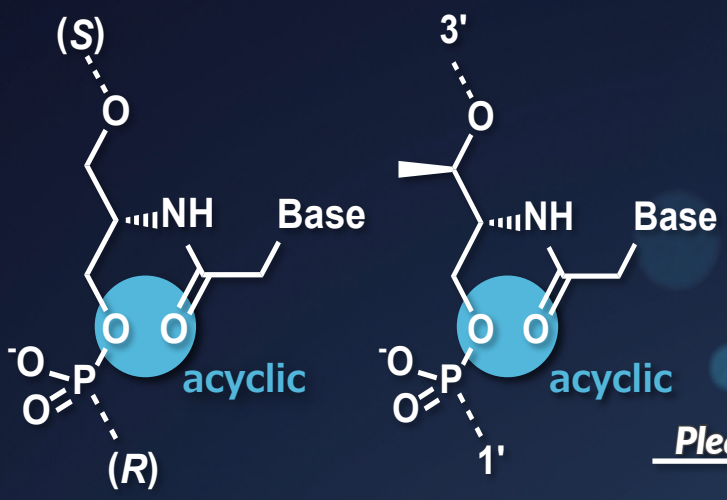
MITSUBISHI GAS CHEMICAL

Acyclic Nucleic Acid
SNA iL-aTNA

An acyclic artificial nucleic acids developed by **Professor Hiroyuki Asanuma's laboratory** at Nagoya University that outperforms conventional artificial nucleic acids in performance and safety.



Key Features

- ✓ **Stable duplexes** with canonical DNA or RNA
- ✓ **Strong resistance to nuclease degradation**
- ✓ **Lower cytotoxicity** than sugar-modified nucleic acids commonly employed in conventional nucleic acid therapeutics

Please feel free to contact us for oligo synthesis using these acyclic nucleic acids.

NOTE : This service is available only under agreement with Nagoya University.

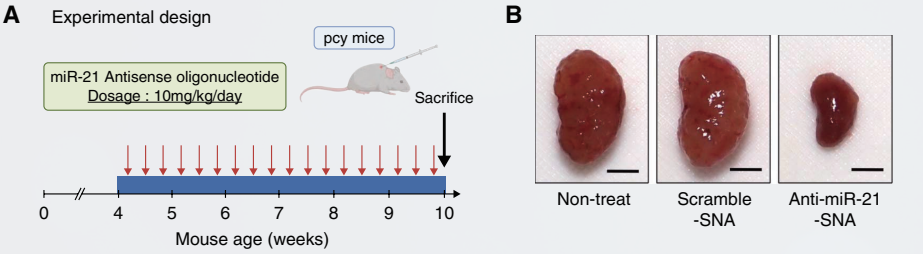
Effect of anti-miR-21 containing acyclic nucleic acid in autosomal dominant polycystic kidney disease (ADPKD)

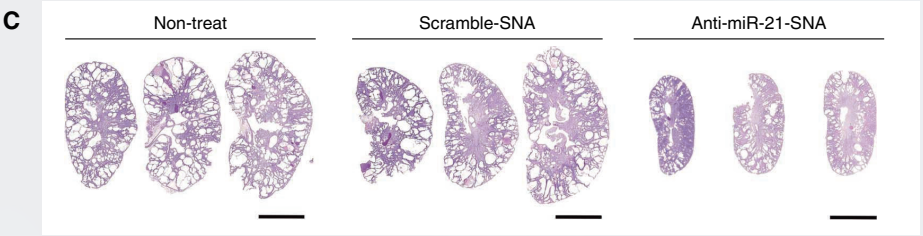
▼ **Synthesized by Hokkaido System Science**

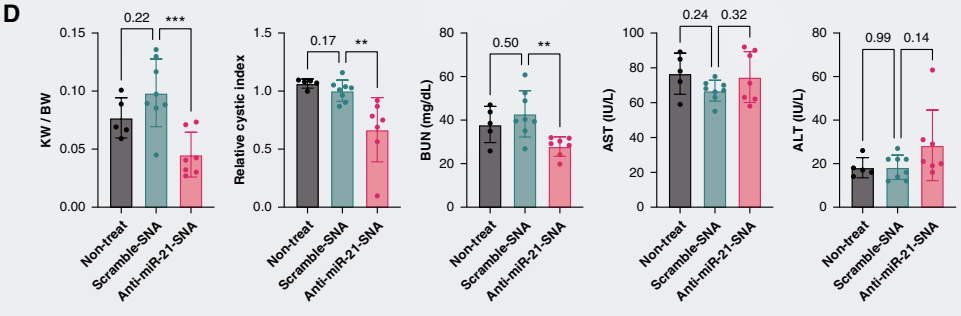
Anti-miR-21-SNA : (*R*) - A T C G a a u a G u C T G a C u a C a a C - (*S*)
Mature miR-21-5p : 5' - U A G C U U A U C A G A C U G A U G U U G A - 3'
a: 2,6-Diaminopurine u: 2-Thiouracil

Noda Y, Kato N, Sato F, et al. Ameliorative effect of an anti-microRNA-21 oligonucleotide on animal and human models of cystic kidney disease. *Kidney360*. 2025;6(6):900–913. doi:10.34067/KID.0000000771

Therapeutic effect of anti–miR-21-SNA in an ADPKD mouse model







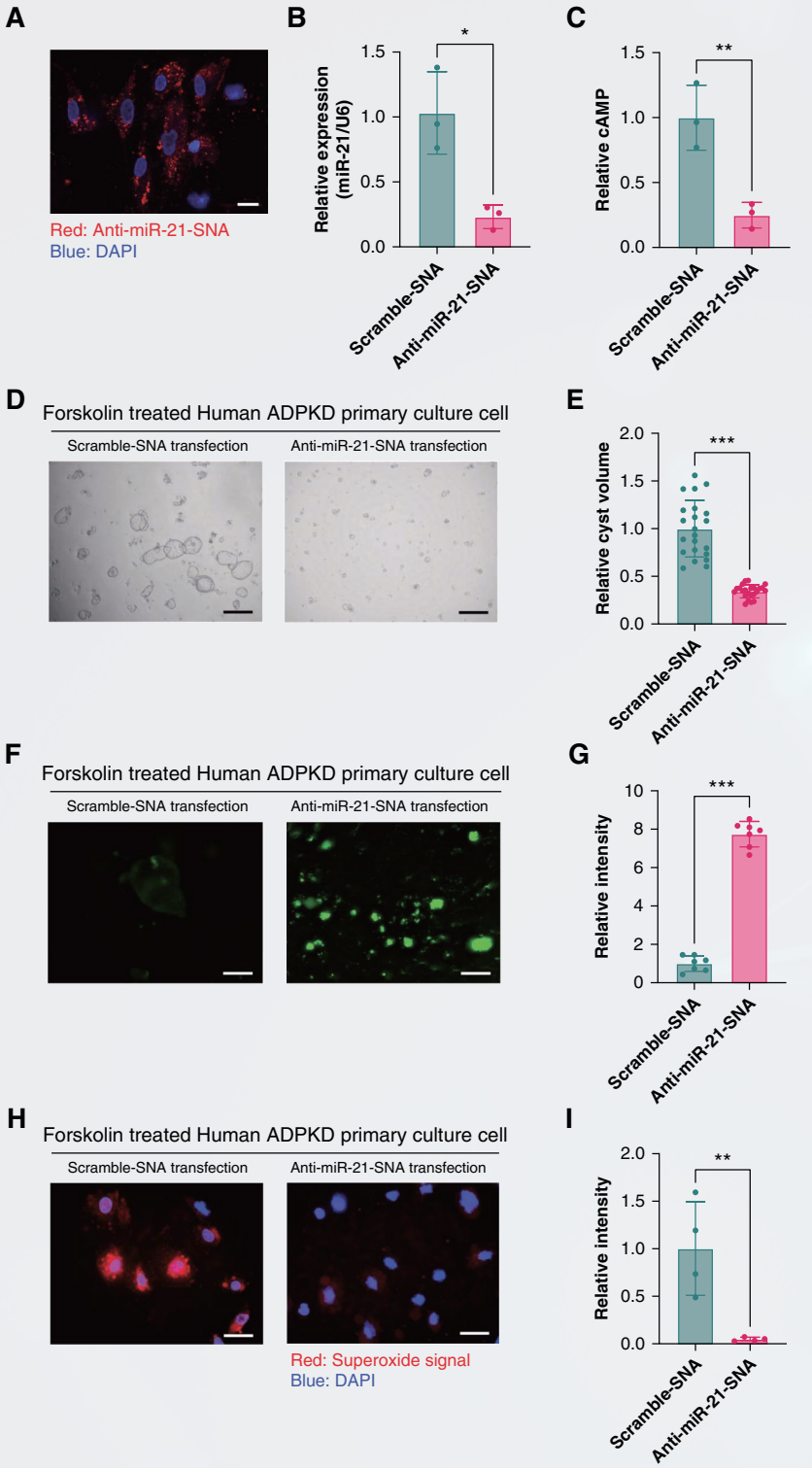
(A,B)

Weekly subcutaneous administration of anti–miR- 21-SNA significantly reduced the kidney weight to body weight (KW/BW) ratio compared to the scrambled control group, indicating suppression of kidney enlargement.

(C,D)

In addition, blood urea nitrogen (BUN) levels were significantly decreased in the treatment group, suggesting a protective effect on renal function and inhibition of disease progression.

Anti–miR-21-SNA suppresses cyst formation, abnormal signaling, and oxidative stress in human ADPKD primary cells



(A)
Cy5-labeled anti–miR-21-SNA was efficiently taken up into human primary renal epithelial cells.

(B)
Transfection resulted in a significant decrease in miR-21 expression.

(C)
Intracellular cAMP levels were significantly reduced, indicating inhibition of cystogenic signaling.

(D,E)
In 3D culture with forskolin stimulation, anti–miR-21-SNA significantly reduced cyst volume.

(F,G)
Intracellular calcium concentration increased following treatment, suggesting improvement in cellular function.

(H,I)
Mitochondrial superoxide production, a marker of oxidative stress, was significantly suppressed.

These findings demonstrate that anti–miR-21-SNA effectively reduces cyst burden, normalizes cell signaling, and alleviates oxidative stress in human ADPKD primary cells, supporting its potential as a therapeutic agent.

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