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Review article

A systematic review and meta-analysis of the effort-reward imbalance model of workplace stress with indicators of immune function☆

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ABSTRACT

Objective: Despite considerable research into associations between the effort reward imbalance (ERI) model and various health outcomes over the past 20 years, the underlying mechanisms responsible for the association remain unclear. Recently, ERI investigations have examined associations with immune sub-systems (e.g., leukocytes, cytokines and immunoglobulins). Synthesis of the amalgamated research evidence will aid clarity to this field of enquiry. We conducted a meta-analysis and reviewed the associations of ERI and over-commitment (OC) in the workplace with immunity.
Method: Electronic databases were searched with the phrase 'effort reward imbalance' which initially yielded 319 studies leading to 57 full text studies being screened. Seven studies that met inclusion criteria were combined using mixed and random effects models.
Results: Greater ERI was associated with lower immunity ($r = -0.09$, CI -0.14, -0.05, $p < 0.001$). Sub-group analyses revealed the effect with mucosal immunity was stronger ($r = -0.33$, CI -0.47 to -0.18) than trends between both cytokine ($r = -0.04$, CI -0.07, -0.01) and leukocyte sub-groups ($r = -0.02$ CI -0.04, 0.01) respectively ($k = 7$, $N = 9952$). Over-commitment was also associated with lower immunity ($r = -0.05$, CI -0.09, 0.01, $p = 0.014$); subgroup (leukocytes, cytokines, mucosal immunity) associations, however, were homogenous ($Q = 1.83$, $df = 2$, $p = 0.400$, $k = 6$, $N = 2358$).
Conclusions: Greater ERI and OC were both associated with lower immunity. The association between mucosal immunity and ERI was stronger than the cytokine and leukocyte sub-groups. OC moderated the relationship between ERI and immunity.

1. Introduction

The effort reward imbalance (ERI) workplace stress model [1] has substantive predictive associations with behavioural outcomes (such as absenteeism, smoking, alcohol) [2], depression [3,4], cardiovascular disease [4], and self-reported health [2,5]. Despite over 20 years of assessing the ERI model with health outcomes, the mechanisms by which ERI relates to manifest ill-health are poorly understood. In recent years, growing emphasis has been accorded to studies of the ERI model with physiological indicators of ill-health, including immune markers. This paper will review and synthesise the evidence examining the association between ERI model elements and immunity.

The ERI model is based on the concept of social reciprocity where perceived efforts at work should confer adequate rewards [1]. Perceived efforts refer to the physical, emotional and psychological demands of the work one does. The rewards component refers to money, esteem, and promotion prospects/job security. The imbalance between effort and reward is expressed as a ratio with higher ERI scores representing an imbalance between a worker's perceived efforts and rewards, which is theorised to increase risk of ill-health outcomes [6]. A further component, over-commitment (OC), is a cognitive style/disposition that refers to the worker's inability to withdraw from work.

The model is set around 3 main hypotheses. The first suggests that all three components of the model (efforts, rewards and OC) will be individually associated with the health outcome. Secondly, the ERI ratio, or efforts compared to rewards, is expected to better predict outcomes than either efforts or rewards alone. And thirdly, OC is expected to moderate the association between ERI and ill-health outcomes [7]. Hypotheses two and three of the ERI model will be the focus of this meta-

Abbreviations: CD4, T-helper cells; CRP, c-reactive protein; ERI, effort reward imbalance; IFN, interferon; IL, interleukin; NKC, natural killer cell; NKCC, natural killer cell cytotoxicity; OC, over commitment; sIgA, salivary immunoglobulin A; TNF, tumour necrosis factor; HDL, High Density Lipoproteins; LDL, Low Density Lipoproteins; PSS, Perceived Stress Scale.

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analysis as they have received the most attention empirically and, are considered to have stronger associations with ill-health than hypothesis 1 [8].

The OC component of the ERI model has evolved over time. Originally referred to as 'need for control' and depicted as a work-related Type A behaviour, it reflected two factors, vigor and immersion (consisting of: need for approval, competitiveness, disproportionate irritability and inability to withdraw from work). Subsequent research found that the 'inability to withdraw from work' component was the primary factor followed by disproportionate irritability, OC was subsequently reconceptualised to reflect this. The evolution of OC involves the factor now being considered an independent concept alongside efforts and rewards [2].

Meta-analytic associations between chronic stress and immune markers have shown the potential of chronic stress to affect the flexibility and balance within the immune system. These effects may contribute to ill-health outcomes and warrant greater empirical consideration. Herbert and Cohen's [9] meta-analysis of stress and immunity included long-term naturalistic stressors ($k = 8$, $N = 1289$) and found a negative association with number of circulating lymphocytes (B-cells $r = -0.33$ $p < 0.001$, T-cells $r = -0.40$ $p < 0.001$, T helper cells $r = -0.24$ $p < 0.01$, T cytotoxic cells $r = -0.67$ $p < 0.001$), lymphocyte proliferation to both concanavalin A ($r = -0.19$, $p < 0.01$) and phytohemagglutinin ($r = -0.25$, $p < 0.001$), whereas a positive relationship was observed with the helper: suppressor ratio ($r = 0.17$ $p < 0.01$). Segerstrom and Miller's [10] meta-analysis of stress and immunity also included chronic stress and they report that chronic stress (dementia caregiver, unemployment or living with a handicap) was negatively associated with both specific and innate immunity equally across genders. Their meta-analysis of chronic stress studies ($k = 31$, $N = 9075$), included leukocyte subset counts ranging from natural killer cells $r = -0.14$ to neutrophils $r = 0.36$ and leukocyte subset percentages ranging from T lymphocytes $r = -0.03$ to monocytes $r = 0.08$. Surprisingly, despite the overall association attained from the meta-analysis of all measures with chronic stress states, none of the individual measures in isolation, were significantly associated with chronic stress. Segerstrom and Miller [10] did not assess sex as a moderator of the chronic stress–immunity relationship but they did recommend these analyses in future studies. Consistent with Herbert and Cohen's [9] meta-analysis, no study assessed the relationship of innate immunoglobulins with chronic stress. However, specific immunoglobulins in the form of antibody to herpes simplex 1 and antibody to influenza after vaccination were included and had medium ($r = 0.44$ $p = 0.17$) and small ($r = -0.22$ $p = 0.001$) relationships respectively with chronic stress. In addition to the influenza antibody, other immune measures that were included and significantly associated with chronic stress were natural killer cell cytotoxicity ($r = -0.12$, $p = 0.04$), lymphocyte proliferation to concanavalin A ($r = -0.13$, $p = 0.02$), lymphocyte proliferation to phytohemagglutinin ($r = -0.16$, $p = 0.004$) and interleukin 2 (IL-2) ($r = -0.21$, $p = 0.001$).

While these meta-analyses [9,10] advance the field by identifying associations between acute, distant, and chronic forms of stress with sub-groups of immune and inflammatory markers, they collapsed diverse stress states such as bereavement, unemployment, and caregiving into a single category of 'chronic stress'. More recently, Nakata's [11] systematic review ($N = 56$) of various psychosocial job stress measures (high job demands, low job control, high job strain, job dissatisfaction, high ERI, OC, burnout, unemployment, organisational downsizing and economic recession) and immune parameters (blood, urine and saliva) found job stress exposure measurably decreased natural killer cell activity, natural killer cell numbers, T cell subsets and CD4+/CD8+ ratios and increased inflammatory markers when exposed to job stress. Building on this approach of specifically assessing the effects of occupational stress upon immunity, we aimed to sharpen the focus and precision of such research by assessing indicators of immunity in the specific context of chronic work stress as assessed by the ERI model. Although the use of prospective research designs have demonstrated an association between ERI and various health outcomes [2], the underlying mechanisms responsible for this association remain unclear. Workplace models such as the ERI can identify the situational and individual antecedents to reduced immunity. Advances in this area may help identify precursors of ill-health and promote evidence based intervention [2]. We will nonetheless compare the findings from the investigations of chronic states and immunity in the ERI literature with the chronic stress and immunity studies reported in the Herbert and Cohen [9] and Segerstrom and Miller [10] meta-analyses.

The aim of this meta-analysis is to summarise and clarify what is currently understood regarding ERI and OC and its association upon immunity, specifically focussing on hypotheses two and three of the ERI model. We will identify which physiological indices of the immune system have the strongest association with ERI and OC and additionally, assess if associations with ERI and OC are better explained according to sub-groups or singular indicators within the immune system. Finally, as ERI and OC relate to perceived external and internal factors respectively, we will assess if ERI and OC differ in their strength of association with these immune markers, and if either OC or sex moderates the link between ERI and the immune indices.

2. Methods

2.1. Procedure

Study selection was based on the PRISMA guidelines [12]. The following databases were used to search for potential studies for inclusion: PsychINFO (319 articles identified), MEDLINE 1996 – (OVID) (530 articles identified), PubMed (372 articles identified), PsychARTICLES (28 articles identified), ProQuest Psychology Journals (151 articles identified), JSTOR (209 articles identified), Science Direct (514 articles identified) and Google Scholar (117 articles identified). Studies incorporating the ERI model were identified using the phrase 'effort* reward* imbalance*'. Duplicates were identified and removed to allow abstracts to be screened in accordance with exclusion criteria (Table 1). Exclusion criteria are listed in order of priority whereby a single failed exclusion criterion is sufficient for a study to be excluded, eliminating the need for subsequent criteria to be considered. A final search was conducted in May 2016 to ensure research published since the initial search was included if eligible (5 articles identified, 1 eligible).

Full text articles were obtained and screened for eligibility, and the reference list of included articles were then checked for any additional studies that met the search criteria (13 articles identified). Studies that only reported effect sizes for response to acute stress and could not provide chronic stress or baseline responses were excluded. For studies that did not report a measure of effect, or information to

Table 1
Exclusion criteria for study inclusion in meta-analysis.

Exclusion criteria
1. The study was not available in English
2. The study contains no original data
3. The study did not utilise the ERI model
4. The study did not measure the ERI ratio of the ERI model.
5. The study did not include a physiological indicator of immune system functioning as an outcome measure of effect
6. The study could only provide a measure of effect in response to acute stress
7. The study did not provide an effect size measure and the corresponding author of the study could not provide the requested effect size measure or information allowing its calculation
8. The study was not published in a peer reviewed journal
9. The study could not be retrieved
10. The study discusses purely descriptive, theoretical or psychometric matters on the ERI model
11. The paper is a presentation, personal communications, unpublished paper or dissertation.