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UNLOCKING CURATIVE POTENTIAL

A New Approach to Harnessing IL-2
to Fight Cancer

Aron Knickerbocker
President and CEO

MARCH 2024

← **Treg**
Immune suppression

IL-2 traffic
HEAVY
toward Tregs

Effector →
Immune activation

ADOPT-A-HIGHWAY
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Treg

Immune suppression

Use AU-007
intelligent GPS
to reroute IL-2
to the desired
destination



Effector



Immune activation

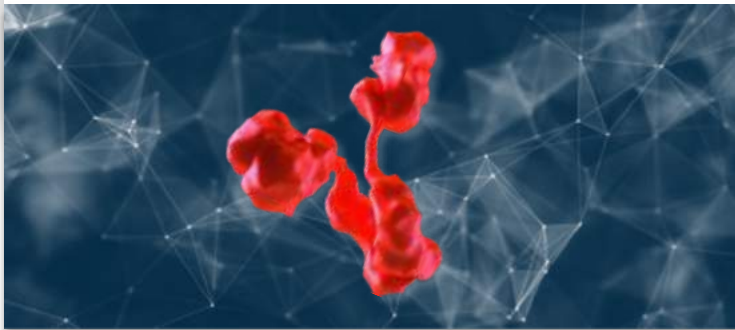
ADOPT-A-HIGHWAY

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Highly Differentiated Approach for Targeting IL-2 in Immuno-Oncology

ENABLED BY ARTIFICIAL INTELLIGENCE

- AU-007, a monoclonal antibody created by Biologic Design's innovative artificial intelligence (AI) antibody design platform



FOCUSED APPROACH

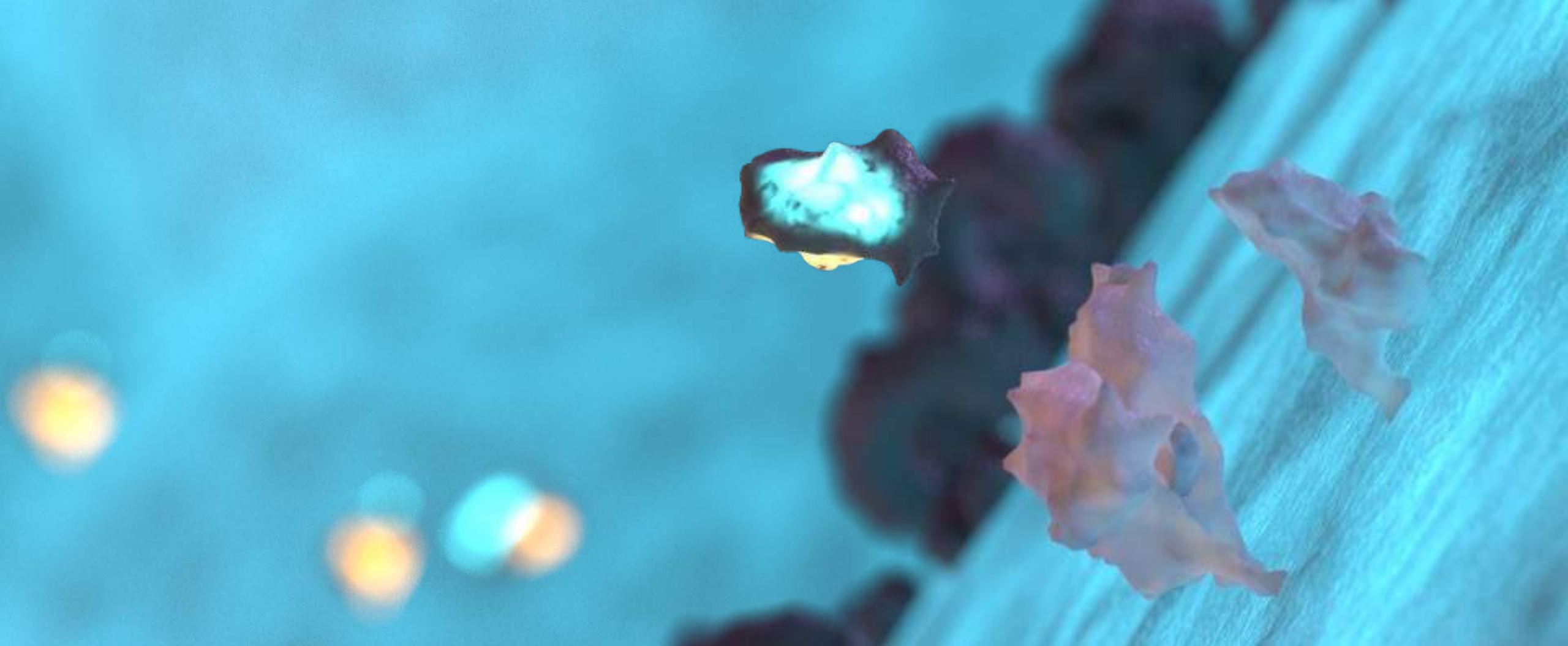
- Addressing high unmet need in solid tumors
- Phase 2 (US and Australia)
- Safe and well tolerated
- Only IL-2 agent to **lower Tregs**
- Evidence of anti-tumor activity



POSITIONED FOR SUCCESS

- Accomplished and experienced leadership team
- \$60M in Total Series A funding from ATP
- Unique competitive advantages
- Multi-indication potential



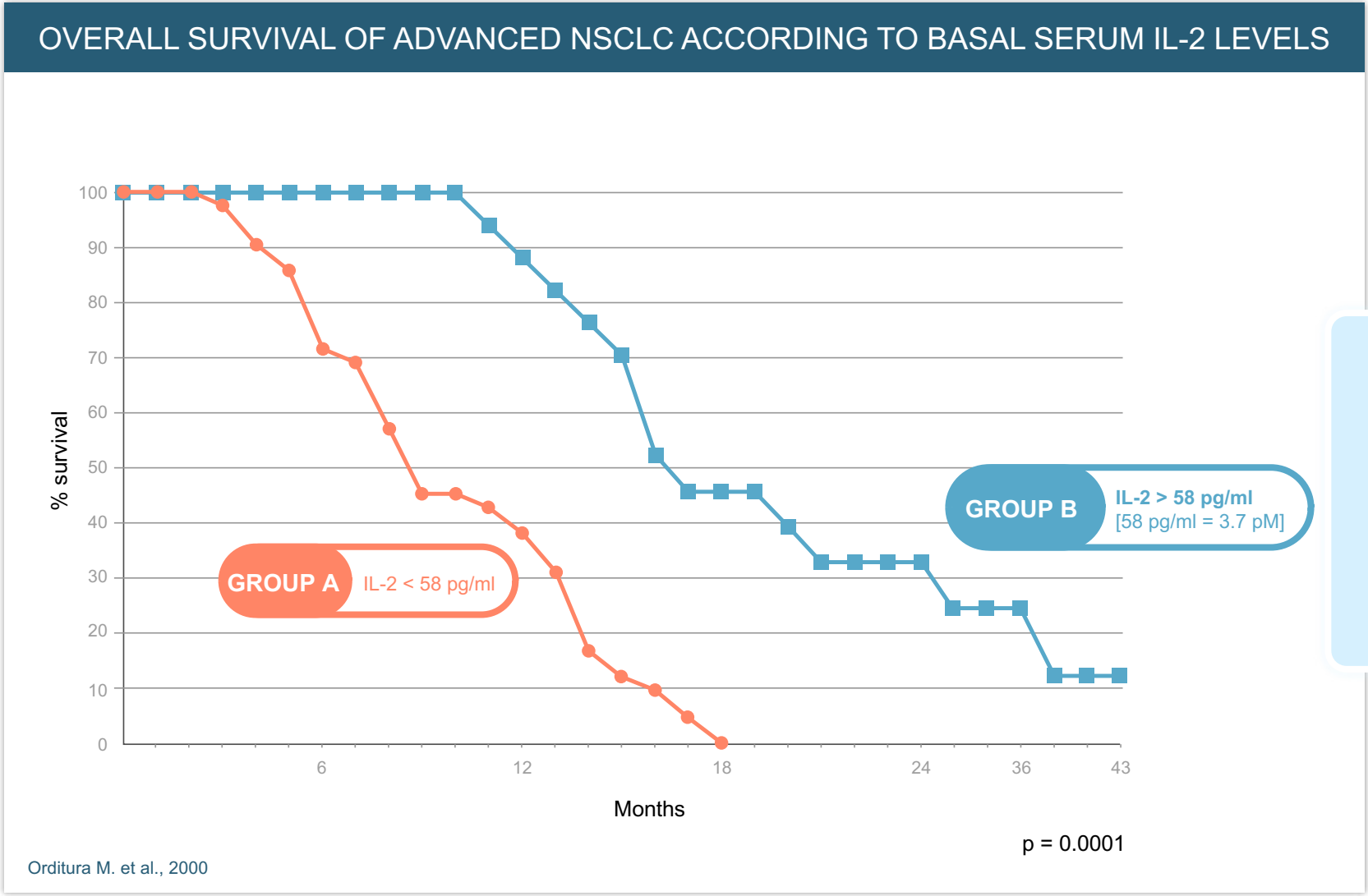


IL-2: A HISTORICALLY ELUSIVE POWER

Potent Immune Attack and Memory Against Cancers

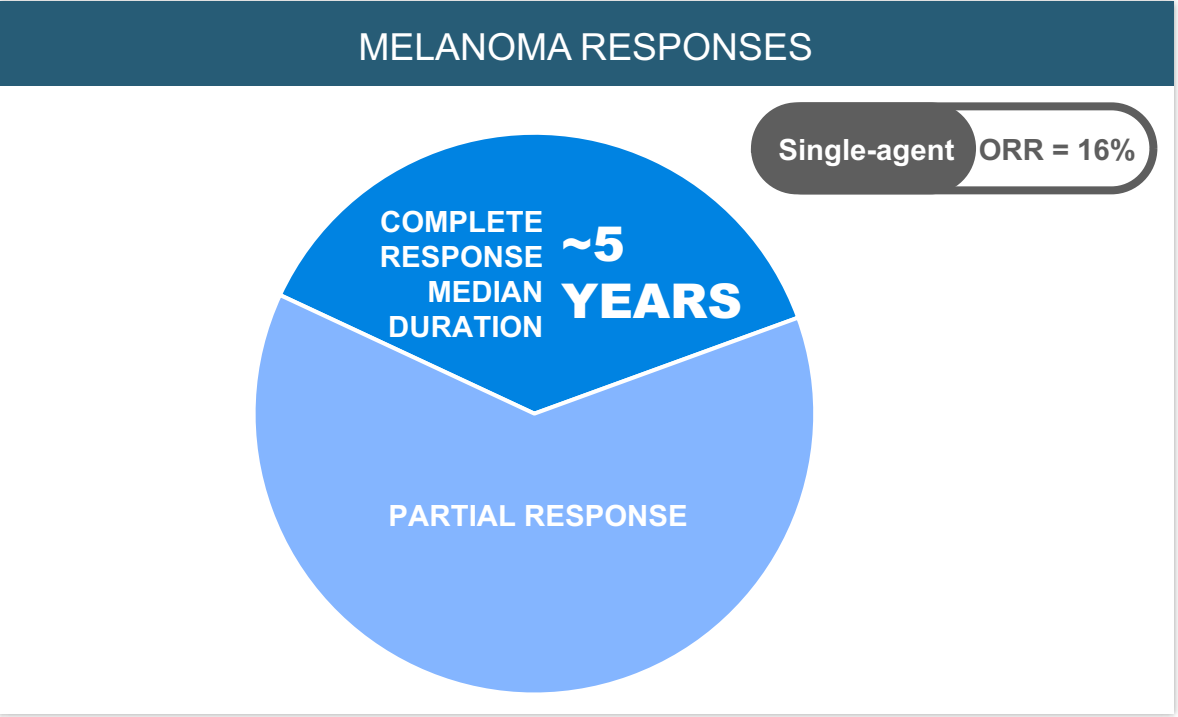
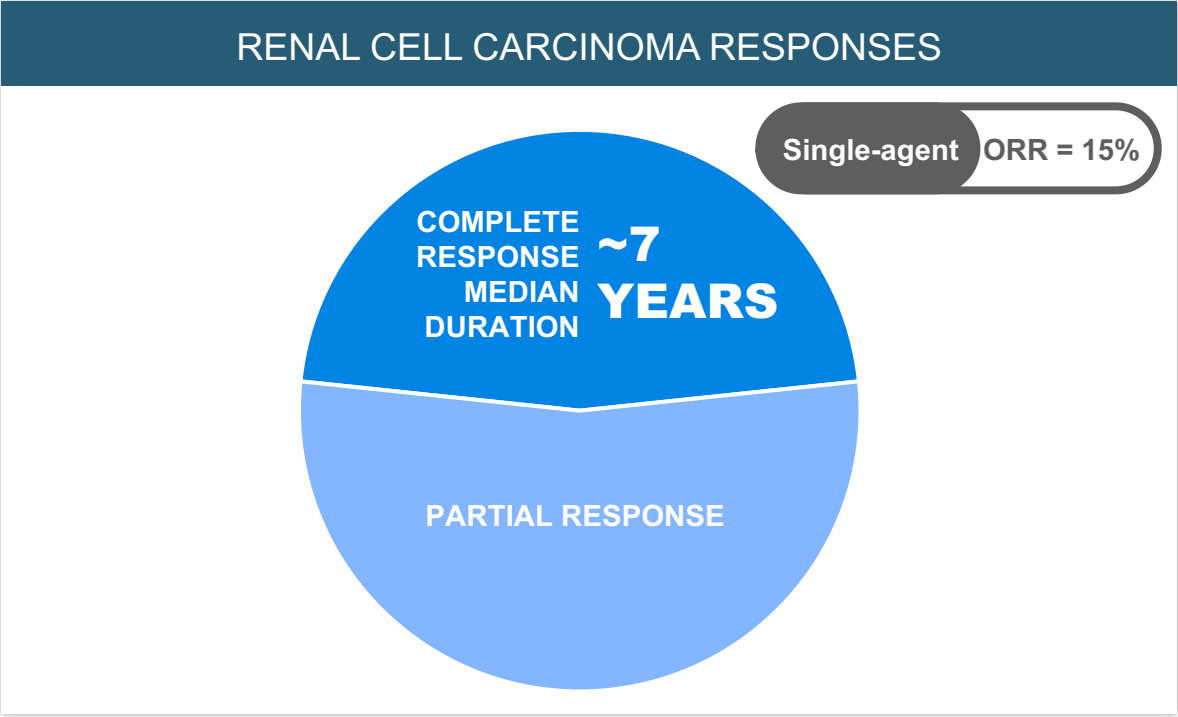
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Why Is IL-2 So Compelling? Higher Endogenous IL-2 Levels in Cancer Patients Correlate With Improved Survival



Patients received no exogenous IL-2 therapy

Why Is IL-2 So Compelling? When Proleukin® (Recombinant Human IL-2) Works, It Can Really Work, Leading to Durable, Complete Responses as a Single Agent



- Remarkable in its ability as a single agent to initiate an anti-tumor attack and generate **immune memory** of the tumor, sometimes leading to profoundly long-lasting complete responses
- Rarely used due to its significant toxicity that limits how much patients can receive, and likely constrains efficacy
- If IL-2's therapeutic index could be widened, Aulos believes that it has clinical potential akin to the PD-(L)1 checkpoint inhibitors

IL-2: Current Limitations

- Natural IL-2 is endogenously produced at low concentrations and suppresses, more than activates, the immune system because it binds to and activates regulatory T cells (Tregs), which express high-affinity receptors
- Therefore, effective treatment with IL-2 historically required very high doses to activate effector T cells, leading to an extremely toxic side effect profile, including:
 - Cytokine storms
 - Increased risk of pulmonary edema and blood vessel leakage
- IL-2 mimetics, variants, pegylated and fusion proteins
 - Create a **negative feedback loop**: the IL-2 mimetic triggers the secretion of more endogenous IL-2, tipping the balance and leading to **Treg expansion** and suppression of the very immune response that the treatment was meant to activate
 - Have an increased **risk of immunogenicity** (anti-drug antibodies)

“IL-2 therapy has a poor safety profile and restricted efficacy in only a fraction of patients.”


Klatzmann D et al., 2015

IL-2 IS A “DOUBLE-EDGED SWORD”



Both suppressing and activating the immune system with many therapeutic challenges

Aulos Accurately Predicted the Current Inadequacies of the IL-2 Competitive Landscape



Based on observations of preclinical and clinical data, we predicted in late 2021 that next generation “non-alpha” IL-2 agents would have limited efficacy and bempeg would fail broadly.

WHY?

Next generation “non-alpha” IL-2 agents:

Have only been able to achieve very low IL-2 equivalent blood concentrations

Do not prevent the negative feedback loop to immunosuppressive Tregs, resulting in **Treg expansion**

Have demonstrated no or limited single-agent clinical activity, and weak or worse combination activity

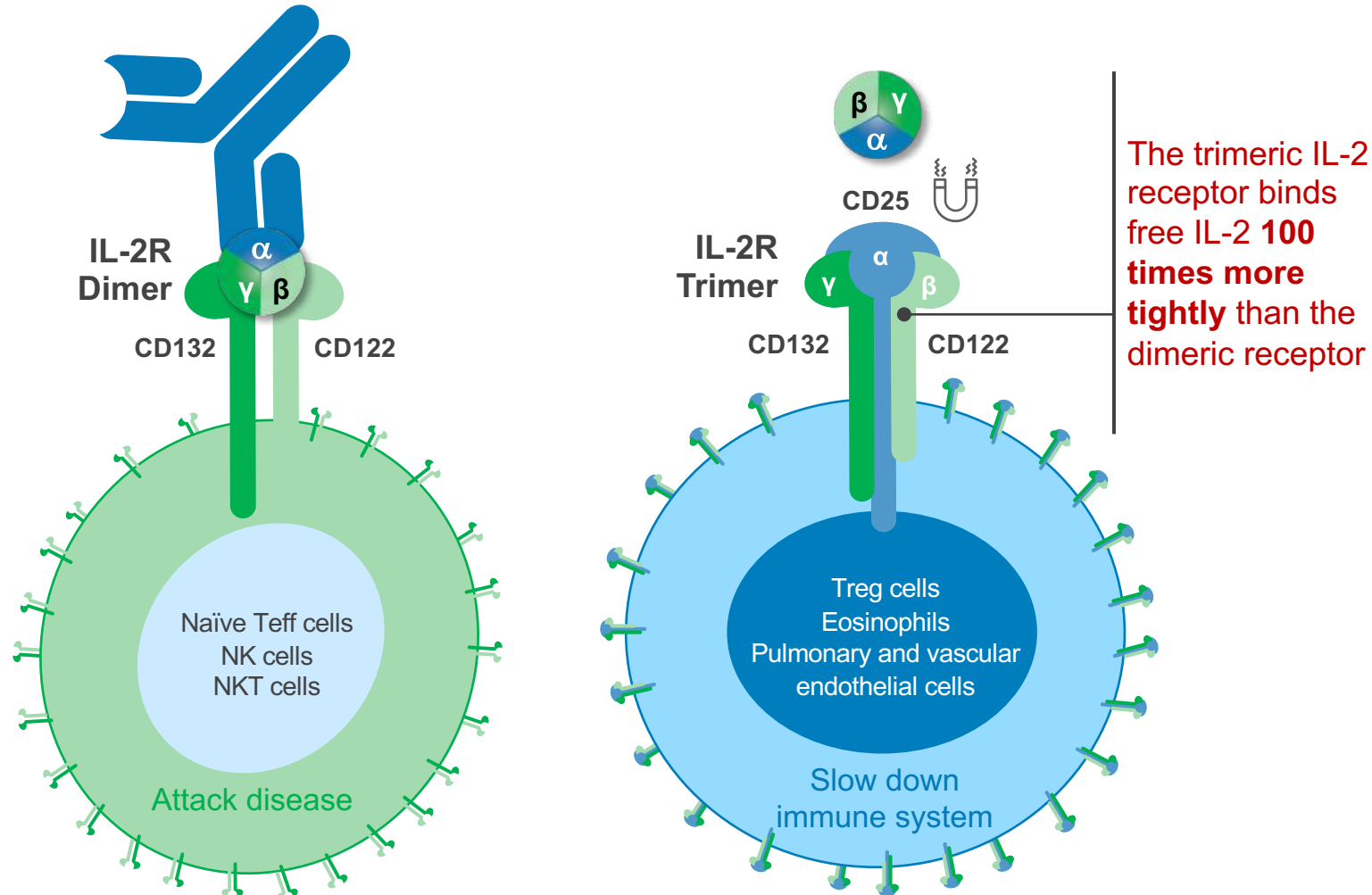


AU-007, Human Monoclonal Antibody That Redirects IL-2
Best-in-Class Potential for Immune-Sensitive Solid Tumor
Treatment

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AU-007 mAb Binds to the Part of IL-2 That Interacts With the Alpha (CD25) Receptor Subunit, Preventing Binding by IL-2 to Trimeric IL-2 Receptors

AU-007 BOUND TO IL-2 SIGNALING THROUGH IL-2R

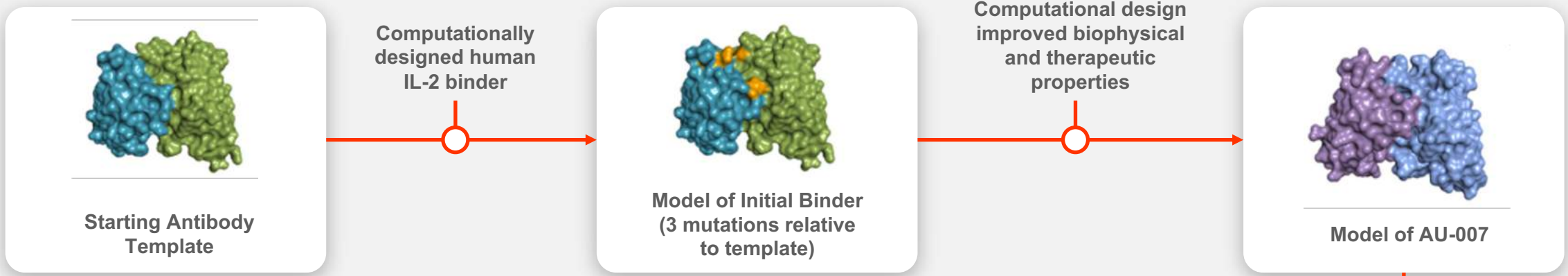


By binding to the portion of IL-2 that interacts with the alpha (CD25) receptor subunit, AU-007 prevents activation of the trimeric IL-2 receptor found on Tregs, eosinophils and vasculature

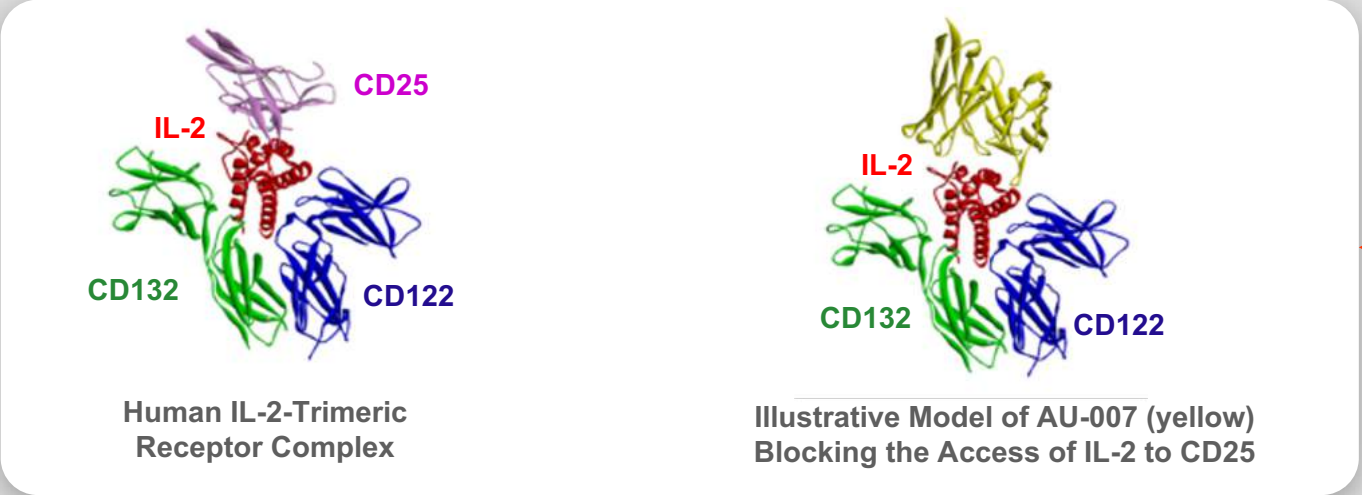
AU-007 redirects IL-2 to bind only to dimeric receptors on effector T cells, yielding significant competitive advantages for efficacy and safety

Computational Design for Precise Blocking of IL-2's Binding to Alpha (CD25) Receptor Subunit Contained in Trimeric Receptors on Tregs, Vasculature and Eosinophils

AU-007 Design

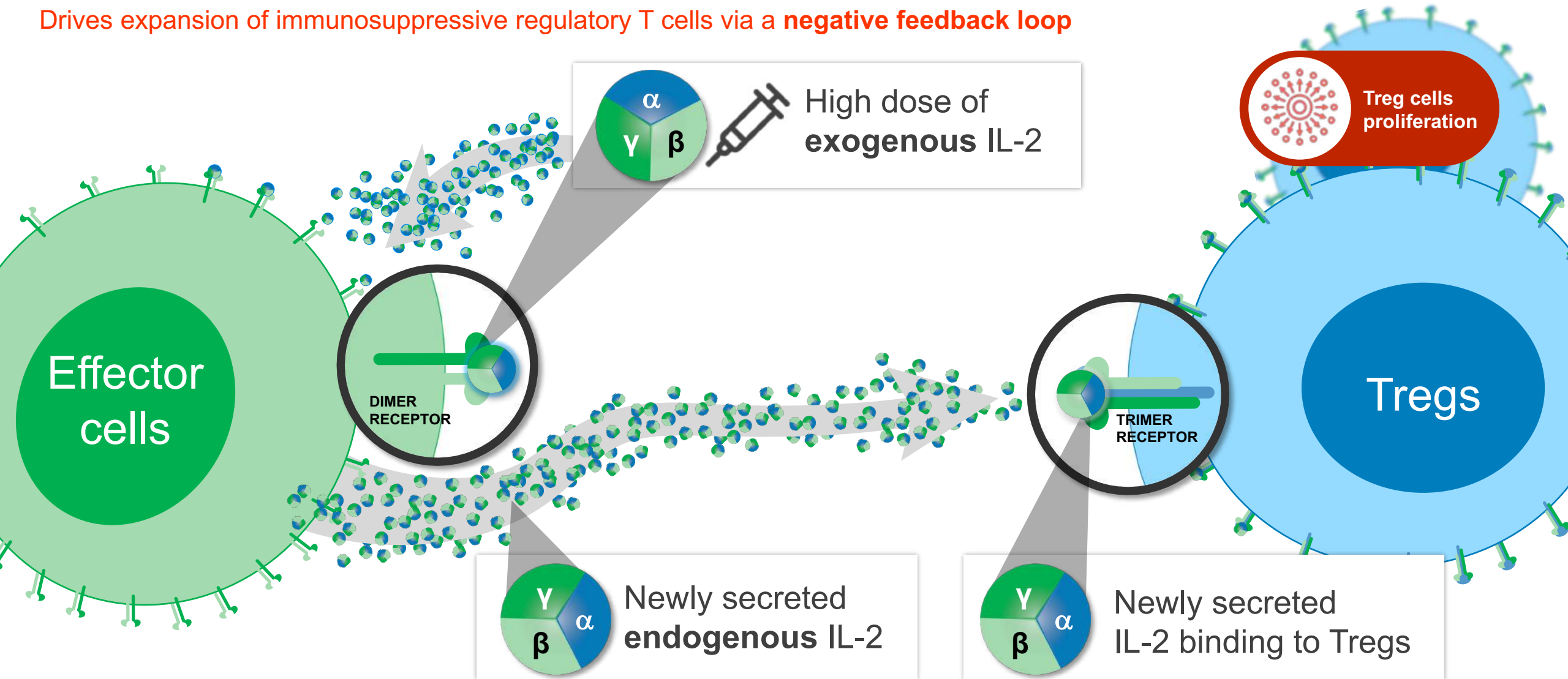


AU-007 Function



Exogenous IL-2 Therapies, Even “Non-Alpha” Therapies, Lead to Production of Endogenous IL-2 by Activated Effector Cells

Drives expansion of immunosuppressive regulatory T cells via a **negative feedback loop**



Clinical Evidence of the Negative Feedback Loop in Action: Competing Products All Drive the Expansion of Immunosuppressive Tregs

| DRUG/PROGRAM | COMPANY | ISSUE(S) |
|---|----------------------------------|---|
| THOR-707 <i>Pegylated IL-2</i> | <i>Sanofi</i> | After first dose: increased peripheral blood Tregs up to 3.5 times ¹ |
| Bempegaldesleukin <i>Pegylated IL-2</i> | <i>Nektar/BMS</i> | 27-fold increase in peripheral blood Tregs ² |
| ANV419 <i>IL-2 fusion to antibody</i> | <i>Anaveon</i> | ~2-fold expansion of Tregs ³ |
| Nemvaleukin alfa <i>IL-2 fusion to CD25</i> | <i>Mural (formerly Alkermes)</i> | ~2-fold expansion of Tregs ⁴ |
| MDNA11 <i>Albuminated IL-2 superkine</i> | <i>Medicenna</i> | After first dose: Tregs rise in peripheral blood, fold change not reported ⁵ |
| WTX-124 <i>Masked IL-2</i> | <i>Werewolf</i> | Tregs rise, fold change not reported ⁶ |

One Treg
can inhibit ~10
cancer-fighting
effector
T cells⁷

AU-007 Uniquely Tips Balance Toward Immune Activation, Away From Immune Suppression by Blocking Negative Feedback Loop to Tregs



Competitive Differentiation

| | Full blockage of IL-2 binding to CD25 | Prevent Treg expansion and binding to vascular endothelium | Avoid negative feedback from endogenous IL-2 | Human IgG1 mAb: Good PK, low potential for immunogenicity |
|---------------------------------|---|---|--|--|
| aulos | ✓ | ✓ | ✓ | ✓ |
| High dose IL-2 | X | X | X | X |
| Modified IL-2 | X / ✓ | X / ✓ | X | X |
| Fusion proteins (incl. mAbs) | X / ✓ | X / ✓ | X | X / ✓ |

Aulos’ approach to IL-2 modulation addresses challenges



Rapidly Advancing Clinical Development of AU-007

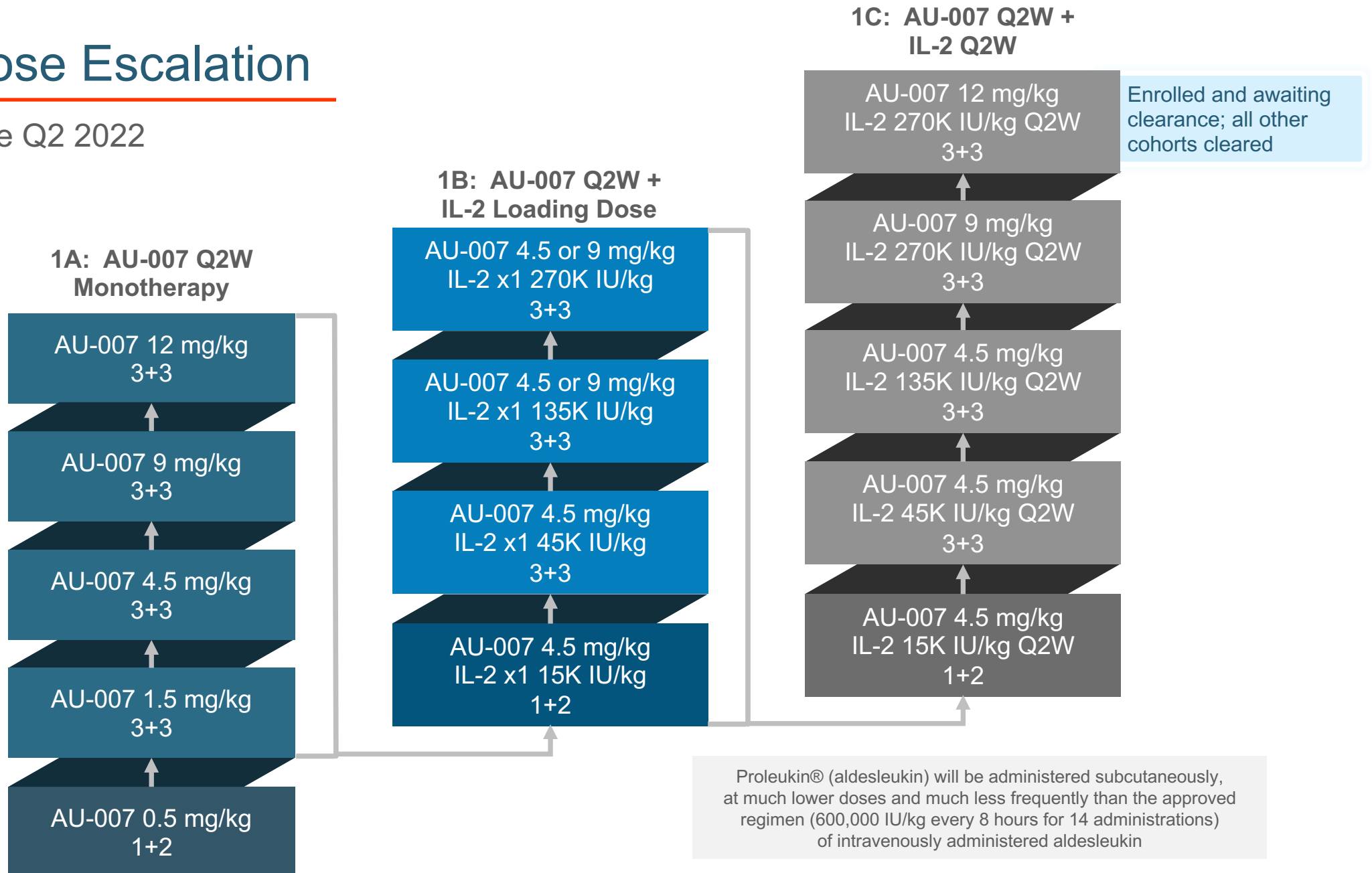
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Summary of Clinical Program and Recent Initiation of Phase 2

- **Stable disease/objective response results in Proleukin[®] (aldesleukin)-containing arms (1B and 1C)**
 - Profound tumor shrinkage in patient with metastatic melanoma who had progressed on two checkpoint inhibitor regimens
 - Tumor shrinkages also observed in NSCLC, renal cell carcinoma, bladder, head & neck (nasopharyngeal), colorectal
 - Additional anti-tumor activity seen since SITC Annual Meeting in November 2023
- **Excellent safety profile; Mostly low-grade AEs related to IL-2 MOA and evidence of immune activation**
- **Pharmacodynamic data show increased immune activation with addition of low-dose, subcutaneous Proleukin[®]**
- **Current status**
 - Phase 2 cohort opened with single administration low-dose, subcutaneous of Proleukin[®]
 - Second-line melanoma and second-/third-line RCC
 - 9 mg/kg AU-007 plus single dose of Proleukin[®] at 135,000 IU/kg
 - Allows for additional dose(s) of Proleukin[®] upon tumor growth (boost dosing)
 - Phase 2 cohort opened with Q2W low-dose, subcutaneous Proleukin[®] regimen
 - Second-line melanoma and second-/third-line RCC
 - 9 mg/kg AU-007 plus Q2W Proleukin[®] at 135,000 IU/kg
- **High enthusiasm and engagement from sites and investigators**

Phase 1 Dose Escalation

Dosing began late Q2 2022



Clinical Development: Speed to Proof of Concept

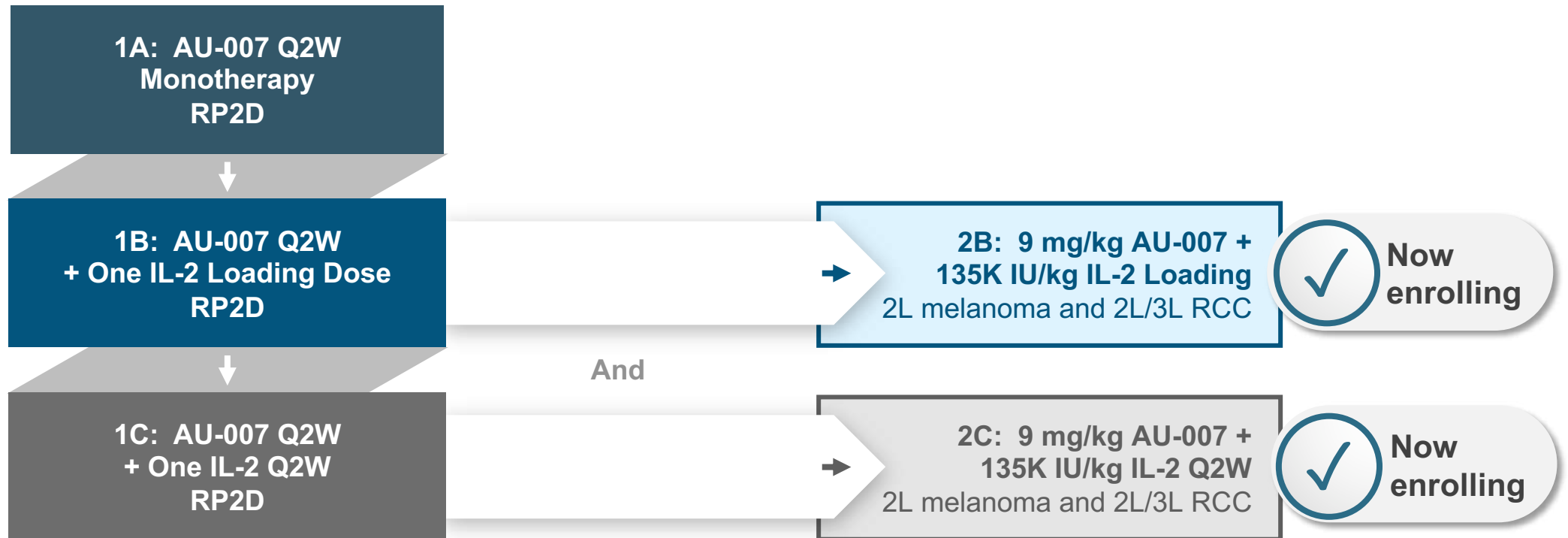
Now Enrolling in Phase 2 in Melanoma and RCC With Two IL-2 Schedules

AU-007 Phase 1 Dose Escalation

Australia initially; IND cleared October 2022

Phase 2 Expansion Cohorts

Australia & US

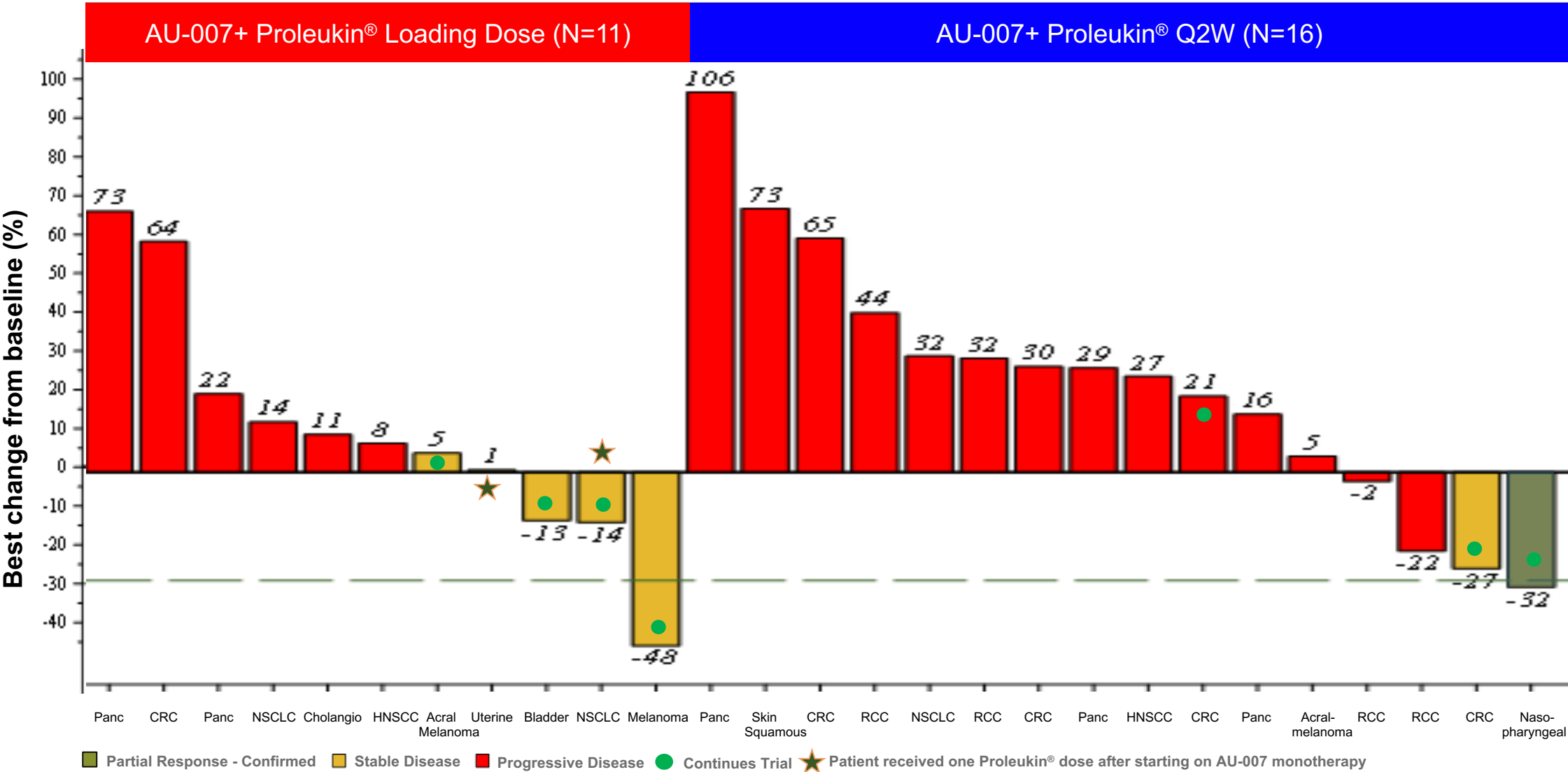


Phase 1 Dose Escalation Data Presented at SITC 2023: Safety by Type of Adverse Event

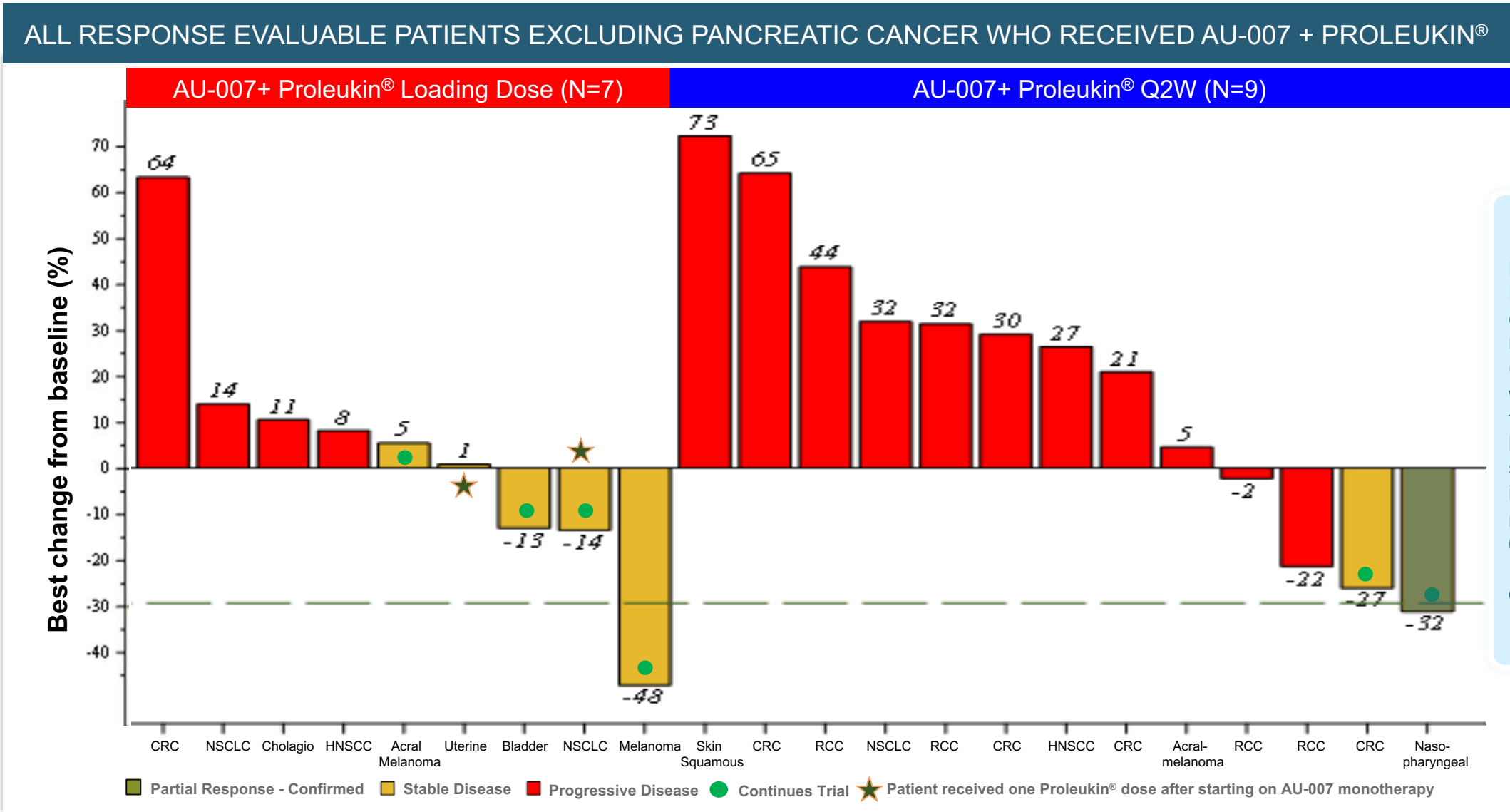
| Drug-related AEs in > 5% of patients n=42 | | |
|---|-----------------------|-----------------------|
| Adverse Event | Grade 1 or 2 n (%) | Grade 3 or 4 n (%) |
| Fatigue | 7 (17) | 0 |
| Nausea | 6 (14) | 0 |
| Pyrexia | 5 (12) | 0 |
| Chills | 4 (10) | 0 |
| Vomiting | 3 (7) | 0 |
| Lymphopenia | 0 | 3 (7) |
| <ul style="list-style-type: none">• 1 patient with Grade 3 lymphopenia, 2 with Grade 4 – all transient (3-7 days)• No DLTs; 1 Related SAE – Grade 2 cytokine release syndrome (CRS) in Arm 1C Cohort 3 | | |

All drug-related AEs were Grade 1 or 2 except for 3 patients receiving AU-007 + aldesleukin with transient (3-7 day) Grade 3 or 4 lymphopenia that were not associated with adverse outcomes. Transient lymphopenia is a known effect of IL-2 treatment as lymphocytes traffic out of blood and into tissue. No patients discontinued for a drug related adverse event; no DLTs observed.

Phase 1 Dose Escalation: AU-007 + Proleukin®: Best Response vs. Baseline



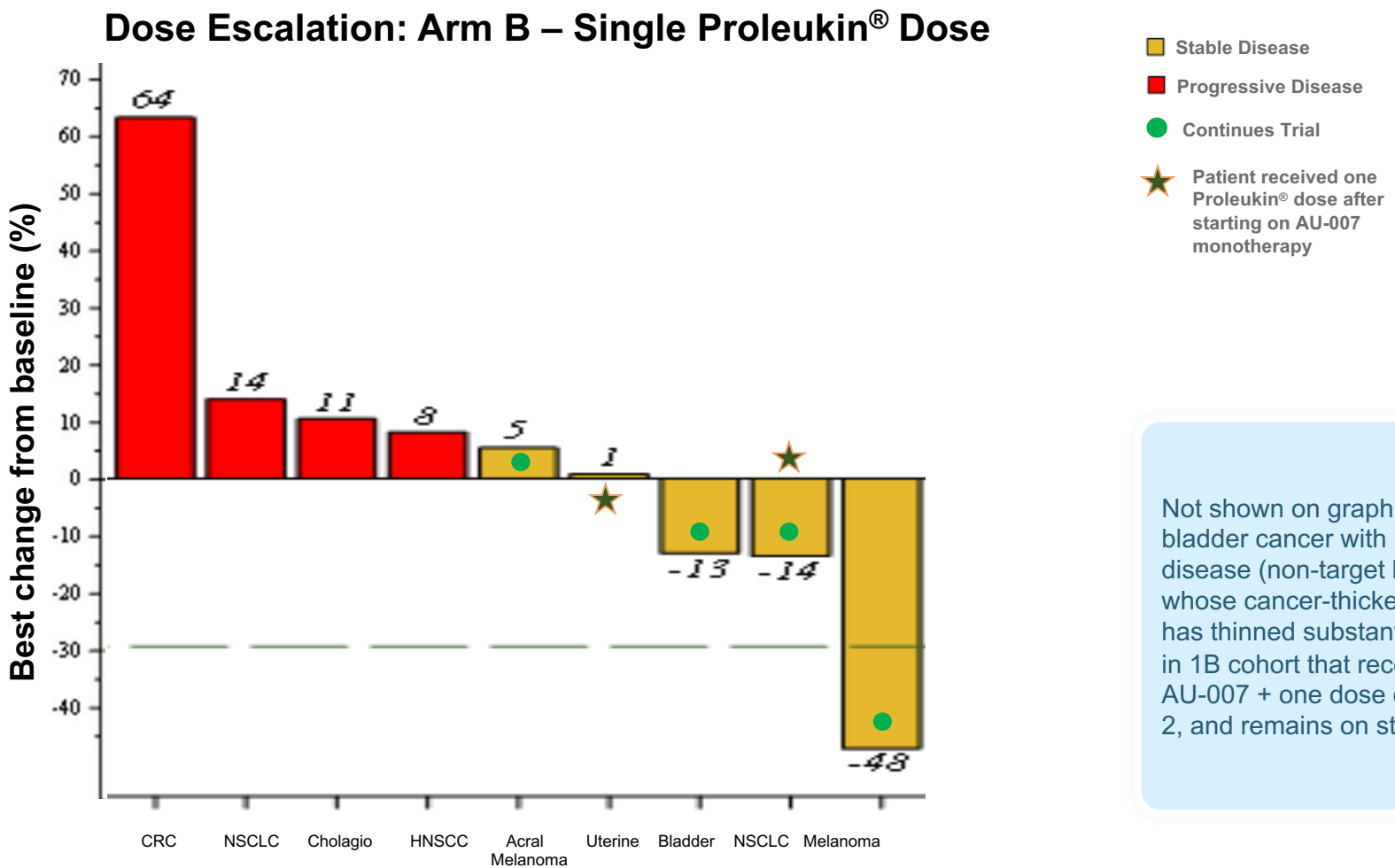
Phase 1 Dose Escalation: AU-007 + Proleukin®: Best Response in Immune-Sensitive Tumors



Not shown on graph: Patient with bladder cancer with non-measurable disease (non-target lesions only) whose cancer-thickened bladder wall has thinned substantially. Patient is in 1B cohort that received 4.5 mg/kg AU-007 + one dose of 45K IU/kg IL-2, and remains on study.

Phase 1 Dose Escalation: AU-007 + Proleukin®: Best Response in Immune-Sensitive Tumors

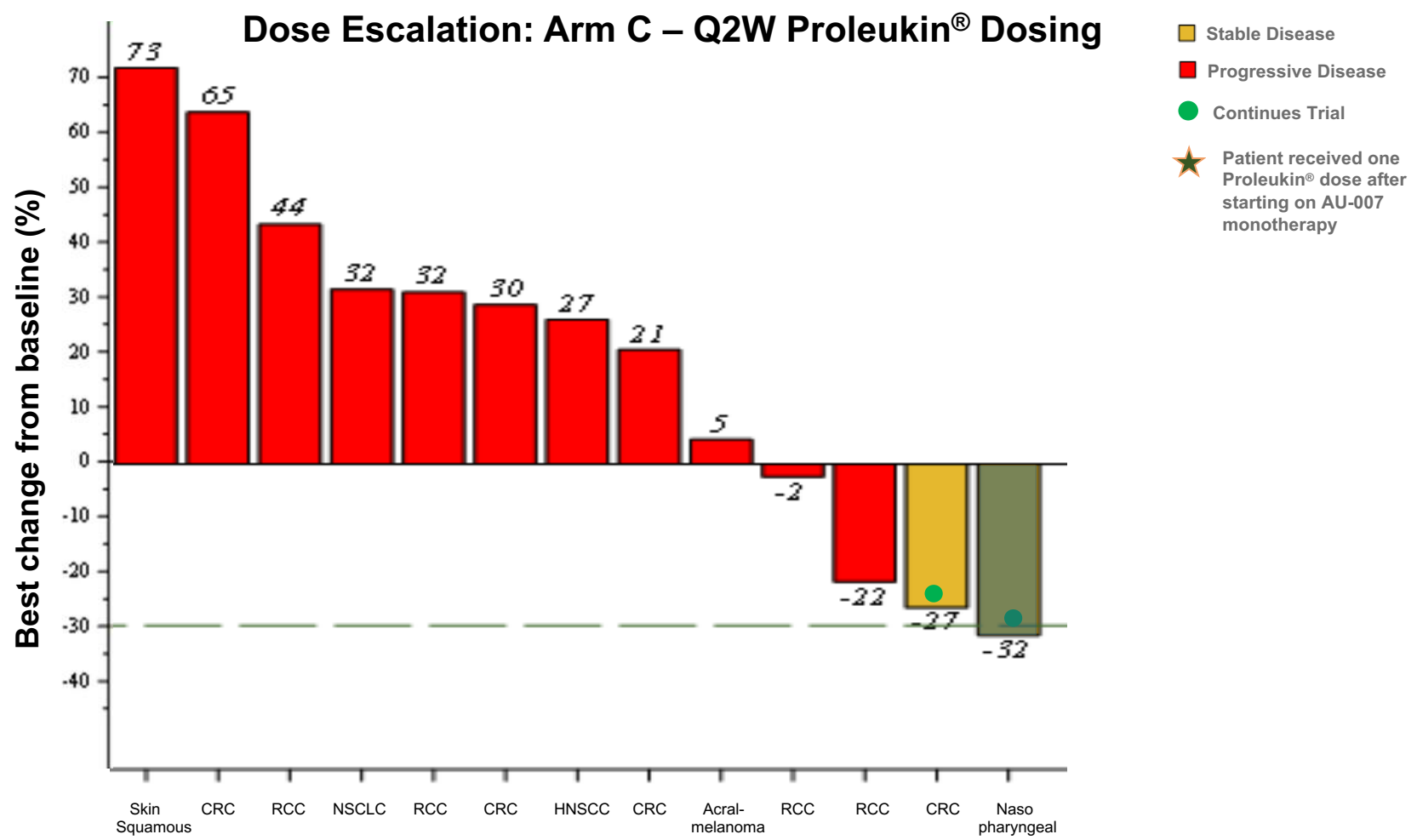
ALL RESPONSE EVALUABLE PATIENTS EXCLUDING PANCREATIC CANCER WHO RECEIVED AU-007 + PROLEUKIN®



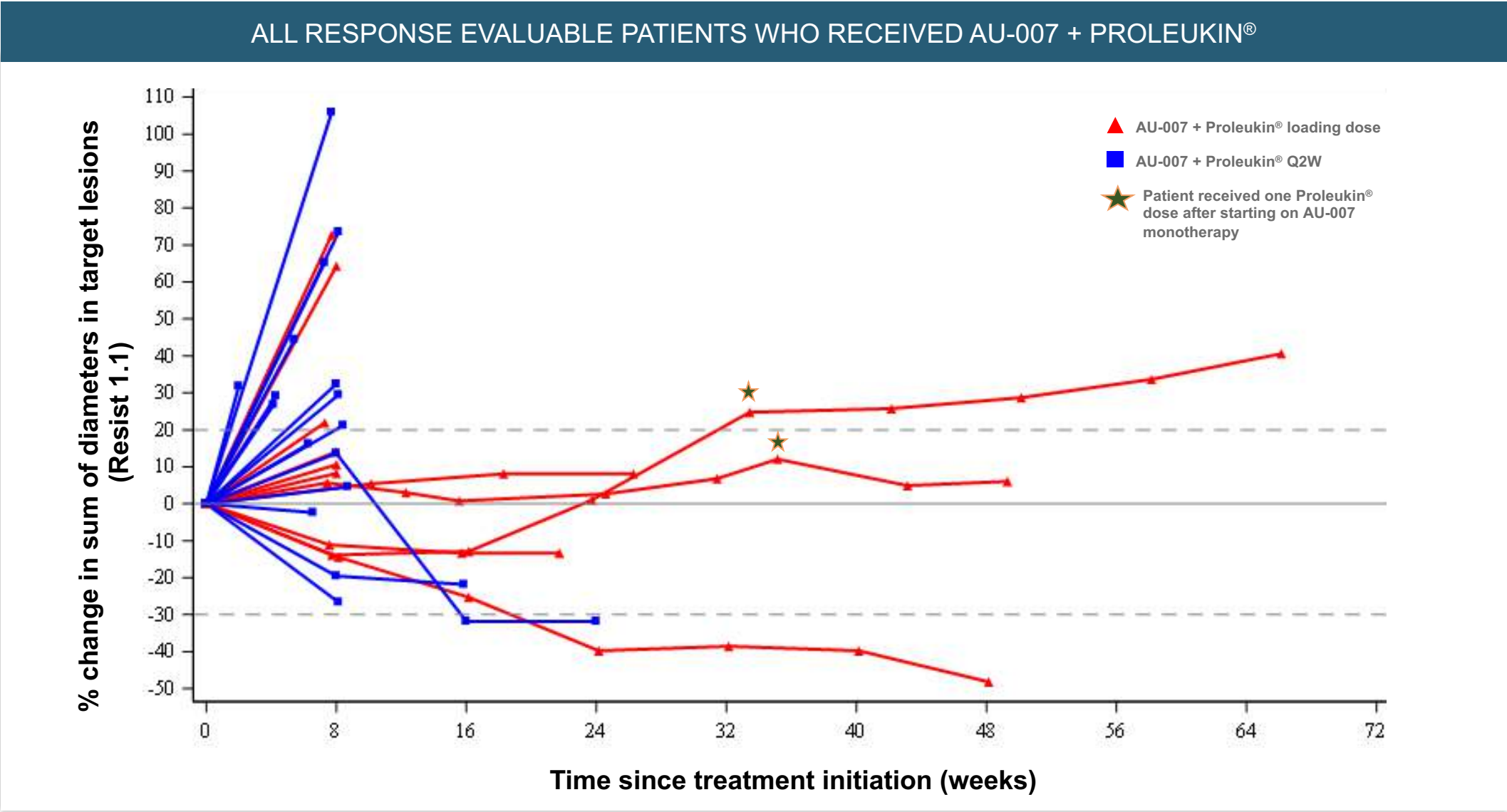
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Phase 1 Dose Escalation: AU-007 + Proleukin®: Best Response in Immune-Sensitive Tumors

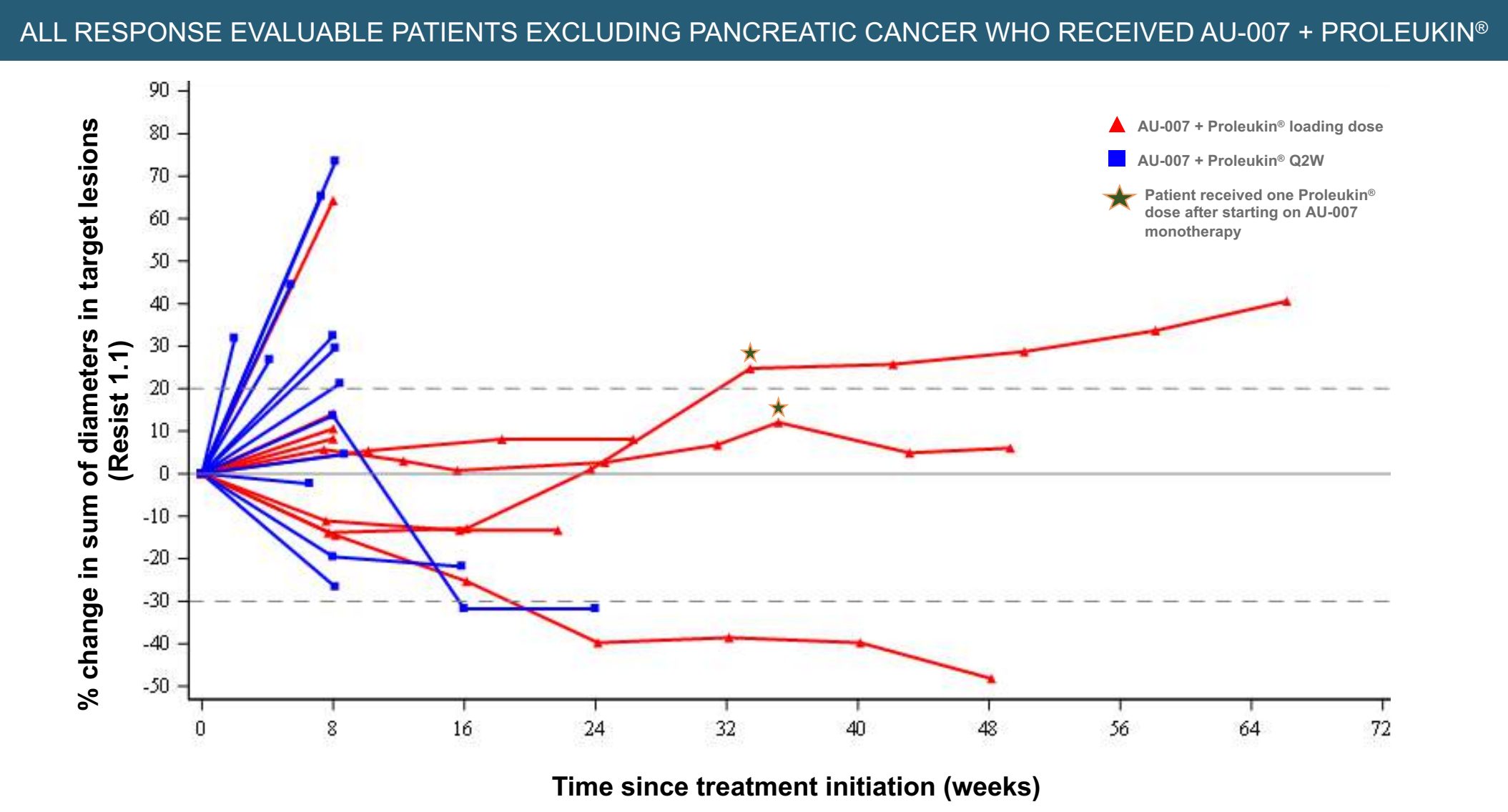
ALL RESPONSE EVALUABLE PATIENTS EXCLUDING PANCREATIC CANCER WHO RECEIVED AU-007 + PROLEUKIN®



AU-007 + Proleukin®: % Change vs. Baseline Over Time

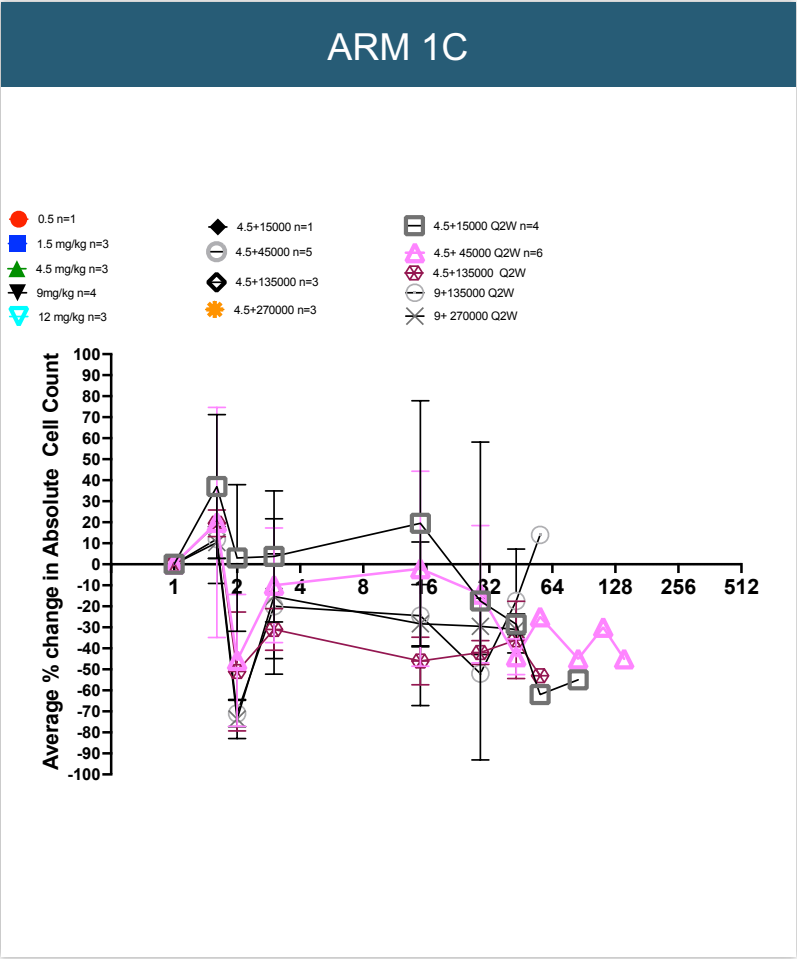
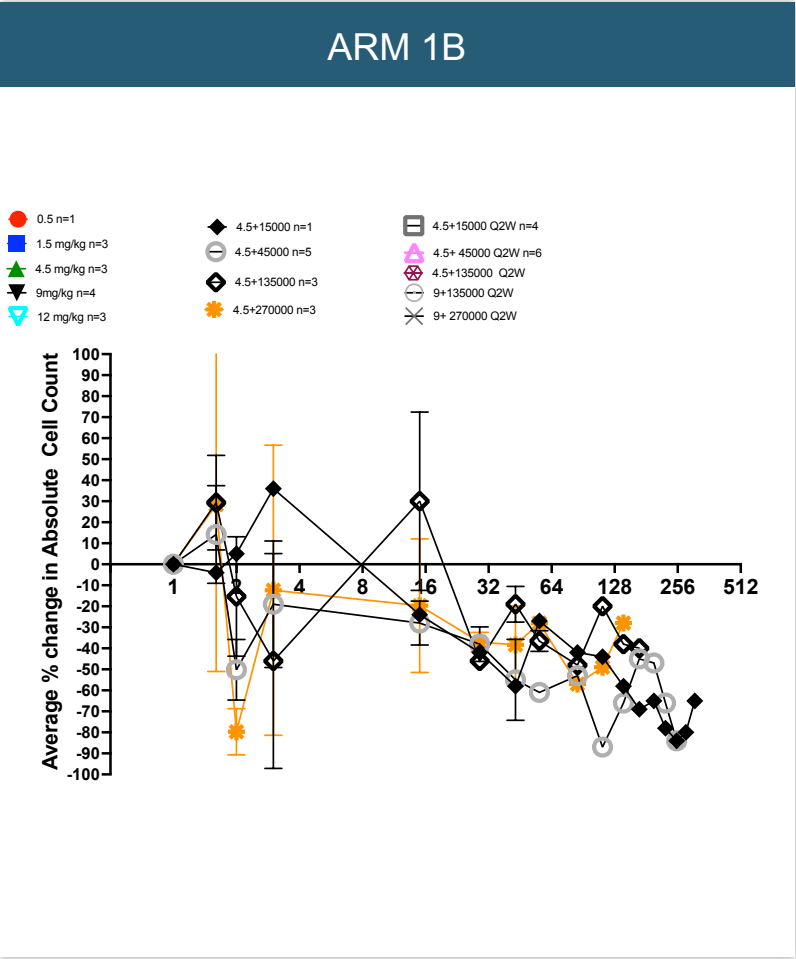
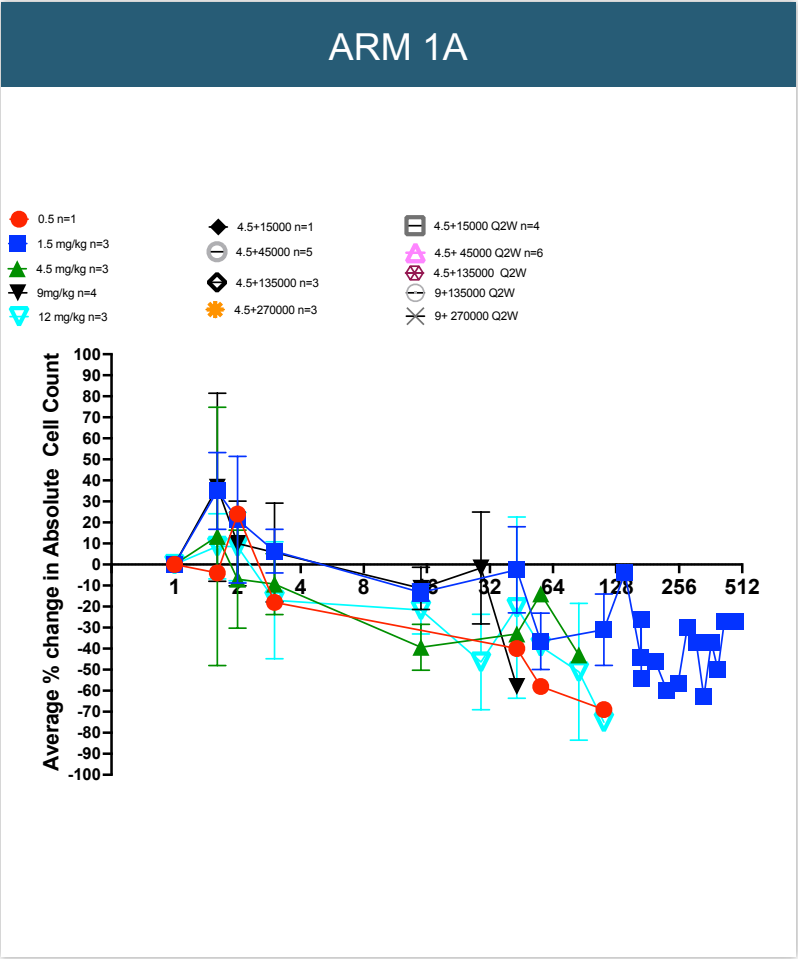


AU-007 + Proleukin®: % Change vs. Baseline Over Time



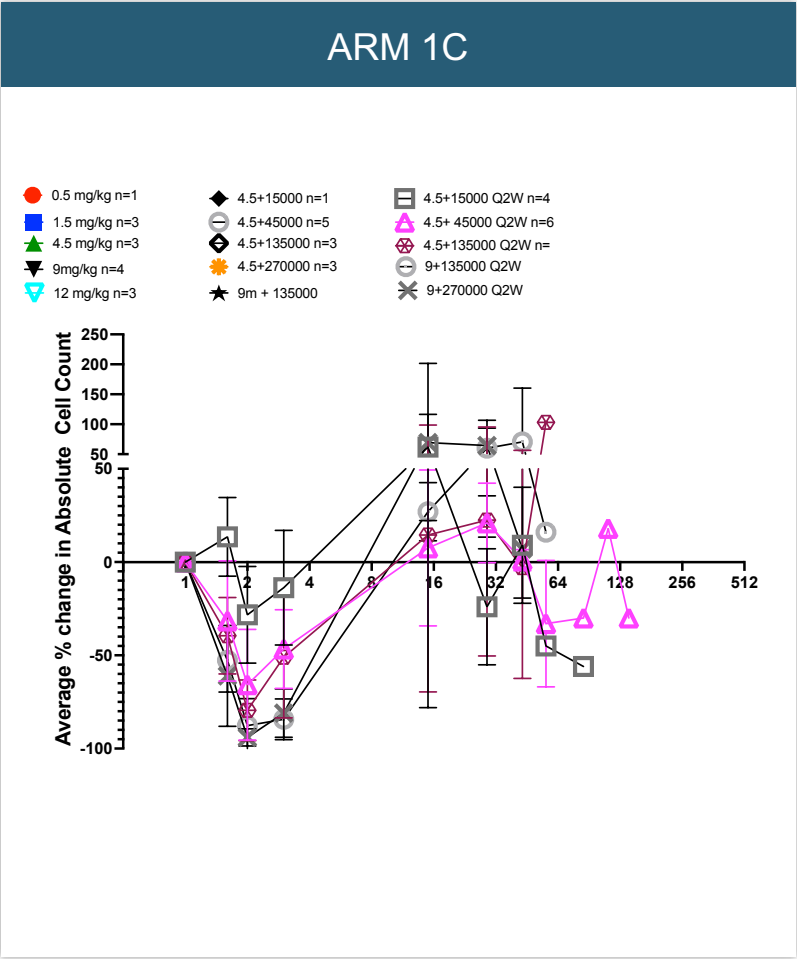
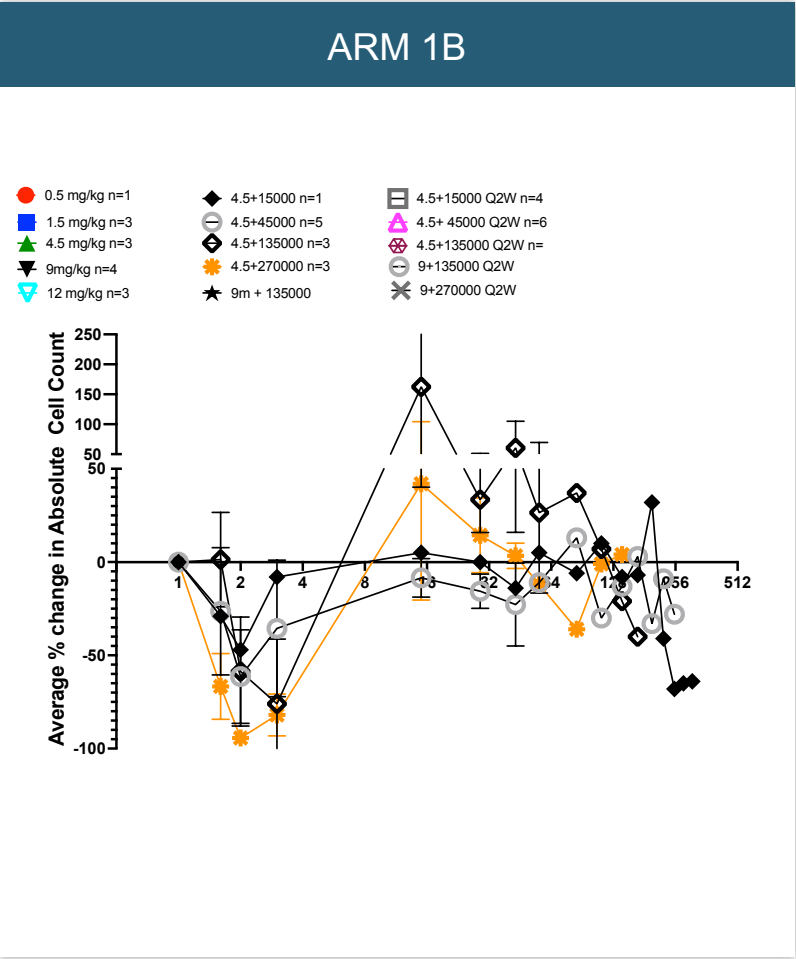
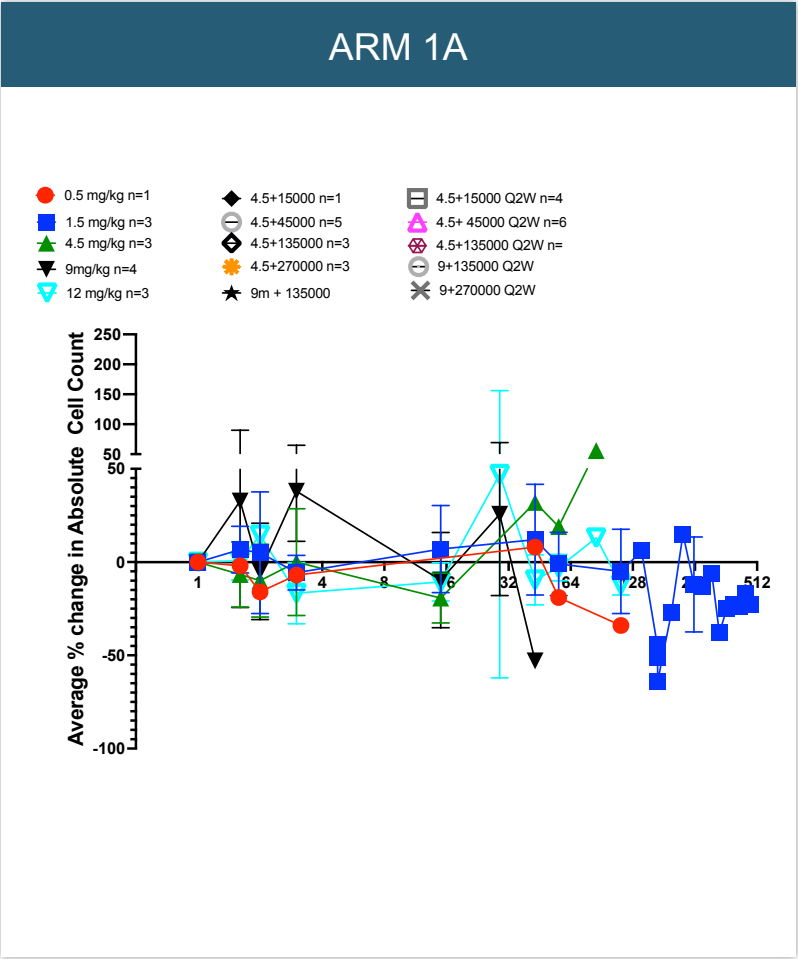
Pharmacodynamics: AU-007 Continues to Demonstrate Decrease in Tregs at Any Proleukin® IL-2 Dose Level

Completely unique profile in the IL-2 therapeutic class

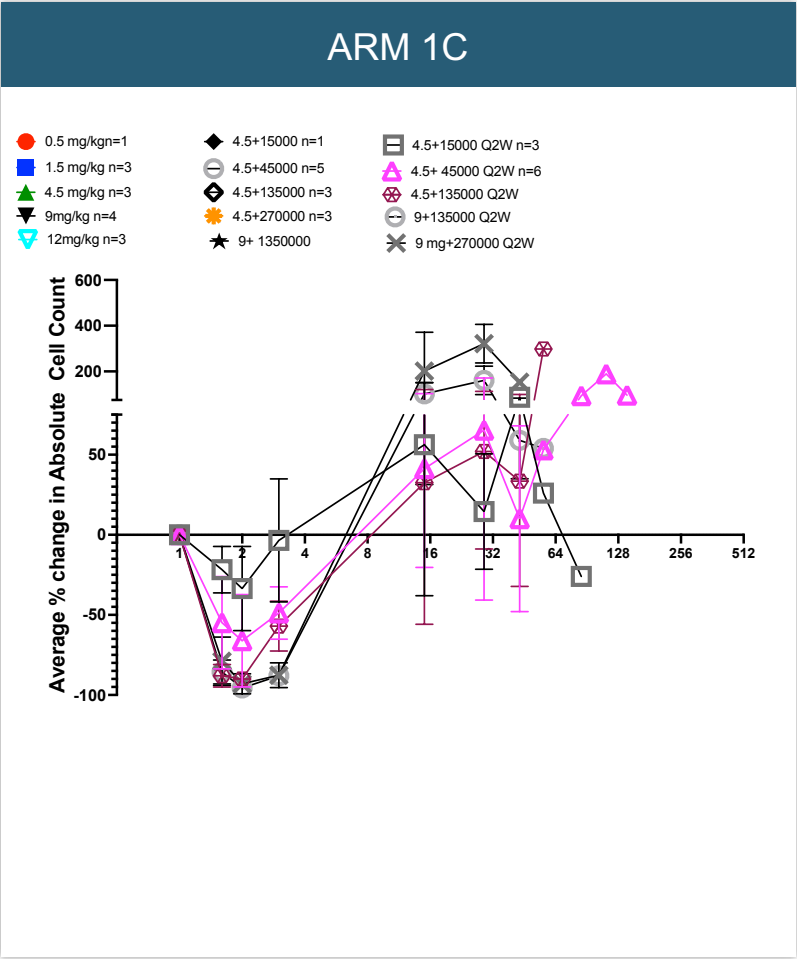
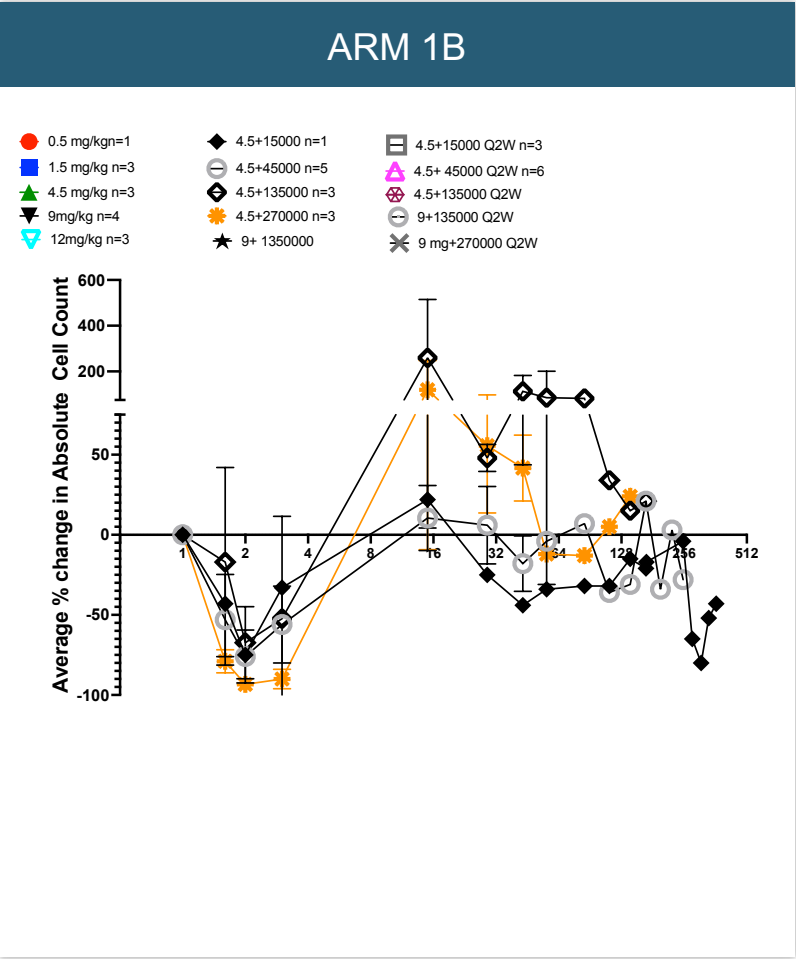
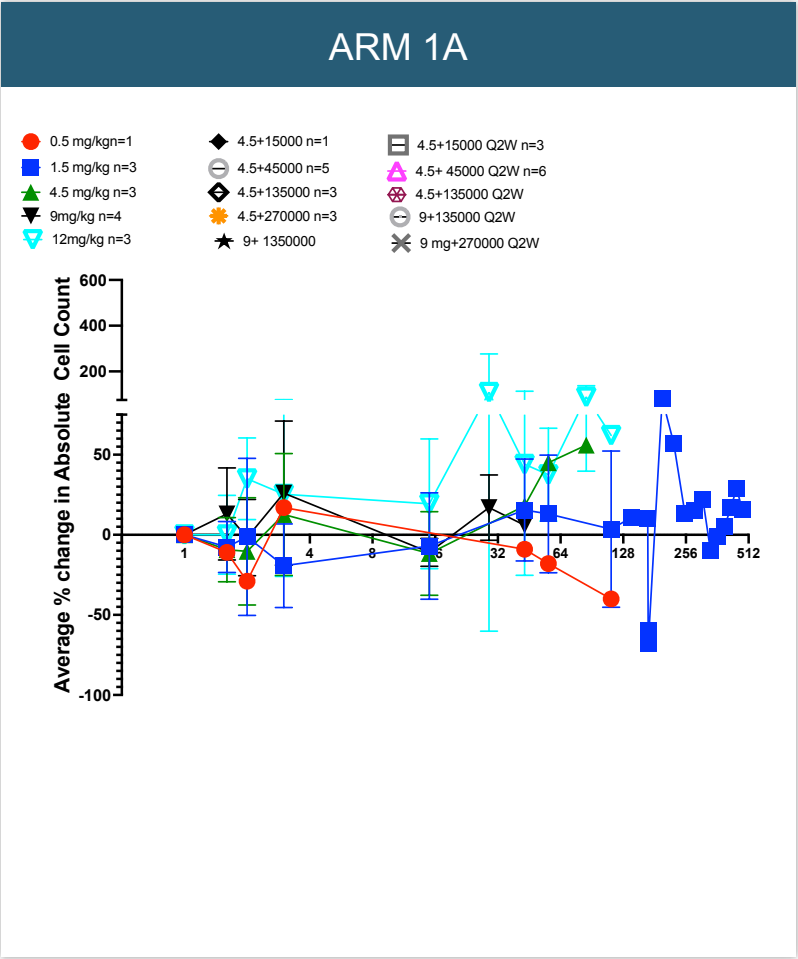


X axis = Days

AU-007 Dose Escalation: Peripheral CD8 Cell Increases by Study Arm

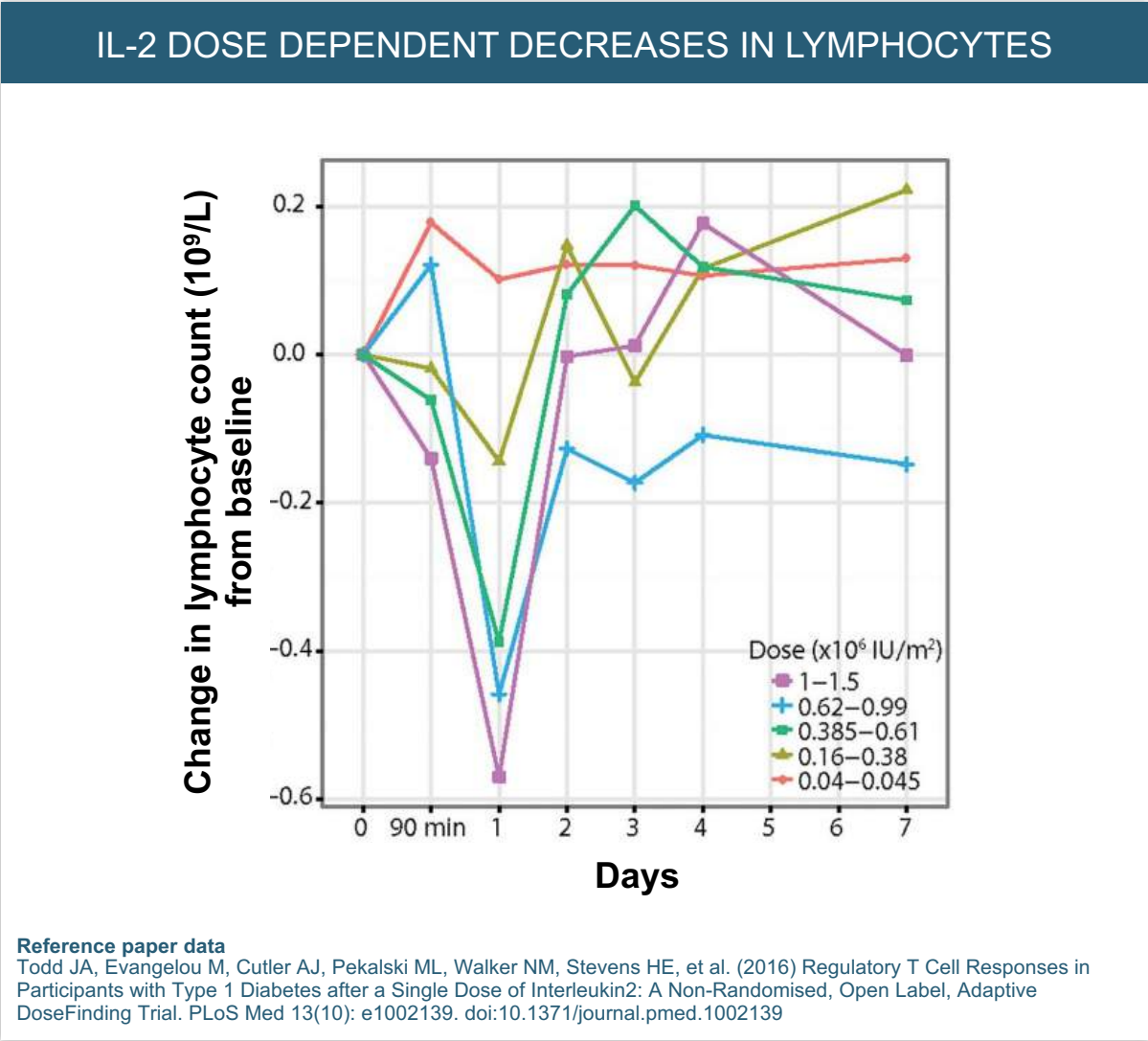


AU-007 Dose Escalation: Peripheral NK Cell Increases by Study Arm

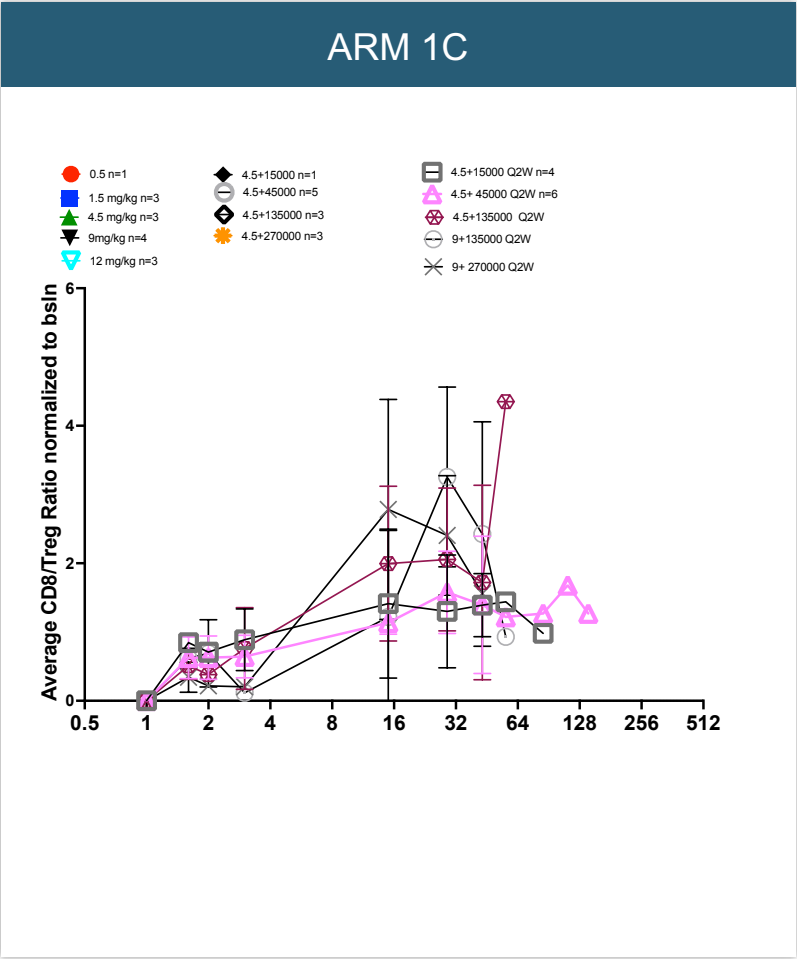
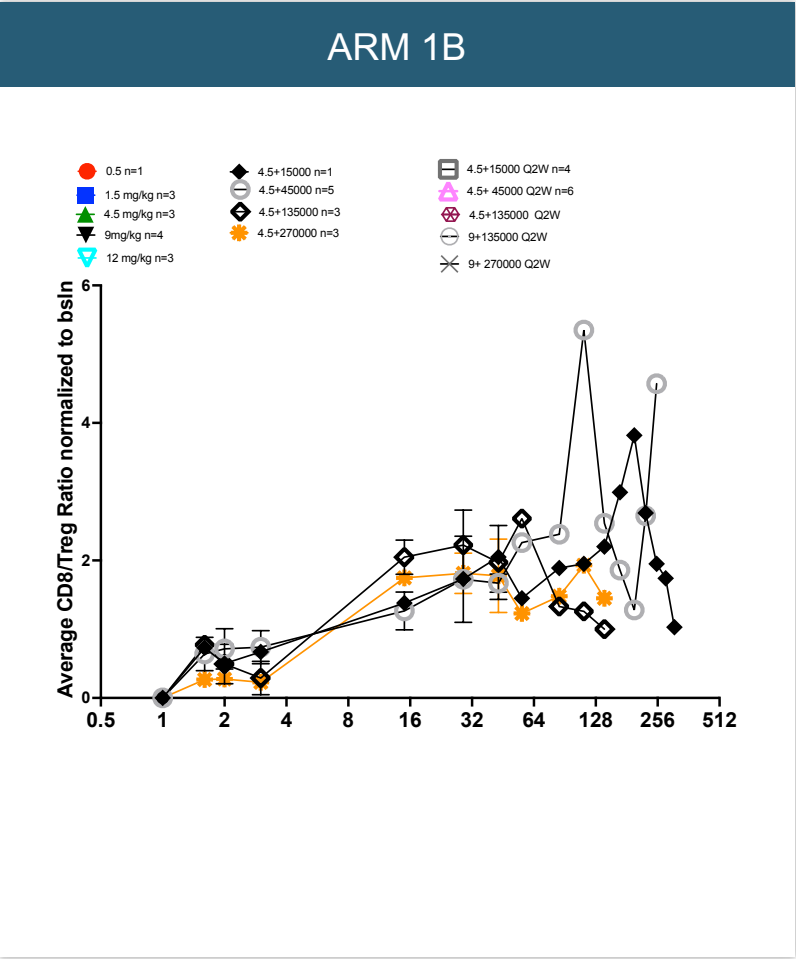
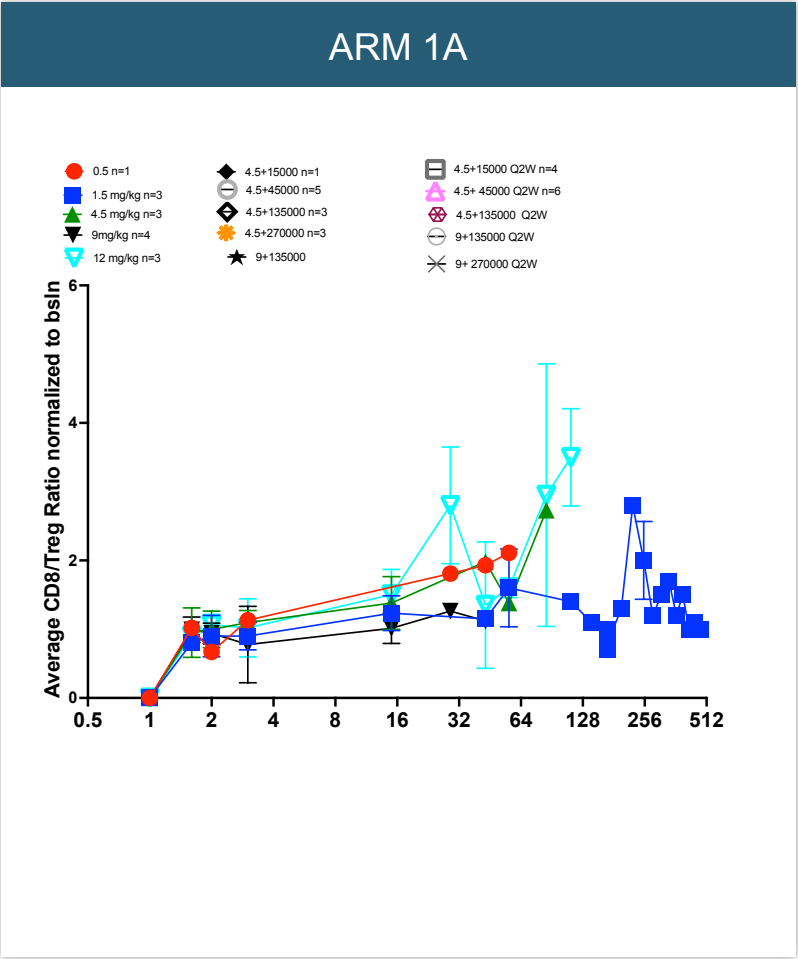


X axis = Days

Transient Lymphopenia Is a Known Phenomenon for Patients Receiving Proleukin®, and Likely Represents Trafficking of Lymphocytes From Vasculature Into Tissue

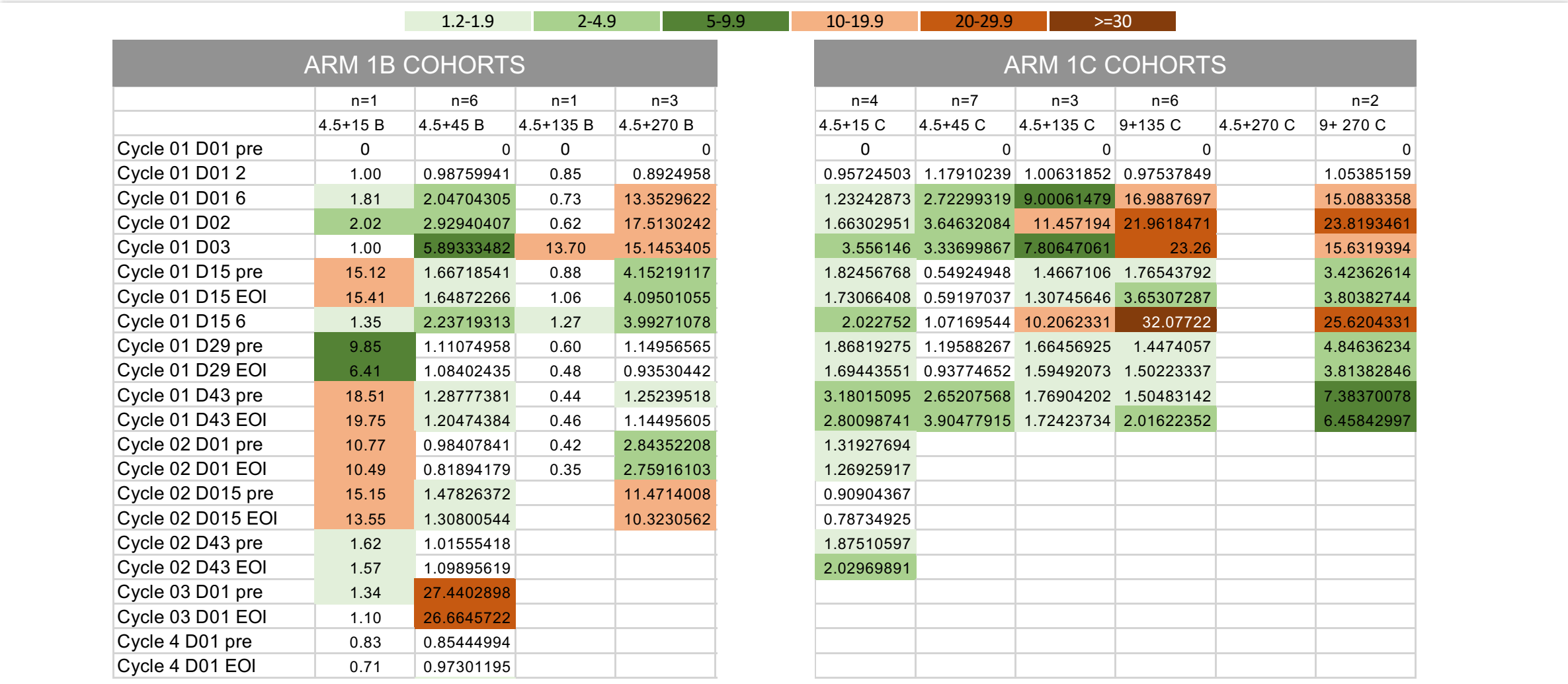


AU-007 Dose Escalation: Strong Increase in CD8+/Treg Ratios, Distinct in the IL-2 Class



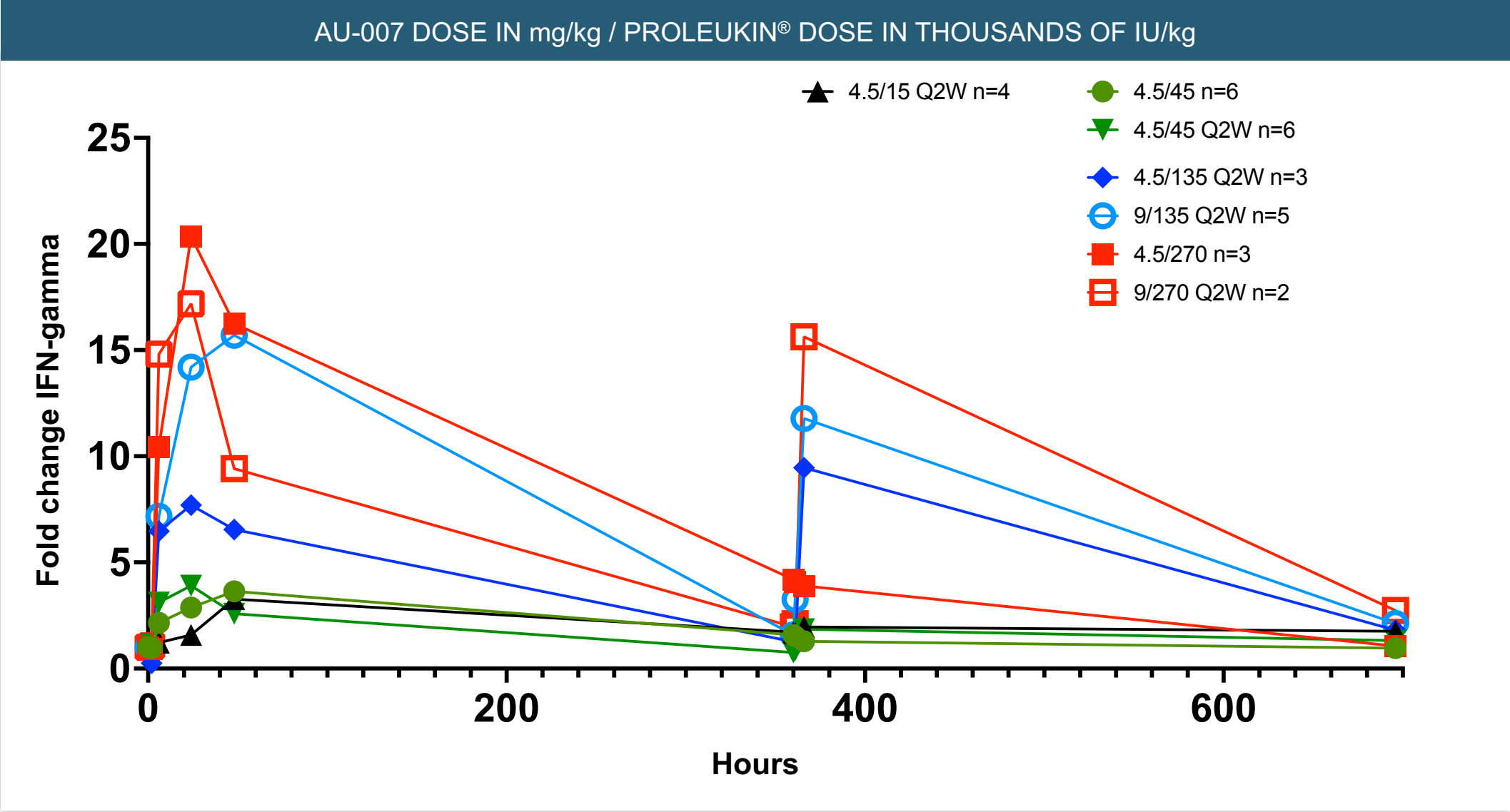
X axis = Days

AU-007 Dose Escalation: Fold Change in the Expression of IFN-γ Seen With 1B (Single) or 1C (Every Two Weeks) Dose Schedule of Proleukin®

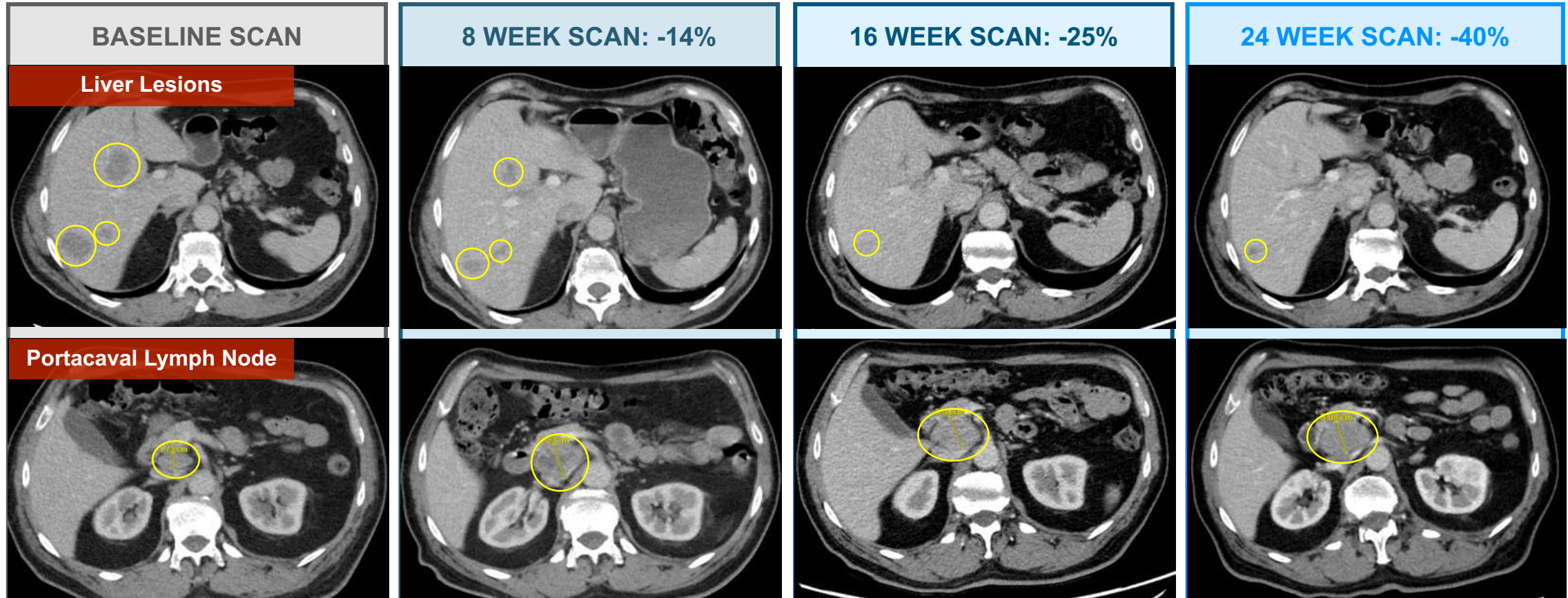


A heat map of the change from baseline in the circulating levels of IFN-γ. Light green represents a 0.2- to 1.9-fold change, mid-green a 2- to 4.9-fold change, dark green a 5- to 9.9-fold change, light red a 10- to 19.9-fold change, mid-red a 20- to 29.9-fold change and dark red a >=30-fold change. These preliminary results demonstrate that the longer a patient receives AU-007, the more likely the patient is to have increases in circulating IFN-γ. This is consistent with the observations in circulating cell populations, particularly Treg and NK cells. The addition of Proleukin® in the presence of AU-007 consistently increases IFN-γ in the peripheral circulation.

Average Fold Change in IFN- γ From Dose Escalation Cohorts With 1B (Single) or 1C (Every Two Weeks) Dose Schedule of Proleukin[®]

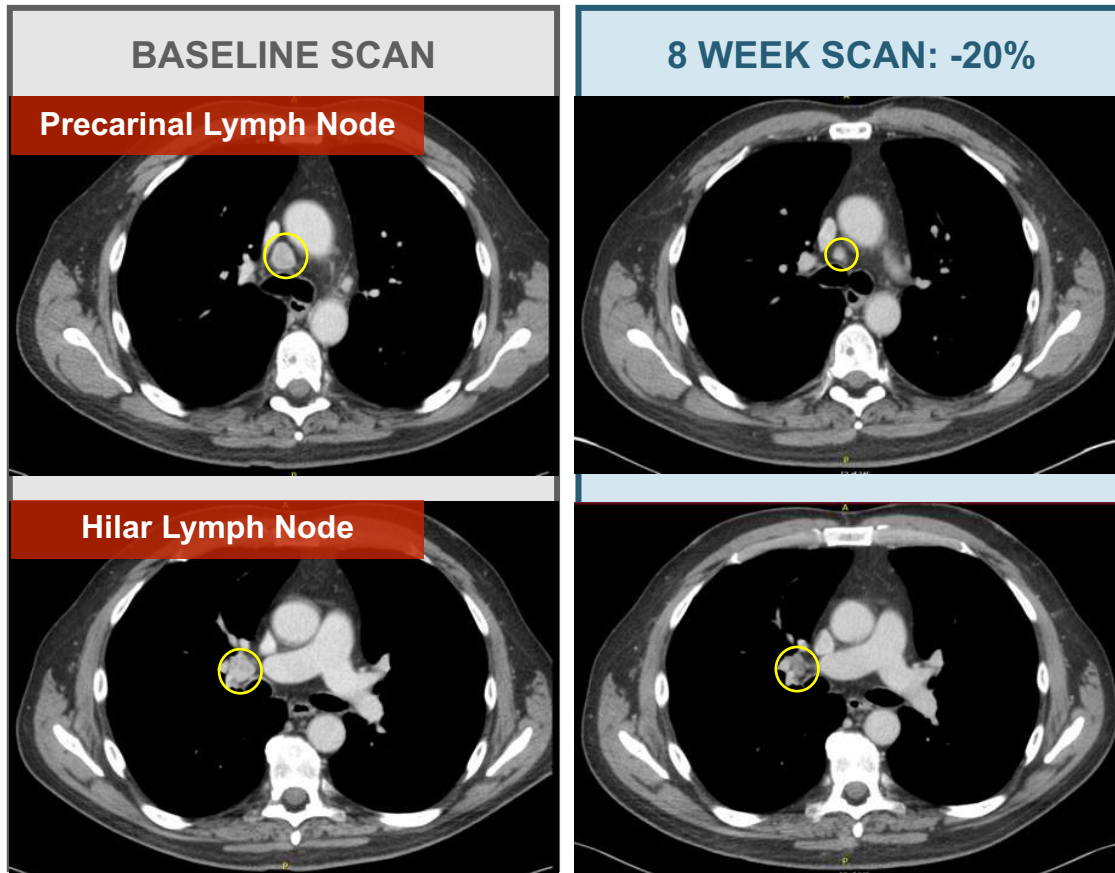


SITC 2023: 40% Tumor Shrinkage in the Target Lesions of a Patient Whose Melanoma Progressed Through Prior Anti-PD-1 + CTLA4 Therapy



- 62-year-old man with progression in the liver, December 2022
- February 2023, initial Q2W AU-007 (4.5 mg/kg) dose + one (and only) 15K IU/kg Proleukin® dose administered
- Initial portacaval LN growth with necrotic center followed by stabilization may represent pseudoprogression

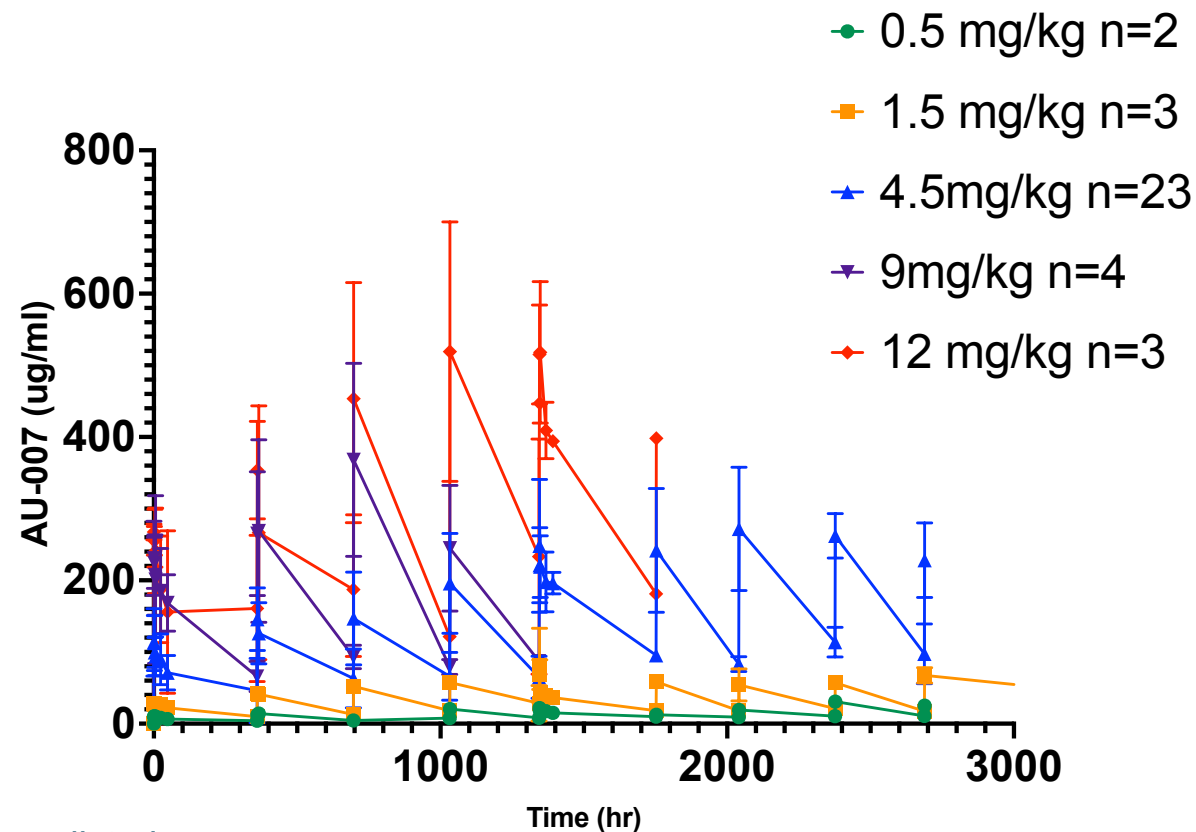
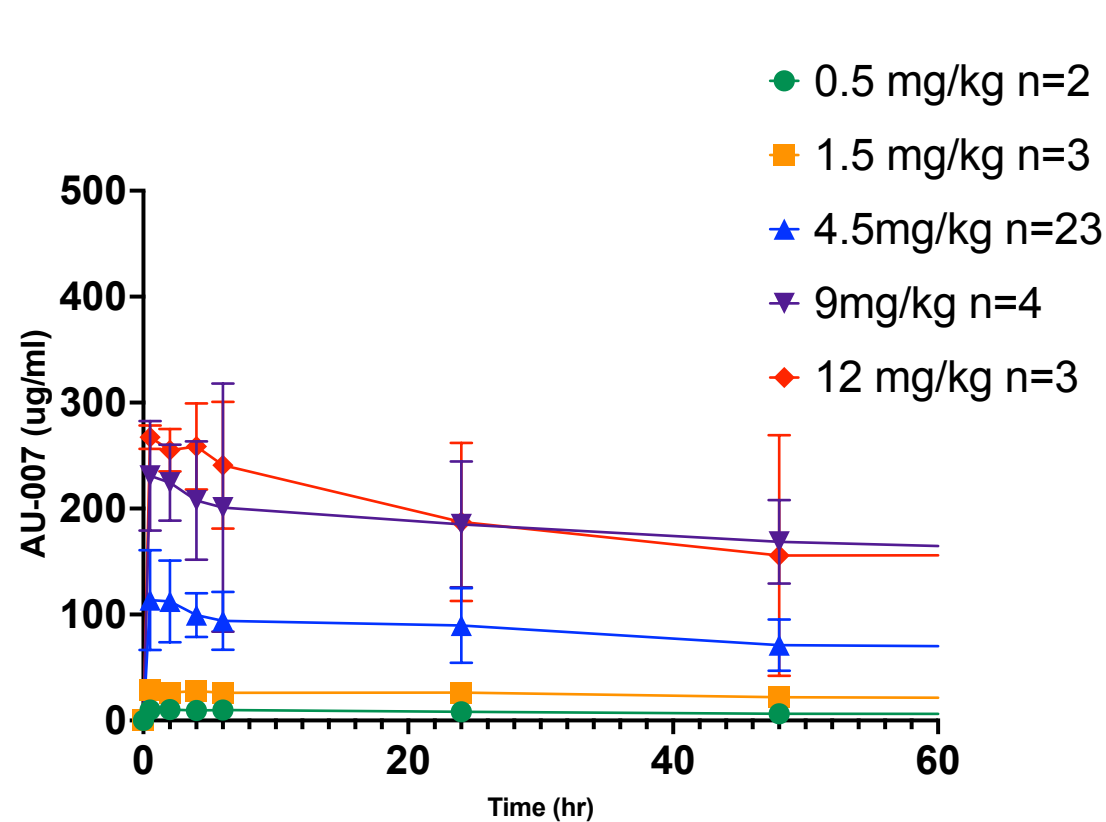
SITC 2023: 20% Tumor Shrinkage in First 8 Weeks in the Target Lesions of a Patient Whose RCC Progressed Through Prior Anti-PD-1 Therapy



- 68-year-old man progressed on anti-PD-1 treatment June 2022
- July 2023, initial AU-007 (4.5 mg/kg) + 15K IU/kg Q2W Proleukin®
- The primary renal cancer remains *in situ* and was stable

AU-007 PK Data Demonstrates IgG1 Therapeutic Characteristics

PK data continues to demonstrate dose proportionality and accumulation; half-life > 14 days



Cmax and step close to predicted

| Dose | Est Cmax (70kg) | Step | Actual Cmax (ug/ml) | Calculated Step |
|------|-----------------|------|---------------------|-----------------|
| 0.5 | 14 ug/ml | | 10.8+/-16 | |
| 1.5 | 42 ug/ml | 3 | 29.6+/-13 | 2.75 |
| 4.5 | 126 ug/ml | 3 | 110+/-15 | 3.7 |
| 9 | 252 ug/ml | 2 | 255+/-21 | 2.3 |
| 12 | 336 ug/ml | 1.3 | 282+/-9 | 1.5 |

AU-007 PK and IL-2 Coverage (For Binding and Redirecting IL-2 to Dimeric Receptors on Effector Cells)

| AU-007 Dose mg/kg | Time Point | Serum AU-007 ug/ml | Serum IL-2 Coverage pM | Coverage of Highest IL-2 Dose (Proleukin® 270K IU/kg) |
|----------------------|----------------------|-----------------------|---------------------------|--|
| 0.5 | Initial Peak | 11 | 150685 | 377 x |
| | Initial Trough | 4.3 | 58904 | 147 x |
| | Steady State Average | 12 | 164384 | 411 x |
| 1.5 | Initial Peak | 30 | 410959 | 1027 x |
| | Initial Trough | 9.8 | 134247 | 336 x |
| | Steady State Average | 32 | 438356 | 1096 x |
| 4.5 | Initial Peak | 110 | 1506849 | 3767 x |
| | 50 Hours | 85 | 1164384 | 2911 x |
| | Steady State Average | 94 | 1287671 | 3219 x |
| 9 | Initial Peak | 255 | 3493151 | 8733 x |
| | 50 Hours | 169 | 2315068 | 5788 x |
| | Steady State Average | 192 | 2630137 | 6575 x |
| 12 | Initial Peak | 282 | 3863014 | 9658 x |
| | 50 Hours | 184 | 2520548 | 6301 x |
| | Steady State Average | 256 | 3506849 | 8767 x |

AU-007 Has Unique Potential to Solve the Challenges of IL-2 by Acting as a Router for IL-2, Redirecting It Toward Effector Cells

Computationally designed, epitope-specific monoclonal antibody therapeutics directing native IL-2 cytokine to specific target cells (drives expansion of effector T cells and downregulation of Tregs)



Potential for higher efficacy, based on unique MOA
Only agent in class that **lowers** Tregs



Potential for lower toxicity —
by blocking IL-2's binding to vascular endothelium



Unique antibody computationally designed by world-class machine learning



Known modality; a well-behaved antibody format with drug-like properties



AULOS

Positioning for Success

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**At Aulos,
our mission is
to extend and improve
the lives of patients
through innovative,
safe and effective
cancer immunotherapy**

Our Values



INGENUITY

We bring a spirit of ingenuity to what we do.



BALANCE

We are a balanced organization that pursues the best idea.



GROWTH

We are committed to grow individually and as a team.



HOPE

We aspire to provide hope to patients and their loved ones with novel therapy.



SUPPORT

We support each other and collaborate efficiently.

Accomplished, Experienced Leadership Team



Aron Knickerbocker
President and Chief
Executive Officer



Yanay Ofran
Chief Scientific
Officer



Jim Vasselli, M.D.
Chief Medical
Officer



Micah Pearlman
Chief Operating
Officer



Leo Redmond
Chief Financial
Officer



Tim Wyant
SVP and Head of
Early Development



Jenny Tang
Head of Clinical
Operations



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AU-007 Value-Driven Milestones

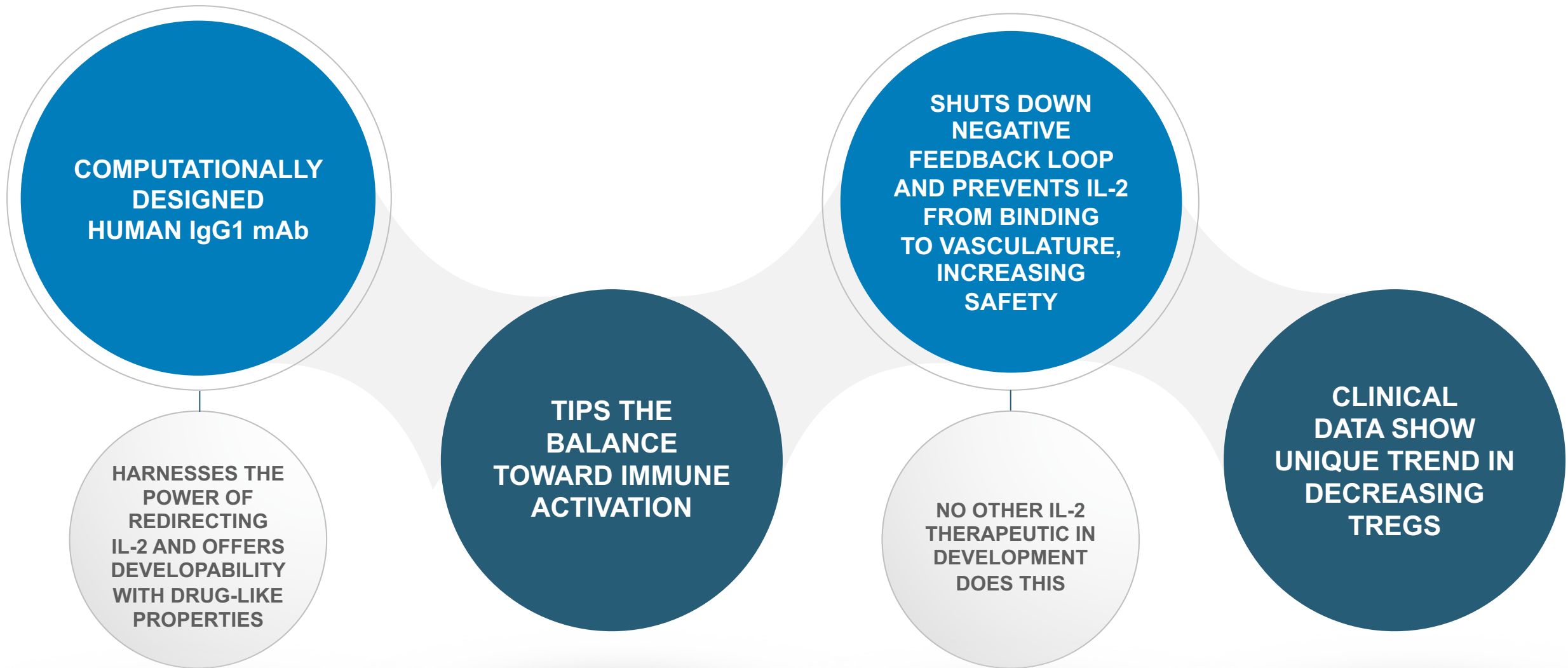
| | |
|---|-----------|
| ✓ Initiated Dosing in Phase 1 in Australia | 2Q 2022 |
| ✓ Received FDA Clearance of IND Application | 4Q 2022 |
| ✓ Began Dosing Patients at US Clinical Sites | 1Q 2023 |
| ✓ Began Phase 2 Dosing in Expansion Cohorts in Melanoma and Renal Cell Carcinoma | 1H 2024 |
| Begin Phase 2 dosing in expansion cohorts in non-small cell lung cancer | 2H 2024 |
| Establish Phase 2 clinical proof of concept in melanoma and renal cell carcinoma | 2H 2024 |
| Establish Phase 2 clinical proof of concept in non-small cell lung cancer | 1H 2025 |
| Seek Breakthrough Designation, begin pivotal trial(s) in melanoma, RCC and/or NSCLC | 2025 |
| Initiate Phase 2 trials in additional indications, as warranted | 2025 |
| Submit marketing approval applications globally | 2027-2028 |
| First commercial sales | 2027-2029 |



A safe and broadly applicable IL-2 regimen has been a “holy grail” of cancer immunotherapy.

If achieved, AU-007 would likely represent the next multi-indication blockbuster cancer immunotherapy – a pipeline in a product.

AU-007: A Compelling New Approach for Harnessing IL-2 to Fight Cancer

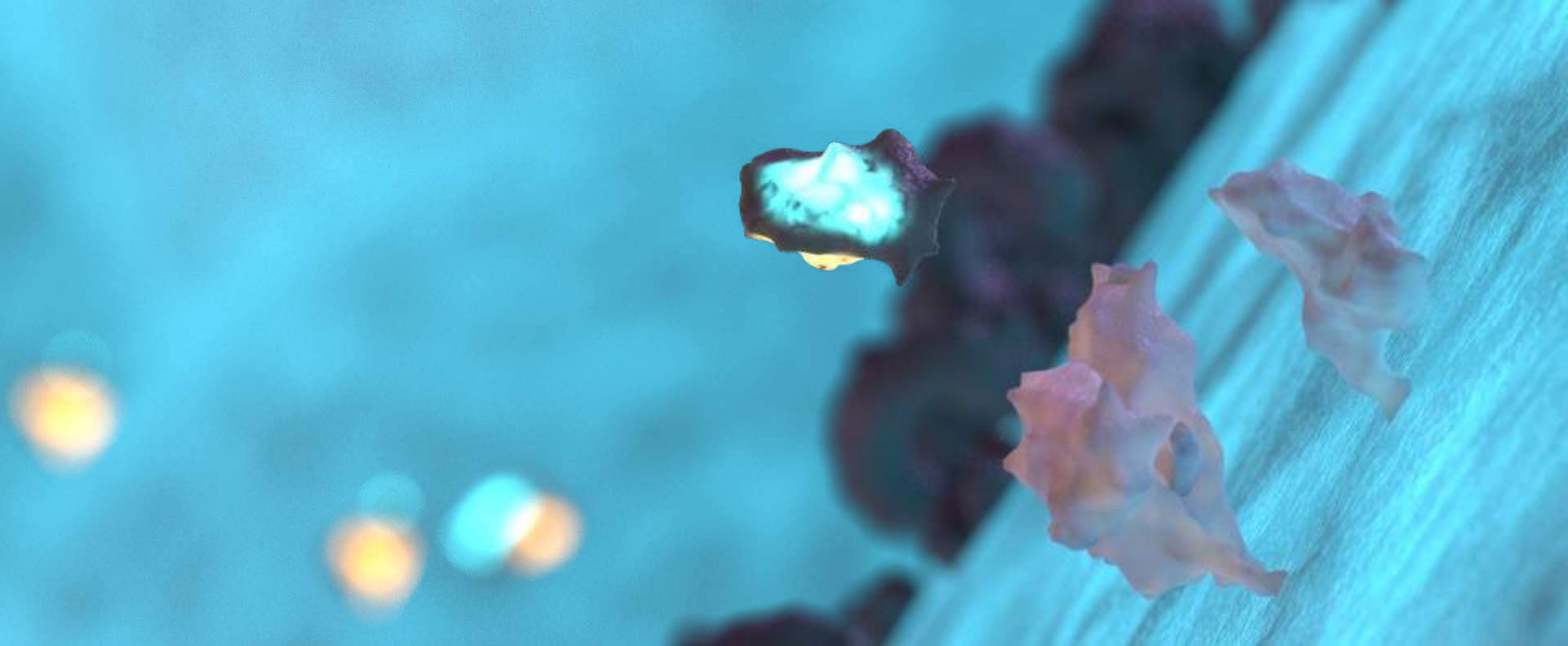


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THANK YOU

www.aulosbio.com



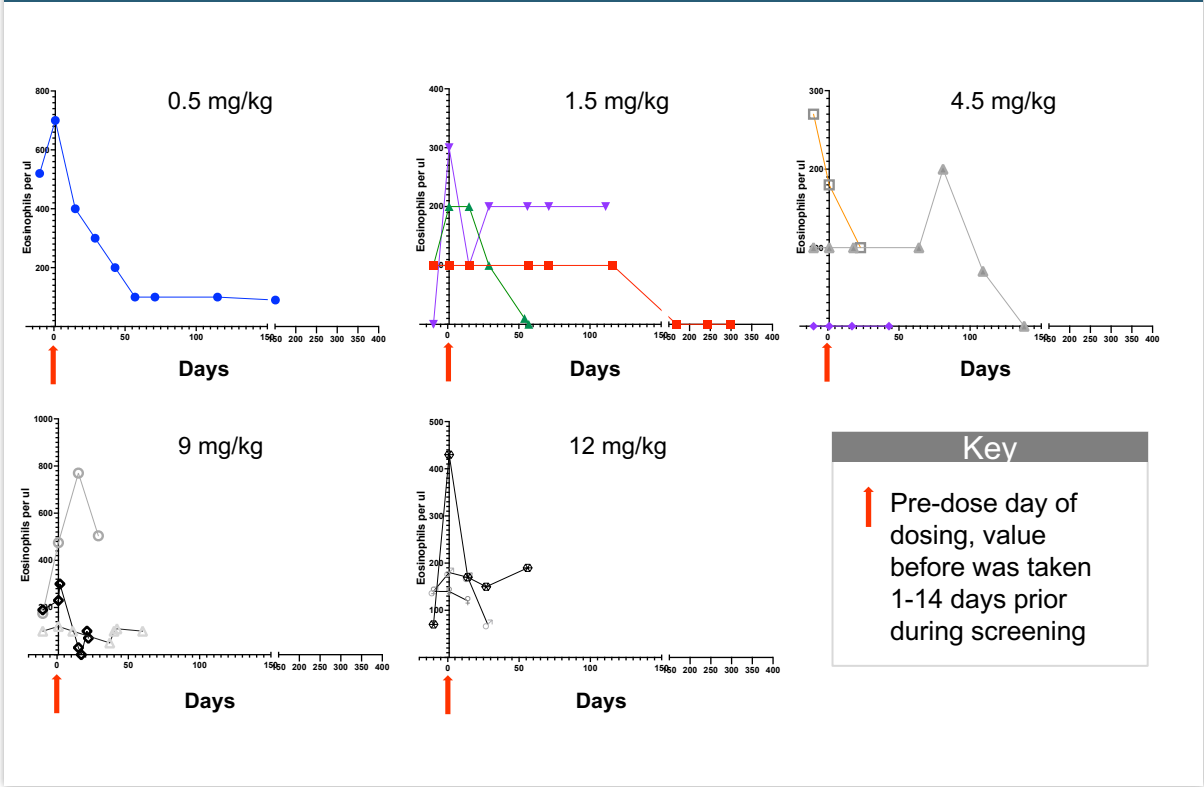


APPENDIX

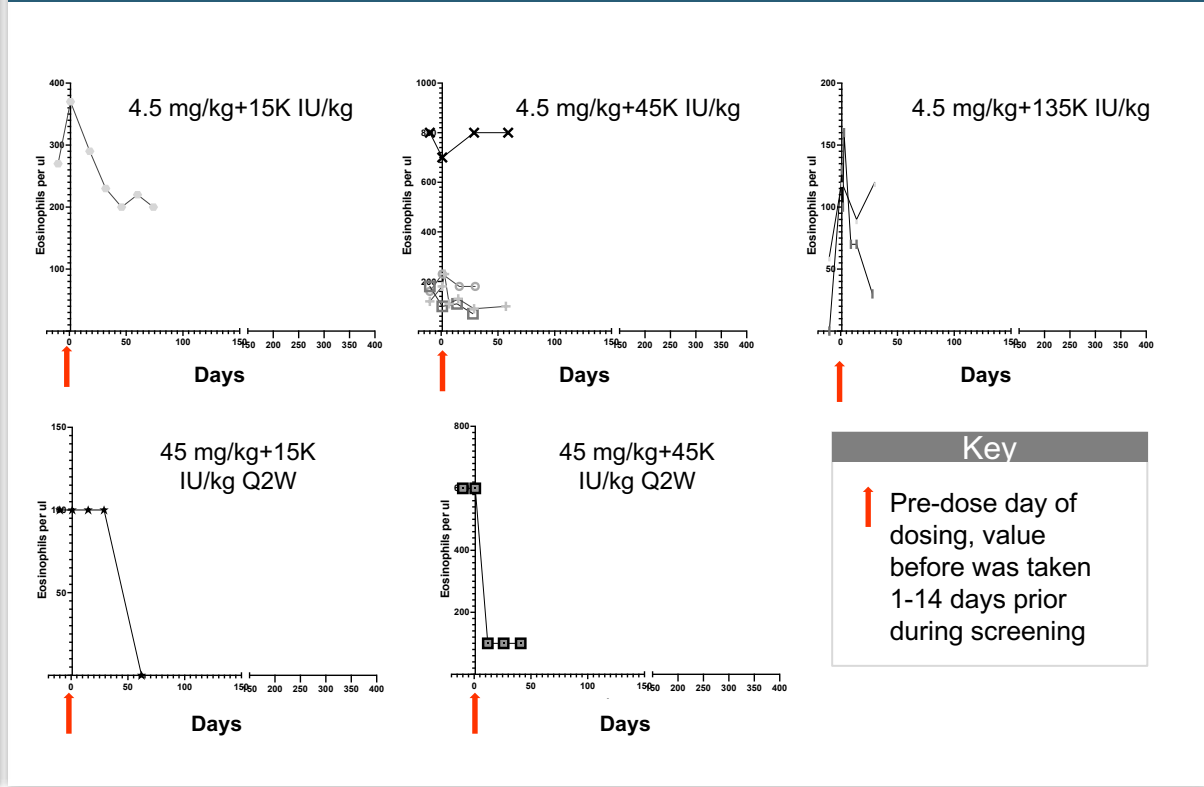
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AU-007 Dose Escalation Study: Change in Eosinophils (Cells That Also Express the Trimeric IL-2 Receptor That Contains CD25)

INDIVIDUAL PERIPHERAL BLOOD EOSINOPHIL COUNTS
IN AU-007-ONLY COHORTS

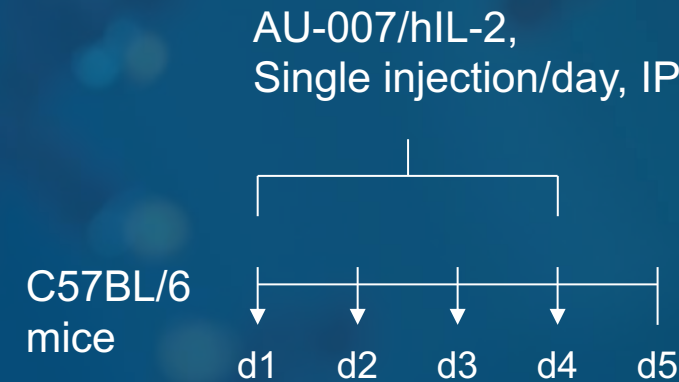
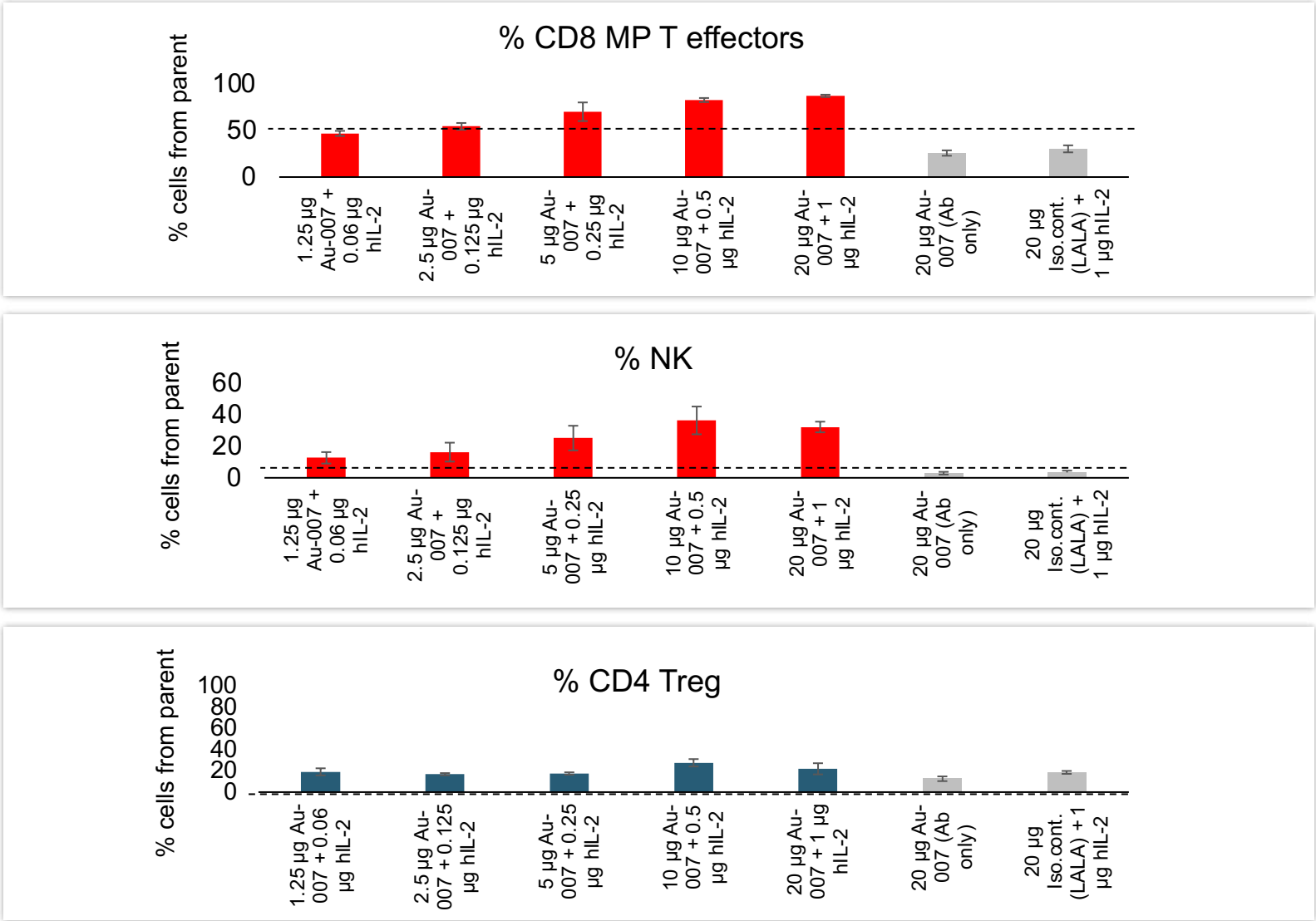


INDIVIDUAL PERIPHERAL BLOOD EOSINOPHIL COUNTS
IN AU-007+PROLEUKIN® COHORTS



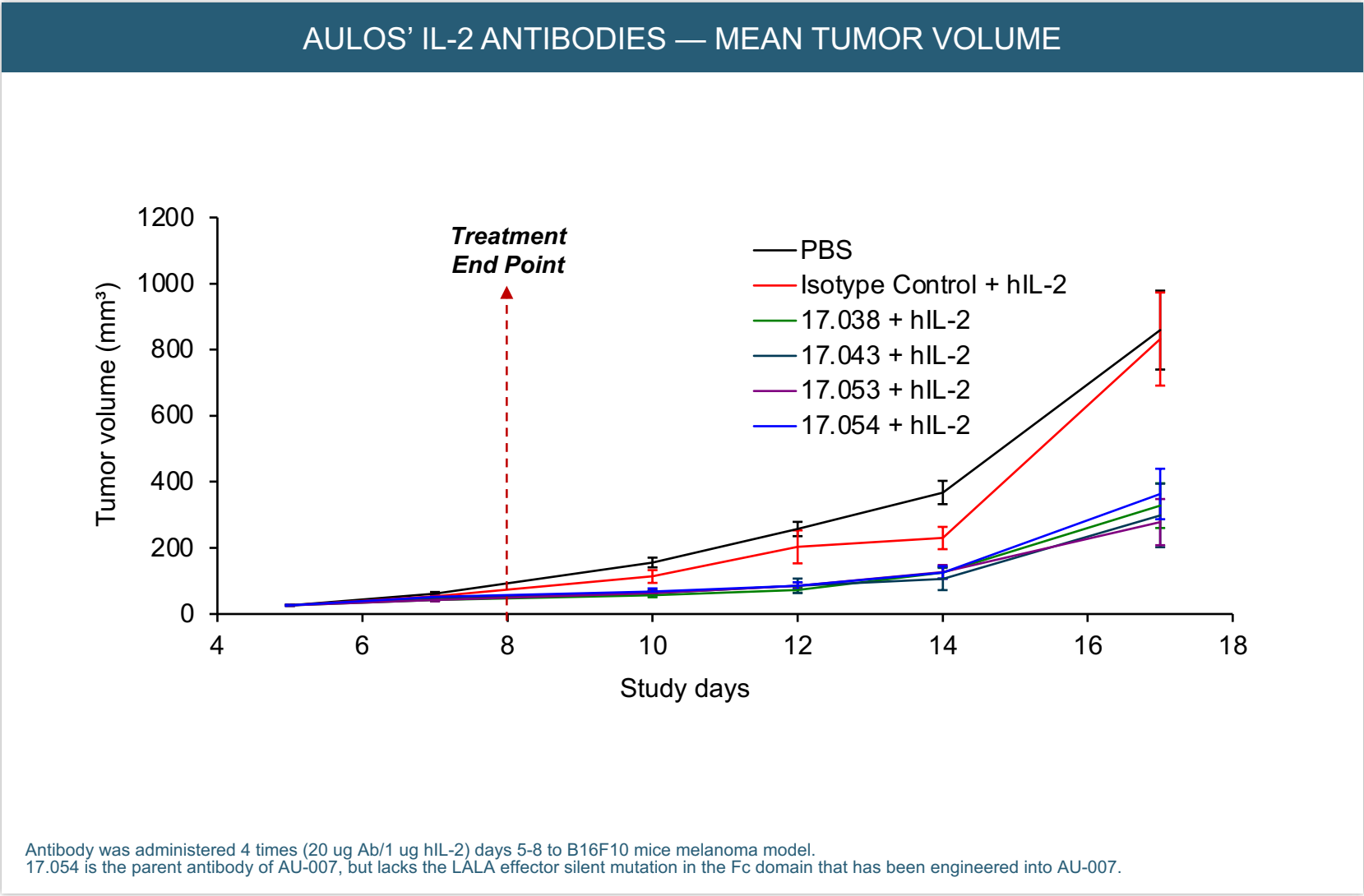
Changes over time in the circulating number of eosinophils. Panel A are the cohorts receiving only AU-007 monotherapy and panel B are cohorts receiving AU-007 with at least 1 dose of Proleukin®. All but one patient in the AU-007 monotherapy and AU-007 with Proleukin® arms demonstrated a decrease or no change in the circulating levels of eosinophils. A patient in the 9 mg/kg cohort had severe seasonal allergies requiring treatment during time on AU-007 treatment and is consistent with a history of being treated for seasonal allergies. The rise in eosinophils was attributed to the allergy reaction. All patients given AU-007 with Proleukin® showed stable or a decrease in circulating eosinophils. This is consistent with the mechanism of action of AU-007 preventing IL-2 from interacting with the IL-2 trimeric receptor on eosinophils.

In Mice, AU-007 Promotes Dose-Dependent Expansion and Activation of Effector T and NK but Not Treg Cells *In Vivo*

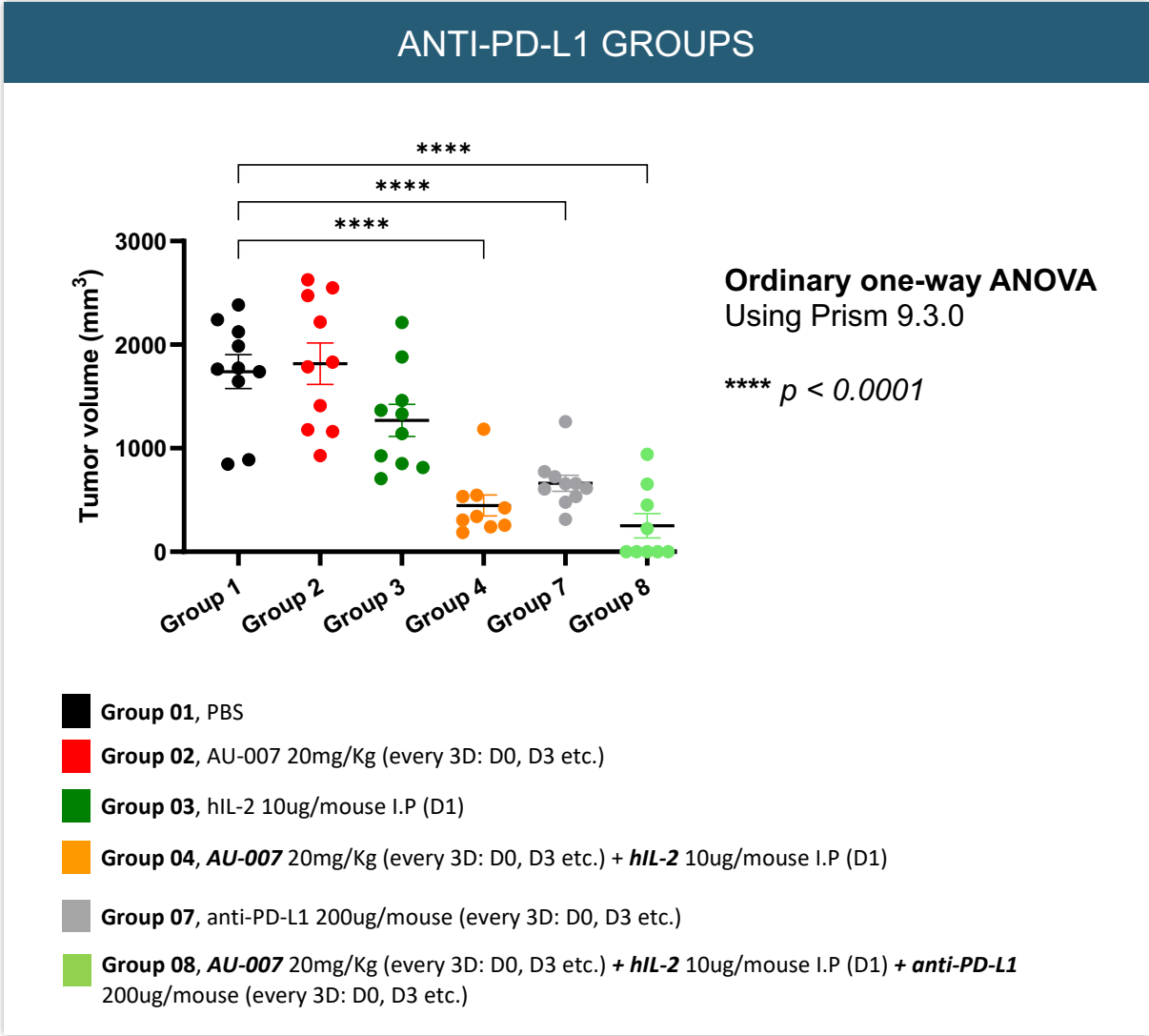
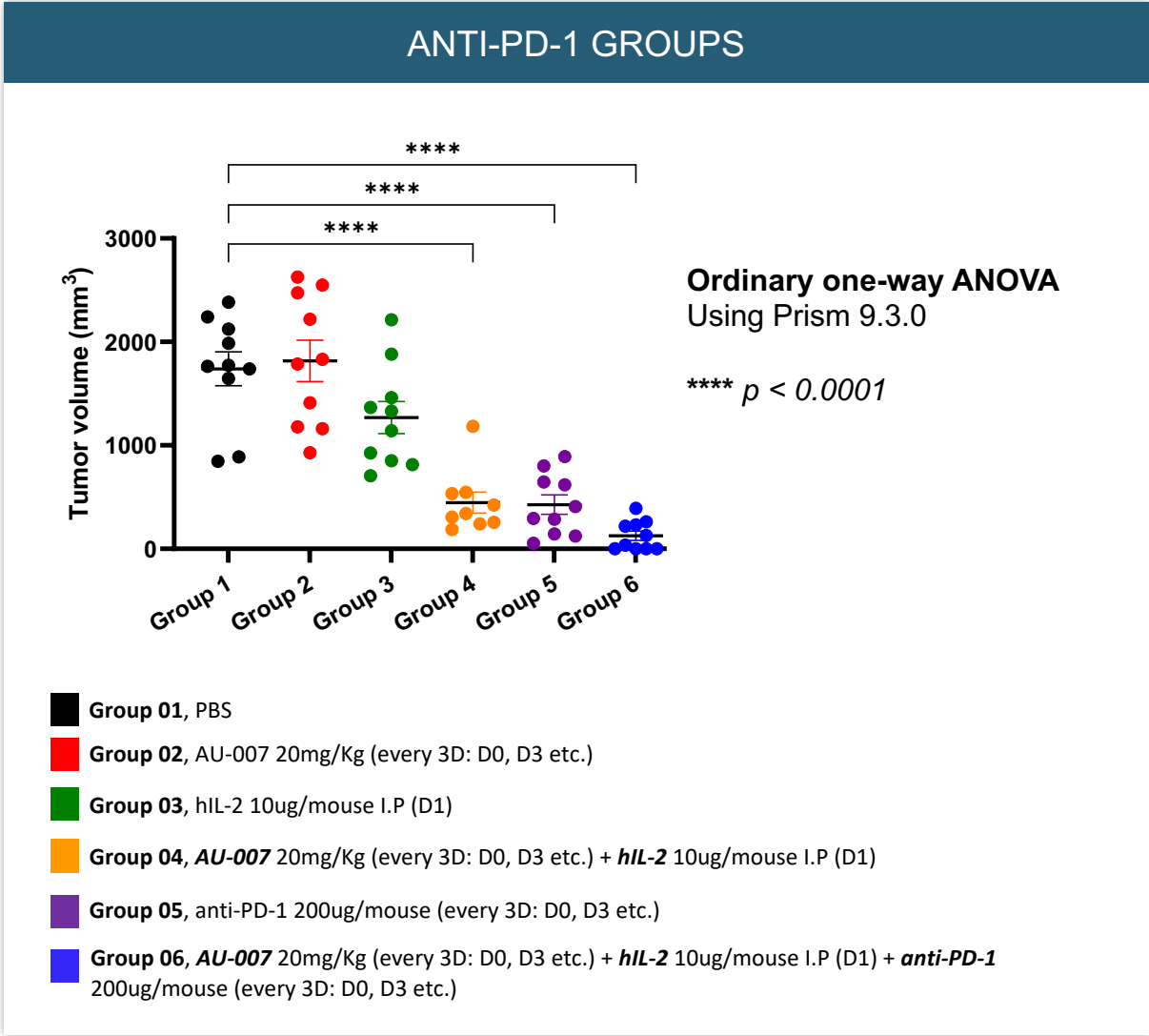


- Splenocytes isolation
- Flow cytometry

Aulos' IL-2 mAbs Show Inhibition of Tumor Growth in Mouse Syngeneic Tumor Model Resistant to Checkpoint Inhibitors (B16F10 Melanoma)

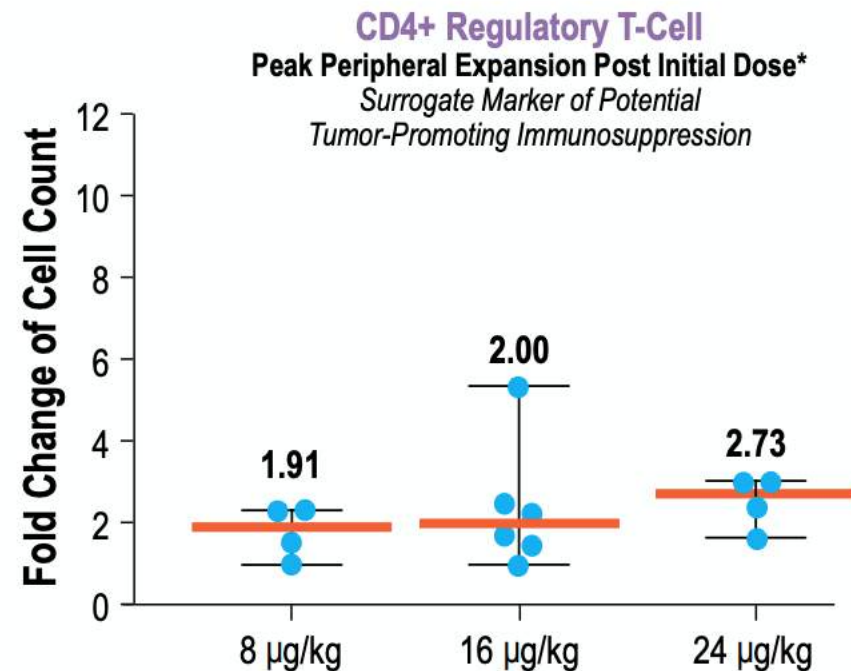


AU-007 Induces Regressions and Some Tumor Eradications in MC38 Colon Cancer Model in Wild-Type Mice When Combined With Anti-PD-(L)1



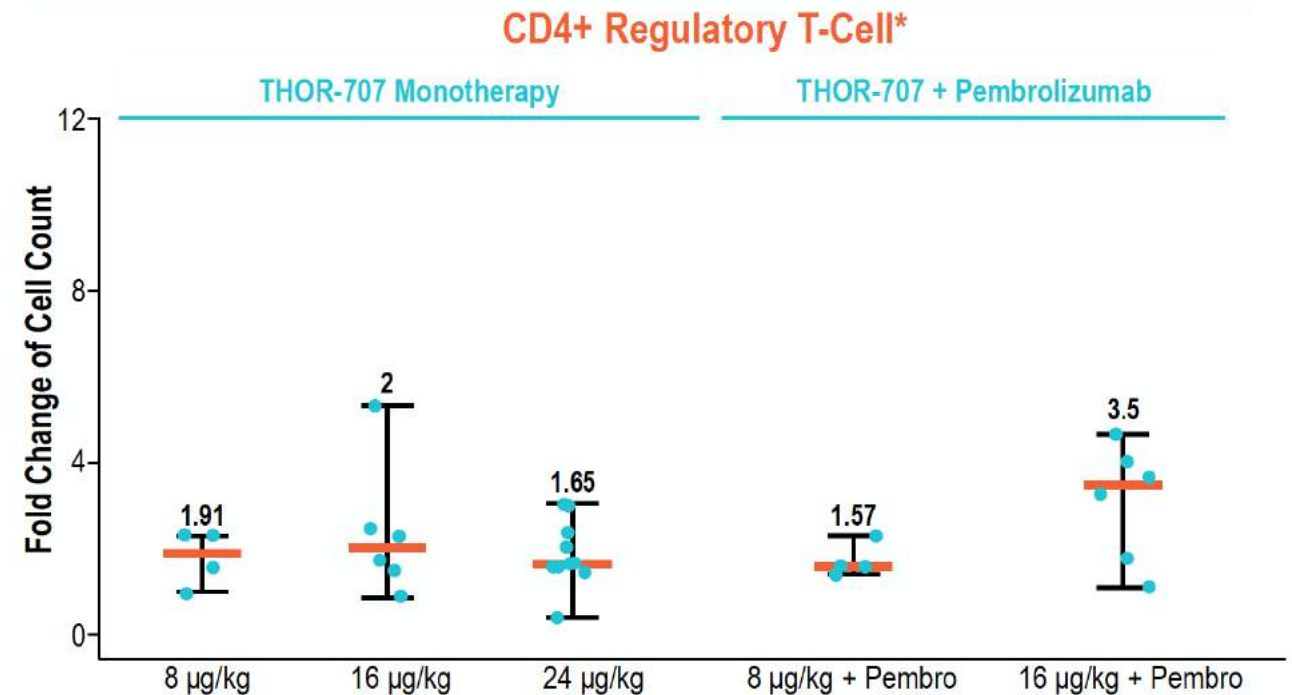
Clinical Evidence of the Negative Feedback Loop in Action: THOR-707 Increases Peripheral Blood Tregs ~2-3x After First Dose

ENA Symposium 2020,
Phase 1/2 Dose Escalation



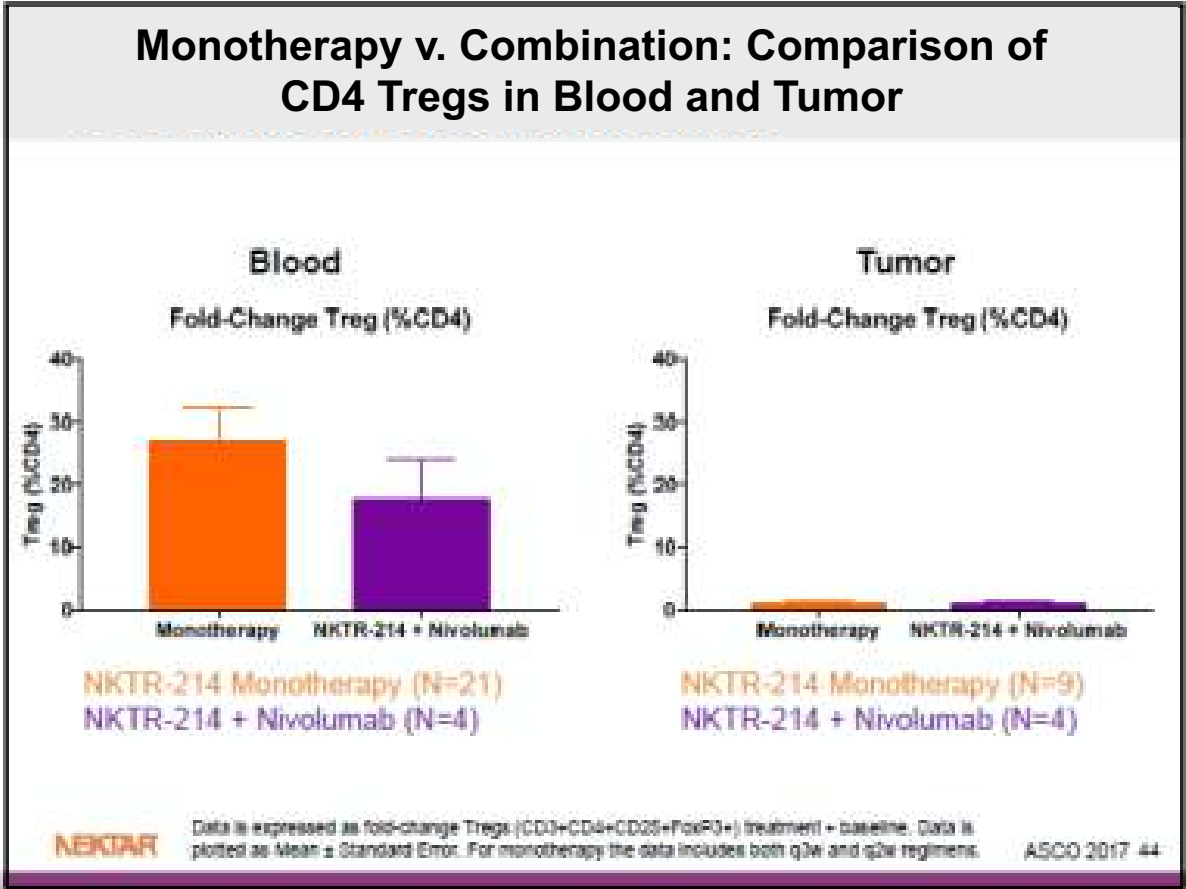
AACR 2021, Phase 1/2

Pharmacodynamic Markers of Not-Alpha Selectivity



Second *In Vivo* Proof of Negative Feedback Loop in Action: Bempegaldesleukin

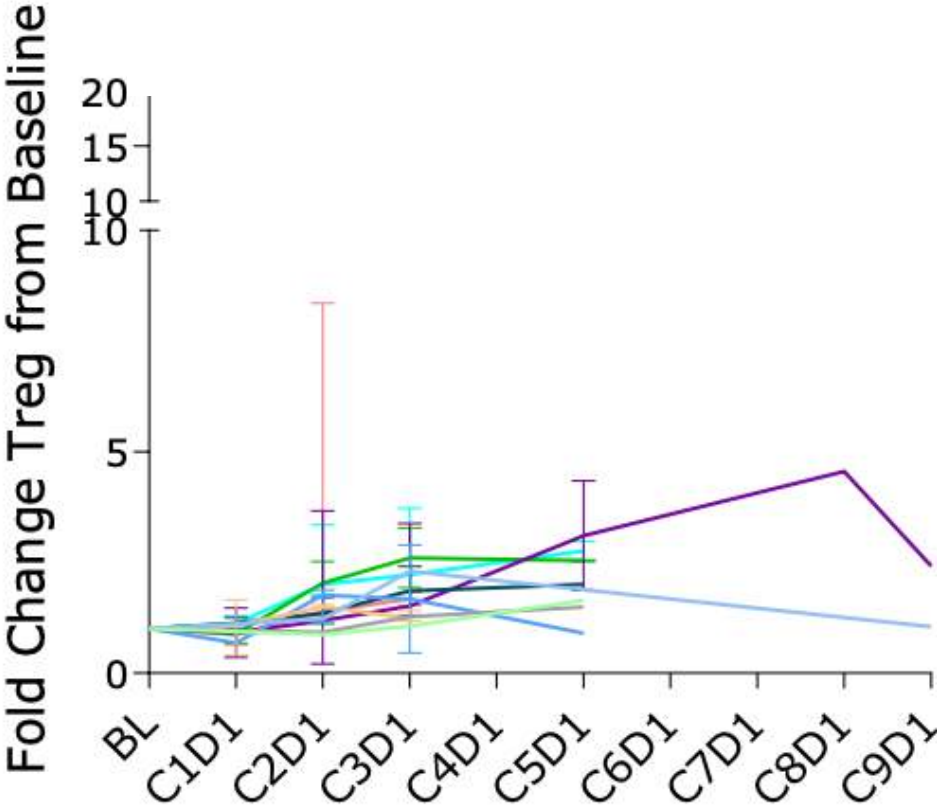
27-fold increase in peripheral Tregs with Bempeg



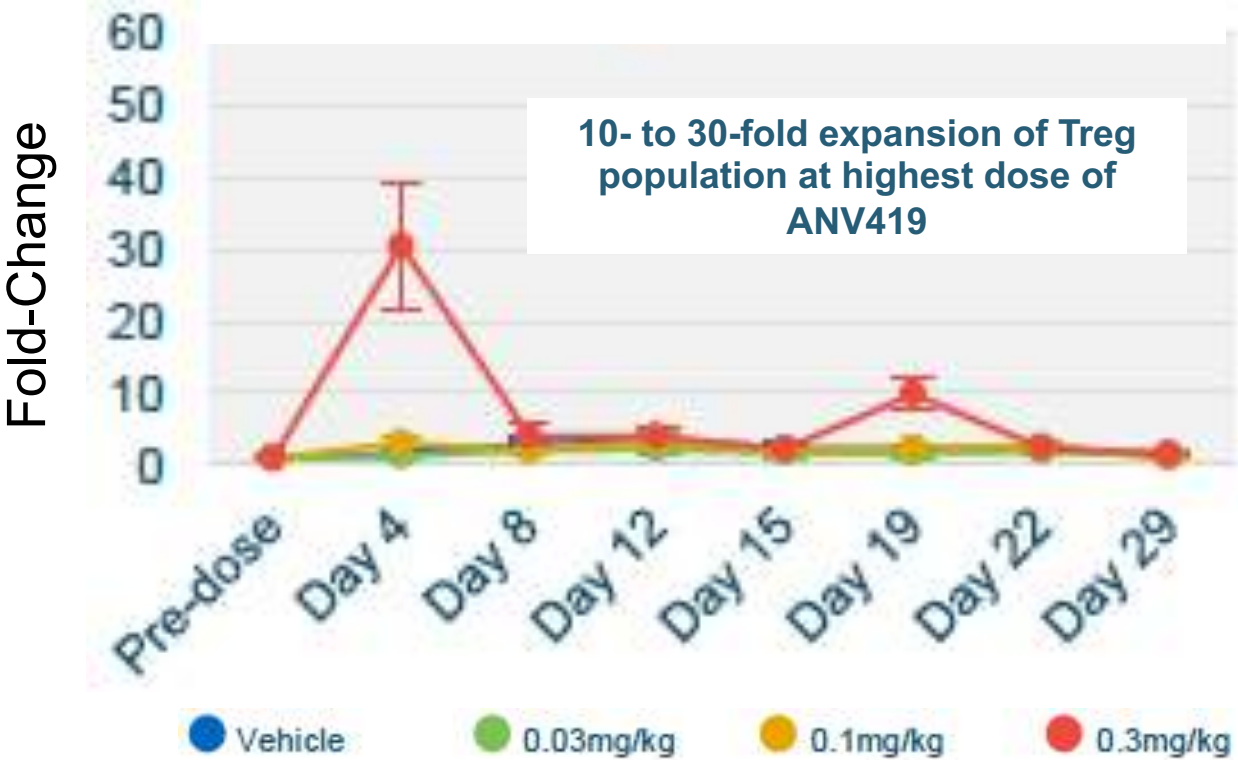
Substantial expansion of Tregs and low delivered doses of IL-2 likely accounts for poor clinical data observed to date with pegylated IL-2 constructs, and the failure in multiple Phase 3 trials

Third *In Vivo* Proof of Negative Feedback Loop in Action: Anaveon's ANV419

Treg fold change at each cycle in patients from Phase 1 clinical trial

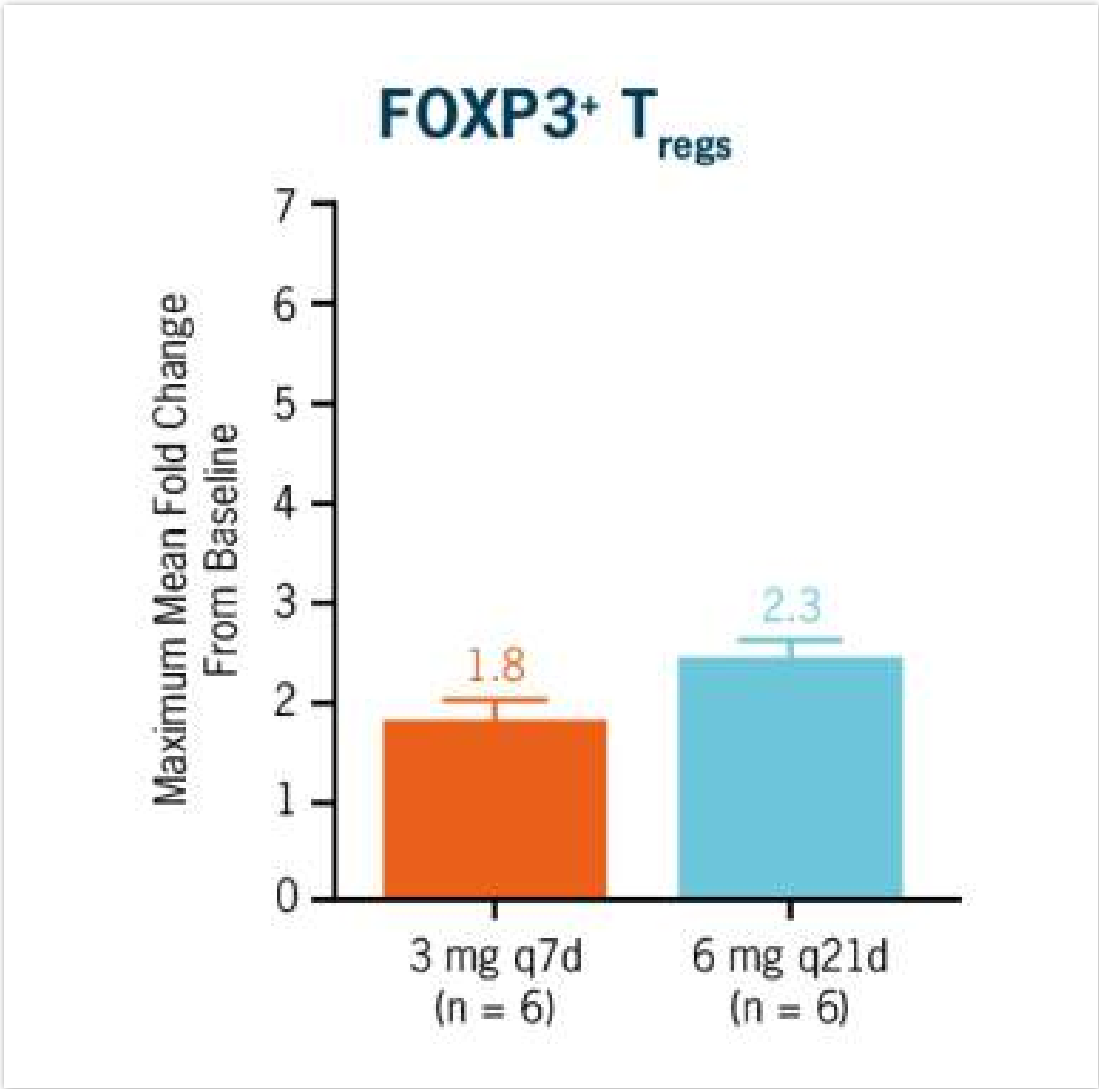


Treg population fold change following dosing on Days 1 and 15 in cynomolgus monkeys

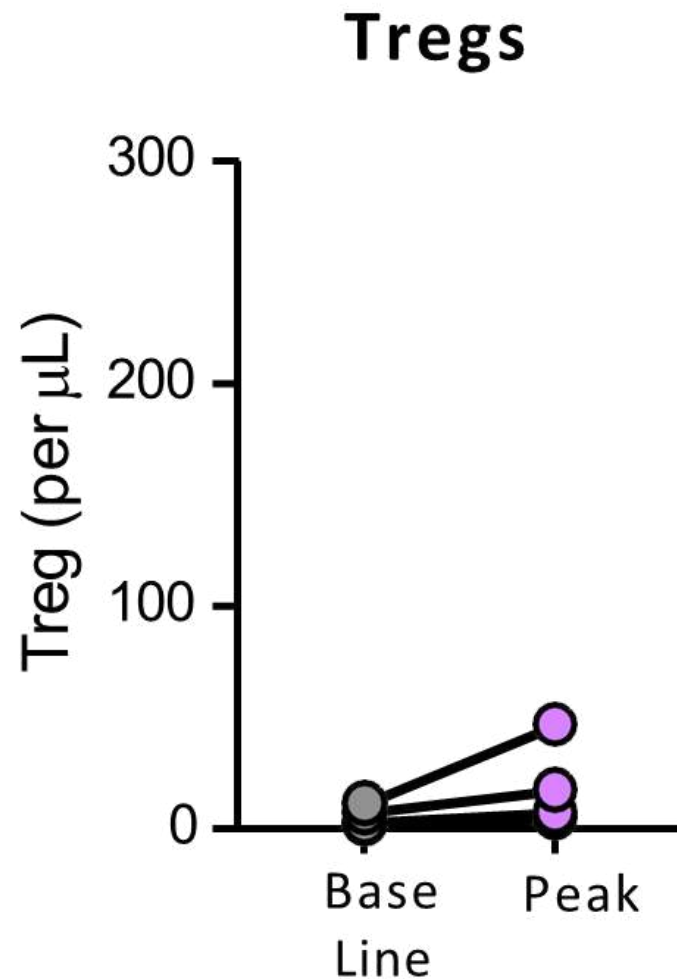


Half-life of ANV419 in cynomolgus monkeys is ~24 hours
Half-life of AU-007 in cynomolgus monkeys is ~15 days

Fourth *In Vivo* Proof of Negative Feedback Loop in Action: Alkermes' Nemvaleukin Alfa

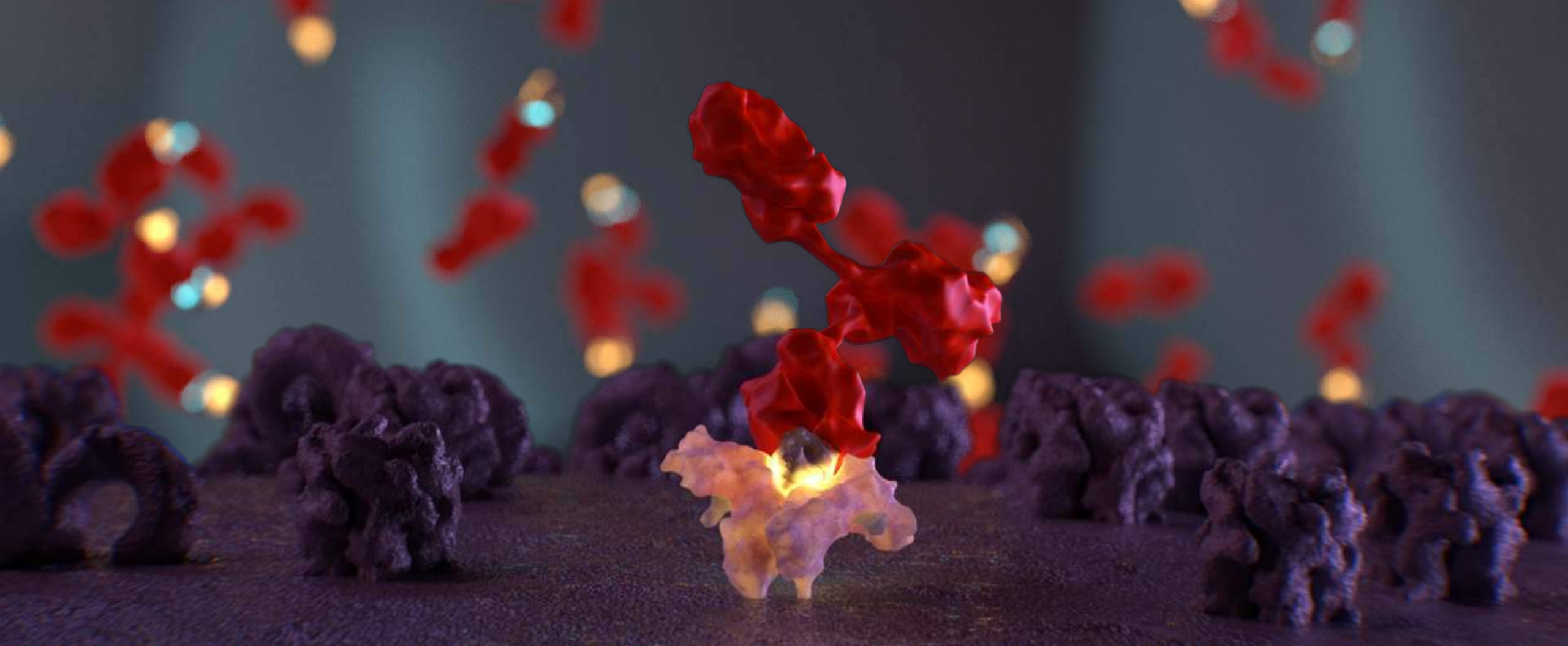


Fifth *In Vivo* Proof of Negative Feedback Loop in Action: Medicenna's MDNA11



Following the very first dose of MDNA11, Tregs begin to rise

Y-axis scaling obscures the significant fold increase in Tregs elicited by MDNA11

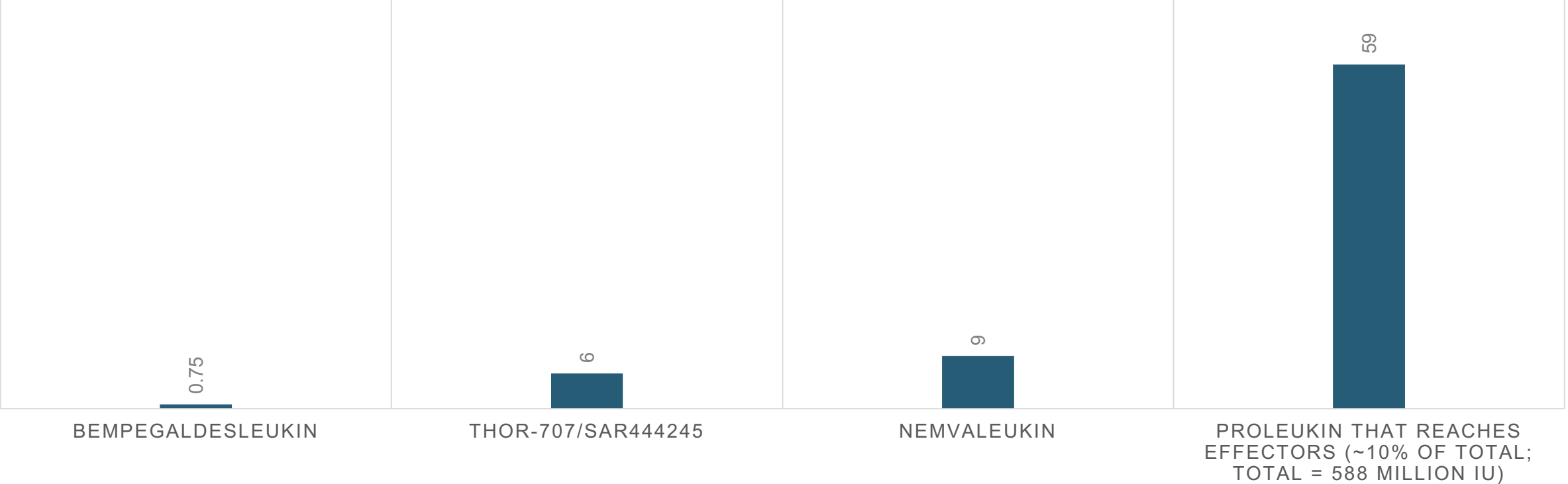


IL-2 equivalent amounts delivered by second generation
("non-alpha") agents is very low

aulos

IL-2 Equivalent Amounts Delivered by Second Generation, “Non-Alpha” Agents is Actually Very Low, and Correlates With Clinical Efficacy

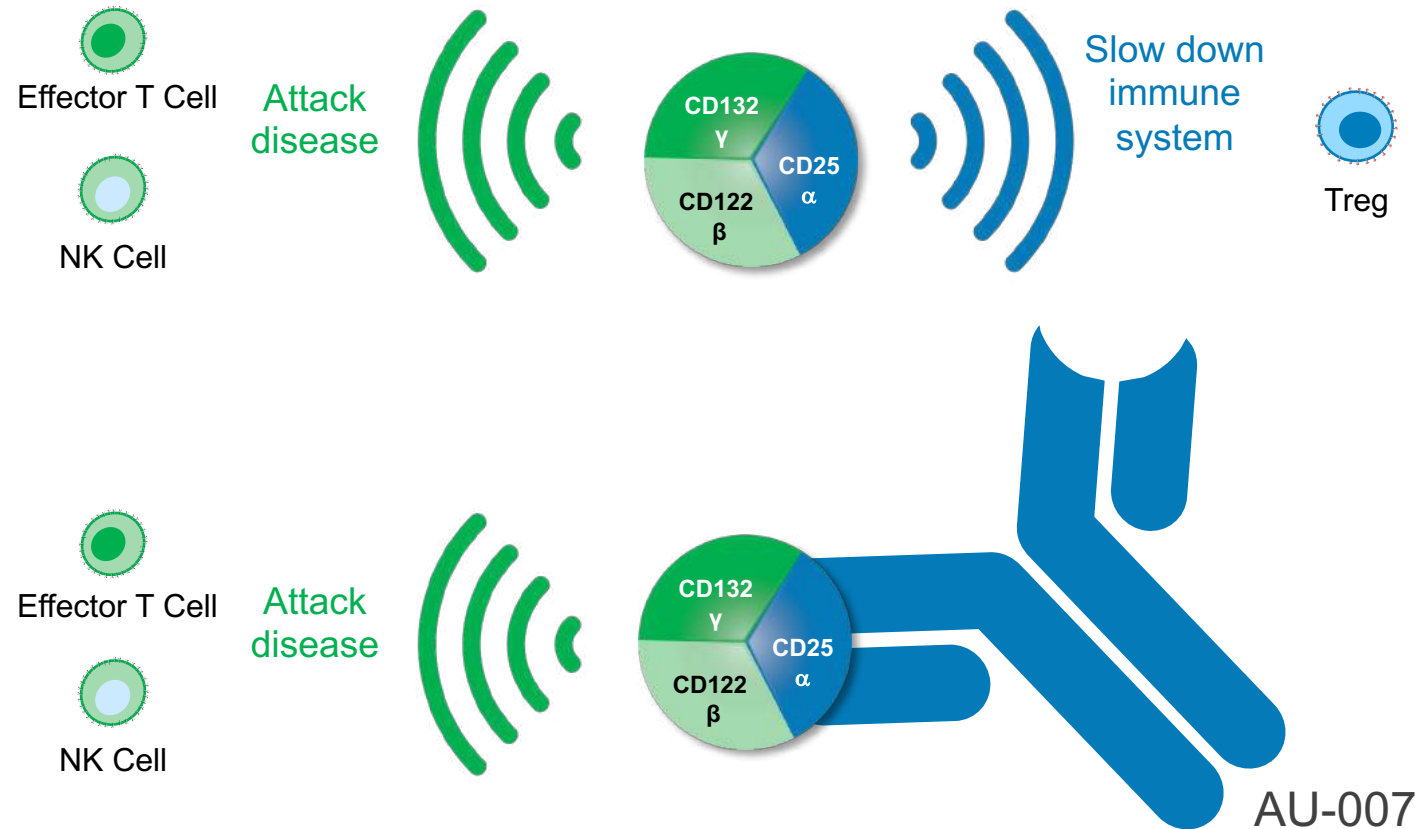
MILLIONS OF INTERNATIONAL UNITS (IU) DELIVERED
PER 2- OR 3-WEEK CYCLE



Modeling suggests that AU-007 will deliver as much or more IL-2 to effector T cells and NK cells as Proleukin®, while redirecting IL-2 away from Tregs, pulmonary endothelium, vasculature and eosinophils

AU-007 mAb Binds to the Part of IL-2 That Interacts With the Alpha (CD25) Receptor Subunit, Preventing Binding by IL-2 to Trimeric IL-2 Receptors

AU-007 BINDS TO CD25 DOMAIN ON IL-2

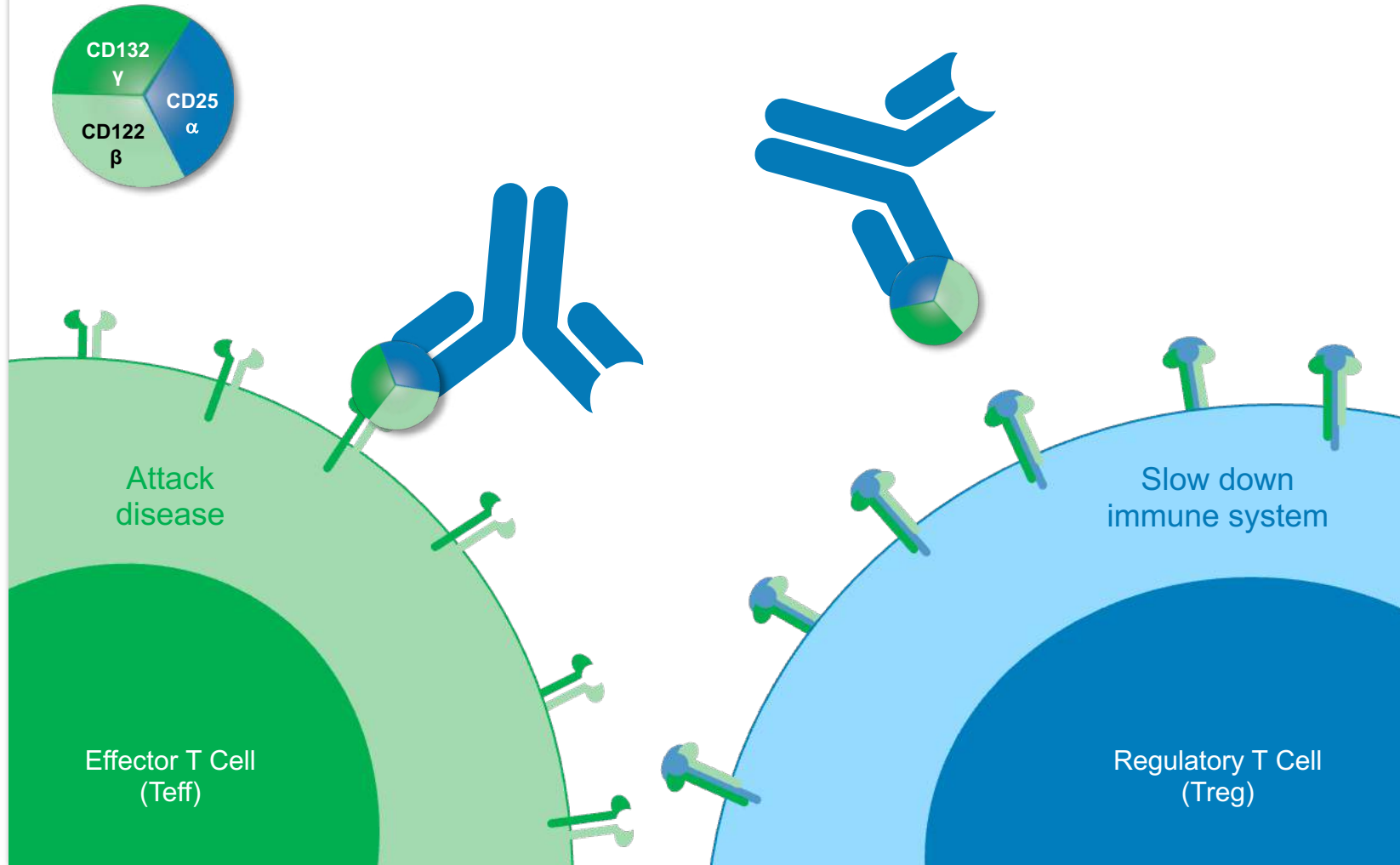


IL-2 signals to effector T cells and NK cells to attack disease, while it signals to Tregs to slow down an immune response

When AU-007 binds to CD25, it inhibits signaling to Tregs while still allowing IL-2 to bind to effector T cells and NK cells, which expand and kill tumor cells

AU-007 mAb Complex Binds to the Part of IL-2 That Interacts With the Alpha (CD25) Receptor Subunit, Preventing Binding by IL-2 to Tregs and Allowing Binding to Effector T Cells

AU-007 BINDS TO CD25 DOMAIN ON IL-2



AU-007 redirects IL-2 to bind only to dimeric receptors on effector T cells, yielding significant competitive advantages for efficacy and safety