KDIGO, NKF, ISRNM, KfH, Baxter, BöhringerIngelheim, Reata Mitsubishi, Noxxon, Keryx, Abbott, Astellas, Fresenius, MSD, Amgen, Sanofi, BMS, AstraZeneca, Genzyme, Roche, Vifor, AMAG, research grant to the institution, consultancy, honoraria, sponsored education,
Objectives

Patterns of dyslipidemia in DKD

Lipid-lowering medications: are all statins the same?

Role of fibrates and HDL-raising therapies, e.g. CETP inhibitors

Novel lipid-lowering agents, e.g. PSCK9 inhibitors

KDIGO Clinical Practice Guideline for Lipid Management in Chronic Kidney Disease

VOLUME 3 | ISSUE 3 | NOVEMBER 2013
http://www.kidney-international.org
Dyslipidemia: 3 different languages

Apolipoproteins
- A-I
- A-II
- B-100
- C-I-III
- E

Lipoprotein particles

Lipids
- Triacylglyceride
- Cholesterolester
- Phospholipids
- Non-esterified Cholesterol

Kidney Disease: Improving Global Outcomes
**CKD:** TG-rich ApoB containing Lp Particles

Non-HDL Cholesterol

**Chylomicron**
- C-Remnant
- Atherogenicity -

**VLDL**
- d: 0.920

**IDL**
- d: 1.019

**LDL**
- d: 1.063

**HDL**
- d: 1.210

**Apo B**
- Apo B

**Apo A**

---

Kidney Disease: Improving Global Outcomes

Otvos J. Clin Cardiol 1999; 22 (Suppl II): 21-27
Kidney Disease: Improving Global Outcomes

Lipoprotein particles

<table>
<thead>
<tr>
<th></th>
<th>CKD</th>
<th>Healthy Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol</td>
<td>200 mg/dl</td>
<td>200 mg/dl</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>180 mg/dl</td>
<td>80 mg/dl</td>
</tr>
</tbody>
</table>

Kidney Disease: Improving Global Outcomes
Modulators of the lipid pattern

- Proteinuria & Nephrotic syndrome
- Peritoneal dialysis & uremic toxins & NS
- Hemodialysis & Heparin administration
- Immunosuppression & polypharmacy
- sHPT & LPL inhibitors
- Insulin resistance
- "method of determination of lipids"
## Pattern of dyslipidemia

<table>
<thead>
<tr>
<th></th>
<th>TC</th>
<th>LCL-C</th>
<th>HDL-C</th>
<th>TG</th>
<th>Lp(a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4D</td>
<td>216</td>
<td>125</td>
<td>34</td>
<td>224</td>
<td>32.6</td>
</tr>
<tr>
<td>AURORA</td>
<td>175</td>
<td>100</td>
<td>45</td>
<td>156</td>
<td></td>
</tr>
<tr>
<td>SHARP</td>
<td>189</td>
<td>107</td>
<td>43</td>
<td>203</td>
<td></td>
</tr>
<tr>
<td>ALERT</td>
<td>249</td>
<td>158</td>
<td>52</td>
<td>192</td>
<td></td>
</tr>
</tbody>
</table>

KDIGO

Kidney Disease: Improving Global Outcomes
Lipoprotein(a)
Lp(a) concentrations in nephrotic syndrome

- **Controls**: Median Lp(a) mg/dL
    - 9
  - Stenvinkel et al. Kidney Int 1993 n=31
    - 7

- **Nephrotic syndrome**: Median Lp(a) mg/dL
  - Kronenberg et al. Kidney Int 2004 n=207
    - 30
Lp(a) and kidney disease

Median Lp(a) mg/dL

- Controls: 6.9
- >90: 11.0
- 45-90: 18.4
- <45: 24.4
- Nephrotic: 29.9
- HD: 14.0
- CAPD: 19.9
- RTX: 8.0

GFR

J Clin Invest 91:397-401, 1993
Arterioscler Thromb 14:1399-1404, 1994
Kidney Int. 65: 606-612, 2004

KDIGO
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Role of fibrates and HDL-raising therapies, e.g. CETP inhibitors

Novel lipid-lowering agents, e.g. PSCK9 inhibitors
Lipid modifying agents

Lovastatin  
Simvastatin  
Atorvastatin  
Rosuvastatin  
Pravastatin  
Fluvastatin  
Pitavastatin

Niacin  
Ezetimibe  
Sevelamer  
Colestevelam  
Cholestyramine

Fenofibrate  
Bezafibrate  
Gemfibrozil

Combinations

Fish oil
Prospective Evaluation of Proteinuria and Kidney Function in diabetic (PLANET I) and non-diabetic (PLANET II) Patients with progressive Kidney Disease

Renal effects of atorvastatin and rosuvastatin in patients with diabetes who have progressive renal disease (PLANET I): a randomised clinical trial

*The Lancet Diabetes & Endocrinology, Available online 4 February 2015,*

Primary Endpoint: Proteinuria

325 patients with T1- or T2-Diabetes

Protein/creatinine (mg/g) on-treatment/baseline ratio

No. of patients
R10 107 98 91 107
R40 116 108 103 116
A80 102 92 81 102

KDIGO
Secondary Endpoint: Change in eGFR

![Graph showing change in eGFR over 52 weeks for different treatments]

- **Atorvastatin 80 mg**
- **Rosuvastatin 10 mg**
- **Rosuvastatin 40 mg**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No. of patients at 0 weeks</th>
<th>No. of patients at 26 weeks</th>
<th>No. of patients at 52 weeks</th>
<th>LOCF</th>
</tr>
</thead>
<tbody>
<tr>
<td>R10 107</td>
<td>107</td>
<td>100</td>
<td>93</td>
<td>107</td>
</tr>
<tr>
<td>R40 116</td>
<td>116</td>
<td>111</td>
<td>103</td>
<td>115</td>
</tr>
<tr>
<td>A80 102</td>
<td>102</td>
<td>92</td>
<td>84</td>
<td>101</td>
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</table>

KDIGO
Table 4 | Recommended doses (mg/d) of statins in adults with CKD

<table>
<thead>
<tr>
<th>Statin</th>
<th>eGFR G1-G2</th>
<th>eGFR G3a-G5, including patients on dialysis or with a kidney transplant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lovastatin</td>
<td>GP</td>
<td>nd</td>
</tr>
<tr>
<td>Fluvastatin</td>
<td>GP</td>
<td>80&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Atorvastatin</td>
<td>GP</td>
<td>20&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Rosuvastatin</td>
<td>GP</td>
<td>10&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Simvastatin/Ezetimibe</td>
<td>GP</td>
<td>20/10&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pravastatin</td>
<td>GP</td>
<td>40</td>
</tr>
<tr>
<td>Simvastatin</td>
<td>GP</td>
<td>40</td>
</tr>
<tr>
<td>Pitavastatin</td>
<td>GP</td>
<td>2</td>
</tr>
</tbody>
</table>

Lower doses than those used in major trials of statins in CKD populations may be appropriate in Asian countries.

1 ALERT, 2 4D, 3 AURORA, 4 SHARP
Objectives

Patterns of dyslipidemia in DKD

Lipid-lowering medications: are all statins the same?

Role of fibrates and HDL-raising therapies, e.g. CETP inhibitors

Novel lipid-lowering agents, e.g. PSCK9 inhibitors
Fibrates: Risk Benefits and Role in Treating Dyslipidemias

Min Jun and Vlado Perkovic
Fibrate characteristics

Fibrates effectively reduce triglycerides and elevate HDL-C targeting the residual CV risk

Jun et al. Lancet 2010;375:1875-84

Fibrates (most data with fenofibrate) raise serum creatinine (reversible process) and the mechanisms are still not settled (creatinine secretion, generation of vasodilatory prostaglandins, metabolic production). It does not translate to longer-term harm (FIELD and ACCORD trials) but potential benefits

Davis et al. Diabetologia 2010;54:280-90
Mychaleckyj et al. Diabetes Care 2012;35:1008-14
## Fibrates in CKD

<table>
<thead>
<tr>
<th>eGFR 30-59.9 ml/min/1.73m²</th>
<th>Fibrates</th>
<th>Placebo</th>
<th>Favours Fibrate</th>
<th>Risk Ratio; 95% CI</th>
<th>P for hetero b/t eGFR subgroups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular events</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VA-HIT</td>
<td>32</td>
<td>199</td>
<td>49</td>
<td>200</td>
<td>0.66 (0.44-0.98), p=0.04</td>
</tr>
<tr>
<td>FIELD</td>
<td>57</td>
<td>295</td>
<td>60</td>
<td>224</td>
<td>0.72 (0.53-0.99), p=0.04</td>
</tr>
<tr>
<td>Overall</td>
<td>89</td>
<td>494</td>
<td>109</td>
<td>424</td>
<td>0.70 (0.54-0.89), p=0.004</td>
</tr>
<tr>
<td>(I² = 0.0%, p for hetero=0.72)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VA-HIT</td>
<td>10</td>
<td>199</td>
<td>13</td>
<td>200</td>
<td>0.77 (0.35-1.72), p=0.529</td>
</tr>
<tr>
<td>FIELD</td>
<td>18</td>
<td>295</td>
<td>26</td>
<td>224</td>
<td>0.53 (0.30-0.94), p=0.028</td>
</tr>
<tr>
<td>Overall</td>
<td>28</td>
<td>494</td>
<td>39</td>
<td>424</td>
<td>0.60 (0.38-0.96), p=0.032</td>
</tr>
<tr>
<td>(I² = 0.0%, p for hetero=0.443)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR =60 ml/min/1.73m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VA-HIT</td>
<td>265</td>
<td>1065</td>
<td>329</td>
<td>1067</td>
<td>0.81 (0.70-0.93), p=0.002</td>
</tr>
<tr>
<td>FIELD</td>
<td>555</td>
<td>4600</td>
<td>623</td>
<td>4676</td>
<td>0.91 (0.81-1.01), p=0.07</td>
</tr>
<tr>
<td>Overall</td>
<td>820</td>
<td>5665</td>
<td>952</td>
<td>5743</td>
<td>0.86 (0.77-0.96), p=0.009</td>
</tr>
<tr>
<td>(I² = 40.4%, p for hetero=0.195)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

J Am Coll Cardiol. 2012;60:2061-71
Fibrates and metabolites accumulate with declining renal function. The risk of developing myopathy alone or in combination with statins (gemfibrocil>fenofibrate) increases.

Jones & Davidson AJ Cardiol 2005;95:120-2

KDIGO 2013 (and KDOQI 2003) recommended that fibrate treatment be avoided in patients with an eGFR < 30 ml/min/1.73m^2 and not used at all in combination with statins.
Cholesteryl ester transfer protein normally transfers cholesterol from HDL to VLDL or LDL. Inhibition of this process results in higher HDL levels. LDL levels are reduced as well as Lp(a) by ~40%.
Anacetrapib: REVEAL study ongoing (30,000 patients)

Studies have recently found low levels of anacetrapib in the blood of people who last took anacetrapib up to 4 years previously.

Evacetrapib: ACCELERATE study ongoing (12,000 patients) started 10/2012 (4 P MACE) (S-creatinine < 2.2 mg/dl)
### Changes in blood lipid and lipoprotein concentrations with CETP inhibitors

<table>
<thead>
<tr>
<th></th>
<th>Torcetrapib 60 mg daily</th>
<th>Dalcetrapib 600 mg daily</th>
<th>Anacetrapib 40 mg daily</th>
<th>Anacetrapib 150 mg daily</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>4%</td>
<td>n/a</td>
<td>1%</td>
<td>3%</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>-24%</td>
<td>-4%</td>
<td>-27%</td>
<td>-40%</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>-9%</td>
<td>-3%</td>
<td>-11%</td>
<td>-11%</td>
</tr>
<tr>
<td>Apo B</td>
<td>-12%</td>
<td>n/a</td>
<td>-20%</td>
<td>-29%</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>61%</td>
<td>25%</td>
<td>86%</td>
<td>139%</td>
</tr>
<tr>
<td>Apo A1</td>
<td>25%</td>
<td>10%</td>
<td>32%</td>
<td>47%</td>
</tr>
</tbody>
</table>
HDL Targeting Therapies


No. at Risk
Placebo 79077685 7498 7272 6959 6436 3650
Dalcetrapib 79107663 7402 7196 6871 6333 3599

Months

HDL Cholesterol (mg/dl)

0 1 3 6 12 24 36
HDL isolated from healthy individuals inhibited the production of inflammatory cytokines by peripheral monocytes and HDL from HD patients did not show this anti-inflammatory property. Many HDL samples even promoted the production of inflammatory cytokines.
HDL in children with CKD promotes endothelial dysfunction and an abnormal vascular phenotype.
Is HDL in CKD patients a toxic protein?

HDL Cholesterol Is Not Associated with Lower Mortality in Patients with Kidney Dysfunction

Stephen Zewinger,* Thimoteus Speer,* Marcus E. Kleber,† Hubert Scharnagl,‡ Rainer Woitas,§ Philipp M. Lepper,‖ Karolin Pfahler,∗ Sarah Seiler,* Gunnar H. Heine,* Winfried März, †‡‖ Günther Silbernagel,** and Danilo Fliser*
HDL and cardiovascular events

- C1: HDL-C ≤25 mg/dl
- HDL-C 26-33 mg/dl
- HDL-C 34-41 mg/dl
- HDL-C 42-49 mg/dl
- HDL-C ≥50 mg/dl

KDIGO
HDL Cholesterol, Apolipoproteins, and Cardiovascular Risk in Hemodialysis Patients

Günther Silber nagel,* Bernd Genser,†++§ Christiane Drechsler,§‖ Hubert Schar nagl,¶
Tanja B. Grammer,† Tatjana Stojakovic,¶ Vera Krane,§‖ Eberhard Ritz,** Christoph Wanner,§‖
and Winfried März†+++†+

- 1255 HD patients with T2DM
- 66 years, BMI 28 Kg/m2
- FU 4 years, 49 % death rate, 31% CV events
- HDL-C, Apo A1 and Apo C3 were not related to outcomes
- Inverse association of ApoA2 with mortality (HR 0.63, 95% CI 0.40-0.89)

JASN 2015 Feb;26:484-492.
Objectives

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Role of fibrates and HDL-raising therapies, e.g. CETP inhibitors

Novel lipid-lowering agents, e.g. PSCK9 inhibitors
Alirocumab and Evolocumab are fully human monoclonal antibodies against PCSK9. They reduce LDL-C by up to 65% and are well tolerated in randomized, placebo-controlled, phase 2 clinical trials up to 1 year in over 3000 hypercholesterolemic patients.1-7

5. J Am Coll Cardiol 2012;59:2344-53
A 52-Week Placebo-Controlled Trial of Evolocumab in Hyperlipidemia

Dirk J. Blom, M.D., Ph.D., Tomas Hala, M.D., Michael Bolognese, M.D., Michael J. Lillestol, M.D., Phillip D. Toth, M.D., Lesley Burgess, M.B., B.Ch., M.Med., Ph.D., Richard Ceska, M.D., Ph.D., Eli Roth, M.D., Michael J. Koren, M.D., Christie M. Ballantyne, M.D., Maria Laura Monsalvo, M.D., Kate Tsirtonis, M.Sc., Jae B. Kim, M.D., Rob Scott, M.D., Scott M. Wasserman, M.D., and Evan A. Stein, M.D., Ph.D., for the DESCARTES Investigators*

This article was published on March 29, 2014, at NEJM.org.
% Change LDL-C From Baseline

UC LDL-C Percent Change from Baseline, Mean (± SE)

Baseline Week 12 Week 52

-80
-70
-60
-50
-40
-30
-20
-10
0
10
20
30
40
50
60
70
80

Number of patients:

Baseline: 302, 599
Week 12: 294, 582
Week 52: 264, 542

Placebo QM (N = 302) Evolocumab 420 mg QM (N = 599)

Treatment difference 57%

Full analysis set, LDL by Ultracentrifugation
Other Lipids at Week 52

**ApoB**
- Percent Change from Baseline, Mean (%): 
  - Placebo QM: 2%
  - Evolocumab 420 mg QM: -42%

**Lp(a)**
- Percent Change from Baseline, Median (%): 
  - Placebo QM: -6% (-21 to 1)
  - Evolocumab 420 mg QM: -28% (-49 to -6)

**HDL-C**
- Percent Change from Baseline, Mean (%): 
  - Placebo QM: 6%
  - Evolocumab 420 mg QM: 2%

**ApoA1**
- Percent Change from Baseline, Mean (%): 
  - Placebo QM: 3% (-49 to -6)
  - Evolocumab 420 mg QM: -28% (-49 to -6)

**Triglycerides**
- Percent Change from Baseline, Median (%): 
  - Placebo QM: 3%
  - Evolocumab 420 mg QM: -9% (-26 to 13)

Error bars represent standard error. Data in parentheses represent Q1 to Q3.
PCSK9 is elevated in CKD and proteinuria

## ODYSSEY LONG TERM

### Difference (alirocumab vs. placebo) in LDL-C % Change from Baseline to W24

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Alirocumab</th>
<th>Placebo</th>
<th>Interaction p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>n=1530</td>
<td>n=780</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -61.0</td>
<td>LS mean 0.8</td>
<td></td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>n=1419</td>
<td>n=722</td>
<td>0.2227</td>
</tr>
<tr>
<td></td>
<td>LS mean -61.6</td>
<td>LS mean 0.8</td>
<td></td>
</tr>
<tr>
<td>Black/African American</td>
<td>n=53</td>
<td>n=24</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -49.5</td>
<td>LS mean 4.3</td>
<td></td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td>0.1313</td>
</tr>
<tr>
<td>&lt; 65</td>
<td>n=963</td>
<td>n=498</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -60.4</td>
<td>LS mean -0.1</td>
<td></td>
</tr>
<tr>
<td>65 to &lt; 75</td>
<td>n=442</td>
<td>n=221</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -62.9</td>
<td>LS mean 3.0</td>
<td></td>
</tr>
<tr>
<td>≥ 75</td>
<td>n=125</td>
<td>n=61</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -59.5</td>
<td>LS mean 0.5</td>
<td></td>
</tr>
<tr>
<td><strong>BMI</strong></td>
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<tr>
<td>&lt; 30</td>
<td>n=852</td>
<td>n=406</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -61.5</td>
<td>LS mean 1.3</td>
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<tr>
<td>≥ 30</td>
<td>n=674</td>
<td>n=371</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -60.3</td>
<td>LS mean 0.1</td>
<td></td>
</tr>
<tr>
<td><strong>Moderate CKD</strong></td>
<td></td>
<td></td>
<td>0.0210</td>
</tr>
<tr>
<td>Yes</td>
<td>n=73</td>
<td>n=174</td>
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<tr>
<td></td>
<td>LS mean -62.0</td>
<td>LS mean 8.7</td>
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<td>No</td>
<td>n=1356</td>
<td>n=707</td>
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<tr>
<td></td>
<td>LS mean -60.9</td>
<td>LS mean 0.0</td>
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</tr>
<tr>
<td><strong>Diabetes</strong></td>
<td></td>
<td></td>
<td>0.0957</td>
</tr>
<tr>
<td>Yes</td>
<td>n=545</td>
<td>n=273</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -60.0</td>
<td>LS mean -1.0</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>n=985</td>
<td>n=507</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -61.6</td>
<td>LS mean 1.8</td>
<td></td>
</tr>
<tr>
<td><strong>Baseline fasting TGs, mg/dL</strong></td>
<td></td>
<td></td>
<td>0.3431</td>
</tr>
<tr>
<td>&lt; 150</td>
<td>n=928</td>
<td>n=450</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -58.8</td>
<td>LS mean 2.2</td>
<td></td>
</tr>
<tr>
<td>≥ 150</td>
<td>n=602</td>
<td>n=329</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LS mean -64.5</td>
<td>LS mean -1.1</td>
<td></td>
</tr>
</tbody>
</table>

All patients on background of maximally tolerated statin ± other lipid-lowering therapy
Controversy & Summary

• Outcomes of the CKD patients within the CETP inhibitor trials are of interest

• Subcategories of CKD patients may show an unmet need for fibrates and PCSK9 inhibitors or a combination of these drugs

• Lipoprotein(a) may become a new target in CKD patients, especially in proteinuric or nephrotic syndrome patients
Thank you ☺

Kidney Disease: Improving Global Outcomes
A Controlled, Prospective Study of the Effects of Atorvastatin on Proteinuria and Progression of Kidney Disease

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<table>
<thead>
<tr>
<th></th>
<th>Start of Run-In Phase</th>
<th>Start of Study Phase</th>
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</thead>
<tbody>
<tr>
<td>No. of patients (M/F)</td>
<td>56 (38/18)</td>
<td>—</td>
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<tr>
<td>Age</td>
<td>55.6 ± 1</td>
<td>—</td>
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<tr>
<td>BMI (w/h²)</td>
<td>27.6 ± 0.26</td>
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<tr>
<td>Hypertension (yes/no)</td>
<td>27/29</td>
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<tr>
<td>Office systolic BP (mm Hg)</td>
<td>144.3 ± 2.4</td>
<td>133.0 ± 1.0</td>
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<tr>
<td>Office diastolic BP (mm Hg)</td>
<td>93.3 ± 1.8</td>
<td>84.8 ± 0.8</td>
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<tr>
<td>CrCl (mL/min)</td>
<td>55.5 ± 1.4</td>
<td>50.4 ± 1.3</td>
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<tr>
<td>UPE (g/24 h)</td>
<td>2.7 ± 0.1</td>
<td>2.2 ± 0.1</td>
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<tr>
<td>Total cholesterol (mg/dL)</td>
<td>320.4 ± 4.7</td>
<td>310.6 ± 3.3</td>
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<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>189 ± 5</td>
<td>198 ± 4.1</td>
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<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>36.2 ± 0.7</td>
<td>36.1 ± 0.6</td>
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<tr>
<td>Serum albumin (g/dL)</td>
<td>3.35 ± 0.06</td>
<td>3.30 ± 0.06</td>
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Controversy & Summary

- Do new treatments provide the opportunity to go from moderate intensity to high intensity LDL lowering in CKD patients?
- Acute fibrate-induced creatinine elevation in T2DM with relatively preserved renal function may confer longer-term renoprotective effects.
- The correction of the abnormal HDL composition and the improvement of its vasoprotective properties remains to be shown.