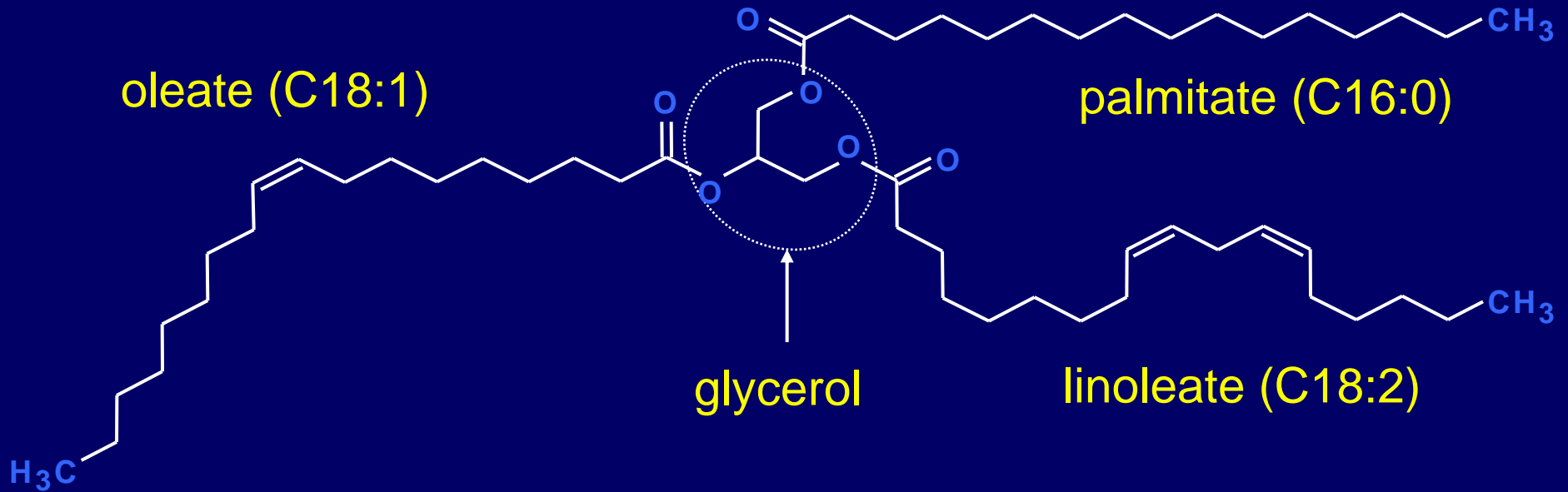


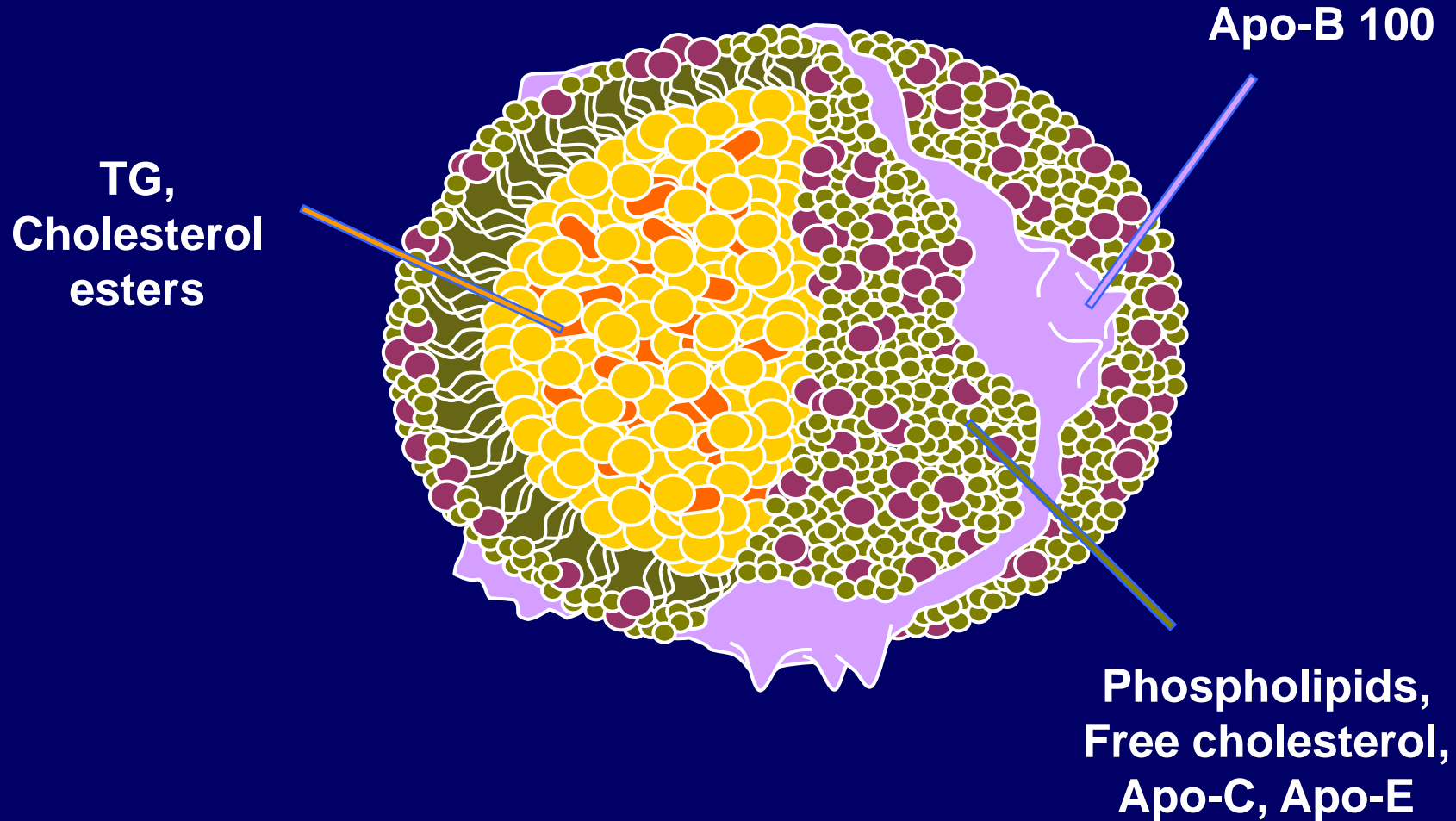
# Responses of blood lipids to aerobic and resistance type of exercise

**Labros Sidossis, Ph.D.**  
**Laboratory of Nutrition and Clinical Dietetics**  
**Harokopio University of Athens, Greece**

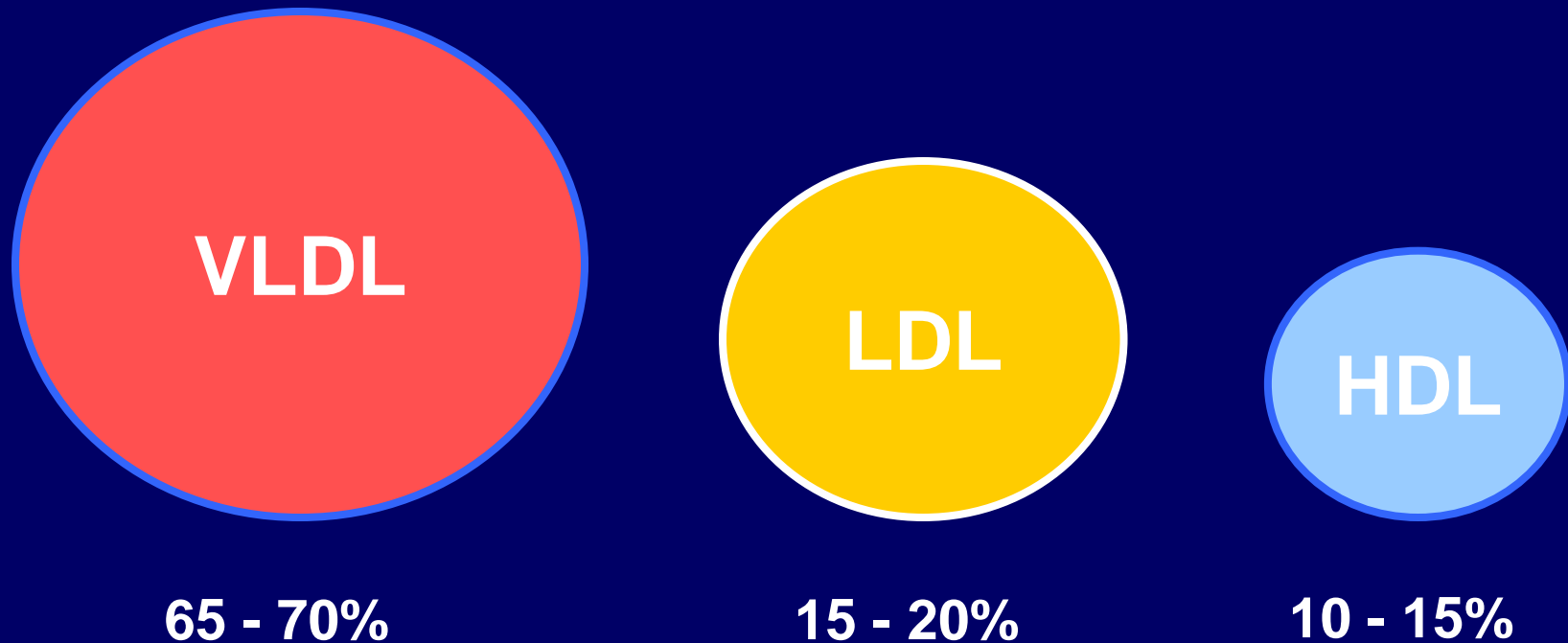
# Triacylglycerol structure



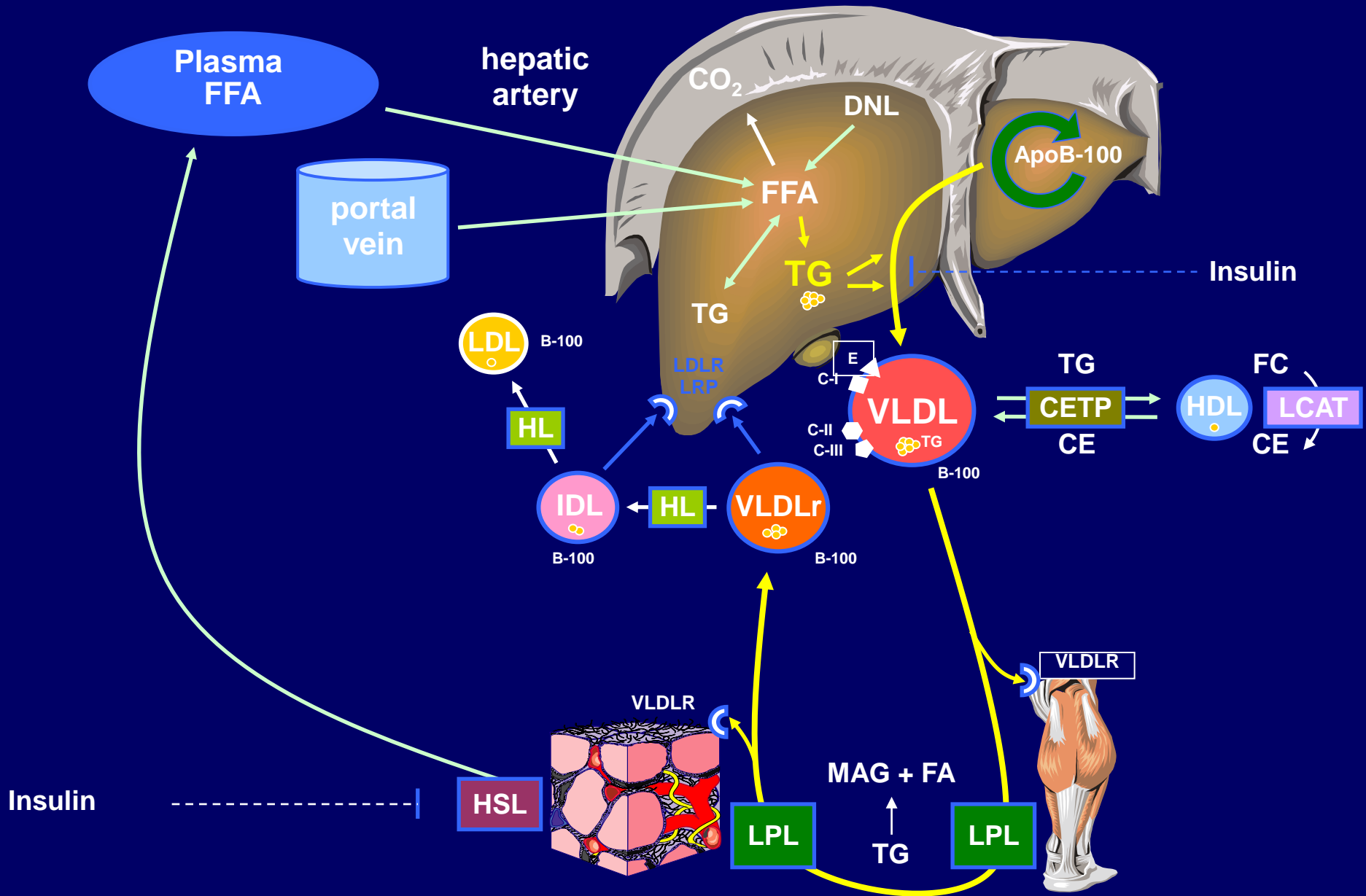
# Very low density lipoproteins (VLDL)



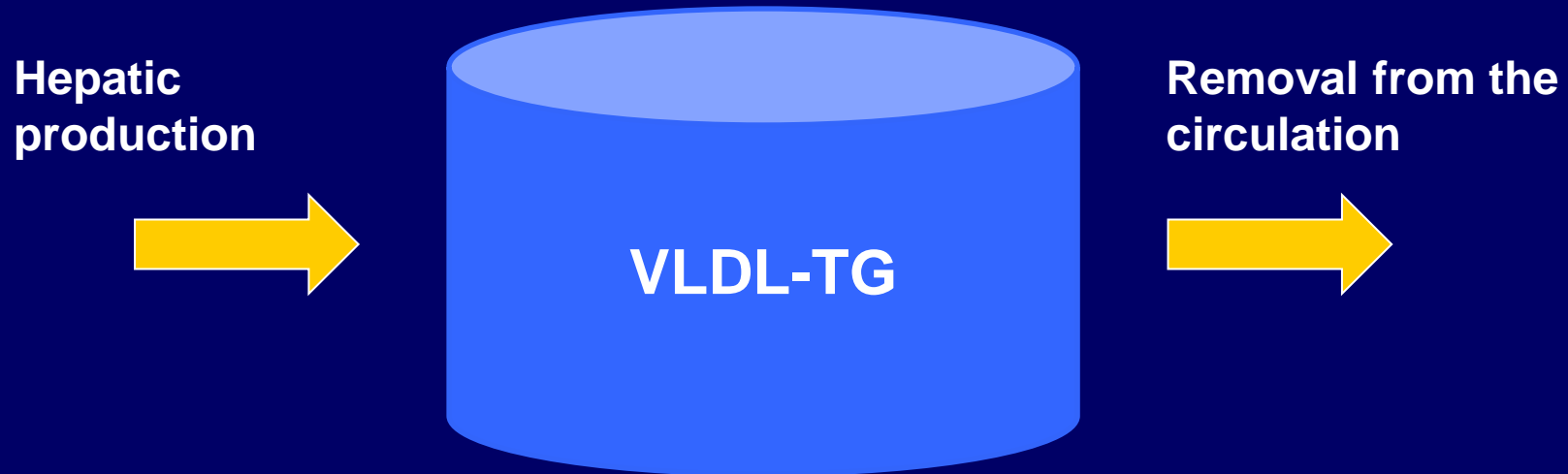
# TG transport in blood (fasting state)



# VLDL metabolism



# Regulation of plasma VLDL-TG concentration

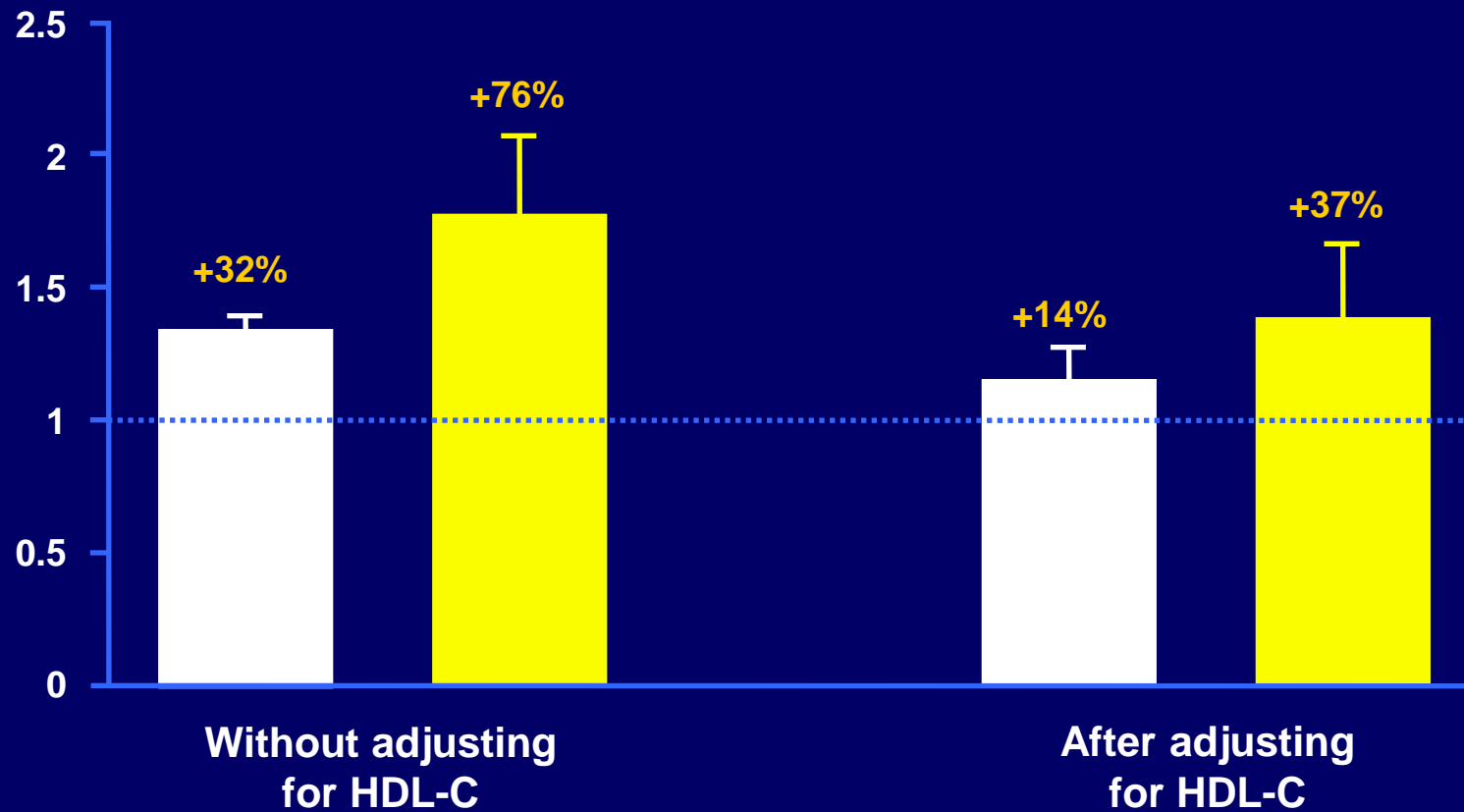


# Triglycerides and CVD

# TG and Cardiovascular Disease (Fasting State)

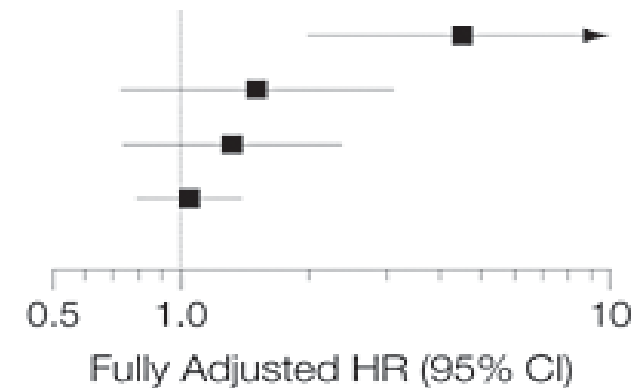
CHD, relative risk  
(for an increase in plasma  
TG by 1 mM or 86 mg/dl)

Men (n = 46413)  
Women (n = 10864)



# TG and Cardiovascular Disease (Stratified by Time Since Last Meal)

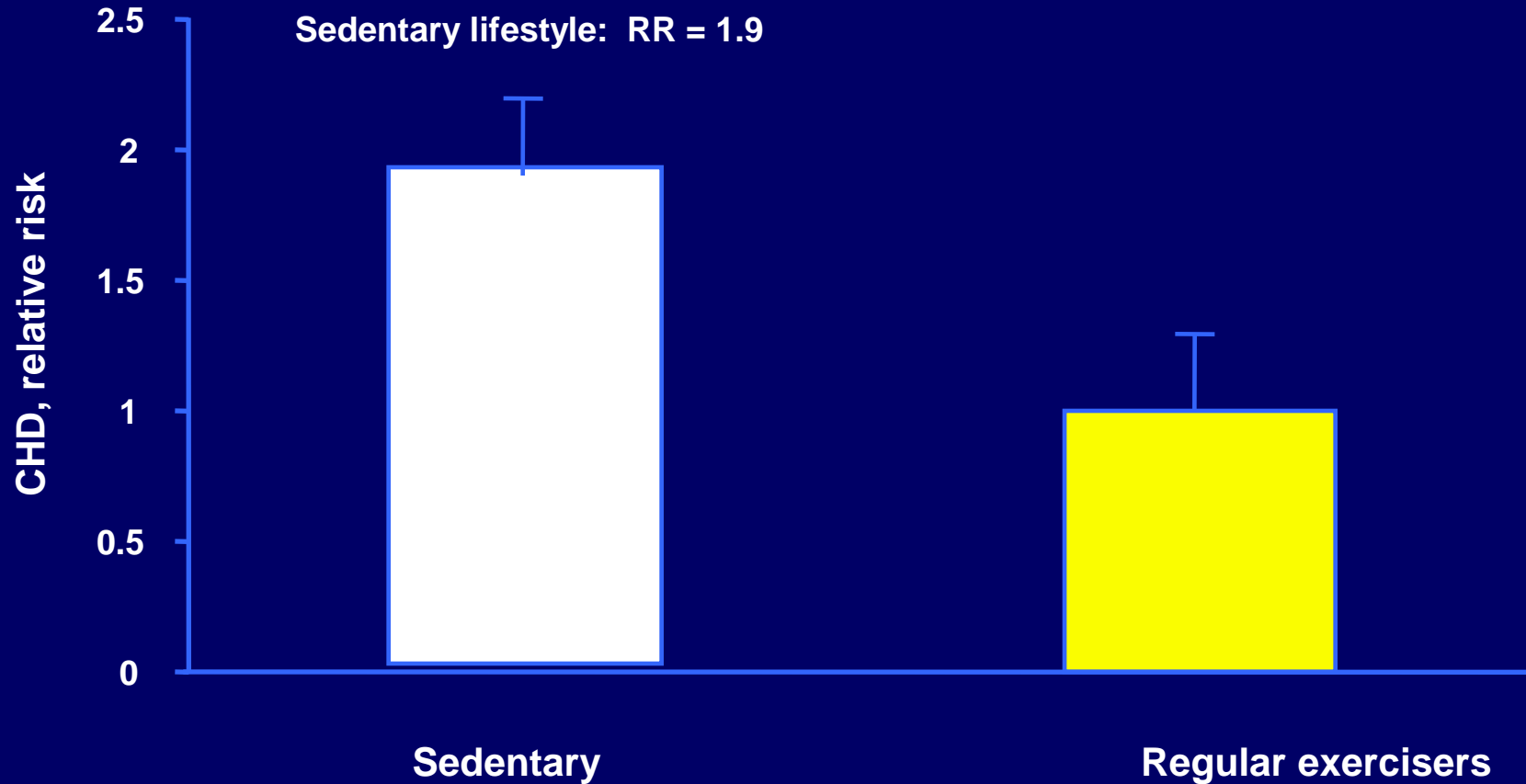
Time From Last Meal, h	No. of Participants	No. of Events	Hazard Ratio (95% Confidence Interval)
2 to <4	2797	98	4.48 (1.98-10.15)
4 to <8	2594	92	1.50 (0.72-3.13)
8 to <12	4846	177	1.31 (0.73-2.36)
≥12	15272	609	1.04 (0.79-1.38)



# Exercise and cardiovascular disease :

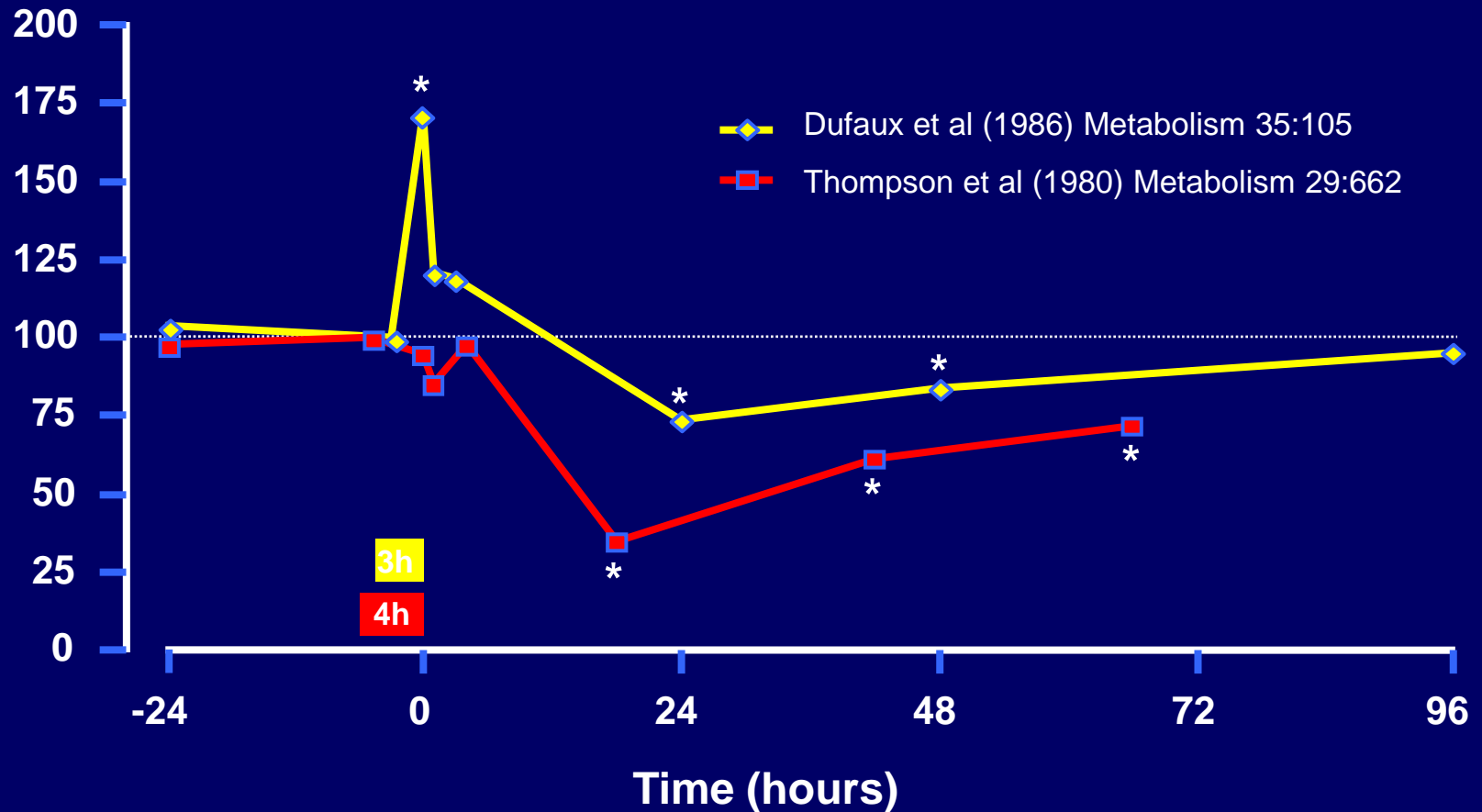
## What do we know

# Exercise and cardiovascular disease



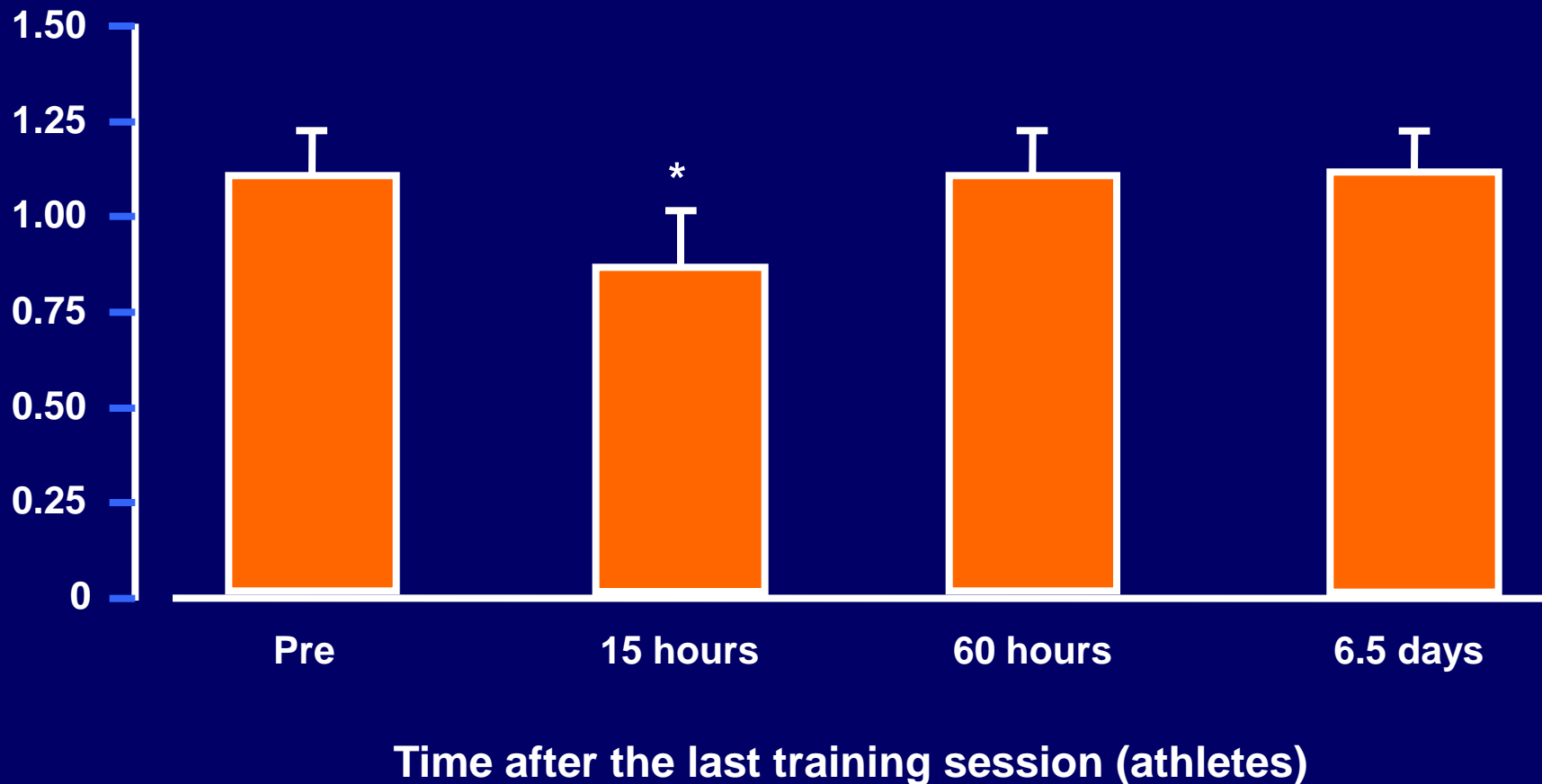
# Hypotriglyceridemia is acute and short-lived

Plasma triglyceride (% of pre-exercise baseline)



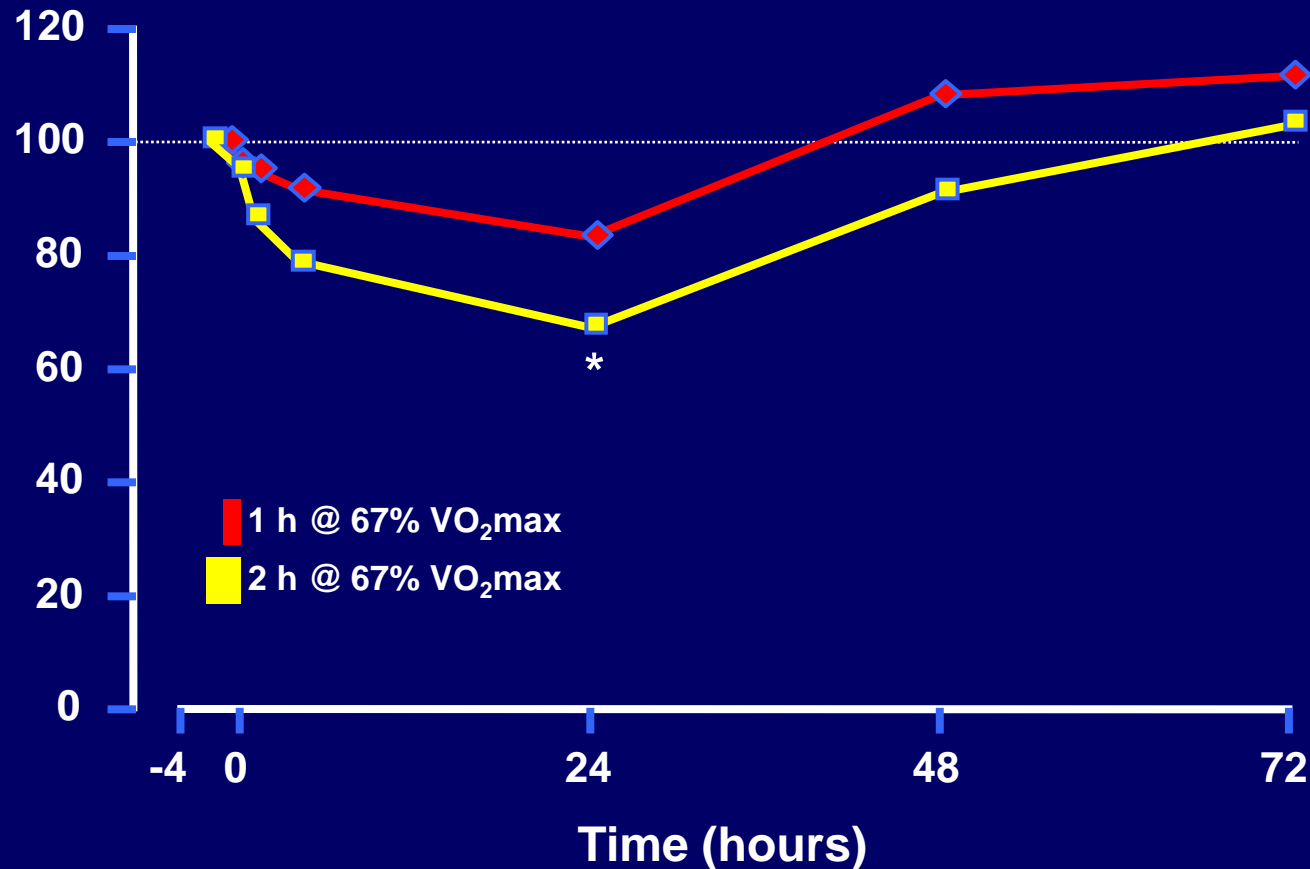
# Hypotriglyceridemia does not result from training

Plasma triglyceride (mM)

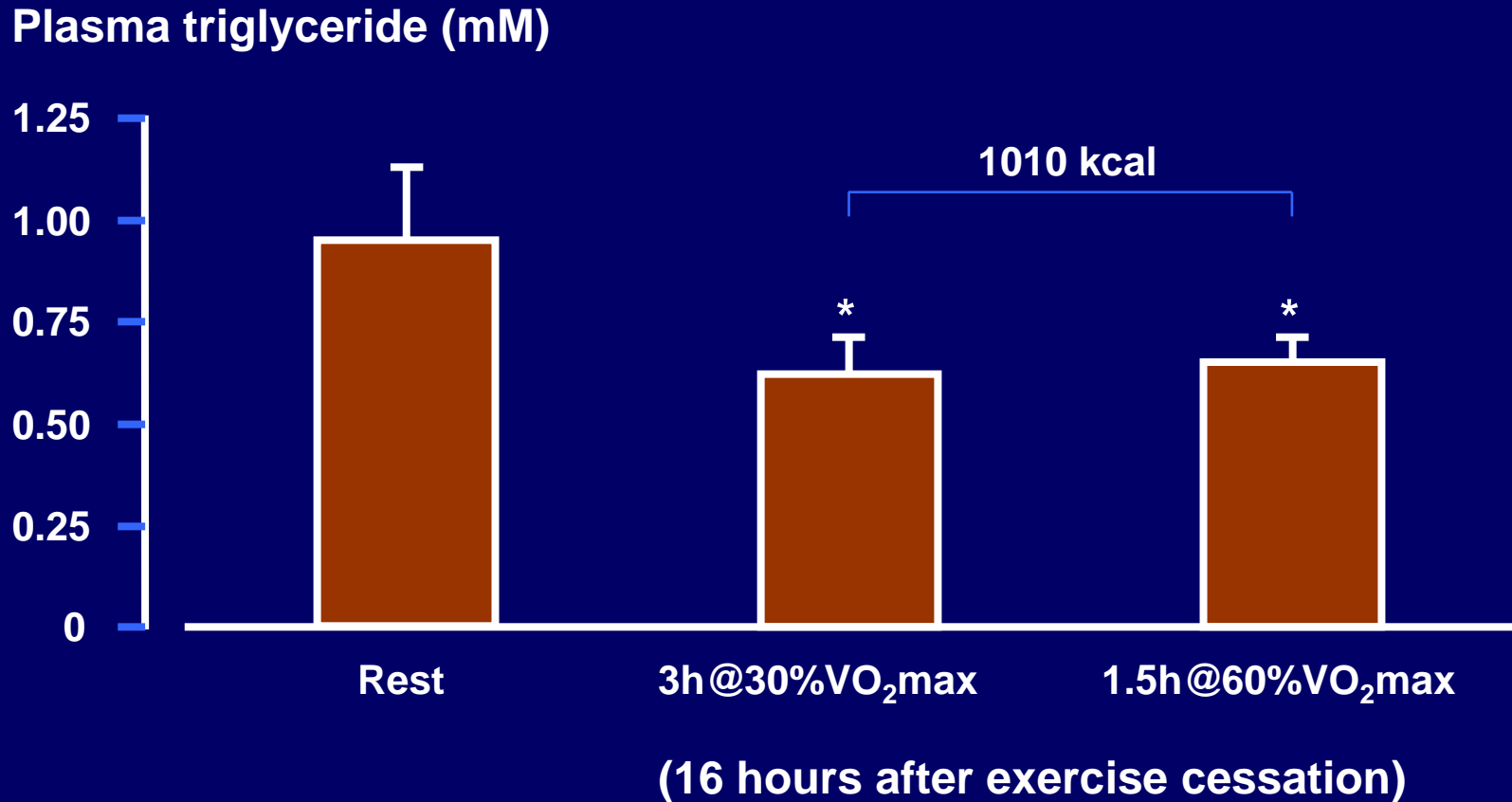


# Hypotriglyceridemia is dose-dependent

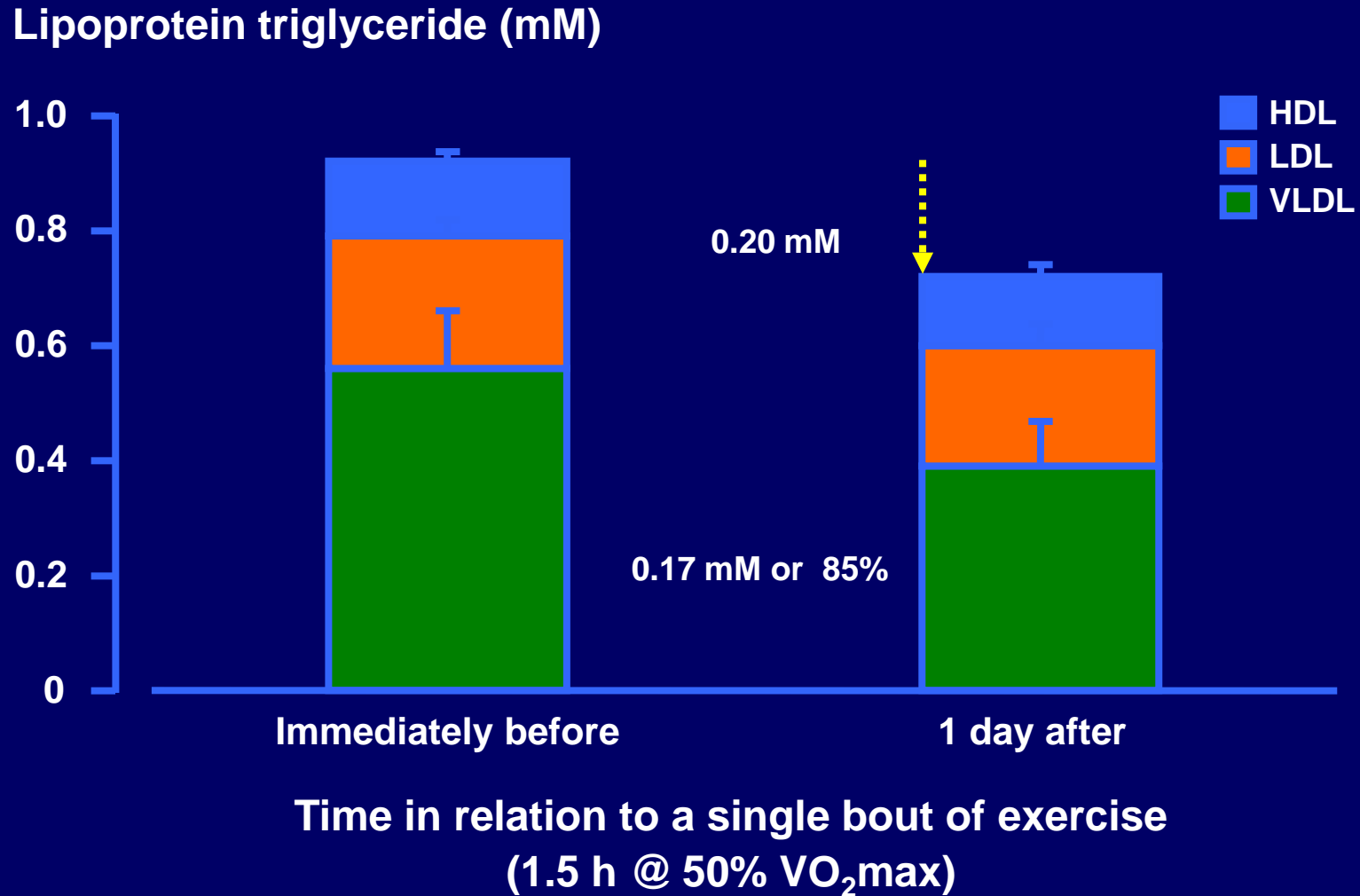
Plasma triglyceride (% of pre-exercise baseline)



# Hypotriglyceridemia depends on energy expenditure



# Hypotriglyceridemia results from hypo-VLDL-triglyceridemia



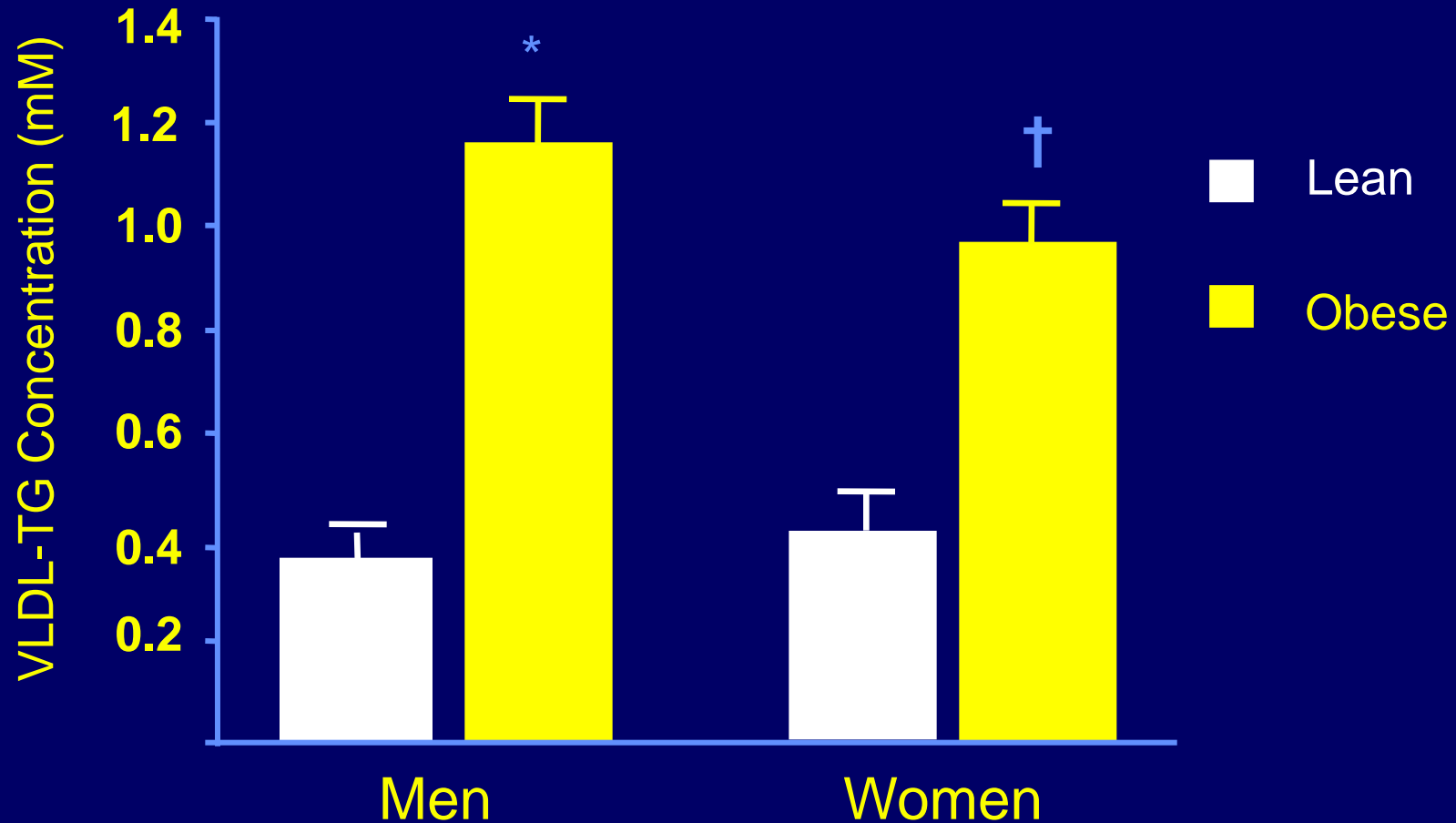
Mechanisms of acute exercise –  
induced decrease in fasting and  
post-prandial lipemia

Why do we need to look at mechanisms?

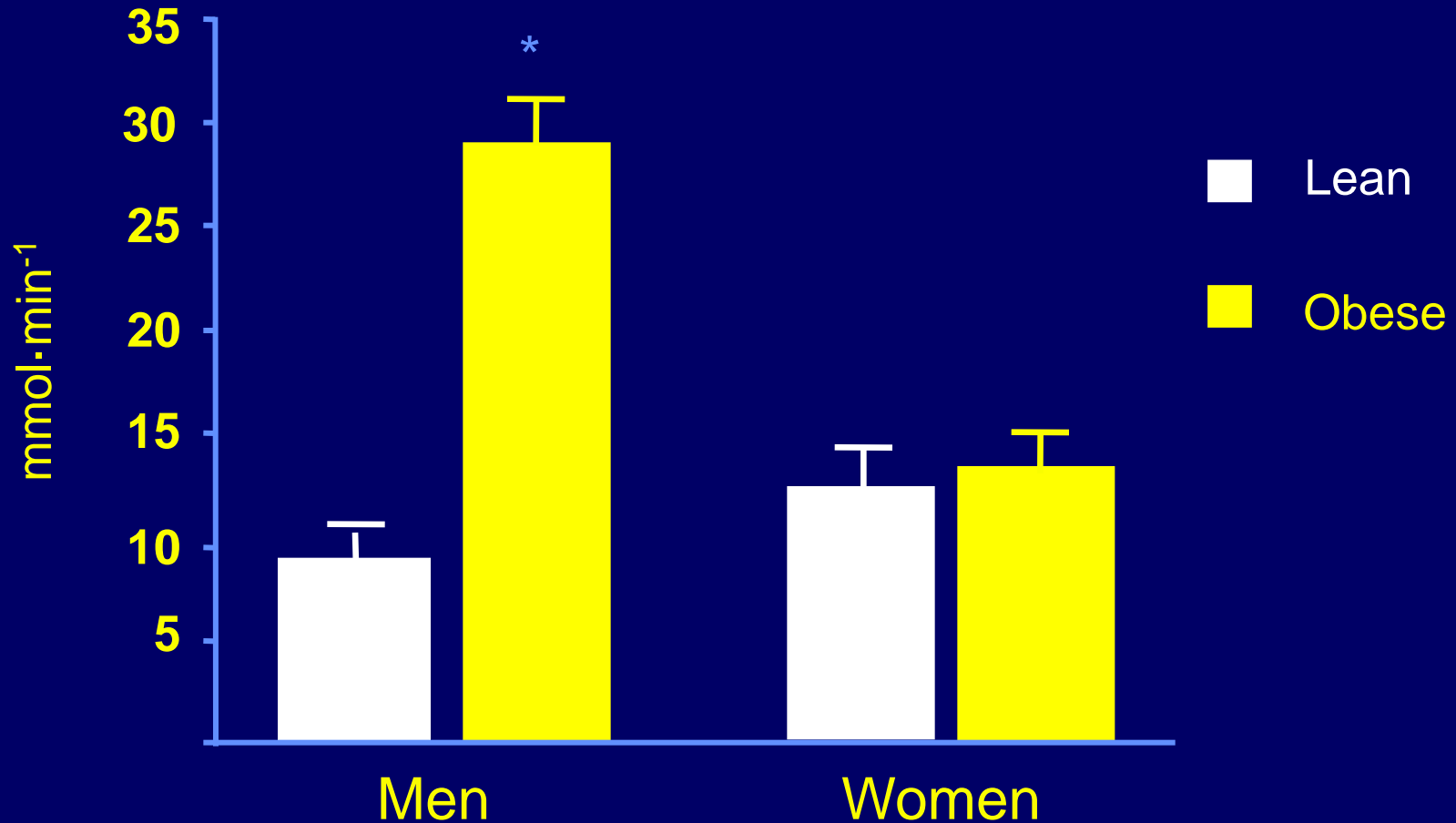
# Regulation of plasma VLDL-TG concentration



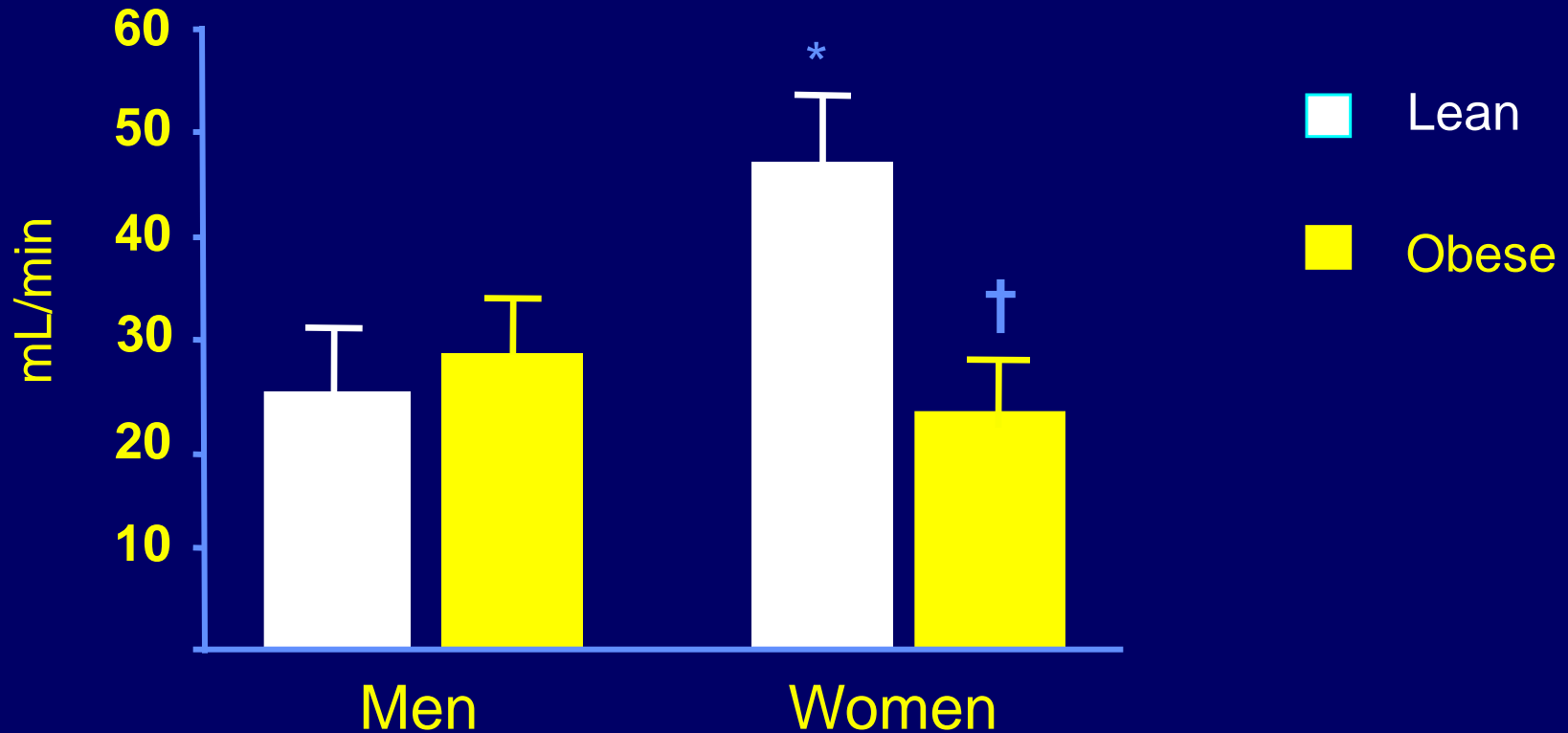
# VLDL-triglyceride concentration: effects of sex and obesity



# Effect of Obesity and Gender On VLDL-TG Production Rate



# Effect of Obesity and Gender On VLDL-TG Clearance



Mechanisms explaining the observed  
hypotriglyceridemic effect of exercise:  
Aerobic exercise



## A single bout of brisk walking decreases basal VLDL-TG concentration

Exercise: 90 min  
at 60%  $\text{VO}_2$  max;  
800 Kcal

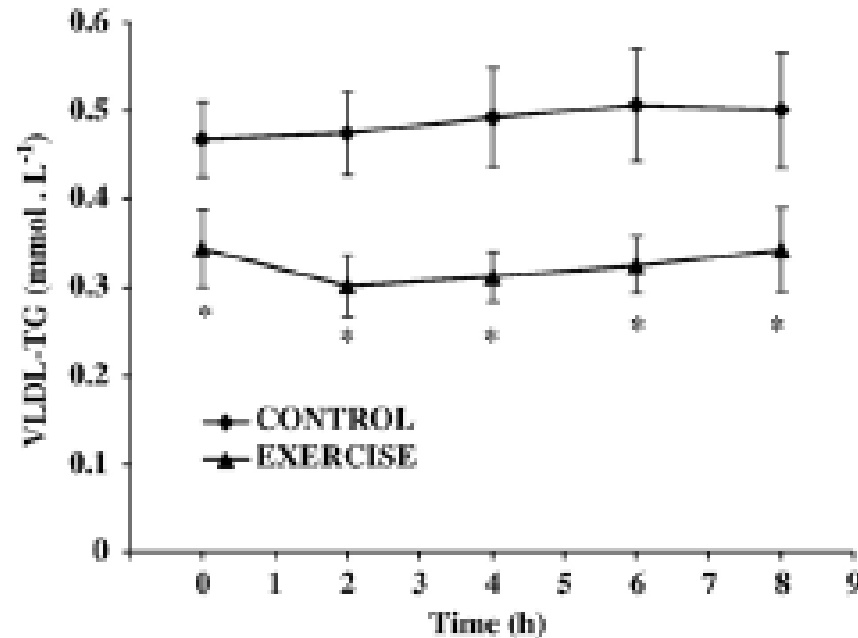
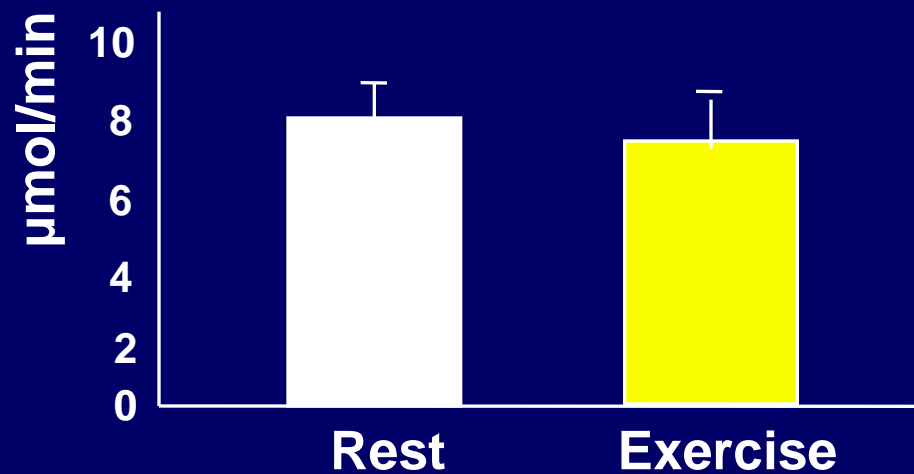


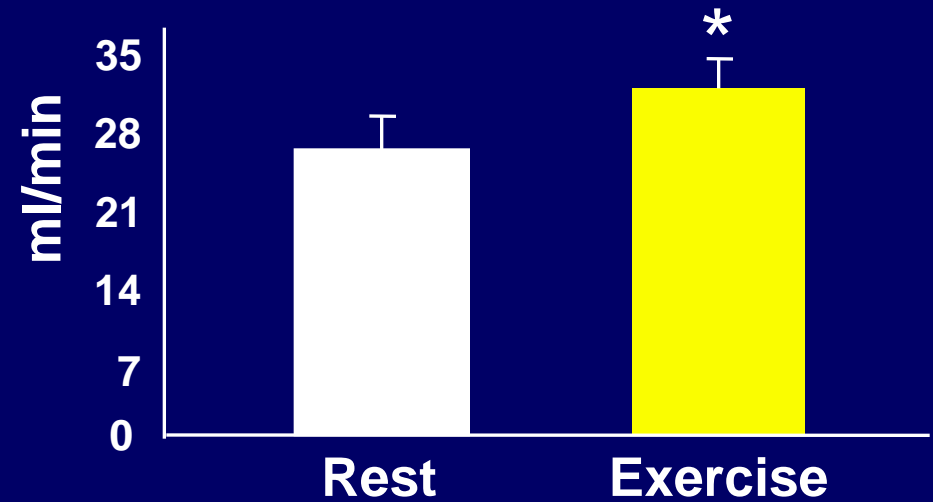
Fig. 3. VLDL-TG concentration throughout the 8-hour study period for the CON and EXE trials, respectively. \* $P < .05$ , significantly different from control.

A single bout of brisk walking (90 min at 60%  $\text{VO}_2$  peak; 800 Kcal) increases basal VLDL-TG clearance in men

(a) VLDL-TAG secretion rate



(b) VLDL-TAG plasma clearance rate

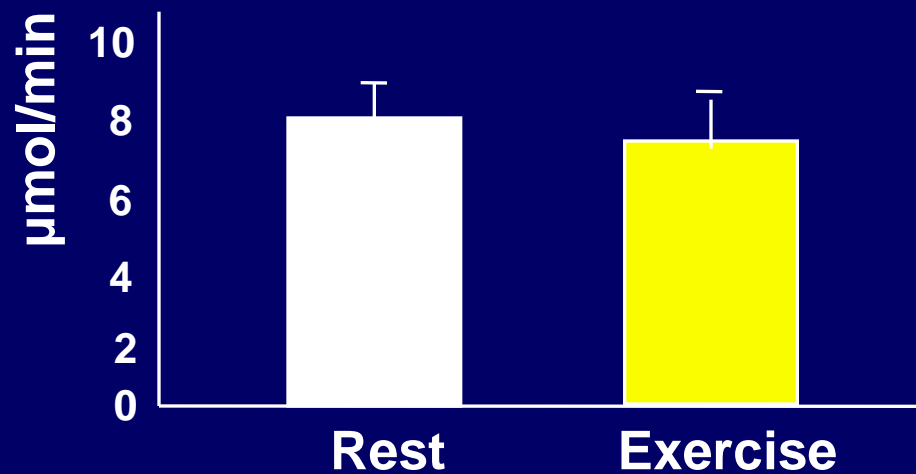


Mechanisms explaining the observed  
hypotriglyceridemic effect of exercise:  
Resistance exercise

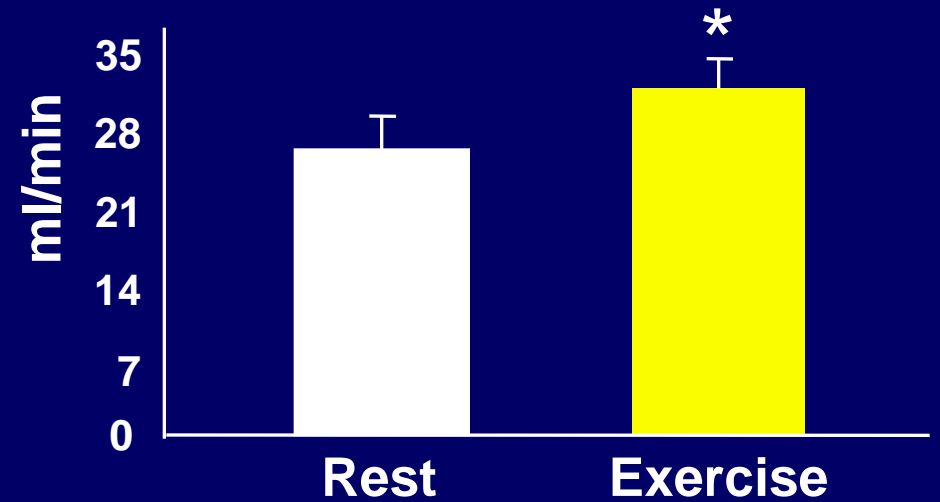


A single bout of resistance exercise  
(90 min resistance exercise; 400 Kcal)  
increases basal VLDL-TG clearance in men

(a) VLDL-TAG secretion rate



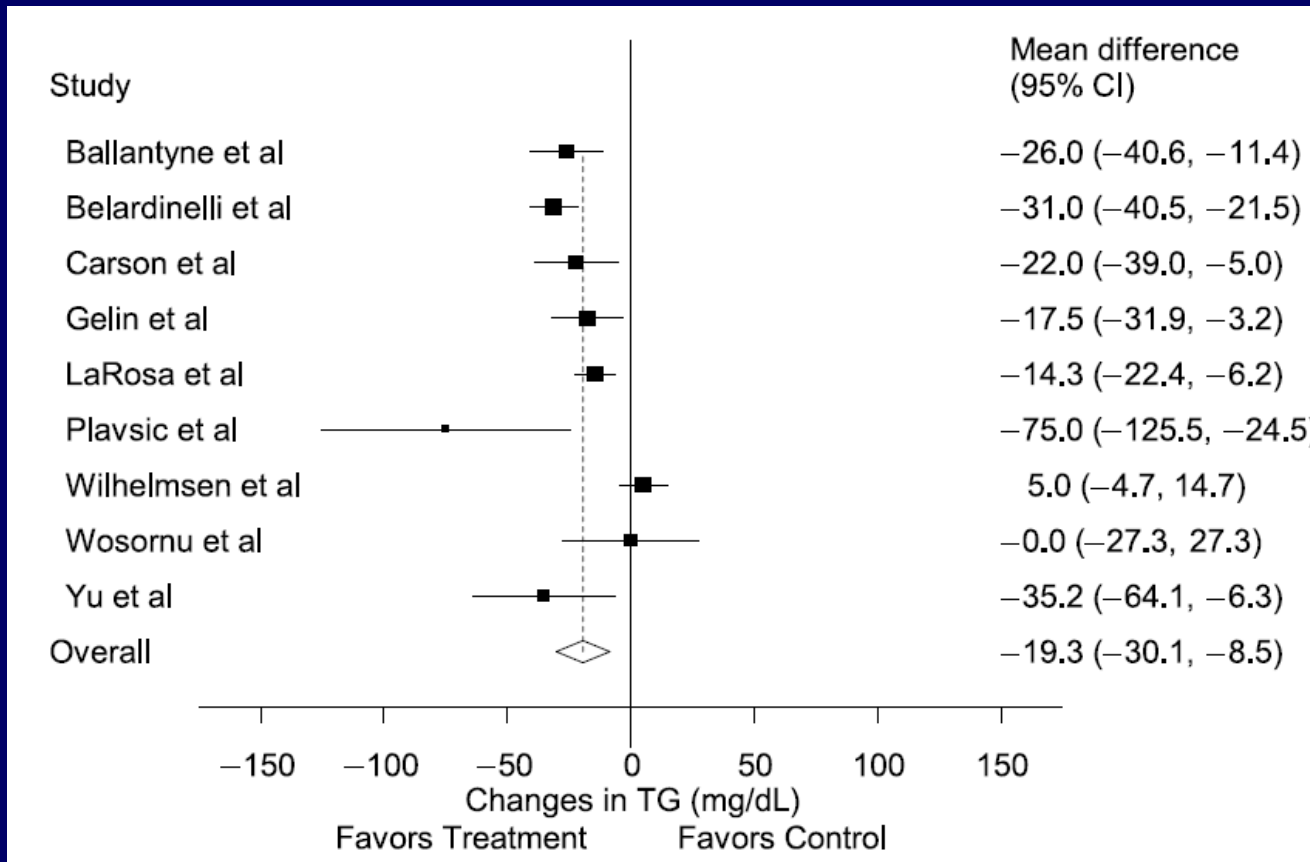
(b) VLDL-TAG plasma clearance rate



# Mechanisms explaining the observed hypotriglyceridemic effect of exercise: Aerobic training

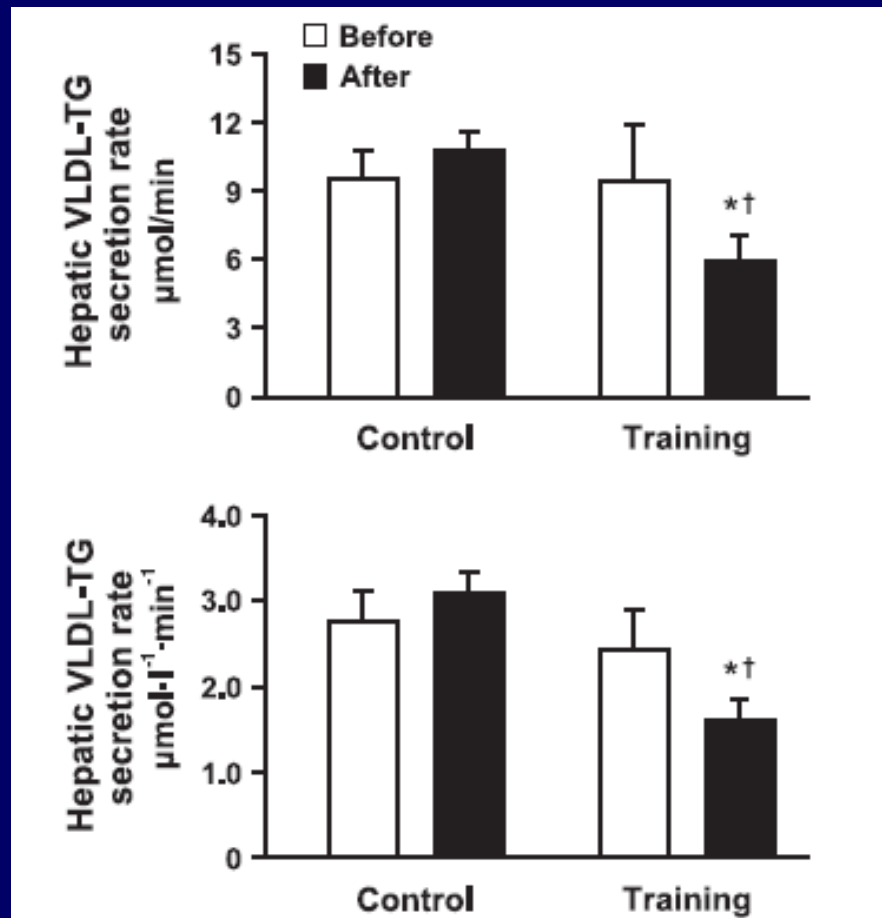


# Effect of exercise training on fasting plasma TG in patients with CVD



**~11% reduction ( $P < 0.05$ )**

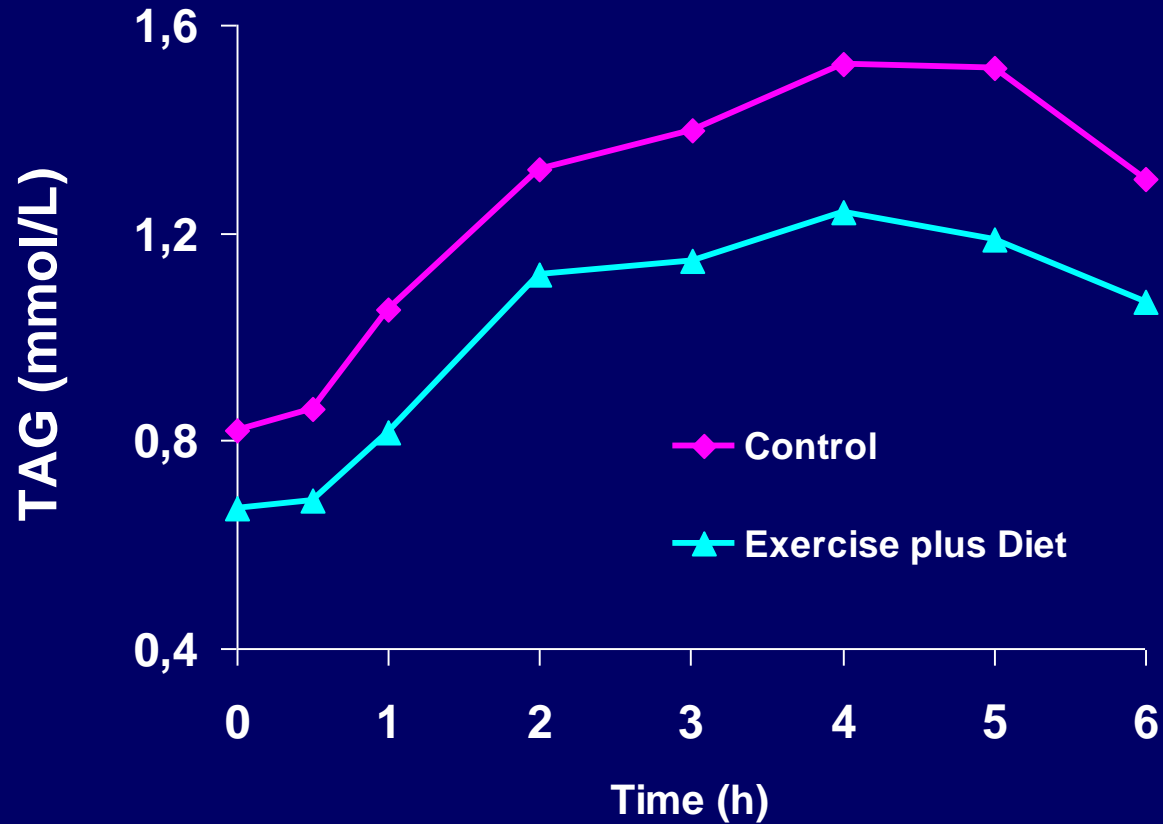
# Aerobic training reduces basal hepatic VLDL-triglyceride secretion rate in healthy men



# Effect of diet and exercise on postprandial triglyceride levels

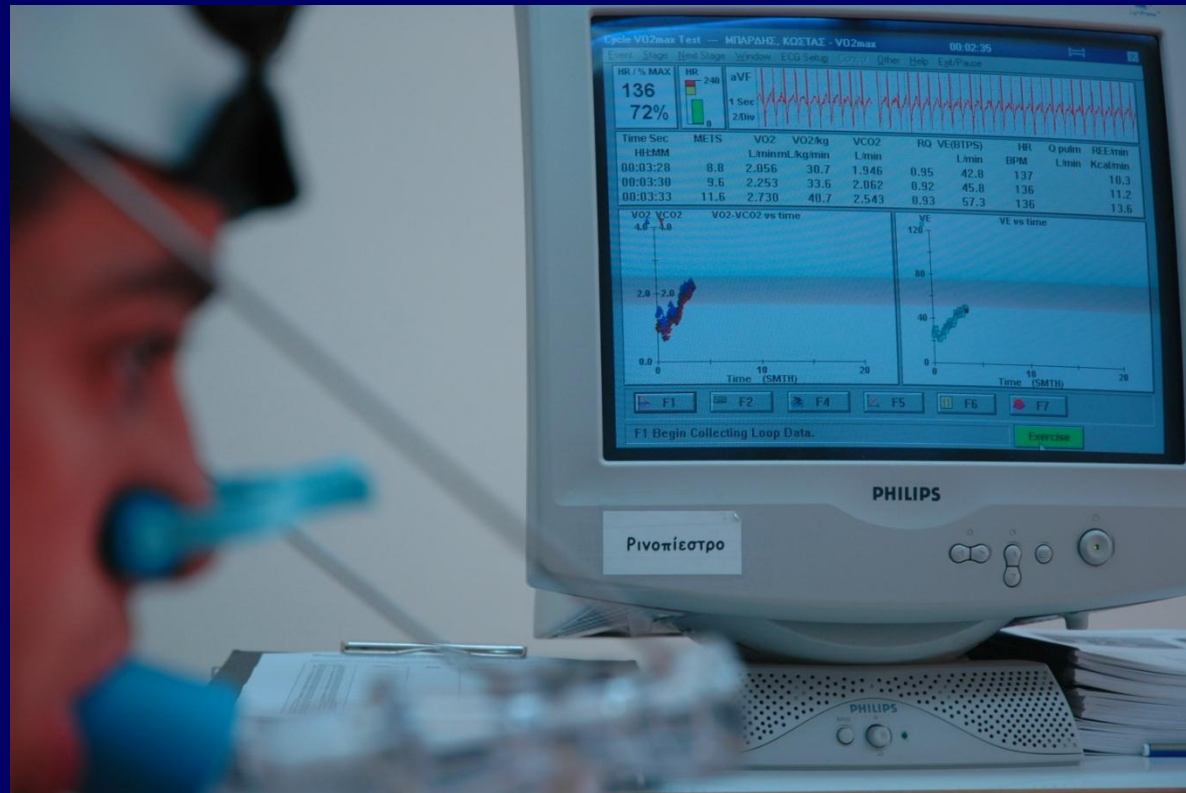


# Exercise (250 Kcal) + Diet (250 Kcal) acutely reduces fasting and postprandial triacylglycerolaemia in young lean women



(Maraki et al, Br J Nutr. 2008)

Is the effect on triglycerides due to exercise or diet *per se* or is it due to the negative energy balance

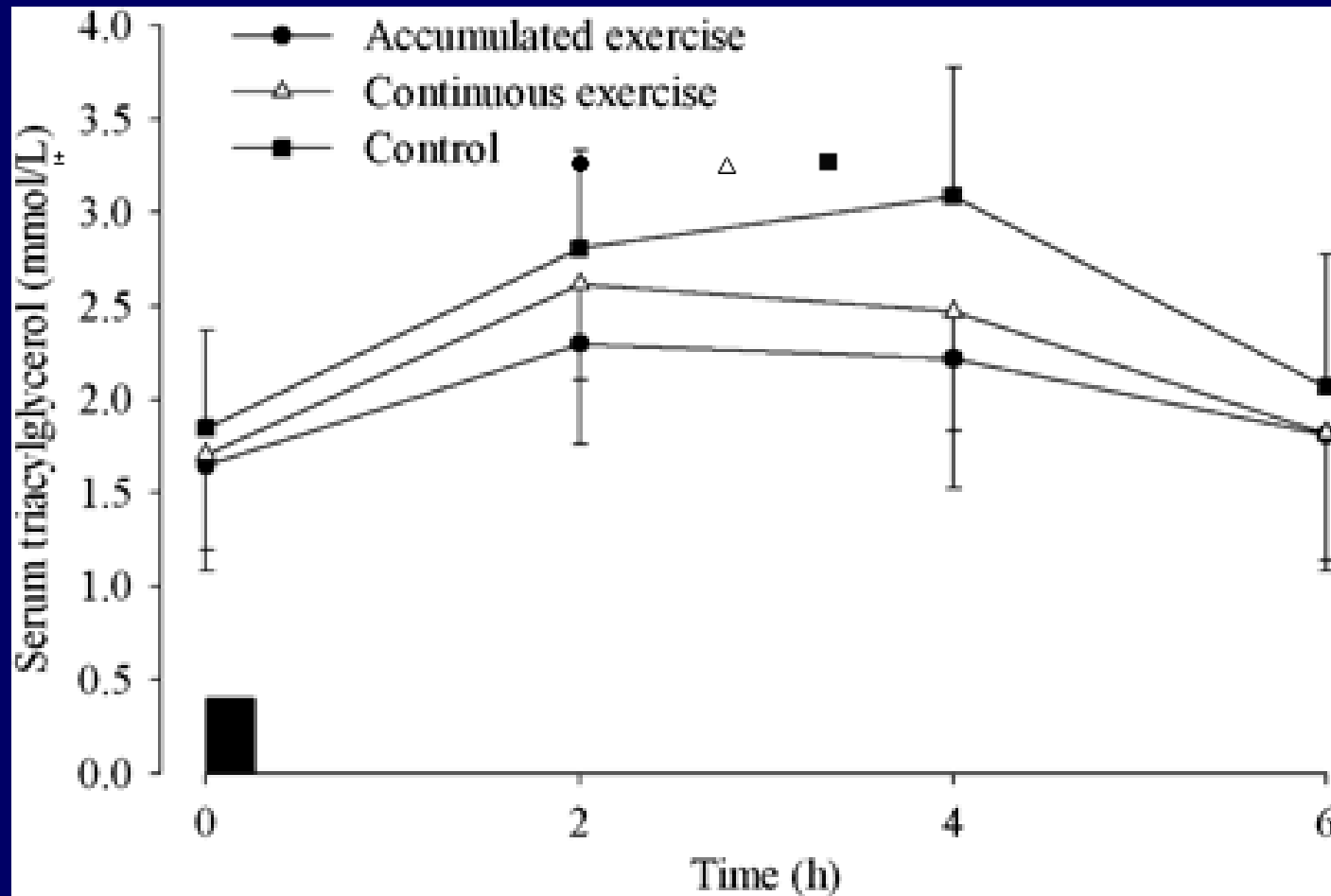


# Negative energy balance from exercise, diet or a combination of both: effect on fasting and postprandial TG



\* p < 0.05 vs Control

# Effects of continuous vs accumulated activity on postprandial TG concentrations in obese men



# Conclusions

- Aerobic and resistance exercise significantly reduce plasma triglycerides
- The effect of exercise
  - is acute (manifests after a single bout of exercise)
  - is short-lived (lasts for 1-2 days)
  - It depends on energy expenditure
- The mechanisms responsible for the diet and exercise-induced effects on TG metabolism need to be further elucidated

# Exercise and VLDL metabolism: future directions

1. What is the optimum / minimum exercise (type – intensity – duration combination) and the proper diet – exercise combination for correcting hyper-TAG in various diseases
2. What are the mechanisms responsible for the observed improvements on lipid / lipoprotein profile brought about by exercise and diet manipulation



Harokopio University, Athens, Greece

S. Kavouras, G. Nassis, F. Magkos, Y.  
Tsekouras, M. Maraki, K. Anastasiou,

K. Skenderi, A. Yianni, D.  
Panagiotakos, E. Bathrellou

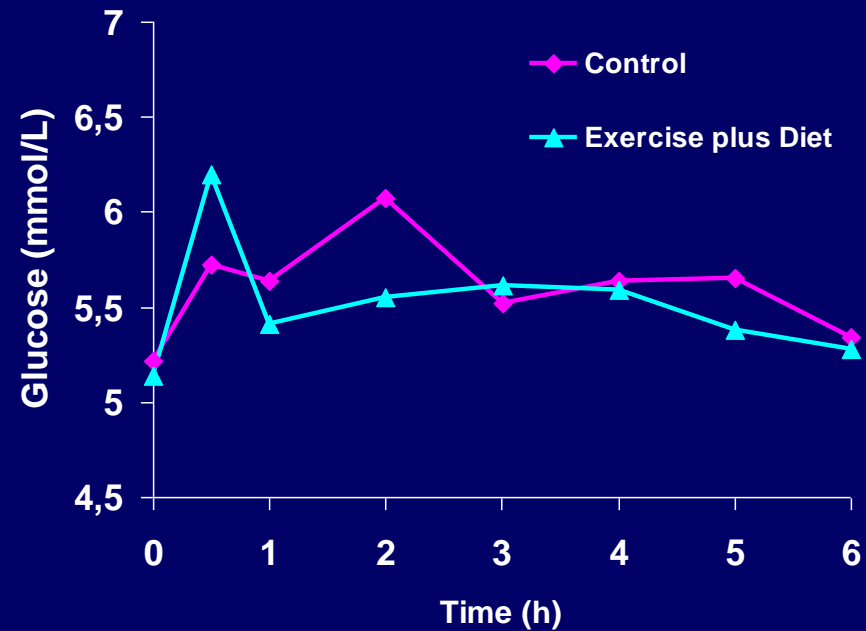
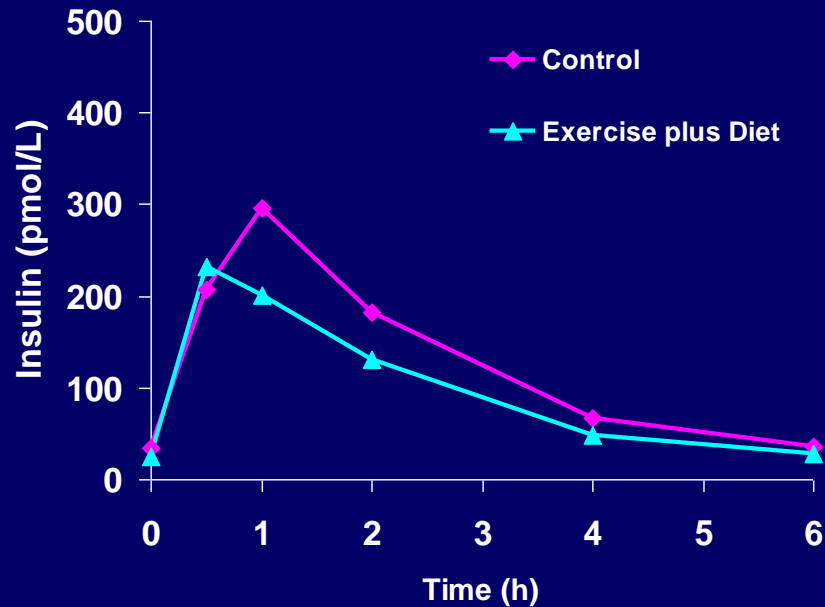
Washington University School of  
Medicine, St. Louis, MO, USA

S. Klein, B. Mittendorfer, B. Patterson  
University of Texas Medical Branch at  
Galveston, TX, USA

R. Wolfe, J. Romijn, A. Gastaldelli, D.  
Chinkes

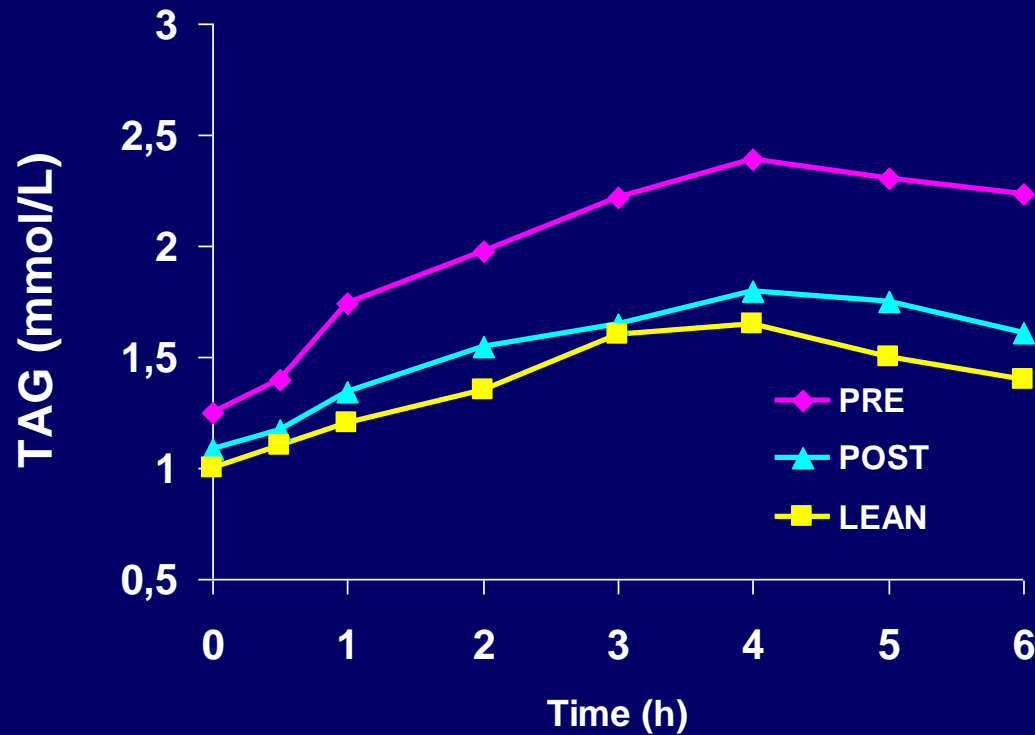
**Thank you for your attention**

# Effect of exercise (250 Kcal) + Diet (250 Kcal) on OFFT insulin and glucose response

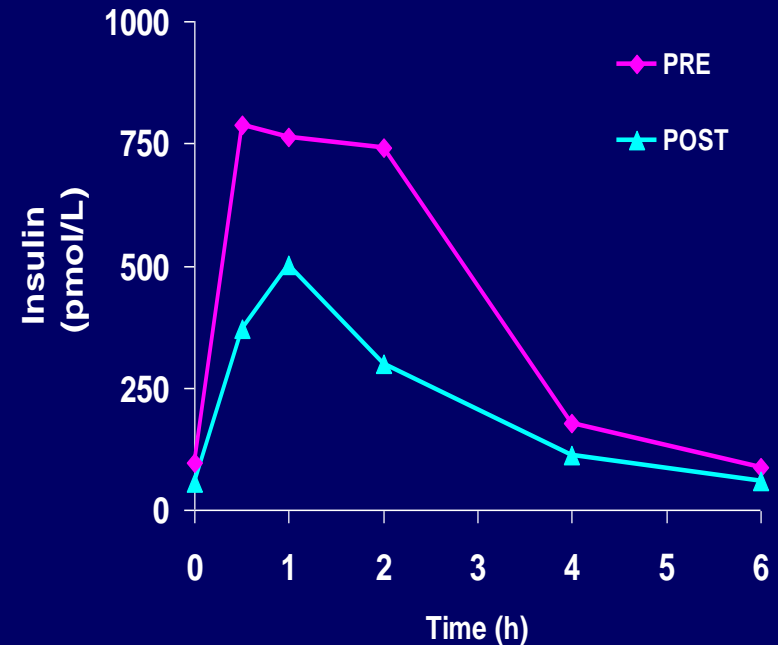
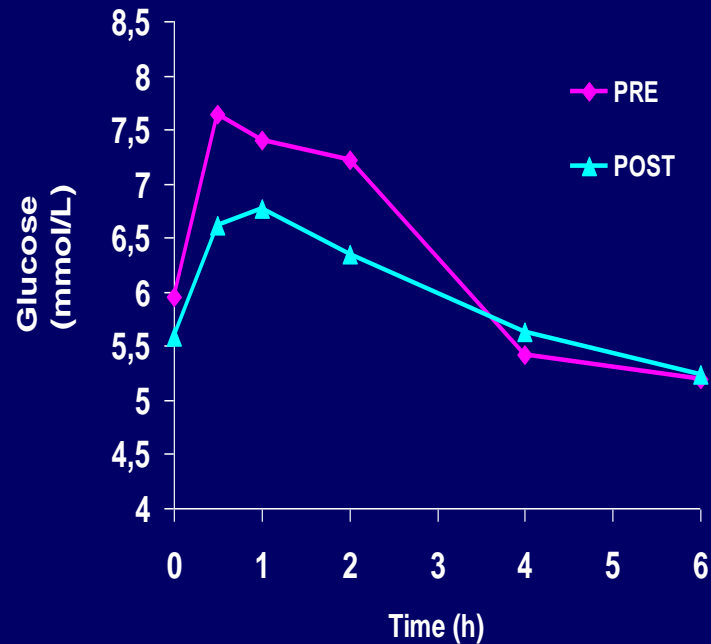


(Maraki et al, *Br J Nutr.* 2008)

## A 10% weight loss reduces postprandial triacylglycerolemia in obese subjects



## Effect of 10% weight loss on OFTT insulin and glucose response



Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls (Nassis et al, Metabolism 2005)

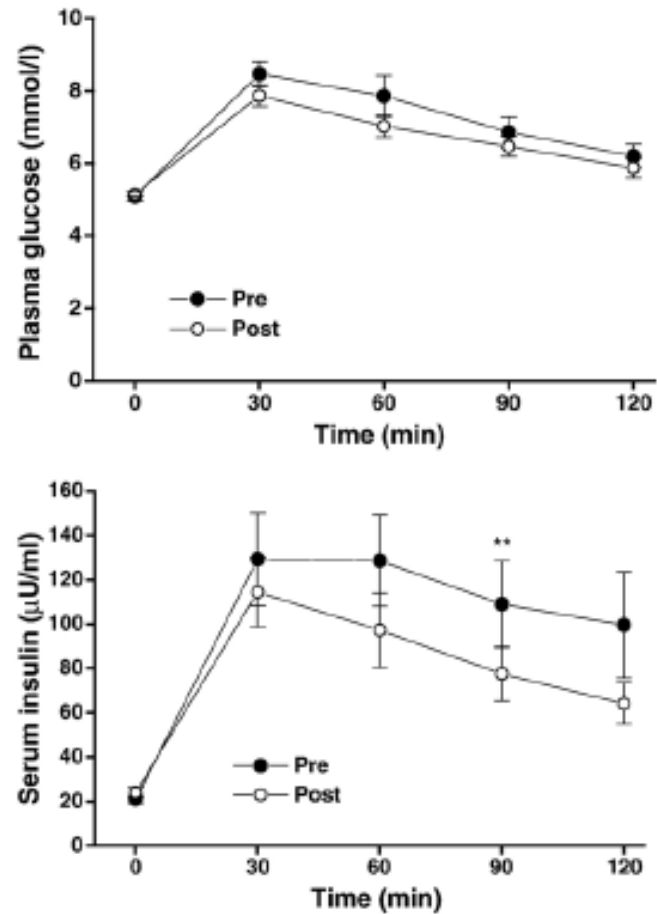
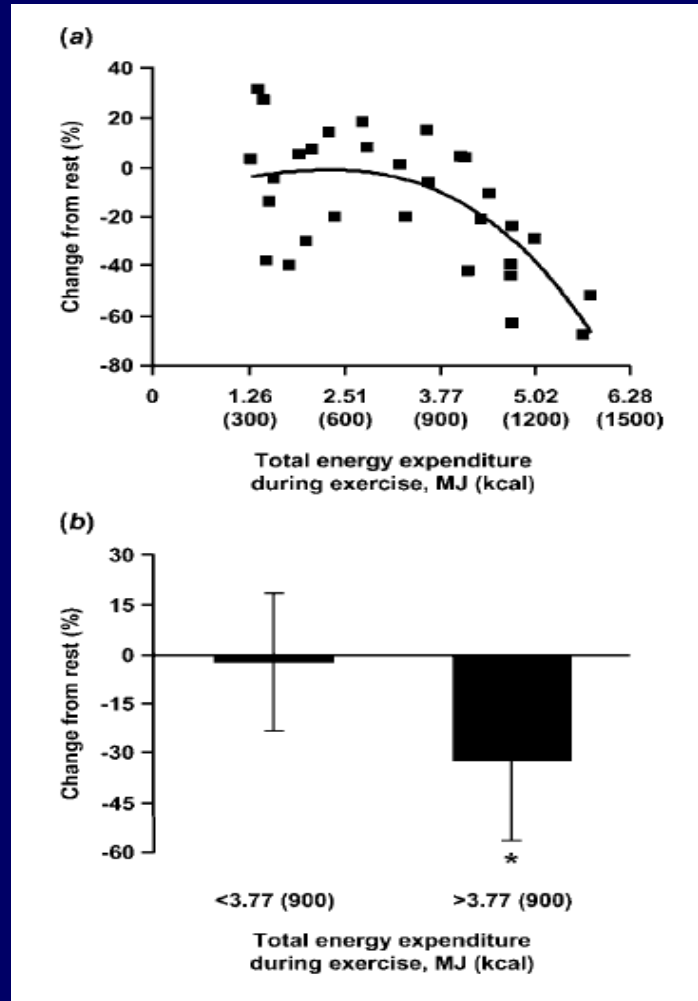


Fig. 1. Plasma glucose (top panel) and serum insulin (bottom panel) responses during the 2-hour OGTT before and after 12 weeks of aerobic training in overweight and obese girls (n = 15). Values are means  $\pm$  SE; double asterisk in the bottom panel indicates  $P < .01$  vs postintervention.

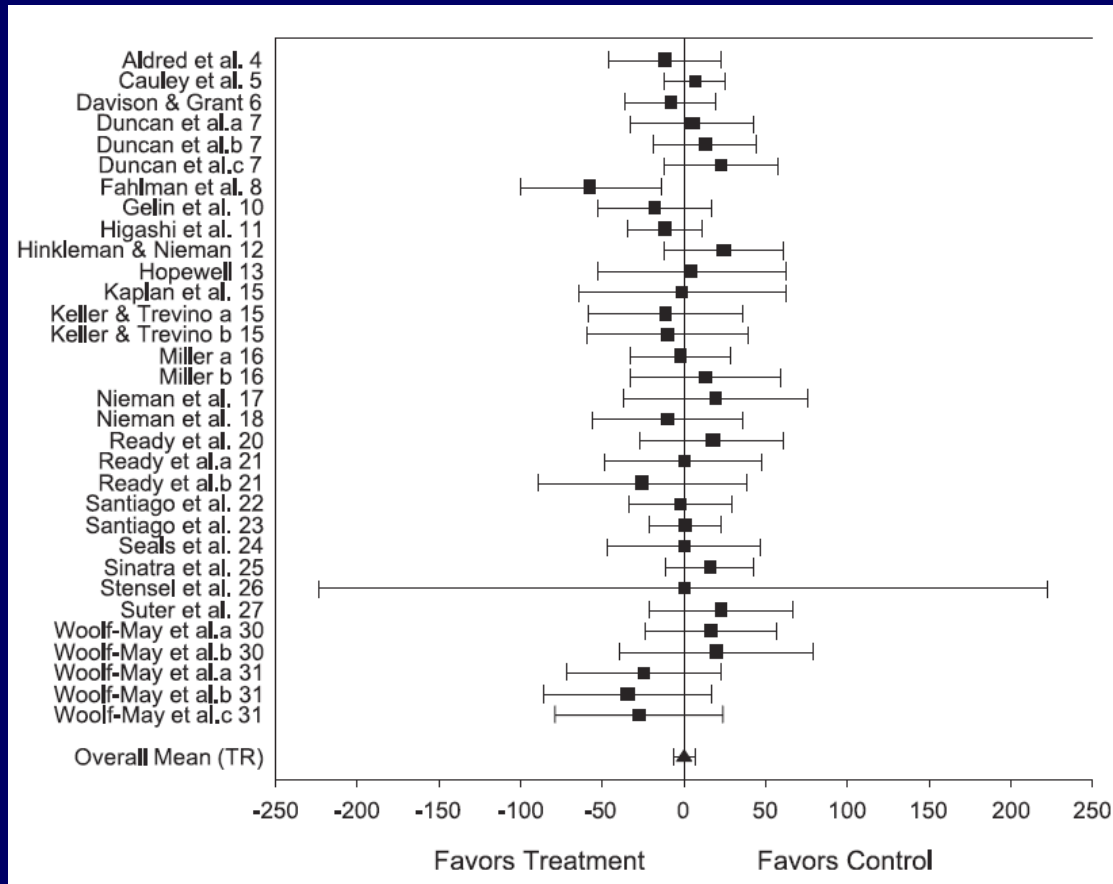
# Improved insulin sensitivity after a single bout of exercise is curvilinearly related to exercise intensity



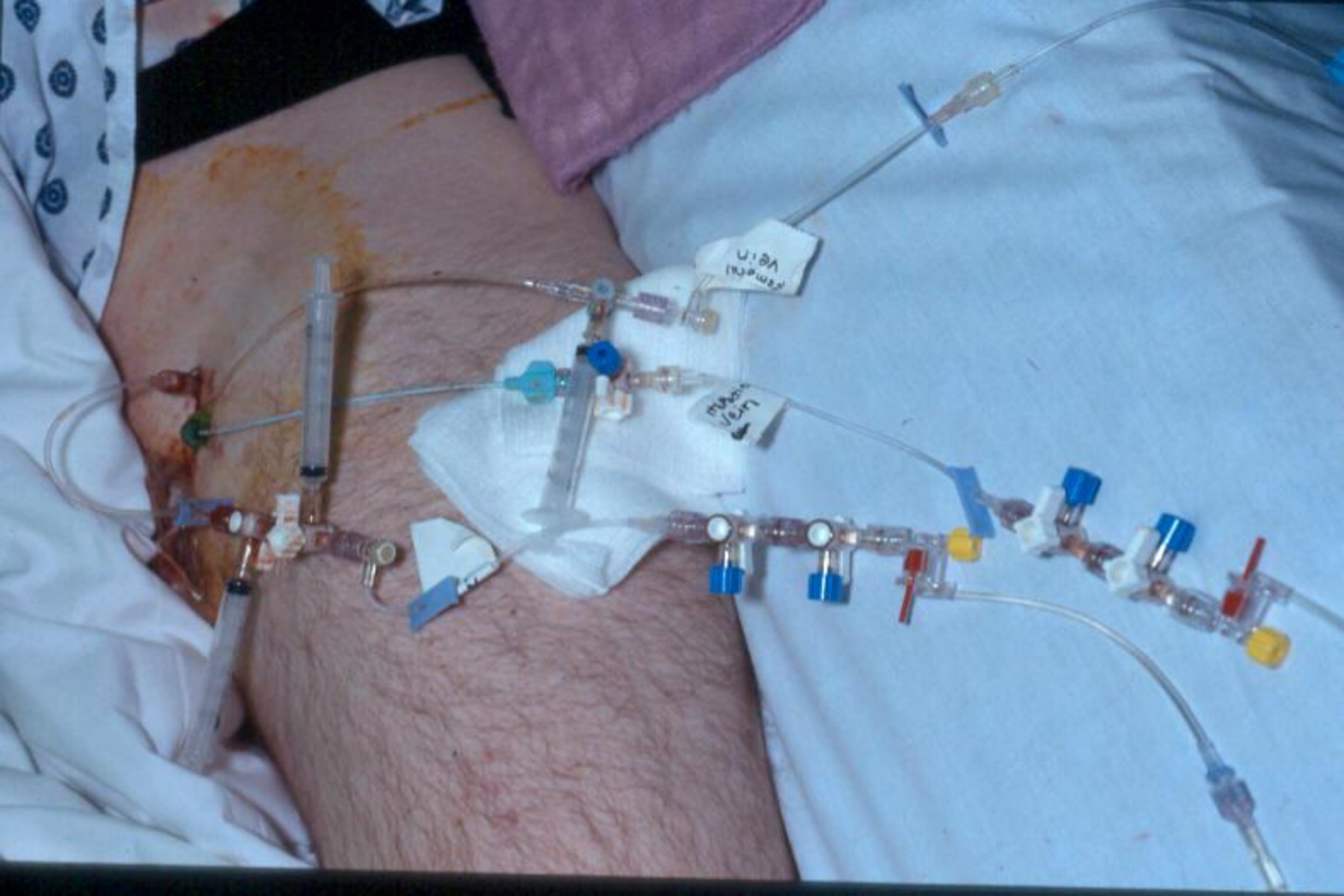
**Figure 1** Exercise-induced changes in  $HOMA_{1R}$  as a function of total energy expenditure during exercise (a), and for subjects who expended less than or more than 3.77 MJ (900 kcal) during the exercise bout (b)

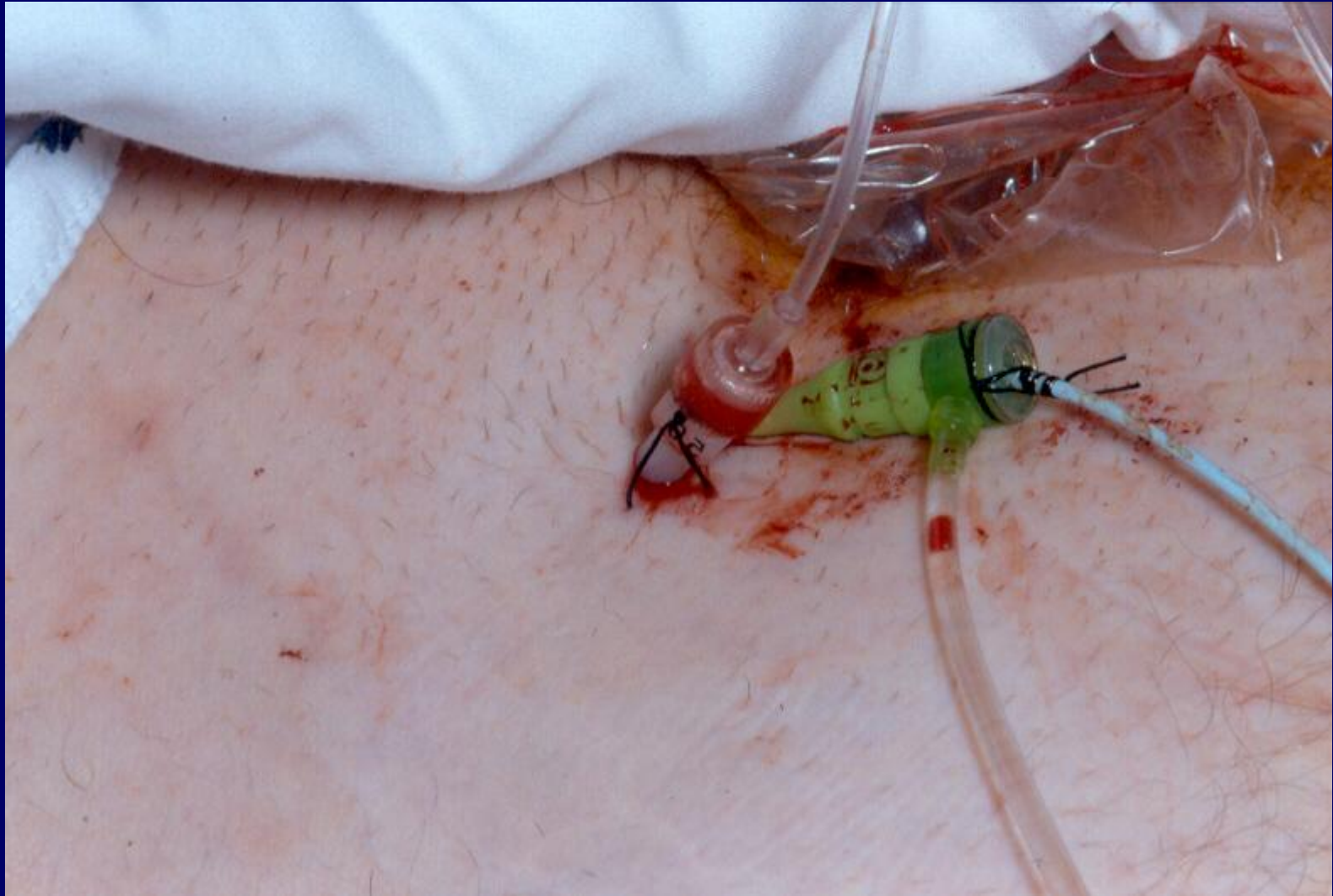
In (b), values are means  $\pm$  S.D. \* $P < 0.05$  compared with rest.

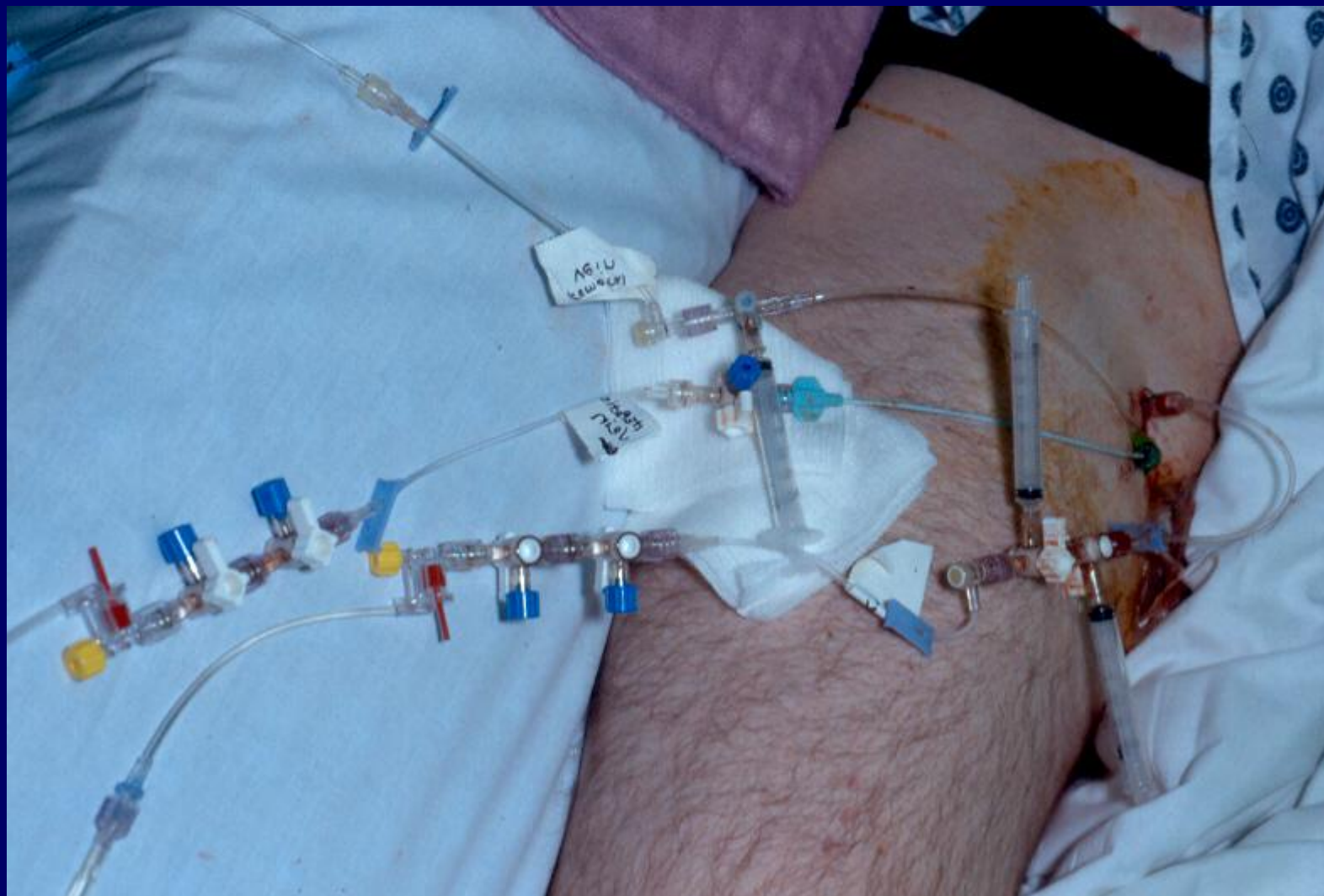
# Effect of walking on fasting plasma TG



( $P=NS$ )







# Exercise-induced hypotriglyceridemia

The hypotriglyceridemic effect of exercise is **acute** (manifests after a single bout of exercise and is not the result of repeated exercise sessions, i.e. training) and **short-lived** (lasts for 1-2 days). Holloszy et al (1964) Am J Cardiol 14:753

	Effect of training	Effect of a single bout
Plasma [TG]	↓ 24% (4 – 37%)	↓ 20% (14 – 50%)

# Possible mechanisms

## Indirect evidence:

### Increased VLDL-TG removal from plasma

- Skeletal muscle LPL mass/activity

Seip & Semenkovich (1998) Exerc Sport Sci Rev 26:191

Kiens & Richter (1998) Am J Physiol 275:E332

- Postheparin plasma LPL activity

- Clearance of exogenous TG (IVFTT)

Sady et al (1986) J Am Med Assoc 256:2552

Annuzzi et al (1987) Metabolism 36:438

### Reduced VLDL-TG secretion from the liver

- Animal studies; isolated hepatic tissue

Simonelli & Eaton (1978) Am J Physiol 234:E221

Mondon et al (1984) J Appl Physiol 57:1466

- Increased ketone body concentrations

Herd et al (2001) Metabolism 50:756

- Reduced [TG] without changes in LPL or IVFTT clearance

Gill et al (2001) Eur J Clin Invest 31:201

# Exercise and VLDL-TG metabolism in men

Exercise	Exercise-induced change (% from resting values)				
	Gross EE	Concentration	Secretion	Clearance	MRT
2 h cycling at 60% VO <sub>2</sub> peak <sup>1</sup>	~1200 kcal	-29.5*	-8.8	29.3*	-21.2*
1.5 h running at 60% VO <sub>2</sub> peak <sup>2</sup>	~925 kcal	-33.5*	-8.0	37.9*	-21.6*
1 h cycling at 60% VO <sub>2</sub> peak <sup>3</sup>	~570 kcal	5.3	15.1	9.4	-7.5
1.5 h resistance exercise <sup>4</sup>	~500 kcal	-30.3*	-9.6	20.8*	-17.9*

<sup>1</sup>Magkos et al (2006) Am J Physiol Endocrinol Metab 290:E355

<sup>2</sup>Tsekouras et al (2007) Metabolism 56:1037

<sup>3</sup>Magkos et al (2007) Am J Physiol Endocrinol Metab 292:E1568

<sup>4</sup>Tsekouras et al (2008) Am J Physiol Endocrinol Metab