Long-Term Outcomes and Molecular Correlates of Sotorasib Efficacy in Patients With Pretreated KRAS G12C-Mutated Non–Small-Cell Lung Cancer: 2-Year Analysis of CodeBreaK 100

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INTRODUCTION

Overall survival (OS) remains poor for molecularly unselected, advanced non–small-cell lung cancer (NSCLC),1,2 with 2-5 months of median progression-free survival (PFS) on second-line plus chemotherapy or immunotherapy.3-6 Sotorasib specifically and irreversibly inhibits KRAS\textsuperscript{G12C},7-10 with approval in over 40 countries11-13 for adults with KRAS G12C-mutated advanced NSCLC after prior systemic therapy.14,15 In CodeBreaK 100 phase II, sotorasib demonstrated an objective response rate (ORR) of 37%, a median duration of response (DOR) of 11.1 months, a median PFS of 6.8 months, a median OS of 12.5 months, and a manageable safety profile in KRAS G12C-mutated advanced NSCLC.16 We report CodeBreaK 100 phase II 2-year pooled analyses representing, to our knowledge, the longest KRAS\textsuperscript{G12C} inhibitor treatment follow-up to date.

METHODS

Patients

The multicenter, single-group, open-label phase I/II CodeBreaK 100 trial (ClinicalTrials.gov identifier: NCT03600883) enrolled patients age 18 years and older with KRAS G12C-mutated locally advanced or metastatic NSCLC after progression on prior therapies (Data Supplement, online only).16 Institutional review board approval before study initiation and participating country regulatory authority approval were received; all patients provided written informed consent.

Study Design

Phase I primary end point was safety and tolerability (key secondary: DOR and PFS). Phase II primary end point was ORR (blinded independent central review; key secondary: DOR, PFS, OS, and safety). Late-onset toxicities were assessed (treatment-related adverse events [TRAEs] occurring after 1 year on treatment).
CONTEXT

Key Objective
To determine the long-term safety, tolerability, and efficacy of sotorasib 960 mg once daily in patients with KRAS G12C-mutated, locally advanced or metastatic non–small-cell lung cancer from the CodeBreaK 100 clinical trial (ClinicalTrials.gov identifier: NCT03600883). Exploratory analyses assessed the relationship of various biomarkers, such as PD-L1 expression level and genomic alterations, with efficacy.

Knowledge Generated
This 2-year pooled analysis of CodeBreaK 100, which is, to our knowledge, the largest clinical data set with the longest follow-up reported for patients treated with any KRASG12C inhibitor to date, showed that sotorasib treatment provided long-term efficacy and was well tolerated, with no new safety signals detected. Long-term benefit with sotorasib (defined as progression-free survival of at least 12 months) was associated with lower baseline circulating tumor DNA levels and was observed across KRAS G12C variant allele frequency levels, PD-L1 expression levels, and in a proportion of patients with STK11 and/or KEAP1 comutations.

Relevance
The findings from this analysis with over 2-year follow-up data demonstrate that nearly a quarter of previously treated advanced stage KRAS G12C-mutated NSCLC patients treated with sotorasib derived long-term benefit, with few late-onset treatment-related toxicities, supporting not only its use in this treatment setting but also additional studies investigating its therapeutic role in earlier lines of therapy.

Exploratory analyses evaluated molecular correlates with efficacy (Data Supplement). PD-L1 and genomic alterations were correlated with long-term benefit (PFS ≥ 12 months) versus early progression (nonresponders with PFS ≤ 3 months).

RESULTS

Patients
As of February 22, 2022, 174 patients (phase I, N = 48; phase II, N = 126) received sotorasib 960 mg once daily (Table 1). Median treatment duration was 5.6 months (range, 0.2-35.9); 13 patients remained on treatment at cutoff. Median prior lines of therapy was 2.0 (range, 0 to 4+). Prior therapies included anti–PD-(L)1 (157 [90%]) and platinum-based chemotherapy plus anti–PD-(L)1 (144 [83%]).

Safety
Any-grade TRAEs were observed in 121 (70%) patients (Data Supplement), with grade 3 in 34 (20%), grade 4 in 2 (1%), and no fatal TRAEs; TRAEs led to treatment reduction or interruption in 39 (22%) and treatment discontinuation in 11 (6%). Most common TRAEs were diarrhea (53 [30%]), increased alanine aminotransferase level (31 [18%]), and increased aspartate aminotransferase level (31 [18%]). Median (range) time to grade ≥ 3 diarrhea and hepatotoxicity onset was 6.1 (1.7-11.1) and 9.1 (3.1-18.7) weeks. All grade ≥ 3 (median [range] duration, weeks) diarrhea resolved (2.9 [0.3-6.0]); grade ≥ 3 hepatotoxicity resolved in all but three of 19 (16%; 5.5 [0.4-39.1]). Trends toward increased hepatotoxicity in patients receiving checkpoint inhibitors ≤ versus >3 months before sotorasib initiation were observed (Data Supplement).

Of 45 patients who continued sotorasib beyond 1 year, 11 (24%) had any-grade TRAEs after 1 year on treatment (new-onset TRAEs), without trends in adverse event type. One grade 3 new-onset TRAE (2%; hemolytic anemia) resolved in 5 days (sotorasib discontinued after disease progression). No grade 4 or 5 new-onset TRAEs occurred. New-onset TRAEs led to dose reduction in one (2%) patient without treatment discontinuation.

Efficacy
ORR was 41% (95% CI, 33.3 to 48.4), and DCR was 84% (95% CI, 77.3 to 88.9; Data Supplement). Of patients with confirmed response, estimated 72.8% (95% CI, 60.0 to 82.2) and 50.6% (37.4 to 62.4) remained in response at 6 and 12 months, respectively. Median DOR was 12.3 months (95% CI, 7.1 to 15.0).

Median PFS was 6.3 months (95% CI, 5.3 to 8.2; Fig 1A); median OS was 12.5 months (10.0 to 17.8; Fig 1B). Kaplan-Meier OS estimate was 51% (95% CI, 42.8 to 58.2) and 33% (95% CI, 25.0 to 40.2) at 12 and 24 months, respectively. At data cutoff, nine of 70 (13%) patients with response remained on study without progression, including five receiving sotorasib for ≥2 years with continued response (Fig 1C).

Sixteen patients had evaluable brain metastases per central Response Assessment in exploratory Neuro-Oncology Brain Metastases review (Data Supplement); three (19%) had complete response and 11 (69%) had stable disease, with 14 (88%) having intracranial disease control overall (Data Supplement).

Across 172 efficacy-evaluable patients, 40 (23%) had long-term clinical benefit (PFS ≥ 12 months) and 62 (36%) had
### TABLE 1. Patient Demographics and Baseline Characteristics

<table>
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<th>Characteristic</th>
<th>Phase I (N = 48)</th>
<th>Phase II (N = 126)</th>
<th>Total (N = 174)</th>
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<td>65.0 (37-86)</td>
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<tr>
<td>&lt;1%</td>
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<td>Duration of sotorasib treatment, months, median (range)</td>
<td>6.0 (0.2-35.9)</td>
<td>5.5 (0.2-26.9)</td>
<td>5.6 (0.2-35.9)</td>
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</table>

Abbreviations: ECOG PS, Eastern Cooperative Oncology Group performance status; VEGF, vascular endothelial growth factor.

aDetermined locally.

bEach patient may have had more than one prior therapy.

cTwo patients with no prior line of therapy in phase I were excluded.
FIG 1. Long-term benefit and outcomes with sotorasib treatment. Kaplan-Meier plot of (A) PFS and (B) OS (median OS follow-up of 24.9 months [range, 0.7-35.9 months]) by central review, and (C) swimmer plot for phase I and phase II responders by central review (one patient with an ongoing response ended treatment because of patient request). BOR, best objective response; CR, complete response; OS, overall survival; PD, progressive disease; PFS, progression-free survival; PR, partial response.
early progression (PFS ≤ 3 months). Baseline characteristics were similar; the latter had slightly higher proportions of patients with visceral metastasis (liver/bone), progressive disease on prior therapy, and received prior platinum-based chemotherapy and immunotherapy.

Biomarker Analysis

Centrally measured PD-L1 and/or genomic data were available for 114 phase II patients (Data Supplement). Most prevalent alterations were TP53 (46%), LRP1B (36%), KDM6A (32%), and STK11 (32%).

Albeit limited in sample size, long-term clinical benefit with sotorasib was observed across PD-L1 expression levels (Fig 2A). There was a nonsignificant trend toward enrichment of longer benefit with PD-L1 tumor proportion score (TPS) <1% versus ≥1% (odds ratio [OR], 0.36 [0.12 to 1.12]), without significant differences between PD-L1 TPS 1%-49% and ≥50% (OR, 0.83 [0.07 to 9.69]). Most significant enrichment in patients with early progression was with mutant KEAP1 (OR, 0.22 [0.06 to 0.87] long-term benefit vs early progression); although not significant, these patients were more likely to have ROS1 (single-nucleotide

**FIG 2.** Association of long-term benefit and early progression with PD-L1 expression and genomic alterations: (A) PD-L1 tumor proportion score, (B) selected genomic alterations, (C) STK11 and KEAP1 mutations, and (D) Mutant versus wild-type STK11. CR, complete response; IHC, immunohistochemistry; PFS, progression-free survival; PR, partial response.
variant (SNVI) and secondary RAS mutations (Fig 2B; Data Supplement). Patients with long-term benefit were more likely to harbor mutations in PI3K, PDGFR, and EPH receptor gene family and RET SNVs. Association of KEAP1 wild-type status with long-term benefit was independent of STK11 mutation status (Fig 2C). Patients with STK11 comutations were as likely to derive long-term benefit as patients with STK11 wild-type (OR, 0.71 [0.25 to 2.02]; Fig 2D).

No difference in tumor tissue median KRAS G12C variant allele frequency (VAF) or tumor mutational burden was observed in long-term benefit or early progression groups (Data Supplement). Patients with long-term benefit tended to have lower baseline median plasma circulating tumor DNA (ctDNA; $P = .01$; Data Supplement).

**DISCUSSION**

In this analysis representing the most mature KRASG12C inhibitor clinical data, sotorasib demonstrated long-term efficacy, without new safety signals. A substantial proportion of patients derived long-term clinical benefit (1- and 2-year OS rates, 51% and 33%, respectively). Once-daily oral sotorasib 960 mg did not result in cumulative late-onset severe or chronic lower-grade toxicities.

Durable sotorasib benefit and safety profiles compare favorably with standard-of-care chemotherapy with docetaxel-based regimens, which historically yielded approximately 10%-23% response rates and a median PFS < 4.5 months. Two-year OS rate (33%) with sotorasib was higher versus docetaxel (historically 14%). In the phase III CodeBreaK 200 randomized controlled trial (Clinical-Trials.gov identifier: NCT04303780), sotorasib showed statistically significant improvement in PFS versus docetaxel in pretreated KRAS G12C-mutated advanced NSCLC, with a 34% decrease in the relative risk of disease progression or death with sotorasib (HR 0.66; $P = .0017$). There was no OS difference, although the study was not powered for OS, and docetaxel arm crossover was permitted, improved quality of life with sotorasib was observed. These findings are encouraging, considering historically poor standard-of-care chemotherapy outcomes.

Long-term benefit with sotorasib was associated with lower baseline ctDNA levels, consistent with ctDNA prognostic roles across therapeutics. Prolonged benefit was observed across KRAS G12C VAF levels, PD-L1 expression, and a proportion of patients with STK11 and/or KEAP1 comutations. However, consistent with studies of other therapies, KEAP1 mutation was negatively prognostic overall. Relatively small sample sizes with available biomarker data were challenging; additional analyses evaluating prognostic and predictive impact of baseline and postprogression genomic alterations are warranted. International collaboration and data sharing are key to uncovering KRAS-mutant cancer molecular complexities.

In this long-term analysis, oral once-daily sotorasib demonstrated favorable safety profile and durable efficacy across subgroups in KRAS G12C-mutated NSCLC.
REFERENCES


AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

Long-Term Outcomes and Molecular Correlates of Sotorasib Efficacy in Patients With Pretreated KRAS G12C-Mutated Non–Small-Cell Lung Cancer: 2-Year Analysis of CodeBreak 100

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Research Funding: Genentech (Inst), Amgen (Inst), Daiichi Sankyo (Inst), Adaptimmune (Inst), Abbvie (Inst), Bayer (Inst), Infinity Pharmaceuticals (Inst), Kite, a Gilead Company (Inst), Medimmune (Inst), National Cancer Institute (Inst), Fate Therapeutics (Inst), Pfizer (Inst), Novartis (Inst), Numab (Inst), Turning Point Therapeutics (Inst), Kyowa (Inst), Loxo (Inst), Merck (Inst), Eisai (Inst), Genmab (Inst), Mirati Therapeutics (Inst), Molagon (Inst), Takeda (Inst), AstraZeneca (Inst), Navne (Inst), VM Pharma (Inst), Erasca, Inc (Inst), Bristol Myers Squibb (Inst), Adial Nortye (Inst), Seagen (Inst), Deciphera (Inst), Pyramid Biosciences (Inst), Lilly (Inst), Endeavor BioMedicines (Inst), F. Hoffmann-La Roche (Inst), Ignity (Inst), Teckro (Inst), TCR2 Therapeutics (Inst)  
Travel, Accommodations, Expenses: Genmab, Society for Immunotherapy of Cancer, Bayer Schering Pharma, ASCO, AACR, Telperian

Piro Lito  
Leadership: Frontier Medicines  
Consulting or Advisory Role: Black Diamond Therapeutics, Repare Therapeutics, AmMax Bio, Revolution Medicines  
Speakers' Bureau: Boehringer Ingelheim  
Research Funding: Mirati Therapeutics (Inst), Revolution Medicines (Inst), Amgen (Inst), Boehringer Ingelheim (Inst), VircPharmaceuticals (Inst)  
Patents, Royalties, Other Intellectual Property: I am listed as an inventor on a patent application filed by MSKCC that describes an approach to treat BRAF mutant cancers (Inst), I am listed as an inventor on a patent application filed by MSKCC that describes an approach to treat KRAS mutant cancers (Inst)

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Employment: Amgen  
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Simon Jones  
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Patents, Royalties, Other Intellectual Property: I am listed as an inventor on several Amgen patents. I do not receive royalties on these patents  
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Travel, Accommodations, Expenses: MORE Health, Jiangsu Hengrui Medicine  
Uncompensated Relationships: Amgen, AstraZeneca, Genentech, Lilly, Boehringer Ingelheim, Daiichi Sanyko  
No other potential conflicts of interest were reported.