

# TECHNOLOGY Selective RAR? Ligand-loaded Nanoparticles for Manipulation of Targeted Bone Growth

#### OVERVIEW

Imbalance in bone length, caused by congenital conditions or fractures, can result in progressive deformity and physical problems. To date, the only way to adjust these bone length problems are surgical procedures. UMB researchers have identified a nuclear retinoid receptor, RAR?, which controls the rate of transition from cartilage to bone and is therefore critical for longitudinal bone growth. They have developed a poly-L-lactide (PLLA)-based nanoparticle formulation for the targeted delivery and release of RAR? agonists and antagonists into the growth plate of specific bones. This represents a new method of treating abnormal bone growth by enhancing or blocking ossification.

This technology allows the control of endochondral bone formation locally, anywhere in the body, through modulation of RAR? receptors. Delivery of a RAR? agonist via nanoparticles to one side of the growth plate, or systemically, induces early closure of the plate, which can be used to correct bone alignment in joints. Conversely, delivery of RAR? antagonists, systemically or via nanoparticles, can prevent the early closure of growth plates and promote cartilage growth, helping heal growth plate fractures by preventing bone-bridge formation. RAR? targeting can be combined with the delivery of bone morphogenetic protein (BMP), bone conducting grafting materials, and BMP/scaffold/skeletal progenitor cells to promote bone repair, heal fractures, and aid spinal fusion.

#### **ADVANTAGES**

Pharmacological treatment can replace surgical standard of care

#### STAGE OF DEVELOPMENT

Proof of concept experiments with RAR? agonist and antagonist nanoparticles have been performed in a tibia defect repair model in wild-type and RAR?-null mice. These studies show bone repair is enhanced in RAR?-null mice, an effect mimicked by pharmacological isotype-specific antagonism of RAR? in a mouse ectopic bone model. In contrast, RAR? agonists inhibit chondrogenesis and reduce cartilage size.

#### LICENSING POTENTIAL

Available for licensing

#### **CONTACT INFO**

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## **Additional Information**

## INSTITUTION

University of Maryland, Baltimore

## PATENT STATUS

PCT application filed, pending

## LICENSE STATUS

Available for licensing and/or sponsored research

### CATEGORIES

- Therapeutics
- Other

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## ATTACHMENTS

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