The Metabolic Syndrome

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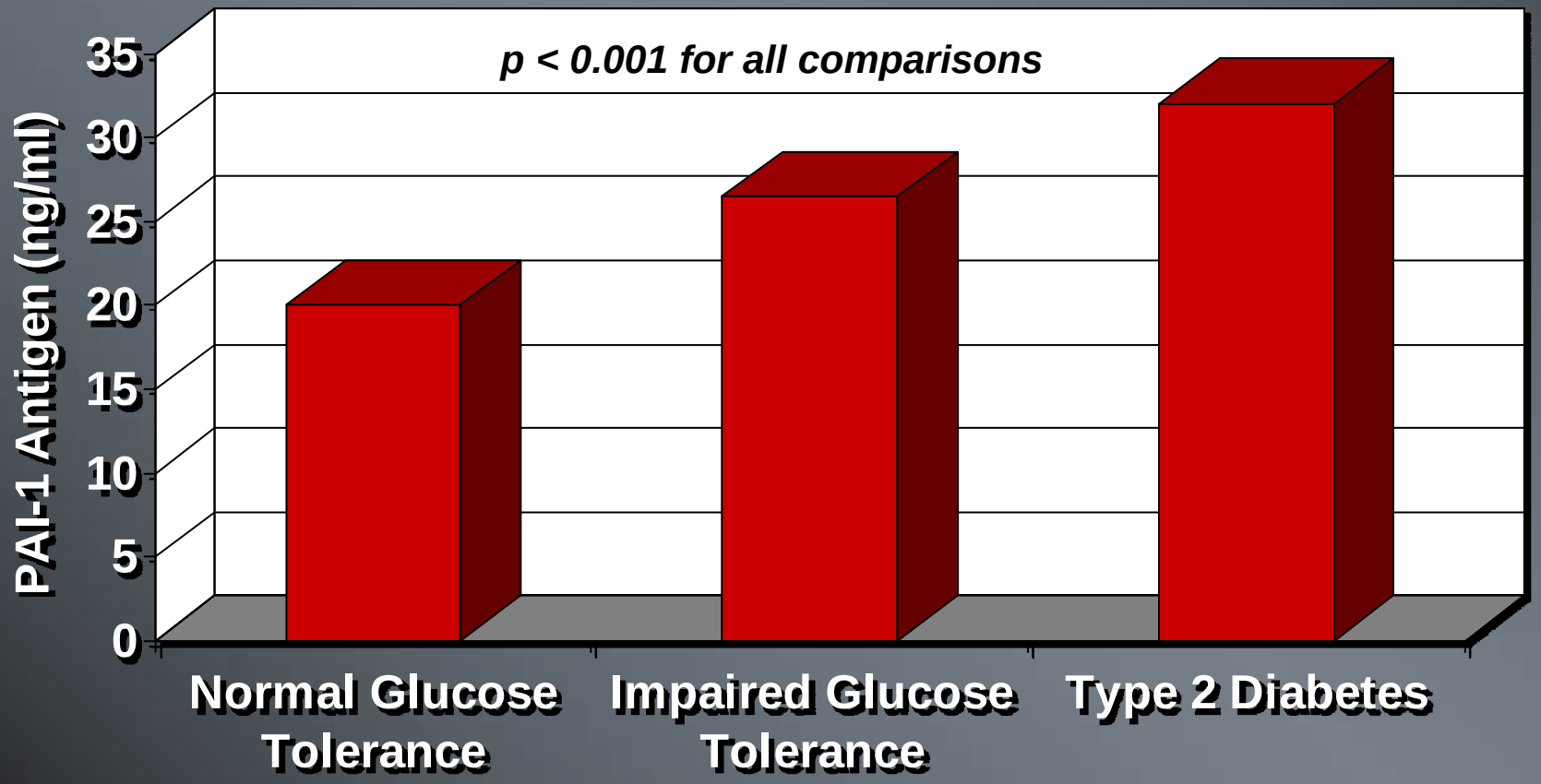
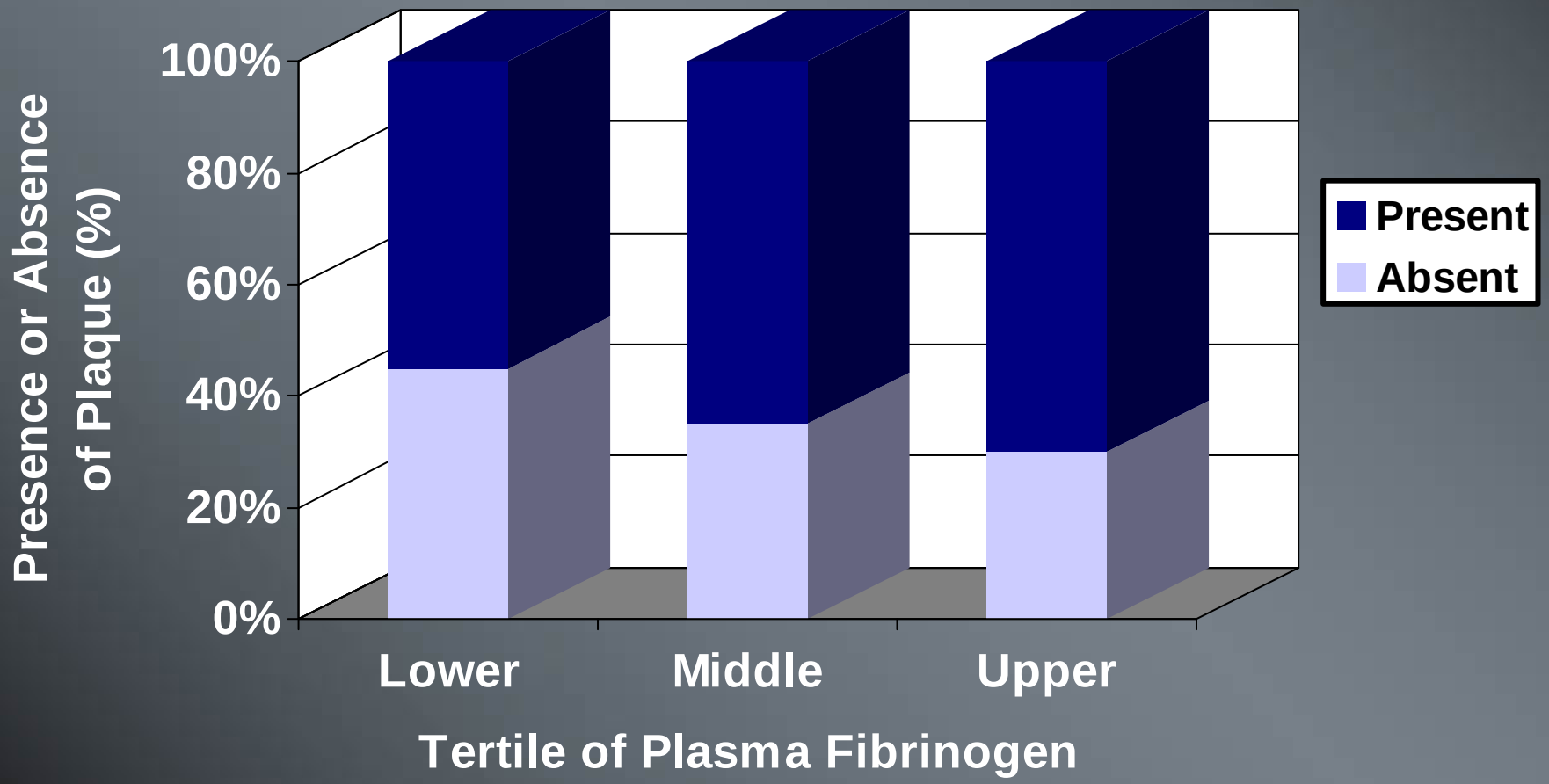
PAI-1 Levels and Glucose Tolerance



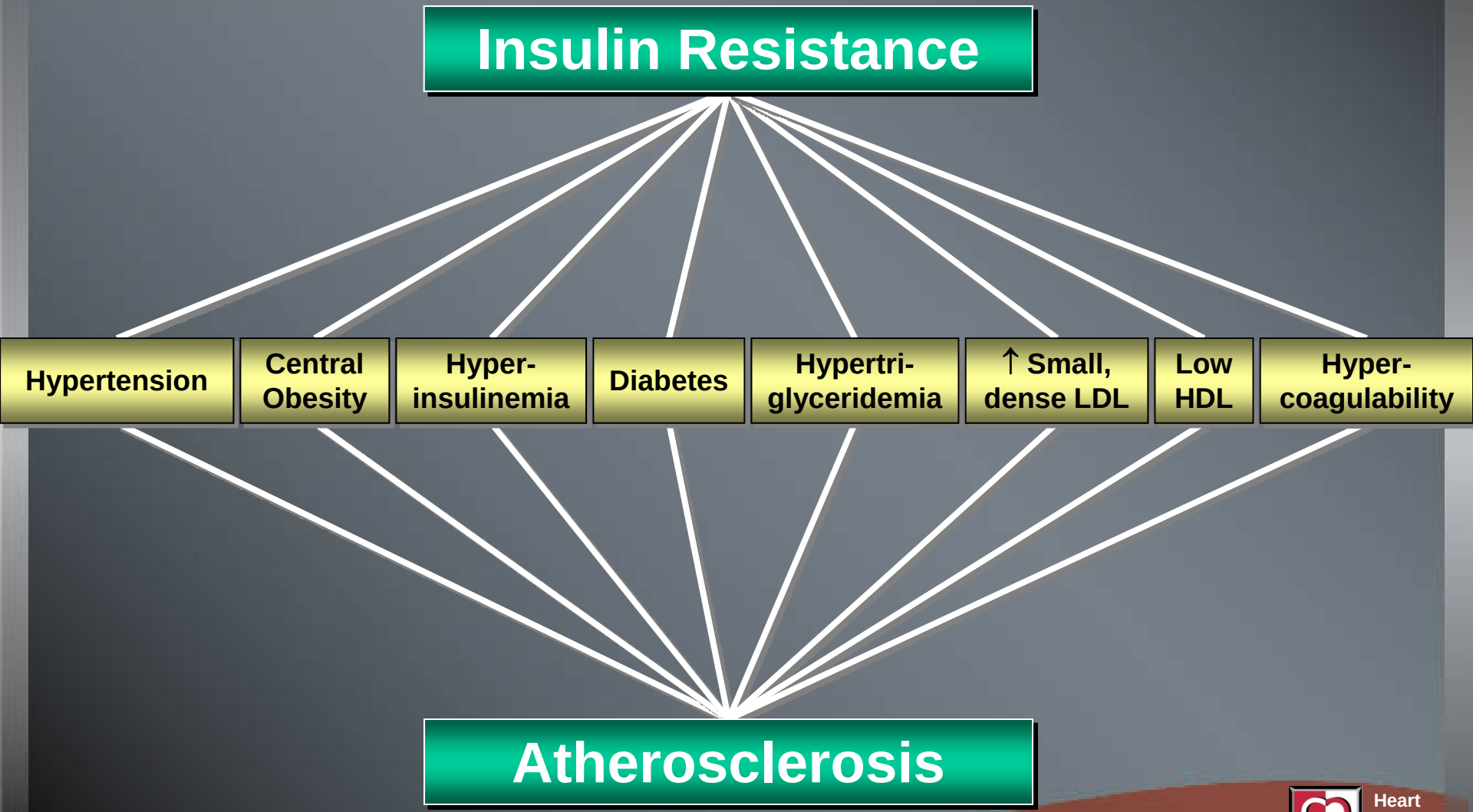
Festa A, et al. *Arterioscler Thromb Vasc Biol*. 1999;19:562–568

Fibrinogen and Atherosclerosis

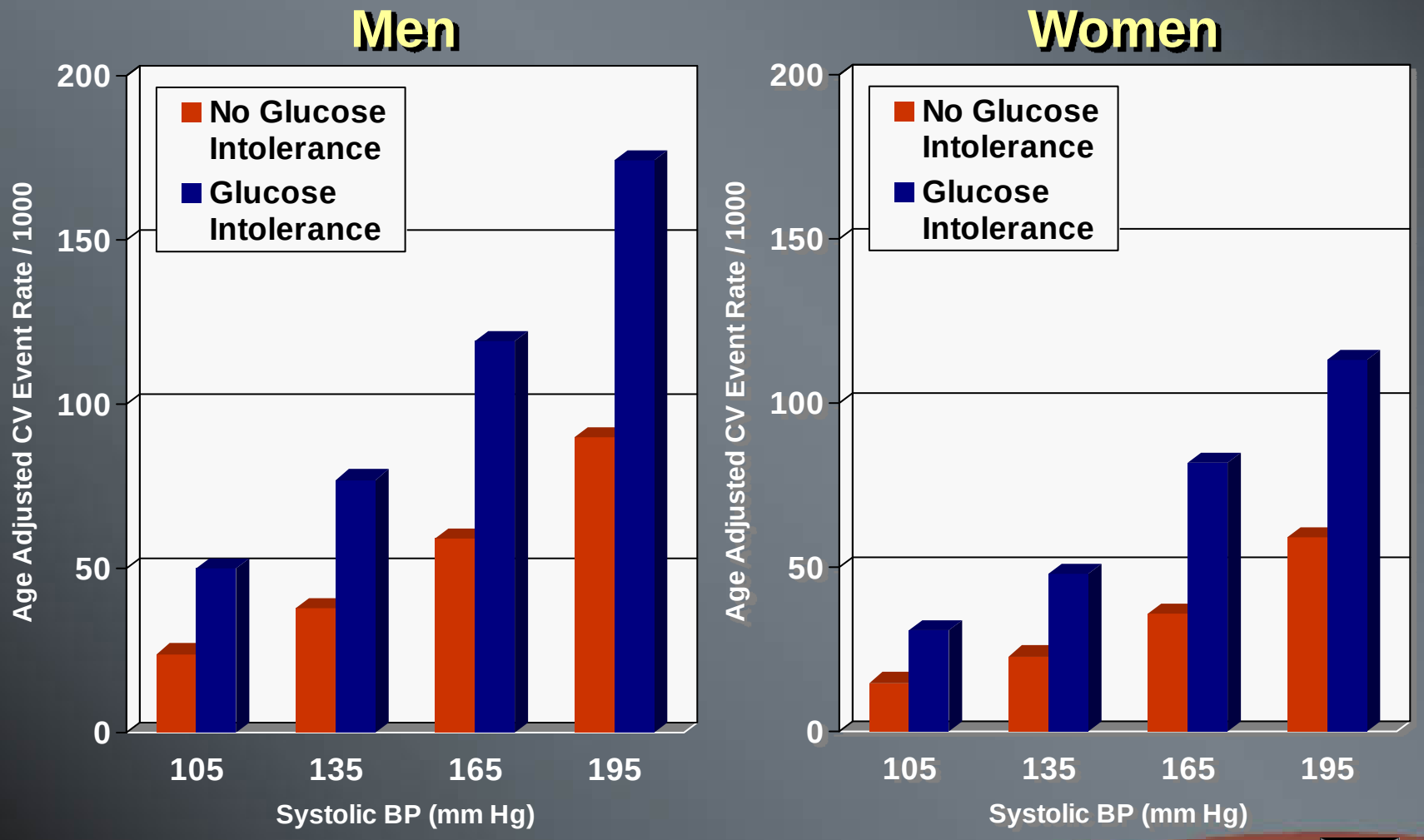


Levenson J, et al. *Arterioscler Thromb Vasc Biol.* 1995;15:1263-1268. Reproduced with permission.

Insulin Resistance and Atherosclerosis

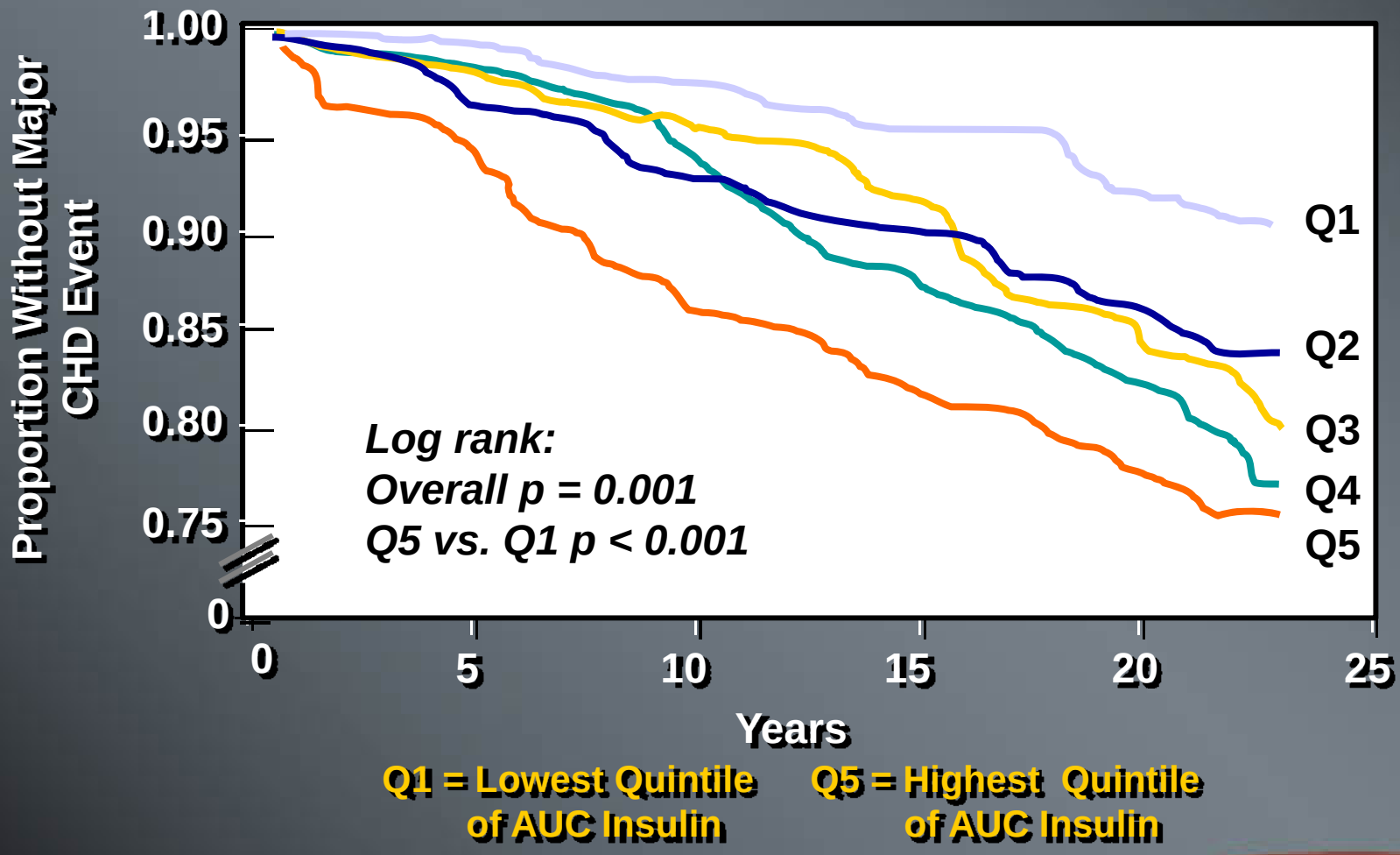


Hypertension, Glucose Intolerance and CHD



Kannel WB, et al. *Am Heart J.* 1991;121:1268-1273.

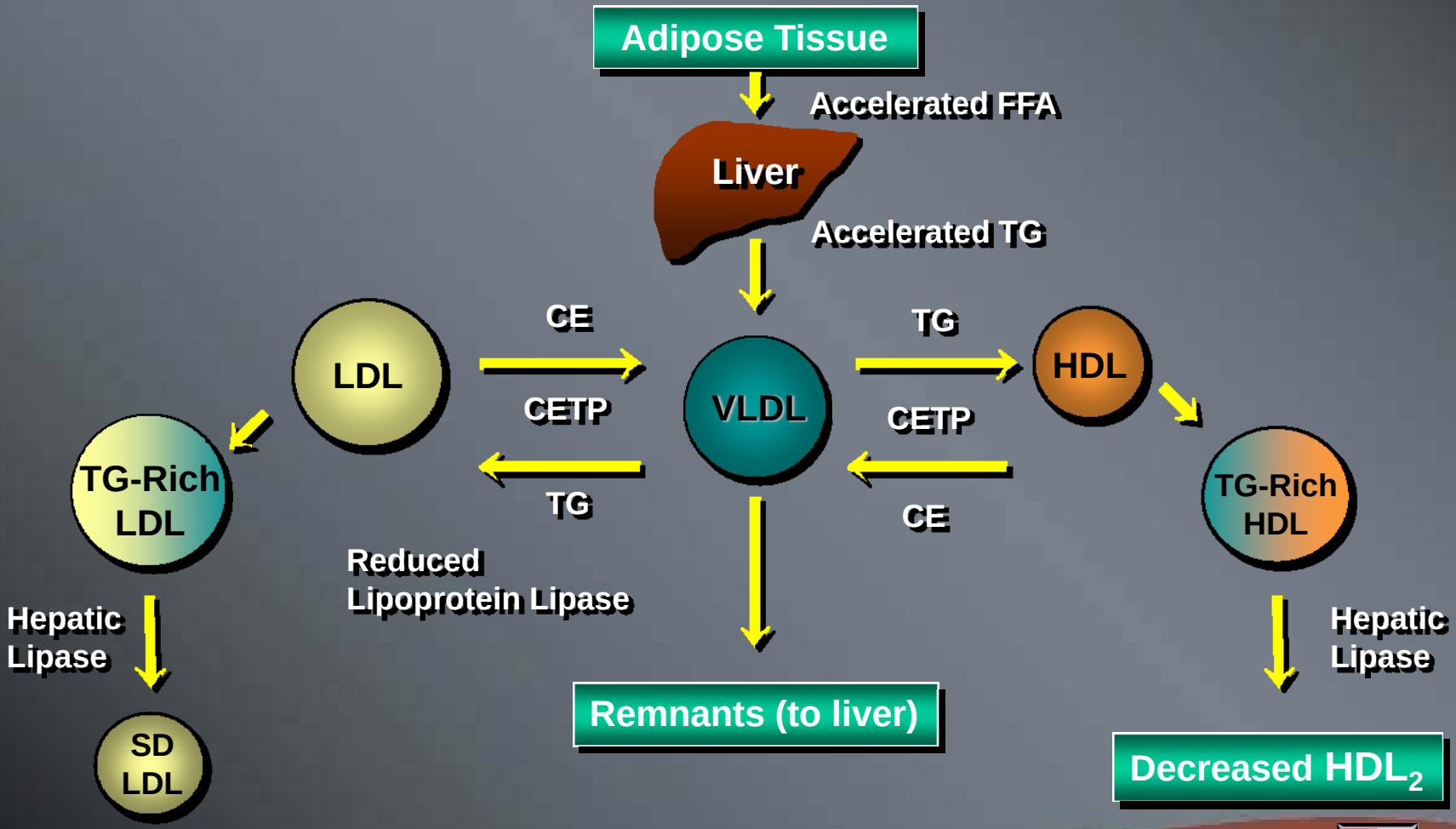
Hyperinsulinemia and CHD



Pyörälä M, et al. *Circulation.* 1998;98:398-404.

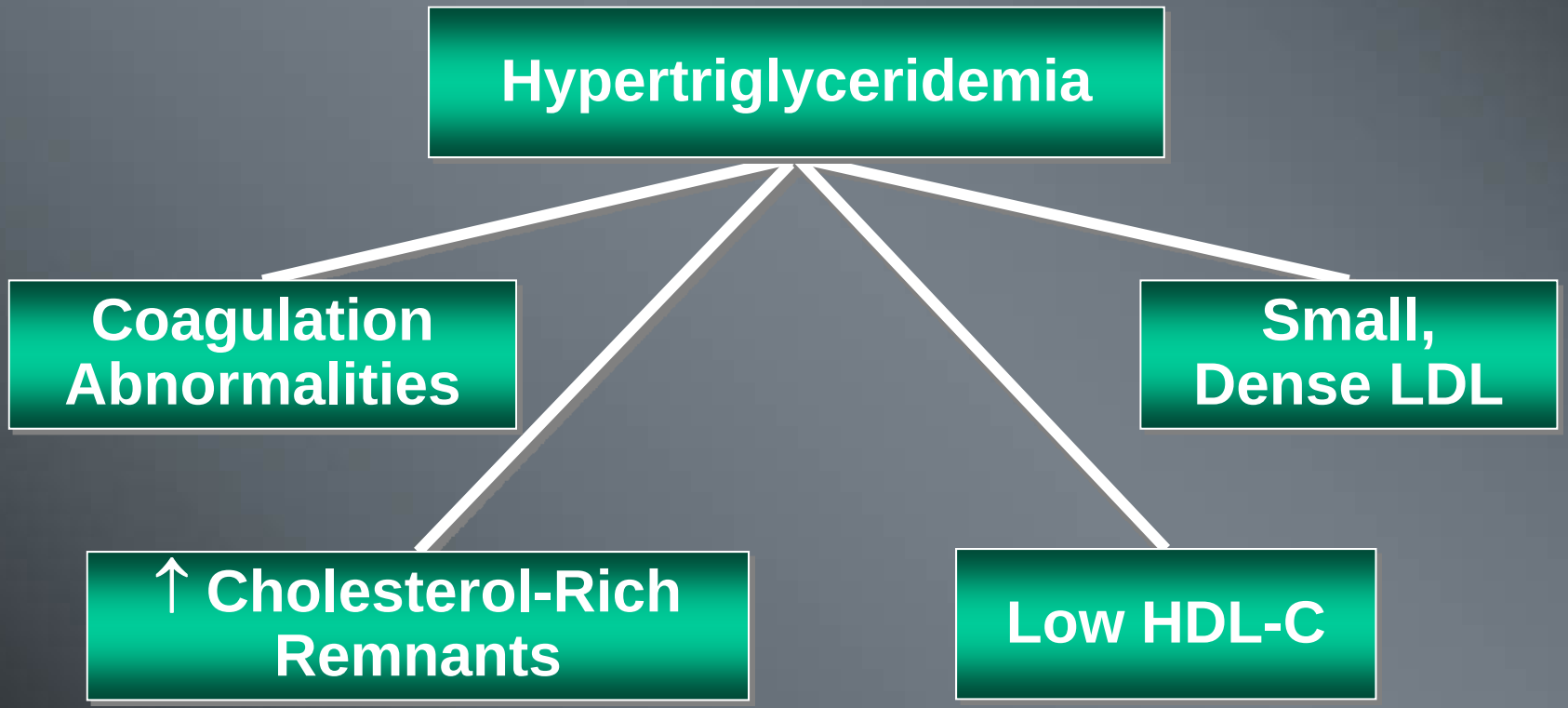
Insulin Resistance

Postprandial Lipid Metabolism



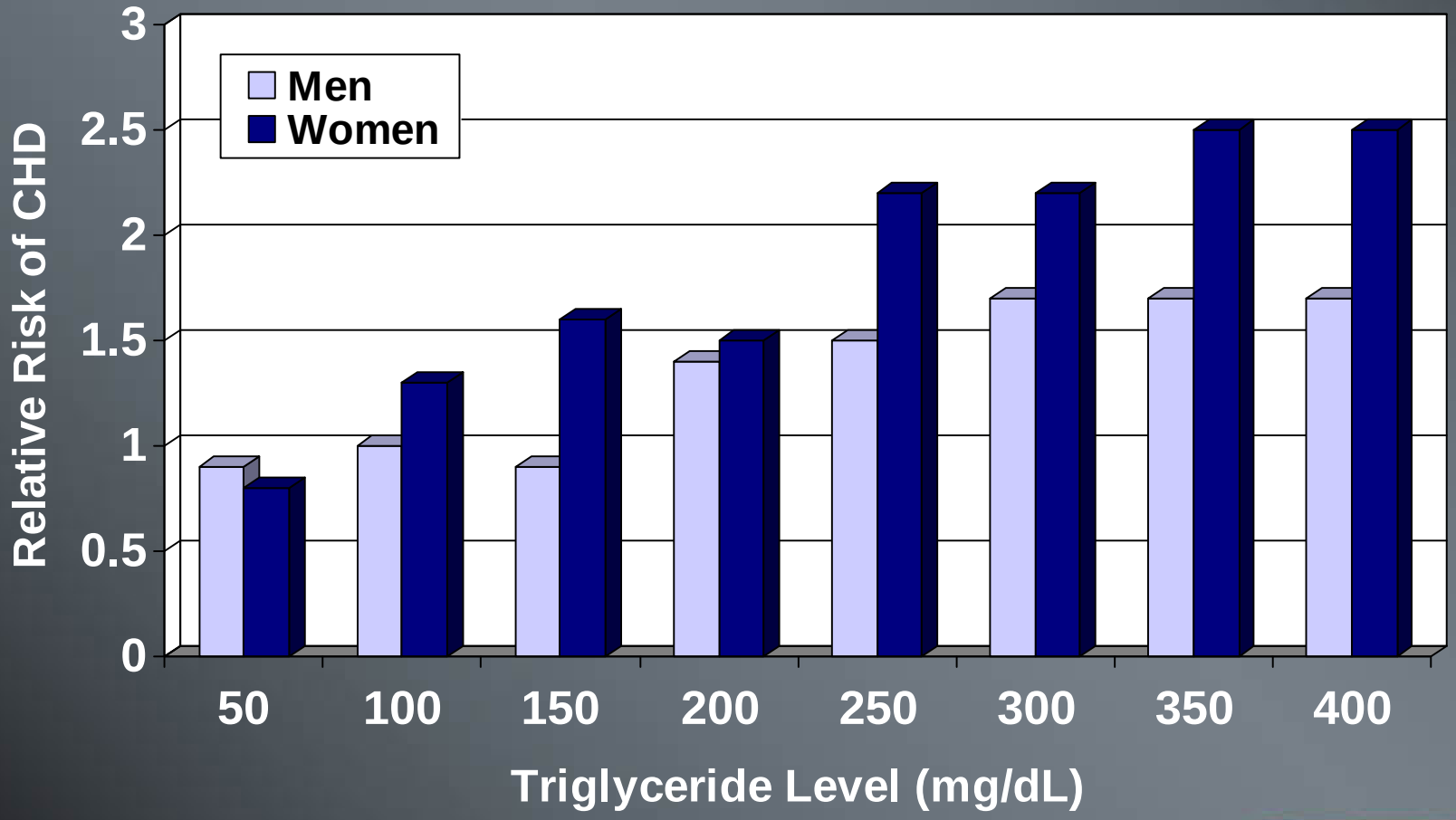
Hypertriglyceridemia

Atherogenic Changes



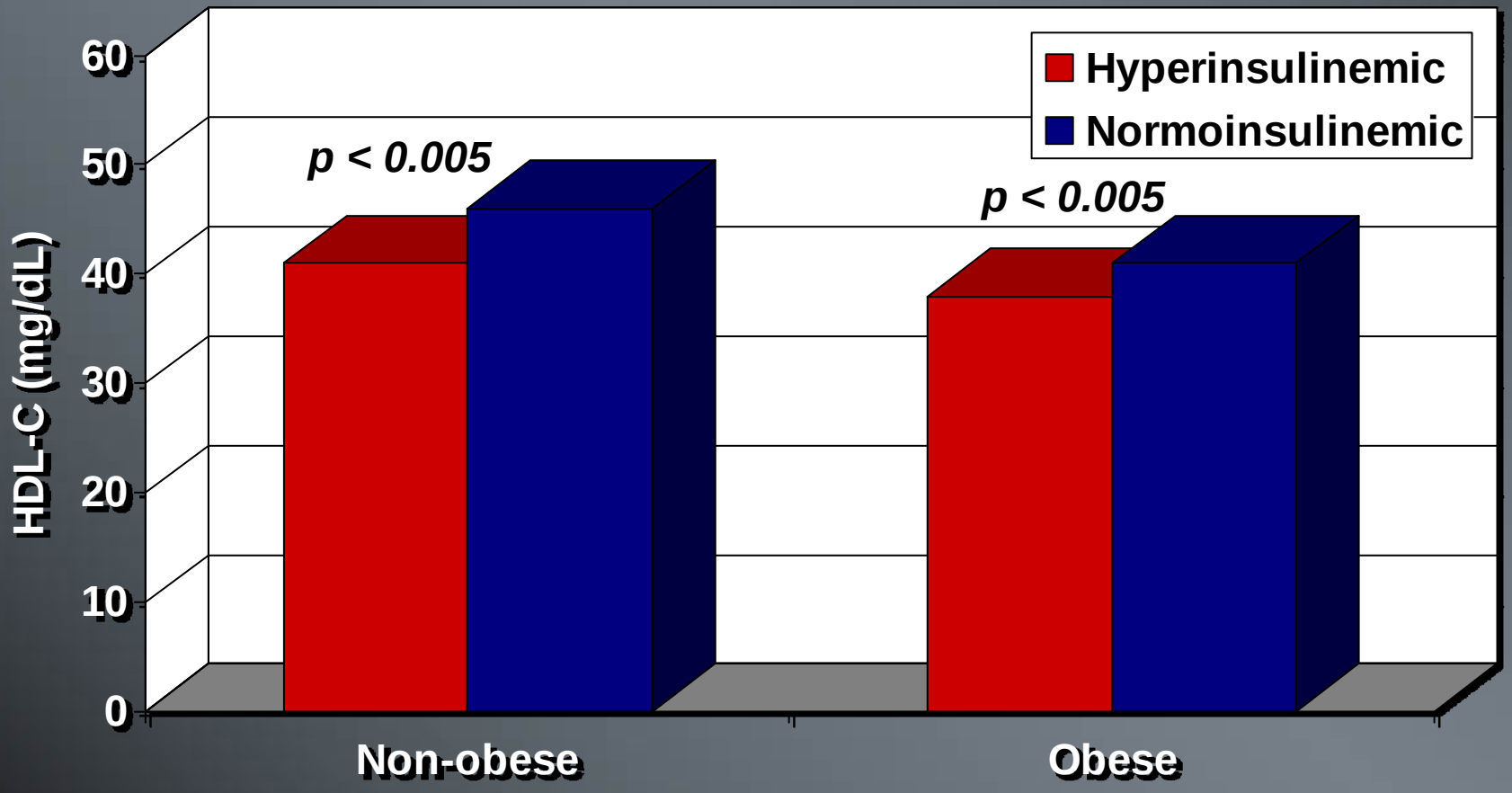
Miller M. *Eur Heart J.* 1998;19(Suppl H):H18-H22.
Reproduced with permission from Harcourt Publishers Limited.

Risk of CHD by Triglyceride Level



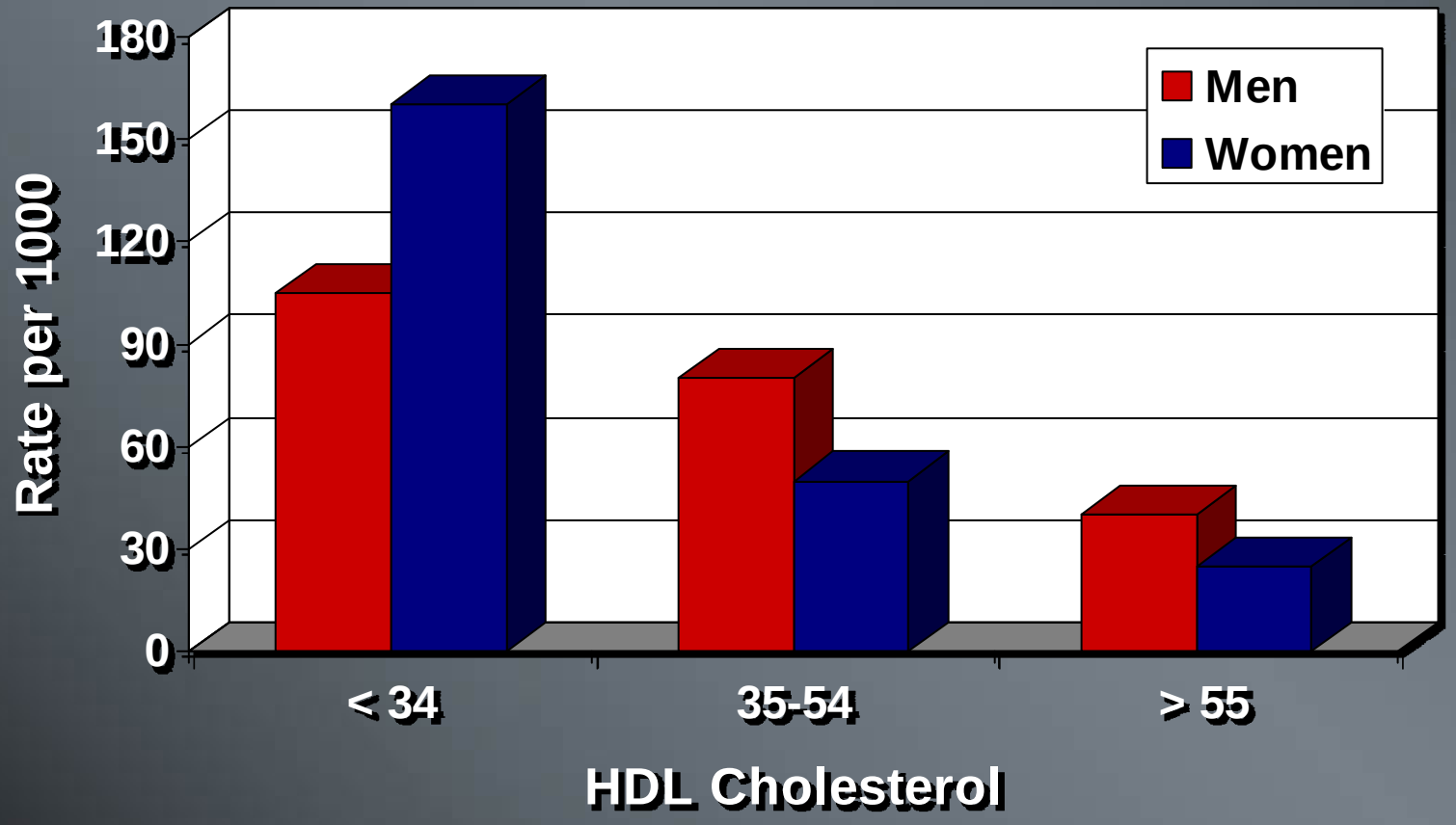
Castelli WP. *Am J Cardiol.* 1992;70:3H-9H.
Reproduced with permission from Elsevier Science.

Hyperinsulinemia and Low HDL



Zavaroni I, et al. *J Intern Med*. 1994;235:51-56

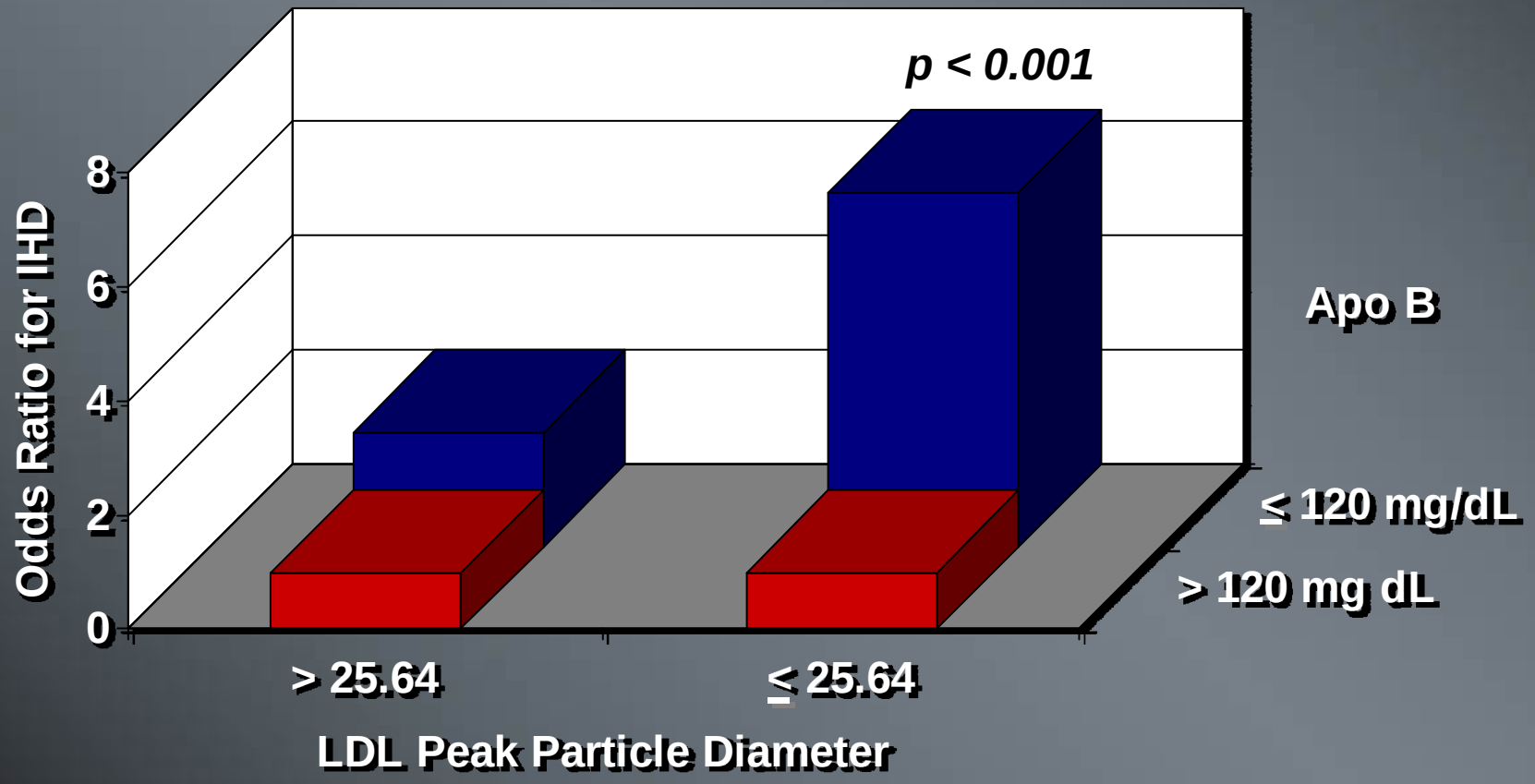
Cardiovascular Disease and HDL-C Levels



Kannel WB, *Am J Cardiol.* 1983;52:9b-12b.

Ischemic Heart Disease Risk

LDL Particle Diameter and Apo-B Levels



Lamarche B, et al. *Circulation.* 1997;95:69-75.

Drug Therapy for Diabetic Dyslipidemia

Mechanisms of Action

Nicotinic acid:

- ↓ hepatic production of VLDL
- ↑ HDL cholesterol levels; relatively contraindicated because it may increase insulin resistance

HMG CoA reductase inhibitors (statins):

- ↓ cholesterol synthesis by inhibition of HMG CoA reductase, resulting in ↓ lipoprotein formation and ↑ LDL receptor synthesis

Bile acid sequestrants:

- Promote removal of LDL cholesterol from circulation by stimulating hepatic LDL receptor synthesis

Fibric acid derivatives (fibrates):

- ↓ hepatic production of VLDL triglycerides
- ↑ lipolysis of serum triglycerides by ↑ lipoprotein lipase activity
- ↑ HDL levels

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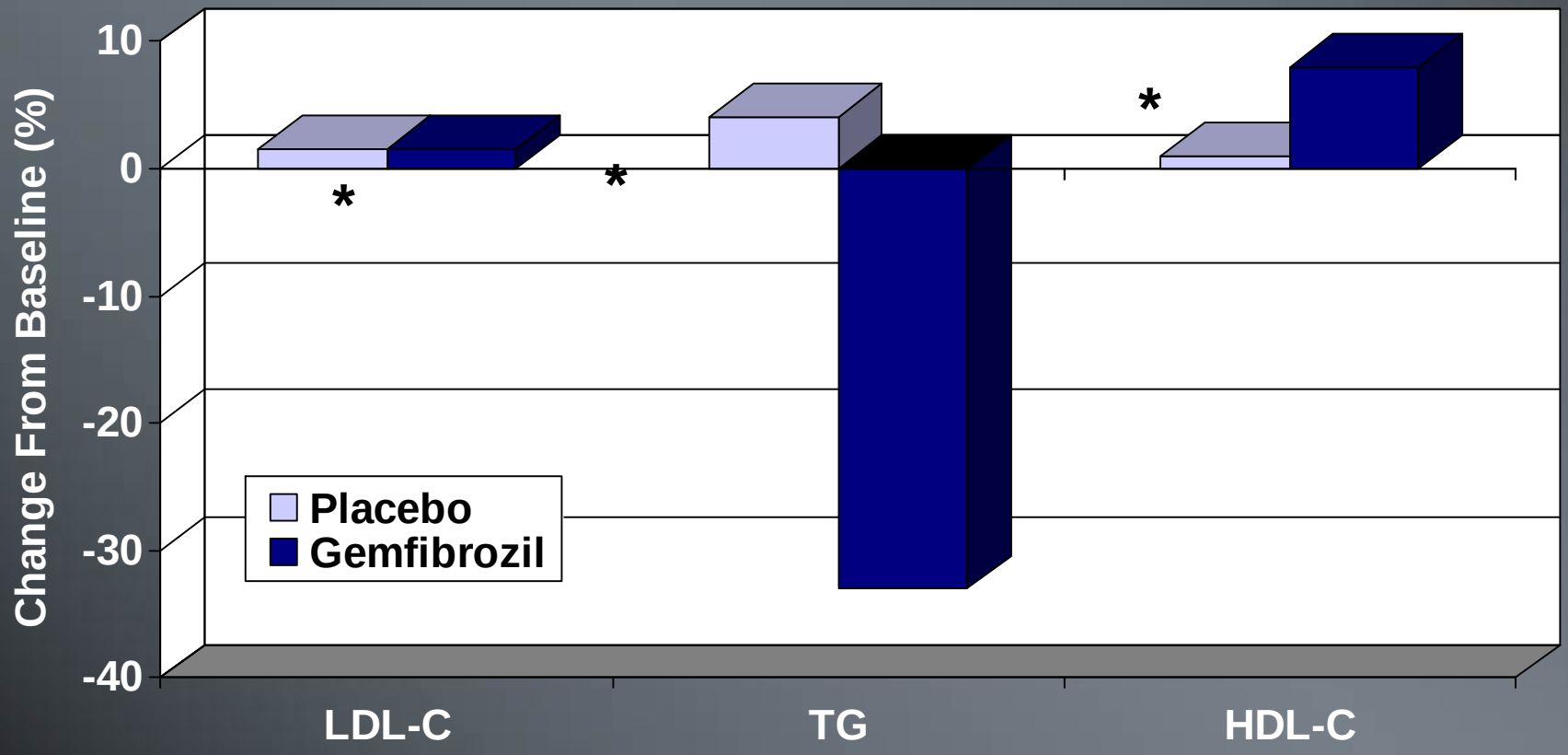
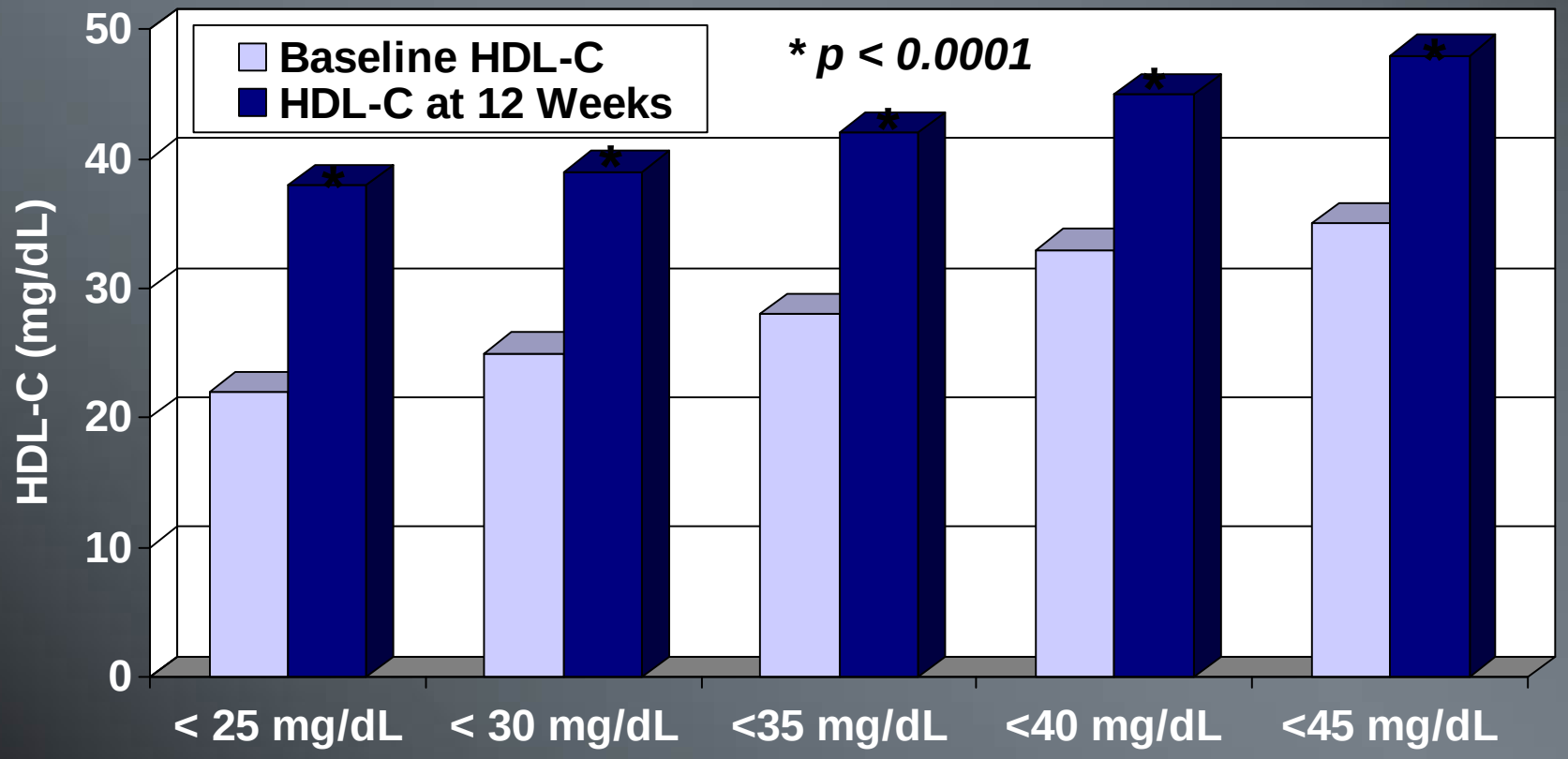
Change in Lipoproteins From Baseline



Rubins HB, et al. *N Engl J Med*. 1999;341:410-418.

Fenofibrate and HDL-C



Poulter N. *Brit J Cardiol* 1999; 6(12): 682–685

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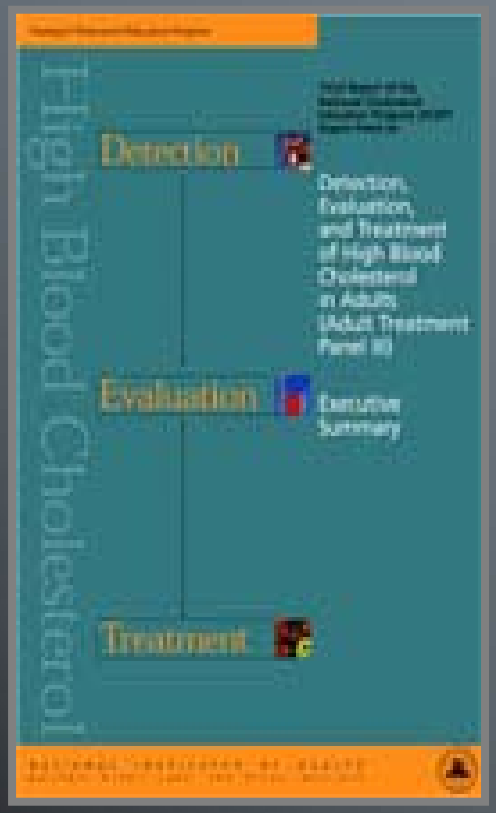
Third Report of the National Cholesterol Education Program (NCEP)



- NCEP's Adult Treatment Panel (ATP) III provides updated clinical guidelines for cholesterol testing and management
- ATP III expands the indications for intensive cholesterol-lowering therapy in clinical practice
- It addresses the metabolic syndrome as a secondary target of risk-reduction therapy, after the primary target, LDL cholesterol

Third Report of the National Cholesterol Education Program Expert Panel. Executive Summary.
NIH Publication No. 01-3670. May 2001.

Dyslipidemia in the Metabolic Syndrome

Summary

- Dyslipidemia in the metabolic syndrome is a CHD risk factor and is characterized by elevated triglyceride, reduced HDL, and an increased incidence of small, dense LDL particles

- For patients with LDL >130 mg/dL, treat with a statin first, then assess triglyceride and HDL levels to determine if a fibrate or niacin* is needed; for patients with LDL <130 mg/dL, a fibrate or niacin is first-line therapy when HDL is <40 mg/dL, then reassess the LDL level to determine if a statin is needed

* Niacin should be used with caution in this patient group because of its negative effect on insulin sensitivity and blood glucose levels.

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