PREVENZIONE CARDIOVASCOLARE

I fattori di rischio cardiovascolare emergenti … rumore, stress, inquinamento acustico, …"spirituality and heart"

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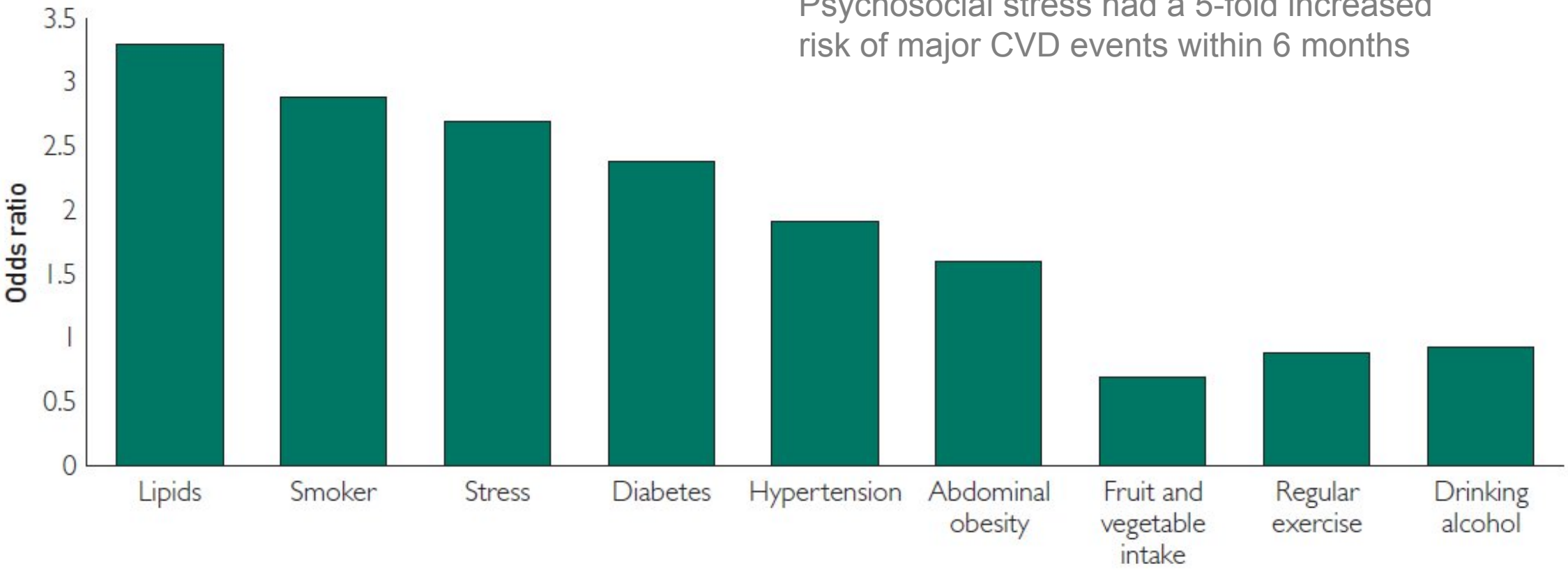
SISTEMA SANITARIO REGIONALE
AZIENDA OSPEDALIERA
SAN CAMILLO FORLANINI

The odds of experiencing a myocardial infarction for individuals in the INTERHEART study

Psychosocial stress had a 5-fold increased risk of major CVD events within 6 months



O'Keefe EL, et al. Mayo Clin Proc. 2019;94:1852-1864

Stress related disorders and risk of cardiovascular disease: population based, sibling controlled cohort study

Huan Song,[1,2] Fang Fang,[2] Filip K Arnberg,[3,4] David Mataix-Cols,[5,6] Lorena Fernández de la Cruz,[5,6] Catarina Almqvist,[2,7] Katja Fall,[2,8] Paul Lichtenstein,[2] Gudmundur Thorgeirsson,[1] Unnur A Valdimarsdóttir[1,2,9]

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ABSTRACT

OBJECTIVE

To assess the association between stress related disorders and subsequent risk of cardiovascular disease.

DESIGN

Population based, sibling controlled cohort study.

SETTING

Population of Sweden.

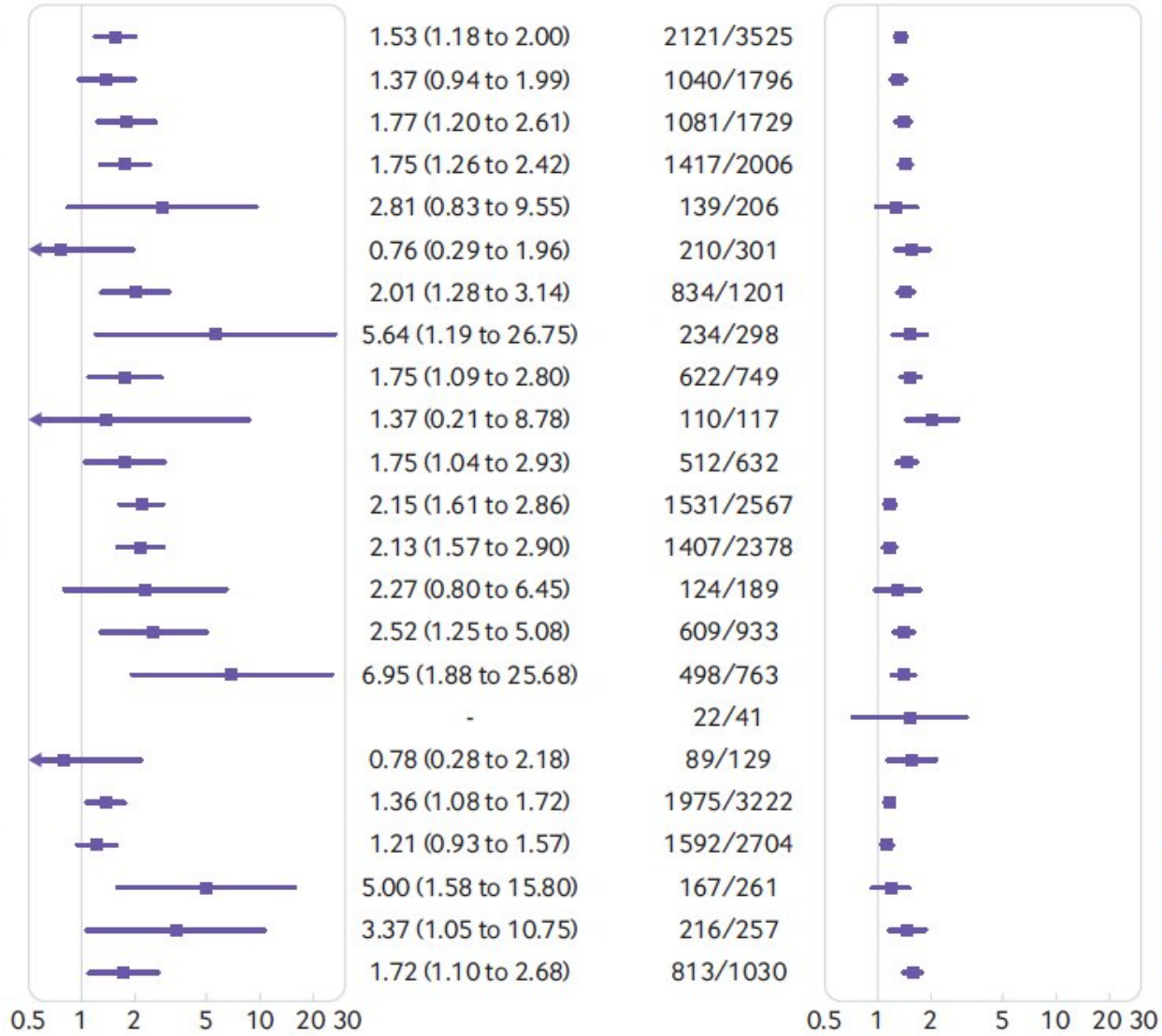
PARTICIPANTS

136 637 patients in the Swedish National Patient Register with stress related disorders, including post-traumatic stress disorder (PTSD), acute stress reaction, adjustment disorder, and other stress reactions, from 1987 to 2013; 171 314 unaffected full siblings of these patients; and 1 366 370 matched unexposed people from the general population. their unaffected full siblings, and the matched unexposed individuals, respectively. In sibling based comparisons, the hazard ratio for any cardiovascular disease was 1.64 (95% confidence interval 1.45 to 1.84), with the highest subtype specific hazard ratio observed for heart failure (6.95, 1.88 to 25.68), during the first year after the diagnosis of any stress related disorder. Beyond one year, the hazard ratios became lower (overall 1.29, 1.24 to 1.34), ranging from 1.12 (1.04 to 1.21) for arrhythmia to 2.02 (1.45 to 2.82) for artery thrombosis/embolus. Stress related disorders were more strongly associated with early onset cardiovascular diseases (hazard ratio 1.40 (1.32 to 1.49) for attained age <50) than later onset ones (1.24 (1.18 to 1.30) for attained age ≥50; P for difference=0.002). Except for fatal cardiovascular diseases, these associations were not modified by the presence of psychiatric comorbidity. Analyses

Song H, et al. BMJ 2019;365:l1255

Relative risks of developing different types of cardiovascular disease among patients with any stress related disorder, compared with their full siblings

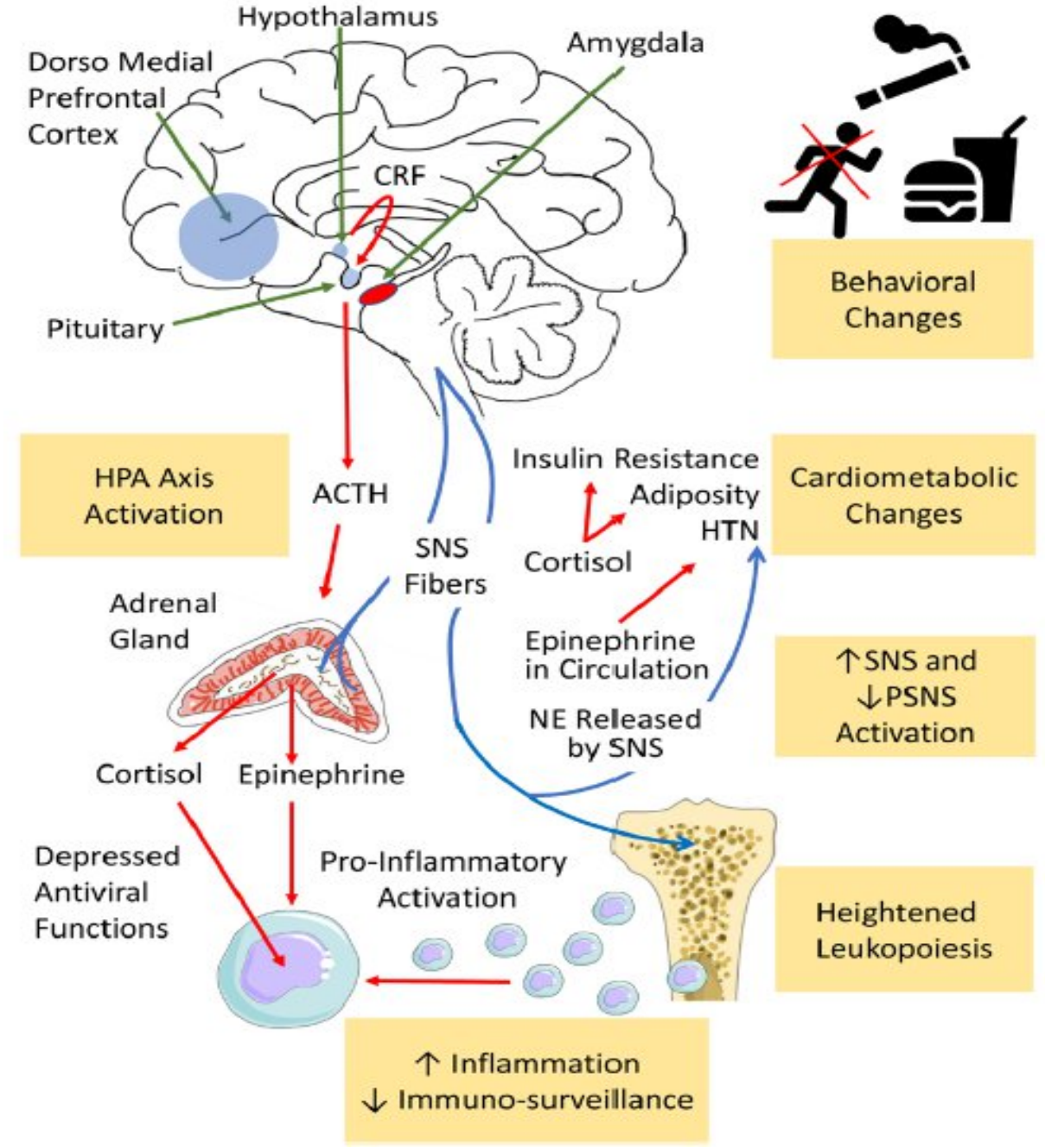
Cardiovascular diseases	<1 year of follow-up: No of cases in patients/siblings	<1 year of follow-up: Hazard ratio (95% CI)	≥1 year of follow-up: No of cases in patients/siblings	≥1 year of follow-up: Hazard ratio (95% CI)
Ischaemic heart disease, all	161/193	1.53 (1.18 to 2.00)	2121/3525	1.34 (1.25 to 1.44)
Acute myocardial infarction	84/104	1.37 (0.94 to 1.99)	1040/1796	1.29 (1.17 to 1.43)
Other ischaemic heart disease	77/89	1.77 (1.20 to 2.61)	1081/1729	1.39 (1.26 to 1.53)
Cerebrovascular disease, all	120/119	1.75 (1.26 to 2.42)	1417/2006	1.42 (1.31 to 1.55)
Arachnoidal bleeding	12/7	2.81 (0.83 to 9.55)	139/206	1.27 (0.97 to 1.65)
Haemorrhagic stroke	11/24	0.76 (0.29 to 1.96)	210/301	1.56 (1.24 to 1.95)
Ischaemic stroke	75/76	2.01 (1.28 to 3.14)	834/1201	1.44 (1.29 to 1.61)
Other cerebrovascular disease	22/12	5.64 (1.19 to 26.75)	234/298	1.51 (1.21 to 1.89)
Emboli and thrombosis, all	61/56	1.75 (1.09 to 2.80)	622/749	1.52 (1.34 to 1.74)
Artery thrombosis/embolus	9/8	1.37 (0.21 to 8.78)	110/117	2.02 (1.45 to 2.82)
Pulmonary emboli	52/48	1.75 (1.04 to 2.93)	512/632	1.45 (1.26 to 1.67)
Hypertensive diseases, all	168/143	2.15 (1.61 to 2.86)	1531/2567	1.16 (1.08 to 1.26)
Essential hypertension	151/129	2.13 (1.57 to 2.90)	1407/2378	1.16 (1.07 to 1.26)
Other hypertensive disease	17/14	2.27 (0.80 to 6.45)	124/189	1.28 (0.95 to 1.73)
Heart failure, all	41/29	2.52 (1.25 to 5.08)	609/933	1.39 (1.22 to 1.60)
Heart failure	32/15	6.95 (1.88 to 25.68)	498/763	1.39 (1.19 to 1.63)
Ischaemic cardiomyopathy	1/1	-	22/41	1.50 (0.71 to 3.15)
Takotsubo cardiomyopathy	8/13	0.78 (0.28 to 2.18)	89/129	1.55 (1.12 to 2.14)
Arrhythmia/conduction disorder, all	196/215	1.36 (1.08 to 1.72)	1975/3222	1.16 (1.08 to 1.24)
Arrhythmia	146/186	1.21 (0.93 to 1.57)	1592/2704	1.12 (1.04 to 1.21)
Conduction disorder	25/14	5.00 (1.58 to 15.80)	167/261	1.17 (0.92 to 1.50)
Cardiac arrest	25/15	3.37 (1.05 to 10.75)	216/257	1.46 (1.16 to 1.85)
Fatal cardiovascular events, all	96/66	1.72 (1.10 to 2.68)	813/1030	1.56 (1.37 to 1.77)



Song H, et al. BMJ 2019;365:l1255

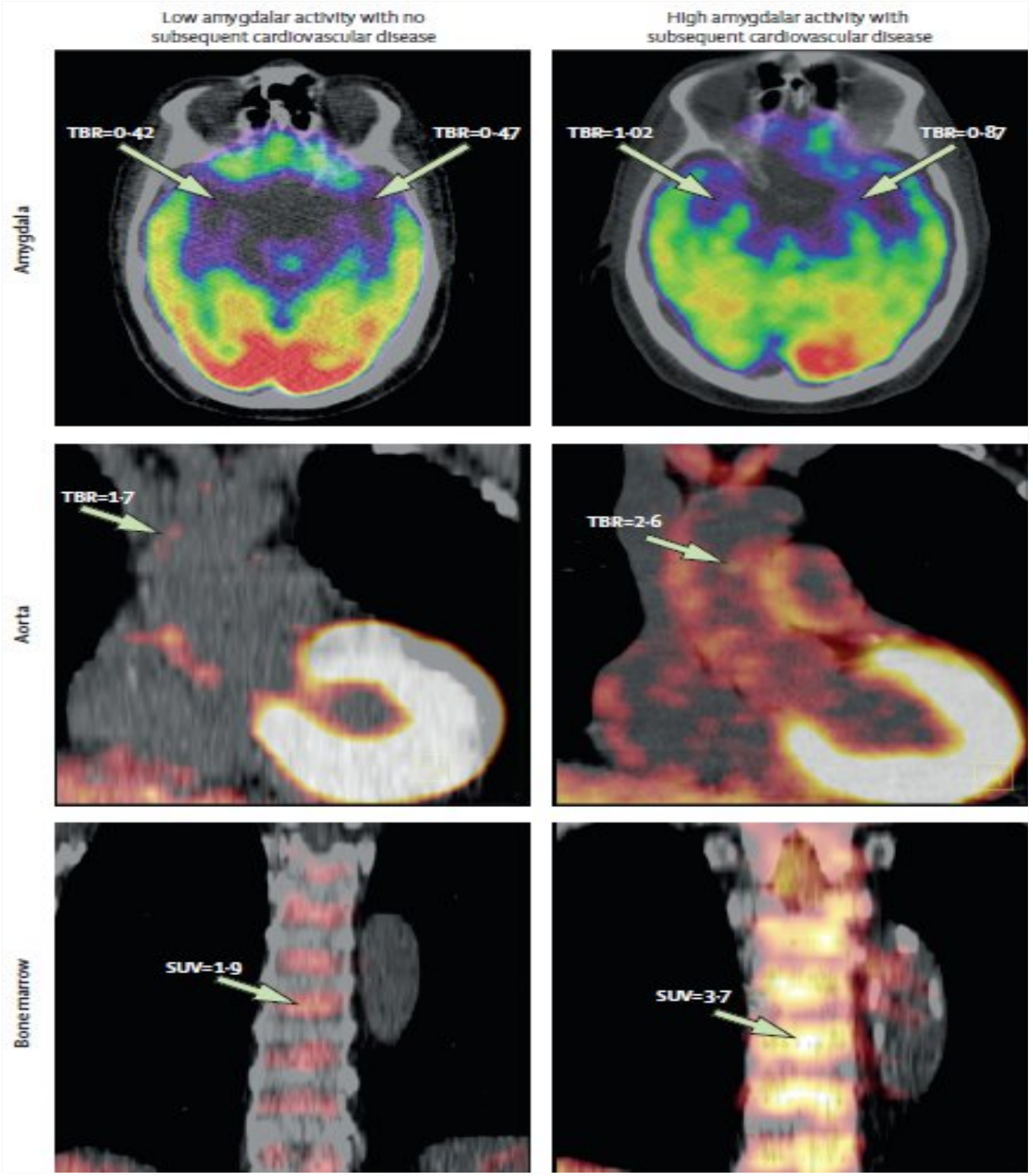
Chronic stress	Strain over months to years
Acute stress	Strain lasting seconds to weeks
Amygdala	Bilateral limbic structure beneath the temporal lobe involved in emotional processing
Environmental stress	Negative response to an environmental stimulus (e.g., noise)
Psychosocial stress	Negative response to an emotional or social stimulus (e.g., low income)
Resilience	Capacity to recover from or tolerate stress

Physiological consequences of stress



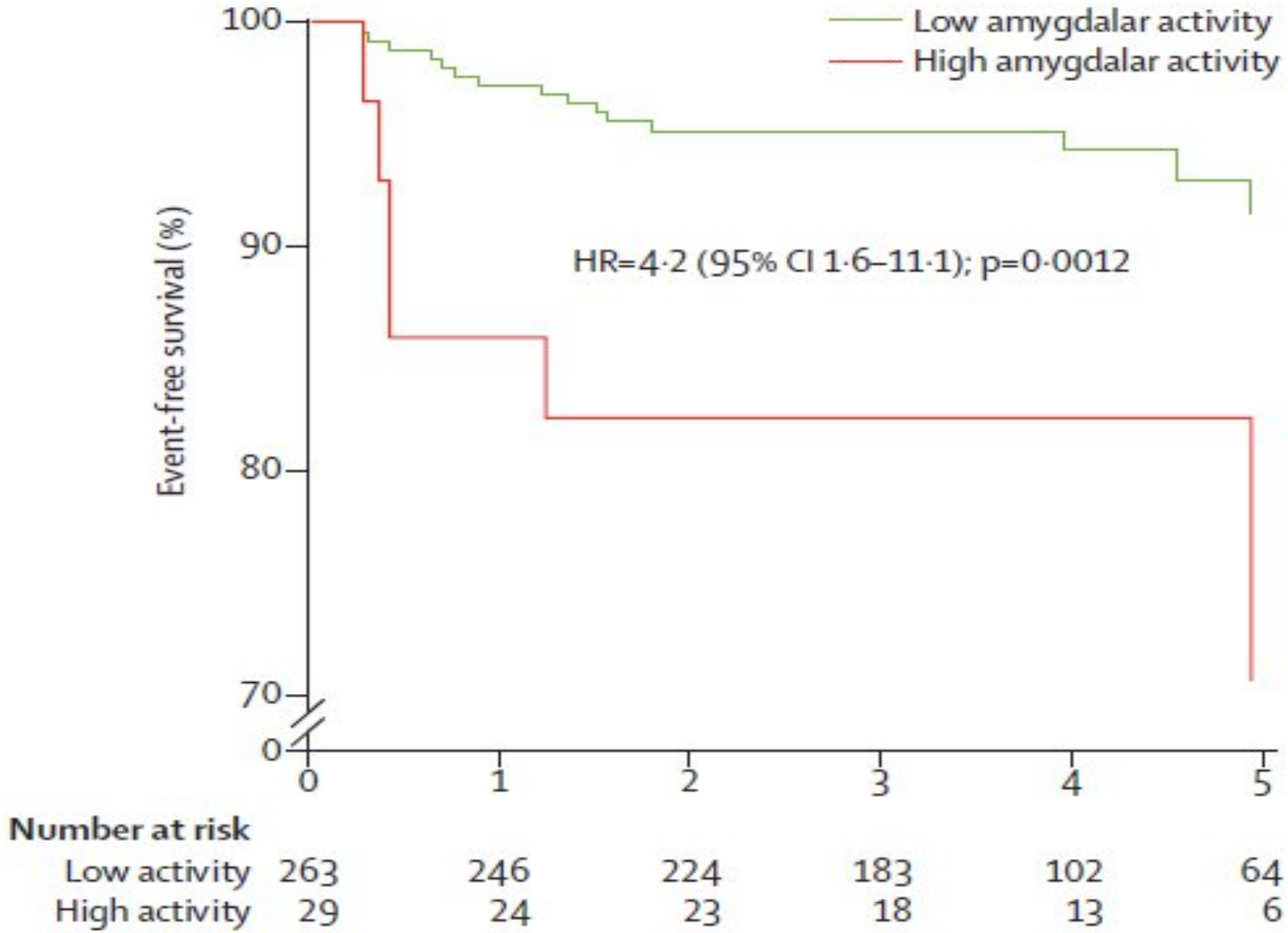
Osborne MT, et al. *Circ Cardiovasc Imaging*. 2020;13:e010931

Amygdalar, arterial, and bone-marrow uptake of ^{18}F-FDG in individuals with and without subsequent cardiovascular disease



Tawakol A, et al.
Lancet 2017;389:834-45

Kaplan-Meier survival curves of low *vs* high amygdalar activity based on the 90th percentile cutoff

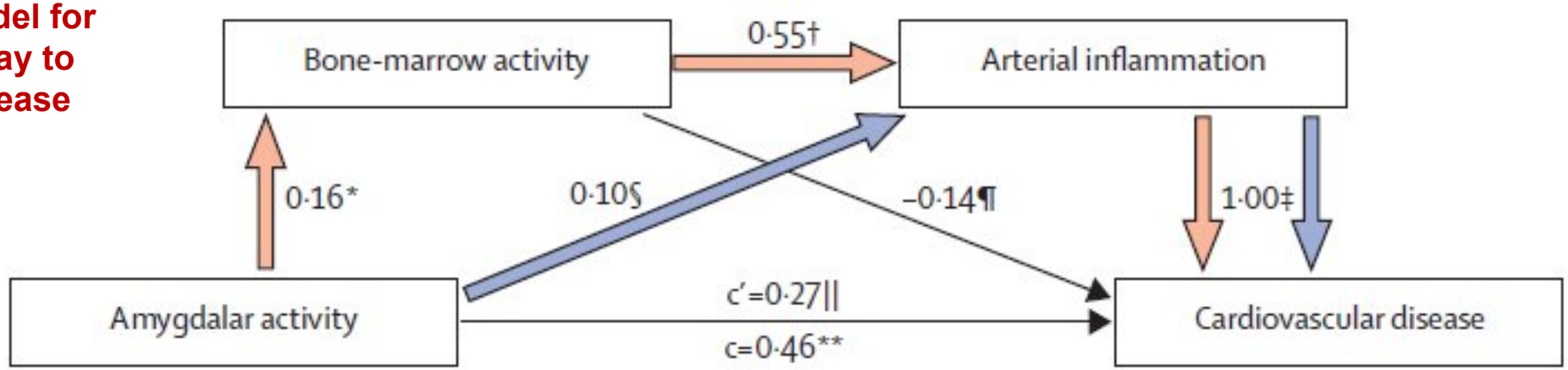


Tawakol A, et al. Lancet 2017;389:834-45

Correlation of amygdalar activity with haemopoietic activity and arterial inflammation

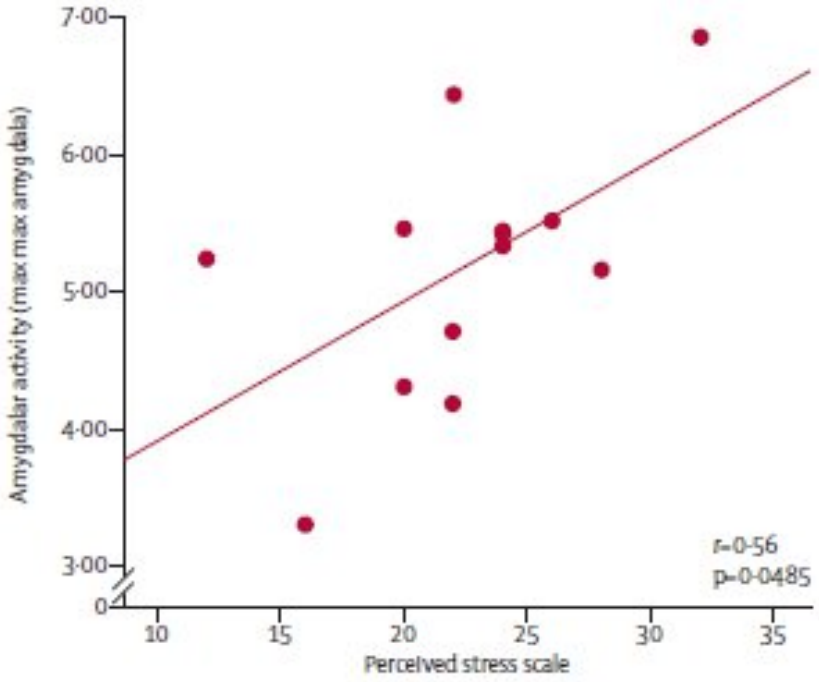
	Mean mean		Mean max		Max max	
	Correlation coefficient	p value	Correlation coefficient	p value	Correlation coefficient	p value
Aortic inflammation	0·49	<0·0001	0·45	<0·0001	0·41	<0·0001
Carotid inflammation*	0·47	<0·0001	0·43	<0·0001	0·40	<0·0001
Splenic activity	0·50	<0·0001	0·47	<0·0001	0·46	<0·0001
Bone-marrow activity	0·44	<0·0001	0·40	<0·0001	0·40	<0·0001
Control tissue uptake of ^{18}F-FDG uptake (subcutaneous fat)	0·02	0·73	0·02	0·80	0·02	0·79

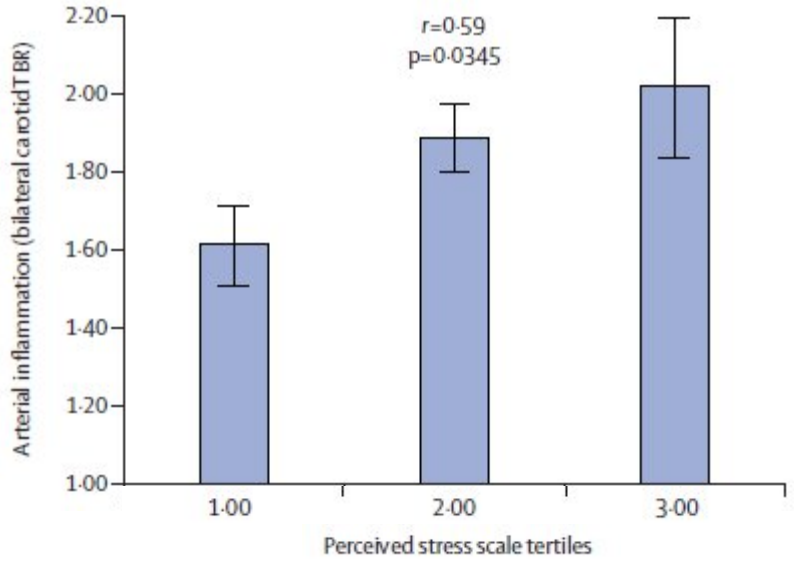
Serial mediation model for hypothesised pathway to a cardiovascular disease event

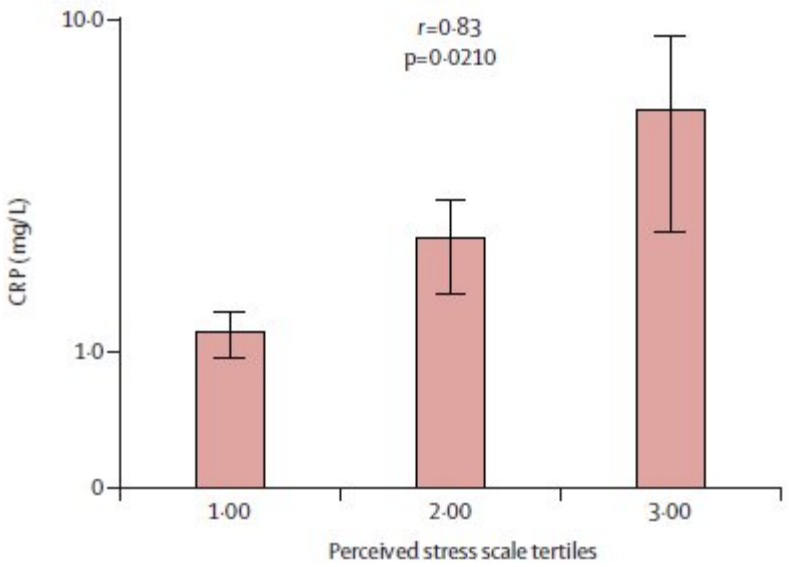


Tawakol A, et al. Lancet 2017;389:834-45

Perceived stress associated with amygdalar activity, arterial inflammation, and CRP in cross-sectional validation sub-study

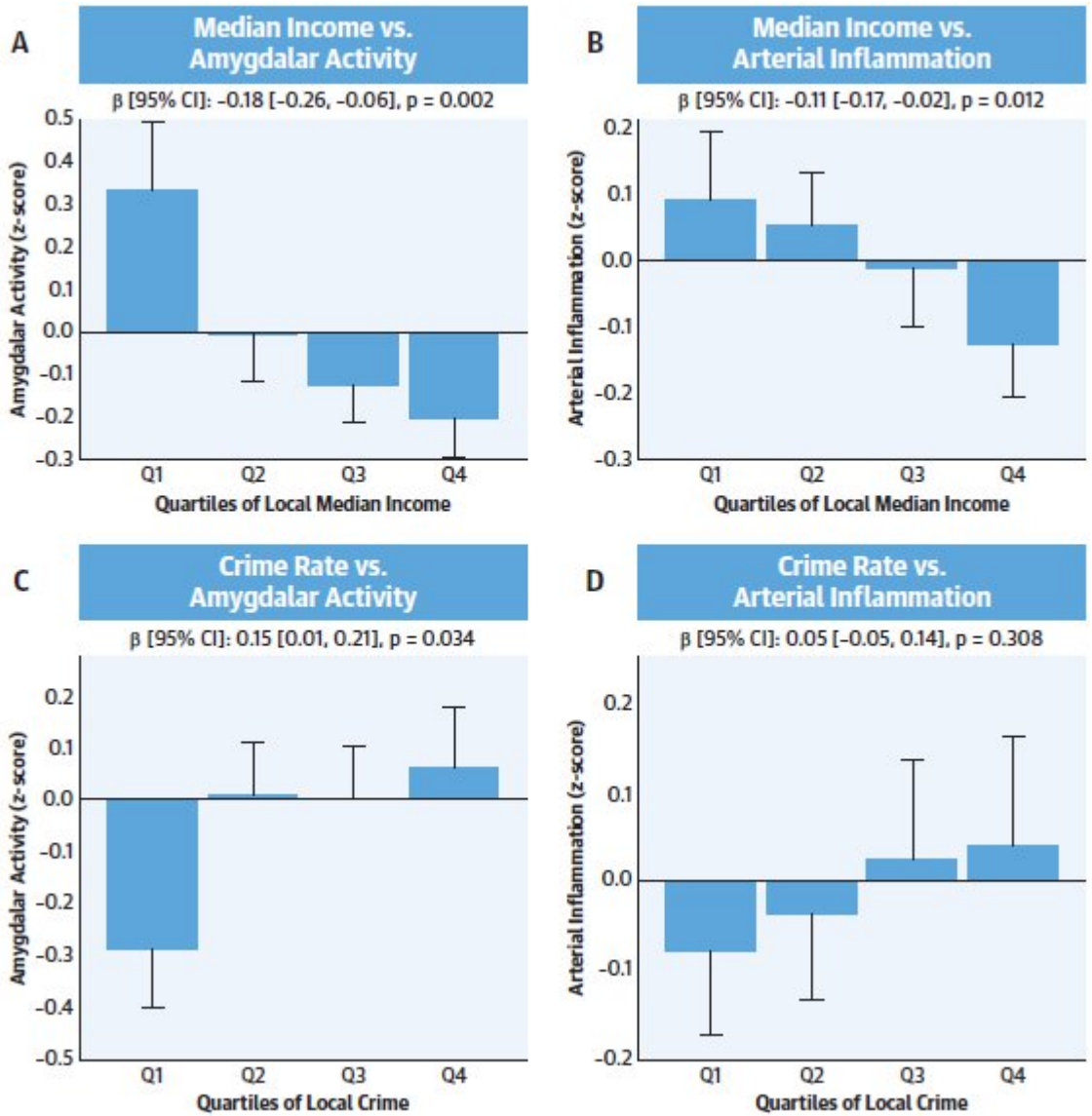




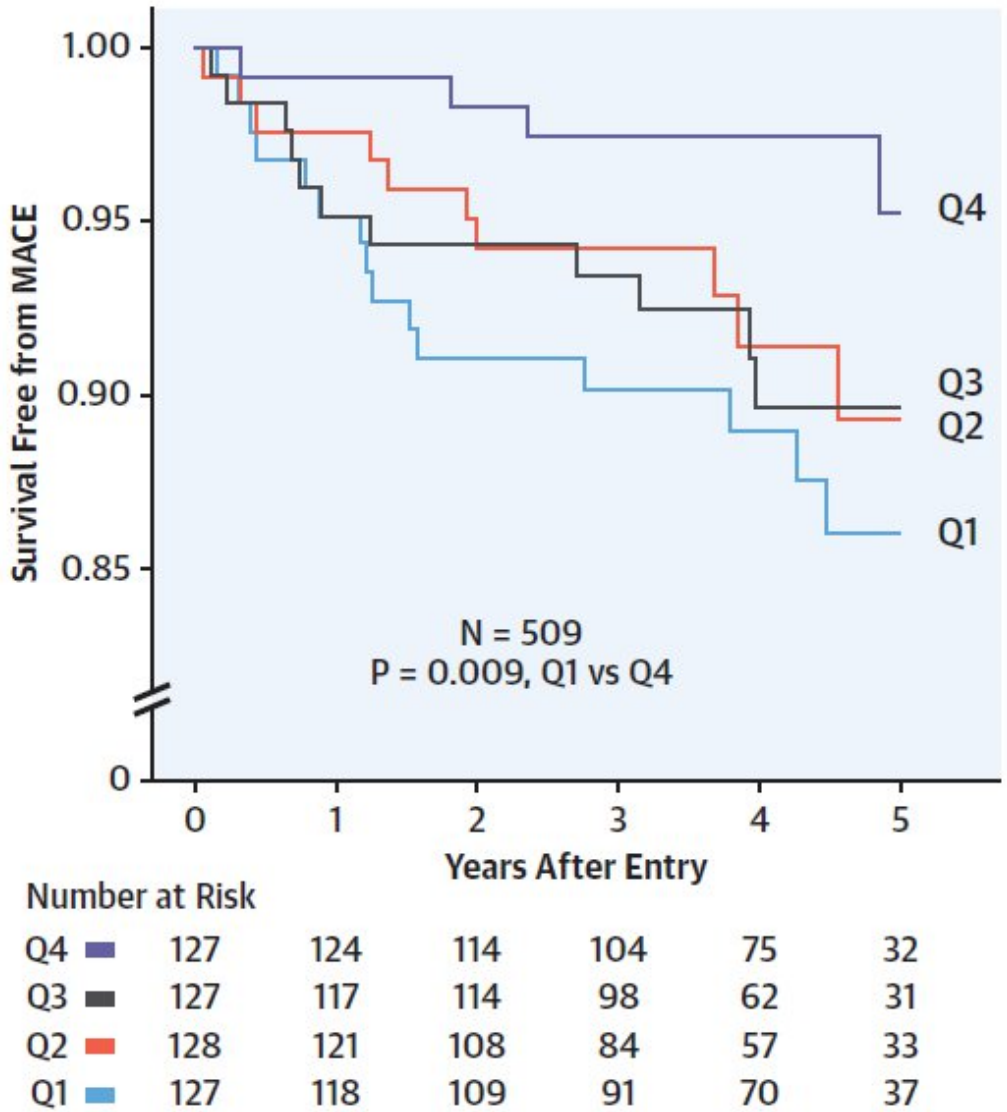


Tawakol A, et al.
Lancet 2017;
389:834-45

Socioeconomic Status Vs Amygdalar Activity and Arterial Inflammation



Socioeconomic Status Vs MACE



Tawakol A, et al. J Am Coll Cardiol 2019;73:3243–55

OPEN

Plasma protein expression profiles, cardiovascular disease, and religious struggles among South Asians in the MASALA study

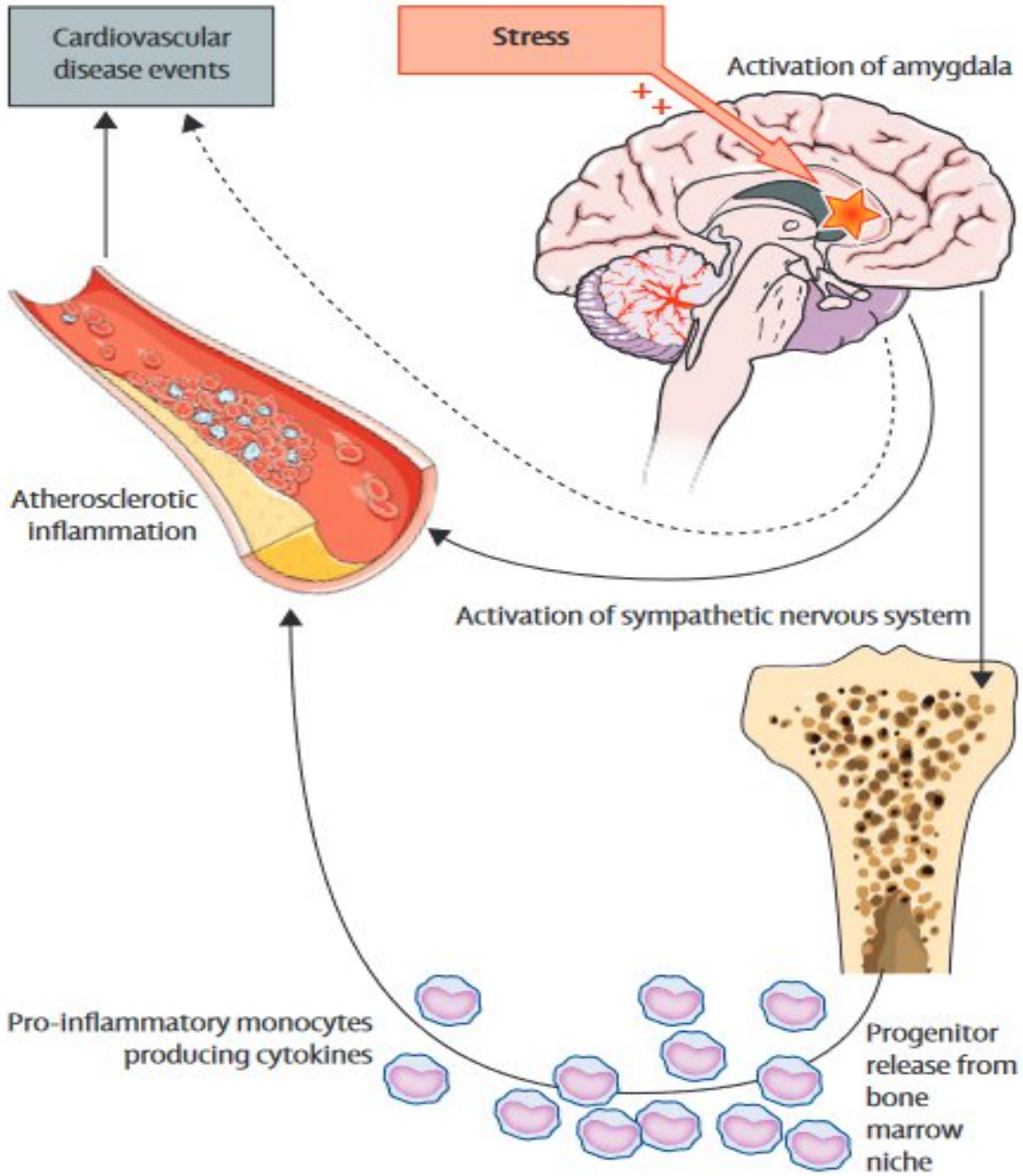
Long H. Ngo[1,2,3]✉, M. Austin Argentieri[4,5], Simon T. Dillon[1,2], Blake Victor Kent[4,6], Alka M. Kanaya[7,8], Alexandra E. Shields[1,4,8] & Towia A. Libermann[1,2,8]

Blood protein concentrations are clinically useful, predictive biomarkers of cardiovascular disease (CVD). Despite a higher burden of CVD among U.S. South Asians, no CVD-related proteomics study has been conducted in this sub-population. The aim of this study is to investigate the associations between plasma protein levels and CVD incidence, and to assess the potential influence of religiosity/spirituality (R/S) on significant protein-CVD associations, in South Asians from the MASALA Study. We used a nested case–control design of 50 participants with incident CVD and 50 sex- and age-matched controls. Plasma samples were analyzed by SOMAscan for expression of 1305 proteins. Multivariable logistic regression models and model selection using Akaike Information Criteria were performed on the proteins and clinical covariates, with further effect modification analyses conducted to assess the influence of R/S measures on significant associations between proteins and incident CVD events.

Religious struggles that exacerbate the adverse impact of stressful life events, significantly modified the effect of Contactin-5 and Complement factor B on risk of CVD. Our research is this first assessment

Complement factor B [CFB]) associated with incident CVD after adjustment for diabetes (AUC = 0.82). Religious struggles that exacerbate the adverse impact of stressful life events, significantly modified the effect of Contactin-5 and Complement factor B on risk of CVD. Our research is this first assessment of the relationship between protein concentrations and risk of CVD in a South Asian sample. Further research is needed to understand patterns of proteomic profiles across diverse ethnic communities, and the influence of resources for resiliency on proteomic signatures and ultimately, risk of CVD.

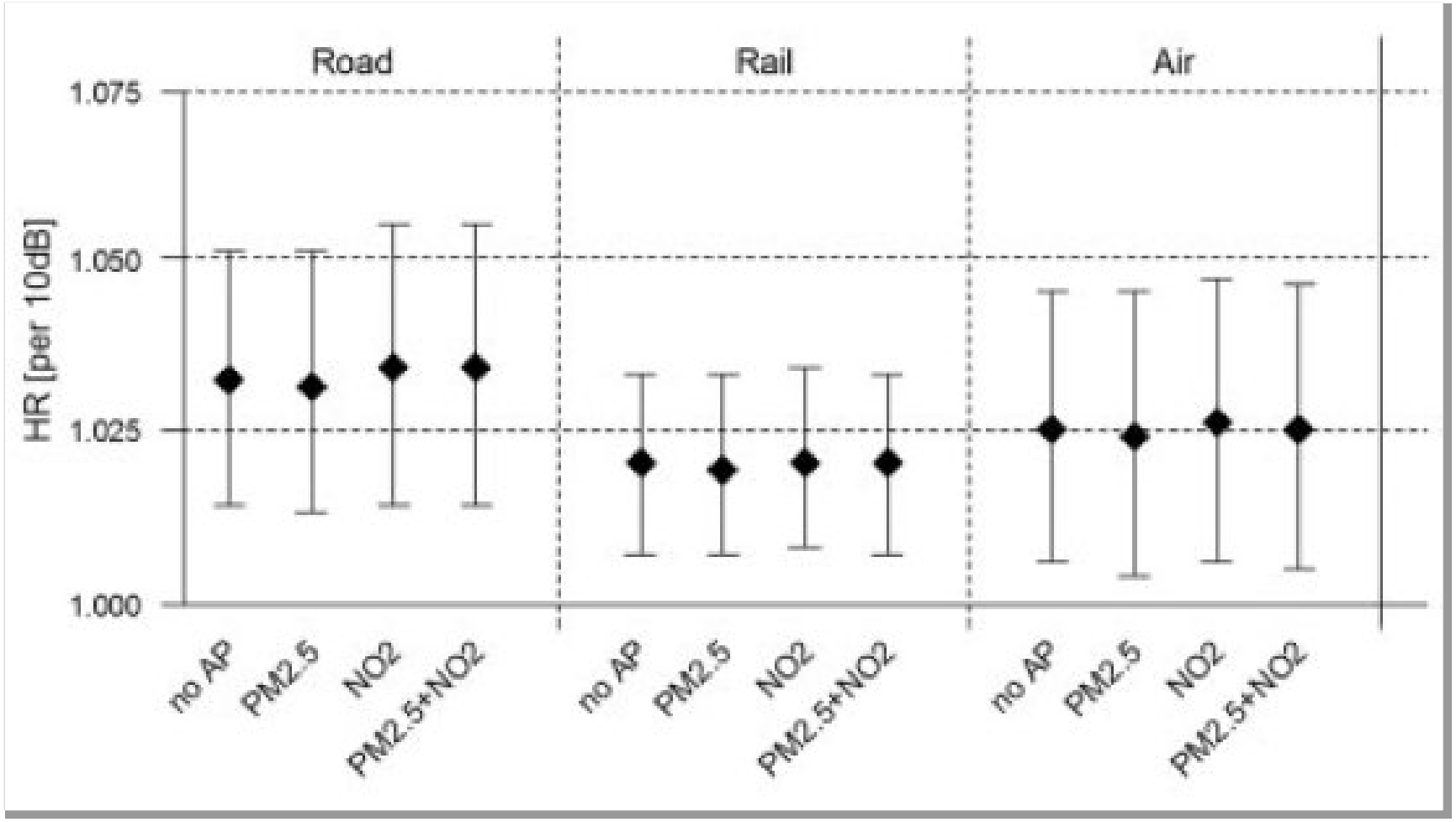
A model of stress leading to atherosclerotic inflammation



Tawakol A, et al.
Lancet 2017;389:834-45

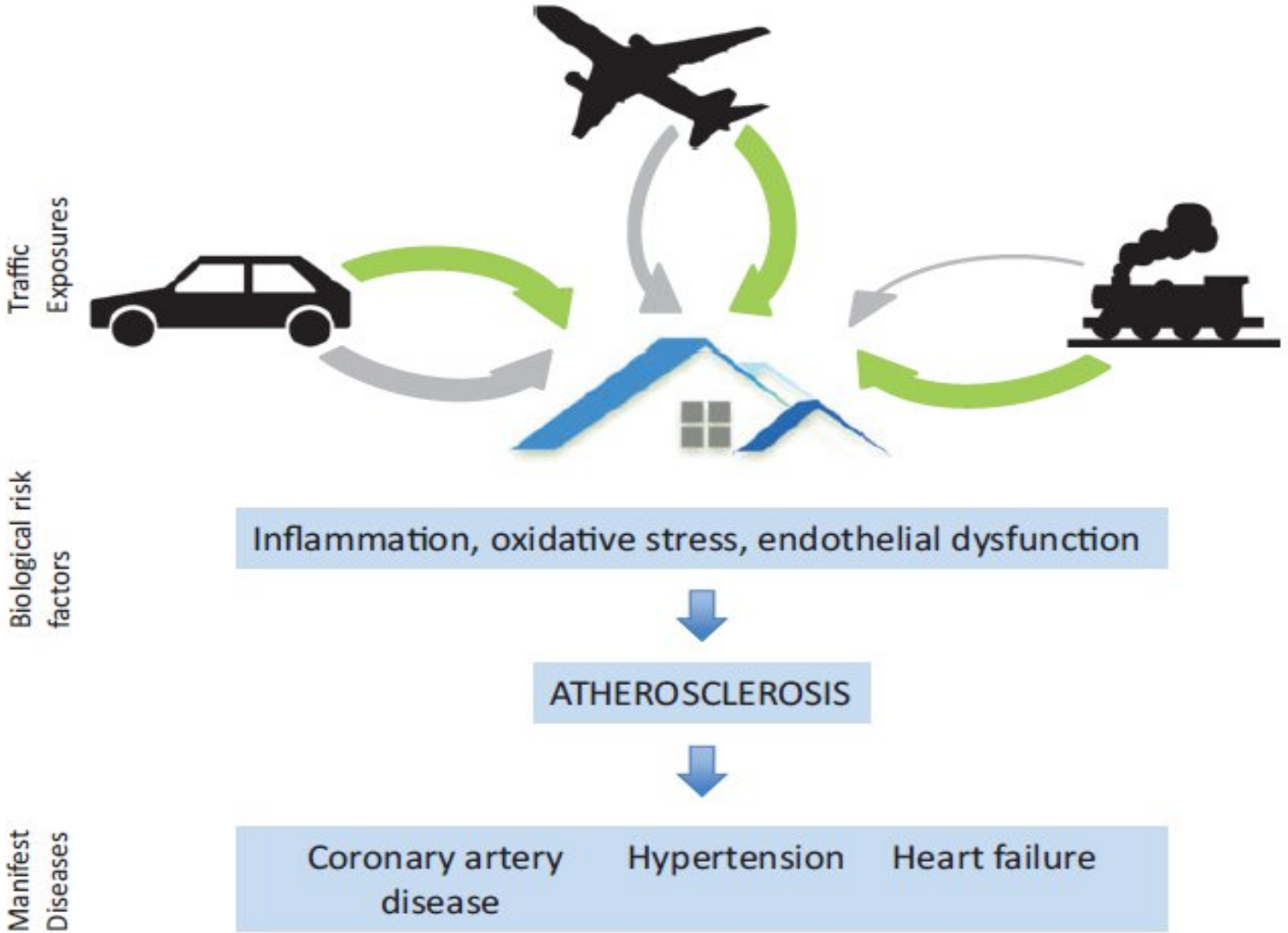
Linear hazard ratios for associations between road, railway, and aircraft noise exposure and myocardial infarction per 10 dB increase in L_{den}

4.40 million adults (>30 years) in the Swiss National Cohort (SNC)



Heritier H, et al. Eur Heart J 2019;40;598–603

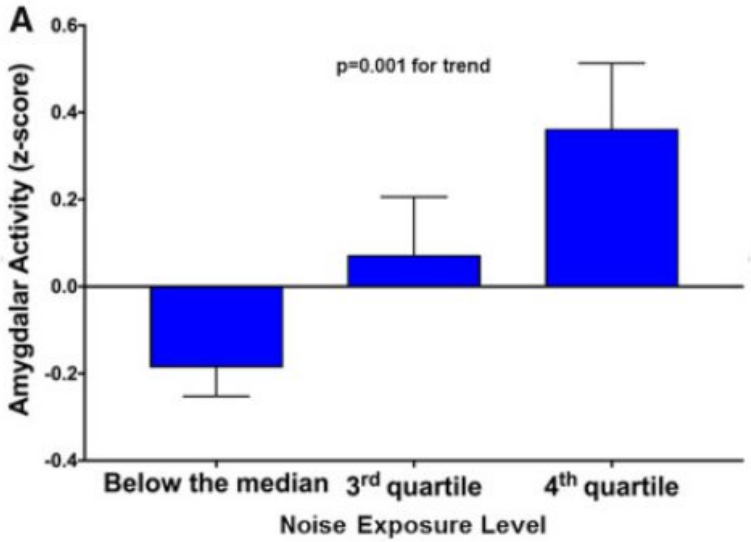
Potential common pathophysiological pathways for induction of cardiovascular disease by air pollution and transportation noise



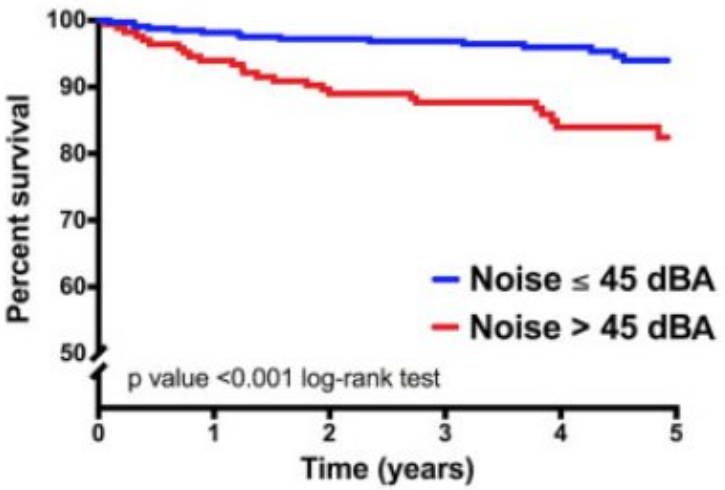
Sorensen M and Pershagen G. Eur Heart J 2019;40;604–606

Neurobiological mechanism linking noise to CVD

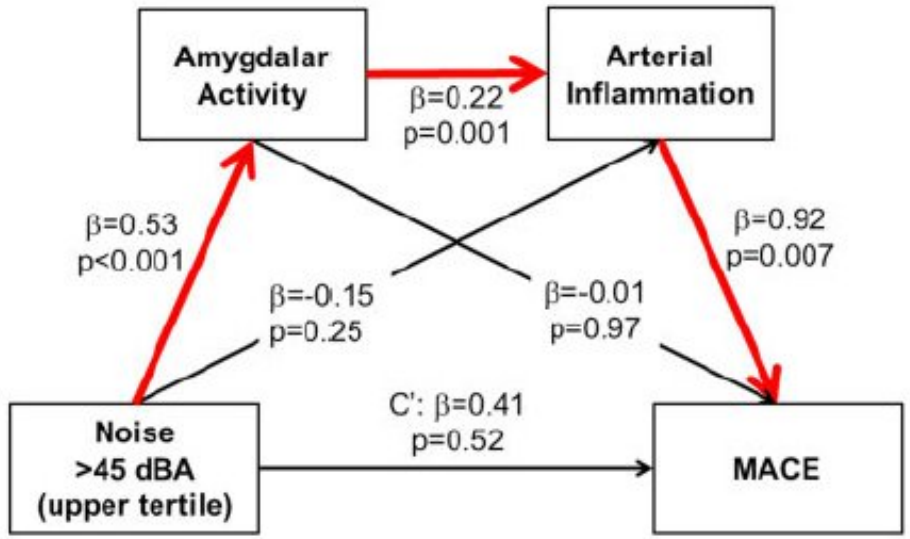
Amygdalar metabolic activity by noise exposure



MACE event-free survival by noise exposure

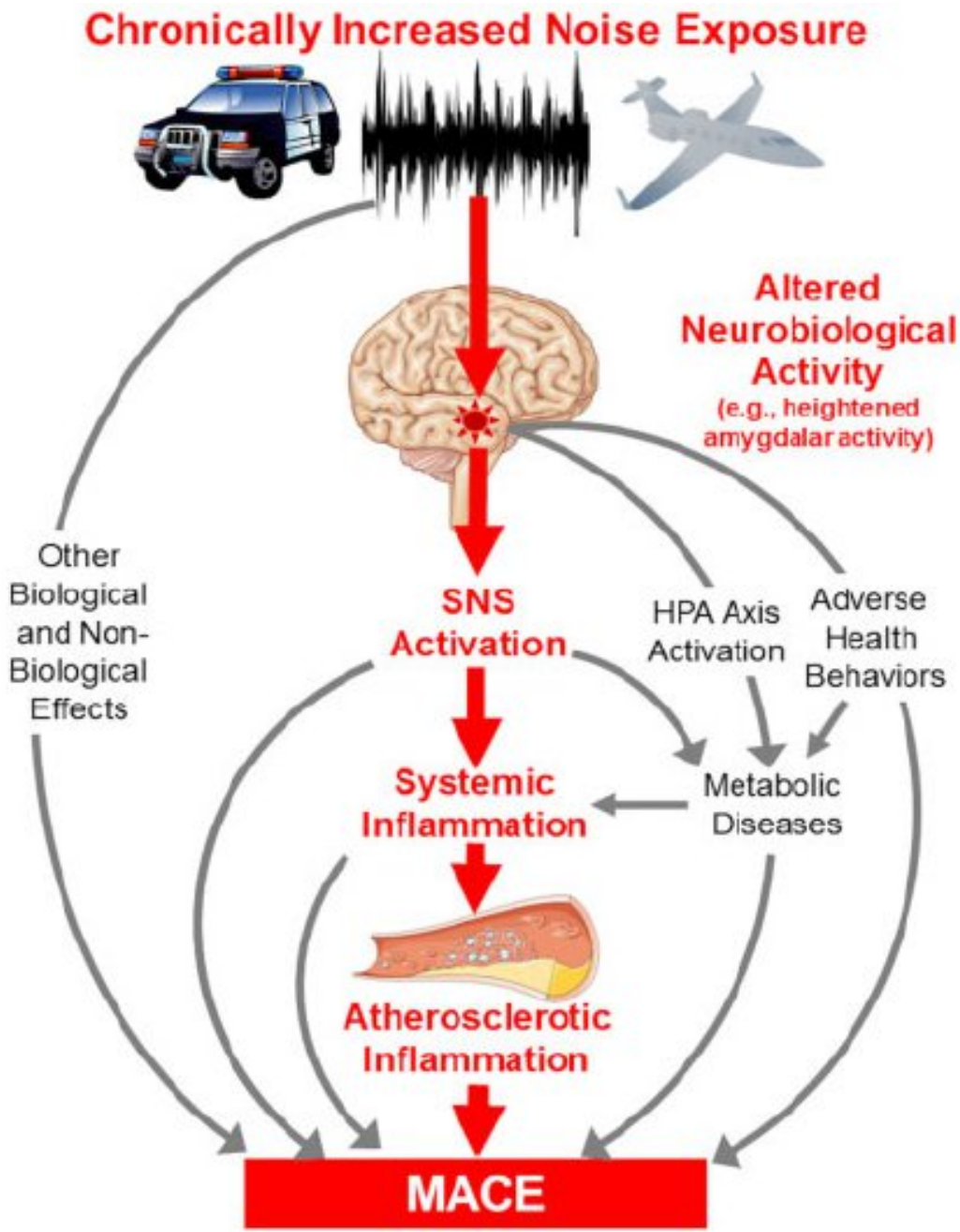


Mediation analysis for the hypothesized pathway from noise exposure to MACE



Osborne MT, et al. *Eur Heart J.* 2020;41:772–782

Mechanism linking noise exposure to major adverse cardiovascular disease events through upregulated amygdalar metabolic activity and arterial inflammation



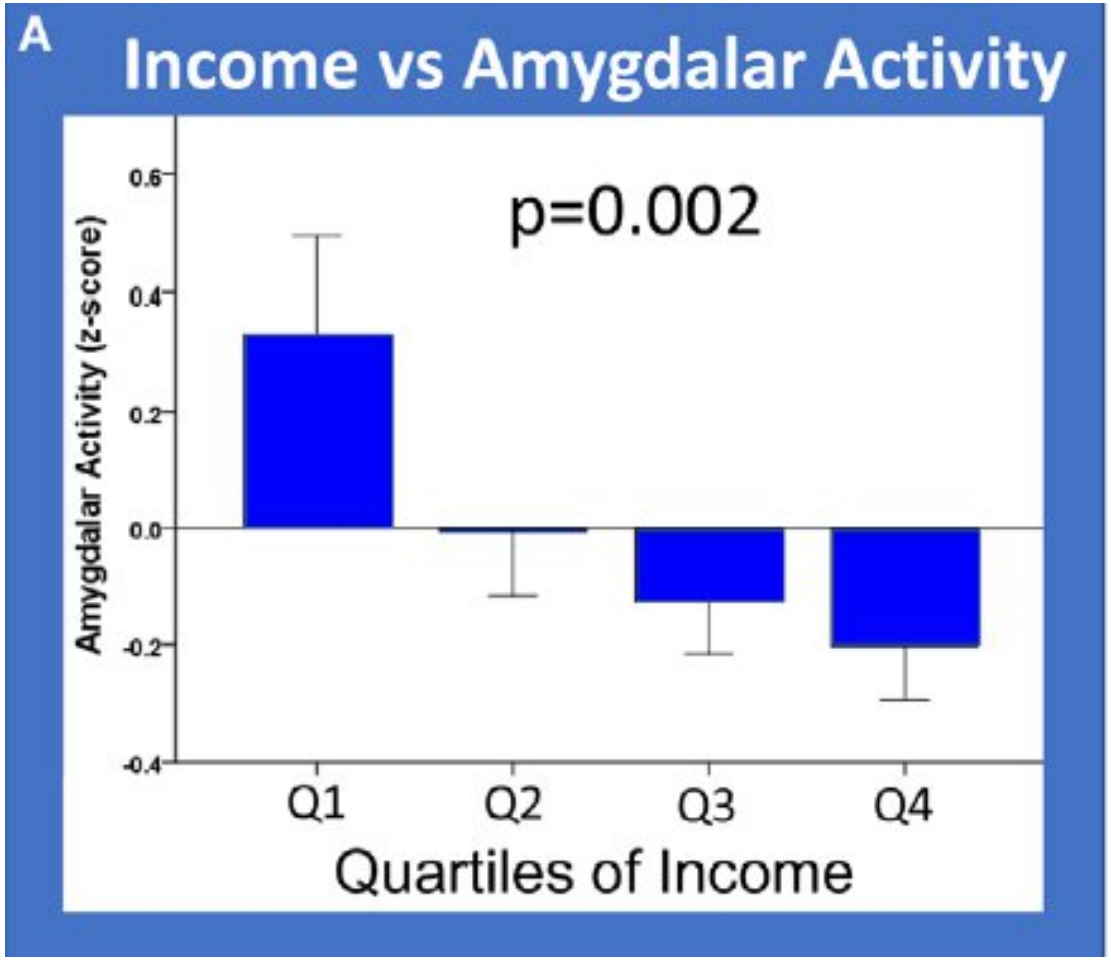
Osborne MT, et al. *Eur Heart J.* 2020;41:772–782

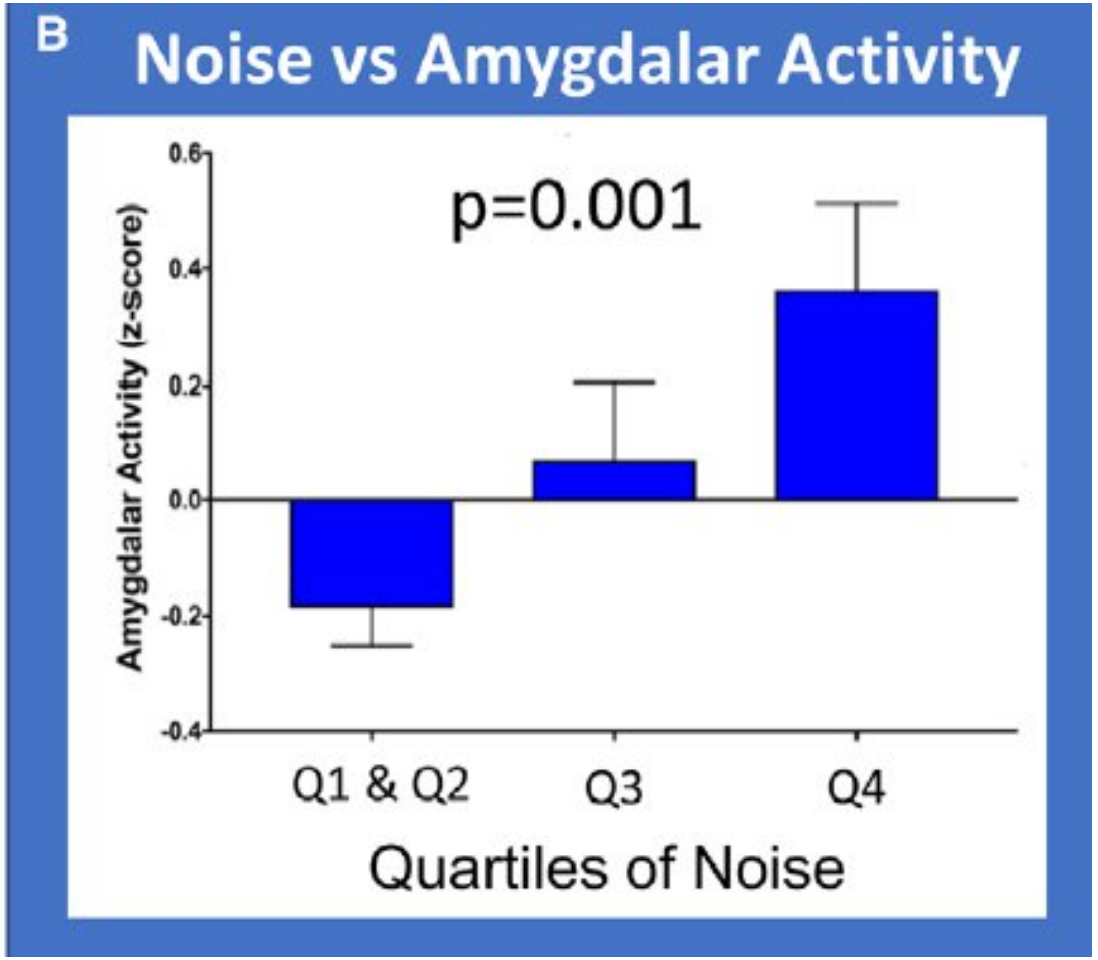
World Health Organization
REGIONAL OFFICE FOR Europe

ENVIRONMENTAL NOISE GUIDELINES

for the European Region

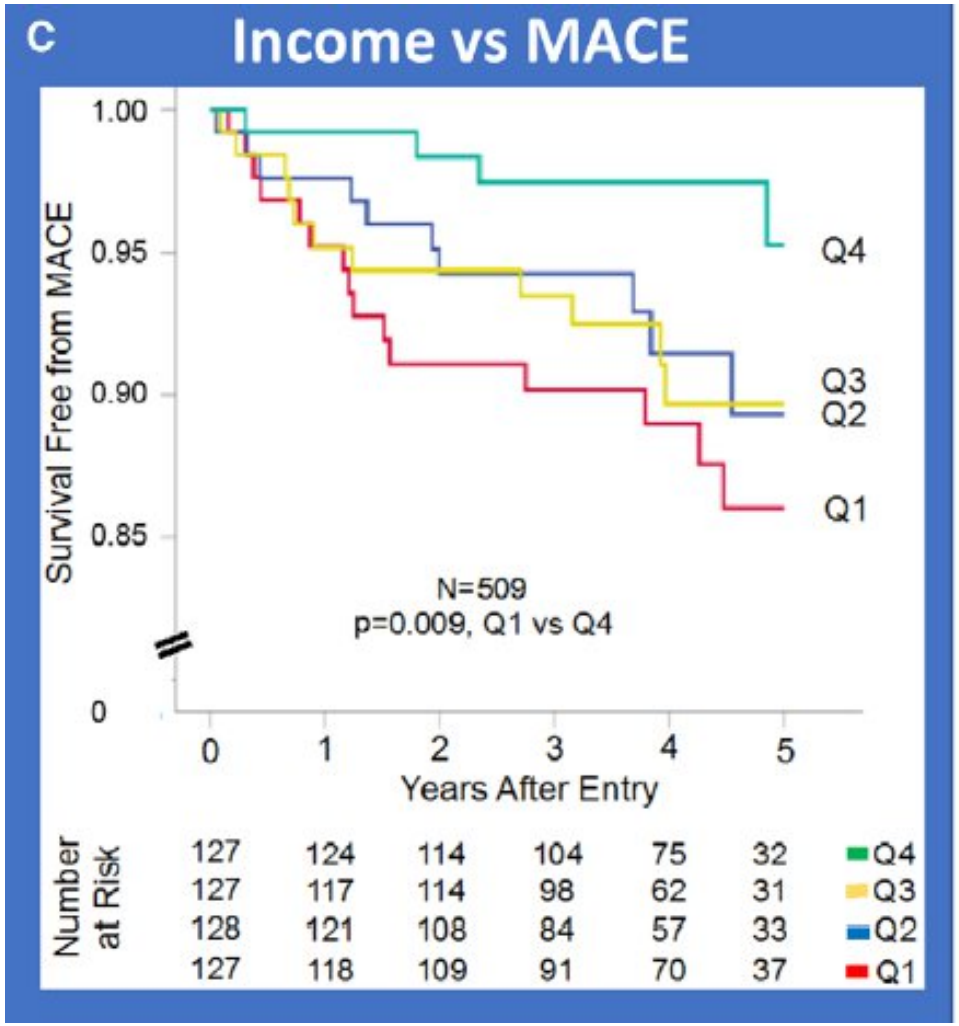
Socioeconomic and environmental stressors vs stress-associated neurobiological activity and cardiovascular disease events

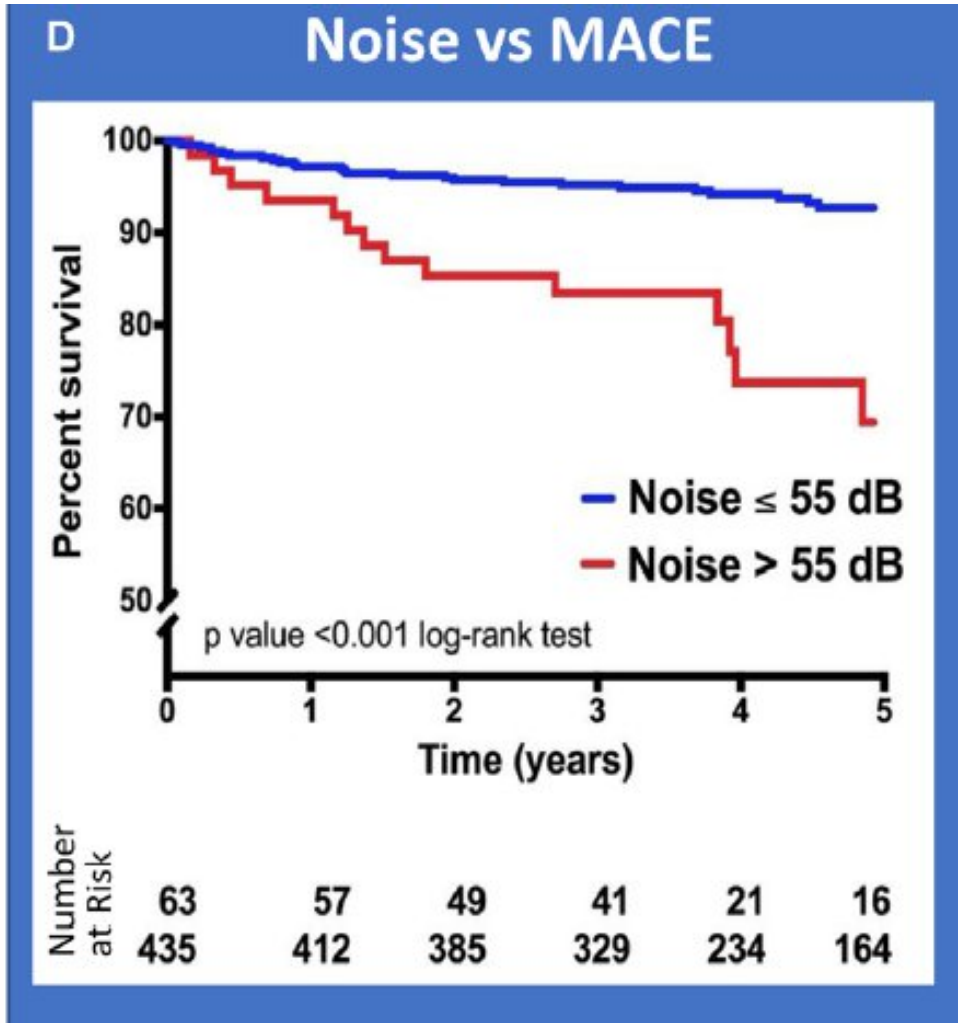




Osborne MT, et al. *Circ Cardiovasc Imaging*. 2020;13:e010931
Tawakol A, et al. *J Am Coll Cardiol*. 2019;73:3243–3255.

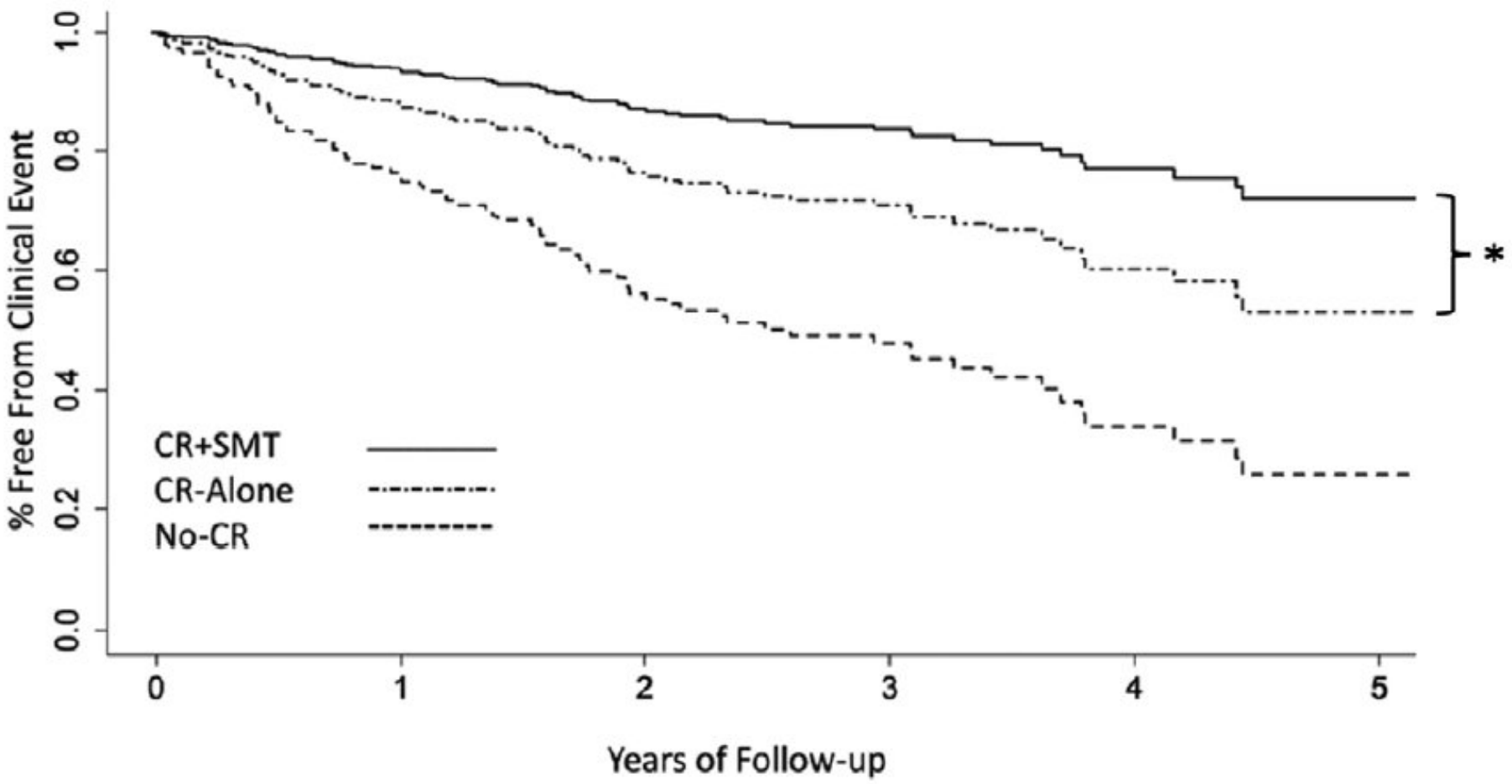
Socioeconomic and environmental stressors vs stress-associated neurobiological activity and cardiovascular disease events





Osborne MT, et al. *Circ Cardiovasc Imaging*. 2020;13:e010931
Tawakol A, et al. *J Am Coll Cardiol*. 2019;73:3243–3255.

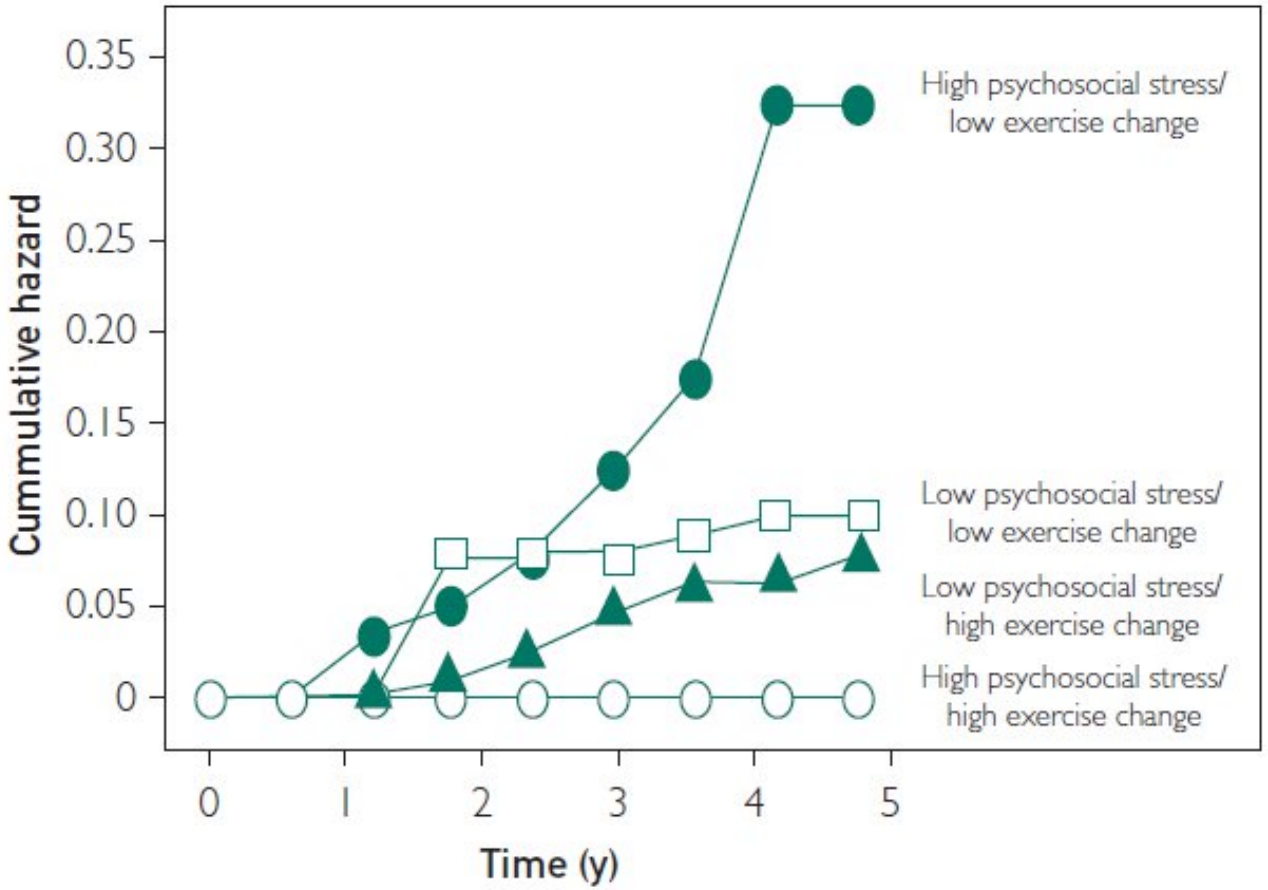
Impact of stress reduction on cardiovascular disease events



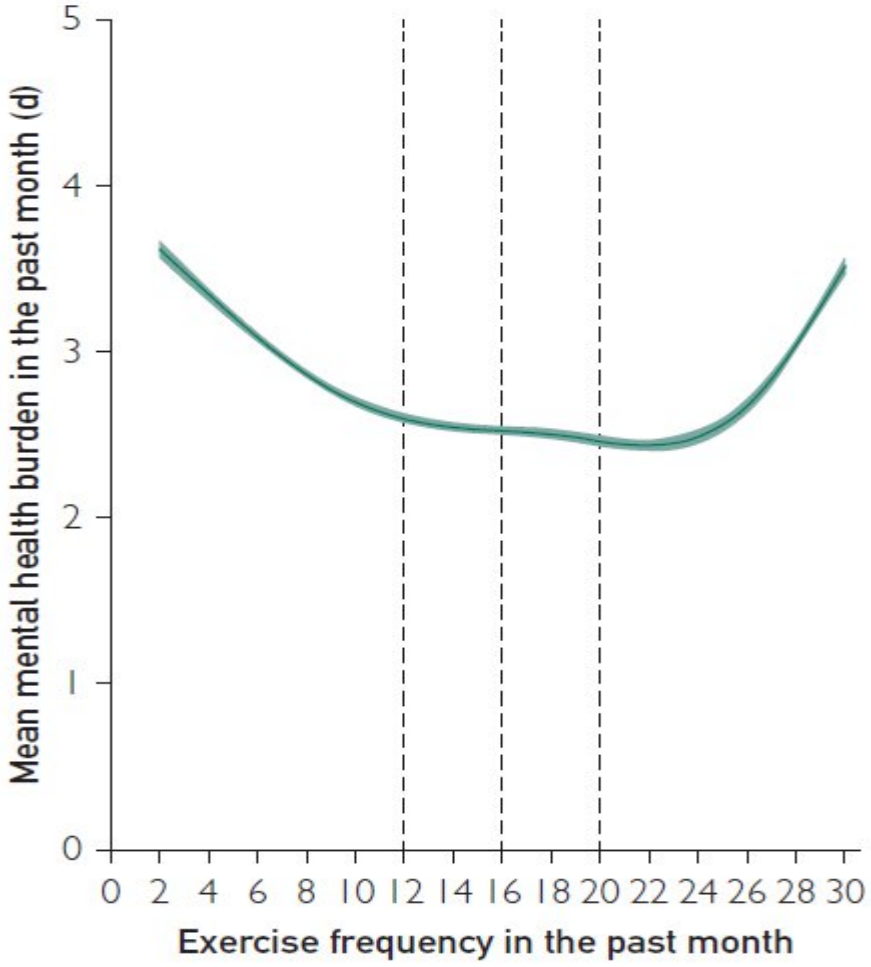
Osborne MT, et al. *Circ Cardiovasc Imaging*. 2020;13:e01093
Blumenthal JA, et al. *Circulation*. 2016;133:1341–1350.1

Exercise Counteracts the Cardiotoxicity of Psychosocial Stress

Actuarial cumulative hazard plot for survival time based on changes in exercise capacity after exercise training split by baseline psycosocial stress



Mental health burden as a function of exercise frequency



O'Keefe EL, et al. Mayo Clin Proc. 2019;94:1852-1864
Milani RV, Lavie CJ. Am J Med. 2009;122:931-938
Chekroud SR, et al. Lancet Psychiatry. 2018;5:P739-P746

NDVI Relationships to Health Outcomes in Unadjusted and Adjusted Models Adjusting for Individual Sociodemographics, Neighborhood Income

Relationship of Neighborhood Greenness to Heart Disease in 249 405 US Medicare Beneficiaries

AMI		
Low NDVI	1	
Medium NDVI	0.94 (0.77–1.14)	0.5231
High NDVI	0.75 (0.63–0.90)§	0.0018§
P value linear trends‖		0.0231§
IHD		
Low NDVI	1	
Medium NDVI	0.97 (0.94–1.01)	0.0961
High NDVI	0.80 (0.77–0.83)§	<0.0001§
P value linear trends‖		<0.0001§
HF		
Low NDVI	1	
Medium NDVI	1.02 (0.97–1.07)	0.4930
High NDVI	0.84 (0.80–0.88)§	<0.0001§
P value linear trends‖		<0.0001§
AF		
Low NDVI	1	
Medium NDVI	0.91 (0.84–0.97)§	0.0062§
High NDVI	0.94 (0.87–1.00)	0.0674
P value linear trends‖		0.3435

Wang K, et al. J Am Heart Assoc 2019;8:e010258. DOI:10.1161/JAHA.118.010258

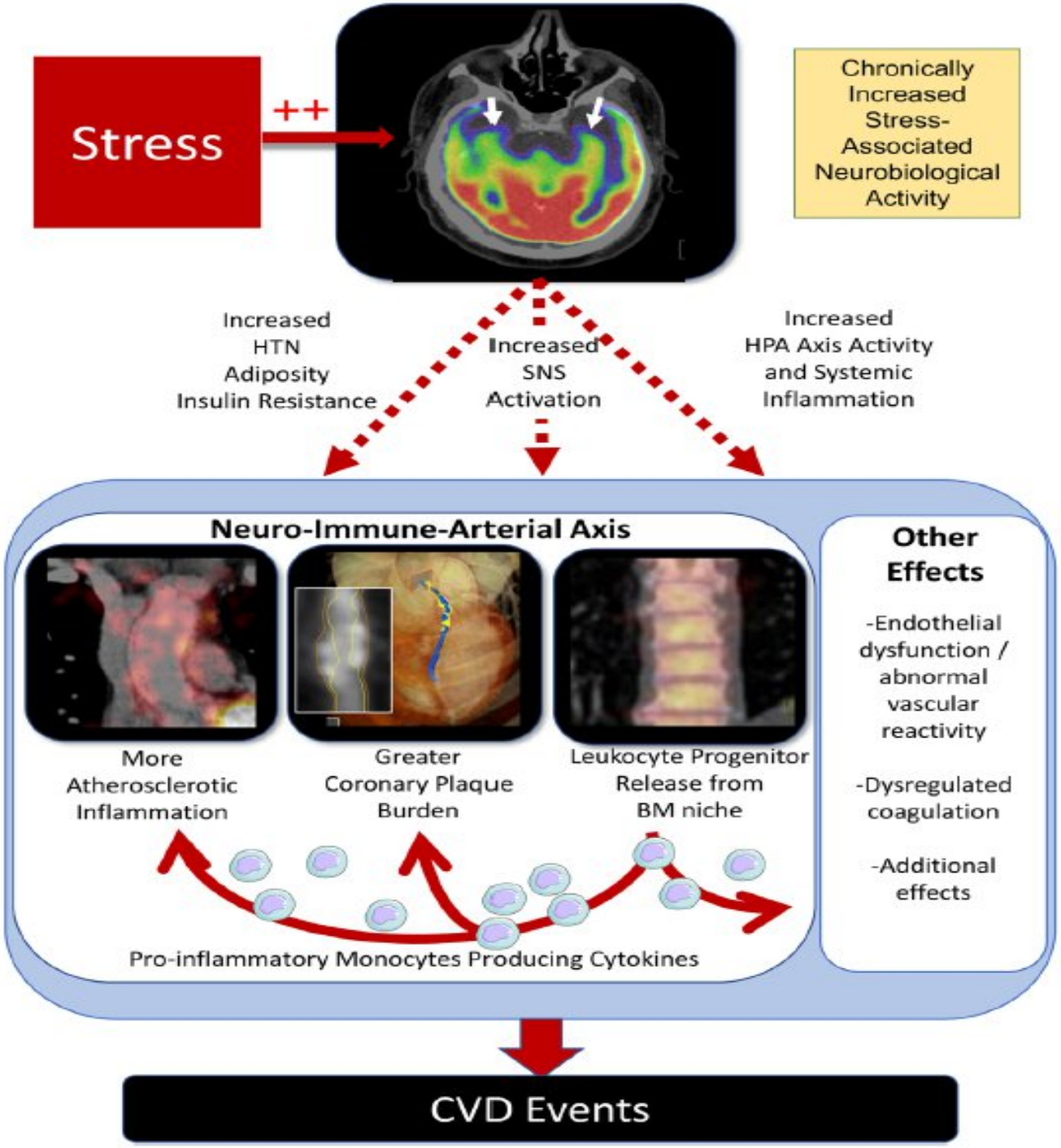
Key points

- **Lo stress è un fattore di rischio cardiovascolare diffuso, pervasivo e largamente sottostimato.**
  Fattori psicosociali (come la condizione socio-economica) e ambientali (come l'inquinamento acustico) attraverso vie neurobiologiche comuni, associate allo stress, che implicano l'iperattività dell'amigdala, l'infiammazione arteriosa e l'attivazione del midollo osseo possono favorire eventi cardiovascolari.

- **Sono necessari interventi mirati a ridurre lo stress nella popolazione più esposta e a maggior rischio e studi ulteriori che confermino le vie biologiche ipotizzate e identifichino gli strumenti migliori per inibirle.**


grazie per l'attenzione
By Renzo Rizzo

Mechanisms linking stress to cardiovascular disease



Osborne MT, et al. *Circ Cardiovasc Imaging*. 2020;13:e010931

religions

MDPI

Article

The Study on Stress, Spirituality, and Health (SSSH): Psychometric Evaluation and Initial Validation of the SSSH Baseline Spirituality Survey

Erica T. Warner [1,2,†], Blake Victor Kent [2,3,*,†], Ying Zhang [4], M. Austin Argentieri [2,5], Wade C. Rowatt [6], Kenneth Pargament [7], Harold G. Koenig [8], Lynn Underwood [9], Shelley A. Cole [10], Martha L. Daviglus [11], Alka M. Kanaya [12], Julie R. Palmer [13], Tianyi Huang [14], Mark A. Blais [15] and Alexandra E. Shields [1,2]

Abstract: This paper describes the development and initial psychometric testing of the baseline Spirituality Survey (SS-1) from the Study on Stress, Spirituality, and Health (SSSH). The SS-1 contains a mixture of items selected from validated existing scales and new items generated to measure important constructs not captured by existing instruments, and our purpose here was to establish the validity of new and existing measures in a racially/ethnically diverse sample. Psychometric properties of the SS-1 were evaluated using standard psychometric analyses in 4563 SSSH participants. Predictive validity of SS-1 scales was assessed in relation to the physical and mental health component scores from the Short-Form 12 Health Survey (SF-12). Scales exhibited adequate to strong psychometric properties and demonstrated construct and predictive validity. Overall, the correlational findings provided solid evidence that the SS-1 scales are associated with a wide range of relevant R/S attitudes, mental health, and to a lesser degree physical health.