

Occupational Medicine

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Psychosocial working conditions and coronary heart disease: new evidence and implications for prevention

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Your lecturer: Johannes Siegrist



- Professor emeritus of Medical Sociology, Heinrich-Heine-University Düsseldorf, Germany (HHU)
- Past Director, Institute of Medical Sociology and Public Health Program, HHU
- Long-standing research on social determinants of health; esp. psychosocial work environment and health
- Collaboration with WHO and ILO
- Recent book: J. Siegrist, J. Li: Psychosocial Occupational Health. Oxford Univ. Press 2024

<https://www.uniklinik-duesseldorf.de/patienten-besucher/klinikeninstitutezentren/institut-fuer-medizinische-soziologie/das-institut/forschung/>

Coronary heart disease (CHD)

- CHD is the most common type of CVD, and, with some 9 million deaths annually, the leading cause of all health loss globally.
(Roth GA et al. JACC 2017, 70(1); Martin SS et al. Circulation 2024, 149(8))
- Despite declining incidence and lethality in developed countries, CHD is still a major contributor to premature mortality and DALYs.
- Due to its multiple risk factors, CHD, as a biopsychosocial disease, continues to be a major challenge for medical and public health-related prevention.
- In addition to established biological, biomedical, and behavioural risk factors, a number of psychological (e.g. depressive mood) and socio-environmental risk factors were documented.
- Among the latter, distinct working conditions were identified.

Being employed or self-employed is a **major goal** in adult life. It determines a wide range of **life chances**:

- It provides continuously earned **income** and economic **independence**
- It confers a **social status** within society and strengthens a person's **social identity**, providing formal **membership** and basic **social security**
- It offers opportunities for **skill development, training and promotion**
- It structures **time**, strengthens **motivation** and pro-active **behaviour**
- It meets important human needs of **autonomy**, self efficacy, **recognition**, and related self- esteem
- Depending on its **quality**, paid **work exerts powerful positive and negative effects on health** and well-being, due to its extended and intrusive **impact over the life course**

Major changes in the modern world of work and employment

- **Growth of the tertiary sector (service and ICT occupations and professions); de-industrialisation**
- **Extended economic globalization:**
 - **Growth** through global expansion of free-marketed principles and technological innovations, generating **large flows of transnational capital, trade, and workforce** (IMF, WTO, World Bank)
 - Increase of **transnational competition, job insecurity, financial instability** and environmental **degradation**
- **Digitization, automation and artificial intelligence (AI):**
 - **Microelectronic revolution** (PCs, Internet), **industry 4.0)**
 - **AI:** Application of Machine Learning Models (GPT-4) and Large Language Models (LLMs) to old and new jobs (**knowledge work**), with increased risks of **job loss**

How does work impair your health? Two main pathways:

➤ Physical / chemical/ biological factors:



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Injury

Hearing loss

Dust (e.g. asbestos)

Heavy lifting

Disability

Heavy noise

Lung disease, Cancer

Back pain

➤ Psychosocial factors:



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Long working hours

High work pressure

Job loss

Cardiovascular disease?

Hypertension?

Depression?

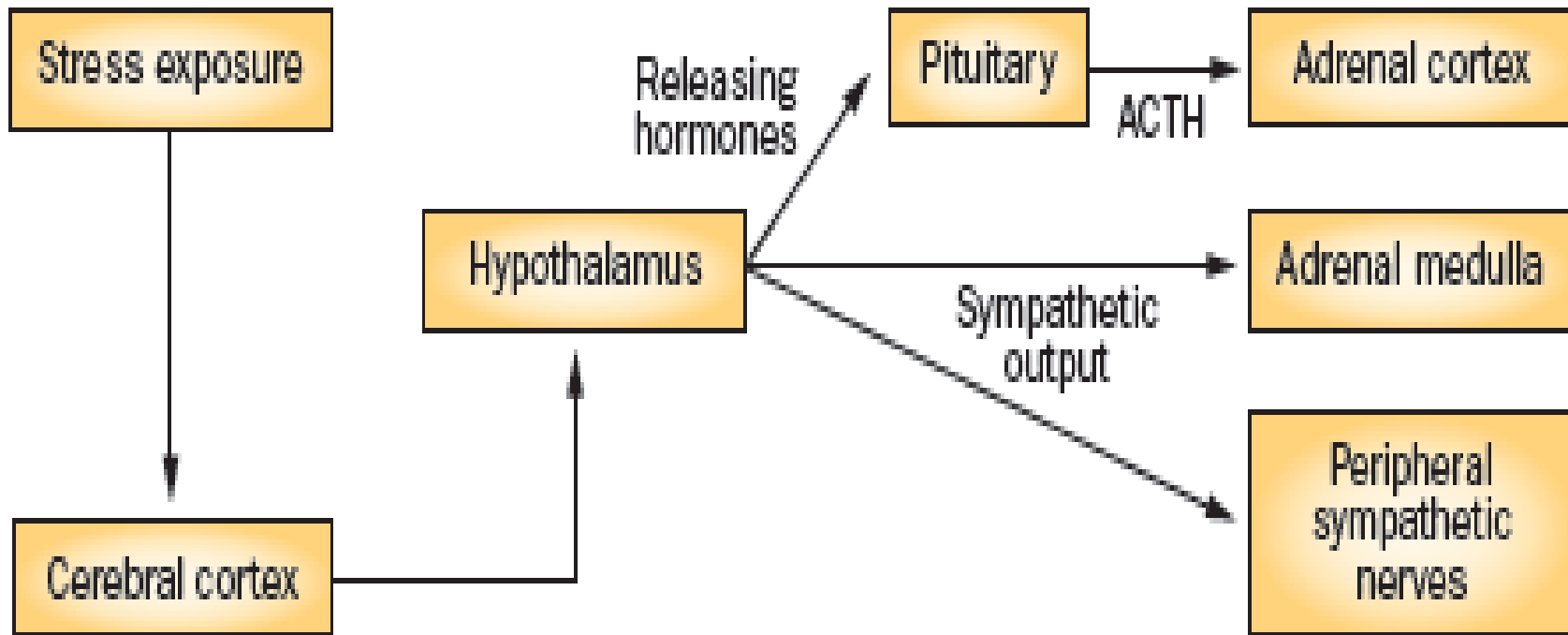
Modern work: A major role of psychosocial work environments

Psychosocial work environment:

- Umbrella term for **non-material working conditions** with relevance for health that are experienced and processed through the **brain's cognitive and emotional appraisal**.
- If defined as **threat**, these experiences **activate** the organism's **stress axes** > Biopsychosocial model of health and disease
- Scientific challenge: How to **define** and **measure threatening psychosocial work environments** within the complexities of modern work and employment?
- To this end, a **theoretical model** is required that selects **distinct components** at a high level of abstraction, where the **interaction** of these components **explains** the pathway to disease

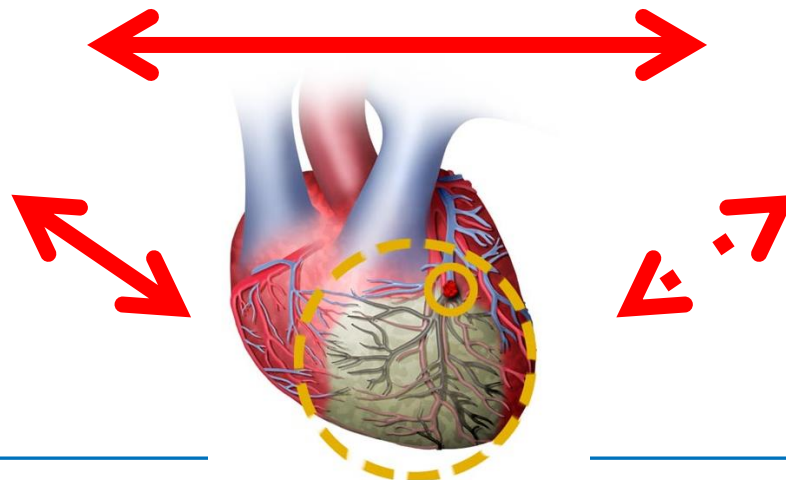
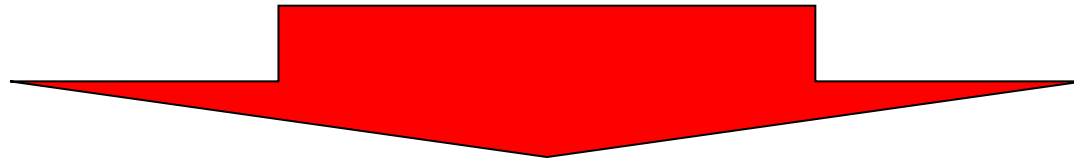
- Recurrent stimuli (,stressors‘) of the work environment that are perceived as **threatening** by the working person.
- Threat:
 - an expected **physical or interpersonal** assault or **harm** (e.g. mobbing, discrimination);
 - an expected **loss of control** over one’s agency (e.g. failed performance)
 - an expected **offense of one’s social identity** (e.g. depreciation, job loss)
- The experience of threat evokes **negative emotions** in the cortico-limbic brain structures that activate two pathways:
 - **Behavioural reactions** (,fight-flight‘, coping activities)
 - Activation of **physiological stress responses** (SAM-, HPA axis) with adverse long-term effects on health
- If **chronically experienced** in everyday working life, **stressors** often **bypass cognitive awareness** (,habituation‘), yet activate the cortico-limbic structures.

Stress-theoretical basis:



Sustained stress reactions → allostatic load
→ disease development

Theoretical models analysing main health outcomes

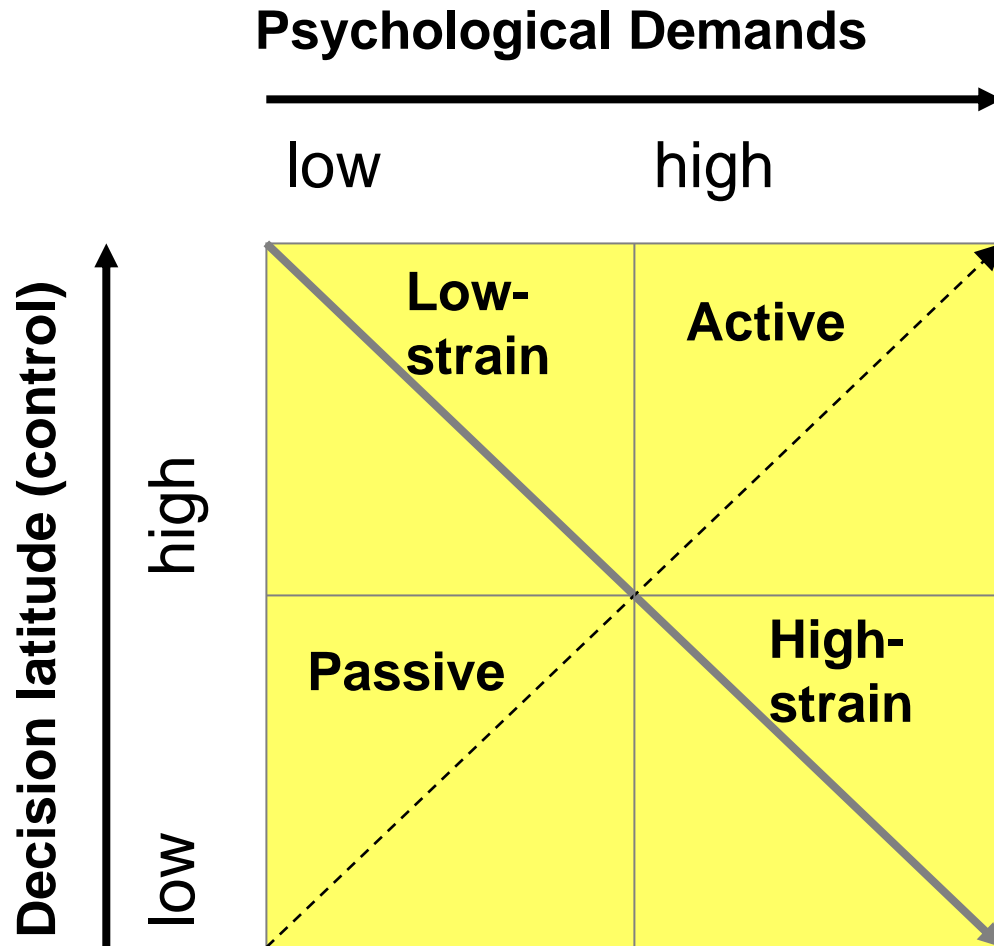


Three complementary models of a stressful psychosocial work environment

- **Demand-control model**
(R. Karasek, 1979;
R. Karasek & T. Theorell,
1990)
 - Focus on job task profile: high demand/low control
- **Effort-reward imbalance model**
(J. Siegrist, 1996;
J. Siegrist et al., 2004)
 - Focus on work contract: high 'cost'/low 'gain'
- **Organizational injustice model**
(J. Greenberg et al., 1982;
M. Elovainio et al., 2002)
 - Focus on unfair procedures and interactions

The demand-control (DC) model

(R. Karasek 1979, R. Karasek, T. Theorell 1990)

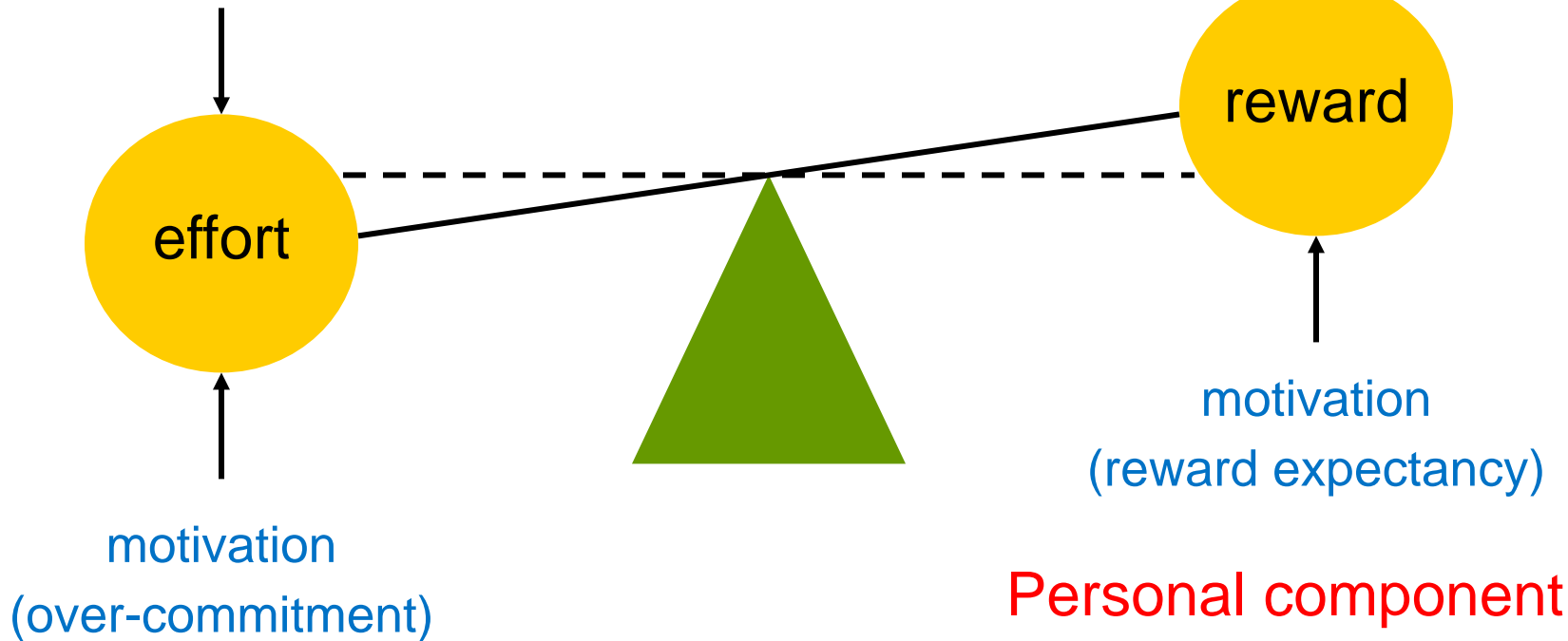


The effort-reward imbalance (ERI) model (J. Siegrist 1996)

Situational components

- Money: wage, salary
- Status: mobility / job security
- Recognition: esteem

Demands / Obligations



Explanation of the model

- Violation of an evolutionary old principle of social exchange – the reciprocity between ,give‘ and ,take‘ – is a stressful experience, especially so in a core social role, the work role.
- Recurrent experience of high effort/low reward at work occurs rather frequently in a globalized economy (e.g. high competition; lack of alternative job; job instability, low skill level).
- Three dimensions of reward are essential – and of similar significance for health and well-being at work: Salary or wage; Control of one’s social status (security, promotion); Esteem/appreciation.
- While extrinsic factors – challenges, threats and rewards –matter most, intrinsic factors of the working person contribute to this imbalance as well, specifically the way of coping with the demands at work (over-commitment).

Reasons to focus on these models

1. They are rooted in basic concepts of psychobiological stress research:

DC model:

High demand: Activation of the sympatho-adrenomedullary axis

Low control: Loss of control over one's agency: Activation of the hypothalamic-pituitary-adrenocortical (HPA) axis

ERI model:

High effort: Activation of the sympatho-adrenomedullary axis

Low reward: Threat to one's self-esteem (depreciation, job loss):
Activation of brain reward circuitry (Insula, Amygdala, HPA axis)

2. They have been studied in many prospective epidemiologic investigations internationally, using validated standardized measures

Whitehall II, IPD-Work consortium, ELSA, SOEP, NAKO, CONSTANCES, HRS, DWCS, FPSS, ELSA-Brasil

Both models are measured by standardized self-administered questionnaires containing psychometrically validated scales:

➤ **Job Content Questionnaire (JDQ)**

Karasek R et al. (1998) J Occup Health Psychol 3: 322

➤ **Effort-Reward Imbalance Questionnaire (ERI-Short):**

Leineweber C et al. (2010) Occup Environ Med 67: 526

These questionnaires are available in a variety of reliable language versions.

Alternatively, the models are measured by a job exposure matrix.

Adequate research design to test causal associations:

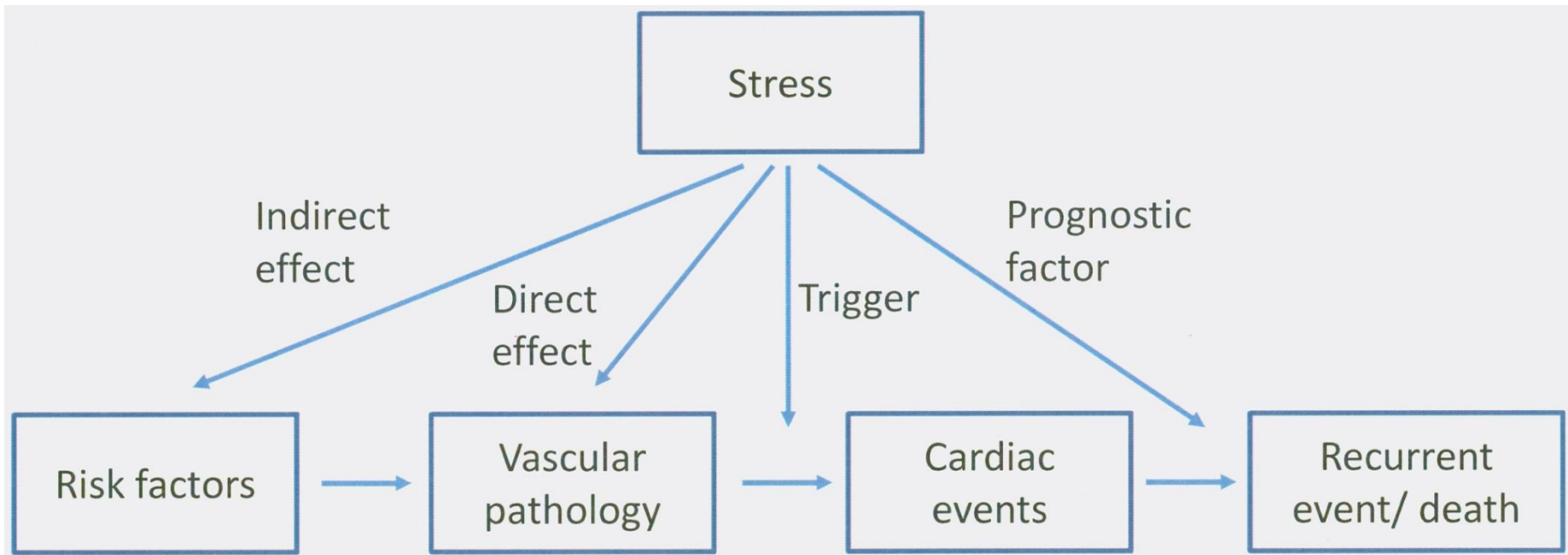
Prospective observational cohort study of a large sample of working population, with adjustments and consecutive assessments of health outcomes

➤ **Bradford Hill criteria of causal associations** in epidemiologic studies

Questions to be answered

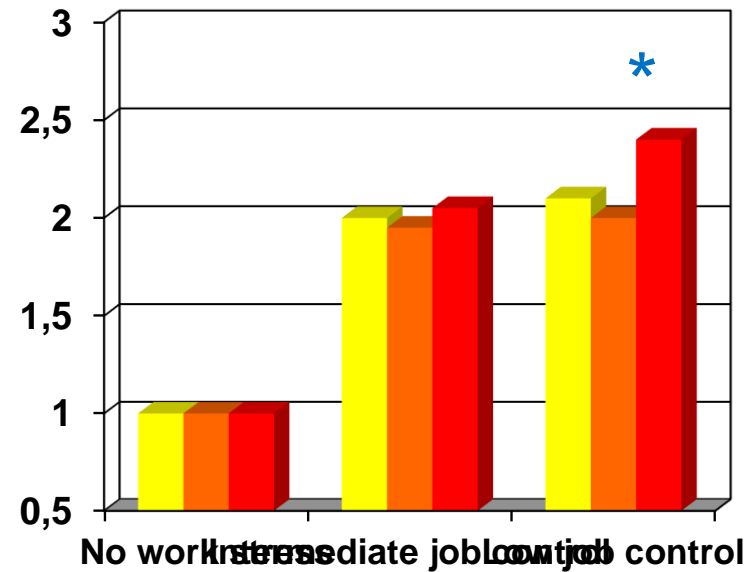
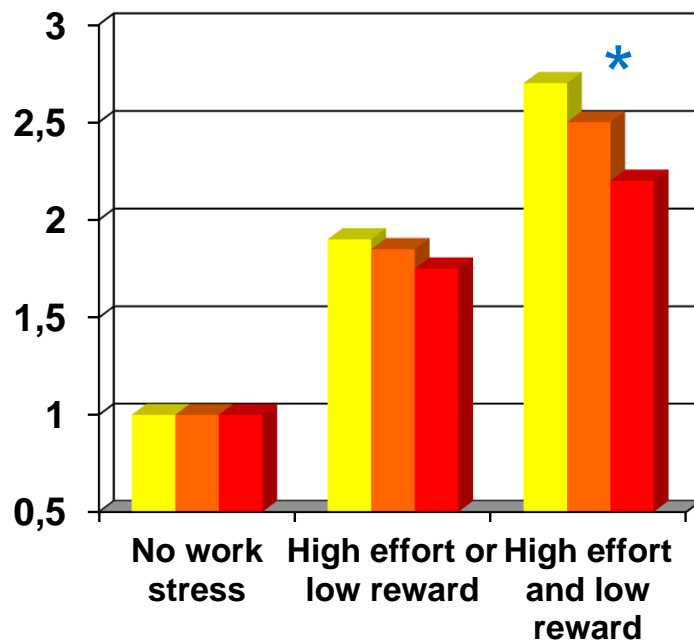
1. What is the **current evidence** on **associations** between stressful work (in terms of these models) and **elevated risks of CHD**?
 - Incident disease (AMI) and recurrent CHD
 - Subclinical disease and major risk factors
 - Psychobiological pathways
 - Links between CHD and depression
2. What are the **practical implications** of these results for recognition, assessment, prevention and treatment of CHD?
 - Occupational health professions and services
 - Prevention strategies at organisational/company level
 - National labour and social policies

1. Work stress and coronary heart disease



Selected empirical evidence: Work stress and incident coronary heart disease

The Whitehall II Study; ORs; N= 10,308 men and women



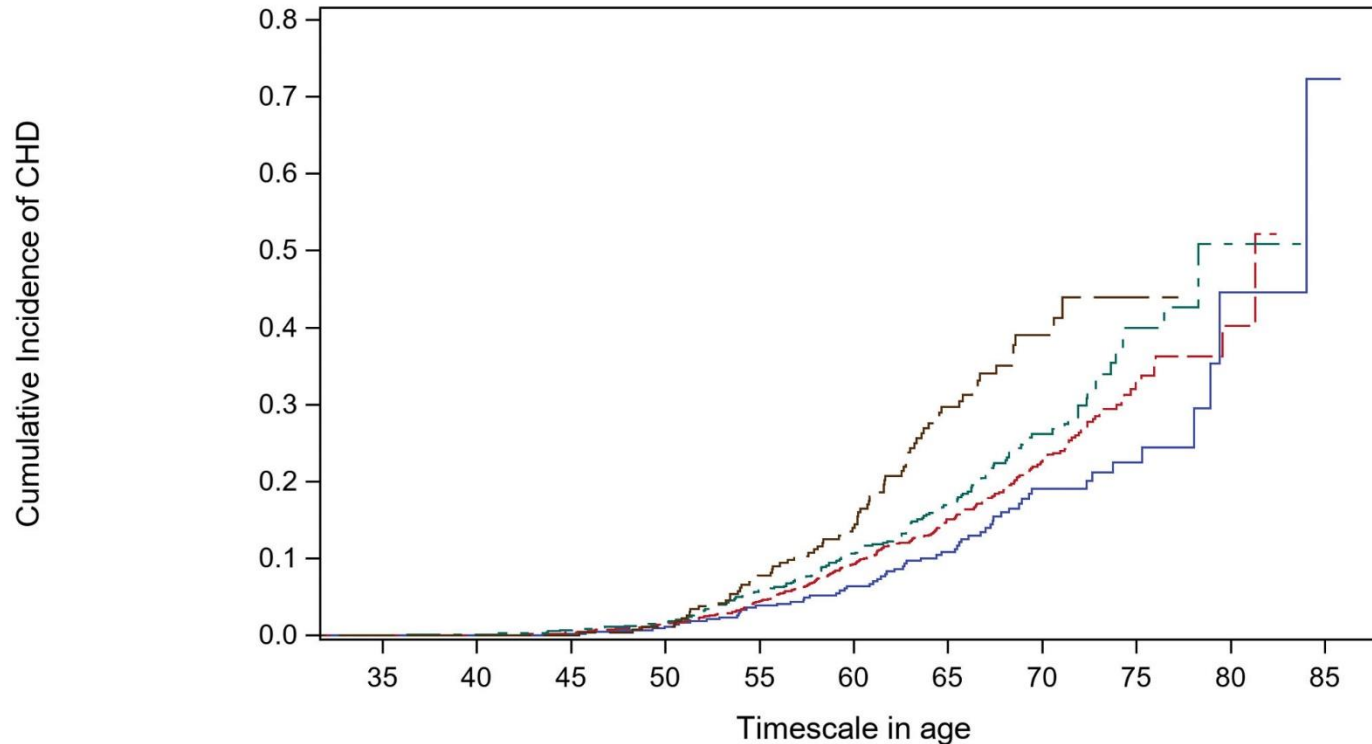
- adjusted for age, sex, length of follow-up
- + alternative work stress model
- + grade, coronary risk factors, negative affect

* $p < .05$

Somewhat lower, but significant risk elevations for IHD: PROQ-Study: 18 year follow-up; N= 3118 men

A Kaplan-Meier survival curves for the age before coronary heart disease among men: combined exposure to job strain and effort-reward imbalance

With Number of Subjects at Risk



HR = **1.95** (1.32: 2.87), adj for 15 risk factors

Unexposed	431	431	429	422	374	308	220	110	45	6	1
Low exposure	1678	1677	1671	1635	1467	1203	810	360	79	10	0
Intermediary exposure	741	740	735	720	648	516	337	130	31	3	0
Double exposure	268	268	268	261	227	172	94	30	5	0	0

Meta-analysis of prospective studies: Work stress (DC, ERI, OJ) and risk of CHD

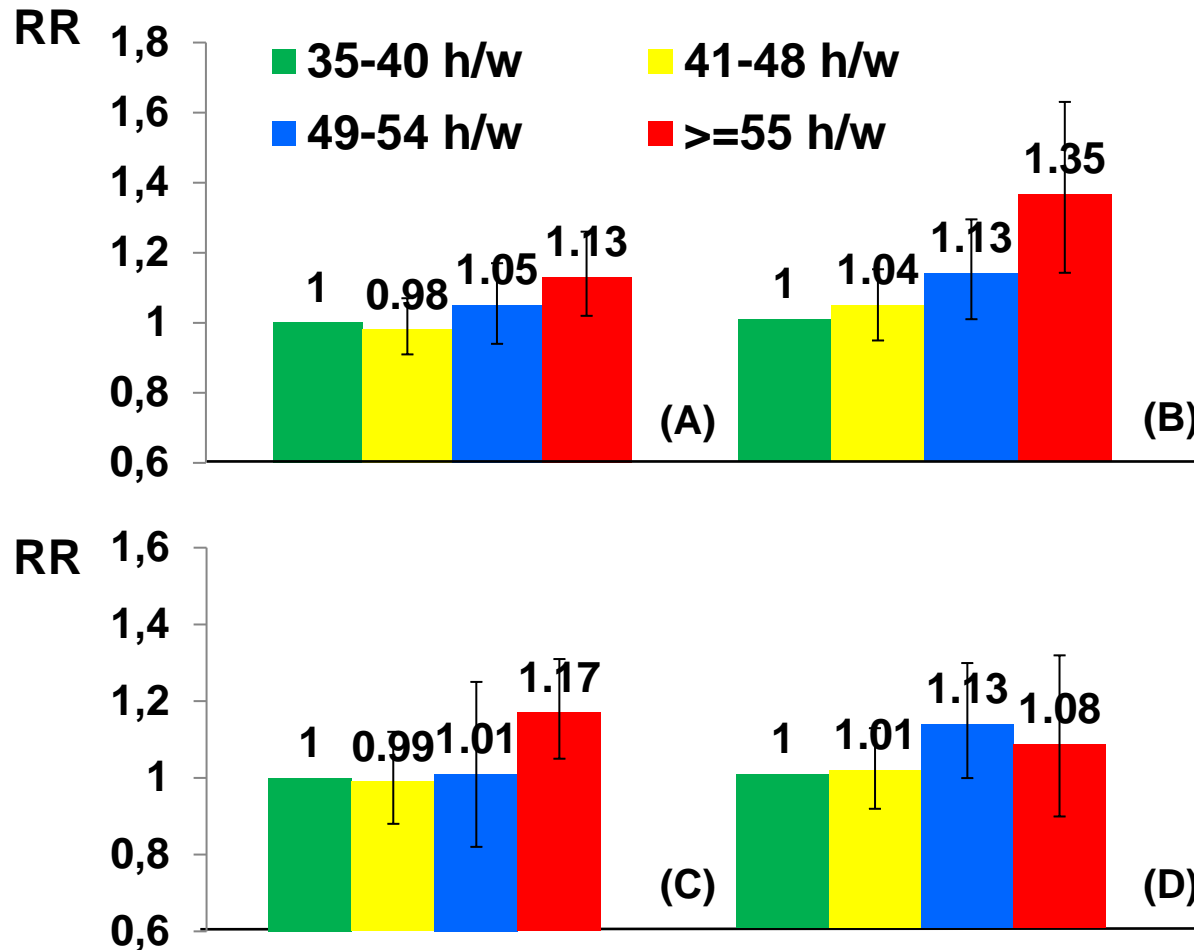
Pooled risk estimates of IHD or mortality according to different psychosocial exposures at work, based on systematic reviews (SR) and individual studies (IS)

First author (year)	Study type	Exposure	Health outcome	RR (95% CI)
Kivimäki et al., (2012)	SR	Job strain	Incident IHD	1.23 (1.10; 1.37)
Dragano et al., (2017)	SR	Effort-reward	Incident IHD	1.16 (1.00; 1.35)
Dragano et al., (2017)	SR	Job strain + Effort reward	Incident IHD	1.41 (1.12; 1.76)
Niedhammer et al., (2021)	SR	Job insecurity	Incident IHD	1.32 (1.09; 1.59)
Li et al., (2015)	SR	Job strain + Effort reward	Recurrent IHD	1.65 (1.23; 2.22)
Trudel et al., (2021)	IS	Job strain + LWH	Recurrent IHD	2.55 (1.30; 4.98)
Kivimäki et al., (2018)	IS	Job strain (with CMD)	Mortality (men)	1.68 (1.19; 2.35)
Kivimäki et al., (2018)	IS	Effort reward (without CMD)	Mortality (men)	1.22 (1.06; 1.41)
Niedhammer et al. (2021)	SR	Organisational justice	Cardiovascular mortality	1.62 (1.24; 2.13)

CMD = cardiometabolic disease; LWH = long working hours; RR = relative risk

Proxy of high demand/effort: Long working hours

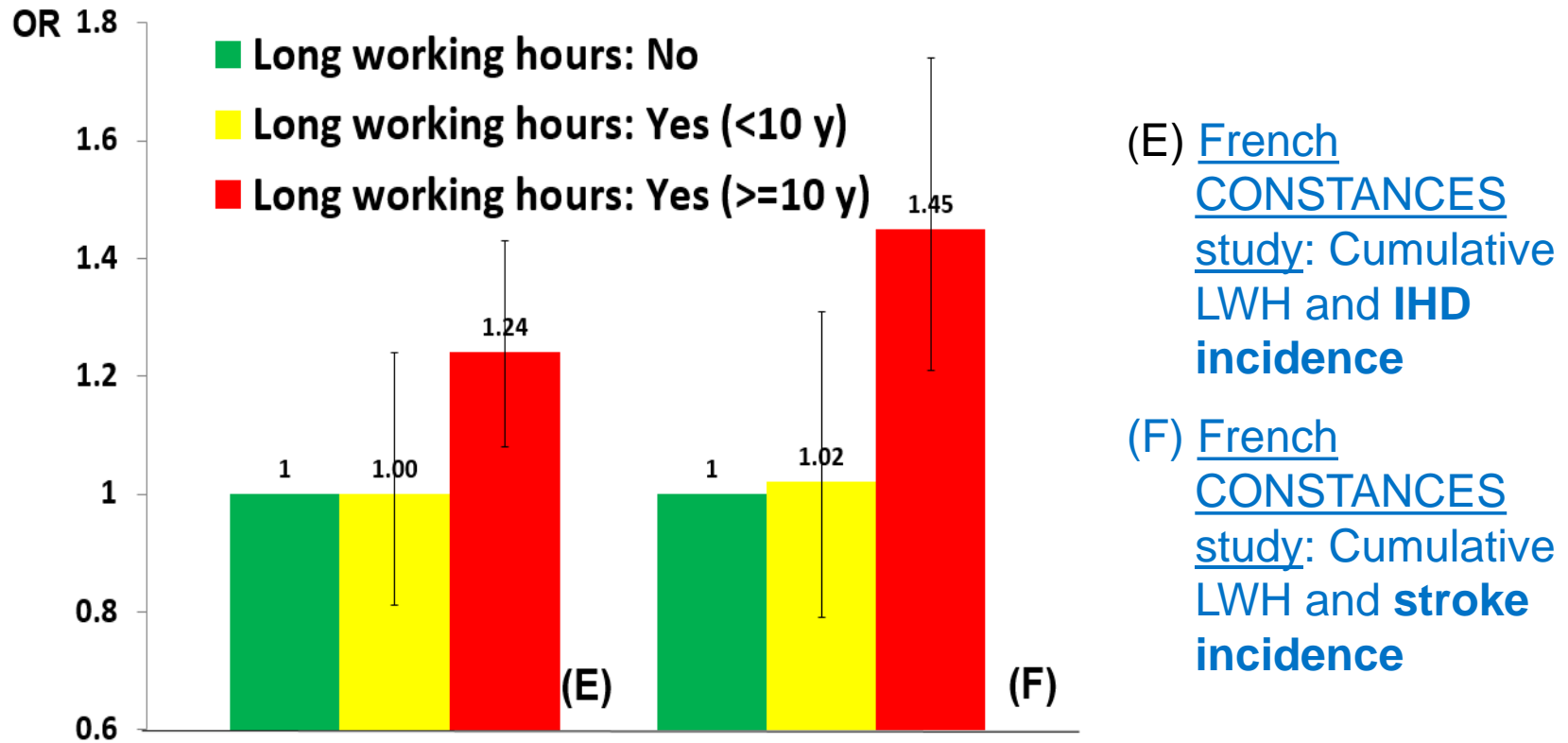
WHO-ILO: meta-analysis: LWH and risk of CHD/stroke



- (A) WHO/ILO review: LWH and **acquired IHD**
- (B) WHO/ILO review: LWH and **acquired stroke**;
- (C) WHO/ILO review: LWH and **dying from IHD**
- (D) WHO/ILO review: LWH and **dying from stroke**.

Source: Li J, et al. Environ Int, 2020, 142: 105739.; Descatha A, et al.. Environ Int, 2020, 142: 105746.

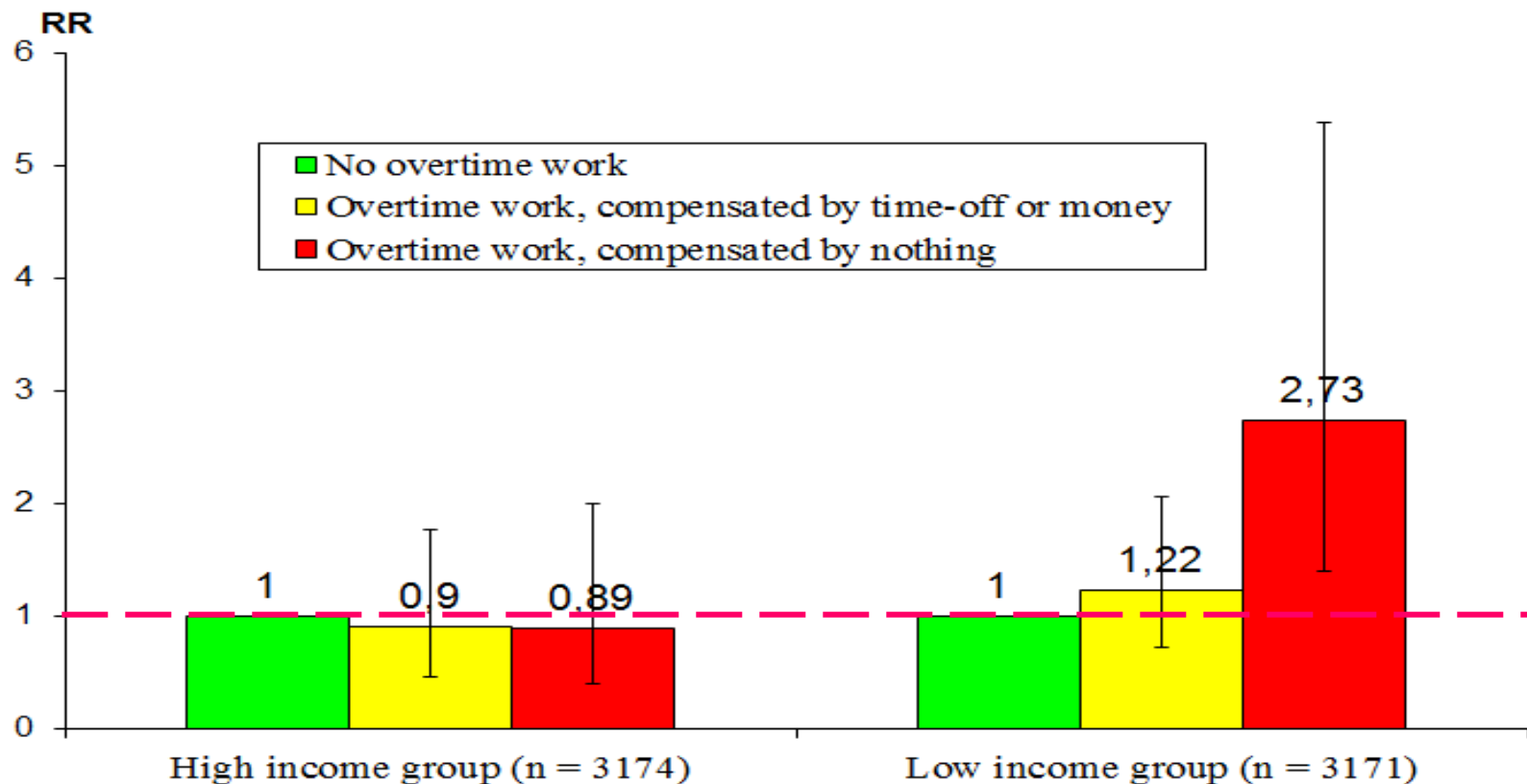
Duration of long working hours and CVD occurrence



Source: Fadel M, et al., J Am Heart Assoc, 2020, 9 (12): e015753.
Fadel M, et al. Stroke, 2019, 50 (7): 1879-1882.

Long working hours with/without compensation

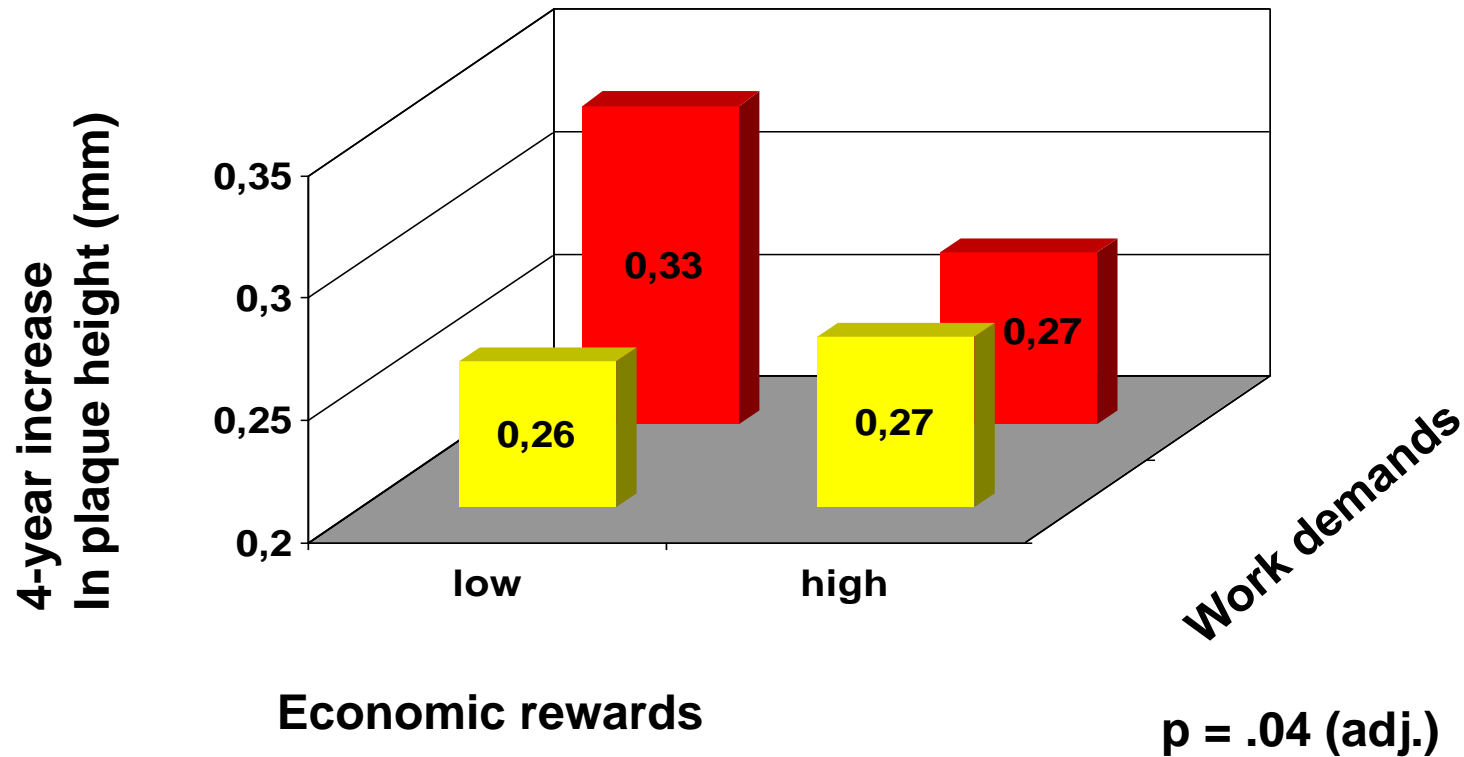
Risk of reported CHD , adjusted for main CVD risk factors
N = 3079 men and women; GSOEP Study; 2011 and 2013



Source: Li J, Siegrist J (2018) Am J Ind Med. 61(10):861-868

- Based on some **20 cohort studies**, exposure to work stress (DC, ERI, OJ) increases the **risk of CHD** by 30% to 60% (**RR 1.3-1.6**).
- These risks add to each other if working people are **simultaneously exposed** to these stressors (**RR: 2.0**).
- Given a **prevalence of work stress** (DC or ERI) of some **25 %** in working populations, a **moderate risk elevation** is relevant in terms of public health.
- **Theoretically**, by avoiding work stress, some **8% of all CHD events** in employed populations could be **prevented** (population-attributable fraction (PAF))

Prospective blue-collar study: Demand/reward and progression of carotid atherosclerosis (4-year period; N= 940 male Finnish workers)



Source: Lynch J et al. (1997), *Circulation*, 96: 302-307.

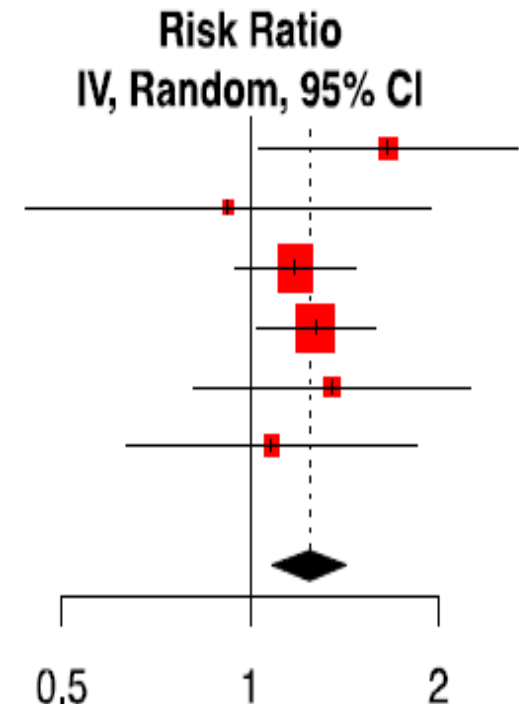
ERI and risk of Typ- 2 Diabetes: Mta-analysis of prospective studies

Study	log (RR)	SE	Weight	Risk Ratio	
				IV, Random	95% CI
Kumari 2004 (M)	0.51	0.2438	8.0%	1.66	[1.03, 2.68]
Kumari 2004 (F)	-0.08	0.3802	3.3%	0.92	[0.44, 1.94]
Mutambudzi 2018	0.17	0.1141	36.7%	1.18	[0.94, 1.48]
Nordentoft 2020	0.24	0.1116	38.4%	1.27	[1.02, 1.58]
Souza Santos 2020 (F)	0.30	0.2584	7.2%	1.35	[0.81, 2.24]
Souza Santos 2020 (M)	0.08	0.2734	6.4%	1.08	[0.63, 1.85]

Total (95% CI) 100.0% 1.24 [1.08, 1.42]

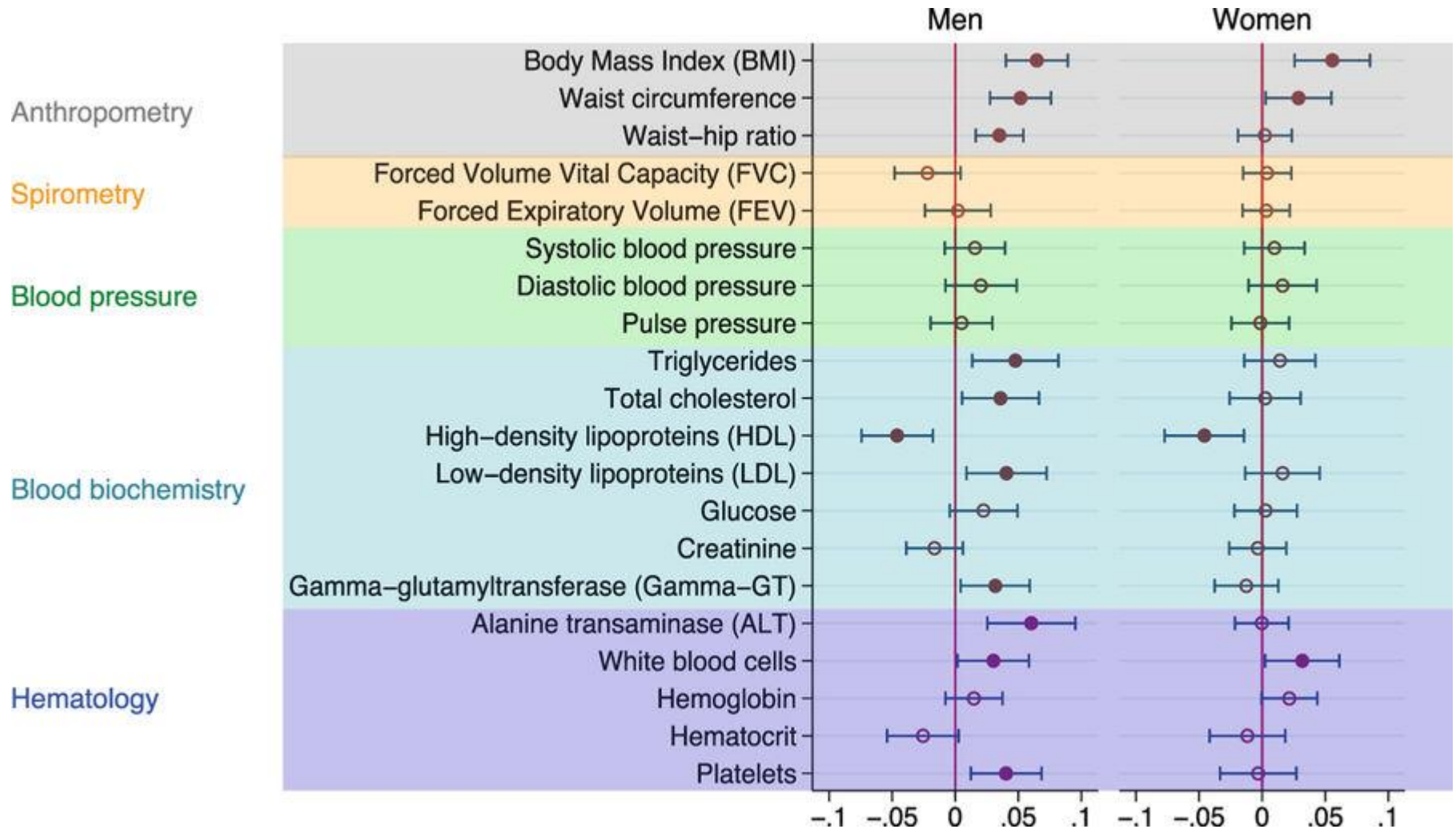
Heterogeneity: $\tau^2 = 0$; $\text{Chi}^2 = 2.65$, $\text{df} = 5$ ($P = 0.75$); $I^2 = 0\%$

Test for overall effect: $Z = 3.14$ ($P < 0.01$)



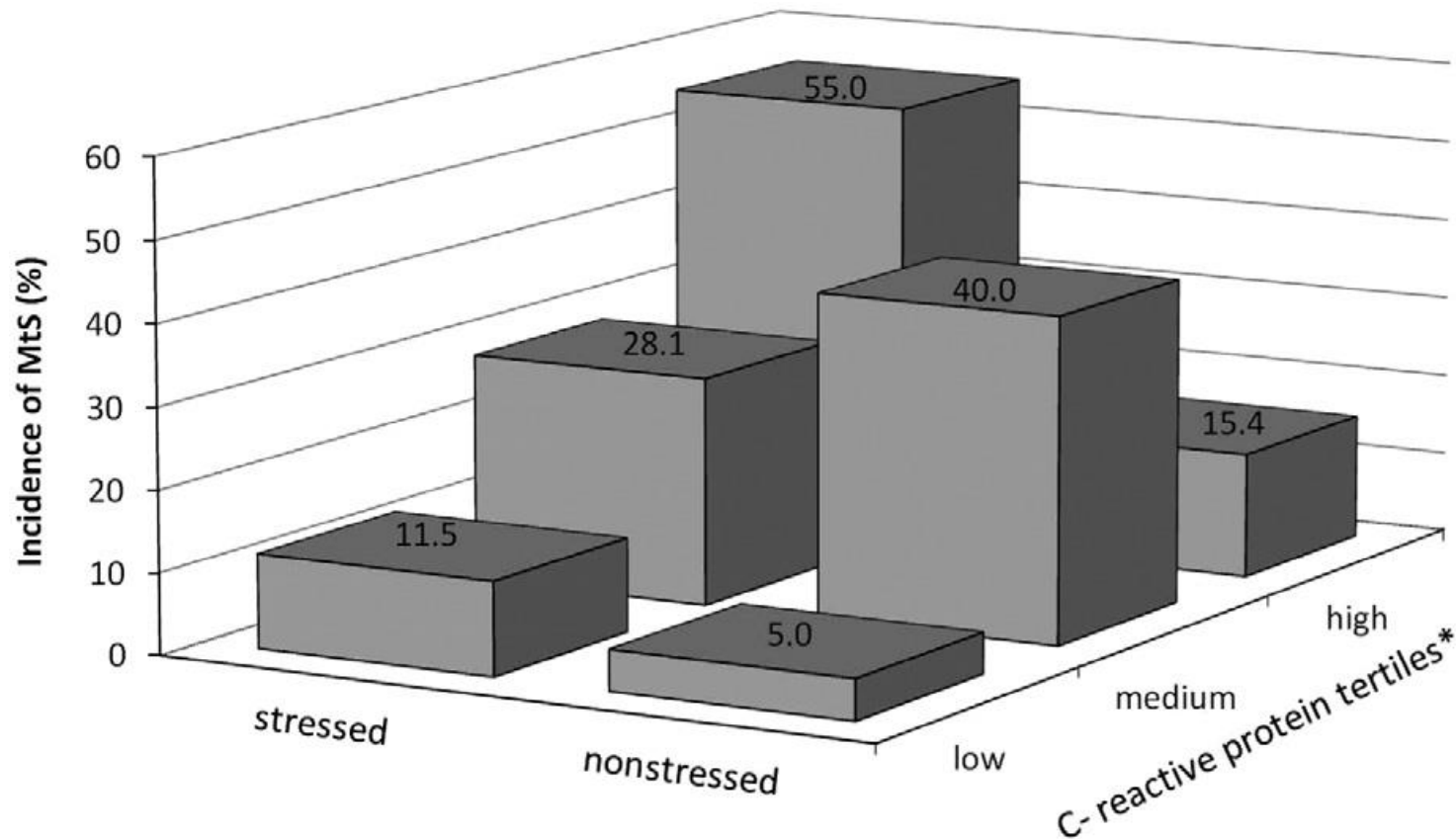
Risk ratios according to gender (OR and HR) transformed in Risk Ratios (RR); SE = Standard error

Work stress (ERI) and cardiometabolic risk factors (CONSTANCES Studie; N = 43.593 M. u. F.)



Source: Magnusson Hanson et al. (2017). Scientific Reports 7: 9282

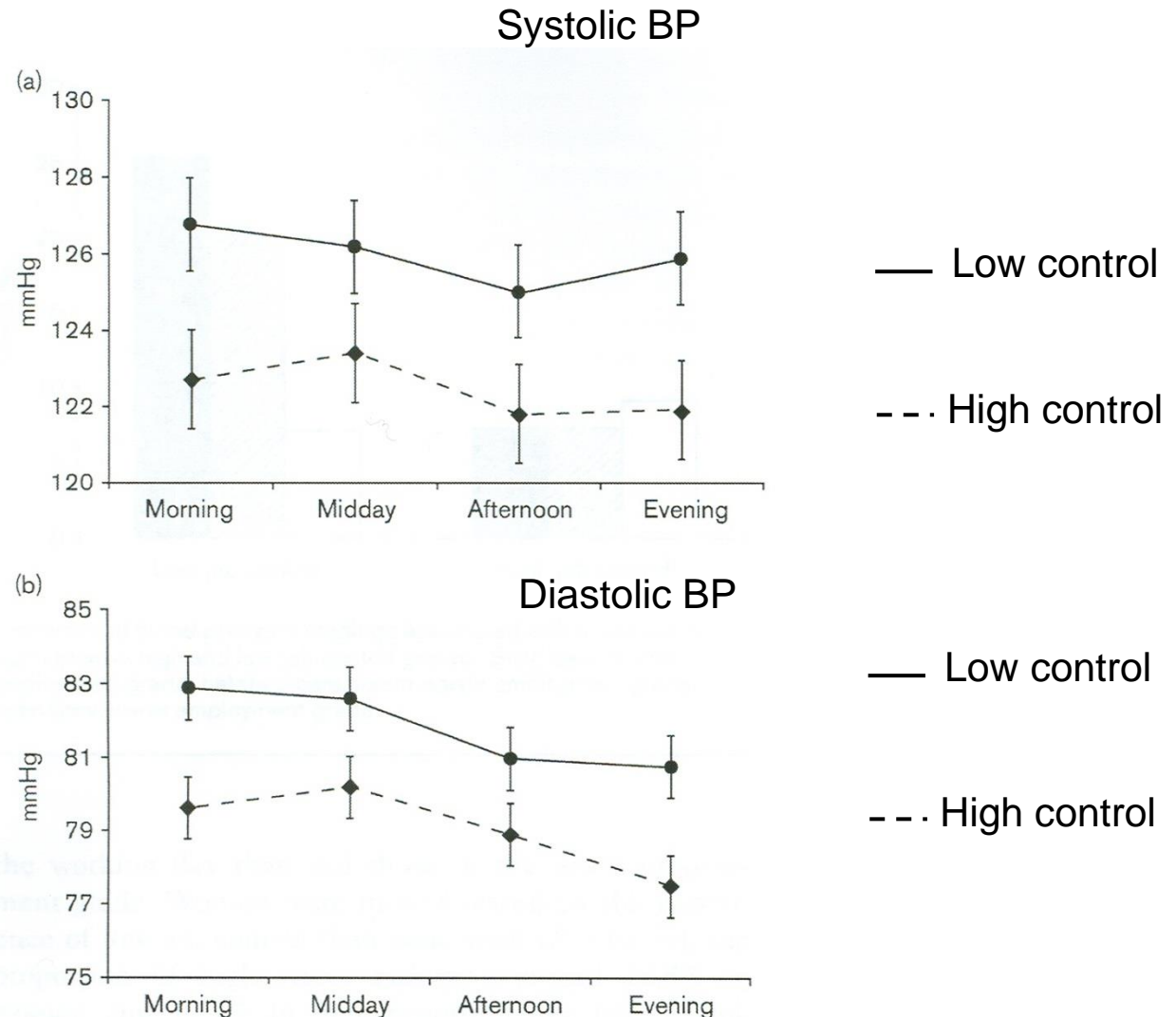
Prevalence of metabolic syndrome according to work stress (ERI) and C-reactive protein



(N=146 male employees, Jordan)

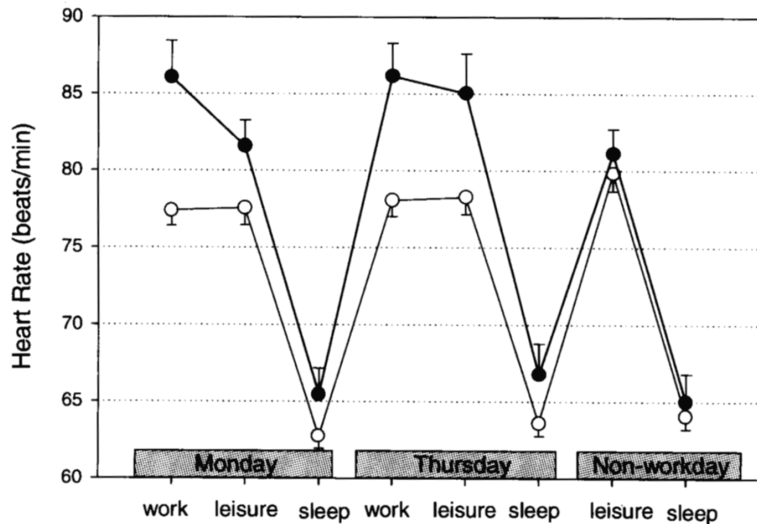
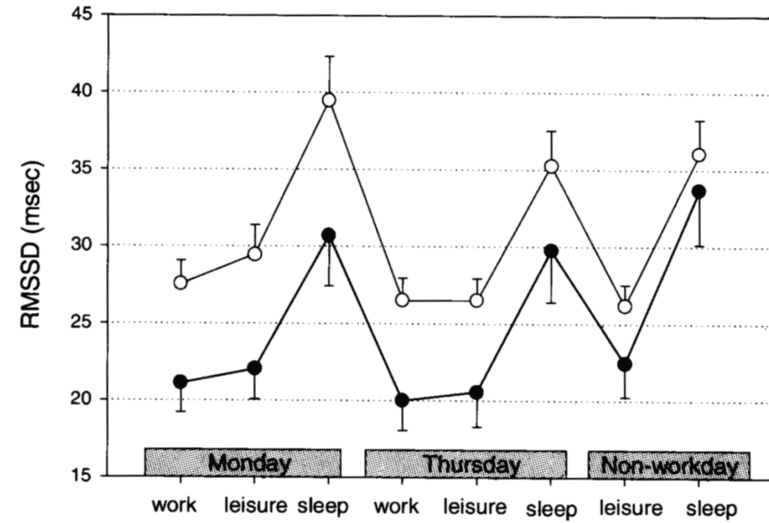
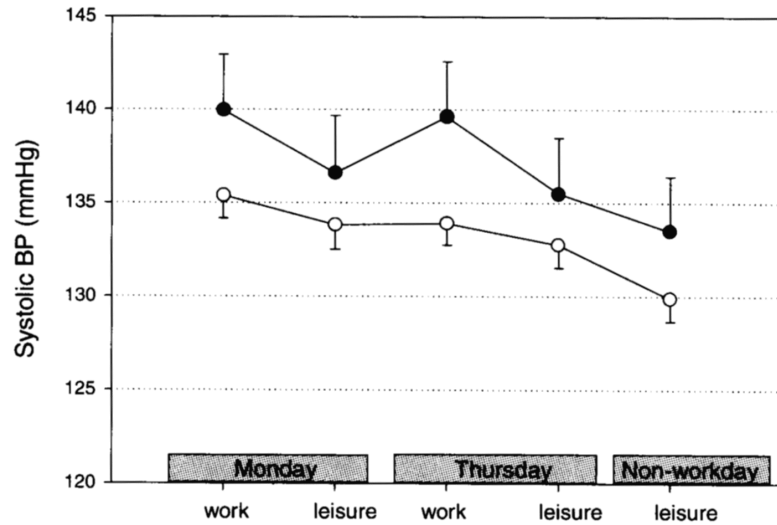
Biological pathways: Control at work and blood pressure

Mean ambulatory blood pressure (low control vs. high control).
N = 227 men and women (47-59 years); Whitehall Cohort Study



Source: Based on Steptoe A et al. (2004) *J Hypertension* 22(5): 915-920

Work stress (ERI) and elevated blood pressure and heart rate in computer workers (3 days)

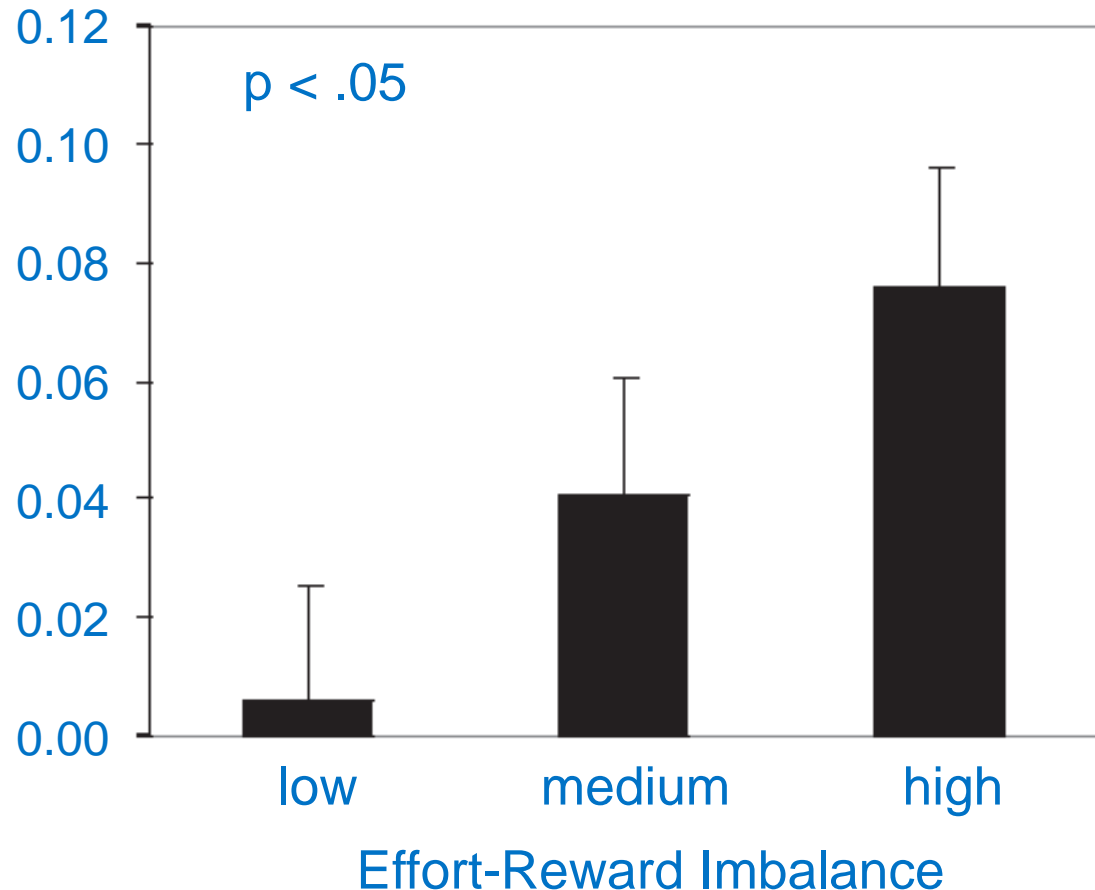


○ Low Imbalance
 ● High Imbalance

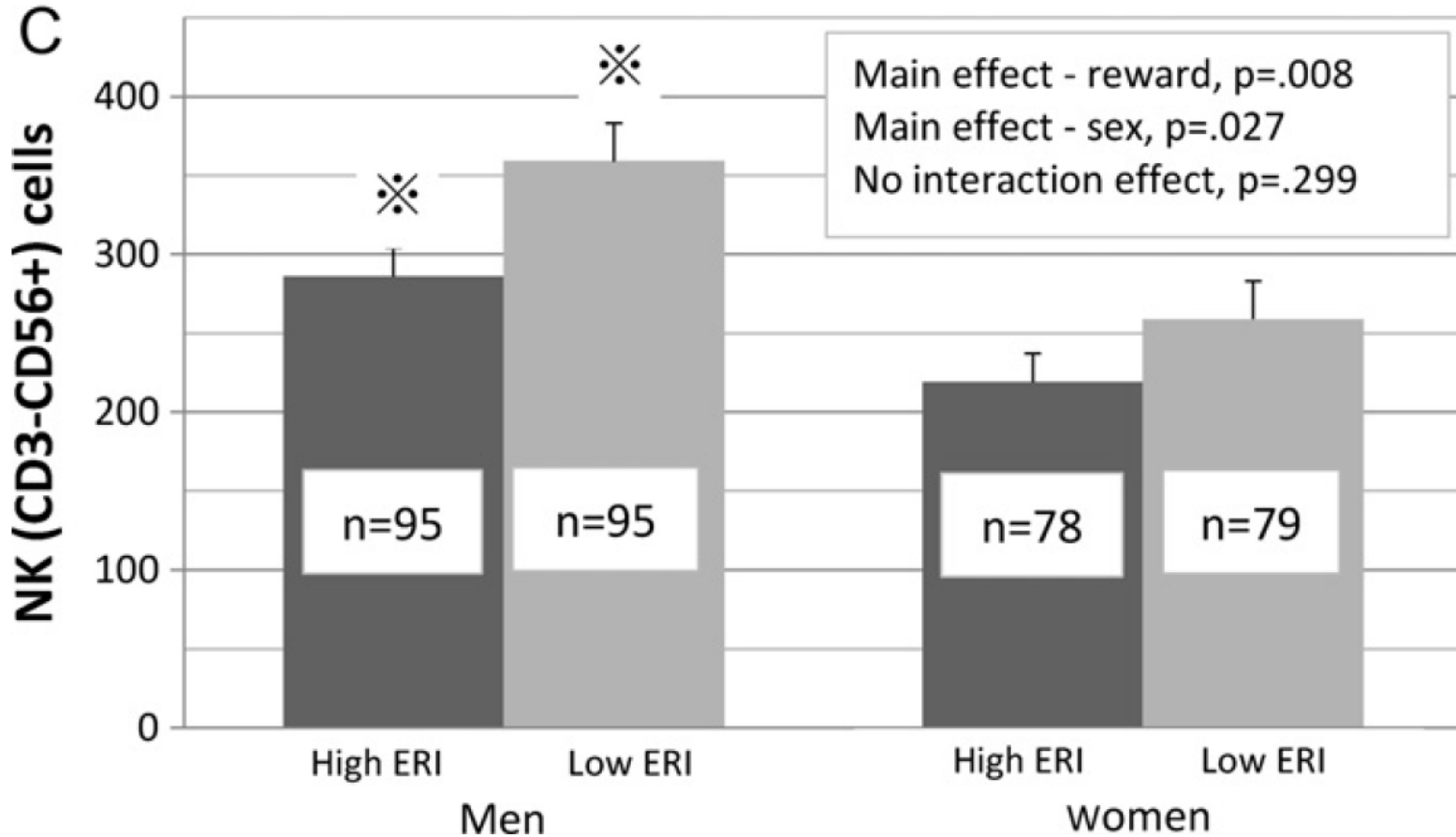
Inflammatory response (CRP) to experimental mental stress according to level of ERI (N=92)

CRP change*
($\mu\text{g/ml}$) as
function of effort-
reward imbalance

* adjusted for age, BMI,
baseline levels



Work stress (ERI) and natural killer cells



347 Japanese employees

Links between CHD and depression: Epidemiologic evidence

Patients with major depression: risk of future CVD:

Meta-analysis of 26 studies:

- incident acute myocardial infarction: OR= 1.28
- Incident stroke: OR= 1.13
- CVD mortality: OR= 1.44

Source: Krittanawong C et al. (2023) Am J Med 136, 881

Cardiac patients with post-AMI depression: risk of future CVD (compared to cardiac patients without depression):

Meta-analysis of 29 studies (follow-up: 24 months):

- Cardiac mortality: OR= 2.71
- Cardiac event: OR= 1.59
- All-cause mortality: OR= 2.25

Source: Meijer A et al. (2011) Gen Hosp Psychiatr. 33(3), 203

Brain reward system:

- Anterior cingulate, insula, nucleus accumbens, amygdala:
Areas involved in the processing of reward, aversion and fear

Chronic psychosocial stress can lead to

- Overactivation with longer-term reduced responsiveness
- Inhibition of activity: functional loss
- Lack of adaptation (e.g. continued firing in case of lack of reward)

Depression: Reduced dopaminergic (DA) responsiveness to reward
(nucleus accumbens; anhedonia) (Baik JH 2020)

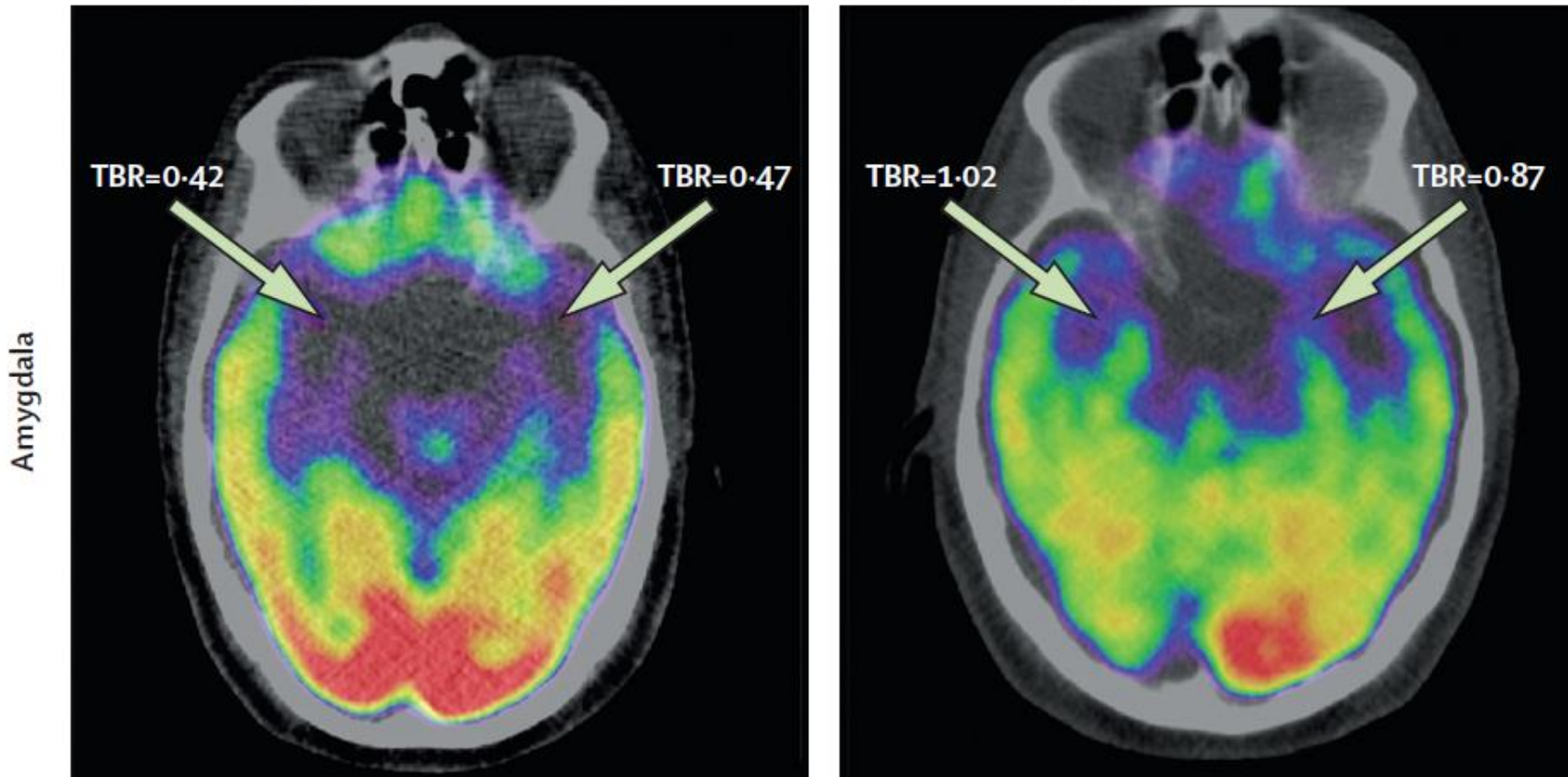
Depression and CHD: Pronounced response to fear (anterior cingulate
and ANS activation) (Bremner et al. 2019)

CHD: overactivation of amygdala (Tawakol et al. 2017);

Stress, activated limbic circuits (amygdala) and elevated CVD risk

Low amygdalar activity with no subsequent cardiovascular disease

High amygdalar activity with subsequent cardiovascular disease

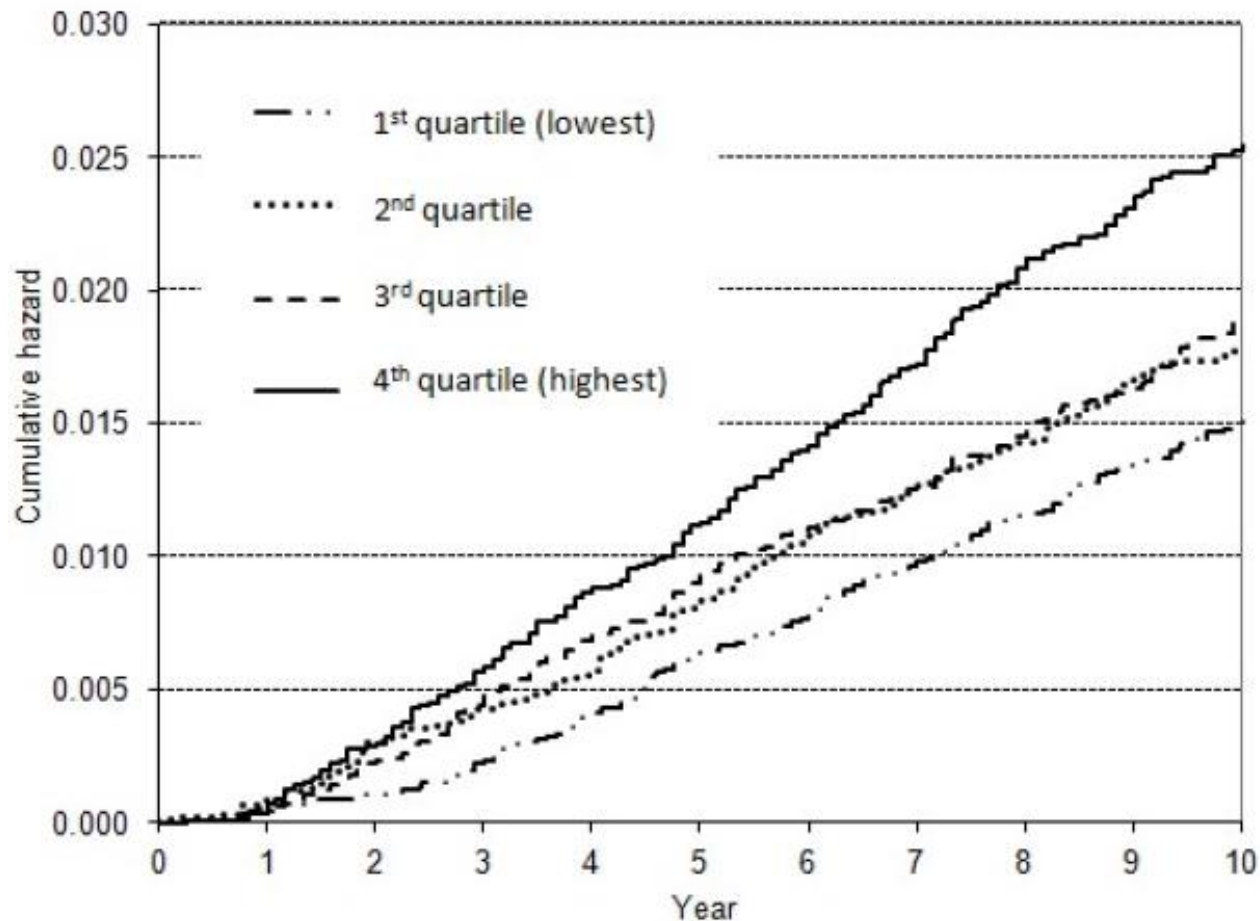


HR 1.59 (95% CI: 1.27 – 1.98) of CVD due to high activity, n = 293; 3.7 years

Source: Tawakol A et al. (2017) *Lancet* 389:834-45

Effort-reward imbalance at work and risk of disability pension due to depression

Cumulative hazard curves of disability pension due to depression by quartiles of work stress (ERI) (N = 51,874 male and female employees in Finland)



Source: Juvani A et al. (2014) *Scand J Work Environ Health* 40:266-77

- Based on some **30 cohort studies**, exposure to **work stress** (DC, ERI, OJ) increases the **risk of depression** by 70% to 100 % (RR 1.7-2.0).
- These risks add to each other if working people are **simultaneously exposed** to these stressors (RR:4.0).
- Epidemiologic evidence is supplemented by **naturalistic studies** on potential **psychobiological pathways**
- Given a **prevalence of work stress** of some **25%** in employed populations, these **risk elevations** are **significant** in terms of prevention.
- **Theoretically**, by avoiding work stress, some **25% of all depressive episodes** in employed populations could be **prevented** (population-attributable fraction (PAF))

Source: Niedhammer I et al. (2022) Int Arch Occup Environ Health
95(1) 233

2. Practical implications

Monitoring of work stress:

Regular monitoring activity:

- **Administrative data** analysis within and beyond companies (role of OSH professionals)
- **Survey** of employees within company (e.g. once/year)
 - apply a feasible, validated tool
 - analyse data on time, respect data protection
 - use results as basis of developing recommendations
- **Examples of tools:**
 - EWCS: European Working Conditions Survey
 - COPSQ: Copenhagen Psychosocial Questionnaire
 - JCQ: Job Content Questionnaire
 - ERI: Effort-Reward Imbalance Questionnaire

- Extend prescribed medical screenings to occupational high risk groups
- Participate in discussion and implementation of preventive measures at company level, derived from screening and monitoring data
- Apply evidence on successful return to work in secondary/tertiary prevention
 - Individual placement and support; early collaboration of company with medical treatment/rehabilitation; stepwise reintegration; psychotherapeutic support
- Develop protected consultation opportunity for stress and depression-related problems among employees (incl. early detection depression)
 - Liaison service with psychiatrist/ psychotherapist

Intervention measures at company level

- **Personal level:** Stress prevention programs; relaxation
- **Interpersonal level:** Leadership training; communication skills;
- **Structural level:** Organizational/personnel development (based on work stress models)
 - Job enrichment / enlargement (autonomy, control, responsibility)
 - Skill utilization / active learning/ cognitive stimulation
 - Participation, shared decisions, esp. work schedules
 - Culture of recognition, good leadership
 - Fair wages / gain-sharing
 - Continued qualification / promotion prospects
 - Reconciliation of work and family/private life

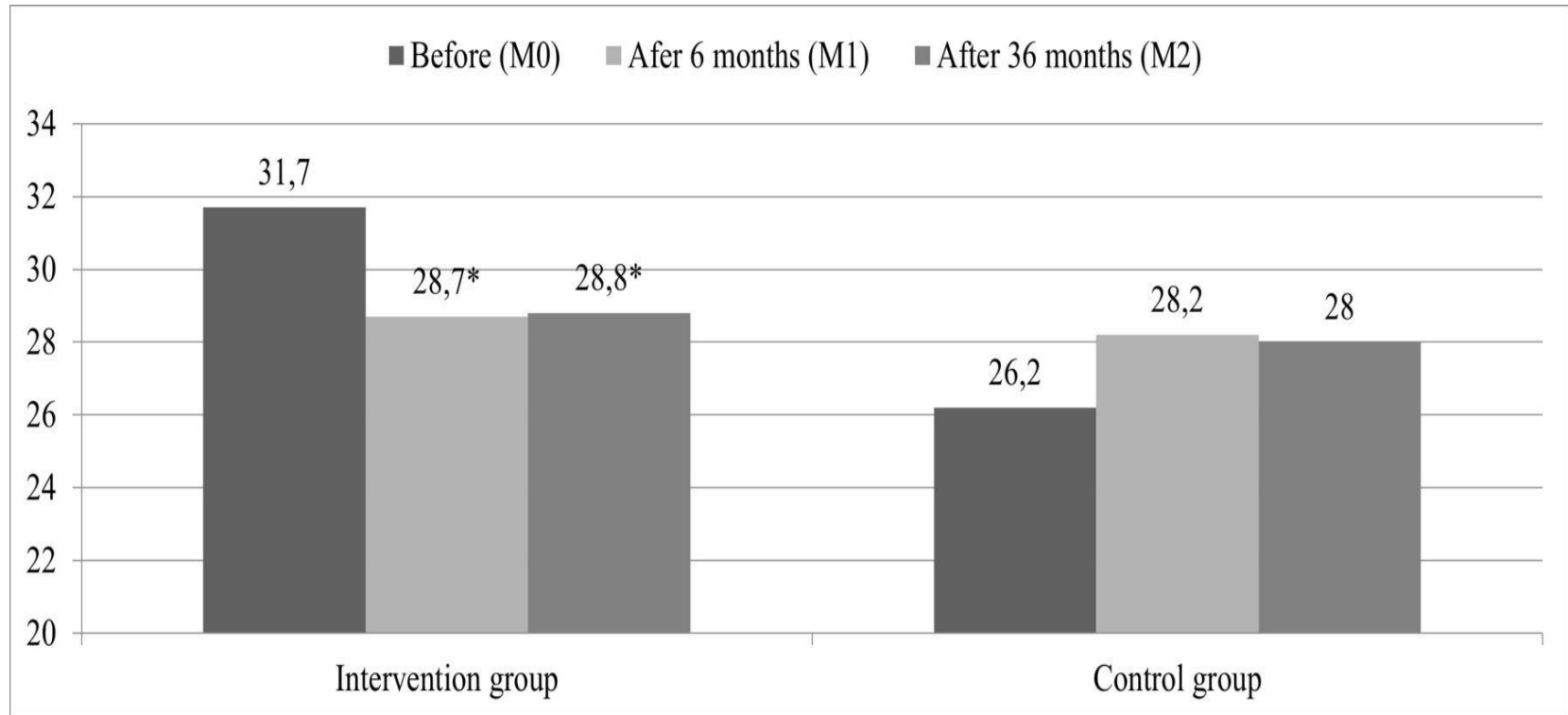
Improved mental health among hospital personnel

Mean values t2

Variable	Intervention- H	Controll -H	p
Demand	11.9	12.6	.008
Control	70.0	68.7	.051
Support	23.7	23.0	.011
Recognition/reward	31.2	30.2	.003
E-R ratio	1.0	1.1	.001
Burnout	43.2	48.3	.003

36 months-Follow-up (t2), Two Canadian hospitals (H), N=248 (Intervention) vs. 240 (Control) (ANCOVA, adj. for baseline values t1)

Reduced hypertension in an organizational intervention [N=1088 (intervention) and N=1068 (control)]



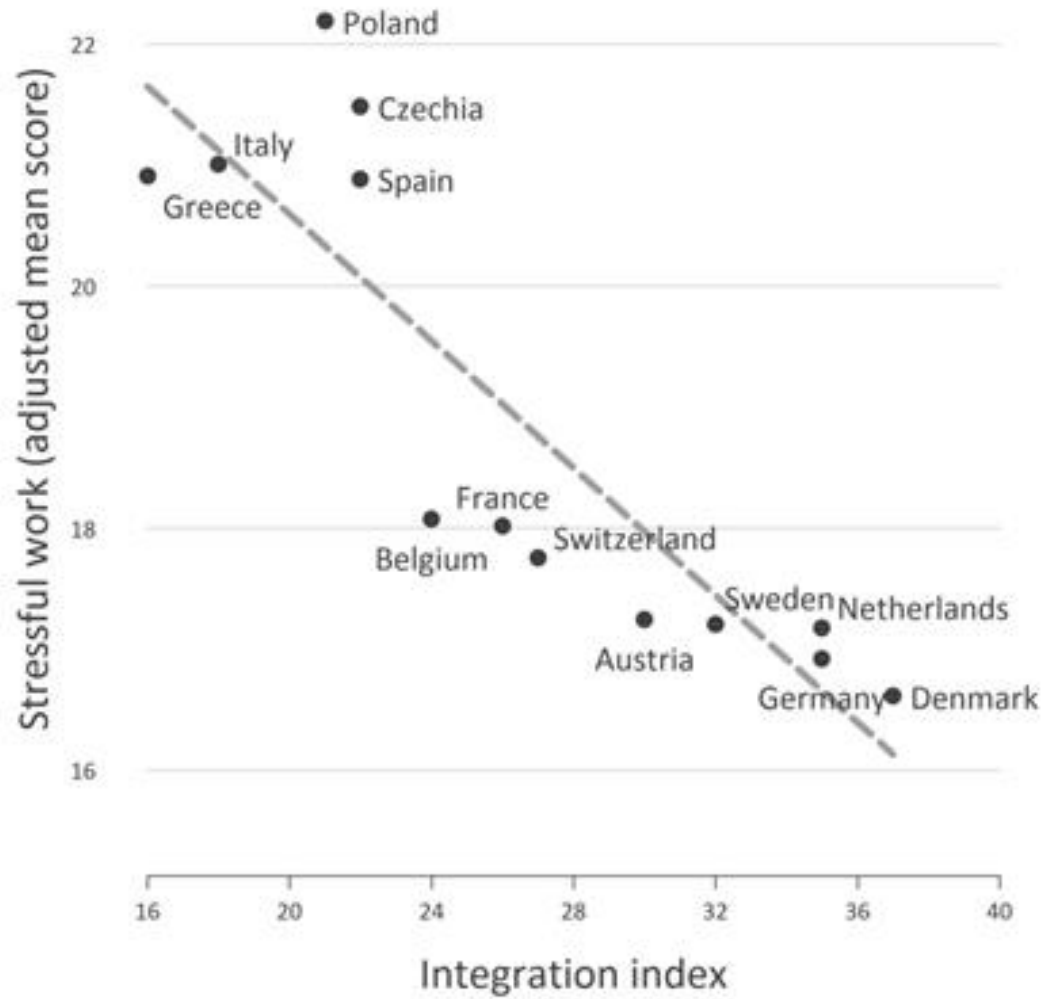
Organisational change based on job strain and effort-reward models;
Three times: baseline (M0); 6m (M1); 36m (M2)
Prevalence ratio IG vs. CG: 0.85 (0.74; 0.98)

Source: Trudel X et al. (2021) *Occup Environ Med* 78(10): 738-744.

Policy implications at the level of national labour and social policies

- **Strengthen distinct labor and social policies**
 - **ALMPs**: Integration policies, e.g. continued education/training; return to work; supported employment and rehabilitation services)
 - **PLMPs**: Protection policies, e.g. generosity and accessibility of benefit programs (compensation of income loss due to unemployment, disability, premature retirement)
- **Improve legislation on employment contracts**
 - Reduce **non-standard employment** and fixed-term contracts
 - Protect **precarious forms** of self-employment (gig economy)
- **Enforce fair work standards, including occupational health and safety measures**
 - **Injury prevention**, health hazards, shift work, long working hours
 - Healthy psychosocial work environments

Mean score of stressful work according to implementation of labour market integration policy



SHARE
N=11.181

Conclusions

- There is considerable new evidence on the impact of adverse psychosocial work environments on the incidence of CHD events and other stress-related disorders (esp. depression).
- Exposure definition is rooted in theoretical models (DC, ERI), is assessed by validated, standardized measurement tools, and is linked with CHD data through prospective cohort studies, thus offering good quality of evidence.
- A substantial gap exists between available knowledge and its application in professional practice and in occupational policies of prevention and health promotion.
- As there are successful models of good practice derived from this knowledge: „Do something, do more, do better“ (Sir Michael Marmot, 2012) !

Many thanks!

