Bale/Doneen Live Chat Session

Amy Doneen MSN, ARNP

August 14, 2013

5:30-6:30 pm PST

Title of today's "chat"
"BEST IN SHOW"

So many studies ……..which ones to select?

Outline for today's discussion

Red Flags:

BP higher in offspring of mothers with Pre-ecclampsia

CAD 2013 Update in Women

Disease:

Recidivism: 30-day prediction model

Root Causes:

Insulin Resistance – Predictors of CVA in IR patients

AF diagnosed with long term monitoring in CVA patients

Smoking and HR – Increased CVA Risk

Treatment:

Exercise – AHA 2013 Statement

Eating habits of men – importance of breakfast

Fiber reduces stroke risk

Prostate Cancer and Omega 3's

Omega's 3's reducing stroke risk in women

Sexual Counseling for patients with CVD

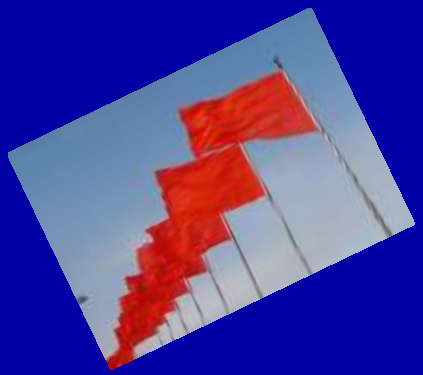
ACE-I and reduced cognitive Decline

Niacin not related to Ischemic Stroke – AIM-HIGH

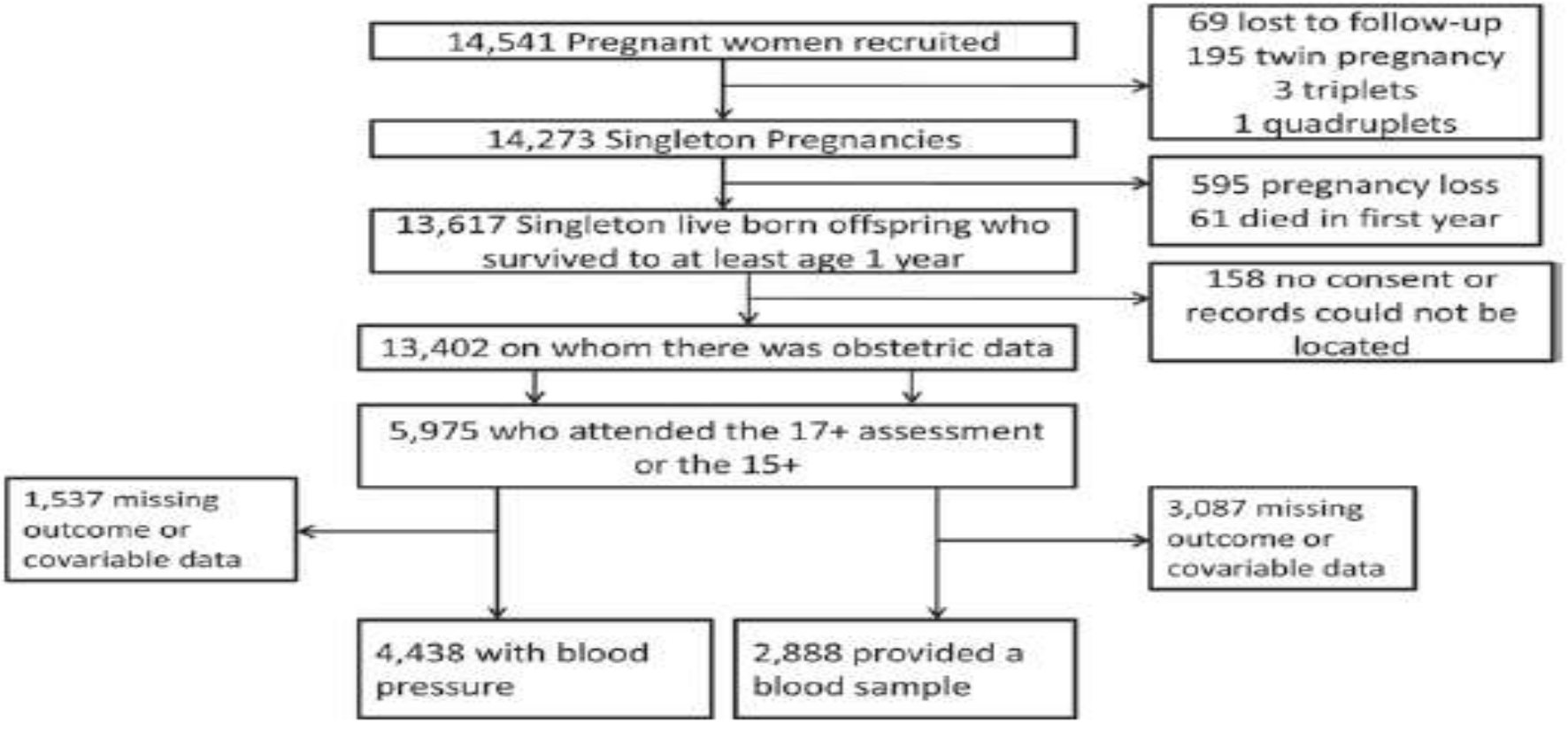
Red Flags

HTN in children of women with pre-ecclampsia

Fraser, A., Nelson, S., et al. (Aug 5, 2013). Hypertensive disorders of pregnancy and cardiometabolic health in adolescent offspring. *Hypertension*;62: OI10.1161.113.01513



Maternal hypertensive disorders of pregnancy (preeclampsia and gestational hypertension) associated with cardiometabolic health measures in adolescent offspring



Fraser, A., Nelson, S., et al. (Aug 5, 2013). *Hypertension*;62: OI10.1161.113.01513

Maternal hypertensive disorders of associated with cardiometabolic health measures in adolescent offspring

SBP and DBP higher in offspring of mothers with:

gestational hypertension

mean difference: 2.06 mmHg & 1.11 mmHg
(95% CI, 1.28-2.84 & CI, 0.54-1.69)

preeclampsia

mean difference: 1.12 mmHg and 1.71 mmHg
(95% CI, -0.89-3.12 & CI, 0.23-3.17)

compared with offspring of mothers without hypertensive disorders during pregnancy.

Fraser, A., Nelson, S., et al. (Aug 5, 2013). *Hypertension*;62: OI10.1161.113.01513

2013 Update: CAD in Women

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112.

CAD in Women: 2013 Update

Prevalence of CAD in Women:

CAD leading cause of death for men and women

Ave age of first MI for women – 70.3 yrs (64.5 for men)

Women have a 2-fold higher mortality rate after MI than men

In ages 45-64, women more likely to have heart failure within five years of MI

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Autoimmune diseases more prevalent in women (RA, SLE)

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CAD in Women: 2013 Update

Hormonal Changes:

1. Dysfunction in ovulation has been assoc. with CAD risk
2. PCOS - IR, pre-diabetes, metabolic syndrome
3. Functional hypothalamic amenorrhea – a cause of ovarian dysfunction – associated with premature CAD
4. Early age at menarche (<12 years) is associated with increased CAD events, CAD mortality & overall mortality.
5. Pre-ecclampsia – double risk of ischemic heart disease, stroke, and VTE over 5-10 years following the pregnancy.
6. Gestational DM – increases risk of DM
7. Gains from breast cancer treatment advances are being attenuated by increased CAD risk.

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

CAD in Women: 2013 Update

Undertreatment of CAD in Women:

1. In 1997 – only 30% of women were aware that CAD was the leading cause of death
2. In 2009 – this increased to 54%
3. In 2004 – fewer than 1 in 5 physicians recognized that more women than men die each year from CAD.
4. Women less likely to be treated to reach goal for LDL.
5. Female diabetics have the greatest sex disparity in achieving LDL targets.
6. Cardiac rehabilitation after MI is underused – women are 55% less likely to participate in cardiac rehab than men.

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

CAD in Women: 2013 Update

Risk Status	Criteria
High risk (≥1 high-risk state)	• Clinically manifest CHD • Clinically manifest cerebrovascular disease • Clinically manifest peripheral arterial disease • Abdominal aortic aneurysm • End-stage or chronic kidney disease • Diabetes mellitus • 10-year predicted CAD risk >10%

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

CAD in Women: 2013 Update

At risk (>1 risk factor)	• Cigarette smoking • SBP >120 mm Hg, DBP >80 mm Hg, or treated hypertension • Total cholesterol >200 mg/dl, HDL-C <50 mg/dl, or treated for dyslipidemia • Obesity, particularly central adiposity • Poor diet • Physical inactivity • Family history of premature CAD occurring in first-degree relatives in men <55 years of age or in women <65 years of age • Metabolic syndrome • Evidence of advanced subclinical atherosclerosis (e.g., coronary calcification, carotid plaque, or thickened IMT) • Poor exercise capacity on treadmill test and/or abnormal heart rate recovery after stopping exercise • Systemic autoimmune collagen-vascular disease (e.g., lupus or rheumatoid arthritis) • History of pre-eclampsia, gestational diabetes, or pregnancy-induced hypertension

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

CAD in Women: 2013 Update

Ideal coronary artery health (all of these)	• Total cholesterol <200 mg/dl (untreated) • BP <120/<80 mm Hg (untreated) • Fasting blood glucose <100 mg/dl (untreated) • Body mass index <25 kg/m^2 • Abstinence from smoking • Healthy (DASH-like) diet

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

CAD in Women: 2013 Update

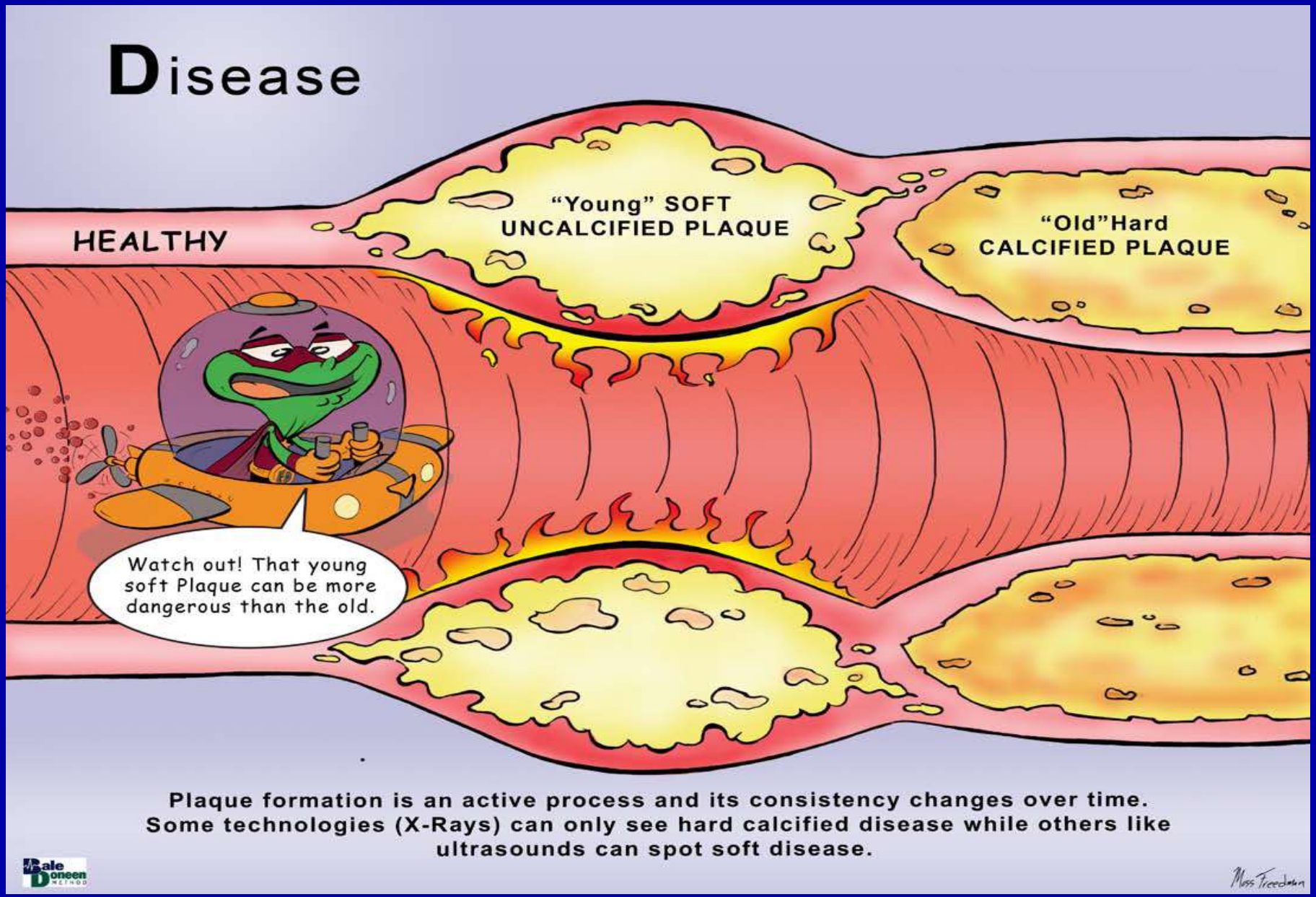
TABLE 2. Diagnostic value of various stress testing modalities in women

Stress Testing Modality	Sensitivity, %	Specificity, %	Negative Predictive Value	Positive Predictive Value
Exercise ECG [63–68]	31–71	66–78	78	47
Exercise echocardiography [68–70]	80–88	79–86	98	74
Exercise SPECT [71–74]	78–88	64–91	99	87
Pharmacological echocardiography [75–77]	76–90	85–94	68	94
Pharmacological SPECT [61,62,78]	80–91	65–86	90	68

ECG, electrocardiogram; SPECT, single-photon emission computed tomography.
Adapted, with permission, from Kohli and Gulati [60].

Sharma, K., Gulati, M. (2013). Coronary artery disease in women: a 2013 update. *Global Heart*. Vol 8, No 2.105-112

Disease

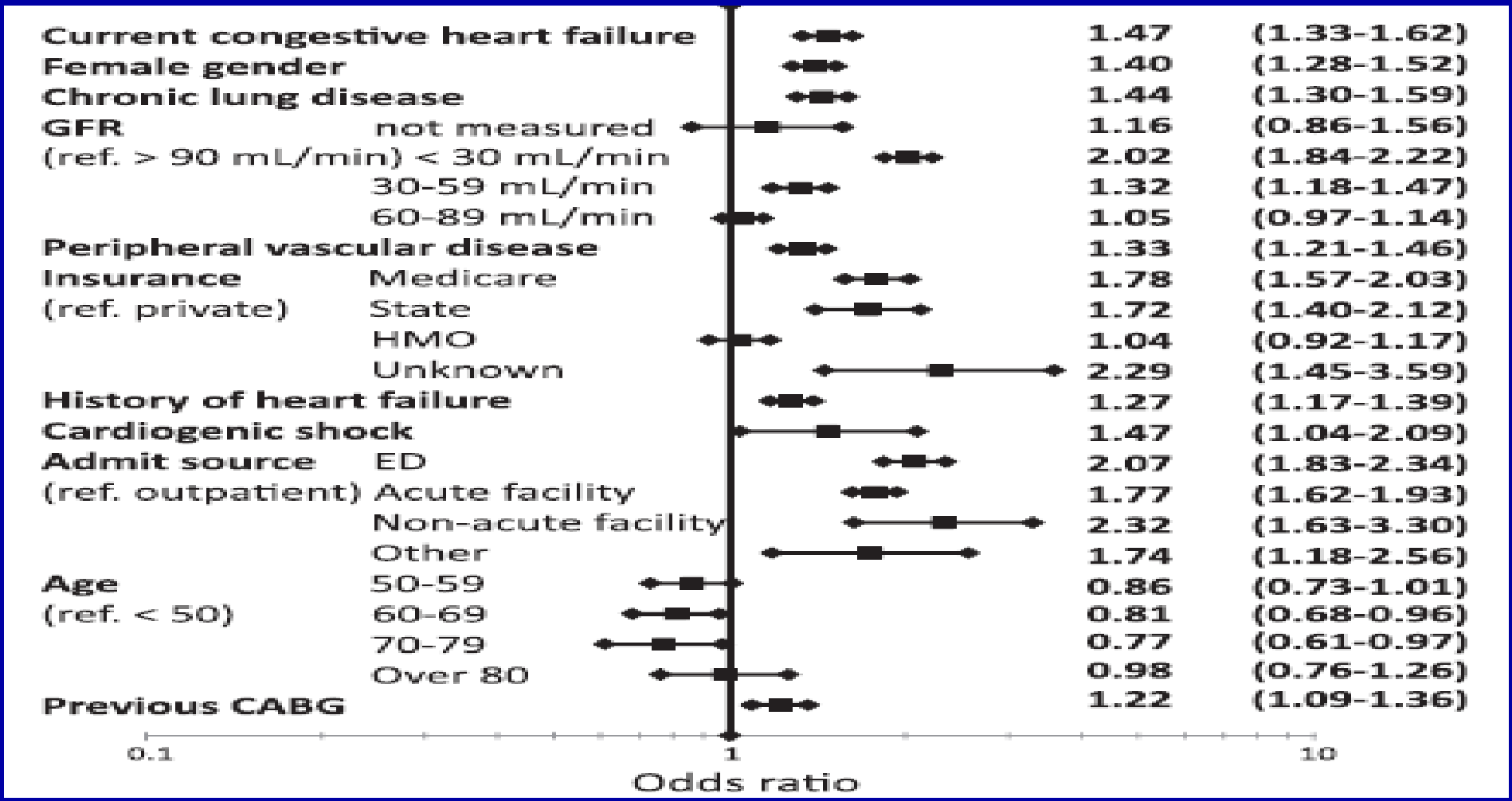


Plaque formation is an active process and its consistency changes over time. Some technologies (X-Rays) can only see hard calcified disease while others like ultrasounds can spot soft disease.

A Prediction Model to Identify Patients at High Risk for 30-Day Readmission After Percutaneous Coronary Intervention

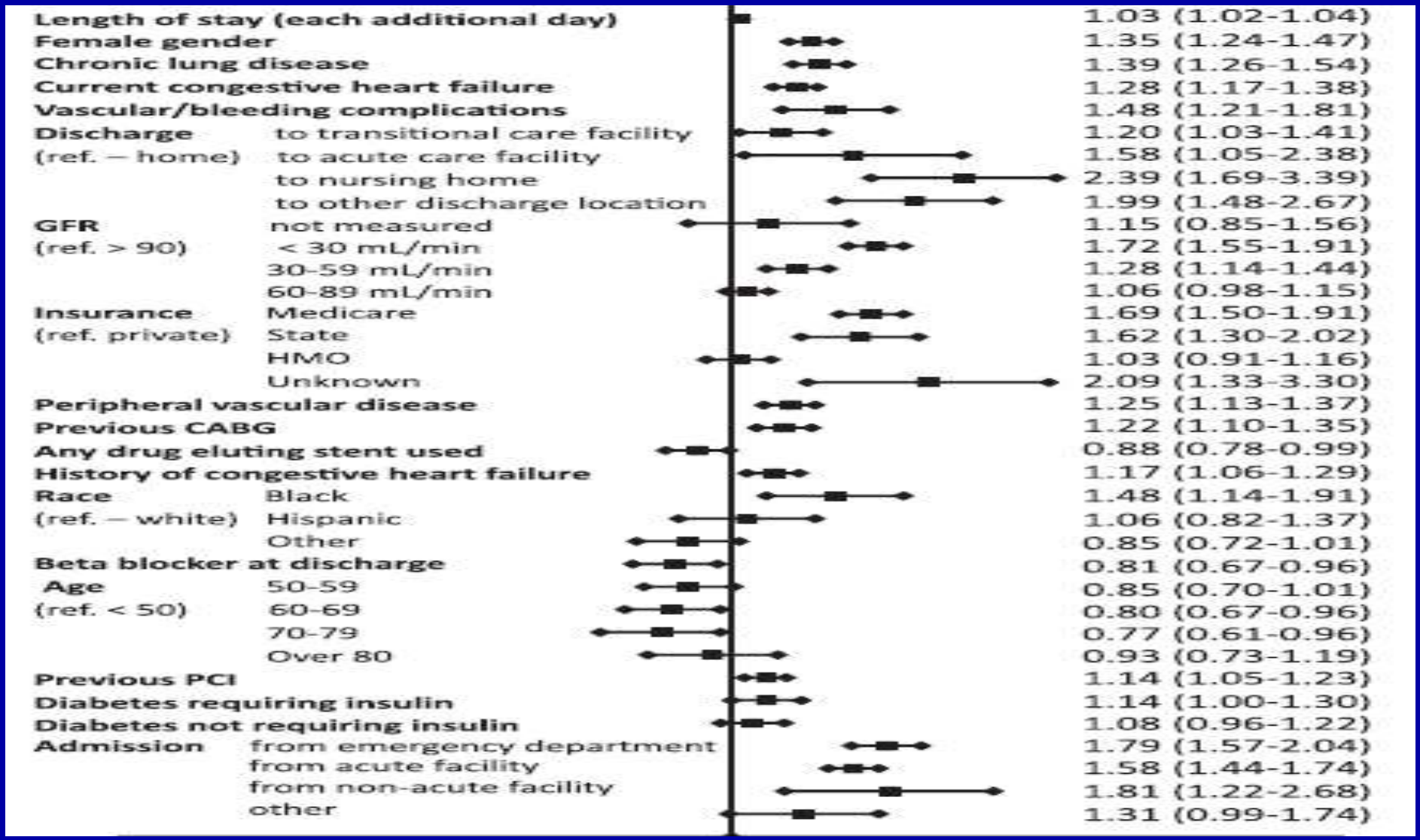
Wasfy, J., Rosenfield, K., et al. (2013). A prediction model to identify patients at high risk for 30-day readmission after percutaneous coronary intervention. *Circ Cardiovasc Qual Outcomes*. Vol 6(4):429-435

A Prediction Model to Identify Patients at High Risk for 30-Day Readmission After Percutaneous Coronary Intervention (pre-PCI Model)



Wasfy, J., Rosenfield, K., et al. (2013).. *Circ Cardiovasc Qual Outcomes*. Vol 6(4):429-435

A Prediction Model to Identify Patients at High Risk for 30-Day Readmission After Percutaneous Coronary Intervention (Discharge Model)



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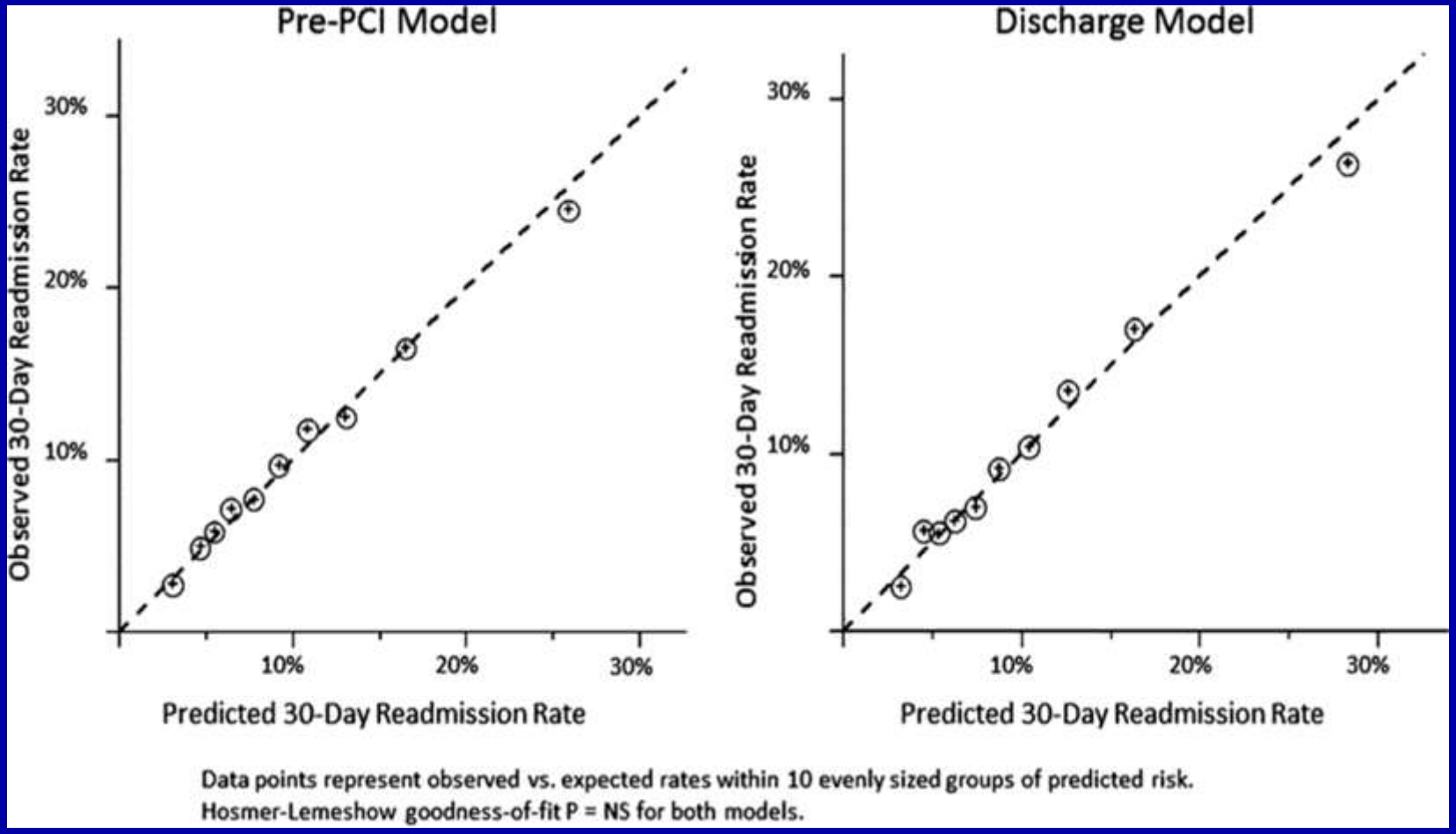
Observed vs predicted rates of 30-day readmission within validation cohort



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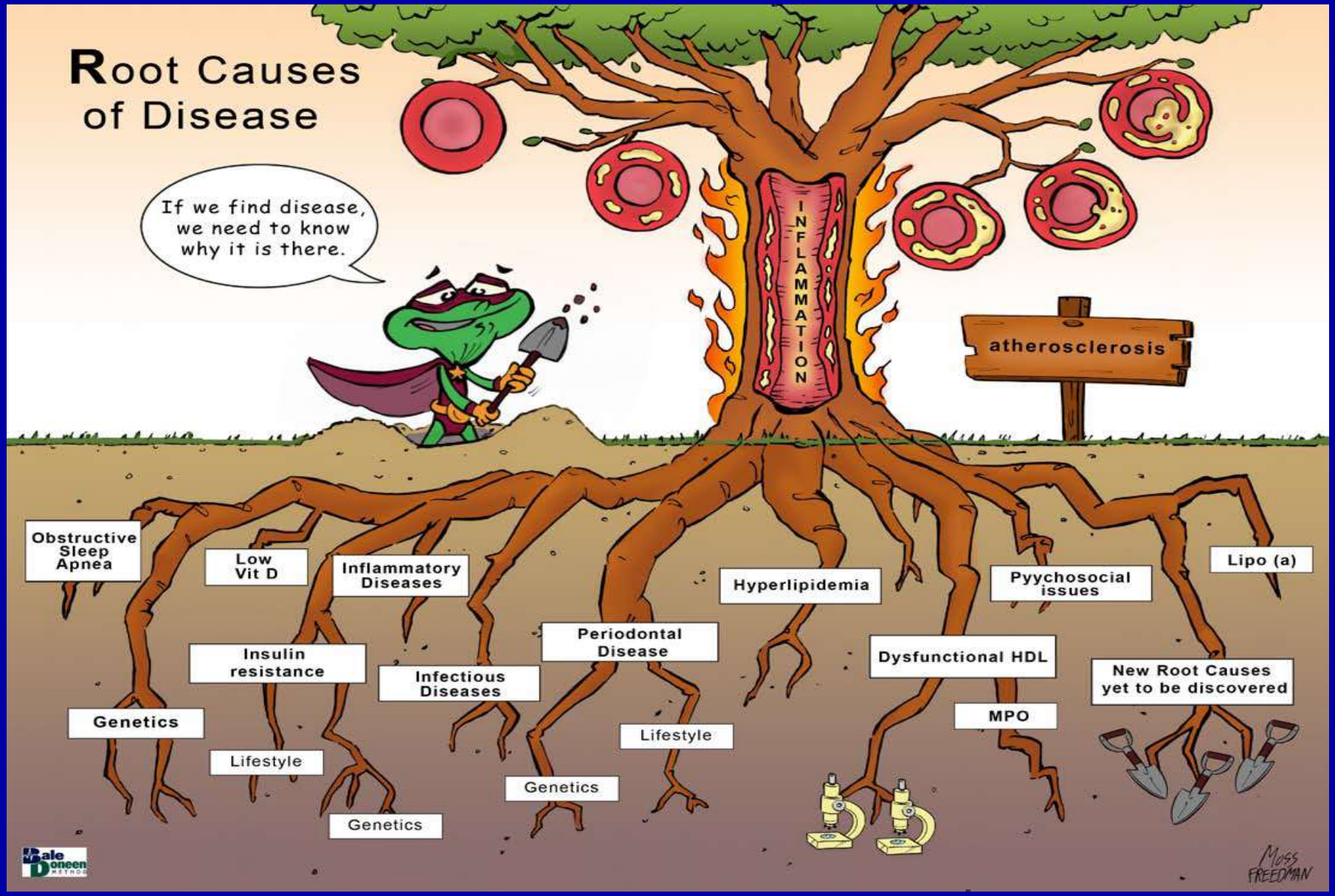


Predictors of stroke in IR patients

Preiss, D., Giles,T., et al. (2013). Predictors of stroke in patients with impaired glucose tolerances results from the nateglinide and valsartan in impaired glucose tolerance outcomes research trial. *Stroke*; 44.

Predictors of stroke in patients with impaired glucose tolerances

NAVIGATOR trial – 9306 participants, 237 strokes over 6.4 years. (202 nonhemorrhagic strokes)

Subjects without history of CVD (n=8570)

193 (2.3%) experienced a stroke

Subjects with history of CVD (n=736)

44 (6.0%) experienced a stroke

Independent predictors: PE or DVT (strongest predictor), previous CVA or TIA, higher pulse pressure and higher waist circumference.

No sugars were predictive.

Preiss, D., Giles,T., et al. (2013). *Stroke*; 44.

Predictors of stroke in patients with impaired glucose tolerances

Table 2. Predictors of Stroke in NAVIGATOR Participants in a Multivariable Cox Proportional-Hazard Stepwise Selection Model

Baseline Variable*	Hazard Ratio (95% CI)	*P* Value
Venous thromboembolism (pulmonary embolism/deep venous thrombosis)	4.09 (2.32–7.22)	<0.001
Cerebrovascular disease	2.15 (1.54–3.01)	<0.001
Pulse pressure (per 1 mm Hg)	1.02 (1.01–1.03)	<0.001
Waist circumference (per 10 cm)	1.28 (1.13–1.46)	<0.001
Coronary heart disease	1.67 (1.28–2.19)	<0.001
LDL cholesterol (per 1 mmol/L increase between 2.5 and 4.0 mmol/L)	1.44 (1.15–1.80)	0.001
eGFR (per 1 mL min^{-1} 1.73 m^{-2} up to 70 mL min^{-1} 1.73 m^{-2})	0.98 (0.96–0.99)	0.006
Heart rate (per bpm up to 70)	0.97 (0.96–0.99)	0.007
Body mass index (per 1 kg/m^2)	0.96 (0.92–0.99)	0.01
Age (per 10 y)	1.24 (1.02–1.51)	0.034
Atrial fibrillation	1.65 (1.04–2.63)	0.035
Black vs all other races	2.16 (1.01–4.65)	0.049
Valsartan vs no treatment	0.80 (0.62–1.03)	0.088
Nateglinide vs no treatment	0.88 (0.68–1.13)	0.31

Preiss, D., Giles,T., et al. (2013). *Stroke*; 44.

Cardiac Event Monitoring for AF after ischemic stroke

Higgins., P., MacFarlane., et al. (2013). Noninvasive cardiac event monitoring to detect atrial fibrillation after ischemic stroke: a randomized controlled trial. *Stroke*. ISSN.1524-4628.

Noninvasive cardiac event monitoring to detect atrial fibrillation after ischemic stroke

Randomized trial of 100 patients presenting in sinus rhythm with no AF history, within 7 days of ischemic stroke symptom onset.

Patients randomized to:

standard practice investigation (SP)

SP plus additional monitoring (SP-AM)

Primary outcome was detection of AF at 14 d

Patients were followed-up at 14 d and 90 d.

Higgins., P., MacFarlane., et al. (2013). Noninvasive cardiac event monitoring to detect atrial fibrillation after ischemic stroke: a randomized controlled trial. *Stroke*. ISSN.1524-4628.j

Noninvasive cardiac event monitoring to detect atrial fibrillation after ischemic stroke

Table 4. Differences Between Groups for Detection of Any-Duration PAF, Sustained PAF and Treatment with Anticoagulation at 14 and 90 Days

End Point	SP Group	SP-AM Group	Difference Between Groups	*P* Value
Any-duration PAF, 14 days	4% (0.0%–13.7%)	44% (30.0%–58.7%)	40% (25.2%–54.8%)	<0.001*
Any-duration PAF, 90 days	10% (3.3%–21.8%)	48% (33.7%–62.6%)	38% (21.8%–54.1%)	<0.001
Sustained PAF, 14 days	2% (0.0%–10.6%)	18% (8.6%–31.4%)	16% (4.7%–27.3%)	<0.05*
Sustained PAF, 90 days	8% (2.2%–19.2%)	22% (11.5%–36.0%)	14% (0.0%–27.7%)	0.09*
AC for any indication, 14 days	0% (0%–5.8%)	18% (8.6%–31.4%)	18% (7.4%–28.6%)	<0.01*
AC for any indication, 90 days	10% (3.3%–21.8%)	26% (14.6%–40.3%)	16% (1.2%–30.7%)	<0.05
AC for AF TE prophylaxis, 14 days	0% (0%–5.8%)	16% (7.2%–29.1%)	16% (5.8%–26.2%)	<0.01*
AC for AF TE prophylaxis, 90 days	6% (1.3%–16.5%)	22% (11.5%–36.0%)	16% (2.8%–29.2%)	<0.05*

Values quoted are% (95% CI). Comparison between groups is with difference in 2 proportions. When observed frequencies were low, Fisher exact test (*) was used. A *P* value of <0.05 was considered statistically significant. AC indicates anticoagulation; CI, confidence interval; PAF, paroxysmal atrial fibrillation; SP, standard practice; SP-AM, standard practice plus additional monitoring; and TE, thromboembolic.

Higgins., P., MacFarlane., et al. (2013). Noninvasive cardiac event monitoring to detect atrial fibrillation after ischemic stroke: a randomized controlled trial. *Stroke*. ISSN.1524-4628.j

Smoking & HR related to Ischemic Stroke

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: a population-based prospective cohort study among inner mongolians in china. *Stroke*. ISSN:1524-4628.

Smoking & Heart Rate are independent risk factors for ischemic stroke.

Prospective cohort study June 2003-July 2012 conducted among 2530 people ≥ 20 years of age from Mongolia, China.

Followed for 9.2 years – total of 23, 292 person-years observed

4 subgoups: related to smoking, heart rate & ischemic stroke. (95% Confidence intervals for all)

Nonsmokers with HR ≥ 80 bpm	1.42 (0.62-3.28)
Smokers with HR < 80 bpm	2.11 (1.06-4.23)
Smoker with HR ≥ 80 bpm	2.86 (1.33-6.14)
Non smokers with HR < 80 bpm	Comparative Group

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: *Stroke*. ISSN:1524-4628

Smoking, HR and Ischemic Stroke

Table 1. Baseline Characteristics of 2530 Participants According to Heart Rate/Smoking Status in Inner Mongolia, China

	Heart Rate <80 bpm Nonsmokers	Heart Rate ≥80 bpm Nonsmokers	Heart Rate <80 bpm Smokers	Heart Rate ≥80 bpm Smokers	*P* Value
n	851	558	757	364	
Age	44.6±12.3	44.0±12.5	48.4±12.4	50.4±12.2	<0.0001*
Male, %	32.1	18.3	64.6	50.0	<0.0001*
Drinking, %	23.5	23.5	39.4	43.6	<0.0001†
Family history of CVD, %	11.6	16.0	10.8	17.2	0.03†
Systolic blood pressure, mm Hg	129.6±22.2	133.2±22.7	126.4±22.8	130.9±22.3	<0.0001†
Diastolic blood pressure, mm Hg	84.2±12.0	87.0±12.5	82.3±12.7	85.7±12.2	<0.0001†
Body mass index, kg/m^2	22.9±3.5	22.7±3.5	21.9±3.6	20.9±3.4	<0.0001†
Fasting glucose, mmol/L	4.90±1.17	5.32±1.18	4.74±1.24	5.19±1.20	<0.0001†
Total cholesterol, mmol/L	3.75±1.16	3.71±1.18	3.73±1.16	3.78±1.13	0.83†
Triglycerides, mmol/L	1.14±1.17	1.40±1.42	1.22±1.38	1.45±1.36	<0.0001†
HDL cholesterol, mmol/L	1.18±0.32	1.15±0.33	1.19±0.33	1.18±0.32	0.37†
LDL cholesterol, mmol/L	2.35±1.02	2.28±1.04	2.30±1.05	2.31±1.03	0.66†

CVD indicates cardiovascular disease; HDL, high-density lipoprotein; and LDL, low-density lipoprotein.
*These *P* values were mutually adjusted for sex and age.
†These *P* values were adjusted for age and sex.

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: *Stroke*. ISSN:1524-4628

Smoking, HR and Ischemic Stroke

Table 2. Age- and Sex-Adjusted and Multivariable-Adjusted Hazard Ratios for Ischemic Stroke Incidence According to Heart Rate/Smoking Status

			Age and Sex Adjusted			Multivariable Adjusted*		
	Cases	Person-Years	Hazard Ratio	95% Confidence Interval		Hazard Ratio	95% Confidence Interval	
Heart rate <80 bpm/nonsmokers	12	7948	1.00 (reference)			1.00 (reference)		
Heart rate ≥80 bpm/nonsmokers	11	5227	1.44	0.64	3.28	1.42	0.62	3.28
Heart rate <80 bpm/smokers	30	6878	1.66	0.84	3.28	2.11	1.06	4.23
Heart rate ≥80 bpm/smokers	21	3239	2.66	1.30	5.42	2.86	1.33	6.14

*Multivariable model adjusted for age, sex, body mass index, drinking status, family history of cardiovascular disease, blood glucose, systolic blood pressure, diastolic blood pressure, and lipids.

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: *Stroke*. ISSN:1524-4628

Smoking, HR and Ischemic Stroke

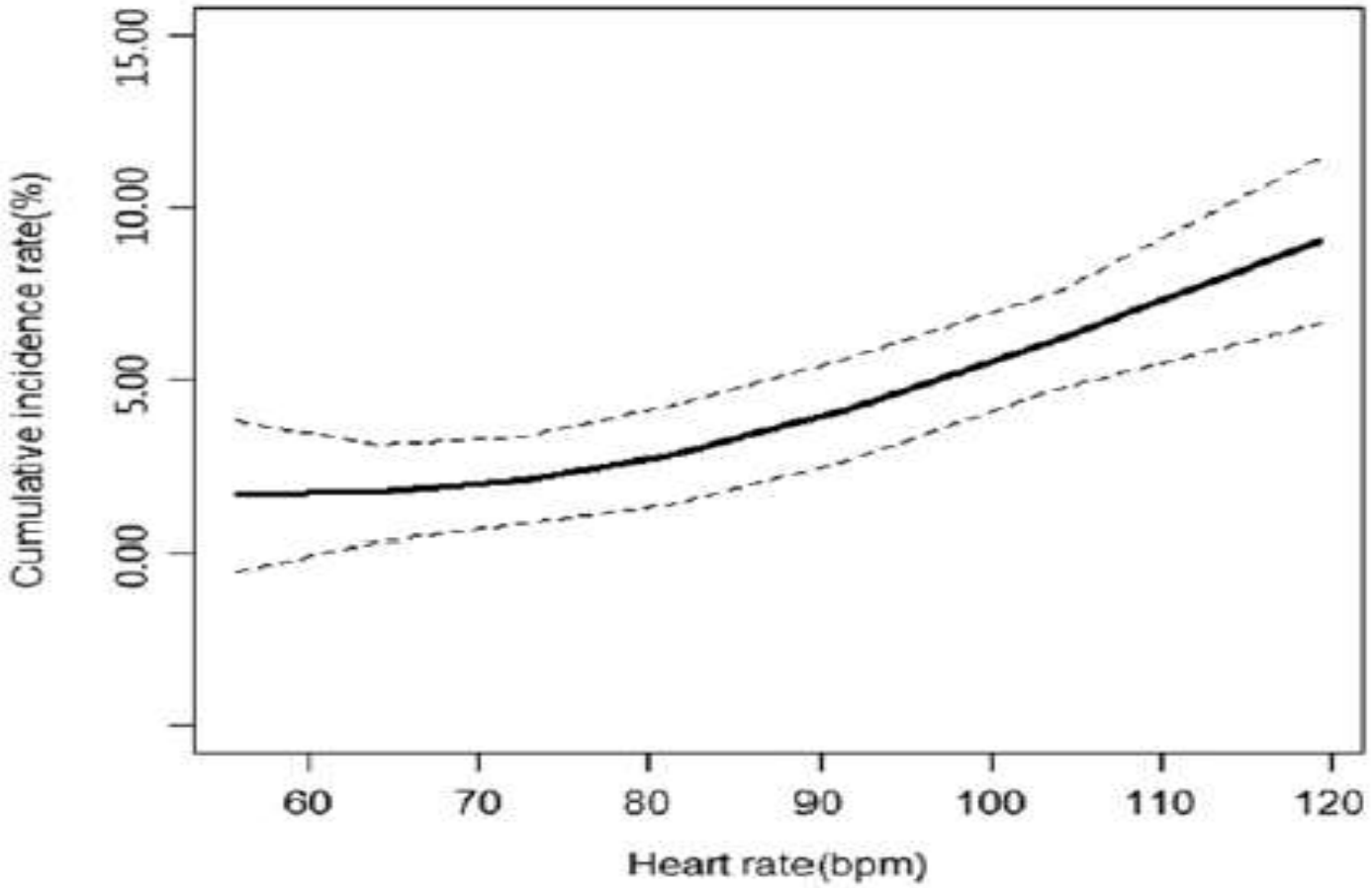


Figure 1. Fitted curve of cumulative incidence rates (solid line) and their 95% confidence intervals (dotted line) for each 10-bpm heart rate interval in an Inner Mongolian population.

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: *Stroke*. ISSN:1524-4628

Smoking, HR and Ischemic Stroke

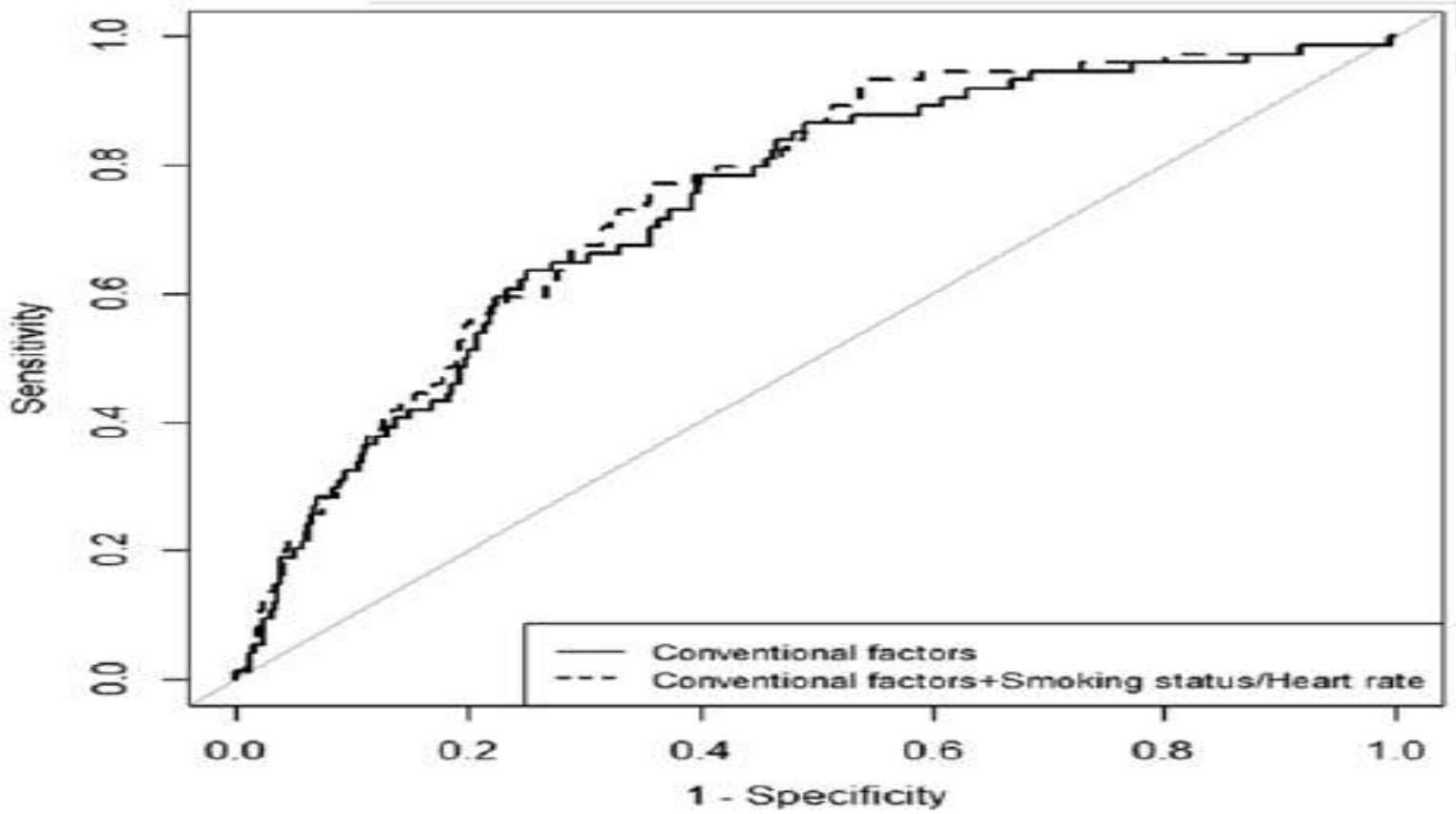
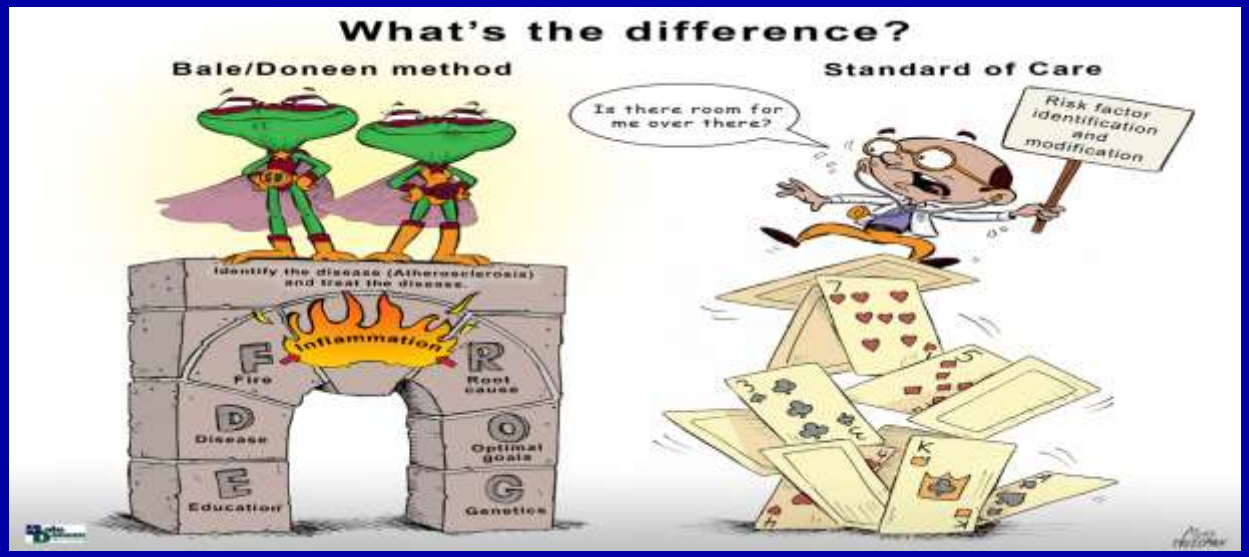


Figure 2. Area under the curve for the prediction of ischemic stroke incidence for baseline conventional risk factors and for the addition of smoking status/heart rate. Risk factors in the conventional model include age, sex, body mass index, drinking status, family history of cardiovascular disease, blood glucose, blood pressure, and lipids.

Tian, Xu, Yiaoqing, Bu, et al. (2013). Smoking, heart rate, and ischemic stroke: *Stroke*. ISSN:1524-4628

Treatment



Exercise Standards 2013 AHA

Fletcher, G., Ades, P., et al. (2013). Exercise standards for testing and training a scientific statement from the american heart association. Circulation: DOI: 10.1161 August 20 2013.

2013 Exercise statement from AHA

Previous standards statement was 2001

2013 standards intended for use by physicians, nurses, exercise physiologists and specialists, technologists and other healthcare providers involved in exercise testing and training.

<u>For our purpose – Review</u>:

Exercise Testing

General Guidelines for Exercise Training

Behavioral change modification

Exercise and Inflammation

Fletcher, G., Ades, P., et al. (2013). Exercise standards for testing and training a scientific statement from the american heart association. Circulation: DOI: 10.1161 August 20 2013.

2013 Exercise statement from AHA

Purposes of Exercise Testing

1. Detection of coronary artery disease (CAD) in patients with chest pain. (chest discomfort) syndromes or potential symptom equivalents.
2. Evaluation of the anatomic and functional severity of CAD.
3. Prediction of cardiovascular events and all-cause death.
4. Evaluation of physical capacity and effort tolerance.
5. Evaluation of exercise-related symptoms.
6. Assessment of chronotropic competence, arrhythmias, and response to implanted device therapy.
7. Assessment of the response to medical interventions.

Fletcher, G., Ades, P., et al. (2013). Exercise standards for testing and training a scientific statement from the american heart association. Circulation: DOI: 10.1161 August 20 2013.

AHA Exercise Statement

- Meta-analyses suggest that exercise-based cardiac rehabilitation reduces total deaths, CV deaths, and hospital readmissions by roughly 25% in post MI pts
- Only 14% to 35% of qualified pts are referred to rehab after MI and ≈31% after CABG
- Nearly all pts with CAD can benefit from an individualized exercise training regimen

Fletcher, G. F., et. al. (2013). *Circulation*. doi: 10.1161/CIR.0b013e31829b5b44

2013 Exercise statement from AHA

Table 7. General Guidelines for Endurance and Resistance Training

Endurance training	
Frequency	≥5 d/wk
Intensity	55%–90% maximum predicted HR* or 40%–80% Vo_2max or HR reserve RPE 12–16
Modality	Walking, treadmill, cycling, etc
Duration	30–60 min
Resistance training	
Frequency	2–3 d/wk
Intensity	50%–80% of 1-RM or RPE 12–16 1–3 sets of 8–15 repetitions per exercise
Modality	Lower extremity: leg extensions, leg curls, leg press. Upper extremity: bench press, lateral pulldowns, biceps curl, triceps extension
Duration	30–45 min

HR indicates heart rate; maximum predicted HR=(220–age); RPE, rating of perceived exertion; and 1-RM, single-repetition maximal lift.

*The HR range recommendation assumes that the individual is not taking β-adrenergic–blocking medications. Modified from Shephard and Balady.[491a]

Fletcher, G., Ades, P., et al. (2013). Exercise standards for testing and training a scientific statement from the american heart association. Circulation: DOI: 10.1161 August 20 2013.

AHA Exercise Statement

Components of behavior change and self-regulation:

1. Setting of realistic and simple goals
2. Self-monitoring of personal behaviors linked to goal attainment
3. Feedback about progress toward goals
4. Self-evaluation of progress
5. Corrective behavior leading to effective movement toward goals

Fletcher, G. F., et. al. (2013). *Circulation*. doi: 10.1161/CIR.0b013e31829b5b44

AHA Exercise Statement

Exercise and Inflammation:

The impact of exercise training on fitness and ultimately on risk of death appears to be attributable to enhanced fibrinolysis, improved endothelial function, decreased sympathetic tone, and likely other factos.

B/D: Autophagy and Senescence (arterial inflammation) !!

Fletcher, G. F., et. al. (2013). July 22, *Circulation*. doi: 10.1161/CIR.0b013e31829b5b44

Eating Breakfast reduces CHD risk in men

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Eating Breakfast and incident CHD in men

Skipping meals is associated with excess body weight, hypertension, IR and elevated lipids.

Trial aimed to prospectively examine eating habits and risk of CHD.

Eating habits, including breakfast eating, assessed in 1992 in 26,902 American men 45 to 82 years of age from the Health Professionals Follow-up Study who were free of CVD and Cancer.

16 year follow-up, 1527 incident CHD cases.

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Eating Breakfast and incident CHD in men

Men who skipped breakfast had a 27% higher risk of CHD compared with men who did not eat breakfast!

(relative risk, 1.27; 95% CI, 1.06-1.53).

Compared with men who did not eat late at night, those who ate late at night had a 55% higher CHD risk.

(relative risk, 1.55; 95% CI, 1.05-2.29).

Adjusted for: BMI, HTN, hyperlipidemia, diabetes.

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Eating Breakfast and incident CHD in men

Table 2. Eating Breakfast and Multivariate RR of CHD With 95% CIs

	Breakfast		
	Yes	No	*P* Value
Cases, n	1356	171	
Person-years	338 074	49 880	
Age-adjusted model: RR (95% CI)	1.00 (Referent)	1.33 (1.13–1.57)	0.0008
+Diet factors*	1.00 (Referent)	1.38 (1.15–1.66)	0.0006
+Demographic factors†	1.00 (Referent)	1.29 (1.07–1.55)	0.007
+Activity factors‡	1.00 (Referent)	1.27 (1.06–1.53)	0.01
Adjustment for potential mediators			
+BMI§	1.00 (Referent)	1.23 (1.02–1.48)	0.03
+Health conditions‖	1.00 (Referent)	1.21 (1.00–1.46)	0.05
+BMI and health conditions	1.00 (Referent)	1.18 (0.98–1.43)	0.08

BMI indicates body mass index; CHD, coronary heart disease; CI, confidence interval; and RR, relative risk.

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Eating Breakfast and incident CHD in men

Table 3. Late-Night Eating and Multivariate RR of CHD With 95% CIs

	Late-Night Eating		
	No	Yes	*P* Value
Cases, n	1498	29	
Person-years	383 584	4370	
Age-adjusted model: RR (95% CI)	1.00 (Referent)	1.61 (1.10–2.36)	0.01
+Diet factors*	1.00 (Referent)	1.59 (1.08–2.35)	0.02
+Demographic factors†	1.00 (Referent)	1.55 (1.05–2.28)	0.03
+Activity factors‡	1.00 (Referent)	1.55 (1.05–2.29)	0.03
Adjustment for potential mediators			
+BMI§	1.00 (Referent)	1.53 (1.04–2.25)	0.03
+Health conditions‖	1.00 (Referent)	1.41 (0.95–2.10)	0.08
+BMI and health conditions	1.00 (Referent)	1.41 (0.95– 2.08)	0.09

BMI indicates body mass index; CHD, coronary heart disease; CI, confidence interval; and RR, relative risk.

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Eating Breakfast and incident CHD in men

Table 5. Comparison of Eating Habits and Multivariate RR of Coronary Heart Disease With 95% CIs Obtained Using Different Methods for Approaching Missing Covariate Data

	Method for Missing Covariate Data		
	Missing Indicator	Multiple Imputation	Complete Case*
Risk Factor	RR (95% CI)	RR (95% CI)	RR (95% CI)
Skipping breakfast†	1.27 (1.06–1.53)	1.29 (1.07–1.56)	1.25 (1.03–1.51)
Late-night eating‡	1.55 (1.05–2.29)	1.53 (1.01–2.32)	1.52 (1.01–2.29)
Eating frequency, times/d			
1–2	1.10 (0.91–1.31)	1.17 (0.86–1.58)	1.08 (0.79–1.47)
3	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
4–5	1.05 (0.94–1.18)	1.05 (0.79–1.38)	1.11 (0.84–1.47)
≥6	1.26 (0.90–1.77)	1.21 (0.56–2.61)	1.57 (0.72–3.42)

Cahill, L., Chiuve, S., et al. (2013). Prospecitve study of breakfast eating and incident coronary heart disease in a cohort of male US health professionals. Circulation;128:337-343.

Dietary Fiber intake decreases risk of first stroke.

Threapleton, D., Greenwood., D., et al. (2013). Dietary fiber intake and risk of first stroke: a systematic review and meta-analysis. Stroke; 44:1360-1368.

Dietary Fiber and Risk of First Stroke

Meta-analysis to determine fiber intake and incidence of first hemorrhagic or ischemic stroke (lit review – 1990-2012) – 8 cohort studies met criteria

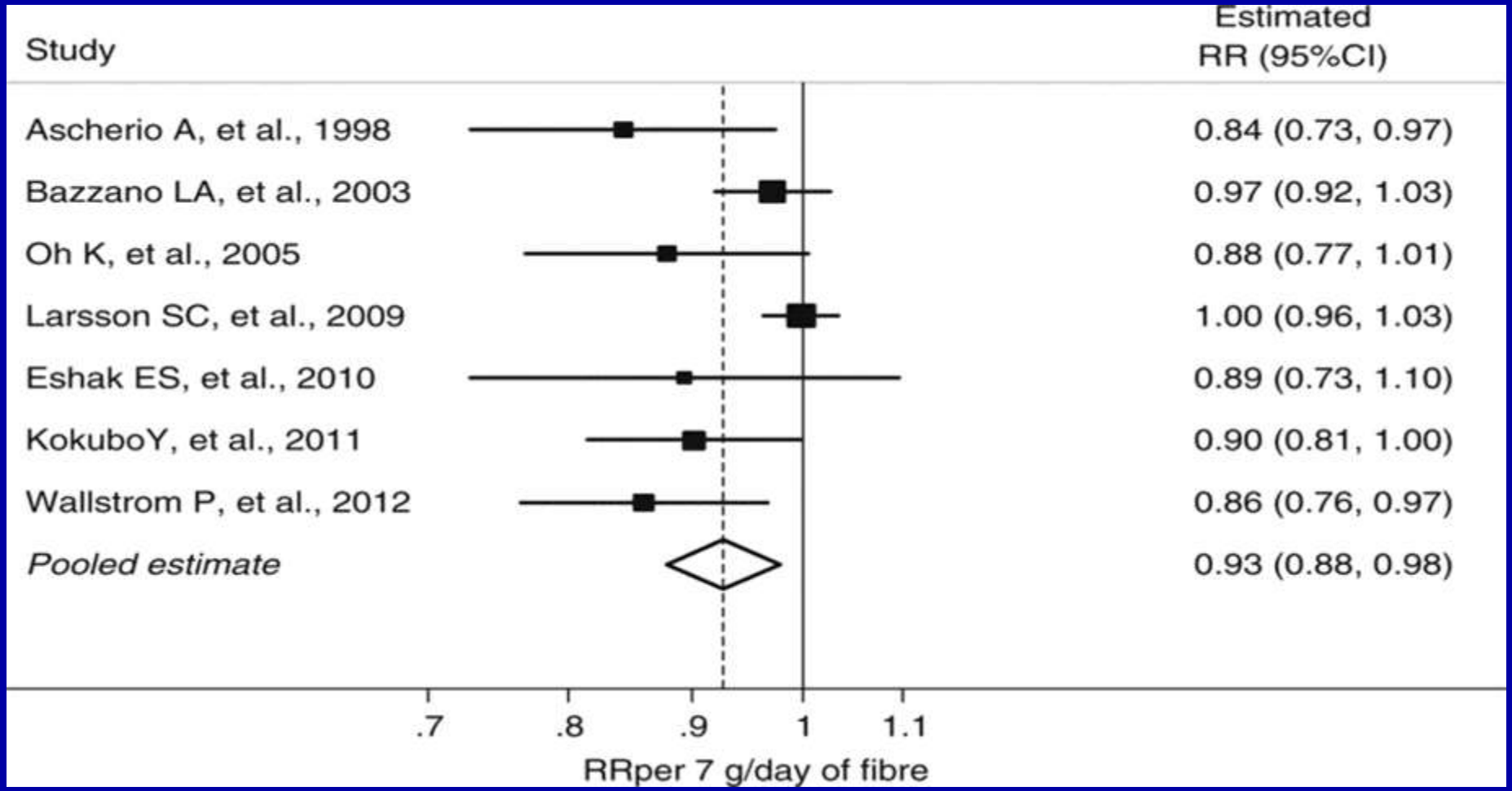
Total dietary fiber intake was inversely associated with risk of hemorrhagic plus ischemic stroke.

Fiber: 7g/day, relative risk 0.93; 95% CI, 0.88-0.98.

Fiber: 4g/day – not assoc. with stroke risk reduction

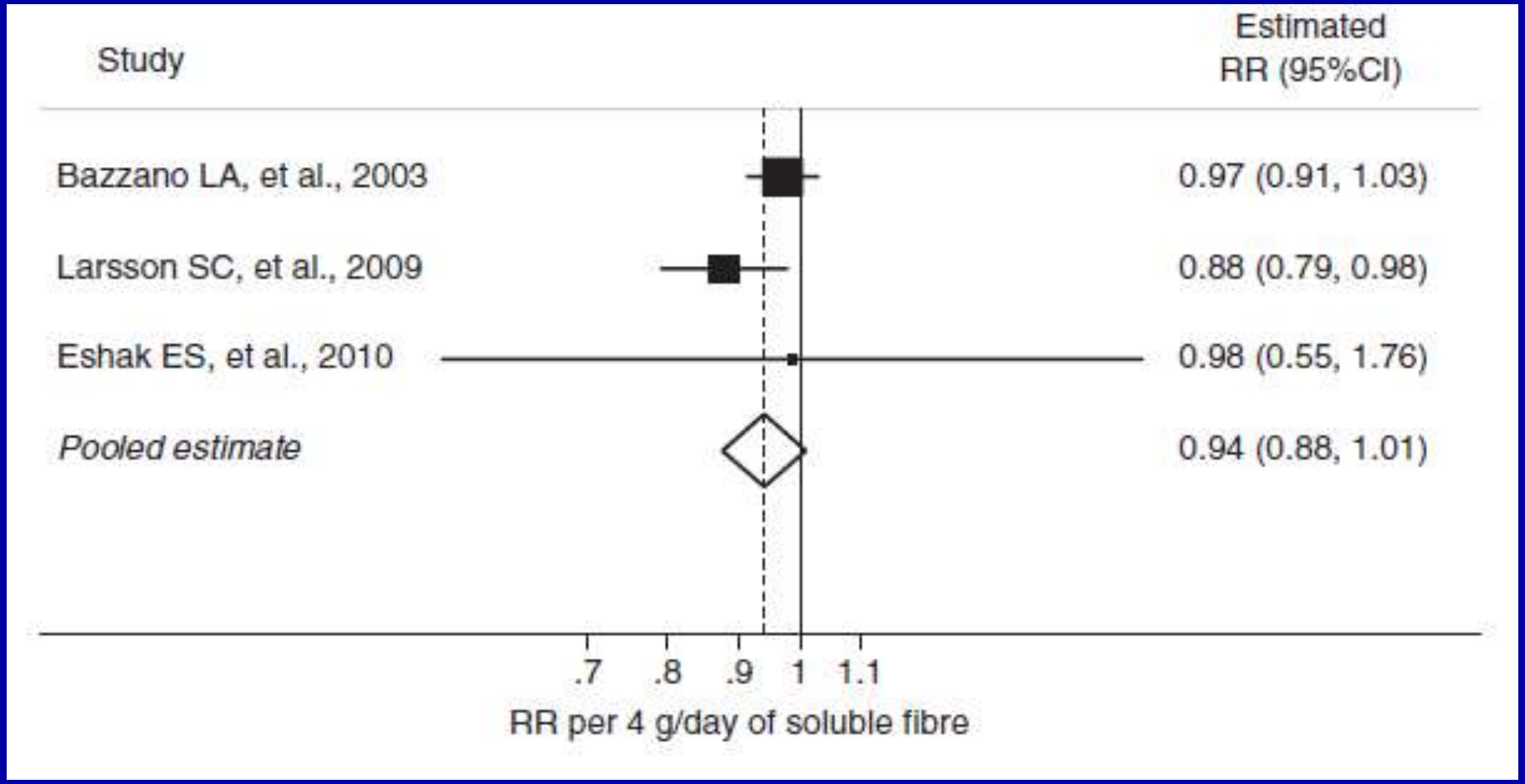
Threapleton, D., Greenwood., D., et al. (2013). Dietary fiber intake and risk of first stroke: a systematic review and meta-analysis. Stroke; 44:1360-1368.

Stroke Risk reduction at 7gm/day of soluble fiber



Threapleton, D., Greenwood., D., et al. (2013). Dietary fiber intake and risk of first stroke: a systematic review and meta-analysis. Stroke; 44:1360-1368.

Stroke Risk reduction at 4gm/day of soluble fiber



Threapleton, D., Greenwood., D., et al. (2013). Dietary fiber intake and risk of first stroke: a systematic review and meta-analysis. Stroke; 44:1360-1368.

Omega 3 and Prostate Cancer?

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M.,Parnes, H. L. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*

Omega 3's and Prostate CA Risk: SELECT

- All subjects came for SELECT trial: a randomized, placebo-controlled trial testing whether selenium and or vitamin E reduced prostate CA risk
- Entry was July 2001 through May 2004 -35,533 men
- Halted Sept. 2008; reported in 2011 vit. E vs placebo increases prostate CA 17%

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M., . . . Parnes, H. L. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

JOURNAL OF Arteriology
PRODUCED BY THE BALE/DONEEN METHOD
VOLUME 4, AUGUST 2013

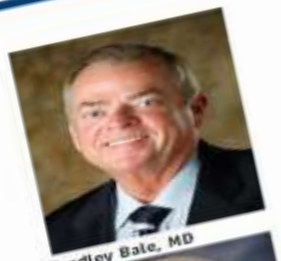
7/18/2013:

THIS WHOLE THING SEEMS FISHY!

HERE ARE THE HEADLINES...

"Omega-3 supplements linked to prostate cancer" — *Fox News*

"Hold the salmon: Omega 3 fatty acids linked to higher risk of cancer" — *CNN*


Bradley Bale, MD


Amy Doneen, MSN, ARNP

Abstract details:

Background: Studies of dietary n-3 fatty acid intake and prostate cancer risk are inconsistent; however, recent large prospective studies have found increased risk of prostate cancer among men with high blood concentrations of long-chain n-3 polyunsaturated fatty acids. This case-cohort study examines associations between plasma phospholipid fatty acids and prostate cancer risk among participants in the Selenium and Vitamin E Cancer Prevention Trial.

Methods:

Case subjects were 834 men diagnosed with prostate cancer, of which 156 had high-grade cancer. The subcohort consisted of 1393 men selected randomly at baseline and from within strata frequency matched to case subjects on age and race. Proportional hazards models estimated ratios (HR) and 95% confidence intervals ions between fatty acids and prostate cancer risk overall and by grade. All statistical tests were two-sided.

Results:

Compared with men in the lowest quartiles of LC 3PUFA, men in the highest quartile had increased risk for low-grade (HR 1.44, 95% CI 1.08 to 1.93), high grade (HR 1.71, 95% 1.00 to 2.94), and total prostate cancer (HR 1.43, 95% CI 1.09-1.88). Associations were similar for individual long-chain n-3 fatty acids. Higher linoleic acid (n-6) was associated with reduced risks of low-grade (HR 0.75, 95% CI 0.56-0.99) and total prostate cancer (HR 0.77, 95% CI 0.59-1.01); however, there was no dose response.

Conclusions:

This study confirms previous reports of increased prostate cancer risk among men with high blood concentrations of LC 3PUFA. The consistency of these findings suggests that these fatty

Bale Doneen METHOD

Omega 3's and Prostate CA Risk: SELECT

Brasky, T. et al, (July 2013). Plasma Phospholipid Fatty Acids and Prostate Cancer Risk in the SELECT Trial. Journal of the National Cancer Institutute

This article certainly merits attention because it challenges our longstanding recommendations to eat a healthy Mediterranean diet rich in Omega 3 nutrients and low in saturated fats.

Additionally, in some cases, we utilize Omega-3 supplementation to treat vascular inflammation (OCEAN trial), plaque stabilization (COMBOS) and prevention of vascular events and death (JELIS and GISSI).

This research (Brasky et at), is challenging from several vantage points – statistically, methodologically and intuitively.

This list is NOT meant to be conclusive but certainly raises points of concern.

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M., . . . Parnes, H. L. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Omega 3's and Prostate CA Risk:SELECT

Methodologically –

1. This is an observational trial – NO cause/effect was reported.
2. We do not know how many of the men in this trial had prostate cancer BEFORE the trial was initiated.
3. This was NOT a trail to test supplement intake – no supplements or dietary intake were evaluated.
4. No documentation was provided regarding intake of fish or Omega-3 supplements were recorded in the trial.
5. Absolute levels of EPA, CHA, DPAn-3 were not reported.

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M., . . . 4. nes, H. L. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Omega 3's and Prostate CA Risk: SELECT

Statistically –

The difference in mean blood plasma phospholipid fatty acids blood level for omega-3 was 4.66% in the cancer group vs 4.48% in the non-cancer case matched group. control arm. The association between Omega-3 and prostate cancer is being based on a 0.2% difference in Omega-3 levels. Some experts have commented that the levels in both groups are well below what would be expected with supplemental omega-3.

Co-founders for prostate cancer were not controlled in this observational study – this data also demonstrated that the prostate cancer positive subjects were smokers (53%), regularly consumed alcohol (64%), had a first degree relative with prostate cancer (30%), and were obese (80%). These confounding variables were not controlled for in the study.

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M., . . . Parnes, H. L. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Omega 3's and Prostate CA Risk: SELECT

Intuitively –

If Omega 3 fatty acid increased prostate cancer, countries with high fish intake would likely show an increase prostate cancer incidence. This does not appear to be the case.

Likewise, if this premise was true, prostate cancer would be higher in countries where fish intake was low and this has never been proven.

Many studies conflict with this data –

- Zheng (2013) – meta analysis with 16 cohort studies showing that an intake of Omega-3 had a dose-related inverse relationship with risk of breast cancer.
- Szymanski (2010) – meta analysis demonstrating that fish consumption had a reduction in late stage or fatal prostate cancer among cohort studies.
- Lietzman (2004), Terry (2001) – population based studies showing increased Omega 3 fatty acid intake and reduction of prostate cancer.

Brasky, T. M., Darke, A. K., Song, X., Tangen, C. M., Goodman, P. J., Thompson, I. M., . . . Parnes, H. L. (2013).. *Journal of the National Cancer Institute*.

Meta-analysis with current results: EPA

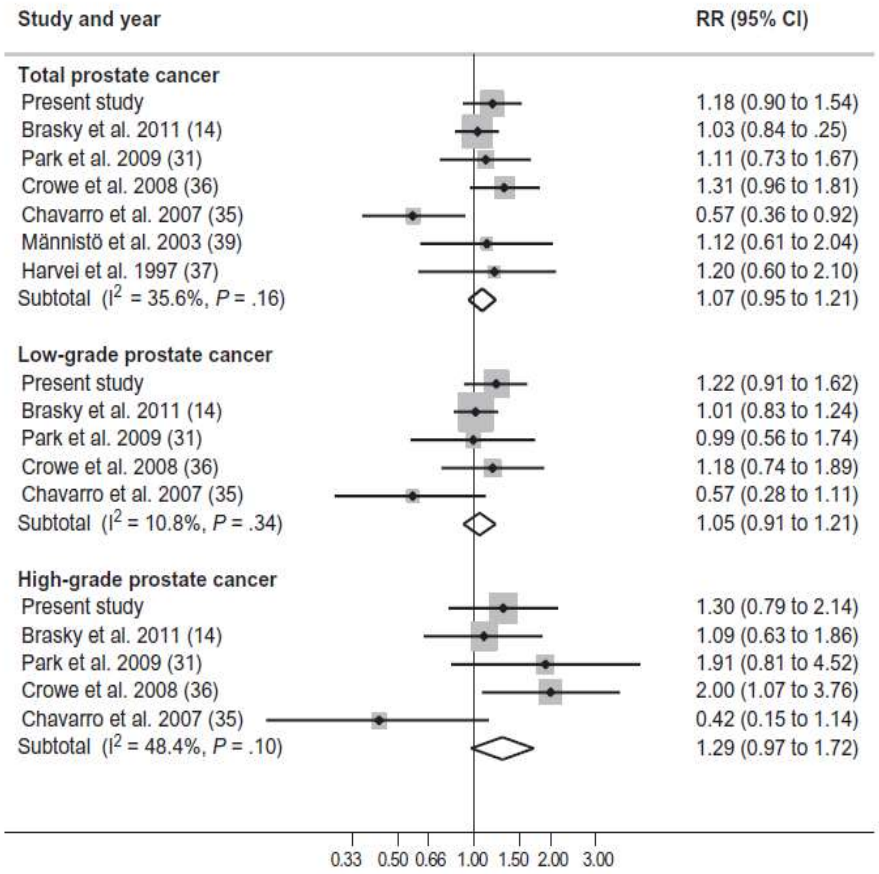


Figure 1. Meta-analysis of prospective biomarker studies examining associations between eicosapentaenoic acid (EPA) and total, low-, and high-grade prostate cancer risk. **Dots** and **horizontal lines** correspond to relative risks (RRs) and 95% confidence intervals (CIs), respectively, comparing the highest vs lowest quantile of EPA measured in blood for each study. The size of the **shaded square** represents the study-specific weight in the meta-analysis. The **diamond** represents the meta-relative risk and 95% confidence interval. Relative risks estimated assuming fixed effects. All statistical tests were two-sided.

Brasky, T. M., et. al. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Meta-analysis with current results: DHA

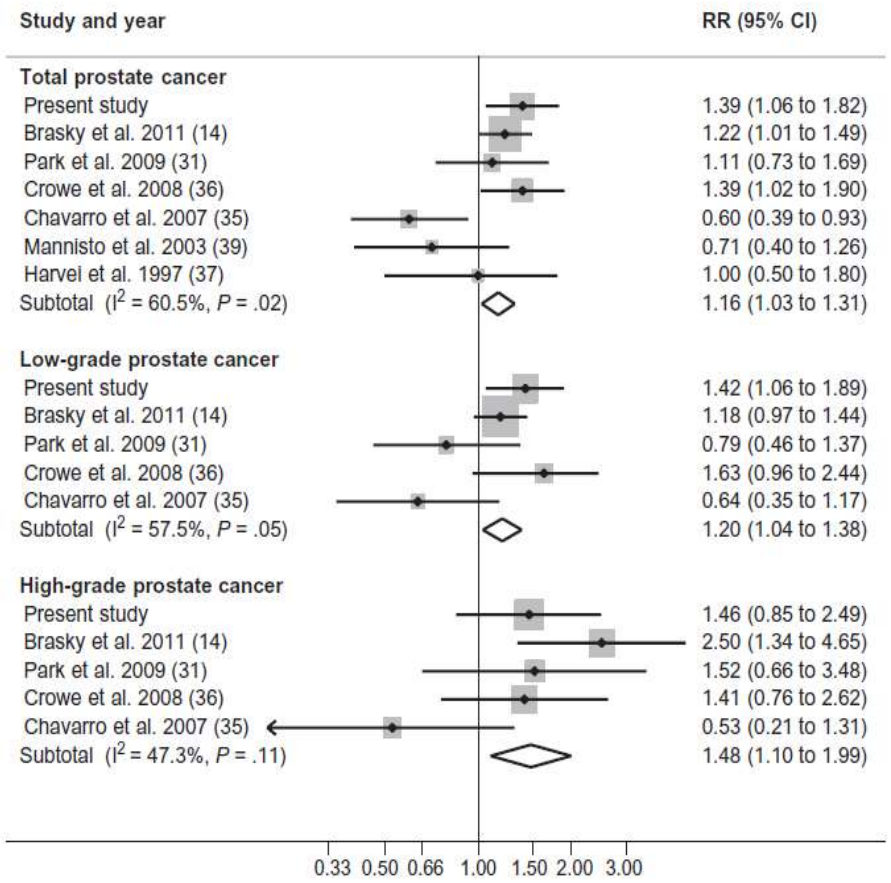


Figure 2. Meta-analysis of prospective biomarker studies examining associations between docosahexaenoic acid (DHA) and total, low-, and high-grade prostate cancer risk. **Dots** and **horizontal lines** correspond to relative risks (RRs) and 95% confidence intervals (CIs), respectively, for each study for comparisons of the highest vs lowest quantile of DHA measured in blood. The size of the **shaded square** represents the study-specific weight in the meta-analysis. The **diamond** represents the meta-relative risk and 95% confidence interval. Relative risks estimated assuming fixed effects. All statistical tests were two-sided.

Brasky, T. M., et. al. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Omega 3's and Prostate CA Risk

- Results contradict the expectation based on inflammation increasing prostate CA risk.
- Would expect long-chain ω-3 fatty acids to reduce risk and ω-6 fatty acids to increase risk.
- Unclear why high levels of long-chain ω-3 PUFA would increase prostate cancer risk.
- Recommendations for increasing long-chain ω-3 PUFA intake should consider potential prostate cancer risk.

Brasky, T. M., et. al. (2013). Plasma Phospholipid Fatty Acids and Prostate cancer risk in the Select trial. *Journal of the National Cancer Institute*.

Serum Fatty Acids and Ischemic Stroke Risk in Women

Yaemsiri, S., Seneta, S., et al. (2013). Serum fatty acids and incidence of ischemic stroke among postmenopausal women. *Stroke*. 44. DOI10.1161.

Serum fatty acids and incidence of ischemic stroke among postmenopausal women

Prospective, case-control study within the Women's Health Initiative Observational Study cohort of postmenopausal US women aged 50-79 years.

Between 1993 and 2003, incident cases of ischemic stroke were matched 1:1 to controls on age, race, and length of follow-up (964 matched pairs).

Yaemsiri, S., Seneta, S., et al. (2013).*Stroke*. 44. DOI10.1161.

Serum fatty acids and incidence of ischemic stroke among postmenopausal women

Individuals with serum trans, saturated and monounsaturated fatty acids are positively associated with ischemic stroke.

Individuals with n3 and n6 polyunsaturated fatty acids are inversely associated.

1.38 (99.9% CI, 1.05-1.83)

Yaemsiri, S., Seneta, S., et al. (2013).*Stroke*. 44. DOI10.1161.

Sexual Counseling with CAD patients

Steinke, E., Jaarsma, T., et al. (2013). Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document from the american heart association and the ESC council on cardiovascular nursing and allied professions. *Circulation*;128: DOI10:1161.

Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document

Recommendations for Sexual Assessment and Counseling

1. Structured counseling strategies to address the psychosexual needs of cardiac and stroke patients can be useful *(Class IIa; Level of Evidence C)*.
2. The use of instruments to assess cardiac and stroke patients' sexual concerns can be beneficial *(Class IIa; Level of Evidence C)*.

Steinke, E., Jaarsma, T., et al. (2013). *Circulation*;128:DOI10:1161

Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document

Table 3. Patient Education Resources for Cardiovascular Sexual Counseling

Resource	Location
Sex and Heart Disease (American Heart Association)	Online resource, available at: http://www.heart.org/HEARTORG/Conditions/More/MyHeartandStrokeNews/Sex-and-Heart-Disease_UCM_436414_Article.jsp Brochure product code 50-1622; order at: http://www.heart.org/HEARTORG/General/Sex-and-Heart-Disease-Brochure_UCM_310082_Article.jsp
Sex, Intimacy, and Heart Disease (Krames Patient Education)	Brochure product code 1900; order at: https://www.kramesstore.com
The Sensuous Heart: Guidelines for Sex After a Heart Attack or Heart Surgery (Pritchett & Hull Associates)	Booklet (24 pages); order at: http://www.p-h.com/product.php?productid=17372&cat=1&page=1
Sex After Stroke: Our Guide to Intimacy After Stroke (American Heart Association/American Stroke Association)	Brochure product code 50-1653; order at: http://www.heart.org/HEARTORG/General/Sex-After-Stroke-Our-Guide-to-Intimacy-After-Stroke_UCM_310558_Article.jsp
Sex after stroke (National Stroke Association)	*StrokeSmart* magazine, January/February 2009 featured article: "Redefining sexuality after stroke." Available at: http://www.stroke.org/site/PageServer?pagename=SS_MAG_jf2009_feature_sexuality National Stroke Association fact sheet: *Recovery After Stroke: Redefining Sexuality*. Available at: http://www.stroke.org/site/DocServer/NSAFactSheet_Sexuality.pdf?docID=999
Intimacy After Stroke (Stroke Foundation of New Zealand)	20-Page document that includes a patient questionnaire on sexual functioning after stroke. Available at: http://www.stroke.org.nz/resources/Sexuality-Booklet.pdf Print copies: send an e-mail message to strokenz@stroke.org.nz
Other stroke resources	Stroke Association - United Kingdom: *Sex after Stroke*. Available at: http://www.stroke.org.uk/factsheet/sex-after-stroke Caswell J. Sex and intimacy after stroke. *Stroke Connection*. March/April 2009: pp 12–15. Available at: http://www.nxtbook.com/nxtbooks/aha/strokeconnection_200903/#/14.
Sex and heart failure (ESC and the Heart Failure Association of the ESC)	Heart Failure Matters Web site. Available at: http://www.heartfailurematters.org/EN/Living-with-Heart-Failure/EN-Sex-and-heart-failure

Steinke, E., Jaarsma, T., et al. (2013). *Circulation*;128:DOI10:1161

Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document

Table 4. Assessment Instruments for Sexual Function

Instrument	Areas Assessed	Attributes
Female Sexual Function Index (FSFI)[106]	Women's sexual functioning: Desire, arousal, lubrication, orgasm, satisfaction	19 Items Discriminates between clinical and nonclinical populations[106,107] Established reliability (test-retest: 0.79–0.86; Cronbach α=0.82), with reported construct and divergent validity Response time 15 min
Brief Index of Sexual Functioning for Women (BISF-W)[108]	Dimensions of sexual functioning: Thoughts/ desires, arousal, frequency of sexual activity, receptivity/initiation, pleasure/orgasm, relationship satisfaction, problems affecting sexual function[109]	22 Items; self-report Subscale and composite score, with major factors of sexual desire, sexual activity, and sexual satisfaction Most items rated on Likert scale Reliability (test-retest, 0.68–0.78; 0.39 [factor 1] to 0.83 [factor 2]) and validity demonstrated[109,110] Response time 15–20 min

Steinke, E., Jaarsma, T., et al. (2013). *Circulation*;128:DOI10:1161

Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document

Changes in Sexual Functioning Questionnaire (CSFQ) and Changes in Sexual Functioning-Short Form (CSFQ-SF)[111,112]	Male and female sexual function in all domains of the sexual response cycle Subscales: Dimensions of pleasure, desire/frequency, desire/interest, arousal/excitement, orgasm/completion, and phases of sexual functioning (desire, arousal, orgasm)	CSFQ: females, 35 items; males, 36 items CSFQ-SF: 14 items for each of the female and male versions 5-Point Likert scale, from "never or no enjoyment" to "every day or always" May be scored on the 3 phases of sexual response: Desire, arousal, orgasm/completion[112] Reliability (0.89–0.90, CSFQ-SF) and construct validity established[112] Response time for CSFQ, 15–20 min; CSFQ-SF appropriate for clinical setting, with response time of 4–5 min
Derogatis Interview for Sexual Functioning (DISF) and Derogatis Interview for Sexual Functioning – Self-Report (DISF-SR)[113]	Patient's perception of overall current sexual functioning 5 Domains: Sexual cognition/fantasy, sexual arousal, sexual behavior/experience, orgasm, sexual drive/relationship	26 Items Rated on 4-point Likert scale Composite score Gender-specific versions Reliability (0.74–0.80) and validity established Response time 15–20 min

Steinke, E., Jaarsma, T., et al. (2013). *Circulation*;128:DOI10:1161

Sexual counseling for individuals with cardiovascular disease and their partners: a consensus document

Brief Male Sexual Function Inventory (BMSFI)[114]	4 Functional domains: Drive, erection, ejaculation, problem with sexual function; and sexual satisfaction	10 Items for 4 domains, 1-item sexual satisfaction Overall score reflects sexual functioning Rated on 0–4 scale, from "no function/big problem" to "no difficulty/no problem" The 4 domains explained 28% of variance in overall sexual satisfaction Reliability: Cronbach α=0.90–0.94[114]
Index of Erectile Function-5 (ILEF-5)[115]	Measure of ED in men	5-Item tool revised from original 15 items Ordinal measurement of items Score ranges from 5–25 Score <21 indicative of ED; further ED scoring: Severe (5–7), moderate (8–11), mild to moderate (12–16), mild (17–21), and none (22–25) Reported instrument sensitivity 0.98 and specificity 0.88; reported weighted κ=0.85 for agreement between the predicted and true ED classes[115]

Steinke, E., Jaarsma, T., et al. (2013). *Circulation*;128:DOI10:1161

Centrally acting ACE-I decreases cognitive decline in dementia patients

Yang, G., O'caolmh, R., et al. (2013). Effects of centrally acting ACE inhibitors on the rate of cognitive decline in dementia. BMJ. Open DOI:10.1136.

ACE-I on rate of cognitive decline in dementia

Centrally acting ACE (CACE-Is) cross the blood-brain barrier: compared the rates of cognitive decline in clinic patients with dementia. Observational case-control study

817 patients diagnosed with Alzheimer's or mixed dementia. Of these, 361 with valid cognitive scores were included for analysis, 85 CACE-Is and 276 NoCACE-I.

Age: 41-104 years,

Mini mental status exam (MMSE) or Quick Mild Cognitive Impairment (Qmci) measured ave 6 months rates of change.

Yang, G., O'caolmh, R., et al. (2013). Effects of centrally acting ACE inhibitors on the rate of cognitive decline in dementia. BMJ. Open DOI:10.1136

ACE-I on rate of cognitive decline in dementia

Rate of decline was compared between groups –

CACE-I and NoCACE-I : baseline and 6 months

6 month rate of decline in Qmci scores between CACE-I (1.8 points) and NoCACE-I (2.1 points) patients (p=0.049). Median SMMSE scores improved by 1.2 points in the first 6 months of CACE treatment compared to 0.8 point decline for the CACE-I (p=0.003)

Cognitive scores can improve in the first 6 months after CACE-I treatment and use of CACE-Is is associated with a reduced rate of cognitive decline in patients with dementia.

Yang, G., O'caolmh, R., et al. (2013). Effects of centrally acting ACE inhibitors on the rate of cognitive decline in dementia. BMJ. Open DOI:10.1136

ACE-I on rate of cognitive decline in dementia

Table 3 Comparison of differences in Qmci and SMMSE scores between baseline and end point

	Groups	Mann-Whitney U test (p Values)
Changes in Qmci	CACE-I (53) vs NoCACE-I (102) median = 1.8* vs median = 2.1*	0.049
Changes in SMMSE	CACE-I (113) vs NoCACE-I (240) median = 0.8* vs median = 1.0*	0.77
	NewCACE-I (30) vs NoCACE-I (240) median = −1.2* vs median = 1.0*	0.001
	NewCACE-I (30) vs CACE-I† (83) median = −1.2* vs median = 0.8*	0.003

*Median score shows the change in six months for CACE-I, NoCACE-I and NewCACE-I
†CACE-I group excluding NewCACE-I patients.
CACE-I, patients currently receiving ACE inhibitors; NewCACE-I, patients who were newly started on CACE-Is; NoCACE-I, patients who are not currently prescribed CACE-Is; Qmci, Quick Mild Cognitive Impairment; SMMSE, Standardised Mini-Mental State Examination.

Yang, G., O'caolmh, R., et al. (2013). Effects of centrally acting ACE inhibitors on the rate of cognitive decline in dementia. BMJ. Open DOI:10.1136

ACE-I on rate of cognitive decline in dementia

Bale/Doneen summary:

Centrally Acting ACE-I showed a 65% less decline in cognitive functioning per year of exposure.

Centrally Acting ACE-I:

- Captopril
- **Ramipril**
- Lisinopril
- Trandolapril
- Perindopril

Non-Centrally Acting ACE-I:

- Enalapril
- Moexipapril
- Quinapril

Yang, G., O'caolmh, R., et al. (2013). Effects of centrally acting ACE inhibitors on the rate of cognitive decline in dementia. BMJ. Open DOI:10.1136

And the Winner is…..

- BEST IN SHOW……..GOES TO…..

Niacin does NOT cause Strokes!

Teo, K., Goldstein, L., et al. (2013). Extended-release niacin therapy and risk of ischemic stroke in patients with cardiovascular disease: the atherothrombosis intervention in metabolic syndrome with low HDL/high Triglycerides: Impact on global health outcome (AIM-HIGH). Stroke. ISSN: 0039-2499.

BEST IN SHOW 2013

Darby!

Extended-release niacin therapy and risk of ischemic stroke in patients with cardiovascular disease: the atherothrombosis intervention in metabolic syndrome with low HDL/high Triglycerides: Impact on global health outcome (AIM-HIGH)

Final analysis of AIM-HIGH to determine if relationship between Niacin therapy and ischemic stroke risk.

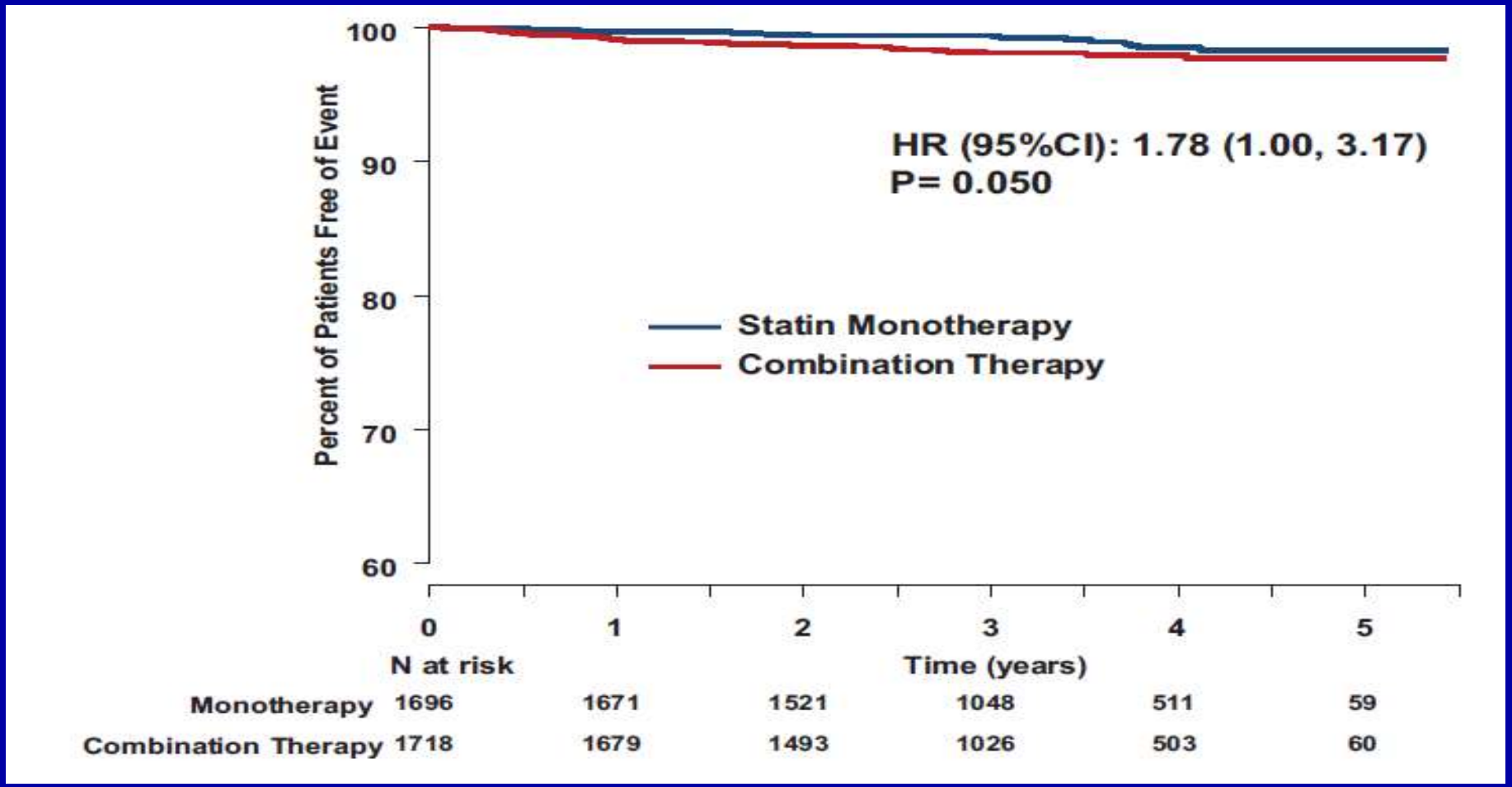
Teo, K., Goldstein, L., et al. (2013).Stroke. ISSN: 0039-2499.

Extended-release niacin therapy and risk of ischemic stroke (AIM-HIGH)

NO GROUP DIFFERENCE IN TRIAL PRIMARY COMPOSITE END POINT (ISCHEMIC STROKE) AT A MEAN 36-MONTH FOLLOW-UP AMONG 3414 PATIENTS.

Teo, K., Goldstein, L., et al. (2013).Stroke. ISSN: 0039-2499.

Extended-release niacin therapy and risk of ischemic stroke (AIM-HIGH)



Teo, K., Goldstein, L., et al. (2013).Stroke. ISSN: 0039-2499.

Extended-release niacin therapy and risk of ischemic stroke (AIM-HIGH)

Table 2. Incidence of Various Stroke Types

Event	Statin Monotherapy, n (%)	Combination Therapy, n (%)	Hazard Ratio (95% CI)	*P* Value
Ischemic stroke (fatal or nonfatal)	18 (1.06)	32 (1.86)	1.78 (1.00–3.17)	0.050
Ischemic stroke+hemorrhagic stroke+confirmed TIA	38 (2.24)	46 (2.68)	1.21 (0.79–1.87)	0.377
Ischemic stroke+hemorrhagic stroke	21 (1.24)	36 (2.10)	1.73 (1.01–2.96)	0.047
Ischemic stroke+confirmed TIA	35 (2.06)	42 (2.44)	1.20 (0.77–1.88)	0.428
Hemorrhagic stroke	3 (0.18)	4 (0.23)	1.36 (0.30–6.08)	0.688
TIA	19 (1.12)	11 (0.64)	0.57 (0.27–1.21)	0.143

CI indicates confidence interval; and TIA, transient ischemic attack.

Teo, K., Goldstein, L., et al. (2013).Stroke. ISSN: 0039-2499.

Upcoming Events

September

14-15:	Bale/Doneen Preceptorship Program – Lubbock, TX
20-21:	American Academy of Oral Systemic Health – Las Vegas
30-10/4:	Osteopathic Medical Conference & Exposition – Las Vegas

October

10-12:	Intern. Acad. of Biomedical Dentistry & Medicine – Houston
16-20:	B/DReunion and CHL Symposium, – Dallas Ft. Worth

November

8-9:	Bale/Doneen Preceptorship Program – Nashville, TN

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