of symptoms. Previous work suggests that treatment for insomnia can have an indirect impact on chronic pain and the neural networks involved in chronic pain. The current work explores the correlational relationship of sleep and pain, and how the underlying neural correlates of pain are affected by ability to obtain adequate sleep. Behavioral measures of sufficient sleep and extent of pain symptoms and disabling effects of pain were obtained from healthy controls and CP patients. Resting state fMRI scans were acquired for all participants. MRI scanning was done using a 3-Tesla Siemens Trio scanner. SPM12 was used for all image processing. The functional data were preprocessed, and coregistered to the EPI template (toolbox/OldNormEPI.nii), followed by coregistering the mean image to the skull-stripped, anatomical T1 scan. A group contrast was used to extract regions of interest (ROIs) associated with chronic pain. We then tested whether the strength of activation in these ROIs varied as a function of sleep measures. We predicted that individuals with chronic pain who obtain sufficient sleep would experience less severe pain symptoms and exhibit reduced activation in brain regions associated with chronic pain. As expected, better sleep quality and efficiency were associated with fewer chronic pain symptoms and reduced pain disability. Preliminary results indicate that sleep measures can be differentially associated with pain severity of various pain conditions in the same patient, and that sleep quality and efficiency were associated with the cognitive and emotional components of chronic pain. These results suggest that poorer sleep quality may be associated with maintaining the chronic pain state. Future work will explore the potential influence of mood on these variables. This research is supported via a NIH-NINR grant: R01 NR015314-01A.

(398) Impaired physical function is associated with feeling older than actual age in obese adults with symptomatic knee osteoarthritis

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Recent research addressing self-perceived age has shown that adults who feel older than their actual age may be at increased risk for premature death. The exact reasons why certain adults might feel older than their actual age remain unclear; however, poor physical function and failure to maintain a healthy weight have been suggested as potential risk factors. Individuals with symptomatic knee osteoarthritis (OA) may be particularly at risk for feeling older than they actually are given that physical disability and obesity are common correlates of knee OA. This study sought to determine whether measures of physical function and evoked pain as well as obesity status predicted self-perceived age in a sample of 81 adults with symptomatic knee OA aged 46-78 years. Participants reported their chronological age and perceived age according to how old he/she is, and how old he/she thinks he/she should be, respectively. Physical function and quality of life were measured using the Short Physical Performance Battery (SPPB). Intensity of evoked pain during completion of the SPPB was also rated. In an adjusted model, results revealed a significant BMI by physical function interaction for self-perceived age (βR = .056, p = .006). Specifically, poorer physical function predicted feeling older than actual age for participants with average and above average BMI (p = .029 and p = .001, respectively), but not for those with below average BMI (p = .697). Similar analyses evaluating the effects of evoked pain intensity were non-significant: Average (30.3, SD = 6.9) and above average BMI (37.2) in this sample both met criteria for obesity; however, below average BMI (23.4) was normal weight. Thus, obesity appears to potentiate the effect that poor physical function has on self-perceived age in adults with symptomatic knee OA. Supported by a grant from NIH/NIA; R37AG033906.

(399) Small Fiber Polyneuropathy: A Big Clue to Etiology and Management of Chronic Pelvic Pain (CPP)

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This study was designed to assess prevalence of small fiber polyneuropathy (SFPN) based upon clinical presentation and skin biopsy findings in patients with refractory CPP and concurrent pain syndromes. We hypothesized that hypersensitivity is thought to have the average age of the patients with multiple pain syndromes like irritable bowel syndrome (IBS), interstitial cystitis (IC), and fibromyalgia (FM). The lack of common etiology complicates the ability to offer effective treatment options. SFPN is emerging as a major contributor to unexplained multi-symptom syndromes involving chronic widespread pain and is often present in patients with IBS and FM. The diagnosis of SFPN can be confirmed via skin biopsy: decreased epidermal nerve fiber density is demonstrated on immunofluorescence. We evaluated refractory CPP patients with 3mm punch biopsies of the lower extremity through Corinithian Reference Lab. The sensitivity and specificity are 78-92% and 65-90% respectively. Comorbid conditions in our population include migraine (39%), IBS (36%), endometriosis (21%), FM (32%), IC (14%), GERD (50%), vulvodynia (7%), lower back pain (32%), and other chronic pain syndromes (35%). In our practice, 19 (68%) of 28 patients were positive for SFPN. The prevalence of SFPN in specialty referral patients with refractory CPP is remarkably high versus published population data, which estimates “minimum prevalence” at 52/100,000. Such a significant proportion of refractory CPP patients with biopsies consistent with SFPN has never been reported in the literature. Consideration for SFPN shifts the focus from syndromes to unifying treatable disorder. Making the diagnosis of SFPN may result in treatments not usually offered such IVIG or other immunomodulatory therapies. Identifying SFPN should be a priority in CPP patients. (1. Krieger, J. N., et al. J Pain 2015; 2. Gondim, A., and exercise; 3. Albrecht, P. J., et al. Pain Medicine 2013; 4. Peters, MJ, et al. Neurology 2013; 5. Caro, X. J., et al. Rheumatol [Oxford], 2008)

(400) Increased keratinocyte Nav subunit expression among painful diabetic peripheral neuropathy patients predicts Lidoderm patch responsiveness

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Currently, nearly 30 million people (~9.3% of the U.S. population) have diabetes. Furthermore, 1 in 4 people are unaware they have the disease, and an estimated 86 million Americans over the age of 20 years have prediabetes. Therefore, cases of chronic painful diabetic peripheral neuropathy (PDPN) continue to rise among both the aging and young population. Pharmacologic treatment for PDPN is limited to only two FDA-approved oral medications (pregabalin [Lyrica], duloxetine [Cymbalta]), although most neuropathic pain analgesics also have benefit, including tricyclics, antidepressants, anti-epileptics, opioids, and others. Yet, most compounds are burdened with unwanted CNS side-effects that limit utility. Therefore, use of topical 5% lidocaine also remains a front-line treatment option for neuropathic pain. The Lidoderm patch (5% lidocaine, ENDO Pharmaceuticals) has demonstrated benefit in the treatment of PDPN, however the mechanism remains unclear. Here, we utilized human skin biopsy evaluations to determine if cutaneous innervation (PGP) or keratinocyte voltage-gated sodium channel subunits (Nav1.6, Nav1.7) and calcitonin gene-related peptide (CGRP) were altered in painful and/or non-painful DPN patients, and if any changes were observed following a 4-week treatment with Lidoderm patch. As expected, there were significant losses of intraepidermal nerve fibers (IENF), subepidermal axons, and upper dermal nerves among both PDPN and non-painful DPN patients compared with control subjects, however no innervation measures differentiated between pain and non-pain patients. Epidermal keratinocyte expression of Nav1.6, Nav1.7, and CGRP were each increased among PDPN patients compared with control, and CGRP was also increased in pain versus non-pain groups. Efficacious treatment with Lidoderm patch reversed these skin increases, and response analysis demonstrated that pretreatment expression levels of keratinocyte Nav1.6 positively-predicted outcome. These findings further implicate keratinocyte mechanisms as likely mediators of painful conditions where small fiber neuropathy is present. This study was funded, in part, by an investigator-initiated grant from ENDO Pharmaceuticals.

(401) A SNP in COL11A2 associated with thermal hyperalgesia in patients with knee osteoarthritis

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Hypersensitivity to painful stimuli is a well-recognized phenomenon in patients with knee osteoarthritis (OA). The degree of overall hypersensitivity is thought to have the average age of 58 years. Despite the high prevalence of hypersensitivity, including the central processing and genetic variability. We hypothesize that there are natural genetic variations within the pain genes affecting the development of such hypersensitivity in patients