



## **PRESS RELEASE**

*Uppsala, Sweden – 8 April 2026*

### **Beactica Therapeutics announces presentation at the American Association for Cancer Research Annual Meeting on its TEAD degrader programme**

Beactica Therapeutics AB, the Swedish precision oncology company, today announced that its TEAD degrader programme has been selected for a late-breaking presentation at the American Association for Cancer Research (AACR) Annual Meeting 2026. The conference will take place on April 17–22 at the San Diego Convention Center in San Diego, California.

The presentation will include new positive results from studies with Beactica's wholly owned TEAD degrader P65-047. This will include in vivo proof-of-concept in clinically relevant models of two cancers of the lung and an ability to overcome resistance to clinically used KRAS inhibitors. The work to be presented contains contributions by Beactica's collaborators at the University of Texas M. D. Anderson Cancer Center in Houston, Texas.

#### **Poster presentation details**

**Title:** *P65-047, a novel TEAD degrader, overcomes KRAS inhibitor resistance through Hippo pathway disruption in NSCLC*

**Session:** Late-Breaking Research: Experimental and Molecular Therapeutics 3

**Location:** Poster Section 53, Board Number 19

**Abstract presentation number:** LB362

**Date:** Tuesday April 21, 2026, at 2:00 PM – 5:00 PM

**Presenter:** Dr Peter Brandt, Head of Chemistry, Beactica Therapeutics

#### **Abstract**

**Background:** Allele-specific KRAS inhibitors (e.g. MRTX849/adagrasib, MRTX1133) show clinical benefit in KRAS-mutant NSCLC but face rapid development of resistance mediated by adaptive YAP–TEAD axis activation. TEAD degraders represent a rational mechanistic partner to intercept this bypass pathway and restore KRAS inhibitor sensitivity.

**Methods:** P65-047, a cereblon-recruiting TEAD degrader, was evaluated in: (1) in vivo subcutaneous CDX models using NCI-H226 (NF2<sup>-/-</sup> mesothelioma, Hippo-dysregulated) and CALU-1 (KRAS<sup>G12C</sup>/TP53<sup>-/-</sup> NSCLC); and (2) in vitro parental and KRASi-resistant NSCLC human cell models H23 KRAS<sup>G12C</sup> and A427 KRAS<sup>G12D</sup>. TEAD protein degradation and target gene suppression were assessed by Western blot and qPCR.

**Results:** P65-047 monotherapy induced dose-dependent tumour regression in both xenograft models (vehicle-treated tumours progressed). In resistant cell models in vitro, P65-047 exhibited minimal single-agent activity but produced marked restoration of KRAS inhibitor-dependent growth suppression in combination settings: P65-047 + MRTX1133 resensitized MRTX1133-resistant A427 KRAS<sup>G12D</sup> cells, and P65-047 + MRTX849 resensitized MRTX849-resistant H23 KRAS<sup>G12C</sup> cells in cell viability assays. Mechanistic studies confirmed on-target TEAD degradation and suppression of TEAD-regulated transcripts in resistant cells.

**Conclusions:** P65-047 displays high efficacy, delivering robust in vivo tumour regression and strong resensitisation in vitro, establishing TEAD degradation as a mechanistically validated strategy to overcome acquired resistance in KRAS-mutant NSCLC. These data support clinical advancement of TEAD degrader P65-047 in patients with allele-specific KRAS inhibitor-refractory disease.

### **About Beactica Therapeutics**

Beactica Therapeutics AB is a privately held precision medicine company with a pipeline of novel small molecule therapeutics aimed at treating diseases with significant unmet medical need. Beactica's approach is centered around the Eclipsor™ platform that enables the efficient development of allosteric modulators and targeted protein degraders. Beactica deliver value to patients and shareholders by advancing its programmes to clinical proof of concept. For more information, please visit [www.beactica.com](http://www.beactica.com).

### **Beactica Therapeutics Contact**

Per Källblad *M.Sc. Ph.D.*

CEO

[per.kallblad@beactica.com](mailto:per.kallblad@beactica.com)

Tel: +46 18 56 08 80