The Art of MNT for CKD: Beyond the Guidelines

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"The art of medicine is making good decisions based on poor information."

Author unknown

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Challenges of Providing MNT

- Limited evidence-based nutrition research
- Dependence on consensus/opinion
- Conflicting recommendations
- Realities of implementing dietary restrictions
- Questionable outcome indicators to measure nutrition status
- Reliance on patient self-report

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AGENDA

- KDIGO Guidelines 2013
- Practice beyond the guidelines
- Hot topics
  - Phosphorus
  - Fructose/Uric Acid
- Putting it all together

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Comparison of Diets for CKD

	CKD	HEMO	PD HOME HEMO	TRANS.
Protein	Low	High	Higher	Varies
K+	Varies	Low	High	Varies
PO4	Low	Low	Low	Liberal
Ca	Supplement?	Supplement?	Supplement?	Supplement?
Na+	1500-2000 mg	2000-2400 mg	2000-3000 mg	2000-3000 mg
Fluids	Varies	Limit	Liberal	Liberal
Calories	Varies	Varies	Limit	Varies

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KDOQI 2000

Table 10. Stages of Chronic Kidney Disease

Stage	Description	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

Kidney International, January 2013, Vol 3, Issue 1, Supplements

kdigo.org

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

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GFR Equations

- Equations use serum creatinine, age, gender, weight, and race
- Cockcroft-Gault equation
- Modification of Diet in Renal Disease (MDRD)
- **New equation**: Chronic Kidney Disease Epidemiology Collaboration **(CKD-EPI)** 2010
- Calculator online: kidney.org

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Evidence Grading

Level 1

*"We **recommend** . . ."*

Level 2

*"We **suggest** . . ."*

Proteinuria Predicts Progression

- Urine Albumin/Creatinine Ratio
  - Normal 0-29
  - Albuminuria >30

- Urine Protein/Creatinine Ratio
  - Measure if albuminuria >300
  - Ratio ≈ g protein excretion (24°)
  - Target <1.0

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GFR and Cr Caveats

Factors affecting serum creatinine levels:

Age	↓
Female	↓
African American	↑
Vegetarian diet	↓
Ingestion of meat	↑
Muscular habitus	↑
Muscle wasting	↓
Obesity	No change
Certain medications	↑

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My favorite guideline:

3.1.22: We recommend that individuals with CKD receive expert dietary advice and information in the context of an education program, tailored to severity of CKD and the need to intervene on salt, phosphate, potassium, and protein intake where indicated (1B)

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KDIGO Suggestions for Protein

Protein intake:

3.1.13: 0.8 g/kg/day in adults, with appropriate education

3.1.14: Avoid high protein intake

(>1.3 g/kg/day)

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What weight to use?

- Ideal Body Weight (IBW)
- Standard Body Weight (NHANES II)*
- Adjusted Body Weight*
  - Amputations
  - Obesity/underweight
  - Edema-free

***KDOQI recommendation, opinion**

KDIGO: no comment

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Hypoalbuminemia May ≠ Malnutrition

- Proteinuric effect
- Albumin is acute phase reactant
- Malnutrition-Inflammation Complex Syndrome (MICS)*
  - May account for high rate of CVD in CKD
  - May contribute to “reverse epidemiology” of dialysis survival

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*Fouque, et al, “A proposed nomenclature and diagnostic criteria for protein-energy wasting in acute and chronic kidney disease”, Kidney International, 2008, 73:391-398

KDIGO: Glycemic Control

3.1.15: Recommend target HbA1c of ~7% to prevent or delay progression of microvascular complications of DM including DKD

3.1.16: Recommend not treating to 7% target in those at risk for hypoglycemia

3.1.17: Suggest HbA1c target extended above 7% in those with co-morbidities or limited life expectancy

3.1.18: Be part of multifactorial intervention

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DM ➔ CKD

- 25-40% of pts w/ T1 or T2 DM develop diabetic nephropathy within 20-25 years of onset
- DM is independent risk factor for death due to CVD
- Twice the mortality in pts w/DM + albuminuria
- In Hawai'i, ~70% ESRD pts have DM

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Interpreting HbA1c in DKD

Remember:

- People with CKD may have red cell life span <90 days resulting in falsely low HbA1c
- May need to review actual blood sugar logs
- Watch for hypoglycemia (and unawareness), with decreased insulin clearance

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DM Medication Caveats

- Avoid renally excreted meds (e.g. glyburide)
- Dose adjustments for insulin when GFR < 30 – watch for hypoglycemia
- Metformin
  - Review when GFR <45
  - Suggest avoid when GFR <30

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KI Supplements (2013) **3**, 91-111

KDIGO: Sodium

3.1.19: Recommend lowering Na intake to <2000 mg/day, unless contraindicated

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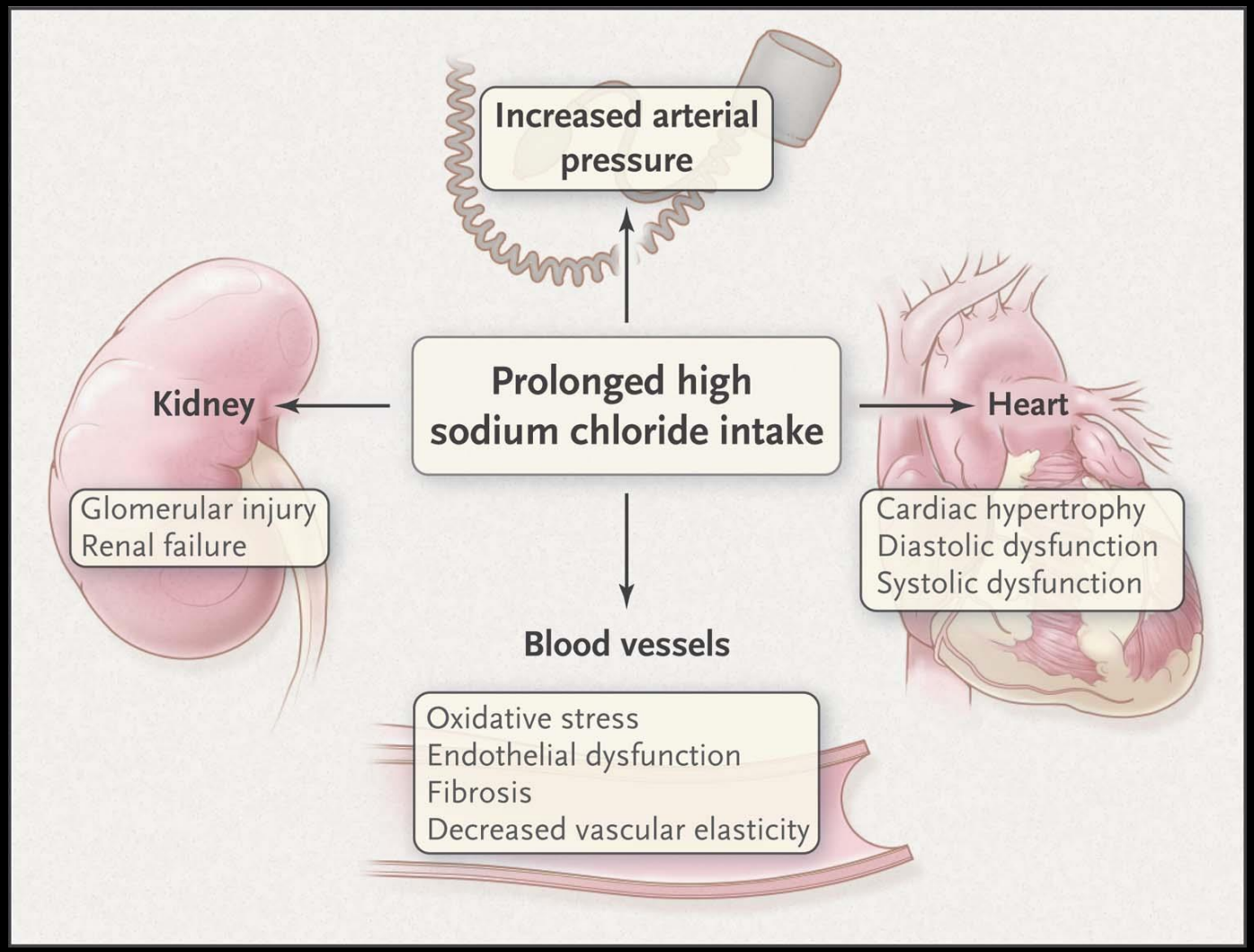
Target-Organ Damage Due to High Intake of Sodium Chloride.



Kotchen TA et al. N Engl J Med 2013;368:1229-1237

Sodium

- HTN: treatment slows progression of CKD
- Target blood pressure: 140/90 mm Hg with microalbuminuria
- Target blood pressure 130/80 mm Hg with albuminuria or proteinuria
- High Na intake ↑ BP, ↑ proteinuria, blunts RAAS blockade
- Na sensitivity

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Kidney International Supplements, KDIGO Clinical Practice Guidelines for the Management of Blood Pressure in CKD; Dec 2012, 2 (5)

KDIGO: Phosphorus

Diagnosis, Evaluation, Prevention and Treatment of Chronic Kidney Disease related Mineral and Bone Disorders

(CKD-MBD)

Kidney International: 76, Supplement 113; August, 2009

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Phosphorus Restriction

- ↓ Urinary excretion and ↑ serum levels when GFR declines to <40-45
- ↑ P levels increase PTH (parathyroid hormone) and FGF23 (fibroblast growth factor
- ↑ Risk for vascular calcifications and mortality

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Understanding Sources of Dietary Phosphorus in the Treatment of Patients with Chronic Kidney Disease

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In individuals with chronic kidney disease, high dietary phosphorus (P) burden may worsen hyperparathyroidism and renal osteodystrophy, promote vascular calcification and cardiovascular events, and increase mortality. In addition to the absolute amount of dietary P, its type (organic *versus* inorganic), source (animal *versus* plant derived), and ratio to dietary protein may be important. Organic P in such plant foods as seeds and legumes is less bioavailable because of limited gastrointestinal absorption of phytate-based P. Inorganic P is more readily absorbed by intestine, and its presence in processed, preserved, or enhanced foods or soft drinks that contain additives may be underreported and not distinguished from the less readily absorbed organic P in nutrient databases. Hence, P burden from food additives is disproportionately high relative to its dietary content as compared with natural sources that are derived from organic (animal and vegetable) food proteins. Observational and metabolic studies indicate nutritional and longevity benefits of higher protein intake in dialysis patients. This presents challenges to providing appropriate nutrition because protein and P intakes are closely correlated. During dietary counseling of patients with chronic kidney disease, the absolute dietary P content as well as the P-to-protein ratio in foods should be addressed. Foods with the least amount of inorganic P, low P-to-protein ratios, and adequate protein content that are consistent with acceptable palatability and enjoyment to the individual patient should be recommended along with appropriate prescription of P binders. Provision of in-center and monitored meals during hemodialysis treatment sessions in the dialysis clinic may facilitate the achievement of these goals.

Clin J Am Soc Nephrol 5: 519–530, 2010. doi: 10.2215/CJN.06080809

Chronic kidney disease (CKD) affects >20 million Americans and is associated with high morbidity and mortality (1). The progressive deterioration of kidney function in CKD leads to retention of many substances, including phosphorus (P), that are normally excreted by the kidney. Serum P concentration, however, is usually maintained within the normal range of 2.5 to 4.5 mg/dl by a variety of compensatory mechanisms until renal disease has progressed to approximately stage 5 CKD or ESRD (2). An effective mechanism is the reduction in renal tubular absorption of phosphate (PO_4; *i.e.*, increased fractional excretion of P regulated by parathyroid hormone [PTH] and the phosphatonin fibroblast growth factor 23) (3,4).

In recent years, a number of epidemiologic studies have shown an association between high serum P levels and increased death risk in both dialysis-dependent patients with ESRD (5,6) and individuals with less advanced stages of CKD (7), hyperphosphatemia in these latter patients also seems to be associated with a faster rate of CKD progression (8). Indeed, emerging data indicate that in individuals who do not have apparent CKD and have high normal serum P levels, the risk for coronary artery calcification and mortality is increased (9–11). Hence, relative hyperphosphatemia may represent a novel cardiovascular and death risk factor (12). Similarly, it is possible, although not yet proved, that interventions aimed at dietary P restriction may improve cardiovascular profile and survival even in individuals with high-normal or borderline elevated serum P levels.

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The Element Phosphorus

P, a multivalent nonmetal element of the nitrogen group (group 15) of the periodic table, is naturally found in inorganic PO_4 rocks. Because of its high reactivity, P is almost never found as a free element in nature but is present almost exclu-

ISSN: 1555-9041 (5/3: 0519

Phosphorus Bioavailability

SOURCE	ABSORPTION
Animal	40-60%
Plant	<50%
Inorganic (additives)	>90%

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P to Protein Ratios

	mg P	g Pro	Ratio
Egg white, 1 large	5	3.6	1.4
Canned tuna, water pack, 3 oz	139	21.7	6.7
Canned tuna, oil pack, 3 oz	265	24.8	10.7
Soy milk, 8 oz	120	6.8	17.4
Cow's milk, 8 oz	229	8.1	28.3
Liquid non-dairy creamer, 1 oz	19	0.3	63.3
Cola soda	65	0	65

KDOQI: Uric Acid

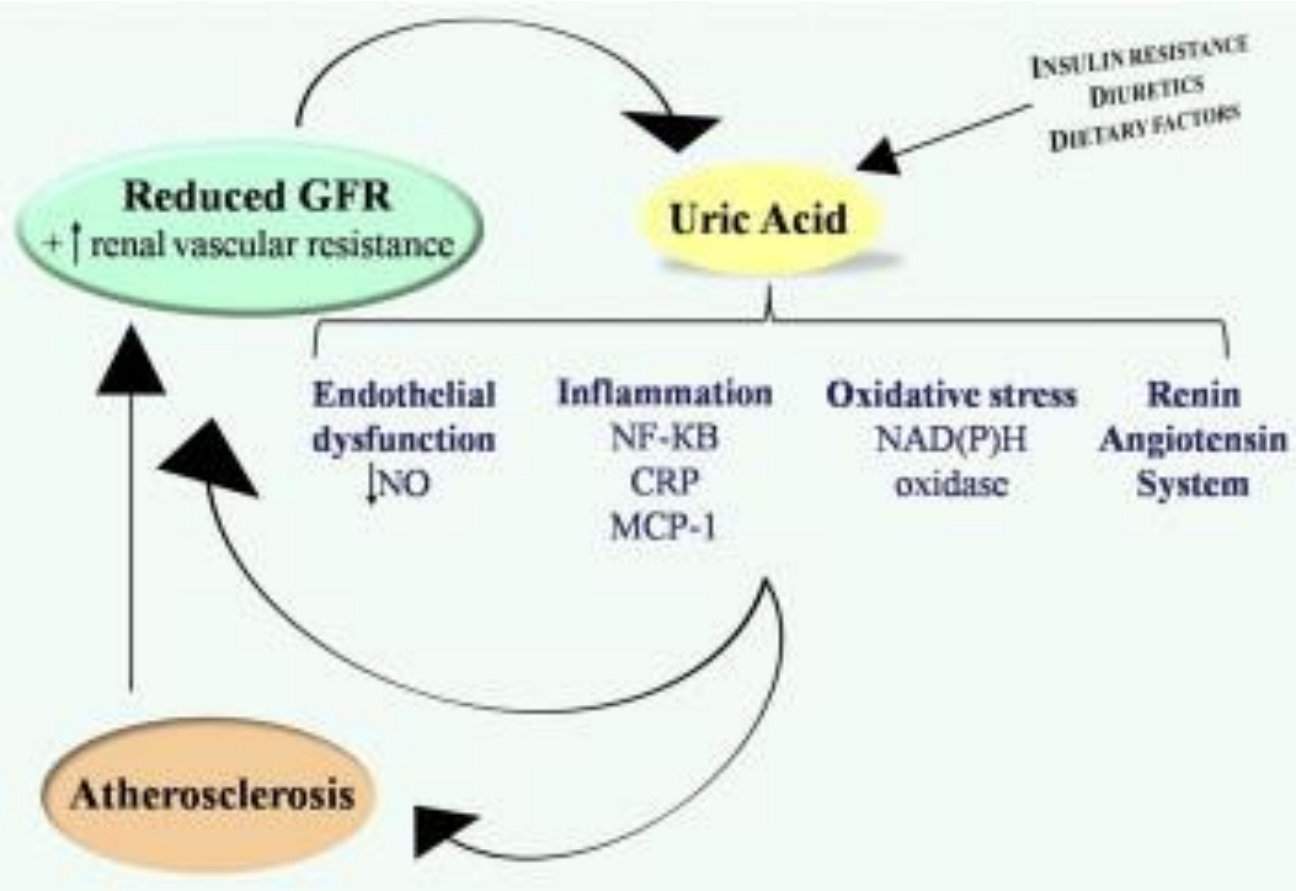
- 3.1.20: Insufficient evidence to support or refute the use of agents to lower serum uric acid concentrations in people with CKD and either symptomatic or asymptomatic hyperuricemia in order to delay progression of CKD (not graded).

However . . .

NHANES 2007-2010

GFR	Prevalence of Gout
≥ 90	1.59%
60-89	5.73%
30-59	12.93%
<30	29.81%

What's the connection?



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Jalal, et al, Uric Acid as a Target of Therapy in CKD, Am J Kidney Dis, 2013; 61(1):134-146

ACKD
Advances in Chronic Kidney Disease

National Kidney Foundation

Vol 19, No 6, November 2012

EDITORIAL

Uric Acid: A Clearer Focus

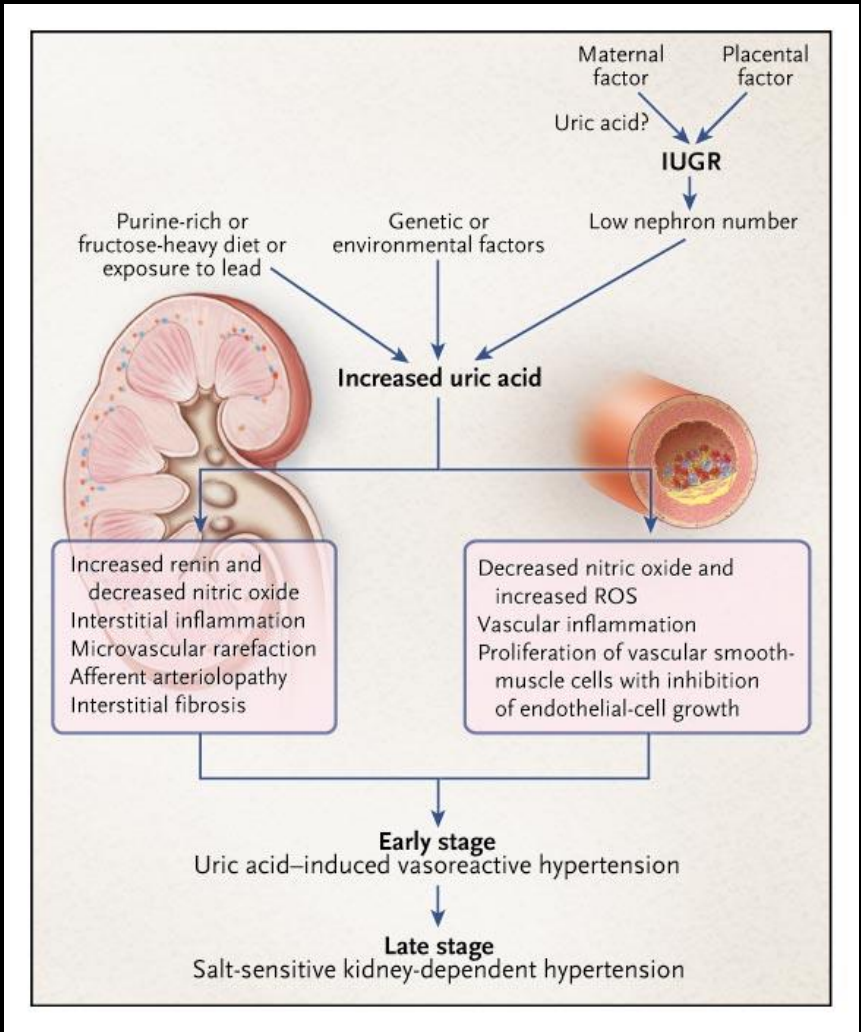
Plasma phosphorus and uric acid/urate levels were removed from clinical laboratories' metabolic panels nearly 3 decades ago. There was insufficient evidence for their continued measurement. Moreover, fears of treating asymptomatic hyperuricemia with allopurinol, with its risk of side effects and hypersensitivity, contributed to uric acid's removal from the metabolic panel. Subsequently, the evidence base for both of these nephrocentric molecules developed. This issue of Advances in *Chronic Kidney Disease* converges on uric acid/urate, which gained preeminence in hominoids by virtue of the loss of the *uricase* gene during evolution through the Miocene Epoch.

The guest editors, Drs. Anthony J. Bleyer and Stanislav Kmoch have, like a large reflecting telescope, collected an impressive amount of information from their respective

URAT1 (uric acid transporter-1 protein)[5] and GLUT9a (glucose transporter-like protein 9a), now referred to simply as GLUT9.[6] It is now clear that urate is not reabsorbed and secreted, contradicting the classic model of presecretory reabsorption, secretion, and postsecretory reabsorption. Also, it is now appreciated that the uricosuric agents losartan, furosemide, probenecid, and benzbromarone inhibit apical URAT1, whereas pyrazinamide stimulates this transporter. On the basolateral aspect, GLUT9 is inhibited by losartan, probenecid, and benzbromarone, thereby inducing uricosuria.[5]

The incidence and prevalence of gout and uric acid stones have risen concomitantly with the ongoing epidemic of obesity, metabolic syndrome, and type 2 diabetes. The culprit for gout is likely endogenous uric acid overproduction fueled by the exogenously introduced

Proposed Mechanism for Uric Acid–Mediated Hypertension



Feig D et al. N Engl J Med 2008;359:1811-1821

Cardiovascular Conditions and Risk Factors Associated with Elevated Uric Acid

Table 1. Cardiovascular Conditions and Risk Factors Associated with Elevated Uric Acid.

Hypertension and prehypertension
Renal disease (including reduced glomerular filtration rate and microalbuminuria)
Metabolic syndrome (including abdominal obesity, hypertriglyceridemia, low level of high-density lipoprotein cholesterol, insulin resistance, impaired glucose tolerance, elevated leptin level)
Obstructive sleep apnea
Vascular disease (carotid, peripheral, coronary artery)
Stroke and vascular dementia
Preeclampsia
Inflammation markers (C-reactive protein, plasminogen activator inhibitor type 1, soluble intercellular adhesion molecule type 1)
Endothelial dysfunction
Oxidative stress
Sex and race (postmenopausal women, blacks)
Demographic (movement from rural to urban communities, Westernization, immigration to Western cultures)

Feig D et al. N Engl J Med 2008;359:1811-1821

The NEW ENGLAND JOURNAL of MEDICINE

Where is the uric acid coming from?

FRUCTOSE metabolism

⬇

rapidly depletes ATP in liver

⬇

generating adenosine (purine)

⬇

uric acid

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ARTICLES

Hypothesis: Could Excessive Fructose Intake and Uric Acid Cause Type 2 Diabetes?

Richard J. Johnson, Santos E. Perez-Pozo, Yuri Y. Sautin, Jacek Manitius, Laura Gabriela Sanchez-Lozada, Daniel I. Feig, Mohamed Shafiu, Mark Richard J. Glassock, Michiko Shimada, Carlos Roncal and Takahiko Nakagawa

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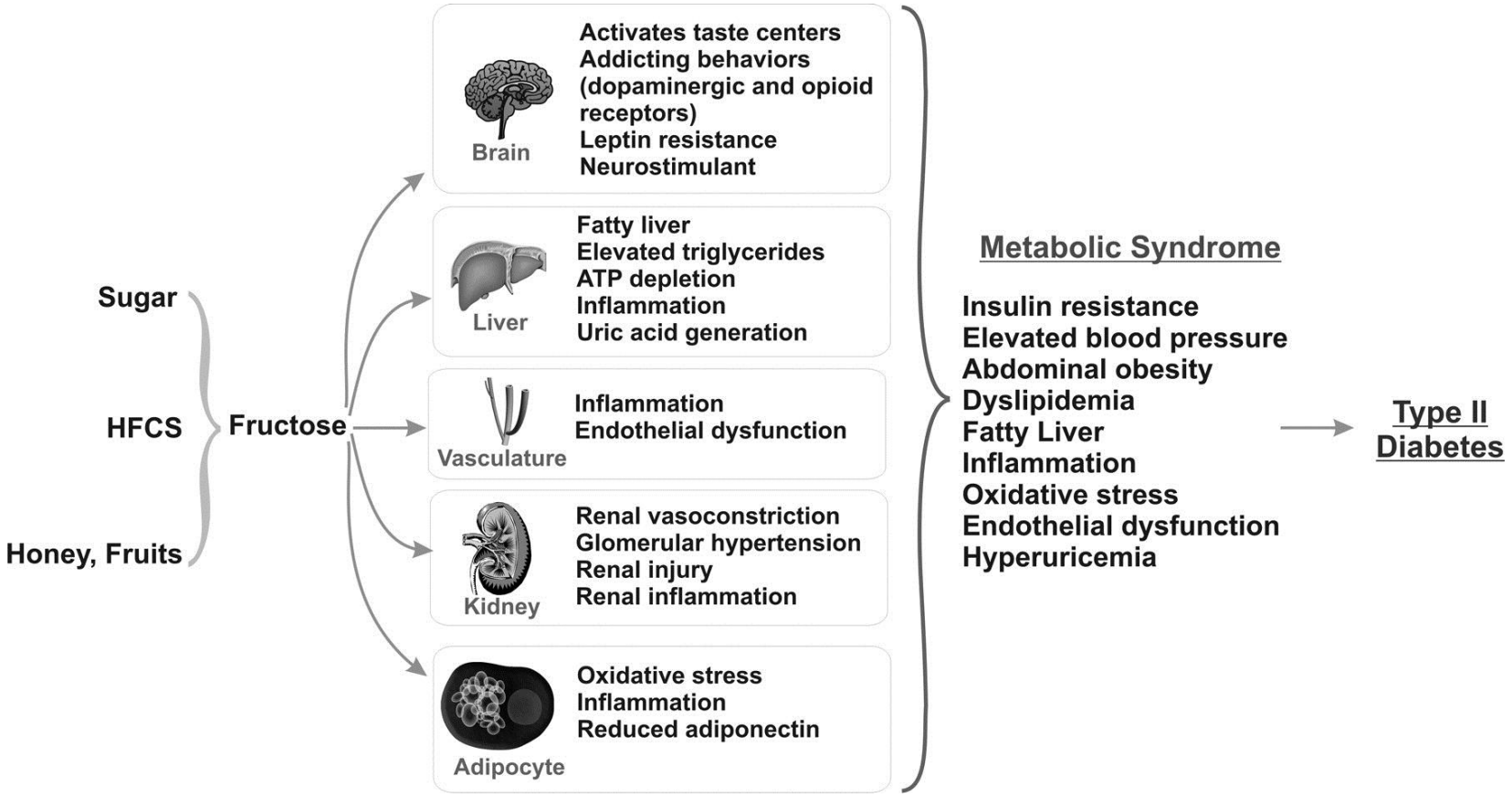
Abstract

We propose that excessive fructose intake (>50 g/d) may be one of the underlying etiologies of metabolic syndrome and type 2 diabetes. The primary sources of are sugar (sucrose) and high fructose corn syrup. First, fructose intake correlates closely with the rate of diabetes worldwide. Second, unlike other sugars, the i of excessive fructose induces features of metabolic syndrome in both laboratory animals and humans. Third, fructose appears to mediate the metabolic syndrom by raising uric acid, and there are now extensive experimental and clinical data supporting uric acid in the pathogenesis of metabolic syndrome. Fourth, enviro and genetic considerations provide a potential explanation of why certain groups might be more susceptible to developing diabetes. Finally, we disc counterarguments associated with the hypothesis and a potential explanation for these findings. If diabetes might result from excessive intake of fructose, the public health measures could have a major impact on improving the overall health of our populace.

Effect of fructose on various organ systems.



Johnson R J et al. Endocrine Reviews 2009;30:96-116

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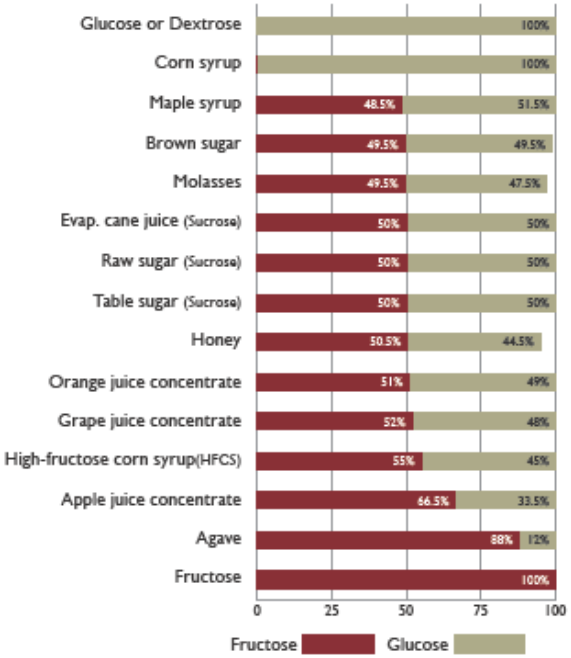
ENDOCRINE REVIEWS

SUGAR IN FOOD

How Much Sugar Should You Eat?

With a few exceptions (like agave and corn syrup), most sweeteners and the naturally occurring sugars in fruit break down into roughly half fructose and half glucose in the body.* The natural sugar in milk (lactose) breaks down into half glucose and half galactose.



*Sucrose is shown as its component sugars (fructose and glucose).
Note: If percentages don't add up to 100, other sugars account for the difference.
Sources: USDA Nutrient Database and company information.

The Bottom Line

- Shoot for 100 calories (6½ teaspoons) a day of added sugars if you're a woman and 150 calories (9½ teaspoons) a day if you're a man. Even less may be better for your heart.
- Don't drink sugar-sweetened beverages. Limit fruit juices to no more than 1 cup a day.
- Limit all added sugars, including high-fructose corn syrup, cane or beet sugar, evaporated cane juice, brown rice syrup, agave syrup, and honey.
- Don't worry about the naturally occurring sugar in fruit, milk, and plain yogurt.
- If a food has little or no milk or fruit (which contain natural sugars), the "Sugars" number on the package's Nutrition Facts panel will tell you how many grams of added sugars are in each serving. Multiply the grams by 4 to get calories from sugar. Divide the grams by 4 to get teaspoons of sugar.

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Visit www.nutritionaction.com for more great advice about sugar in food!

com-sf-1

What labs do you look at?

- Creatinine
- GFR
- BUN
- Albumin
- Albuminuria or proteinuria
- Lytes
- Bone: Ca, P, PTH
- Lipids
- A1c
- Anemia: Hgb, Hct, Tsat, CBC
- Uric acid

How do your prioritize your interventions?

- Hyper/hypokalemia
- Na intake – BP control
- Protein intake: vegetable vs animal
- Glycemic control
- Weight management
- Medication compliance
- Dietary supplements
- Patient readiness

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Putting it Together

Guidelines = Cookbook

Effective **MNT** transforms Recipes
to **DIET** (a way of life)

Are you a COOK or a CHEF?

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Questions???

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RESOURCES

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CKD & Nutrition

The purpose of medical nutrition therapy (MNT) for chronic kidney disease (CKD) is to maintain good nutritional status, slow progression, and to treat complications.

The key diet components to slowing progression of CKD are:

- Controlling blood pressure by reducing sodium intake
- Reducing protein intake, if excessive
- Managing diabetes

CKD & NUTRITION

Overview

CKD Nutrition Management Training Program

CKD and Nutrition for Dietetic Educators

NKF-HAWAII

National Kidney Foundation of Hawaii

1634 South King Street

Phone: 593-1515

http://www.kidneyhi.org/

Patient education/support

Health screenings for CKD

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HMSA Well-Being Connection

Diet counseling by phone – no referral needed

1-855-440-7149

Diana Franklin, RD

Lisa Morita, RD

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