ASN American Society of Nephrology

KIDNEY WEEK EDITION

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ASN's First Kidney Health Guidance Focuses on Improving Care for People With Obesity

By Bridget M. Kuehn

ASN's first-ever kidney health guidance provides nephrologists and all other members of the kidney care team with advice on the best practices for holistic, nonstigmatizing care for obesity that incorporates lifestyle, psychosocial interventions, bariatric surgery, and a mounting arsenal of medications (1).

ASN decided to begin creating kidney health guidance to help speed the implementation of the growing array of treatments available for patients with kidney diseases, explained Holly J. Kramer, MD, MPH, professor in the Division of Nephrology at Loyola University Chicago's Stritch School of Medicine, IL. "We wanted to improve the treatment of kidney diseases by guiding nephrologists on the use of newer therapies," Kramer said.

Kramer, who cochaired the Kidney Health Guidance Workgroup that drafted the guidance on obesity care, explained that clinical guidance differs from a clinical practice guideline. Experts draft clinical practice guidelines based on a systematic literature review and weigh recommendations based on the evidence. These formal documents can take years to complete and publish and are typically updated on a regular 5- to 10-year schedule. Experts can more quickly draft guidance documents as practical guides for clinicians and can respond more rapidly to developments in evidence.

Many fields, including specialties like cardiology, oncology, and pulmonology, use clinical guidance documents to supplement more formal clinical guidelines, noted T. Alp Ikizler, MD, FASN, director of the Division of Nephrology and Hypertension at Vanderbilt University Medical Center, Nashville, TN, and cochair of the workgroup. However, Ikizler said that such documents have not been common in nephrology. ASN saw an opportunity to provide additional day-to-day, accessible clinical guidance for the entire team of clinicians treating patients with kidney diseases. "These are going to be living documents," Ikizler said. "There will be updates as the science and information grows within the field."

Continued on page 6 >

Kidney Week Scientific Sessions

THURSDAY

Accelerating Health Care Innovation in Nephrology With Artificial Intelligence
State-of-the-Art Lecture: David C. Rhew, MD

Molecular Bases of Potassium and Sodium Balance, Electrolyte Disorders, and Hypertension
Robert W. Schrier, MD, Endowed Lectureship: Paul A. Welling, MD

Navigating an Evolving Organ Procurement Landscape
Christopher R. Blagg, MD, Endowed Lectureship in Kidney Diseases and Public Policy: Jesse D. Schold, MEd, MS, PhD

FRIDAY

From Reading the Genome for Risk to Rewriting It for Cardiovascular Health
State-of-the-Art Lecture: Sekar Kathiresan, MD

Innovations in Clinical Trial Design and Conduct for New Therapeutic Approaches in CKD
ASN-AHA Donald W. Seldin Young Investigator Award: Hiddo Jan L. Heerspink, PhD

Disparities Between Men and Women in the Course of Diabetic Kidney Disease
Garabed Eknoyan, MD, Endowed Lectureship: Adeera Levin, MD

There Is No Debate: Patients Should Be Part of the Selection Conference
Celeste Castillo Lee Endowed Lectureship: Nichole M. Jefferson, MBA

Water Disorders
Burton D. Rose, MD, Endowed Lectureship: Roger A. Rodby, MD, FASN

APOL1-Mediated Kidney Disease: Variably Proteinuric, Yet Invariably Progressive
Michelle P. Winn, MD, Endowed Lectureship: Kirk N. Campbell, MD, FASN

SATURDAY

Understanding, Improving, and Applying Human Pluripotent Stem Cell-Derived Kidney Tissues
State-of-the-Art Lecture: Melissa H. Little, PhD

Acute Kidney Injury, Repair, and Generation of Kidney Tissue
Homer W. Smith Award Lecture: Joseph V. Bonventre, MD, PhD, FASN

Selective Targeting of the Innate Immune System in Transplantation: The Time Is Now
Barbara T. Murphy, MB BAO BCh, Endowed Lectureship: Fadi G. Lakkis, MD

CKD-MBD and the Skeleton
Jack W. Coburn, MD, Endowed Lectureship: Thomas L. Nickolas, MD, MS

Unraveling the Mysteries of High GFR in Health and Disease
Barry M. Brenner, MD, Endowed Lectureship: Timothy W. Meyer, MD

SUNDAY

The Curative Therapy for Sickle Cell Disease: The Good, the Bad, and the Future
State-of-the-Art Lecture: Michael R. DeBaun, MD, MPH

Inside

FABHALTA®
(iptacopan) 200 mg capsules

FABHALTA IS NOW FDA APPROVED

for the reduction of proteinuria in adults with *primary IgAN* at risk of rapid disease progression, generally a UPCR ≥1.5 g/g[1]

This indication is approved under accelerated approval based on reduction of proteinuria. It has not been established whether FABHALTA® slows kidney function decline in patients with IgAN. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory clinical trial.[1]

Scan to learn more or visit fabhalta-hcp.com/igan

IMPORTANT SAFETY INFORMATION

WARNING: SERIOUS INFECTIONS CAUSED BY ENCAPSULATED BACTERIA

FABHALTA, a complement inhibitor, increases the risk of serious infections, especially those caused by encapsulated bacteria, such as *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* type b. Life-threatening and fatal infections with encapsulated bacteria have occurred in patients treated with complement inhibitors. These infections may become rapidly life threatening or fatal if not recognized and treated early.

- **Complete or update vaccinations for encapsulated bacteria at least 2 weeks prior to the first dose of FABHALTA, unless the risks of delaying therapy with FABHALTA outweigh the risk of developing a serious infection. Comply with the most current Advisory Committee on Immunization Practices (ACIP) recommendations for vaccinations against encapsulated bacteria in patients receiving a complement inhibitor.**
- **Patients receiving FABHALTA are at increased risk for invasive disease caused by encapsulated bacteria, even if they develop antibodies following vaccination. Monitor patients for early signs and symptoms of serious infections and evaluate immediately if infection is suspected.**

Because of the risk of serious infections caused by encapsulated bacteria, FABHALTA is available only through a restricted program under a Risk Evaluation and Mitigation Strategy (REMS) called the FABHALTA REMS.

CONTRAINDICATIONS

- In patients with serious hypersensitivity to FABHALTA or any of the excipients.
- For initiation in patients with unresolved serious infection caused by encapsulated bacteria, including *Streptococcus pneumoniae*, *Neisseria meningitidis*, or *Haemophilus influenzae* type b.

WARNINGS AND PRECAUTIONS

Serious Infections Caused by Encapsulated Bacteria

- FABHALTA, a complement inhibitor, increases a patient's susceptibility to serious, life-threatening, or fatal infections caused by encapsulated bacteria, including *Streptococcus pneumoniae*, *Neisseria meningitidis* (caused by any serogroup, including nongroupable strains), and *Haemophilus influenzae* type b. Life-threatening and fatal infections with encapsulated bacteria have occurred in both vaccinated and unvaccinated patients treated with complement inhibitors. The initiation of FABHALTA is contraindicated in patients with unresolved serious infections caused by encapsulated bacteria.
- Complete or update vaccination against encapsulated bacteria at least 2 weeks prior to the start of FABHALTA, according to the current ACIP recommendations for patients receiving a complement inhibitor. Revaccinate patients in accordance with ACIP recommendations considering the duration of therapy with FABHALTA. Note that ACIP recommends an administration schedule in patients receiving complement inhibitors that differs from the administration schedule in the vaccine prescribing information. If urgent FABHALTA therapy is indicated in a patient who is not up to date with vaccines against encapsulated bacteria according to ACIP recommendations, provide the patient with antibacterial drug prophylaxis and administer these vaccines as soon as possible. The benefits and risks of treatment with FABHALTA, as well as the benefits and risks of antibacterial drug prophylaxis in unvaccinated or vaccinated patients, must be considered against the known risks for serious infections caused by encapsulated bacteria.

Please see additional Important Safety Information and Brief Summary of full Prescribing Information, including Boxed WARNING on the following pages.

IMPORTANT SAFETY INFORMATION (continued)

WARNINGS AND PRECAUTIONS (continued)

Serious Infections Caused by Encapsulated Bacteria (continued)

- Vaccination does not eliminate the risk of serious encapsulated bacterial infections, despite development of antibodies following vaccination. Closely monitor patients for early signs and symptoms of serious infection and evaluate patients immediately if an infection is suspected. Inform patients of these signs and symptoms and instruct patients to seek immediate medical care if they occur. Promptly treat known infections. Serious infection may become rapidly life threatening or fatal if not recognized and treated early. Consider interruption of FABHALTA in patients who are undergoing treatment for serious infections, depending on the risks of interrupting treatment in the disease being treated.

FABHALTA REMS

- FABHALTA is available only through a restricted program under a REMS called FABHALTA REMS, because of the risk of serious infections caused by encapsulated bacteria.
- Under the FABHALTA REMS, prescribers must enroll in the program; counsel patients about the risks, signs, and symptoms of serious infections caused by encapsulated bacteria; provide patients with the REMS educational materials; ensure patients are vaccinated against encapsulated bacteria; prescribe antibacterial drug prophylaxis if patients' vaccine status is not up to date and treatment must be started urgently; and provide instructions to always carry the Patient Safety Card during treatment and for 2 weeks following the last dose of FABHALTA.
- Further information is available by telephone: 1-833-993-2242 or online at **www.FABHALTA-REMS.com**.

Hyperlipidemia

- FABHALTA may increase total cholesterol, LDL cholesterol, and serum triglycerides. Some patients required cholesterol-lowering medications.
- Monitor serum lipid parameters periodically during treatment with FABHALTA and initiate cholesterol-lowering medications, if indicated.

ADVERSE REACTIONS

- The most common adverse reactions (≥5%) in adults with IgAN receiving FABHALTA were upper respiratory tract infection, lipid disorder, and abdominal pain.

DRUG INTERACTIONS

- Concomitant use of CYP2C8 inducers (eg, rifampin) may decrease iptacopan exposure, which may result in loss of or reduced efficacy of FABHALTA. Monitor the clinical response and discontinue use of the CYP2C8 inducer if loss of efficacy of FABHALTA is evident.
- Concomitant use of strong CYP2C8 inhibitors (eg, gemfibrozil) may increase iptacopan exposure, which may result in an increased risk for adverse reactions with FABHALTA. Coadministration with a strong CYP2C8 inhibitor is not recommended.

USE IN SPECIFIC POPULATIONS

- Because of the potential for serious adverse reactions in a breastfed child, breastfeeding should be discontinued during treatment and for 5 days after the final dose.
- FABHALTA is not recommended in patients with severe hepatic impairment (Child-Pugh class C). No dose adjustment is required for patients with mild (Child-Pugh class A) or moderate (Child-Pugh class B) hepatic impairment.

INDICATION

FABHALTA is indicated to reduce proteinuria in adults with primary immunoglobulin A nephropathy (IgAN) at risk of rapid disease progression, generally a urine protein-to-creatinine ratio (UPCR) ≥1.5 g/g.

This indication is approved under accelerated approval based on reduction of proteinuria. It has not been established whether FABHALTA slows kidney function decline in patients with IgAN. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory clinical trial.

Please see additional Important Safety Information on previous page and Brief Summary of full Prescribing Information, including Boxed WARNING on the following pages.

IgAN, immunoglobulin A nephropathy; UPCR, urine protein-to-creatinine ratio.

Reference: 1. Fabhalta. Prescribing information. Novartis Pharmaceuticals Corp.

NOVARTIS

Novartis Pharmaceuticals Corporation
East Hanover, New Jersey 07936-1080

Printed in USA 9/24 424579

FABHALTA® (iptacopan) capsules, for oral use
Initial U.S. Approval: 2023

BRIEF SUMMARY: Please see package insert for full prescribing information.

WARNING: SERIOUS INFECTIONS CAUSED BY ENCAPSULATED BACTERIA

FABHALTA, a complement inhibitor, increases the risk of serious infections, especially those caused by encapsulated bacteria, such as *Streptococcus pneumoniae, Neisseria meningitidis*, and *Haemophilus influenzae* type b *[see Warnings and Precautions (5.1)]*. Life-threatening and fatal infections with encapsulated bacteria have occurred in patients treated with complement inhibitors. These infections may become rapidly life-threatening or fatal if not recognized and treated early.

- **Complete or update vaccination for encapsulated bacteria at least 2 weeks prior to the first dose of FABHALTA, unless the risks of delaying therapy with FABHALTA outweigh the risk of developing a serious infection. Comply with the most current Advisory Committee on Immunization Practices (ACIP) recommendations for vaccinations against encapsulated bacteria in patients receiving a complement inhibitor. See *Warnings and Precautions (5.1)* for additional guidance on the management of the risk of serious infections caused by encapsulated bacteria.**
- **Patients receiving FABHALTA are at increased risk for invasive disease caused by encapsulated bacteria, even if they develop antibodies following vaccination. Monitor patients for early signs and symptoms of serious infections and evaluate immediately if infection is suspected.**

Because of the risk of serious infections caused by encapsulated bacteria, FABHALTA is available only through a restricted program under a Risk Evaluation and Mitigation Strategy (REMS) called the FABHALTA REMS *[see Warnings and Precautions (5.2)]*.

1 INDICATIONS AND USAGE

1.1 Paroxysmal Nocturnal Hemoglobinuria

FABHALTA is indicated for the treatment of adults with paroxysmal nocturnal hemoglobinuria (PNH).

1.2 Immunoglobulin A Nephropathy

FABHALTA is indicated to reduce proteinuria in adults with primary immunoglobulin A nephropathy (IgAN) at risk of rapid disease progression, generally a urine protein-to-creatinine ratio (UPCR) ≥1.5 g/g.

This indication is approved under accelerated approval based on reduction of proteinuria. It has not been established whether FABHALTA slows kidney function decline in patients with IgAN. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory clinical trial.

4 CONTRAINDICATIONS

FABHALTA is contraindicated:

- in patients with serious hypersensitivity to iptacopan or any of the excipients.
- for initiation in patients with unresolved serious infection caused by encapsulated bacteria, including *Streptococcus pneumoniae*, *Neisseria meningitidis*, or *Haemophilus influenzae* type b.

5 WARNINGS AND PRECAUTIONS

5.1 Serious Infections Caused by Encapsulated Bacteria

FABHALTA, a complement inhibitor, increases a patient's susceptibility to serious, life-threatening, or fatal infections caused by encapsulated bacteria, including *Streptococcus pneumoniae, Neisseria meningitidis* (caused by any serogroup, including non-groupable strains), and *Haemophilus influenzae* type b. Life-threatening and fatal infections with encapsulated bacteria have occurred in both vaccinated and unvaccinated patients treated with complement inhibitors. The initiation of FABHALTA treatment is contraindicated in patients with unresolved serious infections caused by encapsulated bacteria.

Complete or update vaccination against encapsulated bacteria at least 2 weeks prior to administration of the first dose of FABHALTA, according to the current ACIP recommendations for patients receiving a complement inhibitor. Revaccinate patients in accordance with ACIP recommendations considering the duration of therapy with FABHALTA. Note that ACIP recommends an administration schedule in patients receiving complement inhibitors that differs from the administration schedule in the vaccine prescribing information. If urgent FABHALTA therapy is indicated in a patient who is not up to date with vaccines against encapsulated bacteria according to ACIP recommendations, provide the patient with antibacterial drug prophylaxis and administer these vaccines as soon as possible. Various durations and regimens of antibacterial drug prophylaxis have been considered, but the optimal durations and drug regimens for prophylaxis and their efficacy have not been studied in unvaccinated or vaccinated patients receiving complement inhibitors, including FABHALTA. The benefits and risks of treatment with FABHALTA, as well as the benefits and risks of antibacterial drug prophylaxis in unvaccinated or vaccinated patients, must be considered against the known risks for serious infections caused by encapsulated bacteria.

Vaccination does not eliminate the risk of serious encapsulated bacterial infections, despite development of antibodies following vaccination. Closely monitor patients for early signs and symptoms of serious infection and evaluate patients immediately if an infection is suspected. Inform patients of these signs and symptoms and instruct patients to seek immediate medical care if these signs and symptoms occur. Promptly treat known infections. Serious infection may become rapidly life-threatening or fatal if not recognized and treated early. Consider interruption of FABHALTA in patients who are undergoing treatment for serious infections, depending on the risks of interrupting treatment in the disease being treated.

FABHALTA is available only through a restricted program under a REMS *[see Warnings and Precautions (5.2)]*.

5.2 FABHALTA REMS

FABHALTA is available only through a restricted program under a REMS called FABHALTA REMS, because of the risk of serious infections caused by encapsulated bacteria *[see Warnings and Precautions (5.1)]*.

Notable requirements of the FABHALTA REMS include the following:

- Prescribers must enroll in the REMS.
- Prescribers must counsel patients about the risk of serious infections caused by encapsulated bacteria.
- Prescribers must provide patients with the REMS educational materials.
- Prescribers must assess patient vaccination status for vaccines against encapsulated bacteria and vaccinate if needed according to current ACIP recommendations two weeks prior to the first dose of FABHALTA.
- Prescribers must provide a prescription for antibacterial drug prophylaxis if treatment must be started urgently, and the patient is not up to date with vaccines against encapsulated bacteria according to current ACIP recommendations at least two weeks prior to the first dose of FABHALTA.
- Pharmacies that dispense FABHALTA must be certified in the FABHALTA REMS and must verify prescribers are certified.
- Patients must receive counseling from the prescriber about the need to receive vaccinations against encapsulated bacteria per ACIP recommendations, the need to take antibiotics as directed by the prescriber, and the early signs and symptoms of serious infections.
- Patients must be instructed to carry the Patient Safety Card with them at all times during treatment and for 2 weeks following the last dose of FABHALTA.

Further information is available by telephone: 1-833-99FABHA (1-833-993-2242) or online at www.FABHALTA-REMS.com.

5.3 Monitoring of PNH Manifestations After FABHALTA Discontinuation

In PNH patients, after discontinuing treatment with FABHALTA, closely monitor patients for at least 2 weeks after the last dose for signs and symptoms of hemolysis. These signs include elevated lactate dehydrogenase (LDH) levels along with a sudden decrease in hemoglobin or PNH clone size, fatigue, hemoglobinuria, abdominal pain, dyspnea, major adverse vascular events (such as thrombosis, stroke and myocardial infarction), dysphagia, or erectile dysfunction. If discontinuation of FABHALTA is necessary, consider alternative therapy.

If hemolysis occurs after discontinuation of FABHALTA, consider restarting treatment with FABHALTA, if appropriate, or initiating another treatment for PNH.

5.4 Hyperlipidemia

FABHALTA may increase total cholesterol, LDL-cholesterol, and serum triglycerides *[see Adverse Reactions (6.1)]*.

Of the 54 FABHALTA-treated patients who had a normal total cholesterol level at baseline in APPLY-PNH, 43% developed Grade 1 hypercholesterolemia during the randomized treatment period. One FABHALTA-treated patient in APPLY-PNH experienced increased total cholesterol that worsened to Grade 2 from Grade 1 at baseline.

Of the 34 FABHALTA-treated patients who had a normal cholesterol level at baseline in APPOINT-PNH, 24% developed Grade 1 hypercholesterolemia during the core treatment period.

Of the 60 FABHALTA-treated patients who had LDL-cholesterol ≤ 130 mg/dL at baseline in APPLY-PNH, 17% developed LDL-cholesterol > 130-160 mg/dL, 8% developed LDL-cholesterol > 160-190 mg/dL, and 7% developed LDL-cholesterol > 190 mg/dL during the randomized treatment period. Of the 36 FABHALTA-treated patients who had LDL-cholesterol ≤ 130 mg/dL at baseline in APPOINT-PNH, 11% developed LDL-cholesterol > 130-160 mg/dL and 3% developed LDL-cholesterol > 160-190 mg/dL.

Of the 52 patients with normal triglyceride levels at baseline in APPLY-PNH, 23% developed Grade 1 elevated triglycerides during the randomized treatment period. Three FABHALTA-treated patients in APPLY-PNH experienced an increase in triglycerides from Grade 1 to Grade 2.

Of the 37 FABHALTA-treated patients who had a normal triglyceride level at baseline in APPOINT-PNH, 27% developed Grade 1 elevated triglycerides in the core treatment period.

Of the 102 FABHALTA-treated patients in APPLY-PNH and APPOINT-PNH, two patients required cholesterol-lowering medications.

Monitor serum lipid parameters periodically during treatment with FABHALTA and initiate cholesterol-lowering medication, if indicated.

6 ADVERSE REACTIONS

The following clinically significant adverse reaction is discussed in greater detail in other sections of the labeling:

- Serious Infections Caused by Encapsulated Bacteria *[see Warnings and Precautions (5.1)]*.
- Hyperlipidemia *[see Warnings and Precautions (5.4)]*.

6.1 Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

Paroxysmal Nocturnal Hemoglobinuria (PNH)

The data described below reflects the exposure in adults with PNH who received FABHALTA (n = 62) or anti-C5 treatment (US-approved and non-US-approved eculizumab product or US-approved and non-US-approved ravulizumab product, n = 35) in APPLY-PNH [NCT04558918] and adults who received FABHALTA (n = 40) in APPOINT-PNH

[NCT04820530] at the recommended dosing regimen for 24 weeks. In APPLY-PNH, serious adverse reactions were reported in 2 (3%) patients with PNH receiving FABHALTA. Serious adverse reactions included pyelonephritis, urinary tract infection and COVID-19. In APPOINT-PNH, serious adverse reactions were reported in 2 (5%) patients with PNH receiving FABHALTA. Serious adverse reactions included COVID-19 and bacterial pneumonia. The most common adverse reactions (≥ 10%) with FABHALTA were headache, nasopharyngitis, diarrhea, abdominal pain, bacterial infection, viral infection, nausea, and rash.

Table 1 describes the adverse reactions that occurred in > 5% of patients treated with FABHALTA in the APPLY-PNH or APPOINT-PNH studies.

Table 1: Adverse Reactions Reported in > 5% of Patients Treated with FABHALTA in APPLY-PNH or APPOINT-PNH Studies (24-Week Treatment Period)

Adverse reactions	APPLY-PNH		APPOINT-PNH
	FABHALTA (N = 62) n (%)	Anti-C5 (Eculizumab or Ravulizumab) (N = 35) n (%)	FABHALTA (N = 40) n (%)
Headache[a]	12 (19)	1 (3)	11 (28)
Nasopharyngitis[b]	10 (16)	6 (17)	6 (15)
Diarrhea	9 (15)	2 (6)	3 (8)
Abdominal pain[a]	9 (15)	1 (3)	3 (8)
Bacterial infection[c]	7 (11)	4 (11)	2 (5)
Nausea	6 (10)	1 (3)	2 (5)
Viral infection[d]	6 (10)	11 (31)	7 (18)
Arthralgia	5 (8)	1 (3)	0
Thrombocytopenia[a]	4 (6)	0	0
Dizziness	4 (6)	0	1 (3)
Systemic hypertension[a]	4 (6)	0	0
Lipid disorder[e]	4 (6)	0	3 (8)
Rash[f]	2 (3)	0	4 (10)

[a]Includes similar terms.
[b]Nasopharyngitis contains: rhinitis allergic, upper respiratory tract infection, pharyngitis, rhinitis.
[c]Bacterial infection contains: pyelonephritis, urinary tract infection, bronchitis bacterial, bronchitis haemophilus, cholecystitis, folliculitis, cellulitis, arthritis bacterial, sepsis, klebsiella infection, staphylococcal infection, *Pseudomonas* infection, hordeolum, pneumonia bacterial.
[d]Viral infection contains: COVID-19, herpes zoster, oral herpes, nasal herpes, influenza A virus test positive, influenza.
[e]Lipid disorder contains: dyslipidemia, blood cholesterol increased, low density lipoprotein increased, hypercholesterolemia, blood triglycerides increased, hyperlipidemia.
[f]Rash contains: dermatitis allergic, acne, erythema multiforme, rash maculo-papular, rash erythematous.

Clinically relevant adverse reactions reported in less than or equal to 5% of patients includes urticaria in one patient (3%) in APPOINT-PNH.

Description of Select Adverse Reactions (graded per NCI CTCAE Version 4.03 unless noted otherwise)

Platelet Count Decreased
Of the 37 FABHALTA-treated patients who had normal platelet counts at baseline in APPLY-PNH, 43% experienced any Grade thrombocytopenia during the randomized treatment period. Three FABHALTA-treated patients in APPLY-PNH experienced decreased platelets that worsened to Grade ≥ 3 from baseline (one patient with normal platelets that worsened to Grade 4, one patient with baseline Grade 1 that worsened to Grade 4, and one patient with baseline Grade 3 that worsened to Grade 4).

Immunoglobulin A Nephropathy (IgAN)
The safety of FABHALTA was evaluated in APPLAUSE-IgAN, a randomized double-blind clinical study in adults with IgAN (eGFR ≥ 20 mL/min/1.73 m^2 at baseline).

The data below reflect FABHALTA exposure in 235 patients with IgAN (eGFR ≥ 20 mL/min/1.73 m^2 at baseline) with a median duration of 43 weeks (up to 104 weeks) in APPLAUSE-IgAN. Table 2 describes the adverse reactions that occurred in ≥ 3% of patients treated with FABHALTA and were ≥ 2% higher in frequency than placebo. All of these adverse reactions were mild or moderate in severity.

Table 2: Adverse Reactions Reported in ≥ 3% of Adult Patients with IgAN (eGFR ≥ 20 mL/min/1.73 m^2) Treated with FABHALTA and ≥ 2% Higher in Frequency Than Placebo in APPLAUSE-IgAN

Adverse reaction	FABHALTA (N = 235) n (%)	Placebo (N = 235) n (%)
Upper respiratory tract infection	20 (9)	16 (7)
Lipid disorder[1]	15 (6)	10 (4)
Abdominal pain[1]	15 (6)	5 (2)
Nausea	8 (3)	2 (1)
Dizziness	7 (3)	2 (1)

[1] Includes similar terms.

7 DRUG INTERACTIONS

7.1 CYP2C8 Inducers
Concomitant use of CYP2C8 inducers (e.g., rifampin) may decrease iptacopan exposure, which may result in loss of or reduced efficacy of FABHALTA. Monitor the clinical response and discontinue use of the CYP2C8 inducer if loss of efficacy of FABHALTA is evident.

7.2 Strong CYP2C8 Inhibitors
Concomitant use of strong CYP2C8 inhibitors (e.g., gemfibrozil) may increase iptacopan exposure, which may result in an increased risk for adverse reactions with FABHALTA. Coadministration with a strong CYP2C8 inhibitor is not recommended.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy
Risk Summary
Available data from clinical trials with FABHALTA use in pregnant women are insufficient to identify a drug-associated risk of major birth defects, miscarriage, or other adverse maternal or fetal outcomes. There are risks to the mother and fetus associated with untreated PNH and IgAN in pregnancy *(see Clinical Considerations)*. The use of FABHALTA in pregnant women or women planning to become pregnant may be considered following an assessment of the risks and benefits.

In animal reproduction studies, oral administration of iptacopan to pregnant rats and rabbits during organogenesis at exposures 4- to 6-times the human exposure (based on AUC) at the maximum recommended human dose (MRHD) of 200 mg twice daily did not induce embryo or fetal toxicity *(see Data)*.

The estimated background risk of major birth defects and miscarriage for the indicated populations is unknown. All pregnancies have a background risk of major birth defects, loss, or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriages in clinically recognized pregnancies is 2% to 4% and 15% to 20%, respectively.

Clinical Considerations
Disease-Associated Maternal and/or Embryo/Fetal Risk
PNH in pregnancy is associated with adverse maternal outcomes, including worsening cytopenias, thrombosis, infections, bleeding, miscarriages, increased maternal mortality, and adverse fetal outcomes, including fetal death and premature delivery.

IgAN in pregnancy is associated with adverse maternal outcomes, including increased rates of cesarean section, pregnancy-induced hypertension, pre-eclampsia and preterm delivery, and adverse fetal/neonatal outcomes, including stillbirth and low birth weight.

Data
Animal Data
In an embryo-fetal development study in rats, oral administration of iptacopan during organogenesis did not cause embryo-fetal toxicity when given up to the highest dose of 1,000 mg/kg/day, which corresponds to 4-times the MRHD based on AUC.

In an embryo-fetal development study in rabbits, oral administration of iptacopan during organogenesis did not cause embryo-fetal toxicity when given up to the highest dose of 450 mg/kg/day, which corresponds to 6-times the MRHD based on AUC.

In a pre- and postnatal development study in rats, oral administration of iptacopan during gestation, parturition, and lactation did not cause adverse effects in offspring when given up to the highest dose of 1,000 mg/kg/day, which corresponds to 4-times the MRHD based on AUC.

8.2 Lactation
Risk Summary
There are no data on the presence of iptacopan or its metabolites in either human or animal milk, the effects on the breastfed child or on milk production. Since many medicinal products are secreted into human milk, and because of the potential for serious adverse reactions in a breastfed child, breastfeeding should be discontinued during treatment and for 5 days after the final dose.

8.4 Pediatric Use
Safety and effectiveness in pediatric patients with PNH or IgAN have not been established.

8.5 Geriatric Use
There were 29 PNH patients 65 years of age and older in APPLY-PNH and APPOINT-PNH *[see Clinical Studies (14) in the full prescribing information]*. Of the total number of FABHALTA-treated patients during the 24-week treatment period in these studies, 21 (20.6%) were 65 years of age and older, while 7 (6.9%) were 75 years of age and older. There were 8 IgAN patients 65 years of age and older in APPLAUSE-IgAN *[see Clinical Studies (14) in the full prescribing information]*. Of the total number of FABHALTA-treated patients, 3 (2.4%) were 65 years of age and older. Clinical studies of FABHALTA did not include sufficient numbers of patients 65 years of age and older to determine whether they respond differently from younger patients.

8.7 Hepatic Impairment
The use of FABHALTA is not recommended in patients with severe hepatic impairment (Child-Pugh class C). No dose adjustment is required for patients with mild (Child-Pugh class A) or moderate (Child-Pugh class B) hepatic impairment *[see Clinical Pharmacology (12.3) in the full prescribing information]*.

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East Hanover, New Jersey 07936

For more information, visit www.FABHALTA.com or call 1-888-669-6682.

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ASN's First Kidney Health Guidance Focuses on Improving Care for People With Obesity

Continued from cover

ASN selected obesity management as the topic for the first health guidance. Approximately 42% of US adults are classified as having obesity, a proportion that has grown by more than 10% over the past 2 decades (1). The condition can contribute to the development of kidney diseases, worsen a patient's prognosis, and exacerbate comorbidities, Ikizler explained. It has historically been difficult to manage, but he noted that a growing number of tried and true, as well as new, interventions are available to treat the condition.

"Clinicians are having their patients come to them with questions about what the best [weight loss] approach would be for them, whether it's medical therapy, behavioral therapy, [or] lifestyle modifications," said ASN President Deidra C. Crews, MD, ScM, FASN. "That made us think this was a great topic [on which] to begin our Kidney Health Guidance."

Empathetic care

The ASN Kidney Health Guidance on the Management of Obesity in Persons Living with Kidney Diseases emphasizes the importance of discussing weight loss with patients in an empathetic and nonstigmatizing manner. "The first thing would be asking the patient if they feel comfortable discussing weight," Kramer said. "Some people feel uncomfortable discussing their weight, so you have to be really careful about how you discuss it with the patient."

Some tips include avoiding stigmatizing language, such as describing someone as *fat* or *obese*, and using the term *unhealthy weight* instead. Kramer suggested discussing the potential of weight loss to help patients achieve their kidney care goals. For example, she explained that the goal may be losing weight to prevent progression, improve quality of life, or help meet transplant eligibility requirements.

The guidance also emphasizes the importance of addressing the patient's mental health. Patients with obesity and kidney diseases may feel shame or blame related to their conditions. Patients may also have mental health conditions like anxiety, depression, substance use disorders, eating disorders, or other mental health conditions that may make weight management difficult, Kramer explained. "If you don't get in there and figure out the psychological components that are driving the difficulties in managing weight and lifestyle, you are not going to get very far," she explained.

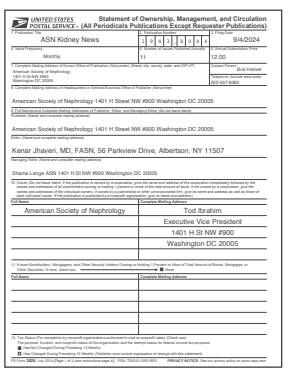
The guidance recommends lifestyle changes, such as dietary patterns, creating an exercise schedule suitable for the patient's circumstances and their comorbidities, and getting adequate sleep, as the foundational steps for patients interested in weight loss. "You need to give the lifestyle a chance to work," she encouraged. It also acknowledges that social determinants of health, such as food or housing insecurity, economic status, health literacy, and access to transportation, may all impede treatment for obesity and kidney diseases. These complexities must be addressed in the treatment plan.

Many patients will not experience sustained weight loss from lifestyle changes; for these patients, the guidance recommends considering medications as part of the treatment plan. The guidance highlights glucagon-like peptide-1 (GLP-1) receptor agonists based on evidence that they can slow chronic kidney disease progression, prevent heart failure, and improve patient quality of life and functioning. The guidance also details other medications commonly used for weight loss that may be appropriate and explains what nephrologists need to consider about their use in patients with kidney diseases.

"There's been a lot of excitement about the studies that have come out showing the benefits of these medications in terms of their ability to help people to lose weight but also how they can actually lead to slower progression of kidney disease as well as reductions in cardiovascular outcomes," Crews stated. "There's still a number a number of challenges around making sure that they're getting to everyone who might benefit from them."

She explained that many patients have struggled to get such medications at an affordable cost, indicating a need for more advocacy. Additionally, some clinicians have been hesitant to prescribe them.

"Most nephrologists may not feel comfortable prescribing antiobesity medications," Kramer acknowledged. "We need to do more to educate nephrologists on how to safely utilize these medications across the entire spectrum of kidney disease stages."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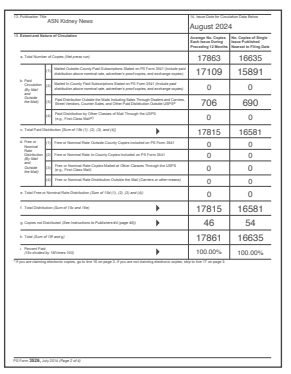
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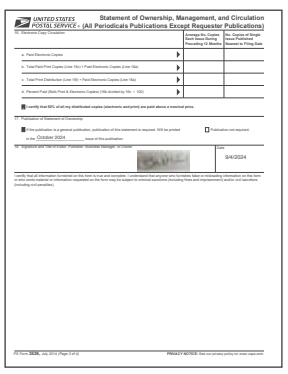
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For example, Kramer explained that for patients with more advanced kidney diseases, nephrologists must more slowly and cautiously escalate doses of weight-loss medications and carefully monitor for side effects, which may be more common in patients with worse kidney function. "Some people just don't tolerate higher doses," she said. "You've got to go slow. People can get nauseous or ill from the medication."

Ikizler noted how essential it is to recognize that these medications, as well as more invasive approaches like metabolic or bariatric surgery, can be effective in patients with kidney diseases. However, nephrologists need to follow up closely. "We need to be very diligent and provide oversight," he said. "We cannot just sort of prescribe and forget."

As noted in the guidance, kidney diseases should not be considered a contraindication for metabolic and bariatric surgery. Patients with advanced kidney diseases or who require dialysis may have higher rates of complications or death after surgery than those with earlier stages of kidney diseases. Still, overall rates of mortality in patients with kidney diseases are low. It also highlights factors that may make some patients poor candidates for bariatric surgery, such as substance use disorders or eating disorders.

In addition to providing practical advice for nephrologists on all of these issues, the guidance also notes that there are likely unappreciated benefits of these interventions for patients with kidney diseases beyond just weight loss. Ikizler said he sees a role for nephrologists in helping the kidney care team and patients understand them. "It may have multiple different impacts [on kidney health], and we need to figure those out," he said. "Just losing weight should not be our primary or only target. We should also look at other metabolic benefits that come from these [therapies]."

Team-based approach

Kramer acknowledged that nephrologists may have limited time for patient consultations and limited training in obesity care, lifestyle modifications, or mental health. "We know that nephrologists have a lot to do," Ikizler said. "We are very thorough and very involved in our patients' care. [Obesity management] will bring an additional [commitment]. The way to manage that is to delegate or refer certain responsibilities and tasks to other health care team members."

The guidance emphasizes the benefits of collaborating with primary care physicians, advanced practice professionals, nurse educators, dieticians, mental health clinicians, mobility and physical activity experts, and others to provide holistic care for obesity. "It's going to take a team approach to help these patients not only get the weight off but keep it off," Kramer said.

Some clinics already hire nurse educators to work with patients with kidney diseases, and they can help patients understand the influence of weight on kidney health, Kramer noted. However, most public and private payers do not currently cover these services. Medicare and private insurance typically pay for medical nutrition therapy for up to 3 years. Still, Kramer said that only 1 in 10 patients with kidney diseases ever meets with a registered dietician.

Ikizler noted that nurses can play a pivotal role in patient follow-up and medication management. He noted that social workers can also help address socioeconomic challenges and psychosocial concerns and help patients with medication access, affordability, and medication adherence.

Kramer also opined that new care models are needed. For example, cardiologists routinely suggest that patients undergo physical rehabilitation after a heart attack, for which they get training and guidance on how to exercise. However, patients with kidney diseases do not typically receive this kind of support. "That is a huge gap in care because exercise is good not only for the physical functioning of the body but also for mental health," she said.

The guidance also attempts to define the nephrologist's role in using these new classes of medications and when they might need to collaborate with other specialties. Ikizler noted that several specialties, including endocrinology, cardiology, and general and internal medicine, also prescribe weight-loss medications. "It is important we talk with them all the time," he said. "We need to communicate [medication decisions] and sometimes allow others to prescribe these medications but also provide some oversight from the kidney perspective."

Advocating for access

Kramer acknowledged that some of the recommendations are aspirational and that some interventions may not be available in every practice context or accessible to every patient. "Not everyone will have access to all of these therapies," she explained. "It depends on your practice." For example, some practices may not have dieticians with expertise in kidney diseases and obesity or clinicians who can help patients overcome physical barriers to exercise. Access to bariatric surgery may also be limited because few centers accept patients who are considered high risk or those who lack insurance coverage. Patients from rural areas may also have limited access to mental health and other types of specialty care. Out-of-pocket costs are also a major barrier to many patients accessing newer weight-loss therapies like GLP-1 receptor agonists and gastric inhibitory polypeptide receptor agonists.

The guidance also lays out the need for policy solutions to address some of these patient access and systemic problems. Ikizler noted that many professional societies, like the American Cancer Society, have successfully leveraged the expert consensus in their clinical guidance documents to help push for policy change. "We felt we had to set the bar high," Ikizler said. "Having these guidance documents is critical to making policy changes."

Kramer notes that through recent government price negotiations, sodium-glucose cotransporter-2 inhibitors will soon be available to Medicare patients at reduced costs. She hopes that policymakers will include GLP-1 agonists in future price negotiations. In the meantime, nephrologists can advocate for coverage and reduced costs for patients covered by Medicaid or private insurance. She argued that there is a strong cost-benefit argument for coverage, which may help keep someone from needing dialysis or may allow them to undergo transplant.

Ikizler highlighted the importance of advocating for policy changes that increase access to weight-loss medications and that prioritize access for patients with kidney diseases who are at high risk of complications. He also said that policies supporting patient access to multidisciplinary care teams—including nurses, social workers, mental health clinicians, dieticians, and other specialties—are also needed. "Policies should allow us to create a health care team that manages our patients," he said. "Obesity management is just one component of kidney care that requires a team effort."

In the meantime, Kramer emphasized that the most critical advice for nephrologists consulting the guidance is to start by talking with their patients about weight in a supportive way. "Ask your patient if they want to discuss their weight," she suggested. "If you ask them in an empathetic way, in a nonthreatening way, I bet the majority of people would say, 'Yes, I want to talk about it.' We really need to talk about it."

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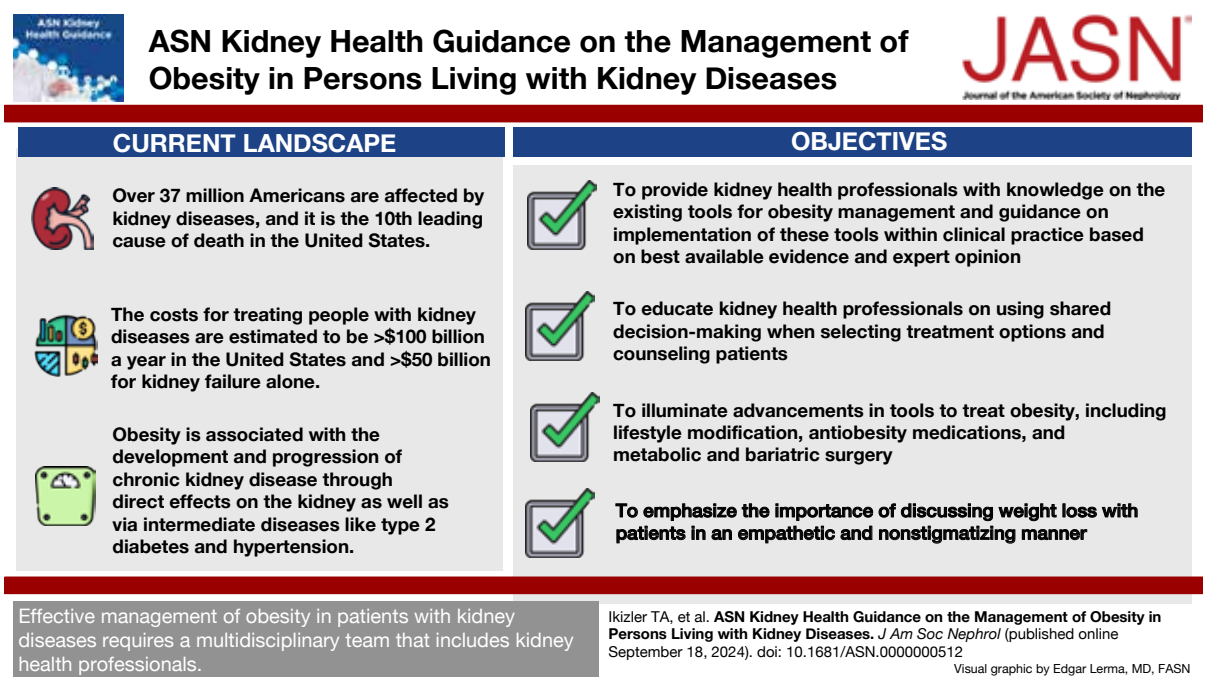

Are you a fellow with a tip or idea you'd like to share with your fellow peers and the broader kidney community?

Send your idea to the ASN *Kidney News* Fellows First column at kidneynews@asn-online.org

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Understanding the TDAPA for Treating Anemia in Patients on Dialysis

By Katherine Kwon

Hypoxia-inducible factor-prolyl hydroxylase inhibitors (HIF-PHIs) are a new class of medications to treat anemia in patients with chronic kidney disease (CKD). Daprodustat was approved in February 2023 (1), and vadadustat was approved in March 2024 (2). Medicare will temporarily pay for these new medications differently, with potentially significant economic impacts on dialysis units. Nephrologists, who are tasked with deciding which anemia treatments to use, should understand how the different payments work.

Medicare pays for dialysis services using the End-Stage Renal Disease Prospective Payment System (ESRD PPS), colloquially known as "the bundle." This per-treatment payment covers all aspects of dialysis except for the physician fees; it includes payment to the dialysis unit, disposable supplies, lab tests, and many dialysis-related medications. The payment amount is adjusted annually.

There has been criticism of the effect that ESRD PPS has on dialysis drug innovation, since any increase in medication costs comes out of the bundle, thereby decreasing dialysis units' margin. In response, the Centers for Medicare & Medicaid Services (CMS) created the Transitional Drug Add-on Payment Adjustment (TDAPA). TDAPA allows for a separate payment for new dialysis medications, outside of the bundle, for a limited time. Currently, TDAPA reimburses dialysis units for the drugs based on 100% of their average sale price. HIF-PHIs, which treat an existing category of CKD-related conditions (e.g., anemia) will be paid for under TDAPA for 2 years. At the end of the 2-year period, payment for HIF-PHIs will fall into the bundle. There is no current plan to adjust the bundle payment once HIF-PHIs are included. CMS has suggested adding an additional 3 years of payments at 65% of actual CMS expenditures on a per-treatment basis, but this rule has not been finalized (3).

Although dialysis facilities are reimbursed for the drugs at their average sale price, there is still a significant economic incentive to use HIF-PHIs during the TDAPA period because their use can substantially replace the erythropoietin analogues (ESAs), which are paid for inside of the bundle. TDAPA payments therefore shift anemia costs outside of the bundle, increasing the profit margin for the dialysis unit. It is not yet clear how aggressively the dialysis companies will shift their anemia management to capture these savings. Daprodustat started the 2-year TDAPA period on October 1, 2023, and will end on September 30, 2025. When CMS analyzed payments for daprodustat for the first quarter of the TDAPA period, it found total CMS expenditures of only $23,705 per month (4).

Only patients with traditional Medicare as their primary coverage will be governed by the CMS rules for TDAPA. Patients with Medicare Advantage or commercial insurance plans will have different coverage for novel drugs that depend on the plans' negotiations with dialysis companies. The plans' experience with TDAPA is variable, and there are reports of an inability to process submitted claims (5).

Another challenge is encountered when patients on dialysis are participating in value-based care, such as Comprehensive Kidney Care Contracting (CKCC). Costs incurred under Medicare Part D, which covers prescriptions, are not included in calculating the total cost of care in patients using CKCC. However, TDAPA drug payments are included in Part B, which does affect the total cost of care. Therefore, value-based care companies may be less likely to advocate for a shift from ESAs to HIF-PHIs because their Part B claims will increase. Several of the larger dialysis companies participate in the CKCC models.

As always, patient safety and drug efficacy are the primary concerns in selecting the appropriate anemia treatment for patients with CKD. The HIF-PHIs provided data of noninferiority compared with ESAs in their applications for US Food and Drug Administration approval, and the black box warnings are the same for both classes of drugs (6). Medical directors may be asked to consider changing the anemia management algorithms based on their different reimbursements. They should be aware of the economic implications of their formulary and clinical decisions, both for the impact on individual patients as well as on the health care system. ■

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The author reports no conflicts of interest.

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ASN Executive Vice President's Update

Highlighting 13 of ASN's Accomplishments in 2024

By Tod Ibrahim

Considered "the brisk philosopher of merchandising," Elmer Wheeler (1903–1968) spent his career advising "merchants how to win sales and influence customers" (1). His first piece of marketing advice was, "Don't sell the steak—sell the sizzle," because "The sizzle has sold more steaks than the cow ever has, although the cow is, of course, mighty important." Recognizing the many different "cuts of beef," this editorial describes 13 of ASN's prime achievements in 2024.

To promote "high-quality, person-directed care across the spectrum of kidney health and diseases spanning the kidney care journey from screening and early detection to diagnosis, treatment, and palliative care," the society produced the first **ASN Kidney Health Guidance** (2). In Management of Obesity in Persons Living with Kidney Diseases, ASN encourages nephrologists to gain knowledge about the tools and interventions available to help adults with obesity and kidney diseases safely lose weight to improve kidney and metabolic health and overall quality of life. The guidance "provides kidney health professionals with knowledge on the existing tools for obesity management and guidance on implementation of these tools within clinical practice based on best available evidence and expert opinion" (3).

Spurred by recommendations from the ASN Task Force on Augmented Intelligence and Digital Health, the **ASN Partnership for Responsible Augmented Intelligence in Kidney Health** launched. As health care adapts to the use of artificial intelligence (AI), and medical specialty societies learn how best to support their members, this initiative advances understanding of AI, machine learning, and generative AI by focusing on clinical domains, such as acute kidney injury, chronic kidney disease, dialysis, and transplantation. The Partnership for Responsible Augmented Intelligence supports patient care, drives research, stimulates innovation, and educates the nephrology workforce.

As a result of advocacy by ASN and other members of the kidney community, the **Centers for Disease Control and Prevention (CDC) designated "chronic renal failure" as an immunocompromising condition**, a determination that offers Americans with kidney failure earlier access to life-saving vaccines (4). Since 2016, ASN has collaborated with CDC to improve care for people living with kidney diseases, including through advancing infection-prevention practices, promoting best practices in vascular access, and educating nephrology fellows on the value of home therapies as a treatment option.

Following several legislative and regulatory successes that are advancing kidney health in the United States, ASN initiated **Transforming Kidney Health Research**. In partnership with the American Association of Kidney Patients, the American Kidney Fund, the American Society of Pediatric Nephrology, and the National Kidney Foundation (NKF), a Blue-Ribbon Panel is developing a bold, comprehensive research plan. To accelerate innovation that will deliver better diagnostics, therapies, and tools for preventing and treating kidney diseases, this community-wide plan will:

- Identify existing and emerging areas of opportunity for kidney research across the entire federal government.
- Develop federal appropriations recommendations to fund these opportunities.
- Become a pillar for the kidney community to advocate for a major increase in federal appropriations over fiscal years 2026–2028.

Similar efforts in other specialties increased federal research appropriations significantly, including by more than $3 billion from the National Institutes of Health to research type 1 diabetes (5).

During the American Nephrology Nurses Association (ANNA) 2024 Nephrology Nursing Summit, ASN cofacilitated a session—"Navigating Challenges Together: A Panel Discussion on **Kidney Care Workforce Solutions**"—with ANNA. This session was an important part of a broader effort to strengthen the nephrology workforce, starting with the dialysis care team in the United States (6). Additionally, ASN joined forces with the American Academy of Family Physicians, the American Academy of Pediatrics, and the American College of Emergency Physicians to lead a plenary discussion, "Physician Workforce Challenges, Strategies, and Future Opportunities for Specialty Societies," at the Council of Medical Specialty Societies (CMSS) Annual Meeting.

To produce the **Diagnostic Excellence: Estimated Glomerular Filtration Rate (eGFR) Toolkit**, ASN was one of 18 societies selected to participate in the CMSS Promoting Diagnostic Excellence Across Medicine program. Funded by the Gordon and Betty Moore Foundation and The John A. Hartford Foundation, this program focuses on "diagnostic safety, quality, and equity" (7). In an effort to reduce disparities in diagnosis and in equitable access to care, ASN's toolkit informs the broader health community of the importance of removal of race from the eGFR calculation. The toolkit explains what the changes are, why they were made, and what they mean. Easily accessible, downloadable, and printable, the toolkit is a "one-stop shop" with needed resources, links to important information, and answers to frequently asked questions.

Additionally, *Time* selected Cynthia Delgado, MD, FASN, and Neil R. Powe, MD, FASN—who cochaired the NKF-ASN Task Force on Reassessing the Inclusion of Race in Diagnosing Kidney Diseases—as two of the 100 "most influential people in health in 2024" for spearheading efforts to remove race from clinical algorithms (8). Ziyad Al-Aly, MD, FASN, also made the list for "detailing the numerous organ systems affected by Long COVID, the risks posed by multiple COVID-19 infections, and how Long COVID compares with chronic symptoms of viruses like the flu."

Funded by a grant from the National Institute of Diabetes and Digestive and Kidney Diseases, ASN originated **Fostering Inclusivity in Dialysis (FinD)**, a groundbreaking leadership training program. To produce future generations of leaders regardless of their current level of practice, FinD focuses on dialysis access—particularly to improve outcomes and experience for people who are living with kidney diseases who are impacted by health care disparities—and offers yearlong leadership training, career-specific mentoring, and networking experience for early career nephrologists, including those who are in fellowship training.

"The best way for ASN to engage its more than 21,000 members—and the broader kidney community, including people living with kidney diseases—is to pursue practical, purposeful, and powerful initiatives that will advance the field."

With support from Amgen; Otsuka; Travere Therapeutics, Inc.; and Vera Therapeutics, Inc., ASN introduced the **Glomerular Diseases Collaborative (GD-C)**. GD-C promotes high-quality care for people living with glomerular diseases and stimulates opportunities to address gaps in knowledge, training, continuing education, and awareness across the spectrum of glomerular diseases. To close these gaps, GD-C improves access to care; increases nephrologists' clinical knowledge, including those who specialize in glomerular diseases; enhances access to clinical trials, which includes engaging people living with kidney diseases, informing nephrologists, and strengthening the infrastructure; and conducts economic analyses.

As part of the We're United 4 Kidney Health campaign, ASN committed to **"transform transplant and increase access to donor kidneys"** (9). Continuing to honor this promise, ASN:

- advocated successfully for the largest funding increase ever ($23 million) to the Health Resources and Services Administration (HRSA) to carry out the Organ Procurement and Transplantation Network Modernization Initiative, implementing a new law to overhaul the transplant system that ASN championed in 2023;
- shaped the Centers for Medicare & Medicaid Servies (CMS) Increasing Organ Transplant Access Model;
- partnered with the American Society of Transplantation (AST) to apply for Accreditation Council for Graduate Medical Education (ACGME) accreditation for transplant nephrology (at press time, ACGME was considering the ASN-AST proposal);
- convinced CMS and HRSA to collect information about what happens between when a nephrologist refers a patient for transplant and when the patient is added (or not added) to the waitlist; and
- worked with NKF to ensure that race was removed from the Kidney Donor Risk Index.

Suzanne Watnick, MD, FASN, is serving as the inaugural **ASN Health Policy Scholar in Residence**. In piloting this unique approach to increasing nephrology's presence within the federal government, Watnick works directly with the society's members, leadership, and staff to further federal legislative and regulatory efforts to ensure excellence in kidney care, increase federal funding for kidney research, and bolster the nephrology workforce. To increase the federal government's commitment to kidney health, she also serves as ASN's point of contact to the US Department of Health and Human Services (HHS) through the Kidney Innovation Accelerator (KidneyX), a public-private partnership between HHS and ASN "to accelerate innovation in the prevention, diagnosis, and treatment of kidney diseases" (10).

An **aligned editorial structure now exists** for the *Journal of the American Society of Nephrology* (*JASN*), the *Clinical Journal of the American Society of Nephrology* (*CJASN*), and *Kidney360*. On January 1, 2024, Rajnish Mehrotra, MD, MS, FASN, started his term as the seventh *JASN* editor-in-chief (EIC); Connie Rhee, MD, MSc, started her term as the fourth *CJASN* EIC; and Michael Allon, MD, started his fifth year as the founding EIC of *Kidney360*. Under the new editorial structure, Mehrotra is also the inaugural senior EIC of the ASN Portfolio of Journals, and the three EICs are "working together to further enhance the author experience by providing clarity regarding the scope of each journal, a simplified submission process, and a process of seamless transfer between journals such that we continue to attract the best kidney-related research from around the world to the ASN journals" (11).

In June, ASN hosted the first **Kidney Innovation Conference**, which brought together the Kidney Health Initiative, KidneyX, and KidneyCure communities. Including keynote addresses stressing the need to accelerate innovation for people living with kidney failure, this cutting-edge meeting included presentations by patients and patient advocates, insights into the ethical and legal considerations for xenotransplantation and other emerging therapies, perspectives on AI and digital health, discussions about real-world evidence and clinical trials, suggestions for integrating health equity into innovations, and a poster session to showcase the research of KidneyCure grant recipients. Between a bipartisan panel of members of Congress and focused discussion with CMS on "Maximizing Reimbursement Pathways for New Kidney Drugs, Devices, and Biologics," the conference also tackled myriad political realities.

As the world's premier nephrology meeting, **ASN Kidney Week will add new features in 2024**. Implementing recommendations from the ASN Late-Breaking Clinical Trials Task Force, these additions will include multiple sessions to highlight late-breaking clinical trials, more presentation time for high-impact kidney research, and increased collaboration with *JASN*, the *Journal of the American Medical Association*, and *The New England Journal of Medicine*. Besides emphasizing exciting science throughout the meeting, ASN strives to ensure that the breakthroughs presented at Kidney Week extend beyond the more than 12,000 people convening in San Diego, CA. Mark your calendars because ASN Kidney Week 2025 will take place Wednesday, November 5, through Sunday, November 9, in Houston, TX.

The best way for ASN to engage its more than 21,000 members—and the broader kidney community, including people living with kidney diseases—is to pursue practical, purposeful, and powerful initiatives that will advance the field. As illustrated in the Table, these 13 selected accomplishments are engaging more than 350 volunteers.

In total, an estimated 1000 members (and other key constituents, such as people living with kidney diseases) contribute to ASN's success by serving on the society's committees, work groups, task forces, and other panels. The society's leadership, staff, and I thank each of them for serving. We encourage you to email operations@asn-online.org if you are interested in volunteering to help ASN achieve its vision of "A world without kidney diseases" (12). ■

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Table. More than 350 member volunteers contributed to 13 selected accomplishments

Accomplishment	Chair/leader	Members[a]
ASN Kidney Health Guidance (KHG) KHG Oversight Committee KHG Obesity and Kidney Diseases Workgroup	K. Lui T. Ikizler H. Kramer	17 18
ASN Partnership for Responsible Augmented Intelligence in Kidney Health Steering Committee	N. Tangri	6
CDC: "Chronic renal failure" is an immunocompromising condition. Nephrologists Transforming Dialysis Safety Project Committee	K. Bryant	21
Transforming Kidney Health Research Blue-Ribbon Panel research plan	S. Parikh	10
Kidney Care Workforce Solutions ASN Workforce and Training Committee	R. Hoover	24
Diagnostic Excellence: eGFR Toolkit Diagnostic Excellence: eGFR Toolkit Steering Committee	C. Gadegbeku	9
Fostering Inclusivity in Dialysis (FinD) FinD Steering Committee FinD Mentors and Advisers	C. Delgado	 12 30
Glomerular Diseases Collaborative (GD-C) GD-C Steering Committee	K. Gibson	8
Transform transplant and increase access to donor kidneys. ASN Transplant Workgroup	M. Josephson	10
ASN Health Policy Scholar in Residence ASN Health Care Justice Committee ASN Policy and Advocacy Committee ASN Quality Committee	S. Watnick R. Bignall R. Mannon M. Mendu	62
Aligned editorial structure now exists. Editorial boards for *JASN*, *CJASN*, and *Kidney360*	R. Mehrotra C. Rhee M. Allon	48
Kidney Innovation Conference Kidney Health Initiative Board of Directors KidneyCure Grants Review Committee	U. Patel B. Kone A. Pozzi	47
ASN Kidney Week 2024 added new features in 2024. ASN Kidney Week Education Committee	R. Gbadegesin T. Isakova	 34
Total	**23**	**356**

[a]Includes people living with kidney diseases and interns (usually nephrology fellows) who serve on the society's committees, work groups, task forces, and other panels.

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Antinephrin Autoantibodies: Advances in Understanding Podocytopathies

By Benjamin Wooden and Gerald B. Appel

In recent decades, there have been major advances in understanding the clinical course, histopathology, and treatment of the two major podocytopathies: minimal change disease (MCD) and primary focal segmental glomerulosclerosis (FSGS). Despite advancements in understanding the pathogenesis of other glomerulopathies, the mechanisms behind podocytopathies have remained elusive, largely due to unidentified pathogenic circulating immune factors. However, new studies offer hope to unravel these immune factors and revolutionize our approach to podocytopathies.

Autoantibodies targeting nephrin—a component of the podocyte slit diaphragm—have emerged as key in the pathogenesis in both MCD and primary FSGS. Antinephrin antibodies were known to produce the nephrotic syndrome in animal models (1). In 2022, Watts et al. (2) reported that 29% of patients with MCD in several cohorts had detectable circulating antinephrin autoantibodies, which correlated with punctate immunoglobulin G (IgG) in podocytes and colocalized with nephrin on biopsies (also known as "dusting" on immunofluorescence). More recently, a report by Hengel and colleagues (3), published in *The New England Journal of Medicine*, documents that an even larger subset of primary podocytopathies may be driven by autoantibodies against nephrin.

In a multicenter cohort of over 500 patients, Hengel et al. (3) found that these antibodies were present in 44% of 105 adults with MCD and in 52% of 182 children with idiopathic nephrotic syndrome (INS). In the adults with MCD, 69% of those with nephrotic range proteinuria and not receiving immunosuppression were positive, and in the children with INS, 90% of those with nephrotic range proteinuria and not receiving immunosuppression were positive. Only 9% of 74 adults with primary FSGS had antinephrin antibodies, and among controls with other diseases, almost no antinephrin autoantibodies were found. Antinephrin antibodies correlated with disease activity and became negative with clinical remission.

In addition, an experimental murine model showed that the development of antinephrin antibodies led to full nephrotic syndrome, with histologic findings of MCD, IgG localized to the podocyte slit diaphragm, nephrin phosphorylation, and podocyte slit diaphragm alterations. Another recent study found all of the 11 kidney transplant patients with recurrent FSGS to have antinephrin autoantibodies, whereas none of the patients with nonrecurrent FSGS or genetic forms of FSGS did (4).

Together, these findings have important implications for the diagnosis and management of primary podocytopathies. It is conceivable that the use of antinephrin antibody testing will become analogous to that of antiphospholipase A2 receptor antibodies in membranous nephropathy, providing noninvasive diagnostics and enabling detection of "immunologic remission," even before clinical remission occurs (5). Accordingly, future studies need to determine the sensitivity and specificity of antinephrin antibodies and whether their depletion reliably predicts clinical remission. ■

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The authors report no conflicts of interest.

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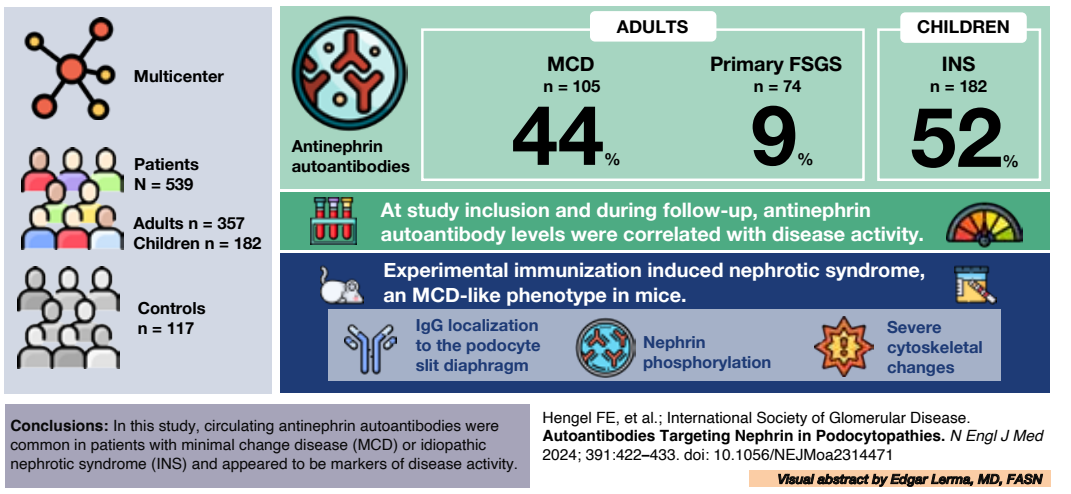
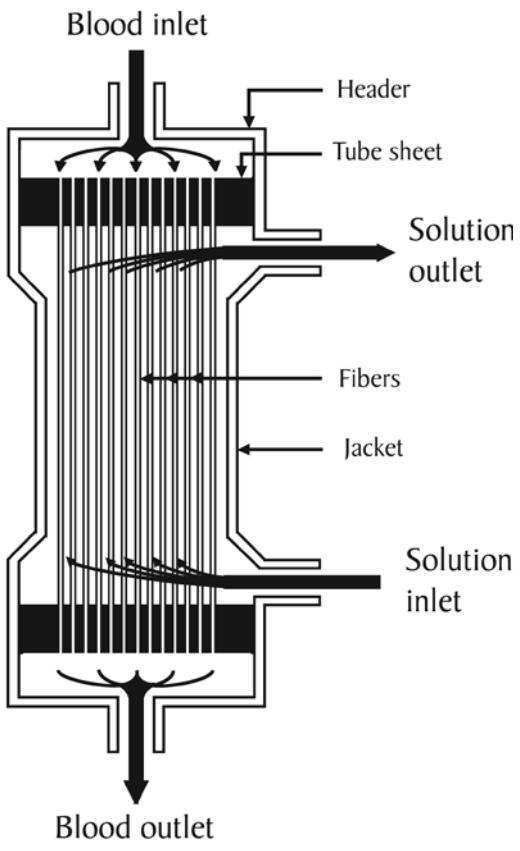


Underutilization of ECT in Acute Intoxication

By Timothy Yung

Extracorporeal therapy (ECT) is a set of modalities that can be used in a variety of settings to tackle issues involving volume, blood pressure, electrolyte imbalance, or any combination of these. In specific instances, the use of ECT, such as hemodialysis, can also be used for its ability to clear poisons. Since the development of the first hollow fiber dialyzer in the 1960s, advances in filter and membrane technology have increased the efficiency and capability of removing endogenous toxins and by extension, exogenous toxins (1). Modern hemodialyzers (Figure), created with synthetic polymers and manufactured with the use of nanotechnology, have increased membrane permeability and allowed for more combined diffusion and convective clearance strategies (2). Despite the infrequency of poisoning and the smaller subset of patients who meet indications for ECT, the role for use remains vital. Data published within the Annual Report of the National Poison Data System demonstrate that ECT constitutes a small fraction of the therapy provided to acute toxin exposures (3, 4). Recognition of clinical scenarios in which use of ECT may be beneficial will increase its utility as a supportive measure for acute poisoning.

Figure. Inside of a hemodialysis dialyzer



Source: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

When prescribing ECT for acute poisoning, several factors must be considered. First, the poisoning must be associated with serious morbidity or mortality. Although various antidotes exist for different toxins, ECT should be considered if these antidotes are unlikely to prevent further deterioration. Additionally, ECT is warranted if the poison continues to cause significant toxicity despite active treatment aimed at minimizing absorption or maximizing elimination (5). Properties of individual toxins also weigh into the clinical decision to pursue ECT. Volume of distribution, molecular weight, and protein binding make certain toxins more amenable to treatment with ECT. If a toxin is more hydrophobic or lipophilic, it will have a larger volume of distribution and be less readily cleared through ECT filtering of plasma volume. Similarly, toxins with larger molecular weight will be physically unable to clear the dialyzer due to size restriction of the membrane. From this perspective, extensive protein binding of toxins will have larger molecular weights when factoring in the molecular weight of blood proteins like albumin; thus, protein-bound toxins are less dialyzable (1, 6).

Lithium is an excellent example that highlights some of the challenges involved in prescribing ECT for poisoning. The EXTRIP (Extracorporeal Treatments in Poisoning) workgroup guidelines recommend initiation of ECT when comorbidities (e.g., arrhythmia, seizure, or coma) are present, and serum levels are elevated (7). Lithium is water-soluble, is not highly protein-bound, and has a low volume of distribution, making it easy to dialyze. However, the volume of distribution does not account for the use of sodium transport channels that slow distribution and contribute to rebound serum levels following cessation of ECT. Furthermore, the toxicity of lithium is not inherently correlated with serum level, which makes clinical practice regarding when to prescribe or stop ECT more difficult (1, 7).

ECT provides a method to rapidly reduce serum concentration and clear toxic levels of many common poisons (6). Use of modalities like hemodialysis or continuous renal replacement therapy (CRRT) has added advantages in restoring physiologic balance and correcting metabolic derangements that frequently occur concomitantly with poisonings. Maximizing the use of extracorporeal filtration and removal involves consideration of time to initiation and the method of ECT used. Hemodialysis is the preferred ECT method in most cases of acute poisoning, having higher efficiency and clearing toxins more rapidly. This efficiency relates to the ability to filter larger volumes of plasma over shorter periods of time as compared with CRRT (1). Its relatively quick set-up time and ready availability in many centers further contribute to its usefulness over other ECT methods such as hemoperfusion and hemofiltration, both which involve the use of solvents.

Despite lower efficiency with CRRT, modalities such as continuous veno-venous hemofiltration and continuous veno-venous hemodialysis have seen increased use over time (1, 5, 6). Easy application within the setting of shock and less hemodynamic effects make CRRT ideal in critical care settings. Additionally, toxins that have large volumes of distribution and are slow to equilibrate between intravascular and extracellular compartments may benefit from more continuous removal. Therapeutic plasmapheresis, another form of ECT, is yet another modality that can be used to tackle toxins that are highly protein-bound (1). Ultimately, as with the decision to pursue ECT, the decision about which modality to use is also dependent on the properties of the toxin (Table).

As technology continues to advance, and the availability of ECT continues to increase, it may be the case that ECT becomes a more feasible first-line alternative to supportive care. Since the inception of modern dialysis, improvements in dialyzer and membrane technology have increased the efficiency of chronic dialysis and consequently efficacy in the context of acute poisoning. As manufacturing techniques become even more refined with the integration of advanced polymers, there exists the potential to dialyze larger molecules or more protein-bound toxins. Increased use will also add to anecdotal experience and expertise in different use cases, generating more evidence for use in various clinical contexts. ■

Table. Toxin properties and amenability of ECT modalities

Toxin	Molecular weight, Da	Water solubility	Protein binding (%)	Volume of distribution (L/kg)	Amenability to HD	Amenability to CRRT
Urea	60	High	Low	Low (~0.6)	High	High
Creatinine	113	High	Low	Low (~0.6)	High	High
Lithium	7	High	Low	Low (~0.5–0.9)	High	High
Methanol	32	High	Low	Low (~0.6)	High	High
Ethylene glycol	62	High	Low	Low (~0.5–0.8)	High	High
Salicylates (aspirin)	180	Moderate	Moderate (~50)	Low (~0.2–0.4)	Moderate	Moderate
Phenobarbital	232	Low	High (~50–60)	Moderate (~0.5–1.0)	Low	Moderate
Digoxin	781	Low	High (~20–30)	High (~6.0–8.0)	Low	Low
Vancomycin	1449	Moderate	Low	Moderate (~0.4–1.0)	Moderate	High
Gentamicin	477	High	Low	Low (~0.2–0.3)	High	High
Theophylline	180	Moderate	Low	Low (~0.3–0.7)	High	High
Cisplatin	300	Low	High (~90)	High (~11.0–12.0)	Low	Low
Carbamazepine	236	Low	High (~70–80)	High (~1.0–2.0)	Low	Moderate

HD, hemodialysis.

Timothy Yung, MD, is a nephrology fellow at the Medical College of Wisconsin, Milwaukee.

The author reports no conflicts of interest.

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INDICATION

XPHOZAH (tenapanor) 30 mg BID is indicated to reduce serum phosphorus in adults with chronic kidney disease (CKD) on dialysis as add-on therapy in patients who have an inadequate response to phosphate binders or who are intolerant of any dose of phosphate binder therapy.

IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS

XPHOZAH is contraindicated in:

- Pediatric patients under 6 years of age
- Patients with known or suspected mechanical gastrointestinal obstruction

WARNINGS AND PRECAUTIONS

Diarrhea

Patients may experience severe diarrhea. Treatment with XPHOZAH should be discontinued in patients who develop severe diarrhea.

MOST COMMON ADVERSE REACTIONS

Diarrhea, which occurred in 43-53% of patients, was the only adverse reaction reported in at least 5% of XPHOZAH-treated patients with CKD on dialysis across trials. The majority of diarrhea events in XPHOZAH-treated patients were reported to be mild-to-moderate in severity and resolved over time, or with dose reduction. Diarrhea was typically reported soon after initiation but could occur at any time during treatment with XPHOZAH. Severe diarrhea was reported in 5% of XPHOZAH-treated patients in these trials.

Please see Brief Summary of full Prescribing Information on the following page.

Reference: XPHOZAH® (tenapanor) full Prescribing Information. Waltham, MA: Ardelyx, Inc.; 2023.

ardelyx®

XPHOZAH (tenapanor) tablets, for oral use
Brief Summary of Prescribing Information

1 INDICATIONS AND USAGE

XPHOZAH is indicated to reduce serum phosphorus in adults with chronic kidney disease (CKD) on dialysis as add-on therapy in patients who have an inadequate response to phosphate binders or who are intolerant of any dose of phosphate binder therapy.

4 CONTRAINDICATIONS

XPHOZAH is contraindicated in patients under 6 years of age because of the risk of diarrhea and serious dehydration *[see Warnings and Precautions (5.1), Use in Specific Populations (8.5)]*.

XPHOZAH is contraindicated in patients with known or suspected mechanical gastrointestinal obstruction.

5 WARNINGS AND PRECAUTIONS

5.1 Diarrhea

Diarrhea was the most common adverse reaction in XPHOZAH-treated patients with CKD on dialysis *[see Dosage and Administration (2) in the full Prescribing Information, Contraindications (4) and Adverse Reactions (6.1)]*. In clinical trials, diarrhea was reported in up to 53% of patients, reported as severe in 5%, and associated with dehydration and hyponatremia in less than 1% of patients. Treatment with XPHOZAH should be discontinued in patients who develop severe diarrhea.

6 ADVERSE REACTIONS

6.1 Clinical Trial Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared with rates in the clinical trials of another drug and may not reflect the rates observed in practice.

The safety data described below reflect data from 754 adults with CKD on dialysis taking XPHOZAH in clinical trials as monotherapy and in combination with phosphate binders. Among the 754 patients, 258 patients were exposed to tenapanor for at least 26 weeks and 75 were exposed to tenapanor for at least one year. *[see Clinical Studies (14) in the full Prescribing Information]*.

Most Common Adverse Reaction

Diarrhea, which occurred in 43-53% of patients, was the only adverse reaction reported in at least 5% of XPHOZAH-treated patients with CKD on dialysis across trials. The majority of diarrhea events in the XPHOZAH-treated patients were reported to be mild-to-moderate in severity and resolved over time, or with dose reduction. Diarrhea was typically reported soon after initiation but could occur at any time during treatment with XPHOZAH. Severe diarrhea was reported in 5% of XPHOZAH-treated patients in these trials *[see Warnings and Precautions (5.1)]*.

7 DRUG INTERACTIONS

7.1 OATP2B1 Substrates

Tenapanor is an inhibitor of intestinal uptake transporter, OATP2B1 *[see Clinical Pharmacology (12.3) in the full Prescribing Information]*. Drugs which are substrates of OATP2B1 may have reduced exposures when concomitantly taken with XPHOZAH. Monitor for signs related to loss of efficacy and adjust the dose of concomitantly administered drug as needed.

Enalapril is a substrate of OATP2B1. When enalapril was coadministered with XPHOZAH (30 mg twice daily for five days), the peak exposure (Cmax) of enalapril and its active metabolite, enalaprilat, decreased by approximately 70% and total systemic exposures (AUC) decreased by 50 to 65% compared to when enalapril was administered alone *[see Clinical Pharmacology (12.3) in the full Prescribing Information]*. However, the decrease in enalaprilat's exposure with XPHOZAH may be offset by the inherently higher exposures observed in patients with CKD on dialysis due to its reduced renal clearance. Therefore, a lower starting dose of enalapril, which is otherwise recommended in patients with CKD on dialysis is not required when enalapril is coadministered with XPHOZAH.

7.2 Sodium Polystyrene Sulfonate

Separate administration XPHOZAH and sodium polystyrene sulfonate (SPS) by at least 3 hours. SPS binds to many commonly prescribed oral medicines.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

Tenapanor is essentially non-absorbed systemically, with plasma concentrations below the limit of quantification (less than 0.5 ng/mL) following oral administration *[see Clinical Pharmacology (12.3) in the full Prescribing Information]*. Therefore, maternal use is not expected to result in fetal exposure to the drug.

The available data on XPHOZAH exposure from a small number of pregnant women have not identified any drug associated risk for major birth defects, miscarriage, or adverse maternal or fetal outcomes. In reproduction studies with tenapanor in pregnant rats and rabbits, no adverse fetal effects were observed in rats at 0.2 times the maximum recommended human dose and in rabbits at doses up to 15 times the maximum recommended human dose (based on body surface area) *[see Nonclinical Toxicology (13.1) in the full Prescribing Information]*.

The estimated background risk of major birth defects and miscarriage for women with CKD on dialysis with hyperphosphatemia is unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the United States general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2% to 4% and 15% to 20%, respectively.

Animal Data

In an embryofetal development study in rats, tenapanor was administered orally to pregnant rats during the period of organogenesis at dose levels of 1, 10 and 30 mg/kg/day. Tenapanor doses of 10 and 30 mg/kg/day were not tolerated by the pregnant rats and was associated with mortality and moribundity with body weight loss. The 10 and 30 mg/kg dose group animals were sacrificed early, and the fetuses were not examined for intrauterine parameters and fetal morphology. No adverse fetal effects were observed in rats at 1 mg/kg/day (approximately 0.2 times the maximum recommended human dose) and in rabbits at doses up to 45 mg/kg/day (approximately 15 times the maximum recommended human dose, based on body surface area). In a pre- and post-natal developmental study in mice, tenapanor at doses up to 200 mg/kg/day (approximately 16.5 times the maximum recommended human dose, based on body surface area) had no effect on pre- and post-natal development.

8.2 Lactation

Risk Summary

There are no data available on the presence of tenapanor in either human or animal milk, its effects on milk production or its effects on the breastfed infant. Tenapanor is essentially non-absorbed systemically, with plasma concentrations below the limit of quantification (less than 0.5 ng/mL) following oral administration *[see Clinical Pharmacology (12.3) in the full Prescribing Information]*. The minimal systemic absorption of tenapanor will not result in a clinically relevant exposure to breastfed infants. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for XPHOZAH and any potential adverse effects on the breastfed infant from XPHOZAH or from the underlying maternal condition.

8.4 Pediatric Use

Risk Summary

XPHOZAH is contraindicated in patients less than 6 years of age. In nonclinical studies, deaths occurred in young juvenile rats (less than 1-week old rats; approximate human age-equivalent of less than 2 years of age) and in older juvenile rats (approximate human age-equivalent of 2 years of age) following oral administration of tenapanor, as described below in Juvenile Animal Toxicity Data.

The safety and effectiveness of XPHOZAH in pediatric patients have not been established.

Juvenile Animal Toxicity Data

In a 21-day oral dose range finding toxicity study in juvenile rats, tenapanor was administered to neonatal rats (post-natal day (PND) 5) at doses of 5 and 10 mg/kg/day. Tenapanor was not tolerated in male and female pups and the study was terminated on PND 16 due to mortalities and decreased body weight (24% to 29% reduction in females at the respective dose groups and 33% reduction in males in the 10 mg/kg/day group, compared to control).

In a second dose range finding study, tenapanor doses of 0.1, 0.5, 2.5, or 5 mg/kg/day were administered to neonatal rats from PND 5 through PND 24. Treatment-related mortalities were observed at 0.5, 2.5, and 5 mg/kg/day doses. These premature deaths were observed as early as PND 8, with majority of deaths occurring between PND 15 and 25. In the 5 mg/kg/day group, mean body weights were 47% lower for males on PND 23 and 35% lower for females on PND 22 when compared to the controls. Slightly lower mean tibial lengths (5% to 11%) were noted in males and females in the 0.5, 2.5, and 5 mg/kg/day dose groups on PND 25 and correlated with the decrements in body weight noted in these groups. Lower spleen, thymus, and/or ovarian weights were noted at the 0.5, 2.5, and 5 mg/kg/day doses. Tenapanor-related gastrointestinal distension and microscopic bone findings of increased osteoclasts, eroded bone, and/or decreased bone in sternum and/or femorotibial joint were noted in males and females in the 0.5, 2.5, and 5 mg/kg/day dose groups.

In juvenile rats administered tenapanor at 0.03, 0.1, or 0.3 mg/kg/day on PND 5 through PND 61, treatment-related mortalities were observed at 0.3 mg/kg/day. Lower mean body weight gains were noted in the 0.3 mg/kg/day group males and females compared to the control group primarily during PND 12–24 but continuing sporadically during the remainder of the dosing period; corresponding lower mean food consumption was noted in this group during PND 21–33. As a result, mean body weights were up to 15.8% and 16.8% lower in males and females, respectively, compared to the control group; the greatest difference was on PND 24 for males and PND 21 for females. Mean body weight in the 0.3 mg/kg/day group males was only 3.9% lower than the control group on PND 61. There were no tenapanor-related effects on mean body weights, body weight gains, or food consumption in the 0.03 and 0.1 mg/kg/day group males and females. A dosage level of 0.1 mg/kg/day was considered to be the no-observed-adverse-effect level (NOAEL) for juvenile toxicity of tenapanor *[see Contraindications (4), Warnings and Precautions (5.1)]*.

In a 21-day oral dose range finding study in older (weaned) juvenile rats administered tenapanor at 0.1, 1, or 5 mg/kg/day on PND 21 through PND 41 (approximate human age-equivalent of 2 to 12 years of age), treatment-related mortalities or moribundities were observed during the first two days of the study in the 1 mg/kg/day males and the 5 mg/kg/day males and females. Watery feces, decreased food consumption, and lower mean body weight were also observed in the 1 and 5 mg/kg/day groups.

In weaned juvenile rats administered tenapanor at 0.1, 0.3, and 0.7 (males) or 1 (females) mg/kg/day on PND 21 through PND 80, no mortalities were observed. Significant decreases in mean body weights were observed in the 0.3 and 0.7 mg/kg/day males throughout the dosing period (up to 20.3% lower than control) and in the 1 mg/kg/day females between PND 23 to 35 (up to 16.7% lower than control), with food consumption notably decreased on PND 21 to 29. There were also reductions in tibia length between PND 76 and 80 in the 0.3 and 0.7 mg/kg/day males, and between PND 36 and 64 in the 0.7 mg/kg/day males, which were not observed during the 14-day recovery period. The NOAEL was considered to be 0.1 mg/kg/day for juvenile toxicity of tenapanor.

8.5 Geriatric Use

Of 1010 adult patients with CKD on dialysis randomized and treated in two randomized, double-blind, placebo-controlled randomized withdrawal clinical trials for XPHOZAH (TEN-02-201 and TEN-02-301) as well as a third randomized, double-blind, placebo-controlled trial (TEN-02-202) for XPHOZAH in combination with phosphate binders, 282 (28%) were 65 years of age and older. Clinical studies of XPHOZAH did not include sufficient numbers of patients aged 65 and older to determine whether they respond differently than younger patients.

10 OVERDOSAGE

No data are available regarding overdosage of XPHOZAH in patients. Based on nonclinical data, overdose of XPHOZAH may result in gastrointestinal adverse effects such as diarrhea, as a result of exaggerated pharmacology with a risk for dehydration if diarrhea is severe or prolonged *[see Warnings and Precautions (5.1)]*.

17 PATIENT COUNSELING INFORMATION

Advise Patients:

Diarrhea

Instruct patients to contact their healthcare provider if they experience severe diarrhea *[see Warnings and Precautions (5.1)]*.

- Instruct patients not to use stool softeners or laxatives with XPHOZAH.

Administration and Handling Instructions

Instruct Patients:

- To take XPHOZAH just prior to the first and last meals of the day *[see Dosage and Administration (2.2) in the full Prescribing Information]*.
- Patients should be counseled not to take XPHOZAH right before a hemodialysis session, and to take XPHOZAH right before the next meal, as some patients may experience diarrhea after taking XPHOZAH.
- If a dose is missed, take the dose just before the next meal. Do not take 2 doses at the same time *[see Dosage and Administration (2.2) in the full Prescribing Information]*.
- To keep XPHOZAH in a dry place. Protect from moisture. Keep in the original bottle. Do not remove desiccant from the bottle. Keep bottles tightly closed *[see How Supplied/Storage and Handling (16) in the full Prescribing Information]*.

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- Complimentary Refreshment Breaks
- Exhibitor Spotlights
- Fellows-in-Training (FIT) Bowl
- Headshot Lounge
- 3,000+ Posters with Top Trainee Poster Sessions
- Attendee Lounges
- Welcome Reception
- Wi-Fi Service

Welcome Reception

Thursday, October 24, 6:00–7:00 p.m.
ASN welcomes you to San Diego with a reception in the exhibit hall.
Support provided by Calliditas Therapeutics.

ASN Communities Lounge – Booth 1529; Aisles 1500 and 1600

A focal point of your exhibit hall experience, visit the lounge to learn more about ASN Communities, meet kidney leaders, network with peers, and unwind at the relaxation zone.

FIT Bowl

Which nephrology training team will reign supreme? Stop by and watch teams test their knowledge against their peers. The FIT Bowl is a two-day, single elimination tournament held in the ASN Futures Theater in the exhibit hall.
Seating is limited.

Thursday, October 24	**10:30 a.m.–12:30 p.m.**	**Elimination Rounds**
Friday, October 25	**10:30 a.m.–11:30 a.m.**	**Semi-Finals**
	11:30 a.m.–12:30 p.m.	**Finals**

Support provided by IgANexus.

Headshot Lounge

Stop by booth 727 Thursday through Saturday from 9:30 a.m.–2:30 p.m. for a complimentary professional headshot.
Support provided by Calliditas Therapeutics.

Exhibitor Spotlight Schedule

Join your colleagues for the latest advances in nephrology practices, products, services, and technologies presented in two theaters on the exhibit hall floor (no continuing education credit). Seating is limited and available on a first-come, first-served basis.

The 10:00 a.m. presentations include breakfast. All other presentations include lunch.

Thursday, October 24

10:00–10:45 a.m. — **Theater 1**

Helping Patients Go Home Successfully: The Home Dialysis Spotlight!
Presented by Baxter

11:00–11:45 a.m. — **Theater 2**

Jardiance® (empagliflozin) tablets: Expert Perspectives on Clinical Trial Data and Approved Indications
Presented by Boehringer Ingelheim | Lilly

12:00–12:45 p.m. — **Theater 1**

A Stronger Foundation: Perspectives on an IgA Nephropathy Therapy with an Updated Indication
Presented by TRAVERE THERAPEUTICS

1:00–1:45 p.m. — **Theater 2**

Discover an Innovative Mechanism of Action: An Oral, Once-Daily HIF-PHI
Presented by Akebia Therapeutics

Friday, October 25

10:00–10:45 a.m. — **Theater 1**

Navigating IgA Nephropathy: Pathogenesis, the Role of APRIL, and Patient Case Study
Presented by Otsuka

11:00–11:45 a.m. — **Theater 2**

A Different Perspective on Hyperphosphatemia Management: Evaluating Current Strategies
Presented by ardelyx

12:00–12:45 p.m. — **Theater 1**

Get to Know FABHALTA® (iptacopan)
Presented by NOVARTIS

1:00–1:45 p.m. — **Theater 2**

Early Intervention in IgA Nephropathy: An Update on a Potential Disease-Modifying Therapy
Presented by calliditas THERAPEUTICS

Saturday, October 26

10:00–10:45 a.m. — **Theater 1**

Transitions of Care in Kidney Transplant with ENVARSUS XR®
Presented by Veloxis PHARMACEUTICALS

11:00–11:45 a.m. — **Theater 2**

Effective Strategies with HIF PH Inhibitors: Insights from Real-World Evidence
Presented by zydus Dedicated To Life

12:00–12:45 p.m. — **Theater 1**

Rare Disease Across the Lifespan: Helping Patients with Cystinosis Thrive into Adulthood
Presented by AMGEN

1:00–1:45 p.m. — **Theater 2**

Potential Approaches to Addressing the Autoimmunity in Autoimmune Glomerulonephritis
Presented by VERTEX | ALPINE ImmuneSciences

New Pharmacologic Therapies for Hypertension on the Horizon

By George Thomas

Almost half of adults in the United States have a diagnosis of hypertension, with declining blood pressure control rates that lead to increased morbidity and mortality (1). A recent flurry of research into novel agents with unique mechanisms to manage hypertension bears promise for better blood pressure control, particularly in the subset of patients with resistant hypertension, summarized in the Table.

Of these, aprocitentan—a dual endothelin receptor antagonist—is approved by the US Food and Drug Administration (FDA) for treatment of resistant hypertension. Unlike other agents studied for systemic hypertension (e.g., bosentan and darusentan), aprocitentan appears to have a better safety profile. A phase 3 trial showed modest but consistent blood pressure reduction in patients with resistant hypertension, with a secondary outcome of albuminuria reduction by –21% and –38%, with 12.5 mg and 25 mg doses, respectively (2). Edema was a concern, particularly with the higher 25-mg dose. The FDA-approved dose is 12.5 mg once daily in patients with an estimated glomerular filtration rate (eGFR) of 15 or greater. Aprocitentan prescribers need to be enrolled in a Risk Evaluation and Mitigation Strategy safety program due to risk of major birth defects.

An increasing recognition of autonomous aldosterone production as a contributor to hypertension has prompted evaluation of agents beyond steroidal mineralocorticoid receptor antagonists (MRAs), like spironolactone. Although the non-steroidal MRA (ns-MRA) finerenone is the most recognizable in this class and has proven cardiorenal benefits in type 2 diabetes, its effect on blood pressure reduction has been more limited (although data suggest more potent effects at a systolic blood pressure >140 mm Hg). It should be noted that the randomized controlled trials examining kidney outcomes for

Table. Summary and status of novel pharmacologic agents undergoing research to manage hypertension

Agent	Class	Trials	Patients studied	Results: BP difference from placebo, mm Hg	Concerns/side effects	Status
Aprocitentan	Dual ERA	PRECISION phase 3 trial	Resistant hypertension, GFR ≥15	• Office SBP at 4 weeks: – 3.8 (12.5 mg) – 3.7 (25.0 mg) • ABPM 24-hour SBP at 4 weeks: – 4.2 (12.5 mg) • Greater BP reduction with older age (≥75 years), CKD, and albuminuria • Withdrawal phase of study—consistent BP reduction compared with placebo	• REMS safety program—negative pregnancy test required in females of childbearing age • Edema • Reversible decrease in hemoglobin • Not recommended in hepatic dysfunction, NYHA stages III–IV, or elevated NTproBNP ≥500 pg/mL	FDA approved, March 2024
Ocedurenone	ns-MRA	BLOCK-CKD phase 2 trial	Uncontrolled hypertension, CKD stages 3b and 4	Office SBP at 84 days: • 7.0 (0.25 mg) • 10.2 (0.50 mg)	Monitor for hyperkalemia	CLARION-CKD phase 3 trial in CKD stages 3b and 4 and uncontrolled hypertension
Baxdrostat	ASI	BrigHTN phase 2 trial	Resistant hypertension, GFR ≥45	Office SBP at 12 weeks: • 8.1 (1 mg) • 11.0 (2 mg)	Monitor for hyperkalemia, cortisol insufficiency	Bax24 phase 3 trial in resistant hypertension
Lorundrostat	ASI	TARGET-HTN phase 2 trial	Uncontrolled hypertension, GFR ≥60	Office SBP at 8 weeks: • 7.8 (100 mg)	Monitor for hyperkalemia, cortisol insufficiency	LAUNCH-HTN phase 3 trial in uncontrolled and resistant hypertension
Zilebesiran	siRNA targeting angiotensinogen	KARDIA-2 phase 2 trial	Uncontrolled hypertension, background of indapamide, amlodipine, or olmesartan	ABPM 24-hour SBP at 3 months: • 12.1 (indapamide) • 9.7 (amlodipine) • 4.0 (olmesartan)	Monitor for hypotension	KARDIA-3 phase 3 trial in uncontrolled hypertension and high cardiovascular risk including CKD

ABPM, ambulatory blood pressure monitoring; ASI, aldosterone synthase inhibitor; Bax24, A Study to Investigate the Effect of Baxdrostat on Ambulatory Blood Pressure in Participants With Resistant Hypertension; BLOCK-CKD, phase 2b Study of KBP-5074 in Subjects With Uncontrolled Hypertension and Advanced Chronic Kidney Disease; BP, blood pressure; BrigHTN, A Study of CIN-107 in Adults With Treatment-Resistant Hypertension (rHTN); CLARION-CKD, Efficacy and Safety of KBP-5074 in Uncontrolled Hypertension and Moderate or Severe Chronic Kidney Disease (CKD); ERA, endothelin receptor antagonist; KARDIA-3, Zilebesiran as Add-on Therapy in Patients With High Cardiovascular Risk and Hypertension Not Adequately Controlled by Standard of Care Antihypertensive Medications; LAUNCH-HTN, Efficacy and Safety of Lorundrostat in Subjects With Uncontrolled and Resistant Hypertension; NTproBNP, N-terminal prohormone of brain natriuretic peptide; NYHA, New York Heart Association; PRECISION, A Research Study to Show the Effect of Aprocitentan in the Treatment of Difficult to Control (Resistant) High Blood Pressure (Hypertension) and Find Out More About Its Safety; REMS, Risk Evaluation and Mitigation Strategy; SBP, systolic BP; TARGET-HTN, Trial on the Safety and Efficacy of MLS-101 in Patients With Uncontrolled Hypertension.

finerenone (FIDELIO-DKD [Efficacy and Safety of Finerenone in Subjects With Type 2 Diabetes Mellitus and Diabetic Kidney Disease] and FIGARO-DKD [Efficacy and Safety of Finerenone in Subjects With Type 2 Diabetes Mellitus and the Clinical Diagnosis of Diabetic Kidney Disease]) were not blood pressure studies but were cardiovascular and kidney outcome studies (3, 4). Ocedurenone is a third-generation ns-MRA, with a small phase 2 trial showing significant blood pressure reduction compared with placebo in patients with uncontrolled hypertension and chronic kidney disease (CKD) stages 3b and 4, with no reported cases of severe hyperkalemia (≥6 mmol/L) (5).

Distinct from ns-MRAs, which block aldosterone binding to MRs, aldosterone synthase inhibitors prevent adrenal aldosterone synthesis by inhibiting the CYP11B2 isoenzyme. The challenge is to specifically inhibit CYP11B2 without inhibiting CYP11B1, which is involved in cortisol production. Osilodrostat, which was the first aldosterone synthase inhibitor in clinical development, inhibited cortisol synthesis and was repurposed to treat hypercortisolism (6). Newer aldosterone synthase inhibitors have more selectivity for CYP11B2, and agents currently under study include baxdrostat and lorundrostat for hypertension, dexfadrostat for primary aldosteronism, and BI 690517 for CKD and albuminuria. Phase 2 trials of baxdrostat in resistant hypertension (7) and lorundrostat in uncontrolled hypertension (8) showed significant blood pressure differences compared with placebo. Both trials showed lower serum aldosterone and an increase in plasma renin activity, with no cortisol insufficiency. Although the incidence of severe hyperkalemia (≥6 mmol/L) was low, it should be noted that these trials were of short duration, and the mean eGFR in these trials was 80–83; the real-world incidence of hyperkalemia could be higher, particularly in those with lower eGFR.

Hepatic angiotensinogen attenuators are one of the more intriguing innovative therapies, which include small interfering RNA (siRNA) targeting hepatic angiotensinogen and antisense oligonucleotide for hepatic angiotensinogen mRNA knockdown. Zilebesiran is a first-in-class siRNA that binds to an hepatic asialoglycoprotein receptor leading to reduction in angiotensinogen mRNA and decreased hepatic angiotensinogen. The phase 2 KARDIA-1 (A Study to Evaluate Efficacy and Safety of ALN-AGT01 in Patients With Mild-to-Moderate Hypertension) study showed blood pressure reduction at 300 mg doses and higher, when injected subcutaneously with dosing schedules of once every 3 months or once every 6 months (approximately a 14-mm Hg blood pressure difference from placebo) and sustained angiotensinogen and blood pressure reduction at 6 months (9). The phase 2 KARDIA-2 (Zilebesiran as Add-on Therapy in Patients With Hypertension Not Adequately Controlled by a Standard of Care Antihypertensive Medication) study evaluated zilebesiran 600 mg vs placebo on background of olmesartan, indapamide, or amlodipine (10). Interestingly, blood pressure reduction was more pronounced in the indapamide and amlodipine groups at 3 months and sustained to 6 months in these groups but not in the olmesartan group. Although the potential for improved adherence with a once-every-6-month dosing schedule is appealing, several questions remain, including concern for refractory hypotension, particularly in states like shock. No severe adverse events of hypotension or orthostasis were noted in studies, but these were of relatively short follow-up, and longer-term safety data are needed.

In addition to pharmacologic agents, device-based therapy, in particular, renal denervation, has emerged as a possible adjunctive option and now has FDA approval for clinical use, with newer catheter designs and more recent studies showing modest but significant blood pressure reductions.

Overall, this is an exciting era for antihypertensive therapy. Although continued development and research into newer therapies are essential to advance the field, it is also critical to remember the basic paradigms of management, including proper blood pressure measurement and continued emphasis on lifestyle modifications. The availability of newer therapies also brings the challenge of access, and effective ways of implementation need to be examined. The exact place of these newer therapies in the armamentarium remains to be determined in future studies and with clinical use, particularly in comparison with older, inexpensive agents like spironolactone. ■

George Thomas, MD, FASN, is an associate professor of medicine at the Cleveland Clinic Lerner College of Medicine and director of the American Heart Association Comprehensive Hypertension Center in the Department of Kidney Medicine, Cleveland Clinic, Cleveland, OH.

The author reports no conflicts of interest.

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Educational Symposia Schedule

KIDNEY WEEK 2024

Marriott Marquis San Diego Marina

Thursday, October 24–Saturday, October 26

Breakfast or lunch will be served at each symposium.

Seating is limited and available on a first-come, first-served basis to fully paid Annual Meeting participants. Doors open approximately 15 minutes prior to each symposium.

Continuing Education Credit
These live activities are eligible for continuing education credit. Please visit www.asn-online.org/KidneyWeek for more information.

Thursday, October 24 12:45–1:45 p.m.

Accepting the Complement: Understanding the Role of Complement and Its Targeted Therapies in IgA Nephropathy
*Support is provided by an educational grant from **Novartis Pharmaceuticals Corporation.***

Hyperkalemia, CKD, and Heart Failure: Dietary and Medical Considerations
*Support is provided by an educational grant from **AstraZeneca**.*

Imaging and Multidisciplinary Care in ADPKD
*Support is provided by an educational grant from **Otsuka America Pharmaceutical, Inc**.*

Mechanisms and Advances in ANCA-Associated Vasculitis
*This activity is supported by educational funding provided by **Amgen**.*

Friday, October 25 6:45–7:45 a.m.

Advances in Inflammation Mechanisms and Therapies to Reduce Atherosclerotic Cardiovascular Disease in Persons with Kidney Diseases
*Support is provided by an educational grant from **Novo Nordisk**.*

Ushering Renal Denervation from Trials into the Real World
*Support is provided by an educational grant from **Medtronic**.*

Friday, October 25 12:45–1:45 p.m.

Expanding Therapeutic Landscape in Lupus Nephritis: Utilization of B Cell Targeted Therapy
*Support is provided by an educational grant from **Genentech, a member of the Roche Group**.*

Targeting the Pathophysiology of Hyperphosphatemia in ESKD: From Physiology to Clinical Innovation
*Support is provided by an educational grant from **Ardelyx, Inc**.*

The Role of Hemodiafiltration in the Management of Dialysis-Dependent Chronic Kidney Failure
*Support is provided by an educational grant from **Fresenius Medical Care Renal Therapies Group LLC**.*

Treatment of Anemia of CKD: What Is the Role of Hypoxia-Inducible Factor Prolyl Hydroxylase Inhibitors?
*Support is provided by an educational grant from **Akebia Therapeutics, Inc.***

Saturday, October 26 12:45–1:45 p.m.

Acute Kidney Dysfunction in Cirrhosis: A Case-Based Conversation about Diagnosis and Treatment
*Support is provided by an educational grant from **Mallinckrodt Pharmaceuticals**.*

Glucagon-Like Peptide-1 Receptor Agonists and Diabetic Kidney Disease
*Support is provided by an educational grant from **Novo Nordisk**.*

Targeting Cytokines in IgA Nephropathy: Understanding and Taking Aim at the “First-Hit”
*Support is provided by an educational grant from **Vera Therapeutics**.*

All symposia will be recorded and available in the ASN eLearning Center for up to three years starting in late November; continuing education credits will not be awarded for the online content.

PLENARY SESSION

STATE-OF-THE-ART LECTURE

Plenary to Focus on Artificial Intelligence in Nephrology

David C. Rhew, MD

A physician and computer scientist with unique experience and expertise will speak on "Accelerating Health Care Innovation in Nephrology With Artificial Intelligence" at a plenary session on Thursday, October 24.

The speaker will be David C. Rhew, MD, Microsoft's global chief medical officer and vice president of health care. He is also adjunct professor at Stanford University; holds six US technology patents that enable authoring, mapping, and integrating clinical decision support into electronic health records; and has been recognized as 1 of the 50 most influential clinician executives by Modern Healthcare.

He served as Microsoft's international coordinator for the pandemic response, in which he worked with the World Health Organization to develop its World Health Data Hub, with the US Centers for Disease Control and Prevention to establish its vaccine data program, and with states to roll out COVID-19 vaccines.

Dr. Rhew says that his goal at Microsoft "is to demonstrate how a large-scale data-driven approach will lead to smarter decision-making, more proactive care, and improved health outcomes and lower costs for patients and populations." His team is pursuing this goal by focusing on health care data interoperability, involving traditional data sets (e.g., electronic health records and claims) along with newer ones (e.g., biosensors, patient-reported outcomes, and social determinants of care), and the maturation of a health care cloud-based platform that is secure, scalable, and intelligent.

Prior to joining Microsoft in 2019, he served as chief medical officer and vice president at Samsung as well as senior vice president and chief medical officer at Zynx Health. He also served on the National Quality Forum's executive committee for consensus standards and approval and chaired the Consumer Technology Association's Health Technology Board. He currently sits on the Governing Committee of the National Evaluation System for Health Technology Coordinating Center.

Dr. Rhew received bachelor's degrees in computer science and cellular molecular biology from the University of Michigan. He received an MD from Northwestern University and completed his internal medicine residency at Cedars-Sinai Medical Center in Los Angeles, CA. He completed fellowships in health services research at Cedars-Sinai and infectious diseases at the University of California, Los Angeles.

Basketball Star and Transplant Recipient to Receive President's Medal

Alonzo Mourning

Hall-of-Fame basketball player and kidney transplant recipient Alonzo Mourning will receive an ASN President's Medal during the opening plenary on Thursday, October 24, in recognition of his work in raising awareness about kidney diseases, as well as raising funds for kidney research and social justice programs. ASN presents this medal to individuals who have advanced the association's mission to fight against kidney diseases by educating health professionals, sharing new knowledge, advancing research, and advocating for patients.

Mr. Mourning was enjoying a stellar basketball career—having won a gold medal as part of the US team at the 2000 Summer Olympics in Sydney, Australia—when he noticed extreme swelling in his legs and feet accompanied by uncharacteristic fatigue. He was soon diagnosed with focal segmental glomerulosclerosis (FSGS), and genetic testing revealed that he carries the APOL1 gene variant. He missed the entire 2002–2003 National Basketball Association (NBA) season due to his kidney disease. A relative donated a kidney for a successful transplant in 2003. He not only returned to play the next year, but in 2006, he won an NBA championship as a key player for the Miami Heat.

Mr. Mourning was an active philanthropist prior to his illness. He cofounded the Mourning Family Foundation in 1997 to focus on youth development through advocacy, education, and enrichment. Since then, he has been instrumental in raising more than $50 million for various programs, including building a state-of-the-art youth center aimed at bridging gaps and strengthening communities in the urban core in the South Florida area of Overtown.

After being diagnosed with FSGS, he also turned his attention to raising money and awareness as a kidney health advocate. He began with a campaign using his nickname, establishing Zo's Fund for Life, which raised $2 million for FSGS, providing research into treatment, education, and financial relief for patients who cannot afford medication. He continues to raise awareness about the importance of becoming an organ donor. He works with the National Kidney Foundation to educate the public on the importance of early detection of kidney diseases.

Mr. Mourning has received numerous honors, including the NBA Community Assist Award, the National Urban League Outstanding Community Service Award, the National Conference for Community and Justice Silver Medallion Community Service Award, the NBA J. Walter Kennedy Citizenship Award, the Children's Trust Excellence Award for Public Policy, and the Florida Blue Foundation Sapphire Award. He was inducted into the Naismith Memorial Basketball Hall of Fame in 2014 after a multiyear career with the Charlotte Hornets, Miami Heat, and New Jersey Nets.

Mr. Mourning graduated from Georgetown University with a degree in sociology. He is a member of the President's Council on Sports, Fitness and Nutrition; a member of his alma mater's board of directors; and vice president of player programs and development for the Miami Heat.

Schrier Lectureship Looks at Electrolyte Disorders

Paul A. Welling, MD

An internationally recognized electrolytes authority will discuss "Molecular Bases of Potassium and Sodium Balance, Electrolyte Disorders, and Hypertension" in the Robert W. Schrier, MD, Endowed Lectureship on Thursday, October 24.

Paul A. Welling, MD, holds the Joseph S. and Ester Handler Endowed Professorship of Laboratory Research in Nephrology at Johns Hopkins School of Medicine. He also serves as codirector of the Maryland Polycystic Kidney Disease Research and Translation Core Center and codirector of the Biomedical Resource Core of the Johns Hopkins O'Brien Center to Advance Kidney Health Equity.

A major focus of Dr. Welling's research is on developing a mechanistic understanding of how potassium in the diet affects blood pressure as well as cardiovascular and kidney health. His laboratory is recognized for elucidating how ion transport molecules in the kidney control salt balance and discovering how these molecules go awry in disorders of electrolyte homeostasis and blood pressure.

In recent years, he has focused on developing a molecular understanding of the relationship between sodium and potassium in the diet, inherited susceptibilities of hypertension, and kidney diseases. His group uses a multidisciplinary approach, combining modern methods of molecular genetics, genomics, cellular biology, and biochemistry with state-of-the-art physiologic and pathophysiologic phenotyping techniques to translate genetic discoveries to mechanistic understandings of electrolyte transport physiology, hypertension, and kidney diseases.

Dr. Welling currently serves as principal investigator on three research projects funded by the National Institutes of Health and is the North American coordinator and principal investigator on the Leducq Foundation Networks of Excellence on Potassium in Hypertension.

He has held leadership and scientific advisory roles with ASN, the National Institutes of Health, and the American Physiological Society. He has served on the editorial boards of the *American Journal of Physiology–Renal Physiology, The Journal of Physiological Sciences*, the *Journal of Biological Chemistry*, and the *Annual Review of Physiology*. Dr. Welling has received many honors, including the Steven Hebert Award and the Carl W. Gottschalk Distinguished Lectureship from the American Physiological Society and the Donald Seldin Lecture from the American Heart Association.

He received his medical degree from The University of Kansas and completed his postdoctoral training at Yale School of Medicine. He worked for many years at the University of Maryland School of Medicine, where he rose to the rank of full professor and director of the Maryland Center for Kidney Discovery. He joined Johns Hopkins in 2019.

Lecture to Focus on Fast-Changing World of Organ Donation

Jesse D. Schold, MEd, MS, PhD

"Navigating an Evolving Organ Procurement Landscape" is the title of the Christopher R. Blagg, MD, Endowed Lectureship in Kidney Diseases and Public Policy on Thursday, October 24.

The speaker will be Jesse D. Schold, MEd, MS, PhD, a visiting professor of surgery and epidemiology at the University of Colorado Anschutz Medical Campus in Denver, where he is also the director of the Center for Outcomes Research and Policy and associate vice chair of policy and outcomes for the Department of Surgery.

Dr. Schold's research interests include large database analyses, quality metrics for health care practitioners, health services research, disparities in health care, and statistical and epidemiologic methods. He is particularly focused on research that promotes access to care for patients with organ failure and use of empirical evidence to improve care delivery and health care policy.

Dr. Schold has been a coinvestigator on multiple studies by the National Institutes of Health, the Health Resources & Services Administration, and the Centers for Disease Control and Prevention. He has given over 200 invited national and international lectures and has authored over 360 peer-reviewed publications primarily in the fields of kidney diseases and organ transplantation.

A former board member of the American Society of Transplantation, he has served on numerous national committees, including at the National Institutes of Health, the National Kidney Foundation, and ASN. He has served as associate editor of the *American Journal of Transplantation* and as statistical editor of *JASN*. Among many honors, he received the Young Investigator Award from the American Transplant Congress, an Excellence in Science Award from the Lerner Research Institute, and a Clinical Science Established Investigator Award from the American Society of Transplantation.

Dr. Schold received master's degrees in statistics and mathematics education from North Carolina State University and a doctorate in health services research from the University of Florida.

PLENARY SESSION

STATE-OF-THE-ART LECTURE

Genetics Researcher to Describe Efforts to Find Gene-Editing Medicines

Sekar Kathiresan, MD

The founder of a biotechnology company will describe efforts to transform medicine's approach to cardiovascular disease. Sekar Kathiresan, MD, will speak on "From Reading the Genome for Risk to Rewriting It for Cardiovascular Health" at a plenary on Friday, October 25.

Dr. Kathiresan is cofounder and chief executive officer of Verve Therapeutics, a biotechnology company pioneering a new approach to the care of cardiovascular disease, with the hope of transforming treatment from chronic management to single-course gene-editing medicines.

Dr. Kathiresan is a cardiologist and scientist who has focused his career on understanding the inherited basis for heart attacks and leveraging those insights to improve the care of cardiovascular disease. In the past decade, genetics research has shown that naturally occurring gene variants can dramatically lower some individuals' lifetime risk of atherosclerotic cardiovascular disease. Based on his groundbreaking discoveries of these genetic mutations that confer resistance to cardiovascular disease, Dr. Kathiresan cofounded Verve Therapeutics with a vision of mimicking the mutations by inactivating specific genes to lower blood lipids through a single course of gene-editing therapy to address the root causes of this disease. Verve is advancing initial programs that target *PCSK9* and *ANGPTL3*—genes that have been extensively validated by Dr. Kathiresan and others as targets for lowering blood lipids, such as low-density lipoprotein cholesterol, which is a major driver of cardiovascular disease.

Prior to joining Verve, Dr. Kathiresan's roles included serving as director of the Massachusetts General Hospital Center for Genomic Medicine, director of the Cardiovascular Disease Initiative at the Broad Institute, and professor of medicine at Harvard Medical School.

At Harvard, Dr. Kathiresan's research laboratory focused on understanding the inherited basis for blood lipids and myocardial infarction. His research contributions have been recognized by the American Heart Association with its Distinguished Scientist Award and by the American Society of Human Genetics with the Curt Stern Award.

Dr. Kathiresan received an MD from Harvard Medical School. He completed his clinical training in internal medicine and cardiology at Massachusetts General Hospital and his postdoctoral research training in human genetics at the Framingham Heart Study and the Broad Institute.

Scott J. Gilbert to Be Given the Robert G. Narins Award for Contributions in Education

Scott J. Gilbert, MD, FASN

Scott J. Gilbert, MD, FASN, will receive the Robert G. Narins Award on Friday, October 25, for his many efforts in the education and training of the next generation of nephrologists. Dr. Gilbert is professor of medicine at Tufts University School of Medicine and a nephrologist at Tufts Medical Center in Boston, MA. He also directs the nephrology fellowship program and core faculty of the internal medicine residency program.

At Tufts University School of Medicine, he has led the first-year renal course for more than 20 years, coordinates the fourth-year consultative nephrology elective, and leads sessions in courses on problem-based learning, introduction to clinical reasoning, and medical ethics. He is an academic coach for 20 medical students through their 4-year medical school experience.

He is active in premedical mentoring at Tufts University, serving as faculty adviser to the Minority Association of Pre-Health Students, an organization for racial and ethnic minorities who are under-represented in health care, with an interest in the health professions, as well as the Kidney Disease Screening and Awareness Program, a student-run organization that provides blood pressure, blood sugar, and urinalysis screening in underserved communities in the Boston area.

Dr. Gilbert recently engaged in programs to promote diversity in medicine as a program leader and career mentor in the Tufts University School of Medicine/University of Massachusetts Boston Enrichment Program and through the development and oversight of a summer clinical immersion program for undergraduate students who are under-represented and underserved and are "first in family" to attend college, showcasing career opportunities in health care that they might not have known about. He is editor of the *National Kidney Foundation's Primer on Kidney Diseases*, previously served as education editor for the *American Journal of Kidney Diseases*, and chaired the ASN Workforce and Training Committee.

He attended Washington University School of Medicine in St. Louis, MO, before completing an internal medicine residency and nephrology fellowship at Beth Israel Deaconess Medical Center.

John P. Peters Award to Honor Susan E. Quaggin

Susan E. Quaggin, MD, FASN

ASN will recognize the wide-ranging contributions of Susan E. Quaggin, MD, FASN, with the presentation of the John P. Peters Award on Friday, October 25. This award is given for outstanding contributions to improving the lives of patients and furthering the understanding of the kidney in health and disease.

Dr. Quaggin is the Irving S. Cutter Professor of Medicine at Northwestern University Feinberg School of Medicine, where she also chairs the Department of Medicine and directs the Feinberg Cardiovascular and Renal Research Institute.

Her research focuses on the fundamental processes needed to establish and maintain the integrity of the specialized vascular beds in the kidney and eye. To understand and identify new therapeutic targets, she developed genetic mouse models that allow cell- and time-specific manipulation of functional genes. Her group's findings about the vasculature have revealed pathogenic mechanisms and have led to new therapeutic targets for a number of diseases, including diabetic kidney disease, nephrotic syndrome, and glaucoma.

Dr. Quaggin has served ASN in many roles, including as president; associate editor of *JASN*; chair of Kidney Week's Program Committee and of the Diabetic Kidney Disease Collaborative Task Force; and member of the Public Policy Board, Education Committee, Grants Review Committee, and more. She has also served on the councils of The American Society for Clinical Investigation and the International Society of Nephrology. She chaired the National Institutes of Health's pathobiology of kidney disease study section. She is on the scientific advisory boards of AstraZeneca and Roche and is cofounder of Mannin Research.

Among her many honors, Dr. Quaggin received the International Society of Nephrology Alfred Newton Richards Award for basic research, a Kidney Foundation of Canada medal for research excellence, an American Heart Association distinguished scientist award, and an honorary doctorate from the University of Southern Denmark. She was corecipient of the Grand Prix Scientifique from the Fondation Lefoulon-Delalande.

Dr. Quaggin received her medical degree from the University of Toronto and completed her residency at the university's St. Michael's Hospital. She completed her nephrology fellowship and additional research training at the University of Toronto and Yale University. She also trained in the developmental biology program at the University of Toronto's Samuel Lunenfeld Research Institute.

PLENARY SESSION

Young Investigator Hiddo Heerspink Recognized for Work on Clinical Trial Design

Hiddo Jan L. Heerspink, PhD

The ASN-American Heart Association Donald W. Seldin Young Investigator Award will be presented to Hiddo Jan L. Heerspink, PhD, who will speak on "Innovations in Clinical Trial Design and Conduct for New Therapeutic Approaches in CKD" on Friday, October 25.

Dr. Heerspink is professor of clinical trials and personalized medicine and a clinical trialist in the Department of Clinical Pharmacy and Pharmacology at the University Medical Center Groningen in The Netherlands. He is also a visiting professor at the University of New South Wales, Sydney, Australia.

Dr. Heerspink's research focuses on optimizing treatment strategies and finding new therapeutic approaches to halt the progression of chronic kidney disease (CKD) and cardiovascular disease. His main expertise includes clinical trial design, personalized medicine, and methodology and statistical analysis of clinical trials. He has successfully led and currently leads numerous clinical trials, including trials of dapagliflozin in CKD (DAPA-CKD), finerenone in nondiabetic CKD (FIND-CKD), atrasentan in patients with immunoglobulin A nephropathy (ALIGN), and a comparison of drugs in treating CKD and high proteinuria (ZENITH-CKD).

Dr. Heerspink has published more than 500 peer-reviewed publications and is an editorial board member of *Diabetes, Obesity and Metabolism* and *CJASN*. He holds five patents in the areas of biomarkers and CKD. He serves as a member of various committees of international professional organizations, including ASN and the American Diabetes Association.

Among many honors, he has received the Foreign Scientist Award from the Japanese Diabetes Society, the Rising Star Award and the Camillo Golgi Prize from the European Association for the Study of Diabetes, a Young Researcher Award from the Canadian Society Nephrology, and a Top Publication Award from the Dutch Society for Clinical Pharmacology & Biopharmacy.

Dr. Heerspink studied pharmacology at the University of Groningen and received his PhD from the University Medical Center Groningen.

Eknoyan Lecture to Focus on Disparities in Diabetic Kidney Disease

Adeera Levin, MD

The Garabed Eknoyan, MD, Endowed Lectureship will be delivered by a leader in kidney disease research and care on Friday, October 25. Adeera Levin, MD, will speak on "Disparities Between Men and Women in the Course of Diabetic Kidney Disease."

Dr. Levin is professor of medicine and head of the Division of Nephrology at The University of British Columbia. She is the principal investigator for the Strategy for Patient-Oriented Research Network grant (Can-SOLVE CKD Network), a pan-Canadian initiative for kidney research. She is also the executive director of BC Renal, an organization that oversees the care, planning, and budgets for kidney services in the province of British Columbia, Canada. In this capacity, she has leveraged her epidemiologic training, clinical knowledge, and health outcomes research to develop an evidence-based transparent system that enhances patient care across the continuum of patient needs.

Dr. Levin is active in international activities across the spectrum of kidney education, research, and administration, including serving as president of the International Society of Nephrology. Her major research interests include cardiovascular disease in patients with chronic kidney disease (CKD), variability in the progression of CKD, and optimal models of care. She collaborates with investigators across Canada and internationally.

She has several hundred peer-reviewed publications and book chapters and is coeditor of the textbook, *Chronic Kidney Disease: A Practical Guide to Understanding and Management*. She serves on several editorial boards, reviews for major kidney journals, and was the inaugural editor-in-chief of the *Canadian Journal of Kidney Health and Disease*.

Dr. Levin has received many teaching and research awards from local and national groups, including an award from the Canadian Society of Nephrology for outstanding contributions to Canadian nephrology; the National Kidney Foundation's Research Medal of Excellence and its International Distinguished Medal for a significant contribution to the field of kidney diseases; the Aubrey J. Tingle Research Award for contributions to the province of British Columbia; and the Order of Canada, the country's highest civilian honor, for her contributions to the life of Canadians.

Dr. Levin received her medical degree from McMaster University, followed by a residency in internal medicine at McMaster University and Henderson General Hospital (currently Juravinski Hospital). She completed a clinical fellowship and a clinical research fellowship in nephrology at Toronto Western Hospital and St. Michael's Hospital of the University of Toronto.

Patient Advocate to Speak on Donor Kidney Selection

Nichole M. Jefferson, MBA

A patient who has been on dialysis and received a kidney transplant will discuss the patient experience in a talk entitled "There Is No Debate: Patients Should Be Part of the Selection Conference" at the Celeste Castillo Lee Endowed Lectureship on Friday, October 25. The speaker, Nichole M. Jefferson, MBA, is a risk management and compliance analyst for Wells Fargo in Dallas, TX.

When Ms. Jefferson was diagnosed with kidney failure in 2003, she was taken by surprise and unaware of its meaning. After experiencing both hemodialysis and peritoneal dialysis, she preferred the convenience of peritoneal dialysis.

She received a kidney transplant in 2008. The many obstacles that she faced following her transplant led her to the realization that a transplant is simply another form of treatment and not a cure. This understanding sparked a quest for knowledge, which turned to enthusiasm for advocacy in kidney care. She relied on this devotion and encouragement for what she describes as her "fight for a second transplant" in April 2024.

Ms. Jefferson has volunteered with local chapters of the National Kidney Foundation. Spurred by the lack of recognition of the prevalence of kidney diseases in the Black community, she sought to raise consciousness in her community and in underserved populations. She began speaking with legislators regarding the need for early detection and other issues relating to chronic kidney disease.

In 2015, she was selected to join the National Kidney Foundation Kidney Advocacy Committee. In 2016, she was invited to get involved with the Kidney Health Initiative's Patient and Family Partnership Council. In 2017, she joined the board of directors of Home Dialyzors United and was elected president of the board. These opportunities have allowed her to broaden her audience to include nephrologists, researchers, scientists, and other key stakeholders in the kidney community.

Rose Lecture Will Examine Fluid and Electrolyte Disorders

Roger A. Rodby, MD, FASN

"Water Disorders" is the title of the Burton D. Rose, MD, Endowed Lectureship, which Roger A. Rodby, MD, FASN, will deliver on Friday, October 25.

Dr. Rodby is a professor in the Division of Nephrology at Rush University, where he is also director of inpatient renal replacement therapies and director of the fellowship training program.

Dr. Rodby's main research focus is on lupus nephritis and diabetic nephropathy. As a member of the Collaborative Study Group, he has authored over 150 articles across a wide spectrum of clinical nephrology topics. A leader in internet-based education through social media, he has more than 14,000 X (formerly Twitter) followers. He has more than 7500 posts as a community leader of ASN's online "Open Community." He is also a member of the *American Journal of Kidney Diseases* Social Media Advisory Group.

He is working with seven other colleagues across the nation in a multiyear podcast, "Channel Your Enthusiasm," which dissects each chapter of Dr. Rose's *Clinical Physiology of Acid-Base and Electrolyte Disorders*. In 2020, he opened the Rush University Medical Center renal biopsy conferences so that anyone could attend virtually through live Zoom presentations. There are now approximately 100 presentations available for viewing on YouTube.

Dr. Rodby was codirector of ASN's Board Review Course and Update for 6 years. In more traditional media, he has served as assistant editor of Seminars in Dialysis and on the editorial boards of the *American Journal of Kidney Diseases* and *Kidney360*. He has also been active in the National Kidney Foundation of Illinois.

Dr. Rodby received his medical degree from the University of Illinois. He completed his residency in internal medicine and served as chief resident at the University of Medicine and Dentistry of New Jersey. After concluding his fellowship training in nephrology at Rush University Medical Center, he joined its faculty, where he has remained for more than 30 years.

Researcher in Pathogenesis of Kidney Diseases to Speak on Genetic Discoveries

Kirk N. Campbell, MD, FASN

A researcher known globally for his work in the genetics of kidney diseases will deliver the Michelle P. Winn, MD, Endowed Lectureship on Friday, October 25. Kirk N. Campbell, MD, FASN, will present a lecture titled "APOL1-Mediated Kidney Disease: Variably Proteinuric, Yet Invariably Progressive."

Dr. Campbell is the Irene and Dr. Arthur M. Fishberg Professor of Medicine in the Division of Nephrology and professor of pharmacological sciences at the Icahn School of Medicine at Mount Sinai in New York City. He is the founding director of the Mount Sinai Center for Kidney Disease Innovation, which has the goal of leveraging state-of-the-art genomic, bioinformatics, and molecular phenotyping technology to identify novel therapeutic targets for drug discovery and biomarker validation while expanding patient registries, biobanking, and clinical trial opportunities.

Dr. Campbell leads a National Institutes of Health-funded research program focused on podocyte cell biology, glomerular disease, and clinical trials in the rare kidney disease space. The work centers on advancing the understanding of the underlying mechanisms involved in kidney disease progression while identifying targets for therapeutic intervention and has resulted in two patents.

Dr. Campbell cochaired the ASN Kidney Week 2022 Education Committee and served on the American Kidney Fund group focused on advancing strategies for inclusive clinical trial enrollment. He has been a principal investigator for multiple clinical trials, testing novel agents to treat focal segmental glomerulosclerosis, immunoglobulin A nephropathy, lupus nephritis, and membranous nephropathy. He leads Mount Sinai's kidney precision medicine recruitment site, overseeing the safe and ethical collection of kidney biopsies, clinical data, and samples from patients with acute kidney injury and chronic kidney disease.

He is the president-elect of the National Kidney Foundation. He is a past president of The New York Society of Nephrology and a member of the board of directors of the NephCure Foundation. Dr. Campbell is also a member of the editorial boards of the *American Journal of Kidney Diseases*, *Kidney360*, *Kidney International*, *Frontiers in Medicine*, and the *American Journal of Physiology–Renal Physiology*. Among many honors, he received an ASN Distinguished Leader Award.

Dr. Campbell received his MD from the University of Connecticut School of Medicine, followed by an internal medicine residency at Yale New Haven Hospital and a nephrology fellowship at the Icahn School of Medicine at Mount Sinai.

PLENARY SESSION

STATE-OF-THE-ART LECTURE

Nephrology Science Pioneer to Speak on Stem Cells in Kidney Tissue Research

Melissa H. Little, PhD

An internationally recognized researcher will describe the state-of-the-art lecture in "Understanding, Improving, and Applying Human Pluripotent Stem Cell-Derived Kidney Tissues" at a plenary session on Saturday, October 26. Melissa H. Little, PhD, is chief executive officer of the Novo Nordisk Foundation Center for Stem Cell Medicine, chief scientist at the Murdoch Children's Research Institute, and leader of the Kidney Regeneration Laboratory in Melbourne, Australia.

A top pioneer in the field of renal stem cell biology and renal regeneration, Dr. Little has spent decades developing new regenerative treatment options. She was part of the first team to successfully grow human kidney organoids in a petri dish and performed pioneering studies into potential regenerative therapies in the kidney. This work has revolutionized the field by providing researchers with a safer and more effective way to study diseases and test drugs. Dr. Little's approach to generating kidney organoids from human pluripotent stem cells has been adopted across the globe, where it is being applied to disease modeling, drug screening, and renal replacement therapies.

Dr. Little is the immediate past president of the International Society for Stem Cell Research and holds an honorary position as professor in the Department of Pediatrics at The University of Melbourne. She has previously been president of the Australasian Society for Stem Cell Research, program leader of Stem Cells Australia, and chief scientific officer of the Australian Stem Cell Centre. She is a companion of the Order of Australia and a fellow of the Australian Academy of Science, the Australian Academy of Health and Medical Sciences, and The Royal Danish Academy of Sciences and Letters. She serves on the editorial boards of *Cell Stem Cell*, *Nature Reviews Nephrology*, *Development*, and *Kidney International*.

Dr. Little has received many awards for her work, including the GlaxoSmithKline Award for Research Excellence, an Eisenhower Fellowship, a Boerhaave Professorship and honorary doctorate from Leiden University, the Eureka Prize from the Australian Museum, the Alfred Newton Richards Award from the International Society of Nephrology for her kidney organoid research, the Julian Wells Medal for her outstanding contribution to understanding the genetic basis of kidney development, and the Homer W. Smith Award from ASN for contributions to the science of nephrology,

Dr. Little received a doctorate in biochemistry from The University of Queensland.

Pioneering Researcher Vesna Garovic to Receive Barbara T. Murphy Award

Vesna D. Garovic, MD, PhD, FASN

ASN will present the Barbara T. Murphy Award to Vesna D. Garovic, MD, PhD, FASN, on Saturday, October 26. This is just the fourth presentation of this award, which was named after the nephrology leader and late ASN president-elect. It honors leaders who strengthen the foundation of nephrology while advancing the field through innovation, creativity, inspiration, and tenacity.

Dr. Garovic is professor of medicine and chair of the Division of Nephrology and Hypertension at the Mayo Clinic in Rochester, MN. She holds a joint appointment in the Department of Obstetrics and Gynecology and is vice chair for research in the Department of Internal Medicine, director of the Mayo Clinic Center for Clinical and Translational Science, and dean of clinical and translational science at the Mayo Clinic.

When she recognized the unmet need for nephrologists to develop expertise in pregnancy-related kidney and cardiovascular diseases, she became a pioneer who dedicated her career to this field of inquiry. She has been funded by the National Institutes of Health (NIH) for the past 15 years and has published more than 200 peer-reviewed papers.

Dr. Garovic's clinical and research interests in hypertension, in general, and hypertensive pregnancy disorders, in particular, span several research areas: diagnosis, treatment, and underlying molecular mechanisms, with a recent focus on the senescence, epigenetics, and epidemiology of cardiovascular and renal complications.

She served as a chartered member of the NIH Pregnancy and Neonatology Study Section and as a member of the American Heart Association (AHA) Councils on Hypertension and on the Kidney in Cardiovascular Disease Science Subcommittee. She chaired the Writing Group for the AHA scientific statement on hypertension in pregnancy. Among many honors, Dr. Garovic received the Marvin Moser Clinical Hypertension Award from the AHA and the Council on Hypertension.

Dr. Garovic earned her medical degree, a master's of science in biochemistry, and a doctorate in molecular biology and obstetrics and gynecology at the University of Belgrade in Serbia, where she also completed a residency and fellowship in obstetrics and gynecology. She earned a second master's in medical genetics from McGill University in Montreal, Quebec, Canada. She then completed an internal medicine residency and nephrology fellowship at the Albert Einstein College of Medicine and Montefiore Medical Center in New York City.

ASN to Bestow Belding H. Scribner Award on Claudio Ronco

Claudio Ronco, MD

The Belding H. Scribner Award will be tendered on Saturday, October 26, to Claudio Ronco, MD. Dr. Ronco's clinical and basic research efforts have significantly advanced the management of patients with kidney diseases and the science of nephrology.

Dr. Ronco is director of the International Renal Research Institute at San Bortolo Hospital in Vicenza, Italy, where he also served as director of the Department of Nephrology and Transplantation. He has also held positions as associate professor of nephrology at San Bartolo Hospital, director of the renal laboratory at the International Renal Research Institute, professor of medicine at the Albert Einstein College of Medicine and Beth Israel Deaconess Medical Center in New York City, and visiting professor at The George Washington University in Washington, DC.

Established in 1995, the Belding H. Scribner Award is presented to individuals who have made outstanding contributions to the care of patients with renal disorders or have substantially influenced the clinical practice of nephrology. Dr. Ronco is receiving the award because of his achievements as a pioneer in many areas of nephrology, including peritoneal dialysis, critical care nephrology, continuous renal replacement therapy, cardiorenal syndromes, and wearable dialysis technology. He has coauthored 1146 papers, 65 books, and 80 book chapters and has delivered more than 1000 lectures and presentations at international meetings and universities.

Dr. Ronco has served on the board of directors of five scientific societies and served as president of the International Society for Hemodialysis. He cofounded the Acute Dialysis Quality Initiative consensus group with colleagues from the University of California and the University of Pittsburgh. He is editor-in-chief of *Blood Purification* and *Contributions to Nephrology*, associate editor of *Critical Care*, and editor emeritus of *The International Journal of Artificial Organs*.

Dr. Ronco has received numerous awards including lifetime achievement awards for hemodialysis and peritoneal dialysis from the Annual Dialysis Conference, the Bywaters Award from the International Society of Nephrology, an international medal of excellence and the J. Michael Lazarus Distinguished Award from the National Kidney Foundation, and the Belding Scribner Award from the International Society for Hemodialysis.

Dr. Ronco graduated in medicine from the University of Padua in Italy and then completed specialty training in nephrology at the University of Padua and in pediatric nephrology at the University of Naples Federico II, Italy.

Joseph Bonventre to Receive Smith Award

Joseph V. Bonventre, MD, PhD, FASN

Prominent investigator Joseph V. Bonventre, MD, PhD, FASN, will be presented the Homer W. Smith Award on Saturday, October 26. This award recognizes outstanding contributions to understanding how kidneys function in normal and diseased states. Dr. Bonventre will speak on "Acute Kidney Injury, Repair, and Generation of Kidney Tissue."

He is the Samuel A. Levine Professor of Medicine at Harvard Medical School and professor of health sciences and technology at the Massachusetts Institute of Technology (MIT). He is also chief of the Renal (Kidney) Medicine Division and founding chief of the Engineering in Medicine Division at Brigham and Women's Hospital. He directed the Harvard-MIT Program in Health Sciences and Technology for 10 years.

Dr. Bonventre's research focuses primarily on the study of kidney injury and repair and signal transduction, with an emphasis on the role of inflammation, biomarkers, and stem cells. His recent work also involves the generation of kidney organoids from stem cells and their use in kidney disease modeling. Among many discoveries, Dr. Bonventre established the origin of epithelial cells that repair the kidney after injury as dedifferentiated surviving proximal tubule cells. He was the first to describe the role of proximal tubule cell cycle arrest in maladaptive fibrosis that can occur after severe injury leading to chronic kidney disease.

He has published more than 420 original research papers; 160 reviews, chapters, and editorials; and 3 books. His work has been referenced more than 94,000 times, and he is one of the most frequently cited authors in the area of acute kidney injury.

Dr. Bonventre is past president of ASN, a founding member of the board of directors of the National Space Biomedical Research Institute, and a member of the Executive Committee of the Harvard Stem Cell Institute. He is a past councilor of the International Society of Nephrology. He has chaired many National Institutes of Health (NIH) study sections, served as a member of the Advisory Council of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), and is editor of *Seminars in Nephrology*. Among many honors, he has received the Osler Medal of the Royal College of Physicians, the Bywaters Award of the International Society of Nephrology, the Massry Award of the National Kidney Foundation, two MERIT awards from the NIH-NIDDK, and honorary doctorates from Mount Saint Mary College and the Norwegian University of Science and Technology.

Dr. Bonventre received an MD and a PhD in biophysics from Harvard University. He completed his residency, clinical fellowship, and research fellowship at Massachusetts General Hospital and Harvard University.

Immune System in Transplantation to Be Subject of Murphy Lectureship

Fadi G. Lakkis, MD

A leader in transplantation immunology will provide his insights in the Barbara T. Murphy, MB BAO BCh, Endowed Lectureship on Saturday, October 26. The speaker, Fadi G. Lakkis, MD, is a distinguished professor of surgery and professor of immunology and medicine at the University of Pittsburgh. His talk is titled "Selective Targeting of the Innate Immune System in Transplantation: The Time Is Now."

The work in Dr. Lakkis' National Institutes of Health-funded laboratory focuses on two areas of transplantation immunology: allorecognition in the innate immune system in both invertebrate and vertebrate models and the biology of memory T cells in organ transplantation in animals and humans.

He has served ASN, the American Society of Transplantation, and the National Kidney Foundation on numerous committees and has been on the board of directors of the American Society of Transplantation. Dr. Lakkis has served as associate editor of *The Journal of Immunology* and the *American Journal of Transplantation*; on the editorial boards of *JASN*, *Graft*, and the *American Journal of Transplantation*; and is currently consulting editor of *The Journal of Clinical Investigation*. Among many honors, early in his career, he received young investigator awards from ASN, The Transplantation Society, and the American Society of Transplantation, which were followed by additional awards for research excellence from ASN, the National Kidney Foundation, and the American Society of Transplantation.

Dr. Lakkis received his medical degree from the American University of Beirut, Lebanon, and completed his fellowship training in nephrology and transplantation immunology at the Beth Israel Deaconess Medical Center of Harvard Medical School.

After serving on the faculties of Emory University and Yale University, Dr. Lakkis joined the University of Pittsburgh in 2005 as the Frank and Athena Sarris Chair in Transplantation Biology and the scientific director of the Thomas E. Starzl Transplantation Institute.

Bone Disease Researcher to Provide Update in Coburn Lecture

Thomas L. Nickolas, MD, MS

Thomas L. Nickolas, MD, MS, a leading researcher into the skeletal effects of kidney diseases, will deliver the Jack W. Coburn, MD, Endowed Lectureship on Saturday, October 26, on the topic of "CKD-MBD and the Skeleton."

Dr. Nickolas is professor of medicine and a member of the Division of Nephrology at Columbia University Irving Medical Center.

Dr. Nickolas' laboratory focuses on improving the understanding of, and approach to, bone disease in adults and children with kidney failure. His research uses advanced methods to phenotype bone disorders in patients with kidney failure and after kidney transplantation, including dual-energy x-ray absorptiometry, high-resolution in vivo bone imaging, bone biopsy, bone cell culture, and transcriptomics.

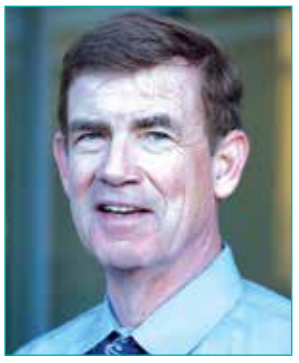
As part of this work, he conducts clinical trials to test the efficacy of therapeutic approaches to protect the skeleton from the effects of kidney diseases. He is also involved in research on the skeletal effects of other systemic diseases such as HIV and on the adverse effects on the skeleton of bone therapies such as bisphosphonates. He collaborates with colleagues in the United States, Brazil, Europe, and Australia.

Dr. Nickolas is a member of the Committee of Scientific Advisors of the International Osteoporosis Foundation, cochaired the bone disease section of the 2023 KDIGO Controversies Conference on Personalizing CKD-MBD Care, and has served as a member of various National Institutes of Health study sections.

He has served ASN as a national meeting abstract reviewer and on the Program Committee for chronic kidney disease (CKD) and mineral and bone disease (MBD). He has been on the editorial boards of *CJASN*, *Osteoporosis International, Bone*, *Bone Reports*, and the *Journal of Bone and Mineral Research*.

After receiving his medical degree from the University of Pittsburgh School of Medicine, Dr. Nickolas completed a residency in internal medicine at the University of Pennsylvania; an epidemiology fellowship at the European Institute of Oncology in Milan, Italy; and a fellowship in nephrology at Columbia University. He has a master's degree in biostatistics/epidemiology from the Columbia University Mailman School of Public Health.

Brenner Lectureship to Feature Glomerular Filtration Researcher



Timothy W. Meyer, MD

A researcher who has spent 20 years studying kidney failure will deliver the Barry M. Brenner, MD, Endowed Lectureship on Saturday, October 26. Timothy W. Meyer, MD, will speak on "Unraveling the Mysteries of High GFR in Health and Disease."

Dr. Meyer recently retired as a professor at Stanford University and as nephrology section chief at VA Palo Alto Health Care to devote himself to the investigation of uremia at his VA Palo Alto laboratory, where he continues to be formally employed part-time.

Dr. Meyer began his research studying kidney disease progression in animals; for the past 20 years, he has focused on kidney failure and its treatment by dialysis. He became interested in kidney failure because, as a practicing academic nephrologist, he saw that dialysis treatment does not restore normal health. Believing that some of the ill health experienced by patients undergoing dialysis must be attributable to the imperfect removal of organic waste solutes, he worked to identify these solutes and devise means to limit their accumulation. His work on quantifying and improving solute removal by dialysis and related therapies has emphasized finding ways to adjust dialysis prescriptions to control solute levels.

Dr. Meyer has also worked on suppressing the production of colon-derived solutes in collaboration with investigators in Stanford's Department of Microbiology and Immunology and Department of Bioengineering. His research has been designed to provide participating fellows with a better understanding of the function of the kidney and of the limitations of current kidney replacement therapies.

Among many professional positions, he has served as associate editor of *JASN*, as chair of the ASN Program Committee, on the Scientific Advisory Council of the former Juvenile Diabetes Research Foundation (now Breakthrough T1D), and on the external advisory committees for multiple studies.

Dr. Meyer received his MD from Harvard University, followed by residencies at Stanford University and Sydney Hospital in Australia. He completed an extended fellowship in adult nephrology at Brigham and Women's Hospital with a clinical year sandwiched between 2 years of metabolism research in the laboratory of George Cahill Jr., MD, and 3 years of research in progressive chronic kidney disease in the laboratory of the late Barry Brenner, MD. Following this fellowship, Dr. Meyer moved to Palo Alto, CA, where he has remained.

PLENARY SESSION

STATE-OF-THE-ART LECTURE

Sickle Cell Disease Expert to Provide Look Into Future

Michael R. DeBaun, MD, MPH

A researcher who has spent his career investigating sickle cell disease will look ahead at coming developments at a plenary session on Sunday, October 27. Michael R. DeBaun, MD, MPH, will speak on "The Curative Therapy for Sickle Cell Disease: The Good, the Bad, and the Future."

Dr. DeBaun is professor of pediatrics and medicine as well as vice chair of clinical and translational research in the Department of Pediatrics at Vanderbilt University School of Medicine. He is also director of the Vanderbilt-Meharry Center of Excellence in Sickle Cell Disease. Before arriving at Vanderbilt in 2010, he was professor of pediatrics, neurology, and biostatistics at Washington University School of Medicine in St. Louis.

For more than 25 years, Dr. DeBaun's research has led to fundamental changes in the understanding of the clinical epidemiology, pathogenesis, prevention, and treatment of overt and silent strokes in children and adults with sickle cell disease. He has been the principal investigator or coleader of seven clinical trials funded by the National Institutes of Health or foundations designed to prevent strokes in patients with sickle cell disease in North America, Europe, and Nigeria.

He recently completed two successful trials to cure sickle cell disease in children and adults. A trial of nonmyeloablative haploidentical bone marrow transplants with thiotepa and post-transplant cyclophosphamide showed that this treatment had efficacy equivalent to myeloablative gene therapy options recently approved by the US Food and Drug Administration—at one-fifth of the cost.

Dr. DeBaun's impact extends beyond his research and advocacy work. In 2017, he created the Vanderbilt-Meharry James Puckette Carter Summer Scholarship Program, designed to spark interest in a career in academic medicine. Over the past 2 decades, he has mentored countless students, medical residents, postdoctoral fellows, and faculty members domestically and in Africa. His mentoring efforts have been recognized by the Maureen Andrew Mentor Award from the Society for Pediatric Research and by the American Society of Hematology Mentor Award for clinical research.

Advocacy is another important component of Dr. DeBaun's work. He was the primary physician author of the American Jobs Creation Act of 2004 to expand services for children and adults with sickle cell disease, which was signed by President George W. Bush. He was the physician consultant for a law authorizing Tennessee Medicaid insurers to provide increased services and education related to sickle cell disease.

Dr. DeBaun received an MD from Stanford University and an MPH from Johns Hopkins School of Medicine. He completed a residency in pediatrics and a fellowship in pediatric hematology/oncology at Washington University School of Medicine in St. Louis and St. Louis Children's Hospital, as well as a fellowship in epidemiology at the National Cancer Institute.

ASN Announces Midcareer Award Winners

ASN's Midcareer Awards recognize individuals who have made substantial and significant contributions in a variety of areas early in their professional lives.

Presented on Sunday, October 27, these awards recognize up to three winners in each of four categories: clinical service, education, leadership, and research.

Distinguished Clinical Service Award

Neera K. Dahl, MD, PhD, FASN

Dr. Dahl is a senior associate consultant in the Division of Nephrology and Hypertension at the Mayo Clinic Rochester and adjunct professor in the Section of Nephrology at Yale School of Medicine. She directs the Mayo Clinic's Polycystic Kidney Disease Foundation Center of Excellence.

During her time as a clinician-educator at Yale from 2007 to 2023, she ran the Yale Inherited Diseases of the Kidney program, which focused on autosomal-dominant polycystic kidney disease (ADPKD). She built a clinical trials program to support research opportunities for her patients. Dr. Dahl was the principal investigator for every major industry-sponsored clinical trial in ADPKD, including the development of treatment with tolvaptan. The program was the clinical arm of a much broader Yale ADPKD research program that includes internationally recognized basic research scientists. In recognition of the comprehensive care that her program delivered, the Yale nephrology program earned a Center of Excellence designation and a Patient Navigator Award from the Polycystic Kidney Disease Foundation.

Her skill at translating basic science research findings into clinical practice has led to her being in demand as a speaker in clinical genetics and cystic kidney disease, both nationally at ASN and the National Kidney Foundation and locally to teach about new developments in these evolving areas.

Dr. Dahl has published 65 original articles. She is associate editor for *Kidney360* and served on the editorial board of ASN's Kidney Self-Assessment Program. She is a member of the Scientific Advisory Committee of the Polycystic Kidney Disease Foundation, for which she cochaired the Center of Excellence Advisory Board.

She received an MD and a PhD in physiology from Tufts University School of Medicine and then completed a residency and fellowship at the Beth Israel Deaconess Medical Center in Boston, MA.

PLENARY SESSION

Distinguished Clinical Service Award

Ladan Golestaneh, MD, MS, FASN

Dr. Golestaneh is professor of medicine in the Division of Nephrology at the Montefiore Medical Center of the Albert Einstein College of Medicine in New York City. She is also medical director of the Williamsbridge Home Dialysis Center and director of end stage kidney disease at the Montefiore Care Management Organization.

Early in her career, Dr. Golestaneh established Montefiore's first continuous replacement therapy program. From 2006 to 2009, she directed the inpatient hemodialysis facility at Montefiore. In 2009, she became medical director of the Bedford Park Dialysis Center. During this time, she developed the intensive care unit nephrology curriculum at Montefiore and became a consultant to the heart transplant program. In 2012, she joined the Montefiore Care Management Organization as the expert consultant for its population with chronic kidney disease and led multiple innovative projects. In 2014, she became medical director of the Williamsbridge Home Dialysis Center of Excellence.

The next year, Dr. Golestaneh enrolled in the clinical research training program at the Albert Einstein College of Medicine and received her master's degree in clinical research methods. Since completing this degree, she has focused her attention on grant-funded research designed to improve outcomes for patients with kidney diseases.

She is globally recognized through multiple speaking engagements, through numerous publications, as an ad hoc reviewer for major scientific journals, as a content expert and leader for the Centers for Medicare & Medicaid Services' Kidney Care program, and through multiple research collaborations.

Dr. Golestaneh received her MD from New York Medical College, after which she completed a residency and fellowship in nephrology at the Albert Einstein College of Medicine and Montefiore Medical Center.

Distinguished Educator Award

Samira S. Farouk, MD, MS, FASN

Dr. Farouk is a transplant nephrologist, associate professor of medicine and medical education, associate program director of the nephrology fellowship program, and social media director of the Division of Nephrology at the Icahn School of Medicine at Mount Sinai in New York City.

Involved in all levels of medical education, Dr. Farouk teaches medical students, residents, and fellows. She is interested in the development and study of innovative medical educational tools and technologies, including free open access medical education and social media. She is a cofounder of the mobile-friendly, teaching-tool website NephSIM, founder of the virtual nephrology mentoring program NephSIM Nephrons, and cofaculty lead of the Renal Fellow Network.

She served on the executive committee of NephMadness and the Nephrology Social Media Collective. She is currently deputy codirector of the ASN Board Review Course and Update, president of The New York Society of Nephrology, associate editor of *Kidney Medicine*, and a member of the editorial boards of several journals, including the *American Journal of Kidney Diseases* and *Clinical Transplantation*. Among many honors, Dr. Farouk has received the ASN William and Sandra Bennett Clinical Scholars Program KidneyCure Award, was the winner of the 2018 ASN Innovations in Kidney Education Contest, and has received many teaching awards.

She received her medical degree from the Rutgers University Robert Wood Johnson Medical School, where she graduated with a distinction in research. She completed her internship, residency, and nephrology and transplant fellowship at the Icahn School of Medicine at Mount Sinai.

Distinguished Educator Award

Abhilash Koratala, MD, FASN

Dr. Koratala is associate professor of medicine and director of clinical imaging for nephrology at the Medical College of Wisconsin. He is internationally known for his expertise in point-of-care ultrasonography (POCUS), particularly in bedside hemodynamic assessment using Doppler techniques, and for his educational efforts supporting it.

Dr. Koratala has delivered more than 100 invited talks on POCUS globally. He has developed comprehensive POCUS curricula for nephrology trainees, which have been published in the *American Journal of Kidney Diseases* and *Kidney360*. Dr. Koratala also teaches POCUS on online platforms such as Twitter/X, in which his handle @NephroP is among the most followed in medical education, boasting over 65,000 followers. He authors posts for "Focus on POCUS" on the Renal Fellow Network, a nephrology blog published in partnership with ASN. He serves as associate editor of the *POCUS Journal*.

His online POCUS teaching tool won the ASN Innovations in Kidney Education Contest in 2020. He won an educational tools contest sponsored by the American Heart Association Council on the Kidney in Cardiovascular Disease and has received exemplary teacher awards from the University of Florida. In addition to his expertise in POCUS, Dr. Koratala's work in all aspects of nephrology has led to more than 200 publications in peer-reviewed journals.

Dr. Koratala received his medical degree from Kakatiya Medical College in Warangal, India. He completed a residency in internal medicine at the BronxCare Health System (formerly Bronx Lebanon Hospital Center) in New York City, a fellowship in nephrology at the University of Florida in Gainesville, and a mini-fellowship in renal ultrasonography at Emory University in Atlanta.

Distinguished Educator Award

Laura J. Maursetter, DO, FASN

Dr. Maursetter is associate professor of medicine in the Division of Nephrology at the University of Wisconsin School of Medicine and Public Health and chief of medicine subspecialties at the William S. Middleton Memorial Veterans' Hospital in Madison.

Her career has focused on educational innovations at both local and national levels. An active member of ASN, she served on the Training Program Directors Committee, in which she helped to develop the initial method for program directors across the country to connect. As a member of the Workforce Committee, she was a leader in getting the Kidney TREKS (Tutored Research and Education for Kidney Scholars) program started and expanded to its present two locations to reach a broader audience. As a member of the Continuing Professional Development Committee, she has helped to lead the Board Review Course and Update over the past 7 years.

At the University of Wisconsin School of Medicine and Public Health, she was a nephrology fellowship program director, in which she created curricula in simulation, asynchronous quality improvement, and mentorship. In addition, she created both just-in-time and longitudinal evaluation systems to more efficiently assess and provide feedback to learners across the system. Dr. Maursetter was also instrumental in creating curricula for the medical school for both the Acute Care block and the Division of Nephrology. Her clinical work focuses on the underserved populations of Veterans Affairs and the Department of Corrections.

She has been honored with excellence in teaching awards from the University of Wisconsin.

Dr. Maursetter received her medical degree from the Chicago College of Osteopathic Medicine before completing her internal medicine residency at Advocate Lutheran General Hospital in Chicago. She completed her nephrology fellowship at the University of Wisconsin before joining the faculty.

Distinguished Leader Award

Amanda Hyre Anderson, PhD, MPH, FASN

Dr. Anderson is professor of epidemiology and medicine at The University of Alabama at Birmingham (UAB); vice chair for research in the Department of Epidemiology at The UAB School of Public Health; codirector of the Biostatistics, Epidemiology and Research Design unit of The UAB Center for Clinical and Translational Science; and adjunct professor of epidemiology at the Perelman School of Medicine at the University of Pennsylvania and at Tulane University.

A renal and cardiovascular disease epidemiologist with over 20 years of experience conducting epidemiological research, with a specialty in chronic kidney disease, Dr. Anderson is an active ASN member, having just completed her fourth year on the Publications Committee.

Her leadership has spanned across research, academics, administration, and service. She has participated in more than 20 National Institutes of Health-funded research projects and training grants. She is a principal investigator (PI) for the Chronic Renal Insufficiency Cohort (CRIC) Study Scientific and Data Coordinating Center. As contact PI for the George M. O'Brien Kidney Resource Alliance National Coordinating Center, she works with seven O'Brien Kidney National Resource Centers in support of kidney disease research nationwide. Dr. Anderson is also contact PI for a National Institutes of Health study investigating the gut microbiome and metabolome among a subset of participants in the CRIC study. She is also engaged in academic and administrative leadership at her university, serving on deans' councils, research advisory councils, and the faculty council.

She has published in high-impact nephrology journals and leading general medicine journals, including *The New England Journal of Medicine*, the *Journal of the American Medical Association*, and *Annals of Internal Medicine*.

Dr. Anderson received her MPH and PhD, both in epidemiology, from Tulane University.

Distinguished Leader Award

Charuhas V. Thakar, MD, FASN

Dr. Thakar is director of the Wellcome-Wolfson Institute for Experimental Medicine in the School of Medicine, Dentistry and Biomedical Sciences at Queen's University Belfast, Northern Ireland.

He has more than 2 decades of administrative and leadership experience in complex health care environments, including governmental, nonprofit, academic, and private sectors. He served as the Robert G. Luke Endowed Chair in Nephrology and director of the Division of Nephrology at the University of Cincinnati from 2013 to 2023 and was also chief of the renal section at the Cincinnati VA Medical Center from 2005 to 2024.

Dr. Thakar is internationally recognized for his research related to acute kidney injury and progression of chronic kidney disease. His research has been funded by the US Department of Veterans Affairs, the National Institutes of Health, and other research foundations. His work has been published in top peer-reviewed journals in nephrology and more general medicine.

He currently serves the National Kidney Foundation on its Scientific Advisory Board and as editor-in-chief of *Advances in Kidney Disease and Health*. He serves on the Scientific Advisory Boards of several other organizations, including the Northern Ireland Kidney Research Fund, Fresenius NxStage critical care, and the Dialysis Clinic, Inc. He served as a member of the ASN Acute Kidney Injury Advisory Group.

Dr. Thakar received his medical degree from Bharati Vidyapeeth Medical College, University of Pune, India. He then completed a residency in internal medicine at Yale University, followed by a clinical and research fellowship in nephrology at the Cleveland Clinic Foundation. He also completed an executive leadership program in managing health care delivery at Harvard Business School.

Distinguished Researcher Award

Michael T. Eadon, MD, FASN

Dr. Eadon is associate professor of medicine, associate professor of medical and molecular genetics, and the David M. and Julie B. DeWitt Scholar in Nephrology Research at the Indiana University School of Medicine in Indianapolis. He is a translational physician-scientist with accomplishments in the realms of pharmacogenomic discovery, construction of a molecular atlas of the kidney in health and disease, and implementation of pharmacogenomics and genomics in chronic kidney disease.

Dr. Eadon's research focuses primarily on the implementation of pharmacogenomics into clinical practice as well as the identification of novel predictors of renal injury from large genomic, transcriptomic, and proteomic data sets. A major focus is the evaluation of renal disease expression patterns with spatial transcriptomics and understanding the genetic variants that affect this expression.

He is an active member of the National Institute of Diabetes and Digestive and Kidney Disease's Kidney Precision Medicine Project, integrating diverse orthogonal data sets to characterize molecular patterns of kidney diseases in individuals who have had a kidney biopsy. A second focus of his research is translating genetic determinants of an antihypertensive drug response and the progression of kidney diseases into clinical practice. He is also an active contributor to the National Human Genome Research Institute's Implementing Genomics in Practice Consortium. He also serves on the ASN Publications Committee. Dr. Eadon is a practicing clinician at Eskenazi Health, Indiana University Health, and the Richard L. Roudebush Veterans' Administration Medical Center.

He received his MD from Rush University, followed by an internal medicine residency at Baylor College of Medicine and a combined research fellowship in nephrology, glomerulonephritis, and clinical pharmacology at The University of Chicago.

PLENARY SESSION

Distinguished Researcher Award

Mara McAdams DeMarco, PhD

Dr. McAdams DeMarco is an epidemiologist and associate professor at the New York University Grossman School of Medicine in New York City. She leads the Epidemiology of Aging in End Stage Kidney Disease laboratory and co-leads the NYU Kidney Disease and Health Community Advisory Board as well as the Kidney Disease and Aging Research Collaborative.

Her research focuses on the intersection of aging and kidney failure with a particular emphasis on older candidates and recipients undergoing kidney transplant. She conducted some of the first studies of frailty, delirium, cognitive function, and Alzheimer disease among older patients undergoing kidney transplant.

Dr. McAdams DeMarco is the principal investigator of the oldest and largest cohort study of frailty among kidney transplant candidates and recipients and of clinical trials of exercise interventions to prevent cognitive aging. Her studies have explored how novel aging metrics (such as frailty, cognitive function, physical function, and quality of life) can help improve risk prediction of adverse outcomes in older recipients of kidney transplant and identify interventions to prevent adverse outcomes of aging. Her overarching career objectives are to improve the care and well-being of older patients with kidney failure. Among many honors, she has received two junior investigator awards from the American Society of Transplant Surgeons.

Dr. McAdams DeMarco received an MS in epidemiology from the Harvard T.H. Chan School of Public Health and a PhD in epidemiology from the Johns Hopkins Bloomberg School of Public Health.

Distinguished Researcher Award

Vishal Patel, MD

Dr. Patel holds the Yin Quan-Yuen Distinguished Professorship in Nephrology in the Department of Internal Medicine at The University of Texas Southwestern Medical Center in Dallas.

Dr. Patel's research focuses on mRNA translation regulation, RNA chemical modifications, and noncoding RNAs in the context of polycystic kidney disease (PKD) and kidney development. His laboratory provided the first evidence that microRNAs directly inhibit PKD1 (the gene mutated in PKD) and thereby promote PKD pathogenesis. The team devised an endogenous, mutation-agnostic PKD1/2 mRNA therapy for autosomal-dominant PKD. This drug is now in clinical trials, marking a major advance in the field. In addition, his laboratory has identified many other microRNAs that may play a pathogenic role in PKD.

The National Institutes of Health (NIH) has funded Dr. Patel's laboratory continuously since 2009, with the Department of Defense providing additional funding. The research has resulted in several patents and has been published in prestigious journals such as the *Proceedings of the National Academy of Sciences USA*, *Nature Communications*, and *Cell Metabolism*.

Dr. Patel is active in several national leadership positions. He serves as a member and chair of several NIH and Department of Defense grant study sections. He chairs the Scientific Advisory Committee and is a member of the board of directors of the PKD Foundation. He serves as a consultant for many pharmaceutical companies.

Dr. Patel obtained his medical degree from Pramukhswami Medical College in Karamsad, India. He completed his internal medicine residency at Northwestern University and a clinical nephrology fellowship at UT Southwestern. Following his clinical training, he was an NIH-funded physician-scientist trainee at UT Southwestern, studying kidney genetics and primary cilia biology.

Wolters Kluwer

ASN
American Society of Nephrology

Discover the Best of ASN Journals at Kidney Week 2024!

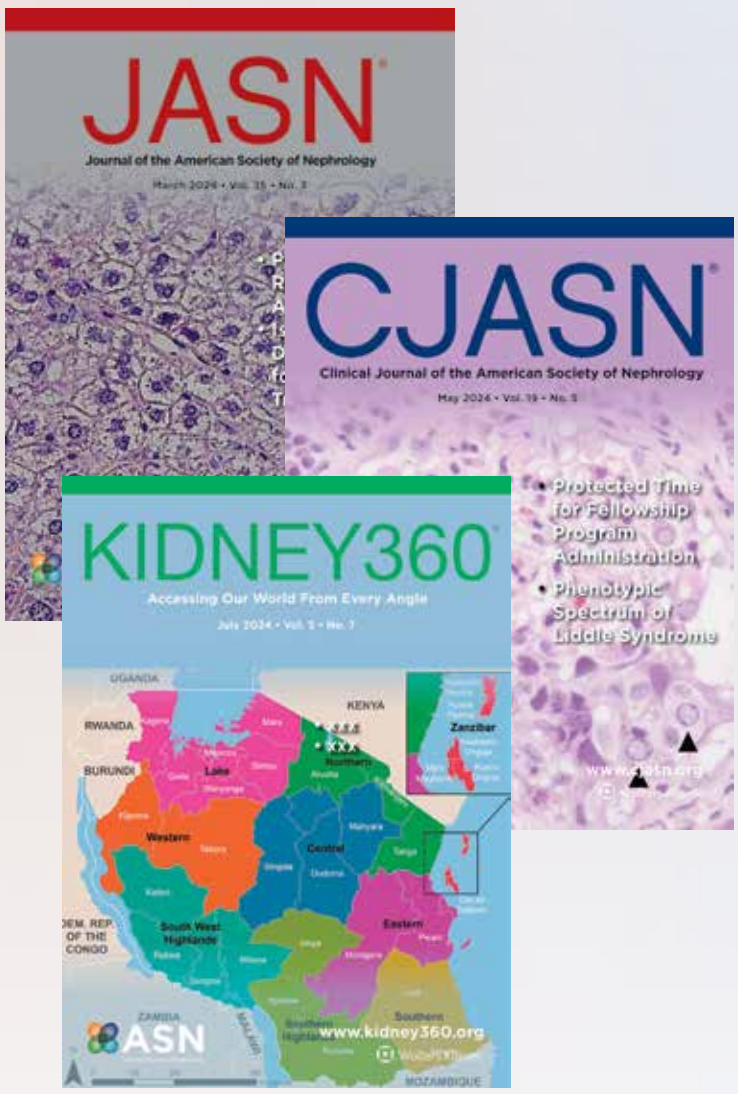
Join us for an unmissable session.

What to Expect:

Four editorial board members from ASN journals will showcase a selection of the high impact articles published over the past year. The session will cover key thematic areas, with the authors on hand to answer your questions.

Why Attend?

- **Summarize Key Advances:**
  Get a comprehensive overview of the high-impact research featured in ASN journals.
- **Explore Therapeutic Breakthroughs:**
  Learn about significant therapeutic advancements for kidney diseases.
- **Understand Mechanistic Insights:**
  Gain a deeper understanding of mechanistic studies that inform kidney disease treatment.



Thematic Areas:

AKI and Intensive Care Unit Nephrology, Glomerular Diseases, Therapeutic Advances in CKD, and Care of Patients Undergoing Long-Term Dialysis.

Don't miss this opportunity to engage with leading experts and enhance your knowledge of cutting-edge research in nephrology.

Mark your calendar and be there!

Date: October 25, 2024
Time: 2 PM
Location: Ballroom 20B/C

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In Memoriam

Investigators Who Revealed Key Aspects of Kidney Physiology, Drove Clinical Advances, Led Major Institutions

Among professionals in the kidney community who have passed away in 2023 and 2024, the contributions of several leaders in nephrology are acknowledged here and will be honored at Kidney Week 2024. George L. Bakris, MD, a creative force with lasting contributions to hypertension care; Barry M. Brenner, MD, a visionary leader whose groundbreaking research changed modern understanding of kidney disease progression; William L. Henrich, MD, FASN, a compassionate clinician who improved dialysis training and kidney care; Nicolaos E. Madias, MD, a charismatic leader with vast expertise in acid-base physiology; Stephen C. Textor, MD, a generous mentor and pioneer in the field of hypertension; and Charles Spurgeon Wingo, MD, a renowned scientist whose pivotal discoveries influenced kidney care, have all been seminal figures in the field.

George L. Bakris, MD

As an "out-of-the-box" thinker, George L. Bakris, MD, made major contributions to kidney and hypertension care. Among the most recent was a landmark study in *The New England Journal of Medicine* demonstrating that semaglutide reduces poor kidney outcomes in patients with type 2 diabetes and chronic kidney disease (1).

Bakris emigrated from Greece to the United States with his parents when he was 6 weeks old (2). He grew up in a Greek Orthodox family in South Bend, IN. He studied medicine at the National and Kapodistrian University of Athens and at the Rosalind Franklin University of Medicine and Science in North Chicago, IL, before completing his residency and an internal medicine and research fellowship at the Mayo Clinic. He then completed a nephrology and clinical pharmacology fellowship at The University of Chicago.

He spent 3 years as the director of renal research at Tulane University's Ochsner Medical Center in New Orleans, LA, before joining the faculty at Rush University in Chicago in 1993. At Rush University, he served as vice chair of the Department of Preventive Medicine and director of the Hypertension/Clinical Research Center. He later became the director of the Comprehensive Hypertension Center at The University of Chicago, one of the American Heart Association's centers of excellence in hypertension care.

"His impact was enormous as it related to improving the lives of those individuals with difficult-to-control blood pressure and secondary hypertension," said longtime colleague Arlene Chapman, MD, professor of medicine and chief of the Section of Nephrology at The University of Chicago, in a statement (3). "During his tenure of training fellows in hypertension, he was able to provide the United States with the next generation of hypertension specialists. His legacy will be felt for a very long time."

Throughout his career, Bakris authored hundreds of publications. They helped lead major clinical trials for blood pressure targets, renin-angiotensin system blockers, sodium-glucose transporter-2 inhibitors, finerenone, and glucagon-like peptide-1 receptor agonists. He helped author many national and international guidelines on hypertension and kidney diseases. He was honored with several national and international awards and honorary doctorates from the Universities of Athens [National and Kapodistrian University of Athens] and Tessaloniki [Aristotle University of Thessaloniki] (4). Bakris was equally devoted to his family and effortlessly balanced the two. He also loved music, comedy, travel, global cuisine, wine, and cooking.

"He will live on through his great work," said Michael Davidson, MD, clinical professor of medicine and director of The University of Chicago Medicine's Lipid (Cholesterol) Clinic, in a statement (3). "I will remember him most as a great friend and colleague who was always eager to help with his amazing mind."

Barry M. Brenner, MD

At ASN's first national meeting in 1967, Barry M. Brenner, MD, and his colleagues at the National Institutes of Health's Laboratory of Kidney and Electrolyte Metabolism made waves by debunking the existing paradigm for resorption by the renal proximal tubules (5). He and colleague Julia Troy would later start a laboratory at the San Francisco Veterans Affairs Medical Center, CA, that would use pioneering techniques to elucidate the mechanisms of glomerular filtration.

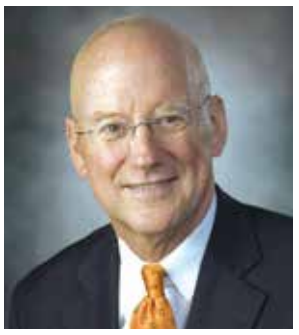
It was just the start of Brenner's illustrious and productive career, which included authoring more than 730 publications and editing or coediting 49 books, including *The Kidney*. Beyond detailing normal glomerular function, Brenner studied the consequences of renal nephron loss in kidney diseases and renal-protective medications and identified reduced renal endowment at birth and how it contributes to an increased vulnerability to kidney diseases later in life. According to colleagues and family members, his tireless work ethic, creativity, compassion, and mentorship leave a lasting legacy in the field.

"Barry was a giant in the field, known and respected nationally and internationally," said Joseph V. Bonventre, MD, PhD, FASN, the Constantine L. Hampers, MD, Distinguished Chair in Renal Medicine at the Brigham and Women's Hospital, Harvard Medical School. "His contributions are enduring and his influence profound."

In addition to his research in San Francisco, Brenner was the founding chief of nephrology at the hospital from 1969 until 1976. In 1976, he joined the faculty at Brigham and Women's Hospital, Boston, MA, where he served in many roles, including director of the Laboratory of Kidney and Electrolyte Physiology, director and director emeritus of the Renal Division, director of the Physician-Scientist program, codirector of the joint postdoctoral Fellowship Training Program in Nephrology, and director emeritus of the Samuel A. Levine Distinguished Professor of Medicine at Harvard Medical School. He also served as an ASN councilor and as the organization's president from 1986 to 1987. Kidney Week recognizes his contributions through the Barry M. Brenner, MD, Endowed Lectureship.

Beyond his professional accomplishments, he was a loving husband, supportive father, and encouraging grandfather. Brenner was a devoted Boston Red Sox fan, and he shared his passion for sports cars, wine, and art as well as his profession with many in his orbit, including his son, Robert M. Brenner, MD, chief medical officer at Vera Therapeutics.

Robert said his father would have liked his career to serve as inspiration for the next generation of talented physician-scientists. "How do you make a real impact?" Robert asked. "It begins at the bench. Developing a detailed understanding of the principles that underlie our clinical observations and carrying that all the way forward with mechanistically sound interventions interrogated in well-designed trials, with the potential to ultimately evolve the practice of medicine. Then, generating the next set of important questions based on clinical experience, and going back to the bench with those questions in hand."



William L. Henrich, MD, FASN

William L. Henrich, MD, FASN, was the founding editor of *Henrich's Principles and Practice of Dialysis*, a seminal textbook on dialysis that helped train generations of nephrologists, and was a transformative leader at The University of Texas Health Science Center (UT Health) at San Antonio.

Henrich, the president of UT Health at San Antonio, died from complications after a second round of stem cell transplantation to treat cancer, according to a statement from the institution (6). He earned a medical degree at Baylor College of Medicine in Houston, TX, and completed his internship and internal medicine residency at the University of Oregon and his fellowship in nephrology at the University of Colorado School of Medicine. Heinrich took on increasingly greater leadership roles as he moved from The University of Texas Southwestern Medical Center's Medical School, where he rose to the rank of professor, before becoming chair of medicine at the Medical College of Ohio (now The University of Toledo) and later chair of medicine at the University of Maryland.

"His ability to make everyone he interacted with feel truly seen and heard was remarkable," said Donna L. Parker, MD, professor and senior associate dean for undergraduate medical education at the University of Maryland, in a statement (7). "His grace under pressure kept those around him calm and focused under the most challenging circumstances."

In 2006, he was appointed dean of the Joe R. & Teresa Lozano Long School of Medicine, UT Health at San Antonio. He served as president of UT Health at San Antonio from 2009 to 2024. Under Henrich's leadership, UT Health at San Antonio underwent a major expansion. He helped grow the institution's multispecialty practices, oversaw building $1 billion in new facilities, and doubled its sponsored research funding from $194.5 to $413 million. He also helped launch the university's newest school, the School of Public Health.

"His integrity, energy, intelligence, humor, and compassion made him one of the finest leaders I have known," said UT System Chancellor James B. Milliken in a statement (6). "I learned from him constantly, and I'm grateful for his friendship over the years. The UT System and the state of Texas are so fortunate to have had Bill Henrich share his many talents and enrich our institutions and lives."

He also left a lasting impact on the field of nephrology as the founding editor of his namesake's textbook on dialysis, published more than 300 articles, and was one of UpToDate's first writers. Henrich served as ASN president from 2006 to 2007. He was dedicated to his family, ran almost every day, and loved to share funny stories or lament the misadventures of his favorite teams: the Spurs, Cowboys, and Rangers (8).

Nicolaos E. Madias, MD

Nicolaos E. Madias, MD, a beloved and charismatic leader at Tufts University School of Medicine and Tufts Medical Center in Boston, MA, for more than 3 decades made seminal contributions to the field's understanding of acid-base physiology.

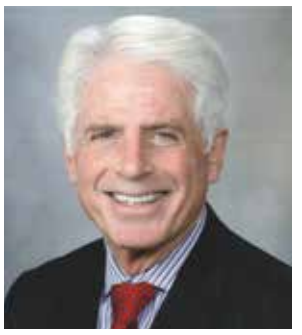
Throughout his career, Madias contributed to over 250 publications, books, or reviews and mentored more than 75 fellows trained in the United States or Greece (9). Madias was born and raised in Greece and received his medical degree at the National and Kapodistrian Athens University School of Medicine in 1968 and completed his internship and residency at Mass General Brigham Healthcare Center (Waltham) and St. Elizabeth Medical Center in Massachusetts. He joined the Division of Nephrology at Tufts Medical Center in 1973 as a fellow, joined the faculty of Tufts University School of Medicine as an assistant professor of medicine in 1976, and later became a professor in 1988. He served as chief of the Division of Nephrology at Tufts Medical Center between 1982 and 1999. He was an executive academic dean at Tufts University School of Medicine from 1999 to 2004 and served as the school's interim dean in 2003. In 2004, he became the chairman of the Department of Medicine at St. Elizabeth's Medical Center, one of Tufts University's teaching hospitals, and served in that role until 2015. He also served as Tufts University School of Medicine's academic dean at St. Elizabeth's Medical Center from 2010–2014.

"A gifted, dedicated mentor, Dr. Nicolaos Madias served Tufts University and Tufts Medicine with great distinction and had a significant impact on Tufts University School of Medicine students, scholarship, and his profession," said Helen Boucher, MD, dean and professor of medicine at Tufts University School of Medicine and chief academic officer at Tufts Medicine. "He will be deeply missed by those who knew him, and his excellence in teaching, exemplary leadership, and contributions to the scientific and medical communities will not soon be forgotten."

Madias' research focused on acid-base and electrolyte physiology and the treatment of hypertensive disorders. He served as a member of the American Board of Internal Medicine's boards of internal medicine and nephrology and was an established investigator for the American Heart Association. ASN is offering a course on acid-base, fluid, and electrolyte balance disorders as part of the Kidney Week program to honor Madias (10).

"He was a brilliant clinician, scientist, speaker, [and] writer," said Mark Sarnak, MD, MS, chief of nephrology at Tufts Medical Center. "But on top of all of that, he was a real gentleman, a very approachable person who cared for everyone in the division."

Madias was also a devoted father and husband and enjoyed sharing humorous stories. He was committed to his Greek Orthodox faith. He was recognized for his contributions to Greek American culture with the Freedom Award from The Federation of Hellenic-American Societies of New England in 2022 (9). He maintained close ties with his colleagues from Greece and was awarded honorary doctorates from the National and Kapodistrian University of Athens, the Aristotle University of Thessaloniki, and the Universities of Patras and Ioannina. Madias also cofounded and served as an editor for the Nephrology Forum of *Kidney International*.



Stephen C. Textor, MD

Internationally recognized hypertension expert Stephen C. Textor, MD, used imaging techniques to reveal the role of hypoxia in ischemic nephropathy.

Textor received his MD from the University of California, Los Angeles, David Geffen School of Medicine in 1973 before completing his residency and nephrology fellowship at Boston University, MA, in 1978. He then served 2 years as an international fellow of the Swiss National Science Foundation Swiss Postdoctoral Fellowships at the Lausanne University Hospital in Switzerland. He later worked at the Cleveland Clinic; City of Hope Hospital in Duarte, CA; and the University of California, Irvine. The Mayo Clinic recruited Textor to join the Division of Nephrology and Hypertension in 1988. He remained at the Mayo Clinic until his retirement. During his tenure there, he served as a professor and was vice chair of the Division of Nephrology and Hypertension for 4 years.

Throughout his career, Textor studied renal hypertension, kidney transplantation, and antihypertensive therapies while caring for patients (11). He became adept at advanced imaging techniques and used them to study renal artery stenosis and renovascular hypertension.

"[Textor] was committed to the care of [patients with hypertension] and left a legacy of innovative strategies that he pioneered to treat patients with renovascular disease," said Lilach O. Lerman, MD, PhD, FASN, director of the Renovascular Disease Laboratory. Lerman explained that Textor developed a stem cell therapy for renal artery stenosis and led the first trial for the intervention. He also spearheaded a clinical trial of a mitochondria-targeting therapy for ischemia-reperfusion injury.

At ASN, Textor served as section editor for the Nephrology Self-Assessment Program and a member of the Hypertension Advisory group. He was also active in the American Heart Association's Council on Hypertension and the American Society of Hypertension. Textor held a pilot's license and enjoyed flying and adventurous outdoor pursuits, including white-water kayaking, canoeing, helicopter skiing, backpacking, and rock climbing.

Charles Spurgeon Wingo, MD

During his 42-year research career, Charles Spurgeon Wingo, MD, became a go-to source for fluid and electrolyte research for his colleagues and trainees at the University of Florida, Gainesville. "Charles was a true triple threat as a knowledgeable and compassionate clinician, the consummate biomedical scientist, and an outstanding teacher and mentor," said David W. Ploth, MD, distinguished university professor at the Medical University of South Carolina in Charleston, in an emailed statement.

Wingo joined the faculty at the University of Florida in 1981 after completing his internship and residency at The University of Texas McGovern Medical School in Houston and a fellowship at The University of Texas Southwestern Medical Center in Dallas. In addition to his research, he provided clinical care at the University of Florida and Veterans Affairs hospitals in Gainesville. During his tenure, he mentored many trainees who have gone on to successful careers.

He made pivotal discoveries on potassium homeostasis in the kidney and detailed the role of H-K-ATPase proteins in the kidney. His laboratory also studied the role of circadian control of sodium channels in the kidney and how it affected blood pressure. Most recently, he documented new information about the role of aldosterone in regulating blood pressure (12). A session honoring Wingo at Kidney Week will focus on potassium stress and tubular injury (13).

Wingo was also a leader in the Southern Society for Clinical Investigation, serving as a fellow, councilor, and president. The organization awarded him its highest honor, the Founder's Award. He was also devoted to his family and was an adventurous world traveler, eager to try new foods and experiences. ■

Continued on page 36 >

In Memoriam

Continued from page 35

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In your adult patients with **SLE with active kidney involvement,** lupus nephritis (LN)[1]

IS *Her* PROTEINURIA AT THERAPEUTIC TARGETS?

Guidelines recommend **therapeutic targets for reductions in proteinuria and renal response**[2]

Recommended therapeutic targets[2a]

- Reduce proteinuria by **≥25%** by 3 months and **≥50%** by 6 months
- Reduce urine protein to **<0.5-0.7 g/day** by 12-24 months
- Taper steroids to **≤5 mg/day** as quickly as possible
- Management should be continued once complete response is achieved for **at least 3 years**

Not an actual patient.

Lupkynis® (voclosporin) capsules 7.9 mg

See how intervention with a multitarget treatment regimen (e.g., LUPKYNIS + MMF + steroids) can help treat to recommended therapeutic targets.[1-4b]

[a]All with eGFR within 10% from baseline.[2]
[b]EULAR recommendations for the management of systemic lupus erythematosus: 2023 update.[2]

eGFR=estimated glomerular filtration rate; MMF=mycophenolate mofetil; SLE=systemic lupus erythematosus.

INDICATION

LUPKYNIS is indicated in combination with a background immunosuppressive therapy regimen for the treatment of adult patients with active lupus nephritis (LN).

Limitations of Use: Safety and efficacy of LUPKYNIS have not been established in combination with cyclophosphamide. Use of LUPKYNIS is not recommended in this situation.

IMPORTANT SAFETY INFORMATION

BOXED WARNINGS: MALIGNANCIES AND SERIOUS INFECTIONS

Increased risk for developing malignancies and serious infections with LUPKYNIS or other immunosuppressants that may lead to hospitalization or death.

Please see full Prescribing Information including Boxed Warning and Medication Guide about LUPKYNIS at LUPKYNISpro.com, Important Safety Information throughout, and Brief Summary on adjacent pages.

Lupkynis®
(voclosporin) capsules 7.9 mg

Actual patients on LUPKYNIS.

Not actual patients.

DEFEND HER

FROM THE THREATS OF LUPUS NEPHRITIS (LN)

Help Protect Her Kidney Function for Tomorrow by Rapidly Reducing Her Proteinuria Today[a]

[a]AURORA 1 was a 52-week randomized, double-blind, placebo-controlled, phase 3 trial of 357 patients evaluating MMF + low-dose steroids with or without the addition of LUPKYNIS in adults with active lupus nephritis. Patients in the LUPKYNIS arm were more likely to achieve a complete renal response at 52 weeks (40.8% LUPKYNIS arm vs. 22.5% active control arm; OR: 2.7, 95% CI: 1.6-4.3, *p*-value <0.001), UPCR ≤0.5 mg/mg at 52 weeks (45.3% LUPKYNIS arm vs. 23.0% active control arm; OR: 3.1, 95% CI: 1.9-5.0), as well as complete renal response at 24 weeks (32.4% LUPKYNIS arm vs. 19.7% active control arm; OR: 2.2; 95% CI: 1.3, 3.7). Time to UPCR of ≤0.5mg/mg was shorter in the LUPKYNIS arm than the active control arm (median time of 169 days vs. 372 days, respectively; HR: 2.0; 95% CI: 1.5, 2.7). Time to achieve a 50% reduction in UPCR was shorter in the LUPKYNIS arm than the active control arm (median time of 29 days vs. 63 days, respectively; HR: 2.1, 95% CI:1.6, 2.6).[1]

INDICATION

LUPKYNIS is indicated in combination with a background immunosuppressive therapy regimen for the treatment of adult patients with active lupus nephritis (LN).

Limitations of Use: Safety and efficacy of LUPKYNIS have not been established in combination with cyclophosphamide. Use of LUPKYNIS is not recommended in this situation.

IMPORTANT SAFETY INFORMATION

BOXED WARNINGS: MALIGNANCIES AND SERIOUS INFECTIONS

Increased risk for developing malignancies and serious infections with LUPKYNIS or other immunosuppressants that may lead to hospitalization or death.

CONTRAINDICATIONS: LUPKYNIS is contraindicated in patients taking strong CYP3A4 inhibitors because of the increased risk of acute and/or chronic nephrotoxicity, and in patients who have had a serious/severe hypersensitivity reaction to LUPKYNIS or its excipients.

LUPKYNIS Can Help Your Patients Achieve Therapeutic Targets[1-4]

More patients achieved a complete renal response[1,3b]

- 2.7x more likely to achieve a complete renal response within 1 year compared with active control (40.8% of patients vs. 22.5% of patients; *P*<0.001; OR: 2,7; 95% CI: 1.6-4.3)[1,3c]

Rapid reductions in proteinuria[3b]

- UPCR ≤0.5 mg/mg by 169 days (median) vs. 372 days with active control (HR: 2.0; 95% CI: 1.5-2.7)[3c]
- 50% decrease in UPCR by 29 days (median) vs. 63 days with active control[3d]

Preserved renal function over 2-year extension period (3 year total)[4e]

- Stable eGFR over 2-year extension period (annualized slope was -0.2 mLmin/1.73 m^2 vs. -5.4 mL/min/1.73 m^2 with active control)[4]

IMPORTANT NOTES: This is a post hoc analysis and should be interpreted with caution. eGFR should be monitored periodically. Please see Brief Summary on adjacent pages for more information.

Endpoints were achieved with a steroid-sparing protocol, targeting ≤2.5 mg/day.[4]

[b]Stringent criteria for complete renal response included: UPCR ≤0.5 mg/mg; eGFR ≥60 mL/min/1.73 m^2 or no confirmed decrease from baseline in eGFR >20% or no treatment- or disease-related eGFR-associated event; sustained low-dose steroids; No administration of rescue medications.[1]
[c]Primary endpoint for AURORA 1.[3]
[d]Secondary endpoint for AURORA 1.[4]
[e]AURORA 2 was a phase 3, double-blind, placebo-controlled, 24-month continuation study that enrolled 216 (84.6%) patients who completed 12 months of treatment in AURORA 1; 116 (64.8%) patients were enrolled in the LUPKYNIS arm, and 100 (56.2%) patients were enrolled in the active control arm. In AURORA 2, the proportion of patients experiencing adverse events was comparable between groups (86.2% vs. 80.0% in the LUPKYNIS and active control arms, respectively); the overall adverse event profile seen in AURORA 2 was similar to AURORA 1. Patients in the LUPKYNIS arm were more likely to achieve a complete renal response at nearly every timepoint. At Month 36, the end of the extension study, more patients in the LUPKYNIS arm than in the active control arm achieved a complete renal response (50.9% vs. 39.0%, respectively; OR 1.74; 95% CI 1.00-3.03).[4]

CI=confidence interval; eGFR=estimated glomerular filtration rate; HR=hazard ratio; MMF=mycophenolate mofetil; OR=odds ratio; UPCR=urine protein-to-creatinine ratio.

LUPKYNIS Can Help Exceed Guideline Goals With Rapid and Sustained Reduction of Proteinuria and Preserved Renal Function[1-4]

GET*Started* Learn how to get your patients started at **LUPKYNISpro.com**

IMPORTANT SAFETY INFORMATION (cont.)

WARNINGS AND PRECAUTIONS

Lymphoma and Other Malignancies: Immunosuppressants, including LUPKYNIS, increase the risk of developing lymphomas and other malignancies, particularly of the skin. The risk appears to be related to increasing doses and duration of immunosuppression rather than to the use of any specific agent.

Serious Infections: Immunosuppressants, including LUPKYNIS, increase the risk of developing bacterial, viral, fungal, and protozoal infections, including opportunistic infections which lead to serious, including fatal outcomes.

Nephrotoxicity: LUPKYNIS, like other calcineurin inhibitors (CNIs), may cause acute and/or chronic nephrotoxicity. The risk is increased when CNIs are concomitantly administered with drugs associated with nephrotoxicity. Monitor eGFR regularly.

Please see full Prescribing Information including Boxed Warning and Medication Guide about LUPKYNIS at LUPKYNISpro.com, Important Safety Information throughout, and Brief Summary on adjacent pages.

References
1. LUPKYNIS® (voclosporin) Package Insert. Aurinia Pharma, U.S., Inc; 04/24. **2.** Fanouriakis A, et al. *Ann Rheum Dis.* 2024;83(1):15-29. **3.** Rovin BH, et al. [Erratum in Lancet. 2021;397(10289):2048.] *Lancet.* 2021;397(10289):2070-2080. **4.** Saxena A, et al. *Arthritis Rheumatol.* 2024;76(1):59-6.

Lupkynis® (voclosporin) capsules 7.9 mg

LUPKYNIS® (voclosporin) capsules, BRIEF SUMMARY
SEE PACKAGE INSERT FOR FULL PRESCRIBING INFORMATION

WARNING: MALIGNANCIES and SERIOUS INFECTIONS

Increased risk for developing malignancies and serious infections with LUPKYNIS or other immunosuppressants that may lead to hospitalization or death.

INDICATION AND USAGE

LUPKYNIS is indicated in combination with a background immunosuppressive therapy regimen for the treatment of adult patients with active lupus nephritis (LN).

Limitations of Use: Safety and efficacy of LUPKYNIS have not been established in combination with cyclophosphamide. Use of LUPKYNIS is not recommended in this situation.

CONTRAINDICATIONS

LUPKYNIS is contraindicated in patients concomitantly using strong CYP3A4 inhibitors (e.g., ketoconazole, itraconazole, clarithromycin) because these medications can significantly increase exposure to LUPKYNIS which may increase the risk of acute and/or chronic nephrotoxicity and also in patients who have had a serious/severe hypersensitivity reaction to LUPKYNIS or its excipients.

WARNINGS AND PRECAUTIONS

Lymphoma and Other Malignancies: Immunosuppressants, including LUPKYNIS, increase the risk of developing lymphomas and other malignancies, particularly of the skin. The risk appears to be related to the intensity and duration of immunosuppression rather than to the use of any specific agent. Examine patients for skin changes and advise to avoid or limit sun exposure and to avoid artificial UV light (tanning beds, sun lamps) by wearing protective clothing and using broad spectrum sunscreen with a high protection factor (SPF 30 or higher).

Serious Infections: Immunosuppressants, including LUPKYNIS, increase the risk of developing bacterial, viral, fungal, and protozoal infections, including opportunistic infections. These infections may lead to serious, including fatal, outcomes. Viral infections reported include cytomegalovirus and herpes zoster infections. Monitor for the development of infection. Consider the benefits and risks for the individual patient; use the lowest effective dose needed to maintain response.

Nephrotoxicity: LUPKYNIS, like other calcineurin inhibitors (CNIs), can cause acute and/or chronic nephrotoxicity. Monitor eGFR regularly during treatment, and consider dose reduction or discontinuation in patients with decreases in eGFR from baseline; persistent decrease in eGFR should be evaluated for chronic CNI nephrotoxicity. Consider the risks and benefits of LUPKYNIS treatment in light of the patient's treatment response and risk of worsening nephrotoxicity, including co-administration with drugs associated with nephrotoxicity. The risk for acute and/or chronic nephrotoxicity is increased when LUPKYNIS is concomitantly administered with drugs associated with nephrotoxicity.

Hypertension: Hypertension is a common adverse reaction of LUPKYNIS therapy and may require antihypertensive therapy. Some antihypertensive drugs can increase the risk for hyperkalemia. Certain calcium-channel blocking agents (verapamil and diltiazem) may increase voclosporin blood concentrations and require dosage reduction of LUPKYNIS. Monitor blood pressure regularly during treatment and treat new-onset hypertension and exacerbations of pre-existing hypertension. If a patient experience increases in blood pressure that cannot be managed with dose reduction of LUPKYNIS or other appropriate medical intervention, consider discontinuation of LUPKYNIS.

Neurotoxicity: LUPKYNIS, like other CNIs, may cause a spectrum of neurotoxicities. The most severe include posterior reversible encephalopathy syndrome (PRES), delirium, seizure, and coma; others include tremor, paresthesias, headache, mental status changes, and changes in motor and sensory functions. Monitor for neurologic symptoms and consider dosage reduction or discontinuation of LUPKYNIS if neurotoxicity occurs.

Hyperkalemia: Hyperkalemia, which may be serious and require treatment, has been reported with CNIs including LUPKYNIS. Concomitant use of agents associated with hyperkalemia (e.g., potassium-sparing diuretics, ACE inhibitors, angiotensin receptor blockers) may increase the risk for hyperkalemia. Monitor serum potassium levels periodically during treatment.

QTc Prolongation: LUPKYNIS prolongs the QTc interval in a dose-dependent manner after single dose administration at a dose higher than the recommended lupus nephritis therapeutic dose. The use of LUPKYNIS in combination with other drugs that are known to prolong QTc may result in clinically significant QT prolongation. Certain circumstances may increase the risk of the occurrence of torsade de pointes and/or sudden death in association with the use of drugs that prolong the QTc interval, including (1) bradycardia; (2) hypokalemia or hypomagnesemia; (3) concomitant use of other drugs that prolong the QTc interval; and (4) presence of congenital prolongation of the QT interval.

Immunizations: Avoid the use of live attenuated vaccines during treatment with LUPKYNIS (e.g., intranasal influenza, measles, mumps, rubella, oral polio, BCG, yellow fever, varicella, and TY21a typhoid vaccines). Inactivated vaccines noted to be safe for administration may not be sufficiently immunogenic during treatment with LUPKYNIS.

Pure Red Cell Aplasia: Cases of pure red cell aplasia (PRCA) have been reported in patients treated with another CNI immunosuppressant. All of these patients reported risk factors for PRCA such as parvovirus B19 infection, underlying disease, or concomitant medications associated with PRCA. A mechanism for CNI-induced PRCA has not been elucidated. If PRCA is diagnosed, consider discontinuation of LUPKYNIS.

ADVERSE REACTIONS

Adverse Reactions in ≥3% of Patients Treated with LUPKYNIS 23.7 mg Twice a Day and ≥2% Higher than Placebo in Studies 1 and 2

Adverse Reaction	LUPKYNIS 23.7 mg twice a day (n=267)	Placebo (n=266)
Glomerular filtration rate decreased	26%	9%
Hypertension	19%	9%
Diarrhea	19%	13%
Headache	15%	8%
Anemia	12%	6%
Cough	11%	2%
Urinary tract infection	10%	6%
Abdominal pain upper	7%	2%
Dyspepsia	6%	3%
Alopecia	6%	3%
Renal impairment	6%	3%
Abdominal pain	5%	2%
Mouth ulceration	4%	1%
Fatigue	4%	1%
Tremor	3%	1%
Acute kidney injury	3%	1%
Decreased appetite	3%	1%

DRUG INTERACTIONS

Effect of Other Drugs on LUPKYNIS

Strong and Moderate CYP3A4 Inhibitors

Voclosporin is a sensitive CYP3A4 substrate. Co-administration with strong or moderate CYP3A4 inhibitors increases voclosporin exposure, which may increase the risk of LUPKYNIS adverse reactions. Co-administration of LUPKYNIS with strong CYP3A4 inhibitors (e.g., ketoconazole, itraconazole, clarithromycin) is contraindicated. Reduce LUPKYNIS dosage when co-administered with moderate CYP3A4 inhibitors (e.g., verapamil, fluconazole, diltiazem). Avoid food or drink containing grapefruit when taking LUPKYNIS.

Strong and Moderate CYP3A4 Inducers

Voclosporin is a sensitive CYP3A4 substrate. Co-administration with strong or moderate CYP3A4 inducers decreases voclosporin exposure, which may decrease the efficacy of LUPKYNIS. Avoid co-administration of LUPKYNIS with strong or moderate CYP3A4 inducers.

Effect of LUPKYNIS on Other Drugs

Certain P-gp Substrates

Voclosporin may be a P-gp inhibitor. Co-administration of voclosporin increases exposure of P-gp substrates, which may increase the risk of adverse reactions of these substrates. For certain P-gp substrates with a narrow therapeutic window, reduce the dosage of the substrate as recommended in its prescribing information, if needed.

OATP1B1 Substrates

Voclosporin is an inhibitor of OATP1B1 and OATP1B3 transporters and information suggests an increase in the concentration of these substrates is possible. Monitor for adverse reactions of OATP1B1/OATP1B3 substrates when used concomitantly with LUPKYNIS.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

Avoid use of LUPKYNIS in pregnant women due to the alcohol content of the drug formulation. The available data on the use of LUPKYNIS in pregnant patients are insufficient to determine whether there is a drug-associated risk for major birth defects, miscarriage, or adverse maternal or fetal outcomes. There are risks to the mother and fetus associated with systemic lupus erythematosus (SLE).

LUPKYNIS may be used in combination with a background immunosuppressive therapy regimen that includes mycophenolate mofetil (MMF). MMF used in pregnant women and men whose female partners are pregnant can cause fetal harm (major birth defects and miscarriage). Refer to the MMF prescribing information for more information on its use during pregnancy.

The estimated background risk of major birth defects and miscarriage for the indicated population is unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the U.S. general population, the background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2% to 4% and 15% to 20%, respectively.

Clinical Considerations

Disease-Associated Maternal and/or Embryo/Fetal Risk

Pregnant women with SLE are at increased risk of adverse pregnancy outcomes, including worsening of the underlying disease, premature birth, miscarriage, and intrauterine growth restriction. Maternal LN increases the risk of hypertension and preeclampsia/eclampsia. Passage of maternal autoantibodies across the placenta may result in adverse neonatal outcomes, including neonatal lupus and congenital heart block.

Lactation

Risk Summary

Available data from the clinical study and published scientific literature are insufficient to determine the effects of LUPKYNIS on the breastfed infant or on milk production. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for LUPKYNIS and any potential adverse effects on the breastfed infant from LUPKYNIS or from the medical condition of the mother.

Females and Males of Reproductive Potential

LUPKYNIS may be used in combination with a background immunosuppressive therapy regimen that includes MMF. If LUPKYNIS is administered with MMF, the information for MMF regarding pregnancy testing, contraception, and infertility also applies to this combination regimen. Refer to MMF prescribing information for additional information.

Pediatric Use

The safety and efficacy of LUPKYNIS in pediatric patients has not been established.

Renal Impairment

Use of LUPKYNIS is not recommended in patients with a baseline eGFR ≤45 mL/min/1.73 m^2 unless the benefit exceeds the risk. If used in patients with severe renal impairment at baseline, LUPKYNIS should be used at a reduced dose. No dosage adjustment is recommended in patients with mild or moderate renal impairment at baseline. Monitor eGFR closely. After initiating therapy, dosing adjustments should be made based on eGFR.

Hepatic Impairment

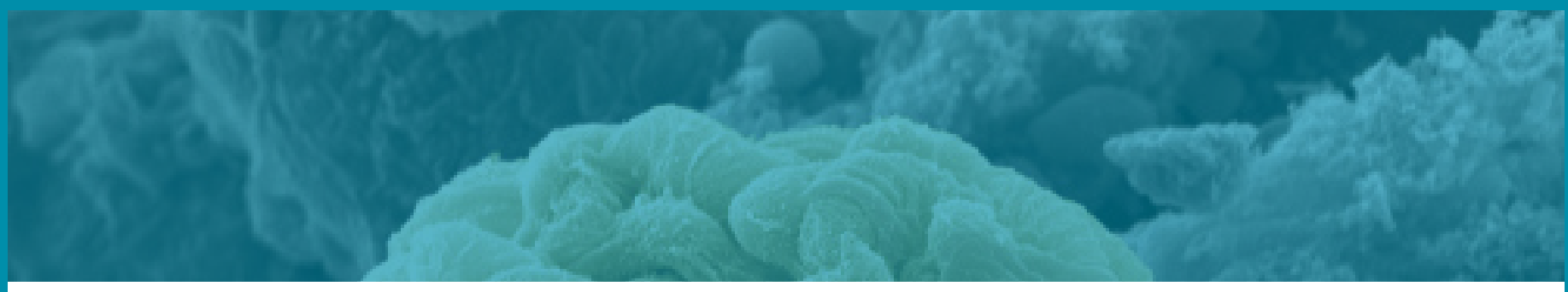
In patients with mild and moderate hepatic impairment (Child-Pugh A and Child-Pugh B), reduce the LUPKYNIS dosage. Avoid LUPKYNIS in patients with severe hepatic impairment (Child-Pugh C).

To report SUSPECTED ADVERSE REACTIONS, contact Aurinia Pharmaceuticals at 1-833-672-0028 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

This brief summary is based on LUPKYNIS Prescribing Information (FPI-0044) issued April 2024.

Aurinia™

Additional information can be found at LUPKYNISpro.com.



With support from ASN members, kidney patients, friends and family, industry partners, and leaders in the field, KidneyCure funds trailblazing investigators who are transforming the future of kidney care.

Thank you to the following donors for their generous support of KidneyCure:

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The Founders Circle recognizes companies and nonprofit organizations that have made significant contributions in support of foundation programs.

Transition to Independence Grants Program Donors

ASN American Society of Nephrology — $15,000,000

Akebia Therapeutics — $1,000,000

Otsuka | Visterra — $1,000,000

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Fresenius Medical Care — $10,000,000

ASN American Society of Nephrology — $6,500,000

American Renal Associates — $1,000,000

Amgen — $1,000,000

Baxter — $1,000,000

PKD Foundation Polycystic Kidney Disease — $500,000

Visionary Circle

The Visionary Circle recognizes individuals who have donated, pledged, or made a bequest of $75,000 or more to the foundation or its programs.

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Revitalizing ASN's Excellence in Patient Care for Clinical Nephrology Today and in the Future

By Alan S. Kliger

The American College of Physicians defines nephrology as "…the subspecialty of internal medicine that focuses on the diagnosis and treatment of diseases of the kidney. Because the kidney performs so many critical functions, nephrologists maintain expertise in primary kidney disorders, but also the management of the systemic consequences of kidney dysfunction. Although the prevention and identification and management of early kidney disease is a large part of general internal medicine practice, **nephrologists are usually called upon to assist and manage more complex or advanced nephrologic disorders**" (1). This has been the picture for decades—in which a nephrologist was consulted in later stages of chronic kidney disease or to manage more complex or advanced disease.

However, our field is undergoing a major change. With the discovery of therapies targeting specific molecular or cellular processes that contribute to disease development or progression; with the growing opportunities to alter defective genes; and with agents such as sodium-glucose cotransporter-2 (SGLT2) inhibitors, glucagon-like peptide-1 (GLP-1) agonists, and nonsteroidal mineralocorticoid receptor antagonists that prevent or ameliorate kidney diseases, nephrologists now have the opportunity to identify and intervene early in the course of many kidney diseases. Furthermore, many of these agents that are highly effective in changing the course of kidney diseases are also useful in reducing heart disease, diabetes, and possibly other diseases as well.

This complex specialty now requires new approaches to best address the vast array of circumstances, diagnoses, complications, and needs of the patient population for whom it cares.

ASN created Excellence in Patient Care (EPC) to help integrate the several clinical working groups aiming to improve patient care across the wide spectrum of nephrology care. EPC has overseen work done by AKINow [Acute Kidney Injury], the Adult Immunization Project, the Diabetic Kidney Disease Collaborative, and the Emergency Partnership Initiative. These efforts were fashioned after ASN's Nephrologists Transforming Dialysis Safety (NTDS) initiative. Launched in July 2016 as a partnership with the Centers for Disease Control and Prevention, NTDS' mission, "engaging nephrologists as team leaders in transformational change that continuously improves the safety of life sustaining dialysis," focused on infection prevention and patient safety.

In the years since 2016, the project progressed to include specific initiatives on:

- Quality, Assessment, Improvement, and Education
- Transforming Dialysis Access Together
- Human Factors
- Home Dialysis
- Project Firstline
- COVID-19 and Emerging Threats

NTDS' and EPC's library of resources includes an Ebola gap analysis; an algorithm for treatment of hepatitis B; a leadership initiative; Targeting Zero Infections webinars on topics such as environmental decontamination; leading a collaboration to respond to hurricanes in the Caribbean; learning modules on antibiotic prescribing, COVID-19, optimizing vascular access planning, patient empowerment, and mental wellness; and over 20 webinars addressing the unique issues prompted by the COVID-19 pandemic.

New tools and new opportunities for nephrologists to transform the lives of our patients now dictate that EPC and its initiatives grow and change to better meet the needs of clinicians who treat kidney diseases. The world has changed, from new viruses and therapies to the rise of misinformation and artificial intelligence. As an example, SGLT2 inhibitors and GLP-1 receptor agonists, which are proven to be highly effective therapies to reduce kidney and cardiovascular risks in diabetic kidney disease, remain vastly underutilized (2).

Now is the time to move nephrology forward once again, embrace such new therapies, implement best practices, and search for like-minded professions with goals that intersect with and support those of clinical nephrology.

The close intersection of hearts and kidneys, particularly with new SGLT2 and GLP-1 therapies that link them, suggests that now is the time to welcome cardiology as a partner in improving the lives of patients who experience multiple and complicated comorbidities.

Excellence in Patient Care

- Acute Kidney Injury (AKINow)
- Adult Immunization Project (AIP)
- COVID-19 and Emerging Threats (C-ET)
- Diabetic Kidney Disease Collaborative (DKD-C)
- eGFR Toolkit
- Emergency Partnership Initiative (EPI)
- Fostering Inclusivity in Dialysis (FinD)
- Glomerular Diseases Collaborative (GD-C)
- Home Dialysis Project (HDP)
- Human Factors Engineering (HFE)
- Kidney Community Vaccination Collaborative (KCVC)
- Nephrologists Transforming Dialysis Safety (NTDS)
- Project Firstline
- Quality, Assessment, Improvement, and Education (QAIE)
- Transforming Dialysis Access Together (TDAT)

ASN is thrilled to announce that it is redesigning EPC to embrace the new and complex issues that nephrology care teams address on a daily basis. This redesign will unite NTDS and EPC with new initiatives, including Fostering Inclusivity in Dialysis, the development of an eGFR [Estimated Glomerular Filtration Rate] Toolkit, the Kidney Community Vaccination Collaborative, and a Glomerular Diseases Collaborative. Similarly, EPC will pursue collaborations to support collective efforts to save hearts and kidneys.

This collective of clinically focused initiatives is meant to be broad, recognizing the variability of conditions that nephrology treats, and nimble in acknowledgment of the constantly evolving environment in which today's clinician practices.

We invite you to partner with us as EPC strives to support the needs of nephrology clinicians today and in the future. To learn more about EPC, please visit https://epc.asn-online.org/ or scan the QR code. ■

Alan S. Kliger, MD, is chair of the EPC Advisory Committee and clinical professor of medicine at Yale University School of Medicine, New Haven, CT.

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Fostering Inclusivity in Dialysis

A Leadership Training Program

By Cynthia Delgado on behalf of the FinD Steering Committee

ASN, in partnership with the National Institute of Diabetes and Digestive and Kidney Diseases, is introducing a 1-year leadership training program for 10 early career individuals practicing in nephrology from across the workforce spectrum. The Fostering Inclusivity in Dialysis (FinD) program is designed to cultivate leadership skills for future leaders in nephrology. The overarching goal of FinD is to create a diverse leadership workforce poised to improve outcomes and experiences for people living with kidney diseases impacted by health care disparities.

ASN is the largest nephrology society in the world, with more than 21,000 members, nearly doubling since 2010. Members represent a diverse workforce, with individuals working within the areas of pharmaceutical and biotech, health policy and advocacy, interventional nephrology, clinical dialysis (both private practice and dialysis organizations), education, and research. Workforce diversity is critical to achieving ASN's goals to accelerate innovation and expand patient choice while leading a culture change that creates pathways for advancing kidney health. ASN's longstanding commitment toward addressing health care inequities has led to a renaissance in recent years. But there is more to be done. Reflecting ASN's workforce diversity, a new generation of leaders is needed to address a multitude of challenges in dialysis (Figure 1).

Dialysis is both the lifeline and the Achilles' heel of care for patients with kidney failure. Nearly 800,000 Americans with kidney failure depend on dialysis for their survival (1). However, a large disproportion of individuals who depend on dialysis are African American, Latinx American, Native American, and from historically marginalized communities (Figure 2A). Apart from racial and ethnic disparities in kidney care, the use of more advanced home therapies on the whole remains flat (Figure 2B).

Figure 1. Leveraging ASN's workforce diversity to identify and train future leaders to address current and future challenges to dialysis therapies

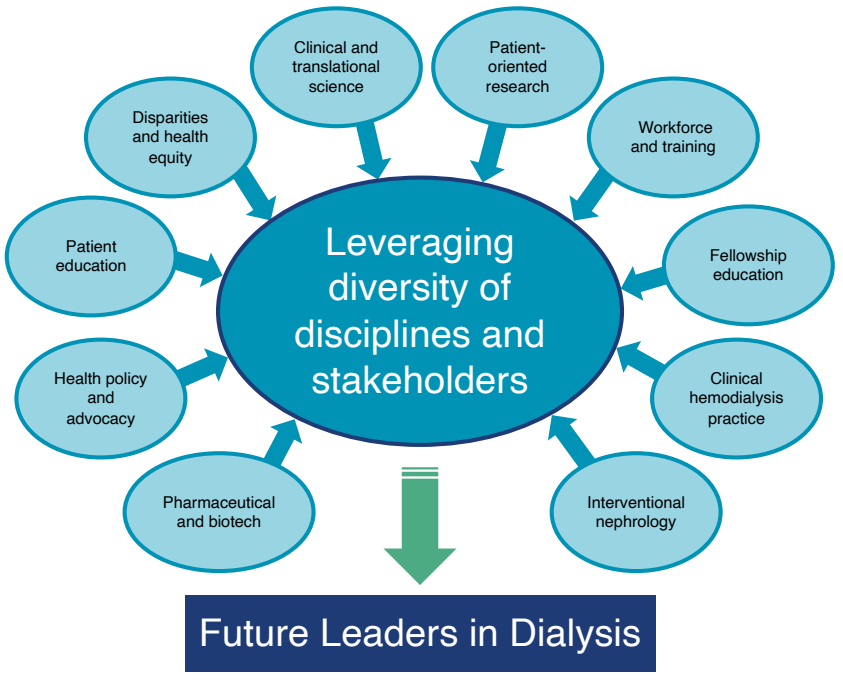
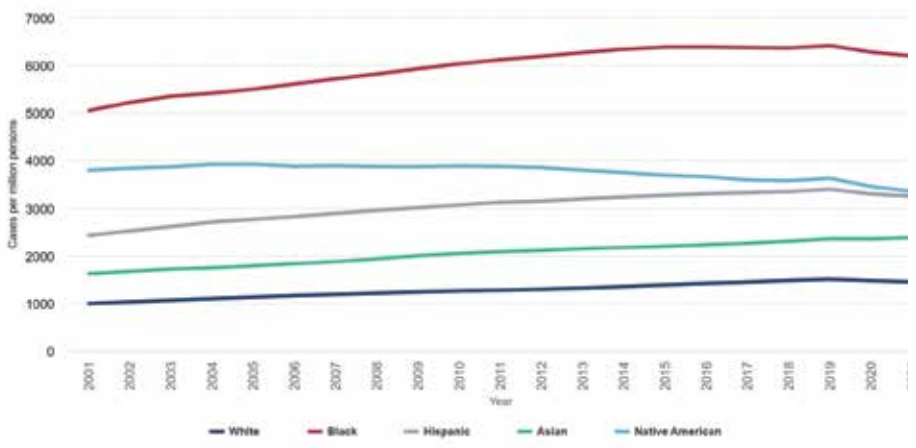


Figure 2A. Adjusted prevalence of kidney failure by race and ethnicity, 2001–2021



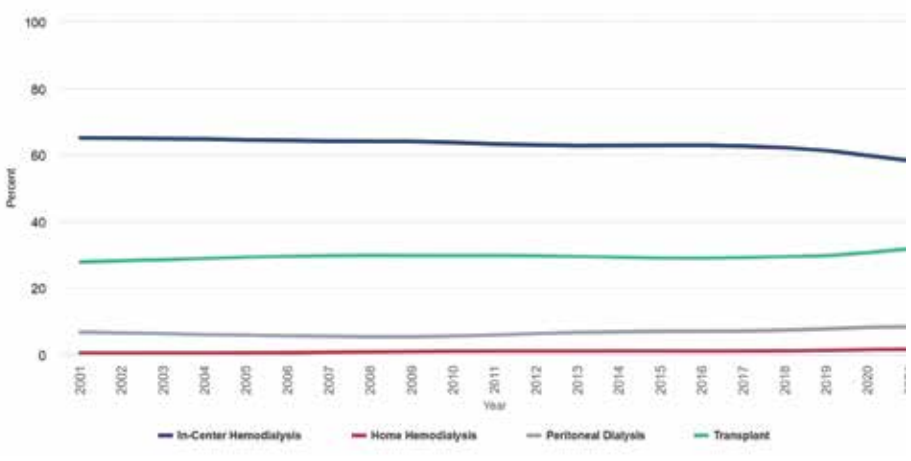
Source: US Renal Data System, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases (1).

Dialysis access placement and its maintenance experience challenges, particularly among patient populations that are historically marginalized. By US Renal Data System estimates, the percentage of patients initiating hemodialysis with a tunneled dialysis catheter has grown progressively to 75% in 2021, with a difference that cannot be accounted for by region (urban versus rural) (Figure 3) (1). Arteriovenous fistula (AVF) is still considered to be the gold standard, owing to fewer long-term complications and a lower overall health care cost, yet 9 in 10 young adults (ages 18–44 years) are starting hemodialysis with a tunneled dialysis catheter. There are a number of barriers to successful AVF use, including issues around placement, maturation, and long-term use (1). Only 44% of all AVF mature without intervention, in which maturation is defined as the ability to use AVF for hemodialysis treatment (1). AV grafts, in contrast, allow for early use of the vascular access but are plagued by a reproducible stenosis at the graft-vein anastomosis, resulting in a 1-year unassisted patency of only 23%. More biomedical research innovation is needed to improve these modalities of dialysis access.

Such challenges go beyond the dialysis treatment itself, as individuals receiving dialysis who live in marginalized communities experience reduced access to the needed steps in the path to kidney transplant (2). Although many nephrology leaders have led efforts to improve health care equity within dialysis, the work is not complete (3–5). Understanding the many levers that drive differences, including the potential contribution of socioeconomic factors, is also needed. Additionally, recent work suggests that policy-incentivized dialysis outcomes may not entirely align with a patient's life goals. A new generation of health policy and advocacy leaders is needed to better align policy-incentivized dialysis outcomes with patient-centered needs (6). More recent social events prompted ASN to further support efforts to increase workforce diversity, recognizing how it can foster innovation (7, 8).

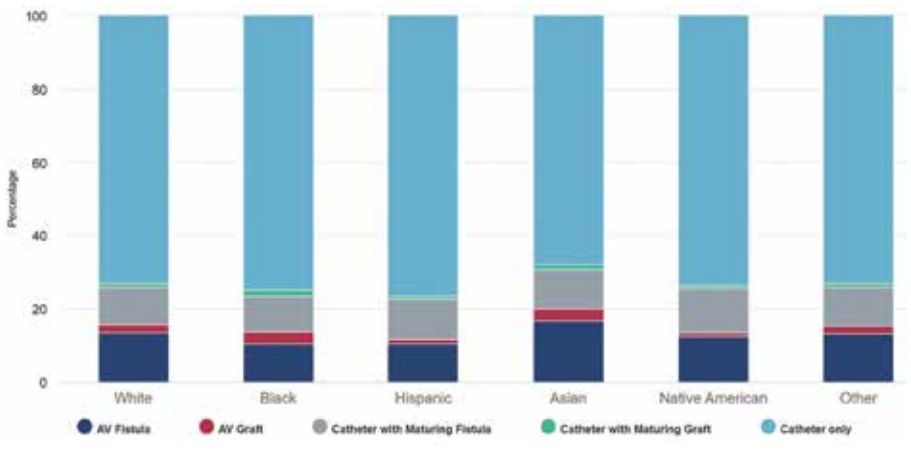
We can do better, and training leaders who will identify and address issues in dialysis care and in the experience of patients on dialysis, both locally and at the national level, is the first step.

Figure 2B. Percent prevalence of kidney failure by modality, 2001–2021



Source: US Renal Data System, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases (1).

Figure 3. Vascular access use at hemodialysis initiation by patient race and ethnicity, 2021



Source: US Renal Data System, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases (1).

Multifaceted approach to leadership training

Participants in the FinD program can expect a multifaceted approach to becoming the next generation of leaders and will be offered a unique opportunity for building relationships with experienced leaders and mentors.

First, participants will be exposed to a blend of one-on-one mentorship and leadership training with carefully selected leaders within nephrology and the broader health care community who are working to eliminate health care disparities. Participants will meet with their assigned mentor team, selected based on participants' career goals. This experience will forge a pathway to becoming leaders in their chosen area of specialty, not only to address equitable, patient-centered dialysis access care but also as future leaders in ASN and the broader kidney community.

Second, the training experience includes a virtual lecture series held at regular intervals, covering topics such as vascular access, current health care disparities in dialysis, dialysis health policy, research, and leadership. Each virtual skill development course will be in a townhall format to allow the exchange of ideas after the didactic presentation.

Third, a leadership capstone project will offer the participants a real-world leadership experience to apply the knowledge gained. The participants may choose grant writing, policy change, quality improvement, innovation, and workforce development as their area of interest to focus their capstone project.

Finally, participants will attend in-person networking events with their mentors and program leaders to facilitate cross-pollination of ideas.

After program completion, participants will be invited to future program alumni events to facilitate ongoing resource access and opportunities with future cohorts of the program.

For more information on FinD, please visit https://epc.asn-online.org/projects/find/ or scan the QR code. ■

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Glomerular Diseases Collaborative

Advancing Care for People With Glomerular Diseases

By Keisha Gibson and Christina Silva on behalf of the GD-C Steering Committee

The Glomerular Diseases Collaborative (GD-C) aims to promote high-quality care for people with glomerular diseases (GDs) and stimulate opportunities to address gaps in knowledge, training, continuing education, and awareness across the spectrum of GDs.

Most forms of GDs develop gradually, often exposing no symptoms for many years. GD pathogenesis varies and is believed to be the result of autoimmune, genetic, epigenetic, infectious, and paraneoplastic factors, as well as exposure to medications and environmental influences.

In 2023, ASN formed a strategic advisory panel of GD experts across the United States. The ASN GD-C Strategic Advisory Panel explored, analyzed, and evaluated the diagnosis and treatment landscape for GD. The panel identified numerous tactical opportunities to address the gaps in GD care, research, and education.

ASN launched its GD-C in April 2024 and will further define the scope of the initiative, building from the strategic framework identified by the strategic advisory panel. The ASN GD-C Steering Committee will develop a framework consisting of guiding principles, project scope, and prioritization of initiatives and tactics.

In addition, the steering committee will build and implement targeted initiatives to support the identified strategic priorities:

Access to care

- Support access to first-line medications by developing clinician tools and resources to assist with prior authorization processes.
- Support identification and referral to GD specialists nationally.
- Collaborate with ASN Policy to advocate for telehealth and licensing across state lines.

Clinical knowledge

- Build a web-based "clinical collection" compendium to include the most up-to-date content on GD, highlight websites focused on GD, and share prioritized educational materials and resource links.
- Develop educational assets focused on treatment pathways and best practices to optimize outcomes, including early diagnosis, clinical trials as a treatment pathway, and emerging therapies.
  - These educational assets include:
    - Disease educational fact sheets
    - Digital educational materials
    - Webinars and podcasts focused on GD identification and treatment best practices
- Submit a GD topic to ASN Kidney Health Guidance to develop clinical guidance.
- Submit a manuscript to a peer-reviewed journal to focus on identification and management of GDs or other prioritized topics determined by the steering committee.

Research/clinical trial infrastructure

- Facilitate enrollment in clinical research trials, and collaborate with ASN Research, Discovery, and Innovation.

Economic analysis

- Perform cost/risk/benefit analyses for early screening in children, using new therapies and licensing across state lines.

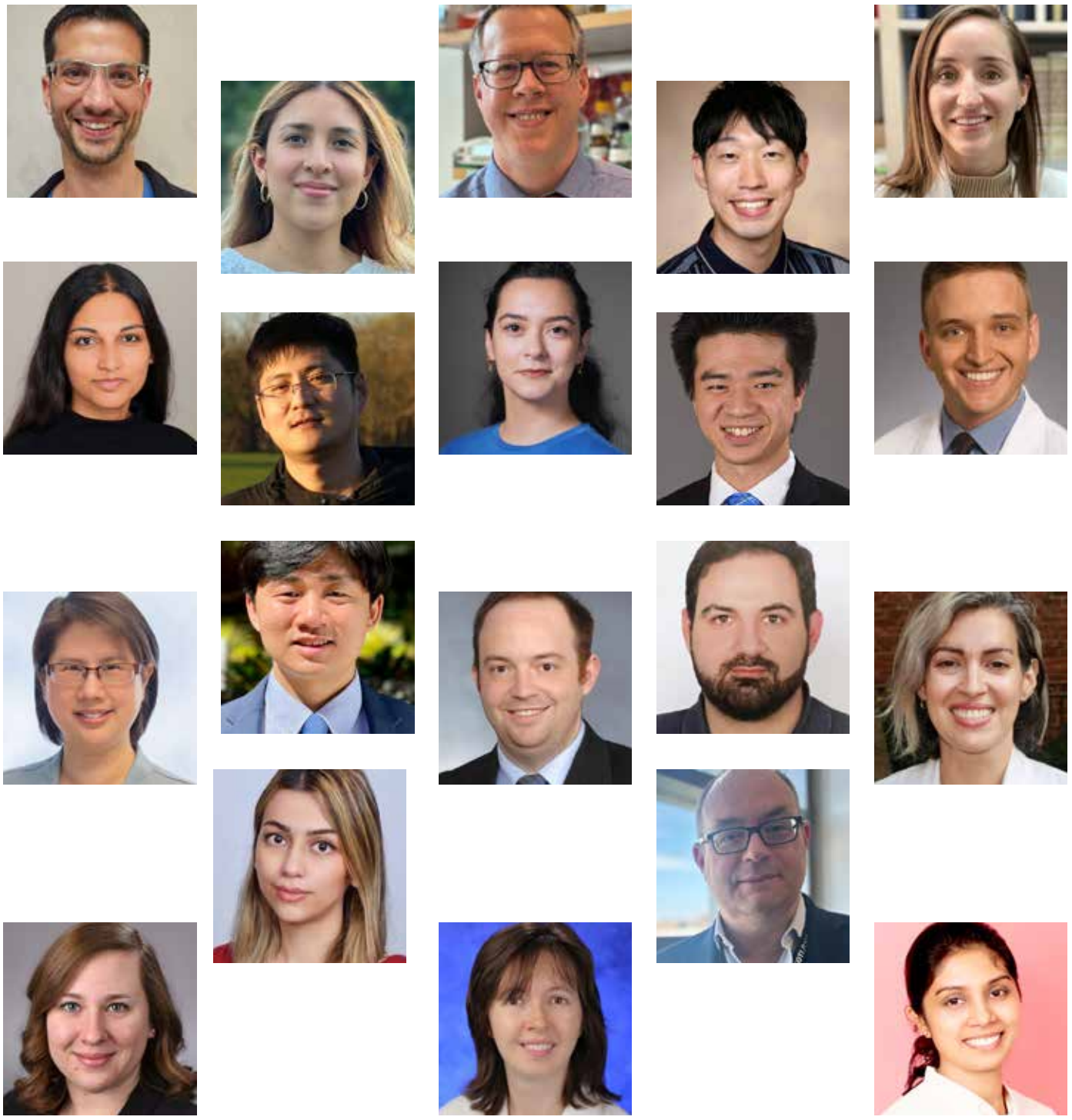
This group is driven by the mission of making sure that all people with GDs, regardless of whether they are a farmer living in the rural midwest, a professor living in a large metropolitan city, or a child, have access to the best care and that their treatment is the same.

To learn more about the Glomerular Diseases Collaborative, please visit https://epc.asn-online.org/projects/gd-c/ or scan the QR code. ■

Support for the Glomerular Diseases Collaborative is provided by Amgen; Otsuka; Travere Therapeutics, Inc.; and Vera Therapeutics, Inc.

KidneyCure congratulates the talented group of individuals awarded grants in 2024.

With support from ASN members, industry partners, and nephrology leaders, KidneyCure provides approximately $3,000,000 annually to young researchers, fellows, and educators at the most critical stages of their careers—giving life and momentum to ideas that would otherwise be lost.



Make a difference today.

Support these trailblazers and future kidney leaders by visiting www.kidneycure.org.

KidneyCure

Fund it. Find it.

ASN-Harold Amos Medical Faculty Development Program

Aiming to increase diversity among future leaders, the ASN-Harold Amos Medical Faculty Development Program provides four years of research and career development support to a nephrologist from a historically disadvantaged background.

Jordan Gabriela Nestor, MD, MS, FASN *
Columbia University
Utilizing Temporal Electronic Health Record (EHR) for Early Detection of Collagen Type IV-Associated Nephropathy (COL4A-AN)

KidneyCure Pre-Doctoral Fellowship Program

The KidneyCure Pre-Doctoral Fellowship Program provides funding to early career-stage PhD students to conduct original research projects and make contributions to the understanding of kidney biology and disease.

Bengucan Gunen, MSPH
Drexel University Dornsife School of Public Health
Addressing Disparities in Renal Diet Adherence: Leveraging Social Support to Improve Dietary Behaviors Among End Stage Kidney Disease Patients with Obesity

Arvin Halim *
Indiana University School of Medicine
Modulating the Focal Adhesion Pathway with KL1 to Inhibit Cardiac Fibrosis in CKD

Ben J. Lipps Research Fellowship Program

The Ben J. Lipps Research Fellowship Program supports nephrology fellows who will advance the understanding of kidney biology and disease and is fully endowed by contributions provided by Fresenius Medical Care, ASN, the American Renal Patient Care Foundation, Inc., Amgen, Baxter, and the PKD Foundation.

Jessica Bahena-López, MD, PhD *
Oregon Health & Science University
Exploring Dual Polarization of CaSR in the Distal Convoluted Tubule: Implications for GPCR Signaling and Sodium Salt Regulation
Ben J. Lipps Research Fellowship Award

Ryoichi Bessho, MD, PhD *
Vanderbilt University Medical Center
Dissecting Mitochondrial Electron Transport Function in Renal Fibrogenesis
Sharon Anderson Research Fellowship Award

Meghana Eswarappa, MD
University of California, San Francisco
Evaluating Racial Disparities in Chronic Kidney Disease Severity and Treatment in Autosomal Dominant Polycystic Kidney Disease
Jared J. Grantham Research Fellowship Award

Chenjian Gu, PhD
Washington University in St. Louis
Novel Regulation of Autophagic Flux for the Treatment of Uromodulin-associated Nephropathy
Ben J. Lipps Research Fellowship Award

Robin Lo, MD *
Stanford University
Studying Mechanisms of Tubular Remodeling–Implications for Physiology
KidneyCure Research Fellowship Award

Samer Mohandes, MD *
University of Pennsylvania
Alternative Splicing in Chronic Kidney Disease
Joseph A. Carlucci Research Fellowship Award

Megan L. Noonan, PhD *
Washington University in St. Louis
The Role of Alternative Splicing Factor Srsf7 in Diabetic Kidney Disease Progression
Ben J. Lipps Research Fellowship Award

Poornima Dilhani Ekanayake Weerasinghe Mudiyanselage, PhD *
Washington University in St. Louis
Chronic Neuroinflammation, Neuropathology and Neurocognitive Dysfunction After Acute Kidney Injury
Donald E. Wesson Research Fellowship Award

Transition to Independence Grants Program

The Transition to Independence Grants Program helps young investigators achieve independent research careers and is supported by contributions provided by ASN, Akebia Therapeutics, Inc., Otsuka and Visterra, and individual donors.

David P. Al-Adra, MD, PhD
University of Wisconsin
Delivery of a Novel Immune Inhibitory Transgene to Kidneys During Normothermic Preservation to Decrease Rejection After Transplantation
John Merrill Grant in Transplantation

Jeffrey A. Beamish, MD, PhD *
University of Michigan
Control of Proximal Tubule Metabolism by Pax8 in Ischemic Kidney Injury
Carl W. Gottschalk Research Scholar Grant

Andrew S. Beenken, MD, PhD
Columbia University
Structural Mechanisms of Ligand Binding by Cubilin in the Proximal Tubule
Carl W. Gottschalk Research Scholar Grant

Maria Castañeda-Bueno, PhD
Salvador Zubiran National Institute of Medical Sciences and Nutrition
Novel Insights Into the Physiology of the Distal Convoluted Tubule
Joseph V. Bonventre Career Development Grant

Scott M. Krummey, MD, PhD
Johns Hopkins University
Role of CD43 Signaling on Effector CD8+ T Cells in Acute Rejection
Carl W. Gottschalk Research Scholar Grant

Jennifer Lai Yee, MD, MPH, PhD *
University of Michigan
Precision Medicine in Kidney Disease: Functionally Classify Variants of Nephrotic Syndrome Genes Using a Multiplex Approach
Norman Siegel Research Scholar Grant

Kyle McCracken, MD, PhD
Cincinnati Children's Hospital Medical Center
FOXI1-dependent Genetic Mechanisms of Human Intercalated Cell Development
Carl W. Gottschalk Research Scholar Grant

Jennifer A. Schaub, MD *
University of Michigan
A Systems Biology Approach to Identify Cortical and Medullary Gene Regulatory Networks Related to Hypoxia Inducible Factor-1A
Carl W. Gottschalk Research Scholar Grant

Nicholas J. Steers, PhD *
Columbia University
Elucidating the Role of T-follicular Like Helper Cells Promoting the Activation of B-cells in IgA Nephropathy
Carl W. Gottschalk Research Scholar Grant

William and Sandra Bennett Clinical Scholars Program

The William and Sandra Bennett Clinical Scholars Program provides funding to clinician educators to conduct a project to advance all facets of nephrology education and teaching.

Sayna Norouzi, MD, FASN
Loma Linda University
Online Glomerular Diseases Fellowship Program: Engaging and Interactive Learning

** Kidney Week 2024 oral and/or poster abstract presenter*

Diagnostic Excellence: eGFR Toolkit

New Implementation Resource for Health Care Professionals

By Crystal Gadegbeku and Christina Silva on behalf of the Diagnostic Excellence: eGFR Toolkit Steering Committee

The Diagnostic Excellence: eGFR Toolkit project, funded by a grant from the Council of Medical Specialty Societies, was awarded to ASN and launched this year. This project aims to promote national implementation of the race-free estimated glomerular filtration rate (eGFR) equation in the screening, early detection, and management of kidney diseases. The Diagnostic Excellence: eGFR Steering Committee was tasked with developing an implementation toolkit to highlight the importance of removing race from the eGFR calculation, devise an easily accessible guide for using the recommended eGFR equations, and serve as a resource for additional information for health care professionals and people living with kidney diseases.

Background

The National Kidney Foundation (NKF) and ASN first collaborated in 2020 to reassess the inclusion of race in the estimation of kidney function. After 1 year of comprehensive deliberation, the NKF-ASN Task Force provided recommendations to adopt the race-free creatinine-based and cystatin C-based eGFR equations for eGFR reporting. Currently, NKF is working diligently with clinical commercial laboratories to adopt the new eGFR equation in reporting eGFR and to make available the cystatin C measurement for cystatin C-based eGFR reporting. To date, approximately two-thirds of the clinical commercial laboratories in the United States are now using the race-free eGFR equation (1).

Identifying needs

The steering committee hosted roundtables, engaging health care professionals representing multiple disciplines (including cardiology, rheumatology, endocrinology, internal medicine, family practice, and nephrology) caring for people at risk for and with kidney diseases to assess foundational knowledge of eGFR and cystatin C.

Among non-nephrologists who participated in the roundtable discussions, a prediscussion survey indicated that there was varied knowledge and insight about the evolution to the race-free 2021 eGFR equation and subsequently, variable levels of comfort in explaining this change to patients. Additionally, not all participants were familiar with the cystatin C-based equations, and some who were familiar with the alternative eGFR measurement did not have it available in their affiliated laboratory system to use for patient care.

Among nephrologists who participated in the roundtable discussions, the prediscussion survey indicated that these physicians were familiar with the new race-free eGFR with some insight into the basis for the change. However, there was varied familiarity with the cystatin C-based equation and a varied level of comfort explaining the change in eGFR reporting to patients.

For development of the eGFR Toolkit, the goals of the roundtable discussions were:

- to determine health care professionals' understanding of the removal of race from the eGFR equation,
- to understand how best to implement the revised eGFR calculation for clinical practice,
- to understand whether there are opportunities to improve patient education about the race-free eGFR equation, and
- to assess health care professionals' understanding and impact of laboratory-reporting changes due to the adoption of the race-free eGFR equation.

Many key themes from both groups emerged (Table), and the discussions from the roundtables have been useful in developing a framework for the eGFR Toolkit.

Toolkit development

Based on the information from the roundtable discussions, the eGFR Toolkit is uniquely designed to be an accessible online resource to facilitate the adoption of the race-free equation in clinical practice. Toolkit chapters will allow the learner to read from beginning to end at their leisure, choose specific topics on eGFR, or link to eGFR calculators and patient-facing materials for patient care management. There is also a certificate of completion for learners who read the toolkit in its entirety. The educational goals include the following:

- enhancing screening and early detection of chronic kidney disease (CKD) in people at risk for CKD, including minoritized populations; the elderly; and those with diabetes, hypertension, cardiovascular disease, and/or other systemic illnesses
- improving management of CKD across a broad range of specialties and health care professionals
- facilitating familiarity and the appropriate use of cystatin C-based eGFR equations as testing availability increases nationally

Evaluation, communication, and dissemination

Providing the opportunity for the completed eGFR Toolkit to be evaluated by nephrology and non-nephrology health care professionals was key to refining the educational materials. For ongoing feedback, an evaluation has been embedded in the online toolkit.

Now that the eGFR Toolkit has been finalized, communication and dissemination are the next essential steps for this project. ASN is holding webinars to share the benefits and availability of the eGFR Toolkit and collaborating with the Council of Medical Specialty Societies to promote the toolkit.

The Diagnostic Excellence: eGFR Toolkit is available and free to access by visiting https://epc.asn-online.org/projects/estimated-glomerular-filtration-rate-egfr or by scanning the QR code. ■

Reference

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Table. Key themes from multidisciplinary roundtable discussions

Non-nephrologist roundtable themes	Nephrologist roundtable themes
Lack of comfort in using online tools for eGFR calculation; reliance on laboratory reporting from various laboratory systems	Integration of historical eGFR reporting missing or inadequately labeled for assessing progression
Lack of standardized reporting and clear labeling	
Lack of awareness and/or accessibility to cystatin C	
Lack of patient educational tools to guide explanation	

Kidney Community Vaccination Collaborative

Promoting Immunization for Respiratory Illnesses

By Rebecca Schmidt on behalf of the KCVC Steering Committee

The importance of vaccination for people living with kidney diseases is well known. However, dialysis facility staff often struggle to persuade patients to receive immunizations for respiratory illnesses including influenza, COVID-19, and pneumonia. Newer vaccines, such as for respiratory syncytial virus, can present additional challenges for the kidney community. Patients' hesitancy is due to many factors, including logistical challenges, confusion about recommendations, distrust in the medical field in general, cultural concerns, and misinformation about the safety of vaccines or the importance of being immunized.

To address these challenges, in 2021, ASN partnered with the Council of Medical Specialty Societies and the Centers for Disease Control and Prevention in their "Specialty Societies Advancing Adult Immunizations" (SSAAI) initiative. The goals of this project were to increase the adoption of the Centers for Disease Control and Prevention's Standards for Adult Immunization Practice and the delivery of recommended vaccinations through the broad spectrum of nephrology care.

In fall 2024, ASN built upon the foundations of the SSAAI initiative to launch the Kidney Community Vaccination Collaborative (KCVC). The focus of the collaborative is to adapt the work begun with the SSAAI initiative to the unique needs of the kidney community, in which clinicians and patients have consistent and often long-term relationships, and medical professionals are well-versed on the unique challenges facing patients with kidney diseases. These relationships offer the opportunity for dialysis facility professionals to serve as the trusted source of information in the evolving landscape of vaccination recommendations.

To lead this work, KCVC has established a steering committee comprised of representatives from nephrology: physicians, nurses, researchers, pharmacists, pediatricians, the patient community, and immunization leaders. The steering committee is supported by a Learning and Action Network, through which community members share best practices and resources for promoting immunization. KCVC is charged with the following goals:

- **Identify** the unique needs of the kidney community.
- **Assemble** best practices for vaccination.
- **Disseminate** resources and strategies for promoting vaccination.
- **Engage** dialysis facility staff to communicate with patients and other staff.
- **Pursue** collaborations with community partners to support vaccination at dialysis facilities.
- **Review** data to track vaccination trends.

This fall, KCVC launched a vaccination campaign targeting respiratory illnesses including influenza, COVID-19, respiratory syncytial virus, and pneumonia. Through its early work with SSAAI, KCVC has already established a library of resources, including:

- ASN Statement on Immunizations in the Kidney Community
- "How to be a Vaccine Advocate" resource for clinicians and staff
- COVID-19 Pocket Guide and Companion Posters (English and Spanish) for dialysis staff
- Video testimonials featuring dialysis facility staff and a patient advocate
- Micro-learning module promoting vaccination for staff and patients

In addition, the steering committee and Learning and Action Network members have used a variety of techniques to confront misinformation and facilitate vaccination. These include:

- "Lunch and Learn" program featuring a dialysis facility medical director
- "Myth Busters" initiative to directly confront patient fears and misinformation
- Vaccine clinics, in which a local health partner provides vaccination at the dialysis facility and completes documentation in the state database
- BINGO game to engage patients and promote understanding of the importance of vaccination

For 2025, KCVC will consider these proposed initiatives:

- **Develop** vaccination checklists: What is available, when to receive, and where.
- **Generate** educational materials for staff and patients for all vaccines recommended for patients on dialysis.
- **Adapt** materials specific to transplant and pediatrics.
- **Pursue** partnerships with kidney organizations.
- **Conduct** a focus group of interdisciplinary representatives to identify the needs of the community.
- **Advocate** for enhanced access to and coverage for vaccination for patients on dialysis.

Immunization is a simple process and has been a cornerstone of the medical field for decades. Its importance and value to patients and public health are well-documented. However, the challenges that staff face in promoting immunization are real and varied. KCVC offers a new opportunity for dialysis professionals to know the most up-to-date vaccine recommendations and embrace their trusted role, promote best practices, and encourage vaccination for all respiratory illnesses that can be deadly to this vulnerable population.

KCVC invites all members of the kidney community to join in support of promoting vaccinations through respectful collaboration and initiatives tailored for the kidney community. "Build Trust. Open Dialogue. Respect Choices."

To learn more about KCVC and explore the resources mentioned, please visit https://epc.asn-online.org/projects/kcvc/ or scan the QR code, or to share your own best practices, email epc@asn-online.org.

INDICATIONS & USAGE

FILSPARI® (sparsentan) is indicated to slow kidney function decline in adults with primary immunoglobulin A nephropathy (IgAN) who are at risk for disease progression.

IMPORTANT SAFETY INFORMATION

BOXED WARNING: HEPATOTOXICITY AND EMBRYO-FETAL TOXICITY

Because of the risks of hepatotoxicity and birth defects, FILSPARI is available only through a restricted program called the FILSPARI REMS. Under the FILSPARI REMS, prescribers, patients and pharmacies must enroll in the program.

Hepatotoxicity

Some Endothelin Receptor Antagonists (ERAs) have caused elevations of aminotransferases, hepatotoxicity, and liver failure. In clinical studies, elevations in aminotransferases (ALT or AST) of at least 3-times the Upper Limit of Normal (ULN) have been observed in up to 3.5% of FILSPARI-treated patients, including cases confirmed with rechallenge.

Measure transaminases and bilirubin before initiating treatment and monthly for the first 12 months, and then every 3 months during treatment. Interrupt treatment and closely monitor patients who develop aminotransferase elevations more than 3x ULN.

FILSPARI should generally be avoided in patients with elevated aminotransferases (>3x ULN) at baseline because monitoring for hepatotoxicity may be more difficult and these patients may be at increased risk for serious hepatotoxicity.

Embryo-Fetal Toxicity

FILSPARI can cause major birth defects if used by pregnant patients based on animal data. Therefore, pregnancy testing is required before the initiation of treatment, during treatment and one month after discontinuation of treatment with FILSPARI. Patients who can become pregnant must use effective contraception before the initiation of treatment, during treatment, and for one month after discontinuation of treatment with FILSPARI.

Contraindications

FILSPARI is contraindicated in patients who are pregnant. Do not coadminister FILSPARI with angiotensin receptor blockers (ARBs), ERAs, or aliskiren.

Warnings and Precautions

- **Hepatotoxicity:** Elevations in ALT or AST of at least 3-fold ULN have been observed in up to 3.5% of FILSPARI-treated patients, including cases confirmed with rechallenge. While no concurrent elevations in bilirubin >2-times ULN or cases of liver failure were observed in FILSPARI-treated patients, some ERAs have caused elevations of aminotransferases, hepatotoxicity, and liver failure. To reduce the risk of potential serious hepatotoxicity, measure serum aminotransferase levels and total bilirubin prior to initiation of treatment and monthly for the first 12 months, then every 3 months during treatment.

  Advise patients with symptoms suggesting hepatotoxicity (nausea, vomiting, right upper quadrant pain, fatigue, anorexia, jaundice, dark urine, fever, or itching) to immediately stop treatment with FILSPARI and seek medical attention. If aminotransferase levels are abnormal at any time during treatment, interrupt FILSPARI and monitor as recommended.

  Consider re-initiation of FILSPARI only when hepatic enzyme levels and bilirubin return to pretreatment values and only in patients who have not experienced clinical symptoms of hepatotoxicity. Avoid initiation of FILSPARI in patients with elevated aminotransferases (>3x ULN) prior to drug initiation because monitoring hepatotoxicity in these patients may be more difficult and these patients may be at increased risk for serious hepatotoxicity.
- **Embryo-Fetal Toxicity:** FILSPARI can cause fetal harm when administered to a pregnant patient and is contraindicated during pregnancy. Advise patients who can become pregnant of the potential risk to a fetus. Obtain a pregnancy test prior to initiation of treatment with FILSPARI, monthly during treatment, and one month after discontinuation of treatment. Advise patients who can become pregnant to use effective contraception prior to initiation of treatment, during treatment, and for one month after discontinuation of treatment with FILSPARI.
- **FILSPARI REMS:** Due to the risk of hepatotoxicity and embryo-fetal toxicity, FILSPARI is available only through a restricted program called the FILSPARI REMS. Prescribers, patients, and pharmacies must be enrolled in the REMS program and comply with all requirements (www.filsparirems.com).
- **Hypotension:** Hypotension has been observed in patients treated with ARBs and ERAs. There was a greater incidence of hypotension-associated adverse events, some serious, including dizziness, in patients treated with FILSPARI compared to irbesartan. In patients at risk for hypotension, consider eliminating or adjusting other antihypertensive medications and maintaining appropriate volume status. If hypotension develops, despite elimination or reduction of other antihypertensive medications, consider a dose reduction or dose interruption of FILSPARI. A transient hypotensive response is not a contraindication to further dosing of FILSPARI, which can be given once blood pressure has stabilized.
- **Acute Kidney Injury:** Monitor kidney function periodically. Drugs that inhibit the renin-angiotensin system (RAS) can cause kidney injury. Patients whose kidney function may depend in part on the activity of the RAS (e.g., patients with renal artery stenosis, chronic kidney disease, severe congestive heart failure, or volume depletion) may be at particular risk of developing acute kidney injury on FILSPARI. Consider withholding or discontinuing therapy in patients who develop a clinically significant decrease in kidney function while on FILSPARI.
- **Hyperkalemia:** Monitor serum potassium periodically and treat appropriately. Patients with advanced kidney disease, taking concomitant potassium-increasing drugs (e.g., potassium supplements, potassium-sparing diuretics), or using potassium-containing salt substitutes are at increased risk for developing hyperkalemia. Dosage reduction or discontinuation of FILSPARI may be required.
- **Fluid Retention:** Fluid retention may occur with ERAs, and has been observed in clinical studies with FILSPARI. FILSPARI has not been evaluated in patients with heart failure. If clinically significant fluid retention develops, evaluate the patient to determine the cause and the potential need to initiate or modify the dose of diuretic treatment then consider modifying the dose of FILSPARI.

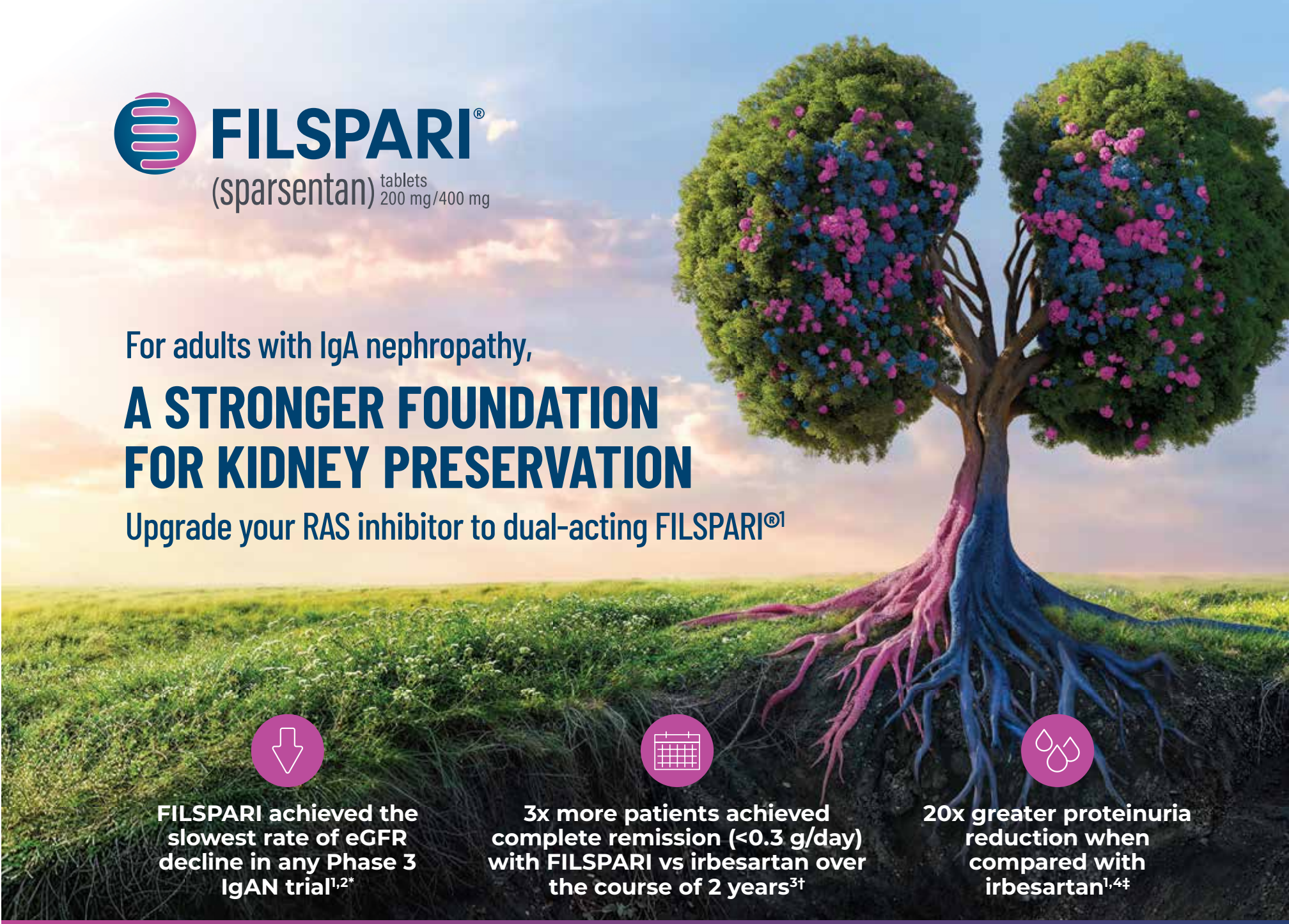
Most common adverse reactions

The most common adverse reactions (≥5%) are hyperkalemia, hypotension (including orthostatic hypotension), peripheral edema, dizziness, anemia, and acute kidney injury.

Drug interactions

- **Renin-Angiotensin System (RAS) Inhibitors and ERAs:** Do not coadminister FILSPARI with ARBs, ERAs, or aliskiren due to increased risks of hypotension, syncope, hyperkalemia, and changes in renal function (including acute renal failure).
- **Strong and Moderate CYP3A Inhibitors:** Avoid concomitant use of FILSPARI with strong CYP3A inhibitors. If a strong CYP3A inhibitor cannot be avoided, interrupt FILSPARI treatment. When resuming treatment with FILSPARI, consider dose titration. Monitor blood pressure, serum potassium, edema, and kidney function regularly when used concomitantly with moderate CYP3A inhibitors. Concomitant use with a strong CYP3A inhibitor increases sparsentan exposure which may increase the risk of FILSPARI adverse reactions.
- **Strong CYP3A Inducers:** Avoid concomitant use with a strong CYP3A inducer. Concomitant use with a strong CYP3A inducer decreases sparsentan exposure which may reduce FILSPARI efficacy.
- **Antacids and Acid Reducing Agents:** Administer FILSPARI 2 hours before or after administration of antacids. Avoid concomitant use of acid reducing agents (histamine H2 receptor antagonist and PPI proton pump inhibitor) with FILSPARI. Sparsentan exhibits pH-dependent solubility. Antacids or acid reducing agents may decrease sparsentan exposure which may reduce FILSPARI efficacy.

FILSPARI®
(sparsentan) tablets 200 mg/400 mg



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Upgrade your RAS inhibitor to dual-acting FILSPARI®[1]

FILSPARI achieved the slowest rate of eGFR decline in any Phase 3 IgAN trial[1,2*]

3x more patients achieved complete remission (<0.3 g/day) with FILSPARI vs irbesartan over the course of 2 years[3†]

20x greater proteinuria reduction when compared with irbesartan[1,4‡]

Upgrade your patients' RASi treatment to FILSPARI[1]
Scan or visit FILSPARIhcp.com to learn more

*The mean eGFR slope from baseline to Week 110 was -3.0 mL/min/1.73 m^2 per year for FILSPARI and -4.2 mL/min/1.73 m^2 per year for irbesartan.[1]
†Complete remission is measured by reaching urinary protein excretion <0.3 g/day at any time during the 110-week PROTECT Study. Remission was achieved more frequently in FILSPARI patients (31%) than in irbesartan patients (11%).[2,5]
‡The adjusted geometric mean (GM) of UPCR at baseline was 1.2 g/g for FILSPARI (n=202) and irbesartan (n=202). At 110 weeks, patients on FILSPARI saw a -40% (0.7 g/g) reduction compared to patients on irbesartan which was -2% (1.2 g/g).[1,4]
CI=confidence interval; eGFR=estimated glomerular filtration rate; IgA=immunoglobulin A; IgAN=immunoglobulin A nephropathy; RAS=renin-angiotensin system; RASi=renin-angiotensin system inhibitor; UPCR=urine protein-to-creatinine ratio.

IMPORTANT SAFETY INFORMATION: Drug interactions (cont'd)

- **Non-Steroidal Anti-Inflammatory Agents (NSAIDs), Including Selective Cyclooxygenase-2 (COX-2) Inhibitors:** Monitor for signs of worsening renal function with concomitant use with NSAIDs (including selective COX-2 inhibitors). In patients with volume depletion (including those on diuretic therapy) or with impaired kidney function, concomitant use of NSAIDs (including selective COX-2 inhibitors) with drugs that antagonize the angiotensin II receptor may result in deterioration of kidney function, including possible kidney failure.
- **CYP2B6, 2C9, and 2C19 Substrates:** Monitor for efficacy of concurrently administered CYP2B6, 2C9, and 2C19 substrates and consider dosage adjustment in accordance with the Prescribing Information. Sparsentan decreases exposure of these substrates, which may reduce efficacy related to these substrates.
- **P-gp and BCRP Substrates:** Avoid concomitant use of sensitive substrates of P-gp and BCRP with FILSPARI. Sparsentan may increase exposure of these transporter substrates, which may increase the risk of adverse reactions related to these substrates.
- **Agents Increasing Serum Potassium:** Monitor serum potassium frequently in patients treated with FILSPARI and other agents that increase serum potassium. Concomitant use of FILSPARI with potassium-sparing diuretics, potassium supplements, potassium-containing salt substitutes, or other drugs that raise serum potassium levels may result in hyperkalemia.

For additional Important Safety Information, please see the Brief Summary of the full Prescribing Information on the following pages, including BOXED WARNING.

References: 1. FILSPARI Prescribing Information. San Diego, CA: Travere Therapeutics, Inc. **2.** Data on file, REF-1302. Travere Therapeutics, Inc. **3.** Data on file, REF-1301. Travere Therapeutics, Inc. **4.** Data on file, REF-1303. Travere Therapeutics, Inc. **5.** Rovin BH, Barratt J, Heerspink HJL, et al. Efficacy and safety of sparsentan versus irbesartan in patients with IgA nephropathy (PROTECT): 2-year results from a randomised, active-controlled, phase 3 trial. *Lancet*. 2023;402(10417):2077-2090.

TRAVERE® THERAPEUTICS

Brief Summary of full Prescribing Information for FILSPARI® (sparsentan) tablets, for oral use

Initial U.S. Approval: 2023

INDICATIONS AND USAGE

FILSPARI is indicated to slow kidney function decline in adults with primary immunoglobulin A nephropathy (IgAN) who are at risk for disease progression.

WARNING: HEPATOTOXICITY and EMBRYO-FETAL TOXICITY

Because of the risks of hepatotoxicity and birth defects, FILSPARI is available only through a restricted program called the FILSPARI REMS. Under the FILSPARI REMS, prescribers, patients, and pharmacies must enroll in the program.

Hepatotoxicity

Some Endothelin Receptor Antagonists (ERAs) have caused elevations of aminotransferases, hepatotoxicity, and liver failure. In clinical studies, elevations in aminotransferases (ALT or AST) of at least 3-times the Upper Limit of Normal (ULN) have been observed in up to 3.5% of FILSPARI-treated patients, including cases confirmed with rechallenge.

Measure transaminases and bilirubin before initiating treatment and monthly for the first 12 months, and then every 3 months during treatment. Interrupt treatment and closely monitor patients who develop aminotransferase elevations more than 3-times ULN.

FILSPARI should generally be avoided in patients with elevated aminotransferases (>3-times ULN) at baseline because monitoring for hepatotoxicity may be more difficult and these patients may be at increased risk for serious hepatotoxicity.

Embryo-Fetal Toxicity

FILSPARI can cause major birth defects if used by pregnant patients based on animal data. Therefore, pregnancy testing is required before the initiation of treatment, during treatment, and one month after discontinuation of treatment with FILSPARI. Patients who can become pregnant must use effective contraception before the initiation of treatment, during treatment, and for one month after discontinuation of treatment with FILSPARI.

CONTRAINDICATIONS

Use of FILSPARI is contraindicated in patients who are pregnant.

Do not coadminister FILSPARI with angiotensin receptor blockers (ARBs), ERAs, or aliskiren.

WARNINGS AND PRECAUTIONS

Hepatotoxicity

Elevations in ALT or AST of at least 3-fold ULN have been observed in up to 3.5% of FILSPARI-treated patients, including cases confirmed with rechallenge. While no concurrent elevations in bilirubin greater than 2-times ULN or cases of liver failure were observed in FILSPARI-treated patients in clinical trials, some endothelin receptor antagonists have caused elevations of aminotransferases, hepatotoxicity, and liver failure. To reduce the risk of potential serious hepatotoxicity, measure serum aminotransferase levels and total bilirubin prior to initiation of treatment and monthly for the first 12 months, then every 3 months during treatment.

Advise patients with symptoms suggesting hepatotoxicity (nausea, vomiting, right upper quadrant pain, fatigue, anorexia, jaundice, dark urine, fever, or itching) to immediately stop treatment with FILSPARI and seek medical attention. If aminotransferase levels are abnormal at any time during treatment, interrupt FILSPARI and monitor as recommended.

Consider re-initiation of FILSPARI only when hepatic enzyme levels and bilirubin return to pretreatment values and only in patients who have not experienced clinical symptoms of hepatotoxicity.

Avoid initiation of FILSPARI in patients with elevated aminotransferases (greater than 3-times ULN) prior to drug initiation because monitoring hepatotoxicity in these patients may be more difficult and these patients may be at increased risk for serious hepatotoxicity.

Embryo-Fetal Toxicity

Based on data from animal reproduction studies, FILSPARI can cause fetal harm when administered to a pregnant patient and is contraindicated during pregnancy. Advise patients who can become pregnant of the potential risk to a fetus. Obtain a pregnancy test prior to initiation of treatment with FILSPARI, monthly during treatment, and one month after discontinuation of treatment. Advise patients who can become pregnant to use effective contraception prior to initiation of treatment, during treatment, and for one month after discontinuation of treatment with FILSPARI.

FILSPARI REMS

For all patients, FILSPARI is available only through a restricted program under a REMS called the FILSPARI REMS because of the risk of hepatotoxicity and embryo-fetal toxicity.

Important requirements of the FILSPARI REMS include the following:

- Prescribers must be certified with the FILSPARI REMS by enrolling and completing training.
- All patients must enroll in the FILSPARI REMS prior to initiating treatment and comply with monitoring requirements.
- Pharmacies that dispense FILSPARI must be certified with the FILSPARI REMS and must dispense only to patients who are authorized to receive FILSPARI.

Further information is available at www.filsparirems.com or 1-833-513-1325.

Hypotension

Hypotension has been observed in patients treated with ARBs and endothelin receptor antagonists (ERAs) and was observed in clinical studies with FILSPARI. In the PROTECT trial, there was a greater incidence of hypotension-associated adverse events, some serious, including dizziness, in patients treated with FILSPARI compared to irbesartan.

In patients at risk for hypotension, consider eliminating or adjusting other antihypertensive medications and maintaining appropriate volume status.

If hypotension develops, despite elimination or reduction of other antihypertensive medications, consider a dose reduction or dose interruption of FILSPARI. A transient hypotensive response is not a contraindication to further dosing of FILSPARI, which can be given once blood pressure has stabilized.

Acute Kidney Injury

Monitor kidney function periodically. Drugs that inhibit the renin-angiotensin system can cause acute kidney injury. Patients whose kidney function may depend in part on the activity of the renin-angiotensin system (e.g., patients with renal artery stenosis, chronic kidney disease, severe congestive heart failure, or volume depletion) may be at particular risk of developing acute kidney injury on FILSPARI. Consider withholding or discontinuing therapy in patients who develop a clinically significant decrease in kidney function while on FILSPARI.

Hyperkalemia

Monitor serum potassium periodically and treat appropriately. Patients with advanced kidney disease or taking concomitant potassium-increasing drugs (e.g., potassium supplements, potassium-sparing diuretics), or using potassium-containing salt substitutes are at increased risk for developing hyperkalemia. Dosage reduction or discontinuation of FILSPARI may be required.

Fluid Retention

Fluid retention may occur with endothelin receptor antagonists and has been observed in clinical studies with FILSPARI. FILSPARI has not been evaluated in patients with heart failure.

If clinically significant fluid retention develops, evaluate the patient to determine the cause and the potential need to initiate or modify the dose of diuretic treatment then consider modifying the dose of FILSPARI.

ADVERSE REACTIONS

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

The safety of FILSPARI was evaluated in PROTECT (NCT03762850), a randomized, double-blind, active-controlled clinical study in adults with IgAN.

The data below reflect FILSPARI exposure in 202 patients with a median duration of 110 weeks.

The most common adverse reactions are presented in the table below.

Adverse Reactions Reported in 2% or More of Subjects Treated with FILSPARI

	FILSPARI (N = 202) n (%)	Irbesartan (N = 202) n (%)
Hyperkalemia[1]	34 (17)	27 (13)
Hypotension (including orthostatic hypotension)	33 (16)	13 (6)
Peripheral edema[1]	33 (16)	29 (14)
Dizziness[1]	32 (16)	14 (7)
Anemia	16 (8)	9 (4)
Acute kidney injury	12 (6)	5 (2)
Transaminase elevations[2]	7 (3.5)	8 (4.0)

[1] Includes related terms.

[2] Elevations in ALT or AST greater than 3-fold ULN.

Laboratory Tests

Initiation of FILSPARI may cause an initial small decrease in estimated glomerular filtration rate (eGFR) that occurs within the first 4 weeks of starting therapy and then stabilizes.

The incidence of a hemoglobin decrease >2 g/dL compared to baseline and below the lower limit of normal was greater for the FILSPARI arm (11%) compared to the irbesartan arm (5%). This decrease is thought to be in part due to hemodilution. There were no treatment discontinuations due to anemia or hemoglobin decrease in the PROTECT study.

DRUG INTERACTIONS

Renin-Angiotensin System (RAS) Inhibitors and ERAs

Do not coadminister FILSPARI with ARBs, ERAs, or aliskiren.

Combined use of these agents is associated with increased risks of hypotension, syncope, hyperkalemia, and changes in renal function (including acute renal failure).

Strong and Moderate CYP3A Inhibitors

Avoid concomitant use of FILSPARI with strong CYP3A inhibitors. If a strong CYP3A inhibitor cannot be avoided, interrupt treatment with FILSPARI. When resuming treatment with FILSPARI, consider dose titration.

Monitor blood pressure, serum potassium, edema, and kidney function regularly when used concomitantly with moderate CYP3A inhibitors. No FILSPARI dose adjustment is needed.

Sparsentan is a CYP3A substrate. Concomitant use with a strong CYP3A inhibitor increases sparsentan C_{max} and AUC, which may increase the risk of FILSPARI adverse reactions.

Strong CYP3A Inducers

Avoid concomitant use with a strong CYP3A inducer. Sparsentan is a CYP3A substrate. Concomitant use with a strong CYP3A inducer decreases sparsentan C_{max} and AUC, which may reduce FILSPARI efficacy.

Antacids and Acid Reducing Agents

Administer FILSPARI 2 hours before or after administration of antacids. Avoid concomitant use of acid reducing agents (histamine H2 receptor antagonist and PPI proton pump inhibitor) with FILSPARI. Sparsentan exhibits pH-dependent solubility. Antacids or acid reducing agents may decrease sparsentan exposure which may reduce FILSPARI efficacy.

Non-Steroidal Anti-Inflammatory Agents (NSAIDs), Including Selective Cyclooxygenase-2 (COX-2) Inhibitors

Monitor for signs of worsening renal function with concomitant use with NSAIDs (including selective COX-2 inhibitors). In patients with volume depletion (including those on diuretic therapy) or with impaired kidney function, concomitant use of NSAIDs (including selective COX-2 inhibitors) with drugs that antagonize the angiotensin II receptor may result in deterioration of kidney function, including possible kidney failure. These effects are usually reversible.

CYP2B6, 2C9, and 2C19 Substrates

Monitor for efficacy of the concurrently administered CYP2B6, 2C9, and 2C19 substrates and consider dosage adjustment in accordance with the Prescribing Information. Sparsentan is an inducer of CYP2B6, 2C9, and 2C19. Sparsentan decreases exposure of these substrates, which may reduce efficacy related to these substrates.

P-gp and BCRP Substrates

Avoid concomitant use of sensitive substrates of P-gp and BCRP with FILSPARI. Sparsentan is an inhibitor of P-gp and BCRP. Sparsentan may increase exposure of these transporter substrates, which may increase the risk of adverse reactions related to these substrates.

Agents Increasing Serum Potassium

Monitor serum potassium frequently in patients treated with FILSPARI and other agents that increase serum potassium. Concomitant use of FILSPARI with potassium-sparing diuretics, potassium supplements, potassium-containing salt substitutes, or other drugs that raise serum potassium levels may result in hyperkalemia.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

Based on data from animal reproductive toxicity studies, FILSPARI can cause fetal harm, including birth defects and fetal death, when administered to a pregnant patient and is contraindicated during pregnancy. Available data from reports of pregnancy in clinical trials with FILSPARI are insufficient to identify a drug-associated risk of major birth defects, miscarriage, or other adverse maternal or fetal outcomes. In animal reproduction studies, oral administration of sparsentan to pregnant rats throughout organogenesis at 10-times the maximum recommended human dose (MRHD) in mg/day caused teratogenic effects in rats, including craniofacial malformations, skeletal abnormalities, increased embryo-fetal lethality, and reduced fetal weights. Advise pregnant patients of the potential risk to the fetus.

The background risk of major birth defects and miscarriage for the indicated population is unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2-4% and 15-20%, respectively.

Lactation

Risk Summary

There are no data on the presence of sparsentan in human milk, the effects on the breastfed infant, or the effect on milk production. Because of the potential for adverse reactions, such as hypotension in breastfed infants, advise patients not to breastfeed during treatment with FILSPARI.

Females and Males of Reproductive Potential

Based on data from animal reproductive toxicity studies, FILSPARI can cause fetal harm, including birth defects and fetal death, when administered to a pregnant patient and is contraindicated during pregnancy.

Pregnancy Testing

Verify that patients who can become pregnant are not pregnant prior to initiating FILSPARI, monthly during treatment, and one month after discontinuation of treatment. The patient should contact their physician immediately for pregnancy testing if onset of menses is delayed or pregnancy is suspected. If the pregnancy test is positive, the physician and patient must discuss the risks to their pregnancy and the fetus.

Contraception

Patients who can become pregnant who are using FILSPARI must use an effective method of contraception prior to initiation of treatment, during treatment, and for one month after discontinuation of treatment with FILSPARI to prevent pregnancy.

Pediatric Use

The safety and efficacy of FILSPARI in pediatric patients have not been established.

Geriatric Use

Of the total number of subjects in the PROTECT study of FILSPARI, 15 (7.4%) were 65 and older. No overall differences in safety or effectiveness were observed between these subjects and younger subjects.

Hepatic Impairment

Avoid use of FILSPARI in patients with any hepatic impairment (Child-Pugh class A-C) because of the potential risk of serious liver injury.

OVERDOSAGE

There is no experience with overdose with FILSPARI. Sparsentan has been given in doses up to 1600 mg/day in healthy volunteers, or up to 400 mg/day in patients. Overdose of FILSPARI may result in decreased blood pressure. In the event of an overdose, standard supportive measures should be taken, as required. Dialysis is unlikely to be effective because sparsentan is highly protein-bound.

PATIENT COUNSELING INFORMATION

Advise patients to read the FDA-approved patient labeling (Medication Guide).

Restricted access

Advise the patient that FILSPARI is only available through a restricted access program called the FILSPARI REMS.

As a component of the FILSPARI REMS, prescribers must review the contents of the FILSPARI Medication Guide with the patient before initiating FILSPARI.

Instruct patients that the risks associated with FILSPARI include:

Hepatotoxicity

Advise patients with symptoms suggesting hepatotoxicity (nausea, vomiting, right upper quadrant pain, fatigue, anorexia, jaundice, dark urine, fever, or itching) to immediately stop taking FILSPARI and seek medical attention.

Embryo-Fetal Toxicity

Educate and counsel patients who can become pregnant about the need to use reliable methods of contraception prior to treatment with FILSPARI, during treatment and for one month after treatment discontinuation. Patients who can become pregnant must have pregnancy tests prior to treatment with FILSPARI, monthly during treatment, and one month after treatment discontinuation.

Patients should be instructed to immediately contact their physician if they suspect they may be pregnant. Patients should seek additional contraceptive advice from a gynecologist or similar expert as needed.

Educate and counsel patients who can become pregnant on the use of emergency contraception in the event of unprotected sex or contraceptive failure.

Advise patients to contact their gynecologist or healthcare provider if they want to change the form of birth control which is used to ensure that another acceptable form of birth control is selected.

Advise the patient that FILSPARI is available only from certified pharmacies that are enrolled in the FILSPARI REMS.

Patients must sign the FILSPARI REMS Patient Enrollment Form to confirm that they understand the risks of FILSPARI.

Lactation

Advise patients not to breastfeed during treatment with FILSPARI.

Drug Interactions

Advise patients to inform their healthcare provider of all concomitant medications, including prescription medications, over-the-counter drugs, vitamins/supplements, herbal products, and grapefruit.

Other Risks Associated with FILSPARI

Inform patients of other risks associated with FILSPARI, including:

- Hypotension: Advise patients to remain hydrated.
- Hyperkalemia: Advise patients not to use potassium supplements or salt substitutes that contain potassium without consulting their healthcare provider.

This information is not comprehensive. Visit FILSPARI.com or call 1-877-659-5518 to obtain the full Prescribing Information.

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TRAVERE® THERAPEUTICS

Multidisciplinary Dialogue Is Needed to Achieve Whole-Person Quality Measures for Dialysis Care

By Sonia Rodriguez-Ramirez, Asad A. Merchant, and Istvan Mucsi

A recent article summarizing outcomes from the 2022 Kidney Disease Outcomes Quality Initiative (KDOQI) Workshop on Patient-Centered Quality Measures for Dialysis Care (1) explores the need for a more patient-centered dialysis quality system in alignment with the US Institute of Medicine Committee on Quality of Health Care in America describing it as: respectful of and responsive to individual patient preferences, needs, and values (2, 3). The authors, who were participants in the KDOQI workshop, suggest including patient priorities and life goals in dialysis quality systems. They also highlight current gaps, particularly in the End-Stage Renal Disease Quality Incentive Program, which focuses on biochemical and administrative outcomes but needs to capture patient-reported outcomes and experiences (1).

The proposal to incorporate a "life goal" quality measure is a notable innovation. The authors of the report (1) acknowledge potential conflicts between individualized care and population-level quality measures. To address this, ongoing dialogue between stakeholders and a periodic revision of goals and care strategies will be required (4, 5).

Another significant insight is the incorporation of social determinants of health into quality measures. However, data collection challenges, patient privacy, and the administrative burden on facilities are substantial. Although the workshop included a broad spectrum of stakeholders, perspectives from marginalized groups likely need better representation (6), which will require proactive outreach, intentional inclusion efforts, and mechanisms to amplify marginalized voices beyond consensus processes (7).

The recommendations for improving the collection and utilization of patient-reported data are particularly valuable. These recommendations can address the current issues of low response rates and delayed feedback and may want to include qualitative and narrative methods which may better capture the complexity and diversity of illness experiences (8–10).

In conclusion, the report provides a thorough and insightful critique of current dialysis quality measures, highlighting the need for a more patient-centered approach. However, it is worth noting that the systems described and approaches considered are unique to the US health care context. There is a clear tension between the quantitative, reductionist, and administrative perspective and the spirit of person- and community-centered, whole-person care. To build more person- and community-centered, whole-person care, we will require a flexible and dynamic quality system that balances the needs of individual patients with population health goals (Figure). To achieve this, an inclusive, participatory, interprofessional, and multidisciplinary dialogue will be needed, and inviting perspectives from other jurisdictions may be helpful. ■

Sonia Rodriguez-Ramirez, MD, MScHQ, and Asad A. Merchant, MD, MScCH, FRCP(C), are assistant professors of medicine, and Istvan Mucsi, MD, PhD, FASN, is a professor of medicine with the Division of Nephrology and Ajmera Transplant Centre, University Health Network, and with the Temerty Faculty of Medicine, University of Toronto, Ontario, Canada.

The authors report no conflicts of interest.

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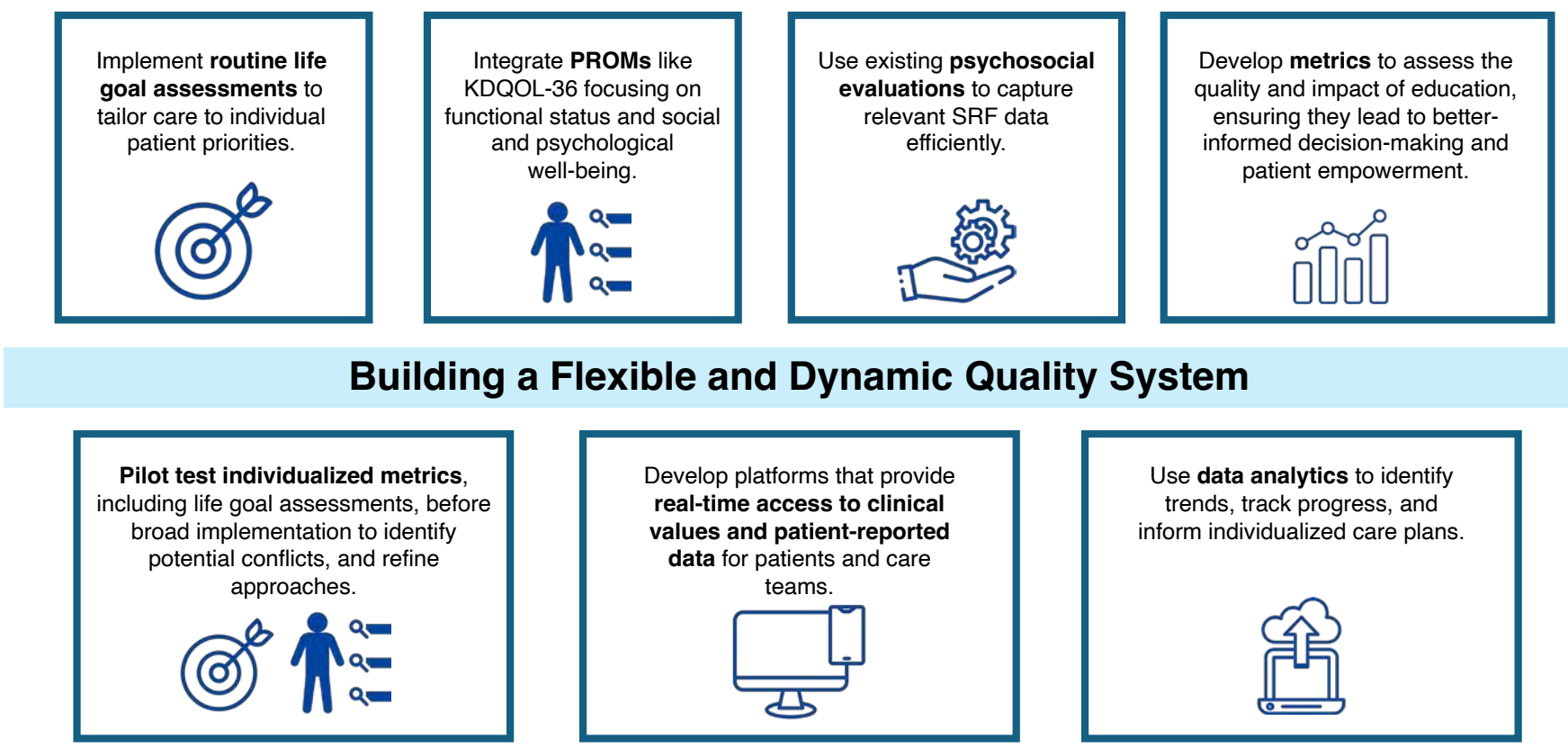
Figure. Factors in a whole-person quality system for dialysis care



KDQOL-36, Kidney Disease Quality of Life survey; PROMs, patient-reported outcome measures; SRF, social risk factor.

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*Velphoro 500 mg tablet; sevelamer 800 mg tablet. Based on an analysis of comparative studies that roughly established an equivalent dose for phosphate binders relative to the phosphate binding capacity of calcium carbonate.[1]

†Percentage of patients at goal increased from 21.2% at baseline with sevelamer (8.4 pills/day) to 41.1% at 1 year after a switch to Velphoro (4.9 pills/day); *P*<.0001.[2]

‡A retrospective analysis of deidentified pharmacy data from 1,792 adult in-center hemodialysis patients who were switched to Velphoro during routine care between May 2018 and May 2019. Subset evaluated included patients taking sevelamer at baseline (n=841). The decision to discontinue baseline phosphate binders (PBs) and switch to Velphoro was made on a clinical basis and the reasons underlying this change were not available. Comparisons were made between the 91-day period before Velphoro initiation (ie, baseline) and the 4 consecutive 91-day intervals of Velphoro treatment (Q1-Q4). Main outcome measures included achievement of target phosphorus levels (≤5.5 mg/dL) and mean number of PB pills/day.[2]

INDICATION

Velphoro® (sucroferric oxyhydroxide) is a phosphate binder indicated for the control of serum phosphorus levels in adult and pediatric patients 9 years of age and older with chronic kidney disease on dialysis.

IMPORTANT SAFETY INFORMATION

- Velphoro chewable tablets must be administered with meals. Velphoro should be chewed or crushed. Do not swallow whole.
- Patients with peritonitis during peritoneal dialysis, significant gastric or hepatic disorders, following major gastrointestinal (GI) surgery, or with a history of hemochromatosis or other diseases with iron accumulation have not been included in clinical studies with Velphoro. Monitor effect and iron homeostasis in such patients.
- In a parallel design, fixed-dose study of 6 weeks duration, the most common adverse drug reactions to Velphoro chewable tablets in hemodialysis patients included discolored feces (12%) and diarrhea (6%).
- Velphoro can be administered concomitantly with oral calcitriol, ciprofloxacin, digoxin, enalapril, furosemide, HMG-CoA reductase inhibitors, hydrochlorothiazide, losartan, metoprolol, nifedipine, omeprazole, quinidine and warfarin. For oral medications where a reduction of bioavailability would be clinically significant consider separating of the timing of administration. Consider monitoring clinical responses or blood levels of the concomitant medications.

Velphoro is available by prescription only. For additional Safety Information, please see full Prescribing Information.

To report SUSPECTED ADVERSE REACTIONS, contact Fresenius Medical Care Customer Service at 1-800-323-5188 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

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FRESENIUS MEDICAL CARE
RENAL PHARMACEUTICALS

Distributed by: Fresenius Medical Care North America, Waltham, MA 02451

US-Velphoro-000086 Rev A 08/2024

VELPHORO®
(sucroferric oxyhydroxide)
chewable tablets

TAKE CHARGE OF PHOSPHATE MANAGEMENT

Brief Summary:
Please see Full Prescribing Information for additional information

VELPHORO®
(sucroferric oxyhydroxide)
chewable tablets

INDICATIONS AND USAGE
Velphoro (sucroferric oxyhydroxide) is a phosphate binder indicated for the control of serum phosphorus levels in patients with chronic kidney disease on dialysis.

DOSAGE AND ADMINISTRATION
Velphoro tablets must be chewed and not swallowed whole. To aid with chewing and swallowing, tablets may be crushed.

The recommended starting dose of Velphoro is 3 tablets (1,500 mg) per day, administered as 1 tablet (500 mg) 3 times daily with meals.

Adjust by 1 tablet per day as needed until an acceptable serum phosphorus level is reached, with regular monitoring afterwards. Titrate as often as weekly.

DOSAGE FORMS AND STRENGTHS
Velphoro (sucroferric oxyhydroxide) chewable tablet 500 mg.

CONTRAINDICATIONS
None.

WARNINGS AND PRECAUTIONS
Patients with peritonitis during peritoneal dialysis, significant gastric or hepatic disorders, following major gastrointestinal surgery, or with a history of hemochromatosis or other diseases with iron accumulation have not been included in clinical studies with Velphoro. Monitor effect and iron homeostasis in such patients.

ADVERSE REACTIONS
In a parallel design, fixed-dose study of 6 weeks duration, the most common adverse drug reactions to Velphoro chewable tablets in hemodialysis patients included discolored feces (12%) and diarrhea (6%).

The following adverse reactions were identified during post approval use of Velphoro and were reported voluntarily from a population of uncertain size.

Gastrointestinal Disorders: tooth discoloration

Skin and Subcutaneous Tissue Disorder: rash

To report SUSPECTED ADVERSE REACTIONS, contact Fresenius Medical Care North America at 1-800-323-5188 or FDA at 1-800-FDA-1088 or *www.fda.gov/medwatch*.

DRUG INTERACTIONS
Velphoro can be administered concomitantly with oral calcitriol, ciprofloxacin, digoxin, enalapril, furosemide, HMG-CoA reductase inhibitors, hydrochlorothiazide, losartan, metoprolol, nifedipine, omeprazole, quinidine and warfarin.

Take acetylsalicylic acid, cephalexin and doxycycline at least 1 hour before Velphoro.

Take levothyroxine at least 4 hours before Velphoro.

For oral medications not listed above where a reduction of bioavailability would be clinically significant consider separation of the timing of administration. Consider monitoring clinical responses or blood levels of the concomitant medication.

USE IN SPECIFIC POPULATIONS

Pregnancy
Pregnancy Category B: Reproduction studies have been performed in rats and rabbits at doses up to 16 and 4 times, respectively, the human maximum recommended clinical dose on a body weight basis, and have not revealed evidence of impaired fertility or harm to the fetus due to Velphoro. However, Velphoro at a dose up to 16 times the maximum clinical dose was associated with an increase in post-implantation loss in pregnant rats. Animal reproduction studies are not always predictive of human response.

There are no adequate and well-controlled studies in pregnant women.

Labor and Delivery
No Velphoro treatment-related effects on labor and delivery were seen in animal studies with doses up to 16 times the maximum recommended clinical dose on a body weight basis. The effects of Velphoro on labor and delivery in humans are not known.

Nursing Mothers
Since the absorption of iron from Velphoro is minimal, excretion of Velphoro in breast milk is unlikely.

Pediatric Use
The safety and efficacy of Velphoro have not been established in pediatric patients.

Geriatric Use
Of the total number of subjects in two active-controlled clinical studies of Velphoro (N=835), 29.7% (n=248) were 65 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects.

OVERDOSAGE
There are no reports of overdosage with Velphoro in patients. Since the absorption of iron from Velphoro is low, the risk of systemic iron toxicity is low. Hypophosphatemia should be treated by standard clinical practice.

Velphoro has been studied in doses up to 3,000 mg per day.

HOW SUPPLIED/STORAGE AND HANDLING
Velphoro are chewable tablets supplied as brown, circular, bi-planar tablets, embossed with “PA 500” on 1 side. Each tablet of Velphoro contains 500 mg iron as sucroferric oxyhydroxide. Velphoro tablets are packaged as follows:

NDC 49230-645-51 Bottle of 90 chewable tablets

Storage
Keep the bottle tightly closed in order to protect from moisture.

Store at 25°C (77°F) with excursions permitted to 15 to 30°C (59 to 86°F).

PATIENT COUNSELING INFORMATION
Inform patients that Velphoro tablets should be chewed or crushed. Do not swallow whole *[see Dosage and Administration]*.

Velphoro should be taken with meals.

Instruct patients on concomitant medications that should be dosed apart from Velphoro *[see Drug Interactions]*.

Inform patients that Velphoro can cause discolored (black) stool, but this discoloration of the stool is considered normal with oral medications containing iron.

Inform patients that Velphoro can stain teeth.

Inform patients to report any rash to their healthcare professional.

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FRESENIUS MEDICAL CARE
RENAL PHARMACEUTICALS

PN 105108-01 Rev A 04/2022

Targeting CD38 in Acute Antibody-Mediated Rejection: A New Hope for Kidney Transplant Recipients?

By Jennifer S. Y. Li and Germaine Wong

Acute antibody-mediated rejection (ABMR) is a significant cause of graft loss in kidney transplant recipients (1–3). Despite its impact, effective treatments are lacking. Recently, targeting CD38 has garnered attention as a therapeutic option for ABMR (4). CD38 is a transmembrane glycoprotein and is expressed on immune cells (5–7) (such as natural killer [NK] cells, plasma cells, B cells, T cells, and macrophages) and nonimmune cells (such as red blood cells and endothelial cells) (8, 9). CD38 expression is upregulated by inflammatory stimuli (e.g., interferon γ [8–10] and nuclear factor κB [11]) and also has immunomodulatory functions through its dual roles as a receptor for CD31 (or platelet-endothelial cell adhesion molecule 1) and as an ectoenzyme regulating nicotinamide metabolism and calcium signaling (Figure) (12). Binding to CD38 can induce cell death through mechanisms including Fc-mediated cross-linking, complement-dependent cytotoxicity, antibody-dependent cell toxicity, and cellular phagocytosis.

A recently published phase 2 randomized controlled trial by Mayer et al. reports promising results for felzartamab, a humanized monoclonal anti-CD38 antibody, in treating ABMR (13). This trial included adult kidney transplant recipients with biopsy-confirmed ABMR diagnosed at least 6 months after transplant, with a detectable donor-specific antibody (DSA) and an estimated glomerular filtration rate ≥20 mL/min/1.73 m^2. A total of 22 patients were recruited and randomly assigned with a 1:1 ratio to receive felzartamab or placebo over a 20-week treatment period, with follow-up extending to 52 weeks. Randomization was stratified by site and categories of ABMR. The primary outcomes were safety and an adverse-effects profile. Secondary outcomes included resolution of ABMR, change in the immunodominant mean fluorescence intensity of DSA, and other biomarkers such as NK cell counts.

Overall, the study found a greater incidence of adverse effects in the treatment arm compared with placebo (119 vs 81 events), predominately driven by infections and infusion-related reactions. However, no deaths were reported. At 24 weeks, patients who received felzartamab showed greater histological resolution of ABMR compared with those receiving placebo. However, at the end of 52 weeks, three of nine patients assigned to the treatment arm experienced a recurrence of ABMR. Notably, there was no indication of increased T cell-mediated rejection, which could be a theoretical concern, as CD38 is also expressed on regulatory T cells. Additionally, there were no cases of allograft loss at 1 year postrandomization, and findings of all other biochemical markers and changes in the mean fluorescence intensity of DSA were relatively similar between the two groups.

Felzartamab appears to be a safe and promising agent for the treatment of ABMR. A large, well-powered and well-designed phase 3 study is underway. If proven effective, felzartamab may offer a novel approach to managing ABMR and improving long-term patient and graft outcomes. ■

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The authors report no conflicts of interest.

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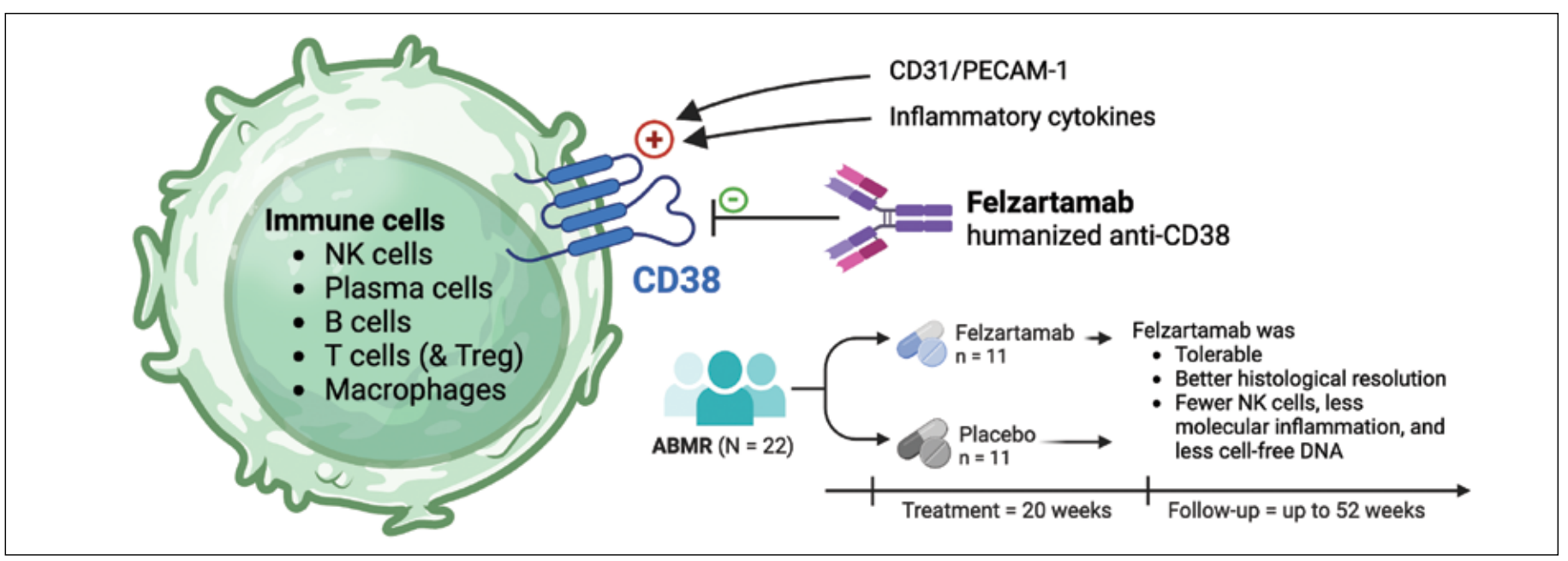
Figure. Felzartamab targets CD38 to treat acute antibody-mediated rejection in kidney transplant recipients



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PECAM-1, platelet-endothelial cell adhesion molecule 1; Treg, regulatory T cell.

ASN Comments on Proposed Rules Governing Payment and Quality Measurement in the ESRD Program

By David L. White

Every summer, the Centers for Medicare & Medicaid Services (CMS) releases the proposed End-Stage Renal Disease (ESRD) Prospective Payment System (PPS) and Quality Incentive Program (QIP) rule that will govern payment and quality measurement in the ESRD program in the calendar year (CY) ahead. This summer was no different, so ASN recommended adjustments to the proposal during the 60-day comment period required by law under the Administrative Procedure Act, which governs rulemaking at the federal level. Currently, more than 800,000 Americans experience kidney failure, including more than 550,000 receiving dialysis and more than 200,000 living with a kidney transplant.

As always, ASN takes advantage of the open comment process to refocus the administration on the vast impact of kidney diseases on 37 million Americans and their family members. It is also an important opportunity to underscore the fact that kidney diseases and kidney failure disproportionally impact historically marginalized populations and minorities, including those who are American Indian or Alaska Native, Asian, Black, Hispanic or Latinx, and Native Hawaiian and Other Pacific Islander; people with lower incomes; and older adults, underlying and exacerbating existing disparities. These and other factors explain why it is critical that the CMS ESRD program promotes equitable access to optimal kidney care.

The proposals discussed in ASN's letter (1) and this article are expected to be finalized around the same time as Kidney Week 2024.

AKI site of care

After several years of advocacy by ASN, CMS proposed to extend the home dialysis benefit to individuals with acute kidney injury (AKI) for either peritoneal dialysis (PD) or home hemodialysis (HD). For patients with AKI requiring kidney replacement therapy, ASN advocated for allowing home dialysis as patients transition to home (from hospitals or postacute or in-center transitionary dialysis facilities), and the corresponding Medicare payment should be allowed when the nephrologist and patient agree that a particular patient with AKI can safely dialyze at home.

ASN views home therapy as supervised care that is of at least similar quality and intensity to in-center HD and highlighted the commitment to ensuring the success of all patients with AKI requiring dialysis (AKI-D), regardless of whether they are receiving dialysis in the home or in an HD facility. In these circumstances, intensive training for home dialysis should also be reimbursed by Medicare, via the addition of Current Procedural Terminology training codes 90989 and 90933 being added to the telehealth list. (Because there is no relative value unit attached to these codes, clinicians who are dependent on the relative value unit system to quantify their clinical work have a difficult time receiving credit for the work that they perform in the supervision of home dialysis training.)

ASN expressed concerns regarding an Add-on Payment Adjustment for Training of patients with AKI being added to the same payment as the proposed payment amount in 2025 for in-center dialysis: $273.20. However, CMS also proposed to extend an add-on payment adjustment for home and self-dialysis training at the same rate as patients with ESRD, on a budget-neutral basis, which results in a proposed AKI CY 2025 base rate (for all dialysis modalities) of $264.70 ($273.20 – $8.50, with $8.50 being the estimated add-on training adjustment). The problem with the adjustment, ASN pointed out, was CMS' math. The agency assumed that the number of patients with AKI going to home dialysis would be the same rate as all patients with ESRD receiving home dialysis in the fourth quarter of 2022: 15.4%. ASN asserted to CMS that "It is highly unlikely that the AKI home dialysis rate will equal the overall ESRD home dialysis rate, especially not in the first years of this new policy." Nephrologists evaluating the proposed rule felt that it is highly unlikely that there will be a significant number of individuals with AKI initiating home PD. ASN expressed concern that such an out-of-portion payment adjustment could impact modality choices for these patients for whom PD is an important patient-centered option. Patients with AKI-D are reimbursed lower than incident patients with ESRD, reflecting noninclusion of the incident patient modifier. While this is required by law, it emphasizes

that further deductions from the reimbursement for HD for AKI-D would be financially unviable.

ASN volunteered its AKINow Committee to work with CMS on accurate predictions of the uptake of home dialysis for AKI-D. Additionally, CMS correctly pointed out that "ESRD" and "AKI" are not interchangeable and that the Conditions for Coverage (CfCs) for dialysis facilities need to be aligned with the changes proposed in the rule. ASN believes that CfCs need updating (last updated in 2008) and urged CMS to engage the kidney care community in a broader dialogue on a range of potential updates to CfCs.

Dialysis bundled payment shortcomings

ASN expressed strong concern over an inadequate Medicare bundled payment for dialysis. In its March "Report to the Congress," the Medicare Payment Advisory Commission (MedPAC) estimated a margin of zero for 2024 (2). MedPAC's finding means that there are many facilities with a margin below zero. Given the significantly increasing costs, it poses challenges for many facilities to be able to adjust to unexpected events when they occur or, in some cases, to be able to continue providing services at their historic levels. A decrease in access to dialysis presents a grave concern for all patients regardless of payor. Other concerns identified by ASN in the proposed rule include the following:

1. **CMS proposed using the outlier policy as payment policy for innovation**. ASN expressed alarm that CMS' proposal to address innovative payment in the proposed rule by expanding products eligible for outlier payments did not represent a sustainable ESRD PPS policy for adequate funding for innovative drugs, biologics, and devices as covered by the Transitional Drug Add-on Payment Adjustment (TDAPA) under the ESRD PPS for certain new renal dialysis drugs and biological products. As MedPAC stated, the outlier policy is essentially stop-loss insurance, and it is not meant to establish accurate and adequate payment for new medically necessary services.

ASN recommended that CMS support the approach in the proposal outlined in section 201 of Senate bill 4469, the "Chronic Kidney Disease Improvement in Research and Treatment Act of 2024" (3). This proposal would require CMS in a nonbudget neutral manner to:

Continued on page 60 >

ASN Comments on Proposed CMS Rules

Continued from page 59

- Establish a permanent post-TDAPA to the base rate for a new drug or biological product that comes within an existing functional category.
- Calculate the post-TDAPA using the most recent 12-month period of utilization data for the product and the most recent available full calendar quarter of average sales price (ASP).
- Update the adjustment amount annually to account for inflationary changes.
- Apply the adjustment amount immediately upon the expiration of the TDAPA period.

ASN maintains that the ESRD PPS as currently structured stifles innovation for a population that already experiences extreme health disparities and issues of access. As a result of recent policies, patients with chronic kidney disease-associated pruritus are not able to access the only US Food and Drug Administration-approved treatment indicated specifically to treat this disease. The manufacturer of difelikefalin (Korsuva) has indicated it will cease all research and development in the area of chronic kidney disease, reflecting low use due to payment policy.

2 **Orals in the ESRD PPS bundle present challenges.** ASN has repeatedly voiced its concerns to CMS about including oral-only phosphate binders and other phosphate-lowering drugs in the ESRD PPS for many reasons and supports efforts in Congress to delay that occurrence, but CMS again requested comments on a payment approach. To maintain consistency with the treatment of calcimimetics during their first 2 years of TDAPA, to align with the way Medicare reimburses for drugs and biologics under the Hospital Outpatient PPS's pass-through policy, and to minimize administrative burden on CMS and care facilities, ASN recommended that CMS adopt the methodology outlined in the Social Security Act section 1847A (4). This methodology sets payment at ASP + 6%; if ASP is not available, the payment is based on the wholesale acquisition cost.

ASN has made clear to CMS that it believes that adding phosphate binders and phosphate-lowering drugs to the bundle will have a negative impact on patients. ASN emphasized that phosphate binders and phosphate-lowering drugs must be taken outside of the facility, typically when a patient eats. Congress has recognized the challenges of including these drugs in the ESRD PPS when it has repeatedly restricted CMS from adding them. Furthermore, there will not be an actionable solution present for the distribution of phosphate-lowering agents to patients residing in nursing homes or other facilities.

3 **There is also the problem of no dispensing fees.** CMS recognized in the proposed rule's preamble that "dispensing fees and other costs are not currently included in the ESRD PPS base rate for phosphate binders" (5). As the Government Accountability Office found in its 2023 report, dialysis facilities will incur significant costs that are not included in the base rate if phosphate binders are added to the ESRD PPS bundled reimbursement (6). These costs include the following:

- paying pharmacy charges to obtain the drugs through them
- mailing fees either in terms of obtaining the drugs from pharmacies or sending the drugs directly to patients' homes, which is where they are taken
- incurring storage costs associated with maintaining the drugs at the dialysis facility if the decision is to distribute the drugs to patients during their dialysis treatment sessions
- complying with state pharmacy laws; for example, some states, like Alabama, do not allow dialysis facilities to distribute oral drugs, so there are additional contracting costs incurred
- supporting the provision of a significant volume of pills to patients so that they have the amount they need at every meal and snack
- adjusting drug supplies when a physician changes a patient's prescription to another product (which often occurs)
- absorbing costs of unused medications when patients are hospitalized, transfer to other facilities, die, or receive a kidney transplant

ASN detailed the differing policies within CMS programs and recommended that CMS adopt the straightforward and transparent ASP + 6% policy that it relies on in other parts of the Medicare program. As an alternative, CMS could consider using the same flat rate supply fee used for other oral Part B drugs that are supplied as part of a physician service.

QIP

CMS added three new health equity-focused quality measures in the CY 2024 ESRD PPS final rule (88 FR 76437–76446 and 76466–76480) to ESRD QIP (5). CMS is now soliciting feedback on the creation of a Health Equity Adjustment. ASN applauded CMS for its efforts to improve health equity among the population with ESRD receiving facility-based dialysis, given longstanding disparities among this population. ASN posed questions to CMS about the efficacy and logistics of the proposal. It remains unclear if CMS will delay this effort to provide time to improve the proposal.

ASN supported CMS' proposal to disaggregate the Kt/V Dialysis Adequacy measure into four distinct components, each evaluated against its own performance standards. ASN's analysis found that the approach acknowledges the complexity of dialysis adequacy and allows for a more nuanced assessment. By distinguishing

among various aspects of Kt/V, this proposal facilitates a more accurate reflection of patient-specific needs and treatment efficacy. However, ASN has longstanding concerns about the application of Kt/V in assessing PD adequacy, particularly considering existing guidelines and patient outcomes.

ASN continued to express concern over the number of measures in QIP particularly the following:

1. **In-Center Hemodialysis Consumer Assessment of Healthcare Providers and Systems (ICH CAHPS) Survey Administration (clinical measure).** ASN continues to express concern over the low response rate to ICH CAHPS. To reduce the burden on patients, ASN requested the form be shortened and that CMS field the survey once a year and not twice. Finally, to empower patients, ASN encouraged CMS to allow facilities to see deidentified results of the surveys so that they can respond to the specific patient concerns with some level of patient permission. Patient members of several technical expert panels have recommended this step.
2. **Standard Readmission Ratio (SRR) (clinical measure).** ASN remains concerned that SRR might mislead patients, care partners, and health care practitioners due to its wide confidence interval. This variability can lead to inaccurate facility classifications and fail to accurately reflect actual performance. ASN noted that the current measure also poses challenges for small facilities, as their scores can be heavily influenced by random variability. ASN urged CMS to transition to the use of the underlying readmission rate, which can be properly risk adjusted in the same way that the standardized mortality rate has been and allow within-facility year-to-year comparisons.
3. **Standardized Transfusion Ratio (STrR) (clinical measure).** ASN expressed concerns that the STrR measure lacks validity and believes it should be suppressed. ASN, while appreciative that CMS has acknowledged this concern, remained troubled that CMS has not addressed the low reliability of the data on transfusions.
4. **Standardized Hospitalization Ratio (clinical measure).** ASN agreed that hospitalization rates are crucial indicators of quality for both patients and facilities but also strongly urged CMS to implement a genuinely risk-standardized hospitalization rate measure to prevent misclassifying facilities and misleading patients.
5. **Clinical Depression Screening and Follow-up (reporting measure).** ASN recognized that identifying and treating mental health conditions among patients receiving dialysis are critical to ensuring optimal health and clinical outcomes. ASN expressed major concerns about the ability of dialysis units to treat depression in isolation, without additional support and resources. As a first step, ASN proposed that CMS consider clarifying opportunities for and supporting expanded access to mental health services that can occur either on-site in the dialysis facility (e.g., in a private room before or after treatments) or via telemedicine for patients on dialysis.
6. **National Healthcare Safety Network (NHSN) Dialysis Event (reporting measure).** ASN supports CMS' proposal to remove the NHSN Dialysis Event reporting measure from the ESRD QIP measure set beginning with payment year 2027.
7. **NHSN Bloodstream Infection (BSI) in Patients on HD (clinical measure).** Research from the Centers for Disease Control and Prevention, the measure's developer, as well as from CMS and other sources, indicates that the measure lacks both validity and reliability. Previously, ASN has recommended that CMS transition the NHSN BSI measure to a reporting measure while forming a technical expert panel to address its shortcomings.
8. **Screen Positive Rate for Social Drivers of Health (reporting measure).** ASN applauds CMS' commitment to addressing health care disparities and supporting these measure concepts. ASN strongly supports the implementation of screening measures for social drivers of health for patients on dialysis, recognizing their potential to improve patient care. However, ASN also encouraged CMS to evaluate the impact of public reporting of the percentage of patients in each dialysis facility who screen positive in various domains. ASN fears that this publicity may lead patients to either avoid answering or provide inaccurate responses, especially within the close-knit environment of a dialysis facility.

To keep track of ASN's policy efforts related to these proposals, follow coverage in *Kidney News* and the ASN podcast feed, and visit ASN's policy webpage (http://www.asn-online.org/policy). For real-time updates from ASN Policy, follow @ASNAdvocacy on X. ■

David L. White is the regulatory and quality officer at ASN.

Summary of ASN recommendations in its CY 2025 comment letter (1)

- Finalize site of care proposal for individuals with AKI.
- Revise proposed Add-on Payment Adjustment for Training.
- Support proposed conditions for coverage for dialysis facilities but need to go further.
- Address dialysis bundled payment shortcomings.
  - Payment policy for innovation does not equate to the outlier policy—do not finalize.
  - Dispensing fees for orals in the bundle are needed if oral-only agents are included in PPS.
  - The policy of the current base rate does not include dispensing fees for phosphate binders.
- Convene community to improve the proposed Health Equity adjustment.
- Support replacing the Kt/V Dialysis Adequacy comprehensive clinical measure (measuring how much urea is removed during dialysis) with four separate measures.
- Address additional ESRD QIP issues.

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From Transplant Recipients to Podcast Hosts, Two Paths Lead to One Passion for Patient Advocacy

By Lisa Schwartz

Valen Keefer and Cristen Wathen know the extreme ups and downs associated with living with chronic kidney disease. Both women have hereditary polycystic kidney disease (PKD), and both are kidney and liver transplant recipients.

They are on a mission to inspire hope in patients like them, teaming up to produce their new podcast, "Unpacking the Gift of Life." The duo, based in California, shares with *Kidney News* (*KN*) how their unique yet parallel experiences of living with PKD led to their chance friendship and passion for patient advocacy while also providing insight and perspective for professionals in the nephrology field on the physical and emotional complexities of living with chronic illness.

About the cohosts

Diagnosed with PKD at age 10, Keefer had been in and out of hospitals for the better part of her childhood and adolescence before receiving a kidney transplant at 19 years old. Feeling alone on her journey as a teenager compelled her to become a strong advocate for others in similar situations. Fourteen years after her kidney transplant, PKD affected her liver, leading to Caroli syndrome, a rare, inherited disorder associated with the genetic mutations of PKD that cause severe liver complications, including multiple bouts of sepsis. In 2018, Keefer received a life-saving liver transplant. As a double organ recipient, Keefer has been a passionate patient advocate and educator for 20 years, speaking around the country about the importance of including the patient's voice in health care innovation, policy, and practice.

Wathen was diagnosed with PKD in her 20s. By her early 40s, she was a busy licensed counselor, an associate professor in the Counseling Department at Palo Alto University in California, and a mother when the disease started to take its toll. She received the shattering news that not only was her PKD and its associated cysts consuming her kidneys, but, like Keefer, she too had Caroli syndrome, leading to her own kidney and liver transplants. Today, as a mental health professional, she focuses on the emotional well-being of patients living with chronic illness and transplant.

KN: How did you both connect, and what compelled you to start this podcast?

Keefer: When my health declined during my liver issues, I started seeing a hepatologist in California. It turned out [that] the hepatologist was also Cristen's liver specialist, and when she reached out to me, it was perfect timing. We had the same rare manifestation of [Caroli] syndrome and PKD. This connection led to a deep friendship and a shared desire to create authentic conversations through this podcast to support and educate patients and the nephrology community.

Wathen: My extensive online research and motivation to find information and support through my PKD journey led me to Valen's blog and story. I was struck by her vibrancy, passion, and experience with the same challenges [that] I was facing. It gave me hope that I could be okay too. When she wrote about her liver transplant, I knew I had to reach out.

KN: How does your podcast inform the nephrology community?

Keefer: Through open conversations and resources like our podcast, we help the medical community understand patients better. By sharing our experiences and stories, we inspire other patients and show them [that] they're not alone while providing clinicians with the patient perspective needed to influence care and treatment.

Wathen: We provide genuine insight into the emotional aspects of living with chronic illness, dialysis, transplant, and post-transplant life. As a counselor and patient, I have seen first-hand how much power nephrologists and clinicians have in shaping a patient's emotional well-being simply through tone of voice or validation of how a person is feeling. These actions have a profound impact on the patient experience.

KN: What discussions are relevant to nephrologists and the kidney professional community?

Keefer: In season one of the podcast, we discuss the importance of emotional well-being as well as topics like family planning, barriers to health care, living well with chronic disease, and how care [practitioners] can understand and support a patient's plan for the future so [that] they can thrive. The emotional toll of chronic kidney disease is real. A transplant, for instance, is a life-changing experience with a wide range of emotions. While our conversations focus on how we as patients can advocate for ourselves, we need our doctors to advocate for us too. We also emphasize the importance of partnership between patients and the care team as we all work toward the same goal.

Wathen: Doctors and nurses do an excellent job of explaining what to expect pre- and post-transplant. It is equally important for clinicians to check in on the emotional aspects of life before and after transplant. Living with a chronic illness is like having a full-time job [that] you never wanted. Our podcast offers [practitioners] a deeper understanding of the challenges [that] patients face so that we can deal with these complexities together.

KN: How can kidney care teams better educate and inform patients about chronic kidney disease and the transplant process?

Keefer: It's a delicate balance for doctors who try not to overwhelm patients with too much information while still preparing them for what lies ahead. I speak to nephrologists around the country about the fine line between providing too much information and not enough. Patients feel empowered by knowledge, especially when it is presented in a sensitive and personal way. By recognizing that patients need to be mentally prepared for every stage of their journey, the care team can help them take control of their health.

KN: What is one important takeaway from your podcast?

Keefer: In sharing my story, I always emphasize being grounded in gratitude. Transplant patients are overwhelmingly grateful for a second chance at life. By sharing our experiences and engaging in open dialogue, we celebrate the advancements in modern medicine and revel in the hope that future innovations will result in better care for generations to come.

Wathen: Understanding the importance of hope, education, and validating a patient's feelings is very powerful for [the health care team]. This journey is a true partnership, physically and emotionally. ■

"Unpacking the Gift of Life" is a podcast series hosted by Valen Keefer and Cristen Wathen, two transplant survivors and friends sharing stories of hope, health, and emotional well-being along the kidney disease and transplant journey. Listen here: https://unpackingthegiftoflife.buzzsprout.com/2342454.

To learn more about Valen Keefer's lifelong journey with PKD and transplant, visit her website: https://valenkeefer.com/.

Reforms Needed in Nephrology Fellowship Programs to Support Pregnancy and Family Leave

By Suman Behera

It is well known that long and inconsistent working hours and night-call shifts can significantly affect physician trainees' health, and these demands can have adverse effects on maternal and fetal health during pregnancy (1). Unfortunately, one in three medical and surgical fellows had unfavorable pregnancy outcomes during their training period (2). Beyond the physical and emotional toll during pregnancy, taking more than 6 weeks of leave from a fellowship program often results in an extension of the training period, which delays graduation and can affect one's level of compensation. It also delays board certification and can lead to difficulty matching into secondary fellowships.

What can be done to make it more conducive and appealing for individuals in medicine to choose nephrology fellowships? Do we have the right systems and policies in place to provide a supportive environment for individuals who become pregnant as well as for those who become caretakers with childcare needs? A recent survey-based study published in *CJASN* (3) sheds light on these important questions surrounding pregnancy and parenthood during nephrology fellowship.

Policies for parental leave vary across the globe, and the United States does not mandate paid parental leave. As the study demonstrated, nephrology fellows are often unaware of these policies even when they exist (3). Approximately three-fourths of fellows included in the study were unaware of Accreditation Council for Graduate Medical Education (ACGME) parental leave policies, and approximately 4 out of 10 respondents did not know about the duration of parental leave, accommodations for lactation, and access to subsidized childcare.

Surprisingly, one-fourth of the female fellows who took family leave reported experiencing "negative emotions" from fellow trainees upon return (4). Coverage of service obligations during leave is a topic of concern for co-fellows who are juggling their own shifts and responsibilities. Are financial incentives adequate motivators for trainees to cover for their colleagues while on leave? They may be for some, but they do not necessarily alleviate all concerns. Coordinating coverage results in additional shifts for the other fellows, worsening potential burnout and resulting in conflicting schedules with their other clinical and nonclinical responsibilities. However, if a system is built around sharing the coverage for the individual on leave among all nephrologists—both attendings and fellows-in-training—it may decrease the burden for everyone (4).

Additionally, individuals may have anxiety after a prolonged leave of absence, questioning their capabilities and ability to keep up with colleagues, resulting in poorer performance (5). With this in mind, a government initiative in the United Kingdom, called Springboard, aims to support individuals after prolonged leave and restore confidence when returning to practice. It offers a 2-day course for trainees who have taken time out of training for 3 months to 6 years, providing clinical and nonclinical practice support as well as a forum for sharing experiences (4, 5). Similar practical programs would be beneficial if made available in North America.

Medical trainees have work shifts lasting approximately 80 hours per week, and there are no special considerations during pregnancy per ACGME (3). Inconsistent and unconventional working hours pose an additional hindrance in the postpregnancy childcare phase. What may help with this? Should hospital facilities be required to offer in-hospital childcare or daycare centers for fellows/trainees? Some institutions have on-site childcare, but the demand is higher than hospitals can accommodate.

Women in Nephrology also offers a few suggestions to aid nephrologists returning from family leave. To improve participation at conferences, for example, Women in Nephrology suggests that nephrology conferences provide on-site childcare for parents attending the sessions, publicize the availability of breastfeeding/lactation areas at the meeting sites, and "allow infants in arms" during sessions (6).

The *CJASN* study's authors emphasize that programs should ultimately be advocating for their trainees (3). Specific recommendations include:

- Offering subsidized childcare for fellows
- Enacting clear policies for training programs
- Providing trainees with program-specific resources about pregnancy and parental leave policies, as well as national and local policies, early within their programs
- Mandating breastfeeding/lactation suites at every hospital
- Standardizing the duration for parental leave across the different governing bodies such as the American Board of Internal Medicine, ACGME, the American Board of Medical Specialties, and the Family and Medical Leave Act.

After overcoming societal, cultural, and socioeconomic factors that can influence pregnancy and its outcomes on health, a supportive workplace can enable fellows' success both professionally and personally. We have an opportunity to create a safe and supportive environment for nephrology trainees who are or may become pregnant and for future trainees who are considering nephrology fellowships. ■

Suman Behera, MD, MBBS, is an adjunct assistant clinical professor at McMaster University, Hamilton, Ontario, Canada.

The author reports no conflicts of interest.

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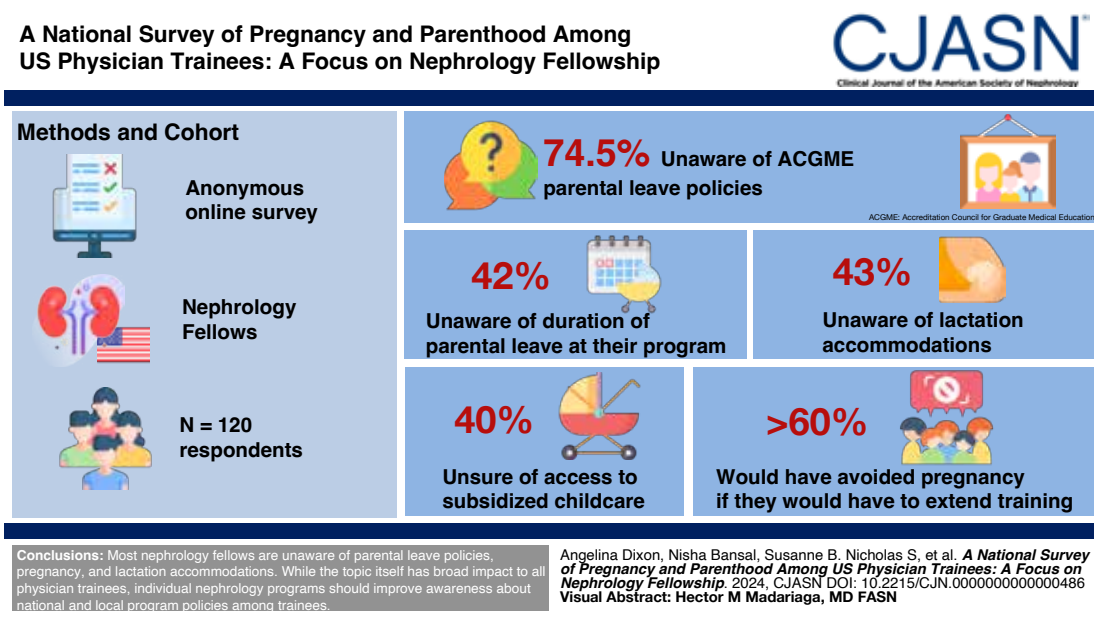

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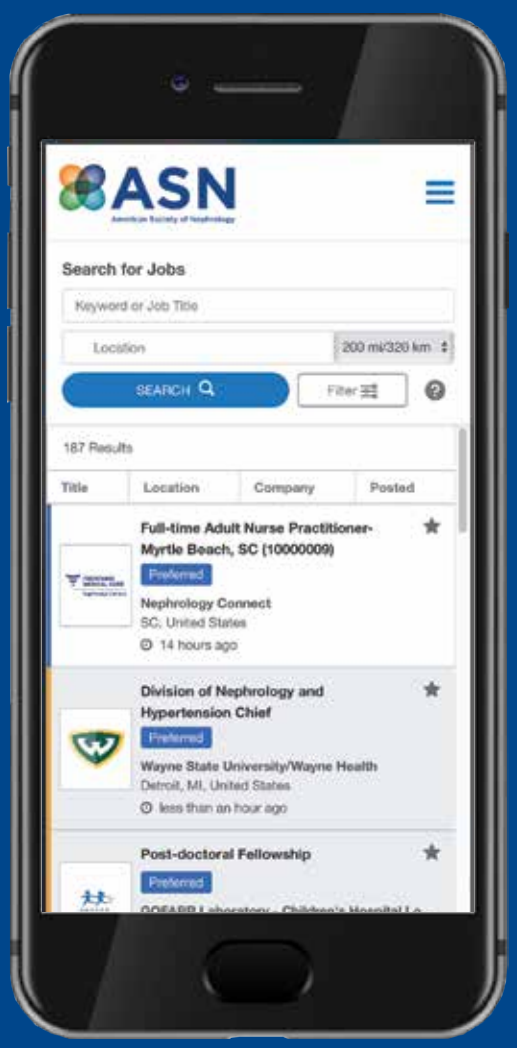

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ASN Expresses Concerns About Medicare Physician Fee Schedule, Support for Dental Services Coverage for ESRD Beneficiaries

By David L. White

ASN President Deidra C. Crews, MD, ScM, FASN, on behalf of the society provided comments and recommendations to the "CY2025 Payment Policies under the Physician Fee Schedule and Other Changes to Part B Payment and Coverage Policies" in September (1). ASN's recommendations touched on numerous provisions of the proposed rule (Table), but for this article, two are of particular interest:

1. Overall concerns regarding the Medicare Physician Fee Schedule (MPFS)
2. Support to expand coverage for dental services for individuals undergoing dialysis

Concerns about the plagued MPFS

The MPFS proposed rule included a 2.8% reduction to the MPFS conversion factor (CF). CF is a multiplier used to convert relative value units into dollars for medical services. Reductions to CF impact all clinicians providing care to patients on Medicare.

At the same time, clinicians continue to face ongoing financial challenges in operating their practices since the Medicare payment system has failed to keep pace with inflation. In 2025, the proposed 2.8% payment reduction will coincide with an expected 3.6% increase in medical practice cost inflation, as measured by the Medicare Economic Index. Nephrology will experience an overall 2.2% cut for 2025. When adjusted for inflation, Medicare physician payments have declined by 29% from 2001 to 2024 (2). This is clearly not a sustainable trajectory.

ASN has supported efforts by Congress since 2020 to mitigate and/or eliminate reductions caused by the application of Medicare's budget-neutrality adjustment, which statutorily prohibits any net increase in cost to the federal government when adjustments to MPFS exceed $20 million. Most recently, in the Consolidated Appropriations Act, 2024, Congress provided 2.93% of relief to help offset 2024's payment cut, once again mitigating but not eliminating the reduction and failing to keep up with medical inflation for 2024 (3). Unfortunately, the additional 2.93% expires at the end of 2024, which is the main contributor to the proposed 2.8% reduction for 2025.

MPFS is the only payment system within Medicare lacking an annual inflationary update, even though clinicians contend with a wide range of shifting economic factors (such as increasing administrative burdens, staff salaries, building rent, and purchasing essential technology) when determining their ability to provide care to patients on Medicare. The absence of an annual inflationary update, combined with statutory budget neutrality requirements, further compounds the difficulties that nephrologists face in managing resources to continue caring for patients in their communities.

ASN beseeched the Biden-Harris Administration to work directly with Congress and across the US Department of Health and Human Services to find solutions to the plagued payment system.

Coverage expansion for dental services for ESRD beneficiaries

In February 2024, ASN and the National Kidney Foundation jointly urged the Centers for Medicare & Medicaid Services (CMS) to use its existing authority or its broad waiver authority to allow Medicare payment for diagnostic and therapeutic dental services for Medicare End-Stage Renal Disease (ESRD) beneficiaries receiving dialysis when, because of immunosuppression, poorly controlled diabetes, heart disease, malnutrition, and/or other relevant comorbidities, dental treatment can be integral and substantially related to the clinical success of such covered nephrology-related medical services as:

- Current Procedural Terminology (CPT) codes 36901–36906: Dialysis circuit procedures
- CPT codes 90935, 90937, and 90940: Hemodialysis procedures
- CPT code 90961: Physician or other qualified health care professional visits for ESRD
- CPT codes 90989–90999: Other dialysis procedures
- CPT codes 99212–99215: Evaluation and management services
- Diagnosis-Related Group code 872: Hospitalization for septicemia or severe sepsis

Identifying and resolving dental infections can be similarly integral and related to the clinical success of other covered medical services for comorbidities that are frequently associated with kidney failure. Currently, many individuals covered under the ESRD benefit lack access to these essential services due to inadequate Medicare coverage.

Research has consistently demonstrated that oral health can be a crucial determinant of overall health outcomes in patients with kidney failure. Treatment of dental infections risking or causing bloodstream infections, poor glycemic control, and other complications can be integral and substantially related to the clinical success of medical therapies to manage ESRD.

Patients with kidney failure have higher rates of decayed, missing, and filled teeth; dental plaque; loss of attachment; xerostomia; gingivitis; periodontitis; as well as mouth and jaw-bone lesions compared with the general population. The consequences of poor oral health are worse for patients with kidney failure due to their advanced age, diabetes, polypharmacy, and/or impaired immune function. As a result, adults with kidney failure experience more severe oral diseases compared with the general population, which can contribute to increased mortality rates.

ASN wrote in its comments, "Given these findings, the expansion of dental services in Medicare should be considered a fundamental component of standard care for all [patients undergoing dialysis] just as it is for patients being evaluated for a kidney transplant. Providing this coverage is essential for improving health outcomes and ensuring comprehensive care for those affected by kidney failure" (1).

ASN thanked CMS for proposing to make necessary oral and dental care available for comorbidities frequently associated with kidney failure. In so doing, CMS will significantly reduce the risk of medical complications currently faced by individuals with kidney failure and avoid the costly interventions now borne by Medicare, beneficiaries, and taxpayers. ASN has long supported the expansion of Medicare coverage to include dental services.

To keep track of ASN's policy efforts related to these proposed rules, follow coverage in *Kidney News* and the ASN podcast feed, and visit http://www.asn-online.org/policy to read the full comment letter. For real-time updates from ASN Policy, follow @ASNAdvocacy on X. ■

David L. White is the regulatory and quality officer at ASN.

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Table. Topics ASN addressed in the comment letter

Expanding Caregiver Training Services to include home dialysis training services for caregivers
Expanding Hospital Inpatient or Observation Evaluation and Management Add-on for Infectious Disease (Healthcare Common Procedure Coding System code GIDXX) to include nephrologists
Expanding coverage for dental services for ESRD beneficiaries
Incorporating certain Advanced Primary Care Management services into the traditional Medicare program to support individuals with chronic kidney disease
Expanding coverage of compounded immunosuppressive drugs
Increasing uptake of Kidney Disease Education
Expanding audio-only coverage, additions to the telehealth list, and lifting of frequency limits
Incorporating new MIPS Quality Measures proposed for the CY2025 performance period/2027 MIPS payment year and future years

CY, calendar year; MIPS, Merit-based Incentive Payment System.

Symptom Burden Affects Quality of Life in Patients With Nondialysis CKD

In patients with nondialysis chronic kidney disease (CKD), high symptom burden is strongly associated with decreased health-related quality of life, reports a paper in the *American Journal of Kidney Diseases*.

The cross-sectional study included 4430 patients with stage 3 to 5 nondialysis CKD enrolled in the Chronic Kidney Disease Outcomes and Practice Patterns Study (CKDopps). Participants were drawn from Brazil, France, and the United States. Among the patients, 60% were men, the mean age was 68 years, and the mean baseline estimated glomerular filtration rate was 30 mL/min/1.73 m^2.

The Kidney Disease Quality of Life Short Form was used to assess the presence and severity of 13 individual symptoms and to classify overall symptom burden as low, intermediate, or high. Associations of symptom burden with the Kidney Disease Quality of Life Short Form physical and mental component scores were assessed.

Patients reported being "very much to extremely bothered" by a median of six symptoms. Muscle soreness and feeling "washed out or drained" were the most common bothersome symptoms, followed by cramps, shortness of breath, dry skin, diminished sex life, and numbness in hands or feet. With adjustment for other factors, symptoms showing the greatest increase in prevalence with CKD stage were lack of appetite, nausea or upset stomach, and feeling washed out or drained. Women were more likely to report high bothersome symptoms from all symptoms except impact on sex life, which was three times more frequent in men.

High symptom burden was more common among women, in France, and in patients with severe albuminuria and various comorbid conditions, although not in patients with a lower estimated glomerular filtration rate. High symptom burden compared with low symptom burden was associated with lower quality of life: a 13.4-point difference in the physical component scores and a 7.7-point difference in the mental component scores.

The CKDopps data show a high symptom burden among patients with nondialysis CKD, with associated reductions in disease-specific quality of life. The findings "indicate the importance and need to assess symptoms more systematically and to enhance provider [clinician]/patient communication as part of ND-CKD [nondialysis-CKD] care aiming to improve patients' quality of life and prioritized clinical outcomes," the investigators conclude [Speyer E, et al.; CKDopps Investigators. Symptom burden and its impact on quality of life in patients with moderate to severe CKD: The international Chronic Kidney Disease Outcomes and Practice Patterns Study (CKDopps). *Am J Kidney Dis*, published online August 6, 2024. doi: 10.1053/j.ajkd.2024.06.011].

Medical Management Versus Dialysis for Older Adults With Kidney Failure

Older adults who are frail with chronic kidney failure and who opt for continuing medical management rather than dialysis have only a slight reduction in survival time while spending significantly more days at home, reports a study in the *Annals of Internal Medicine*.

The study included data on 20,440 older adults (mean age, 77.9 years) with chronic kidney failure. All had an estimated glomerular filtration rate of <12 mL/min/1.73 m^2 and were not referred for kidney transplantation. Patients were treated in the US Department of Veterans Affairs health system between 2010 and 2018.

Using data from this observational cohort, the researchers performed a target trial emulation, mimicking the design principles of a randomized clinical trial, to compare the outcomes of initiating dialysis versus continuing medical management. Patient survival and number of days spent at home, rather than in a hospital or other care facility, were compared between groups.

The median time to dialysis was 8 days in patients who initiated dialysis versus 3 years in those continuing medical management. Survival was 770 days in the group starting dialysis versus 761 days in those continuing medical management—a nonsignificant difference of 9.3 days. On an "intention to treat" analysis, dialysis was associated with less time at home—a significant difference of 13.6 days—compared with continued medical management. In a "per-protocol" analysis, starting dialysis

was associated with increased survival, with a significant difference of 77.6 days, but 14.7 fewer days at home.

Older adults with kidney failure who are not referred for kidney transplantation face a difficult choice between starting dialysis or remaining on medical management.

Few studies have provided empirical data to guide this decision.

The clinical trial emulation suggests a modest increase in survival time for patients in this situation who start dialysis but with fewer days spent at home. The findings suggest "a more favorable risk-benefit profile of starting dialysis at an eGFR [estimated glomerular filtration rate] less than 12 mL/min/1.73 m^2 among subgroups aged 80 years or older and those with more advanced kidney failure," the researchers write. They discuss the study implications for clinical practice and policy [Montez-Rath ME, et al. Effect of starting dialysis versus continuing medical management on survival and home time in older adults with kidney failure: A target trial emulation study. *Ann Intern Med*, published online August 20, 2024. doi: 10.7326/M23-3028]. ■

Continued on page 68 >

 continued from page 67

Belzutifan Improves Response in Advanced Clear-Cell RCC

For patients with advanced clear-cell renal-cell carcinoma (RCC), the hypoxia-inducible factor 2α inhibitor belzutifan improves outcomes compared with everolimus, reports a randomized trial in *The New England Journal of Medicine*.

The phase 3, open-label LITESPARK-005 trial enrolled 746 patients with advanced clear-cell RCC at 147 study sites. All patients had received first-line treatment with immune checkpoint inhibitors and antiangiogenic drugs. Patients were randomly assigned to once-daily treatment with belzutifan (120 mg) or the mammalian target of rapamycin inhibitor, everolimus (10 mg). Progression-free and overall survival were evaluated as coprimary outcomes.

On interim analysis after a median of 18.4 months, progression-free survival was 24.0% for patients assigned to belzutifan versus 8.3% in the everolimus group. Belzutifan also produced a higher objective response rate: 21.9% versus 3.5%. At a second interim analysis, the median overall survival was 21.4 months with belzutifan and 18.1 months with everolimus. Eighteen-month survival was 55.2% and 50.6%, respectively; the difference was nonsignificant.

In both groups, more than 60% of patients experienced grade 3 or higher adverse events. Rates of treatment discontinuation due to adverse events were 5.9% with belzutifan and 14.7% with everolimus. Belzutifan was associated with a longer time to worsening of RCC-specific symptoms and to a decline in health-related quality of life.

In early-phase trials, belzutifan showed clinical activity against clear-cell RCC. The LITESPARK-005 results demonstrate improvements in progression-free survival and an objective response rate with belzutifan in patients who were heavily pretreated with advanced clear-cell RCC compared with everolimus. The authors discuss the clinical implications of their findings; studies comparing belzutifan combinations with other RCC therapies are ongoing [Choueiri TK, et al.; LITESPARK-005 Investigators. Belzutifan versus everolimus for advanced renal-cell carcinoma. *N Engl J Med* 2024; 391:710–721. doi: 10.1056/NEJMoa2313906].

Cannabis Cream May Reduce Itching in Patients on Dialysis

A topical cannabis cream reduces pruritus severity in patients with chronic kidney disease (CKD) receiving dialysis, according to a study in *Kidney Medicine*.

The randomized, double-blind trial included 60 patients on hemodialysis (mean age, 61.6 years) with CKD-associated pruritus. All scored 3 or higher on the Worst Itch Numeric Rating Scale, with a mean baseline score of 6.7.

Patients were assigned to receive a specially prepared cannabis cream incorporating 5% cannabis oil containing cannabidiol and tetrahydrocannabinol. Controls received a placebo cream that did not contain cannabis.

At 4 weeks, the mean Worst Itch Numeric Rating Scale score decreased to 2.6 in patients receiving cannabis cream and to 3.6 in the placebo group. After adjustment for baseline scores, use of the cannabis cream was associated with a –1.1 difference. Patients assigned to the cannabis cream also showed improvement in itch-related quality, based on reduction in the Skindex-10 score. However, the difference was nonsignificant after adjustment for baseline scores. No adverse effects were recorded in either group.

Pruritus is a bothersome symptom associated with CKD, for which current medications are often ineffective. By acting against inflammation, oxidation, and peripheral nerve activation, cannabinoids might be useful treatments for CKD-associated pruritus.

The randomized trial shows a promising reduction in severity with a cannabis-containing topical cream in patients on dialysis with CKD-associated pruritus. The researchers call for further studies with longer follow-up in larger patient samples, including assessment of itch-related quality of life [Anumas S, et al. Cannabis-containing cream for CKD-associated pruritus: A double-blind, placebo controlled trial. *Kidney Med*, published online August 16, 2024. https://www.kidneymedicinejournal.org/article/S2590-0595(24)00105-5/fulltext].

Sacubitril-Valsartan Shows Benefits for Patients on Dialysis With Heart Failure

Combination therapy with sacubitril and valsartan reduces mortality and the hospitalization rate in patients with heart failure and reduced ejection fraction (HFrEF) receiving dialysis for kidney failure, suggests a report in *JAMA Network Open*.

The retrospective study included propensity-matched groups of Medicare-enrolled patients with HFrEF and kidney failure who were on dialysis: 1434 receiving sacubitril-valsartan and 1434 receiving angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Approximately two-thirds of patients were male, one-third were Black, and more than half were White; the median time on dialysis was 3.8 years. Primary outcomes of all-cause mortality, cardiovascular mortality, all-cause hospitalization, and heart failure hospitalization were compared at a median follow-up of 0.9 years.

Overall mortality was 39% in patients receiving sacubitril-valsartan versus 43% in those with angiotensin-converting enzyme inhibitor or angiotensin receptor blocker therapy. On a propensity score-matched analysis, sacubitril-valsartan was associated with significant reductions in all-cause mortality (hazard ratio [HR], 0.82) and all-cause hospitalization (HR, 0.86). Cardiovascular mortality and the heart failure hospitalization rate were similar between groups. As-treated and subgroup analyses showed a comparable pattern. Sacubitril-valsartan was associated with a lower risk of hyperkalemia (HR, 0.71), with no significant difference in hypertension. Only 14% of patients received the maximum dose of sacubitril-valsartan at any time during the study.

Previous trials have reported reductions in mortality and hospitalization with sacubitril-valsartan for patients with HFrEF. The recent study extends these findings to suggest improved all-cause mortality and hospitalization rates with sacubitril-valsartan in patients with HFrEF on dialysis. The researchers conclude: "[S]acubitril-valsartan may have significant therapeutic potential among patients with HFrEF requiring hemodialysis, but further studies are needed before changes in clinical practice" [Le D, et al. Sacubitril-valsartan in patients requiring hemodialysis. *JAMA Netw Open* 2024; 7:e2429237. doi: 10.1001/jamanetworkopen.2024.29237].

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Thursday, October 24

General & Clinical Nephrology Poster Tour — **10:30 — 11:30 a.m.**
Led by Edgar Lerma, MD, FASN, and Roger Rodby, MD, FASN.

Overview of the Increasing Organ Transplant Access (IOTA) Model — **11:30 a.m. — 12:30 p.m.**
A synopsis and discussion of the new payment model with CMMI: what it means for your practice and role. Relevant for both general and transplant nephrologists. (Session 1)

Kidney Care Choices (KCC) Model Meeting — **12:30 — 1:30 p.m.**
An intimate discussion with CMMI about the KCC model, its future, and potential scenarios for your practice. (Session 1)

Phairify Demonstration — **1:30 — 2:00 p.m.**
Learn about the Physician Value Exchange and how to leverage this exclusive ASN member benefit. (Session 1)

Friday, October 25

Overview of the Increasing Organ Transplant Access (IOTA) Model — **10:00 — 11:00 a.m.**
A synopsis and discussion of the new payment model with CMMI: what it means for your practice and role. Relevant for both general and transplant nephrologists. (Session 2)

New Features of Communities — **11:00 a.m. — 12:30 p.m.**
Overview of the new platform: intuitive widgets, personalized links, navigation tips, and more.

Phairify Demonstration — **12:30 — 1:00 p.m.**
Learn about the Physician Value Exchange and how to leverage this exclusive ASN member benefit. (Session 2)

Forum on Transplant Nephrology Training — **1:00 — 2:00 p.m.**
ASN-AST effort to secure ACGME accreditation for transplant nephrology: briefing, questions and answers, and networking.
Led by Roy Bloom, MD, FASN, and Neeraj Singh, MD, FASN.

Kidney Care Choices (KCC) Model Meeting — **2:00 — 3:00 p.m.**
An intimate discussion with CMMI about the KCC model, its future, and potential scenarios for your practice. (Session 2)

Saturday, October 26

Becoming a Community Leader — **10:00 — 11:00 a.m.**
Engagement strategies, moderation, replies, and follow-through.

Communities Library — **11:00 a.m. — 12:00 p.m.**
Keyword searching, studies, reports, and credible research.

Themed Communities — **12:00 — 1:00 p.m.**
Onco-nephrology, Kidney Transplantation, Women's Health and Research.

Mentor Match Program — **1:00 — 2:00 p.m.**
Enrollment basics, search criteria, and building relationships.

Listen and partake in conversations.

ASN Communities

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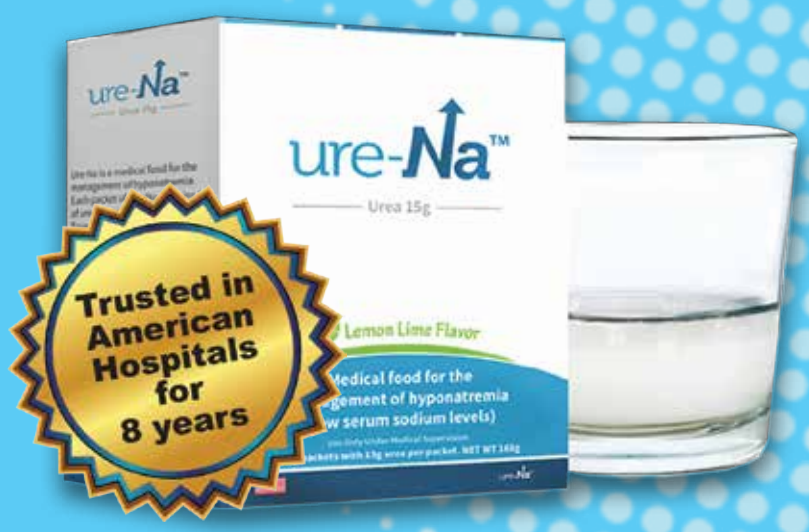

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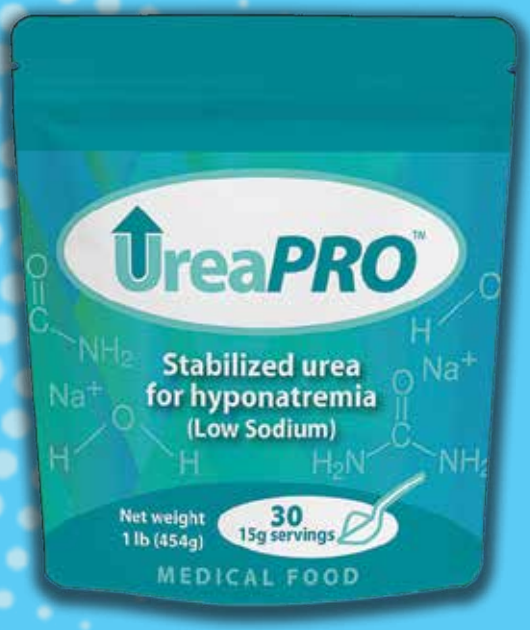

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*2014 European Clinical Practice Guideline On The Diagnosis & Treatment Of Hyponatremia, UpToDate: Overview Of The Treatment of Hyponatremia in Adults, AAP Pediatrics 2021 Vasopressin Dependent Disorders
**Current EAU Guideline on Urolithiasis
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