Mezigdomide, carfilzomib, and dexamethasone vs carfilzomib and dexamethasone in relapsed/refractory multiple myeloma: results from the phase 3 SUCCESSOR-2 trial

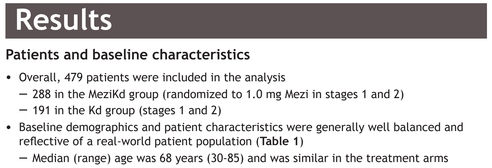
Paul G. Richardson,[1] Fredrik Schjesvold,[2] Chengcheng Fu,[3] Monique A. Hartley-Brown,[1] Cesar Gomez,[4] Donna Reece,[5] Gabor Mikala,[6] Omar Alkharabsheh,[7] Christopher Parrish,[8] Darrell White,[9] Hang Quach,[10] Claudio Cerchione,[11] Vania Hungria,[12] Amitabha Mazumder,[13] Marc S. Raab,[14] Albert Oriol,[15] Zehua Zhou,[16] Alberto Rocci,[17] Brian Yu,[16] Meletios A. Dimopoulos[18]

[1]Dana-Farber Cancer Institute, Boston, MA, USA; [2]Oslo Myeloma Center, Department of Hematology, Oslo University Hospital, Oslo, Norway; [3]The First Affiliated Hospital of Soochow University, Jiangsu Institute of Hematology, Suzhou, China; [4]Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, UK; [5]The Princess Margaret Cancer Centre, Toronto, ON, Canada; [6]Semmelweis University, Budapest, Hungary; [7]USA Health Mitchell Cancer Institute, University of South Alabama, Mobile, AL, USA; [8]Leeds Teaching Hospitals NHS Trust, Leeds, UK; [9]Queen Elizabeth II Health Sciences Centre, Dalhousie University, Halifax, NS, Canada; [10]St Vincent's Hospital Melbourne, University of Melbourne, Melbourne, VIC, Australia; [11]IRCCS Istituto Romagnolo per lo Studio dei Tumori, Meldola, Italy; [12]São Germano Clinic, São Paulo, Brazil; [13]The Oncology Institute of Hope and Innovation, Pasadena, CA, USA; [14]Heidelberg University Hospital, Heidelberg, Germany; [15]Catalan Institute of Oncology and Josep Carreras Institute, Hospital Germans Trias i Pujol, Badalona, Spain; [16]Bristol Myers Squibb, Princeton, NJ, USA; [17]Celgene International Sàrl, a Bristol-Myers Squibb Company, Boudry, Switzerland; [18]National and Kapodistrian University of Athens, Athens, Greece

Introduction

- An increasing number of patients with multiple myeloma (MM) entering second-line treatment are anti-CD38 monoclonal antibody (mAb)- and lenalidomide (LEN)-exposed and/or refractory, with associated poor outcomes and limited treatment options[1-3]
- Carfilzomib plus dexamethasone (Kd) is a widely adopted standard of care in relapsed/refractory MM (RRMM) often used following anti-CD38 mAb and LEN exposure[2]
- Mezigdomide (Mezi) is a potent oral cereblon E3 ligase modulator (CELMoD) that induces maximal, rapid Ikaros/Aiolos degradation leading to enhanced MM-cell death and immune stimulation vs immunomodulatory drug (IMiD) agents[4] (**Figure 1**)
  - Preclinically, Mezi demonstrated synergistic antitumor activity with proteasome inhibitors (PIs)[5]
- In a phase 1/2 trial, Mezi in combination with Kd (MeziKd) yielded promising efficacy with a manageable safety profile in patients with RRMM[6]
- We report results from the SUCCESSOR-2 trial (NCT05552976), evaluating MeziKd vs Kd alone in anti-CD38 mAb- and LEN-exposed patients with RRMM from first relapse. This is the first phase 3 CELMoD trial to report findings

Figure 1. Mechanism of action of the CELMoD Mezi[4]



T/NK, T-cell/natural killer cell; Ub, ubiquitin.

Objective

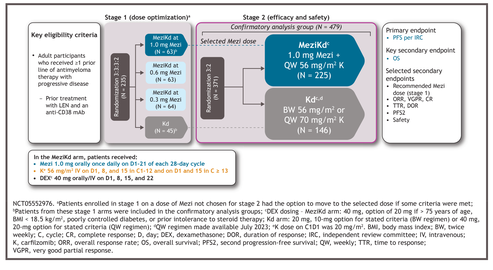
- To assess the efficacy and safety of MeziKd compared with Kd in patients with RRMM who have had prior treatment with an anti-CD38 mAb and LEN

Methods

Study design and treatment

- SUCCESSOR-2 is a randomized phase 3, two-stage, inferentially seamless trial
  - In stage 1, patients were randomized 3:3:3:2 to 1 of 3 Mezi doses (0.3 mg, 0.6 mg, and 1.0 mg) plus Kd or to Kd to identify the optimal Mezi dose for the combination
  - In stage 2, patients were randomized 3:2 to the selected MeziKd dose or Kd to evaluate the efficacy and safety of the selected Mezi dose (1.0 mg)
- The primary endpoint was progression-free survival (PFS)
- Details of the two-stage study design, treatment dosing, and endpoints are shown in **Figure 2**

Figure 2. SUCCESSOR-2 study design



NCT05552976. [a]Patients enrolled in stage 1 on a dose of Mezi not chosen for stage 2 had the option to move to the selected dose if some criteria were met; [b]Patients from these stage 1 arms were included in the confirmatory analysis groups; [c]DEX dosing – MeziKd arm: 40 mg, option of 20 mg if > 75 years of age, BMI < 18.5 kg/m², poorly controlled diabetes, or prior intolerance to steroid therapy; Kd arm: 20 mg, 10-mg option for stated criteria (BW regimen) or 40 mg, 20-mg option for stated criteria (QW regimen); [d]QW regimen made available July 2023; [e]K dose on C1D1 was 20 mg/m². BMI, body mass index; BW, twice weekly; C, cycle; CR, complete response; D, day; DEX, dexamethasone; DOR, duration of response; IRC, independent review committee; IV, intravenous; K, carfilzomib; ORR, overall response rate; OS, overall survival; PFS2, second progression-free survival; QW, weekly; TTR, time to response; VGPR, very good partial response.

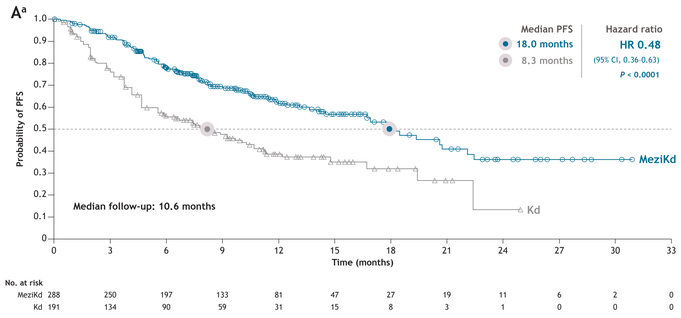
Results

Patients and baseline characteristics

- Overall, 479 patients were included in the analysis
  - 288 in the MeziKd group (randomized to 1.0 mg Mezi in stages 1 and 2)
  - 191 in the Kd group (stages 1 and 2)
- Baseline demographics and patient characteristics were generally well balanced and reflective of a real-world patient population (**Table 1**)
  - Median (range) age was 68 years (30-85) and was similar in the treatment arms
  - 25.1% of patients were ≥ 75 years of age
- Overall, median (range) number of prior lines of therapy (LOTs) was 2 (1-9)
  - 92.1% of patients were triple-class-exposed
  - 37.2% were exposed to pomalidomide (POM) and 7.3% to anti-B-cell maturation antigen (BCMA) treatment
  - Most patients were refractory to anti-CD38 mAbs (85.8%), LEN (75.8%), anti-CD38 mAbs and LEN (68.1%), or their last LOT (92.9%)
- Prior exposure and refractory status per treatment arm are shown in **Table 2**

MeziKd significantly improved PFS vs Kd, reducing the risk of progression or death by 52%; the benefit with MeziKd was consistent across all prespecified subgroups

Figure 3. (A) PFS (primary endpoint), (B) PFS subgroup analysis



B[b]

Subgroup	N	Favors MeziKd / Favors Kd	HR (95% CI)
Age, years			
< 65	181		0.52 (0.34-0.80)
65 to < 75	178		0.43 (0.27-0.67)
≥ 75	120		0.52 (0.29-0.94)
Baseline ISS disease stage			
I	245		0.42 (0.28-0.64)
II	146		0.51 (0.31-0.84)
III	88		0.60 (0.34-1.05)
Cytogenetic risk groups			
High risk[c]	136		0.49 (0.30-0.80)
Standard risk[d]	280		0.51 (0.35-0.74)
Soft-tissue plasmacytomas			
Yes	118		0.34 (0.21-0.54)
No	361		0.58 (0.42-0.82)
Extramedullary plasmacytomas			
Yes	45		0.33 (0.16-0.67)
No	434		0.51 (0.38-0.69)
Prior LOTs			
≤ 2	253		0.54 (0.37-0.80)
> 2	226		0.44 (0.30-0.64)
Refractory to anti-CD38 mAbs			
Yes	411		0.48 (0.36-0.64)
No	68		0.50 (0.23-1.08)
Refractory to LEN			
Yes	363		0.50 (0.36-0.67)
No	116		0.40 (0.21-0.76)
Refractory to anti-CD38 mAbs and LEN			
Yes	326		0.47 (0.34-0.65)
No	153		0.48 (0.28-0.82)
Refractory to POM			
Yes	155		0.51 (0.33-0.78)
No	324		0.44 (0.31-0.64)

Data cutoff: January 15, 2026. [a]In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd; [b]In subgroups of the confirmatory analysis group; [c]Defined as presence of del(17p), and/or translocation t(4;14), and/or translocation t(14;16); [d]Defined as absence of del(17p), translocation t(4;14), and translocation t(14;16).

Table 1. Baseline characteristics

	MeziKd (N = 288)	Kd (N = 191)
Age, median (range), years	68.0 (40-84)	67.0 (30-85)
≥ 75 years	75 (26.0)	45 (23.6)
Male sex, n (%)	146 (50.7)	106 (55.5)
Race, n (%)		
White	162 (56.3)	114 (59.7)
Asian	90 (31.3)	57 (29.8)
Black or African American	11 (3.8)	13 (6.8)
American Indian	10 (3.5)	3 (1.6)
Unknown or missing	15 (5.2)	4 (2.1)
Time since initial diagnosis, median (range), years	4.6 (0.4-30.9)	4.5 (0.5-20.9)
ECOG PS score, n (%)[a]		
0	116 (40.3)	74 (38.7)
1	154 (53.5)	103 (53.9)
2	16 (5.6)	14 (7.3)
ISS stage at study entry, n (%)		
I	149 (51.7)	96 (50.3)
II	87 (30.2)	59 (30.9)
III	52 (18.1)	36 (18.8)
Soft-tissue plasmacytomas, n (%)	66 (22.9)	52 (27.2)
Extramedullary plasmacytomas, %	8.7	10.5
Parasketetal plasmacytomas, %	17.7	17.8
High-risk cytogenetics, n (%)[b]	74 (25.7)[c]	62 (32.5)[d]
Expanded high-risk cytogenetics, n (%)[e]	162 (56.3)[f]	112 (58.6)[g]

Data cutoff: January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd. Percentages may not total 100 due to rounding. [a]2 patients in the MeziKd arm had ECOG PS score 1 during screening per eligibility requirements of 0-2 and ECOG PS score 3 at baseline; [b]Defined as presence of del(17p), and/or translocation t(4;14), and/or translocation t(14;16); [c]36/288 patients were NE; [d]27/191 patients were NE; [e]Defined as presence of del(17p), and/or translocation t(4;14), and/or translocation t(14;16) and/or amplification 1q21; [f]38/288 patients were NE; [g]9/191 were NE. ECOG PS, Eastern Cooperative Oncology Group performance status; ISS, International Staging System; NE, not evaluable or data missing.

Table 2. Prior therapy

	MeziKd (N = 288)	Kd (N = 191)
Prior LOTs, median (range)	2.0 (1-9)	2.0 (1-8)
No. of prior LOTs, n (%)		
1 prior LOT	43 (14.9)	33 (17.3)
2 prior LOTs	109 (37.8)	68 (35.6)
3 prior LOTs	64 (22.2)	35 (18.3)
4+ prior LOTs	72 (25.0)	55 (28.8)
Prior therapy exposure, n (%)		
Anti-CD38 mAbs	288 (100)	191 (100)
LEN	288 (100)	191 (100)
Triple-class exposed	265 (92.0)	176 (92.1)
POM	114 (39.6)	64 (33.5)
BCMA-targeted therapy	23 (8.0)	12 (6.3)
T cell-redirecting therapy	28 (9.7)	12 (6.3)
Refractory status, n (%)[a]		
To last prior LOT	264 (91.7)	181 (94.8)
Anti-CD38 mAbs	248 (86.1)	163 (85.3)
LEN	220 (76.4)	143 (74.9)
POM	99 (34.4)	56 (29.3)
PI	138 (47.9)	102 (53.4)
Anti-CD38 mAbs and LEN	199 (69.1)	127 (66.5)
Triple-class refractory[b]	111 (38.5)	75 (39.3)

Data cutoff: January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd. Percentages may not total 100 due to rounding. [a]Refractory is defined as disease that is nonresponsive on therapy (failure to achieve minimal response or development of progressive disease while on therapy) or progresses within 60 days of last dose; [b]Triple-class refractory is defined as refractory to an IMiD, PI, and anti-CD38 mAb.

- At data cutoff (January 15, 2026), median follow-up was 10.6 months, discontinuations due to adverse events (AEs) were infrequent, and Mezi relative dose intensity was high (83.1%) (**Table 3**)
- Median treatment duration was 8.9 months for MeziKd vs 6.2 months for Kd
  - 52.4% of patients in the MeziKd arm and 31.4% in the Kd arm remained on treatment at data cutoff

Table 3. Patient disposition and treatment exposure

	MeziKd (N = 288)	Kd (N = 191)
Patients who received treatment, n (%)	288 (100)	186 (97.4)
Ongoing treatment	151 (52.4)	60 (31.4)
Discontinued treatment	137 (47.6)	126 (66.0)
Progressive disease	85 (29.5)	93 (48.7)
AE	28 (9.7)	11 (5.8)
Physician decision	12 (4.2)	6 (3.1)
Withdrawal	8 (2.8)	16 (8.4)
Treatment duration, median (range), months	8.9 (0.3-32.1)	6.2 (0.2-25.0)
Mezi relative dose intensity, median, %	83.1	-
Patients with Mezi dose reductions, n (%)	122 (42.4)	-
K relative dose intensity, median, %	86.8	94.0
Patients with K dose reductions, n (%)	101 (35.1)	41 (22.0)[a]
DEX relative dose intensity, median, %	78.1	93.0
Patients with DEX dose reductions, n (%)	106 (36.8)	58 (31.2)[a]

Data cutoff January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd. Percentages may not total 100 due to rounding. [a]5 patients were randomized but not treated. AE, adverse event.

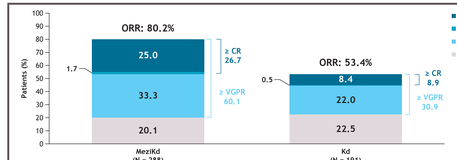
Efficacy: primary endpoint

- MeziKd significantly improved PFS vs Kd, reducing the risk of progression or death by 52% (median [95% confidence interval, CI], 18.0 [14.5-22.1] vs 8.3 [5.6-10.7] months; hazard ratio [HR], 0.48 [95% CI, 0.36-0.63]; $P < 0.0001$) (**Figure 3A**)
- The superior PFS survival benefit with MeziKd was consistent across all prespecified subgroups (**Figure 3B**)
  - This included patients with > 2 prior LOTs, prior treatment exposure/refractoriness, high-risk cytogenetics, extramedullary disease, and age ≥ 75 years

Efficacy: secondary endpoints

- The addition of Mezi to Kd deepened response, doubling the rate of VGPR or better and tripling the rate of complete response (CR) or better (**Figure 4**)
  - Higher ORR (80.2% vs 53.4%) and CR or better (26.7% vs 8.9%) was observed with MeziKd vs Kd
  - Median time to response was 1.1 months with each treatment
  - The 12-month DOR rate with MeziKd was numerically higher than with Kd

Figure 4. Treatment response

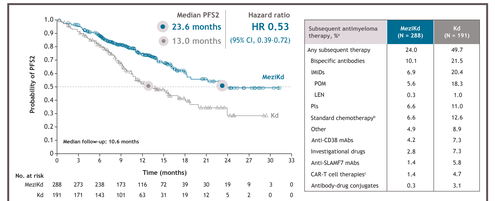


	MeziKd (N = 288)	Kd (N = 191)
Time to response, median (range), months[a]	1.1 (0.7-7.4)	1.1 (0.9-7.9)
12-month DOR, % (95% CI)	72 (65-78)	54 (41-66)

Data cutoff: January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd. Best overall response was evaluated by an IRC. Percentages may not total 100 due to rounding. [a]Time to response is defined as the time from randomization to the first documentation of response (PR or better). PR, partial response; sCR, stringent complete response.

- PFS2 with MeziKd was superior to Kd (**Figure 5**)
  - MeziKd demonstrated sustained clinical benefit beyond first progression

Figure 5. PFS2

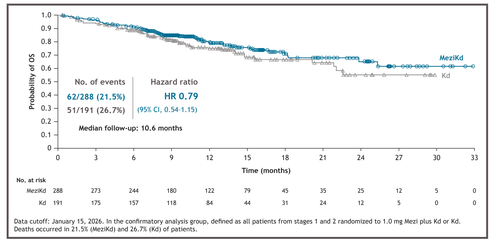


Subsequent antimyeloma therapy, %[a]	MeziKd (N = 288)	Kd (N = 191)
Any subsequent therapy	24.0	49.7
Bispecific antibodies	10.1	21.5
IMiDs	6.9	20.4
POM	5.6	18.3
LEN	0.3	1.0
PIs	6.6	11.0
Standard chemotherapy[b]	6.6	12.6
Other	4.9	8.9
Anti-CD38 mAbs	4.2	7.3
Investigational drugs	2.8	7.3
Anti-SLAMF7 mAbs	1.4	5.8
CAR-T cell therapies[c]	1.4	4.7
Antibody-drug conjugates	0.3	3.1

Data cutoff: January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd. PFS2 was per investigator assessment. [a]Includes subsequent antimyeloma therapies that were received by ≥ 1% of patients in either treatment arm in the confirmatory analysis group; [b]Standard chemotherapy includes: nitrogen mustard analogs, anthracyclines and related substances, podophyllotoxin derivatives, platinum compounds, purine analogs, and vinca alkaloids and analogs; [c]Refers to the output of "Cell and Gene therapies". CAR, chimeric antigen receptor.

- The planned OS futility analysis demonstrates a positive OS trend favoring MeziKd with no crossover of the curves (**Figure 6**)
  - Deaths (21.5% MeziKd vs 26.7% Kd) were mostly due to progressive disease

Figure 6. OS



Data cutoff: January 15, 2026. In the confirmatory analysis group, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd. Deaths occurred in 21.5% (MeziKd) and 26.7% (Kd) of patients.

Safety

- The most common treatment-emergent AEs (TEAEs) and grade 3/4 AEs are shown in **Table 4**
- Neutropenia, which is a known on-target and reversible AE for Mezi, was the most common grade 3/4 AE (61.1%) with MeziKd
  - Neutropenia was managed effectively with dose interruptions/modifications and/or granulocyte colony-stimulating factor (G-CSF)
  - Only 1 patient discontinued due to neutropenia with MeziKd (none with Kd)
- A reduction in grade 3/4 hypertension was observed with MeziKd, potentially associated with the lower K dose
- Grade 3/4 venous thromboembolism occurred in 2.8% (MeziKd) and 0.5% (Kd) of patients

Table 4. Most common TEAEs

TEAE,[a] n (%)	MeziKd (N = 288) Any grade	MeziKd (N = 288) Grade 3/4	Kd (N = 186)[b] Any grade	Kd (N = 186)[b] Grade 3/4
Any TEAE	286 (99.3)	241 (83.7)	178 (95.7)	105 (56.5)
Hematologic				
Neutropenia[c]	199 (69.1)	176 (61.1)	32 (17.2)	17 (9.1)
Febrile neutropenia	24 (8.3)	23 (8.0)	0	0
Thrombocytopenia[d]	174 (60.4)	113 (39.2)	77 (41.4)	42 (22.6)
Anemia	149 (51.7)	75 (26.0)	66 (35.5)	28 (15.1)
White blood cell count decrease	67 (23.3)	54 (18.8)	23 (12.4)	7 (3.8)
Nonhematologic				
Diarrhea	112 (38.9)	10 (3.5)	33 (17.7)	1 (0.5)
URTI	79 (27.4)	13 (4.5)	31 (16.7)	3 (1.6)
Fatigue	67 (23.3)	9 (3.1)	39 (21.0)	7 (3.8)
Cough	66 (22.9)	1 (0.3)	22 (11.8)	0
Pneumonia	58 (20.1)	45 (15.6)	21 (11.3)	11 (5.9)
Dyspnea	58 (20.1)	11 (3.8)	26 (14.0)	5 (2.7)
Hypertension	31 (10.8)	11 (3.8)	40 (21.5)	17 (9.1)

Data cutoff: January 15, 2026. [a]Includes TEAEs of any grade that occurred in ≥ 20% of patients and grade 3/4 TEAEs that occurred in ≥ 10% of patients in either treatment arm in the confirmatory analysis group of the safety population, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd who received ≥ 1 dose of study treatment. TEAEs were graded per the National Cancer Institute Common Terminology Criteria for Adverse Events version 5.0; [b]Excludes 5 patients who were randomized but not treated; [c]Includes preferred terms of neutropenia and neutrophil count decrease; [d]Includes preferred terms of thrombocytopenia and platelet count decrease. URTI, upper respiratory tract infection.

- Grade 5 TEAEs were observed in 7.3% (MeziKd) and 4.3% (Kd) of patients, mostly in the context of myeloma progression
- Infections were mostly well managed following standard clinical practice and supportive care with a low incidence of grade 4 events (**Table 5**)
  - Most infections (68.6% MeziKd; 98.0% Kd) were not associated with grade 3/4 neutropenia
- The incidence of hypogammaglobulinemia was low (10.1% MeziKd; 6.5% Kd)
  - 31.6% of patients in the MeziKd group received ≥1 dose of immunoglobulin replacement therapy (IGRT) vs 21.0% in the Kd group
- The incidence of fatal infections was low (2.4% MeziKd; 1.1% Kd)

Table 5. Summary of infections

TEAE,[a] n (%)	MeziKd (N = 288) Any grade	MeziKd (N = 288) Grade 3	MeziKd (N = 288) Grade 4	Kd (N = 186)[b] Any grade	Kd (N = 186)[b] Grade 3	Kd (N = 186)[b] Grade 4
Any infection	210 (72.9)	82 (28.5)	16 (5.6)	100 (53.8)	28 (15.1)	1 (0.5)
URTI	79 (27.4)	13 (4.5)	0	31 (16.7)	3 (1.6)	0
Pneumonia	58 (20.1)	39 (13.5)	6 (2.1)	21 (11.3)	10 (5.4)	1 (0.5)
Influenza	30 (10.4)	9 (3.1)	0	18 (9.7)	2 (1.1)	0
COVID-19	29 (10.1)	5 (1.7)	0	11 (5.9)	0	0
RTI	29 (10.1)	7 (2.4)	0	8 (4.3)	0	0
UTI	26 (9.0)	8 (2.8)	0	9 (4.8)	1 (0.5)	0
Nasopharyngitis	17 (5.9)	0	0	18 (9.7)	0	0
Bronchitis	17 (5.9)	6 (2.1)	0	7 (3.8)	0	0

Data cutoff: January 15, 2026. As of December 2024, *Pneumocystis jirovecii* pneumonia prophylaxis was required in both arms. [a]TEAEs that occurred in ≥ 5% of patients in either treatment arm in the confirmatory analysis group of the safety population, defined as all patients from stages 1 and 2 randomized to 1.0 mg Mezi plus Kd or Kd who received ≥ 1 dose of study treatment; [b]Excludes 5 patients who were randomized but not treated. RTI, respiratory tract infection; UTI, urinary tract infection.

Conclusions

- MeziKd showed clinically meaningful PFS benefit with a predictable and manageable safety profile in predominantly triple-class-exposed, anti-CD38 mAb- and LEN-refractory patients
  - MeziKd significantly reduced risk of progression or death by 52% vs Kd (PFS 18.0 vs 8.3 months)
  - This benefit was sustained across all prespecified subgroups, regardless of prior therapy, age, refractory status, cytogenetic risk, or presence of extramedullary disease
  - Neutropenia was the most common grade 3/4 AE, manageable with dose modifications and/or G-CSF
  - Infections were generally well managed following standard clinical practice and supportive care
  - There were no new safety signals
- These data support MeziKd as a potential new standard of care for RRMM that can be easily used across diverse care settings, including community practice

References

1. Ramasamy K, et al. *Clin Lymphoma Myeloma Leuk* 2025;25:337-348.
2. Mancuso K, et al. *Cancers (Basel)* 2025;17:1168.
3. Gay F, et al. *Front Oncol* 2025;15:1550644.
4. Watson ER, et al. *Science* 2022;378:549-553.
5. Dimopoulos M, et al. *Clin Lymphoma Myeloma Leuk* 2024;24(suppl 2):S258.
6. Sandhu I, et al. *Blood* 2024;144(suppl 1):1025.

Acknowledgments

- The patients and families who are making the study possible
- The clinical study teams who are participating
- All the investigators of the SUCCESSOR-2 trial
- This study was supported by Celgene, a Bristol-Myers Squibb Company
- All authors contributed to and approved the presentation; writing and editorial assistance were provided by Mauro Locati, PhD, and Sarah Spaeh, PhD, of the Publications Division of Omnicom Health Medical Communications, funded by Bristol Myers Squibb

Presented at the Society of Hematologic Oncology (SOHO) Annual Meeting; September 9-12, 2026, Chicago, IL, USA.
Previously presented at the 2026 American Society of Clinical Oncology (ASCO) annual meeting; May 29-June 2, 2026; Chicago, IL, USA.