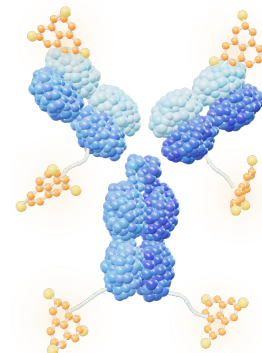


HUMAN NAMkind™ ADC

Which part of your ADC drives the signal?

Efficacy and safety testing for antibody-drug conjugates in **normal human** and **patient-derived disease** tissue — liver, ileum and colon.



● Antibody ● Linker ● Payload

91%

Liver safety accuracy
DILI, 92 clinical drugs

96%

GI safety accuracy
vs clinical diarrhea

100%

MASH concordance
6 reference drugs

7

Clinical ADCs tested
Human liver & gut

VivoSim platform validation data. Metrics apply to the evaluated reference sets.

One platform. Two questions.

SAFETY · NORMAL HUMAN TISSUE

Will it hurt the patient?

Healthy-donor liver, ileum and colon.

Liver	ATP · LDH · Albumin · GSH
Ileum & colon	TEER · ATP · Alamar Blue · histology
Output	Margin of safety vs clinical C_{max}
Validation	91% DILI · 96% GI accuracy

EFFICACY · HUMAN DISEASE MODELS

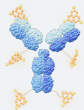
Does it work in diseased tissue?

Patient-derived MASH liver, UC & Crohn's.

MASH liver	P1NP · Sirius Red · Albumin
IBD intestine	TEER · IL-6 / IL-8 · P1NP · histology
Benchmarks	Resmetirom · Tofacitinib · Upadacitinib
Donors	15–16 MASH and IBD patient donors

Same primary human cells, 3D architecture and 96-well format for safety and efficacy.

Deconstruct your ADC. Find what drives the signal.



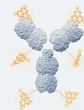
1 · Intact ADC
The molecule you plan to dose



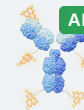
2 · Free payload
Or linker-payload / key catabolite



3 · Naked antibody
Does target binding alone hurt tissue?



4 · Non-binding ADC
Isotype control conjugate



5 · Benchmark ADC
Known clinical profile, plus vehicle

Same donor tissue · same plate · matched exposure around clinical C_{max} · repeat dosing 7–21 days.

Bring your ADC program.

DETAILS ON REVERSE →

Start with an ADC you already understand — a conjugate with a known clinical profile, plus its free payload.

HUMAN NAMkind™ ADC

Resolve your ADC into antibody and payload.

One study, five arms, matched exposure — same donor tissue, same plate, in liver, ileum, colon or all three.

What the ADC data already shows

Linker and payload change the liver signal

T-DXd (cleavable, DAR 8): full concentration–response; albumin already low at clinical C_{max} . **T-DM1** (non-cleavable, DAR 4): partial effect, albumin ~60% of vehicle at C_{max} . **Trastuzumab alone**: no significant change.

Payload drives the gut risk

T-DXd is toxic in ileum and colon, concentration-dependently; **free deruxtecan** alone is toxic; naked trastuzumab is only mildly active in ileum. Ileum responds at lower concentrations than colon.

A negative you can interpret

BCMA is not expressed in gut. **Belantamab mafodotin** gave no signal up to its 43 $\mu\text{g/mL}$ clinical C_{max} , while **free mafodotin** was toxic (TEER IC_{50} 115 $\mu\text{g/mL}$).

ADCs profiled: gemtuzumab ozogamicin, trastuzumab emtansine, trastuzumab deruxtecan, trastuzumab rezetecan, brentuximab vedotin, enfortumab vedotin, belantamab mafodotin.

Built around your program

Exposure, done properly

Dose in ADC molarity and payload-equivalent molarity, bracketing a justified range around clinical C_{max} .

What your team receives

Margin of safety for every arm vs C_{max} , a clear read on which component drives the signal, raw data and slides.

Chronic, not a snapshot

Repeat dosing for 7–21 days; target expression checked in the tissue before dosing.

No campaign queue

Work starts when compounds and PO arrive. Report in three weeks, with a senior-scientist readout call.

Build a study around your next decision.

01 / MEET

20 minutes to agree the construct, the tissue and the decision the data should inform.

02 / BOUNDED PILOT

Intact ADC, free payload, naked antibody and vehicle at matched exposure — liver, gut or both.

03 / RANK CANDIDATES

Compare linker-payload options across your pipeline on the same human tissue.

An exploratory, fit-for-purpose in-vitro comparison. It informs and prioritizes in-vivo toxicology; it does not replace it.

Let's design your ADC study.

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SCAN TO
CONTACT US

