CVD is the number one killer

- Leading cause of death in men and women
- Hyperlipidemia is a major modifiable risk factor for CVD
- Discussion today will include recommendations from ATP III (2004 update), the AHA/ACC guidelines on primary and secondary prevention and the Expert Panel of Lipid specialists consensus statement on advanced lipoprotein testing, convened by the National Lipid Association

Lipids - Definition

- Lipids are organic compounds (include fats, oils, sterols, triglycerides)
- Principal structure of living things (along with proteins and carbohydrates)
- Essential to the body’s function
- Transport system evolved to allow delivery of lipids to the organs that need them

Lipid Management

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Clinical Director, Lipid Clinics
Assistant Professor
Division of Cardiovascular Medicine
The Ohio State University Wexner Medical Center

- LDL
  - Primary carriers of cholesterol
  - Provide it to the body
- HDL
  - Carries cholesterol back to the liver
- VLDL and chylomicrons - lipids that are triglyceride rich
- Non-HDL is Total cholesterol – HDL
- Non-HDL also reflects cholesterol that is atherogenic
Overnutrition

- With overnutrition, lipids can become harmful
- Lipids (in particular LDL and chylomicron remnants) become “stuck” to the subendothelium of the vasculature
- Forms a “fatty streak,” the first step in the development of atherosclerosis

Risk Assessment

- ATP III- fasting lipid profiles
- Step-wise approach

Step 1

- Identify patients with clinical atherosclerosis
  - Clinical coronary artery disease
  - Peripheral arterial disease
  - Symptomatic carotid artery disease
  - Abdominal aortic aneurysm

Step 2

- Determine the presence of major risk factors
  - Smoking
  - Hypertension
  - Low HDL
  - Age (men > 45 years, women > 55 years)
  - Family history of premature coronary artery disease (males ≤ 55 years, females ≤ 65)
Step 3

• Assess 10 year risk in patients without coronary artery disease or an equivalent
• Framingham score- three levels
  – > 20% = coronary equivalent
  – 10% - 20%
  – < 10%

Other Risk Assessments

• Reynolds Risk Score
  – Gender, age, family history, blood pressure, smoking, HDL, hs-CRP
• PROCAM
  – Gender, age, family history, blood pressure, glucose > 120, weight, height and hypertension meds

Step 4

• Identify metabolic syndrome
  – Abdominal obesity
  – Elevated triglycerides
  – Low HDL
  – Elevated blood pressure
  – Elevated fasting glucose

Step 5: NCEP ATP III - LDL-C goals

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>Risk Factors</th>
<th>10 year CHD-risk</th>
<th>LDL-C Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very High-Risk</td>
<td>CHD or CHD RE + RF</td>
<td>&gt;20%</td>
<td>&lt;70mg/dL</td>
</tr>
<tr>
<td>High-Risk</td>
<td>CHD or CHD RE</td>
<td>&gt;20%</td>
<td>&lt;100mg/dL</td>
</tr>
<tr>
<td>Moderately-high risk*</td>
<td>2 + RF</td>
<td>10-20%</td>
<td>&lt;100mg/dL</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>2 + RF</td>
<td>&lt;10%</td>
<td>&lt;130mg/dL</td>
</tr>
<tr>
<td>Low Risk</td>
<td>0-1</td>
<td></td>
<td>&lt;160mg/dL</td>
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</table>
Step 6: ADA/ACC 2008 Consensus Statement

<table>
<thead>
<tr>
<th></th>
<th>LDL</th>
<th>Apo B</th>
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<tbody>
<tr>
<td>Highest risk patients</td>
<td>&lt; 70</td>
<td>&lt; 80</td>
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<tr>
<td>- known CHD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- DM with an</td>
<td></td>
<td></td>
</tr>
<tr>
<td>additional CV risk factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Risk patients</td>
<td>&lt; 100</td>
<td>&lt; 90</td>
</tr>
<tr>
<td>- no known CHD or DM but 2 clinical risk factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- DM but no other major CV risk factors</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CV risk factors: smoking, hypertension, family history of premature CHD

Therapeutic Lifestyle Change

- Reduce saturated fat and cholesterol intake
- Plant stanols/sterols (2 g/d), increased viscous (soluble) fiber intake (10-25 g/d)
- Weight reduction
- Physical activity

Prioritize targets

- If TG <500, LDL is primary target
- If TG >500, TG is primary target
- When LDL is at goal and TG 200-500, non-HDL is target

Pharmacologic Therapy

- Statins
- Bile Acid Sequestrants
- Niacin
- Fibric Acid Derivatives
- Ezetimibe
- Omega-3 Fatty Acids
Statins

- **Mechanism of action**
  - Inhibits B-hydroxy beta methyl glutaryl Co A reductase
  - Therefore decreases production of cholesterol
  - Leads to upregulation in LDL receptors that take up LDL out of the blood

- **Statin effects**
  - LDL levels ↓30-60%, TG levels ↓~20%, HDL levels ↑5-15%

- **Side effects:** Myopathy, increased liver enzymes

Muscle and Statin Safety

- **Baseline CK only for high risk pts**
- **Counsel pt to report any muscle symptoms**
- **Do not measure CK if asymptomatic**
- **Check CK in symptomatic pts**
- **Evaluate muscle symptoms or ↑ CK level for other causes**

### Relative LDL Lowering of Statins

<table>
<thead>
<tr>
<th></th>
<th>Atorva 40 mg</th>
<th>Fluva 1 mg</th>
<th>Pitava 20 mg</th>
<th>Lovar 20 mg</th>
<th>Prava 10 mg</th>
<th>Rosuv 40 mg</th>
<th>Vytorin 10 mg</th>
<th>Simva 10 mg</th>
<th>% LDL decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 mg</td>
<td>--</td>
<td>40 mg</td>
<td>20 mg</td>
<td>--</td>
<td>20 mg</td>
<td>--</td>
<td>--</td>
<td>10 mg</td>
<td>30%</td>
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<tr>
<td>20 mg</td>
<td>80 mg</td>
<td>4 mg</td>
<td>80 mg</td>
<td>80 mg</td>
<td>5 mg</td>
<td>10/10 mg</td>
<td>40 mg</td>
<td>41%</td>
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<tr>
<td>40 mg</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>10 mg</td>
<td>10/20 mg</td>
<td>80 mg</td>
<td>47%</td>
<td></td>
</tr>
<tr>
<td>80 mg</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>10 mg</td>
<td>10/40 mg</td>
<td>55%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>20 mg</td>
<td></td>
<td>10/80 mg</td>
<td>40 mg</td>
<td></td>
<td>63%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Liver and Statin Safety

- **Check LFTs at baseline and as clinically indicated**
- **Evidence of liver injury**
  - d/c statin, etiology should be sought/refer pt to gastroenterologist/hepatologist
- **Isolated, asymptomatic ↑ LFTs**
  - 1-3 X ULN, no need to d/c statin
  - > 3 X ULN, test should be repeated
- **According to the Expert Liver Panel, pts with chronic liver disease, nonalcoholic fatty liver disease, or NASH may safely receive statin therapy.**

Risk versus Benefit of Statins

- Risks from fatal and non-fatal rhabdomyolysis are ~ 0.3 and 3 per 100,000 person years respectively
- Acute Liver Failure 0.5-1 per 100,000 person years (~ equal to background rate of liver failure in the general population)
- No evidence that statins cause:
  - Acute or chronic kidney damage
  - Peripheral neuropathy
  - Impairment of memory or cognition

Secondary Prevention Statin Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Population</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIRACL</td>
<td>3086 patients with ACS</td>
<td>16% relative risk reduction in death, nonfatal acute MI, cardiac arrest, recurrent symptomatic myocardial ischemia with objective evidence and requiring emergency hospitalization</td>
</tr>
<tr>
<td>4S</td>
<td>4444 men and women with prior MI</td>
<td>30% relative risk reduction in death, 42% reduction in coronary deaths, 37% reduction in revascularization procedures</td>
</tr>
<tr>
<td>CARE</td>
<td>4159 men and women with prior MI</td>
<td>CHD death and nonfatal MI reduced 24%; 26% reduction in CABG; 22% reduction in PTCA</td>
</tr>
</tbody>
</table>

Primary Prevention Statin Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Population</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>WOSCOPS</td>
<td>High-risk men</td>
<td>31% reduction in fatal and nonfatal MI; 32% reduction from all CV causes; 22% reduction in all-cause mortality</td>
</tr>
<tr>
<td>AFCAPS/TexCAPS</td>
<td>6605 men and women</td>
<td>36% reduction in first coronary event; 26% reduction in fatal and nonfatal MI; 33% reduction in revascularization (CABG, PTCA)</td>
</tr>
<tr>
<td>CARDS</td>
<td>2638 Type 2 diabetic patients without known CHD</td>
<td>Cardiovascular events were reduced by 37%</td>
</tr>
</tbody>
</table>
ASCOT-LLA
10, 305 increased risk patients
Intervention: Atorva 10
34% reduction in nonfatal MI and fatal CHD
21% reduction in total cardiovascular events
27% reduction in fatal and nonfatal stroke

JUPITER
17, 802 men and women with LDL < 130 and CRP > 2
Intervention: Rosuva 20
Primary endpoint of a first major CV event reduced by 44%
Mortality reduced by 20%.

Nicotinic acid
- Decrease LDL 5-25%
- Raise HDL 15-35%
- Decrease TG by 20-25%
- Side effects: Flushing, hyperglycemia, hepatotoxicity

Bile acid sequestrants
- Colesevelam, cholestyramine, and colestipol
- Decrease LDL 15-30%
- Raise HDL 3-5%
- No change or increase in TG
- Side effects: GI distress, constipation, decreased absorption of other drugs
- Contraindicated in hypertriglyceridemia

Fibric acids
- Decrease LDL 5-20% (may increase LDL in patients with high TG)
- Raise HDL 10-20%
- Decrease TG 20-50%
- Side effects: GI distress
**Ezetimibe**

- Cholesterol absorption inhibitor
- Decreases LDL by 18%
- Increases HDL by 1%
- Decreases TG by 2%
- Additive to statins

**Outcome Data on Non-statins**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholestryamine</td>
<td>• Effective in primary prevention (Lipid Research Clinics 1984) and secondary prevention in men (Watts 1992)</td>
</tr>
<tr>
<td>Colestipol</td>
<td>• Significantly reduces cardiovascular events compared to placebo (Insull 2006)</td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>• No cardiac outcomes data and ezetimibe did not reduce regression of carotid intima-media thickness (surrogate marker) when added to a statin (Kastelein 2008, Taylor 2009)</td>
</tr>
</tbody>
</table>

**Omega-3 Fatty Acids**

- **Mechanism of Action**
  - Slows the release of TG rich VLDL into plasma
  - Accelerates clearance of TG rich lipoproteins via enhanced lipolysis

- **Effects**
  - 2000-4000mg of EPA + DHA, TG ↓17-47% (45% if TG >500)
  - Most OTC 1-g fish oil contain 300mg to 500mg of EPA + DHA

- **Side effects**
  - GI (burping/belching, diarrhea), fishy after taste
  - ↑ALT and LDL-C
  - Monitor BS
  - Mildly inhibit platelet fxn at high doses

**Fenofibrate**

- In type 2 diabetics, did not reduce primary outcome of fatal MI or CHD mortality. Improved secondary outcomes of non-fatal MI and coronary revascularization, a reduction in albuminuria, reduced laser treatments for retinopathy (FIELD 2005)
- Added on to statin, did not lower risk of non-fatal MI, non-fatal stroke, or CV death, more than statin alone in patients with type 2 diabetes at high risk for CV disease. May be a subgroup (high TG, low HDL) that benefits (ACCORD 2010)

**Gemfibrozil**

- Effective in primary prevention in men (Helsinki Heart Study 1987) and in secondary prevention in men with low HDL (VA-HIT 1999)
### Conclusions

- Hyperlipidemia is a major modifiable risk factor
- Risk assessment
- Cholesterol targets
- Pharmacologic therapy

### Niacin

- Effective in secondary prevention (Coronary Drug Project 1975)
- Niacin and simvastatin decreased atherosclerosis, coronary death, MI, stroke, or revascularization (HATS 2001)
- Added on to statin, niacin decreased CIMT (ARBITER-2 2004, ARBITER-6 HALTS 2009)
- In patients with stable CVD and LDL < 70, no benefit to addition of niacin to statin therapy (AIM-HIGH 2011)

### Omega-3 fatty acids

- Effective in secondary prevention (GISSI-Prevenzione 2002); however, recent meta-analysis did not show that omega-3 fatty acids reduce cardiovascular events or mortality (Rizos 2012)

### Future Directions

- PCSK9 inhibitors
- PCSK9 enzyme that degrades the LDL receptor, raising serum LDL levels.
- If PCSK9 is inhibited, LDL levels fall.
- Dramatic drops in LDL in Phase II trials
- Phase III trials underway

### Lipid Cases

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Patient Case Outline

- Case #1
  - High TG
  - Drug Interactions
  - Statin Adverse Effects
- Case #2 – Intermediate Risk

Patient Case #1

- Nutrition Assessment reveals
  - No breakfast
  - Sweet tea and fruit throughout the day
  - Few crackers, pop, for lunch
  - Large dinner, including white meat, large portion of white pasta/rice/bread/potatoes

Patient Case #1

- 55 yo male
- PMH: HTN, newly diagnosed DM
- FH/SH/ROS: Non contributory
- Labs:

<table>
<thead>
<tr>
<th>Component</th>
<th>11/15/2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOLESTEROL</td>
<td>280</td>
</tr>
<tr>
<td>TRIGLYCERIDES</td>
<td>567 (H)</td>
</tr>
<tr>
<td>HDL CHOLESTEROL</td>
<td>28 (L)</td>
</tr>
<tr>
<td>LDL CHOLESTEROL</td>
<td>---</td>
</tr>
<tr>
<td>NON-HDL CHOLESTEROL</td>
<td>182</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>135</td>
</tr>
<tr>
<td>HbA1c</td>
<td>7.3</td>
</tr>
</tbody>
</table>

- Step 1: Define Risk
  - Diabetes
  - Metabolic Syndrome
- Step 2: Prioritize goals
**Step 3: Treatment Considerations**
- Secondary causes of dyslipidemia
  - Impaired Fasting Glucose
  - Hypothyroidism
- Therapeutic Lifestyle Changes
- Medication Considerations

**Patient Case #1**

- Discuss TLC w/ small, quantifiable goals
- Started on gemfibrozil 600mg twice daily
- Returns to clinic in 8 wks with following labs:

<table>
<thead>
<tr>
<th>Component</th>
<th>1/15/2012</th>
<th>11/15/2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOLESTEROL</td>
<td>240</td>
<td>280</td>
</tr>
<tr>
<td>TRIGLYCERIDES</td>
<td>350 (H)</td>
<td>567 (H)</td>
</tr>
<tr>
<td>HDL CHOLESTEROL</td>
<td>31 (L)</td>
<td>28 (L)</td>
</tr>
<tr>
<td>LDL CHOLESTEROL</td>
<td>139 (H)</td>
<td>----</td>
</tr>
<tr>
<td>NON-HDL CHOLESTEROL</td>
<td>209</td>
<td>182</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>132</td>
<td>135</td>
</tr>
<tr>
<td>HbA1c</td>
<td>7.1</td>
<td>7.3</td>
</tr>
</tbody>
</table>

**Combination Therapy**
- Compelling Indications
- Cost
- Drug Interactions
Statins and Drug Interactions

- Risk of myopathy ↑ when statins are coadministered with medications that inhibit their metabolism
- Choosing a noninteracting medication or switching to a non-interacting statin may be the safest option

Statins and Drug Interactions

- Focus on Simvastatin

<table>
<thead>
<tr>
<th>Contraindicated with simvastatin:</th>
<th>Itraconazole, Ketoconazole, Posaconazole, Erythromycin, Clarithromycin, Telithromycin, HIV protease inhibitors, Nefazodone, Gemfibrozil, Cyclosporine, Danazol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do not exceed 10mg daily of simvastatin with:</td>
<td>Verapamil, Diltiazem</td>
</tr>
<tr>
<td>Do not exceed 20mg daily of simvastatin with:</td>
<td>Amiodarone, Alodipine (New), Ranolazine (New)</td>
</tr>
<tr>
<td>Limit use of simvastatin 80mg daily</td>
<td>Increased risk of muscle damage may exceed benefits, and safer alternatives are available</td>
</tr>
</tbody>
</table>

Patient Case #1

- Returns to clinic in 8 wks, labs below
- Pt complains of constant myalgias bilaterally and difficulty standing up from the seated position

<table>
<thead>
<tr>
<th>Component</th>
<th>3/15/2012</th>
<th>1/15/2012</th>
<th>11/15/2011</th>
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</thead>
<tbody>
<tr>
<td>CHOLESTEROL</td>
<td>199</td>
<td>240</td>
<td>280</td>
</tr>
<tr>
<td>TRIGLYCERIDES</td>
<td>249 (H)</td>
<td>350 (H)</td>
<td>567 (H)</td>
</tr>
<tr>
<td>HDL CHOLESTEROL</td>
<td>34 (L)</td>
<td>31 (L)</td>
<td>28 (L)</td>
</tr>
<tr>
<td>LDL CHOLESTEROL</td>
<td>115 (H)</td>
<td>139 (H)</td>
<td>----</td>
</tr>
<tr>
<td>NON-HDL CHOLESTEROL</td>
<td>165</td>
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<td>182</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>120</td>
<td>132</td>
<td>135</td>
</tr>
<tr>
<td>HbA1c</td>
<td>7.0</td>
<td>7.1</td>
<td>7.3</td>
</tr>
</tbody>
</table>
**Patient Case #1**

Statin-related muscle effects occur in the significant minority of pts, mostly myalgias with normal CK

- **Myalgia management**
  - Re-challenge/Reduce statin
  - Try different statin
    - Ultra-low dose statin and/or Hydrophilic statin
  - Assess 25-OH Vitamin D
    - Consider replacement if low
  - Consider non-statin or combination

<table>
<thead>
<tr>
<th>Component</th>
<th>7/15/12</th>
<th>3/15/12</th>
<th>1/15/12</th>
<th>11/15/11</th>
</tr>
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<tbody>
<tr>
<td>CHOLESTEROL</td>
<td>178</td>
<td>199</td>
<td>240 (H)</td>
<td>280 (H)</td>
</tr>
<tr>
<td>TRIGLYCERIDES</td>
<td>230 (H)</td>
<td>249 (H)</td>
<td>350 (H)</td>
<td>567 (H)</td>
</tr>
<tr>
<td>HDL-C</td>
<td>37 (L)</td>
<td>34 (L)</td>
<td>31 (L)</td>
<td>28 (L)</td>
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<tr>
<td>LDL CHOLESTEROL</td>
<td>95</td>
<td>115 (H)</td>
<td>139 (H)</td>
<td>---</td>
</tr>
<tr>
<td>NON-HDL</td>
<td>141 (H)</td>
<td>165 (H)</td>
<td>209 (H)</td>
<td>252 (H)</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>140</td>
<td>120</td>
<td>132</td>
<td>135</td>
</tr>
<tr>
<td>HbA1c</td>
<td>7.3</td>
<td>7.0</td>
<td>7.1</td>
<td>7.3</td>
</tr>
</tbody>
</table>

**Patient Case #1: Niaspan Education**

- Non-HDL still not at target
- Consider add-on therapy
- Options & considerations of each
  - Lovaza
  - BAS
  - Zetia
  - Niaspan – yes!

- Describe as “prickly heat”
  - Head, neck, and shoulders
  - 15-30 min after ingestion of IR, 30-120 after ER, highly variable after SR
- Expect it
  - Reassure it is “normal” and “harmless”
  - Short-lived
- Prevent it
  - Concurrent ASA 81-325mg or ibuprofen 200, 30-60 min before niacin
  - Bedtime snack
    - Whole wheat crackers or skim milk
  - Avoid high-fat meals, alcohol, spicy food
- Rapidly abort flushing with NSAID
  - For example, ibuprofen 200 mg
  - Caution in pts w/ renal or active peptic disease

- Patient was switched to rosuvastatin and was able to titrate up to 20mg daily
- Most recent labs reveal:
**Patient Case #1**

- Niaspan was added and pt was able to titrate up to 1500mg daily
- Most recent labs reveal all at goal levels:

<table>
<thead>
<tr>
<th>Component</th>
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<th>7/15/12</th>
<th>3/15/12</th>
<th>1/15/12</th>
<th>11/15/11</th>
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<td>280 (H)</td>
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<td>TG</td>
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<td>249 (H)</td>
<td>350 (H)</td>
<td>567 (H)</td>
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<tr>
<td>HDL-C</td>
<td>40</td>
<td>37 (L)</td>
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<tr>
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<tr>
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<tr>
<td>HbA1c</td>
<td>6.9</td>
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<td>7.0</td>
<td>7.1</td>
<td>7.3</td>
</tr>
</tbody>
</table>

**Patient Case #2**

- 51 yo male presents to clinic on no meds
- BP 137/85, waist 38”, sedentary, father with MI at age 53
- Laboratory values

**Step 1: Define Risk**

- Risk Factors that modify LDL-C goal = 2
- Framingham 10-year risk score = 6%
- Metabolic Syndrome
- ATP III & Update recommend LDL-C target <130mg/dL and non-HDL target of <160mg/dL

*In pt with intermediate risk, esp with positive family history, are we doing enough??*
Patient Case #2

Step 1: Define risk (cont)
- LDL-P or ApoB
  - Direct measure of atherogenic particles
  - Measures residual risk
  - Useful in positive family history
  - Useful in intermediate risk
    - Framingham 5-20%

When LDL-P is discordant, consideration should be given to intensifying LDL-C lowering therapy
- Discordance expected in:
  - Hypertriglyceridemia
  - Abdominal obesity
  - Metabolic Syndrome
  - Insulin resistance
  - Low HDL

Patient Case #2

- LDL-C & LDL-P discordance

For this patient, NMR reveals:

<table>
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<tr>
<th>Component</th>
<th>Value</th>
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<tr>
<td>CHOLESTEROL</td>
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<tr>
<td>TRIGLYCERIDES</td>
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<tr>
<td>HDL CHOLESTEROL</td>
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<td>LDL CHOLESTEROL</td>
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<tr>
<td>NON-HDL CHOLESTEROL</td>
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<tr>
<td>GLUCOSE</td>
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<tr>
<td>Total LDL-P</td>
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</table>
Patient Case #2

Step 1: Define risk (cont)
- Intermediate Risk
  - Consider other advanced testing
    - Lp(a)
    - Crp-hs
  - Consider imaging
    - CIMT
    - Coronary Calcium Scoring

Step 2: Prioritize / Set goals
- At least LDL-C <130mg/dL
- Consider LDL-C <100mg/dL
- Consider target LDL-P/ApoB <1000/<80

Step 3: Treatment Considerations
- Medication
  - Statins, Zetia, and BAS tend to lower LDL-C more
  - Niacin and Fibrates tend to lower LDL-P more
- Combination therapy may be necessary

Therapeutic Lifestyle Changes
- 10 week follow-up laboratory values

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<th>Component</th>
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<td>Total LDL-P</td>
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Cardiovascular Risk Reduction and Lipid Clinic

- Cardiovascular event risk reduction
- Medication intolerance
- Management of drug interactions
- Familial hypercholesterolemia
- Hypertriglyceridemia

To make an appointment: 888-293-7677