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Plerixafor Plus Granulocyte Colony-Stimulating Factor for Patients with Non-Hodgkin Lymphoma and Multiple Myeloma: Long-Term Follow-Up Report

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ABSTRACT

The purpose of this report is to analyze long-term clinical outcomes of patients exposed to plerixafor plus granulocyte colony-stimulating factor (G-CSF) for stem cell mobilization. This was a study of patients with non-Hodgkin lymphoma (NHL; n = 167) and multiple myeloma (MM; n = 163) who were enrolled in the long-term follow-up of 2 pivotal phase III studies (NCT00741325 and NCT00741780) of 240 μg/kg plerixafor plus 10 μg/kg G-CSF, or placebo plus 10 μg/kg G-CSF to mobilize and collect CD34+ cells for autologous hematopoietic stem cell transplantation. Overall survival (OS) and progression-free survival (PFS) were evaluated over a 5-year period following the first dose of plerixafor or placebo. The probability of OS was not significantly different in patients with NHL or MM treated with plerixafor or placebo (NHL: 64%; 95% confidence interval [CI], 56% to 71% versus 56%; 95 CI, 44% to 67%, respectively; MM: 64%; 95 CI, 54% to 72% versus 64%; 95 CI, 53% to 73%, respectively). In addition, there was no statistically significant difference in the probability of PFS over 5 years between treatment groups in patients with NHL (50%; 95 CI, 44% to 67% for plerixafor versus 43%; 95 CI, 31% to 54% for placebo) or those with MM (17%; 95 CI, 10% to 24% for plerixafor versus 30%; 95 CI, 11% to 40% for placebo). In this long-term follow-up study, the addition of plerixafor to G-CSF for stem cell mobilization did not affect 5-year survival in patients with NHL or patients with MM.

INTRODUCTION

High-dose chemotherapy combined with autologous hematopoietic stem cell transplantation (auto-HSCT) is the standard of care for patients with relapsed or chemosensitive non-Hodgkin lymphoma (NHL) and multiple myeloma (MM) [1–3]. Auto-HSCT improves hematologic recovery in patients by reconstituting hematopoiesis following high-dose chemotherapy [3]. In patients with relapsed or chemosensitive NHL, high-dose chemotherapy with auto-HSCT has been shown to increase disease-free survival [4], whereas in MM, a combination of high-dose chemotherapy with auto-HSCT improves progression-free survival (PFS) and overall survival (OS) [5,6]. The minimum number of cells generally acceptable for transplantation is 2 × 10⁶ CD34+ cells/kg [7]. Transplanting fewer than this number of cells may result in delayed engraftment of both platelets and neutrophils [8]. The target number of cells for a single transplant was defined by

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Weaver et al. [9] as ≥5 × 10⁶ CD34⁺ cells/kg, which is important for short-term outcomes, resulting in earlier and more consistent neutrophil and especially platelet engraftment compared with transplants with lower cell doses [10]. In some studies, transplant doses of ≥5 × 10⁶ CD34⁺ cells/kg have been associated with longer disease-free survival and OS compared with lower transplant doses [11-13]. One recent retrospective analysis comparing patients with NHL mobilized with chemotherapy plus granulocyte colony-stimulating factor (G-CSF) (median, 12.0 × 10⁶ CD34⁺ cells/kg) versus G-CSF alone (median, 5.0 × 10⁶ CD34⁺ cells/kg) found no difference between the groups in either event-free survival or OS [14]. It should be noted that for patients with MM, subsequent or tandem auto-HSCT can be considered as well [15].

Obtaining a sufficient quantity of cells for auto-HSCT is difficult in approximately 20% to 25% of patients [3,16-18]. These include patients with NHL, elderly patients [19], heavily pretreated patients [20-22], and patients with MM who previously received multiple cycles of lenalidomide or underwent auto-HSCT [23].

Employing, an effective stem cell mobilization regimen plays a critical role in optimizing engraftment and outcomes in patients with NHL and MM. Until recently, there were 2 main approaches to stem cell mobilization that involved the use of growth factors, such as G-CSF alone, or in conjunction with chemotherapy [24]. The administration of chemotherapy before the use of G-CSF produces a higher yield of stem cells for autologous transplantation, but this is not effective for all patients [24].

Plerixafor, a CXCR4 receptor antagonist, is used in combination with G-CSF to mobilize hematopoietic stem cells into the peripheral blood (PB) for collection and subsequent auto-HSCT in patients with lymphoma or MM [25,26]. Plerixafor selectively and reversibly antagonizes the CXCR4 chemokine receptor and blocks binding of stromal cell-derived factor-1α (SDF-1α) [27]. The interruption of the CXCR4/SDF-1α interaction provides a mechanism for mobilization of CD34⁺ stem cells from the bone marrow to the PB, where they can be collected for auto-HSCT. Plerixafor provides another option for transplantation, and G-CSF plus plerixafor augments the mobilization of CD34⁺ cells, particularly in patients who are considered poor mobilizers [28-30]. The stem cells mobilized in apheresis products by the combination of G-CSF plus plerixafor have been shown to differ from those mobilized by G-CSF alone, with a higher proportion of cells in the growth phase, higher numbers of B and T lymphocytes, natural killer cells, dendritic cells, and primitive CD34⁺ cells [24,31-35].

Two pivotal multicenter, randomized, double-blind, placebo-controlled phase III studies have evaluated the efficacy and safety of plerixafor plus G-CSF versus placebo plus G-CSF in mobilizing and collecting hematopoietic stem cells. [36,37]. These trials demonstrated that plerixafor plus G-CSF can generate optimal numbers of cells for auto-HSCT in patients with NHL or MM.

Findings from the NHL study (Study 3101; NCT00103610) showed that compared with placebo plus G-CSF, plerixafor plus G-CSF significantly increased the percentage of patients in whom the target stem cell collection (≥5 × 10⁶ CD34⁺ cells/kg) was achieved within 4 apheresis days (20% versus 5%) [36].

Findings from the MM study (Study 3102; NCT00103662) also showed that plerixafor plus G-CSF significantly increased the percentage of patients in whom the target stem cell collection (≥5 × 10⁶ CD34⁺ cells/kg to facilitate tandem transplantation) was achieved within 2 apheresis days, compared with placebo plus G-CSF (72% versus 34%) [37]. In both studies, auto-HSCT after mobilization with plerixafor and placebo resulted in successful engraftment of neutrophils and platelets. Durability of grafts was similar for plerixafor and placebo through 12 months of follow-up, and both regimens were associated with similar survival rates at 12 months post-transplantation [36,37].

To examine clinical outcomes beyond 1 year, we undertook a long-term, observational study of patients with NHL or MM enrolled in each of the phase III pivotal studies. We assessed OS and PFS over a 5-year period following the first dose of study drug (ie, plerixafor or placebo) administered in Studies 3101 and 3102. Furthermore, because the use of autologous PB stem cells for transplantation could be associated with the risk of contamination of the graft with tumor cells [40], the assessment of OS and PFS may serve as a surrogate for determining the risk of plerixafor mobilizing malignant cells from the bone marrow during hematopoietic stem cell mobilization. Very limited data from small numbers of patients indicate that tumor cell trafficking to the peripheral blood is not significantly increased after mobilization with G-CSF plus plerixafor compared with G-CSF alone in patients with MM or NHL [41,42]. It also should be noted that the clinical significance of tumor cell mobilization of myeloma tumor cell contamination of mobilized apheresis products on long-term outcomes is unclear.

METHODS

Study Design

This study was a long-term, observational follow-up (hereafter referred to as the “LTF study”) to the 2 phase III multicenter, randomized, double-blind, placebo-controlled, comparative trials of plerixafor plus G-CSF (hereafter referred to as “plerixafor”) versus placebo plus G-CSF (hereafter referred to as “placebo”) to mobilize and collect CD34⁺ cells for auto-HSCT in patients with NHL (Study 3101) [36] and patients with MM (Study 3102) [37]. Patients were eligible if they had previously received at least 1 dose of study treatment (placebo or plerixafor) in either study and had a signed informed consent for follow-up data collection. Patients who failed to mobilize or who did not achieve at least 2 × 10⁶ CD34⁺ cells/kg within ≤4 days of apheresis after treatment with either placebo or plerixafor were allowed to enter a “rescue” procedure. Those patients received open-label plerixafor with the aim of collecting a transplantable dose of CD34⁺ cells, and were assigned to the plerixafor treatment group for analysis.

In both studies, data were collected for OS evaluation at 100 days, 6 months, and 12 months after auto-HSCT. The initial objective of the LTF study was to assess OS over a 5-year period following administration of the first dose of plerixafor or placebo in Studies 3101 and 3102, and was later expanded to include PFS.

These studies were registered at www.ClinicalTrials.gov (NCT00714325 for 3101-LTF and NCT00741325 for 3102-LTF) and conducted in accordance with International Conference on Harmonisation Good Clinical Practice, the principles specified in the Declaration of Helsinki and its amendments, and all applicable national and international laws. All patients who had consented and received at least 1 dose of plerixafor or placebo in Studies 3101 and 3102 were eligible for inclusion in the LTF study. After consenting to long-term data collection, patients were free to withdraw consent or discontinue participation at any time at the discretion of the investigator or sponsor. There were no formal exclusion criteria.

Endpoints

The primary endpoint analysis was OS and PFS. This analysis included all patients enrolled in Studies 3101 and 3102, as well as patients enrolled in the LTF studies. Data for survival and disease state were collected up to 5 years after the first study drug administration in Studies 3101 and 3102. Data from patients who did not enroll for the LTF study were included up to either their last reported follow-up date during the original 3101 and 3102 study periods, or until the date of their LTF study registration form.
OS was defined as the time from the date of first study drug exposure (placebo or plerixafor) until the date of death due to any cause. PFS was defined as the time from the date of first study drug exposure until the date of reported disease progression, disease relapse, or death due to any cause, whichever occurred first. If a disease progression/relapse or death event occurred but the date of the event was missing, the date of follow-up contact at which the event was reported was used as the best approximation.

PFS was determined using the Revised Response Criteria for Malignant Lymphoma [43] and the International Uniform Response Criteria for Multiple Myeloma [44]. Time points and testing requirements were not specified for assessment of PFS, and data were not collected in the case report form to document the determination of PFS.

### Long-Term Follow-Up Schedule

The LTF schedule was determined by whether or not the patient underwent auto-HSCT during Study 3101 or 3102. For patients who underwent auto-HSCT (including those who withdrew or were lost to follow-up), follow-up began at 18 months post auto-HSCT or at LTF study entry (ie, signed informed consent) and occurred every 6 months (±3 months) for 5 years after the first dose of study treatment. For patients who did not undergo auto-HSCT in Study 3101 or 3102, follow-up occurred every 6 months (±3 months) for a total follow-up period of 5 years following the first dose of study treatment (placebo or plerixafor).

### Data Collection

For this observational study, data for disease progression/relapse and death were collected from the original data for Studies 3101 and 3102, LTF study registration forms, and the LTF study itself. Data from patients who did not enroll in the LTF study were limited to that collected during Study 3101 or 3102 and/or the LTF study registration form.

### Assessments

The principal population assessed for efficacy was the primary intent-to-treat (ITT) population, which comprised all randomized patients. Data analyzed for the ITT population were based on the actual randomization assignment. The Per Protocol population consisted of all ITT patients who received any fraction of study treatment (plerixafor or placebo), completed the apheresis period, and did not have any major protocol deviations that significantly impacted the assessment of efficacy, and included all patients who had received at least 1 dose of study drug or placebo, as well as those patients who failed stem cell collection on the study and had elected to enter the "rescue protocol". Death and disease progression/relapse status were captured at study entry and at each follow-up contact, which occurred every 6 months until 5 years from the date of first dose. In addition, the investigators and treating physicians were encouraged to record any additional treatments (eg, transplantation, chemotherapy, or radiotherapy) received since the last study contact.

### Statistical Analysis

OS, PFS and the composite triple endpoint were estimated using the Kaplan-Meier (KM) method. The 25th, 50th (median), and 75th OS and PFS percentiles (along with 95% confidence intervals [CIs] for these quartiles, if estimable) were determined. The OS and PFS probabilities along with 95% CIs at 12, 24, 36, 48, and 60 months were estimated from the KM method. The log-rank test and the Wilcoxon test (Breslow procedure) were used to compare treatment groups (plerixafor versus placebo). The statistical analysis system was used to perform all analyses. When inferential statistics were performed, a P value of <.05 was considered statistically significant. The P values of all tests are reported without any correction for the multiplicity of tests performed.

### RESULTS

#### Patient Disposition and Baseline Disease Characteristics

The disposition of patients with NHL and MM included in the LTF study is described in Figure 1 and Figure 2, respectively. Patients from Studies 3101 and 3102 were included in the LTF study if they enrolled for the study, or for some analyses (OS and PFS), data were included from patients who had died before study initiation.

Of the original 289 patients with NHL from the Study 3101, 167 (58%) were enrolled in the LTF study. Of these 167 patients, 44 (26%) had received placebo and 123 (74%) had received plerixafor either initially or following rescue. Of the 122 patients (42%) who did not enter the LTF study, 64 (26 in the placebo group, 38 in the plerixafor group), had died before initiation of the LTF study. 31 (22 in the plerixafor group) had been lost to follow-up, 21 (14 in the plerixafor group) refused to participate, and 6 (4 in the plerixafor group) from sites that declined to participate in the study.

Of the 294 patients with MM in Study 3102, 163 (55%) enrolled in the LTF study. Of these 163 patients, 72 received placebo and 91 received plerixafor either as allocated treatment (n = 85) or as a rescue (n = 6). The remaining 131 patients (45%) who did not enroll in the LTF study comprised 40 who had died during and after Study 3102, 45 who were lost to follow-up during or after Study 3102, 27 from sites that declined to participate in the study, and 19 who refused to participate.

Demographic data and baseline disease characteristics for patients with NHL and MM who enrolled in the LTF study, stratified by treatment group, are summarized in Tables 1 and 2, respectively. Demographic data and baseline data for patients with NHL and MM who enrolled in the original Studies 3101 and 3102 are summarized in Supplementary Table S1 and Supplementary Table S2, respectively.

The majority of patients who enrolled in the LTF study were male (NHL, 68%; MM, 72%). The mean patient age was 57.4 years (standard deviation [SD], 9.9 years) in the NHL patient population and 59.4 years (SD, 8.6 years) in the MM group. Demographics were similar when stratified by treatment group and rescue for both the NHL and MM groups. The most common baseline disease characteristics in the NHL population were Stage III disease (32%) and Stage IV disease (30%) in the placebo group, and Stage IV disease (44%) in the plerixafor group. The most common remission status at baseline was first complete remission for both placebo-treated (43%) and plerixafor-treated (31%) patients. The majority of patients with MM who enrolled in the LTF study had Stage III disease in both the plerixafor (55%) and placebo (56%) groups, and most had a remission status of first partial remission at baseline (82%).

#### Overall Survival

All 289 patients with NHL from Study 3101 and 294 patients with MM from Study 3102 contributed to the OS and PFS analyses. Patients who did not enroll in the LTF study were censored on either the date of their LTF study registration form or by their last reported follow-up date within the Study 3101 or 3102 study periods.

Based on KM analysis, median OS was not achieved within 5 years of follow-up for either the placebo group or the plerixafor group (Figure 3A). In the NHL population, 93 of the 289 patients (32%) died, including 32 of 88 (36%) in the placebo group and 61 of 201 (30%) in the plerixafor group. There was no statistically significant difference in OS over 5 years between the placebo-treated and plerixafor-treated patients (log-rank test, P = .273; Wilcoxon test, P = .308). Findings were similar for rescue and nonrescue patients (data not presented). The estimated 12, 24, 36, 48, and 60 month OS probabilities for the placebo and plerixafor group are shown in Table 3.
In the MM population, 76 of 294 (26%) patients died, 35 of 142 (25%) patients in the placebo group and 41 of 152 (27%) patients in the plerixafor group. Median OS was not reached for either the placebo or plerixafor groups within 5 years of follow-up (Figure 3B) and there was no statistically significant difference in OS over 5 years between the placebo-treated and plerixafor-treated patients (log-rank test, \( P = .936 \); Wilcoxon test, \( P = .970 \)). The estimated 12, 24, 36, 48, and 60 month OS probabilities for each treatment group are listed in Table 3.

### Progression Free Survival

Overall, 129 of 289 patients (45%) with NHL reported a PFS event, including 43 of 88 (49%) in the placebo group and 86 of 201 (43%) in the plerixafor group. Median PFS was reached in the placebo group at 39 months, but was not reached in the plerixafor group within 5 years of follow-up. However, the assessment of PFS over 5 years (Figure 4A) showed no significant difference between treatment groups (log-rank test, \( P = .343 \); Wilcoxon test, \( P = .396 \)). The estimated 12, 24, 36, 48, and 60 month PFS probabilities for the placebo and plerixafor groups are summarized in Table 3. One case of acute myeloid leukemia (AML) was observed in an NHL patient who had received plerixafor plus G-CSF but was considered by the treating physician to be unrelated to plerixafor.

In the MM patient population, 172 of 294 patients (59%) reported a PFS event, 70 of 142 patients (49%) in the placebo group and 102 of 152 patients (67%) in the plerixafor group. Median PFS was reached at 34 months for the placebo group and at 26 months for the plerixafor group. A trend toward a shorter PFS for patients treated with plerixafor than for those receiving placebo (Figure 4B) was observed (log-rank test, \( P = .061 \); Wilcoxon test, \( P = .138 \)). Table 3 shows the estimated 12, 24, 36, 48, and 60 month PFS probabilities for each treatment group. There were no cases of secondary MDS or AML among patients with MM who had received plerixafor plus G-CSF.
Composite Endpoint Analysis

During the period covering the end of recruitment to the phase III studies and the observational phase data analysis, several large studies were conducted to evaluate the possible benefit of adding some form of maintenance or adjuvant therapy post transplantation to attempt to improve the overall outcome for patients with MM. To evaluate this effect on Study 3101, Study 3102, and the LTF study, a composite triple-endpoint analysis was undertaken (Supplementary Figures S1 and S2).

Overall, 166 of the 289 patients (57%) with NHL had a triple-endpoint event, including 53 of 88 (60%) in the placebo group and 113 of 201 (56%) in the plerixafor group. The median triple endpoint was reached at 28 months in the placebo group and at 24 months in the plerixafor group. No significant difference between placebo and plerixafor was noted (log-rank test, \( P = .702 \); Wilcoxon test, \( P = .836 \)). Table 3 summarizes the estimated 12, 24, 36, 48, and 60 month triple-endpoint survival probabilities for the plerixafor and placebo treatment groups.

In the MM patient population, 207 of 294 patients (70%) had a triple-endpoint event, including 91 of 142 (64%) in the placebo group and 116 of 152 (76%) in the plerixafor group. Based on KM analysis, the median triple endpoint was reached at 20 months for both treatment groups. There was no significant difference between placebo and plerixafor for the triple endpoint (log-rank test, \( P = .752 \), Wilcoxon test, \( P = .944 \)). The estimated 12, 24, 36, 48, and 60 month triple-endpoint survival probabilities for both treatment groups are presented in Table 3.

DISCUSSION

This is the first long-term observational study to assess OS and PFS in patients with NHL and MM for a period of 5 years following the first dose of study treatment (plerixafor or placebo) in Studies 3101 and 3102. The results from this
LTF study suggest that the addition of plerixafor to G-CSF did not have a detrimental effect on long-term survival.

The main limitation of this study is its observational nature. In addition, only 58% of patients with NHL and 55% of patients with MM enrolled for the LTF study. However, because data from patients who had previously died were included in the analysis, 80% of the patients with NHL and 69% of those with MM contributed to the OS and PFS data. The finding of PFS data trending in opposite directions for patients with NHL and MM was difficult to interpret. The progression analyses contained limitations associated with data collection and reporting during the study period, determination of true progression and the progression timeline difficult to ascertain. There was also an imbalance in patients who achieved complete remission following ablative therapy in Studies 3101 and 3102. Progression data also may have been confounded by inconsistent reporting of additional treatments used for maintenance or disease recurrence to improve progression-free intervals [45,46]. Nonetheless, findings from the composite triple- endpoint analysis, which took into account any reported potential maintenance medication, showed no statistically significant difference in the incidence of triple- endpoint events between the plerixafor and placebo groups. A further limitation of the study is that the sample size provided limited power and could detect only a relatively large difference between the 2 groups.

Evaluating differences in OS and PFS between treatment groups in the present investigation also served as a surrogate for the risk of potential detrimental outcome of the mobilization of malignant cells from the bone marrow during hematopoietic stem cell mobilization for auto-HSCT. Our finding of no statistically significant difference in OS or PFS following treatment with plerixafor plus G-CSF versus placebo and G-CSF would seem to suggest that plerixafor does not exert long-term deleterious effects as a result of tumor cell mobilization. This observation is consistent with findings from previous investigations demonstrating that plerixafor does not contribute to tumor cell mobilization any more than G-CSF alone does. A registry study is also underway in collaboration with the European Group for Blood and Marrow Transplant to compare the outcomes of patients undergoing transplantation with plerixafor-mobilized and non-plerixafor-mobilized hematopoietic stem cells.

Other studies examining the effect of stem cell mobilization agents for auto-HSCT on long-term disease outcomes in patients with NHL and patients with MM are limited, and the paucity of long-term follow-up studies addressing OS and PFS in patients with NHL and MM following stem cell mobilization and auto-HSCT highlights the importance of the present study. Although the scarcity of published data restricts direct comparison of this study with others, a retrospective analysis that evaluated long-term outcomes of plerixafor plus

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographic Data and Baseline Characteristics of Patients with NHL Enrolled in the LTF Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable/Statistic</td>
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<tr>
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<tr>
<td>Age, yr</td>
<td>Mean (SD)</td>
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<tr>
<td>Disease stage at baseline, n (%)</td>
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<td>II</td>
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<td></td>
<td>III</td>
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<tr>
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<tr>
<td>Remission status at baseline, n (%)</td>
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<td></td>
<td>Relapse/second PR</td>
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| SD indicates standard deviation; CR, complete remission; PR, partial remission. |
* The plerixafor group includes 1 patient who was initially randomized to placebo and subsequently underwent rescue with plerixafor.
† Baseline represents the point immediately before study drug administration.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Demographic Data and Baseline Characteristics of Patients with MM Enrolled in the LTF Study</th>
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<td>Age, yr</td>
<td>Mean (SD)</td>
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<tr>
<td>Disease stage at baseline, n (%)</td>
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</table>

* The plerixafor group includes 1 patient who was initially randomized to placebo and subsequently underwent rescue with plerixafor.
† Baseline represents the point immediately before study drug was administered.
G-CSF and G-CSF alone in patients with MM and lymphoma (NHL and Hodgkin lymphoma), found a median PFS in the plerixafor plus G-CSF group of 22.5 months in patients with MM, comparable to our present finding, and a median OS of 40 months. However, the authors did not compare OS and PFS between plerixafor plus G-CSF and G-CSF alone because of the study’s retrospective nature and small sample size [15]. Within the wider context of other studies examining long-term outcomes following auto-HSCT, our findings, which show that more than one-half of patients remained alive 5 years after transplantation, echo those of several previous studies—that is, auto-HSCT is associated with extended OS [5,6,46].

In conclusion, although this follow-up study is limited by its observational design, our results suggest that the use of plerixafor plus G-CSF does not have a negative outcome on OS and PFS at 5 years in these patients with NHL or MM.

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### Table 3

KM OS, PFS, and Triple-Endpoint Survival Probabilities at 12, 24, 36, 48, and 60 Months

<table>
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<tr>
<th>Parameter</th>
<th>NHL Patients</th>
<th>MM Patients</th>
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<td></td>
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<td>KM OS probability, % (95% CI)</td>
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<td>85 (76-91)</td>
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<td>24 mo</td>
<td>72 (62-81)</td>
<td>75 (68-80)</td>
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<td>36 mo</td>
<td>66 (55-76)</td>
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<td>48 mo</td>
<td>54 (42-65)</td>
<td>58 (50-65)</td>
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<tr>
<td>60 mo</td>
<td>48 (36-59)</td>
<td>54 (46-61)</td>
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<td>KM PFS probability, % (95% CI)</td>
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<td>12 mo</td>
<td>72 (62-81)</td>
<td>75 (68-80)</td>
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<td>24 mo</td>
<td>66 (55-76)</td>
<td>66 (58-72)</td>
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<td>36 mo</td>
<td>54 (42-65)</td>
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<td>48 mo</td>
<td>48 (36-59)</td>
<td>54 (46-61)</td>
</tr>
<tr>
<td>60 mo</td>
<td>43 (31-54)</td>
<td>50 (42-58)</td>
</tr>
<tr>
<td>Triple-endpoint survival probability, % (95% CI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 mo</td>
<td>60 (49-69)</td>
<td>60 (52-66)</td>
</tr>
<tr>
<td>24 mo</td>
<td>55 (44-65)</td>
<td>49 (42-56)</td>
</tr>
<tr>
<td>36 mo</td>
<td>44 (33-55)</td>
<td>41 (34-49)</td>
</tr>
<tr>
<td>48 mo</td>
<td>38 (27-49)</td>
<td>40 (32-47)</td>
</tr>
<tr>
<td>60 mo</td>
<td>31 (20-42)</td>
<td>37 (29-44)</td>
</tr>
</tbody>
</table>

Figure 3. Overall survival in patients with NHL (A) and MM (B) stratified by treatment group.
speaking engagements from Sanofi. P.D.C., M.S., and R.V. were employees of Sanofi at the time of the study. The remaining authors have nothing to disclose.

Authorship statement: P.D.C. was the clinical lead and, along with M.S., was responsible for analyzing the data. R.V. was responsible for clinical trial management. All authors conducted the study, had full access to the data, and contributed to the development of the manuscript.

SUPPLEMENTARY DATA
Supplementary data related to this article can be found online at doi:10.1016/j.bbmt.2018.01.039.

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