# Article information:

RUNX2 stabilization by long non-coding RNAs contributes to hypertrophic changes in human chondrocytes - PMC  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9760429/>

# Article summary:

1. This study identified two long non-coding RNAs (lncRNAs), LINC02035 and LOC100130207, which play important roles in hypertrophic changes in normal chondrocytes.

2. The expression level of RUNX2, a master regulator of chondrocyte hypertrophy, was regulated at the post-translational level during hypertrophic differentiation of the normal human chondrocyte cell line, TC28a2.

3. Knockdown (KD) of LINC02035 or LOC100130207 promoted ubiquitin-mediated proteasomal degradation of RUNX2 and prevented hypertrophic differentiation of normal chondrocytes, suggesting that these two novel lncRNAs could be potential therapeutic targets for delaying or preventing OA development.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article is generally reliable and trustworthy as it provides evidence to support its claims through experiments such as western blotting, real-time quantitative polymerase chain reaction, osteocalcin reporter assay, RNA-immunoprecipitation (RNA-IP), RNA-in situ hybridization, and IP. Furthermore, the article is well written with clear explanations of the methods used in the experiments and their results.

However, there are some potential biases that should be noted. Firstly, the study only focuses on two lncRNAs (LINC02035 and LOC100130207) without exploring other possible lncRNAs that may also contribute to hypertrophic changes in normal chondrocytes. Secondly, the study does not provide any information on possible risks associated with using these lncRNAs as therapeutic targets for OA development. Lastly, the study does not present both sides equally as it only focuses on how these lncRNAs can be used to prevent OA development without exploring other possible treatments or therapies for OA.

# Topics for further research:

* Osteoarthritis treatments
* Osteoarthritis risk factors
* Other long non-coding RNAs
* Hypertrophic chondrocyte changes
* Therapeutic targets for OA
* RNA-in situ hybridization techniques

# Report location:

<https://www.fullpicture.app/item/df5e0ae4f87b9e73f37482ba629188af>