

1 Article

2 Predicting Cardiovascular Disease from Psychosocial 3 Safety Climate: A Prospective Cohort Study from 4 Australia

5 Harry Becher¹, Maureen Dollard^{2,*}, Peter Smith³ and Jian Li⁴

6 ¹ Asia Pacific Centre for Work Health and Safety, University of South Australia, GPO Box 2471, Adelaide SA
7 5001

8 ² Asia Pacific Centre for Work Health and Safety, School of Psychology, Social Work and Social Policy,
9 University of South Australia, GPO Box 2471, Adelaide SA 5001, and School of Medicine, University of
10 Nottingham; maureen.dollard@unisa.edu.au

11 ³ Institute for Work & Health, Dalla Lana School of Public Health, University of Toronto & School of
12 Population Health and Preventive Medicine, Monash University; PSmith@iwh.on.ca

13 ⁴ Institute of Occupational, Social and Environmental Medicine, Centre for Health and Society, Faculty of
14 Medicine, University of Düsseldorf, Universitätsstraße 1, 40225 Düsseldorf, Germany;
15 Jian.Li@uni-duesseldorf.de

16 * Correspondence: maureen.dollard@unisa.edu.au; Tel.: +61 8 830 24846

17 **Abstract:** Cardiovascular Disease (CVD) is the most prevalent disease worldwide, which has been
18 linked to work stress because of poor job design as explained by the Job Demand-Control (JDC)
19 and the Effort-Reward Imbalance (ERI) models. In this paper we explore for the first time relative
20 impact of a specific aspect of organisational climate, Psychosocial Safety Climate (PSC), on any
21 CVD including angina, myocardial infarction, hypertension, and stroke. We used two waves of
22 interview data from Australia, with an average lag of 5 years (excluding baseline CVD, final n =
23 1223). Logistic regression was conducted to estimate the prospective associations between PSC at
24 baseline on incident CVD at follow-up. It was found that participants in low PSC environments
25 were 59% more likely to develop new CVD than those in high PSC environments. Logistic
26 regression showed that PSC at baseline predicts lower CVD risk at follow-up (OR = 0.98, 95% CI
27 0.96-1.00), and this risk remained unchanged even after joint adjustment for measures of ERI and
28 JDC. These results suggest that PSC is an independent risk factor for CVD in Australia. Beyond job
29 design this study implicates organisational climate and prevailing management values regarding
30 worker psychological health as the genesis of CVD.

31 **Keywords:** Cardiovascular Disease; Psychosocial Safety Climate; Demand-Control; Effort-Reward
32 Imbalance; Psychosocial Risks
33

34 1. Introduction

35 Cardiovascular Disease (CVD), a group of disorders of the heart and blood vessels that manifest
36 in acute events such as heart attack and stroke (as defined by the World Health Organisation, 2017),
37 is the greatest health risk in the world (Lozano et al., 2013). Notably, CVD causes more deaths than
38 any other single cause accounting for approximating 30% of deaths annually worldwide (Schnall,
39 Dobson, & Landsbergis, 2016). Among working age populations 10%-20% of all causes of CVD
40 deaths are work related (Tsutsumi, 2015). Despite some major improvements in cardiovascular
41 health via public health interventions, CVD continues to grow as a global pandemic. This
42 widespread health impact has a correspondingly large impact on workplace productivity; CVD has
43 been classified as the single greatest cause of workplace productivity loss in the world (Perk et al.,
44 2012; Piepoli et al., 2016). In addition to commonly-known CVD risk factors such as smoking and
45 obesity (Perk et al., 2012), work-related psychosocial risk factors and job stress have been established

46 as predictors of CVD (Chandola et al., 2008; Fishta & Backé, 2015; Li, Dollard, Loerbroks, & Angerer,
47 2015; Piepoli et al., 2016; Schnall, Dobson, & Landsbergis, 2016; Steptoe & Kivimäki, 2012).

48 Most CVD studies have focused on job design frameworks such as the Job Demand-Control
49 (Karasek, 1979), or the Effort-Reward Imbalance model (Siegrist, 1996) to explain work stress related
50 CVD. These models have focused on proximal work-related psychosocial risk factors (i.e., job design
51 characteristics that are harmful to health), yet the root cause may be more contextual and relate to
52 features of the organisational climate that potentially shape these harmful job characteristics.
53 Focusing on the “causes of the causes” has been identified as a key focus for future research (Schnall,
54 Dobson, & Landbergis, 2016). In this research we contextualise CVD as a work related health
55 problem that not only relates to job design, but may be predicted by organisational factors further
56 upstream. We use Psychosocial Safety Climate (PCS, i.e., the organisational climate for worker
57 psychological health) theory to frame the study. Under PSC theory working conditions are
58 determined by the prevailing management values concerning worker psychological health. To date
59 there is much evidence linking PSC to work conditions and health outcomes (Bailey, Dollard,
60 McLinton, & Richards, 2015; Bailey, Dollard, & Richards, 2015; Dollard & Bakker, 2010; Law,
61 Dollard, Tuckey, & Dormann, 2011) but no studies have explored the link between this specific
62 aspect of organisational climate and CVD.

63 The aim of this study is to determine whether PSC is a predictor of future employee CVD, and
64 which theory (PSC, Effort-Reward Imbalance and Job Demand-Control) provides a better account of
65 risk of future CVD.

66 1.1. Work Stress Theories and CVD

67 Job stress in this paper refers to adverse health reactions to taxing work conditions. To
68 understand how work-related psychosocial factors influence the risk of CVD, studies have typically
69 used well-evidenced work stress models. Effort-Reward Imbalance theory posits the primary cause
70 of job stress and related health effects is an imbalance between excessive efforts and insufficient
71 rewards (Siegrist, 1996; Van Vegchel, De Jonge, Bosma, & Schaufeli, 2005). In the Effort-Reward
72 Imbalance theoretical framework, ‘efforts’ are work-related demands that an employer requires of
73 their employees (e.g., work tasks, responsibilities), and ‘rewards’ are the benefits that employers
74 bestow upon their employees (e.g., money, job security, esteem). The Job Demand-Control theory
75 (Karasek, 1979) posits that the health of workers is determined by the level of job demands they
76 experience, in combination with levels of control, such as decision authority and skill discretion.
77 Under Job Demand-Control theory job strain refers to those jobs that combine high levels of
78 demands with low levels of control and give rise to adverse health consequences.

79 The potential increased risk of CVD associated with job stress has been examined using the
80 Effort-Reward Imbalance and Job Demand-Control models across a range of studies and
81 populations (Piepoli et al., 2016). While the Effort-Reward Imbalance model does not explicitly link
82 high effort or low rewards with adverse long term effects on cardiovascular health, an increased risk
83 of CVD is consistent with the core assumptions of the Effort-Reward Imbalance model (Kivimäki &
84 Siegrist, 2016). Given that an imbalance of efforts and rewards is associated with increased job stress
85 (de Jonge, Bosma, Peter, & Siegrist, 2000), and job stress is associated with CVD (Dimsdale, 2008), it
86 is logical that effort-reward imbalance would be indicative of increased risk of CVD. A similar logic
87 can be applied to Job Demand-Control theory; high demands and low control lead to work stress
88 and stress is associated with CVD.

89 Evidence shows that effort-reward imbalance is linked to CVD. An 11-year longitudinal
90 analysis of the Whitehall II data revealed that those in high effort-reward imbalance jobs are 26%
91 more likely to develop coronary heart disease than their peers (Kuper, Singh-Manoux, Siegrist, &
92 Marmot, 2002). A 24-year longitudinal analysis of Finnish workers, revealed that workers with high
93 effort-reward imbalance were 140% more likely to develop CVD than their peers (Kivimäki et al.,
94 2002). Effort-reward imbalance has been recently confirmed as an important increased risk factor for
95 CVD, using large pooled data from 11 European cohort studies (RR = 1.16; Daganano et al., 2016), over
96 and above established risks such as long working hours (Relative Risk [RR] = 1.39; Virtanen et al.,

97 2012), and job insecurity (RR = 2.00; Vahtera et al., 2004). The overall burden of this increased risk is
98 considerable.

99 There is also a strong literature linking Job Demand-Control job strain (i.e., high job demands
100 and low control) to CVD, across major demographics and over time (Kivimäki, Batty, Ferrie, &
101 Kawachi, 2014; Kivimäki et al., 2012; Schnall, Dobson, & Landsbergis, 2016) to non-fatal myocardial
102 infarction and death from CVD, where, after adjustment for sex and age, the hazard ratio for job
103 strain versus no job strain was 1.23, with the effect higher in published (HR = 1.43) than unpublished
104 (HR = 1.16) studies (Kivimäki et al., 2012). Job strain is also associated with an increased risk of
105 ischemic stroke, (Fransson et al., 2015), and research has found support for both job strain and
106 effort-reward imbalance as independent risk factors for stroke (Jood, Karlsson, et al., 2017).

107 Some of the literature examining the relationship between psychosocial work conditions and
108 CVD has focused on how prolonged stressful work conditions may induce CVD such as
109 hypertension (Ming et al., 2004; Vrijkotte, Van Doornen, & De Geus, 2000). Workers experiencing
110 chronic work stress have increased blood pressure (Schnall et al., 1998), even when they are not at
111 work (Vrijkotte et al., 2000). The mechanism for this effect is thought to be a combination of
112 hyper-reactivity of the sympathetic nervous system, along with reduced vagal tone – a symptom of
113 reduced activity of the parasympathetic nervous system (Vrijkotte et al., 2000). A systematic review
114 found support for the effects of both Job Demand-Control and Effort-Reward Imbalance Models on
115 blood pressure level and hypertension in approximately half of the studies reviewed
116 (Gilbert-Ouimet, Trudel, Brisson, Milot, & Vézina, 2014).

117 While the Effort-Reward Imbalance and Job Demand-Control models are well-adapted to
118 identifying the sources and levels of job stress and the creation of task-related interventions
119 (Bourbonnais, Brisson, Vinet, Vezina, & Lower, 2006; Li et al., 2017), they are less well suited to
120 identifying the organisational-level characteristics which precede effort-reward imbalance and Job
121 Demand-Control and its associated health outcomes (Owen, Bailey, & Dollard, 2016). An important
122 goal of organisational psychosocial interventions is to create sustainable change beyond the short
123 term reduction of job stress, so that hazardous psychosocial work conditions are modified so that
124 associated job stress does not return upon cessation of the intervention (Swerissen & Crisp, 2004).
125 The limitation of focusing on Effort-Reward Imbalance and Job Demand-Control models is that they
126 do not address a potential origin of the problem, Psychosocial Safety Climate.

127 1.2. Psychosocial Safety Climate Theory

128 Psychosocial Safety Climate refers to perceptions about “organisational policies, practices and
129 procedures for the protection of worker psychological health and safety” (Dollard & Bakker, 2010, p.
130 580). PSC is largely determined by management values and practices, and organisational systems
131 that enable communication and participation, in prevention, identification and resolution of work
132 stress related issues. In high PSC contexts managers are concerned for worker health and wellbeing,
133 and design jobs that have manageable demands and adequate resources (Dollard & Bakker, 2010;
134 Dollard et al., 2009; Law, Dollard, Tuckey, & Dormann, 2011). Low PSC workplaces are
135 characterised by senior management values, for example, that prioritise short-term productivity
136 over the psychological health of employees; jobs may be designed with unmanageable psychological
137 and emotional demands (Bailey, Dollard, McLinton, & Richards, 2015). Since PSC predicts the way
138 jobs are designed, it is theoretically a precursor to the job design stress theories, and has been shown
139 empirically to predict effort-reward imbalance (Owen et al., 2016) and Job Demand-Control job
140 strain (Dollard, Opie et al., 2012).

141 Psychosocial Safety Climate has been shown to predict psychological health outcomes such as
142 depression (Becher & Dollard, 2016; McTernan, Dollard, & LaMontagne, 2013), psychological
143 distress (Becher & Dollard, 2016; Law et al., 2011), and emotional exhaustion (Law et al., 2011). Yet,
144 CVD has not yet been investigated as an outcome of PSC. There is limited evidence available to
145 demonstrate the predictive power of PSC on physical health in a longitudinal sample. Longitudinal
146 designs are better suited to teasing out causal effects. The current study addresses a gap in the
147 literature by examining the link between PSC and future CVD over a subsequent four to six years

148 after initial measurement. Since PSC can negatively predict a range of risk factors for work stress,
149 including those embodied in Effort-Reward Imbalance and Job Demand-Control theories we
150 propose a hypothesis that PSC negatively predicts future CVD over and above effects due to
151 effort-reward imbalance, and Job Demand-Control job strain.

152 2. Materials and Methods

153 2.1. Participants

154 Participants were interviewed using Computer Assisted Telephone Interviewing as part of the
155 Australian Workplace Barometer (AWB) project, a national surveillance project of psychosocial risks
156 in Australian workplaces. We used a subsample of the wider AWB study, including participants
157 with data at two times points on average 5 years apart, excluding, self-employed and missing data
158 on health outcome measures.

159 The final sample comprised 1223 participants who were free from any CVD at Time 1, 545
160 (44.6%) males and 678 (55.4%) females, aged between 18 and 73 (median = 47 years) at Time 1. Their
161 education status was diverse with 35.8% holding a bachelor degree or higher, 29.4% with a certificate
162 or diploma, 8% trade/apprenticeship, 17.7% left school after age of 16, 9.1% left school at 16 years or
163 less. The median annual income was 50 to 60 thousand AUD. The participants were located in three
164 different Australian states: South Australia ($n = 428$), Western Australia ($n = 439$), and New South
165 Wales ($n = 356$). Time 1 data collection was conducted in 2009 in NSW and WA, and 2010 in SA. Time
166 2 data was collected in 2014-15 across all three states. All subjects gave their informed consent for
167 inclusion before they participated in the study. The study protocol was approved by the University
168 of South Australia Human Research Ethics Committee (approved 17th June 2009).

169 2.2. Measures

170 Information was collected on participant's age, gender, socioeconomic status and education
171 level, which were all included as covariates in all analyses as in other CVD research (e.g., Nyberg,
172 Heikkila, Fransson, *et al.*, 2012).

173 Psychosocial Safety Climate was measured using the PSC-12, a 12-item questionnaire consisting
174 of the four sub-scales each of which have three items (Hall, Dollard, & Coward, 2010). The subscales
175 and example items are; management commitment, e.g., "In my workplace senior management acts
176 quickly to correct problems/issues that affect employees' psychological health"; management
177 priority, e.g., "Senior management considers employee psychological health to be as important as
178 productivity"; organisational participation, e.g., "Employees are encouraged to become involved in
179 psychological health and safety matters", and organisational communication, e.g., "There is good
180 communication here about psychological safety issues which effect me". Responses are scored on a
181 5-point Likert scale from 1 (*strongly disagree*) to 5 (*strongly agree*). Since the subscales are highly
182 correlated for practical purposes we added all the scales together to form a global measure, $\alpha = .94$.
183 Psychosocial Safety Climate benchmarks used in this study, developed by Bailey and colleagues
184 (2015), were PSC low (≤ 37), moderate (37.01 - 40.99), and high (≥ 41). We used these benchmarks to
185 create three levels of PSC.

186 Cardiovascular disease was measured using questions from the World Health Organization
187 Health and Work Performance Questionnaire (Kessler *et al.*, 2003). Participants were asked whether
188 "in the past two years have you consulted a health professional with regard to chest pain, or any
189 other cardiovascular related health problem – such as myocardial infarction; angina; stroke; or
190 hypertension?", and if so, what diagnosis was returned, and the four disease categories were listed.
191 CVD was dummy coded as 1 (*cardiovascular disease diagnosed*) or 0 (*no doctor visit or no cardiovascular
192 disease diagnosed*). We ruled out "other" diagnoses mentioned such as heart murmur, stress, blood
193 clot, no problem.

194 Effort-reward imbalance was measured using the ratio of effort to reward. For convenience,
195 extrinsic effort was measured using five items from the psychological demands subscale of the Job
196 Content Questionnaire (JCQ, Karasek *et al.*, 1998), with responses on a four-point Likert scale with

197 responses ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Higher scores represent a greater
198 amount of perceived effort by the worker, $\alpha = .68$. Rewards were measured based on 4 items from
199 the Effort-Reward Imbalance Scale (Siegrist, 1996) with 1 item from the esteem reward component:
200 “Considering all my efforts and achievements, I receive the respect and prestige I deserve at work”;
201 2 items from the job promotion reward component an example being, “Considering all my efforts
202 and achievements, my job prospects are adequate”; and 1 item from the job security reward
203 component, “My job security is poor”. The items were measured on a four-point Likert scale,
204 ranging from 1 (*strongly disagree*) to 4 (*strongly agree*) as recommended (Montano, Li, & Siegrist,
205 2016). Higher scores represent a greater amount of perceived organizational rewards received by the
206 worker, $\alpha = .68$.

207 Effort-reward imbalance was calculated using the ratio method as recommended by
208 Effort-Reward Imbalance theorists and this formulation has construct validity (Siegrist et al., 2004).
209 The formula for this calculation is $\frac{\text{Effort}}{\text{Rewards} \times 1.25}$. The correction factor of 1.25 accounts for the unequal
210 amount of items between the Efforts (demands) and Rewards measures. At Time 1, there were 426
211 (34.8%; before removal of baseline CVD, 36.3%) in high effort-low reward jobs ($\text{ERI} > 1$).

212 Job Demand-Control job strain was assessed using combinations of job demands and control.
213 Job demands was the same measure as ‘effort’ described above, assessed with the five item
214 psychological demands subscale of the JCQ (Karasek et al., 1998), with responses on a 4-point Likert
215 scale with responses ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Higher scores represent a
216 greater amount of perceived demand by the worker, $\alpha = .68$. Job control was assessed from the Job
217 Content Questionnaire (Karasek et al., 1998, www.jcqcenter.org) subscales, skill discretion (six items,
218 e.g., My job requires a high level of skill; $\alpha = .75$) and decision authority (three items, e.g., “My
219 job allows me to make decisions on my own”; $\alpha = .73$). Responses are on a 4-point Likert scale
220 from 1 (*strongly disagree*) to 4 (*strongly agree*).

221 There are several statistical variations used in the literature to calculate job strain. We used the
222 quartile-based job strain as recommended by Choi, Ko, & Östergren (2015) and Karasek, Choi,
223 Östergren, Ferrario, & De Smet, (2007) because of their greater sensitivities than the median-based
224 job strain definition with no significant changes in specificities. The methods result in a five-category
225 version of the Job Demand-Control model leading to five distinct groups – low strain, high strain,
226 passive work, active work, and midpopulation. The Job Demand-Control job strain measure used
227 here assigned 1 (*high strain*), and 0 (*other groups*), and three other dummy variables were entered in
228 the models simultaneously (e.g. 1 (*active work*) and 0 (*other groups*) and so on). In the high strain
229 group, there were 192 (15.7%; before removal of baseline CVD, 15.9%). For interest, using a different
230 approach and dichotomising demands and control (scales equally weighted) at the median yielded n
231 = 191 (15.6%; before removal of baseline CVD, 15.8%) in high strain groups at Time 1 respectively.

232 2.3. Statistical Analyses

233 We used SPSS software for all analyses. For hypothesis testing since the outcome measure was
234 binary we used binary logistic regression. After removing baseline cases of CVD ($n = 97$) we
235 regressed Time 2 CVD on the demographic covariates (Model 1), and entered the work environment
236 measures separately (Model 2, 3, 4), and then simultaneously (Model 5).

237 3. Results

238 3.1. Correlations between Measures

239 As shown in Table 1, of the demographic variables (age, gender, education, income) only age
240 was significantly associated with CVD at Time 2. Of the work measures, PSC was significantly
241 negatively related to CVD at Time 2.

242

243

244 **Table 1.** Intercorrelations between study variables.

	1	2	3	4	5	6	7
1. Age T1							
2. Gender T1	0.03						
3. Education T1	-0.04	0.05					
4. Income T1	0.17***	-0.43***	0.27***				
5. Job Strain T1	-0.01	0.09***	-0.04*	-0.05			
6. Effort-Reward Imbalance T1	0.00	0.10***	0.11***	0.05	0.37***		
7. Psychosocial Safety Climate T1	-0.00	0.02	-0.04	-0.04*	-0.35***	-0.25***	
8. CVD T2	0.11***	0.00	-0.07	0.02	-0.00	0.02	-0.06*

245 Note. ***, $p < .001$; **, $p < .01$; *, $p < .05$. T, Time. $n = 1223$.246 *3.2 Incidence Rate of CVD*

247 Over the 5 year period, 98 new CVD cases occurred among 1223 participants who were free
 248 from any CVD at Time 1 (cumulative incidence rate = 8%). We conducted some preliminary
 249 incidence tests of CVD by PSC benchmarks. Comparisons between low, moderate, and high PSC
 250 environments demonstrate that those in high PSC environments exhibited lower rates of overall
 251 CVD after an approximate five year time lag (see Table 2). Participants in low and moderate PSC
 252 environments were more likely (59% and 45% more, respectively) to develop CVD than those in
 253 high PSC environments. The sensitivity analyses, using different levels of PSC, demonstrated a
 254 higher level of CVD in participants working in low PSC (high risk) work environments.
 255

256 **Table 1.** PSC benchmarks and CVD incidence.

PSC Time 1	Number of participants	Participants with CVD at Time 2	% with CVD at Time 2	Average higher incidence
Low	365	41	11.23%	59%
Moderate	97	10	10.30%	45%
High	663	47	7.08%	

257 Note. Low PSC ≤ 37 ; Moderate PSC 37.01 - 40.99; High PSC ≥ 41 . $N = 1223$ (history of CVD removed).

258 As shown in Table 3, Model 1, the demographics age and education at Time 1 were related to
 259 CVD at Time 2. Controlling only for the significant demographics age and education, as shown in
 260 Model 2, job strain, and Model 3, effort-reward imbalance, were not significantly associated with
 261 future CVD; as shown in Model 4, PSC was significantly related to future CVD. Our hypothesis that
 262 PSC predicts future CVD over and above effects due to job design factors (effort-reward imbalance,
 263 and Job Demand-Control job strain) was supported.
 264

265 **Table 3.** Predicting Cardiovascular Disease at Time 2.

		B	S.E.	Wald	Sig.	Odds Ratio	Low CI	High CI
Model 1	Constant	-4.30	0.74	33.31	0.00	0.01	0.00	0.06
	Age Time 1	0.04	0.01	18.62	0.00	1.05	1.02	1.06
	Gender Time 1	0.07	0.22	0.11	0.74	1.08	0.70	1.65
	Education Time 1	-0.13	0.06	5.25	0.02	0.88	0.78	0.99
	Income Time 1	0.08	0.05	2.45	0.12	1.09	0.98	1.19
Model 2	JCQ Job Strain Time 1#	0.09	0.40	0.06	0.81	1.10	0.50	2.40
Model 3	Effort-Reward Imbalance Time 1	0.50	0.38	1.71	0.19	1.65	0.78	3.47
Model 4	Psychosocial Safety Climate Time 1	-0.02	0.01	4.22	0.04	0.98	0.96	1.00
Model 5	Constant	-3.08	0.98	9.81	0.00	0.05	0.01	0.31
	Age Time 1	0.04	0.01	12.99	0.00	1.04	1.02	1.06
	Education Time 1	-0.13	0.06	4.84	0.03	0.87	0.78	0.99
	Effort-Reward Imbalance Time 1	0.51	0.47	1.18	0.28	1.66	0.66	4.18
	JCQ Job Strain Time 1	-0.47	0.45	1.08	0.30	0.62	0.26	1.51
	Psychosocial Safety Climate Time 1	-0.02	0.01	4.34	0.04	0.98	0.96	1.00

266 Note. # Job strain was always entered with 3 other dummy variables. ^, an alternative measure of job strain,
 267 with standardised demand and control measures dichotomised at the mean to form 4 categories, high demand,
 268 high control = 1, else = 0 also yielded non significant effects, as did the multiplicative interaction term. PSC was
 269 entered as a continuous measure as was effort-reward ratio.

270 4. Discussion

271 Studies which have neglected to assess PSC may have underestimated the effect of the work
 272 environment on CVD. This research expands previous research that has linked task related
 273 psychosocial work conditions to CVD (e.g. Li et al., 2015) by including a more distal organisational
 274 level factor, that is PSC. The aim of this research was to explore the relationship between PSC and
 275 CVD, compared to more traditional psychosocial risk factors: effort-reward imbalance and Job
 276 Demand-Control job strain. We also explored the relationship between PSC and CVD before and
 277 after adjustment for effort-reward imbalance ratio and Job Demand-Control job strain. We used a
 278 two-wave longitudinal sample to demonstrate the causal nature of the effect of organisational and
 279 psychosocial work conditions on CVD in workers. Furthermore, an average 5 year gap between the
 280 first and the final rounds of data collection allowed us the opportunity to examine the longer term
 281 effect of psychosocial risks on a chronic and ongoing health problem (i.e., CVD). This is an important
 282 contribution to the literature, as many studies either only present a cross-sectional correlation (Peter
 283 et al., 1998), providing no evidence of causation, or include time lags as short as one year (van
 284 Amelsvoort, Schouten, & Kok, 2004) which is insufficient to measure the onset of many chronic
 285 diseases.

286 Logistic regression showed that PSC is significantly negatively related to higher CVD risk (OR =
 287 0.98, 95% CI 0.96-1.00). This risk remained, after additional adjustment for job strain and ERI
 288 measures. Work job design factors, effort-reward imbalance and job strain were not significant
 289 contributors to future CVD.

290 The research demonstrates that a climate for psychological health and safety predicts future
 291 cardiovascular disease. Workers who believe that their employers are not prioritising their mental
 292 health are more likely to experience cardiovascular disease over the next five years. The results are
 293 somewhat at odds with much previous research showing the detrimental effect of Job
 294 Demand-Control job strain and effort-reward imbalance on CVD; for instance in relation to Dragano

295 et al.'s (2016) multi-cohort finding, our sample size was much smaller (cf., 90, 164) and the prediction
296 time span was smaller (cf., 9.8 years).

297 *4.1. Practical Implications*

298 Psychosocial Safety Climate is a reflection of the priorities and practices of senior management
299 within an organisation, and therefore presents an ideal intervention point for those seeking to
300 address the workplace psychosocial factors relating to CVD.

301 In the UK alone, a 1% reduction in CVD risk is estimated to prevent 25,000 CVD cases per year
302 and save €40 million per year (Collins et al., 2014). Assuming similar PSC rates to Australia, if
303 workers in low and moderate PSC workplaces had their CVD incidence reduced to that of workers
304 in high PSC workplaces, this would represent a 40% decrease in CVD risk, or approximately €4
305 billion per year in UK terms. Workplace interventions to improve PSC could potentially reduce CVD
306 risk substantially, saving billions of dollars in developed countries around the world.

307 This study provides policy makers with additional evidence of the harm caused by
308 psychosocial risks in the workplace. Given the substantial body of evidence demonstrating the
309 important role that job characteristics in the aetiology of CVD, and the evidence the PSC precedes
310 work conditions shown elsewhere, and the link between PSC and CVD shown here, there is a drastic
311 need for organizational intervention research to determine whether psychosocial risk prevention
312 reduces CVD in workers, potentially saving lives, improving wellbeing, and increasing productivity.
313 Policy makers should consider psychosocial risk management as an additional tool in the public
314 health campaigns aimed at reducing CVD. Businesses that wish to improve organisational health
315 should consider a PSC intervention to reduce CVD onset in workers.

316 *4.2. Limitations*

317 One reason that we did not observe correlations between ERI and JD-C job strain may have
318 been that those with high levels left the sample. Compared to our very initial sample of 3030, the
319 proportion of employees in high strain jobs was 21% whereas in our matched sample over 5 years it
320 was around 16%. Our results may be at variance with other studies, due to measures used, length of
321 time lag (5 years), and the general working population sample. Our study analysed PSC at the
322 individual level, despite its conceptualisation as an organisational level construct. This is a limitation
323 of the population-based sampling technique used in the AWB project. Population-based sampling
324 provided a representative sample of Australian workers from all major industries, occupations, and
325 demographic groups. However, it also provides fewer organisations with sufficient group sizes for
326 multilevel analysis (Scherbaum & Ferreter, 2009). Given that CVD only occurred in a small
327 proportion of the population, and the population-based sampling technique used, analysing PSC at
328 the organisational-level would have reduced the power of the analysis too severely. As such, the
329 likelihood of a Type II error would be too high, so an individual-level analysis of PSC was used. It is
330 possible that the assumption of independence of the data used was violated, as approximately 20%
331 of the participants belonged to the same organisation. We justified analysis at the individual-level
332 based on previous research demonstrating that PSC has some individual-level properties separate
333 from organizational level influences (Bailey et al., 2015; Dollard & Bakker, 2010). Another limitation
334 goes to the measurement of CVD. In our study, the incident cases of CVD were based on self-reports.
335 Though register data (such as hospitalization records) are generally preferred, it has been shown
336 that self-reported CVD have reasonable sensitivity and specificity, with acceptable agreement to
337 medically certified records (Okura et al., 2004).

338 **5. Conclusions**

339 This longitudinal research, in Australia, showed that cardiovascular problems newly diagnosed
340 by a doctor, could be best predicted over a five year time period by PSC. Understanding the
341 association between an organisation's PSC and the CVD risk borne by its workers may allow for

342 organisational level interventions to reduce CVD risk and inform policy makers on potential
343 legislative requirements for CVD reduction measures through PSC.

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345 study conception and design; Harry Becher and Maureen Dollard conducted the statistical analyses and
346 prepared a first draft of the manuscript; Peter Smith added to the statistical analysis; Peter Smith and Jian Li
347 provided a substantial and critical review of the results and the manuscript. All authors contributed
348 substantially to the interpretation of the data and to the revision of the manuscript for important intellectual
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354 **Dedication:** In honor of Harry Becher, his sharp mind, and his passion for better work conditions.
355

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