A Phase 1/2 Study of CFT1946, a Novel BiDAC™ Degrader Targeting Mutant BRAF V600X Solid Tumors Including Metastatic Colorectal Cancer (CRC) in Combination with Cetuximab

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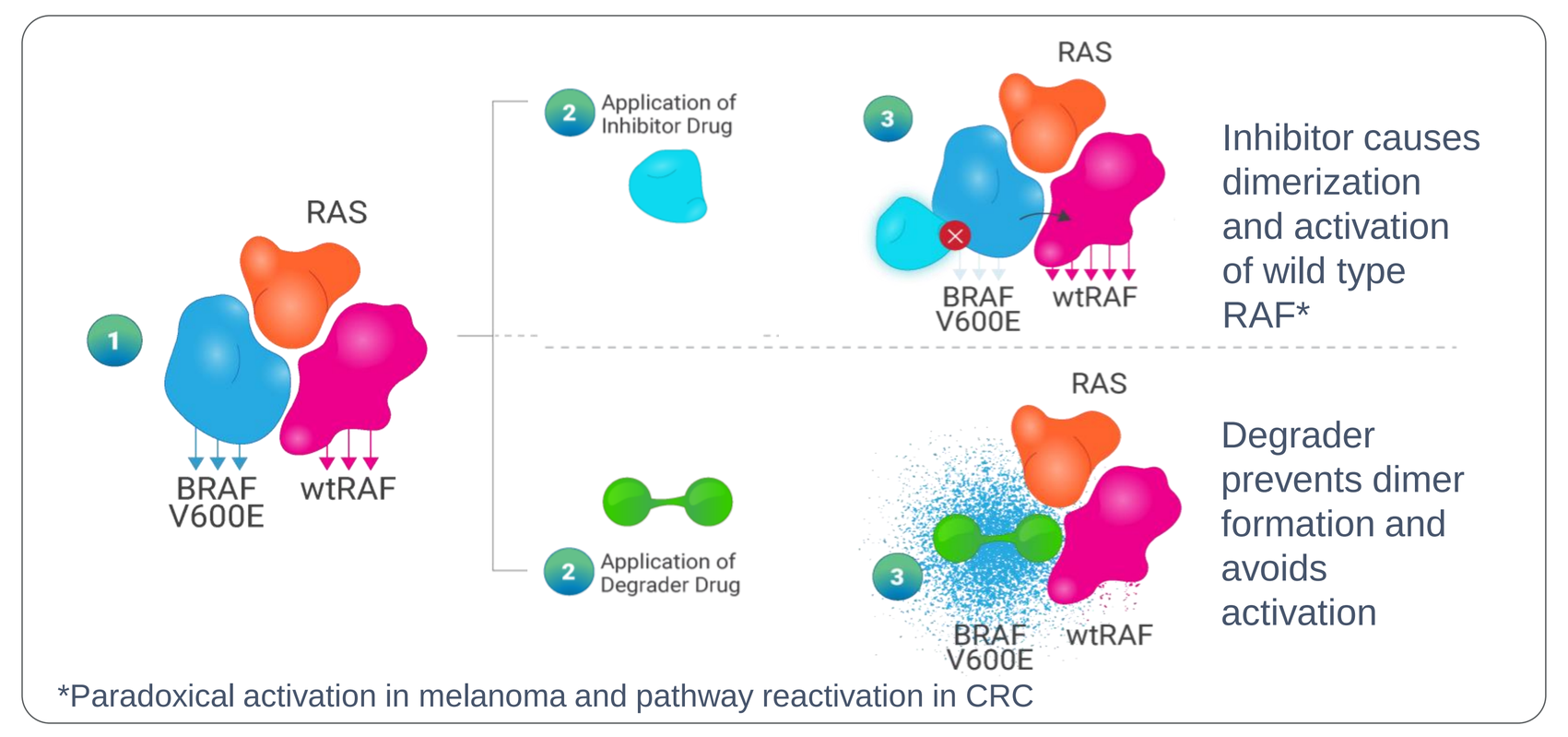
#160TiP

BACKGROUND

- BRAF is a protein kinase that acts as a signal transducer/amplifier in RTK signaling pathways, specifically, the MAPK pathway that promotes cell proliferation and survival when activated through extracellular signals[1,2]
- Constitutively active mutated BRAF, specifically BRAF V600X, is capable of uncontrolled signaling which signals as a monomer, resulting in hyperactivation of MEK, ERK, and dysregulation of cellular proliferation[2]
- BRAF V600X is a clinically validated oncology target in multiple tumor types including[1-3] CRC, melanoma, NSCLC, and ATC
- Currently approved BRAFi result in pathway reactivation in CRC and paradoxical RAF activation in melanoma as the mutant BRAF protein is still able to dimerize with wtRAF, resulting in a dimeric signaling complex and resistance[4] (Figure 1)

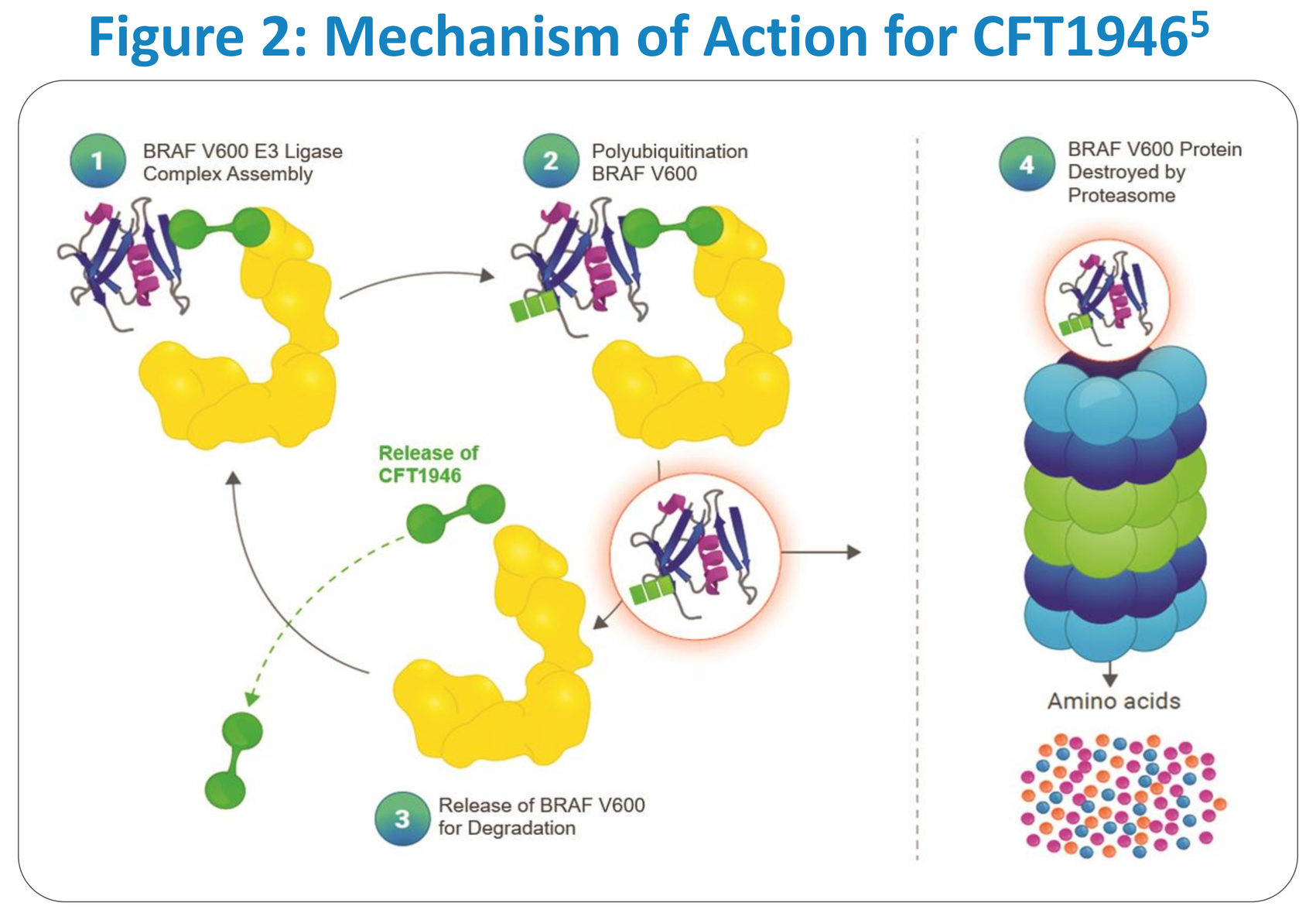
Figure 1: Utilizing a Degrader Approach to Overcome Limitations of BRAF Inhibition[5]



CFT1946 BACKGROUND

- CFT1946 is a novel, orally bioavailable, heterobifunctional, or BiDAC— degrader
  - CFT1946 selectively degrades mutant BRAF V600X protein and inhibits the MAPK pathway while sparing wtBRAF
  - Distinct from approved BRAFi, CFT1946 avoids paradoxical RAF activation as the degraded BRAF V600X mutant protein can no longer incorporate into a dimeric signaling complex (Figure 1)
- Mechanism of action (Figure 2):
  - CFT1946 induces ternary complex formation with BRAF and cereblon E3 ligase (step 1)
  - BRAF V600X is polyubiquitinated and subsequently released for degradation in the proteasome (steps 2-4)
  - CFT1946 is released to form another ternary complex; this catalytic property increases potency
- CFT1946 has demonstrated preclinical activity in BRAF V600X mutant *in vitro* and *in vivo* models, including models resistant to BRAFi (Fig 3-6)

Figure 2: Mechanism of Action for CFT1946[5]



PRE-CLINICAL DATA: *IN VITRO*

Figure 3: CFT1946 is an On-Mechanism, CRBN-Based, Highly Selective BRAF V600X BiDAC— Degrader[5]

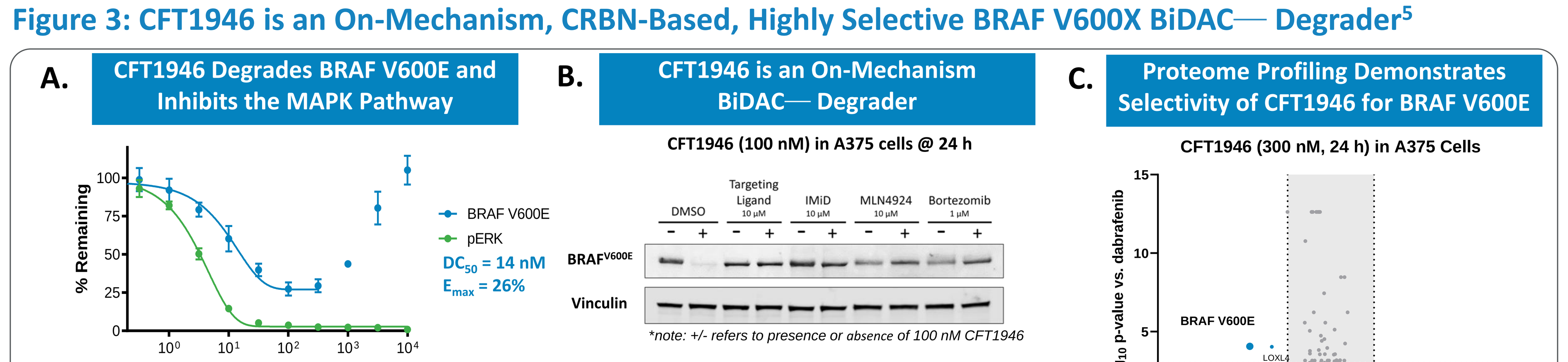


Figure 3. CFT1946 acts as a degrader of BRAF V600E as demonstrated in the HiBiT assay reported in panel **(A)** Phospho-ERK levels decrease in a CFT1946 dose-dependent manner. BRAF V600E levels decrease in a CFT1946 dose-dependent manner until "hook effect" concentrations are achieved. **(B)** Demonstrates that CFT1946 is on-mechanism for a CRBN-based BiDAC™ degrader and Figure **(C)** shows the selectivity of CFT1946 in A375 cells using global proteomic profiling.

PRE-CLINICAL DATA: *IN VIVO*

Figure 4: Dose Proportional PK and PD Profile in the BRAF V600E A375 Xenograft Mouse Model for CFT1946[5]

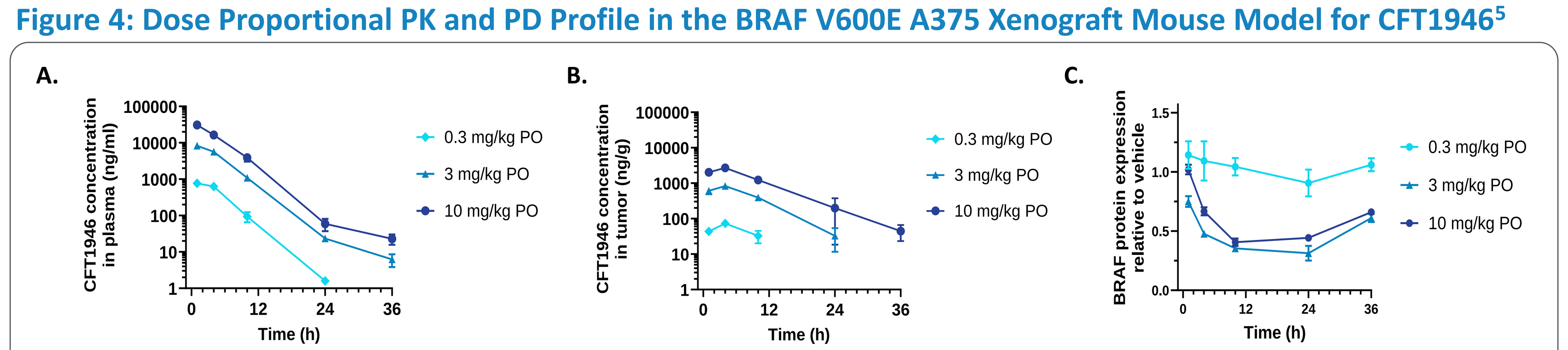


Figure 4. CFT1946 demonstrates a dose proportional pharmacokinetic exposure in plasma **(A)** and tumor xenografts **(B)**. Panel **(C)** demonstrates a dose proportional loss of BRAF V600E protein over time after a single dose.

PRE-CLINICAL DATA: CRC *IN VIVO* CDX

Figure 5: BRAF V600E HT-29 CRC CDX Model Exhibits Greater Response to CFT1946 Alone and in Combination with Cetuximab Compared to Approved BRAFi/EGFRmAb[5]

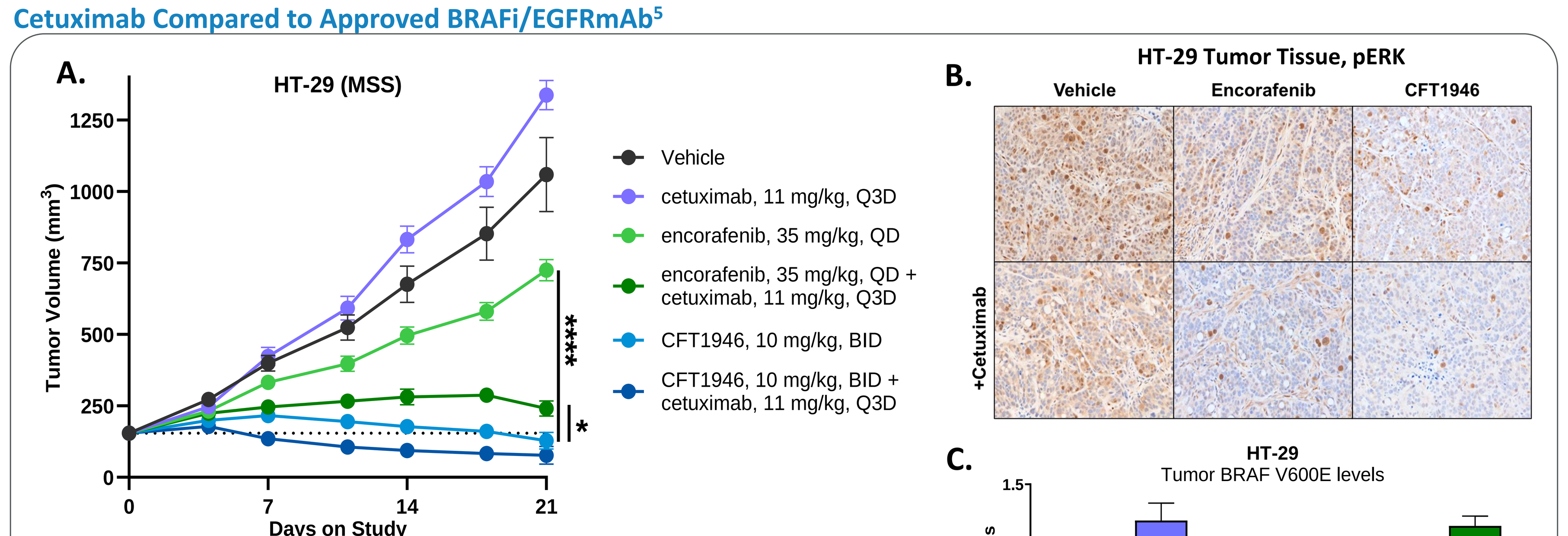


Figure 5. (A) *In vivo* efficacy of CFT1946 or encorafenib with or without cetuximab combination demonstrates degrader advantage in the BRAF V600E heterozygous colorectal cancer model HT-29, microsatellite stable (MSS) model. Dotted line indicates starting tumor size. Unpaired t-test was performed at the last time point (∗p < 0.05 and ∗∗∗∗p < 0.0001). Efficacy data are expressed as mean tumor volumes ± SEM. n= 6 mice per group. **(B)** Representative images of tumor tissue collected at end of study at 20x magnification. CFT1946 treatment results in greater suppression of pERK than the inhibitor or cetuximab alone. CFT1946 in combination with cetuximab has the greatest pERK suppression by IHC, correlating with the deepest level of tumor growth inhibition in the efficacy study. **(C)** Corresponding end of study tumor BRAF V600E protein levels from efficacy study demonstrate BRAF degradation in CFT1946 treated mice.

Figure 6: Robust Efficacy Response Observed in Multiple CDX Models in Colorectal Cancer[5]

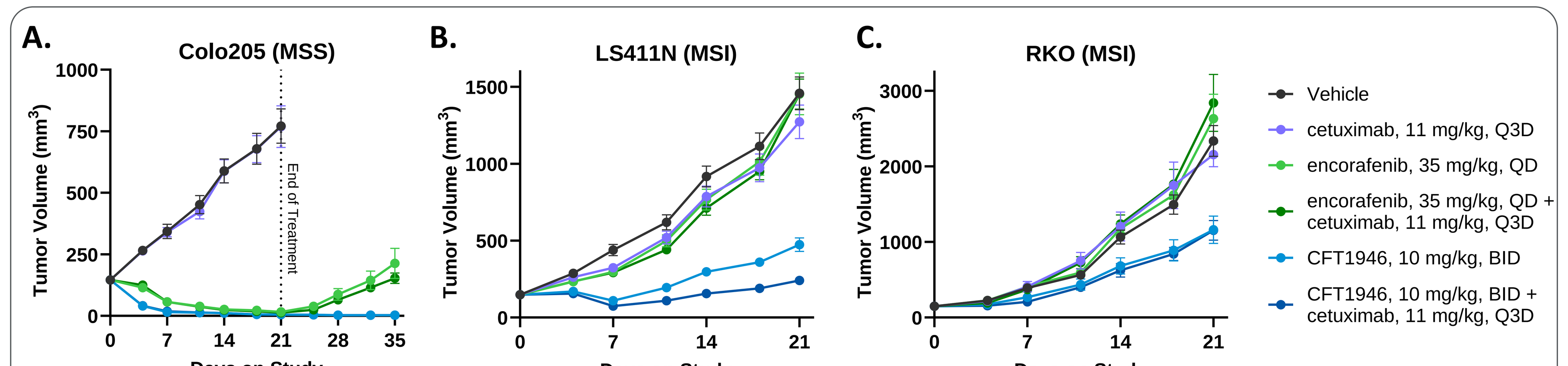
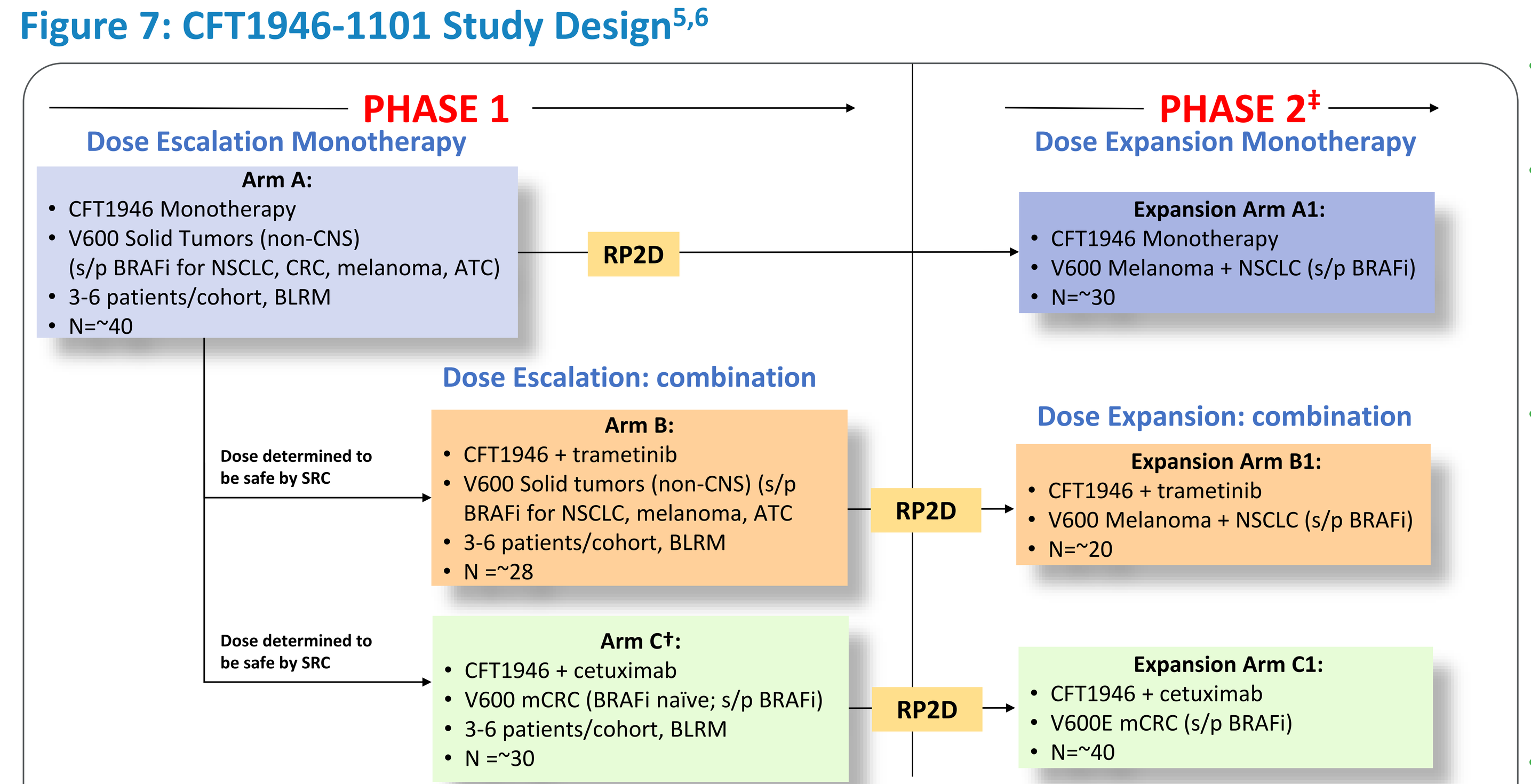


Figure 6. (A) Efficacy study of the BRAF V600E CRC model Colo205. Treatment was ceased at day 21 and monitored for outgrowth. CFT1946 treatment resulted in a more durable suppression of tumor growth with tumors still undetectable 14 days after removal of treatment in 7 out of 8 mice. **(B)** *In vivo* efficacy study with the microsatellite instable (MSI) CRC model LS411N demonstrated strong tumor growth inhibition with CFT1946 treatment and further suppression of tumor growth with addition of cetuximab. **(C)** CFT1946 treatment in the MSI CRC model RKO suppressed tumor growth but was not further benefited by addition of cetuximab. Efficacy data are expressed as mean tumor volumes ± SEM. n= 6 mice per group.

Phase 1/2: FIRST-IN-HUMAN CLINICAL STUDY DESIGN[6]

Figure 7: CFT1946-1101 Study Design[5,6]



CFT1946 is administered orally in 28-day cycles until progression or intolerable toxicity; additional patients may be enrolled at dose levels deemed safe by SRC (N=15-30)
Phase 1 expansion cohorts: additional patients to be enrolled at selected doses and tumor types (N=30)
†Phase 1 arm will include BRAF V600X mutant CRC with prior BRAFi or BRAFi naïve, if BRAFi is not available per local SoC.
‡Phase 2 will be initiated once the RP2D has been identified. Eligible subjects are ≥18 years-old with documented BRAF V600X mutant cancers who have received ≥1 prior therapy.

- Open-label, multicenter, Phase 1/2 clinical trial with dose escalation and expansion phases
- Dose escalation Phase 1 includes 3 arms:
  - CFT1946 (Arm A) with a starting oral dose of 20 mg twice daily, currently enrolling dose level 5
  - CFT1946+trametinib (Arm B)
  - CFT1946+cetuximab (Arm C)
- Dose expansion Phase 2 includes 3 arms: CFT1946 (Arm A1), CFT1946+trametinib (Arm B1), and CFT1946+cetuximab (Arm C1)
  - Arms A1 and B1 will include BRAF V600X mutant melanoma or NSCLC with prior BRAFi therapy
  - while Arm C1 will enroll CRC with prior BRAFi therapy
- N≈206 across 16 US and European sites will be enrolled
- Registered on ClinicalTrials.gov as NCT05668585; study is open for enrollment

FIRST-IN-HUMAN STUDY DESIGN[5,6]

KEY ELIGIBILITY CRITERIA

KEY INCLUSION CRITERIA

- ≥18 years of age at time of informed consent
- Documented evidence of a BRAF V600X mutation obtained from tumor tissue or liquid biopsy
- Received ≥1 prior line of SoC therapy for unresectable locally advanced or metastatic disease, melanoma, NSCLC, CRC, ATC or other BRAF V600X mutation-positive tumors
- Adequate bone marrow, liver, renal, and cardiac organ function

KEY EXCLUSION CRITERIA

- Subject has had major surgery within 21 days prior to the planned first dose.
- Subject with CNS involvement (primary tumor or metastatic disease), except if clinically stable
- Subject with known malignancy other than trial indication that is progressing or has required treatment within the past three years, except for conditions that have undergone potentially curative therapy

STUDY ENDPOINTS[6]

PRIMARY ENDPOINT

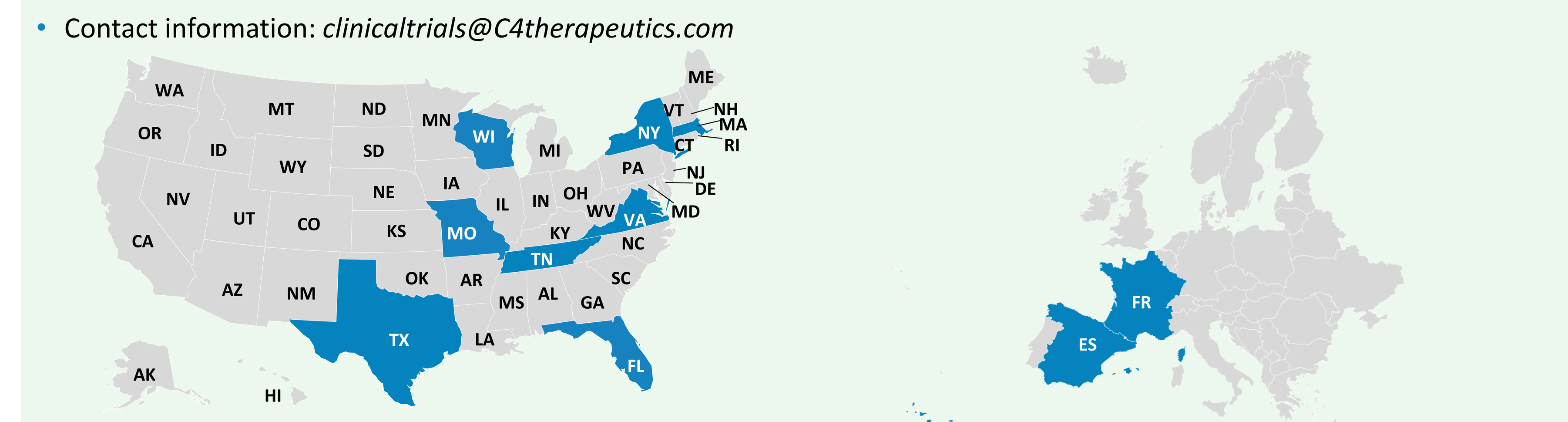
- Frequency and severity of AEs and SAEs of CFT1946 (Phase 1)
- Incidence of DLTs (Phase 1)
- Number of participants with changes between baseline and post-baseline safety assessments (Phase 1)
- Frequency of dose interruptions and dose reductions (Phase 1)
- Frequency of AEs leading to discontinuation (Phase 1)
- ORR (Phase 2)

SECONDARY ENDPOINTS

- Frequency and severity of AEs and SAEs of CFT1946 (Phase 2)
- Frequency of dose interruptions and dose reductions (Phase 2)
- Assessment of PK and PD
- PK-QTcF relationship
- ORR (Phase 1)

STUDY STATUS/ENROLLMENT

- The study opened to accrual in December 2022 and is currently enrolling dose level 5
- N≈206 patients from 16 sites* in the US and Europe[6]
- Trial registration: NCT05668585
- Contact information: *clinicaltrials@C4therapeutics.com*



*Blue colored states/countries indicate clinical trial sites

Abbreviations
AE, adverse event; **ATC**, anaplastic thyroid cancer; **BLRM**, Bayesian logistic regression model; **BRAF**, v-raf murine sarcoma viral oncogene homolog B1; **BRAFi**, BRAF inhibitor, **BRAFV600,** BRAF with Valine 600 mutation; **CDX**, cell-derived xenografts; **CNS**, central nervous system; **CRBN**, cereblon; **CRC**, colorectal cancer; **DLT**, dose limiting toxicities; **ERK**, extracellular signal-regulated kinases; **HiBiT**, high affinity bioluminescent tag; **MAPK**, mitogen-activated protein kinase; **MEK**, mitogen-activated protein kinase kinase; **NSCLC**, non-small cell lung cancer; **ORR**, overall response rate; **pERK**, phospho-ERK; **PD**, pharmacodynamics; **PK**, pharmacokinetics; **PO**, by mouth; **PoM**, proof of mechanism; **QTcF**, Fridericia heart-rate-corrected QT interval; **RAF**, rapidly accelerated fibrosarcoma; **RP2D**, recommended phase 2 dose; **RTK**, receptor tyrosine kinase; **SAE**, serious adverse event; **s/p**, status post; **SoC**, standard-of-care; **SRC**, Safety Review Committee; **TGI**, tumor growth inhibition; **wt**, wild-type.

Disclosures
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References
1. Ascierto PA, et al. *J Transl Med*. 2012;10:85.
2. Zheng G, et al. *BMC Cancer*. 2015;15:779.
3. Chang CF, et al. *Am J Cancer Res*. 2022;12(11):5342-5350.
4. Hoyer S, et al. *Int J Mol Sci*. 2021;22(21):11951.
5. C4 Therapeutics data on file.
6. NCT05668585. www.clinicaltrials.gov. Accessed June 11, 2024.