



Full-length Article

Unemployment and inflammatory markers in England, Wales and Scotland, 1998–2012: Meta-analysis of results from 12 studies

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ABSTRACT

Introduction: Unemployment represents for many affected individuals a substantial source of psychosocial stress, and is linked to both increased risk of morbidity and mortality and adverse health-related behaviours. Few studies have examined the association of unemployment with systemic inflammation, a plausible mediator of the associations of psychosocial stress and health, and results are mixed and context dependent. This study examines the association of unemployment with C-reactive protein (CRP) and fibrinogen, two markers of systemic inflammation.

Methods: A random-effects meta-analysis was performed using a multilevel modelling approach, including 12 national UK surveys of working-age participants in which CRP and fibrinogen were measured between 1998 and 2012 (N = 30,037 economically active participants). The moderating impact of participant age and UK country was explored.

Results: CRP and fibrinogen were elevated in unemployed compared to employed participants; jobseekers were also more likely (Odds Ratio: 1.39, $p < 0.001$) to have CRP levels corresponding to high cardiovascular risk (>3 mg/L), after adjustment for age, gender, education, long-term illness, smoking, and body mass index. Associations were not explained by mental health. Associations peaked in middle-age, and were stronger in Scotland and Wales than in England.

Conclusions: Our study demonstrates that systemic inflammation is associated with an important but little-studied aspect of the social environment, as it is elevated in unemployed compared to employed survey participants. Modifications suggest the association of unemployment and inflammation is substantially influenced by contextual factors, and may be especially strong in Wales, where further investigation of this relationship is needed.

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1. Introduction

There is an established association of stressful experiences with systemic inflammation. Laboratory studies involving both animals (Zhou et al., 1993) and humans (Maes et al., 1998, 1999) have shown production of pro-inflammatory cytokines can be directly stimulated by psychologically stressful experiences (Kiecolt-Glaser et al., 2003), while in human observational studies an elevated inflammatory state has been associated with ‘stressful life events’ including bereavement (Buckley et al., 2012) and caregiving (Kiecolt-Glaser et al., 2003). More generally, markers of systemic inflammation are associated with disadvantaged socioeconomic position, a finding attributed both to psychosocial stress associated with socioeconomic disadvantage, and to social

patterning of inflammation-related health behaviours (Gimeno et al., 2008; McDade et al., 2011; Marmot et al., 2008; Jones et al., 2015). Unemployment is hypothesised to represent a substantial source of stress firstly through its financial impact, and secondly due to loss of non-financial benefits of employment, for example its role in supporting status and personal identity (Jahoda, 1981). An influence of unemployment on systemic inflammation through psychosocial stress might therefore be expected. Since systemic inflammation is an established risk factor for cardiovascular disease (Yudkin et al., 2000; Ridker, 2003) and also associated with depression (Dowlati et al., 2010), such a pathway could also help to explain the elevated risk of both psychological morbidity (Paul and Moser, 2009) and mortality (Roelfs et al., 2011) among jobseekers.

However, any such associations must be shown to be independent of confounding by demographic, and health-related factors. Socioeconomic background may confound unemployment-health associations, because unemployment disproportionately affects

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more disadvantaged individuals (Montgomery et al., 1996). Since systemic inflammation is higher in women than men and increases with age (Krabbe et al., 2004), gender and age could either confound or suppress unemployment-inflammation associations, depending on the distribution of unemployment in a population. Diverse chronic illnesses including cancer (Bower, 2014), Parkinson's disease (Friedman et al., 2007), diabetes (Lasselin et al., 2012) and major depression (Dowlati et al., 2010) are associated with inflammation, and could therefore confound unemployment-inflammation associations if they also impact on employability.

Finally, differences in inflammation-related health behaviours could contribute to elevations in inflammatory markers among jobseekers. Of particular importance are smoking and adiposity: smoking increases inflammatory activity by augmenting production of pro-inflammatory cytokines (Arnson et al., 2010), while adiposity is an important determinant of systemic inflammation because key pro-inflammatory cytokines are made in adipose tissue (Lyon et al., 2003). Physical activity appears to have direct and immediate anti-inflammatory effects in addition to longer-lasting ones mediated through adiposity (Gleeson et al., 2011), while the relationship of alcohol appears U-shaped, with anti-inflammatory effects for moderate consumption (Pai et al., 2006; Raum et al., 2007). Thus, inflammation-related health behaviours could confound unemployment-inflammation associations, if smokers or heavier people are more likely to become unemployed or to stay unemployed for longer (Puhl and Heuer, 2009), but could also act as mediators if unemployment triggers changes health-related behaviours, a possibility which has been called the 'coping hypothesis' (Roelfs et al., 2011). This is plausible for systemic inflammation, given evidence that unemployment may alter both smoking (Marcus, 2014) and adiposity (Monsivais et al., 2015; Deb et al., 2011; Hughes and Kumari, 2017).

To date, only a handful of studies have investigated inflammatory markers in relation to unemployment. These used data from Finland, the US and the UK, and all report elevations in inflammatory markers for currently or recently unemployed participants (Hintikka et al., 2009; Janicki-Deverts et al., 2008; Hughes et al., 2015). However, these associations appear to be area specific and require further investigation. The largest study used English and Scottish data and found elevations in the inflammatory markers C-reactive protein (CRP) and fibrinogen after taking account of age, gender, occupational social class, housing tenure, smoking, body mass index and alcohol consumption, consistent with an influence of unemployment on inflammation which is independent of social background, chronic illness and health behaviours. Elevations were more apparent among older jobseekers and in Scotland (Hughes et al., 2015), where unemployment had been higher at the time of data collection, suggesting moderation by contextual effects. This paper seeks to extend these analyses: an individual-participant data meta-analysis is presented of 12 national studies, incorporating data collected between 1998 and 2012 from all countries in Great Britain. To isolate a possible direct influence of unemployment on inflammation via psychosocial stress, we investigate jobseekers' inflammatory markers after accounting for a wide range of possible confounders including age, gender, education, long-term illness, mental health, BMI, and multiple dimensions of smoking and alcohol consumption.

2. Methods

2.1. Participants

This meta-analysis aims to be as comprehensive and representative as possible for a UK-based analysis. It therefore incorporates all usable observations from national English, Scottish or Welsh

surveys to date which included working-age populations and measured C-reactive protein and fibrinogen from blood samples taken from the general population sample (as opposed to, for example, only an ethnic minority boost sample). No such data was available from Northern Ireland. Hence, we include data from the Health Survey for England (HSE), the Scottish Health Survey (SHeS), the National Child Development Study (NCDS) and *Understanding Society: The UK Household Longitudinal Survey* (UKHLS). The HSE (Mindell et al., 2012) and SHeS (Gray et al., 2010) are annually repeated cross-sectional surveys, comprising a new sample each year. Detailed sociodemographic and health-related information is collected annually; blood samples were collected from adult participants in the general population sample at HSE 1998, 2003, 2006, 2009 and SHeS 2003, 2008, 2009, 2010 and 2011, which are included in the current analysis. HSE 1999 was not included as the biomedical component of this survey was focused specifically on the health of certain ethnic minority groups, and as such its CRP and fibrinogen measurements are not representative of the English population at the time. The NCDS is a longitudinal birth cohort study, which began as a study of over 17,000 children born in England, Wales and Scotland in one week in 1958 (Power and Elliott, 2006). Sociodemographic information was collected at ages 7, 11, 16, 23, 33, 42, 50 and 55. When cohort members were aged 44–45, blood samples and anthropometric measurements were collected at a biomedical visit involving 9377 remaining participants. UKHLS is a longitudinal nationally-representative UK study of over 40,000 households which began in 2009 (Knies, 2015). Sociodemographic information is collected annually from the same participants, and in 2010–12, 13,258 adult participants gave blood samples at an additional biomedical visit (Benzeval et al., 2014).

The initial sample included men and women aged 22–64 (of working age but likely to have finished full-time education) and in the active labour force, i.e. currently employed or currently unemployed and seeking work. Participants not working due to sickness or disability, retired people, full-time students, and otherwise economically inactive participants were excluded. Participants whose CRP exceeded 10 mg/L were excluded, since this is considered to reflect current or recent infection rather than chronic processes (Pearson et al., 2003). In HSE and SHeS surveys fibrinogen was not measured for participants taking statins, fibrates or beta-blockers, so for consistency NCDS and UKHLS participants known to be taking these medications were excluded from fibrinogen analyses. The initial sample therefore numbered 52,117 for CRP and 49,747 for fibrinogen. Of these, due primarily to lack of consent for blood samples, 32,259 (62.0%) had usable CRP measurements and 30,069 (60.4%) had usable fibrinogen measurements. Further exclusions for missing employment status and covariates resulted in combined complete-case sample size of 30,037 for CRP analyses and 28,661 for fibrinogen analyses.

2.2. Measures

2.2.1. Inflammatory markers

In HSE and SHeS surveys, serum CRP concentrations were analysed by the Biochemistry Department of the Royal Victoria Infirmary, Newcastle (RVI), using the N Latex CRP mono Immunoassay on the Behring Nephelometer II Analyser (Ingle et al., 2013) until SHeS 2011 when a Roche Modular P analyser was introduced (Bradshaw et al., 2012). Fibrinogen was analysed at the RVI Haematology Department on the Organon Teknika MDA 180 analyser until HSE 2006 (Bajekal et al., 1999, 2001; Blake et al., 2004; Bromley et al., 2005) and subsequently the Auto Coagulation lab (TOP) CTS analyser (Corbett et al., 2009, 2010; Aresu et al., 2011; Bromley et al., 2011; Ali et al., 2008). In NCDS, CRP was measured on citrated plasma by high-sensitivity nephelometric analysis of latex particles coated with CRP-monoclonal antibodies on the BN ProSpec protein

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