Recent work suggests that the targeted degradation of cancer related factors has several advantages over other treatment methods. Proteolysis Targeting Chimeras (PROTACs) are a prominent method of degradation, where the three-part structure – consisting of an E3 ligase binder, a target protein binder, and a linker – facilitates the ubiquitination of a target protein, directing it to the 26S proteasome for degradation. PROTACs have had success, however, they are limited in target range by their ability to recruit few E3 ligases. Indeed, out of over 600 E3 ligases in the human proteome, only 4 are in common use among PROTACs. To solve this issue, our lab has developed Ubiquitin Variant Induced Proximity (UbVIP), which involves a similar three-part structure but is instead protein-based. UbVIP overcomes the challenges of limited E3s by incorporating non-inhibitory ubiquitin variants to act as the E3 ligase binder, allowing for unique E3s to be recruited. UbVIP may give greater opportunities for targeted degradation of cancer related proteins, however UbVIP is a largely unexplored topic. Therefore, I propose to utilize the UbVIP technology in conjunction with the recently developed SH2 superbinding domains (sSH2s) to target receptor tyrosine kinases (RTKs) for degradation as a novel cancer therapeutic. I also intend to advance the capacity of UbVIPs to act as therapeutics by introducing 9-arginine penetrating peptides in the UbVIP structure to act as a delivery method. This would serve to advance the knowledgebase of UbVIPs, expand their functionality as typical therapeutics, and simultaneously develop a potential novel cancer therapeutic.