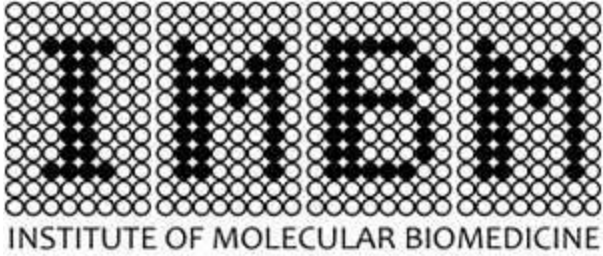
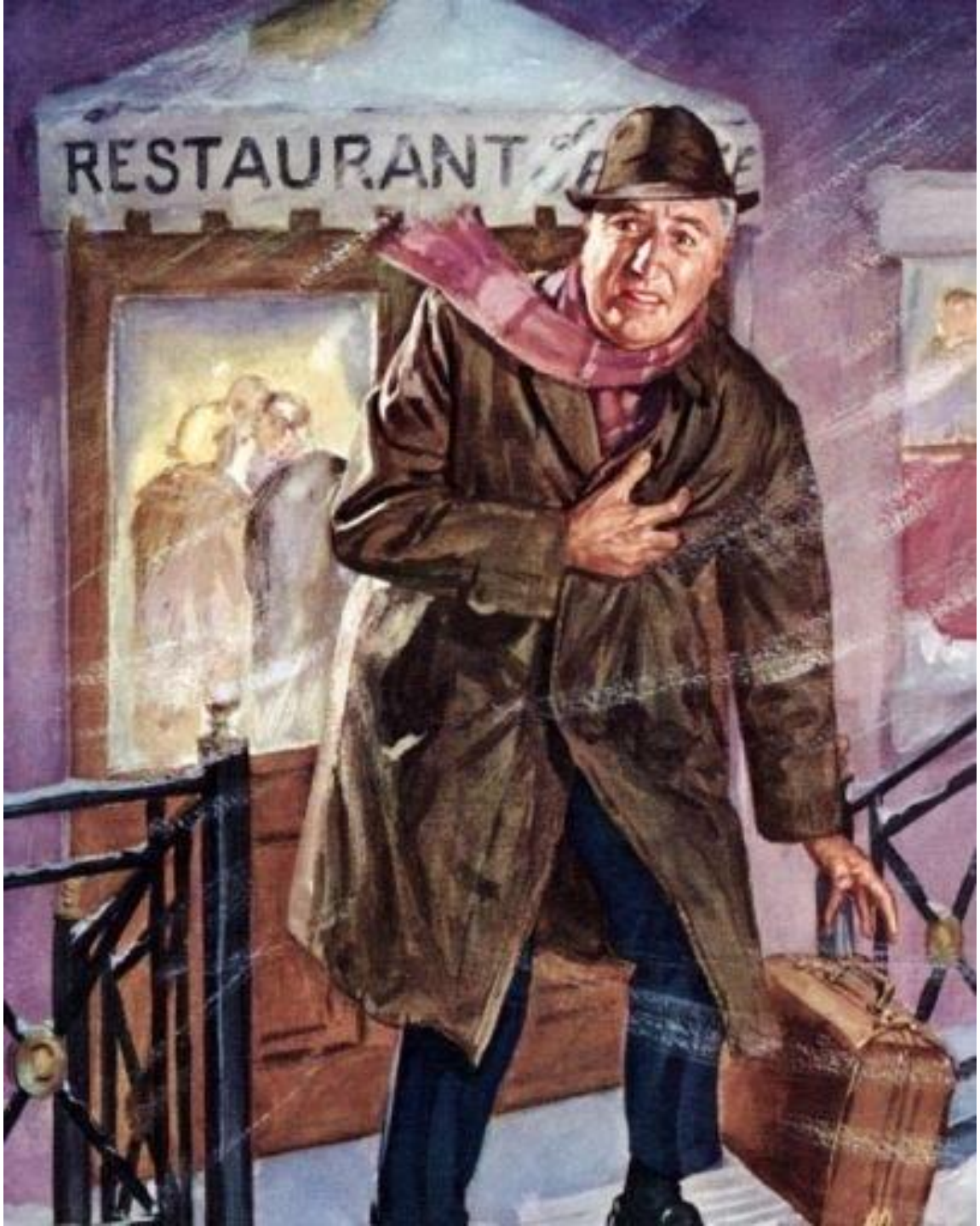


Peter Celec

petercelec@gmail.com
www.imbm.sk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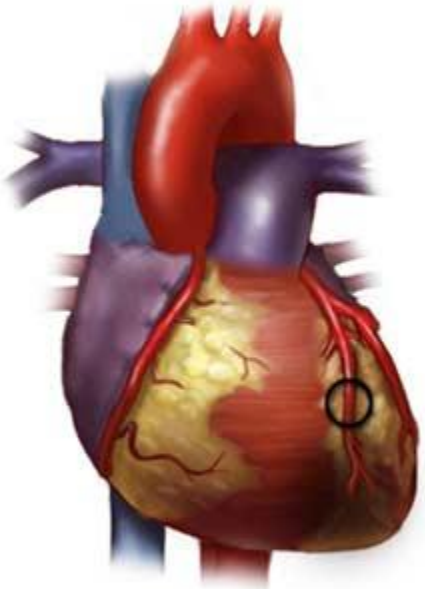
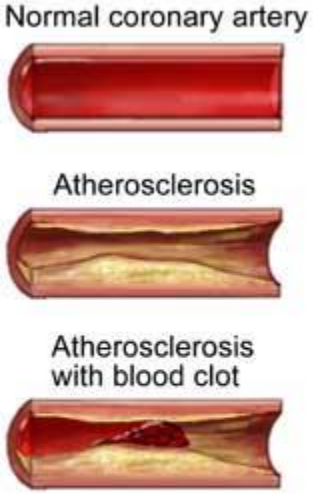
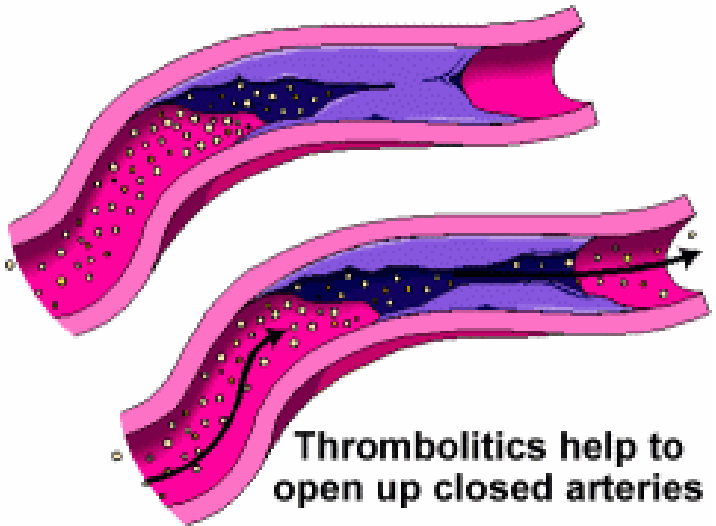
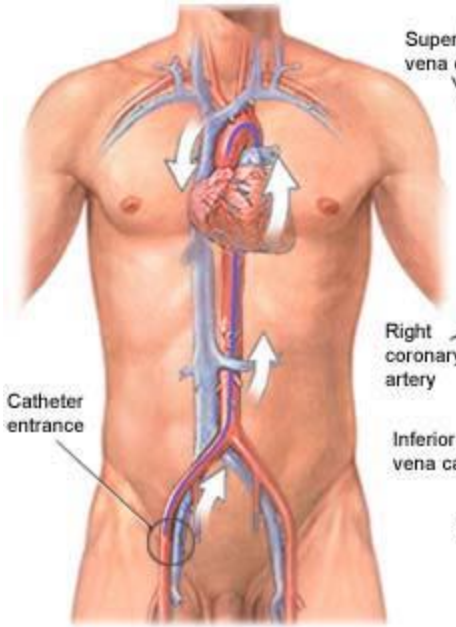
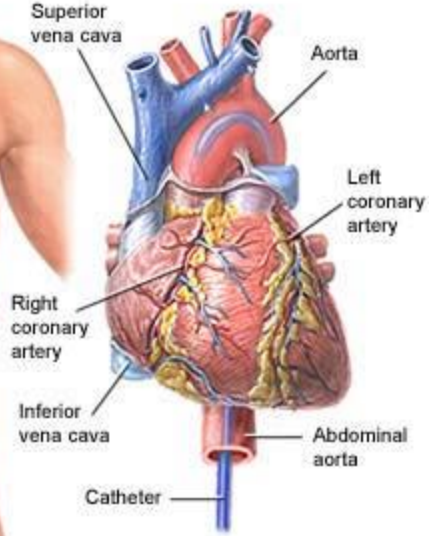



Normal coronary artery
Atherosclerosis
Atherosclerosis
with blood clot


Thrombolitics help to
open up closed arteries

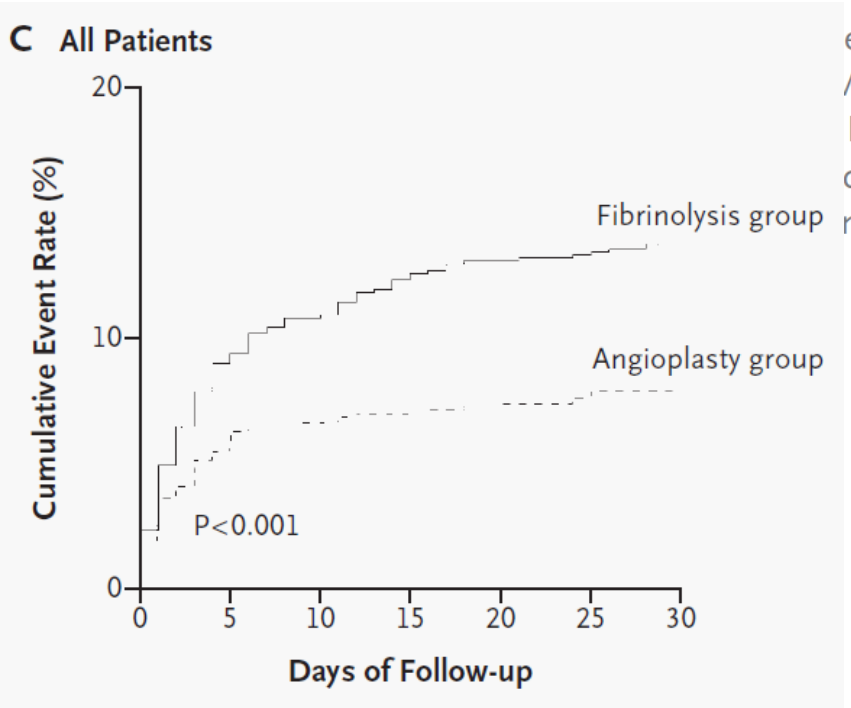

Catheter
entrance


Superior
vena cava
Aorta
Left
coronary
artery
Right
coronary
artery
Inferior
vena cava
Abdominal
aorta
Catheter

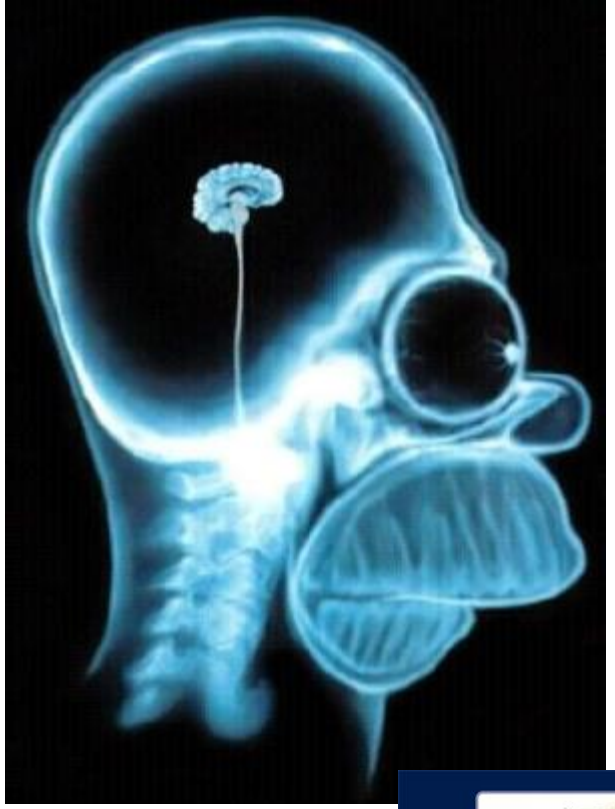
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 AUGUST 21, 2003 VOL. 349 NO. 8

A Comparison of Coronary Angioplasty with Fibrinolytic Therapy in Acute Myocardial Infarction



elsen, M.D., Klaus Rasmussen, M.D., Leif Thuesen, M.D.,
M.D., Ulrik Abildgaard, M.D., Flemming Pedersen, M.D.,
B. Villadsen, M.D., Lars R. Krusell, M.D., Torben Haghfelt, M.D.,
d, M.D., Else Vigholt, M.D., Henrik K. Kjaergard, M.D.,
n, M.Sc., for the DANAMI-2 Investigators*



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The NEW ENGLAND JOURNAL of MEDICINE

VOL. 348 NO. 12

ESTABLISHED IN 1812 MARCH 20, 2003 WWW.NEJM.ORG

Kiňová, Hulín et al.
INTERNÁ MEDICÍNA

PRINCÍPY INTERNEJ MEDICÍNY

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EDITORI
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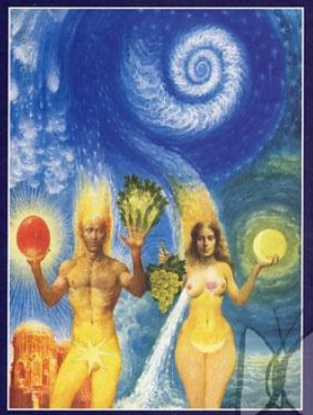
HULÍN
BERNADIČ



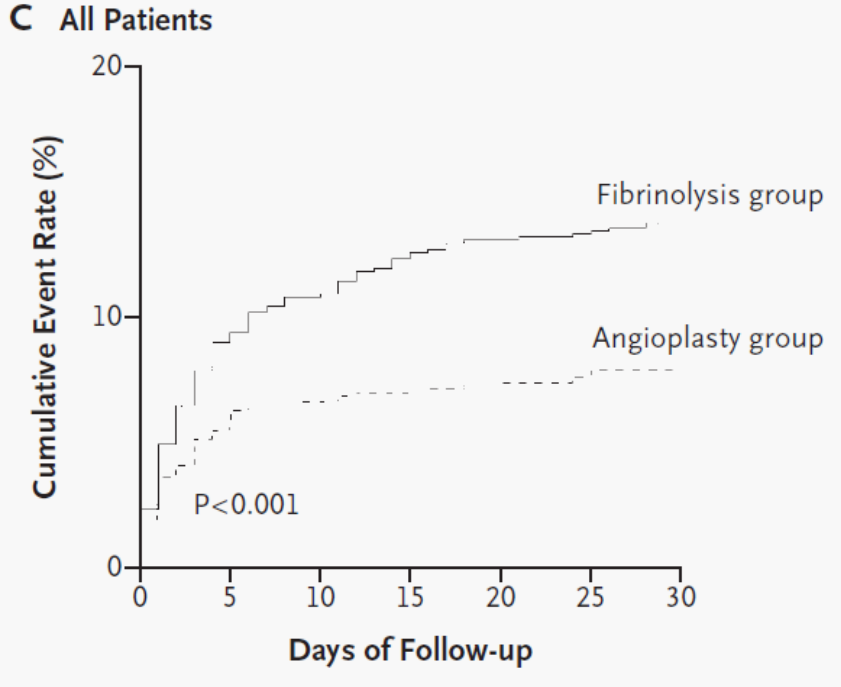
Ako sa dostať k novým poznatkom?

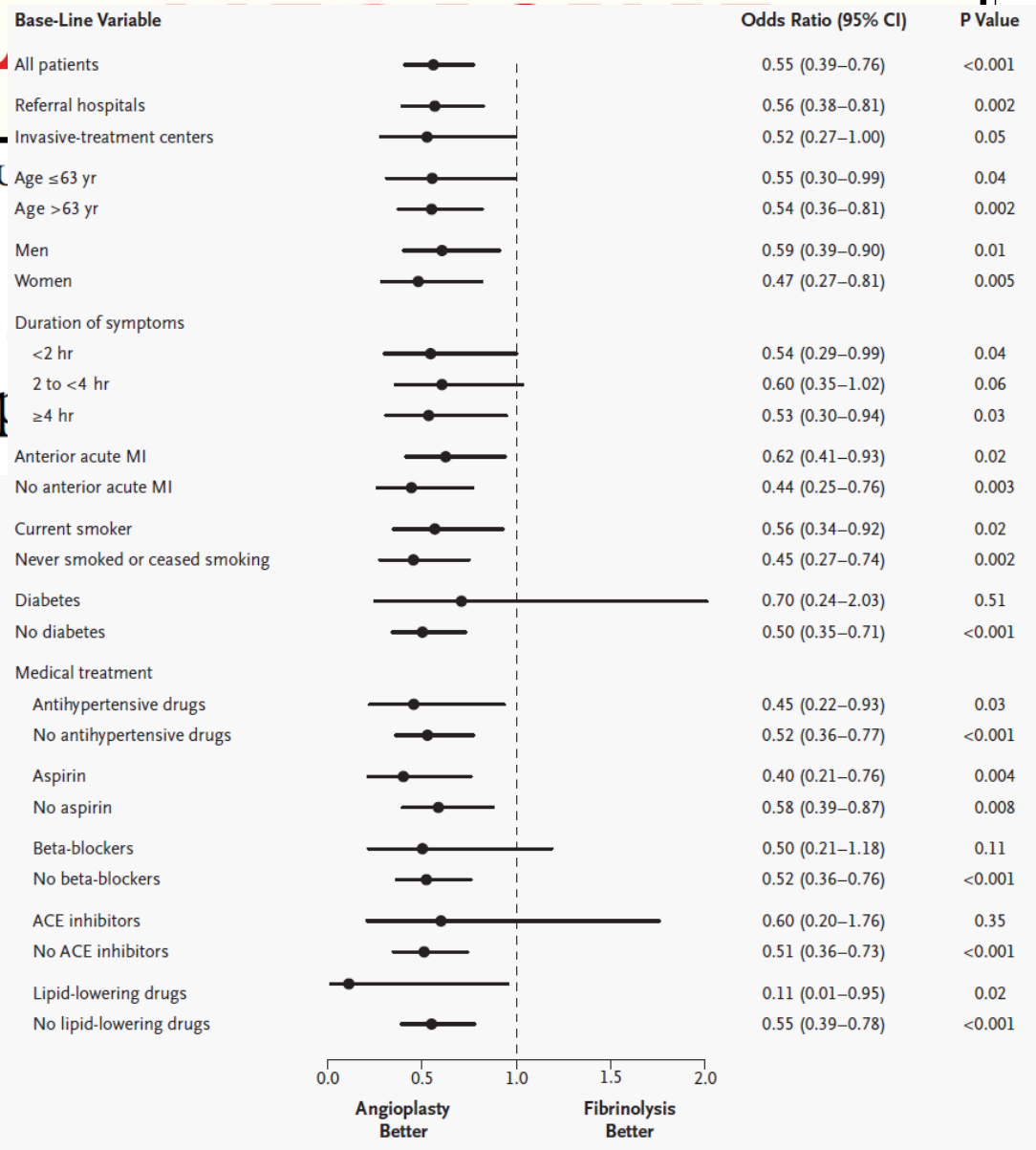
- Hypotézy
- Prevencia vs diagnostika vs terapia
- Pozorovanie vs experimenty
- Klinické fázy
- Merania
- Štatistická analýza

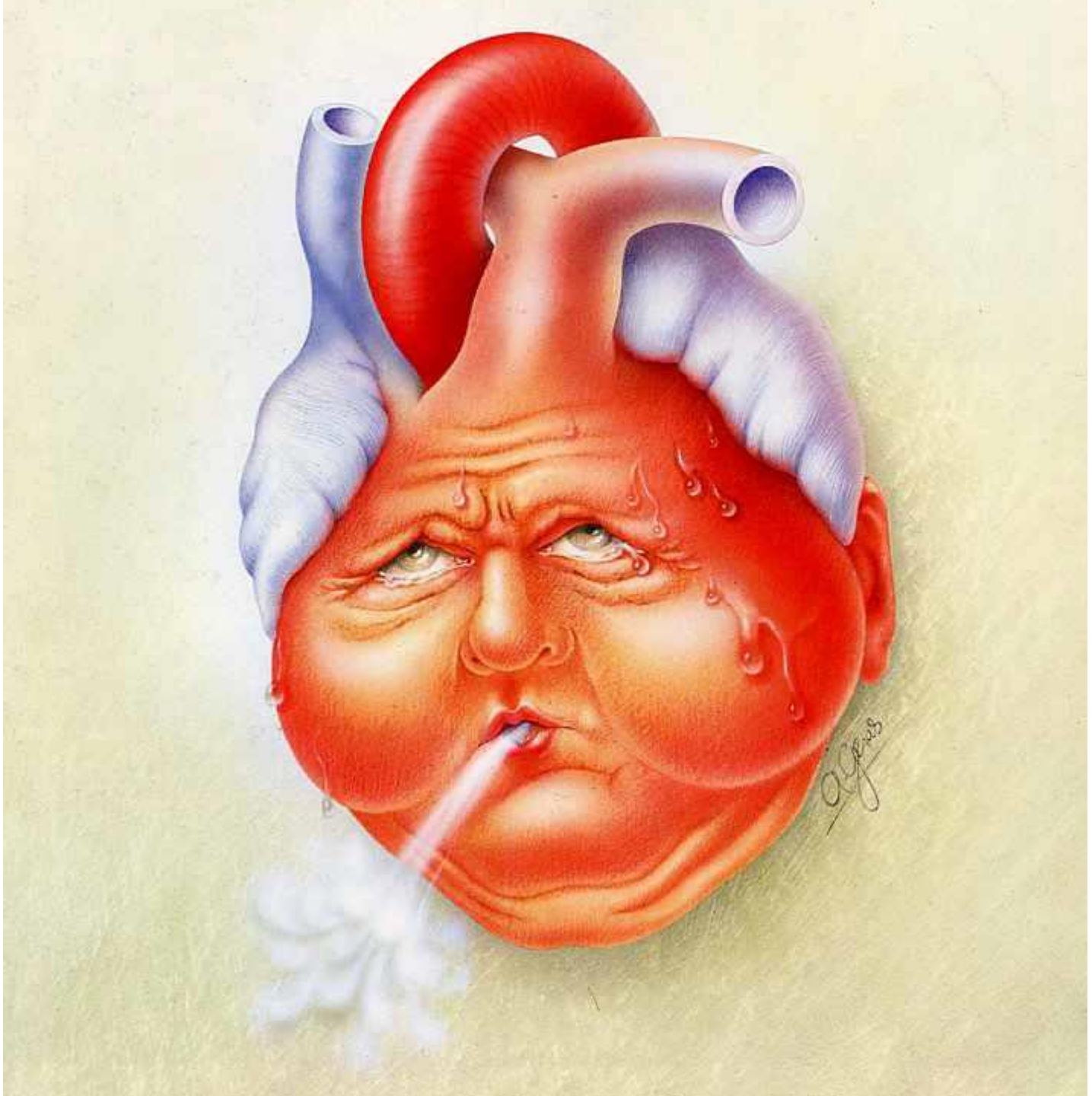
The NEW ENGLAND JOURNAL

ESTABLISHED IN 1812

A Comparison with Fibrinolytic Therap







Br Heart J **(Supplement)** 1994; 72: 92–99

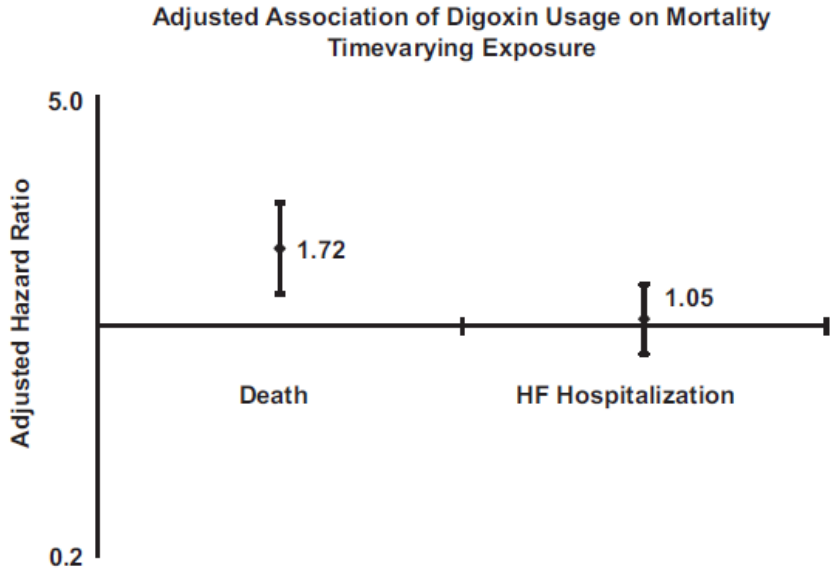
Inotropic agents for heart failure: what if digoxin increases mortality?

W J Remme

Effectiveness and Safety of Digoxin Among Contemporary Adults With Incident Systolic Heart Failure

James V. Freeman, MD, MPH; Jingrong Yang, MA; Sue Hee Sung, MPH; Mark A. Hlatky, MD; Alan S. Go, MD

	Death From Any Cause	Hospitalization for Heart Failure
Therapy	Adjusted Hazard Ratio (95% Confidence Interval)	
Overall cohort	1.72 (1.25–2.36)	1.05 (0.82–1.34)
Men (digoxin vs nondigoxin users)	1.64 (1.09–2.46)	1.11 (0.82–1.48)
Women (digoxin vs nondigoxin users)	1.69 (0.98–2.90)	1.04 (0.71–1.53)
Concurrent β-blockers (digoxin vs nondigoxin users	1.55 (1.11–2.18)	1.08 (0.83–1.42)
No concurrent β-blockers (digoxin vs nondigoxin users	2.49 (1.20–5.17)	0.88 (0.46–1.69)



Klinické štúdie

- Preklinické/Fázy I, II, III, IV

- Observačné / Intervenčné
  - Asociácie vs kauzálne vzťahy
- Placebom kontrolované
- Randomizované
- Dvojito slepé
- Multicentrické
- Prospektívne / retrospektívne
- Longitudinálne / prierezové
- Exklúzne / inklúzne kritériá

Meta-analýzy

- Kohorty z viacerých klinických štúdií
- Variabilita medzi štúdiami
- Štatistická analýza

- Vysoká informačná hodnota
- Váha veľkých čísel

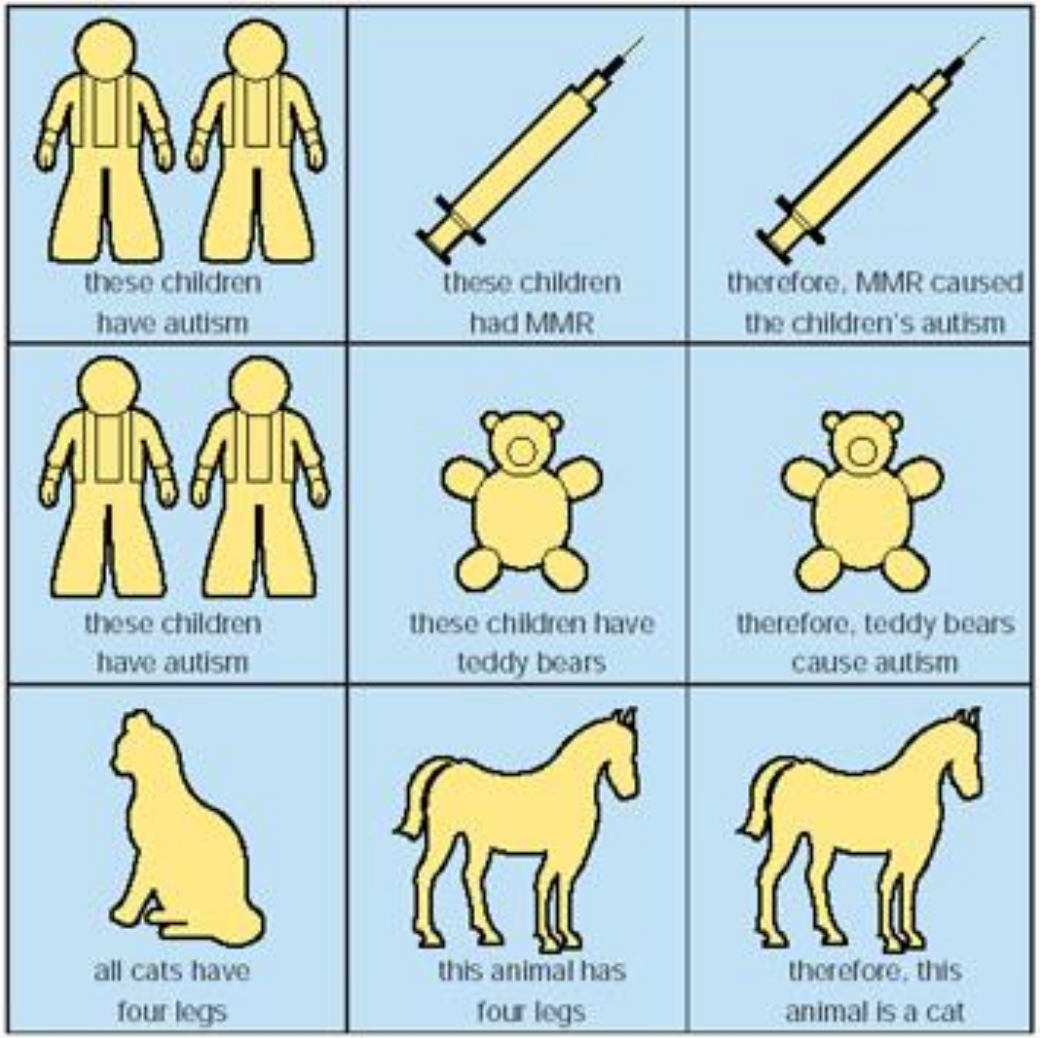
Prístup ku klinickej medicíne

- Empírická medicína
- Expert opinion

- Evidence-based medicine (EBM)
  - Medicína založená na dôkazoch
  - Guidelines – protokoly

Kto je dobrý lekár?

- http://howdovaccinescauseautism.com/



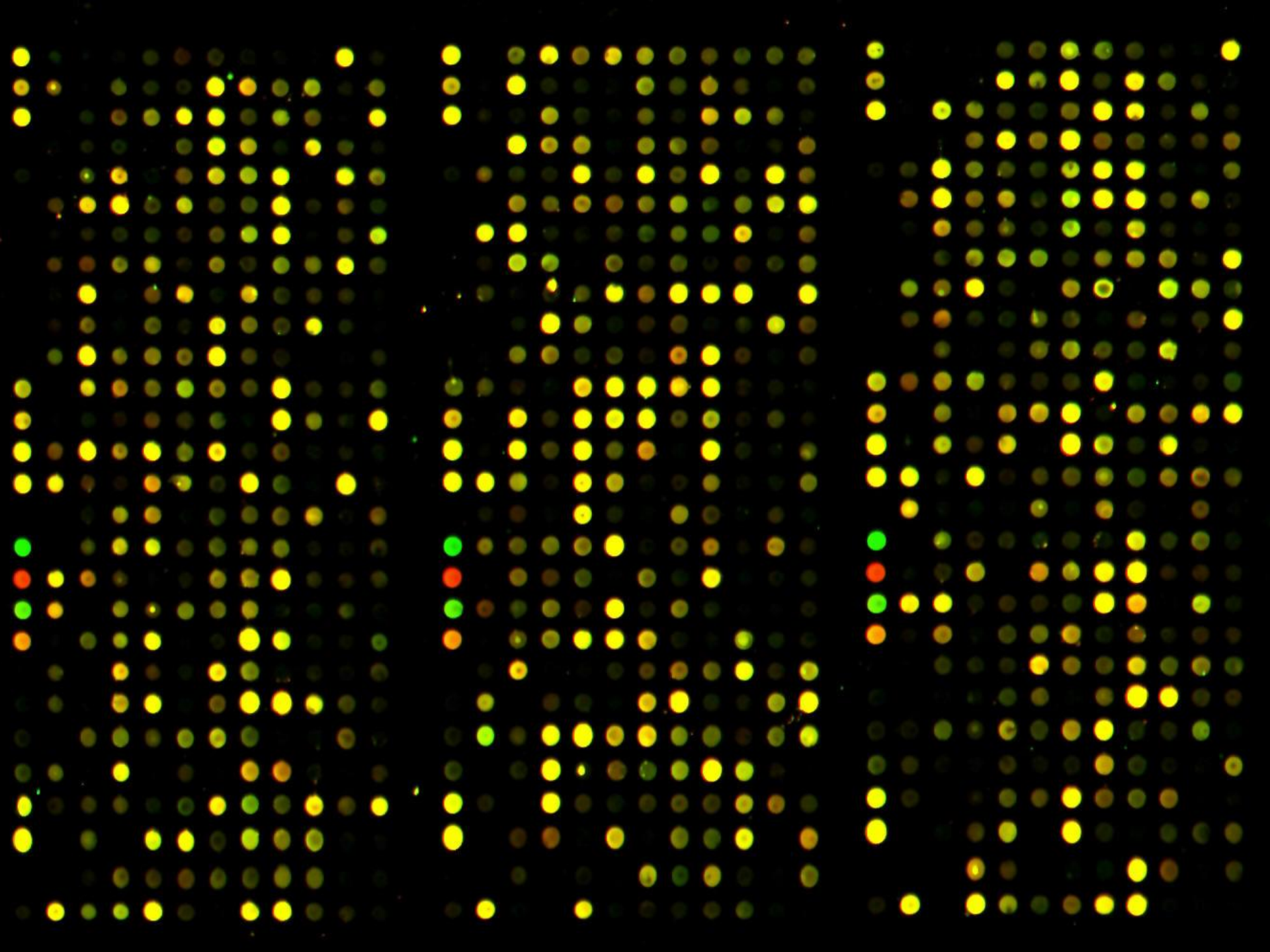
Kto je dobrý lekár?

- Číta
- Má aktuálne informácie (updated)
- Postupuje podľa aktuálnych protokolov

- Zdravotné poisťovne

Veda a medicína

- Čo je cieľom medicíny?
  - Uzdravený pacient? (onkológia, geriatria)
  - Spokojný pacient? (dotazníky, čakacie doby)
  - Maximalizovať benefit pre pacienta (protokoly)
- Čo je cieľom medicínskej vedy?
  - Zlepšovať klinickú prax? (aplikovaný výskum)
  - Hľadať pravdu? (veda)
- BIOmedicínsky výskum
  - Veľké zmeny – systémová biológia
  - Vieme, ale nerozumieme – vadí to?



Vol 447|7 June 2007|doi:10.1038/nature05911 nature

ARTICLES

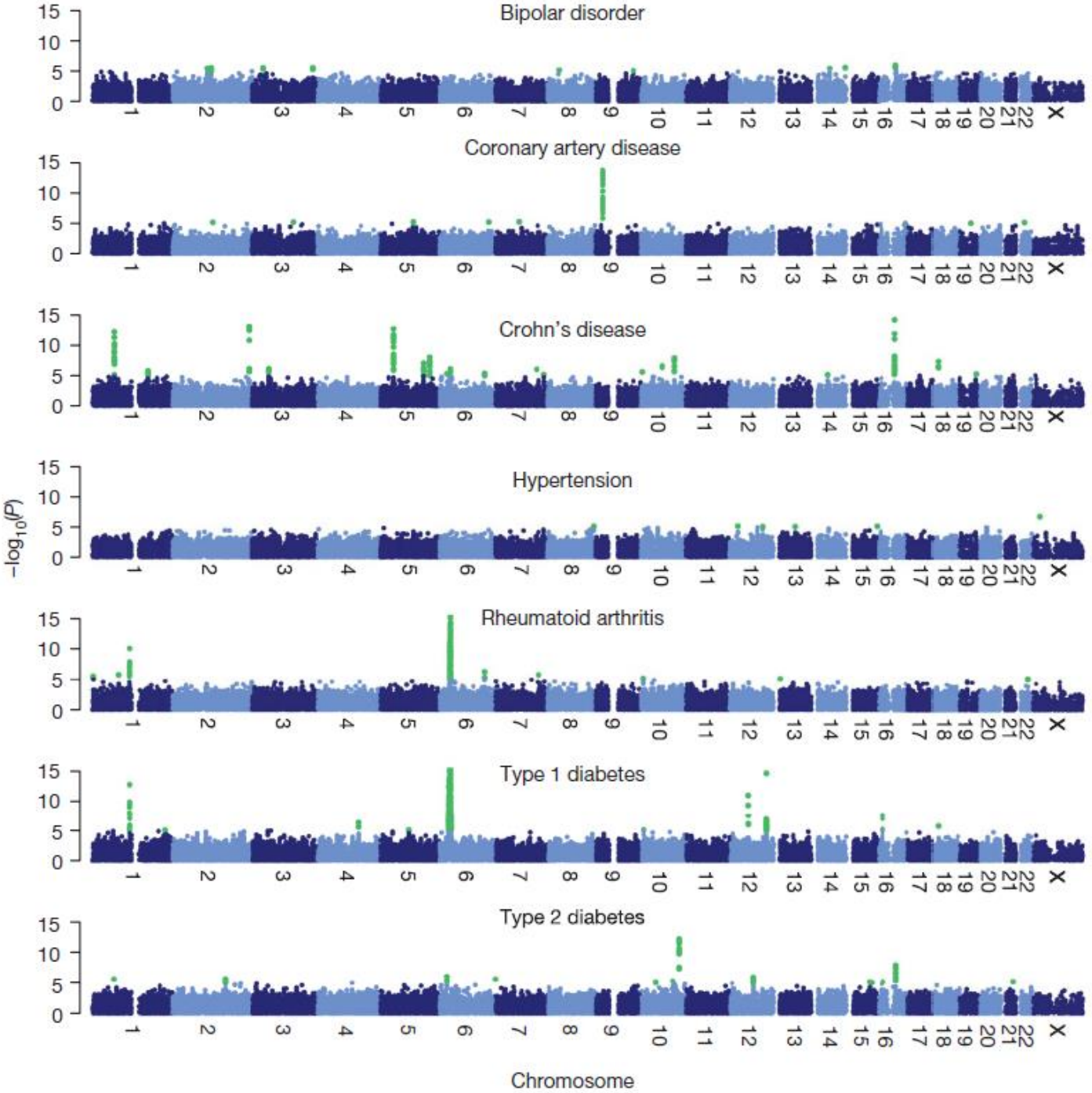
Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls

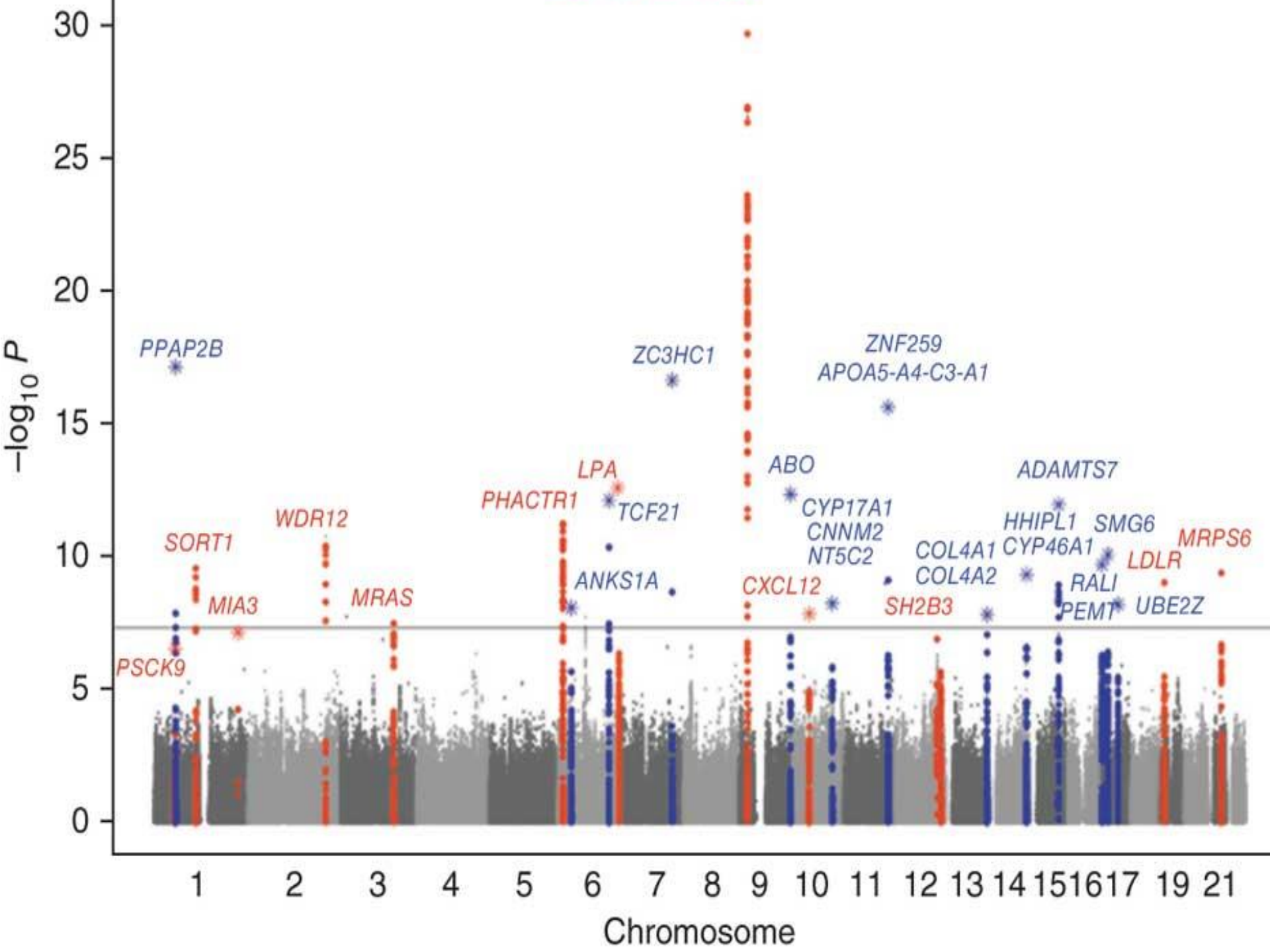
The Wellcome Trust Case Control Consortium*

Vol 464|1 April 2010|doi:10.1038/nature08979 nature

ARTICLES

Genome-wide association study of CNVs in 16,000 cases of eight common diseases and 3,000 shared controls


Bipolar disorder
Coronary artery disease
Crohn's disease
Hypertension
Rheumatoid arthritis
Type 1 diabetes
Type 2 diabetes
−log10(P)
Chromosome


30
25
20
15
10
5
0
$-\log_{10} P$
PPAP2B
ZC3HC1
ZNF259
APOA5-A4-C3-A1
LPA
ABO
ADAMTS7
PHACTR1
TCF21
CYP17A1
CNNM2
NT5C2
HHIPL1
CYP46A1
SMG6
MRPS6
WDR12
SORT1
COL4A1
COL4A2
LDLR
ANKS1A
CXCL12
RALI
MIA3
MRAS
SH2B3
PEMT
UBE2Z
PSCK9
1
2
3
4
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7
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12
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15
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17
19
21
Chromosome

Table 3 | Regions of the genome showing the strongest association signals

Collection	Chromosome	Region (Mb)	SNP	Trend *P* value	Genotypic *P* value	$\log_{10}$(BF), additive	$\log_{10}$(BF), general	Risk allele	Minor allele	Heterozygote odds ratio	Homozygote odds ratio	Control MAF	Case MAF
					Standard analysis								
BD	16p12	23.3–23.62	rs420259	2.19×10^{-04}	6.29×10^{-08}	1.96	4.79	A	G	2.08 (1.60–2.71)	2.07 (1.6–2.69)	0.282	0.248
CAD	9p21	21.93–22.12	rs1333049	1.79×10^{-14}	1.16×10^{-13}	11.66	11.19	C	C	1.47 (1.27–1.70)	1.9 (1.61–2.24)	0.474	0.554
CD	1p31	67.3–67.48	rs11805303	6.45×10^{-13}	5.85×10^{-12}	10.07	9.41	T	T	1.39 (1.22–1.58)	1.86 (1.54–2.24)	0.317	0.391
CD	2q37	233.92–234	rs10210302	7.10×10^{-14}	5.26×10^{-14}	11.11	11.28	T	C	1.19 (1.01–1.41)	1.85 (1.56–2.21)	0.481	0.402
CD	3p21	49.3–49.87	rs9858542	7.71×10^{-07}	3.58×10^{-08}	4.24	5.22	A	A	1.09 (0.96–1.24)	1.84 (1.49–2.26)	0.282	0.331
CD	5p13	40.32–40.66	rs17234657	2.13×10^{-13}	1.99×10^{-12}	10.41	9.89	G	G	1.54 (1.34–1.76)	2.32 (1.59–3.39)	0.125	0.181
CD	5q33	150.15–150.31	rs1000113	5.10×10^{-08}	3.15×10^{-07}	5.36	5.01	T	T	1.54 (1.31–1.82)	1.92 (0.92–4.00)	0.067	0.098
CD	10q21	64.06–64.31	rs10761659	2.68×10^{-07}	1.75×10^{-06}	4.69	4.13	G	A	1.23 (1.05–1.45)	1.55 (1.3–1.84)	0.461	0.406
CD	10q24	101.26–101.32	rs10883365	1.41×10^{-08}	5.82×10^{-08}	5.91	5.48	G	G	1.2 (1.03–1.39)	1.62 (1.37–1.92)	0.477	0.537
CD	16q12	49.02–49.4	rs17221417	9.36×10^{-12}	3.98×10^{-11}	8.93	8.47	G	G	1.29 (1.13–1.46)	1.92 (1.58–2.34)	0.287	0.356
CD	18p11	12.76–12.91	rs2542151	4.56×10^{-08}	2.03×10^{-07}	5.42	5.00	G	G	1.3 (1.14–1.48)	2.01 (1.46–2.76)	0.163	0.208
RA	1p13	113.54–114.16	rs6679677	4.90×10^{-26}	5.55×10^{-25}	22.36	21.99	A	A	1.98 (1.72–2.27)	3.32 (1.93–5.69)	0.096	0.168
RA	6	MHC	rs6457617*	3.44×10^{-76}	5.18×10^{-75}	74.84	73.18	T	T	2.36 (1.97–2.84)	5.21 (4.31–6.30)	0.489	0.685
T1D	1p13	113.54–114.16	rs6679677	1.17×10^{-26}	5.43×10^{-26}	23.07	22.83	A	A	1.82 (1.59–2.09)	5.19 (3.15–8.55)	0.096	0.169
T1D	6	MHC	rs9272346*	2.42×10^{-134}	5.47×10^{-134}	141.9	142.2	A	G	5.49 (4.83–6.24)	18.52 (27.03–12.69)	0.387	0.150
T1D	12q13	54.64–55.09	rs11171739	1.14×10^{-11}	9.71×10^{-11}	8.89	8.24	C	C	1.34 (1.17–1.54)	1.75 (1.48–2.06)	0.423	0.493
T1D	12q24	109.82–111.49	rs17696736	2.17×10^{-15}	1.51×10^{-14}	12.53	11.88	G	G	1.34 (1.16–1.53)	1.94 (1.65–2.29)	0.424	0.506
T1D	16p13	10.93–11.37	rs12708716	9.24×10^{-08}	4.92×10^{-07}	5.15	4.70	A	G	1.19 (0.97–1.45)	1.55 (1.27–1.89)	0.350	0.297
T2D	6p22	20.63–20.84	rs9465871	1.02×10^{-06}	3.34×10^{-07}	4.15	3.98	C	C	1.18 (1.04–1.34)	2.17 (1.6–2.95)	0.178	0.218
T2D	10q25	114.71–114.81	rs4506565	5.68×10^{-13}	5.05×10^{-12}	10.14	9.43	T	T	1.36 (1.2–1.54)	1.88 (1.56–2.27)	0.324	0.395
T2D	16q12	52.36–52.41	rs9939609	5.24×10^{-08}	1.91×10^{-07}	5.35	5.05	A	A	1.34 (1.17–1.52)	1.55 (1.3–1.84)	0.398	0.453
					Multi-locus analysis								
T1D	4q27	123.26–123.92	rs6534347	4.48×10^{-07}	1.83×10^{-06}	5.15	4.69	A	A	1.30 (1.10–1.55)	1.49 (1.25–1.78)	0.351	0.402
T1D	12p13	9.71–9.86	rs3764021	7.19×10^{-05}	5.08×10^{-08}	2.12	4.55	C	T	1.57 (1.38–1.79)	1.48 (1.25–1.75)	0.467	0.426
					Sex differentiated analysis								
RA	7q32	130.80–130.84	rs11761231	3.91×10^{-07}	1.37×10^{-06}	-	-	G	A	1.44 (1.19–1.75)	1.64 (1.35–1.99)	0.375	0.327
					Combined cases								
RA+T1D	10p15	6.07–6.17	rs2104286	5.92×10^{-08}	2.52×10^{-07}	5.26	4.45	T	C	1.35 (1.11–1.65)	1.62 (1.34–1.97)	0.286	0.245

Regions with at least one SNP with a *P* value of less than 5×10^{-7} for our primary analyses. The $\log_{10}$ value of the Bayes factor (BF) for the bayesian analysis corresponding to the trend and genotypic tests is also given. Region marks the boundaries of signal defined by recombination and return of test statistics to background levels. The minor allele is defined in the controls and its frequency in that group as well as the case sample is reported. MAF, minor allele frequency. Cluster plots for each SNP have been inspected visually, and are shown in Supplementary Fig. 10. Positions are in NCBI build-35 coordinates *Multiple SNPs in the MHC region are significant, we report the most extreme.

A Common Allele on Chromosome 9 Associated with Coronary Heart Disease

Ruth McPherson,[1]*† Alexander Pertsemlidis,[2]* Nihan Kavaslar,[1] Alexandre Stewart,[1] Robert Roberts,[1] David R. Cox,[3] David A. Hinds,[3] Len A. Pennacchio,[4,5] Anne Tybjaerg-Hansen,[6] Aaron R. Folsom,[7] Eric Boerwinkle,[8] Helen H. Hobbs,[2,9] Jonathan C. Cohen[2,10]†

Coronary heart disease (CHD) is a major cause of death in Western countries. We used genome-wide association scanning to identify a 58-kilobase interval on chromosome 9p21 that was consistently associated with CHD in six independent samples (more than 23,000 participants) from four Caucasian populations. This interval, which is located near the *CDKN2A* and *CDKN2B* genes, contains no annotated genes and is not associated with established CHD risk factors such as plasma lipoproteins, hypertension, or diabetes. Homozygotes for the risk allele make up 20 to 25% of Caucasians and have a ~30 to 40% increased risk of CHD.

Arteriosclerosis, Thrombosis, and Vascular Biology

Journal of the American Heart Association

American Heart Association

Arterioscler Thromb Vasc Biol. 2009;29:1671-1677

Functional Analysis of the Chromosome 9p21.3 Coronary Artery Disease Risk Locus

Olga Jarinova, Alexandre F.R. Stewart, Robert Roberts, George Wells, Paulina Lau, Thet Naing, Christine Buerki, Bradley W. McLean, Richard C. Cook, Joel S. Parker, Ruth McPherson

Targeted deletion of the 9p21 non-coding coronary artery disease risk interval in mice

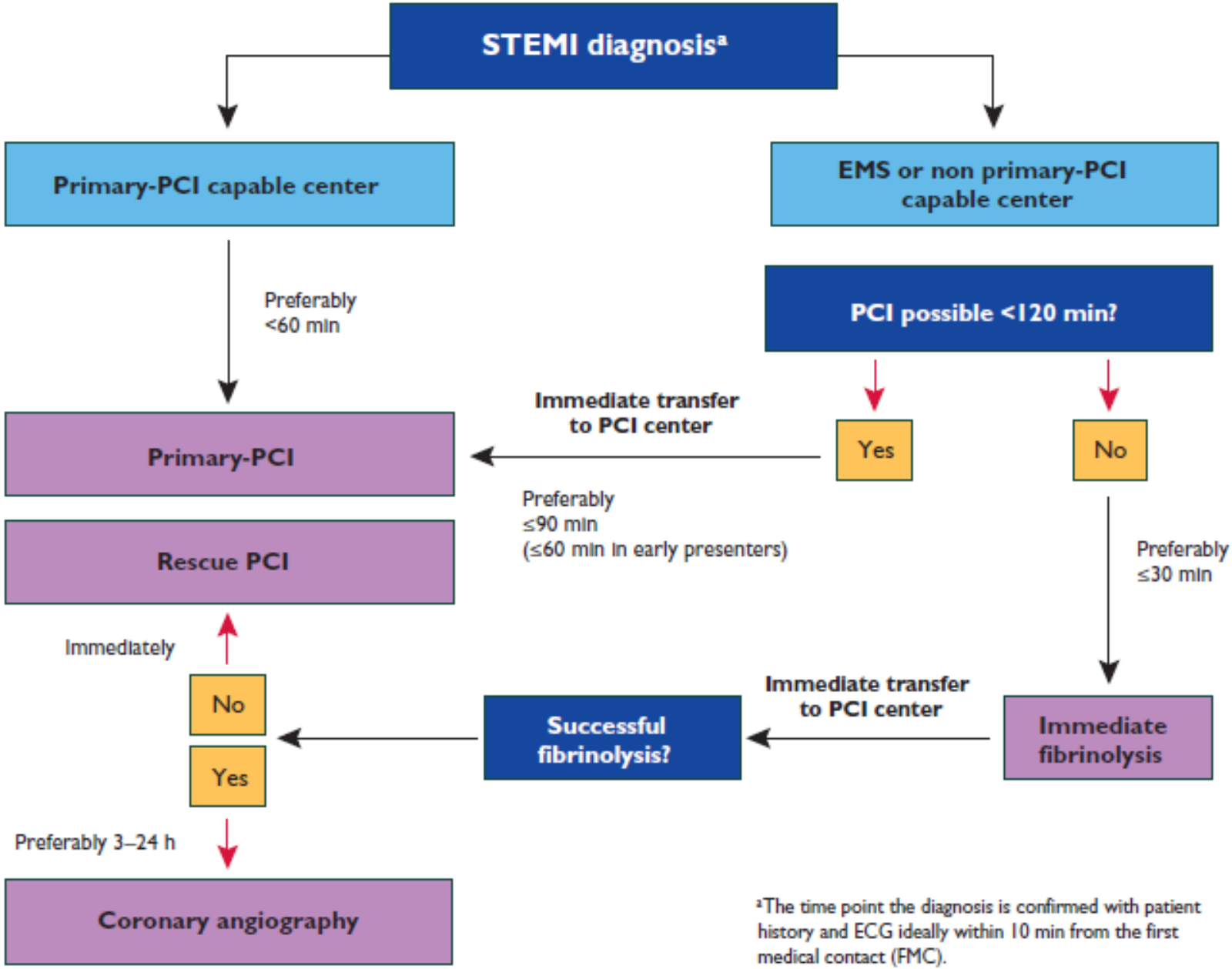
Axel Visel[1,2], Yiwen Zhu[1], Dalit May[1], Veena Afzal[1], Elaine Gong[1], Catia Attanasio[1], Matthew J. Blow[1,2], Jonathan C. Cohen[3], Edward M. Rubin[1,2] & Len A. Pennacchio[1,2]

Vol 464|18 March 2010|doi:10.1038/nature08801

nature

European Heart Journal (2012) **33**, 2569–2619
doi:10.1093/eurheartj/ehs215

ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

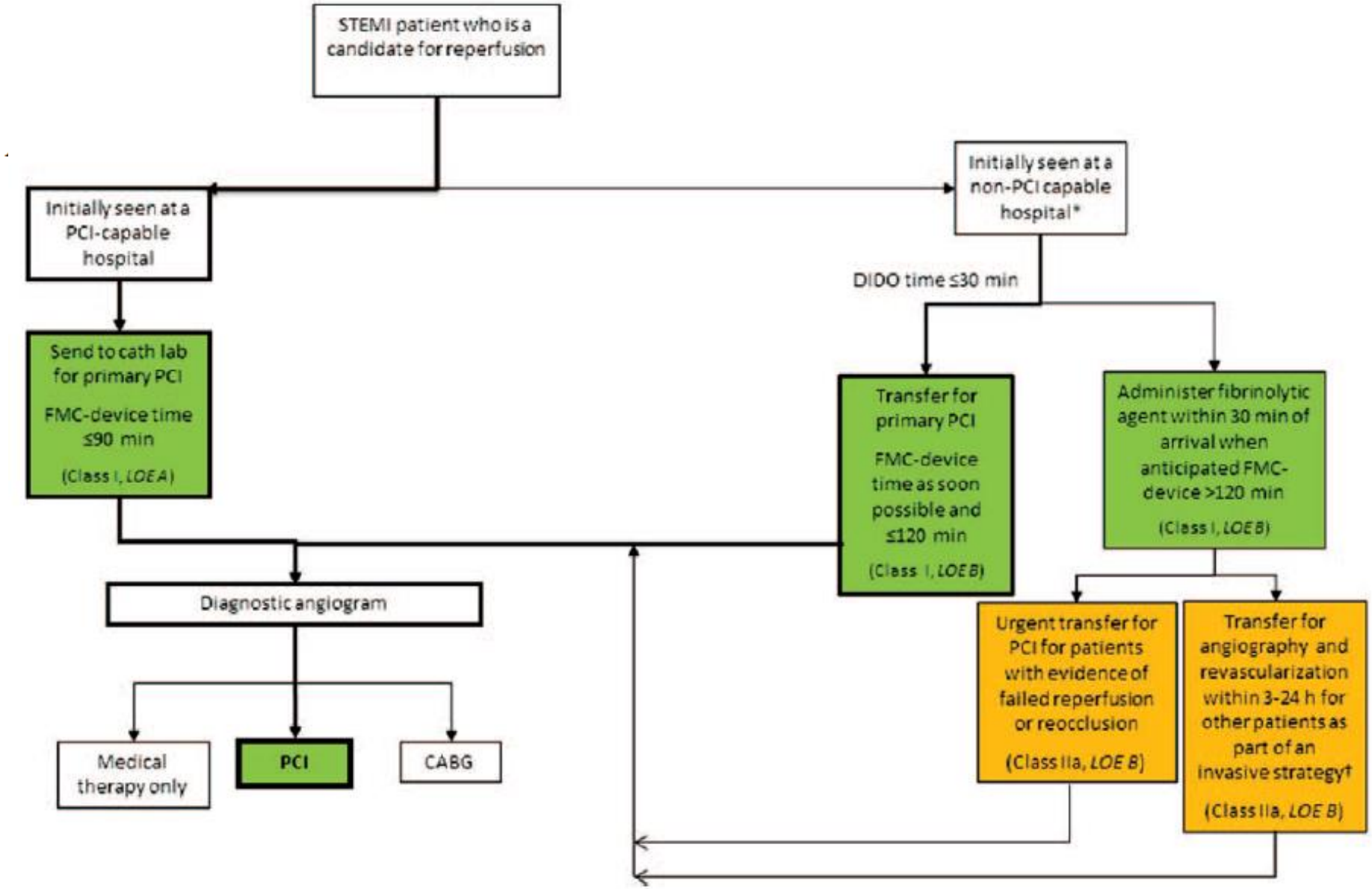


[a]The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from the first medical contact (FMC).
All delays are related to FMC (first medical contact).

Cath = catheterization laboratory; EMS = emergency medical system; FMC = first medical contact; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

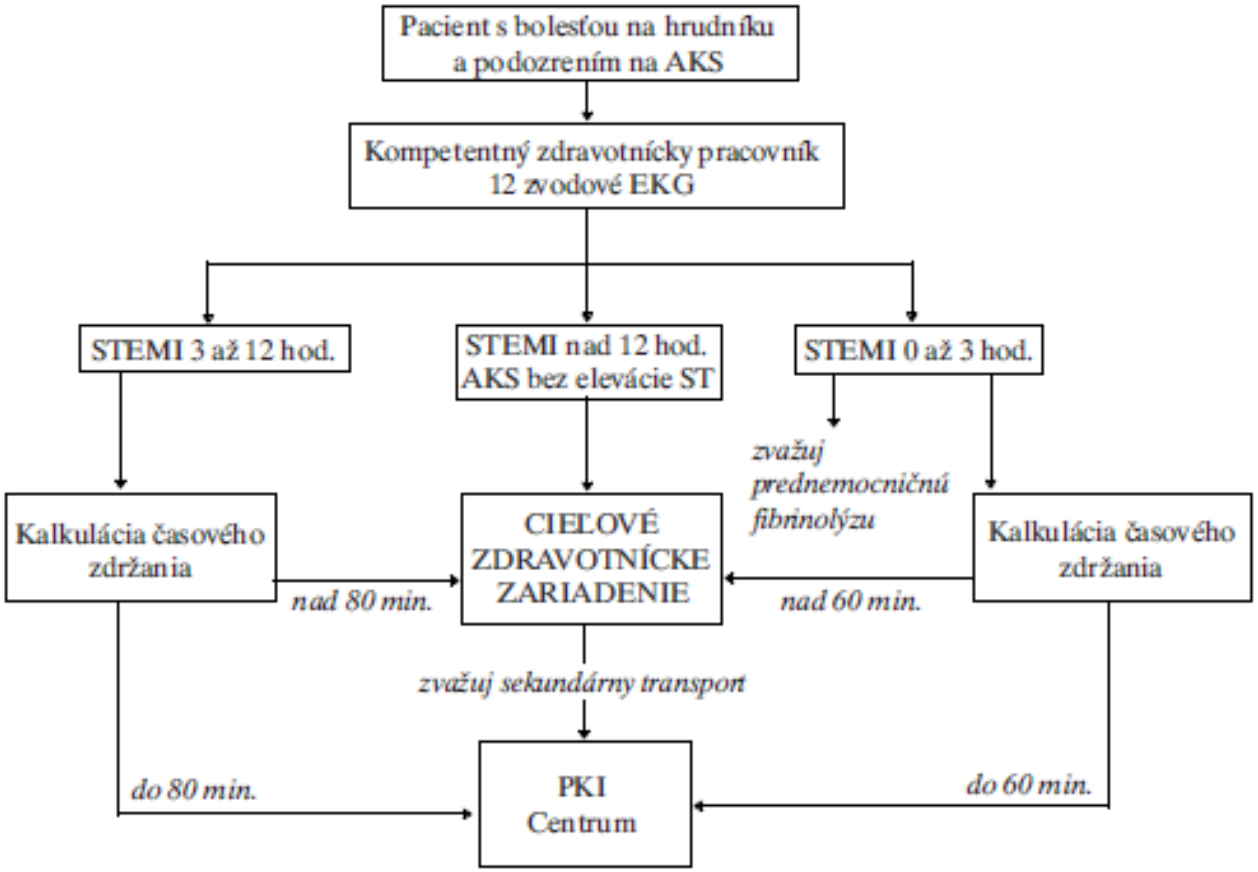
Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association®



Odborné usmernenie Ministerstva zdravotníctva Slovenskej republiky

pre reperfúznu liečbu akútneho infarktu myokardu s eleváciami ST segmentu



Prístup ku klinickej medicíne

- Empírická medicína
- Expert opinion

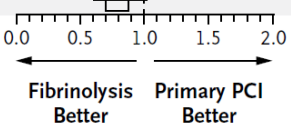
- Evidence-based medicine (EBM)
  - Medicína založená na dôkazoch
  - Guidelines – protokoly
- **Individualizovaná medicína**

J
ESTA

Fibr

Paul W. A
Thierry Danay
Miodrag Ostojic
Hans-Richard A
Claudio Fr
Kris Bo

Subgroup	Fibrinolysis (N=944)	Primary PCI (N=948)	Relative Risk (95% CI) of Primary End Point	P Value	P Value for Interaction
	no. of patients/total no. (%)				
Overall event rate	116/939 (12.4)	135/943 (14.3)	0.86 (0.68–1.09)	0.21	
Age					0.63
<75 yr	80/805 (9.9)	100/823 (12.2)	0.82 (0.62–1.08)	0.16	
≥75 yr	36/134 (26.9)	35/120 (29.2)	0.92 (0.62–1.37)	0.68	
Time to randomization					0.81
0 to <2 hr	79/642 (12.3)	93/640 (14.5)	0.85 (0.64–1.12)	0.24	
≥2 hr	37/297 (12.5)	42/303 (13.9)	0.90 (0.60–1.36)	0.61	
Sex					0.71
Male	77/746 (10.3)	90/737 (12.2)	0.85 (0.63–1.13)	0.25	
Female	39/193 (20.2)	45/206 (21.8)	0.93 (0.63–1.35)	0.69	
Systolic blood pressure					0.16
<100 mm Hg	5/36 (13.9)	14/44 (31.8)	0.44 (0.17–1.10)	0.08	
100 to <140 mm Hg	56/465 (12.0)	68/444 (15.3)	0.79 (0.57–1.09)	0.15	
140 to <160 mm Hg	34/269 (12.6)	27/266 (10.2)	1.25 (0.77–2.00)	0.37	
≥160 mm Hg	21/169 (12.4)	25/188 (13.3)	0.93 (0.54–1.61)	0.81	
Killip class					0.23
I	95/838 (11.2)	104/839 (12.4)	0.91 (0.70–1.17)	0.46	
II–IV	16/52 (30.8)	24/50 (48.0)	0.64 (0.39–1.06)	0.08	
Infarct location					0.06
Anterior	80/449 (17.8)	72/429 (16.8)	1.06 (0.79–1.42)	0.69	
Inferior	34/467 (7.3)	61/494 (12.3)	0.59 (0.40–0.88)	0.01	
Other	2/21 (9.5)	2/18 (11.1)	0.86 (0.13–5.48)	0.87	
Hypertension					0.34
No	48/494 (9.9)	67/516 (13.0)	0.76 (0.54–1.08)	0.12	
Yes	65/431 (15.2)	66/412 (15.9)	0.96 (0.70–1.31)	0.78	
Diabetes					0.24
No	93/816 (11.6)	115/813 (14.1)	0.82 (0.63–1.06)	0.12	
Yes	21/113 (18.5)	19/122 (15.6)	1.19 (0.68–2.09)	0.55	
Weight					0.35
<60 kg	12/47 (25.5)	9/53 (17.0)	1.50 (0.70–3.25)	0.30	
60 to <90 kg	81/648 (12.5)	99/647 (15.3)	0.82 (0.62–1.07)	0.15	
≥90 kg	23/244 (9.4)	27/243 (11.1)	0.85 (0.50–1.44)	0.54	
Place of randomization					0.68
Ambulance	91/764 (11.9)	107/757 (14.1)	0.84 (0.65–1.09)	0.20	
Community hospital	25/175 (14.3)	28/186 (15.1)	0.95 (0.58–1.56)	0.84	
TIMI risk score					0.71
<5	73/749 (10.0)	82/745 (11.2)	0.89 (0.67–1.19)	0.43	
≥5	31/114 (28.2)	41/118 (34.6)	0.82 (0.56–1.19)	0.29	
Time of randomization					0.13
Before amendment	31/192 (16.1)	25/187 (13.4)	1.21 (0.74–1.97)	0.45	
After amendment	85/747 (11.4)	110/756 (14.6)	0.78 (0.60–1.02)	0.07	



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Publikácie!!!

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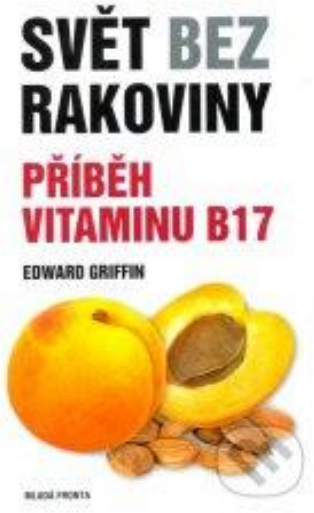
necenzurované informácie o očkovaní

Dr. Andrew Wakefield mal pravdu: MMR vakcína spôsobuje autizmus a zápalové ochorenie čriev



diva.sk

Rakovinu liečia vitamínom C už aj na Slovensku a v Česku, výsledky sú vraj veľmi povzbudivé

16.07.2013 Autor: Alex Berger, Diva.sk, Foto: Dreamstime

Odporučiť 2,8 tis.

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Rodičia detí po očkovaní šokujú: Prejavil sa u nich autizmus aj slepota!

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1. Guardian of **truth**.
Brooks A.
Nurs Manag (Harrow). 2014 Jan;20(9):15. doi: 10.7748/nm2014.02.20.9.15.s16.
PMID: 24479919 [PubMed - in process]
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2. Biomarker accuracy: exploring the **truth**.
Ray CA.
Bioanalysis. 2014 Feb;6(3):269-71. doi: 10.4155/bio.13.322. No abstract available.
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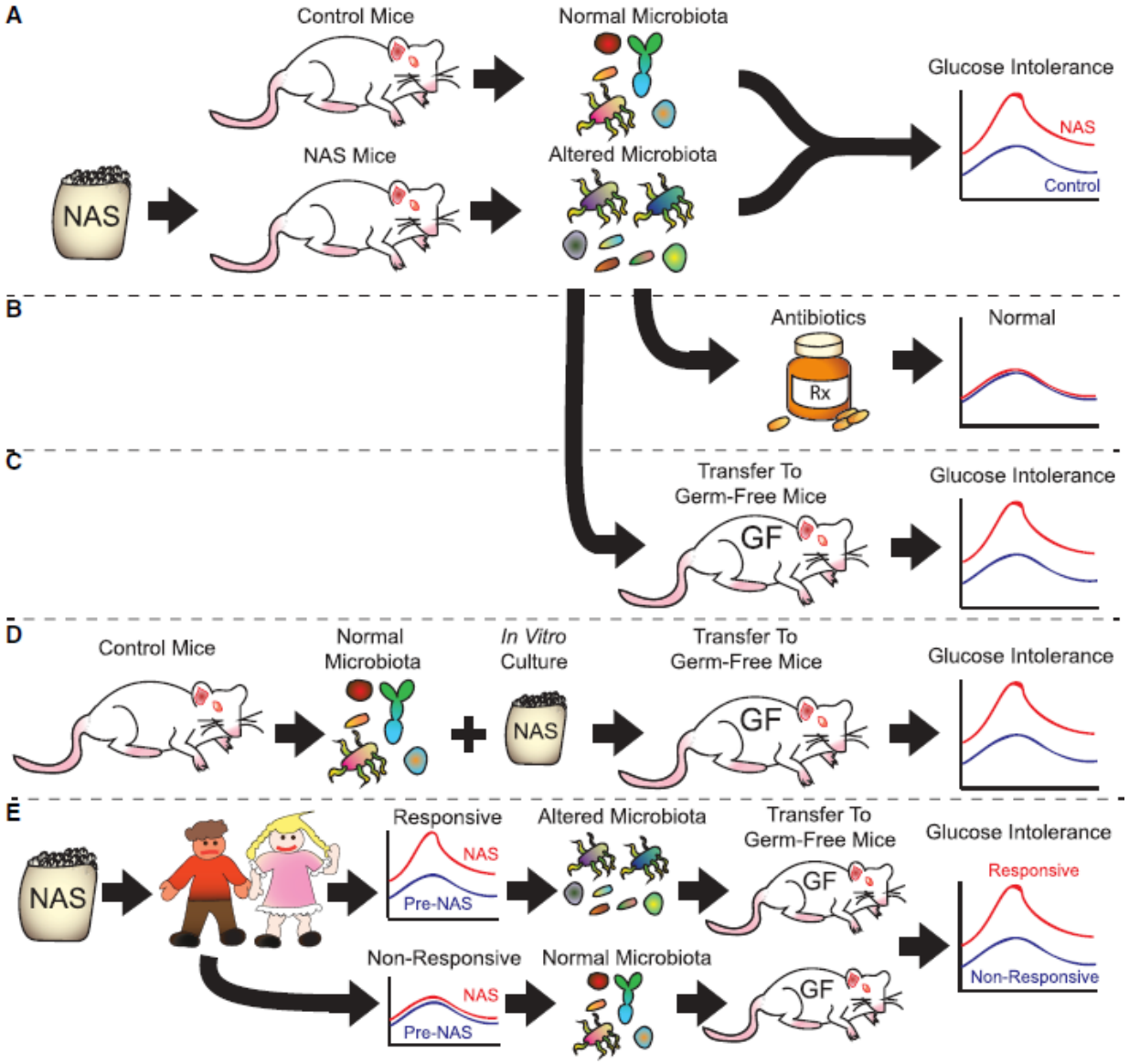
Tell the **truth**, but be gentle.

Prevencia nesprávnej prevencie



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15 January 2014 Last updated at 16:58 GMT

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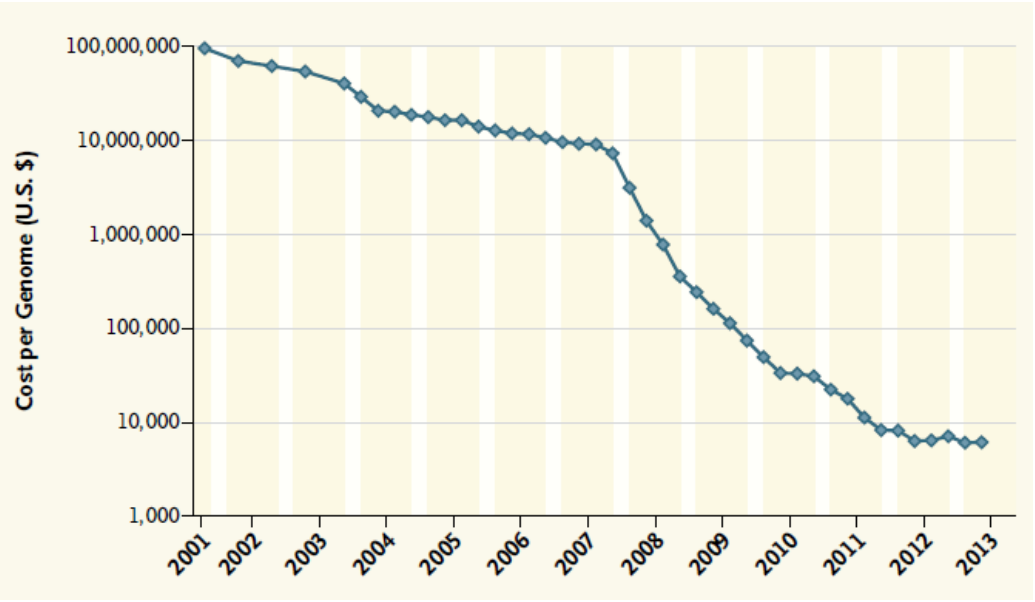

Dennis Lo

THE LANCET

Early report

Presence of fetal DNA in maternal plasma and serum

Y M Dennis Lo, Noemi Corbetta, Paul F Chamberlain, Vik Rai, Ian L Sargent, Christopher W G Redman, James S Wainscoat

- Fetus is similar to a tumor
- Non-invasive prenatal diagnosis
  - Sex
  - RhD
  - Aneuploidies
  - Fetal genome

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PLoS one

The Sorcerer II Global Ocean Sampling Expedition: Metagenomic Characterization of Viruses within Aquatic Microbial Samples

Shannon J. Williamson[1]*, Douglas B. Rusch[1], Shibu Yooseph[1], Aaron L. Halpern[1], Karla B. Heidelberg[1,2], John I. Glass[1], Cynthia Andrews-Pfannkoch[1], Douglas Fadrosh[1], Christopher S. Miller[3], Granger Sutton[1], Marvin Frazier[1], J. Craig Venter[1]

1 J. Craig Venter Institute, Rockville, Maryland, United States of America, 2 University of Southern California, Los Angeles, California, United States of America, 3 Molecular Biology Institute, University of California at Los Angeles, Los Angeles, California, United States of America

Viruses are the most abundant biological entities on our planet. Interactions between viruses and their hosts impact several important biological processes in the world's oceans such as horizontal gene transfer, microbial diversity and biogeochemical cycling. Interrogation of microbial metagenomic sequence data collected as part of the Sorcerer II Global Ocean Expedition (GOS) revealed a high abundance of viral sequences, representing approximately 3% of the total predicted proteins. Cluster analyses of the viral sequences revealed hundreds to thousands of viral genes encoding various metabolic and cellular functions. Quantitative analyses of viral genes of host origin performed on the viral fraction of aquatic samples confirmed the viral nature of these sequences and suggested that significant portions of aquatic viral communities behave as reservoirs of such genetic material. Distributional and phylogenetic analyses of these host-derived viral sequences also suggested that viral acquisition of environmentally relevant genes of host origin is a more abundant and widespread phenomenon than previously appreciated. The predominant viral sequences identified within microbial fractions originated from tailed bacteriophages and exhibited varying global distributions according to viral family. Recruitment of GOS viral sequence fragments against 27 complete aquatic viral genomes revealed that only one reference bacteriophage genome was highly abundant and was closely related, but not identical, to the cyanomyovirus P-SSM4. The co-distribution across all sampling sites of P-SSM4-like sequences

Syntetická biológia

Generating a synthetic genome by whole genome assembly: ϕX174 bacteriophage from synthetic oligonucleotides

Hamilton O. Smith, Clyde A. Hutchison III†, Cynthia Pfannkoch, and J. Craig Venter‡

Institute for Biological Energy Alternatives, 1901 Research Boulevard, Suite 600, Rockville, MD 20850

Contributed by J. Craig Venter, November 3, 2003

We have improved upon the methodology and dramatically shortened the time required for accurate assembly of 5- to 6-kb segments of DNA from synthetic oligonucleotides. As a test of this methodology, we have established conditions for the rapid (14-day) assembly of the complete infectious genome of bacteriophage ϕX174 (5,386 bp) from a single pool of chemically synthesized oligonucleotides. The procedure involves three key steps: (*i*) gel purification of pooled oligonucleotides to reduce contamination

truncated species. Although
useful as primers for PCR ar
only small (a few hundred
generally be accurately and di
repair/selection steps. For ex
assembly of a partially active
segments of DNA from which
could be transcribed was quit

RESEARCH ARTICLE

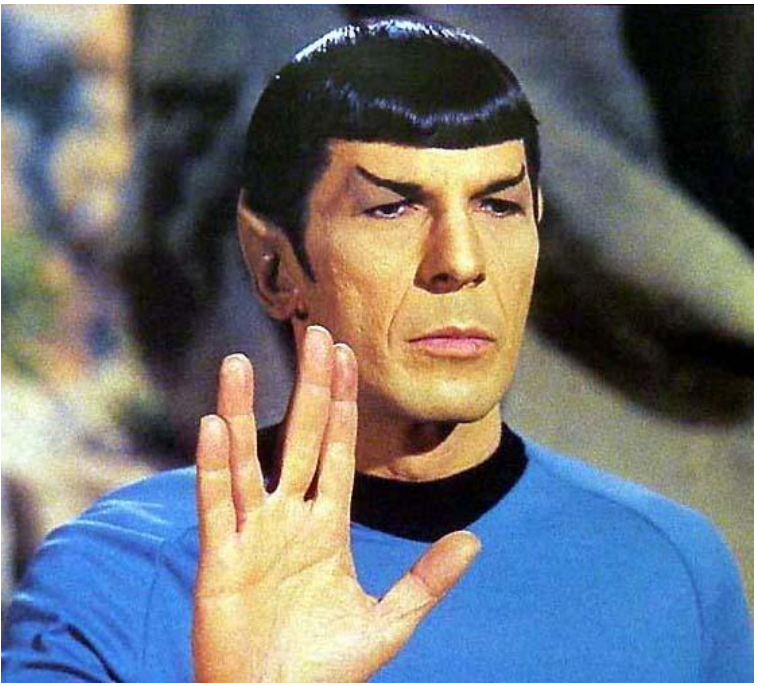
Complete Chemical Synthesis, Assembly, and Cloning of a *Mycoplasma genitalium* Genome

..., Evgeniya A. Denisova,
...wnley, David W. Thomas,
...I. Glass, J. Craig Venter,

...me. This synthetic genome,
...*enitalium* G37 except
...nicity and to allow for
..." at intergenic sites known
...bases (kb), assembled from
...ination to produce
..., and 144 kb ("1/4
Escherichia coli. Most of
...enomes with the correct
...ed by transformation-
...e, then isolated and
...ods described here will be
...ynthesized pieces and also

Creation of a Bacterial Cell Controlled by a Chemically Synthesized Genome

Génová terapia už nie je sci-fi



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2 November 2012 Last updated at 11:00 GMT

Gene therapy: Glybera approved by European Commission

European Medicines Agency recommends first gene therapy for approval

EUROPEAN MEDICINES AGENCY
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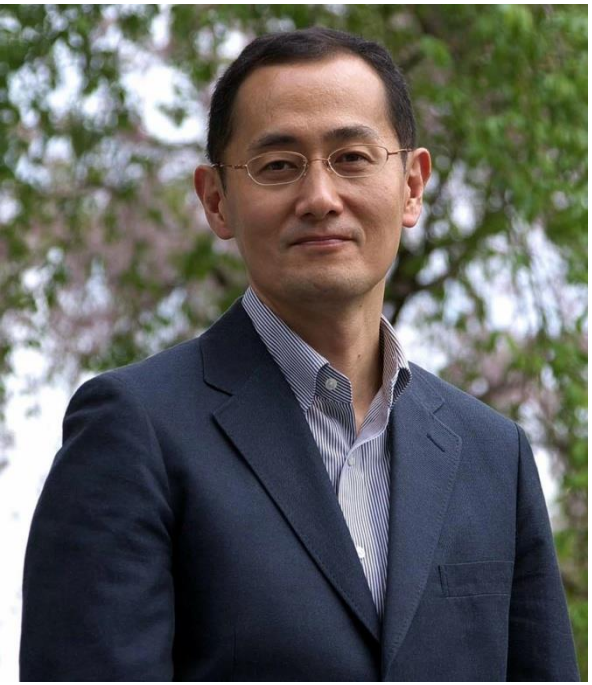
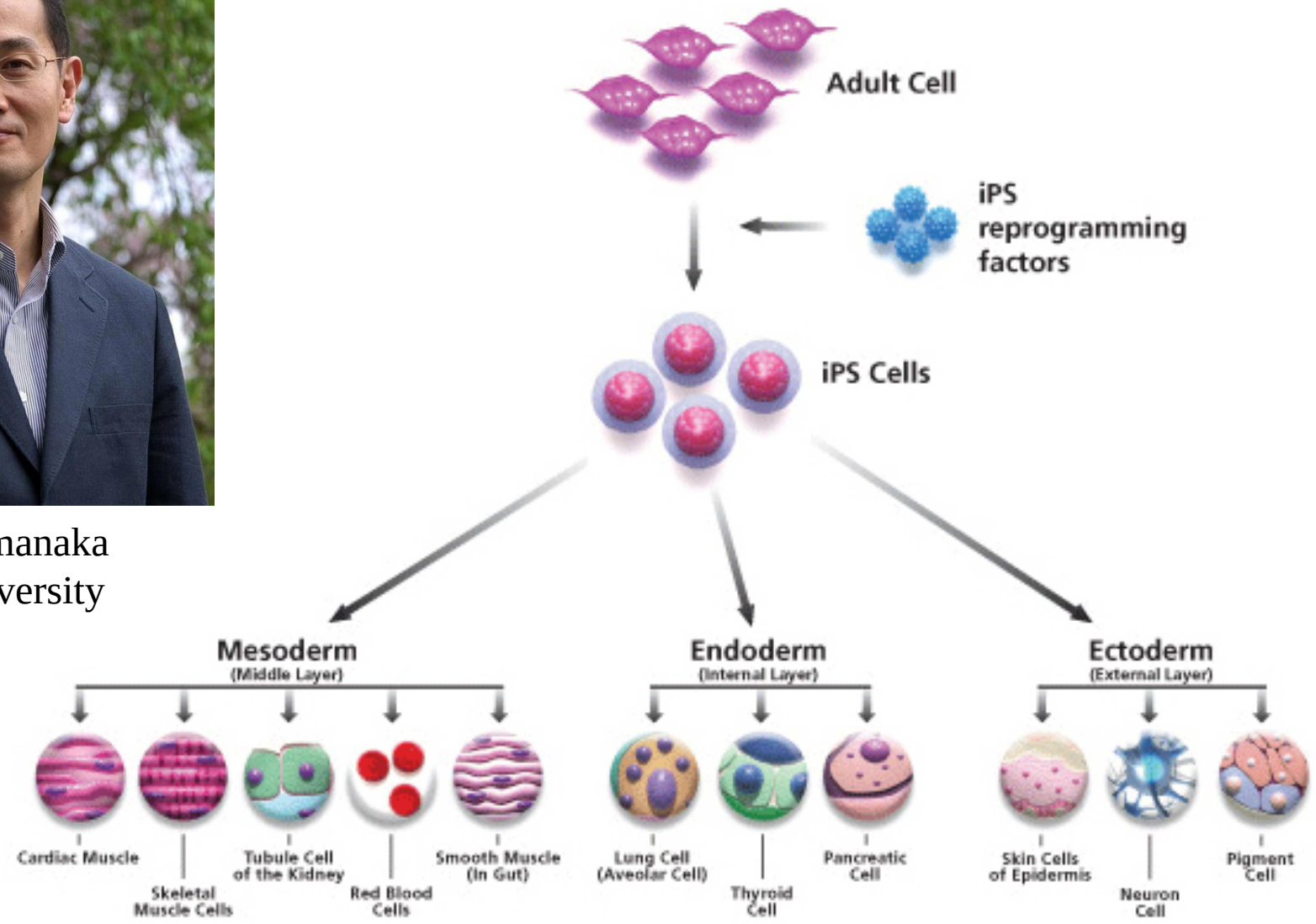


Shinya Yamanaka
Kyoto University



Personalizovaná medicína

NAME	CONFIDENCE	STATUS
Hemochromatosis (HFE-related)	★★★★	Variant Present
Familial Hypercholesterolemia Type B	★★★★	Variant Present
Phenylketonuria	★★★★	Variant Absent
Familial Dysautonomia	★★★★	Variant Absent
Canavan Disease	★★★★	Variant Absent

Usher Syndrome Type III	★★★★	Variant Absent
TTR-Related Familial Amyloid Polyneuropathy	★★★★	Variant Absent
Pendred Syndrome	★★★★	Variant Absent
Tyrosinemia Type I	★★★★	Variant Absent
Hereditary Fructose Intolerance	★★★★	Variant Absent

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NAME	CONFIDENCE	STATUS
Hemochromatosis (HFE-related)	★★★★	Variant Present
Familial Hypercholesterolemia Type B	★★★★	Variant Present

Congenital Disorder of Glycosylation Type 1a (PMM2-CDG)	★★★★	Variant Absent
DPD Deficiency	★★★★	Variant Absent
Dihydrolipoamide Dehydrogenase Deficiency	★★★★	Variant Absent
Neuronal Ceroid Lipofuscinosis (PPT1-related)	★★★★	Variant Absent
Medium-Chain Acyl-CoA Dehydrogenase (MCAD) Deficiency	★★★★	Variant Absent
Glycogen Storage Disease Type 1a	★★★★	Variant Absent
Glycogen Storage Disease Type 1b	★★★★	Variant Absent
Gaucher Disease	★★★★	Variant Absent
ARSACS	★★★★	Variant Absent
G6PD Deficiency	★★★★	Variant Absent
Cystic Fibrosis	★★★★	Variant Absent
Factor XI Deficiency	★★★★	Variant Absent
Zellweger Syndrome Spectrum	★★★★	Variant Absent
Nijmegen Breakage Syndrome	★★★★	Variant Absent
D-Bifunctional Protein Deficiency	★★★★	Variant Absent
Usher Syndrome Type I (PCDH15-related)	★★★★	Variant Absent
LAMB3-related Junctional Epidermolysis Bullosa	★★★★	Variant Absent
Familial Mediterranean Fever	★★★★	Variant Absent

Maple Syrup Urine Disease Type 1B	★★★★	Variant Absent
Tay-Sachs Disease	★★★★	Variant Absent
Agenesis of the Corpus Callosum with Peripheral Neuropathy (ACCPN)	★★★★	Variant Absent
Neuronal Ceroid Lipofuscinosis (CLN5-related)	★★★★	Variant Absent

Decreased Risk

NAME	CONFIDENCE	YOUR RISK	AVG. RISK	COMPARED TO AVERAGE
Gout	★★★★	17.1%	22.8%	0.75x
Age-related Macular Degeneration	★★★★	4.4%	6.5%	0.67x
Alzheimer's Disease	★★★★	4.3%	7.2%	0.60x
Psoriasis	★★★★	4.1%	11.4%	0.36x
Rheumatoid Arthritis	★★★★	1.2%	2.4%	0.51x
Parkinson's Disease	★★★★	0.94%	1.61%	0.58x
Restless Legs Syndrome	★★★★	0.86%	1.96%	0.44x
Melanoma	★★★★	0.74%	2.86%	0.26x
Ulcerative Colitis	★★★★	0.46%	0.77%	0.59x
Multiple Sclerosis	★★★★	0.24%	0.34%	0.69x
Exfoliation Glaucoma	★★★★	0.16%	0.75%	0.22x
Crohn's Disease	★★★★	0.13%	0.53%	0.24x
Type 1 Diabetes	★★★★	0.12%	1.02%	0.12x
Celiac Disease	★★★★	0.07%	0.12%	0.58x
Primary Biliary Cirrhosis	★★★★	0.04%	0.08%	0.48x

Elevated Risk

NAME	CONFIDENCE	YOUR RISK	AVG. RISK	COMPARED TO AVERAGE
Atrial Fibrillation	★★★★	**41.6%**	27.2%	1.53x
Prostate Cancer ♂	★★★★	**29.0%**	17.8%	1.63x
Venous Thromboembolism	★★★★	**17.9%**	12.3%	1.45x
Colorectal Cancer	★★★★	**8.0%**	5.6%	1.44x
Esophageal Squamous Cell Carcinoma (ESCC)	★★★★	**0.43%**	0.36%	1.21x
Stomach Cancer (Gastric Cardia Adenocarcinoma)	★★★★	**0.28%**	0.23%	1.22x
Bipolar Disorder	★★★★	**0.15%**	0.10%	1.44x

NAME	CONFIDENCE	STATUS
Proton Pump Inhibitor (PPI) Metabolism	★★★★	Rapid
Warfarin (Coumadin®) Sensitivity	★★★★	Increased
Sulfonylurea Drug Clearance (Type 2 Diabetes Treatment)	★★★★	Reduced
Phenytoin (Dilantin®) Sensitivity (Epilepsy Drug)	★★★★	Increased
Fluorouracil Toxicity	★★★★	Typical

23andme

23andMe provides ancestry-related genetic reports and raw genetic data. At this time we do not offer health-related genetic reports. If you are a current customer please go to the health page for more information. Close alert

Department of Health and Human Services

Public Health Service
Food and Drug Administration
10903 New Hampshire Avenue
Silver Spring, MD 20993

Nov 22, 2013

Ann Wojcicki
CEO
23andMe, Inc.
1390 Shoreline Way
Mountain View, CA 94043

Document Number: GEN1300666
Re: Personal Genome Service (PGS)

WARNING LETTER

23andme

Should the FDA stop you from scaring yourself with 23andMe's DNA test?

BY EZRA KLEIN December 6, 2013 at 9:19 am

FDA identifies the idiot gene: 23andMe users

Published: Sunday, 1 Dec 2013 | 7:00 AM ET

The FDA vs. 23andMe: A Lesson for Health Care Entrepreneurs

New evidence shows the FDA was wrong to halt 23andMe testing

@qz

Regulating 23andMe to Death Won't Stop the New Age of Genetic Testing

By Larry Downes and Paul Nunes

Radšej nevedieť?

Preventívne rozhodnutie

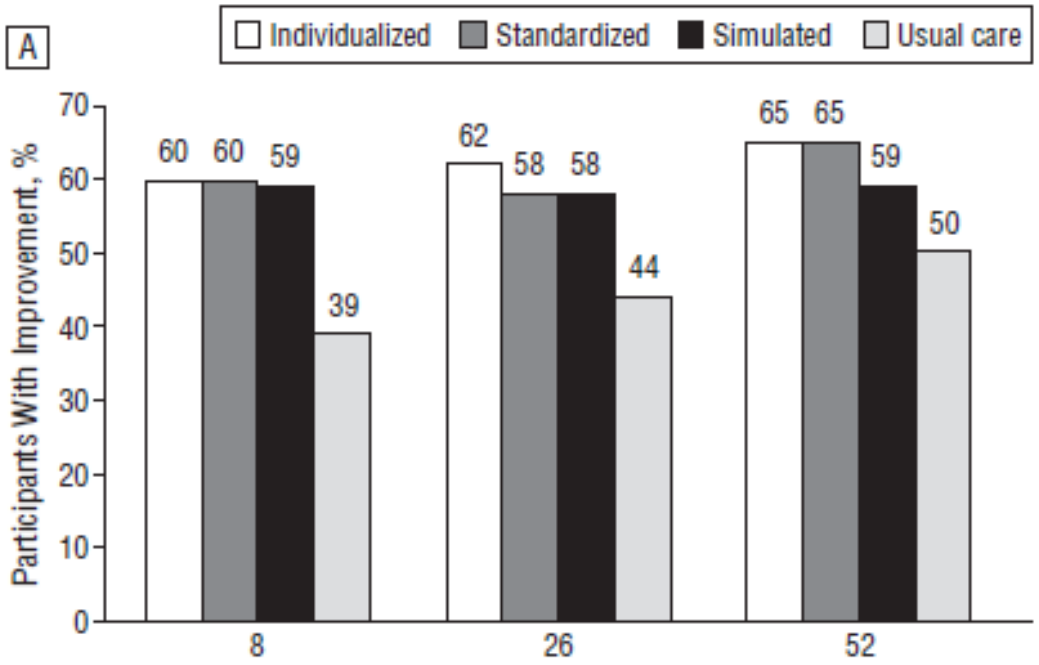
Lekári majú strach z nesprávnych interpretácií

Cholesterol? Tlakomery?

Strach pramení z neznalosti

A Randomized Trial Comparing Acupuncture, Simulated Acupuncture, and Usual Care for Chronic Low Back Pain

Daniel C. Cherkin, PhD; Karen J. Sherman, PhD; Andrew L. Avins, MD, MPH; Janet H. Erro, RN, MN; Laura Ichikawa, MS; William E. Barlow, PhD; Kristin Delaney, MPH; Rene Hawkes, BA; Luisa Hamilton, MD; Alice Pressman, MS; Partap S. Khalsa, DC, PhD; Richard A. Deyo, MD, MPH



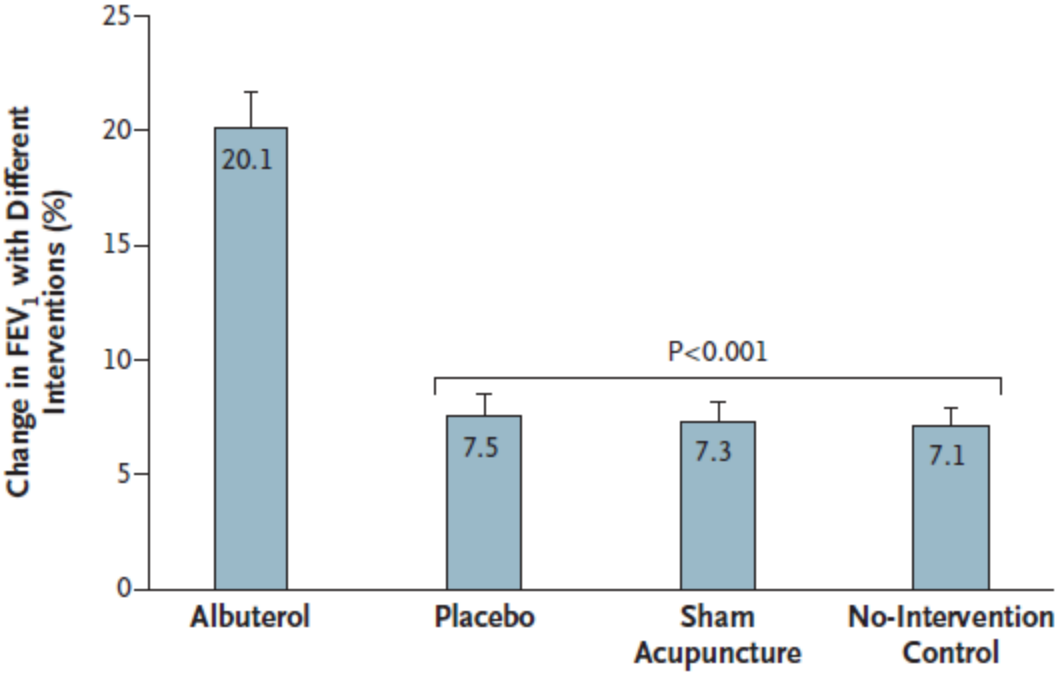
The NEW ENGLAND JOURNAL of MEDICINE

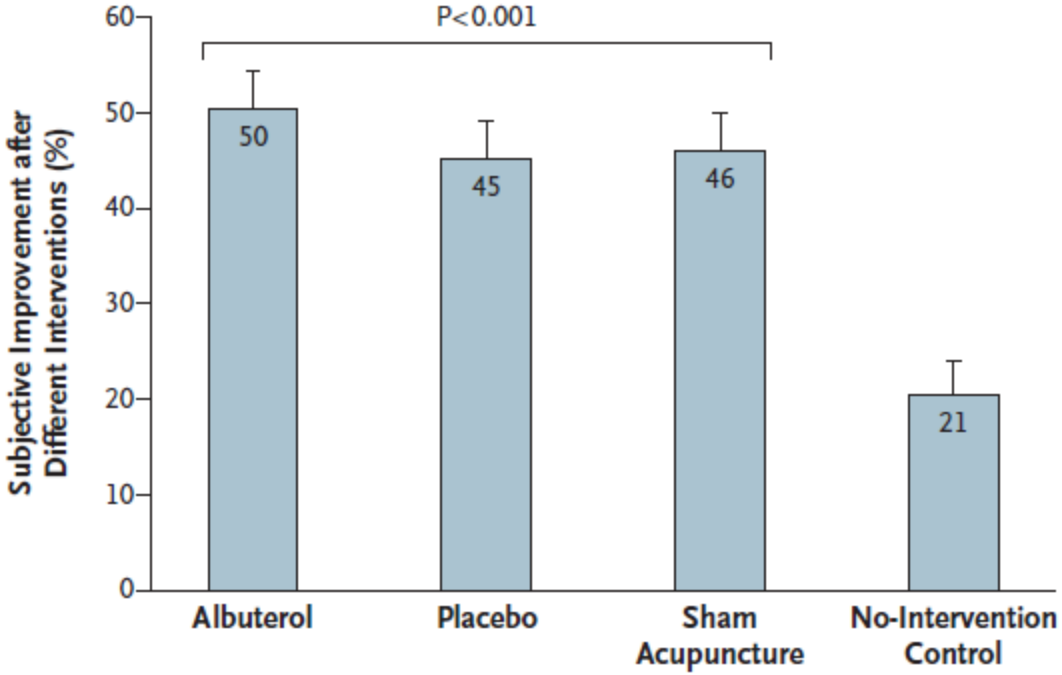
ORIGINAL ARTICLE

Active Albuterol or Placebo, Sham Acupuncture, or No Intervention in Asthma

Michael E. Wechsler, M.D., John M. Kelley, Ph.D., Ingrid O.E. Boyd, M.P.H., Stefanie Dutile, B.S., Gautham Marigowda, M.B., Irving Kirsch, Ph.D., Elliot Israel, M.D., and Ted J. Kaptchuk

The New England Journal of Medicine


Change in FEV_1 with Different Interventions (%)
25
20
15
10
5
0
20.1
P<0.001
7.5
7.3
7.1
Albuterol
Placebo
Sham Acupuncture
No-Intervention Control


Subjective Improvement after Different Interventions (%)
60
50
40
30
20
10
0
P<0.001
50
45
46
21
Albuterol
Placebo
Sham Acupuncture
No-Intervention Control

Rebecca L. Waber, BS
Massachusetts Institute of Technology
Cambridge, Massachusetts
Baba Shiv, PhD
Stanford University
Stanford, California
Ziv Carmon, PhD
INSEAD
Singapore
Dan Ariely, PhD
ariely@mit.edu
Massachusetts Institute of Technology

RESEARCH LETTER

Commercial Features of Placebo and Therapeutic Efficacy

Table. Comparison of Participants Assigned to Regular-Price Placebo vs Low-Price (Discounted) Placebo

	Regular Price (n = 41)	Low Price (n = 41)	*P* Value
Women, No. (%)	27 (65.9)	24 (58.5)	.50
Age, mean (SD), y	30.9 (12.4)	30.0 (11.4)	.74
Calibrated maximum tolerance, mean (SD), V	51.8 (18.7)	54.9 (23.3)	.50
Shocks received, No. (SD)	18.2 (7.2)	18.6 (9.1)	.80
Change in pain scores[a]			
All shocks, No. (%) [95% CI]			
Pain reduction	35 (85.4) [74.6-96.2]	25 (61.0) [46.1-75.9]	.02[b]
Pain increase	6 (14.6) [3.8-25.5]	16 (39.0) [24.1-54.0]	

populations.[4] Substantial evidence indicates that when the value of improved quality of life and productivity are considered, investments in health technology offer great returns.[5] Assessing medical needs, building capacity, and promoting cost-effective innovation will help in realizing technology's potential for achieving better health in low-resource settings.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

From the Biodesign Program (S.R.S), the Division of Gastroenterology and Hepatology (S.R.S.), Department of Medicine (S.R.S., M.B), and the Center for Innovation in Global Health (M.B.), Stanford University, Stanford, CA.

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The Art of Doing Nothing

Lisa Rosenbaum, M.D.

N ENGL J MED 365;9 NEJM.ORG SEPTEMBER 1, 2011

The New England Journal of Medicine

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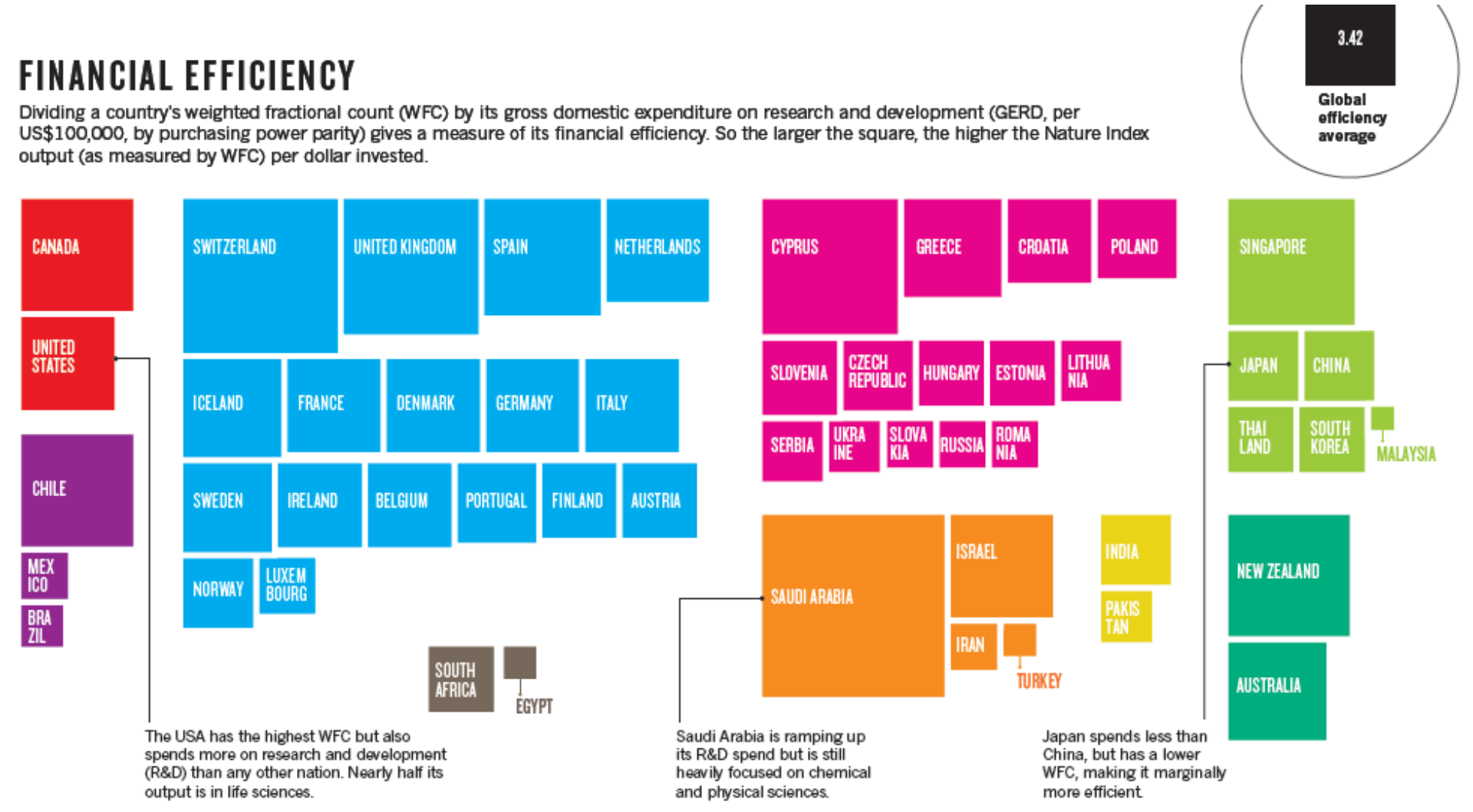

petercelec@gmail.com