**Introduction:** In this study, characteristics of DRG neurons innervating the hip joint in inflammatory states were investigated in rats using retrograde neurotransport and immunohistochemistry. We first investigated the ratios of CGRP-IR and IB4-binding neurons per retrogradely labeled DRG neurons innervating the hip joint under physiologic conditions. We then examined the effects of the hip joint inflammation on each type of neuron innervating the hip joint.

**Materials and Methods:** Twenty male SD rats weighing 250–300 g were used. 30 μl of saline (control group: n=10) or 30μl of complete Freund’s adjuvant (inflammatory group: n=10) was injected into the left hip. Seven days after first operation, Bilateral DRGa from the T12 to L6 levels were resected. The specimens were processed for CGRP immunohistochemistry and IB4-binding using biotin-labeled IB4 (1:1000) and rabbit antibody to CGRP (1:1000). After incubation with labeled isoelectric or antibody, sections were incubated with goat anti-rabbit Alexa 594 (for CGRP-IR; 1:1000) or streptavidin Alexa 488 fluorescent antibody conjugate (for IB4-binding; 1:1000). FG-labeled neurons (Fig.1A), and FG-labeled and CGRP-IR (Fig.1B) or IB4-binding (Fig.1C) neurons were counted. Hip joint was decaclfied stained with haematoxylin and eosin and assessed by light microscopy.

**Results:**

**Histological appearance:** Inflammatory group exhibit development of histopathology. Articular cartilage and subchondral bone are generally intact. Synovial membranes were hyperplastic and accompanied by mononuclear inflammatory cell infiltrates in synovial/subsynovial tissues. FG-labeled DRG neurons: In control group, the numbers of FG-labeled neurons in L1, L2, L3, L4 were significantly higher than the numbers of labeled neurons in T13 and L5(Fig.2). In inflammatory group, the numbers of FG-labeled neurons in L3**, L4** were significantly higher than the numbers of labeled neurons in T12, T13, L1, L2 and L5 (Fig.3). (**p<0.05, ***p<0.01**) FG-labeled neurons expressing CGRP and binding IB4: In control group, CGRP-IR neurons in DRG were present at 36.0±26.9% in L1, 24.0±24.8% in L2, 36.0±26.8% in L3, 48.0±28.3% in L4, and 26.7±46.2% in L5. IB4-binding neurons were found at 2.8±2.6% in L2, 3.9±7.3% in L3 and 1.7±4.7% in L4(Fig.4). In inflammatory group, CGRP-IR neurons in DRG were present at 75.0±41.8% in L1, 57.2±20.7% in L2, 33.3±10.5% in L3, 37.7±9.1% in L4 and 33.4±28.8% in L5. IB4-binding neurons were found at 4.4±10.7% in L2, 3.9±2.6% in L3, 2.0±1.8% in L4 and 4.4±8.4% in L5(Fig.5). Comparison Between the Control and Inflammatory groups: In the control group, 33.3± 31.2% were CGRP-IR and 1.2± 3.6% were IB4-binding. In the Inflammatory group, 45.1± 26.3% were CGRP-IR and 2.3± 4.4% were IB4-binding. The mean percentage of CGRP-IR neurons innervating the hip joint in inflammatory states, relative to the total number of neurons, was significantly higher than the mean percentage innervating the normal Hip joint (p<0.05). In contrast, there was no significant difference between the two groups in the mean percentage of IB4-binding neurons relative to the total number of neurons.

**Discussion:** The results of this study indicate that the rat hip joint under each condition are innervated from the ipsilateral T13 to L5 DRG. Our observations showed that most of the DRG neurons innervating the rat hip joint under physiologic conditions are distributed in L1, L2, L3, and L4, and those innervating the hip joint in inflammatory states are distributed in L3 and L4. These results support the findings from anatomic human studies, and may explain the clinical referred pain in the thigh and lower leg that originates from hip joint pathologies. CGRP-IR neurons may have a significant role in hip joint pain sensation via peptidergic DRG neurons involved with sensation of inflammatory pain. The hip joint inflammation causes an increase in the percentage of DRG neurons innervating the hip joint that are CGRP-IR, but not in the percentage that IB4-binding. These results suggest that NGF-dependent but CGRP-negative neurons exist in the DRG and that inflammation induces CGRP in these neurons. In contrast, GDNF-dependent IB4-binding neurons were not likely to be affected by the hip joint inflammation. The results of this study lead us to believe that NGF is one of the key molecules involved in the hip joint inflammatory pain.

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**Characteristics of sensory DRG neurons innervating the Hip joint related to Inflammatory Pain**

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