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Published in:
Social Science & Medicine

DOI:
10.1016/j.socscimed.2022.114805

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2022

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
Högnäs, R. S., Bijlsma, M. J., Högnäs, U., Blomqvist, S., Westerlund, H., & Hanson, L. M. (2022). It's giving me the blues: A fixed-effects and g-formula approach to understanding job insecurity, sleep disturbances, and major depression. *Social Science & Medicine*, *297*, [114805]. https://doi.org/10.1016/j.socscimed.2022.114805

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Social Science & Medicine 297 (2022) 114805

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

It's giving me the blues: A fixed-effects and g-formula approach to understanding job insecurity, sleep disturbances, and major depression☆

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ARTICLE INFO

Keywords:
Job insecurity
Sleep disturbances
Major depression
Causal mediation

ABSTRACT

Research suggests that work-related factors like job insecurity increases the risk of major depression (MD), although it is unclear whether the association is causal. Research further suggests that job insecurity increases sleep disturbances, which is also a risk factor for MD. Based on current knowledge, it is possible that job insecurity operates through sleep disturbances to affect MD, but this pathway has not been examined in the literature. The current study extends the literature by using two complementary, counterfactual approaches (i.e., random- and fixed-effects regression and a mediational g-formula) to examine whether job insecurity causes MD and whether sleep disturbances mediate the relationship. A methodological triangulation approach allowed us to adjust for unobserved and intermediate confounding, which has not been addressed in prior research. Findings suggest that the relationship between job insecurity and MD is primarily direct, that hypothetically intervening on job insecurity (in our g-formula) would reduce MD by approximately 10% at the population level, and this relationship operates via sleep disturbances to some degree. However, the indirect pathway had a high degree of uncertainty.

1. Introduction

According to the World Health Organization, more than two-hundred and fifty million people worldwide suffer from depression (WHO, 2017). Depression can affect a wide range of daily activities, negatively alter one's life course, and may even lead to suicide (Lépine and Briley, 2011). Approximately 4.9% of the Swedish population suffered from depression in 2015 (WHO, 2017), although the true prevalence may be higher if it is concealed or undiagnosed (e.g., Goldman et al., 1999). Several overlapping factors increase the risk of depression, including family history, childhood abuse, personal relationships, and the focus here, work-based circumstances (e.g., Köhler et al., 2018). Indeed, the quality of working life, encompassing the work environment and labor market attachment, is an important risk factor throughout the life course (Bonde, 2008; Kim and von dem Knesebeck, 2016).

Work provides pecuniary benefits (e.g., income) and can provide nonpecuniary benefits including a sense of purpose and work-based social connections. Research suggests that feelings of isolation and exclusion from society may follow job loss, particularly over long spells of unemployment (Korpi, 1997; Ochsen and Welsch, 2011; Pohlan, 2019; see also Rözer et al., 2020). Unemployment is also negatively associated with physical health (e.g., Herber et al., 2019), e.g., lower self-rated health, smoking, and weight gain (Golden and Perreira, 2015; Minelli et al., 2014; Monsivais et al., 2015). It further increases the risk of mental health problems (Zhang and Bhavsar, 2013), including the use of psychotropic medication (Bijlsma et al., 2017; Bijlsma et al., 2019) and depression (Zuelke et al., 2018). A growing body of research suggests that *job insecurity* may be an even stronger predictor of mental

☆ This study was funded by Nordforsk (#75021). SLOSH data collection and management has also been supported by the Swedish Research Council, and the Swedish Research Council for Health, Working Life and Welfare (# 2019-01321) partly through Stockholm Stress Center of Excellence. Please direct questions to Robin S. Högnäs, robin.hognas@su.se, Stress Research Institute, Stockholm University, Sweden. The authors would like to thank Ben Wilson for valuable advice during the early stages of this project.

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https://doi.org/10.1016/j.socscimed.2022.114805
Received 3 October 2021; Received in revised form 7 February 2022; Accepted 10 February 2022
Available online 12 February 2022
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health problems (Kim and von dem Knesebeck, 2016; LaMontagne et al., 2021; Milner et al., 2016; Watson and Osberg, 2018; de Witte, 1999).

1.1. Job insecurity, health, & depression

The concept of job insecurity is distinct from unemployment. It is defined as the feeling that employment is in jeopardy, or a perception or fear of involuntary job loss (e.g., Caroli and Godard, 2016; de Witte, Pienaar and de Cuyper, 2016; Mohr, 2000). Such fears may exist for many reasons, including the structural conditions of the labor market. For example, recent estimates suggest that job tenure, an important marker of employment security, has decreased by more than 17% in Sweden over the past decade (OECD, 2019). Moreover, the number of temporary contracts in Sweden and the U.S. increased between 2005 and 2019 (Kratz and Krueger, 2019; Hellsing and Samuelsson, 2020). These labor market conditions potentially affect how workers view the security of their jobs, regardless of whether they are objectively more or less secure (Keim et al., 2014; Milner et al., 2014).

Job insecurity is conceptualized as a psychosocial working condition, influenced by economic, social, political, and proximal workplace structures within which employment contracts are negotiated and carried out. When workers feel insecure in their employment, these psychosocial working conditions may negatively affect cognition, emotion, behavior, and ultimately physical or mental health (Rugulies, 2019). Reactions to repeated arousal, strain, or “allostatic load” (McEwen, 2000) may induce primary effects like sleep problems, anxiety, and/or mood changes and secondary effects like abnormal metabolism, cardiovascular risk factors, and/or inflammation. Over time, these reactions could increase the risk of chronic stress dysregulation associated with tertiary effects, or clinical disorders like cardiometabolic diseases, chronic pain, cancer, and depression (Mauss et al., 2015).

Indeed, research shows that job insecurity increases the risk of disease, including diabetes (Ferrie et al., 2016) and coronary heart disease (Magnusson Hanson et al., 2020; Virtanen et al., 2013). Mental health may also be important along the causal pathway between job insecurity and physical health. Magnusson Hanson et al. (2020) found that job insecurity increased the risk of heart disease primarily via psychological distress (i.e., symptoms of depression and anxiety). Moreover, research from several countries underscores the significance of job insecurity in psychological distress (Niedhammer et al., 2012; Burgard and Seelye, 2017; de Witte et al., 2016; Kim and von dem Knesebeck, 2016; Watson and Osberg, 2018), including purchases of psychotropic drugs (Blomqvist et al., 2020). In fact, LaMontagne et al. (2021) recently found that improving job security elevated Australian workers’ mental health even after adjusting for unobserved confounding.

Studies further suggest that job insecurity increases the risk of major depression (e.g., Blackmore et al., 2007; Wang et al., 2008; Wang et al., 2012). Magnusson Hanson et al. (2015), for example, found that repeated threats of dismissal from work increased major depressive symptoms, which subsequently increased threats of dismissal. While this issue of reverse causality raised by Magnusson Hanson et al. (2015) remains unclear (Griep et al., 2021; Shoss, 2017), a systematic review of 57 longitudinal studies on job insecurity and several mental health indicators (e.g., psychological distress, depression) suggested that reverse causality was not supported in the few studies that addressed it (de Witte et al., 2016).

1.2. Job insecurity and sleep disturbances

There are multiple reasons to hypothesize that the causal pathway through which job insecurity affects major depression includes sleep disturbances. Theoretically, job insecurity likely contributes to one’s allostatic load (e.g., McEwen, 2000), the primary effects of which may decrease the quantity and quality of sleep. Fears of job loss may lead to rumination and worry that may affect sleep. Rumination or perseverative cognitions—an inability to turn off work-related stressful thoughts—may stall recovery, lead to poor sleep, and adversely affect health (e.g., Kivimäki et al., 2006; Berset et al., 2011). Work stressors like effort-reward imbalance, overload, hectic and physically strenuous work, shift work, and even the anticipation of work stress can negatively affect sleep (Åkerstedt et al., 2002; Fahlén et al., 2006; Kecklund and Åkerstedt, 2004). Greubel and Kecklund (2011) also found that downsizing—linked to job insecurity—slightly increased sleep disturbances and subsequently depressive symptoms.

Only a few studies have examined the relationship between job insecurity and sleep problems, and overall, findings have been mixed. For example, Burgard and Ailshire (2009) found a negative association between job insecurity and sleep quality, but more immediate psychosocial work factors (e.g., upsetting experiences at work) were more important. Using French data, Chazelle et al. (2016) found no significant association between job insecurity in 2006 and sleep problems in 2010, although the time lag in this study limits any causal interpretation. Other studies, however, have used a diversity of data sources and sleep measures (e.g., hours of sleep, consistent sleep, quality of sleep), and have found a significant positive association between job insecurity and sleep problems (Ferrie et al., 1998; Magnusson Hanson et al., 2020; Virtanen et al., 2011).

1.3. Sleep disturbances and depression

A long line of research suggests that disentangling the direction of causality between sleep disturbances (primarily insomnia) and depression is difficult because the two are neurobiologically interconnected (e.g., Alvaro et al., 2013; Jansson-Fröjmark and Lindblom, 2008). There is, however, evidence that sleep disturbances may cause depression. For example, a psychiatric community-based study of adolescents found that insomnia predicted depression, but depression did not predict insomnia (Johnson et al., 2006). A meta-analysis also found that people without a history of depression were two times more likely to become depressed if they had trouble with insomnia. Baglioni and Riemann (2012) further underscored well-documented clinical connections between insomnia and the onset of depression for adolescents, adults, and aging populations. Moreover, brain functions connected to mood are inherently linked to circadian rhythms (e.g., Pandi-Perumal et al., 2020).

1.4. The current study

Research shows that work stressors disrupt sleep and sleep disturbances increase the risk of depression. The current study focuses on job insecurity and major depression (MD) and assesses direct and indirect (via sleep disturbances) associations between the two. An examination of sleep disturbances as a potential causal mediator stands to advance overall knowledge about the risk factors of MD. In addition, causal analyses of the relationship between job insecurity and MD have been limited, particularly in terms of adjusting for unobserved and intermediate confounding. Sleep disturbances may mediate the relationship between job insecurity (the exposure) and MD (the outcome), but may also confound the relationship between subsequent job insecurity, MD, and time-varying covariates (e.g., VanderWeele et al., 2014). This intermediate confounding makes it difficult to determine the indirect effect of job insecurity via sleep disturbances on MD, and is therefore important to address in causal analyses.

We extend the literature by examining the causal relationship between job insecurity and MD using two complementary, counterfactual approaches: 1) fixed-effects regression that conditions on unobserved time-constant confounding; 2) a longitudinal g-formula that conditions on observed time-varying confounding and assesses whether removing job insecurity at the population-level (our hypothetical intervention) reduces the risk of MD (e.g., Robins, 1986; Robins and Hernán, 2009; Bijlsma et al., 2017); and 3) decomposing the average total effect of the “intervention” into the average direct effect of removing job insecurity and the average indirect effect of sleep disturbances.

Specifically, we examine the following research questions: Is job insecurity associated with MD among working adults, accounting for unobserved time-constant and observed time-varying demographic and confounding factors? If we intervene on job insecurity, while also adjusting for intermediate confounding, what effect would this have on MD at the population level? To what extent does the effect operate through sleep disturbances?

2. Data and measures

2.1. Data

Data are from the Swedish Longitudinal Occupational Survey of Health (SLOSH), a nationally representative cohort study on work life, health, and individual well-being. SLOSH began following participants ages 16–64 from the randomly sampled 2003 Swedish Work Environment Survey (SWES) ($N = 9214$) in 2006. There are 7 follow-up waves with two-year intervals up to 2020, but data were available for the current study up to 2018. Refresher samples were added from SWES 2005, 2007, 2009, and 2011 (see Magnusson Hanson et al., 2018 for a detailed description). Participants self-administered one of two surveys at each wave depending on whether they were: 1) *in gainful employment* (i.e., ≥30% full-time); or 2) *not in gainful employment* (<30%) temporarily, or permanently out of the labor force.

This study uses a panel of SLOSH participants observed over five waves, from 2010 (when job insecurity was first observed) to 2018. Among those with non-missing on major depression in all five waves ($N = 4879$), participants who were not in gainful employment for two or more consecutive waves were excluded ($N = 1932$). Item missing for remaining participants ($N = 2947$) ranged from 3% to 14% across waves. Missing on covariates, sleep disturbances, and job insecurity for the continuously gainfully employed (77%) appeared to be missing at random (MAR), and therefore multiply imputing missing values may be less biased compared to complete case analysis (IRDE, 2021).

Among the gainfully employed with nonconsecutive or temporary gaps in employment (23%), average missing across waves was 5% with the highest proportion in the 5th analytic wave (14%). Most of these missing cases were attributed to job insecurity, which was not asked if participants were not gainfully employed at the time of the survey. We wanted to retain participants with temporary gaps. Thus, we ran regression models with and without this group and found neither substantive nor significant differences in the relationship between job insecurity and MD. Therefore, we imputed missing values this group. The MI Impute chained command in Stata (10 imputations) and MICE package in R (an imputation for each bootstrap iteration) was used to multiply impute missing values on covariates (except age, which had no missing), sleep disturbances, and job insecurity. We did not find significant nor substantive differences between the imputed and unimputed regression results, so we retained the analytic sample of $N = 2947$ participants by using the imputed data.

2.2. Major depression (MD)

MD was operationalized in terms of whether or not participants had a high severity of depressive symptoms. First, a 6-item subscale from the Symptom Checklist Core Depression Scale (SCL-CD_6) was derived from the Hopkins Symptom Checklist (SCL-90). Items included: 1) felt blue or sad; 2) had no interest in things; 3) had low levels of energy; 4) felt like everything was an effort; 5) worried too much; and 6) had self-blame (alpha reliability = .90). Responses ranged from 1 = *not at all* to 5 = *extremely*. Systematic analyses in prior research found the subscale to be a valid and reliable measure of the severity of depression; and using ROC analyses determined that a score of 17 out of a range of 0–24 is an appropriate cut-point for MD (see Magnusson Hanson et al., 2014 for details). The final measure of MD was a dichotomous variable drawn from the validated SCL-CD_6 symptoms cut-point, where 1 = *major depression* and 0 = *no major depression*.

2.3. Main exposure

Job insecurity was measured in terms of whether participants worried about being laid off, keeping their job, and/or a fear of job loss. Response categories ranged from 1 = *completely disagree* to 5 = *completely agree*. If participants agreed (i.e., a 4 or 5) with any of the three indicators, they were coded as exposed to job insecurity. The final measure was a dichotomous variable, where 1 = *job insecurity* and 0 = *no job insecurity*.

2.4. Time-varying mediator

Sleep disturbance was measured using the Karolinska Sleep Questionnaire, which included indicators for whether participants experienced: 1) difficulty falling asleep; 2) repeated awakenings; 3) early awakening; and 4) disturbed sleep (Magnusson Hanson et al., 2011). Categories ranged from 1 = *never* to 6 = *always or five or more times per week*. Consistent with prior research, sleep disturbances were present if participants experienced any one of the four items three or more times per week (Magnusson Hanson et al., 2017; Mallon et al., 2014).

2.5. Covariates

Time-varying covariates included personal income and civil status as these factors potentially confound the exposure-mediator and mediator-outcome relationships.[1] Research shows that income is strongly associated with work life; those with lower skills and lower income tend to be more vulnerable in the labor force and to mental health problems than their higher earning counterparts (e.g., Hernández-Quevedo et al., 2006; Oesch, 2010). Income was linked to SLOSH from the Longitudinal Integrated Database for Health Insurance and Labor Market Studies (LISA) registers and was reported in Swedish kronor; it is measured here in quintiles, where 1 = *1st quintile* and 5 = *5th quintile*. Civil status is also a potential confounder. Marriage tends to be positively associated with employment stability, income, and mental health; changes in civil status (e.g., from married to single) may decrease employment stability and women's earnings, and increase short and long-term risks of depression (Kamiya et al., 2013; Raz-Yurovich, 2013; Schoeni, 1995). From SLOSH data, civil status is measured as a dichotomous variable, where 1 = *married or cohabiting* and 0 = *single*.

We controlled for demographic factors on which job insecurity, sleep disturbances, and/or mental health tend to vary, i.e., sex, education, and age (Arber et al., 2009; Kuehner, 2017; Näswall and de Witte, 2003). Sex was measured using a dichotomous variable, where 1 = *female* and 0 = *male*. Three dichotomous variables for *primary and lower secondary*, *upper secondary*, and *university* measured education. Age was measured in years.

3. Analytic approach

In the first stage of the analysis, we estimated logistic regression, random-effects, and fixed-effects models in three steps: 1) MD (outcome) as a function of job insecurity (exposure); 2) sleep disturbances (mediator) as a function of job insecurity (exposure); and 3) MD (outcome) as a function of sleep disturbances (mediator). In the second stage, we begin with a causal directed acyclic graph (DAG) and estimate the relationships illustrated in the DAG. We then define the intervention scenarios and use multivariable models in the g-formula to simulate these scenarios.

[1] We included a time-varying variable for the presence of any one of six chronic diseases. It did not change the models estimated in Table 2 in any substantive nor significant way and was therefore not included.

3.1. Random- and fixed-effects

We conducted several analyses to evaluate the robustness of the exposure-mediator and mediator-outcome associations. Our concern was whether job insecurity reasonably had causal effects on MD, or whether this measure of job insecurity was a proxy for other omitted variables. For example, does job insecurity affect MD through one's ability to sleep well, as prior research would imply? Or, does job insecurity include an emotional element (e.g., anxiety about job loss) that reflects a source of unobserved heterogeneity across individuals that determines MD, such as a genetic predisposition or early childhood experience?

To better address issues of selectivity, we estimated random-effects (RE) and fixed-effects (FE) using repeated measures pooled over time. RE models capture variation between and within subjects, denoted as $log\left[\frac{Pit}{1-Pit}\right] = \mu_t + \beta X_{it} + \gamma Z_i + \delta W_{it} + \alpha_i + \varepsilon_{it}$. Where, $log\left[\frac{Pit}{1-Pit}\right]$ represents the log odds of MD, X is time-varying exposures to job insecurity, Z is time-invariant covariates (i.e., sex and education), W is time-varying covariates (i.e., marital status, income, and sleep disturbances), α is a set of random variables each with a mean of 0 and constant variance, and ε is random error across individuals and time. FE models capture within-person variation and reflect how changes in job insecurity affect changes in MD over time. They are denoted as $log\left[\frac{Pit}{1-Pit}\right] = \beta X_{it} + \delta W_{it} + \alpha_i$, where X is job insecurity, W is the vector of time-varying covariates, and α_i is the individual fixed effect. FE is a more conservative technique and reduces bias by adjusting for unobserved, time-constant confounding or unmeasured fixed individual characteristics that may affect both job insecurity and MD (Allison, 2009).

3.2. Longitudinal g-formula

In stage two, we evaluate the possible causal relationship between job insecurity and MD using a longitudinal g-formula embedded within a counterfactual framework (e.g., Robins and Hernán, 2009). There are several advantages to computing the g-formula as a complement to RE and FE. First, while FE adjust for unobserved time-constant confounding and observed time-varying covariates, the potential for intermediate confounding—that prior exposure, mediator, confounders, and MD affect one another over time—is still present (e.g., VanderWeele et al., 2014). Second, the g-formula here is based on sample statistics from SLOSH, which were used to simulate what happens to MD in the population if we hypothetically intervened on, or eliminated, job insecurity. Third, we could assess the average direct effect of this "intervention" on MD in the population, and what proportion of the effect, on average, operates through sleep disturbances. This approach is computationally lengthy and intensive, but allows for estimating these direct and indirect effects while simultaneously adjusting for intermediate confounding. Our implementation of the g-formula, however, does not adjust for unmeasured confounding—it complements the RE and FE models where unmeasured confounding is adjusted.

3.2.1. Causal DAG

The first part of the g-formula approach includes the multivariable relationships shown in the causal DAG (Fig. 1). Job insecurity, income, civil status, and our mediator, sleep disturbances, in year k and k-1 (the prior observation year) were allowed to affect MD in observation year k. Because we were interested in causal relationships—requiring that exposures precede mediators and outcomes—we also allowed job insecurity, income, civil status, and sleep disturbances from k-1 to affect the time-varying covariates and MD in k. The model includes the parsimonious set of time-varying confounders, and the relationships illustrated were estimated using logistic or multinomial logistic regression.

3.2.2. Total and indirect/mediated effects

For the total effect, we use simulation (Monte Carlo integration) within the g-formula counterfactual framework to compare three scenarios. The *natural course scenario* is based on the empirical observations of job insecurity directly from the SLOSH data. In counterfactual notation, this scenario is represented by $MD(J, S_J)$, where MD represents major depression under the scenario where J (job insecurity) is as observed, and the mediator S (sleep disturbances) is affected by the observed J levels. The counterfactual total or *intervention scenario* assesses what would happen to major depression at the population-level if all observed to have had job insecurity in our sample population did not have job insecurity. This is denoted by $MD(J^*, S_{J^*})$, representing major depression MD when job insecurity is set to 0 and sleep disturbances are affected by this intervention on J. The *counterfactual direct scenario* (CF direct) includes our examination of whether sleep disturbances mediate the association between job insecurity and major depression, denoted as $MD(J^*, S_J)$, MD represents major depression, affected by the intervention on J, but with sleep disturbances following the natural course distribution.

Confidence intervals were determined using 999 bootstrap iterations (e.g., Keil et al., 2014). Models from the DAG were estimated using random draws of individuals with replacement for each bootstrap iteration. The estimated models were then used with observations from the first wave to simulate observations in the next period, and those simulated observations were then used with the multivariable models to simulate subsequent observations up to 2018, the final observation year. This process was done for both scenarios similarly, but with the difference of whether or not job insecurity was "intervened" or observed empirically. The difference in the predictions for the two yields the total effect (TE) of MD if job insecurity were removed at the population-level; using counterfactual notation, $TE: \quad E[MD(J^*, S_{J^*}) - MD(J, S_J)]$.

For the CF direct scenario or mediated effect, the values for individuals with sleep disturbances were set to values in the natural course. Thus, mediator values were observed empirically, and therefore made independent of the values simulated in the intervention scenario. This so-called "blocking" of the mediator allows us to examine how much of the total effect on MD is explained by sleep disturbances. In other words, we decomposed the TE into direct effects (DE) and indirect effects (IE) operating via sleep disturbances (e.g., Bijlsma and Wilson, 2020; Wang and Arah, 2015); using counterfactual notation, $DE: E[MD(J^*, S_J), - MD(J, S_J)]$, and. $IE = TE - DE$.

4. Results

4.1. Descriptive statistics

Table 1 shows baseline summary statistics separately by job insecurity. Approximately 13% who reported job insecurity, versus 4% who did not, experienced MD. Job insecurity was more frequent among those with upper secondary or lower education (54% combined) compared to those with a university or higher level of education (46%). Among those with a university education, 46% reported job insecurity compared to 53% who did not. There were no substantive age differences by job insecurity. There were, however, significant differences in job insecurity by income, marital status, and sleep disturbances. Sixty-four percent in the 1st and 2nd income quintiles reported job insecurity compared to 54% in the same group who reported no job insecurity. Fifty-one percent of married or cohabiting participants reported job insecurity compared to 58% who did not. Finally, job insecurity varied significantly by sleep disturbances—42% with job insecurity versus 25% without it, reported sleep disturbances.

4.2. Multivariable models

Table 2 shows the results from the multivariable logistic regression,

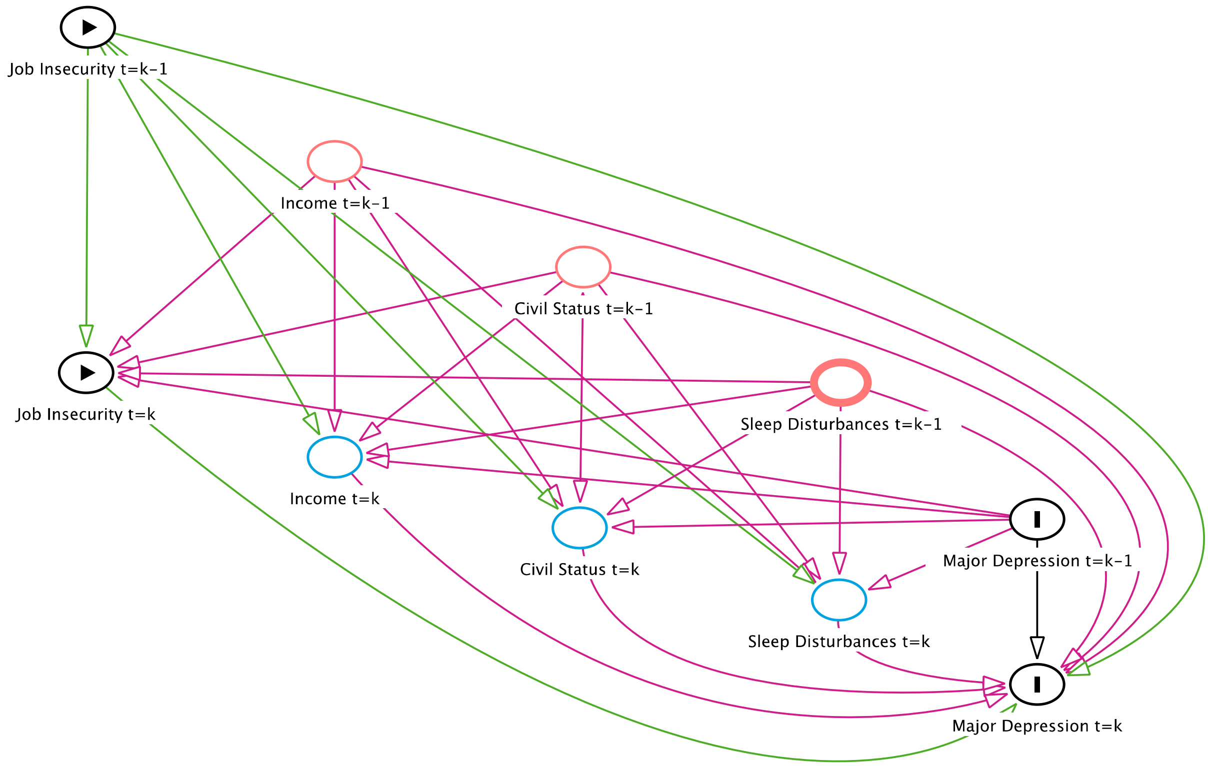

Fig. 1. Causal directed acyclic graph (DAG).

Table 1
Baseline descriptive statistics for the full (unimputed) analytic sample, separately by job insecurity ($N = 2774$).

	Job Insecurity % or Mean (sd)	No Job Insecurity % or Mean (sd)	Sig.[a]
Major Depression (MD)	13	4	***
Sex			
Female	58	59	
Male	42	41	ns
Education			
Primary & Lower Secondary	7	6	
Upper Secondary	47	41	
University	46	53	**
Age	47 (8.7)	48 (8.3)	*
Income			
1st Quintile (ref.)	40	33	
2nd Quintile	24	21	
3rd Quintile	18	18	
4th Quintile	11	15	
5th Quintile	7	14	**
Civil Status			
Married or Cohabiting	51	58	
Single	49	42	*
Sleep Disturbances	42	25	***
N	248	2526	

Notes. *p < .05; **p < .01; ***p < .001.
[a] Chi-square tests where percentages are reported; t-tests where means are reported.

Table 2
Logistic, random-, and fixed-effects estimates for major depression (MD) as a function of job insecurity, sleep disturbances as a function of job insecurity, and MD as a function of sleep disturbances.

	Logistic (95% CI)	RE (95% CI)	FE (95% CI)
Exposure - > Outcome			
Model 1: MD by Job Insecurity + Covariates	**2.48*** ** (1.92–3.19)	**2.70*** ** (1.88–3.87)	**1.94** ** (1.30–2.95) *n* = 348
Exposure - > Mediator			
Model 2: Sleep Disturbances by Job Insecurity + Covariates	**2.02*** ** (1.73–2.36)	1.79*** (1.40–2.28)	**1.30*** (1.01–1.68) *n* = 1262
Mediator - > Outcome			
Model 3: MD by Sleep Disturbances + Covariates	**8.08*** ** (6.71–9.73)	**8.93*** ** (6.94–11.49)	**3.62*** ** (2.67–4.90) *n* = 348

Note: *p < .05; **p < .01; ***<0.001. Odds ratios are reported in all models. Models 1–3 were estimated separately. Model 1 estimates MD as a function of job insecurity and adjusts for the mediator and all covariates, i.e., sleep disturbances and income, civil status, age, education, and sex. Models 2 estimates sleep disturbances (mediator) as a function of job insecurity and adjusts for income, civil status, age, education and sex. Model 3 estimates MD as a function of sleep disturbances and adjusts for job insecurity (exposure), income, civil status, age, education and sex. FE models adjust for time-varying covariates and *n* includes only those cases out of the total analytic sample, $N = 2{,}947$, in which each outcome (y) and predictor (x) change over the observation period.

RE, and FE models.[2] Where appropriate, we adjusted for the same covariates across the three models— time-constant and time-varying covariates were adjusted in the Logit and RE models, and only time-

[2] The Hausman test suggested that FE versus RE results may be preferable. However, we report results from both models as they provide different types of information.

varying covariates were adjusted in the FE models. Starting with Model 1, results from the logistic and RE models suggest that job insecurity increases MD by more than 2 times (OR = 2.48, CI = 1.92–3.19 and 2.70, CI = 1.88–3.87, respectively), net of confounding factors (e.g., income, civil status), sleep disturbances (mediator), and covariates. The FE models suggest that job insecurity increases MD by about 1.94 (CI = 1.30–2.95) times even once unobserved time-constant confounding is adjusted.

Next, Model 2 shows estimates predicting sleep disturbances (mediator) by job insecurity. These results are also consistent across approaches and suggest that job insecurity significantly increases sleep disturbances. After taking into account both within- and between-person differences in the RE models, job insecurity versus no job insecurity increases sleep disturbances by nearly a factor of 2. The FE model further shows a significant association between job insecurity and sleep disturbances, with an approximate 30% increase among those who report job insecurity.

Lastly, Model 3 shows estimates of MD by sleep disturbances, the mediator-outcome association. Regardless of the analytic approach, sleep disturbances significantly increase the odds of MD. In the logistic and RE models, those with sleep disturbances increased MD by over a factor of 8. Even in the most conservative FE models, which adjust for job insecurity, changes in sleep disturbances increased MD by more than 3 times (OR = 3.62, CI = 2.67–4.90, in FE model).

Combined, the results from the first stage of the analysis are consistent with prior research that shows that job insecurity is significantly associated with MD (e.g., Magnusson Hanson et al., 2015; de Witte et al., 2016). These results further suggest that sleep disturbances, at least in part, may mediate the association between job insecurity and MD, although a formal examination of mediation was not possible in the regression analyses. Thus, we turn to the g-formula results where causal mediation was more formally examined (see Table 3 for odds ratios from the g-formula).

Table 3
Odds ratios from logistic regression models in the G-formula predicting major depression (MD) by job insecurity, sleep disturbances, and covariates[a].

	Adjusted Odds Ratio	95% CI
Job Insecurity	2.02	(1.37, 2.91)
Job Insecurity (lagged)	1.07	(.73, 1.52)
Male (ref.)		
Female	1.44	(1.11, 1.87)
Primary and Lower Secondary (ref.)		
Upper Secondary	1.27	(.73, 2.33)
University	1.06	(.60, 1.98)
Age	.98	(.97, .99)
Income		
1st Quintile (ref.)		
2nd Quintile	.57	(.35, .91)
2nd Quintile (lagged)	1.46	(.95, 2.26)
3rd Quintile	.75	(.44, 1.24)
3rd Quintile (lagged)	1.04	(.63, 1.73)
4th Quintile	.63	(.35, 1.13)
4th Quintile (lagged)	1.28	(.72, 2.26)
5th Quintile	.62	(.31, 1.22)
5th Quintile (lagged)	.79	(.39, 1.59)
Civil Status		
Unmarried Single (ref.)		
Married or Cohabiting	1.15	(.64, 2.01)
Married or Cohabiting (lagged)	.74	(.42, 1.31)
Sleep Disturbances	6.17	(4.68, 8.02)
Sleep Disturbances (lagged)	1.42	(1.10, 1.83)
Major depression (lagged)	7.51	(5.70, 9.85)

[a] These ORs were used to predict the probability of MD and were estimated using the full dataset. In the g-formula simulation, probabilities were estimated based on each of the 999 bootstrapped samples from the data.

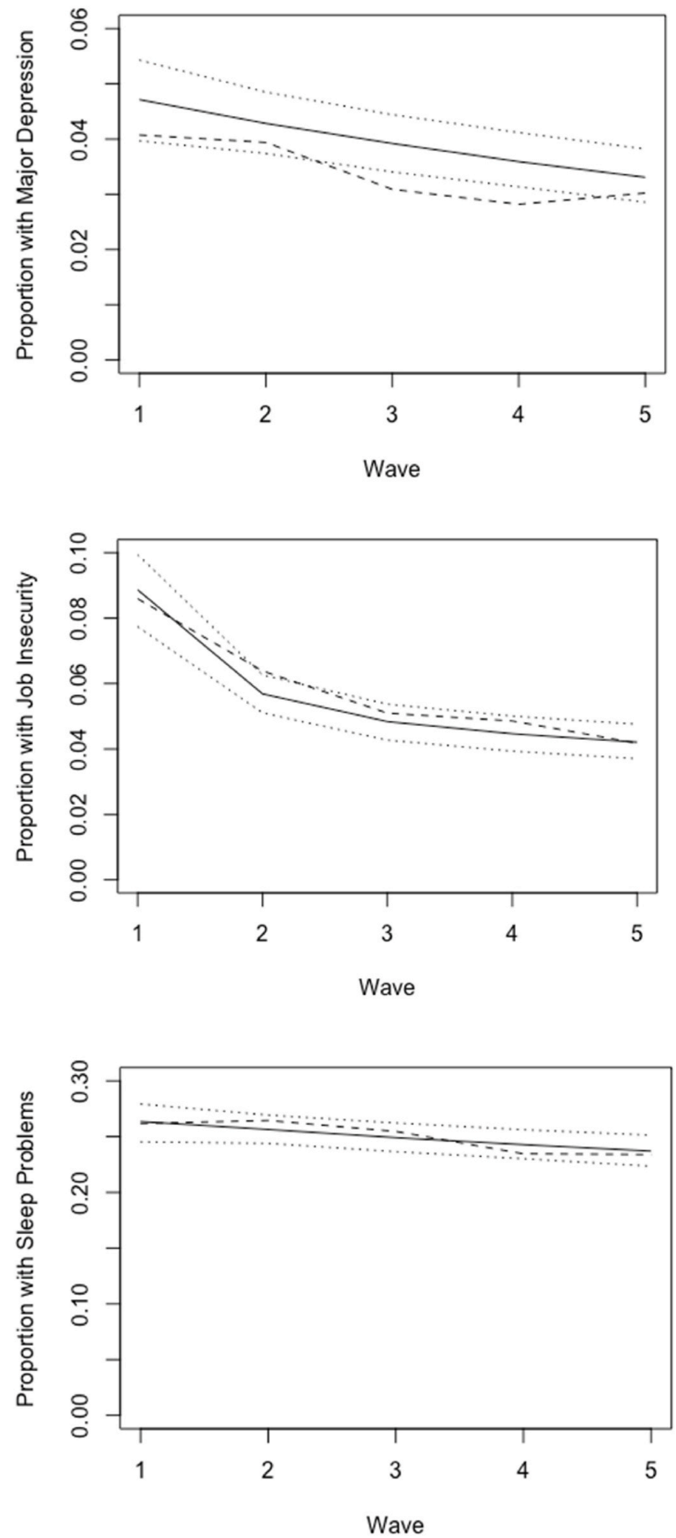

Fig. 2. Natural Course (solid line) with 95% Confidence Intervals (dotted lines) and SLOSH Data (dashed line).

4.3. G-formula results

Fig. 2 shows the descriptive results from the natural course scenario, plotted against the empirical SLOSH data, and 95% confidence intervals for outcome, main exposure, and mediator (i.e., MD, job insecurity, and sleep disturbances). Approximately 5% experienced MD during the 1st observation and 4% by the 5th. Approximately 9% reported job

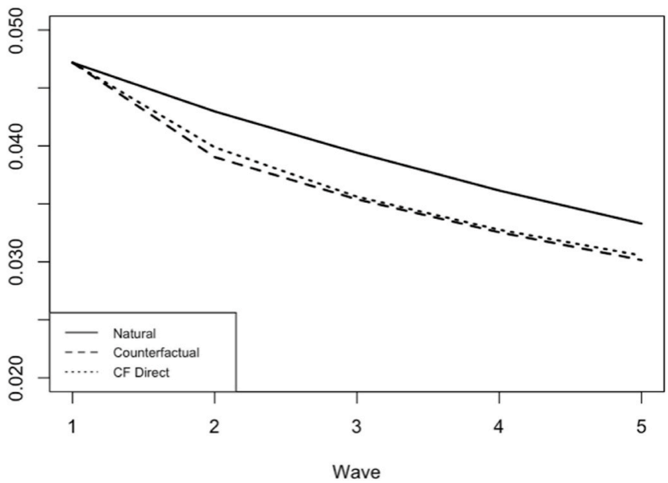

Fig. 3. Proportion with major depression in the natural, counterfactual, and counterfactual direct scenarios. The CF direct scenario via sleep disturbances.

insecurity in the 1st observation but this decreases to about 5% by the 5th. There is only a slight decrease from 26% among those who report sleep disturbances over the five waves. Importantly, these results show that data simulated in the natural course fit well with SLOSH data, with only a slight deviation in MD between the 3rd and 4th observations.

Fig. 3 shows results from the three scenarios in the g-formula: the natural course, counterfactual, and CF direct scenarios. In the natural course and counterfactual scenarios, results suggest that eliminating job insecurity would result in a significant decrease in MD at the population-level. Results suggest that subtracting the natural course from the counterfactual scenario—the hypothetical intervention at the population-level—would decrease MD by about an average of 10% or 0.10 (CI 0.04, 0.15) or 0.5 percentage points at each wave. That is, when we "intervene" on job insecurity, depression decreases from 5% to 4.5%, a reduction of approximately 10% of MD in the population after the first wave. In other words, 0.5% of the population shifts from experiencing MD to not experiencing MD if there were no job insecurity. Results from the CF direct scenario, the mediation model, suggest that this association is partly indirect, via sleep disturbances (IE = 11%, 95% CI = −0.31, 0.53). However, the CIs for the averaged IE are wide and include zero, suggesting a high degree of uncertainty. Importantly, however, the share of the IE is calculated as the ratio between the IE and TE. Since both of these estimates are close to zero (relative to their standard deviations) and similar in magnitude, the size of the ratio is highly sensitive to sampling error, widening the CIs.

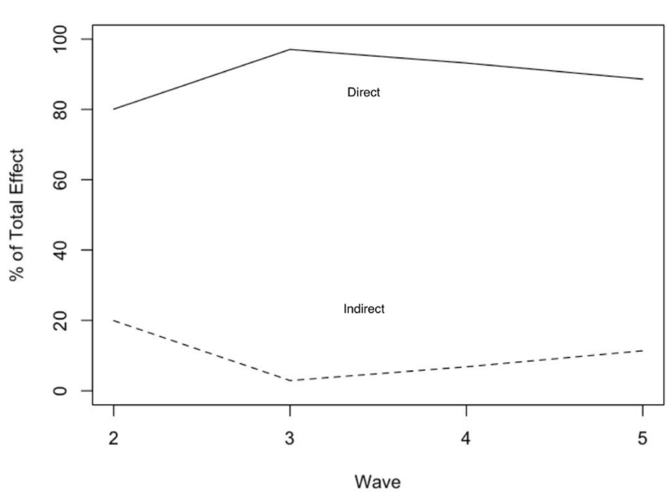

Fig. 4. Median Direct and Indirect Effects of Job Insecurity on Major Depression (as a Percentage of the Total Effect), operating via Sleep Disturbances.

Finally, Fig. 4 shows the percentage point change in the DE and IE (via sleep disturbances) of job insecurity on MD over the 10-year period (5 waves, 2 years apart). These results suggest that the majority of the decrease in MD happens in our counterfactual scenario. In other words, over the period of observation, the effect of job insecurity on MD is direct. However, job insecurity may very well operate via sleep disturbances, particularly if job insecurity at time k affects MD at time k via sleep problems at k. The assumptions underlying our model (shown in the causal DAG in Fig. 1), combined with a lack of information on the very precise timing of events, do not allow us to assess this possibility.

5. Discussion

This study extends knowledge on the relationship between job insecurity and MD (e.g., Magnusson Hanson et al., 2015) by adjusting for important sources of unobserved time-constant (FE models) and time-varying intermediate confounding (g-formula) in two separate, innovative and complementary causal modeling approaches. We further extend the literature by examining the previously unexplored question of whether sleep disturbances mediate this relationship, derived from prior research (Ferrie et al., 1998; Johnson et al., 2006; Virtanen et al., 2011; Baglioni and Riemann, 2012; Magnusson Hanson et al., 2020; Virtanen et al., 2011). To summarize, we address our first research question, "Is job insecurity associated with major depression (MD), accounting for unobserved time-constant and observed time-varying demographic and confounding factors?" in the first stage of our analysis. Findings consistently showed that the relationship between job insecurity and MD was robust to adjustments for demographic characteristics, observed time-varying, and unobserved time-constant confounding. As expected, job insecurity significantly increased sleep disturbances and sleep disturbances significantly increased the odds of MD by more than a factor of 3 (FE results). The regression results indeed suggested that sleep disturbances may mediate the association between job insecurity and MD.

The finding that job insecurity increases the odds of sleep disturbances may be the immediate (or primary) effect of the cognitive or emotional strain attributed to that fear (e.g., McEwen, 2000). Sleep disturbances in turn significantly predict MD, suggesting that the relationship between job insecurity and MD may be partially explained by sleep disturbances—all of which may be the product of chronic stress dysregulation (e.g., Mauss et al., 2015). Whether objective contractual agreements, co-worker discussions about future layoffs, workplace isolation, or neglect on the part of one's superior underlie fears of job loss, workers may frequently worry and ruminate about their future financial and productive lives (e.g., Berset et al., 2011). Such an intense burden could impact workers cognitively and emotionally, disturb sleep, and be severely debilitating psychologically.

In stage two of the analysis, we used a counterfactual causal inference approach with the g-formula to address the second research question "If we intervene on job insecurity, while also adjusting for intermediate confounding, what effect would this have on major depression at the population level?" Findings confirmed the results from stage one, suggesting that our hypothetical intervention on job insecurity—removing it at the population-level—decreased MD by about an average of 10%. The findings further suggest that the relationship between job insecurity and MD is primarily direct. However, we examined our third research question, "To what extent does the effect operate through sleep disturbances?" in the CF direct scenario and found that approximately 11% of the relationship operated via sleep disturbances, which we interpret with caution given the associated high degree of uncertainty.

The g-formula results were robust to adjusting for intermediate confounding due to the possibility that previous job insecurity, sleep disturbances, and time-varying confounders affected each other over time. For example, we adjusted for prior income and civil status affecting (or affected by) sleep disturbance or job insecurity in a previous two-year period. We further adjusted for prior MD given that participants with symptoms in 2010, for example, may have been at a greater risk of MD in 2012.

The RE and FE results were consistent with those from the g-formula. Risk ratios from the g-formula can be approximated with odds ratios (OR) when incidence of the outcome is small. Thus, we estimate the population attributable fractions (PAF) = 7.8% (with OR 2.7) and 4.5% (with OR 1.94), respectively.[3] Approximately 7.8% (or 4.5%) of the MD cases in the population were due to job insecurity. This is comparable to the estimated 10% reduction of cases with MD in the hypothetical g-formula intervention on job insecurity, so results were corroborated across approaches. Our findings were further consistent with Niedhammer et al. (2021) who estimated that 8.2% of depression in Sweden was attributed to job insecurity.

Overall, findings were consistent with prior studies showing a positive association between job insecurity and depression (e.g., Blackmore et al., 2007; Watson and Osberg, 2018). In particular, this study partially replicates and extends LaMontagne et al.'s (2021) RE and FE results, which showed a dose-response relationship between improvements in job insecurity and decreases in depression in Australia. Both studies, however, suffer from potential bias due to unobserved time-varying confounding. Still, the two studies were conducted in two distinct social, economic, and policy contexts, and jointly suggest a causal link between job insecurity and depression. We further extend this evidence by showing a reduction in MD with a hypothetical intervention on job insecurity that adjusts for intermediate confounding.

Along with all observational studies, this one includes limitations. First, the proportion with job insecurity (9%) in this study differed from prior research showing an average of 17% across European countries (Niedhammer et al., 2021). The difference may be attributed to measurement—job insecurity here was derived from three items and Niedhammer et al. relied on one item. A lack of a standardized measure of job insecurity may result in important differences in the estimated prevalence, which is an important issue for future research. Next, our FE models adjusted for potentially important unobserved time-constant individual characteristics that may affect job insecurity and MD (e.g., childhood experiences, born in another country), and the g-formula adjusted for intermediate time-varying confounding. However, unaccounted for time-varying confounders may still limit our study.

Macro and meso level ebbs and flows in unemployment, precarious work, and short-term contracts, along with job stressors like sexual harassment, may adversely affect workers regardless of whether or not their jobs are in jeopardy (Keim et al., 2014; Milner et al., 2014). Personal networks with disproportionately high unemployment could increase perceptions of job insecurity and affect one's mood or risk of MD. The same is true regarding contractual details of employment that change over time, particularly if contracts are frequently re-negotiated. Moreover, mild versus chronic job insecurity could have few or severe impacts on MD, but data did not allow us to examine these nuances. Overall, such time-varying factors that increasingly characterize contemporary jobs may confound the effects of job insecurity on MD.

Research further underscores the importance of a long history of perceived job insecurity in psychological distress (Burgard and Seeyle, 2017). Our study spans 10 years, or a full decade of observations that help to establish temporal ordering of exposures, mediators, and outcomes—a necessary requirement to assess causal pathways and relationships (Hill, 1965; Rothman and Greenland, 2005). Even so, it is possible that some workers in our sample were exposed to job insecurity for more than a decade, which may have affected them more or less severely. Burgard and Seelye (2017), for example, argue that consistent exposure over a long period either has few psychological effects because workers find ways to cope and adjust; or, workers are worse off psychologically due to cumulative adverse effects of prolonged exposure. In either case, 10-years of observations do not allow us to account for these potential differences. On the other hand, temporary or periodic exposure to job insecurity may increase the risk of MD in important, disruptive, and lasting ways. For example, if one starts to feel that previously secure employment is no longer secure, this could arouse a fear that may be severely debilitating psychologically.

Next, it is possible that important events associated with job insecurity, sleep disturbances, and MD occur between SLOSH waves. The 2-year time lag may mean that short-term worries, rumination, and/or ability to wind down at the end of a work day (e.g., Berset et al., 2011) are not captured in our models. If the lag time in our data does not correspond with the natural course of these phenomena, our causal interpretations may be affected. Moreover, given the average age of 48 in our sample, the DE and IE may be underestimated if job security improves with age. Where data are available, future research would benefit from using a similar causal approach for younger workers.

Finally, we cannot rule out the possibility of common method bias as job insecurity and MD are self-rated. However, we use repeated measures over time which are preferable to cross-sectional data for estimating causal relationships. Our g-formula model also included lagged MD on job insecurity, which somewhat adjusts for the possibility of reverse causality. Overall, despite limitations, this study makes three important contributions to the literature: 1) it uses innovative methodological techniques to address important sources of potential bias from unobserved heterogeneity and confounding in estimating the relationship between job insecurity and MD; 2) it methodologically "triangulates" or corroborates results, strengthening causal interpretations; and 3) it assesses sleep disturbances as a potential mediator. This study further highlights the possible severe consequences of job insecurity and emphasizes a need to better understand and address it. Policies aimed at prevention—e.g., limiting temporary contracts and promoting communication and support from employers to workers—may help to reduce job insecurity and associated risks of MD.

CRediT authorship contribution statement

Robin S. Högnäs: Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing. **Maarten J. Bijlsma:** Methodology, Formal analysis, Writing – review & editing. **Ulf Högnäs:** Conceptualization, Formal analysis, Writing – original draft. **Sandra Blomqvist:** Conceptualization, Writing – review & editing. **Hugo Westerlund:** Conceptualization. **Linda Magnusson Hanson:** Conceptualization, Methodology, Writing – review & editing.

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[3] The approximations are calculated as $(0.05^*(2.7–1))/(0.05^*(2.7–1)+1) = 0.078$ and $(0.05^*(1.94–1))/(0.05^*(1.94–1)+1) = 0.045$.

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