Arbeitsbedingungen als Ursache kardialer Erkrankung

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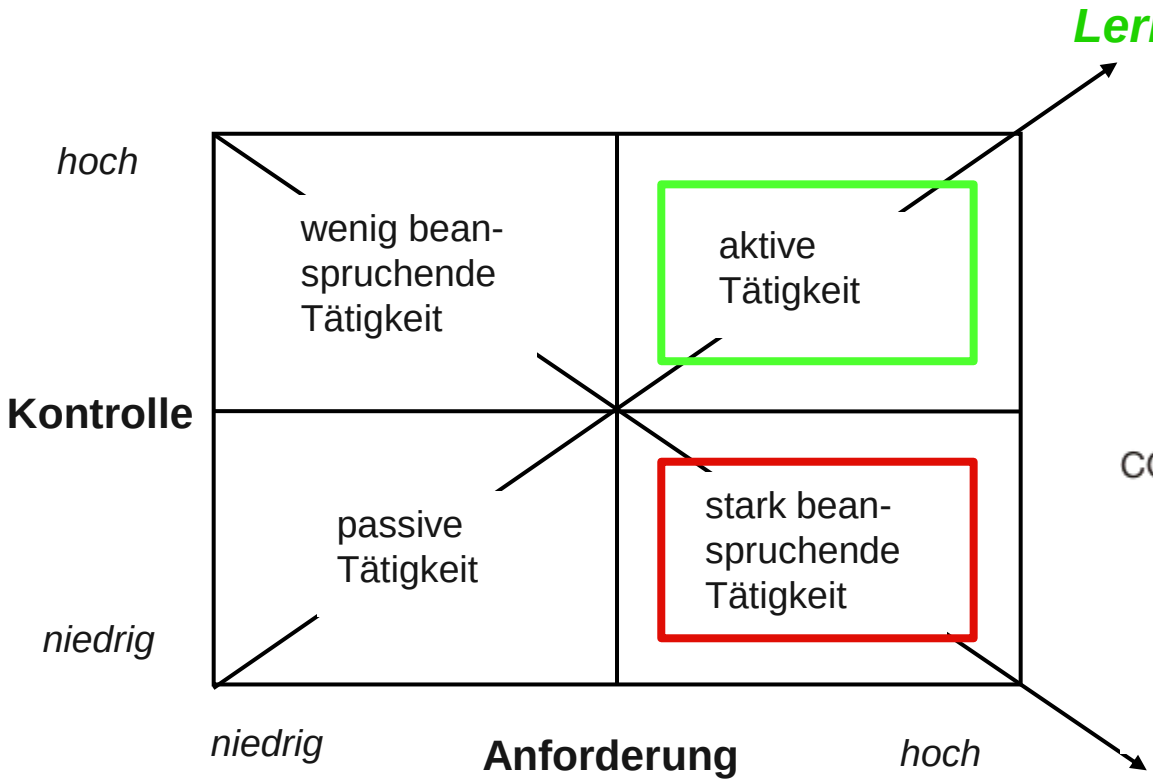
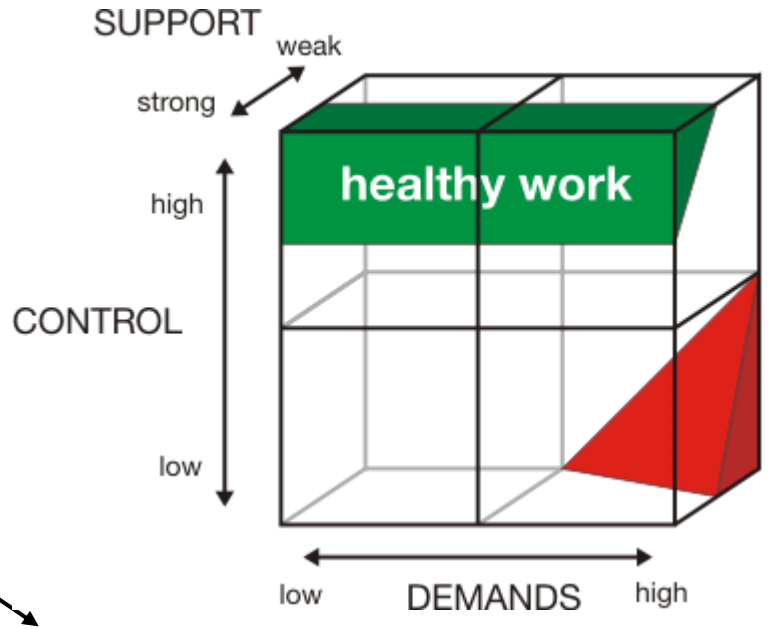
Anforderungs-Kontroll-Modell

(Demand-Control-(Support) Model)





(nach Karasek, 1979; Johnson & Hall, 1988)

[1]z.B. Kivimäki & Kawachi 2015, Theorell et al. 2016

Die Gratifikationskrise

(Effort-Reward Imbalance Model)

Anforderungen

- zu viel Arbeit
- Unterbrechungen
- Überstunden
- Zeitdruck

Belohnung

- Wertschätzung
- Arbeitsplatz-sicherheit
- Karriereaussichten

Verausgabungsneigung

→ Erkrankung[1]

(nach Siegrist, 1996)

[1]z.B. Theorell et al. 2016, Gilbert-Ouimet et al. 2014

Arbeitsunsicherheit

(Job Insecurity)

Unsicherheit in Bezug auf… [1]

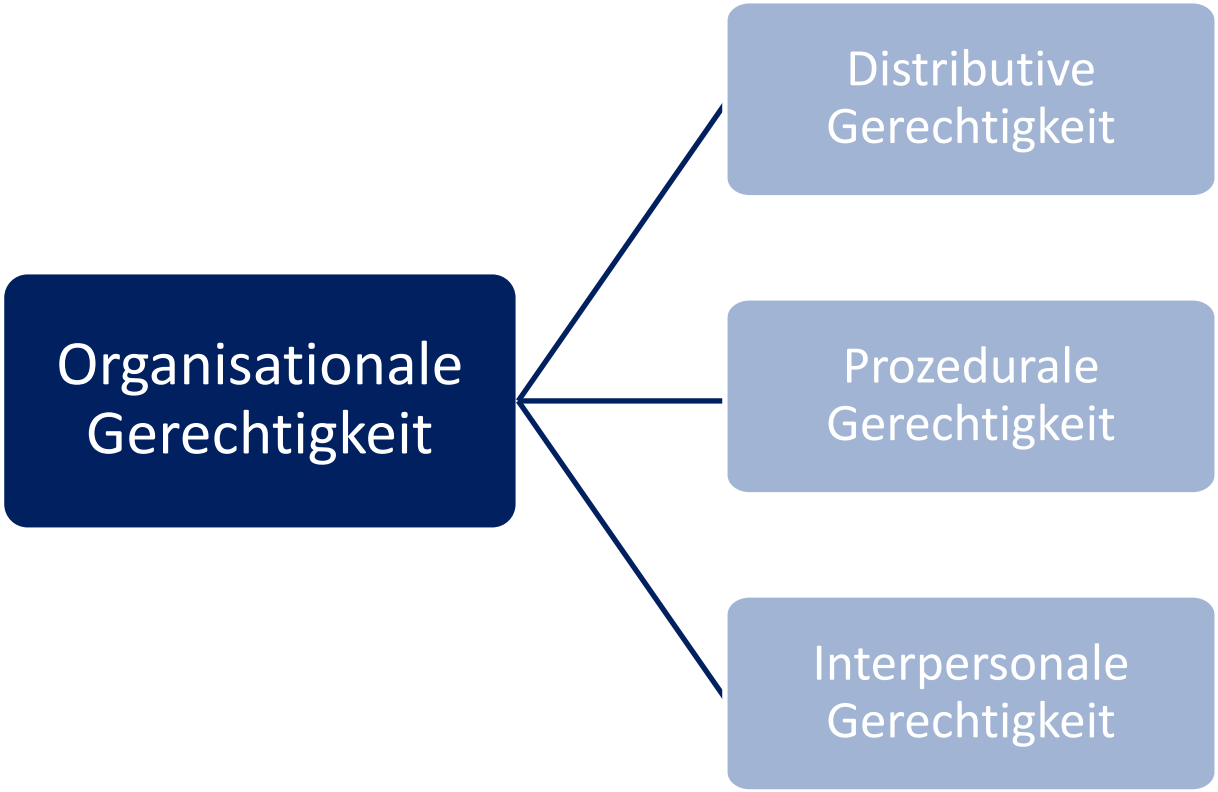
→ Erkrankung[3]

- **Subjektive** Einschätzung einer Situation [1]
- Antizipierte Veränderung ist **unfreiwillig** [2]
- Empfundene **Machtlosigkeit** [2]

[1] Hellgren et al. 1999; Sverke et al. 2002; De Witte 2005 [2] Greenhalgh & Rosenblatt 1984, 2010; De Witte 2005
[3] z.B. Sverke et al. 2002, Theorell et al. 2016

Gerechtigkeit in Organizationen

(Organizational Justice)



(nach Greenberg, 1990; Bies & Moag, 1986)

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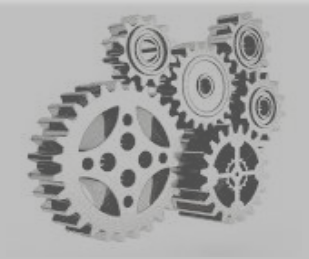
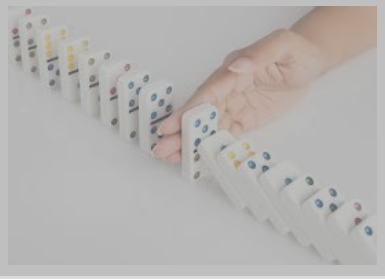
Psychosoziale Stressoren bei der Arbeit

Psychosoziale Stressoren und kardiale Erkrankungen



Wirkungsmechanismen



Präventive Ansätze

A systematic review of studies in the contributions of the work environment to ischaemic heart disease development

Töres Theorell[1], Katarina Jood[2], Lisbeth Slunga Järvholm[3], Eva Vingård[4], Joep Perk[5], Per Olov Östergren[6], Charlotte Hall[7]

Table 1 Degree of scientific evidence for different studied work environment factors

Work-related factor	Participants	Studies	Scientific evidence
Relationship between occupational environment and ischaemic heart disease			
Low decision latitude	804 086	25	⊕⊕⊕○
Job strain (low decision latitude, high job demands)	237 273	18	⊕⊕⊕○
Iso-strain (job strain + low job support)	24 645	2	⊕⊕○○
High pressure job	1 024 128	7	⊕⊕○○
Effort reward imbalance	29 917	5	⊕⊕○○
Low support at the work place	167 307	11	⊕⊕○○
Low work place justice	20 296	3	⊕⊕○○
Poor skill discretion	1 012 008	5	⊕⊕○○
Job insecurity	64 527	4	⊕⊕○○
Night work	34 413	3	⊕⊕○○
Long working week	1 013 046	7	⊕⊕○○
Noise	584 735	9	⊕⊕○○
The scientific evidence is insufficient (⊕○○○) to determine if there is a relationship between the following occupational factors and IHD			
Demands at work, Active job (high decision latitude, high job demands), Passive job (low decision latitude, low job demands), Poor social climate at the work place, Bullying at work, Conflicts at work, Shift work, Physically demanding work, Sitting work, Manual handling—lifts, Electromagnetic fields, Radiation (gamma- and ionizing radiation), Radon			

⊕⊕⊕○, There is scientific evidence for an association between exposure and outcome. The result is based on studies of high or moderate quality. The quality of evidence has been upgraded due to consistency of the data (control and job strain) or large magnitude of effect (bullying).

⊕⊕○○, There is scientific evidence for an association between exposure and outcome. The result is based on studies of high or moderate quality.

Theorell et al., 2016.

Curr Cardiol Rep (2015) 17: 74
DOI 10.1007/s11886-015-0630-8

Work Stress as a Risk Factor for Cardiovascular Disease

Mika Kivimäki[1] · Ichiro Kawachi[2]

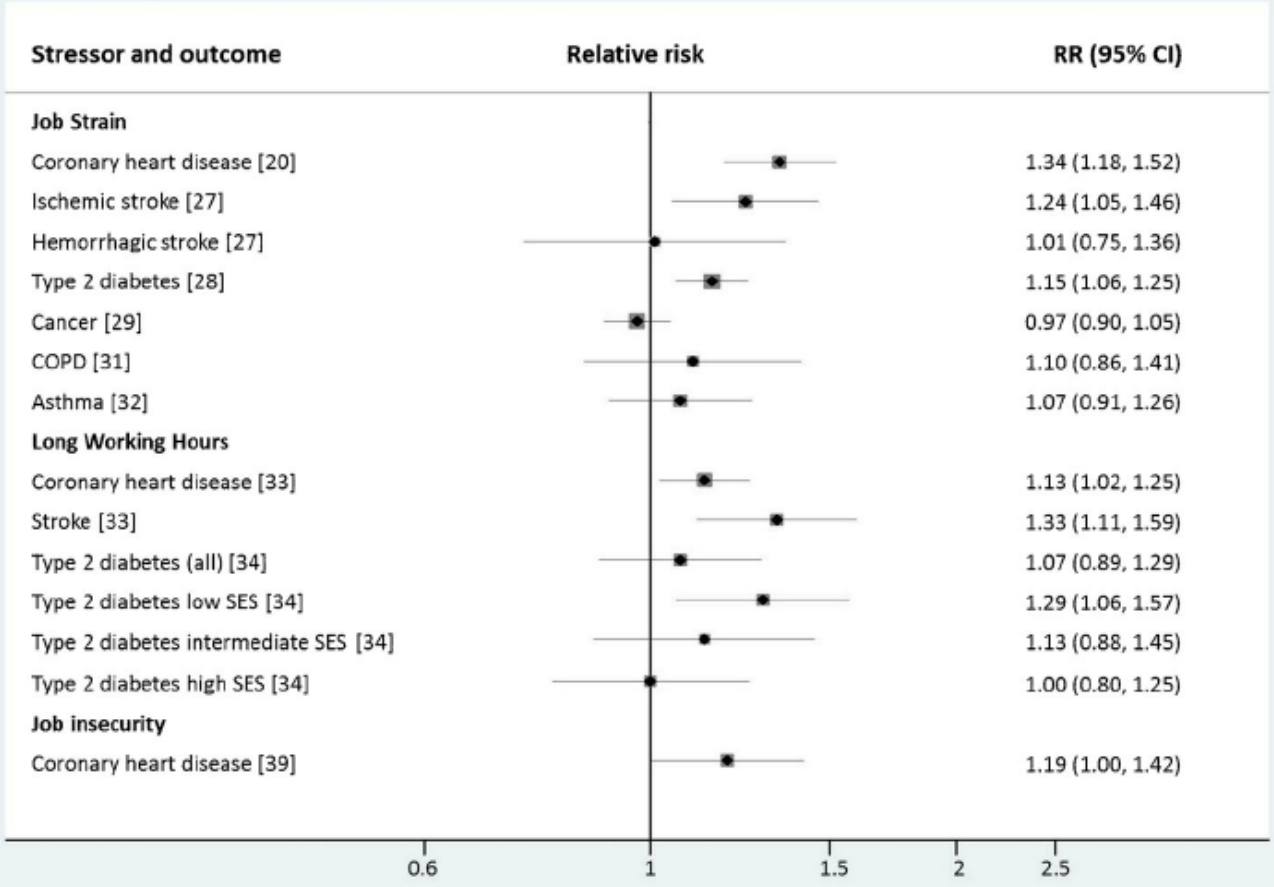
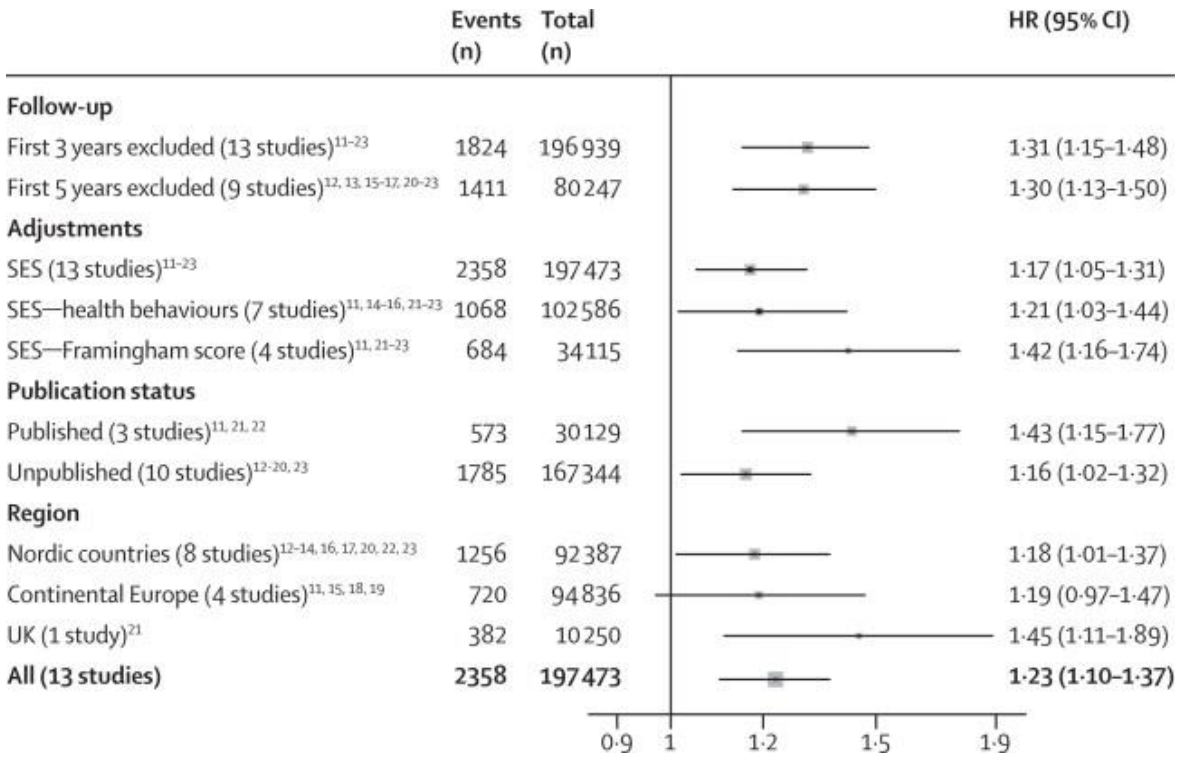


Fig. 1 Associations of work stressors with cardiovascular disease and other chronic conditions in recent meta-analyses of prospective cohort studies. Reference in *parenthesis*

Lancet 2012; 380: 1491–97

Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data

Mika Kivimäki, Solja T Nyberg, G David Batty, Eleonor I Fransson, Katriina Heikkilä, Lars Alfredsson, Jakob B Bjorner, Marianne Borritz, Hermann Burr, Annalisa Casini, Els Clays, Dirk De Bacquer, Nico Dragano, Jane E Ferrie, Goedele A Geuskens, Marcel Goldberg, Mark Hamer, Wendela E Hooftman, Irene L Houtman, Matti Joensuu, Markus Jokela, France Kittel, Anders Knutsson, Markku Koskenvuo, Aki Koskinen, Anne Kouvonen, Meena Kumari, Ida E H Madsen, Michael G Marmot, Martin L Nielsen, Maria Nordin, Tuula Oksanen, Jaana Pentti, Reiner Rugulies, Paula Salo, Johannes Siegrist, Archana Singh-Manoux, Sakari B Suominen, Ari Väänänen, Jussi Vahtera, Marianna Virtanen, Peter J M Westerholm, Hugo Westerlund, Marie Zins, Andrew Steptoe, Töres Theorell, for the IPD-Work Consortium



- 13 europäischen Kohortenstudien
- 197‘473 Teilnehmer (davon gaben 30’214 job strain an)
- 50% Frauen
- Ø follow-up zwische 7.5 Jahre
- Baseline: 1985-2006

Figure 2. Association of job strain with incident coronary heart disease in relation to study follow-up periods, adjustments, publication status for data, and geographical region. Estimates are adjusted for age and sex unless otherwise stated.

Lancet 2012; 380: 1491–97

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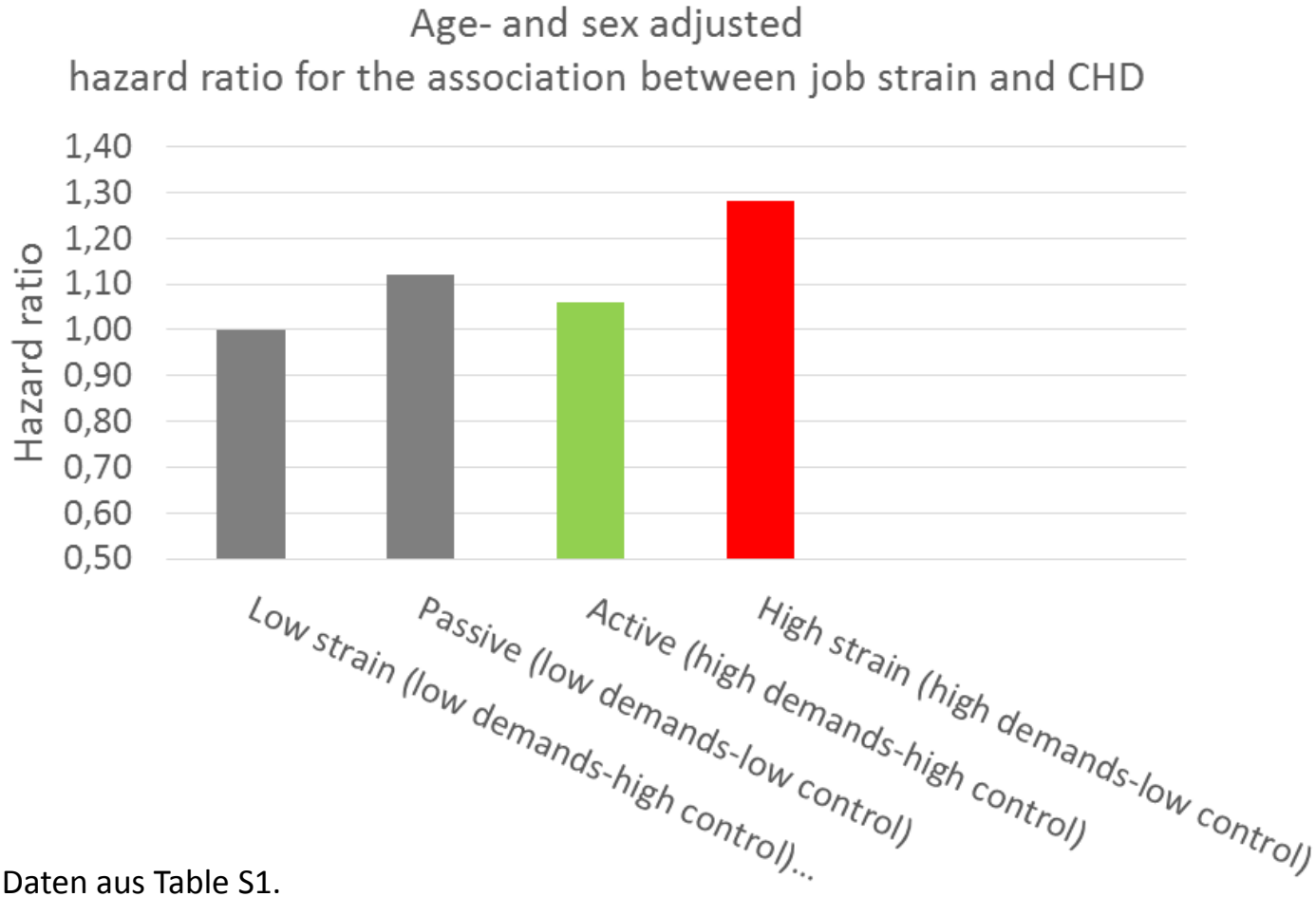

Basierend auf Daten aus Table S1.

Scand J Work Environ Health 2006;32(6, special issue):431–442

Work stress in the etiology of coronary heart disease—a meta-analysis

by Mika Kivimäki, PhD,[1,2] Marianna Virtanen, PhD,[2] Marko Elovainio, PhD,[3] Anne Kouvonen, PhD,[1] Ari Väänänen, PhD,[2] Jussi Vahtera, MD[2]

- 14 prospektive Kohortenstudien, 101‘788 Teilnehmer
- 48% Frauen
- Ø follow-up zwische 4 und 26 Jahren

Table 3. Relative risk (RR) of incident coronary heart disease or cardiovascular mortality and its summary estimate in observational cohort studies on the effort–reward imbalance model and the organizational injustice model. (95% CI = 95% confidence interval)

Stress model	Age- and gender-adjusted		Multiple adjusted [a]	
	RR	95% CI	RR	95% CI
Effort-reward imbalance				
Siegrist et al, 1990 (6) (men)	..	..	4.53	1.43–14.3
Bosma et al, 1998 (48) (men, women)	2.68	1.46–4.91	2.15	1.15–4.01
Kivimäki et al, 2002 (43) (men, women)	2.36	1.26–4.42	2.42	1.02–5.73
Kuper et al, 2002 (47) (men, women)	1.22	1.01–1.46	1.26	1.03–1.55
Combined summary estimate excluding Bosma et al, 1998 (48) [b]	1.58	0.84–2.97	2.05	0.97–4.32
Combined summary estimate excluding Kuper et al, 2002 (47) [b]	2.52	1.63–3.90	2.51	1.58–3.98
Organizational injustice				
Kivimäki et al, 2005 (8) (men)	1.54	1.10–2.13	1.41	1.01–1.96
Elovainio et al, in press (15) (men, women)	1.81	1.14–2.94	1.64	1.00–2.78
Combined summary estimate	1.62	1.24–2.13	1.47	1.12–1.95

[a] The covariates varied between studies. See table 1 for adjustments in each study.

[b] Bosma et al (48) and Kuper et al (47) are from the same cohort, the first having a more comprehensive assessment of effort–reward imbalance, but the latter having a more objective assessment of outcome. Thus two meta-analyses were conducted.

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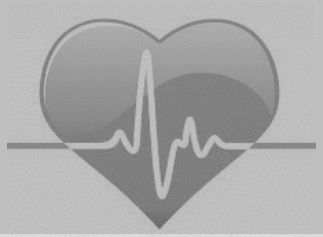
Psychosoziale Stressoren bei der Arbeit



Psychosoziale Stressoren und kardiale Erkrankungen

Wirkungsmechanismen

Präventive Ansätze

Wirkungsmechanismen

Psychosoziale Arbeitsbedingungen als Stressoren

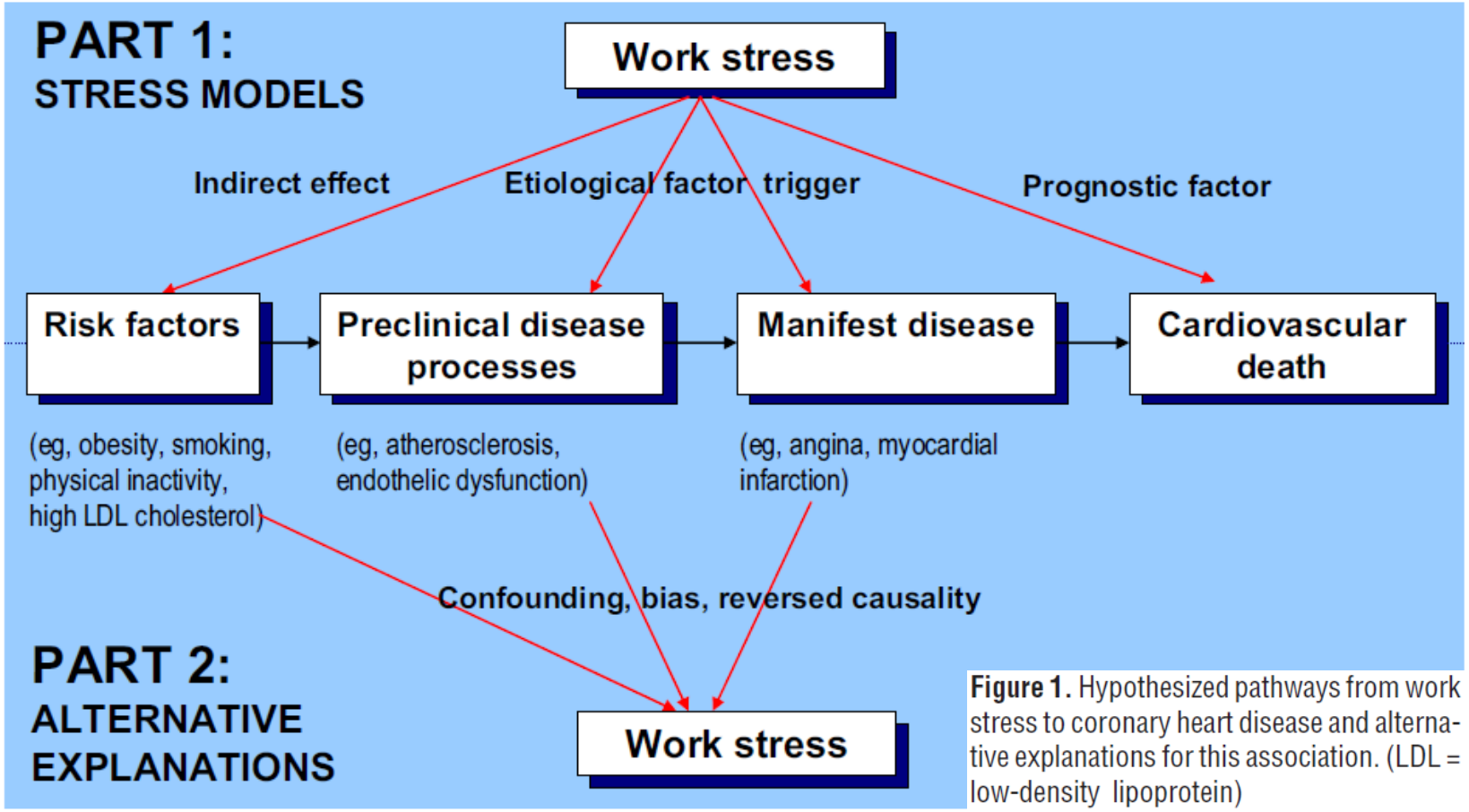


Figure 1. Hypothesized pathways from work stress to coronary heart disease and alternative explanations for this association. (LDL = low-density lipoprotein)

Quelle: Kivimäki et al., 2006, S. 432

Wirkungsmechanismen

Psychosoziale Arbeitsbedingungen als Stressoren

Chronic stress at work and the metabolic syndrome: prospective study

Tarani Chandola. Eric Brunner. Michael Marmot

Cite this article as: BMJ, doi:10.1136/bmj.38693.435301.80 (published 20 January 2006)

Table 3 Odds ratios (95% confidence intervals) of the metabolic syndrome. Multivariate multiple imputation logistic regression models: non-retired men and women in the Whitehall II cohort at phase 5

	Including patients who were obese at baseline			Excluding patients who were obese at baseline	
	No of cases/total	Adjusted for age+employment grade	Adjusted for age+employment grade+health behaviours	No of cases/total	Adjusted for age+employment grade+health behaviours
Men and women:					
No exposures	491/5178	1.00	1.00	388/4881	1.00
1 exposure	134/1253	1.13 (0.70 to 1.82)	1.12 (0.70 to 1.82)	103/1165	1.11 (0.60 to 2.03)
2 exposures	54/383	1.55 (0.85 to 2.85)	1.53 (0.87 to 2.69)	41/356	1.47 (0.74 to 2.92)
≥3 exposures	41/220	2.25 (1.31 to 3.85)	2.39 (1.36 to 4.21)	30/198	2.29 (1.27 to 4.12)
P for linear trend		<0.01	<0.00		0.01
Men:					
No exposures	341/3564	1.00	1.00	281/3407	1.00
1 exposure	95/900	1.11 (0.73 to 1.67)	1.11 (0.73 to 1.69)	77/851	1.12 (0.67 to 1.87)
2 exposures	37/252	1.64 (0.98 to 2.73)	1.57 (0.92 to 2.65)	31/238	1.56 (0.93 to 2.63)
≥3 exposures	32/181	2.01 (0.88 to 4.58)	2.17 (0.92 to 5.09)	24/166	2.04 (0.86 to 4.85)
P for linear trend		0.03	0.03		0.04
Women:					
No exposures	150/1614	1.00	1.00	107/1474	1.00
1 exposure	40/353	1.23 (0.40 to 3.74)	1.27 (0.42 to 3.84)	25/314	1.22 (0.28 to 5.37)
2 exposures	17/131	1.27 (0.34 to 4.83)	1.45 (0.45 to 4.75)	10/118	1.09 (0.15 to 7.94)
≥3 exposures	9/39	3.73 (0.88 to 15.75)	3.72 (0.79 to 17.53)	6/32	4.69 (0.79 to 27.86)
P for linear trend		0.23	0.11		0.26

Chandola et al., 2006

Wirkungsmechanismen

Psychosoziale Arbeitsbedingungen als Stressoren

Job Strain–Associated Inflammatory Burden and Long-Term Risk of Coronary Events: Findings from the MONICA/KORA Augsburg Case-Cohort Study

Rebecca T. Emeny, PhD, Astrid Zierer, BS, Maria Elena Lacruz, PhD, Jens Baumert, PhD, Christian Herder, PhD, Gabriele Gornitzka, Wolfgang Koenig, MD, Barbara Thorand, PhD, Karl-Heinz Ladwig, PhD, MD, for the KORA Investigators

TABLE 3. Multivariate Associations of Job Strain, Adjusted for Classical CHD Risk Factors, and each Inflammatory Biomarker, on CHD Outcome (n = 913 Noncases and n = 114 Cases)

	Biomarker, HR* (95% CI)	Job Strain, HR (95% CI)	Effect Change, ΔHR (SE)	Interaction Term, p Value
MCP-1, pg/mL	1.26 (1.03–1.54)	2.69 (1.12–6.42)	14.5 (2.1)	0.07
Log IL-8, pg/mL	1.12 (0.89–1.41)	2.57 (1.04–6.40)	9.4 (1.8)	0.04
Log IL-18, pg/mL	1.06 (0.87–1.28)	2.39 (1.01–5.65)	1.7 (0.8)	0.10
Log CRP, mg/L	1.27 (1.02–1.58)	2.12 (0.91–4.98)	−9.8 (1.8)	0.76
Log IL-6, pg/mL	1.38 (1.01–1.88)	2.30 (1.01–5.26)	−2.1 (0.9)	0.90
Log sICAM-1, ng/mL	1.18 (0.89–1.56)	2.22 (0.94–5.24)	−5.5 (1.4)	0.90
Log sE-selectin, ng/mL	1.02 (0.81–1.29)	2.33 (0.99–5.48)	−0.9 (0.6)	0.91
Log MIF, ng/mL	1.07 (0.87–1.32)	2.37 (1.02–5.52)	0.9 (0.6)	0.91
Log IP-10, pg/mL	1.17 (0.96–1.44)	2.42 (1.04–5.64)	3.0 (1.0)	0.39

CHD = coronary heart disease; HR = hazard ratio; CI = confidence interval; MCP-1 = monocyte chemotactic protein-1; IL = interleukin; CRP = high-sensitivity C-reactive protein; sE-selectin = soluble endothelial selectin; MIF = macrophage migration inhibitory factor; IP-10 = interferon-γ–inducible protein-10.
HR and 95% CI are presented in Cox analyses. Each model is adjusted for age, sex, survey, smoking status, actual hypertension, total cholesterol/high-density lipoprotein cholesterol, and body mass index. ΔHR = % change in HR from model 2 of Job Strain without any biomarker = ((Job Strain HR with biomarker − 2.35)/2.35) × 100. All standardized biomarker variables were continuous; therefore, the HR represents risk change per 1 standard deviation.

Emeny et al., 2013

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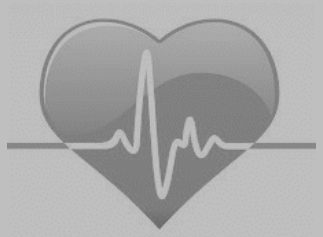
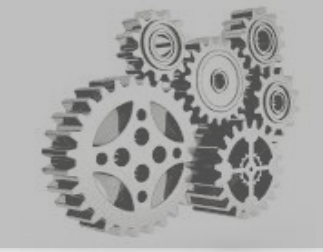
Psychosoziale Stressoren bei der Arbeit



Psychosoziale Stressoren und kardiale Erkrankungen



Wirkungsmechanismen

Präventive Ansätze

Präventive Ansätze

Prävention am Arbeitsplatz

- Sekundäre Interventionen (SMI) am Arbeitsplatz , unter Verwendung von Verhaltenstherapeutischen Elementen, zeigen größte Wirkung bei der Reduktion von erlebtem Stress und Verbesserung von

Original article

Stress management interventions in the workplace improve stress reactivity: a randomised controlled trial

Heribert Limm,[1] Harald Gündel,[2] Mechthild Heinmüller,[3] Birgitt Marten-Mittag,[1,3] Urs M Nater,[4] Johannes Siegrist,[5] Peter Angerer[3]

Limm et al., 2011

5%

son

Zusammenfassung

Psychosoziale Risikofaktoren bei der Arbeit

- Arbeitsbedingter Stress ist ein eigenständiger Risikifaktor bei der Entstehung von KHK (ORs zwischen 1.20-2.50), Gratifikationskrisen scheinen stärker mit KHK assoziiert zu sein als Job Strain (Anforderungs-Kontroll-Modell)
- Arbeitsunsicherheit und Ungerechtigkeit bei der Arbeit sind eigenständige Risikofaktoren

Potentielle Wirkungsmechanismen:

- Dysregulation des ANS und der HHNA-Achse durch anhaltende Aktivierung bei chronischem Arbeitsstress

Validierte Stresspräventionsprogramme am Arbeitsplatz

Weiterer Forschungsbedarf:

- Untersuchung weiterer Paradigmen von arbeitsbedingtem Stress
- Untersuchung möglicher biologischer Wirkungsmechanismen im Zusammenhang zwischen arbeitsbedingtem Stress und gesundheitlichen Folgen

VIELEN DANK FÜR IHRE AUFMERKSAMKEIT

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