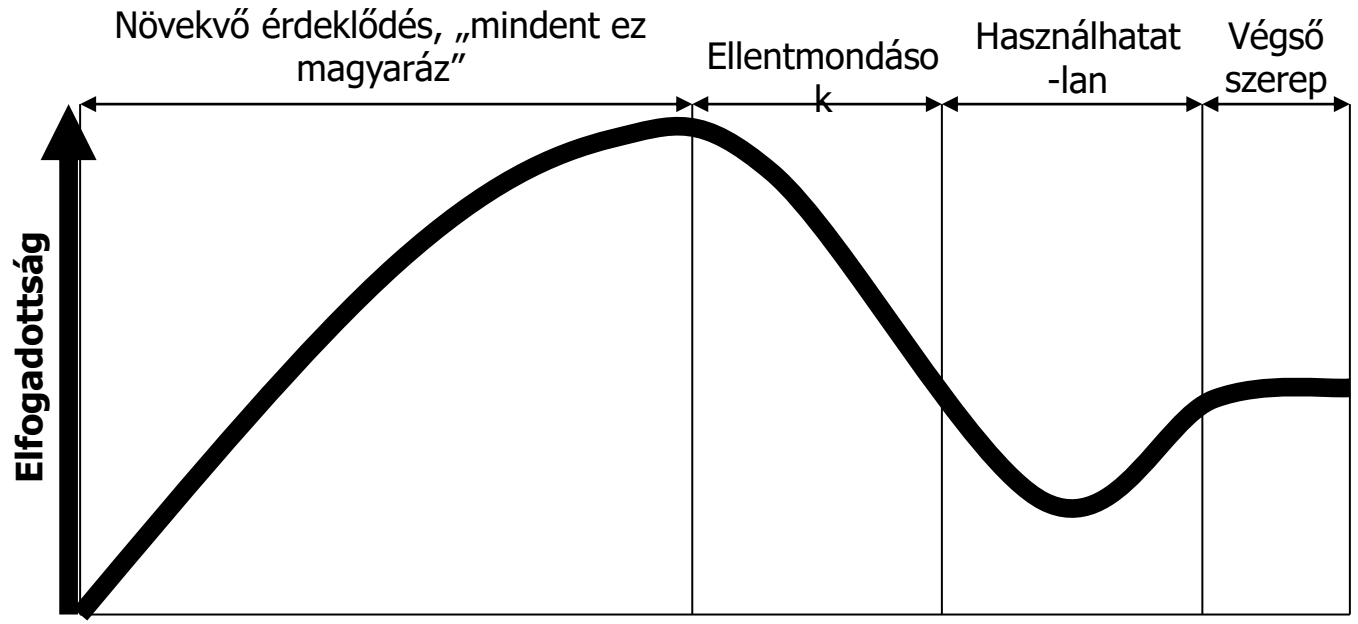
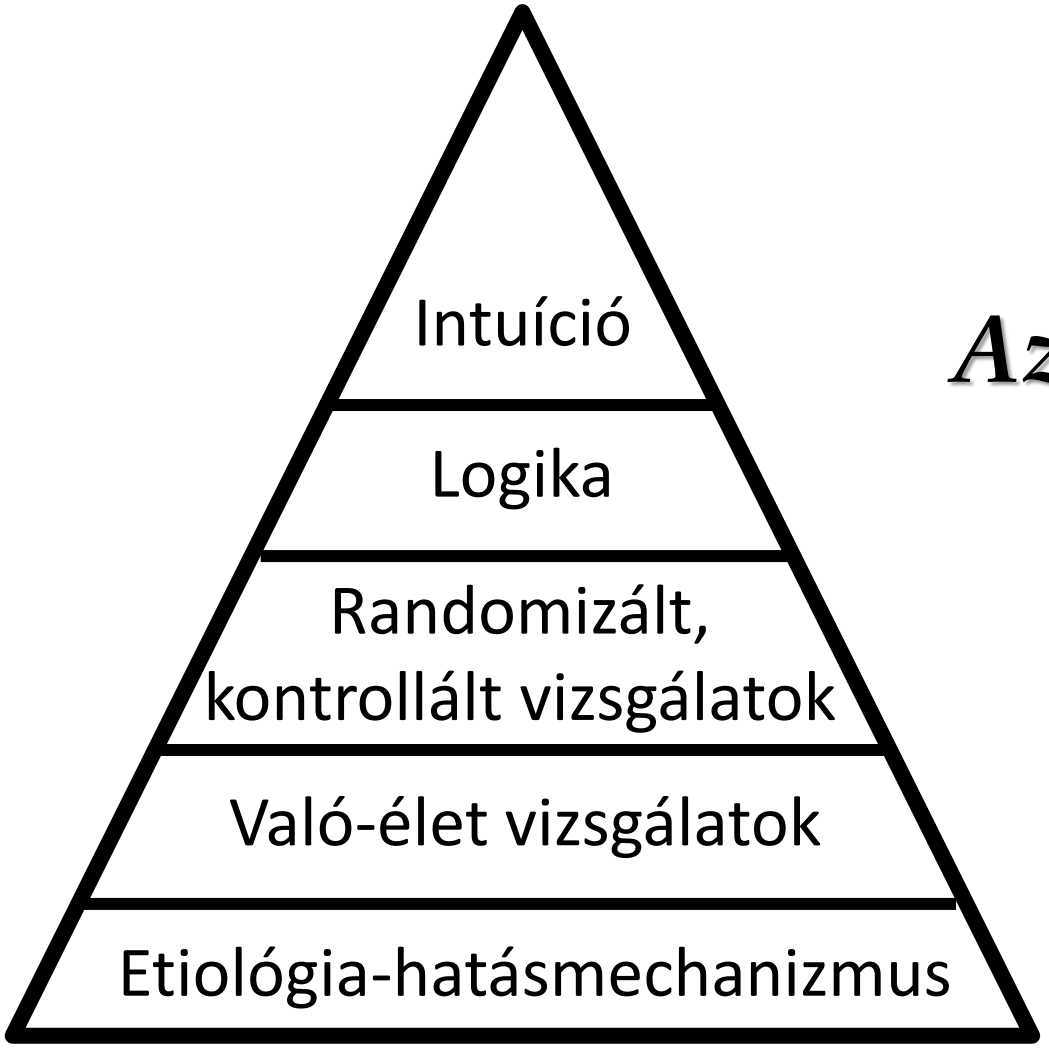
Újdonságok a diabetológiában és a diabeteses vesebetegség

Wittmann István
II. sz. Belgyógyászati Klinika és Nephrologiai Centrum
PTE ÁOK KK

Az orvosi ismeretek elfogadottságának természetrajza

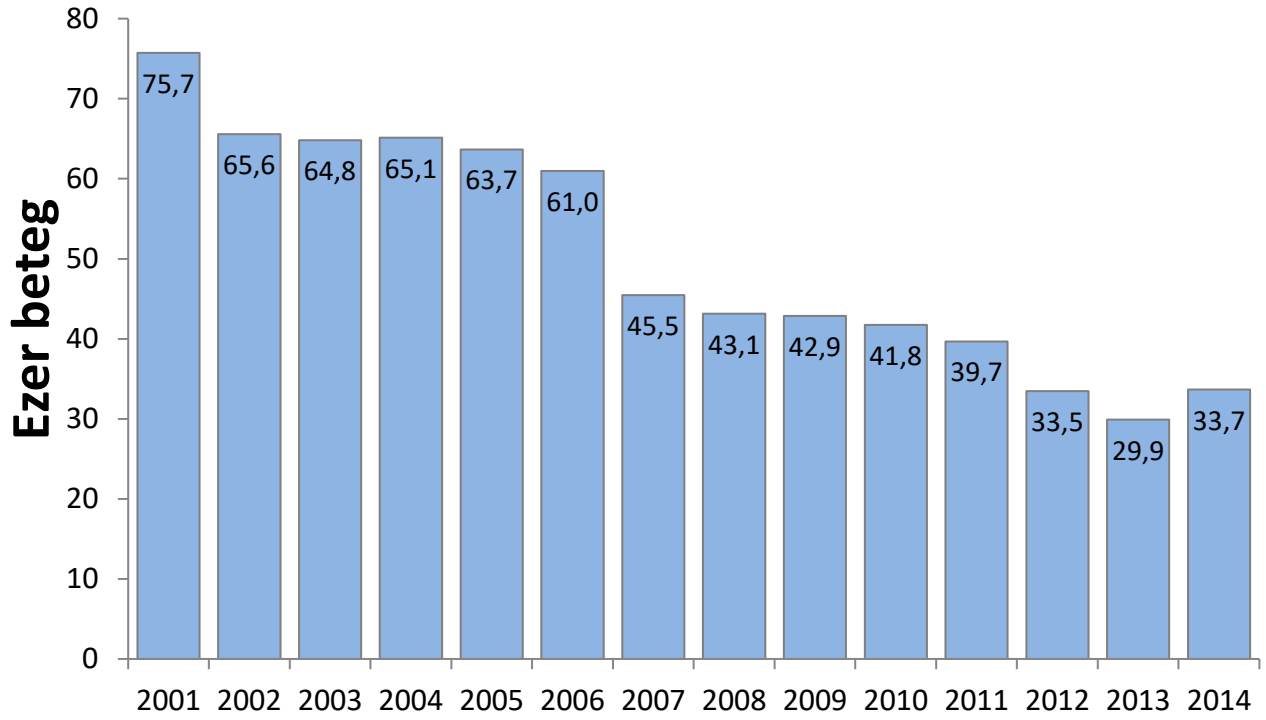


A szerző saját véleménye

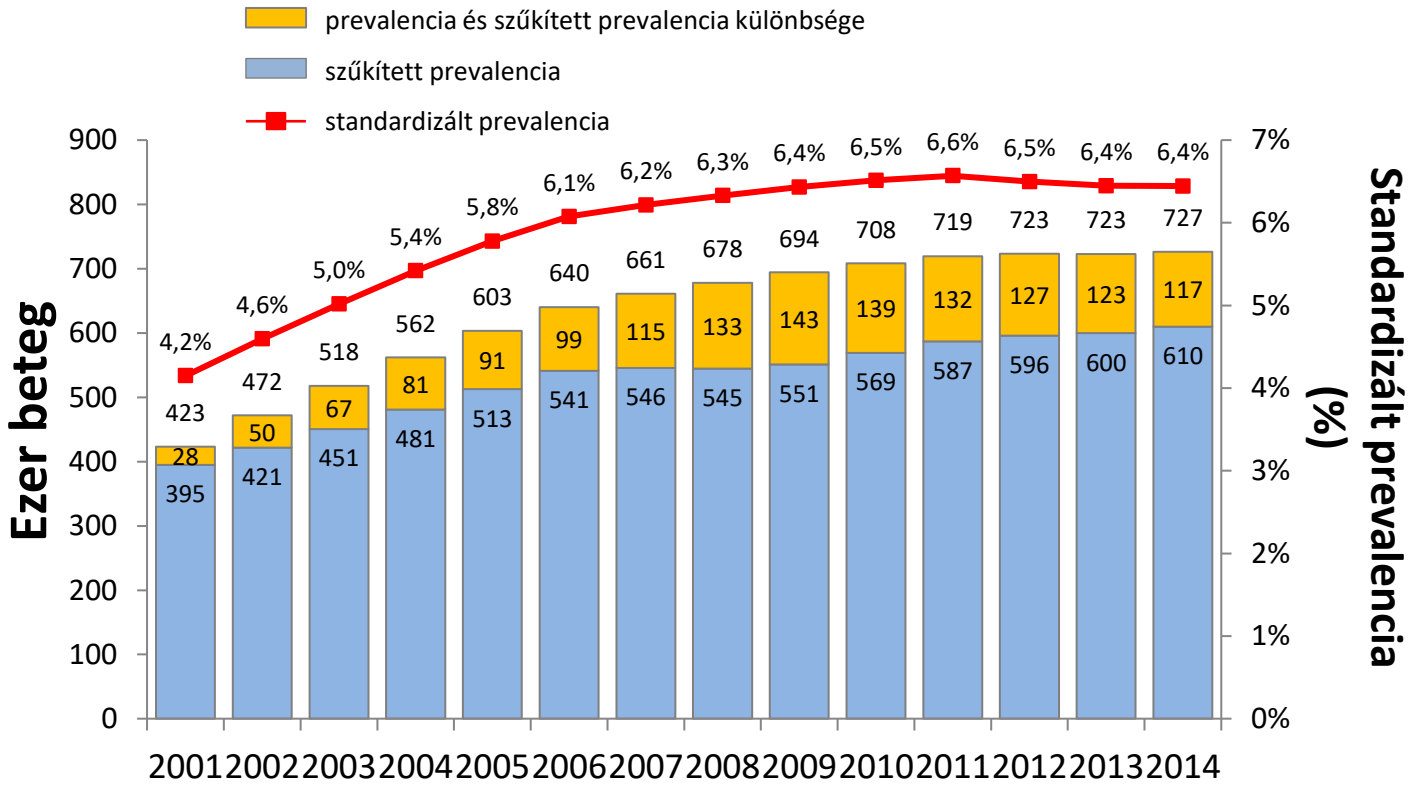


Az orvosi művészet alapjai

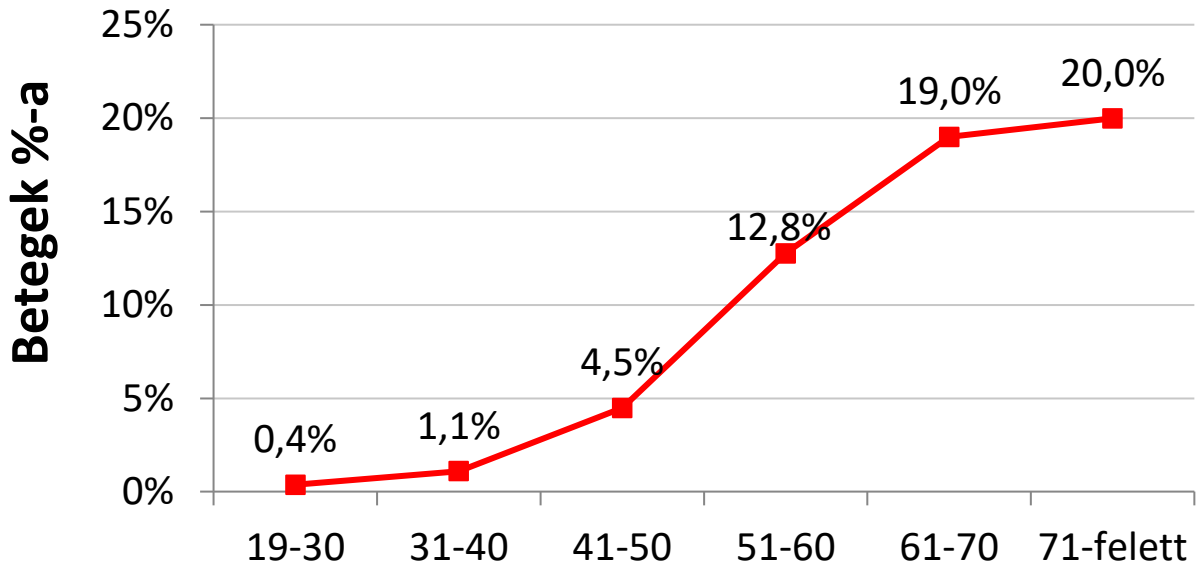
A 2-es típusú diabetes incidenciája (az OEP adatbázisban új betegként megjelenő esetek száma) 2001-2014 között



A 2-es típusú diabetes prevalencia-adatai 2001-2014 között



A 2-es típusú diabetesben szenvedők teljes lakosság számához viszonyított részaránya életkori dekádok szerint (2011. évi adatok)



T2DM, NAFLD, CVD, CKD, karcinogenesis

Túlzott energiabevitel

↓

Dysbacteriosis

↓

Abdominális zsírsejtfenotípus-váltás (hormonok, cytokinek)

↓

Szisztémás szubklinikus gyulladás és oxidatív stressz

↓

T2DM, NAFLD, CVD, CKD, karcinogenezis

A prediabetes diagnózisa

Emelkedett éhomi vércukor (IFG)

Éhomi vércukor 6,1 – 6,9 mmol/l

2 h vércukor <7,8 mmol/l

Csökkent glukóz tolerancia (IGT)

Éhomi vércukor <6,1 mmol/l

2 h vércukor 7,8-11,1 mmol/l

IFG + IGT

Éhomi vércukor 6,1-6,9 mmol/l

2 h vércukor 7,8-11,1 mmol/l

HbA1c

5,7-6,4%

A diabetes diagnózisa

1. Tünetek + random plazma glukóz≥11.1 mM

2. Éhomi plazma glukóz ≥7.0 mM

3. OGTT: 2 órás plazma glukóz ≥11.1 mM

4. HbA1c ≥ 6,5%

biomedicines

MDPI

Article

Incorporation of Oxidized Phenylalanine Derivatives into Insulin Signaling Relevant Proteins May Link Oxidative Stress to Signaling Conditions Underlying Chronic Insulin Resistance

Judit Mohás-Cseh [1], Gergő Attila Molnár [1], Marianna Pap [2,3], Boglárka Laczy [1], Tibor Vas [1], Melinda Kertész [1], Krisztina Németh [4], Csaba Hetényi [5], Orsolya Csikós [6], Gábor K. Tóth [6], Attila Reményi [4] and István Wittmann [1,*]

[1] 2nd Department of Medicine and Nephrology-Diabetes Center, University of Pécs Medical School, 7624, Pécs, Hungary; mohas-cseh.judit@pte.hu (J.M.-C.); molnar.gergo@pte.hu (G.A.M.); laczy.boglarka@pte.hu (B.L.); vas.tibor2@pte.hu (T.V.); meli871106@gmail.com (M.K.)
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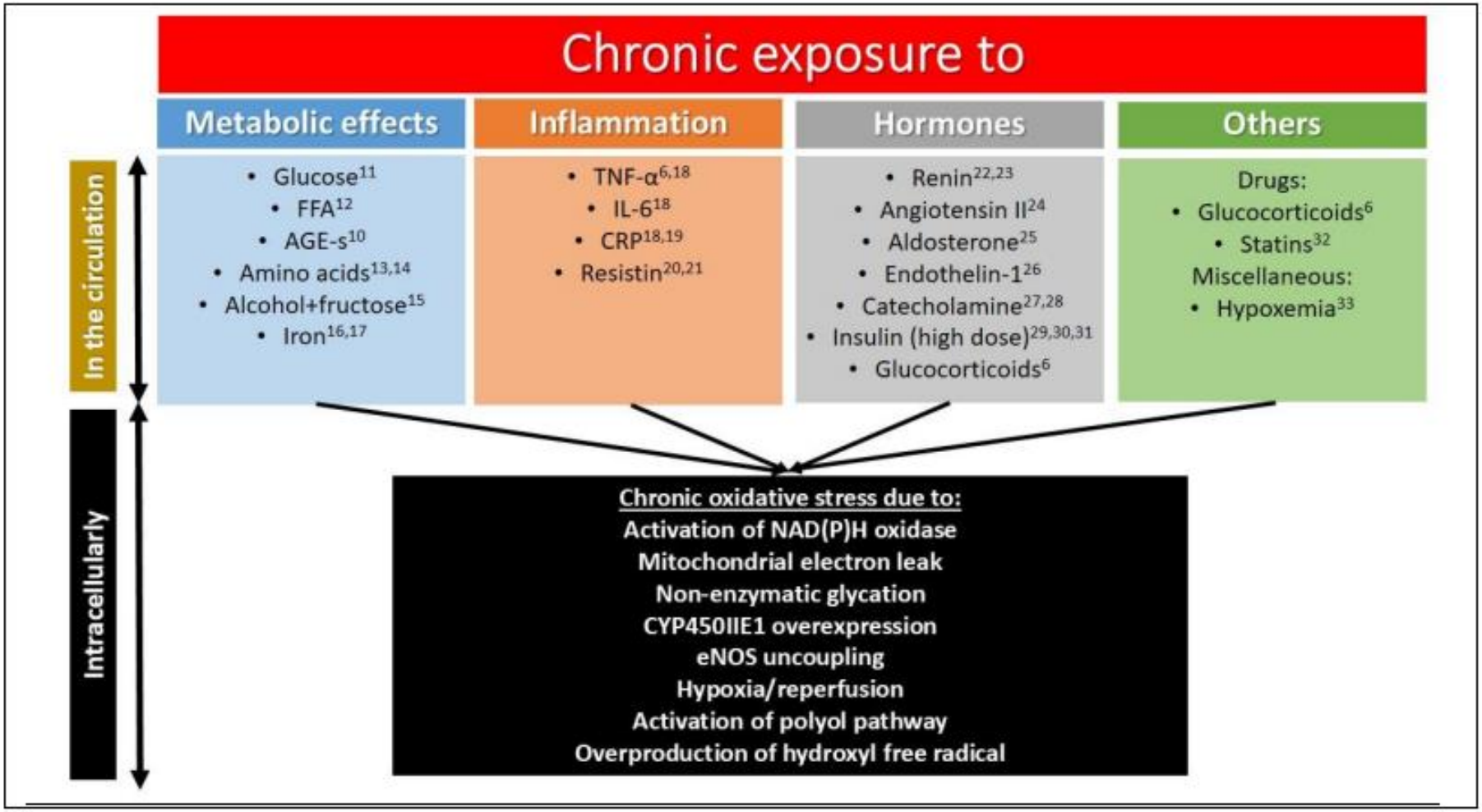
[3] Signal Transduction Research Group, Szentágothai Research Centre, University of Pécs, 7624, Pécs, Hungary
[4] Institute of Organic Chemistry, Research Centre for Natural Sciences, 1117, Budapest, Hungary; nemeth.krisztina@ttk.hu (K.N.); remenyi.attila@ttk.mta.hu (A.R.)
[5] Department of Pharmacology and Pharmacotherapy, University of Pécs Medical School, 7643, Pécs, Hungary; hetenyi.csaba@pte.hu
[6] Department of Medical Chemistry, Albert Szent-Györgyi Medical School, University of Szeged, 6725, Szeged, Hungary; csikos.orsolya@icloud.com (O.C.); toth.gabor@med.u-szeged.hu (G.K.T.)
* Correspondence: wittmann.istvan@pte.hu; Tel.: +36-72/536-050; Fax: + 36-72/536-051

Citation: Mohás-Cseh, J.; Molnár, G.A.; Pap, M.; Laczy, B.; Vas, T.; Kertész, M.; Németh, K.; Hetényi,

Biomedicines 2022, 10, 975. https:// doi.org/10.3390/ biomedicines10050975



Biomedicines 2022, 10, 975. https:// doi.org/10.3390/ biomedicines10050975

Effects of a chronic treatment by antioxidant lipoic acid on the metabolism, a metaanalysis (n=24 studies)

Parameter	Average difference	p
Fasting plasma glucose	-0,54	0,003
$HOMA_{IR}$	-0,76	<0,001
HbA_{1C}	-1,22	0,002
Triglyceride	-0,58	0,006

Metabolism Clinical and Experimental
2018; 87, 56-69

diabetesresearchandclinicalpractice 201 (2023) 110734

Contents lists available at ScienceDirect

Diabetes Research and Clinical Practice

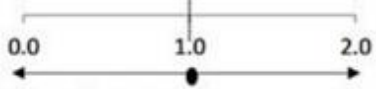
journal homepage: www.journals.elsevier.com/diabetes-research-and-clinical-practice

Morbidity and mortality of patients with diabetic neuropathy treated with pathogenetically oriented alpha-lipoic acid versus symptomatic pharmacotherapies – A nationwide database analysis from Hungary

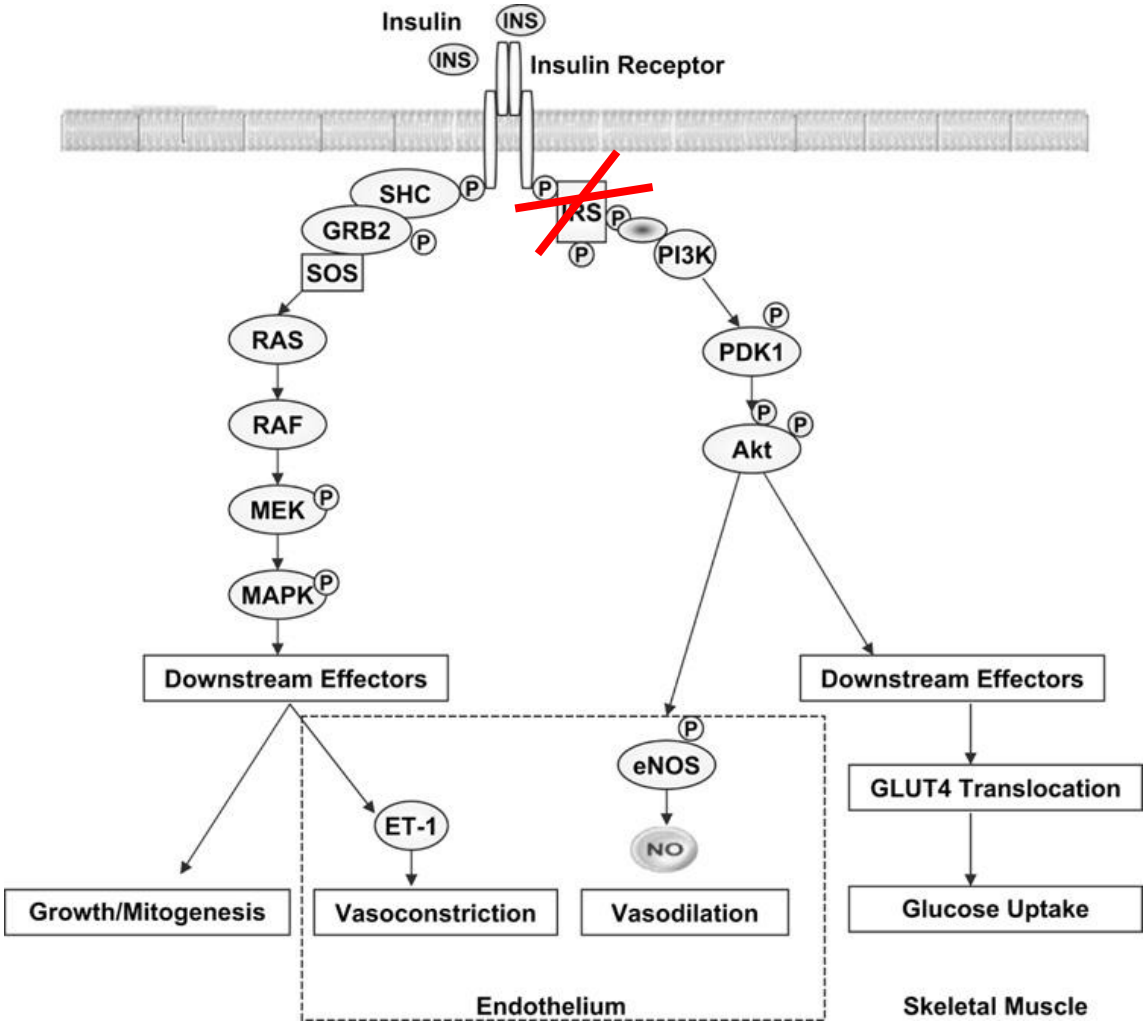
György Jermendy [a,*], György Rokszin [b], Ibolya Fábián [b], Péter Kempler [c], István Wittmann [d]

Effects of alpha-lipoic acid on the hard endpoints

	Population (n)			Events (n)						
	alpha-lipoic acid	vs	symptomatic treatment	alpha-lipoic acid	vs	symptomatic treatment	Hazard ratio	95%CI		p value
AMI with PCI	23,843	vs	23,843	219	vs	181	0.73	(0.60	- 0.89)	0.0016
Stroke with CT	23,843	vs	23,843	414	vs	379	0.71	(0.62	- 0.82)	<0.0001
HHF	23,843	vs	23,843	1,296	vs	1,168	0.72	(0.66	- 0.78)	<0.0001
LLA	23,843	vs	23,843	339	vs	217	1.05	(0.89	- 1.25)	0.5455
Cancer	23,843	vs	23,843	957	vs	777	0.83	(0.76	- 0.92)	0.0002
All-cause mortality	23,843	vs	23,843	647	vs	677	0.55	(0.49	- 0.61)	<0.0001



Favors alpha-lipoic acid cohort Favors symptomatic treatment cohort



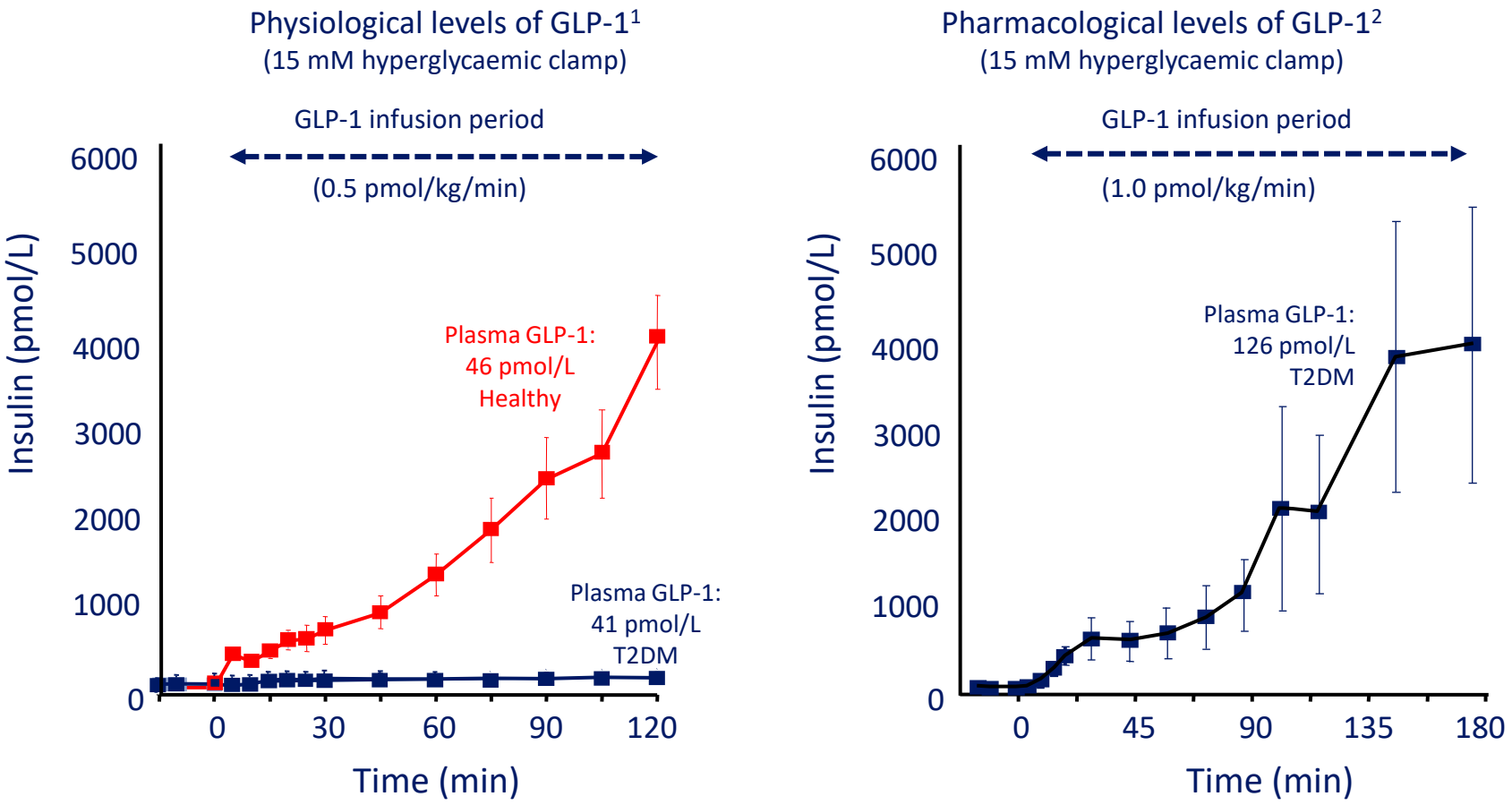
Muniyappa R et al. Endocrinol Metab Clin North Am. 2008 September ; 37(3): 685–x. doi:10.1016/j.ecl.2008.06.001.

NI

Más hormonok jelátvitele és a multi-hormonális rezisztencia

A GLP-1 rezisztencia

Bizonyíték humán vizsgálatból a GLP-1-rezisztenciára

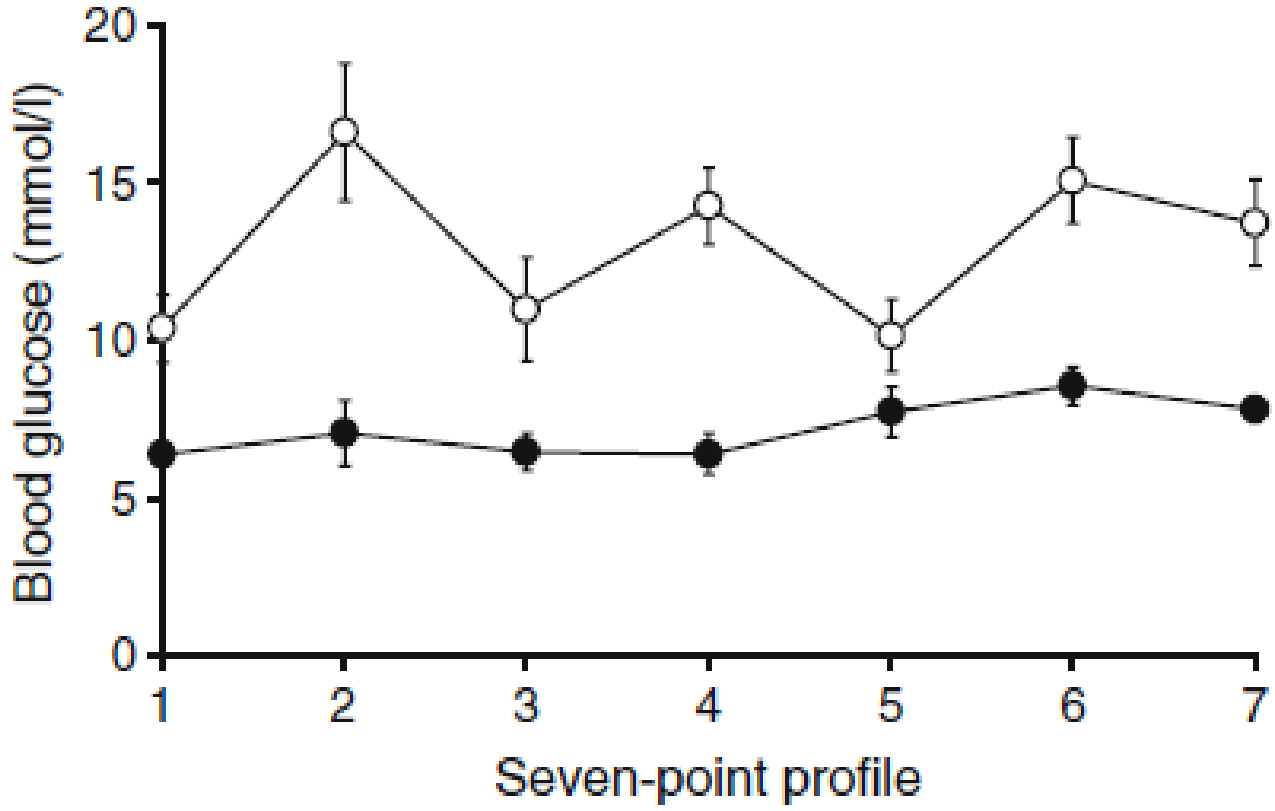


GLP-1, glucagon-like peptide 1, T2DM, type 2 diabetes mellitus

1. Højberg PV et al. *Diabetologia* 2009;52:199–207; 2. Vilsbøll T et al. *Diabetologia* 2002;45:1111–1119

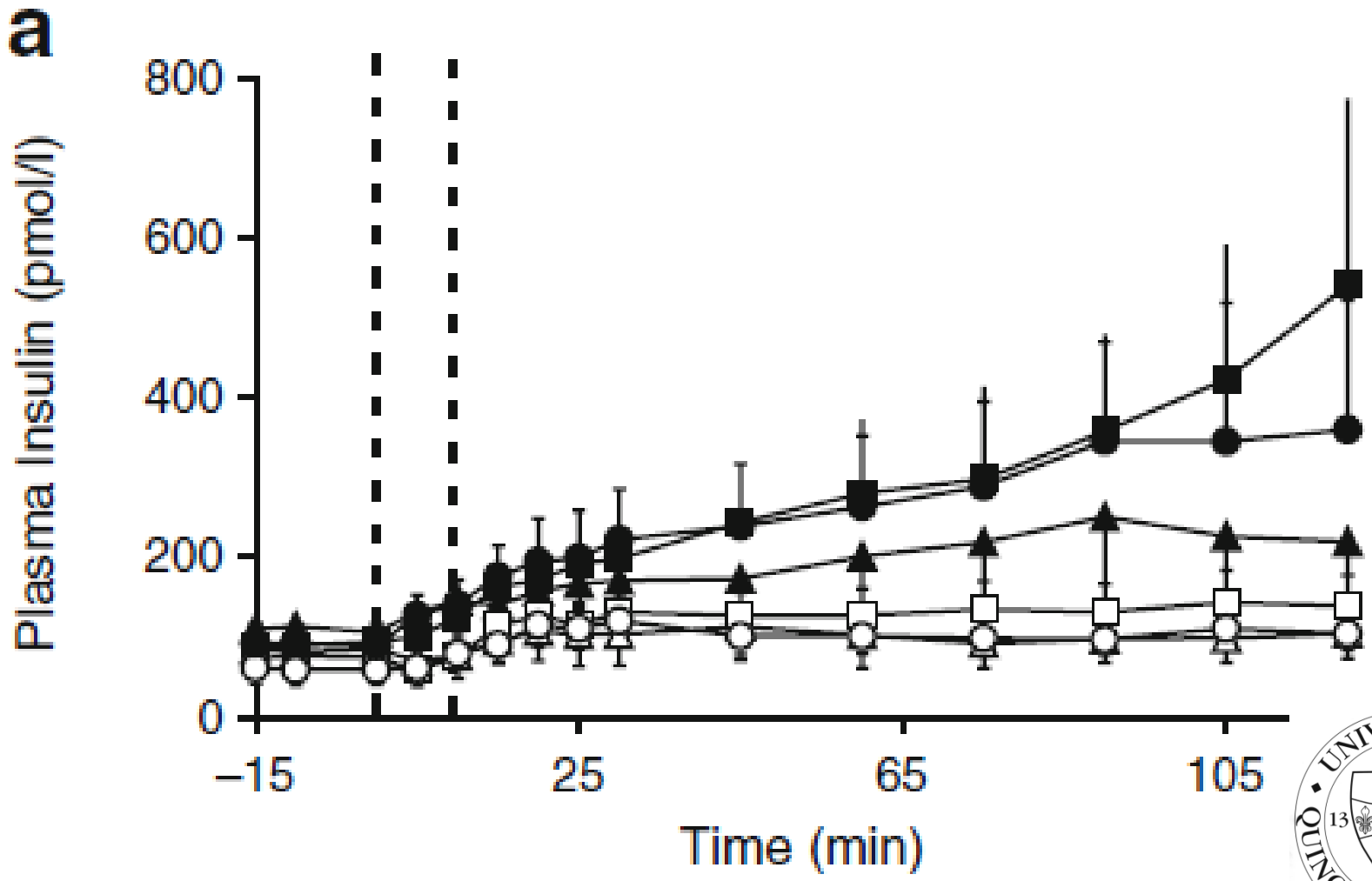
A GLP-1- és a GIP-rezisztencia áttörése

Vércukorprofil 4 hetes inzulinkezelés előtt (üres kör) és után (fekete kör)



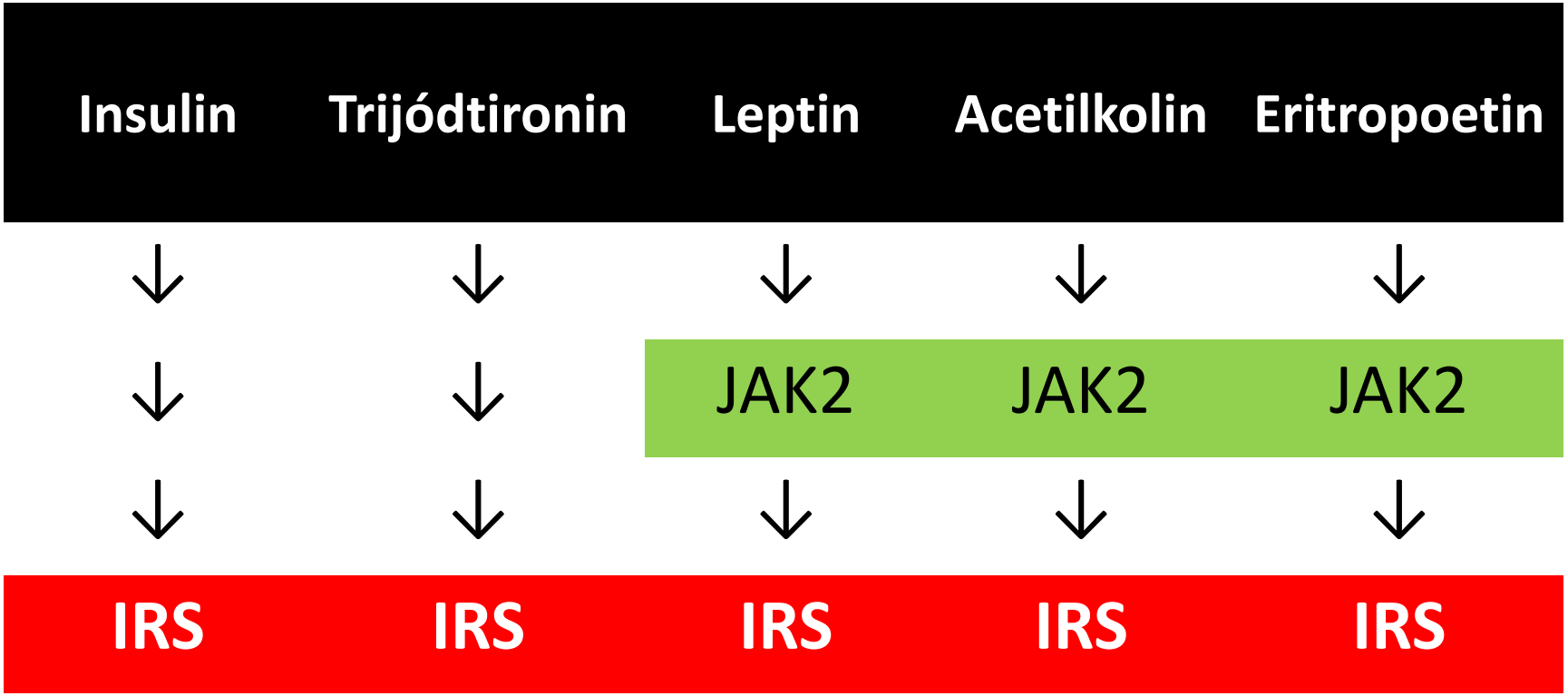
Diabetologia 2009,52,199-207

A plazmainzulin GLP-1 hatására a 4-hetes euglikémia előtt (üres), után (tele négyzet), GIP hatására (üres és tele kör) és fizsó hatására (üres és tele háromszög) T2DM-ben



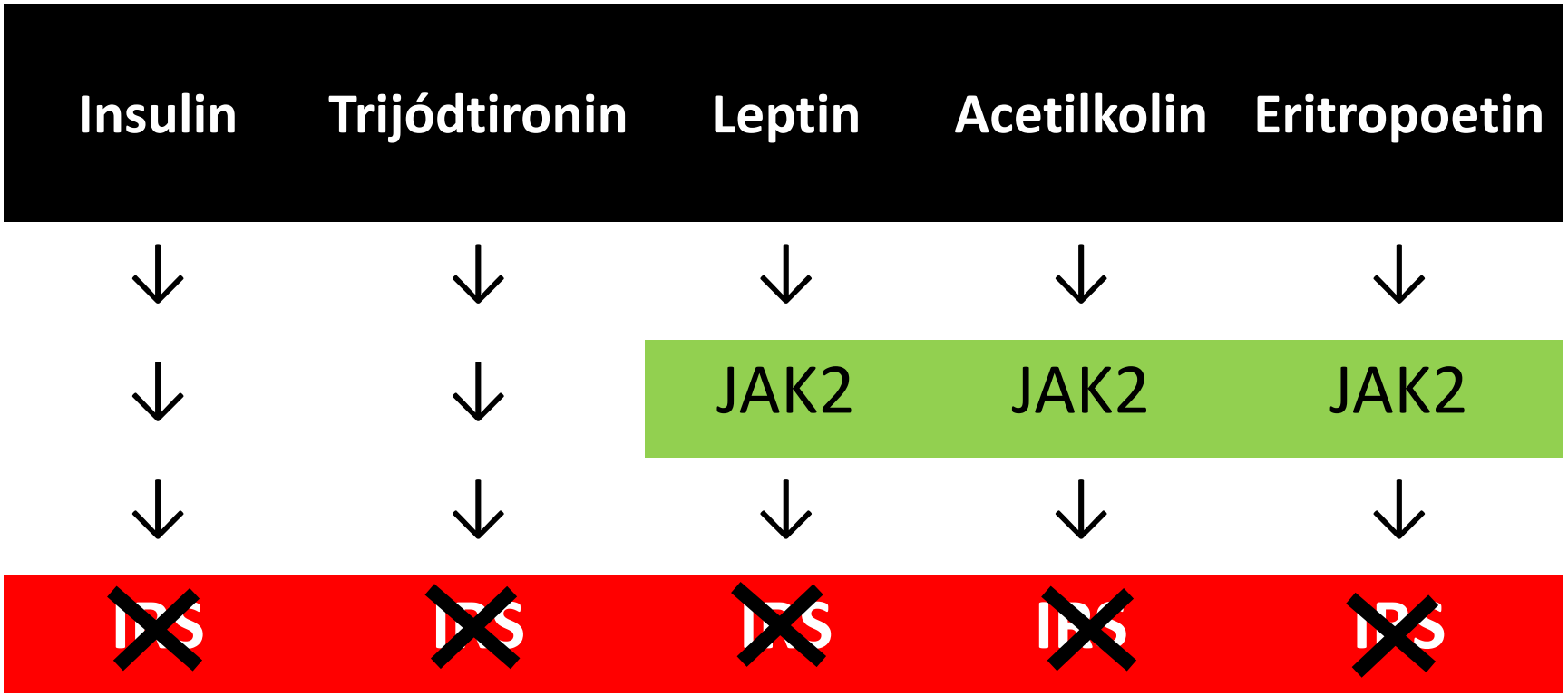
Diabetologia 2009,52,199-207

Néhány hormon közös jelátvitele



Antioxidants 2023, 12, 75. https://doi.org/10.3390/ antiox12010075

Oxidatív stress okozta krónikus, multihormonális rezisztencia



Antioxidants 2023, 12, 75. https://doi.org/10.3390/ antiox12010075

Az idült multihormonális rezisztencia kialakulása

Diabetes mellitus, dyslipidaemia and inflammation

↓

Intracellular oxidative stress

↓

Overproduction of hydroxyl free radical

↓

Accumulation of abnormal ortho- and meta-tyrosine

↓

Chronic and end-stage kidney disease, dialysis therapy

↓

Reduced kidney function and oxidative stress

↓

Overproduction of hydroxyl radical and reduced elimination of abnormal ortho- and meta-tyrosine

↓

Accumulation of abnormal ortho- and meta-tyrosine

↓

Incorporation of ortho- and meta-tyrosine in the signalling proteins

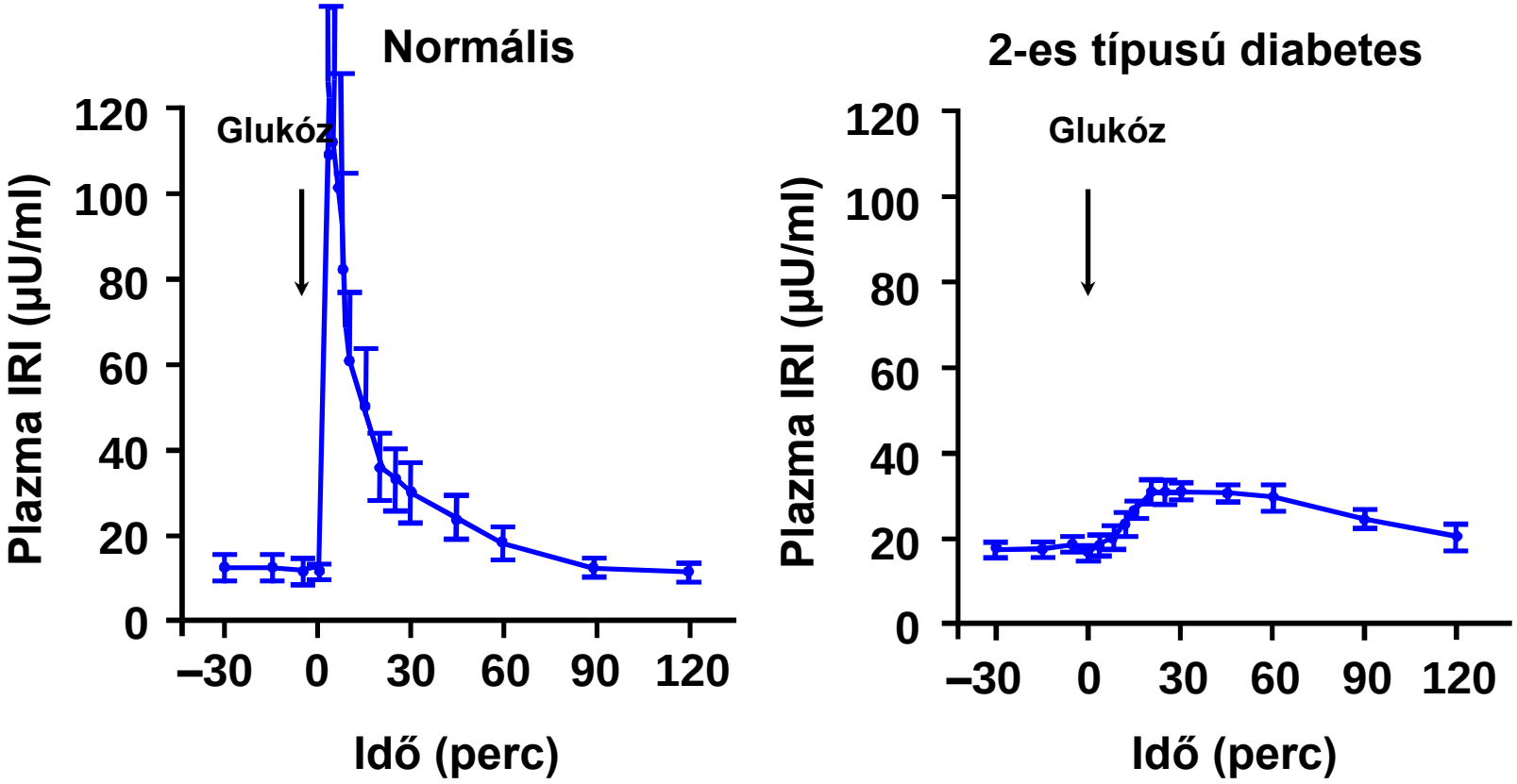
↓

Resistance to insulin, triiodothyronine, leptin, acetylcholine, erythropoietin, GLP-1

Antioxidants 2023, 12, 75. https://doi.org/10.3390/ antiox12010075

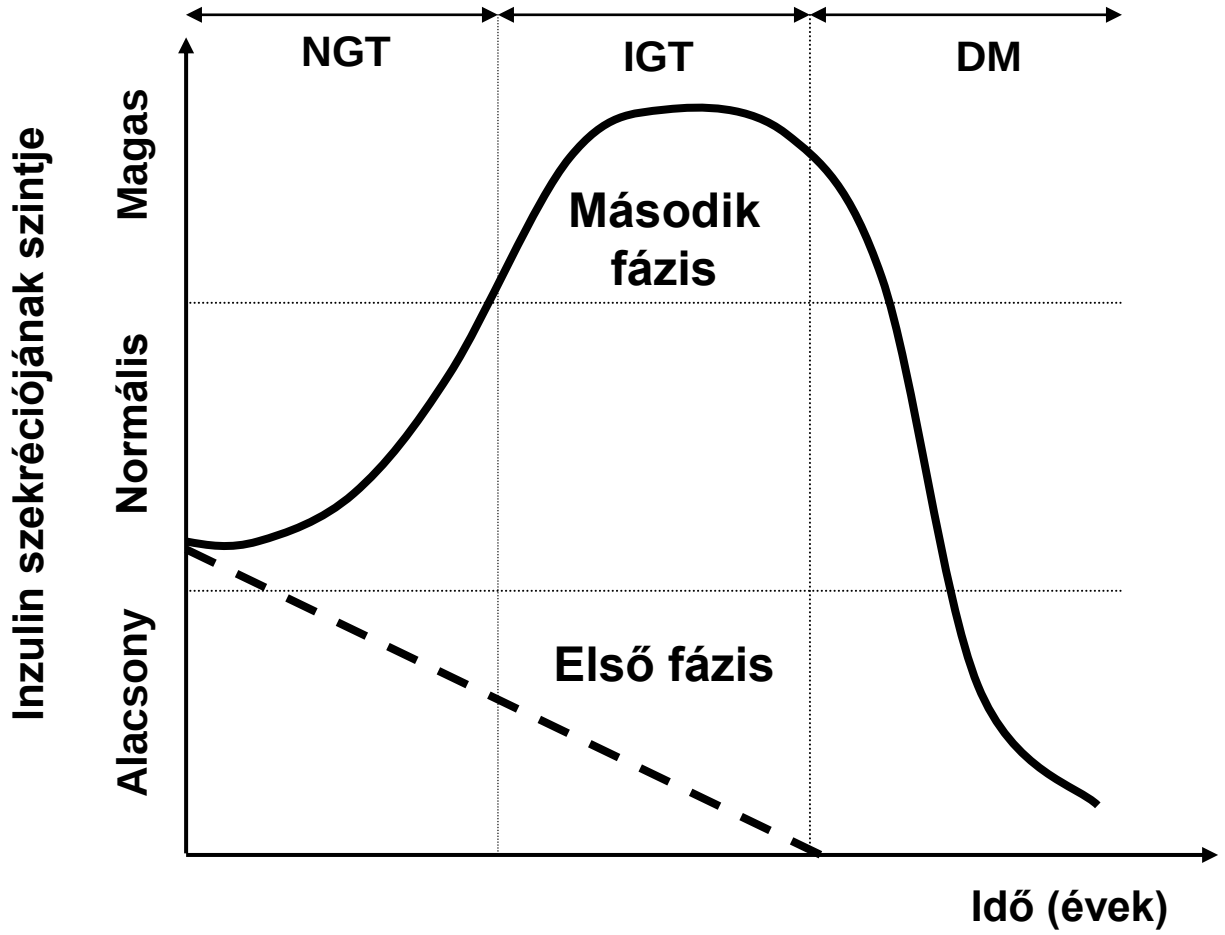
Az inzulinszekréció károsodása

2-es típusú diabetesben a korai inzulinválasz elvész



Ward WK et al. Diabetes Care 1984;7:491–502

Az inzulin szekréciójának változása



USE OF GLUCOSE-LOWERING MEDICATIONS IN THE MANAGEMENT OF TYPE 2 DIABETES

HEALTHY LIFESTYLE BEHAVIOURS; DIABETES SELF-MANAGEMENT EDUCATION AND SUPPORT (DSMES); SOCIAL DETERMINANTS OF HEALTH (SDOH)

TO AVOID THERAPEUTIC INERTIA REASSESS AND MODIFY TREATMENT REGULARLY (3–6 MONTHS)

Goal: Cardiorenal Risk Reduction in High-Risk Patients with Type 2 Diabetes (In addition to comprehensive CV risk management)*

+ASCVD†

Defined differently across CVOTs but all included individuals with established CVD (e.g. MI, stroke, any revascularisation procedure). Variably included: conditions such as transient ischaemic attack, unstable angina, amputation, symptomatic or asymptomatic coronary artery disease.

+Indicators of high risk

While definitions vary, most comprise ≥ 55 years of age with two or more additional risk factors (including obesity, hypertension, smoking dyslipidaemia or albuminuria)

+ASCVD/Indicators of High Risk

GLP-1 RA# with proven CVD benefit **EITHER/OR** SGLT2i§ with proven CVD benefit

If HbA_{1c} above target

- For patients on a GLP-1 RA consider adding SGLT2i with proven CVD benefit or vice versa
- TZD^

+HF

Current or prior symptoms of HF with documented HFrEF or HFpEF

+HF

SGLT2i§ with proven HF benefit in this population

+CKD

eGFR < 60 ml/min per 1.73 m^2 OR albuminuria (ACR ≥ 3.0 mg/mmol (30mg/g)). These measurements may vary over time; thus, a repeat measure is required to document CKD.

+CKD (on maximally tolerated dose of ACEi/ARB)

PREFERABLY

SGLT2i§ with primary evidence of reducing CKD progression

Use SGLT2i in people with an eGFR ≥ 20 ml/min per 1.73 m^2; once initiated should be continued until dialysis or transplantation

— — — — — OR — — — — —

GLP-1 RA with proven CVD benefit if SGLT2i not tolerated or contraindicated

If HbA_{1c} above target, for patients on SGLT2i, consider incorporating a GLP-1 RA or vice versa

If additional cardiorenal risk reduction or glycaemic lowering needed

Goal: Achievement and Maintenance of Glycaemic and Weight Management Goals

Glycaemic Management: Choose approaches that provide the efficacy to achieve goals:

Metformin OR Agent(s) including COMBINATION therapy that provide adequate EFFICACY to achieve and maintain treatment goals

Consider avoidance of hypoglycaemia a priority in high-risk individuals

In general, higher efficacy approaches have greater likelihood of achieving glycaemic goals

Efficacy for glucose lowering

Very High:
Dulaglutide (high dose), Semaglutide, Tirzepatide
Insulin
Combination Oral, Combination Injectable (GLP-1 RA/Insulin)

High:
GLP-1 RA (not listed above), Metformin, SGLT2i, Sulfonylurea, TZD

Intermediate:
DPP-4i

Achievement and Maintenance of Weight Management Goals:

Set individualised weight management goals

- General lifestyle advice: medical nutrition therapy/eating patterns/ physical activity
- Intensive evidence-based structured weight management programme
- Consider medication for weight loss
- Consider metabolic surgery

When choosing glucose-lowering therapies:
Consider regimen with high-to-very-high dual glucose and weight efficacy

Efficacy for weight loss

Very High:
Semaglutide, Tirzepatide

High:
Dulaglutide, Liraglutide

Intermediate:
GLP-1RA (not listed above), SGLT2i

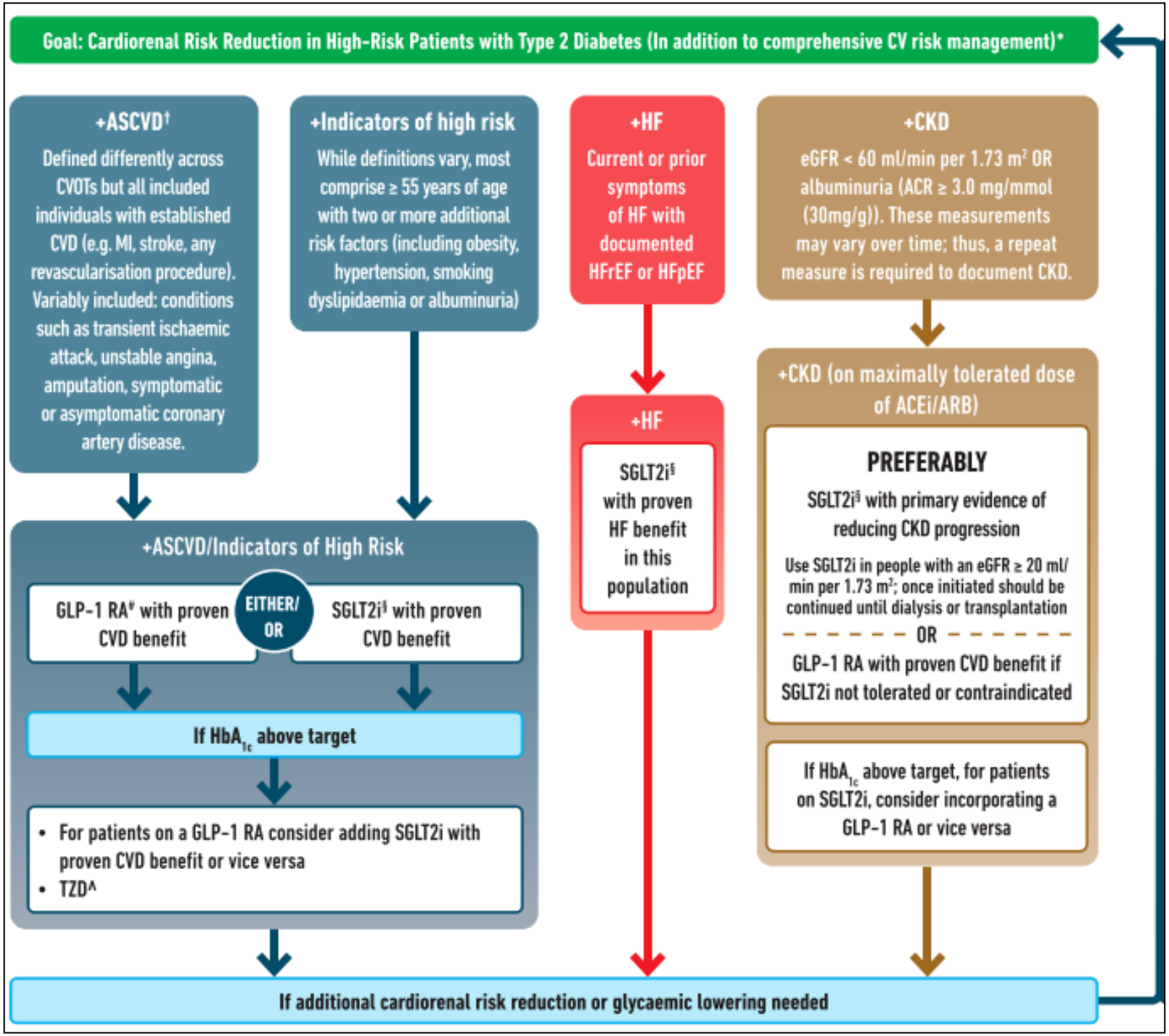
Neutral:
DPP-4i, Metformin

If HbA_{1c} above target

Identify barriers to goals:

- Consider DSMES referral to support self-efficacy in achievement of goals
- Consider technology (e.g. diagnostic CGM) to identify therapeutic gaps and tailor therapy
- Identify and address SDOH that impact on achievement of goals

* In people with HF, CKD, established CVD or multiple risk factors for CVD, the decision to use a GLP-1 RA or SGLT2i with proven benefit should be independent of background use of metformin; † A strong recommendation is warranted for people with CVD and a weaker recommendation for those with indicators of high CV risk. Moreover, a higher absolute risk reduction and thus lower numbers needed to treat are seen at higher levels of baseline risk and should be factored into the shared decision-making process. See text for details; ^ Low-dose TZD may be better tolerated and similarly effective; § For SGLT2i, CV/renal outcomes trials demonstrate their efficacy in reducing the risk of composite MACE, CV death, all-cause mortality, MI, HHF and renal outcomes in individuals with T2D with established/high risk of CVD; # For GLP-1 RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke and renal endpoints in individuals with T2D with established/high risk of CVD.


Goal: Cardiorenal Risk Reduction in High-Risk Patients with Type 2 Diabetes (In addition to comprehensive CV risk management)*
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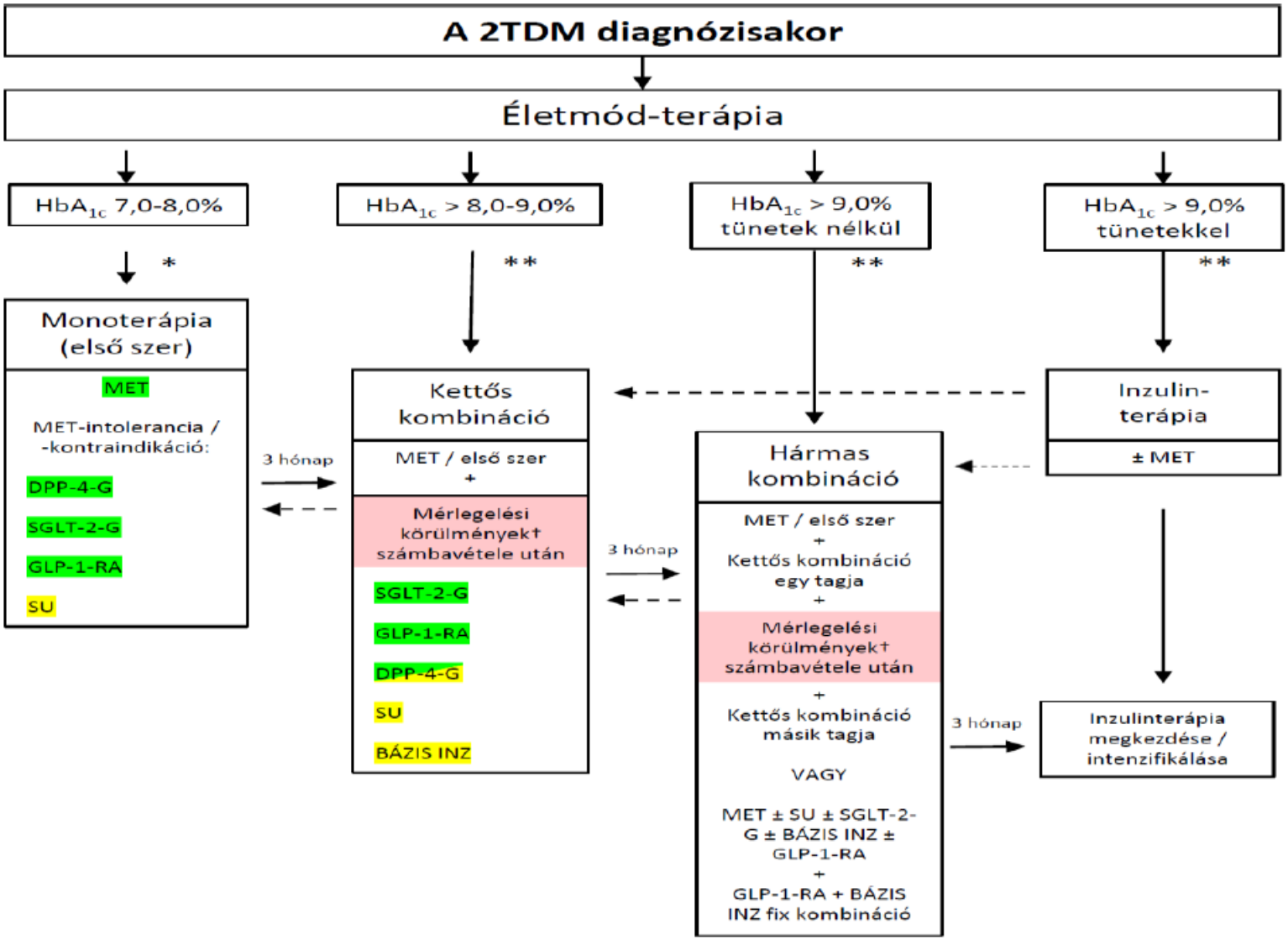
Defined differently across CVOTs but all included individuals with established CVD (e.g. MI, stroke, any revascularisation procedure). Variably included: conditions such as transient ischaemic attack, unstable angina, amputation, symptomatic or asymptomatic coronary artery disease.
+Indicators of high risk
While definitions vary, most comprise ≥ 55 years of age with two or more additional risk factors (including obesity, hypertension, smoking dyslipidaemia or albuminuria)
+HF
Current or prior symptoms of HF with documented HFrEF or HFpEF
+CKD
eGFR < 60 ml/min per 1.73 m² OR albuminuria (ACR ≥ 3.0 mg/mmol (30mg/g)). These measurements may vary over time; thus, a repeat measure is required to document CKD.
+ASCVD/Indicators of High Risk
GLP-1 RA¥ with proven CVD benefit
EITHER/ OR
SGLT2i§ with proven CVD benefit
If HbA1c above target
• For patients on a GLP-1 RA consider adding SGLT2i with proven CVD benefit or vice versa
• TZD^
+HF
SGLT2i§ with proven HF benefit in this population
+CKD (on maximally tolerated dose of ACEi/ARB)
PREFERABLY
SGLT2i§ with primary evidence of reducing CKD progression
Use SGLT2i in people with an eGFR ≥ 20 ml/min per 1.73 m²; once initiated should be continued until dialysis or transplantation
OR
GLP-1 RA with proven CVD benefit if SGLT2i not tolerated or contraindicated
If HbA1c above target, for patients on SGLT2i, consider incorporating a GLP-1 RA or vice versa
If additional cardiorenal risk reduction or glycaemic lowering needed

2. ábra. Szakmailag megalapozott terápiás lépések az újonnan felismert 2-es típusú diabetes mellitus (2TDM) kezelésekor



Hazai adatok az atherosclerosisról 2-es típusú diabetesben

Kiss *et al. Cardiovasc Diabetol (2018) 17:107*
https://doi.org/10.1186/s12933-018-0751-7

Cardiovascular Diabetology

ORIGINAL INVESTIGATION

Open Access

Dissimilar impact of type 2 diabetes on cardiovascular outcomes according to age categories: a nationwide population study from Hungary

Zoltán Kiss[1], György Rokszin[2], Zsolt Abonyi-Tóth[2,3], György Jermendy[4], Péter Kempler[5], Dániel Aradi[6] and István Wittmann[1*]

T2DM vs Control	Number of population			Number of events			Hazard ratio (95% CI)	p value
Mortality	**T2DM**		**Control**	**T2DM**		**Control**		
71 and over	30 082	vs.	60 164	6 753	vs.	9 875	1.29(1.24-1.34)	p<0.0001
61-70	42 243	vs.	84 486	3 571	vs.	5 716	1.20(1.14-1.26)	p<0.0001
51-60	46 659	vs.	93 318	2 379	vs.	3 699	1.22(1.14-1.30)	p<0.0001
41-50	21 827	vs.	43 654	621	vs.	783	1.48(1.30-1.69)	p<0.0001
31-40	8 621	vs.	17 242	85	vs.	70	1.98(1.34-2.92)	p=0.0002
19-30	2 852	vs.	5 704	6	vs.	10	1.20(0.44-3.30)	p=0.725
Total	152 678	vs.	305 356	13 417	vs.	20 154	1.26(1.22-1.29)	p<0.0001
Interaction								p=0.01
Myocardial Infarction	**T2DM**		**Control**	**T2DM**		**Control**		
71 and over	30 082	vs.	60 164	1 041	vs.	872	1.78(1.59-2.00)	p<0.0001
61-70	42 243	vs.	84 486	948	vs.	763	1.57(1.39-1.79)	p<0.0001
51-60	46 659	vs.	93 318	922	vs.	628	1.85(1.62-2.11)	p<0.0001
41-50	21 827	vs.	43 654	360	vs.	162	2.76(2.16-3.52)	p<0.0001
31-40	8 621	vs.	17 242	55	vs.	16	3.50(1.70-7.23)	p=0.0002
19-30	2 852	vs.	5 704	4	vs.	1	8.02(0.90-71.71)	p=0.0627
Total	152 678	vs.	305 356	3 330	vs.	2 442	1.81(1.69-1.94)	p<0.0001
Interaction								p=0.003
Stroke	**T2DM**		**Control**	**T2DM**		**Control**		
71 and over	30 082	vs.	60 164	3 326	vs.	4 220	1.31(1.24-1.39)	p<0.0001
61-70	42 243	vs.	84 486	2 707	vs.	3 114	1.30(1.21-1.39)	p<0.0001
51-60	46 659	vs.	93 318	1 958	vs.	1 799	1.62(1.49-1.76)	p<0.0001
41-50	21 827	vs.	43 654	440	vs.	319	2.12(1.77-2.55)	p<0.0001
31-40	8 621	vs.	17 242	68	vs.	21	4.64(2.55-8.45)	p<0.0001
19-30	2 852	vs.	5 704	3	vs.	2	3.00(0.50-7.96)	p=0.229
Total	152 678	vs.	305 356	8 502	vs.	9 475	1.40(1.35-1.46)	p<0.0001
Interaction								p<0.0001

Fig. 2 Adjusted hazard ratios for all-cause mortality, myocardial infarction and stroke according age cohorts comparing T2DM with matched control. Age-dependent interactions: mortality: $p_{interaction} = 0.01$, myocardial infarction: $p_{interaction} = 0.003$ and stroke: $p_{interaction} < 0.0001$

Young-onset T2DM

Diabetes & Metabolism 48 (2022) 101389

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Original article

Prevalence and risk of diabetic complications in young-onset versus late-onset type 2 diabetes mellitus

Yongin Cho[a,1], Hye-Sun Park[b,1], Byung Wook Huh[c], Seong Ha Seo[a], Da Hea Seo[a], Seong Hee Ahn[a], Seongbin Hong[a], Young Ju Suh[d], So Hun Kim[a,*]

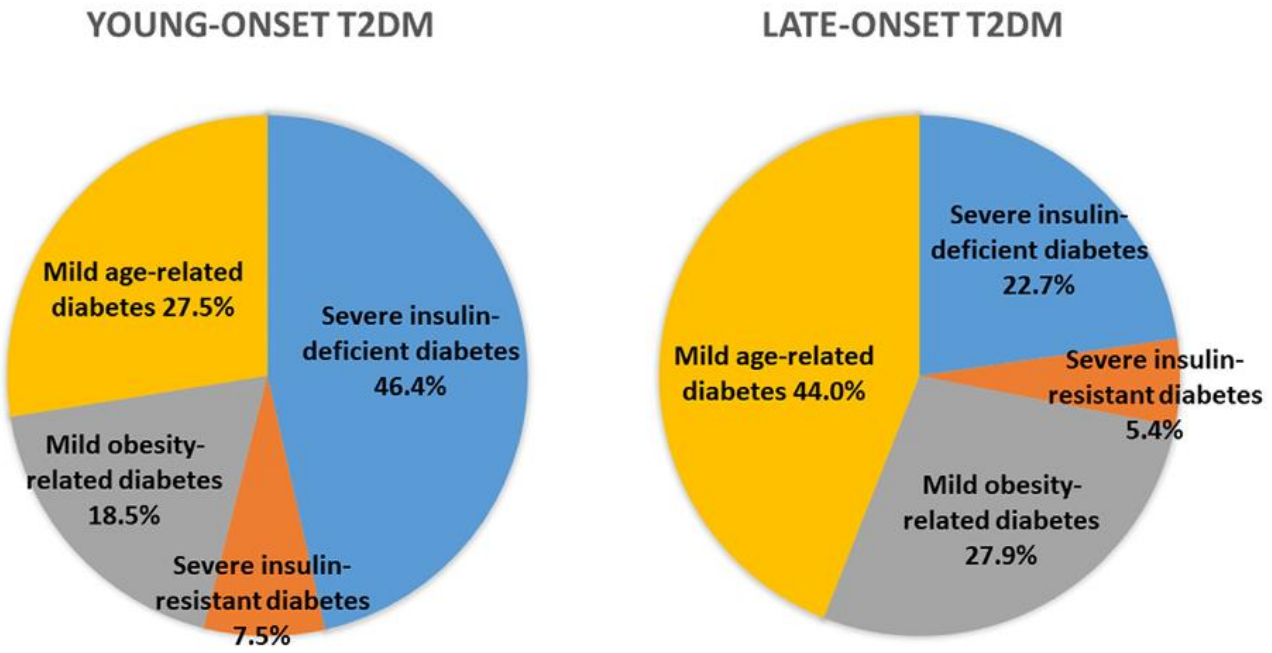

Fig. 2. Distribution of people with YOD and LOD according to the cluster classification.

YOD, young-onset type 2 diabetes mellitus; LOD, late-onset type 2 diabetes mellitus; SIDD, severe insulin-deficient diabetes; SIRD, severe insulin-resistant diabetes; MOD, mild obesity-related diabetes; MARD, mild age-related diabetes.

PLOS MEDICINE

RESEARCH ARTICLE

Age at diagnosis, glycemic trajectories, and responses to oral glucose-lowering drugs in type 2 diabetes in Hong Kong: A population-based observational study

Calvin Ke[1,2], Thérèse A. Stukel[3,4], Baiju R. Shah[2,3,4,5], Eric Lau[1,6], Ronald C. Ma[1,7], Wing-Yee So[1], Alice P. Kong[1,7], Elaine Chow[1], Juliana C. N. Chan[1,6,7]*, Andrea Luk[1,6,7]

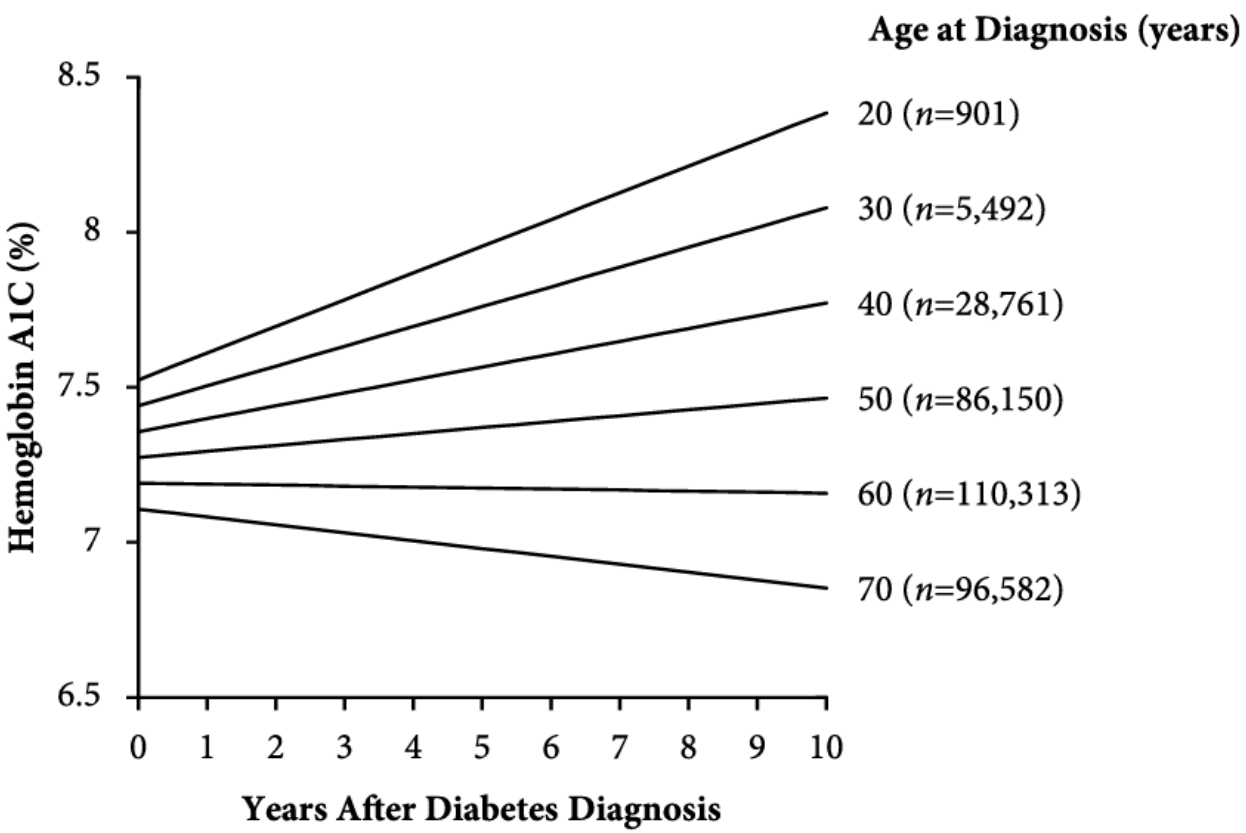

Fig 2. Glycemic deterioration during the first decade after type 2 diabetes diagnosis. Results are stratified by age at

Life expectancy associated with different ages at diagnosis of type 2 diabetes in high-income countries: 23 million person-years of observation

*Emerging Risk Factors Collaboration**

Summary

Background The prevalence of type 2 diabetes is increasing rapidly, particularly among younger age groups. Estimates suggest that people with diabetes die, on average, 6 years earlier than people without diabetes. We aimed to provide reliable estimates of the associations between age at diagnosis of diabetes and all-cause mortality, cause-specific mortality, and reductions in life expectancy.

Lancet Diabetes Endocrinol 2023; 11: 731–42

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https://doi.org/10.1016/

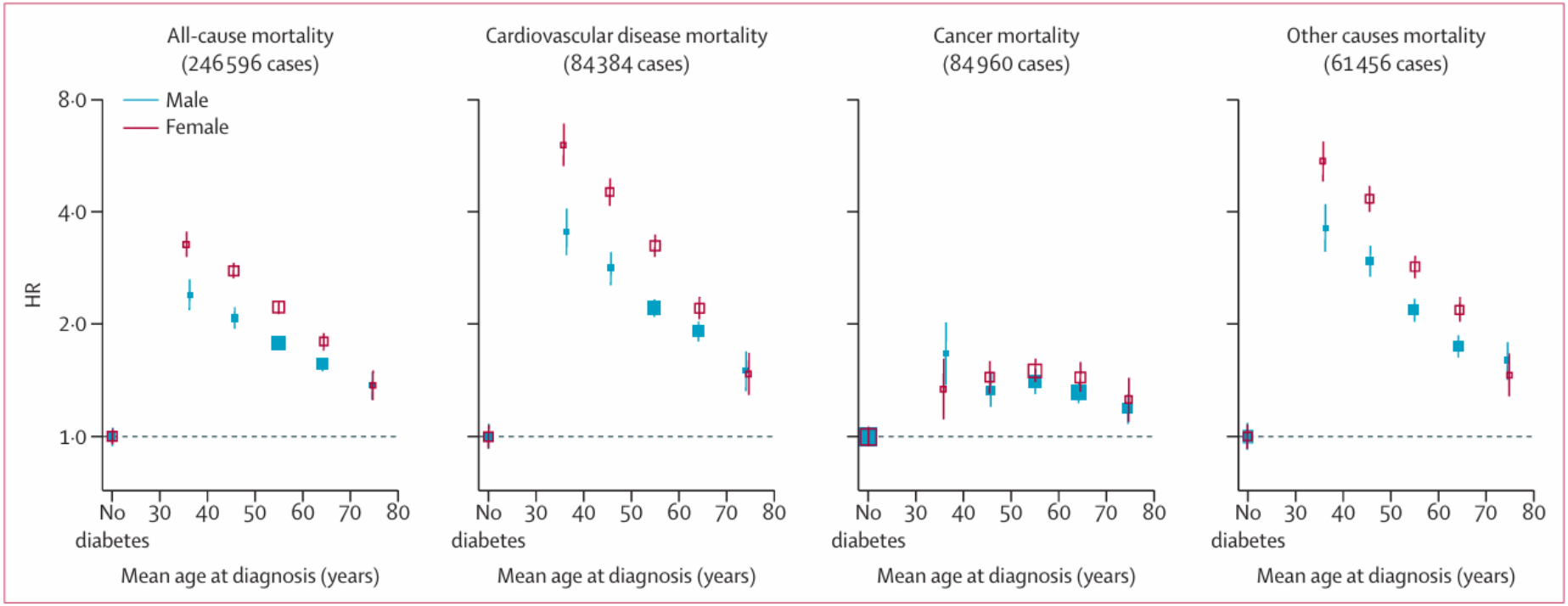
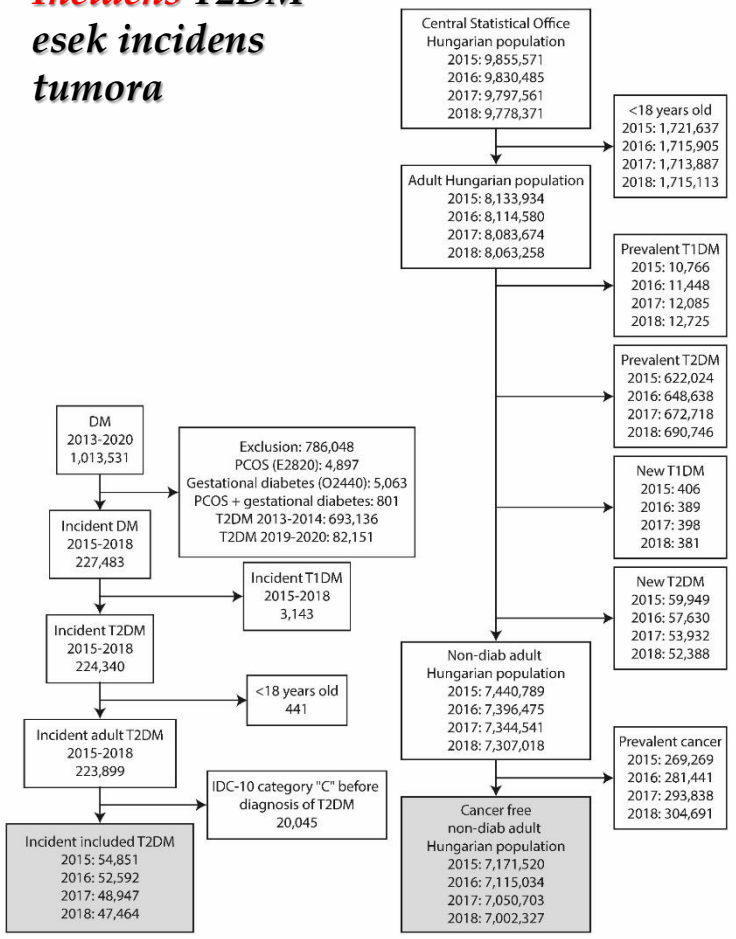


Figure 1: **Sex-specific HRs for all-cause and cause-specific mortality according to age at diagnosis of type 2 diabetes**

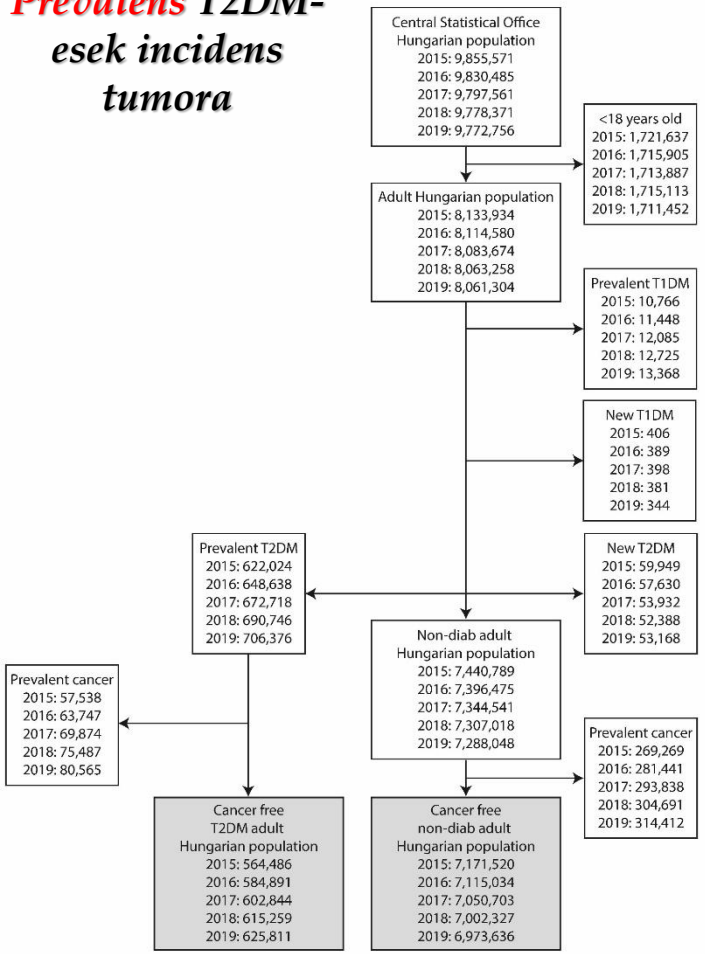
The mean age at diagnosis for the categories 30 to <40 years, 40 to <50 years, 50 to <60 years, 60 to <70 years and ≥70 years is plotted on the x axis. HRs are adjusted for age, and the reference (1·0) is people without diabetes. Studies with fewer than ten events of any outcome were excluded from the analysis of that outcome. The sizes of the boxes are proportional to the inverse of the variance of the log-transformed HRs. Vertical lines represent 95% CIs. HR=hazard ratio.

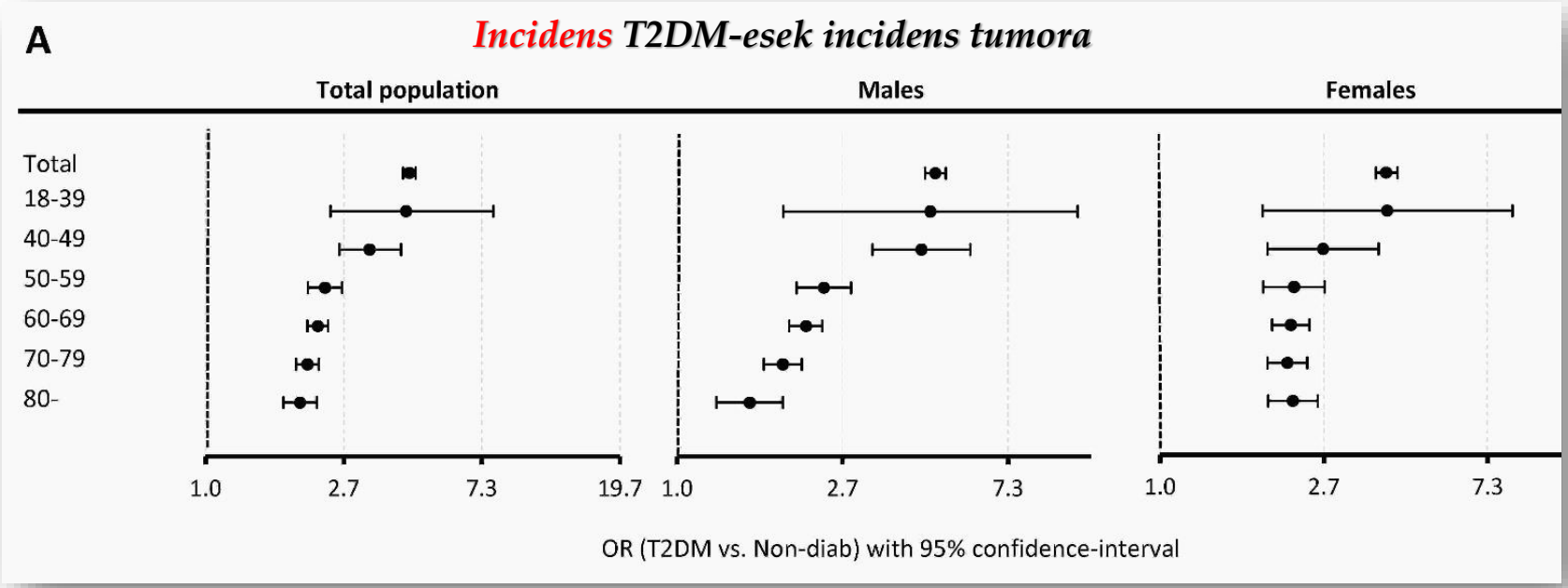
Diabetes és tumor
Hazai új eredményeink

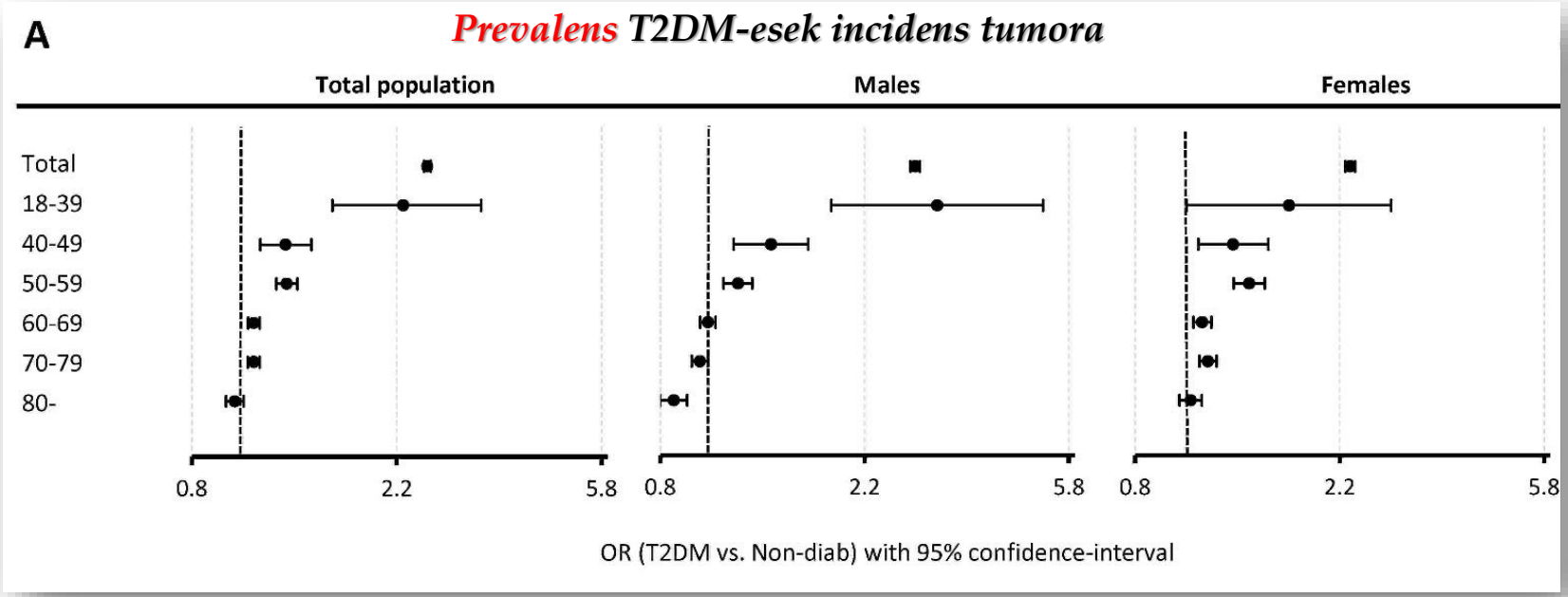
Incidens T2DM-esek incidens tumora



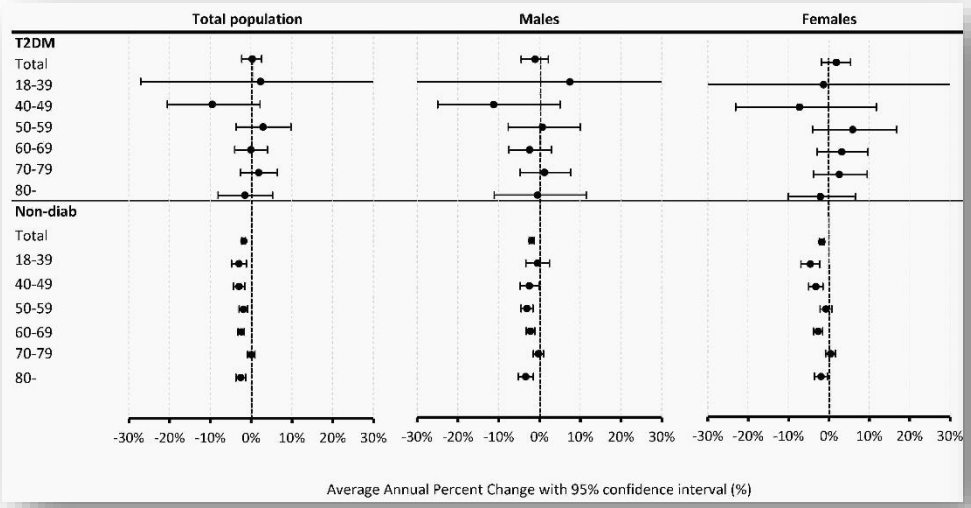
Prevalens T2DM-esek incidens tumora



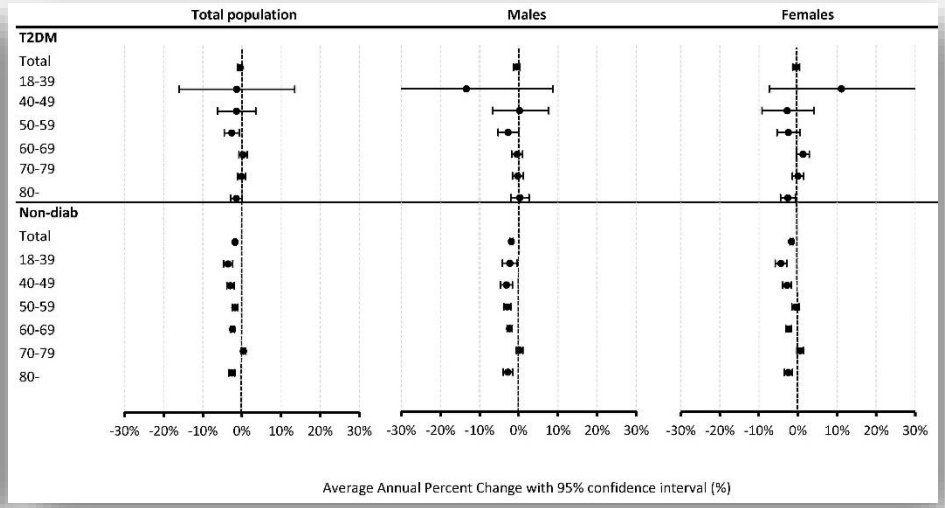

A
Incidens T2DM-esek incidens tumora
Total population
Males
Females
Total
18-39
40-49
50-59
60-69
70-79
80-
1.0
2.7
7.3
19.7
1.0
2.7
7.3
1.0
2.7
7.3
OR (T2DM vs. Non-diab) with 95% confidence-interval


A
Prevalens T2DM-esek incidens tumora
Total population
Males
Females
Total
18-39
40-49
50-59
60-69
70-79
80-
0.8
2.2
5.8
0.8
2.2
5.8
0.8
2.2
5.8
OR (T2DM vs. Non-diab) with 95% confidence-interval

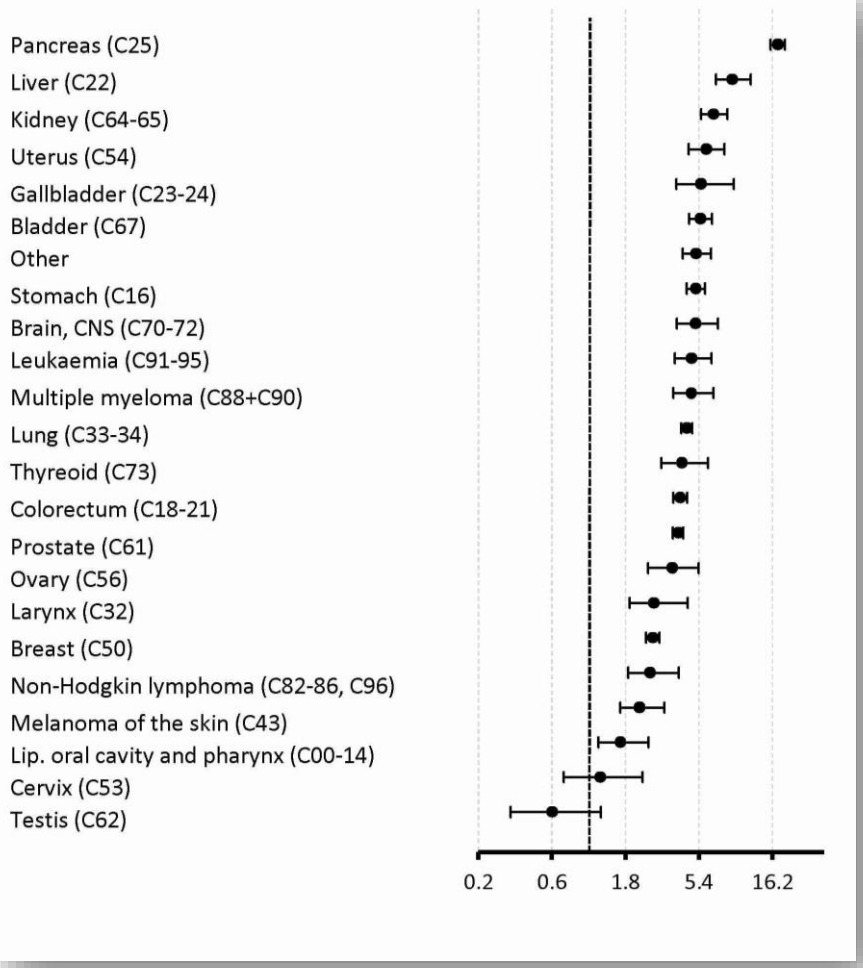
Incidens T2DM-esek incidens tumorának éves változása



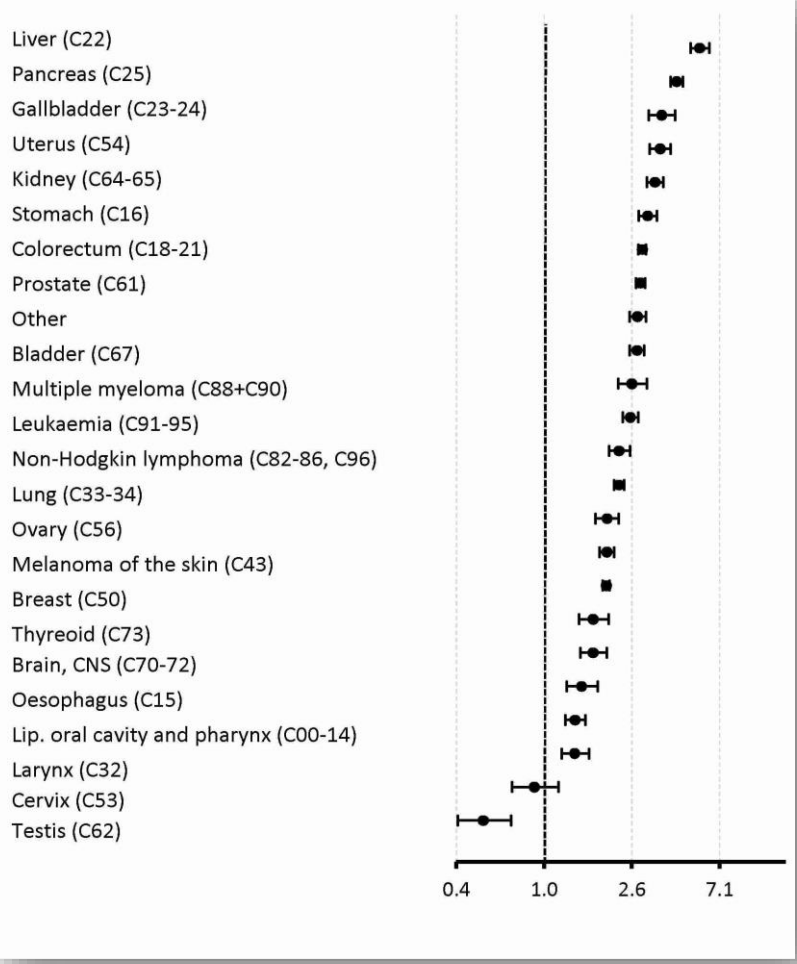
Prevalens T2DM-esek incidens tumorának éves változása



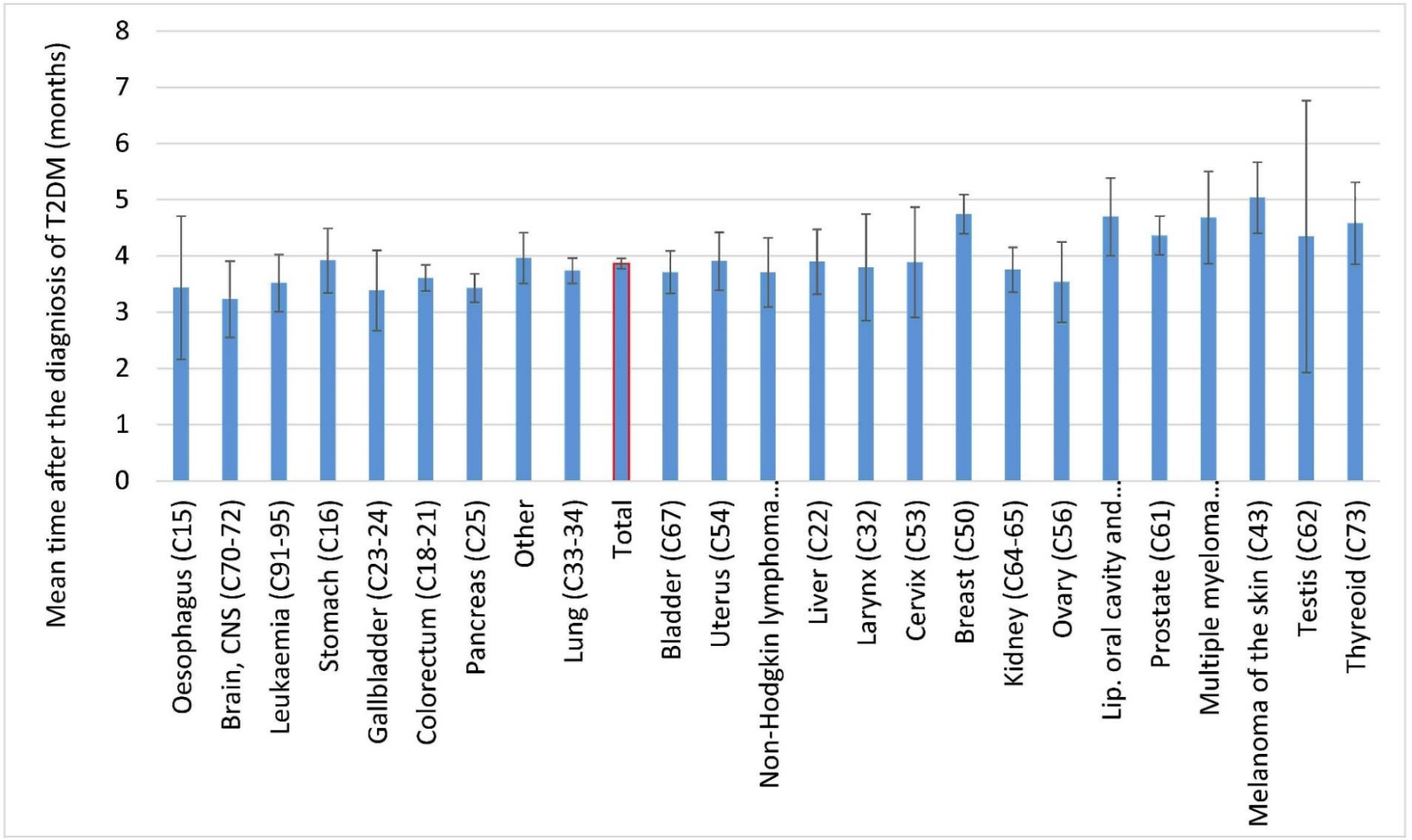
Incidens T2DM-esek incidens tumora



Prevalens T2DM-esek incidens tumora



Incidens T2DM-esek incidens tumorának átlagos felfedezési ideje



Diabeteszes vesebetegség

IJPH | SSPH+ SWISS SCHOOL OF PUBLIC HEALTH

International Journal of Public Health
ORIGINAL ARTICLE
published: 30 March 2023
doi: 10.3389/ijph.2023.1605635

Check for updates

Prevalence, Cardiometabolic Comorbidities and Reporting of Chronic Kidney Disease; A Hungarian Cohort Analysis

Antal Zemplényi[1], *Eszter Sághy*[1], *Anna Kónyi*[1], *Lilla Szabó*[2], *István Wittmann*[3]* *and Boglárka Laczy*[3]

[1]*Center for Health Technology Assessment and Pharmacoeconomic Research, Faculty of Pharmacy, University of Pécs, Pécs, Hungary,* [2]*AstraZeneca Ltd., Budapest, Hungary,* [3]*Second Department of Medicine and Nephrology-Diabetes Center, University of Pécs Medical School, Pécs, Hungary*

A laboratóriumi értékekkel igazolt CKD esetek BNO kódolási aránya az orvosi dokumentációban

Csak 28,6% a felismert CKD-s

	N18, N19+	N18, N19–	Total
CKD confirmed by laboratory test+	3,893	9,703	13,596
CKD not confirmed by laboratory test–	2,374	81,316	83,690
Total	6,267	91,019	97,286

Amennyiben a CKD-nak lejelentett, de laborral nem CKD-sokat is beleszámoljuk a CKD-s populációba, akkor **16,4%** a CKD előfordulása

IJPH | SSPH+ SWISS SCHOOL OF PUBLIC HEALTH

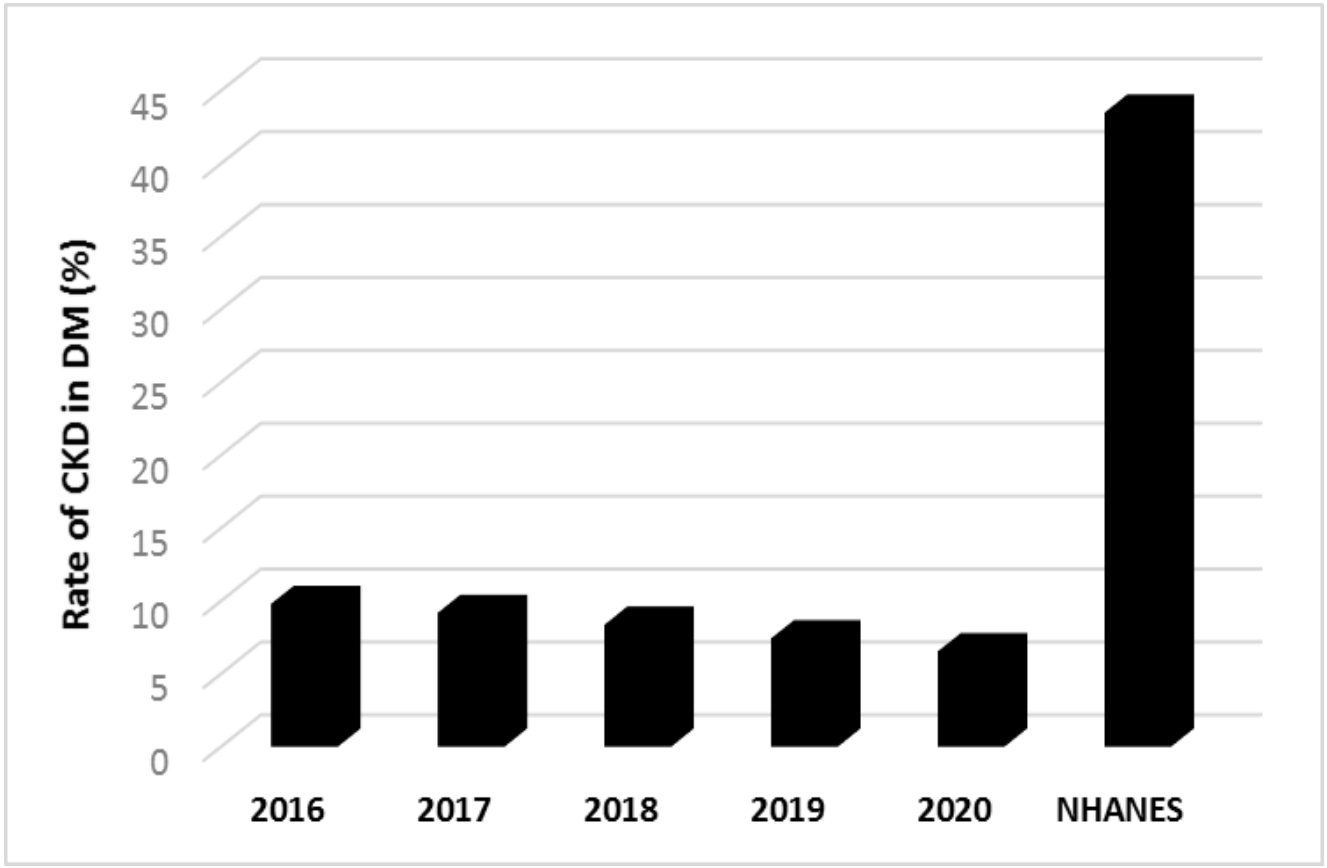
International Journal of Public Health
ORIGINAL ARTICLE
published: 29 August 2023
doi: 10.3389/ijph.2023.1606151

Check for updates

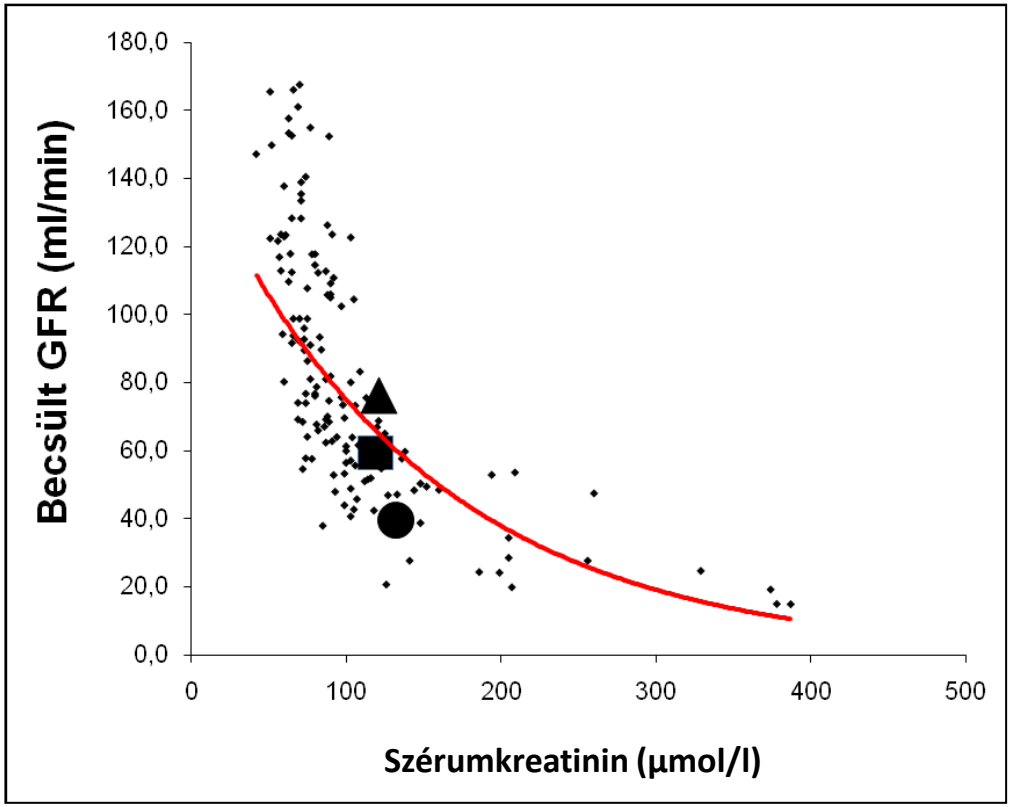
Deficiencies in the Recognition and Reporting of Chronic Kidney Disease in Patients With Type 2 Diabetes Mellitus; A Hungarian Nationwide Analysis

Erzsébet Ladányi[1], Balázs Salfer[2], József Balla[3], István Kárpáti[3], György Reusz[4], Lilla Szabó[2], Péter Andriska[5], László Németh[5], István Wittmann[6] and Boglárka Laczy[6]*

A 2-es típusú cukorbetegek körében lejelentett CKD-sok aránya hazánkban



Szérumkreatinin és becsült GFR (Cockroft-Gault, MDRD, CKD-EPI)



▲: Szérumkreatinin=**121** µmol/l, GFR=**76** ml/min;
■: Szérumkreatinin=**123** µmol/l, GFR=**56** ml/min;
●: Szérumkreatinin=**132** µmol/l, GFR=**39** ml/min.

Emberi Erőforrások Minisztériuma
EGÉSZSÉGÜGYI SZAKMAI KOLLÉGIUM

Egészségügyi szakmai irányelv–
„A felnőttkori idült vesebetegség diagnózisa és kezelése”

Típusa:	Klinikai egészségügyi szakmai irányelv
Azonosító:	002169
Megjelenés dátuma:	év. hónap. nap
(Közlönykiadó adja meg)	
Érvényesség időtartama:	megjelenést követő 3 évig érvényes
Kiadja:	Emberi Erőforrások Minisztériuma
Megjelenés helye	
Nyomtatott verzió:	Egészségügyi Közlöny
Elektronikus elérhetőség:	https://kollegium.aeek.hu

A CKD diagnózisa, stádium-megállapítása, aktivitása és prognózisa

Hosszabban, mint 3 hónapja fennálló

becsült GFR < 60 ml/perc/1,73m^2, vagy kóros albuminürítés, vagy kóros vizeletüledék, vagy kóros szövettan, vagy eltérés a vese képalkotóvizsgálatában, vagy ionzavar, vagy vese-transzplantáció az anamnézisben

↓

Ha a bGFR nem áll rendelkezésre, ennek meghatározása

↓

CKD stádiumának megállapítása

↓

CKD aktivitási jelként az albuminuria, vizeletüledék vizsgálata

↓

A prognózishoz a vese ultrahangos vizsgálata

Az SGLT-2-G és a GLP-1-RA-k alkalmazása CKD-ban szenvedő cukorbetegekben

CKD* kezelése RAAS-gátlóval

↓

A HbA_{1c}-től függetlenül SGLT-2-G** elkezdése

bGFR≥25 ml/perc/1,73m^2 esetén***

↓

A bGFR és a HbA_{1c} ellenőrzése

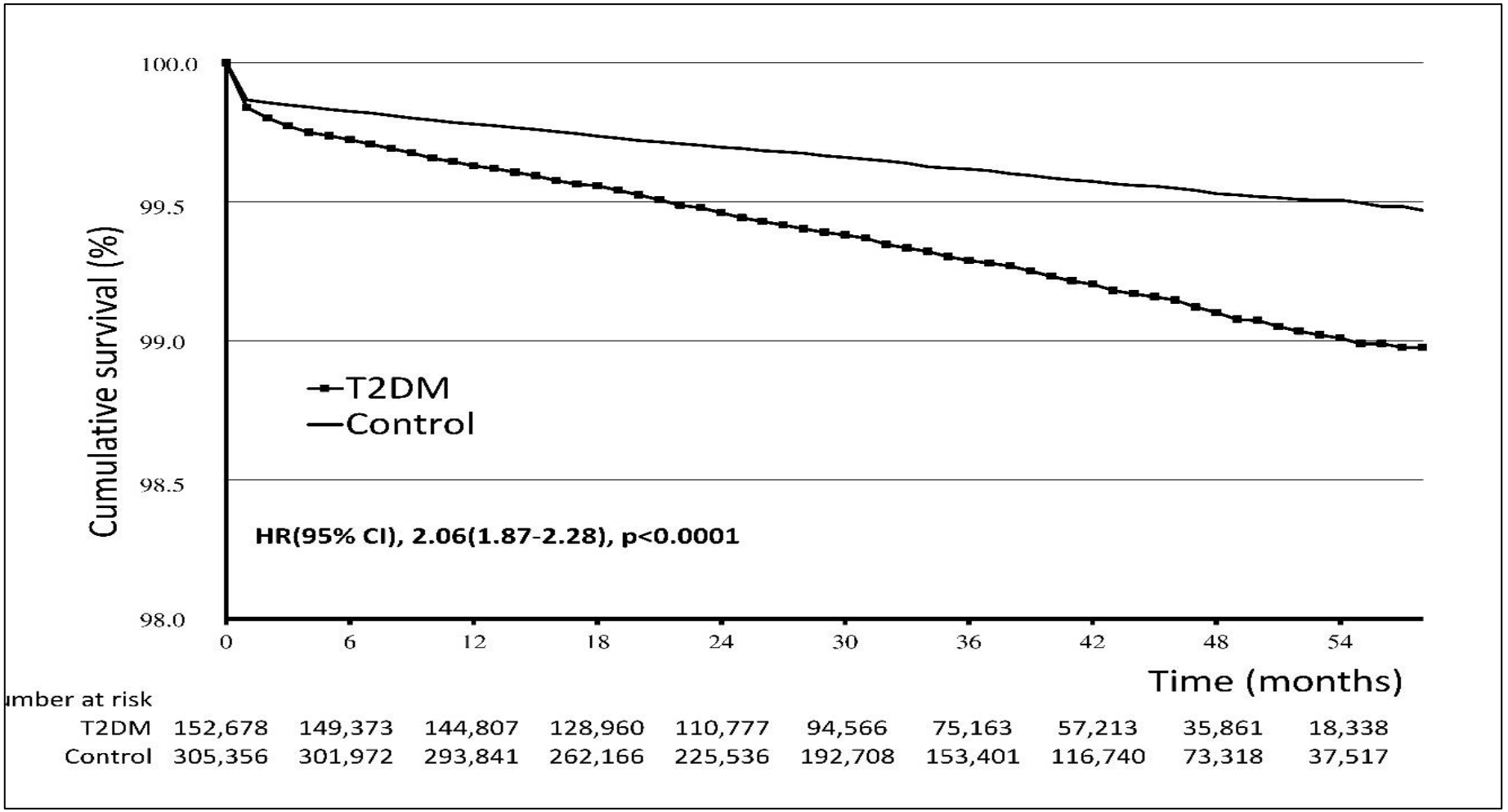
↓

Kontraindikáció és bGFR < 25ml/perc/1,73m^2 esetén SGLT-2-G elhagyása, elégtelen HbA_{1c}-válasz esetén GLP-1-RA-val történő kiegészítése

Szűrés diagnózis stádium	Kezelés	Orvos
CKD-szűrés[1] CKD-diagnózis[2]		Nefrológus, nem nefrológus, nem háziorvos szakorvos, háziorvos
CKD1[3]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, SGLT-2-gátlás*, antihipertenzívum, statin	Nefrológus, nem nefrológus, nem háziorvos szakorvos, háziorvos
CKD2[4]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, SGLT-2-gátlás*, antihipertenzívum, statin	Nefrológus, nem nefrológus, nem háziorvos szakorvos, háziorvos
CKD3[5]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, SGLT-2-gátlás*, antihipertenzívum, statin	Nefrológus, nem nefrológus, nem háziorvos szakorvos, háziorvos
CKD4[6]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, SGLT-2-gátlás*, antihipertenzívum, statin, kacs-diuretikum, ESA, MBD, alkalizálás	Nefrológus
CKD5[7]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, antihipertenzívum, statin, kacs-diuretikum, ESA, MBD, alkalizálás	Nefrológus
CKD5D[8]	Specifikus kezelés Nem specifikus kezelés: RAAS-gátlás, antihipertenzívum, statin, kacs-diuretikum, ESA, MBD, alkalizálás Vesepótló-kezelés	Nefrológus

T2DM – dialízis

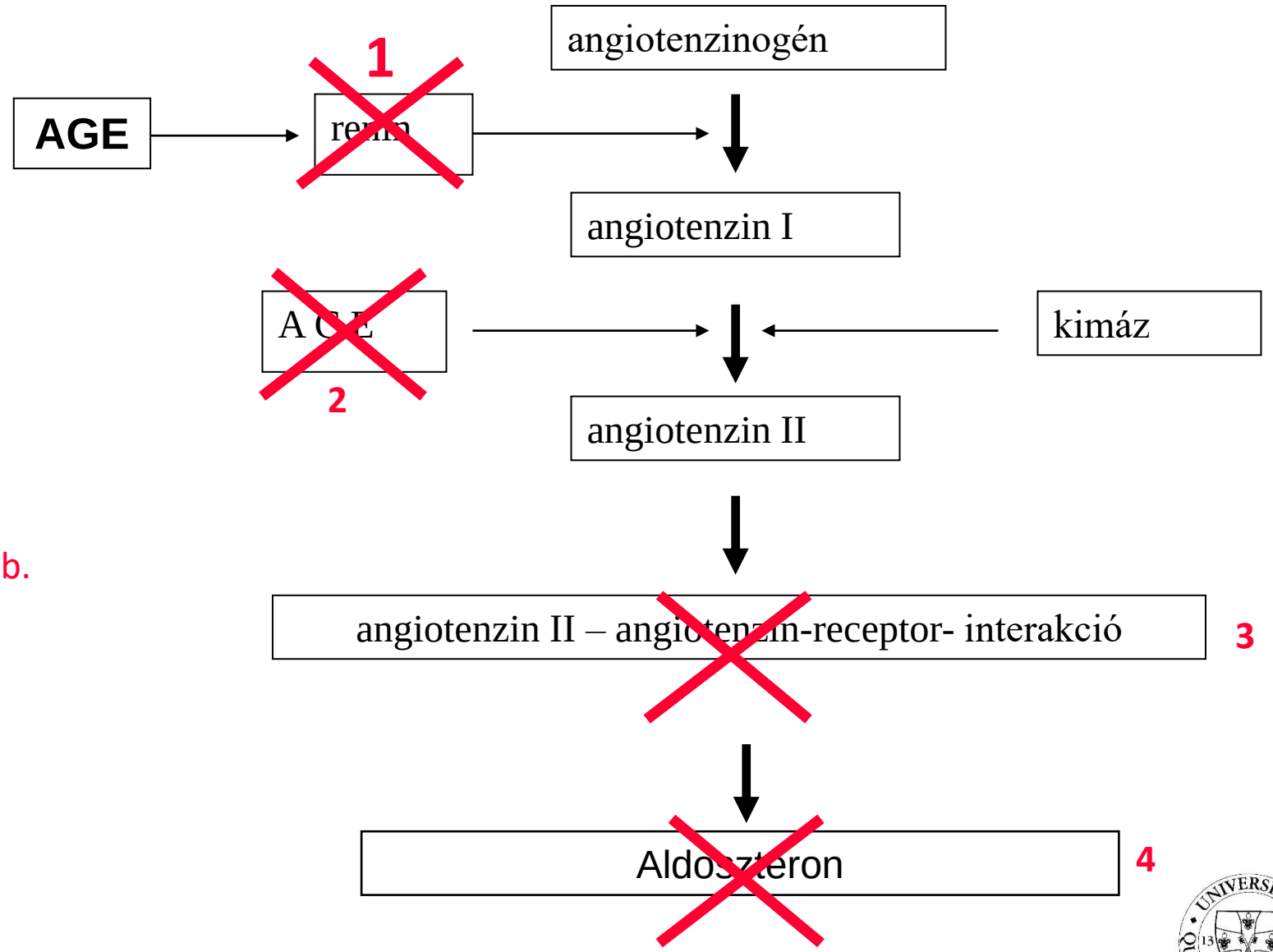
T2DM-ben nagyobb a dialízis kockázata, mint a nem cukorbetegek körében



umber at risk

T2DM	152,678	149,373	144,807	128,960	110,777	94,566	75,163	57,213	35,861	18,338
Control	305,356	301,972	293,841	262,166	225,536	192,708	153,401	116,740	73,318	37,517

A renin-angiotensin-aldosteron-rendszer gátlói



1, Renin-inhib.

2, ACEI

3, ARB

4, Ald-inhib.

Endocrine (2024) 83:285–301
https://doi.org/10.1007/s12020-023-03566-6

MINI REVIEW

Extra-adrenal aldosterone: a mini review focusing on the physiology and pathophysiology of intrarenal aldosterone

Chuanming Xu[1]

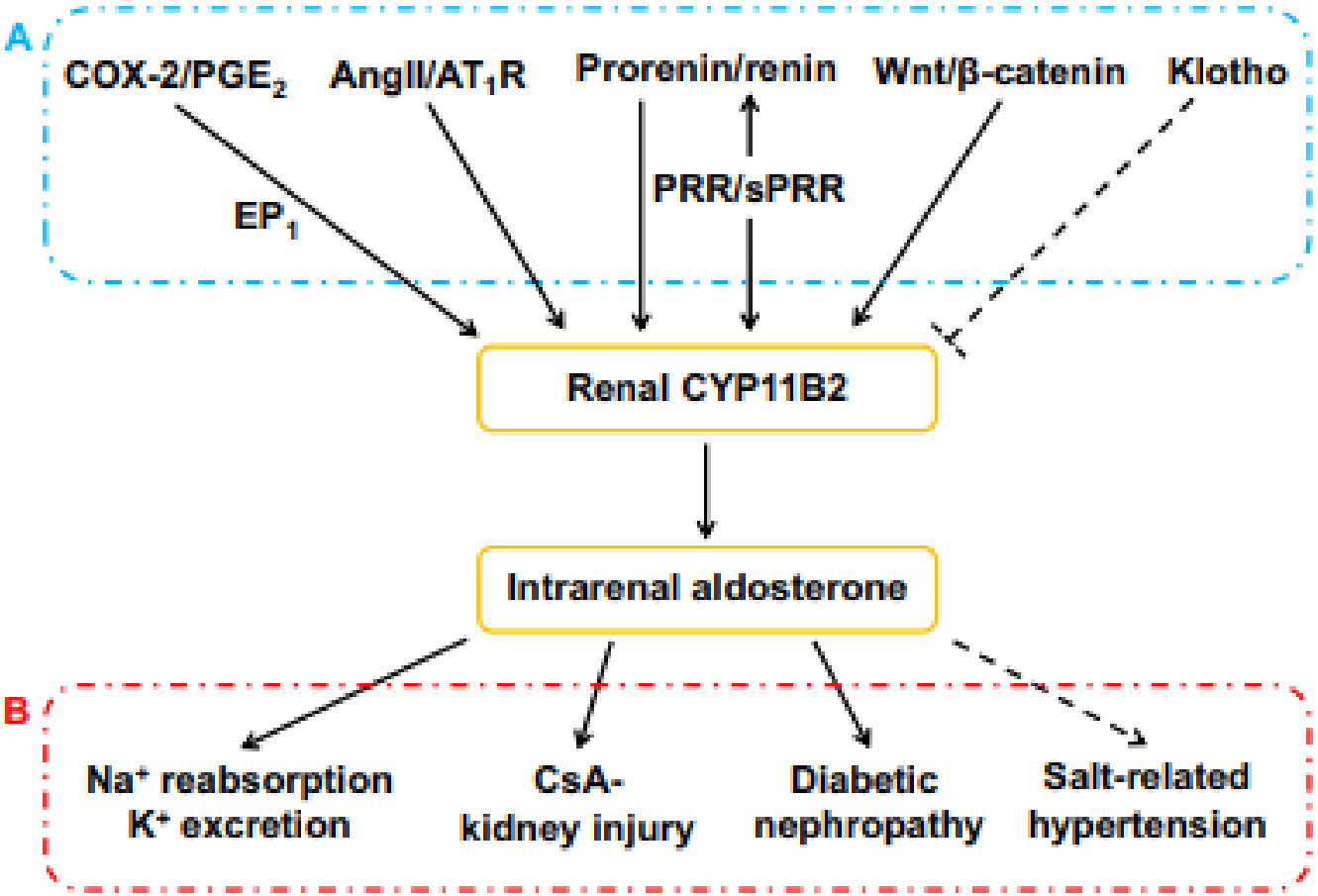


Fig. 3 Regulators (**A**) and physio-pathological effects (**B**) of intrarenal aldosterone. Studies have demonstrated the stimulation of intrarenal CYP11B2/aldosterone by (pro)renin receptor (PRR)/(pro)renin, angiotensin II (AngII)/Angiotensin II type 1 receptor (AT_1R), wnt/β-catenin, and cyclooxygenase-2 (COX-2)/prostaglandin E_2 (PGE_2)/E-prostanoid subtype 1 (EP_1) signaling. Klotho may indirectly inhibit

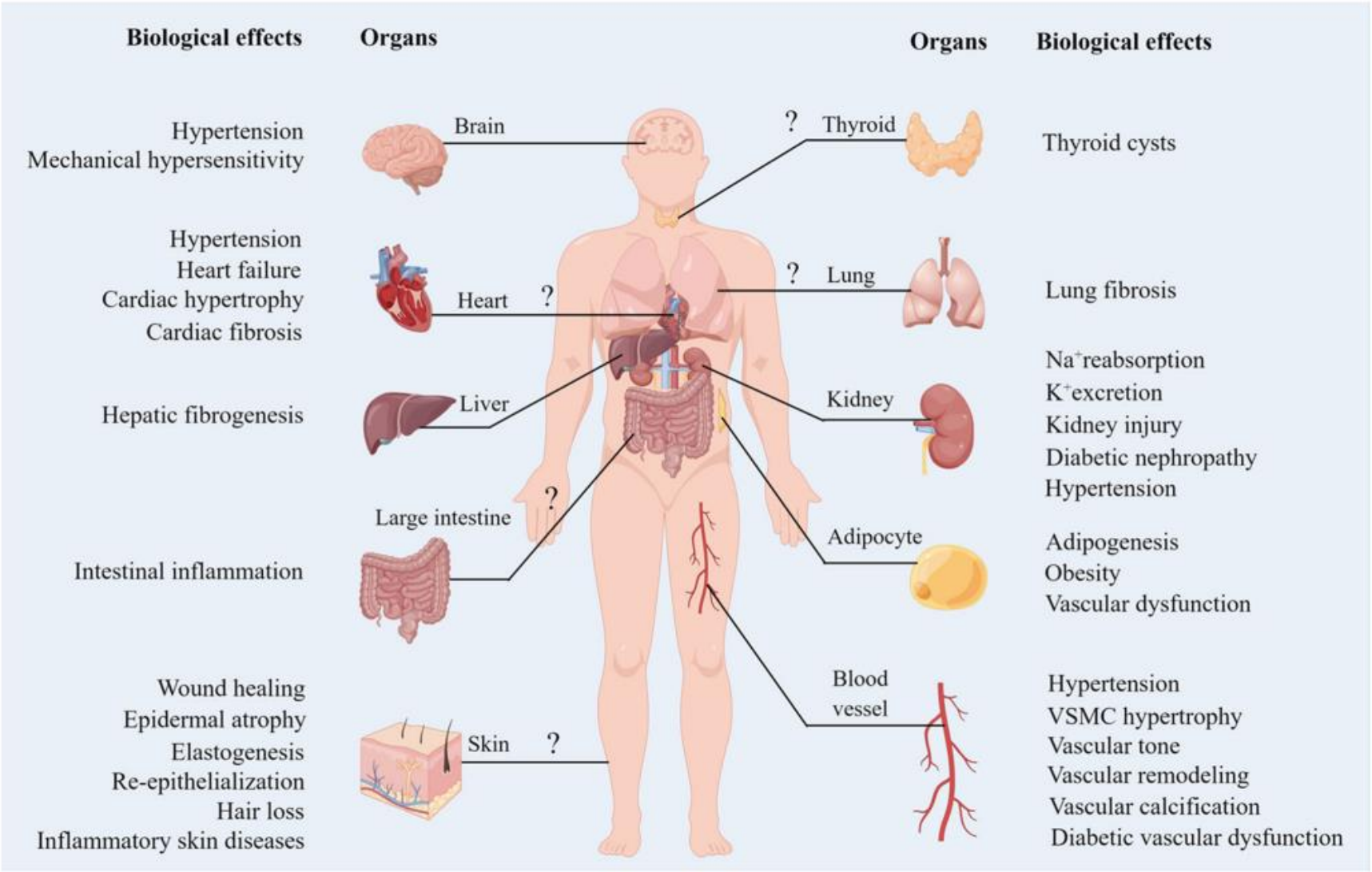


Fig. 2 An overview of the distribution and biological effects of extra-adrenal aldosterone. VSMC vascular smooth muscle cell

Highlights

- Local aldosterone synthesis may present in extra-adrenal tissues like the kidneys and play significant physiological and pathophysiological significance.
- Wnt/β-catenin pathway interacts with PRR/sPRR contributing to intrarenal aldosterone synthesis by activating intrarenal RAS.
- COX-2/PGE_2/EP_1 pathway stimulates intrarenal aldosterone production by activating sPRR/β-catenin signaling.
- Intrarenal aldosterone may contribute to Na^+/K^+ homeostasis, kidney injury, and salt-related hypertension.

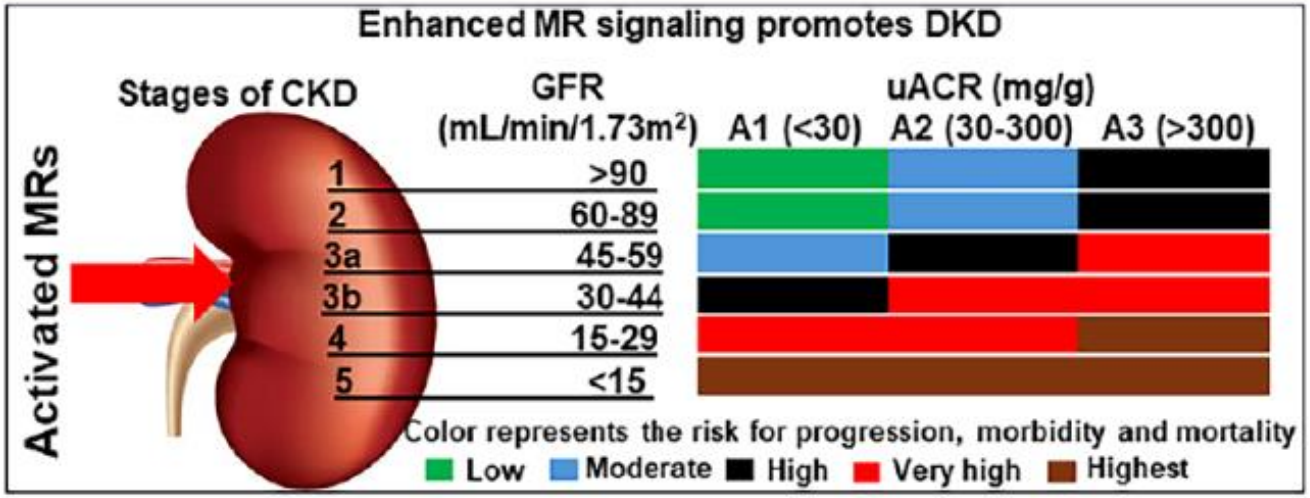
Review > Am J Physiol Renal Physiol. 2024 Sep 1;327(3):F519-F531.
doi: 10.1152/ajprenal.00135.2024. Epub 2024 Jul 18.

The mineralocorticoid receptor in diabetic kidney disease

Guanghong Jia [1 2], Guido Lastra [1 2], Brian P Bostick [3], Nihay LahamKaram [4], Johanna P Laakkonen [4], Seppo Ylä-Herttuala [4 5], Adam Whaley-Connell [1 2 6]

Affiliations + expand

PMID: 39024357 DOI: 10.1152/ajprenal.00135.2024



Molecular mechanisms in DKD

Activated MRs

↓

Renal insulin resistance, microcirculatory dysfunction, mitochondria dysfunction, oxidative stress, inflammation, dyslipidemia, abnormal release of extracellular vesicles, and gut dysbiosis

↓

Epithelial-mesenchymal transition and kidney fibrosis

↓

DKD

Conclusion

Enhanced MR signaling contributes to the development of DKD and targeting the MR is an effective preventive and therapeutic strategies in DKD.

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