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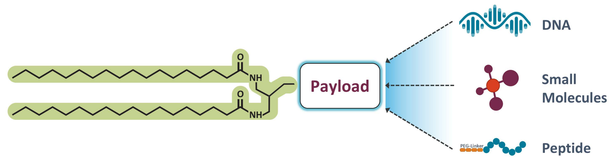
Direct Intratumoral Delivery of Amphiphilic ImmunoModulators (AIM) Induces Durable Anti-Tumor Efficacy

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Nature Medicine
Phase 2 Trial Analysis

AIM Therapy for Intratumoral Administration



Membrane/Albumin Binding Lipid
- ✓ Tumor Retention [1]
- ✓ Lymph Node Draining [2,3]

Immunostimulatory Adjuvant
- ✓ Activate Tumor-Resident APCs
- ✓ Induce Immune Activation within TME

AIM Therapy Rationale and Potential Benefits

In Situ Anti-Tumor Immune Response

Intratumoral AIM administration results in **immune activation** within the TME with the potential to:
- **Release antigen** from inflammation-damaged tumor cells
- **Stimulate tumor-resident APCs** that present antigen to **tumor infiltrating T cells (TILs)**
- **Reverse immuno-suppressive tumor microenvironment (TME)** and recruit immune cells

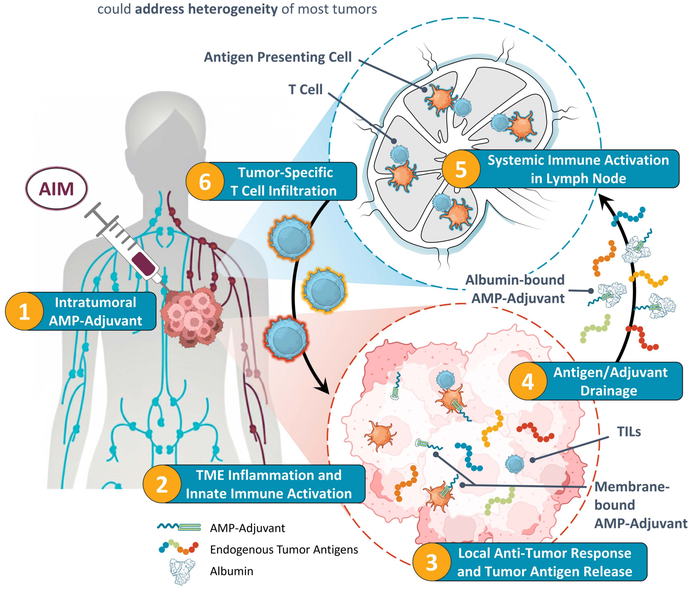
Enhanced Activity and Improved Safety due to Tissue Retention

AMP-lipid-mediated retention[1] of adjuvant within the tumor has the potential to ensure:
- Maximal adjuvant concentration at tumor site and **prolonged immunostimulation**
- **Minimal systemic toxicity**

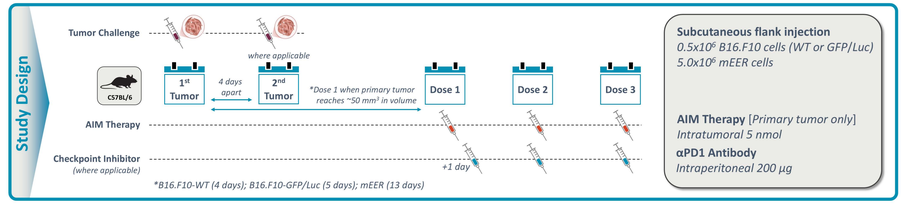
Excess adjuvant and antigen may collect at draining lymph nodes to activate systemic responses[2,3]

Off-the-Shelf and Personalized

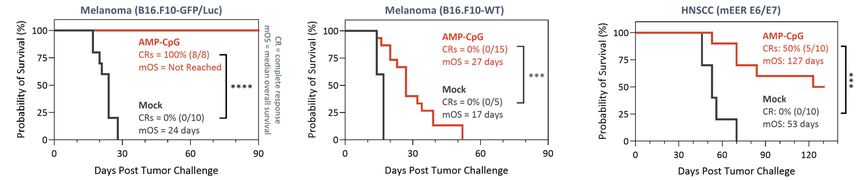
AIM Therapy is antigen- and tumor-agnostic:
- No antigen specification or manufacturing necessary as **antigen is supplied by tumor** itself
- Immunity is not limited to pre-specified antigens but may raise **polyclonal responses**, which could **address heterogeneity** of most tumors



AIM Therapy: Intratumoral (i.t.) Repeat-Dose Administration and Abscopal Effect Studies

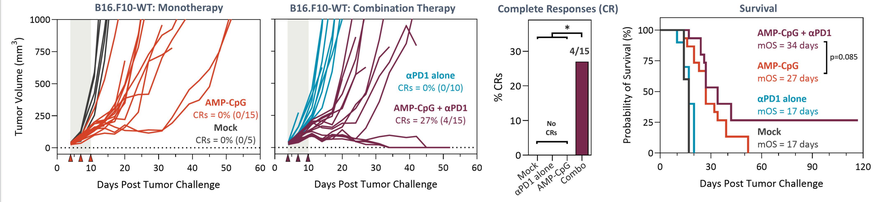


AMP-CpG Monotherapy Elicits Anti-Tumor Effect in Multiple Solid Tumor Models



AMP-CpG eradicated 50% of HNSCC, 100% of B16.F10-G/L tumors, and significantly delayed growth of B16.F10-WT

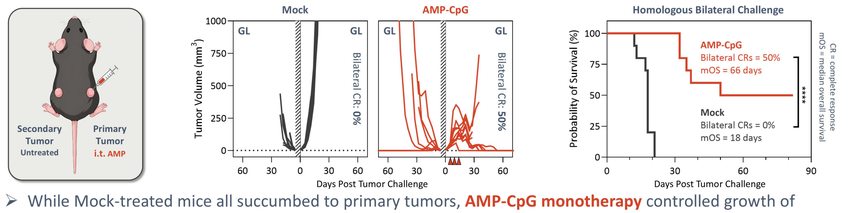
AMP-CpG Synergizes with Checkpoint Inhibitor Therapy to Eradicate B16.F10-WT Tumors



AMP-CpG (▲) plus αPD1 **Combo Therapy** (▲) induced complete responses in 27% of B16.F10-WT tumors.

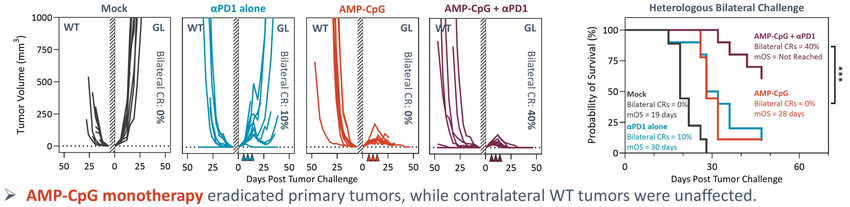
i.t. AMP-CpG Treatment Demonstrates Abscopal Effect in Bilateral Tumor Challenge

Homologous Bilateral Model: B16.F10-GFP/Luc (GL) tumors on both flanks; secondary tumor 4 days delayed (▩)



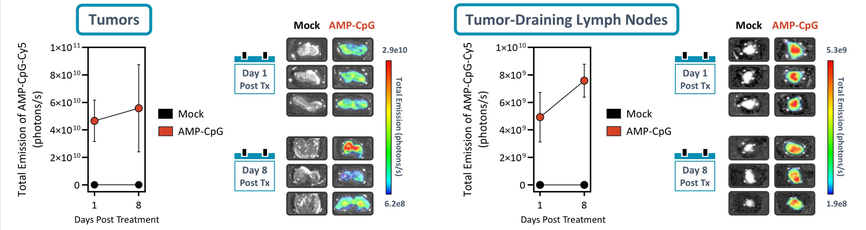
- While Mock-treated mice all succumbed to primary tumors, **AMP-CpG monotherapy** controlled growth of primary tumors and 50% (5/10) of mice achieved bilateral complete responses (CR).

Heterologous Bilateral Model: Treated B16.F10-GFP/Luc (GL); untreated B16.F10-WT (WT) with 4 days delay (▩)



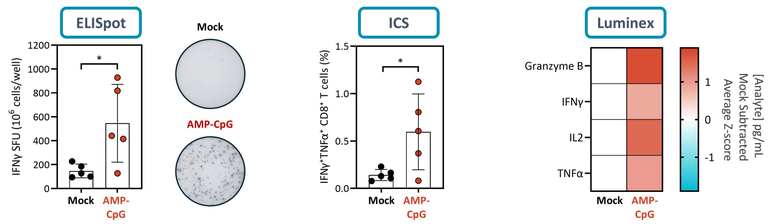
- **AMP-CpG monotherapy** eradicated primary tumors, while contralateral WT tumors were unaffected.
- However, **AMP-CpG + αPD1** delayed WT tumors as well and achieved complete bilateral responses in 40% of mice.

AMP-CpG Supports Tumor Retention as well as Lymph Node Accumulation in Mice



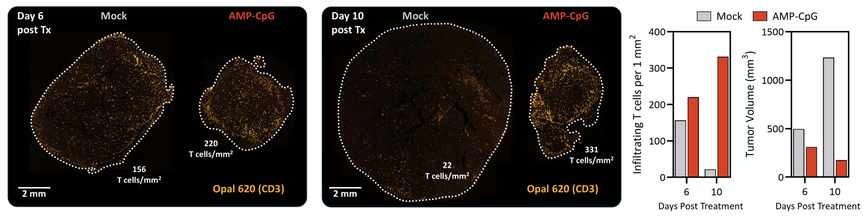
IVIS imaging of intratumorally administered, Cy5-labeled AMP-CpG demonstrates sustained tumor retention alongside efficient transport to tumor-draining lymph nodes and persistent accumulation therein.

Antigen-Agnostic AMP-CpG Treatment Generates Tumor-Specific T cell Responses in dLN



In mice, intratumoral AMP-CpG enhanced T cell cytokine responses in tumor-draining LNs against 5 selected TAAs 5 days post treatment, suggesting robust APC activation by AMP-CpG and efficient tumor antigen processing in dLNs.

AMP-CpG Facilitates Prolonged T Cell Survival and Anti-Tumor Effect in Murine Tumors



Intratumoral AMP-CpG treatment promoted retention of T cells in tumor tissue and regressed tumor size.

In Clinical Trials AMP-CpG Demonstrates Safety and Improved Prognosis in PDAC [4-6]

Camupepimut, an AMP-CpG immunotherapy targeting mKRAS, was investigated in a randomized Phase 2 clinical trial in patients with KRAS mutated adjuvant pancreatic ductal adenocarcinoma (PDAC) (**AMPLIFY-7P** Phase 1/2: NCT05726864).
- Safety: Camupepimut was generally well tolerated, with no new safety signals observed.
- Immunity: Post-hoc analyses show robust mKRAS-specific **T cell responses in 99% of evaluable treated patients**.
- Survival: Post-hoc analyses show that stronger immune responses were associated with improved disease-free survival (DFS). Median DFS was not reached in those above the previously identified immune-response threshold versus 6.08 months in those below the threshold (HR 0.22; p<0.0001). **Camupepimut improved prognosis with 49% lower recurrence risk** when compared to observation in baseline ctDNA^{neg} patients.

TAKE HOME MESSAGE
- **AIM-Therapy is an off-the-shelf approach with the potential to treat various tumors in an antigen-agnostic manner.**
- **AMP-CpG monotherapy eradicates solid tumors in mice and synergizes with checkpoint inhibitor therapy to eliminate more aggressive tumor models, including at distant, untreated sites, indicating a systemic antitumor effect.**
- **i.t. administered AMP-CpG efficiently drains to LNs eliciting tumor-specific T cell responses associated with T cell infiltration into the tumor, local retention, and antitumor activity, culminating in tumor regression and complete responses in mice.**

References:
1. Liu H, *et al.* **Angew. Chem. Int. Ed.** (2011) 50: 7052-7055
2. Liu H, *et al.* **Nature**. (2014) 27;507(7493):519-22
3. Moynihan KD, *et al.* **Nat Med.** (2016) 22(12):1402-1410
4. Pant S, *et al.* **Nat Med.** (2024) 30: 531–542
5. Wainberg ZA, *et al.* **Nat Med.** (2025) 31: 3648–3653
6. George B, *et al.* **Nat Med**. (2026)