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## Usefulness of intra-articular Botulinum toxin injections: A literature review

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**Background:** Botulinum toxin is a proven and widely used treatment for numerous conditions characterized by excessive muscular contractions. Recent studies have assessed the analgesic effect of Botulinum toxin in joint pain and started to unravel its mechanisms.

**Literature Search Methodology:** We searched the international literature via the Medline database using the term “intraarticular botulinum toxin injection” combined with any of the following terms: “knee”, “ankle”, “shoulder”, “osteoarthritis”, “adhesive capsulitis of the shoulder”.

**Results:** Of 16 selected articles about intraarticular botulinum toxin injections, 7 were randomized con-trolled trials done in patients with osteoarthritis, adhesive capsulitis of the shoulder, or chronic pain after joint replacement surgery. Proof of anti-nociceptive effects was obtained in some of these indications and the safety and tolerance profile was satisfactory. The studies are heterogeneous. The comparator was usually a glucocorticoid or a placebo; a single study used hyaluronic acid. Pain intensity was the primary outcome measure.

**Discussion & Conclusion:** The number of randomized trials and sample sizes are too small to provide a satisfactory level of scientific evidence or statistical power. Unanswered issues include the effective dosage and the optimal dilution and injection modalities of botulinum toxin.

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## Identification of miR-26a as a target gene of bile acid receptor GPBAR-1/TGR5

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**Objective:** This study is to identify whether miR-26a is a target gene of bile acid receptor TGR5 in BAT in spontaneous and diet-induced obesity animal models, thus providing a potential link between TGR5 and miR-26a expression.

**Methods:** We used spontaneous and diet-induced obesity mice to be determined whether TGR5 is the only receptor required to mediate the effect of OA on obesity. Mouse body weights were monitored during the entire process. Histological examination and metabolic measurements were also performed.

**Results:** TGR5 partially mediated the effect of OA on obesity and glucose regulation. TGR5 activation increased the expression of miR-26a. TGR5 activation up-regulated miR-26a expression in macrophages. JNK pathway was downstream of TGR5 in miR-26a induction. TGR5 responsible DNA sequences were in the proximal regions of miR-26a promoter.

**Conclusion:** These findings suggest the activation of TGR5 by OA can modulate the expression of miR-26a through a JNK-dependent pathway for the treatment of obesity-associated metabolic diseases.

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