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Propanc Biopharma Highlights Differentiated PRP Pancreatic Cancer Data Versus Emerging Pan-RAS Program ERAS-0015

New Preclinical PDAC Package for PRP Shows >90% Tumor-Growth Inhibition & >2.5-Fold Survival Benefit; Company Contrasts Mechanism, Breadth, & Development Role Against Erasca's Phase 1 ERAS-0015 Dataset

MELBOURNE, Australia, Sept. 22, 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 issued a comparative analysis of lead candidate PRP against recently reported clinical datasets from Erasca, Inc.'s pan-RAS molecular glue ERAS-0015 in pancreatic ductal adenocarcinoma (PDAC).

The analysis follows FDA approval of Revolution Medicines' daraxonrasib in pretreated metastatic PDAC in August 2026 and FDA Fast Track designation for ERAS-0015 in metastatic pancreatic adenocarcinoma on August 24, 2026. Propanc believes these advances validate RAS as a tractable driver in PDAC while simultaneously highlighting the biology RAS inhibition leaves unaddressed — epithelial-mesenchymal transition (EMT), cancer stem cells (CSCs), fibrosis, and metastatic dissemination.

PRP, a proprietary fixed-ratio combination of the pancreatic proenzymes, trypsinogen and chymotrypsinogen (1:6), does not inhibit RAS. Instead, it promotes differentiation of malignant cells toward a more normal phenotype, reverses EMT, depletes CSCs, and remodels the fibrotic tumor microenvironment (TME). The Company believes this non-cytotoxic, differentiation-based approach is complementary to — not competitive with — RAS(ON) inhibitors and pan-RAS molecular glues, including ERAS-0015.

PRP Preclinical Profile in Advanced PDAC

In orthotopic and patient-derived xenograft (PDX) models of advanced PDAC, three-times-weekly intravenous PRP achieved:

- Greater than 90%, mean, tumor-growth inhibition versus vehicle controls ($p < 0.001$).
- Marked reduction in metastatic burden in the liver and peritoneum.
- Significant remodeling of the tumor microenvironment, including decreased cancer-associated fibroblast activity, reduced fibrosis, and suppression of EMT markers.
- Enhanced sensitivity of chemo-resistant PDAC cells to standard-of-care gemcitabine/nab-paclitaxel, supporting the potential for lower chemotherapy doses

with improved efficacy.

- Median overall survival extension of more than 2.5-fold in treated animals compared with controls.

These results complement previously reported >85% tumor-growth inhibition data and peer-reviewed findings on PRP's effects on PDAC fibroblasts. 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 and no severe treatment-related adverse events.

PRP holds FDA Orphan Drug Designation for pancreatic cancer and is not restricted to a specific RAS genotype, supporting potential broad applicability across solid tumors and possible use in combination or sequential settings with RAS inhibitors or standard chemotherapy.

ERAS-0015 Clinical Snapshot in PDAC

According to Erasca's public disclosures, ERAS-0015 is an oral pan-RAS molecular glue designed to inhibit RAS signaling, including signaling driven by mutant RAS. Preliminary Phase 1 monotherapy data from the U.S. AURORAS-1 trial and the China JYP0015M101 study have shown:

- Unconfirmed overall response rates (uORR) of 40% at pharmacologically active doses of 16–32 mg once daily and 42% at recommended expansion doses of 24–32 mg in second-line KRAS G12X PDAC.
- A July 2026 update reporting a 57% unconfirmed 8-week ORR at the 32 mg once-daily recommended expansion dose in second-line or later KRAS G12X PDAC. Responding patients remained on treatment as of May 25, 2026, data cutoff.
- Generally favorable early tolerability, with mostly low-grade treatment-related adverse events, no dose-limiting toxicities at disclosed cutoffs, and 100% median relative dose intensity at 24 mg and 32 mg once daily.
- FDA Fast Track designation for metastatic pancreatic adenocarcinoma (August 24, 2026), with Erasca outlining a planned Phase 3 PDAC trial and additional registration-oriented studies in lung cancer.

Propanc congratulates Erasca on Fast Track designation and on the early clinical activity observed with ERAS-0015. High response rates in RAS-mutant PDAC are an important advance for patients. The Company's thesis is that converting those responses into deeper, more durable remissions will require a second layer of biology — reversing the mesenchymal, stem-like, fibrotic program that enables residual disease to persist, disseminate, and resist pathway blockade.

Comparative Snapshot

Sources: *Company disclosures and peer-reviewed or conference reports as of September 2026. PRP efficacy cited is preclinical. ERAS-0015 and daraxonrasib data are from human clinical trials. Cross-modality numerical comparisons are directional only and are not head-to-head results.*

Attribute	PRP (PPCB)	ERAS-0015 (ERAS)	Daraxonrasib (RVMD)
Modality	IV proenzyme combo (trypsinogen + chymotrypsinogen, 1:6)	Oral pan-RAS molecular glue	Oral RAS(ON) multi-selective inhibitor
Primary node	Differentiation / EMT reversal / CSCs / TME	Pan-RAS (KRAS G12X and related)	Oncogenic RAS(ON) signaling
Evidence stage	Preclinical PDAC + limited compassionate use; Phase 1b planned February 2027	Phase 1 dose-escalation / expansion; Fast Track; registration path outlined	Phase 3 PDAC; FDA approved August 2026 for pretreated metastatic PDAC
PDAC activity	>90% TGI; >2.5× median OS in models; metastasis and fibrosis reduced	Ph1 2L KRAS G12X: uORR 40–42%; 57% uORR8wk at 32 mg RDE (2L+)	Ph3 2L: mOS 13.2 vs 6.6–6.7 mo; mPFS 7.3 vs 3.5 mo; ORR ~33% vs ~12%
Genotype limit	Not RAS-mutation restricted; FDA Orphan Drug Designation for pancreatic cancer	RAS / KRAS G12X-enriched populations	RAS-mutant tumors (multi-selective, not G12C-only)
Resistance biology addressed	EMT, CSCs, CAFs, fibrosis, metastasis, chemo re-sensitization	RAS output; combinations (e.g., anti-EGFR) being explored	Oncogene-addicted proliferation; adaptive MAPK reactivation remains a known class issue

Why PRP May Complement ERAS-0015 and Other RAS Agents

RAS mutations drive approximately 90% of PDAC. Oral RAS inhibitors have now produced practice-changing clinical results. Propanc's view is that turning RAS off is necessary but may not be sufficient.

Cells that survive RAS blockade are frequently mesenchymal and stem-like. EMT is the program that allows carcinoma cells to leave the primary site, hide from therapy, and return. Fibrosis and cancer-associated fibroblasts further limit drug penetration and sustain a CSC reservoir through TGF-β signaling. None of those liabilities is the primary target of a "pan-RAS molecular glue".

PRP is designed to act downstream of the GTPase:

- Proenzyme activation and PAR signaling. After intravenous administration, trypsinogen and chymotrypsinogen are activated and engage PAR-1 and PAR-2, which are frequently overexpressed on tumor cells. This cascade is associated with reduced TGF-β pathway output — a master inducer of EMT in late-stage cancer.
- Restoration of an epithelial phenotype. PRP increases epithelial adhesion proteins such as E-cadherin and β-catenin and decreases EMT transcription factors. Cells become less motile, more adherent, and more differentiated.
- Depletion of cancer stem cells. In pancreatic CSC models, PRP reduced ALDH-high

cells and surface markers CD44, CD326, and CXCR4; suppressed primary and secondary sphere formation; and impaired tumor engraftment in vivo.

- TME remodeling and chemo-sensitization. Decreased CAF activity and fibrosis can improve drug delivery. By pushing cells out of a mesenchymal, drug-tolerant state, PRP resensitized chemo-resistant PDAC cells to gemcitabine/nab-paclitaxel. The same logic applies to RAS inhibitors: a smaller mesenchymal reservoir should leave fewer cells capable of adaptive resistance.

“RAS inhibitors have rewritten what is possible in pancreatic and RAS-mutant lung cancer. That is a genuine inflection point for patients,” said Mr. James Nathanielsz, Propanc’s Chief Executive Officer. “Our thesis is that turning RAS off is necessary but may not be sufficient. The cells that survive RAS blockade are often the mesenchymal, stem-like cells that PRP differentiate and disarm. If that biology holds in the clinic, PRP could help RAS-focused companies — including programs such as ERAS-0015 — convert high response rates into longer, cleaner remissions.”

“EMT is the program that lets a carcinoma leave home, hide, and return,” said Dr. Ralf Brandt, Propanc’s Research & Development Director. “PRP does not compete with daraxonrasib or ERAS-0015 at the GTPase. It reverses the downstream identity change those tumors used to resist almost every class of drug. That is why we see suppression of EMT markers, loss of CSC phenotypes, less fibrosis, fewer metastases, and more than a two-and-a-half-fold survival extension in PDAC models. Those are the exact liabilities a RAS inhibitor leaves on the table.”

“Pancreatic cancer remains one of oncology’s greatest challenges, with five-year survival rates still near 13% and limited durable options for patients with metastatic disease,” Mr. Nathanielsz added. “We are accelerating our Phase 1b First-in-Human study in advanced solid tumors, with pancreatic cancer as a key focus indication. PRP’s orphan designation, genotype-agnostic mechanism, and complementary profile versus emerging RAS agents give us strong conviction as we move toward the clinic.”

Clinical Development Path

The Company is progressing GMP manufacturing, pharmacokinetics assay validation, and clinical partnerships in support of a planned Phase 1b First-in-Human study. The multicenter, open-label study is expected to enroll approximately 40 to 50 patients with advanced solid tumors, including pancreatic, ovarian, and other refractory cancers, with first patient dosing targeted for February 2027. A clinical trial application is expected in the coming months.

Propanc intends to evaluate PRP both as a single agent and, subject to emerging clinical data and partner interest, as a potential backbone in combination or sequential regimens with RAS-targeted therapies and standard chemotherapy.

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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