Bale/Doneen Live Chat Session

Amy Doneen MSN, ARNP

April 10, 2013

5:30-6:30 pm PST

Puerto Vallarta
April 4, 2013

Bibliography for today – 4/10/2013 1 of 2

- Daniels, L., Grady, D., et al. Circ Cardiovasc Qual Outcomes March 12 2013;6;164-170

- Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

- Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology. April 2013; 80:1300-1306.

- Hermann, D., Gronewold, J., et al. Stroke. March 25, 2013;44:1008-1013

- Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

Bibliography for today – 4/10/2013 2 of 2

- Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea JAMA March 13, 213, Vol. 309. No 10. 997-1004

- Kokubo, Y et al. Impact of Green Tea and Coffee and Stroke Incidence. Stroke. AHA Published Online March 14, 2013, AHA ISSN: 0039-2499. Online ISSN: 1524—4628

- Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

- Koeth, R., Hazen, S., et al. Intestinal microbiota metabolism of L-carnitine, a nutrient red meat, promotes atherosclerosis. Nature Medicine. April 7, 2013. doi 10.1038.

- Rassmussen-Torvik, L., Shay, C., et al., Circulation March 18, 2013 ISSN 1524-4539

Red Flags

DM and CHD in women

RUTH trial – international, multicenter, double-blind, randomized, placebo-controlled Trial of raloxifene and CVD outcomes in 10 101 postmenopausal women selected for high CHD risk.

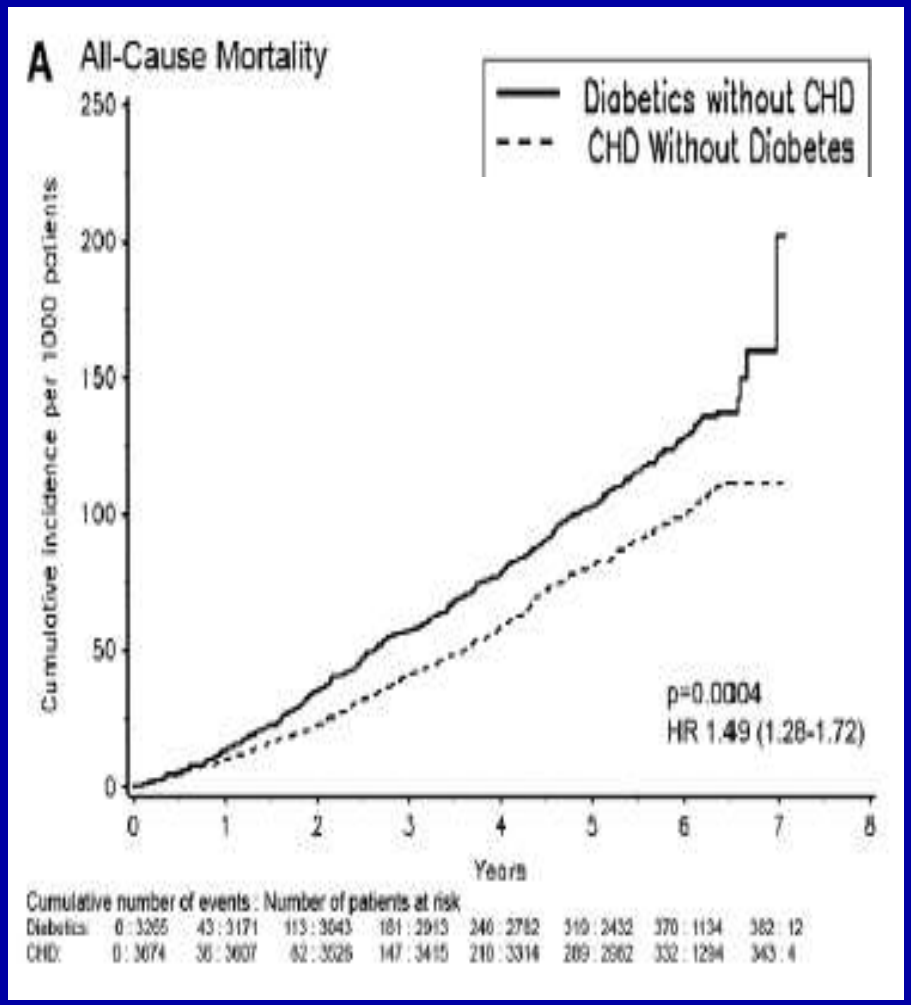
3672 had a history of diabetes without known CHD
3265 had history of CHD without known diabetes.

Mean age 67.5 years – follow-up was 5.6 years.

Goal: Compare the two groups for CHD and CVD fatal and non-fatal.

Daniels, L., Grady, D., et al. Circ Cardiovasc Qual Outcomes March 12 2013;6:164-170

DM and CHD in Women



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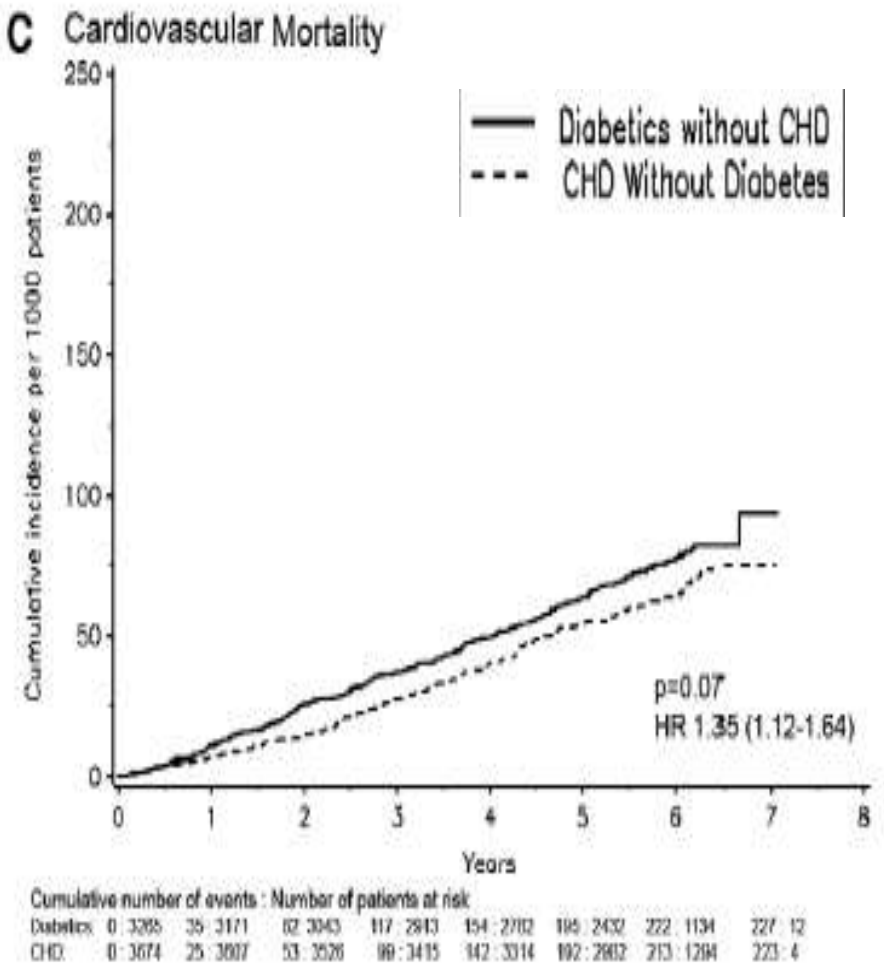
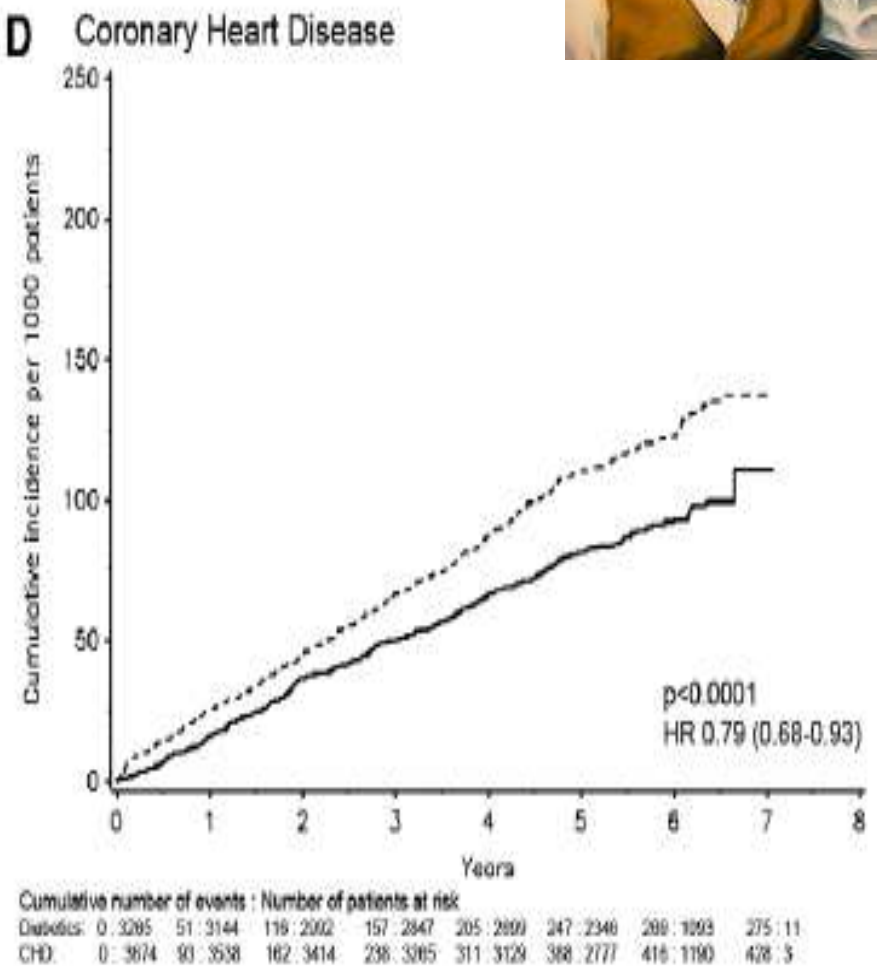
DM and CHD in Women





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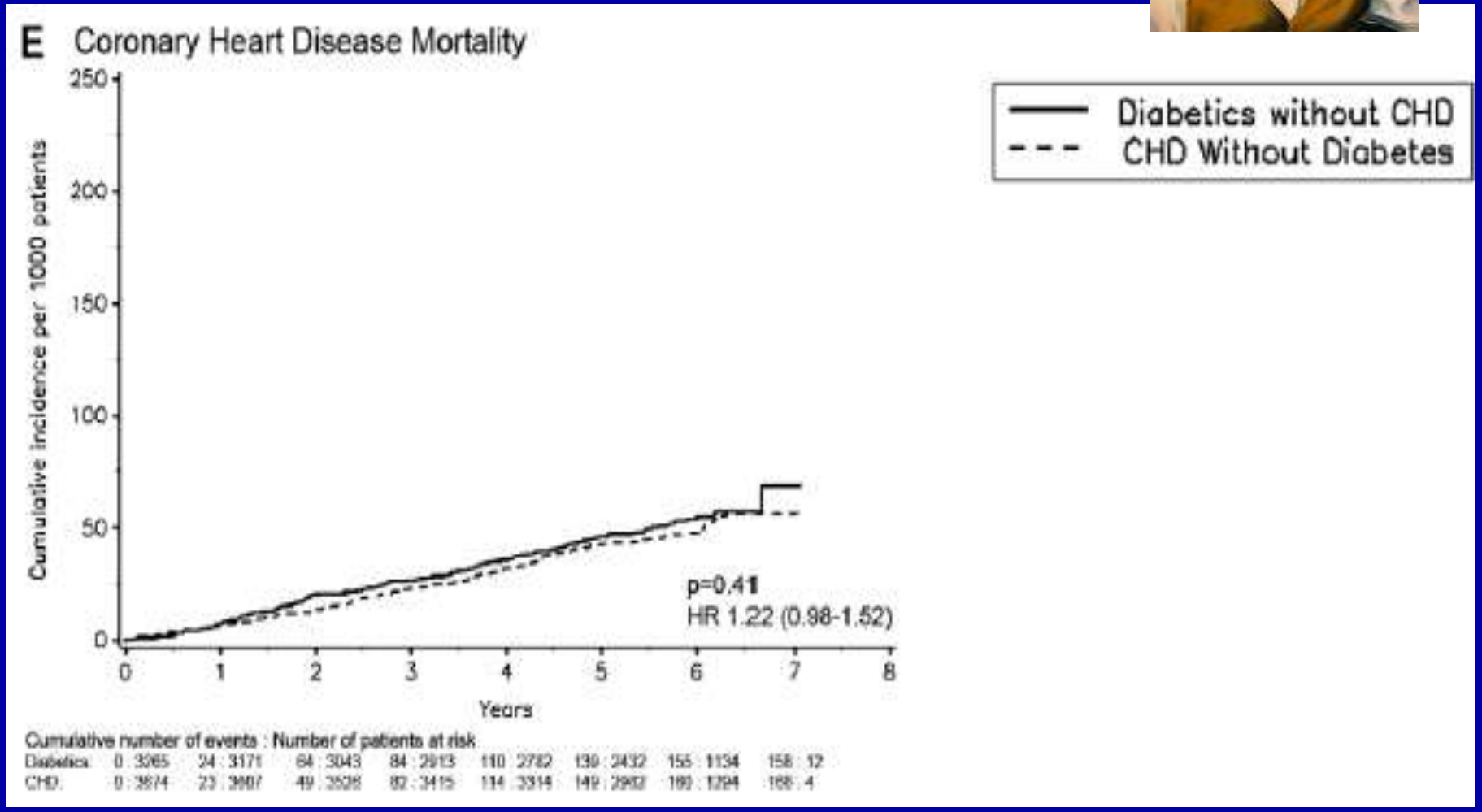
DM and CHD in Women



Daniels, L., Grady, D., et al. Circ Cardiovasc Qual Outcomes March 12 2013;6;164-170

DM and CHD in Women

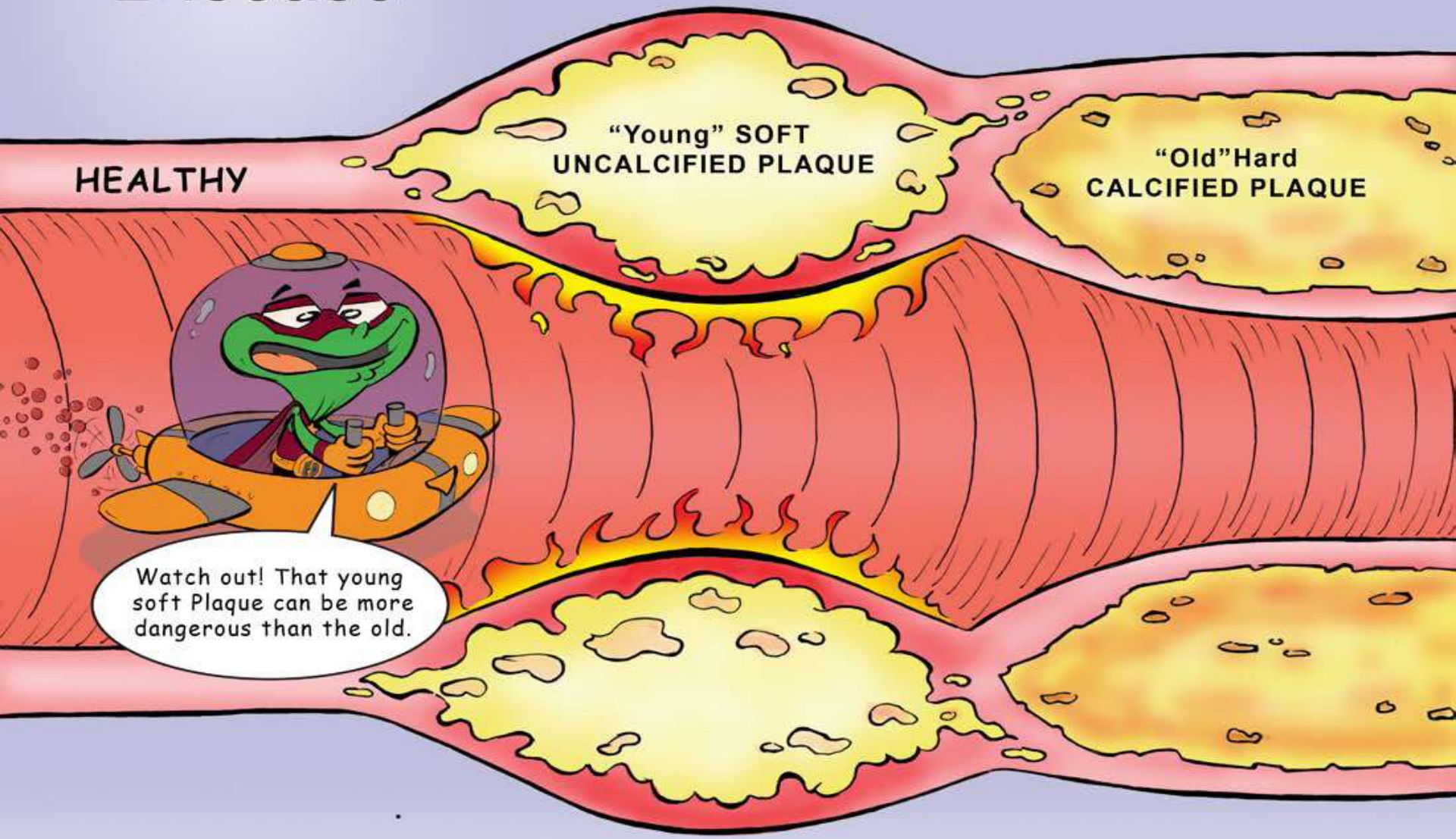
Diabetic women without “known” CHD had a lower risk of nonfatal CHD and CVD Events compared with non diabetic women with CHD but their risk of CV Death and all-cause mortality was similar.

BD Take-Away:

1. Women with DM may have a more severe or delayed presentation of CHD and CVD, small-vessel disease, odd clinical presentations….
2. Follow a Disease/Inflammatory Paradigm –Always!

Daniels, L., Grady, D., et al. Circ Cardiovasc Qual Outcomes March 12 2013;6;164-170

Disease



Plaque formation is an active process and its consistency changes over time. Some technologies (X-Rays) can only see hard calcified disease while others like ultrasounds can spot soft disease.

Dale Doneen METHOD

Moss Freedman

Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service Members, 2001-2011

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

Autopsies of US service members killed in battle.

First demonstrated in Korean was in 1953 (Enos et al), demonstrated fatty streaks in 77% of service men.

Cross sectional study of all US service members who died of combat or unintentional injuries in operations Enduring Freedom and Iraqi Freedom/New Dawn between Oct 2001 and Aug 2011.

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

Table 2. Prevalence of Coronary and/or Aortic Atherosclerosis by *ICD-9-CM* Diagnoses of Major Cardiovascular Risk Factors

ICD-9-CM Diagnosis	No. of Patients With Atherosclerosis/Total No.	Atherosclerosis Prevalence, % (95% CI)	Prevalence Ratio (95% CI)	
			Unadjusted	Age-Adjusted
None	389/3506	11.1 (10.1-12.1)	1 [Reference]	1 [Reference]
Obesity	37/166	22.3 (15.9-28.7)	2.01 (1.49-2.71)	1.47 (1.10-1.96)
Smoking	18/128	14.1 (8.0-20.2)	1.27 (0.82-1.96)	1.12 (0.73-1.74)
Hypertension	17/39	43.6 (27.3-59.9)	3.93 (2.72-5.68)	1.88 (1.34-2.65)
Dyslipidemia	14/28	50.0 (30.3-69.7)	4.51 (3.08-6.60)	2.09 (1.43-3.06)
Diabetes/IFG	2/10	20.0 (0.0-50.2)	1.80 (0.52-6.25)	0.58 (0.17-1.97)
Obesity and hypertension	4/8	50.0 (5.3-94.7)	4.51 (2.24-9.07)[a]	3.14 (1.54-6.44)[a]

Abbreviations: *ICD-9-CM*, *International Classification of Diseases, Ninth Revision, Clinical Modification*; IFG, impaired fasting glucose.
[a]Reflects odds ratio.

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

Table 3. Coronary Autopsy Studies in the US Armed Forces: Korean War, Vietnam War, and OEF and OIF/OND

	Korean War[5,21]	Vietnam War[7]	OEF and OIF/OND
US deaths, No.	36 574[a]	58 220[a]	6191[b]
Autopsies included in the study, No. (% of total deaths)	300 (0.8)	105 (0.2)	3832 (61.9)
Mean (SD) age, y	22.1 (NR)	22.1 (4.4)	25.9 (6.6)
Race, % white	NR	86.7	72.7
Coronary lesion grading			
Minimal	Fibrous thickening or streaking causing insignificant narrowing	Fibrous thickening or single plaques <5 mm in greatest diameter	Streaking causing insignificant luminal narrowing
Moderate	10%-49% narrowing	Single plaques <1 cm	10%-49% narrowing
Severe	≥50% narrowing	Single plaques >1 cm or confluent smaller plaques	≥50% narrowing
Prevalence of coronary atherosclerosis, %			
Minimal	35	NR	1.5
Moderate	27	NR	4.7
Severe	15	5	2.3
Any	77	45	8.5

Abbreviations: OEF, Operation Enduring Freedom; OIF, Operation Iraqi Freedom; OND, Operation New Dawn; NR, not reported.
[a] Per Congressional Research Service.[22]
[b] During case identification period of October 2001 through August 2011, per Armed Forces Medical Examiner System.

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

Results summary – sign. Assoc. risk factors:

Age: strongest association with atherosclerosis

40 years and older had a 7 times increased prevalence of disease compared with those aged 24 years and younger (45.9% vs 6.6%, unadjusted PR, 6.95; 95% CI, 5.49-8.80).

Lower Education level & **higher military entrance**

Higher BMI obese 15.8% vs 7.6% [95% CI, 1.35-2.60]

HTN: (43.6% vs 11.1% [95% CI, 1.30-1.65]

After Adjusting for age: only obesity and HTN remained significant for association.

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

BD Take-Away

Of the 464 service members with atherosclerosis at autopsy, only 2 (0.5%) had been clinically diagnosed with atherosclerosis and none had been diagnosed with ischemic heart disease.

Identify patients as Primary, Secondary, Tertiary!

Promote Wellness within all settings – including military.

Webber, B., Seguin, P., et al. Prevalence of and Risk Factors for Autopsy-Determined Atherosclerosis Among US Service members, 2001-2011. JAMA December 26, 2012. Vol. 308, No 24. 2577-2583

Predicting Cognitive Decline with Framingham Risk Scoring

Predicting Cognitive Decline with Framingham Risk Scoring

Comparing 2 FRS scores with dementia risk score (CAIDE) in relation to 10-year cognitive decline in late middle age.

Men and women mean age 55.6 at baseline – Whitehall II study (longitudinal British cohort study).

Cognitive tests included reasoning, memory, verbal fluency, vocabulary, and global cognition, assessed 3 times over 10 years.

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

Assessing cognitive function

Assessed 3 times over 10 years –

5 standard cognitive tasks:

1. Alice Heim 4-I (reasoning, identify patterns, infer principles)
2. Short-term verbal memory with 20 word recall test
3. Two measures of verbal fluency: phonemic and semantic
4. Vocabulary assessed with Mill Hill Vocabulary Test
5. Global Cognitive Score using all 5 tests above.

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

Comparing the cognitive score with FRS:

Two analytic samples taken:

First: comparison of FRS CVD risk score with the dementia risk score based on participants free of CVD at baseline with data on all components of risk scores

Second: comparison of FRS stroke risk score with the dementia risk score based on individuals without a history of stroke or TIA who had data on all components of the risk scores.

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

Table 2 Associations of dementia and CVD risk (1997/1999) with 10-year cognitive change (1997/1999, 2002/2004, 2007/2009) (n = 4,374)

	Risk groups					
	Low	Intermediate	High		Standardized risk	
Cognitive test	10-year cognitive change (95% CI)			p	β (95% CI)	Δ (95% CI)[a]
Reasoning						
Dementia risk	−0.28 (−0.31, −0.26)	−0.35 (−0.38, −0.32)	−0.36 (−0.39, −0.33)	<0.001	−0.05 (−0.06, −0.03)[c]	
CVD risk	−0.26 (−0.29, −0.23)	−0.31 (−0.34, −0.28)	−0.41 (−0.44, −0.38)	<0.001	−0.06 (−0.08, −0.04)[c]	0.01 (−0.004, 0.03), NS
Memory						
Dementia risk	−0.24 (−0.28, −0.19)	−0.27 (−0.33, −0.22)	−0.26 (−0.32, −0.19)	0.46	−0.01 (−0.04, 0.01)	
CVD risk	−0.20 (−0.25, −0.15)	−0.29 (−0.34, −0.24)	−0.27 (−0.32, −0.21)	0.09	−0.03 (−0.06, 0.00)	0.02 (−0.02, 0.06), NS
Phonemic fluency						
Dementia risk	−0.34 (−0.38, −0.31)	−0.37 (−0.42, −0.33)	−0.36 (−0.41, −0.31)	0.42	−0.01 (−0.04, 0.01)	
CVD risk	−0.31 (−0.35, −0.27)	−0.36 (−0.40, −0.32)	−0.39 (−0.44, −0.35)	0.01	−0.03 (−0.06, −0.01)[b]	0.02 (−0.005, 0.05), NS
Semantic fluency						
Dementia risk	−0.29 (−0.33, −0.26)	−0.32 (−0.37, −0.28)	−0.29 (−0.35, −0.24)	0.85	0.001 (−0.02, 0.02)	
CVD risk	−0.31 (−0.35, −0.27)	−0.36 (−0.40, −0.32)	−0.39 (−0.44, −0.35)	<0.001	−0.05 (−0.07, −0.02)[c]	0.05 (0.02, 0.08)
Vocabulary						
Dementia risk	0.05 (0.03, 0.07)	0.004 (−0.02, 0.03)	−0.02 (−0.05, 0.01)	<0.001	−0.02 (−0.04, −0.01)[b]	
CVD risk	0.05 (0.03, 0.08)	0.03 (0.002, 0.05)	−0.02 (−0.05, 0.001)	<0.0001	−0.04 (−0.05, −0.03)[c]	0.02 (−0.004, 0.04), NS
Global cognition						
Dementia risk	−0.31 (−0.33, −0.28)	−0.36 (−0.39, −0.34)	−0.35 (−0.39, −0.32)	0.01	−0.03 (−0.04, −0.01)[b]	
CVD risk	−0.26 (−0.28, −0.23)	−0.34 (−0.37, −0.32)	−0.40 (−0.43, −0.37)	<0.001	−0.06 (−0.08, −0.05)[c]	0.03 (0.01, 0.05)

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

Table 3 Associations of dementia and stroke risk (1997/1999) with 10-year cognitive change (1997/1999, 2002/2004, 2007/2009) (n = 5,157)

	Risk groups					
	Low	Intermediate	High		Standardized risk	
Cognitive test	10-year cognitive change (95% CI)			*p*	β (95% CI)	Δ (95% CI)[a]
Reasoning						
Dementia risk	−0.28 (−0.30, −0.26)	−0.35 (−0.38, −0.32)	−0.37 (−0.40, −0.33)	<0.001	−0.05 (−0.06, −0.04)[c]	
Stroke risk	−0.27 (−0.29, −0.24)	−0.34 (−0.36, −0.31)	−0.42 (−0.45, −0.38)	0.001	−0.05 (−0.06, −0.03)[c]	0.00 (−0.02, 0.02), NS
Memory						
Dementia risk	−0.24 (−0.28, −0.20)	−0.27 (−0.32, −0.22)	−0.27 (−0.33, −0.20)	0.33	−0.02 (−0.04, 0.01)	
Stroke risk	−0.24 (−0.28, −0.20)	−0.27 (−0.31, −0.22)	−0.25 (−0.32, −0.19)	0.56	−0.03 (−0.06, 0.00)	0.01 (−0.01, 0.03), NS
Phonemic fluency						
Dementia risk	−0.34 (−0.37, −0.30)	−0.37 (−0.41, −0.33)	−0.37 (−0.41, −0.31)	0.27	−0.02 (−0.04, 0.01)	
Stroke risk	−0.32 (−0.36, −0.29)	−0.36 (−0.39, −0.32)	−0.42 (−0.47, −0.37)	0.003	−0.03 (−0.06, −0.01)[b]	0.01 (−0.01, 0.04), NS
Semantic fluency						
Dementia risk	−0.29 (−0.32, −0.26)	−0.34 (−0.38, −0.30)	−0.30 (−0.35, −0.26)	0.43	−0.01 (−0.03, 0.01)	
Stroke risk	−0.26 (−0.29, −0.22)	−0.33 (−0.37, −0.29)	−0.40 (−0.44, −0.34)	<0.001	−0.05 (−0.08, −0.03)[c]	0.04 (0.02, 0.06)
Vocabulary						
Dementia risk	0.05 (0.03, 0.07)	0.006 (−0.02, 0.03)	−0.03 (−0.06, −0.002)	<0.001	−0.03 (−0.04, −0.01)[c]	
Stroke risk	0.04 (0.03, 0.07)	0.02 (−0.001, 0.04)	−0.05 (−0.08, −0.02)	<0.001	−0.04 (−0.05, −0.02)[c]	0.01 (−0.004, 0.03), NS
Global cognition						
Dementia risk	−0.22 (−0.24, −0.21)	−0.27 (−0.29, −0.25)	−0.27 (−0.29, −0.24)	<0.001	−0.02 (−0.03, −0.01)[c]	
Stroke risk	−0.21 (−0.23, −0.19)	−0.26 (−0.28, −0.24)	−0.31 (−0.34, −0.29)	<0.001	−0.04 (−0.05, −0.03)[c]	0.02 (0.01, 0.04)

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

Results:

Higher cardiovascular disease risk and higher stroke risk were associated with greater cognitive decline in ALL tests except memory.

Compared with dementia risk score, cardiovascular and stroke risk scores showed stronger associations with 10 year cognitive decline.

CVD and Stroke risk displayed stronger associations with cognitive decline than dementia risk.

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

BD Take-Away

Cardiovascular disease is a Systemic Disease

Educate patients on the value of optimal CV prevention of lifelong wellness.

Incorporate memory/dementia testing in your practice.

Author of this paper: sara.kaffashian@inserm.fr

Kaffashian, S. Dugravor, Al, et al. Predicting Cognitive Decline. Neurology April 2013; 80:1300-1306.

CACS and Stroke Risk

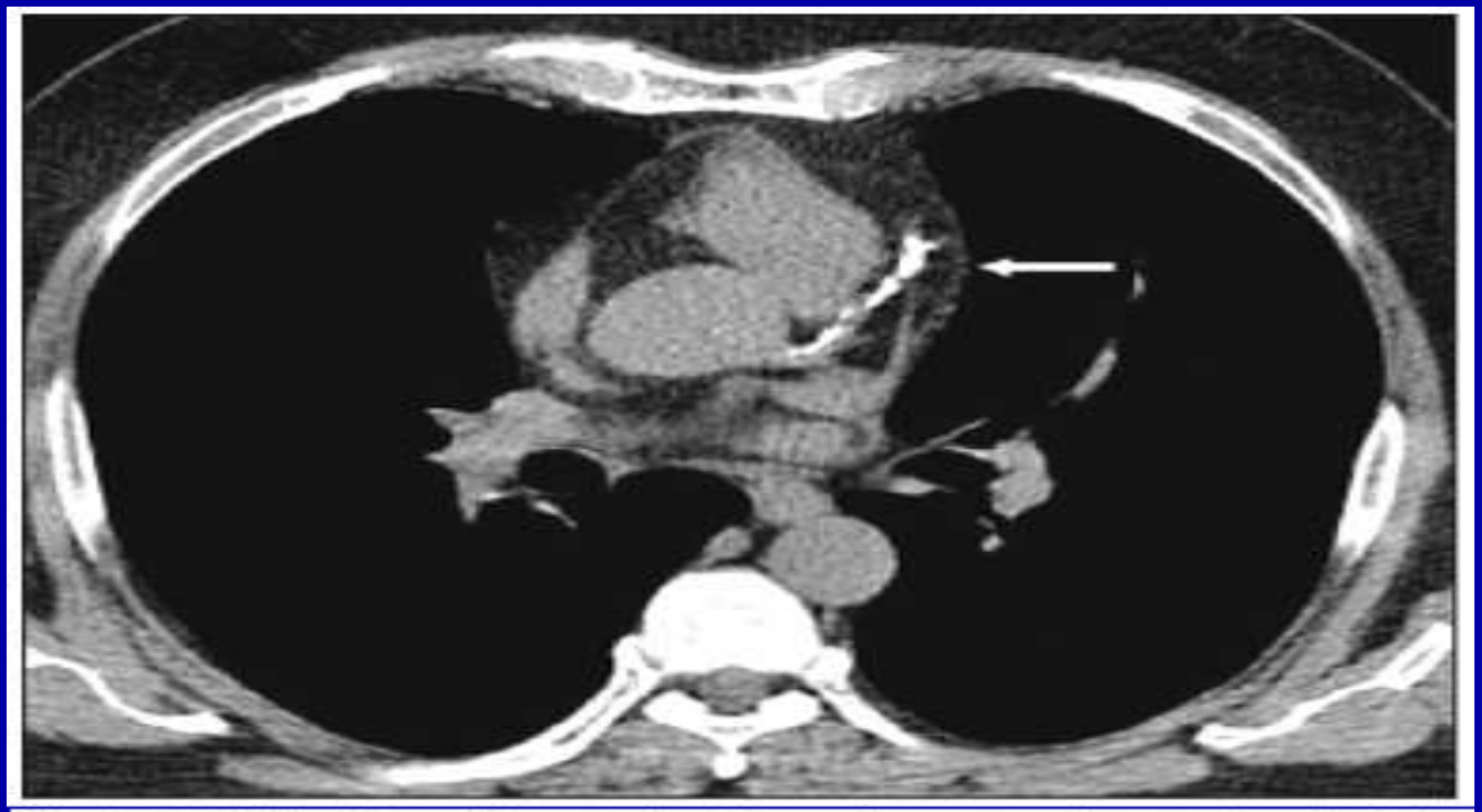


Fig. 1 - Calcification of the anterior descending artery detected on ultrafast tomography in an asymptomatic man (arrow).

CAC predicts stroke risk

4180 subjects from the Heinz Nixdorf Recall Study (45-75 years of age; 47.1% men) without previous stroke, coronary heart disease, or MI were Evaluated for stroke events over 94.9 ± 19.4 months.

Determine whether CAC is a stroke predictor in addition to established vascular risk factors (age, Sex, SBP, LDL, HDL, DM, smoking and AF).

92 subjects (55 men and 37 women) developed a stroke during follow-up period (82 ischemic and 10 hemorrhagic).

Hermann, D., Gronewold, J., et al. Stroke. March 25, 2013;44:1008-1013.

CAC predicts stroke risk

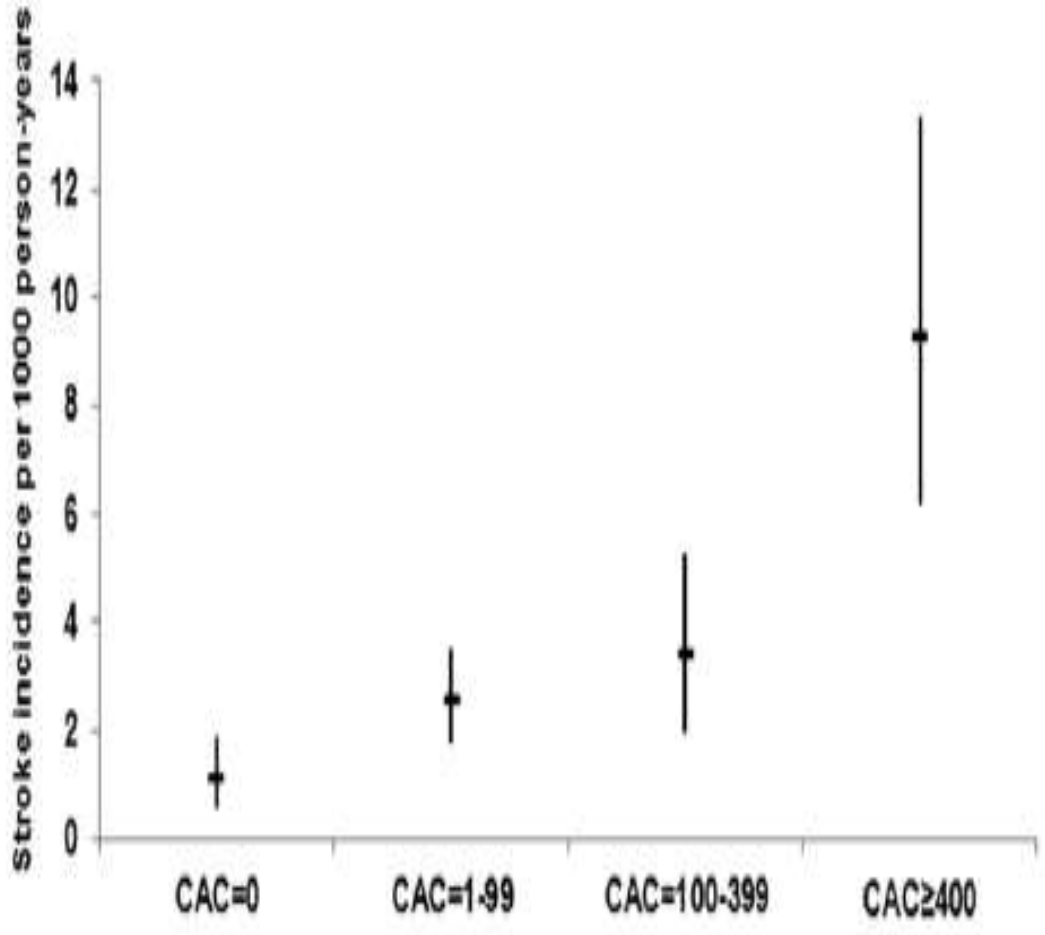


Figure 1. Stroke incidence rates with 95% confidence intervals within coronary artery calcification (CAC) categories. Data are given as number of stroke events per total person time-at-risk in years multiplied with 1000 (strokes per 1000 person-years). Note the markedly increased stroke incidence in subjects exhibiting CAC values ≥400.

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CAC predicts stroke risk

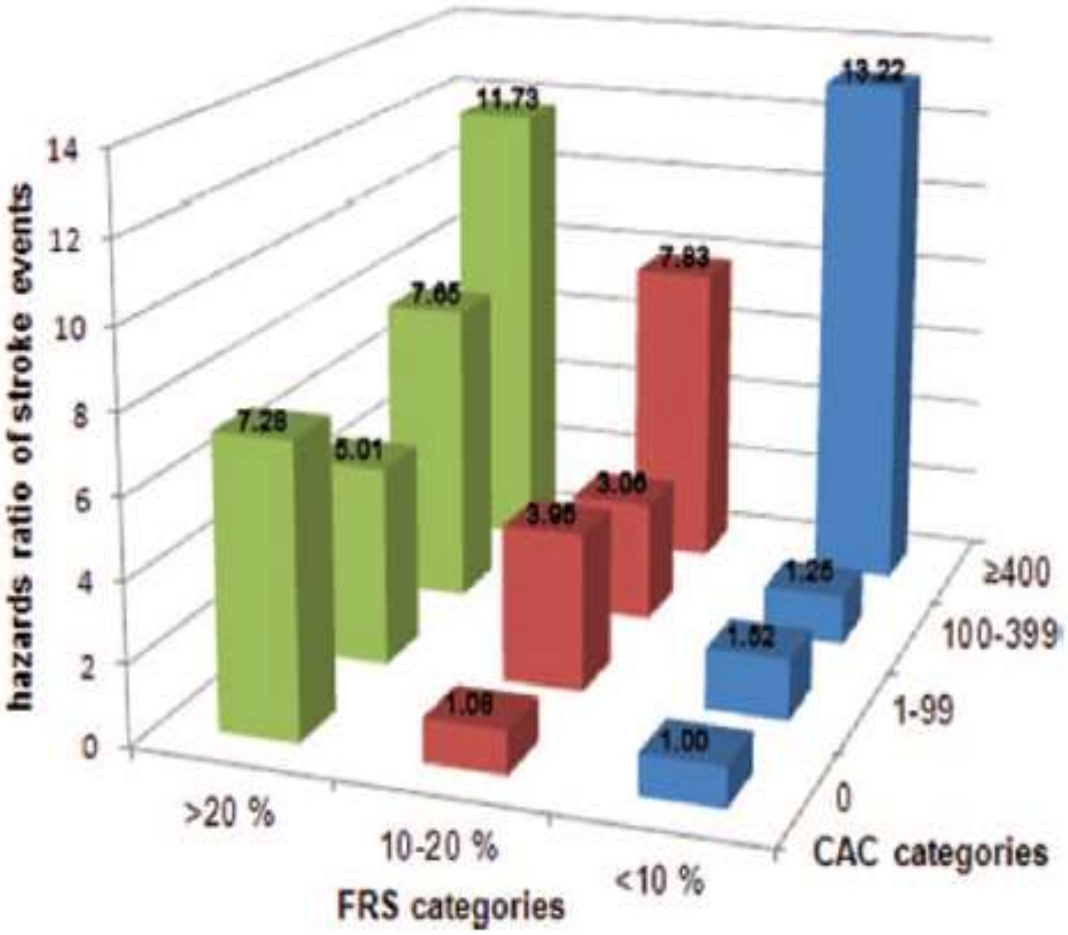


Figure 2. Stroke risk in subjects belonging to the Heinz Nixdorf Recall study stratified on Framingham risk score (FRS) and coronary artery calcification (CAC) categories. Hazards ratios of stroke events in the different combinations of FRS and CAC categories are shown, with the lowest CAC and FRS category as reference. For the low and intermediate FRS categories, log-rank tests for trends revealed significant differences between CAC categories, indicating that CAC discriminates stroke hazard in subjects at low and intermediate vascular risk.

Hermann, D., Gronewold, J., et al. Stroke. March 25, 2013;44:1008-1013.

CAC predicts stroke risk

CAC is an independent predictor of future stroke events in the general population.

CAC predicted stroke in men and women – more significantly in subjects < 65 yrs.

CAC predicted stroke independent of AF

CAC discriminated stroke risk specifically in subjects with FRS <10% and FRS 10-20%.

Hermann, D., Gronewold, J., et al. Stroke. March 25, 2013;44:1008-1013.

CAC predicts stroke risk

BD Take-Away:

Disease ANYWHERE in the vascular system documents risk for a vascular EVENT!

Atherosclerosis = risk for an event.

This discussion goes both ways –

plaque in the coronary tree = risk for stroke.

plaque in the carotid bed = risk for MI.

Hermann, D., Gronewold, J., et al. Stroke. March 25, 2013;44:1008-1013.

Long-term Mortality After Stroke Among Adults age 18-50 years.

FUTURE study – prospective cohort of prognosis after TIA, ischemic stroke or hemorrhagic stroke in adults aged 18-50 years admitted to Radbound University Nijmegen Med Center in Netherlands between Jan 1, 1980- Nov 1, 2010.

Survival status of 959 consecutive patients with first-ever TIA (n=262), ischemic stroke (n=606), or intercerebral hemorrhage (n=91) were assessed as of Nov 1, 2012.

Mean follow-up duration was 11.1 (SD 8.7 yrs).

Cumulative 20-year mortality among 30-day survivors of stroke.

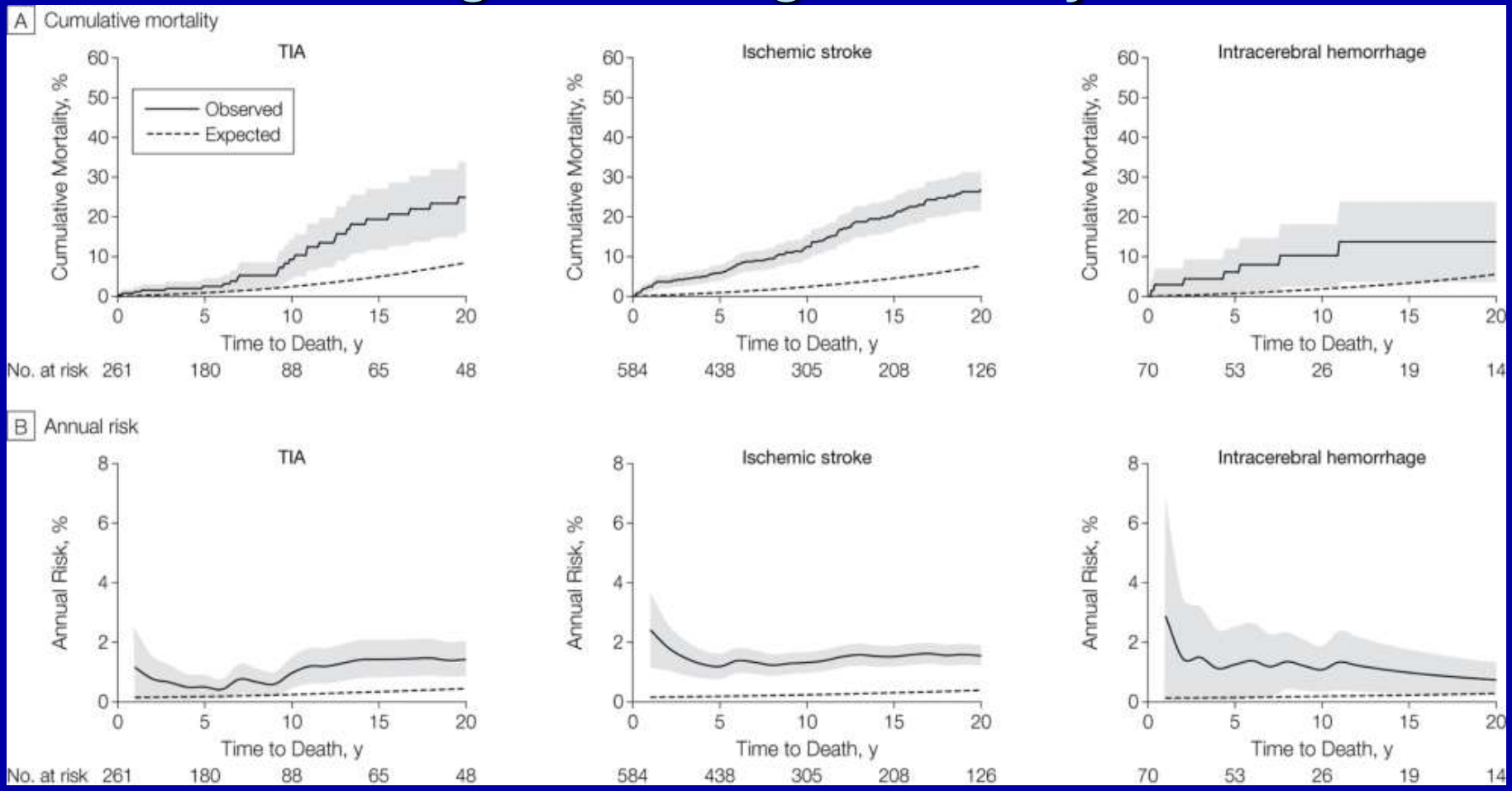
Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

Long-term Mortality After Stroke Among Adults age 18-50 years.

	No.	30-d Survivors, No.	Deaths Among 30-d Survivors, No.	Cumulative 20-y Risk of Death Among 30-d Survivors, % (95% CI)	Observed vs Expected Standardized Mortality Ratio (95% CI)
TIA	262	261	29	24.9 (16.0-33.7)	2.6 (1.8-3.7)
Ischemic stroke	606	584	111	26.8 (21.9-31.8)	3.9 (3.2-4.7)
Intracerebral hemorrhage	91	71	9	13.7 (3.6-23.9)	3.9 (1.9-7.2)

Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

Long-term Mortality After Stroke Among Adults age 18-50 years.



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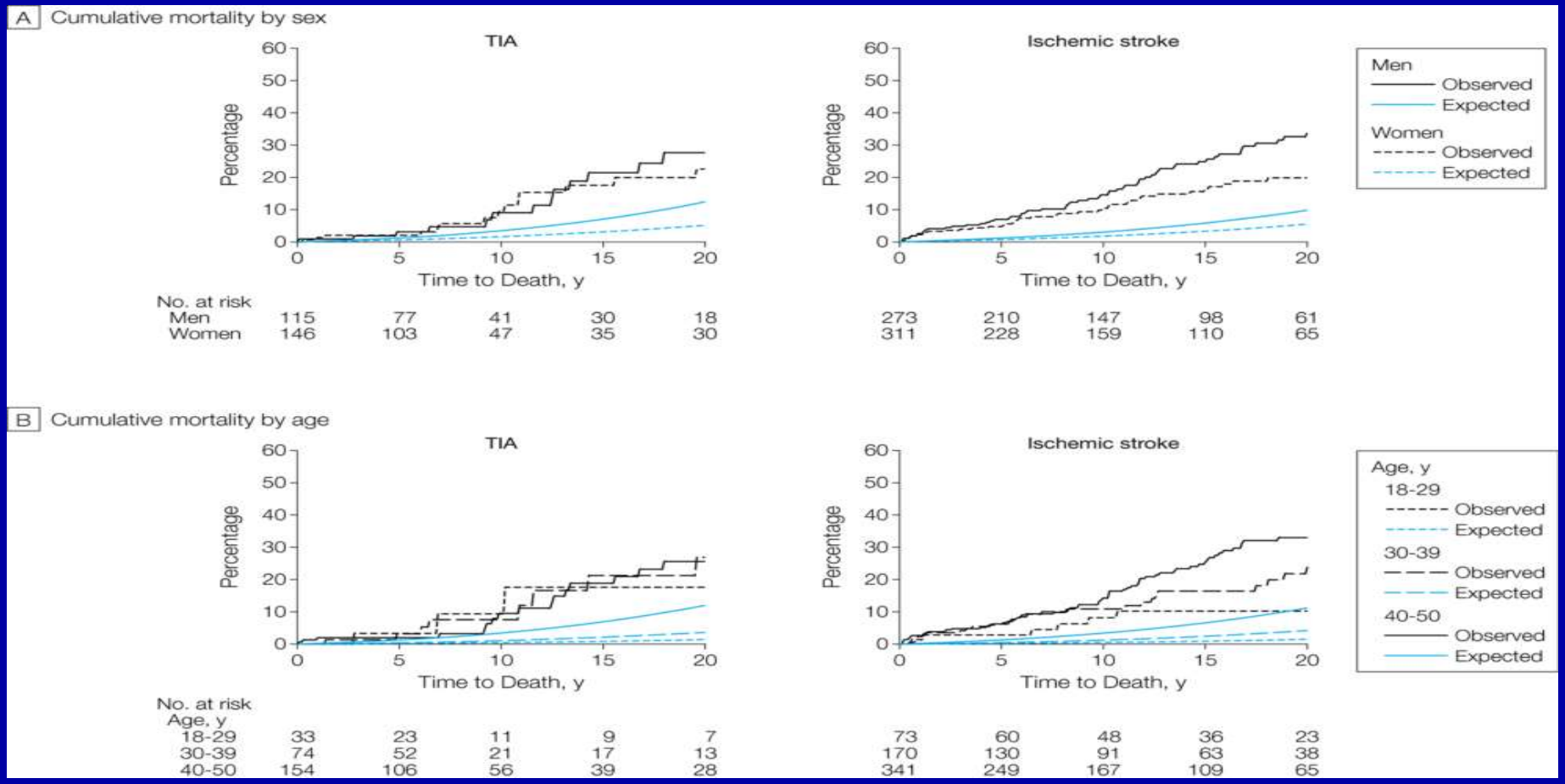
Long-term Mortality After Stroke Among Adults age 18-50 years.



Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

Long-term Mortality After Stroke Among Adults age 18-50 years.

Half of the deaths were attributable to a vascular origin, suggesting that the underlying disease causing the stroke at a young age continues to be active throughout life.

Cardioembolic stroke was the most important predictor of mortality in ALL subgroups and in ALL ages.

Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

Long-term Mortality After Stroke Among Adults age 18-50 years.

Table 4. Causes of Death Among 30-Day Survivors

	No. (%)			
		Index Event		
Cause of Death[a]	Total (n = 145)	TIA (n = 29)	Ischemic Stroke (n = 107)	ICH (n = 9)
Ischemic stroke	20 (13.8)	4 (13.8)	16 (15.0)	0
ICH	8 (5.5)	1 (3.4)	5 (4.7)	2 (22.2)
Cardiac cause	38 (26.2)	4 (13.8)	31 (29.0)	3 (33.3)
Other vascular[b]	9 (6.2)	1 (3.4)	7 (6.5)	1 (11.1)
Malignancies	34 (23.4)	12 (41.4)	21 (19.6)	1 (11.1)
Infections	21 (14.5)	2 (6.9)	17 (15.9)	2 (22.2)
Miscellaneous	15 (10.3)	5 (17.2)	10 (9.3)	0

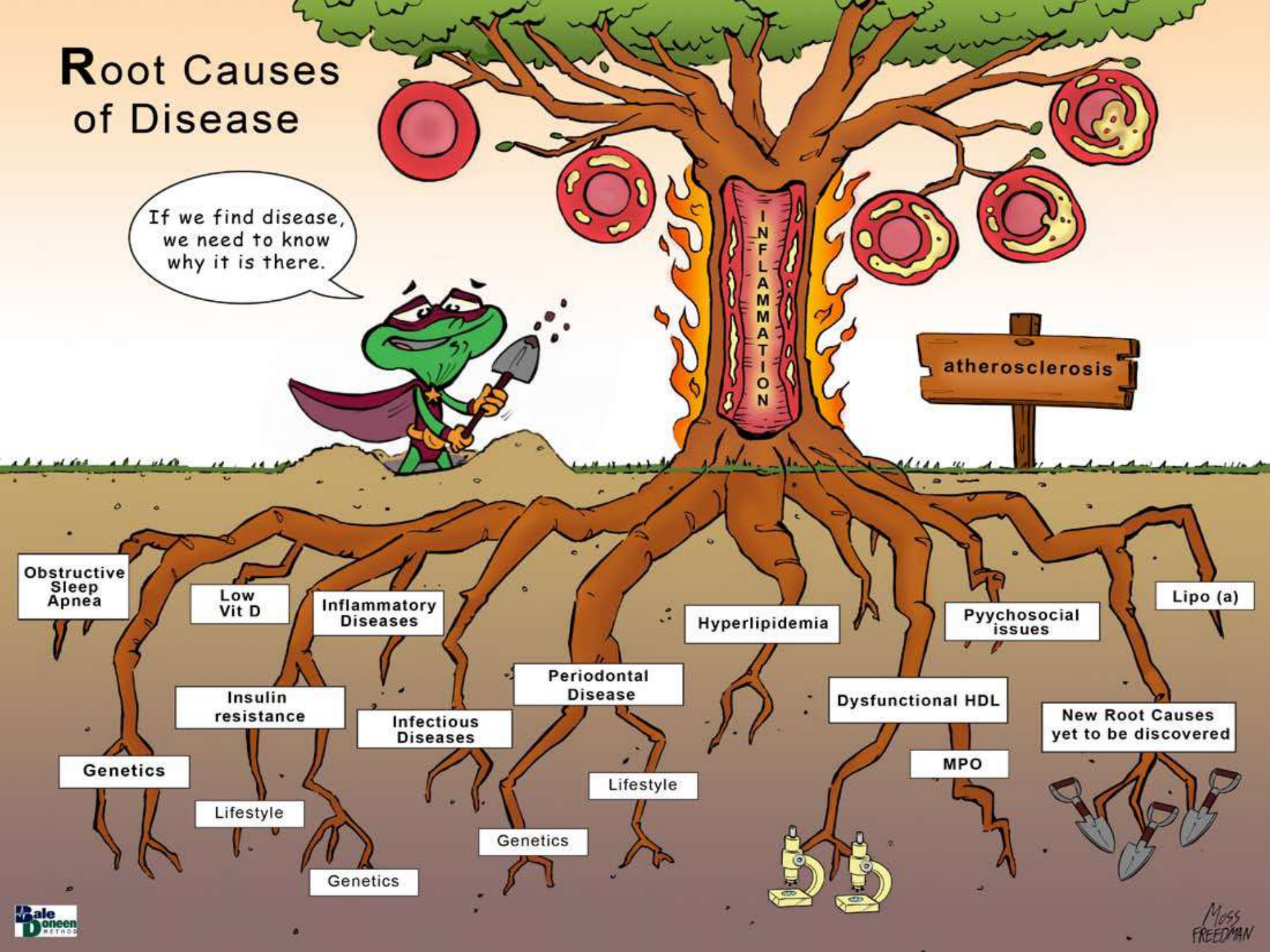
Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.

B/D Take-Away

The underlying vascular disease that caused the stroke or TIA at a young age continues to place these patients at increased risk for recidivistic vascular events throughout their lives.

Primary – Secondary – TERTIARY!

Rutten-Jacobs, L.R., et al., Long-term Mortality after stroke among adults aged 18-50 Years. JAMA, March 20, 2013-Vol 309, No. 11. 1136-1144.


Root Causes of Disease
If we find disease, we need to know why it is there.
INFLAMMATION
atherosclerosis
Obstructive Sleep Apnea
Low Vit D
Inflammatory Diseases
Hyperlipidemia
Pyychosocial issues
Lipo (a)
Periodontal Disease
Insulin resistance
Infectious Diseases
Dysfunctional HDL
New Root Causes yet to be discovered
Genetics
Lifestyle
Genetics
Lifestyle
MPO
Genetics
Lifestyle
Genetics
Dale Doneen METRO
MOSS FREEDMAN

Primary Care vs Specialist Sleep Center Management of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Primary Care vs Specialist Sleep Center management of OSA

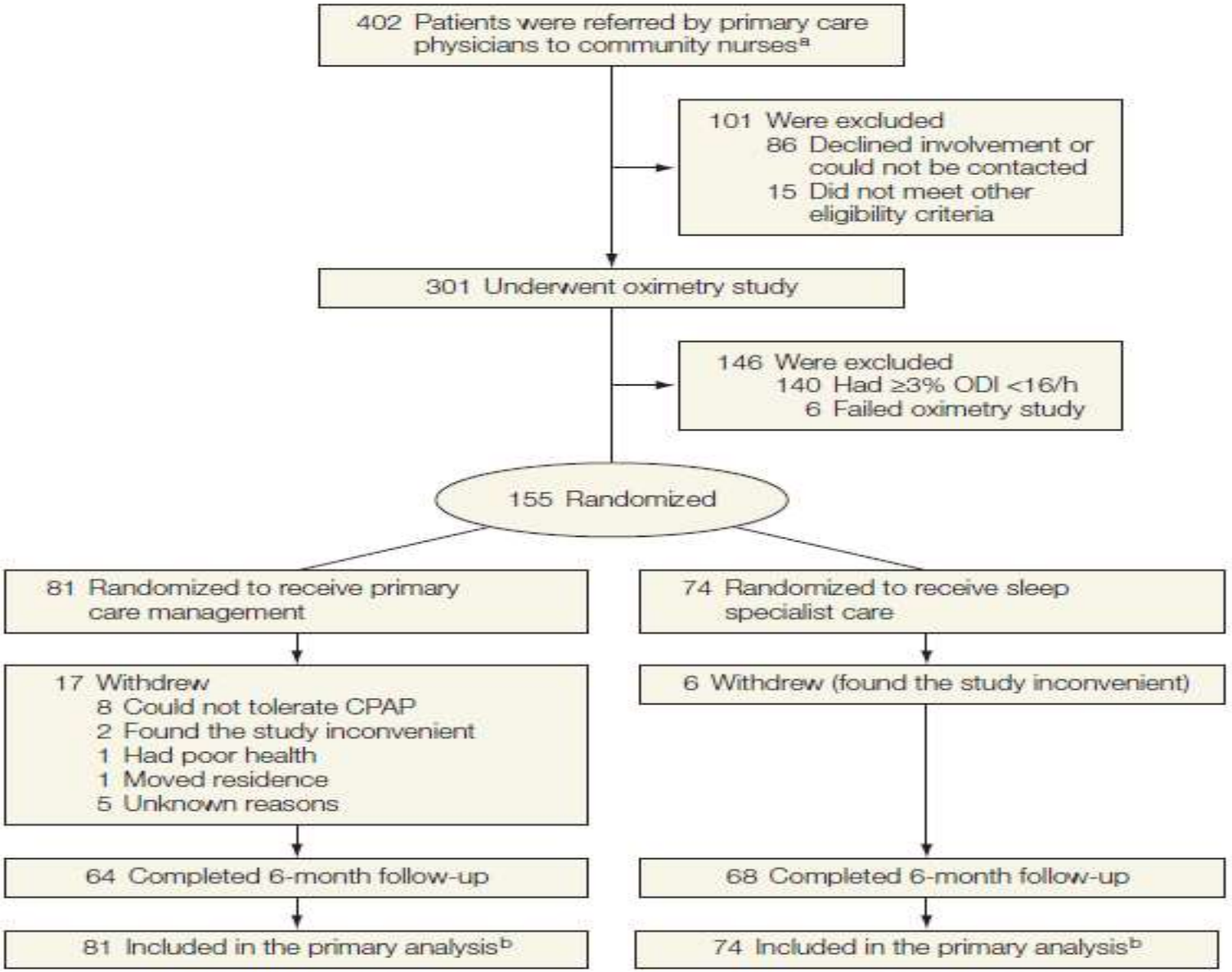
Randomized, controlled, noninferiority study inolving 155 patients with OSA that were treated at primary care practices (n=81) or at university hospital sleep medicine centers (n=74) between Sept 2008 and June 2010.

Primary outcome: 6 mo change in Epworth Sleepiness Scale Score.

Secondary outcome: disease specific and general quality of life measures, OSA symptoms, adherence to CPAP, patient satisfaction and health costs.

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Figure. Flow Diagram of Participant Recruitment and Randomization



Primary Care vs. Specialty management of OSA

Table 1. Baseline Characteristics of Patients[a]

	Primary Care (n = 81)	Specialist Sleep Center (n = 74)
Men, No. (%)	69 (85)	57 (77)
Age, mean (SD), y	57.2 (10.9)	54.5 (11.8)
Region, No. (%)		
Metropolitan	27 (33)	18 (24)
South Coast	3 (4)	1 (1)
Riverland	27 (33)	29 (39)
Barossa Valle	24 (30)	26 (35)
BMI, mean (SD)	33.1 (5.5)	33.7 (5.6)
Waist circumference, mean (SD), cm	111.2 (13.6)	113.1 (14.5)
OSA 50 questionnaire score, mean (SD)	8.2 (1.5)	8.1 (1.7)
ESS total score, mean (SD)	12.8 (3.9)	12.5 (3.9)
Oximetry ≥3% ODI, events/h	32.7 (18.2)	35.7 (17.4)

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Primary Care vs. Specialty management of OSA

Table 2. Principal Treatment Recommended to Patients at Baseline and Used at 6 Months

	No. (%) of Patients	
	Primary Care (n = 81)	Specialist Sleep Center (n = 74)
Baseline recommended treatment		
Principal treatment		
CPAP	73 (90)	52 (70)
Conservative measures only	2 (2)	18 (24)
MAS	1 (1)	3 (4)
Patient withdrew	5 (7)	1 (1)
6-Month principal treatment		
No. of patients[a]	64	68
CPAP	51 (63)	45 (61)
Conservative measures only	7 (9)	12 (16)
MAS	6 (7)	11 (15)

Abbreviations: CPAP, continuous positive airway pressure; MAS, mandibular advancement splint.
[a] At 6 months, 17 patients had withdrawn from primary care group and 6 dropped out of the specialist group.

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Primary Care vs. Specialty management of OSA

Table 3. Change in Epworth Sleepiness Scale Score at 6 Months

Epworth Sleepiness Scale Score	Mean (95% CI)		*P* Value[b]	Adjusted Difference in Mean Change[a]	Lower Bound of 1-Sided 95% CI
	Primary Care (n = 81)	Specialist Sleep Center (n = 74)			
Baseline	12.8 (12.0-13.6)	12.5 (12.4-13.5)			
6-mo[c]	7.0 (6.0-8.0)	7.0 (6.0-8.0)			
Change[d]	5.8 (4.4-7.2)	5.4 (4.2-6.6)	.43	−0.13	−1.50

Abbreviation: ESS, Epworth Sleepiness Scale.
[a] Based on analysis of covariance with adjustment for baseline ESS score and region.
[b] 1-Sided *P* value.
[c] Missing values replaced by multiple imputation.
[d] $P < .001$ for paired *t* test comparison of ESS examining change from baseline to 6 months.

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Table 4. Secondary Outcome Measures at 6 Months

	Primary Care			Specialist Sleep Center				
		Mean (95% CI)			Mean (95% CI)			
	No. of Patients	Baseline Score	Change at 6 mo	No. of Patients	Baseline	Change at 6 mo	Adjusted Difference[a]	*P* Value
FOSQ								4
SASQ								5
SF-36								
Vit								4
Me								4
Blood mr								0
								8
Weight								8

Abbrevi
[a] Based
[b] Measu levels
[c] $P < .0$
[d] Measu indica
[e] Measures the general health status of a patient using 8 subscales, each of which have a total score out of a possible 100 points, with higher scores indicating a higher level of functioning. Only 2 of the 8 SF-36 subscales (ie, vitality and mental health) are reported herein.

<u>There were significant changes in quality of life</u> for both groups although not statistically different from one another.

<u>No statistical difference between PCP and specialty fo</u>r:

1. CPAP use 4.8 hrs (PCP) vs 5.4 hrs (Specialty)
2. SBP and DBP
3. Weight

Chai-Coetzer, C., et al. JAMA March 13, 213, Vol. 309. No 10. 997-1004

Costs for OSA Management

The average total costs per patient were estimated at $1819.44 in the primary care group and $3067.86 in the specialist group.

Sleep study costs, sleep physician consultations and travel costs were main contributors.

Primary care management of OSA was 40% cheaper and non-inferior to specialist care in both the Australian and US contexts.

Chai-Coetzer, C., Antic, N., et al. Primary Care vs Specialist Sleep Center Management Of Obstructive Sleep Apnea and Daytime Sleepiness and Quality of Life. JAMA March 13, 213, Vol. 309. No 10. 997-1004

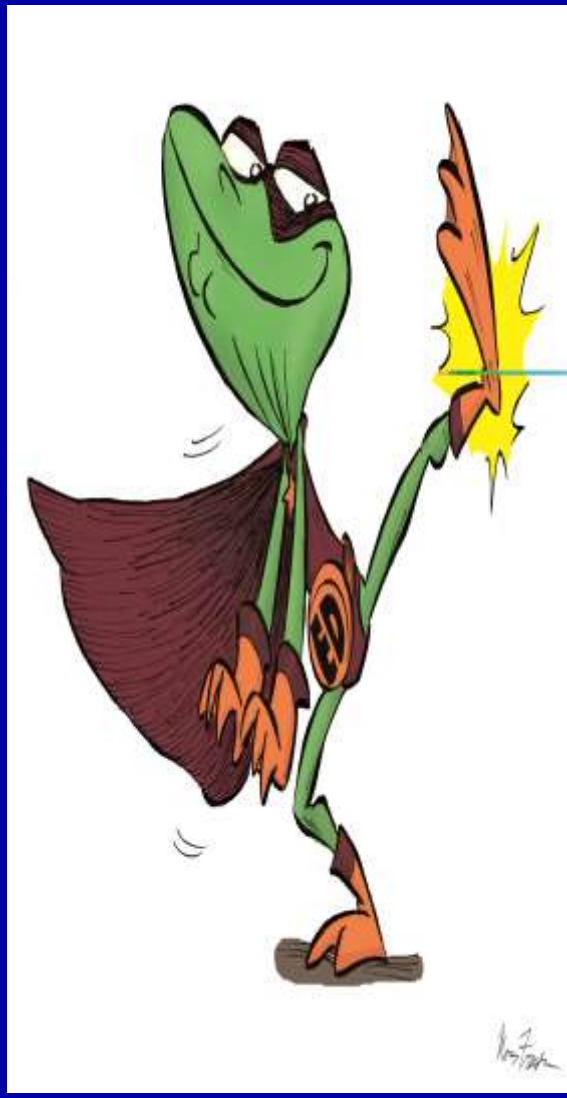
BD Take-Away

Primary Care management of OSA is NOT inferior to specialty care management of OSA and promotes a significant cost savings opportunity to the patient.

OSA is a root cause of atherosclerosis.

EDFROG IRA

Education	Genetics	Individual management
Disease	Optimal	Risk factor response
Fire	Roots	Assess Disease Annually



TREATMENT

The Impact of Green Tea and Coffee Consumption on the Reduced Risk of Stroke Incidence in Japanese Population

The Japan Public Health Center-Based Study Cohort

Yoshihiro Kokubo, MD, PhD, FAHA; Hiroyasu Iso, MD, PhD; Isao Saito, MD, PhD; Kazumasa Yamagishi, MD, PhD; Hiroshi Yatsuya, MD, PhD; Junko Ishihara, PhD; Manami Inoue, MD, PhD; Shoichiro Tsugane, MD, PhD

Kokubo, Y et al. Impact of Green Tea and Coffee and Stroke Incidence. Stroke. AHA Published Online March 14, 2013, AHA ISSN: 0039-2499. Online ISSN: 1524--4628

Impact of both green tea and coffee consumption on strokes

82,369 Japanese (45-79 years) without CVD or cancer in 1995 and 1998.

13 years f/u through the end of 2007.

Green Tea and Coffee assessed by questionnaire at baseline.

Kokubo, Y et al. Impact of Green Tea and Coffee and Stroke Incidence. Stroke. AHA Published Online March 14, 2013, AHA ISSN: 0039-2499. Online ISSN: 1524--4628

Table 1. Baseline Characteristic Variables in a Cohort Subjects According to Green Tea and Coffee Consumption

	Green Tea						Coffee				
	(Times/Week)			(Cups/d)			(Times/Week)			(Cups/d)	
	0	1–2	3–6	1	2–3	≥4	0	1–2	3–6	1	≥2
Number of subjects	17606	8497	7490	8103	17426	23247	19841	18762	13364	15128	15019
Age at baseline, y	54.1	52.7	52.7	53.1	53.8	55.4	56.6	55.2	53.5	53.2	50.4
Sex, % of men	47.3	47.6	50.3	48.9	47.5	42.3	42.2	45.0	51.7	44.3	51.3
Body mass index, kg/m^2	23.8	23.7	23.8	23.6	23.5	23.4	23.5	23.8	23.7	23.6	23.5
Current smoker, %	24.6	25.4	26.3	25.8	24.6	23.4	16.9	20.2	26.7	25.0	37.2
Current drinker, %	39.8	44.1	49.0	45.6	44.9	40.7	37.3	41.6	47.5	42.8	47.6
Sports at leisure time 1>time/week, %	17.2	18.7	20.5	20.0	20.0	21.7	18.8	21.0	21.0	21.4	22.5
Antihypertensive drug users, %	20.1	16.8	17.6	18.1	19.0	20.0	25.2	22.1	17.0	17.0	11.0
Antilipidemic drug users, %	4.5	4.6	5.0	4.7	5.4	5.5	5.9	5.7	4.4	4.7	3.0
History of diabetes mellitus, %	5.1	5.1	5.0	4.9	5.0	5.0	7.1	4.8	4.1	3.9	3.5
coffee >1 time/d	38	39	36	41	39	27	–	–	–	–	–
Green tea >1 time/d, %	–	–	–	–	–	–	60	63	57	59	54

Kokubo, Y et al. Impact of Green Tea and Coffee and Stroke Incidence. Stroke. AHA Published Online March 14, 2013, AHA ISSN: 0039-2499. Online ISSN: 1524--4628

Results Trends

Higher Green Tea consumption tended to have higher prevalence of exercise

- Inverse relationship with green tea at ≥4cup/day and incidences of CVD and strokes.

Higher Coffee consumption tended to be younger, higher prevalence of smoking and exercise and had a lower prevalence of antihypertensive drug users and history of diabetes.

- Inverse relationship with coffee at ≥ 2 cups/d and incidences of CVD and strokes (disappeared after age and smoking adjustments)

Kokubo, Y et al. Impact of Green Tea and Coffee and Stroke Incidence. Stroke. AHA Published Online March 14, 2013, AHA ISSN: 0039-2499. Online ISSN: 1524--4628

Green Tea

Table 2. Age and Multivariable-Adjusted Hazard Ratios of Cardiovascular Disease and its Subtypes According to Green Tea Consumption

	Green Tea Consumption						
	None	1–2 Times/Week	3–6 Times/Week	1 Cup/d	2–3 Cups/d	≥4 Cups/d	*P* for Trend
Person-years	228 788	108 408	95 222	105 019	226 579	302 703	
Cardiovascular disease							
Number of cases	1070	434	372	436	839	1184	
Age-adjusted HRs	1	0.95 (0.85–1.06)	0.91 (0.81–1.02)	0.93 (0.83–1.03)	0.81 (0.74–0.88)	0.78 (0.72–0.84)	<0.001
Multivariable-adjusted HRs	1	0.94 (0.84–1.05)	0.93 (0.82–1.05)	0.94 (0.84–1.05)	0.85 (0.78–0.93)	0.84 (0.77–0.92)	<0.001
All strokes							
Number of cases	848	361	289	346	672	909	
Age-adjusted HRs	1	0.99 (0.88–1.12)	0.90 (0.79–1.02)	0.94 (0.83–1.06)	0.81 (0.74–0.90)	0.75 (0.69–0.82)	<0.001
Multivariable-adjusted HRs	1	0.97 (0.86–1.10)	0.91 (0.80–1.05)	0.94 (0.83–1.07)	0.86 (0.78–0.95)	0.80 (0.73–0.89)	<0.001

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Coffee

Table 3. Age and Multivariable-Adjusted Hazard Ratios of Cardiovascular Disease and its Subtypes According to Coffee Consumption

	Coffee Consumption					
	None	1–2 Times/Week	3–6 Times/Week	1 Cup/d	≥2 Cups/d	*P* for Trend
Person-years	254 006	242 850	172 976	199 104	196 914	
Cardiovascular disease						
Number of cases	1282	1045	678	679	616	
Age-adjusted HRs	1	0.84 (0.78–0.91)	0.84 (0.77–0.92)	0.77 (0.71–0.85)	0.84 (0.76–0.92)	<0.001
Multivariable-adjusted HRs	1	0.93 (0.86–1.01)	0.89 (0.81–0.98)	0.84 (0.76–0.92)	0.89 (0.80–0.99)	0.004
All strokes						
Number of cases	1038	843	534	529	441	
Age-adjusted HRs	1	0.85 (0.78–0.92)	0.82 (0.74–0.91)	0.74 (0.67–0.82)	0.75 (0.67–0.84)	<0.001
Multivariable-adjusted HRs	1	0.94 (0.85–1.02)	0.89 (0.80–0.99)	0.80 (0.72–0.90)	0.81 (0.72–0.91)	<0.001

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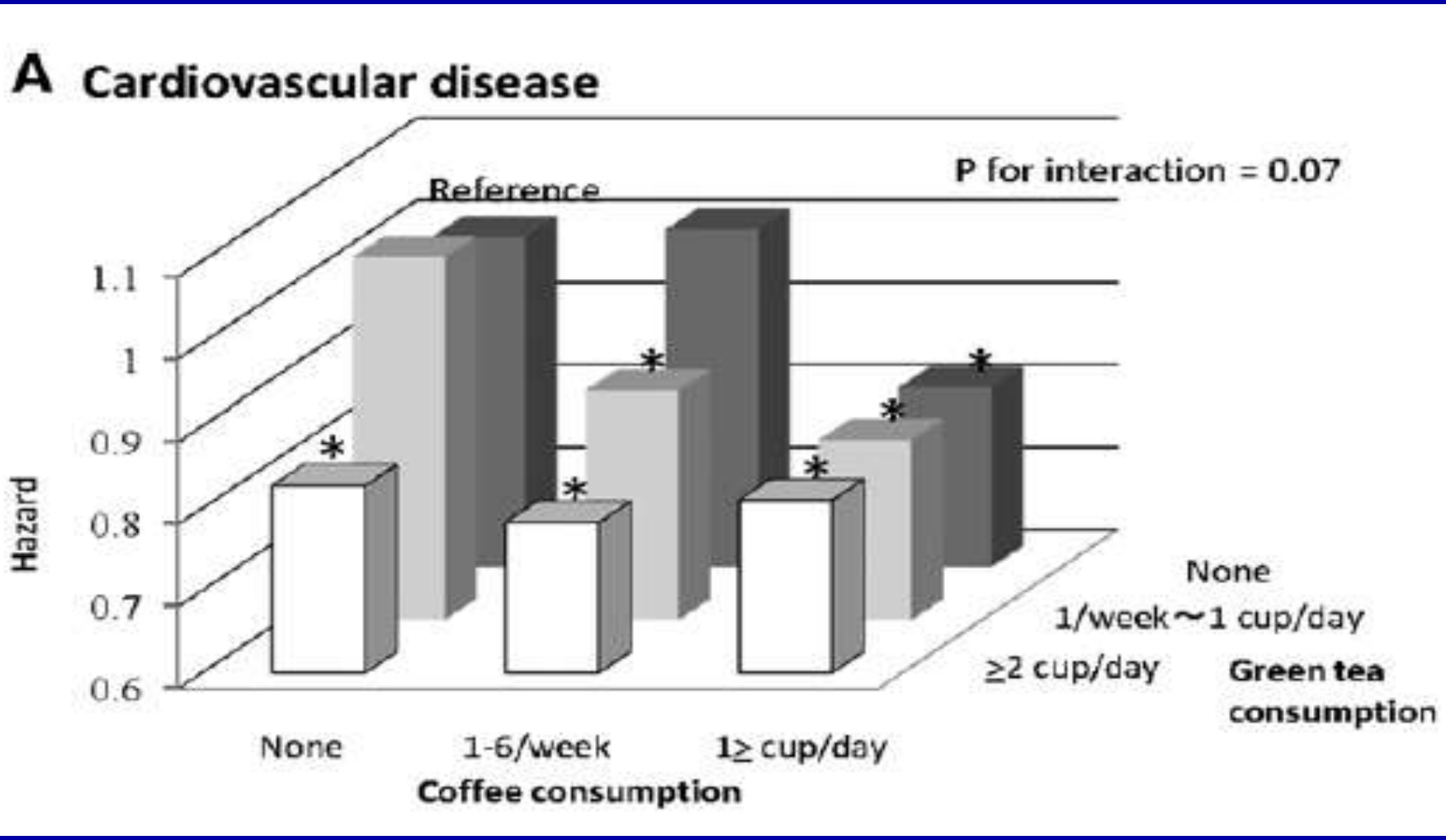

*indicates $P<0.05$ compared with seldom green tea or coffee (reference)

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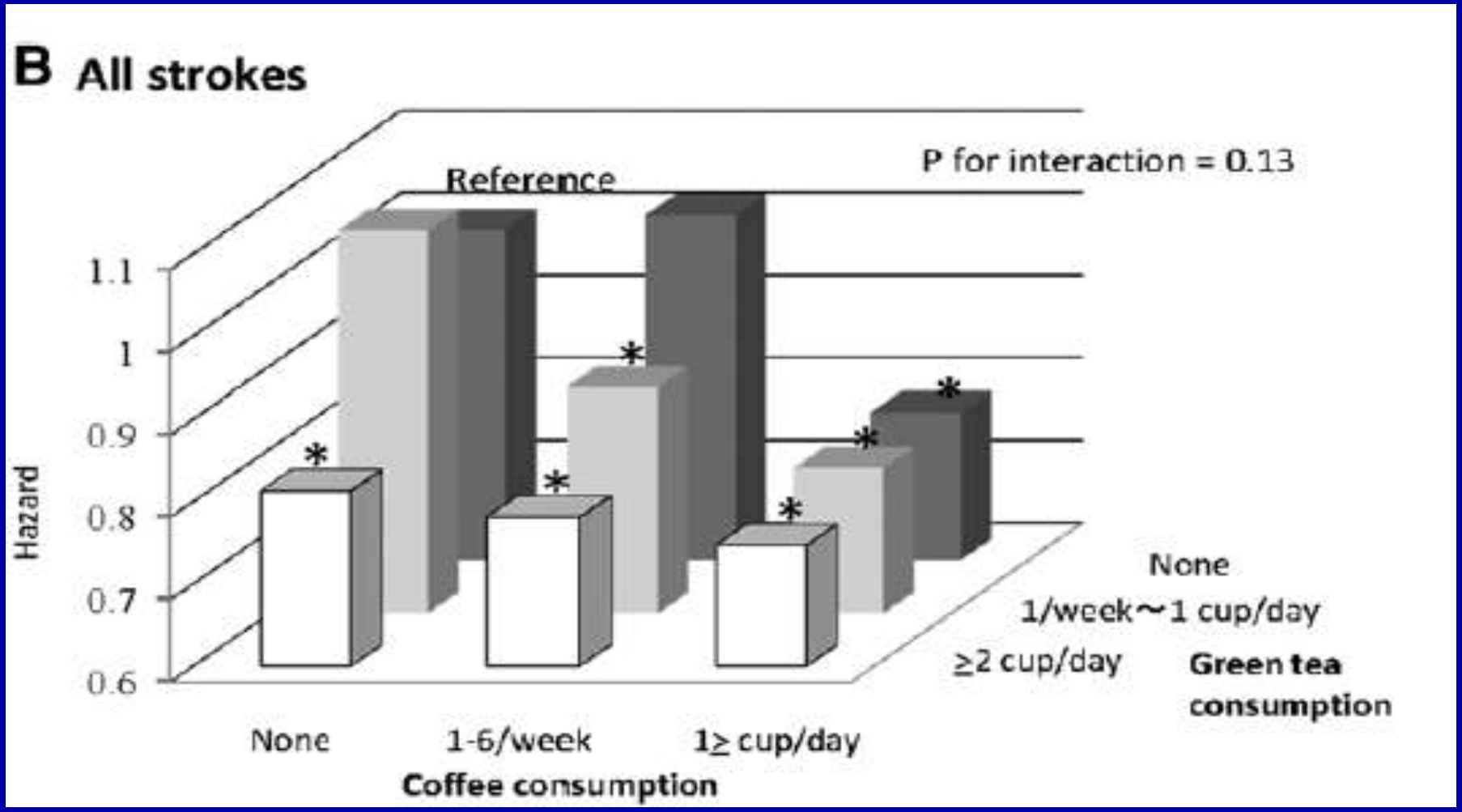

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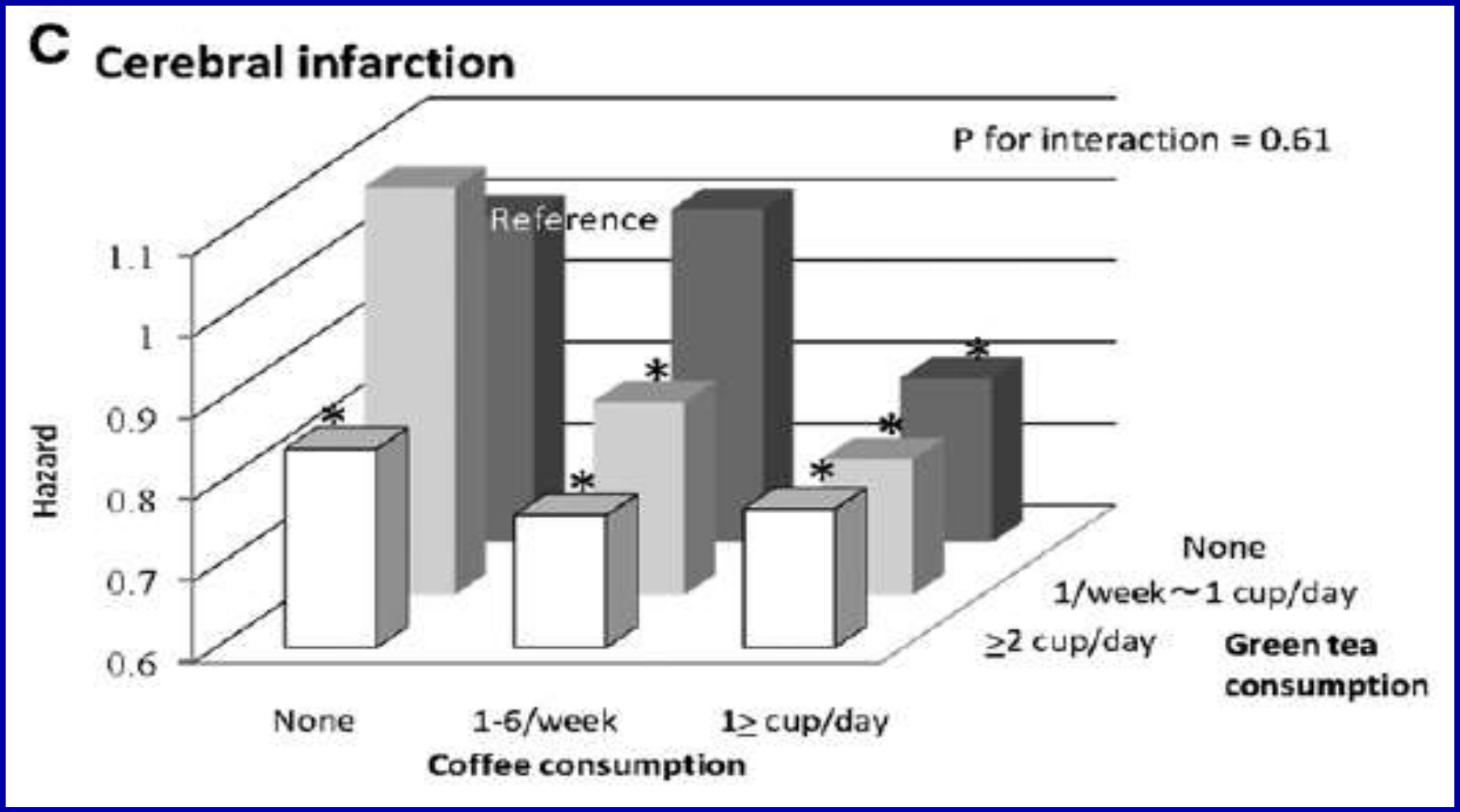

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Why??

Green Tea: catechins (epigallocatechin 3-gallate) exerts vascular-protective effects:

- antioxidative
- anti-inflammatory
- antiproliferative
- increase plasma antioxidant capacity
- antithrombogenic effects
- Less likely to develop hypertension

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Why??

Coffee:

caffeine and diterpene –shown to affect lipids, blood pressure and insulin sensitivity

Chlorogenic acid and quinides: may reduce body weight and blood glucose.

**combination of green tea and coffee benefit is not yet clear but positive.

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BD Take-Away

Green Tea and Coffee are GOOD to have on a daily basis and certainly easy to obtain for our patients.

Walking vs Running for HTN, Cholesterol and DM Risk Reduction

Test whether equivalent energy expenditure by moderate-intensity (walking) and vigorous-intensity (running) provides equivalent health benefits.

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636.

National Runners' and Walkers' Health Study Cohorts

n=33 060 (runners)

n=15 945 (walkers)

Utilized METh/d (metabolic equivelant hours per day) compared with self-reported, physician diagnosed HTN, hyperlipidemia, DM and CHD during 6.2 year follow-up.

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

Walking Versus Running for Hypertension, Cholesterol, and Diabetes Mellitus Risk Reduction

	Men		Women	
	Runners	Walkers	Runners	Walkers
Sample, n	16 983	3349	16 077	12 596
Age, y	48.28±10.98	61.77±11.10	40.89±10.66	53.08±12.05
Follow-up, y	6.30±0.91	5.60±1.17	6.55±0.94	5.69±1.26
Education, y	16.79±2.46	16.31±2.72	16.35±2.31	15.27±2.54
Current smokers, %	1.22	3.40	1.69	3.68
Meat, servings/d	0.44±0.40	0.46±0.41	0.27±0.30	0.37±0.34
Fruit, pieces/d	1.53±1.18	1.62±1.22	1.53±1.06	1.70±1.14
Alcohol, g/d	9.85±13.47	9.16±13.40	5.88±8.21	4.93±9.09
BMI, kg/m^2	24.09±2.59	26.63±4.05	21.62±2.51	25.48±5.18
Energy expenditure, METh/d				
Running	5.29±3.12		4.74±3.03	
Walking		2.20±1.66		2.14±1.63
Other vigorous exercise	1.70±3.21	1.69±3.34	2.06±3.34	1.46±2.95
Other exercise, moderate	0.76±1.63	0.43±1.49	0.83±1.73	0.36±1.26
Other exercise, light	0.02±0.30	0.04±0.59	0.03±0.36	0.03±0.25
Other exercise, strength	0.53±1.26	0.20±0.86	0.54±1.26	0.20±0.75

BMI indicates body mass index; and METh/d, metabolic equivant hours per day.

Table 2. Hazard Ratios (95% Confidence Intervals) From Cox Proportional Hazard Analyses of Self-Reported Incident Hypertension, Hypercholesterolemia, Diabetes Mellitus, and CHD

	Hypertension	Hypercholesterolemia	Diabetes Mellitus	CHD
Sample size, n	43 341	44 216	48 116	47 921
Incident events	3874	6637	647	530
Runners (0,1)	0.623 (0.552–0.704)§	0.640 (0.583–0.702)¶	0.294 (0.214–0.405)§	0.478 (0.342–0.666)§
Energy expenditure at baseline (risk reduction per METh/d)				
Running	0.958 (0.944–0.973)§	0.957 (0.946–0.968)¶	0.879 (0.832–0.929)§	0.955 (0.912–1.000)*
Walking	0.928 (0.899–0.957)§	0.930 (0.908–0.953)§	0.877 (0.824–0.934)§	0.907 (0.839–0.981)†
Other vigorous	0.983 (0.972–0.994)†	0.986 (0.978–0.994)‡	0.980 (0.950–1.007)	0.994 (0.966–1.024)
Other moderate	0.997 (0.976–1.018)	0.998 (0.982–1.014)	0.969 (0.908–1.024)	0.984 (0.927–1.044)
Other light	0.886 (0.739–1.006)	1.011 (0.955–1.061)	0.992 (0.736–1.121)	0.983 (0.807–1.197)

Analyses of runners and walkers combined adjusted for baseline age (age, age^2), sex, and race (self-identified black, Hispanic, Asian, Native American), education, smoking, and intakes of red meat, fruit, and alcohol. Analyses of hypertension, hypercholesterolemia, and diabetes mellitus also included adjustment for preexisting CHD at baseline. CHD indicates coronary heart disease; and METh/d, metabolic equivant hours per day.

Significance levels for individual coefficients are coded: *$P<0.05$; †$P<0.01$; ‡$P<0.001$; §$P<0.0001$; ¶$P<10^{-15}$.

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

Table 3. Hazard Ratios (95% Confidence Intervals) From Cox Proportional Hazard Analyses of Self-Reported Incident Hypertension, Hypercholesterolemia, Diabetes Mellitus, and CHD, Adjusted for BMI

	Hypertension	Hypercholesterolemia	Diabetes Mellitus	CHD
Sample size, n	42 853	43 683	47 584	47 339
Incident events	3811	6520	629	509
BMI, kg/m^2	1.087 (1.079–1.095)¶	1.061 (1.055–1.067)¶	1.138 (1.125–1.150)¶	1.070 (1.048–1.093)§
Runners (0,1)	0.862 (0.759–0.979)*	0.819 (0.743–0.903)§	0.587 (0.420–0.821)†	0.569 (0.401–0.808)†
Energy expenditure at baseline (risk reduction per METh/d)				
Running	0.977 (0.962–0.992)†	0.968 (0.957–0.979)§	0.912 (0.861–0.963)‡	0.978 (0.934–1.025)
Walking	0.987 (0.957–1.018)	0.976 (0.952–1.000)*	1.013 (0.950–1.078)	0.946 (0.873–1.025)
Other vigorous	0.988 (0.977–0.999)*	0.990 (0.982–0.998)*	0.995 (0.965–1.022)	0.997 (0.968–1.027)
Other moderate	0.995 (0.974–1.016)	0.996 (0.980–1.013)	0.965 (0.904–1.020)	0.983 (0.925–1.044)
Other light	0.920 (0.776–1.034)	1.026 (0.973–1.075)	1.040 (0.801–1.158)	0. 998 (0.828–1.204)

Analyses of runners and walkers combined adjusted for baseline age (age, age^2), sex, and race (self-identified black, Hispanic, Asian, Native American), education, smoking, and intakes of red meat, fruit, and alcohol. Analyses of hypertension, hypercholesterolemia, and diabetes mellitus also included adjustment for preexisting CHD at baseline. BMI indicates body mass index; CHD, coronary heart disease; and METh/d, metabolic equivant hours per day.

Significance levels for individual coefficients are coded: *$P<0.05$; †$P<0.01$; ‡$P<0.001$; §$P<0.0001$; ¶$P<10^{-15}$. Samples sizes differ slightly from Table 2 because of missing BMI.

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

Results:

Final Conclusion:

Risk reductions were NOT statistically different for running than walking for diabetes mellitus (P=0.94), hypercholesterolemia = (P=0.06), or CHD (P=0.26) and marginally greater for walking than running or hypercholesterolemia (P=0.04).

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

Interesting findings….

Runners had 38% lower risk for HTN, 36% lower risk for hyperlipidemia and 71% lower risk for DM than walkers.

The average caloric expenditure was more than twice as great for those who chose running over walking.

Hard to compare ‘time’ spent – quantify distance rather than duration: more accurate.

BD Take-Away: All exercise is beneficial! Pedometers are a great idea for walkers and distance is more accurate.

Williams, P., Thompson, P, Walking Versus Running for HTN, Cholesterol and DM Risk Reduction. ATVB. April 5, 2013, Published online AHAjounals. ISSN: 1524-4636

In the headlines…what our patients are reading…..

In the headlines…what our patients are reading…..

New Culprit in Red Meat Linked with Heart Disease

By Cari Nierenberg, MyHealthNewsDaily Contributor | LiveScience.com – Mon, Apr 8, 2013

"The high amounts of saturated fat and cholesterol in red meat have long been blamed for increasing people's risk of heart disease. But now, new research points a finger at another culprit in meat that may be more closely tied to this leading killer.

A new study reveals that a nutrient called l-carnitine, which is found in red meat and is also popular as a dietary supplement, may also play a role in the development of heart disease."

http://news.yahoo.com/culprit-red-meat-linked-heart-disease-122309519.html

Intestinal microbiota metabolism of L-Carnitine, a nutrient in red meat, promotes atherosclerosis.

? Environmental elements other than saturated fat in meat that may increase CVD risk.

Explored the participation of commensal intestinal microbiota in modifying the diet-host interaction with reference to red meat consumption.

Koeth, R., Hazen, S., et al. Intestinal microbiota metabolism of L-carnitine, a nutrient red meat, promotes atherosclerosis. Nature Medicine. April 7, 2013. doi 10.1038.

Intestinal microbiota metabolism of L-Carnitine, a nutrient in red meat, promotes atherosclerosis.

Pathway in both humans and mice linking microbiota metabolism of dietary choline and phosphatidylcholine to CVD pathogenesis.

Choline is metabolized by gut microbiota to produce TMA which is rapidly oxidized by hepatic flavin monoxygenases to form TMAO which is proatherogenic and associated with CVD risk.

Koeth, R., Hazen, S., et al. Intestinal microbiota metabolism of L-carnitine, a nutrient red meat, promotes atherosclerosis. Nature Medicine. April 7, 2013. doi 10.1038.

Intestinal microbiota metabolism of L-Carnitine, a nutrient in red meat, promotes atherosclerosis.

TMAO has been proposed to:

Induce upregulation of macrophage scavenger receptors => increase cholesterol transport.

L-Carnitine –

Abundant in red meat and contains trimethylamine structure similar to choline.

Also – It is endogenous in mammals and is essential in transporting fatty acids into the mitochondrial compartment of the cell.

Koeth, R., Hazen, S., et al. Intestinal microbiota metabolism of L-carnitine, a nutrient red meat, promotes atherosclerosis. Nature Medicine. April 7, 2013. doi 10.1038.

Intestinal microbiota metabolism of L-Carnitine, a nutrient in red meat, promotes atherosclerosis.

Studied gut bacteria – dependent on metabolism of L-carnitine to produce TMAO in Rodents and Humans.

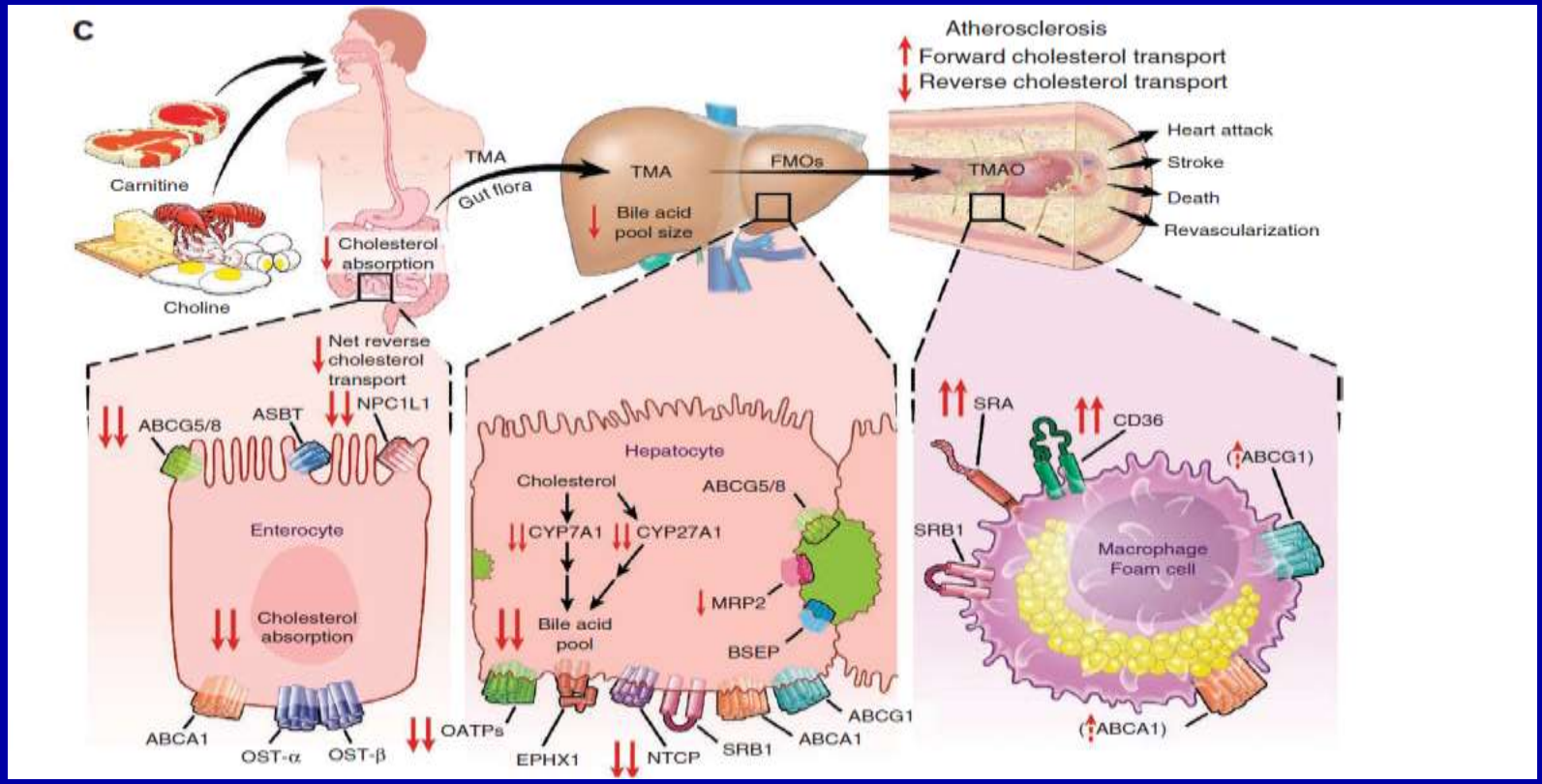
Used isotope tracers in humans – clinical studies for CVD risk

Demonstrated that: TMAO and its dietary precursors choline and carnitine, suppress reverse cholesterol transport (RCT) through gut microbiota-dependent mechanisms.

*Dietary L-Carnitine might be metabolized to produce TMA and TMAO in a gut microbiota-dependent fashion and be associated with atherosclerotic risk.

Koeth, R., Hazen, S., et al. Intestinal microbiota metabolism of L-carnitine, a nutrient red meat, promotes atherosclerosis. Nature Medicine. April 7, 2013. doi 10.1038.

TMAO and L-carnitine



BD Concern: L-Carnitine for lipo(a) –
stop/hold until further understanding of this pathway.

Koeth, R. A., et. al. published online ***Nature Medicine*** 7 April 2013
doi:10.1038/nm.3145

Last one….preventing Cancer!!!

Cardiovascular Disease Prevention Reduces Cancer Risk!

Examined adherence to ideal levels of the seven AHA cardiovascular health metrics

Compared with incident cancers in the Atherosclerosis Risk In Communities (ARIC) study over 17-19 years of follow-up.

A total of 13,253 participants included for analysis.

Combined cancer incidence (excluded non-melanoma skin cancers) from 1987-2006. A total of 2880 incident cancers occurred during follow-up.

Rassmussen-Torvik, L., Shay, C., et al., Circulation March 18, 2013. ISSN 1524-4539

AHA 7 Essentials for Heart Health

- Do not smoke
- BMI ≤25
- Exercise/wk: 150’ moderate or 75’ vigorous.
- Diet- at least four of these five:
  1) 4 – 1/2 cups/day of fruit and vegetables
  2) ≥two 3.5-ounce fish/wk
  3) ≤ three sugar-sweetened 12 oz. beverages/wk
  4) ≥ three 1-ounce servings of fiber-rich whole grains/day
  5) <1,500 milligrams salt/day
- TC <200 mg/dL
- BP < 120/80
- FBG <100 mg/dL

Published on line 1/20/2010: AHA ***Circulation***

Cardiovascular Disease Prevention Reduces Cancer Risk!

Table 2. Incident combined cancer rates by number of ideal health metrics: The ARIC Study, 1987-2006

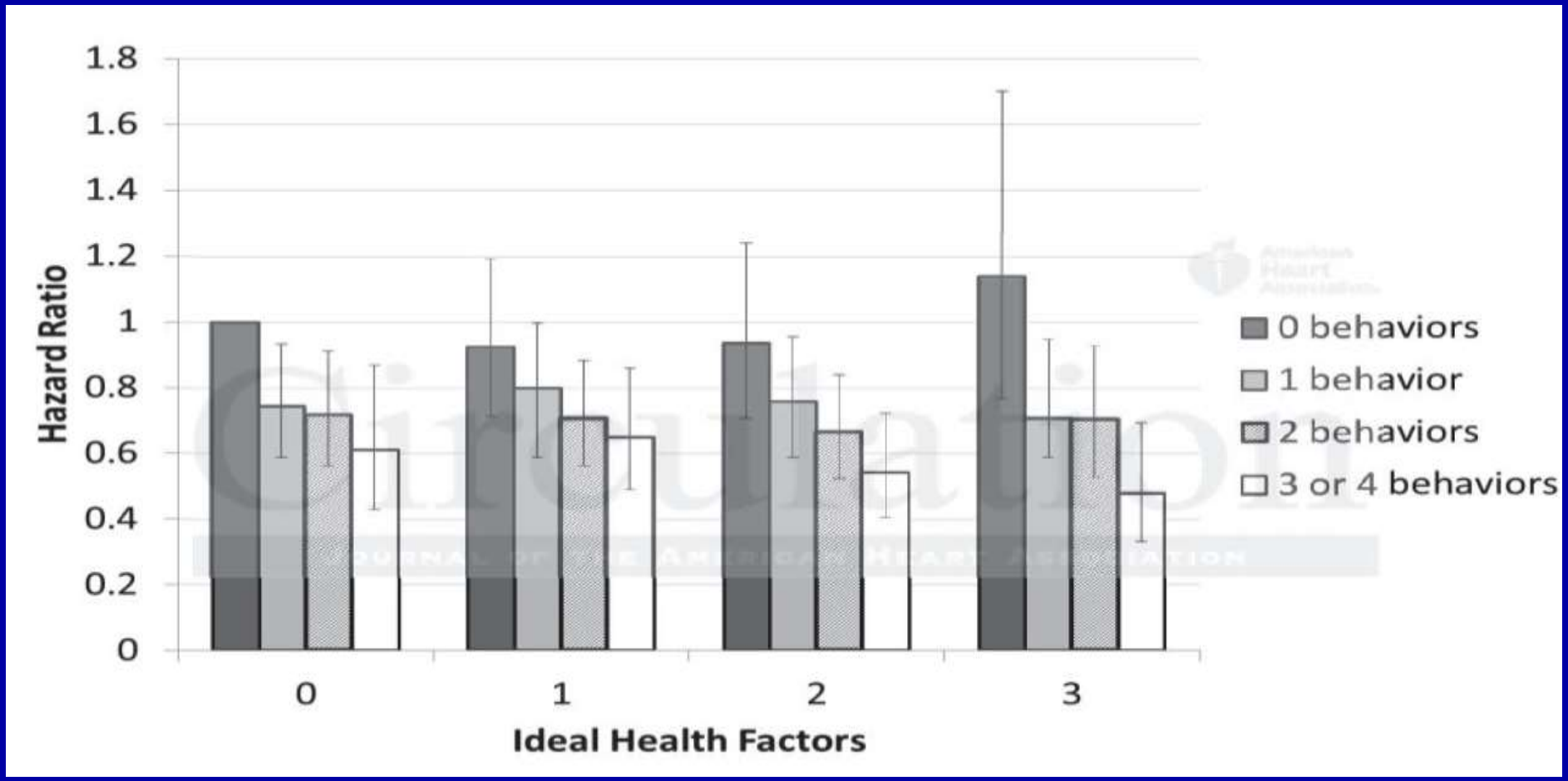
# Ideal health metrics	Total sample % (n= 13253)	# Cancer cases	Incidence rate per 1000 person-years*	Hazard Ratio (95% C.I)*†
0	2.8	95	17.3	1.0 (referent)
1	15.7	475	14.3	0.79 (0.64-0.98)
2	25.9	815	14.3	0.79 (0.64-0.98)
3	26.3	779	13.4	0.74 (0.59-0.91)
4	17.8	463	12.3	0.67 (0.54-0.84)
5	8.8	203	11.3	0.61 (0.48-0.79)
6-7	2.7	50	9.0	0.49 (0.35-0.69)

*adjusted for age, sex, race, and ARIC center

†trend test for this association: Hazard Ratio per 1 increase in number of ideal heath metrics = 0.92, p-trend < .0001

Rassmussen-Torvik, L., Shay, C., et al., Circulation March 18, 2013. ISSN 1524-4539

Cardiovascular Disease Prevention Reduces Cancer Risk!



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Cardiovascular Disease Prevention Reduces Cancer Risk!

# Ideal health metrics	Hazard Ratio for lung cancer (95% C.I)*†	Hazard Ratio for colorectal cancer (95% C.I)* †	Hazard Ratio for breast cancer (95% C.I)* †
0	1.0 (referent)	1.0 (referent)	1.0 (referent)
1	0.46 (0.30-0.73)	0.81 (0.41-1.59)	0.69 (0.42-1.16)
2	0.42 (0.28-0.65)	0.97 (0.50-1.86)	0.71 (0.43-1.17)
3	0.37 (0.24-0.57)	0.84 (0.44-1.63)	0.59 (0.36-0.98)
4	0.27 (0.17-0.44)	0.63 (0.31-1.25)	0.60 (0.36-1.00)
5	0.18 (0.09-0.33)	0.64 (0.30-1.37)	0.68 (0.40-1.16)
6-7	0.04 (0.01-0.27)	0.20 (0.04-0.91)	0.52 (0.26-1.03)

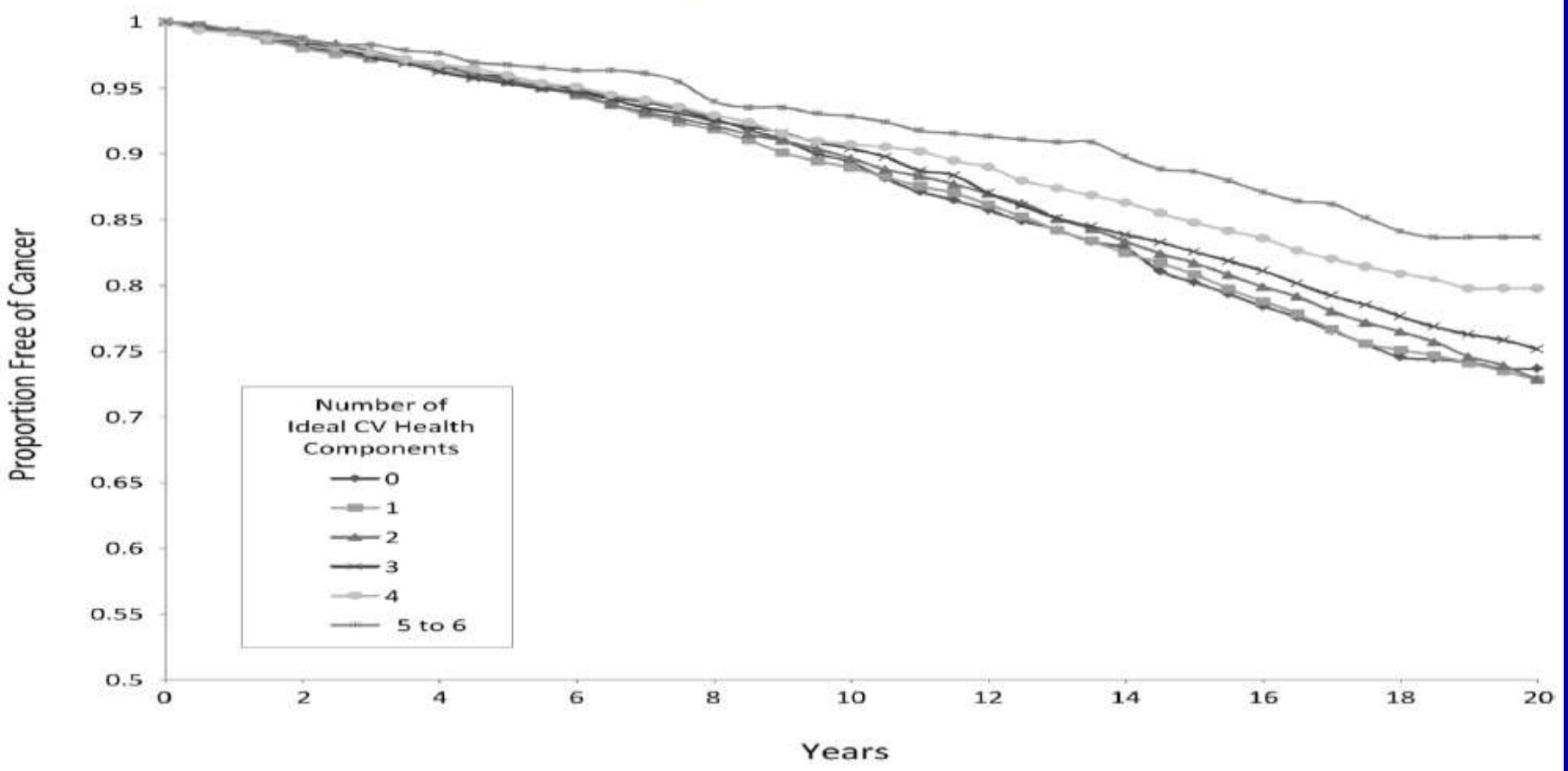
*adjusted for age, sex, race, and ARIC center

†p-trend for the lung cancer association < .0001 , p-trend for the colorectal cancer association = .0092 , p-trend for the breast cancer association = .11

Rassmussen-Torvik, L., Shay, C., et al., Circulation March 18, 2013. ISSN 1524-4539

Cardiovascular Disease Prevention Reduces Cancer Risk!

Supplementary Figure 1. Survival curves for combined cancer incidence by total number of ideal health metrics (with the ideal smoking metric omitted).



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Cardiovascular Disease Prevention Reduces Cancer Risk!

Having 6 or more ideal CV health metrics was associated with a 51% reduction in cancer risk!!!

Smoking was removed (due to suspicion that this was the main player of association) and cancer reduction remained statistically significant for cancer incidence. However smoking remains the most powerful modifiable health metric for CVD and cancer.

BD Take-Away: Optimal CV Prevention = Optimal Systemic Health. This is a good business model.

Rassmussen-Torvik, L., Shay, C., et al., Circulation March 18, 2013. ISSN 1524-4539

Have a great evening everyone!