

Propanc Biopharma Publishes New Data: PRP Reprograms Chemotherapy-Resistant Pancreatic Cancer Cells & Restores Therapeutic Vulnerability

Peer-Reviewed Scientific Reports Data & Academic Collaboration Show Potent Anti-Tumor Activity, EMT Reversal, & Sequential Chemo-Sensitization; Phase 1b First-In-Human Study Planned for Q1 2027

MELBOURNE, Australia, Oct. 26, 2026 (GLOBE NEWSWIRE) -- Propanc Biopharma, Inc. (Nasdaq: PPCB) ("Propanc" or the "Company"), a biopharmaceutical company focused on developing novel treatments for chronic diseases including recurrent and metastatic cancer, today announced new preclinical data supporting the potential of its lead asset, PRP (a proprietary fixed-ratio 1:6 intravenous formulation of trypsinogen and chymotrypsinogen), to address chemotherapy resistance in pancreatic cancer.

In collaborative research led by Dr. Maria Belén Toledo at the Universities of Jaén and Granada, PRP was shown to remodel the resistant cellular state in pancreatic cancer models with acquired chemotherapy resistance. The study used a sequential design (chemoresistant cells → PRP pre-treatment → chemotherapy challenge) and demonstrated that PRP functions as a resistance-modulating intervention rather than a conventional cytotoxic agent. Key preclinical findings:

- Proliferation and survival: PRP monotherapy reduced cancer cell proliferation by ~50–54% and suppressed long-term clonogenic survival by up to 59%.
- Metastatic behavior: Migration limited to 21–57% wound closure (vs. 100% in untreated controls); invasive capacity reduced by 47–81%.
- EMT reversal: Strong epithelial differentiation with E-cadherin upregulated up to 5.29-fold and EPCAM up to 4.39-fold; mesenchymal drivers Vimentin reduced to ~0.31–0.32-fold and Snail to 0.28–0.62-fold.
- Pathway effects: Significant downregulation of TGF- β /SMAD signaling, induction of pro-apoptotic BAX (up to 2.74-fold), and suppression of oncogenic stemness drivers MYC and SOX2 (down to 0.11–0.22-fold in resistant cells).
- Combination synergy: PRP plus TGF- β inhibition further amplified responses, including 80% invasion inhibition and stronger apoptotic signaling.

These results complement the Company's recent peer-reviewed pilot study published in Scientific Reports (Nature Publishing Group), which reported an 85.9% reduction in mean tumor weight in an orthotopic pancreatic (Pan 02) model at the higher dose, ~50% tumor-

weight inhibition in an ovarian (A2780) model, anti-angiogenic activity, near-complete blockade of invasion in select lines, and EMT reversal. The Pan 02 cancer cell line is particularly noteworthy since it is a syngeneic (genetically identical or similar) tumor model conducted in immunocompetent mice and is clinically relevant for patients where cytotoxic agents induce immunosuppression and are at risk of infection—one of the main causes of death for cancer patients. Conversely, PRP reduces chemoresistance whilst enhancing immunorecognition.

In addition, earlier orthotopic and patient-derived xenograft data have also shown >90% mean tumor growth inhibition and >2.5-fold median survival extension in advanced PDAC models, with reduced metastatic burden and tumor microenvironment remodeling.

In vivo anti-tumor activity of PRP

A Orthotropic Pancreatic Tumors

Control Group (PBS)



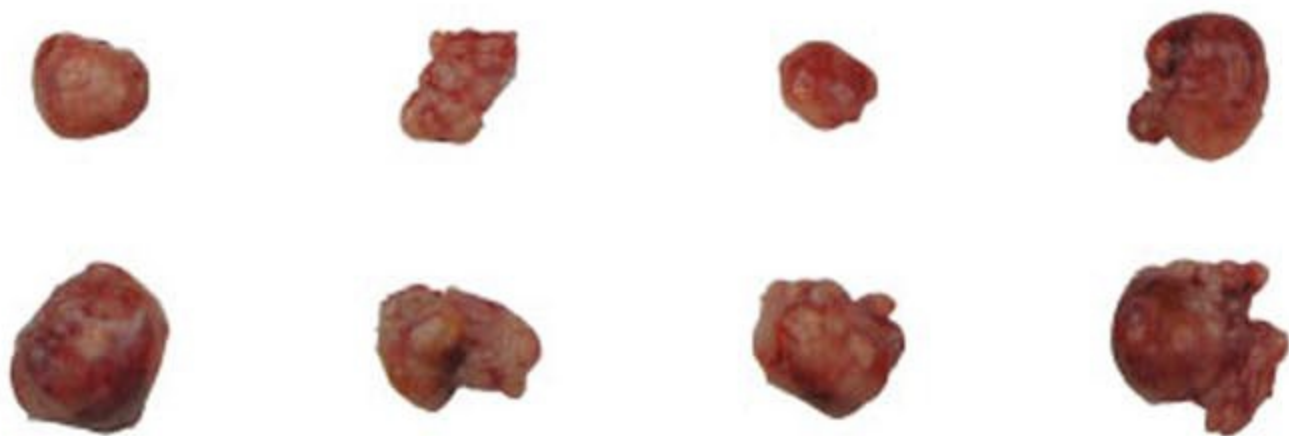
T/C: 83.3/500 mg/kg



1 cm

B Orthotropic Ovarian Tumor

Control Group (PBS)



T/C: 27.5/165 mg/kg




1 cm

(A) Images of excised tumors at study termination of orthotopic pancreatic tumors. Mean tumor weight at termination day after daily I.V. injection of PBS, T/C 27.5/165 (mg/kg) or T/C 83.3/500 (mg/kg) (P < 0.05). (B) Images of excised tumors at study termination of orthotopic ovarian tumors. Mean tumor weight at termination day after daily I.V. injection of PBS, T/C 9.1/54 (mg/kg) or T/C 27.5/165 (mg/kg) (* P < 0.05)*

PRP's differentiation-based mechanism—targeting cancer stem cells, EMT, fibrosis, and metastatic dissemination—is distinct from and potentially complementary to emerging RAS-targeted approaches. Revolution Medicines' daraxonrasib (RASONQUE), recently FDA-approved for metastatic pancreatic adenocarcinoma, has set a clinical benchmark with median overall survival of 13.2 months versus 6.6–6.7 months for chemotherapy in the Phase 3 RASolute 302 trial. Erasca's pan-RAS molecular glue ERAS-0015 similarly focuses on oncogenic RAS signaling. PRP does not inhibit RAS and is not genotype-restricted; its preclinical package of deep tumor control, fewer metastases, a less fibrotic microenvironment, and chemo re-sensitization, addresses residual biology that RAS pathway blockade does not directly target. Limited prior compassionate-use experience with related proenzyme formulations has shown signals of prolonged survival in advanced solid-tumor patients with a favorable safety profile. PRP holds FDA Orphan Drug Designation for pancreatic cancer.

“These findings are highly significant as we progress PRP to our world-first Phase 1b First-in-Human study for advanced solid-tumor patients planned for Q1 2027,” said Mr. James Nathanielsz, Propanc's Chief Executive Officer. “We envision PRP as a long-term, metronomic therapy that minimizes the threat of recurrence while enabling patients to better respond to standard treatments when resistance becomes critical. The data positions PRP as a potential sequential or combination partner to RAS inhibitors from companies such as Revolution Medicines and Erasca, addressing biology—cancer stem cells, EMT, fibrosis, and metastatic spread—that those agents do not primarily target.”

The Company continues advancing manufacturing, pharmacokinetics assay validation, and clinical preparations for the multicenter, open-label Phase 1b study (dose escalation followed by expansion) expected to enroll up to 50 patients with advanced solid tumors, including pancreatic, ovarian, and refractory prostate cancers, at sites in Australia.

About Propanc Biopharma, Inc.

Propanc Biopharma, Inc. (Nasdaq: PPCB) is developing a novel approach to preventing cancer recurrence and metastasis by targeting and eradicating cancer stem cells through proenzyme activation. The Company's lead product candidate, PRP, is designed to address the underlying drivers of cancer proliferation and spread.

More information: www.propanc.com

Forward-Looking Statements

All statements in this press release that are not historical are forward-looking statements, including, among other things, statements relating to the Company's expectations regarding its market position and market opportunity, expectations and plans as to its product development, manufacturing and sales, and relations with its partners and investors, made in reliance upon the safe harbor provisions of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. These statements are not historical facts but rather are based on the Company's current expectations, estimates, and projections regarding its business, operations and other similar or related factors. Words such as "may," "will," "could," "would," "should," "anticipate," "predict," "potential," "continue," "expect," "intend," "plan," "project," "believe," "estimate," and other similar or related expressions are used to identify these forward-looking statements, although not all forward-looking statements contain these words. You should not place undue reliance on forward-looking statements because they involve known and unknown risks, uncertainties, and assumptions that are difficult or impossible to predict and, in some cases, beyond the Company's control. Forward-looking statements are not guarantees of future actions or performance. Actual results may differ materially from those in the forward-looking statements because of several factors, including, without limitation, risks and uncertainties related to market conditions, as well as those risks described under "Risk Factors" in the prospectus related to the proposed offering and those described in the Company's filings with the SEC. The Company undertakes no obligation to revise or update information in this release to reflect events or circumstances in the future, even if new information becomes available.

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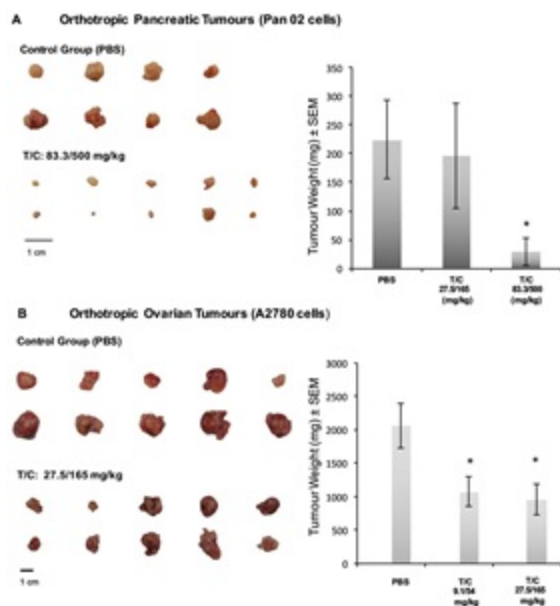
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In vivo anti-tumor activity of PRP



(A) Images of excised tumors at study termination of orthotropic pancreatic tumors. Mean tumor weight at termination day after daily I.V. injection of PBS, T/C 27.5/165 (mg/kg) or T/C 83.3/500 (mg/kg) (* P < 0.05). (B) Images of excised tumors at study termination of orthotropic ovarian tumors. Mean tumor weight at termination day after daily I.V. injection of PBS, T/C 9.1/54 (mg/kg) or T/C 27.5/165 (mg/kg) (* P < 0.05)

Source: Propanc Biopharma, Inc.