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FDG/Raclopride-PET neuroimaging in work-related stress – a systematic review

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Dear Editor,

Work-related stress is a major cause of decline in mental health, lost earnings, reduced productivity and long-term sickness absence. It can also be a precursor to disorders such as depression and anxiety (Stansfeld and Candy, 2006) as well as an increased risk of dementia, cardiovascular disease, and accidents (Wang et al., 2012). Stress can lead to diverse brain malfunctions extending from somatic disruption to affective and high-end executive abilities of cognitive functions. The etiologies behind work-induced mental disorders is well described and defined by prevailing stressors that cause these conditions (Flachs et al., 2015). However, to the best of our knowledge, there is very little evidence linking psychosocial work stressors to alterations in the neurobiology of the brain. We theorize that these effects can be detected as altered glucose metabolism in relation to neural activity and in the dopaminergic mesocorticolimbic projections in patients with work-related stress, measured by Positron Emission Tomography (PET), respectively with the radiotracers [¹⁸ F] Fluorodeoxyglucose (FDG) and [¹¹C] Raclopride (Raclopride). To this end, we conducted a systematic review in PubMed/MEDLINE, Scopus, Embase, Cochrane, and APA PsykInfo on December 12, 2022, to provide a literature overview of these neurobiological effects of work-related stress addressed with FDG and Raclopride PET neuroimaging.

Astonishingly, we could not identify a single relevant article (Figure 1).

We identified 13 articles in the literature search (see Supplemental Material for details). After the exclusion of seven doublets, five articles did not comply with the inclusion criteria in relation to diagnosis or molecular neuroimaging intervention, and one was the protocol article for the PhD project associated with this review (Madsen et al., 2018).

The absence of studies in this field might be explained by work-related stress in general only within recent years having obtained recognition and status as a clinical condition. Additionally, it is our impression that stress related conditions are still somewhat stigmatized and related to assumed weakness in the character of the individual not coping with the strains of work-related psychosocial stressors. Furthermore, stress, let alone work-related stress, is not a unified diagnosis throughout the world, and different ICD sub codes are applied in diagnosing the disorder across nations. The lack of neurobiological investigations into work-related stress may be reinforced by another aspect of the PhD project related to this systematic review (Madsen et. al., 2018). Here, we investigated the genotype variation of the COMT-gene in patients with work-related stress. The COMT-gene breaks down dopamine in the prefrontal cortex, and polymorphisms of the gene are believed to influence stress resilience. To the best of our knowledge, no other studies have addressed this subject yet. Finally, the availability of PET scanners in general and of the tracer Raclopride especially may be further impeding factors for FDG and Raclopride PET neuroimaging in work-related stress.

There is an evidence gap as to how work-related stress effects the neural activity in the brain and the binding potential of dopamine in the mesocorticolimbic projections of the prefrontal cortex underlying affective and high-end executive abilities of human existence.

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Contributors

Conceptualization: SSM and OG; Literature search: SSM, MNB, and RP; Paper appraisal: SSM, MNB, and RP; manuscript preparation and finalization: SSM and OG.

Conflict of Interest

Neither of the authors reports any conflict of interest.

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Supplementary material

Methodology, PICOS questions, and full search strategies for PubMed, MEDLINE, Scopus, Embase, Cochrane, and APA Psychinfo.

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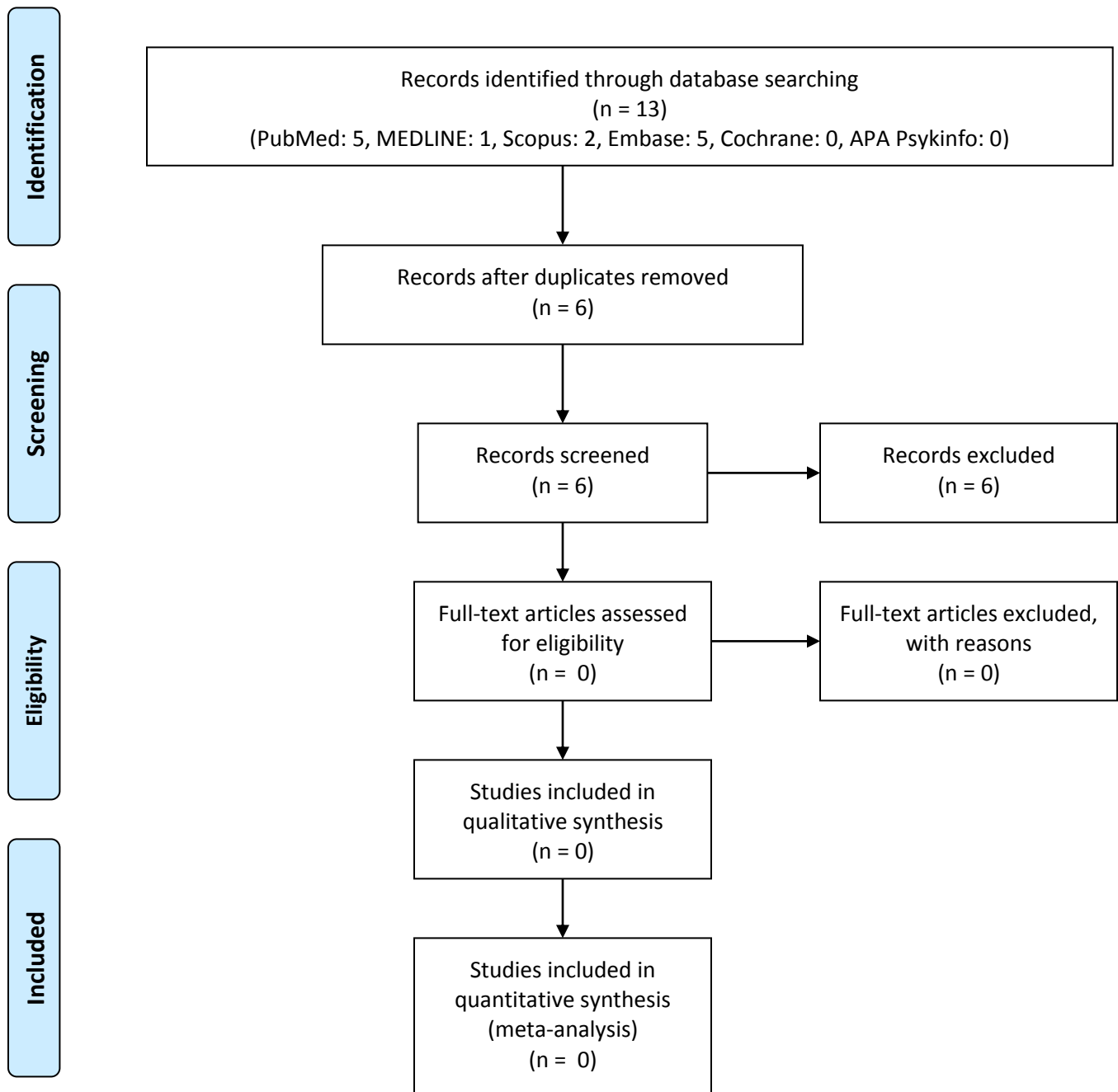
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Figure legend

Figure 1 Flow diagram for study selection



From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(6): e1000097. doi:10.1371/journal.pmed1000097

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