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Fatty Acid Binding Protein 5 Inhibitor ART26.12 Provides Immediate and Lasting Analgesia for Osteoarthritic Pain

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Introduction

Chronic pain associated with knee osteoarthritis (OA) lacks safe, efficacious long-term analgesic treatments due to the complexity of pain signaling pathways. Non-steroidal anti-inflammatory drugs (NSAIDs) have short-term efficacy but suffer gastrointestinal risks. Fatty-acid binding protein 5 (FABP5) inhibition has been shown to exhibit analgesic activity by modulating fatty acid transfer within the endocannabinoid system (ECS). This study investigated efficacy of FABP5 inhibition in reducing OA pain using the novel drug, ART26.12, in a rat model.

Materials and Methods

Experiments were approved by the Stony Brook University Institutional Animal Care and Use Committee (#277150). 51 Female Sprague Dawley rats underwent surgical destabilization of the medial meniscus (DMM). Eight weeks post-surgery, five rat groups (N=10) received acute dosing (PO BID) of vehicle, 8 mg/kg naproxen, and three concentrations of ART26.12 (10, 25, and 50 mg/kg). Static incapacitance (reported as an averaged ratio of ipsilateral to contralateral hindlimb weight bearing) and grip strength (averaged ratio of ipsilateral force (g) to rat weight (g)) were measured at 1 and 4 hours after acute dosing, and weekly from weeks 8-12 post-DMM during the chronic dosing phase (same dosing regime). X-rays were taken to track OA progression. Results were presented as dot plots with mean, significance, and 95% confidence intervals overlaid.

Results/Case Report

Expectedly, the vehicle group exhibited no differences in incapacitance with acute or chronic dosing (Fig. 1,2). In acute dosing, the ART26.12 groups demonstrated significantly increased weight bearing on the ipsilateral (arthritic) limb versus the contralateral limb, indicated by increased weight bearing ipsilateral/contralateral ratios between baseline and post-dose timepoints. Specifically, rats dosed with 10 mg/kg ART26.12 showed a significant increase in weight bearing on the ipsilateral limb only at the 1-hour post-dose time point, while the 25 mg/kg and 50 mg/kg ART26.12 groups showed significant increases at both 1-hour and 4-hour post-dose timepoints (Fig. 1). With higher ART26.12 concentrations, from 10 mg/kg to 25 mg/kg and 50 mg/kg, there was a more pronounced, statistically significant increase in ipsilateral weight bearing from baseline to 1-hour and 4-hour post-dose timepoints. In acute dosing, the 8 mg/kg naproxen group showed a significant increase in ipsilateral weight bearing only at the 4-hour post-dose time point (Fig. 1). In chronic dosing, there were significant increases in ipsilateral weight bearing between weeks 0.5-4 for ART26.12 treatment groups, with similar increases observed in the naproxen group (Figure 2). No significant differences were observed in acute or chronic grip strength measurements across groups or timepoints (Figure 3,4).

Discussion

Increased weight bearing ratios across all timepoints suggest that ART26.12 reduced OA-induced pain acutely and chronically, with chronic incapacitance data demonstrating that ART26.12 effectiveness did not decrease with time. Absent significant grip strength variation may be due to minimal isolated pressure to the knee during grip action. Despite similar effectiveness of naproxen and ART26.12 in chronic incapacitance, ART26.12 may still be more favorable than NSAIDs if histological analysis reveals healthier gastrointestinal tracts in ART26.12 groups.

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