D vitamīns un hipertensija, kardiovaskulārie notikumi, dislipidēmija – kas jauns 2021. gadā?

Asoc. prof. Kārlis Trušinskis
Latvijas Kardioloģijas centrs, Paula Stradiņa Klīniskā universitātes slimnīca
Rīgas Stradiņa universitāte

HA
dzīve ir tā vērta

LATVIJAS HIPERTENSIJAS UN ATEROSKLEROZES BIEDRĪBA

Asinsspiediena kontrole pasaulē

Long-term and recent trends in hypertension awareness, treatment, and control in 12 high-income countries: an analysis of 123 nationally representative surveys

*NCD Risk Factor Collaboration (NCD-RisC)**

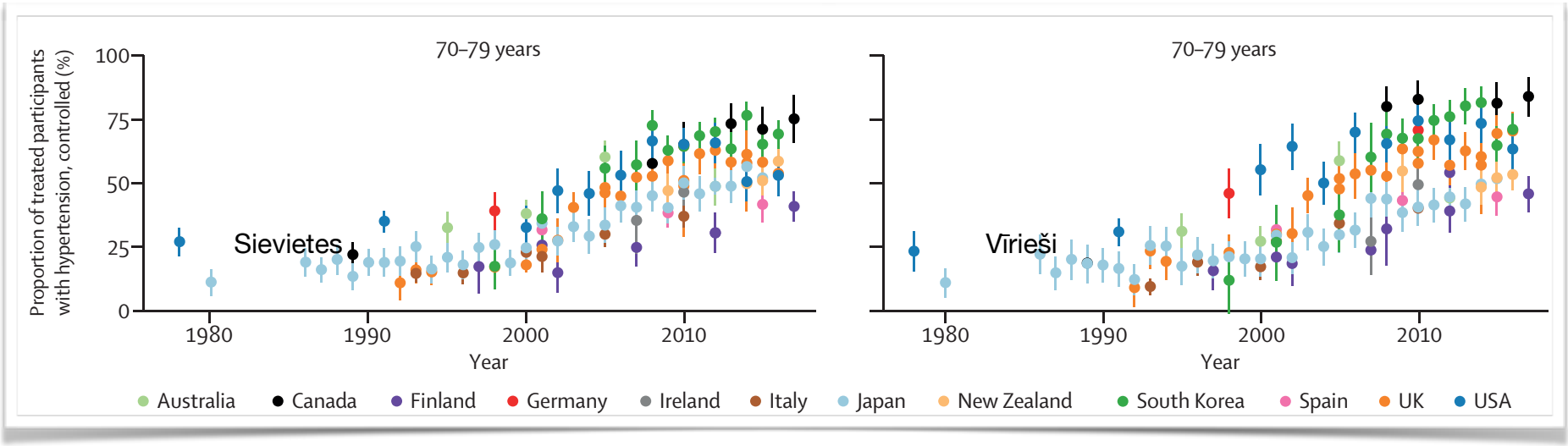
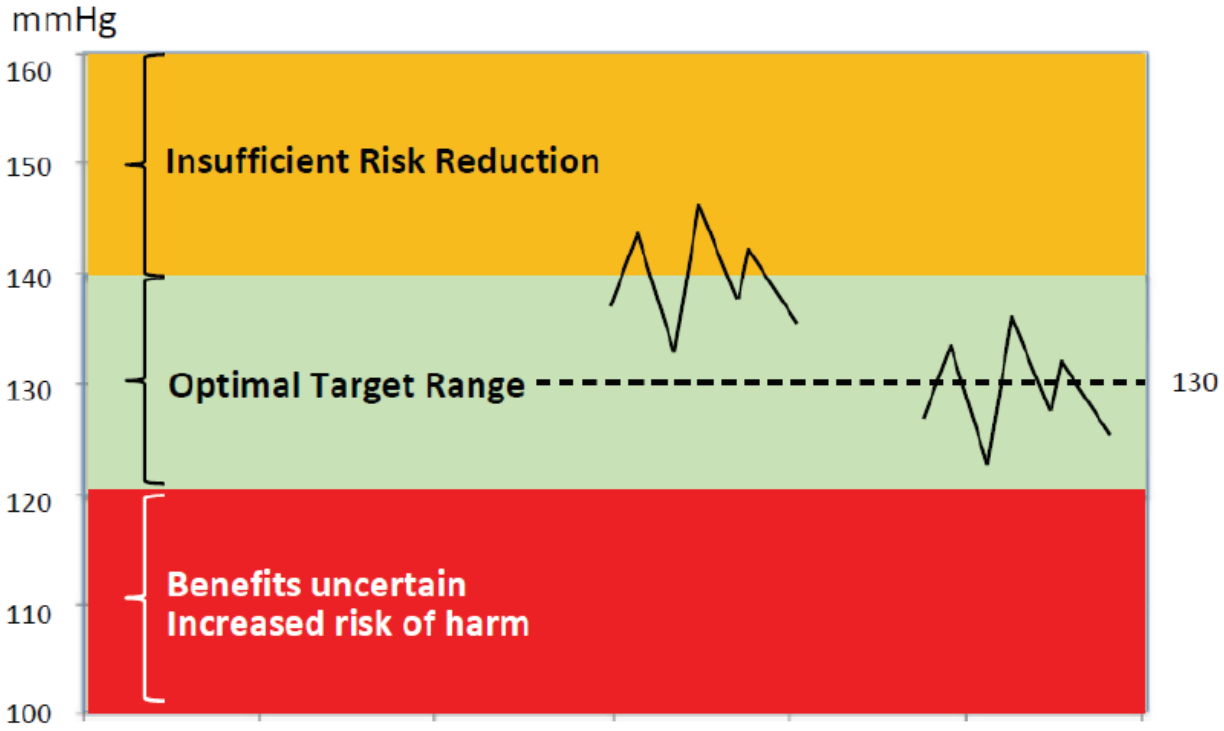


Figure 6: **Trends in hypertension control, by country, sex, and age group**
See appendix (pp 29–41) for country-by-country results. Error bars indicate 95% CIs.

Princips asinsspiediena samazināšanai



Target Range below 140mmHg, aiming for 130mmHg

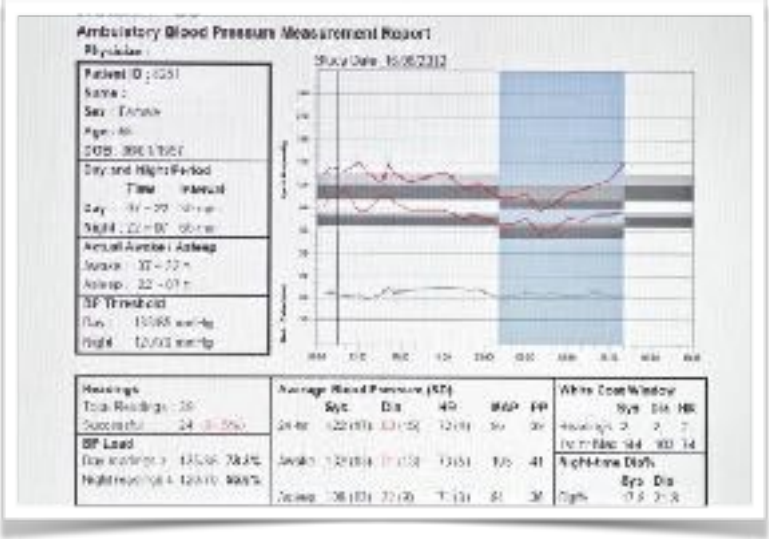
Hipertensijas diagnostika: ambulatorā asinsspiediena monitorēšana

ESC European Society of Cardiology
European Heart Journal (2018) **00**, 1–98
doi:10.1093/eurheartj/ehy339

ESC/ESH GUIDELINES

2018 ESC/ESH Guidelines for the management of arterial hypertension

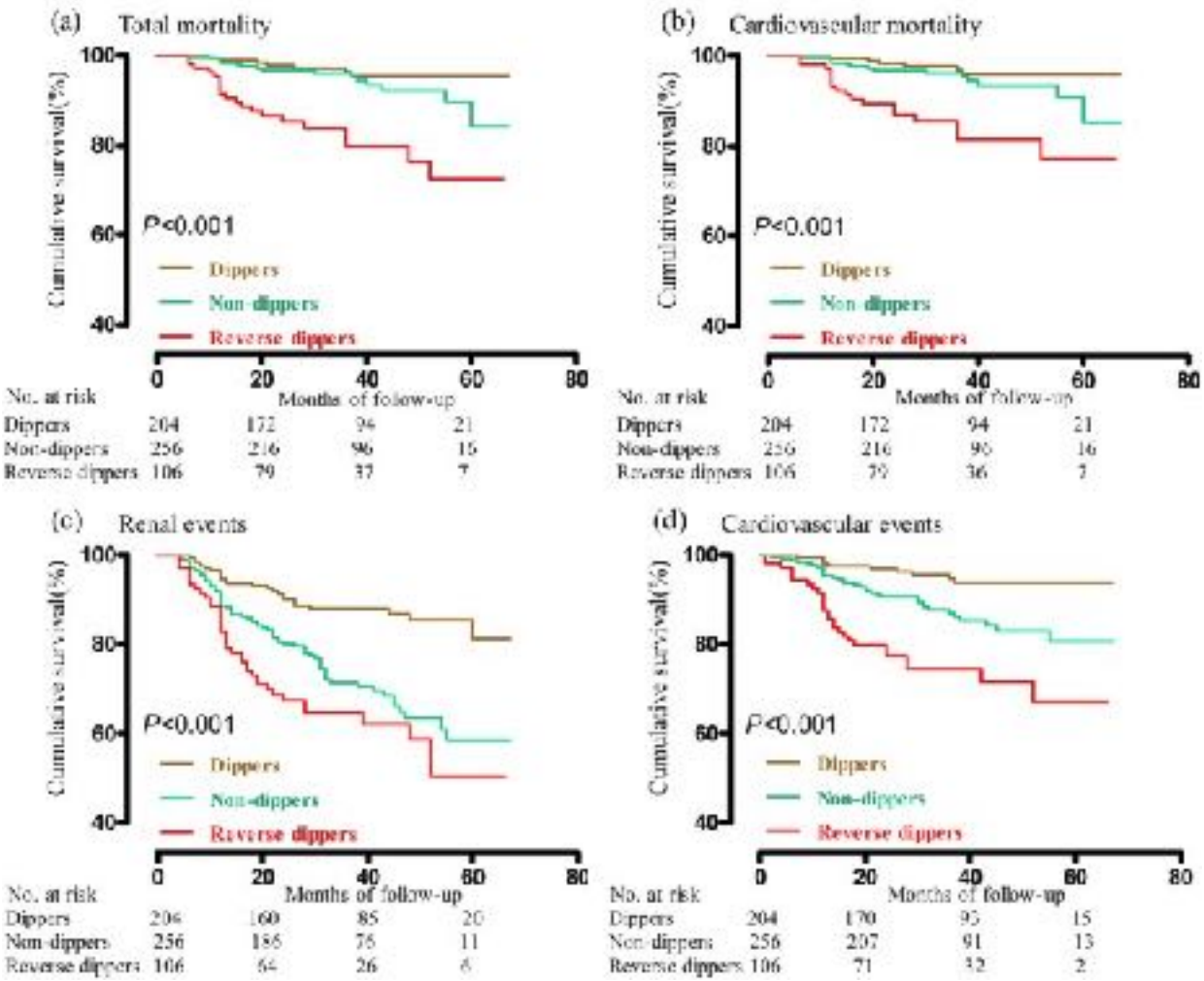
The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH)



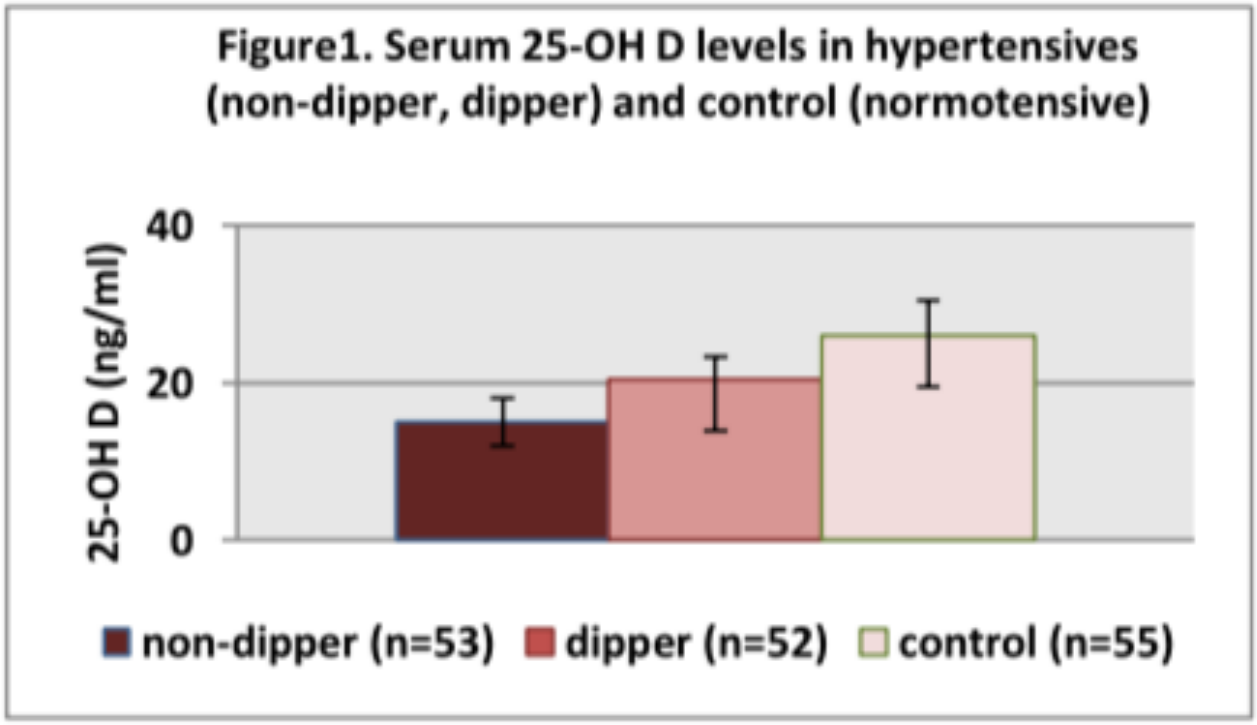
Changes in recommendations	
2013	**2018**
Diagnosis	**Diagnosis**
Office BP is recommended for screening and diagnosis of hypertension.	It is recommended to base the diagnosis of hypertension on: • Repeated office BP measurements; or • Out-of-office BP measurement with ABPM and/or HBPM if logistically and economically feasible.

Prognostic Value of Reverse Dipper Blood Pressure Pattern in Chronic Kidney Disease Patients not Undergoing Dialysis: Prospective Cohort Study

Cheng Wang[1,*], Zengchun Ye[1,*], Yan Li[2,*], Jun Zhang[1], Qunzi Zhang[1], Xinxin Ma[1], Hui Peng[1] & Tanqi Lou[1]



Vitamin D plasma concentration and VDR gene polymorphisms in patients with non-dipper hypertension



IS. Tatarnikova[1], LA. Shpagina[2], ON. Gerasimenko[2]

(1) *Institute of Biological Chemistry and Fundamental Medicine*, Novosibirsk, Russian Federation

(2) *Novosibirsk State Medical University*, Novosibirsk, Russian Federation

Journal of Steroid Biochemistry & Molecular Biology 175 (2018) 29–43

Contents lists available at ScienceDirect

Journal of Steroid Biochemistry & Molecular Biology

journal homepage: www.elsevier.com/locate/jsbmb

Review

Vitamin D and cardiovascular diseases: Causality

Sunil J. Wimalawansa*

Professor of Medicine, Endocrinology & Nutrition, Cardio Metabolic Institute, NJ, USA

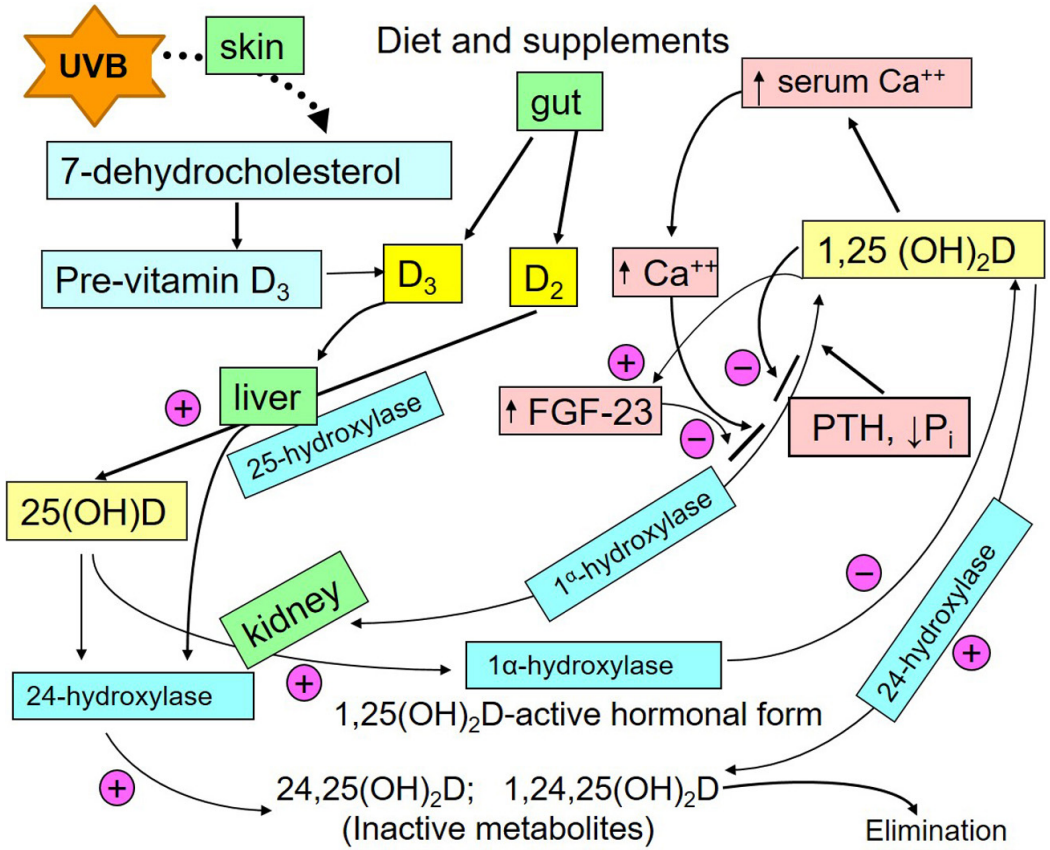


Fig. 1. Metabolic activation of vitamin D.
The generation of pre-vitamin D in the skin from the precursor 7-dehydrocholesterol, following skin exposure to UVB is illustrated. Pre-vitamin D together with the vitamin D absorbed via the gastrointestinal tract are transported to the liver, where 25-hydroxylase enzyme (CYP24A1) converts it to 25(OH)D, the body's storage form of vitamin D. 1α-hydroxylase enzyme (CYP27B1) is predominantly located in renal tubules (also present in other cells, such as in macrophage), converts 25(OH)D into its active hormonal form, 1,25(OH)D. Any excess vitamin D is converted to an inactive metabolite through 24-hydroxylation.

Dermato-Endocrinology
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/kder20

Influence of drugs on vitamin D and calcium metabolism

Uwe Gröber[a] & Klaus Kisters[b]
[a] Academy of Micronutrient Medicine; Essen, Germany
[b] Westphalian Wilhelms University Münster; Academy of Micronutrient Medicine and Hypertension Excellence Center (ESH); Medizinische Klinik I; Herne, Germany
Published online: 01 Apr 2012.

Vitamin D-deficiency
(25-OH-D < 20 ng/ml)

Renin ↑

Parathormone ↑

Angiotensin II ↑

Intracellular calcium activity ↑

Aldosterone ↑

Vasoconstriction ↑

Inhibition of GLUT-4

Synthesis of Adipocytes ↑

Na^+/H_2O-Retention ↑

Insulin-induced uptake of glucose ↓

Risk for hypertension and/or preeclampsia ↑

Risk for obesity and/or insulinresistance ↑

International Journal of
Molecular Sciences

MDPI

Review

The Impact of Vitamin D in the Treatment of Essential Hypertension

Christian Legarth [1], Daniela Grimm [1,*], Markus Wehland [2], Johann Bauer [3] and Marcus Krüger [2]

[1] Institute of Biomedicine, Pharmacology, Aarhus University, Wilhelm Meyers Allé 4, DK-8000 Aarhus C, Denmark; chr_brod@hotmail.com

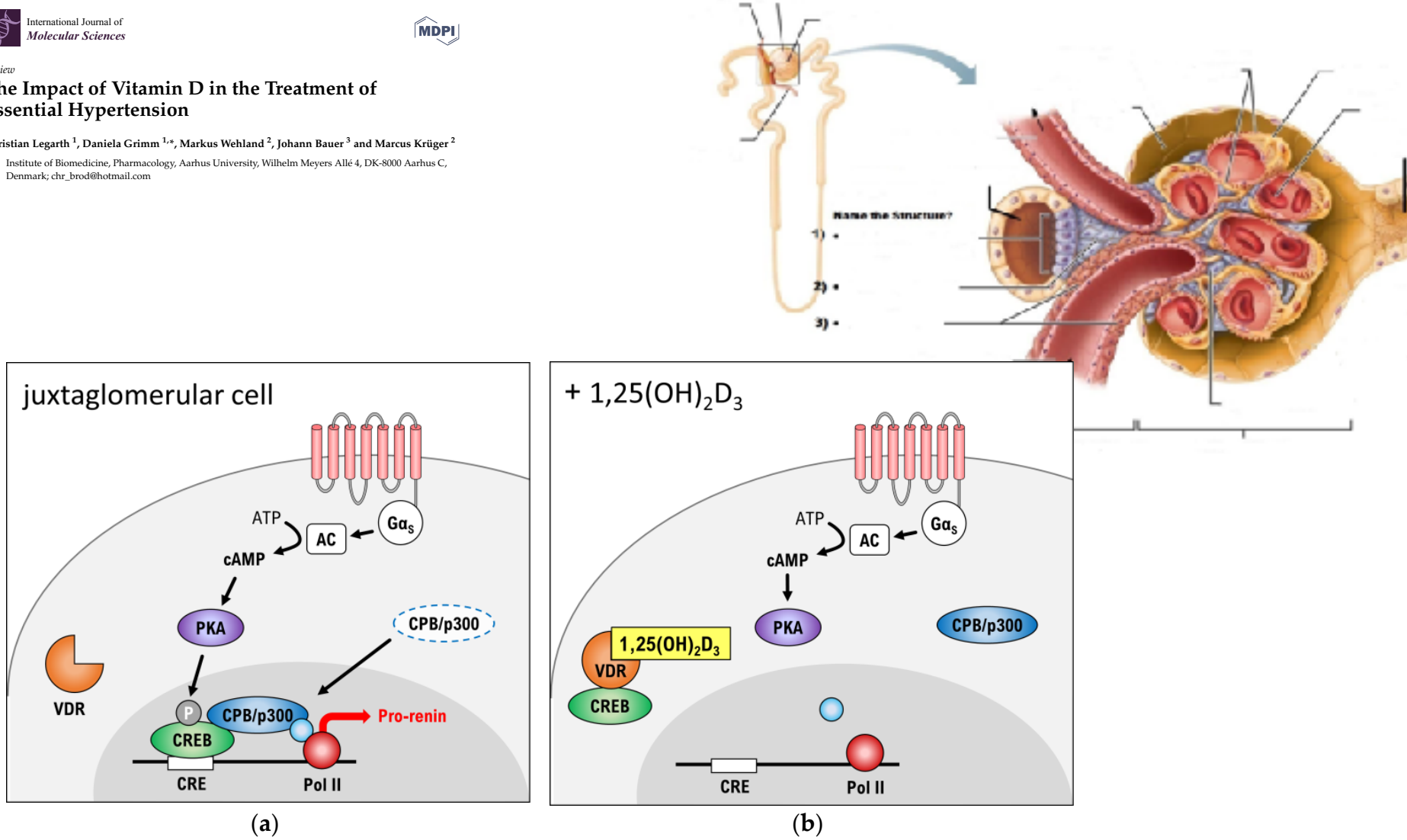


Figure 3. cAMP-PKA pathway. (**a**) Signalling in a juxtaglomerular cell in absence of $1,25(OH)_2$-vitamin D_3; (**b**) Signalling in presence of $1,25(OH)_2$-vitamin D_3. cAMP: cyclic adenosine monophosphate, CBP: CREB-binding protein, CRE: cAMP-dependent response element, CREB: cAMP response element-binding protein, $G\alpha_S$: G_S-protein alpha subunit, P: phosphate, PKA: protein kinase A, Pol II: RNA polymerase II, VDR: vitamin D receptor. The "+" stands for "JG in presence of".

AP&T Alimentary Pharmacology & Therapeutics WILEY

Letter: does vitamin D have a potential role against COVID-19?

Deepak Kumar[1]
Prakamya Gupta[2]
Dibyajyoti Banerjee[1]

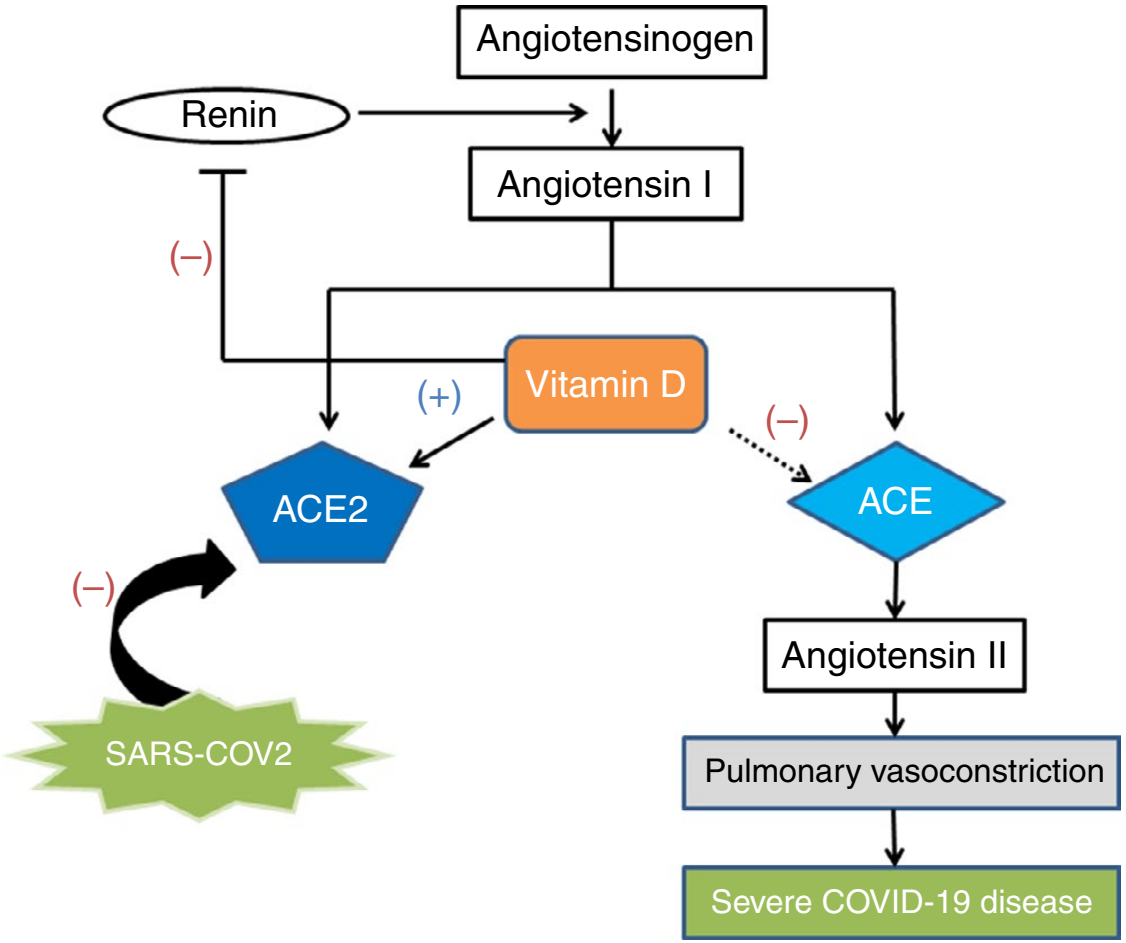
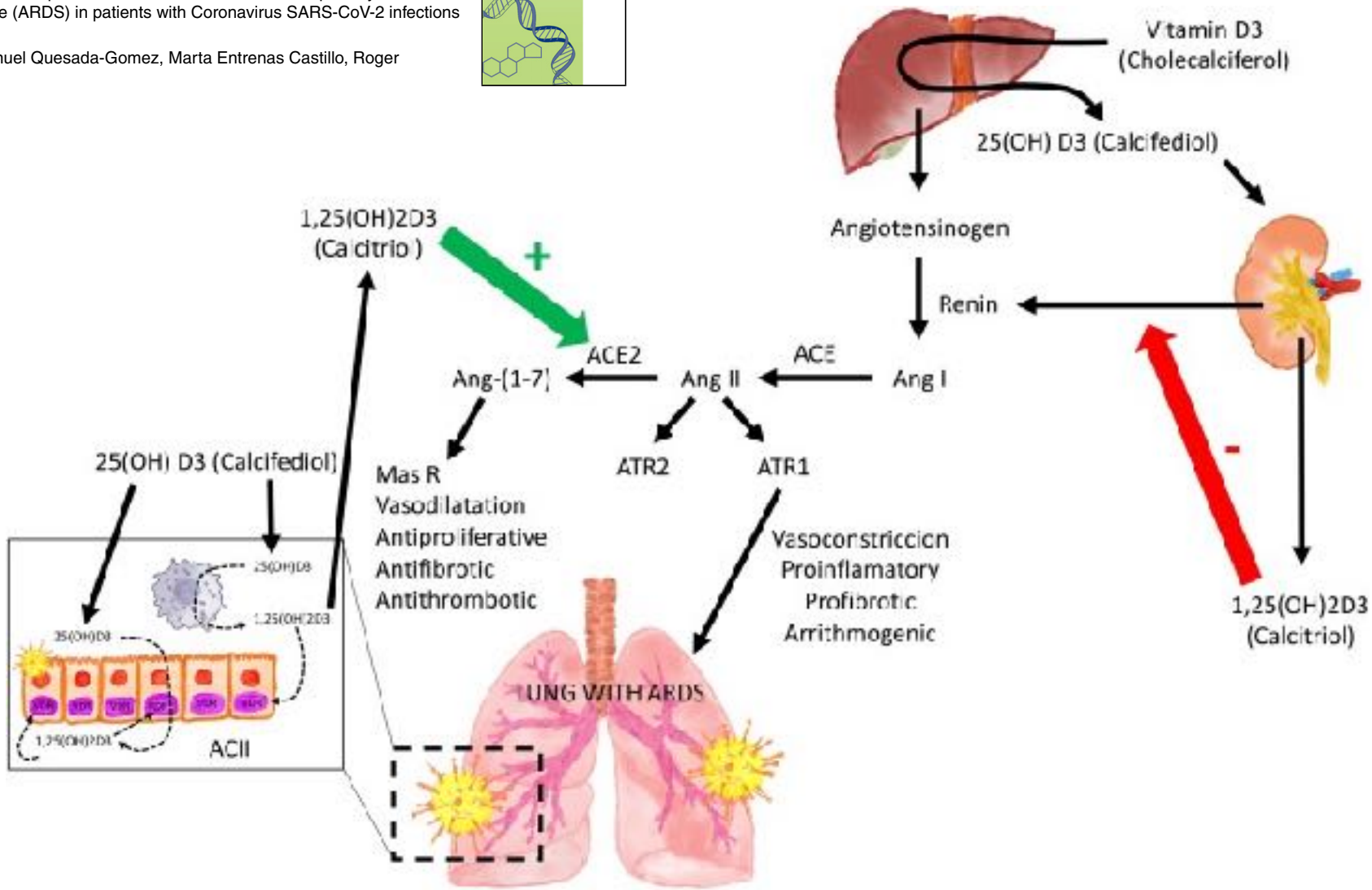


FIGURE 1 The role of vitamin D in COVID-19. SARS-CoV-2 binds to the ACE2 of alveolar cells and disturbs the ratio of ACE2/ACE activity. It increases ACE activity and, in turn, results in more angiotensin II formation causing pulmonary vasoconstriction to precipitate severe COVID19. Vitamin D induces ACE2 expression, which limits the formation of angiotensin II via ACE and reduces lung injury. Besides, vitamin D also acts on renin and inhibits its activity, which further contributes to the reduction in angiotensin II. Therefore, vitamin D supplementation may have a protective role against COVID-19. (Dotted line indicates indirect effect)

Journal Pre-proof

Vitamin D Receptor stimulation to reduce Acute Respiratory Distress Syndrome (ARDS) in patients with Coronavirus SARS-CoV-2 infections

Jose Manuel Quesada-Gomez, Marta Entrenas Castillo, Roger Bouillon



DIABETICMedicine

DOI: 10.1111/j.1464-5491.2007.02360.x

Original Article: Clinical Care

Vitamin D improves endothelial function in patients with Type 2 diabetes mellitus and low vitamin D levels

J. A. Sugden, J. I. Davies, M. D. Witham*, A. D. Morris† and A. D. Struthers

Department of Clinical Pharmacology, Division of Medicine and Therapeutics, *Section of Ageing and Health, Division of Medicine and Therapeutics and †Division of Medicine and Therapeutics, Ninewells Hospital and Medical School, Dundee, UK

Accepted 1 October 2007

Table 2 Change from baseline of parameters during treatment with vitamin D or placebo, $n = 34$

Parameter	Change with vitamin D	Change with placebo	P (between groups)
Vitamin D homeostasis			
Vitamin D (nmol/l)	22.9 ± 16.6	7.6 ± 12.5	0.02
Calcium (mmol/l)	0.01 ± 0.08	–0.04 ± 0.10	0.07
Phosphate (mmol/l)	0.05 ± 0.14	–0.04 ± 0.14	0.08
PTH (pmol/l)	–0.14 ± 0.99	–0.18 ± 0.94	0.89
Glycaemic control			
HbA_{1c} (%)	0.01 ± 0.60	–0.05 ± 0.39	0.74
HOMA (IS)	–39.7 ± 79.3	–25.6 ± 139.0	0.72
Endothelial function			
FMD response to hyperaemia (%)	2.35 ± 3.12	0.06 ± 3.39	0.048
FMD flow (%)	1.17 ± 26.68	3.55 ± 21.55	0.78
FMD response to GTN (%)	–1.33 ± 2.72	–0.98 ± 5.65	0.82
Blood pressure			
Systolic BP (mmHg)	–7.3 ± 11.8	6.6 ± 9.7	0.001
Diastolic BP (mmHg)	–2.2 ± 8.6	2.3 ± 5.7	0.08
Renin–angiotensin levels			
Renin (ng/ml)	1.85 ± 0.48	–0.79 ± 2.04	0.06
Angiotensin II (pg/ml)	–6.3 ± 19.0	6.8 ± 30.0	0.14

Mean ± SD.

BP, blood pressure; FMD, flow-mediated vasodilation; GTN, glyceryl trinitrate; HbA_{1c}, glycated haemoglobin; HOMA, homeostatic model assessment; IS, insulin sensitivity; PTH, parathyroid hormone; SD, standard deviation.

Eur J Epidemiol (2013) 28:205–221
DOI 10.1007/s10654-013-9790-2

META-ANALYSIS

Vitamin D and risk of future hypertension: meta-analys of 283,537 participants

Setor Kwadzo Kunutsor · Tanefa Antoinette Apekey · Marinka Steur

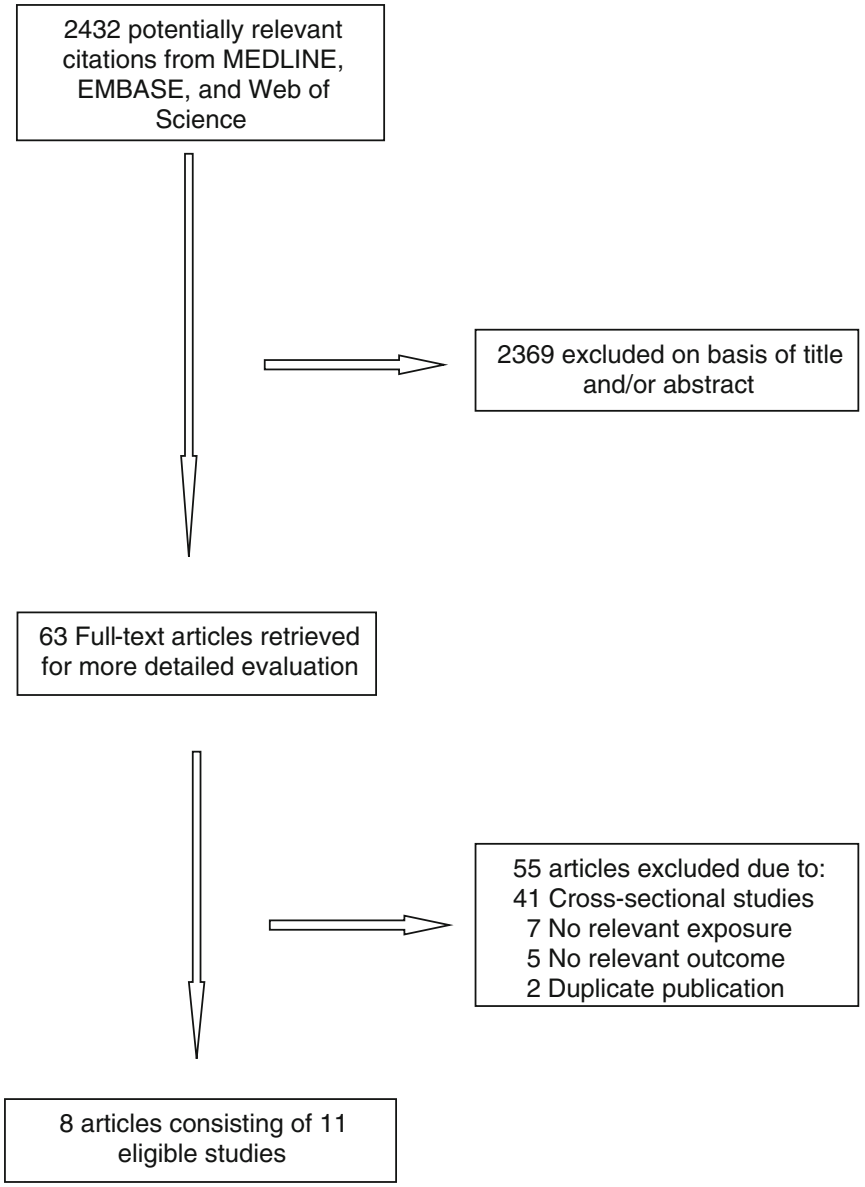


Fig. 1 Search strategy for studies included in current review, 2005–2012

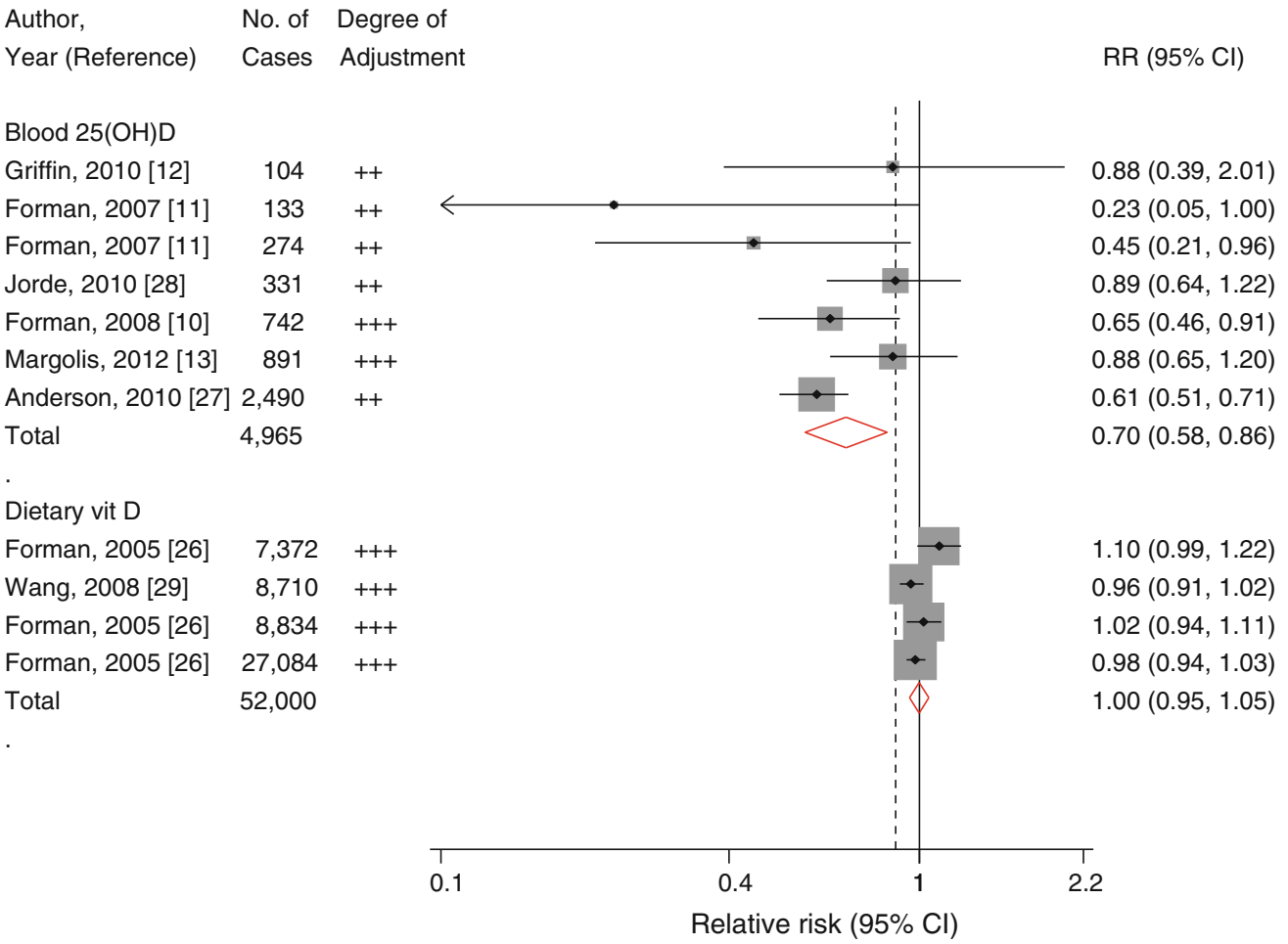


Fig. 2 Relative risk for incident hypertension in individuals in the *top* compared to the *bottom* third of *baseline* vitamin D status in Western populations, 2005–2012. The summary estimate presented was calculated using a random effects model; using a fixed effects model was 0.68 (0.60–0.77) for blood 25(OH)D and 0.99 (0.96–1.02) for dietary vitamin D. Degree of adjustment: +, adjusted for age and/or sex; ++, adjusted for + and major hypertension risk factors; +++, adjusted for ++ and parathormone/diet/Calcium/renal function/factors associated with sun exposure (e.g. season of blood draw, location, latitude); *CI* confidence interval (*bars*), *RR* relative risk

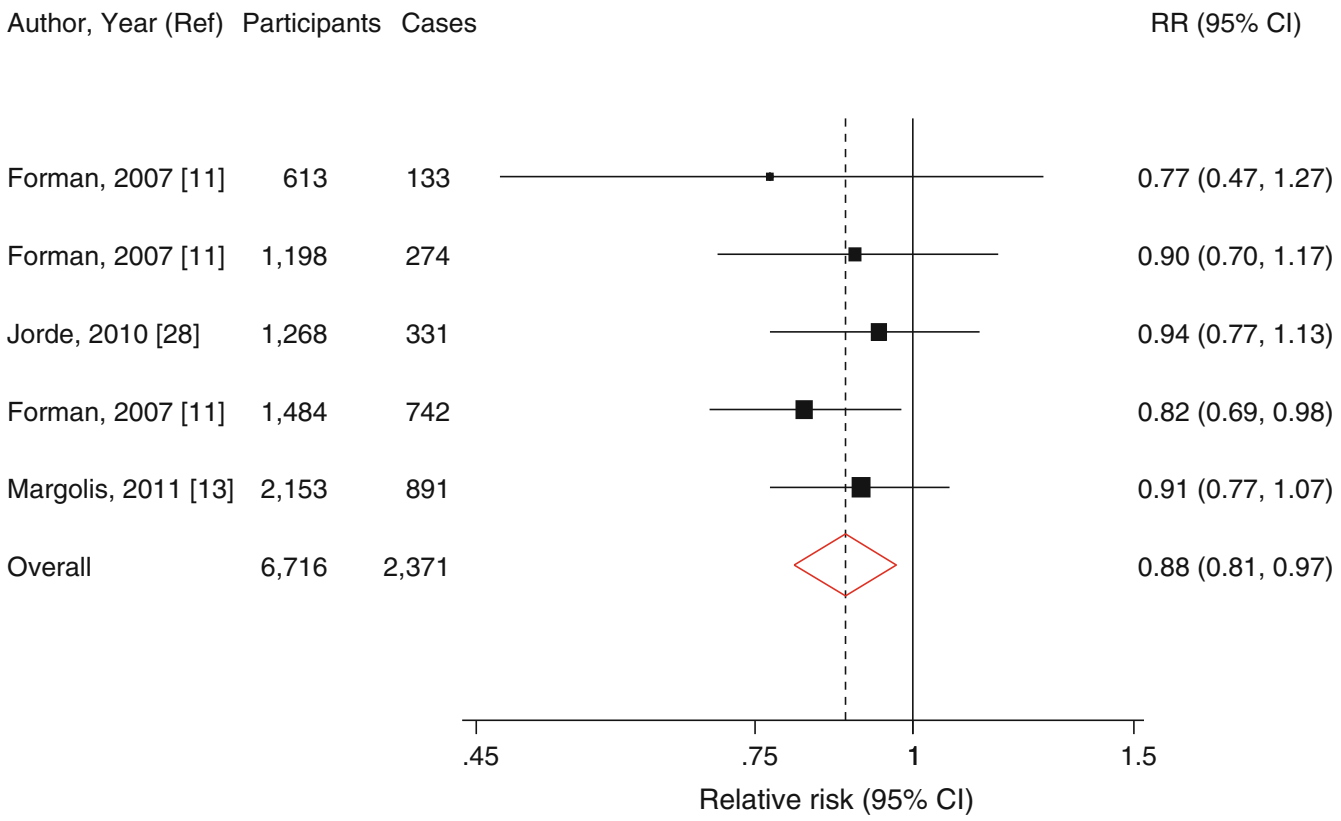


Fig. 3 Relative risk for hypertension per 10 ng/mL increment in 25(OH) D levels in studies with relevant data in Western populations, 2005–2012. *CI* confidence interval (*bars*), *RR* relative risk, *Ref* Reference

Effect of Cholecalciferol Supplementation During Winter Months in Patients With Hypertension: A Randomized, Placebo-Controlled Trial

5 mēn terapija

Thomas Larsen[1], Frank H. Mose[1], Jesper N. Bech[1], Annebirthe Bo Hansen[2] and Erling B. Pedersen[1]

METHODS

We investigated the effect of 75 µg (3,000 IU) cholecalciferol per day in a randomized, placebo-controlled, double-blind study in 130 hypertensive patients residing in Denmark (56° N). Ambulatory BP (24-h BP) and arterial stiffness were measured before and after 20 weeks of treatment, that took place between October and March.

Central SBP (mm Hg)[b]			
Placebo ($n = 55$)	132 ± 13	133 ± 15	0.007
D3 ($n = 52$)	135 ± 16	130 ± 18*	
Central DBP (mm Hg)[b]			
Placebo ($n = 55$)	82 ± 8	81 ± 8	0.15
D3 ($n = 52$)	83 ± 8	80 ± 9*	
Office SBP (mm Hg)[b]			
Placebo ($n = 55$)	142 ± 13	143 ± 15	0.02
D3 ($n = 52$)	144 ± 16	139 ± 18*	
Office DBP (mm Hg)[b]			
Placebo ($n = 55$)	81 ± 8	80 ± 7	0.18
D3 ($n = 52$)	81 ± 11	79 ± 9*	

CONCLUSIONS

Cholecalciferol supplementation, by a dose that effectively increased vitamin D levels, did not reduce 24-h BP, although central systolic BP decreased significantly. In a post-hoc subgroup analysis of 92 subjects with baseline p-25(OH)D levels <32 ng/ml, significant decreases in 24-h systolic and diastolic BP occurred during cholecalciferol supplementation.

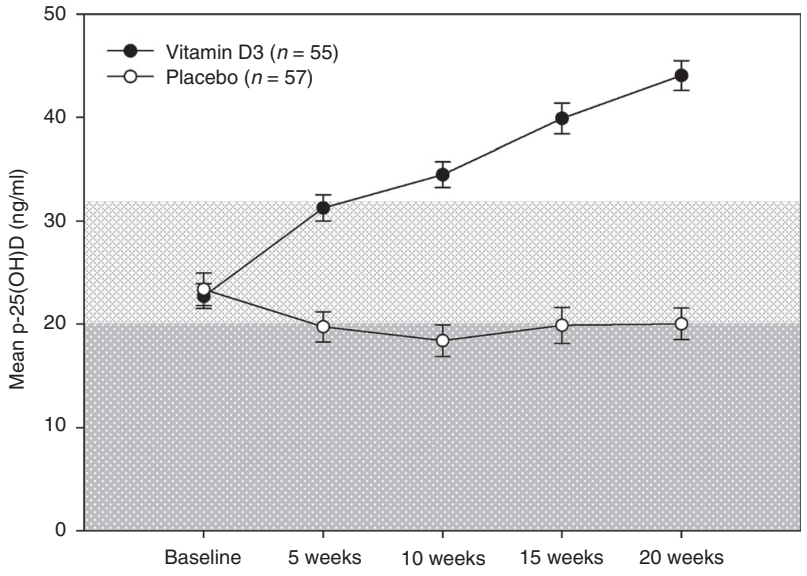


Figure 2 | Mean plasma concentrations of 25-hydroxy-vitamin D (p-25(OH)D) with s.e. From "5 weeks" onward p-25(OH)D was significantly improved ($P < 0.001$), whereas p-25(OH)D dropped significantly in the placebo group ($P < 0.001$). Light gray area represents vitamin D insufficiency (p-25(OH)D <32 ng/ml). Dark gray area represents vitamin D deficiency (p25(OH)D <20 ng/ml).

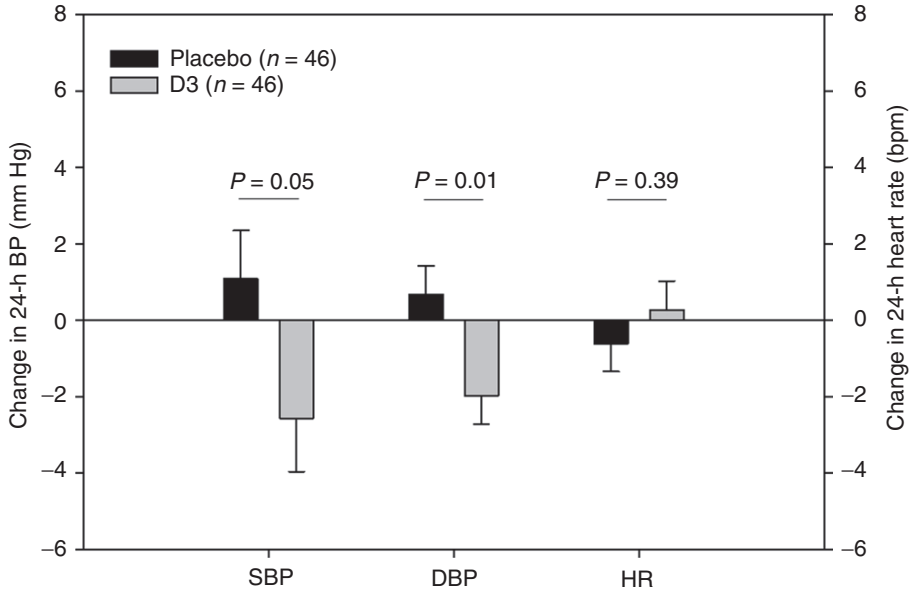
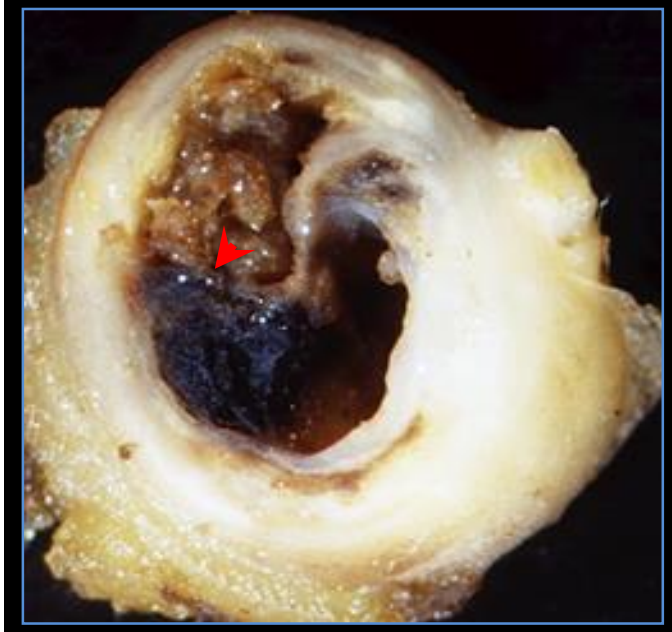
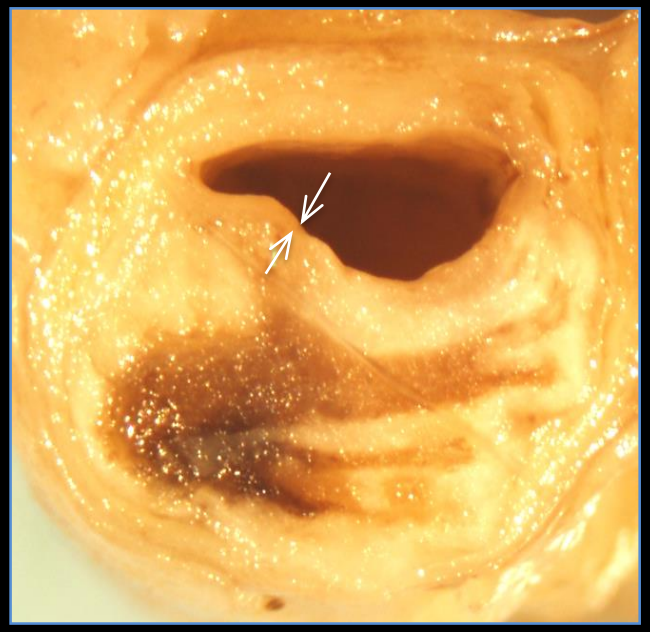
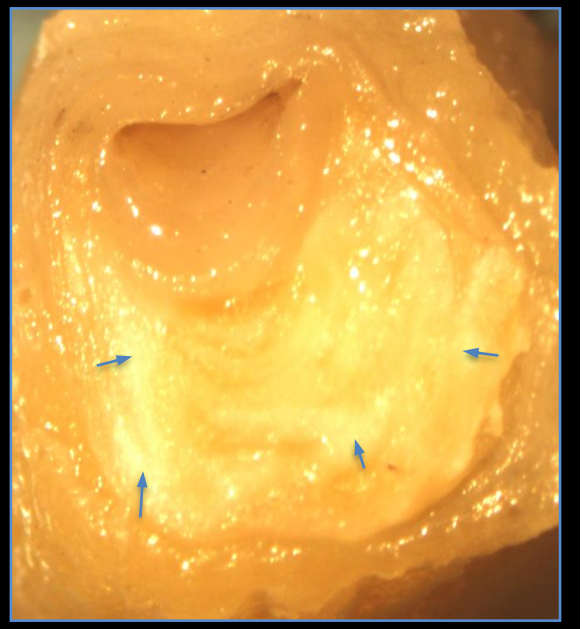


Figure 3 | Ambulatory blood pressure (BP) and heart rate in patients with plasma concentrations of 25–hydroxy–vitamin D <80 nmol/l at baseline (n = 92). Mean values and s.e.m. after treatment with cholecalciferol and placebo. bpm, beats per minute; DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure.

Augsta riska aterosklerotiskas pangas histomorfoloģija



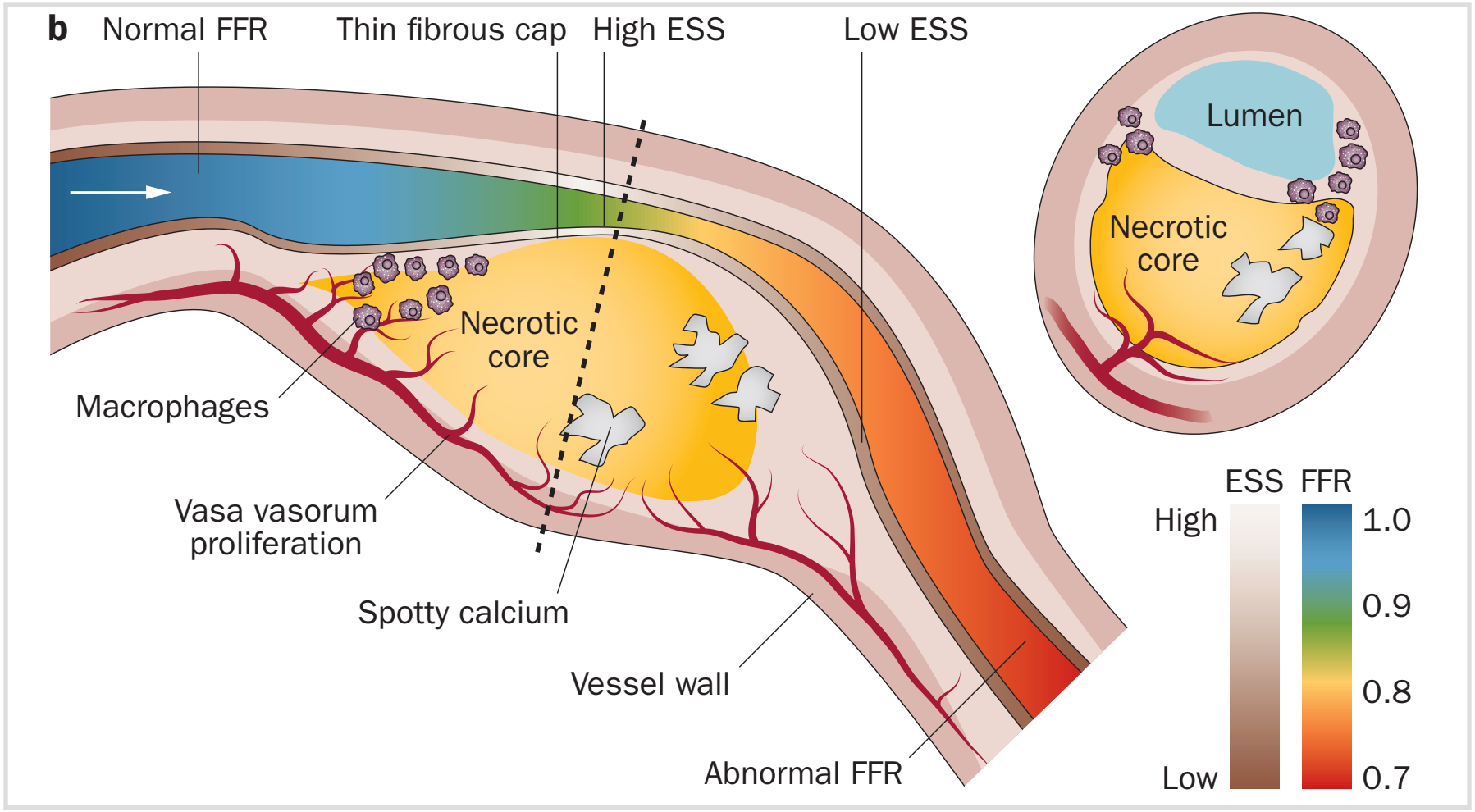




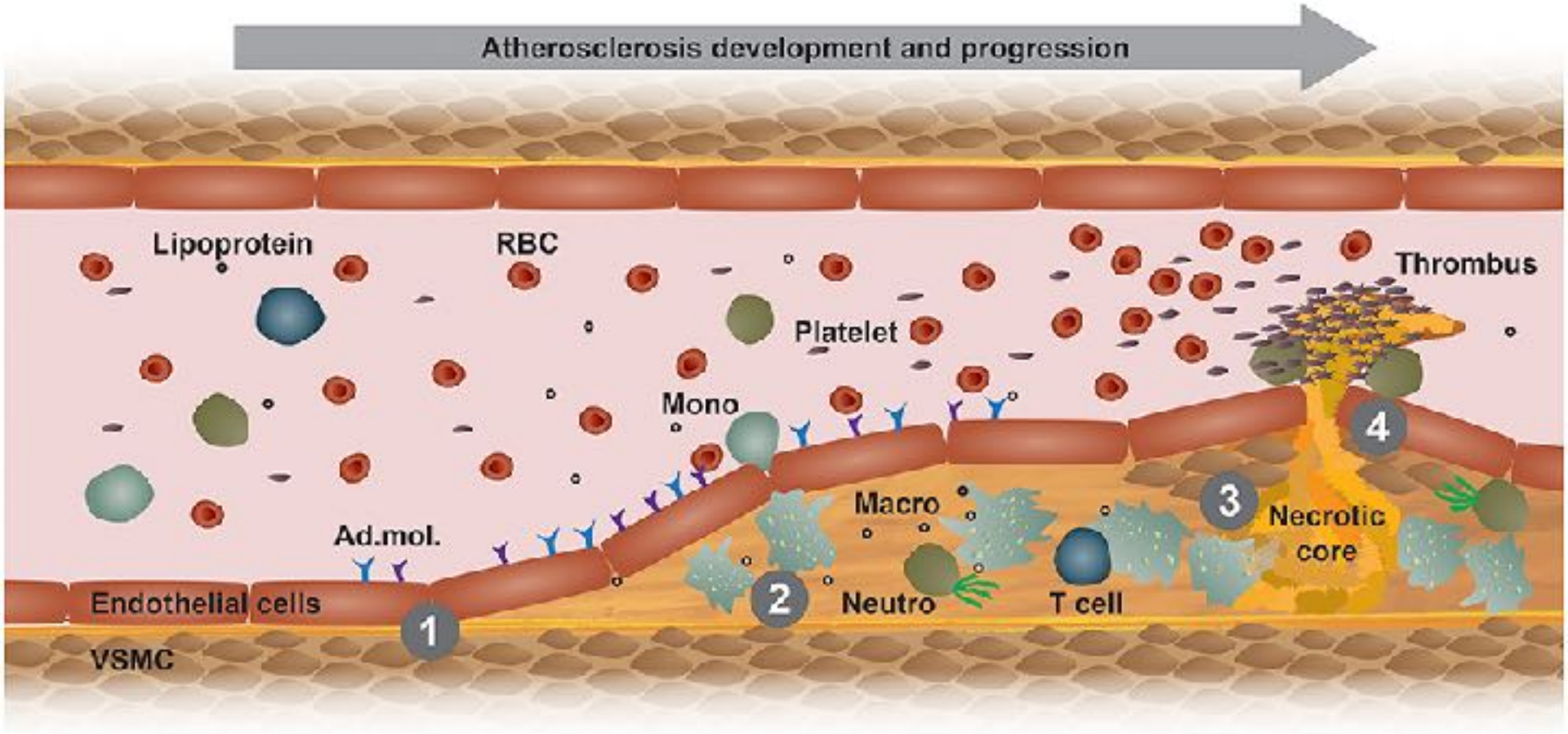
Narula et al. Braunwald's CT Atlas 2008

Comprehensive plaque assessment by coronary CT angiography

Pál Maurovich-Horvat, Maros Ferencik, Szilard Voros, Béla Merkely and Udo Hoffmann



Maurovich-Horvat, P. *et al. Nat. Rev. Cardiol.* advance online publication 22 April 2014;

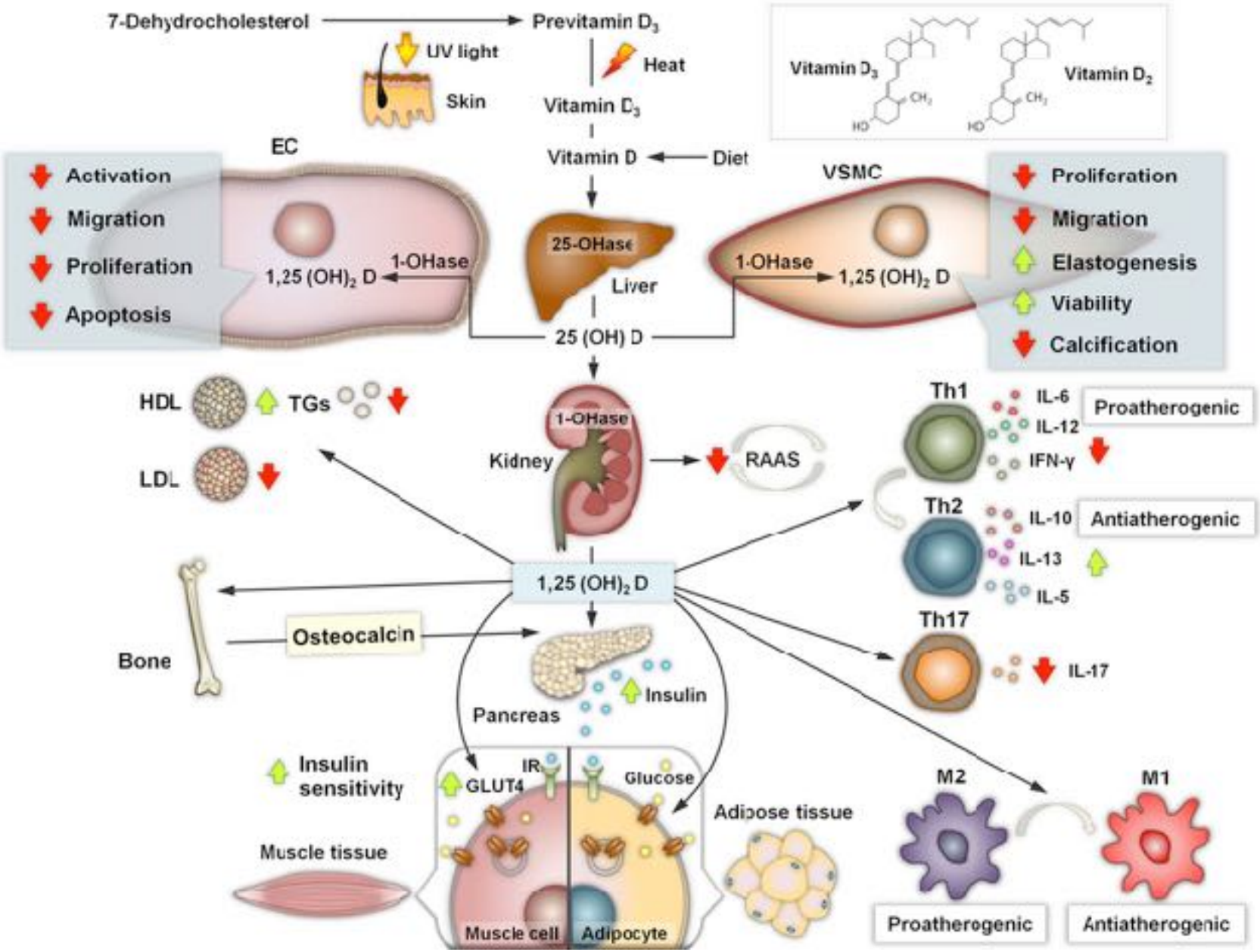


Oppi S, Lüscher TF and Stein S(2018) Mouse models for atherosclerosis research – which is my line?. *Front. Cardiovasc. Med.* 6:46. doi:10.3389/fcvm.2019.00046

Circulation December 3/10, 2013

Role of Vitamin D in Atherosclerosis

Eva Kassi, MD, PhD; Christos Adamopoulos, BSc, MSc; Efthimia K. Basdra, DDS, PhD; Athanasios G. Papavassiliou, MD, PhD



Contemporary Reviews in Cardiovascular Medicine

Role of Vitamin D in Atherosclerosis

Eva Kassi, MD, PhD; Christos Adamopoulos, BSc, MSc; Efthimia K. Basdra, DDS, PhD; Athanasios G. Papavassiliou, MD, PhD

Vitamin D deficiency affects almost 50% of the population worldwide. It has been suggested that this pandemic might contribute to the worldwide increased prevalence of CVD.[9–11]

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Vitamin D Supplements and Prevention of Cancer and Cardiovascular Disease

JoAnn E. Manson, M.D., Dr.P.H., Nancy R. Cook, Sc.D., I-Min Lee, M.B., B.S., Sc.D., William Christen, Sc.D., Shari S. Bassuk, Sc.D., Samia Mora, M.D., M.H.S., Heike Gibson, Ph.D., David Gordon, M.A.T., Trisha Copeland, M.S., R.D., Denise D'Agostino, B.S., Georgina Friedenberg, M.P.H., Claire Ridge, M.P.H., Vadim Bubes, Ph.D., Edward L. Giovannucci, M.D., Sc.D., Walter C. Willett, M.D., Dr.P.H., and Julie E. Buring, Sc.D., for the VITAL Research Group*

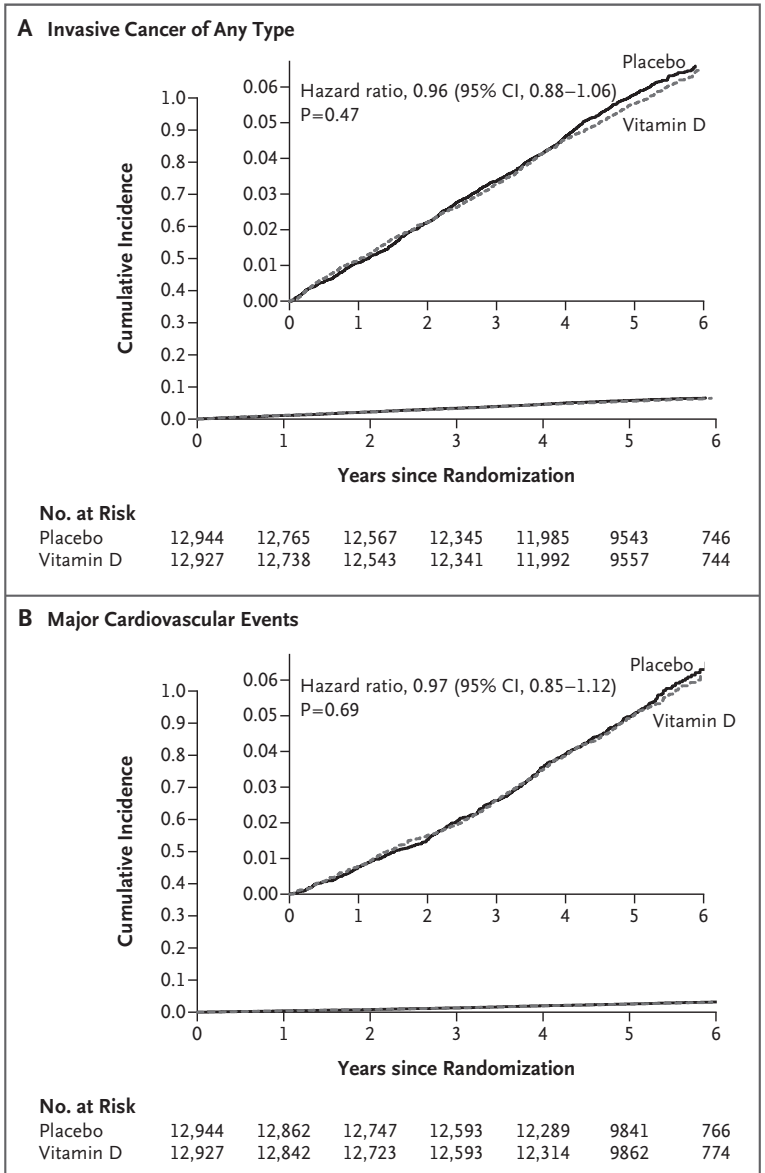


Figure 2. Cumulative Incidence Rates of Invasive Cancer of Any Type and Major Cardiovascular Events, According to Year of Follow-up, in the Vitamin D Group and Placebo Group.

Analyses were from Cox regression models that were controlled for age, sex, and randomization group in the n–3 fatty acid portion of the trial (intention-to-treat analyses). The insets show the same data on an enlarged y axis.

ORIGINAL ARTICLE

Vitamin D Supplements and Prevention of Cancer and Cardiovascular Disease

JoAnn E. Manson, M.D., Dr.P.H., Nancy R. Cook, Sc.D., I-Min Lee, M.B., B.S., Sc.D., William Christen, Sc.D., Shari S. Bassuk, Sc.D., Samia Mora, M.D., M.H.S., Heike Gibson, Ph.D., David Gordon, M.A.T., Trisha Copeland, M.S., R.D., Denise D'Agostino, B.S., Georgina Friedenberg, M.P.H., Claire Ridge, M.P.H., Vadim Bubes, Ph.D., Edward L. Giovannucci, M.D., Sc.D., Walter C. Willett, M.D., Dr.P.H., and Julie E. Buring, Sc.D., for the VITAL Research Group*

Analyses excluding the first 2 yr of follow-up			
Invasive cancer of any type	490	522	0.94 (0.83–1.06)
Death from cancer	112	149	0.75 (0.59–0.96)
Major cardiovascular event	274	296	0.93 (0.79–1.09)
Death from any cause	368	384	0.96 (0.84–1.11)

TRIAL DESIGN AND OVERSIGHT

We conducted this randomized, double-blind, placebo-controlled trial, with a two-by-two factorial design, to examine the benefits and risks of vitamin D_3 (cholecalciferol) at a dose of 2000 IU per day and marine n–3 fatty acids at a dose of 1 g per day in the primary prevention of cancer and cardiovascular disease among 25,871 men who were 50 years of age or older and women who were 55 years of age or older. The trial protocol has been described elsewhere[4,12] and is available with the full text of this article at NEJM.org.

Eur J Clin Nutr. 2015 February ; 69(2): 193–197. doi:10.1038/ejcn.2014.209.

The effect of a single, large bolus of vitamin D in healthy adults over the winter and following year: a randomized, double-blind, placebo-controlled trial

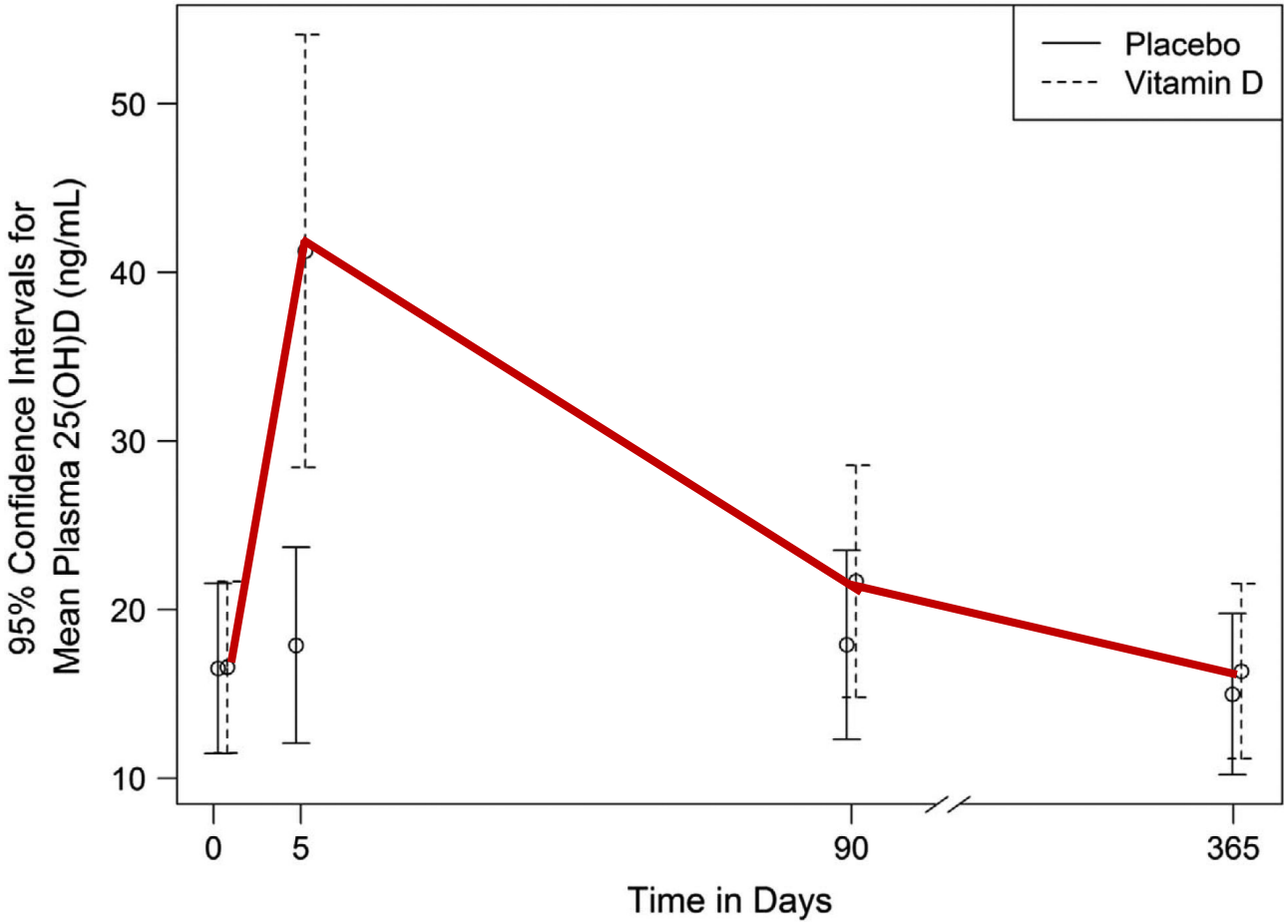
MD Kearns[1], **JNG Binongo**[2], **D Watson**[2], **JA Alvarez**[1], **D Lodin**[1], **TR Ziegler**[1], and **V Tangpricha**[1,3]

[1]Division of Endocrinology, Metabolism and Lipids, Emory University School of Medicine, Atlanta, GA, USA

[2]Department of Biostatistics and Bioinformatics, Rollins School of Public Health, Atlanta, GA, USA

[3]Atlanta Veterans Affairs Medical Center, Section of Endocrinology, Atlanta, GA, USA.

250 000 DV holekalciferols



Participant demographics

100% līdzestība:)

Baseline characteristics	Study arm	
	Vitamin D (n = 14)	Placebo (n = 14)
Age, year, mean (s.d.)	28.2 (6.7)	26.5 (5.2)
Female, *n* (%)	12 (86)	10 (71)
White, *n* (%)	9 (64)	9 (64)
Weight, kg, mean (s.d.)	66.6 (9.3)	65.5 (10.2)
Height, m, mean (s.d.)	1.7 (0.1)	1.7 (0.1)
BMI, mean (s.d.)	23.7 (2.9)	22.3 (2.2)
Fitzpatrick scale, n		
Type 2	5	6
Type 3	4	4
Type 4	2	2
Serum calcium level, mg/dl, mean (s.d.)	9.3 (0.3)	9.2 (0.3)
Hours outdoors, week, mean (s.d.)	9.0 (5.2)	7.0 (5.4)
Current vitamin D supplementation, *n*	4	1

Osteoporos Int (2015) 26:2863–2868
DOI 10.1007/s00198-015-3205-z

ORIGINAL ARTICLE

A phase IV, two-armed, randomized, cross-over study comparing compliance with once-a-month administration of vitamin D3 to compliance with daily administration of a fixed-dose combination of vitamin D3 and calcium during two 6-month periods

O. Bruyère[1] · R. Deroisy[2] · N. Dardenne[1] · E. Cavalier[3] · M. Coffiner[4] · S. Da Silva[4] · S. De Niet[4] · J.-Y. Reginster[1]

Iedzert ampulu vai sakožļāt tableti?

100 pacienti 6 mēn 25 000 DV 1xmēn vs 800 DV tabletes katru dienu

Table 2 Association between chosen reasons of preference and chosen treatment

		VD group *N* (%)	VDCa group *N* (%)	Fisher exact test *p*
Reason	Taste	2 (3.0)	3 (18.8)	0.030
	Ease of use	17 (34.0)	10 (62.4)	
	Frequency of use	21 (42.0)	3 (18.8)	
	No adverse events	7 (14.0)	0 (0.0)	
	Treatment reputation	0 (0.0)	0 (0.0)	
	Other	3 (6.0)	0 (0.)	
	Total	50 (76.5)	16 (23.5)	

25-Hydroxyvitamin D and Risk of Myocardial Infarction in Men

A Prospective Study

Edward Giovannucci, MD, ScD; Yan Liu, MS; Bruce W. Hollis, MD, PhD; Eric B. Rimm, ScD

Table 3. Estimated RRs of MI by Level of 25(OH)D at Baseline During 10 Years of Follow-up

Variable	Plasma 25(OH)D, ng/mL				*P* Value (Trend)
	≤15.0	15.1-22.5	22.6-29.9	≥30.0	
Cases/controls, No.	63/87	156/307	165/299	70/207	NA
RR (95% CI)					
Matching variables	2.42 (1.53-3.84)	1.65 (1.15-2.37)	1.72 (1.22-2.42)	1 [Reference]	<.001
MV1[a]	2.01 (1.22-3.30)	1.45 (0.99-2.12)	1.56 (1.09-2.22)	1 [Reference]	.02
MV2[b]	2.09 (1.24-3.54)	1.43 (0.96-2.13)	1.60 (1.10-2.32)	1 [Reference]	.02

Abbreviations: CI, confidence interval; MI, myocardial infarction; MV, multivariate; NA, not applicable; 25(OH)D, 25-hydroxyvitamin D; RR, relative risk.

SI conversion factor: To convert 25(OH)D to nanomoles per liter, multiply by 2.496.

[a] MV1: matching variables (age, month and year of blood collection, and smoking status) and family history of MI before the age of 60 years, history of diabetes mellitus, history of hypertension, alcohol intake, body mass index, physical activity, region, race, multivitamin use, marine ω-3 intake, and fasting status.

[b] MV2: all the variables in MV1 and high- and low-density lipoprotein cholesterol and triglyceride levels.

Cardiovascular Therapeutics

STATIN-D Study: Comparison of the Influences of Rosuvastatin and Fluvastatin Treatment on the Levels of 25 Hydroxyvitamin D

Derun Taner Ertugrul,[1] Bunyamin Yavuz,[2] Hicran Cil,[1] Naim Ata,[1] Kadir Okhan Akin,[3] Metin Kucukazman,[1] Ahmet Arif Yalcin,[2] Kursad Dal,[1] Burcu Balam Yavuz[4] & Emre Tutal[1]

1 Department of Internal Medicine, Kecioren Teaching and Research Hospital, Ankara, Turkey
2 Department of Cardiology, Kecioren Teaching and Research Hospital, Ankara, Turkey
3 Department of Biochemistry, Kecioren Teaching and Research Hospital, Ankara, Turkey
4 Department of Internal Medicine, Hacettepe University Faculty of Medicine, Ankara, Turkey

Table 3 Bone parameters before and after rosuvastatin and fluvastatin treatment

8 nedēļas

	Rosuvastatin 10 mg (n = 69)			Fluvastatin 80 mg XL (n = 65)			*P
	Baseline	After treatment	**P	Baseline	After treatment	**P	
25-OHvitD (ng/mL)	11.8 (3.7–30.0)	35.2 (4.0–101.0)	**<0.001**	9.6 (4.0–67.0)	10.2 (3.9–83.0)	0.557	**<0.001**
1,25 OHvitD (pg/mL)	18.3 (5.6–145.0)	24.0(10.5–51.0)	**0.008**	19.4 (2.8–43.0)	20.7 (6.4–56.4)	0.241	0.144
BALP (U/L)	18.4 (2.6–214.0)	9.6 (0.9–21.6)	**<0.001**	17.0 (2.99–258.0)	12.8 (0.7–167.0)	**0.004**	0.368
OCL (ng/mL)	4.3 (1.0–35.0)	4.5 (1.0–24.7)	0.927	4.8 (1.0–32.0)	4.0 (1.2–35)	0.178	0.123
Ca (mg/dL)	9.4 ± 0.6	9.4 ± 0.5	0.774	9.6 ± 0.5	9.4 ± 1.0	**0.041**	0.056
P (mg/dL)	3.1 ± 0.7	3.1 ± 0.5	0.768	3.1 ± 0.7	3.2 ± 0.6	0.181	0.222

25-OHvitD: 25 Hydroxyvitamin D; 1,25 OHvitD: 1,25-hydroxyvitamin D.
BALP, bone alkaline phosphatase; OCL, osteocalcin; P, phosphorus; Ca, calcium.
**P*: *P* value between rosuvastatin and fluvastatin.
***P*: *P* value between baseline and after treatment.
Continuous variables with normal distribution were expressed as mean ± SD. Variables with skew distribution are expressed as median (minimum–maximum), and categorical variables are expressed as percentage.

 © 2010 Blackwell Publishing Ltd

Effects of *Atorvastatin* on Vitamin D Levels in Patients With Acute Ischemic Heart Disease

José L. Pérez-Castrillón, MD[a,*], Gemma Vega, MD[a], Laura Abad, MD[a], Alberto Sanz, MD[b], José Chaves, MD[c], Gonzalo Hernandez, MD[c], and Antonio Dueñas, MD[a]

Table 1
Effect of atorvastatin on vitamin D and other laboratory data

Variable	Baseline	12 mos	p Value
Total cholesterol (mg/dl)	182 ± 48	161 ± 33	0.0001
HDL cholesterol (mg/dl)	39 ± 12	49 ± 11	0.0001
LDL cholesterol (mg/dl)	114 ± 41	90 ± 31	0.0001
Triglycerides (mg/dl)	152 ± 91	117 ± 66	0.003
Calcium (mg/dl)	9.5 ± 0.6	9.6 ± 0.5	NS
Phosphorus (mg/dl)	3.6 ± 0.6	3.4 ± 0.6	NS
25-Hydroxycholecalciferol (nmol/L)	41 ± 19	47 ± 19	0.003

LDL = low-density lipoprotein.

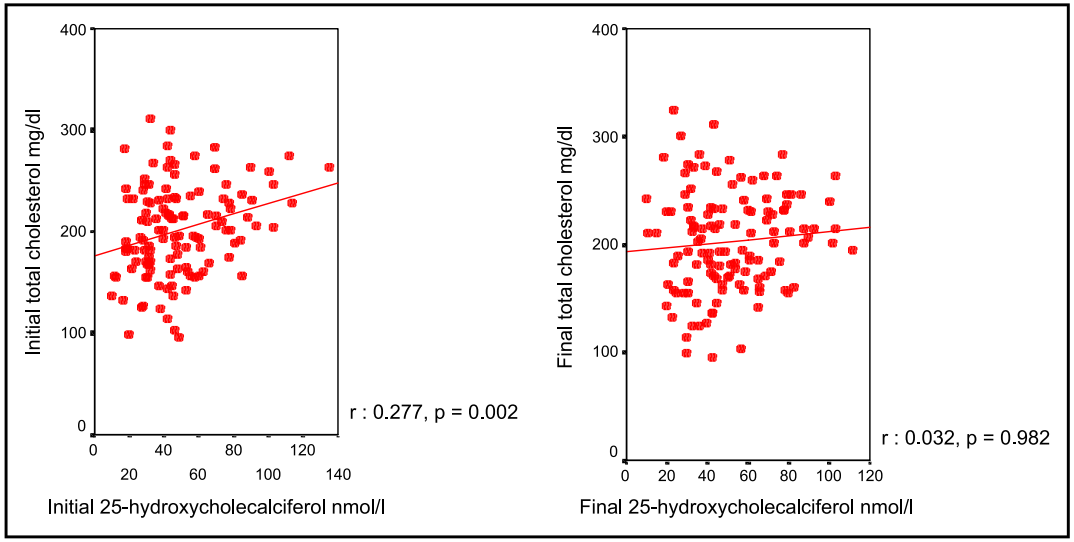
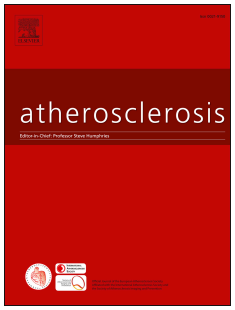


Figure 1. Relation between baseline and final levels of total cholesterol and 25-hydroxycholescalciferol.

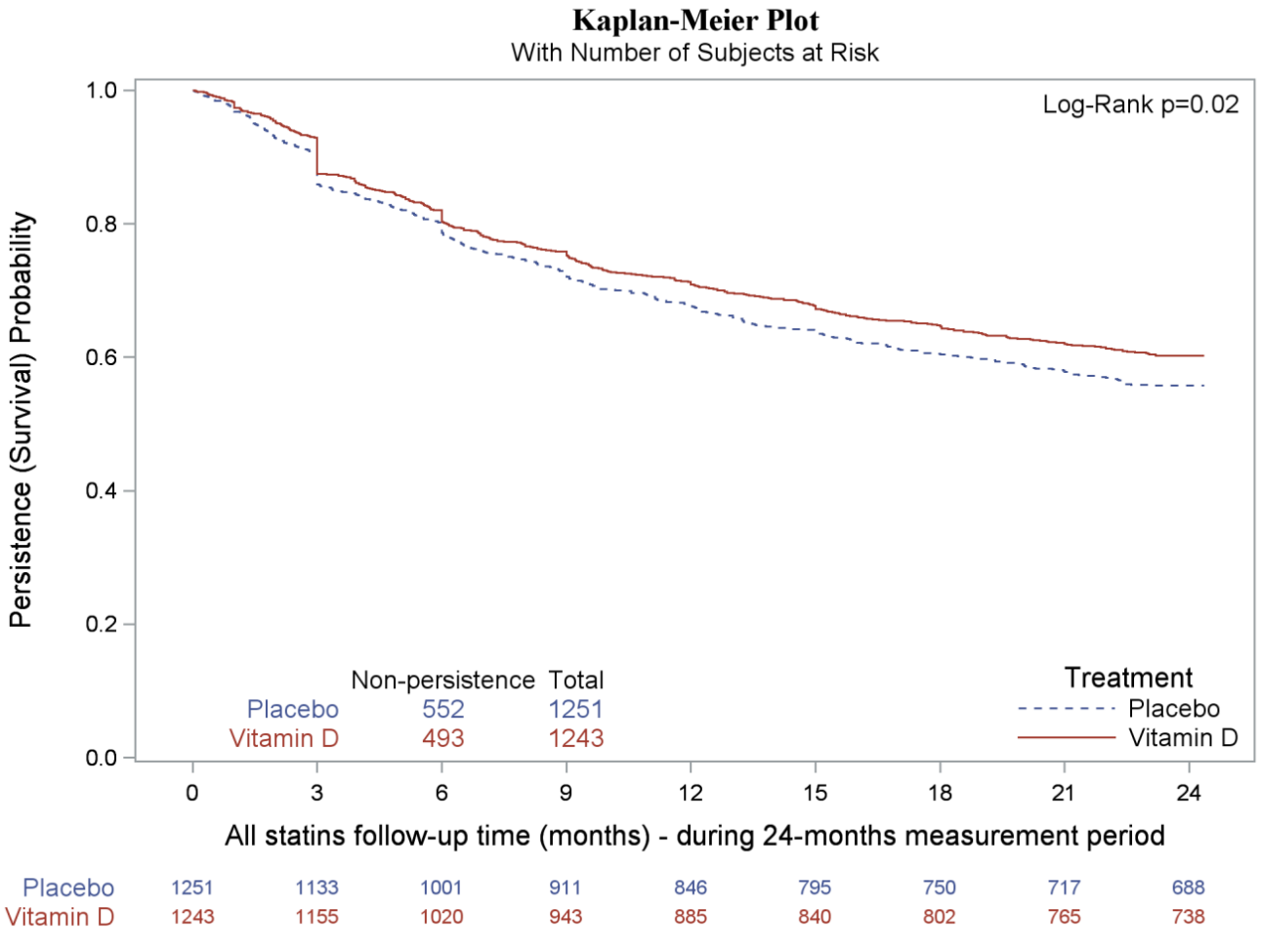
Accepted Manuscript



Effects of vitamin D supplementation on adherence and persistence with long-term statin therapy: Secondary analysis from the randomized, double-blind, placebo-controlled ViDA study

Zhenqiang Wu, Carlos A. Camargo, Jr., Kay-Tee Khaw, Debbie Waayer, Carlene M.M. Lawes, Les Toop, Robert Scragg

2018



Journal of Clinical & Translational Endocrinology 6 (2016) 56–59

Contents lists available at ScienceDirect

Journal of Clinical & Translational Endocrinology

journal homepage: www.elsevier.com/locate/jcte

Original research

Impact of vitamin D status on statin-induced myopathy

Krista D. Riche Pharm.D. [a,b], Justin Arnall Pharm.D. [c], Kristin Rieser Pharm.D. [a], Honey E. East M.D. [d], Daniel M. Riche Pharm.D., FCCP, BCPS, CDE, ASH-CHC, CLS [a,e,*]

[a] The University of Mississippi, School of Pharmacy, Jackson, MS, United States
[b] St. Dominic Hospital, Jackson, MS, United States
[c] Wake Forest University, Winston-Salem, NC, United States
[d] Premier Medical Group, Jackson, MS, United States
[e] The University of Mississippi School of Medicine, Jackson, MS, United States

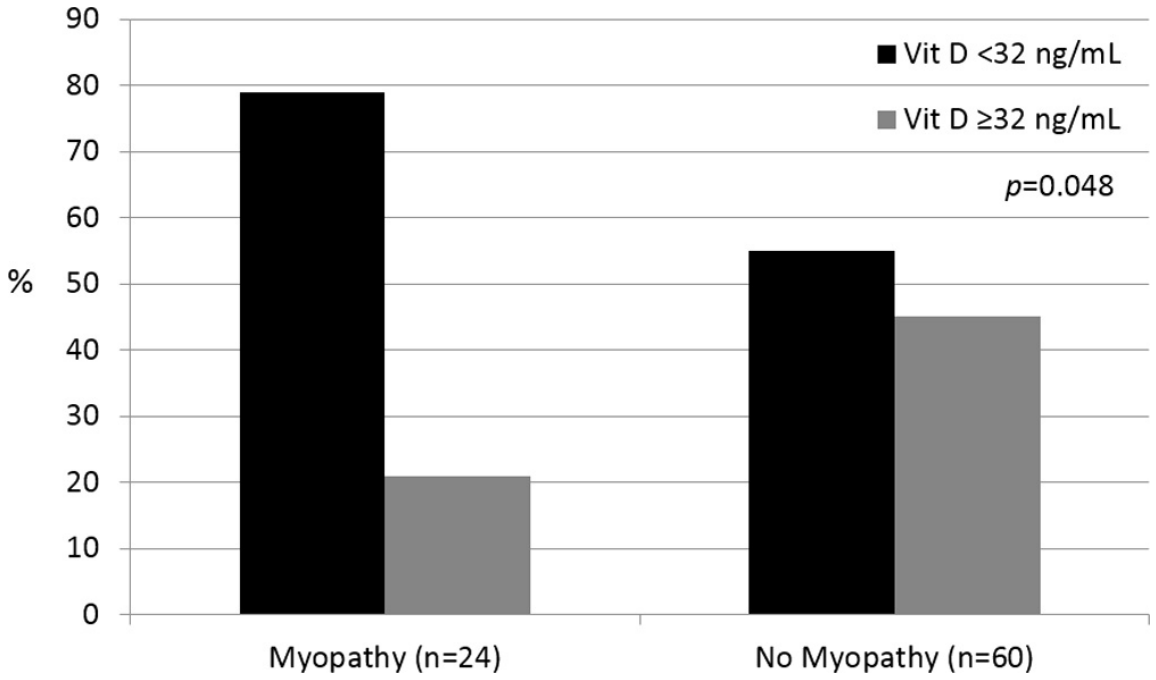
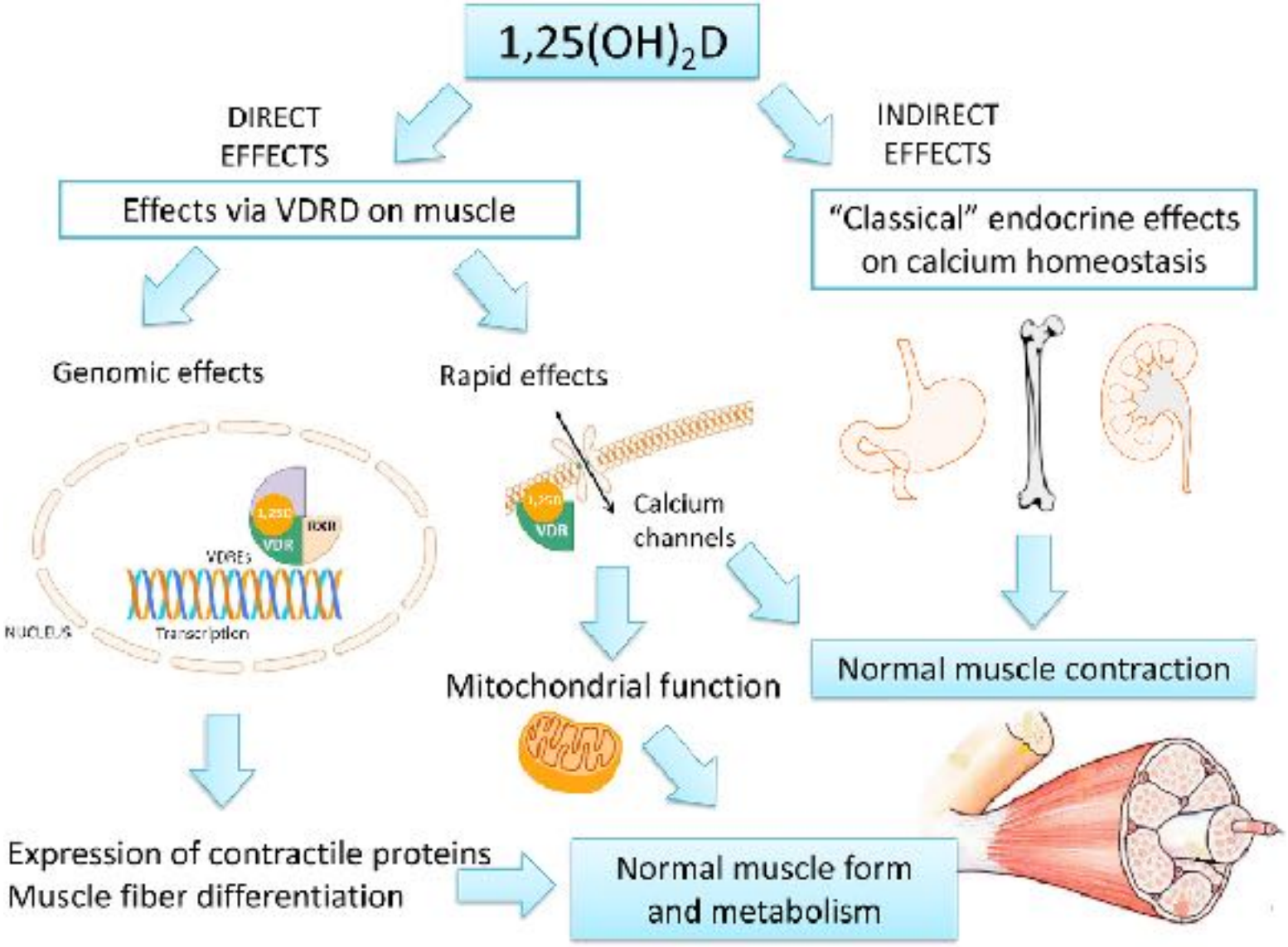


Fig. 1. Rate of statin-induced myopathy by vitamin D Status before vitamin D supplementation


1,25(OH)2D
DIRECT EFFECTS
INDIRECT EFFECTS
Effects via VDRD on muscle
"Classical" endocrine effects on calcium homeostasis
Genomic effects
Rapid effects
1,25D
VDR
RXR
VDRE
Transcription
NUCLEUS
Calcium channels
Mitochondrial function
Normal muscle contraction
Expression of contractile proteins
Muscle fiber differentiation
Normal muscle form and metabolism

ESC European Society of Cardiology
European Heart Journal (2019) **00**, 1–78
doi:10.1093/eurheartj/ehz455

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

Recommendations for treatment goals for low-density lipoprotein cholesterol

Recommendations	Class[a]	Level[b]
In secondary prevention for patients at very-high risk,[c] an LDL-C reduction of ≥50% from baseline[d] and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended.[33–35,119,120]	I	A

Ļoti augsta riska pacientiem ZBLH jāsamazina par >50% un mērķis ir <1.4 mmol/l

For patients with ASCVD who experience a second vascular event within 2 years (not necessarily of the same type as the first event) while taking maximally tolerated statin-based therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered.[119,120]	IIb	B
In patients at high risk,[c] an LDL-C reduction of ≥50% from baseline[d] and an LDL-C goal of <1.8 mmol/L (<70 mg/dL) are recommended.[34,35]	I	A

Ja atkārtojas notikums, apsvērt ZBLH mērķi <1.0 mmol/l

ESC European Society of Cardiology
European Heart Journal (2019) 00, 1–78
doi:10.1093/eurheartj/ehz455
ESC/EAS GUIDELINES

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

Recommendations for pharmacological low-density lipoprotein cholesterol lowering

Recommendations	Class[a]	Level[b]
It is recommended that a high-intensity statin is prescribed up to the highest tolerated dose to reach the goals set for the specific level of risk.[32,34,38]	I	A
If the goals[c] are not achieved with the maximum tolerated dose of a statin, combination with ezetimibe is recommended.[33]	I	B
For primary prevention patients at very-high risk, but without FH, if the LDL-C goal is not achieved on a maximum tolerated dose of a statin and ezetimibe, a combination with a PCSK9 inhibitor may be considered.	IIb	C
For secondary prevention, patients at very-high risk not achieving their goal[c] on a maximum tolerated dose of a statin and ezetimibe, a combination with a PCSK9 inhibitor is recommended.[119,120]	I	A

Statīns augstākajā rekomendētajā/tolerētajā devā

↓

Ezetimībs

↓

PCSK9i

Effect of the inhibition of a cholesterol membrane transporter on vitamin D absorption:

a double-blind randomized placebo-controlled study

Mariana Costa Silva, Gustavo Adolpho Moreira Faulhauber, Érica Neves Leite, Kamila Ramborger Goulart, Jorge Mario Ahumada Ramirez, Fernanda Mariani Cocolichio, Tania Weber Furlanetto

Table 2. Biochemical responses 14 days after 50,000 IU oral vitamin D3

Measure	Ezetimibe	Placebo	p
25OHD, ng/mL	24.67 ± 5.24	24.49 ± 6.16	0.391
Δ25OHD, ng/mL	8.75 ± 3.74	10.02 ± 3.84	0.26
Calcium, mg/dL	9.32 ± 0.45	9.41 ± 0.39	0.475
Albumin, g/dL	4.51 ± 0.27	4.61 ± 0.33	0.356
PTH, pg/mL	33.51 ± 14.44	34.90 ± 12.85	0.729

Endocrine Abstracts (2015)

17th European Congress of Endocrinology