PROGRAMMABLE PROTEIN STABILIZATION WITH LANGUAGE MODEL-DERIVED PEPTIDE GUIDES

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ABSTRACT

Dysregulated protein degradation through the ubiquitin proteasomal pathway can induce numerous disease phenotypes, including cancer, neurodegeneration, and diabetes. Although small molecule-based targeted protein degradation (TPD) and targeted protein stabilization (TPS) platforms can address this dysregulation, they rely on structured and stable binding pockets, which do not exist to classically undruggable targets. Here, we expand the TPS target space by engineering "deubiquibodies" (duAbs) via fusion of computationally-designed peptide binders to the catalytic domain of the potent OTUB1 deubiquitinase. In human cells, duAbs effectively stabilize exogenous and endogenous proteins in a DUB-dependent manner. Using protein language models to generate target-binding peptides, we engineer duAbs to conformationally diverse target proteins, including key tumor suppressor proteins p53 and WEE1, and heavily disordered fusion oncoproteins, such as PAX3::FOXO1. We also encapsulate duAbs targeting p53 as mRNA in lipid nanoparticles and demonstrate effective intracellular delivery, p53 stabilization, and apoptosis activation, motivating further *in vivo* translation.

1 INTRODUCTION

The ubiquitin-proteasomal pathway regulates critical processes, including protein folding, DNA repair, and cell differentiation, thus helping to maintain proteostasis (Zhao et al., 2022). Dysregulation of this pathway – such as improper degradation of tumor suppressors or mutant, misfolded proteins – can lead to severe pathogenic phenotypes, such as cancer, neurodegenerative disease, cystic fibrosis, and diabetes (Sabapathy & Lane, 2018; Ward et al., 1995; Rao et al., 2015; Costes et al., 2011). Stabilizing improperly ubiquitinated proteins via target-specific deubiquitination is thus a critical therapeutic goal. Building off the major advances in targeted protein degradation (TPD) using bifunctional small-molecule degraders (Zhao et al., 2022), targeted protein stabilization (TPS) modalities, such as DUBTACs (Henning et al., 2022), have been described recently. However, these strategies rely on a limited set of chemical linkers and warheads, which are difficult to generate, synthesize, and screen for new targets and do not exist for classically "undruggable" targets (Chen et al., 2023). To address the limited reach of small molecule-based degraders, we previously engineered ubiquibodies (uAbs) by fusing computationally-designed peptides to E3 ubiquitin ligase domains for modular TPD (Portnoff et al., 2014; Chatterjee et al., 2020; Brixi et al., 2023; Bhat et al., 2025; Chen et al., 2024a).

Here, we expand the TPS target space by engineering "deubiquibodies" (duAbs) via fusion of computationally-designed protein binders to the catalytic domain of the potent OTUB1 deubiquitinase. Our duAbs potently stabilize exogenous and endogenous proteins in a DUB-dependent

manner. We further demonstrate duAb programmability by leveraging our group's generative protein language models to design target-specific peptide binders to conformationally diverse target proteins, namely transcription factors FOXP3 and β-catenin, tumor suppressor kinase WEE1, tumor suppressor p53, and "undruggable" fusion oncoprotein PAX3::FOXO1. We also showcase our novel p53-stabilizing duAb can be delivered as mRNA in lipid nanoparticles (LNPs) and corroborate effective intracellular delivery and modulation of downstream signaling pathways, thereby demonstrating the ease and speed with which new duAbs can be designed to diverse proteins in the proteome and be applied to biotherapeutics and overall proteome research applications.

2 METHODS

Binder Design The βcat-SnP-7 peptide peptide (Brixi et al., 2023), P60D2A peptide (Lozano et al., 2017), and YFP nanobody (Kanner et al., 2020) were described in previous works and obtained from respective manuscript metadata. Novel binding peptides designed in this study were either generated by the previously-described SaLT&PepPr algorithm (Brixi et al., 2023) via input of an interacting partner sequence, by the *de novo* PepPrCLIP algorithm (Bhat et al., 2025) via input of the target protein sequence, or by the target sequence-conditioned PepMLM algorithm (Chen et al., 2024a). All binder sequences can be found in Supplementary Table A2.

Generation of Plasmids All duAb plasmids were generated from the standard pcDNA3 vector, harboring a cytomegalovirus (CMV) promoter. An Esp3I restriction site was introduced immediately upstream of the OTUB1 catalytic domain and flexible GSGSG linker via Gibson Assembly using synthesized gene fragments (Azenta). For duAb assembly, oligos for candidate peptides were annealed and ligated via T4 DNA Ligase into the Esp3I-digested duAb backbone. Assembled constructs were transformed into 50 µL NEB Turbo Competent Escherichia coli cells, and plated onto LB agar supplemented with the appropriate antibiotic for subsequent sequence verification of colonies and plasmid purification (Azenta).

Cell Culture and Flow Cytometry The HEK293T, HeLa, and HepG2 cells were maintained in Dulbecco's Modified Eagle's Medium (DMEM) supplemented with 100 units/mL penicillin, 100 mg/mL streptomycin, and 10% fetal bovine serum (FBS). The alveolar rhabdomyosarcoma cell line, RH4, was maintained in RPMI 1640 supplemented with 100 units/mL penicillin, 100 mg/mL streptomycin, and 10% FBS. For duAb screening in reporter cell lines, pcDNA-duAb (500 ng) plasmids were transfected into cells ($2x10^5$/well in a 24-well plate) with Lipofectamine 2000 in serum-free Opti-MEM medium. The pan-DUB inhibitor 4 µM PR-619 was added to applicable cells, and subsequently cells were harvested within 24 hours post-treatment and analyzed on a Attune NxT Flow Cytometer (ThermoFisher) for GFP fluorescence (488-nm laser excitation, 530/30 filter for detection). 10,000 events were gated for data analysis based on default FSC/SSC parameters for the analyzed cells. Cells expressing eGFP were normalized to a sample transfected with a non-targeting duAb using the FlowJo software (`https://flowjo.com/`). Representative flow cytometry gating strategies are indicated in Supplementary Figure A2. For endogenous target screening in cell lines, pcDNA-duAb (800 ng) plasmids were transfected into cells as triplicates ($3x10^5$/well in a 12-well plate) with Lipofectamine 2000 in Opti-MEM. Cells were harvested after 72 hours for subsequent cell fractionation and immunoblotting.

Lipid Nanoparticle Formulation for p53-targeting duAbs mRNA with ARCA cap and poly(A) tail additions for p53-pMLM-4-OTUB1 was synthesized via *in vitro* transcription using the HiScribe T7 ARCA mRNA Kit (Qiagen). The mRNA was then concentrated and cleaned of impurities using the RNEasy MinElute Cleanup Kit (Qiagen). CleanCap® FLuc mRNA (NEB) was used as a positive control. Lipid nanoparticles (LNPs) were prepared by diluting DLIN-MC3-DMA, DSPC, cholesterol, and DMG-PEG2000 in ethanol using standard molar ratios 50:10:38.5:1.5, respectively. The prepared mRNA for p53-pMLM-4-OTUB1 and luciferase were diluted in citrate buffer (10 mM, pH 4) in a 2:1 volume ratio with the lipid mixture, and mRNA was loaded in a 1:20 mass ratio with the lipid, respectively. After LNP production, they were transfected into HeLa cells and were extracted 72 h post-transfection for immunoblot analysis.

Immunoblotting On the day of harvest, cells were detached were washed twice with ice-cold 1X PBS. Cell lysis and immunoblotting were performed according to standard protocols. Pro-

teins were probed with rabbit anti-WEE1 antibody (Abcam, Cat no. ab137377; diluted 1:1000), mouse anti-p53 antibody (Santa Cruz Biotechnology, Cat no. sc-126; diluted 1:1000), rabbit anti-FOXO1 antibody (Cell Signaling Technology, Cat no. 2880S; diluted 1:1000), rabbit anti-Cl-PARP-1 (Cell Signaling Technology, Cat no. 5625T, diluted 1:750), rabbit anti-Vinculin (Invitrogen, Cat no. 700062; diluted 1:2000), or mouse anti-GAPDH (Santa Cruz Biotechnology, Cat no. sc-47724; diluted 1:10000) overnight at 4C. The blots were washed and probed with a secondary antibody, donkey anti-rabbit IgG (H+L), horseradish peroxidase (HRP) (Abcam, Cat no. ab7083, diluted 1:5000) or goat anti-mouse IgG (H+L) Poly-HRP (ThermoFisher, Cat no. 32230, diluted 1:5000) for 1-2 h at room temperature. Blots were detected by chemiluminescence using a BioRad ChemiDoc™ Touch Imaging System (Biorad). Densitometry analysis of protein bands in immunoblots was performed using ImageJ software as described here: (`https://imagej.nih.gov/ij/docs/examples/dot-blot/`).

Statistical Analysis and Reproducibility Sample sizes were not predetermined based on statistical methods but were chosen according to the standards of the field (three independent biological replicates for each condition), which gave sufficient statistics for the effect sizes of interest. All data were reported as average values with error bars representing standard deviation (SD). For individual samples, statistical analysis was performed using the two-tailed Student's t test using GraphPad Prism 10 software, with calculated p values are represented as follows: *, $p<0.05$, **, $p<0.01$, ***, $p<0.001$, ****, $p<0.0001$. No data were excluded from the analyses. The experiments were not randomized. The investigators were not blinded to allocation during experiments and outcome assessment.

3 RESULTS

duAb Engineering Previous work has shown that a yellow fluorescent protein-targeting nanobody (YFP Nb) used in enDUBO1 constructs stabilized target-YFP fusion proteins (Figure 1A) (Kanner et al., 2020). We have also demonstrated our βcat-SnP-7 peptide derived from our SaLT&PepPr algorithm exhibited nanomolar binding affinity to β-catenin (Brixi et al., 2023). Thus, we sought to design a programmable, peptide-guided stabilization system (Figure 1A). We isolated catalytic domains (uni, 2025) within a subset of highly active deubiquitinases (Supplementary Table A1) (Poirson et al., 2024), linked them to the aforementioned binders using different linker candidates (Supplementary Table A1), and evaluated reporter-target protein fusions, specifically potassium ion channel protein, KCNQ1-YFP and β-catenin-sfGFP. Our results show that our OTUB1 fusions significantly increased exogenous protein expression and exceeded the stabilization via other DUB-fusion candidates (Figures 1B and A1A). We exhibit DUB-dependent stabilization by employing pan-DUB inhibitor PR-619 (Figures 1B and A1A) (Hsu et al., 2021) and mutating key OTUB1 catalytic residues (Figures 1C and A1B) (Seo et al., 2023; Wu et al., 2021). We additionally desmonstrate that we achieve potent duAb activity within 48-72 hours post-transfection by monitoring β-catenin-sfGFP expression using our βcat-SnP-7-L2-OTUB1 fusion (Figure 1D). Using TOP-GFP, a fluorescent reporter that serves as a reliable readout of β-catenin–dependent transcriptional activity (Figure 1E) (Horst et al., 2012), we corroborated increased β-catenin expression with significantly higher Wnt signaling than either untransfected cells or cells transfected with other β-catenin-targeting DUB fusions (Figure 1F), and either OTUB1 mutants (Figure 1G). Given these results, we refer to binder-L2-OTUB1 fusions as deubiquibodies, or duAbs (Figure 1A).

duAb Programmability Having shown that our duAb architecture is compatible with guide peptide binder sequences for target-specific stabilization, we sought to demonstrate duAb programmability by designing new peptides to conformationally-diverse target proteins using generative peptide design language models, which are not constrained by the requirement of stable tertiary structures (Figure 2A). We first focused our attention on FOXP3, a classically undruggable transcription factor that plays a central role in the development and function of regulatory T cells (Tregs) (Hori, 2021). We applied our SaLT&PepPr interface-prediction algorithm to isolate guide peptides from its well-known interacting partner, NFAT (Supplementary Table A2) (Barbi et al., 2015; Wu et al., 2006), and subsequently tested the peptide-guided duAbs in a FOXP3-mCherry HEK293T stable cell line. We demonstrated that SaLT&PepPr-derived duAbs induced statistically significant stabilization of FOXP3-mCherry in a DUB-dependent manner, and outperformed a duAb composed of a previously-designed P60D2A FOXP3-targeting peptide (Figure 2B) Lozano et al. (2017).

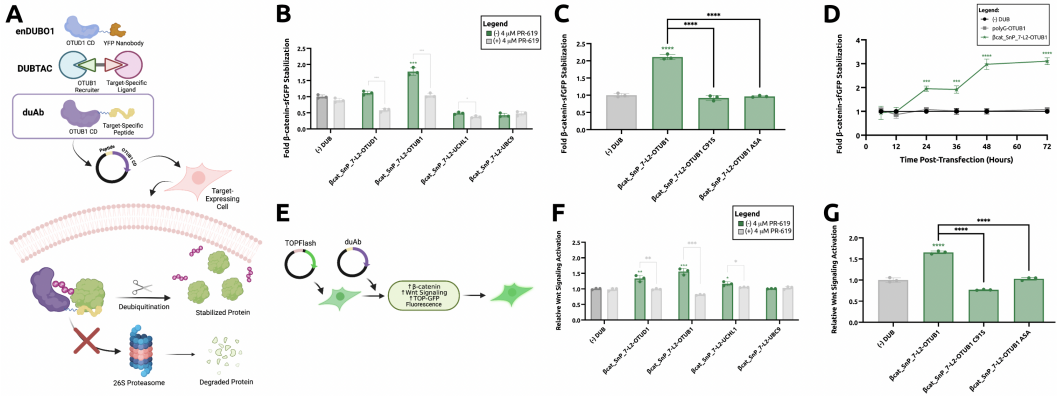

Figure 1: **duAb architecture engineering.** A) Current TPS strategies. B) β-catenin-sfGFP stabilization in HEK293T cells comparing DUB domains and PR-619 treatment. C) β-catenin-sfGFP stabilization comparing OTUB1 mutants. D) Time-course experiment for potent duAb activity. E) TOP-GFP reporter correlates with active Wnt signaling. F) Relative Wnt activity comparing DUB-fusions and PR-619 treatment. G) Relative Wnt activity comparing OTUB1 mutants.

Next, we targeted WEE1, a tumor suppressor kinase in non-cancerous eukaryotic somatic cells and is regulated by the ubiquitin-proteasomal pathway in hepatocellular carcinoma cell lines (Henning et al., 2022; Ghelli Luserna di Rorà et al., 2020; Hashimoto et al., 2003; Cruz et al., 2021). We designed six WEE1-specific peptides via our *de novo* peptide design algorithm, PepPrCLIP (Supplementary Table A2) (Bhat et al., 2025), and we reveal that each of our peptide-guided duAbs induced statistically-significant stabilization of endogenous WEE1 in heptocellular carcinoma HepG2 cells (Figure 2C). Additionally, it has been reported that fusion oncoproteins which drive pediatric cancers, such as EWS::FLI1 for Ewing sarcoma, exhibit a "Goldilocks" phenomenon, where suppression of their ubiquitination can induce fusion oncoprotein overdose and cancer cell death (Seong et al., 2021). However, pharmacologically stabilizing these proteins is highly difficult, as these proteins exhibit almost complete structural disorder with no discernable binding pockets (Figure 2D) (Tripathi et al., 2023). To overcome this structural disorder, we used our most recent peptide generator, PepMLM (Chen et al., 2024a), which only requires the target sequence as input, to generate ten PAX3::FOXO1-targeting, the main driver of pediatric alveolar rhabdomyosarcoma (ARMS) (Linardic, 2008). After transfecting plasmids encoding these peptide-guided duAbs into fusion-positive RH4 ARMS cells, we observed stable increases in the levels of both PAX3::FOXO1 fusion oncoprotein and FOXO1 for five of the duAb designs (Figure 2D). Our results demonstrate the facile programmability of duAbs to stabilize proteins with diverse tertiary structures and functional properties.

duAb Delivery in Lipid Nanoparticles for Functional p53 Stabilization Finally, we sought to stabilize p53, a key tumor suppressor protein that regulates cell cycle arrest, apoptosis, and DNA repair (Kastenhuber & Lowe, 2017). duAb-mediated p53 stabilization would ensure its availability to suppress tumor formation and growth (Figure 3A) (Lavin et al., 2006). p53 is largely disordered (Figure 3B), thus we designed eight peptides again using PepMLM (Chen et al., 2024a). As p53 is destabilized via ubiquitination in human cervical carcinoma (Crinelli et al., 2008), amongst many other cancers, we transfected HeLa cells with plasmid DNA encoding eight different duAb designs and exhibited potent duAb-dependent stabilization with peptides p53-pMLM-4 and p53-pMLM-5 (Figure 3B).

Previous studies have shown lipid nanoparticle (LNP) delivery of genetically-encodable TPD strategies as mRNA, motivating the extensibility of our duAbs delivered via LNPs for biotherapeutics applications (Figure 3A) (Cheng et al., 2020; Ghosal et al., 2024; Riley et al., 2021). We encapsulated our best performing p53 stabilizer – p53-pMLM-4-duAb – in LNPs, delivered them in HeLa cells, and demonstrated significant increases in endogenous p53 levels (Figure 3C). We also evaluated downstream functional effects via PARP-1 cleavage, which has been shown to be a hallmark of apoptosis activation (Mashimo et al., 2021), and exhibited significant cleaved PARP-1 expression

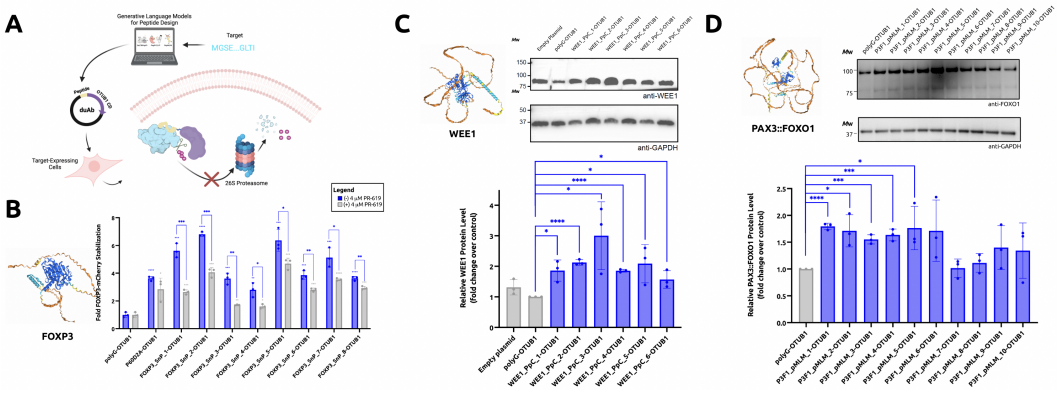

Figure 2: **Extending duAb programmability.** A) duAb design using generative language models for peptide design. B) FOXP3-mCherry stabilization in HEK293T cells using SalT&PepPr-designed duAbs in a DUB-dependent manner. C) Endogenous WEE1 stabilization in HepG2 cells using PepPrCLIP-designed duAbs. D) Endogenous PAX3::FOXO1 stabilization in RH4 cells using PepMLM-designed duAbs.

upon treatment of our p53-stabilizing duAbs (Figure 3A and 3D). Thus, our results indicate that we can programmably design a genetically-encodable TPS strategy for diverse proteins and translate them into deliverable modalities for downstream clinical applications.

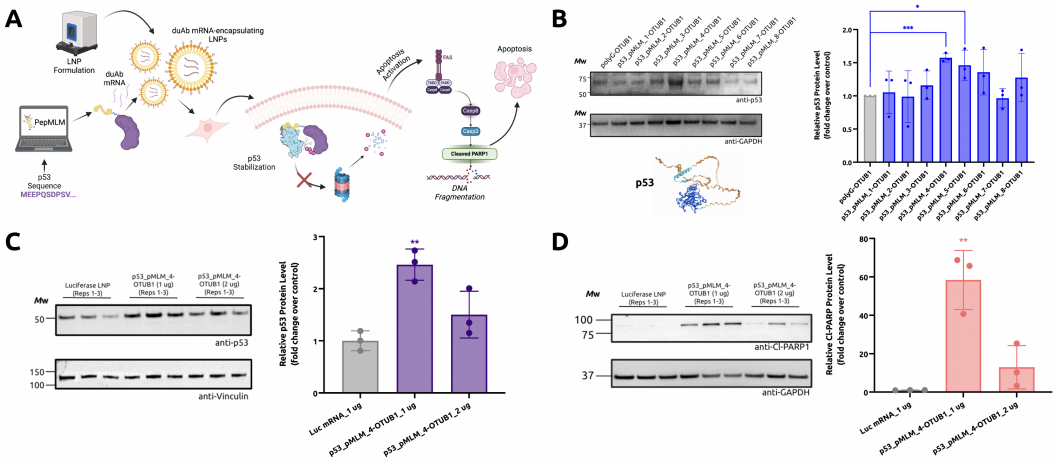

Figure 3: **p53 stabilization and apoptosis activation mediated by mRNA-encoded duAb-LNP delivery.** A) Programmable design of p53-targeting duAbs, encapsulation, and mRNA-LNP-based delivery. B) Endogenous p53 stabilization in HeLa cells. C) mRNA-LNP duAb delivery yields endogenous p53 stabilization. D) Apoptosis activation via PARP-1 cleavage upon duAb-LNP delivery.

4 Conclusion

In this work, we have demonstrated that our engineered deubiquibodies (duAbs) are a modular architecture for rescuing improperly degraded proteins via the ubiquitin-proteasome pathway, particularly those that are otherwise "undruggable" by conventional small molecule-based strategies. Integrated with our rapid binder generation algorithms (Brixi et al., 2023; Bhat et al., 2025; Chen et al., 2024a), our results demonstrate the simplicity and programmability of duAbs to stabilize diverse target substrates intracellularly.

With the rapid advancements of targeted lipid nanoparticle (LNP) platforms (Hou et al., 2021), our duAbs can be readily encapsulated as mRNA and delivered to specific tissues of interest for therapeutic applications. More interestingly, as a genetically-encoded tool, peptide-guided duAbs, alongside uAbs, may together comprise a powerful proteome screening tool for drug discovery, allowing for combinatorial protein activation and inhibition screening. Finally, we envision that our language model-generated peptides can be augmented to specifically bind post-translational and mutant isoforms of target proteins (Peng, 2024; **?**; Chen et al., 2024b), and can be fused to other PTM domains, including kinases, phosphatases, and deglycosylases, to name a few (Chen et al., 2023). This study, enabling modular peptide-guided protein stabilization, represents a next step towards this eventual goal of a fully programmable proteome editing system.

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A SUPPLEMENTARY FIGURES

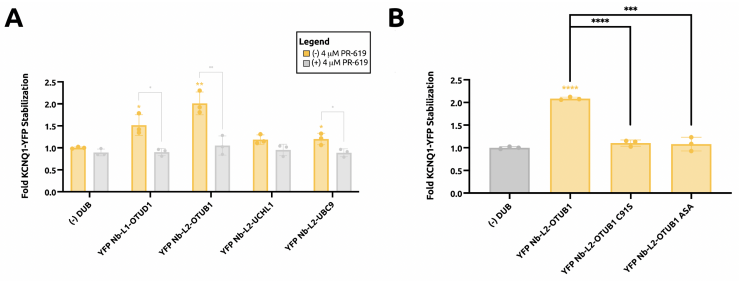

Figure A1: Engineering of the duAb architecture. A) KCNQ1-YFP stabilization in HEK293T cells using different deubiquitinase catalytic domains. B) KCNQ1-YFP stabilization in HEK293T cells using different OTUB1 mutants.

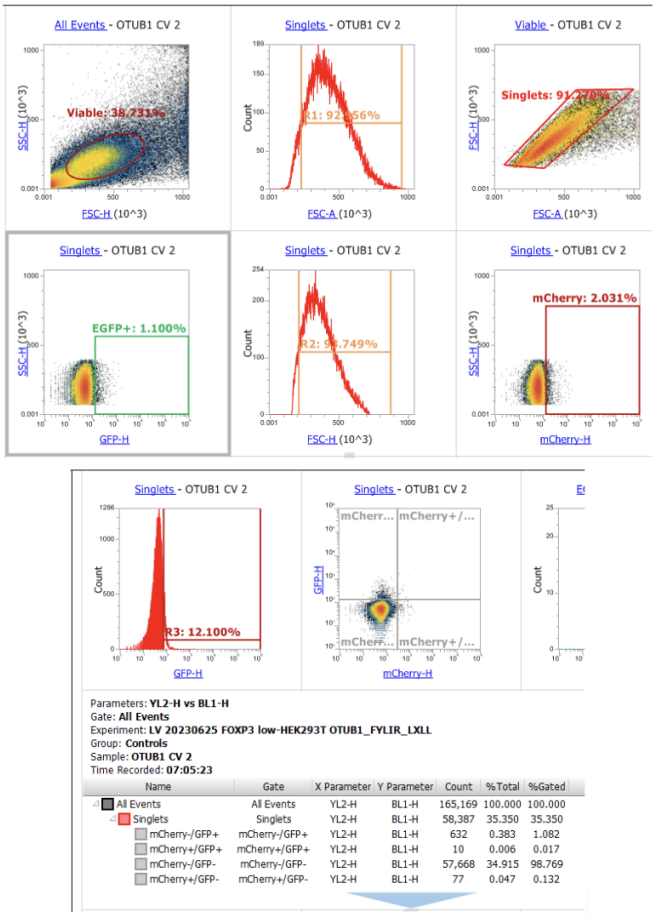

Figure A2: Example gating strategy for flow cytometry.

B SUPPLEMENTARY TABLES

Table A1: Linker and DUB catalytic domain sequences used in study.

Label/Name	Type	Amino Acid Sequence	UniProt ID
L1	Linker	GAPGSG	N/A
L2	Linker	GSGSG	N/A
OTUB1	DUB Catalytic Domain	SYIRKTRPDGNCFYRAFGFSHLEALLDDSKELQRFKAVSAKSKEDLVSQGFTEFTIEDFHNTFMDLIEQVEKQTSVADLLASFNDQSTSDYLVVYLRLLTSGYLQRESKFFEHFIEGGRTVKEFCQQEVEPMCKESDHIHIIALAQALSVSIQVEYMDRGEGGTTNPHIFPEGSEPKVYLLYRPGHYDILYK	Q96FW1
OTUD1	DUB Catalytic Domain	SDSEGVNCLAYDEAIMAQQDRIQQEIAVQNPLVSERLELSVLYKEYAEDDNIYQQKIKDLHKKYSYIRKTRPDGNCFYRAFGFSHLEALLDDSKELQRFKAVSAKSKEDLVSQGFTEFTIEDFHNTFMDLIEQVEKQTSVADLLASFNDQSTSDYLVVYLRLLTSGYLQRESKFFEHFIEGGRTVKEFCQQEVEPMCKESDHIHIIALAQALSVSIQVEYMDRGEGGTTNPHIFPEGSEPKVYLLYRPGHYDILYK	Q5VV17
UCHL1	DUB Catalytic Domain	MQLKPMEINPEMLNKVLSRLGVAGQWRFVDVLGLEEESLGSVPAPACALLLLFPLTAQHENFRKKQIEELKGQEVSPKVYFMKQTIGNSCGTIGLIHAVANNQDKLGFEDGSVLKQFLSETEKMSPEDRAKCFEKNEAIQAAHDAVAQEGQCRVDDKVNFHFILFNNVDGHLYELDGRMPFPVNHGASSEDTLLKDAAKVCREFTEREQGEVRFSAVALCKAA	P09936
UBC9	SUMOlase Catalytic Domain	MSGIALSRLAQERKAWRKDHPFGFVAVPTKNPDGTMNLMNWECAIPGKKGTPWEGGLFKLRMLFKDDYPSSPPKCKFEPPLFHPNVYPSGTVCLSILEEDKDWRPAITIKQILLGIQELLNEPNIQDPAQAEAYTIYCQNRVEYEKRVRAQAKKFAPS	P63279

Table A2: Binder sequences used in this study.

Binder Name	Target UniProt ID	Target	Binder Sequence	Derivation
YFP Nb	N/A	YFP	MQVQLVESGGALVQPGGSLRLSCAA SGFPVNRYSMRWYRQAPGKEREW VAGMSSAGDRSSYEDSVKGRFTISR DDARNTVYLQMNSLKPEDTAVYYCN VNVGFEYWGQGTQVTVSS	Kanner, et al.[11]
βcat_SnP_7	P35222	β-catenin	DPTAPPYDSLLVFDYEGS	Brixi, et al.[10]
βcat_SnP_8	P35222	β-catenin	PTAPPYDSLLVFDYEG	Brixi, et al.[10]
P60D2A	Q9BZS1	FOXP3	RAFQSFRKMWPFFAM	Lozano, et al.[20]
FOXP3_SnP_1	Q9BZS1	FOXP3	YVINGKRK	SaLT&PepPr
FOXP3_SnP_2	Q9BZS1	FOXP3	NFYVINGKRK	SaLT&PepPr
FOXP3_SnP_3	Q9BZS1	FOXP3	KVNFYVINGKRK	SaLT&PepPr
FOXP3_SnP_4	Q9BZS1	FOXP3	VKVNFYVING	SaLT&PepPr
FOXP3_SnP_5	Q9BZS1	FOXP3	TPVKVNFY	SaLT&PepPr
FOXP3_SnP_6	Q9BZS1	FOXP3	FYVINGKRKRSQ	SaLT&PepPr
FOXP3_SnP_7	Q9BZS1	FOXP3	NGKRKRSQ	SaLT&PepPr
FOXP3_SnP_8	Q9BZS1	FOXP3	RSQPQHFTYH	SaLT&PepPr
WEE1_PpC_1	P30291	WEE1	VMVVYPPGSYLTASGSN	PepPrCLIP
WEE1_PpC_2	P30291	WEE1	SGGAGGYGFCTFSGSAL	PepPrCLIP
WEE1_PpC_3	P30291	WEE1	APMYRGYMRDSPGLPVK	PepPrCLIP
WEE1_PpC_4	P30291	WEE1	EMPGCKTHKGELVWETFNG	PepPrCLIP
WEE1_PpC_5	P30291	WEE1	YPGGGSWGQPHGGSWGQK	PepPrCLIP
WEE1_PpC_6	P30291	WEE1	RMPDDLYHTGELVQCTPHA	PepPrCLIP
P3F1_pMLM_1	N/A	PAX3::FOXO1	MVTQLSSDDYTSKLVCSN	PepMLM
P3F1_pMLM_2	N/A	PAX3::FOXO1	ALTSSTSVLYTTYLLISN	PepMLM
P3F1_pMLM_3	N/A	PAX3::FOXO1	MVTQLSDDLSTYYDDLPK	PepMLM
P3F1_pMLM_4	N/A	PAX3::FOXO1	NVTSDTSDSGSTYDDCPS	PepMLM
P3F1_pMLM_5	N/A	PAX3::FOXO1	ALVSSSDVLYTYSLLISN	PepMLM
P3F1_pMLM_6	N/A	PAX3::FOXO1	NLVDLSSVLYSSYDLCPK	PepMLM
P3F1_pMLM_7	N/A	PAX3::FOXO1	NLVSLSSDLSTYYLLCKS	PepMLM
P3F1_pMLM_8	N/A	PAX3::FOXO1	ALVQDTSVLGSYYLLCSN	PepMLM
P3F1_pMLM_9	N/A	PAX3::FOXO1	NLTSLDDDLGSYKDLLKK	PepMLM
P3F1_pMLM_10	N/A	PAX3::FOXO1	NLTSLSSDSYTYKLDLPN	PepMLM
p53_pMLM_1	P04637	p53	GRTRRLGGGKLCRGS	PepMLM
p53_pMLM_2	P04637	p53	SDTYLTGAGCCCCGR	PepMLM
p53_pMLM_3	P04637	p53	KPTRRYGGGKLRCRR	PepMLM
p53_pMLM_4	P04637	p53	SPTYPLSACKKCRGR	PepMLM
p53_pMLM_5	P04637	p53	GPTRRTGAGRKTCRK	PepMLM
p53_pMLM_6	P04637	p53	SDTYRLLGRKKRCGS	PepMLM
p53_pMLM_7	P04637	p53	EPTYLIDCCY	PepMLM
p53_pMLM_8	P04637	p53	DDTYRQGPCPCT	PepMLM