3340

Population Pharmacokinetic and Exposure-response Analyses for Daratumumab in Combination Therapies for Patients With Multiple Myeloma Who Have Received 1 or More Prior Lines of Therapy

Xu Steven Xu,[1,*] Sam Liao,[2] Meletios A. Dimopoulous,[3] Pieter Sonneveld,[4] P. Joy Ho,[5] Andrew Belch,[6] Merav Leiba,[7] Marcelo Capra,[8] David Gomez,[9] Eva Medvedova,[10] Shinsuke Iida,[11] Chang-Ki Min,[12] Ming Qi,[13] Jordan Schecter,[1] Nushmia Z. Khokhar,[13] Xiaoyu Yan,[1] Liping Zhang,[13] Pamela L. Clemens[13]

[1]Janssen Research & Development, LLC, Raritan, NJ, USA; [2]Pharmax Research, Inc, Irvine, CA, USA; [3]National and Kapodistrian University of Athens, Athens, Greece; [4]Department of Hematology, Erasmus MC, Rotterdam, The Netherlands; [5]Royal Prince Alfred Hospital, Camperdown, Australia; [6]Cross Cancer Institute, Edmonton, AB, Canada; [7]Sheba Medical Center Tel Hashomer, Ramat Gan, Israel; [8]Instituto do Cancer COR Hospital Mae de Deus, Porto Alegre, Brazil; [9]Hospital Universitario de la UANL, Monterrey, Nuevo Leon, Mexico; [10]Oregon Health & Science University, Portland, OR, USA; [11]Nagoya City University Hospital, Nagoya, Japan; [12]Seoul St. Mary's Hospital, Seoul, Korea; [13]Janssen Research & Development, LLC, Spring House, PA, USA.

*Presenting author.

INTRODUCTION

- Multiple myeloma (MM) is an incurable disease and, despite advances in treatment such as proteasome inhibitors (PIs) and immunomodulatory drugs (IMiDs), patients inevitably relapse[1,2]
  - Patients who are refractory to PIs and IMiDs have particularly poor prognoses and limited treatment options[3,4]
  - Thus, new treatments and novel treatment combinations are needed
- Daratumumab (DARA) is a human monoclonal antibody targeting CD38 that has direct on-tumor and immunomodulatory mechanisms of action[5-8]
- In monotherapy studies, patients with heavily treated relapsed and refractory MM who were treated with DARA achieved deep and durable responses[9-11]
  - Population pharmacokinetic (PK; PPK) modeling of data from DARA monotherapy studies informed the current recommended dose of DARA 16 mg/kg[12]
- As a monotherapy and in combination with other established regimens, DARA treatment induced rapid, deep, and durable responses with significant clinical benefit in patients treated with ≥1 prior line of therapy[13-15]
  - The FDA recently approved DARA for the treatment of patients with ≥1 prior line of therapy[16]
- Here, DARA PK were evaluated using data from a series of clinical trials, including two phase 3 studies, a phase 1/2 study, and a phase 1b study that combined the drug with standard of care regimens

OBJECTIVES

- To describe the PK characteristics of DARA following its administration in combination therapies
- To evaluate the influence of covariates on the disposition of DARA in patients with MM who had received ≥1 prior line of therapy
- To compare the PK parameters of DARA in combination therapies with those of DARA monotherapy
- To investigate the relationship between DARA exposure and selected efficacy and safety endpoints

METHODS

Patients

- Data from 4 clinical trials of DARA in combination with standard of care therapies (GEN503 [ClinicalTrials.gov Identifier: NCT01615029], MMY1001 [NCT01998971], POLLUX [NCT02076009], and CASTOR [NCT02136134]) were pooled for PPK analyses
- DARA was administered intravenously in all studies
- Key eligibility criteria for these studies are listed in **Table 1**

Table 1. Eligibility Criteria for Clinical Studies

Study	Key eligibility criteria
GEN503	• Age ≥18 years with measurable, documented MM • ECOG performance status ≤2 ***Part 1:*** • Between 2 and 4 prior lines of therapy • Eligible for treatment with Rd ***Part 2:*** • ≥1 prior line of therapy and a PR or better with a prior treatment • Disease progression on the last line of treatment
MMY1001	• Age ≥18 years with measurable, documented MM • ECOG performance status ≤2 ***Vd and VTd arms:*** • Newly diagnosed with symptomatic disease fulfilling CRAB criteria ***VMP arm:*** • Newly diagnosed with symptomatic disease fulfilling CRAB criteria and ineligible for transplantation ***Pom-d arm:*** • ≥2 prior lines of therapy, including R and V • Disease refractory to last line of treatment
POLLUX	• Age ≥18 years with measurable, documented MM • ECOG performance status ≤2 • ≥1 prior line of therapy and a PR or better with a prior treatment • Disease progression on last line of treatment • Not intolerant or refractory to R
CASTOR	• Age ≥18 years with measurable, documented MM • ECOG performance status ≤2 • ≥1 prior line of therapy and a PR or better with a prior treatment • Disease progression on last line of treatment • Not intolerant or refractory to V or other PIs

MM, multiple myeloma; ECOG, Eastern Cooperative Oncology Group; Rd, lenalidomide and dexamethasone; PR, partial response; Vd, bortezomib and dexamethasone; VTd, bortezomib, thalidomide, and dexamethasone; CRAB, calcium elevated, renal failure, anemia, and bone lesions; VMP, bortezomib, melphalan, and prednisone; Pom-d, pomalidomide and dexamethasone; R, lenalidomide; V, bortezomib; PI, proteasome inhibitor.

Study Design

- **GEN503:** a phase 1/2, open-label, multicenter, dose-escalation (Part 1) and dose-extension (Part 2) study that evaluated the combination of DARA with lenalidomide and dexamethasone (Rd)
- **MMY1001:** a phase 1/2, open-label, multicenter, multi-arm study that evaluated DARA in combination with a variety of standard of care therapies, including bortezomib and dexamethasone (Vd); bortezomib, melphalan, and prednisone (VMP); bortezomib, thalidomide, and dexamethasone (VTd); and pomalidomide and dexamethasone (Pom-d)
- **POLLUX:** a phase 3, randomized, open-label, active-controlled, multicenter study that compared Rd to DARA plus Rd (DRd)
- **CASTOR:** a phase 3, randomized, open-label, active-controlled, multicenter study that compared Vd to DARA plus Vd (DVd)
- DARA doses and dose schedules for these studies are listed in **Table 2**

Table 2. Dosing Schedules for Clinical Studies

Study	DARA dose and schedule
GEN503	***Dose:*** DARA 2, 4, 8, and 16 mg/kg (Part 1) OR DARA 16 mg/kg (Part 2) ***Schedule[a]:*** qw for Cycles 1-2, q2w for Cycles 3-6, then q4w thereafter
MMY1001	***Dose:*** DARA 16 mg/kg ***Schedule:*** qw for 6 weeks, then q3w thereafter (Vd,[b] VTd,[b] and VMP[c] arms) OR qw for Cycles 1-2, q2w for Cycles 3-6, then q4w thereafter (Pom-d arm[d])
POLLUX	***Dose:*** DARA 16 mg/kg ***Schedule[a]:*** qw for Cycles 1-2, q2w for Cycles 3-6, then q4w thereafter
CASTOR	***Dose:*** DARA 16 mg/kg ***Schedule:*** qw for Cycles 1-3,[b] q3w on Cycles 4-8,[b] then q4w thereafter[a]

DARA, daratumumab; qw, weekly; q2w, every 2 weeks; q4w, every 4 weeks; q3w, every 3 weeks; Vd, bortezomib and dexamethasone; VTd, bortezomib, thalidomide, and dexamethasone; VMP, bortezomib, melphalan, and prednisone; Pom-d, pomalidomide and dexamethasone.
[a]28-day cycles.
[b]21-day cycles.
[c]42-day cycles.

PPK Analyses

- PPK analyses were performed on pooled datasets from the POLLUX, CASTOR, GEN503, and MMY1001 studies
- Serum DARA concentrations were evaluated using a validated enzyme-linked immunosorbent assay (lower limit of quantitation = 0.2 µg/mL; BioAnalytical Research Corporation Global Central Laboratory, Ghent, Belgium; Janssen Research & Development, LLC, Spring House, PA, USA)
- A PPK model, which was previously developed using data from DARA monotherapy studies,[12] was used to fit concentration-time data from the combination studies
  - PPK modeling was performed using NONMEM® 7.2 (ICON, Dublin, Ireland)
  - Data management, post-processing, and graphic analyses of NONMEM® runs were performed using the software package R (version 2.15.3)
- Subgroup analyses were performed to evaluate the relationship between patient/disease characteristics and DARA exposure
- Exposure-response analyses examined the relationship between DARA exposure (maximal pre-infusion [trough] concentration [$C_{pre\text{-}infusion,max}$]) and efficacy (progression-free survival, duration of response [DOR], overall response rate [ORR]) and safety (thrombocytopenia, anemia, neutropenia, lymphopenia, and infection) endpoints
  - Exposure-efficacy analyses were performed by study (ie, POLLUX, CASTOR, and MMY1001)
  - Exposure-safety analyses were performed according to combination regimen (ie, DRd [POLLUX and GEN503], DVd [CASTOR], and DARA plus Pom-d [MMY1001])
  - Because the majority of infusion-related reactions (IRRs) occurred during the first infusion, the predicted end-of-infusion concentration after the first infusion ($C_{max,1st}$) was used for exposure-safety analysis for IRRs

RESULTS

Patients

- The PPK dataset included 4,426 measurable PK samples for 694 patients, 684 of whom received DARA 16 mg/kg
  - 2.5% of the PK samples were below the limit of quantitation
- Descriptive statistics of continuous baseline patient and disease covariates are summarized in **Table 3**, and categorical covariates are summarized in **Table 4**

Table 3. Continuous Baseline Covariates[a]

	GEN503 N = 44	MMY1001 N = 128	POLLUX N = 282	CASTOR N = 240	Combined N = 694
Weight, kg					
N	44	127	282	240	693
Median (range)	81.5 (57.0-120.0)	77.3 (41.2-137.0)	73.0 (37.0-132.0)	77.8 (45.0-134.8)	75.9 (37.0-137.0)
Age, y					
N	44	128	282	240	694
Median (range)	61.0 (41.0-76.0)	65.0 (35.0-86.0)	65.0 (34.0-89.0)	63.0 (30.0-84.0)	64.0 (30.0-89.0)
Albumin, g/L					
N	44	128	282	240	694
Median (range)	38.0 (16.0-46.5)	36.0 (21.0-50.0)	38.0 (17.0-51.0)	39.0 (3.7-52.0)	38.0 (3.7-52.0)
Creatinine clearance, mL/min					
N	44	127	282	240	693
Median (range)	92.2 (42.6-224.7)	72.8 (26.1-176.4)	74.0 (24.0-266.5)	80.6 (21.1-208.7)	77.8 (21.1-266.5)
Total bilirubin, µmol/L					
N	44	128	280	240	692
Median (range)	5.7 (1.7-11.5)	7.1 (2.6-60.0)	7.8 (2.9-29.0)	8.1 (2.0-100.7)	7.7 (1.7-100.7)
Estimated glomerular filtration rate, mL/min/1.73 m²					
N	44	128	282	240	694
Median (range)	76.1 (27.9-278.2)	68.4 (8.5-139.1)	74.2 (24.0-231.8)	72.2 (18.3-165.4)	72.6 (8.5-278.2)

[a]Statistics were calculated before imputation of missing values.

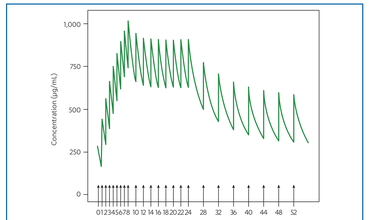
Table 4. Categorical Baseline Covariates

	GEN503 N = 44	MMY1001 N = 128	POLLUX N = 282	CASTOR N = 240	Combined N = 694
Dose group (N = 694), n (%)					
2 mg/kg	3 (6.8)	–	–	–	3 (0.4)
4 mg/kg	3 (6.8)	–	–	–	3 (0.4)
8 mg/kg	4 (9.1)	–	–	–	4 (0.6)
16 mg/kg	34 (77.3)	128 (100)	282 (100)	240 (100)	684 (98.6)
Sex (N = 694), n (%)					
Men	31 (70.5)	71 (55.5)	170 (60.3)	131 (54.6)	403 (58.1)
Women	13 (29.5)	57 (44.5)	112 (39.7)	109 (45.4)	291 (41.9)
Race (N = 694), n (%)					
White	42 (95.5)	95 (74.2)	203 (72.0)	201 (83.8)	541 (78.0)
African American	–	15 (11.7)	5 (1.8)	13 (5.4)	33 (4.8)
Hispanic/Latino	1 (2.3)	9 (7.0)	1 (0.4)	6 (2.5)	17 (2.4)
Asian	1 (2.3)	–	53 (18.8)	12 (5.0)	66 (9.5)
Pacific Islander	–	–	–	1 (0.4)	1 (0.1)
Native American	–	–	–	1 (0.4)	1 (0.1)
Other	–	9 (7.0)	20 (7.1)	6 (2.5)	35 (5.0)
Age (N = 694), n (%)					
<65 years	29 (65.9)	63 (49.2)	131 (46.5)	129 (53.8)	352 (50.7)
65 to <75 years	13 (29.5)	51 (39.8)	122 (43.3)	92 (38.3)	278 (40.1)
≥75 years	2 (4.5)	14 (10.9)	29 (10.3)	19 (7.9)	64 (9.2)
Creatinine clearance (N = 693), n (%)					
≥90 mL/min	23 (52.3)	42 (33.1)	97 (34.4)	89 (37.1)	251 (36.2)
≥60 to <90 mL/min	17 (38.6)	45 (35.4)	106 (37.6)	96 (40.0)	264 (38.1)
≥30 to <60 mL/min	4 (9.1)	38 (29.9)	77 (27.3)	47 (19.6)	166 (24.0)
≥15 to <30 mL/min	–	2 (1.6)	2 (0.7)	8 (3.3)	12 (1.7)
Hepatic dysfunction (N = 687), n (%)					
Normal	38 (86.4)	107 (83.6)	254 (92.4)	199 (83.3)	598 (87.2)
Mild	6 (13.6)	19 (14.8)	20 (7.3)	38 (15.9)	83 (12.1)
Moderate	–	2 (1.6)	1 (0.4)	1 (0.4)	4 (0.6)
Severe	–	–	–	1 (0.4)	1 (0.1)
ECOG status (N = 693), n (%)					
0	26 (59.1)	43 (33.6)	137 (48.6)	103 (43.1)	309 (44.6)
1	17 (38.6)	71 (55.5)	134 (47.5)	123 (51.5)	345 (49.8)
2	1 (2.3)	14 (10.9)	11 (3.9)	13 (5.4)	39 (5.6)
Lines of prior therapy (N = 665), n (%)					
1	15 (34.1)	3 (3.0)	146 (51.8)	118 (49.2)	282 (42.4)
2	14 (31.8)	20 (20.2)	85 (30.1)	64 (26.7)	183 (27.5)
3	12 (27.3)	25 (25.3)	37 (13.1)	37 (15.4)	111 (16.7)
≥3	3 (6.8)	51 (51.5)	14 (5.0)	21 (8.8)	89 (13.4)
Type of MM (N = 694), n (%)					
IgG	26 (59.1)	81 (63.3)	162 (57.4)	132 (55.0)	401 (57.8)
Non-IgG	18 (40.9)	47 (36.7)	120 (42.6)	108 (45.0)	293 (42.2)
Refractory status (N = 307), n (%)					
None	30 (68.2)	–	7 (8.9)	5 (5.9)	42 (13.7)
PI only	7 (15.9)	8 (8.1)	55 (69.6)	3 (3.5)	73 (23.8)
IMiD only	3 (6.8)	21 (21.2)	10 (12.7)	69 (81.2)	103 (33.6)
PI and IMiD	4 (9.1)	70 (70.7)	7 (8.9)	8 (9.4)	89 (29.0)
Combination therapy (N = 694), n (%)					
Rd	44 (100)	–	282 (100)	–	326 (47.0)
Vd	–	6 (4.7)	–	240 (100)	246 (35.4)
VTd	–	12 (9.4)	–	–	12 (1.7)
VMP	–	11 (8.6)	–	–	11 (1.6)
Pom-d	–	99 (77.3)	–	–	99 (14.3)

ECOG, Eastern Cooperative Oncology Group; MM, multiple myeloma; IgG, immunoglobulin G; PI, proteasome inhibitor; IMiD, immunomodulatory drug; Rd, lenalidomide and dexamethasone; Vd, bortezomib and dexamethasone; VTd, bortezomib, thalidomide, and dexamethasone; VMP, bortezomib, melphalan, and prednisone; Pom-d, pomalidomide and dexamethasone.

Effect of Patient and Disease Characteristics on DARA Exposure

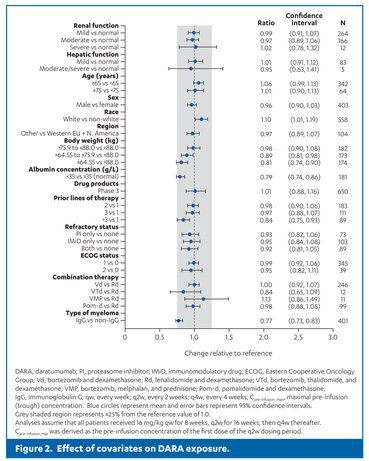
- PK of DARA were similar between monotherapy and combination therapy studies
  - Concentration-time data were adequately described by a 2-compartment PPK model with parallel linear and non-linear Michaelis-Menten eliminations
  - The model-derived half-life associated with linear elimination was 23.3 ± 11.8 days in the combination studies and 18 ± 9 days in the monotherapy studies
  - As in the monotherapy studies, steady state was reached at approximately 5 months into the every 4 weeks dosing period (**Figure 1**)



PK, pharmacokinetic; DARA, daratumumab; qw, weekly; q2w, every 2 weeks; q4w, every 4 weeks.
Black arrows represent dosing events.

Figure 1. Typical PK profile of DARA under the recommended dose and dosing schedule of POLLUX (16 mg/kg qw for 8 weeks, q2w for 16 weeks, and q4w thereafter).

- Similar to findings from the monotherapy studies, the concentration of DARA was statistically lower in patients with an abnormal albumin level and immunoglobulin G myeloma; however, the magnitude of the effects was small (<25%) and not clinically relevant
- The effects of other intrinsic and extrinsic factors (age, sex, race, region, renal and hepatic impairment, type of combination therapy, Eastern Cooperative Oncology Group status, refractory status, and number of prior lines of therapy) on DARA exposure were not clinically important (**Figure 2**)
  - These data were similar (within <25%) to the results of subgroup analyses of data from the monotherapy studies
- Consistent with results from the monotherapy studies, clearance and volume of distribution of DARA increased with increasing body weight (**Figure 2**); however, DARA exposure was consistent across a range of body weights when administered on a mg/kg basis

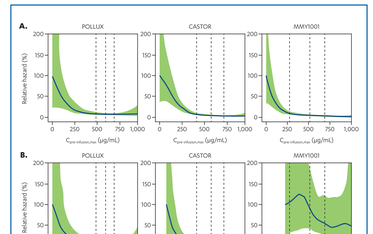


DARA, daratumumab; PI, proteasome inhibitor; IMiD, immunomodulatory drug; ECOG, Eastern Cooperative Oncology Group; Vd, bortezomib and dexamethasone; Rd, lenalidomide and dexamethasone; VTd, bortezomib, thalidomide, and dexamethasone; VMP, bortezomib, melphalan, and prednisone; Pom-d, pomalidomide and dexamethasone; IgG, immunoglobulin G; qw, every week; q2w, every 2 weeks; q4w, every 4 weeks; $C_{pre\text{-}infusion,max}$, maximal pre-infusion (trough) concentration. Blue circles represent mean and error bars represent 95% confidence intervals.
Grey shaded region represents ±25% from the reference value of 1.0.
Analyses assume that all patients received 16 mg/kg qw for 8 weeks, q2w for 16 weeks, then q4w thereafter.
$C_{pre\text{-}infusion,max}$ was derived as the pre-infusion concentration of the first dose of the q2w dosing period.

Figure 2. Effect of covariates on DARA exposure.

Exposure-efficacy Analysis

- The risk of disease progression and death decreased with increasing DARA exposure, based on data from the POLLUX, CASTOR, and MMY1001 studies
  - The rate of decrease of relative hazard appeared to slow down when $C_{pre\text{-}infusion,max}$ was >250 µg/mL, suggesting that there would be limited additional benefit to DARA $C_{pre\text{-}infusion,max}$ >250 µg/mL (**Figure 3A**)
    - >90% of patients in POLLUX and CASTOR and >80% of patients in MMY1001 had $C_{pre\text{-}infusion,max}$ >250 µg/mL
    - These data are consistent with the concentration at which 90% maximal effect on ORR (274 µg/mL) was observed in the monotherapy studies
- Similarly, risk of disease progression and death in responders decreased with DARA exposure based on data from the POLLUX and CASTOR studies
  - When $C_{pre\text{-}infusion,max}$ was >250 µg/mL, the rate of decrease of relative hazard appeared to slow down, suggesting a limited benefit to DARA concentrations above 250 µg/mL (**Figure 3B**)
    - >90% of patients in POLLUX and CASTOR had $C_{pre\text{-}infusion,max}$ >250 µg/mL
    - No relationship was observed between $C_{pre\text{-}infusion,max}$ and DOR in MMY1001; this may be due to the smaller overall patient numbers or the narrower range of concentrations observed in responders

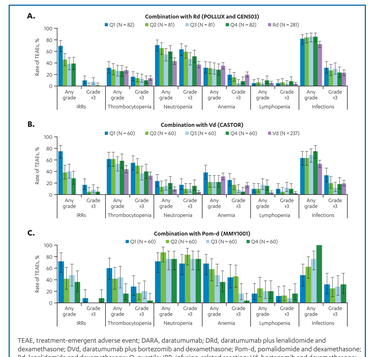


The relationships between exposure and PFS (A) and DOR (B) are shown for the POLLUX, CASTOR, and MMY1001 studies.
$C_{pre\text{-}infusion,max}$, maximal pre-infusion (trough) concentration; PFS, progression-free survival; DOR, duration of response.
Solid blue lines represent point estimates. Green shaded regions in panels A and B represent the 95% confidence interval. Black dotted lines separate the quartiles of $C_{pre\text{-}infusion,max}$.

Figure 3. Relative hazard of disease progression and death at different $C_{pre\text{-}infusion,max}$.

Exposure-safety Analysis

- No relationship between $C_{max,1st}$ and IRRs was observed within the studied DARA concentration range, regardless of whether DARA was combined with Rd, Vd, or Pom-d (**Figure 4**)
- Similarly, no relationship was apparent between $C_{pre\text{-}infusion,max}$ and thrombocytopenia, anemia, neutropenia, lymphopenia, or infection within the studied concentration range, across all combination regimens (**Figure 4**)
  - A trend toward an increased rate of infections of any grade was observed, but did not reach statistical significance; this trend was not mirrored in the rate of grade ≥3 infections
  - These analyses are consistent with the results from similar exposure-safety analyses of data from monotherapy studies



TEAE, treatment-emergent adverse event; DARA, daratumumab; DRd, daratumumab plus lenalidomide and dexamethasone; DVd, daratumumab plus bortezomib and dexamethasone; Pom-d, pomalidomide and dexamethasone; Rd, lenalidomide and dexamethasone; Q, quartile; IRR, infusion-related reaction; Vd, bortezomib and dexamethasone; CI, confidence interval; $C_{max,1st}$, end-of-infusion concentration after the first infusion; $C_{pre\text{-}infusion,max}$, maximal pre-infusion (trough) concentration.
Error bars represent 95% CIs.
End-of-infusion concentration after $C_{max,1st}$ was used as the exposure measure for analyses of IRRs. $C_{pre\text{-}infusion,max}$ was used as the exposure measure for all other analyses.
The quartiles for $C_{max,1st}$ were: 1st quartile (<247 µg/mL), 2nd quartile (247-303 µg/mL), 3rd quartile (303-347 µg/mL), and 4th quartile (347-464 µg/mL).
The quartiles for $C_{pre\text{-}infusion,max}$ are: 1st quartile (<740 µg/mL), 2nd quartile (740-870 µg/mL), 3rd quartile (870-1,008 µg/mL), and 4th quartile (1,008-1,470 µg/mL).

Figure 4. Rate of TEAEs by DARA-exposure quartiles for DRd (A), DVd (B), and DARA plus Pom-d (C).

CONCLUSIONS

- **The PK of DARA were similar between monotherapy and combination therapy studies**
- **No clinically relevant demographic or clinical characteristics were identified and, thus, no dose adjustments based on patient or disease characteristics are recommended**
- **This exposure-response analysis supports the use of the recommended dose and dose schedule of DARA in combination therapy**
  - **Maximum clinical benefit was maintained throughout dosing**
  - **No relationship between exposure and safety signals was identified**

REFERENCES

1. Kurcda J, et al. *Expert Rev Anticancer Ther.* 2013;13(9):1081-1088.
2. Laubach JP, et al. *Expert Opin Investig Drugs.* 2014;23(4):445-452.
3. Kumar SK, et al. *Leukemia.* 2012;26(1):149-157.
4. Usmani SZ, et al. *Oncologist.* 2016;21(11):1355-1361.
5. de Weers M, et al. *J Immunol.* 2011;186(3):1840-1848.
6. Krejcik J, et al. *Blood.* 2016;128(3):384-394.
7. Overdijk MB, et al. *J Immunol.* 2016;197(3):807-813.
8. van de Donk NW, et al. *Immunol Rev.* 2016;270(1):95-112.
9. Lokhorst HM, et al. *N Engl J Med.* 2015;373(13):1207-1219.
10. Lonial S, et al. *Lancet.* 2016;387(10027):1551-1560.
11. Usmani SZ, et al. *Blood.* 2016;128(1):37-44.
12. Yan X, et al. *Blood.* 2015;126(23):4222.
13. Dimopoulos MA, et al. *N Engl J Med.* 2016;375(14):1319-1331.
14. Palumbo A, et al. *N Engl J Med.* 2016;375(8):754-766.
15. Usmani SZ, et al. *Blood.* 2016;128(1):37-44.
16. DARZALEX™ (daratumumab) injection, for intravenous use [package insert]. Horsham, PA: Janssen Biotech, Inc.; 2016.

ACKNOWLEDGMENTS

The authors thank the patients participating in these studies and their families, as well as the global network of investigators, research nurses, study coordinators, and operations staff. These studies (POLLUX [ClinicalTrials.gov Identifier: NCT02076009], CASTOR [NCT02136134], GEN503 [NCT01615029], and MMY1001 [NCT01998971]) were sponsored by Janssen Research & Development, LLC. Editorial and medical writing support were provided by Erica Chevalier-Larsen, PhD, of MedErgy, and were funded by Janssen Global Services, LLC.

DISCLOSURES

XSX, MQ, JS, NZK, XY, LZ, and PLC are employees of Janssen Research & Development, LLC. XSX, JS, XY, LZ, and PLC own stock in Johnson & Johnson. SL is an employee of Pharmax Research, Inc., has consulted for Janssen Research & Development, LLC, and owns stock in Johnson & Johnson. MAD reports honoraria from and participation in advisory committees for Celgene, Janssen, Novartis, and Amgen. PS reports honoraria from and consultancy for Amgen, Celgene, Janssen, Karyopharm, and Takeda; and research funding from Amgen, Celgene, Janssen, and Karyopharm. PJH reports honoraria from and participation in advisory committees for Celgene and Janssen. AB reports honoraria from and participation in advisory committees for Amgen, Celgene, and Takeda. MC reports participation in a speakers bureau for Janssen. DG reports consultancy for Janssen, BMS, Celgene, and Amgen. EM is an employee of Oregon Health and Science University. SI reports research funding from Janssen, Takeda, Celgene, BMS, Chugai Pharmaceuticals, Kyowa Hakko Kirin Co., Eli Lilly Japan, Novartis, Sanofi, Bayer Yakuhin, Toyama Chemical Co., Teijin Pharma, and Astellas Pharma; and honoraria from Janssen, Takeda, Celgene, BMS, and Ono Pharmaceuticals. ML and CM report no conflicts.

An electronic version of the poster can be viewed by scanning the QR code. The QR code is intended to provide scientific information for individual reference. The PDF should not be altered or reproduced in any way.
http://jjd_ash.scientificpresentations.org/Xu_JJD61074.pdf

POSTER PRESENTED AT THE 58TH AMERICAN SOCIETY OF HEMATOLOGY (ASH) ANNUAL MEETING & EXPOSITION; DECEMBER 3-6, 2016; SAN DIEGO, CALIFORNIA.