Technology from Kyoto University

Novel Nucleic Acid Therapeutics for Inflammatory Diseases

We are looking to out-license the technology for its commercialization.

Reduces inflammatory cytokines through a different mechanism, offering a new treatment option for inflammatory diseases such as atherosclerosis and MASH.

◆Background

Inflammatory diseases affect a vast global population and drive chronic conditions like atherosclerosis and MASH. Macrophage-driven inflammation plays a key role in plaque formation and liver fibrosis, yet current treatments are limited, highlighting the need for new molecular approaches targeting this pathway.

◆Description

Researchers at Kyoto University and Osaka University, working collaboratively, identified a long non-coding RNA (lncRNA) whose expression in human macrophages changes in response to cholesterol. They developed a synthetic nucleic acid that suppresses this lncRNA. When introduced into human macrophages, it inhibits the maturation of inflammatory cytokine mRNAs, reducing the expression of cytokines such as IL-6 and TNF. This work highlights a novel therapeutic target and a promising nucleic acid–based approach.

- **Reduced atherosclerotic lesions and fibrotic areas in MASH liver confirmed**
  Deleting the homologous lncRNA in mice suppressed macrophage-driven inflammation and slowed progression of atherosclerosis and MASH (Fig. 1). Treatment with the synthetic nucleic acid also improved inflammatory markers and liver function.

- **Compatible with existing therapies**
  This new approach targets macrophages to regulate inflammation via a mechanism distinct from conventional lipid- or glucose-focused therapies. It can be combined with existing treatments and may extend to inflammatory diseases beyond atherosclerosis and MASH.

◆Development Status

- In-vitro anti-inflammatory effect confirmed with synthetic nucleic acid
- In vivo efficacy demonstrated by reduced atherosclerosis and liver fibrosis in disease mouse models lacking the homologous lncRNA

TRL: Level 3

◆Applications

Drug development for inflammatory diseases such as
- atherosclerosis
- MASH
- others

◆Offer

- Patent License
- Option for Patent License
- Collaborative Research

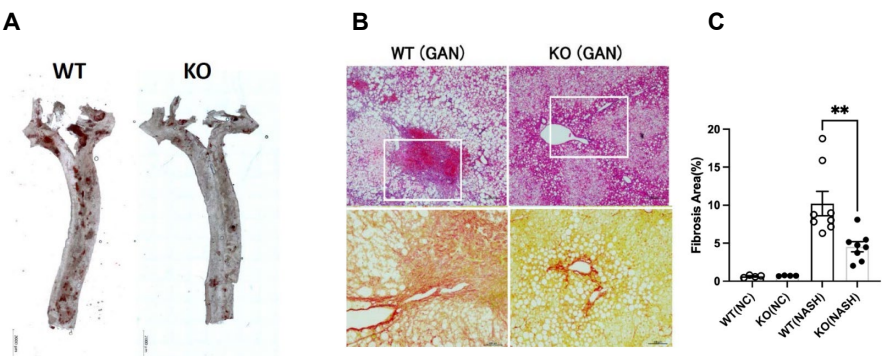

Fig. 1: Suppressive effects on disease lesions caused by lncRNA deficiency

A: Atherosclerosis model mice *(ApoE-deficient mice)*:
In lncRNA knockout (KO) mice, the amount of plaque stained with Sudan IV was reduced compared with lncRNA wild-type (WT) mice.

B&C: MASH/NASH disease model mice (GAN diet):
In lncRNA knockout (KO) mice, a reduction in the fibrosis area of the liver was observed.

◆Contact

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IAC Institutional Advancement and Communications

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