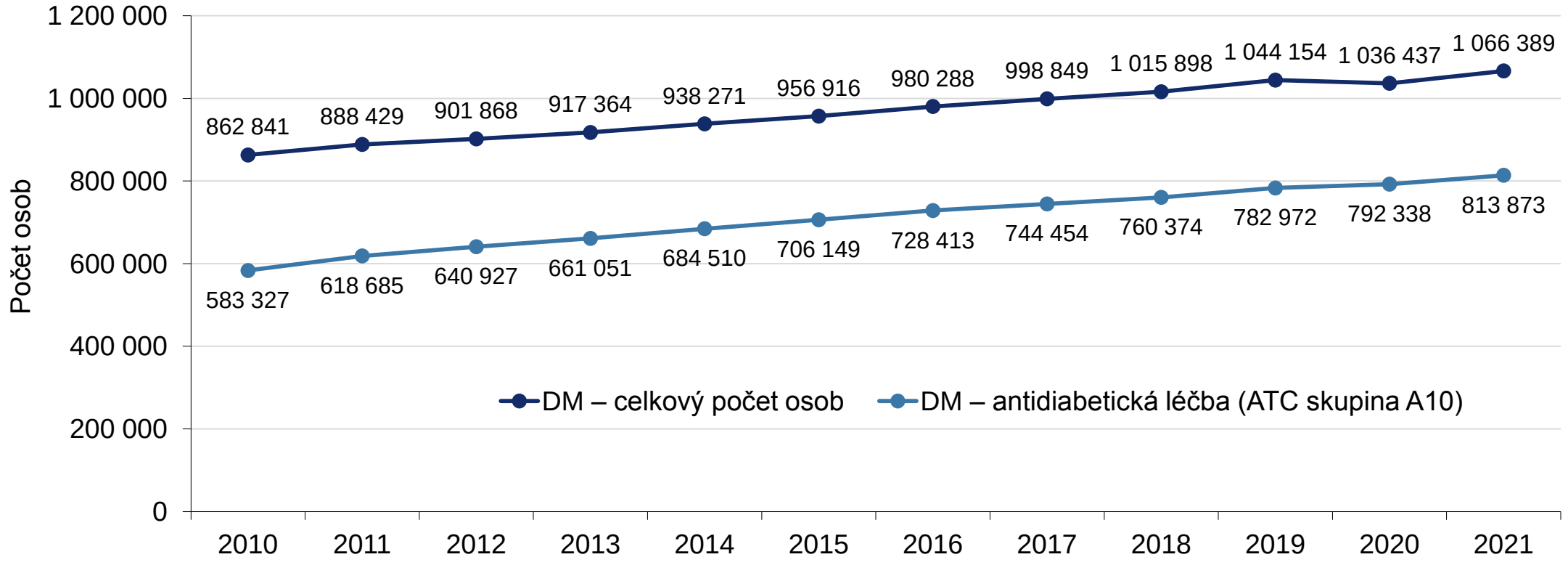
Metabolické aspekty léčby arteriální hypertenze

Martin Prázný

Prevalence diabetes mellitus v české populaci

Zdroj: NRHZS 2010–2021



	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
Podíl léčených	67,6 %	69,6 %	71,1 %	72,1 %	73,0 %	73,8 %	74,3 %	74,5 %	74,8 %	75,0 %	76,4 %	76,3 %

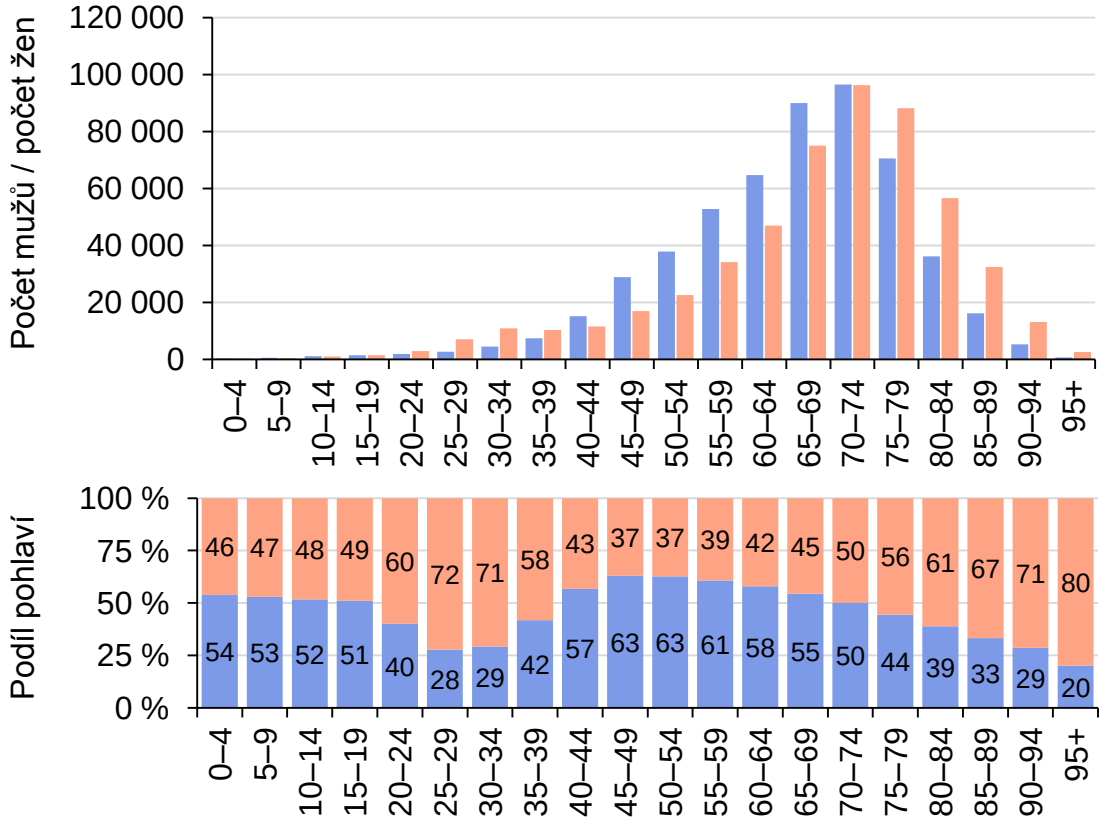
Celkový počet diabetiků zahrnuje všechny osoby, které na základě vykázaných dat v daném roce splnily definiční kritérium pro DM.
Za léčené diabetiky považujeme pacienty, kteří mají v daném roce záznam o léčbě inzulínem a/nebo perorálními antidiabetiky (léčiva z ATC skupiny A10A a/nebo A10B). Osoby, u kterých není dostupný žádný záznam o antidiabetické léčbě, mohou být léčeny pouze dietou a/nebo se jedná o prediabetes.

Demografický profil osob s diabetes mellitus (2021) – celkem / s antidiabetiky

Zdroj: NRHZS 2010–2021

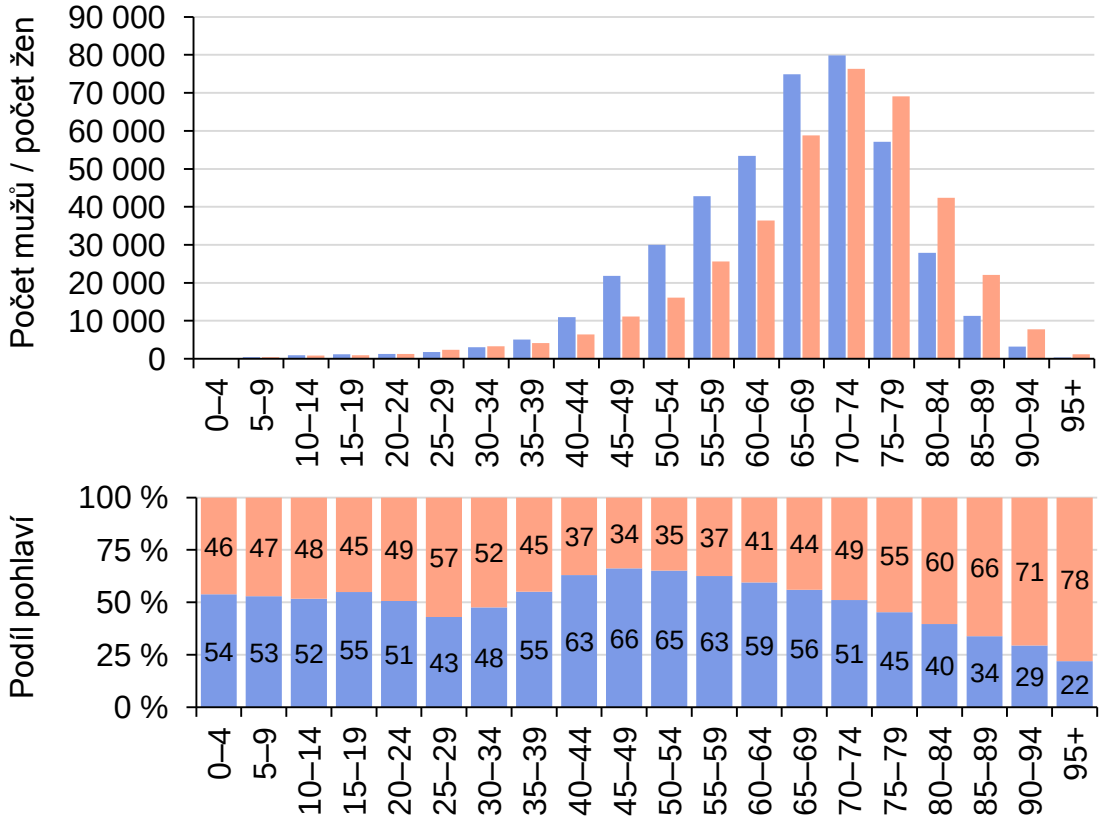
Pohlaví a věková struktura osob, u kterých byl v roce 2021 zaznamenán diabetes mellitus (bez ohledu na přítomnost antidiabetické léčby):

Věk	N (%)	Průměr (SD)	Medián (IQR)
Muži	535 085 (50,2 %)	65,2 (13,2)	67 (58; 74)
Ženy	531 304 (49,8 %)	67,8 (15,0)	71 (61; 78)
Celkem	1 066 389 (100 %)	66,5 (14,2)	69 (59; 76)



Pohlaví a věková struktura osob, které v roce 2021 užívaly léčiva z ACT skupiny A10 = antidiabetika:

Věk	N (%)	Průměr (SD)	Medián (IQR)
Muži	427 302 (52,5 %)	65,4 (12,6)	67 (58; 74)
Ženy	386 571 (47,5 %)	69,1 (13,2)	71 (63; 78)
Celkem	813 873 (100 %)	67,2 (13,0)	69 (60; 76)



Komorbidity u pacientů s DM – dle věku

Zdroj: NRHZS 2010–2021

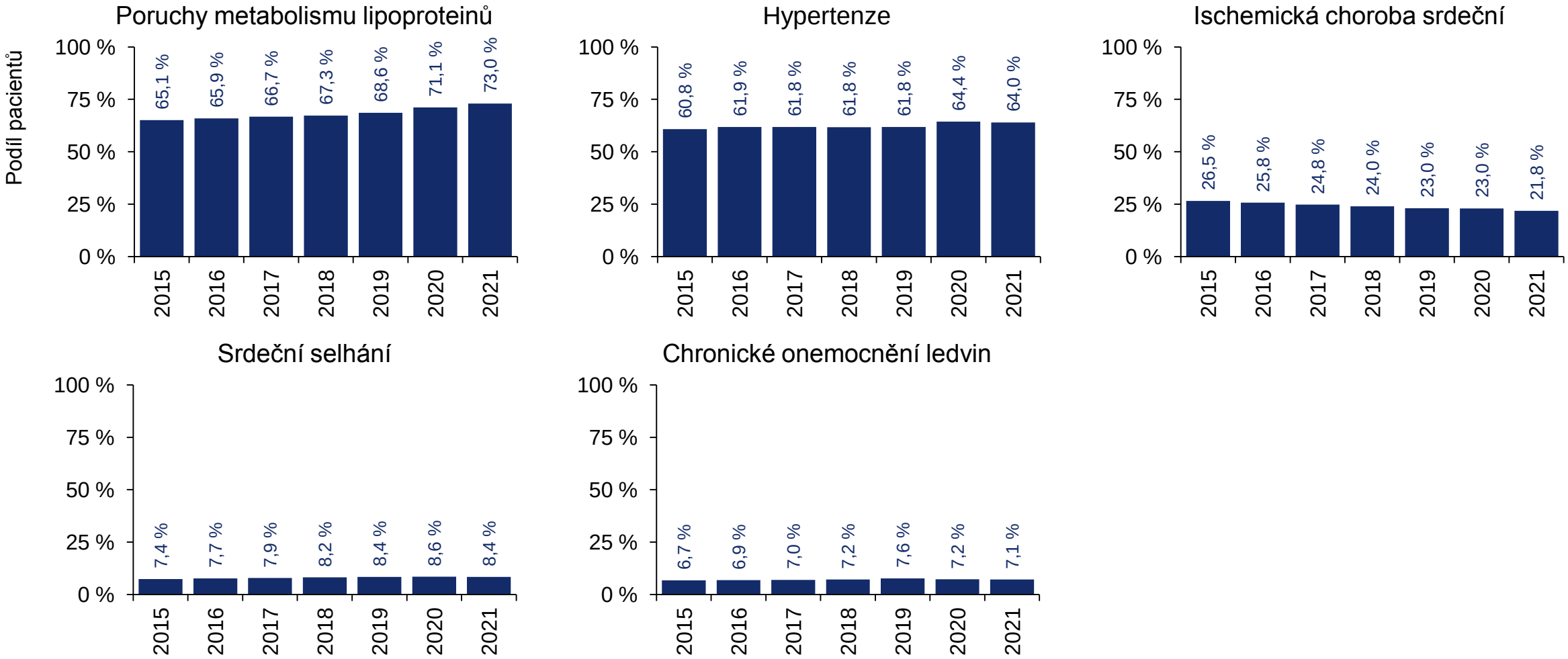
Relativní četnost výskytu komorbidit u pacientů, kteří byli v roce 2021 léčení antidiabetiky (ATC skupina A10):

	Celkem	< 40 let	40–49 let	50–59 let	60–69 let	70–79 let	80+ let
Počet pacientů (2021)	813 873	27 054	50 319	114 391	223 549	282 426	116 134
Poruchy metabolismu lipoproteinů	73,0 %	21,6 %	55,3 %	69,2 %	77,5 %	79,8 %	71,0 %
Hypertenze	64,0 %	14,0 %	42,2 %	55,2 %	64,7 %	71,8 %	73,5 %
Ischemická choroba srdeční	21,8 %	1,5 %	5,8 %	11,1 %	18,0 %	27,1 %	38,5 %
Srdeční selhání	8,4 %	0,6 %	1,9 %	3,3 %	5,8 %	10,1 %	19,1 %
Chronické onemocnění ledvin	7,1 %	2,7 %	2,9 %	3,6 %	5,1 %	8,8 %	13,3 %

Komorbidity u pacientů s DM – časový vývoj (2015–2021)

Zdroj: NRHZS 2010–2021

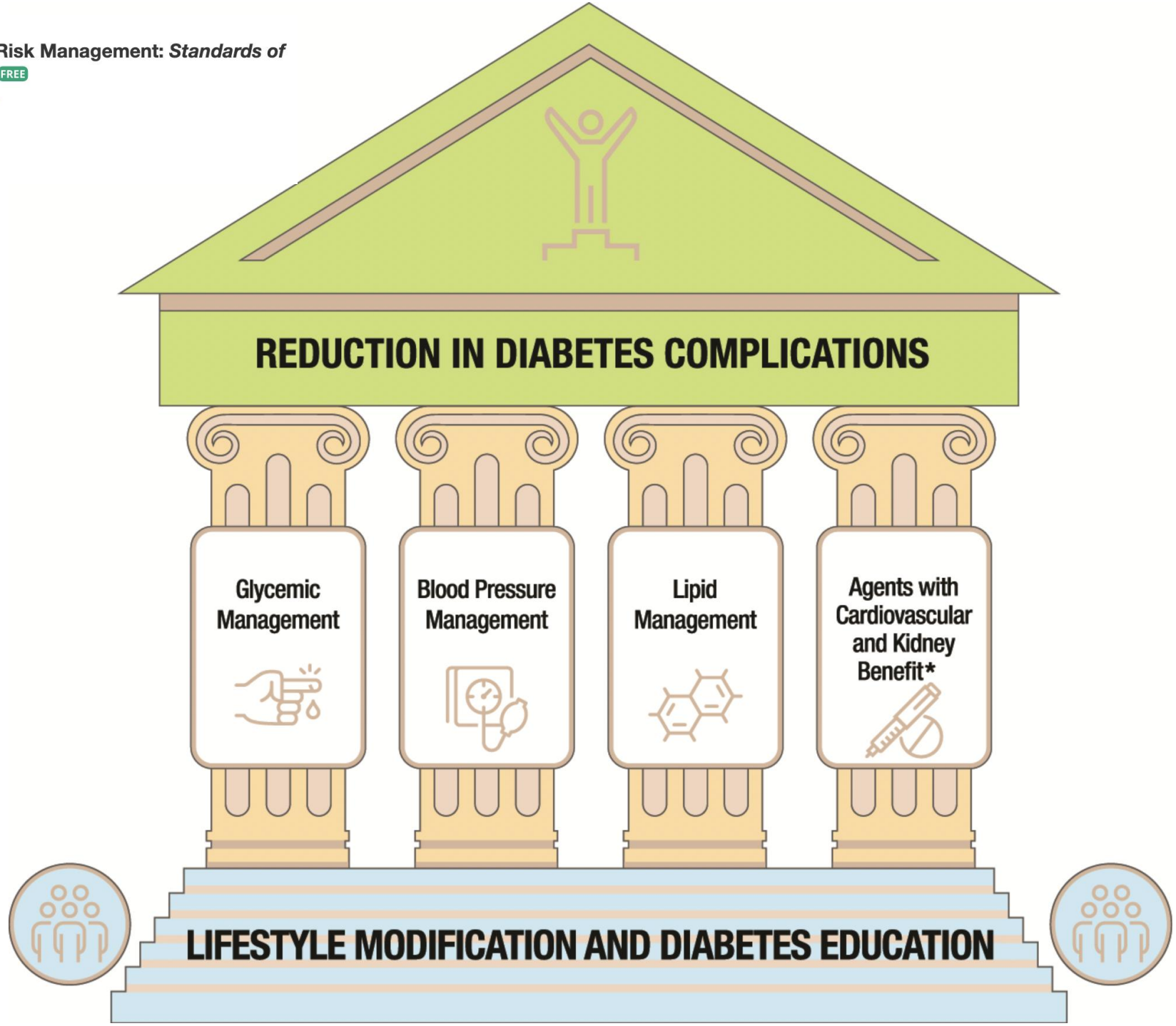
Relativní četnost výskytu komorbidit u pacientů, kteří byli v letech 2015–2021 léčení antidiabetiky (ATC skupina A10):



10. Cardiovascular Disease and Risk Management: *Standards of Medical Care in Diabetes—2022* FREE

American Diabetes Association Professional Practice Committee

Diabetes Care 2022;45(Supplement_1):S144–S174
https://doi.org/10.2337/dc22-S010



STANDARDS OF CARE | DECEMBER 16 2021

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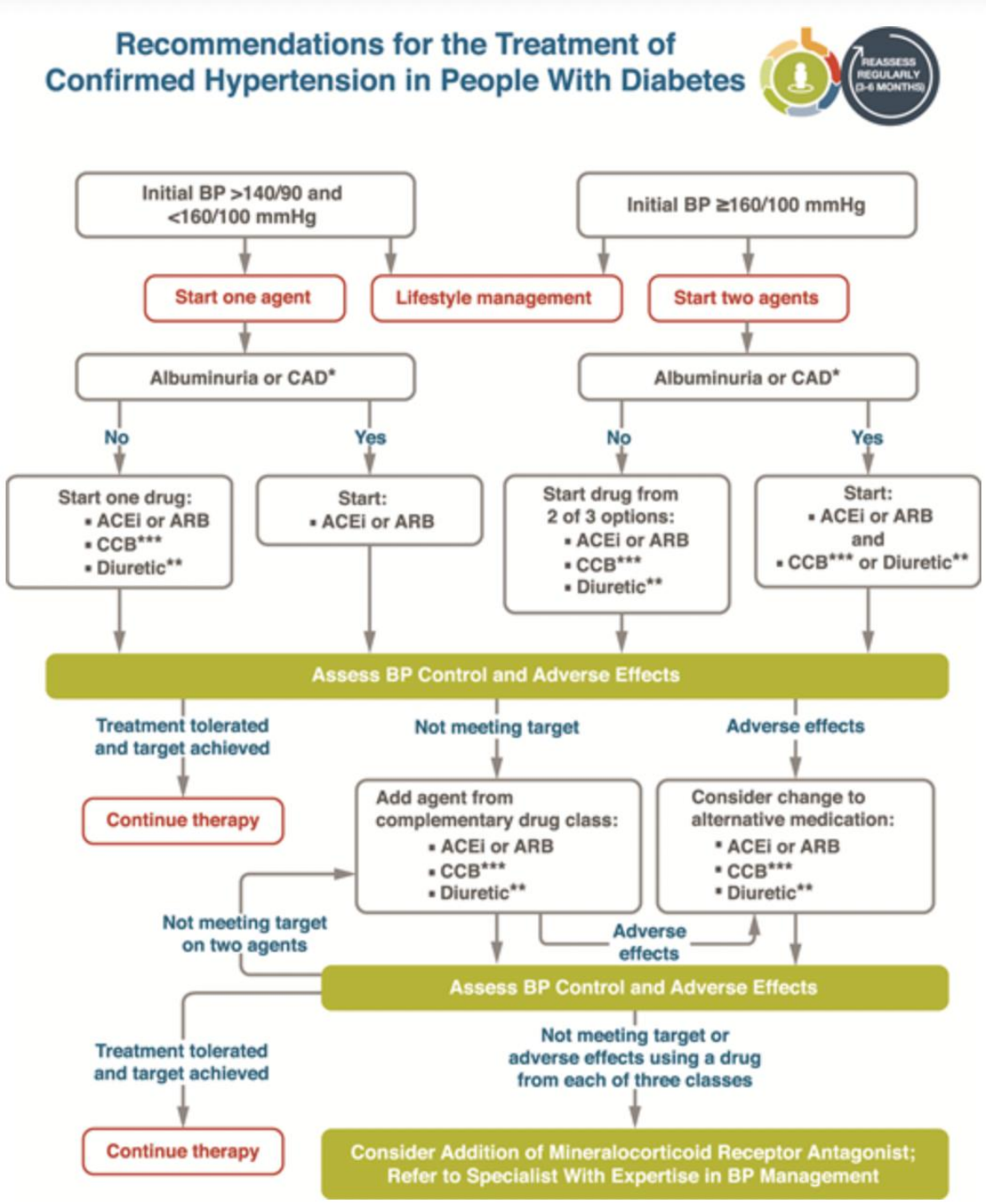

Recommendations for the treatment of confirmed hypertension in people with diabetes. *An ACE inhibitor (ACEi) or angiotensin receptor blocker (ARB) is suggested to treat hypertension for patients with coronary artery disease (CAD) or urine albumin-to-creatinine ratio 30–299 mg/g creatinine and strongly recommended for patients with urine albumin-to-creatinine ratio ≥300 mg/g creatinine. **Thiazide-like diuretic; long-acting agents shown to reduce cardiovascular events, such as chlorthalidone and indapamide, are preferred. ***Dihydropyridine calcium channel blocker (CCB). BP, blood pressure. Adapted from de Boer et al. (17).

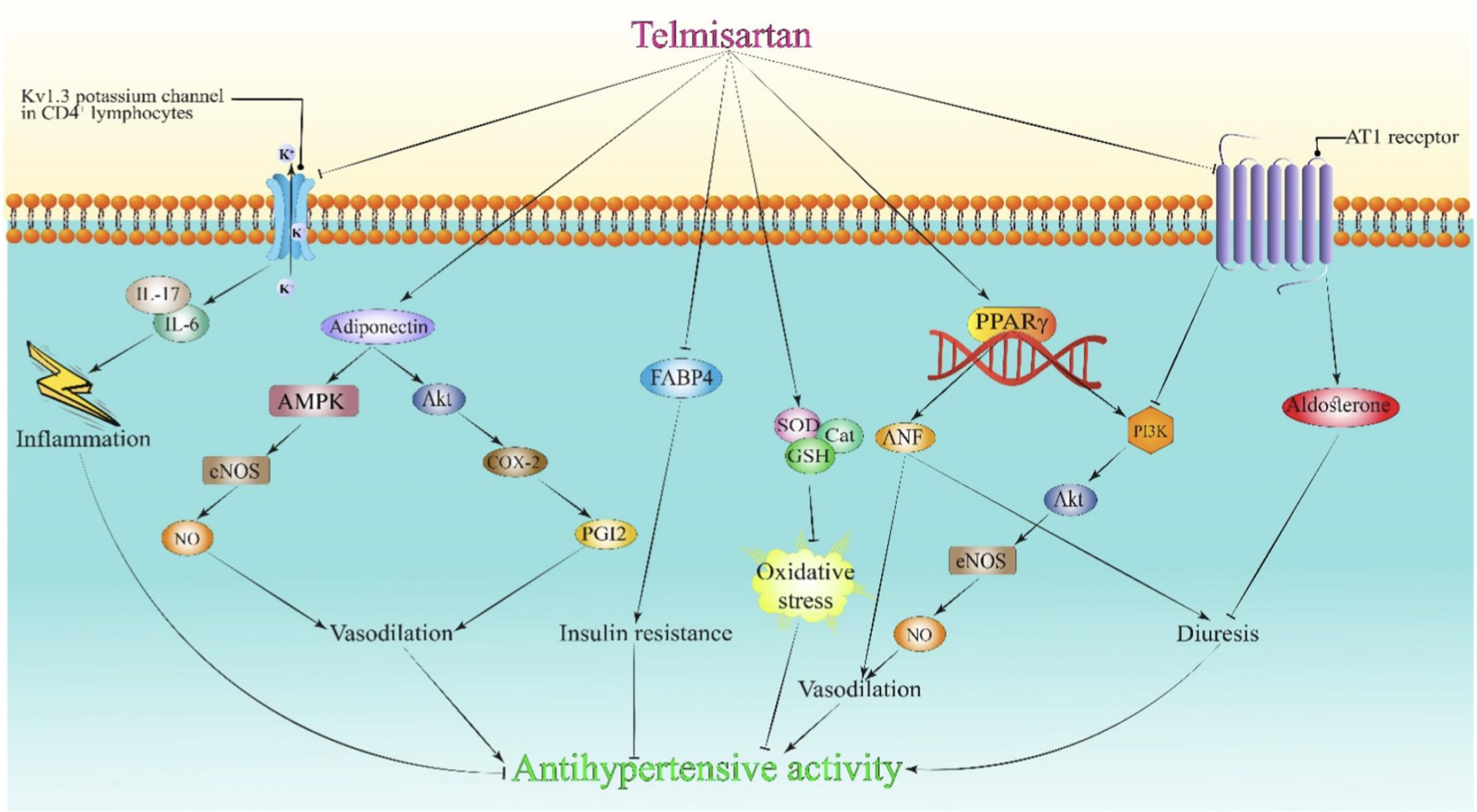

Fig. 2. Antihypertensive mechanisms of telmisartan. Schematic illustration of different mechanisms of antihypertensive activity of telmisartan; Telmisartan has antagonistic activity on AT1 receptors and a partial PPARγ-agonistic effect. In addition, telmisartan modulates adipokine levels and attenuates oxidative stress. Moreover, telmisartan reduces the expression of K_V 1.3 voltage-gated potassium channel in lymphocytes. Telmisartan also exerts diuretic activity via reducing aldosterone secretion and increases the expression of cyclooxygenase-2 via activation of Akt which ultimately leads to the production of prostacyclin and vasodilation activity.

Imenshahidi M. et al.: Effects of telmisartan on metabolic syndrome components: a comprehensive review. Biomedicine & Pharmacotherapy 171 (2024). https://doi.org/10.1016/j.biopha.2024.116169

International Journal of Cardiology 156 (2012) 92–122

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Letters to the Editor

Telmisartan improves insulin sensitivity: A meta-analysis of randomized head-to-head trials

Hisato Takagi *, Takuya Umemoto

Department of Cardiovascular Surgery, Shizuoka Medical Center,

	Telmisartan			Other ARB				Std. Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI
Bahadir 2007	14.1	4	21	13.2	4.3	21	3.9%	0.21 [-0.39, 0.82]
Clodi 2009	18.1	16	32	15.2	6.5	29	4.8%	0.23 [-0.27, 0.73]
de Luis 2010	11.6	8.2	34	14.8	8.4	31	4.9%	-0.38 [-0.87, 0.11]
Derosa 2004	19.1	3.9	40	19	4	39	5.4%	0.03 [-0.42, 0.47]
Derosa 2007	17.1	3.6	92	18	3.7	90	7.1%	-0.25 [-0.54, 0.05]
Fogari 2009	9.7	2.6	50	9.9	2.8	50	5.9%	-0.07 [-0.47, 0.32]
Hong 2011	67	27	36	77	44	37	5.2%	-0.27 [-0.73, 0.19]
Ichikawa 2007	9.3	4.5	26	11.7	7.4	27	4.4%	-0.38 [-0.93, 0.16]
Makita 2008	7.37	5.62	46	5.67	5.51	44	5.7%	0.30 [-0.11, 0.72]
Minami 2009	18.1	19.8	21	18.2	15.1	21	3.9%	-0.01 [-0.61, 0.60]
Nakayama 2008	9	6.8	20	10.4	1.6	20	3.8%	-0.28 [-0.90, 0.35]
Negro 2006	32	3.4	22	39	3.8	21	3.1%	-1.91 [-2.64, -1.18]
Ozaki 2010	11.4	19	74	9.4	31.3	153	7.3%	0.07 [-0.21, 0.35]
Perl 2010	5.1	4.9	24	7.3	7.8	24	4.2%	-0.33 [-0.90, 0.24]
Rizos 2010	8.2	4.1	52	10.6	5.1	99	6.5%	-0.50 [-0.84, -0.16]
Tomiyama 2007	8.1	5	40	9.6	6.6	40	5.4%	-0.25 [-0.69, 0.19]
Vitale 2005	16.93	8.91	20	20.14	9.49	20	3.8%	-0.34 [-0.97, 0.28]
Watanabe 2010	9.3	5.5	38	12.8	10.3	47	5.5%	-0.41 [-0.84, 0.02]
Yamada 2008	13.9	7.1	28	13.4	11.5	28	4.6%	0.05 [-0.47, 0.58]
Yano 2007	11.8	8	30	11.2	7.6	21	4.3%	0.08 [-0.48, 0.63]
Total (95% CI)			**746**			**862**	**100.0%**	**-0.19 [-0.35, -0.03]**

Heterogeneity: $Tau^2 = 0.07$; $Chi^2 = 43.51$, df = 19 (P = 0.001); $I^2 = 56\%$

Test for overall effect: Z = 2.35 (P = 0.02)

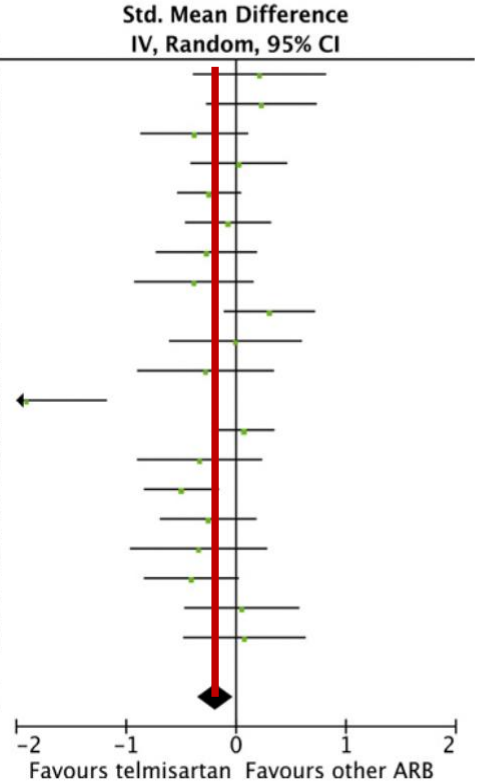

Fig. 1. Final fasting insulin level among patients randomized to telmisartan versus other angiotensin II type 1 receptor blocker (ARB) therapy. CI, Confidence interval; IV, inverse variance; SD, standard deviation.

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Letters to the Editor

Telmisartan improves insulin sensitivity: A meta-analysis of randomized head-to-head trials

Hisato Takagi *, Takuya Umemoto

Department of Cardiovascular Surgery, Shizuoka Medical Center

	Telmisartan			Other ARB				Std. Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI
ATHLETE 2011	2.2	1	14	2.1	0.9	14	3.4%	0.10 [-0.64, 0.84]
Bahadir 2007	1.9	0.5	21	1.8	0.6	21	4.1%	0.18 [-0.43, 0.78]
Clodi 2009	10.2	16.7	32	4.2	2.3	29	4.5%	0.49 [-0.03, 1.00]
de Luis 2010	3.9	3.1	34	4.5	3.7	31	4.6%	-0.17 [-0.66, 0.31]
Derosa 2004	4.9	3.5	40	5.1	3.6	39	4.9%	-0.06 [-0.50, 0.39]
Derosa 2007	5.1	2.7	92	5.7	3	90	5.6%	-0.21 [-0.50, 0.08]
Fogari 2009	-5.73	1.96	50	-3.77	0.78	50	4.9%	-1.30 [-1.74, -0.87]
Georgescu 2009	4.48	1.11	28	5.95	1.12	26	4.1%	-1.30 [-1.89, -0.71]
Hong 2011	2.7	2.7	36	2.9	2.7	37	4.8%	-0.07 [-0.53, 0.39]
Ichikawa 2007	2.6	1.5	26	3.3	2.4	27	4.4%	-0.34 [-0.89, 0.20]
Makita 2008	1.98	1.75	46	1.46	0.71	44	5.0%	0.38 [-0.03, 0.80]
Minami 2009	5.87	7.61	21	5.5	5.89	21	4.1%	0.05 [-0.55, 0.66]
Nakayama 2008	2.4	1.4	20	2.3	1.2	20	4.0%	0.08 [-0.54, 0.70]
Negro 2006	202.3	25.4	22	225.3	23.9	21	3.9%	-0.91 [-1.55, -0.28]
Ozaki 2010	4.5	9.6	74	3.3	13	153	5.7%	0.10 [-0.18, 0.38]
Perl 2010	2.2	2.3	24	3.37	4.12	24	4.2%	-0.34 [-0.92, 0.23]
Rizos 2010	1.8	1.2	52	2.8	1.6	99	5.4%	-0.67 [-1.02, -0.33]
Tomiyama 2007	2.3	0.5	40	2.7	0.6	40	4.8%	-0.72 [-1.17, -0.26]
Vitale 2005	4.24	2.36	20	5.82	2.66	20	3.9%	-0.62 [-1.25, 0.02]
Watanabe 2010	3.49	0.62	38	3.98	1.03	47	4.9%	-0.56 [-0.99, -0.12]
Yamada 2008	4.1	2.2	28	4.4	3.6	28	4.5%	-0.10 [-0.62, 0.43]
Yano 2007	3.6	2.5	30	3.1	1.9	21	4.3%	0.22 [-0.34, 0.78]
Total (95% CI)			**788**			**902**	**100.0%**	**-0.26 [-0.47, -0.06]**

Heterogeneity: $Tau^2 = 0.17$; $Chi^2 = 84.59$, df = 21 ($P < 0.00001$); $I^2 = 75\%$

Test for overall effect: $Z = 2.54$ ($P = 0.01$)

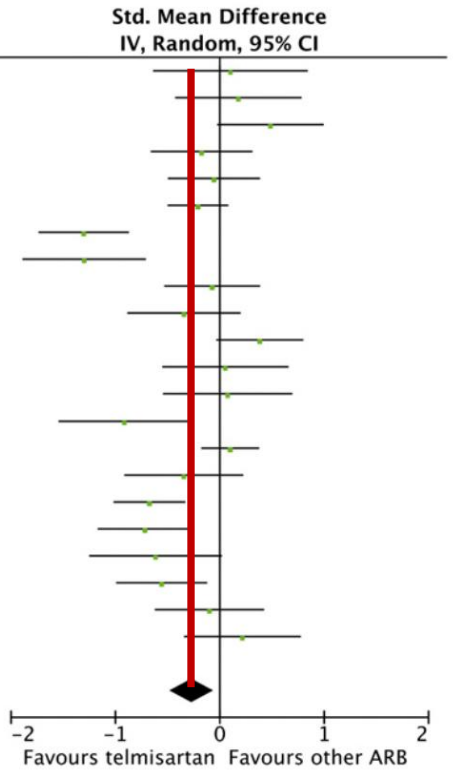

Fig. 2. Final index of insulin sensitivity among patients randomized to telmisartan versus other angiotensin II type 1 receptor blocker (ARB) therapy. CI, Confidence interval; IV, inverse variance; SD, standard deviation.

Journal of the American Society of Hypertension
Volume 7, Issue 3, May–June 2013, Pages 229-235

Research Article

Telmisartan as a metabolic sartan: The first meta-analysis of randomized controlled trials in metabolic syndrome

Hisato Takagi MD, PhD, Masao Niwa MD, Yusuke Mizuno MD, Shin-nosuke Goto MD, Takuya Umemoto MD, PhD, ALICE (All-Literature Investigation of Cardiovascular Evidence) Group

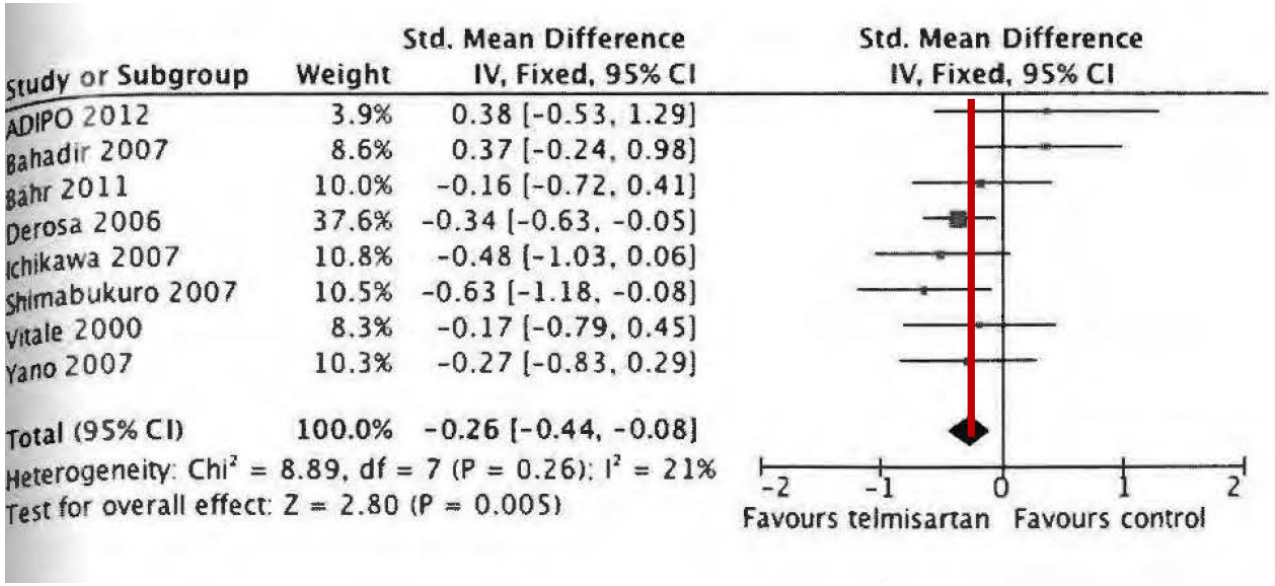

Figure 3. Percent changes from baseline to final levels of glycosylated hemoglobin among patients with metabolic syndrome randomized to telmisartan versus control therapy. *CI, confidence interval; df, degrees of freedom; IV, inverse variance; Std, standardized.*

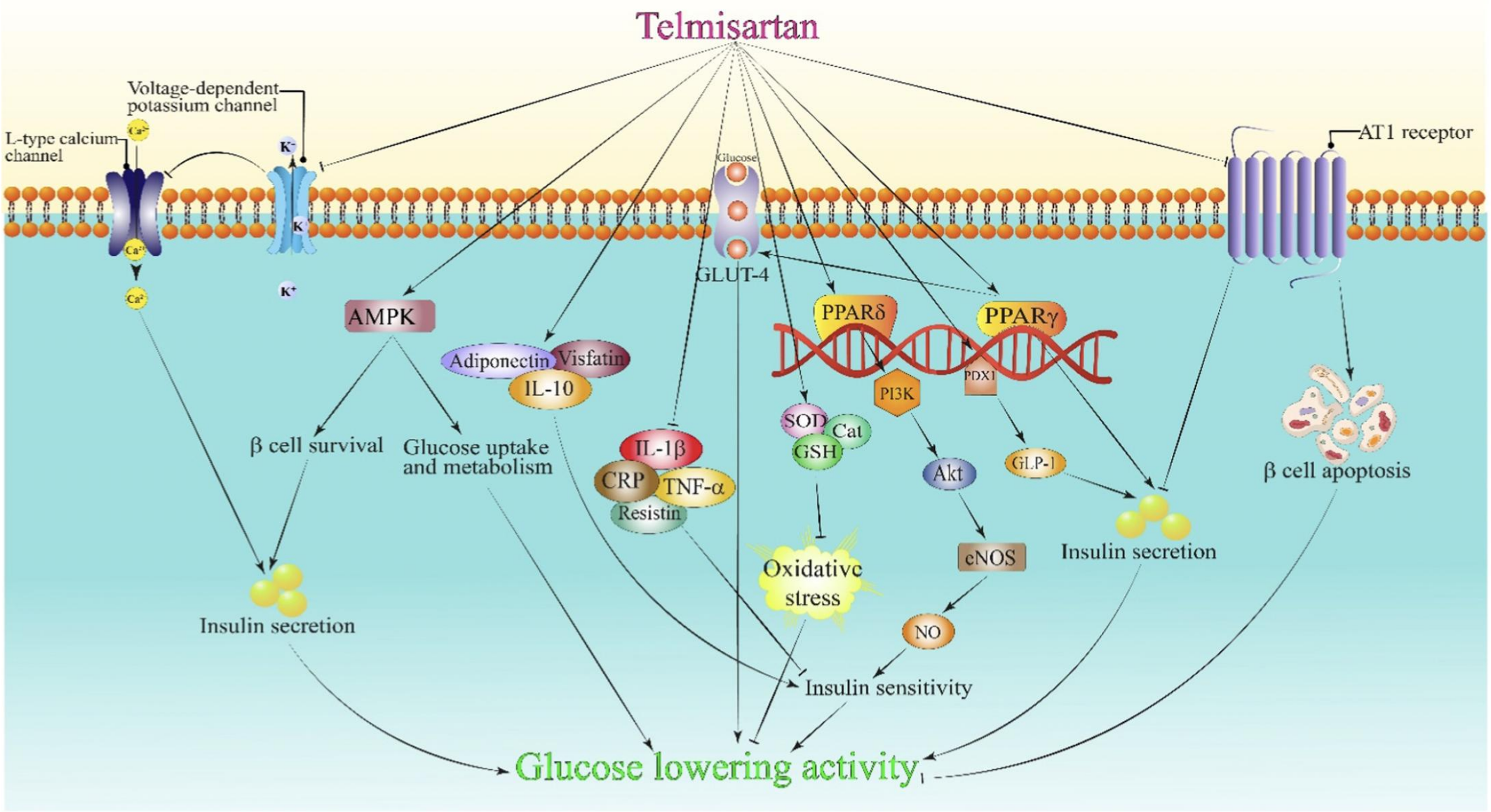

Fig. 1. Antidiabetic mechanisms of telmisartan. Schematic illustration of different mechanisms of antidiabetic activity of telmisartan; Telmisartan has a partial PPARγ-agonistic effect and antagonistic activity on AT1 receptors. Moreover, telmisartan has been reported to have an agonist activity on PPARα and PPARδ receptors. In addition, telmisartan modulates adipokine levels and attenuates oxidative stress.

Imenshahidi M. et al.: Effects of telmisartan on metabolic syndrome components: a comprehensive review. Biomedicine & Pharmacotherapy 171 (2024). https://doi.org/10.1016/j.biopha.2024.116169

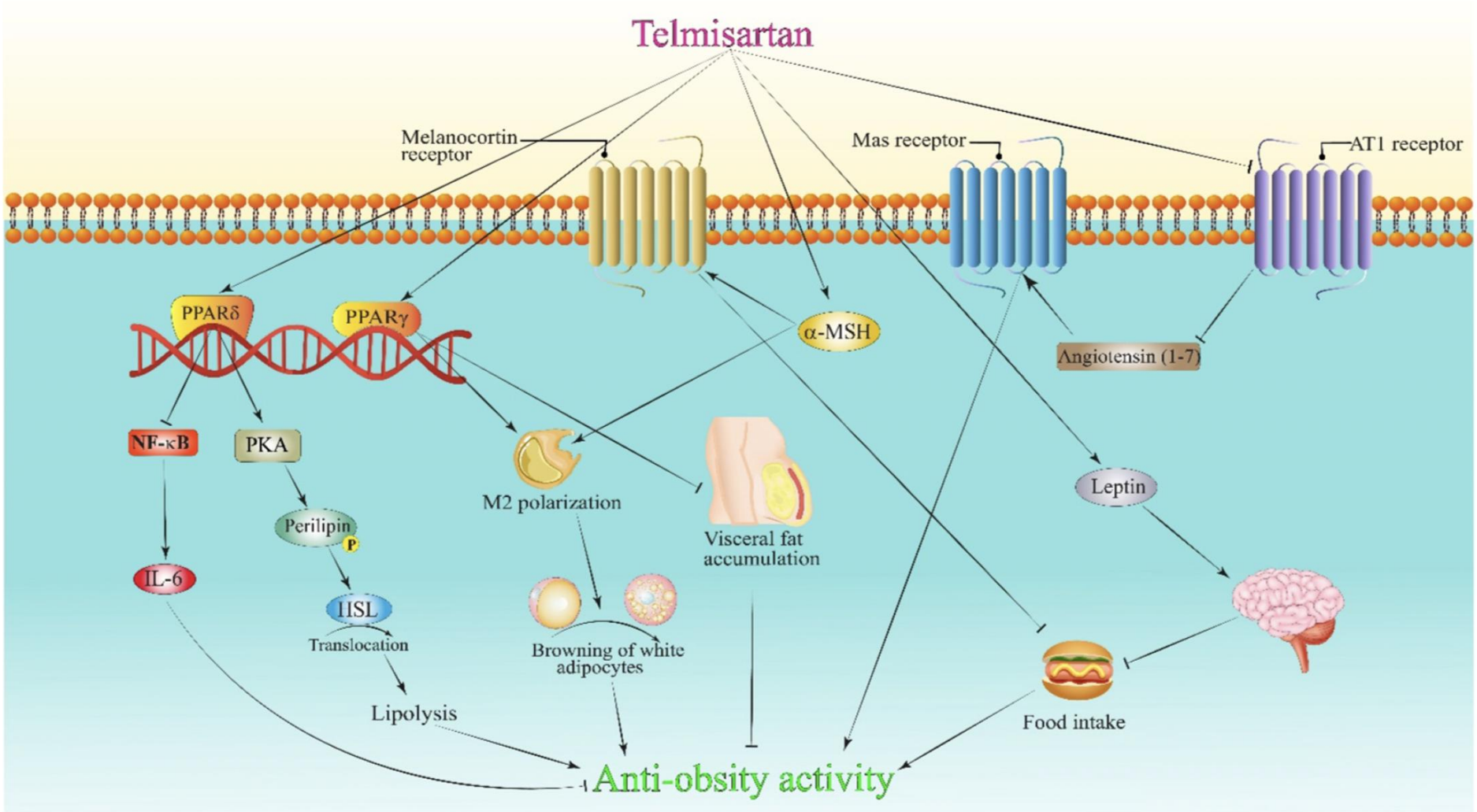

Fig. 4. Antiobesity mechanisms of telmisartan. Schematic illustration of different mechanisms of antiobesity activity of telmisartan; Telmisartan has a partial PPARγ-agonistic effect that induces M2 polarization and the browning of white adipocytes. Telmisartan has an antagonistic activity on AT1 receptors activates the ACE2/rMAS axis and improves weight control via the activation of the Mas receptors. Moreover, telmisartan preserves leptin transport across the blood-brain barrier and increases leptin sensitivity and prevents diet-induced obesity. In addition, telmisartan induces the PKA-dependent phosphorylation of perilipin via activation of PPAR-δ receptors and thereby stimulates lipolysis.

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