AcademicLabs Report on Rising Stars in Protein Degraders

1. Introduction and Background

This report evaluates 50 researcher profiles for one specific business purpose: identifying emerging external leaders in **protein degraders** who are most relevant for collaboration, consulting, co-development, or adjacent strategic engagement. The emphasis is therefore not on general scientific excellence alone, but on a combination of **direct targeted-protein-degradation relevance, differentiated capability, translational trajectory, and practical partnering utility**.

The field itself is broadening rapidly. What was once dominated by classical bifunctional PROTACs now includes **molecular glues, covalent glues, bioPROTACs, lysosome-directed chimeras, autophagy-enabled degradation concepts, E3-expansion strategies, programmable peptide-based degraders, and increasingly sophisticated delivery or spatiotemporal-control systems**. This expansion is clearly reflected in the cohort. The strongest profiles are no longer only those making another degrader against an established target; they are increasingly those who can:

- open new target classes,
- exploit underused E3 ligases,
- solve delivery and selectivity problems,
- generate translationally credible in vivo data, or
- build enabling platforms that increase the odds of success across programs.

At the same time, a large fraction of the cohort sits in the adjacent but strategically important zone of **ERAD, autophagy, proteasome biology, degron discovery, lysosomal trafficking, and protein-homeostasis regulation**. These researchers are often less suitable for immediate asset-centric co-development, but they can still be highly valuable for mechanism, resistance biology, target-space expansion, and biomarker strategy.

The comparative ranking in **Chapter 3** already harmonized the individual evaluations into five conserved criteria: recent momentum, differentiated niche, visibility/reputability, translational signal, and external validation. The chapters below build on that ranking and convert it into a practical decision framework: **how to interpret the scores, how to tier the field, which Tier 1 profiles are the best near-term fits, what patterns are emerging across the landscape, and what actions should follow**.

A final methodological note is important. The underlying evidence is uneven across profiles. Many records provide strong publication and collaboration signals but **limited explicit evidence on authorship leadership, grant capture, awards, or independence**. Consistent with the profile-level instructions, judgments in this report are therefore intentionally conservative where evidence is limited, and direct degrader relevance is weighted more heavily than broader proteostasis relevance for this specific mandate.

2. Evaluation Framework and Scoring Legend

2.1 Framework used across profiles

To maintain consistency with the underlying profile reviews and the ranking table in Chapter 3, each profile was assessed against five criteria:

Criterion	Core question	What a high score means	What a low score means
Momentum	Is there strong, recent activity in protein degraders or directly relevant adjacent science?	Dense, current 2024–2026 activity with clear continuity and multiple relevant outputs	Sparse, older, or only indirectly relevant work
Niche	Is the researcher differentiated in a strategically useful way?	Distinct platform, modality, E3-ligase angle, target class, or enabling biological specialty	Broad but undifferentiated work or weak relevance to degrader strategy
Visibility	Are the outputs credible, visible, and field-relevant?	Reputable, high-interest outputs with strong methodological or conceptual weight	Limited evidence of field visibility or mainly lower-signal outputs
Translational signal	Is there evidence of movement toward practical application?	In vivo activity, disease-relevant models, patents, lead-like chemistry, delivery, or platform deployability	Mostly mechanistic/basic work with limited partnering readiness
Validation	Is there evidence of external credibility?	Competitive funding, strong collaborations, patents, industry links, or signs of independence	Limited evidence on grants, honors, leadership, or external uptake

2.2 Scoring legend

The profile-level scores use a **1–10 scale** and should be interpreted as follows:

- **1–3:** weak or highly indirect fit; limited evidence for this mandate
- **4–6:** moderate or mixed fit; scientifically credible but with clear gaps
- **7–8:** strong fit on the criterion; good evidence with some remaining uncertainty
- **9–10:** exceptional fit on the criterion; unusually strong and decision-relevant evidence

2.3 Overall fit legend

The overall labels used throughout the report are qualitative summaries of the five criteria and are **not purely arithmetic**:

- **Excellent:** direct degrader relevance plus repeatedly high scores across momentum, niche, and translational/partnering value
- **Good:** strong and decision-relevant, but with one or two material limitations such as unclear independence, earlier translation, or narrower visibility
- **Ok:** scientifically credible, but either more degrader-adjacent than direct, or limited by weak leadership/validation evidence
- **Poor:** limited recent direct relevance to the protein degrader mandate

2.4 How scores were interpreted for this report

For this company use case, scores were interpreted through a **partnering lens** rather than a purely academic lens. In practice, that means:

1. **Direct targeted-protein-degradation relevance was prioritized** over broader degradation biology when ranking near-term external targets.
2. **Translational evidence mattered disproportionately**, especially in vivo efficacy, modality/platform deployability, and patent or industry-facing signals.
3. **Novel E3-ligase or modality expansion** was treated as strategically valuable because it can diversify future discovery options.
4. **Evidence limitations were handled conservatively**. When grant, authorship, or leadership evidence was not available, scores for validation/independence were kept lower rather than inferred.

This framework is especially important when comparing chemistry-led degrader builders with biology-led enablers. Both can be valuable, but they are valuable in **different ways** and should not be engaged through the same business model.

3. Comparative Ranking of All Evaluated Profiles

The table below harmonizes the individual profile evaluations into five conserved criteria: **Mom** (recent momentum), **Nich** (differentiated niche), **Vis** (visibility/reputability of outputs), **Trans** (early translational signal), and **Val** (external validation, collaboration, and—where available—funding/independence).
Ranking prioritizes direct protein-degrader relevance and near-term partnering utility, not just arithmetic averages; biology-enabling but less direct degrader profiles were therefore ranked somewhat lower even when scientifically strong.

Highest-priority external tracking pool: Shipeng He, Pranam Chatterjee, Wenchao Lu, William Farnaby, Hojong Yoon, Woong Sub Byun, Ling Xie, George M. Burslem, Baishan Jiang, Nicole Trainor.

Rank	Researcher	Fit	Mom	Nich	Vis	Trans	Val	Best-fit opportunity / concise justification	Key citation
1	**Shipeng He**	Excellent	10	10	9	9	6	Precision degradation platforms; unusually dense output with strong in vivo efficacy.	HerTACs (2025)
2	**Pranam Chatterjee**	Excellent	9	10	8	9	8	AI-designed peptide degraders/stabilizers; differentiated platform with in vivo/LNP and patents.	Programmable β-catenin degraders (2024)
3	**Wenchao Lu**	Excellent	9	9	8	9	6	Strong degrader/glue discovery chemistry with durable preclinical signals.	TEAD degrader (2025)
4	**William Farnaby**	Good	8	9	9	9	8	Structure-guided degraders for KRAS/CNS targets; strong translational and network profile.	Pan-KRAS degrader (2024)
5	**Hojong Yoon**	Good	8	9	7	8	9	Molecular-glue discovery and chemoproteomics; clear NIH-backed independence trajectory.	CRBN glue interactome (2025)
6	**Woong Sub Byun**	Good	9	9	8	7	8	Rational glue/covalent degrader design across noncanonical E3s; very concentrated recent momentum.	BRD9 glue degrader (2025)
7	**Ling Xie**	Good	9	9	8	8	6	Broad next-gen modality innovator: TF-PROTACs, bridged PROTACs, DUBTACs.	AURKB degrader (2025)
8	**George M. Burslem**	Good	8	8	7	9	8	Induced proximity, bioPROTACs, and glue translation; especially attractive for platform collaboration.	LRF degrader (2025)
9	**Baishan Jiang**	Good	9	8	8	9	6	Kinase degrader/glue chemistry with strong PK/xenograft evidence and IP.	Selective CDK6 degrader (2026)
10	**Nicole Trainor**	Good	8	8	9	9	6	Strong collaborative programs in KRAS/SMARCA/TEAD degraders with patent support.	KRAS(on) degrader (2025)
11	**Scott J. Hughes**	Good	8	9	8	9	6	Mechanistic innovator in targeted glues/PROTAC architecture; independence evidence remains limited.	BRD9 targeted glue (2025)
12	**Dean Clift**	Good	8	9	7	9	6	TRIM21/TRIMTAC state-selective degradation; differentiated niche with strong patenting.	TRIMTACs (2025)
13	**Xing Qiu**	Good	8	9	7	8	6	Nontraditional nucleotide/TF-directed degraders plus anti-cancer degrader patents.	LSD1 degrader (2025)
14	**Zhengnian Li**	Good	8	9	7	8	6	Distinctive antiviral degraders plus WEE1 glue biology and granted IP.	Dengue capsid degraders (2026)
15	**Justin M. Reitsma**	Good	8	8	7	8	6	Valuable upstream collaborator on E3 evaluation, ternary analysis, and degradable-proteome expansion.	In-cell E3 evaluation (2025)
16	**Matthias Hinterndorfer**	Good	8	9	8	8	6	Strong proximity-pharmacology profile beyond CRBN/VHL; good E3-expansion angle.	High-throughput CIPs (2026)
17	**Kheewoong Baek**	Good	8	9	8	7	6	First-author CRBN interactome work and strong glue/E3-biology positioning.	CRBN glue interactome (2025)
18	**Wenyi Mi**	Good	8	9	7	8	5	New E3 recruitment strategies (GID4, ZYG11B); structurally grounded platform potential.	GID4 PROTACs (2025)
19	**Vincenzo D'Angiolella**	Good	8	9	8	7	6	KBTBD4/UM171 molecular-glue mechanism expert; especially strong for E3 biology collaborations.	UM171/KBTBD4 glue mechanism (2025)
20	**Ryan D. Baldridge**	Good	8	9	7	8	7	ERAD degrons and membrane-target degradation biology; nonclassical but strategically useful.	ER-lumen degrons (2024)
21	**Matthew E. R. Maitland**	Good	8	9	7	7	6	Alternative E3 ligases (CTLH/GID4/FBXO22) plus PROTAC specificity tools.	FBXO22–NSD2 degradation (2024)
22	**Martin Peter Schwalm**	Good	8	9	7	7	6	Strong assay/mechanism/quality-control partner for PROTAC and autophagy degrader programs.	Rapid PROTAC discovery workflow (2026)
23	**Jiwon Hwang**	Good	8	9	7	8	6	ER-lumen degron discovery expands targetable biology; enabling rather than chemistry-led.	ER-lumen degrons (2024)
24	**Jonathan St. Germain**	Good	7	8	7	8	6	ProtacID and FBXO22 recruitment make him useful for MOA de-risking.	ProtacID (2025)
25	**Ka Yang**	Good	8	8	7	8	7	Chemoproteomics plus dual degrader/E3-expansion work; good enabling-translational mix.	MDM2/GSPT1 dual degrader (2025)
26	**Huilun Wang**	Good	8	9	8	8	8	High-quality ERAD disease biology; strong science but indirect to canonical degrader discovery.	SEL1L rescue via ASO (2026)
27	**Ting-Wei Mu**	Good	7	8	7	8	8	ER-phagy/CNS proteostasis specialist; attractive biology collaborator, less direct TPD fit.	GluN2B autophagy (2026)
28	**Florian Wilfling**	Good	7	9	8	5	8	Selective-autophagy degradability rules; strong enabling biology, limited direct degrader modality work.	Autophagy initiation hubs (2025)
29	**Xiaoqiong Wei**	Good	7	8	7	7	6	ERAD substrate/disease biology with occasional TPD adjacency; indirect but credible.	ERAD substrate proteomics (2024)
30	**Siavash Vahidi**	Good	8	9	8	7	6	ClpP/ClpXP structural degradation machinery; mechanistically strong but adjacent to canonical TPD.	ClpP activation mechanism (2025)
31	**Yongwang Zhong**	Good	7	8	6	7	6	ERAD/ubiquitin small-molecule modulation; useful biology-led opportunity, less direct platform fit.	RNF5 inhibitor/degrader (2022)
32	**Hong Yue**	Ok	8	7	8	8	5	Strong contributor on glue/E3 discovery studies; leadership/independence evidence limited.	CRBN glue interactome (2025)
33	**Vinay V. Eapen**	Good	7	8	8	7	6	Selective autophagy and lysosomal degradation in inflammation/immunology; noncanonical relevance.	WSTF nuclear autophagy (2025)

Rank	Researcher	Fit	Mom	Nich	Vis	Trans	Val	Best-fit opportunity / concise justification	Key citation
34	**Ryan J. Lumpkin**	Good	8	8	7	7	6	DCAF16/CRBN glue chemoproteomics and ubiquitylation mapping; solid enabling profile.	E-STUB ubiquitylation mapping (2024)
35	**Shoshiro Hirayama**	Good	8	8	7	6	7	Proteasome/microaggrephagy/co-translational QC; funded enabling biology, not chemistry-led TPD.	Microaggrephagy (2025)
36	**Jun Hamazaki**	Good	6	8	8	5	6	Proteasome regulation and PROTAC-response biology; useful for combination/resistance questions.	Intrinsic signaling modulates TPD (2024)
37	**Yuanzhong Wu**	Good	7	8	7	8	5	Cancer E3/substrate turnover with translational links; indirect to degrader chemistry.	ASB7/SUV39H1 degradation (2025)
38	**Alexandra Stolz**	Good	7	8	7	6	8	ATTEC/LC3-GABARAP validation and screening infrastructure; enabling collaborator more than asset owner.	LC3/GABARAP ligands (2024)
39	**Yulia Jitkova**	Good	7	8	6	8	5	Mitochondrial ClpP/ClpXP AML biology; strong science, limited independent-lead evidence.	ClpXP phospho-substrates (2025)
40	**Yuuki Fujiwara**	Good	6	8	7	6	6	SIDT2/RNautophagy lysosomal uptake biology; differentiated but not drug-like degrader focused.	SIDT2 protein uptake (2020)
41	**Koraljka Husnjak**	Ok	6	7	8	6	5	Strong ubiquitin/E3 scientist; selective recent degrader exposure rather than sustained TPD leadership.	Intramolecular bivalent glues (2024)
42	**Qian Chen**	Ok	5	8	7	7	5	Plant ERAD/E2-E3 turnover expert; mechanistically relevant, low pharma degrader proximity.	Potato E3-mediated turnover (2026)
43	**Nico Dissmeyer**	Ok	6	8	7	4	6	Plant N-degron specialist; valuable mechanism knowledge, limited therapeutic degrader translation.	PRT1 N-degron (2025)
44	**Vishwajeeth Pagala**	Ok	6	7	6	6	5	Proteomics-heavy contributor with one notable PROTAC repurposing study; leadership evidence limited.	PXR degrader repurposing (2025)
45	**Mauricio Andres Torres-Gutierrez**	Ok	4	7	7	6	5	Tissue ERAD/autophagy biology is relevant but largely degrader-adjacent.	ERAD–autophagy crosstalk (2026)
46	**Rachel Byerley Reinert**	Ok	4	7	6	4	8	NIH-backed endocrine ERAD/autophagy program; strong biology, indirect to TPD partnering.	NIH K08 ERAD project
47	**Neha Shrestha**	Ok	4	7	6	3	5	ERAD/autophagy in islet proteostasis; adjacent biology with limited direct degrader evidence.	α-cell ERAD paper (2026)
48	**Hongsheng Lin**	Ok	4	6	5	6	3	Neuronal ubiquitination/stability biology around spastin; not a direct degrader profile.	Spastin ubiquitination (2025)
49	**Eileithyia Swanton**	Poor	2	7	6	5	5	Strong ERAD/proteostasis background, but weak recent direct degrader momentum.	HFpEF proteostasis (2025)
50	**Yoichiro Harada**	Poor	1	7	6	2	4	Glycan quality-control specialist; minimal evidence for degrader-specific opportunity.	Mannose pathway QC (2026)

Brief decision takeaways - **Best near-term collaboration / consulting targets:** ranks **1–10**, especially where the need is direct degrader discovery, modality innovation, and preclinical translation. - **Best enabling-biology targets:** Baldridge, Hwang, Huilun Wang, Florian Wilfling, Ting-Wei Mu, Alexandra Stolz, and Jun Hamazaki—valuable for mechanism, resistance, ERAD/autophagy, or new target-space questions, but less direct for immediate degrader asset generation. - **Lower-priority for this specific mandate:** the bottom tier is scientifically credible in degradation/proteostasis biology, but the supplied evidence shows limited direct engagement with modern therapeutic degrader modalities or limited independent leadership signals.

4. Tiering of Profiles: Tier 1, Tier 2, and Tier 3

4.1 Tiering logic

The tiering below converts the Chapter 3 ranking into an action-oriented segmentation. It is based on a blend of:

- the five harmonized criteria,
- the comparative ranking,
- direct degrader relevance,
- translational maturity, and
- likely near-term usefulness for collaboration, consulting, or co-development.

Tiering is not a statement of absolute scientific quality. A number of Tier 3 researchers are scientifically strong, but fall lower for this specific mandate because their work is more adjacent to proteostasis biology than directly positioned for degrader-focused partnering.

4.2 Tier definitions

Tier 1: Highest-priority external tracking and engagement pool

Definition: Best near-term fit for direct degrader-related collaboration, consulting, or co-development. These profiles combine strong topical momentum, differentiated platform or modality value, and credible translational signals.

Included profiles (Ranks 1–10): Shipeng He, Pranam Chatterjee, Wenchao Lu, William Farnaby, Hojong Yoon, Woong Sub Byun, Ling Xie, George M. Burslem, Baishan Jiang, Nicole Trainor.

Why they are Tier 1: - They are the clearest matches to the company mandate of tracking emerging leaders in degraders. - Most are working directly in **molecular glues, PROTACs, induced proximity, next-generation delivery, or modality expansion**. - Several already show **in vivo efficacy, disease-model validation, or patent-linked translational direction**, for example HerTACs (2025), programmable β-catenin degraders (2024), TEAD degrader (2025), and pan-KRAS degrader (2024).

Tier 2: Strong follow-up pool with clear value but at least one meaningful constraint

Definition: Strong candidates for targeted follow-up, selective collaboration, or scientific advisory engagement, but with one or more limitations relative to Tier 1 such as narrower direct relevance, earlier translational maturity, or limited evidence of independent leadership.

Included profiles (Ranks 11–31): Scott J. Hughes, Dean Clift, Xing Qiu, Zhengnian Li, Justin M. Reitsma, Matthias Hinterndorfer, Kheewoong Baek, Wenyi Mi, Vincenzo D'Angiolella, Ryan D. Baldridge, Matthew E. R. Maitland, Martin Peter Schwalm, Jiwon Hwang, Jonathan St. Germain, Ka Yang, Huilun Wang, Ting-Wei Mu, Florian Wilfling, Xiaoqiong Wei, Siavash Vahidi, Yongwang Zhong.

Why they are Tier 2: - Many have strong scientific value, but are either **more enabling than asset-centric**, or have weaker evidence on independence and translational ownership. - This tier includes two distinct subgroups: - **Tier 2A: direct degrader and E3-expansion contributors**, such as Scott J. Hughes, Dean Clift, Zhengnian Li, Wenyi Mi, and Matthias Hinterndorfer. - **Tier 2B: enabling biology specialists**, such as Ryan D. Baldridge, Jiwon Hwang, Huilun Wang, Ting-Wei Mu, Florian Wilfling, and Siavash Vahidi. - These researchers are particularly useful where the company needs **mechanistic depth, new E3 or degron biology, assay systems, or noncanonical degradation routes** rather than immediate compound-centric co-development.

Tier 3: Lower-priority for this specific mandate, but selectively relevant as biology or platform specialists

Definition: Scientifically credible profiles with either indirect relevance to modern degrader modalities, limited recent direct TPD evidence, or limited evidence of independent external leadership in the supplied record.

Included profiles (Ranks 32–50): Hong Yue, Vinay V. Eapen, Ryan J. Lumpkin, Shoshiro Hirayama, Jun Hamazaki, Yuanzhong Wu, Alexandra Stolz, Yulia Jitkova, Yuuki Fujiwara, Koraljka Husnjak, Qian Chen, Nico Dissmeyer, Vishwajeeth Pagala, Mauricio Andres Torres-Gutierrez, Rachel Byerley Reinert, Neha Shrestha, Hongsheng Lin, Eileithyia Swanton, Yoichiro Harada.

Why they are Tier 3: - The most common reason is **adjacency rather than directness**: strong work in ERAD, autophagy, lysosomal uptake, proteasome biology, plant degradation systems, or ubiquitin-mediated regulation, but not clearly in current therapeutic degrader modalities. - Another recurring issue is **leadership ambiguity**: several profiles appear on strong papers but the metadata provide limited evidence for first/last-author ownership, grants, or independent program-building. - This tier should not be ignored; it is the right pool for **problem-specific biological consultation** rather than broad external prioritization.

4.3 Practical interpretation of the tiers

- **Tier 1:** active outreach and structured business/scientific engagement now
- **Tier 2:** selective follow-up, topic-specific diligence, and monitor-for-escalation

- **Tier 3:** maintain on radar for narrow biology questions, but not as the primary external focus for degrader discovery partnerships

Overall, the tiering shows a relatively small but high-value Tier 1, a broad and strategically useful Tier 2, and a Tier 3 that is scientifically respectable but less aligned with the company's immediate degrader-partnering objectives.

5. In-Depth Assessment of Tier 1 Profiles

Tier 1 contains the most actionable external targets in this cohort. These profiles combine **direct degrader relevance, differentiated strategic positioning, and tangible partnering value**. The highest-conviction use cases fall into three broad buckets: **precision degradation platforms**, **molecular-glue/E3-expansion discovery**, and **structure-guided translational degrader chemistry**.

5.1 Shipeng He

Why Tier 1: Shipeng He is the clearest example in this cohort of a researcher building a **precision-degradation platform portfolio**, not just individual compounds. The work spans tumor-selective lysosomal degradation of membrane and extracellular proteins, targeted delivery enhancement, photoswitchable control, and sonodynamic activation. Particularly strong signals come from HerTACs (2025), which showed potent in vivo antitumor activity with low systemic toxicity, and SDPTAC (2025), which reported near-complete tumor growth inhibition in vivo.

Strategic relevance: This is one of the most attractive profiles for companies that want to expand beyond standard intracellular PROTAC logic into **membrane, extracellular, delivery-constrained, or spatially controlled degradation**. He is especially relevant if the organization values platform differentiation over another crowded CRBN/VHL program.

Best-fit engagement mode: - co-development on precision-delivery degradation concepts, - consulting on spatiotemporal degradation strategies, - collaboration on membrane/extracellular target classes.

Main caveat: Formal independence and competitive-funding evidence are limited in the supplied record, so diligence should focus on ownership, institutional freedom, and reproducibility rather than assuming mature independence.

5.2 Pranam Chatterjee

Why Tier 1: Chatterjee is arguably the most differentiated platform innovator in the set. His core advantage is **AI/language-model-enabled peptide binder design linked to programmable degradation and stabilization**, with validation in multiple target classes. Programmable β-catenin degraders (2024) demonstrated selective knockdown of pathogenic β-catenin pools and in vivo activity after LNP-mRNA delivery, while PepPrCLIP (2025) advanced sequence-only design of peptide binders to conformationally diverse targets.

Strategic relevance: This profile is especially strong for companies interested in **undruggable proteins, intracellular biologics, RNA/LNP-enabled delivery, and next-generation induced-proximity modalities**. The platform can be relevant both to degradation and stabilization, which broadens its strategic value.

Best-fit engagement mode: - strategic platform partnership, - co-development around hard-to-drug oncology or immunology targets, - advisory engagement on AI-enabled degrader design workflows.

Main caveat: The science is unusually compelling, but the supplied evidence is lighter on formal grant/award signals than on publications and patents. As a result, technical diligence should include manufacturability, delivery scalability, and IP boundaries.

5.3 Wenchao Lu

Why Tier 1: Wenchao Lu combines exactly the features that make a strong near-term degrader collaborator: **high recent output, chemistry-led modality breadth, and convincing preclinical signals**. The profile includes a durable TEAD degrader (2025), orally active CK1α degraders for AML (2024), and a highly selective NEK7 molecular glue degrader (2025).

Strategic relevance: Lu is highly relevant for **oncology-focused degrader discovery**, especially where the company wants access to both heterobifunctional and molecular-glue approaches. The profile also suggests strong value in **first- or best-in-class lead generation** rather than only mechanism papers.

Best-fit engagement mode: - co-development or sponsored research on new oncology targets, - consulting on glue-versus-PROTAC route selection, - external scouting anchor for fast-moving degrader chemistry in Asia.

Main caveat: The record is strong scientifically, but the metadata provide limited direct evidence on independent recognition. Practical engagement should therefore test how much of the translational momentum is profile-owned versus embedded in larger programs.

5.4 William Farnaby

Why Tier 1: Farnaby stands out for **structure-guided, translationally mature degrader science**. The profile includes pan-KRAS degraders (2024) with in vivo tumor regression, a CNS-active GSK3 degrader (2025), and funded work on rational degrader-glue design through the KinoGlu project.

Strategic relevance: This is one of the strongest profiles for **consulting and collaboration at the interface of medicinal chemistry, ternary-complex design, and target selection**. He is particularly attractive if the organization has interest in **KRAS, CNS delivery, or structure-biophysics-led degrader design**.

Best-fit engagement mode: - high-level scientific advisory role, - targeted collaboration on difficult oncology or CNS targets, - mechanistic consulting on cooperativity and ternary design.

Main caveat: Much of the record is collaborative and may involve complex institutional/partner entanglement. That does not reduce scientific value, but it may affect exclusivity and transaction simplicity.

5.5 Hojong Yoon

Why Tier 1: Yoon's key strategic advantage is **molecular-glue discovery infrastructure**, especially the ability to identify and characterize new induced-proximity interactions at scale. The strongest evidence comes from CRBN molecular glue interactome mapping (2025) and the NIH-backed chemoproteomic molecular-glue discovery project.

Strategic relevance: This is a particularly good fit for companies that want to build **glue discovery capability rather than chase single targets**. Yoon is valuable both for target identification and for E3-ligase opportunity mapping.

Best-fit engagement mode: - consulting on molecular-glue discovery strategy, - joint platform work around CRBN and noncanonical glue discovery, - external scientific board role focused on induced proximity.

Main caveat: Although the scientific niche is excellent and the funding trajectory is stronger than most in the cohort, the metadata do not always establish him as the senior driver on the most visible papers.

5.6 Woong Sub Byun

Why Tier 1: Byun is one of the strongest chemistry-first rising profiles in **rational molecular glue and covalent degrader design**. He has concentrated recent momentum in BRD9 molecular glue degraders (2025), FBXO22 degraders and recruiters (2025), and SNAr-enabled electrophilic degraders (2024).

Strategic relevance: He is especially relevant where the company wants to **expand beyond standard E3 chemistry** and systematically explore noncanonical recruiter space. The profile is well suited to innovation-oriented collaboration in glue and covalent induced proximity.

Best-fit engagement mode: - medicinal-chemistry collaboration, - consulting on noncanonical E3 recruitment, - targeted scouting for early but inventive degrader concepts.

Main caveat: Independence appears to be emerging rather than fully established; many outputs are still within elite, senior-led collaborative networks.

5.7 Ling Xie

Why Tier 1: Ling Xie brings breadth across **next-generation degrader and stabilizer modalities**: TF-PROTACs, bridged PROTACs, DUBTACs, nucleotide-based concepts, and multiple first-in-class target programs. Representative outputs include the AURKB

degrader (2025), ETV6 TF-PROTACs (2025), and PRO-DUBTAC stabilization strategy (2025).

Strategic relevance: Xie is a strong fit where the company cares about **modality expansion for transcription factors or other difficult targets**, and where stabilization as well as degradation may be strategically useful.

Best-fit engagement mode: - collaboration on nonclassical target classes, - advisory role on transcription-factor degradation or stabilization, - exploratory partnership for platform diversification.

Main caveat: The translational signals are good, but the supplied record contains limited explicit evidence of independent grant support or PI-style leadership.

5.8 George M. Burslem

Why Tier 1: Burslem combines **induced-proximity platform building** with credible early translational execution. The strongest examples are the preclinical LRF molecular glue degrader (2025), lipid-delivered bioPROTACs (2024), and systematic induced-proximity discovery work in pooled endogenous tagging and recruitment screens (2024).

Strategic relevance: He is a particularly attractive profile for companies interested in **platform collaboration rather than only single-target chemistry**, especially around induced proximity, alternative effectors, and biologic-style degradation routes.

Best-fit engagement mode: - consulting on induced-proximity platform strategy, - collaboration on bioPROTAC or intracellular delivery concepts, - translational partnering around new degradation effectors.

Main caveat: The profile is strong but not as clearly elite on visibility/leadership evidence as the top three. This looks more like a high-value platform collaborator than a de-risked franchise leader at present.

5.9 Baishan Jiang

Why Tier 1: Baishan Jiang is one of the strongest kinase-focused degrader chemists in the cohort, with repeated evidence of **selectivity, PK, oral exposure, xenograft efficacy, and IP-linked translation**. Key outputs include the selective CDK6 degrader (2026), NEK7 molecular glue degrader (2025), and multitarget CDK12/7/9 degrader in prostate cancer (2025).

Strategic relevance: This is a strong co-development profile for **oncology kinase degradation**, especially where resistance, selectivity, and pharmacology are central. He is also relevant for groups that want **chemically tractable programs with translational intent** rather than only conceptual novelty.

Best-fit engagement mode: - co-development on kinase degrader programs, - medicinal-chemistry collaboration on oncology targets, - competitive intelligence tracking for Asia-based degrader chemistry.

Main caveat: The main uncertainty is not scientific value but ownership and independence. That should be resolved early in diligence.

5.10 Nicole Trainor

Why Tier 1: Trainor's profile is driven by participation in **very high-value translational degrader programs**: KRAS, TEAD, and SMARCA among them. Particularly relevant are KRAS(on) degraders (2025), IAP-based TEAD degraders (2026), and prior in vivo-active SMARCA programs.

Strategic relevance: She is a strong match for organizations that want exposure to **mechanism-informed design of difficult oncology degraders**, especially where biophysics, cooperativity, and translational chemistry intersect.

Best-fit engagement mode: - consulting or collaboration around oncology degrader design, - scientific advisory role on KRAS/TEAD/SMARCA programs, - diligence target for highly collaborative, industry-style teams.

Main caveat: This is another case where the translational science is strong, but the supplied metadata provide limited clarity on independent leadership versus major-team contribution.

5.11 Tier 1 synthesis

Across Tier 1, three practical conclusions stand out:

1. **The strongest immediate opportunities are platform-led, not target-by-target.** Shipeng He, Pranam Chatterjee, Wenchao Lu, Hojong Yoon, and George Burslem are especially attractive where the company wants reusable capability.
2. **The most translationally ready chemistry-centered profiles are William Farnaby, Baishan Jiang, Nicole Trainor, and Wenchao Lu.**
3. **Several Tier 1 profiles still require diligence on independence and ownership.** That is a manageable issue, but it argues for staged engagement rather than immediate large-scope commitments.

6. Cross-Profile Trends, Patterns, and Clusters

6.1 The field is shifting decisively beyond CRBN and VHL

One of the clearest cross-profile signals is a move away from over-reliance on the classical CRBN/VHL axis. Multiple profiles are building value around **new E3 ligases or noncanonical recruitment routes**, including DCAF16, DCAF15, FBXO22, GID4, ZYG11B, CTLH/GID, SPOP, TRIM21, KEAP1, and YPEL5/GID-CTLH. Representative examples include CRBN molecular glue interactome mapping by Hojong Yoon (2025), FBXO22 recruitment by Woong Sub Byun and collaborators (2025), GID4-based PROTACs by Wenyi Mi and collaborators (2025), and TRIMTACs from Dean Clift (2025).

Implication: this cohort contains meaningful external options for E3 diversification, which is strategically important for avoiding modality crowding and expanding target tractability.

6.2 Target scope is broadening from soluble intracellular proteins to harder target classes

A second strong pattern is the expansion of degradation into **transcription factors, membrane proteins, extracellular proteins, viral proteins, ER-luminal proteins, and pathway-defined subpopulations of proteins**. Examples include TF-PROTACs from Ling Xie (2025), HerTACs and GLTACs from Shipeng He (2025), dengue degraders from Zhengnian Li and Wenchao Lu-linked networks, and ER-lumen degrons from Ryan D. Baldridge and Jiwon Hwang (2024).

Implication: the best external opportunities are increasingly those that can open **new biological compartments and target classes**, not just improve potency against already tractable nuclear or cytosolic proteins.

6.3 Precision, delivery, and context selectivity are becoming core differentiators

A notable cluster of top-ranked profiles is focused less on whether degradation can happen and more on **where, when, and in which cells it should happen**. This includes aptamer/drugtamer strategies, tumor-selective lysosomal systems, tissue-sparing glues, reversible or stimulus-responsive degraders, and LNP-enabled intracellular delivery. Strong examples include Shipeng He's precision-degradation portfolio, Pranam Chatterjee's LNP/mRNA-enabled programmable degraders, and BRD9 glue degraders that spare cardiomyocytes from Woong Sub Byun (2025).

Implication: future partnering value will likely depend increasingly on **delivery, tissue selectivity, and therapeutic window engineering**, not only degrader potency.

6.4 There is a large enabling-biology cluster that matters strategically even when it ranks lower

Many mid-ranked and lower-ranked profiles are not weak scientists; they are simply more focused on **degradation biology than degrader modalities**. This cluster includes ERAD, autophagy, lysosomal uptake, proteasome regulation, and mitochondrial protease biology. Representative profiles include Huilun Wang on SEL1L-HRD1 rescue (2026), Ting-Wei Mu on ER-phagy in neuroreceptors (2026), Florian Wilfling on selective-autophagy initiation hubs (2025), and Alexandra Stolz on LC3/GABARAP ligand validation for ATTEC development (2024).

Implication: these researchers may not be the first call for degrader asset generation, but they are highly relevant for **mechanism-of-action studies, resistance hypotheses, biomarker strategy, and access to non-proteasomal degradation routes**.

6.5 Molecular-glue science is one of the fastest-moving subthemes in the cohort

A particularly dense cluster centers on **molecular glues**, including target discovery, structural mechanism, covalent glue chemistry, and proteome expansion. Key contributors include Hojong Yoon, Woong Sub Byun, Kheewoong Baek, Ryan J. Lumpkin, Vincenzo D'Angiolella, Zhengnian Li, and Wenchao Lu. This is supported by studies such as Unveiling the hidden interactome of CRBN molecular glues (2025), Template-assisted covalent

modification underlies activity of covalent molecular glues (2024), and UM171/KBTBD4 structural mimicry work (2025).

Implication: if the company wants differentiated entry points, **glue discovery and glue-enabling biology** appear richer in this cohort than classical incremental PROTAC optimization.

6.6 A recurring weakness across the cohort is incomplete evidence on independence

One consistent pattern across many otherwise strong profiles is **limited explicit evidence of independent funding, formal leadership, or senior-author ownership**. This is especially true for researchers embedded in elite collaborative networks. The result is that the cohort contains many people who look like excellent **contributors or emerging operators**, but fewer who are fully de-risked as stand-alone external franchise leaders.

Implication: engagement strategy should be tiered. High-science profiles with uncertain independence are still worth engaging, but often through **consulting, focused pilot collaborations, or expert-network relationships first**, rather than broad long-term commitments.

7. Key Risks, Challenges, Constraints, and Opportunities

7.1 Main risks and constraints

Risk / constraint	Why it matters	Practical implication
Leadership ambiguity	Many strong profiles are embedded in large teams and the metadata often do not resolve first/last-author or PI-level ownership	Scientific quality may be high while transaction simplicity is lower
Direct-relevance dilution	A substantial subset of the cohort is stronger in ERAD, autophagy, proteasome biology, or lysosomal mechanisms than in drug-like degraders	These profiles are valuable, but usually not first-line co-development candidates
Novel E3 risk	Expansion beyond CRBN/VHL is strategically attractive, but not every new E3 will be chemically or pharmacologically tractable	Early novelty should not be mistaken for near-term developability
Translation gap	Many profiles show preclinical promise, but few have clear clinical-stage progression in the supplied evidence	Several opportunities remain platform- or probe-stage rather than asset-stage
Platform fragmentation	The field is splitting across glues, PROTACs, lysosomal chimeras, bioPROTACs, peptide degraders, and autophagy concepts	A diffuse external strategy risks spreading effort too thinly
IP / partnering complexity	Many top profiles are linked to multi-institutional or industry-linked collaborations	Freedom-to-operate and exclusivity questions may be material early in diligence

7.2 The most important opportunities

Opportunity 1: Build a diversified modality network rather than a single-platform network

The top-ranked group is unusually complementary. For example: - **Shipeng He** offers precision delivery and membrane/extracellular degradation (HerTACs (2025)) - **Pranam**

Chatterjee offers AI-designed peptide-guided degradation and stabilization (programmable β-catenin degraders (2024)) - **Wenchao Lu** and **Baishan Jiang** offer translational degrader chemistry and molecular-glue discovery (TEAD degrader (2025); selective CDK6 degrader (2026)) - **Hojong Yoon** offers glue-target discovery infrastructure (CRBN glue interactome (2025))

Decision value: a balanced network reduces dependence on any single degrader modality and expands optionality for future pipelines.

Opportunity 2: Use Tier 2 to strengthen mechanism, not just sourcing

Tier 2 contains many of the cohort's best **mechanistic de-riskers**. Profiles such as Ryan D. Baldridge, Jiwon Hwang, Huilun Wang, Florian Wilfling, and Alexandra Stolz can help on questions involving **new compartments, degron discovery, autophagy, ERAD, or lysosome-enabled degradation**. For example, ER-lumen degrons (2024) and LC3/GABARAP ligand validation (2024) are not asset programs in the conventional sense, but they can materially improve targetability strategy.

Decision value: this tier is highly useful for reducing platform risk and opening target spaces that standard medicinal chemistry alone cannot unlock.

Opportunity 3: Focus on molecular glues as a high-yield white space

The cohort contains an unusually dense glue-related network spanning **target discovery, structural mechanism, covalent glue chemistry, and novel ligases**. This includes Hojong Yoon, Woong Sub Byun, Kheewoong Baek, Ryan J. Lumpkin, Vincenzo D'Angiolella, Zhengnian Li, and Wenchao Lu.

Decision value: glue biology may offer the best combination of novelty and strategic differentiation relative to crowded classical PROTAC programs.

Opportunity 4: Pair chemistry-centric and biology-centric external partners on the same problem

The strongest strategic pattern is not chemistry versus biology; it is the **combination** of both. For example, a company could pair a Tier 1 degrader innovator with a Tier 2/Tier 3 biology expert in ERAD, autophagy, or proteasome control to address resistance, compartment access, or tissue selectivity.

Decision value: this creates a more robust external innovation model than treating each profile as a standalone supplier of ideas.

7.3 Bottom-line interpretation

The portfolio-level risk is not lack of scientific quality. The real challenge is **choosing the right engagement type for each profile**. If the company treats all high-quality researchers as equivalent asset partners, it will overcommit to some profiles and underuse others. The best outcome will come from matching:

- **Tier 1** to asset-centric collaboration and strategic scientific access,

- **Tier 2** to mechanism, platform, or targeted advisory roles,
- **Tier 3** to narrow biological consultation or watchlist monitoring.

8. Strategic Recommendations and Priority Actions

8.1 Recommended portfolio strategy

The company should adopt a **three-lane external-engagement model** rather than a single undifferentiated partner list.

Lane A: Immediate asset and platform engagement

Target the highest-conviction Tier 1 profiles whose work is both directly relevant and practically partnerable.

Priority names: - **Shipeng He** for precision degradation and lysosomal/membrane targeting (HerTACs (2025)) - **Pranam Chatterjee** for AI-designed peptide-guided degradation and stabilization (programmable β-catenin degraders (2024)) - **Wenchao Lu** for translational degrader and glue chemistry (TEAD degrader (2025)) - **William Farnaby** for structure-guided translational consulting and difficult targets (pan-KRAS degrader (2024)) - **Hojong Yoon** for molecular-glue discovery infrastructure (CRBN glue interactome (2025))

Lane B: Mechanism and E3-expansion partnerships

Use selected Tier 1 and Tier 2 profiles to strengthen internal capability around **new E3 ligases, degrader specificity, induced proximity, and mechanism-of-action de-risking**.

Priority names: - **Woong Sub Byun** and **Kheewoong Baek** for glue and noncanonical E3 recruitment - **Wenyi Mi** and **Matthew E. R. Maitland** for GID4/CTLH and new E3 routes - **Justin M. Reitsma**, **Martin Peter Schwalm**, and **Jonathan St. Germain** for E3 evaluation, mechanistic assays, and specificity tools - **George M. Burslem** for induced proximity and bioPROTAC strategy

Lane C: Enabling biology and future-option scouting

Create a smaller, lower-cost watchlist of enabling biologists who can be activated when specific technical questions arise.

Priority names: - **Ryan D. Baldridge** and **Jiwon Hwang** for ER-lumen and ERAD degrons - **Huilun Wang** and **Ting-Wei Mu** for ERAD/ER-phagy disease biology - **Florian Wilfling** and **Alexandra Stolz** for autophagy/lysosomal degradability rules - **Jun Hamazaki** and **Shoshiro Hirayama** for proteasome capacity and turnover biology

8.2 Recommended actions over the next 90 days

Action 1: Start immediate outreach to the top five profiles

Open science-first conversations with **Shipeng He, Pranam Chatterjee, Wenchao Lu, William Farnaby, and Hojong Yoon**. The goal should not initially be a broad deal

discussion; it should be to test: - strategic fit with internal target classes, - willingness to engage in consulting versus collaboration, - degree of IP flexibility, - and technical maturity of the relevant platform.

Action 2: Build a focused external advisory cell around modality expansion

Recruit 4–6 external experts from Tier 1 and upper Tier 2 into a lightweight advisory network covering: - molecular glues, - alternative E3 ligases, - precision-delivery degradation, - and noncanonical degradation routes.

A strong initial mix would include **William Farnaby, Hojong Yoon, George M. Burslem, Woong Sub Byun, Justin M. Reitsma, and Alexandra Stolz**.

Action 3: Launch two pilot collaboration concepts, not five

To avoid fragmentation, keep the first wave tightly scoped:

1. **Precision and hard-target degradation pilot**
   - anchor profiles: Shipeng He, Pranam Chatterjee, Ling Xie
   - focus: membrane/extracellular, transcription-factor, or undruggable targets
2. **Glue and E3-diversification pilot**
   - anchor profiles: Hojong Yoon, Woong Sub Byun, Wenyi Mi, Kheewoong Baek
   - focus: unexplored E3s, new recruiter chemistry, target-discovery workflows

Action 4: Add a formal diligence checklist before any deeper commitment

For each candidate, confirm: - actual leadership role on the cited programs, - patent ownership and institutional constraints, - reproducibility of the most decision-relevant results, - translational readiness of the platform, - and overlap or conflict with existing company programs.

This step is essential because the current evidence is strong on science but uneven on independence and ownership.

Action 5: Establish escalation triggers for Tier 2 and Tier 3

Profiles should move up in priority if any of the following appear in the next 6–12 months: - first- or senior-author leadership on flagship degrader papers, - new in vivo or patent-linked translation, - competitive grants or clearer PI independence, - demonstration of a usable new E3 ligase or degradation route.

8.3 Final strategic recommendation

The company should treat this cohort not as a simple ranked vendor list, but as an **external capability map**. The most value will come from:

- doing **deep engagement with a small Tier 1 core**,
- using **Tier 2 to improve internal technical odds of success**,

- and maintaining **Tier 3 as a targeted biological reserve** for mechanism-heavy questions.

That approach is more likely to produce differentiated external advantage than attempting broad, low-intensity engagement across all 50 profiles.

9. Executive Summary

This review of 50 researcher profiles identified a **small but high-value Tier 1 group** that is most relevant for near-term external engagement in protein degraders. The strongest immediate targets are **Shipeng He, Pranam Chatterjee, Wenchao Lu, William Farnaby, and Hojong Yoon**, with the broader Tier 1 completed by **Woong Sub Byun, Ling Xie, George M. Burslem, Baishan Jiang, and Nicole Trainor**. These researchers collectively cover the most strategically important areas of the current landscape: **precision degradation, molecular glues, programmable peptide degraders, new E3-ligase recruitment, translational degrader chemistry, and hard-target expansion**. Representative high-value signals include HerTACs (2025), programmable β-catenin degraders (2024), TEAD degrader (2025), and pan-KRAS degrader (2024).

Across the full set, three cross-cutting patterns stand out. First, the field is moving **beyond CRBN and VHL**, with meaningful activity around DCAF16, DCAF15, FBXO22, GID4, ZYG11B, TRIM21, CTLH/GID, and other noncanonical routes. Second, the most differentiated programs increasingly address **delivery, tissue selectivity, membrane/extracellular targets, transcription factors, viral proteins, and difficult cellular compartments** rather than only classical soluble targets. Third, a large fraction of the cohort is stronger in **enabling degradation biology** than in direct degrader asset creation; those profiles remain valuable, but usually for mechanism, resistance, or platform support rather than primary co-development.

The main constraint across the dataset is **uneven evidence on independence and ownership**. Many top scientific contributors are embedded in strong collaborative networks, but the supplied evidence is often limited on grants, formal leadership, and PI-level control of the most relevant programs. This should not disqualify them, but it does argue for **staged diligence and engagement** rather than immediate broad commitments.

Recommended action: initiate immediate outreach to the top five Tier 1 profiles; build a small external advisory network around modality expansion and E3 diversification; and run two focused pilot workstreams, one centered on **precision and hard-target degradation** and the other on **molecular-glue and E3-expansion strategy**. Tier 2 should be used selectively for mechanism and platform de-risking, while Tier 3 should remain a targeted watchlist for problem-specific biological consultation.