drinking water or regular water. Resected livers were stained for H&E for features of NASH and F4/80 for macrophages analysis. Liver RNA was evaluated for the expression of cytokines and chemokines using an 84-gene Profiler array (Qiagen). Oxidative stress was analyzed by qRT-PCR for heat shock proteins (HSPs) 27, 60, 70 and 90 and for glutathione by a fluorometric assay. Differences in CDE fed and CDE/proglumide-treated mouse livers were evaluated. RESULTS/ANTICIPATED RESULTS: Livers from mice on the CDE diet displayed histologic features of NASH that were prevented by proglumide. Cytokines and chemokines expression, especially CCL20 and CCL2, were increased in the CDE fed mice and these levels were reduced greater than 20-fold with proglumide. Infiltration of F4/80+ macrophages was markedly increased in the CDE livers and these were reduced by > 50% ($p < 0.0001$) with proglumide. RNA expression of HSP70 ($p = 0.006$) and HSP27 ($p = 0.011$) were reduced with proglumide. Hepatic glutathione concentration more than doubled in the CDE/proglumide treated mice compared to CDE mice. CCK-B receptor expression increased in the CDE-fed mouse livers compared to controls. DISCUSSION/SIGNIFICANCE OF IMPACT: CCK receptor blockade decreases NASH by reducing hepatic macrophages, oxidative stress, and blocking inflammatory cytokines and chemokines. This data supports our novel hypothesis that CCK receptors play a role in NASH and proglumide may provide an innovative treatment for this condition.

4095

Concurrent assessment of experimental pain and self-reported pain intensity with acute exercise intervention in fibromyalgia; clarifying or obscuring clinical outcomes?

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OBJECTIVES/GOALS: Experimental pain testing is used to identify changes in nociceptive processing and outcomes with intervention. This study investigated exercise induced changes in experimental pain and self-reported pain intensity after an acute bout of exercise in participants with fibromyalgia. METHODS/STUDY POPULATION: Ten females with fibromyalgia (55±10yr) were familiarized to study procedures and underwent submaximal (20% maximal voluntary contraction) intermittent eccentric muscle contractions isolated to the right arm for 10-minutes. Self-reported pain intensity (0-10 numerical pain rating scale [NPRS]) of the exercising arm was measured before, during, and after exercise; whole-body pain intensity was measured before and after exercise. Experimental pain testing included measurement of pressure pain thresholds (kPa [PPTs]); temporal summation (TS) of pressure pain with a constant mechanical pressure; and TS of punctate pressure with repeated application of monofilaments before and after exercise. RESULTS/ANTICIPATED RESULTS: Participants reported minimal to moderate arm pain (3.1±2.1) during exercise. Following exercise, arm pain and whole-body pain significantly increased (3.1±2.2 and 1.6±0.5, respectively) [$p<0.05$]. No change occurred with PPTs at the bicep (138.9±49.5 to 142.8±55.3), PPTs at the quad (212.0 ±105.4 to 228.1±100.0), TS of mechanical pressure pain (7.6±2.1 to 7.9±1.5), TS of punctate pressure pain at the bicep (2.6±1.7 to 3.0±1.5), and TS of punctate pressure pain at the quad (3.6±1.5 to 3.7±1.4) before to after exercise respectively [$p>0.05$]. The change in self-reported arm and whole-body pain did not correlate with the change in experimental pain testing. DISCUSSION/SIGNIFICANCE OF IMPACT: In people with fibromyalgia, there is no relation between self-reported clinical pain and experimental pain following a single exercise session. Further research should identify the influence of exercise training on pain perception and if experimental pain testing translates to clinical insight.

4302

Decreased structural basal ganglia motor loop connections in Vascular Parkinsonism compared to Parkinson's disease and healthy aging

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OBJECTIVES/GOALS: This study uses diffusion kurtosis imaging (DKI) to investigate the structural profiles of basal ganglia (BG) motor circuitry in Vascular Parkinsonism (VP), Parkinson's disease (PD), and healthy aging controls (HC). VP is a clinical diagnosis of lower body predominant parkinsonism without significant benefit from levodopa. VP is distinct from PD, yet the concept of VP remains debated due to the inability of prior studies to identify specific causative changes. One reason for this may be limitations in measuring intricate BG connectivity in vivo. Given the predominant lower body parkinsonism symptoms in VP, we hypothesized that VP would be associated with decreased connectivity specifically within the BG motor loop. METHODS/STUDY POPULATION: We obtained DKI brain imaging in subjects with VP ($N = 7$), PD ($N = 21$), and HCs ($N = 58$), the latter of which had cardiovascular risk factors but no neurological symptoms. The VP and PD groups were evaluated by a parkinsonism-focused motor exam and brief cognitive testing. We compared BG motor loop connectivity between groups and investigated for correlation between connectivity and clinical scores. To account for differences in fiber counts due to the different imaging scanners and protocols between cohorts, we used a BG motor loop proportion, which was the ratio of the BG motor loop fiber count over a control loop, the visual processing pathway. We used Kruskal-Wallis rank sum test with post-hoc Dunn tests to assess imaging findings between subject groups, and Pearson's correlation to look for correlation between clinical scores and fiber counts. RESULTS/ANTICIPATED RESULTS: The whole brain connectome showed the fewest number of fibers in VP, followed by PD, and then HC ($p<0.0001$). The BG motor loop proportion fiber count of the BG motor loop was lower in the VP group, compared to the PD and HC cohorts ($p = 0.031$). In the VP group, the whole brain connectome fiber count correlated with a gait and balance subscore of the Movement Disorders Society - Unified Parkinson Disease Rating Scale ($R = -0.87$, $p = 0.01$). DISCUSSION/SIGNIFICANCE OF IMPACT: This study indicates that VP is associated with decreased structural connectivity, with a disproportionate degree of loss in the BG motor circuitry. While the etiology for this susceptibility to injury and preferential damage to BG remains to be defined, these findings can provide an important starting point for a biological understanding of VP, and a potential future marker for diagnosis and tracking disease progression.

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