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Study Protocol

Relationship between Burnout, Cardiovascular Risk Factors, and Inflammatory Markers: A Protocol for Scoping Review

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Abstract: Background: Burnout is increasingly being recognized as a contributory factor to the erosion of a positive psychological state. Studies have examined the relationship between burnout and various inflammatory markers such as IL-1, IL-6, and TNF-alpha. Burnout is also associated with increased systemic inflammation along a continuum of symptom severity. This protocol is for a scoping review looking at the link between burnout, inflammatory markers, and cardiovascular risks or diseases. Methods: This study will be based on the preferred reporting items for systematic reviews and meta-analyses guidelines/checklists and the report of the review will be based on the same guideline. The study seeks to address the following principal questions. (i) What are the relevant inflammatory biomarkers that mediate cardiovascular risk factors in burnout? (ii) How do inflammatory biomarkers mediate cardiovascular risk factors in burnout? The outputs obtained from the literature search will be deduplicated using the Rayyan software. Results: We would create table summaries of findings to inform a narrative synthesis of the evidence from the papers included. Conclusion: The review article would help to concisely synthesize the available evidence on the relationship between burnout, inflammatory markers, and cardiovascular diseases.

Keywords: occupational burnout; cardiovascular diseases; cytokines; vital exhaustion

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1. Introduction

1.1. Background

Freudenberger defined burnout as "a state of mental and physical exhaustion brought on by one's professional life" in 1974 [1]. Whereas, in the International Classification of Diseases (ICD-11) 11th revision, it was defined as a syndrome resulting from "chronic workplace stress that has not been successfully managed" [2]. It is a state of mental weariness that describes the disconnect between the worker and the workplace due to consistent stressful demands in the latter [3]. It is also a type of chronic stress that causes physical exhaustion, emotional exhaustion, and cognitive fatigue [1,4]. In the last fifty years, burnout is increasingly being recognized as a strong contributory factor for the erosion of a positive psychological state [5]. While it is also a psychological construct like depression and anxiety, it does not overlap with these related affective dysfunctions [6]. There are three core dimensions as proposed by Malash which include (i) emotional exhaustion (refers to

feelings of depleted emotional resources), (ii) depersonalization (refers to negative, cynical, or excessively detached responses to other people at work), and (iii) reduced personal accomplishment (refers to feelings of decline in competence and productivity and to a lowered sense of self-efficacy) [6,7]. These dimensions can also be described as the cardinal symptom of burnout. Other considered dimensions are (i) physical fatigue, (ii) emotional exhaustion (EE), and (iii) cognitive weariness [8,9]. Notwithstanding, central to all of these dissimilarities is exhaustion as the main component.

1.2. Prevalence and Predisposing Factors of Occupational Burnout

There is a high degree of burnout and other psychosocial stressors among many occupational groups such as white-collar (civil servants), blue-collar (manual workers), and the "helping" professions (health care workers, caregivers, and teachers) globally [4,10–14]. Estimates for general surgery residents in the US ranged from 3.2% to 91.4% according to a national study [13,14]. An estimated burden of between 23.6 and 51.7% was found in a systematic review of articles among Nigerian physicians. The prevalence rate of overall burnout was calculated to be 23.9% based on a pool study of 7 studies and 1101 physical education teachers in another study [15].

This high observed burden is not unconnected with their continuous and prolonged exposure to stress, particularly work-related stress [6]. Several factors, including situational factors such as job demands and resources, predispose many working populations to burnout. High job demands cause fatigue and psychological distancing from the jobs [16]. Other important factors that contribute to burnout in many of these populations include the presence of ambiguity, conflict, stress, workload, tension, and the complexity of the work environment [16–20]. Some stressful environments are also competitive and have a high component of a continuous training process, thereby leading to an unhealthy lifestyle, especially sleep deprivation, and predisposing people to cognitive impairment and emotional fragility [20]. Other factors are perfectionism in many work environments [19]. Generally, long-term job stress can cause burnout, persistent weariness, anxiety symptoms, and health problems [21].

1.3. Effect of Burnout on Cardiovascular Health

Until recently, researchers studying burnout focused on the attitudinal, interpersonal, and organizational consequences of burnout [6]. However, growing evidence has pointed to burnout's negative effects on physical health and well-being over the last decade [8,9]. Occupational burnout can cause persistent fatigue symptoms as well as an increased risk of cardiovascular disease [8,21,22]. There is also evidence that suggests stressors quickly and strongly put people at risk for increased risk of cardiovascular (CV) diseases, CV risk factors, and cardiovascular-related events [5,6,8,21].

Burnout is an independent risk factor for future and recurrent incidences of coronary heart disease [21]. Since the first systematic research study to unravel the effect of burnout (vital exhaustion) on cardiovascular morbidity (myocardial infarction) independent of the classic risk factors by Appel et al. between the late 1980s and 1990s, there have been more insights on more CV outcomes [6]. Other identified CV-related events are recurrent myocardial infarction [MI], coronary bypass surgery, percutaneous transluminal coronary angioplasty [PTCA], repeat PTCA, increase in coronary atherosclerosis, coronary stenosis and restenosis, and cardiac death [6]. Other CV diseases studied by other groups included the impact of the syndrome on stroke outcome and sudden cardiac death [6]. While other studies have also identified associations between the syndrome and various CV risk factors such as dyslipidaemia, dysglycaemia, high alcohol intake, reduced physical activity, and metabolic syndrome [6,23,24].

Postulated pathways include the interplay of metabolic syndrome, hypothalamic–pituitary–adrenal (HPA) axis dysregulation, inflammation, immunity, sleep disturbances, blood coagulation and fibrinolysis, and poor health behaviors such as low physical activity level and increased alcohol intake [6].

1.4. Burnout, Inflammatory Markers, and Cardiovascular Diseases

Certain inflammatory markers such as interleukin (IL-1), interleukin-6 (IL-6), and TNF-alpha have been noted to mediate burnout and cardiovascular morbidities in a few studies [9,25]. Pockets of studies have examined the relationship between burnout and various inflammatory makers which, by extension, are also implicated in CV diseases [6,25]. There appears to be some consistency in the few available evidence of the implication of the first two of these inflammatory markers; however, there are still some disparities, especially with TNF-α [25]. Furthermore, changes with burnout are not merely absolute values but some ratios of these inflammatory markers like TNF-α/IL-4 ratio and TNFα/IL-10 ratios appear to be relevant [25]. Each inflammatory markers are also differentially studied compared to others [25].

Burnout was associated with increased systemic inflammation along a continuum of symptom severity rather than categorically; since low-grade systemic inflammation promotes atherosclerosis, this may explain the increased cardiovascular risk in burned-out individuals [9,25]. There are also some observed gender differences in inflammatory responses, with females with burnout appearing to have enhanced inflammatory responses and oxidative stress [9,25].

Overall, there is wide inconsistency in the findings of which biomarkers have any relationship between burnout and cardiovascular diseases [25]. Thus, no conclusion can be drawn regarding a plausible relationship between burnout and cytokine/biomarker levels, partly due to a lack of adequate studies but primarily due to discrepancies among existing studies [25]. However, it would be interesting to exhaustively identify all inflammatory markers that have been noted in the literature to be involved in this interplay. In addition to exploring the consistency of the findings for each of the biomarkers and the pattern.

There is a dearth of studies that have been conducted around the world synthesizing the link between burnout, stress at work, inflammatory markers, and cardiovascular risks or diseases. Therefore, this scoping review would provide a great opportunity to synthesize all available evidence.

2. Methods

2.1. Review Design

The design of this study will be based on the preferred reporting items for systematic reviews and meta-analyses guideline/checklist [26]. The report of this review would be based on the same guidelines (Table S1).

2.2. Review Questions

This study seeks to address the following principal questions. (i) What are the relevant inflammatory biomarkers that mediate cardiovascular risk factors in burnout? (ii) How do the inflammatory biomarkers mediate cardiovascular risk factors in burnout?

2.3. Literature Selection Criteria

The inclusion or exclusion of the literature into this review will be based on the criteria listed below:

2.3.1. Inclusion Criteria

1. All peer-reviewed original articles investigating the relationship between burnout, CV diseases, CV risk factors, and inflammatory markers among humans;
2. Articles in which their full texts are accessible and in English language;
3. Relevant articles published between 1 January 1990 and 31 December 2022.

2.3.2. Exclusion Criteria

All peer-reviewed original articles that investigated the relationship between burnout, cardiovascular risk factors, and inflammatory markers among non-humans.

2.4. Literature Search Strategy

The literature search will be based on the PCC framework which is made up of population [p], concept [c], and context [C]. The following databases would be searched: APA PsycInfo, PubMed, SCOPUS, and CINAHL (Tables 1–4, Figure S1). The search item would include (i) burnout, (ii) inflammatory markers, (iii) cardiovascular diseases, and (iv) cardiovascular risk factors. The combination of relevant search terms aided by Boolean operators and truncations and without year limiters will be used to retrieve relevant literature published in the duration of the inclusion criteria.

Table 1. Search string for the PubMed database search.

Tag	Subject Search	Search String
#1	Burnout	(“burnout s”[All Fields] OR “burnout, psychological”[MeSH Terms] OR (“burnout”[All Fields] AND “psychological”[All Fields]) OR “psychological burnout”[All Fields] OR “burnout”[All Fields] OR “burnouts”[All Fields]) AND ((clinicalstudy[Filter] OR clinicaltrial[Filter] OR observationalstudy[Filter] OR randomizedcontrolledtrial[Filter]) AND (fft[Filter]) AND (humans[Filter]) AND (1990/1/1:2022/12/31[pdat]) AND (english[Filter]))
#2	Inflammatory markers	((“inflammatories”[All Fields] OR “inflammatory”[All Fields]) AND (“marker”[All Fields] OR “markers”[All Fields])) AND ((clinicalstudy[Filter] OR clinicaltrial[Filter] OR observationalstudy[Filter] OR randomizedcontrolledtrial[Filter]) AND (fft[Filter]) AND (humans[Filter]) AND (1990/1/1:2022/12/31[pdat]) AND (english[Filter]))
#3	Cardiovascular diseases	(“cardiovascular diseases”[MeSH Terms] OR (“cardiovascular”[All Fields] AND “diseases”[All Fields]) OR “cardiovascular diseases”[All Fields]) AND ((clinicalstudy[Filter] OR clinicaltrial[Filter] OR observationalstudy[Filter] OR randomizedcontrolledtrial[Filter]) AND (fft[Filter]) AND (humans[Filter]) AND (1990/1/1:2022/12/31[pdat]) AND (english[Filter]))
#4	Cardiovascular risk factors	(“heart disease risk factors”[MeSH Terms] OR (“heart”[All Fields] AND “disease”[All Fields] AND “risk”[All Fields] AND “factors”[All Fields]) OR “heart disease risk factors”[All Fields] OR (“cardiovascular”[All Fields] AND “risk”[All Fields] AND “factors”[All Fields]) OR “cardiovascular risk factors”[All Fields]) AND ((clinicalstudy[Filter] OR clinicaltrial[Filter] OR observationalstudy[Filter] OR randomizedcontrolledtrial[Filter]) AND (fft[Filter]) AND (humans[Filter]) AND (1990/1/1:2022/12/31[pdat]) AND (english[Filter]))
#1 AND #2 AND #3 AND #4		

Table 2. Search string for the SCOPUS database search.

Tag	Subject Search	
S1	Burnout	(TITLE-ABS-KEY [Burnout] OR TITLE-ABS-KEY [“Burn out”])
S2	Inflammatory markers	(TITLE-ABS-KEY [Inflammatory] OR TITLE-ABS-KEY [“inflammatory marker”] OR TITLE-ABS-KEY [biomaker] OR TITLE-ABS-KEY [“molecular marker”])
S3	Cardiovascular diseases	(TITLE-ABS-KEY [cardiovascular] OR TITLE-ABS-KEY [cardiac] OR TITLE-ABS-KEY [vascular] OR TITLE-ABS-KEY [“circulatory”])
S4	Cardiovascular risk factors	(TITLE-ABS-KEY [“cardiovascular risk factors”] OR TITLE-ABS-KEY [“heart disease risk factor”] OR TITLE-ABS-KEY [“vascular disease risk factor”])
S1 AND S2 AND S3 AND S4		

2.5. Deduplication of the Literature

The outputs obtained from the literature search will be deduplicated using the Rayyan software, Rayyan System Inc. Cambridge, MA, USA.

2.6. Literature Screening and Selection

All deduplicated literature will be screened using the Rayyan software based on the established selection criteria. The screening process will be two-staged with at least three independent reviewers who are experienced researchers, particularly those with a theme

involved: two reviewers will screen all the deduplicated literature while the third reviewer, who is more experienced, will resolve any conflicts in the screening decisions made by the other two reviewers as a consensus. The first stage will consist of a title and abstract screening while the second stage will consist of full-text screening. This scoping review will include only the literature that met the inclusion criteria.

Table 3. Search string for the CINAHL database search (via EBSCOHost interface).

Tag	Subject Search	
#1	Burnout	AB burnout OR AB burn out
#2	Inflammatory markers	AB inflammatory OR AB inflammatory marker OR AB biomarker OR AB molecular marker
#3	Cardiovascular diseases	AB cardiovascular OR AB cardiac OR AB vascular OR AB circulatory
#4	Cardiovascular risk factors	AB cardiovascular risk factors OR AB heart disease risk factor OR AB vascular disease risk factor
#1 AND #2 AND #3 AND #4		

Table 4. Search string for the APA PsycInfo search (via EBSCOHost interface).

Tag	Subject Search	
#1	Burnout	AB burnout OR AB burn out
#2	Inflammatory markers	AB inflammatory OR AB inflammatory marker OR AB biomarker OR AB molecular marker
#3	Cardiovascular diseases	AB cardiovascular OR AB cardiac OR AB vascular OR AB circulatory
#4	Cardiovascular risk factors	AB cardiovascular risk factors OR AB heart disease risk factor OR AB vascular disease risk factor
#1 AND #2 AND #3 AND #4		

2.7. Data Extraction, Data Summary, and Data Analyses

The titles of relevant documents identified through a database search would be itemized in Microsoft Excel after which duplicates would be removed and the abstracts of the remaining titles would be screened to determine if they meet the eligibility requirements. The remaining full articles of selected articles would be read to confirm the eligibility. Two independent reviewers would read each final article and extract data into the templates in Tables 5 and 6. We would create table summaries of findings to inform a narrative synthesis of the papers included. The template shown in Table 1 would be used for data extraction. We would assess the heterogeneity by visually inspecting forest plots and quantitatively using a funnel plot; we use a funnel plot to assess potential publication bias.

Table 5. List of the literature considered for full-text screening and the screening outcomes.

Serial Number	References	Outcomes

Table 6. Summary of the reviewed publications template.

Serial Number	Authors	Year of Publication	Sample Size	Country of Study	Study Design	Work Population	Burnout Measure Used	Biomarker Assessed	CVD/CV Risk Factors Assessed	Major Findings

2.8. Sensitivity Analyses

2.8.1. Quality Assessment/Risk of Bias

The abstract of each identified study would be analyzed by one of the authors and any article that did not meet the eligibility criteria would be excluded. The studies that met the inclusion criteria would be evaluated regarding the rationale, method design, primary outcome, assessment of fatigue, statistical analysis, results, discussion, and conclusions in order to improve data quality. Any study or studies that presented any bias in the methodology, results, or interpretation of the exposed data which could be reflected in the analysis of the study as a whole would be excluded. The exclusion of such biased studies would improve the overall analysis' reliability and precision. Also, we would assess the risk of bias by employing the quality appraisal checklist for quantitative studies reporting correlations and associations [27]. Studies would be assessed against 18 criteria in this tool [27]. This would be conducted independently by two authors and disagreements would once again be resolved through consensus among authors referencing the original studies.

2.8.2. Ethical Considerations

Since this study is a review, ethical approval is not required because it did not use data from human or animal subjects but rather from an open research repository.

3. Conclusions

The review article would help to concisely synthesize the available evidence on the relationship between burnout, inflammatory markers, and cardiovascular diseases. This synthesis of evidence can potentially inform future research and interventions in this area. Specifically, all the inflammatory markers implicated in this relationship should be further investigated to determine their role in the development of cardiovascular diseases. It would also help to elucidate how this comes into play in the pathophysiologic process. Additionally, studying the mechanisms by which these markers contribute to the pathophysiology of cardiovascular diseases could provide valuable insights. Grey literatures that may not be obtained may present a strong limitation in conducting an exhaustive review on this subject that requires comprehensive and up-to-date information.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/jmp4030017/s1, Figure S1: Flow chart of literature search and sorting process: Table S1. PRISMA checklist.

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