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White Paper

Induced-Proximity Therapeutics

Targeted protein degradation (TPD)

has crossed a historic threshold. Proteolysis-targeting chimeras (PROTACs) finally delivered pivotal proof with vepdegestrant (ARV-471, Pfizer/Arvinas, VERITAC-2) positive data from a phase III trial in *ESR1*-mutant ER+/HER2– breast cancer versus fulvestrant (HR 0.57, p<0.001).[1] A decision from the U.S. Food and Drug Administration (FDA), anticipated in 2026, could establish PROTACs as clinically validated.[2]

But while PROTACs mature, the gravitational pull of the field is shifting toward molecular glues (MGs). Why? Molecular glues promise smaller, more drug-like molecules and simpler pharmacology, and the discovery process—once serendipitous—is now becoming systematic (**Table 1**). The deal flow tells the story: Monte Rosa Therapeutics, one of the leaders in rational glue design, initially entered a $2B collaboration with Roche focused on oncology, [3] followed by a second major agreement with Novartis for immunology and inflammation (I&I) totaling $5.7B.[4] Meanwhile, Roche is moving deeper into molecular glue space, with Genetech announcing another multi-year partnership with Orionis Biosciences for its Allo-Glue™ platform—potentially exceeding $2B—pivoting from a broad 2023 collaboration to a cancer-centric strategy.[5-6] These moves underscore a clear signal: molecular glues are no longer a curiosity—they are becoming a priority (**Figure 1**).

As the spotlight shifts from early proof-of-concept to a wave of clinical and strategic milestones, the real question is: how will these advances—across PROTACs, molecular glues, and other emerging approaches—reshape the therapeutic landscape? Which programs will set the pace for the next chapter of TPD?

BTK Degraders: The Next Frontier or a Tactical Reserve?

Few targets in the TPD space have captured attention like BTK, likely because of the prior success of BTK inhibitors (BTKi). The first wave of selective degraders—Nurix's NX-5948 and BeOne Medicines (formerly BeiGene) BGB-16673—are already rewriting expectations in early trials. In relapsed/refractory (post BTKi/BCL2i) B-cell malignancies, they are delivering response rates of 80–94%,[7-8] on par with what BTKi achieve in less resistant disease. However, these data are still early with durability remaining an open question as only BGB-16673 reports an initial PFS rate at 12m of 74%.[9] For reference, approved non-covalent BTKi pirtobrutinib showed 16.8m mPFS (~60% 12m PFS rate) in CLL patients post BTKi/BCL2i. [10]

For now, the logic of BTK degraders starting in later lines to tackle resistant mutations is sound: high unmet need, a clear regulatory path, and a compelling value proposition for a new modality. But what happens if (or when) these drugs succeed? Do they remain "salvage" therapies, or move forward to challenge BTKi head-to-head, finally providing the evidence for superiority of degradation over inhibition? Preclinical potency suggests they could compete, perhaps even surpass BTKi. On the other hand, from a strategic standpoint, preserving better therapy for later may provide patients with an effective option when they become refractory. Will degraders become a new backbone or remain the ace up the sleeve?

Mechanistic and Drug-like Properties

Features	PROTACs	Molecular Glues
Mechanism of Action	Heterobifunctional: Binds both the E3 ligase AND the target to induce proximity	Largely monovalent: Binds E3 ligase OR target protein to induce new or stabilize protein-protein interactions
Linker	Yes	No
Rule of Five	Beyond (not compliant)	Usually within
MW	700–1,500	200–600
RoA	Oral, IV	Oral

Strategic Assessment

Features	PROTACs	Molecular Glues
Key Advantages	(1) Complete inhibition of the target possible; (2) Targeting scaffolding function; (3) Potential to target undruggable targets • Easier to rationally design	• Low molecular weight • Good oral bioavailability • Better cell permeability • Binding pocket not essential
Key Limitations	Targets with high turnover are difficult to degrade • High molecular weight • Low oral bioavailability • Low brain penetration	• Unpredictable target may induce toxicity • Limited rational design

Table 1. Comparison Between PROTACs and Molecular Glues. Key differences and similarities

TPD: Magic Bullet or Precision Tool?

Direct comparisons between degraders and inhibitors remain one of the field's most debated questions. Where degradation does not offer an obvious edge—think proteins without scaffolding roles and broad interaction surfaces, or targets already druggable—does the modality truly add value? The community's high expectations met initial disappointment earlier this year when Arvinas/Pfizer's ER degrader, vepdegestrant (ARV-471), failed to meet its primary endpoint in the ITT population of ER+/HER2– breast cancer.[1] For some, this was a sobering moment. ARV-471 benefit was observed only in *ESR1*-mutant disease (*ESR1* gene encodes for ERα), where constitutively active ER remains the dominant oncogenic driver, i.e., conformational changes in mutated *ESR1* lead to the reduced binding of inhibitors, increased coactivator recruitment, and enhanced proteolytic stability, inducing resistance.[11] By degrading ER rather than merely inhibiting it, the drug can overcome mutation-driven resistance.

Yet VERITAC-2 tells a more nuanced story. First, it marks a historic milestone: the first PROTAC to succeed in a phase III trial. Second, it tempers the "one-size-fits-all" hype. Even a potent degrader must align with tumor biology—here, ESR1 mutation status—to outperform standards.

But also, could we truly state that TPD "fallen short of hype"? Maybe the hype just needs calibration. The promise was sweeping: undruggable targets, broad efficacy, mutation resistance. The reality is likely more targeted and context-dependent: a focused win in ER+ breast cancer, contingent on a specific mutation. This does not diminish the platform—it refines our understanding of where its strengths and weaknesses lie. And let's not forget: the comparator, fulvestrant, is itself a degrader, a SERD, making this more of a comparison between two degradation strategies—not a true head-to-head with an inhibitor.

Ultimately, TPD should complement existing modalities rather than replace them, broadening the therapeutic toolkit to tackle biology where other approaches fall short. This could include unlocking

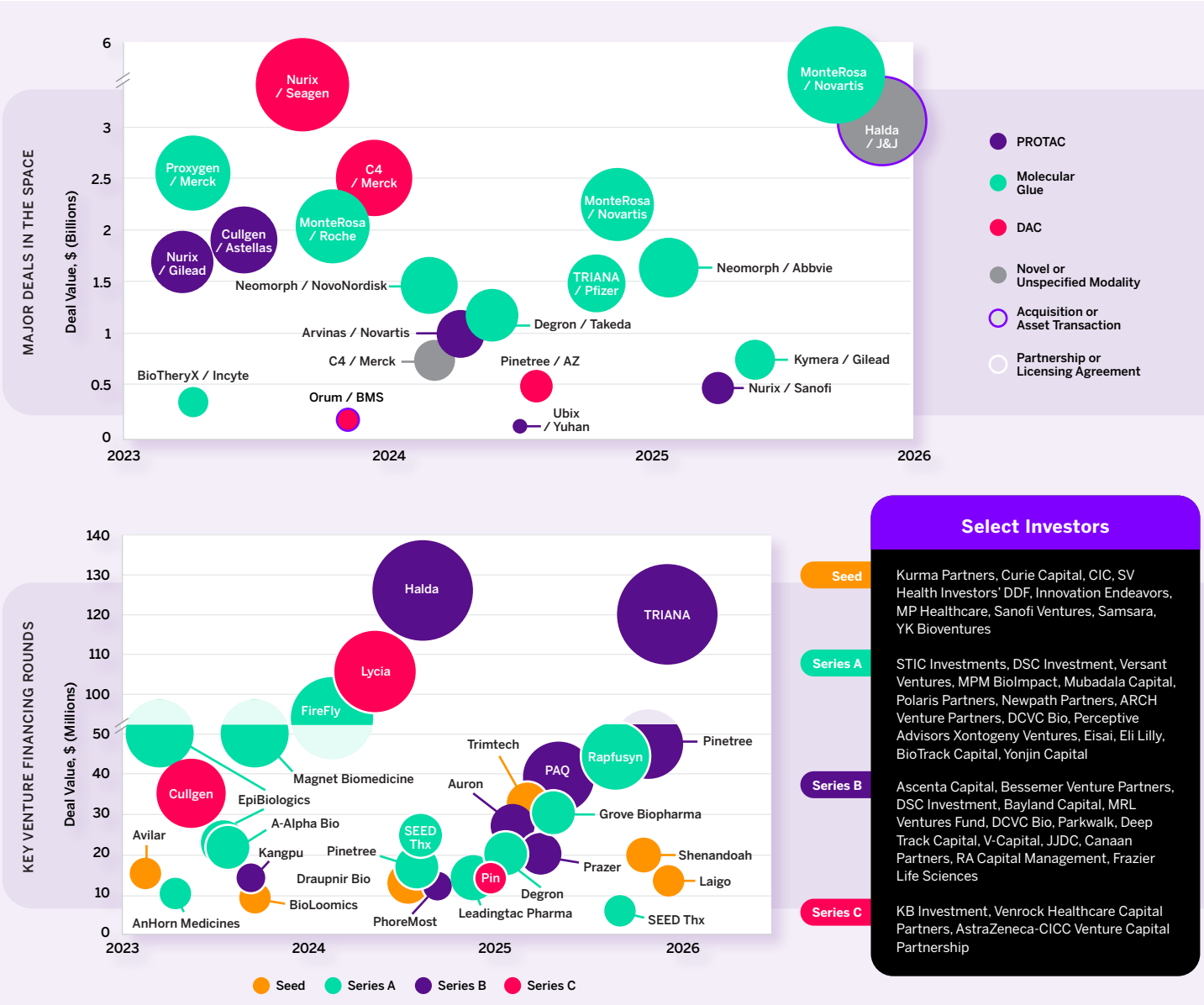


Figure 1: Top Deals between 2023 and 2026. The deal value indicates the maximum amount a company is eligible to receive under the agreement. In case of venture financing (VC), it reflects the funds raised during given investment round at a given time. VC deal values ($ M): Seed (8.7–21.5), Series A (10–94), Series B (12–126), Series C (13.8–106). Select investors from recent funding rounds are listed on the bottom right. GlobalData filters applied include Deal Type: Merger & Acquisitions, Strategic Alliance, Venture Financing. **DAC:** degrader antibody conjugates; **TPD:** targeted protein degradation

previously inaccessible targets such as transcription factors and intrinsically disordered proteins, which have historically resisted small-molecule inhibition—this promise remains to be established in the clinic.

Resistance: From Wishful Thinking to Reality Check

When TPDs first entered the spotlight, the hope was almost utopian: by erasing the protein rather than merely blocking its activity, could we finally outsmart resistance? Biology had other plans.

Resistance is not just possible—it is already here. Long-term lenalidomide use taught us tumors can mutate CRBN impairing degradation.[12] And that is not the only route. Targets themselves can change—mutations or splice variants can strip away the degrader's binding site. Clinical evidence now extends this to BTK degraders. In 2024, a patient on BGB-16673 developed a BTK A428D mutation in the kinase domain, conferring resistance. Structural modeling suggests this would also likely impact Nurix's BTK/IKZF degrader NX-2127.[13]

But this does not mean TPD has lost its excitement, it just simply shifts the narrative: degraders, like inhibitors, exert evolutionary pressure, and tumors adapt. These insights reframe the challenge—moving from hype to strategy: anticipating resistance, expanding ligase diversity, and designing smarter combination approaches.

Beyond Cancer: TPD's Expanding Horizon

TPD is no longer confined to oncology (**Figure 2**). PROTACs and molecular glues are rapidly moving into immunology, neurology, dermatology, and beyond. One of the earliest signals came from IRAK4 degradation. Kymera's KT-474, developed under its collaboration with Sanofi, advanced into phase II for atopic dermatitis (AD) and hidradenitis suppurativa. Yet Sanofi recently pulled back, prioritizing a more potent and selective IRAK4 degrader still in preclinical development.[14] This has not slowed the field: Nurix and Gilead's GS-6791/NX-0479 announced IND clearance for phase I in healthy volunteers,[15] and BeOne's BGB-45035 is now starting in phase II vs placebo for rheumatoid arthritis in China. Other programs are pushing oncology boundaries: Nurix's BTK degraders are moving into autoimmune indications, while Monte Rosa explores VAV1 and NEK7 for inflammatory diseases.

STAT6 is emerging as a compelling example of TPD's expansion beyond oncology. Kymera's KT-621 leads the charge in phase II.[16] While STAT6 may seem underexplored, the JAK/STAT pathway is central to Th2-driven inflammation. In atopic dermatitis (AD), JAK inhibitors deliver high response rates but carry black box safety warnings. Could STAT6 degradation replicate JAKi efficacy while reducing toxicity? Early clinical data presented in December 2025 in moderate-to-severe AD reported mean EASI (Eczema Area and Severity Index) reduction of ~60% and P-NRS reduction of 47%—on par if not better than dupilumab at week 4—and earlier findings hinted at a better safety profile than JAKi, though patient numbers remain small.[17] The possibility of STAT6 degradation challenging dupilumab is becoming more real. Investor confidence underscores the momentum: Sanofi recently exercised a license extension option for Nurix's STAT6 program, paying $15M upfront, with up to $465M in potential milestones.[18]

TPD Meets the Brain: Breaking Barriers, Raising Stakes

For years, the blood–brain barrier was seen as a brick wall for large molecules like PROTACs. Yet recent data suggest the wall may be cracking. Arvinas' LRRK2 degrader, ARV-102, provides one of those examples. Preclinical NHP models showed "deep brain" penetration, and now early human data confirm CNS engagement: dose-dependent LRRK2 reduction in CSF from healthy volunteers and CSF accumulation in patients with Parkinson's disease.[1] Additionally, Nurix's BTK degrader (NX-5948), also designed for brain penetration, opens the door to treating diseases with a CNS component. Very early data suggests this is likely more than theoretical, reporting initial responses in CLL/SLL patients with CNS involvement.[19]

Moreover, as the field shifts toward smaller molecular glues, CNS penetration is becoming even less of a barrier. For example, Monte Rosa initially reported their

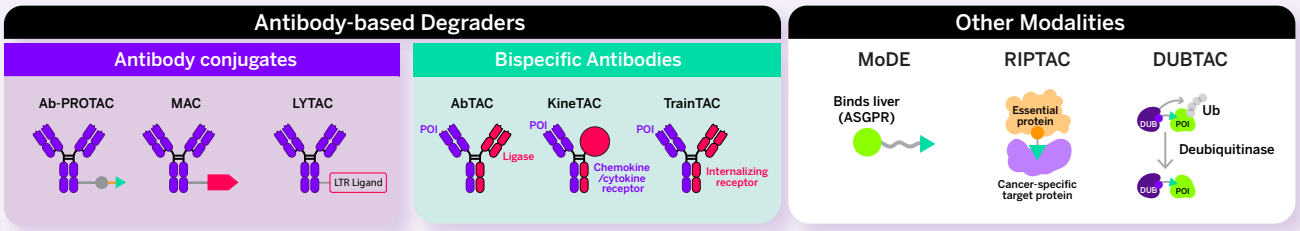
Figure 2: Development in the Oncology and Ex-oncology Space. Pipeline categorized by degrader type (left) and target (right), according to GlobalData (Drug Descriptor: "Proteolysis Targeting Chimeras," "Targeted Protein Degradation"). Oncology pipeline by target: "Other" group includes but not limited to the modalities targeting BCL2L1, BRD4, EGFR, MET, SNF2L2, STAT3, MYC, CDK2, CDK9. Ex-oncology pipeline by target: "Other" group includes but not limited to the modalities targeting ATE1, BRD9, CD47, CDK2, EGFR, GSK3b, GSPT1/IL3RA, Huntingtin, KRAS, STAT3, TDP43, TYK2, Ataxin 3 and E3 ligase or UPS activators. **UCH:** Ubiquitin Carboxyl Terminal Hydrolase; **DUB:** Deubiquitinating Enzyme

NEK7 MG (MRT-8102) to show robust NEK7 knockdown in brain tissue, translating into reduced IL-1β and IL-6 in LPS-induced neuroinflammation models. Now, interim results of the first-in-human phase I study also describe reduced IL-6 levels in CSF in two subjects, consistent with CNS penetration.[20]

The takeaway? CNS was once considered off-limits for degraders, especially larger PROTACs. Evidence is stacking up that this is no longer theoretical. If these programs succeed, it will not just expand TPD's reach but rewrite the rules of what these modalities can do.

What's Next for TPD?

The next chapter of TPD is not just about new targets and new indications—it is about new modalities that redefine what can be drugged and how (**Figure 3**). The field is moving beyond intracellular proteins to extracellular and membrane targets, leveraging alternative cellular pathways like lysosomal-mediated degradation, and other delivery systems such as antibodies or peptides.



Degraders	Target Location	Composition	Degradation Pathway
Ab-PROTAC	Intracellular	Ab conjugated to PROTAC	Proteasomal
MAC	Intracellular	Ab conjugated to molecular glue	Proteasomal
LYTAC	Extracellular/membrane	Ab conjugated to LTR ligand	Lysosomal
AbTAC	Membrane	bsAb: Binding to POI and membrane-bound E3 ligase	Proteasomal, Lysosomal
KineTAC	Extracellular/membrane	bsAb: Binding to POI and chemokine/cytokine receptor	Lysosomal
Internalizing receptor binder/TrainTAC	Extracellular/membrane	bsAb: Binding to POI and internalizing receptor	Lysosomal

Modality		Company	Deal
Antibody-based Degraders	Antibody Conjugates	Nurix	$3.4B from Seagen
		C4	$2.5B from Merck
		Lycia	$107M Series C
	Bispecific Antibody	EpiBiologics	$73M total in Series A
Other Modalities	RIPTAC	Halda	Bought out by J&J for $3B
	DUBTAC	Stablix	Vertex collaboration

Figure 3: Novel Platforms in TPD Space and Beyond. Antibody-based degraders are categorized into two groups: antibody conjugates (left) and bispecific antibodies (middle). The accompanying table provides further information distinguishing these two categories. Additional modalities of interest are shown on the right. Some of the key companies involved, along with significant deals and funding raised, are emphasized. **Ab-PROTAC** refers to antibody-conjugated PROTACs; **DAC** stands for degrader-antibody conjugate; **LTR** is lysosome-targeting receptor; **LYTAC** means lysosome-targeting chimera; **MAC** denotes molecular glue-antibody conjugate; **POI** represents protein of interest; **MoDE** is molecular degrader of extracellular protein; **RIPTAC**, regulated induced proximity targeting chimera, **DUBTAC**, deubiquitinase-targeting chimeras

Technologies that enable targeting proteins beyond the intracellular space are prime examples of these novel formats. Among the most promising are LYTACs, pioneered by Nobel laureate Carolyn Bertozzi's lab and now advanced by Lycia Therapeutics (backed by Lilly), LYTACs recruit lysosomal machinery to clear extracellular proteins. With a $106M Series C,[21] Lycia aims to enter the clinic with LYTACs for autoimmune and inflammatory diseases—potentially offering degraders to deplete soluble factors like autoantibodies or cytokines, targets previously untouchable by degraders. Another move comes from Biohaven's MoDE™ and TRAP™ platforms. Its lead auto-injectable IgG degrader, BHV-1300, achieved up to 83% IgG reduction in phase I and is planning to advance to phase II for Graves' disease.[22] Antibody-based approaches are also gaining traction with companies like EpiBiologics and Laigo Bio developing degrader bispecific antibodies—one arm binds the target (often cell-surface or circulating), the other recruits an endogenous receptor to mediate degradation. These strategies potentially blur the line between biologics and TPD.

Another exciting frontier lies in E3 ligase/target matching. Most clinical degraders rely on CRBN or VHL, but the human genome encodes more than 1,000 E3 ligases with tissue-specific expression.[23] Choosing the right ligase could improve tissue selectivity potentially unlocking stronger degradation. This is an area ripe for innovation.

Beyond Degradation: Induced Proximity Evolves

The concept of induced proximity is proving remarkably versatile. It is spawning a new generation of "TACs" that go beyond degradation. DUBTACs recruit deubiquitinases to stabilize proteins rather than destroy them, while RIPTACs create an interaction between tumor-specific protein and a protein with essential function, leading to cancer cell death. Halda Therapeutics, which entered the clinic in February 2025 with their AR/BRD4-targeting RIPTAC for prostate cancer, attracted a lot of attention after recent acquisition by J&J for $3.05B in cash.[24] These approaches hint at a future where induced proximity becomes a broad therapeutic principle, not just a degradation trick.

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Closing Thoughts

After the initial surge of hype, the TPD field has entered a phase of maturity. Expectations have been recalibrated: degraders are not magic bullets, but precision tools whose success likely depends on biology, patient selection, and smart positioning. Yet the potential remains vast. We now have initial clinical proof, expanding target classes, and a pipeline pushing into I&I, CNS, and even extracellular spaces. A few programs to watch in 2026 include readouts in ex-oncology space with Arvinas' LRRK2 degrader in Parkinson's disease, Monte Rosa's NEK7 in healthy volunteers with elevated cardiovascular risk—and, of course, the potential first-in-class approval of ARV-471 in ESR1-mutant ER+/HER2– breast cancer.

As the field moves forward the questions that remain are not just scientific, they are strategic. Will molecular glues deliver on their promises? Can ligase diversity unlock tissue selectivity? Will induced proximity evolve into a universal principle? These questions will shape portfolios, partnerships, and therapeutic paradigms for the next decade. TPD is no longer a speculative bet, it is a platform in motion. Where it ultimately lands—cornerstone or complement, oncology or multi-system—remains undefined as clinical data and mechanistic insights continue to shape its trajectory.

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