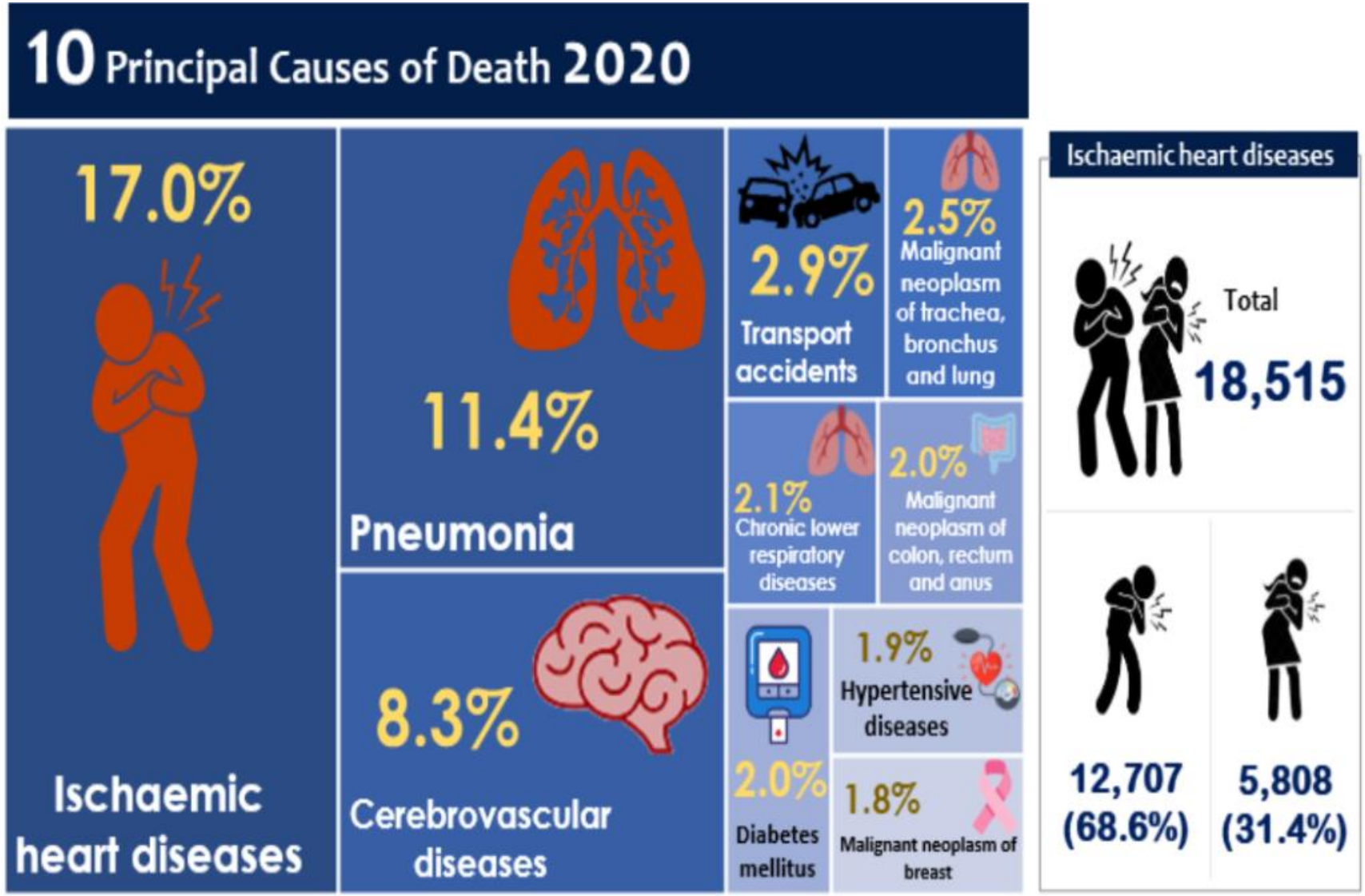
RỐI LOẠN LIPID MÁU NGƯỜI CHÂU Á CÓ GÌ ĐẶC BIỆT ?

Góc nhìn chuyên ngành Nội Khoa

GS.TS.Nguyễn Hải Thuỷ
PCT Hội Nội Khoa Việt Nam

1. ĐẶT VẤN ĐỀ

Bệnh tim mạch do xơ vữa là nguyên nhân tử vong hàng đầu trên thế giới (2020)



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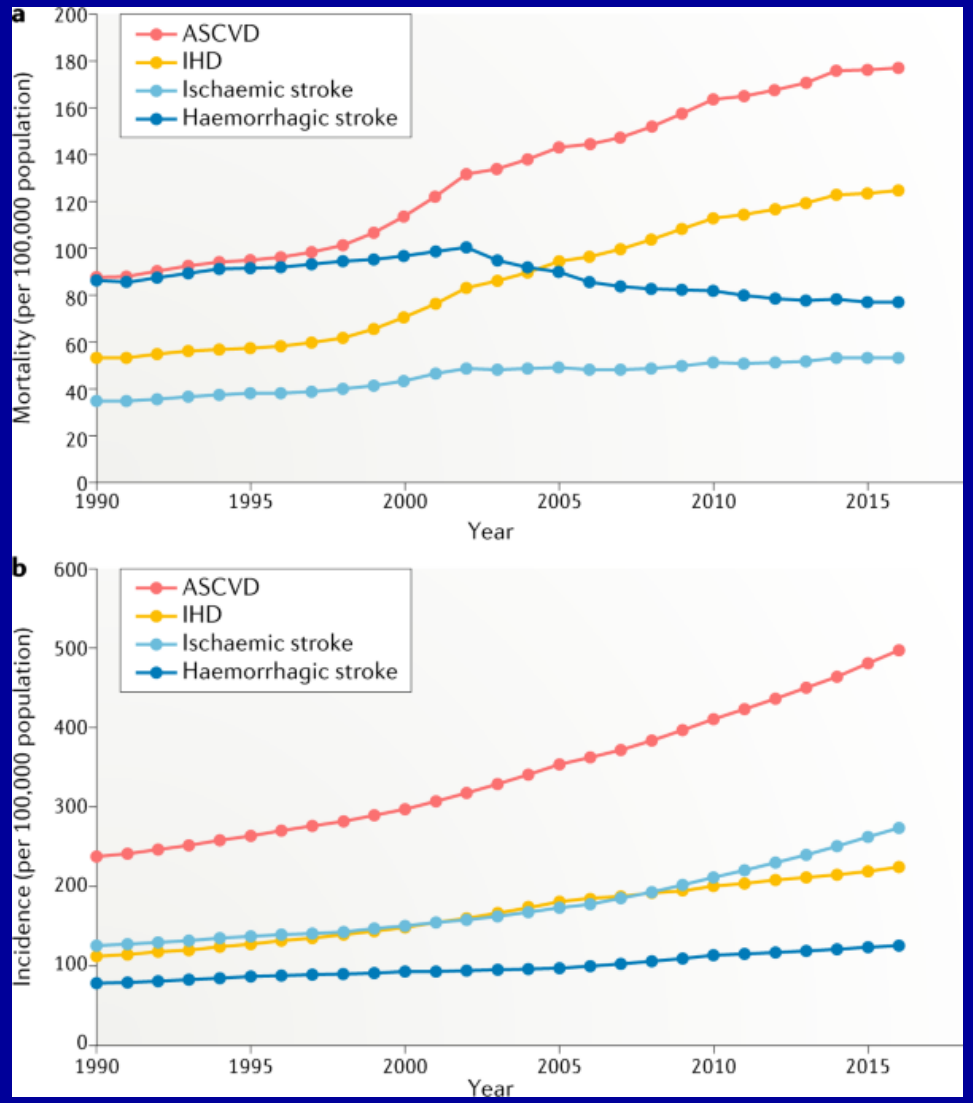

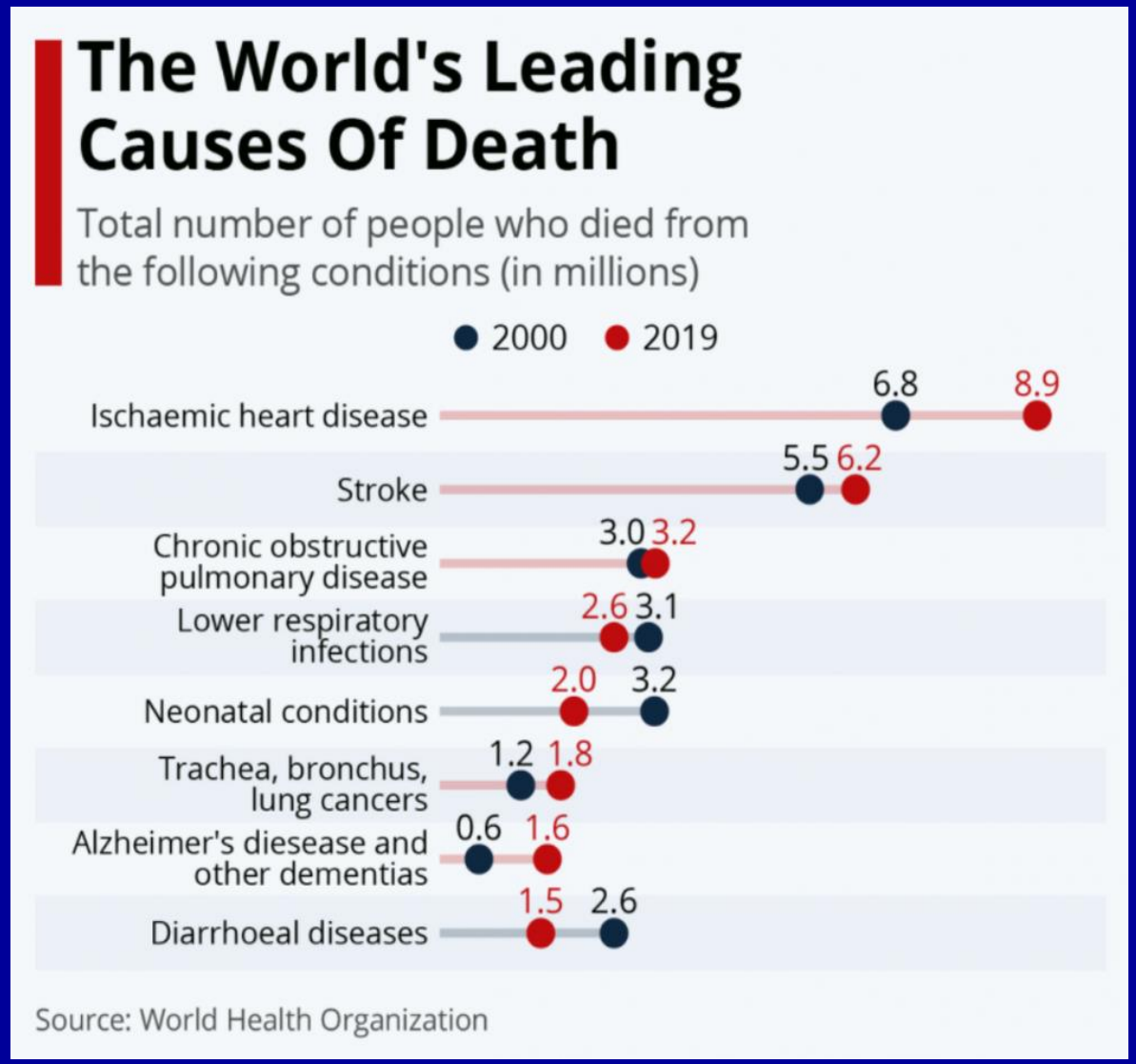

Mặc dù y học ngày càng tiến bộ nhưng tỷ lệ tử vong do BTMXV có khuynh hướng ngày càng tăng

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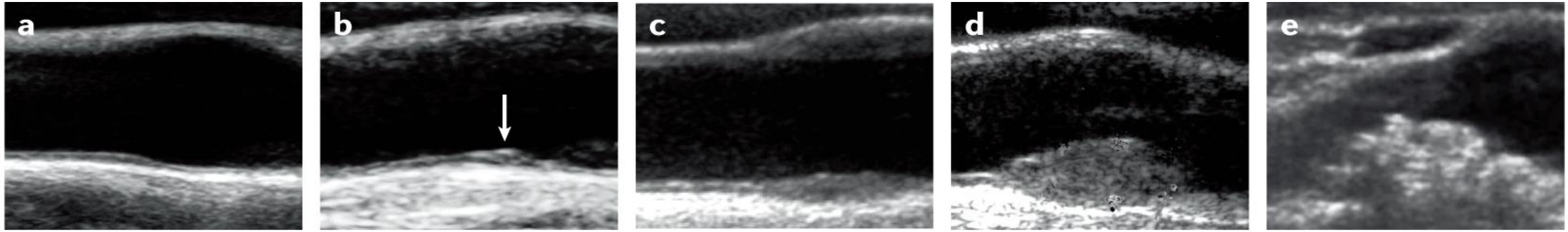
a
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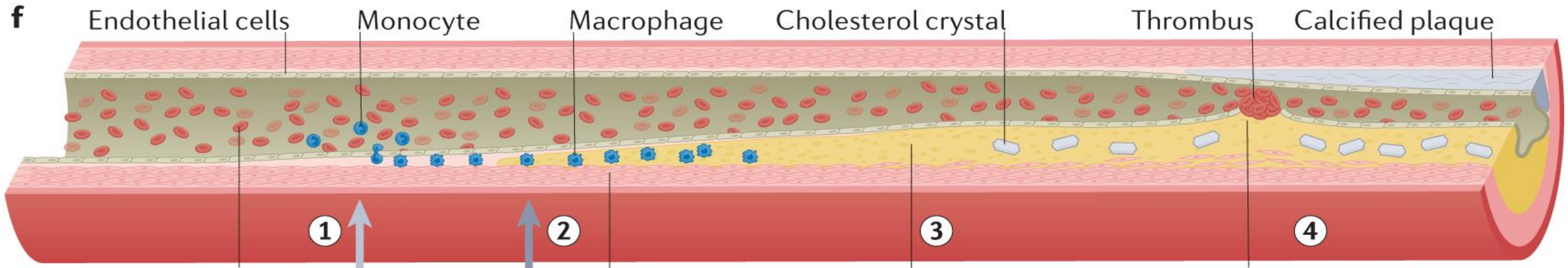
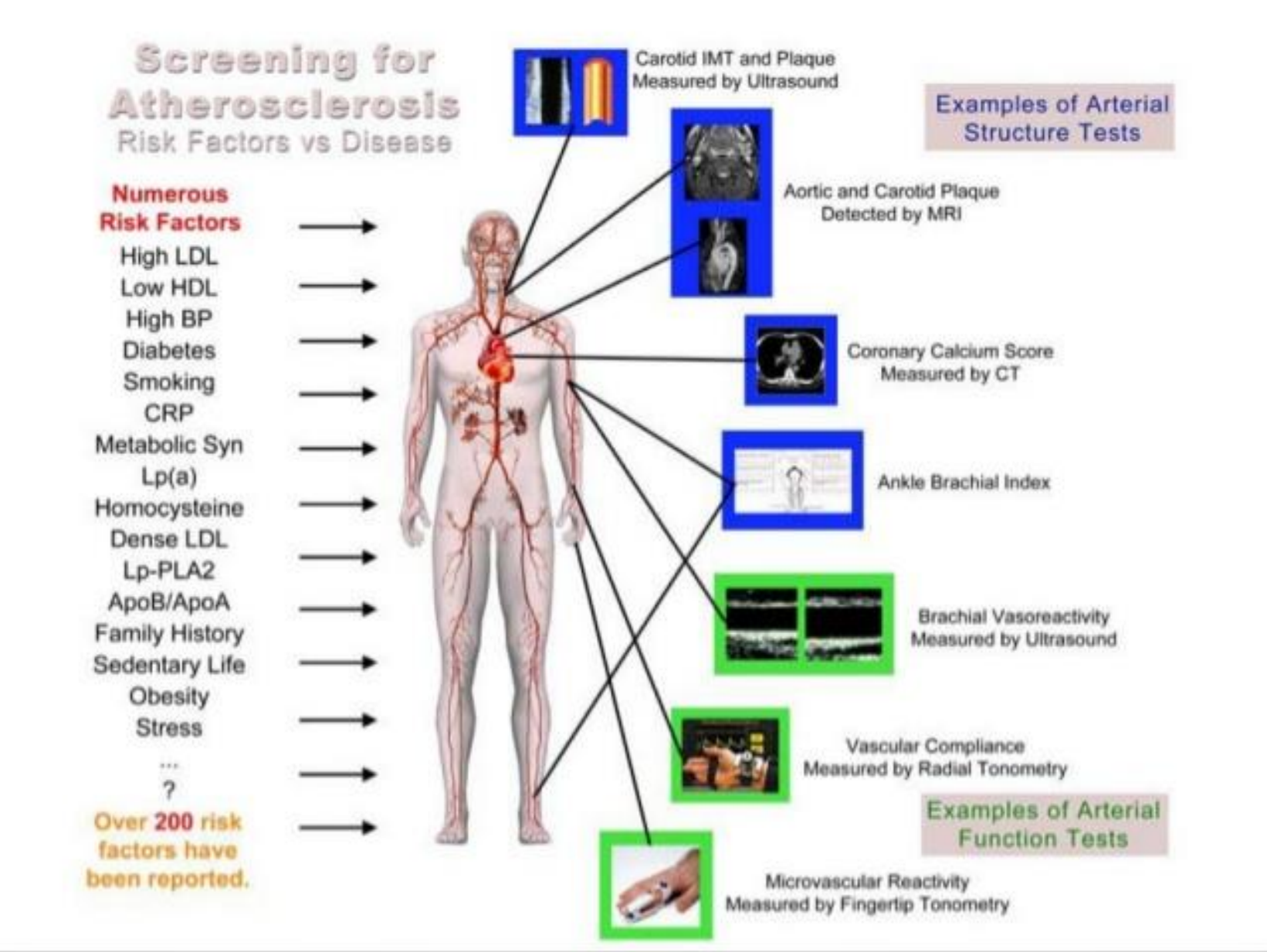
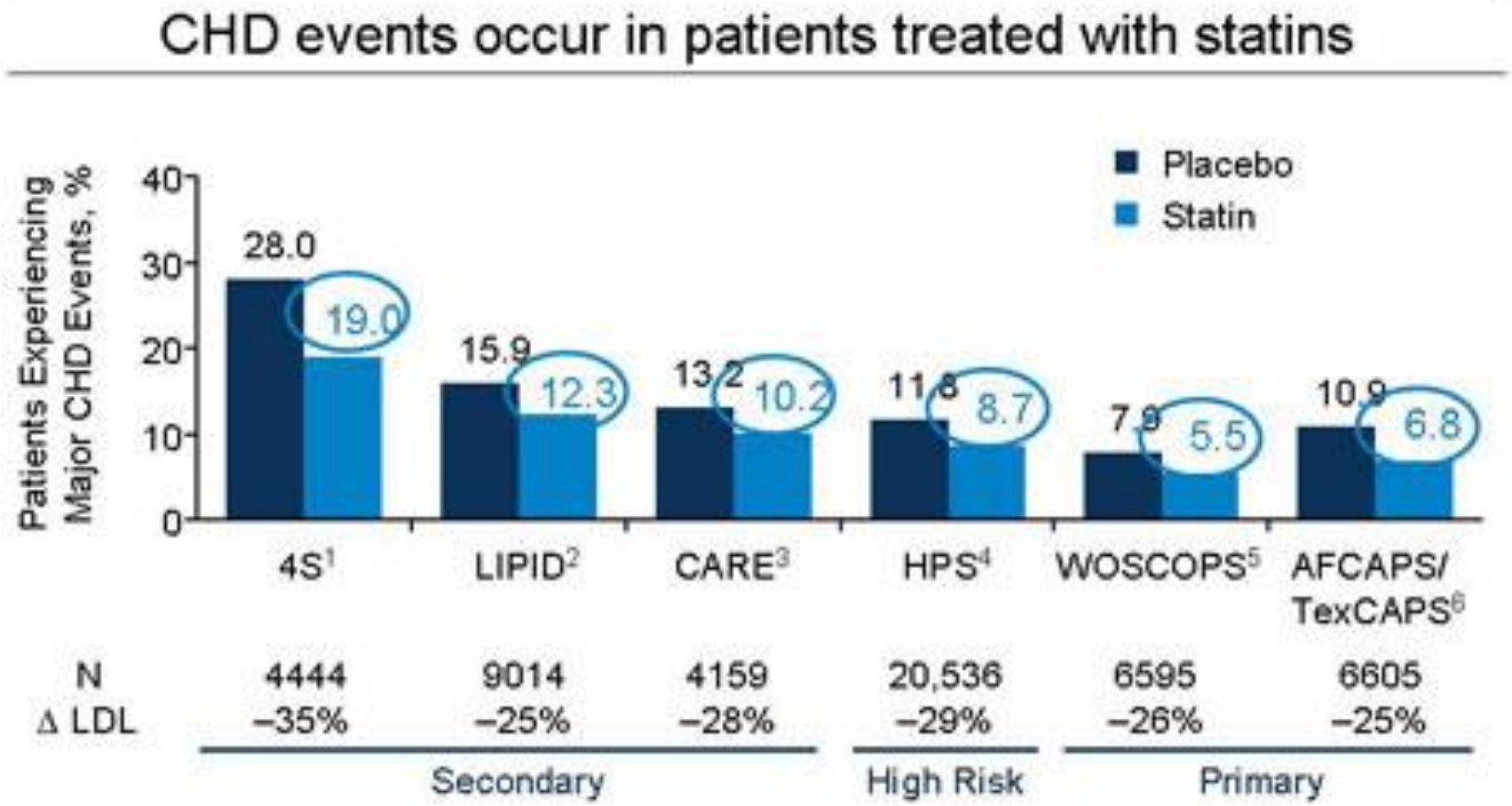

f
Endothelial cells
Monocyte
Macrophage
Cholesterol crystal
Thrombus
Calcified plaque
1
2
3
4
Red blood cell
Smooth muscle cells
Atherosclerotic plaque
Plaque rupture

g
Maternal
(in utero)
Childhood
Adulthood
Old age
• Dyslipidaemia
• Obesity
• Elevated blood pressure
• Active and passive smoking
• Physical inactivity
• Unhealthy diet
h
Primordial prevention
Primary prevention
Secondary prevention
Tertiary prevention

Rối loạn Lipid Máu luôn ưu tiên kiểm soát LDL.C



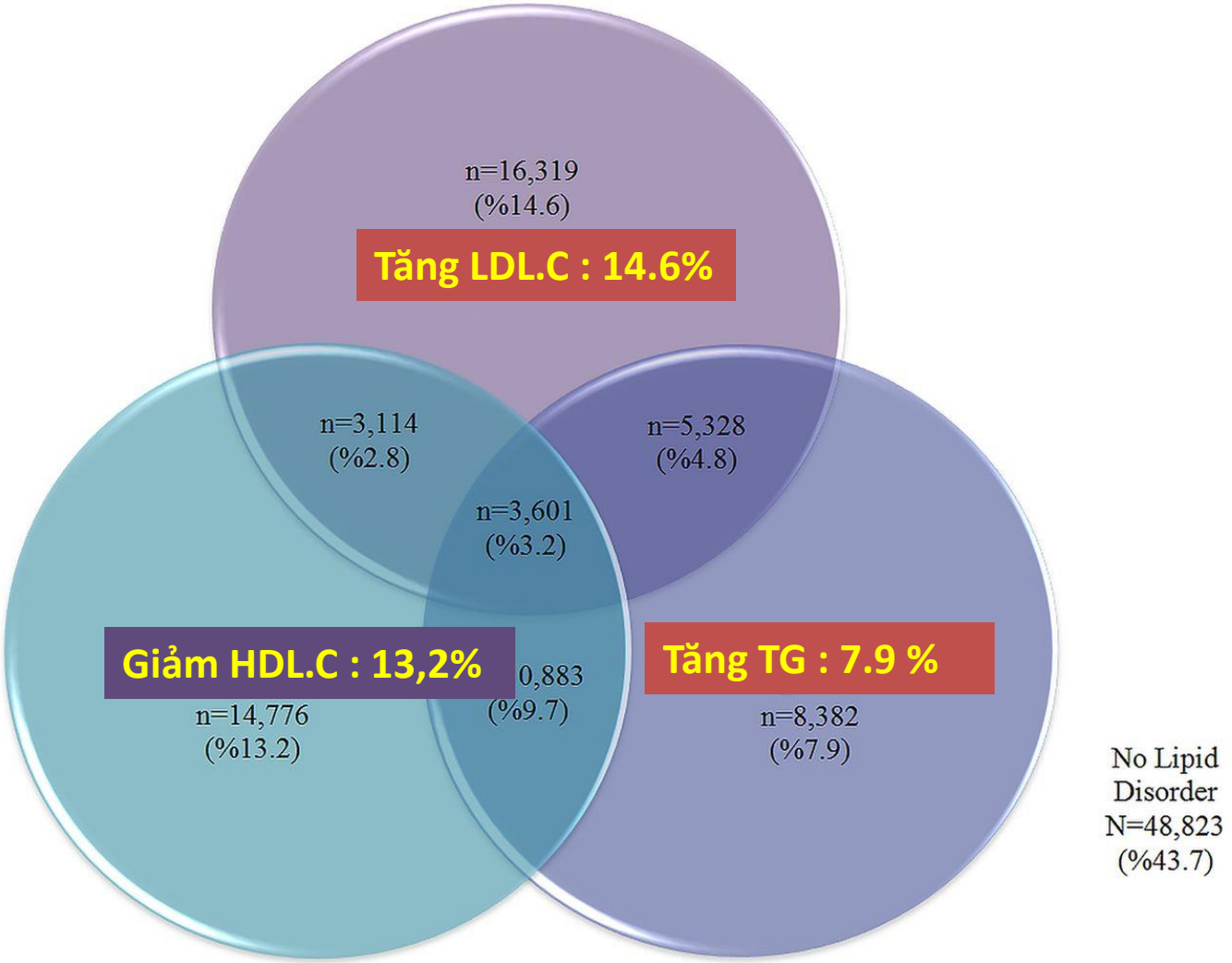
Vì sao biến cố bệnh tim mạch vẫn xãy ra trên người bệnh sử dụng Statin ?



4S, Scandinavian Simvastatin Survival Study; HPS, Heart Protection Study; WOSCOPS, West of Scotland Coronary Prevention Study; AFCAPS/TexCAPS, Air Force/Texas Coronary Atherosclerosis Prevention Study.
[1]4S Group. *Lancet.* 1994;344:1383-1389; [2]LIPID Study Group. *N Engl J Med.* 1998;339:1349-1357; [3]Sacks FM, et al. *N Engl J Med.* 1996;335:1001-1009. [4]HPS Collaborative Group. *Lancet.* 2002;360:7-22; [5]Shepherd J, et al. *N Engl J Med.* 1995;333:1301-1307; [6]Downs JR, et al. *JAMA.* 1998;279:1615-1622.

Medscape

Tỷ lệ RLLP kết hợp (64%) cao hơn đáng kể so với tăng LDL.C đơn độc (14,6%) (khảo sát 48.823 người dân CANADA)



. Asghari S, et al. BMJ Open 2015;5:e007954. doi:10.1136/bmjopen-2015-007954

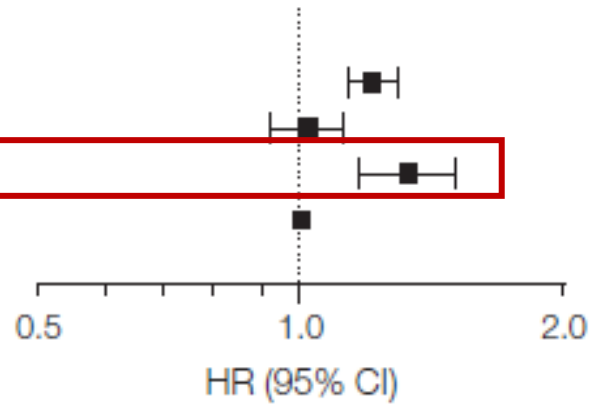
Dù kiểm soát LDL.C nhưng Biến Cố TM vẫn tăng nếu chưa kiểm soát Non-HDL.C

Meta-analysis : 8 RCTs với 38 153 bệnh nhân được chỉ định liệu pháp statin.

- **Mục đích:** Để đánh giá mối tương quan mạnh mẽ của các liên kết của LDL-C, non-HDL-C và apoB với nguy cơ tim mạch ở những bệnh nhân được điều trị bằng liệu pháp statin
- **Thời gian theo dõi** : 1 năm
- **Kết cục chính của meta-analysis:** thời gian xuất hiện biến cố tim mạch mạch chính đầu tiên (nhồi máu cơ tim tử vong /không tử vong, bệnh mạch vành khác tử vong, nhập viện do đau thắt ngực không ổn định, đột quỵ tử vong/không tử vong).

So sánh nguy cơ biến cố tim mạch chính giữa LDL-C và non-HDL-C

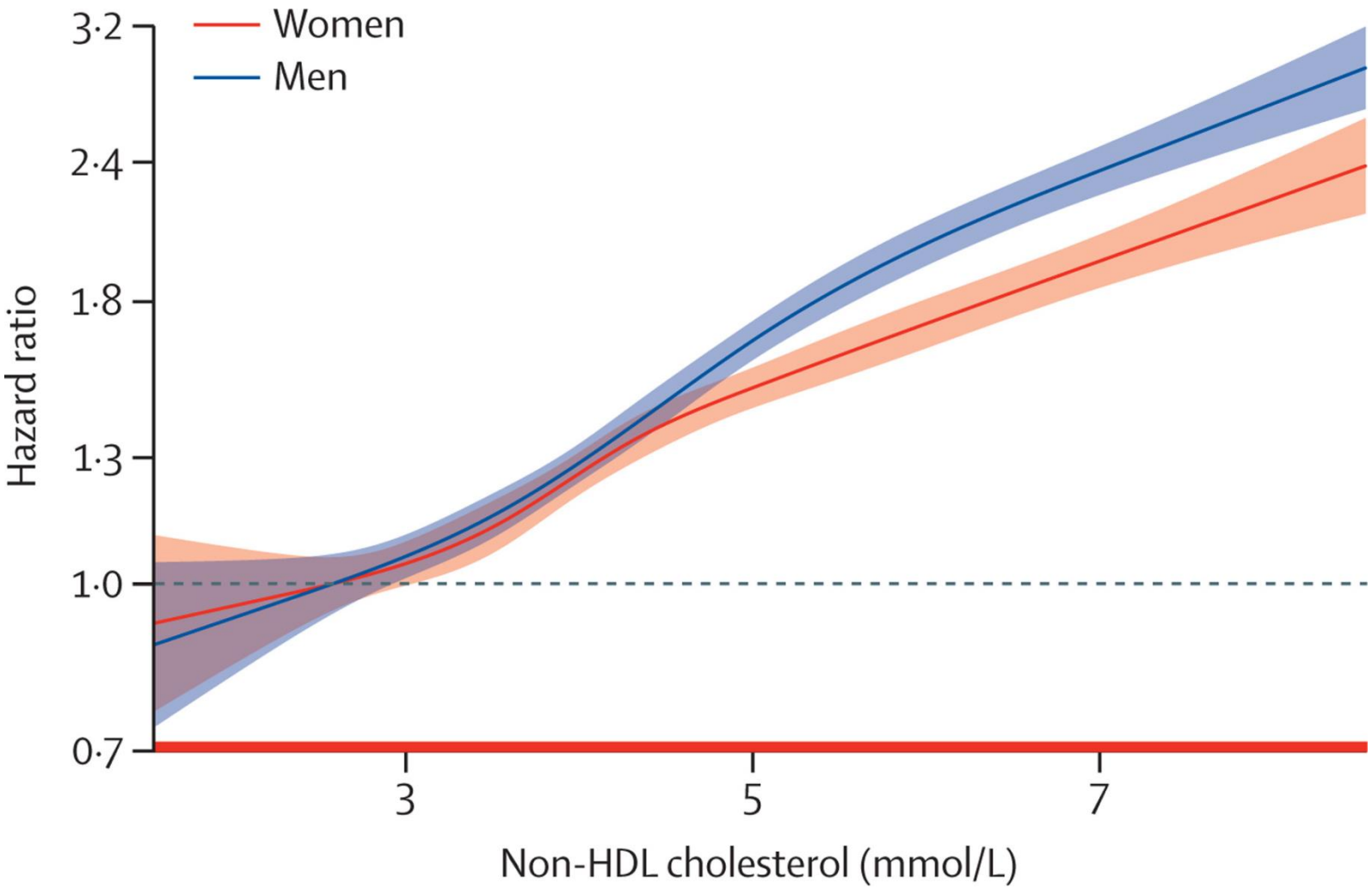
Target Level		No. of Major Cardiovascular Events	Total No. of Participants	HR (95% CI)
LDL-C	Non–HDL-C			
≥100 mg/dL	≥130 mg/dL	1877	10 419	1.21 (1.13-1.29)
≥100 mg/dL	<130 mg/dL	467	2873	1.02 (0.92-1.12)
<100 mg/dL	≥130 mg/dL	283	1435	1.32 (1.17-1.50)
<100 mg/dL	<130 mg/dL	2760	23 426	1.00 [Reference]



HRs đã được điều chỉnh về giới tính, tuổi, hút thuốc, đái tháo đường, tăng huyết áp và thử nghiệm.

Non-HDL-C = TC – HDL-C
1 mmol/L= 38.7 mg/dL
(nồng độ Cholesterol)

JAMA. 2012;307(12):1302-1309



524. 444 individuals in the 44 cohorts from 19 countries (Europe,Australia, and North America) . **Non-HDL.C** concentrations in blood are strongly associated with **long-term risk of ASCVD**.

Fabian J Brunner et al. Lancet 2019; 394: 2173–83

2. Bệnh Tim Mạch và RLLP máu người Châu Á

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JACC: ASIA VOL. 1, NO. 1, 2021

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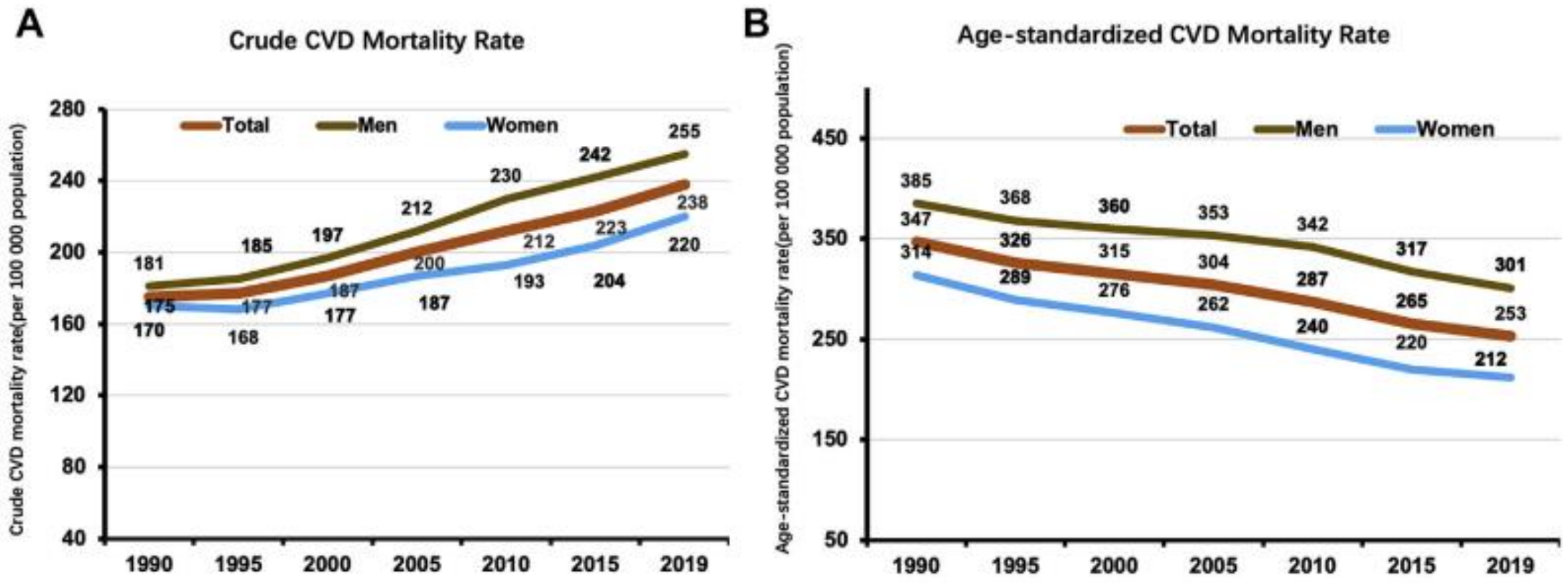
STATE-OF-THE-ART REVIEW

Epidemiological Features of Cardiovascular Disease in Asia

Dong Zhao, MD, PhD

FIGURE 1 **CVD Mortality From 1990 to 2019 in Asia**



(A) Crude CVD mortality (per 100,000 population) from 1990 to 2019 in men **(green line)**, women **(blue line)**, and the total population **(red line)** in Asia. **(B)** Age-standardized CVD mortality (per 100,000 population) from 1990 to 2019 in men **(green)**, women **(blue)**, and total population **(red)** in Asia. All data were obtained from the open database of the Global Burden of Disease Study in the Global Health Data Exchange (2). CVD = cardiovascular diseases.

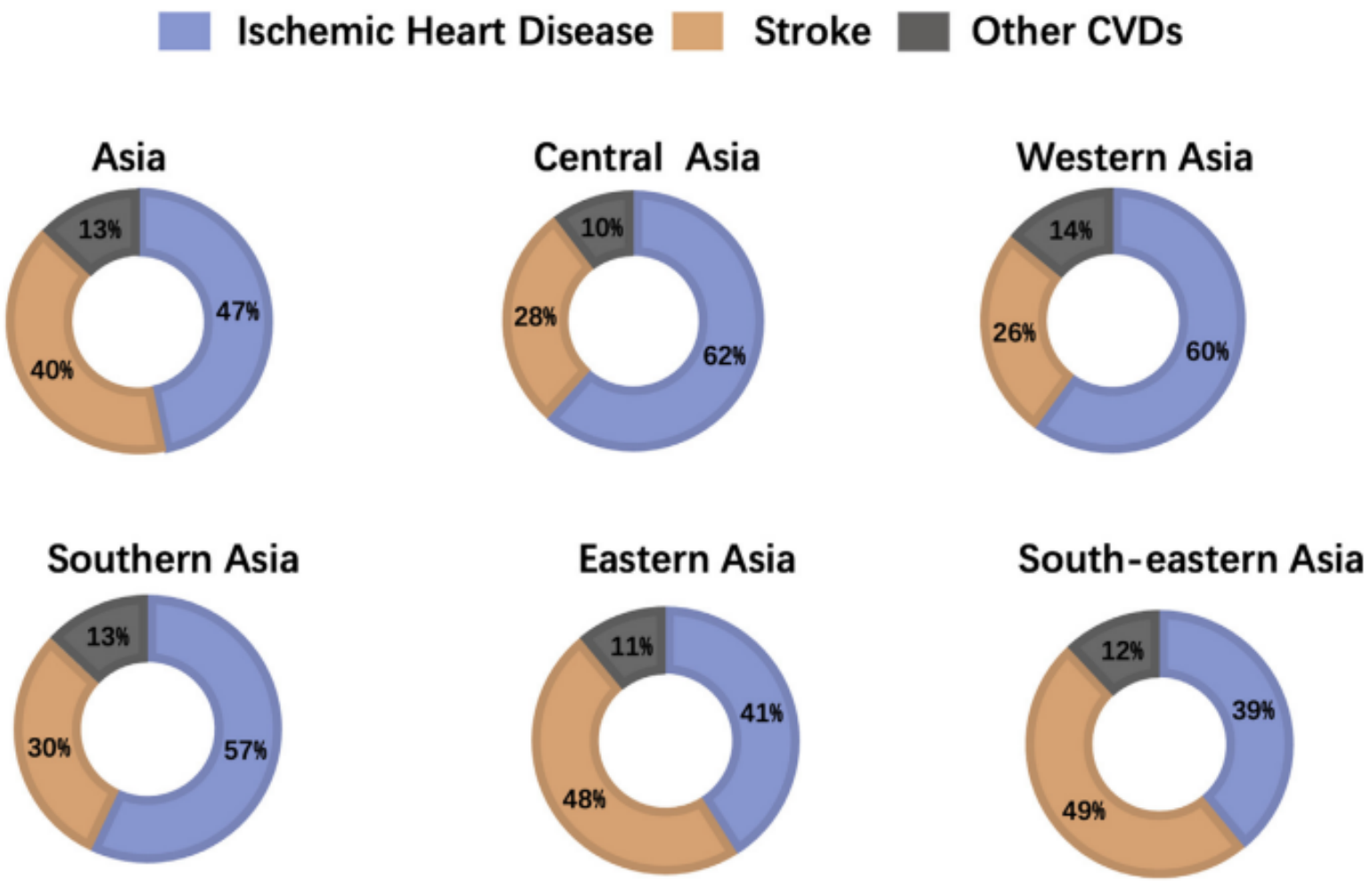
CENTRAL ILLUSTRATION Crude Cardiovascular Disease Mortality Rates in Asian Countries, by Region

Zhao, D. JACC: Asia. 2021;1(1):1-13.

Crude CVD mortality rates (per 100,000 population) in
(A) Western Asian countries,
(B) Central Asian countries,
(C) Southern Asian countries,
(D) Eastern Asian countries, and
(E) Southeastern Asian countries.
All data (2019) were obtained from the open database of the Global Burden of Disease Study in the Global Health Data Exchange.

Việt nam là một trong ba nước vùng Đông Nam Á có tỷ lệ cao về tử vong do bệnh tim mạch

FIGURE 2 The Proportions of IHD and Stroke Deaths in Total CVD Deaths



Vùng Đông Nam Á có tỷ lệ đột quỵ cao hơn so với bệnh mạch vành

Theo NCD-RisC, 10 nước xếp hạng có nồng độ Non HDL-C cao nhất vào năm 1980 (Bỉ, Phần Lan, Greenland…) có giảm Non-HDL-C từ 1980 đến 2018.

Nồng độ TC tăng cao nhất ở các nước Đông Á (Trung Quốc) và các nước Đông Nam Á (Indonesia, Thái Lan, Malaysia và Campuchia). Trung Quốc là quốc gia có nồng độ cholesterol trung bình thấp nhất vào năm 1980 nhưng đã đạt hoặc vượt qua nồng độ Non-HDL-C của nhiều nước phương Tây vào năm 2018.

Báo cáo NCD-RisCreport ghi nhận tử vong do BTMXV do nồng độ cao Non-HDL-C ở các nước phương Tây giảm từ 950.000 (năm 1990) xuống còn 480.000 (năm 2017), trong khi tử vong do BTMXV khắp châu Á đã tăng lên trong giai đoạn này, tăng gấp ba ở Đông Á (từ 250.000 lên 860.000) và tăng gấp đôi ở Đông Nam Á (từ 110.000 lên 310.000).

Trong số 3,9 triệu ca tử vong trên thế giới do Bệnh Mạch Vành và Đột Quỵ liên quan nồng độ cao Non HDL.C trong năm 2017, một nửa trong số này xảy ra ở các nước Đông, Đông Nam và Nam Châu Á.

Sự khác biệt Rối loạn lipid máu người cao tuổi và quần thể chung người Châu Á

International Journal of Gerontology 13 (2019) 100–102
https://doi.org/10.6890/IJGE.201903_13(1).0021

International Journal of Gerontology

journal homepage: http://www.sgecm.org.tw/ijge/

Short Communication

Epidemiology of Dyslipidemias in Asia Pacific Countries: Differences between General Population and the Elderly

Chao-Feng Lin [a, b], Shih-Chieh Chien [c], Hung-I Yeh [a, b *]

The prevalence of dyslipidemia in general population and in the elderly across 10 Asia Pacific countries.

Lipid component	Cut-off value to define "abnormal" levels	Country	Prevalence	
			General population	Elderly
TC	≥240 mg/dL	China	12.2% (Males 12.1%, Females 12.3%)	23.9% (Males 12.9%, Females 28.0%)
		Japan	14.5% (Males 10.3%, Females 17.4%)	13.1% (Males 7.7%, Females 17.2%)
		Korea	14.5% (Males 13.8%, Females 15.1%)	27.8% (60–69 years of age) (Males 17.9%, Females 37.0%) 22.7% (≥70 years of age) (Males 12.5%, Females 29.5%)
		Malaysia	44.9% (Males 46.7%, Females 43.5%)	59.7% (Males 56.5%, Females 64.4%)
		Philippines	46.9%	58.8% (60–69 years of age) 54.6% (≥70 years of age)
		Singapore	17.4%	23.3%
		Thailand	20.4%	Not available*
		Taiwan	11.2% (Males 11.3%, Females 11.1%)	16.7% (Males 12.0%, Females 20.8%)
	≥210 mg/dL	Australia	7.1%	25.2%
	Medicated	New Zealand	11.5%	33.0%
LDL-C	≥160 mg/dL	China	17.9% (Males 18.6%, Females 17.5%)	33.2% (Males 25.1%, Females 37.3%)
		Japan	9.0% (Males 7.4%, Females 10.1%)	7.6% (Males 4.9%, Females 9.6%)
		Korea	14.8% (Males 14.3%, Females 15.2%)	26.9% (60–69 years of age) (Males 17.7%, Females 35.3%) 23.4% (≥70 years of age) (Males 12.4%, Females 30.5%)
		Philippines	47.2%	59.8% (60–69 years of age) 57.5% (≥70 years of age)
		Singapore	15.2%	20.3%
		Taiwan	7.8% (Males 8.6%, Females 7.2%)	8.5% (Males 8.0%, Females 8.9%)
	According to ATP III. High LDL-C are defined as follows: LDL-C ≥100 mg/dL if having prior CHD or CHD equivalent or having 10-year CHD risk >20%; LDL-C ≥ 130 mg/dL if having ≥2 risk factors (RF) and/or 10-year CHD risk 10% to 20%; and LDL-C ≥160 mg/dL if having 0-1 RF.	Thailand	29.6%	Not available*

The prevalence of dyslipidemia in general population and in the elderly across 10 Asia Pacific countries.

Lipid component	Cut-off value to define "abnormal" levels	Country	Prevalence	
			General population	Elderly
HDL-C	<40 mg/dL	China	12.0% (Males 19.5%, Females 6.7%)	12.5% (Males 15.3%, Females 8.0%)
		Japan	6.2% (Males 11.3%, Females 2.6%)	7.4% (Males 12.5%, Females 3.4%)
		Korea	41.2% (Males 34.1%, Females 48.2%)	51.4% (60–69 years of age) (Males 41.8%, Females 60.2%) 57.3% (≥70 years of age) (Males 39.5%, Females 69.1%)
		Singapore	8.1%	6.8%
		Taiwan	10.1% (Males 15.7%, Females 5.3%)	12.5% (Males 18.3%, Females 7.4%)
	<40 mg/dL for males; <50 mg/dL for females	Philippines	71.3%	74.4% (60–69 years of age) 76.5% (≥70 years of age)
		Thailand	47.1%	~50%
TG	≥200 mg/dL	China	15.1% (Males 20.6%, Females 11.3%)	16.0% (Males 12.2%, Females 19.2%)
		Japan	19.4% (Males 26.3%, Females 14.7%)	21.8% (Males 25.9%, Females 18.4%)
		Korea	28.7% (Males 38.0%, Females 19.5%)	34.0% (60–69 years of age) (Males 38.9%, Females 29.5%) 37.1% (≥70 years of age) (Males 32.4%, Females 40.2%)
		Taiwan	15.3% (Males 19.5%, Females 11.6%)	19.0% (Males 16.6%, Females 21.2%)
	≥150 mg/dL	Philippines	38.6%	42.8% (60–69 years of age) 34.4% (≥70 years of age)
		Thailand	38.6%	~45%

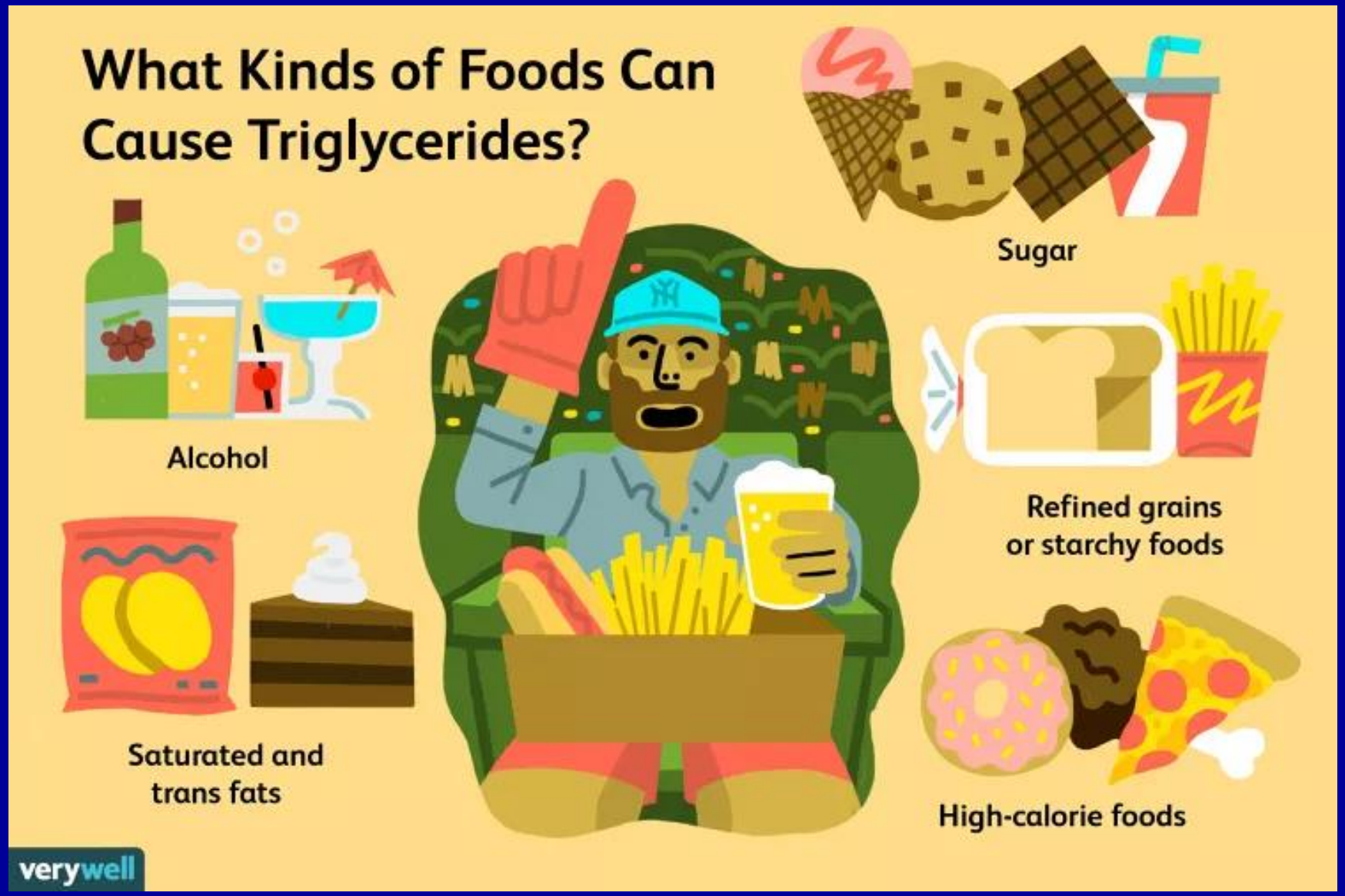
ATP III, The Third Adult Treatment olesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; TG, t

* All lipid levels, except HDL-C, were hi

RỐI LOẠN LIPID MÁU NGƯỜI CHÂU Á

cần chú ý RLLP dạng kết hợp

Chế độ ăn uống liên quan rối loạn lipid máu



Triglycerides

Causes of high triglycerides in the general population

- Overweight and obesity
- Physical inactivity
- Cigarette smoking
- Excess alcohol intake
- Very high carbohydrate diets (>60% of energy)
- Other disease (diabetes, renal failure, nephrosis)
- Drugs: steroids, protease inhibitors, estrogen, etc
- Genetic factors

NCEP JAMA 2001;285:2486 Final Report Circulation 2002;106:3143-3421

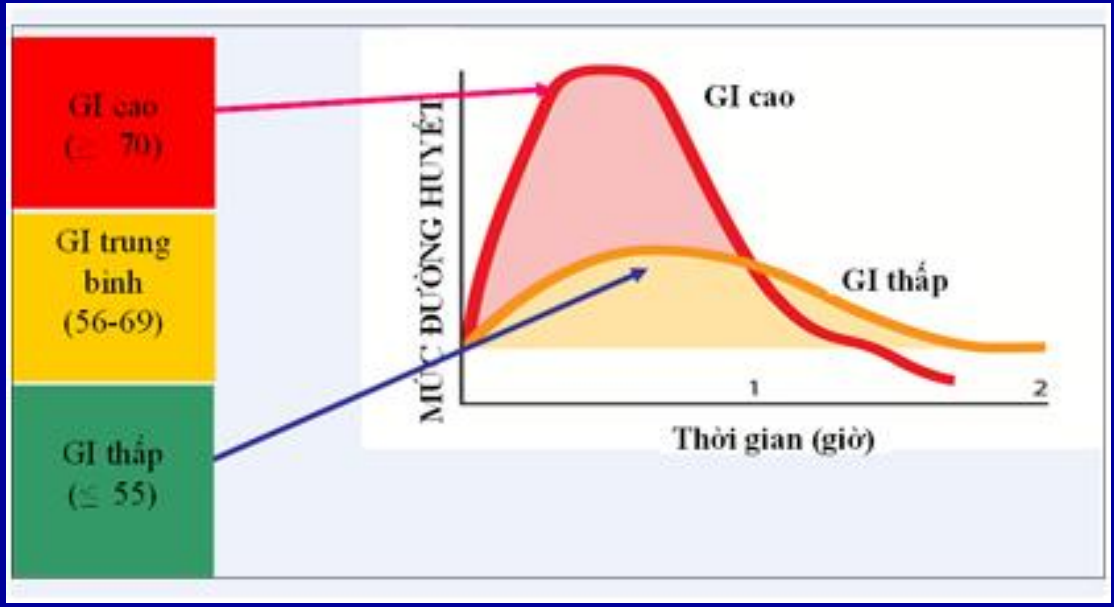
DIETARY CARBOHYDRATES (QUANTITY AND QUALITY) AND DIABETES RISK

Carbohydrate-rich cereals account for 60% of daily caloric intake among Asian nations, which is one of the highest levels in the world.[20] Owing to rapid industrialization and to the green

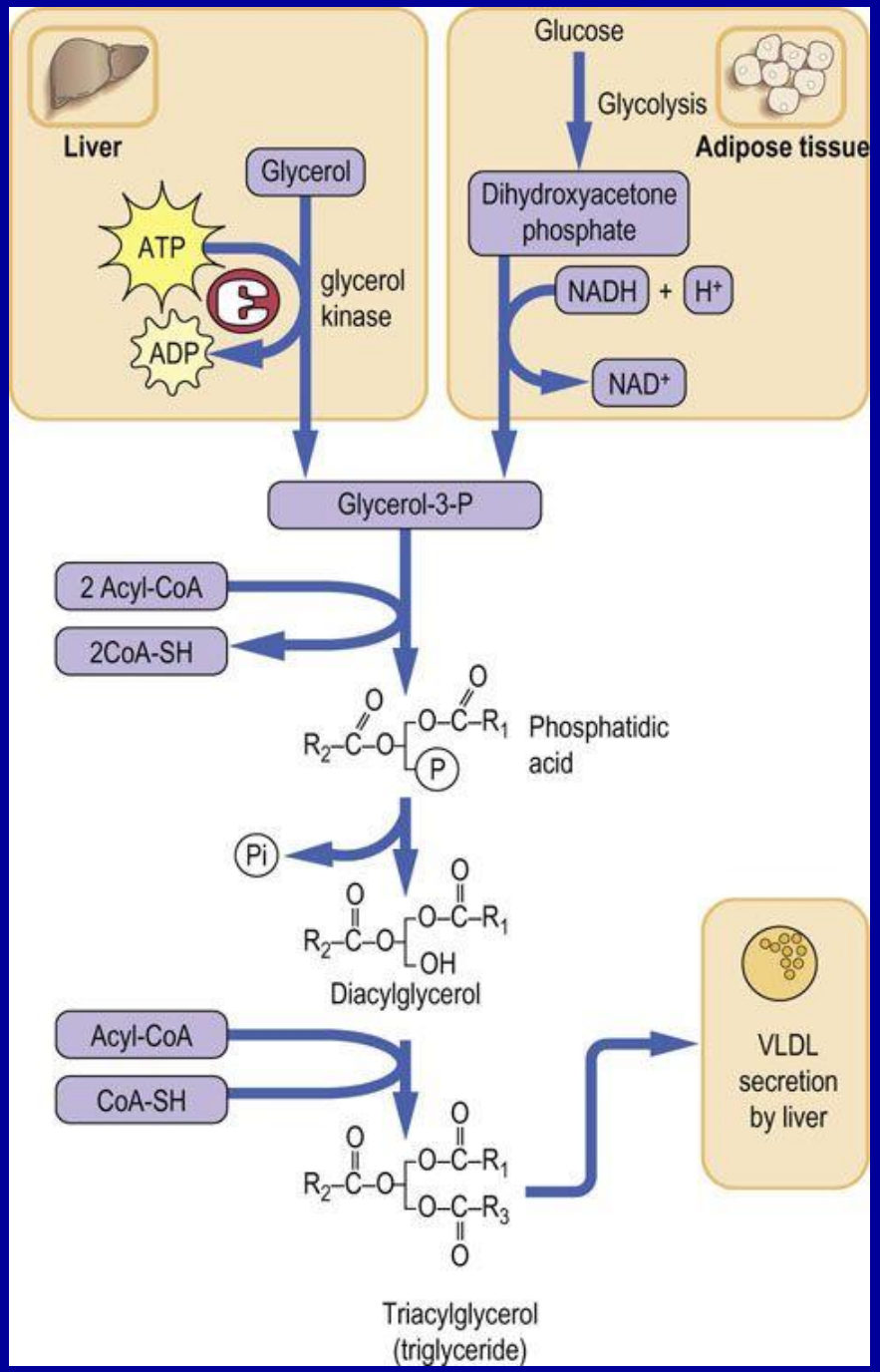
Chế độ ăn nhiều CHO (≥60% tổng năng lượng)
là nguyên nhân tăng Triglycerides

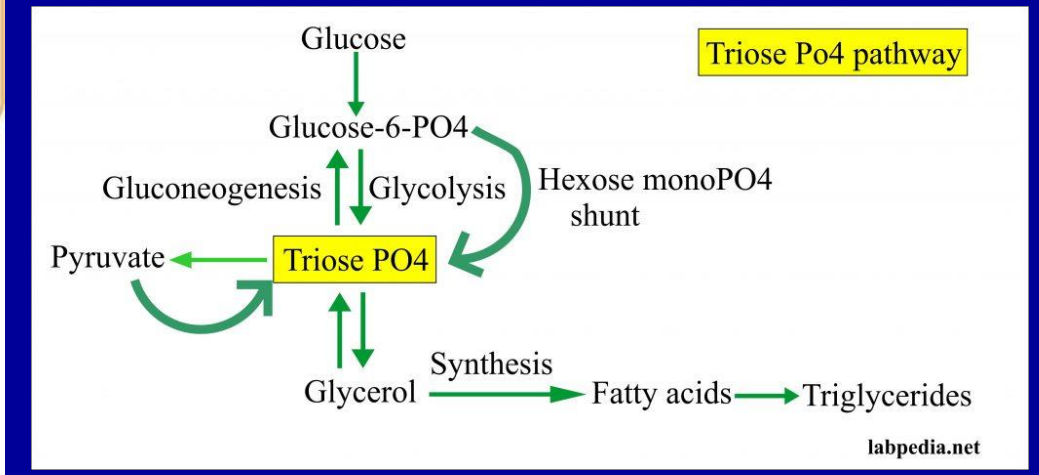
Cơm trắng : CHO bị cắt ra nhanh chóng, vì thế có chỉ số đường huyết cao, gây đường huyết tăng cao và nhanh sau các bữa ăn.

Cơm gạo lứt : CHO được cắt ra chậm, vì thế chỉ số đường huyết thấp sẽ phóng thích đường glucose vào máu chậm hơn.



Cơ chế tăng TG do glucose máu





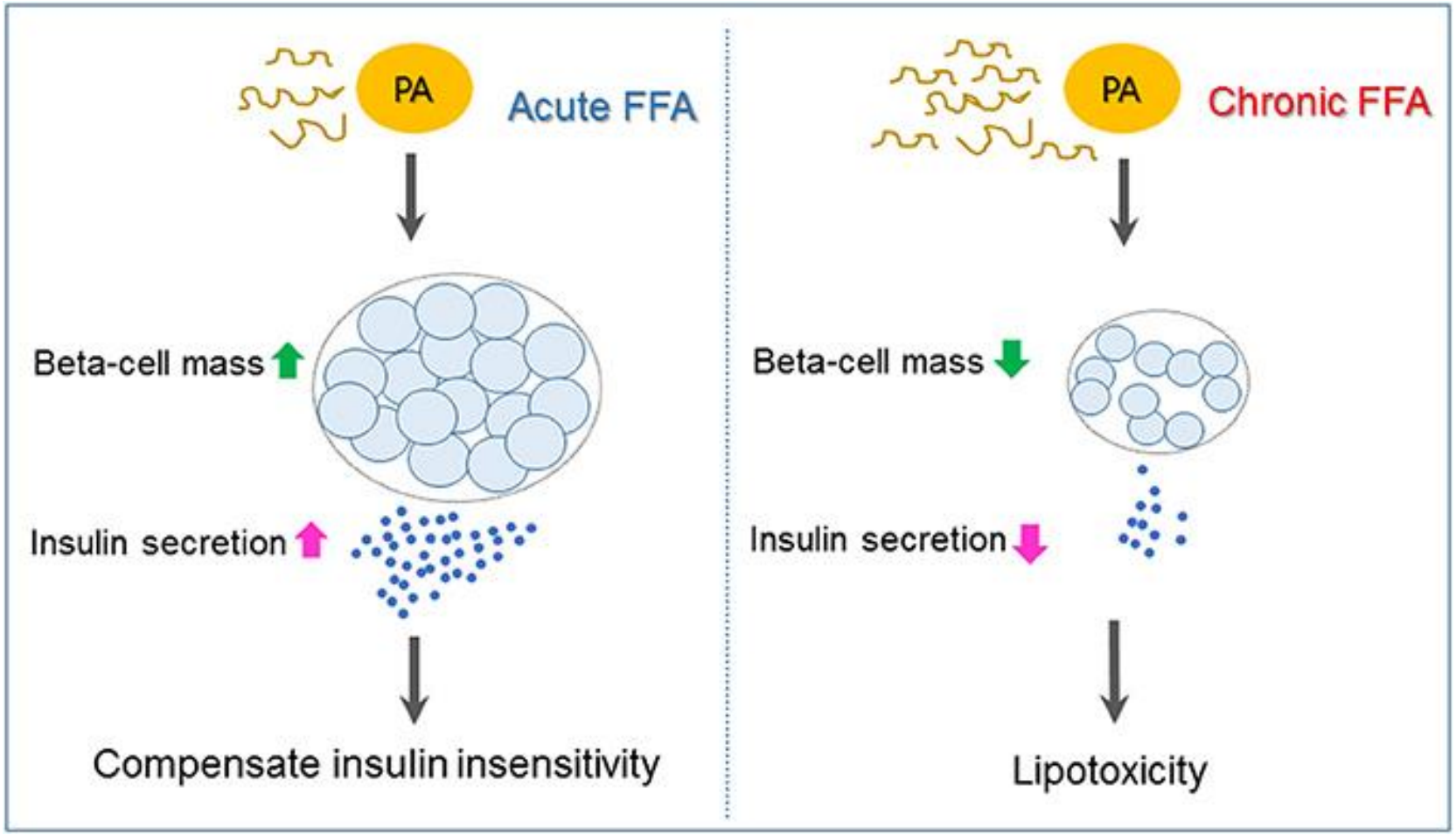
Cơm (CHO)

↓

Tăng Glucose sau ăn

↓

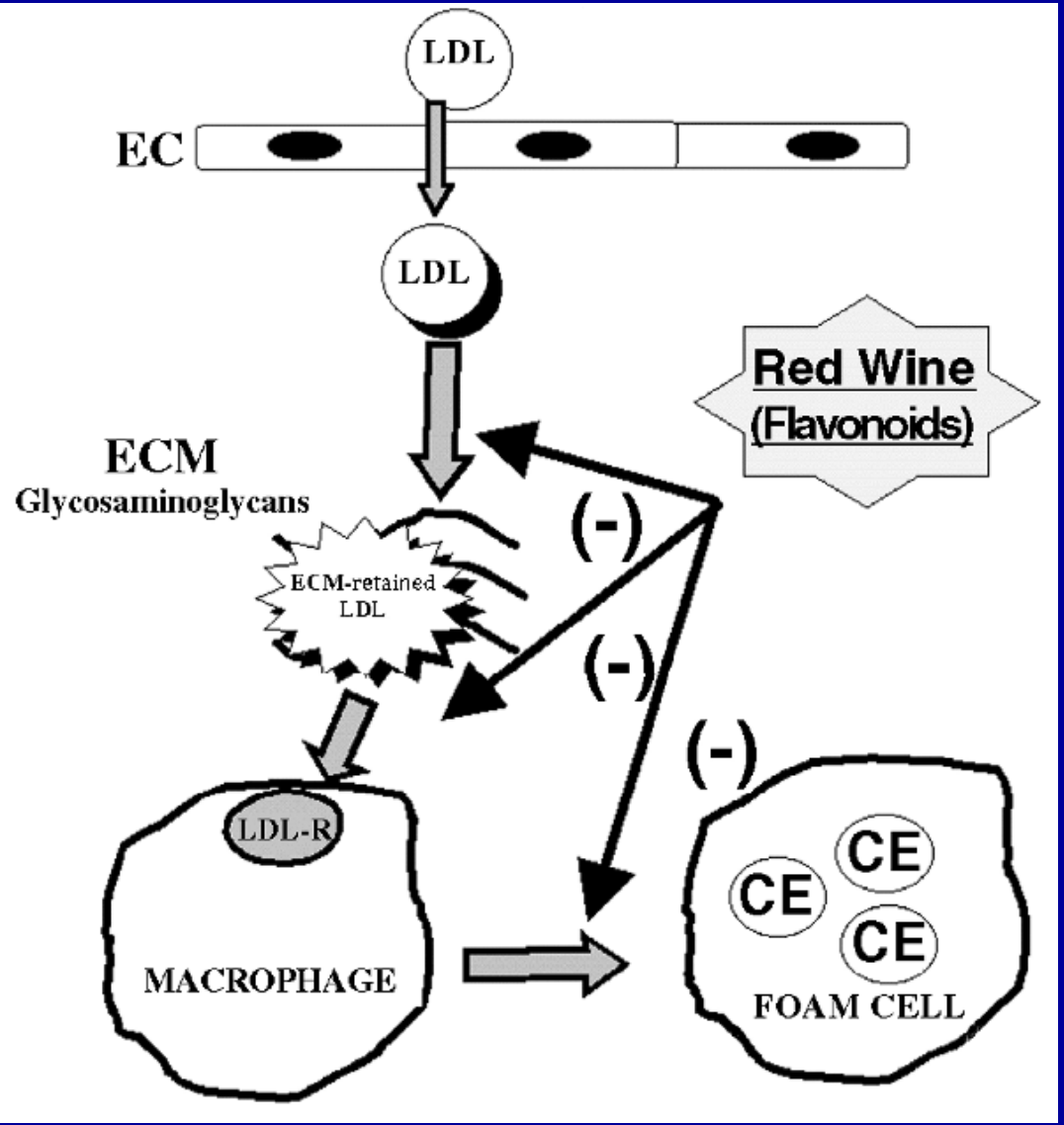
Tăng TG

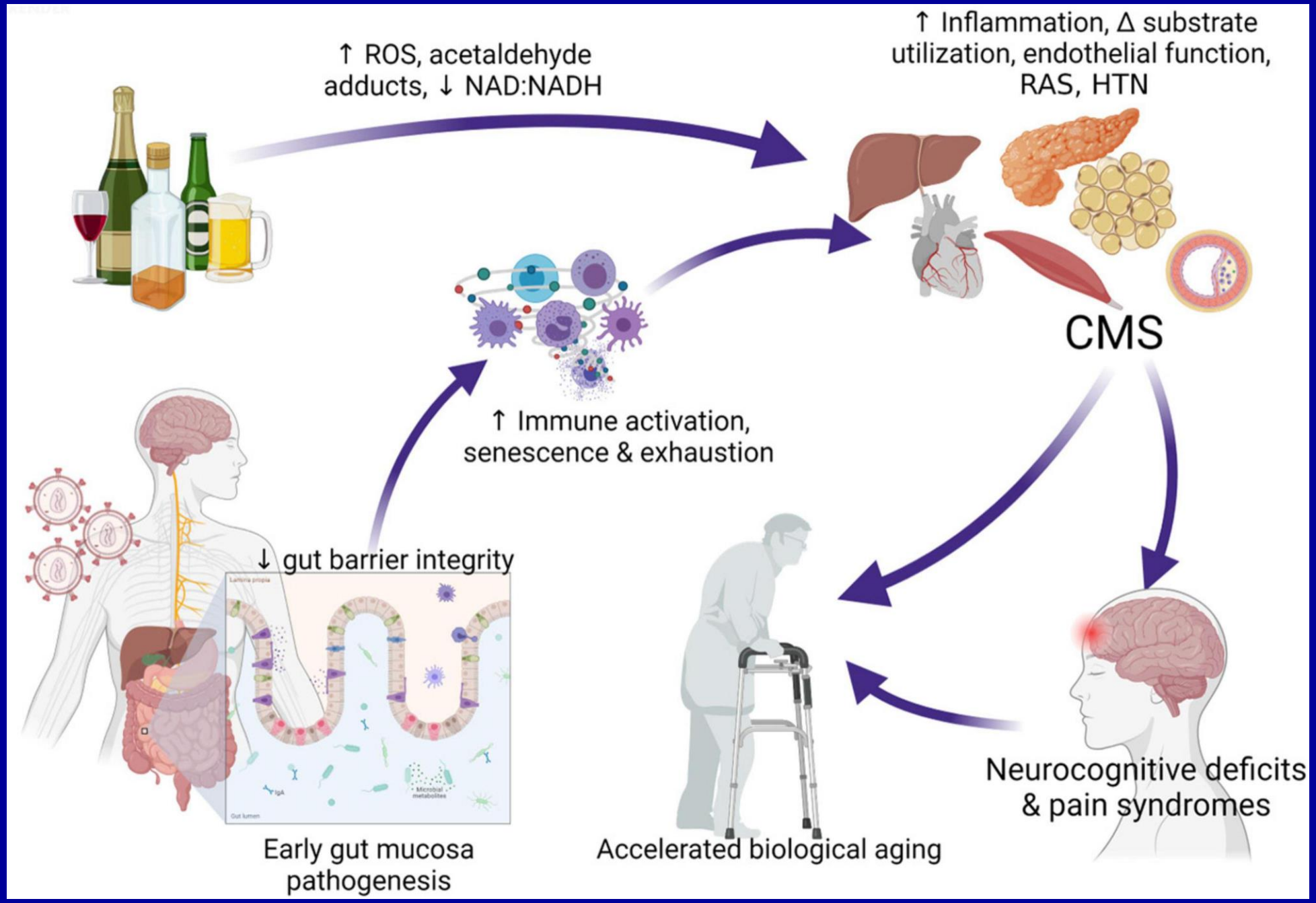


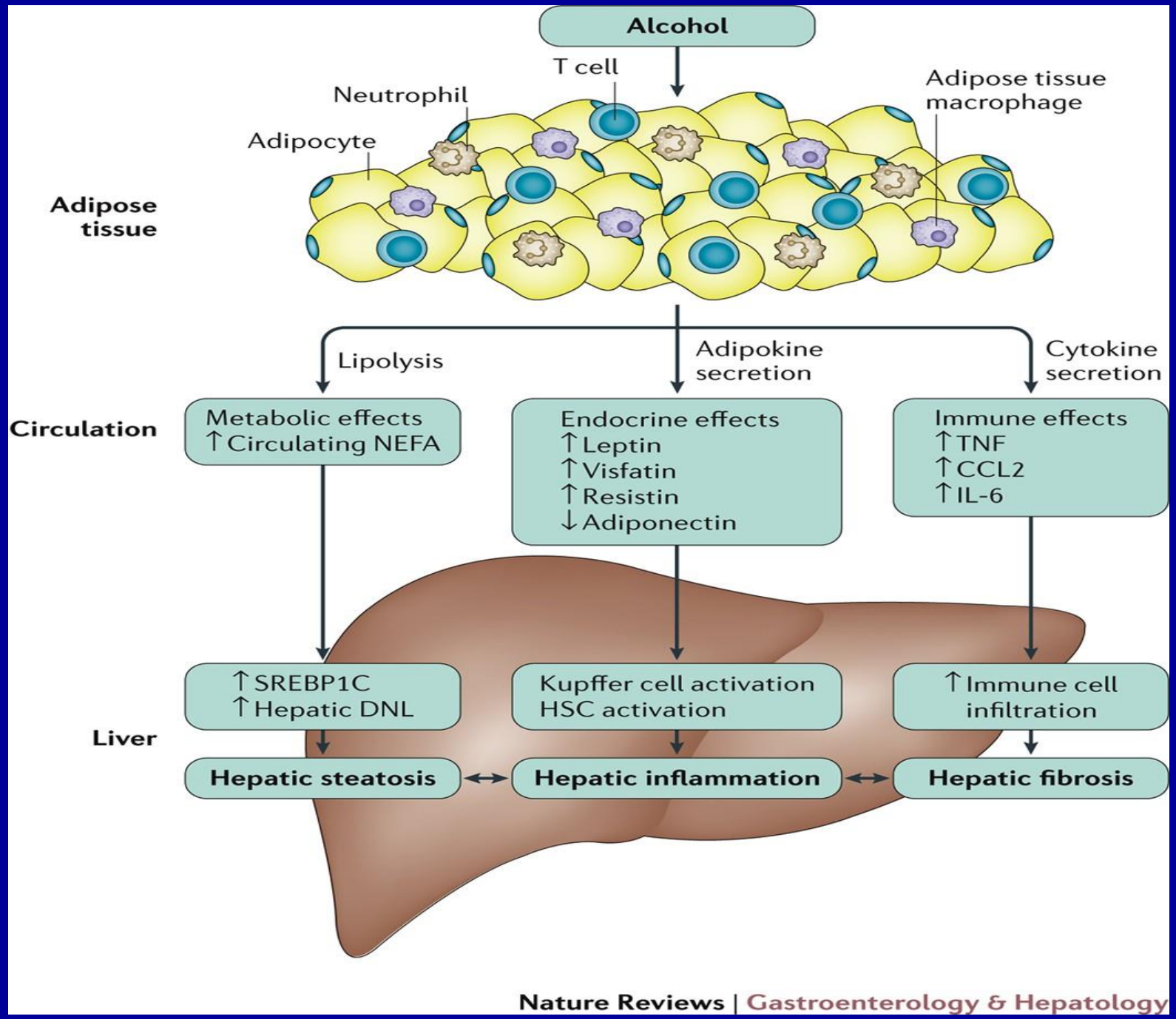
Cơ chế suy TB beta tụy do nhiễm độc lipid.

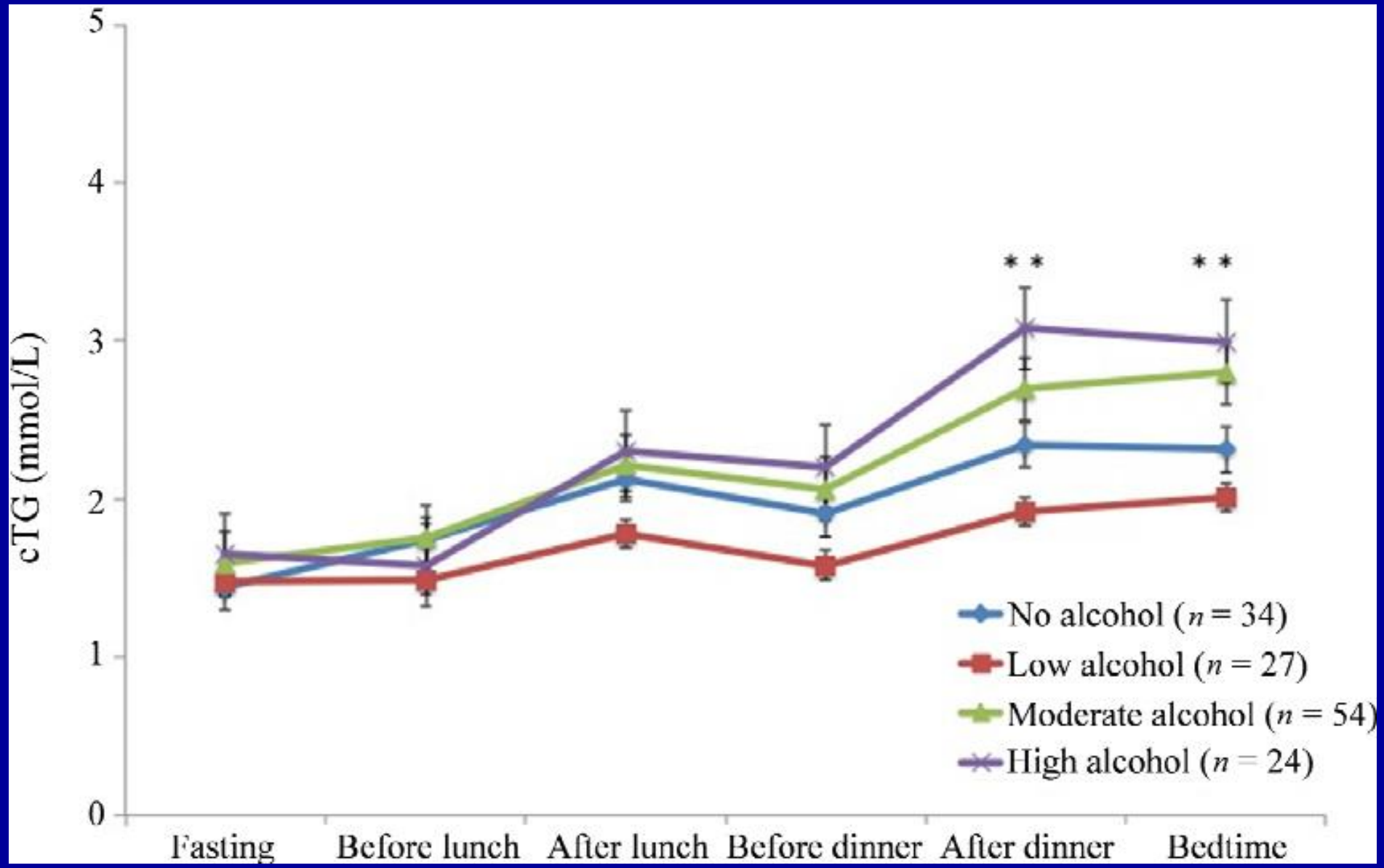
Kháng insulin xảy ra, **axit béo tự do (FFA)** như **palmiate (PA)** tăng sẽ làm **gia tăng khối lượng TB beta** và **tăng bài tiết insulin** để bù vào tình trạng giảm nhạy cảm với insulin. Sự tăng FFA huyết tương kéo dài gây nhiễm độc lipid gây RL chức năng TB beta và tăng quá trình **Apoptosis**, hậu quả gây ra **ĐTĐ típ 2.**

Rượu Bia


LDL
EC
LDL
Red Wine
(Flavonoids)
ECM
Glycosaminoglycans
ECM-retained
LDL
(-)
(-)
(-)
LDL-R
MACROPHAGE
CE
CE
CE
FOAM CELL


↑ ROS, acetaldehyde adducts, ↓ NAD:NADH
↑ Inflammation, Δ substrate utilization, endothelial function, RAS, HTN
CMS
↑ Immune activation, senescence & exhaustion
↓ gut barrier integrity
Early gut mucosa pathogenesis
Accelerated biological aging
Neurocognitive deficits & pain syndromes

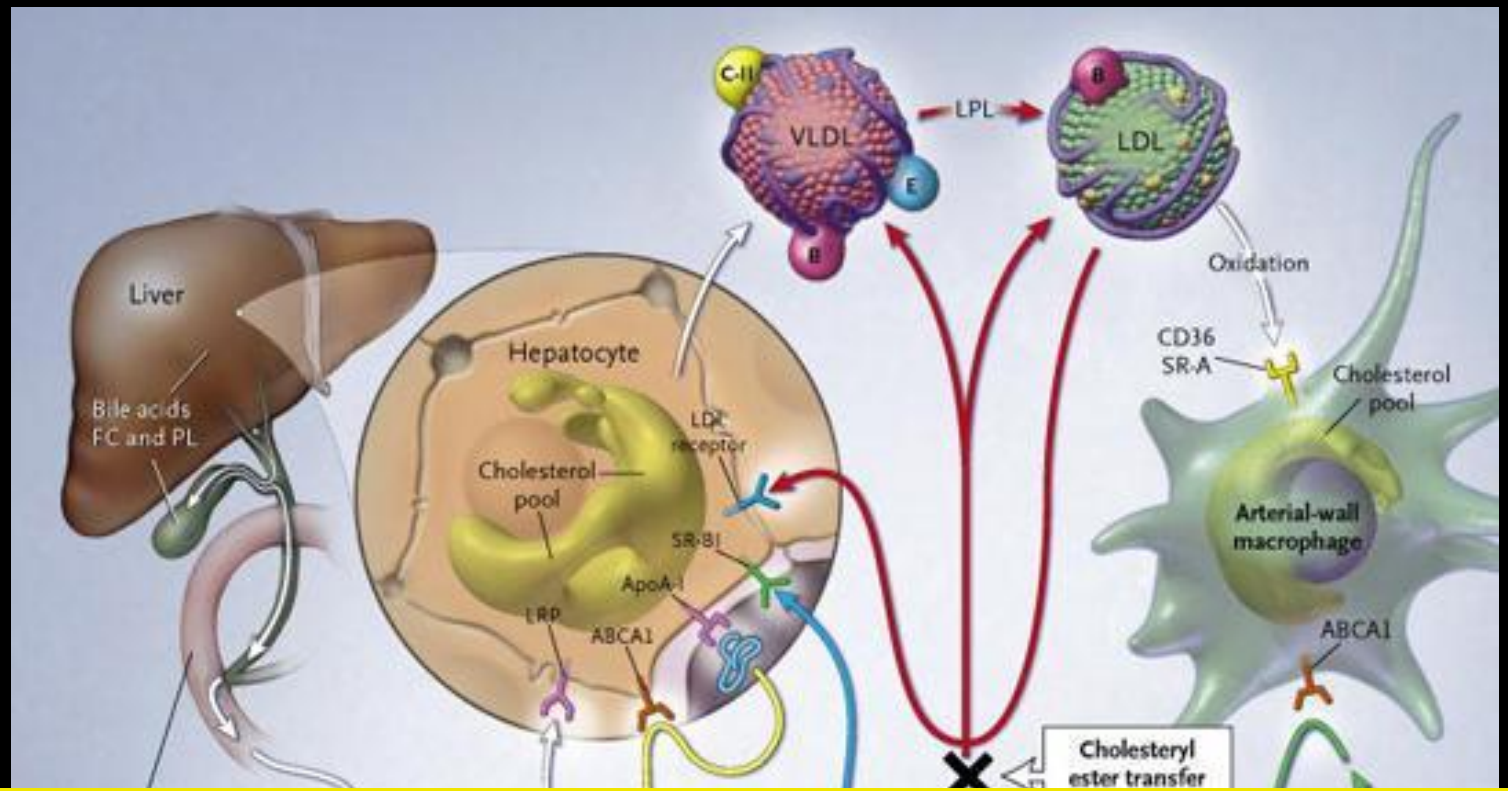

Alcohol
T cell
Neutrophil
Adipocyte
Adipose tissue macrophage
Adipose tissue
Lipolysis
Adipokine secretion
Cytokine secretion
Circulation
Metabolic effects
↑Circulating NEFA
Endocrine effects
↑Leptin
↑Visfatin
↑Resistin
↓Adiponectin
Immune effects
↑TNF
↑CCL2
↑IL-6
Liver
↑SREBP1C
↑Hepatic DNL
Kupffer cell activation
HSC activation
↑Immune cell infiltration
Hepatic steatosis
Hepatic inflammation
Hepatic fibrosis
Nature Reviews | Gastroenterology & Hepatology



Diurnal capillary triglyceride patterns according to alcohol intake. Data are shown as mean ± SEM.
** Significant differences in cTG for a specific time-point (p for trend <0.001).

Tăng Non HDL.C và Rối Loạn lipid máu kết hợp Chế độ ăn uống (nhiều tinh bột, rượu bia..) là những quan tâm cho người Châu Á.

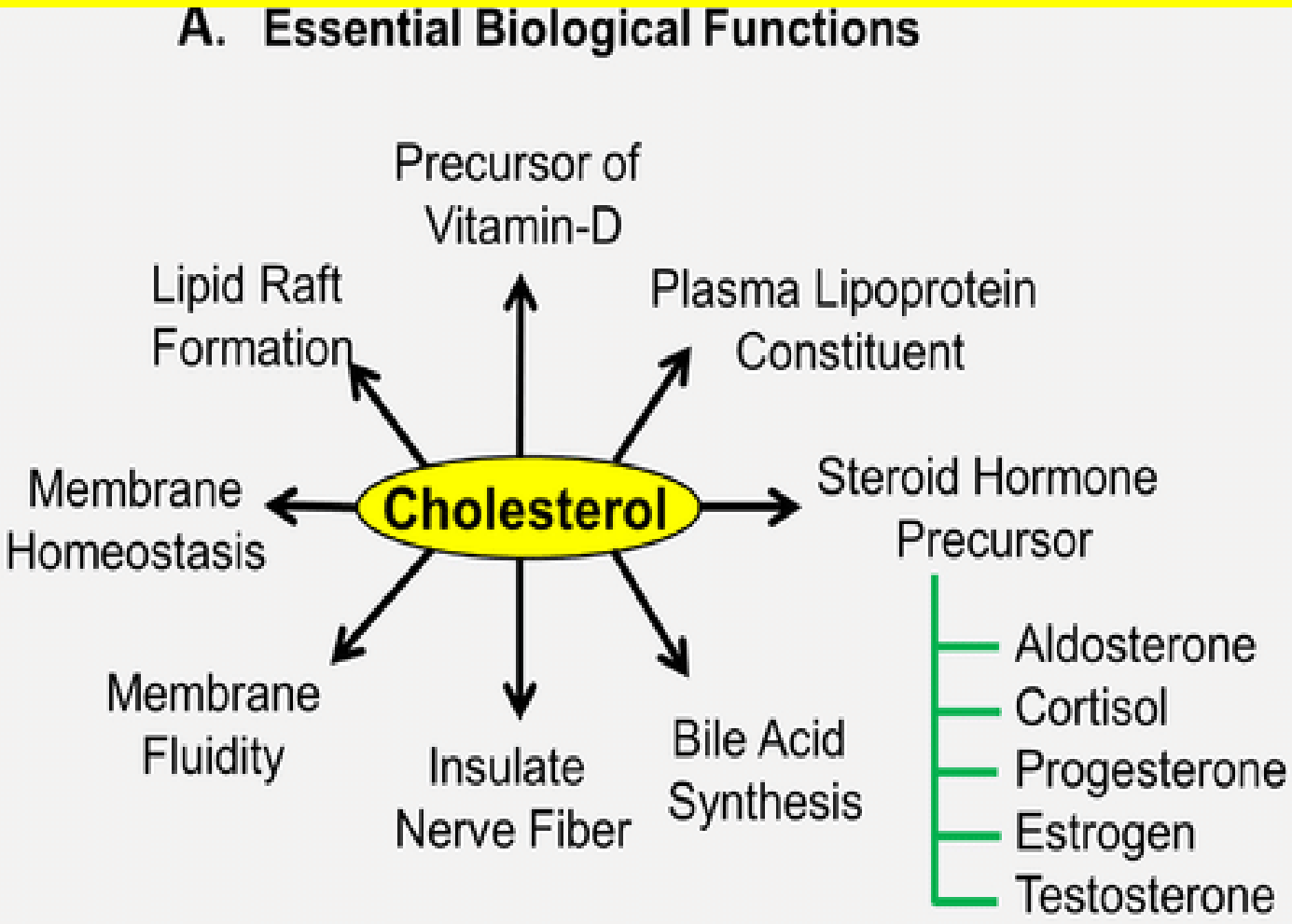
3. Vì sao tăng Non-HDL.C và RLLP máu kết hợp có nguy cơ cao bệnh Tim Mạch Xơ Vữa ?

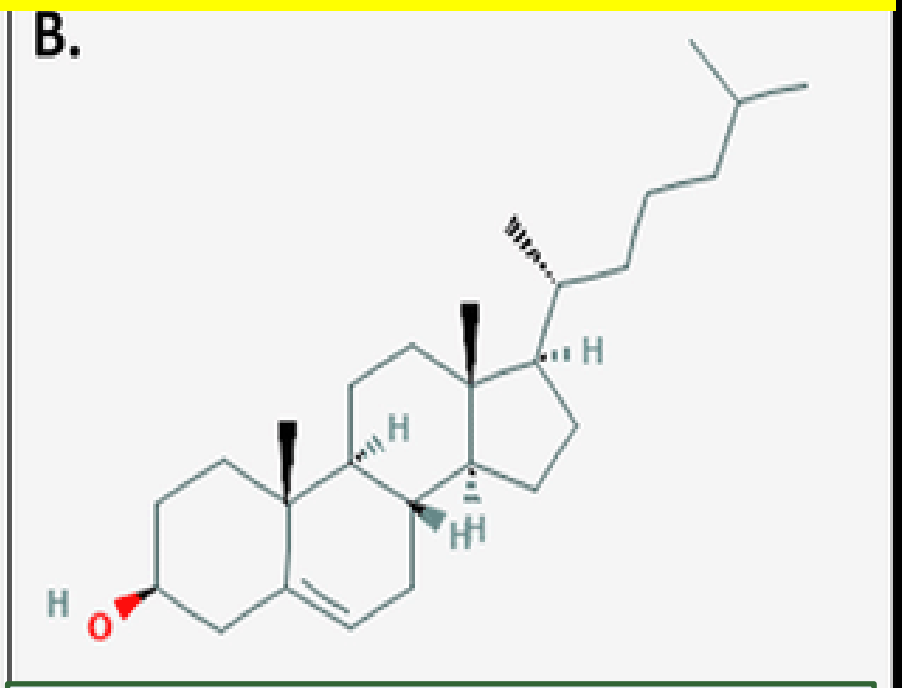


Lipid máu là nguồn năng lượng cung cấp khoảng 30% tổng năng lượng cơ thể và lưu hành dưới 2 dạng chính gồm

- Lipid đơn :cholesterol, acid béo bảo hoà, đơn và đa không bảo hoà
- Lipid phức :cholesterol ester, triglycerides và phospholipid
- Cholesterol và Triglyceride (TG) máu là thành phần chính.

Chức năng Cholesterol

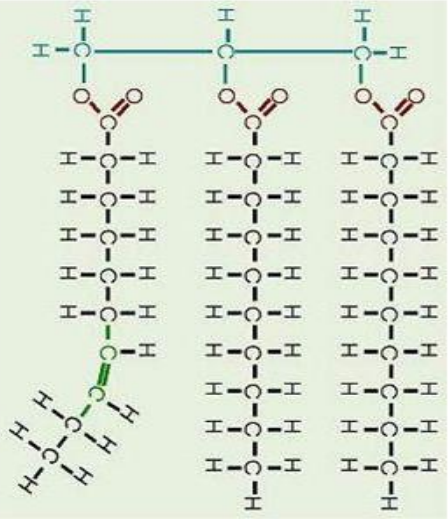


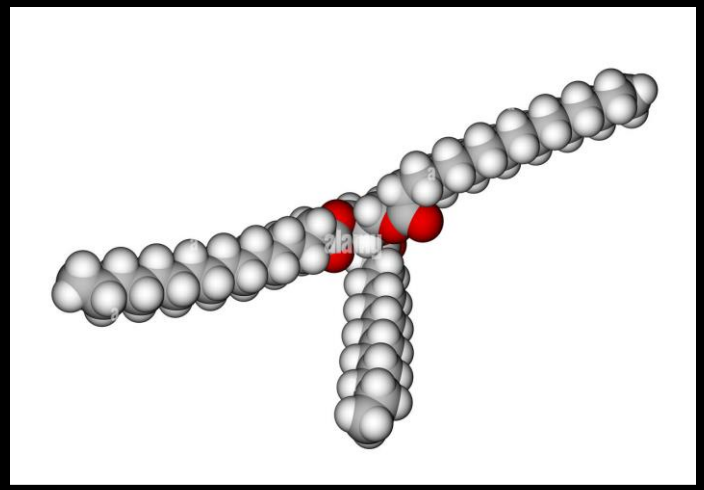


Cholesterol là thành phần cơ bản màng tế bào, tổng hợp acid mật tại gan và hormone steroid nội tiết sinh dục. Ester hoá của cholesterol với FA tạo phân tử (cholesterol ester) hoà tan trong mỡ .

Chức năng Triglycerides

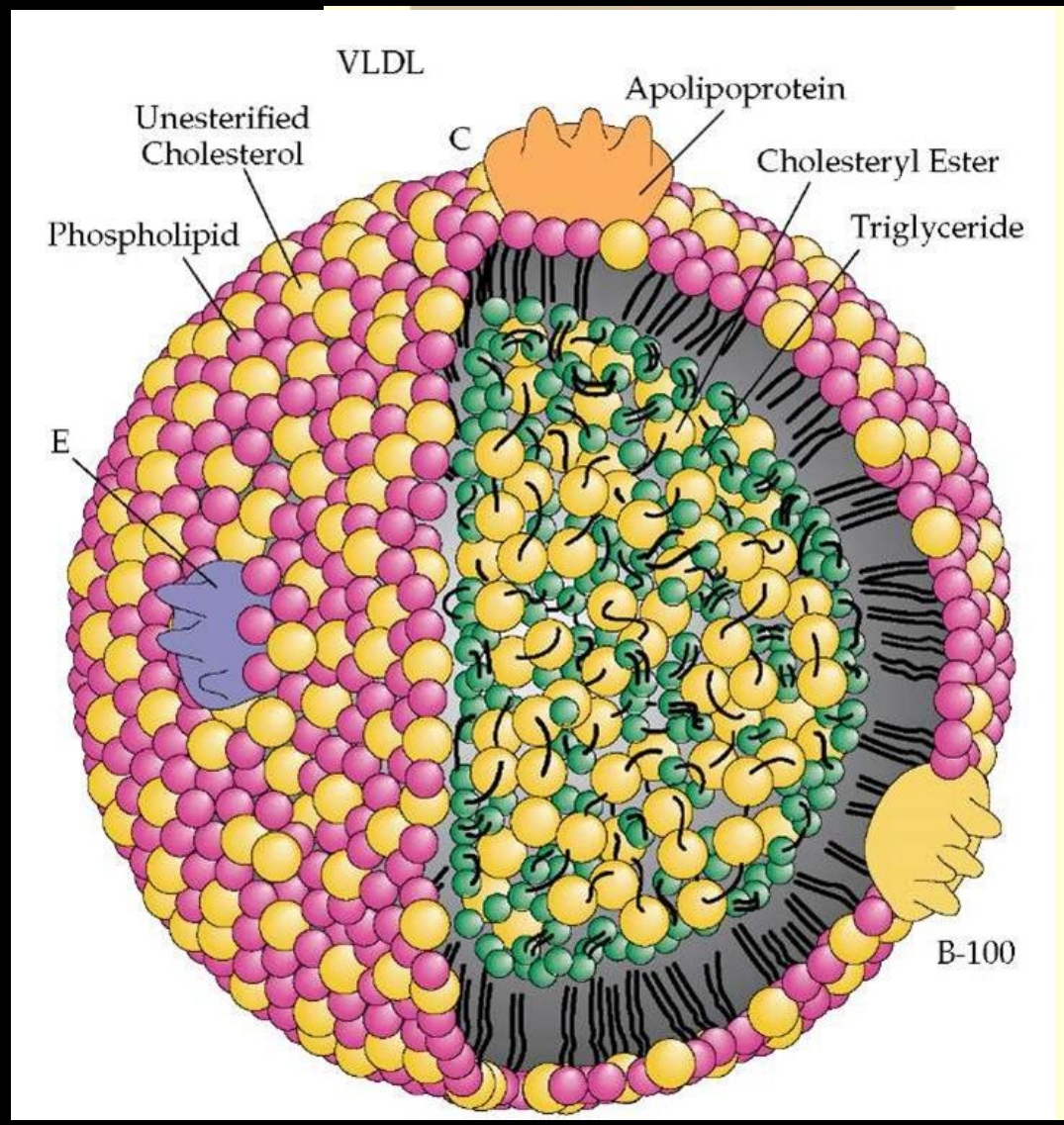
1. Stores and transfers genetic information
2. Long term energy storage, as well as insulation & protection
3. Control the rate of reactions
4. Help to fight disease

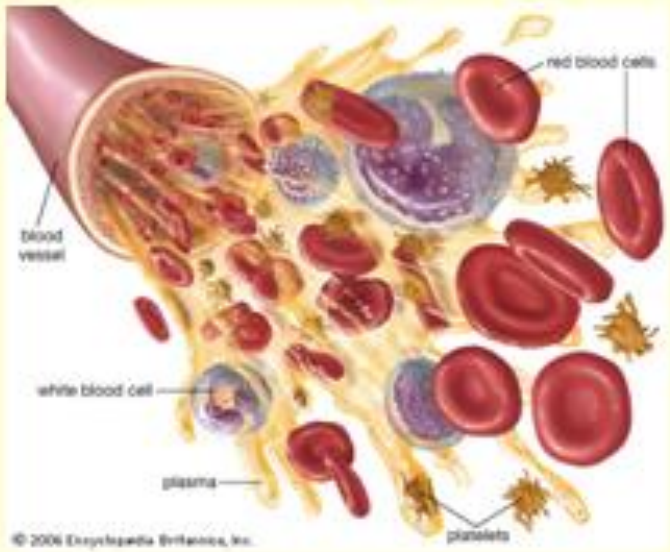




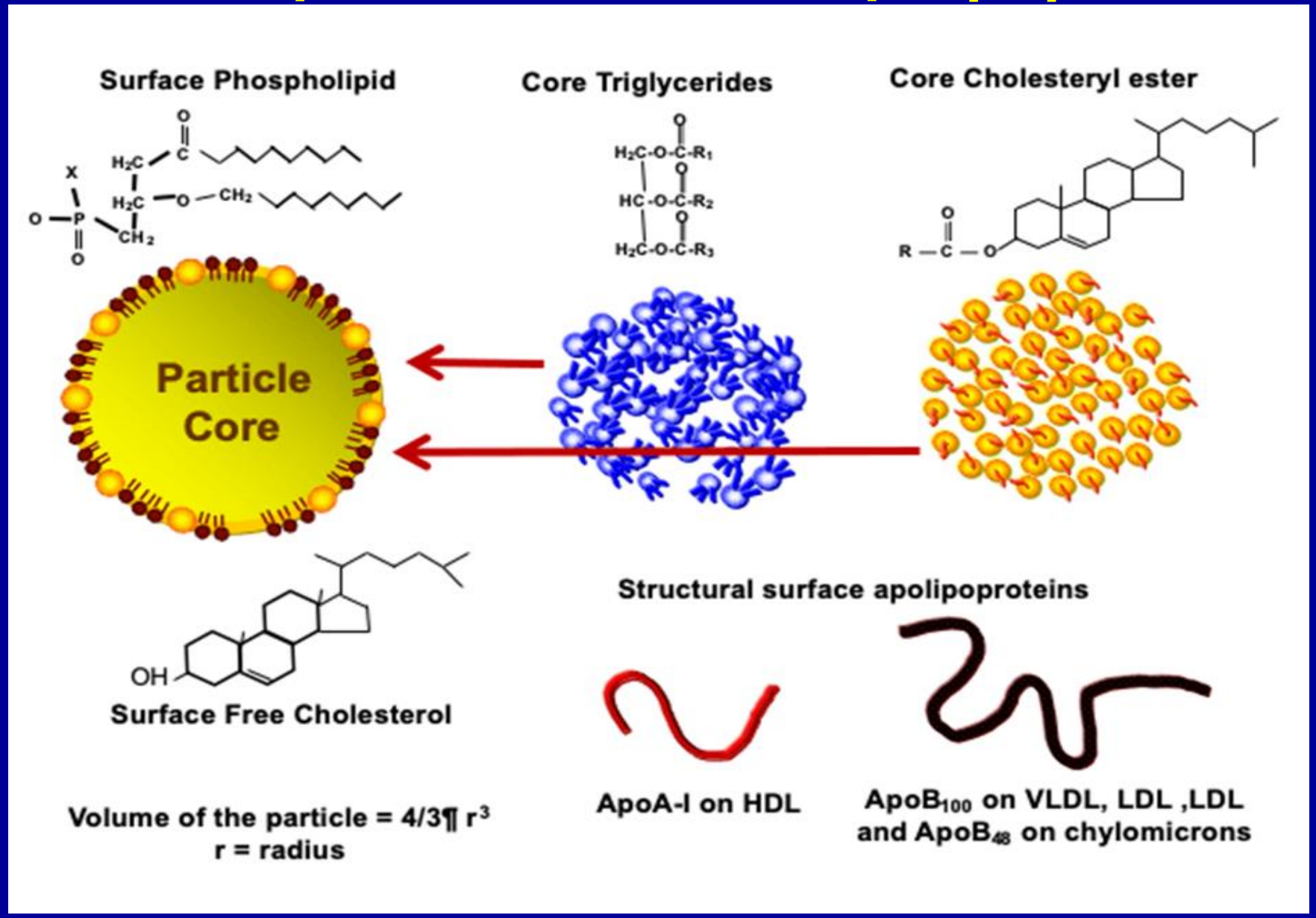
Nồng độ TG gia tăng do tăng tổng hợp và /hoặc giảm thoái biến

Thành phần Lipid máu được vận chuyển tới các cơ quan nhờ các hạt protein (lipoprotein)





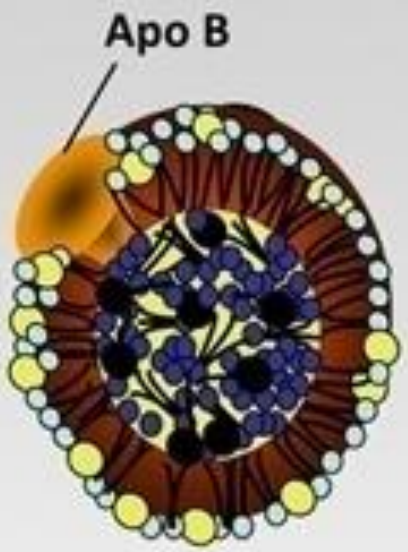
Thành phần cơ bản của hạt lipoprotein



"LDL" Should Not Imply LDL-Cholesterol

LDL là Lipoprotein vận chuyển cholesterol

LDL



Polar Surface Coat

Phospholipid

Free cholesterol

Nonpolar Lipid Core

Cholesterol ester

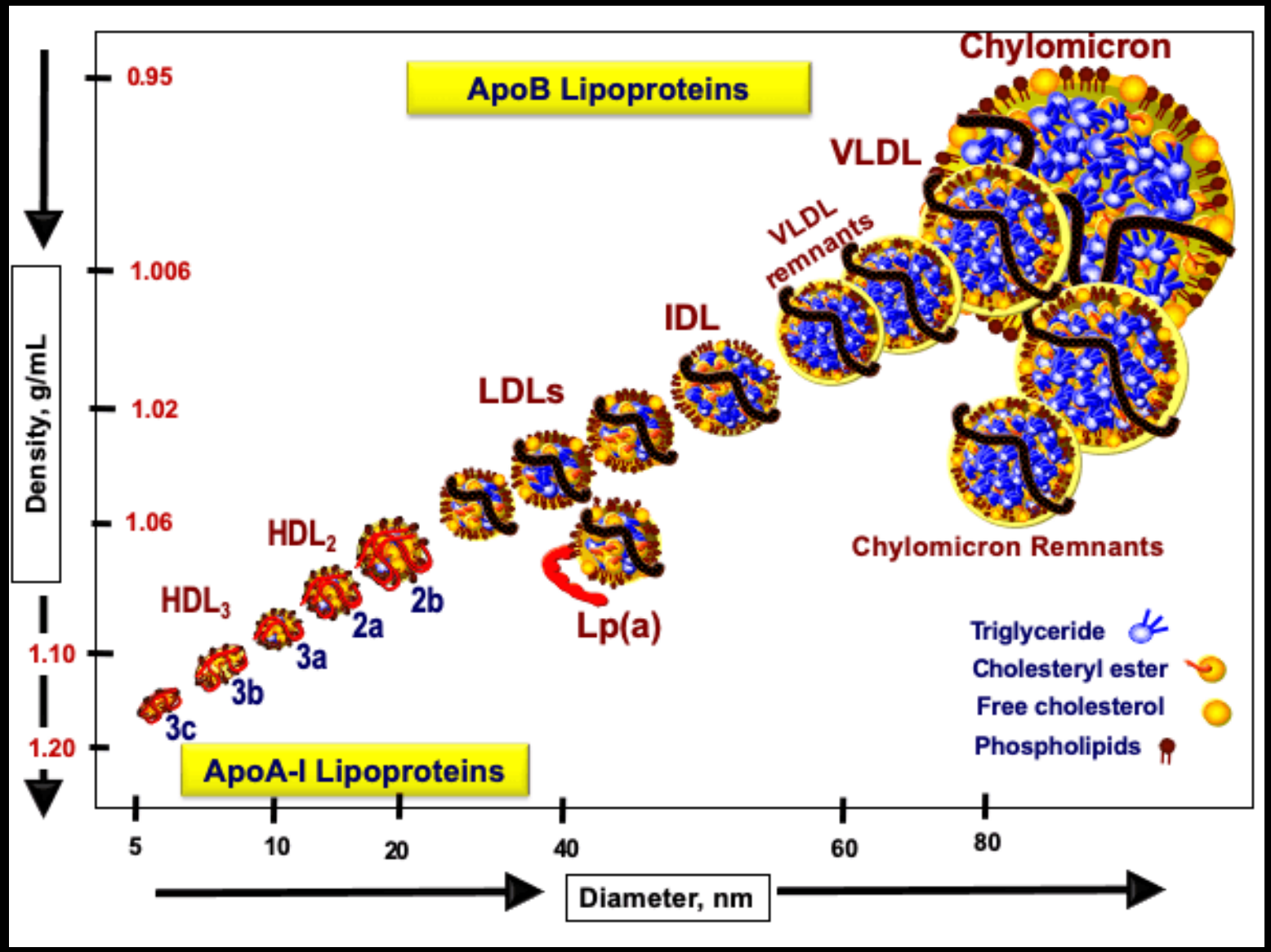
Triglyceride

LDL.C Cholesterol chứa trong hạt LDL

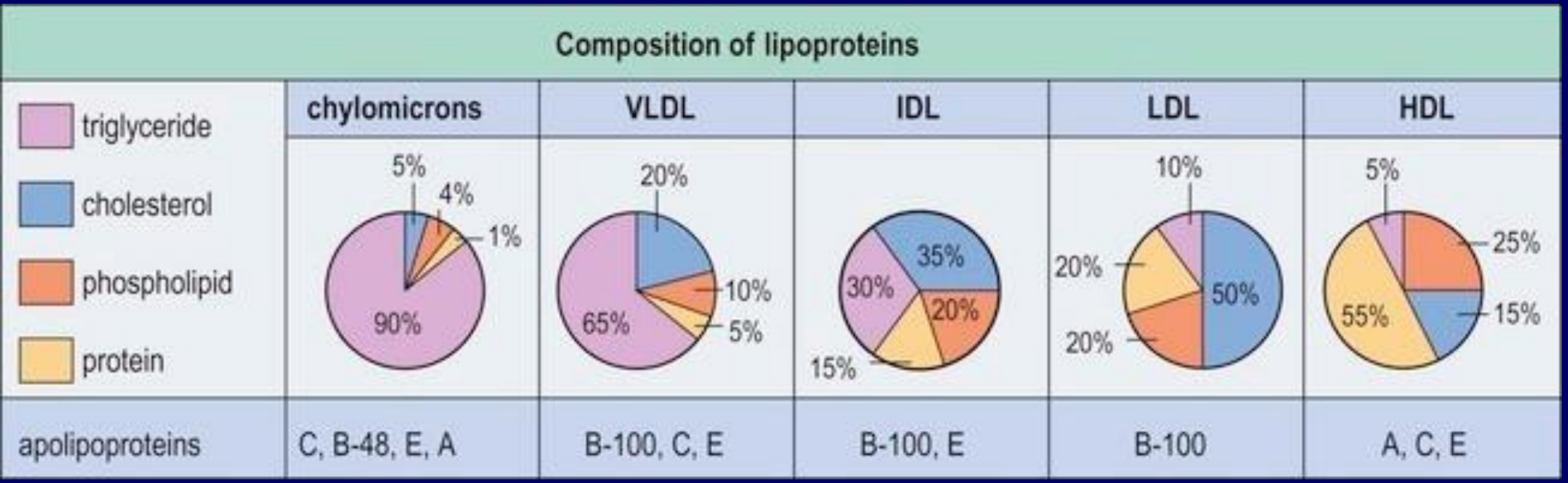
LDL.C

A convenient analytic surrogate of LDL since 1972

Các hạt lipoprotein vận chuyển thành phần lipid



Thành phần (Cholesterol, TG) trong các hạt lipoprotein



Tất cả các lipoprotein đều chứa **Cholesterol và TG** với tỷ lệ khác nhau

Lượng cholesterol chứa trong **LDL lên đến 50%**

Lượng TG chứa nhiều trong **Chylomicron (90%), VLDL(65%), IDL (30%)**

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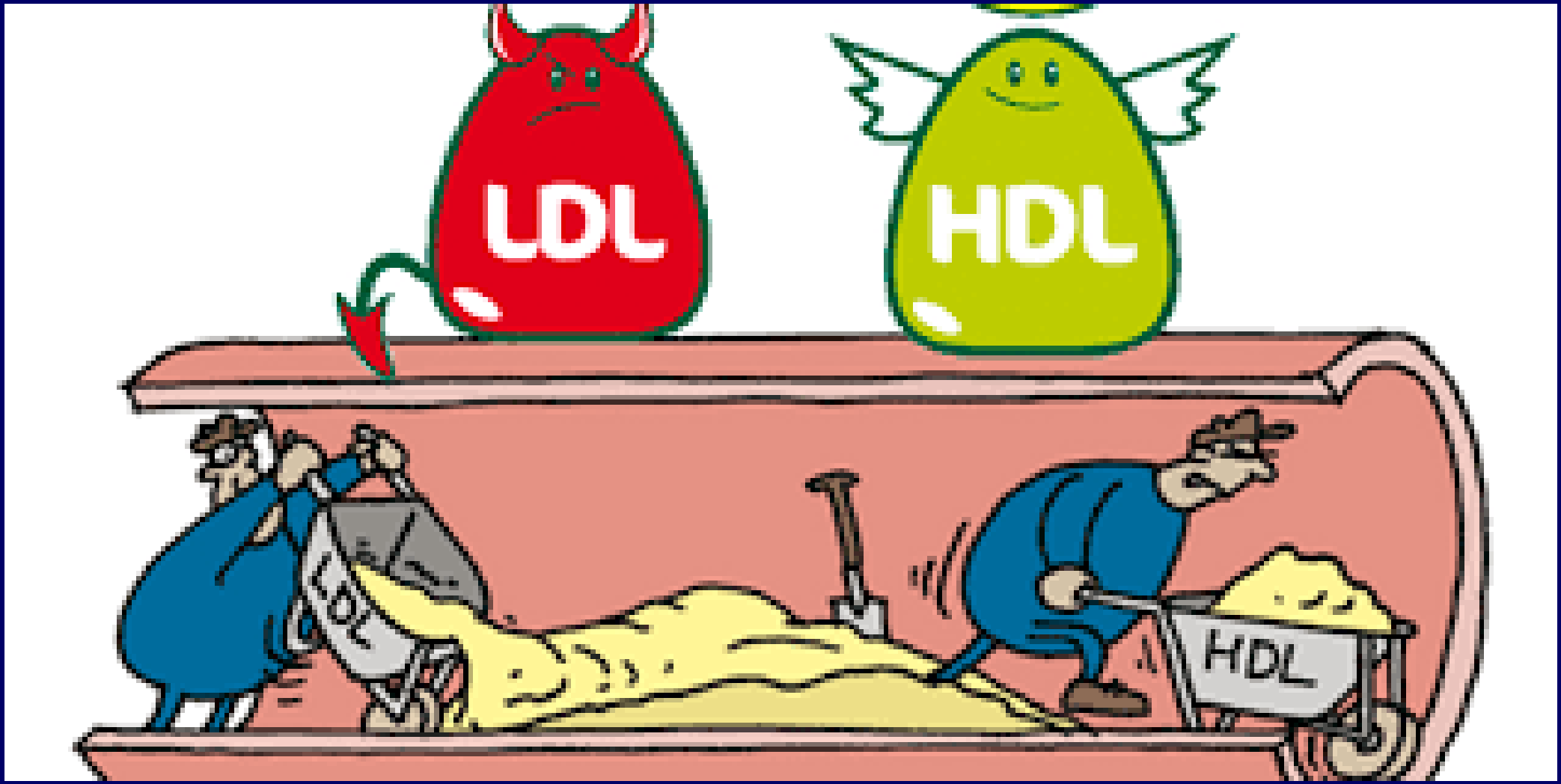
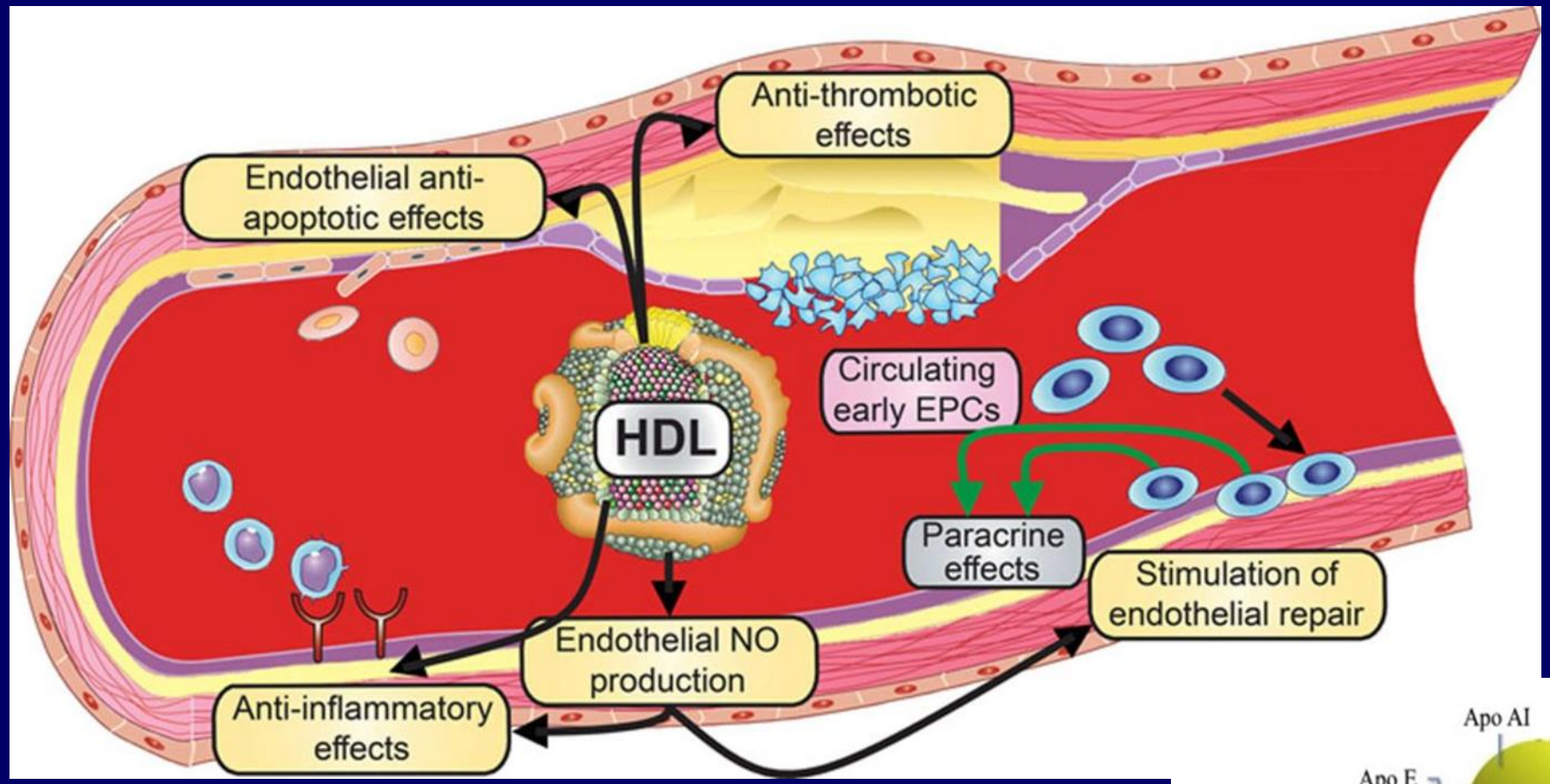
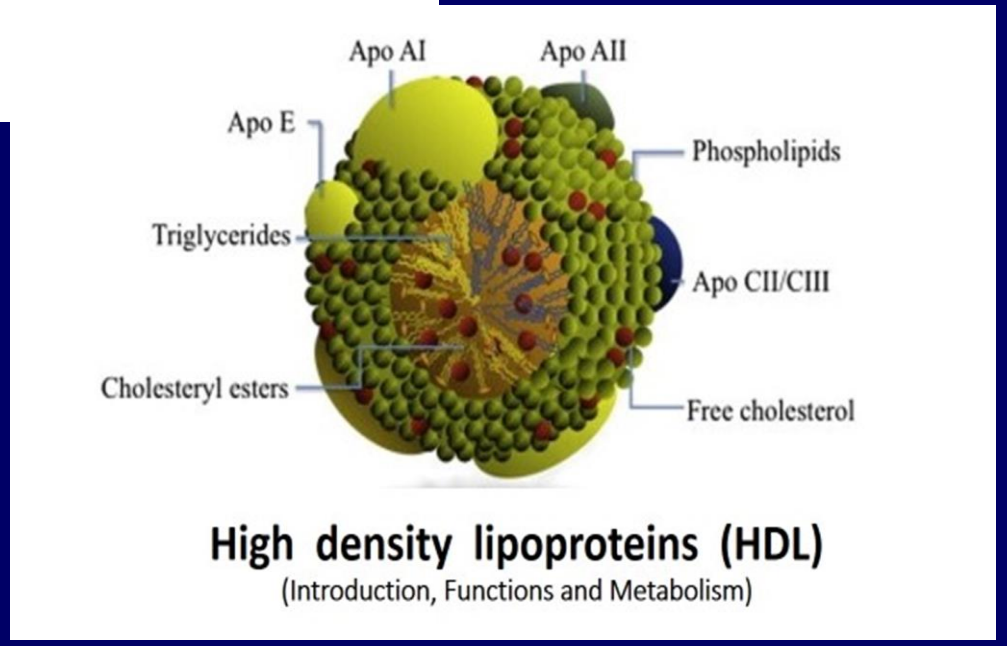
LDL gây tổn thương và HDL bảo vệ thành mạch.







Chức năng của hạt HDL

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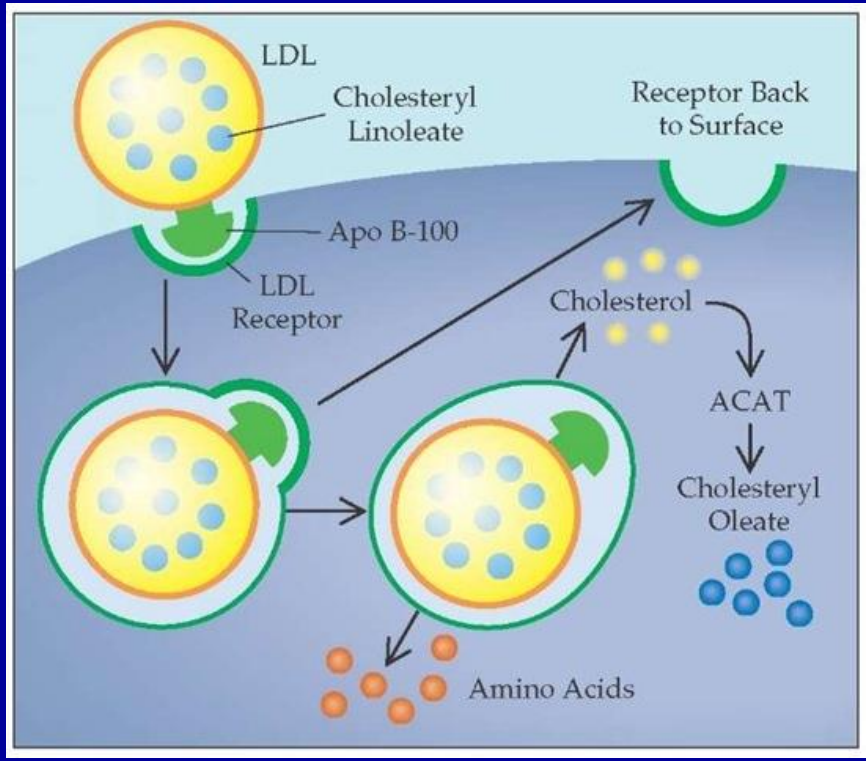
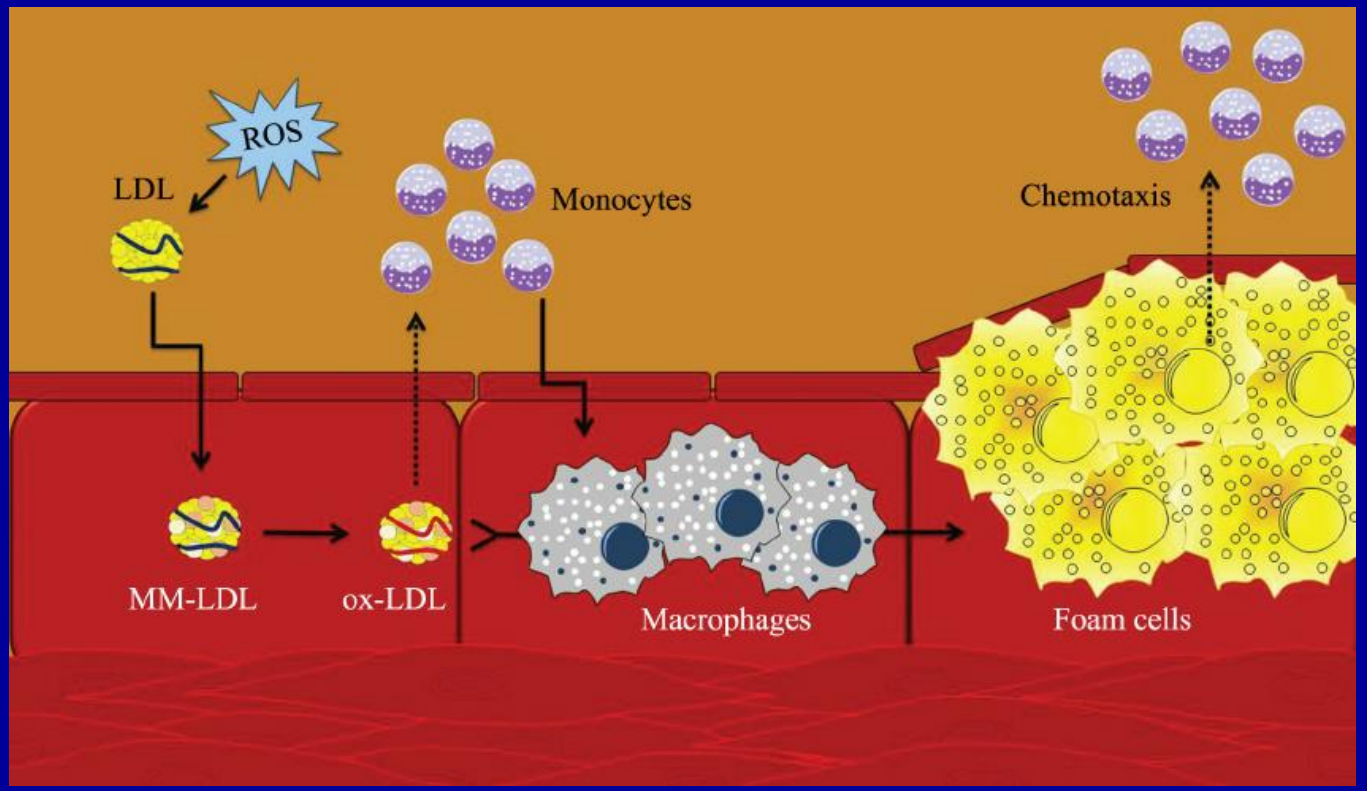
LDL không phải lúc nào cũng là nguyên nhân gây tổn thương mạch máu



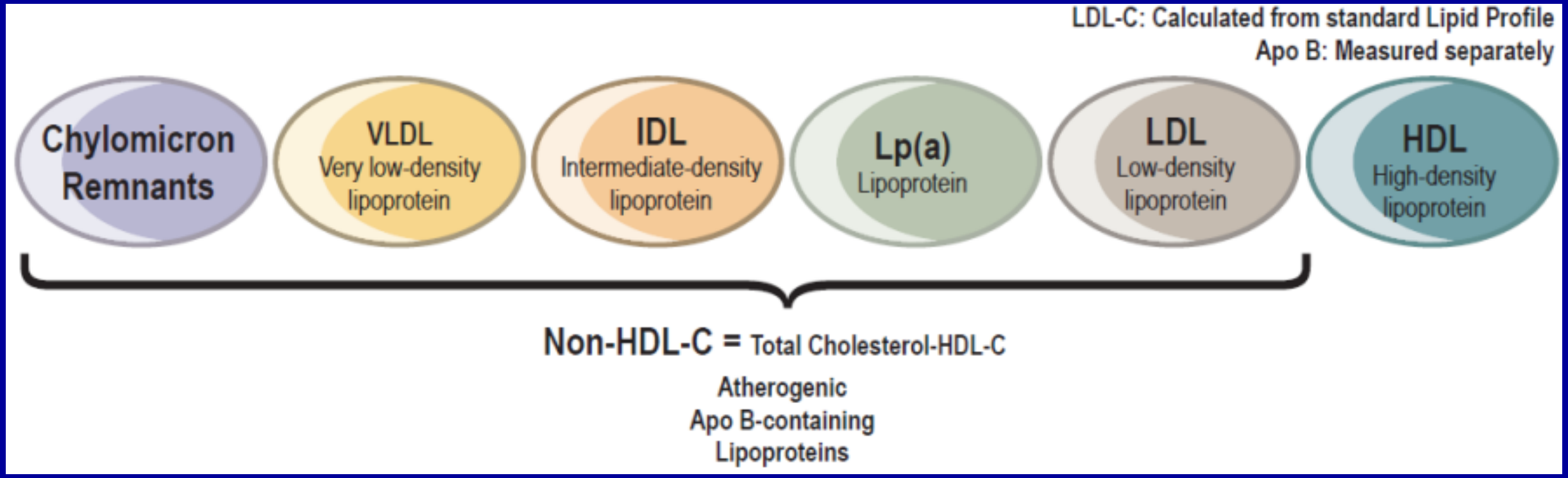


Chức năng sinh lý LDL cung cấp cholesterol cho các cơ quan

LDL cũng là nguyên nhân gây tổn thương mạch máu

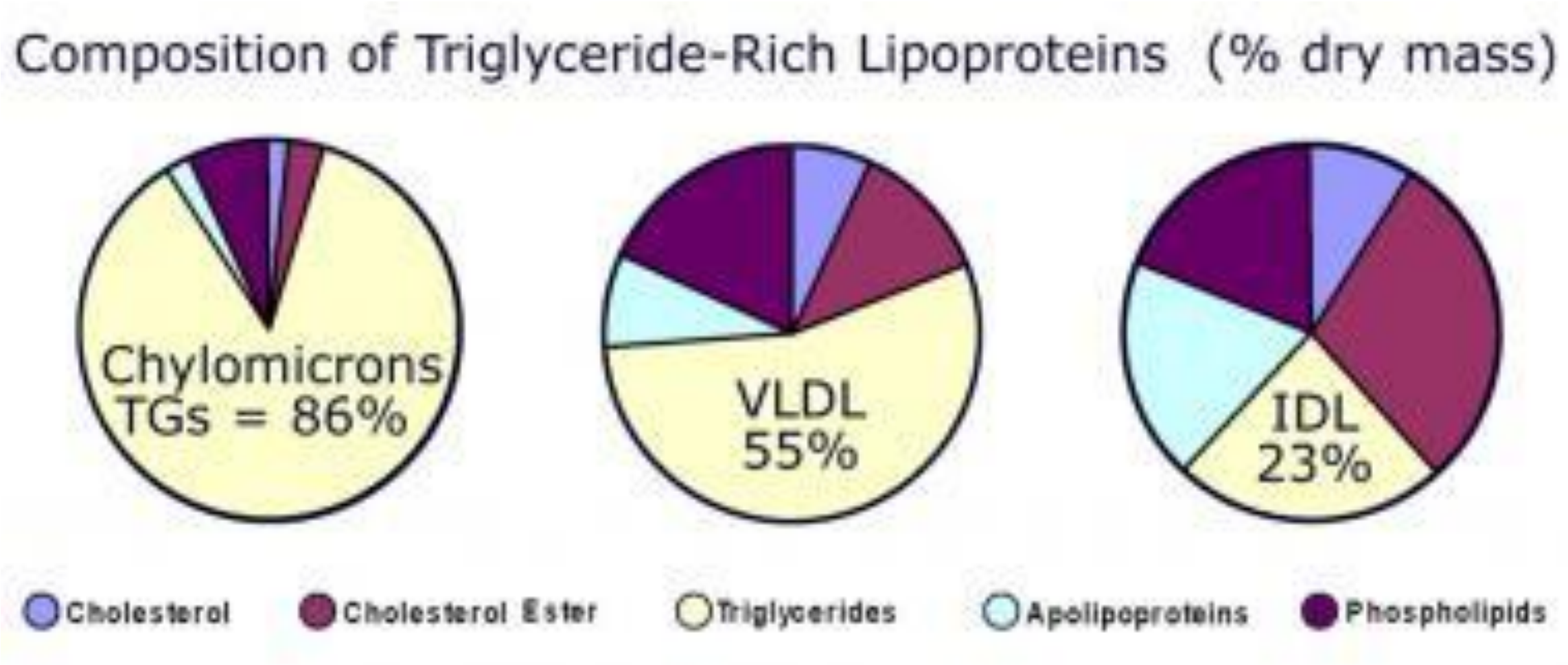
4. Có phải Non-HDL.C và Rối loạn lipid máu kết hợp tuy hai nhưng chỉ là một không ?

Non HDL.C là lượng cholesterol chứa trong các lipoprotein Non HDL (bao gồm LDL) ngoài trừ HDL.



Non HDL.C = TC – HDL.C

Non HDL bao gồm các lipoprotein giàu TG (IDL, VLDL, Chylomicron thừa)



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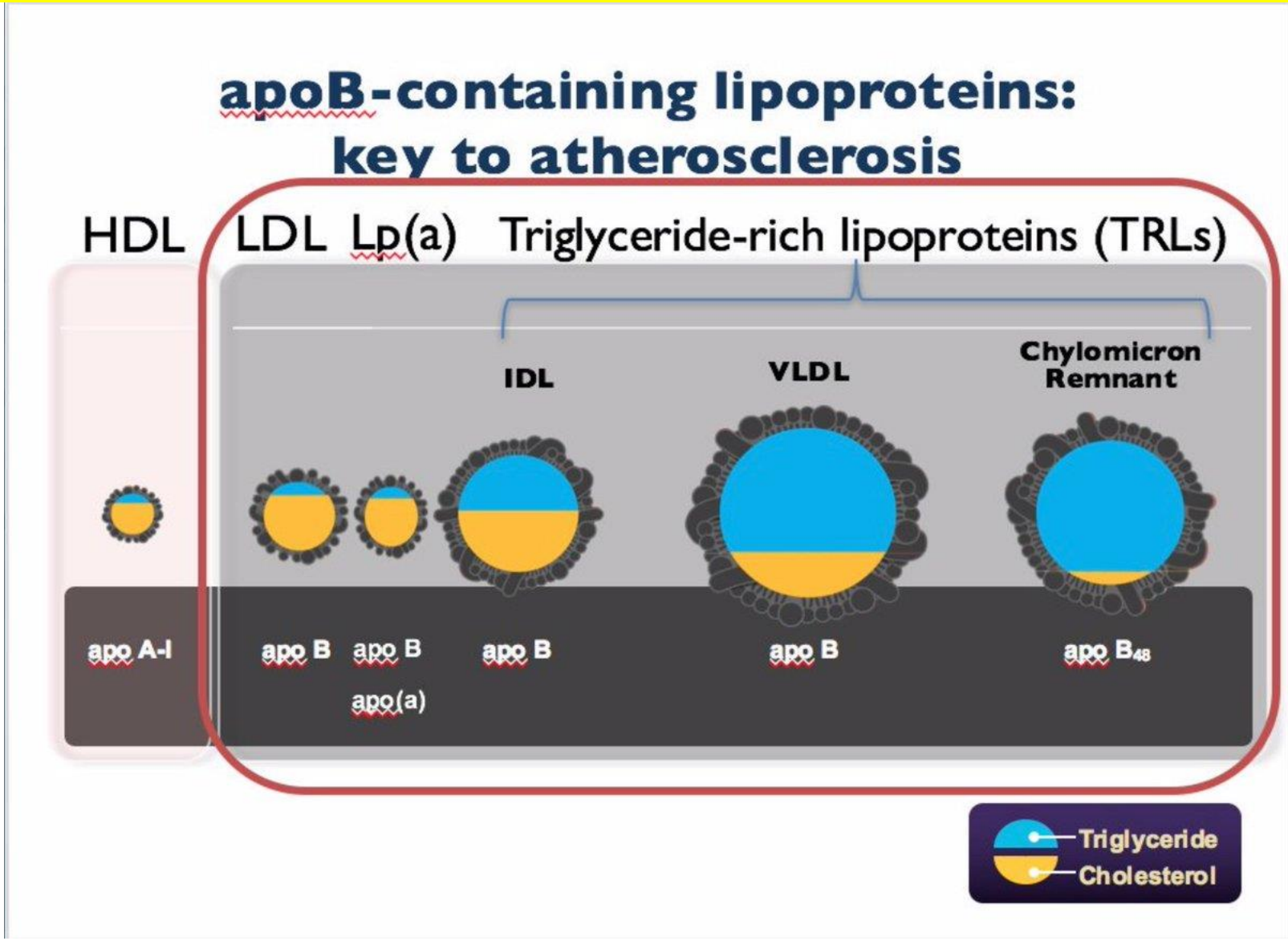
Non HDL là các Lipoprotein giàu TG (TRLs) có gắn apoB



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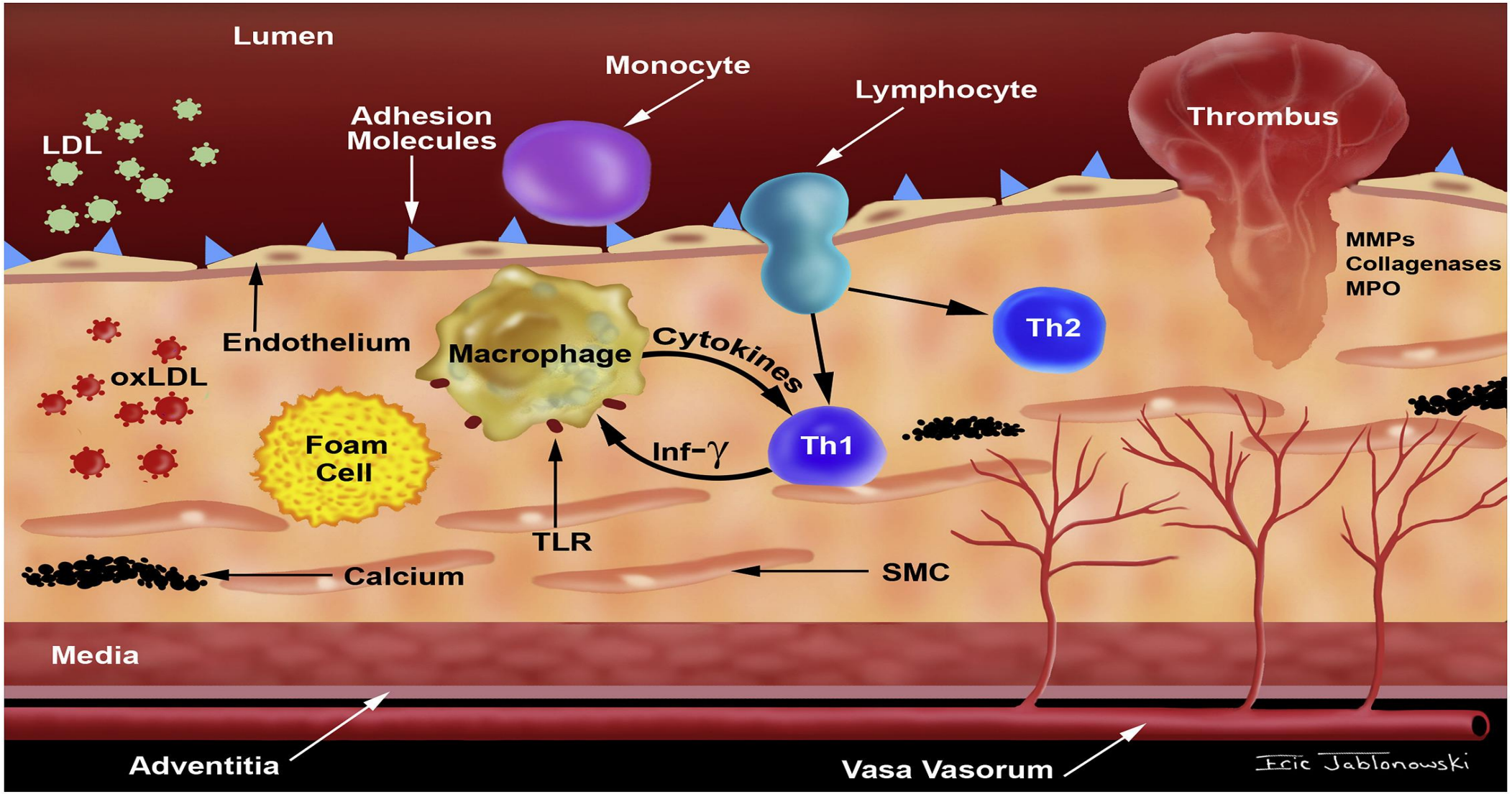
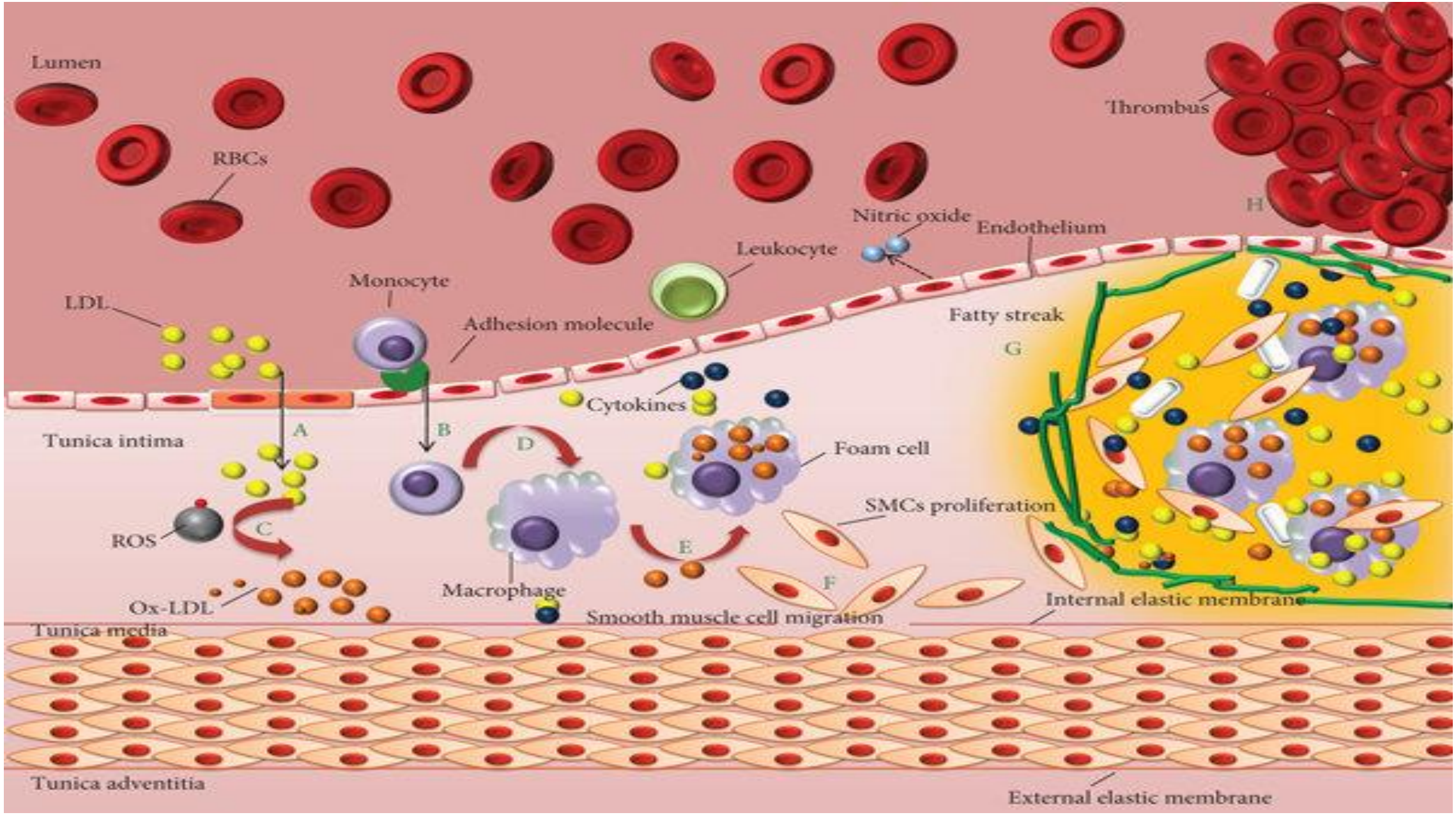
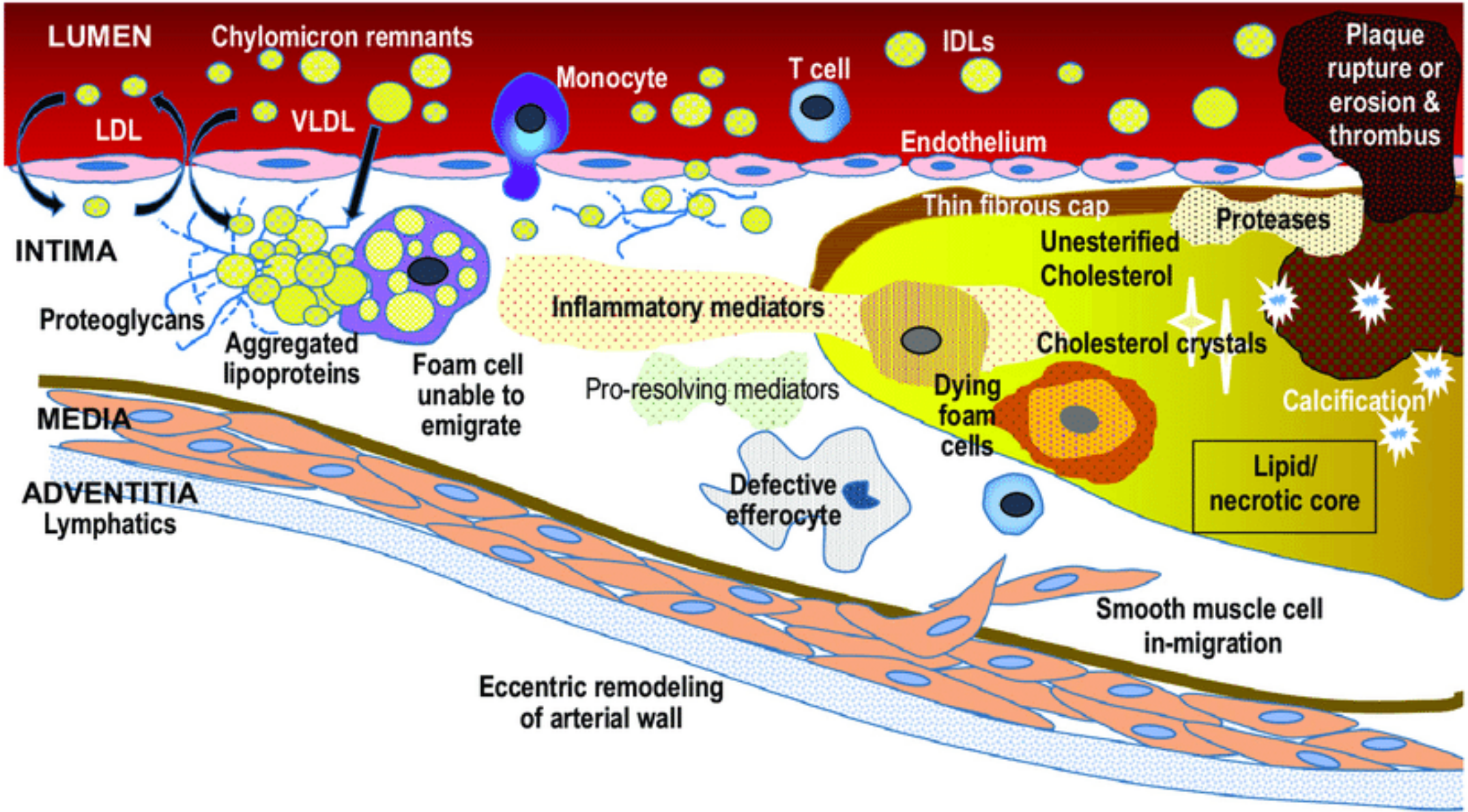
1. Cơ chế xơ vữa động mạch do LDL



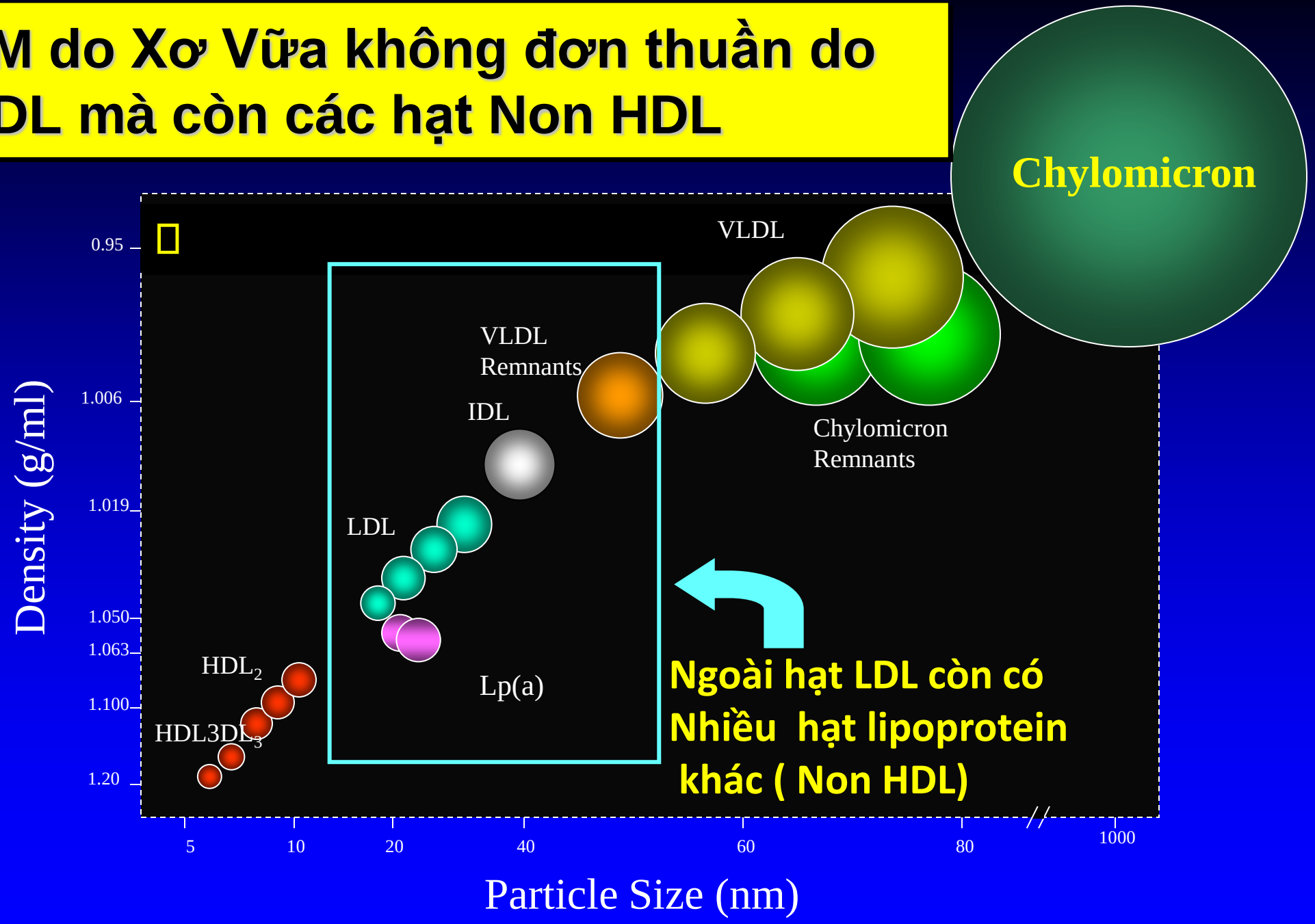

Lumen
RBCs
Thrombus
H
Nitric oxide
Endothelium
Leukocyte
Monocyte
LDL
Adhesion molecule
Fatty streak
G
Cytokines
Tunica intima
A
B
D
Foam cell
SMCs proliferation
ROS
C
E
F
Macrophage
Ox-LDL
Internal elastic membrane
Smooth muscle cell migration
Tunica media
Tunica adventitia
External elastic membrane

Trong mãng xơ vữa động mạch còn phát hiện nhiều loại lipoprotein ngoài LDL

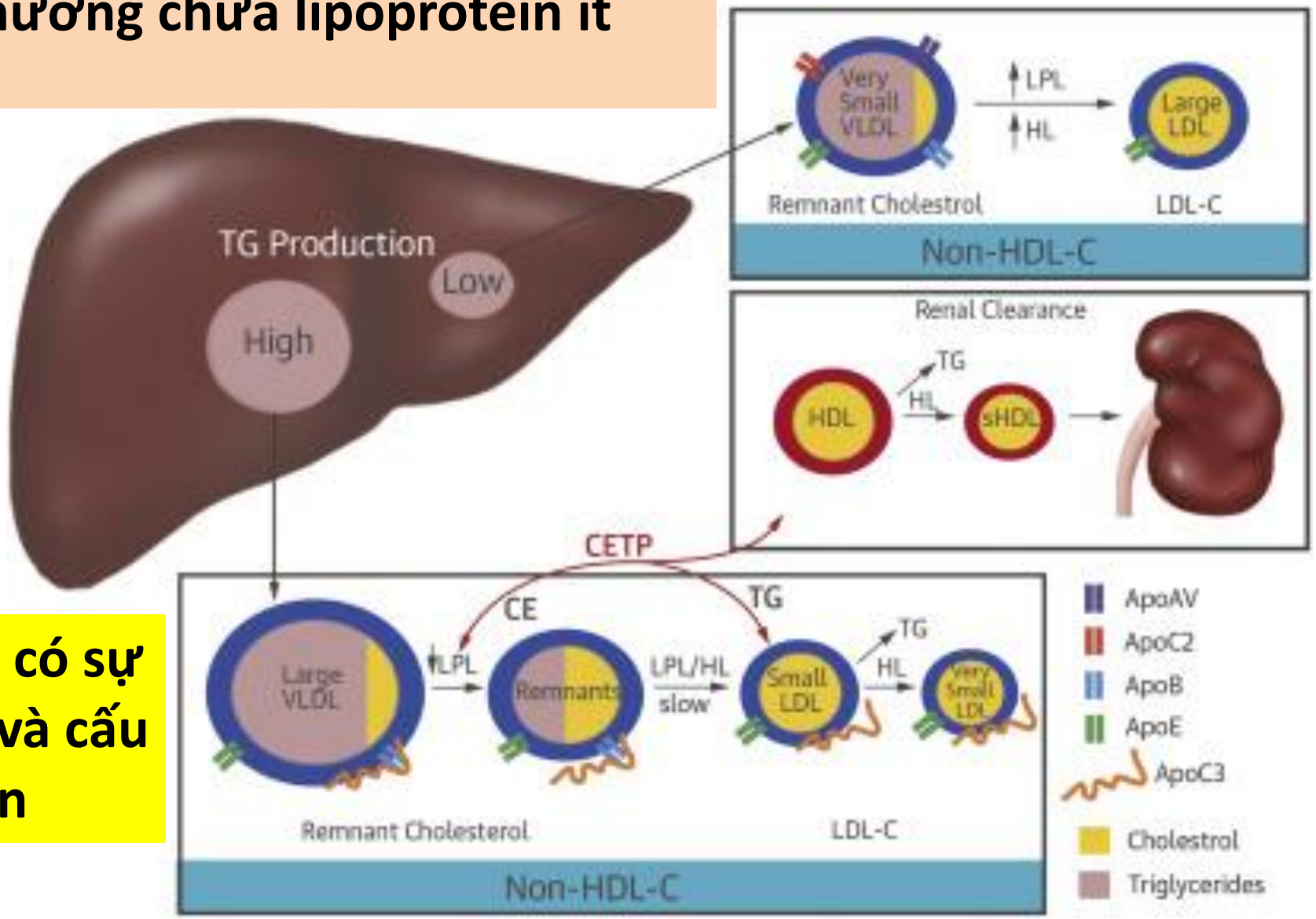


V

Bệnh TM do Xơ Vữa không đơn thuần do LDL mà còn các hạt Non HDL



Nồng độ TG bình thường chứa lipoprotein ít gây xơ vữa



Khi nồng độ TG cao có sự thay đổi hình dạng và cấu trúc lipoprotein

LDLsd và Lipoprotein giàu TG đều gây xơ vữa nặng

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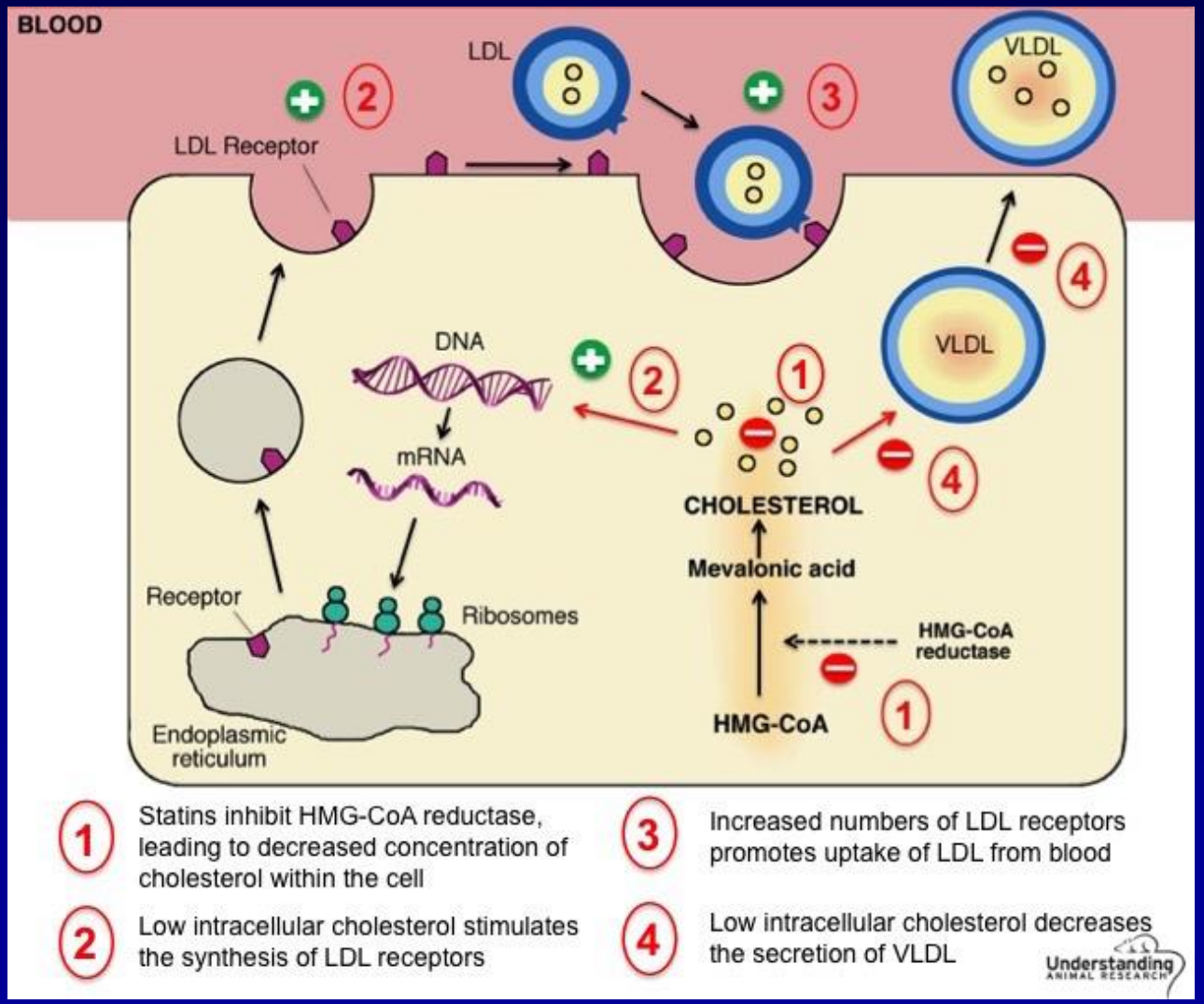
2. Cơ chế sinh xơ vữa của LDLsd (LDLnhỏ đậm đặc)



LDLsd

-Tác động trên nội mạc mạch làm tăng superoxide (O^{2-}) , giảm NO

-Xuyên qua thành mạch

-Tăng nhạy cảm oxy hoá

-Tăng liên kết với proteoglycans nội mạc

-Tạo các chất trung gian tiền ngưng tập và các chất co mạch như thromboxane A2, 8-epi-PGF2α…

- Giảm ái lực thụ thể LDL tại gan

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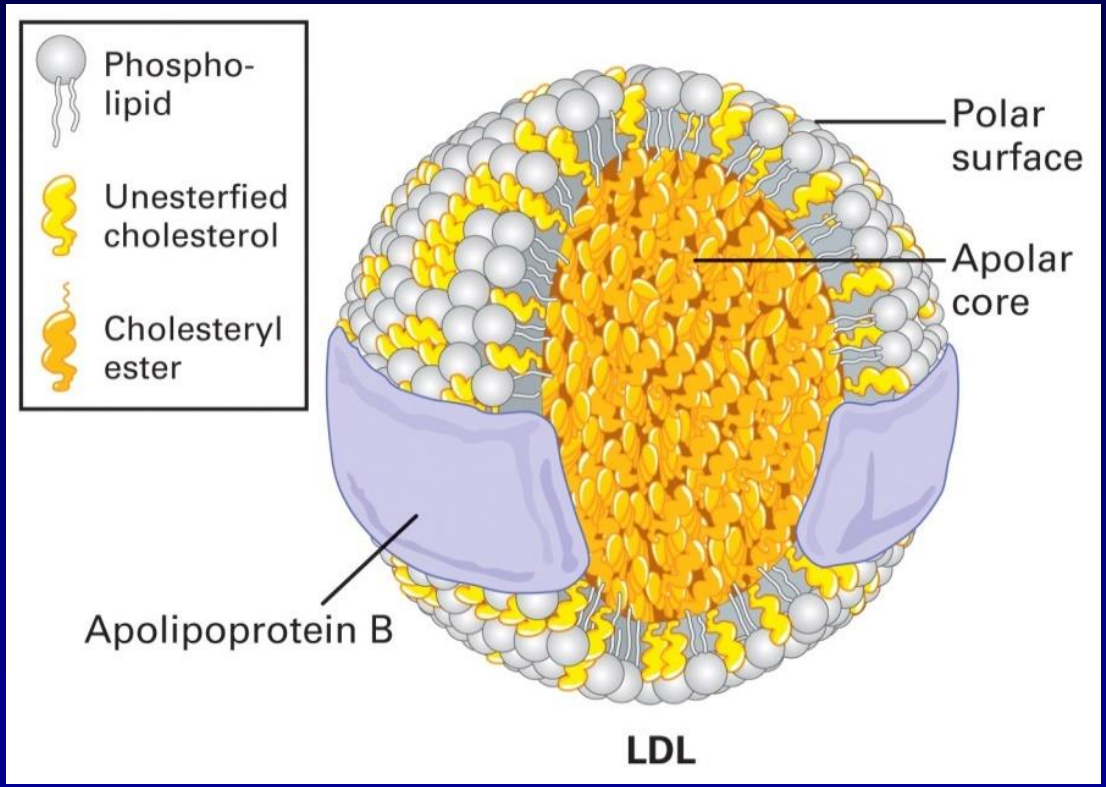
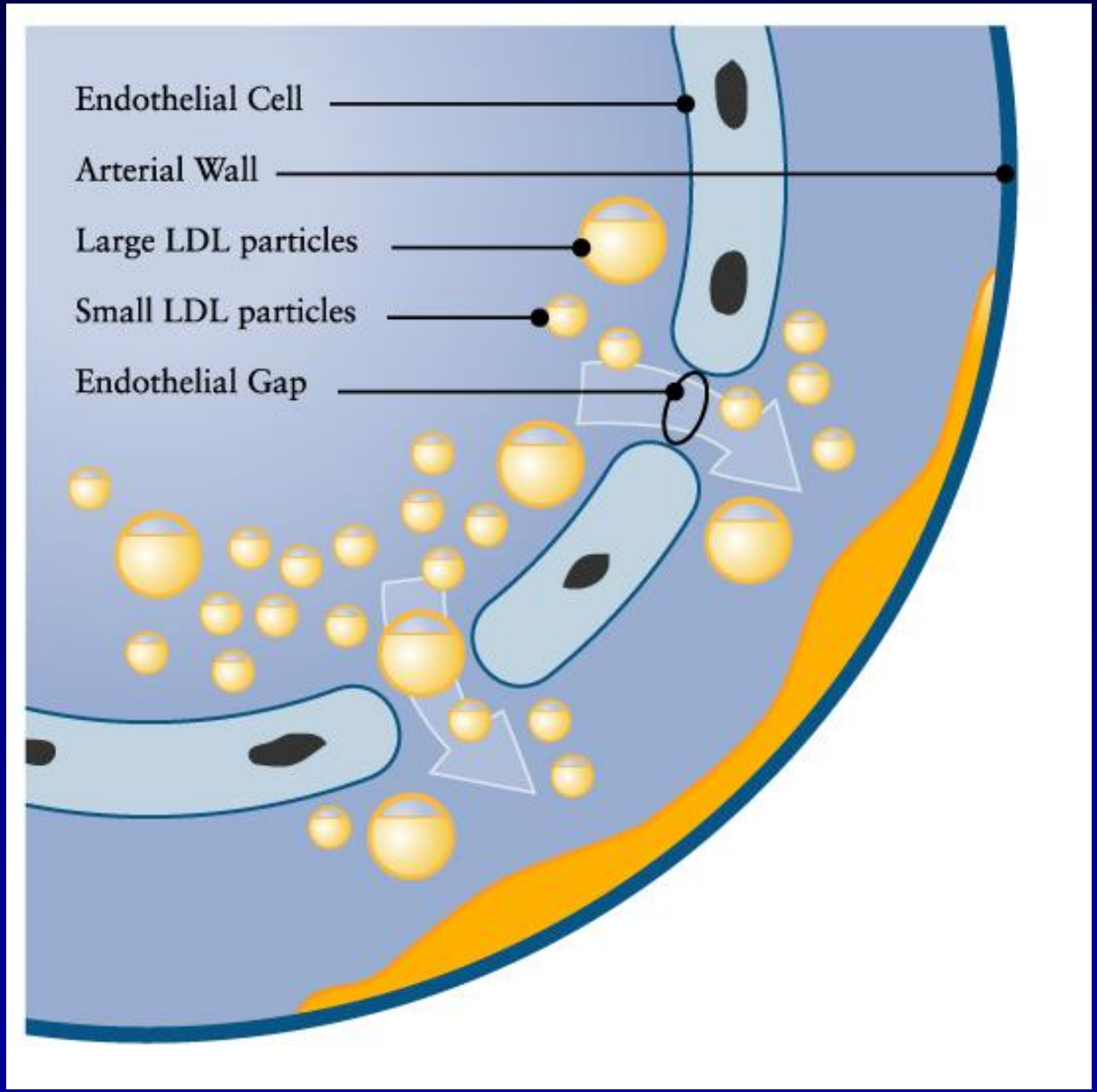
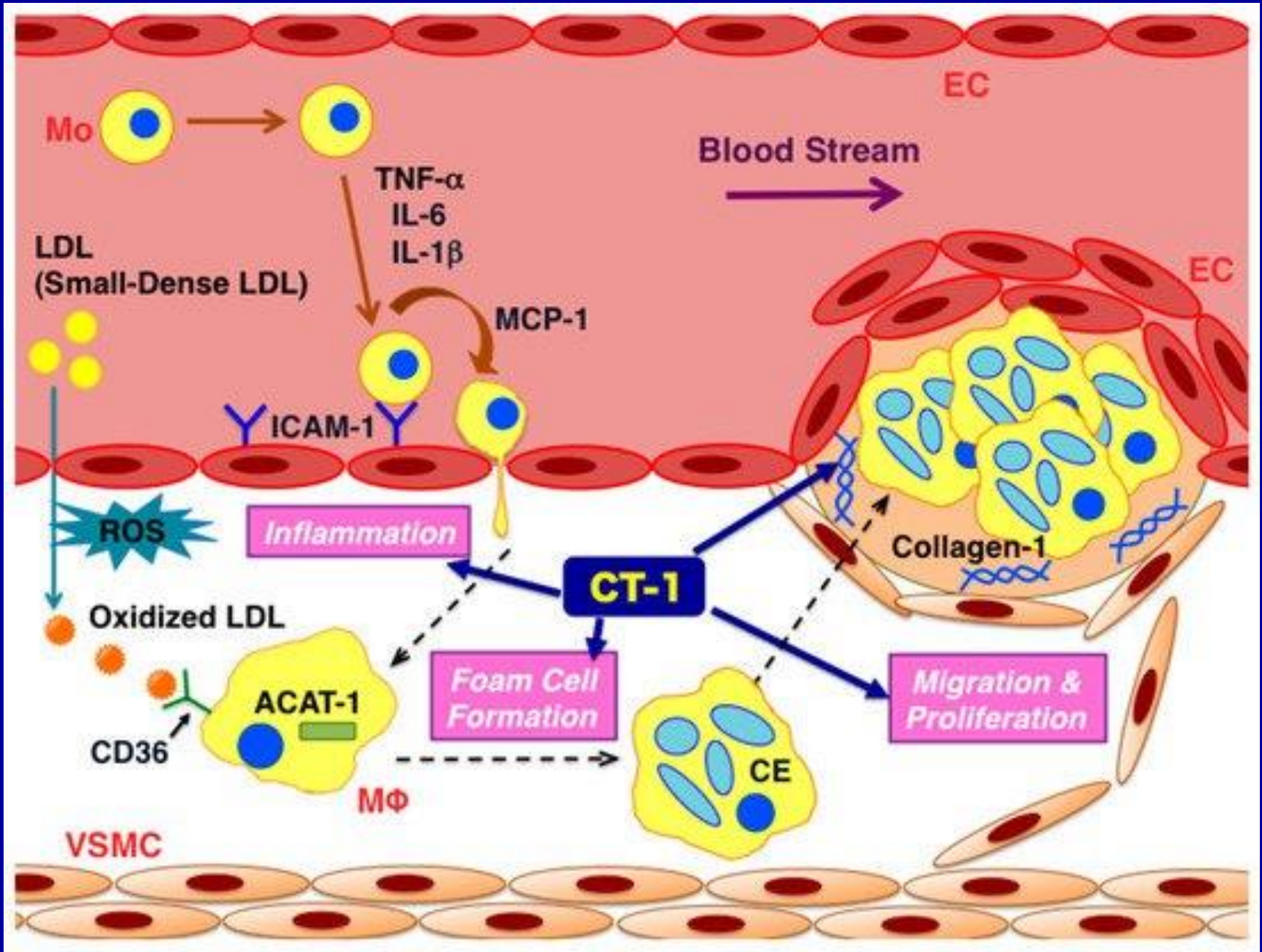




1 Fredrickson et al. NEJM 1967; 276: 148


EC
Mo
Blood Stream
TNF-α
IL-6
IL-1β
LDL
(Small-Dense LDL)
MCP-1
EC
ICAM-1
ROS
Inflammation
Collagen-1
CT-1
Oxidized LDL
ACAT-1
Foam Cell
Formation
Migration &
Proliferation
CD36
CE
MΦ
VSMC

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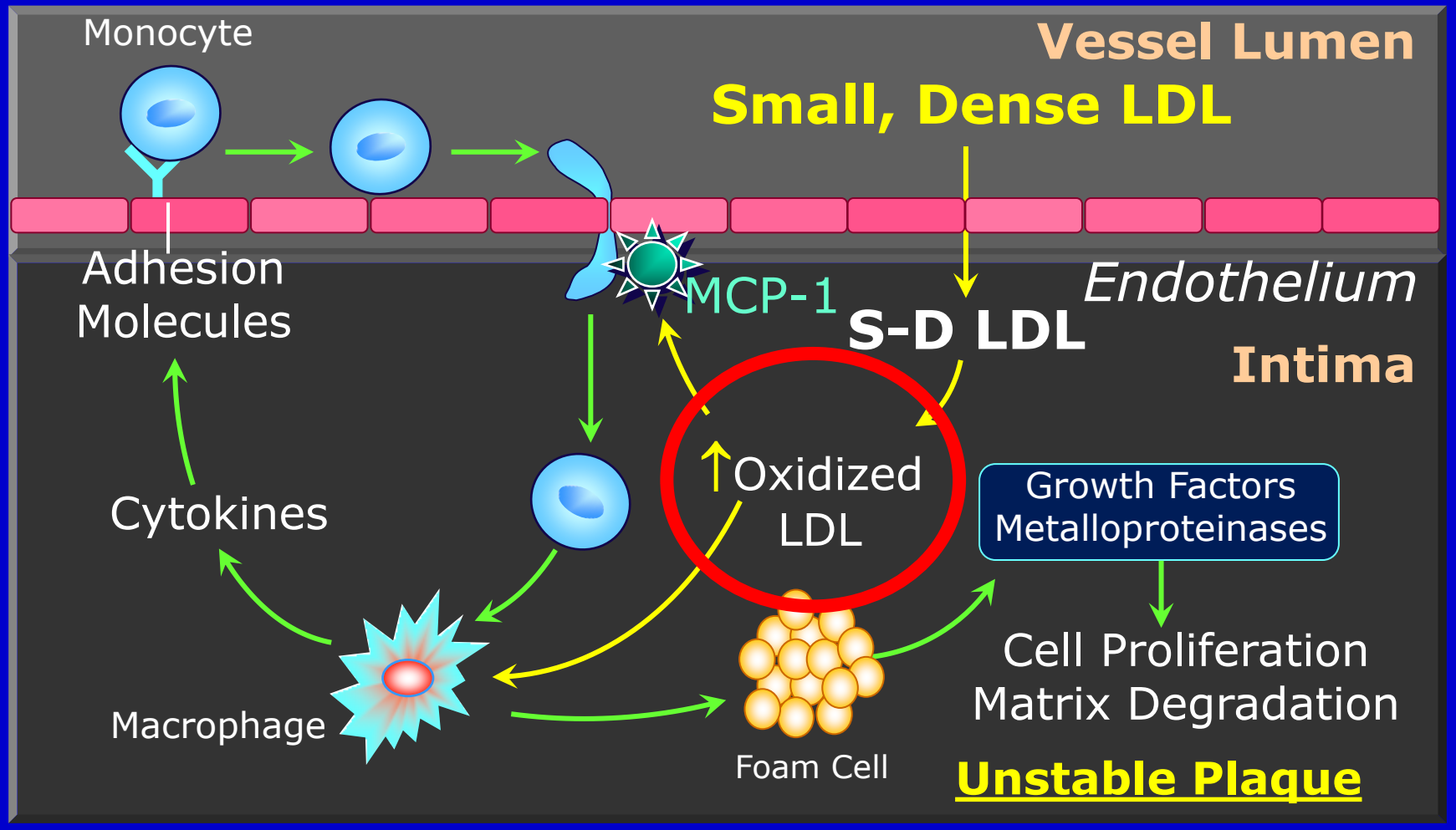
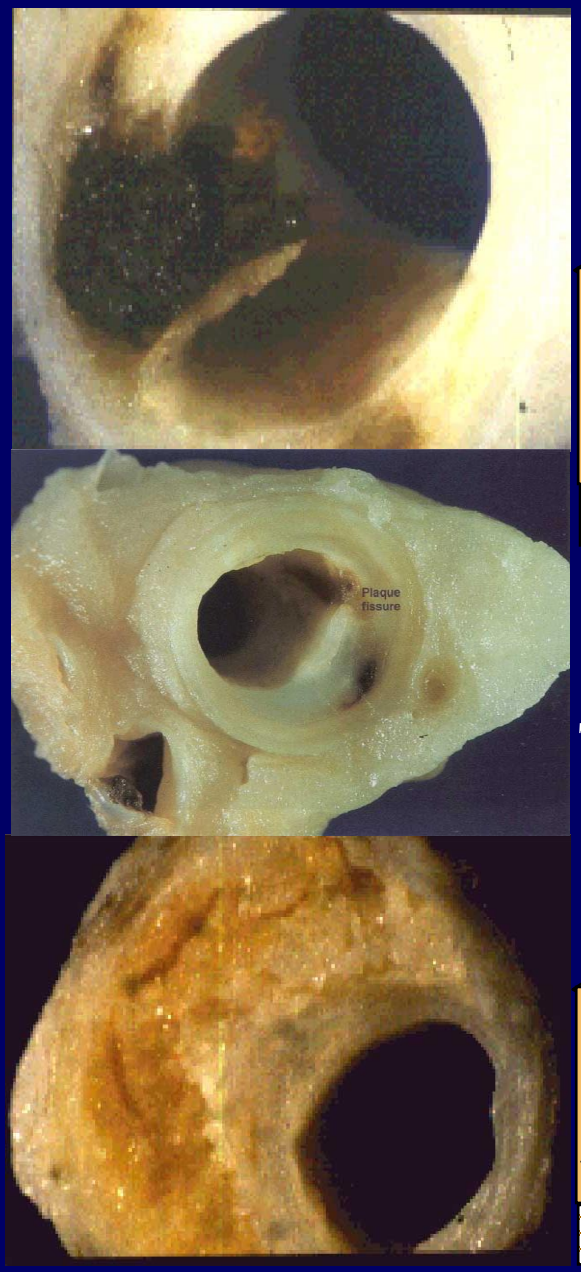
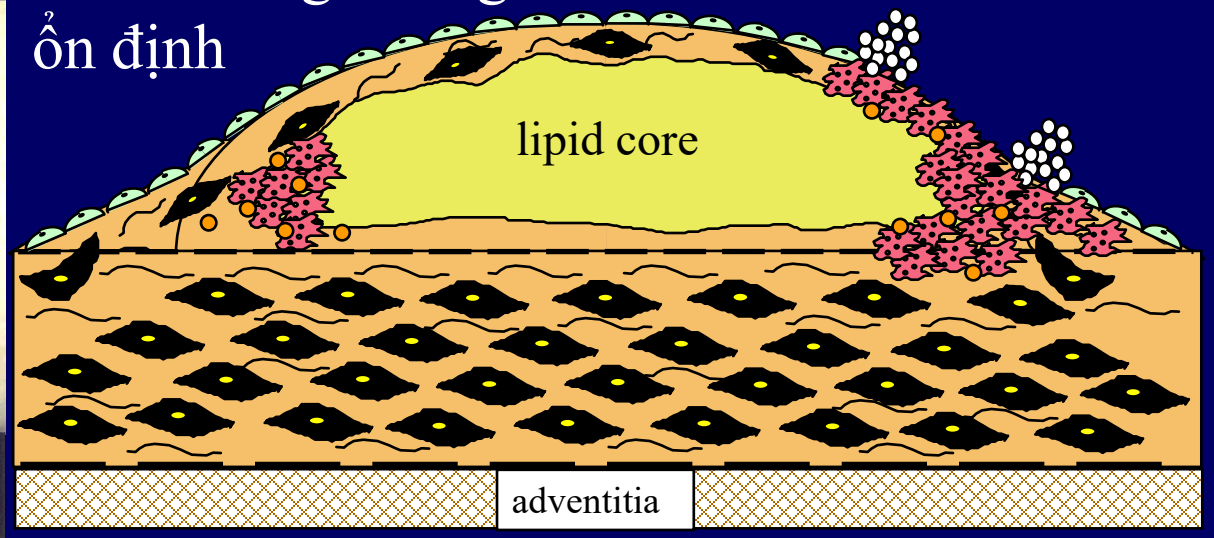
LDLsd và mãng xơ vữa không ổn định



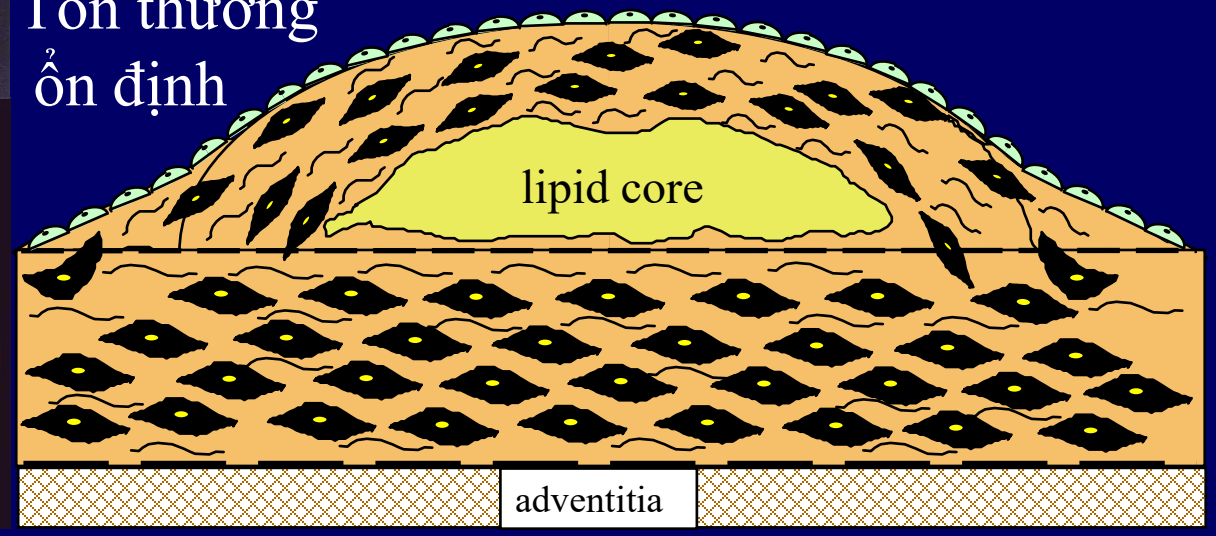


Tổn thương không ổn định



Điều trị chống xơ vữa

Tổn thương ổn định



From Davies et al (1998)

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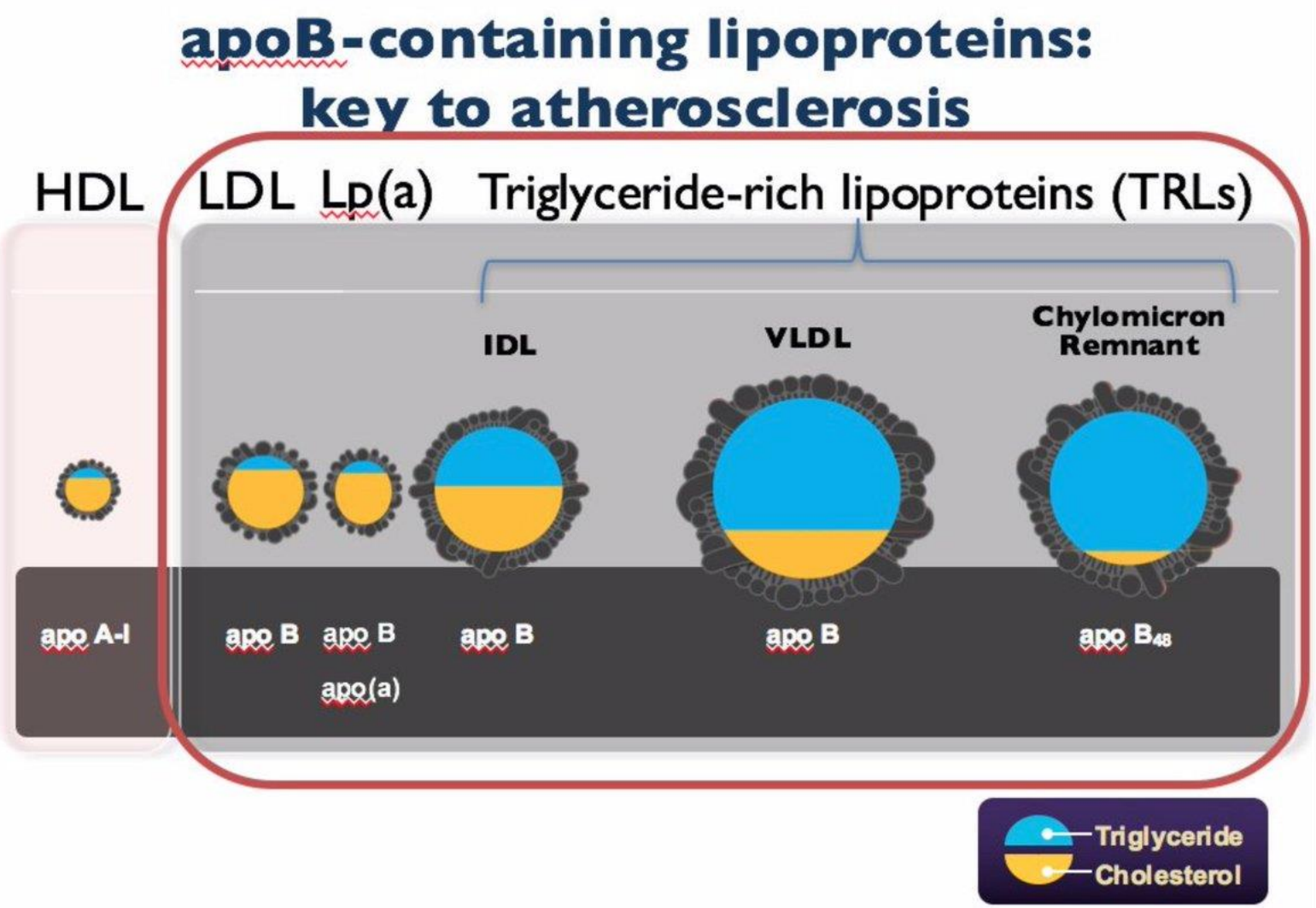
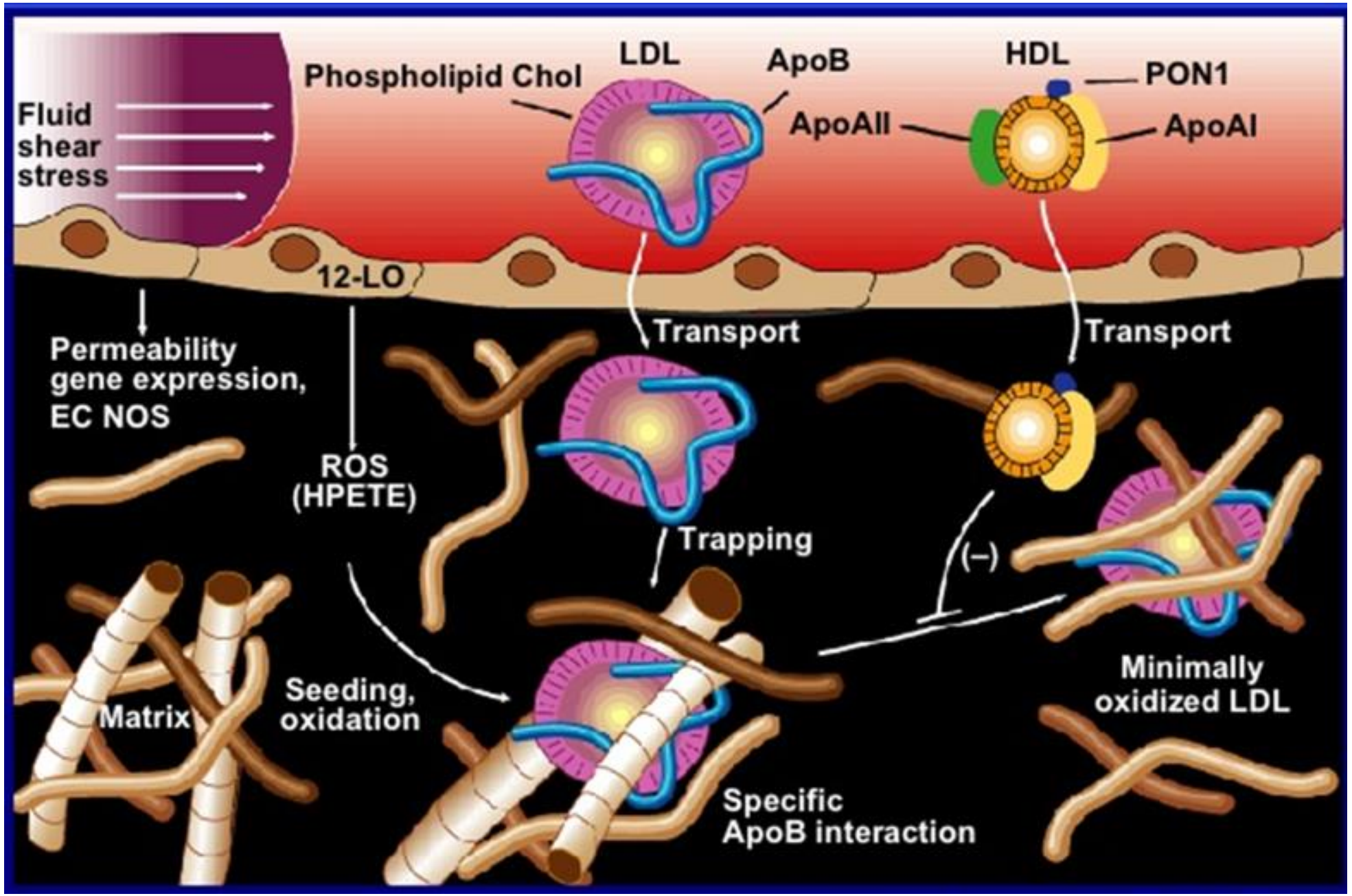
3. Cơ chế sinh xơ vữa Lipoprotein giàu TG (TRLs) có gắn apoB




Fluid
shear
stress
Phospholipid Chol
LDL
ApoB
HDL
PON1
ApoAII
ApoAI
12-LO
Permeability
gene expression,
EC NOS
ROS
(HPETE)
Transport
Transport
Trapping
(–)
Matrix
Seeding,
oxidation
Minimally
oxidized LDL
Specific
ApoB interaction

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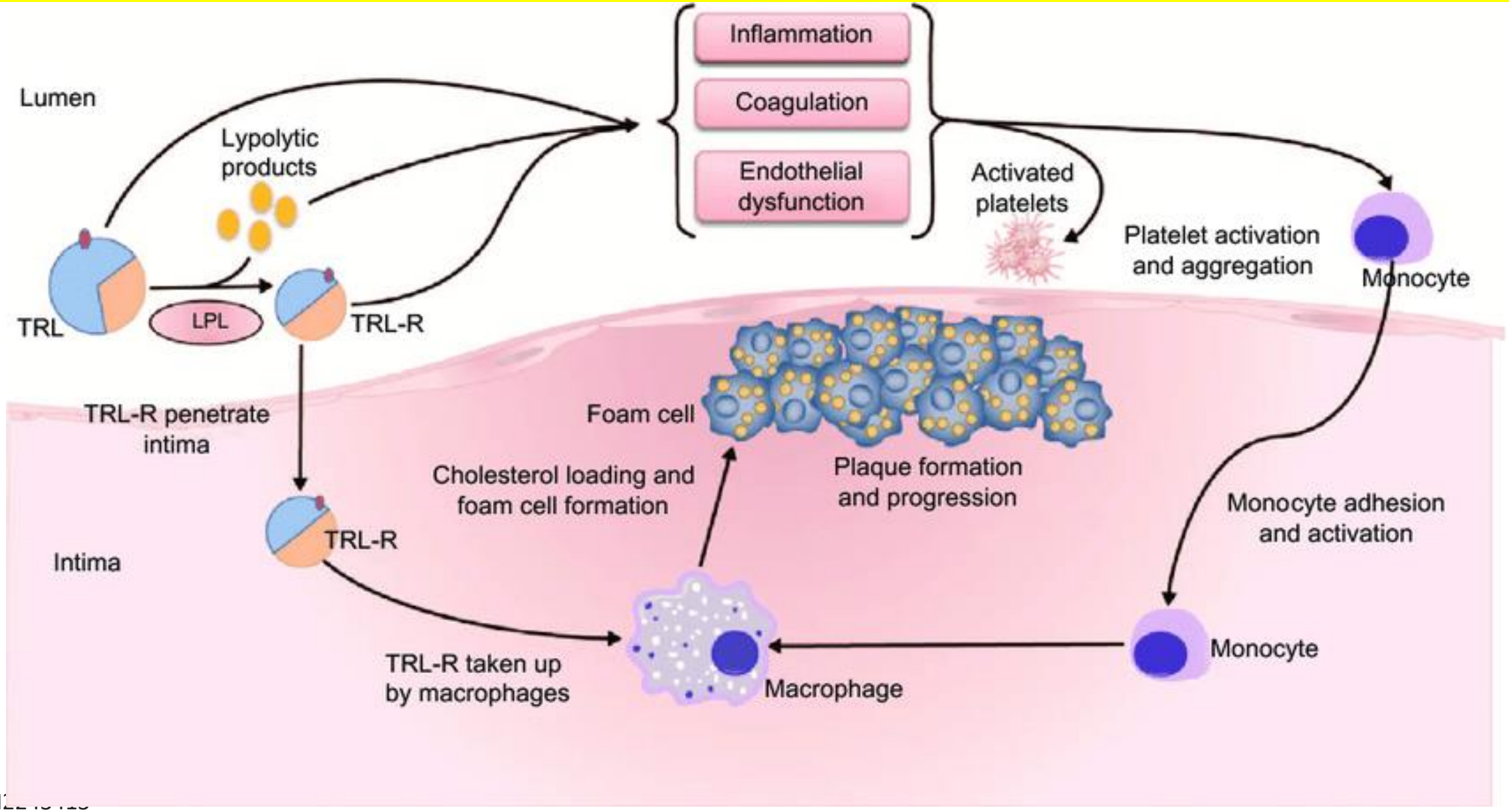
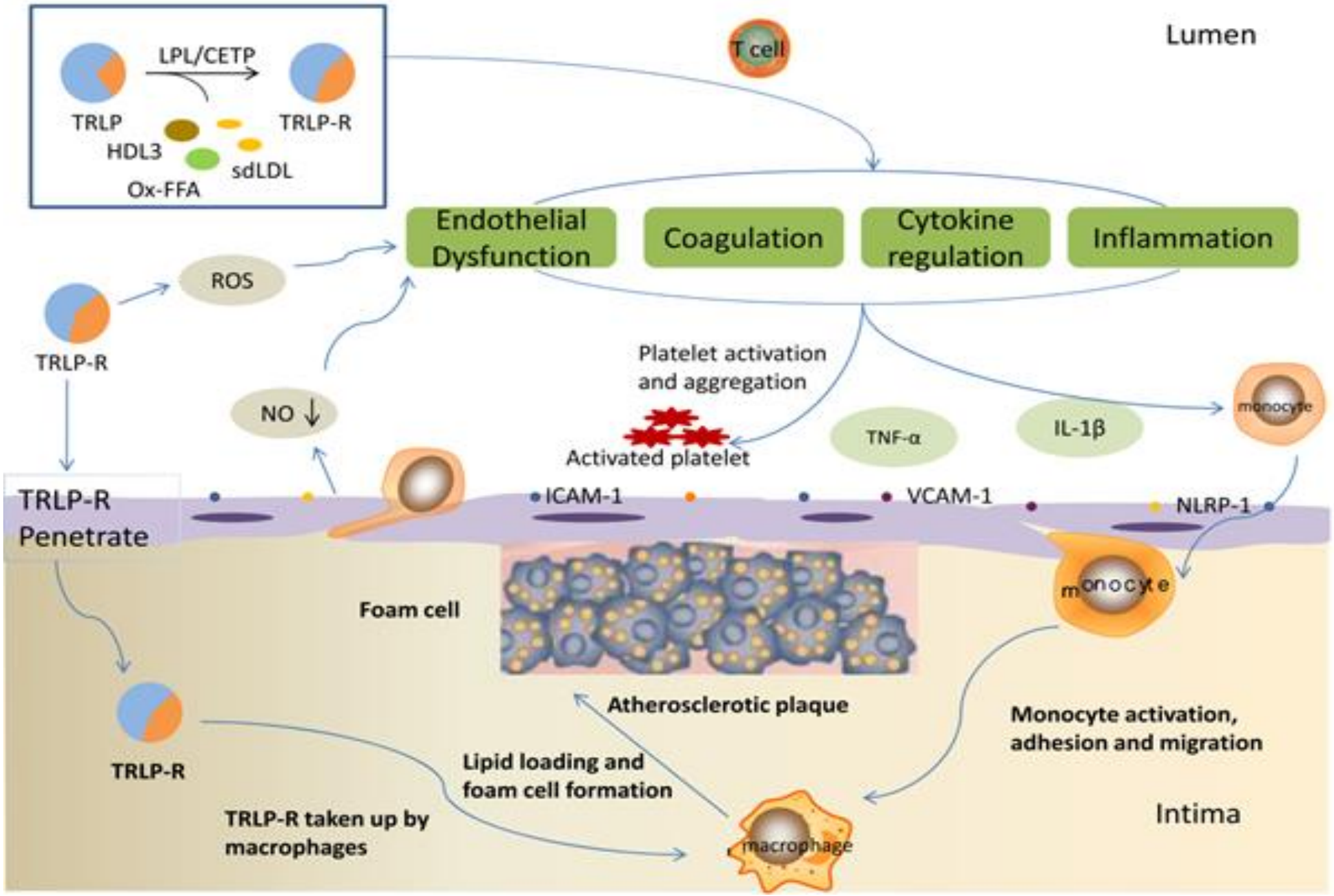
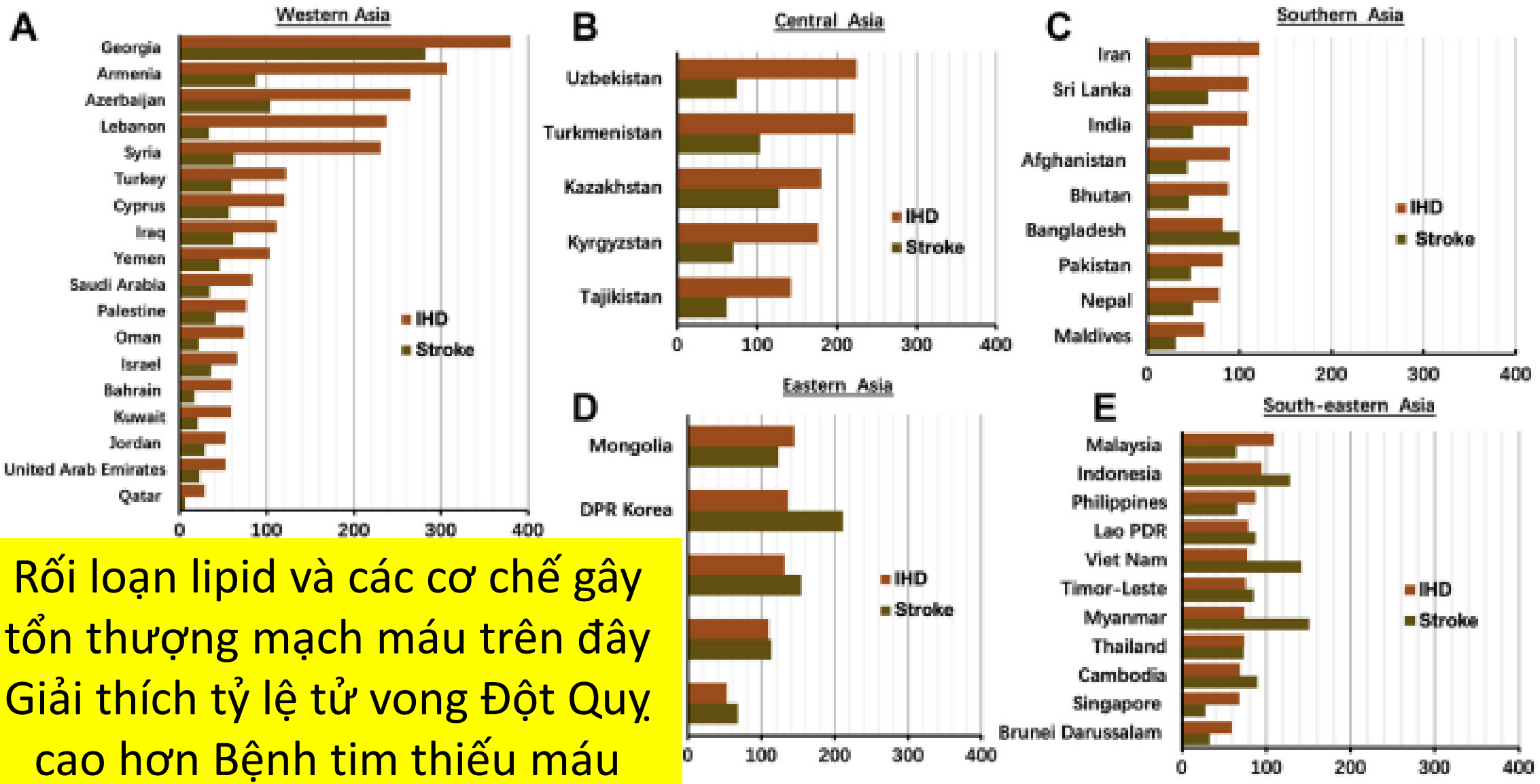
Cơ chế sinh xơ vữa hạt Lipoprotein giàu TG (TRPs)




LPL/CETP
TRLP
TRLP-R
HDL3
sdLDL
Ox-FFA
T cell
Lumen
Endothelial Dysfunction
Coagulation
Cytokine regulation
Inflammation
ROS
TRLP-R
NO ↓
Platelet activation and aggregation
Activated platelet
TNF-α
IL-1β
monocyte
TRLP-R Penetrate
ICAM-1
VCAM-1
NLRP-1
Foam cell
monocyte
Atherosclerotic plaque
Monocyte activation, adhesion and migration
TRLP-R
Lipid loading and foam cell formation
TRLP-R taken up by macrophages
macrophage
Intima

VTM2243413

FIGURE 3 IHD Mortality Rates and Stroke Mortality in Asian Countries



Rối loạn lipid và các cơ chế gây tổn thương mạch máu trên đây Giải thích tỷ lệ tử vong Đột Quỵ cao hơn Bệnh tim thiếu máu

XVĐMCa chiếm tỷ lệ 77,6 % phát hiện qua SA ở 116 Bn ĐTĐ típ 2 . Nguyễn Hải Thủy (1994-1996)

5. Biện pháp can thiệp rối loạn lipid máu ở bệnh nhân người Châu Á

5.1. Thay đổi lối sống

Giảm cân

Hạn chế yếu tố nguy cơ

Giấc ngũ

Liệu pháp dinh dưỡng

Tăng hoạt động thể lực

Dietary Approaches to Stop Hypertension (DASH)
Chế độ ăn hạ huyết áp

- Ngũ cốc: 7-8 phần ăn hàng ngày
- Rau: 4-5 phần ăn hằng ngày
- Trái cây: 4-5 phần ăn hàng ngày
- Các sản phẩm từ sữa ít chất béo hoặc không có chất béo: 2-3 bữa ăn hàng ngày
- Thịt, gia cầm và cá: 6 hoặc ít hơn hàng ngày phần ăn
- Hạt và đậu khô: 4-5 phần ăn mỗi tuần
- Chất béo và dầu: 2-3 phần ăn hàng ngày
- Đồ ngọt: dưới 5 phần ăn mỗi tuần

Chế độ ăn Địa Trung Hải

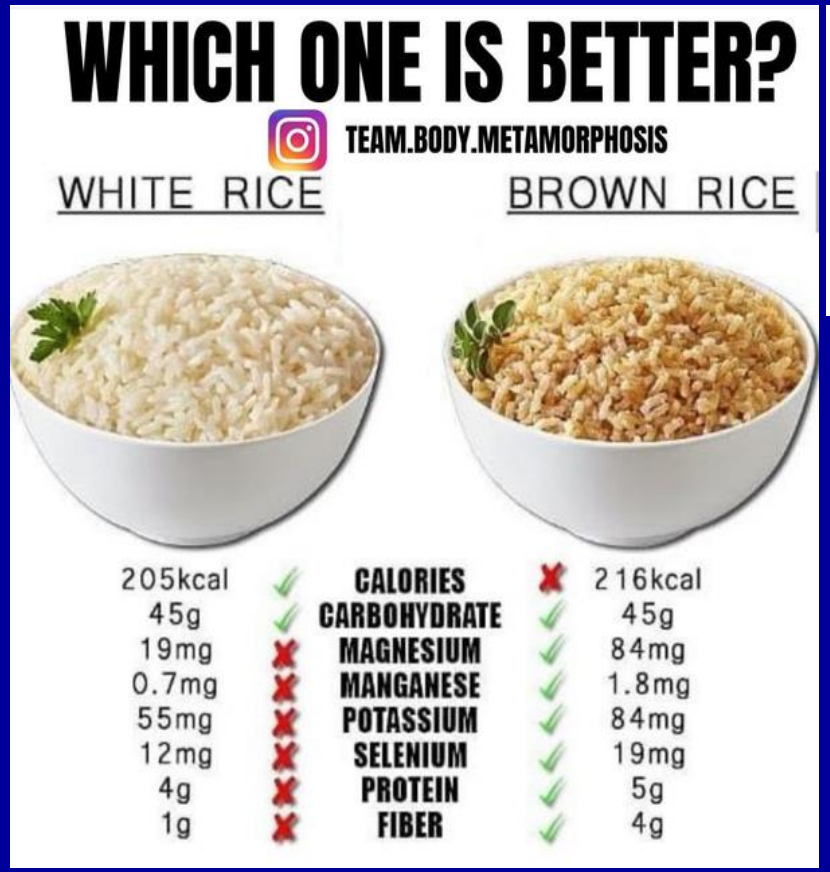
- - Ăn tự nhiên, nên ăn các loại thực phẩm chưa qua chế biến như: hoa quả, rau, ngũ cốc và các loại hạt.
  - Dầu ô liu là nguồn chất béo chính trong chế độ ăn uống.
  - Giảm tiêu thụ thịt đỏ hàng tháng, thay vào là các loại thịt gia cầm.
  - Ăn cá thường xuyên vào mỗi tuần. Tránh các loại cá rán.
  - Uống một lượng rượu vang vừa phải.

Giấc ngũ

- **Nghỉ ngơi đầy đủ là quan trọng cho việc duy trì năng lượng và thoải mái.**
- **Tư vấn về giấc ngủ 6-9 giờ/mỗi đêm (trung bình khoảng 7 giờ).**
- **Có sự liên quan giữa thời gian ngũ với giảm YTNC tim mạch chuyển hóa.**
- **Thiếu ngủ làm trầm trọng thêm tình trạng kháng insulin, THA, tăng đường huyết và RLLP máu và tăng yếu tố viêm cytokine**

A Global Perspective on White Rice Consumption and Risk of Type 2 Diabetes

Diabetes Care 2020;43:2625–2627 | https://doi.org/10.2337/dci20-0042



grown in arsenic-rich areas (19). In U.S. cohorts, replacement of white rice with brown rice was associated with lower diabetes risk, although the risk reduction

Currently, reducing white rice consumption to achieve a more balanced dietary pattern with a variety of whole grains, fruits, vegetables, legumes, nuts, and nontropical vegetable oils can be recommended to reduce the risk of type 2 diabetes and cardiovascular diseases (6,25).

BẢNG QUI ĐỔI TỪ OZ SANG ML(KIỂU MỸ)			
Oz	**ml**	**Oz**	**ml**
2.5	74	**11**	325
3	89	**12**	354
3.5	103	**14**	413
4	118	**16**	472
4.5	133	**18**	531
5	148	**20**	590
6	177	**22**	649
6.5	192	**24**	708
7	207	**30**	885
9	266	**32**	944
10	295	**34**	1003
1oz = 29,5ml			

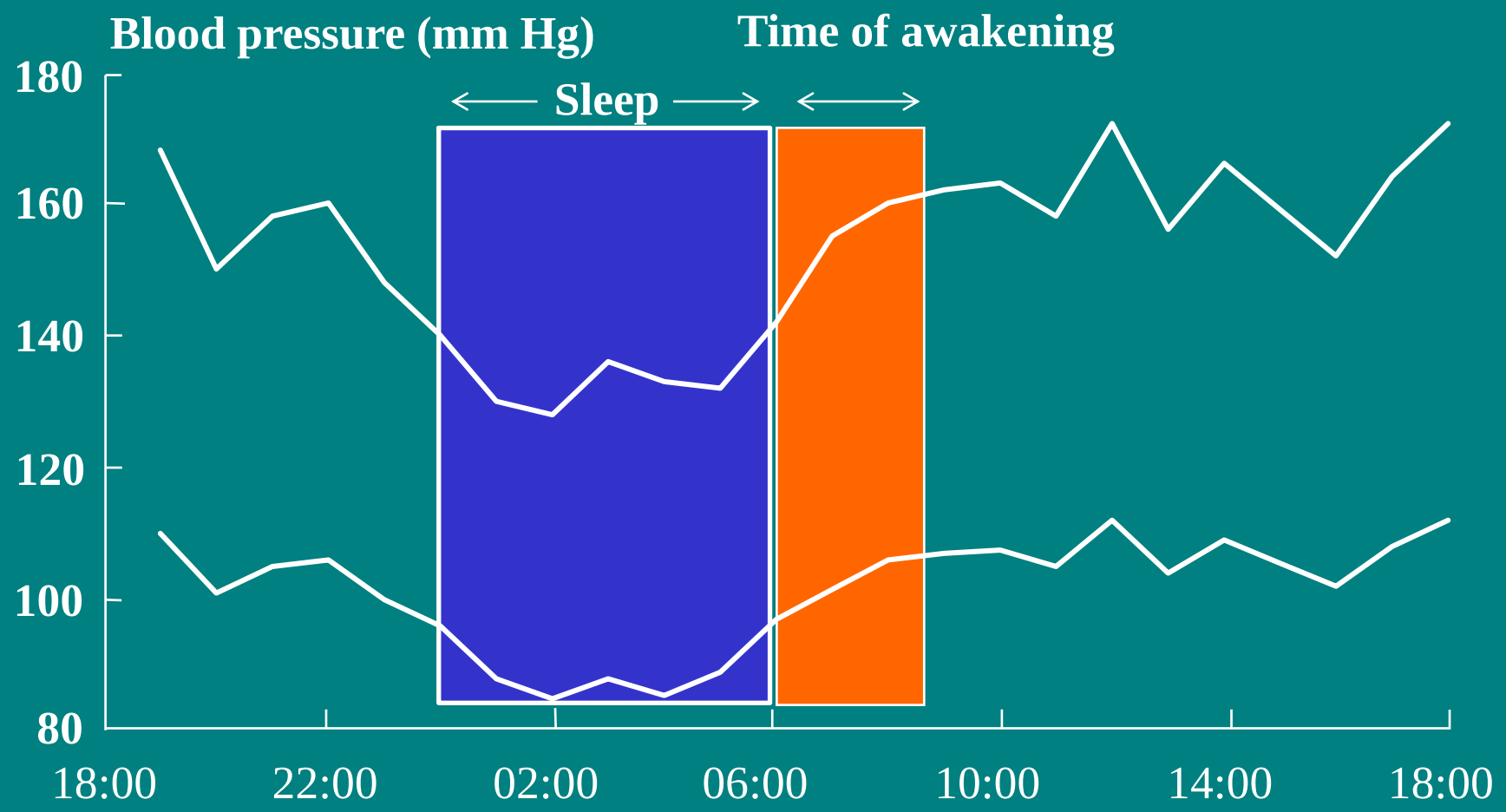
Nam < 20 g alcool/ngày và Nữ < 15 gam alcool/ngày

Nam < 2 drink/ngày và nữ < 1 drink/ngày

1 drink = 12-oz beer = 5-oz rượu = 1.5 oz rượu nặng

Hoạt động thể lực

24-Hour Blood Pressure Profile: The Morning Blood Pressure 'Surge'



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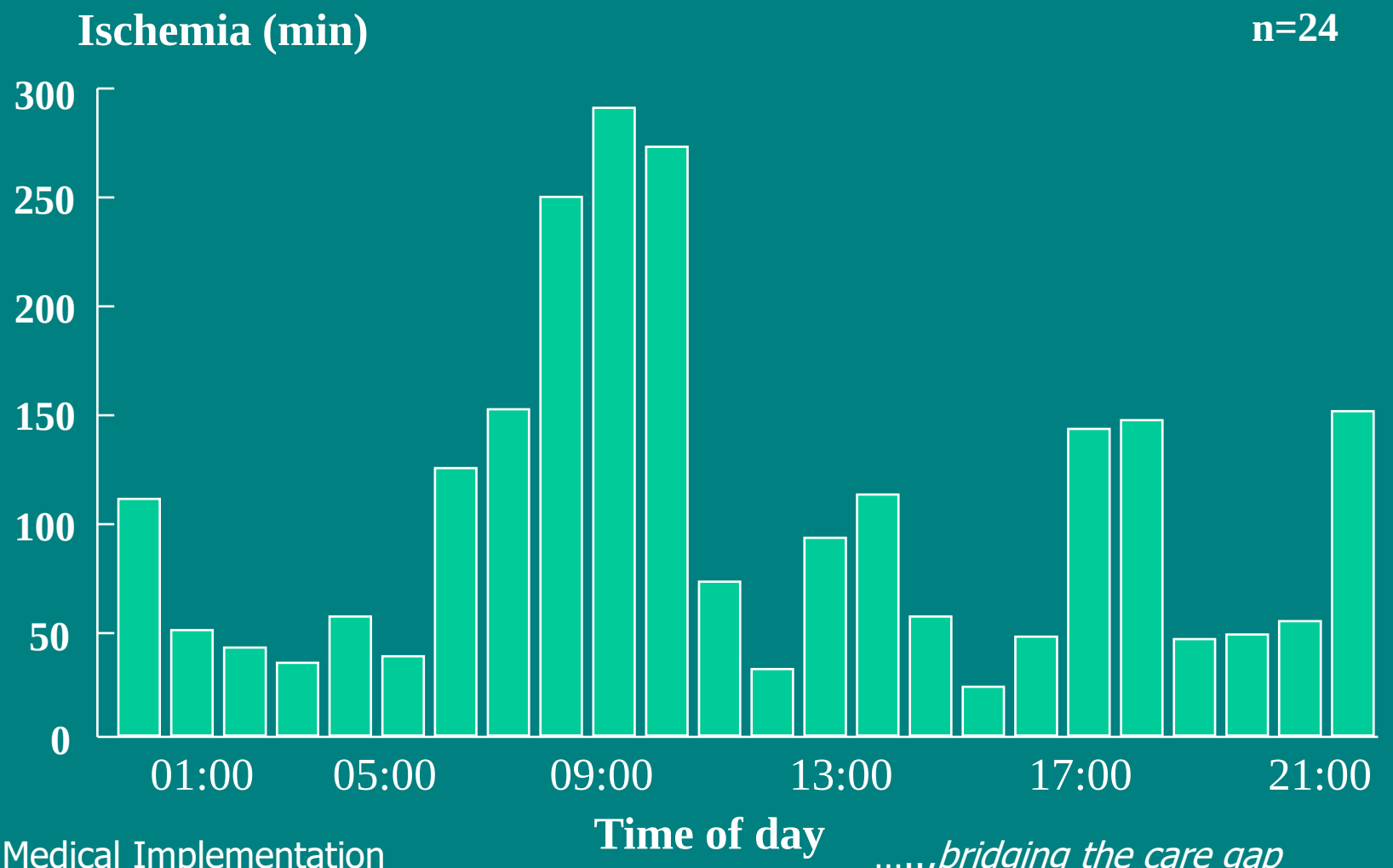
.....bridging the care gap

Adapted from: Millar-Craig, et al. 1978; Mancia, et al. 1983.

Circadian Incidence of Cardiovascular Events: Myocardial Ischemia



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……bridging the care gap

Adapted from: Rocco, et al. 1987.

Glucose Regulation During Exercise & Stress
Role of epinephrine

Exercise or stress

↓

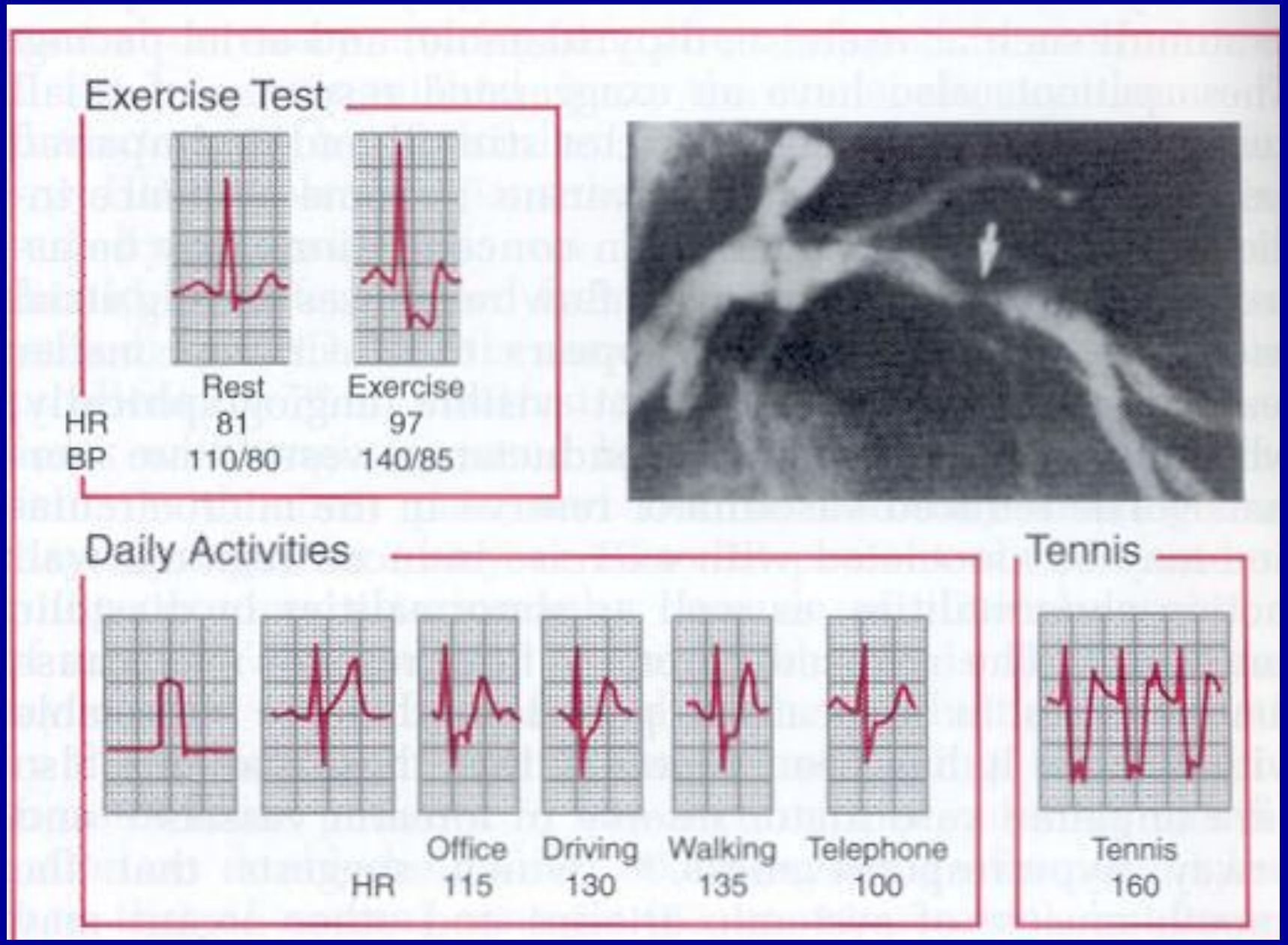
↑ Adrenergic activity

↓

↑Epinephrine

+ α cell → ↑ Glucagon → ↑ Liver glucose output

− β cell → ↓ Insulin → ↓ Glucose uptake


Exercise Test
Rest
Exercise
HR
81
97
BP
110/80
140/85
Daily Activities
Office
Driving
Walking
Telephone
HR
115
130
135
100
Tennis
Tennis
160

Tần số tim tối đa = 220 – tuổi x % thích nghi

Thí dụ : Bệnh nhân 60 tuổi có tần số tim tối đa

220 – 60 = 160 lần/ phút

Tần số tim giao động *(65-85%)*:

Tần số tim tối đa x mục tiêu % = **Mục tiêu tần số tim**

160 x 50% = **80 nhịp/phút**

160 x 70% = **112 nhịp/phút**

220 – Tuổi = Tần số tim tối đa

Age	50% (220-Age) x .5	55% (220-Age) x .55	65% (220-Age) x .65	75% (220-Age) x .75	85% (220-Age) x .85	95% (220-Age) x .95
20	100	110	130	150	170	190
25	97.5	107.25	126.75	146.25	165.75	185.25
30	95	104.5	123.5	142.5	161.5	180.5
35	92.5	101.75	120.25	138.75	157.25	175.75
40	90	99	117	135	153	171
45	87.5	96.25	113.75	131.25	148.75	166.25
50	85	93.5	110.5	127.5	144.5	161.5
55	82.5	90.75	107.25	123.75	140.25	156.75
60	80	88	104	120	136	152
65	77.5	85.25	100.75	116.25	131.75	147.25

5.2. Tham khảo các khuyến cáo liên quan rối loạn lipid máu của các hiệp hội Y học 2021-2023

Khuyến cáo kiểm soát thành phần lipid máu ESC 2021

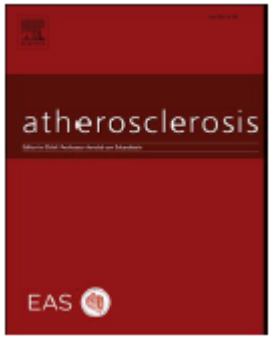
Table 10 Corresponding non-high-density lipoprotein cholesterol and apolipoprotein B levels for commonly used low-density lipoprotein cholesterol goals

LDL-C	Non-HDL-C	Apolipoprotein B
2.6 mmol/L (100 mg/dL)	3.4 mmol/L (131 mg/dL)	100 mg/dL
1.8 mmol/L (70 mg/dL)	2.6 mmol/L (100 mg/dL)	80 mg/dL
1.4 mmol/L (55 mg/dL)	2.2 mmol/L (85 mg/dL)	65 mg/dL

HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

© ESC 2021

Khuyến cáo EAS 2021
Quan tâm đến các Lipoprotein giàu TG (TRLs)



Box 5

Managing elevated TG-rich lipoproteins.

- Elevated TG-rich lipoproteins associate with increased cardiovascular risk, even if patients are at LDL-C goal.
- Contemporary evidence suggests that the atherogenic risk derived from TG-rich particles and their remnants approximates the risk from an LDL particle.
- In type 2 diabetes mellitus patients with elevated TG on LDL-lowering therapy, fenofibrate may be considered for beneficial macro- and microvascular effects.
- High-dose omega-3 fatty acids (icosapent ethyl) may be considered, weighing the benefit versus risks of treatment.

Khuyến cáo lưu ý ngoài biến chứng mạch lớn nên chú ý biến chứng vi mạch

Khuyến cáo sử dụng Fenofibrate và liều cao omega-3 Fatty Acids

Atherosclerosis 325 (2021) 99–109

•Cập nhật theo CCS 2021 CANADA

FRAMINGHAM RISK SCORE (FRS)

Estimation of 10-year Cardiovascular Disease (CVD) Risk

Date: _____

Patient's Name: _____

Step 1[1]

In the "points" column enter the appropriate value according to the patient's age, HDL-C, total cholesterol, systolic blood pressure, and if they smoke or have diabetes. Calculate the total points.

Risk Factor		Risk Points				Points
		Men		Women		
Age						
30-34		0		0		
35-39		2		2		
40-44		5		4		
45-49		7		5		
50-54		8		7		
55-59		10		8		
60-64		11		9		
65-69		12		10		
70-74		14		11		
75+		15		12		
HDL-C (mmol/L)						
>1.6		-2		-2		
1.3-1.6		-1		-1		
1.2-1.29		0		0		
0.9-1.19		1		1		
<0.9		2		2		
Total Cholesterol						
<4.1		0		0		
4.1-5.19		1		1		
5.2-6.19		2		3		
6.2-7.2		3		4		
>7.2		4		5		
Systolic Blood Pressure (mmHg)		Not Treated	Treated	Not Treated	Treated	
<120		-2	0	-3	-1	
120-129		0	2	0	2	
130-139		1	3	1	3	
140-149		2	4	2	5	
150-159		2	4	4	6	
160+		3	5	5	7	
Smoker	Yes	4		3		
	No	0		0		
Diabetes	Yes	statin-indicated condition				
	No	0		0		
Total Points						

1 Adapted from: D'Agostino RB et al.(i). General cardiovascular risk profile for use in primary care. The Framingham Heart Study. Circ 2008;117:743-53.
2 Adapted from: Genest J et al.(i). 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult. Can J Cardiol. 2009;25(10):567-579.
3 Adapted from: Anderson T et al.(i). 2012 Update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia for the prevention of cardiovascular disease in the adult. Can J Cardiol. 2013;29(2):151-167.

† apoB: apolipoprotein B stat, CVD: cardiovascular disease, FRS: Framingham Risk Score, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol.
* Statins indicated as initial therapy
** Consider LDL-C < 1.8 mmol/L for subjects with acute coronary syndrome (ACS) within past 3 months

Provided courtesy of **Canadian Cardiovascular Society**
Leadership. Knowledge. Community.

Step 2[1]

Using the total points from Step 1, determine the 10-year CVD risk (%).*

Total Points	10-Year CVD Risk (%)*	
	Men	Women
-3 or less	<1	<1
-2	1.1	<1
-1	1.4	1.0
0	1.6	1.2
1	1.9	1.5
2	2.3	1.7
3	2.8	2.0
4	3.3	2.4
5	3.9	2.8
6	4.7	3.3
7	5.6	3.9
8	6.7	4.5
9	7.9	5.3
10	9.4	6.3
11	11.2	7.3
12	13.3	8.6
13	15.6	10.0
14	18.4	11.7
15	21.6	13.7
16	25.3	15.9
17	29.4	18.51
18	>30	21.5
19	>30	24.8
20	>30	27.5
21+	>30	>30

Step 3[1]

Using the total points from Step 1, determine heart age (in years).

Heart Age, y	Men	Women
<30	<0	<1
30	0	
31		1
32	1	
34	2	2
36	3	3
38	4	
39		4
40	5	
42	6	5
45	7	6
48	8	7
51	9	8
54	10	
55		9
57	11	
59		10
60	12	
64	13	11
68	14	12
72	15	
73		13
76	16	
79		14
>80	≥17	15+

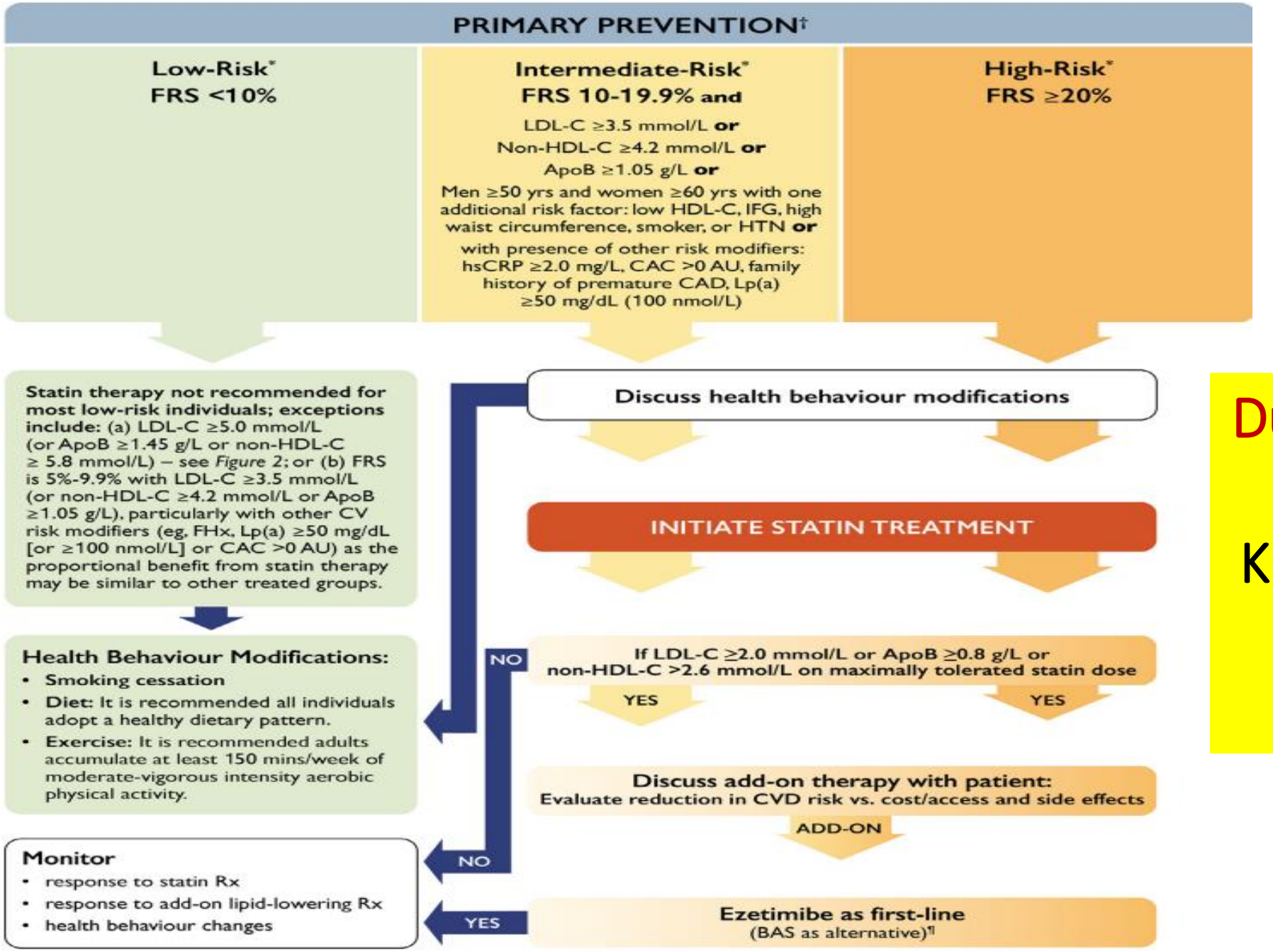
* Double cardiovascular disease risk percentage for individuals between the ages of 30 and 59 without diabetes if the presence of a positive history of premature cardiovascular disease is present in a first-degree relative before 55 years of age for men and before 65 years of age for women. This is known as the modified Framingham Risk Score.[3]

Step 4[2,3]

Using 10-year CVD risk from Step 2, determine if patient is Low, Moderate or High risk.† Indicate Lipid and/or Apo B targets

Risk Level†	Initiate Treatment If:	Primary Target (LDL-C)	Alternate Target
High FRS ≥20%	• Consider treatment in all *(Strong, High)*	• ≤2 mmol/L or ≥50% decrease in LDL-C *(Strong, Moderate)*	• Apo B ≤0.8 g/L or • Non-HDL-C ≤2.6 mmol/L *(Strong, High)*
Intermediate FRS 10-19%	• LDL-C ≥3.5 mmol/L *(Strong, Moderate)* • For LDL-C <3.5 mmol/L consider if: • Apo B ≥1.2 g/L • OR Non-HDL-C ≥4.3 mmol/L *(Strong, Moderate)* • *Men ≥50 and women ≥60 with 1 risk factor: low HDL-C, impaired fasting glucose, high waist circumference, smoker, hypertension*	• ≤2 mmol/L or ≥50% decrease in LDL-C *(Strong, Moderate)*	• Apo B ≤0.8 g/L or • Non-HDL-C ≤2.6 mmol/L *(Strong, Moderate)*
Low FRS <10%	• statins generally not indicated	• statins generally not indicated	• statins generally not indicated
Statin-indicated conditions**	• *Clinical atherosclerosis** • *Abdominal aortic aneurysm* • *Diabetes mellitus* *Age ≥ 40 years* *15-Year duration for age ≥ 30 years (DM1) Microvascular disease* • *Chronic kidney disease (age ≥ 50 years) eGFR <60 mL/min/1.73 m2 or ACR > 3 mg/mmol*		

Lipid targets LDL-C: _____ or Apo B: _____



Dự phòng tiên phát
Ngoài LDL.C
Khuyến cáo đề cập
ApoB và
Non HDL.C

Patients with Atherosclerotic Cardiovascular Disease (ASCVD)
Receiving maximally tolerated statin dose

If LDL-C is ≥1.8 mmol/L or if ApoB ≥0.70 g/L or if non-HDL-C ≥2.4 mmol/L**

If TG is ≥1.5 to 5.6 mmol/L

LDL-C 1.8-2.2 mmol/L or ApoB 0.70-0.80 g/L or non-HDL-C 2.4-2.9 mmol/L

LDL-C >2.2 mmol/L or ApoB >0.80 g/L or non-HDL-C >2.9 mmol/L or high PCSK9i benefit patient*

Consider
ezetimibe ± PCSK9 inhibitor

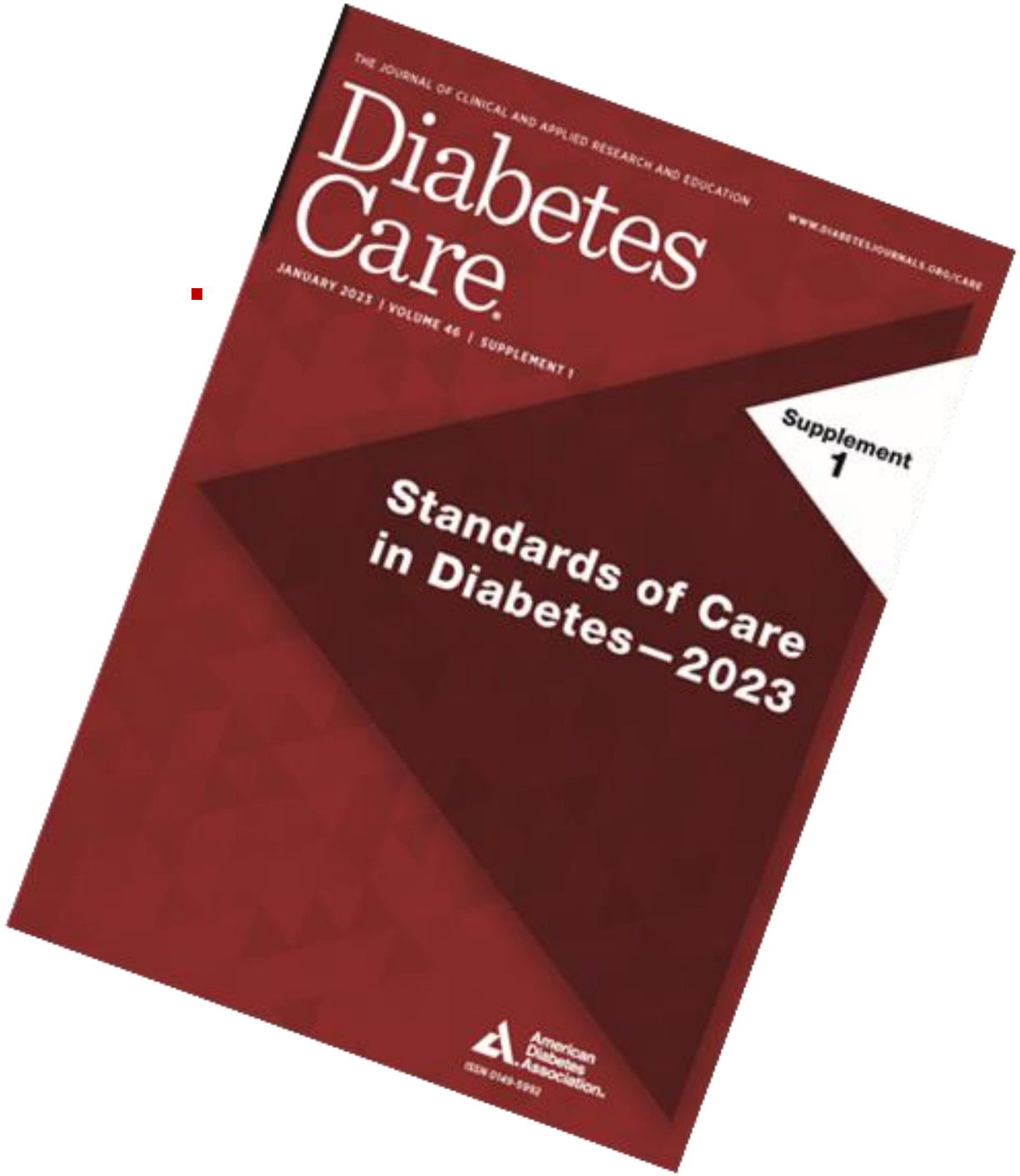
Consider
PCSK9 inhibitor ± ezetimibe

Consider
Icosapent ethyl 2000 mg BID†

*Patients shown to derive largest benefit form intensification of statin therapy with PCSK9 inhibitor therapy are identified in Table 3.
**At low levels of LDL-C or non-HDL-C, measurement of apoB is more accurate than other markers.

†May also be considered for patients without ASCVD but with DM requiring medication treatment in patient ≥50 years of age, and ≥1 additional CV risk factor (*from REDUCE-IT*[166]):

- *men ≥55 y and women ≥65 y;*
- *cigarette smoker or stopped smoking within 3 months;*
- *hypertension (≥140 mmHg systolic OR ≥90 mmHg diastolic) or on BP medication;*
- *HDL-C ≤1.04 mmol/L for men or ≤1.3 mmol/L for women;*
- *hsCRP >3.0 mg/L;*
- *Renal dysfunction eGFR >30 and <60 mL/min;*
- *Retinopathy;*
- *Micro- or macroalbuminuria;*
- *ABI <0.9 without symptoms of intermittent claudication)*


THE JOURNAL OF CLINICAL AND APPLIED RESEARCH AND EDUCATION
Diabetes Care
WWW.DIABETESJOURNALS.ORG/CARE
JANUARY 2023 | VOLUME 46 | SUPPLEMENT 1
Supplement 1
Standards of Care in Diabetes—2023
American Diabetes Association.

Lipid Management—Lifestyle Intervention

10.14 Lifestyle modification focusing on weight loss (if indicated); application of a Mediterranean or Dietary Approaches to Stop Hypertension (DASH) eating pattern; reduction of saturated fat and trans fat; increase of dietary n-3 fatty acids, viscous fiber, and plant stanols/sterols intake; and increased physical activity should be recommended to improve the lipid profile and reduce the risk of developing atherosclerotic cardiovascular disease in people with diabetes. A

Statin Treatment—Primary Prevention

10.18 For people with diabetes aged 40–75 years without atherosclerotic cardiovascular disease, use moderate-intensity statin therapy in addition to lifestyle therapy. A

10.20 For people with diabetes aged 40–75 at higher cardiovascular risk, including those with one or more atherosclerotic cardiovascular disease risk factors, it is recommended to use high-intensity statin therapy to reduce LDL cholesterol by ≥50% of baseline and to target an LDL cholesterol goal of <70 mg/dL. B

10.23 In adults with diabetes aged >75 years, it may be reasonable to initiate moderate-intensity statin therapy after discussion of potential benefits and risks. B

10.24 Statin therapy is contraindicated in pregnancy. B

Statin Treatment—Secondary Prevention

10.25 For people of all ages with diabetes and atherosclerotic cardiovascular disease, highintensity statin therapy should be added to lifestyle therapy. A

10.26 For people with diabetes and atherosclerotic cardiovascular disease, treatment with highintensity statin therapy is recommended to target an LDL cholesterol reduction of ≥50% from baseline and an LDL cholesterol goal of <55 mg/dL. Addition of ezetimibe or a PCSK9 inhibitor with proven benefit in this population is recommended if this goal is not achieved on maximum tolerated statin therapy. **B**

Table 10.2—High-intensity and moderate-intensity statin therapy*

High-intensity statin therapy (lowers LDL cholesterol by ≥50%)	Moderate-intensity statin therapy (lowers LDL cholesterol by 30–49%)
Atorvastatin 40–80 mg	Atorvastatin 10–20 mg
Rosuvastatin 20–40 mg	Rosuvastatin 5–10 mg
	Simvastatin 20–40 mg
	Pravastatin 40–80 mg
	Lovastatin 40 mg
	Fluvastatin XL 80 mg
	Pitavastatin 1–4 mg

*Once-daily dosing. XL, extended release.

Cardiovascular Disease and Risk Management:
Standards of Care in Diabetes - 2023. *Diabetes Care* 2023;46(Suppl. 1):S158-S190

Treatment of Other Lipoprotein Fractions or Targets

10.30 In individuals with atherosclerotic cardiovascular disease or other cardiovascular risk factors on a statin with controlled LDL cholesterol but elevated triglycerides (135–499 mg/dL), the addition of icosapent ethyl can be considered to reduce cardiovascular risk. A

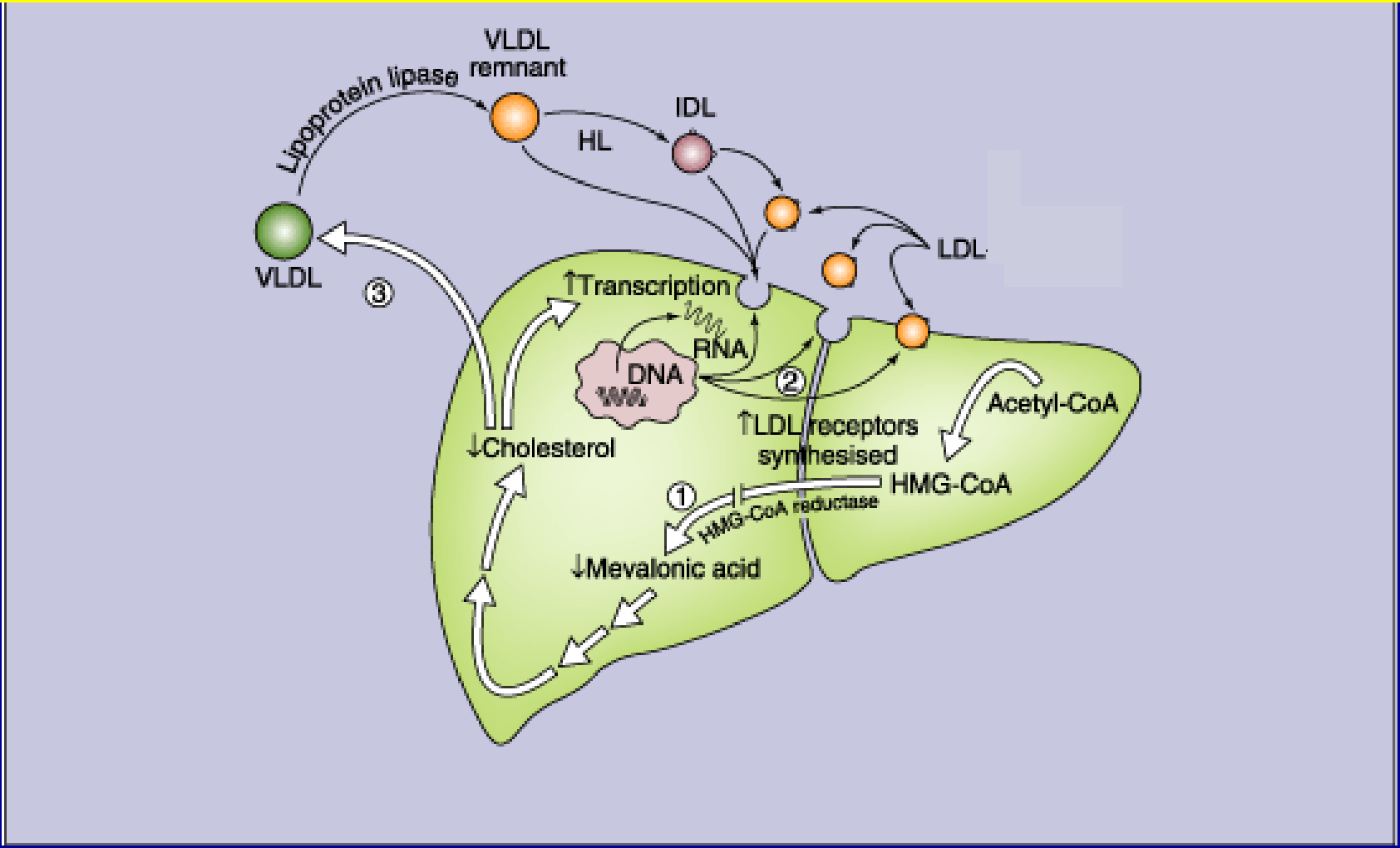
Other Combination Therapy

10.31 Statin plus fibrate combination therapy has not been shown to improve atherosclerotic cardiovascular disease outcomes and is generally not recommended. A

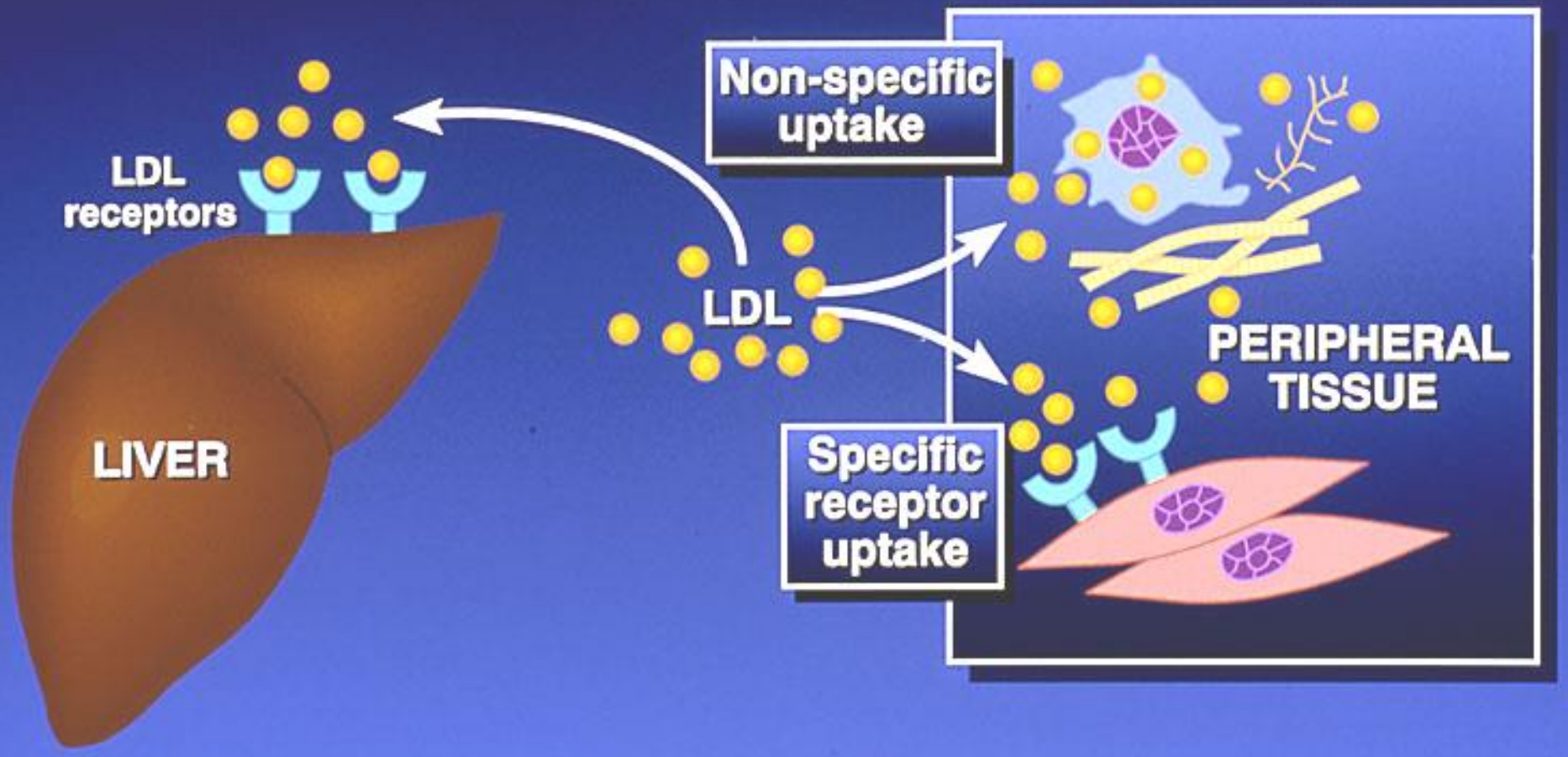
10.32 Statin plus niacin combination therapy has not been shown to provide additional cardiovascular benefit above statin therapy alone, may increase the risk of stroke with additional side effects, and is generally not recommended. A

6. Chọn thuốc Điều Trị RLLP máu dựa theo cơ chế bệnh sinh

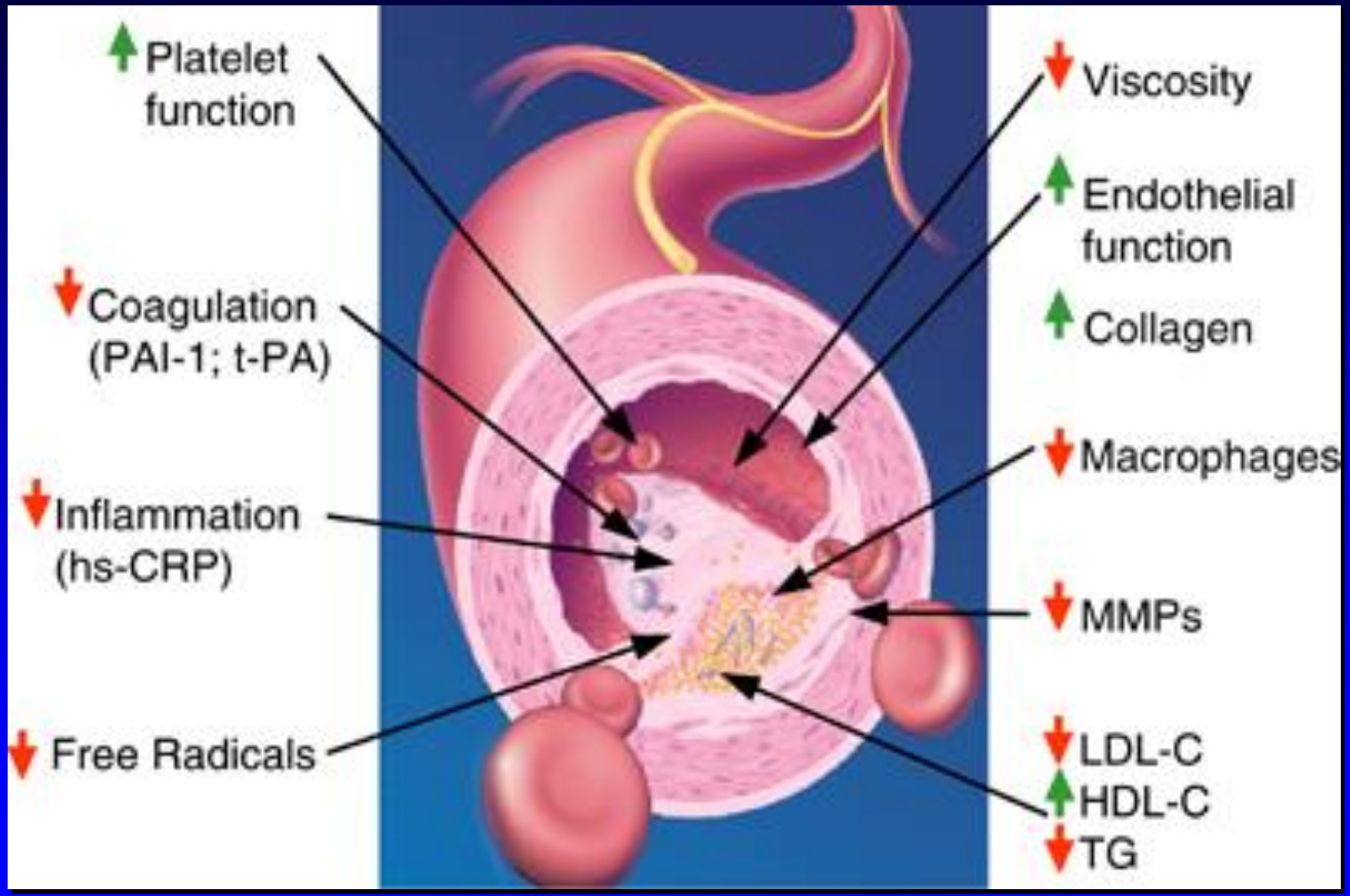
Nhóm ức chế tổng hợp cholesterol và tăng thụ thể LDL
1. Statins



LIPOPROTEIN PATHWAYS
Endogenous
(LDL Uptake)



Tác dụng của statin

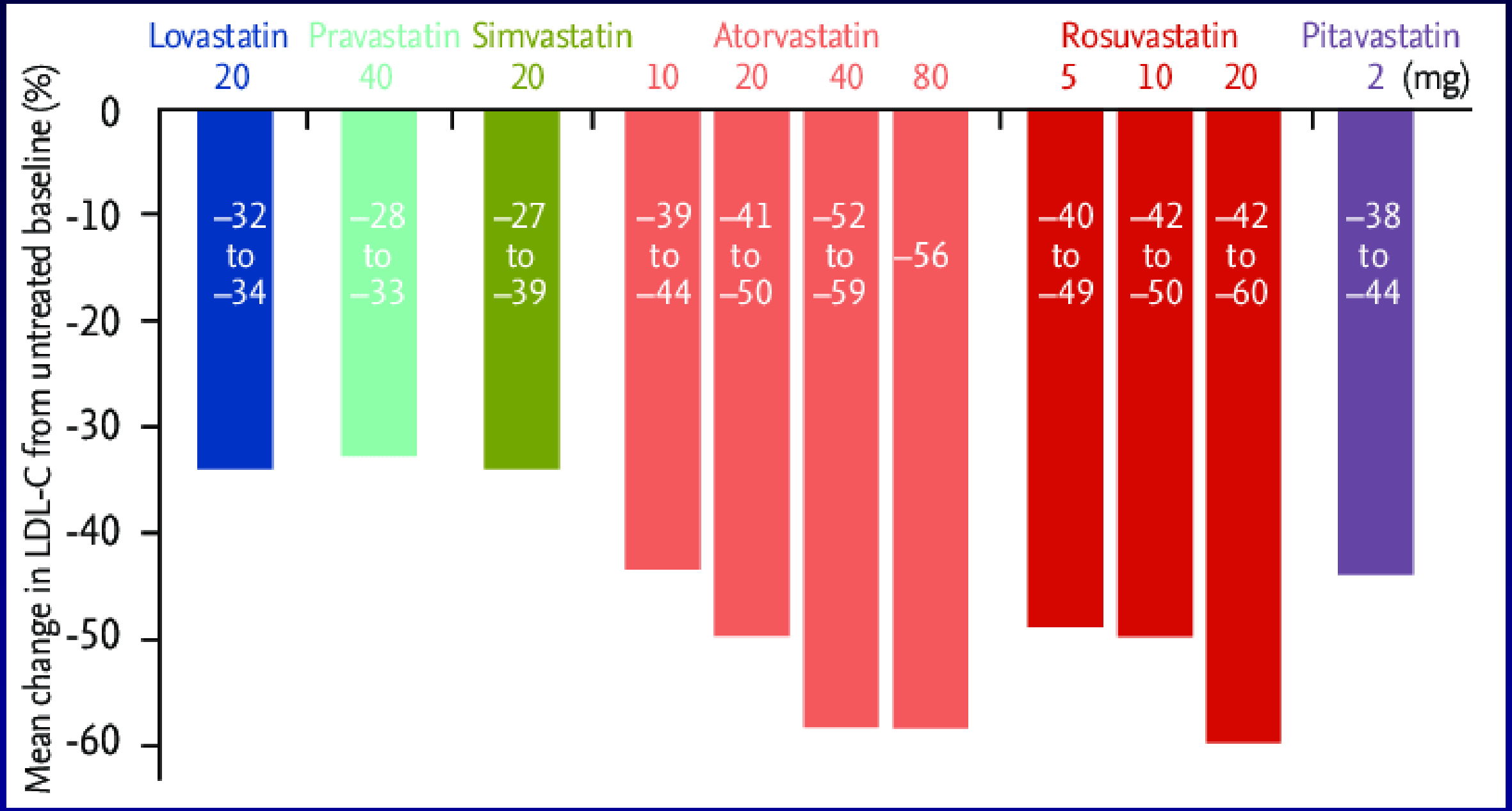


Classification by solubility	Medicines
Water soluble (hydrophilic)	pravastatin, pitavastatin , Rosuvastatin
Fat soluble (lipophilic)	Atorvastatin, fluvastatin, Lovastatin, simvastatin.

Generation statins	**Drugs**
The first (FGS)	pravastatin,lovastatin, fluvastatin
The second (SGS)	Atorvastatin, simvastatin
The Third (TGS)	Rosuvastatin, pitavastatin

Classification by solubility	Medicines
Water soluble (hydrophilic)	Pravastatin, Rosuvastatin
Fat soluble (lipophilic)	Atorvastatin, Fluvastatin, Lovastatin, Simvastatin.

	Lovastatin	Fluvastatin	Pitavastatin	Simvastatin	Atorvastatin	Rosuvastatin	Pravastatin
Dose range (mg/daily)	10–80	20–80	1–4	5–40	10–80	5–40	20–80
Bioavailability (%)	<5	6	>60	<5	12	20	17
Active metabolites	Yes	No	No	Yes	Yes	Yes (minimal)	No
Protein binding (%)	>95	98	96	95	≥90	89	50
Half-life (hours)	2	4.7	12	1–2	14	19	1–2
Faecal excretion (%)	83	90	75	58	90	90	71
Renal excretion (%)	10	<6	2	13	<2	10	20
Liver metabolisation	CYP450 3A4	CYP450 2C9 (minor)	CYP450 2C9	CYP450 3A4	CYP450 3A4	CYP450 2C9 and 2C19	Sulphation
Solubility	Lipophilic	Lipophilic	Lipophilic	Lipophilic	Lipophilic	Hydrophilic	Hydrophilic



Comparison of low density lipoprotein cholesterol (LDL-C) reduction effects of statins between foreigners and Koreans . Modified from Cholesterol Treatment Trialists' Collaboration et al. [2018].

2. Bempedoic acid

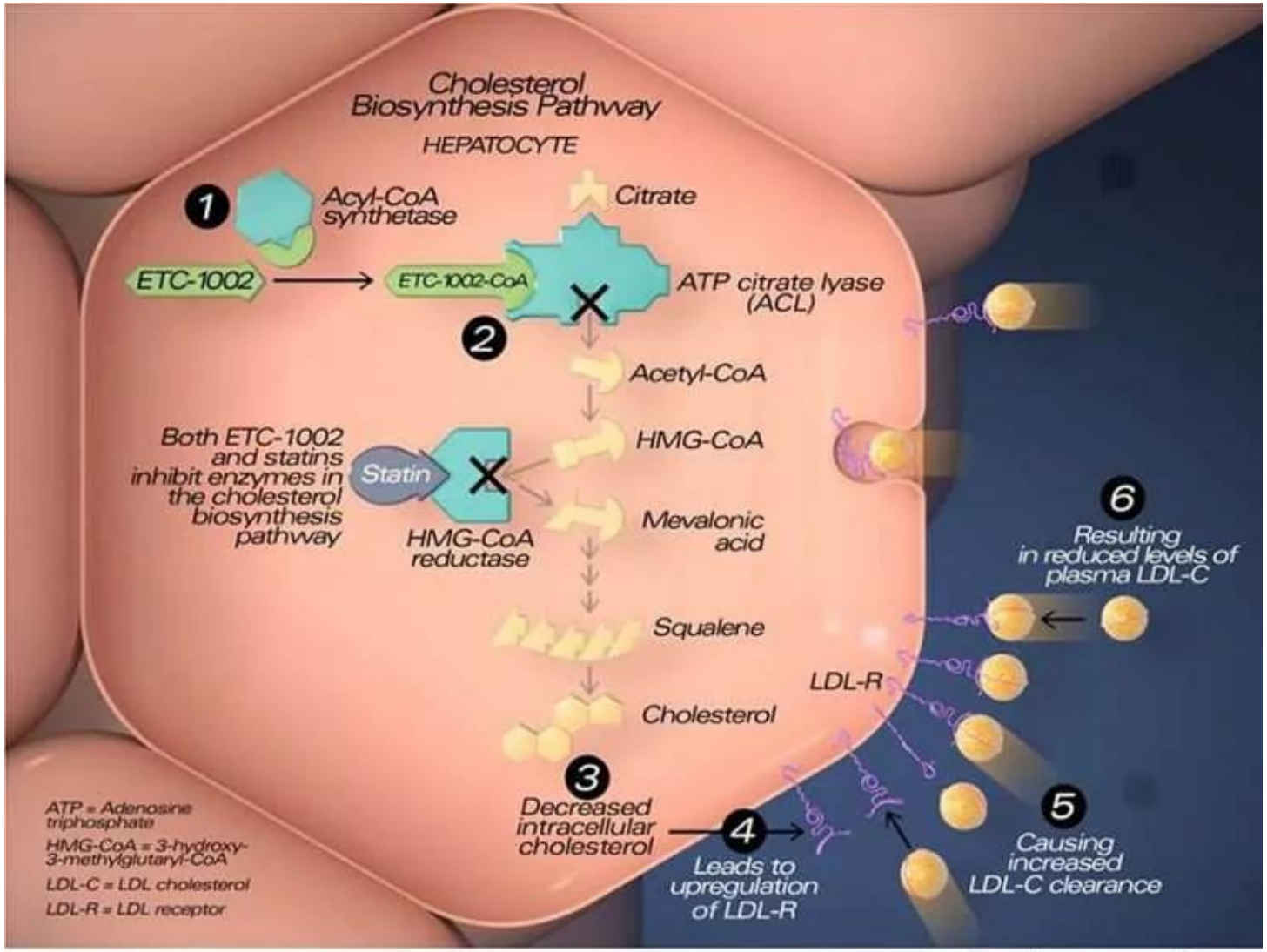


Image from www.esperion.com

Bempedoic acid làm giảm nồng độ cholesterol trong máu bằng cách giảm tổng hợp cholesterol trong gan.

Bempedoic acid

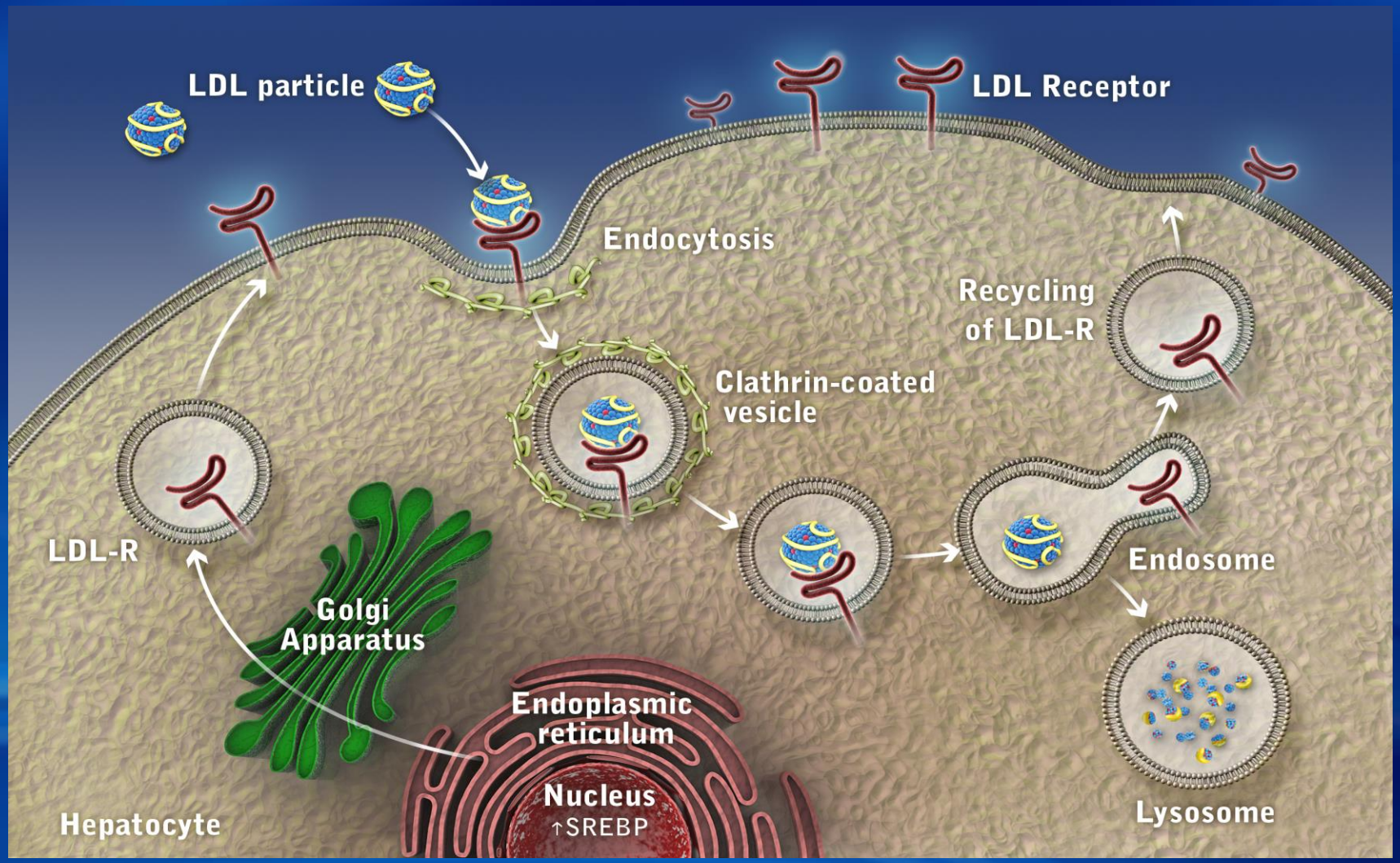
- ức chế enzym ATP citrate lyase tại gan chịu trách nhiệm tạo ra cholesterol.

- làm tăng thụ thể LDL

2. Nhóm tăng thụ thể LDL tại gan

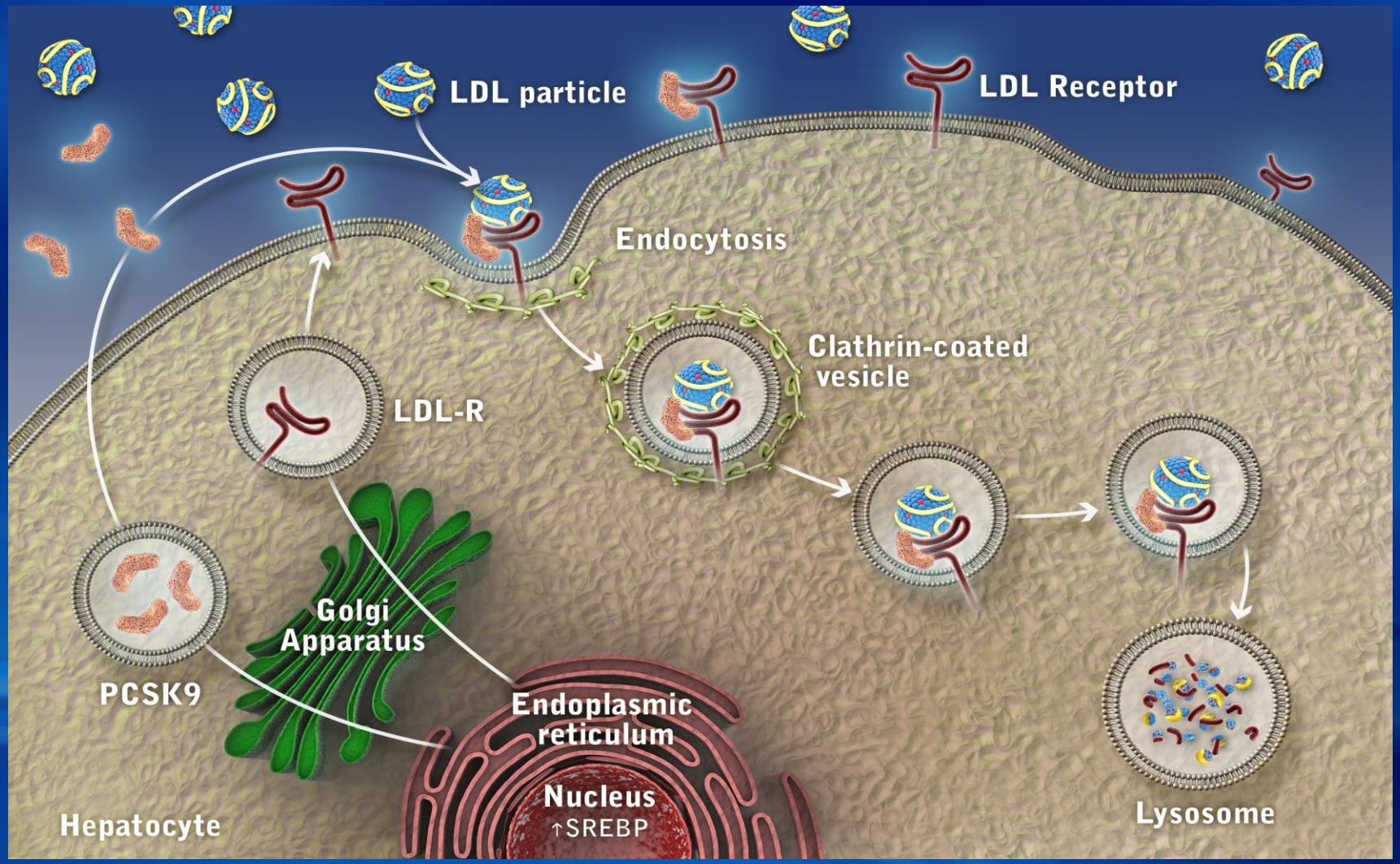
Monoclonal antibody inhibitors of Proprotein Convertase Subtilisin–Kexin type 9 (PCSK9)

Chức năng thụ thể LDL và chu kỳ đời sống (Life Cycle)



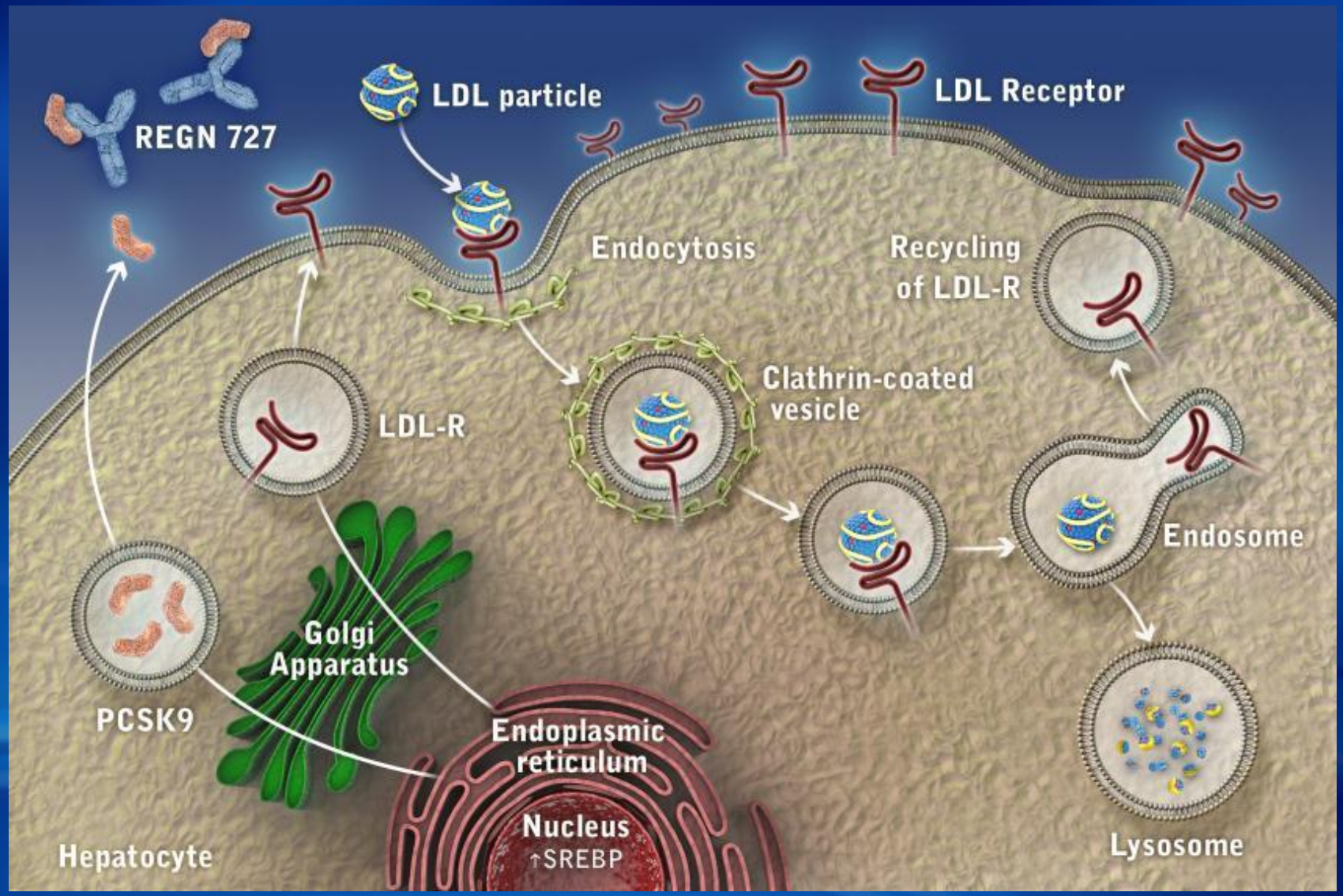
100

Vai trò PCSK9 trong điều hòa sự trình diện thụ thể LDL



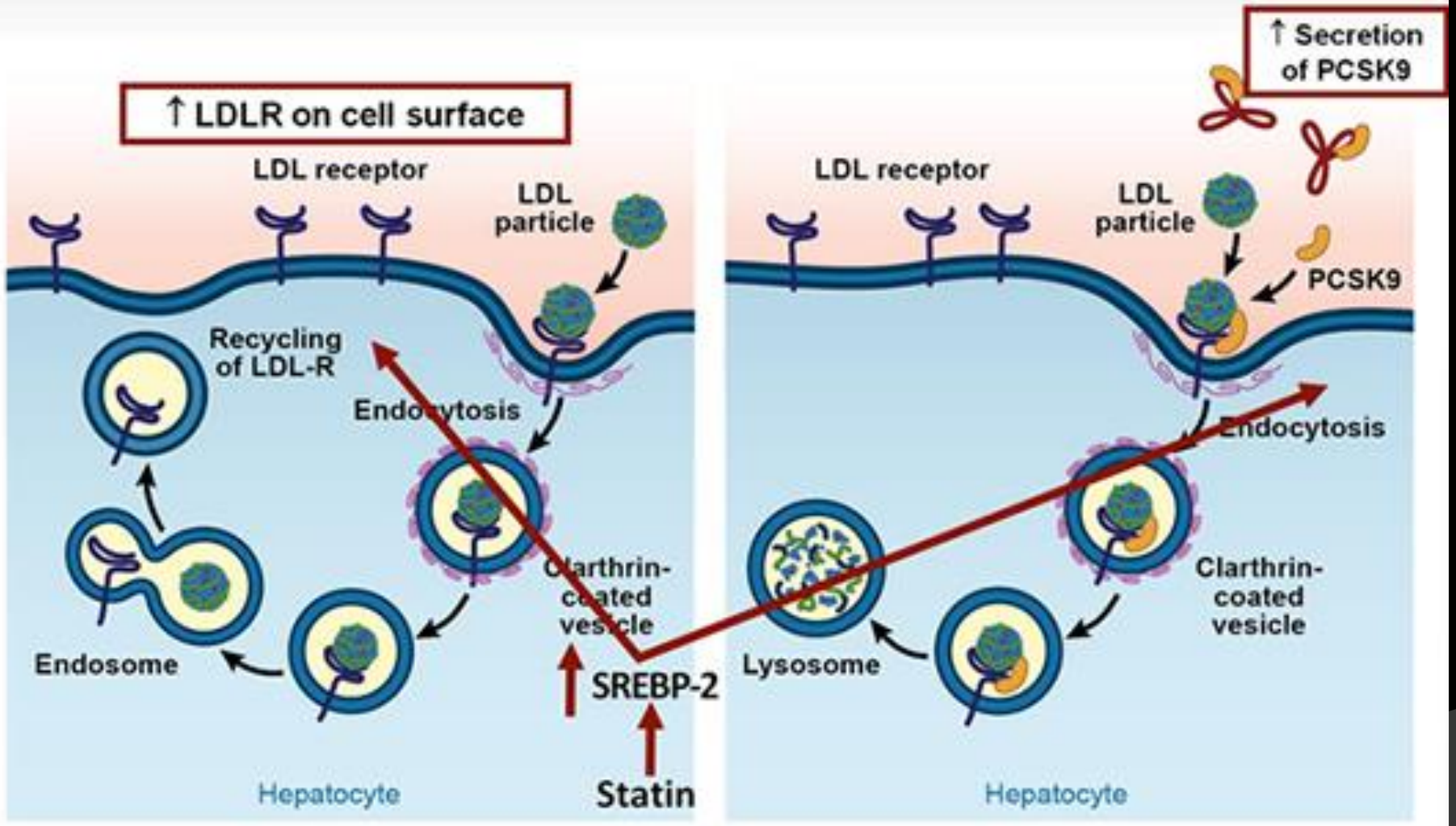
101

Cơ chế tác dụng chất ức chế PCSK9 mAb trên sự trình diện thụ thể LDL

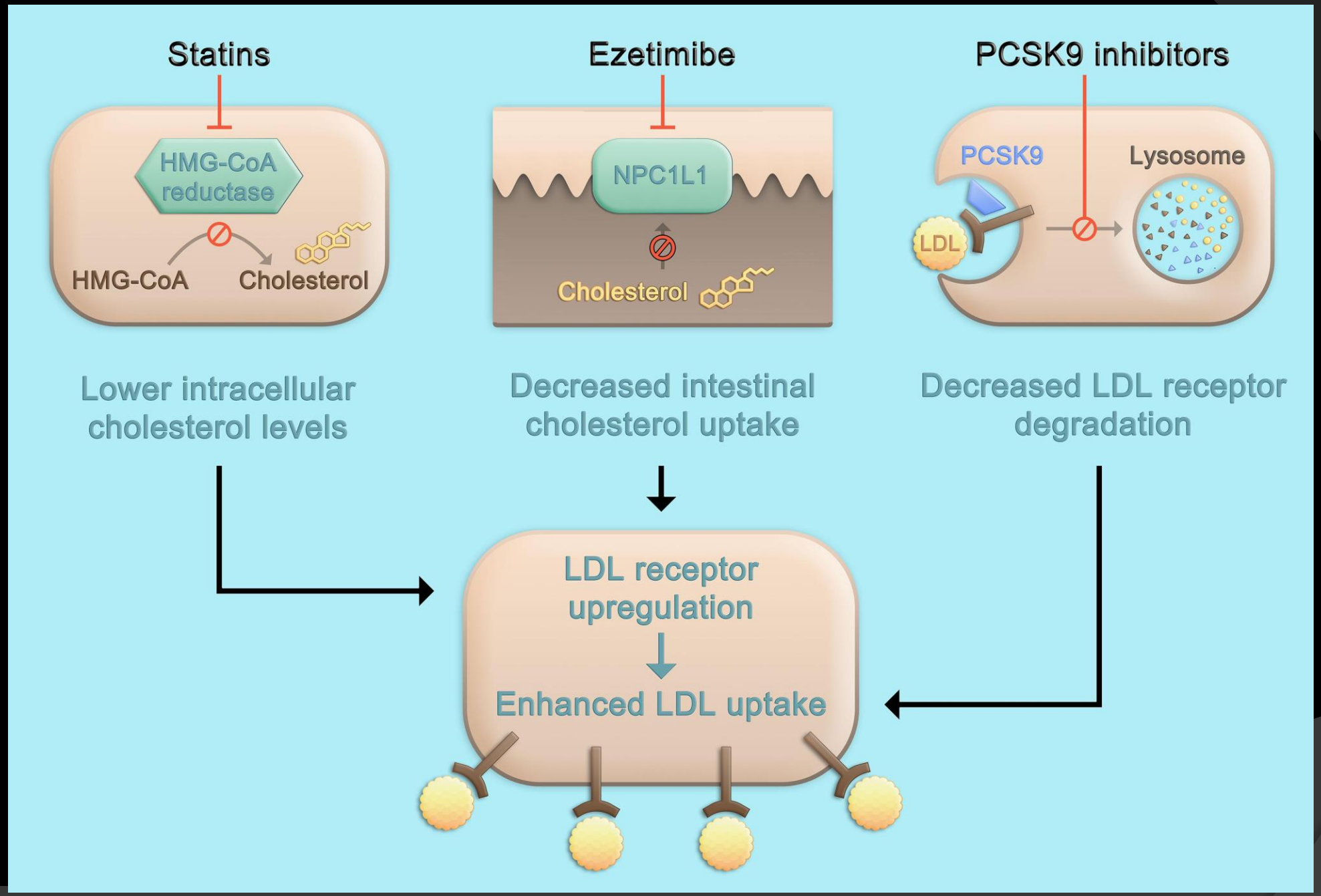


102

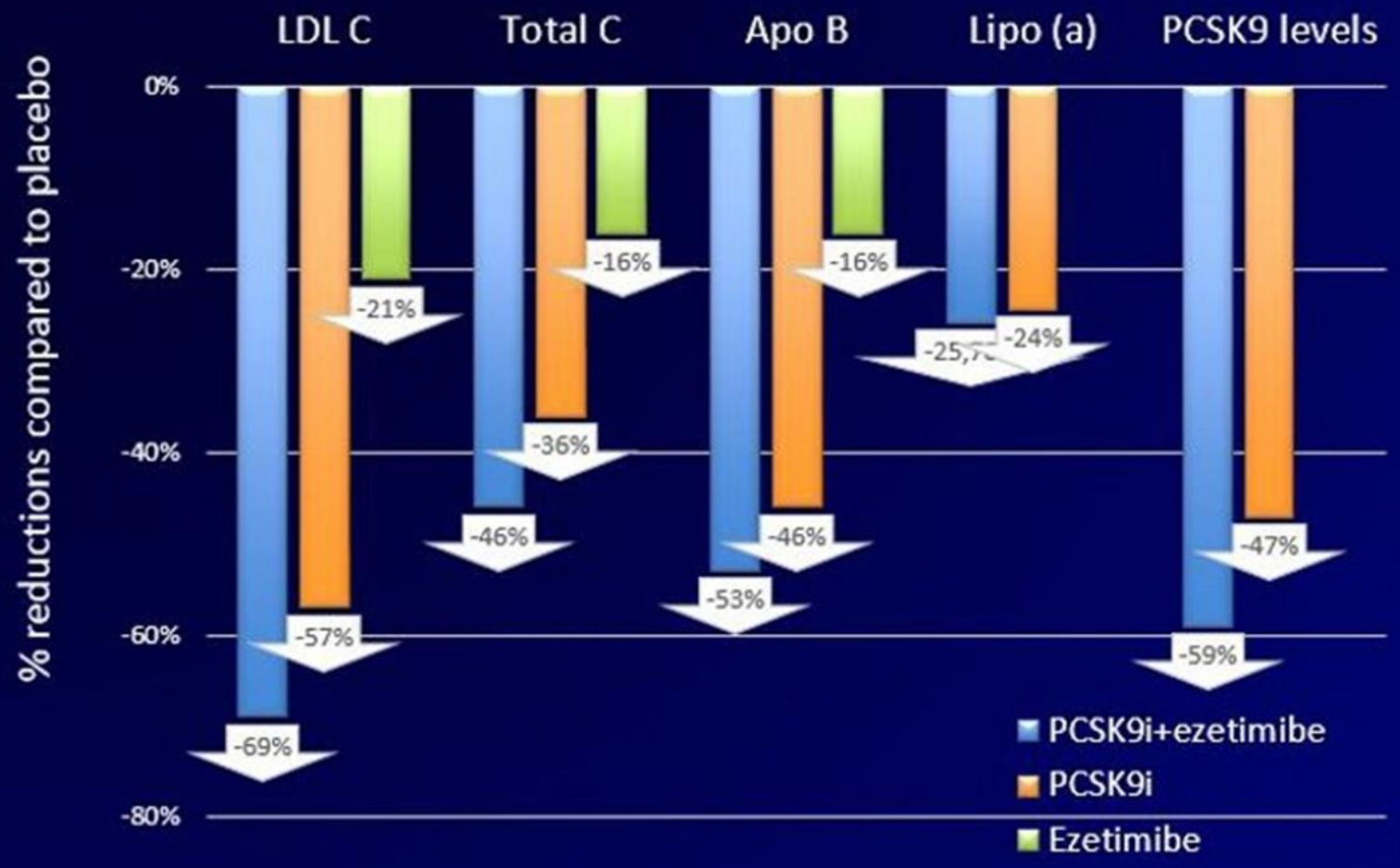
Phối hợp Statin và ức chế PCSK9



Lambert G, et al. *J. Lipid Res.* 2012;53:2515-2524.[6]


Statins
HMG-CoA reductase
HMG-CoA
Cholesterol
Lower intracellular cholesterol levels
Ezetimibe
NPC1L1
Cholesterol
Decreased intestinal cholesterol uptake
PCSK9 inhibitors
PCSK9
Lysosome
LDL
Decreased LDL receptor degradation
LDL receptor upregulation
Enhanced LDL uptake

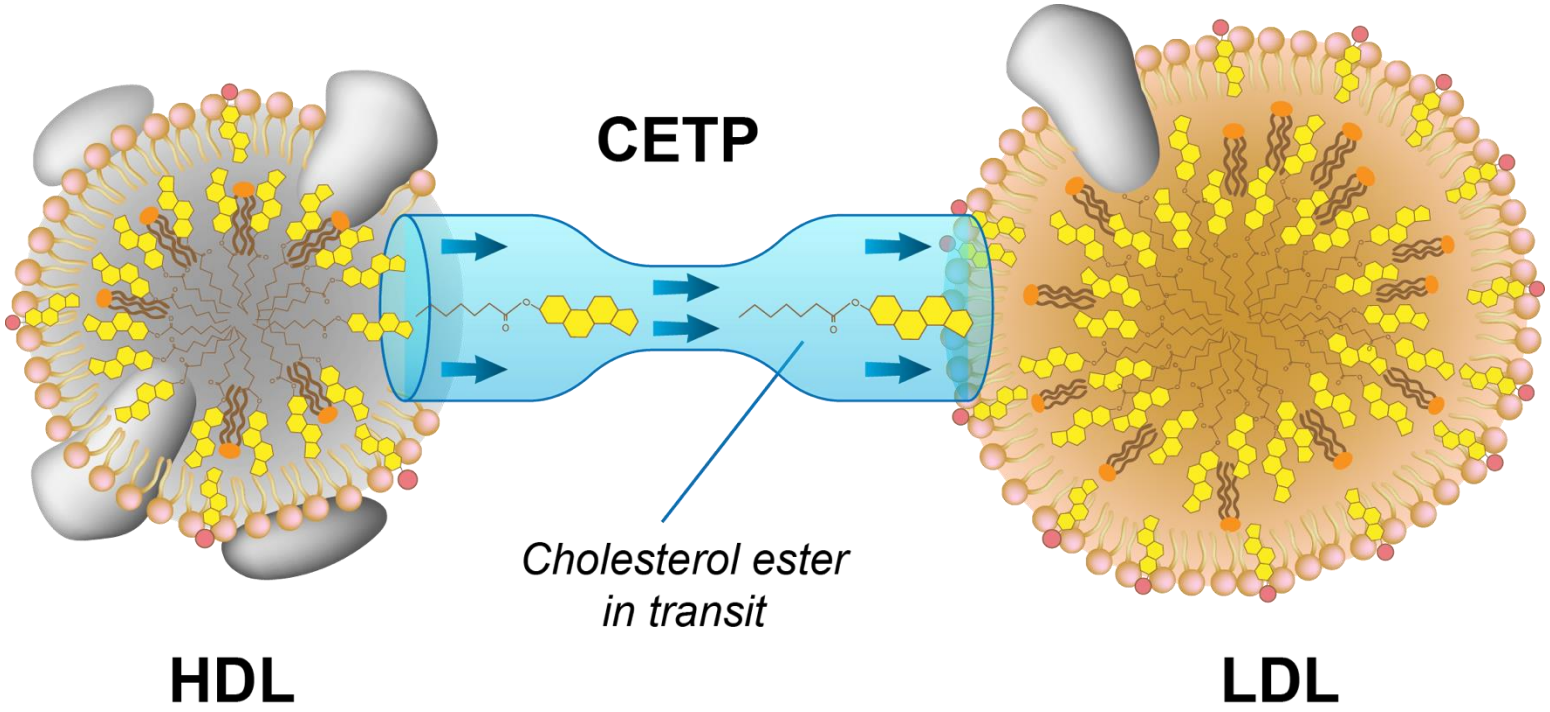
Effect of treatment with PCSK9 inhibition, ezetimibe, or both on 5 lipid measures

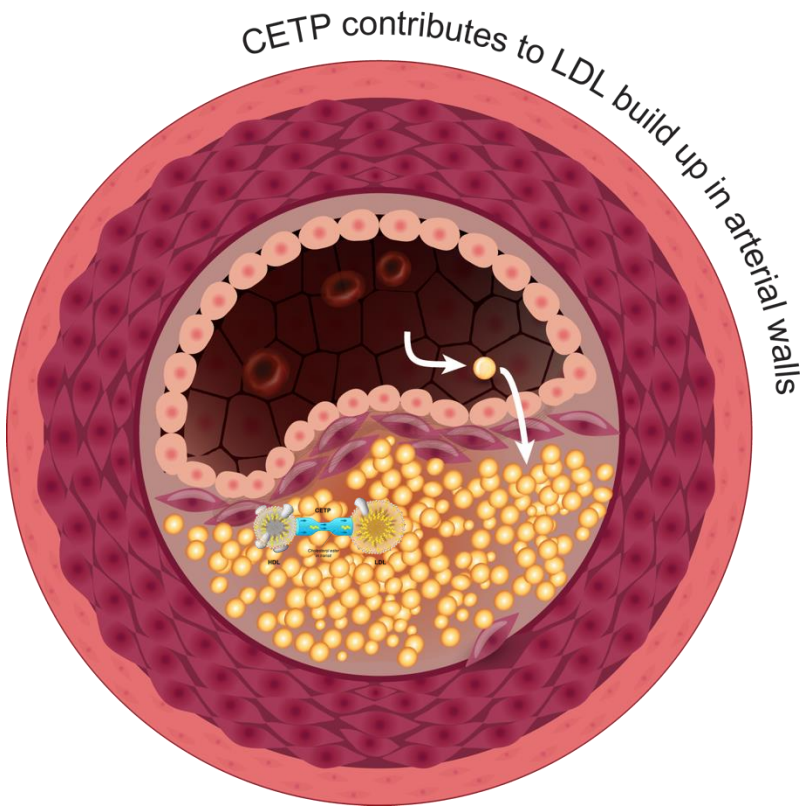


Based on Lipinski *et al.*, Eur Heart J. 2016

VTM2243413

3. Nhóm Ức chế Chuyển Giao cholesterol cho LDL
Cholesteryl ester transfer protein (CETP.I)





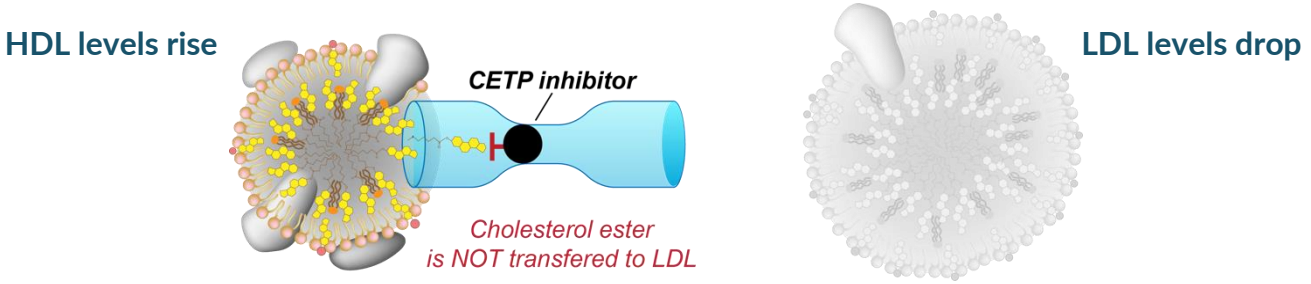
CETP transfers cholesterol esters from HDL to LDL

Cholesteryl ester transfer protein (CETP) promotes the transfer of cholesterol esters from ***anti-atherogenic HDLs*** to ***pro-atherogenic LDLs***, causing LDL-C to build up in the walls of arteries

Biology: CETP inhibition has multiple actions that are beneficial for CHD risk

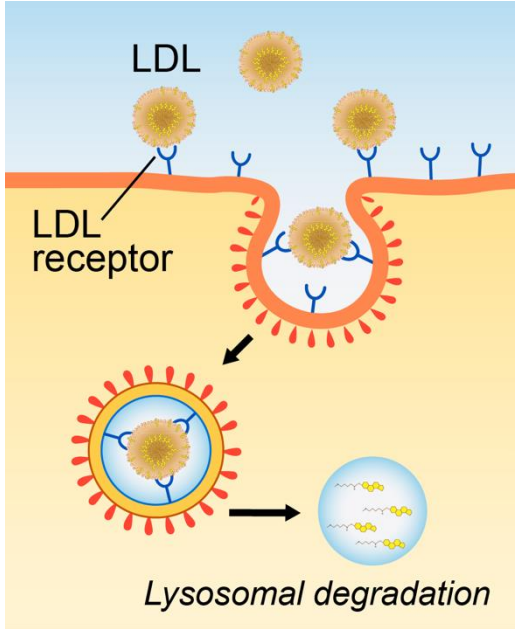
1 In Circulation

CETP inhibition ***blocks transportation of cholesterol esters*** from HDL > LDL, lowering LDL and increasing HDL



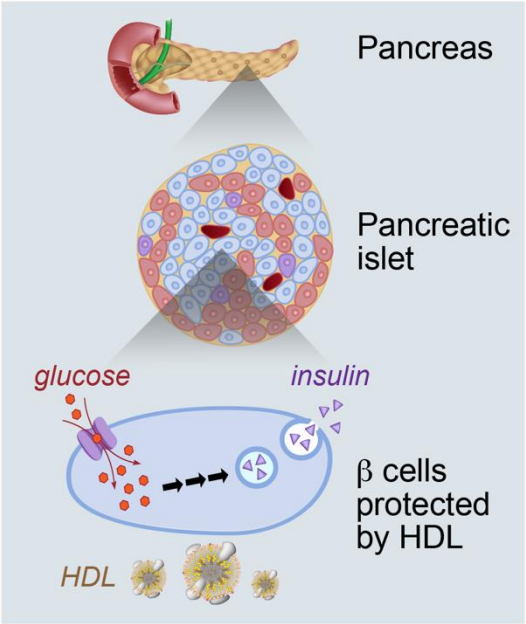
2 In Liver

CETP inhibition also upregulates LDL catabolism in the liver by causing an ***increase in LDL receptors***, resulting in drop in LDL levels in circulation



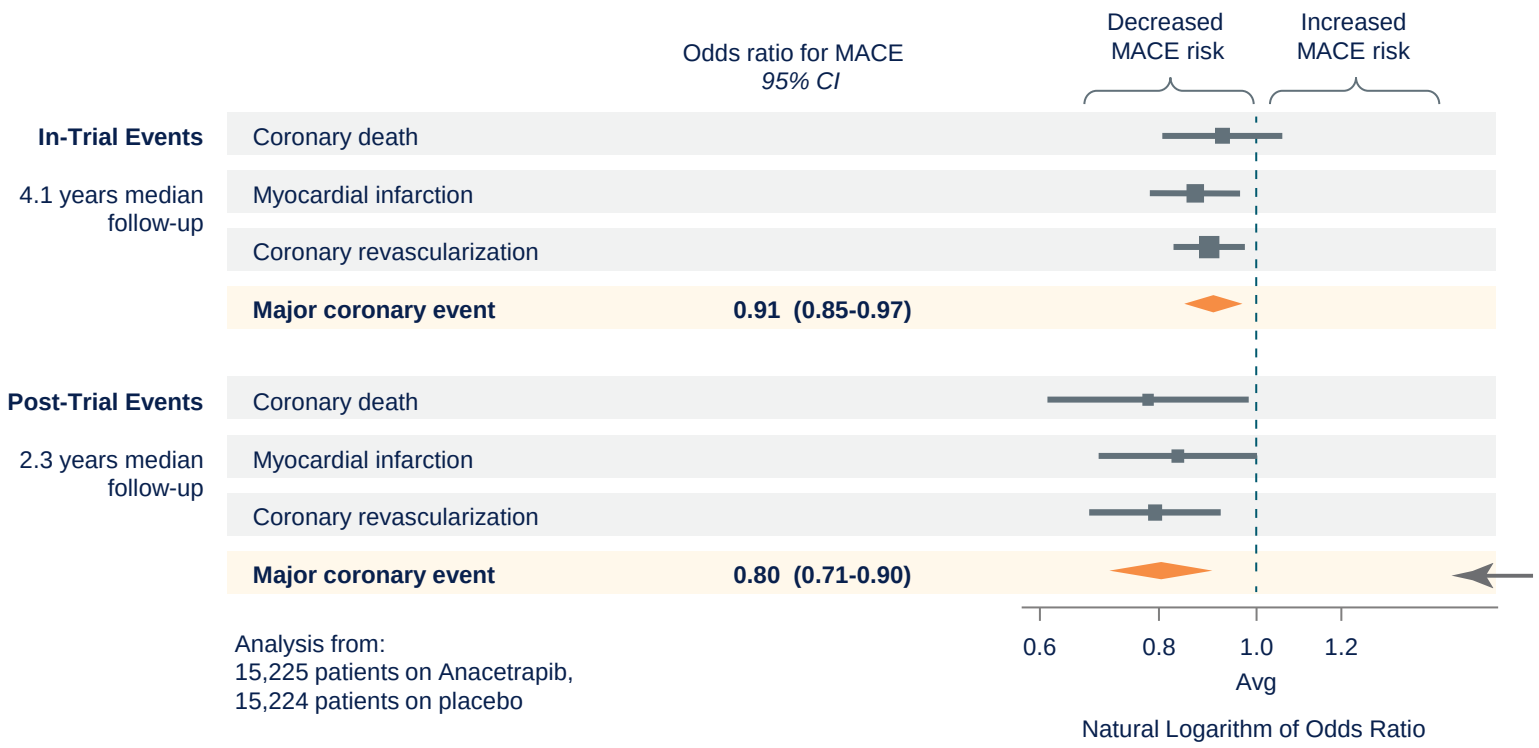
3 In Pancreas

CETP inhibition also protects vital pancreatic β cells by increasing small functional HDLs, ***supporting islet cell survival*** and ***increasing insulin production*** in the pancreas



CETP inhibition has been proven **clinically** to reduce MACE

A 2.3 year follow-up of the REVEAL trial (Phase 3) with Anacetrapib showed that CETP inhibition drove a MACE reduction of 20% compared to placebo



Analysis from:
15,225 patients on Anacetrapib,
15,224 patients on placebo

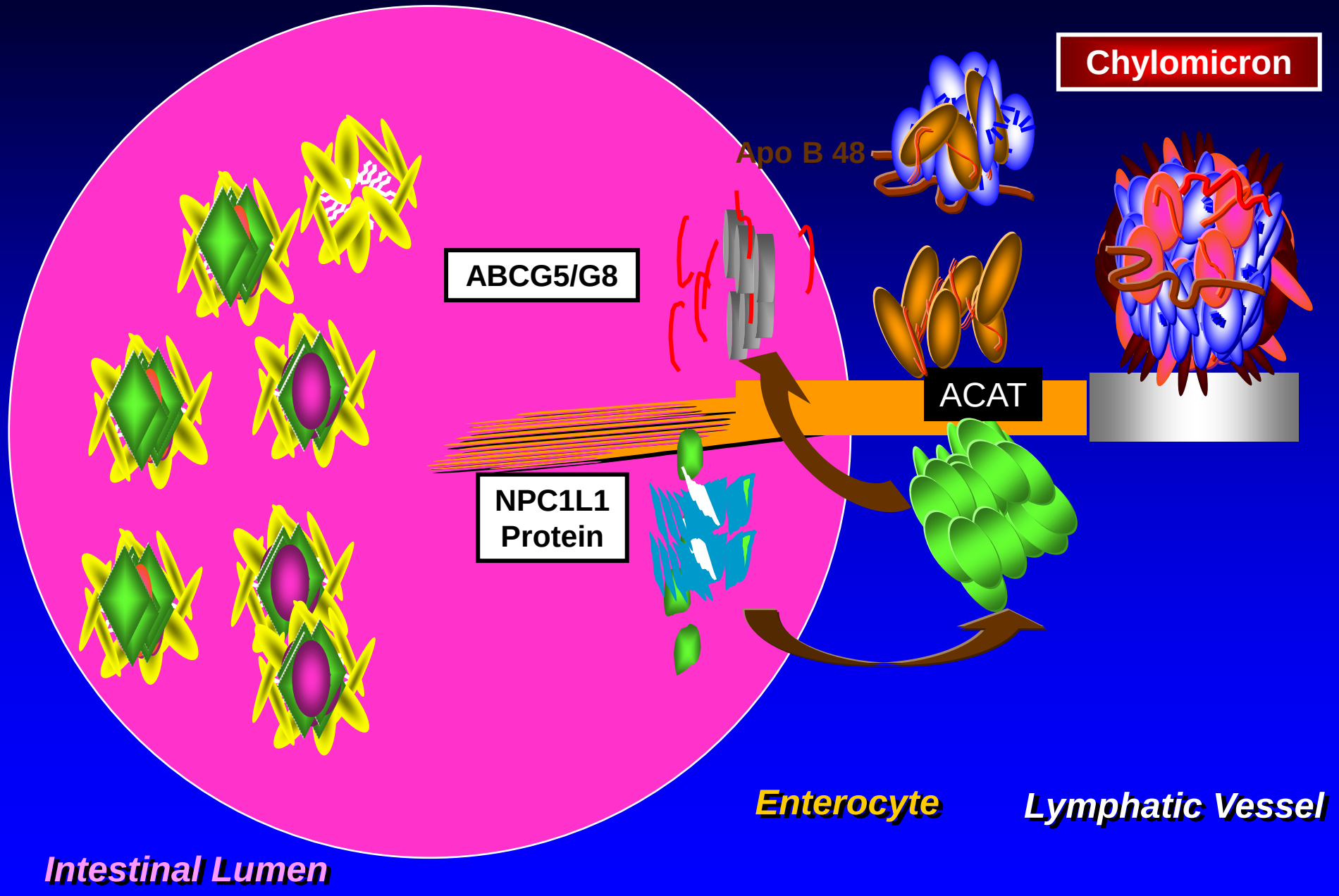
Robust, similar decrease of MACE risk observed across all composites of the primary endpoint

MACE reduction of 20% compared to placebo

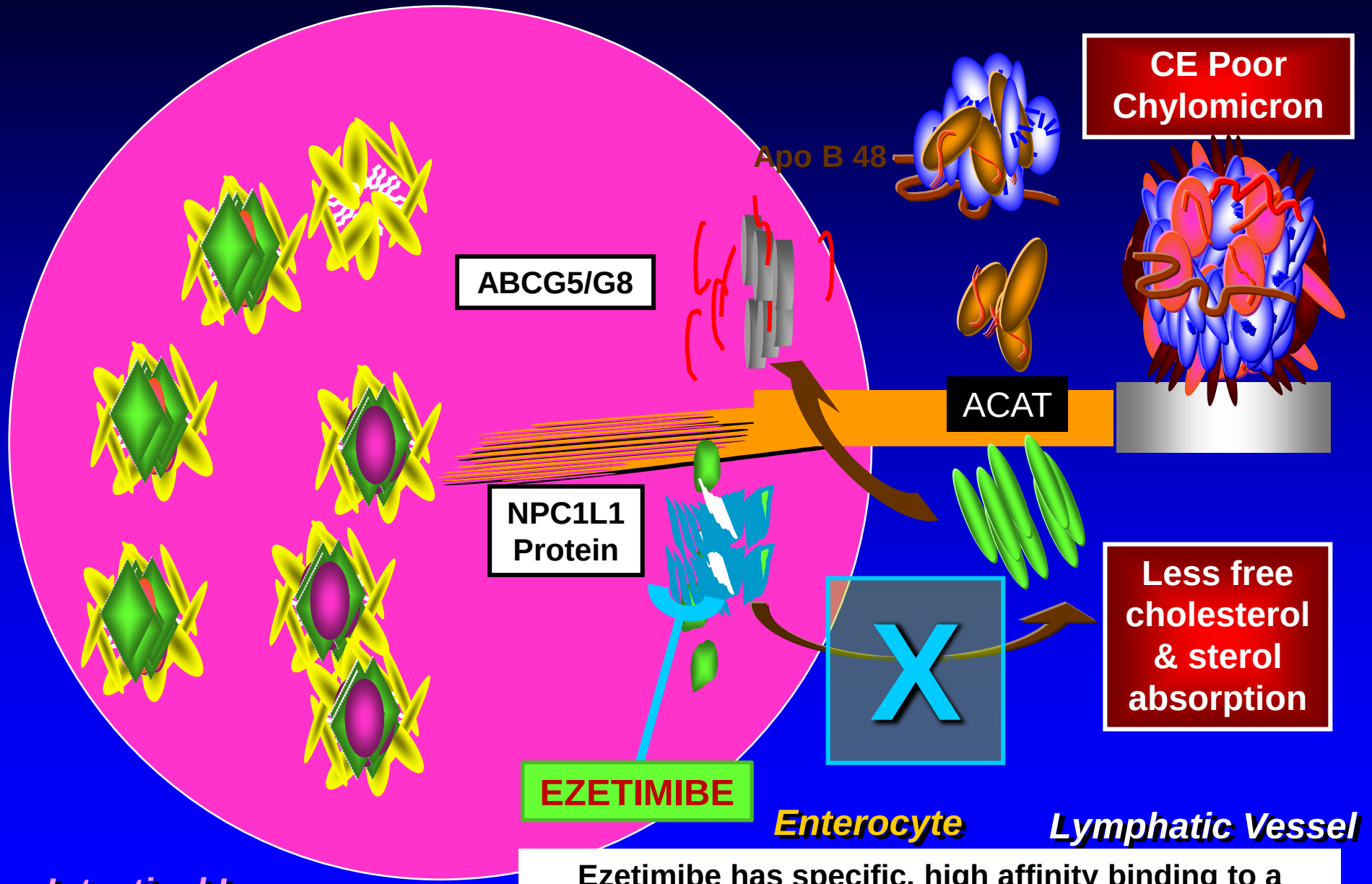
Sources: 1) HPS 3/TIMI 55-REVEAL Collaborative Group. Am Heart J 2017;187:182-90; 2) The HPS3/TIMI55–REVEAL Collaborative Group. N Engl J Med 2017; 377:1217-1227

4.NHÓM ỨC CHẾ HẤP THU CHOLESTEROL NGOẠI SINH EZETIMIBE

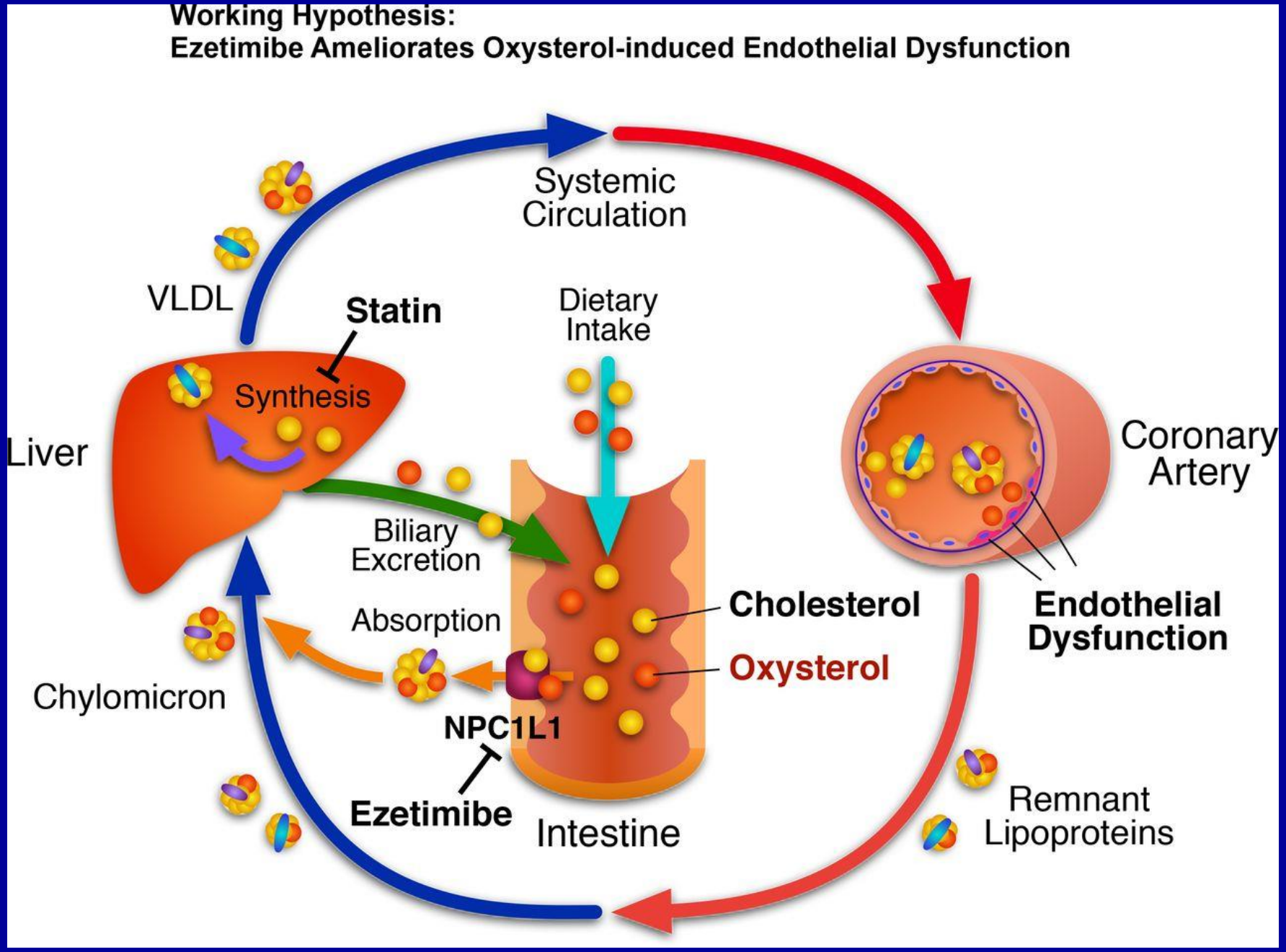
Mechanism of Cholesterol Absorption



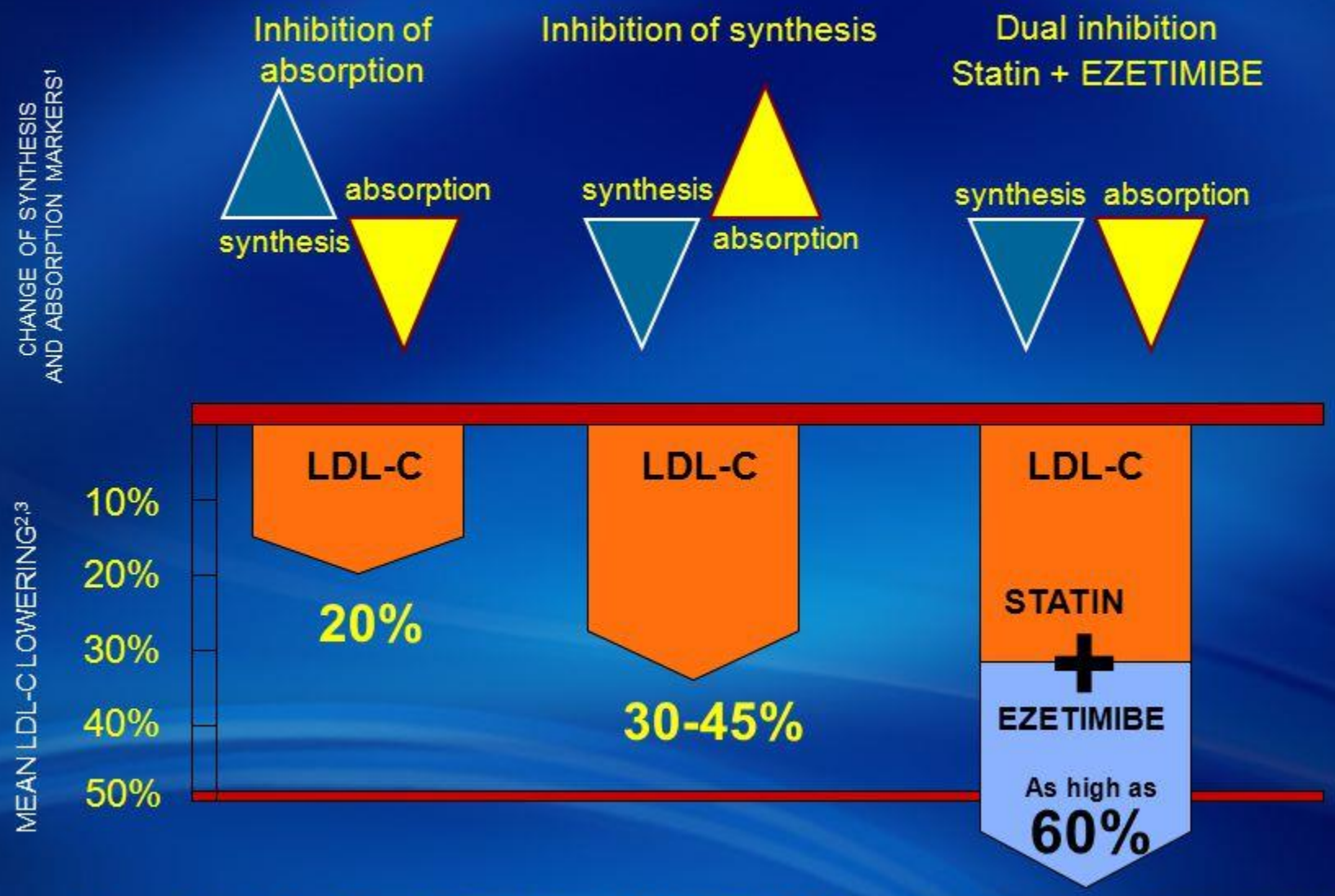
Ezetimibe : Mechanism of Action



Ezetimibe has specific, high affinity binding to a structural protein on the brush border


Working Hypothesis:
Ezetimibe Ameliorates Oxysterol-induced Endothelial Dysfunction
Systemic Circulation
VLDL
Statin
Dietary Intake
Synthesis
Liver
Coronary Artery
Biliary Excretion
Absorption
Cholesterol
Endothelial Dysfunction
Oxysterol
Chylomicron
NPC1L1
Ezetimibe
Intestine
Remnant Lipoproteins

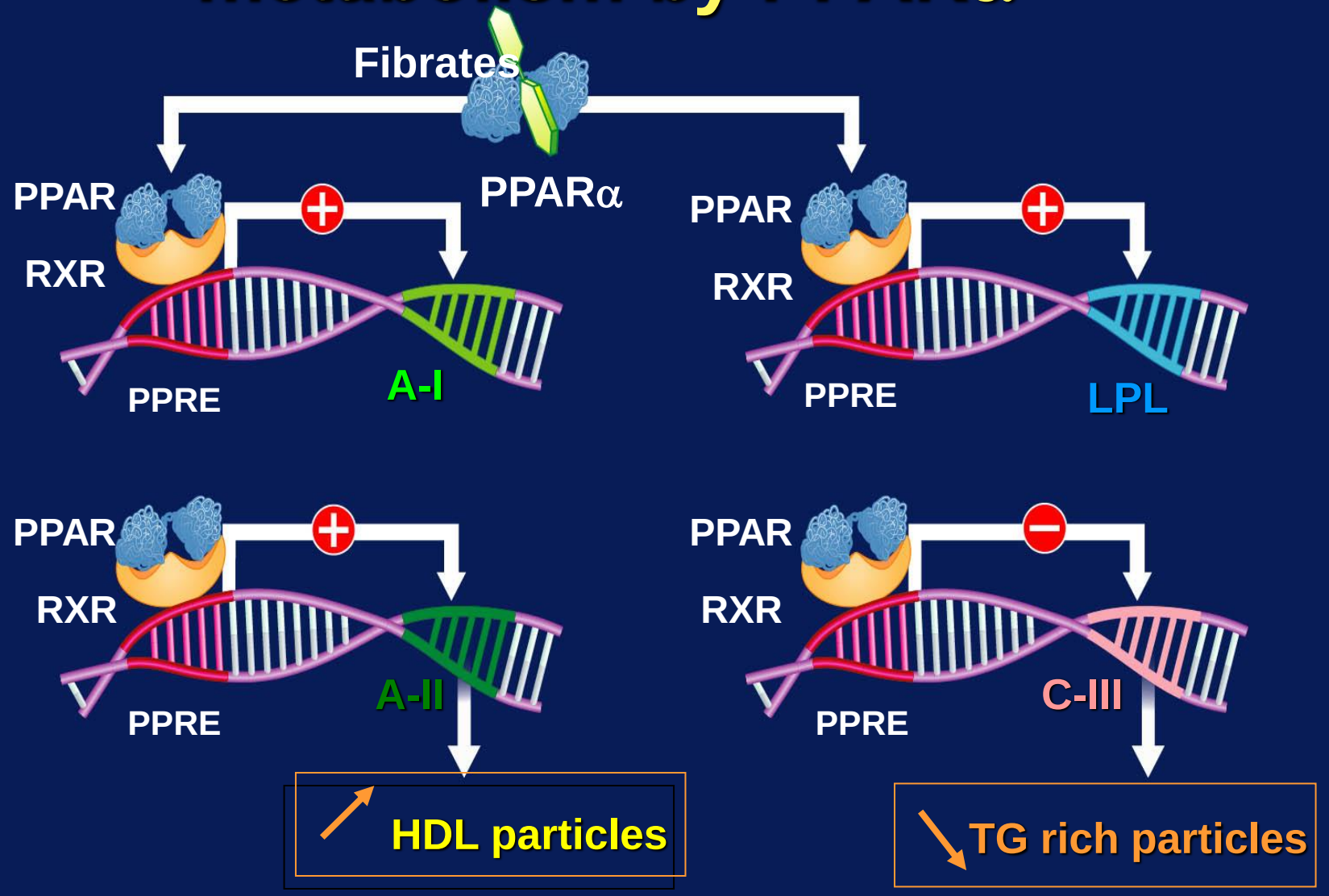
As high as 60% LDL-C lowering via dual inhibition



1. Assmann G, et al. *J Am Coll Cardiol* 2004;43(5, Suppl. 2):A445-A446; **2.** Goldberg AC, et al. *Mayo Clin Proc.* 2004 May;79(5):620-9.; **3.** Davidson M et al. J Am Coll Cardiol 2002; 40:2125-34.

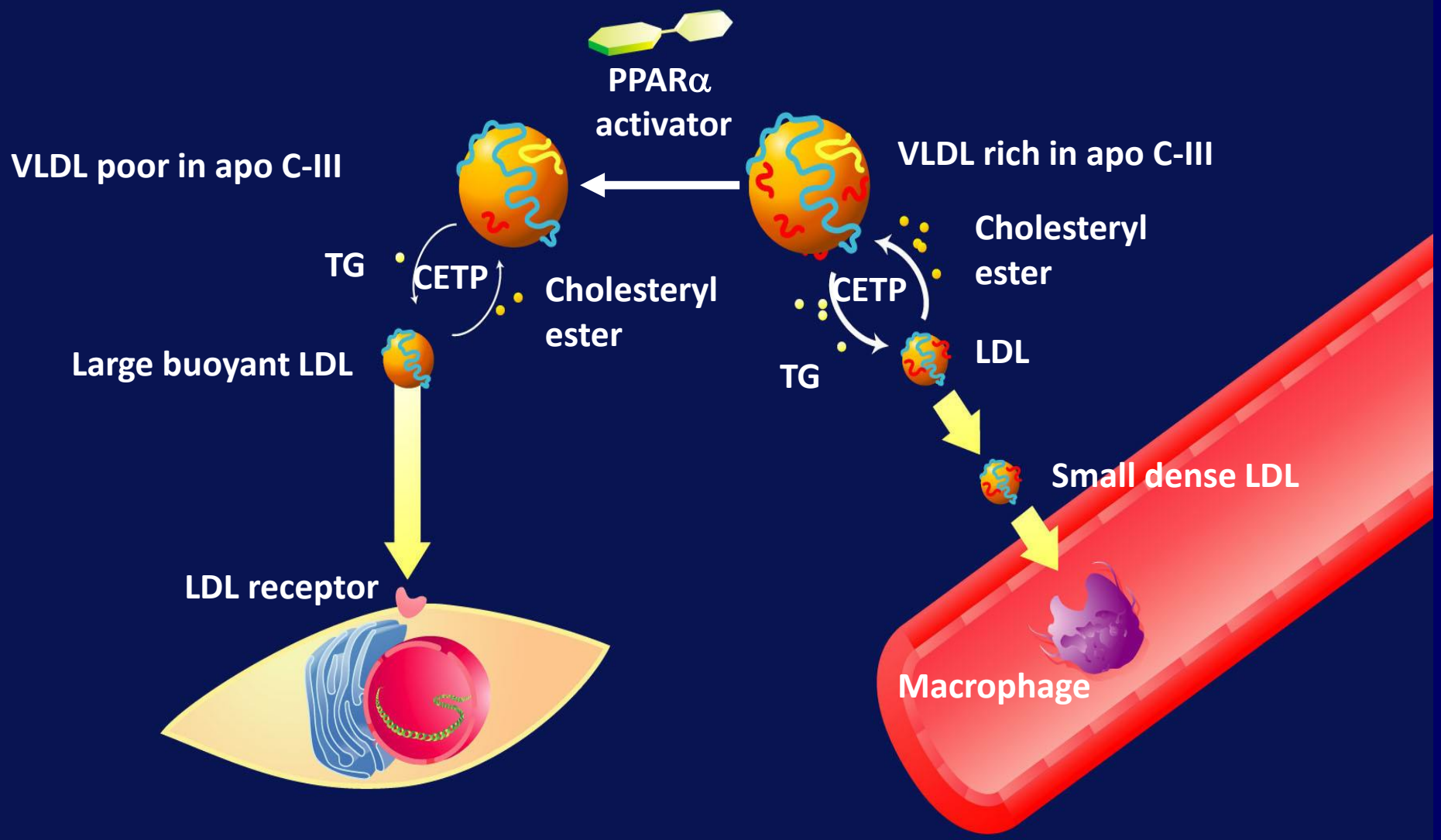
5. Nhóm giảm TG
Fibrate

PPARs : regulation of lipoprotein metabolism by PPARα

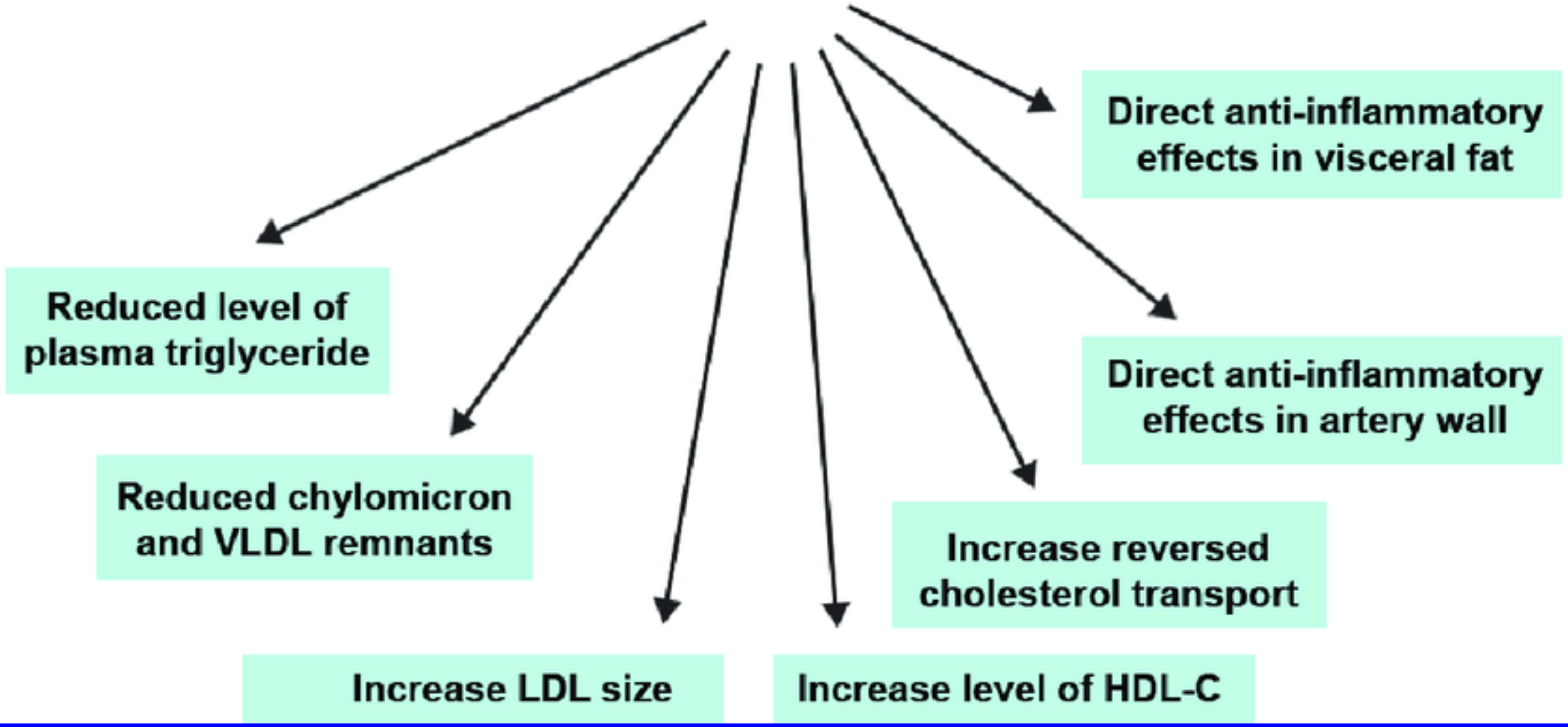


Staels B, Dallongeville J, Auwerx J, Schoonjans K, Leitersdorf E, Fruchart JC, Circulation, 1998, 98, 2088-2093

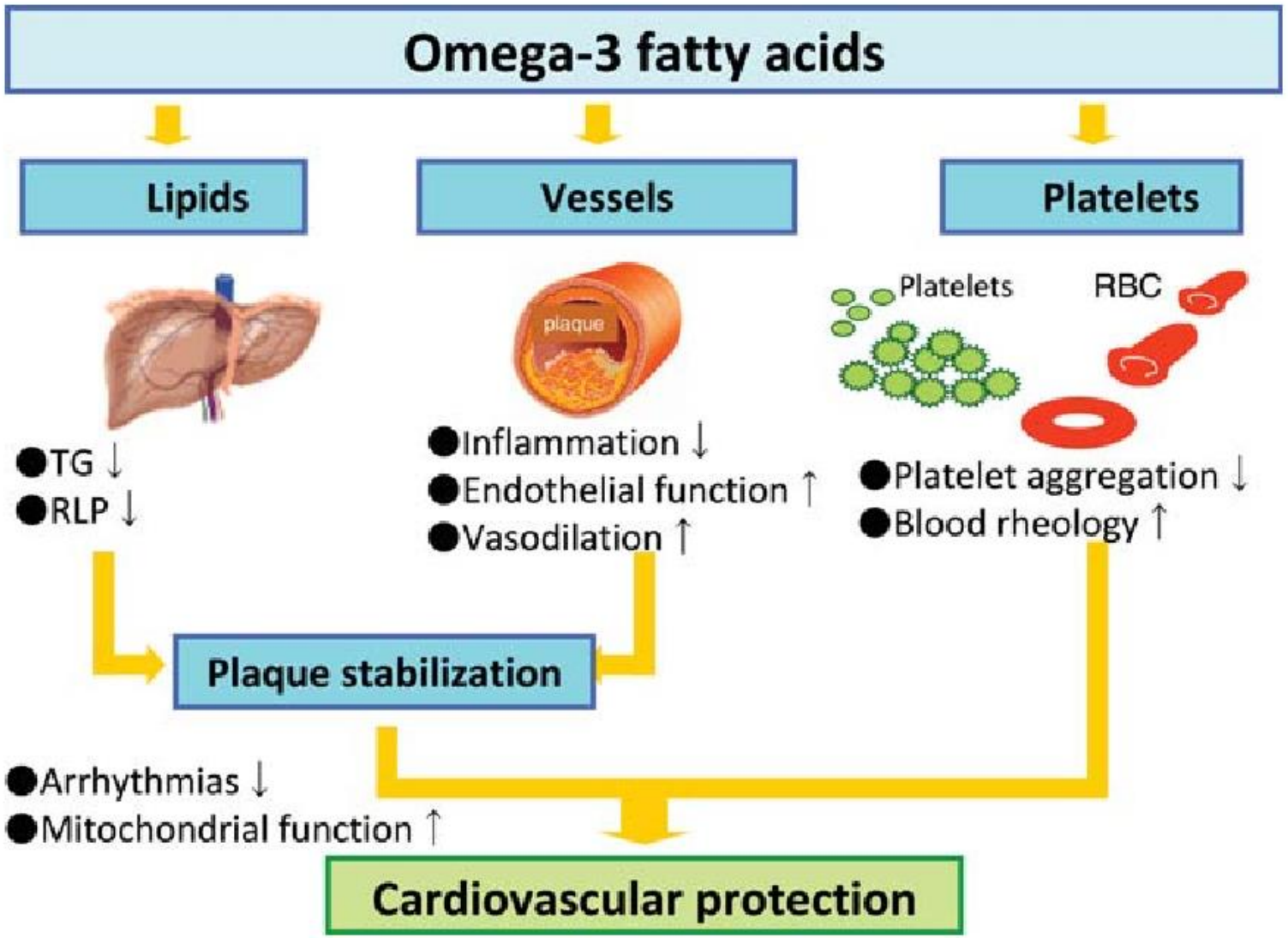
PPARα activators lower small dense LDL

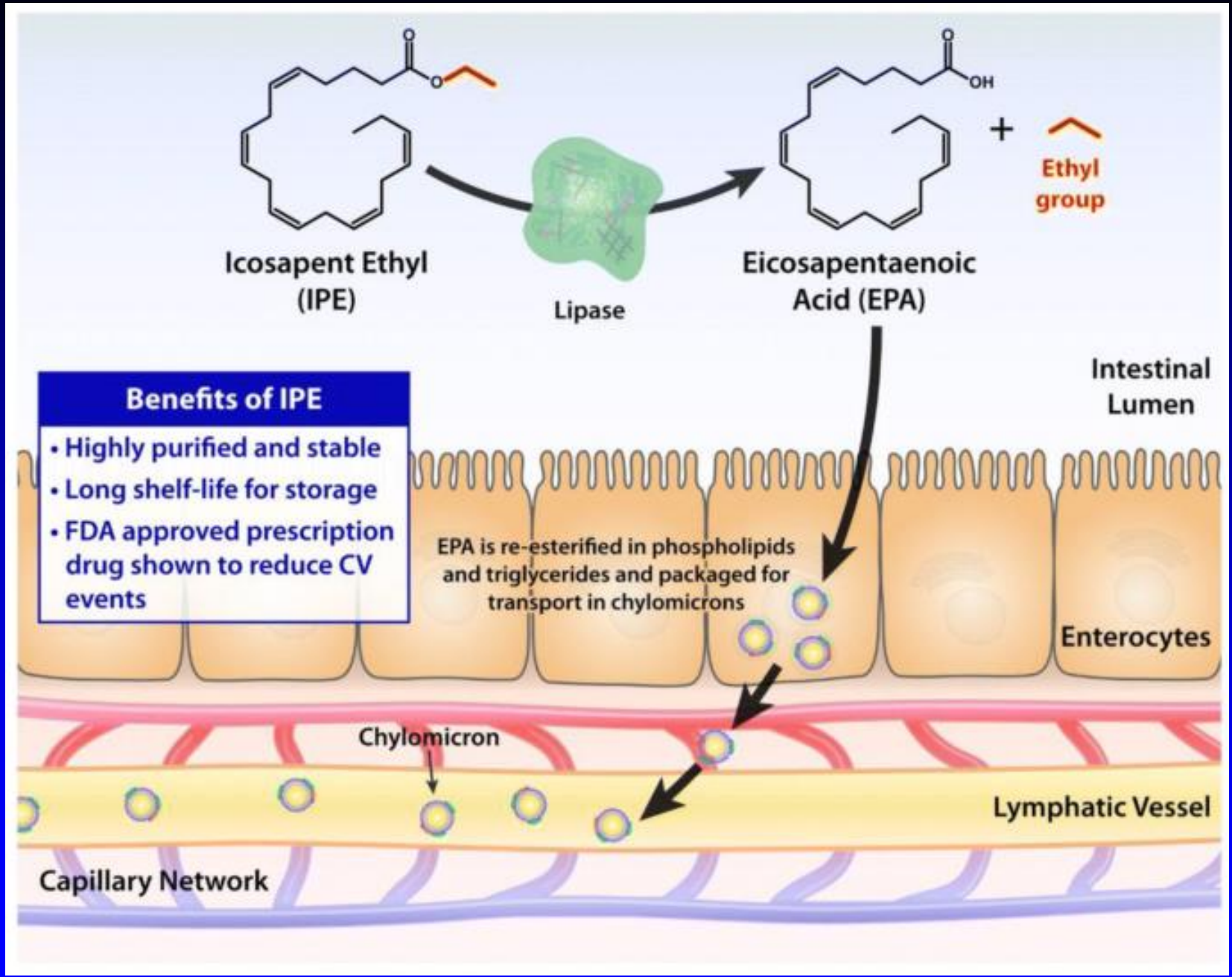


Possible anti-atherogenic mechanisms of fibrates



Nhóm Omega 3


Omega-3 fatty acids
Lipids
Vessels
Platelets
plaque
Platelets
RBC
●TG ↓
●RLP ↓
●Inflammation ↓
●Endothelial function ↑
●Vasodilation ↑
●Platelet aggregation ↓
●Blood rheology ↑
Plaque stabilization
●Arrhythmias ↓
●Mitochondrial function ↑
Cardiovascular protection


Icosapent Ethyl
(IPE)
Lipase
Eicosapentaenoic
Acid (EPA)
OH
+
Ethyl
group
Benefits of IPE
• Highly purified and stable
• Long shelf-life for storage
• FDA approved prescription drug shown to reduce CV events
Intestinal
Lumen
EPA is re-esterified in phospholipids and triglycerides and packaged for transport in chylomicrons
Enterocytes
Chylomicron
Lymphatic Vessel
Capillary Network

6. Thuốc giảm TC và TG bằng men vi sinh

LACTOBACILLUS PLANTARUM
KABP™ 011 (CECT 7527)

LACTOBACILLUS PLANTARUM
KABP™ 012 (CECT 7528)

LACTOBACILLUS PLANTARUM
KABP™ 013 (CECT 7529)

AB LIFE-Men vi sinh giảm cholesterol từ châu Âu-

Sản phẩm là sự kết hợp của 3 chủng lợi khuẩn tự nhiên Lactobacillus plantarum 7527, 7528 và 7529

có khả năng tiêu hóa cholesterol dư thừa trong cơ thể, đồng thời ức chế sự hấp thu cholesterol thức ăn.

Cơ chế 1

Lợi khuẩn trong chế phẩm AB Life sử dụng Cholesterol như một nguồn dinh dưỡng

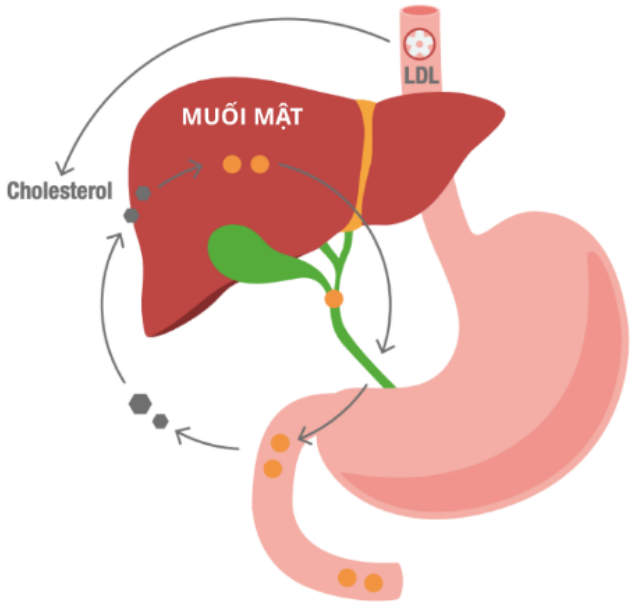
Các lợi khuẩn của AB-LIFE sử dụng Cholesterol như 1 nguồn thực phẩm, qua đó giảm lượng cholesterol mà cơ thể phải hấp thu

Cơ chế 2

Ức chế quá trình tái hấp thu muối mật

A

Khi muối mật bị phân hủy, gan sẽ huy động các phân tử Cholesterol từ máu để tạo ra muối mật mới.



B

Muối mật giải phóng Cholesterol, sau đó được đưa về gan để tái sử dụng

C

Muối mật được đưa vào ruột để giúp hấp thụ chất béo và Cholesterol

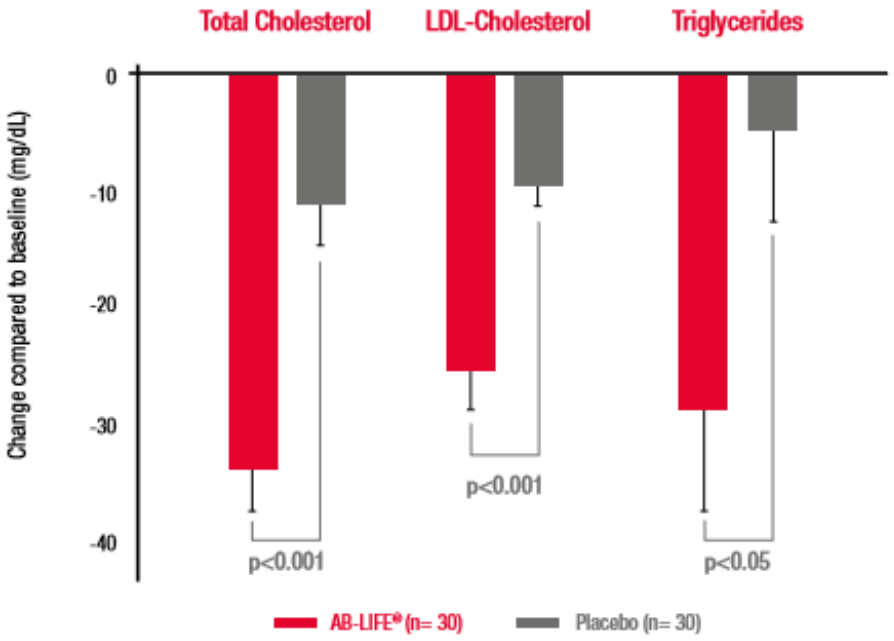
AB-LIFE®

AB-LIFE thuỷ phân các phân tử muối mật, ức chế hấp thụ Cholesterol.

Các lợi khuẩn AB-LIFE cũng sử dụng Cholesterol như một nguồn thực phẩm

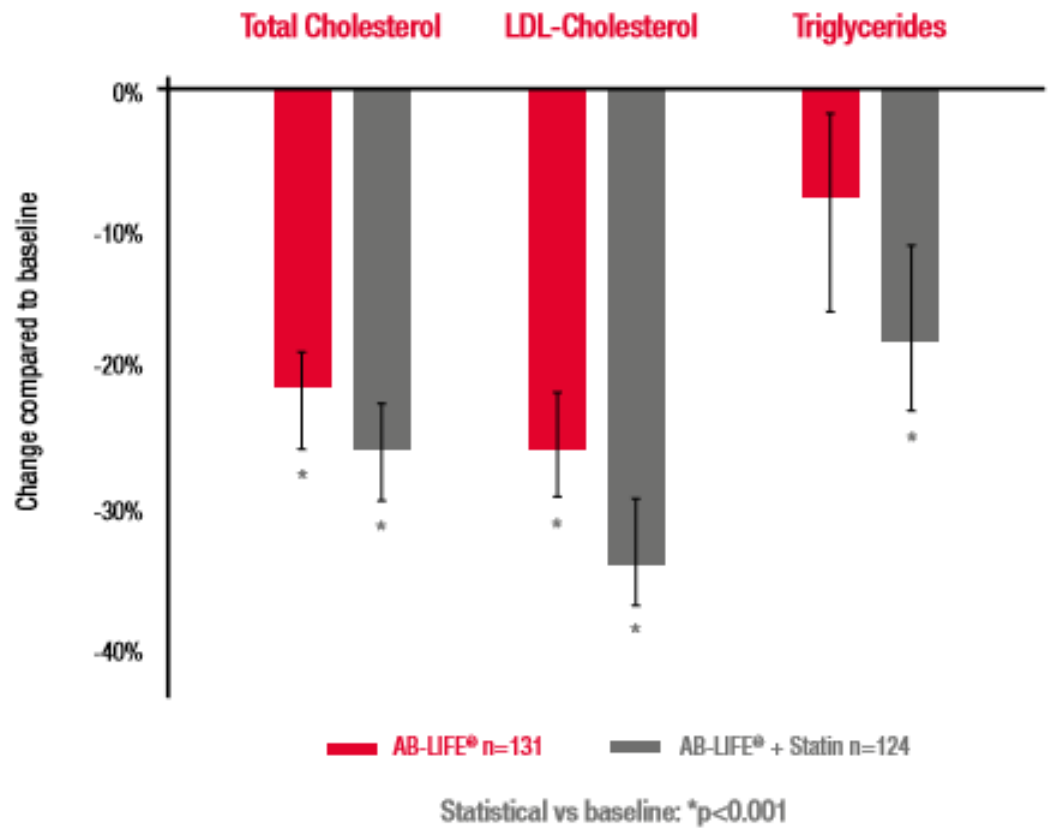
AB-LIFE®, clinical efficacy demonstrated

AB-LIFE® showed a significant reduction of Total Cholesterol, LDL-Cholesterol and Triglycerides[8-9]



Randomized, double-blind placebo-controlled, parallel arm trial

- Giảm TC 11.6%
- Giảm LDL.C 10.2%
- Giảm TG 18.6%



Open-label, multicentric, parallel, comparing AB-LIFE® to AB-LIFE® plus statins

Kết hợp statin tăng hiệu quả

Điều trị Tăng Lipid máu Kết Hợp

- Elevated LDL and/ or triglycerides
- Objective is to achieve LDL goals
- With very high TG> 400, start with fibrate or niacin
- Then treat LDL with statin
- If LDL-C goal achieved, but TG>200, non-HDL-C should be targeted
- Non-HDL goal 30 above LDL goal
- Statin titration dose OR Statin-fibrate OR Statin-Niacin combinations more effective in this type of dyslipidemia but adverse reactions more common with combined treatment so benefit/risk ratio considered
  - Titration for mild TG elevations
  - Combination TX for moderate to severe TG elevations

Treatment with drug combinations

- **Hypercholesterolemia**
  - Cholestyramine + Reductase inhibitors
- **Hypertriglyceridemia**
  - Niacin + Fibrates
- **Familial combined hyperlipidemia**
  - Cholestyramine + Fibrates.
  - Cholestyramine + Niacin.
  - Reductase inhibitors + Fibrates (but this combination may ↑ risk of myopathy).

KẾT LUẬN

- Rối Loạn lipid máu ở người Châu Á có những đặc điểm sau
- Tăng Non-HDL.C (bao gồm LDL.C)
- Rối Loạn Lipid máu kết hợp (Tăng TC và tăng TG)
- Bệnh nguyên chú ý chế độ ăn nhiều tinh bột và rượu bia
- Điều trị cần chú ý điều trị rối loạn lipid máu kết hợp nên phối hợp thuốc.

Chân thành cám ơn
sự theo dõi của quý đồng nghiệp

Kính mời quý đồng nghiệp tham gia báo cáo và tham dự

1. Hội Nghị Nội Tiết- Đái Tháo Đường Đà Nẵng lần thứ I tại Đà Nẵng ngày **16-17/06/2023**

2. Hội Nghị Nội Tiết – ĐTĐ Miền Trung và Tây Nguyên Mỡ Rộng lần thứ XIV tại Đà Nẵng ngày **9-10/9/2023**

VTM2243413