



*Alternatives to trans fat:
Implications of interesterification*

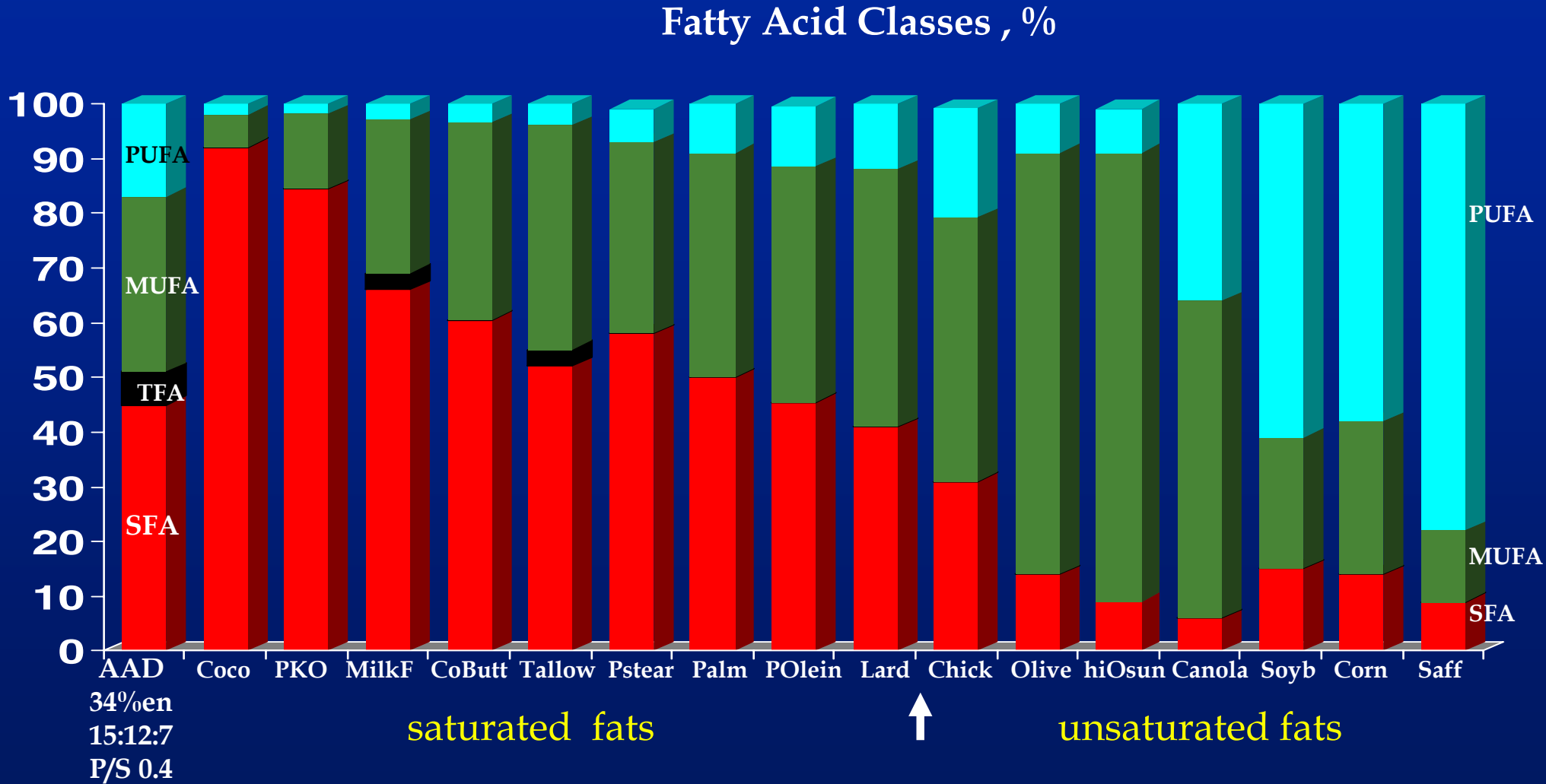
KC Hayes, DVM, PhD

Brandeis University

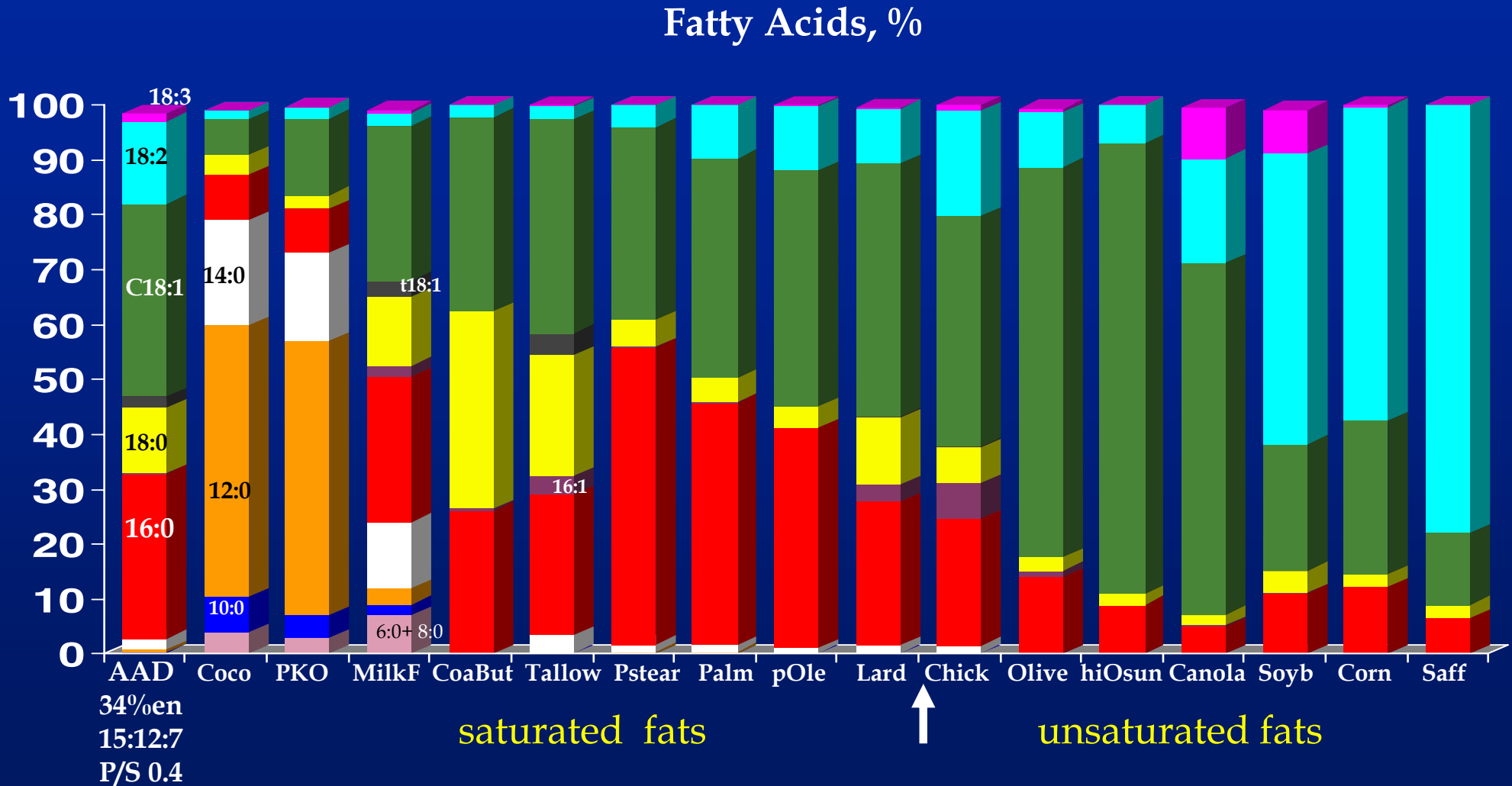
Waltham, MA



Dietary fat composition: by fatty acid classes



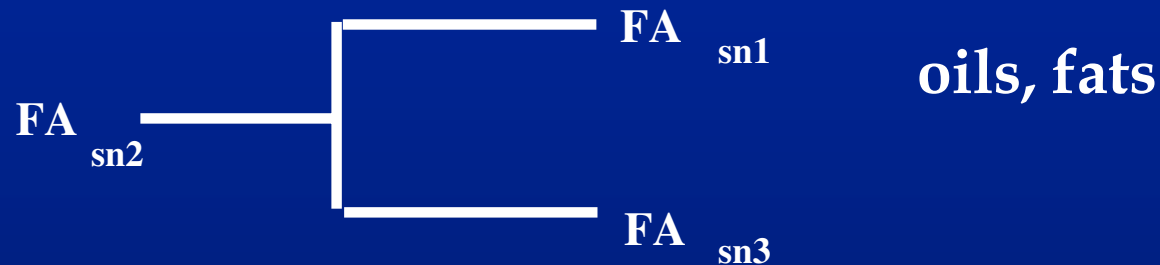
Dietary fat composition: by fatty acids



FAT FORMS we eat are important...

- FATTY ACIDS (16:0, 18:1, 18:2, 18:3...etc.)

and TRIGLYCERIDES (TG-MS):



- PHOSPHOLIPIDS



- sphingolipids, ceramides (sn2-amide-FA)



First, let's consider intake and balance among dietary fatty acids on lipoproteins...





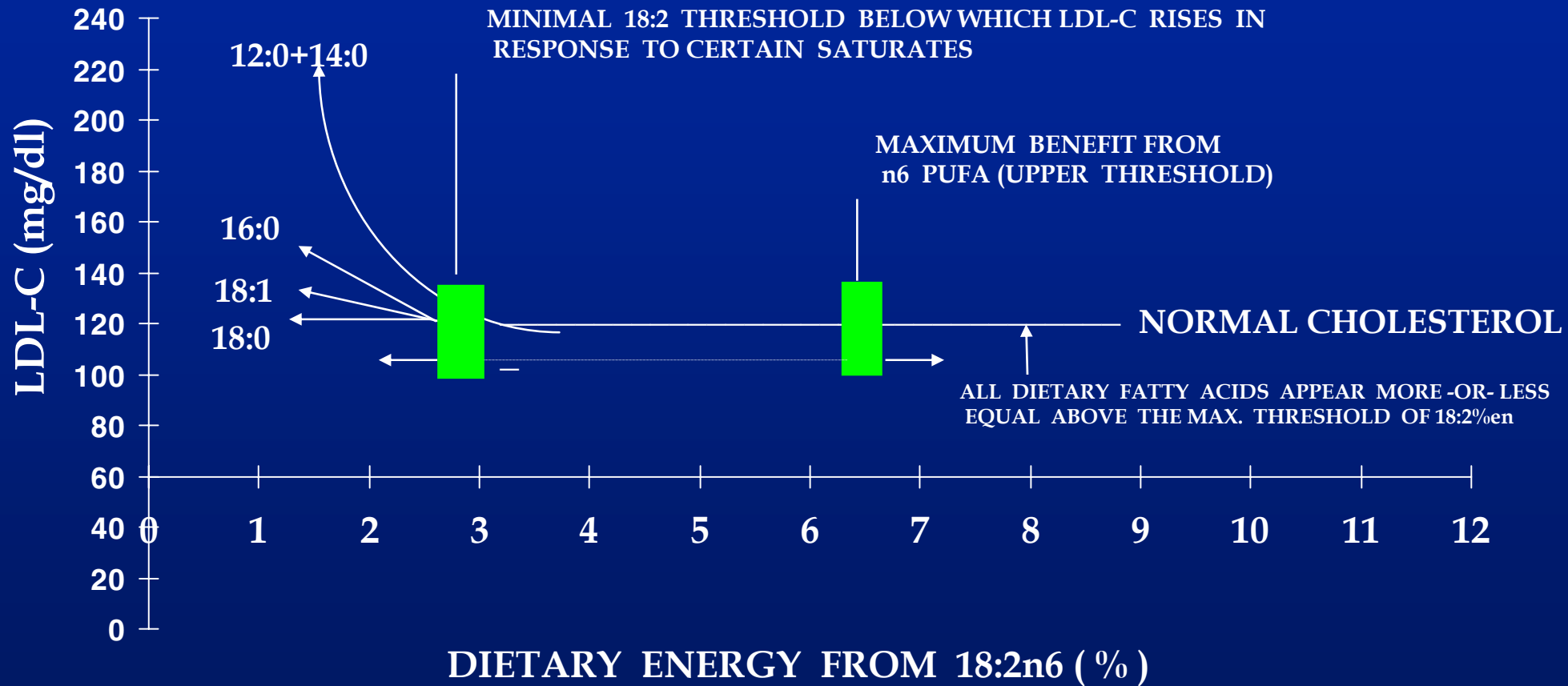
PLASMA CHOLESTEROL RESPONSE (mg/dL) TO DIETARY FATTY ACIDS (PER 1% en

Neutral 0

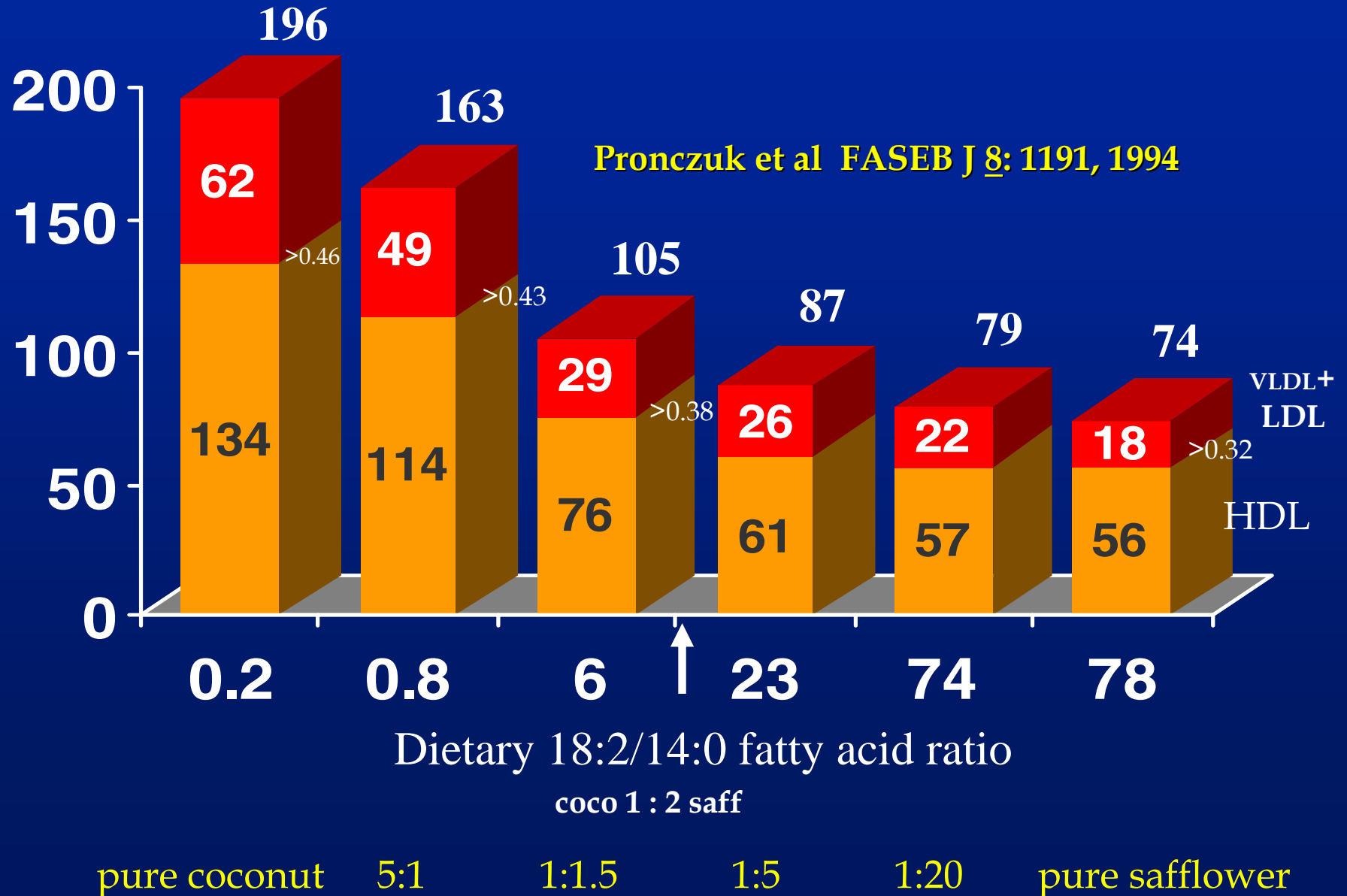
↑
(12:0, 16:0, 18:0, 18:1?)

(varies by Log %en)

PUTATIVE RELATIONSHIP BETWEEN THE DIETARY 18:2 THRESHOLD AND LDL-C IN HUMANS



Natural fat dietary 18:2/14:0 ratio impacts plasma cholesterol in gerbils

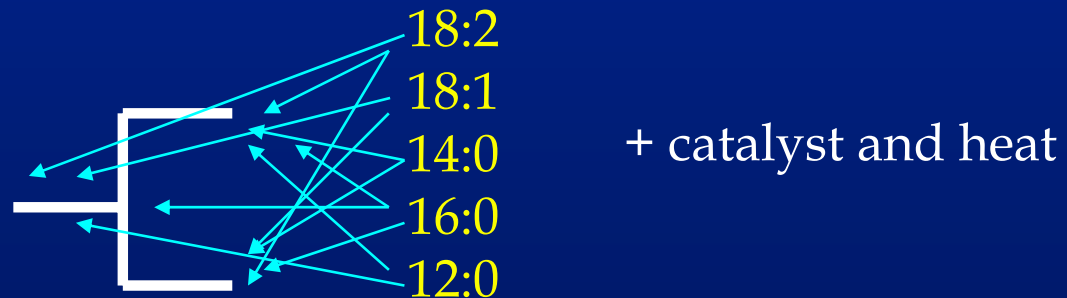




*Second, let's consider TG structure....
..ie. the relative position of fatty acids on
glycerol, especially SFA positions.*



Triglyceride structure itself is important... evidenced by interesterification (IE) process

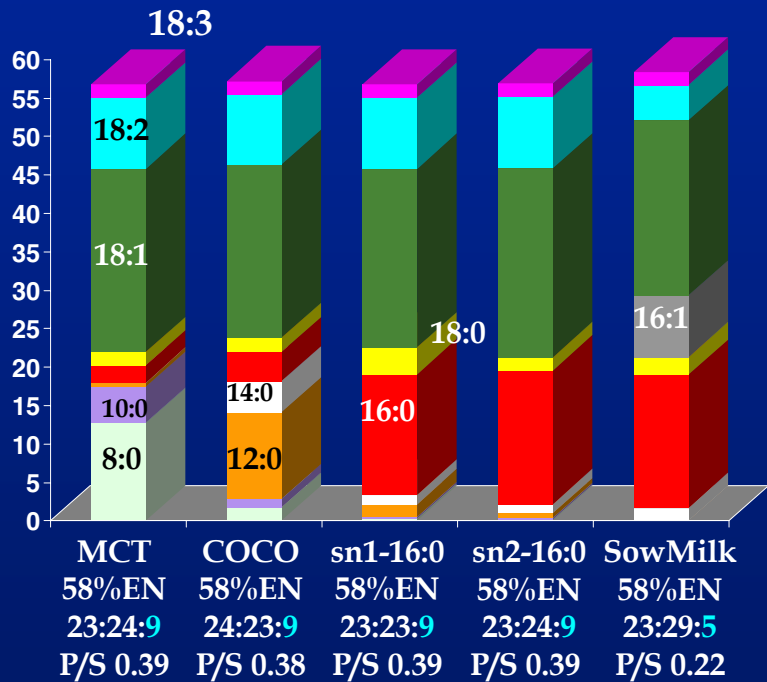


random inter-esterification
"unnatural reshuffle" ...brand new TG molecules

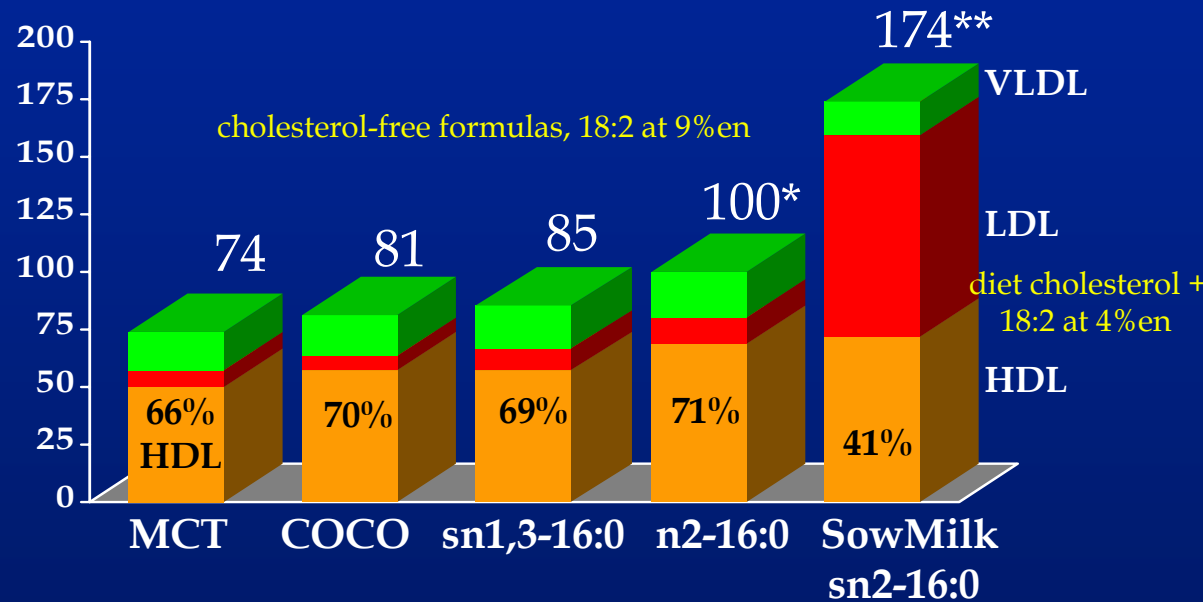
Q: If you interesterify fats, do they behave normally in the body?

Newborn piglets (0-18d) cholesterol response to 8:0 thru 16:0-rich fats after 18d

DIETARY FATTY ACIDS
(percent energy)



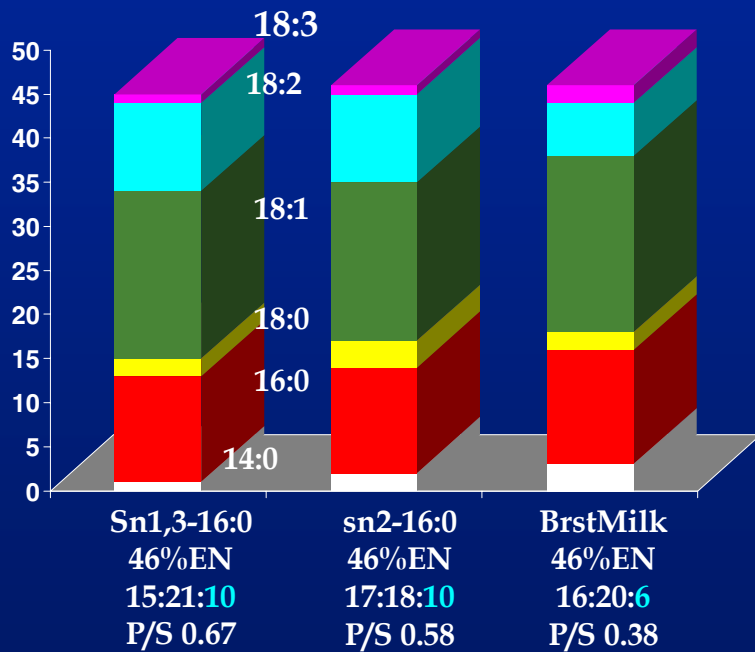
SERUM CHOLESTEROL



Point is, sn2 SFA appears most cholesterol-raising and can be exacerbated by low 18:2 + diet cholesterol.

Human Infant (120d) cholesterol response to sn1,3 or sn2-16:0 formulas vs breast milk

DIETARY FATTY ACIDS
(percent energy)



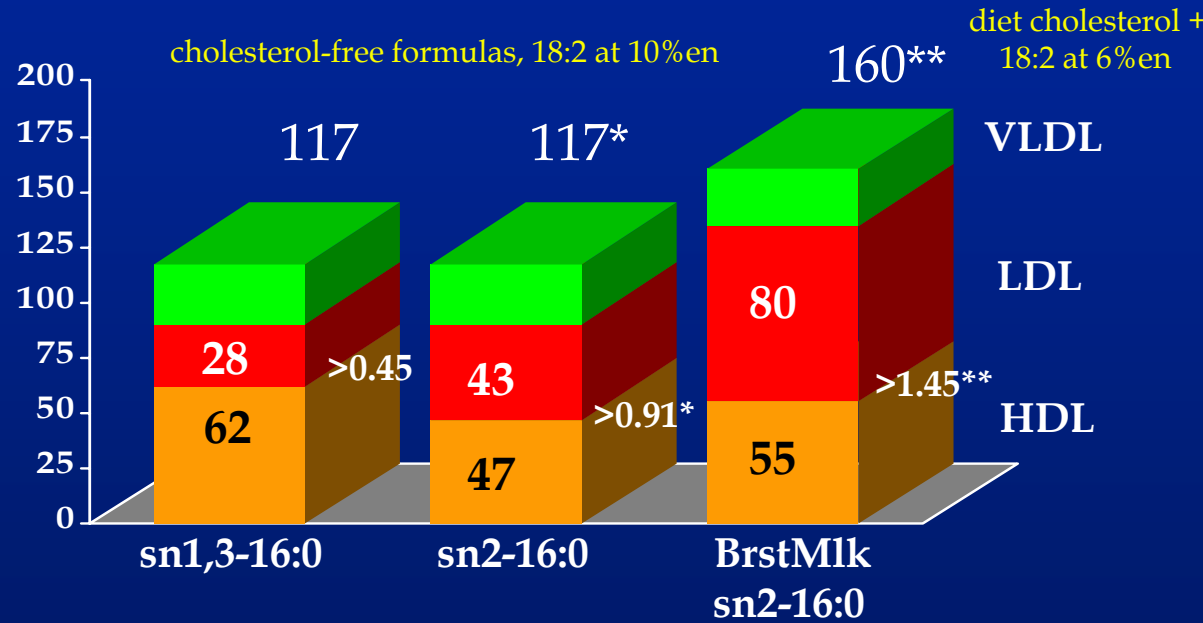
Sn1,3-16:0
46%EN
15:21:10
P/S 0.67

sn2-16:0
46%EN
17:18:10
P/S 0.58

BrstMilk
46%EN
16:20:6
P/S 0.38

Palm Betapol

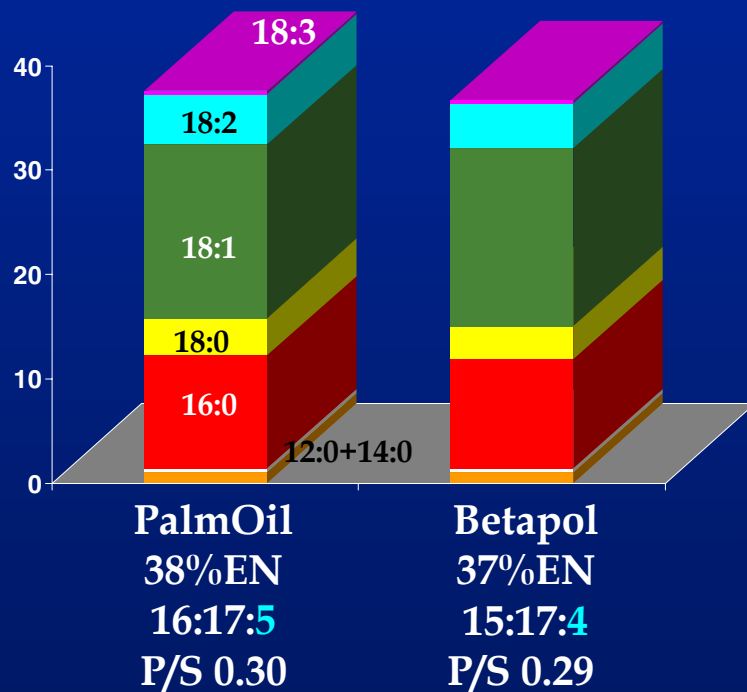
SERUM CHOLESTEROL



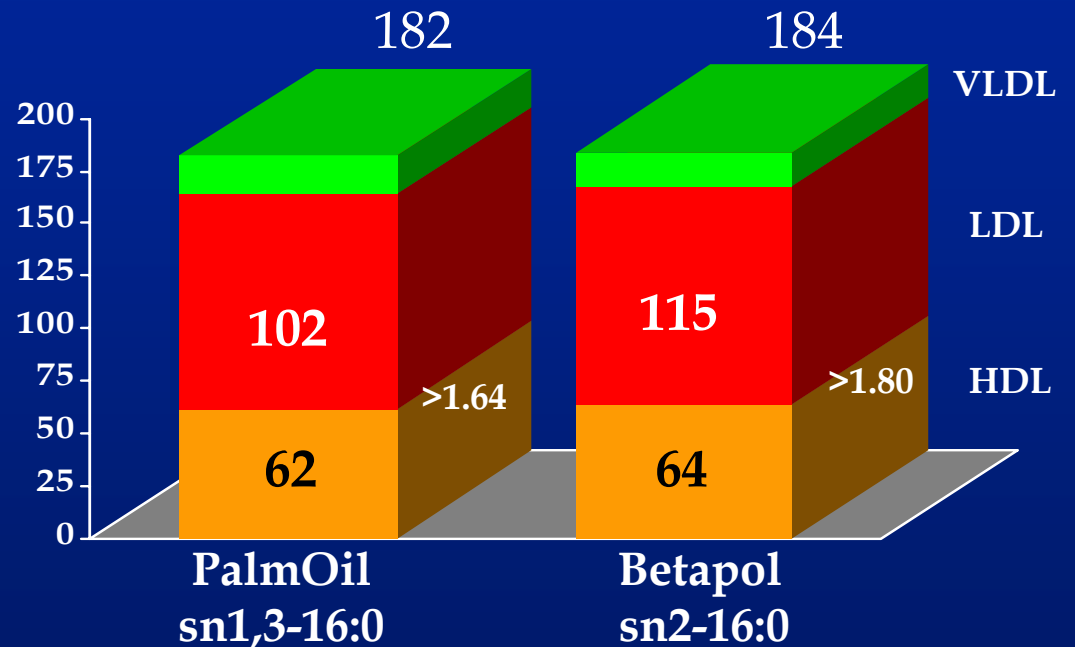
Point is, sn2 SFA appears to distort the LDL/HDL ratio and can be exacerbated by low 18:2 + diet chol, especially in infants without established 18:2 reserves.

Human response to dietary fats with sn1,3-16:0 vs sn2-16:0

DIETARY FATTY ACIDS
(percent energy)



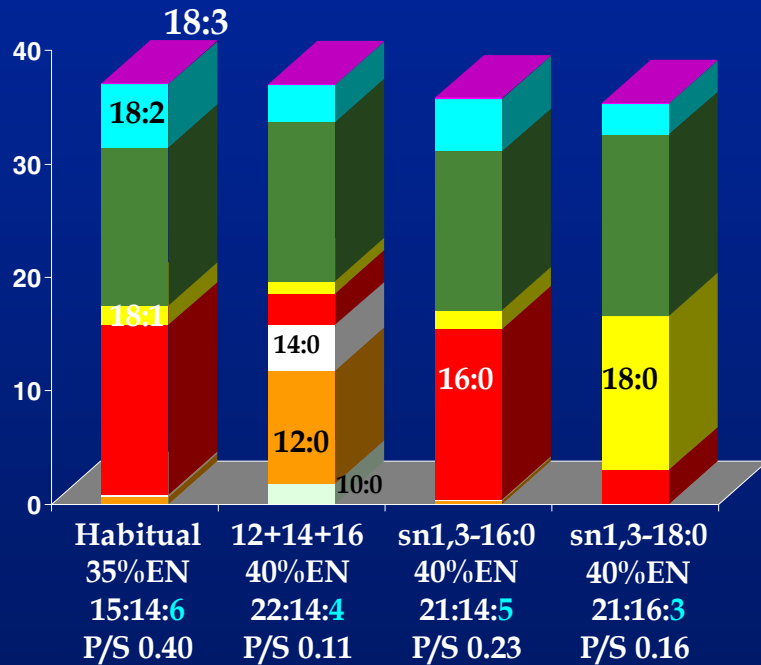
SERUM CHOLESTEROL



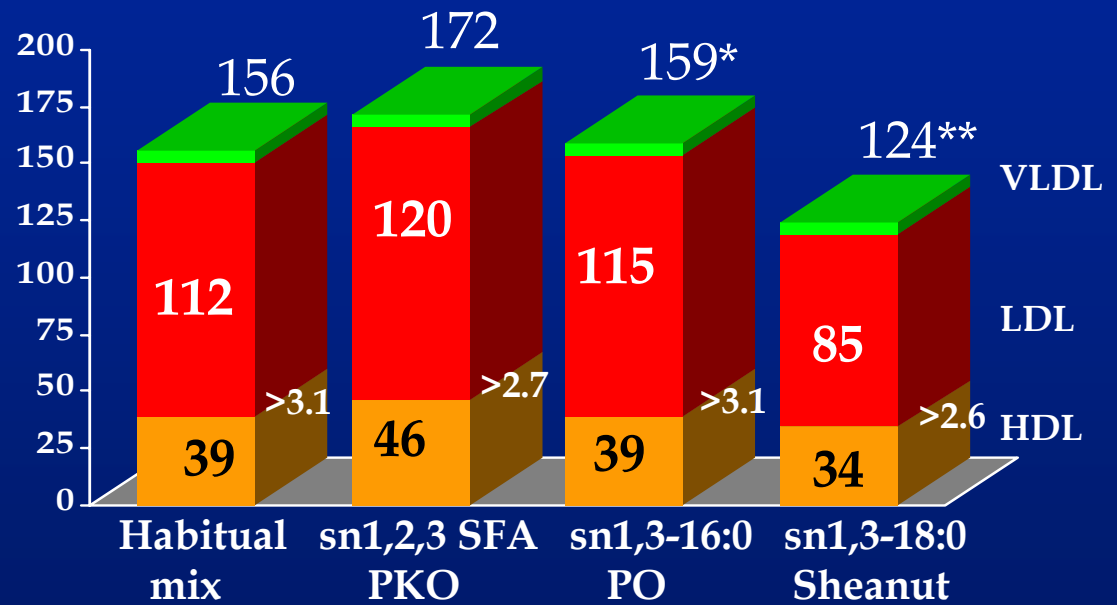
Point is, sn1,3-16:0 can appear equal to sn2-16:0 in adults, especially in women, at modest 18:2 intake.

Human response to natural saturated fats rich in 12+14:0, 16:0, or 18:0

DIETARY FATTY ACIDS
(percent energy)



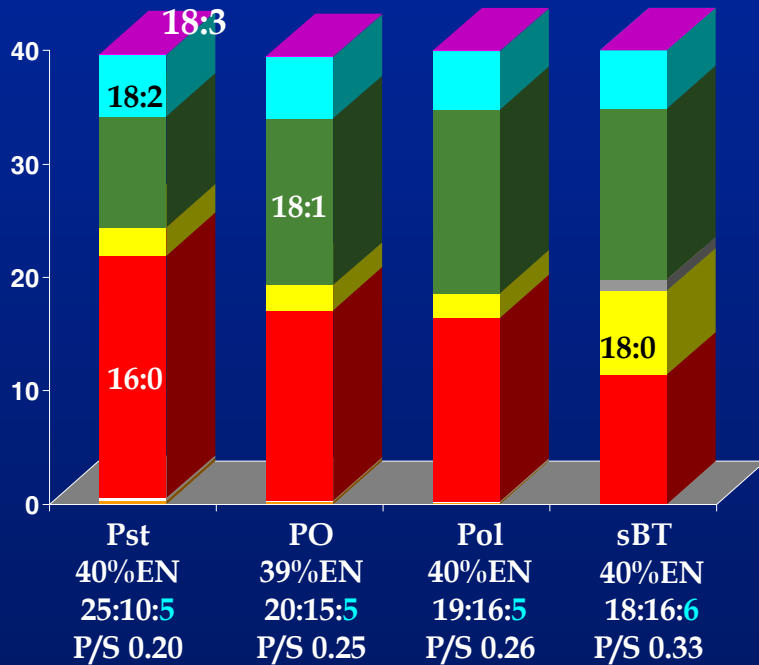
SERUM CHOLESTEROL



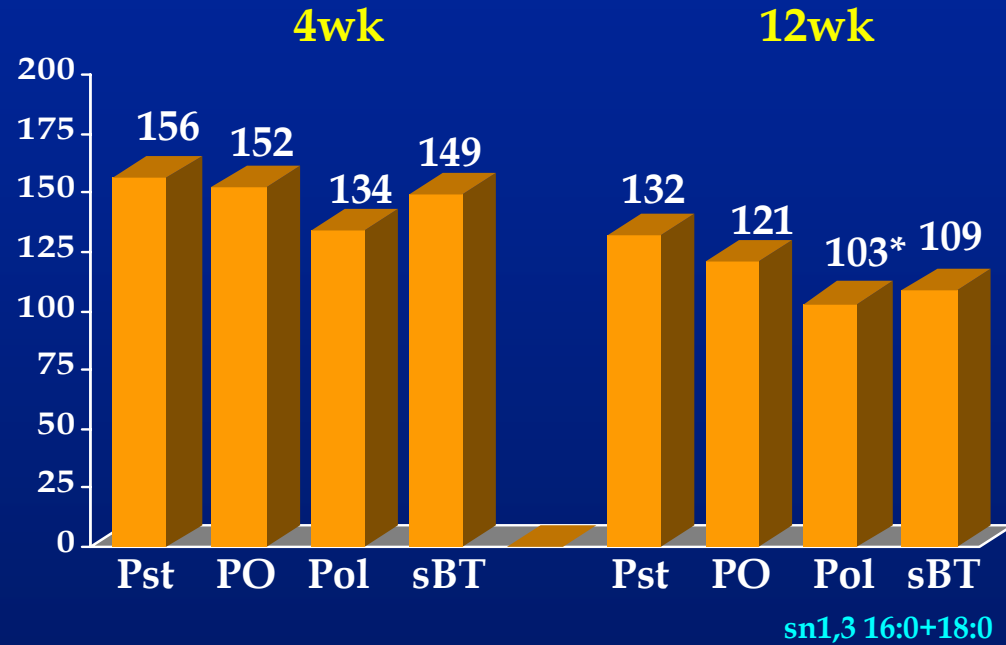
Point is, sn1,3 18:0 appears to lower cholesterol relative to sn1,3 16:0 at low 18:2 intake. Sn1,2,3-SFA most cholesterolemic, but HDL up too.

Gerbil response to 16:0 from various natural fat sources

DIETARY FATTY ACIDS (percent energy)



SERUM CHOLESTEROL



Point is, removing tri-16:0 (as Pst + PO) induces the largest cholesterol decline, even tho the total 16:0 was about the same and 18:2 intake was constant..



Re-examined McGandy and Hegsted 1970 AJCN
“Interesterified various fats with triSFA”

Provided

- Total serum cholesterol
- and beta-lipoproteins from acetate electrophoresis (est LDL),
- and TG

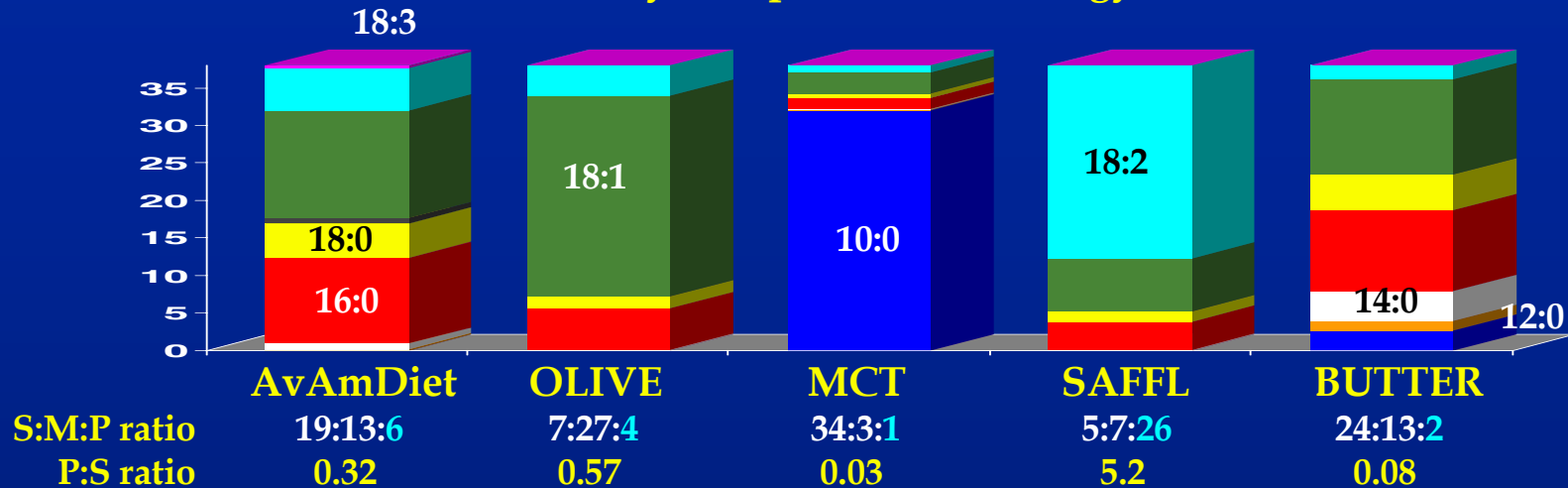
Calculated **VLDL-C** using the Friedwald equation:
as total TG/5

Calculated **HDL-C** = TC - (LDL-C + VLDL-C)

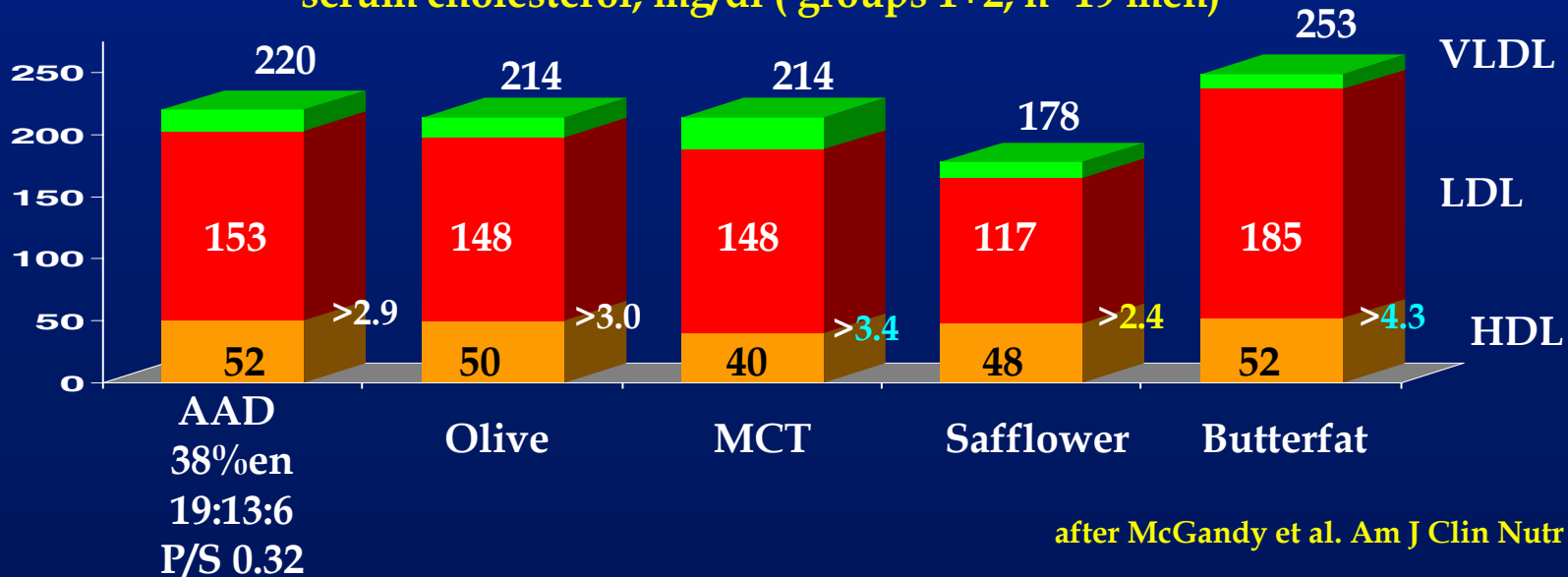


Cholesterol response to fats of different saturation in humans

diet fatty acid profile, 38%energy as fat



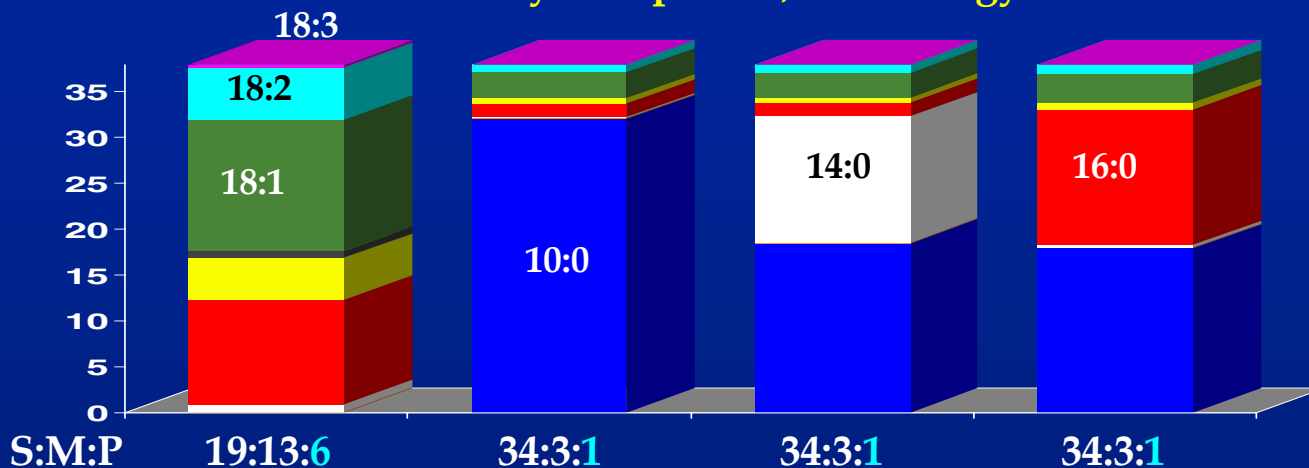
serum cholesterol, mg/dl (groups 1+2, n=19 men)



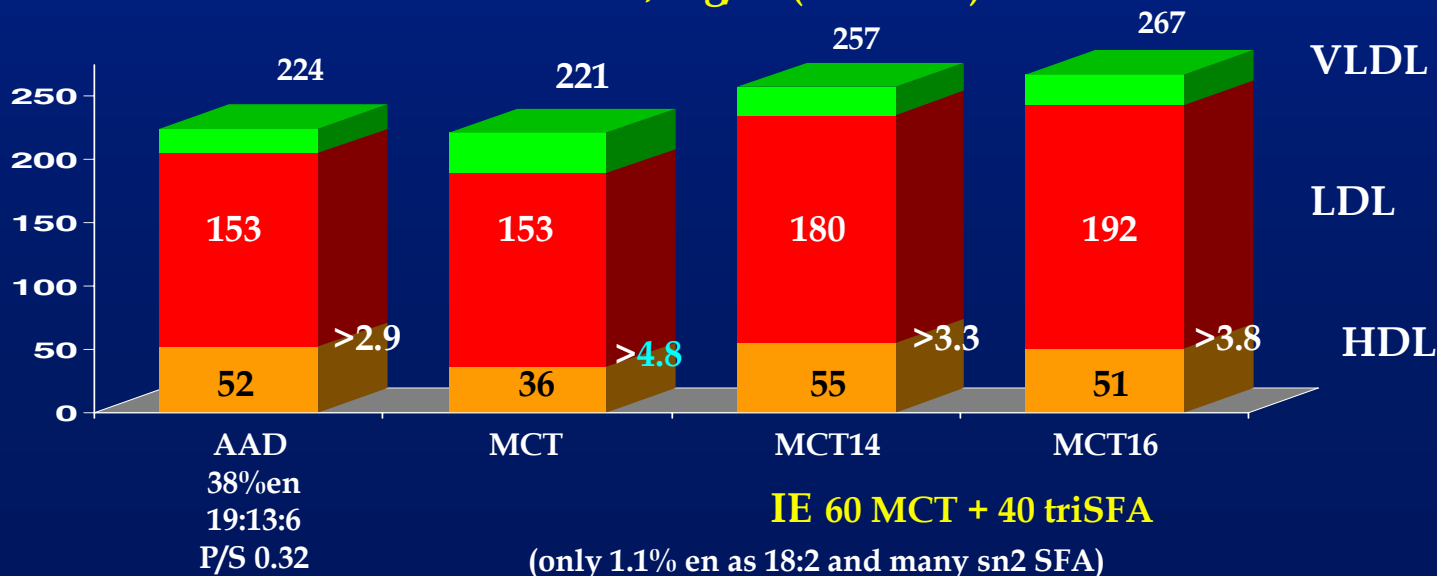
Cholesterol response to IE fats based on MCT + tri14, tri16 in humans (group1)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat



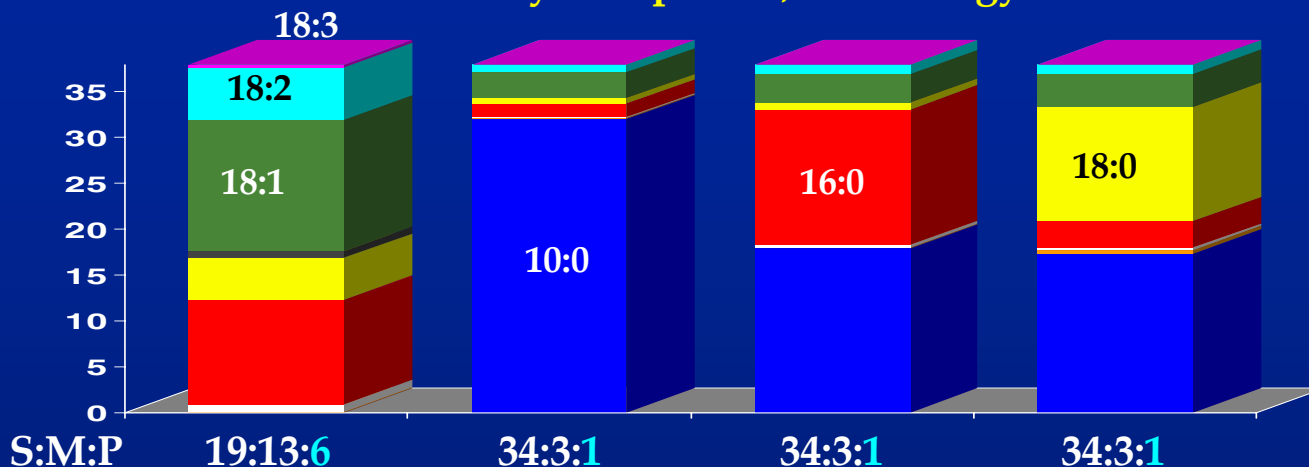
serum cholesterol, mg/dl (n=9 men)



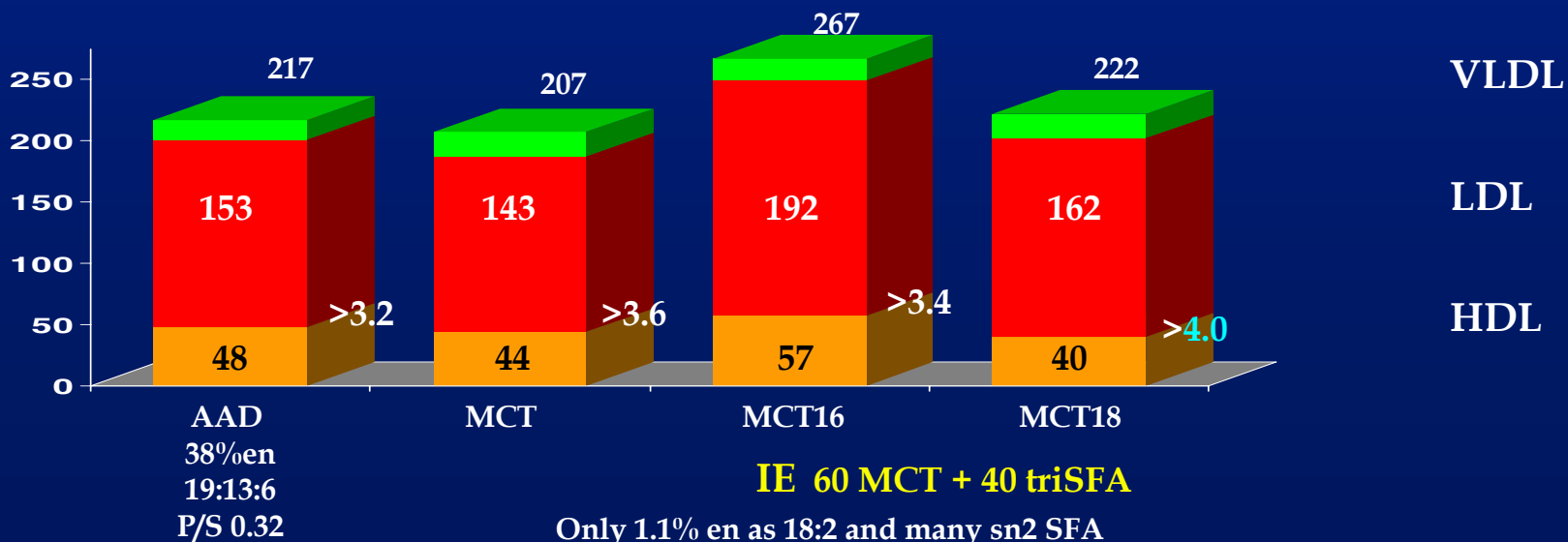
Cholesterol response to IE fats based on MCT + tri16, tri18 in humans (group2)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat



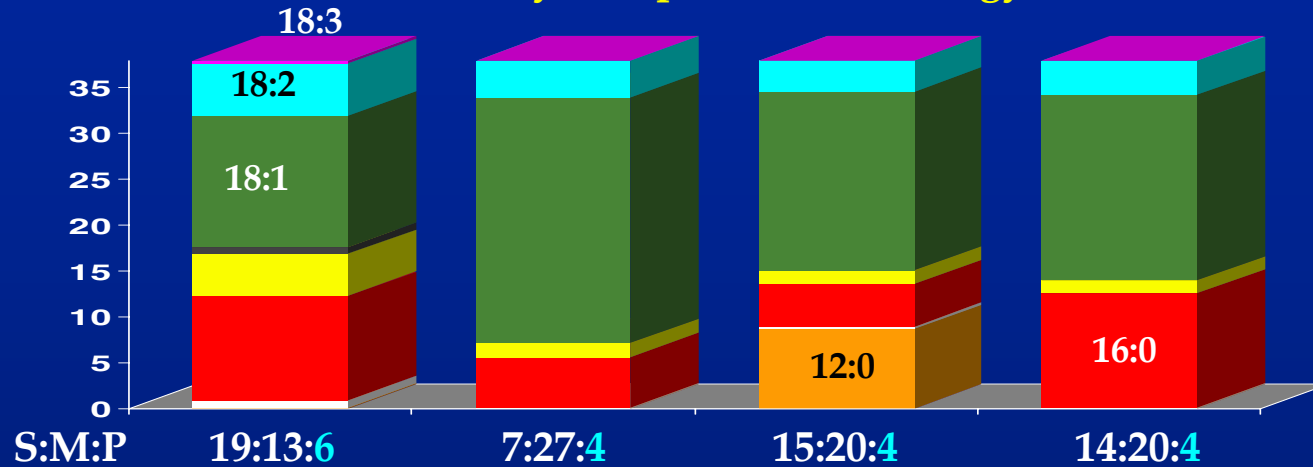
serum cholesterol, mg/dl (n=9 men)



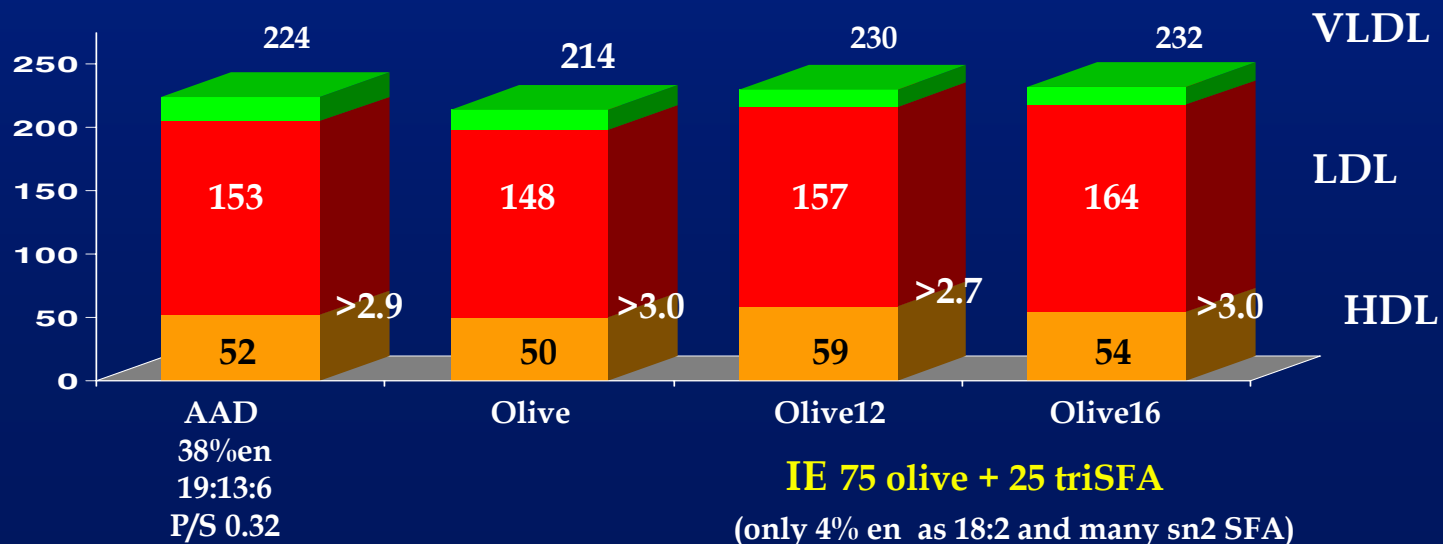
Cholesterol response to IE fats based on Olive Oil + tri12, tri16 in humans (group1)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat



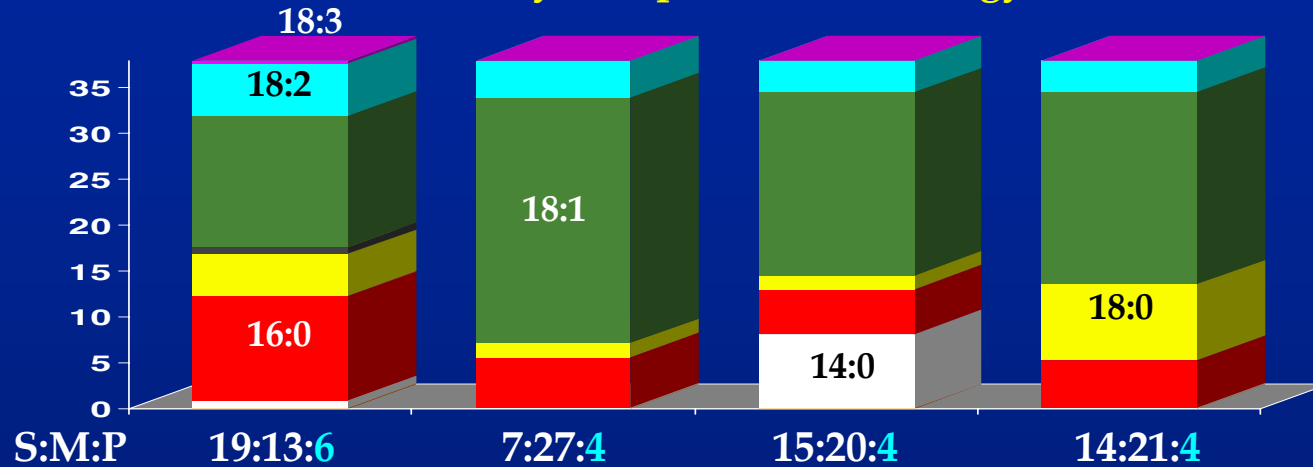
serum cholesterol, mg/dl (n=9 men)



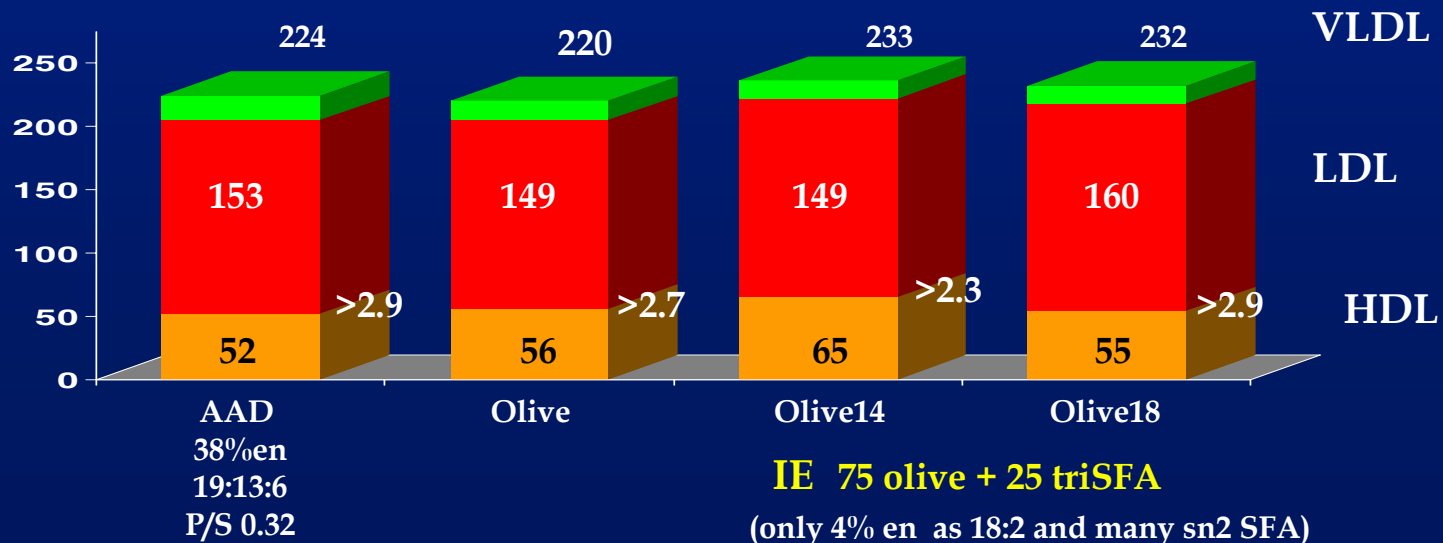
Cholesterol response to IE fats based on Olive Oil + tri14, tri18 in humans (group2)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat



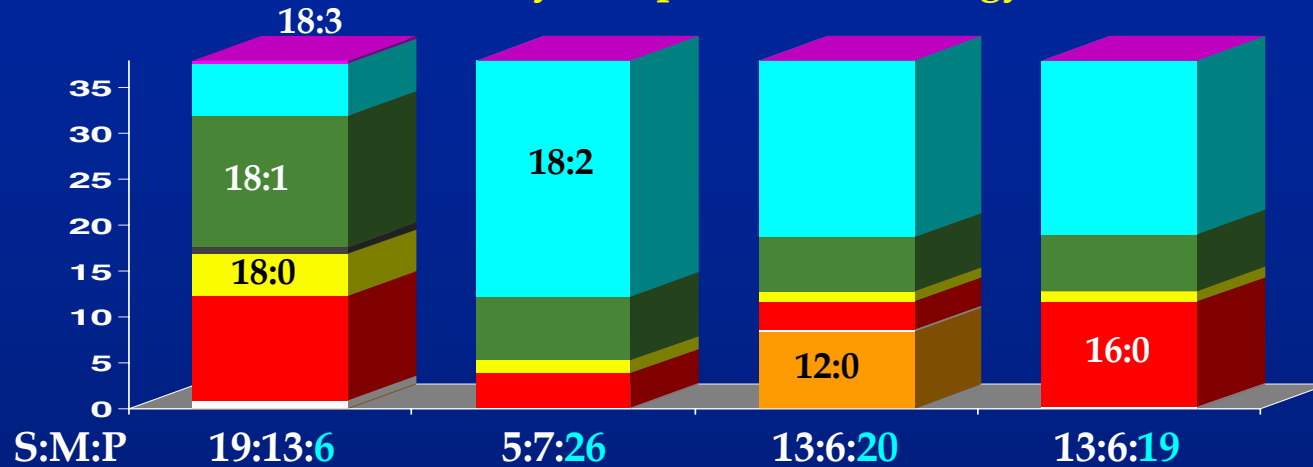
serum cholesterol, mg/dl (n=9 men)



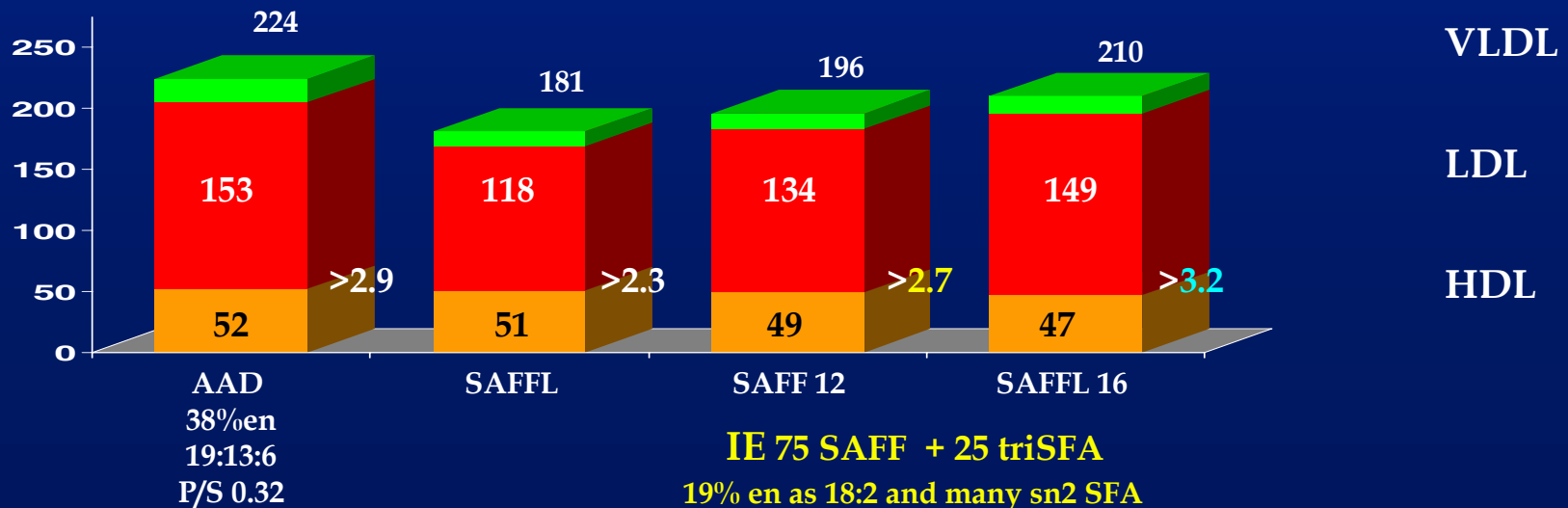
Cholesterol response to IE fats based on SAFFL + tri12, tri16 in humans (group1)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat



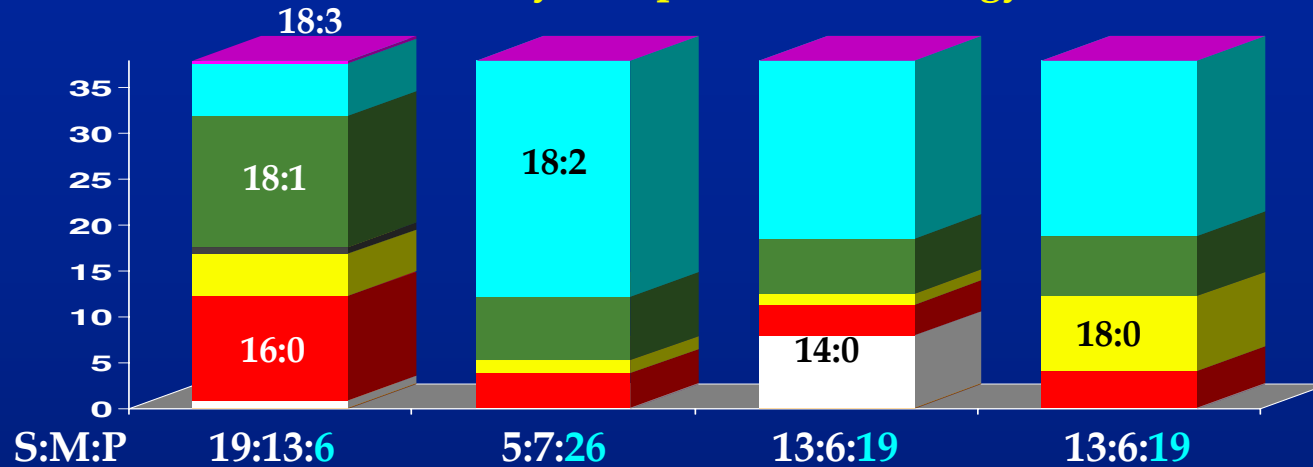
serum cholesterol, mg/dl (n=9 men)



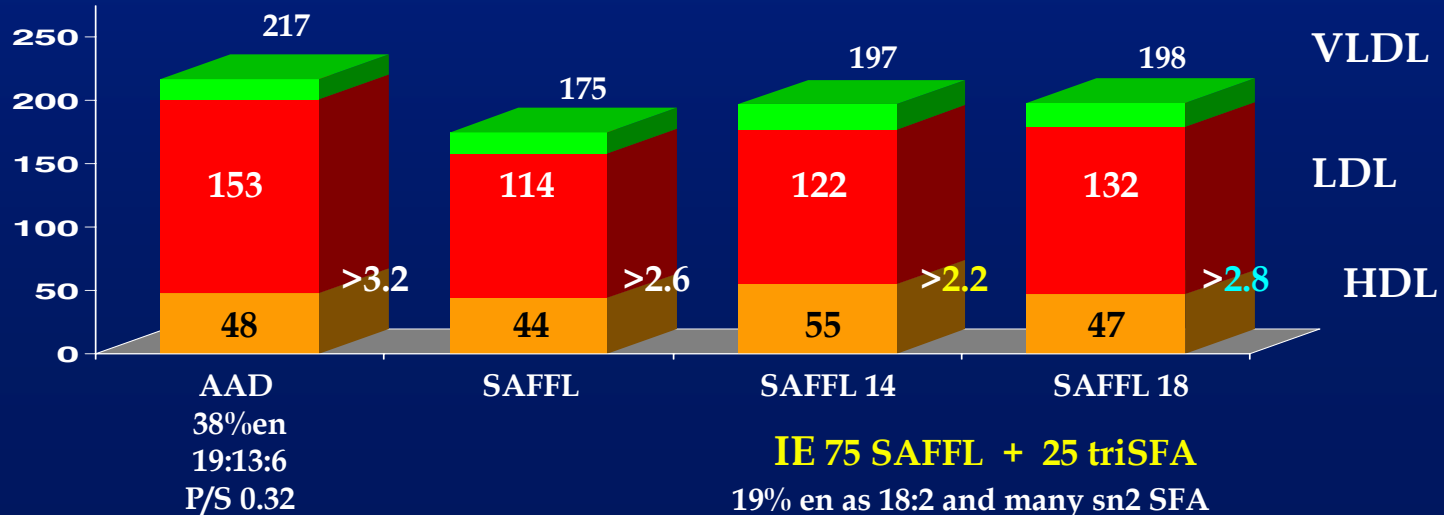
Cholesterol response to IE fats based on SAFFL + tri14, tri18 in humans (group2)

after McGandy et al. Am J Clin Nutr 23: 1288, 1970

diet fatty acid profile, 38%energy as fat

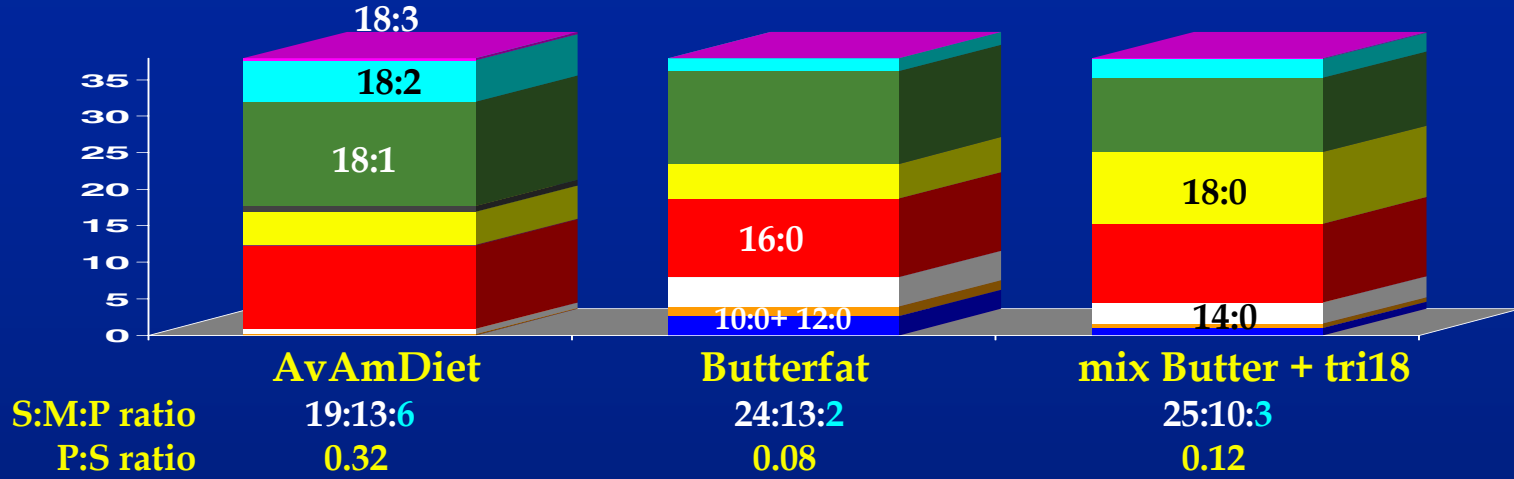


serum cholesterol, mg/dl (n=9 men)

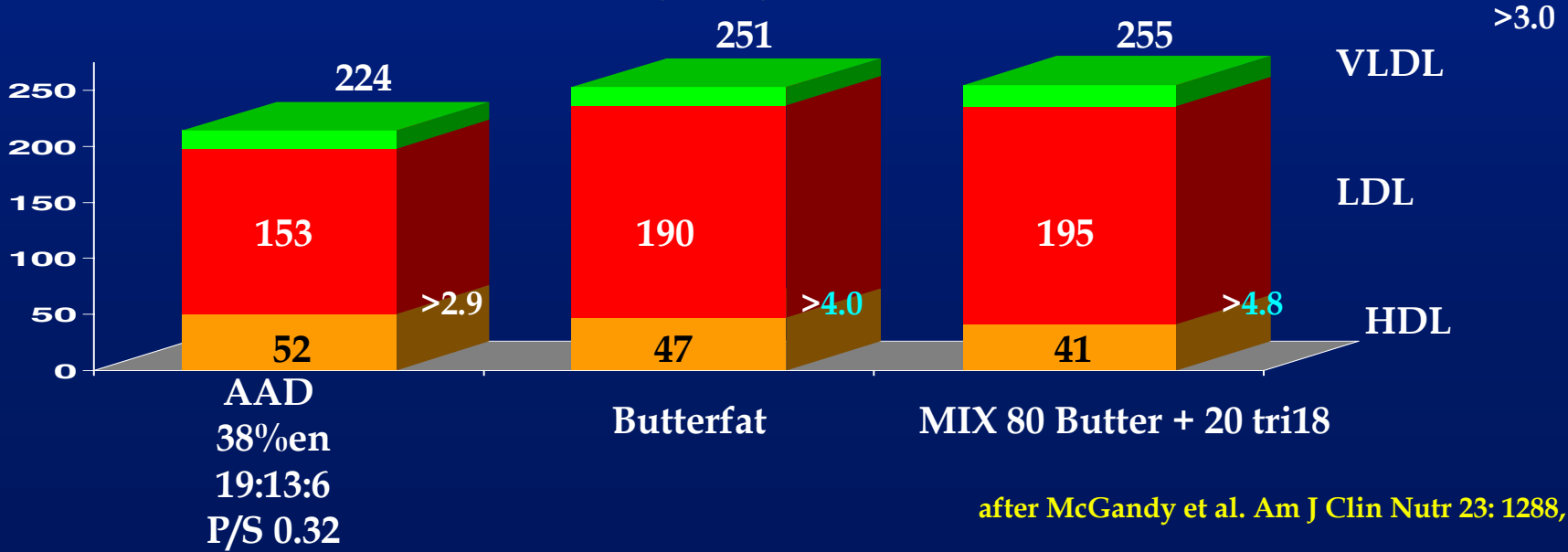


Cholesterol response to butterfats +/- tri18:0 in humans

diet fatty acid profile, 38%energy as fat



serum cholesterol, mg/dl (groups 1+2, n=19 men)





Summary McGandy et al

Adding SFA into MCT or monounsaturated or polyunsaturated oils has a deleterious effect on lipoprotein cholesterol

12:0 and 14:0 seem more HDL-raising than 16:0 or 18:0.

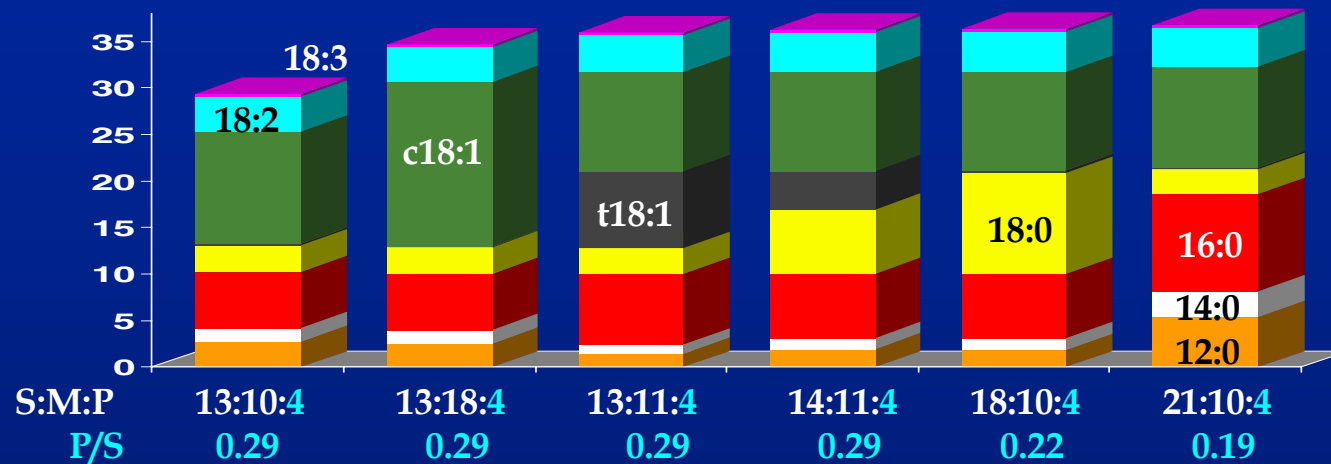
High intake of 18:0 seems to have the biggest HDL lowering potential (like TFA?)



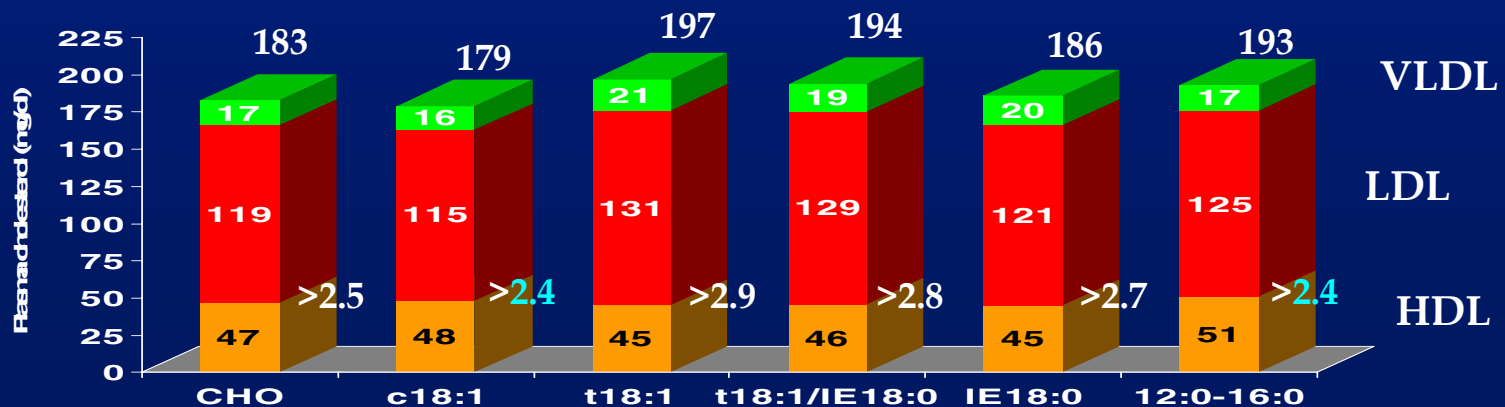
Cholesterol response to t18:1 + IE-18:0 fats in humans

50 men@ 42y+26 BMI/ 5wk periods/crossover

diet fatty acid profile, 38%energy as fat



serum cholesterol, mg/dl (n=50 men)

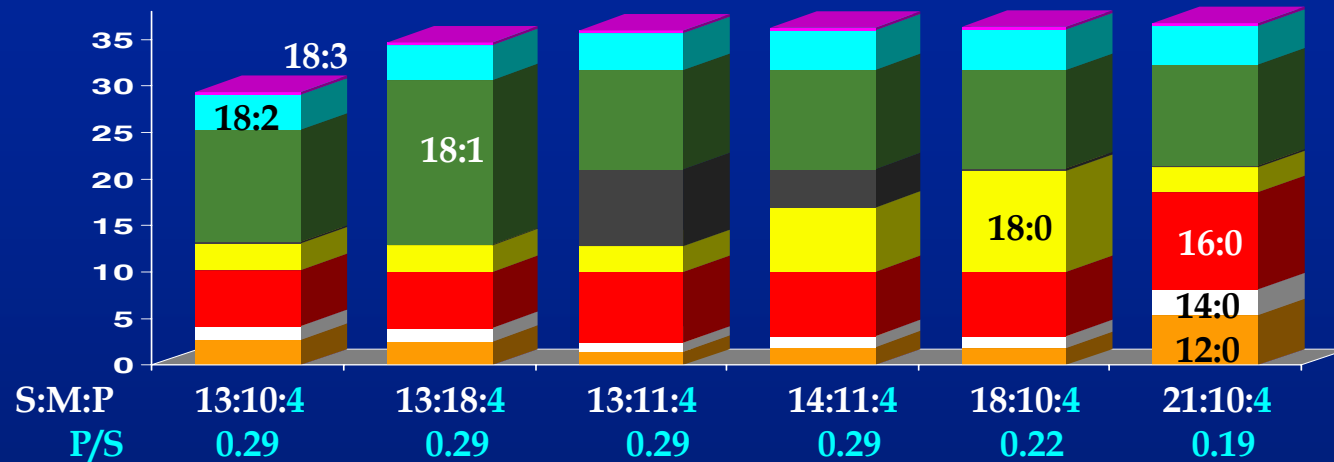


Point is, TFA and IE18:0 depress HDL and raise LDL/HDL ratio.

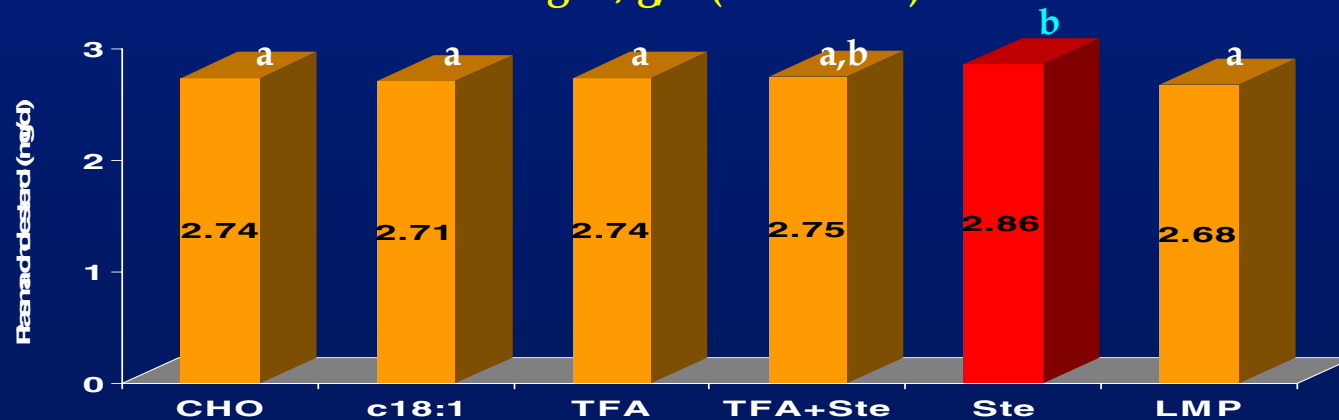
Judd et al. Lipids 37:123, 2002

Fibrinogen response to t18:1, IE-18:0, and LC SFA in humans

diet fatty acid profile, 38%energy as fat

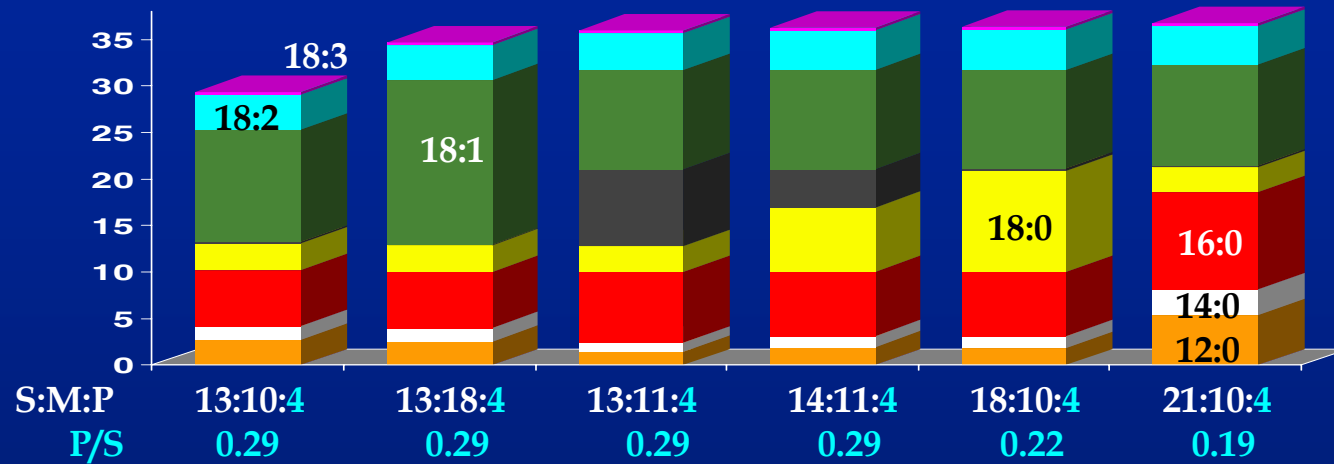


fibrinogen, g/L (n=50 men)

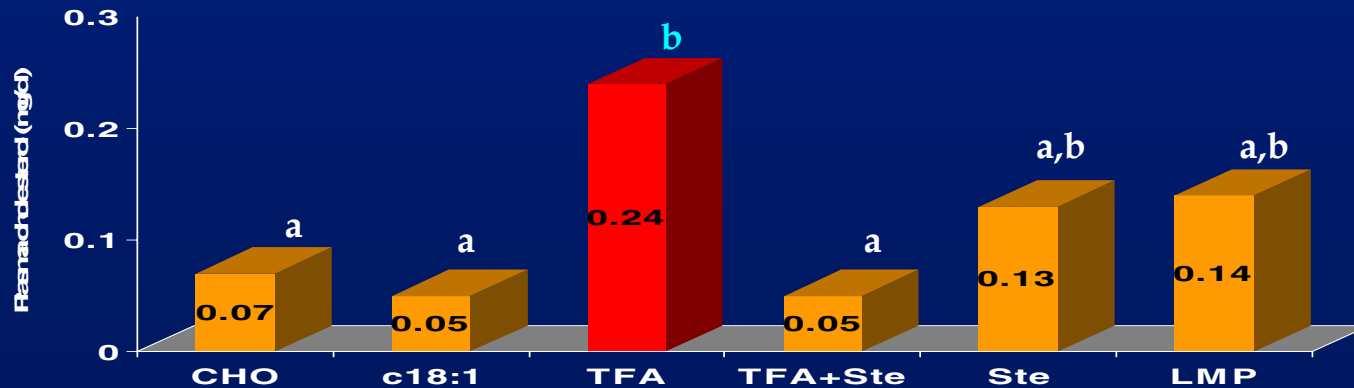


C-reactive protein response to t18:1, IE-18:0, LC SFA in humans

diet fatty acid profile, 38%energy as fat

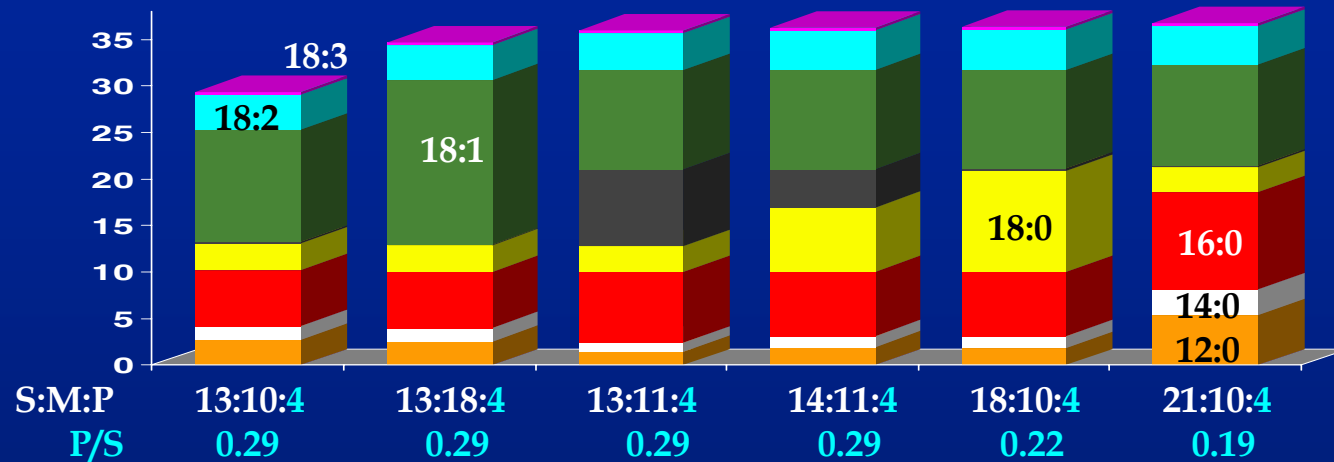


C-reactive protein, mg/L (n=50 men)

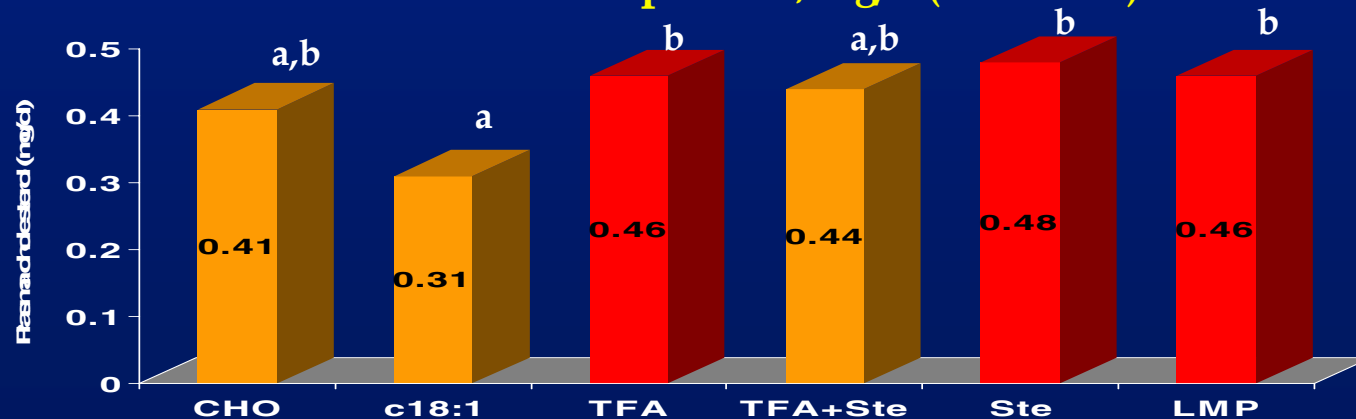


IL-6 response to t18:1, IE-18:0, LC SFA in humans

diet fatty acid profile, 38% energy as fat

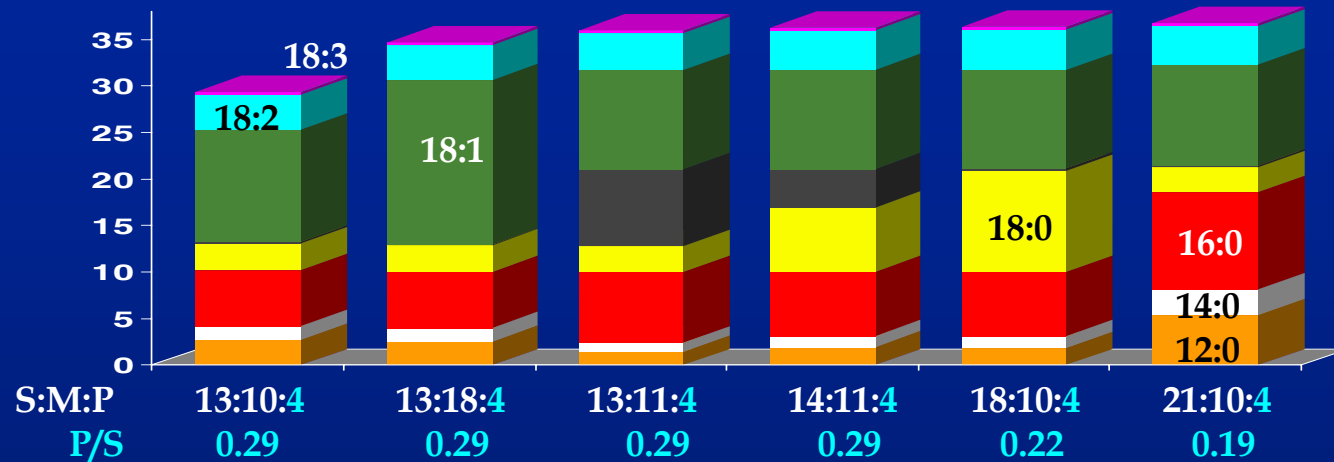


C-reactive protein, mg/L (n=50 men)

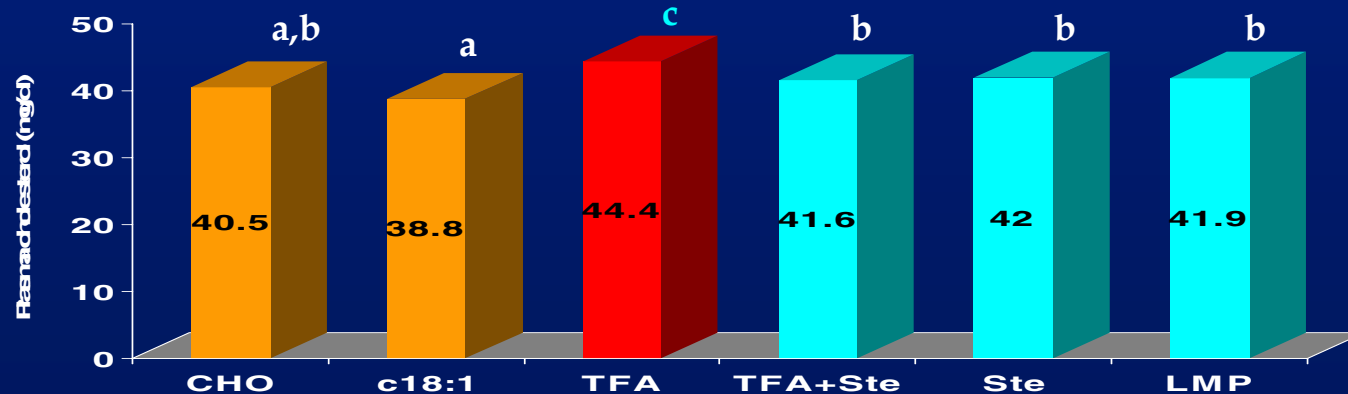


C-reactive protein response to t18:1, IE-18:0, LC SFA in humans

diet fatty acid profile, 38% energy as fat

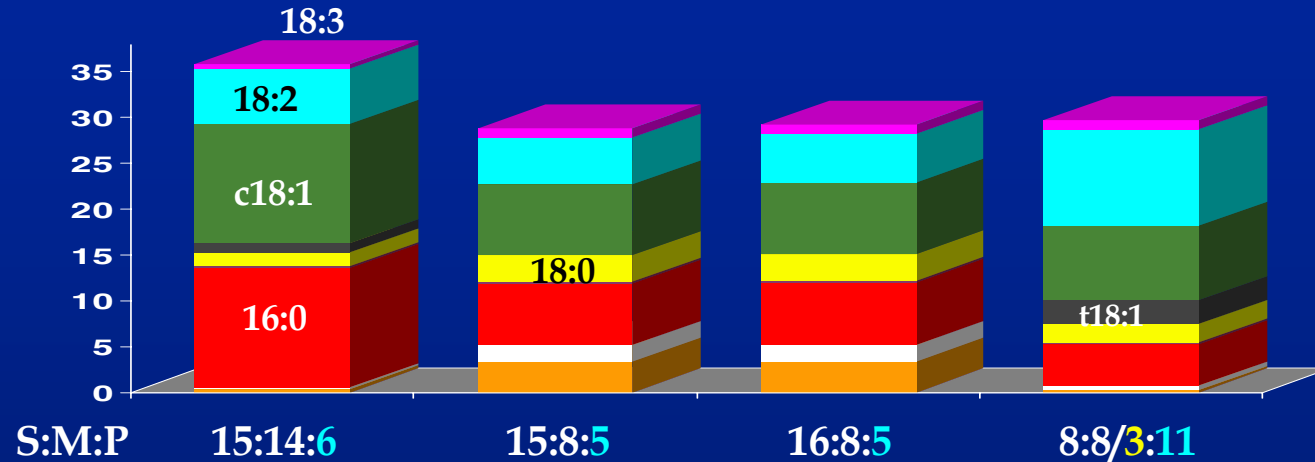


E-selectin ng/mL (n=50 men)

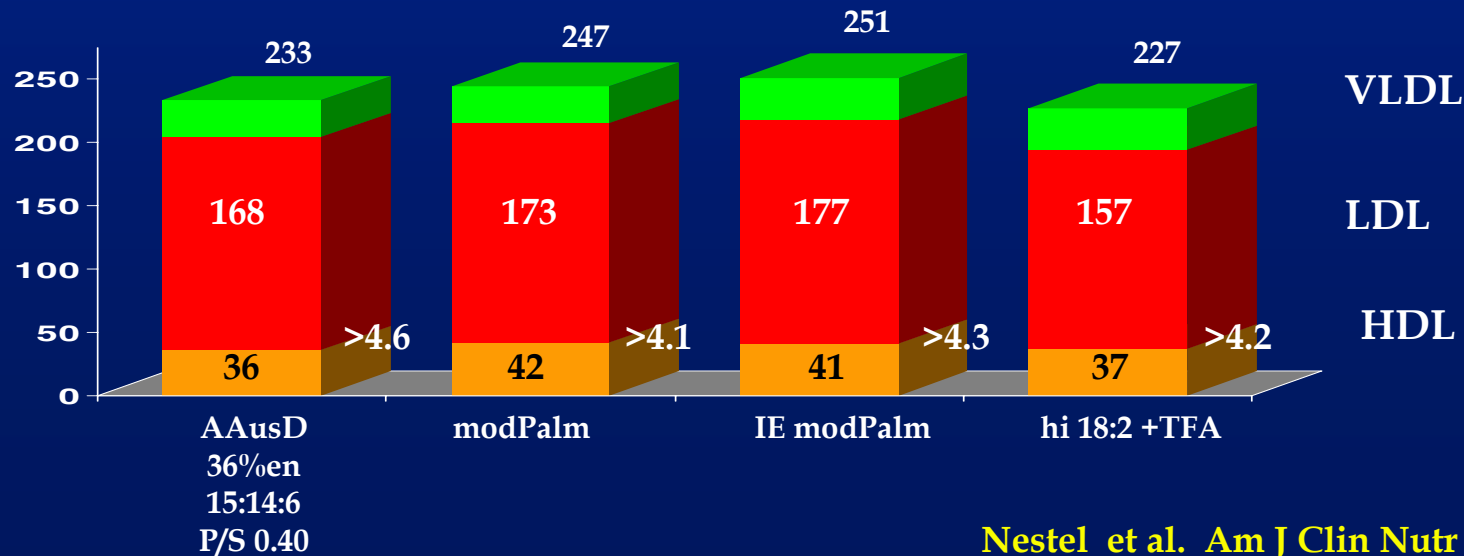


Cholesterol response to modPalm, IE-modPalm, and hi-18:2+TFA in humans

diet fatty acid profile, 31%energy as fat

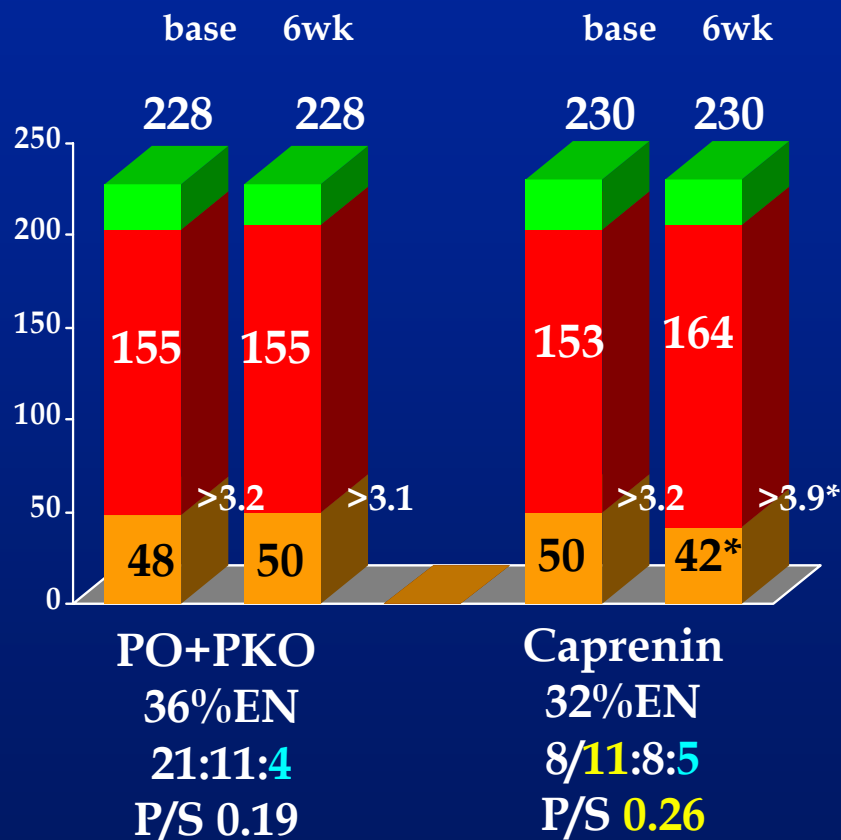


serum cholesterol, mg/dl (n=27 men)

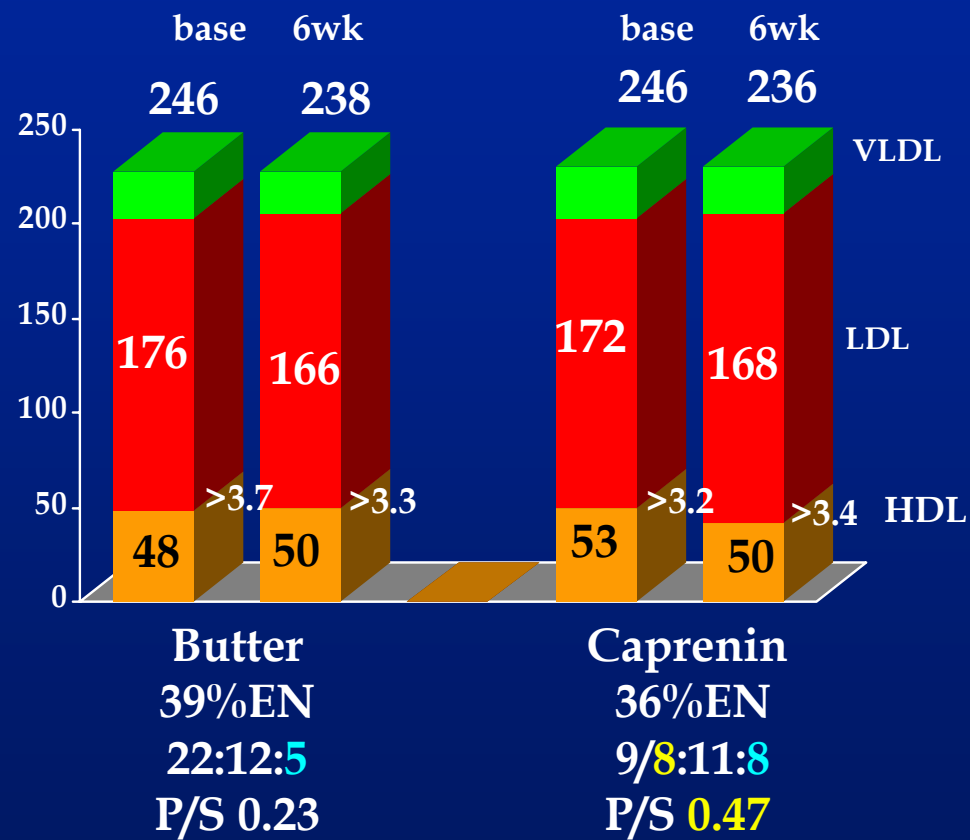


Human plasma cholesterol response to PKO or Butter vs Caprenin at two P/S ratios

Study 1 (n=17M+17M)



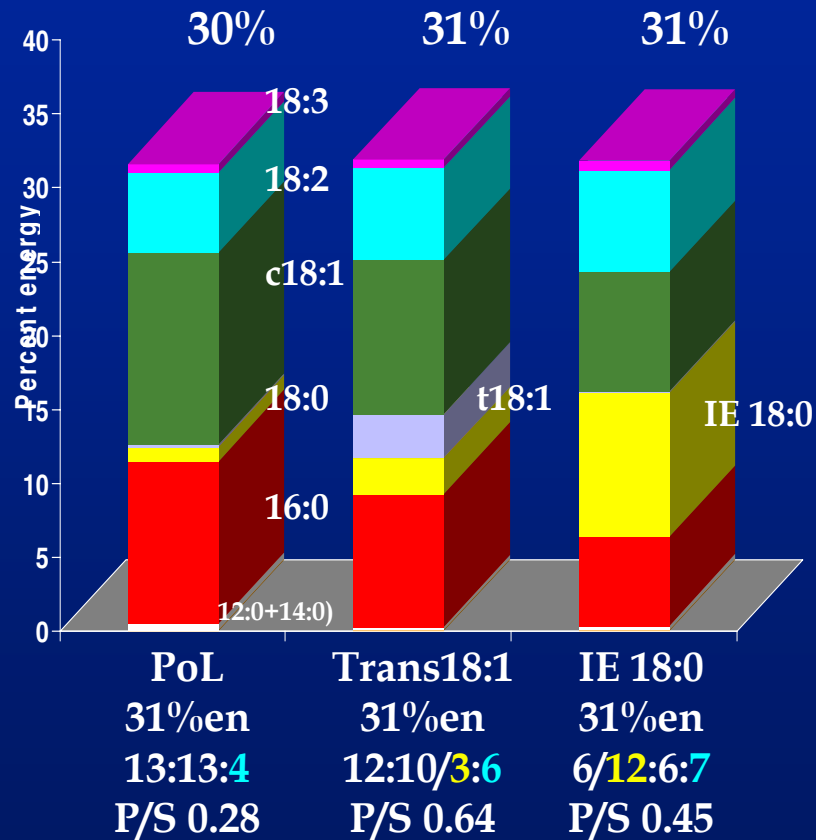
Study 2 (n=7M+7M)
hyper-responders from Study 1



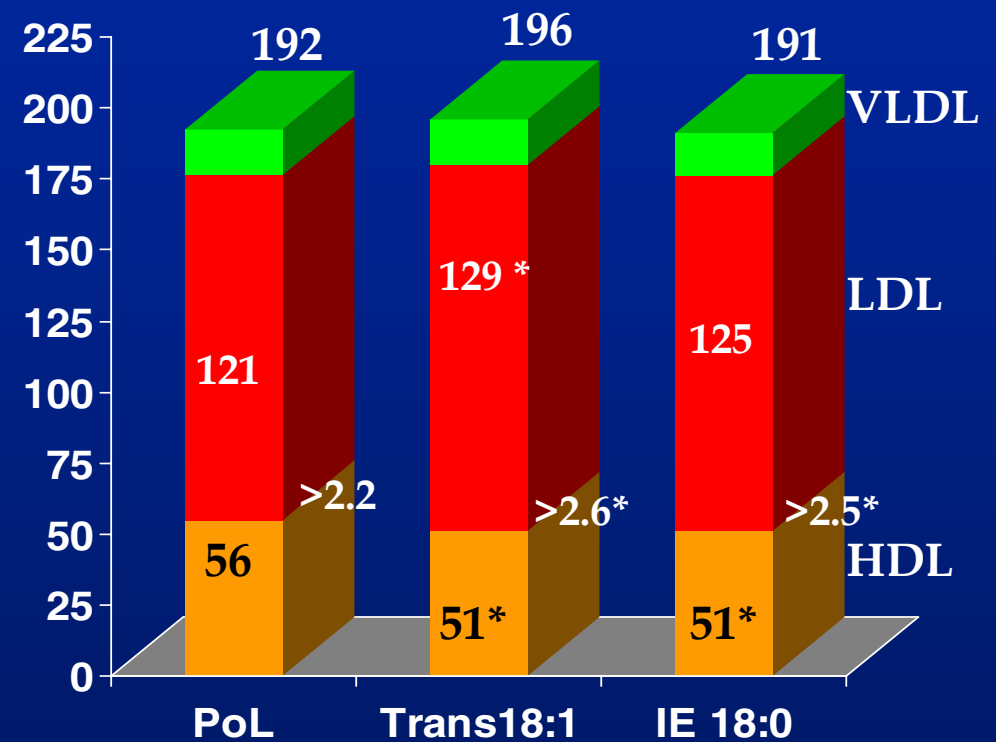
Point is, sn2 SFA can raise LDL/HDL ratio and may be exaggerated at low 18:2.

Natural 16:0 vs TRANS 18:1 and IE 18:0 in HUMANS

DIETARY FATTY ACIDS, %en



LIPOPROTEIN PROFILE



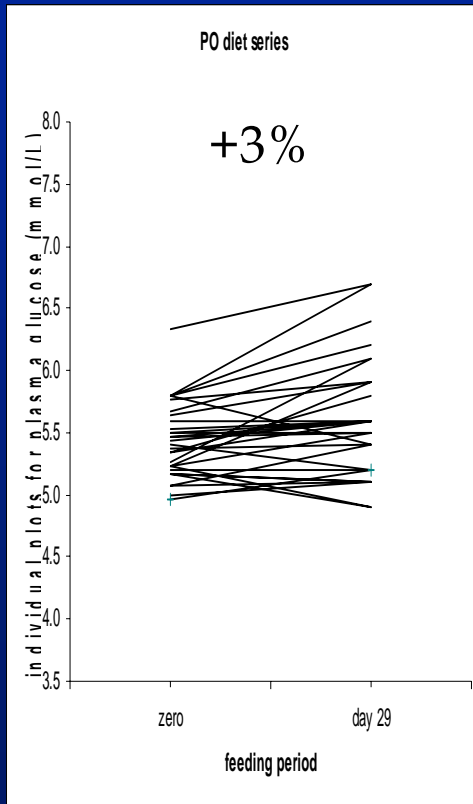
Individual fasting glucose response in humans to three test fats from 0wk to 4wk

insulin (microUnits/ml)

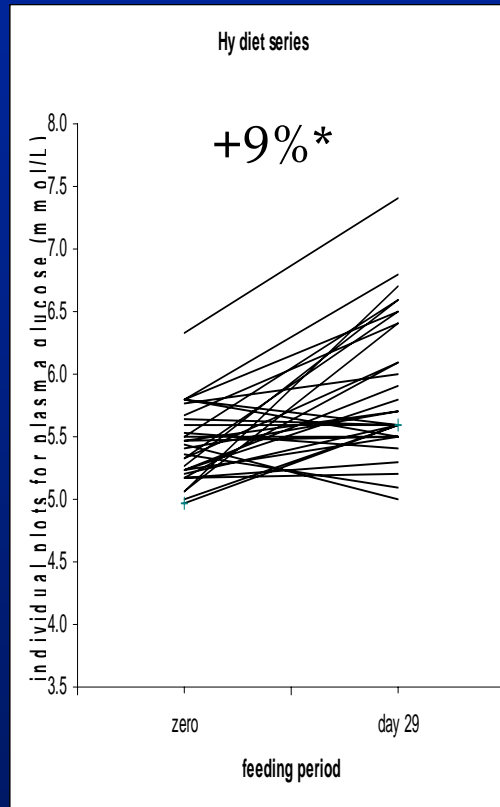
10.1(control)

9.1 (-10%)

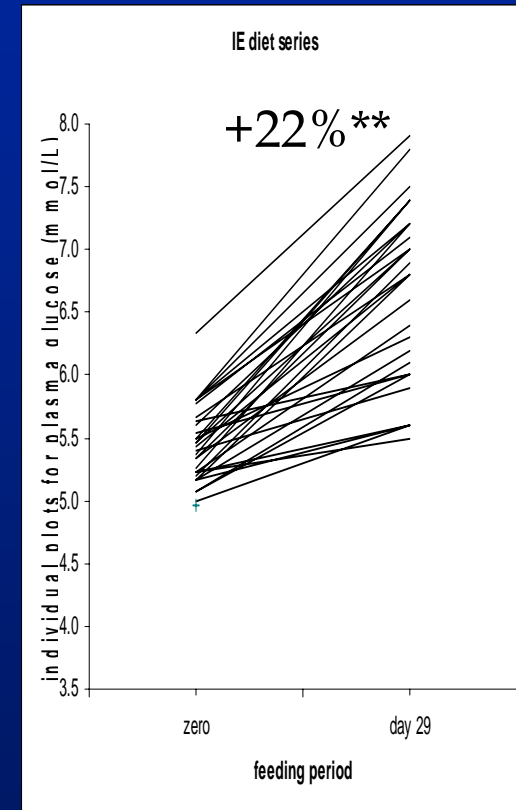
7.9 (-22%)



POL



PHSO



IE

Conclusions

"Balance the technical need with the biology"

It's not the **FAT**, but the **fatty acids**.. and , in addition, **the TG-MS**.

18:2 more critical than **SFA**....not **14:0** per se, but total SFA on TG.

Tri-SFA are the most offensive fats (TG).... because they lack **18:2** and **sn2** is SFA.

So **saturated fats** are a problem because they do not contain enough **sn2-PUFA** to counter their **SFA** content....but it's fixable . Add **POLYS**.

Introducing modified **sn2-SFA** is problematic....trans? **IE-18:0**? **IE-SFA**?

Gender seems important; total level of diet fat; diet **P/S** ratio; lipoprotein profile at entry.



Summary

Two alternatives to trans fat ?

- natural saturates with adequate PUFA
- IE fats leaving sufficient PUFA?

18:0 or 16:0 for interesterification?...not clear if equally bad...seem different.

Important that sn2 be UNSAT, 18:2n6 (+sn2-n3FA)

avoid sn2 SFA, or problems can develop at low 18:2 intake

