# Same fire, different smoke

Obesity, kidney disease, and the food that puts it out

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WHERE THIS DISEASE ACTUALLY ARRIVES

## A lab panel. No name, no history.

| eGFR | uACR |
|---|---|
| 58 | 180 |
| estimated glomerular filtration rate | urine albumin-to-creatinine ratio, mg/g |

**Both values are abnormal.**

Whoever this belongs to almost certainly feels completely well. The kidney produces no symptom at this stage.

This panel lands in an inbox somewhere in this county several hundred times a day.

**Show of hands**
Who is comfortable making a diagnosis from this?

2

# Chronic kidney disease?

## Yes, no, or not yet?

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THE DEFINITION WE GET WRONG CONSTANTLY

# Not yet. Three months.

**Chronic kidney disease means the abnormality persists at least three months.**

One panel raises the question. Repeat testing answers it.

**The CDC says this about its own headline number:**

> *"The estimates were based on a single measure of urinary albumin-to-creatinine ratio and serum creatinine; they do not account for persistence … Thus, CKD in this report might be overestimated."*

### So we repeat the panel.

Three months later, both numbers are still abnormal.

**Now there is a diagnosis.**

Still no symptoms. Still no idea.

If the CDC has to flag that limitation about its own prevalence estimate, so do we at the bedside.

CDC. Chronic Kidney Disease in the United States. 2026. | 2026 AHA/ACC/ADA/ASN CKM guideline. JACC. 2026.

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WHO THOSE TWO NUMBERS BELONG TO

## 37 million U.S. adults. About 1 in 7.

**87%**

of adults with CKD
did not know they had it

**21%**

of adults with high
blood pressure have CKD

**11%**

of adults with
prediabetes have CKD

**Look around this room and do that math. This is not a talk about other people.**

High blood pressure and prediabetes are not exotic categories. Between them they describe a very large share of the adults in this county.

Kidney function can decline substantially before any symptom appears. Your heart gives you chest pain. Your kidney does not.

CDC. Chronic Kidney Disease in the United States. 2026. Prevalence from NHANES Aug 2021 to Aug 2023, race-free CKD-EPI.

5

PART ONE

# The fire

It is not weight. It is fat tissue that has started behaving badly.

6

WHAT WE WERE TAUGHT WAS A WAREHOUSE

# Fat is an active endocrine and immune organ.

**It releases, continuously:**

- Leptin
- Adiponectin
- IL-6
- TNF-alpha
- MCP-1
- Signals that activate sodium retention and the renin-angiotensin-aldosterone system

So there are two questions here, not one.

## How much fat is there? How is it behaving?

The scale answers only the first.

Some people carry excess fat with preserved metabolic function. Others carry modest excess with badly behaved fat.

**BMI cannot tell them apart.**

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CARDIOLOGY, DIABETES AND NEPHROLOGY NOW AGREE

# Cardiovascular-kidney-metabolic syndrome

| | |
|---|---|
| **Stage 0** | No CKM risk factors |
| **Stage 1** | Excess or dysfunctional adiposity, including prediabetes |
| **Stage 2** | Metabolic risk factors or chronic kidney disease |
| **Stage 3** | Subclinical cardiovascular disease |
| **Stage 4** | Clinical cardiovascular disease |

### Stage 1 begins before diabetes and before kidney disease.

Prediabetes counts as evidence of dysfunctional fat even at a normal BMI.

**That lab panel we opened with, eGFR 58 with albumin in the urine, is already Stage 2.**

Whoever that was skipped the entire stage where this is easy to catch. The ordinary course of this disease, not an unlucky exception.

Ndumele CE, et al. Circulation. 2023;148(20):1606-1635. | 2026 AHA/ACC/ADA/ASN CKM guideline. JACC. 2026.

8

PART TWO

# Five roads from fat to kidney

All five run at the same time. Not one of them produces a symptom you would notice.

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ROAD 1 OF 5 · PRESSURE

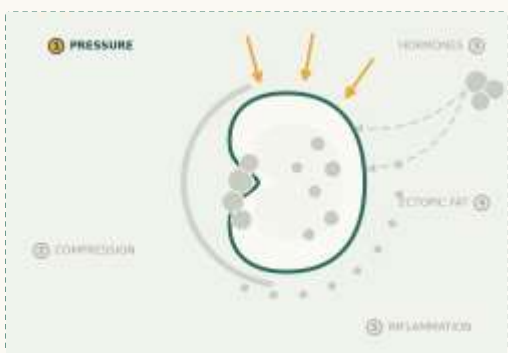
## The filter that works hardest fails first.

Your kidney is a bundle of roughly a million filters called nephrons.

More body mass means more waste to clear, so each filter raises its own rate to keep up. This is called hyperfiltration.

**The result is sustained high pressure inside a structure built for less.**

Nothing about this hurts. There is no early warning.

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WHY TWO IDENTICAL PATIENTS ARE NOT IDENTICAL

## You do not know your own reserve. Neither do we.

**860,000**

nephrons per kidney, on average
Estimated from CT imaging and biopsy in 1,388 1,388 living kidney donors

**People begin adulthood with strikingly different nephron reserves.**

Two people, same BMI, same blood pressure. One has substantial reserve. One is running near the edge.

Neither of them knows, and neither do we.

***Same delivery route. One truck has eight tires, the other has four. Both are legal. Only one survives the potholes.***

Denic A, et al. Single-nephron glomerular filtration rate in healthy adults. N Engl J Med. 2017;376(24):2349-2357.

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ROAD 2 OF 5 · COMPRESSION

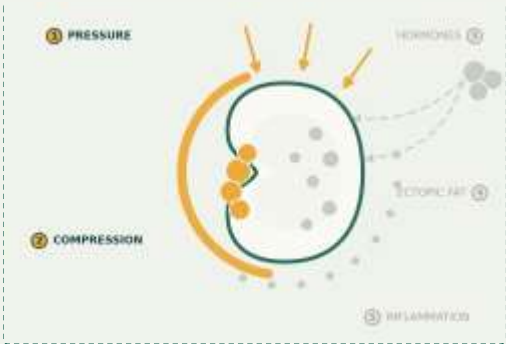
## A plumbing problem stacked on a chemistry problem.

Fat does not only sit under the skin. It accumulates around the the kidney and inside it, in the renal sinus.

That raises pressure within the organ and compresses small vessels and tubules.

**This tracks with hypertension and kidney disease independent of overall BMI.**

Which is part of why a normal-looking BMI is not reassuring.




12

ROAD 3 OF 5 · HORMONES

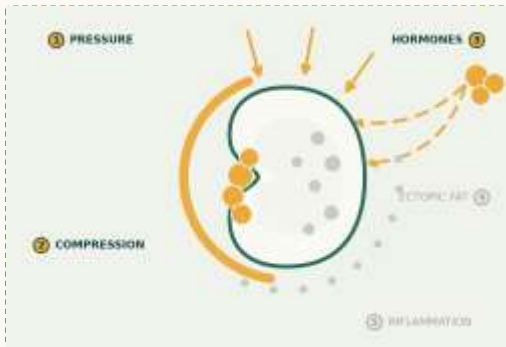
## Dysfunctional fat gives orders.

More aldosterone. More sympathetic nervous system drive. Less adiponectin, which is the protective one.

**Net effect: sodium retention, higher glomerular pressure, vascular remodeling.**

This is part of why blood pressure in obesity is salt-sensitive and rarely controlled on a single drug.

It is also why the same patient often needs three agents and still runs high.

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ROAD 4 OF 5 · ECTOPIC FAT

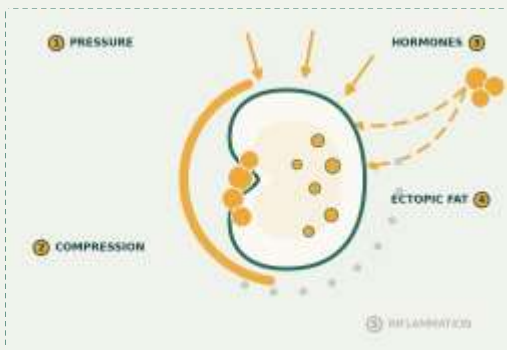
## Podocytes build the filter. They cannot divide.

When storage capacity is exceeded, lipid deposits in tissues never designed to hold it, including kidney cells.

Podocytes form the filtration barrier. Stress them and they enlarge, then detach.

**You do not grow new ones.**

The lesion has a name, obesity-related glomerulopathy, and biopsy series show it rising sharply across recent decades. A multi-society consensus proposed a formal classification in 2025, so this is a live area, not settled history.




Kambham N, et al. Kidney Int. 2001;59(4):1498-1509. | Rico-Fontalvo J, et al. Kidney Int. 2025;108(4):572-583.

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ROAD 5 OF 5 · INFLAMMATION

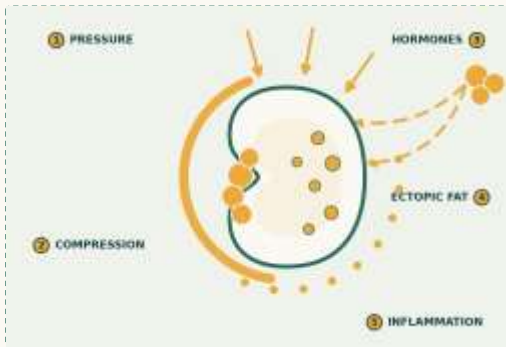
## No resolution phase.

Visceral fat recruits immune cells and produces inflammatory inflammatory signals continuously.

**This is not an infection. Nothing turns it off.**

And it accelerates the other four roads rather than running alongside them.

Five roads, all open at once, none of them painful. That is the whole problem in one sentence.

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WHAT THE FIVE ROADS ADD UP TO

## Risk of kidney failure, by body mass index

| Category | | Relative risk | 95% CI |
|---|---|---|---|
| **Overweight** | BMI 25.0 to 29.9 | **1.87x** | 1.64 to 2.14 |
| **Obesity class I** | BMI 30.0 to 34.9 | **3.57x** | 3.05 to 4.18 |
| **Obesity class II** | BMI 35.0 to 39.9 | **6.12x** | 4.97 to 7.54 |
| **Obesity class III** | BMI 40 or above | **7.07x** | 5.37 to 9.31 |

**320,252 people**

8.3 million person-years. 1,471 cases of kidney failure. Held after adjusting for blood pressure and diabetes.

**The limitation, from the authors:**

single measurements of BMI, and BMI cannot distinguish muscle from fat or say where fat sits. The gradient is real. The ruler is crude.

Hsu CY, McCulloch CE, Iribarren C, Darbinian J, Go AS. Body mass index and risk for end-stage renal disease. Ann Intern Med. 2006;144(1):21-28.

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BEYOND ONE COHORT

# Obesity and incident kidney disease

### Meta-analysis of 39 cohorts, 630,677 participants, mean 6.8 years

Obesity raised risk of low eGFR by 28% (RR 1.28, 95% CI 1.07 to 1.54) and albuminuria by 51% (RR 1.51, 95% CI 1.36 to 1.67). Each additional BMI unit raised both.

### Obesity-related glomerulopathy in biopsy series

In 6,818 native kidney biopsies, incidence rose from 0.2% of biopsies in 1986 to 1990, to 2.0% in 1996 to 2000. A tenfold rise across fifteen years, and that series ended twenty-five years ago.

### Nephron measurement method

1,388 living kidney donors. Iothalamate-measured GFR, CT cortical volume, and biopsy-determined glomerular density. Mean 860,000 nephrons per 860,000 nephrons per kidney with wide interindividual variation. Low birth weight and prematurity reduce nephron endowment.

Garofalo C, et al. Kidney Int. 2017;91(5):1224-1235. | Kambham N, et al. Kidney Int. 2001;59(4):1498-1509. | Denic A, et al. N Engl J Med. 2017;376(24):2349-2357.

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HOW YOU SEE INVISIBLE SMOKE

# Urine albumin-to-creatinine ratio

**It can reveal glomerular injury while eGFR is still preserved.**

One urine sample.

Follow the diabetes-specific and CKD-specific guidance on top of the intervals at right.

**Screening frequency is risk-stratified. It is not annual uACR for everyone with obesity.**

### Testing interval by CKM stage

- **Stage 0**
  Kidney function every 5 years
- **Stage 1**
  eGFR every 2 to 3 years
- **Stage 2 and above**
  eGFR and uACR at least yearly

The same guideline also directs clinicians to screen for food insecurity, housing instability and financial strain. It is months old, and most people in this county who qualify have never had the test.

2026 AHA/ACC/ADA/ASN guideline for cardiovascular-kidney-metabolic syndrome. J Am Coll Cardiol. 2026.

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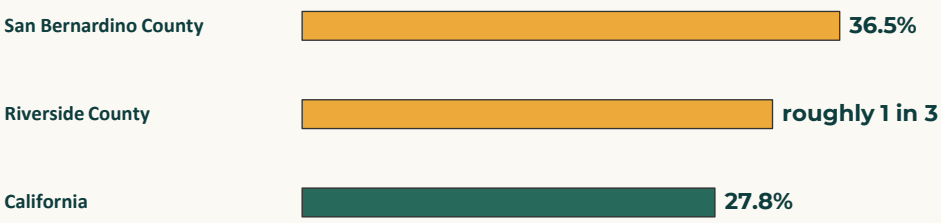
NOT NATIONAL NUMBERS. HERE.

## The fire does not burn evenly




Adult obesity prevalence, age-adjusted

More than 1 in 8 residents of both counties is food insecure. San Bernardino's obesity rate runs about nine nine percentage points above the state's.

San Bernardino County Community Indicators. Riverside County figure pending confirmation from SHAPE Riverside. Food insecurity figure is 2019, the most recent comparable county data.

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INCIDENCE OF KIDNEY FAILURE VS NON-HISPANIC WHITE ADULTS

## Four times. And twice.

**4x**
Black adults

**2x**
Hispanic adults

**Both of those matter in this county.**

APOL1 gene variants explain a real share of the non-diabetic portion of the Black to White difference.

They do not explain all of it, and they do not explain the Hispanic disparity as a whole.

In cohorts matched for income and education, the gap narrows and does not close.

**Genetics is part of the explanation. It has been used as an excuse for the rest.**

CDC 2026, citing USRDS 2025 Annual Data Report. | Cerdeña JP, Tsai J, Grubbs V. APOL1, Black race, and kidney disease: turning attention to structural racism. Am J Kidney Dis. 2021;77(6):857-Kidney Dis. 2021;77(6):857-860.

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WHAT WE ARE WILLING TO PAY FOR

# $141 billion

Medicare fee-for-service spending on beneficiaries with chronic kidney disease, 2023

About 831,000 Americans were living with kidney failure in 2023, about 1 in 400 people. 67% were on dialysis, 33% were living with a transplant. They account for a wildly disproportionate share of Medicare spending relative to their number.

**We will pay for the dialysis chair.**
**We will not pay for the produce.**

USRDS 2025 Annual Data Report. | CDC. Chronic Kidney Disease in the United States. 2026.

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PART THREE

# The food that puts it out

Starting with the strongest study design, not the best headline.

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FIRST THE MECHANISM, THEN THE FOOD

# A failing kidney cannot clear acid well.

**Metabolic acidosis is associated with faster CKD progression.**

Correcting it slowed kidney decline in randomized trials, though not every acidosis trial has been positive.

Here is the strongest randomized evidence.

**Kidney function decline over 2 years**

Standard care
**5.93**
Sodium bicarbonate
**1.88**

mL/min/1.73 m squared

134 patients, randomized. Roughly two thirds slower decline, from baking soda.

## If baking soda can do that, what else lowers dietary acid load?

**Fruits and vegetables are base-producing.**

**Meat are acid-producing.**

Which turns a pharmacologic question into a dietary one, and that is testable.

de Brito-Ashurst I, Varagunam M, Raftery MJ, Yaqoob MM. J Am Soc Nephrol. 2009;20(9):2075-2084.

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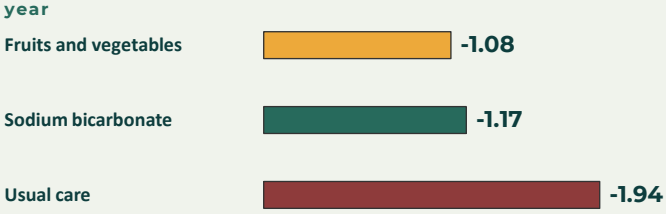
RANDOMIZED · 5 YEARS · 153 PARTICIPANTS

# Produce matched the drug on kidney function.

**Annual eGFR change over five years, mL/min/1.73 m squared per year**




Longer bar is worse. Participants had hypertension and protein in the urine, on standard standard protective medication. Produce dosed to cut dietary acid in half.

**The qualifier**

Those were risk markers and medication medication use. Not heart attacks, not strokes, not deaths. And the study population was highly selected.

**Produce also improved blood pressure and cardiovascular risk markers more than bicarbonate did, on lower doses of medication.**

Goraya N, Madias NE, Simoni J, Kahlon M, Aksan N, Wesson DE. Am J Med. 2024;137(11):1114-1127.e8.

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WHERE THIS EVIDENCE FALLS SHORT

## Not yet definitive.

| | |
|---|---|
| **Small trials** | Tens to low hundreds of participants, not thousands. thousands. |
| **Selected patients** | Hypertensive or non-diabetic, macroalbuminuric, carefully monitored. |
| **One research group** | Most of this randomized work comes from a single single investigative team. |
| **Some analyses exploratory exploratory** | Not every supporting outcome was pre-specified. specified. |

### And yet.

This is still more randomized evidence than most of what we recommend confidently in kidney nutrition.

Which tells you something uncomfortable about kidney nutrition.

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READ THE LABELS, NOT THE FOOD NAMES

## Which of these has potassium added to it?


**You cannot answer this from the food category.** Potassium content and additive use vary brand to brand. Two of these four list potassium chloride or potassium phosphate. The answer is on the package, not in the food group.

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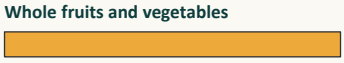
RETIRING FORTY YEARS OF ADVICE

# Additives, not apples.

**Potassium recovered in urine**

**Whole fruits and vegetables**



**77%**

**Higher-bioaccessibility diet**

**96%**

11 healthy volunteers, short crossover feeding study. The higher-bioaccessibility arm was built from animal foods and processed or juiced fruit.

### Two caveats

Urinary recovery is not the same as gastrointestinal absorption. And this experiment did not specifically test test potassium chloride additives.

### Why the conclusion holds

KDIGO 2024 reviewed this and concluded clinicians should limit highly processed foods rich in bioavailable potassium rather than indiscriminately restricting fruits, vegetables, legumes and whole grains.

### Where restriction genuinely is right

- Advanced CKD
- Low urine output
- RAAS inhibitor plus mineralocorticoid antagonist
- Hypoaldosteronism
- Any prior hyperkalemia

**These patients need a plan built for them. Not a blanket ban on apples. on apples.**

Naismith DJ, Braschi A. Int J Food Sci Nutr. 2008;59(5):438-450. | KDIGO 2024 CKD guideline. Kidney Int. 2024;105(4S):S117-S314. | Phosphorus follows the same logic; more in a moment.

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FIBER AND THE GUT-KIDNEY AXIS

# Fiber works on the kidney through the gut.

### The mechanism

Fermentable fiber feeds the gut bacteria that ferment carbohydrate and starves the ones that ferment protein into uremic toxins. The result is lower production of indoxyl sulfate and p-cresyl sulfate, toxins a failing kidney cannot clear.

### The trial data

Randomized fiber supplementation lowered free indoxyl sulfate by 29% in hemodialysis patients. Across 14 controlled feeding trials, added fiber reduced serum urea and creatinine. In NHANES III, each 10 g/day of fiber was linked to 38% lower odds of elevated C-reactive protein in kidney disease.

### The bottom line

Fiber earns its place through the gut-kidney axis, delivered as beans, whole grains, fruits and vegetables. The long-term CRIC cohort did not confirm an association with hard outcomes, so those trials still need to be run. That is a reason to run them, not a reason to skip the beans.

Sirich TL, et al. Clin J Am Soc Nephrol. 2014;9(9):1603-1610. | Chiavaroli L, et al. Eur J Clin Nutr. 2015;69(7):761-768. | Krishnamurthy VM, et al. Kidney Int. 2012;81(3):300-306. | Pradhan N, et al. J Ren Nutr. 2024;35(1):110-117.

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THE REST OF THE FOOD EVIDENCE

## Diet quality, phosphorus, and TMAO

### Plant-based diet quality in established CKD (CRIC, n=2,539)

Highest versus lowest adherence to a healthy plant-based diet: 21% lower all-cause mortality (HR 0.79, 0.66 to 0.95). Each 10-point higher unhealthy plant-based index score: 14% higher risk of CKD progression. Plant-based is not automatically protective. Whole is doing the work.

### Ultra-processed food

Roughly 24% higher incident CKD in ARIC and 25% in UK Biobank, highest versus lowest quartile of intake.

### Phosphorus source

Inpatient crossover, n=9, mean eGFR 32. One week vegetarian versus one week meat protein with equivalent nutrients produced lower serum phosphorus and phosphorus and lower FGF23 on the vegetarian week. A mechanism demonstration, not an outcome trial. Plant phosphorus is largely phytate-bound and poorly phytate-bound and poorly absorbed; animal-source is more available; inorganic additives most of all.

### TMAO

Higher plasma levels predicted mortality in 521 patients with stable CKD, but the analysis is observational and kidney clearance strongly influences TMAO. Do not influences TMAO. Do not present animal food to TMAO to kidney death as an established causal chain.

Amir S, Am J Kidney Dis 2024 | Ren Fail 2024 | Moe SM, CJASN 2011 | Tang WHW, Circ Res 2015. Full citations on the references slide.

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FOOD OR MEDICATION? WRONG QUESTION.

## Semaglutide, major kidney events

**23.2%**

Placebo
410 of 1,766

**18.7%**

Semaglutide
331 of 1,767

### Both numbers are true. Ask for the second one.

**24% relative reduction.** About 4 to 5 percentage points absolute over 3.4 years. Roughly 19 of every 100 treated people still reached the outcome. You will almost always be handed the relative number alone.

### These drugs work.

They are the most important advance in kidney medicine in a generation. Anyone who who says food replaces them is not doing you doing you a favor.

SGLT2 inhibitors and finerenone point the same direction in their own randomized trials.

Perkovic V, Tuttle KR, Rossing P, et al. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med. 2024;391(2):109-121.

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THE LIMIT OF PHARMACOLOGY

# A GLP-1 does not build a grocery store.

**It does not change the food environment**

Not what is served at a church supper, a school lunch, or a clinic vending machine. machine.

**Weight recurrence reactivates the biology**

Hypertension, diabetes, albuminuria and the other obesity-related pathways can return. Kidney-specific recurrence estimates remain uncertain.

**Long-term protection needs a durable plan**

Food quality, physical activity, medication or surgery when indicated, behavioral support, and continued follow-up. Not one thing.

### Not a nephrology opinion.

The 2025 multi-society consensus on obesity-related kidney disease says the the same, explicitly calling for multidisciplinary management including including nutrition, physical activity, pharmacotherapy and bariatric surgery surgery where indicated.

Three societies across two continents, and none of them framed this as a choice between food and medicine.

Rico-Fontalvo J, Ciudin A, Correa-Rotter R, et al. Kidney Int. 2025;108(4):572-583.

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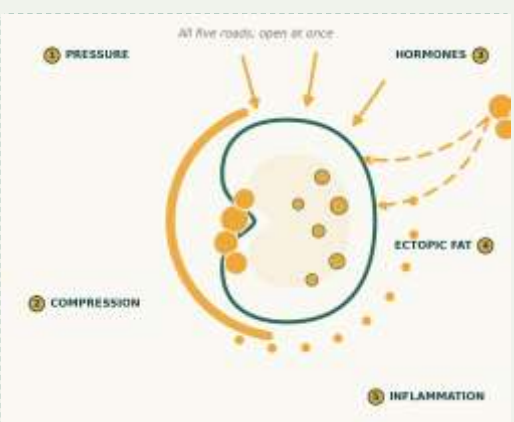
PLAUSIBLE MECHANISTIC REACH. NOT ALL LINKS ARE PROVEN IN OUTCOME TRIALS.

# Which intervention reaches which road?




| | |
|---|---|
| **Dietary acid reduction** | Pressure · Hormones |
| **Weight reduction, any means** | All five |
| **SGLT2 inhibitor** | Pressure |
| **GLP-1 receptor agonist** | Pressure · Hormones · Ectopic fat · Inflammation |
| **Finerenone** | Hormones · Inflammation |
| **Whole-food dietary pattern** | Hormones · Ectopic fat · Inflammation |
| **Less ultra-processed food** | Hormones · Ectopic fat · Inflammation |

**No single row covers all five roads, and the rows overlap. That is why the argument is not food versus drugs.**

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THREE LANES IN THIS ROOM. PICK YOURS.

## Choose your lane

### Your body

- Ask whether you need a uACR
- Beans instead of processed meat, one meal a day
- Read the ingredient list for “phos” and potassium chloride
- Drop sugary drinks first
- Stop fearing produce unless a nephrologist named it

### Your clinic

- eGFR and uACR at least yearly at CKM Stage 2 and above
- No blanket produce restrictions in early CKD
- Stage CKM. Treat Stage 1 as a diagnosis
- Screen for food insecurity. Know two local resources by name
- Prescribe both. Drugs and food

### Your institution

- Change what your building serves before you change what you tell people to eat
- Make beans and whole grains the default, not the option
- Host uACR screening where people already gather
- Read your food procurement contract

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## The kidney will not tell you when the fire starts.

## Go looking.

We opened with two numbers and no story attached, because that is how this disease actually arrives. Not as a
Not as a symptom. As a lab value that somebody either follows up or does not.

The only signal you get is the one you go looking for. And food quality is one of the few interventions that can influence several of these mechanisms at the same time.

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THANK YOU

# Questions

**SELFPrinciple.org**

YouTube: @SeanHashmiMD

Evidence-based resources. No products, no sponsors.

**Sean Hashmi, MD, MS, FASN**

Triple board certified: internal medicine, nephrology, obesity medicine

**Clinical disclaimer**

This content is for general education only and is not medical advice. It doesn't replace a conversation with your own your own physician. Talk to your doctor before changing your diet, medications, or treatment.

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