Longitudinal analysis of inflammatory, psychological, and sleep-related factors following an acute low back pain episode: A potential factor in tissue effects in low back pain

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Background

Systemic inflammation is observed in chronic low back pain (LBP) [3] and may contribute to the transition from acute to persistent LBP [2]. Lower back connective tissues are a possible source of this inflammation and can be modified by inflammation. Fibroblasts secrete inflammatory cytokines in response to stretch or injury of the connective tissues [1, 4], which can “spill over” into the bloodstream, leading to widespread secondary tissue damage and activation of pain pathways in the central nervous system [5]. Whether an early increase in systemic cytokines is related to LBP outcome is unclear. This study aimed to determine whether systemic cytokines and C-reactive protein (CRP) during an acute episode of LBP differ between individuals who did and did not recover by 6 months and to identify subgroups based on patterns of inflammatory, psychological, and sleep features associated with recovery/non-recovery.

Methods

Acute LBP (<2 weeks since onset) and pain-free participants without known inflammatory diseases/disorders provided blood for assessment of CRP, tumor necrosis factor (TNF), interleukin-6 (IL-6) and interleukin-1β after completing questionnaires related to pain/disability, sleep and psychological status. LBP participants repeated measurements at 6 months. Biomarkers were compared at baseline between LBP and control participants, and longitudinally (baseline/6 months) between unrecovered (≥pain and disability), partially recovered (<pain and/or disability) and recovered (no pain and disability) participants at 6 months. We assessed baseline patterns of inflammatory, psychological, sleep and pain data using hierarchical clustering and related the clusters to recovery (% change in pain) at 6 months.

Results

CRP was higher in acute LBP than controls at baseline. In LBP, baseline CRP was higher in the recovered than non-recovered groups. Conversely, TNF was higher at both time-points in the non-recovered than recovered groups. Two subgroups were identified that associated with more ("inflammatory and poor sleep") or less ("high TNF and depression") recovery.

Conclusion

In summary, high inflammation (CRP/IL-6) was associated with good recovery, but specific elevation of TNF, with or without depressive symptoms, was associated with bad recovery. Whether this systemic cytokine response is mediated by the connective tissue response to back
injury and whether it impacts long-term connective tissue health require further investigation.

References


