

A Commentary on “Efficacy and Potential Mechanisms of Yi-Ping Lin Circum-Knee Acupuncture in the Treatment of Knee Osteoarthritis” [Letter]

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Dear editor

I read with great interest the exploratory study by Lijun Huang et al, on the efficacy and mechanisms of Yi-Ping Lin circum-knee acupuncture (CKA-L) for knee osteoarthritis (KOA).¹ The authors report that CKA-L and piroxicam both attenuate KOA symptoms and foster chondrocyte regeneration, with CKA-L producing a significantly greater drop in TNF- α . Their transcriptomic work further shows that CKA-L curbs inflammation and cartilage loss while promoting cartilage and muscle repair by enhancing DNA repair, propelling the cell cycle, inhibiting chondrocyte-apoptosis-linked cysteine-endopeptidase activity, down-regulating pro-inflammatory genes, and up-regulating the anabolic gene Bgn. We commend this innovative study and would appreciate the authors' clarification of the following points.

First, regarding animal modeling methods, the authors employed the DMM procedure to excise ligaments and menisci in mice to establish a KOA model. However this model is more indicative of acute mechanical injury rather than chronic metabolic-inflammatory degeneration. Literature^{2,3} indicates that female estrous cycles and estrogen-mediated chondro-protection retard cartilage loss in mice, so males are the species/sex of choice for DMM studies. However, the authors exclusively used female mice in this study, exclusive use of females here risks underestimating baseline damage and overestimating therapeutic benefit. It is recommended to adopt an age-related spontaneous KOA model and use both male and female mice to reduce extrapolation bias.

Regarding the samples submitted for testing, the methods section merely states “joint tissue extraction” or “cartilage sectioning” without specifying the exact sampling sites. Cartilage, synovium, and bone marrow participate in different mechanisms within KOA. Pooling these compartments blurs tissue-specific transcriptional signatures and can artifactually skew DEG enrichment. It is recommended to employ laser capture or microdissection techniques combined with RNA sequencing of specific regions, which can maintain spatial resolution while revealing mechanistic insights—something hybrid tissue sequencing cannot achieve.

About the piroxicam group, it served only as an efficacy control. No tissue was subjected to RNA-seq, so all transcriptomic, GO-KEGG, PPI and hub-gene data reflect CKA-L exclusively. Western Blots and qPCR compare Sham, Model and CKA-L only, and the ELISA panel for piroxicam is limited to two cytokines without pathway context. Thus, the conclusion that “both drugs can reduce inflammation” does not reveal the similarities and differences in the mechanisms of action between CKA-L and the drugs.

Finally, the CKA-L protocol lists points, depth and retention time, but omits needle angle and manipulation force; operator identity and intra- and inter-operator consistency were also not reported. It is recommended that these acupuncture parameters be refined to enhance the reproducibility of the experiment. Another key limitation is the absence of

a sham-acupuncture control. Without non-penetrating or superficial needling, the specific contribution of CKA-L cannot be disentangled from the nonspecific effects of needle insertion, clinician contact, or patient expectation. Future trials should include a credible sham arm to establish whether CKA-L offers benefits beyond placebo in knee osteoarthritis.

In summary, I commend the authors for their valuable clinical observations. I would be most grateful if the author could kindly address my concerns, and I trust that my suggestions will also be taken into consideration when planning large-scale animal experiments in the future.

Disclosure

The author reports no conflicts of interest in this communication.

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