For Primary Prevention- Balancing efficacy & safety for lipid lowing therapy

馬偕醫院 心臟內科 院長室

洪大川 醫師

12-06-2020

Outline

1. What’s difference in lipid control ?

   - Primary & Secondary prevention

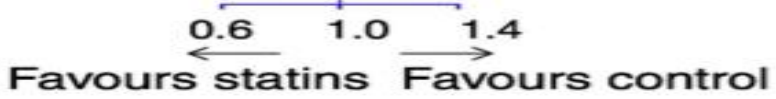
2. Racial difference in statin sensitivity

3. Primary prevention of CVD in lipid treatment

4. Statin Safety & Adherence

Statin reduce all-cause mortality in primary prevention

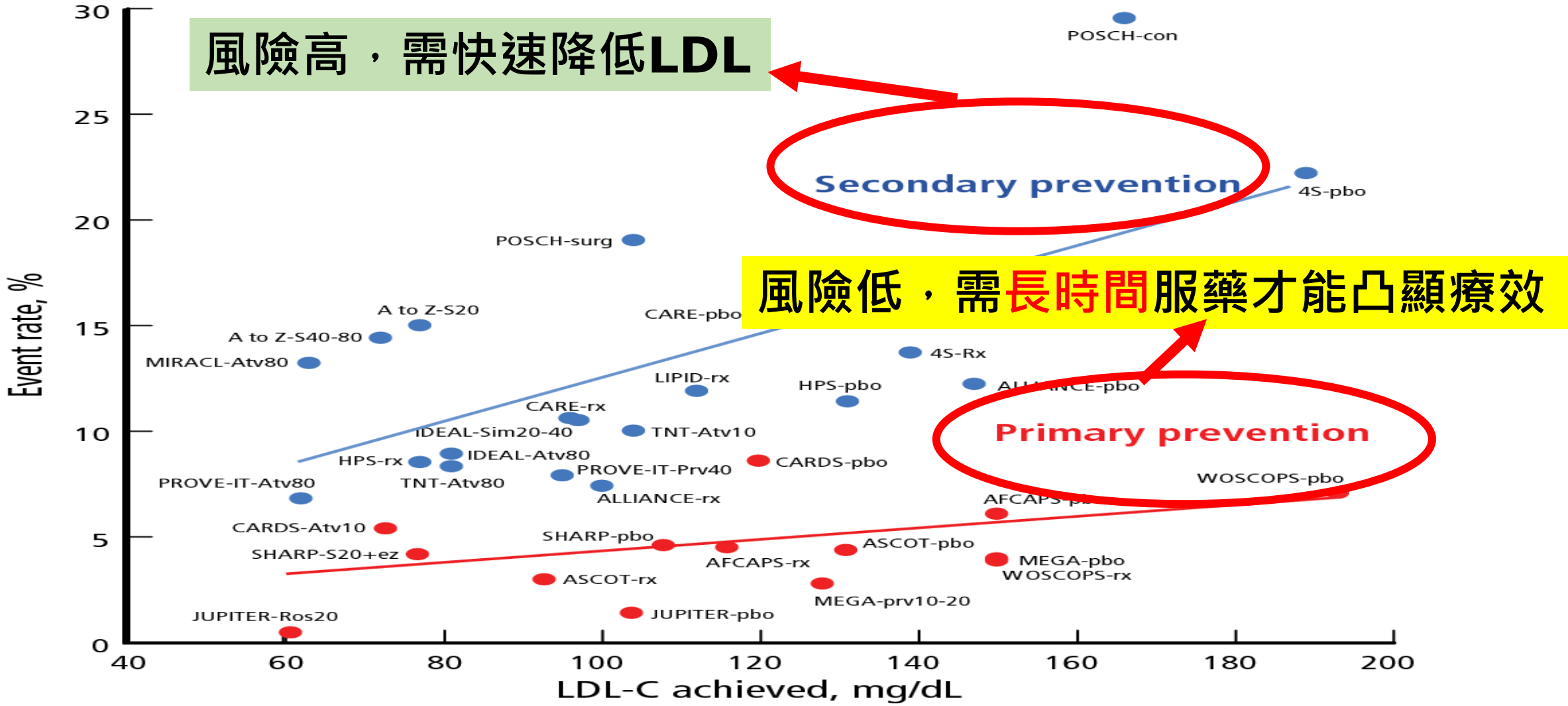
Outcomes related to statins		RR (95% CI)	Quality	RD per 1000 over 10–yrs (95% CI)
Events prevented	Nonfatal MI	0.62 (0.53,0.72)	Very low	–20 (–25 to –15)
	Fatal MI	0.72 (0.50,1.03	Moderate	–4 (–7 to 0)
	Major CV events	0.74 (0.67,0.81)	Very low	–12 (–15 to –9)
	Unstable angina	0.75 (0.63,0.91)	High	–6 (–9 to –2)
	CVD mortality	0.80 (0.71,0.91)	Moderate	–11 (–16 to –5)
	Fatal stroke	0.79 (0.53,1.19)	High	–2 (–4 to 2)
	Nonfatal stroke	0.83 (0.75,0.92)	Low	–7 (–10 to –3)
	Heart failure	0.84 (0.71,1.02)	High	–3 (–6 to 0)
	All–cause mortality	0.89 (0.85,0.93)	Low	–14 (–20 to –9)
Events in excess	Trt. discontinuation	1.00 (0.78,1.24)	Very low	0 (–34 to 38)
	All cancers	1.01 (0.93,1.09)	Moderate	+1 (–7 to 9)
	Diabetes type 2	1.04 (0.91,1.19)	Very low	+3 (–7 to 14)
	Myopathy	1.08 (1.01,1.15)	Low	+13 (2 to 24))
	Headache/Nausea	1.13 (0.97,1.31)	Moderate	+20 (–5 to 49)
	Renal dysfunction	1.14 (1.02,1.29)	Moderate	+20 (3 to 42)
	Hepatic dysfunction	1.16 (1.02,1.31)	Low	+8 (1 to 16)



AHJ American Heart Journal

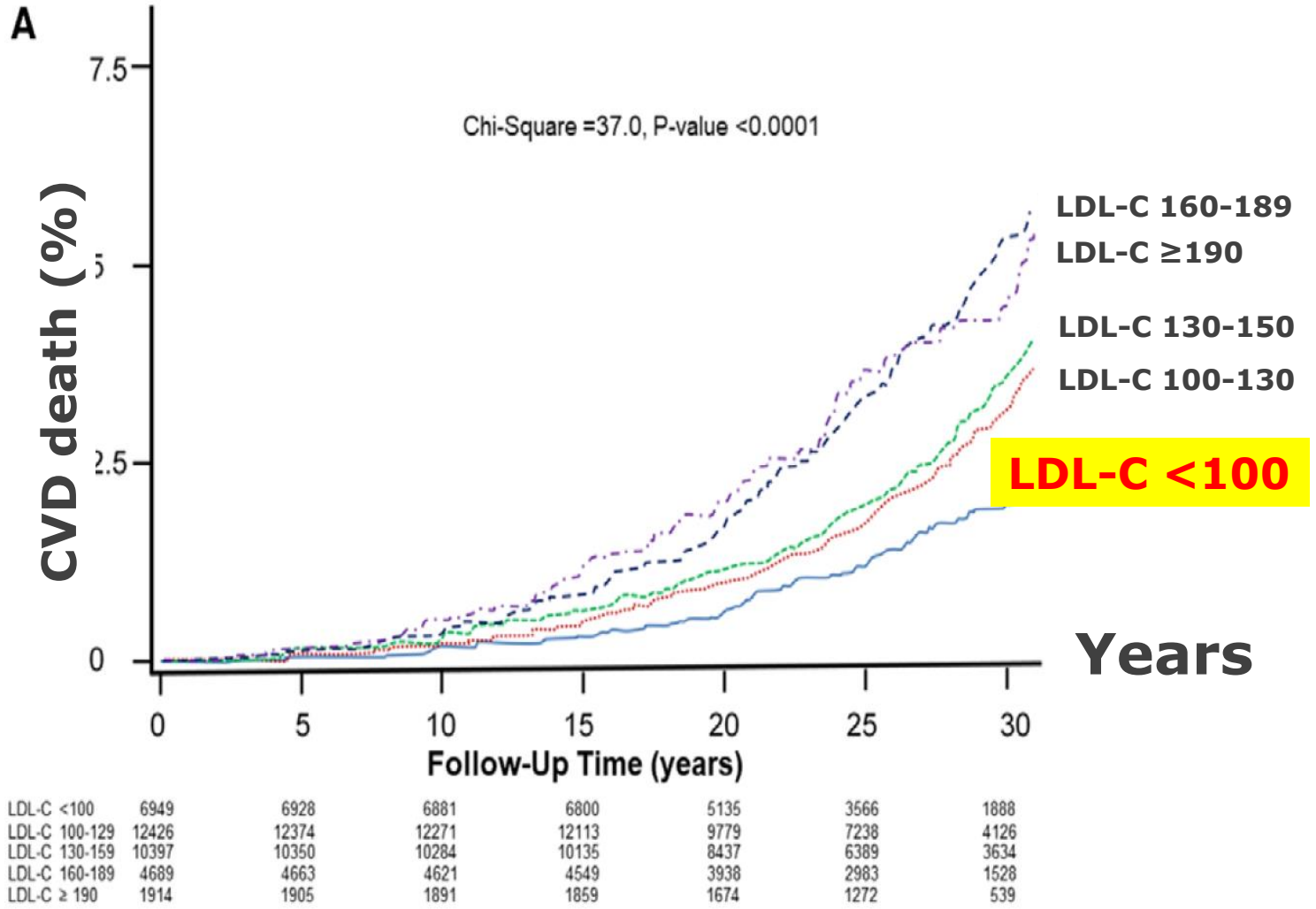
Am Heart J. 2019 Apr;210:18-28

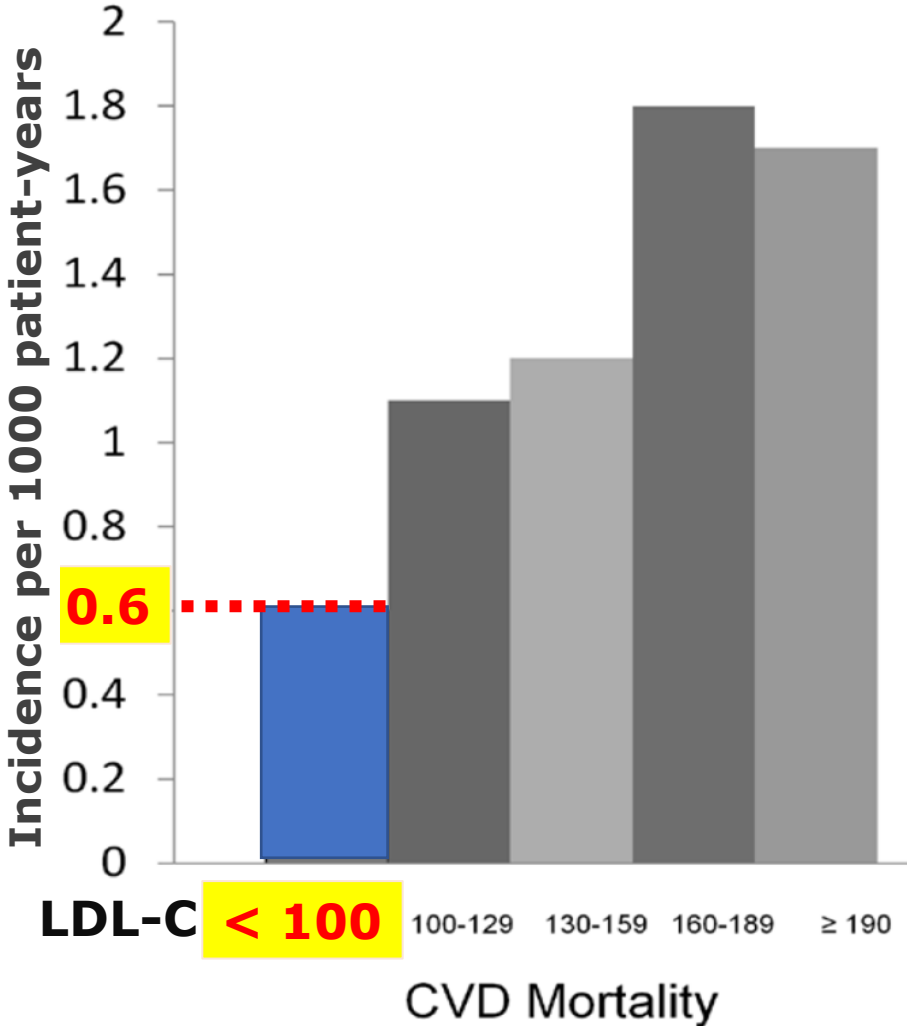
Primary prevention have lower CV event risk



Br Cardiol . 2005 Sherboume Gibbs, Ltd

Correlation between LDL and CVD mortality in primary prevention

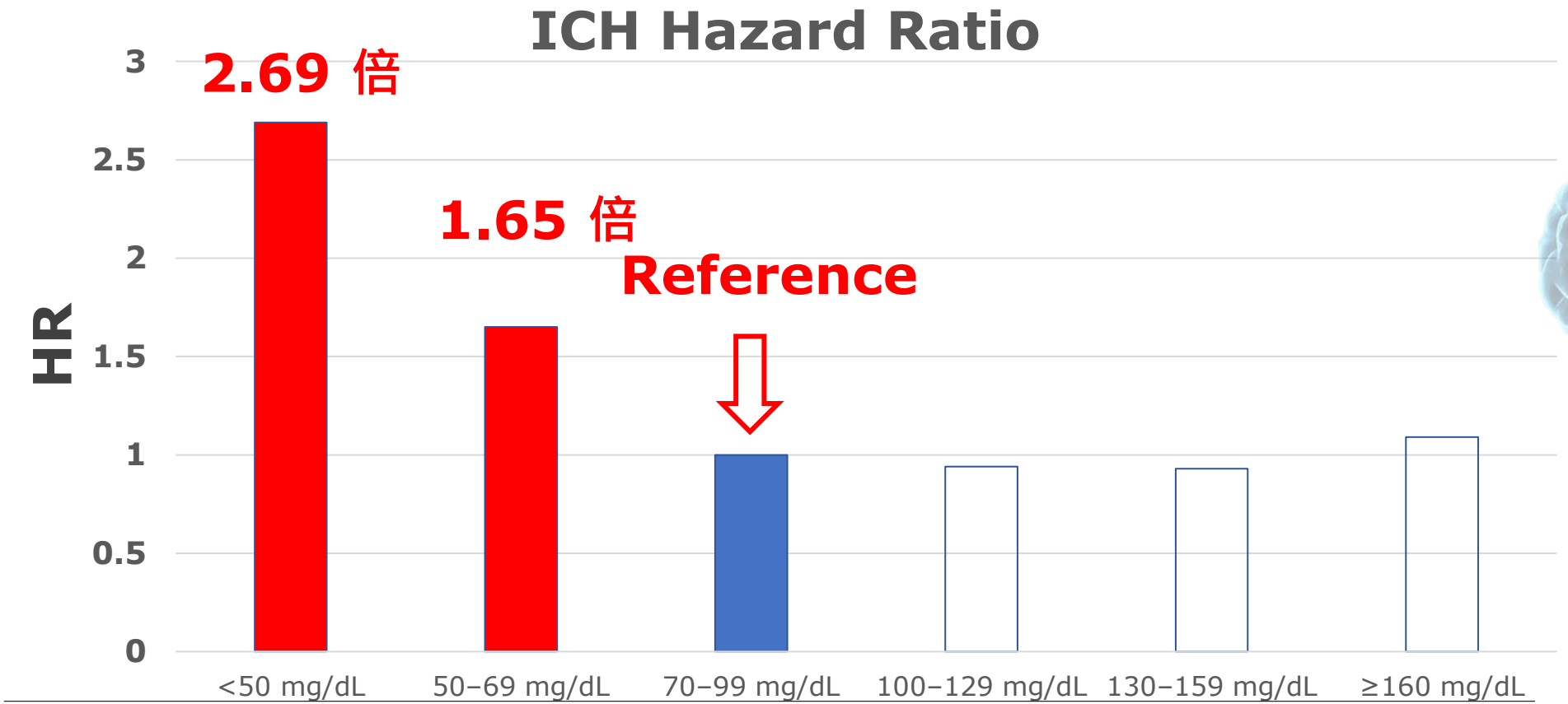




Circulation. 2018;138:2315–2325.

LDL-C lower than 70mg/dl have higher ICH risk

Kailuan (開灤) cohort study, conducted in Tangshan (唐山), China included 96,043 participants (51.3 years) who were free of stroke, MI, and cancer at baseline



	<50 mg/dL	50–69 mg/dL	70–99 mg/dL	100–129 mg/dL	130–159 mg/dL	≥160 mg/dL
SBP,[a] mm Hg	133	129	131	133	137	139
DBP,[a] mm Hg	84	83	84	85	86	87
eGFR,[a] mL/min/1.73 m^2	87.4	89	85.5	83.3	80.2	78.4

AMERICAN ACADEMY OF NEUROLOGY®

Neurology. 2019;93:e1-e13.

www.nature.com/scientificreports

SCIENTIFIC REPORTS

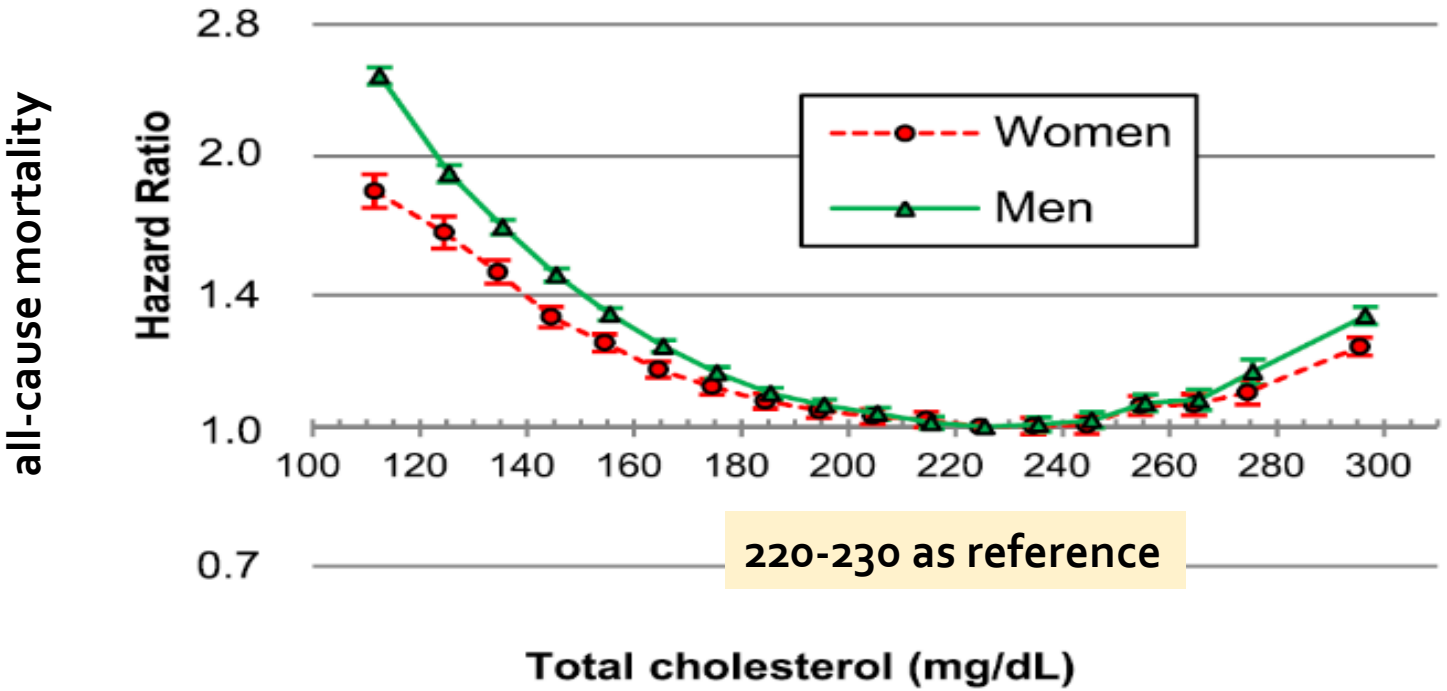
OPEN

Total cholesterol and all-cause mortality by sex and age: a prospective cohort study among 12.8 million adults

Received: 8 May 2018
Accepted: 28 December 2018
Published online: 07 February 2019

Sang-Wook Yi[1,2], Jee-Jeon Yi[3] & Heechoul Ohrr[4]

study included 12,845,017 NHIS beneficiaries 18–99 years of age who underwent routine health examinations



220-230 as reference

Sci Rep. 2019; 9: 1596

American Heart Association

American Diabetes Association

Primary prevention

長期服用，療效與安全性兼顧

Different Lipid Control Strategy

Secondary prevention

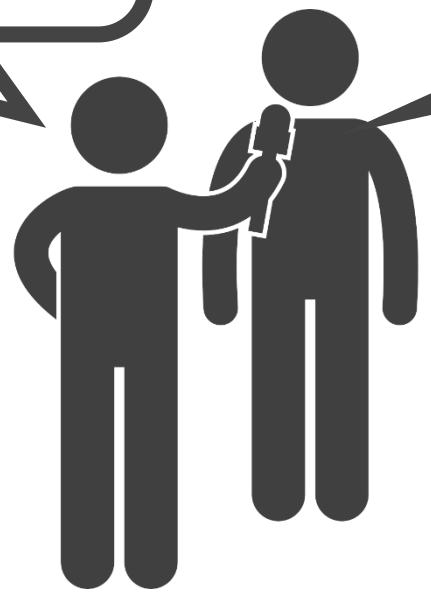
快速降低LDL

Outline

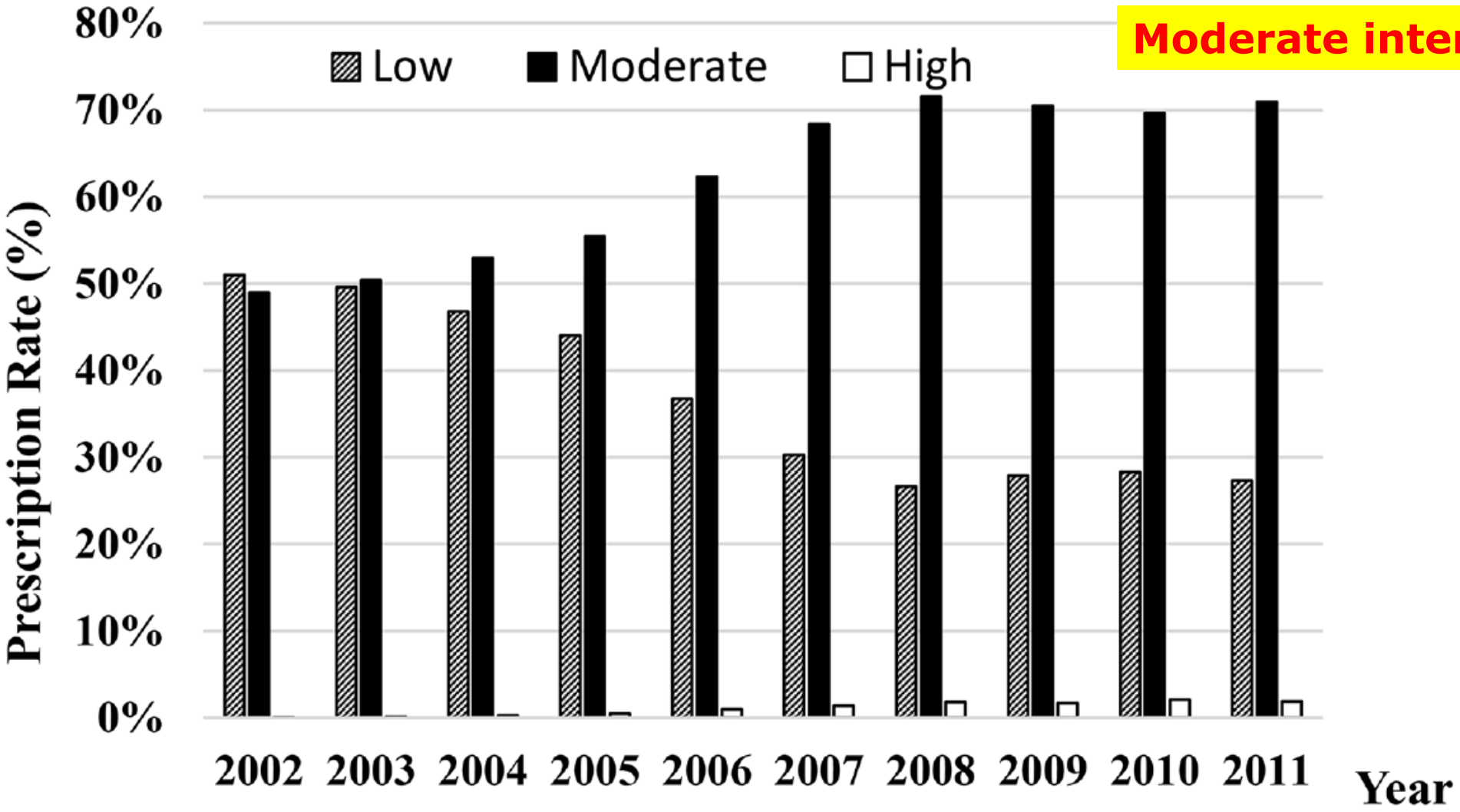
1. What's difference in lipid control ?
   - Primary & Secondary prevention
2. Racial difference in statin sensitivity
3. Primary prevention of CVD in lipid treatment
4. Statin Safety & Adherence

台灣人Primary prevention
需要使用高劑量statin?

其實……



Taiwan 10-years trends in statin utilization



BMJ Open 2017;7:e014150

Asians have higher statin response from gene difference

Gene difference

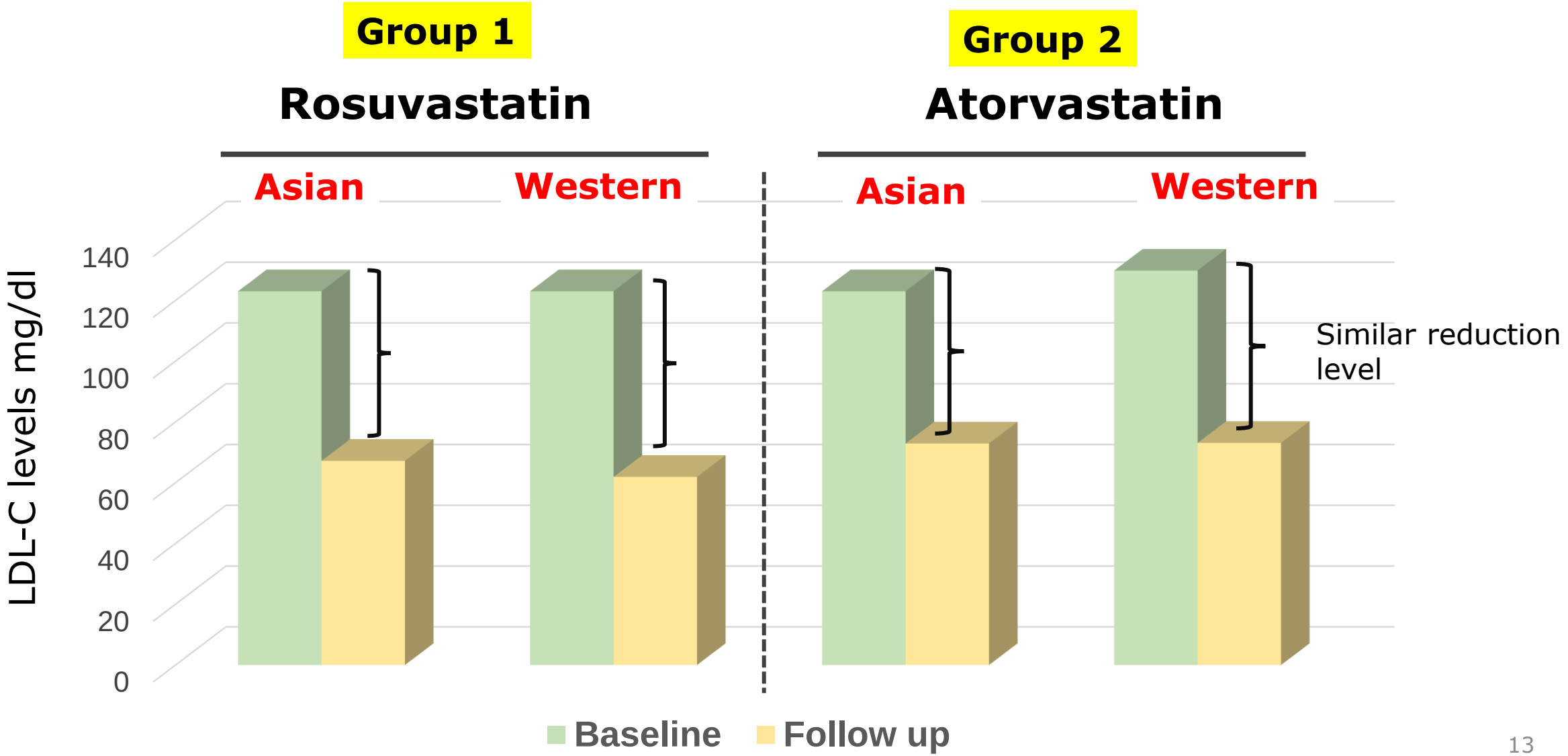
Table 2. Variant allele frequency (percentage) of polymorphisms having effects on statin pharmacokinetics in different ethnic groups

SNP	Chinese	Japanese		Caucasian	Indian[a]
SLCO1B1 521T>C	14.6-15.1	11.0		15.0	2.3
SLCO1B1 388A>G	81.7-83.7	65.1	>	40.3	55.7
ABCG2 421C>A	28.9-29.3	31.1-34.3		11.1-11.7	6.2

The *SLCO1B1* 521C allele results in the *SLCO1B1***5*, **15* and **17* haplotypes.
Data from HapMap. [a]Gujarati Indians in Houston, Texas.

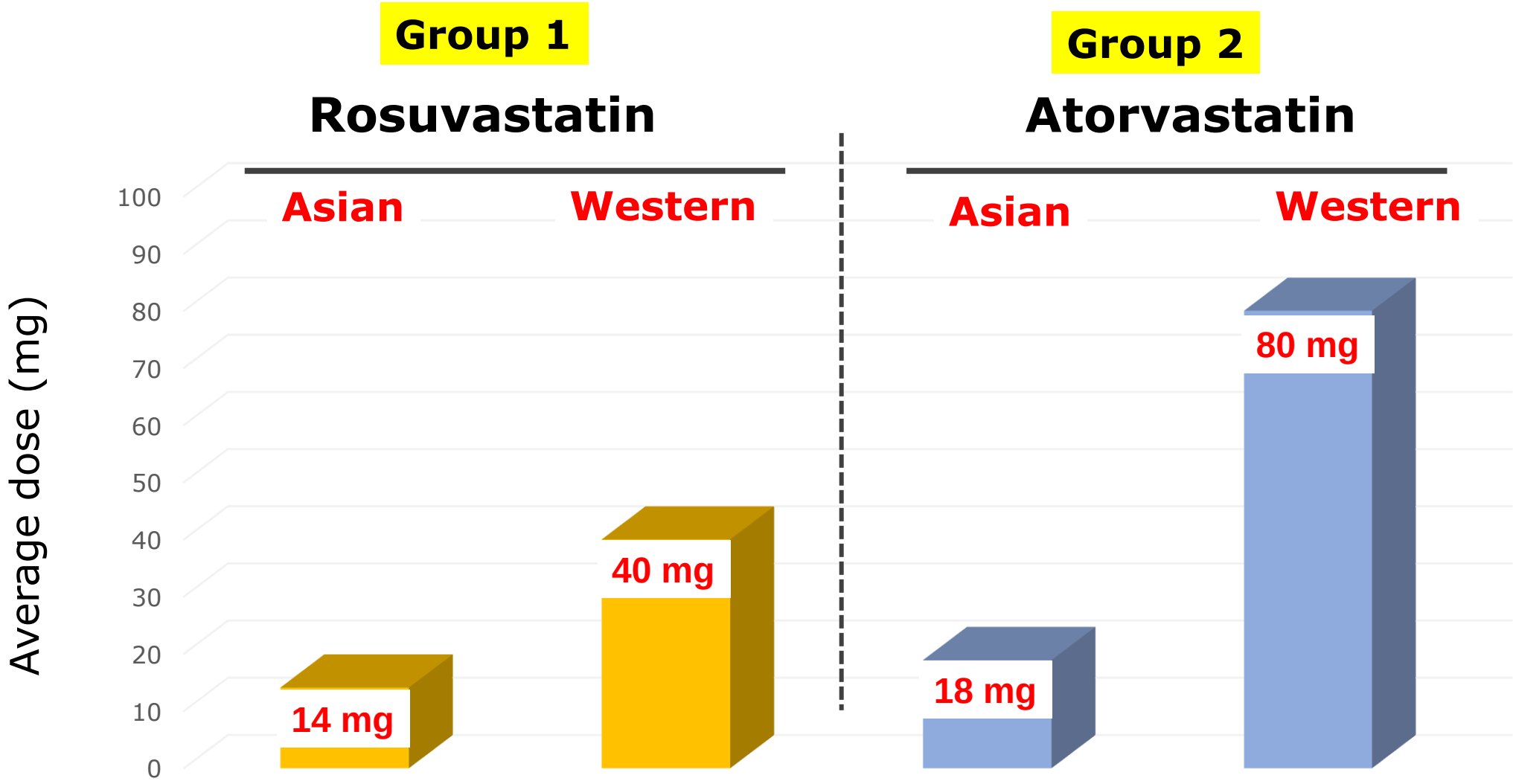
J Atheroscler Thromb, 2017; 24: 00-00

Asian and Western have similar LDL reduction level



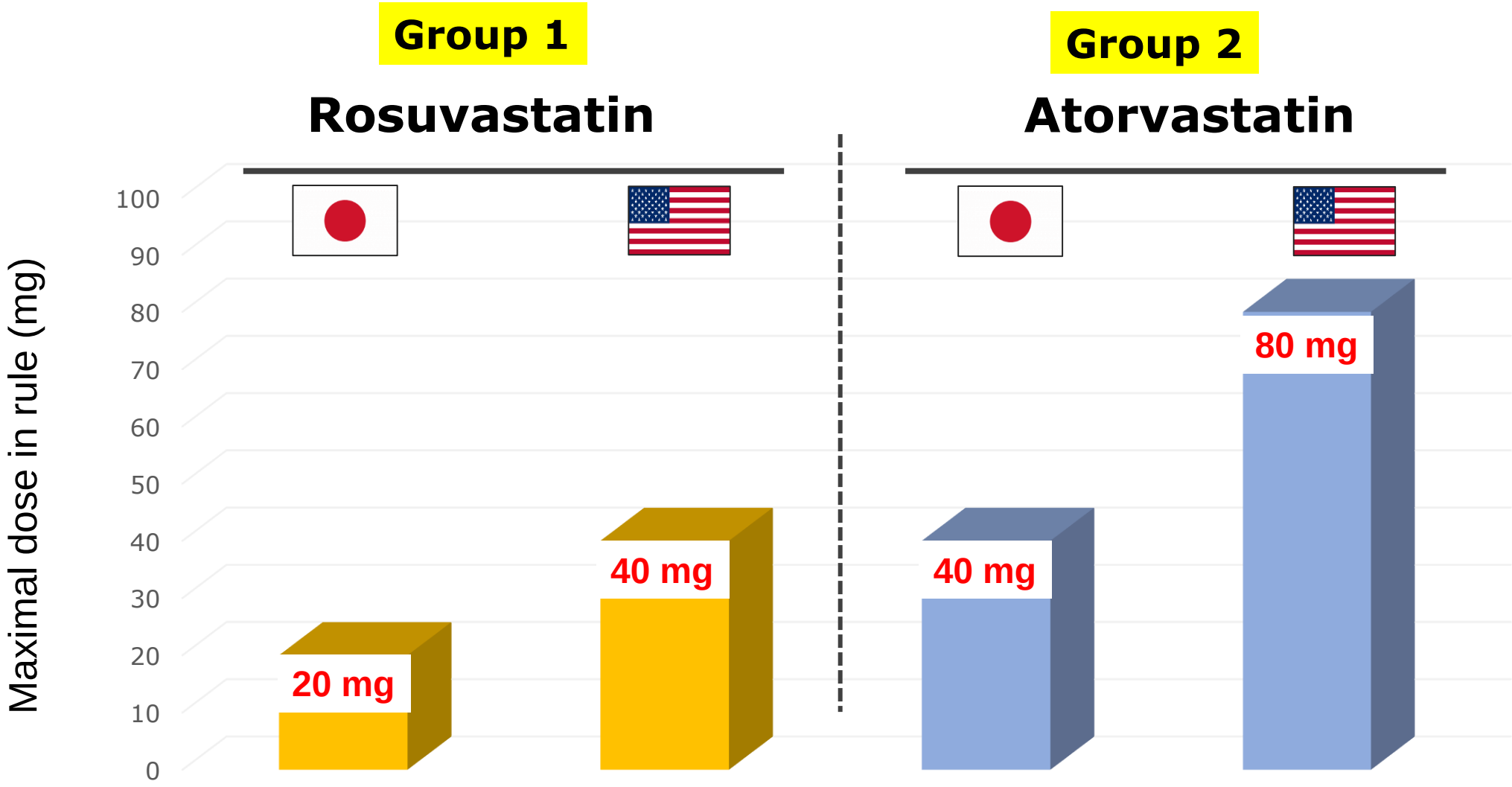
J Atherosclerosis Thromb, 2017; 24: 00-00

Asian have lower statin dosage



J Atheroscler Thromb, 2017; 24: 00-00

Maximal dose of statins in Japan and U.S rule



J Atheroscler Thromb, 2017; 24: 00-00

2018 guideline imply racial difference in statin sensitivity

Racial/ethnic issues in intensity of statin therapy & response to LDL-C lowering

- Japanese patients may be sensitive to statin dosing.

- Using a lower statins intensity(dose) in Japanese patients may give results similar to those seen with higher intensity(dose) in non- Japanese patients

American Heart Association.

2018 ACC/AHA Guideline on the Management of Blood Cholesterol

Outline

1. What's difference in lipid control ?
   - Primary & Secondary prevention
2. Racial difference in statin sensitivity
3. Primary prevention of CVD in lipid treatment
4. Statin Safety & Adherence

2017台灣高風險病人血脂異常臨床治療指引

疾病 / 狀態	低密度膽固醇 (LDL-C) 之目標
急性冠心症候群	< 70 mg/dL
急性冠心症候群+ 糖尿病	< 55 mg/dL 可以考慮
穩定冠狀動脈疾病	< 70 mg/dL
缺血性腦中風或暫時性腦部缺氧	< 100 mg/dL
糖尿病	<100 mg/dL
糖尿病+心血管疾病	< 70 mg/dL
慢性腎臟病(階段 3a–5, eGFR < 60)	> 100 mg/dL 時開始治療
家族性高膽固醇血症	成人: < 100 mg/dL 小孩: < 135 mg/dL 有心血管疾病: < 70 mg/dL

中華民國血脂及動脈硬化學會
Taiwan Society of Lipids & Atherosclerosis

全民健康保險降血脂藥物給付規定表

	非藥物治療	起始藥物治療血脂值	血脂目標值	處方規定
1.有急性冠狀動脈症候群病史 2.曾接受心導管介入治療或外科冠動脈搭橋手術之冠狀動脈粥狀硬化患者	與藥物治療可並行	LDL-C≧70mg/dL	LDL-C＜70mg/dL	第一年應每3-6個月抽血檢查一次，第二年以後應至少每6-12個月抽血檢查一次，同時請注意副作用之產生如肝功能異常，橫紋肌溶解症。
心血管疾病或糖尿病患者	與藥物治療可並行	TC≧160mg/dL或LDL-C≧100mg/dL	TC＜160mg/dL或LDL-C＜100mg/dL	
2個危險因子或以上	給藥前應有3-6個月非藥物治療	TC≧200mg/dL或 LDL-C≧130mg/dL	TC＜200mg/dL或 LDL-C＜130mg/dL	
1個危險因子	給藥前應有3-6個月非藥物治療	TC≧240mg/dL或 LDL-C≧160mg/dL	TC＜240mg/dL或 LDL-C＜160mg/dL	
0個危險因子	給藥前應有3-6個月非藥物治療	LDL-C≧190mg/dL	LDL-C＜190mg/dL	

● **心血管疾病定義：**

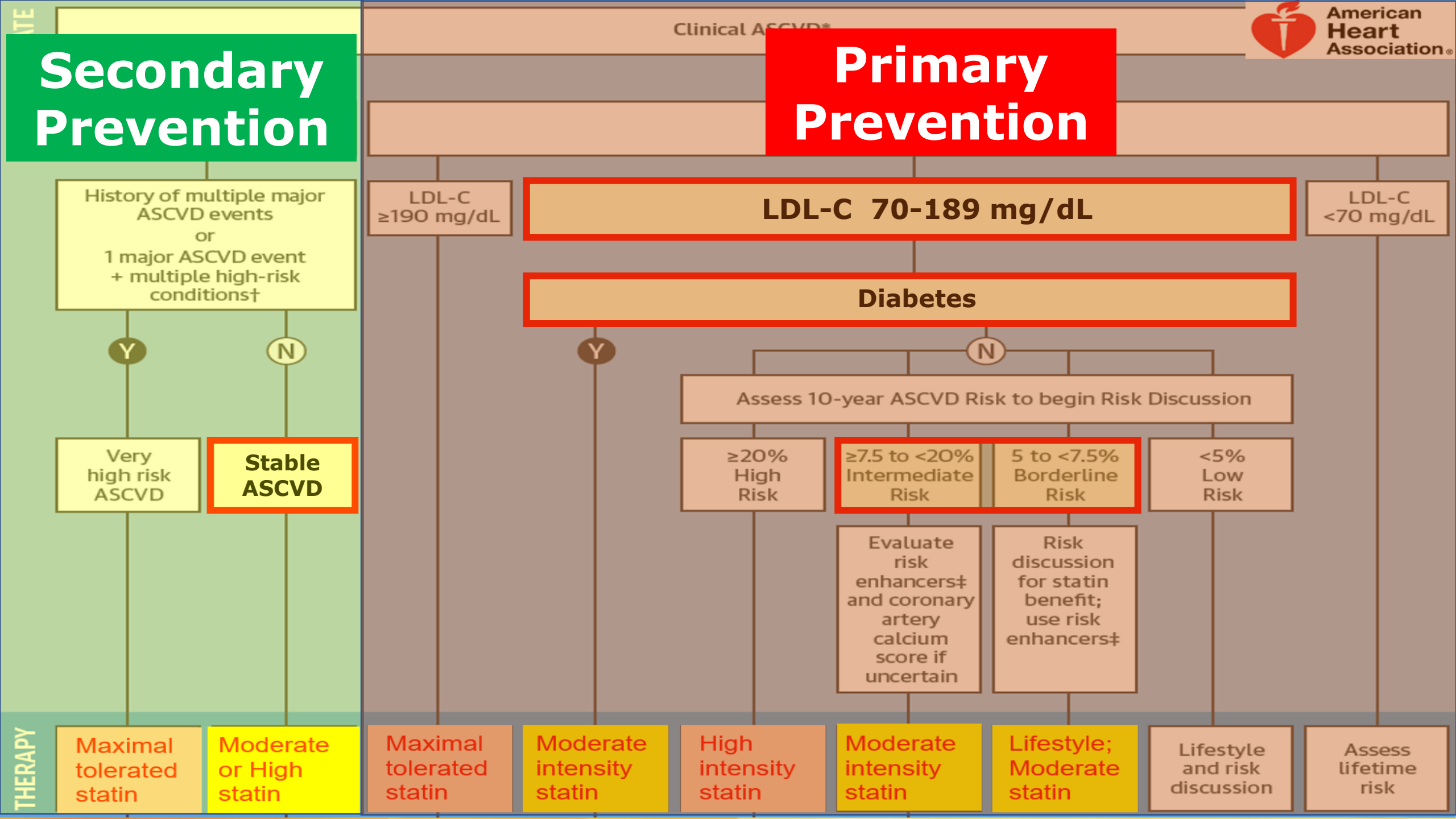
(一)冠狀動脈粥狀硬化患者包含：心絞痛病人，有心導管證實或缺氧性心電圖變化或負荷性試驗陽性反應者(附檢查報告)

(二)缺血型腦血管疾病患者包含：

1. 腦梗塞。
2. 暫時性腦缺血患者(TIA)。（診斷須由神經科醫師確立）
3. 有症狀之頸動脈狹窄。（診斷須由神經科醫師確立）

● **危險因子定義：**

1. 高血壓
2. 男性≧45歲，女性≧55歲或停經者
3. 有早發性冠心病家族史(男性≦55歲，女性≦65歲)
4. HDL-C<40mg/dL
5. 吸菸(因吸菸而符合起步治療準則之個案，若未戒菸而要求藥物治療，應以自費治療)。


Secondary Prevention
Primary Prevention
American Heart Association
Clinical ASCVD*
History of multiple major ASCVD events or 1 major ASCVD event + multiple high-risk conditions†
LDL-C ≥190 mg/dL
LDL-C 70-189 mg/dL
LDL-C <70 mg/dL
Diabetes
Y
N
Y
N
Assess 10-year ASCVD Risk to begin Risk Discussion
Very high risk ASCVD
Stable ASCVD
≥20% High Risk
≥7.5 to <20% Intermediate Risk
5 to <7.5% Borderline Risk
<5% Low Risk
Evaluate risk enhancers‡ and coronary artery calcium score if uncertain
Risk discussion for statin benefit; use risk enhancers‡
THERAPY
Maximal tolerated statin
Moderate or High statin
Maximal tolerated statin
Moderate intensity statin
High intensity statin
Moderate intensity statin
Lifestyle; Moderate statin
Lifestyle and risk discussion
Assess lifetime risk

2020 Guideline update for DM dyslipidemia treatment

Intermediate risk

DM patients without CVD

40-75 age

Lipid control

Moderate intensity statin

(AHA: Class I)

(ADA: Level A)

May High intensity statin

(AHA: Class IIa)

Diabetes-Specific Risk Enhancers **(ADA: Level B)**

American Heart Association.

American Diabetes Association.

Grundy SM, et al. 2018 AHA Cholesterol Clinical Practice Guidelines

2020 ADA guideine

Diabetic risk enhancers

Risk Enhancers
• Long duration (≥10 years for type 2 diabetes mellitus (S.4.3-20) or ≥20 years for type 1 diabetes mellitus (S4.3-6))
• Albuminuria ≥30 mcg of albumin/mg creatinine (S4.3-25)
• eGFR <60 mL/min/1.73 m^2 (S4.3-25)
• Retinopathy (S4.3-19)
• Neuropathy (S4.3-16)
• ABI <0.9 (S4.3-22, S4.3-24)

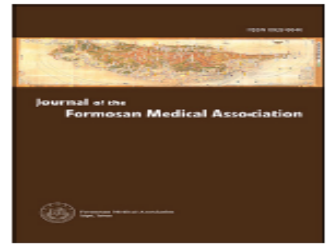
Grundy SM, et al. 2018 AHA Cholesterol Clinical Practice Guidelines
2020 ADA guideine

Journal of the Formosan Medical Association (2016) xx, 1—32

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.jfma-online.com



REVIEW ARTICLE

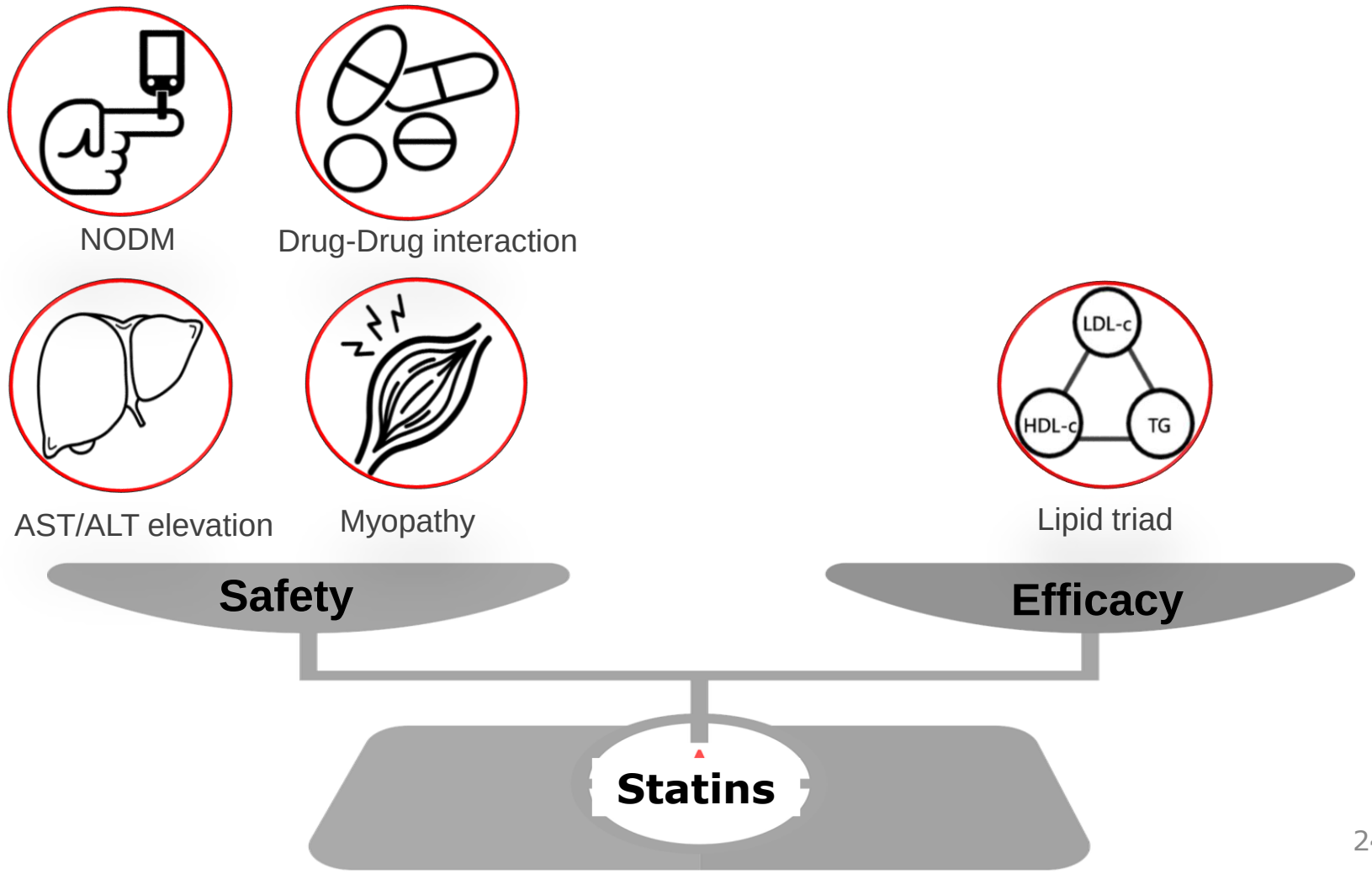
2017 Taiwan lipid guidelines for high risk patients☆

Yi-Heng Li [a], Kwo-Chang Ueng [b,c], Jiann-Shing Jeng [d], Min-Ji Charng [e,f], Tsung-Hsien Lin [g,h], Kuo-Liong Chien [i,j], Chih-Yuan Wang [j], Ting-Hsing Chao [a], Ping-Yen Liu [a], Cheng-Huang Su [k,l], Shih-Chieh Chien [k], Chia-Wei Liou [m],

Recommendation

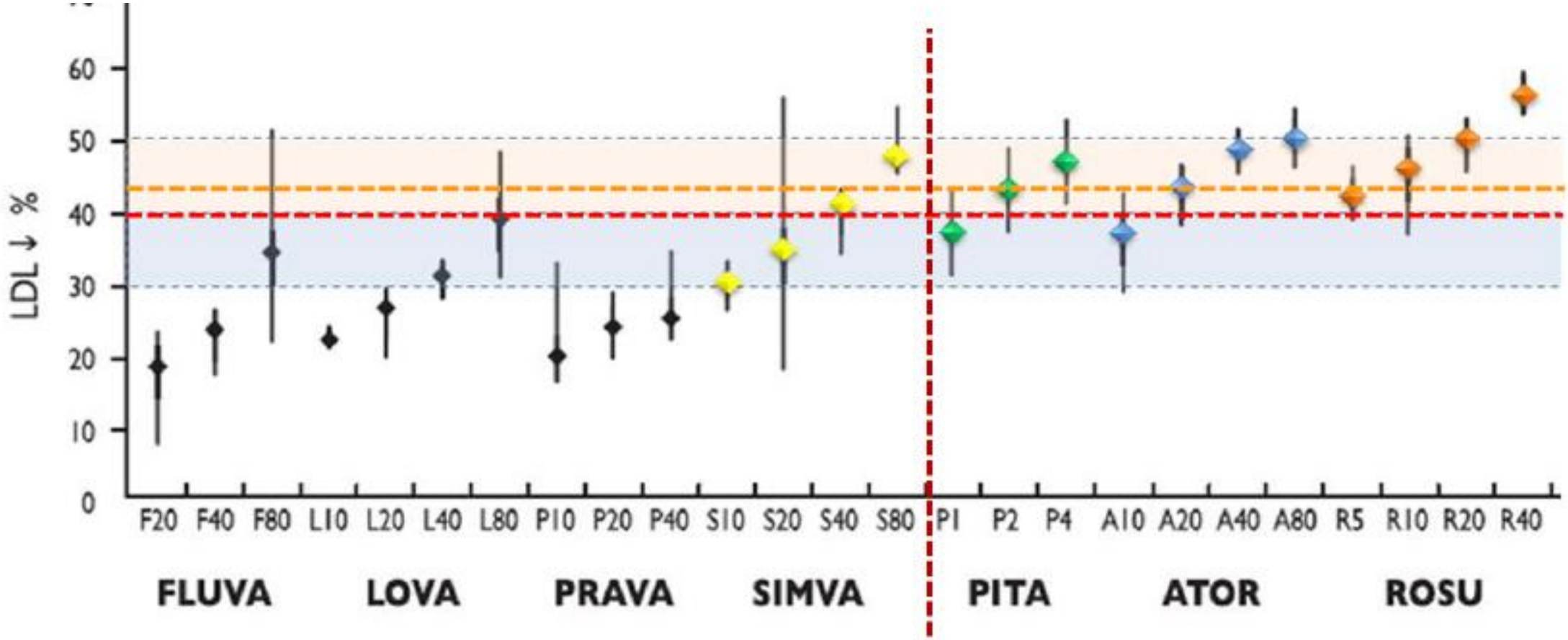
- The LDL-C target for diabetic patients who do not have overt CV disease is <100 mg/dL. (COR I, LOE A)
- The LDL-C target for diabetic patients with overt CV disease is <70 mg/dL. (COR I, LOE B)

Primary prevention, We Care



PITA, ATOR, ROSU all can reduce LDL > 40%

2016 ESC/EAS Guidelines



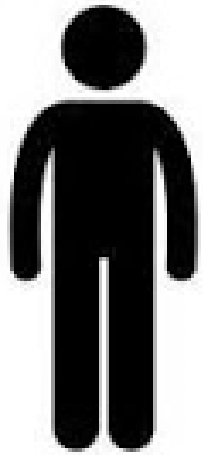
25

1. Weng TC, et al. J Clin Pharm Ther . 2010;35:139-151 2. Mukhtar RY, et Al. Int J Clin Pract . 2055;59(2):239-252

2020 Asian TOHO-LIP study

(Prospective, Randomized, open label)

Hyperlipidemia patients
Age: 65
LDL: 149 mg/dl



Pitavastatin 2mg (n=312)

5 Years

Atorvastatin 10mg (n=310)

MACE

Primary prevention: 74%
Secondary prevention: 26%

The primary outcome:
cardiovascular death, sudden death of unknown origin, nonfatal myocardial infarction, nonfatal stroke, transient ischemic attack, or heart failure requiring hospitalization.

Int J Cardiol. 2020 S0167-5273(19)34045-8

Baseline of trial participants

Table 1
Baseline characteristics of trial participants.

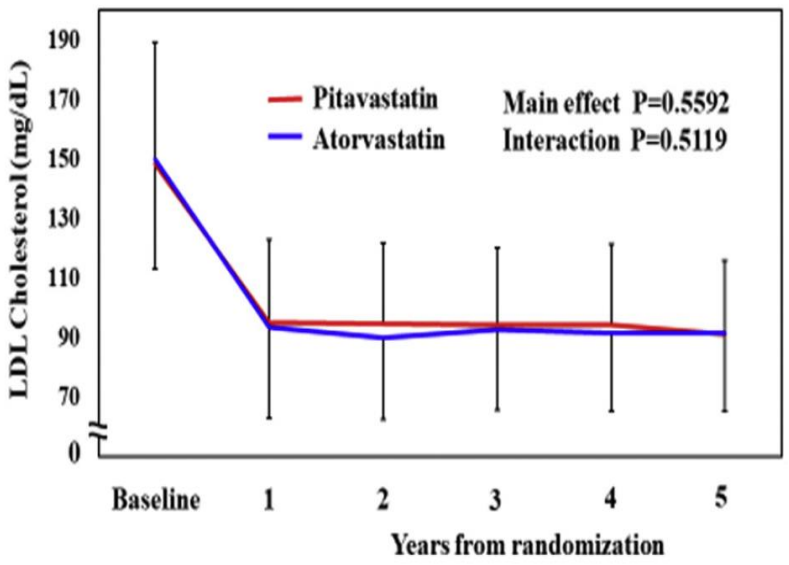
	Pitavastatin (n = 312)	Atorvastatin (n = 310)	P-value
Male (%)	168 (53.8)	168 (54.2)	0.936
Age (years)	65.3 ± 10.1	65.4 ± 9.4	0.932
BMI (kg/m^2)	24.8 ± 3.9	24.3 ± 3.2	0.144
Systolic blood pressure (mmHg)	131 ± 16	132 ± 16	0.364
Diastolic blood pressure (mmHg)	77 ± 11	77 ± 10	0.624
Blood test			
HbA1c, JDS (%)	6.7 ± 1.2	6.8 ± 1.3	0.393
HbA1c, NGSP (%)	7.1 ± 1.2	7.2 ± 1.3	0.393
AST (IU/L)	24 ± 10	23 ± 8	0.266
ALT (IU/L)	25 ± 15	23 ± 12	0.066
γ-GTP (IU/L)	37 ± 51	35 ± 42	0.591
Uric acid (mg/dL)	5.4 ± 1.5	5.4 ± 1.5	0.890
Creatinine (mg/dL)	0.80 ± 0.22	0.80 ± 0.21	0.900
Total cholesterol (mg/dL)	236 ± 45	236 ± 41	0.926
Triglyceride (mg/dL)	172 ± 107	165 ± 95	0.434
HDL-cholesterol (mg/dL)	55 ± 14	55 ± 14	0.934
NonHDL-cholesterol (mg/dL)	182 ± 46	181 ± 40	0.949
LDL-cholesterol (mg/dL)	148 ± 41	150 ± 37	0.652
Prevalence of diabetes, hypertension, and prior atherosclerotic vascular events			
Diabetes	238 (76.3)	234 (75.5)	0.852
Hypertension	232 (74.4)	231 (74.5)	1.000
Prior ACS (Myocardial infarction/Unstable angina)	66 (21.2)	67 (21.6)	0.922
Coronary revascularization	70 (22.4)	70 (22.6)	1.000
Stroke	50 (16.0)	46 (14.8)	0.739
Peripheral artery disease	11 (3.5)	7 (2.3)	0.474

Int J Cardiol. 2020 S0167-5273(19)34045-8

LDL, TC, Non-HDL have no difference

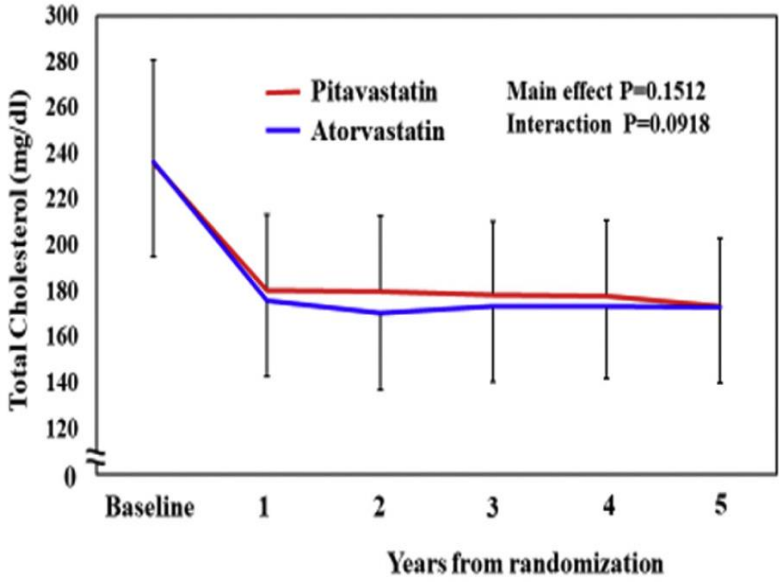
LDL-C

LDL-C: -36%



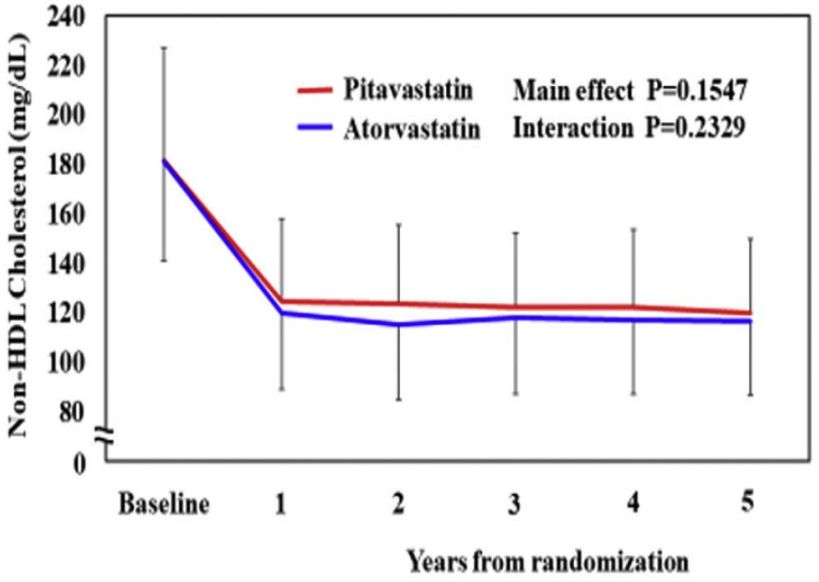
Number of data						
Pitavastatin	291	291	273	260	239	63
Atorvastatin	292	288	278	253	238	87

TC



Number of data						
Pitavastatin	310	305	283	268	250	66
Atorvastatin	306	296	282	262	242	89

Non-HDL



Number of data						
Pitavastatin	304	300	281	267	247	65
Atorvastatin	302	291	280	258	241	87

Int J Cardiol. 2020 S0167-5273(19)34045-8

Atorvastatin have more muscle complaints

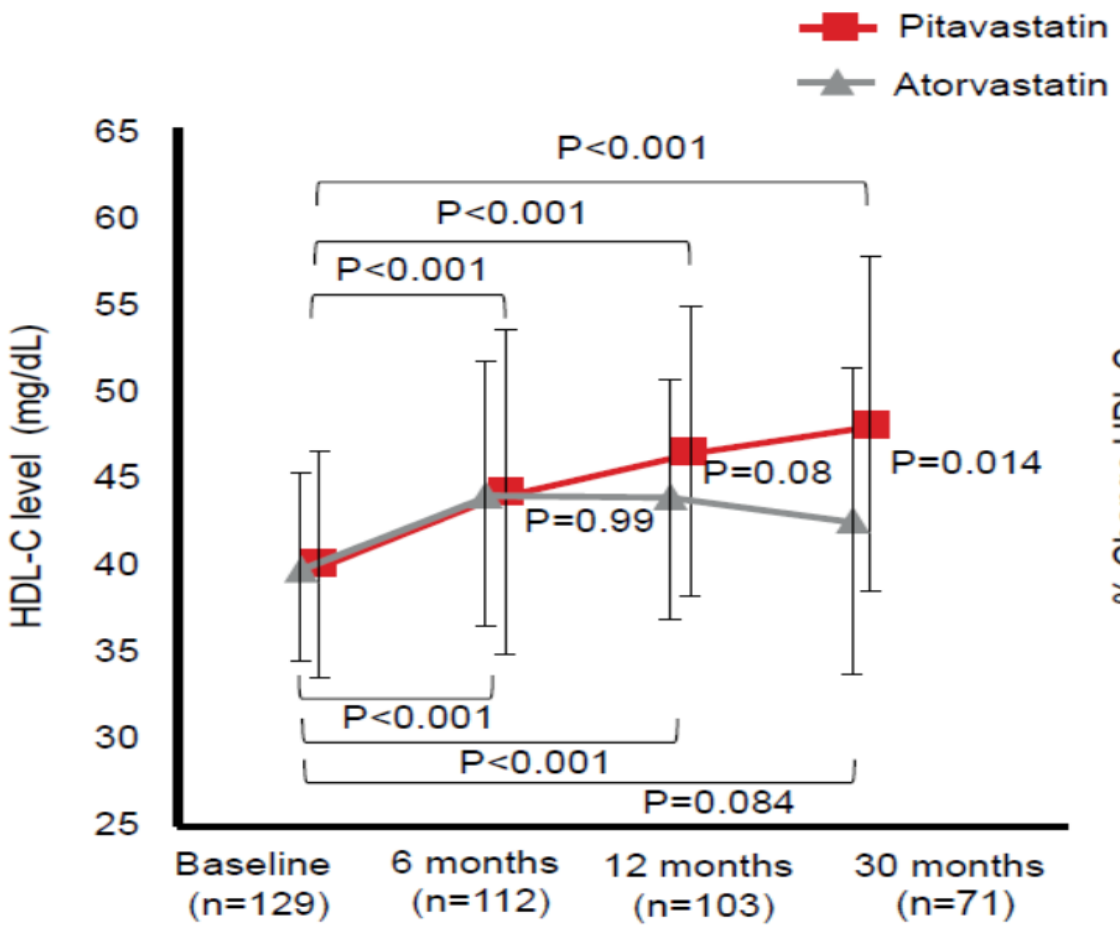
Table 3. Adverse events and laboratory test abnormalities

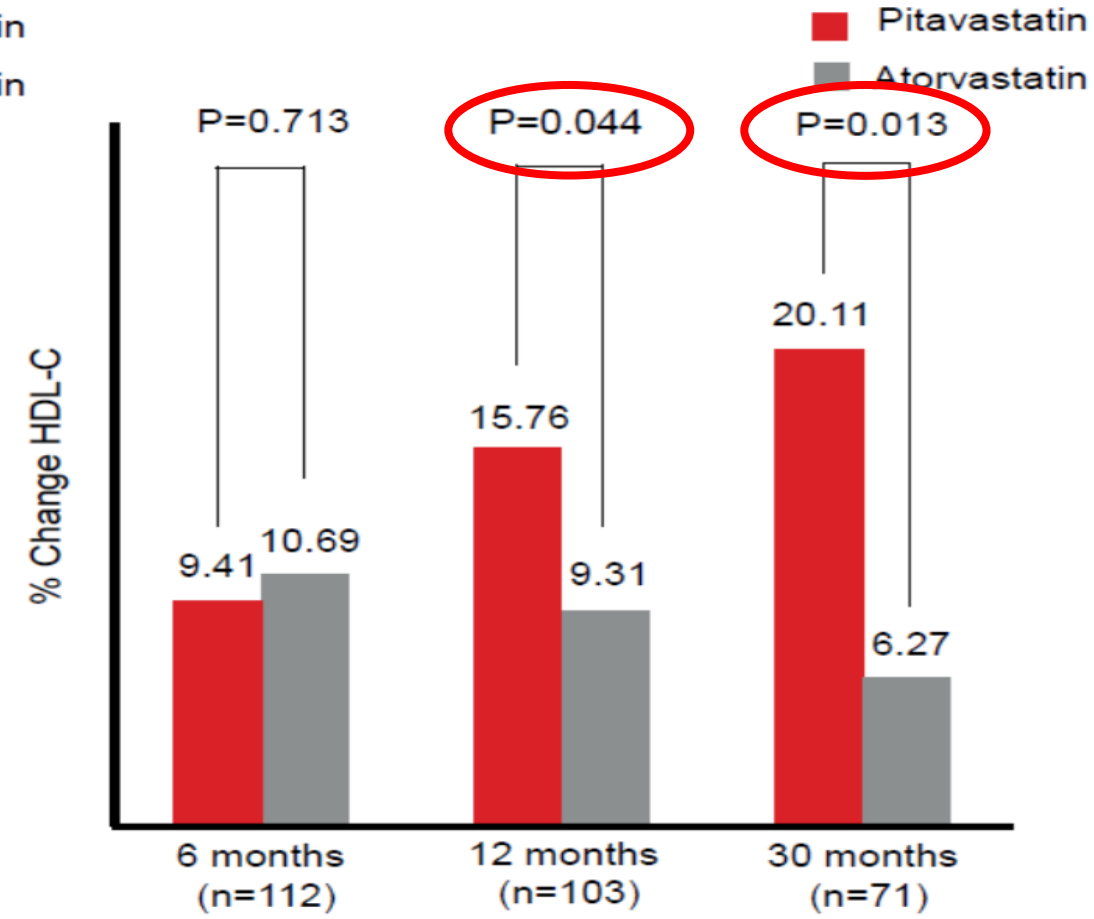
Event	Pitavastatin (n=312), n (%)	Atorvastatin (n=310), n (%)	P value
Adverse events			
Rhabdomyolysis*	1 (0.3)	0	1.000
Muscle complaints	4 (1.3)	12 (3.9)	0.036
Gallbladder-related events	1 (0.3)	1 (0.3)	1.000
Cholecystecomy	0	1 (0.3)	0.498
New onset of diabetes mellitus†	4 (1.3)	5 (1.6)	0.752
Psychiatric disorders	0	2 (0.6)	0.248

Int J Cardiol. 2020 S0167-5273(19)34045-8

Pitavastatin can elevate HDL-C levels

(COMPACT-CAD Study)

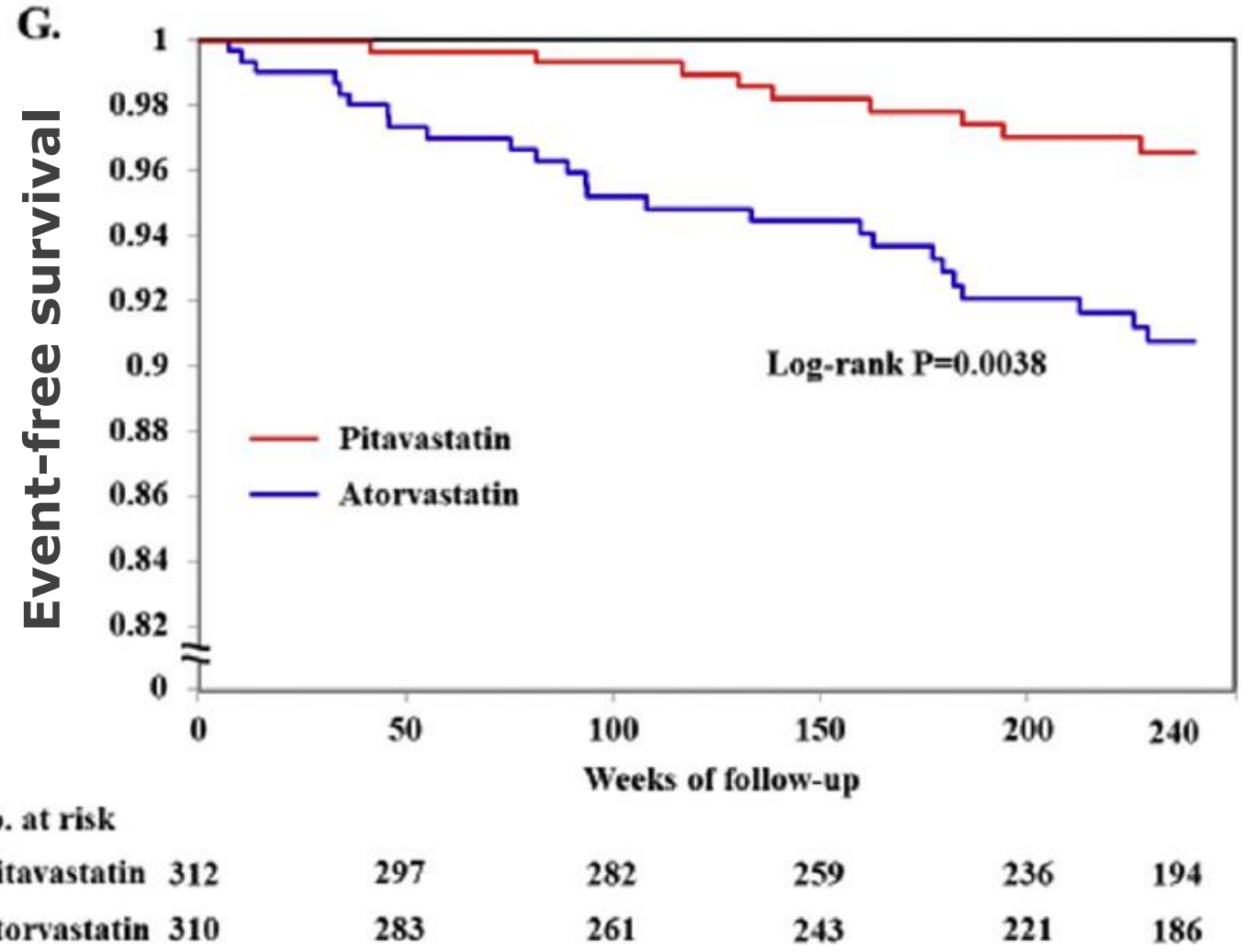




Baseline HDL-C (mg/dL):
Pitavastatin 39.9 ±6.5 Atorvastatin 40.1±5.5

Journal of Cardiology 62 (2013) 87–94

Pitavastatin have better CV outcome



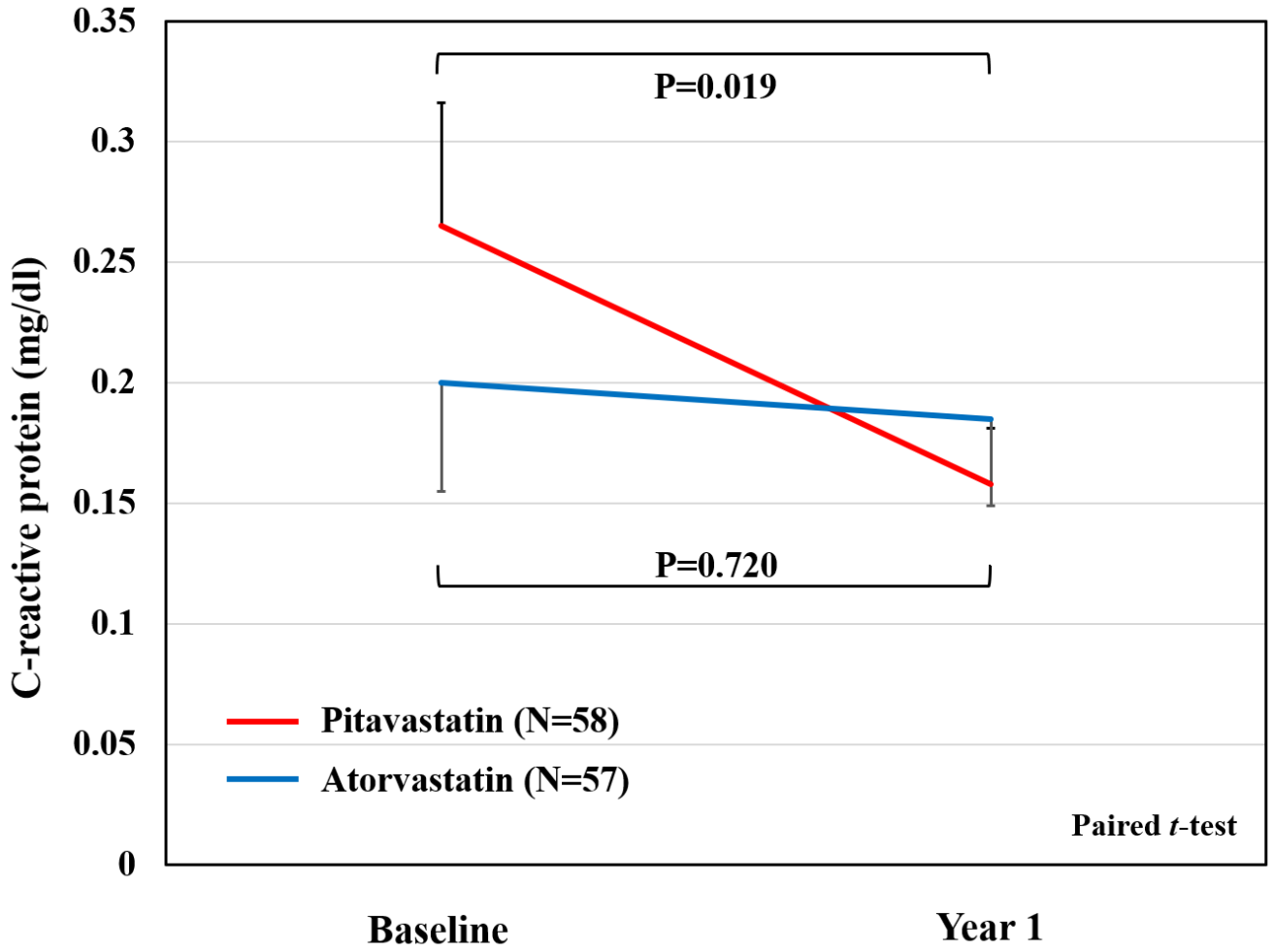
97.1%

91.9 %

Int J Cardiol. 2020 S0167-5273(19)34045-8

Suggesting reason for pitavastatin superior effect

Pleiotropic effects



Int J Cardiol. 2020 S0167-5273(19)34045-8

Original Article

Differential Effects of Atorvastatin and Pitavastatin on Inflammation, Insulin Resistance, and the Carotid Intima-Media Thickness in Patients with Dyslipidemia

Prospective, randomized, open-label trial.146 patients hypercholesterolemia without known cardiovascular disease were randomly assigned to receive **5 mg/day of atorvastatin** (n=73) or **1 mg/day of pitavastatin** for 12 months.

Conclusions:
These data indicate that, while these agents significantly and equally reduce the LDL cholesterol levels, atorvastatin and pitavastatin have different effects on inflammation, insulin resistance, and the progression of carotid atherosclerosis in patients with dyslipidemia.

Table 3. Changes in the clinical characteristics and blood chemistry parameters from baseline to after 12 months of treatment in the patients treated with atorvastatin and pitavastatin

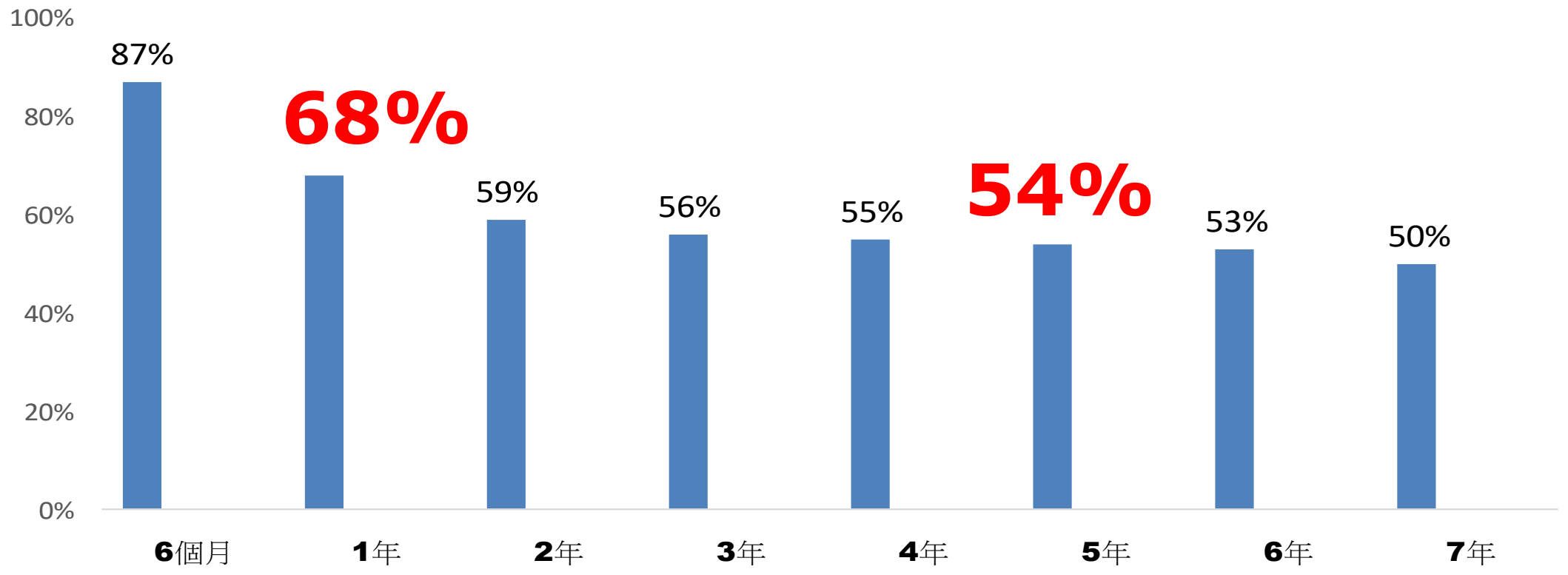
	Atorvastatin ($n=73$)		Pitavastatin ($n=73$)	
	Baseline	At 12 months (the % change)	Baseline	At 12 months (the % change)
Total-C (mg/dL)	244 ± 27	190 ± 25 (−21.3)[&]	247 ± 25	199 ± 22 (−19.5)[&]
LDL-C (mg/dL)	161 ± 29	113 ± 29 (−29.2)[&]	162 ± 23	113 ± 29 (−28.6)[&]
HDL-C (mg/dL)	55 ± 15	55 ± 15 (+1.8)	59 ± 17	62 ± 16 (+6.8)[$ #]
TG (mg/dL)	132 (96, 164)	108 (86, 126) (−17.6)[&]	132 (91, 178)	108 (73, 124) (−15.2)[&]
LDL-C/HDL-C ratio	3.1 ± 0.9	2.2 ± 0.8 (−30.2)[&]	2.9 ± 0.7	2.0 ± 0.5 (−33.0)[$ #]
non HDL-C (mg/dL)	189 ± 27	135 ± 27 (−28.2)[&]	189 ± 21	137 ± 22 (−27.5)[&]
FPG (mg/dL)	104 ± 15	107 ± 18 (+3.3)[$]	104 ± 13	101 ± 14 (−3.1)[$ #]
IRI (μU/mL)	8.6 ± 3.8	9.9 ± 6.0 (+21.9)[&]	8.9 ± 7.3	7.5 ± 5.8 (−9.4)[$ #]
HOMA-IR	2.2 ± 1.2	2.6 ± 1.7 (+25.6)[&]	2.3 ± 2.2	1.9 ± 1.7 (−13.0)[& *]
HbA1c (JDS; %)	5.5 ± 0.6	5.5 ± 0.6 (−0.2)	5.4 ± 0.5	5.3 ± 0.4 (−2.5)[#]
hs-CRP (mg/L)	0.90 (0.35, 2.80)	0.50 (0.24, 2.20) (−23.6)[&]	0.58 (0.38, 2.26)	0.50 (0.24, 2.20) (−32.1)[&]
TNF-α (pg/mL)	12.5 (8.5, 16.0)	9.5 (6.1, 11.3) (−21.1)[&]	15.2 (9.4, 17.3)	9.5 (6.1, 11.3) (−36.0)[& *]
MCP-1 (pg/mL)	105.3 (67.8, 221.0)	78.0 (55.7, 152.3) (−10.9)[&]	133.0 (87.1, 254.0)	78.0 (55.7, 152.3) (−27.9)[& #]
Mean CIMT (mm)	0.884 ± 0.166	0.875 ± 0.180 (−0.4)	0.902 ± 0.196	0.857 ± 0.212 (−4.9)[$ #]

[$] $p<0.05$ vs. baseline, [&] $p<0.01$ vs. baseline [#] $p<0.05$, [*] $p<0.001$ vs. patients treated with atorvastatin at 12 months, The values in *parentheses* represent the percent changes from baseline to after 12 months of treatment, The data are expressed as the mean ± SD or median (interquartile range), C: cholesterol, LDL: low-density lipoprotein, HDL: high-density lipoprotein, FPG: fasting plasma glucose, IRI: immunoreactive insulin, HOMA-IR: homeostasis model assessment of insulin resistance, JDS: Japan Diabetes Society, hs-CRP: high-sensitivity C-reactive protein, TNF: tumor necrosis factor, MCP: monocyte chemoattractant protein, CIMT: carotid intima-media thickness

Outline

1. What's difference in lipid control ?
   - Primary & Secondary prevention
2. Racial difference in statin sensitivity
3. Primary prevention of CVD in lipid treatment
4. Statin Safety & Adherence

Statin, ~70%



*MPR越高表示服藥依順性越好

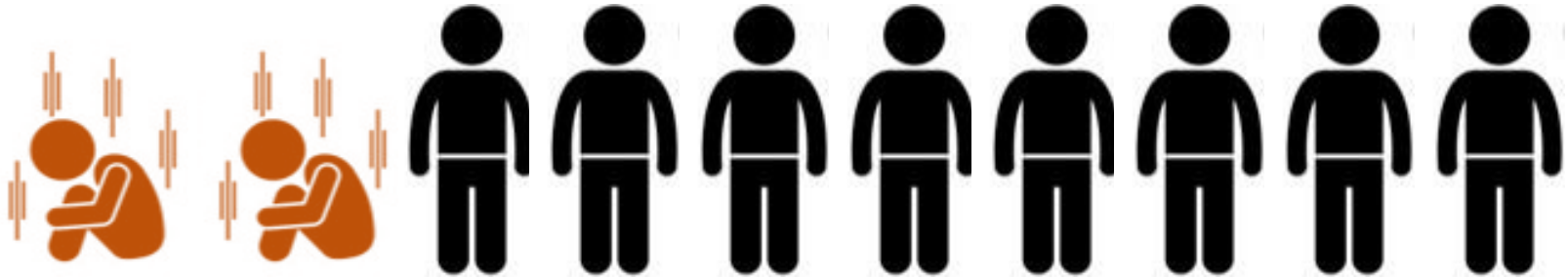
BMC Cardiovasc Disord. 2019 Mar 15;19(1):62.

Myalgia is most common side effect in statin user

2019 STATE study: Experience of patients reporting side effects of statin therapy (N=1500)

2 in 10

Experienced statin-related side effect

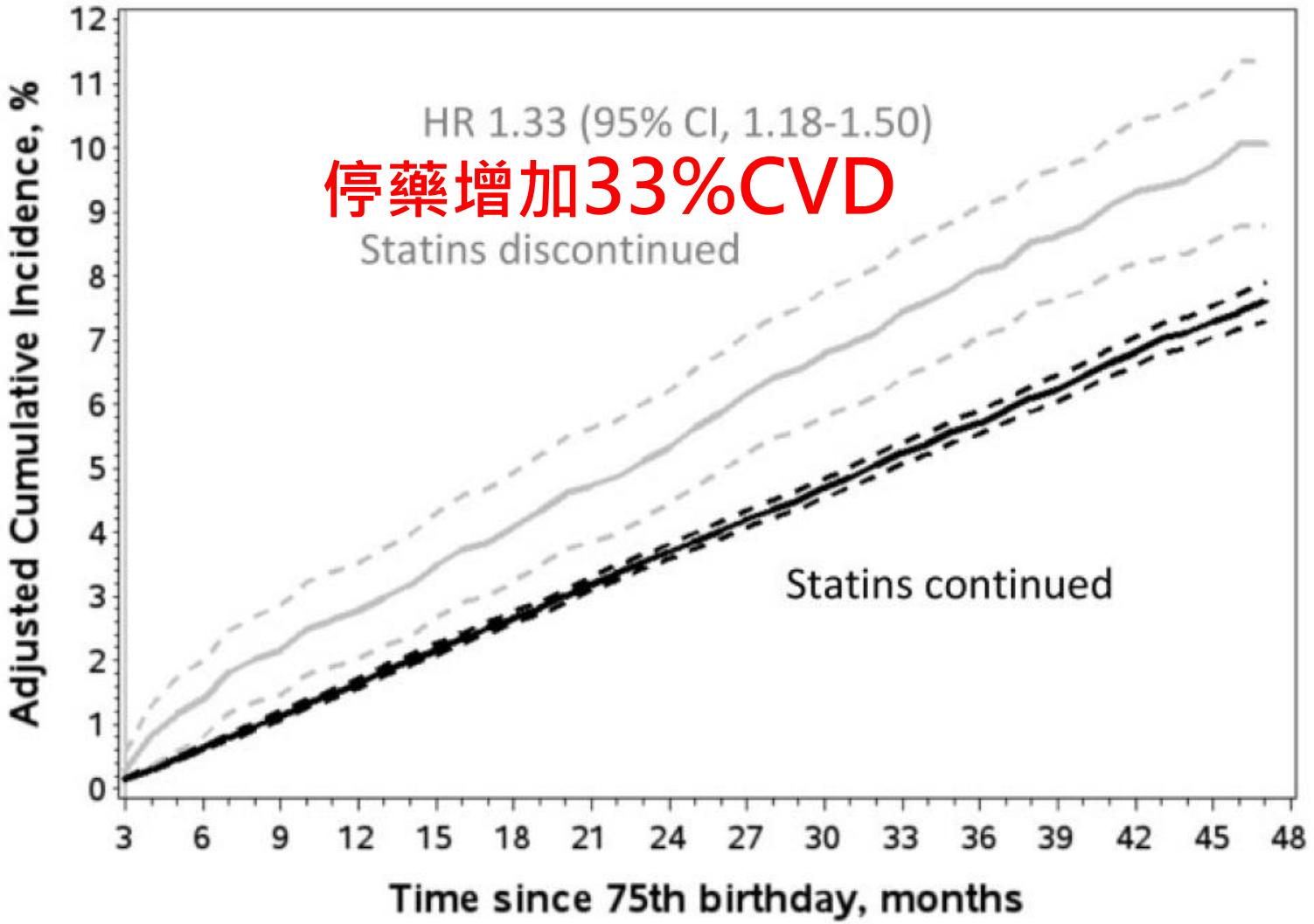


-Muscle aches is the main reason

Journal of Clinical Lipidology (2019) 13, 415–424

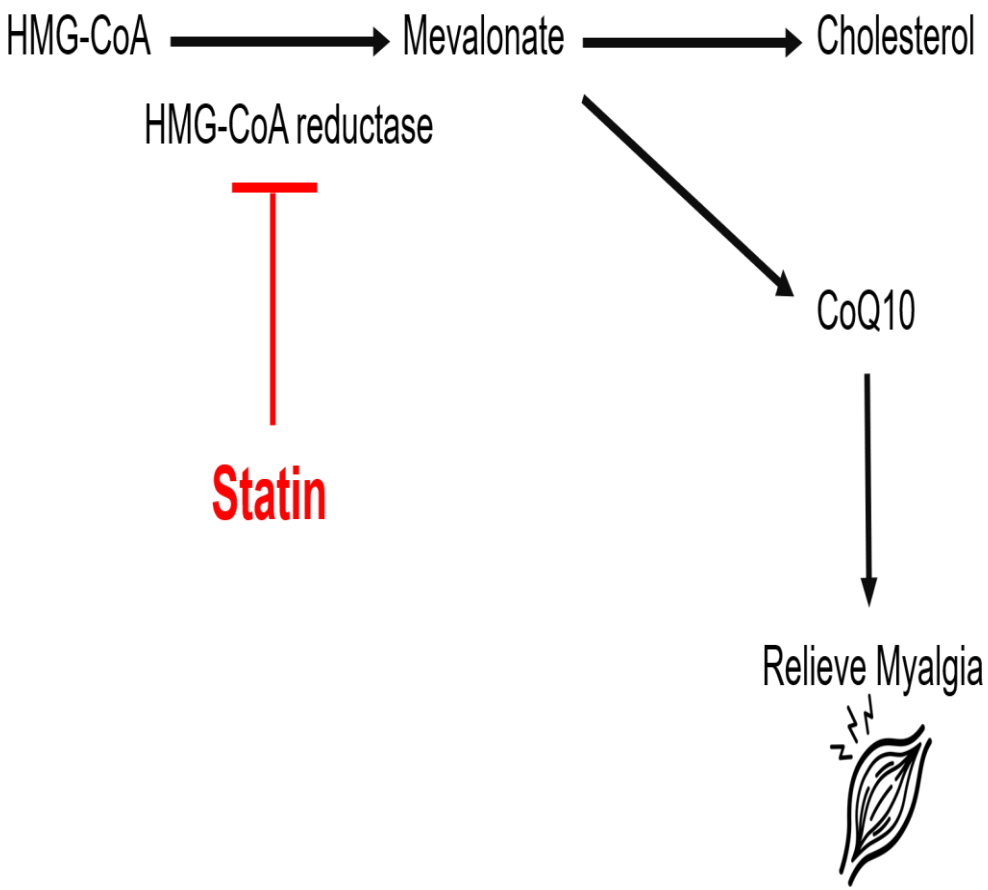
膽固醇治療

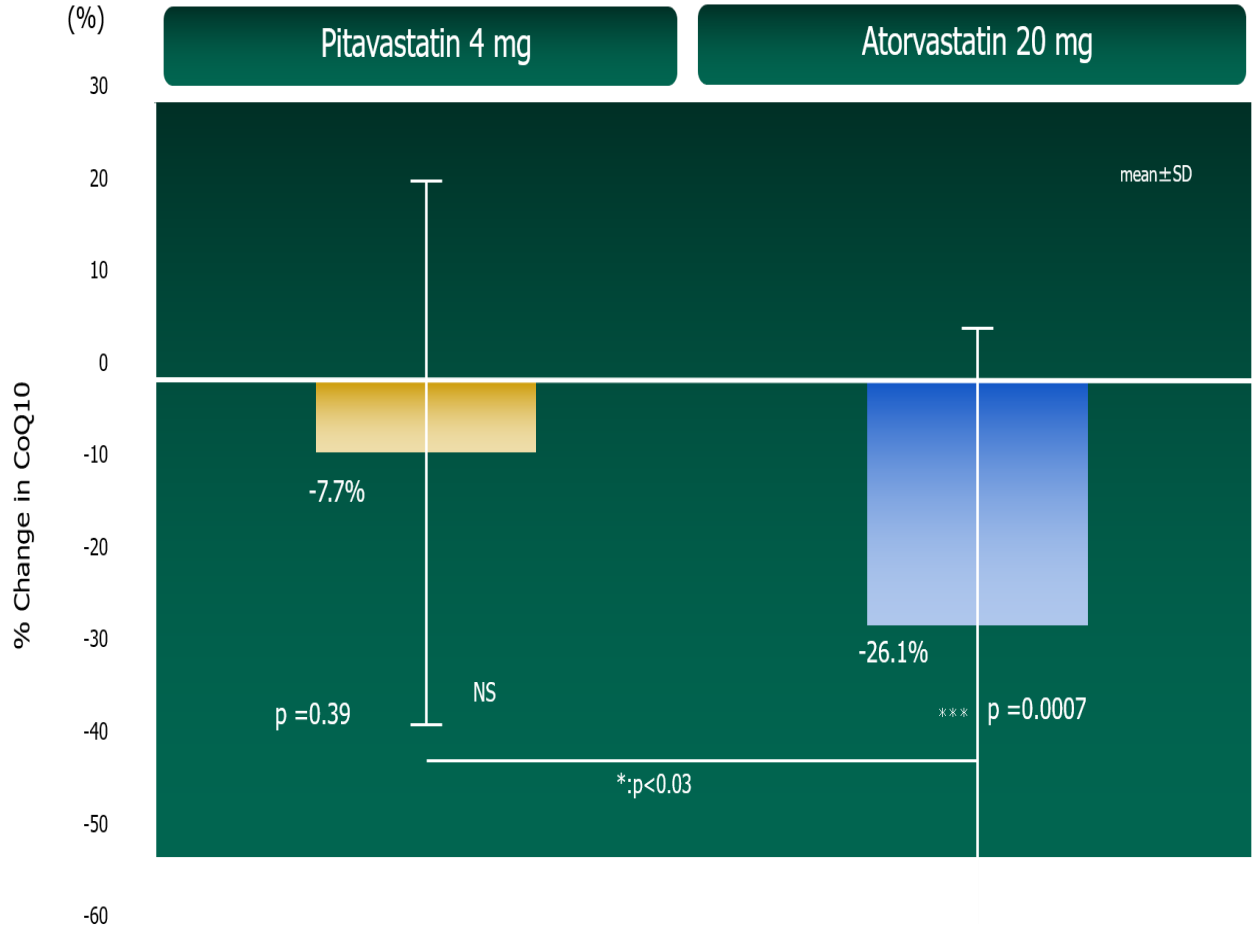
儘量不要停藥



European Heart Journal (2019) 40, 3516–3525

Pitvastatin may have lower myalgia Rate





Clin Pharmacol Ther. 2007, 731-739

NODM

Incidence of NODM according to statin exposure

Table 2

Incidence of NODM according to statin exposure.

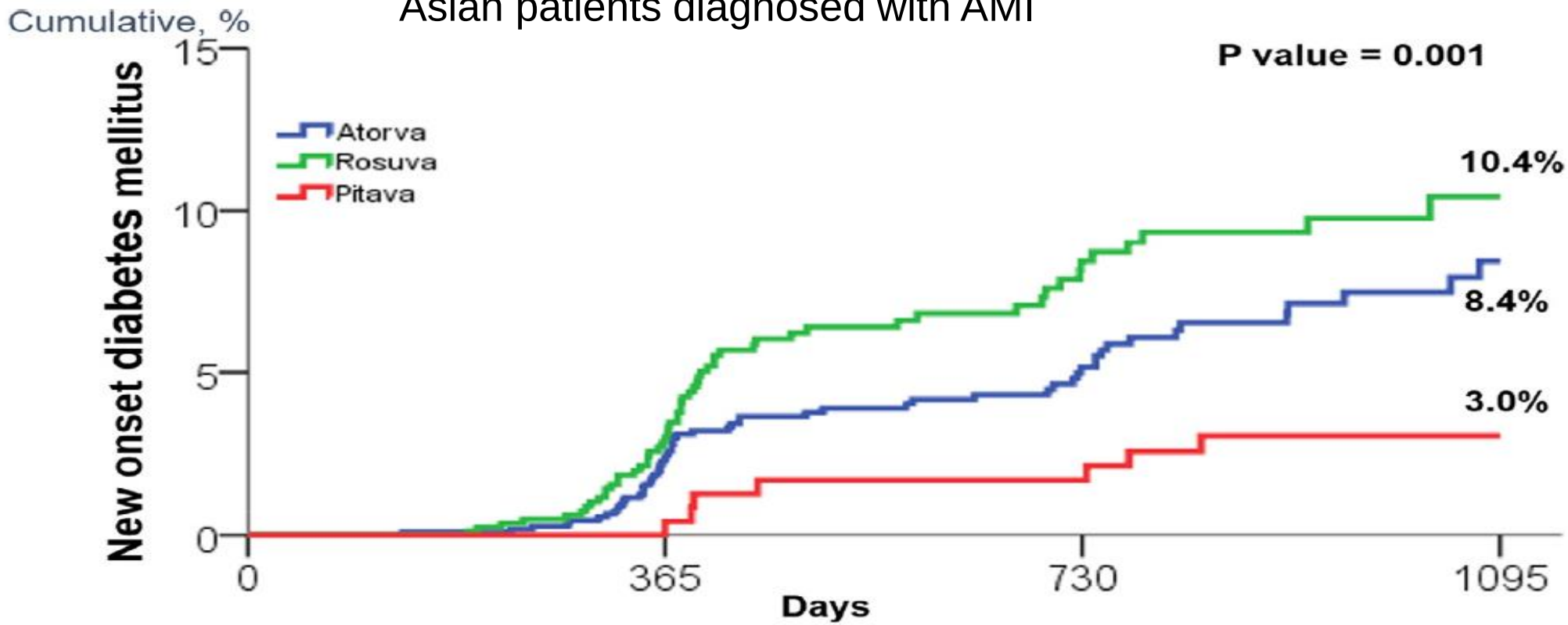
Drug	Incidence, per 1000 PY	Study population, PY
Atorvastatin	4.196	8342
Fluvastatin	4.176	718
Pitavastatin	1.321	757
Pravastatin	4.716	3181
Rosuvastatin	4.770	2935
Simvastatin	6.131	2773
Statin-exposed	6.000	13,669
Matched nonexposed	3.244	55,183

NODM = new-onset diabetes mellitus, PY = patient-years.

Yoon et al. Medicine (2016) 95:46 Medicine.

Pitavastatin have lower NODM rate in AMI patients

2018 Korean prospective, multicenter, real-world treatment, Asian patients diagnosed with AMI



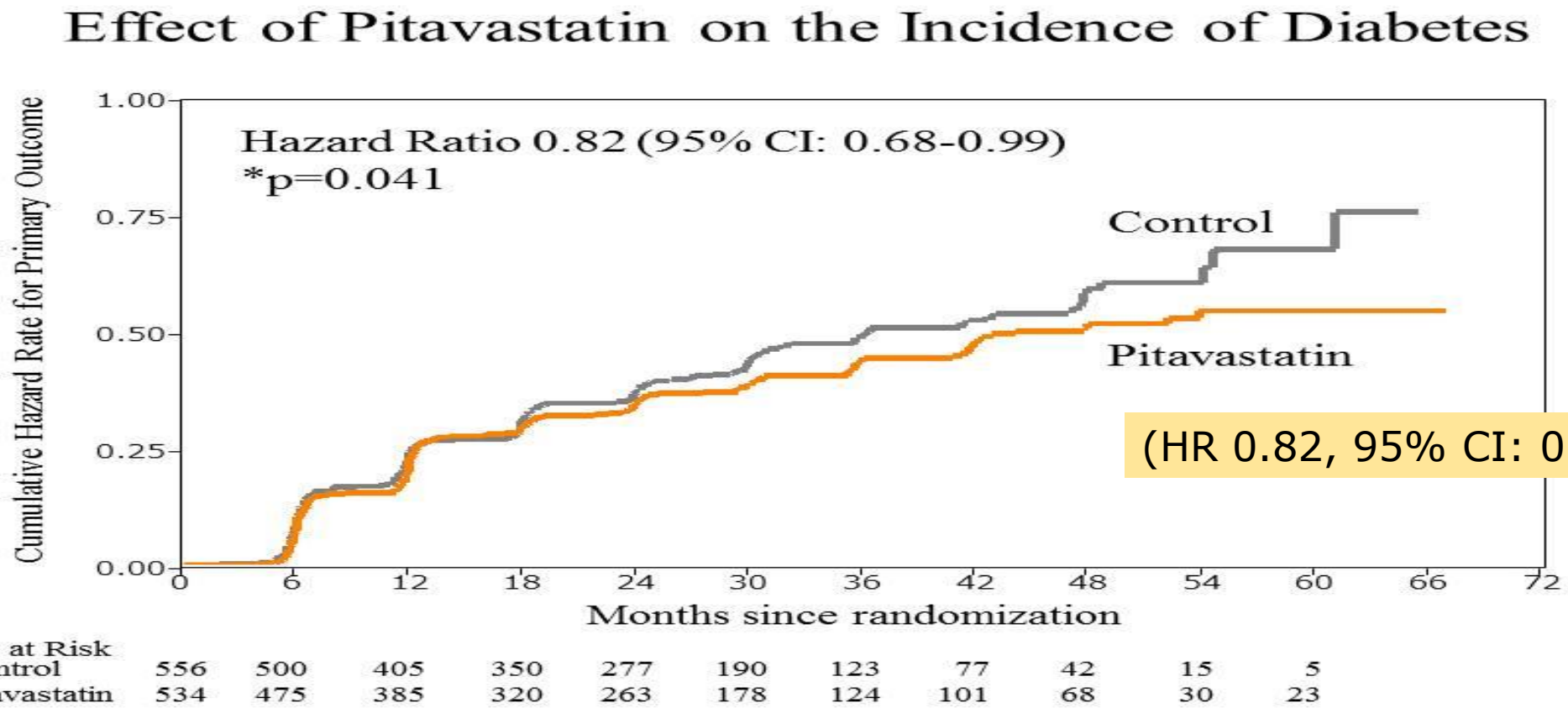
Rosuvastatin 5-10 mg/d

Atorvastatin 10-20 mg/d

Pitavastatin 2-4 mg/d

Choi JY, et al. Am J Cardiol2018;122:922 928

Pitavastatin decrease the development of DM in IGT patients

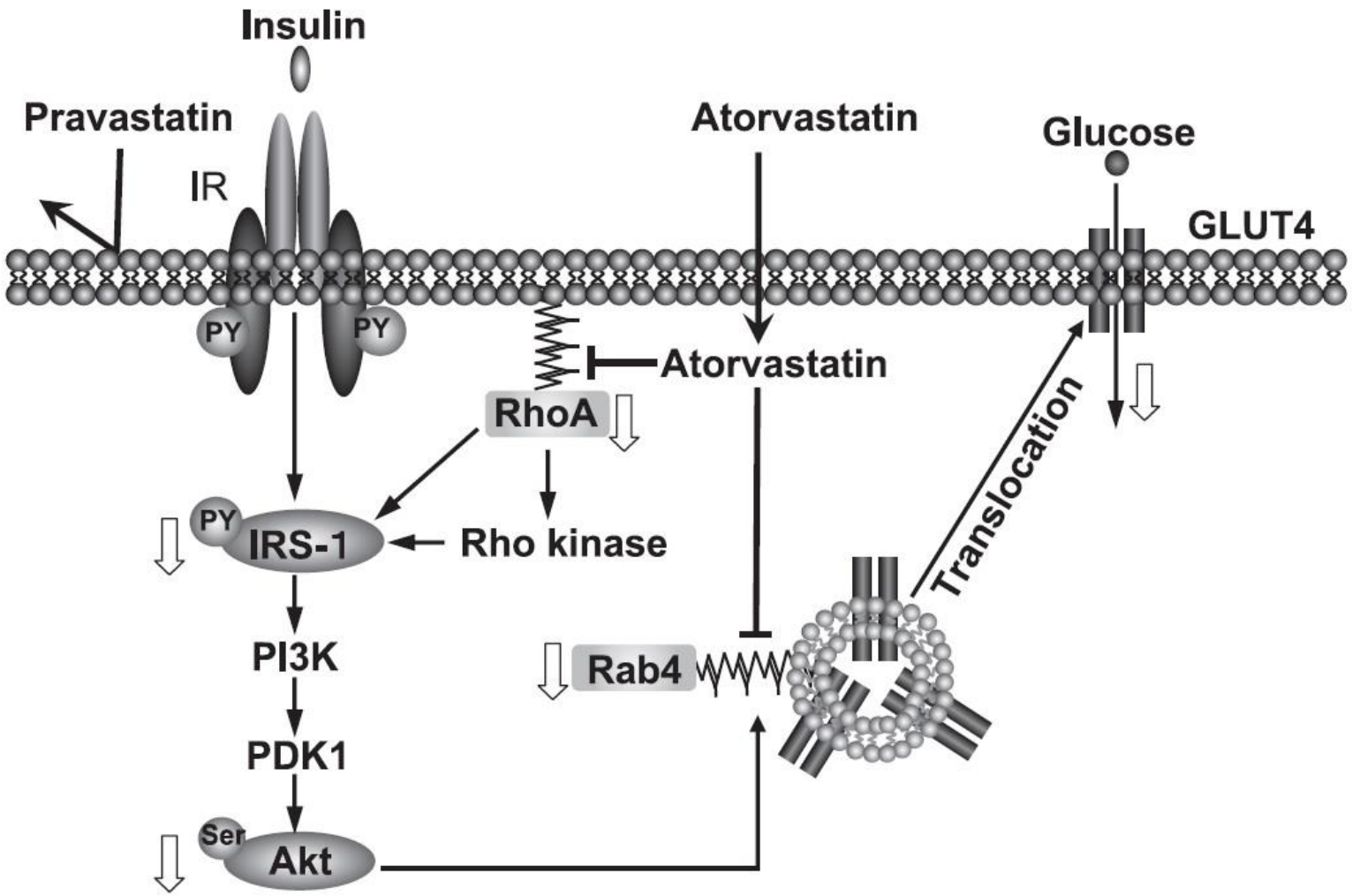


(HR 0.82, 95% CI: 0.68-0.99)

*P value was calculated using a log-rank test that was stratified according to the 5 assignment factors (sex, age, Body mass index, 2-h plasma glucose, and presence of hypertension).

2013 EASD Presentation Abstract Wednesday, Sep 25, 2013, 3:15 PM - 3:30 PM

Atorvastatin inhibits GLUT4 translocation and glucose uptake



Potent statins台灣仿單

Livalo

病患接受HMG-CoA 還原酶抑制劑治療後，曾有醣化血色素/或空腹血漿血糖上升情況，**但依上市後安全監測或預測性研究，pitavastatin 並未有明確造成糖尿病徵兆**

Lipitor

醣化血色素上升，病患接受HMG-CoA 還原酶抑制劑治療後，曾有醣化血色素/或空腹血漿血糖上升情況

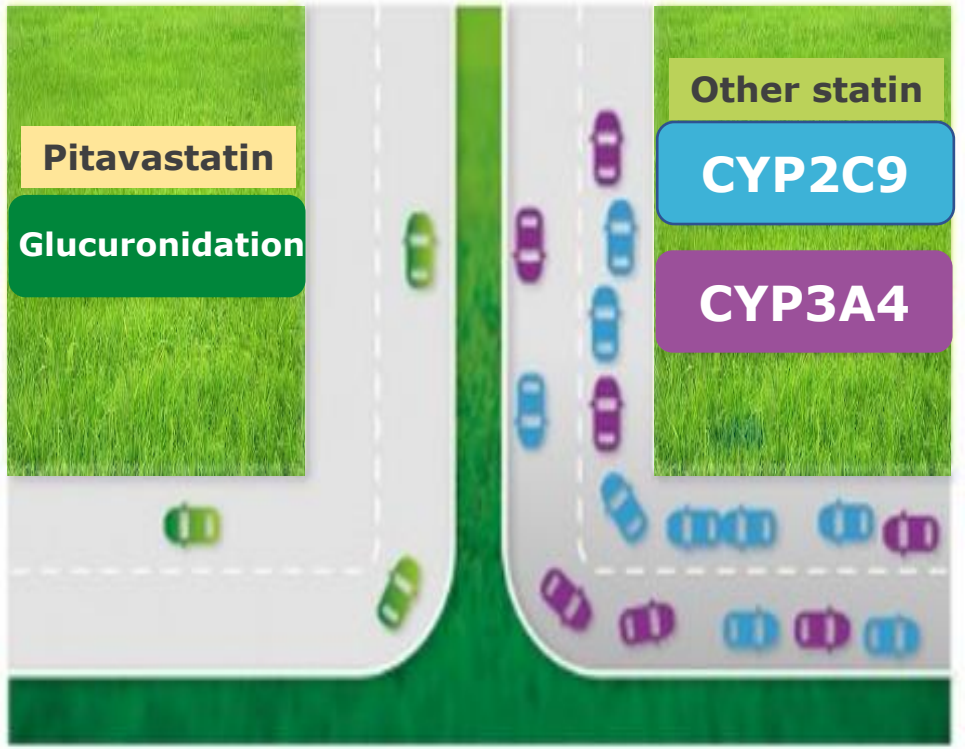
Crestor

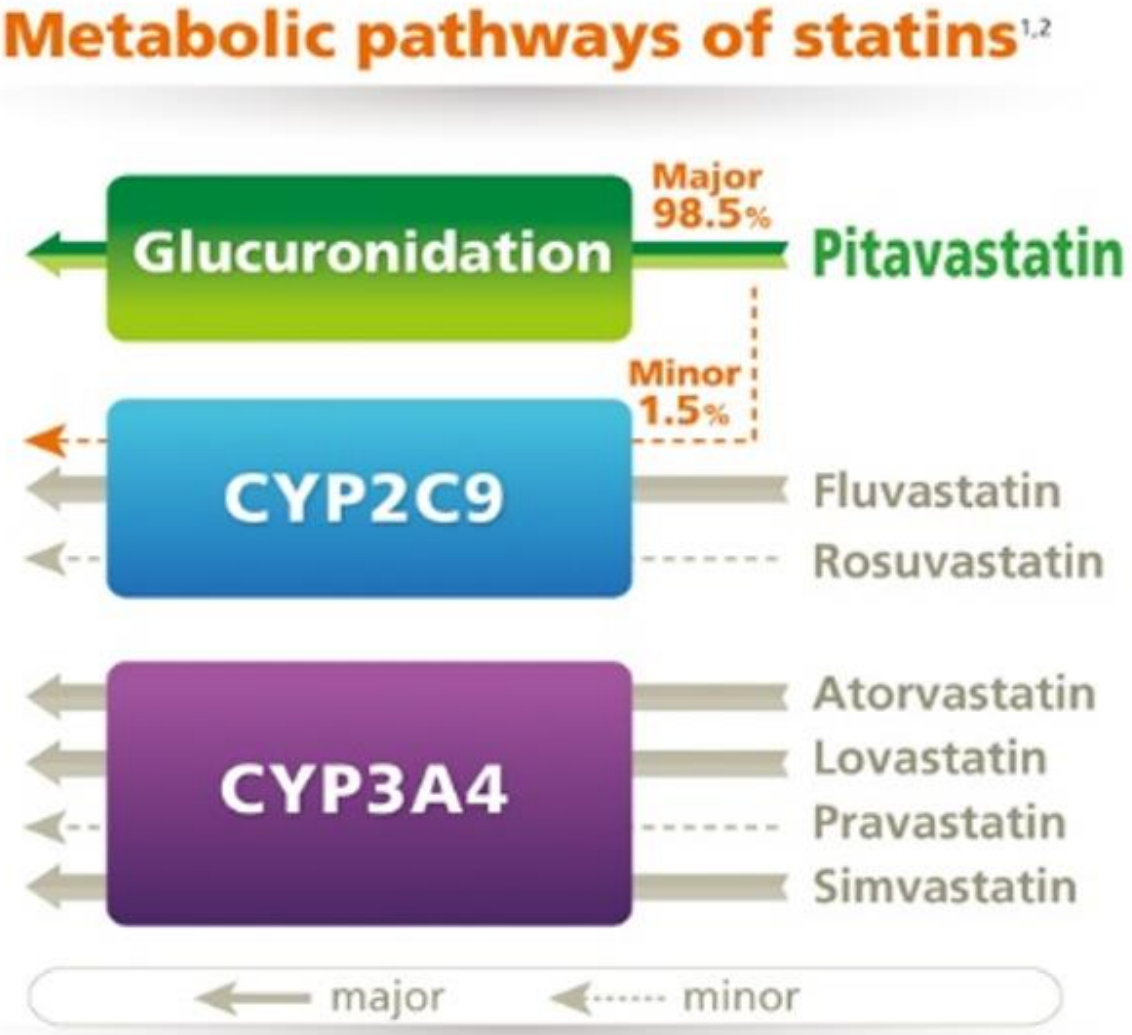
醣化血色素上升，病患接受HMG-CoA 還原酶抑制劑治療後，曾有醣化血色素/或空腹血漿血糖上升情況。使用 HMG-CoA 還原酶抑制劑(包括Crestor) 曾有HbA_{1c}和空腹血糖值增加的報告

Metabolic pathway of statins

Statin	Metabolism
Pitavastatin	Glucuronidation 2C9 minimally
Atorvastatin	3A4
Fluvastatin	2C9 (major)
Lovastatin	3A4
Pravastatin	Sulfation
Rosuvastatin	2C9
Simvastatin	3A4

Pitavastatin: unique metabolic profile
Minor metabolism via CYP pathways

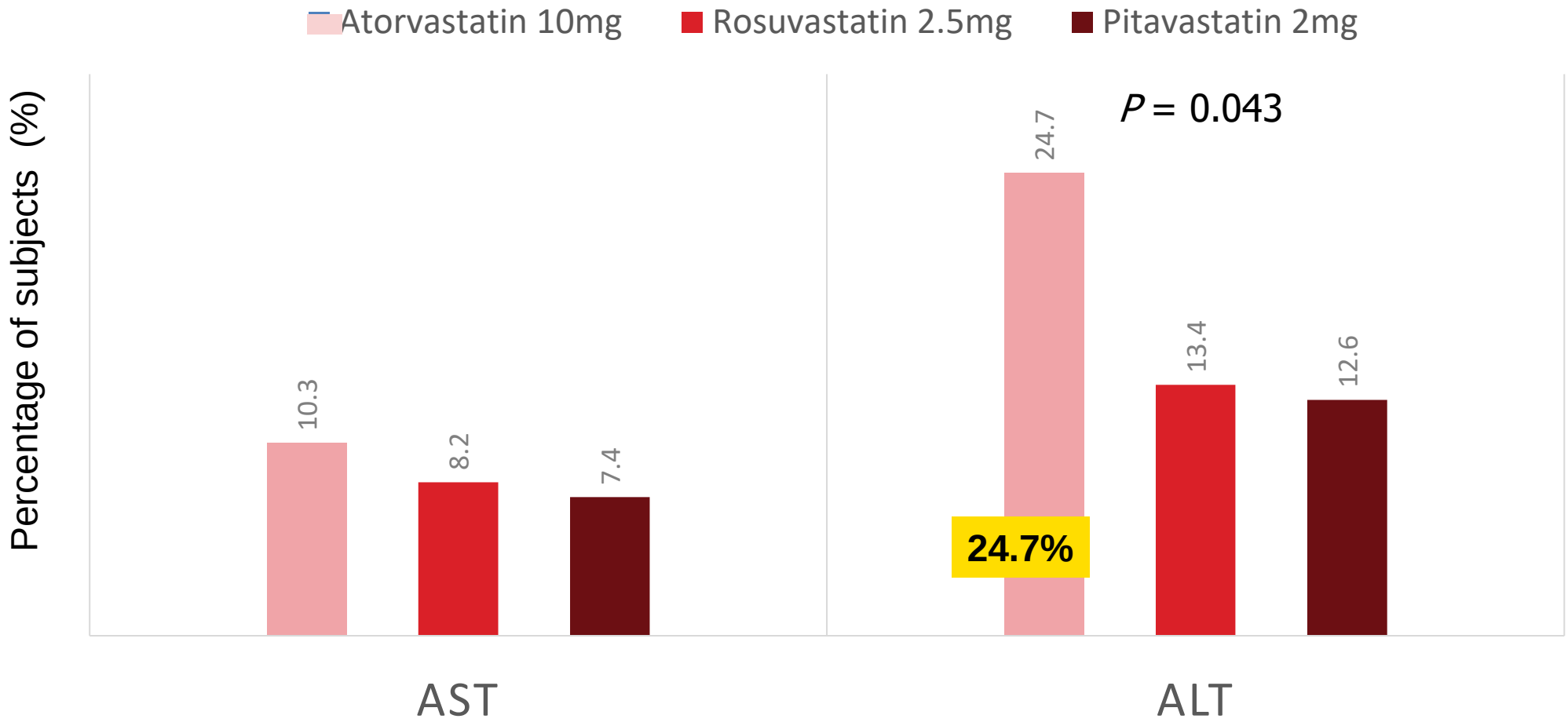




ADR=adverse drug reaction; CYP=cytochrome P450; DDI=drug-drug interaction.

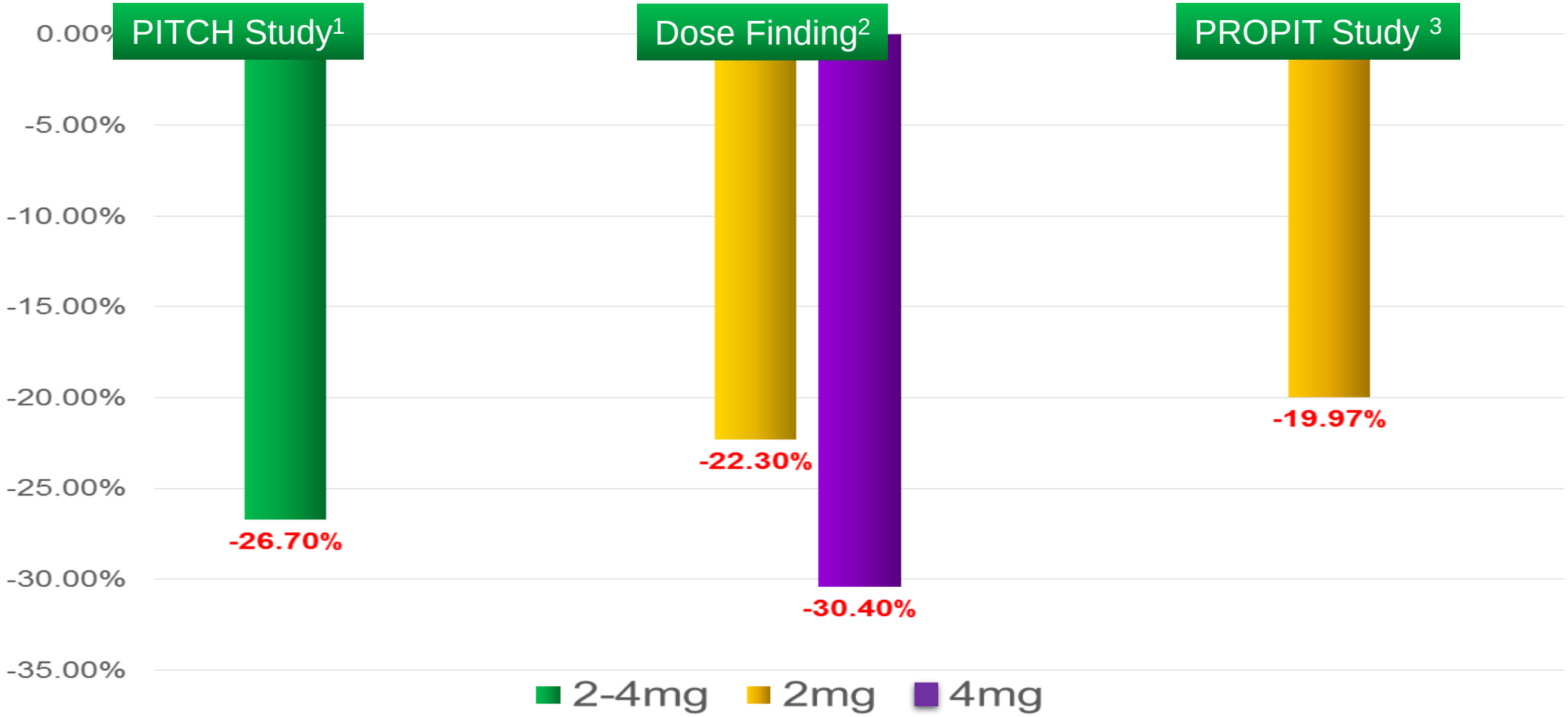
1. Corsini A, Ceska R. Curr Med Res Opin. 2011;27(8):1551-62. 2. Kawai Y, et al. Drug Des Devel Ther. 2011;5:283-97.

PATROL study:
Atorvastatin have more patients experienced in ALT abnormality

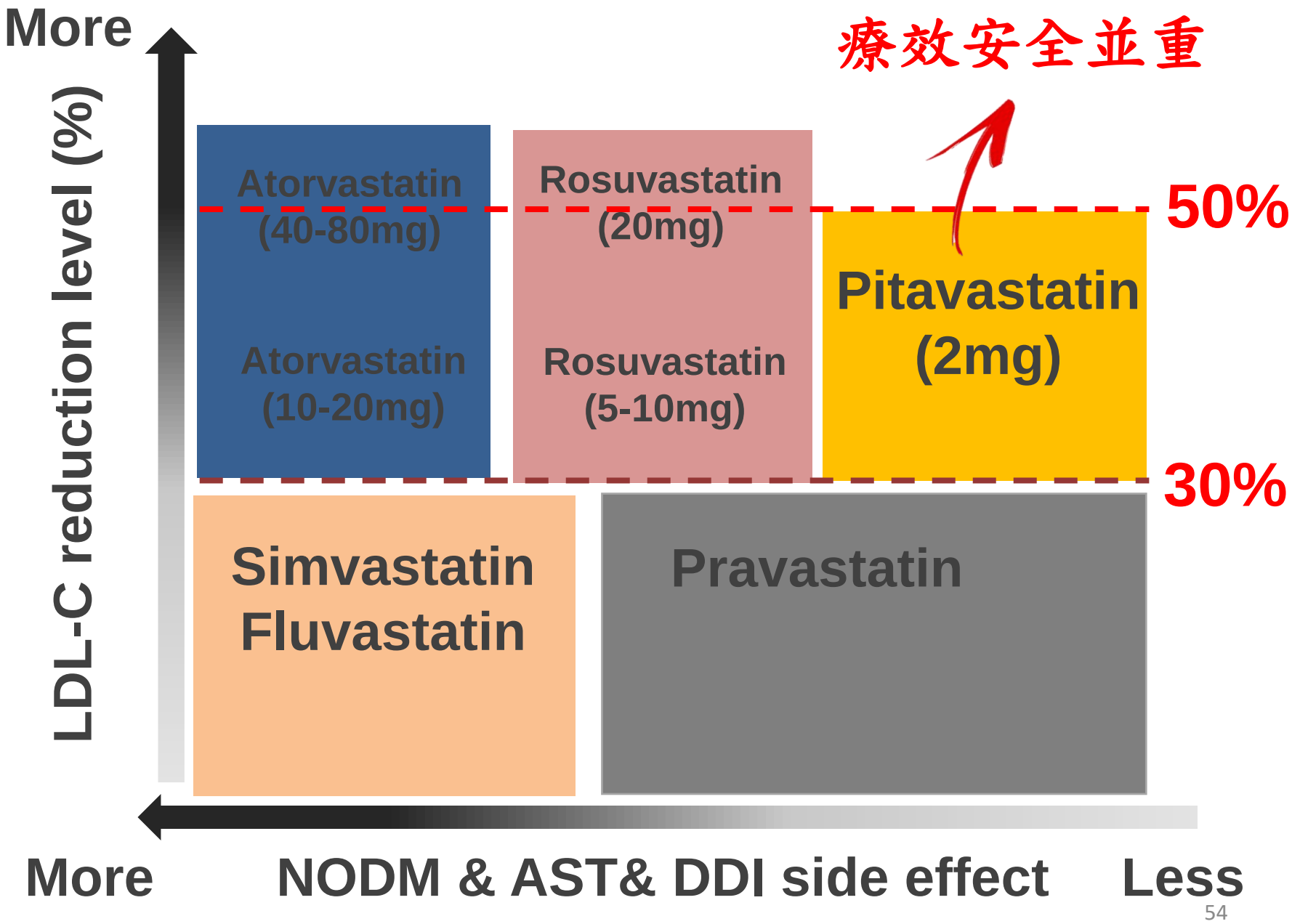


Circ J 2011; 75: 1493–1505

Efficacy of Pitavastatin on TG



1.J of Clin. Lipidol. 2012, vol.6, 340-351
2.Drug Res. 2002,vol.52,NO.4 : 251-255
3.Clin. Endo. 2014,vol.82,NO.5 : 670-677


More
LDL-C reduction level (%)
療效安全並重
Atorvastatin
(40-80mg)
Atorvastatin
(10-20mg)
Rosuvastatin
(20mg)
Rosuvastatin
(5-10mg)
Pitavastatin
(2mg)
50%
30%
Simvastatin
Fluvastatin
Pravastatin
More
NODM & AST& DDI side effect
Less

ESC/EAS Guideline for the management Of dyslipidemias - Summary

	Pitava	Atorva	Rosuva	Prava	Fluva	Simva
TG lowering	**V**	V	V	-	-	-
Lower risk of Myopathy	**V**	-	V	V	-	-
Non-CYP via	**V**	-	V	V	-	-
CKD preferred	**V**	V	-	-	V	-
HIV	**V**	-	V	V	V	-

Modified from European Heart Journal 2011, 32, 1769- 1818

.

Take Home Message

1. 亞洲人對Statin 有較佳反應與療效。使用Moderate intensity statin 即可有效達標
2. Primary Prevention CVD 的血脂控制，需長時間服用才能凸顯藥物效果。更應重視藥物安全性與遵醫囑性。
3. Statin 可有效降低LDL-C ，有效降低TG、提升HDL-C。

,不易造成新生糖尿病、Myalgia 機率較低、藥物交互作用較低者 ,較適合用來長期CVD Primary Prevention之血脂控制。


Thank you for your attention
Mackay Memorial Hospital Tamsui branch