Navigating the Crossroads: CKD Management & Dialysis Decisions in PA-LTC

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Disclosures

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Clinical Context & Session Objectives

- **The PA-LTC Challenge:** High burden of CKD stages 3–5 combined with advanced multimorbidity and frailty.
- **Cellular Aging & Inflammaging:** Connecting sterile chronic inflammation to progressive renal senescence.
- **Cardio-Renal Protection:** Reviewing the 4 pillars of organ preservation and landmark clinical trial data.
- **The Gut-Kidney Axis:** Understanding uremic toxin generation and therapeutic modulation.
- **Dialysis & Goals of Care:** Balancing longevity against functional quality of life and patient values.

Core Philosophy

Renal health in long-term care cannot be isolated from systemic aging pathways. Successful management requires combining organ protection with holistic comfort and shared decision-making.

Reference: Franceschi C, et al. Inflammaging: a new immune-metabolic viewpoint for age-related diseases. Nat Rev Endocrinol. 2018;14(10):576-590.

Epidemiology of CKD in Post-Acute & Long-Term Care

High Prevalence & Multimorbidity

- CKD stages 3–5 affect over 40% of residents aged 65+ in skilled nursing facilities.
- Frequently coupled with ischemic heart disease, type 2 diabetes, and heart failure.
- Age-related nephron loss compounded by cumulative microvascular ischemic injury.

Competing Mortality Risks

- Traditional eGFR risk models overestimate end-stage renal failure risk relative to competing mortality.
- Focus must shift from delaying dialysis at all costs to preventing AKI and managing uremic symptoms.

Reference: O'Hare AM, et al. Mortality risk among slow versus rapid decliners in kidney function in older adults. J Am Soc Nephrol. 2021;32(7):1711-1720.

The "Silver Tsunami" and CKD

- **Epidemiology:** 38% of adults >70 have CKD.
- **The Nexus:** Diabetes is the leading cause, but aging is the leading *accelerant*.
- **Risk:** Mortality in elderly CKD is higher from Cardiovascular Disease (CVD) than progression to dialysis.

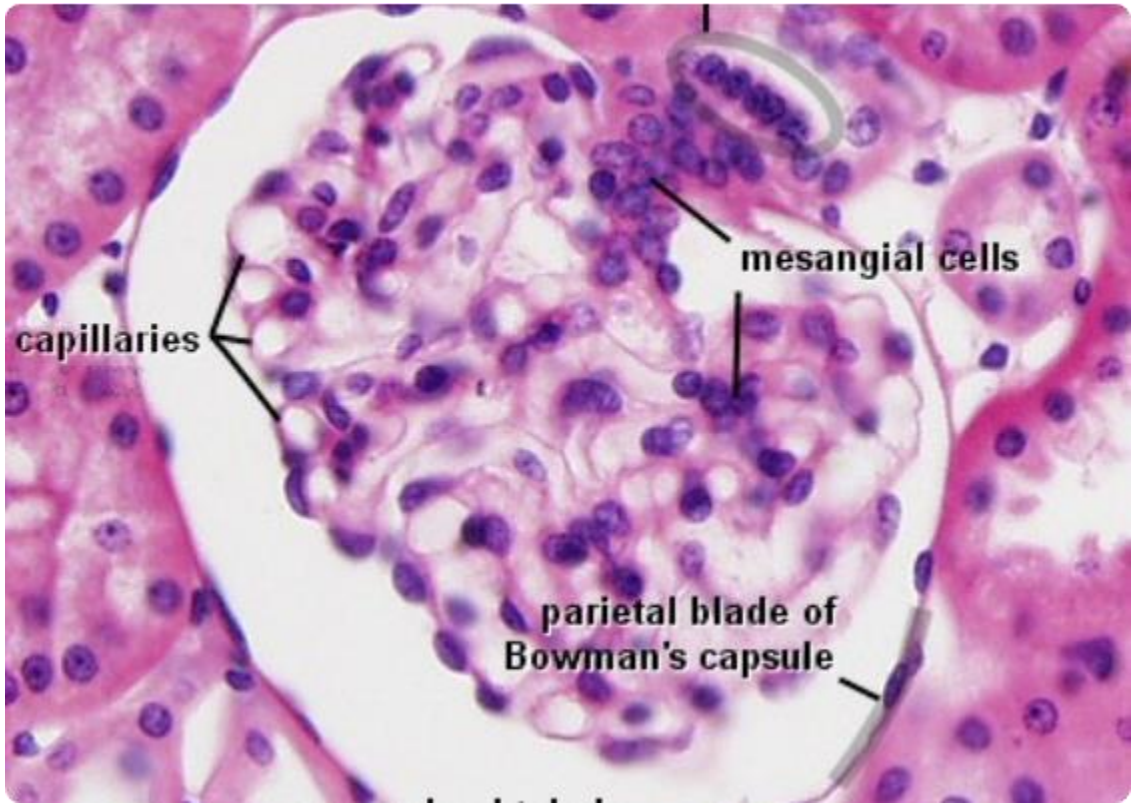
IDF Diabetes Atlas Report: Diabetes and Kidney Disease. 2024

Defining the Aging Kidney

- **Structural decline:** 10% loss of nephrons per decade after age 40.
- **Functional decline:** Decreased renal blood flow and impaired autoregulation.
- **Clinical Pearl:** "Biological age" of the kidney often exceeds "Chronological age" in diabetics.

Chronic Kidney Disease and Risk Management: Standards of Care in Diabetes—2026. Diabetes Care. 2026;49(Suppl 1):S246-S260.

Biology of Aging: Structural Renal Senescence



Microvascular and Glomerular Alterations

- **Global Glomerulosclerosis:** Progressive loss of functional nephrons and interstitial fibrosis with advancing age.
- **Vascular Stiffness:** Hyaline arteriolosclerosis reducing renal blood flow reserve and autoregulation.
- **Impaired Repair:** Decreased telomere length and reduced cellular autophagy limit recovery from minor ischemic insults.

Reference: Denic A, et al. The structural basis of a progressive decline in renal function with age. J Am Soc Nephrol. 2017;28(8):2293-2301.

Pathophysiology of Diabetic Kidney Disease (DKD)

- **Hyperglycemia:** Leads to Advanced Glycation End-products (AGEs).
- **Hemodynamics:** Afferent dilation + Efferent constriction = Glomerular Hyperfiltration.
- **Result:** Podocyte death and basement membrane thickening.

Diabetic Kidney Disease: From Pathophysiology to Regression of Albuminuria. Int. J. Mol. Sci. 2026;27(1):8224.

Diagnostic Nuances: Interpreting eGFR in Frail Adults

Creatinine Pitfalls

- Serum creatinine-based eGFR equations **overestimate** true GFR in frail residents with sarcopenia and low muscle mass.
- A deceptively normal serum creatinine can mask advanced renal impairment.

Cystatin C Utility

- Cystatin C is independent of muscle mass and provides a more accurate estimate of renal function in immobilized residents.
- Serial clinical trends outweigh single lab snapshots.

Reference: Inker LA, et al. New creatinine- and cystatin C-based equations to estimate GFR without race. N Engl J Med. 2021;385(19):1737-1749.

CKD as a Systemic Inflammatory State

- **Concept:** CKD is not just a plumbing issue; it is a "Cytokine Storm" in slow motion.
- **Markers:** Elevated CRP, IL-6, and TNF-.
- **Impact:** Inflammation drives anemia, muscle wasting, and depression.

Defining "Inflammaging": The Chronic Immune Shift

What is Inflammaging?

A chronic, sterile, low-grade inflammation characteristic of advanced aging, driven by senescent cell accumulation (SASP), mitochondrial dysfunction, and persistent antigenic load.

Key Biochemical Markers

Chronically elevated baseline levels of IL-6, TNF-alpha, C-reactive protein (CRP), and high-mobility group box 1 (HMGB1).

Systemic Consequences

Accelerates vascular calcification, cognitive decline, muscle wasting (sarcopenia), and progressive renal tubulointerstitial fibrosis.

Reference: Franceschi C, Campisi J. Chronic inflammation (inflammaging) and its potential contribution to age-associated diseases. J Gerontol A Biol Sci Med Sci. 2014;69(Suppl 1):S4-S9.

Inflammaging as a Driver of Accelerated CKD

- **Endothelial Dysfunction:** Pro-inflammatory cytokines promote vascular permeability, arterial stiffening, and capillary rarefaction.
- **Oxidative Stress:** Mitochondrial dysfunction in tubular cells generates excessive reactive oxygen species (ROS), driving apoptosis.
- **Fibrogenic Signaling:** Macrophage polarization toward pro-inflammatory phenotypes stimulates TGF-beta pathways, accelerating interstitial fibrosis.
- **Uremic Amplification:** Reduced renal clearance traps inflammatory mediators, creating a self-amplifying feedback loop.

The Uremic-Inflammatory Loop

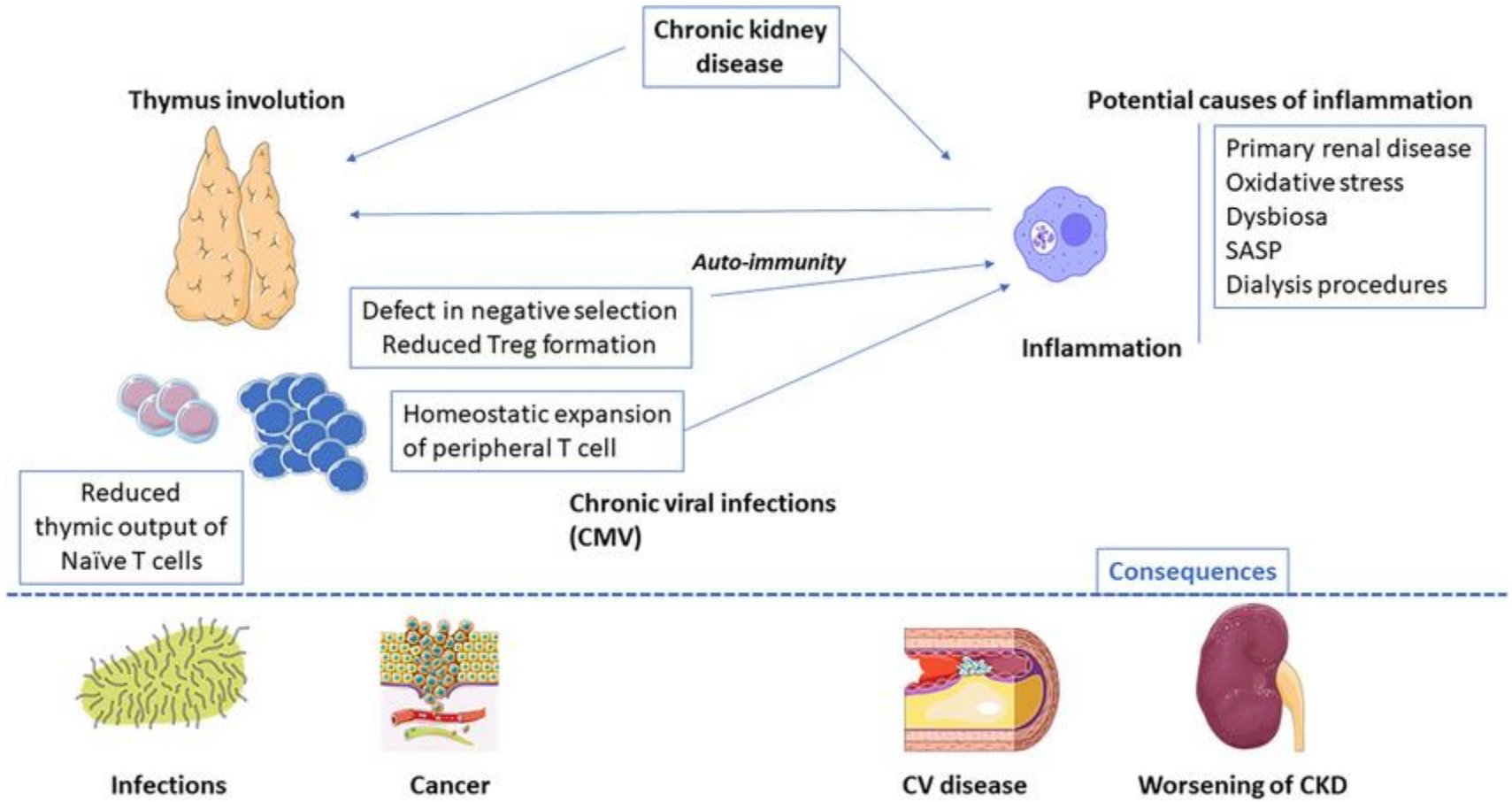
Decreased kidney function impairs clearance of inflammatory cytokines, which in turn damages remaining nephrons—cementing the link between aging biology and renal failure.

Reference: Stenvinkel P, et al. The Toxin-Inflammation-Cachexia Axis in Chronic Kidney Disease. J Intern Med. 2019;285(6):639-652.

"Inflammaging" Explained

- **Definition:** The intersection of age-related immune dysfunction and chronic disease.

- **NLRP3 Inflammasome:** A protein complex triggered by metabolic stress that causes cell death (pyroptosis) and scarring.

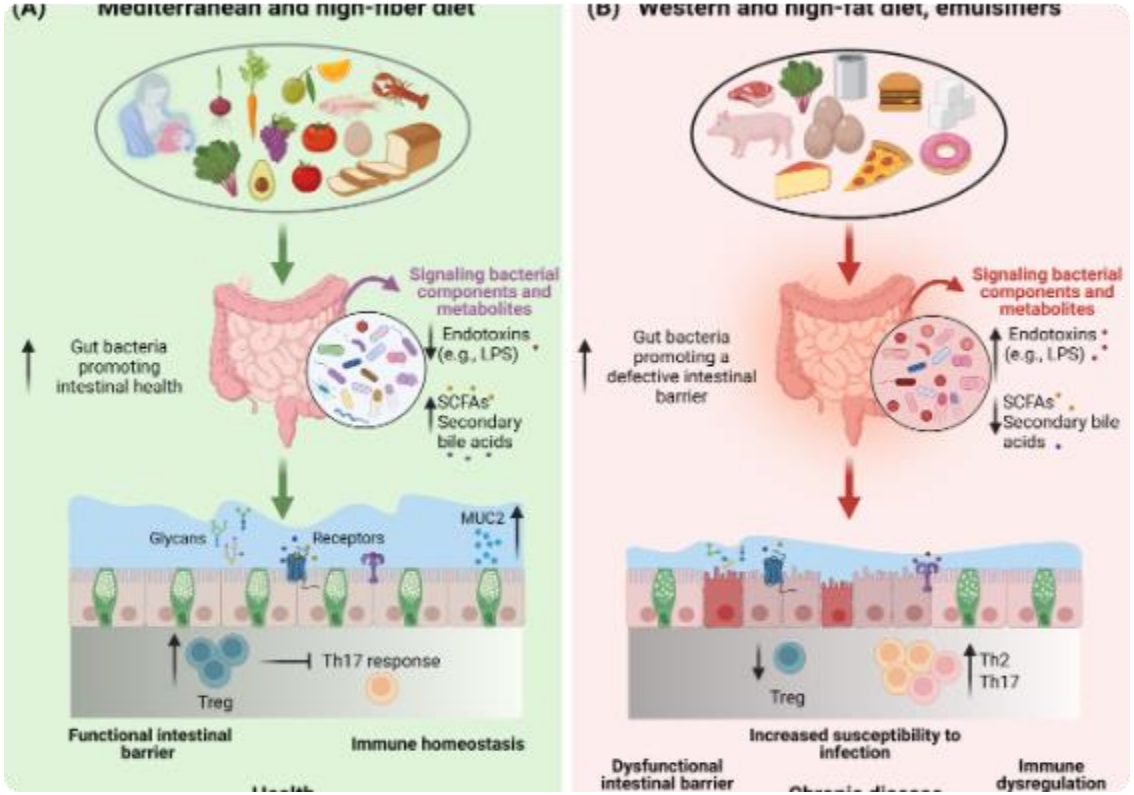


End-Stage Renal Disease-Related Accelerated Immune Senescence, 2021

The Gut-Kidney Axis

- **Dysbiosis:** CKD alters gut flora.
- **Leaky Gut:** Uremic toxins break down the intestinal barrier.
- **Endotoxemia:** Gut bacteria "leak" into the blood, fueling systemic inflammation.

The Gut-Kidney Axis: Uremic Toxin Generation



Dysbiosis & Protein-Bound Toxins

- **Dysbiosis & Epithelial Breakdown:** Advanced CKD induces intestinal dysbiosis and tight-junction breakdown, causing endotoxin translocation.
- **Colonic Precursors:** Fermentation of unabsorbed amino acids yields precursors converted in the liver to **Indoxyl Sulfate (IS)** and **p-Cresyl Sulfate (pCS)**.
- **Dialysis Resistance:** Protein-bound toxins resist standard clearance and directly drive vascular damage and renal fibrosis.

Reference: Ramezani A, Raj DS. The gut microbiome, kidney disease, and targeted interventions. J Am Soc Nephrol. 2014;25(4):657-670.

Targeting the Gut-Kidney Axis in PA-LTC

- **Mitigating Systemic Inflammation:** Reducing gut-derived uremic toxins dampens systemic oxidative stress and vascular endothelial injury.
- **Nutritional & Prebiotic Modulations:**
  - High-fiber diets (where tolerated) promote beneficial short-chain fatty acid (SCFA) producing bacteria.
  - Oral sorbents (e.g., AST-120) bind precursor molecules in the gut lumen, reducing systemic absorption.
- **Balancing Restrictions:** Careful balance between avoiding excessive toxin load and preventing restrictive malnutrition in frail seniors.

Clinical Insight

Targeting the gut microbiome offers a novel, non-dialytic avenue to reduce systemic inflammation and slow tubular injury in vulnerable long-term care residents.

Reference: Vaziri ND, et al. Gut microbiota-metabolite axis in chronic kidney disease. Nephrol Dial Transplant. 2016;31(1):1-7.

Vascular Stiffening & Calcification

- **Process:** Inflammation transforms vascular smooth muscle into osteoblast-like cells.
- **Outcome:** "Stiff pipes" lead to Left Ventricular Hypertrophy (LVH) and high stroke risk.
- **Medial Calcification:** A hallmark of the aging diabetic kidney.

Overview of the 4 Pillars of Cardio-Renal Protection

Pillar I

RAAS Blockade (ACEi / ARB): Reducing intraglomerular pressure and halting progressive albuminuria.
The Foundation

Pillar II

SGLT2 Inhibitors: Profound cardio-renal protection via tubular hemodynamic modulation and anti-inflammatory pathways.
Reduces Pressure and inflammation

Pillar III

Non-steroidal MRA (Finerenone): Combating mineralocorticoid receptor overactivation, inflammation, and fibrosis.
Specifically blocks fibrosis

Pillar IV

GLP-1 Receptor Agonists: Glycemic control paired with robust vascular, weight, and renal outcomes.
Potent anti-inflammatory and weight benefits

Reference: Heerspink HJL, et al. Rationale and protocol for the EMPA-KIDNEY trial. Nephrol Dial Transplant. 2018;33(1):169-177.

Pillar I: Renin-Angiotensin-Aldosterone System (RAAS) Blockade

- **Mechanism of Action:** ACE inhibitors and ARBs dilate the efferent renal arteriole, lowering glomerular capillary hydraulic pressure.
- **Clinical Benefits:** Slows GFR decline and substantially reduces albuminuria in proteinuric CKD.
- **PA-LTC Considerations:**
  - Monitor serum creatinine and potassium closely upon initiation or dose titration.
  - Accept an initial serum creatinine rise up to 30% as a hemodynamic adjustment rather than true acute injury.

Landmark Trial Data

The RENAAL and IDNT trials established ARBs as cornerstone therapy for slowing nephropathy progression in type 2 diabetic kidney disease.

Reference: Brenner BM, et al. Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. N Engl J Med. 2001;345(12):861-869.

Pillar II: SGLT2 Inhibitors – Cardio-Renal Game Changers

Hemodynamic & Metabolic Effects

- Restores tubuloglomerular feedback by increasing distal sodium delivery, causing afferent arteriolar constriction and reducing intraglomerular pressure.
- Lowers intraglomerular hypertension independently of blood glucose reduction.

Landmark Trial Evidence

- **DAPA-CKD & EMPA-KIDNEY:** Demonstrated ~30% reduction in CKD progression, cardiovascular death, or end-stage renal disease across diabetic and non-diabetic cohorts.
- Substantial reduction in heart failure hospitalizations.

The "Dip": Anticipate a 2-5 mL/min drop in GFR initially—this is a sign of success, not injury

Anti-inflammatory: Lowers oxidative stress in the proximal tubule

Reference: Heerspink HJL, et al. Dapagliflozin in Patients with Chronic Kidney Disease. N Engl J Med. 2020;383(15):1436-1446.

Pillar III: Non-Steroidal Mineralocorticoid Receptor Antagonists

- **Mechanism:** Selective non-steroidal MRA (Finerenone) with potent anti-inflammatory and anti-fibrotic properties in both heart and kidney tissue.
- **FIDELIO-DKD & FIGARO-DKD Trials:** Showed significant reductions in kidney failure endpoints and cardiovascular events in CKD associated with type 2 diabetes.
- **Safety Profile:** Lower risk of hyperkalemia compared to steroidal MRAs (spironolactone), though baseline potassium and eGFR monitoring remain essential in PA-LTC.

Cardio-Renal Synergy

Targeting mineralocorticoid receptor overactivation reduces macrophage infiltration and interstitial fibrosis in aging kidneys.

Reference: Bakris GL, et al. Effect of Finerenone on Chronic Kidney Disease Outcomes in Type 2 Diabetes. N Engl J Med. 2020;383(23):2219-2229.

Pillar IV: GLP-1 Receptor Agonists

Vascular & Renal Benefits

- Reduces major adverse cardiovascular events (MACE) and slows albuminuria progression.
- Promotes natriuresis, weight reduction, and endothelial anti-inflammatory signaling.

FLOW Trial Findings

- The landmark FLOW trial demonstrated definitive renal outcome benefits with semaglutide in patients with type 2 diabetes and CKD.
- Particularly useful in managing concurrent obesity and metabolic syndrome in early-stage PA-LTC residents.

Reference: Perkovic V, et al. Effects of Semaglutide on Chronic Kidney Disease in Type 2 Diabetes. N Engl J Med. 2024;391(2):109-121.

GLP-1 RA & The FLOW Trial

- **New Evidence:** Semaglutide significantly reduces major renal events.
- **Mechanism:** Beyond glucose; it reduces systemic inflammation and slows GFR decline.
- **Elderly Use:** Excellent for those needing weight management and CVD protection.

Perkovic V, et al. *Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes (FLOW Trial)*. N Engl J Med. 2024;391:607-621

De-prescribing and Tailoring Therapy in PA-LTC

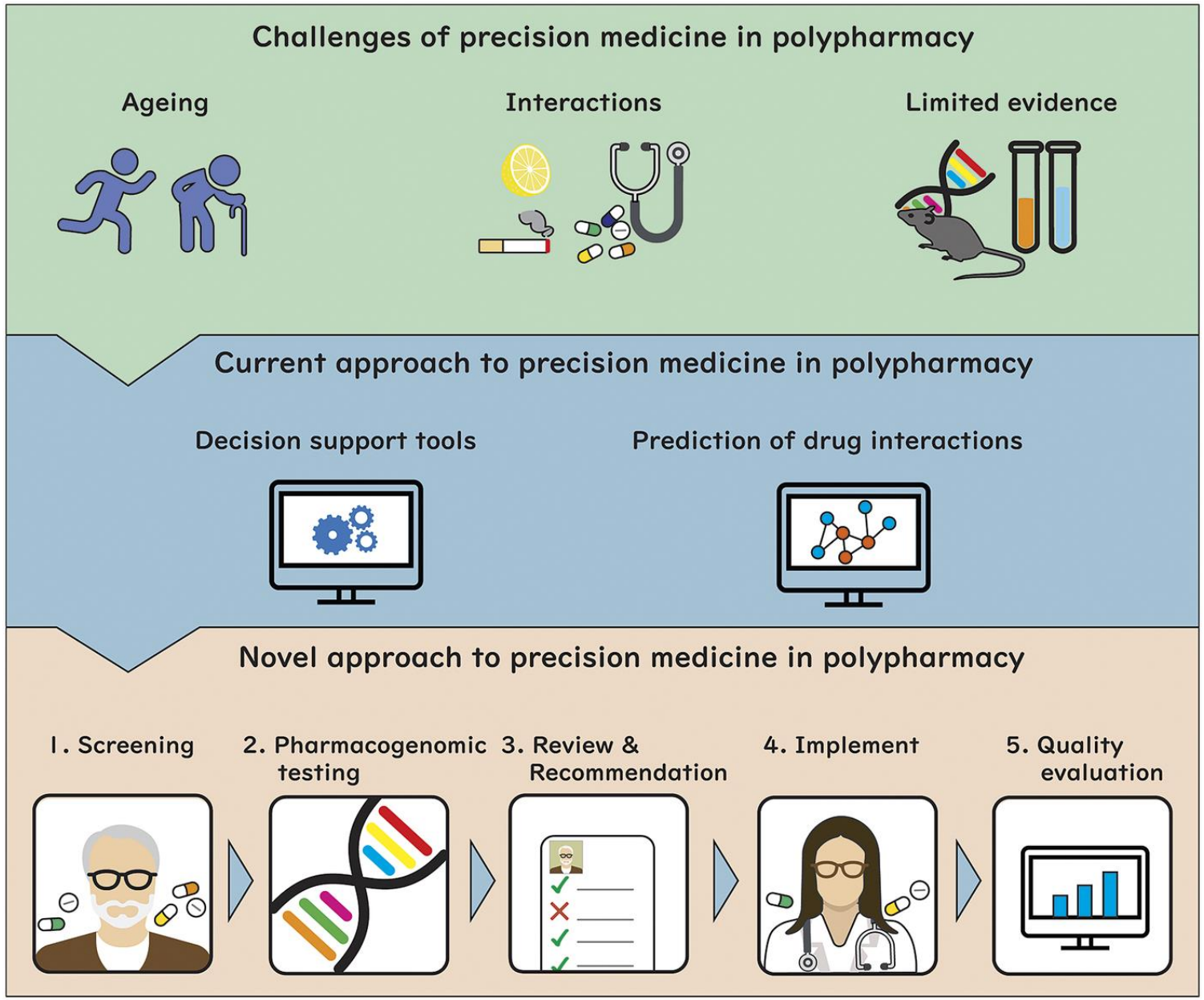
- **The Polypharmacy Burden:** Frail older adults with CKD are uniquely vulnerable to adverse drug reactions due to reduced clearance. Average 8-12 meds
- **Candidates for De-escalation:**
  - Statins prescribed solely for primary prevention in advanced life-limiting illness.
  - Overly aggressive anti-hypertensives causing orthostasis and falls.
  - Supplements and vitamins with minimal net clinical benefit.
- PPI Associated with interstitial nephritis
  NSAIDS Avoid as much as possible

Deprescribing Principle

Every medication added in advanced CKD must pass a rigorous risk-benefit audit regarding its impact on daily comfort and functional capacity.

Preserving Quality of Life: Retain medications that directly control symptoms (analgesics, antiemetics, loop diuretics).

Reference: Muth C, et al. The Quest to Deprescribe in Frail Older Adults with Multimorbidity. Arch Intern Med. 2018;178(5):639-646.


Challenges of precision medicine in polypharmacy
Ageing
Interactions
Limited evidence
Current approach to precision medicine in polypharmacy
Decision support tools
Prediction of drug interactions
Novel approach to precision medicine in polypharmacy
1. Screening
2. Pharmacogenomic testing
3. Review & Recommendation
4. Implement
5. Quality evaluation

Overview of Sick-Day Protocols in PA-LTC

- **The Vulnerability Window:** Intercurrent acute illnesses (gastroenteritis, sepsis, pneumonia) put PA-LTC residents at extreme risk for rapid-onset acute kidney injury (AKI).
- **The "SADMANS" Rule:** Medications that require temporary withholding during acute dehydration risks:
  - **S**ulfonylureas
  - **A**CE inhibitors / **A**RBs
  - **D**iuretics
  - **M**etformin
  - **A**ngiotensin Receptor-Neprilysin Inhibitors (ARNI)
  - **N**SAIDs
  - **S**GLT2 inhibitors

Clinical Objective

Prevent prerenal azotemia and tubular necrosis by temporarily withholding nephrotoxic or volume-depleting agents during acute dehydrating illness.

Reference: Thomas MC, et al. Sick day protocols for kidney protection: rationale and implementation. Diabetes Care. 2019;42(5):791-799.

Clinical Data & Evidence Supporting Sick-Day Management

Risk of Euglycemic DKA & Lactic Acidosis

- SGLT2 inhibitors during acute volume contraction significantly increase risk of euglycemic diabetic ketoacidosis.
- Metformin combined with acute renal hypoperfusion markedly elevates lactic acidosis risk.

Trial & Registry Insights

- Real-world registry data shows that failure to hold RAS inhibitors and diuretics during acute gastroenteritis accounts for >40% of preventable hospital admissions for AKI in older adults.

Reference: Lupsa BC, Inzucchi SE. Use of SGLT2 inhibitors in older adults: safety and clinical management. Diabetes Obes Metab. 2018;20(11):2542-2555.

Clinical Resources for Sick-Day Protocols

Canadian SGLT2 / RAS Guidance

Widely adopted clinical pocket cards and facility algorithms detailing exact hold and restart criteria during acute illness.

Kidney Health Australia Toolkit

Comprehensive patient and nursing staff resources outlining clear "Stop" and "Go" medication checklists during intercurrent infections.

Facility Standing Orders

Implementing automatic nursing notification protocols to hold diuretics and nephrotoxins when oral intake drops below 50% for 24 hours.

Reference: Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. Clinical Practice Guideline. Kidney Int Suppl. 2023;13(1):1-126.

Case Study – The Balancing Act

Patient Profile: "Mrs. J"

- **Age:** 82-year-old female.
- **History:** Type 2 Diabetes (25 years), Hypertension, Osteoarthritis, and Frailty (uses a walker).
- **Labs:**
  - **Serum Creatinine:** 106 mmol/L (1.2 mg/dL) — *Looks "normal" to the untrained eye.*
  - **eGFR (Creatinine):** 44 mL/min/1.73m
  - **UACR (Albuminuria):** 250 mg/g (Moderately increased).
  - **HbA1c:** 7.8%.
- **Current Meds:** Metformin, Lisinopril, Amlodipine, Naproxen (for knee pain).

The Clinical Dilemma:

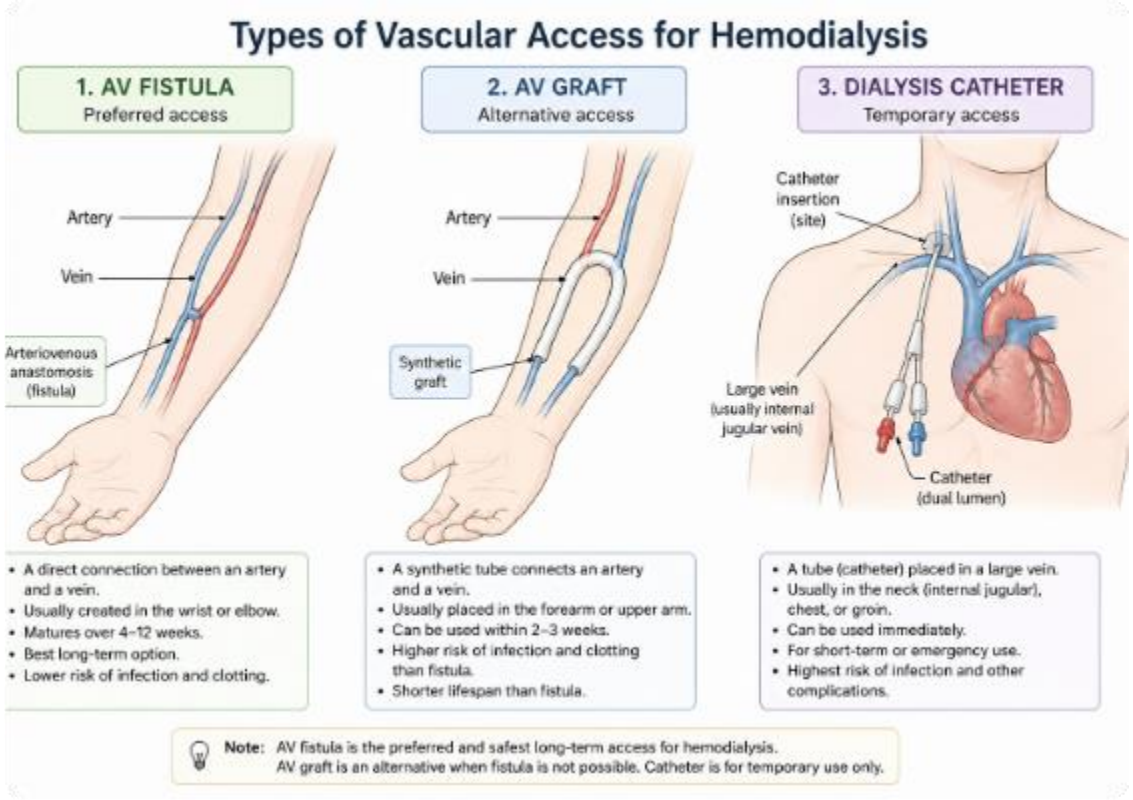
1. **The Hidden Risk:** Her "normal" creatinine masks Stage 3b CKD because of her low muscle mass.
2. **The Inflammatory Driver:** Chronic NSAID use (Naproxen) is likely accelerating her renal decline and worsening her BP.
3. **The Opportunity:** She is not on an SGLT2 inhibitor or a non-steroidal MRA (Finerenone).

Management Plan (The Discussion Points):

- **Deprescribing:** Transition from Naproxen (NSAID) to topical treatments or acetaminophen to remove the nephrotoxic hit.
- **Optimization:** Introduce a low-dose SGLT2i. Discuss the "initial dip" in GFR with the patient so she doesn't panic.
- **Safety Education:** Provide a "Sick Day" card. If she gets a stomach flu, she must stop the Lisinopril and SGLT2i immediately.
- **Target Shift:** Instead of aiming for an HbA1c <7.0% (which risks dangerous hypoglycemia in the elderly), aim for <8.0% to prioritize safety over "perfect" numbers.

The Dialysis Dilemma: Survival vs. Quality of Life

- **High Post-Initiation Mortality:** Over 50% mortality within the first year of starting chronic hemodialysis among nursing home residents with severe baseline frailty.
- **Functional Freefall:** Transitioning to dialysis frequently accelerates loss of independent ambulation and cognitive engagement.
- **The "Dialysis Hangover":** Post-treatment fatigue consumes 12 to 24 hours post-session, robbing the resident of active waking hours.
- **Hospitalization Burden:** Vascular access failures and fluid overloads drive frequent ED visits and readmissions.



Reference: Kurella Tamura M, et al. Functional status of elderly adults before and after initiation of dialysis. N Engl J Med. 2009;361(16):1539-1547.

Comparative Survival: Dialysis vs. Conservative Management

Patient Cohort / Clinical Profile	Median Survival on Dialysis	Median Survival on Conservative Care	Clinical Interpretation
Age 80+, Frailty Score > 6, Advanced Dementia	6 – 12 Months	6 – 10 Months	Minimal or no survival gain with chronic dialysis; increased treatment burden.
Age 75+, Multiple Comorbidities (CHF, Diabetes)	12 – 24 Months	10 – 18 Months	Modest survival extension offset by heavy hospital and transport burden.
Younger, Robust PA-LTC Resident with Good Mobility	36+ Months	12 – 18 Months	Potential survival advantage; candidacy depends heavily on personal values.

Reference: Carson RC, et al. Association between renal replacement therapy and mortality in older patients with stage 5 CKD. Nephrol Dial Transplant. 2009;24(9):2900-2907.

Conservative Kidney Management (CKM) as an Active Strategy

Active Treatment

CKM is not "doing nothing." It is an intensive medical, nutritional, and palliative strategy aimed at maximizing comfort and preserving remaining renal function without dialysis.

Symptom Mastery

Aggressive management of uremic symptoms (pruritus, nausea, restless legs, pain) and meticulous fluid volume balance via loop diuretics.

Dietary Freedom

Relaxation of severe, unpalatable renal restrictions (potassium/phosphorus limits) to encourage eating and maintain nutritional status and pleasure of food.

Reference: Murtagh FE, et al. Conservative management of end-stage renal disease without dialysis: a systematic review. BMC Nephrol. 2007;8:25.

Palliative Management of Uremic Symptoms

Volume & Acidosis Control

Diuretics: Titrate loop diuretics combined with thiazides for refractory edema and mild uremic volume expansion.

Bicarbonate: Oral sodium bicarbonate supplementation to correct metabolic acidosis, improve energy, and preserve muscle mass.

Pruritus & GI Distress

Itch Relief: Emollients, gabapentin at low doses, and targeted therapies for uremic pruritus.

Nausea & Fatigue: Short courses of antiemetics (ondansetron, metoclopramide) and addressing secondary anemia when appropriate for comfort.

Reference: Davison SN. Palliative care for patients with advanced chronic kidney disease not suited for dialysis. Clin J Am Soc Nephrol. 2006;1(3):531-538.

Proactive Timing of Goals of Care (GOC) Discussions

Why Proactive Timing Matters

- **Avoid Crisis Decisions:** Discussions initiated during acute uremic encephalopathy or pulmonary edema lead to reactive, fear-driven choices.
- **Admission Integration:** Incorporate renal prognosis assessments upon SNF/LTC admission or when eGFR drops persistently below 20 mL/min/1.73m².
- **Long-Term Alignment:** Allows families and residents time to process trajectories without the pressure of an imminent emergency room transfer.

Reference: Laky B, et al. Advance care planning in advanced kidney disease: A systematic review. Palliat Med. 2021;35(4):689-703.

Eliciting Personal Values over Clinical Projections

Shifting the Question

Instead of asking abstract medical questions like *"Do you want to live longer?"*, frame inquiries around what gives life meaning and what burdens are unacceptable.

"What does a good day look like?"

Uncovering daily joys: visiting with family, enjoying favorite foods, sitting outside, or remaining pain-free.

"What are you hoping to avoid?"

Identifying feared outcomes: frequent hospital admissions, mechanical ventilation, or being tethered to a dialysis chair.

"The Time Trade-off"

Reflecting on spending 12 hours weekly traveling to and undergoing treatments versus staying in the familiar LTC environment.

Reference: Schell JO, et al. Conversations about dialysis: a systematic review of qualitative studies. Clin J Am Soc Nephrol. 2012;7(7):1100-1109.

Practical Communication: The "Ask-Tell-Ask" Approach

1. ASK (Assess Understanding)
Explore what the resident and family currently understand about their kidney function and overall trajectory.

Example: "What is your understanding of how your kidneys are holding up right now?"

2. TELL (Share Compassionately)
Deliver clear, concise prognostic information framed within the context of daily energy and functional goals.

Example: "Given your overall strength, starting dialysis would likely take more time than it gives back."

3. ASK (Check Response & Align)
Invite feedback and ensure emotional alignment before moving to actionable orders. *"How does that sit with you and your family?"*

Reference: Back AL, et al. Approaching difficult communication tasks in oncology. CA Cancer J Clin. 2005;55(3):164-181.

Navigating Surrogates & Overcoming Guilt

- **The "Abandonment" Myth:** Families often equate choosing conservative care with "giving up" or withholding essential medical support.
- **Clarifying Active Care:** Reassure surrogates that conservative management is a robust, attentive, and comforting treatment plan focused on symptom relief.
- **Substituted Judgment:** Guide families away from what *they* would choose for themselves and toward what the resident explicitly stated or would have wanted.
- **Mitigating Burnout:** Validate family stress and offer multidisciplinary support via social work and chaplaincy.

Reference: Davison SN, et al. The role of surrogate decision-makers in end-stage renal disease treatment choices. Am J Kidney Dis. 2017;69(2):245-253.

Translating Discussions into Actionable Orders

POLST / MOLST Integration

Ensure that choices regarding dialysis, hospital transfers, and resuscitation are explicitly captured on portable medical orders rather than vague blanket statements.

Specific Parameter Orders

Define clear triggers for comfort measures, oxygen, and sublingual medications if uremic symptoms or acute fluid overload arise in the facility.

Care Team Handoffs

Communicate goals clearly across nursing shifts, covering physicians, and covering hospitalists to prevent default emergency transfers.

Reference: Hickam DH, et al. Use of Physician Orders for Life-Sustaining Treatment (POLST). Ann Intern Med. 2013;158(2):113-120.

The Interprofessional PA-LTC Team Approach

Nursing & Therapy Synergy

Nurses: Frontline observers of subtle fluid overload, skin integrity, and behavioral expressions of uremic discomfort.

Therapists (PT/OT): Monitor functional decline, mobility losses, and fatigue thresholds associated with renal failure.

Social Work & Dietary Support

Social Workers: Facilitate family meetings, mediate emotional conflicts, and navigate surrogate dynamics.

Dietitians: Transition residents from restrictive renal diets to pleasurable, comfort-oriented nutrition plans.

Reference: Moss AH. Revised dialysis decision-making recommendations: incorporating palliative care. Clin J Am Soc Nephrol. 2010;5(12):2314-2321.

Mathematical Risk Estimation in Advanced Renal Aging

$$\text{Risk Score} = \beta_1 (\text{Age}) + \beta_2 (\text{Frailty Index}) + \beta_3 (\text{Comorbidity Count})$$

Quantitative scoring models help clinicians estimate 1-year mortality risks in stage 4/5 CKD, guiding discussions away from burdensome treatments when competing mortality risks exceed 80%.

Clinical Utility: Objective risk scores supplement clinical gestalt and reduce decisional conflict among surrogate decision-makers.

Reference: Tangri N, et al. A predictive model for progression of chronic kidney disease to kidney failure. JAMA. 2016;315(15):155-163.

Take-Home Messages: Biology & Protection

Inflammaging & Gut Axis

- Cellular senescence and sterile chronic inflammation drive accelerated CKD progression in older adults.
- Targeting gut-derived uremic toxins and systemic inflammation offers novel therapeutic avenues.

The 4 Pillars of Protection

- RAAS blockade, SGLT2 inhibitors, non-steroidal MRAs, and GLP-1 RAs provide robust cardio-renal protection.
- Always pair aggressive protection with proactive sick-day protocols during acute illness.

Reference: KDIGO 2024 Clinical Practice Guideline. Kidney Int. 2024;105(4S):S117-S314.

Take-Home Messages: Dialysis & GOC Excellence

Dialysis Reality in Frailty

- In frail PA-LTC residents, chronic dialysis frequently fails to extend survival and severely impairs functional quality of life.
- Conservative Kidney Management (CKM) is an active, evidence-backed strategy centered on comfort and symptom control.

Proactive Communication

- Use the Ask-Tell-Ask framework and values-based questioning to guide families through complex decisions before crises occur.
- Honor resident dignity by focusing on what matters most in daily care.

Reference: Tonelli M, et al. Comparison of survival and quality of life in older adults with stage 5 CKD. Kidney Int. 2022;101(3):580-589.

Summary & Take-Home Message

- Treat the patient, not the Creatinine.
- CKD = Systemic Inflammation.
- Prioritize safety: Sick Day Rules and Deprescribing.
- **Closing:** "Protecting the kidney is protecting the heart."

Bibliography and Resources

These are the "Gold Standard" resources for screening, diagnosis, and treatment protocols.

American Diabetes Association (ADA). *11. Chronic Kidney Disease and Risk Management: Standards of Care in Diabetes—2026*. Diabetes Care. 2026;49(Suppl 1):S246-S260. Link

Kidney Disease: Improving Global Outcomes (KDIGO). *2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease*. Kidney International. 2024;105(4S):S117-S314. Executive Summary

ADA/KDIGO Consensus Report. *Diabetes Management in Chronic Kidney Disease: A Consensus Report by the American Diabetes Association and Kidney Disease: Improving Global Outcomes*. (Updated 2024).

Pathophysiology and Mechanisms

•**Frontiers in Medicine.** *Diabetic kidney disease:*
•*from pathogenesis to multimodal therapy–*
•*current evidence and future directions*. 2024;11:1368481. Link

•**American Journal of Physiology.**
•*Advancements in diabetic kidney disease management:*
•*Integrating innovative therapies and targeted drug development*. 2024;326(4).

•**Nature Reviews Nephrology.**
•*Mechanisms of SGLT2 inhibitor-mediated kidney protection*. 2024.

Landmark Clinical Trials(The “Four Pillars”)

SGLT2 Inhibitors	Perkovic V, et al. *Canagliflozin and Renal Outcomes in Type 2 Diabetes and Nephropathy (CREDENCE)*. N Engl J Med.
GLP-1 RAs	Perkovic V, et al. *Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes (FLOW Trial)*. **N Engl J Med. 2024;391:109-121.**
ns-MRA	Agarwal R, et al. *Effect of Finerenone on Chronic Kidney Disease Outcomes in Type 2 Diabetes (FIDELIO-DKD)*. N Engl J Med.
Combination Therapy	Neuen BL, et al. *Estimated Lifetime Benefits of Combination Treatment in Patients With Type 2 Diabetes and Albuminuria*. **JACC. 2024.**

Online Resources & Tools

- **KDIGO CKD Risk Path:**
- An interactive tool for clinicians to stage CKD based on GFR and Albuminuria.

- **NIDDK Health Information:** Kidney Disease Statistics for the United States.

- **Diabetes Atlas (IDF):** Global statistics on the prevalence of DKD