BET Inhibitor Molibresib for the Treatment of Advanced Solid Tumors: Final Results From an Open-label Phase I/II Study

Poster No. 348

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Background and Aims

Molibresib is an orally available, small molecule bromodomain and extra-terminal domain (BET) protein inhibitor investigated for treatment of advanced solid tumors.

Molibresib has been shown in preclinical studies to inhibit the proliferation of patient-derived nuclear protein in testes (NUT) carcinoma (NC),[1] small cell lung cancer (SCLC),[2] castration-resistant prostate cancer (CRPC),[3] triple negative breast cancer (TNBC),[2,4] estrogen receptor–positive breast cancer (ER+BC),[4,5] and gastrointestinal stromal tumor (GIST) cell lines[6] (in particular MYC-amplified solid tumors).

Results from Part 1 of the first-time-in-human (FTIH) study of molibresib in various tumor types showed that molibresib could be tolerated up to 100 mg once daily.[7]

- The 80-mg once-daily dose was selected as the recommended Phase II dose (RP2D) as it showed greater tolerability than the 100-mg once-daily dose.[7]
- Among 19 patients with NUT carcinoma, molibresib treatment resulted in 2 patients achieving a confirmed partial response (PR).

Part 2 of the FTIH study investigated the safety, efficacy, pharmacokinetics, and pharmacodynamics of molibresib at the RP2D for the treatment of NC and other solid tumors.

Methods

Study design

This was an open-label, single- and repeat-dose, two-part, Phase I/II study (115521; NCT01587703) conducted in 22 centers across 8 countries.

Part 1 (Phase I): patients with solid tumors received oral molibresib (2–100 mg once daily; 20–40 mg twice daily) as an amorphous free-base formulation, or 30–80 mg of an oral besylate formulation in a bioequivalence substudy (**Figure 1**).

Part 2 (Phase II): patients were treated with the RP2D from Part 1: 75 mg of the besylate salt formulation, providing similar exposure as 80 mg of the amorphous free-base formulation.

Key inclusion criteria

- ≥16 years of age; diagnosed with NC, SCLC, CRPC, TNBC, ER+BC, or GIST
- Measurable disease as per Response Evaluation Criteria In Solid Tumors (RECIST) v1.1 criteria
- Prior treatment-related toxicities Grade ≤1 (Common Terminology Criteria for Adverse Events v4.0)
- Eastern Cooperative Oncology Group (ECOG) performance status 0–2 for patients with NC; 0–1 for all others

Key exclusion criteria

- Primary malignancy of the central nervous system
- Malignancies related to solid organ transplant
- History of HIV, known hepatitis B surface antigen, or positive hepatitis C antibody
- Recent use of investigational drugs, cancer-related therapy within 14 days, or major surgery within 28 days of the first dose of study medication
- Current anticoagulant therapy; blood or cardiac abnormalities

Primary endpoints

Safety: adverse events (AEs), serious AEs (SAEs), dose reduction or delays (timing and duration), withdrawals due to toxicities, and changes in various safety assessments.

Overall response rate (RR).

Secondary endpoints

Progression-free survival (PFS) and overall survival (OS).

Pharmacokinetic evaluation in patients with different solid tumors.

Gene set expression analysis for a subset of patients with metastatic CRPC and ER+BC.

Statistical analysis

Descriptive statistics were provided for demographic and safety data.

RR was defined as the percentage of patients who achieved a complete response (CR) or PR among patients who received ≥1 dose of treatment.

- NC and other solid tumors: RECIST v1.1; CRPC: prostate-specific antigen 50% decrease (PSA50) using Prostate Cancer Working Group 2 guidelines; GIST: disease control rate (CR + PR + stable disease [SD] ≥16 weeks).

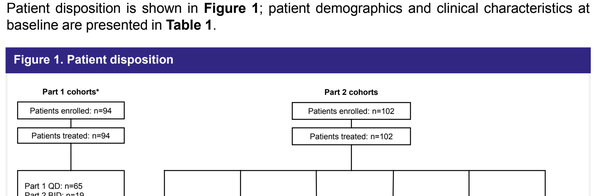
PFS and OS data were summarized descriptively by dose and disease cohort using Kaplan–Meier medians and quartiles.

Results

Patients

Patient disposition is shown in **Figure 1**; patient demographics and clinical characteristics at baseline are presented in **Table 1**.

Figure 1. Patient disposition



*In Part 1, 1 patient participated twice in two different dosing regimens (93 patients received ≥1 dose of molibresib).
BID, twice daily; QD, once daily

Table 1. Patient demographics and clinical characteristics

	Part 2 cohorts							
	NC (n=12)	SCLC (n=14)	CRPC (n=23)	TNBC (n=19)	ER+BC (n=21)	GIST (n=13)	Total Part 2 population (n=102)	Total study population (N=196)
Mean age, years (standard deviation)	42.9 (18.1)	58.3 (11.0)	63.8 (6.1)	50.8 (8.7)	59.7 (10.3)	61.0 (13.2)	57.0 (12.6)	55.4 (14.4)
Female sex, n (%)	7 (58)	9 (64)	0 (0)	19 (100)	21 (100)	6 (46)	62 (61)	105 (54)
ECOG performance status, n (%)*								
0	1 (8)	5 (36)	7 (30)	3 (16)	7 (33)	3 (23)	26 (25)	61 (31)
1	10 (83)	9 (64)	16 (70)	16 (84)	14 (67)	10 (77)	75 (74)	130 (67)
2	1 (8)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (<1)	4 (2)
Prior cancer-related therapy, n (%)								
Surgery	10 (83)	5 (36)	16 (70)	18 (95)	15 (71)	9 (69)	73 (72)	143 (73)
Radiotherapy	7 (58)	10 (71)	21 (91)	18 (95)	16 (76)	1 (8)	73 (72)	129 (66)
Systemic therapy	9 (75)	14 (100)	23 (100)	19 (100)	21 (100)	13 (100)	99 (97)	189 (96)
Prior systemic therapy lines, n (%)								
0	3 (25)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	3 (3)	7 (4)
1	4 (33)	2 (14)	0 (0)	0 (0)	0 (0)	0 (0)	6 (6)	17 (9)
2	2 (17)	6 (43)	0 (0)	0 (0)	0 (0)	3 (23)	11 (11)	20 (10)
≥3	3 (25)	6 (43)	23 (100)	19 (100)	21 (100)	10 (77)	82 (80)	152 (78)

*ECOG performance status was not available for a single patient in the Part 1 population.

Safety

Overall, the safety profile of the Total Part 2 population was similar to the Total study population (including Part 1 and Part 2; **Tables 2** and **3**).

Individual AE data for the Total Part 2 population (n=102) showed that:

- Overall, AEs were reported in 101 (99%) patients; 85 (83%) patients had AEs leading to dose interruption, 22 (22%) patients had AEs leading to discontinuation.
- The most frequent treatment-related AEs were thrombocytopenia (64%), nausea (43%), decreased appetite (37%), diarrhea (32%), dysgeusia (32%), and anemia (31%).
- The most frequent treatment-related SAEs were thrombocytopenia (22%), anemia (6%), vomiting (5%), nausea (4%), and decrease of factor VII level (3%).
- In total, 79 (77%) patients died; the time to death from last dose was >28 days for 63 patients (62%).

Table 2. Safety overview

	Part 2 cohorts							
	NC (n=12)	SCLC (n=14)	CRPC (n=23)	TNBC (n=19)	ER+BC (n=21)	GIST (n=13)	Total Part 2 population (n=102)	Total study population (N=196)
Any AE, n (%)	11 (92)	14 (100)	23 (100)	19 (100)	21 (100)	13 (100)	101 (99)	193 (98)
Treatment-related AEs, n (%)	10 (83)	13 (93)	23 (100)	19 (100)	21 (100)	12 (92)	98 (96)	180 (92)
AEs leading to discontinuation, n (%)	1 (8)	3 (21)	6 (26)	4 (21)	6 (29)	2 (15)	22 (22)	38 (19)
AEs leading to dose reduction, n (%)	5 (42)	4 (29)	9 (39)	5 (26)	4 (19)	3 (23)	30 (29)	53 (27)
AEs leading to dose interruption, n (%)	8 (67)	12 (86)	22 (96)	17 (89)	18 (86)	8 (62)	85 (83)	140 (71)
Grade 1/2 AEs, n (%)	1 (8)	2 (24)	1 (14)	2 (11)	1 (10)	5 (38)	13 (13)	45 (23)
Grade 3/4 AEs, n (%)	10 (83)	11 (79)	22 (96)	15 (79)	18 (86)	8 (62)	84 (82)	142 (72)
Any SAE, n (%)	6 (50)	9 (64)	16 (70)	11 (58)	15 (71)	8 (62)	65 (64)	106 (54)
Treatment-related SAEs, n (%)	5 (42)	6 (43)	13 (57)	8 (42)	8 (38)	5 (38)	45 (44)	77 (39)
Fatal SAEs, n (%)	0 (0)	1 (7)	0 (0)	2 (11)	1 (5)	0 (0)	4 (4)	6 (3)
Fatal treatment-related SAEs, n (%)	0 (0)	0 (0)	0 (0)	1 (5)	0 (0)	0 (0)	1 (1)	1(<1)

Laboratory parameters

Laboratory investigations of the Total Part 2 population showed the following:

- Three of 100 (3%) patients had alanine aminotransferase ≥3 x upper limit of normal (ULN) and 6/100 (6%) patients had bilirubin levels ≥3 x ULN; hepatocellular injury was detected in a single patient.
- Grade 2 changes in serum creatinine were observed from Week 2; 1 patient experienced a Grade 3 value at Week 9.
- Grade 3 reductions in platelets were observed from Week 2 (1%–33% during Weeks 2–45); Grade 4 reductions were observed from Week 3 (2%–6% during Weeks 3–17).
- Twelve (12%) patients had any grade increase of the corrected QT interval by Fredericia at worse case post baseline; these changes were considered within the expected variation.
- For worst-case left ventricular ejection fraction absolute change from baseline, 44/86 (51%) patients had an absolute decrease of >0% to <10%, 14/86 (13%) patients had an absolute decrease of 10% to 19%, and 1/86 (1%) had a decrease of ≥20%.

Efficacy

In the Total study population, 4/196 patients achieved a confirmed PR; 3 patients with NC (2 patients from Part 1 and 1 from Part 2) and 1 patient with CRPC (Part 2; **Table 4**).

- None of the 23 patients with CRPC (Part 2) had a PSA50 response.

Table 3. Summary of AEs occurring in ≥15% of the Total study population by toxicity grade

	Total Part 2 population (n=102)			Total study population (N=196)		
	Grade 1/2 AEs	Grade 3/4 AEs	Total AEs	Grade 1/2 AEs	Grade 3/4 AEs	Total AEs
Any event, n (%)	13 (13)	84 (82)	101 (>99)	45 (23)	142 (72)	193 (98)
Thrombocytopenia	24 (24)	44 (43)	68 (67)	44 (22)	81 (41)	125 (64)
Nausea	42 (41)	8 (8)	50 (49)	84 (43)	12 (6)	96 (49)
Decreased appetite	46 (45)	2 (2)	48 (47)	83 (42)	8 (4)	91 (46)
Anemia	25 (25)	21 (21)	46 (45)	43 (22)	39 (20)	82 (42)
Diarrhea	39 (38)	1 (<1)	40 (39)	69 (35)	4 (2)	73 (37)
Fatigue	31 (30)	8 (8)	39 (38)	60 (31)	10 (5)	70 (36)
Vomiting	30 (29)	7 (7)	37 (36)	61 (31)	9 (5)	70 (36)
Asthenia	24 (24)	14 (14)	38 (37)	40 (20)	22 (11)	62 (32)
Dysgeusia	33 (32)	0 (0)	33 (32)	62 (32)	0 (0)	62 (32)
Hyperbilirubinemia	25 (25)	6 (6)	31 (30)	42 (21)	17 (9)	59 (30)
Dyspnea	21 (21)	8 (8)	29 (28)	38 (19)	8 (4)	46 (23)
Hyperglycemia	17 (17)	2 (2)	19 (19)	31 (16)	6 (3)	37 (19)
Constipation	17 (17)	0 (0)	17 (17)	35 (18)	0 (0)	35 (18)
International normalized ratio increased	19 (19)	0 (0)	19 (19)	35 (18)	0 (0)	35 (18)
Aspartate aminotransferase increased	17 (17)	5 (5)	22 (22)	28 (14)	6 (3)	34 (17)
Prothrombin time prolonged	17 (17)	1 (<1)	18 (18)	29 (15)	1 (<1)	30 (15)

Pharmacokinetics/pharmacodynamics

In Part 2, the 0.5–2.0-h post-dose total active moiety median plasma concentration at Week 1 was 2960 nM (min: 64.5 nM, max: 8990 nM), and it was 2622 nM at Week 4 (min: 110 nM, max: 6234).

- Both Week 1 (0.5–2.0 h post dose) and Week 4 (pre-dose and 0.5–2.0 h post dose) median plasma concentrations for molibresib and total active moiety were similar across individual tumor cohorts.

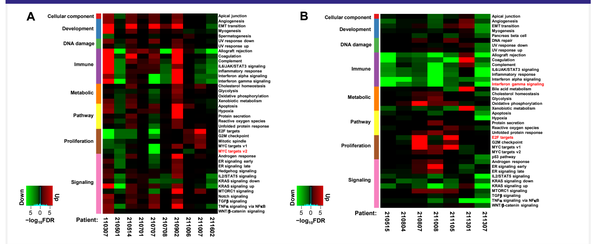
Gene set expression analysis from pre- and post-dose biopsy samples from patients with CRPC and ER+BC (Part 2) showed transcriptional downregulation of N-myc, and interferon gamma and E2F target genes, respectively, upon treatment with molibresib (**Figure 2**).

Table 4. Efficacy overview

	Part 2 cohorts							
	NC (n=12)	SCLC (n=14)	CRPC (n=23)	TNBC (n=19)	ER+BC (n=21)	GIST (n=13)	Total Part 2 cohort (n=102)	Total study population (N=196)
RR, % (95% CI)	8 (0.2, 38.5)	0 (0.0, 23.2)	4 (0.1, 21.9)	0 (0.0, 17.6)	0 (0.0, 16.1)	0 (0.0, 24.7)	2 (0.2, 6.9)	2 (0.6, 5.1)
Best response, n (%)								
CR	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
PR	1 (8)	0 (0)	1 (4)	0 (0)	0 (0)	0 (0)	2 (2)	4 (2)
SD	6 (50)	3 (21)	5 (22)	5 (26)	6 (29)	5 (38)	30 (29)	67 (34)
PD	1 (8)	9 (64)	11 (48)	10 (53)	11 (52)	7 (54)	49 (48)	81 (41)
Not evaluable	4 (33)	2 (14)	5 (22)	4 (21)	4 (19)	1 (8)	20 (20)	39 (20)
Non-CR/Non-PD*	0 (0)	0 (0)	1 (4)	0 (0)	0 (0)	0 (0)	1 (<1)	5 (3)
Median PFS, months (95% CI)	4.8 (2.5, 5.1)	2.2 (1.1, 5.3)	8.0 (5.5, 11.7)	2.4 (1.4, 6.5)	4.7 (3.3, 9.2)	3.4 (1.9, 7.3)	4.7 (3.6, 5.6)	5.0 (3.9, 5.9)
Median OS, months (95% CI)	5.0 (4.3, NR)	2.6 (1.1, 9.4)	9.1 (6.7, 11.7)	5.0 (2.3, 10.3)	8.8 (3.6, 13.1)	7.3 (3.4, NR)	6.5 (5.1, 9.2)	6.7 (5.6, 8.6)

*Persistence of one or more nontarget lesion(s) and/or maintenance of tumor marker level above the normal limits as defined in RECIST v1.1.[8]
CI, confidence interval; NR, not reached; PD, progressive disease

Figure 2. Gene set enrichment analysis across (A) CRPC and (B) ER+BC patients



Heat maps represent FDR for gene sets. Green indicates enrichment of downregulated genes, and red indicates enrichment of upregulated genes.
The vertical bar shows the broad biological process associated with each gene set.
FDR, false discovery rate

Conclusions

Molibresib 75 mg was found to be generally tolerable; however, there were a number of safety issues requiring dose reductions and dose interruptions.

In total, 4 patients achieved a confirmed PR following molibresib treatment; the predefined clinically meaningful response criteria were not met for any tumor type.

Molibresib exposure following administration of 75 mg was similar across individual tumor cohorts (Part 2) and to the findings reported in Part 1 of the study.

Gene set expression analysis suggested that molibresib had cytostatic and anti-inflammatory effects dependent on tumor type.

Disclosures

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