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Equity Research
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# AACR26: Key Highlights from NUVL, NRIX, ALLO, STRO, PRLD, WHWK, ACRV

We summarize key AACR 2026 takeaways across NUVL, NRIX, ALLO, STRO, PRLD, WHWK, and ACRV.

*Key highlights on the Front Page and many details on ~20 Content Pages.*

*Reach out for our coverage posters.*

**NUVL** shared clinical data showing zidesamtinib delivers durable systemic and intracranial activity in heavily pretreated ROS1+ NSCLC, with ORR ~41-47%, median DOR ≥15.7mo, and frequent, durable CNS responses (including G2032R) in patients previously exposed to next-gen TKIs (covered in our NOTE). Preclinical CNS benchmarking independently supports this clinical durability, with superior brain exposure and sustained intracranial control versus repotrectinib and taletrectinib, ***reinforcing a durability-led value proposition heading into the 2L NDA (PDUFA 9/18/26) and planned 1L expansion***.

**NRIX** showed that targeted protein degradation can **meaningfully expand biology across multiple mechanisms: pan-mutant BRAF, AURKA, and CBL-B**, addressing clear limitations of inhibitors such as resistance, incomplete pathway suppression, and non-enzymatic functions. Across programs, the data emphasize depth (degradation vs inhibition), breadth (Class 1/2/3 BRAF, CNS activity), and early translational alignment (on-target PD for CBL-B).

**ALLO** shared preclinical data on a BCMA/ CD70 dual CAR T that showed potential in high-risk multiple myeloma. With recent positive data from Cema-cel's interim data readout (NOTE1, NOTE2), this preclinical data help further underscore the power of ALLO's platform to generate multi-target allogeneic off-the shelf cell Tx for oncology and immunology especially as it advances its CD19/ CD70 asset through its Ph1 trial for autoimmune diseases.

**STRO** data across TF, ITGB6, PTK7, and dual-payload programs highlight engineering-driven ADC innovation -- DAR8 exatecan and dual-payload formats -- capable of step-function gains in efficacy depth, tumor breadth, and durability versus first-generation ADCs. STRO-004 is emerging as the lead value driver, with preclinical data showing materially improved efficacy and exposure vs TF-MMAE benchmarks (64% ORR across 70 PDX models) alongside a ~50 mg/kg NHP HNSTD, ***supporting a wider therapeutic window ahead of mid-2026 STRIVE-1 Phase 1 topline***.

**PRLD** showed preclinical evidence supportive of PRT13722's KAT6A-specific degrader mechanism, which demonstrated deeper responses with reduced neutropenia vs. KAT6A/B inhibitor in a breast cancer xenograft model. Co also showed synergy between PRT13722 and SoC endocrine-, CDK4/6-, and PI3Kα-therapies, leading to complete responses, supporting the potential for improved activity as a combination regimen.

**WHWK** shared preclinical data on their PTK7-, MUC16-, and SEZ6-targeting ADCs, demonstrating potent targeting binding, cellular internalization, cytotoxic activity in cancer cell lines, and tumor regression in CDX/PDX models. Data is mixed on whether Co's proprietary Topo1 inhibitor payload is superior to exatecan, but Co's ADCs overall show comparable or stronger activity vs. previous ADC benchmarks and provide validation for tolerability and stability in NHP, ***supporting their ongoing evaluation in endometrial, ovarian, and SCLC settings***.

**ACRV** highlighted preclinical data suggesting Co's cell cycle checkpoint inhibitors (ACR-368 and ACR-2316) enhance anti-tumor activity when combined with PD-L1 blockade or ADC Topo1

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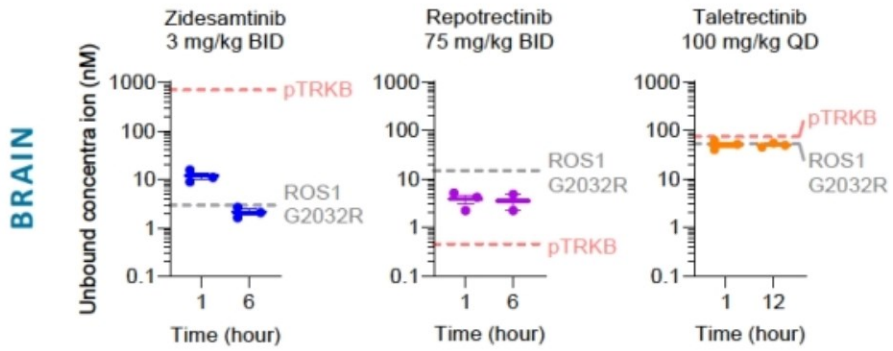
inhibitors. Data show enhanced immune signaling, increased NK/CD8+ activity, and reduced T cell exhaustion from ACR-368/ACR-2316 combination with PD-L1 inhibition, resulting in complete and durable responses in CDX models. Overall, ***the dataset supports continued development of checkpoint inhibitors in combination regimens, including potential future solid-tumor strategies with ICIs.***

## NUVL

**Abstract LB366: Zidesamtinib shows differentiated brain penetrance and intracranial efficacy versus other ROS1 inhibitors**

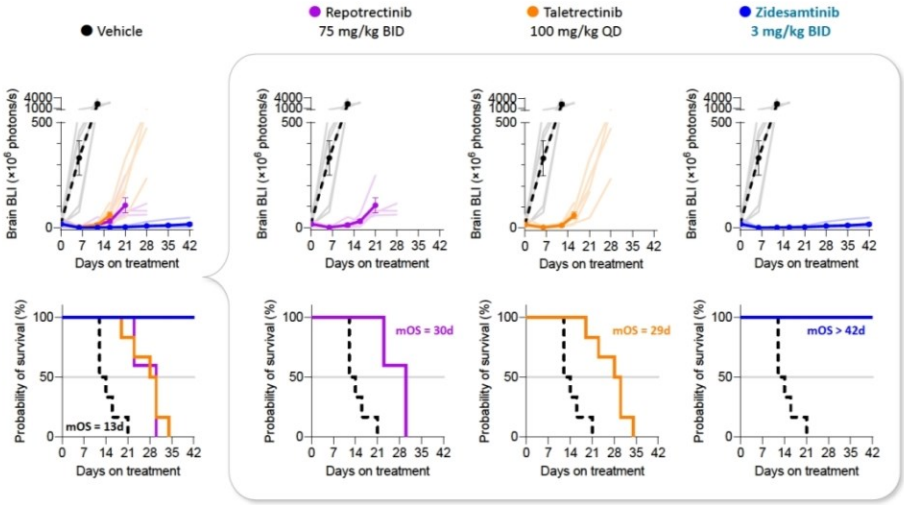
Zidesamtinib (NVL-520) was benchmarked against repotrectinib and taletrectinib in preclinical ROS1 G2032R CNS models to assess brain penetrance and intracranial durability. Zidesamtinib demonstrated superior CNS exposure, with unbound brain concentrations at or above the ROS1 G2032R $IC_{50}$ while remaining below TRK inhibition thresholds, whereas repotrectinib and taletrectinib achieved brain levels below (repotrectinib) or near (taletrectinib) the ROS1 G2032R $IC_{50}$ Chart 1. In an aggressive intracranial Ba/F3 CD74-ROS1 G2032R luciferase model (vehicle mOS 13 days), zidesamtinib produced sustained suppression of brain tumor burden with all mice surviving beyond 42 days, compared with median OS of 30 days for repotrectinib and 29 days for taletrectinib, both showing transient responses Chart 2. Switching to zidesamtinib after progression on taletrectinib re-elicited tumor control and extended survival beyond 42 days, supporting activity post-ROS1 TKI failure Chart 3. Data suggest zidesamtinib delivers the most durable intracranial efficacy among ROS1 inhibitors tested in this resistance model, consistent with its differentiated CNS exposure profile.

**Chart 1 - Zidesamtinib Levels in CNS show favorable PK**



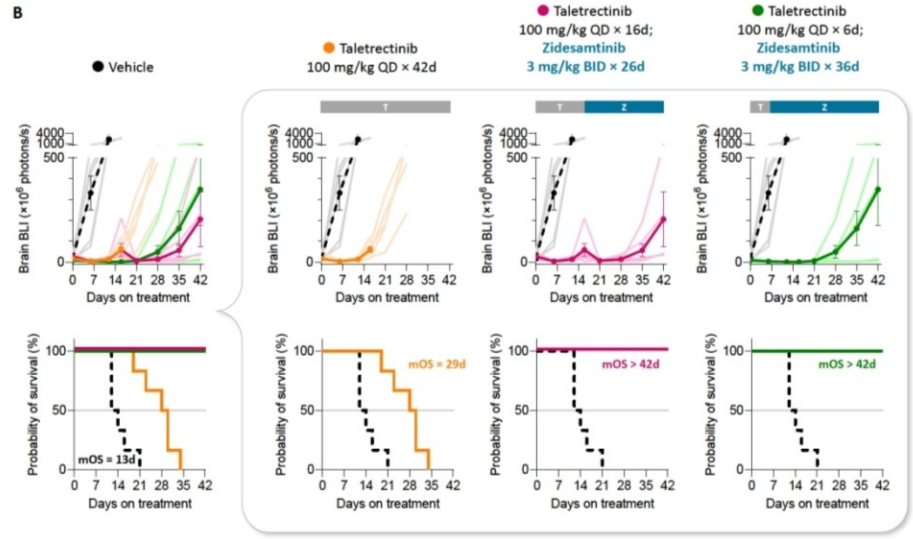

Source: AACR 2026

**Chart 2 - Efficacy in intracranial Ba/F3 CD47-ROS1 G2032 Luciferase Model**




Source: AACR 2026

**Chart 3 - Treatment switching from teletrectinib to zidesamtinib improved trreatment outcomes**



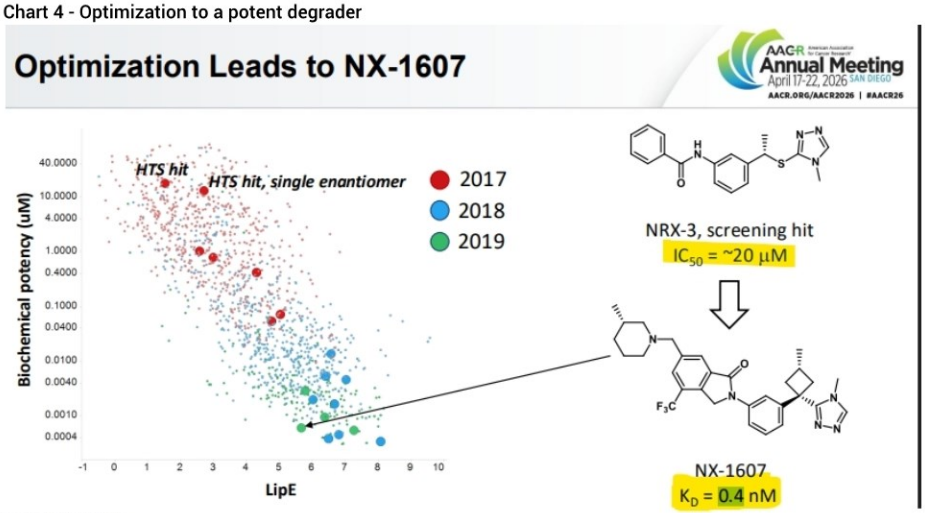

Source: AACR 2026

**Zidesamtinib demonstrates durable systemic and intracranial activity post-next-gen ROS1 TKIs.** In heavily pretreated ROS1+ NSCLC patients previously exposed to repotrectinib or taletrectinib, zidesamtinib delivered ORR ~41-47% with median DOR 15.7 months or not reached, and frequent, durable intracranial responses (IC-ORR 44-71%; IC-DOR not reached), including G2032R disease, supporting durability-driven value in post-TKI settings where prior agents have historically shortened treatment duration. See our NOTE for more details.

**Abstract # Discovery and Characterization of CBL-B Intramolecular Glue Inhibitors (NX-1607)**

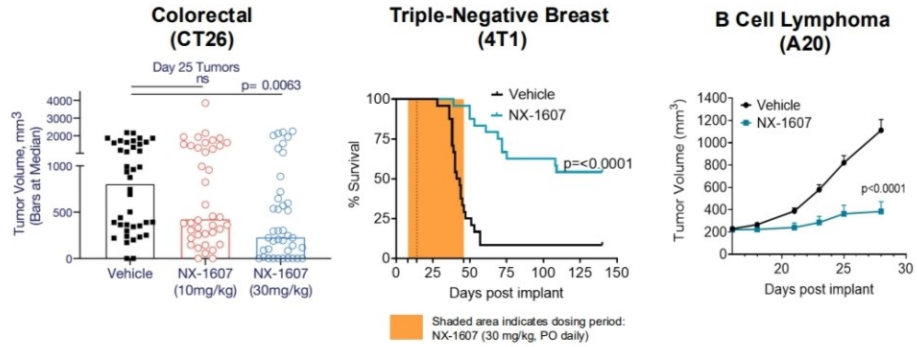
Co described the discovery and optimization of CBL-B intramolecular glue inhibitors, culminating in NX-1607, an oral small molecule that stabilizes CBL-B in a closed, inactive conformation rather than inducing degradation. A mechanism-agnostic HTS (~250k compounds) yielded a singleton hit that was structure-guided to NX-1607, improving affinity from ~20 μM to sub-nanomolar binding (KD ~0.4 nM) with confirmed intramolecular glue binding by co-crystallography Chart 4. Pharmacologic CBL-B inhibition recapitulated genetic ligase-inactivation phenotypes, enhancing IL-2 and IFN-γ secretion and producing single-agent antitumor activity across syngeneic models (CT26, 4T1, A20) Chart 5. NX-1607 showed strong survival synergy with anti-PD-1 in multiple models, including reduced metastatic burden in 4T1. Preclinical PK supported oral dosing, and early clinical data demonstrated dose-dependent PK with on-target PD, evidenced by increased pHS1-positive CD8 T cells Chart 6. Our net read-through is the data position NX-1607 as a differentiated, non-degrader intracellular checkpoint inhibitor with genetic validation, scalable chemistry, and early human biomarker engagement, with clinical durability and immune safety remaining key open questions.

Chart 4 - Optimization to a potent degrader



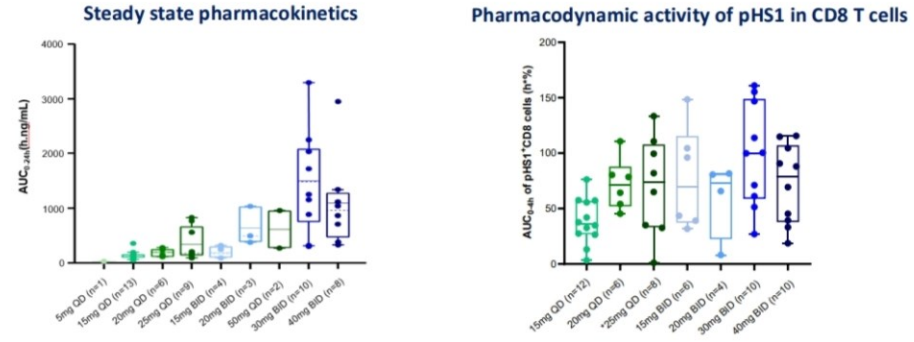

Source: AACR 2026

Chart 5 - Single-Agent NX-1607 Induces Anti-tumor Response in Multiple Models




Source: AACR26

Chart 6 - PK Profile Supports Oral Dosing




Source: AACR 2026

**Abstract #5801: NRX-0305 is an orally bioavailable, pan-mutant BRAF degrader that exhibits single-agent and combination efficacy with MEKi or anti-EGFR across Class 1/2/3 BRAF-mutant cancers**

NRX-0305 was profiled across Class 1, 2, and 3 BRAF-mutant systems to demonstrate breadth beyond V600 disease and combination positioning. In vitro, NRX-0305 degraded Class 1/2/3 mutant BRAF, suppressed pERK signaling, and avoided WT BRAF degradation or paradoxical MAPK activation. In vivo, single-agent activity was observed across multiple CDX and PDX models, including BRAFi-resistant Class 1 tumors. Combination studies showed enhanced efficacy with anti-EGFR (cetuximab) in a BRAF V600E CRC xenograft (HT-29) Chart 7 and with MEK inhibitors (binimetinib or trametinib)

in Class 2 (G469A) and Class 3 (G466V/D594N) PDX models, with TGI approaching ~90-95% in select combinations alongside on-tumor BRAF depletion and pERK suppression Chart 8. A 14-model PDX screen spanning Class 1 Tx-resistant, Class 2, and Class 3 mutations showed consistent tumor volume reduction over 7-14 days at a fixed dose Chart 9. Net-net, the dataset frames NRX-0305 as a potential breadth-expansion BRAF agent with rational MEKi and anti-EGFR combinations; however, efficacy readouts are short-duration, exposure-unmatched, and derived from a non-optimized compound.

**Chart 7 - NRX-0305 is efficacious in Class 1 (V600E) CRC subcutaneous xenograft model as a single agent and demonstrates enhanced anti-tumor activity in combination with anti-EGFR, Cetuximab**

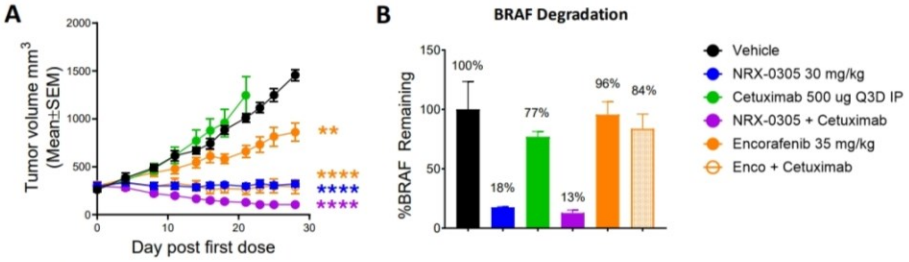



**Figure 4. (A)** Mice bearing subcutaneous Class 1 V600E HT-29 xenograft tumors were dosed daily with NRX-0305^, Cetuximab, Encorafenib as single agents or in combination at the indicate doses (PO, QDx28 or IP, Q3D). Two-way ANOVA, mixed effects model with Dunnett's multiple comparisons test vs vehicle. p value: ** ≤ 0.01, *** ≤ 0.001, **** ≤ 0.0001. **(B)** BRAF levels were assessed in tumors collected 4 hours after the third dose by Simple Western (Jess).

Source: AACR26

**Chart 8 - NRX-0305 synergy with MEK inhibition is maintained at lower Trametinib dose levels in Class 3 (D594N) subcutaneous bladder cancer PDX model**

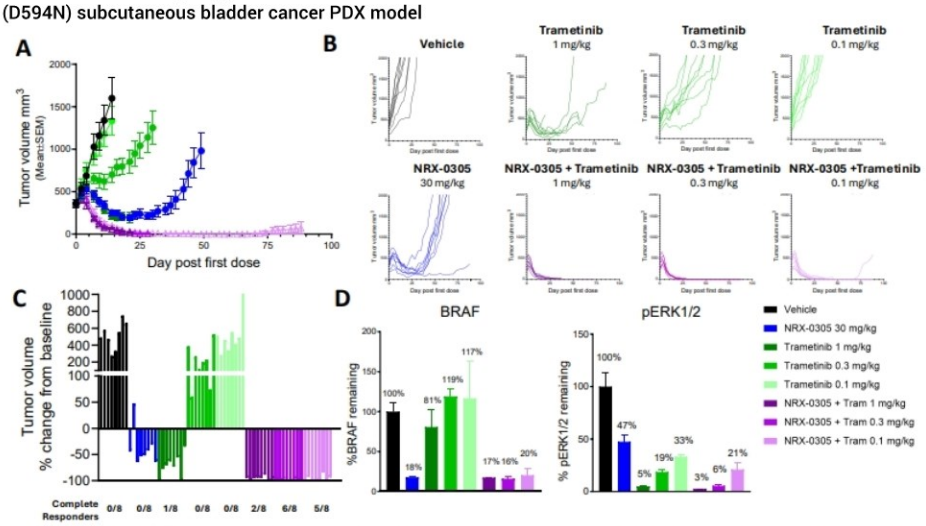



**Figure 8. (A)** Mice bearing subcutaneous Class 3 (BRAF D594N) bladder PDX tumors were dosed daily with NRX-0305^, trametinib or a combination at the indicated doses (PO, up to QDx89). Grey shading indicates dosing interval. **(B)** Spider plots associated with each of the dosing groups. **(C)** Mean tumor volume percent change from baseline for each dosing group at day 28 of the study or the last measured timepoint (for animals that came down before day 28). **(D)** BRAF and pERK levels were assessed in tumors collected 4 hours after the third dose by Simple Western (Jess).

Source: AACR26

**Chart 9 - NRX-0305 demonstrates anti-tumor activities across a wide range of Class 1 BRAF inhibitor-resistant and Class 2/3 mutant PDX models**

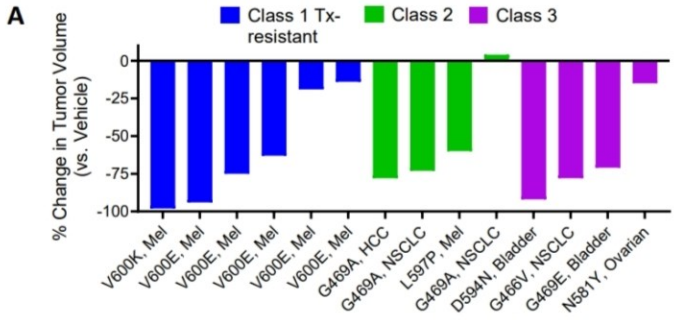


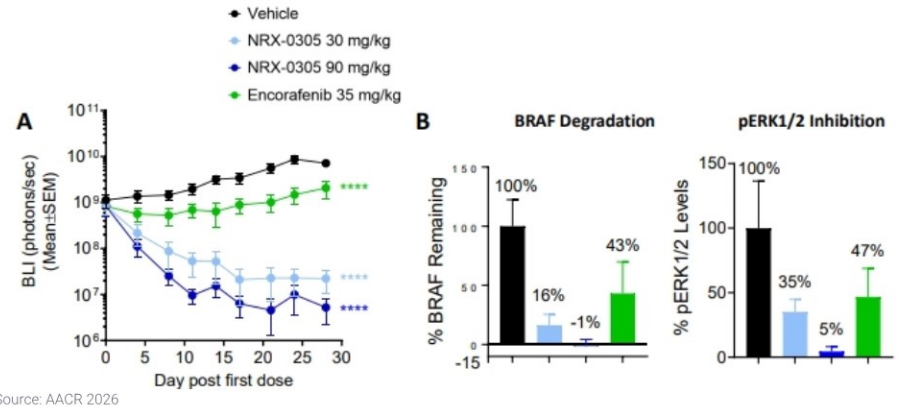

**Figure 5. (A)** NRX-0305 was dosed in 14 different PDX models spanning Class 1 treatment-resistant, Class 2 and Class 3 BRAF mutations. Percent change in tumor volume versus vehicle was determined following 14 or 7 (D594N Bladder model only) consecutive days of dosing at 90 mg/kg. Mel, melanoma; HCC, hepatocarcinoma cancer; NSCLC, non-small cell lung cancer

Source: AACR26

**Abstract #4617: NRX-0305, an orally bioavailable, CNS penetrant pan-mutant BRAF degrader demonstrates robust efficacy in intracranial models of melanoma brain metastasis and primary glioma**

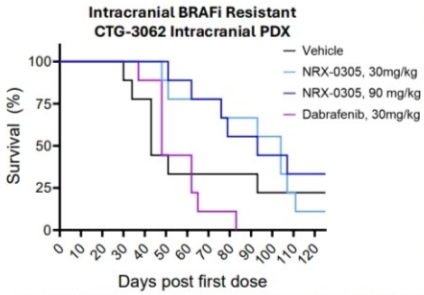
NRX-0305, a non-optimized, orally bioavailable pan-mutant BRAF degrader, was evaluated in intracranial melanoma and glioma models to address CNS penetration and BRAFi resistance. PK/PD studies showed dose-proportional plasma, tumor, and brain exposure, with brain:plasma ratios ~0.2-0.6 at Cmin, resulting in near-complete mutant BRAF degradation (to ~1-16% remaining) and sustained pERK1/2 suppression (to ~5-35%) in tumor tissue Chart 10. In intracranial Class 1 BRAF V600E CDX models (A375, AM38), NRX-0305 reduced tumor burden versus vehicle and BRAFi comparators. In a BRAFi-resistant intracranial melanoma brain metastasis PDX (CTG-3062; V600K; progressed on dabrafenib), daily NRX-0305 significantly prolonged survival, increasing median survival from 43-48 days (vehicle/dabrafenib) to 93-104 days at 30-90 mg/kg QD (~116-142% lifespan increase) Chart 11. Overall, data support mutant-selective BRAF degradation with functional CNS exposure and activity in BRAFi-resistant brain disease limitations include mg/kg dosing, absence of therapeutic-index data, and use of a non-optimized lead.

**Chart 10 - NRX-0305 treatment demonstrated dose-dependent, anti-tumor activity in an intracranial, Class 1 BRAF (V600E) melanoma CDX model**




Source: AACR 2026

Chart 11 - NRX-0305 extends survival in a BRAFi resistant intracranial melanoma metastasis PDX model




| Sample | Median Survival (days) | Significance vs Vehicle | | Significance vs Dabrafenib | | Increase in Lifespan (%) |
|---|---|---|---|---|---|---|
| | | Day 65 | Day 125 | Day 65 | Day 125 | |
| Vehicle | 43 | - | - | ns | ns | 0 |
| NRX-0305 (30mg/kg) | 104 | * | ns | * | ** | 142% |
| NRX-0305 (90mg/kg) | 93 | * | ns | * | ** | 116% |
| Dabrafenib | 48 | ns | ns | - | - | 12% |

**Figure 7.** Mice bearing intracranial BRAFi resistant CTG-3062 PDX tumors were dosed once daily with NRX-0305˚ or dabrafenib at the indicated doses (PO, QDx125). Kaplan Meier survival analysis was performed, and significance was calculated by Log-Rank (Mantel-Cox) test vs vehicle or Dabrafenib at days 65 and 125. Percent increase in lifespan was calculated by the following formula: [Median survival (treated)-median survival (control)/median survival (control)x100]. Statistical significance: *p ≤ 0.05, ** p ≤ 0.01, ns=not significant.

Source: AACR 2026

**Abstract #5166: NRX-4972, a selective, oral, Aurora kinase A degrader, demonstrates increased efficacy in an SCLC tumor model, and greater in vitro synergy than an AURKA inhibitor**

NRX-4972, an orally bioavailable AURKA degrader, was evaluated to address limitations of AURKA inhibitors that do not eliminate kinase-independent scaffolding functions. NRX-4972 achieved potent, proteasome-dependent AURKA degradation (DC50 ~6 nM; Dmax ~95%) with minimal off-target signal in PBMC proteomics and demonstrated higher tumor and brain exposure than the AURKA inhibitor LY3295668 Chart 12. In MYC-driven models (IMR32 neuroblastoma; H82 SCLC), NRX-4972 outperformed inhibitors as monotherapy, with the most pronounced benefit observed under BID dosing, which improved target coverage and depth of PD effects ( ↓ total and p-AURKA to ~10-20%, ↑ γH2AX, ↑ cleaved PARP, ↓ MYC). In the H82 SCLC model, ~60% of mice remained alive at study end (>70 days) with BID NRX-4972, versus 0% survival for BID inhibitor arms Chart 13. An in-vitro Bliss synergy screen (8 cell lines × 28 agents) showed more frequent and stronger synergy for NRX-4972 versus LY3295668, with peak excess-over-Bliss scores up to ~0.8 (e.g., paclitaxel combinations) Chart 14. Overall, degradation confers efficacy and PD advantages over inhibition, with dosing strategy critical; combination data remain in vitro, and clinical feasibility of sustained BID dosing is unaddressed.

Chart 12 - AURKA PD

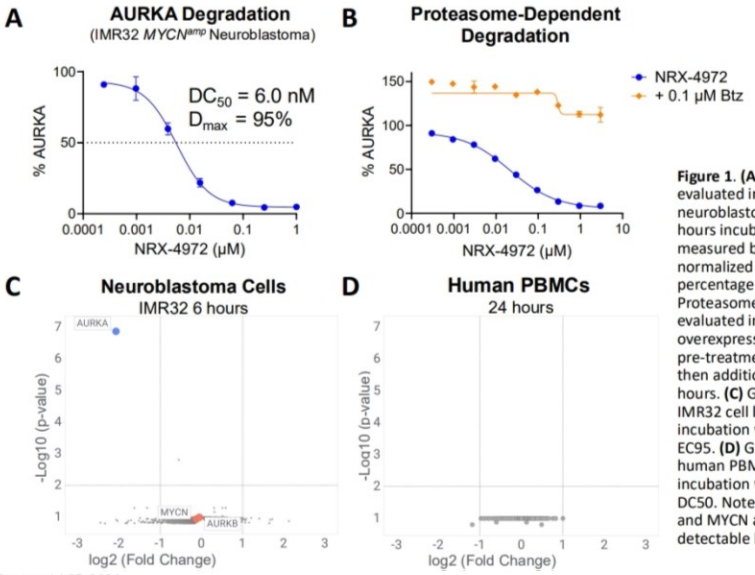
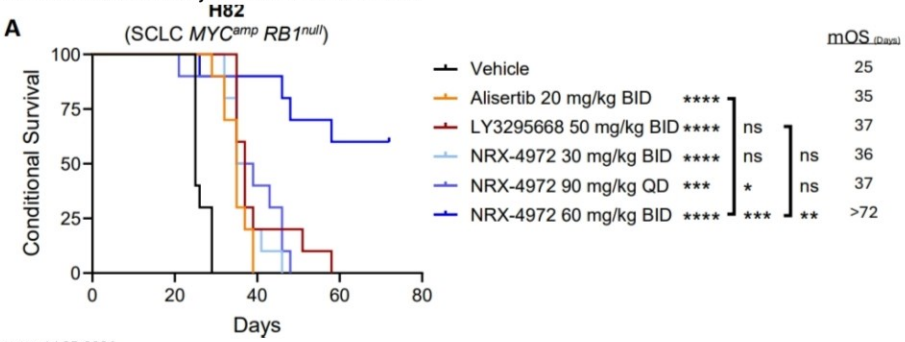



**Figure 1. (A)** NRX-4972 potency evaluated in human IMR32 neuroblastoma cell line after 6 hours incubation; AURKA measured by Jess Simple Western normalized to vinculin as a percentage of DMSO control. **(B)** Proteasome-dependence was evaluated in an MV4-11 AURKA-overexpression HiBiT line; 1 hour pre-treatment of bortezomib, then addition of NRX-4972 for 6 hours. **(C)** Global proteomics in IMR32 cell line after 6 hours incubation with NRX-4972 at EC95. **(D)** Global proteomics in human PBMCs after 24 hours incubation with NRX-4972 at 50x DC50. Note that AURKA, AURKB, and MYCN are not expressed at detectable levels in PBMCs.

Source: AACR 2026

Chart 13 - In Vivo Efficacy shows extended survrival




Source: AACR 2026

Chart 14 - Peak excess-over-Bliss scores up to ~0.8

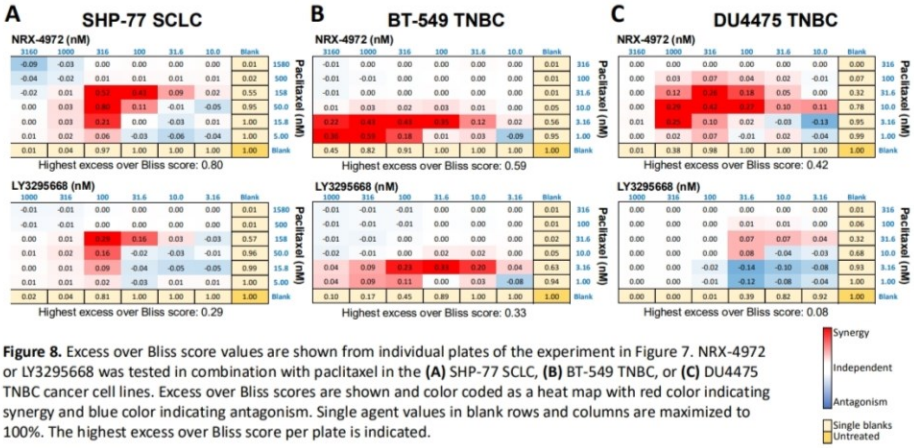



**Figure 8.** Excess over Bliss score values are shown from individual plates of the experiment in Figure 7. NRX-4972 or LY3295668 was tested in combination with paclitaxel in the **(A)** SHP-77 SCLC, **(B)** BT-549 TNBC, or **(C)** DU4475 TNBC cancer cell lines. Excess over Bliss scores are shown and color coded as a heat map with red color indicating synergy and blue color indicating antagonism. Single agent values in blank rows and columns are maximized to 100%. The highest excess over Bliss score per plate is indicated.

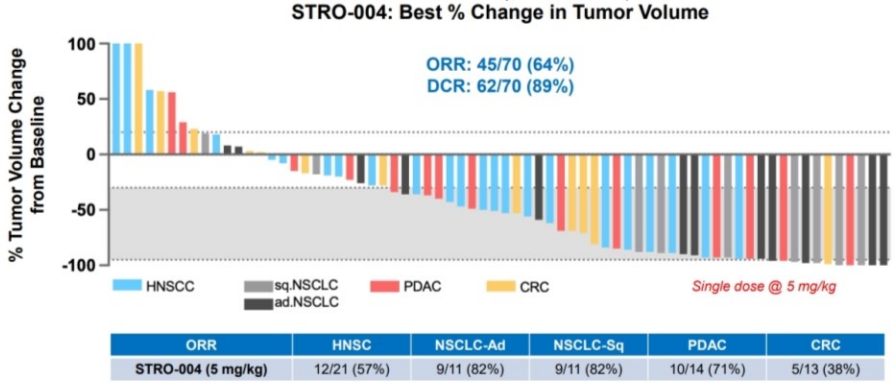
Source: AACR 2026

# STRO

**Abstract #9950: STRO-004 (DAR8 Exatecan TF-ADC) Shows Broad, Durable Activity Across TF-Expressing PDX Models**

STRO-004, a next-generation DAR8 exatecan Tissue Factor (TF) ADC, demonstrated broad and consistent antitumor activity across a large single-mouse PDX “mouse clinical trial” spanning HNSCC, NSCLC (adeno and squamous), PDAC, and CRC. In a single-dose 5 mg/kg study across 70 PDX models, STRO-004 achieved an ORR of 64% (45/70) and DCR of 89% (62/70) Chart 15, with responses observed even in TF-low tumors. STRO-004 outperformed benchmark TF-MMAE ADCs across multiple tumor types, including higher ORR in HNSCC (57% vs 38%) and PDAC (71% vs 50%), and longer median survival (e.g., 45 vs 27 days in HNSCC) Chart 16. The DAR8 exatecan design enabled higher payload delivery and deeper tumor regressions at lower doses than first-generation TF ADCs. Biomarker analyses showed TF expression correlates with response depth, while combined TF + SLFN11 expression better stratified responders, highlighting payload sensitivity as a key determinant. Data support STRO-004 as a differentiated TF ADC with potential to surpass the efficacy ceiling of first-generation TF-MMAE constructs.

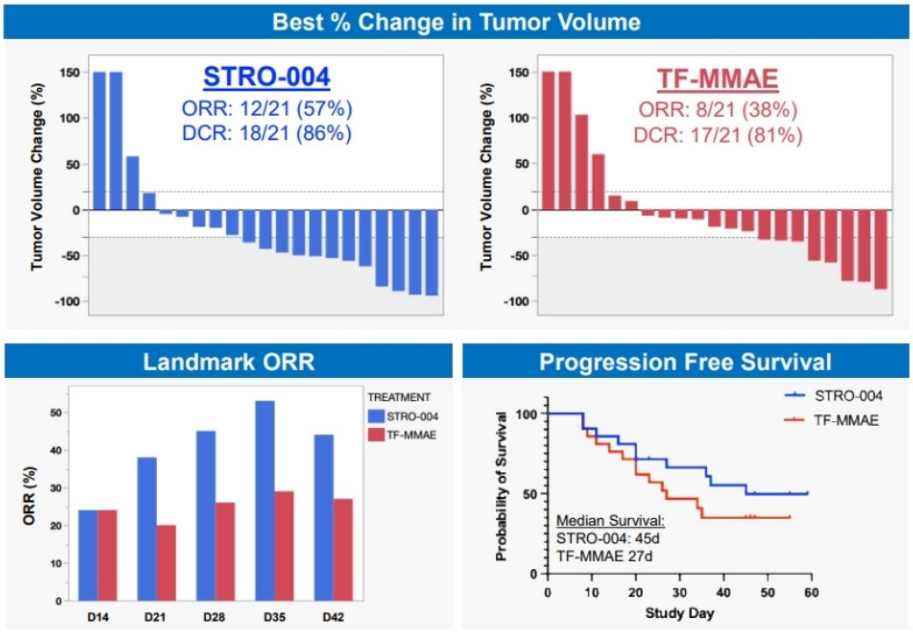
Chart 15 - STRO-004 Demonstrates Robust Anti-tumor Activity Across Multiple Solid Tumor PDX Models




| ORR | HNSC | NSCLC-Ad | NSCLC-Sq | PDAC | CRC |
|---|---|---|---|---|---|
| STRO-004 (5 mg/kg) | 12/21 (57%) | 9/11 (82%) | 9/11 (82%) | 10/14 (71%) | 5/13 (38%) |

Source: AACR 2026

Chart 16 - HNSCC: STRO-004 Drives Deeper Response and Longer Survival vs. TF-MMAE

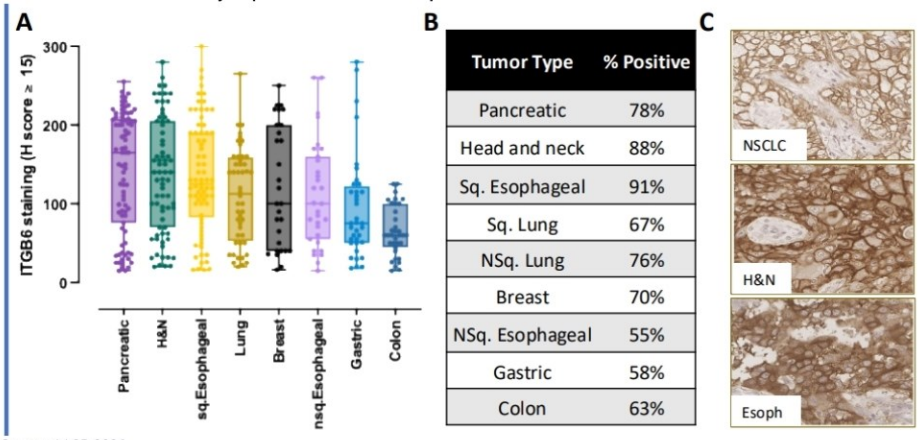



Source: AACR 2026

**Abstract #3299: An Integrin beta-6–Targeting DAR8 ADC Demonstrates Favorable Safety Profile and Potent Antitumor Activity in Preclinical Solid Tumors**

STRO-006 is a highly selective αvβ6-targeting DAR8 exatecan ADC designed to deliver tumor-selective cytotoxicity while preserving physiologic TGF-β signaling. ITGB6 expression was prevalent across solid tumors (e.g., 88% HNSCC, 91% esophageal, 78% pancreatic) Chart 17, supporting a broad addressable population. In CDX and PDX models of NSCLC and HNSCC, STRO-006 produced robust, dose-dependent tumor regressions at clinically relevant doses and outperformed αvβ6 benchmark ADCs. PK studies showed low clearance, extended half-life (~7–8 days), stable DAR, and minimal free exatecan exposure, supporting an improved therapeutic index Chart 19, Chart 20. In NHPs, STRO-006 was well tolerated up to 25 mg/kg, with no myelosuppression and only mild skin/GI effects. Data position STRO-006 as a differentiated αvβ6 ADC, with IND filing planned for 2026.

Chart 17 - ITGB6 is Broadly Expressed Across Multiple Solid Tumors Indications



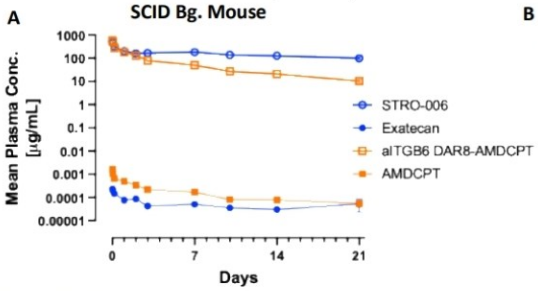

B

| Tumor Type | % Positive |
|---|---|
| Pancreatic | 78% |
| Head and neck | 88% |
| Sq. Esophageal | 91% |
| Sq. Lung | 67% |
| NSq. Lung | 76% |
| Breast | 70% |
| NSq. Esophageal | 55% |
| Gastric | 58% |
| Colon | 63% |




Source: AACR 2026

Chart 18 - STRO-006 Exhibits Superior PK, Low Clearance and Low Payload Exposure

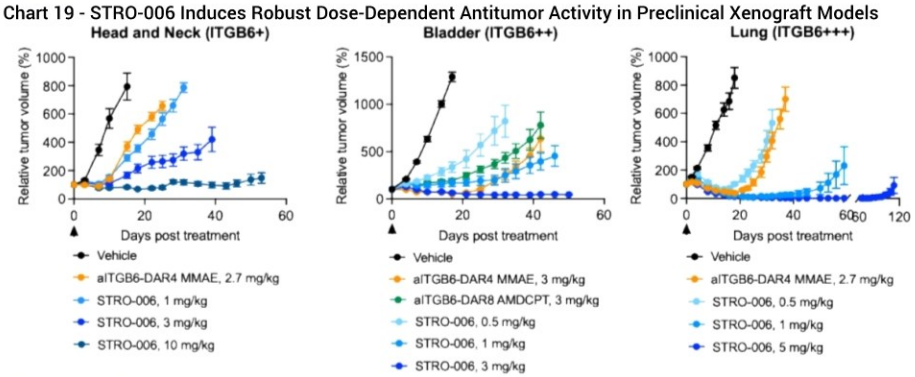



B

| aITGB6 ADC | Analyte | $C_{max}$ (ug/mL) | $CL_{obs}$ (mL·d$^{-1}$/kg) |
|---|---|---|---|
| STRO-006 | TAb | 550 | **2.33** |
| | ADC | 530 | **2.30** |
| | Exatecan | **0.000232** | NA |
| aITGB6 DAR8-AMDCPT | TAb | 533 | 13.3 |
| | ADC | 578 | 23.8 |
| | AMDCPT | 0.00160 | NA |

Source: AACR 2026

Chart 19 - STRO-006 Induces Robust Dose-Dependent Antitumor Activity in Preclinical Xenograft Models

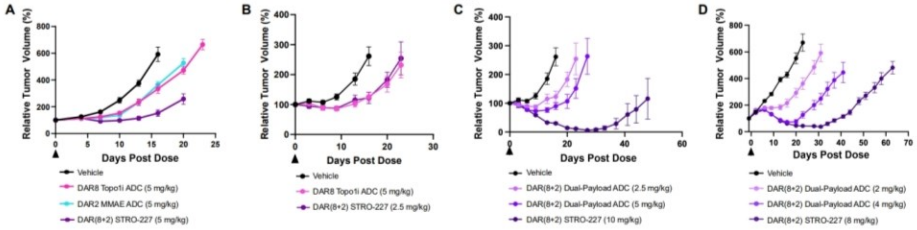



Source: AACR 2026

**Abstract #2025-227: STRO-227 (PTK7 Dual-Payload ADC) Shows Superior Efficacy Versus Single-Payload ADCs**

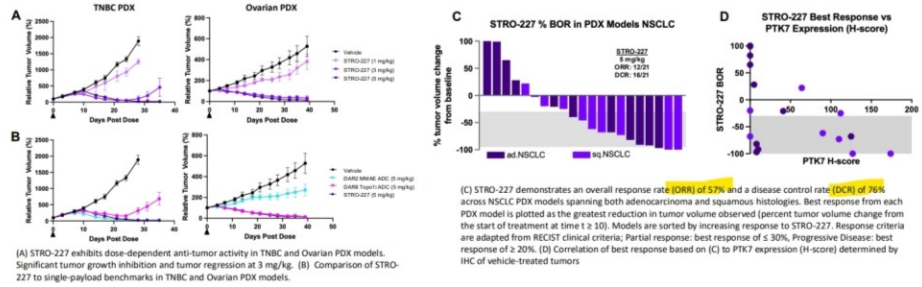
STRO-227 is a PTK7-targeting dual-payload ADC (DAR10; 8 exatecan + 2 MMAE) designed to address tumor heterogeneity and payload resistance. PTK7 is broadly expressed and enriched in tumor-initiating cells, supporting pan-tumor relevance. In TNBC, ovarian, and NSCLC CDX/PDX models, STRO-227 demonstrated greater tumor regression and higher ORR/DCR versus matched single-payload exatecan or MMAE ADCs. Efficacy was achieved at ~half the dose required for single-payload exatecan ADCs, consistent with additive cytotoxic mechanisms Chart 20, Chart 21. PK and toxicology studies showed stable DAR, low free payload exposure, and NHP HNSTD ~25 mg/kg, comparable to single-payload benchmarks Chart 22. Data support STRO-227 as a potential best-in-class PTK7 ADC, with an IND anticipated in late 2026.

Chart 20 - STRO-227 Delivers Robust Anti-Tumor Activity in Breast Cancer CDX Models



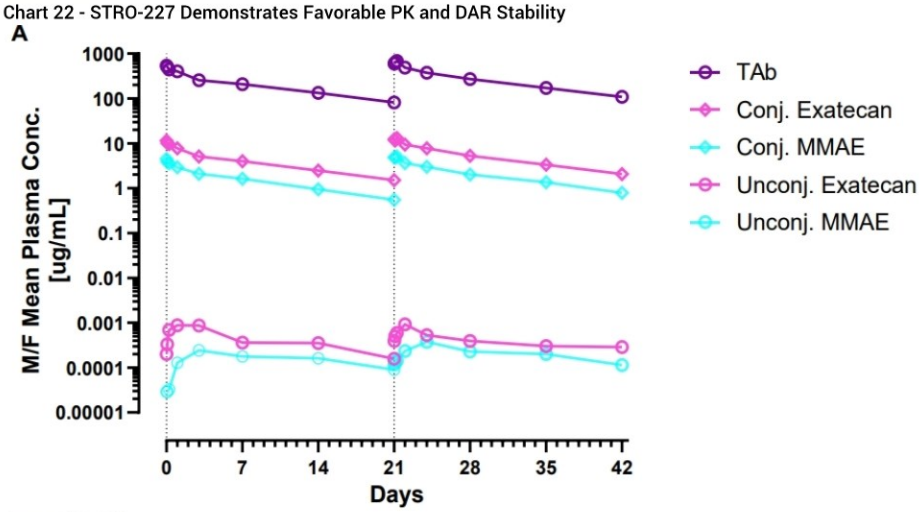

Source: AACR 2026

Chart 21 - Broad Anti-Tumor Activity of STRO-227 Across PDX Models




Source: AACR 2026

Chart 22 - STRO-227 Demonstrates Favorable PK and DAR Stability

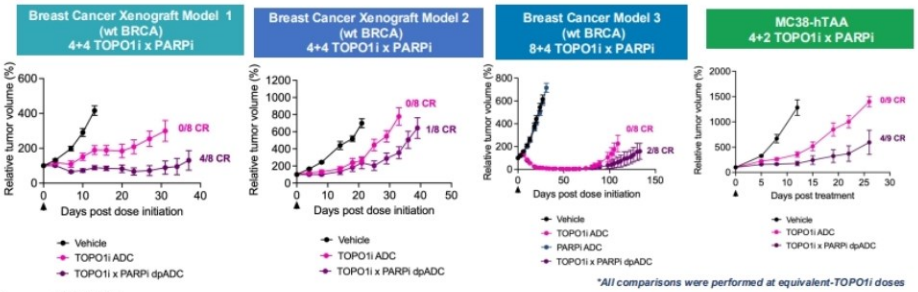



Source: AACR 2026

**Abstract #4436: Dual-Payload TOPO1i × DDRi ADCs Sustain DNA Damage and Overcome Resistance**

Co's site-specific high-DAR dual-payload ADCs co-delivering TOPO1 inhibitors and DDR inhibitors (PARPi) to overcome resistance and toxicity limitations of single-payload ADCs. Mechanistic studies showed TOPO1i-induced replication stress is rapidly repaired via PARP-dependent pathways, while dual-payload ADCs sustained γH2AX signaling and enhanced cell death. In vitro and in vivo models demonstrated greater cytotoxicity and antitumor efficacy versus TOPO1i-only ADCs at equivalent TOPO1i doses Chart 23. High-DAR dpADCs exhibited favorable mouse PK, stable DAR, and controlled systemic payload exposure, mitigating historical safety concerns. Proprietary pan-PARPi payloads were not substrates for major efflux pumps, potentially reducing resistance. Data support dpADCs as a platform strategy to improve durability and therapeutic index in HRD and HRP solid tumors.

**Chart 23 - Site-Specific aTAA TOPO1i x PARPi dpADCs Outperform TOPO1i Single-Payload ADCs Across Varied DAR Formats**

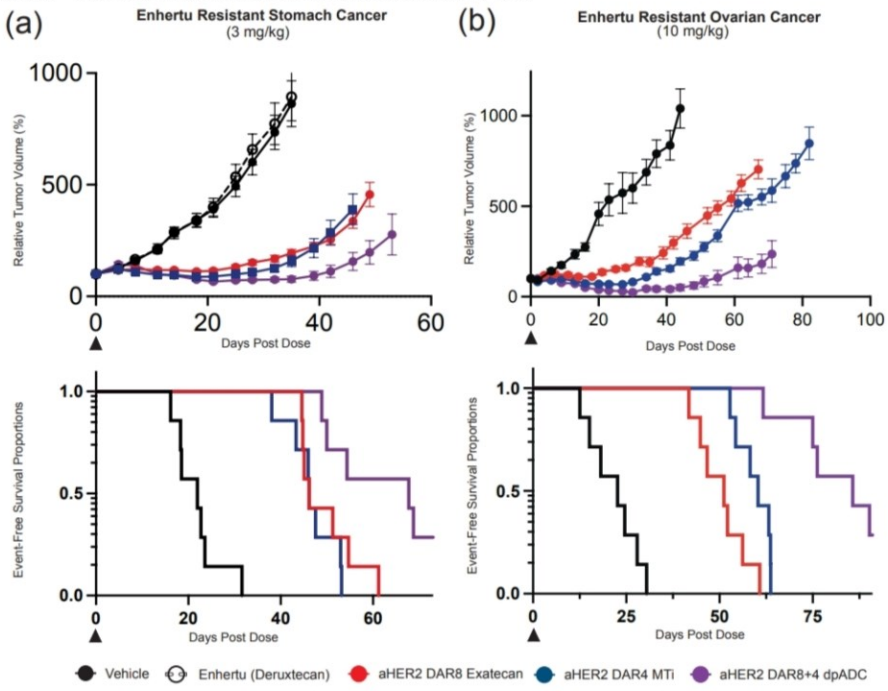



Source: AACR 2026

**Abstract #1685: HER2 Dual-Payload dpADC Overcomes Enhertu Resistance Preclinically**

Co's DAR12 HER2-targeting dual-payload ADC (Topo1i + microtubule inhibitor) demonstrated superior efficacy versus Enhertu and single-payload ADCs in Enhertu-resistant xenograft models. The dpADC induced tumor regressions and significantly delayed time-to-tumor-volume-quadrupling in gastric, ovarian, and breast cancer models where sequential TOPO1i and MTi exposure failed Chart 24. Head-to-head studies showed the dpADC outperformed both individual components and their co-administration, indicating benefit from synchronized intracellular payload delivery. PK studies showed stable DAR over 21 days, and NHP toxicology supported an HNSTD of ~12.5 mg/kg with manageable, transient neutrophil effects. Data suggest dual-payload HER2 ADCs can overcome sequential payload resistance that limits current HER2 ADC durability. The work positions dpADCs as a differentiated, mechanism-driven evolution beyond single-payload ADCs.

**Chart 24 - Sutro's dpADC is efficacious in Enhertu-Resistant CDX**



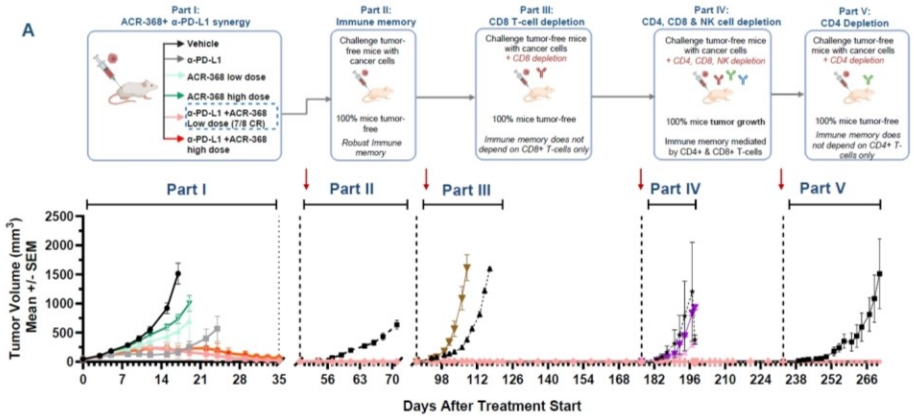

Source: AACR 2026

## ACRV

**Abstract LB152: ACR-368 synergizes with PD-L1 blockade by coordinated activation of adaptive and innate immunity pathways to achieve robust anti-tumor efficacy**

Nonclinical studies in the syngeneic MC38 colorectal model demonstrate robust synergy between ACR-368 (anti-CHK1/2) and anti-PD-L1, with the combination producing complete tumor regression in 7/8 mice, vs 74% TGI with ACR-368 monotherapy and CR in 2/8 mice with anti-PD-L1 alone Chart 25, and generating durable immune memory persisting >200 days across 4 serial tumor rechallenges. ACR-368 + anti-PD-L1 broadly enhanced innate and adaptive immune signaling and synergistically pushed macrophages towards a proinflammatory M1 state (iNOS+), with increased NK/CD8+ activity (Granzyme B) and reduced T cell exhaustion. Data overall support mechanistic rationale for ACR-368 combination with ICIs to augment anti-tumor activity.

**Chart 25 - ACR-368 reduces tumor volume in combo with PD-L1 inhibitor**




Source: AACR 2026

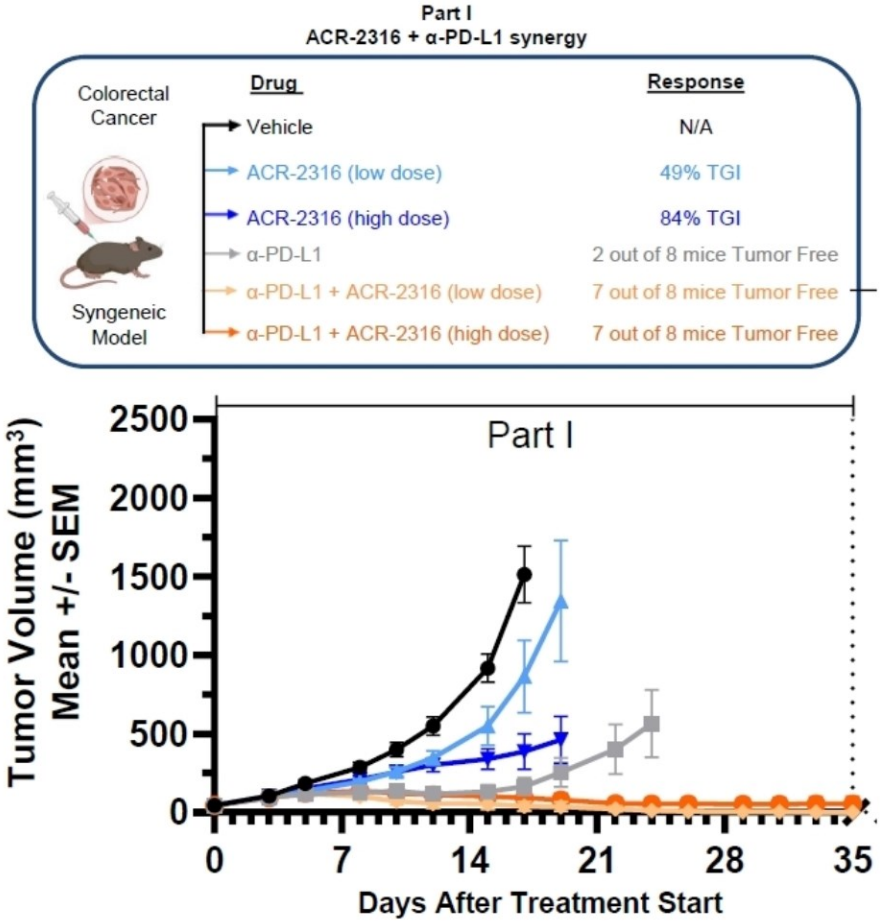
**Abstract 239: Potent Synergy Between CHK1/2 Inhibitor ACR-368 and the ADC Payload Topoisomerase I Inhibitor: Rationale for ADC + ACR-368 Combination Therapy**

Pathway analysis supported by *in vitro* experiments identify CHK1/2 inhibition as a mechanism for improving sensitivity to Topo1 inhibition, by preventing cell cycle checkpoint and DNA damage repair following Topo1 inhibitor-mediated replication fork collapse. ACR-368 + exatecan (Topo1 inhibitor) combination enhances apoptosis from replication fork collapse and improves cytotoxicity IC50 by ~100X in endometrial cancer cell lines. Data suggest potential for ACR-368 to strongly enhance activity of Topo1 ADCs.

**Abstract 3789: Treatment with ACR-2316, a potential first- and best-in-class WEE1/PKMYT1 inhibitor, combined with anti-PD-L1 induces complete tumor regression with durable immune memory in a preclinical syngeneic model**

ACR-2316, an oral WEE1/PKMYT1 inhibitor, demonstrates synergy with anti-PD-L1 in a syngeneic mouse tumor model, with the combination leading to CR in 7/8 mice, vs 49% TGI with low dose ACR-2316 monotherapy and CR in 2/8 mice with anti-PD-L1 alone Chart 26. Durable immune memory persisted >250 days. ACR-2316 + anti-PD-L1 led to reduced T cell exhaustion markers and increased CD8+ cytotoxicity vs. anti-PD-L1 alone, supporting improved anti-tumor activity with the combination.

Chart 26 - ACR-2316 reduces tumor volume in combo with PD-L1 inhibition



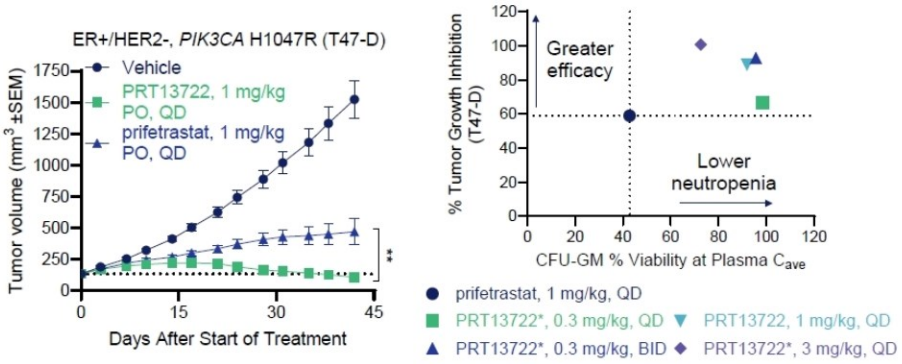

Source: AACR 2026

## PRLD

**Abstract 5793: First-in-class potent and selective oral KAT6A degrader development candidate, PRT13722, drives complete tumor regressions as a monotherapy with an improved preclinical hematological safety profile**

PRT13722, a KAT6A degrader in preclinical development by PRLD, induces deeper cellular perturbation (more differentially regulated genes) in a breast cancer cell model than non-selective KAT6A/B inhibition by prifetrastat. PRT13722 monotherapy drives complete tumor regression in KAT6A-amplified breast cancer xenograft mouse models; prifetrastat resulted only partial TGI Chart 27. Treatment with PRT13722 also reduced neutropenia 2X vs. prifetrastat at equivalent dose in this xenograft model. PRT13722 shows synergy with endocrine therapies and CDK4/6 and PI3Kα-inhibitors, yielding deeper regressions than partner drugs alone. Data overall point to superior anti-tumor activity and safety with PRT13722's selective KAT6A degrader mechanism.

Chart 27 - PRT13722 and prifetrastat evaluated in T47-D breast cancer xenograft mouse model

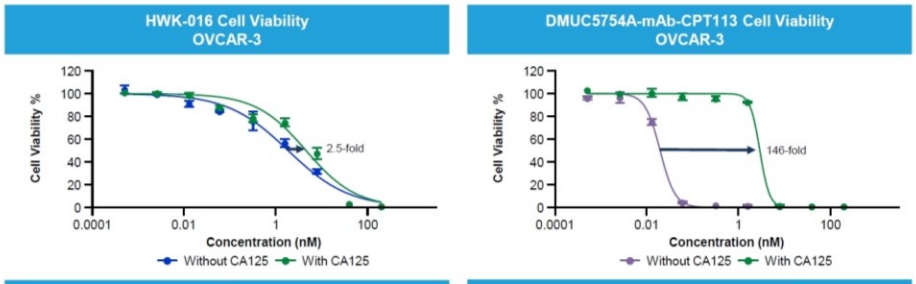



## WHWK

**Abstract 1324: Preclinical assessment of HWK-016, a next-generation, MUC16-targeting ADC with novel bioconjugation and linker-payload technology**

HWK-016, a MUC16-targeted ADC in Ph1 development, is designed to bind membrane-bound MUC16 to favor tumor delivery over targeting of circulating CA125 (shed MUC16 fragment). Cell assays show minimal interference from high CA125 on HWK-016 binding, internalization, and cytotoxicity vs. DMUC5754A, a previous MUC16 ADC developed by Genentech. However, DMUC65754A shows ~100X higher cell potency vs. HWK-016 in the absence of CA125 (0.02nM vs. 1.86nM) Chart 28, suggesting differences in drug payload release. Despite this, HWK-016 demonstrated superior tumor regression in ovarian cancer DCX and PDX models vs. DMUC65754A, with 4/7 CRs 108% TGI in OVCAR-3 and 1/8 CR 103% TGI in CTG-3423 Chart 29. HWK-016 was well tolerated in NHP with an HNSTD of 60mg/kg and showed high stability with $T_{1/2}$ of 10–13 days, supporting PK in trials.

Chart 28 - Cytotoxic activity of MUC16-targeting ADCs



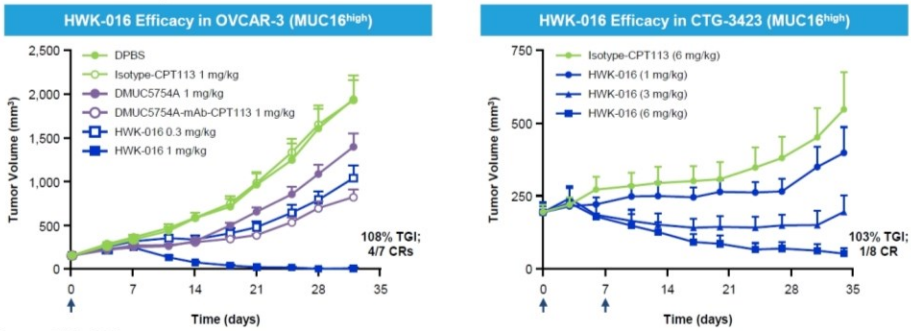

| CA125 Concentration | HWK-016 mAb $EC_{50}$ (nM) | Fold Change |
|---|---|---|
| 0 U/mL | 1.86 | N/A |
| 10,623 U/mL (304× ULN) | 4.57 | 2.5 |

| CA125 Concentration | DMUC5754A-mAb-CPT113 $EC_{50}$ (nM) | Fold Change |
|---|---|---|
| 0 U/mL | 0.02 | N/A |
| 10,623 U/mL (304× ULN) | 2.93 | 146.5 |

Note: In patients with cancer, high CA125 is defined as 2× ULN (>70 U/mL).

Source: AACR 2026

Chart 29 - Tumor regression in ovarian cancer CDX and PDX models

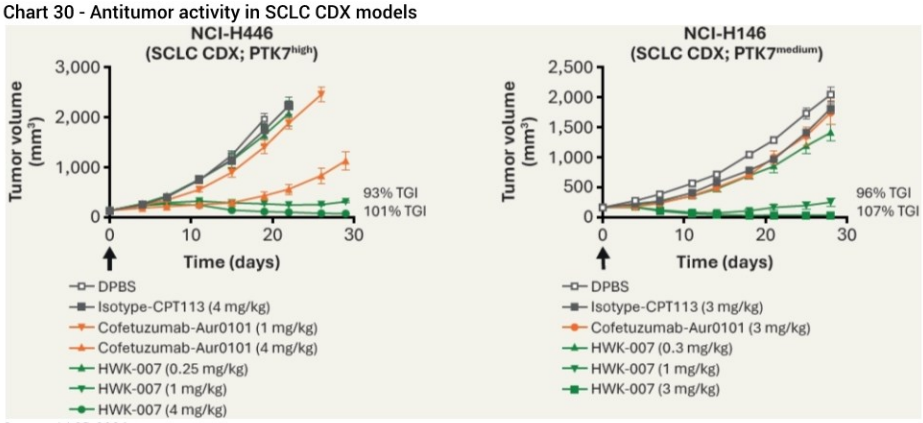



Source: AACR 2026

**Abstract 4439: Preclinical assessment of HWK-007, a next-generation, PTK7-targeting ADC with novel bioconjugation and linker-payload technology**

HWK-007, a PTK7-targeted ADC with a proprietary CPT116 Topo1 inhibitor payload, was evaluated vs. a previous PTK7 ADC, cofetuzumab pelidotin, across cancer cell line and DCX models. HWK-007 showed similar cellular internalization vs. cofe-p, but induced cytotoxicity in cancer cell lines (A204, NCI-H146, OVCAR-3) at greater potency, suggesting improved payload release or activity. HWK-007 also demonstrated stronger antitumor activity in SCLC CDX models vs. cofe-p, with 93-107% tumor regressions at 1 and 3 mg/kg Chart 30. HWK-007 showed good stability in human and cyno plasma, with <1% free CPT116 release, and was well tolerated in NHP (HNSTD of 60mg/kg) with a half-life of 9–11 days.

Chart 30 - Antitumor activity in SCLC CDX models

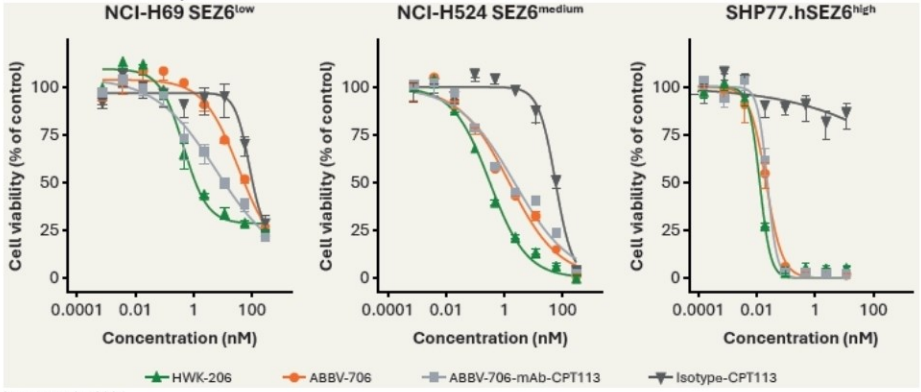



Source: AACR 2026

**Abstract 4440: Preclinical assessment of HWK-206, a next-generation, biparatopic, SEZ6-targeting ADC with novel bioconjugation and linker-payload technology**

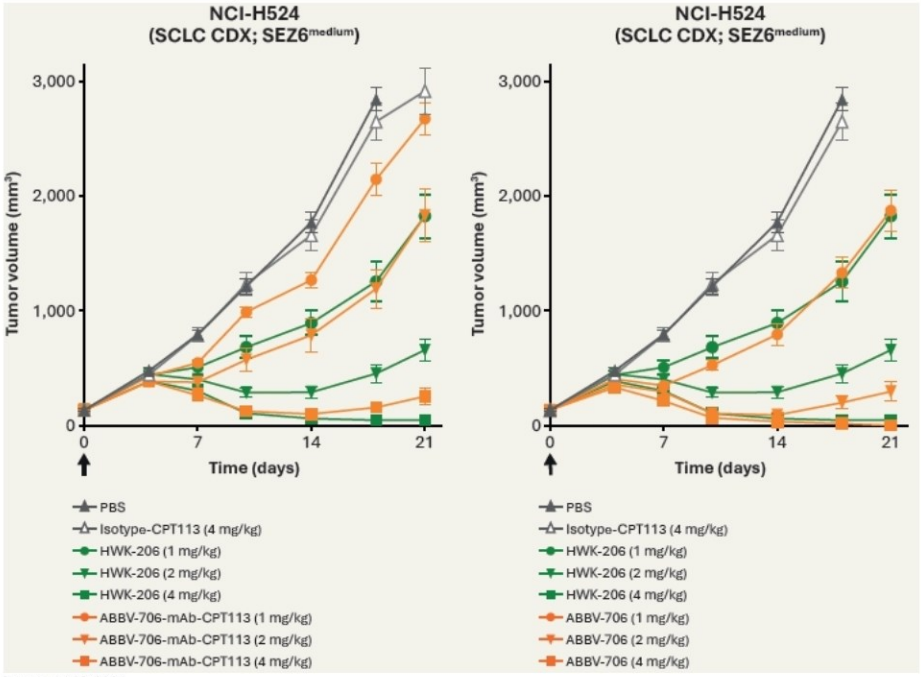
HWK-206 is a biparatropic ADC that binds 2 distinct domains on SEZ6 to release a proprietary CPT116 Topo1 inhibitor payload. HWK-206 shows stronger cell-viability inhibition vs. ABBV-706, a previous anti-SEZ6 exatecan ADC, with ~77X higher potency in cell lines with low SEZ6 expression Chart 31. In an SCLC CDX model, HWK-206 demonstrates greater antitumor activity vs. ABBV-706, but comparable activity vs. ABBV-706 mAB conjugated to CPT116 Chart 32, suggesting weaker cytotoxicity with CPT116 vs. exatecan. In NHP, HWK-206 is well tolerated (HNSTD of 60 mg/kg) and stable (half-life of 7–9 days).

Chart 31 - Cell viability inhibition in NCI-H69 cell culture




Source: AACR 2026

Chart 32 - Antitumor activity in SCLC CDX model

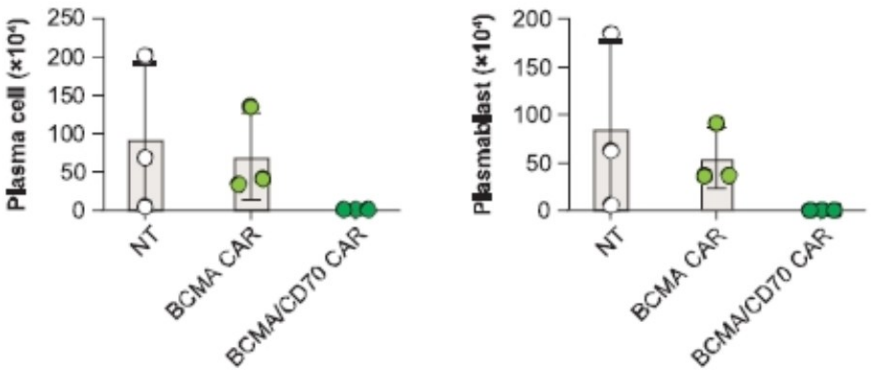



Source: AACR 2026

**Abstract 1535: BMCA/ CD70 dual CAR T cells show ability to overcome immune rejection, exhibit cytotoxic activity, and eliminate tumor cells in humanized mice.**

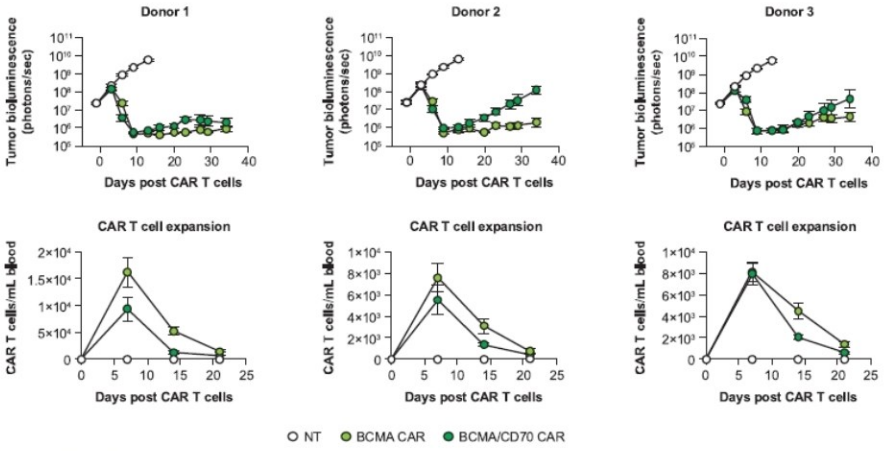
Allogene's BMCA/ CD70 dual CAR T is a gene-edited TRAC KO dual CAR construct designed to increase the durability and efficacy of CAR T therapies in oncology. CD70 CAR reduced allorejection pressure and killed activated host T cells to improve survival and persistence. Allogeneic BMCA/ DC70 Dual CAR T cells showed effective depletion of plasma cells in a humanized mouse model, superior to BCMA CAR alone (Chart 33). In a MOLP-8 in vivo mouse model, BCMA/ CD70 dual CAR T cells showed rapid CAR T cell expansion over 20 days and anti-tumor activity in MOLP-8 tumor xenografts from multiple donors superior to BMCA CAR alone across 40 days. (Chart 34).

Chart 33 - BCMA/ CD70 Dual CAR T Effect on Plasma Cells and Plasmablast Cells vs BCMA CAR Treatment




Source: AACR 2026

Chart 34 - BCMA/ CD70 Dual CAR T Effect on NSG Mice with MOLP-8 Tumor Xenografts




Source: AACR 2026

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**(Article 3(1)e and Article 7 of MAR)**

| | |
|---|---|
| Recommendation Published | April 22, 2026 20:46 P.M. |
| Recommendation Distributed | April 22, 2026 20:46 P.M. |

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- Allogene Therapeutics, Inc. (ALLO: $2.39, BUY)
- NURIX THERAPEUTICS, INC. (NRIX: $16.63, BUY)
- Nuvalent Inc (NUVL: $108.85, BUY)
- Prelude Therapeutics, Inc. (PRLD: $4.31, BUY)
- Sutro Biopharma, Inc. (STRO: $35.53, BUY)
- Whitehawk Therapeutics (WHWK: $4.23, HOLD)

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| | | | IB Serv./Past12 Mos. | | JIL Mkt Serv./Past12 Mos. | |
|---|---|---|---|---|---|---|
| | Count | Percent | Count | Percent | Count | Percent |
| BUY | 2173 | 61.82% | 364 | 16.75% | 109 | 5.02% |
| HOLD | 1182 | 33.63% | 102 | 8.63% | 16 | 1.35% |
| UNDERPERFORM | 160 | 4.55% | 1 | 0.62% | 1 | 0.62% |

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