# UPDATED RESULTS OF THE ASPEN TRIAL FROM A COHORT OF PATIENTS WITH MYD88 WILD-TYPE WALDENSTRÖM MACROGLOBULINEMIA

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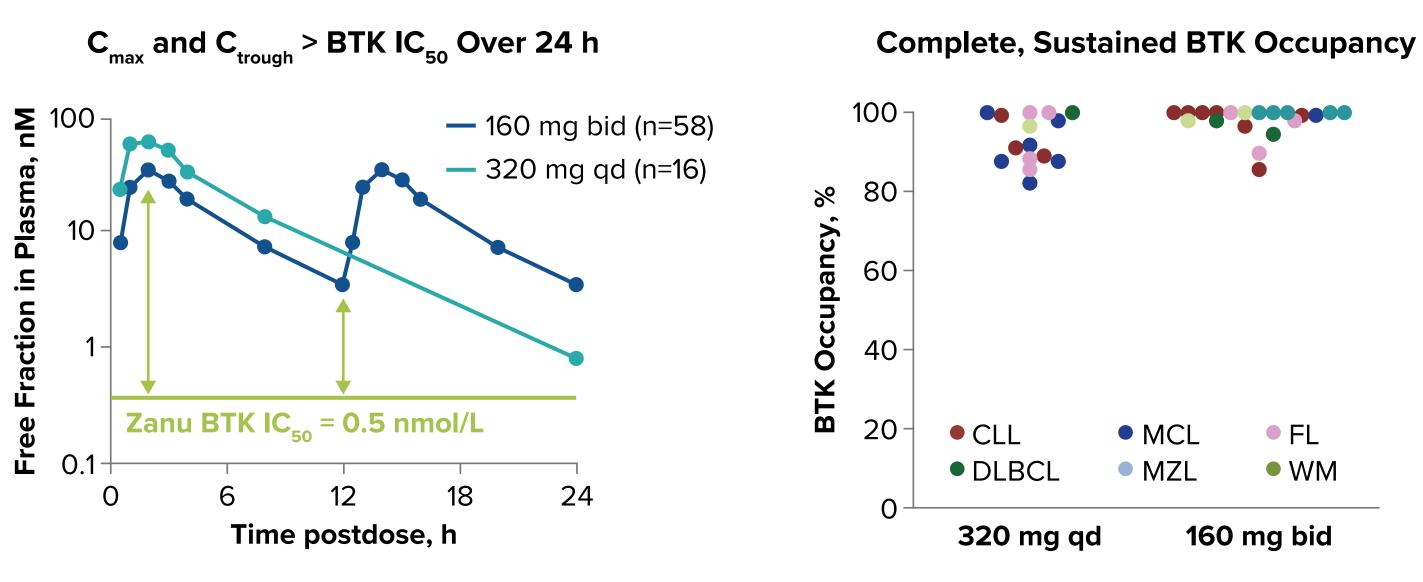
## INTRODUCTION

- Bruton tyrosine kinase (BTK) plays a critical role in B-cell receptor signaling, which mediates B-cell proliferation, migration, and adhesion<sup>1-3</sup>
- First-generation BTK inhibitor ibrutinib has shown activity in Waldenström macroglobulinemia (WM) and has become a standard of care<sup>4</sup>
- However, lower response rates,<sup>5</sup> no major responses,<sup>5,6</sup> and shorter survival<sup>7</sup> have been reported in patients (pts) who lack  $MYD88^{L265P}$  or other activating mutations ( $MYD88^{WT}$ )
- Zanubrutinib is a next-generation BTK inhibitor designed to maximize BTK occupancy and minimize off-target inhibition of
- TEC- and EGFR-family kinases (Figure 1)
- Potent, selective, and irreversible<sup>8</sup>
- Equipotent against BTK compared with ibrutinib; higher selectivity vs EGFR, ITK, JAK3, HER2 and TEC9
- Advantageous pharmacokinetic/pharmacodynamic properties: complete and sustained BTK occupancy in
- peripheral blood mononuclear cells and lymph nodes<sup>8</sup> - Favorable drug-drug interaction properties: can be
- co-administered with strong/moderate CYP3A inhibitors at a reduced dose, proton pump inhibitors, acid-reducing agents, and anti-thrombotic agents 10,11
- Approved for treatment of pts with relapsed/refractory (R/R) mantle cell lymphoma in the United States 11/2019<sup>12</sup>
- Figure 1a. Zanubrutinib: A Potent and Selective BTK Inhibitor<sup>8,9</sup>

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	Targets	Assays	Zanubrutinib IC <sub>50</sub> (nM)	Ibrutinib IC <sub>50</sub> (nM)	Ratio (Zanubrutinib:Ibrutinib)
		BTK-pY223 Cellular Assay	1.8	3.5	0.5
ON TARGET	BTK	Rec-1 Proliferation	0.36	0.34	1.1
		BTK Occupation Cellular Assay	2.2	2.3	1
		BTK Biochemical Assay	0.22	0.2	1.1
	EGFR	p-EGFR HTRF Cellular Assay	606	101	6
		A431 Proliferation	3210	323	9.9

	EGFR	p-EGFR HTRF Cellular Assay	606	101	6
		A431 Proliferation	3210	323	9.9
	ITK	ITK Occupancy Cellular Assay	606	189	17
TARGET		p-PLCγ1 Cellular Assay	3433	77	45
OFF TA		IL-2 Production Cellular Assay	2536	260	9.8
		ITK Biochemical Assay	30	0.9	33
	JAK3	JAK3 Biochemical Assay	200	3.9	51
	HER2	HER2 Biochemical Assay	661	9.4	70
	TEC	TEC Biochemical Assay	1.9	0.8	2.4

Figure 1b. Complete, Sustained BTK Occupancy With bid or qd Dosing<sup>8,9</sup>



bid, twice daily; BTK, Bruton tyrosine kinase; CLL, chronic lymphocytic leukemia; C<sub>max</sub>, maximum concentration; C<sub>trough</sub>, trough concentration; DLBCL, diffuse large B-cell lymphoma; EGFR, epidermal growth factor receptor; FL, follicular lymphoma; HER2, human epidermal growth factor receptor 2; HTRF, homogeneous time resolved fluorescence; IC<sub>50</sub>, half maximal inhibitory concentration; ITK, IL-2–inducible T-cell kinase; JAK3, Janus-associated kinase 3; MCL, mantle cell lymphoma; MZL, marginal zone lymphoma; PLC, phospholipase C; qd: once daily; WM, Waldenström macroglobulinemia; Zanu, zanubrutinib.

# OBJECTIVE

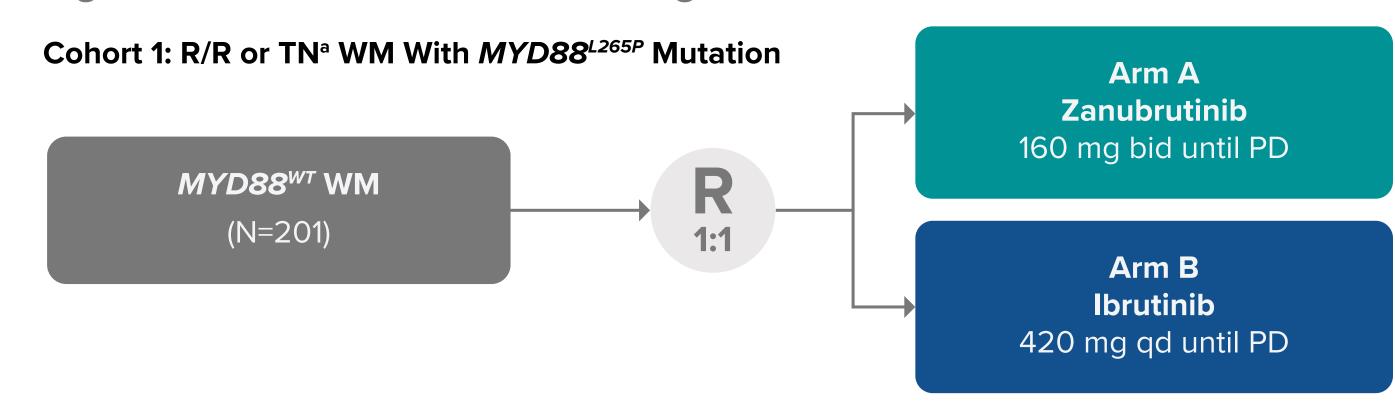
• To assess the safety and efficacy of zanubrutinib in WM pts with MYD88WT from an exploratory cohort of the ongoing phase 3 study of zanubrutinib vs ibrutinib in pts with WM (ASPEN;

# METHODS

Zanubrutinib (BGB-3111)

• ASPEN is an open-label, multicenter, randomized, phase 3 study of zanubrutinib vs ibrutinib in pts with WM (Figure 2)

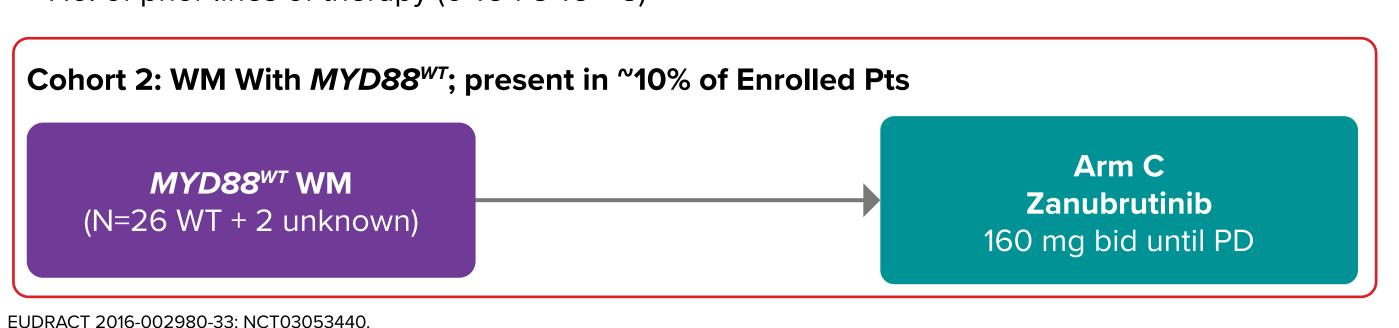
### Figure 2. Phase 3 ASPEN Trial Design



**Stratification factors:** • CXCR4 mutational status (CXCR4WHIM vs CXCR4WT vs missing)

<sup>a</sup>TN must be unsuitable for standard chemoimmunotherapy.

No. of prior lines of therapy (0 vs 1-3 vs > 3)



- Clinical and definitive histologic diagnosis of WM, with measurable disease (serum IqM >0.5 g/dL), and meeting ≥1 criterion for treatment according to consensus panel criteria from the
- If treatment naïve, must be considered by treating physician unsuitable for standard chemoimmunotherapy regimens
- Eastern Cooperative Oncology Group performance status 0-2

bid, twice daily; PD, progressive disease; pt, patient; qd, once daily; R/R, relapsed/refractory; TN, treatment-naïve.

- Absolute neutrophil count ≥750/µL, platelets ≥50000/µL (independent of growth factor/ transfusions)
- Adequate renal, hepatic, and coagulation function

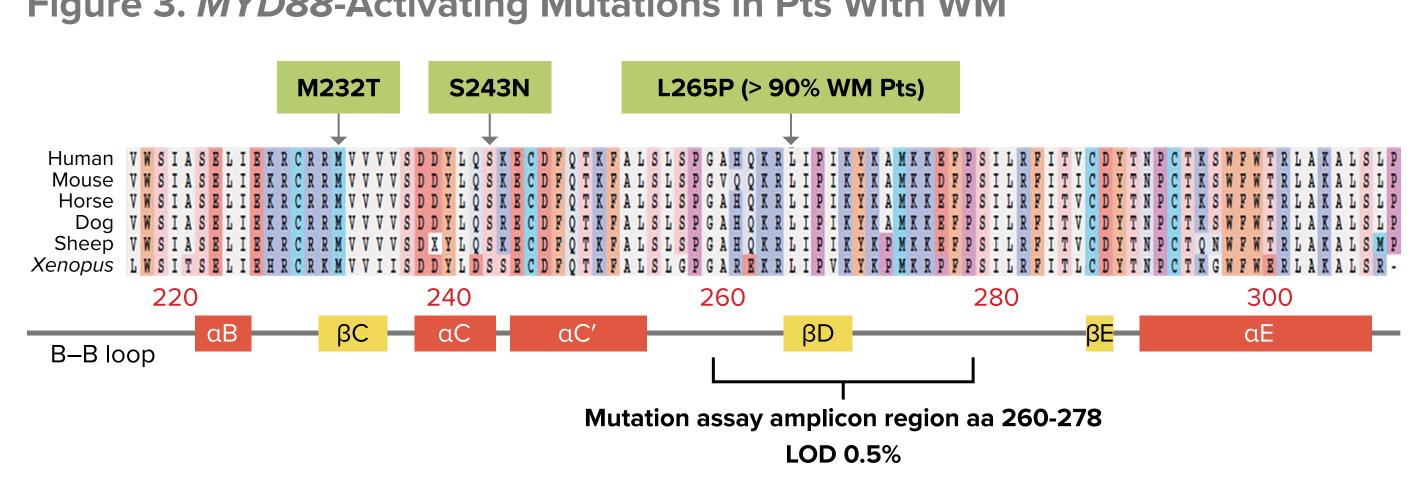
Seventh International Workshop on WM<sup>13</sup>

• No significant cardiac disease, active central nervous system involvement, or prior BTK inhibitors

### **Cohort Assignment**

- Bone marrow MYD88 and CXCR4 mutations were assessed centrally at study entry (NeoGenomics Laboratory, Aliso Viejo, CA, USA)<sup>14</sup>
- The MYD88 mutation assay used detects all mutations in the region encompassing amino acid Ala<sup>260</sup>-Pro<sup>278</sup>, which includes the predominant mutation in WM, *MYD88*<sup>L265P</sup> (**Figure 3**)
- Pts were assigned to cohort 1 (MYD88 mutated; randomized) or exploratory cohort 2 (MYD88<sup>WT</sup> or MYD88 unknown, nonrandomized) based on the central laboratory MYD88 mutation assay results

Figure 3. MYD88-Activating Mutations in Pts With WM



- Detection in the MYD88 amplicon (Ala<sup>260</sup>-Pro<sup>278</sup>) by the NeoGenomics LDT assay includes a wild-type-allele-blocking approach (limit of detection [LOD], 0.5%)<sup>14</sup> versus standard polymerase chain reaction/bidirectional Sanger sequencing assay used to detect CXCR4 mutations (LOD, 10%-15%)
- For MYD88<sup>WT</sup> pts with available samples (12 of 26), MYD88 mutations were also evaluated by next-generation sequencing (200×; LOD, 5%); no other activating mutations were detected

Adapted from Treon et al<sup>6</sup> and Ngo et al<sup>15</sup> LOD, limit of detection; pt, patient; WM, Waldenström macroglobulinemia.

### **Exploratory End Points for Cohort 2**

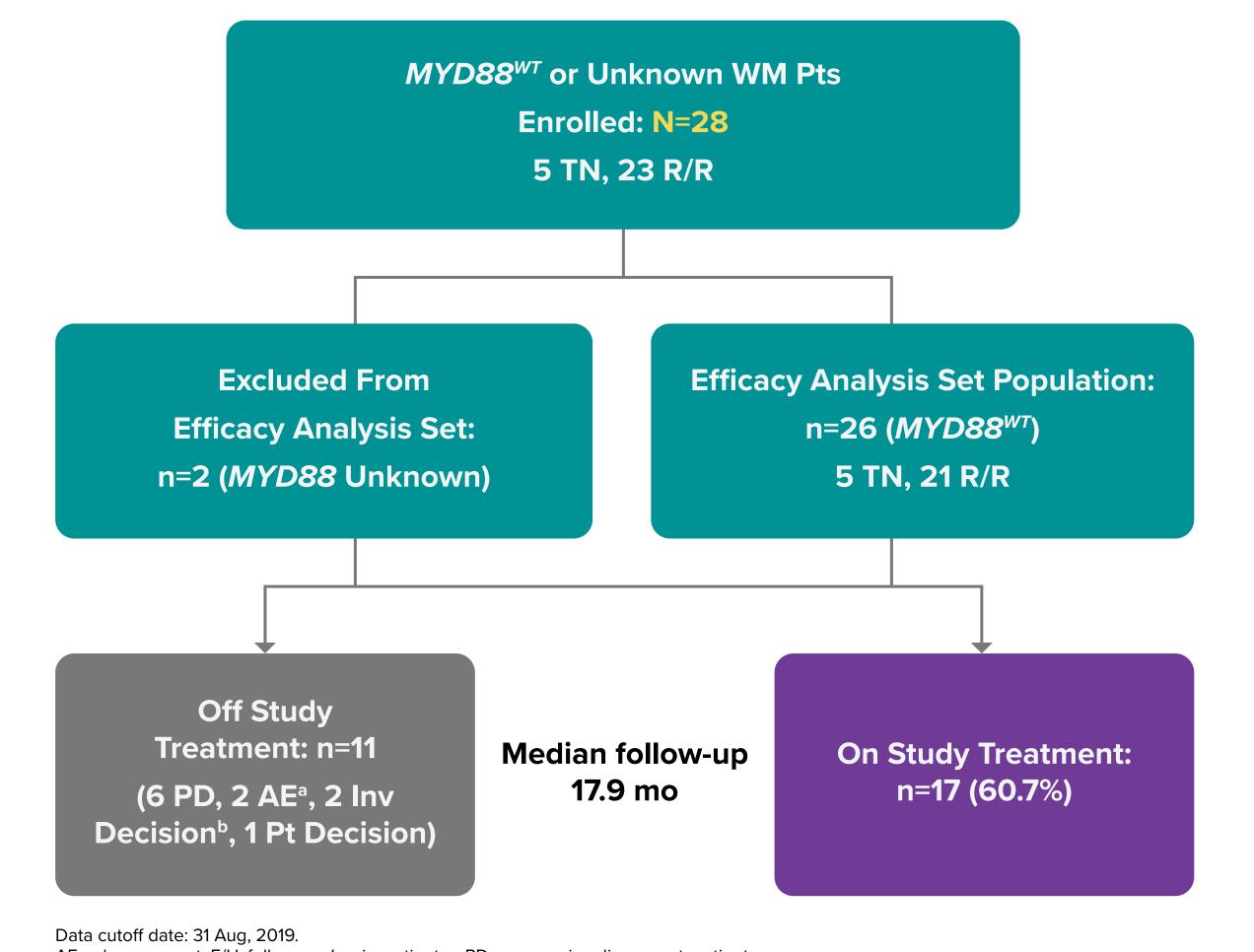
- Responses were assessed monthly by immunoglobulin M (IgM) with extramedullary disease assessment every 3 months, according to response criteria in the National Comprehensive Cancer Network WM guidelines<sup>16</sup> and modified Owen criteria<sup>17</sup> as assessed by the independent review committee
- Efficacy: response rates (overall and major response rate), duration of response, progression-free survival, and overall survival; safety assessed according to National Cancer Institute Common Terminology Criteria for Adverse Events

# RESULTS

version 4.03

- In total, 28 pts (n=26 MYD88<sup>WT</sup>; n=2 MYD88 mutation status unknown) were enrolled into cohort 2
- The safety analysis set includes all 28 pts, and the efficacy analysis set includes 26 MYD88<sup>WT</sup> pts, with a median follow-up of 17.9 months (range, 2.3-27.8; Figure 4 and Table 1)

Figure 4. Disposition of Pts in Cohort 2



AE, adverse event; F/U, follow-up; Inv, investigator; PD, progressive disease; pt, patient. <sup>a</sup>Grade 4 subdural hemorrhage; grade 3 diarrhea. blnvestigator decided no further treatment needed (n=1); pt discharged to hospice for palliative care (n=1).

### **Table 1. Pt and Disease Characteristics**

Characteristic	Total (N=28)
Age, median (range), y	70.1 (39-87)
>65 years, n (%)	19 (67.9)
>75 years, n (%)	12 (42.9)
Sex, n (%)	
Men	14 (50)
Women	14 (50)
IPSSWM, n (%)	
Low	5 (17.9)
Intermediate	11 (39.3)
High	12 (42.9)
Prior treatment status	
Treatment-naïve, n (%)	5 (17.9)
R/R, n (%)	23 (82.1)
No. of prior therapies for R/R pts, median (range)	1 (1-5)
Extramedullary disease present at baseline by IRC, n (%)	21 (75.0)
Genotype, n (%)	
MYD88WT/CXCR4WT	23 (82.1)
MYD88 <sup>WT</sup> /CXCR4 <sup>WHIM</sup>	1 (3.6)
MYD88 <sup>WT</sup> /CXCR4 unknown	2 (7.1)
MYD88 unknown/CXCR4 unknown	2 (7.1)
Hemoglobin ≤110 g/L, n(%)	15 (53.6)

### **Table 2. AE Overview**

Treatment Emergent AE	n (%)		
Pts with ≥1 AE grade ≥3	18 (64.3)		
Pts with ≥1 serious AE	11 (39.3)		
AE leading to death	Ο		
AE leading to treatment discontinuation	2° (7.1)		
AE leading to dose reduction	2 <sup>b</sup> (7.1)		
AE, adverse event; pt, patient; SAE, serious adverse event.			

<sup>a</sup>Grade 4 subdural hemorrhage (related) and grade 3 diarrhea (related). <sup>b</sup>Grade 3 pneumonitis resolved and followed by grade 2 pneumonia (n=1); grade 1 diarrhea (n=1).

- No treatment-emergent AEs leading to death (Table 2)
- 2 Pts discontinued because of AEs
- Grade 4 subdural hemorrhage
- Grade 3 diarrhea

### Major hemorrhage occurred in 2 pts (Table 3)

- Gastric ulcer haemorrhage
- Periorbital hematoma, subdural hematoma, and subdural hemorrhage; treatment was permanently discontinued per protocol
- Atrial fibrillation/flutter occurred in 1 pt (grade 1)
- Most common adverse events (AEs; in >15% pts) were diarrhea, anemia, contusion, pyrexia, upper respiratory tract infection, respiratory tract infection, and cough (Figure 5)

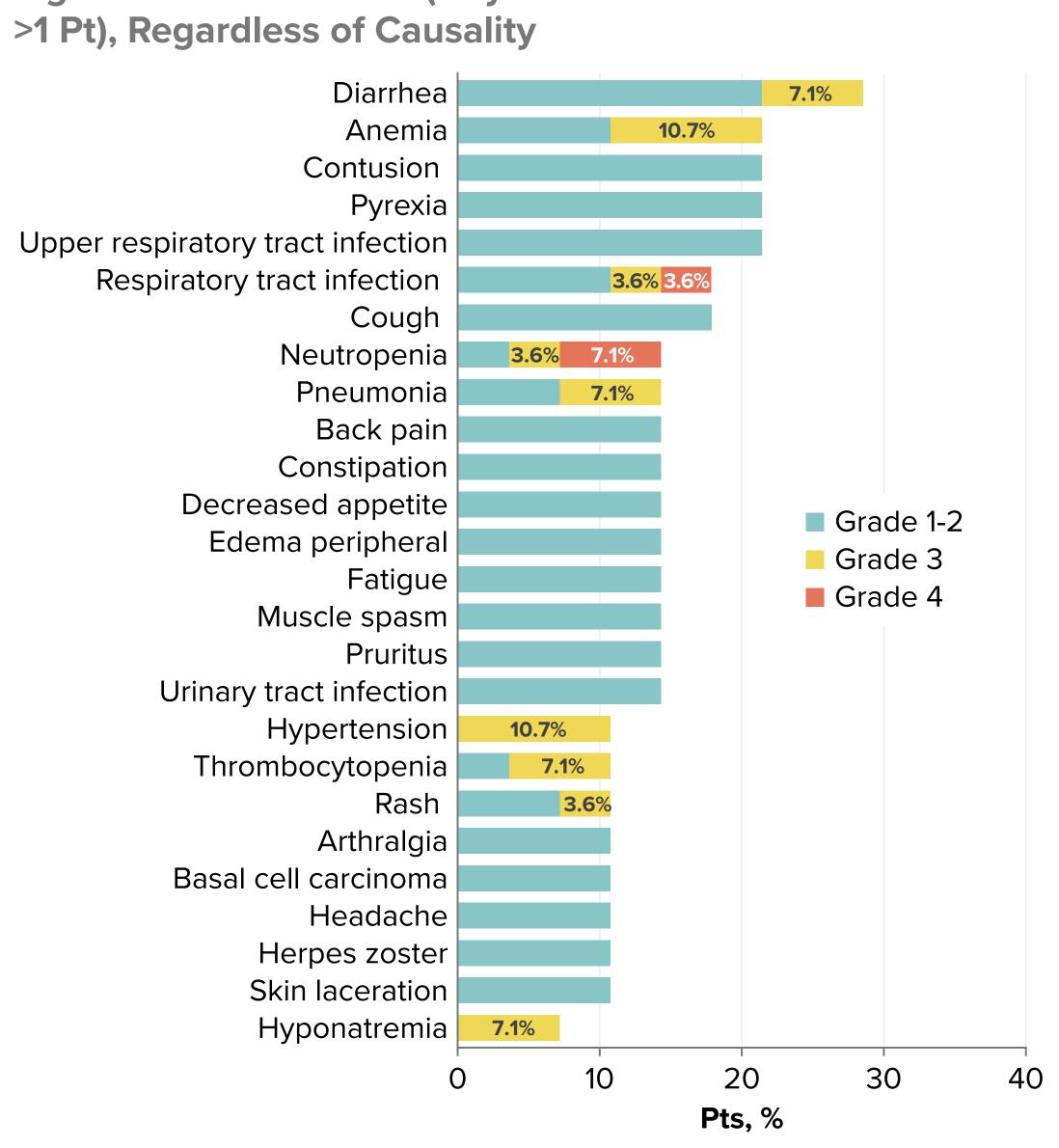
Table 3. AE Categories of Interest (BTKi Class AEs)

All Grade	Grade ≥3
1 (3.6)	0
8 (28.6)	2 (7.1)
11 (39.3)	2 (7.1)
2 (7.1)	2 (7.1)
3 (10.7)	3 (10.7)
5 (17.9)	3 (10.7)
21 (75.0)	8 (28.6)
4 (14.3)	0
	1 (3.6) 8 (28.6) 11 (39.3) 2 (7.1) 3 (10.7) 5 (17.9) 21 (75.0)

No tumor lysis syndrome or opportunistic infection was reported. AE, adverse event; BTKi, Bruton tyrosine kinase inhibitor; PT, preferred term.

<sup>a</sup>Defined as any grade ≥3 hemorrhage or any grade central nervous system hemorrhage: gastric ulcer hemorrhage; and 1 patient had periorbital hematoma, subdural hematoma, and subdural hemorrhage. blncluding PT terms of neutropenia, neutrophil count decreased, febrile neutropenia, agranulocytosis, neutropenic infection, and <sup>c</sup>Basal cell carcinoma (n=3) and Queyrat erythroplasia (n=1).

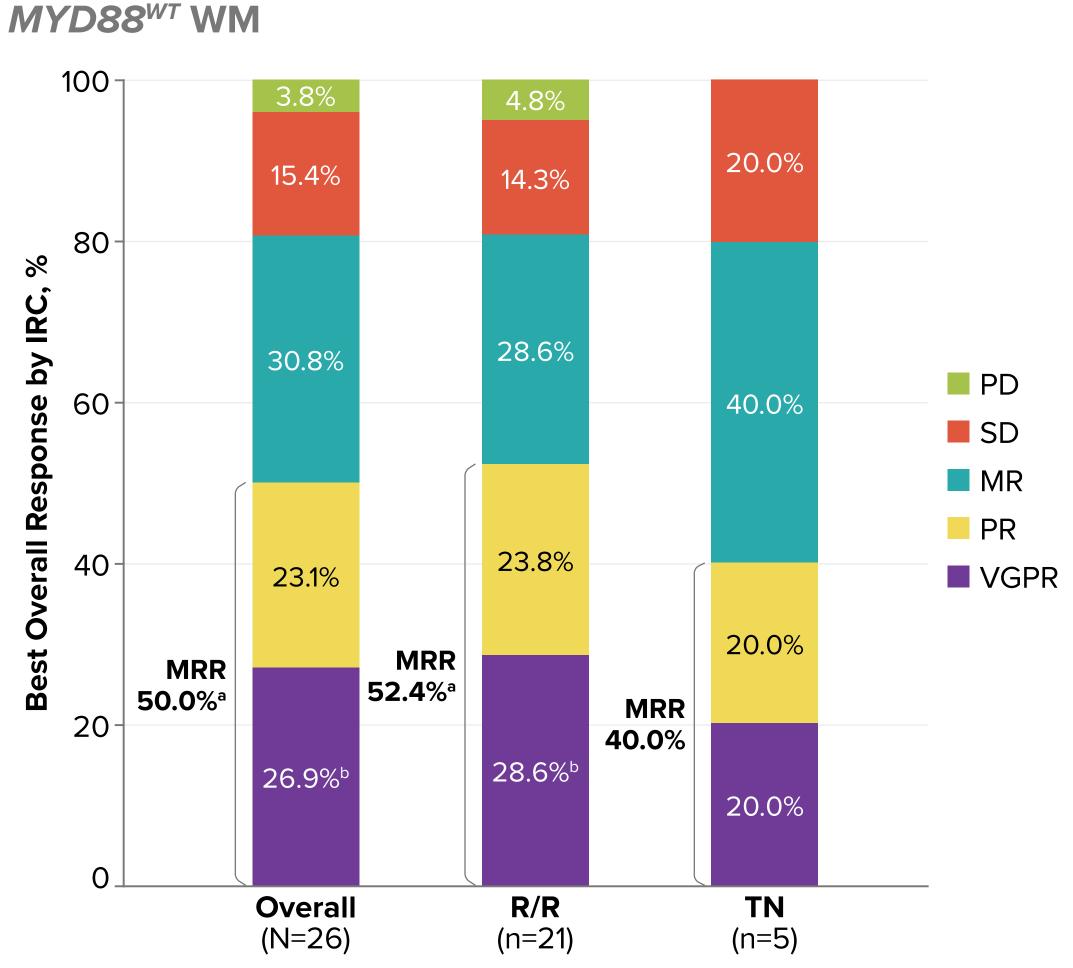
Figure 5. Common AEs (Any Grade >10% or Grade ≥3 in >1 Pt), Regardless of Causality



AE, adverse event; pt, patient.

- Major response rate of 50.0% including 26.9% with VGPR (Figure 6)
- Median time to first major response (partial response or better, requiring) reduction in extramedullary disease if present at baseline) was 2.9 mo (range, 1.9-16.1; **Figure 7**)
- IgM complete response (requiring normal IgM and immunofixation negative) was achieved in 1 pt
- Median progression-free and overall survival were not yet reached (Figure 8)

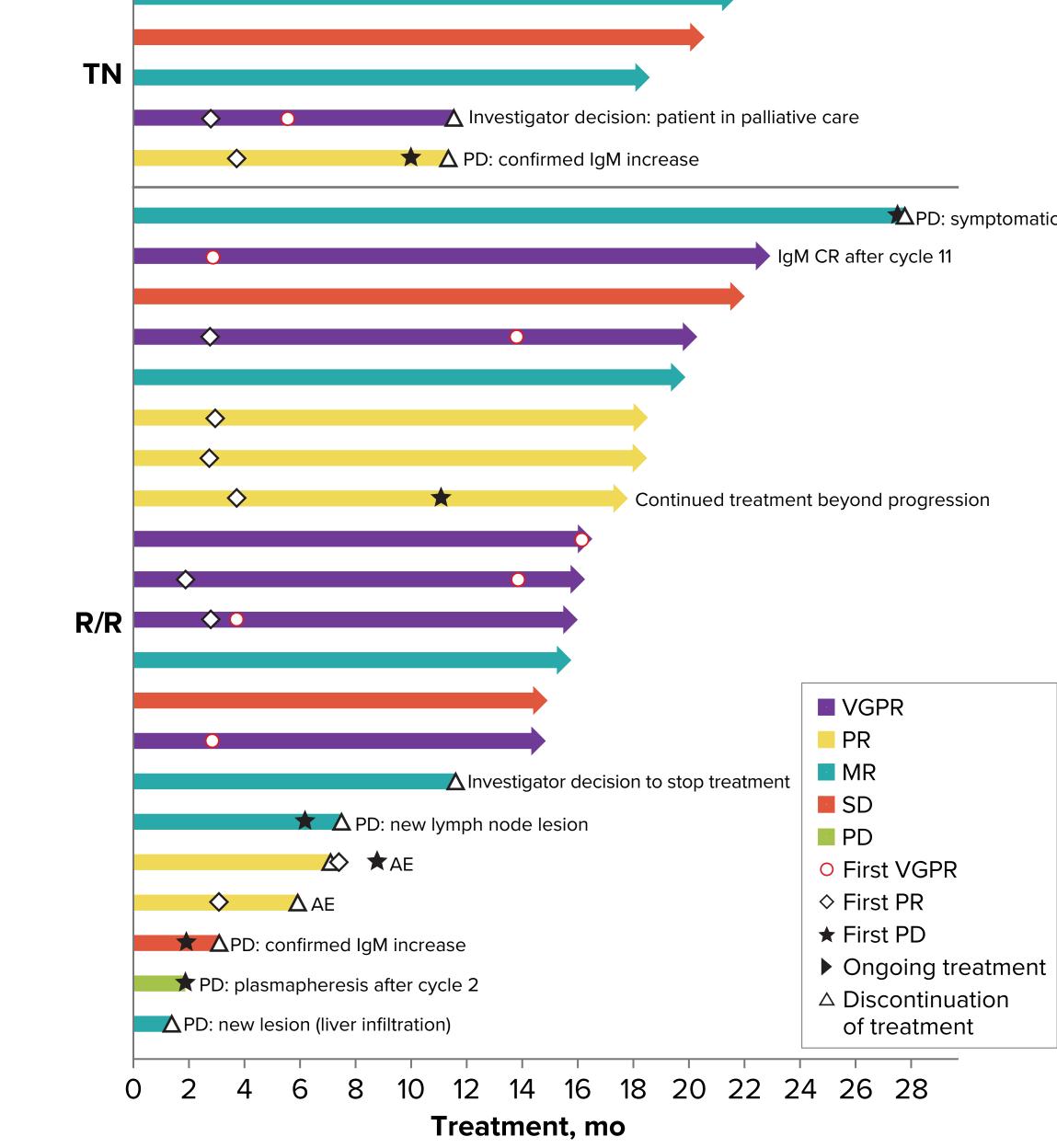
Figure 6. Best Responses by IRC in Patients With



response rate (≥PR); PD, progressive disease; PR, partial response; pt, patient; R/R, relapsed/refractory; SD, stable disease; TN, treatment-naïve: VGPR. verv good PR. <sup>a</sup>lncluding pts confirmed by next-generation sequencing of no other activating MYD88 mutations. bOne pt achieved IgM complete response (normalized IgM and negative immunofixation since cycle 11, with bulky extramedullary disease improving).

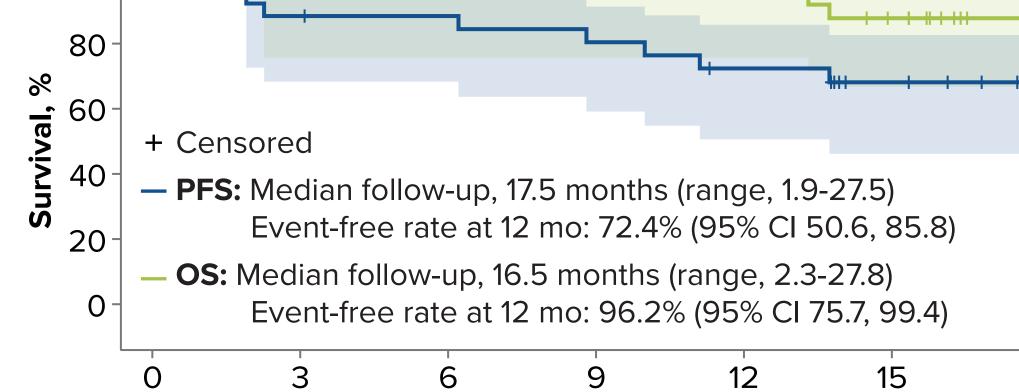
CR, complete response; IgM, immunoglobulin M; IRC, independent review committee; MR, minor response; MRR, major

# Figure 7. Responses Over Time on Treatment



Note: color of bars represents the best response for each patient. AE, adverse event; MR, minor response; PD, progressive disease; PR, partial response; R/R, relapsed/refractory; SD, stable disease; TN, treatment-naïve; VGPR, very good PR.

Figure 8. Progression-Free and Overall Survival



No. of pts at risk **OS** 26 Shaded areas show the 95% CI OS, overall survival; PFS, progression-free survival.

# CONCLUSIONS

- Largest cohort of pts with WM with confirmed MYD88<sup>WT</sup> (n=26)
- studied in terms of safety and efficacy of BTKi treatment Single-agent zanubrutinib resulted in major responses
- (including very good partial response)
- Major response rate of 50.0% including 26.9% with very good partial response
- IgM complete response achieved in 1 pt Median time to first major response was 2.9 months
- (range, 1.9-16.1)
- Zanubrutinib was well tolerated
- Discontinuation because of AEs occurred in 7.1% of pts (2/28)
- Primary reason for discontinuation was progressive disease
- (3 of 6 within first 3 cycles)
- No fatal AEs reported Low incidences of atrial fibrillation
- AE profile is consistent with cohort 1 finding in the ASPEN study [abstract S225]

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### CORRESPONDENCE

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### DISCLOSURES

and Amgen; consulting/advisory role with Janssen; research funding from Gilead Sciences and Incyte; and travel expenses from Janssen and Takeda. HL: stock options with CSL and honoraria and advisory board with Janssen and MSD. MT: employment with Charles University General Hospital; honoraria from Janssen, Gilead Sciences, Takeda, Bristol-Myers Squibb, Amgen, AbbVie, Roche, MorphoSys, and Incyte and consulting/advisory role with Takeda, Bristol-Myers Squibb, Incyte, AbbVie, Amgen, Roche, Gilead Sciences, Janssen, Celgene, and MorphoSys. MV: consulting/advisory role with Janssen and Roche and travel expenses from AbbVie and Janssen. SO: honoraria from Roche, Janssen, AbbVie, Gilead, Merck, AstraZeneca, and Novartis; consulting/advisory role with Roche, AstraZeneca, Janssen, AbbVie, Gilead, Merck, and Novartis; research funding from BeiGene, Roche, AstraZeneca, Janssen, Merck, Amgen, and Epizyme; and travel expenses from Roche. SD: research funding from Janssen and BeiGene; speakers' bureau with Amgen; and travel expenses with Janssen. RGO: honoraria from Janssen and Celgene; consulting/advisory role with Janssen; and travel expenses from Janssen. GC: research funding from BeiGene, Acerta, and GlycoMimetics. SM, JC, EA, SG, SR, and JK: nothing to disclose. JC: consulting/advisory role with BeiGene, Janssen, Kymera, and Pharmacyclics and research funding from AbbVie BeiGene, Janssen, Pharmacyclics, and TG Therapeutics, MM: consulting/advisor role with Roche and Jannsen. **TS:** consulting/advisory role with AstraZeneca, Kite Pharma, Juno Therapeutics, BeiGene; research funding with PCYC, Juno, BeiGene, AstraZeneca, TG Therapeutics, and Celgene; and speakers' bureau with PCYC, Jannsen, AstraZeneca, and Seattle Genetics. MGM: speakers' bureau with Celgene and travel expenses from Janssen, Celgene, Amgen, and Takeda. MGG: consulting advisory role with Celgene and Janssen and travel expenses from Janssen. DT: research funding and honoraria from Amgen, Janssen, Roche and Takeda; advisory board membership for Roche, Janssen, and Amgen. PLZ: consulting/advisory role with Verastem, Celltrion, Gilead, Janssen-Cilag, BMS, Servier, Sandoz, MSD, Immune Design, Celgene, Portola, Roche, Eusapharma, Sanofi, and Kyowa Kirin and speakers' bureau with Verastem, Celltrion, Gilead, Janssen-Cilag, BMS, Servier, MSD, Immune Design, Celgene, Portola, Roche, Eusapharma, and Kyowa Kirin. AO: consulting/advisory role with Celgene and Amgen; speakers' bureau with Celgene, Amgen, and Janssen. AT: honoraria with Janssen, AstraZeneca, and AbbVie and consulting/advisory role with Janssen spa, AstraZeneca, and AbbVie. CB: honoraria with Roche, Janssen, Celltrion, and BeiGene; consulting/advisory role with Roche, Janssen, Celltrion, and BeiGene; research funding from Roche, Janssen, and BeiGene; and speakers' bureau with Roche, Janssen, Celltrion, and BeiGene. VL: honoraria from AstraZeneca, Roche, Janssen, Gilead, AbbVie, and Amgen; consulting/advisory role with AstraZeneca, Roche, Janssen, and AbbVie; speaker's bureau for AbbVie and Janssen; and travel expenses from AbbVie, Roche, and Janssen. WYC: employment with BeiGene and equity ownership with BeiGene and Bristol Myers Squibb. JS: employment and equity ownership with BeiGene. SR: employment with BeiGene and equity ownership with BeiGene and Amgen. AC: employment with, equity ownership with, and travel expenses from BeiGene. JH: employment, leadership role, and equity ownership with BeiGene. CST: honoraria from BeiGene, Janssen, AbbVie, Novartis, and Roche and research funding from Janssen and AbbVie.

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