Antitumor activity of amivantamab by consensus molecular subtypes in *RAS/BRAF* wild-type metastatic colorectal cancer: Secondary analyses from the phase 1b/2 OrigAMI-1 study

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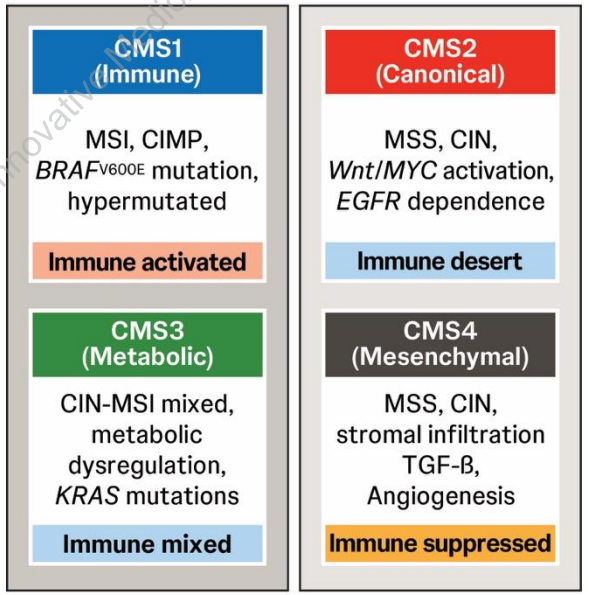
BACKGROUND

- Colorectal tumors can be classified into 4 CMS (**Figure 1**)[1,2]
- In *RAS/BRAF* WT mCRC, the majority of tumors are classified as canonical *EGFR*-dependent (CMS2) and mesenchymal *EGFR*-independent (CMS4), with the latter associated with poor prognosis and limited response to traditional EGFR inhibition[2,3]
  - In prior analyses, cetuximab monotherapy demonstrated greater antitumor activity against CMS2 versus CMS4 tumors[4]
  - Additionally, CMS2 to CMS4 subtype switching has been described as a potential cetuximab monotherapy resistance mechanism[3]
- Amivantamab, an EGFR-MET bispecific antibody, has demonstrated antitumor activity in refractory mCRC, independent of sidedness[5]
- **Given the role of *HGF/MET* signaling in the mesenchymal subtype (CMS4),[3] targeting with amivantamab could demonstrate antitumor activity in both CMS2 and CMS4 mCRC**

Figure 1: CMS in colorectal cancer[1]



CIMP, CpG Island Methylator Phenotype; CIN, chromosomal instability; CMS, consensus molecular subtype; EGFR, epidermal growth factor receptor; mCRC, metastatic colorectal cancer; MSI, microsatellite instability; MSS, mixed MSI status; WT, wild-type.

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METHODS

- OrigAMI-1 (ClinicalTrials.gov Identifier: NCT05379595) is assessing amivantamab as monotherapy or combined with FOLFOX or FOLFIRI in *RAS/BRAF* WT mCRC (**Figure 2**)
- For Cohorts A, B, and C (third- or later-line population):
  - Exploratory analyses involving CMS groups were conducted
  - CMS assignment and expression changes in key EGFR/MET ligands at baseline (n=76) and C3D1 (n=17) were analyzed by whole-transcriptome RNA-sequencing of biopsies
- Data cutoff was October 31, 2024; primary results from the monotherapy cohorts were published in 2026 in *J Clin Oncol*[5]

C, Cycle; CMS, consensus molecular subtype; D, Day; EGFR, epidermal growth factor receptor; mCRC, metastatic colorectal cancer; WT, wild-type.

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METHODS

Figure 2: OrigAMI-1 study design

OrigAMI-1 eligibility criteria

- Unresectable mCRC
- WT *KRAS*, *NRAS*, *BRAF*, *EGFR* ectodomain, without *ERBB2*/*HER2* amplification by central testing

For monotherapy cohorts:

- Amivantamab 1050 mg IV (1400 mg if body weight ≥80 kg) weekly for the first 4 weeks, then every 2 weeks

Focus of this poster

Cohort A: Amivantamab monotherapy in L-sided
2–3 prior lines in metastatic setting (no prior anti-EGFR therapy)

Cohort B: Amivantamab monotherapy in L-sided
2–3 prior lines in metastatic setting (with prior anti-EGFR therapy)

Cohort C: Amivantamab monotherapy in R-sided
2–3 prior lines in metastatic setting

Enrolling

Cohort D: Amivantamab plus FOLFOX in L- and R-sided
Max 1 prior line in metastatic setting (no prior anti-EGFR therapy)

Cohort E: Amivantamab plus FOLFIRI in L- and R-sided
Max 1 prior line in metastatic setting (no prior anti-EGFR therapy)

Cohort F: 1L subcutaneous amivantamab plus FOLFOX in R-sided

1L, first line; EGFR, epidermal growth factor receptor; IV, intravenous; L-sided, left-sided; mCRC, metastatic colorectal cancer; R-sided, right-sided; WT, wild-type.

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RESULTS

- Among biopsied samples at baseline, CMS2 (n=42) and CMS4 (n=31) comprised 96% (73/76) of tumors
- Baseline characteristics were generally similar between CMS2 and CMS4, except for a higher percentage of liver metastases in CMS2 (**Table 1**)

Table 1: Demographic and baseline disease characteristics

Characteristic, n (%)	CMS2 (n=42)	CMS4 (n=31)
Median (range) age, years	63.5 (40–80)	62.0 (35–78)
Male	30 (71)	19 (61)
Race		
Asian	29 (69)	18 (58)
White	11 (26)	11 (35)
Black or African American	1 (2)	1 (3)
Other[a]	1 (2)	1 (3)
ECOG PS score: 0 / 1	22 (52) / 20 (48)	14 (45) / 17 (55)
Tumor side: left / right	34 (81) / 8 (19)	22 (71) / 9 (29)
No. of prior lines[b]: 2 / 3	24 (57) / 18 (43)	18 (58) / 13 (42)
Prior bevacizumab	40 (95)	29 (94)
Prior anti-EGFR therapy[c]	25 (60)	22 (71)
Liver metastases	37 (88)	21 (68)

[a]Includes American Indian or Alaska Native and not reported. [b]In the metastatic setting. [c]Includes cetuximab and panitumumab.
CMS, consensus molecular subtype; ECOG PS, Eastern Cooperative Oncology Group performance status; EGFR, epidermal growth factor receptor.

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RESULTS: Efficacy

- Clinical outcomes with amivantamab for CMS2 and CMS4 were consistent (**Table 2**)

Table 2: Efficacy outcomes in refractory *RAS/RAF* WT mCRC

Endpoint	CMS2 (n=42)	CMS4 (n=31)
Objective response rate	26% (95% CI, 14–42)	16% (95% CI, 5–34)
Disease control rate[a]	83% (95% CI, 69–93)	74% (95% CI, 55–88)
PFS	4.2 months (95% CI, 3.6–5.6)	5.3 months (95% CI, 3.5–5.7)
OS	11.3 months (95% CI, 8.1–16.2)	13.5 months (95% CI, 8.1–NE)

[a]Disease control rate is defined as the percentage of participants achieving a best overall response of CR, PR, or SD (with a minimum duration of 7 weeks).
CI, confidence interval; CMS, consensus molecular subtype; CR, complete response; mCRC, metastatic colorectal cancer; NE, not estimable; OS, overall survival; PFS, progression-free survival; PR, partial response; SD, stable disease; WT, wild-type.

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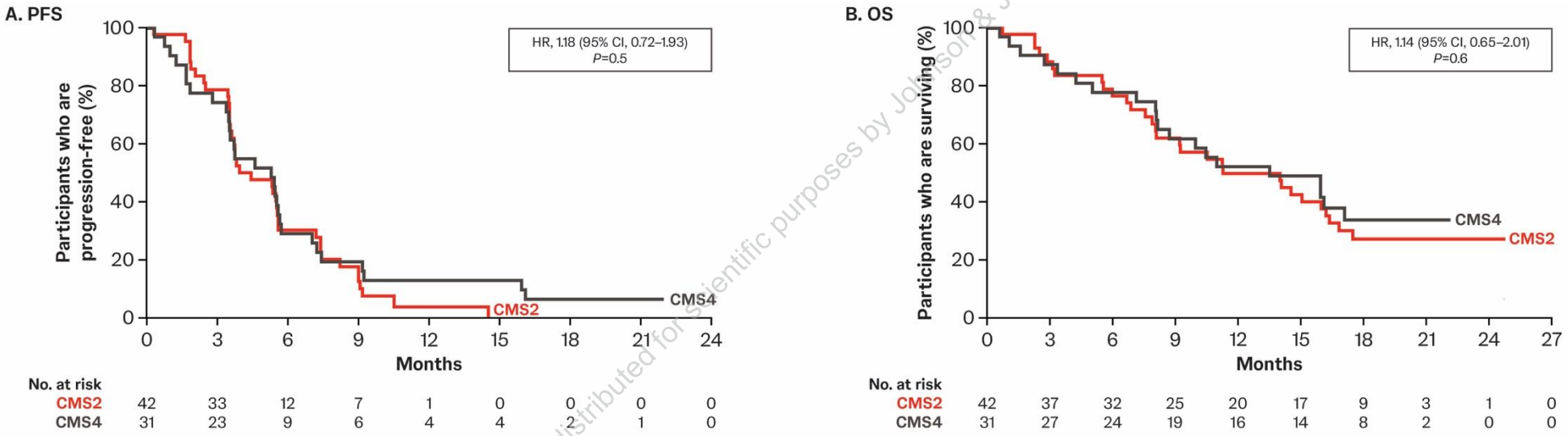
RESULTS: Efficacy

- PFS and OS were not significantly different between the CMS2 and CMS4 subgroups (**Figure 3**; *P*=0.5 for PFS and *P*=0.6 for OS)

Figure 3: (A) PFS and (B) OS among CMS2/CMS4 subgroups following amivantamab treatment in refractory *RAS/RAF* WT mCRC



CI, confidence interval; CMS, consensus molecular subtype; HR, hazard ratio; mCRC, metastatic colorectal cancer; OS, overall survival; PFS, progression-free survival; WT, wild-type.

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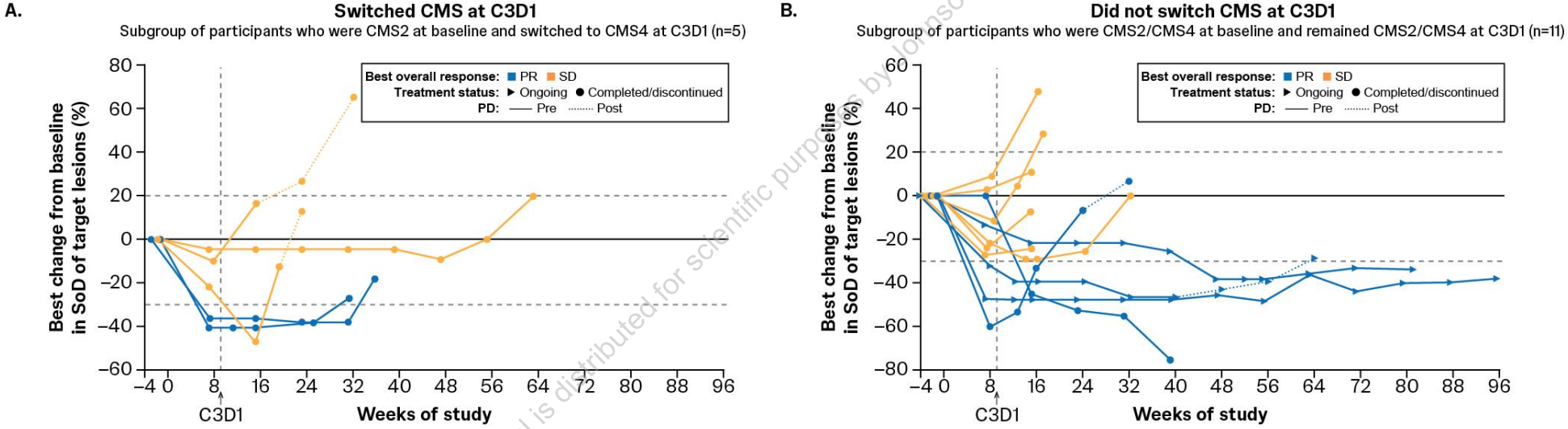
RESULTS: Biomarkers

- At baseline (n=73):
  - Genomic profiles derived from ctDNA were similar
  - *AREG* and *EREG* mRNA expression was higher in CMS2
- Among participants with paired biopsies at baseline and C3D1 (n=17)
  - At C3D1, antitumor activity was seen in participants with subtype switching (2/5 responded) and without subtype switching (5/11 responded; **Figure 4**)

Figure 4: Antitumor activity in participants (A) with or (B) without CMS subtype switching following amivantamab treatment[a]



[a]One participant switched from CMS2 to CMS1 in C3D1 (not shown).
C, Cycle; CMS, consensus molecular subtype; ctDNA, circulating tumor DNA; D, Day; mRNA, messenger RNA; PD, progressive disease; PR, partial response; SD, stable disease; SoD, sum of diameters.

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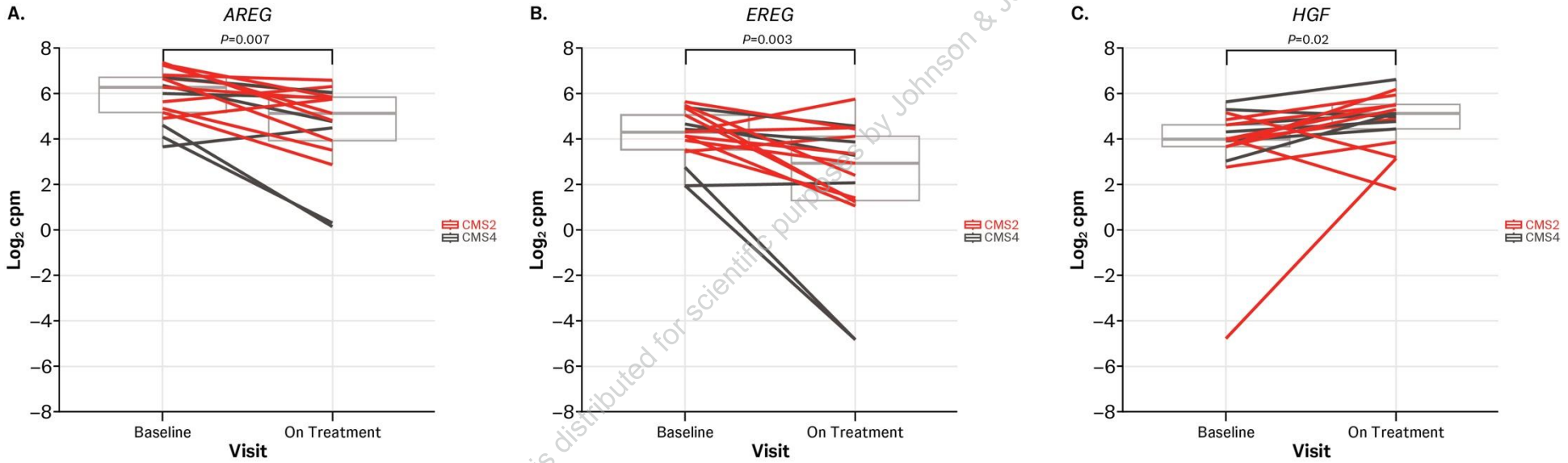
RESULTS: Biomarkers

- Among participants with paired biopsies at baseline and C3D1 (n=17)
  - Amivantamab treatment generally decreased *AREG* and *EREG* expression and increased *HGF* expression, independent of subtype or response (**Figure 5**)

Figure 5: (A) *AREG*, (B) *EREG*, and (C) *HGF* mRNA expression changes following amivantamab treatment



CMS, consensus molecular subtype; mRNA, messenger RNA.

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CONCLUSIONS

- Majority of biopsied *RAS/BRAF* WT mCRC tumors in this refractory setting were CMS2 (canonical *EGFR*) or CMS4 (mesenchymal)
- Unlike traditional EGFR inhibitors, PFS (4.2 vs 5.3 months) and OS (11.3 vs 13.5 months) were similar in CMS2 versus CMS4, respectively, among participants treated with amivantamab in this refractory *RAS*/*BRAF* WT setting
- Antitumor activity was observed with or without subtype switching; amivantamab led to decreases in *AREG* and *EREG* and an increase in *HGF* expression, independent of subtype or response

KEY TAKEAWAY

In a refractory 3L+ population, amivantamab monotherapy, with its dual targeting of EGFR and MET, demonstrated consistent antitumor activity in both canonical *EGFR*-dependent (CMS2) and mesenchymal *EGFR*-independent (CMS4) mCRC tumors

3L+, third- or later-line; CMS, consensus molecular subtype; EGFR, epidermal growth factor receptor; mCRC, metastatic colorectal cancer; OS, overall survival; PFS, progression-free survival; WT, wild-type.

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