

A Critical Evaluation of Cardiff's Pipeline:

Why Cardiff Oncology's onvansertib will fail Phase 2 clinical trials and the stock will trade to \$1

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NOTE: This is a VERY rough draft written just days before a key catalyst (data readout on the 29th) and was not finished beforehand. Due to this there is a continuation section discussing the data and future of this company. Data not finished before the readout is left in its unfinished state.

Executive Summary

In this work, I will prove that the onvansertib Phase 2 trial will fail to meet its primary endpoint. The overwhelming odds that onvansertib can redo a trial and achieve significance this time due to post-hoc data mining in certain subgroups is low to none and the preliminary data that management says is indicative of a benefit is lacking due to small sample sizes and given a larger data set should repeat the same outcomes as before.

Introduction

Small tech biotech stocks trying to find the next investment or get more funding to stay alive usually try to find correlations in the data and come up with subgroups in post-hoc analysis. However, this is very flawed because hypothesis is meant to be tested prospectively and retrospective analysis against any set of data will more likely find false positives than actual correlation. In the case of Cardiff after failure in RAS-mut mCRC they found that Bev naïve patients (a drug used in combination of chemo regimens) respond better than Bev-exposed patients. Obviously with only one drug and many failures they need to show any promise in their pipeline for investments and capital raising if they just gave up on the trial, they'd be left with no way to raise equity. They have done this before in a leukemia trial for the same drug where they claimed all patients with CR hadn't been previously treated with HMA although no significance or further studies were conducted. Sounding familiar? It's a Sisyphean struggle for small biotech's lacking capital, buy a drug, fail, find a subgroup, raise capital, fail in the subgroup, find a new drug. Drugs don't work by accident, most drugs fail.

History of Onvansertib

Originally developed by Nerviano Medical Sciences in 2011 (Beria I, 2011)? And licensed by Trovogene (now Cardiff Oncology) in 2017 according to prospectus. Originally developed for Leukemia. Cardiff Oncology did multiple trials in mCRC, Leukemia, and Prostate Cancer.

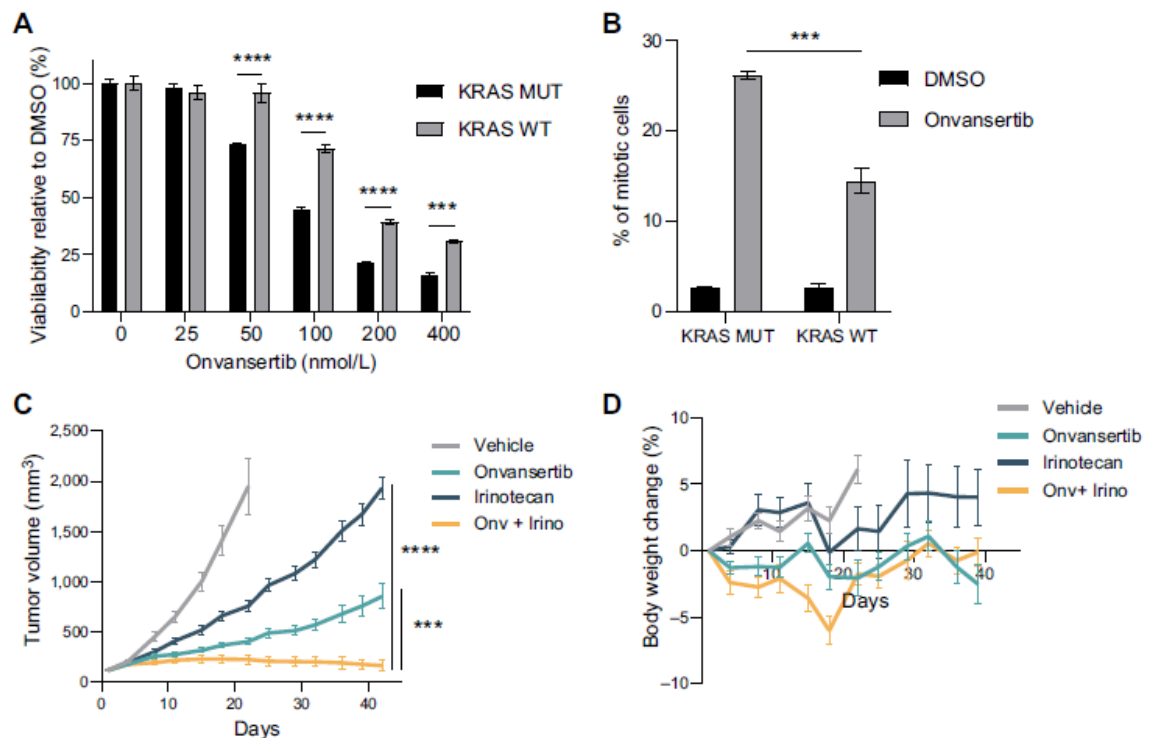
NMS-P937 -> NMS-1286937 -> PCM-075 -> onvansertib

Mechanism of Action

(Smith, 2017).

PLK1 inhibitor ATP competitive (Weiss GJ, 2017) to bind to PLK1 instead of ATP and inducing mitotic arrest and apoptosis at certain thresholds of concentration, however very toxic at high doses and induces thrombocytopenia through apoptosis bone marrow.

KRAS-mut cells are more sensitive to PLK1 inhibition (Ahn, 2024). Phase 1b



The data suggests PLK1 inhibition to be effective in KRAS-mut cells. However, the high toxicity seen in human trial's failures achieve significance seems to show that the doses at which the drug is effective are too toxic. 37.5% ORR n=7? or 16; PFS and DOR not S.S.

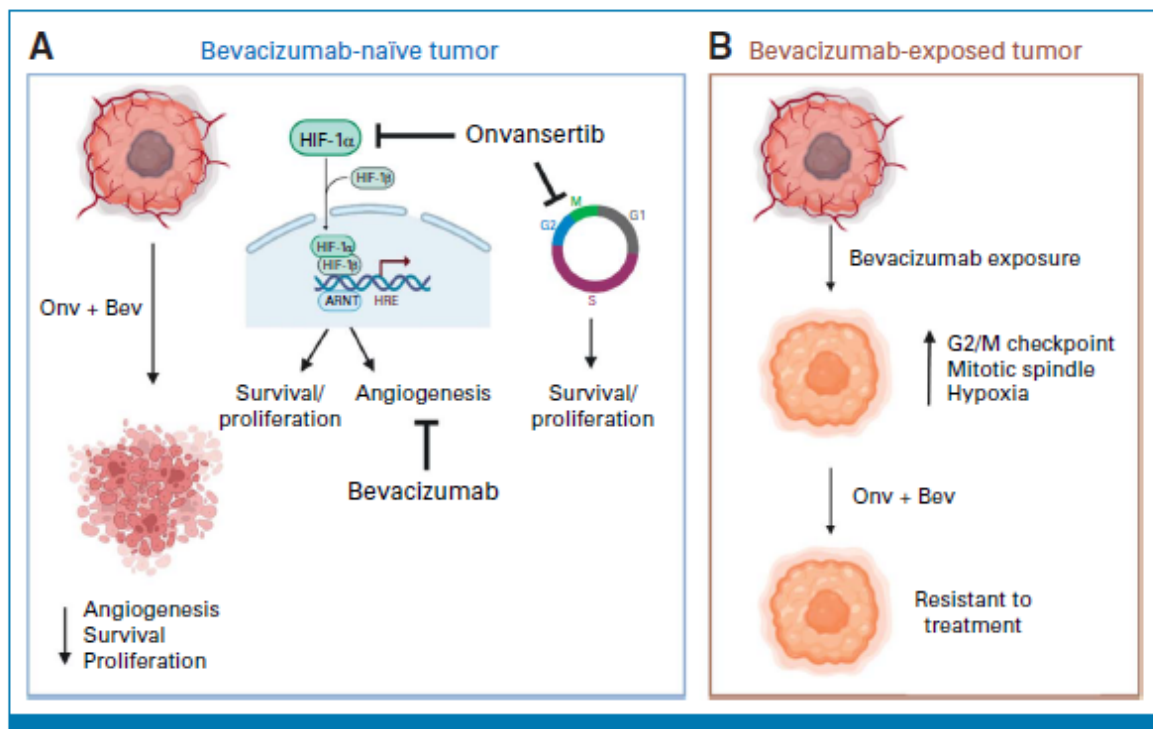


FIG A5. Graphical summary of the proposed mechanisms of Onv and Bev combination therapy in Bev-naïve and Bev-exposed tumors. (A) In Bev-naïve tumors, the combination of Onv and Bev effectively inhibits tumor cell survival, proliferation, and angiogenesis through the indicated mechanisms. (B) In Bev-exposed tumors, Bev exposure leads to upregulation of mitotic and hypoxia pathways resulting in resistance to both Onv and Bev. Figure was created with BioRender.com. Bev, bevacizumab; HIF1 α , hypoxia-inducible factor 1 α ; Onv, onvansertib.

Fabricated explanation for why a Bev-exposed tumor would be resistant to onvansertib. It is important to note that the company never mentioned anything about angiogenesis being key to the drugs effectiveness before the post-hoc data mining.

Failure of another PLK1 inhibitor BI-2536 in pancreatic cancer (Mross, 2012).

Preclinical

Preclinical data showed $F=0.23$ or 23% bioavailability and significant anti-tumor growth in the 7g compound (60mg/kg). The toxic dose was 17mg/kg^2 and the ones used in the Phase II trials are 15mg/kg^2 . In an average human if we use allometric scaling $60\text{kg } 1.62\text{m}^2$ we find a 0.41mg/kg dosing. (Beria I, 2011) Inhibits the hypoxia pathway and shows antitumor activity through angiogenesis. (Ahn DH, 2025)

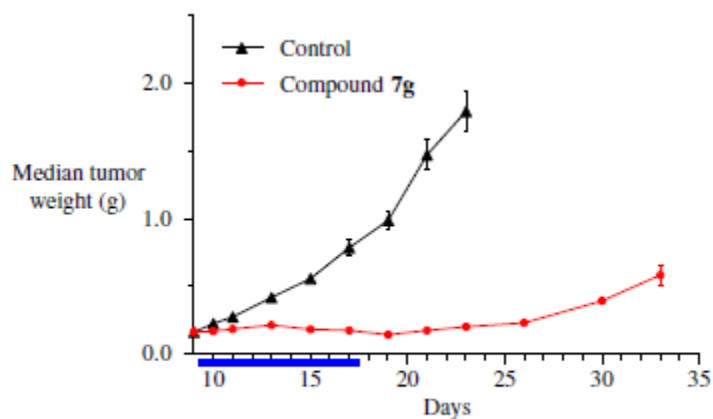


Figure 2. In vivo oral antitumor activity of 7g at 60 mg/kg, once a day, 1–10 consecutive days (blue line) on HCT116 xenograft model.

Image taken from (Ye, 2005)

Compound	R ²	PLK1 IC ₅₀ ^a (μM)	PLK2 IC ₅₀ ^a (μM)	PLK3 IC ₅₀ ^a (μM)	A2780 IC ₅₀ ^a (μM)	Solubility pH 7 (μM)
7g	-(CH ₂) ₂ -OH	0.002 ± 0.001	>10	>10	0.042 ± 0.007	201
2	-CH ₃	0.003 ± 0.001	3.519 ± 0.063	1.439 ± 0.078	0.021 ± 0.005	72
7e	-(CH ₂) ₂ -NH ₂	0.033 ± 0.012	>10	>10	1.307 ± 0.388	202
7d	-(CH ₂) ₂ -O-THP	0.043 ± 0.003	>10	1.002 ± 0.264	0.126 ± 0.027	35
7f	-(CH ₂) ₃ -NH ₂	0.052 ± 0.021	>10	>10	0.846 ± 0.186	186
7a	-(CH ₂) ₃ -N-(CH ₃) ₂	0.279 ^b	>10	>10	2.501 ^b	200

^a Values are means of three experiments.

^b Single data.

Table 3
In vitro ADME^a properties of selected compounds

Compound	Solubility 10% Tween 80 (mg/mL)	Permeability Caco-2 ^b (P _{app})	PAMPA ^c (P _{app} 10 ⁻⁶ cm/s)	Cl _{int} (mL/min/kg) rat hepatocytes (1 μM)	Cl _{int} (mL/min/kg) HLM ^d (1 μM)
2	>3.2	High	50.00	600 ± 15	25.70 ± 0.20
5a	3.7	Moderate	46.41	638 ± 21	24.50 ± 0.31
5b	3.8	High	36.78	603 ± 12	15.95 ± 0.06
7g	>3.4	Moderate	49.59	165 ± 7	16.90 ± 0.15

^a Absorption, distribution, metabolism and excretion.

^b Permeability class has been ascribed using Ranitidine and *N*-acetyl-α-phenylalaninamide as low or high permeable reference compounds, respectively.

^c Parallel artificial membrane permeability assay.

^d Human liver microsomes.

Table 4
In vivo pharmacokinetic parameters^a ± standard deviation of selected compounds in CD1 nu/nu mice^b

Compound	PK data (iv), dose ^c : 10 mg/kg				PK data (po), dose ^c : 10 mg/kg			
	AUC _∞ (μM h)	CL (L/h/kg)	V _{ss} (L/kg)	t _{1/2} (h)	C _{max} (μM)	AUC _∞ (μM h)	t _{1/2} (h)	F ^d (%)
2	4.97 ± 0.71	4.21 ± 0.45	3.39 ± 0.03	0.92 ± 0.12	0.29 ± 0.04	1.04 ± 0.10	1.44 ± 0.29	21
7g	8.57 ± 1.18	2.36 ± 0.33	1.71 ± 0.11	0.89 ± 0.01	0.64 ± 0.24	2.04 ± 0.50	1.60 ± 0.57	24

^a Data of compounds **2** and **7** were compared statistically using Student's *t*-test; *P* < 1% for CL and V_{ss}, *P* = 1.1% for AUC_∞ and *P* > 5% for t_{1/2} after iv administration were found; *P* = 2.5% for AUC_∞ and *P* > 5% for C_{max} and t_{1/2} after oral administration were calculated.

^b *n* = 3 animals per study.

^c Dosed as HCl in situ salt/glucosate.

^d Bioavailability.

Image taken from (Ye, 2005)

As we see in the image above the multiple compounds tested the 7g one being our key candidate named onvansertib we are discussing today. This compound seems acceptable at best (probably why the drug was sold to now Cardiff after the Leukemia failure in phase I). The low bioavailability, low half-life, and low AUC in the oral 7g compound shows some PK challenges.

Clinical Trials

Phase I n=21 NCT01014429

Before Cardiff licensed the drug from Nerviano Medical Sciences hence the compound name (NMS-P937/ NMS-1286937) they ran a Phase I in 2011 to determine the PK/PD and the MTD and DLTS (Weiss GJ, 2017). This Phase I led to the subsequent discontinuation of development by Nerviano and acquisition by now known as Cardiff. In the Phase I post-hoc analysis Nerviano found RAS-mut mCRC patients to seemingly respond better due to 3 out of 5 instances of stable disease being in KRAS mutant tumors, which doesn't seem to be much of a correlation due to small n, but this is probably what convinced Cardiff decided to do the trials in this population and it is interesting to note that the company that claimed this didn't proceed with the trials in this population.

Open Label metastatic solid tumors first-in-human trial. PLK1 inhibitors are highly sensitive to thrombocytopenia TEAEs and bone marrow. (Weiss GJ, 2017) MTD and RP2D at 24mg/m²/day. "PLK inhibitors to induce mitotic cell cycle in rapidly proliferating blood cells and have also been observed with BI-2536 and volasertib"

Biomarkers in both pThr199 and pHH3 did not statistically increase in skin samples collected from patients, n=2 24mg/m² and 36 mg/m², and concentrations were below the threshold expected to modulate biomarkers in tumor and skin tissue based on preclinical studies. (Weiss GJ, 2017) This was from a Nerviano study which led to subsequent discontinuation and leasing of the drug to Cardiff, the weak biomarker data in high dosages (more than double the 15mg/m² in our upcoming trial) is likely a key reason.

"In the present study with NMS-1286937, disease stabilization was observed as the best response in 5 out of the 16 evaluable patients (26.3%), while no objective responses were observed." "3 out of 5 instances of stable disease in our study were observed in patients with KRAS mutant tumors" (Weiss GJ, 2017)

Phase Ib n=

Leukemia P.E failed (Zeidan AM, 2020)

Table 3. Cycle 1 day 5 mean \pm SD pharmacokinetic parameters of onvansertib.

Dose (mg/m ²)	Combination treatment	Number of patients	t_{\max} (hour)	C_{\max} (nmol/L)	AUC ₍₀₋₂₄₎ (nmol/L·hour)	$t_{1/2}$ (hour)
12	Decitabine	4	2.5 \pm 0.6	163 \pm 90	2,270 \pm 1,440	26 \pm 16
12	LDAC	3	2.0 \pm 1.0	153 \pm 84	2,150 \pm 833	32 \pm 21
18	Decitabine	3	2.7 \pm 0.6	230 \pm 129	3,380 \pm 1,740	21 \pm 7
18	LDAC	3	3.0 \pm 1.0	109 \pm 39	1,730 \pm 847	33 \pm 18
27	Decitabine	3	2.0 \pm 1.7	411 \pm 118	5,420 \pm 402	16 \pm 6
27	LDAC	3	2.7 \pm 1.5	340 \pm 219	4,470 \pm 1,450	18 \pm 5
40	Decitabine	4	3.3 \pm 1.0	539 \pm 228	8,050 \pm 3,380	25 \pm 15
40	LDAC	3	1.7 \pm 1.2	350 \pm 104	4,270 \pm 1,950	16 \pm 7
60	Decitabine	3	3.3 \pm 1.2	1,040 \pm 534	16,300 \pm 6,690	47 \pm 8
60	LDAC	5	2.4 \pm 1.1	905 \pm 330	12,700 \pm 4,850	37 \pm 13
90	Decitabine	6	3.3 \pm 0.8	1,310 \pm 806	21,800 \pm 18,400	30 \pm 11

Phase Ib/II n=

Acute myeloid leukemia P.E failed (Croucher PJP, 2023) Was expected due to the failure of Volasertib (same MOA) also in acute myeloid leukemia with a 25% ORR p=0.071 OS p=0.757 (Dohner, 2021).

Claimed that because all patients with CR or Cri hadn't previously been treated with an HMA that could be the group that its effective in. 27% ORR (Croucher PJP, 2023).

Phase Ib n=23 NCT05593328

In RAS-mut mCRC (Ahn DH, 2024)

Phase II n=72 NCT03414034

In mCRPC Failed P.E

Phase II n=68 NCT03829410

1L ORR in NCT03829410 = 10%; 2L = 77%; Even if the drug were to show a response in 2L (Bev naïve) patients the population ~9,500 as per press release, it wouldn't be worth the funding required for further research and Pfizer? would stop working with them ~80m in cash probably down to <60m by now is not enough for further trials and requires heavy dilution. In the overall population however, Failed P.E of ORR>30% ORR was 26.4% n=53 DOR not S.S. Used post-hoc analysis (n=4; wide SD; according to my t-test calculations p=0.8 in entire subgroup, p=0.1 in 30mg, 20mg even worse) to say Bev naïve patients responded better (they should respond better anyways?) Grade $\frac{3}{4}$ AE in 62%. (Ahn, 2025) In clinical studies you have equal populations in this case of Bev-exposed and Bev-naïve due to the better responses to Bev seen in Bev naïve patients so it doesn't affect the outcome of the trial but I hypothesize that comparing Bev naïve to Bev naïve populations they will have a higher ORR in both control and active arms but won't be a significant difference.

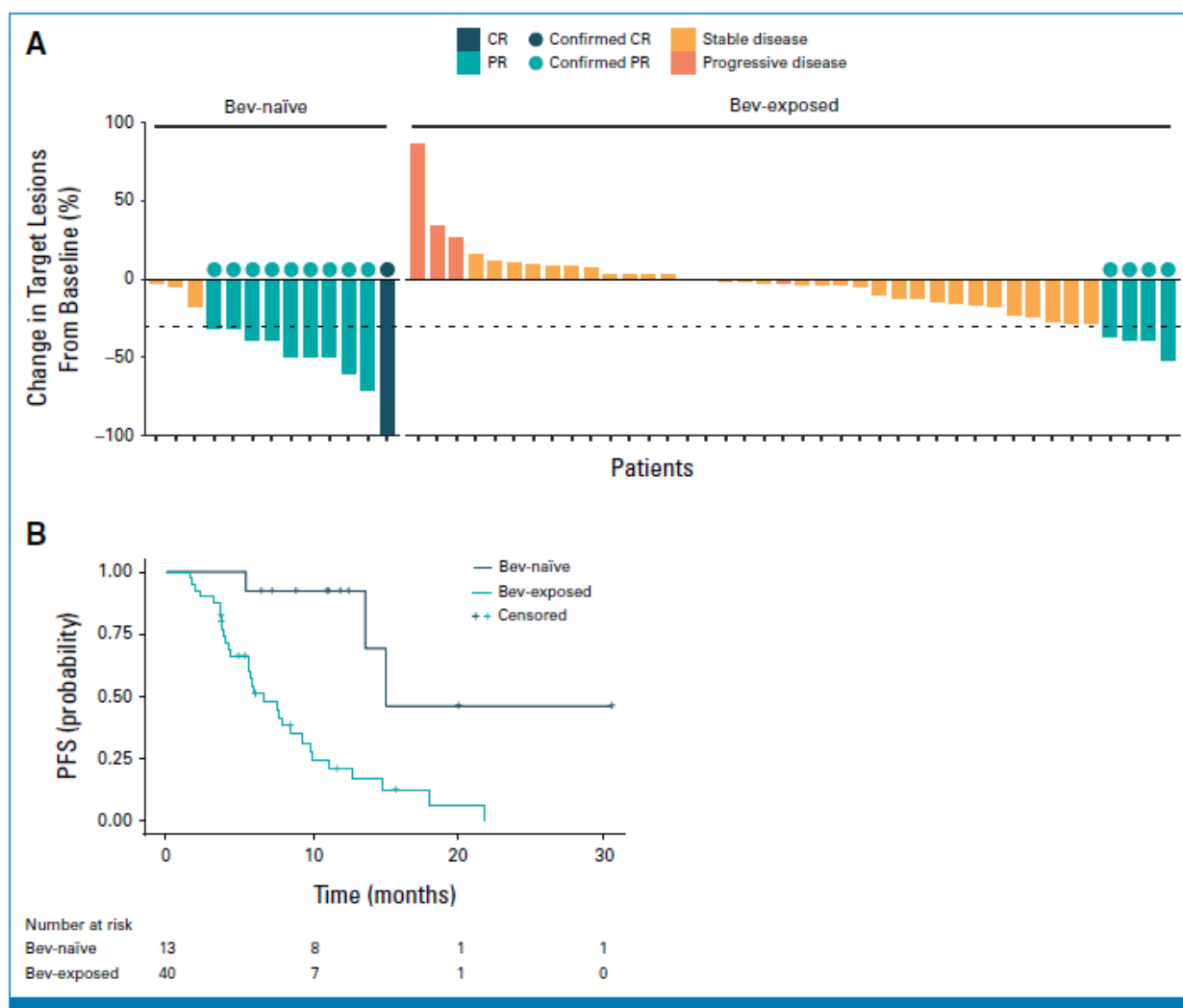


FIG 2. Antitumor activity according to prior bevacizumab treatment. (A) Best percentage change from baseline in target lesions. The dashed line at -30% change represents the RECIST v1.1 cutoff to define PR. Confirmed responses are indicated. (B) Kaplan-Meier curve of the PFS. Bev, bevacizumab; CR, complete response; PFS, progression-free survival; PR, partial response.

Patients not previously exposed to Bev are shown to have a better response than Bev-exposed and this post-hoc analysis seems uncorrelated to the MOA of PLK1 and how it would be more effective in a Bev naïve only population the ORR of SoC vs SoC+onvansertib should not be a significant difference.

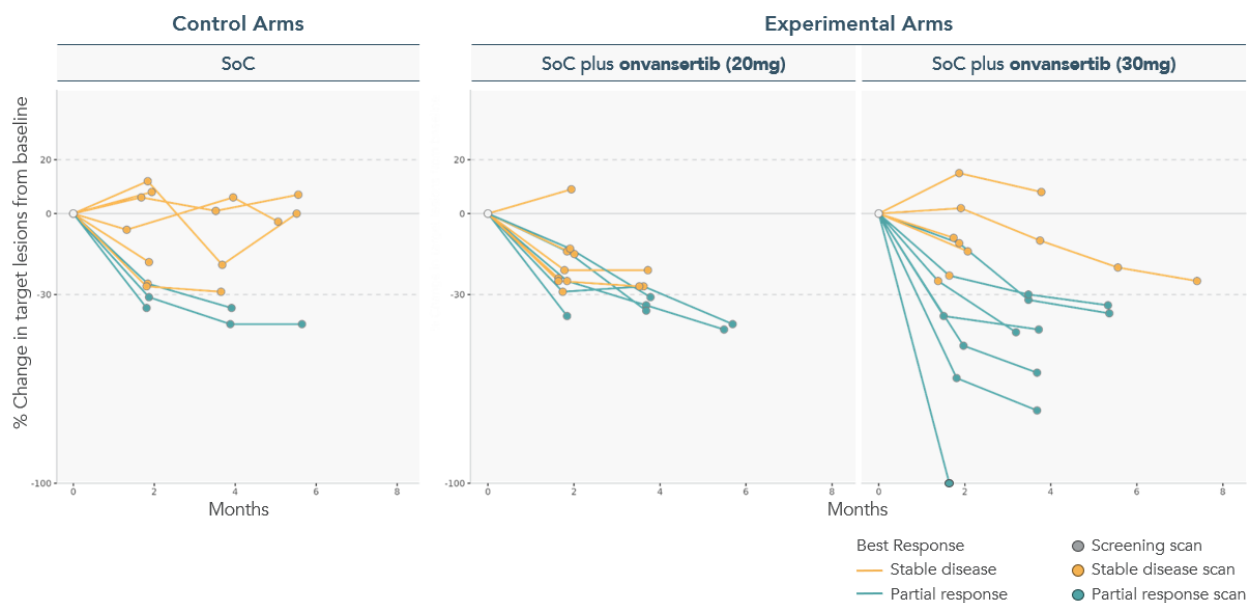
Phase II n=113 NCT06106308

In the FORWARD study for paclitaxel, they found Bev-naïve patients with a 59% ORR compared to 35% in BEV pretreated (Husam, 2025); comparatively in the onvansertib study ORR was 77% for Bev naïve and 10% for BEV-exposed showing a clear distinction between patients' response to Bev being a lot higher in naïve than pretreated. So, is onvansertib driving some or none of the ORR? It seems likely from previous trials and PK that Bev is driving most if

not all the ORR at least enough that it would not achieve significance comparing control to onvansertib.

Control Arm (SoC alone)	20mg dose of onvansertib + SoC	30mg dose of onvansertib + SoC	All onvansertib patients
33% ORR (3 of 9)	50% ORR (5 of 10)	64% ORR (7 of 11)	57% ORR (12 of 21)

Radiographic Response over Time* – as of November 26, 2024



Management likes to claim the preliminary data was good 64% ORR vs 33% in 30mg vs control! That must be great data, right? However, doing a fisher's exact test (instead of a t-test due to small sample size) we find the p values for each group as $p=0.65$, $p=0.37$, $p=0.427$; respectively for 20mg, 30mg, and combined doses. This preliminary data is lacking due to small sample size and no statistical significance. The overwhelming odds are that if you run a trial with the same drug in the same population that failed if the previous trial was structured well, it should not work this time.

But can the new data or the full data show significance? We cannot draw any conclusions on a dataset this low sample size but looking at previous observations it seems highly unlikely to show a significant benefit.

Conclusion

Condition 1: Bev-naïve patients were responding better due to onvansertib and not because they will naturally respond better to the SoC (Bev) anyways even though the n=4 post-hoc wasn't S.S. The previous failure in whole population of RAS-mut mCRC can be thrown out and previous failures of PLK1 can be thrown out. Probability at most 20%

Condition 2: The drug can overcome the PK challenges at the current dosing and the results of previous trials and biomarker data can be thrown out. Probability at most 20%

Condition 3: The preliminary data is indicative of a benefit in the 30mg, and large sample sizes will show the same benefit without high PK. Probability at most 50%

The total probability of this drug succeeding in the RAS-mut mCRC Phase 2 is at most 2%. Their sole asset is onvansertib and once the drug fails again, I'm sure they will not find any partners to do further studies on this drug and will trade at book value.

Continuation

As of July 29th, 2025, more data from Cardiff's trial have been released and I will now begin to analyze and compare it to previous interim results. Since last time the stock has been trading from 4\$ down to 2.40\$ in the AH of the 29th. However, management has decided to go forward with a phase 3 trial. So, what warrants this trial? Also keep in mind the data released on July 29th is from patients collected up to July 8th as listed below images.

	Control Arm (SoC alone) (n=37)	20mg dose of onvansertib + SoC (n=36)	30mg dose of onvansertib + SoC (n=37)
Confirmed ORR ^a	30% (11 of 37)	42% (15 of 36)	49% (18 of 37)
Confirmed ORR at 6-months ^a	22% (8 of 37)	33% (12 of 36)	46% (17 of 37)
ORR ^b	43% (16 of 37)	50% (18 of 36)	59% (22 of 37)

July 8, 2025, Interim Data (above)

The new data as I suggested before shows a deterioration of data with increasing ORR in control and decreasing ORR in the drug from before.

Control Arm (SoC alone)	20mg dose of onvansertib + SoC	30mg dose of onvansertib + SoC	All onvansertib patients
33% ORR (3 of 9)	50% ORR (5 of 10)	64% ORR (7 of 11)	57% ORR (12 of 21)

November 26, 2024, Interim Data (above)

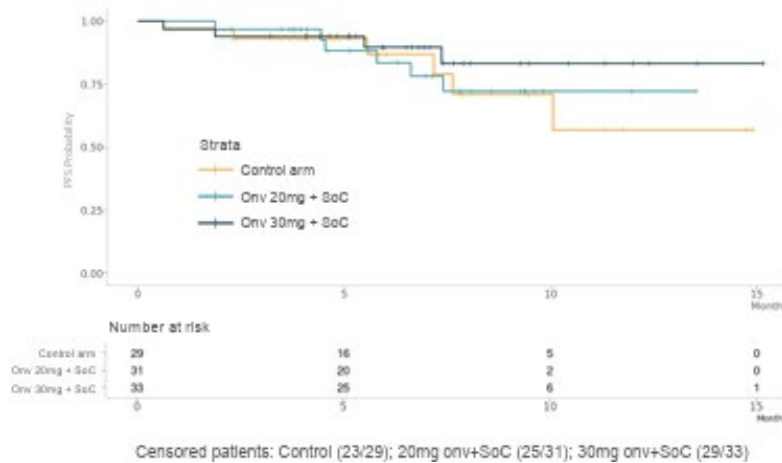
The original data with low n suggested a dose response. However, with the new data the difference has deteriorated from a 31% difference between control and 30mg to a 16% difference. The newly added patient data comes in at 46%, 50%, and 58% ORR respectively for control, 20mg, and 30mg. With further studies I hypothesize an even greater shrinkage of difference due to the small sample size of this population, and we are currently seeing that. The 20mg has become indistinguishable from control and the 30mg difference is statistically insignificant and this drug seems doomed to fail in larger studies.

Intent-to-treat (ITT) (N=110)	Control (SoC alone) (n=37)	Onv 20mg + SoC (n=36)	Onv 30mg + SoC (n=37)	Onv 30mg vs. Control
Confirmed ORR ¹ n, [95% CI]	30% n=11 [16-47]	42% n=15 [26-59]	49% n=18 [32-66]	19% p=0.018

July 8, 2025, Interim Data showing p value mistake (above)

One mistake the company seemed to have made was the p=0.018 in the Confirmed ORR table. We currently do not have an explanation for this and running a Fischer's exact test and a Chi Test on the data we find the respective p values to be 0.15 and 0.1, not statistically significant as the presentation would like to say.

Progression Free Survival – Median PFS Not Reached*



Hazard Ratio (HR)	HR	95% CI
Control vs. all onv arms	0.69	0.25, 1.90
Control vs. onv 20mg + SoC	0.89	0.28, 2.77
Control vs. onv 30mg + SoC	0.52	0.15, 1.83

Median follow up is ~6 months

It's also important to note that as seen above PFS is not statistically different between groups which is necessary to determine efficacy, and the data is very similar at 15 months.

In conclusion, I think the new data confirms the stance before that the drug is doomed to fail and if the company continues with this data to do a phase 3 for this it is likely to fail. The stock currently is trading at ~2x book value which I think will dilute the stock to raise money for phase 3 and their cash will deteriorate.



July 29, 2024, Stock Price down around ~35% on the day (above)

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