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NEUROPATHIC PAIN IS A WEAK PREDICTOR OF NEW ONSET CHRONIC WIDESPREAD PAIN

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My abstract has been or will be presented at a scientific meeting during a 12 months period prior to EULAR 2017: No

Is the first author applying for a travel bursary and/or an award for undergraduate medical students?: No

Background: Regional pain (e.g. back pain) predicts incident chronic widespread pain (CWP), the clinical hallmark of fibromyalgia. Up to 20% of patients with CWP have neuropathic pain (NP). People with CWP and NP report similar pain characteristics including allodynia (pain in response to normal touch), have common risk factors (age, sex, body mass index, smoking and socioeconomic status) and a shared genetic predisposition. Whether NP is a risk factor for CWP is not known.

Objectives: To test the hypothesis that among persons free of CWP, NP would increase the risk of developing CWP.

Methods: In a population based study participant's pain reports were coded and those free of CWP (ACR criteria: pain lasting ≥3 months in the axial skeleton and contralateral body quadrants) identified. Participants also completed the Douleur Neuropathique 4 (DN4) (which has 7 sensory descriptors of pain (burning, painful cold, electric shocks, tingling, pins and needles, itching, and numbness), scores ≥3 indicating NP); demographics (date of birth, sex, English Index of Multiple Deprivation, occupational status); Hospital Anxiety and Depression (HAD) scale; Estimation of Sleep Problem Scale (ESPS); self-reported pain medications (summed to give a total count); and signed consent. Participants were classified as no pain, having some pain that wasn't neuropathic (NP-; DN4 score <3), or neuropathic pain (NP+; DN4 score ≥3). A follow-up questionnaire mailed 12 months later gathered pain data using the methods in the baseline survey. Based upon their pain reports at follow up participants were classified as "new CWP" for those who reported pain that satisfied the criteria for CWP, or "not CWP". Logistic regression estimated the odds of developing new CWP in the NP-, and NP+ groups compared to the no pain group. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI). Population attributable fractions (PAF) estimated the % of new CWP that would be avoided if participants were not exposed to NP- or NP+.

Results: A total of 1162 participants who were free of CWP, completed the DN4 and provided pain data at follow up. Of those 523 (45.0%) had no pain at baseline, 562 (48.4%) had NP- and 77 (6.6%) had NP+. New onset CWP was reported by 153 (13.2%) participants; 19 (3.6%) of the no pain group, 108 (19.2%) of the NP- group, and 26 (33.8%) of the NP+ group. After adjusting for age and sex, compared to the no pain group, the NP- group was 3 times (OR 2.9, 95% CI (2.0, 4.2)) and the NP+ group 4 times (3.9 (2.3, 6.4)) more likely to have new CWP at follow up. These relationships were attenuated but persisted after adjustments for demographics, HAD, ESPS and medication use (NP- (2.9 (1.9, 4.3); NP+ (2.1 (1.1, 4.0)). The PAF was 41.3% (95% CI (25.2, 54.0)) for NP- and 6.0% (0.1, 11.6) for NP+. All of the individual DN4 characteristics except painful cold and itching predicted new CWP with PAF's ranging from 1.6% (0.1, 3.8) for pins and needles to 5.0% (1.1, 8.8) for burning.

Conclusions: NP predicts a small number of new onset CWP cases. CWP is highly prevalent in the general population and effective treatment of pain not of NP origin will have a significant impact on population levels of CWP.

Disclosure of Interest: None declared