AILERON

Transforming the Experience of Chemotherapy for Cancer Patients

CORPORATE PRESENTATION

JANUARY 2022

Forward Looking Statements

Statements in this presentation about Aileron's future expectations, plans and prospects, as well as any other statements regarding matters that are not historical facts, may constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. These statements include, but are not limited to, statements about the Company's strategy and clinical development plans. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "project," "should," "target," "would" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including whether Aileron's cash resources will be sufficient to fund its continuing operations for the periods anticipated; whether the Company will obtain sufficient cash resources to conduct its planned clinical trials; whether results obtained in clinical trials will be indicative of results obtained in future clinical trials; whether third-party data would be indicative of the data that would be obtained in a randomized clinical trial; whether Aileron's product candidates will advance through the clinical trial process on a timely basis, or at all; whether the results of such trials will be accepted by and warrant submission for approval from the United States Food and Drug Administration or equivalent foreign regulatory agencies; whether Aileron's product candidates will receive approval from regulatory agencies on a timely basis or at all; whether, if product candidates obtain approval, they will be successfully distributed and marketed; what impact the coronavirus pandemic may have on the timing of our clinical development, clinical supply and our operations; and other factors discussed in the "Risk Factors" section of Aileron's quarterly report on Form 10-Q for the period ended September 30, 2021, filed on November 12, 2021, and risks described in other filings that Aileron may make with the Securities and Exchange Commission. Any forward-looking statements contained in this presentation speak only as of the date hereof, and Aileron specifically disclaims any obligation to update any forward-looking statement, whether because of new information, future events or otherwise.

We Aspire to Make Chemotherapy Safer and Thereby More Effective to Save More Lives

ALRN-6924: best-in-class potential among chemoprotective agents

- Demonstrated ‘triple-play protection’ against neutropenia, thrombocytopenia and anemia
- Potential to protect multiple tissue types against chemotherapeutic side effects

Core differentiator: p53 biomarker-enabled selective chemoprotection

Nearly 1 million patients in the U.S. are diagnosed annually with p53-mutated cancer

Planned 2022 clinical development milestones; 3 data readouts

Ongoing randomized, placebo-controlled Phase 1b trial in 1st-line p53-mutated NSCLC:

Interim results -- 2Q22; Topline results -- 4Q22

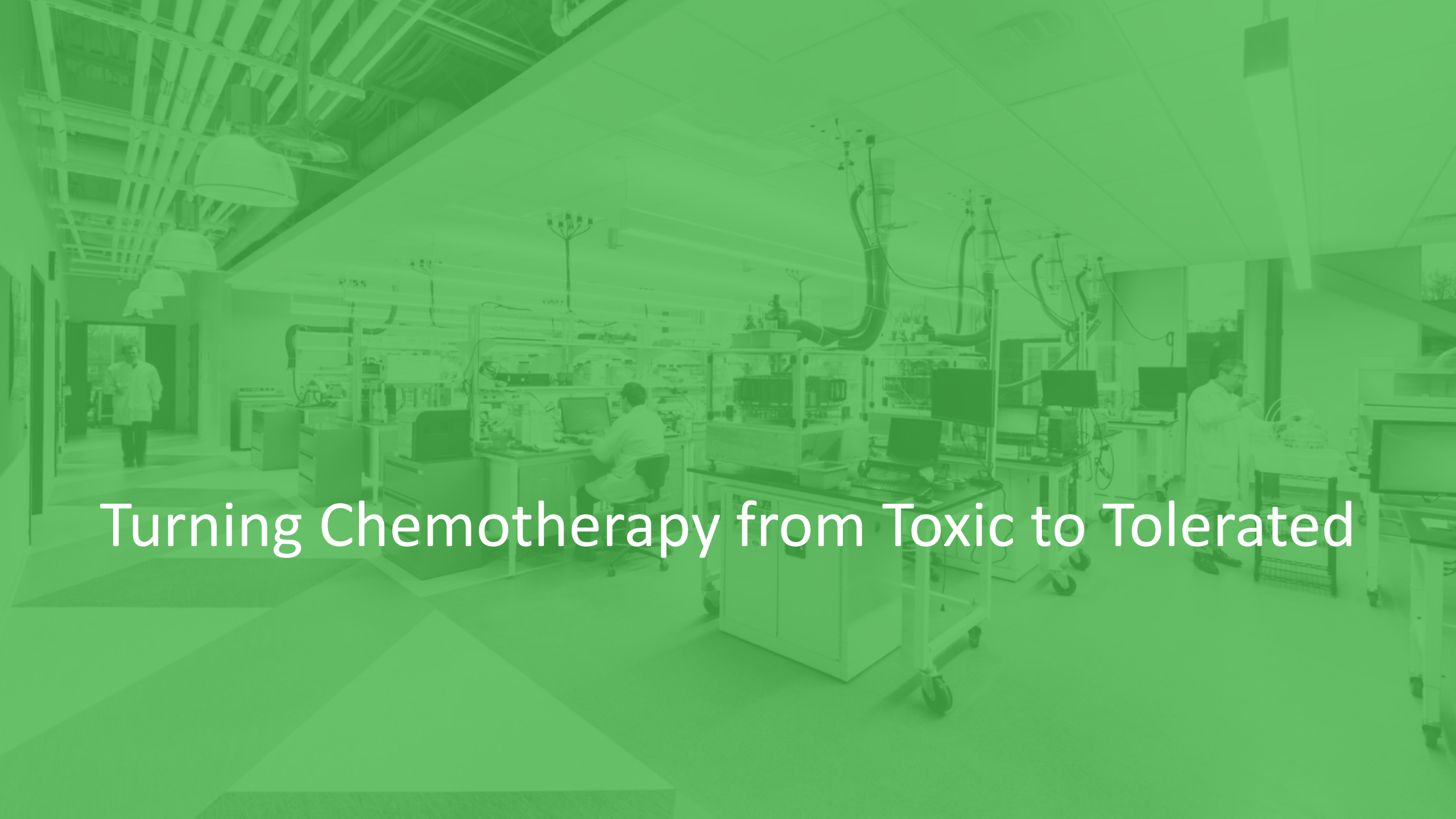
Upcoming parallel group design Phase 1b trial in neoadjuvant p53-mutated breast cancer:

Interim results -- 4Q22

Vision: pursue tumor-agnostic indication for patients with p53-mutated cancer

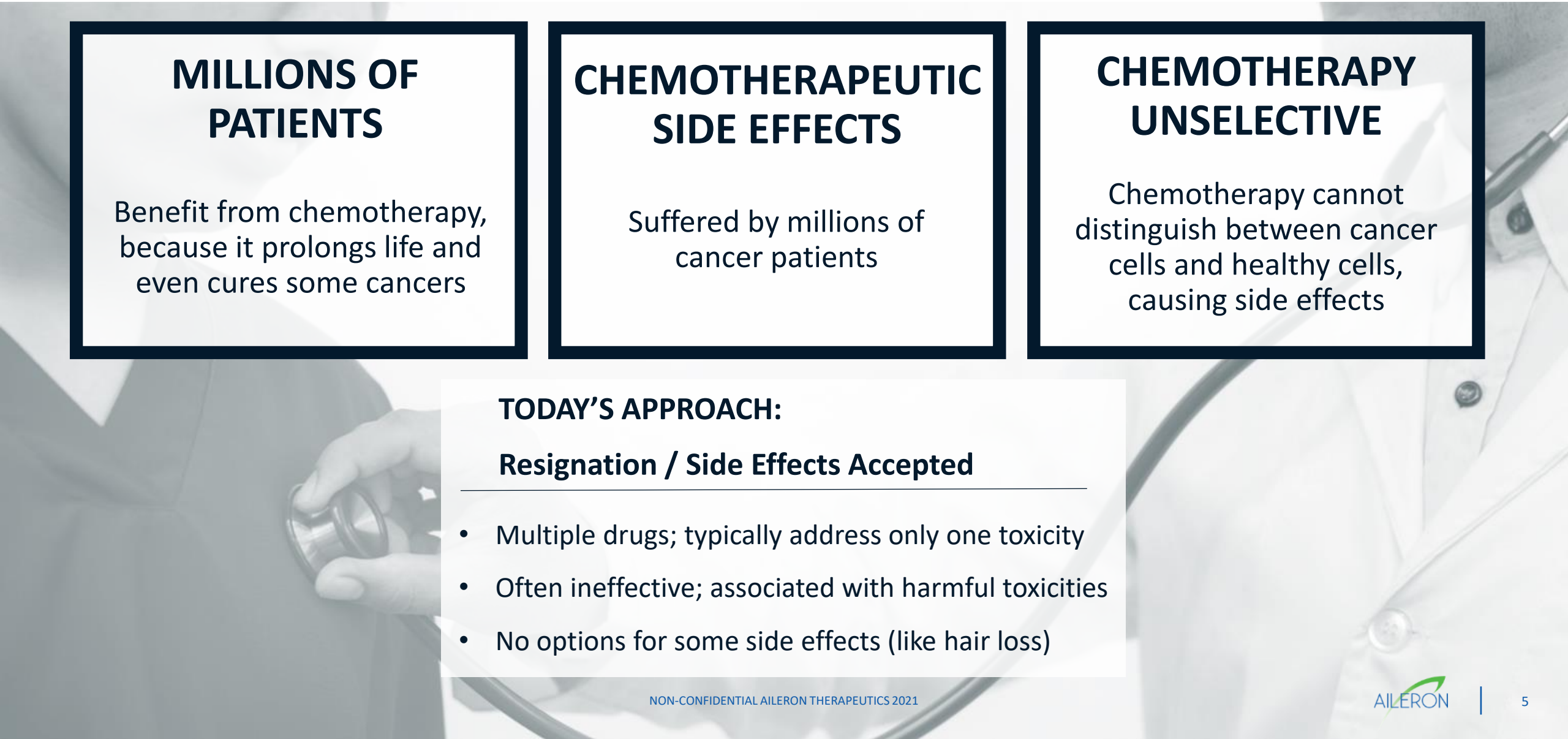
Selective chemoprotection regardless of type of cancer or chemotherapy

NSCLC = non-small cell lung cancer



Turning Chemotherapy from Toxic to Tolerated

Chemotherapy Remains the Essential Backbone of the Majority of Medical Cancer Treatment, but Its Side Effects Severely Limit Its Benefits to Patients



MILLIONS OF PATIENTS

Benefit from chemotherapy, because it prolongs life and even cures some cancers

CHEMOTHERAPEUTIC SIDE EFFECTS

Suffered by millions of cancer patients

CHEMOTHERAPY UNSELECTIVE

Chemotherapy cannot distinguish between cancer cells and healthy cells, causing side effects

TODAY'S APPROACH:

Resignation / Side Effects Accepted

- Multiple drugs; typically address only one toxicity
- Often ineffective; associated with harmful toxicities
- No options for some side effects (like hair loss)

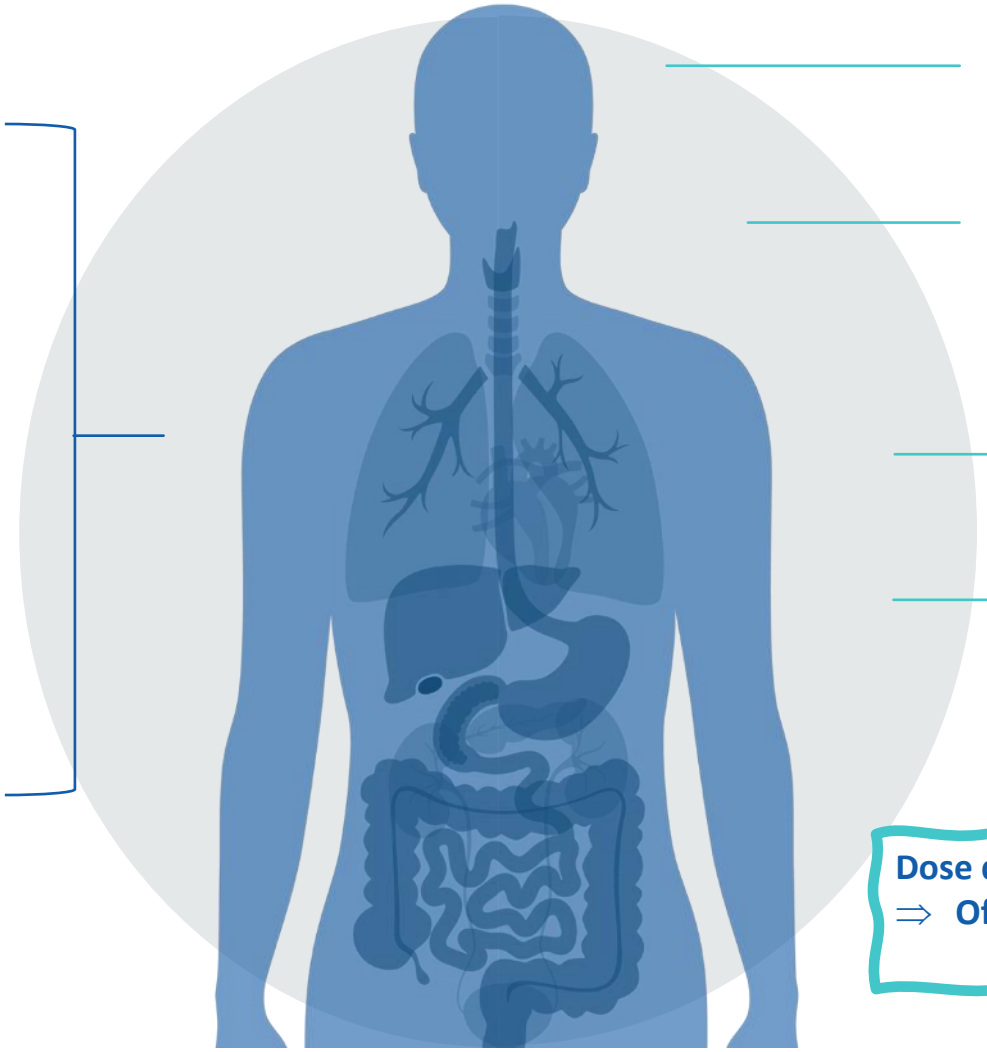
Chemotherapy's Lack of Selectivity Causes Side Effects – from Unpleasant to Life-Threatening

ALRN-6924 harnesses a universal mechanism common to normal cells to protect them against chemotherapy side effects

Severe Anemia (weakness, fatigue)

Severe Thrombocytopenia (excessive bleeding)

Severe Neutropenia (serious infection, fever, sepsis)



Alopecia (hair loss)

Stomatitis (mouth sores)

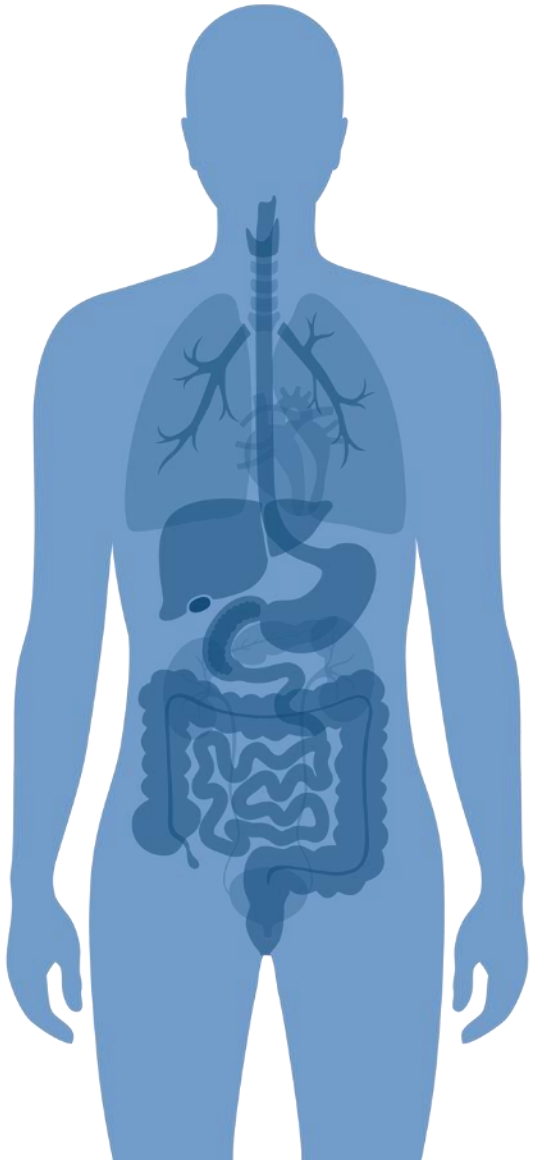
Fatigue, Weakness, Asthenia

Vomiting, Diarrhea, Bloating

Dose delays & dose reductions of chemotherapy
⇒ Often lead to incomplete chemotherapy
⇒ Often reduce response rates & survival

Poor Standard of Care for Management of Bone Marrow Toxicities

Opportunity for ALRN-6924 to be Best-in-Class Medicine to Protect Against Chemotherapy Side Effects



SEVERE ANEMIA

Standard of Care: "EPOs" (e.g., Aranesp®/Epogen®); Blood transfusions
Clinical Issues:

- EPOs: promote tumor growth and thrombo-embolic events (black box warning ⚠)
- Limited efficacy
- Indicated only for hemoglobin < 10g/dL
- Transfusions: risk of infection; limited supply

SEVERE NEUTROPENIA

Standard of Care: G-CSF (e.g. Neulasta® /Neupogen®)
Clinical Issues:

- Can promote tumor growth, can cause spleen rupture and bone pain
- Limited efficacy

SEVERE THROMBOCYTOPENIA

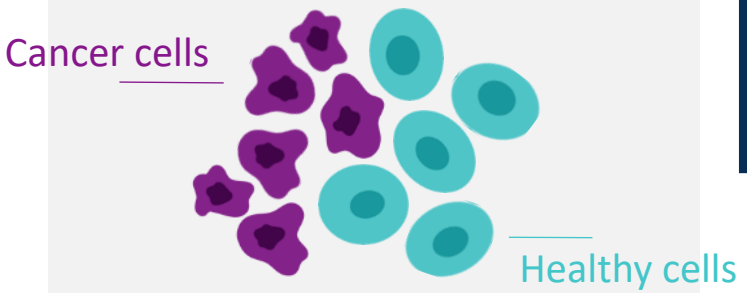
Standard of Care: No drugs used as standard of care – platelet transfusions instead
Clinical Issues:

- Effects limited to 2-5 days maximum
- Patients can become transfusion-refractory
- Transfusion-related risk of infection; limited supply

Basic Principles to Successfully Protect Against Chemotherapy-Induced Side Effects

CURRENT PARADIGM:

Chemotherapy targets healthy cells and cancer cells that are cycling (= undergoing cell division process)

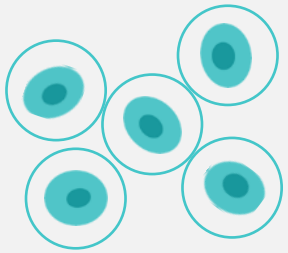


Normally, both healthy cells and cancer cells are destroyed by chemotherapy

PARADIGM SHIFT

AILERON PARADIGM:

Temporarily pause cycling in healthy cells, shielding them from chemotherapy



Healthy cells always have normal p53 thus can be protected

No interruption of cycling in p53-mutant cancer cells, thus not protecting cancer cells from chemotherapy

Cancer cells with mutant p53 are not protected

A COUNTERINTUITIVE APPROACH:

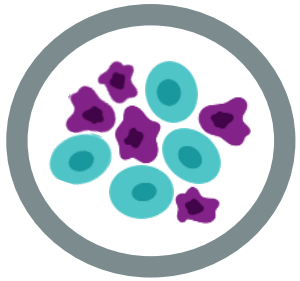
Treat healthy cells, not cancer cells

Healthy Cells Have Normal p53. ALRN-6924 Activates Normal p53, Thereby Upregulating p21, Which Pauses Cell Cycle in Normal Cells, but not in Cancer Cells with Mutant p53

Patient with p53-mutant cancer receives ALRN-6924 before chemotherapy

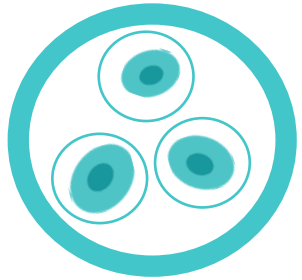
IV administration;
1-hour infusion

→



ALRN-6924 activates normal p53 in healthy cells

→

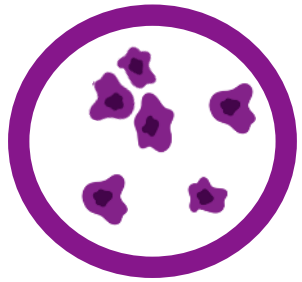


Activated normal p53 upregulates p21, which pauses cell cycling in healthy cells

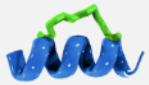
→

Patient with p53-mutant cancer receives chemotherapy

→



Chemotherapy's attack on cancer cells with mutant p53 is uninterrupted



ALRN-6924

Selective chemoprotection of healthy cells (always normal p53)

No protection of p53-mutant cancer cells

- p53 = most common cancer mutation†
- > 50% of all cancer patients have p53 mutation†

†Hoe, K., et al. Nat. Rev. Drug Discov.; 13: 217-236, 2014.

Potential Path to Tumor-Agnostic Indication, 3 Planned Readouts in 2022

p53-mutant SCLC
2nd line topotecan

Phase 1b (Completed)
Achieved proof of concept

Healthy Volunteer Study
MOA confirmed
Study ongoing

p53-mutant NSCLC
1st line carboplatin + pemetrexed +/- immune checkpoint inhibitor

Registration Program:

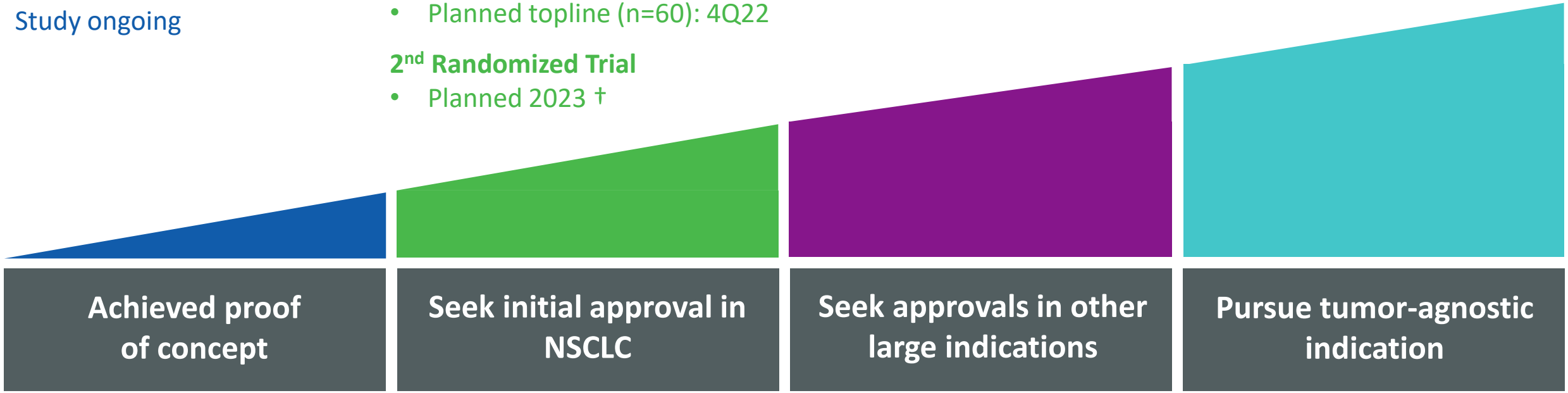
Ongoing Randomized, Placebo-Controlled Trial
- Planned interim (n=20): 2Q22
- Planned topline (n=60): 4Q22

2nd Randomized Trial
- Planned 2023 †

p53-mutant Breast Cancer
Neoadjuvant doxorubicin + cyclophosphamide and docetaxel

- Planned initiation: 1H22
- Planned interim: 4Q22
- Up to 30 patients

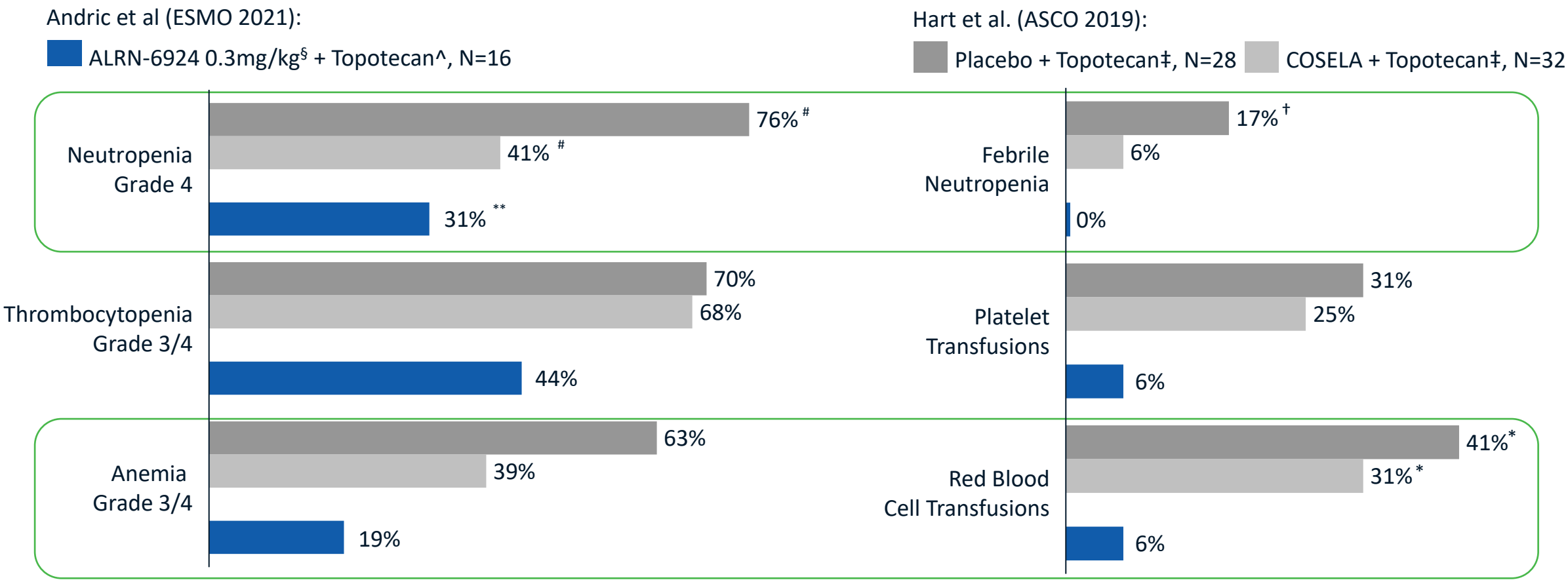
Additional p53-mutant Cancers †
Chemotherapies TBD



† Future trials are subject to clinical, regulatory, financial and other considerations
*SCLC=small cell lung cancer; MOA=mechanism of action

ALRN-6924 Proof-of-Concept Chemoprotection Data in SCLC Patients Receiving Topotecan^

Results of ALRN-6924 Phase 1b Trial and Results of COSELA Trial – Both in SCLC Patients Receiving Topotecan



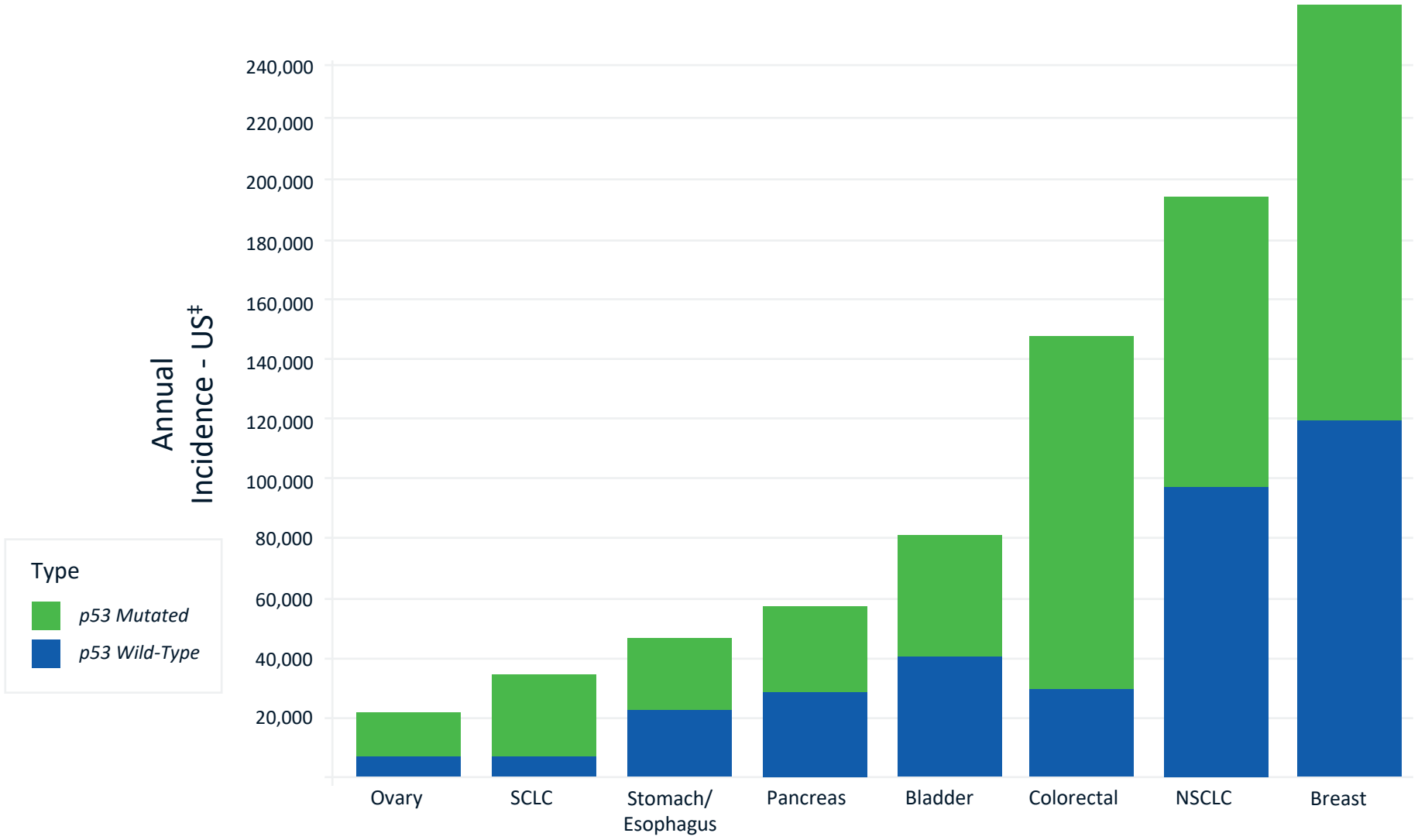
AEs based on laboratory values, as applicable

^ ALRN-6924 data cut July 29, 2021 (-24h cohort)
§ Identified as optimal dose
** In first treatment cycle
# Cycle not characterized

‡ Hart et al. ASCO 2019 (Slide 9) – G1 Therapeutics; clinical trial in SCLC patients receiving topotecan
* Excludes any RBC transfusions administered in the first 5 weeks
† Febrile neutropenia assessed for 29 patients

ALRN-6924 Opportunity and Clinical Development Strategy

Large Market Opportunities in p53-Mutated Cancers Across Most Cancer Types



Newly diagnosed cancer patients per year:

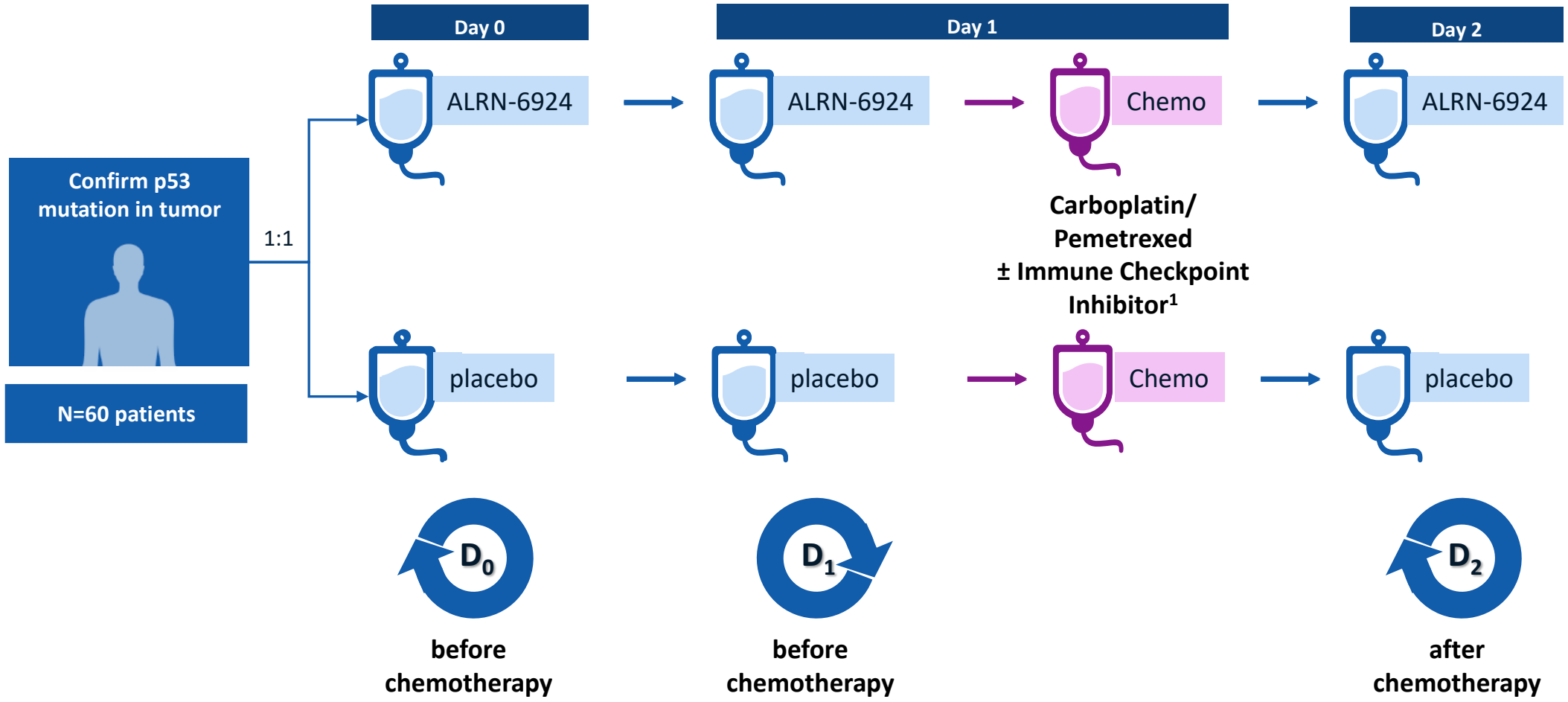
- 18 M Worldwide
- 1.8 M in the US
  - ~50%, i.e. nearly 1M diagnosed annually with p53-mutant cancer in the US

‡ Siegel et al, "Cancer statistics, 2020" American Cancer Society journal CA: A Cancer Journal for Clinicians. Genetic alterations from Foundation "Insights" Database 18-May-2020 (297,209 sample set).

* Hoe, K., et al. Nat. Rev. Drug Discov.; 13: 217-236, 2014.

Advanced p53-Mutated NSCLC Patients Treated in First Line with Carboplatin/Pemetrexed ± Immune Checkpoint Inhibitor[1] and ALRN-6924 or Placebo

Ongoing randomized, double-blind, placebo-controlled Phase 1b clinical trial; planned interim data 2Q22; planned topline results 4Q22



[1] Combination of ALRN-6924 with immune checkpoint inhibitors evaluated in Zhou X. et al, Cancer Discovery, July 6, 2021; **DOI:** 10.1158/2159-8290.CD-20-1741

Strong Intellectual Property Portfolio

- In 2021, Aileron was issued 7 new international patents, including new patent protection for ALRN-6924 in China, and 4 U.S. patents, adding to its strong intellectual property portfolio comprising over 170 U.S. and foreign patents.

- These patents and applications include ALRN-6924 methods of manufacture, methods of use, drug product formulations, and compositions of matter.

- Composition of matter patent in the US expires in 2033 with up to 5 additional years subject to patent term extensions.

Aileron maintains exclusive worldwide rights to its proprietary peptide drug technology and ALRN-6924.

Key Financial Highlights

Cash, Cash Equivalents and Investments (as of September 30, 2021)	$52.2M
Expected to Support Operations Into	2nd Half 2023
Common Shares Outstanding (September 30, 2021)	90.6M
Analyst Coverage by William Blair, H.C. Wainwright and Jones Trading	

We Aspire to Make Chemotherapy Safer and Thereby More Effective to Save More Lives

ALRN-6924: Selective chemo-protection without protecting cancer cells

Demonstrated protection against multiple heme toxicities & blood transfusions

Potential to protect multiple tissue types against chemotherapeutic toxicities

NSCLC trial ongoing; Breast cancer trial to start 1H22

OUR VISION

Chemoprotection for patients with p53-mutated cancers across all cancer types and chemotherapies

~1 million

patients diagnosed annually in U.S. with p53-mutated cancer

Chemoprotection May Transform Chemotherapy Like Anesthesia Transformed Surgery

NASDAQ: ALRN

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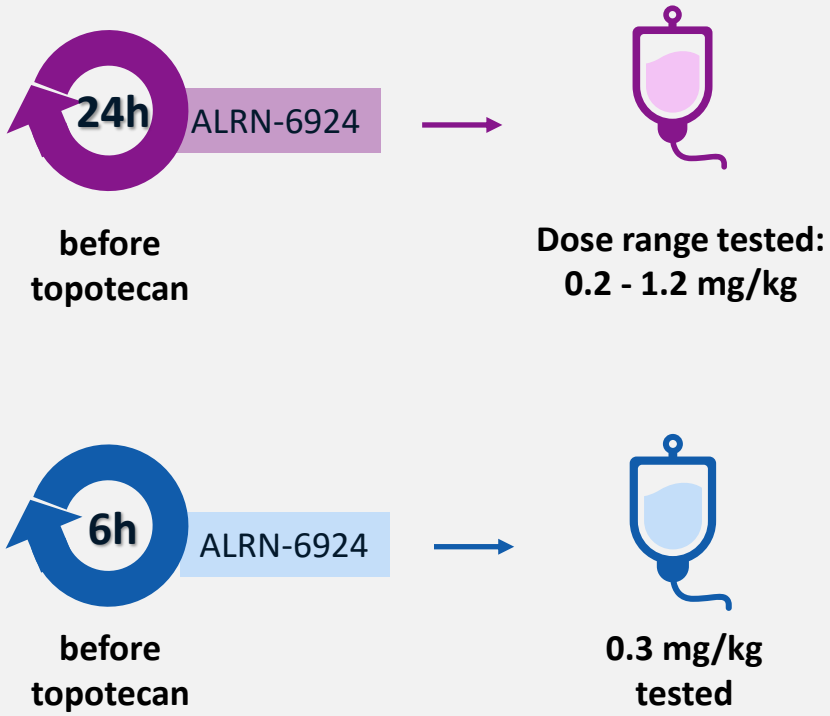
Supplementary Information

ALRN-6924 Phase 1b Trial in SCLC
Design & Final Results

European Society of Medical Oncology (ESMO) Virtual Congress
September 2021

ALRN-6924 Proof-of-Concept Phase 1b SCLC Trial Schema



Trial Completed

0.3 mg/kg at 24h before chemotherapy selected for future trials

Final Results Data Presentation ESMO – Sept. 2021

Protocol highlights: Topotecan (1.5 mg/m^2) administered on days 1 through 5 of every 21-day treatment cycle. Prophylactic G-CSF treatment not permitted in cycle 1; Hb >9 g/dL, ANC >1500/µL, Platelets >100k/µL at baseline

Phase 1b SCLC Study: Demographics and Key Baseline Disease Characteristics

		24H SCHEDULE				6H SCHEDULE	TOTAL
		0.2 N=4	0.3 N=16	0.6 N=6	1.2 N=6	0.3 N=7	N=39
AGE, MEDIAN		65	68.5	66.5	58	69	67
GENDER N (%)	MALE	2 (50)	16 (100)	3 (50)	4 (67)	4 (57)	29 (74)
	FEMALE	2 (50)	0	3 (50)	2 (33)	3 (43)	10 (26)
BASELINE LDH N (%)	<ULN	1 (25)	9 (56)	2 (33)	4 (67)	1 (14)	17 (44)
	≥ULN	3 (75)	7 (44)	4 (67)	2 (33)	6 (86)	22 (56)
TIME SINCE COMPLETION OF PREVIOUS THERAPY N (%)	<60 DAYS	1 (25)	8 (50)	1 (17)	5 (83)	3 (43)	18 (46)
	≥60 DAYS	3 (75)	7 (44)	5 (83)	1 (17)	4 (57)	20 (51)
	MISSING	0	1 (6)	0	0	0	1 (3)
BASELINE ECOG STATUS N (%)	0	3 (75)	12 (75)	3 (50)	3 (50)	2 (29)	23 (59)
	1	1 (25)	4 (25)	3 (50)	3 (50)	4 (57)	15 (39)
	2	0	0	0	0	1 (14)	1 (2)
	≥3	0	0	0	0	0	0
STAGE AT SCLC DIAGNOSIS N (%)	LIMITED	0	0	0	0	0	0
	EXTENSIVE	4 (100)	16 (100)	6 (100)	6 (100)	7 (100)	39 (100)
p53 MUTATION STATUS N (%)	MUTATED	4 (100)	16 (100)	6 (100)	6 (100)	7 (100)	39 (100)
	WILD TYPE	0	0	0	0	0	0

Chemoprotection Led to Fewer Dose Reductions of Chemotherapy

Topotecan dose reductions in SCLC patients:

- Without Chemoprotection: 29%[^] to 32%[*] of patients
- With Chemoprotection:
  - with COSELA[#]: 19% of patients
  - with ALRN-6924[§]: 19% of patients

Hart et al., Adv Ther 2020; topotecan + *COSELA*-treated patients (G1-Therapeutics' clinical trial in SCLC patients receiving topotecan)

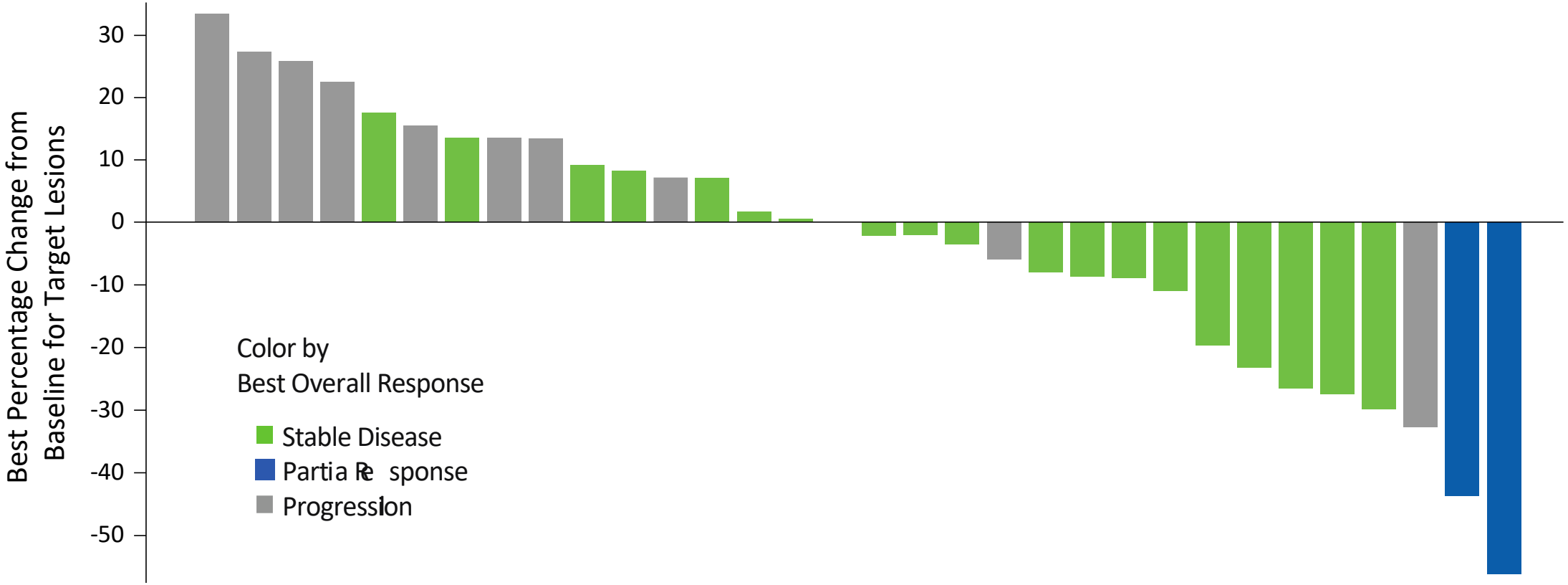
* Hart et al., Adv Ther 2020; topotecan + *placebo*-treated patients (G1-Therapeutics' clinical trial in SCLC patients receiving topotecan)

^ U.S. Prescribing Information for topotecan (2019)

§ Andric et al, ESMO 2021; patients receiving topotecan + ALRN-6924 (0.3 mg/kg -24 h cohort, N=16)

Vision: Chemoprotection → fewer dose reductions → better outcomes

Preservation of Chemotherapy's Anti-Cancer Effects During Treatment with ALRN-6924



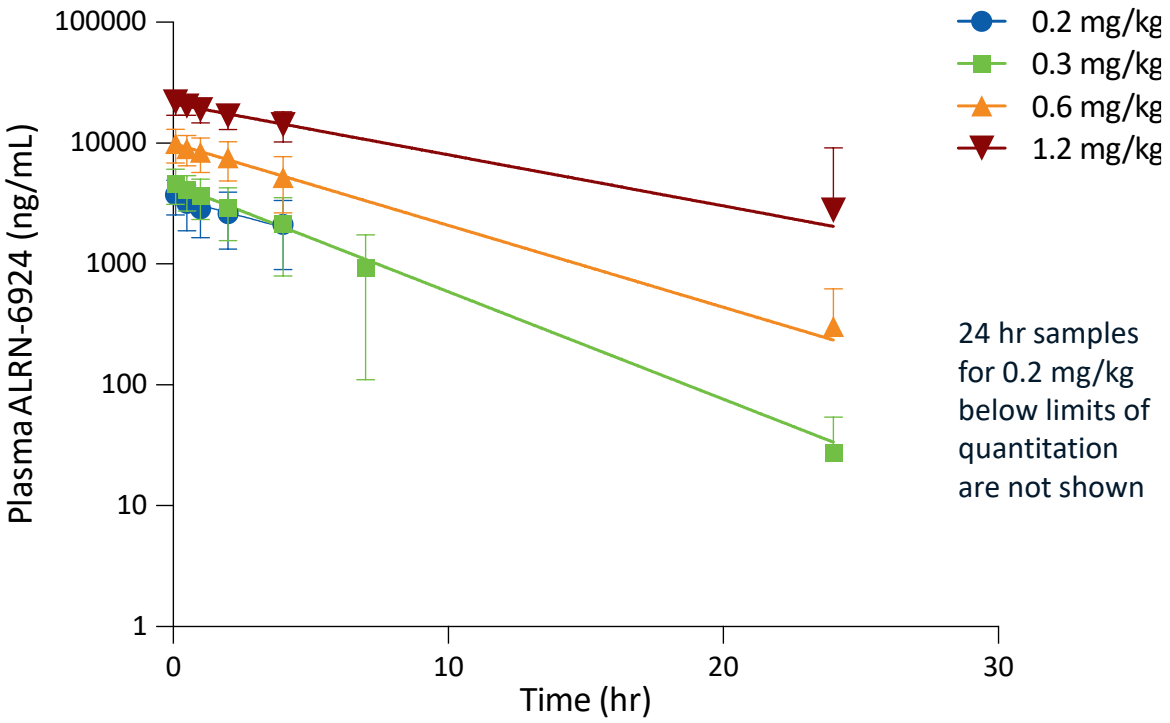
- **The disease control rate (DCR) was 65.6% for 32 patients with radiological evaluation***
- **In historical trials of SCLC patients receiving topotecan, the DCR was between 47% and 61.5%‡**

‡ O'Brien M., et al. J. Clinical Oncol. 24(34): 5441-5447, 2006.
Eckhardt J.R., et al. J. Clin. Oncol. 25(15): 2086-2092, 2007.
Inoue A., et al. J. Clin. Oncol. 26(33): 5401-5406, 2008
Jotte R., et al. J. Clin. Oncol. 29(3): 287-293, 2011.
Pawel J., et al. J. Clinical Oncol. 32(35): 4012-4019, 2014.

- As measured by CT-based tumor imaging per RECIST 1.1; 7 patients did not have a post-baseline evaluation

ALRN-6924 Shows Dose-Dependent PK Profile in SCLC Patients

ALRN-6924 Plasma PK for Patients Dosed 0.2, 0.3, 0.6, and 1.2 mg/kg



24 hr samples for 0.2 mg/kg below limits of quantitation are not shown

- Mean ± st. dev. following first dose of ALRN-6924

	0.2 mg/kg	0.3 mg/kg	0.6 mg/kg	1.2 mg/kg
n of patients	4	22†	6	6
$AUC_{0\text{-}24\ hr}$ µg·hr/mL (% CV)	31.8 (53.6)	34.4 (57.0)	83.0 (45.4)	211.9 (26.3)
C_{max} µg/mL (% CV)	3.8 (32.2)	4.9 (31.3)	10.0 (30.9)	22.0 (21.6)
$t_{½}$, hr (95% C.I.)	4.9 (2.0 - n.c.)	3.4 (2.5 - 4.7)	4.4 (2.6 - 9.2)	7.1 (3.9 - 12.4)

- Slower clearance (longer $t_{½}$) at higher ALRN-6924 doses. 3.4 to 7.1 hr half-life yields no accumulation on repeated dosing
- Plasma exposure after a single ALRN-6924 dose: Dose-proportional C_{max}, slightly greater than dose-proportional AUC

† PK samples unavailable for one 0.3 mg/kg patient

Supplementary Information

Phase 1 Pharmacology Study of ALRN-6924 in Healthy Volunteers (Ongoing)

Design & Initial Results

European Society of Medical Oncology (ESMO) Virtual Congress
September 2021

Phase 1 Study of ALRN-6924 in Healthy Human Volunteers: Study Schema

PART 1

Determination of Optimal Dose (Completed)

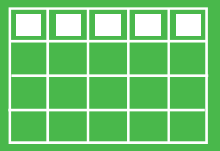
3 groups of subjects were treated with a single dose of placebo (n=6), ALRN-6924 0.3 mg/kg (n=4), or ALRN-6924 0.6 mg/kg (n=4)

PART 2

Kinetics of Pharmacodynamic Effects in the Bone Marrow (Completed)

23 subjects were treated with a single dose of 0.3 mg/kg ALRN-6924, with bone marrow sampled from **3** subjects at each successive time points 4-48 hrs post-infusion

Ongoing



Confirmation of a Universal Treatment Schedule for ALRN-6924; Testing Effects of ALRN-6924 on Epithelial Cells

ALRN-6924 Phase 1 Healthy Volunteer Study: Key Takeaways

Study findings (Parts 1 and 2 completed to date) consistent with prior PK-PD non-clinical results

Optimal dose (0.3 mg/kg) and MOA (p53-activated p21 upregulation, p21-mediated cell cycle arrest) confirmed

Time to onset, duration and magnitude of PD effects to inform dosing schedules for future clinical trials of ALRN-6924

Adverse Events: All Events Were Grade 1 Only; No SAEs or AEs Led to Discontinuation of Study Participation

	Part 1			Part 2	
Parameter	Placebo (N=6)	ALRN-6924 0.3 mg/kg (N=4)	ALRN-6924 0.6 mg/kg (N=4)	ALRN-6924 0.3 mg/kg (N=23)	Total ALRN-6924 0.3 mg/kg (N=27)
TEAEs Occurring in >10% of Subjects					
ANY TEAE	3 (50)	2 (50)	4 (100)	21 (91)	23 (85)
NAUSEA	0	1 (25)	3 (75)	5 (22)	6 (22)
VOMITING	0	1 (25)	1 (25)	2 (9)	3 (11)
BIOPSY SITE PAIN	3 (50)	0	0	11 (48)	11 (41)
FATIGUE	0	0	1 (25)	1 (4)	1 (4)
HEADACHE	0	0	2 (50)	9 (39)	9 (33)
DIZZINESS	0	1 (25)	1 (25)	3 (13)	4 (15)
SAEs Occurring in >10% of Subjects					
ANY SAE	0	0	0	0	0

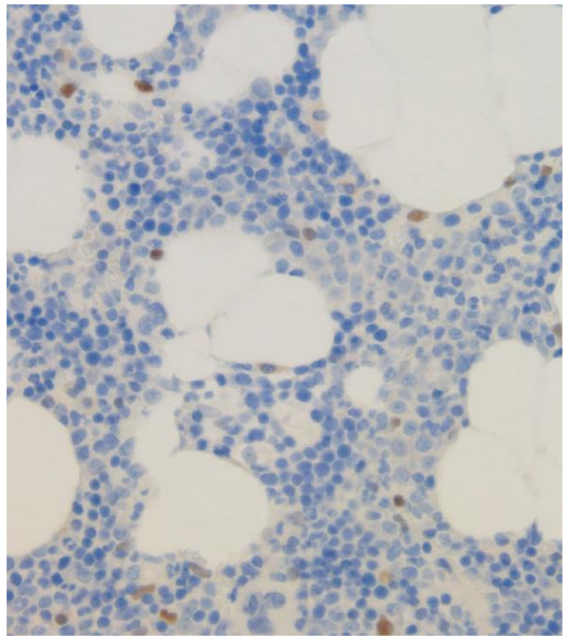
*Following submission of poster to ESMO Virtual Congress 2021, a “totals” column mistakenly combining placebo- and ALRN-6924-treated subjects was removed from this table.

Both 0.3 and 0.6 mg/kg ALRN-6924 Doses Induced p21 in Bone Marrow Cells†

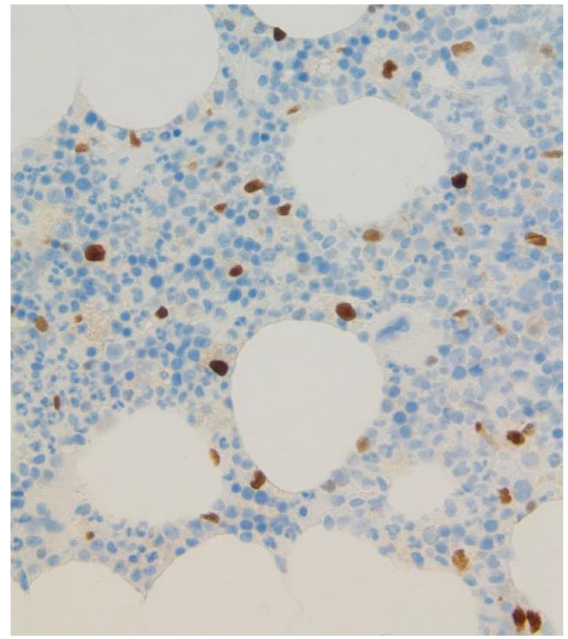
Subject	Group	p21, % at Differential Intensities† 0	1+	2+	3+	cPARP‡ # cells per HPF
1	Plac.	100	0	0	0	2
2	"	100	0	0	0	1
3	"	100	0	0	0	19
4	"	100	0	0	0	12
5	"	100	0	0	0	19
6	"	100	0	0	0	21
7	0.3 mg/kg	94	0	2	4	19
8	"	96	0	2	2	21
9	"	96	0	1	3	7
10	"	95	0	1	4	85
11	0.6 mg/kg	94	0	1	5	17
12	"	93	0	2	5	61
13	"	97	0	3	0	70
14	"	95	3	2	0	96

Representative p21 Staining

Placebo-treated Subject



ALRN-6924-treated Subject



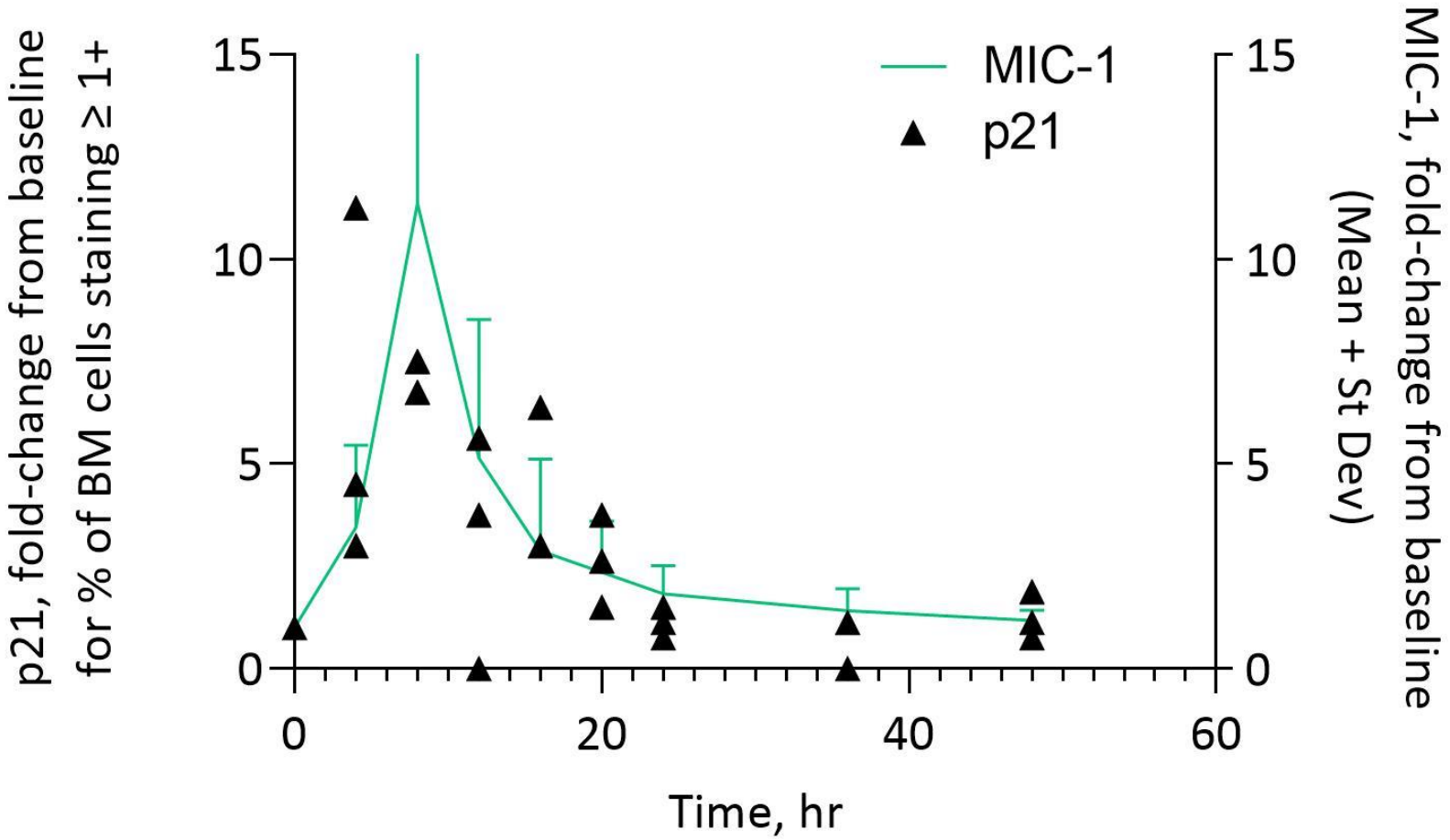
†Note: bone marrow is largely composed of quiescent stem cells or nonproliferating immature, differentiating cells; only 5% to 20% of cells are actively cycling[1,2]

‡Due to low % of cells with positive staining, results are shown as actual number of cells per high-power field (HPF). Scoring in bone marrow core biopsies sampled 8 hrs post-dose was conducted by a pathologist blinded to experimental conditions.

1.Zhang S., et al. Study of different phases in growth cycle of human bone marrow cells and their growth speed, using rat-human cell hybridization, premature chromosome condensation, and sister chromatid differentiation techniques. *Exp Hematol*. 16(3):221-5, 1988.

2.Hayman et al. Distribution Of Proliferating Bone Marrow in Adult Cancer Patients Determined Using FLT-PET Imaging. *Int. J. Radiation Oncology Biol. Phys*. 79(3):847–852, 2011.

p21 Protein in Bone Marrow and Serum MIC-1, Both p53-Dependent, Were Transiently Elevated up to 20 hrs Following a Single 0.3 mg/kg Dose of ALRN-6924



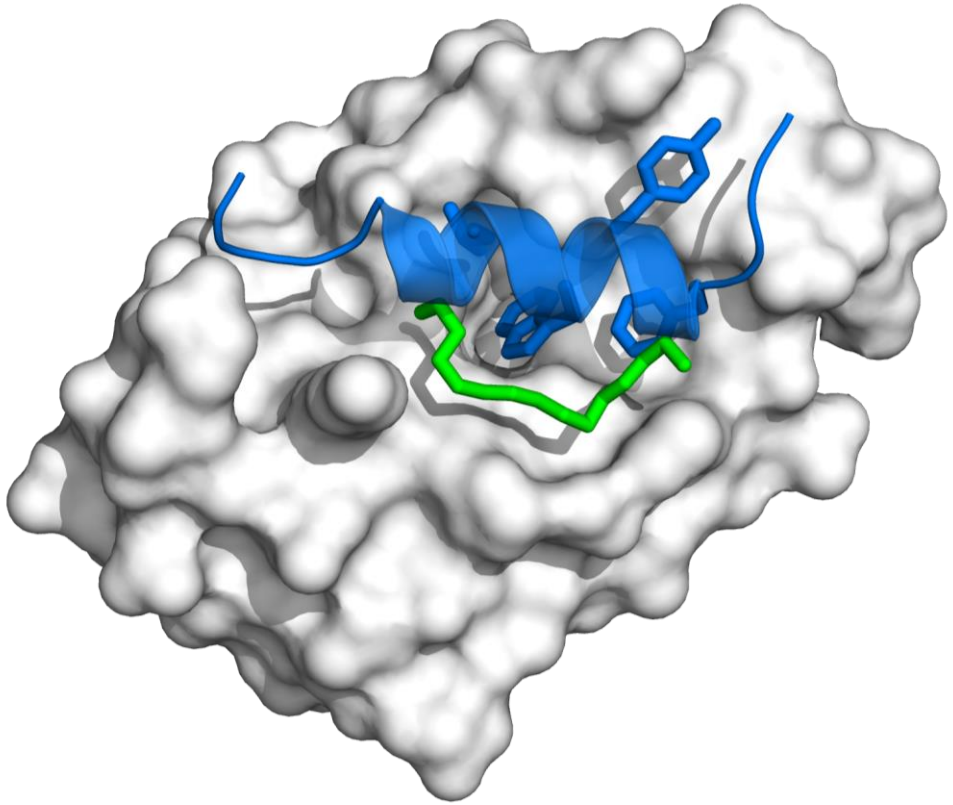
- p21 immunohistochemistry scoring was conducted by an independent pathologist blinded to experimental conditions
- Macrophage Inhibitory Cytokine-1 (MIC-1, also known as Growth Differentiation Factor-15, or GDF-15) was quantified by a qualified ELISA assay
- MIC-1 results are shown as mean change from baseline value prior to first ALRN-6924 dose

MIC-1 = macrophage inhibitory cytokine-1

Supplementary
Non-Clinical Information About ALRN-6924

Structure and Key Design Properties of ALRN-6924

Structure based on the α-helical domain of p53, with chemical modifications



ALRN-6924·MDMX co-crystal structure

The hydrocarbon staple and other modifications ensure:

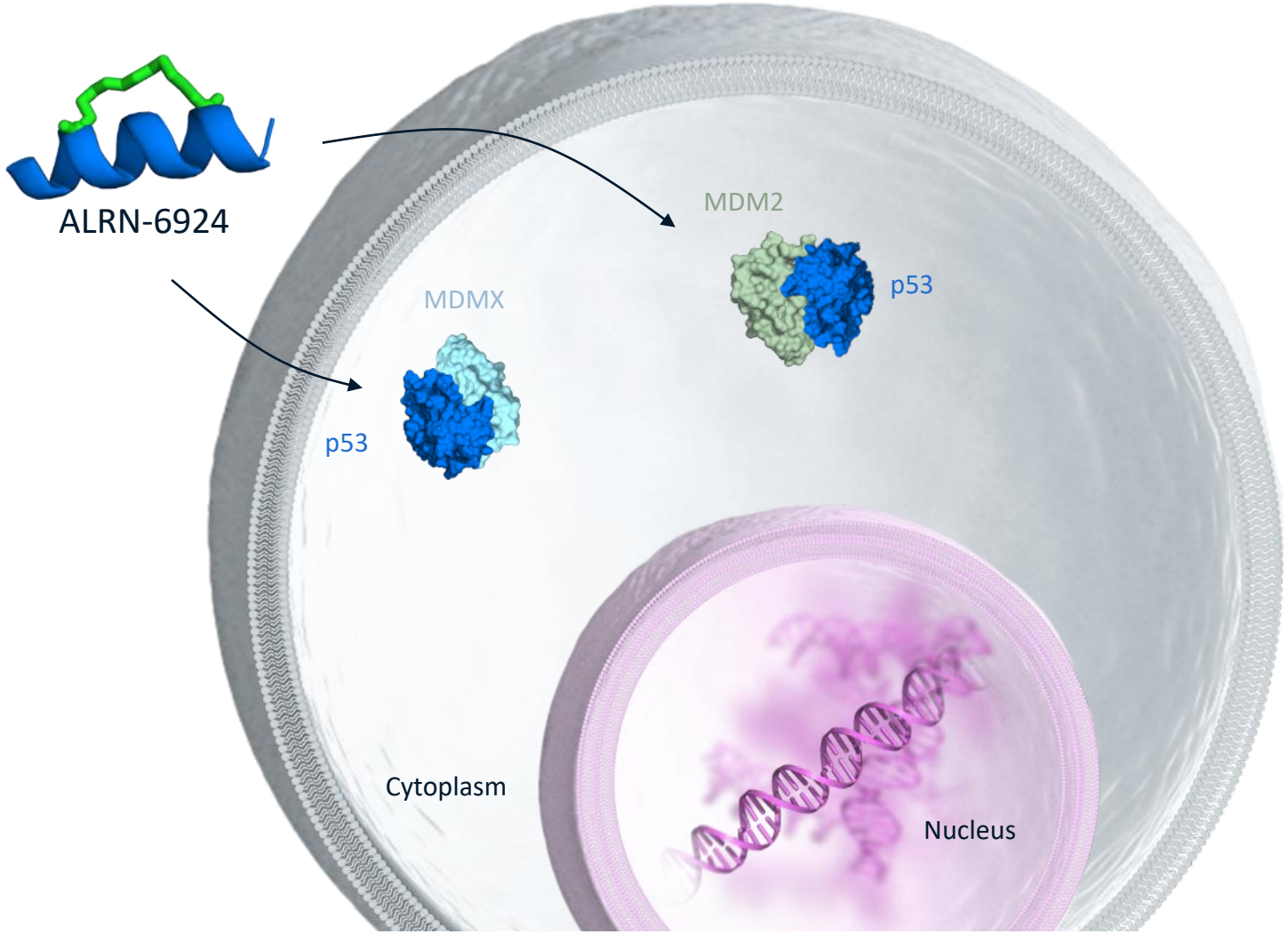
I. Protection from proteolytic cleavage

II. Permeation of cell membranes and cell entry

III. High affinity binding to its targets

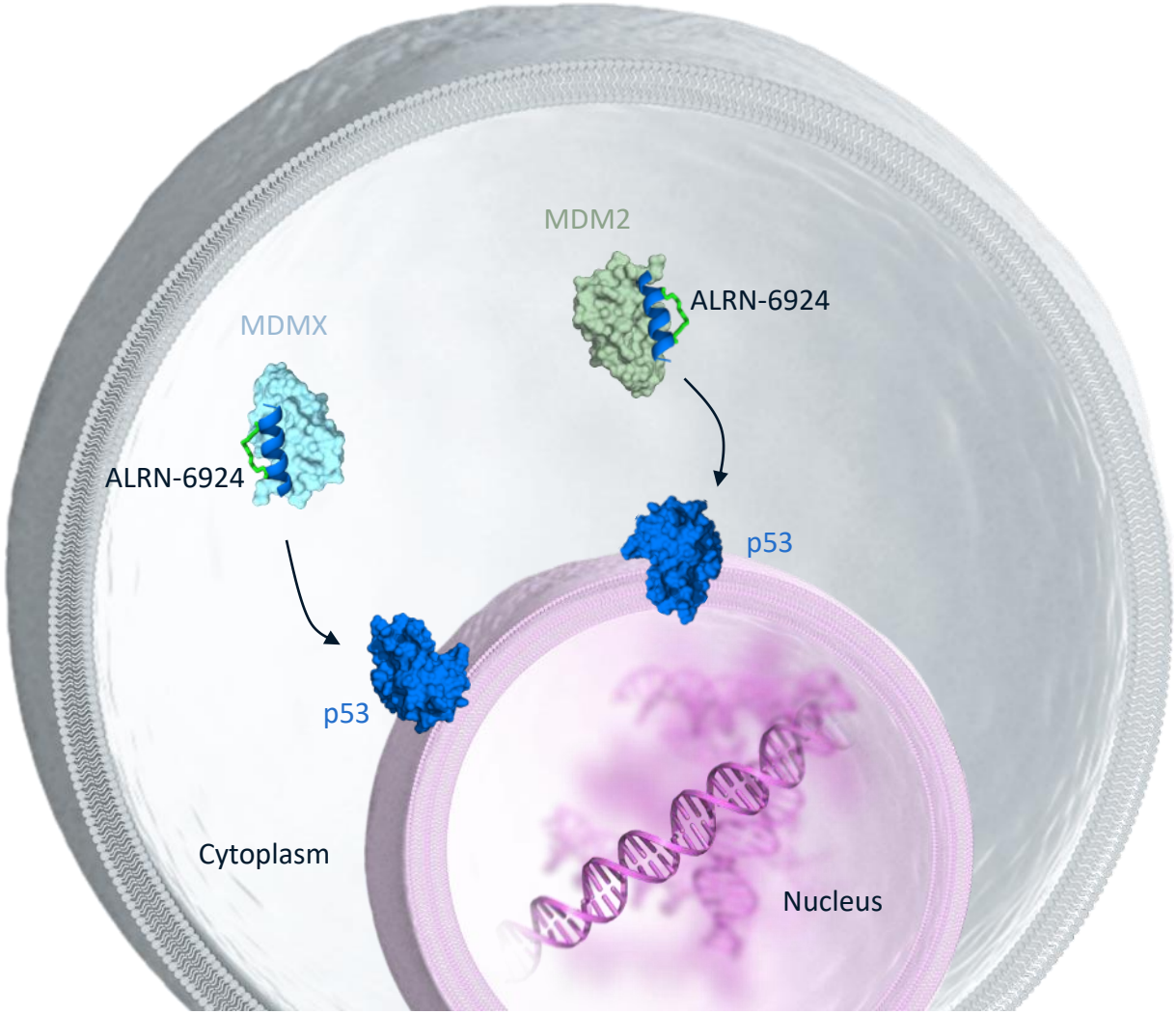
IV. Preclinical and clinical on-target, on-mechanism effects

ALRN-6924 Mechanism of Action in Wild-Type p53 Cells



ALRN-6924 is a decoy that mimics p53 and selectively binds to MDMX + MDM2, releasing p53 and, thereby, upregulating p21 to induce cell cycle arrest

ALRN-6924 Mechanism of Action in Wild-Type p53 Cells



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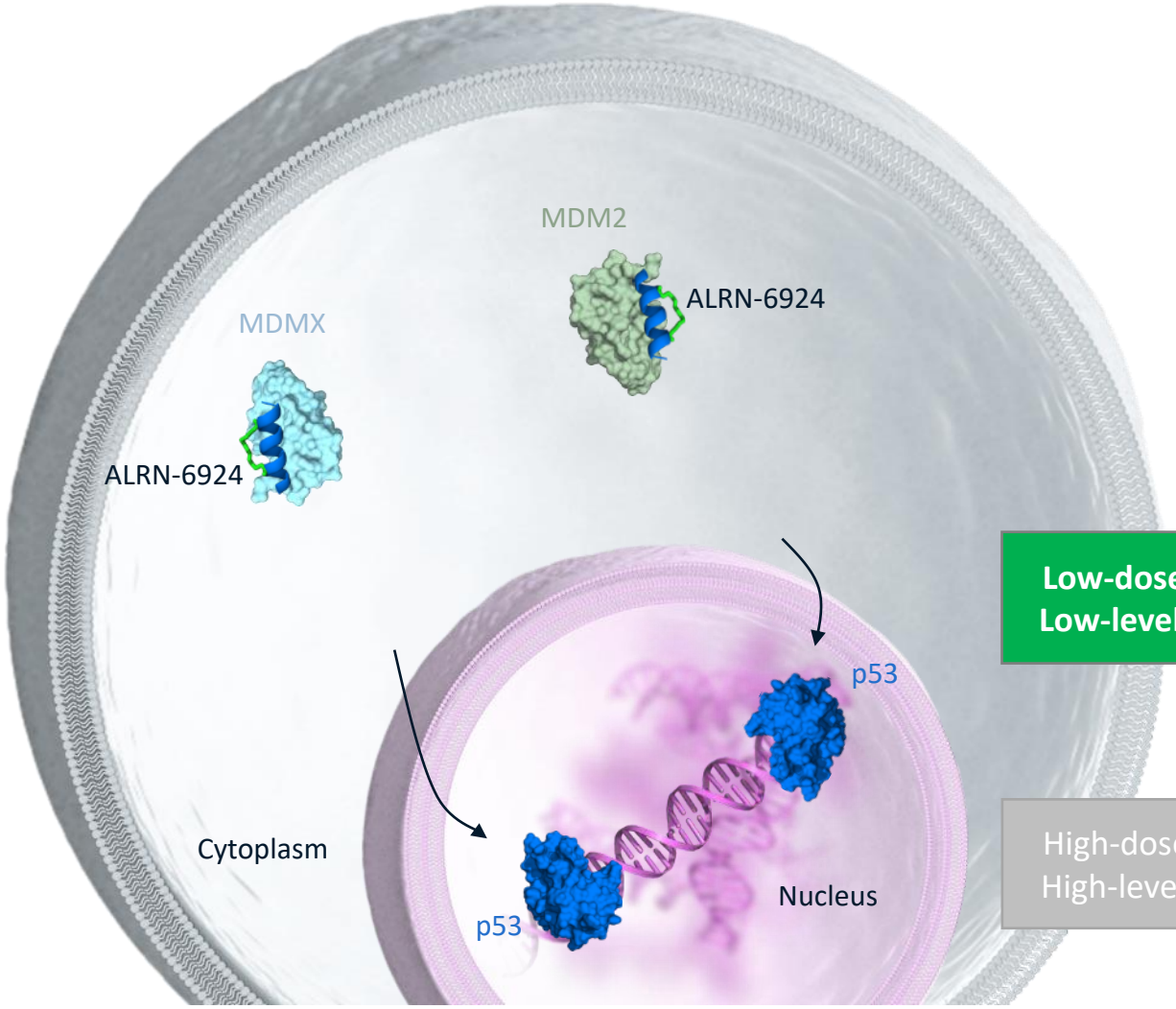
ALRN-6924 Mechanism of Action in Wild-Type p53 Cells



Low-dose ALRN-6924
Low-level p53 release

→ **p21 mediated-cell cycle arrest[1]**

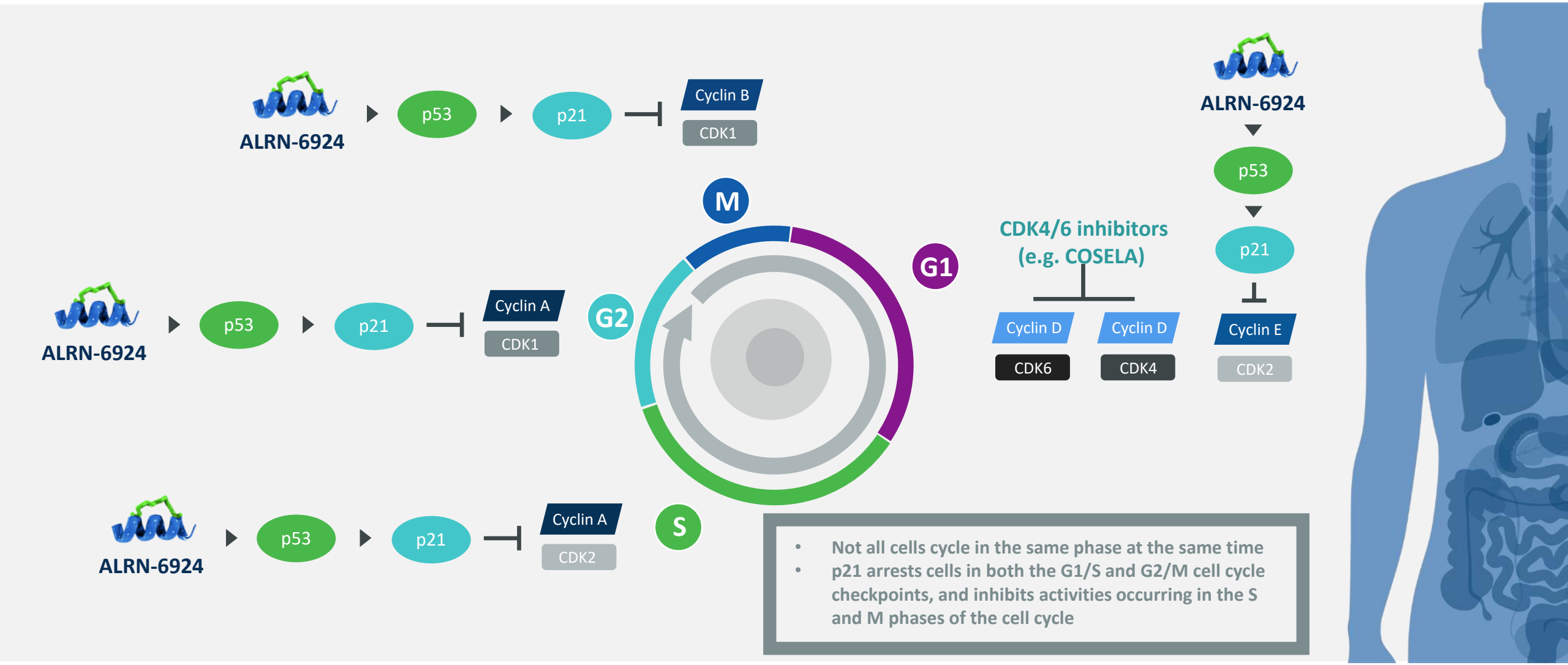
High-dose ALRN-6924
High-level p53 release

→ PUMA-, NOXA-mediated apoptosis[1]

ALRN-6924 is a decoy that mimics p53 and selectively binds to MDMX + MDM2, releasing p53 and, thereby, upregulating p21 to induce cell cycle arrest

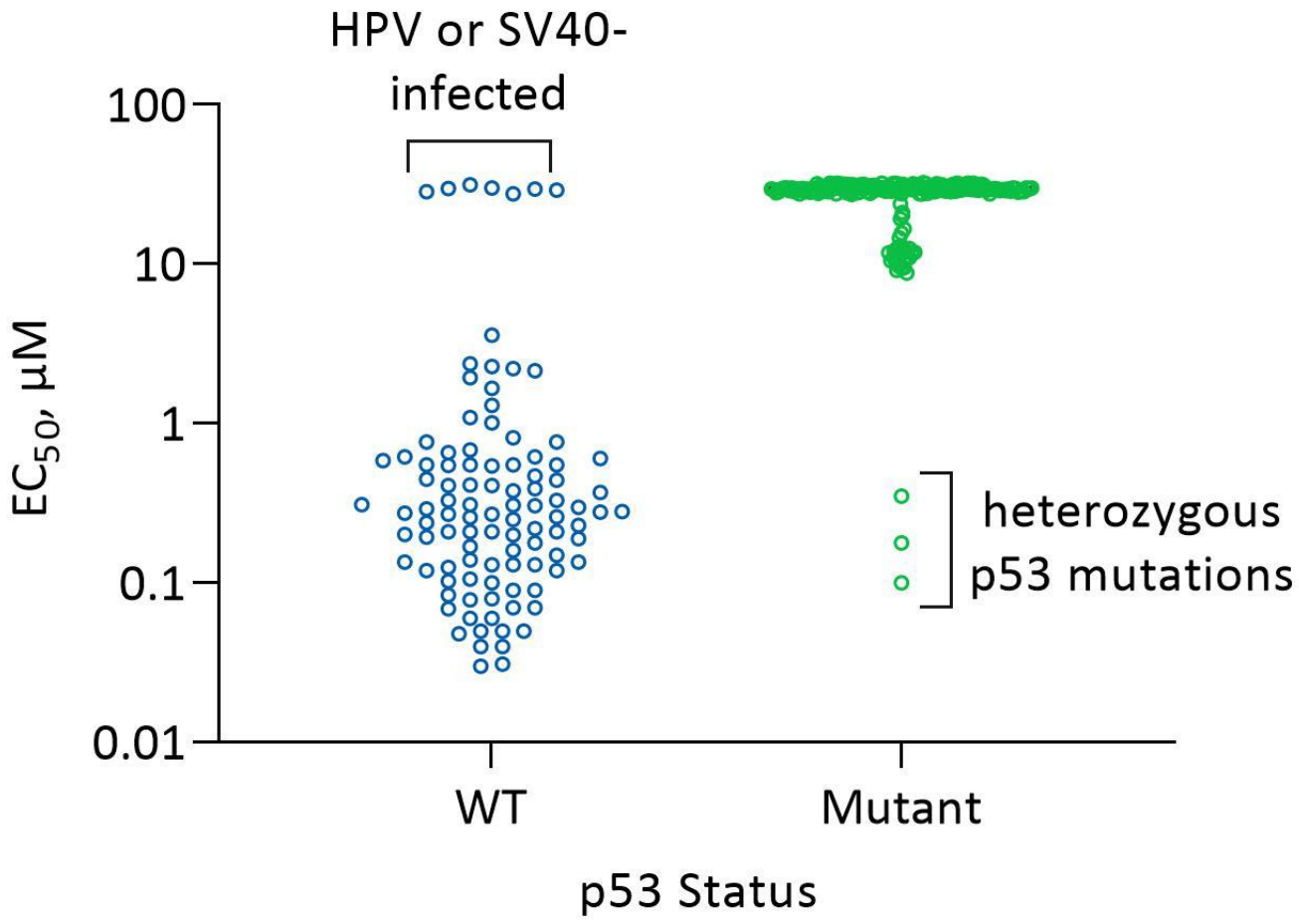
[1] Chen; Cold Spring Harb Perspect Med. 2016 Mar 1

ALRN-6924 Best-in-Class Potential in Chemoprotection: Effects on All Phases of Cell Cycle



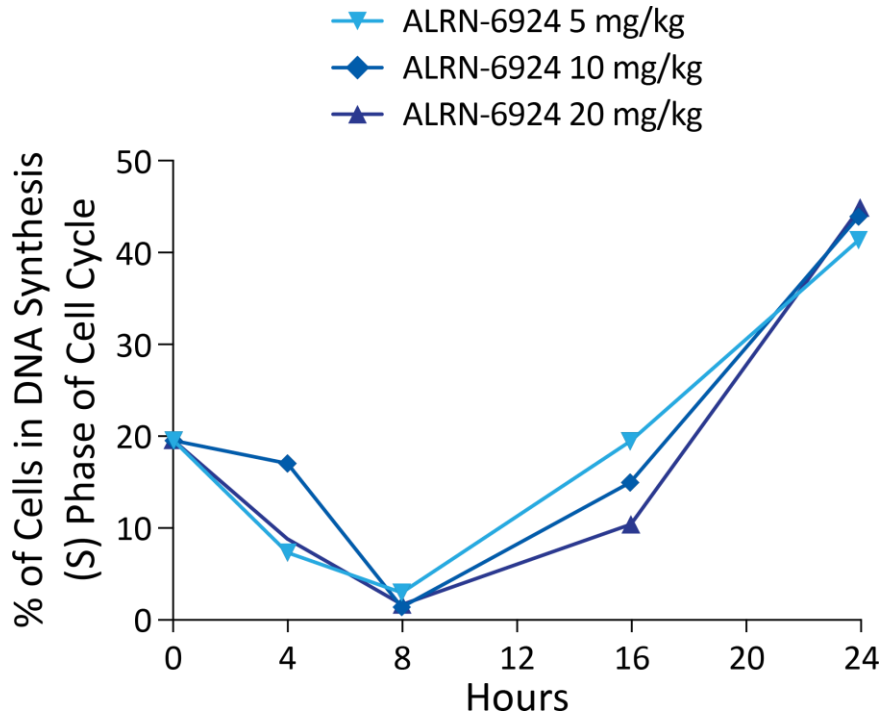
ALRN-6924 Potently Inhibits Proliferation in Wild-Type p53 Cells; Mutant Cells Are Insensitive

ALRN-6924 studies in 300 cell lines show potent, on-mechanism cellular activity



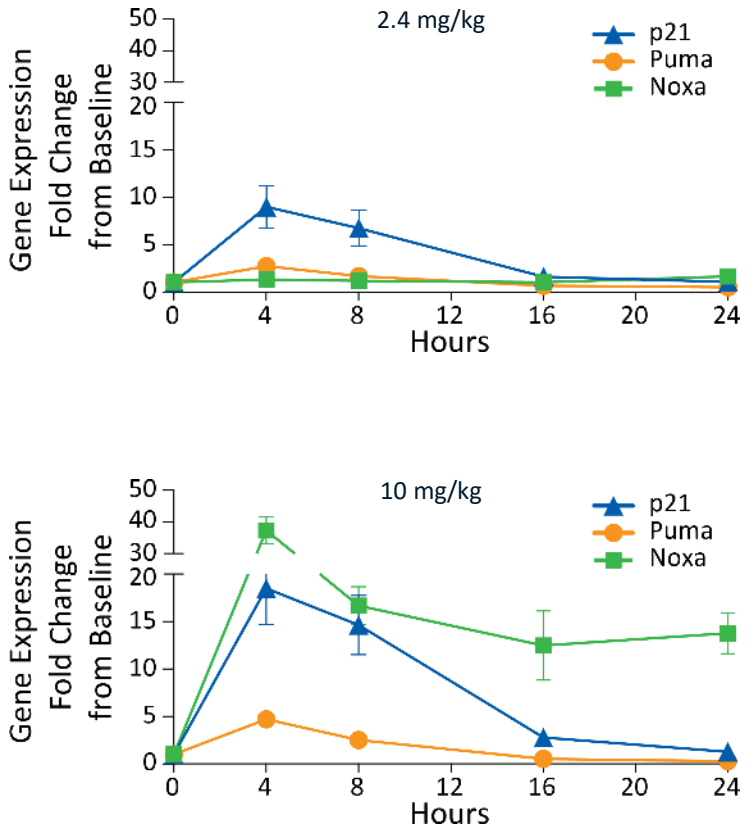
Pairawan et al, "First in class dual MDM2/MDMX inhibitor ALRN-6924 enhances antitumor efficacy of chemotherapy in TP53 wild-type hormone receptor-positive breast cancer models." Breast Cancer Research, 2021; 23: 29.

ALRN-6924 Induces Transient, Reversible Cell Cycle Arrest in Murine Hematopoietic Stem and Progenitor Cells (HSPCs) *in vivo*

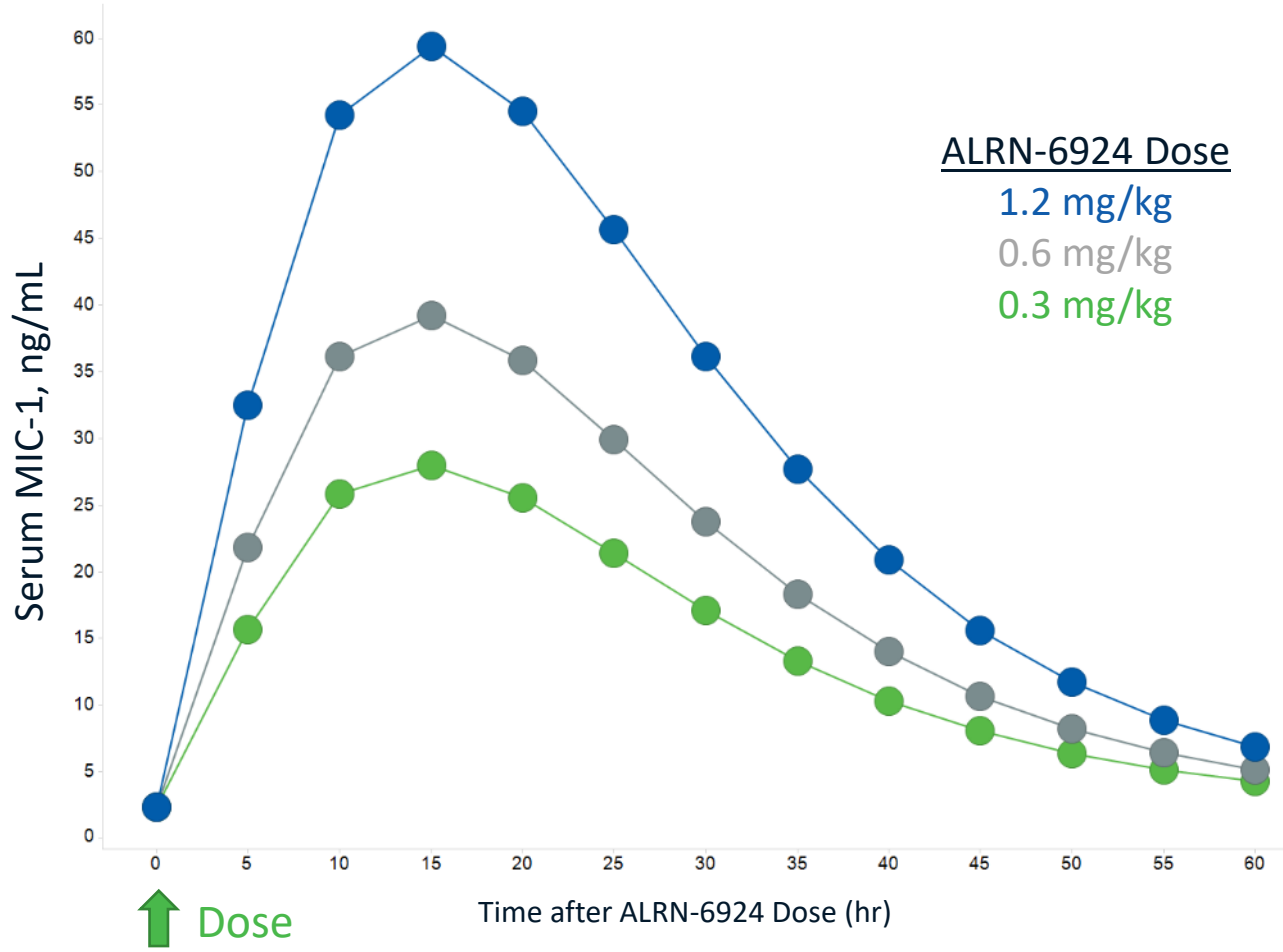


Biomarkers of Cell Cycle Arrest (p21) and Apoptosis (Puma, Noxa, And MIC-1)

Non-clinical studies†



MIC-1 levels in SCLC trial‡



† Carvajal et al, "The Investigational Peptide Drug ALRN-6924, a Dual Inhibitor of MDMX and MDM2, is an Effective Myelopreservation Agent. AACR-NCI-EORTC Conference October 2019

‡ Data modelled from this trial and other ALRN-6924 clinical studies: Meric-BernstamF., et al. Phase I trial of a novel stapled peptide ALRN-6924 disrupting MDMX and MDM2-mediated inhibition of WTp53 in patients with solid tumors and lymphomas. J. Clin. Oncol. 35(15): 2505, 2017.

Chemoprotection May Transform Chemotherapy Like Anesthesia Transformed Surgery

NASDAQ: ALRN

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