Chronic Kidney Disease: A collaborative team approach

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Objective

- Discuss the prevalence and incidence of CKD and its impact on global, national, local health-care resources
- Define Chronic Kidney Disease (CKD) and review current standard of care for CKD
- Define stages of CKD and identify factors that influences CKD progression
- Discuss the evaluation and management of CKD both in early and advanced stages
- Develop strategies for improving CKD outcomes including identifying indications for referral to nephrology

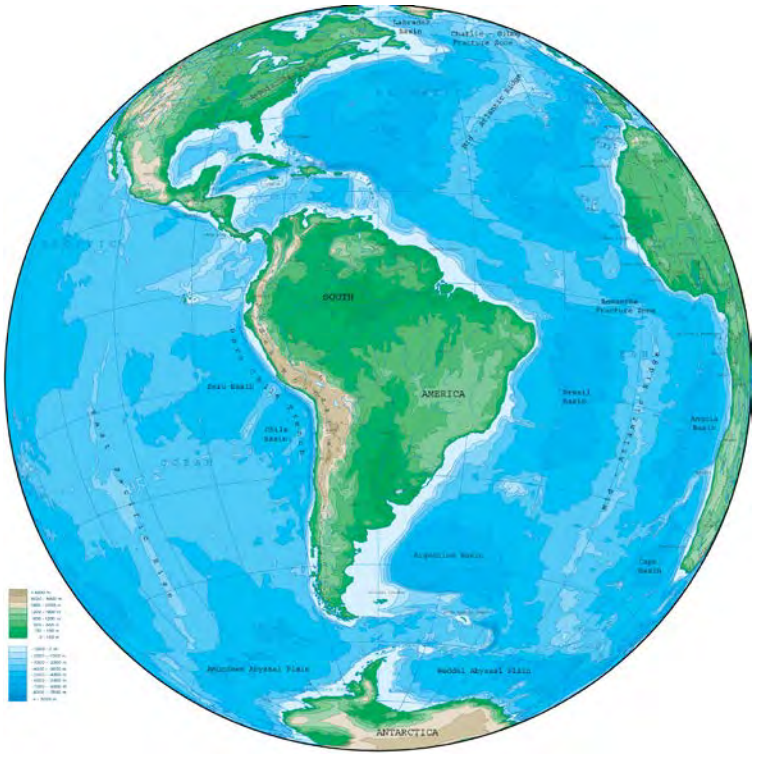
Introduction

- **Chronic Kidney Disease is an epidemic of worldwide proportion. It's progression to ESRD is a substantial burden on global health-care resources** [1]
- **CKD is a progressive disease, often accompanied by multiple co-morbidities. Early recognition and management of complication can improve patient's long-term outcome** [1]
- **Significant deficiencies in the care of CKD still exist. Opportunities for improvement include increasing recognition of CKD in primary care and improving collaborative care with nephrology** [2]

1 A M Nahas, A K Bello. Lancet, 2005
2 Allen, et.al. Journal of Gen. Int Med, 2010

Prevalence: CKD a global health issue [1]

- 240 M with Diabetes
  - Expected to increase to 380 M by year 2025
- 1 Billion people with HTN
  - 1.56 Billion by year 2025



1 George L. Bakris and Eberhard Ritz, Hypertension and Kidney Disease, A Marriage that Should Be Prevented, Kidney International 75, 449-452 (13 February 2009)
2 National Diabetes Information Clearing House, NDIC 2011
3 CDC. Vital signs: prevalence, treatment, and control of hypertension—United States, 1999-2002 and 2005-2008. *MMWR*. 2011;60(4):103-8.

Prevalence and Incidence

- CKD is recognized as a global health issue [1]
  - Increasing prevalence of Diabetes and HTN worldwide
  - DM prevalence over 240 M people worldwide and expected to increase to 380 M by year 2025
  - Nearly 1billion people worldwide have HTN and is expected to increase to 1.56 billion by year 2025
  - US – 28.5M affected by Diabetes. It is the 7th leading cause of death in the US
  - About 1 in 3 US adults – an estimated 68 M people – has high blood pressure

1 George L. Bakris and Eberhard Ritz, Hypertension and Kidney Disease, A Marriage that Should Be Prevented, Kidney International 75, 449-452 (13 February 2009)
2 National Diabetes Information Clearing House, NDIC 2011
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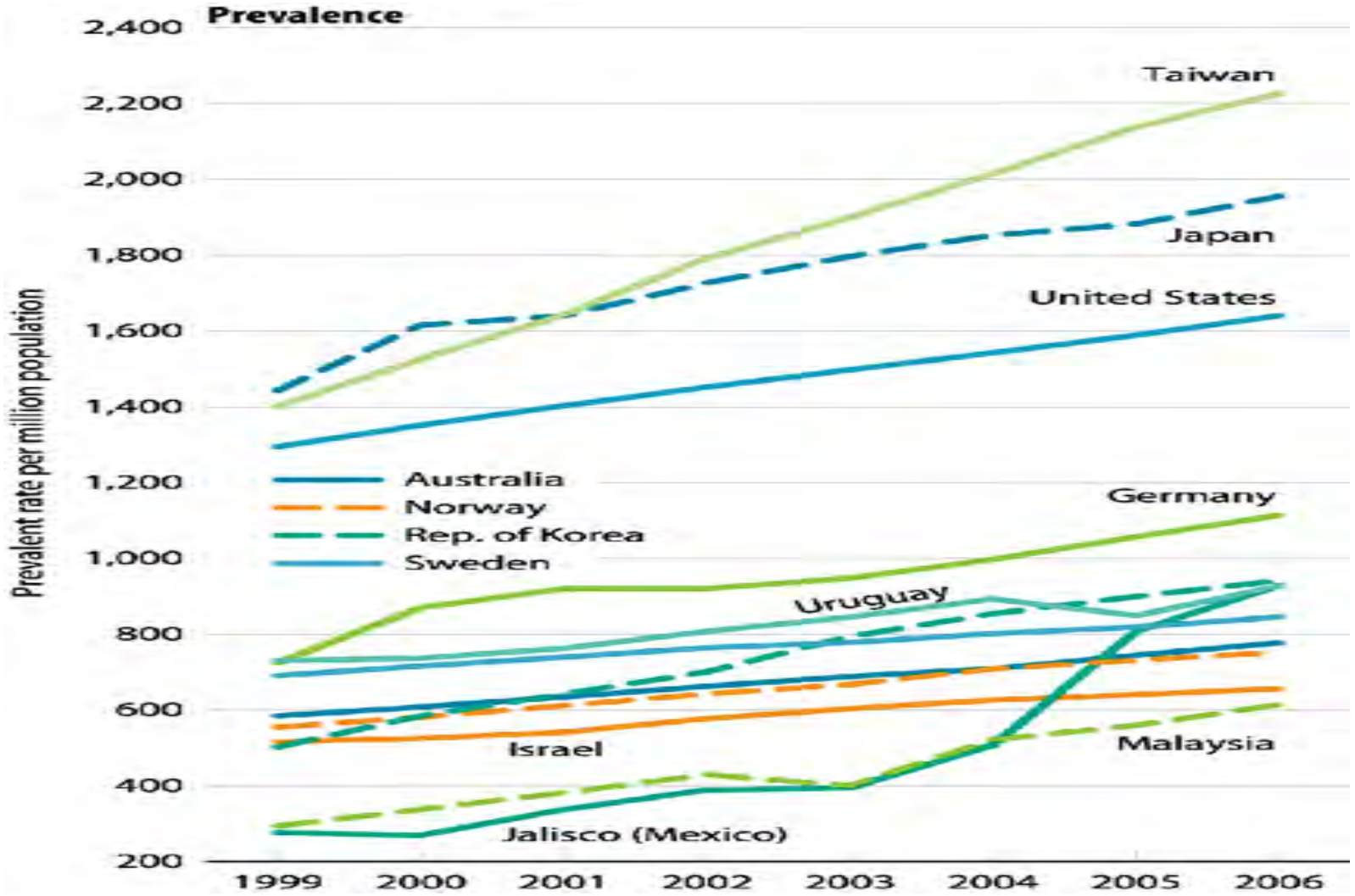
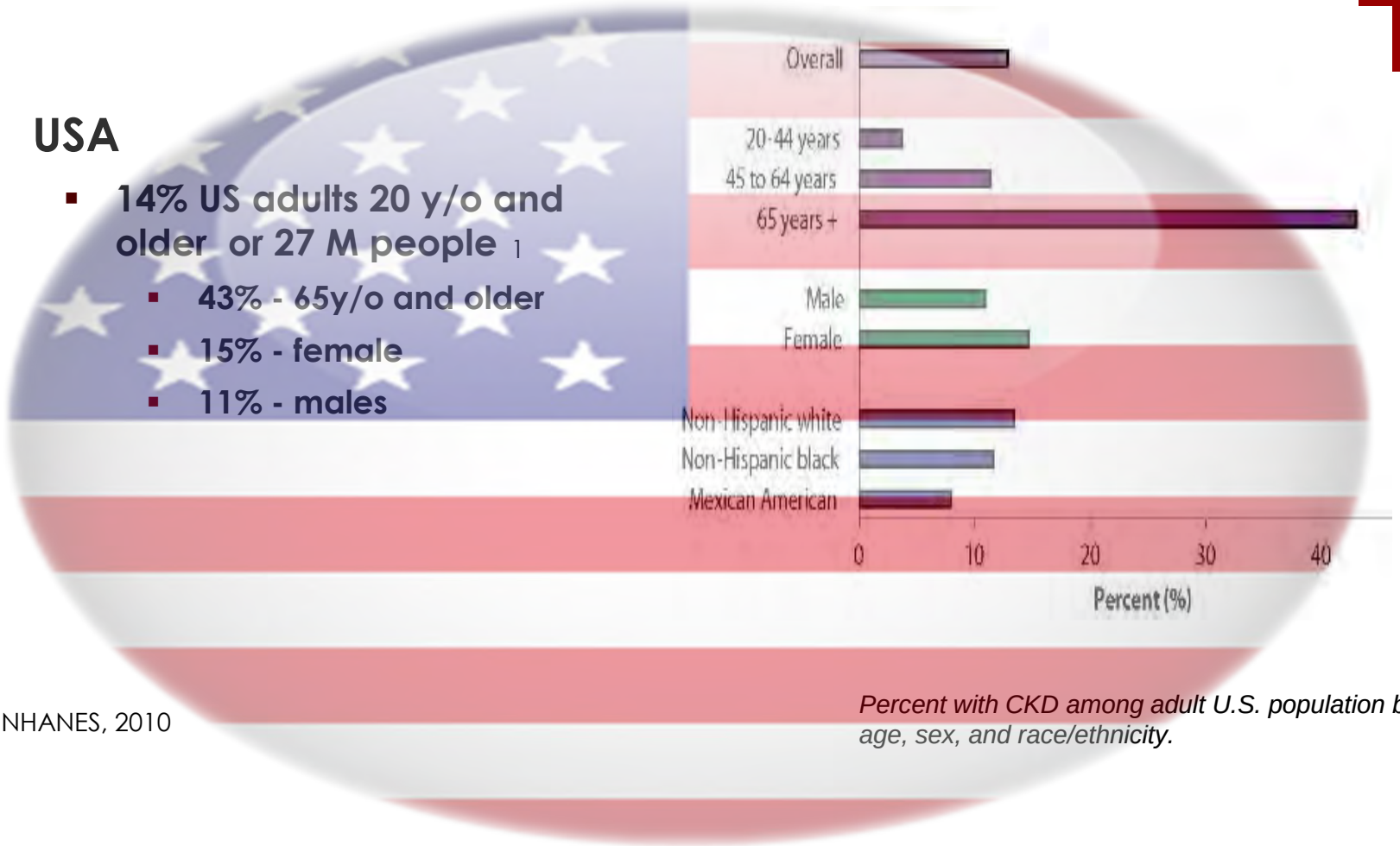


Allan J. Collins, CKD and the Public Health Agenda for Chronic Diseases, Figure 12.1 (continued; Volume Two) USRDS 2008 ADR, CDRG presentation, 25 March 2009

Prevalence of CKD: USA

- **USA**
  - **14% US adults 20 y/o and older or 27 M people** [1]
    - **43% - 65y/o and older**
    - **15% - female**
    - **11% - males**



Percent with CKD among adult U.S. population by age, sex, and race/ethnicity.

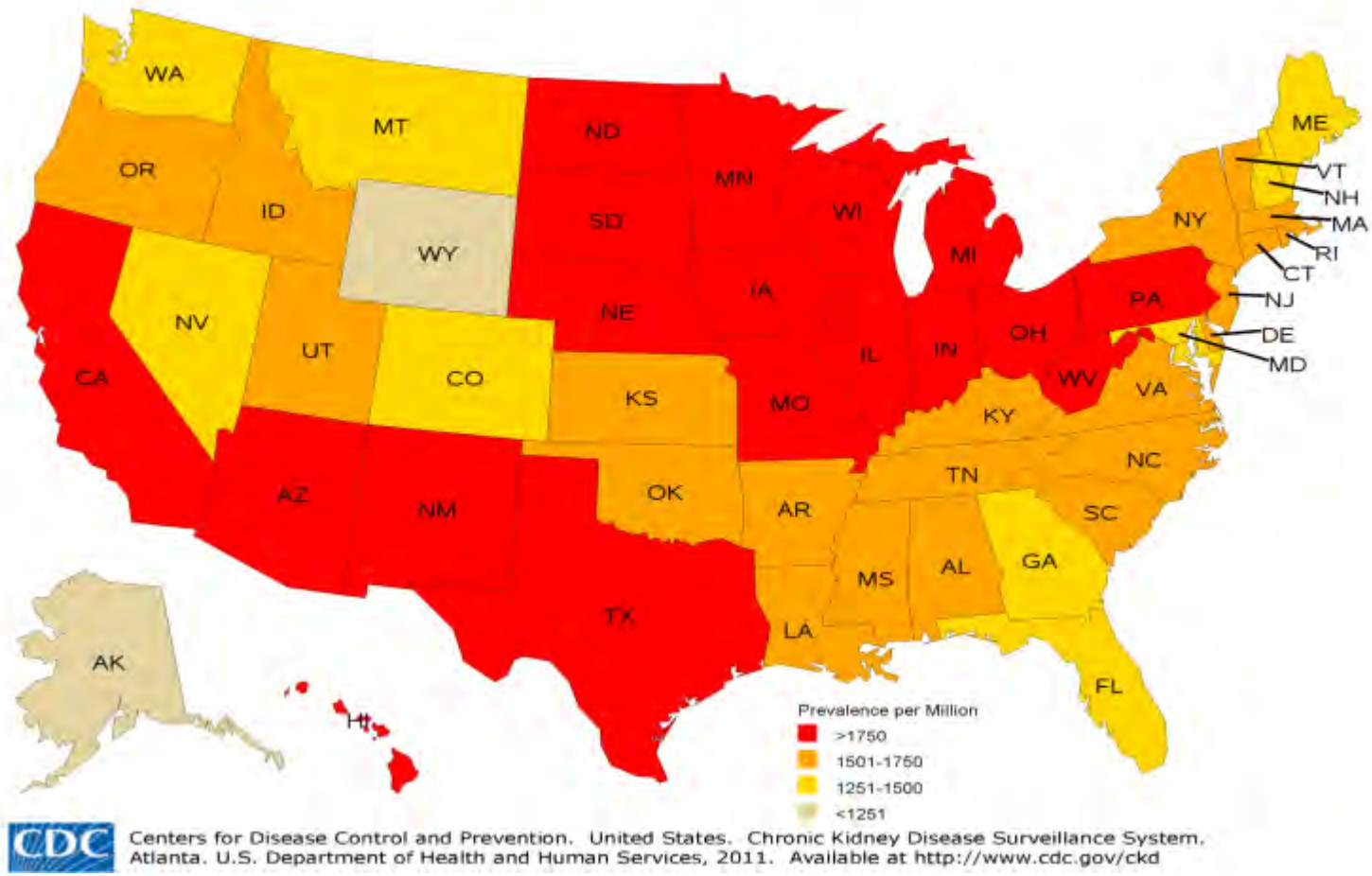
1 NHANES, 2010

Prevalence of CKD

Stages of CKD	GFR (ml/min/1.73m2)	Prevalence by Stage
1	≥90	3,600,000
2	60-89	6,500,000
3	30-59	15,500,000
4	15-29	700,000
5	<15	341,000

Coresh J, et al. *JAMA*. 2007

Prevalence of ESRD by U.S. State (2009)
US Renal Data System



Centers for Disease Control and Prevention. United States. Chronic Kidney Disease Surveillance System. Atlanta. U.S. Department of Health and Human Services, 2011. Available at http://www.cdc.gov/ckd

Incidence of ESRD by U.S. State (2009)
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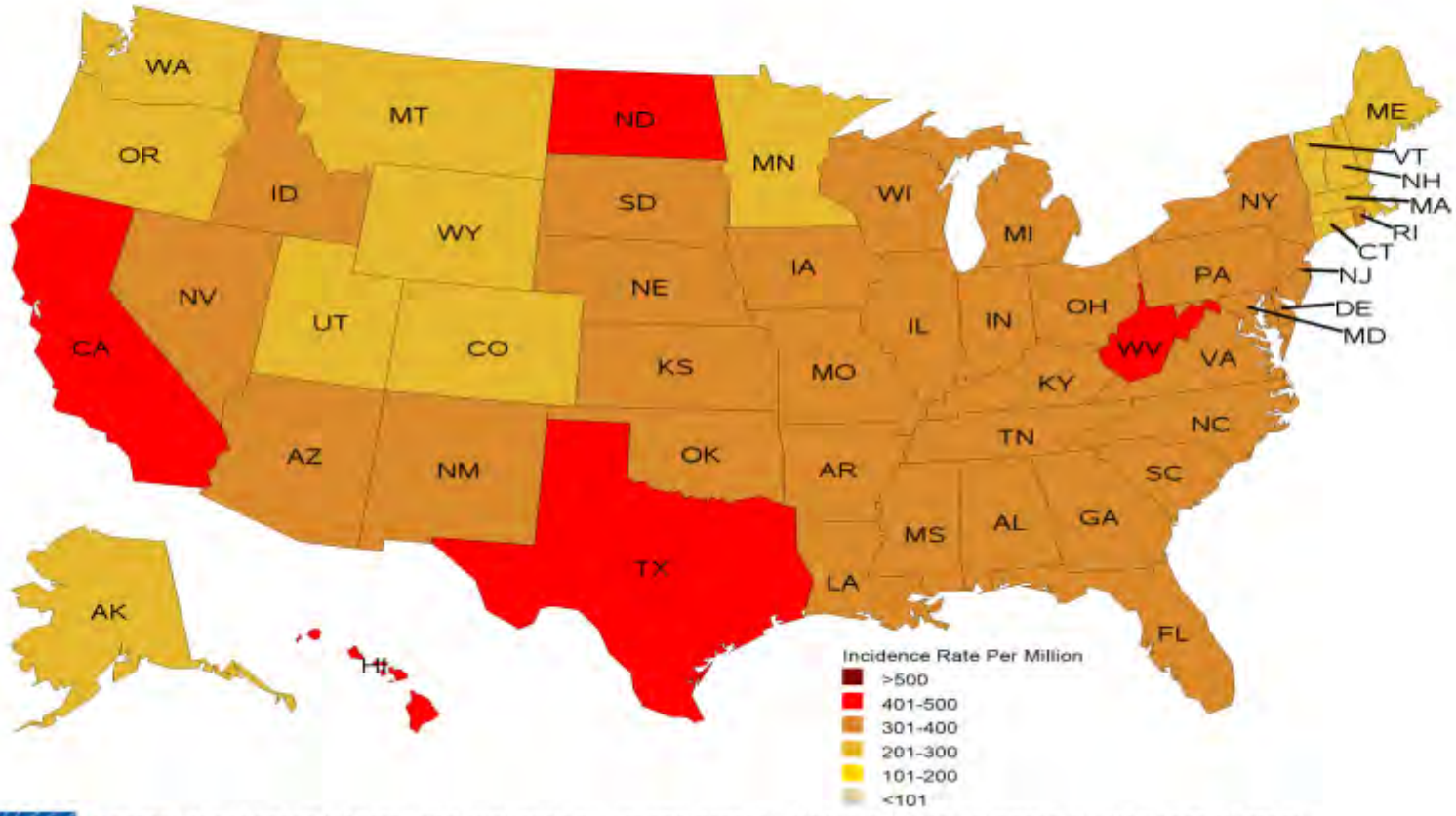


Centers for Disease Control and Prevention. United States. Chronic Kidney Disease Surveillance System. Atlanta. U.S. Department of Health and Human Services, 2011. Available at http://www.cdc.gov/ckd

Prevalence and Incidence of CKD in California

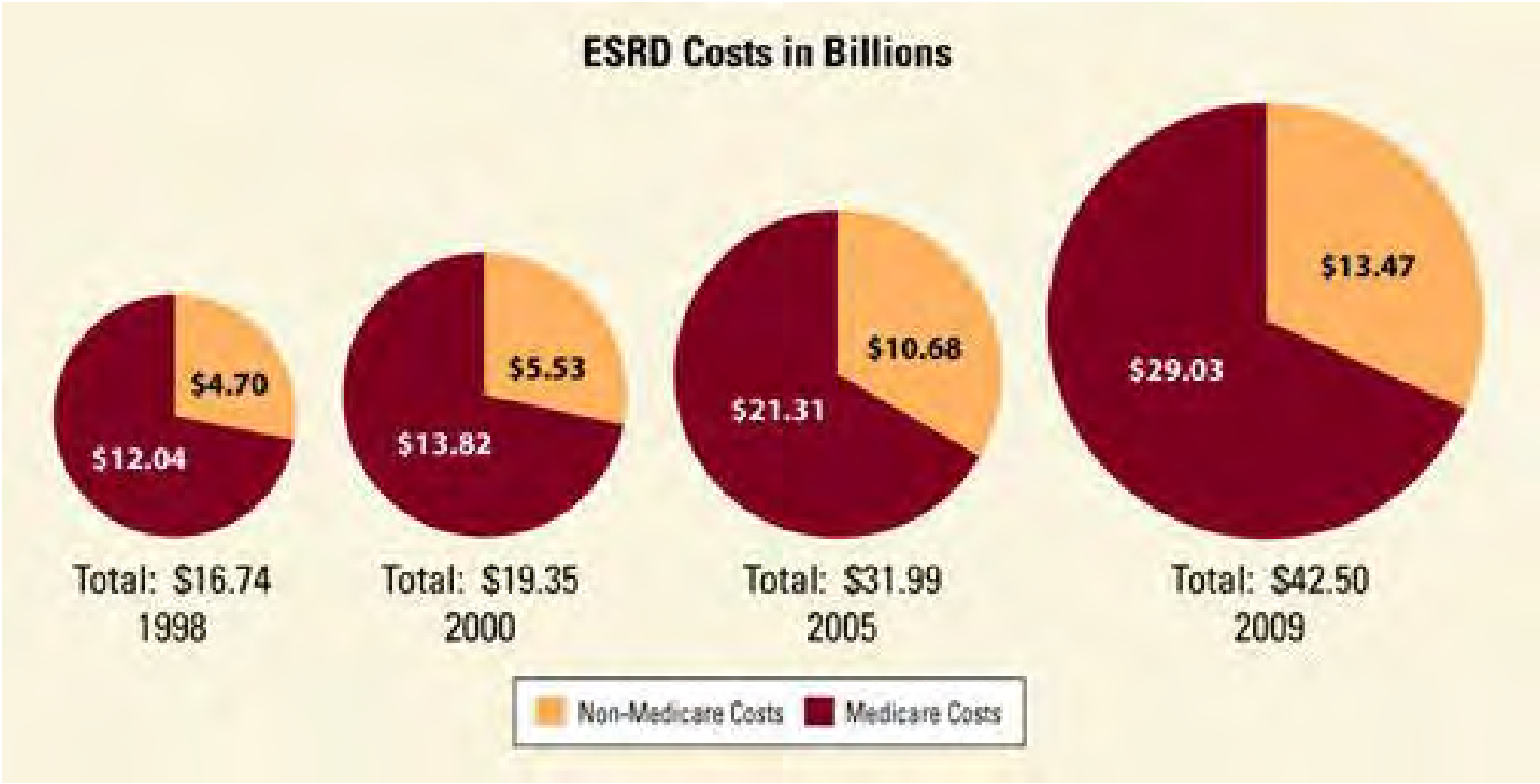
- 2009 USRDS data for California
  - Prevalence of CKD in California

    2021.02 per million population

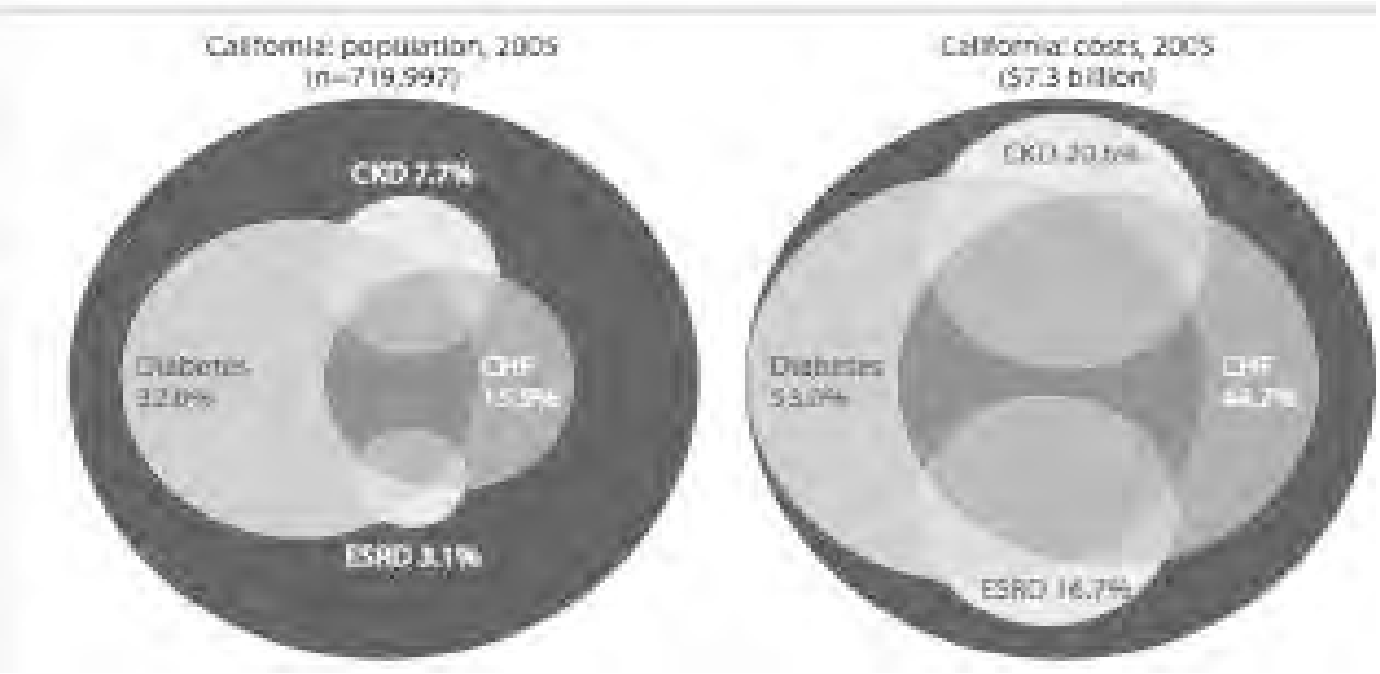
    4th highest in all of US
  - Incidence of CKD in California

    407.948 per million population

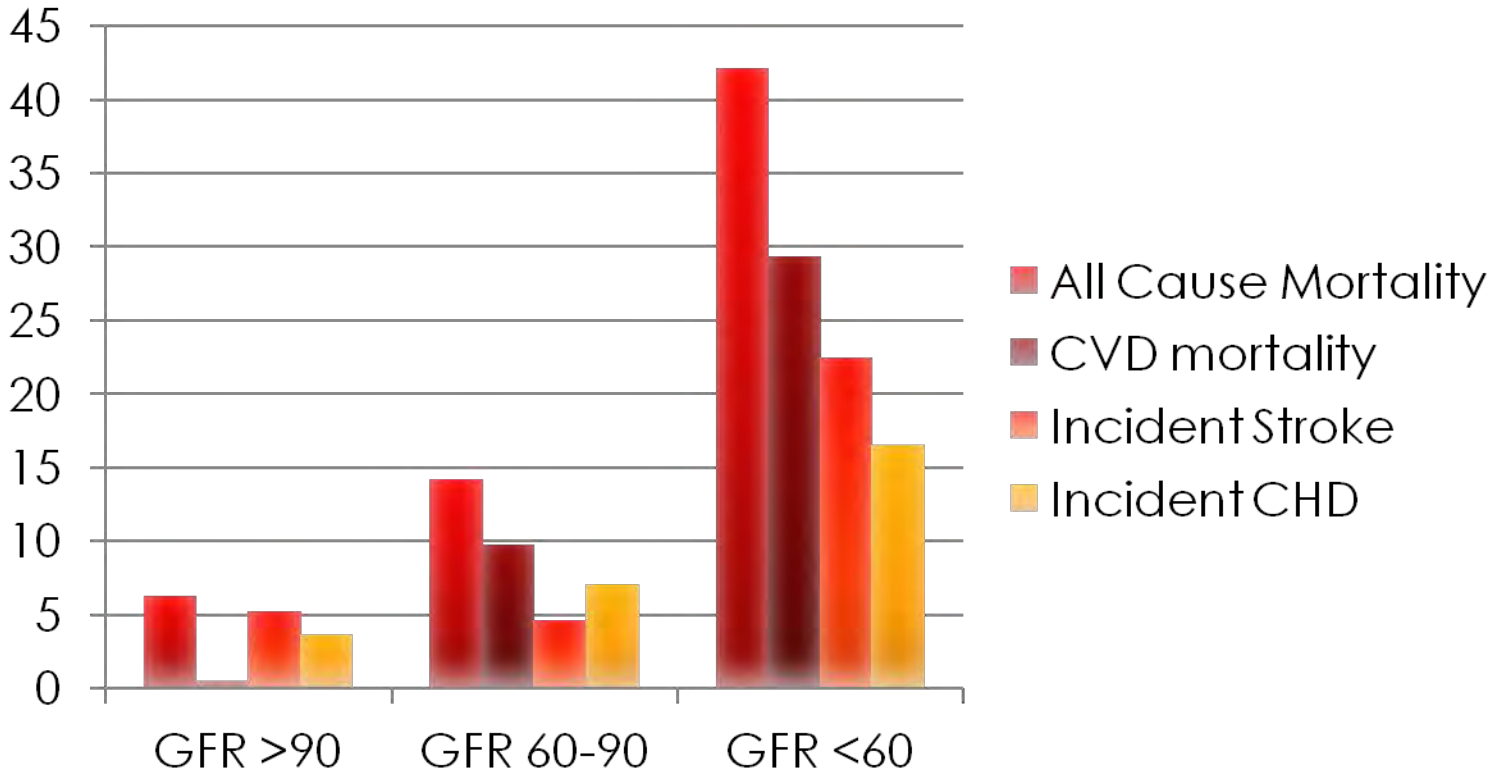
Economic Burden of Kidney Disease



Economic Burden of Kidney Disease



Mortality rate



Zhang R, et al. *Plosone.org*. 2011. Decreased Glomerular filtration rate is associated with mortality and cardiovascular events in patients with HTN: A Prospective Study

CKD: Standards of Care

- Kidney Disease Outcomes Quality Initiative KDOQI
  - Established in 1995 by National Kidney Foundation (NKF) to develop clinical practice guidelines for the management of all stages of CKD
  - 1997 – Published the first KDOQI guidelines. Currently have 13 guidelines
  - 2007 – Publication of Clinical Practice Recommendations for Diabetes and CKD.
  - 2012 – Release updates of guidelines

CKD: Standard of care

- Kidney Disease: Improving Global Outcomes (KDIGO)
  - Established in 2003 as an independent incorporated non-profit foundation governed by an international Board.
  - Published KDIGO Clinical Practice Guidelines
    - 2008 – Prevention, Diagnosis, Evaluation and Treatment of Hep C in CKD
    - 2009 – Diagnosis, Evaluation, Prevention, and treatment of CKD-MBD
    - 2009 – Care of Transplant Recipients
    - 2012 – Acute Kidney Injury

CKD: Definition

- Kidney damage for > 3months
  - Defined by structural or functional abnormalities of the kidney, with or without decreased GFR manifest by either:
    - Pathological abnormalities (Polycystic kidney dz, Obstructive uropathy)
    - Markers of kidney damage – abnormalities in composition of blood or urine, imaging abnormalities (increase creatinine, decrease eGFR, proteinuria)
- GFR less than 60 ml/min for > 3 months with or without kidney damage

Glomerular Filtration Rate (GFR)

- GFR is equals to the sum of the filtration rates in all functioning nephrons
- Can not be measured directly in clinical setting
- eGFR gives a rough estimate of the number of functioning nephron
  - MDRD equation
  - Cokcroft-Gault equation
  - CKD-EPI equation – CKD Epidemiology Collaboration Equation

Estimated GFR

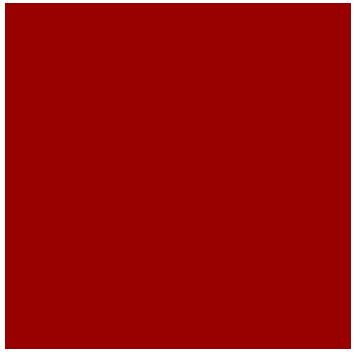
- Based on the physiology of renal blood flow(RBF) and renal plasma flow (RPF)
- RBF – Volume of blood delivered to the kidneys per unit time.
  - 20% of the total cardiac output
  - Composition is 50% cells and 50% plasma
- RPF – volume of plasma that reaches the kidneys per unit time.

Estimated GFR

- Cardiac output = 6 L/min
- RBF – 20% of cardiac output = 1.2 L/min
  - 50% plasma and 50% cells = 600 ml plasma/600 ml cells
- RPF – 20% of plasma filtered by glomerulus per unit time = 120 ml/min
- Estimated normal GFR = 120 ml/min

Estimated GFR

- Calculating eGFR – resources available online or Apps on your smart phones.
  - MDRD – Modification of Diet in Renal Disease
  - CKD-EPI – Chronic Kidney Dz Epidemiology Collaboration Equation
    - Based on 4 variables
      - Serum creatinine, age, sex, and race
  - NKF, NKDEP, ASN all recommends the automatic reporting of eGFR whenever serum creatinine level is ordered
  - In 2011 NKF recommended that clinical laboratories use CKD-EPI equation to report eGFR



- Shaq and betty white and their lab

Stages of CKD

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.

Stages of CKD

Stage	Description	eGFR	Action Plan
1	Kidney Damage with normal eGFR	Normal or >90 ml/min with other evidence of CKD	Diagnose and treat type of kidney disease; treat comorbid conditions; slow progression of CKD; treat modifiable CVD risk factors; periodically restage
2	Mild Impairment	60-89 ml/min with other evidence of CKD damage	Above plus Adjust drug dosages for level of GFR
3	Moderate Impairment	30-59 ml/min	Evaluate for and treat complications of CKD; avoid nephrotoxic drugs
4	Severe Impairment	15-29 ml/min	Above (stage 3) plus Prepare for kidney replacement therapy
5	Renal Failure	<15 ml/min	Assessment of uremia Start of RRT when uremia is present

National Kidney Foundation. *K/DOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification and Stratification*. Am J Kidney Dis 39:S19, 2002

Screening Recommendations

- US Preventative Task Force (2012)
  - Concludes that the evidence is insufficient to assess the balance of benefits and harms of routine screening for chronic kidney disease (CKD) in asymptomatic adults
- NKF Recommendation
  - Risk Assessment of CKD in all patients
  - High Risk Patients: Measure BP, Serum creatinine level, Check urine albumin levels, check urine erythrocytes, leucocyte
- American Diabetes Association
  - Annual screening of all diabetics using urine albumin and serum creatinine testing
- The National Institutes of Health's Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
  - Patients with HTN should have urinalysis and serum creatinine testing. Urine albumin is optional

CKD

- Benefits of Early Detection:
  - Delaying onset of CKD in those at increased risk
  - Slowing CKD progression
  - Decreasing the development of cardiovascular disease in people with CKD

- Late Recognition:
  - Rapid Progression of Disease
  - Worse health status at time of initiation of dialysis
  - Higher mortality rate
  - Delayed referral for transplant

Collaborative roles between PCP and Nephrologist

Control of HTN
Diabetes Management
Modification of co-morbidities
Anemia Management
Renal Osteodystrophy Management
Renal replacement education
Access Placement
Transplant evaluation

Assessment of CKD: Risk Factors

- Clinical Factors
  - Diabetes
  - HTN
  - Family hx of CKD
  - Autoimmune Dz
  - Sx of Urinary obstruction/Frequent UTI
  - Analgesic abuse/exposure to certain drugs
  - GU malignancy
  - Other Malignancies
  - Systemic infections
- Sociodemographic Factors
  - Older age
  - Ethnicity: African American, American-Indian, Hispanic, Pacific Islander
  - Exposure to certain chemicals and environmental conditions
  - Low income/education

National Kdiney Foundation. *Am J Kidney Dis. 2003*

Assessment of CKD

- Physical Examination
  - Significant findings include:
    - Obesity
    - HTN
- Laboratory Exams: Markers of Kidney Disease
  - Serum Creatinine and eGFR
  - Assessing Proteinuria, UACR
- Staging of CKD

Progression of CKD

- Microalbuminuria
- Proteinuria
- Decline in GFR
- Increase in creatinine level

Proteinuria

- Proteinuria
  - Marker of kidney injury
  - Marker for progressive cardiovascular disease
  - Persistent increase in proteinuria indicates: [1]
    - Defect in glomerular membrane
    - Impaired tubular protein reabsorption
    - Increase filtration of low molecular weight protein

1 Carroll, M. MD, et. Al. Proteinuria in Adults: A diagnostic approach. AM Fam Phy, 2000

Proteinuria

- Proteinuria definitions
  - Normal - <150 mg/day
  - Overt proteinuria - Urine total protein ≥300 mg/day
    - In CKD, larger amounts are associated with worse renal survival
  - Nephrotic range proteinuria - Urine total protein: ≥ 3.5 g/day; with serum albumin: < 3.0 g/dL
    - Presence of nephrotic range proteinuria with edema, hypoalbuminemia, and hyperlipidemia is defined as nephrotic syndrome

Proteinuria

- Urine Dipstick proteinuria ranges:
  - 0 mg/dL – Negative
  - 15-30 mg/dL – Trace
  - 30-100 mg/dL – 1+
  - 100-300 mg/dL – 2+
  - 300-1000 mg/dL – 3+
  - >1000 mg/dL – 4+
- Limitations: Not sensitive to detect microalbuminuria

Urine Albumin-Creatinine Ratio

- Ratio between 2 measured substances – urine albumin and urine creatinine
- UACR estimates 24-hr urine albumin excretion
- Reported in mg/g and approximates albumin excretion in mg/day
- Unaffected by variations of urine concentration

NKF-K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Dis 2002; 39(Suppl 1):S1.

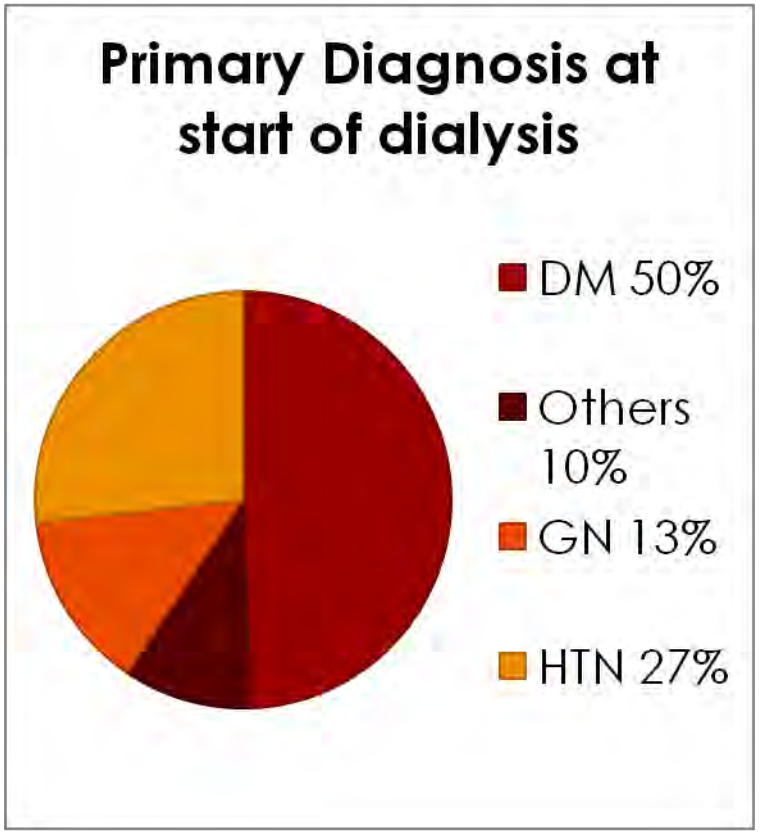
Urine Albumin-Creatinine Ration

- Interpreting UACR results
  - Normal – 30 mg albumin/g Creatinine
  - Microalbuminuria – 30-300 mg albumin/g Creatinine
  - Macroalbuminuria - >300 mg albumin/g Creatinine

NKF-K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Dis 2002; 39(Suppl 1):S1.

Evaluation of CKD

- Etiology of CKD
  - DM – most common cause of CKD
  - HTN
  - Glomerulonephritis
  - Auto-Immune Dz
  - Urinary Tract Obstruction
  - Analgesic Nephropathy
  - HIV and HCV related kidney disease
  - Renovascular disease
  - Malignancy – Multiple Myeloma, renal cancer



United States Renal Data System (USRDS) 2008 Annual Data Report

Evaluation of CKD

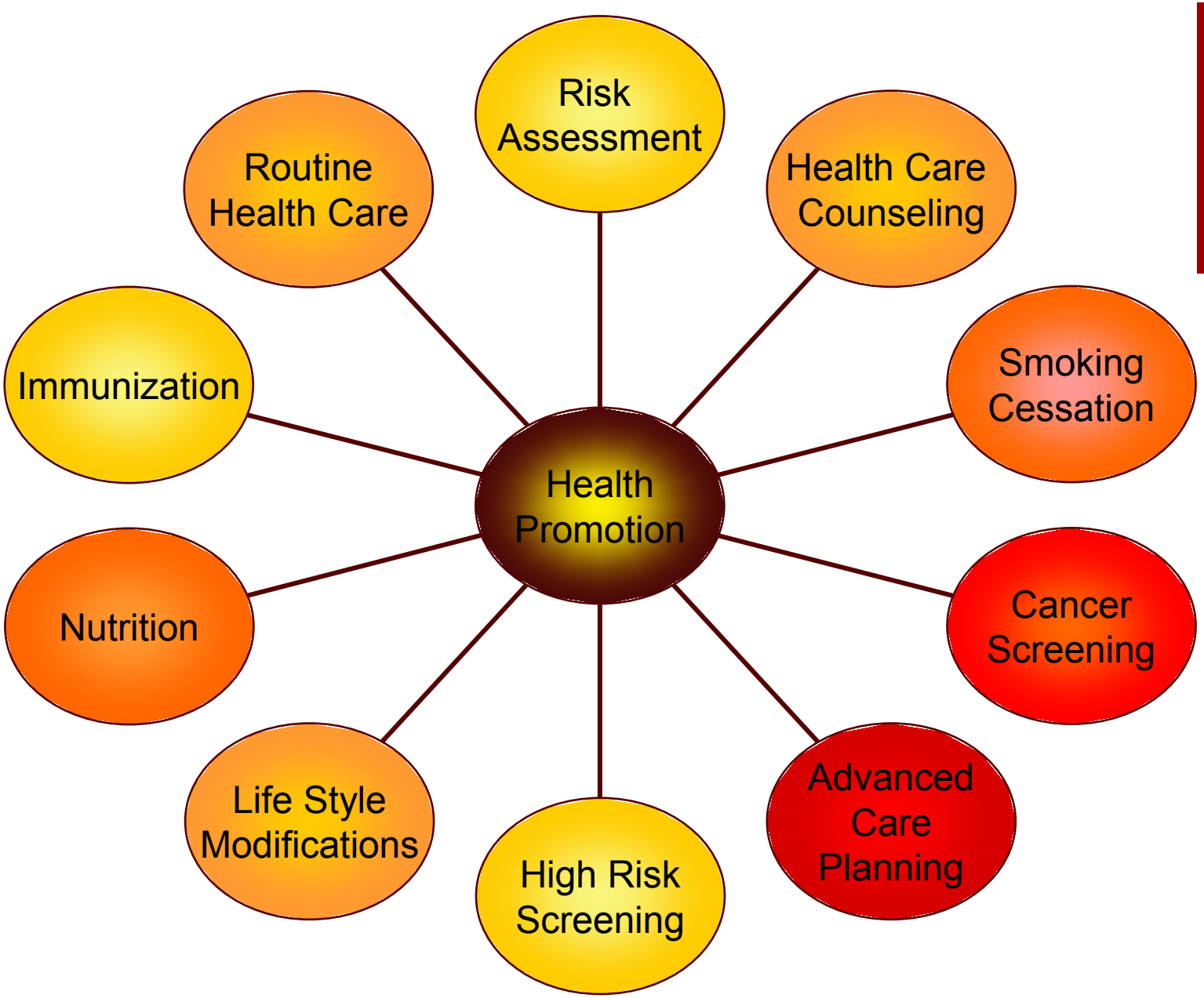
- Identifying complications of CKD
  - Mineral bone disease
  - HTN
  - Anemia of CKD
  - Malnutrition
  - Vitamin D deficiency
  - Electrolyte Imbalance – hyperkalemia
  - Metabolic acidosis

Evaluation of CKD

- Measurement of disease progression
  - Serum Creatinine and eGFR – at least annually
  - More often if:
    - Faster GFR decline (≥ 4ml/min/1.73 m2 per year
    - Risk factor for faster progression
    - Ongoing treatment to slow progression
    - Exposure to risk factors for acute eGFR decline

Management of CKD in Primary Care

The Front Line


Risk Assessment
Routine Health Care
Health Care Counseling
Immunization
Smoking Cessation
Health Promotion
Nutrition
Cancer Screening
Life Style Modifications
Advanced Care Planning
High Risk Screening

Health Care Promotion

- ✓ **Immunizations:**
  - Annual Flu Vaccine
  - Hepatitis screening and vaccination
  - Varicella
  - MMR (Measles/ mumps/ rubella)
  - Pneumonia Vaccine
  - Zoster (age appropriate)
  - Td (Tetanus/diphtheria)
  - Meningococcal

FIGURE 2. Recommended vaccinations indicated for adults based on medical and other indications[1]

VACCINE ▼ INDICATION ▶	Pregnancy	Immuno-compromising conditions (excluding human immunodeficiency virus [HIV])[4,6,7,10,15]	HIV infection CD4+ T lymphocyte count[4,6,7,10,14,15] < 200 cells/μL	HIV infection CD4+ T lymphocyte count[4,6,7,10,14,15] ≥ 200 cells/μL	Men who have sex with men (MSM)	Heart disease, chronic lung disease, chronic alcoholism	Asplenia (including elective splenectomy and persistent complement component deficiencies)[10,14]	Chronic liver disease	Kidney failure, end-stage renal disease, receipt of hemodialysis	Diabetes	Healthcare personnel
Influenza[2,*]	1 dose IIV annually				1 dose IIV or LAIV annually	1 dose IIV annually					1 dose IIV or LAIV annually
Tetanus, diphtheria, pertussis (Td/Tdap)[3,*]	1 dose Tdap each pregnancy	Substitute 1-time dose of Tdap for Td booster; then boost with Td every 10 yrs									
Varicella[4,*]	Contraindicated			2 doses							
Human papillomavirus (HPV) Female[5,*]		3 doses through age 26 yrs				3 doses through age 26 yrs					
Human papillomavirus (HPV) Male[5,*]		3 doses through age 26 yrs				3 doses through age 21 yrs					
Zoster[6]	Contraindicated			1 dose							
Measles, mumps, rubella (MMR)[7,*]	Contraindicated			1 or 2 doses							
Pneumococcal polysaccharide (PPSV23)[8,9]		1 or 2 doses									
Pneumococcal 13-valent conjugate (PCV13)[10]		1 dose									
Meningococcal[11,*]	1 or more doses										
Hepatitis A[12,*]	2 doses										
Hepatitis B[13,*]	3 doses										

*Covered by the Vaccine Injury Compensation Program

- For all persons in this category who meet the age requirements and who lack documentation of vaccination or have no evidence of previous infection; zoster vaccine recommended regardless of prior episode of zoster
- Recommended if some other risk factor is present (e.g., on the basis of medical, occupational, lifestyle, or other indications)
- No recommendation

These schedules indicate the recommended age groups and medical indications for which administration of currently licensed vaccines is commonly indicated for adults ages 19 years and older, as of January 1, 2013. For all vaccines being recommended on the Adult Immunization Schedule: a vaccine series does not need to be restarted, regardless of the time that has elapsed between doses. Licensed combination vaccines may be used whenever any components of the combination are indicated and when the vaccine's other components are not contraindicated. For detailed recommendations on all vaccines, including those used primarily for travelers or that are issued during the year, consult the manufacturers' package inserts and the complete statements from the Advisory Committee on Immunization Practices (www.cdc.gov/vaccines/pubs/acip-list.htm). Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

Hypertension Management

- **Core Concepts of Treatment**

- Hypertension and proteinuria (albuminuria) are both independent variables that predict long-term decline in renal function

➢ Renal disease is both a cause and consequence of hypertension

➢ Reduction of blood pressure reduces cardiovascular risk and renal risk

➢ Reduction of proteinuria (albuminuria) may lower both cardiovascular and renal risk.

JNC VII Classification of BP

- Classification of Blood Pressure:

Category	SBPmmHg		DBPmmHg
Normal	<120	and	< 80
Pre-hypertension	120-139	or	80-89
Hypertension, Stage 1	140-159	or	90-99
Hypertension, Stage 2	≥ 160	or	≥ 100

http://www.hlbi.nih.gov/guidelines/hypertension

Hypertension

- The progression of CKD is strongly linked to hypertension control.
- Patients being treated with an angiotensin-converting enzyme (ACE) inhibitor have more effective preservation of renal function at similar levels of blood pressure reduction. This effect was most profound in those patients with the highest levels of baseline proteinuria.
- The reduction in systemic hypertension slows or prevents progression of proteinuric and nonproteinuric renal disease.
- treating isolated systolic hypertension in older patients slows the progression of CKD.
- ACE inhibitors are more protective, particularly in proteinuric disease. In patients who cannot tolerate ACE inhibitors, an angiotensin receptor blocker (ARB) may reasonably be prescribed.
- JNC 7 Guidelines: <130/80 mm Hg for patients with diabetes or chronic kidney disease

Diagnostic Workup of Hypertension

- Assess risk factors and co-morbidities
- Reveal identifiable causes of hypertension
- Assess presence of target organ damage
- Conduct history and physical examination
- Obtain laboratory tests:
- Obtain EKG

http://www.hlbi.nih.gov/guidelines/hypertension

Anti-Hypertensive Drugs: Sites of Action

Blood Pressure = **Cardiac Output** X **Total Peripheral Resistance**

β-Blockers

CCBs*

Diuretics

→

ACE Inhibitors
AT_1 Blockers
a-Blockers
a_2-Agonists
CCBs
DA_1 Agonists
Diuretics
Sympatholytics
Vasodilators

* = non-dihydropyridine CCBs

Indications for individual drug classes

Heart Failure	THIAZ, BB, ACEI, ALDO ANT
Post Myocardial Infarction	BB,ACE, ALDO ANT
High CVD Risk	THIAZ, BB, ACEI, CCB
Diabetes	THIAZ, BB, ACEI, ARB, CCB
Chronic Kidney Disease	ACEI, ARB
Recurrent Stroke Prevention	THIAZ, ACEI

http://www.hypertensiononline.org

Average Number of Anti-Hypertensive Agents Used to Achieve Target BP

	MDRD	ABCD	HOT	UKPDS
Goal BP	<92 mmHg MAP*	<75 mmHg DBP	<80 mmHg DBP	<85 mmHg DBP
Achieved BP	93	~75	81	82
Avg # of drugs per patient	3.6	2.7	3.3	2.8

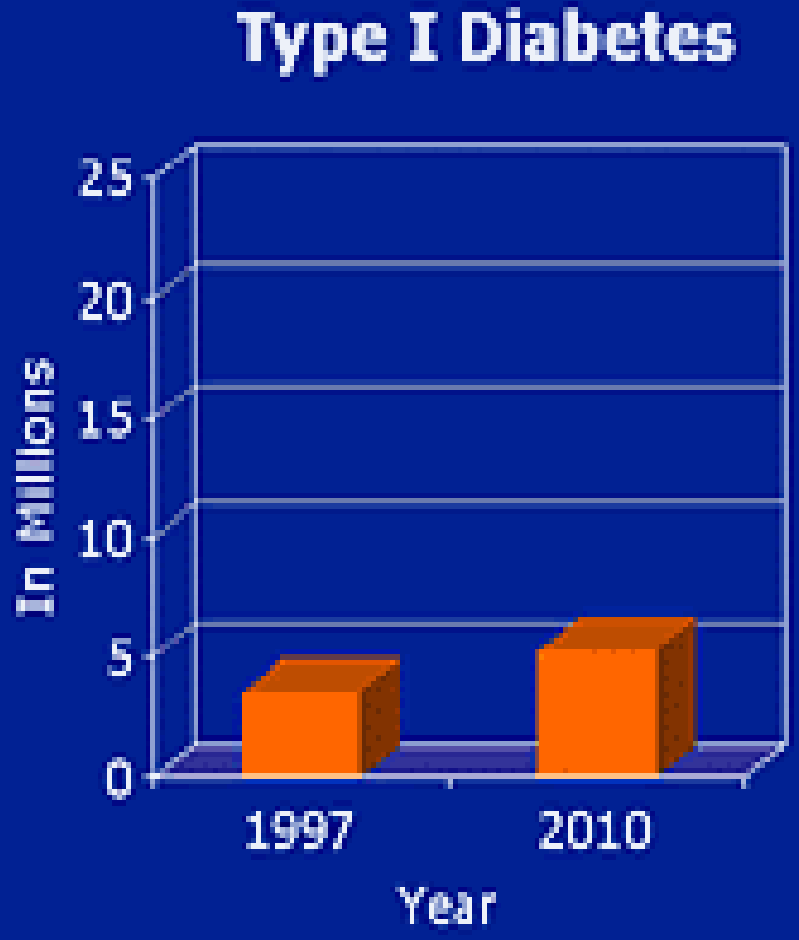
*The goal mean arterial pressure (MAP) of <92 mmHg specified in the MDRD trial corresponds to a systolic/diastolic blood pressure of approximately 125/75 mmHg.

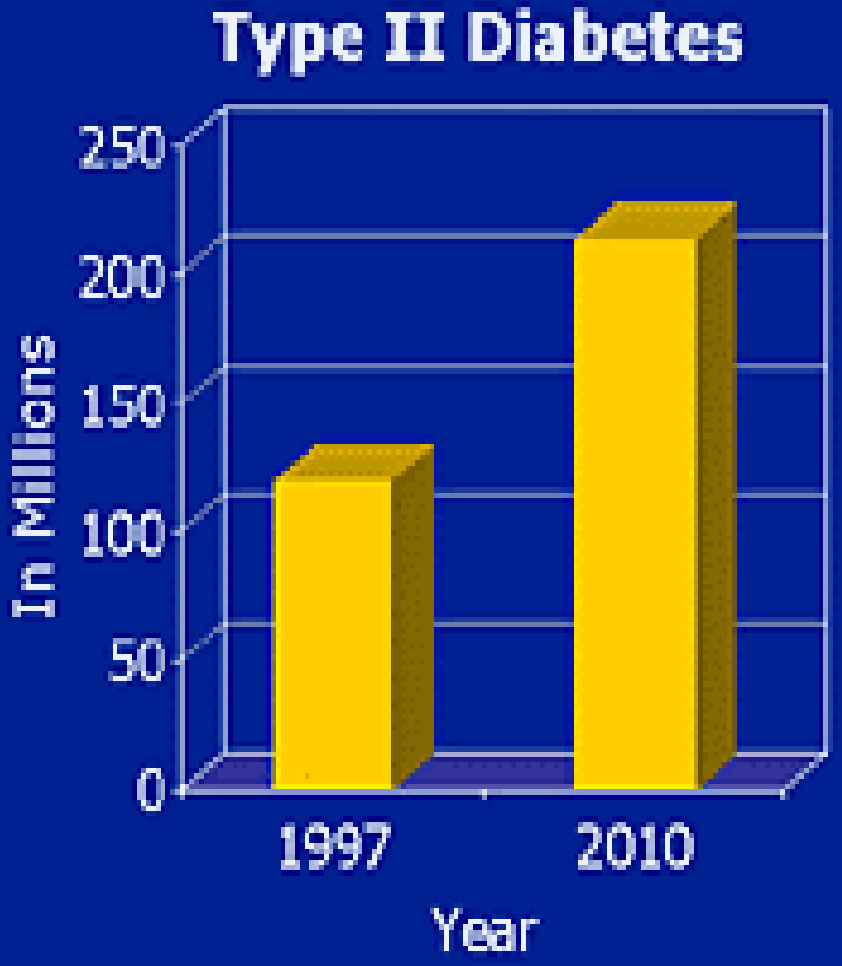
The therapeutic goal of reducing the blood pressure to target, regardless of agent used, should not be sacrificed.

Diabetes

- #1 Cause of Chronic Kidney Disease
- If one improves glycemic control, can lead to decreased rates of retinopathy & neuropathy
- Fewer long-term microvascular complications
- Reduction in Cardiovascular disease
- Goal HgbA1C <7%
- Pre-prandial 90-130
- Post-prandial<180

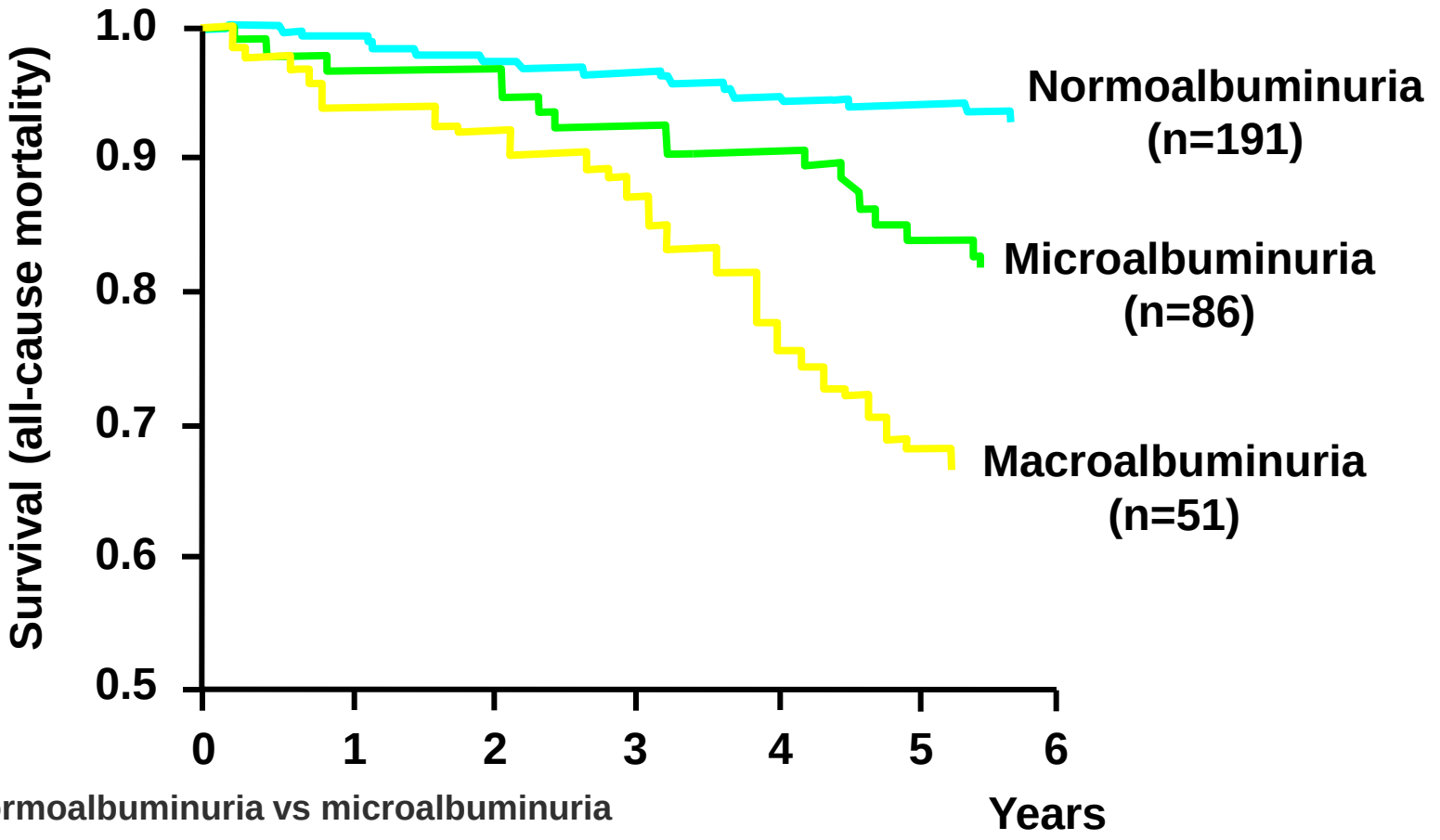
Global Estimates and Projections for Incidence of Diabetes Mellitus





Amos A, McCarty D, Zimmet P.
Diabetes Medicine. 1997;14[Suppl5]:81-85.

Proteinuria as a Risk Factor for Mortality in Type 2 Diabetes



P<0.01 normoalbuminuria vs microalbuminuria

P<0.001 normoalbuminuria vs macroalbuminuria

P<0.05 microalbuminuria vs macroalbuminuria

Gall MA, et al. Diabetes. 1995;44:1303-1309.

Goals for Diabetes and CKD

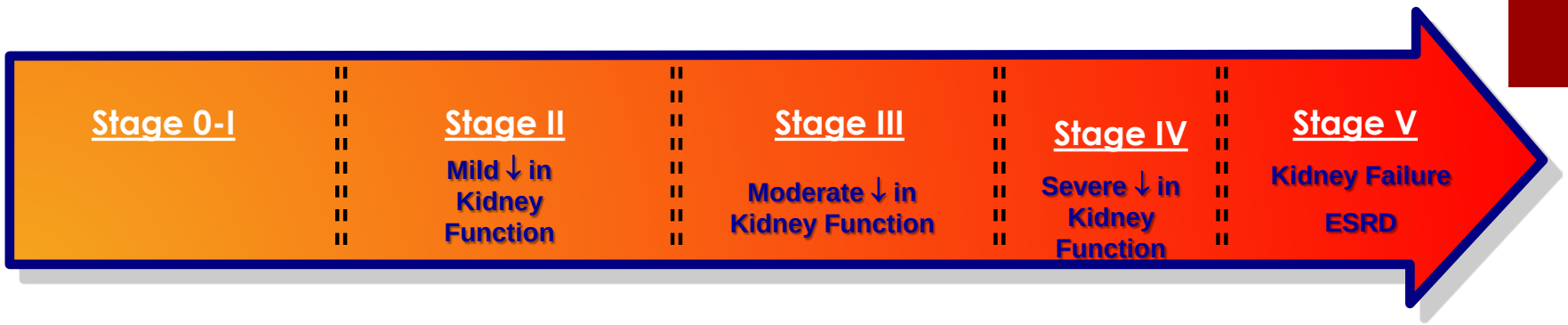
- ✓ Glycemic control for type 1 and type 2
- ✓ Monitoring for Urinary Albumin Excretion
- ✓ Managing the Cardiovascular Risk Factors
- ✓ General Health Screening for diabetics – eye/foot exams, dental care
- ✓ Note:

  35% of Type 1 or 2 will develop nephropathy

  45% of pts starting dialysis have diabetic nephropathy

  2007 USRDS projects increase of ESRD > 50% by 2020

When to Consult?



When to Consult

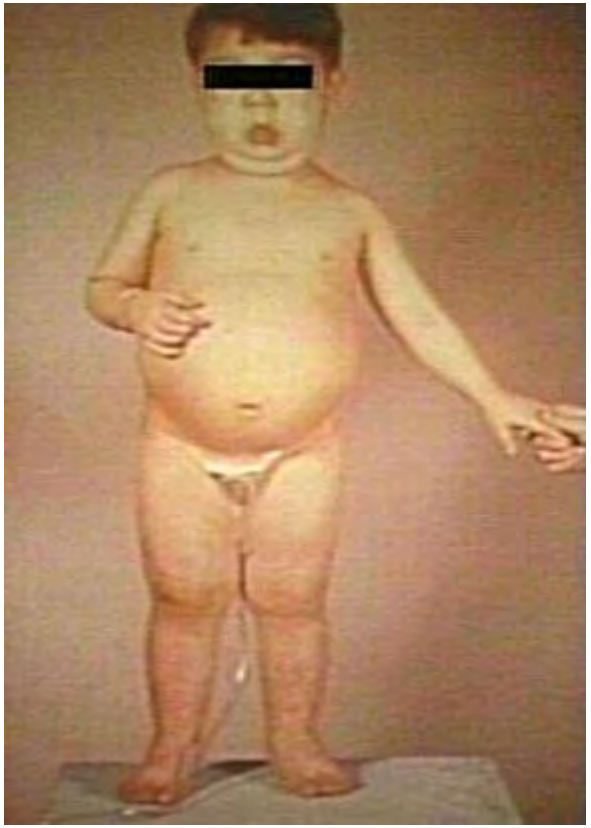
- Patients that require assessment for complications of decreased GFR including
  - Moderate (Stage 3) or severely (stage 4) decreased GFR
- Stage 1-2 – Consider referral for following conditions:
  - Proteinuria - >1000 mg/d
  - Refractory HTN
  - Clarification of diagnosis
  - Unexplained acute decrease in eGFR

Role of the Nephrologist

- The First Goal of the Nephrologist:

a. Establish or Confirm a Diagnosis

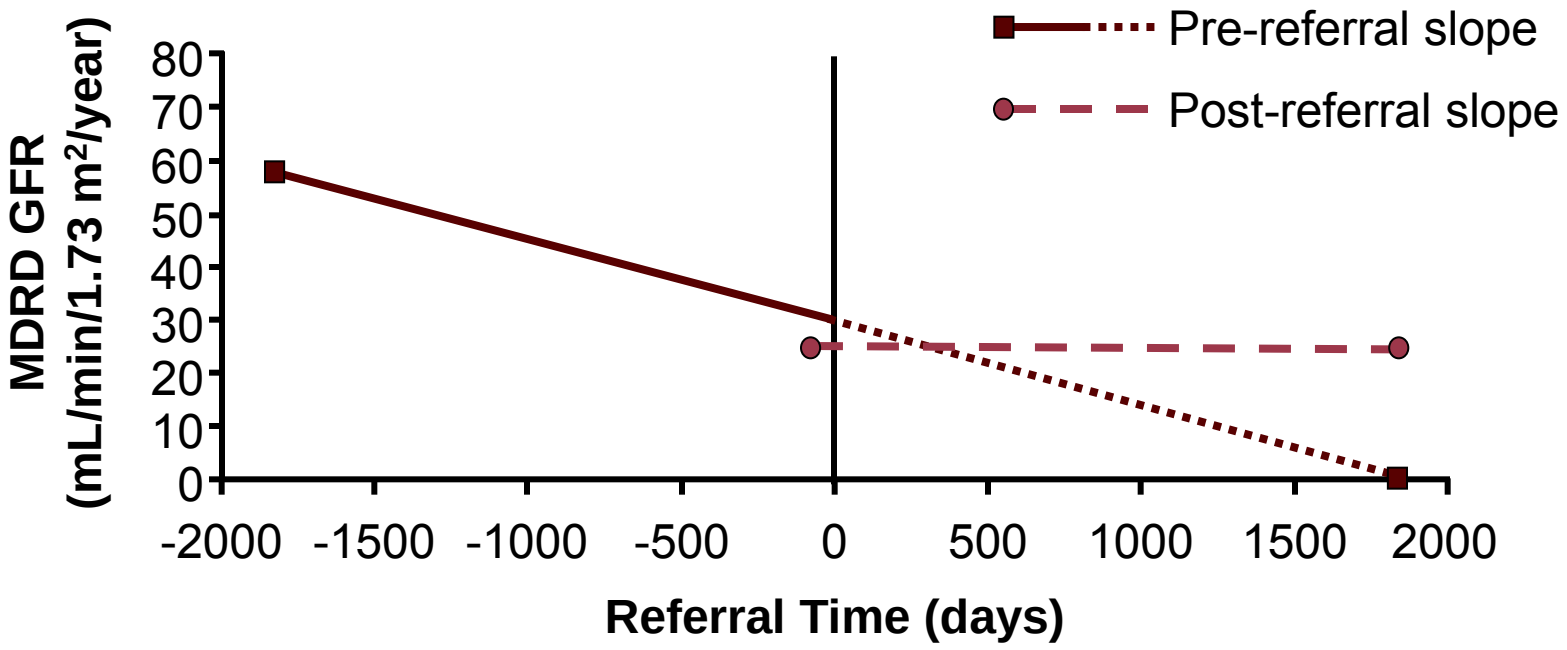
b. Determine the rate of progression.[1]



1. Mendelssohn, DC etal: Elevated levels of serum creatinine: Recommendations for management and referral. Can Med Assoc J 1999; 161(4):413-417.

Benefits of Early Nephrology Referral

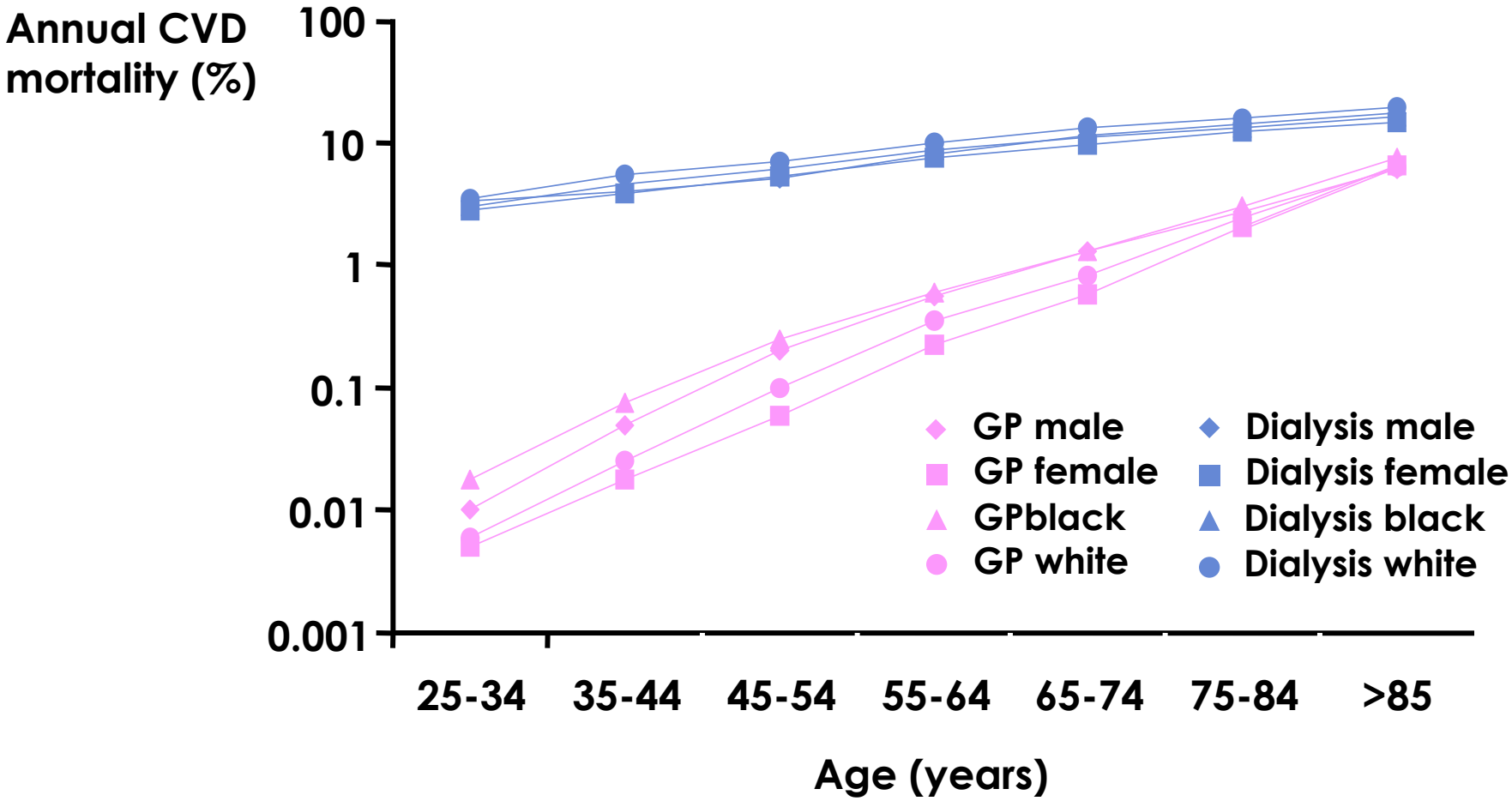
Retrospective observational study of 726 new referrals with stage 3-5 CKD, to a single renal unit in Southampton, UK. Study participants were a mean age of 72 years.



A slower rate of GFR decline was associated with a survival benefit

Adapted from Jones C, et al. *Nephrol Dial Transplant.* 2006:21;2133-2143.

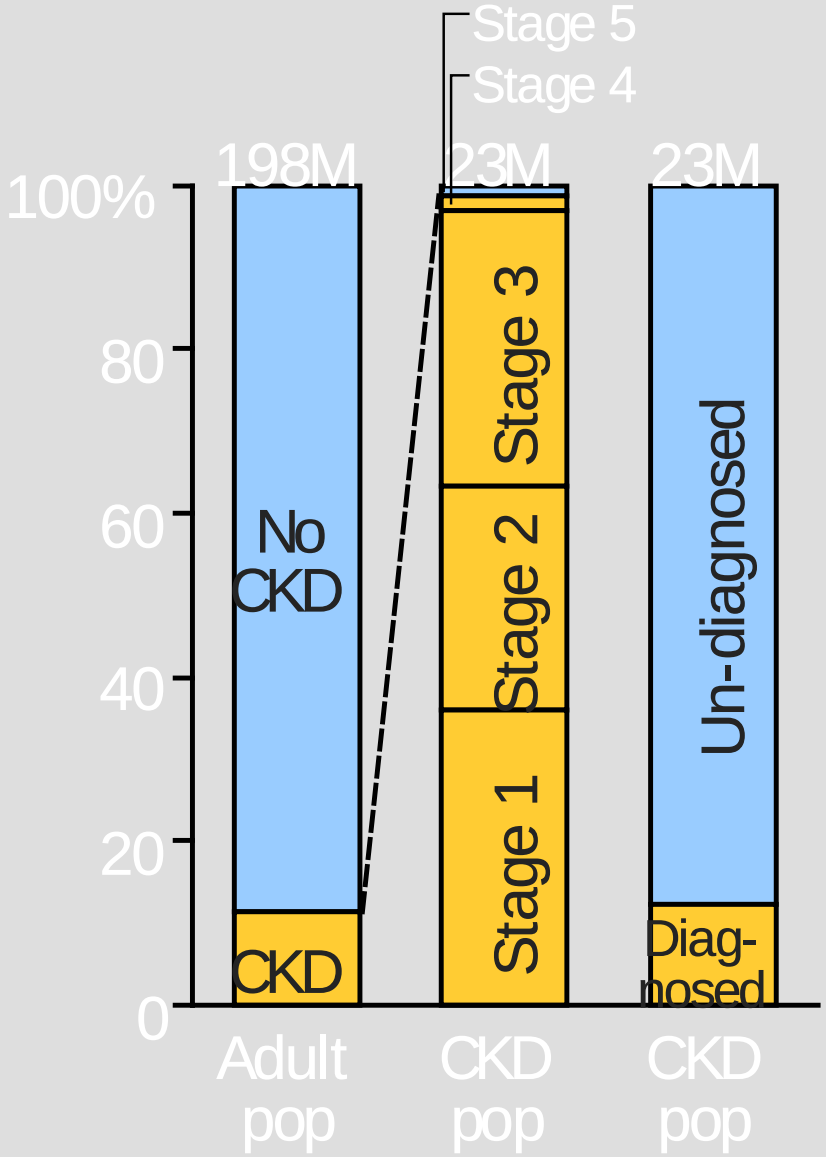
Cardiovascular Disease (CVD) Mortality General Population versus ESRD Patients



GP=General Population
ESRD=End-Stage Renal Disease

Foley RN, Parfrey PS, Sarnak MJ. *Am J Kidney Dis*. 1998;32(suppl):S112-S119.

Broader base: Still large "ghost" population



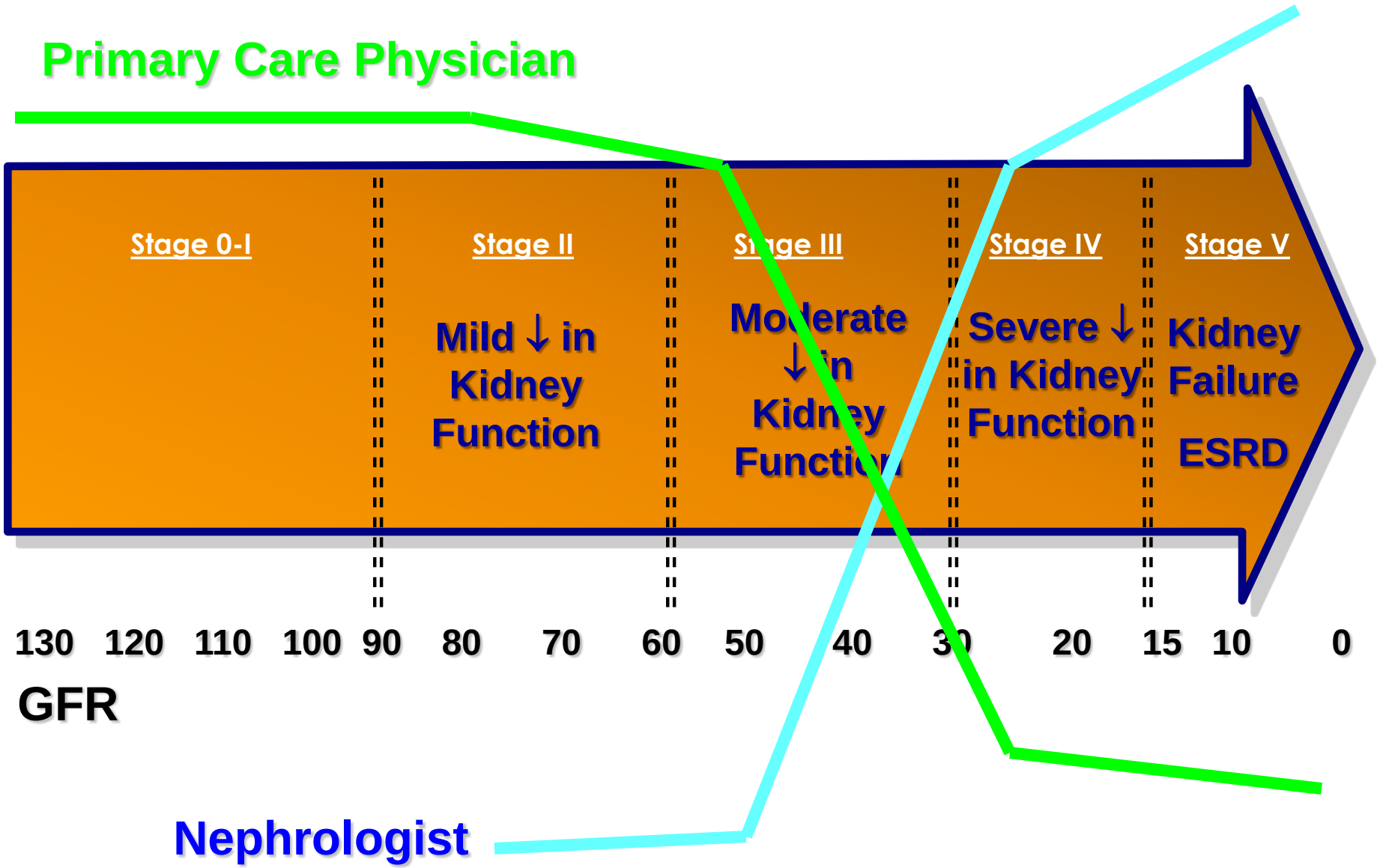
Avg of ~5 months between diagnosis and kidney failure

	Early Diagnosis/ Referral		Late Diagnosis/ Referral
Average Hospital Costs	$18K	⇔	$44K
Average days in hospital	8.6	⇔	17.0
5-Yr Mortality	23%	⇔	41%

Source: K/DOQI, National Kidney Foundation; Analyst reports; Expert interviews; "The timing of referral of patients with end-stage renal disease", *Dial Transplant* 1989; "Chronic Kidney Disease, the silent epidemic" *Hospital Physician* March, 2003

- CKD carries a significant burden of morbidity and mortality.
- Care of patients with CKD requires a team approach
- Managing co-morbidities e.g., hypertension, diabetes, hyperlipidemia should occur early at stages 1 to 3 CKD. Continued by the primary care provider, with assistance from the Nephrologist.
- The strongest evidence for the importance of referral to a Nephrologist is for patients with CKD stages 4 and 5 (GFR <30 mL/min/1.73 m^2).

Co-Management of CKD



Complications of CKD/Secondary Diagnosis

- Glomerulonephropathy
- Anemia secondary to Chronic Kidney Disease
- Renal Bone Mineral Management
- Acid/Base Disorders
- Electolyte Imbalances
- Hyper/Hypovolemia
- Nutritional Deficiencies

Clinical Manifestations of Glomerular Disease

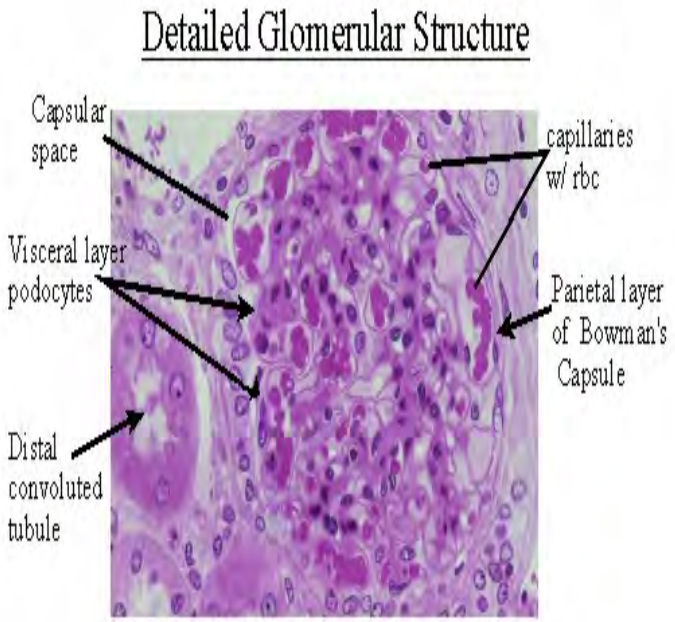
- Asymptomatic Proteinuria
- Nephrotic Syndrome (proteinuria, hypoprotienemia, hyperlipidemia, edema)
- Asymptomatic Hematuria
- Glomerulonephritis (hematuria, proteinuria, renal failure, hypertension)
- Acute Glomerulonephritis (nephritis with short-term renal failure)
- Crescentic Glomerulonephritis (nephritis with rapidly progressive renal failure)
- Chronic Glomerulonephritis (chronic progression of renal failure)
- End Stage Renal Disease (irreversible renal failure)

Causes of Glomerular Diseases

- Diabetic Nephropathy- leading cause, elevated glucose levels scars the kidney, expected that the elevation increases blood flow through the kidneys, thus putting a strain on the filtering system and raising blood pressure.
- Direct result of an infection- post-streptococcal glomerulonephritis can occur after an episode of strep throat, occurs by the immune system overproducing antibodies.
- Drug toxic to the kidneys
- Systemic Lupus- ? Sex-linked genetic factor, autoantibodies form or are deposited in the glomeruli.
- Autoimmune- creation of autoantibodies that attack the body itself, could be specific organs or regions of body.
- Idiopathic

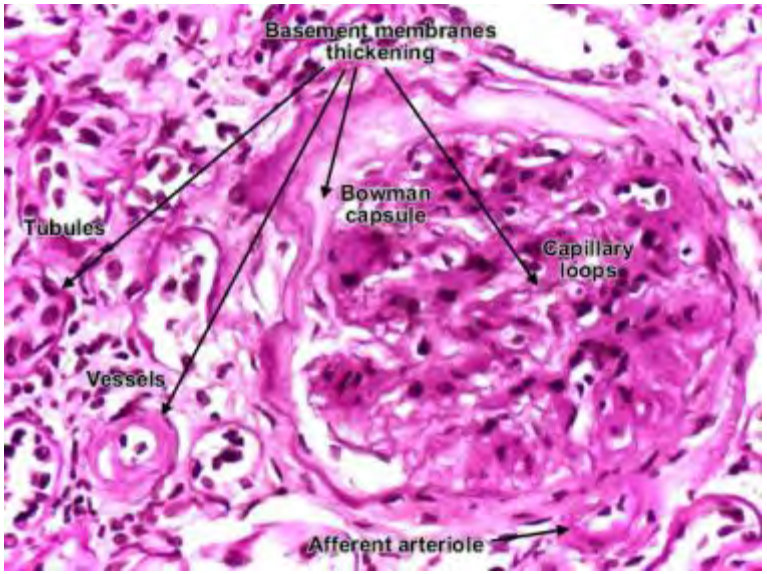
Glomerulonephritis

- Inflammation of the membrane tissue in the kidney that serves as a filter, separating wastes and extra fluid from the blood.
- Damage to the glomeruli occurs letting protein and occasionally blood cells leak into the urine.
- Interference with clearance of waste products occurs which leads to build up of uremic condition in the blood.



http://kidney.niddknih.gov/kudiseases/pubs/glomerular/index.htm

Diabetic Nephropathy



- Seen in patients with Diabetes Mellitus (types I,II)
- Most common cause of ESRD in adults
- Very high frequency in Native Americans
- Earliest clinical manifestation – asymptomatic proteinuria
- Increased abnormal glycosylation of IgG
- Therapy – ACE, Tx with DM drugs, BP maintenance, dietary restrictions.

http://kidney.niddknih.gov/kudiseases/pubs/glomerular/index.htm

Glomerulonephropathies

- **Nephrotic Syndromes**

  Minimal Change Glomerulopahty

  Focal Segmental Glomerulosclerosis

  Diabetic Glomerulosclerosis

  Membranous Glomerulopathy

- **Nephritic Syndromes**

  Glomerulonepritis – Three mechanisms are incriminated as the major pathogenic mediators of glomerulonephritis

  1. Circulating Immune Complex (Lupus, Post infectious GN and IgA nephropathy)

  2. In-Situ – localization of the immune complex (Anti-GBM disease)

  3. Anti-neutrophil cytoplasmic antibodies (ANCA)

- **Primary FSGS**

  - Children and Adults

  - Mostly idiopathic

  - Hereditary (related to problems with podocytopathy

- **Secondary FSGS**

  -Hypertension

  - Obesity

  - Sickle Cell Disease

  - Hypoxic States

  - HIV infection (other viral infections – Parvo B19)

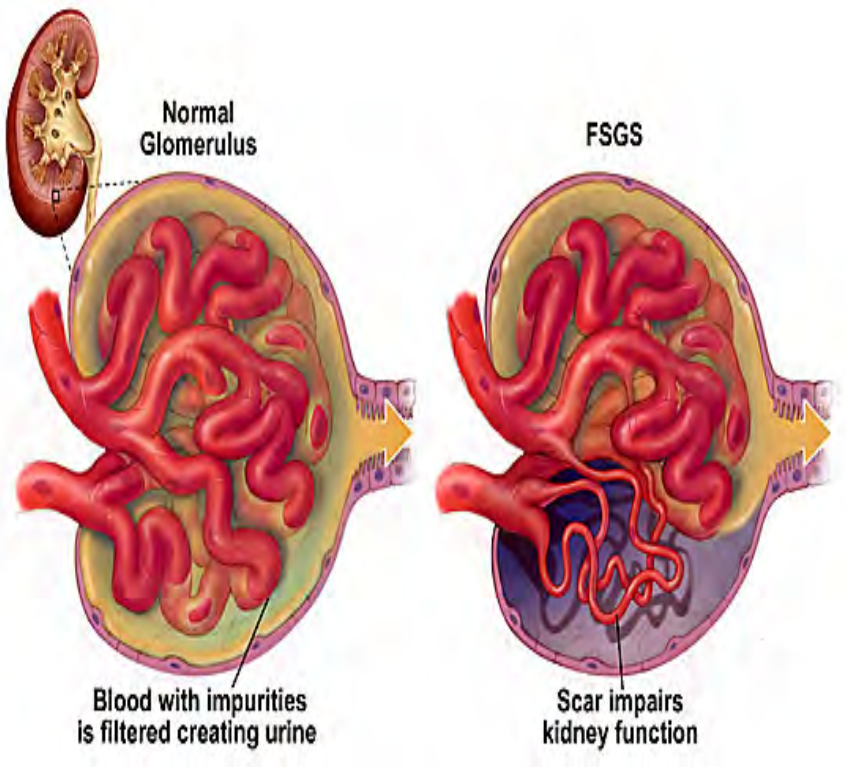
  - IV Drug, heroin – induced

  - Congenital or acquired reduction of renal parenchyma

  - Reflux nephropathy

  - Steroids (anabolic)

  - Lithium



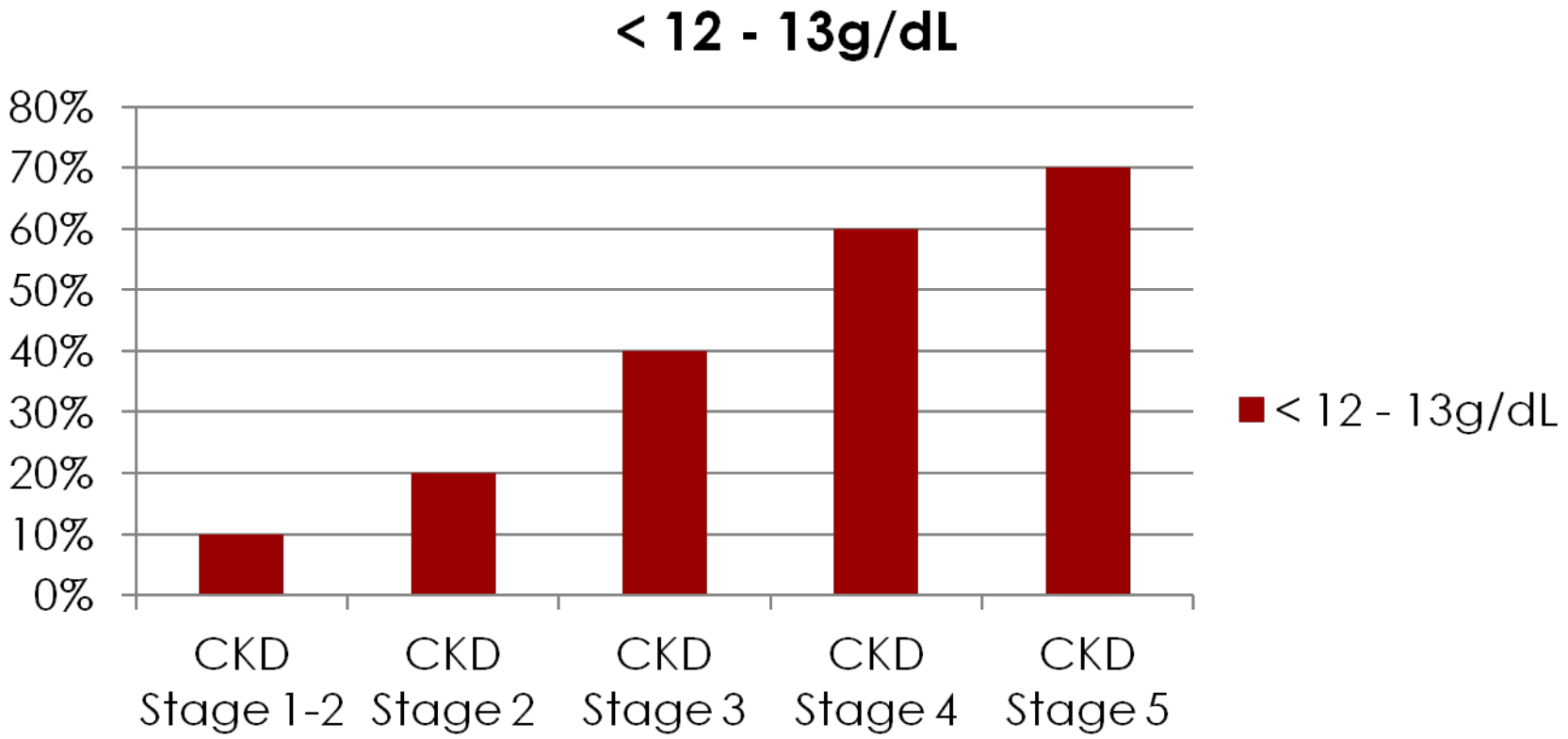
Diagnosis and Initial Assessment of Anemia in CKD

- Prevalence of anemia in CKD depends on both the CKD Stage and the definition of Anemia
- World Health Definition: Hbg< 13.0 g/L Adult Men/Women (non-Menstr) Hbg < 12.0g/L Women
- A Hemoglobin < 10g/dl or Hematocrit < 30% and a reduced CrCl < 40ml/min. This is the standard for the definition of anemia in CKD (CMS reimbursement rules tend to drive the therapy)

- Assess for other causes of Anemia
  - Myelodysplastic syndromes
  - Iron Deficiency
  - GI Blood Losses
  - Folic Acid or Vitamin B 12 deficiency
  - Hemolysis

National Kidney Foundation. K/DOQI clinical practice recommendations for anemia in chronic kidney disease: 2007 Update of hemoglobin target. *Am J Kidney Dis*. 2007;50: 471-530.
McFarlane SI, Chen SC, et al. Prevalence and associations of anemia of ckd: Kidney early evaluation program (KEEP) and National health and nutrition examination survey (NHANES) 1999-2004. Am. J Kidney Dis. 2008; 51: s46-s55.

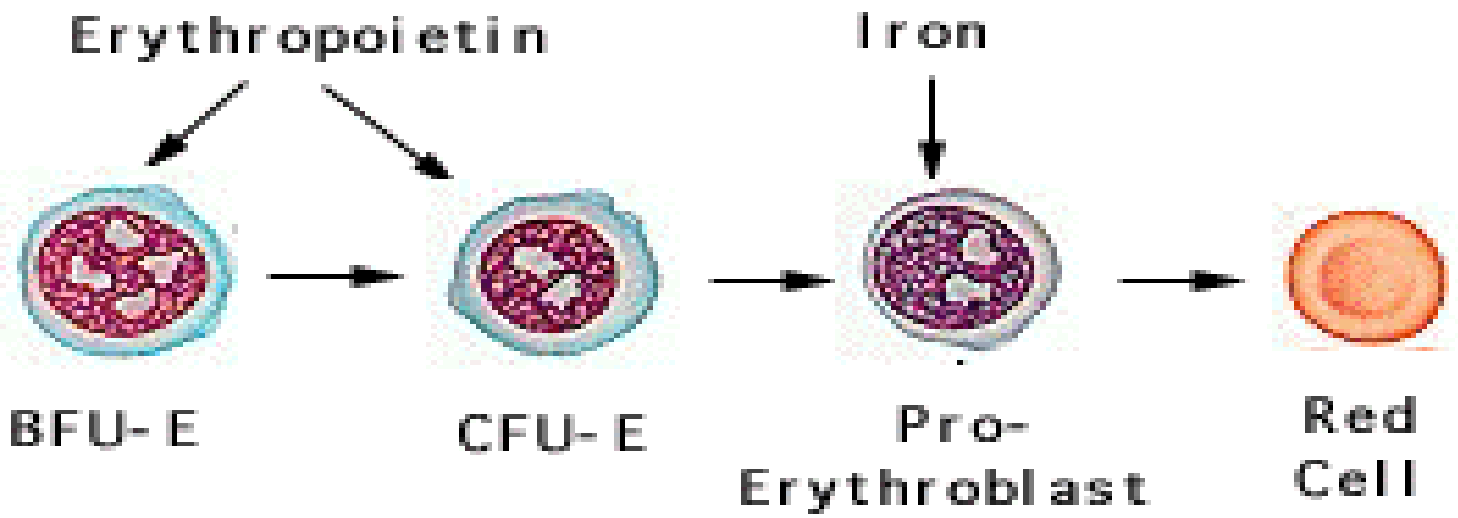
Anemia Starts Early in CKD



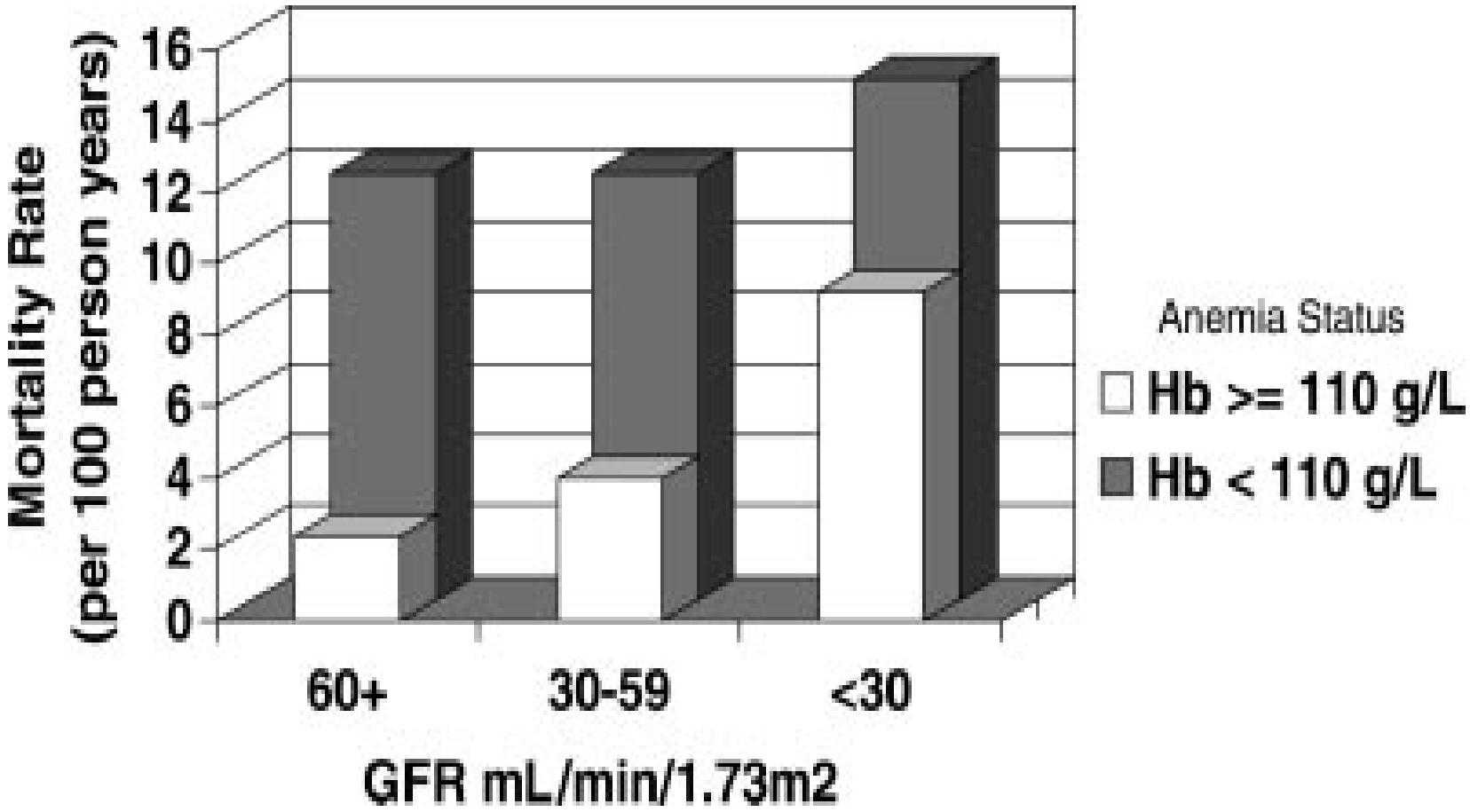
National Kidney Foundation. K/DOQI clinical practice recommendations for anemia in chronic kidney disease: 2007 Update of hemoglobin target. *Am J Kidney Dis*. 2007;50: 471-530.

- Characterized by the reduced production of erythrocytes with cells of normal size and shape.

Figure 3



Increased Risk of Mortality in Older Adults



Mortality rate (per 100 person-years) by stage of kidney function and the presence or absence of anemia (adjusted for age and sex).

Culleton, BF, et al. bloodjournal.hematologylibrary.org, 2006 107: 3841-3846.

Benefits Associated with Anemia Correction in CKD and ESRD

↓ Transfusion Requirements

↑ Quality of Life

↑ Cardiac Function

↑ Cognitive Function

↑ Exercise Capacity and Physical Performance

↓ Hospitalization

↔ Mortality

National Kidney Foundation. K/DOQI clinical practice recommendations for anemia in chronic kidney disease: 2007 Update of hemoglobin target. *Am J Kidney Dis*. 2007;50: 471-530.
McFarlane SI, Chen SC, et al. Prevalence and associations of anemia of ckd: Kidney early evaluation program (KEEP) and National health and nutrition examination survey (NHANES) 1999-2004. Am. J Kidney Dis. 2008; 51: s46-s55.
Culleton, BF, et al. bloodjournal.hematologylibrary.org, 2006 107: 3841-3846.

Hemoglobin is a more Accurate Measure of Anemia than Hematocrit

- Hemoglobin (Hgb)
  - Directly Measured
  - Standard for Quality Control
- Hematocrit (Hct)
  - Indirectly Measured
  - Calculated Value in Automated Equipment (MCV X RBC Count)
  - No comparable Standard of Calibration for Hct as compared with Hgb

National Kidney Foundation. K/DOQI clinical practice recommendations for anemia in chronic kidney disease: 2007 Update of hemoglobin target. *Am J Kidney Dis*. 2007;50: 471-530.

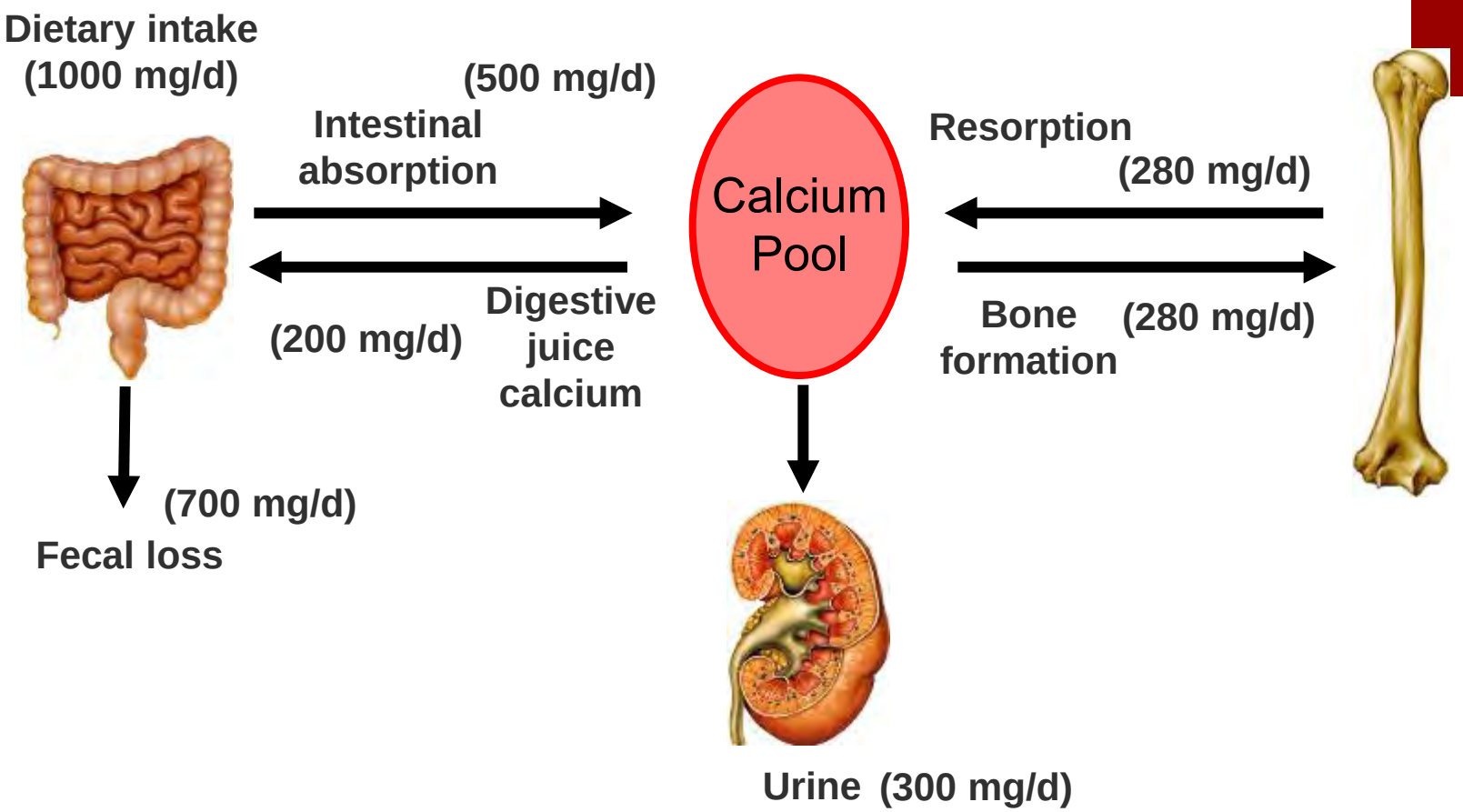
Bone Mineral Management in CKD (Renal Osteodystrophy)

- Derangements occurs with CKD in the balance of Calcium, Phosphorous, Parathyroid Hormone (PTH) and Calcitriol (1,25(OH)2D
- Occurs quickly and early in CKD (before the initiation of dialysis/ESRD).

Results in:

- Abnormal serum concentrations of Calcium & Phosporous
- Impaired Bone Remodeling
- Increased of fractures
- Extraskeletal Manifestations

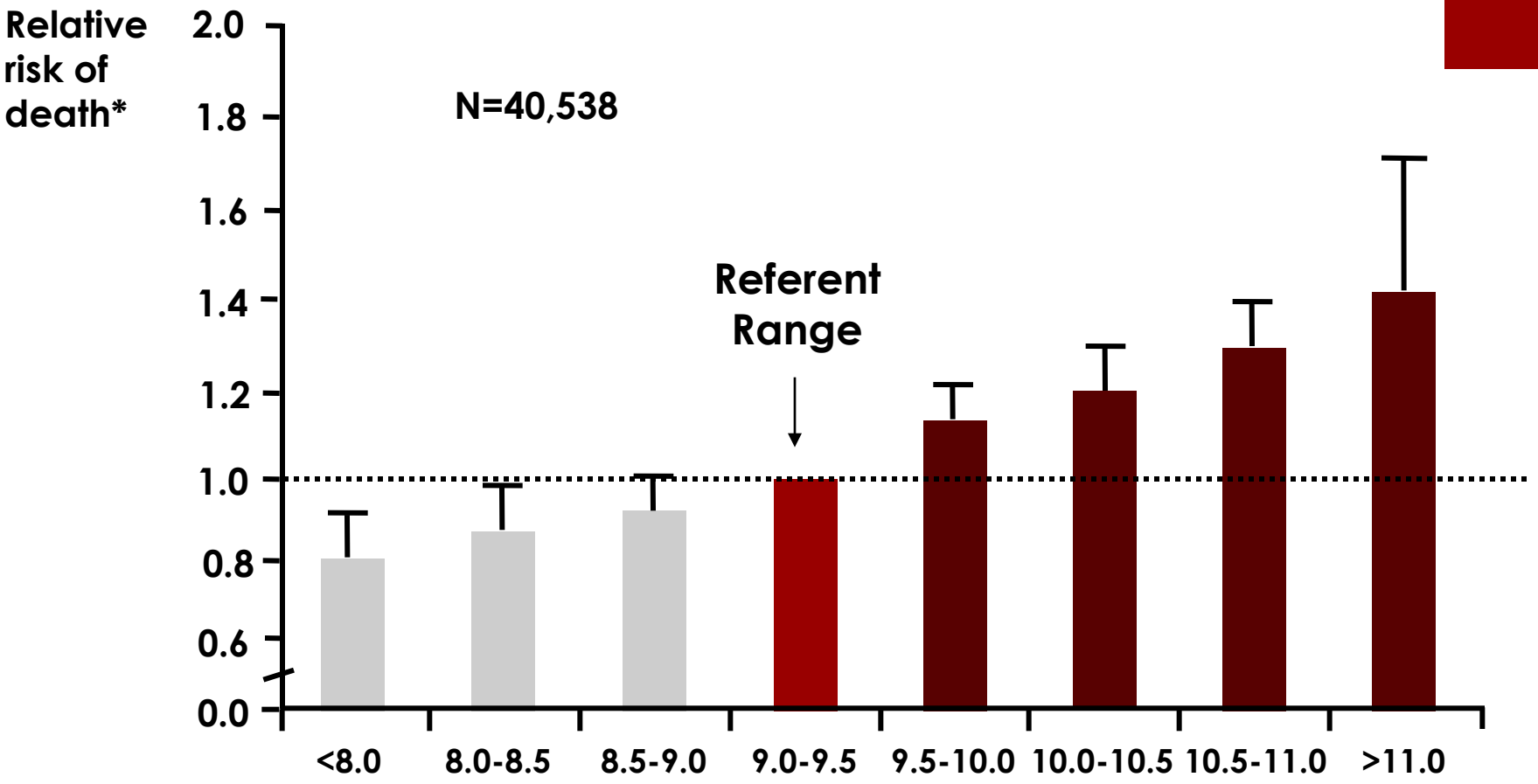
Calcium Metabolism



1000 mg Input = 700 mg Fecal loss + 300 mg Renal excretion
Net balance = 0

Adapted from Nordin BEC. *Calcium, Phosphate, Magnesium Metabolism*. Churchill Livingstone. 1976.

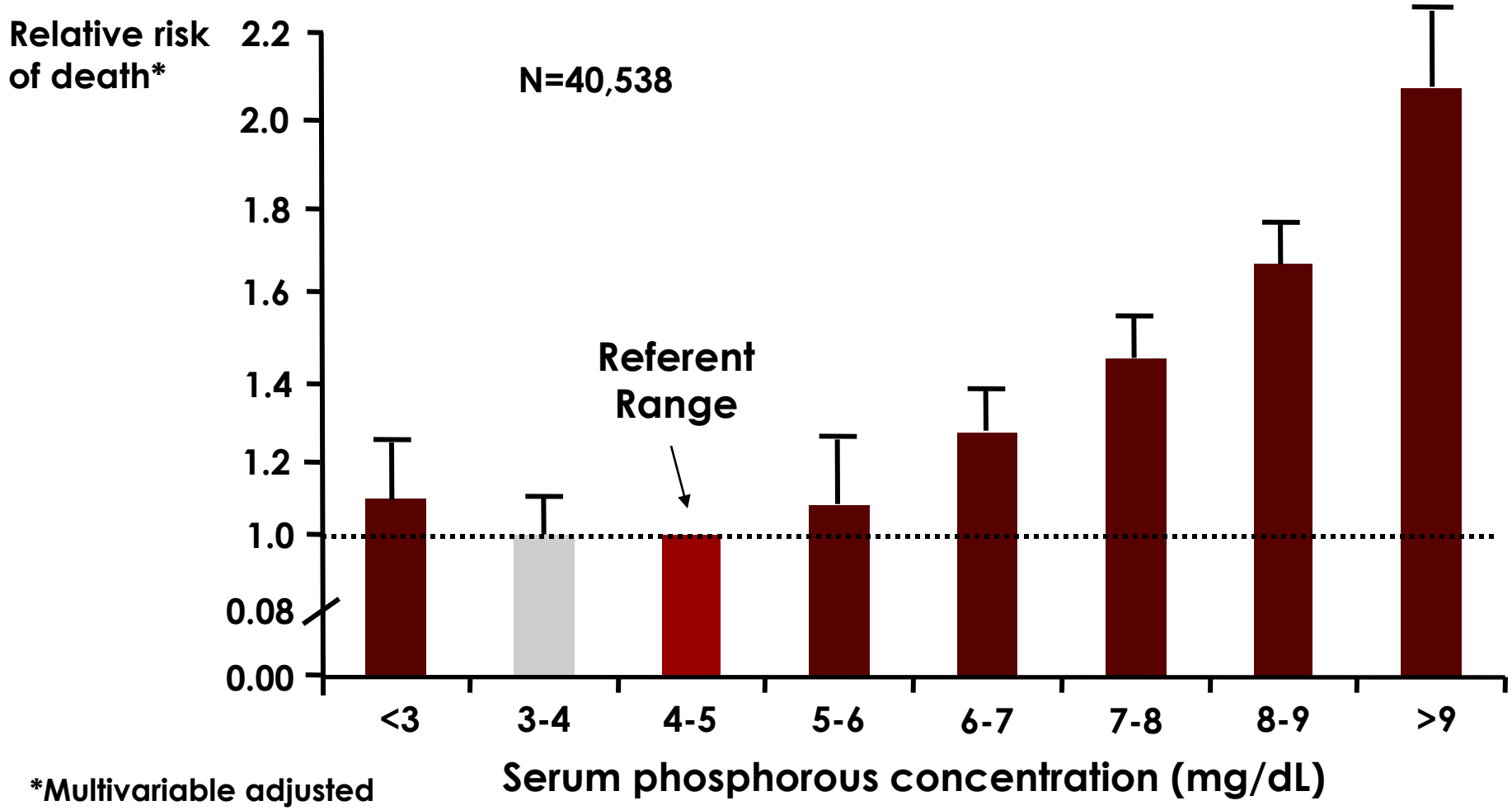
Elevated Serum Calcium Increases Mortality Risk



*Multivariable adjusted

Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. *J Am Soc Nephrol*. 2004;15:2208-2218.

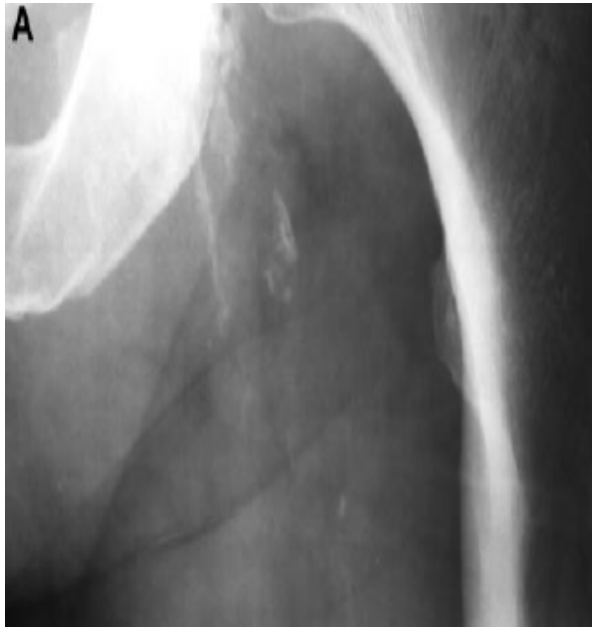
Elevated Serum Phosphorus Increases Mortality Risk



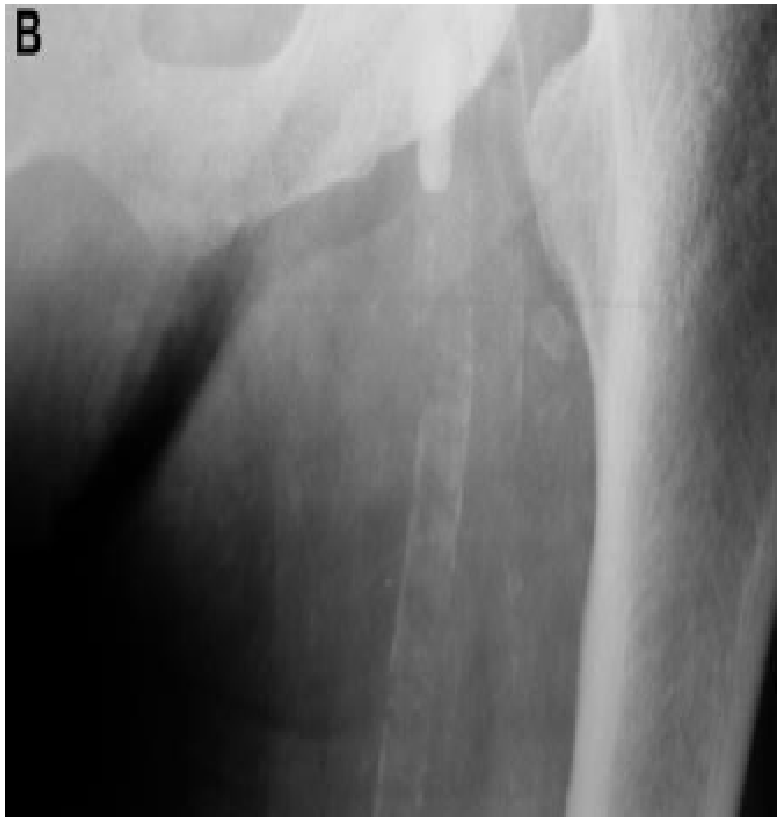
*Multivariable adjusted

Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. *J Am Soc Nephrol*. 2004;15:2208-2218.

Arterial Calcification in End-Stage Renal Disease:



Intimal Plaques

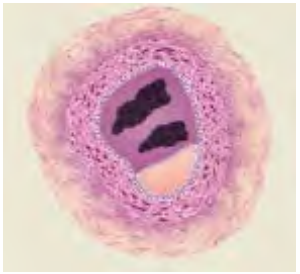


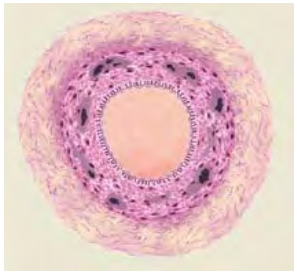
Medial Calcifications

London GM, Guerin AP, Marchais SJ, Metivier F, Pannier B, Adda H. *Nephrol Dial Transplant*. 2003;18:1731-1740.

Arterial Media Calcification in ESRD: Impact on All-Cause and Cardiovascular Mortality

Arterial Intimal Calcification





Arterial Medial Calcification

- usually observed in…
  - older patients with a clinical history of atherosclerosis before starting HD
  - those with typical risk factors associated with atherosclerotic disease

- usually observed in…
  - young and middle-aged patients without conventional atherosclerotic risk factors
  - associated with
    - duration of HD
    - calcium-phosphate disorders
    - oral dose of elemental calcium prescribed as a phosphate binder ($CaCO_3$)

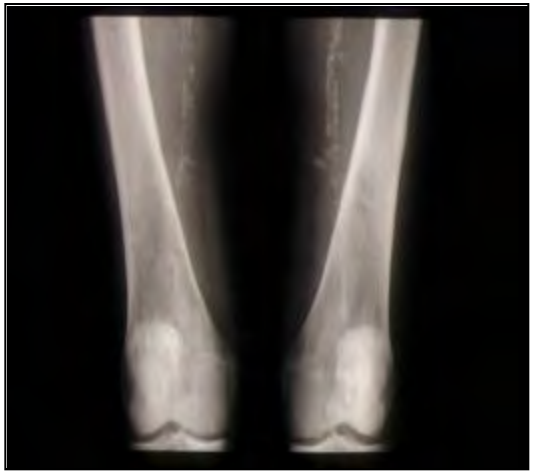
N=202
ESRD=End-Stage Renal Disease
HD=Hemodialysis

For illustrative purposes only

London GM, Guerin AP, Marchais SJ, Metivier F, Pannier B, Adda H. *Nephrol Dial Transplant*. 2003;18:1731-1740.

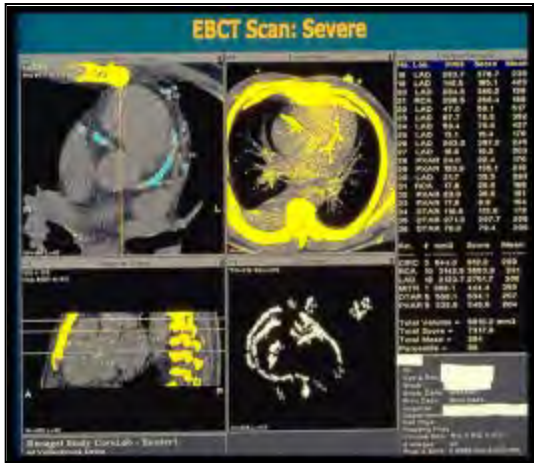
How to Assess Vascular Calcification

Blood Pressure

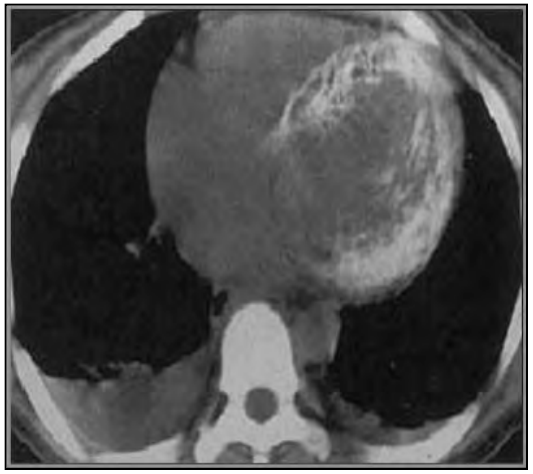


Plain Film

Ultrasound



EBCT



Spiral CT

Pulse Pressure

Cardiovascular Mortality in CKD: Summary

- Risks of CVD and CV calcification are far greater in patients with CKD on dialysis than in the general population
  - Arterial calcification increases mortality risk
  - CV mortality rate is 10-20x greater in dialysis patients
  - All dialysis patients should be evaluated for vascular calcification
- Factors associated with coronary artery calcification include
  - Serum P
  - Ca intake from binders
  - Ca x P product
  - Duration of dialysis
  - Age

Acid Base

- The kidneys' ability to regenerate bicarbonate consumed in buffering the daily net acid production diminishes as nephron mass decreases.
- As GFR Declines, the anion gap widens
- Buffering of excess hydrogen ions occurs in bone
- Chronic acidosis leads to muscle protein breakdown and reduced albumin synthesis.
- The goal of therapy is to maintain the serum bicarbonate concentration at or above 22 mEq/L
- Avoiding the deleterious effects of acidosis on bone histology and protein catabolism.

Bailey JL, et al. The acidosis of chronic renal failure activates muscle proteolysis in rats. J. Clin. Invest 1996; 97: 1447-1453.
Alpem RJ, Sakhaee K. The clinical spectrum of chronic metabolic acidosis, homeostatic mechanisms produce significant morbidity. Am. J. Kidney Dis 1997; 29-291
Rose BD. Pathogenesis and treatment of metabolic acidosis in chronic kidney disease. 2011; www.uptodate.com

Electrolyte Imbalances

- Hyperkalemia generally develops in oliguric patients with a GFR lower than 10 mL/min/1.73 m^2.
- Hypokalemia can develop with aggressive diuresis, Malnutrition or hyperfiltration.
- Calcium/Phosphorous – development in later stages of CKD
- Dietary restriction is the mainstay of management of chronic hyperkalemia in these patients.
- Dietary Supplementation and correction of malnutrition for the management of hypokalemia.

Hyper/Hypovolemia in CKD

- Sodium and intravascular volume balance are usually well maintained until the GFR falls below 15 mL/min/1.73.
- Less than 6 g/day of sodium chloride (<2 g/day of sodium) is the typical initial recommendation.
- Reminder: Adjustments need to be made depending on the patient's volume status, aiming to achieve normotension and only trace pedal edema.
- The uses of diuretic therapy, usually a loop diuretic are considered to maintain.
- Reminder: The ability to concentrate or dilute the urine maximally becomes progressively impaired as GFR.
- Considerations regarding fluid restrictions in the CKD, non-ESRD patients.

Medical Nutrition Therapy (MNT)

- As of January 2002, Medicare provides coverage for Medical Nutrition Therapy to CKD patients
- This provides a pathway for early nutritional interventions in order to improve the outcome and quality of life for CKD patients

MNT Education Monitoring

- Regular monitoring of nutritional status should be a part of the routine care of CKD patients
- 1-3 month interval between nutritional counseling visits is recommended. If the patient is found to have inadequate nutrient intake, or a complication that impacts nutritional status negatively, then a more frequent nutritional intervention is indicated.

Nutritional Considerations in CKD

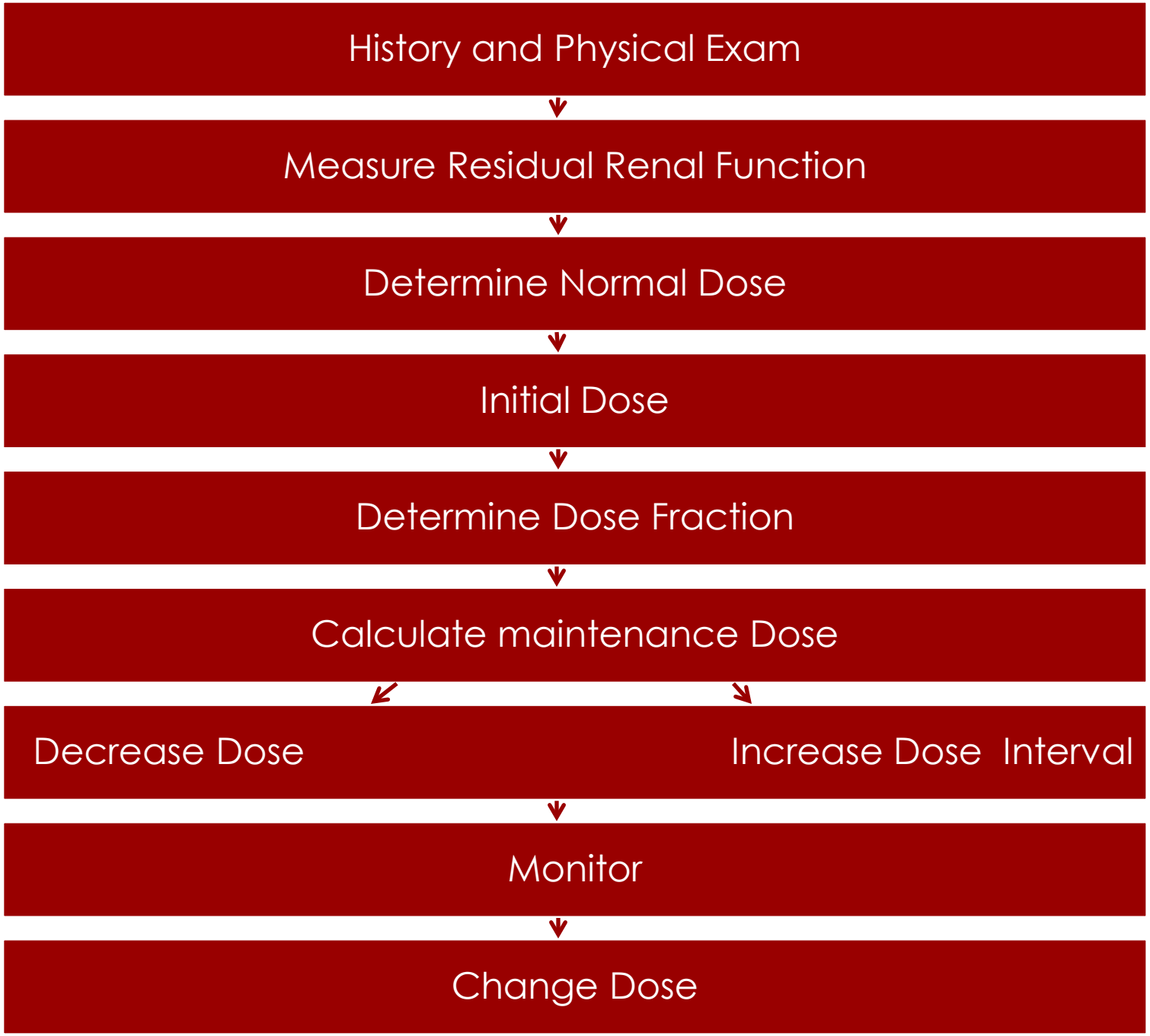
- Individualized to each patient's CKD stage and co-morbid conditions
- Research utilizing animal models have shown that reduced dietary protein is associated with a reduction in glomerular hyperfiltration and a slowing of renal disease progression.
- Successful compliance in the MDRD recommendation required intensive regular interaction by dietitians.
- Typical Dietary Recommendations for Chronic Kidney Disease
  Protein: 0.6-0.8 g/kg/day
  Sodium: <2 g/day (<6 g/day of salt)
  Potassium: 40-70 mEq/day
  Phosphate: 600-800 mg/day
  Calcium: 1400-1600 mg/day (not to exceed 2000 mg/day)
  Free water (in excess of urine output): 1-1.5 L/day

Medication Dosing in CKD

Drug adjustment in CKD

- Uremic affects alters the pharmacology of many drugs
- Caregivers must consider the following in CKD
  1. Bioavailability
  2. Distribution
  3. Metabolism
  4. Elimination

  For medications and the active or toxic metabolites
- Further complicated by other co-morbid conditions such as Diabetes, Hypertension or Heart Disease
- Utilize resources that assist with dosing for pre-ESRD and Dialysis


History and Physical Exam
Measure Residual Renal Function
Determine Normal Dose
Initial Dose
Determine Dose Fraction
Calculate maintenance Dose
Decrease Dose
Increase Dose Interval
Monitor
Change Dose

Effects or Uremia on Drug Disposition

- Bioavailability

Changes with gastrointestinal absorption in CKD : ie phospate binders, nausea, vomiting, gastroparesis

Little is known about bowel function in CKD – thought that salivary urea is converted to ammonia buffering gastric acid

- Distribution

Altered by factors common in CKD

✓ Edema/Ascites

✓ Dehydration/muscle wasting

✓ Decreased protein binding capability

- Metabolism

CKD slows the rate of non renal drug elimination

Changes in the CYP450 protein expression in Uremia

Metabolite Accumulation in CKD probable cause for increased frequency of adverse reactions.

Difference in stable CKD and Acute CKD

- Drug Dosing Calculations

Loading Dose= Vd X IBW X Cp
consider: edema, ascites, dehydration etc

Subsequent Doses
Df = t½ normal / t½ renal failure

Dosing Interval
DI in CKD = Normal Dose X DF
good for drugs with narrow therapeutic range and short plasma half life
DI in CKD = Normal Dose / Df
Wide therapeutic range and long plasma half life ** care with toxic or subtherapeutic levels

Most common combining dose reduction and prolonged intervals

Monitor therapeutic peak/trough

- Drug Removal by Dialysis

Occurs primarily by diffusion across the dialysis membrane

Moves down a concentration gradient from plasma to dialysate

Very effective for non-protein bound and <50 Daltons (small molecular weight)

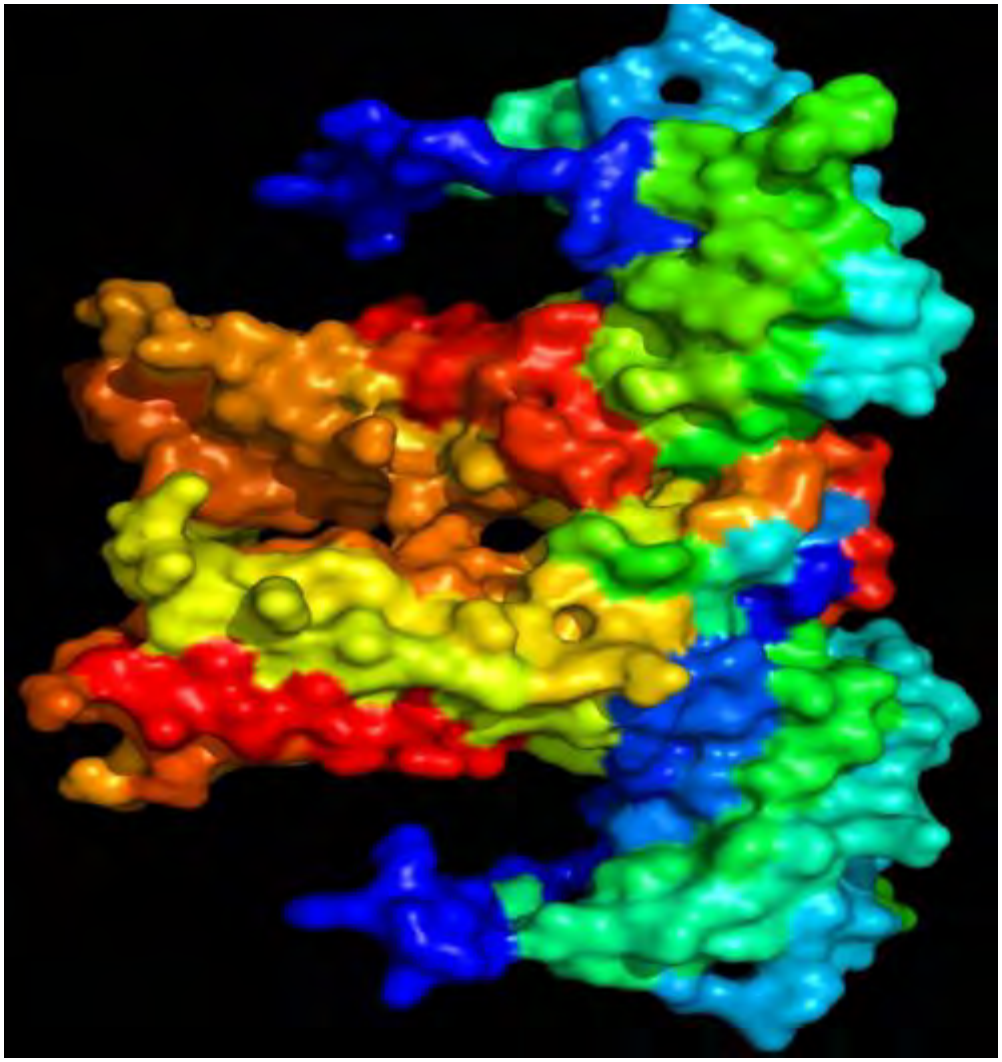
Changes with dialyzer membrane characteristics, surface area, blood flow rates and dialysate flow rates

Difference between hemodialysis and peritoneal dialysis

- General rule of thumb – not removed by hemodialysis will not be removed by peritoneal

Drug	Dose Method	GFR>50 mL/min	GFR 10-50 mL/min	GFR<10 mL/min	Supplement dose p HD	CAPD	CRRT
Acetominophen	I	q4hr	q6hr	q8hr	None	None	Dose for GFR 10-50
Alendronate	D	100%	Avoid	Avoid	Avoid	Avoid	Avoid
Amlodipine	D	100%	100%	100%	None	None	Dose for GFR 10-50
Amoxicillin	I	q8hr	q8-12hr	q24hr	Dose p HD	250mg q 12hr	N/A
Atenolol	D,I	100% q24hr	50% q48hr	30-50% q96hr	25-50mg	None	Dose for GFR 10-50
Benazepril	D	100%	50-75%	25-50%	None	None	Dose for GFR 10-50
Carvedilol	D	100%	100%	100%	None	None	Dose for GFR 10-50
Codiene	D	100%	75%	50%	Unknown	Unknown	Dose for GFR 10-50
Digoxin	D,I	100% q24hr	25-75% q36hrs	10-25% q48hr	None	None	Dose for GFR 10-50
Glipizide	D	100%	100%	100%	Unknown	Unknown	Avoid
Glyburide	D	Unknown	Avoid	Avoid	None	None	Avoid
Ibandronate	D	100%	Avoid	Avoid	Unknown	Unknown	Avoid
Metformin	D	50%	25%	Avoid	Unknown	Unknown	Avoid
Omeprazole	D	100%	100%	100%	Unknown	Unknown	Unknown
Simvastatin	D	100%	100%	100%	Unknown	Unknown	NA
Warfarin	D	100%	100%	100%	None	None	None

Pharmacology Specific to CKD



Common Medications in CKD

- Erythropoetic agents
- Ergocalciferol/Cholecalciferol
- Iron Replacement
- Vitamin D analogs
- Phosphorous binders
- Calcium Metabolic/Hyperparathyroid

Anemia Secondary to CKD

- Erythropoietin

A Glycoprotein hormone synthesized and released by the peritubular type I cells in the renal cortex

- Recombinate cloned by Jacobs etal in 1985
- First released by FDA 1989
- Currently available as; epoetin-α, epoetin-ß, epoetin-Ω

Replacement Therapy

- ✓ Epoetin alfa
- ✓ Darbepoetin alfa
- ✓ Peginesatide
- ✓ Several Newer Medications in the pipeline

- **Dosing Considerations**

✓ Route: IV vs. SQ

✓ Frequency: ESRD vs. CKD/PD

✓ Initial Dose: 50 – 100 u/Kg 3x wk

✓ Monitoring: Q1 -2 wks
Hemogram at initiation
Once goal reached Q 2-4 wks
Check Iron Stores Q 3 months

✓ Target for Therapy: An increase of Hgb by 0.5 to 1.5 per week
Maintenance of Hgb 10 – 11g/dL

✓ Main Treatment Goal – Avoid blood transfusions

- **Adverse Effects of Treatment**

i. ****FDA Black Box Warning****

ii. Restricted Use to CKD/ESRD, HIV associated, some chemo-associated, transfusion reduction (surgery associated)

iii. Increased Mortality Cardiovascular, Tumor Progression, Thromboembolic Events

iv. Common Reactions: Hypertension, Headache, Arthralgias, Tachycardia, AVF/AVG clotting

Iron Therapy in CKD

- Iron deficiency can occur at any stage of CKD
- An Essential component of Anemia Management
- Balance reflects a homeostatic system
- Diagnosis of Iron Deficiency:
  HD – Ferritin < 200ng/mL
  TSAT < 20%
  CKD/PD – Ferritin <100ng/mL
  TSAT < 20%

- Monitoring:
  a. Initially when starting ESA
  b. Monthly for those not receiving Iron on ESA
  c. 3 Months on stable ESA and iron dosing
- Limitation of Lab Studies
  a. Acute phase reactant
  b. Limited sensitivity in CKD
- Used in Ferritin/TSAT together with Hgb for iron dose requirements

Iron Therapy in CKD

- Iron Replacement oral/IV

- Oral Replacement:
  a. Simple/cost effective with multiple oral iron drugs
  b. Limited efficacy in HD[1]
  c. Daily dose:
  Elemental Iron 200mg/day total in divided doses[2]

- IV Replacement:
  a. Iron Sucrose 1mL=20mg elemental iron
  HD – 100mg IVQ HD tx X 10 doses
  PD – 300mg IV X 1 on days 1 and 15 then 400mg IV on day 28
  Non-dialysis – 200mg IV X 5 doses with 14 days.
  Total 1000mg dose

  b. Sodium Ferric Gluconate
  1mL=12.5mg elemental iron
  HD – 125mg IV Q HD tx X 8 doses. Total 1000mg dose

  c. Iron Dextran 1mL=50mg elemental iron
  Black Box Warning

1. Macdougal, et al: A randomized controlled study of iron supplements in patients treated with erythropoietin. Kidney Int 1996.
2. NKF- KDOQI Clinical Practice Guidelines for Anemia of CKD, 2006.

Iron Therapy in CKD

- **Adverse Effects:**
- **Oral Agents – GI side effects**
- **IV Agents -**
  a. Iron Dextran – Associated with severe anaphylaxis = 0.6 -0.7% of all pts treated.
  b. Iron Sucrose/Ferric Gluconate – reduced risk 0.04% if all pts treated.[1]

- **Preferred Route of Iron administration for Hemodialysis is IV[2]**
- **Monitor for Iron Overload Hold for Serum Ferritin >800ng/mL[2]**
- **Consider Holding Infusions in the Presence of Infections[3]**

1. Kosch,M etal: A randomized, controlled parallel-group trial. Nephrol Dial Transplant 2001: 16(6):1239-1244.
2. NKF-KDOQI Clinical Practice Guidelines for Anemia of CKD, 2006.
3. Kessler,M etal: Bacteremia in patients on chronic hemodialysis. A multicenter prospective survey. Nephron 1993; 64:95-100.

Bone Mineral Management

- **Phosphate Binders**

  a. Sevelamer Hydrochloride: Initial dose – 800mg PO with meals. Maximum dose 14g/day

  b. Lanthanum carbonate: Initial dose – 750 – 1500mg/ day PO divided with meals. Maximum dose 3750 mg/ day. **Must be crushed or chewed**

  c. Calcium acetate: Initial dose – 1334mg with meals. Maximum dose 2668mg PO each meal.

  d. Aluminum Hydroxide: Initial dose – 300-600mg PO with meals. **No longer used 2nd to Aluminum Toxicity**

- **Calcium Disorders / Hyperparathyroid**

  a. Cinacalcet: Initial Dose – 30mg PO Q day with largest meal. Titrate dose Q 2-4 weeks until target iPTH reached. Maximum dose 180mg PO Q day for ESRD. **Only indicated for patients on dialysis**

Binder Characteristics

	Aluminum hydroxide	Calcium acetate/ carbonate	Magnesium hydroxide/ carbonate	Lanthanum carbonate	Sevelamer hydrochloride
Efficacious	**Yes**	**Yes**	**N/A***	**Yes**	**Yes**
Absorbed	**Yes**	**Yes**	**Yes**	**Yes**	**No**
Accumulates	**Yes**	**Yes**	**Yes**	**Yes**	**No**
Contributes to Ca x P product	**No**	**Yes**	**No**	**No**	**No**
Lipid effect †	**No**	**No**	**No**	**No**	**Yes**

***Not indicated for the treatment of hyperphosphatemia** **† Reductions in LDL and total cholesterol**

National Kidney Foundation. KDOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. *Am J Kidney Dis*. 2003;42(suppl):S1-S201;
Bleyer AJ, Burke SK, Dillon M, et al. *Am J Kidney Dis*. 1999;33:694-701;
Chertow GM, Burke SK, Raggi P. Treat to Goal Working Group. *Kidney Int*. 2002;62:245-252;
Coladonato JA. *J Am Soc Nephrol*. 2005;16:S107-S114;
Hutchison AJ, Maes B, Vanwalleghem J, et al. *Nephron Clin Pract.* 2006;102(2):c61-71. Epub 2005 Oct 14;
Joy MS, Finn WF. *Am J Kidney Dis*. 2003;42:96-107.

- Vitamin D Analogs

a. Paricalcitrol – PO/IV
Initial dose -1mcg po Q day.
0.04 – 0.1mcg/kg IV 3 X wk.
Titrate based on iPTH levels.
Maximum dose 0.24mcg/kg

b. Calcitriol – PO/IV
Initial dose - 0.25 mcg PO Q day. 1– 2mcg IV 3 X wk.
Titrate based on iPTH levels.

c. Doxercalciferol – PO/IV
Initial dose - 1mcg PO Q day.
4 mcg IV 3 X wk. Titrate based on iPTH levels.
Maximum dose PO 3.5mcg
CKD, IV 18 mcg/wk

- Adverse Reactions

a. Cautions:
Hypercalcemia,
Hyperphosphatemia,
Adynamic bone disease,
Hypervitaminosis D

- Require Close Monitoring for dose titration

Vitamin D 2,3

- Ergocalciferol – (Vitamin D_2)
- Cholecalciferol – (Vitamin D_3)

Initial dose recommendations per Vitamin D_{25} levels.

Serum D_{25} ng/mL (nmol/L)	Definition	Ergocalciferol Dose	Duration (months)	Comment
<5 (12)	Severe deficiency	50,000 IU/wk po X 12 wks then monthly x 3	6 months	Check D_{25} after 6 months
5 -15 (12-37)	Mild deficiency	50,000 IU/wk po X 4 wks then monthly X 5	6 months	Check D_{25} after 6 months
16 – 30 (40-75)	Insufficiency	50,000 IU/month X 6	6 months	Check D_{25} after 6 months

NKF- KDOQI: Clinical Practice Guidelines for Chronic Kidney Disease. Am J Kidney Dis 2002; 39(2 suppl 1): S1 – S266

CKD patient education

- Goals of CKD Education

✓ Aligns with the goals of therapy:
  a. Delay progression
  b. Delay/treat known CVD comorbidities
  c. Manage uremic complications
  d. Ensure modality choice
  e. Initiate timely kidney replacement therapy

❖ Each of these goals require education of the patient and caregivers

❖ Communication and co-management essential

Cutis B, Levin A: The role of the chronic kidney disease clinic. Chronic Kidney Disease, Dialysis & Transplantation 2nd ed. 2005; 71-82
NKF- KDOQI: Clinical Practice Guidelines for Chronic Kidney Disease. Am J Kidney Dis 2002; 39(2 suppl 1): S1 – S266

CKD Education

- **General Statement**

  To maintain health by providing the greatest opportunity to reduce morbidity and mortality.

- **Mission Statement**

  Nephrology Associates Medical Group, Inc (NAMG)

  We are a dedicated group of compassionate healthcare professionals providing excellent care for all patients with kidney and associated diseases.

- **Core Values**

- ✓ Quality
- ✓ Teamwork
- ✓ Compassion
- ✓ Professionalism
- ✓ Accountability
- ✓ Integrity

Why is it Important to Educate?

- Important for decision-making
- Alleviate fear and psychologic suffering
- Taking an active part in care
- Better out-come in chronic diseases[1,2,3]

1. Loveman,E etal: The clinical and cost effectiveness of patient education models for diabetes: A systemic review. Health Technol Assess 2003; 7(22):iii, 10190. 2. Latos D, Schatell D: The nephrologists' critical role in patient education. Adv Ren Replace Ther 2003; 10(2): 146-149. 3. Wright SP etal: Uptake of self-management strategies in a heart failure management programme. Eur J Heart Fail 2003; 5(3): 371-380.

CMS Guidelines for CKD Education

Effective Date: January 1, 2010

- MIPPA Section 152(b) adds Kidney Disease Patient Education services as a Medicare covered benefit for Medicare beneficiaries diagnosed with Stage IV chronic kidney disease (CKD). The services are designed to provide beneficiaries with comprehensive information regarding the management of comorbidities, including for purposes of delaying the need for dialysis; prevention of uremic complications; and each option for renal replacement therapy. The benefit is also designed to be tailored to individual needs and provide the beneficiary with the opportunity to actively participate in his/her choice of therapy.
- Face-to-face educational services either individual or group per one hour

http://www.cms.gov/Regulations-and-Guidance/Guidance/Transmittals/downloads/r1876cp.pdf

Considerations in CKD Education

- **Tools Needed**

✓ Lesson plans/structure
✓ Patient handouts/ Videos/ Internet Resources/ binders or folders
✓ Educational word games, puzzles, quizzes, and take-home exercises
✓ Evaluation and continuous improvement
✓ Outcomes instruments

- Internet Resources
- http://www.kidney.org
- http://www.davita.com
- http://www.kidneyschool.org
- http://ww.aakp.org

NAMG CKD Education Structure

- **Formal Multidisciplinary Team**
- **Utilizes Informal Multidisciplinary Resources**
- **Structured Education –** including but not limited to the following:

✓ Explanation of normal kidney function, blood pressure, lab tests and their significance

✓ Explanation of specific disease conditions, symptoms, and complications of CKD

✓ Dietary teaching and diabetes education (if appropriate)

✓ Ensuring that patient understanding of medications is adequate

✓ Discussions about vein preservation

✓ Eythropoietin hormone therapy teaching including importance of anemia and its treatment.

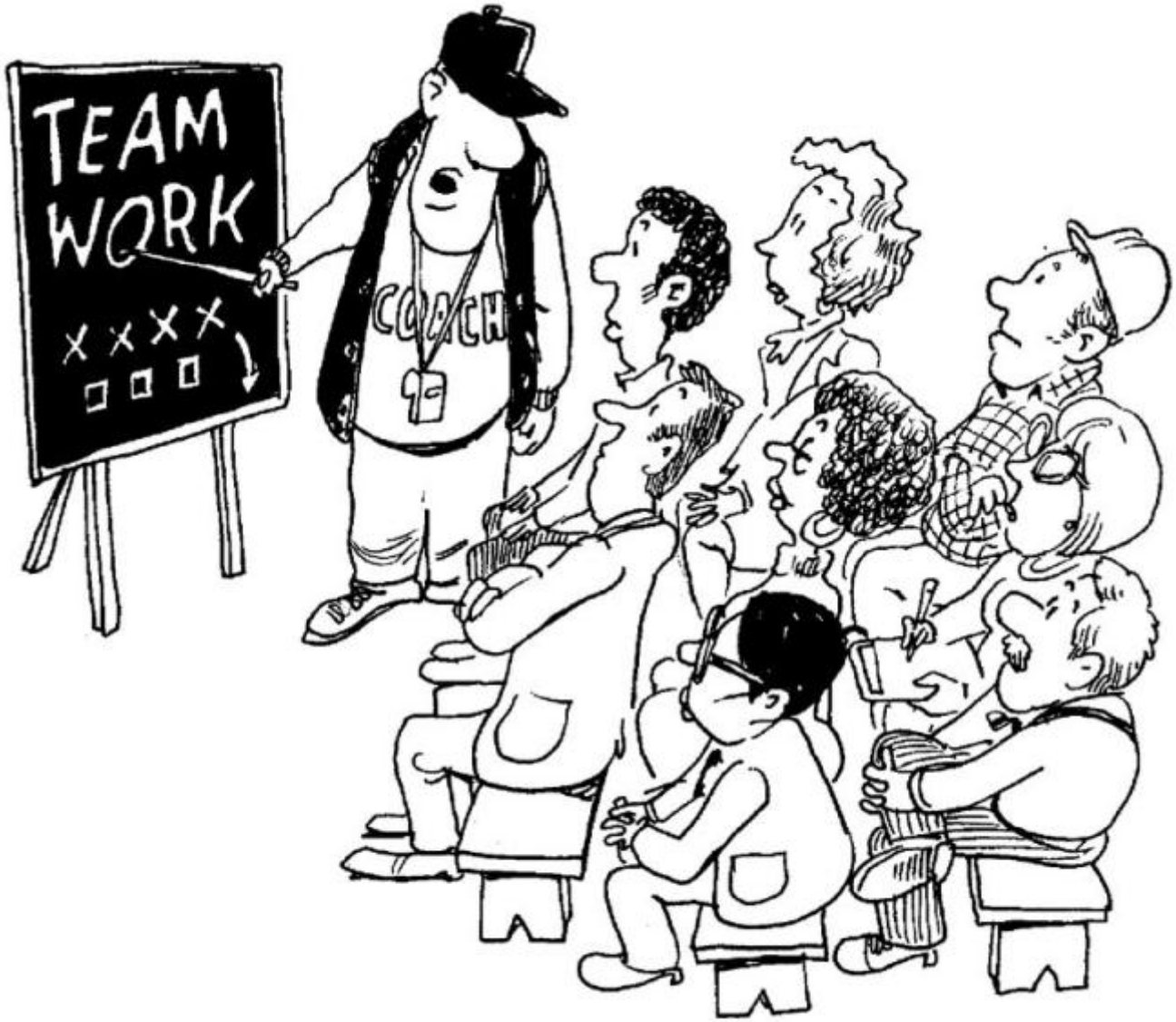
✓ Discussion of choices in treating ESRD

Education utilizes the principles of adult learning, and regular reinforcement are incorporated in the program

Follow-up

- **Summary**
- Early detection, evaluation, and diagnosis of chronic kidney disease allow for the timely initiation of measures aimed at slowing disease progression.
- Classification of CKD into stages 1 to 5 facilitates patient care through the application of stage-specific clinical action plans.
- During early CKD stages, aggressive blood pressure control is the mainstay of therapy and has a proven effect in limiting the progression of disease.
- The progressive loss of renal function is accompanied by a wide range of adaptive and maladaptive processes as the body attempts to maintain homeostasis resulting in a complex medical clinical picture, with its main manifestations in the cardiovascular, neurologic, hematologic, musculoskeletal, and immunologic systems.
- Given the complexities involved in the care of CKD patients, referral to a nephrologist for evaluation and comanagement is recommended.

It Takes a Team to Get Here!



CKD/MNT @ NAMG	Without CKD/MNT @ NAMG
42 yo White Male	41 yo White Male
Diabetic type 1	Diabetic type 1
HTN	HTN
CVA	PVD/DVT
Hyperlipidemia	CHF
Hx Methampetamine abuse	LLE BKA
AVF functioning at start of HD tx	Access – Right IJ CVC
Hgb ……..13.1 PO4 …….. 4.0 PTH-I …….. 218 Albumin ….. 4.6 GFR …… 13 BUN …… 72 Hgb A1C …. 6.7	Hgb ……..10 PO4 …….. 2.3 PTH-I …….. 33 Albumin ….. 2.1 GFR …… 9 BUN …… 31 Hgb A1C …. 7.5
Lives at home with mother	Lives at home with Mother

Case Study

- WC 67y/o Caucasian male referred from PCP due to increase creatinine
- Hx of Present Illness; c/o sob with minimal activity x 4 mos, fatigue for few weeks, unintentional wt loss. Nuclear stress test positive for ischemia, cardiologist would like nephrology clearance prior to coronary angiogram
  - Significant Labs
    - Creat 2.55; eGFR 15/ml/min
    - Calcium – 10.5; PO4; iPTH
    - Total Cholesterol – 114; TG – 99; HDL – 34
    - UACR – not done
    - Hgb - ; Hct
  - PMHx: s/p aortic valve replacement – on anticoagulation, Chronic LBP. Denies HTN, DM, Dyslipidemia