BOUNDLESS BIO™

Unlocking a New Paradigm in Cancer Treatment via ecDNA-Directed Therapies (ecDTx)

Corporate Presentation

May 2026

Nasdaq: BOLD

@BoundlessBio

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This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

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Boundless Bio (BOLD): clinical-stage public company establishing a new category in oncology that addresses oncogene amplified cancers

Oncogene amplified cancer:

- Significant unmet medical need (worse survival)
- Generally unresponsive to targeted therapy and immunotherapy
- ~1.3M new patients per year in major markets[1]

Extrachromosomal DNA (ecDNA):

- Cancer-specific circular DNA—a root cause of oncogene amplification
- Transformative emerging area of cancer biology
- Spyglass drug discovery platform leverages ecDNA to identify synthetic lethal targets in cancer

ecDNA-directed therapies (ecDTx):

- BBI-940: oral Kinesin degrader development candidate; FIH KOMODO-1 Phase 1 clinical trial underway
- Preclinically validated ecDNA-selective targets for ecDTx discovery

Strong Foundation:

- Experienced team; track record of precision oncology drug approvals, multi-$B M&A
- Leading scientific founders, board, advisors
- Cash runway into 2H28; through expected clinical POC for BBI-940

1. United States, EU, and Japan

ecDTx: ecDNA-directed therapy
FIH: first in human
POC: proof of concept

ecDNA are cancer-specific, circular units of DNA that are a frequent driver of oncogene amplified cancer

Normal chromosome

EGFR

Unaltered gene

Transformation to cancer

Altered oncogene

Mutation

EGFR

OR

Fusion

BCR

ABL

- ~14-18% of cancer patients have mutations or fusions
- 60+ FDA approved drugs
- ~$50B in annual sales globally

Amplified oncogene

Chromosomal amplification

EGFR

extrachromosomal DNA (ecDNA) amplification

EGFR EGFR EGFR EGFR

- 25% of cancer patients have amplified oncogenes
- No approved therapies, except HER2 inhibitors (over $10B in annual sales)
- **~50% of high-copy number amplifications are on ecDNA**

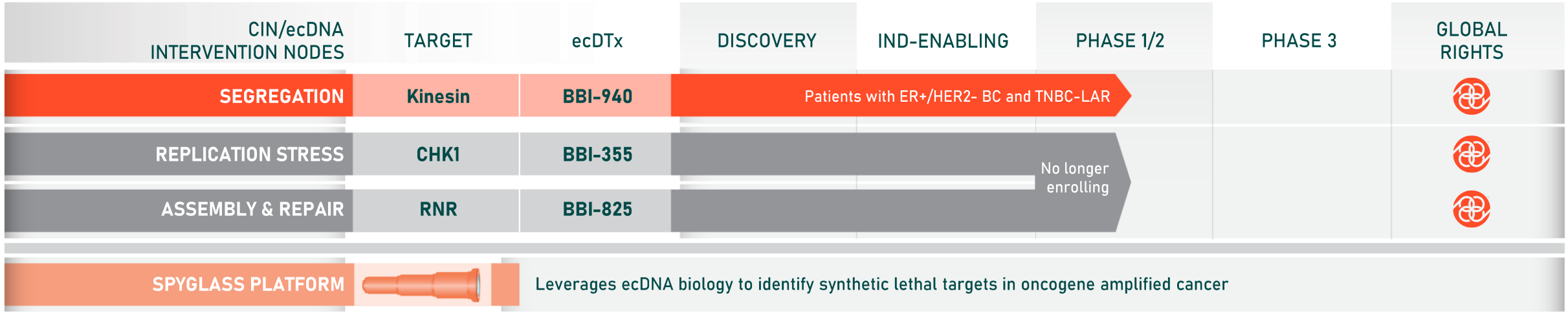
NEXT FRONTIER OF ONCOLOGY

~1.3m patients per year[1]
Oncogene amplified cancer

~750k patients per year[1]
ecDNA-enabled cancer™

1) Patients in US, EU, and Japan; numbers of patients based on third-party sources and internal estimates

Next-generation precision oncology pipeline to address high unmet needs in oncogene amplified cancer



ER+: ER positive; HER2-: HER2 negative; BC: breast cancer; TNBC: triple-negative breast cancer; LAR: luminal androgen receptor; ecDTx: ecDNA-directed therapeutic candidates

BC: breast cancer
CHK1: checkpoint kinase 1
CIN: chromosomal instability
ecDNA: extrachromosomal DNA
RNR: ribonucleotide reductase
TNBC-LAR: triple negative breast cancer, luminal androgen receptor

Accomplished leadership team has proven experience delivering value for patients and shareholders

Zachary Hornby

Chief Executive Officer, President, Director

ignyta · Roche · Fate Therapeutics · Halozyme · Neurocrine Biosciences · ROZLYTREK entrectinib 100mg | 200mg capsules

Christian Hassig, PhD

Chief Scientific Officer

SIERRA ONCOLOGY · Sanford Burnham Prebys · kalypsys · Scripps Research · Ojjaara (momelotinib) 200mg · 150mg · 100mg tablets

Robert Doebele, MD, PhD

Chief Medical Officer

rain ONCOLOGY INC · Anschutz · ROZLYTREK entrectinib 100mg | 200mg capsules · VITRAKVI (larotrectinib)

Jessica Oien, JD

Chief Legal Officer

CIDARA THERAPEUTICS · OTONOMY · élan · Somaxon PHARMACEUTICALS

David Hinkle, CPA

SVP, Finance & Controller

Crinetics Pharmaceuticals · ignyta · receptos · Somaxon PHARMACEUTICALS

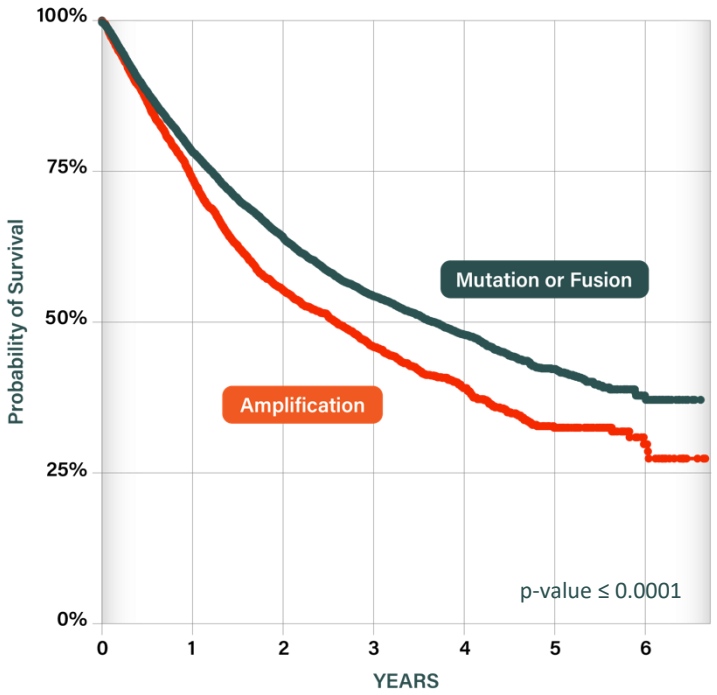
Significant unmet need in oncogene amplified cancers

Cancers with oncogene amplifications: more aggressive, difficult to treat, and worse prognosis

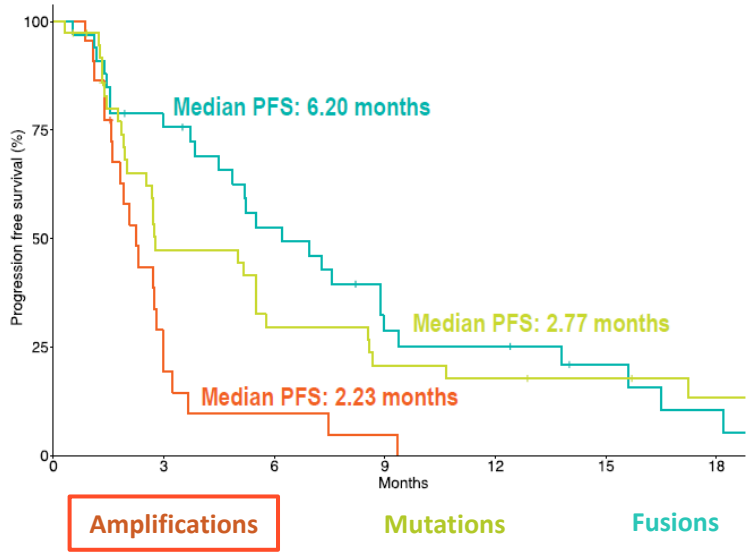
Oncogene amplified cancers

- Oncogene amplification is an oncogenic alteration where extra copies (>2) of a gene (e.g., *EGFR*) drive tumor growth or resistance
- Patients with oncogene amplifications have worse survival than other cancer patients
- Unlike other alterations, oncogene amplified tumors are generally unresponsive to targeted therapies and immunotherapies

Survival of cancer patients, segmented by oncodriver status[1]



PFS of cancer patients with *FGFR* alterations treated with FGFR inhibitors



*Patients with primary or metastatic cancers with **amplifications, point mutations, skipping deletions or fusions** of these genes: AR, ALK, ARAF, BRAF, CCND1, CDK4, CDK6, EGFR, ERBB2, FGFR1, FGFR2, FGFR3, FGFR4, HRAS, IDH1, IDH2, KIT, KRAS, MAP2K1, MAP2K2, MAP2K4, MDM2, MET, MYC, NF1, NRAS, NTRK1, NTRK2, NTRK3, PDGFB, PDGFRA, PDGFRB, PIK3CA, RAF1, RET, ROS1*

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1. cBioPortal analysis using MSK-IMPACT (N=14,674 patients) and MSK-MET (N=1,115 patients) data; p-value = < 0.0001
PFS: progression free survival

Pobel et. al. *ESMO TAT Virtual Congress*. 2021

Despite advancements in precision medicine, cancers with oncogene amplifications generally do not respond to targeted therapies

TARGETED THERAPY	TARGET	APPROVED	NO APPROVAL
IBRANCE palbociclib, Verzenio abemaciclib, KISQALI ribociclib 200 mg tablets	CDK4/6	HR+/HER2- breast cancer	Amplification
IRESSA gefitinib, GILOTRIF (afatinib) tablets, RYBREVANT (amivantamab-vmjw) Injection for IV Use | 350 mg/7 mL (50 mg/mL), Tarceva erlotinib, TAGRISSO osimertinib, EXKIVITY mobocertinib 40 mg capsules	EGFR	L858R NSCLC T790M NSCLC Exon 19 deletion NSCLC Exon 20 insertion NSCLC	Amplification
Balversa (erdafitinib) 3, 4, 5 mg tablets, Pemazyre (pemigatinib) tablets 13.5mg-9mg-4.5mg, LYTGOBI (futibatinib) tablets	FGFR	*FGFR3* mutation bladder cancer *FGFR2/3* fusion cholangiocarcinoma	Amplification

A new approach is needed to treat cancers driven by oncogene amplifications

Improvements in cancer drugs drive increased rates of resistance via oncogene amplification

Patients with ≥ 1 amplification at resistance

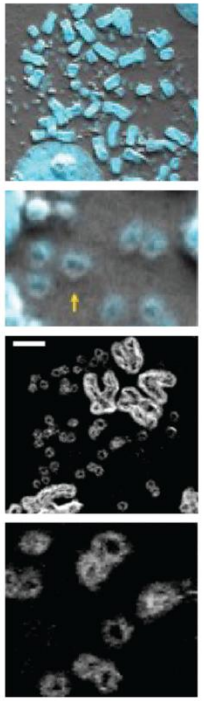
Indication	Previous-gen inhibitors	Next-gen inhibitors	References
EGFR-mutated non-small cell lung cancer (NSCLC)	erlotinib or gefitinib **15/145 (10%)**	osimertinib **24/109 (22%)**	Chmielecki et al., *Nat Comm* (2023)
ALK+ NSCLC	crizotinib **6/90 (7%)**	lorlatinib **9/31 (29%)**	Solomon et al., *JCO* (2024)
ROS1 fusion NSCLC	crizotinib **7/42 (17%)**	crizotinib → lorlatinib **8/28 (29%)**	Lin et al., *Clin Cancer Res* (2021)
KRAS^{G12C} colorectal cancer (CRC)	adagrasib + cetuximab **13/34 (38%)**	divarasib + cetuximab **11/14 (79%)**	Desai et al., *Nat Med* (2023); Yaeger et al., *Canc Disc* (2024)
BRAF^{V600E} CRC	chemo + cetuximab **0/94 (0%)**	encorafenib + binimetinib + cetuximab **22/112 (20%)**	Kopetz et al., *Nat Med* (2024)
Pancreatic ductal adenocarcinoma (PDAC)	adagrasib or sotorasib (in KRAS^{G12C}) **6/22 (27%)**	daraxonrasib **18/44 (41%)**	Dilly, et al., *Cancer Discovery* (2024); Aronchik, et al., *ANE poster* (2025)
Additionally, emerging preclinical evidence and retrospective clinical studies indicate **ABC transporter** amplification/up-regulation may drive resistance to **chemotherapy** and **ADCs**, revealing a **new targetable resistance pathway**.			Bergonzini, et al., *J Exp Clin Cancer Res* (2024); Sledge, et al., *SABCS poster* (2024); *Boundless Bio unpublished data*

Despite advanced targeted therapies, **amplification-driven resistance** remains a challenge across many solid tumor indications

ecDNA: a key driver of oncogene amplifications

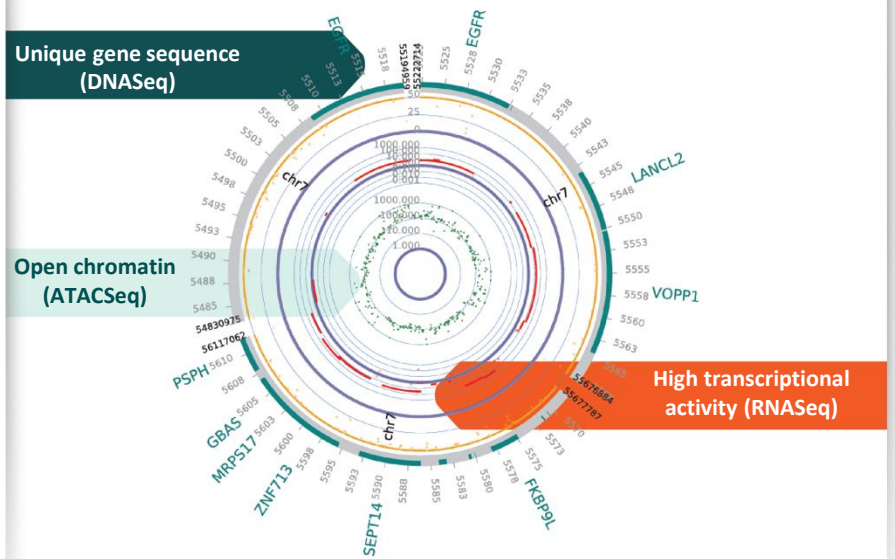
ecDNA are a primary driver of oncogene amplified cancers and enable resistance to targeted therapies

ecDNA are circles of DNA, distinct from chromosomes, that amplify full-length genes and regulatory elements



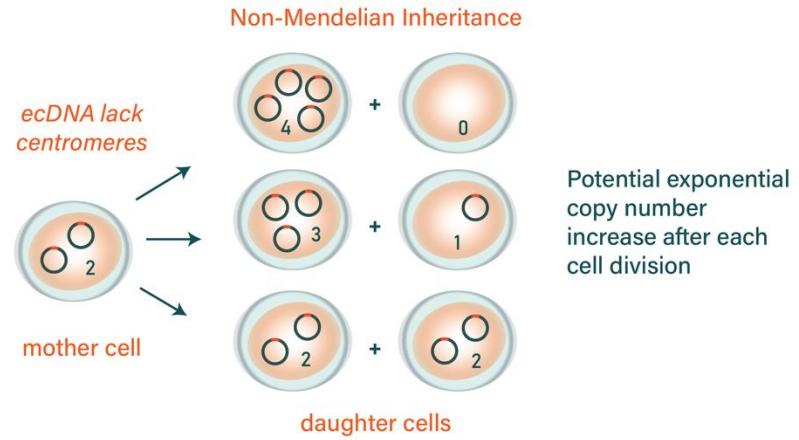
Large size: 2 – 5 Mbp

Circular shape of ecDNA enhances transcriptional activity, leading to high oncogene expression

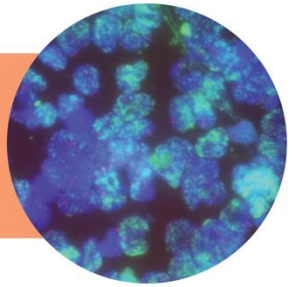


Active Gene Expression

ecDNA asymmetrically segregate during mitosis, enabling exponential gene copy number increase or decrease during cellular division



Protein products of genes amplified on ecDNA can provide a fitness advantage – driving cancer growth and resistance



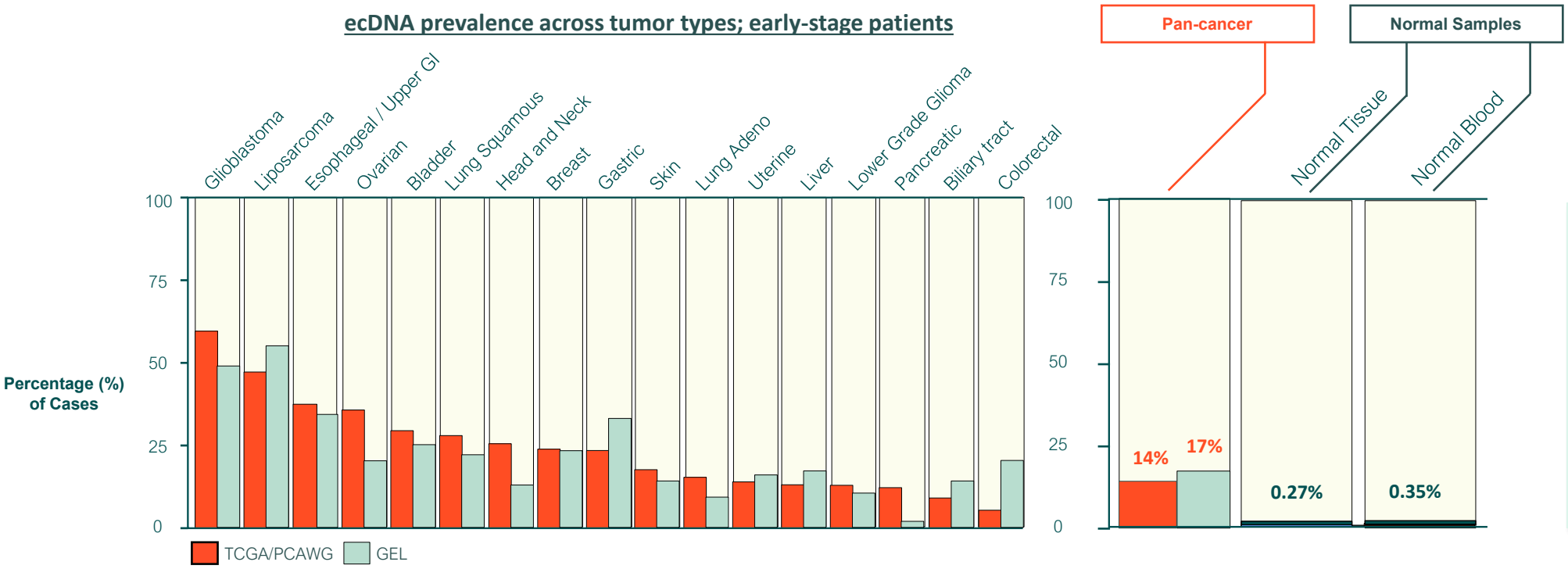
MYCN amplifications on ecDNA in pediatric neuroblastoma

Mbp: mega base pairs

Wu, Turner… Verhaak, Chang, Bafna, Mischel. 2019 Nature

Multiple clinical datasets demonstrate that ecDNA are detected broadly across cancers

ecDNA are not found in normal tissue or blood



- **ecDNA are present in 14-17% of cancer specimens at diagnosis, across multiple tumor types**
- **ecDNA have negligible presence in normal tissue or blood**

Analysis of WGS data from >3,000 tumor and matched normal samples TCGA and PCAWG; ~15,000 tumor samples from GEL

Kim, Nguyen, Turner, et al. 2020 Nature Genetics
Bailey, Mischel, Swanton, et al. 2024 Nature

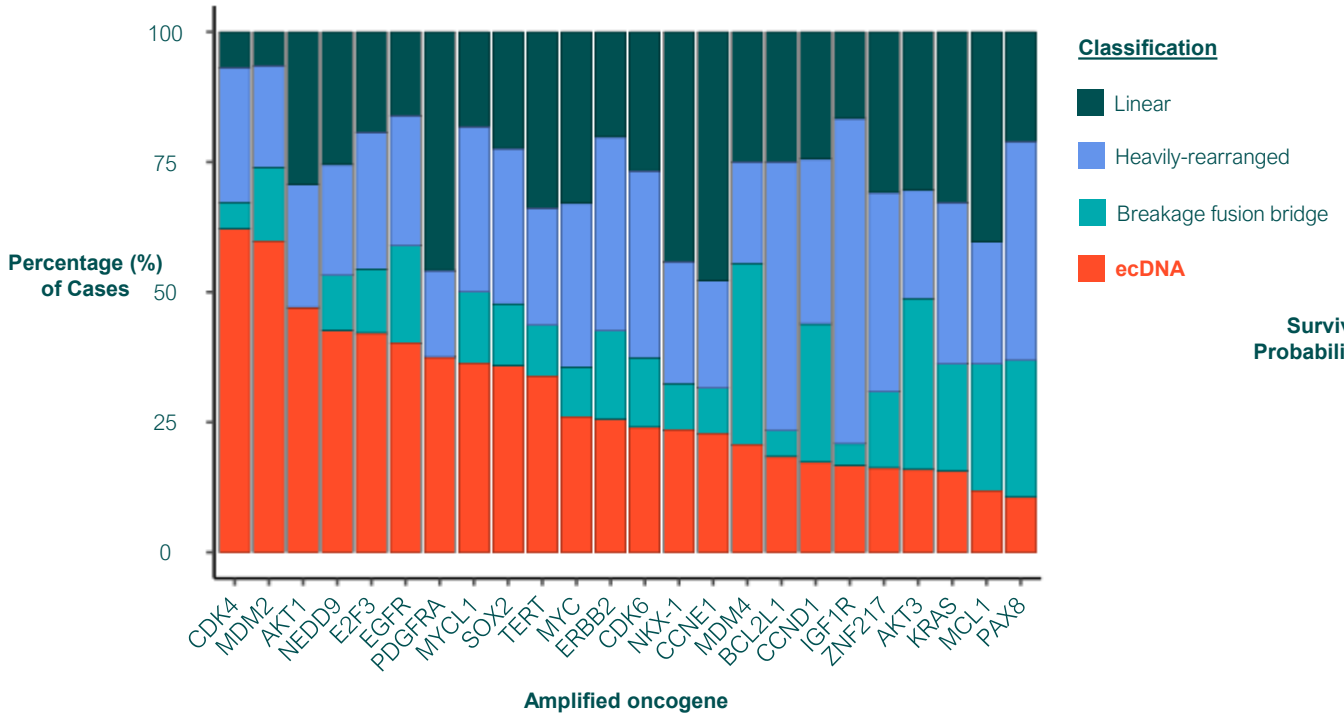
GEL: Genomics England
PCAWG: Pan-Cancer Analysis of Whole Genomes

TCGA: The Cancer Genome Atlas
WGS: whole genome sequencing

Cancer's most common high copy number oncogene amplifications frequently occur on ecDNA; when they do, it is associated with worse survival

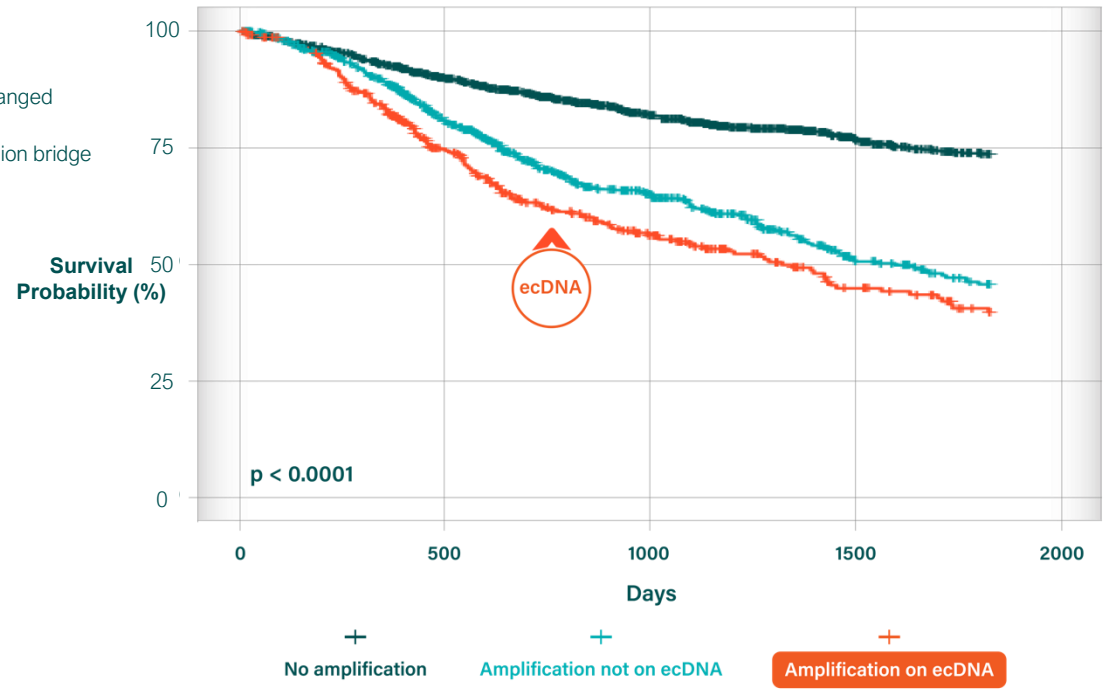
~54% of high-copy number oncogene amplifications are detected on ecDNA

Most frequently amplified oncogenes, segmented by amplification type



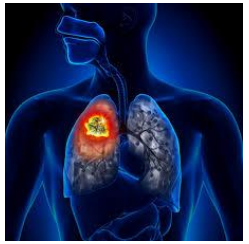
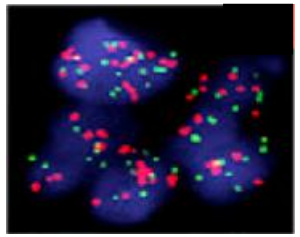
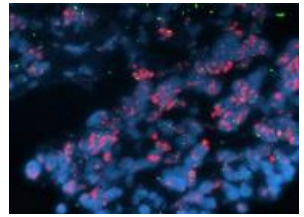
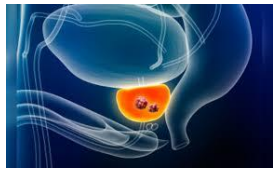
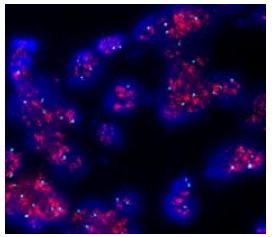
Patients with oncogene amplification *on ecDNA* have worse survival

Survival of cancer patients, segmented by gene amplification status



Adapted from Kim, Nguyen, Turner, et al. 2020 Nature Genetics
High copy number defined as ≥8 gene copies

Oncogene amplifications on ecDNA are a frequent mechanism of clinical resistance to multiple therapeutic modalities

CANCER	INITIAL ONCOGENE DRIVER	CLINICAL THERAPY	ONCOGENE AMPLIFICATION DRIVING RECURRENCE	
NSCLC 	*ALK*^{fusion} *EGFR*^{mut} *ROS1*^{fusion} *KRAS*^{G12C}	ALK, EGFR, ROS1, KRAS^{G12C} inhibitors →	***KRAS*** ***ERBB2*** ***MET***	 *MET* (on ecDNA)
CRC 	*BRAF*^{mut} *KRAS*^{G12C}	BRAF^{V600E}, KRAS^{G12C} inhibitors → Anti-EGFR mAbs →	***KRAS*** ***MET*** ***BRAF*** ***EGFR***	 *KRAS* (on ecDNA)
CRPC 	*AR*	ASI →	***AR*** ***MYC***	 *AR* (on ecDNA)

- Wang 2019 J Hem Onc
- Takezawa 2012 Canc Disc
- Dagogo-Jack 2020 Clin Canc Res
- Schoenfeld 2020 Clin Canc Res
- Roper 2020 Cell Reports Med
- Priest 2023 npj Prec Onc
- Park 2024 Eur J of Can

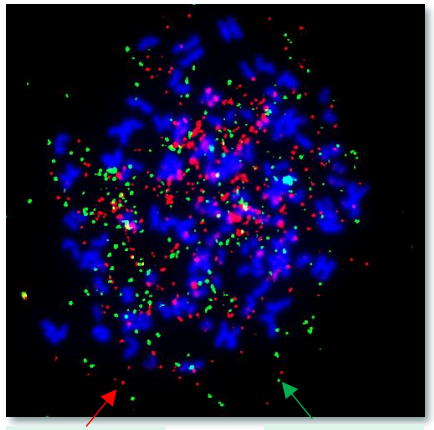
- Misale 2012 Nature
- Bardelli 2013 Canc Disc
- Yaeger 2017 Canc Res
- Awad 2021 NEJM
- Tan 2023 Clin Can Res
- Sacher 2023 NEJM

- Zhao 2024 Nat Gen
- Zivanovic 2023 NAR Can

ASI: androgen signaling inhibitor

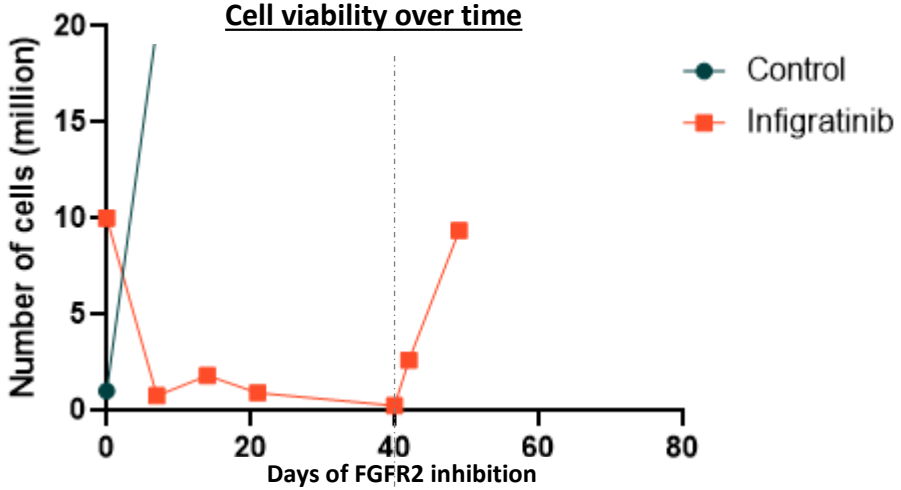
ecDNA enable cancer cells to become resistant to therapies by rapidly adapting oncogene dependency

Gastric cancer cells pre-treatment

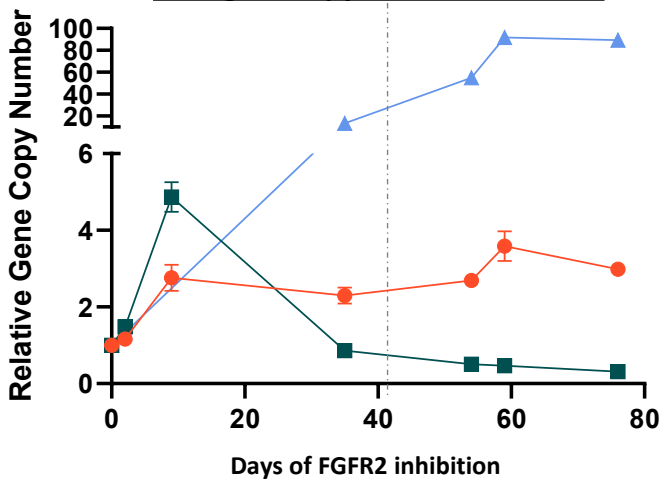


FGFR inhibitor (infigratinib)

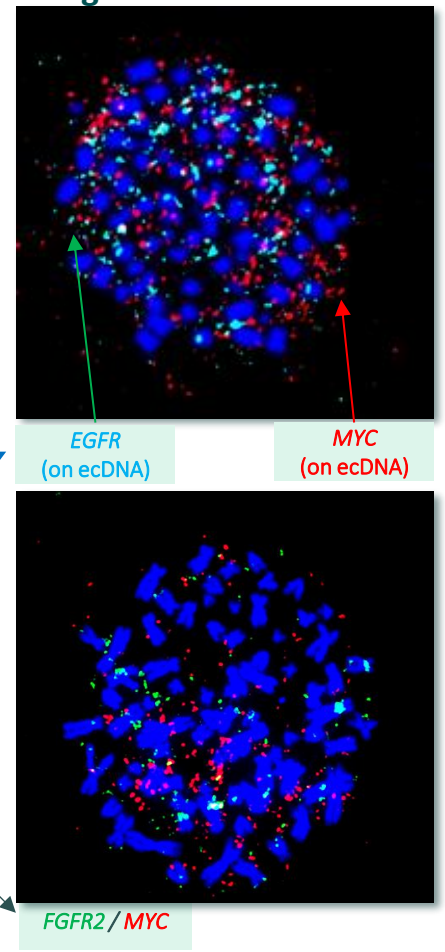
Cell viability over time



Oncogene copy number over time



FGFR inhibitor resistant gastric cancer cells

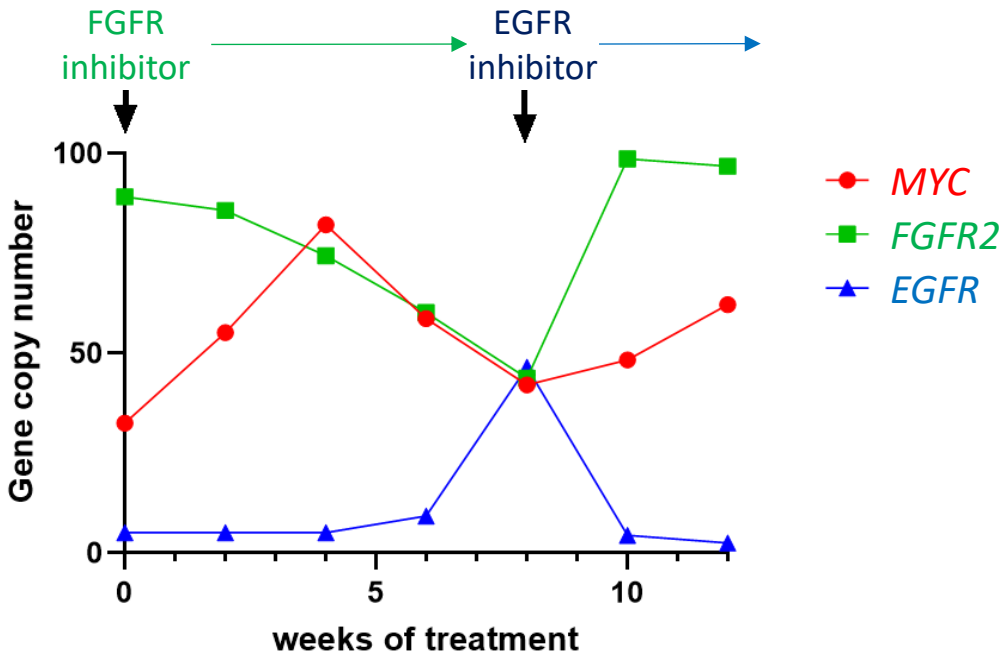


In these gastric cancer cells, ecDNA enable a rapid switch of oncogene dependency from *FGFR2* to *EGFR* under therapeutic pressure

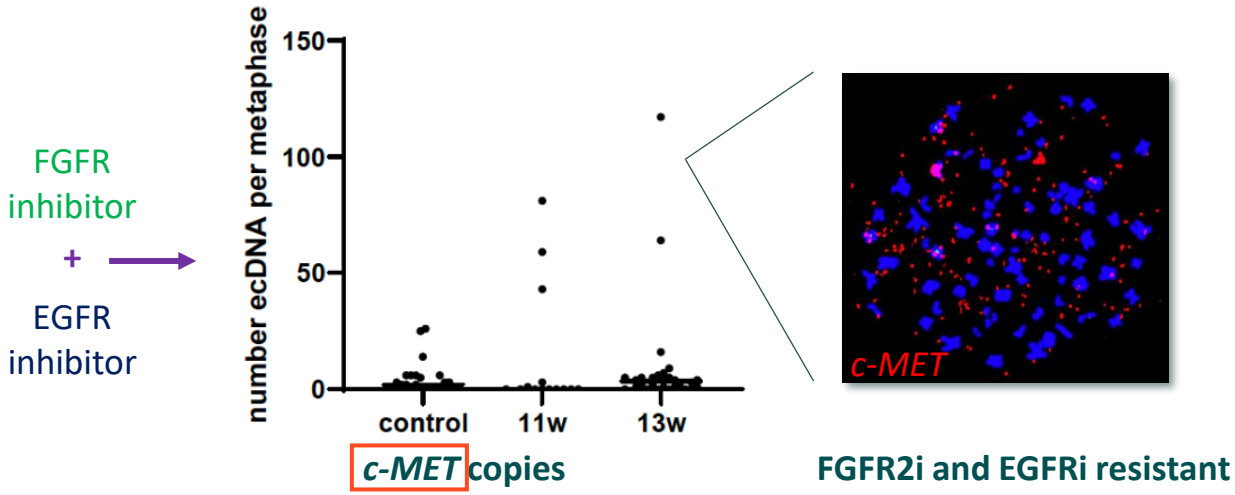
Turner, et al. Cancer Res 2021.

Tumors employ ecDNA to rapidly enable new oncogenic drivers, even under combination target inhibition

Oncogene copy on ecDNA changes dynamically in response to *sequential targeted therapeutic pressure*



New oncogene populations can arise on ecDNA in response to *combination targeted therapeutic pressure*



Inhibition of EGFR results in return of *FGFR2* => ecDNA amplification supports oncogenesis

Simultaneous dual inhibition of FGFR2 and EGFR leads to ecDNA driven amplification of new oncogene (*c-MET*)

Targeting only the oncogenes amplified on ecDNA is a futile therapeutic approach due to ecDNA-enabled process of continuous and rapid oncogene dependency switching

Turner, et al. Cancer Res 2021.

Driving a new treatment paradigm by targeting ecDNA pathways that enable tumor evolution and resistance

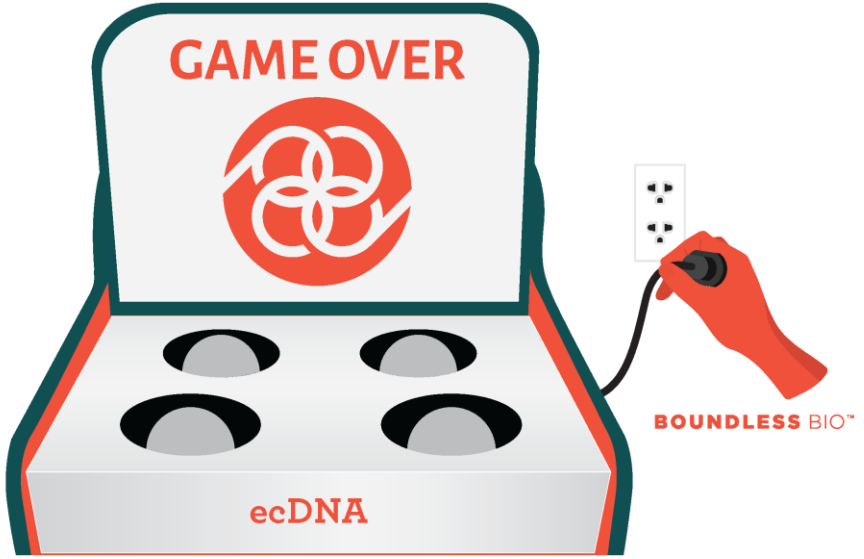
Traditional Targeted Therapy:

Develop targeted inhibitors against activated oncodriver targets, but cells typically develop **resistance**

Applying traditional targeted therapy approach to ecDNA enabled cancers is clinical 'Whac-a-Mole'

Next Generation Precision Oncology:

Exploit underlying vulnerabilities in **ecDNA-driven cells** to drug targets essential for ecDNA functionality in cancer

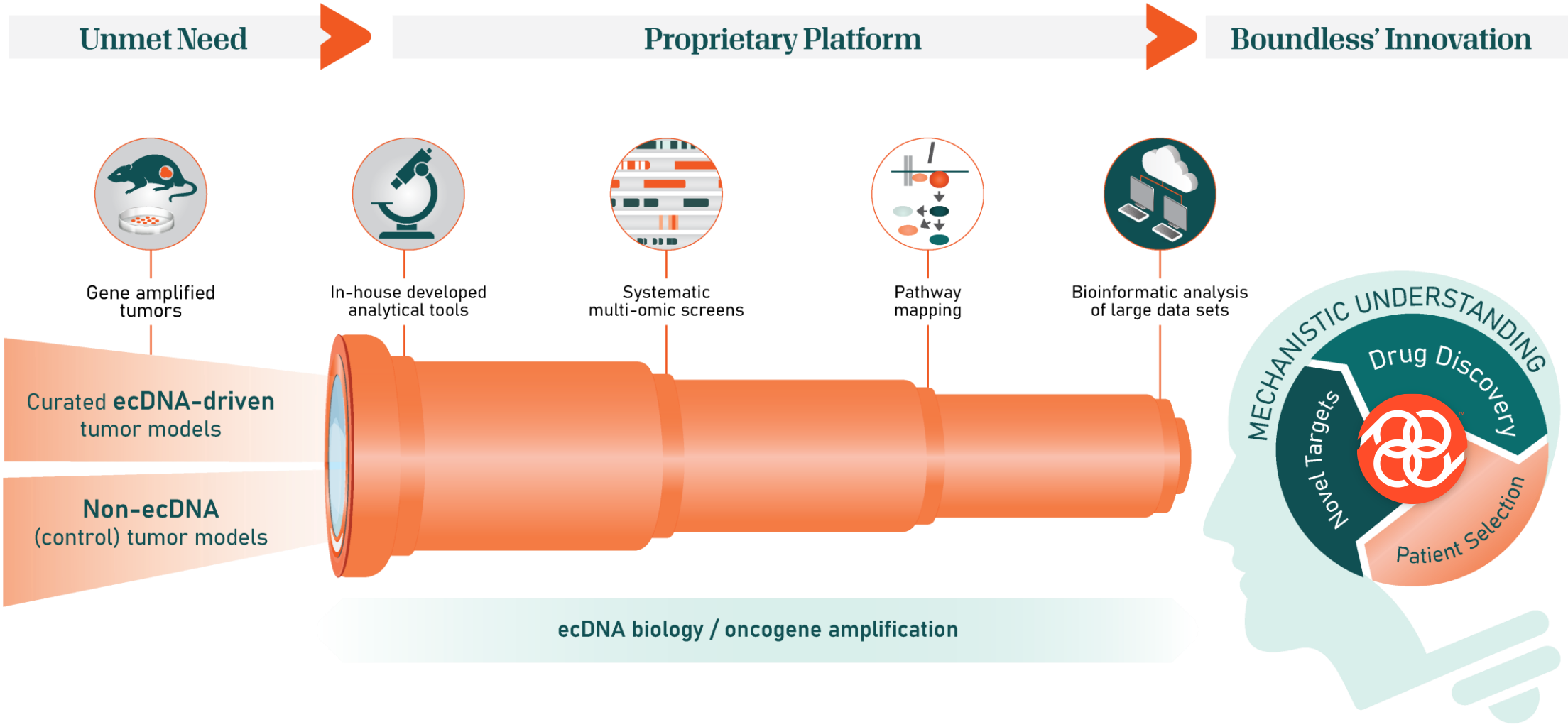


Disable ecDNA plasticity and/or function

- **Replication & transcription**
- **Assembly & repair**
- **Segregation**

Spyglass: unique platform that interrogates ecDNA biology to identify synthetic lethal targets in cancer

Proprietary target and drug discovery engine



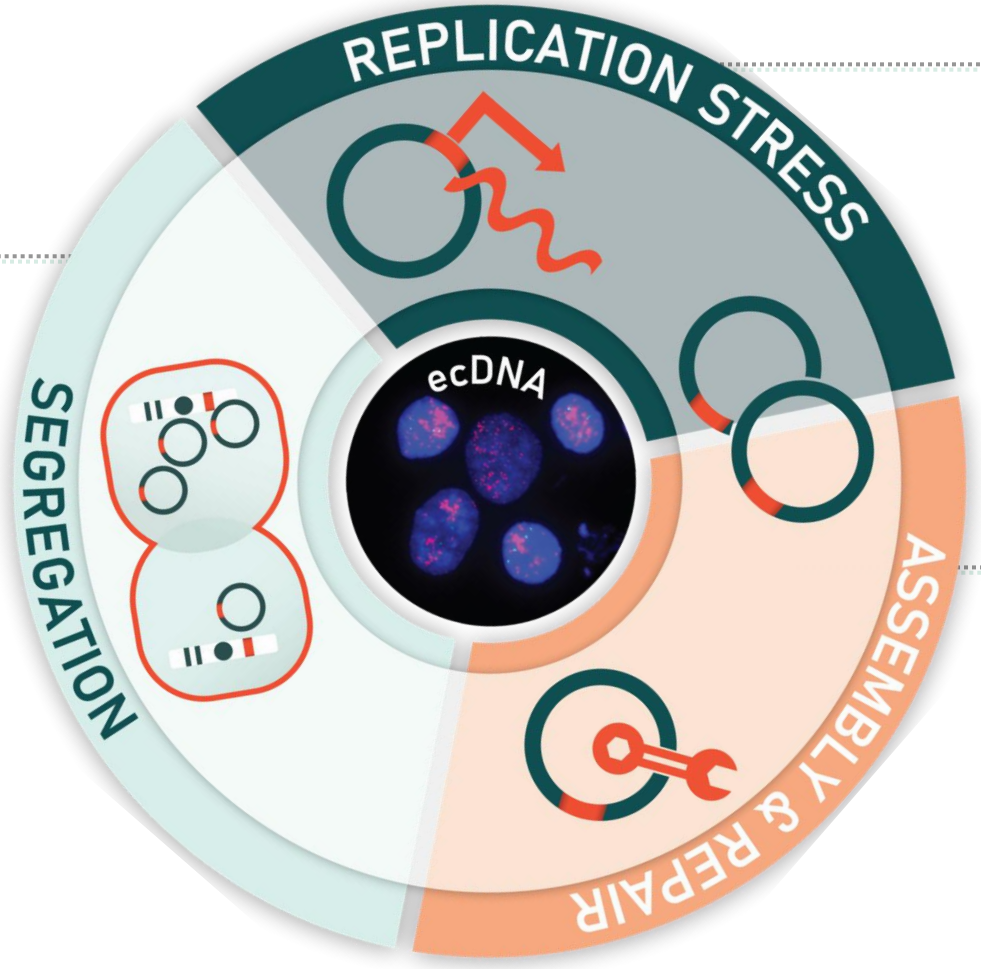
Spyglass reveals cancer targets, both novel and validated, that intersect distinct nodes of ecDNA lifecycle

Kinesin

BBI-940 (Phase 1 clinical)

Novel, oral, selective degrader of Kinesin

Kinesin is a target involved in segregation of DNA and critical for ecDNA segregation



CHK1

BBI-355

Novel, oral, selective inhibitor of CHK1

CHK1 is master regulator of replication stress, including that induced by ecDNA

RNR

BBI-825

Novel, oral, selective inhibitor of RNR

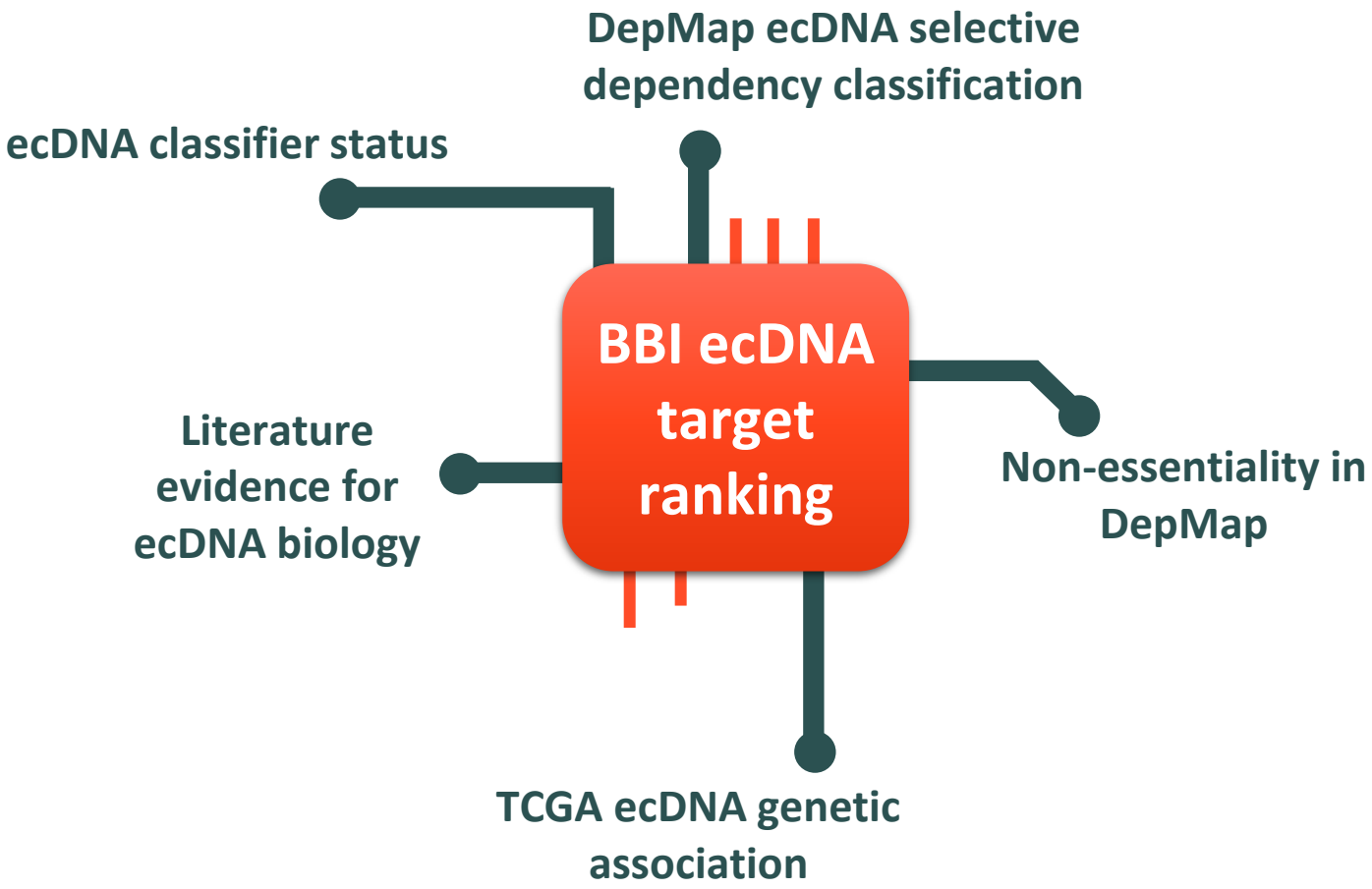
RNR is a rate-limiting enzyme for *de novo* synthesis of dNTPs, the raw materials of DNA, including ecDNA

CHK1: checkpoint kinase 1
dNTPs: deoxyribonucleotide triphosphates
RNR: ribonucleotide reductase

BBI-940: novel Kinesin oral degrader

Targets DNA segregation in cancer

Spyglass screening identified Kinesin as an ecDNA-associated target



BBI ecDNA target ranking

Heuristic scoring method combining internal data sets with large public resources identifying genes and pathways critical for ecDNA-high cancer lines

Pathway Enrichment Analysis

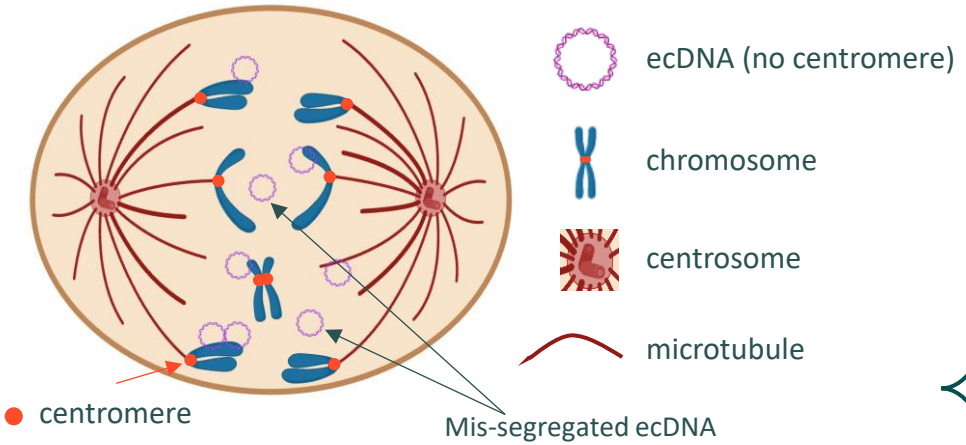
Kinesin
RRM1, RRM2

Revealed specific pathways strongly associated with ecDNA dependency

Novel kinesin target and RNR validated as ecDNA targets

RRM1/2: ribonucleotide reductase M1/2 subunits of RNR

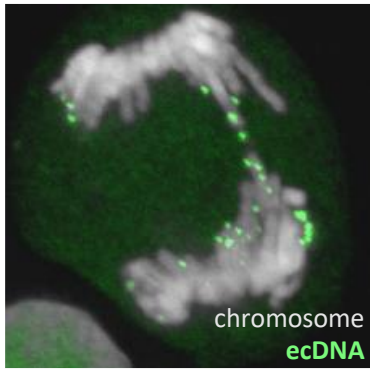
Kinesin regulates DNA segregation and the viability of ecDNA dependent cells



BioRender

- Chromosome segregation is primarily dependent on interactions between the mitotic spindle microtubules and the centromere
- ecDNA lack centromeres and likely rely on distinct mitotic machinery for proper segregation and ‘chromosomal hitchhiking’
- Spyglass has revealed a kinesin (“Kinesin”) that is non-essential for chromosome segregation in healthy cells, but is essential for proper ecDNA segregation and inheritance in cancer cells
- Genetic knockdown or degradation of “Kinesin” reduced ecDNA and showed synthetic lethality and robust anti-tumor activity in chromosomally unstable (CIN) and ecDNA-enabled cancer models
- We are unaware of any other efforts to drug Kinesin

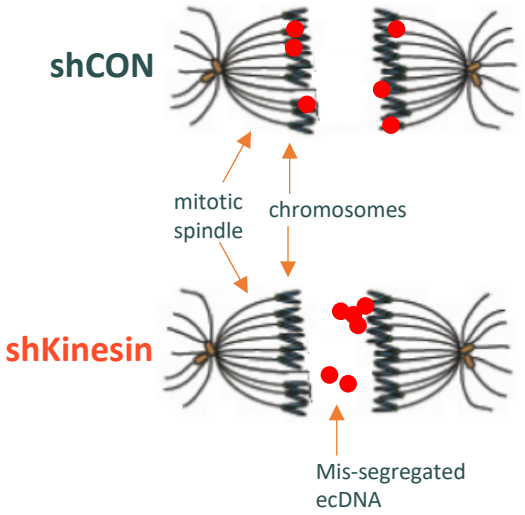
ecDNA ‘hitchhikes’ with chromosomes during mitosis



Oobatake and Shimizu, Genes Chrom Canc 2019

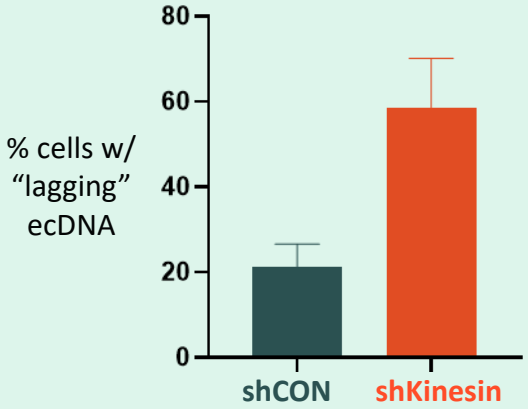
Genetic knockdown of Kinesin resulted in mis-segregation of ecDNA during mitosis and reduced cellular ecDNA levels over time

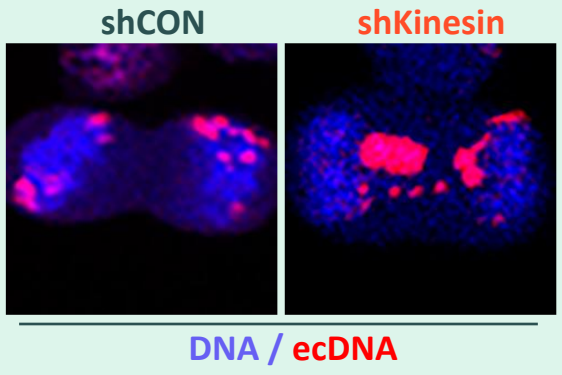
Model for Kinesin inhibition



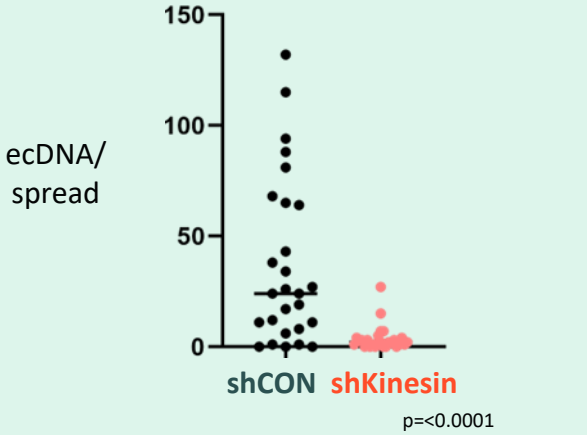
Kinesin hypothesized to interact with both ecDNA and chromosomes, independently of centromeres, to align DNA at the metaphase plate and promote segregation during mitosis

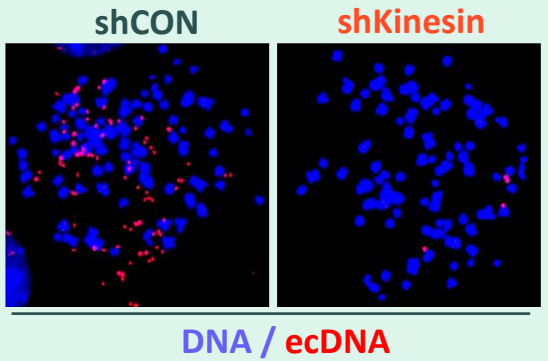
ecDNA displayed a "lagging" phenotype during mitosis in the absence of Kinesin



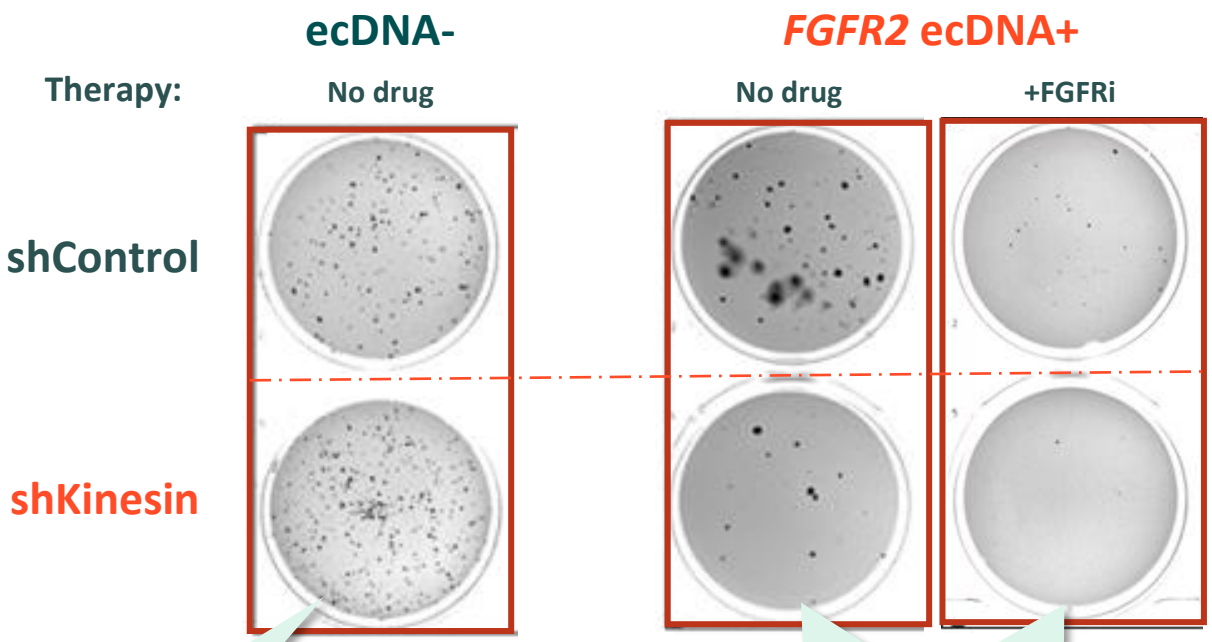


Knock down of kinesin led to reduced levels of ecDNA in cancer cells





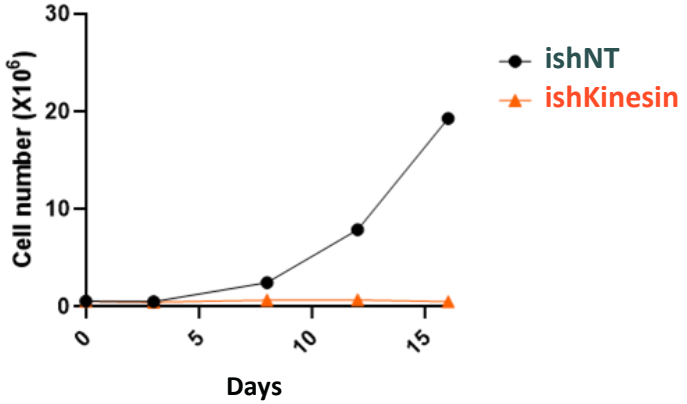
Genetic inactivation of Kinesin resulted in significant anti-proliferation of oncogene amplified tumor cell lines



No negative impact on colony growth in ecDNA- cells

Kinesin KD resulted in significant reduction in viability of ecDNA+ cells. Combination with targeted therapy substantially abrogated colony formation

Anti-tumor activity in *MYCN* amplified cancer cell line



Genetic inhibition resulted in antiproliferation and cytotoxicity in multiple cancer cell lines

ishNT: non-targeting in situ hybridization
KD: knockdown
sh: short hairpin (RNA interference)

Boundless identified a Kinesin inhibitor scaffold via high-throughput screens of >1M compounds

Extensive medicinal chemistry effort optimized potent, cell-active, heterobifunctional degraders

HTS/HTL

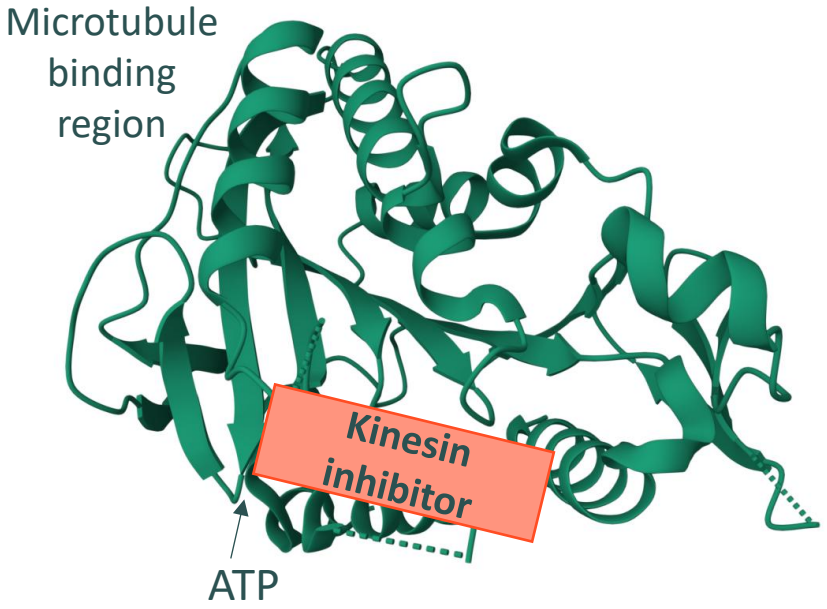
uM biochemical inhibitor series

Inhibitor/ligand SAR

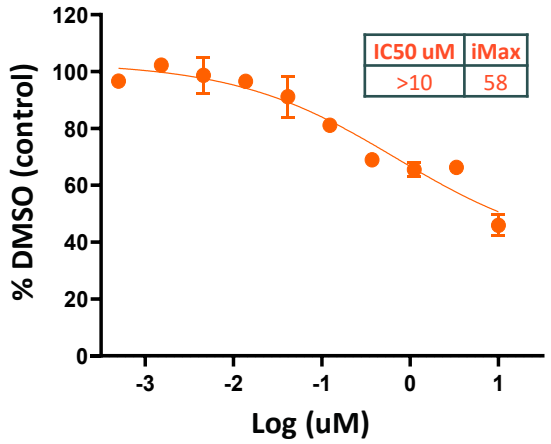
- nM inhibitors
- Selective motor domain binding
- Weak cellular activity

Degrader optimization

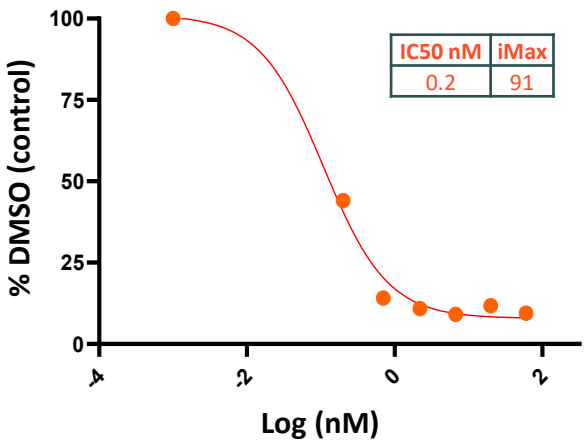
- nM heterobifunctional degraders
- Robust cellular and *in vivo* activity



Kinesin motor domain bound to inhibitor



Potent inhibitors



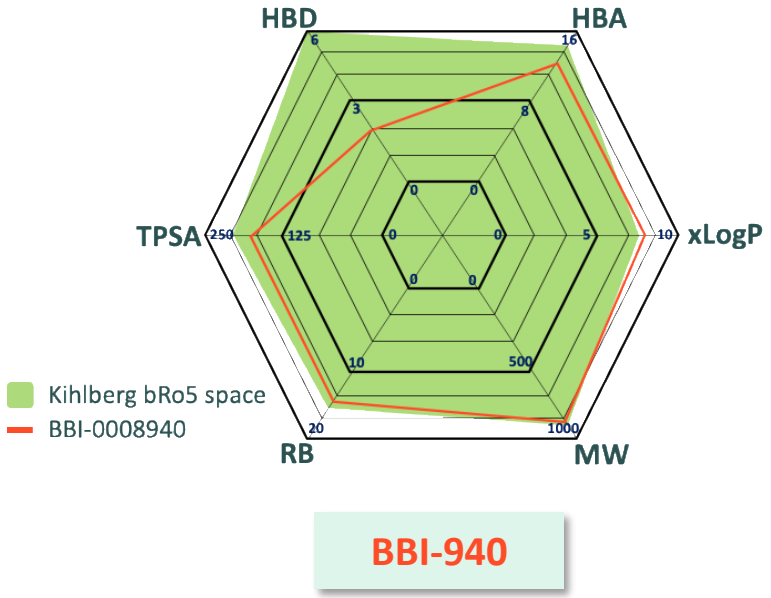
Potent degraders

HTL: hit-to-lead
HTS: high throughput screen
SAR: structure activity relationship

BBI-940 is a novel Kinesin oral degrader

Product Preclinical Property	TPP	BBI-940
Route of administration PO bioavailability (mouse/rat/dog)	Oral >20%	✓
In vitro potency (HiBit DC_{50}/DC_{90}, Dmax%)	<10nM/<100nM, >90%	✓
CYPi	>1uM	✓
hERG	>1000x over target	✓
Kinesin Selectivity/CEREP panel/proteomics	>100x/>100x/clean	✓
PD Response (*in vivo* % degradation)	>80%	✓
Efficacy	>70% TGI single agent	✓

Oral Druggable Space beyond Lipinski's Rule of Five*



BBI-940

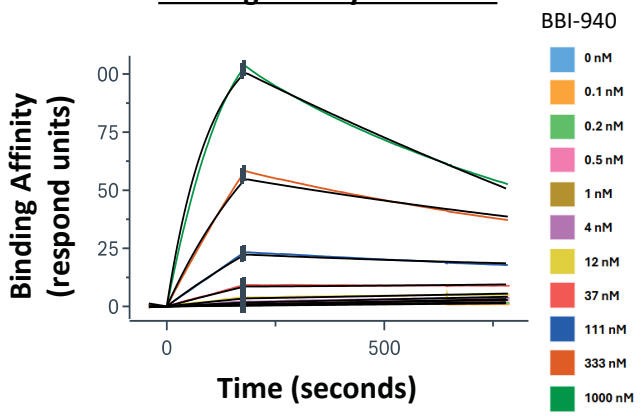
*Commonly used to develop oral degraders

HBA: hydrogen bond acceptors
HBD: hydrogen bond donors
xLogP: hydrophobicity

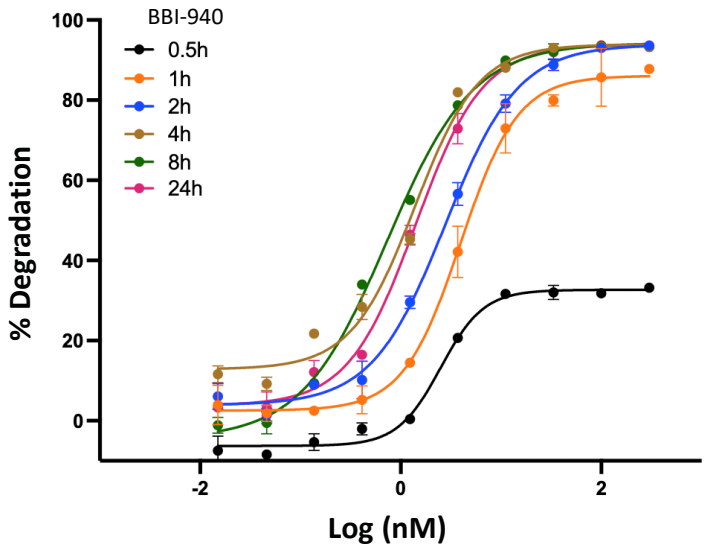
MW: molecular weight
RB: rotatable bonds
TPSA: topological polar surface area

BBI-940 demonstrated strong binding, potent inhibition, and remarkable degradation of Kinesin target, with favorable kinetics

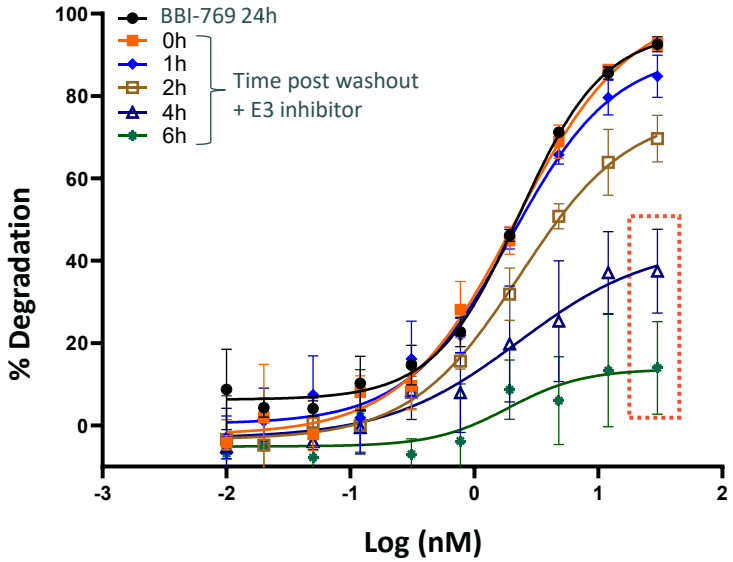
Binding affinity over time



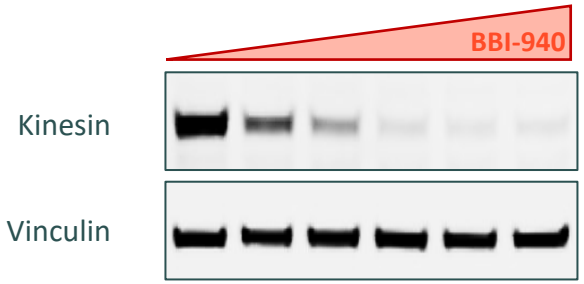
Max Kinesin degradation achieved by 2 hr



Near complete Kinesin resynthesis by 6 hr



Dose-dependent degradation

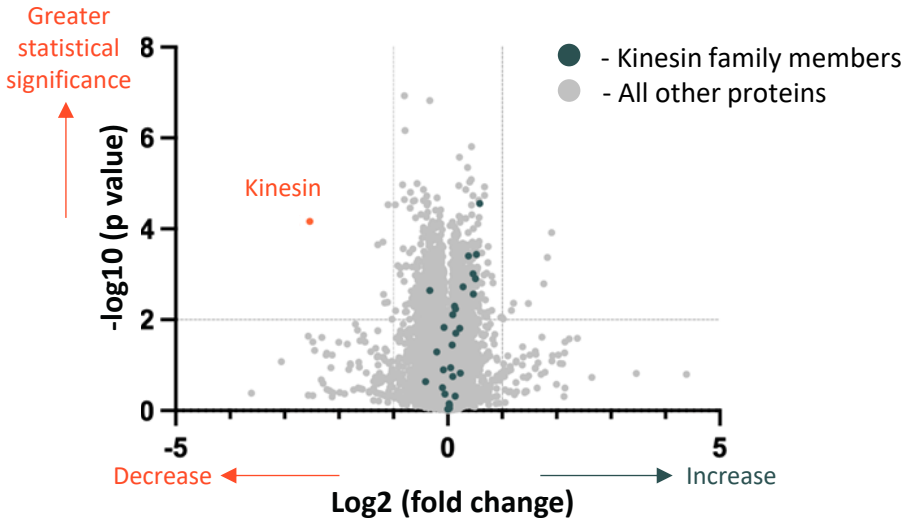


Fast kinetic degradation of Kinesin supports sufficient target coverage to overcome resynthesis rates of the protein

BBI-769: analog to BBI-940

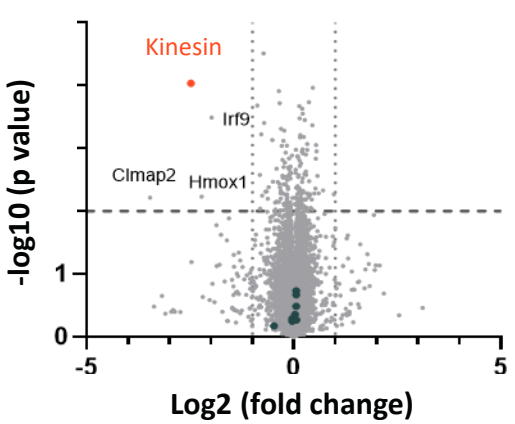
BBI-940 degraded cellular Kinesin with exquisite selectivity over other kinesins and cellular targets

Differentially expressed proteins after treatment with BBI-940 (100 nM following 8 hrs in CAL51 cells)

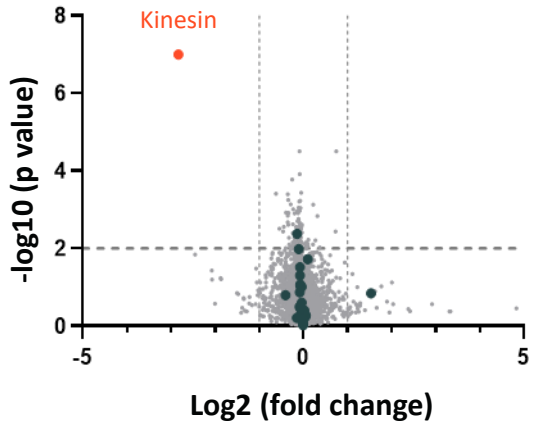


- **BBI-940 elicited rapid and selective degradation of only Kinesin in human breast cancer cell line**
- **BBI-940 demonstrated selectivity against broad receptor target panel**
- **BBI-940 displayed selective biochemical inhibition over all other kinesin family members, including KIF18A**
- **Proteomics in rat and dog cells demonstrated selective degradation and suitability as toxicology species**

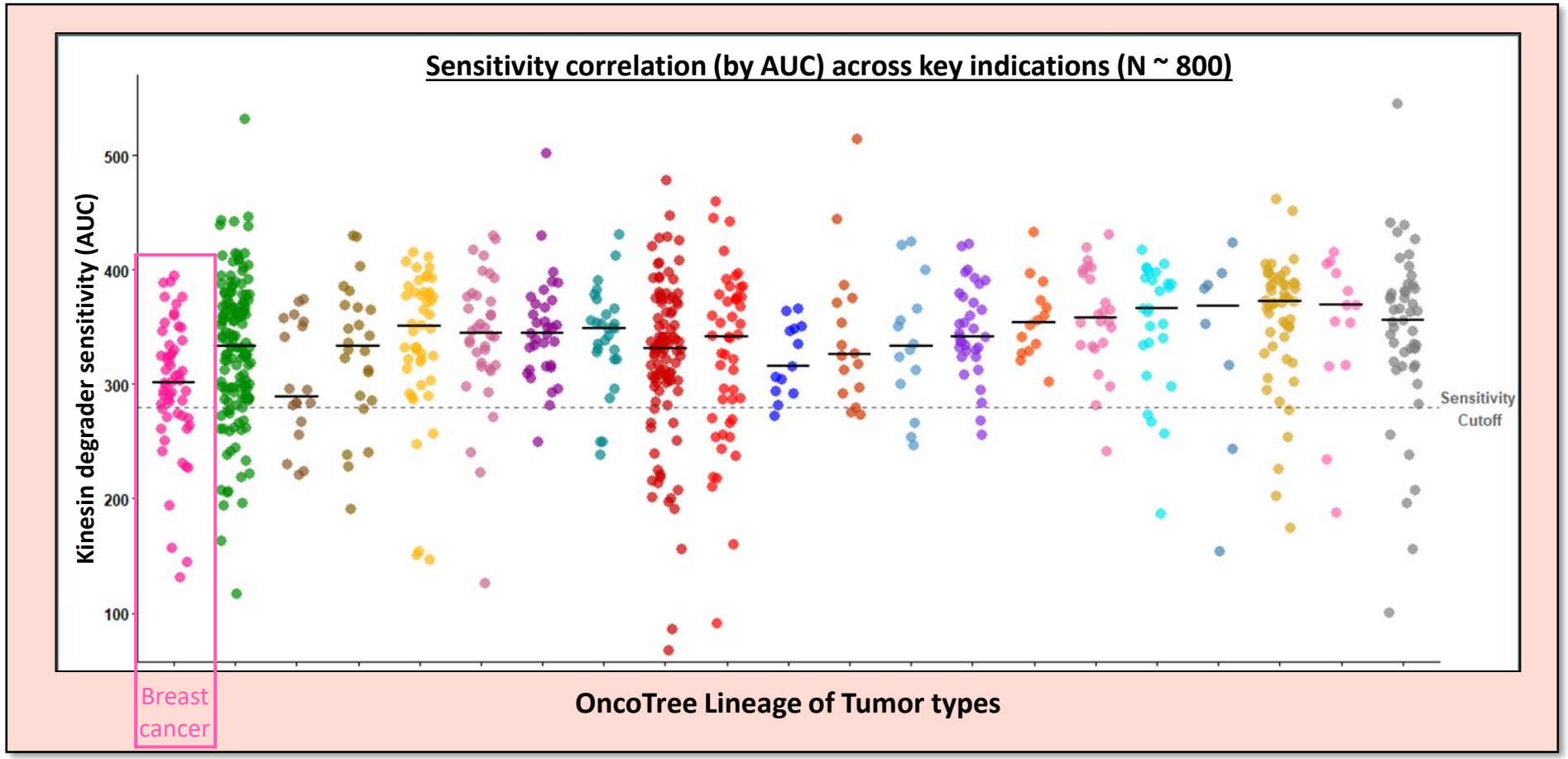
Rat model



Dog model



Cancer cell line screen identified multiple tumor types with high sensitivity to Kinesin degradation, including breast cancer

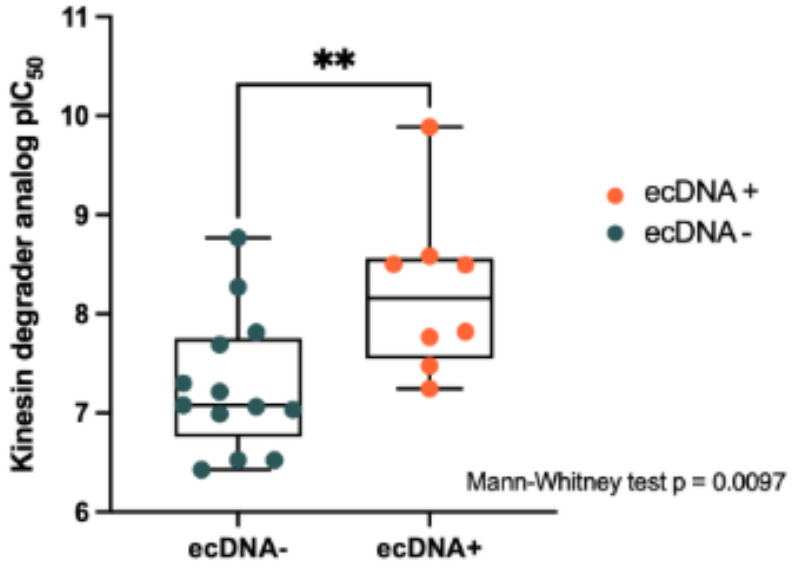


~15% of cell lines sensitive to Kinesin degradation

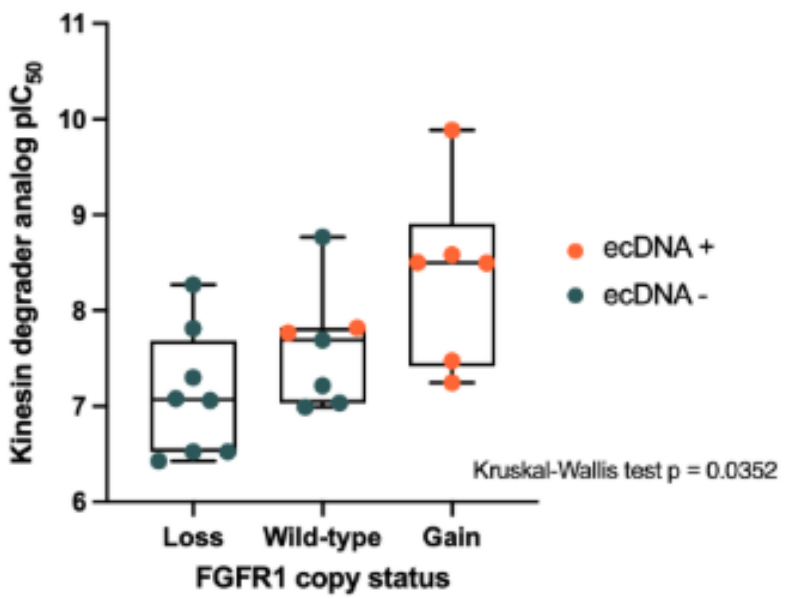
AUC: area under curve

In vitro sensitivity to Kinesin degradation in breast cancer cell lines is correlated with ecDNA status

Cancer cell line sensitivity to Kinesin degradation segmented by ecDNA status



Cancer cell line sensitivity to Kinesin degradation segmented by *FGFR1* and ecDNA status



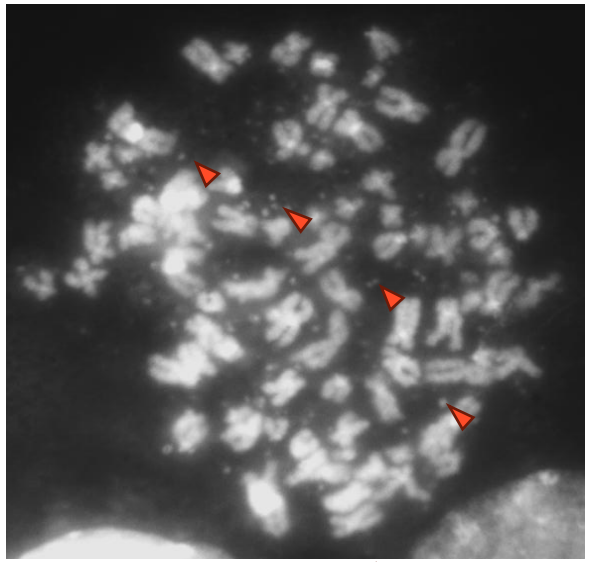
- **Breast cancer cell lines were profiled for sensitivity to Kinesin degraders**
- **ecDNA status was determined by DAPI staining of metaphase spreads**
- **ecDNA+ and *FGFR1* copy gain cell lines are correlated with higher sensitivity**

pIC50: proliferation log IC50 in multi-week 2D cell culture

DAPI: blue-fluorescent DNA stain

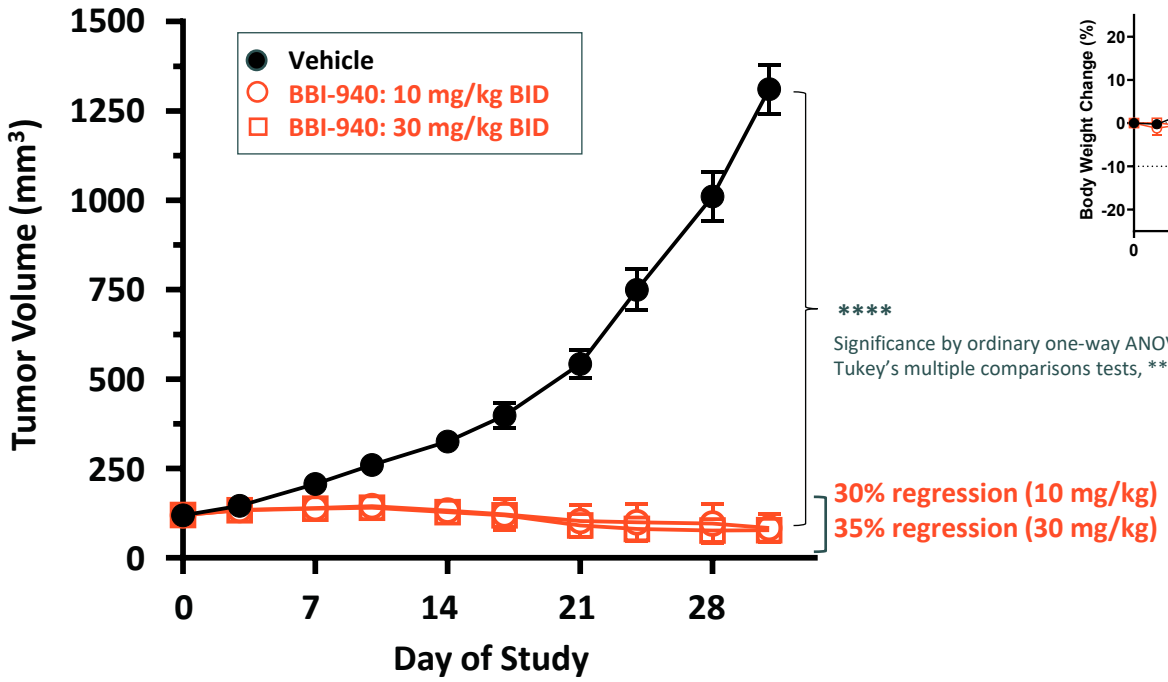
BBI-940 induced tumor regressions as a single agent in breast cancer CDX *in vivo* model

DNA staining of metaphase spread

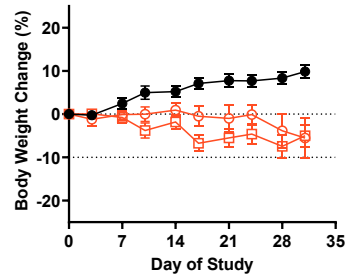


Referring to ecDNA

Tumor volume over time in TNBC-LAR CDX



Body weight over time

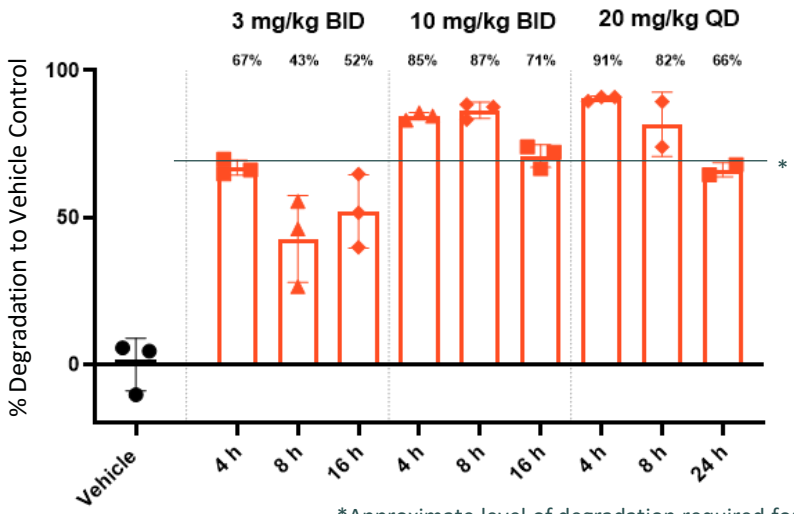


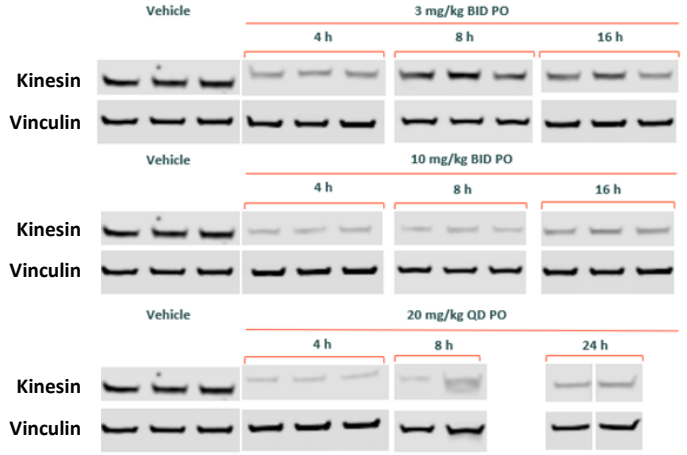
- **Monotherapy regression demonstrated with oral administration in a gene amplified ecDNA+ breast cancer model**
- **BBI-940 demonstrated sustained regressions at both doses tested**
- **BBI-940 generally well tolerated (body weight loss <10%)**

CDX: cell derived xenograft
TNBC-LAR: triple-negative breast cancer, luminal androgen receptor

Steady state PK/PD of BBI-940 tracked with anti-tumor activity in tumor bearing mice

Kinesin degradation at BBI-940 doses vs. time



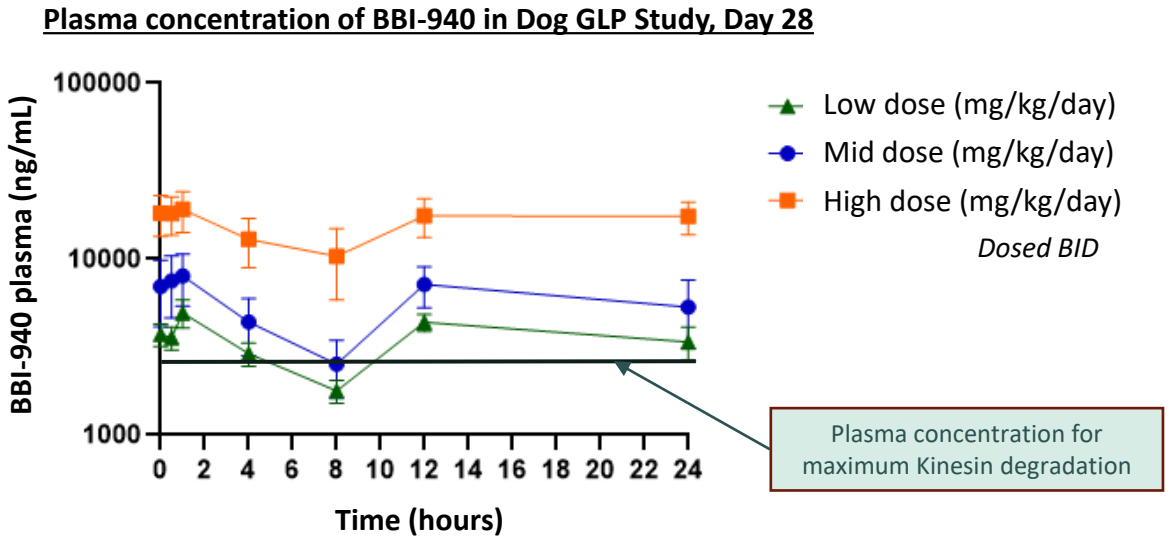


*Approximate level of degradation required for maximal TGI

- **Orally administered BBI-940 displayed dose-proportional exposure in plasma and tumor**
- **10 mg/kg BID results in >70% target degradation over the dosing interval**

BBI-940 was well tolerated in 28-day GLP-toxicity and toxicokinetic studies in rat and dog

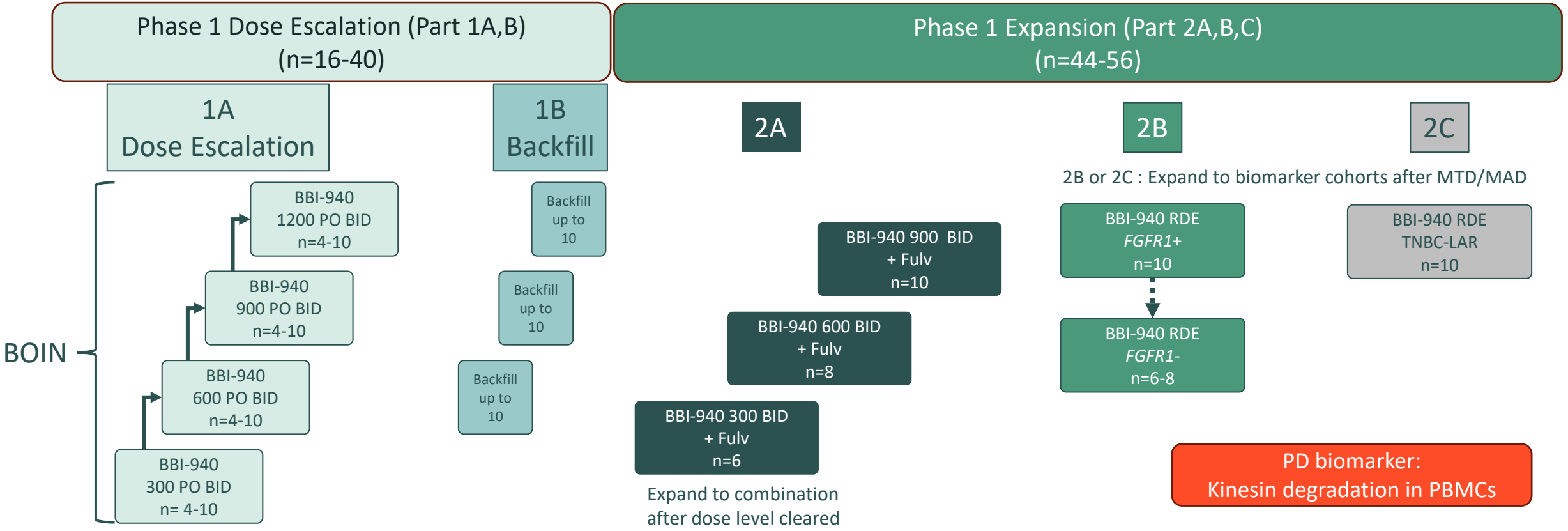
- No deaths
- No dose-related findings in:
  - Body weight or food consumption
  - Cage-side observations or clinical exams
  - Gross necropsy (no lesions)
  - Ophthalmic exams
- Safety pharmacology: no changes in ophthalmology or CNS (FOB)
- Histopathology: no concerning findings
- Toxicokinetics: covers or exceeds efficacious exposure in mouse CDX models



BW: Body weight; FOB: Functional Observation Battery (Central Nervous System); CBC: complete blood count; CVS: cardiovascular; FC: Food consumption; TK: toxicokinetic

First-in-human, phase 1 study of BBI-940 in metastatic breast cancer

Kinesin Oral Molecular Degrader for Oncology (KOMODO-1)



Part 1 Inclusion Criteria

- Advanced, metastatic ER+HER2- breast cancer OR
- TNBC-LAR

Part 2 Inclusion Criteria

- Advanced ER+/HER2- breast cancer without ESR1 mutation post CDK4/6 + ET (2A) or *FGFR1* amp+/-(2B) OR
- TNBC-LAR (2C)

NCT07408089

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DL: dose level
DE: dose escalation
Dose levels using modified Fibonacci

BOIN: Bayesian Optimal Interval Design
Fulv: fulvestrant
FGFR1+: *FGFR1* amp via tissue-based NGS

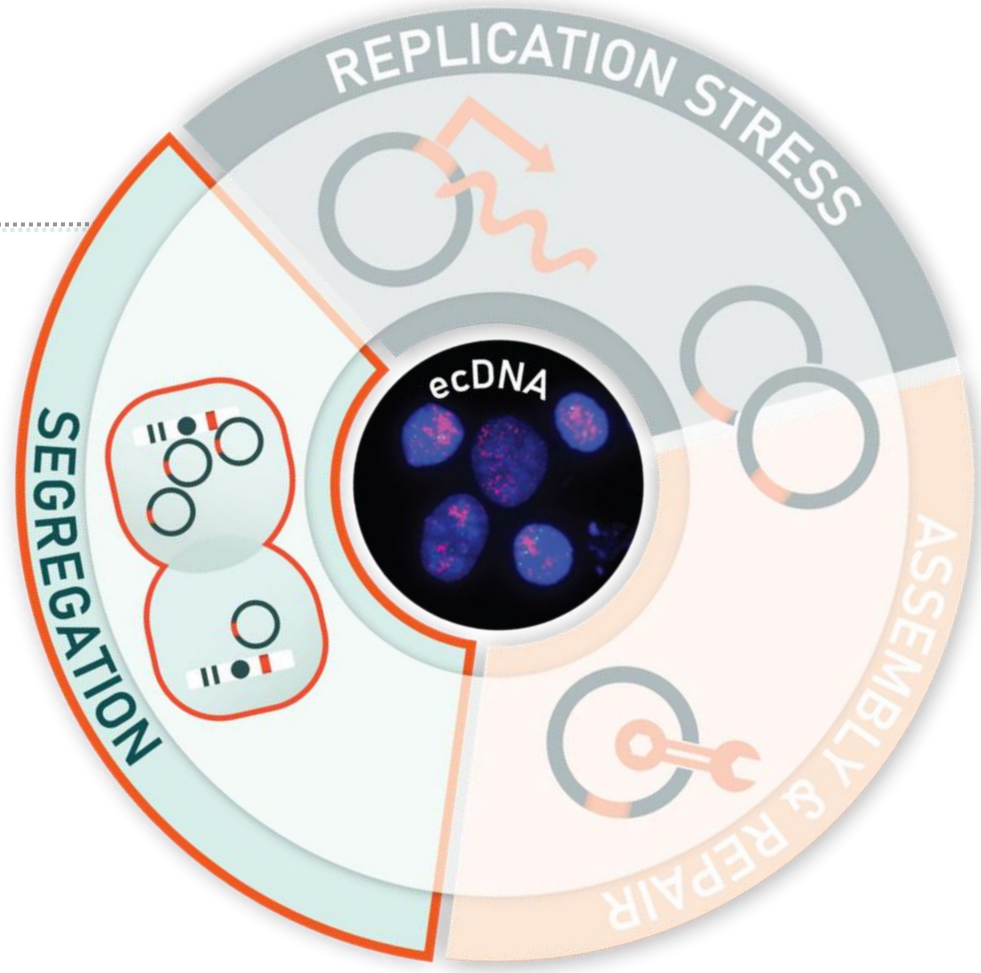
MTD: Maximum Tolerated Dose
MAD: Maximum Administered Dose
RDE: Recommended Dose for Expansion

BBI-940 summary: first-in-class Kinesin degrader advancing in the clinic

Novel Kinesin

BBI-940 (Phase 1 clinical)

- Potentially **first-in-class**, **oral**, **selective** Kinesin degrader
- Kinesin is a novel cancer target **essential for ecDNA segregation** but **non-essential** for normal chromosome segregation
- *In vitro* cytotoxicity and *in vivo* anti-tumor activity established in oncogene amplified cancer models, including **tumor regressions** in breast cancer models
- **FIH KOMODO-1 Phase 1 clinical trial underway**
- **No evidence of competitor programs** globally



Boundless Bio: leading a new area of cancer biology and targeting a large unmet need

Boundless has identified additional novel ecDNA targets for ecDTx discovery efforts

CHK1

BBI-355

Novel, oral, selective inhibitor of CHK1

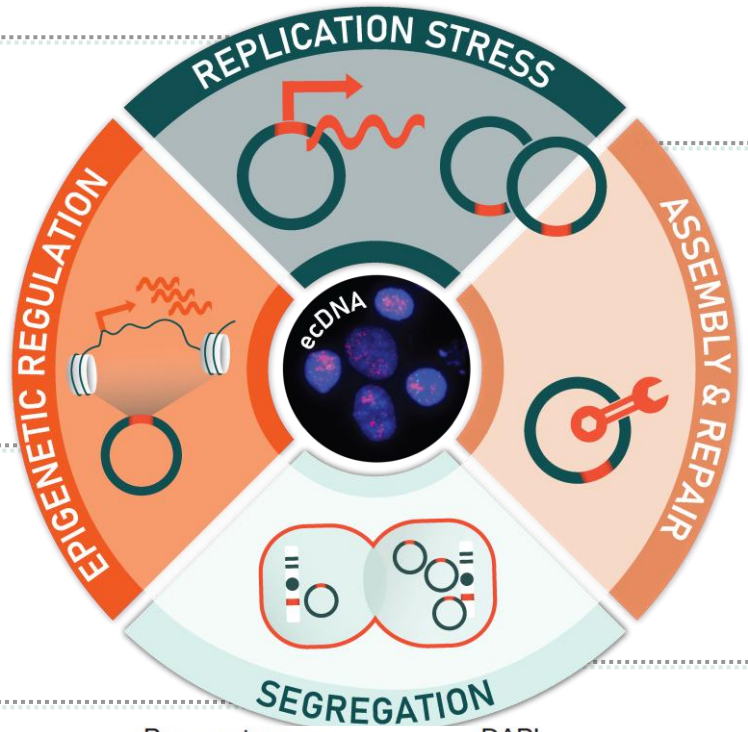
CHK1 is master regulator of replication stress, including that induced by ecDNA

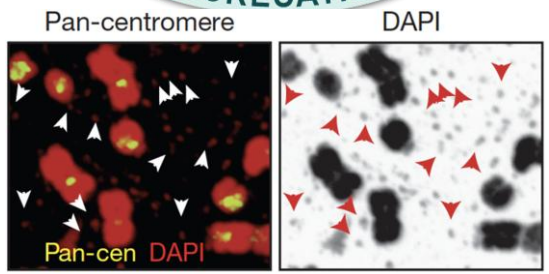
Target D: epigenetic regulator

Stage: Hit to Lead
Selective molecular glue degrader

New discovery targets:

Target A: ubiquitin enzyme
Stage: Hit Identification
Target B: Kinesin interactor
Stage: Assay Development
Target C: ATPase
Stage: Target Validation





Exploit non-centromeric ecDNA segregation pathways and chromosomal instability

RNR

BBI-825

Novel, oral, selective inhibitor of RNR

RNR is a rate-limiting enzyme for *de novo* synthesis of dNTPs, the raw materials of DNA, including ecDNA

Kinesin

BBI-940 (Phase 1 clinical)

Novel, oral, selective degrader of Kinesin

Kinesin is a target involved in segregation of DNA and critical for ecDNA segregation

Boundless Bio is leading a compelling and differentiated approach to address oncogene amplified cancers

Dedicated to Oncogene Amplified Cancers by Targeting Unique Cancer Biology

- **Oncogene amplifications**: one of cancer's highest unmet medical needs; expansive addressable market
- **ecDNA**: a root cause of amplification; Boundless Bio's unique lens into differentiated cancer biology
- **Spyglass**: ecDNA-focused platform to identify synthetic lethal targets in oncogene amplified / CIN cancer
- **ecDTx**: potentially first-in-class therapeutic programs (wholly-owned)

Fortress Position, Well-Funded, Track Record

- Founded by world's leading ecDNA experts
- Experienced team: track record of precision oncology drug approvals, multi-$B M&A
- Approximately $93M in cash and equivalents*, expected to provide cash runway into 2H28

Highly-Differentiated Value Drivers

ecDTx	Target	Intervention Node	Anticipated Milestones
BBI-940	Kinesin	DNA Segregation	KOMODO-1 clinical trial initiated in Q1:26 Initial clinical POC expected within existing cash runway

CIN: chromosomally instable

*Total pro forma cash, cash equivalents, and short-term investments as of 3/31/2026

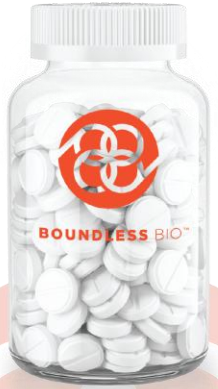
Boundless Bio: a breakthrough biotech company leading the next wave of innovation in cancer treatment

CANCER TREATMENT BREAKTHROUGHS

1940s CHEMOTHERAPY

1990-2000s TARGETED THERAPY

2010s IMMUNOTHERAPY



BIOTECH BREAKTHROUGH AWARD 2022
OVERALL BIOPHARMA COMPANY OF THE YEAR

2020s-2030s
ecDNA-DIRECTED THERAPIES (ecDTx)

Each prior wave of therapeutic innovation has been unable to address a critical population:

PATIENTS WITH ONCOGENE AMPLIFIED CANCERS

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Unbound by convention, bound to save lives

www.boundlessbio.com
@BoundlessBio

Bibliography of recent reviews and publications covering ecDNA—active hyperlinks

2025	Paul Mischel, Howard Chang	*Nature*: Genetic elements promote retention of extrachromosomal DNA in cancer cells
2025	Lillian Siu	NEJM: Extrachromosomal DNA – Amping Up Cancer
2024	Paul Mischel, Howard Chang	*Nature*: Enhancing transcription-replication conflict targets ecDNA-positive cancers
2024	Howard Chang, Paul Mischel, Charles Swanton	*Nature*: Origins and impact of extrachromosomal DNA
2024	Ben Cravatt, Paul Mischel, Howard Chang	*Nature*: Coordinated inheritance of extrachromosomal DNAs in cancer cells
2024	Vineet Bafna, Roel Verhaak	Nature Genetics: Mapping extrachromosomal DNA amplifications during cancer progression
2024	Paul Mischel, Howard Chang	*Nature Reviews Cancer*: Extrachromosomal DNA in cancer
2023	Paul Mischel, Vineet Bafna, Howard Chang, Roel Verhaak	*Nature*: Extrachromosomal DNA in the cancerous transformation of Barrett's oesophagus
2022	Vineet Bafna, Paul Mischel	*Annual Reviews*: Extrachromosomal DNA in Cancer
2022	Paul Mischel, Howard Chang	*Nature Structural and Molecular Biology*: Gene regulation on extrachromosomal DNA
2022	Rene Medema (Netherlands Cancer Inst.)	*Chromosoma*: Life of double minutes: generation, maintenance, and elimination
2022	Vineet Bafna, Howard Chang, Paul Mischel	*Annual Reviews*: Extrachromosomal DNA: An Emerging Hallmark in Human Cancer
2020	Anton Henssen, Howard Chang, Paul Mischel, Vineet Bafna	*Nature Genetics*: Extrachromosomal DNA is associated with oncogene amplification and poor outcome across multiple cancers
2020	Paul Mischel, Charles Swanton (Crick Inst.)	*Annals of Oncology*: Extrachromosomal DNA—relieving heredity constraints, accelerating tumour evolution
2020	Christopher Ott (Mass Gen)	*Cancer Cell*: Circles with a Point: New Insights into Oncogenic Extrachromosomal DNA
2019	Roel Verhaak, Vineet Bafna, Paul Mischel	*Nature Reviews*: Extrachromosomal oncogene amplification in tumour pathogenesis and evolution

PubMed