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VETERINARY MEETING & EXPO

AKI to CKD and everything in between: getting to grips with confusing renal diagnostics

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VETERINARY MEETING & EXPO

Learning objectives

By the end of this presentation, participants should be able to:

1. Compare and contrast the significance of kidney injury vs. chronic kidney function
2. Identify biomarkers of acute and active kidney injury, including urinary cystatin B
3. Understand the cellular sources for cystatin B and the significance of elevated urinary concentrations
4. Apply acute injury concepts to clinical cases

Timeline of renal biomarkers

400 B.C.

Proteinuria

Described by Hippocrates

1886

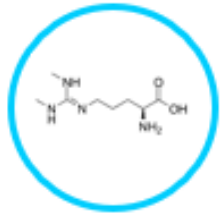
Creatinine

Jaffe reaction

1973

Glomerular filtration rate

Cockcroft-Gault equation for estimating GFR

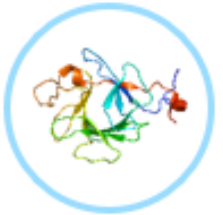


2015

IDEXX SDMA testing

Earlier biomarker of GFR

Launched by IDEXX



2022

IDEXX FGF-23 Test

Early biomarker of CKD

Launched by IDEXX for felines

December 2023

IDEXX Cystatin B Test

Detects both active and acute kidney injury

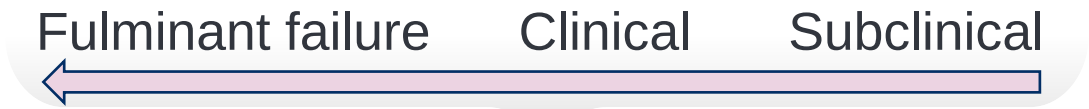
Launched by IDEXX

Centuries

Decades

Years

Terminology can be confusing; ARF, AKI, ATI, ATN



Acute Renal Failure

- Failure has specific definition of >90% loss of function with clinical effects
- Was a term often used to characterize significant loss of kidney function
  - Retention of uremic toxins
  - Dysregulation of fluid
  - Electrolyte and acid-base imbalance

Acute Kidney Injury

- Acute → short time span, generally ≤ 24-48 hours
- Impact disrupts renal cellular structure and function
- Injury does not necessarily = failure, or even azotemia
- Damage can be reversible

Acute tubular injury (ATI) or necrosis (ATN) is the most common cause of AKI in dogs and cats

Active Kidney Injury

- Often cannot characterize the timeline
- Inciting cause and time of onset are not always known
- Diagnostics indicate ongoing and sustained injury
- Distinguish **active kidney injury** from **kidney dysfunction**

J. Himmelfarb, T.A. Ikizler, Acute kidney injury: changing lexicography, definitions, and epidemiology, Kidney International,Volume 71, Issue 10,2007,Pages 971-976,ISSN 0085-2538, https://doi.org/10.1038/sj.ki.5002224.
Kellum, John A., Claudio Ronco, and Rinaldo Bellomo. "Conceptual advances and evolving terminology in acute kidney disease." Nature Reviews Nephrology 17.7 (2021): 493-502.

Acute kidney injury (AKI) vs. chronic kidney disease (CKD): Why do we care?

AKI

- Early detection to prevent progression
- Institute supportive care and specific therapy when possible
- Determine resolution or progression
- Short-term financial and emotional investments are intense
- Prolonged hospitalization: associated with higher morbidity and mortality

CKD

- Early detection/intervention in attempt to slow progression
- Institute dietary therapy, supportive care
- Determine likelihood of rapid progression
- Long-term financial, emotional, and time commitments
- Usually outpatient therapy; when hospitalization is required, usually associated with low morbidity and mortality

Veterinary criteria – IRIS AKI grading

Table 1: IRIS AKI Grading Criteria

AKI Grade	Blood Creatinine	Clinical Description
Grade I	<1.6 mg/dl (<140 µmol/l)	Nonazotemic AKI: a. Documented AKI: (historical, clinical, laboratory, or imaging evidence of AKI, clinical oliguria/anuria, volume responsiveness‡) and/or b. Progressive nonazotemic increase in blood creatinine: ≥ 0.3 mg/dl (≥ 26.4 µmol/l) within 48 h c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade II	1.7 – 2.5 mg/dl (141 – 220 µmol/l)	Mild AKI: a. Documented AKI and static or progressive azotemia b. Progressive azotemic: increase in blood creatinine; ≥ 0.3 mg/dl ≥ 26.4 µmol/l) within 48 h),or volume responsiveness‡ c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade III	2.6 – 5.0 mg/dl (221 – 439µmol/l)	
Grade IV	5.1 – 10.0 mg/dl (440 – 880 µmol/l)	Moderate to Severe AKI: a. Documented AKI and increasing severities of azotemia and functional renal failure
Grade V	>10.0 mg/dl (>880 µmol/l)	

(‡Volume responsive is an increase in urine production to >1 ml/kg/h over 6 h; and/or decrease in serum creatinine to baseline over 48 h)

Risk
Nonazotemic

Injury
Mildly azotemic

Failure
Moderately to severely azotemic

IRIS International Renal Interest Society

http://www.iris-kidney.com/education/index.html

Subgrade

Each grade of AKI is further subgraded as:
1. Non oliguric (NO) or oligo-anuric (O)
2. Requiring renal replacement therapy (RRT)

http://www.iris-kidney.com/guidelines/grading.html

Hallmarks of AKI (vs. CKD)

History and physical exam

+ Acute onset—hours to days
+ Toxin exposure (lily, grapes, NSAIDs, anesthetics…)
+ Renomegaly, renal pain
+ Lack of other PE change
+ Bradycardia (if severe hyperkalemia)
+ Hypothermia

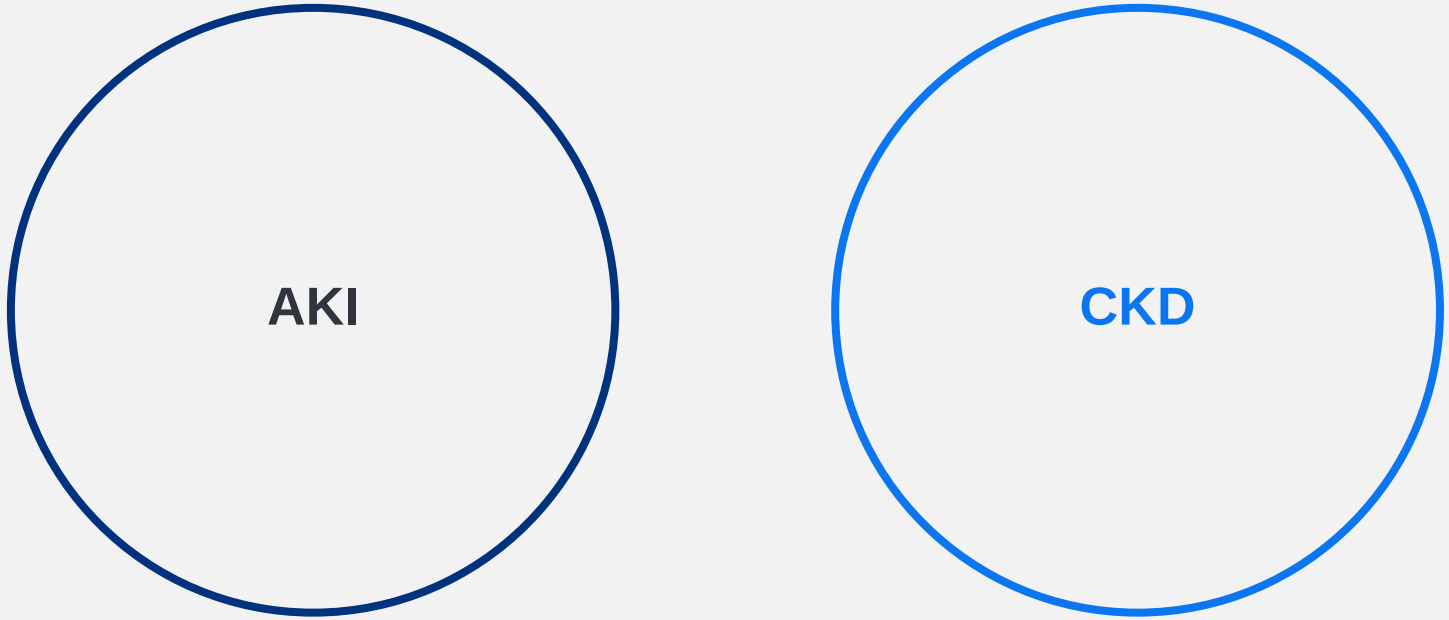
Lab findings

+ Hyperkalemia
+ Urinary granular casts, normoglycemic glucosuria

Imaging

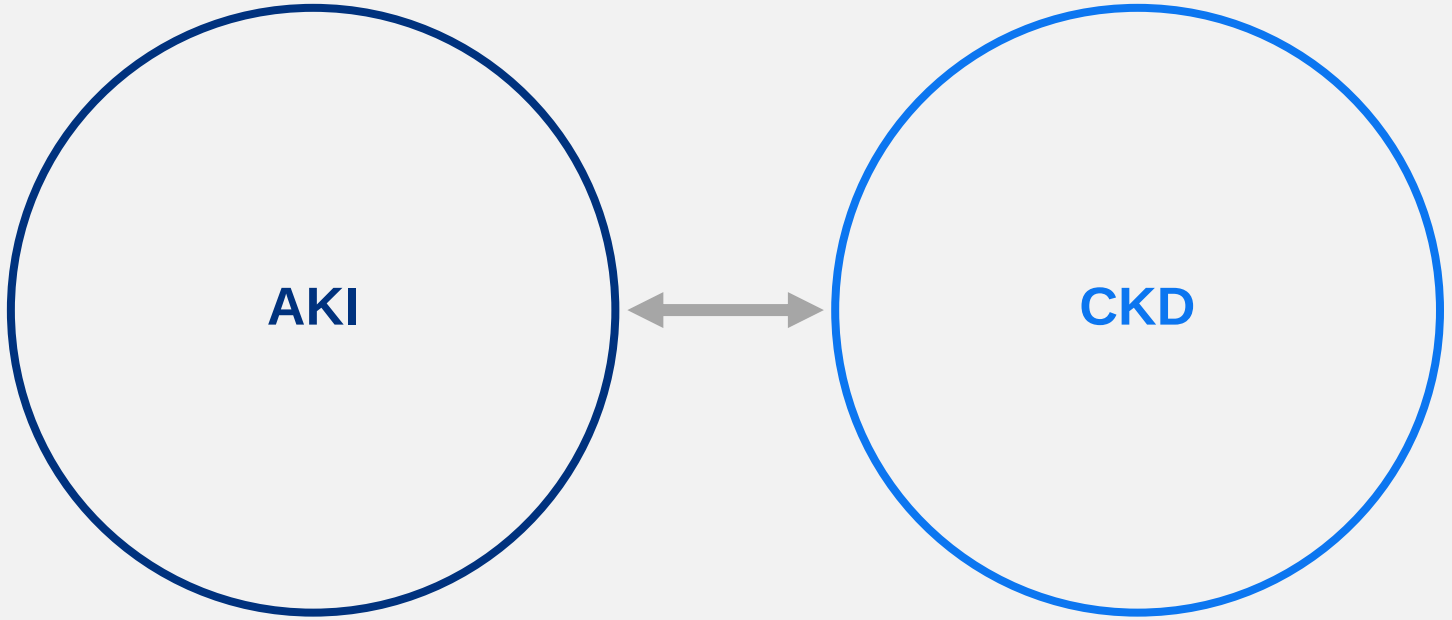
+ Renomegaly in 70%
+ Hydroureter, pyelectasia, hydronephrosis
+ Ureteral calculi
+ Normal parathyroid gland

Back in the day…



More contemporary view…

Your AKI patient may have or develop CKD

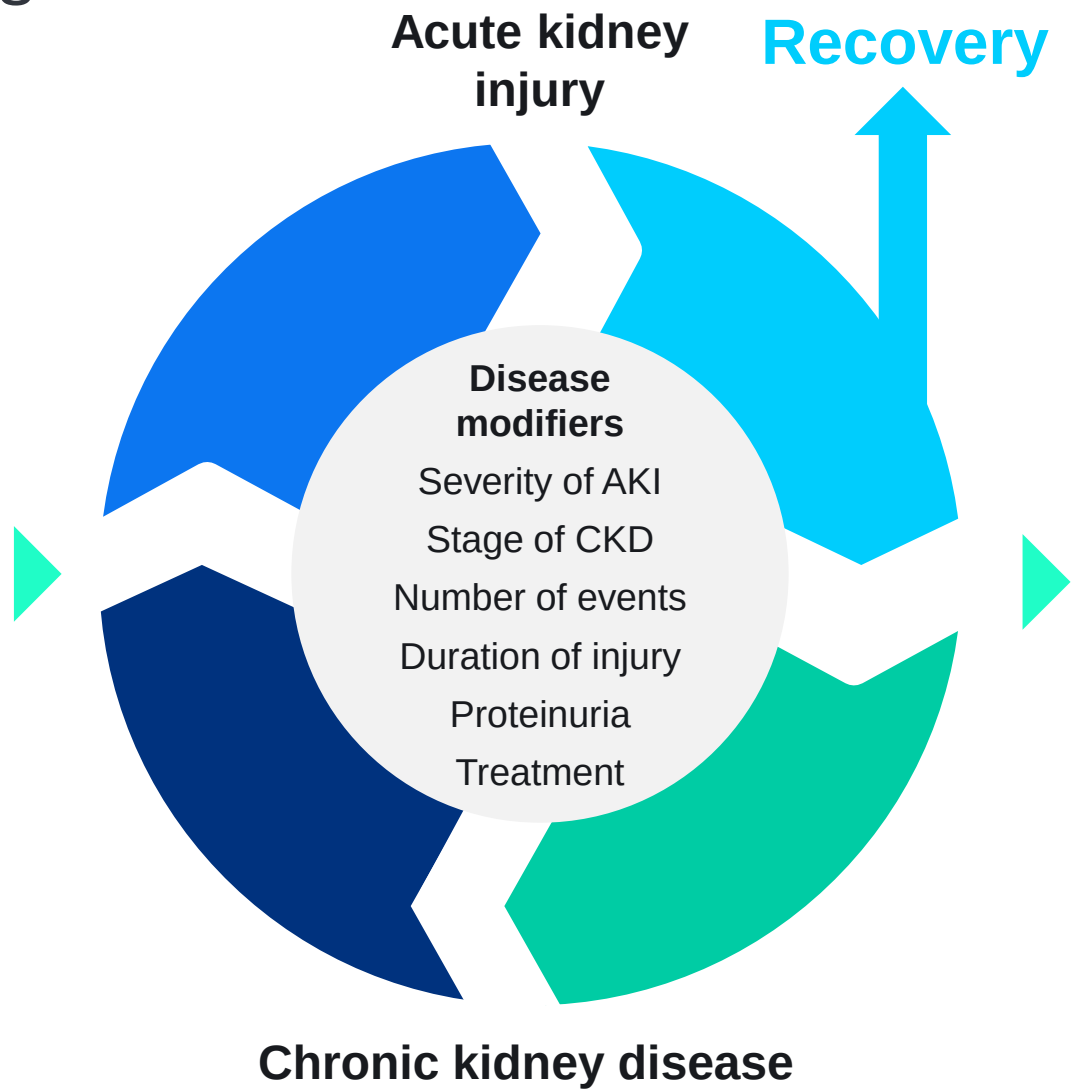


Your CKD patient may have concurrent active kidney injury

Kidney function in health and disease is impacted by risk factors, injury, and outcomes

Risk factors

+ Breed
+ Age
+ Sex
+ Diet
+ Drugs
+ Pre-existing disease
  + CKD
  + Hypertension
  + Metabolic disease
  + Cardiac disease

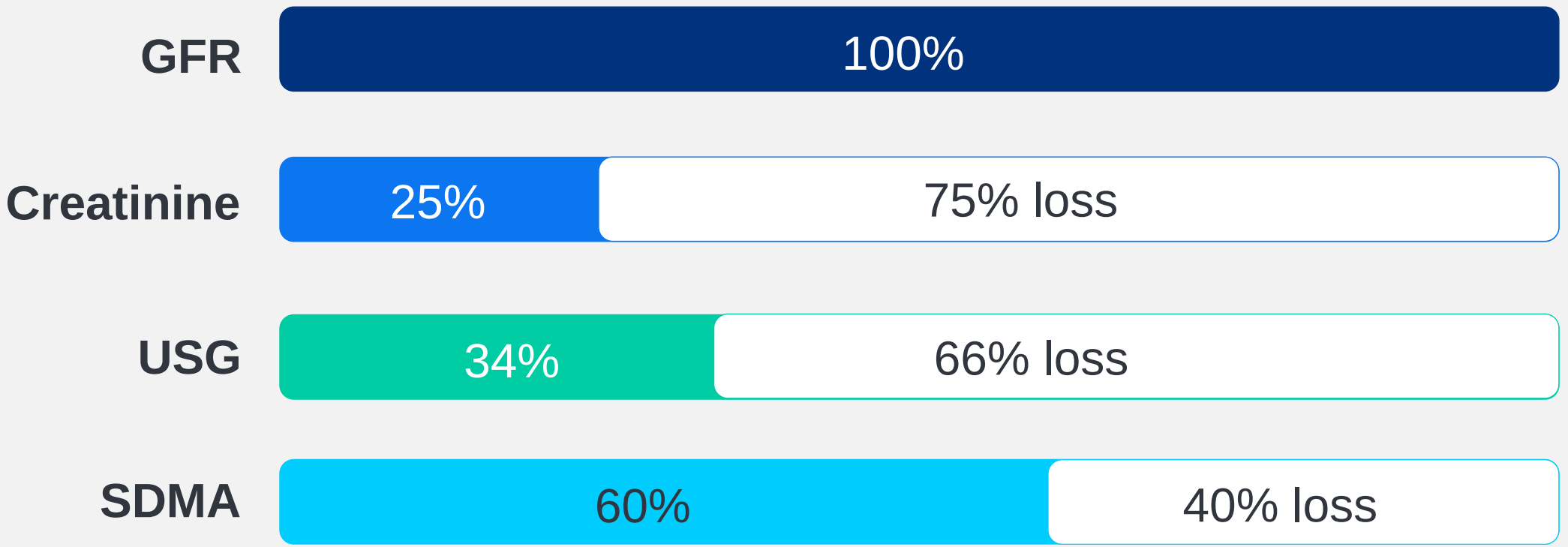


Potential outcomes

+ Persistent damage
+ Cardiovascular events
+ Additional kidney events
+ Diminished quality and quantity of life
+ Cost events

Source: Chawla LS, Eggers PW, Star RA, Kimmel PL. Acute kidney injury and chronic kidney disease as interconnected syndromes. N Engl J Med. 2014;371(1):58–66. doi:10.1056/NEJMra1214243

Performance of current renal “functional” biomarkers (estimates of GFR)



GFR biomarkers fall short as early detectors of kidney disease

Categorization of biomarkers and analytes used to evaluate kidney function and injury

Indirect markers of function	Urine-based markers	Other important analytes	Acute kidney injury markers
Most specific (limited extrarenal impact): + SDMA + Creatinine **Less specific (more extrarenal impact):** + BUN + Phosphorus	**Urinalysis** + Physical + Chemical + Sediment **UPC**	+ Potassium + Sodium/chloride + Calcium + Albumin/TP + Hematocrit + FGF-23	+ Cystatin B + Urine Clusterin + NGAL

You need broad assessment to understand kidney health

What can we measure in clinical practice?

Glomerular function

How well are the kidneys clearing waste from the body (GFR)

Creatinine, SDMA, BUN

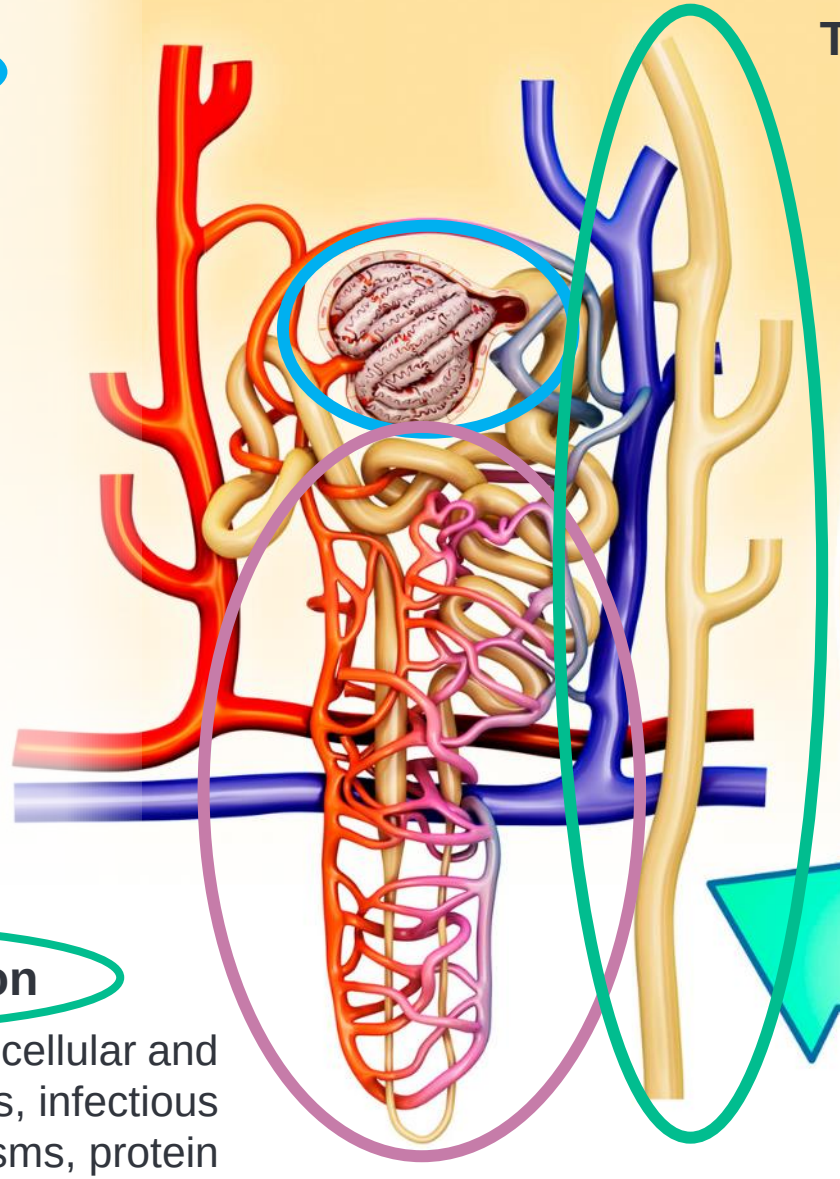
Tubular function

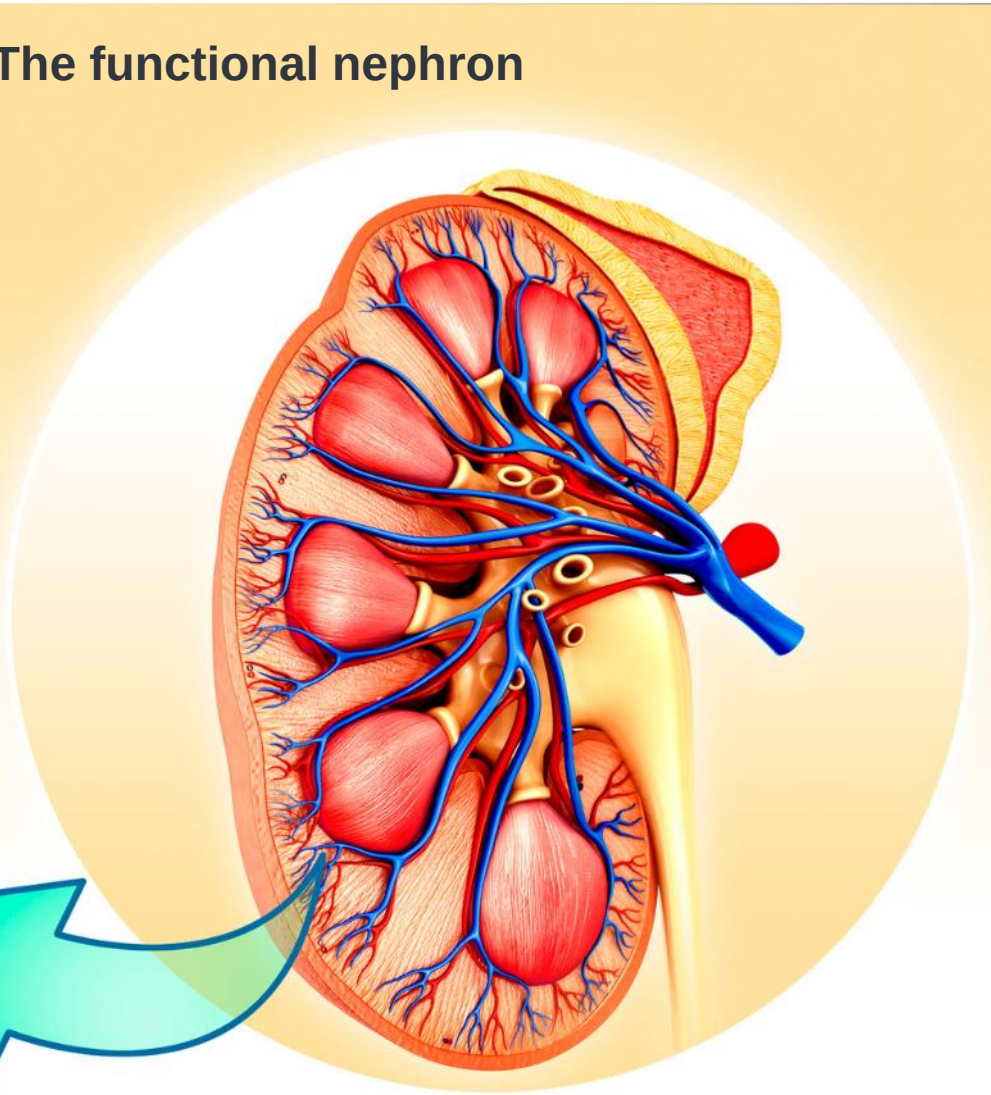
Important in solute and water management

Urine concentration and protein; serum and urine electrolytes, glucose, acid-base

Urine composition

Concentration/volume, pH, cellular and crystalline elements, infectious organisms, protein

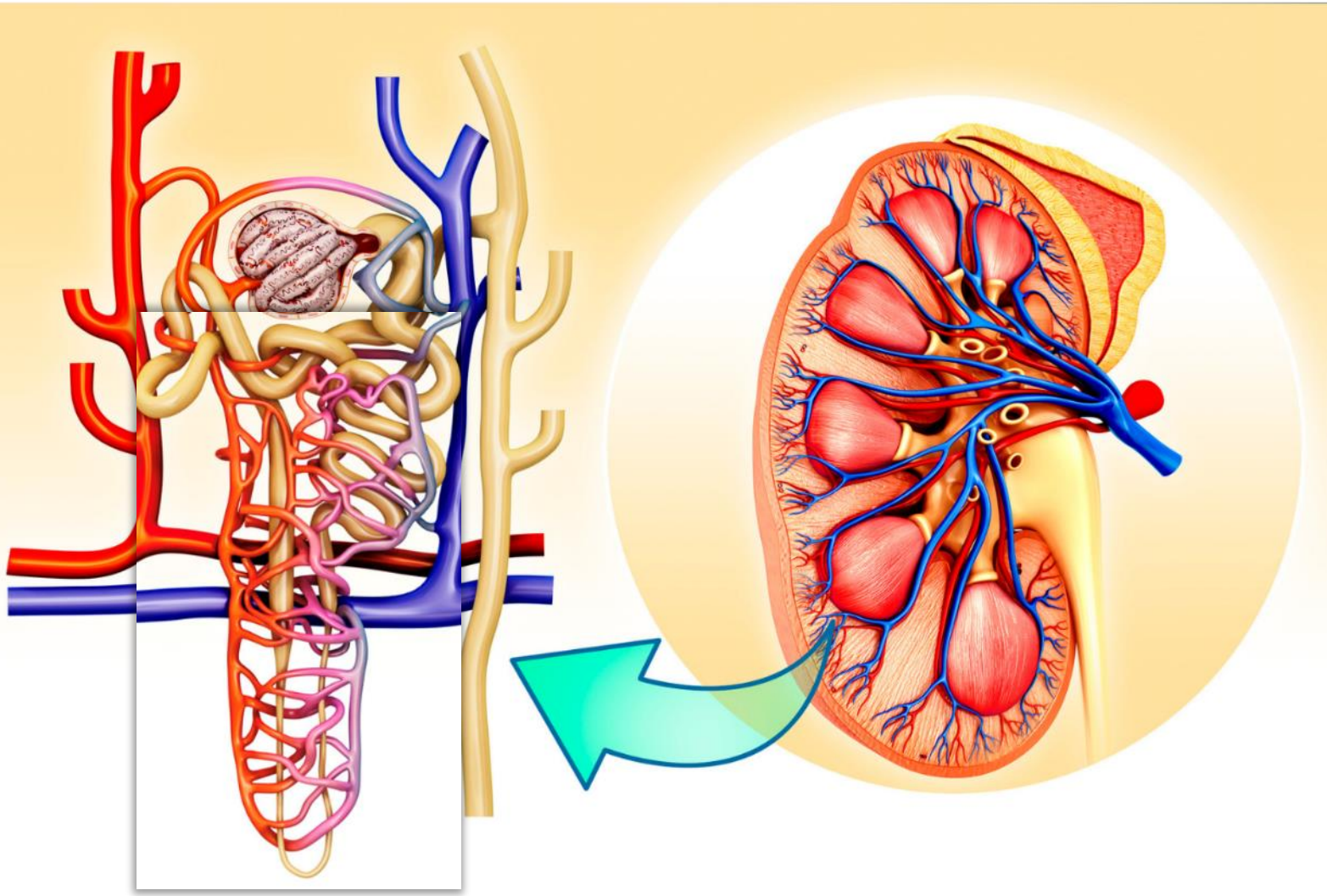




Renal tubules are where the action **really** is

Tubular function

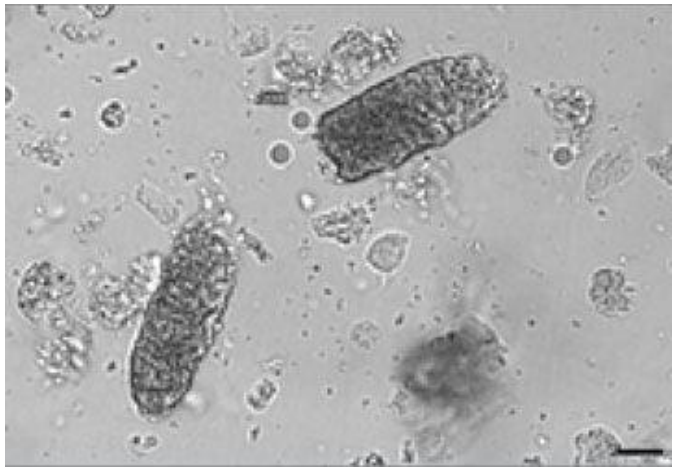
- The actual work of the kidney primarily takes place here. Filtering, reabsorbing, and secreting solutes and water
- Impact urine concentration and what is excreted
- Dysfunction can impact electrolytes, protein levels, glucose, acid-base balance
- Captured in chemistry panel and urinalysis

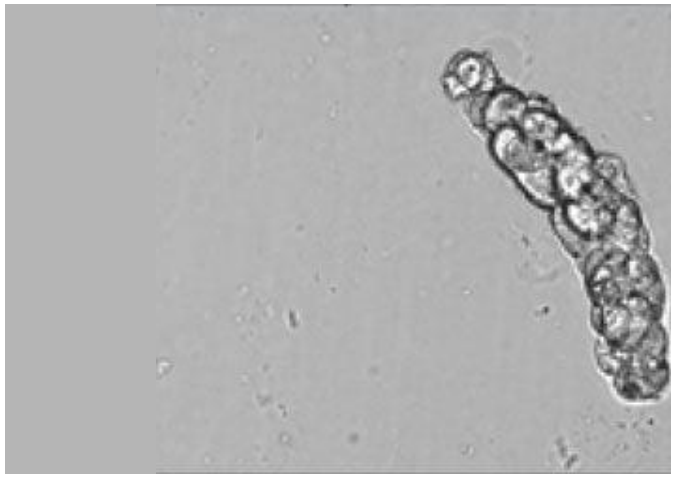


Traditional renal **injury** markers are good, not great

+ Proteinuria
+ Hematuria, pyuria
+ Bacteriuria
+ Renal epithelial cells in the urine
+ Glucosuria (normoglycemia)
+ Cylindruria (casts)
+ Decreased USG

Granular casts





Cellular cast

Source: IDEXX SediVue Dx® images

Functional markers are in blood

Take-home message:

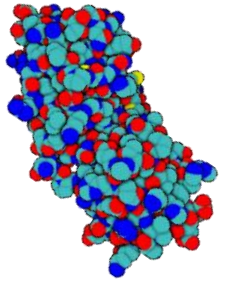
You can’t assess kidney health without urine

Can we do any better?

YES!!! We can!

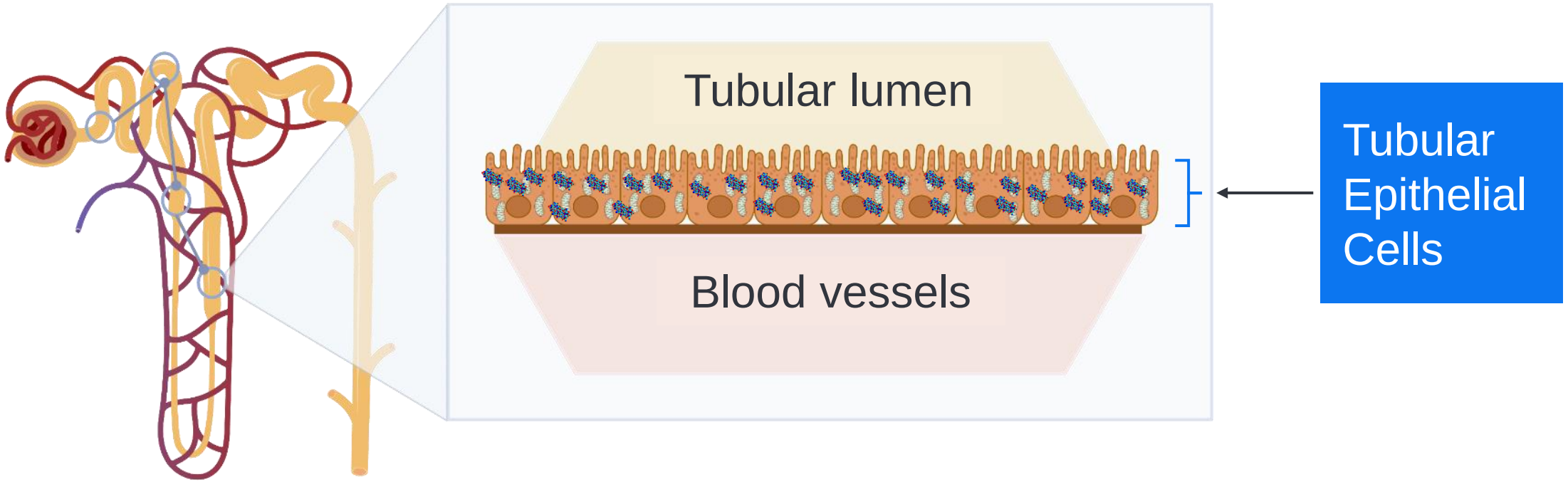
Cystatin B bridges the gap in our abilities to detect early and active renal injury

What is cystatin B?

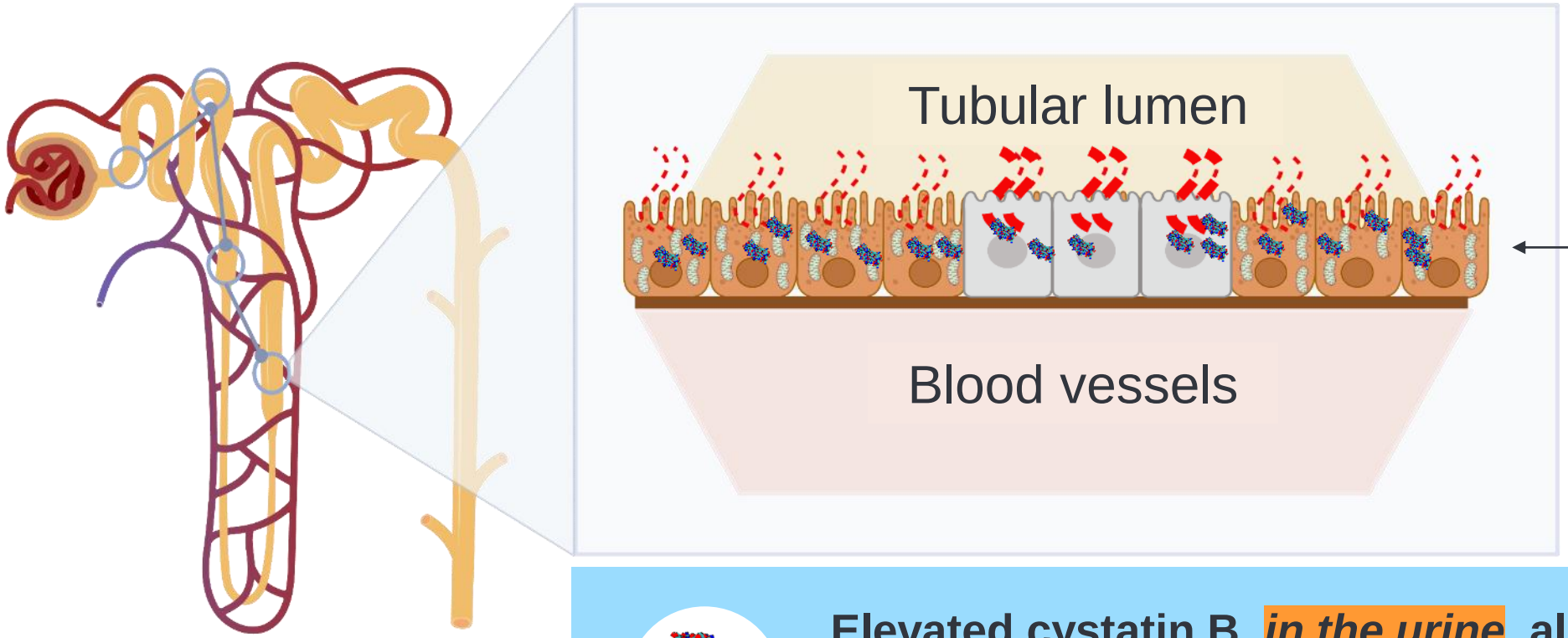


- Member of cystatin family
  - Protease inhibitors that help protect against leakage of proteolytic enzymes from lysosomes
  - Trace amounts in the serum of healthy subjects
- A small, intracellular protein
  - 11 kDa (11,000 daltons)
  - Ubiquitous in many cells, including proximal renal tubular cells
- Freely filtered at the glomerulus
- Increased urinary [cystatin B] indicates active, ongoing tubular injury
  - Think of it as the ALT of the kidney

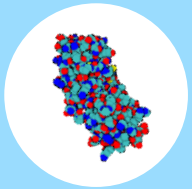
Cystatin B is a very small protein contained in epithelial cells of the renal tubules



During **active or acute kidney injury**, renal tubular epithelial cells can be damaged

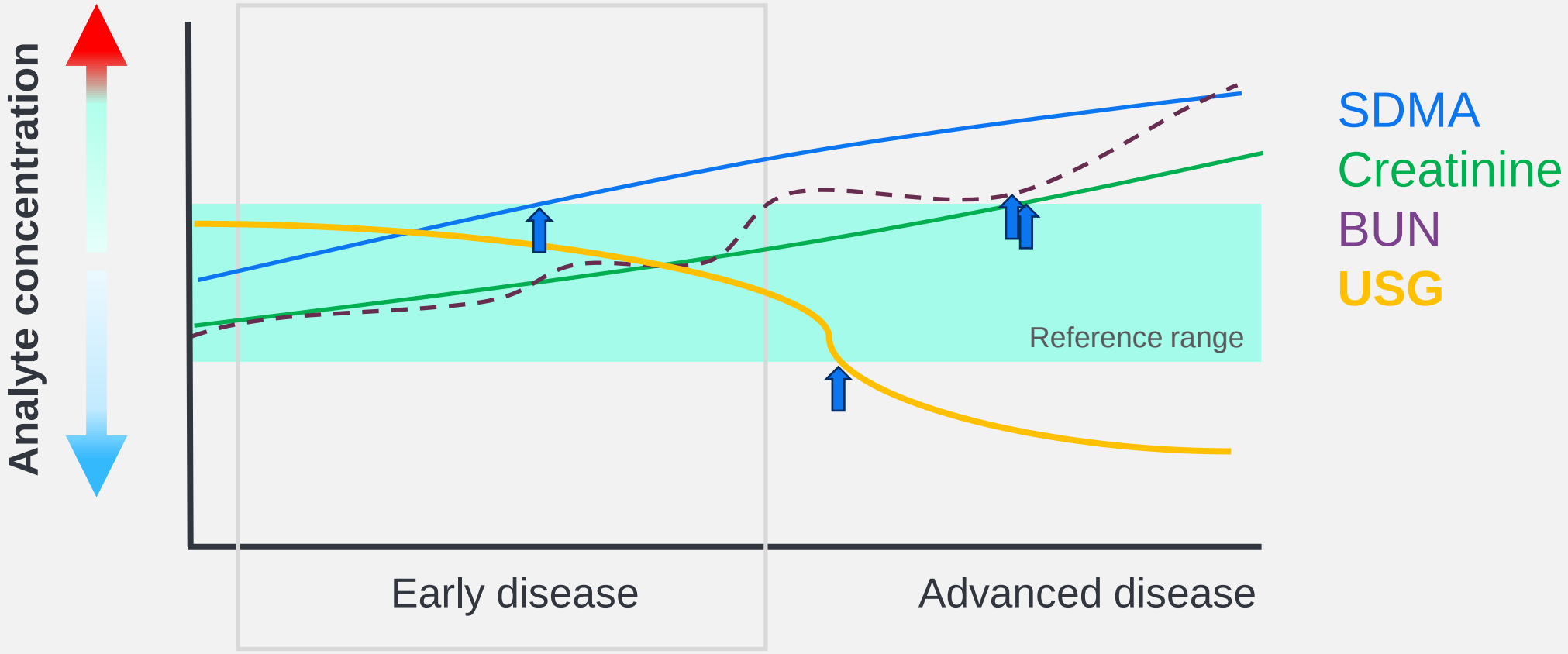


Cystatin B leakage from injured or necrosed tubular epithelial cells

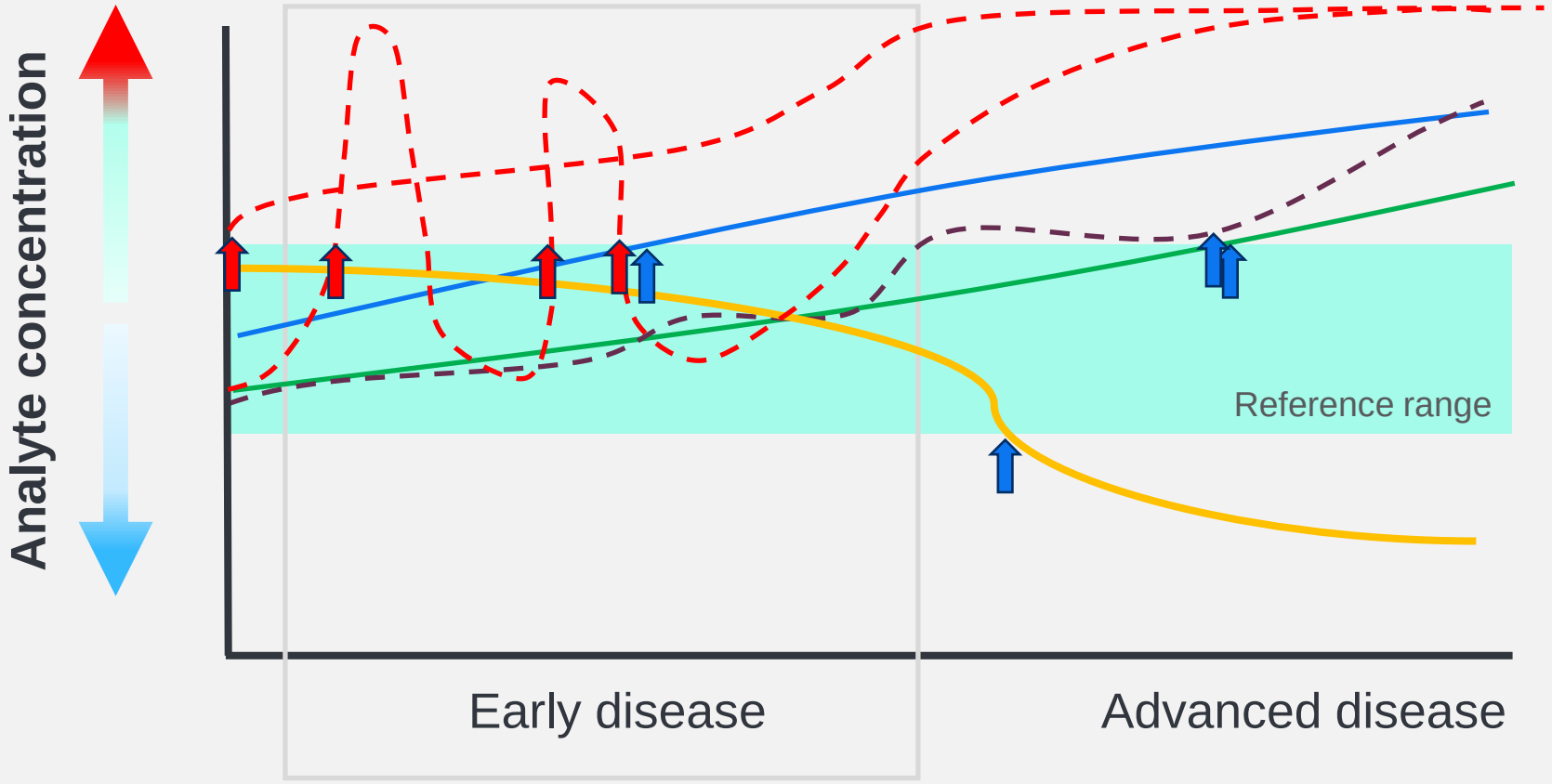


Elevated cystatin B, *in the urine*, alerts to earlier, ongoing, and unresolved kidney injury, and can occur with or without increases in functional markers,

Kidney injury markers are additive to current indirect functional markers

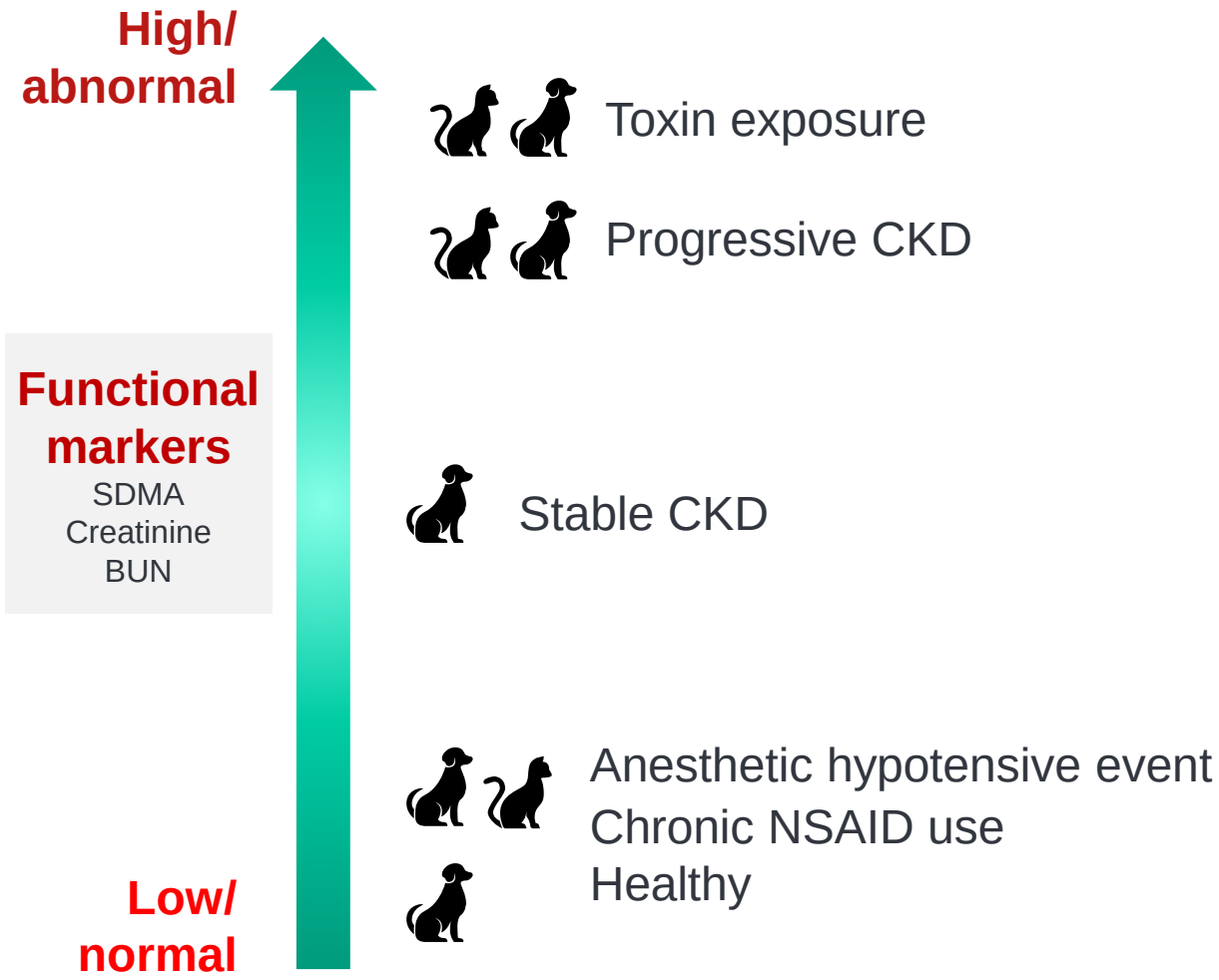


Kidney injury markers are additive to current indirect functional markers

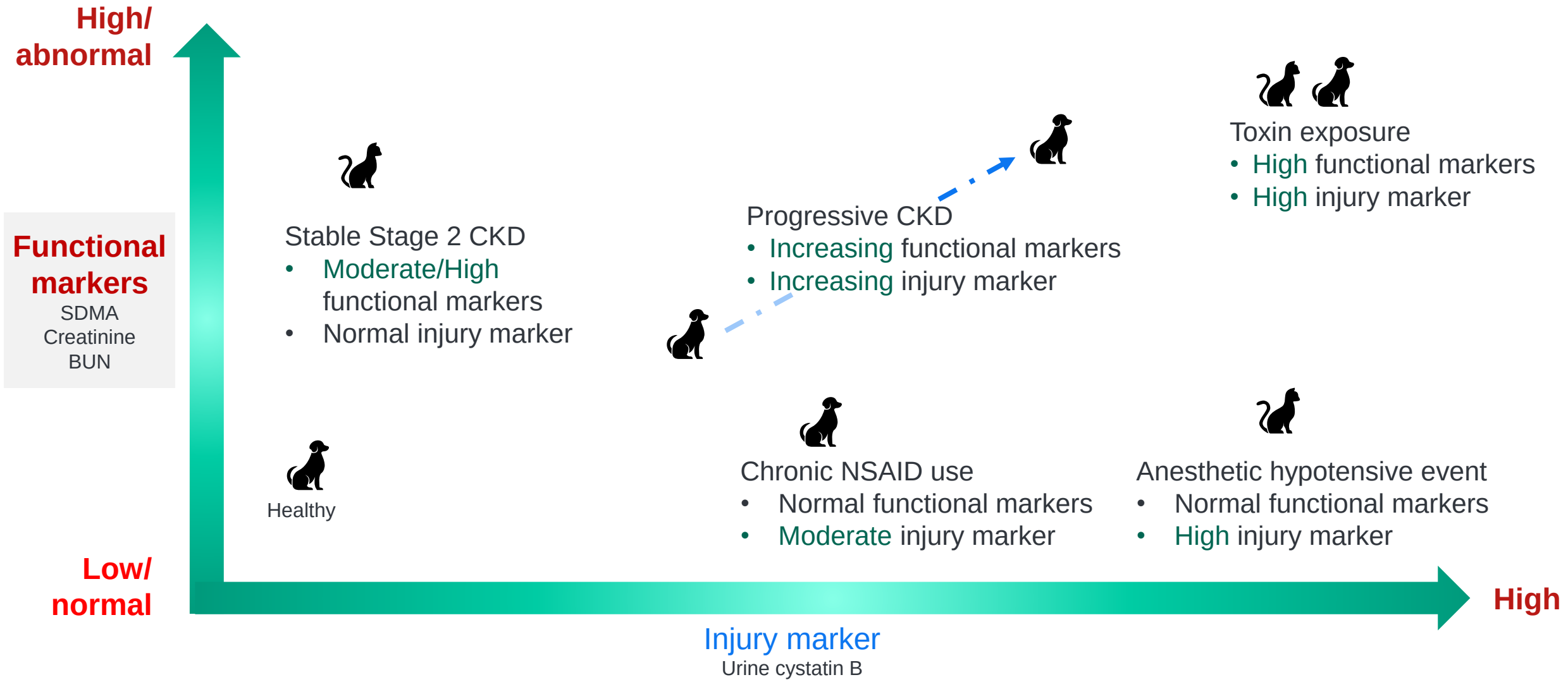


Renal tubular markers
SDMA
Creatinine
BUN
USG

Traditional diagnostics only allow for case evaluation by functional markers



Addition of an injury marker provides better case discrimination and management

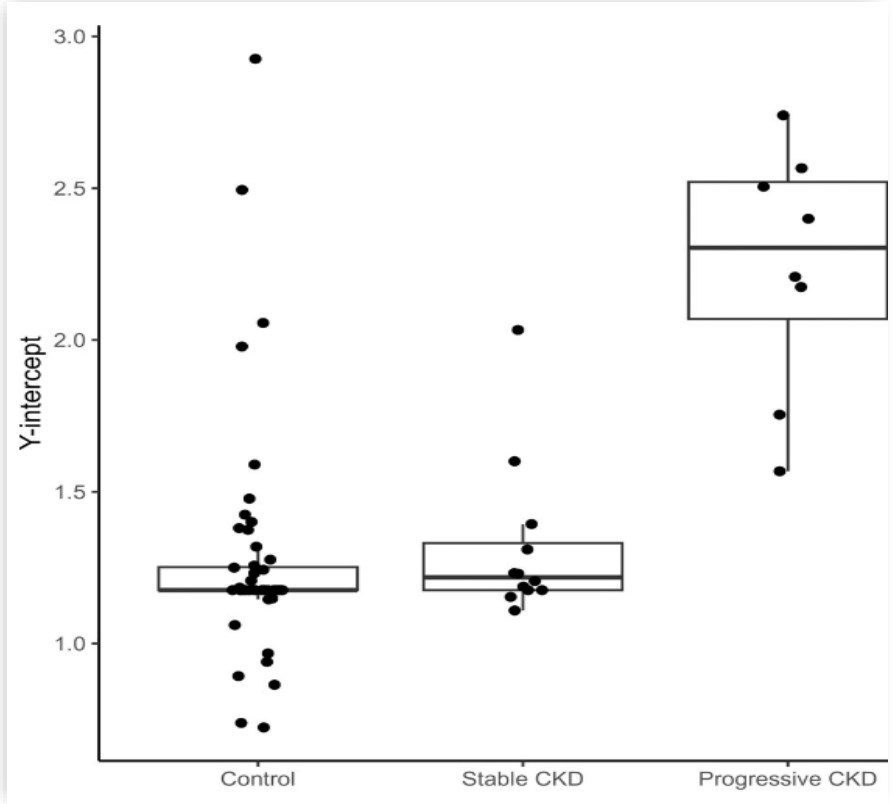


Therefore, markers of tubular injury are earlier indicators of damage than functional markers

By up to 2 days…

Cystatin B has value with evaluating patients with CKD as well!

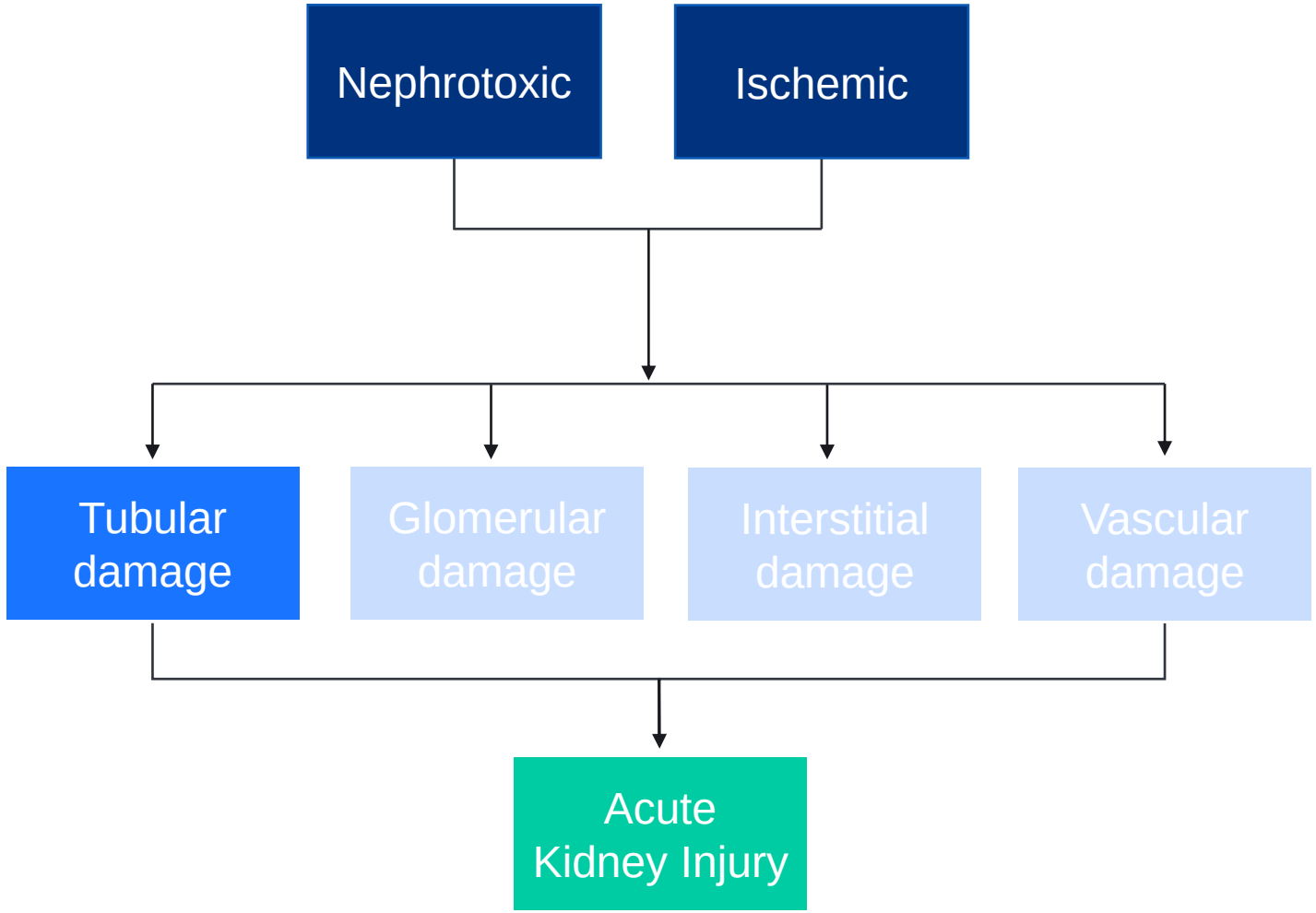
- CKD progressive and irreversible
- Rate of progression unpredictable
- Cystatin B identifies active, progressive injury in dogs with CKD
- Increased urinary cystatin B (uCysB) in dogs with IRIS Stage 1 CKD predicts rapid progression
- Identifies which dogs need more frequent monitoring



y-intercepts calculated from inverse urinary cystatin B (uCysB) vs. time

Segev, et al. J Vet Intern Med. 2023

Pathophysiology of AKI



Common etiologies:

+ Infectious diseases
+ Nephrotoxins
+ Systemic diseases with secondary renal involvement (inflammation)
+ Alterations in hemodynamics
+ Obstructive disorders

Causes of AKI include:

Cat

+ Toxins (plants, chemotherapeutics)
+ Pyelonephritis
+ Acute pancreatitis
+ Marked dehydration
+ Obstructive disorders
+ Etiology unknown ~30%

Dog

+ Toxins (plants, chemotherapeutics, foods)
+ Pyelonephritis
+ Acute pancreatitis
+ Marked dehydration
+ Obstructive disorders
+ Leptospirosis
+ Lyme nephritis
+ Congestive heart failure

AKI can develop in hospitalized patients: Monitor and grade daily

+ Dehydration
+ Age > very young or old
+ Diuretic or nephrotoxic drug therapy
+ Hypokalemia or hypercalcemia
+ Sepsis
+ Congestive heart failure
+ Acute pancreatitis
+ Systemic hypertension
+ CKD

Avoid iatrogenic AKI!
Nephrotoxic drugs
Hemodynamic instability
Fluid overload

Initial treatment of AKI

Initial therapy for AKI; back to the basics

- Address hydration and volemic status
- Institute disease-specific therapy whenever possible
- Address complications of AKI
- Avoid and monitor for complications of therapy
- Keep the patient comfortable
- Prevent further renal injury
- Address nutrition

Use serial evaluations of IRIS AKI grading for objective measure of response to therapy; adapt/adjust accordingly

Fluid therapy for kidney disease: Less may be more

- Fluids are drugs—avoid overdose
- Fluids do not improve kidney function
- Hypervolemia causes AKI and kills patients that already have it
- Not every patient with kidney disease (acute or chronic) needs fluids!!!

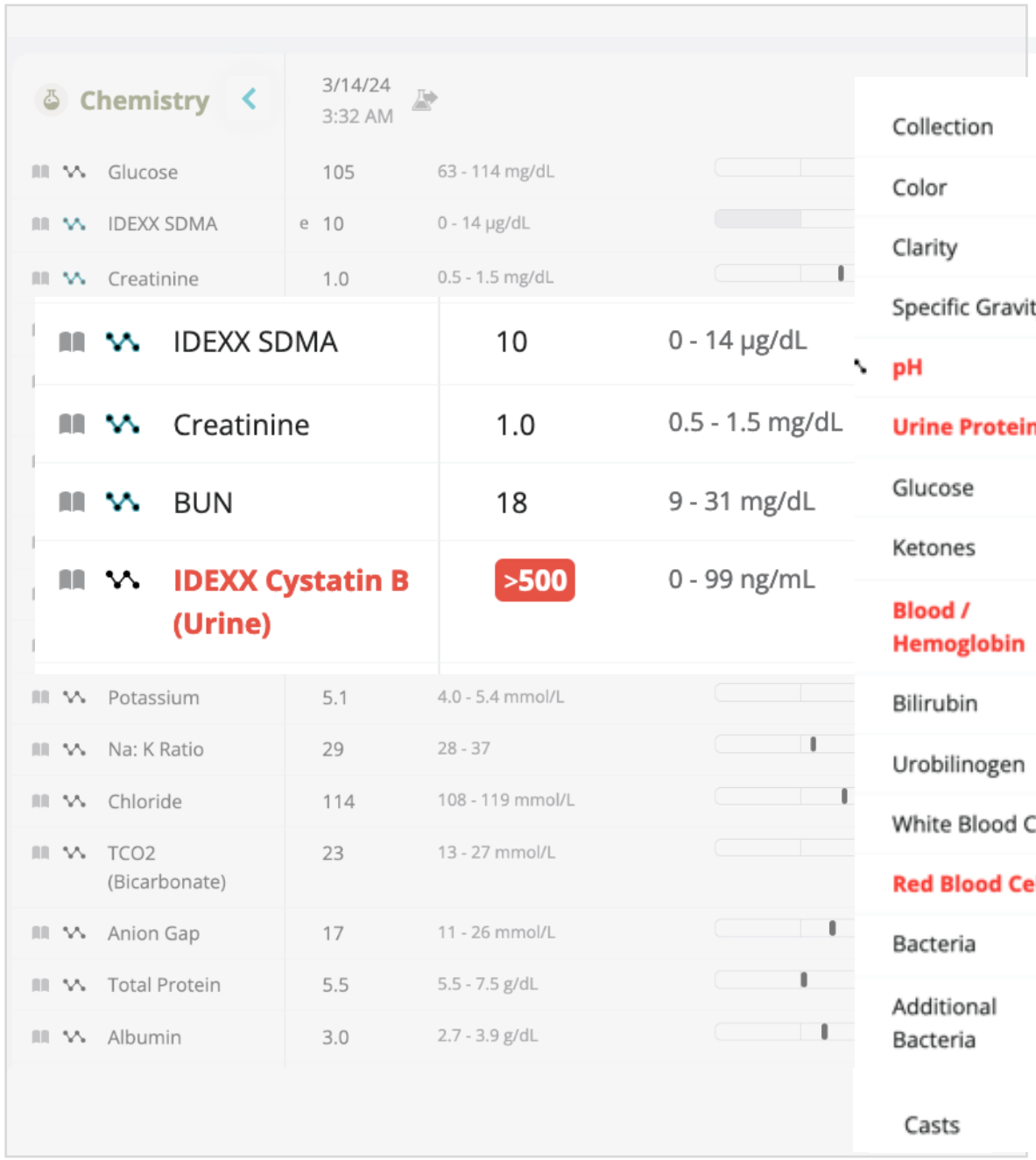
Assessment of fluid therapy success is essential

- Perfusion parameters: HR, CRT, mucous membranes, pulses, lactate, base excess
- Body weight 2-4x/day: >5%–10% increase slow or stop fluids
- Lung auscultation: ≥ q12 hrs, more frequently if any changes in RR/RE

If azotemia worsens with IV fluid therapy, consider **decreasing** fluid rate.

Especially if total daily volume exceeds maintenance or if weight gain.

My dog ate some raisins.



Rover

+ 3-year-old MC MixB
+ Confirmed raisin ingestion
+ Amount/time prior to presentation uncertain
+ Previously healthy

Elevated urinary cystatin B, proteinuria, cylindruria

Three days later, after IV fluids for 48 hours

Chemistry

3/17/24 1:07 AM

IDEXX SDMA	8	0 - 14 µg/dL
Creatinine	1.2	0.5 - 1.5 mg/dL
BUN	26	9 - 31 mg/dL
IDEXX Cystatin B (Urine)	<50	0 - 99 ng/mL
Ratio		
Phosphorus	5.1	2.5 - 6.1 mg/dL
Calcium	9.5	8.4 - 11.8 mg/dL
Sodium	148	142 - 152 mmol/L
Potassium	5.1	4.0 - 5.4 mmol/L
Na: K Ratio	29	28 - 37
Chloride	114	108 - 119 mmol/L
TCO2 (Bicarbonate)	25	13 - 27 mmol/L
Anion Gap	14	11 - 26 mmol/L
Total Protein	**5.1**	5.5 - 7.5 g/dL
Albumin	2.7	2.7 - 3.9 g/dL
Globulin	2.4	2.4 - 4.0 g/dL

Urinalysis

3/17/24 1:07 AM

Collection	FREECATCH
Glucose	NEGATIVE
Ketones	a RACE
Blood / Hemoglobin	**3+**
Bilirubin	1+
Urobilinogen	NORMAL
White Blood Cells	0-2
Red Blood Cells	**30-50**
Bacteria	NONE SEEN
Additional Bacteria	
Epithelial Cells	1+ (1-2)/HPF
Mucus	NONE SEEN
Casts	NONE SEEN
Crystals	NONE SEEN

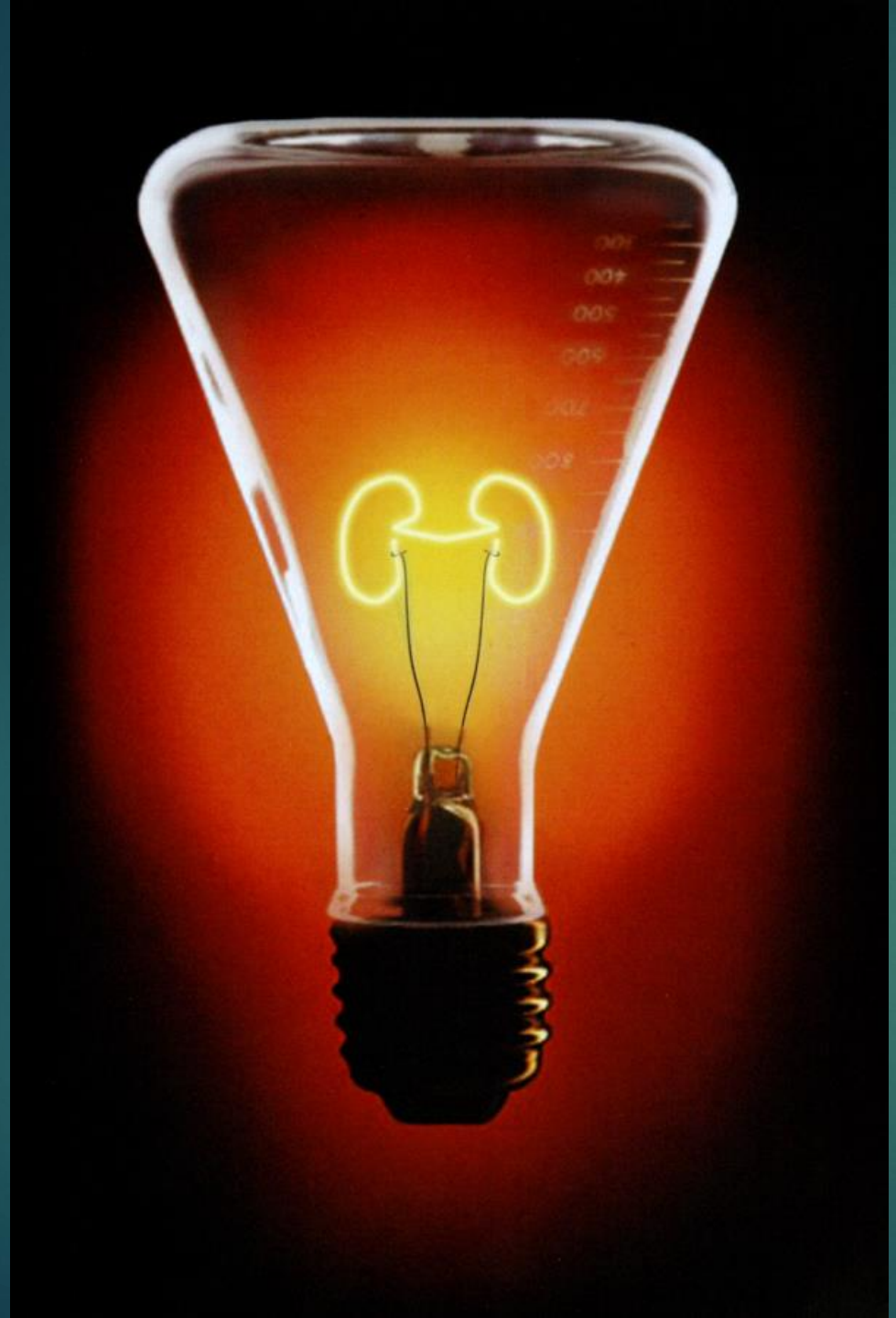
J Vet Diagn Invest 17:223–231 (2005)

Canine renal pathology associated with grape or raisin ingestion: 10 cases

All dogs had degeneration or necrosis (or both) of proximal renal tubules with basement membranes remaining intact, and epithelial regeneration was observed in 5 out of 10 cases.

Take-home

- Acute kidney injury and chronic kidney disease are a continuum.
- A COMPLETE urinalysis is of UTMOST importance when evaluating kidney (as well as systemic) disorders.
- IDEALLY, patients at risk for renal injury (IRIS AKI grade I) are identified and managed BEFORE azotemia develops.
- Fluid therapy paradigms have changed…dramatically.
- Cystatin B, a *urine* biomarker, is a marker of ACTIVE renal tubular injury.



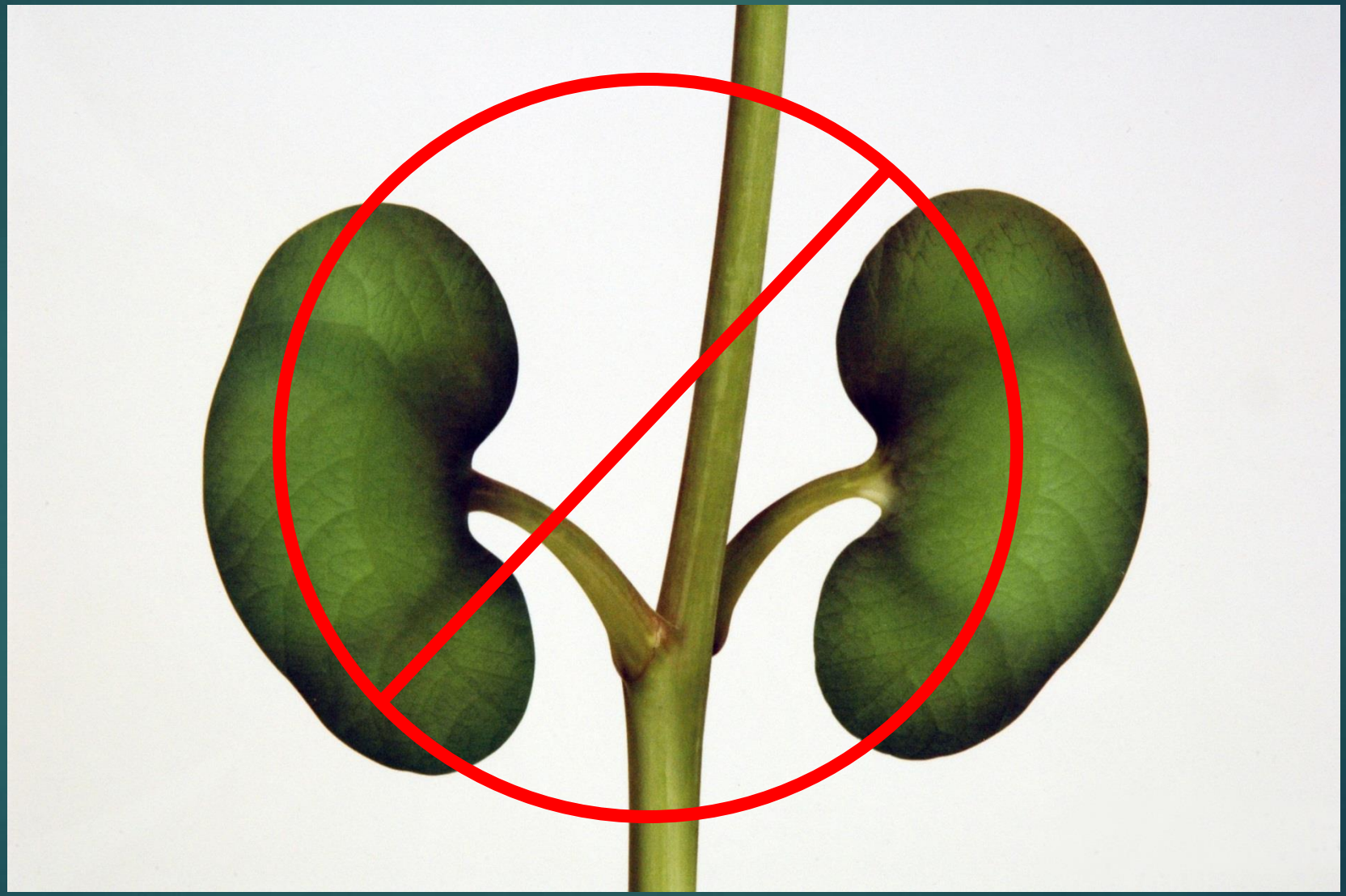
New Tests for Improved Early Diagnosis and Management of CKD

Gregory F. Grauer, DVM, MS
Diplomate, ACVIM (SAIM)
Founding Member ACVNU
Professor Emeritus
Kansas State University

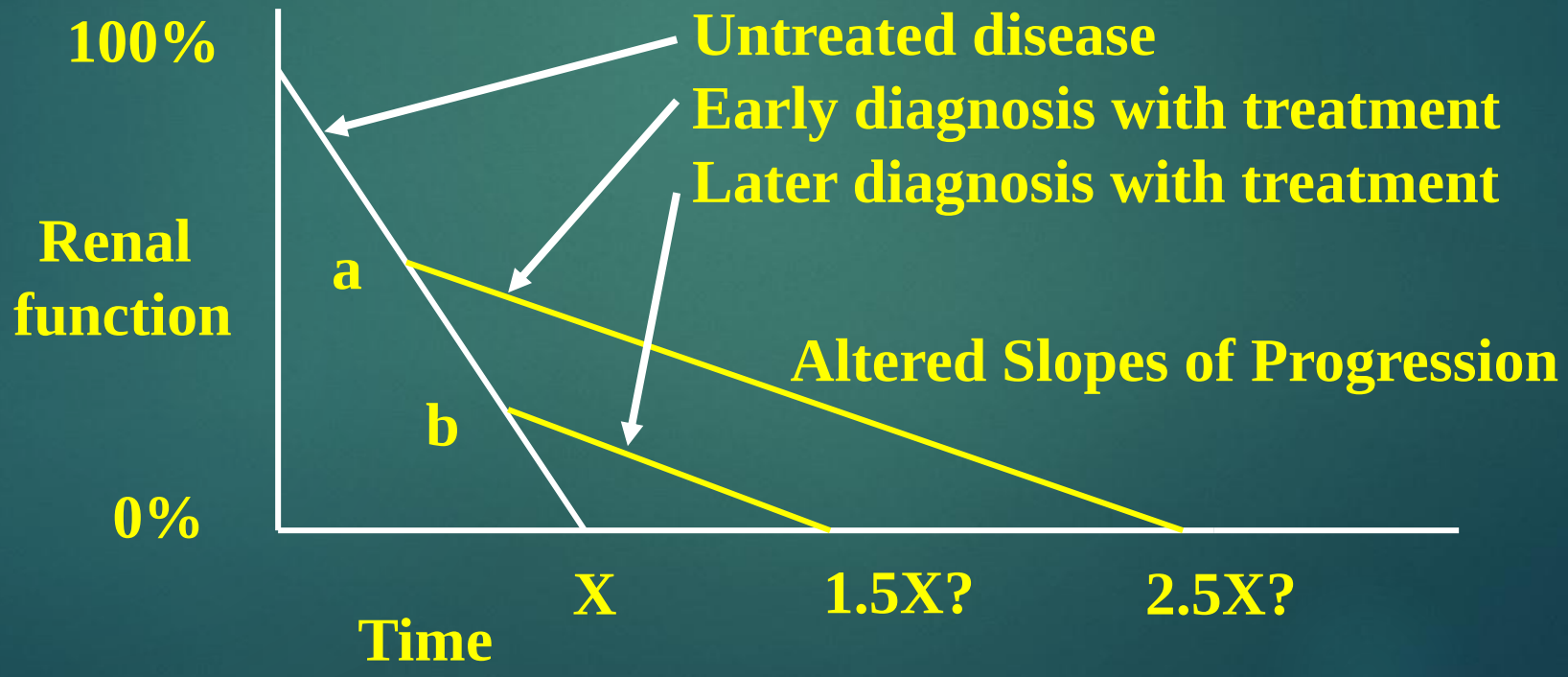
CKD Diagnosis and Treatment: (Begin with the end in sight)

- Closer monitoring of SrCr and SDMA will facilitate early diagnosis of CKD
  - Longitudinal assessment of these parameters when available will provide better data than one-time evaluations
  - Monitoring serum SDMA concentrations will improve early diagnosis of CKD (2x more sensitive than SrCr)
- Use of FGF-23 will improve management of Phosphate imbalance/Phosphate overload
- Think “Inside the Reference Interval”

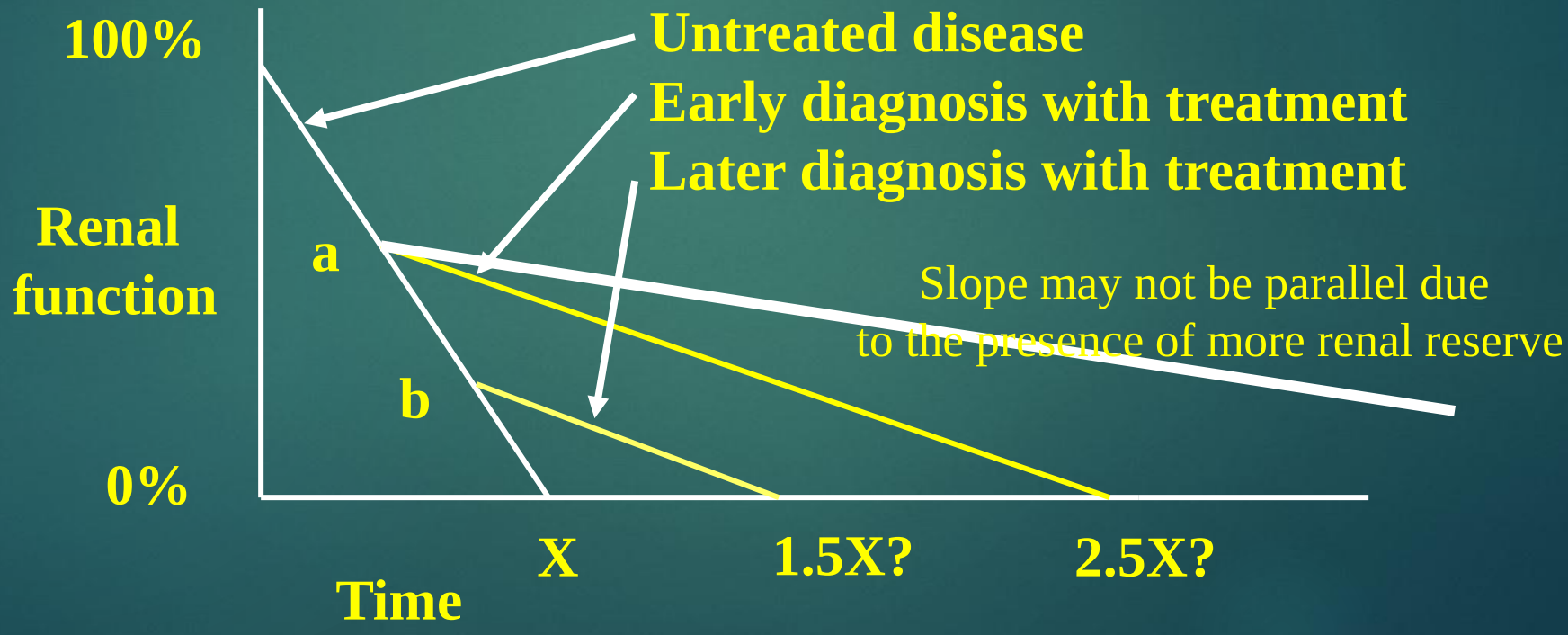
CKD is Irreversible and Often Progressive: No New Nephrons Can Be Produced



Potential effects of early detection and treatment of CKD



Potential effects of early detection and treatment of CKD



IRIS* Classification of Canine and Feline CKD

Creatinine SDMA**	Stage 1 Non-azotemic CKD	Stage 2 Normal to mild renal azotemia	Stage 3 Moderate renal azotemia	Stage 4 Severe renal azotemia
Creatinine Dogs/Cats (mg/dL)	<1.4/<1.6	1.4-2.8/1.6-2.8	2.9-5.0	>5.0
SDMA Dogs/Cats (µg/dL)	<18/<18	18-35/18-25	36-54/26-38	>54/>38
Prevalence in Cats	33.3 %	37.2%	15.4 %	14.1 %

Further sub-classify based on the presence or absence of proteinuria and systemic hypertension. **www.IRIS-kidney.com*

***In the case of discrepancy between Creatinine and SDMA, consider body condition, and re-testing in 2-4 weeks. If values are persistent, consider assigning the patient to the higher stage.*

IRIS* Classification of Canine and Feline CKD

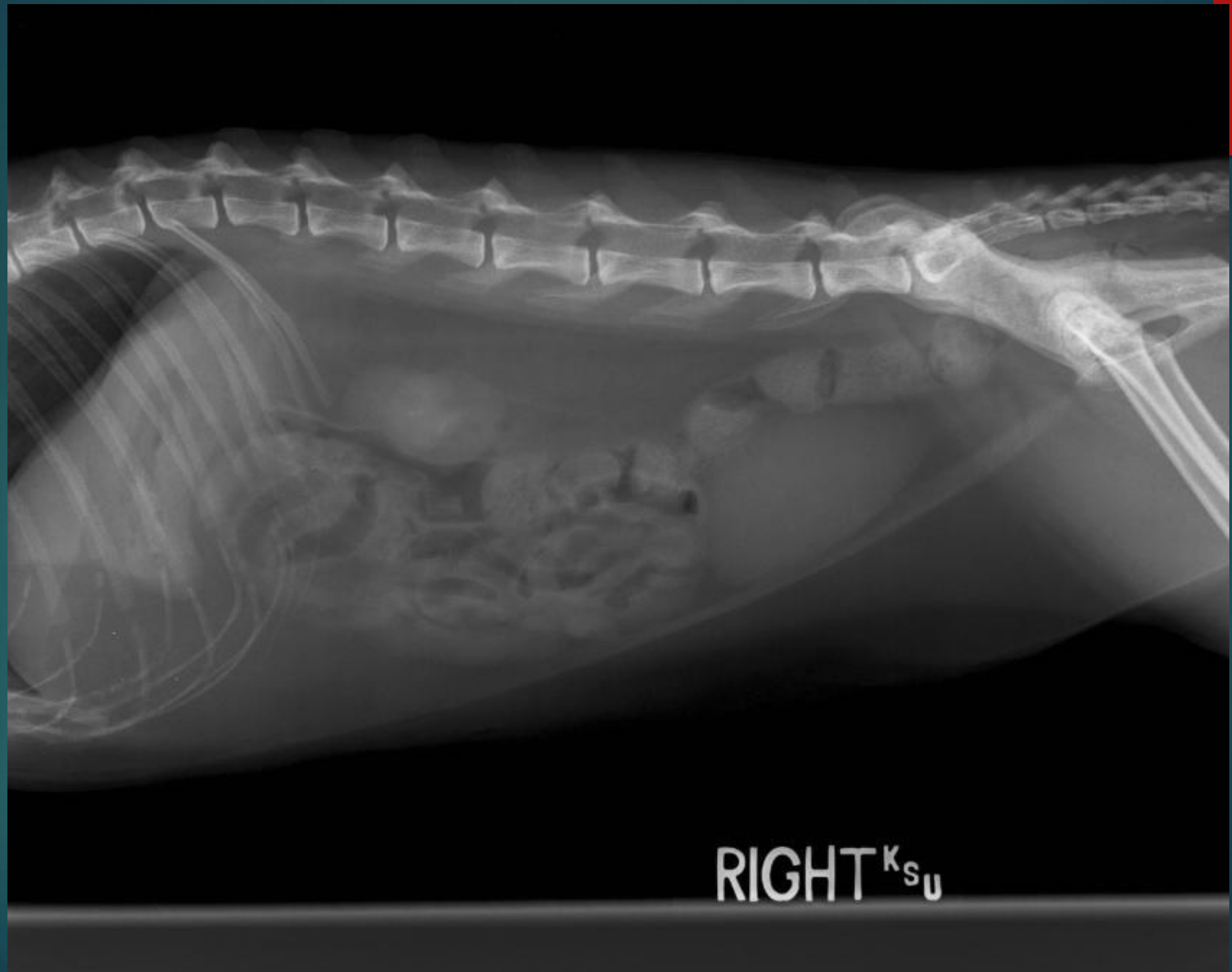
Creatinine SDMA**	Stage 1 Non-azotemic CKD	Stage 2 Normal to mild renal azotemia	Stage 3 Moderate renal azotemia	Stage 4 Severe renal azotemia
Dogs/Cats (mg/dL)	<1.4/<1.6	1.4-2.8/1.6-2.8	2.9-5.0	>5.0
Dogs/Cats (µg/dL)	<18/<18	18-35/18-25	36-54/26-38	>54/>38
Prevalence in Cats	33.3 %	37.2%	15.4 %	14.1 %

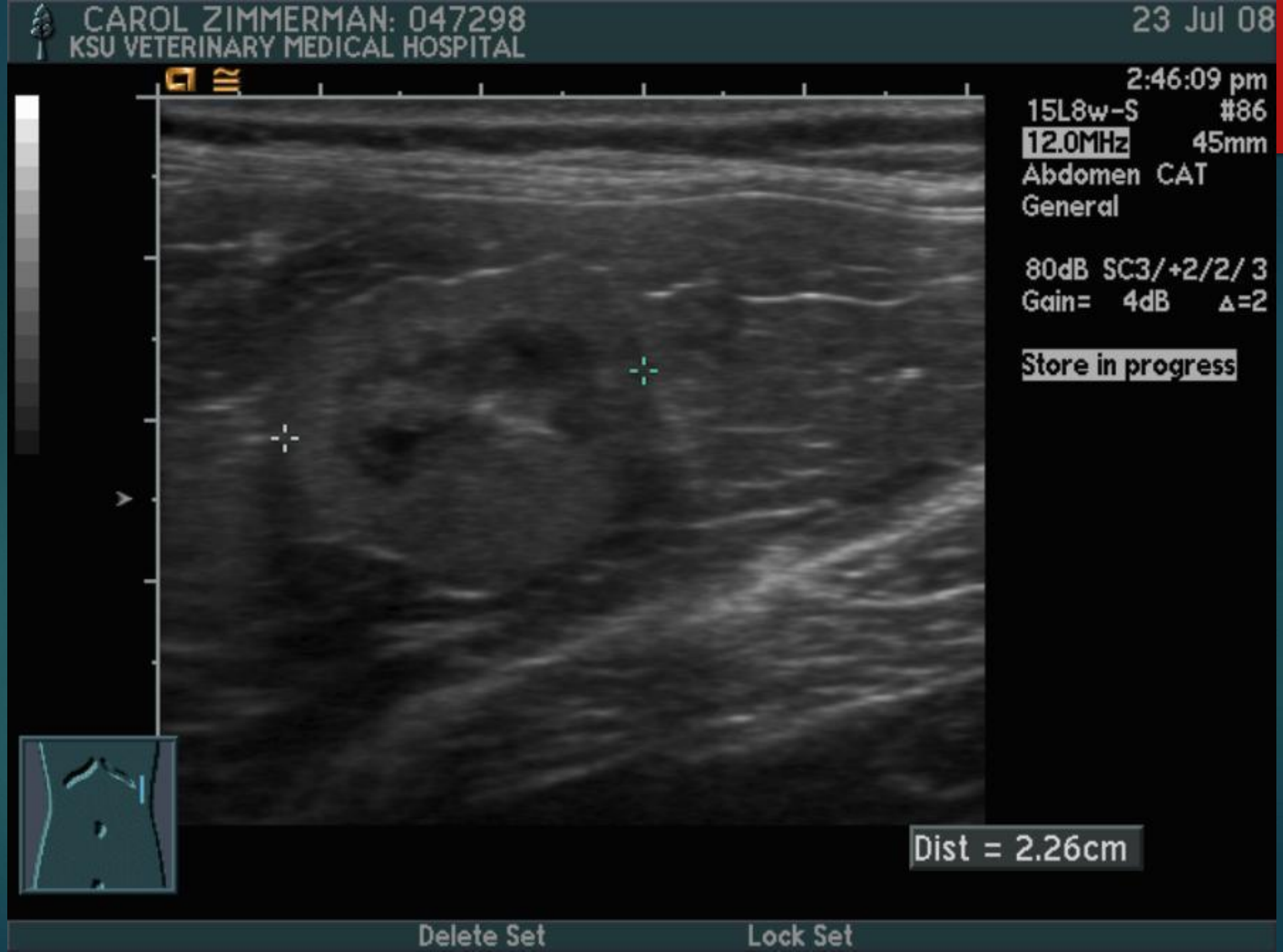
Further sub-classify based on the presence or absence of proteinuria and systemic hypertension. **www.IRIS-kidney.com*

***In the case of discrepancy between Creatinine and SDMA, consider body condition, and re-testing in 2-4 weeks. If values are persistent, consider assigning the patient to the higher stage.*

IRIS Guidelines: Diagnosis of Stage 1/Early Stage 2 CKD

- Kidney palpation or imaging abnormalities
- PU/PD due to loss of nephrons? (dogs > cats)
- Renal proteinuria (persistent w/ normal sediment)
- A persistent ↑ in SDMA (≥14 µg/dl) with sCr <1.6 mg/dl (cats) or <1.4 (dogs)
- ↑ in sCr within the RI without changes in muscle mass or hydration. Same for ↑ in SDMA w/in RI
  - e.g., an increase in sCr from 0.7 to 1.4 mg/dl over several years could indicate ≥ 50% nephron loss. > 50% because remaining nephrons have had time to undergo compensatory hypertrophy.


RIGHT K S U


CAROL ZIMMERMAN: 047298
KSU VETERINARY MEDICAL HOSPITAL
23 Jul 08
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15L8w-S #86
12.0MHz 45mm
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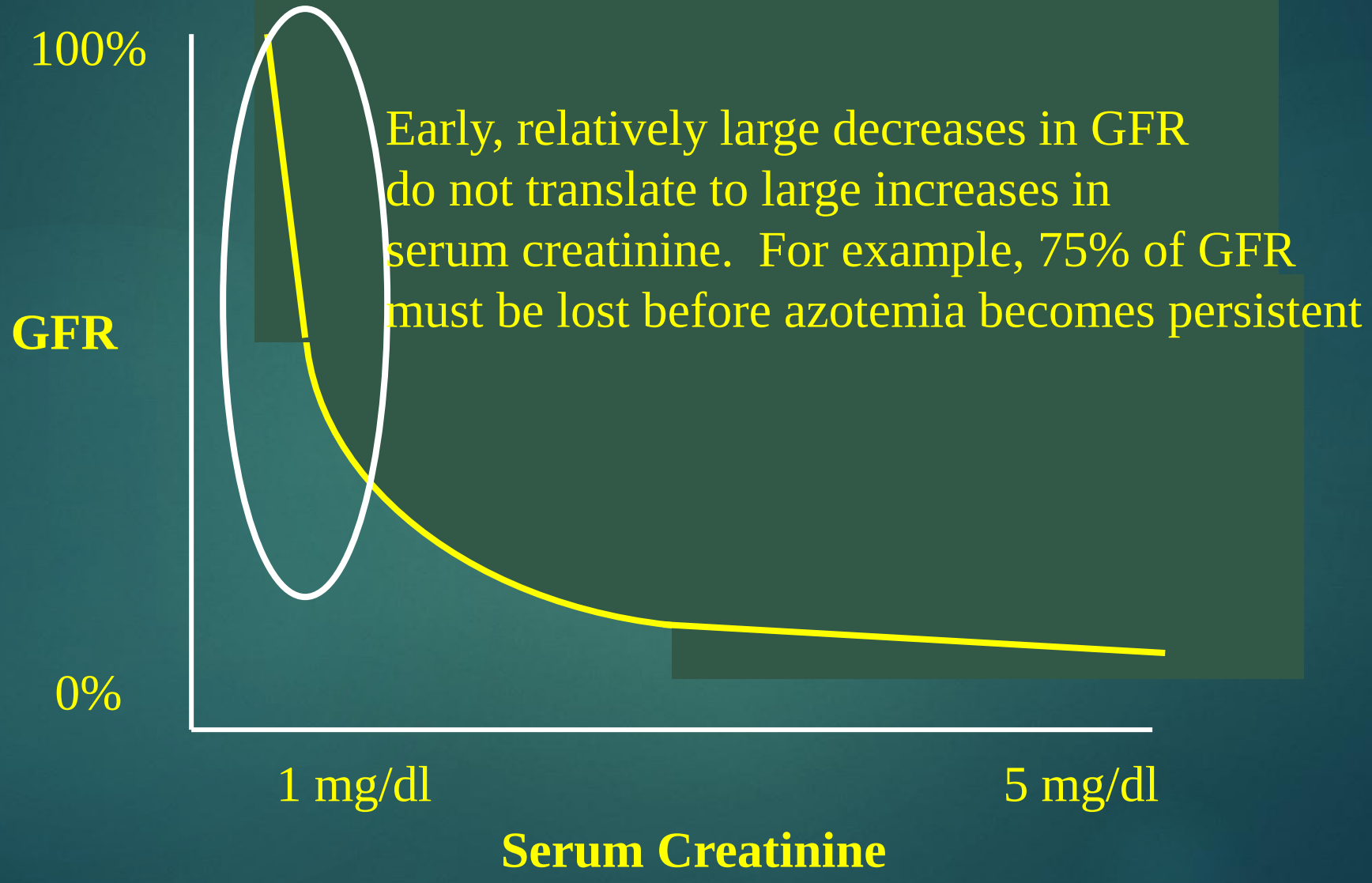
80dB SC3/+2/2/3
Gain= 4dB Δ=2
Store in progress
Dist = 2.26cm
Delete Set
Lock Set

Serum Creatinine Concentration: Limitations

- Reflects muscle mass as well as GFR
- Influenced by methodology
  - Jaffe reaction vs. enzymatic; bench top vs. reference lab
- Variability in inter-laboratory reference ranges can lead to both false negative and false positive results for diagnosis of azotemia[1,2]
  - Longitudinal assessment (individualized baseline) with consistent methodology = best practice

1. Boozer L, et al. JVIM, 2002;16:354 (abstract)
2. Ulleberg T, et al. Acta Vet Scand 2011;53:25

Relationship Between GFR and Serum Creatinine Concentration Is Not Linear



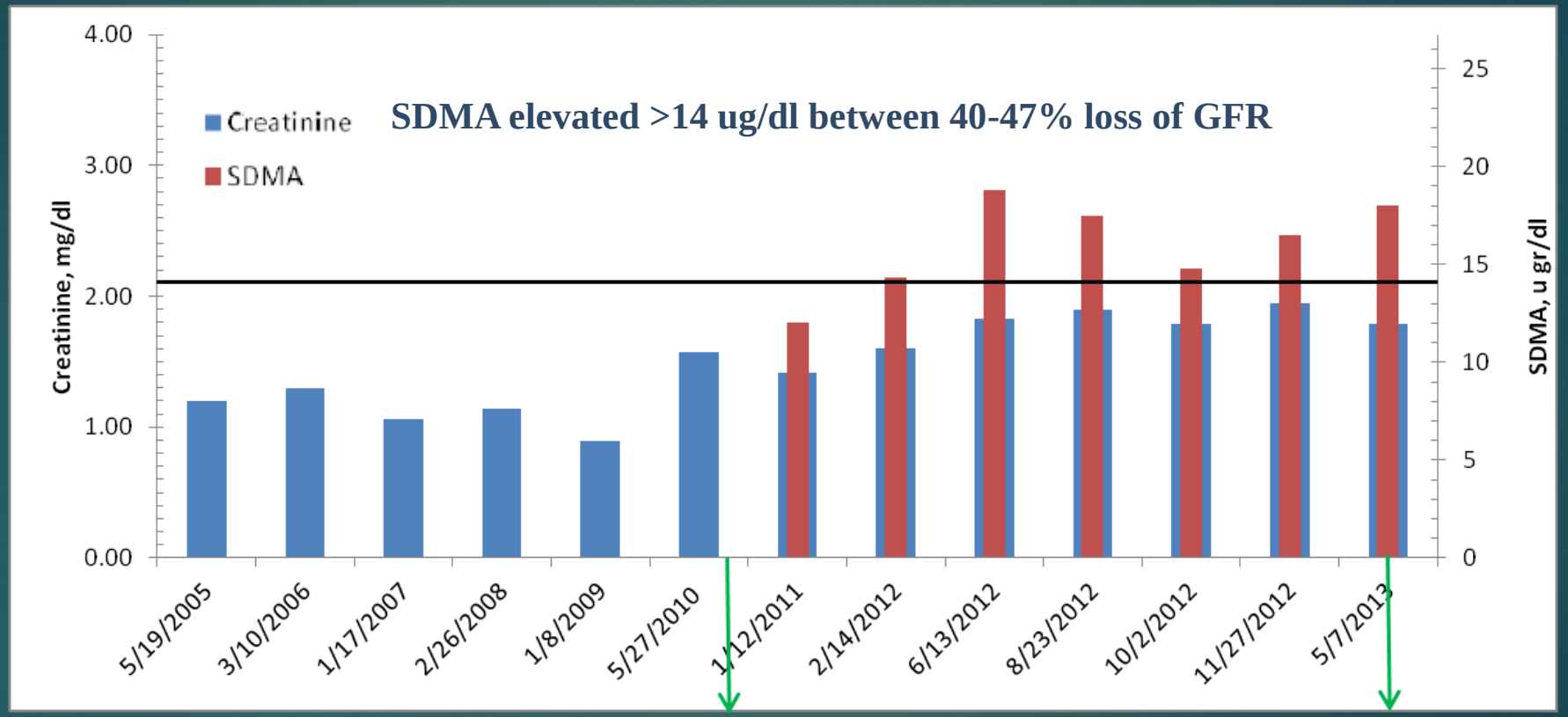
Early, relatively large decreases in GFR do not translate to large increases in serum creatinine. For example, 75% of GFR must be lost before azotemia becomes persistent

Serum Symmetric Dimethylarginine (SDMA)

- Derived from intranuclear methylation of L-arginine by protein-arginine methyltransferases
- Released into circulation after proteolysis
- > 90% eliminated by glomerular filtration
  - Freely filtered with no tubular reabsorption
  - Non-renal influences appear to be minimal
- More sensitive than sCr: In two longitudinal studies, SDMA increased an average of 9 and 17 months prior to sCr, respectively in dogs and cats with CKD[1,2]

1. Hall JA, et al. JVIM 2014; 28:1676 (Feline)
2. Hall JA, et al. JVIM 2016; 30:794 (Canine)

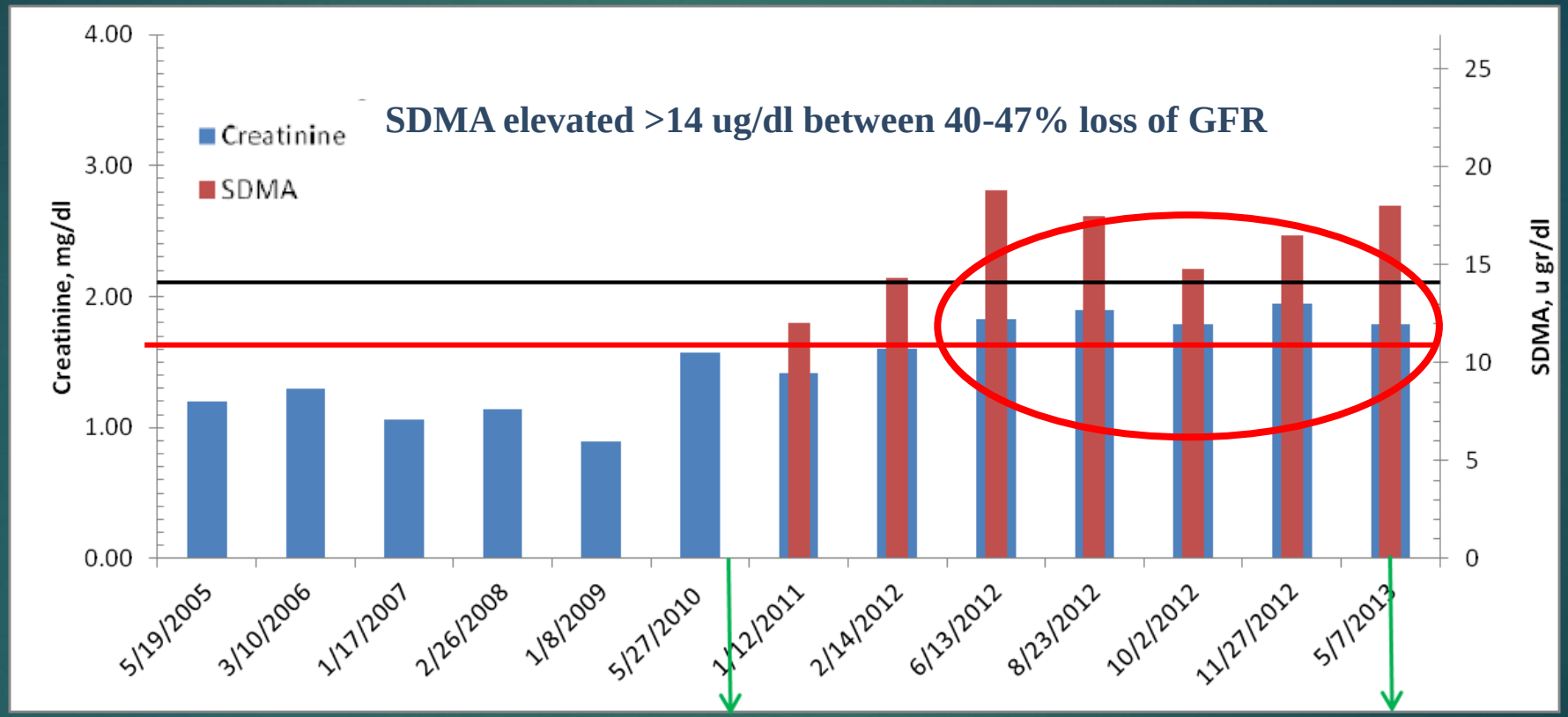
“Monkey” born 1996, FS, DSH



CKD diagnosed 6/16/2010
GFR 1.161 ml/min/kg
40% loss from expected mean (1.94 ml/min/kg)

47% loss
GFR 1.024 ml/min/kg

“Monkey” born 1996, FS, DSH



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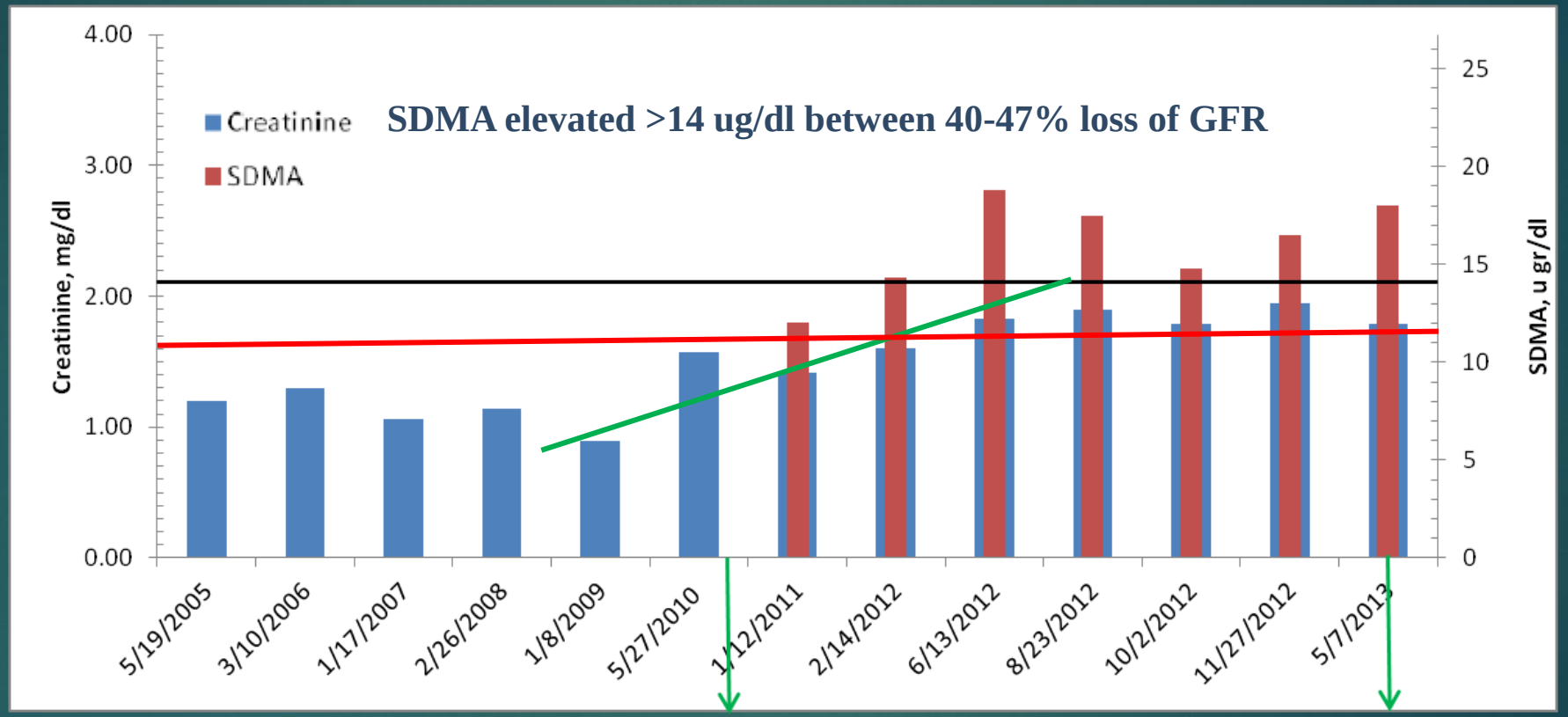

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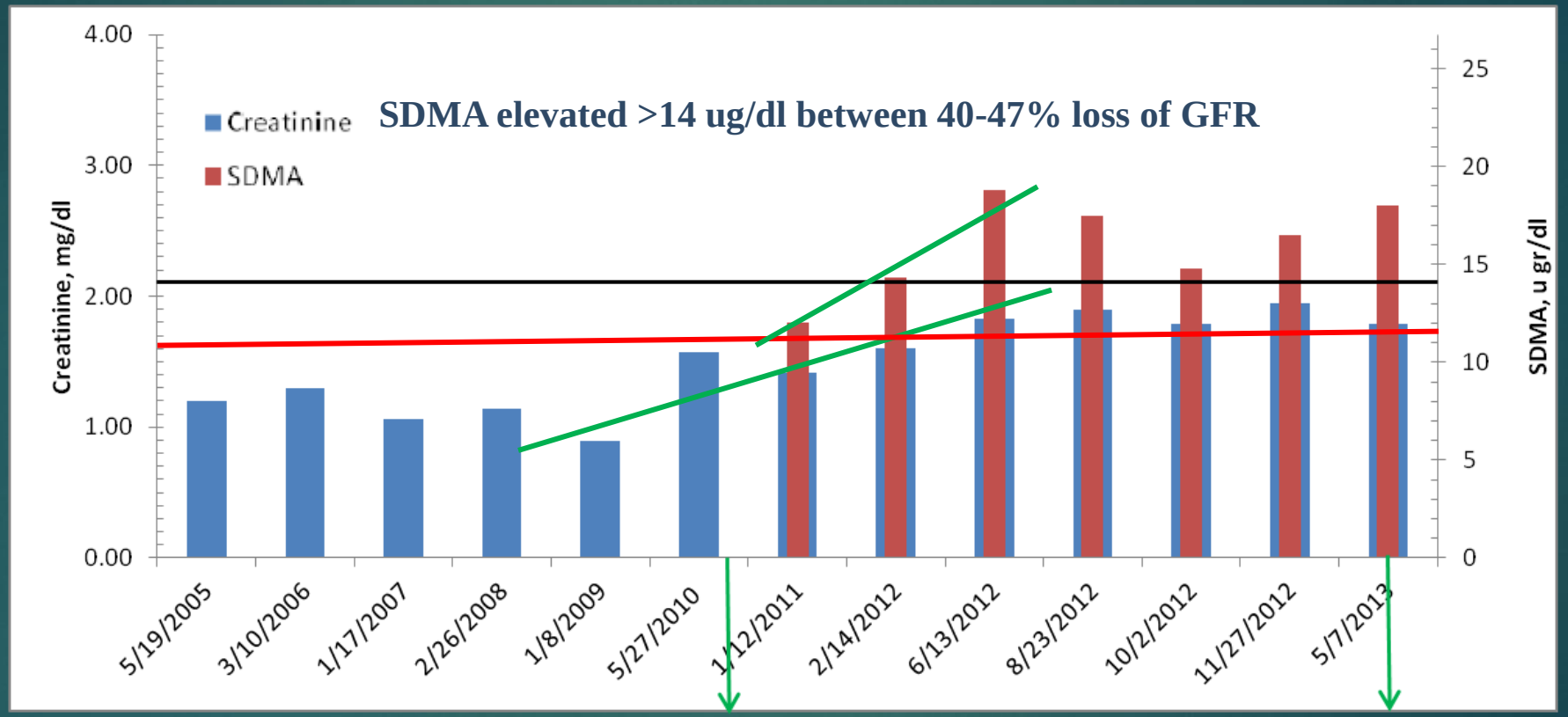

CKD diagnosed 6/16/2010
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40% loss from expected mean (1.94 ml/min/kg)

47% loss
GFR 1.024 ml/min/kg

Age-Specific Reference Intervals in Elderly Cats

Mortier F, et al JFMS 2023, Vol 25, Issue 11

- Using age-specific R is for sCr in mature adult (7-10 yrs) and senior cats (> 10 yrs) improves health screening. (developed using ASVCP Guidelines)
- Standard RI for feline sCr was **0.9-2.3 mg/dl**
- Age-specific RI for mature adult cats would be **0.8-1.85 mg/dl**
- Age-specific RI for senior cats would be **0.7-1.86 mg/dl**

Early Detection of CKD

- Serial determination of SrCr and SDMA concentrations
  - An increase ≥ 0.3 mg/dl is potentially real (vs. laboratory variation – when using the same lab/technique) for serum creatinine
  - An increase > 3.0 µg/dl is potentially real (vs. laboratory variation) for SDMA

Summary of SCr and SDMA for Early Diagnosis of CKD

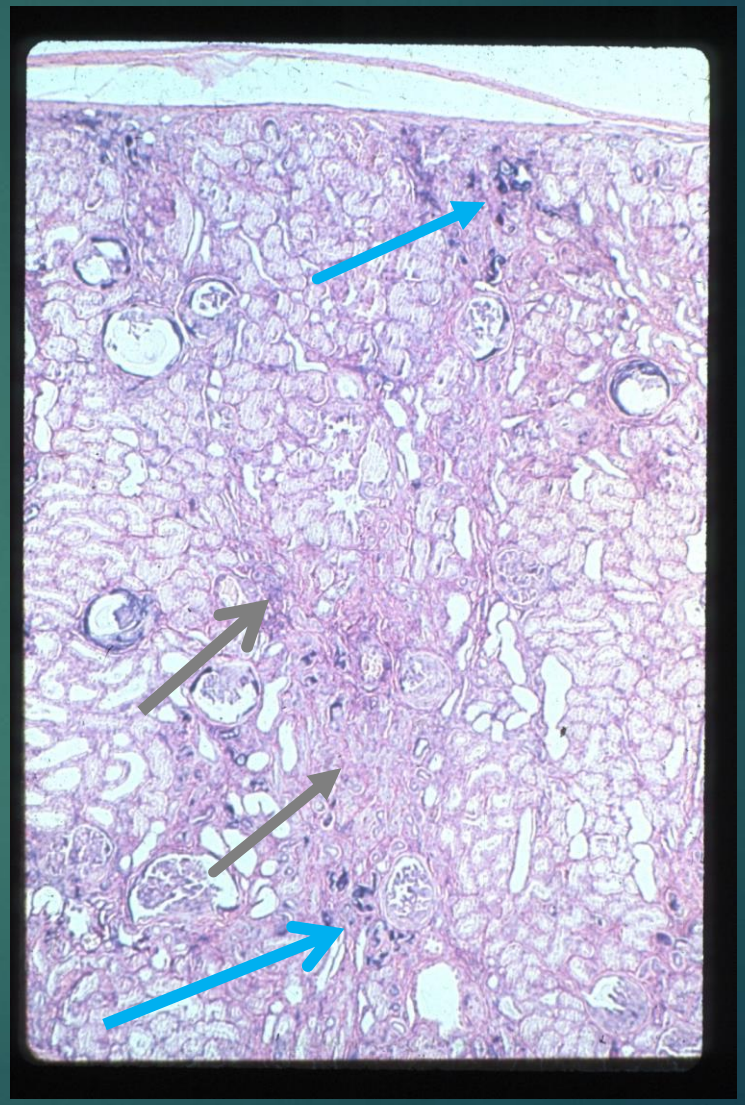
- SDMA is a more sensitive surrogate GFR marker than creatinine
- Longitudinal assessment of sCr and SDMA improves interpretation over single values
- Establishing baselines for subsequent longitudinal evaluation in individual patients is important
- Look for trends within the reference interval

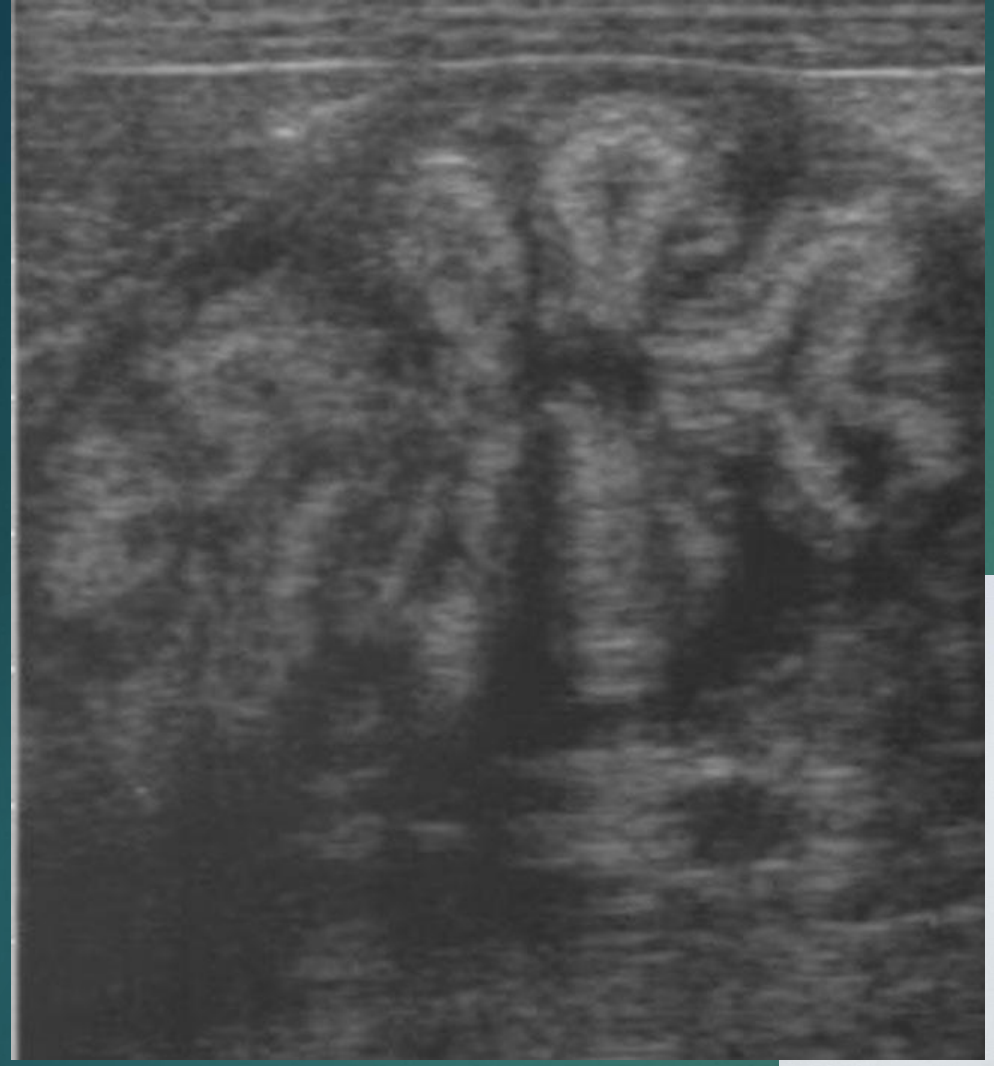
Renal Mineralization

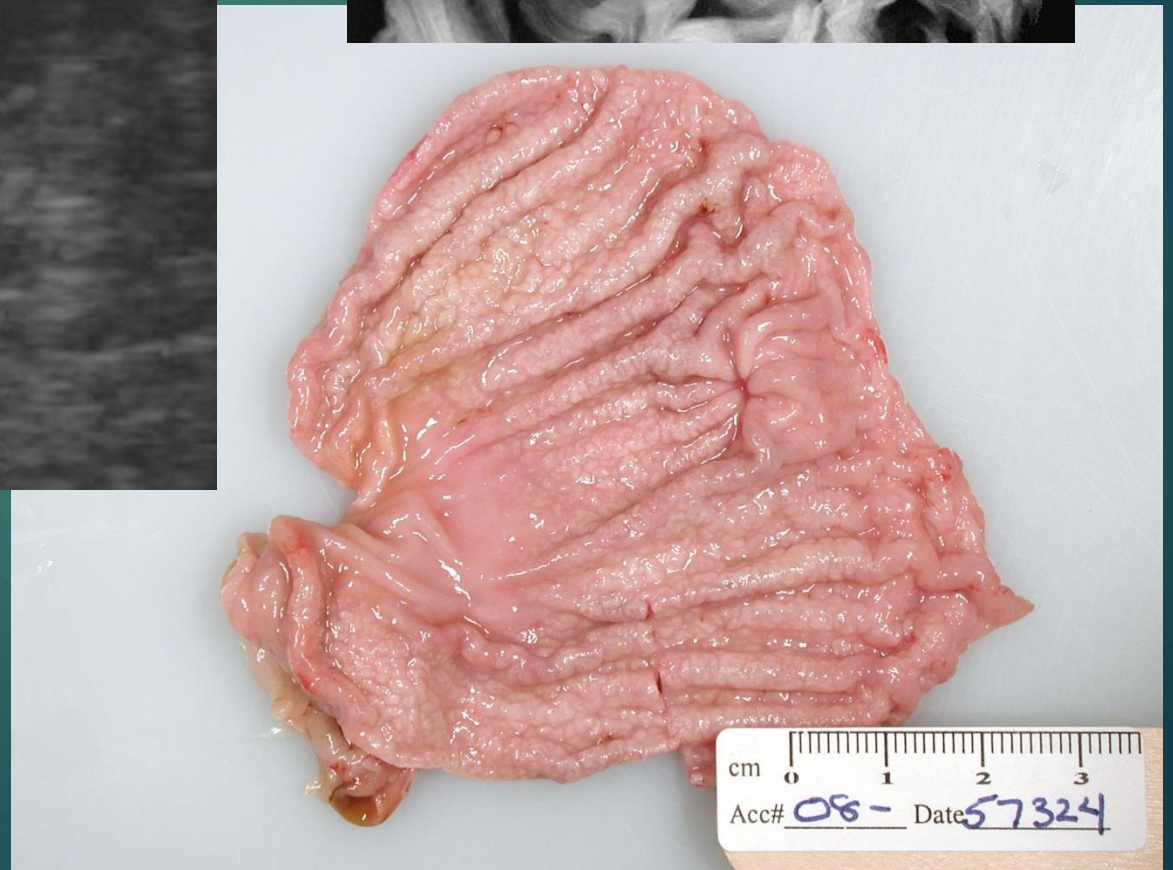
Failure to control dietary phosphorus in dogs and cats with CKD can result in renal mineralization and fibrosis and progressive loss of nephrons

Ross, AJVR 43:1023, 1982

Finco, AJVR 53:2264, 1992



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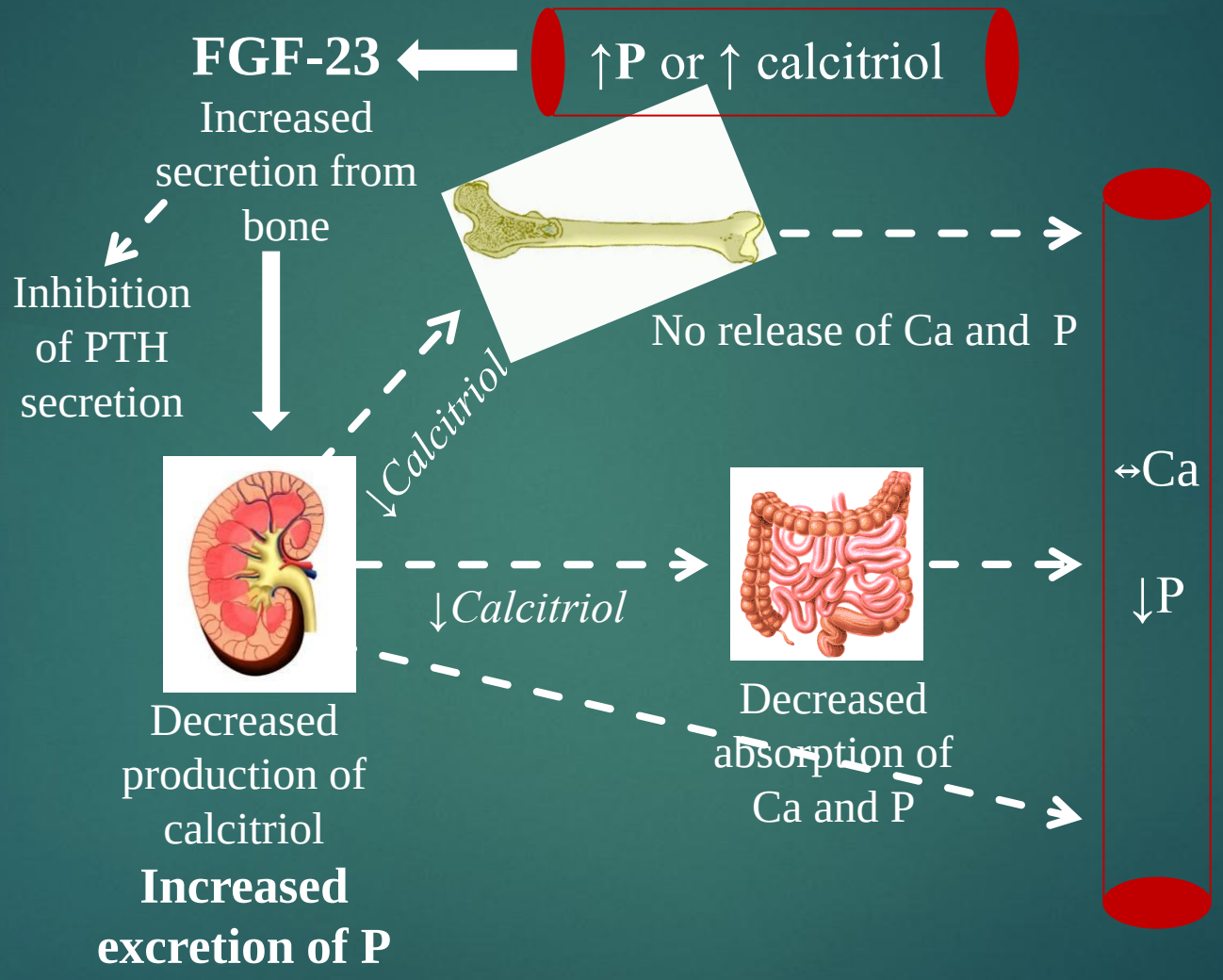
IRIS Therapeutic Targets for Management of Hyperphosphatemia in CKD

IRIS Stage	Target Serum Phosphorus (mg/dl)	Management Options
1	2.5-4.5	Renal diet or Normal ration + binder
2	2.5-4.5	Renal diet ± binder
3	2.5-5.0	Renal diet + ↑binder
4	2.5-6.0	Renal diet + ↑↑binder

FGF-23 regulates Phos much like PTH regulates Ca



FGF-23, secreted from bone in response to ↑ phosphorus. FGF-23 ↓ the density of renal Na-Phos cotransporters resulting in phosphaturia

Utility of FGF-23 in Early-Stage CKD

- Plasma Phos >4.5 mg/dl: Reduce Phos to <4.5 for 30 days then measure FGF-23
- Plasma Phos <4.5 mg/dl: Measure FGF-23
  - »FGF-23 >400 pg/ml: Restrict Phos intake and/or increase binder dose to ↓ FGF-23
  - »FGF-23 >300 <400 pg/ml: Continue to monitor q 2-3 months
  - »FGF-23 <300 pg/ml: No need for further Phos restriction

Utility of FGF-23 in Azotemic CKD

- Transition to a renal diet: Evaluate after 4-6 weeks
- Plasma Phos > Stage target range: Further reduce Phos intake and/or increase binder dose
  - »Phos within serum target range and FGF-23 >400 pg/ml: Further restrict Phos intake
  - »Phos within serum target range and FGF-23 <400 pg/ml: Continue current treatment

Summary/Interpretation of Serum Phosphorus

- Phosphorus within the reference interval deserves more scrutiny – use IRIS Stage-specific Phos target guidelines
- Control with progressive use of renal therapeutic diets and enteric phosphate binders
- Once stage specific target intervals are achieved, further assess treatment efficacy with plasma FGF-23 concentrations


Oh, crap! Was that TODAY?
SS Phos Control
REGAN

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