PHP Biotech Identifies uPAR as the Cellular Entry Receptor for Its PHP53-nb Anti-Tumor Nanobody

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Receptor finding supports tumor selectivity, opens a path to imaging-guided patient selection, and positions the p53-restoring nanobody within a target now being pursued by leading CAR T programs.

PHP Biotech International Inc., a US-based biotechnology company developing intracellular nanobody therapeutics for aggressive solid tumors, today announced that it has identified the urokinase plasminogen activator receptor (uPAR) as the entry point for its investigational lead candidate, PHP53-nb. uPAR is the cell-surface protein that mediates the binding and internalization of the nanobody into the tumor cell, where PHP53-nb is designed to restore the p53 pathway. The identification answers one of the central questions for any intracellular therapeutic: not only what the molecule does once inside the cell, but how it gets there.

The finding matters well beyond a single molecule. For three decades, uPAR has been one of the most consistent markers of aggressiveness in cancer: the more a tumor expresses it, the more it invades, spreads and recurs. It is found in breast, pancreatic, colorectal and brain tumors, and not only on malignant cells but on the supporting stroma around them ? the tissue that feeds and protects the tumor.

What has changed recently is how the field regards the receptor. Rather than attempting to block uPAR, researchers increasingly use it as a delivery address. More than 450 patients have already been imaged with uPAR-directed PET agents across nine Phase 2 trials, making uPAR one of the rare oncology targets to be visualized in humans before being treated therapeutically at scale.

The same pathway is now being pursued by CAR T cell programs. In 2026, teams at Memorial Sloan Kettering and Columbia published in Cell, and an independent group at McMaster University and King's College London published in Science Translational Medicine, both directing engineered T cells against uPAR ? in lung, pancreatic and ovarian models, and in recurrent glioblastoma, respectively.

For PHP53-nb, the identification opens three avenues. First, it supports the nanobody's tumor selectivity documented in prior research, offering a molecular explanation for why the molecule concentrates its activity on tumor cells rather than healthy tissue. Second, it raises the prospect of patient selection using a uPAR imaging agent that already exists at clinical grade and is currently used in clinical trials ? a companion-imaging logic that most early-stage programs cannot borrow from day one. Third, it defines a dual profile: p53 pathway restoration inside the cell, with uPAR on the cell surface serving as the route of internalization. This is a sharper logic than the single-antigen approach prevailing in much of the field, in which one target must carry the burden of both recognition and therapeutic effect.

The company's internalization model describes a four-step cascade. PHP53-nb binds uPAR at the cell surface, forms a ternary complex with the LRP receptor, enters the cell through clathrin-mediated endocytosis, and is released from the early endosome into the cytoplasm for intracellular activity. Each step represents a checkpoint that a purely extracellular therapeutic never has to pass, and each is now mapped.

PHP53-nb is a first-in-class humanized camelid nanobody designed to restore the p53 axis, which is lost in roughly half of all human cancers. Beyond that therapeutic objective, nanobodies are highly modular by nature ? small, stable, easily conjugated and readily engineered into multispecific formats. A binder with confirmed uPAR-mediated internalization is, in principle, a valuable delivery mechanism. The same molecule that carries a p53-restoring mechanism today could be developed into a radioligand or an antibody-drug conjugate, combining biological tumor-suppressor restoration with a cytotoxic payload against the most aggressive tumors.

PHP53-nb is investigational and has not been approved by the U.S. Food and Drug Administration or any other regulatory authority.

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PHP Biotech

Our goal: better treatments for aggressive cancers. We developed PHP53-nb?pairing our 3-NAntC p53-reactivating peptide with the GEN01-AD nanobody platform to restore tumor-suppressing function. Promising results in TNBC and pancreatic cancer.

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