original reports

FORT-1: Phase II/III Study of Rogaratinib Versus Chemotherapy in Patients With Locally Advanced or Metastatic Urothelial Carcinoma Selected Based on *FGFR1/3* mRNA Expression

Cora N. Sternberg, MD[1]; Daniel P. Petrylak, MD[2]; Joaquim Bellmunt, MD[3,4]; Hiroyuki Nishiyama, MD[5]; Andrea Necchi, MD[6]; Howard Gurney, MBBS[7]; Jae-Lyun Lee, MD[8]; Michiel S. van der Heijden, MD[9]; Eli Rosenbaum, MD[10]; Nicolas Penel, MD[11]; See-Tong Pang, MD[12]; Jian-Ri Li, MD[13]; Xavier García del Muro, MD[14]; Florence Joly, MD[15]; Zsuzsanna Pápai, MD[16]; Weichao Bao, PhD[17]; Peter Ellinghaus, PhD[18]; Chengxing Lu, PhD[17]; Mitchell Sierecki, MD[17]; Sabine Coppieters, MD[19]; Keiko Nakajima, MD[17]; Tatiane Cristine Ishida, MD[17]; and David I. Quinn, MBBS, PhD[20]

abstract

PURPOSE Rogaratinib, an oral pan-fibroblast growth factor receptor (FGFR1-4) inhibitor, showed promising phase I efficacy and safety in patients with advanced urothelial carcinoma (UC) with *FGFR1-3* mRNA overexpression. We assessed rogaratinib efficacy and safety versus chemotherapy in patients with *FGFR* mRNA-positive advanced/metastatic UC previously treated with platinum chemotherapy.

METHODS FORT-1 (ClinicalTrials.gov identifier: NCT03410693) was a phase II/III, randomized, open-label trial. Patients with *FGFR1/3* mRNA-positive locally advanced or metastatic UC with ≥ 1 prior platinum-containing regimen were randomly assigned (1:1) to rogaratinib (800 mg orally twice daily, 3-week cycles; n = 87) or chemotherapy (docetaxel 75 mg/m^2, paclitaxel 175 mg/m^2, or vinflunine 320 mg/m^2 intravenously once every 3 weeks; n = 88). The primary end point was overall survival, with objective response rate (ORR) analysis planned following phase II accrual. Because of comparable efficacy between treatments, enrollment was stopped before progression to phase III; a full interim analysis of phase II was completed.

RESULTS ORRs were 20.7% (rogaratinib, 18/87; 95% CI, 12.7 to 30.7) and 19.3% (chemotherapy, 17/88; 95% CI, 11.7 to 29.1). Median overall survival was 8.3 months (95% CI, 6.5 to not estimable) and 9.8 months (95% CI, 6.8 to not estimable; hazard ratio, 1.11; 95% CI, 0.71 to 1.72; $P = .67$). Grade 3/4 events occurred in 37 (43.0%)/4 (4.7%) patients and 32 (39.0%)/15 (18.3%), respectively. No rogaratinib-related deaths occurred. Exploratory analysis of patients with *FGFR3* DNA alterations showed ORRs of 52.4% (11/21; 95% CI, 29.8 to 74.3) for rogaratinib and 26.7% (4/15; 95% CI, 7.8 to 55.1) for chemotherapy.

CONCLUSION To our knowledge, these are the first data to compare FGFR-directed therapy with chemotherapy in patients with *FGFR*-altered UC, showing comparable efficacy and manageable safety. Exploratory testing suggested *FGFR3* DNA alterations in association with *FGFR1/3* mRNA overexpression may be better predictors of rogaratinib response.

J Clin Oncol 41:629-639. © 2022 by American Society of Clinical Oncology

ASSOCIATED CONTENT

Appendix

Data Supplement

Protocol

Author affiliations and support information (if applicable) appear at the end of this article.

Accepted on July 4, 2022 and published at ascopubs.org/journal/jco on October 14, 2022: DOI https://doi.org/10.1200/JCO.21.02303

INTRODUCTION

Patients with locally advanced or metastatic urothelial carcinoma (UC) have high recurrence rates following first-line platinum-based chemotherapy and poor prognosis.[1,2] Second-line treatments include immunotherapy and antibody-drug conjugates, with three immune checkpoint inhibitors approved by the US Food and Drug Administration in this setting.[1-4] The pan-fibroblast growth factor receptor (FGFR) inhibitor erdafitinib is approved for patients with susceptible *FGFR3* or *FGFR2* alterations following a phase II study reporting a 40% objective response rate (ORR) and median progression-free survival (PFS) and overall survival (OS) of 5.5 and 13.8 months, respectively.[5,6] Despite new options, many patients do not benefit from immunotherapy,[1,2] and metastatic UC remains a deadly disease in patients who relapse or progress during first-line chemotherapy.

Aberrant activation of FGFR signaling by genetic alterations affects tumorigenesis and progression of various cancers, including late-stage muscle-invasive UC.[7-9] Of the four known FGFR subtypes, *FGFR3* mutations have been identified in up to 42% of all UCs, up to 20% of metastatic disease cases, and up to 15%

CONTEXT

Key Objective

This interim analysis of the phase II FORT-1 trial evaluated the efficacy and safety of the fibroblast growth factor receptor (FGFR) inhibitor rogaratinib versus chemotherapy in patients with advanced or metastatic urothelial carcinoma selected on the basis of overexpression of *FGFR1* or *FGFR3* mRNA previously treated with platinum chemotherapy.

Knowledge Generated

Comparable efficacy outcomes were observed with rogaratinib versus chemotherapy in patients selected on the basis of *FGFR1/3* mRNA positivity. An exploratory analysis suggested that rogaratinib may yield greater antitumor benefit in patients with both *FGFR3* mRNA overexpression and an *FGFR* DNA alteration, warranting further investigation.

Relevance

To our knowledge, these are the first reported data comparing FGFR-targeted therapy with standard-of-care chemotherapy in patients selected on the basis of *FGFR* mRNA-positive urothelial carcinoma.

of muscle-invasive bladder tumors.[9-12] However, one study showed that 42% of bladder tumors without a detectable *FGFR3* DNA mutation had FGFR3 protein overexpression, suggesting that patients with wild-type or *FGFR3*-mutated tumors could benefit from FGFR-targeted therapies.[11] Activating mutations in *PIK3CA* and *RAS* have been associated with resistance to FGFR inhibition in solid tumors, including UC,[13-16] implying that patients with wild-type *PIK3CA* or *RAS* may demonstrate improved responses to FGFR inhibition.

Rogaratinib (Bayer AG, Berlin, Germany) is an oral FGFR1-4 inhibitor that showed promising efficacy and safety in a phase I study (ClinicalTrials.gov identifier: NCT01976741) of patients with advanced cancers selected on the basis of *FGFR1-3* mRNA overexpression and/or *FGFR3*-activating mutations/translocations.[17] ORR was 24% (12/51) in a subset of patients with advanced muscle-invasive UC. Retrospective analysis supported the association of *PIK3CA* or *RAS* mutations with resistance to FGFR inhibition.[17]

We present the results from an unplanned interim analysis of the phase II part of FORT-1 (ClinicalTrials.gov identifier: NCT03410693), a phase II/III, randomized, open-label study evaluating rogaratinib efficacy versus chemotherapy in patients with *FGFR* mRNA-positive advanced or metastatic UC previously treated with platinum chemotherapy.

METHODS

Study Design and Patients

This prospective, phase II, randomized, open-label, multicenter trial comprised *FGFR* testing, screening, treatment, and follow-up (Data Supplement, online only). The study was conducted at 161 academic medical centers/hospitals in Asia, Europe, North America, and Australia. *FGFR* testing was performed at the investigator's discretion $\leq$ 90 days before screening in patients age $\geq$ 18 years with locally advanced or metastatic UC, histologically or cytologically confirmed (including urinary bladder, renal pelvis, ureters, and urethra). Patients had to have an Eastern Cooperative Oncology Group performance status of 0 or 1 and availability of archival or fresh tumor biopsy. Only patients with *FGFR1/3* mRNA-positive tumors (high expression of *FGFR1* or *FGFR3* mRNA) were eligible to continue screening (Data Supplement).

All patients provided written, informed consent. The study site designated Institutional Review Board or equivalent approved the Protocol (online only) before the start of the study, according to Good Clinical Practice guidelines and the Declaration of Helsinki.

Random Assignment

Eligible patients with *FGFR1/3* mRNA-expressing tumors were randomly assigned 1:1 to rogaratinib or investigator-determined intravenous chemotherapy. Random assignment was stratified by the presence/absence of *PIK3CA-/RAS*-activating mutations, presence/absence of previous immunotherapy, and high/low modified four-factor Bellmunt risk score.[18]

Procedures

We centrally assessed *FGFR1* or *FGFR3* mRNA expression using in situ hybridization (RNAscope; developed by Advanced Cell Diagnostics, Newark, CA, in partnership with Leica Biosystems, Newcastle, United Kingdom), with high *FGFR* mRNA expression defined as an RNAscope score of 3+ or 4+.[17] During *FGFR* testing, we centrally tested DNA from tumor biopsies for *PIK3CA* and/or *RAS* mutations using polymerase chain reaction–based clinical trial assays (LabCorp, Burlington, NC). Because of lower-than-expected prevalence of *PIK3CA* and/or *RAS* resistance mutations, we reconfirmed absence or presence in all enrolled patients using a targeted Illumina MiSeq panel (Illumina, Inc, San Diego, CA) performed by TARGOS Molecular Pathology GmbH (Kassel, Germany). Exploratory *FGFR3* DNA mutation and fusion testing used the OmniSeq

Comprehensive targeted next-generation sequencing panel (OmniSeq, Buffalo, NY), with a minor allele frequency cutoff of 5%.

The starting dose of rogaratinib was 800 mg orally twice daily in continuous 3-week cycles. Chemotherapy could include intravenous docetaxel 75 mg/m^2, paclitaxel 175 mg/m^2, or vinflunine 320 mg/m^2 once every 3 weeks. Patients continued treatment until radiologic or clinical disease progression, unacceptable toxicity, or withdrawal.

We centrally assessed tumors using Response Evaluation Criteria in Solid Tumors version 1.1 at baseline, every 6 weeks up to week 18, and every 9 weeks thereafter. Assessments continued for at least 30 days for patients who discontinued treatment without disease progression.

We collected blood samples for biomarker analyses at screening, before dosing on day 1 of each cycle, and within 14 days of discontinuation, and plasma samples for pharmacokinetic assessment in patients treated with rogaratinib on day 1 of cycles 1-5 before dosing and 0.5-1.5 hours after dosing.

Outcomes

The primary end point for the planned phase II/III study was OS. Secondary end points included PFS, ORR, disease control rate (DCR), duration of response, safety, and tolerability. Safety was assessed throughout the treatment period, within 14 days of discontinuation, and up to 30 days after the last study treatment, including evaluation for retinopathy. Grade ≥ 2 retinal disorders were considered of special interest and monitored throughout the study (Data Supplement). Treatment-emergent adverse events (TEAEs) were classified using Medical Dictionary for Regulatory Activities version 22.1 and graded using National Cancer Institute Common Terminology Criteria for Adverse Events version 4.03.

Statistical Analyses

The phase II part of the study was designed to achieve 90% power to detect the difference in ORR between rogaratinib (assumed ORR = 30%) and chemotherapy (assumed ORR = 10%) in patients who were *FGFR* mRNA-positive with wild-type *PIK3CA*/*RAS*. Assuming a one-sided alpha of 0.1, a power of 90%, a mutation rate of approximately 25% for *PIK3CA* and *RAS* in the study population, and a random assignment ratio of 1:1, approximately 116 such patients were to be included in the planned analysis of ORR, on the basis of Fisher's exact test. phase II was planned to end after these first 116 enrolled patients completed 4.5 months of treatment, at which time the planned ORR analysis would be performed. Patients recruited to phase II were to automatically continue to phase III without interruption if futility was not demonstrated (Data Supplement).

We analyzed efficacy in all randomly assigned patients (full analysis set). The safety population comprised patients who received ≥ 1 dose of study treatment. ORR and DCR were compared using Fisher's exact test. Median OS and PFS were estimated using Kaplan-Meier methods. Hazard ratios and 95% CIs for OS and PFS were calculated using stratified Cox proportional hazards and a stratified log-rank test. Median follow-up time was calculated using the reverse Kaplan-Meier method.

Following a potential imbalance of deaths during the study period, the Data Monitoring Committee recommended a pause in enrollment and a reduction in the daily dose of rogaratinib from 800 to 600 mg twice daily for further evaluation. Because of similar efficacy between the treatment groups, the sponsor decided to stop further enrollment into the study on March 8, 2019. This report describes an interim analysis of efficacy and safety at a data cutoff date of November 25, 2019 (Data Supplement).

We performed a retrospective exploratory rescoring of the tumor samples from randomly assigned patients because of a higher-than-expected proportion of patients testing positive for tumors with high *FGFR1* or *FGFR3* mRNA expression (RNAscope score 3+ or 4+; Data Supplement). We analyzed the relationship between rogaratinib exposure and safety using logistic regression models (Data Supplement).

RESULTS

Patients

From May 31, 2018, to March 8, 2019, we tested 683 patients for *FGFR1/3* mRNA overexpression and 664 (97.2%) had a valid test result. Of these, 456 (68.7%) had *FGFR* overexpression at initial testing; 175 of these patients met the screening eligibility criteria, with 87 randomly assigned to rogaratinib and 88 to chemotherapy (Data Supplement). Of the 82 patients treated with chemotherapy, 40 (48.8%) received vinflunine, 24 (29.3%) received paclitaxel, and 18 (22.0%) received docetaxel. Overall, 7.4% of patients had wild-type *PIK3CA*/*RAS*, 10.9% were confirmed to have *PIK3CA* and/or *RAS* mutations, and 17.7% were unknown (Table 1). A higher percentage of patients receiving rogaratinib had stage IV B disease at study entry (Table 1).

Treatment

At the cutoff date (median follow-up 10.8 months; 95% CI, 10.1 to 11.7), median treatment duration was 12.0 weeks (range, 2.1-40.7 weeks) with rogaratinib and 9.4 weeks (range, 0.1-39.1 weeks) with chemotherapy, corresponding to a median of four treatment cycles (range, 1-14 cycles) in both treatment groups. One patient (1.1%) assigned to rogaratinib and six (6.8%) assigned to chemotherapy did not receive treatment and were excluded from the safety analyses. Most patients receiving rogaratinib (83/86 [96.5%]) had a starting dose of 800 mg. At the analysis cutoff date, six patients (6.9%) were ongoing with rogaratinib and four (4.5%) were ongoing with chemotherapy. The most common primary reason for treatment

TABLE 1. Patient Demographics and Baseline Cancer Characteristics

Characteristic	Rogaratinib (n = 87)	Chemotherapy (n = 88)	Total (N = 175)
Male, No. (%)	75 (86.2)	70 (79.5)	145 (82.9)
Age, years, median (range)	69.0 (36-82)	68.5 (37-89)	69.0 (36-89)
Country/geographic region, No. (%)			
North America, Western Europe, Israel, Australia	61 (70.1)	57 (64.8)	118 (67.4)
Asia	23 (26.4)	24 (27.3)	47 (26.9)
Rest of the world	3 (3.4)	7 (8.0)	10 (5.7)
ECOG performance status, No. (%)			
1	49 (56.3)	52 (59.1)	101 (57.7)
0	38 (43.7)	36 (40.9)	74 (42.3)
Location of primary cancer, No. (%)			
Bladder	56 (64.4)	45 (51.1)	101 (57.7)
Renal pelvis	12 (13.8)	28 (31.8)	40 (22.9)
Ureter	17 (19.5)	14 (15.9)	31 (17.7)
Proximal urethra	2 (2.3)	1 (1.1)	3 (1.7)
Time from most recent progression/relapse, months, median (range)	1.6 (0.3-19.1)	1.6 (0.3-8.1)	1.6 (0.3-19.1)
Liver metastases, No. (%)			
Absent	53 (60.9)	63 (71.6)	116 (66.3)
Present	34 (39.1)	25 (28.4)	59 (33.7)
Stage at study entry, No. (%)			
Stage III B	1 (1.1)	3 (3.4)	4 (2.3)
Stage IV	5 (5.7)	12 (13.6)	17 (9.7)
Stage IV A	13 (14.9)	24 (27.3)	37 (21.1)
Stage IV B	67 (77.0)	48 (54.5)	115 (65.7)
Unknown	1 (1.1)	1 (1.1)	2 (1.1)
Prior immunotherapy, No. (%)	39 (44.8)	39 (44.3)	78 (44.6)
PIK3CA-/*RAS*-activating mutations, No. (%)			
Absent	62 (71.3)	63 (71.6)	125 (71.4)
Present/unknown	25 (28.7)	25 (28.4)	50 (28.6)
Present	9 (10.3)	10 (11.4)	19 (10.9)
Unknown	16 (18.4)	15 (17.0)	31 (17.7)
Modified four-factor Bellmunt risk score, No. (%)			
High risk	16 (18.4)	15 (17.0)	31 (17.7)
Low risk	71 (81.6)	73 (83.0)	144 (82.3)

Abbreviation: ECOG, Eastern Cooperative Oncology Group.

discontinuation was disease progression, including radiologic progression in 53 patients (60.9%) receiving rogaratinib and 47 (53.4%) receiving chemotherapy, and clinical progression in zero and six (6.8%) patients, respectively.

Efficacy

In the overall population, ORRs of 20.7% (18/87) and 19.3% (17/88) were observed for patients assigned to rogaratinib and chemotherapy, respectively (rate difference = 1.4; one-sided $P = .48$), with similar DCRs between groups (Table 2). Efficacy was similar in patients whose *FGFR* expression was confirmed by retrospective exploratory rescoring compared with the overall population (Data Supplement). Of responders, 12/18 (66.7%) assigned to rogaratinib and 9/17 (52.9%) assigned to chemotherapy had previously received immunotherapy.

Median duration of response was 4.9 months (95% CI, 3.5 to 9.1) with rogaratinib and 5.8 months (95% CI, 3.5 to 7.7) with chemotherapy. Median OS was 8.3 months (95% CI, 6.5 to not evaluable) with rogaratinib and 9.8 months (95% CI, 6.8 to not evaluable) with chemotherapy (one-sided $P = .67$; Fig 1A). Median PFS was similar (Fig 1B).

TABLE 2. Objective Tumor Response (full analysis set)

Tumor Response	Rogaratinib (n = 87)	Chemotherapy (n = 88)
Complete response, No. (%)	2 (2.3)	3 (3.4)
Partial response, No. (%)	16 (18.4)	14 (15.9)
Stable disease, No. (%)	24 (27.6)	31 (35.2)
Progressive disease, No. (%)	27 (31.0)	22 (25.0)
Noncomplete response/nonprogressive disease, No. (%)	2 (2.3)	1 (1.1)
Missing/not evaluable[a], No. (%)	16 (18.4)	17 (19.3)
ORR[b], No. (%) [95% CI]	18 (20.7) [12.7 to 30.7]	17 (19.3) [11.7 to 29.1]
Rate difference	1.4 (−10.5 to 13.2)	
One-sided *P* value	.48	
Disease control rate[c], No. (%) [95% CI]	44 (50.6) [39.6 to 61.5]	49 (55.7) [44.7 to 66.3]
Rate difference	−5.1 (−19.9 to 9.7)	
One-sided *P* value	.80	

Abbreviation: ORR, objective response rate.

[a]Includes patients with missing data (no postbaseline assessment in 13 patients each assigned to rogaratinib and chemotherapy) and patients who were not evaluable (three and four patients, respectively).

[b]ORR = complete response + partial response.

[c]Disease control rate = complete response + partial response + stable disease + noncomplete response/nonprogressive disease.

No differences in ORR or DCR were observed in the subset of patients with confirmed *PIK3CA* and *RAS* mutations (Data Supplement).

A post hoc analysis of patients with high *FGFR3* mRNA expression identified 21/82 (25.6%) assigned to rogaratinib and 15/79 (19.0%) assigned to chemotherapy who also had *FGFR3* DNA alterations (four unique hotspot mutations [*FGFR3*-G370C, *FGFR3*-R248C, *FGFR3*-S249C, and *FGFR3*-Y373C] and two unique fusions [*FGFR3*-TACC3v1 and *FGFR3*-TACC3v3]). Retrospective exploratory analysis showed higher ORRs for patients assigned to rogaratinib than to chemotherapy (Table 3). Median OS at the later data cutoff was not reached in either group in this subset of patients (Data Supplement).

Safety

Grade 3 TEAEs occurred in 37 patients (43.0%) receiving rogaratinib and 32 (39.0%) receiving chemotherapy; grade 4 events occurred in 4 (4.7%) and 15 (18.3%), respectively (Table 4). Grade ≥ 2 retinal disorders were reported in six patients (7.0%) with rogaratinib and zero patients with chemotherapy; events included retinal pigment epithelium detachment in three patients (3.5%; all grade 2) and chorioretinopathy (grade 2), retinopathy (grade 3), and serous retinopathy (grade 2) in one patient each (1.2%). Including grade 1 events, 26 patients (30.2%) receiving rogaratinib and three (3.7%) receiving chemotherapy experienced a retinal disorder (Data Supplement). TEAEs remained similar following rescoring of *FGFR* expression (Data Supplement). Table 5 summarizes drug-related TEAEs.

Grade 5 TEAEs occurred in 19 patients (11.3%), 14 of whom (16.3%) received rogaratinib and five of whom (6.1%) received chemotherapy. The most common grade 5 events with rogaratinib were general physical health deterioration (n = 3) and dyspnea (n = 3), with no events considered drug-related. One grade 5 event in a patient receiving chemotherapy (respiratory tract infection) was considered drug-related. Of patients with grade 5 TEAEs, 11/14 (78.6%) receiving rogaratinib and 4/5 (80.0%) receiving chemotherapy had stage IV B disease at study entry. The Data Supplement provides further details on deaths during the study.

An exploratory analysis showed no significant relationships between area under the curve from 0 to 12 hours at steady state and adverse events such as diarrhea, vomiting, nausea, fatigue, retinal disorder, nail disorder, and increased serum lipase, or between exposure and grade ≥ 3 TEAEs (Data Supplement).

The Data Supplement summarizes dose modifications (interruptions or reductions). The most common TEAEs leading to dose modification were hyperphosphatemia (n = 18 [20.9%]) and diarrhea (n = 10 [11.6%]) with rogaratinib, and neutropenia/decreased neutrophil count (n = 6 [7.3%]) and fatigue (n = 4 [4.9%]) with chemotherapy. TEAEs led to permanent discontinuation in 15 patients (17.4%) receiving rogaratinib, most commonly asthenia in four patients (4.7%), with all other events occurring in one patient each (1.2%), and nine patients (11.0%) receiving chemotherapy, most commonly constipation, fatigue, and peripheral neuropathy in two patients each (2.4%).

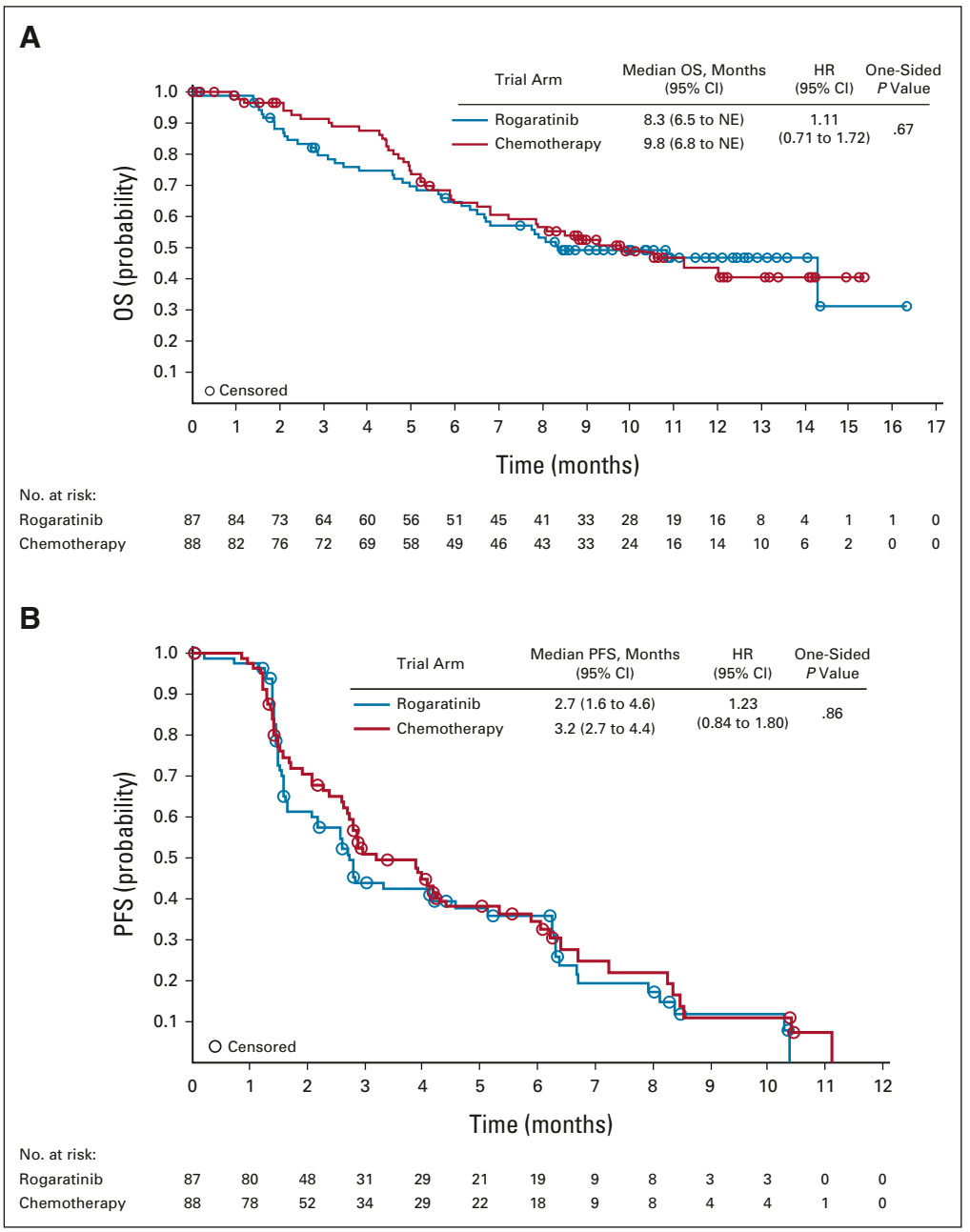

FIG 1. Kaplan-Meier curves of (A) OS and (B) PFS (full analysis set). Median (95% CI) from Kaplan-Meier estimates; *P* value is taken from stratified log-rank test; HR (95% CI) on the basis of stratified Cox proportional hazards model. HR, hazard ratio; NE, not evaluable; OS, overall survival; PFS, progression-free survival.

TEAE incidence was similar for those who received the planned rogaratinib dose of 800 mg twice daily or the reduced dose of 600 mg twice daily.

DISCUSSION

Interim results from the phase II part of the FORT-1 study of rogaratinib versus chemotherapy provide, to our knowledge, the first reported data comparing FGFR-targeted therapy with standard-of-care chemotherapy in patients selected for *FGFR* mRNA-positive UC, defined as overexpression of *FGFR1* or *FGFR3* mRNA.

This interim analysis of 175 patients revealed no significant differences in ORRs between patients assigned to rogaratinib or chemotherapy (20.7% and 19.3%, respectively). The ORR observed with rogaratinib is comparable with that in the subset of patients with UC selected on the basis of *FGFR1-3* mRNA overexpression in the phase I study of rogaratinib (23.5%),[17] and is in line with ORRs reported in

TABLE 3. Exploratory Analysis of Tumor Response by *FGFR3* DNA Alteration (full analysis set)

	Rogaratinib (n = 82)[a]		Chemotherapy (n = 79)[a]	
Tumor Response	***FGFR3* DNA[alt] (n = 21)**	***FGFR3* WT (n = 61)**	***FGFR3* DNA[alt] (n = 15)**	***FGFR3* WT (n = 64)**
ORR, No. (%) [95% CI]	11 (52.4) [29.8 to 74.3]	7 (11.5) [4.7 to 22.2]	4 (26.7) [7.8 to 55.1]	11 (17.2) [8.9 to 28.7]
Disease control rate, No. (%) [95% CI]	16 (76.2) [52.8 to 91.8]	28 (45.9) [33.1 to 59.2]	10 (66.7) [38.4 to 88.2]	33 (51.6) [38.7 to 64.2]

Abbreviations: alt, alteration; FGFR, fibroblast growth factor receptor; ORR, objective response rate; WT, wild-type.
[a]Patients with *FGFR1* mRNA-positive but mRNA-negative results were excluded from the analysis.

early-phase trials of other pan-FGFR inhibitors in patients with UC with *FGFR3* mutations.[19,20] Similarly, no significant differences in DCRs were observed in the rogaratinib and chemotherapy groups. The DCR observed with rogaratinib was slightly lower than that reported in other studies of early-phase FGFR inhibitors in patients with advanced UC selected on the basis of *FGFR3* DNA alterations[19,20] and in the phase I study of rogaratinib (71% overall and 73% in the subset with UC).[17]

OS and PFS were not statistically different within the treatment groups. OS with rogaratinib in patients selected on the basis of high expression of *FGFR1/3* mRNA was broadly similar to that reported with the FGFR1-3 inhibitor infigratinib in patients with advanced UC with *FGFR3* genetic alterations (7.75 months), with a comparable duration of treatment.[19] PFS was similar to that in the phase I study of rogaratinib (3.3 months).[17] In a phase II trial of the pan-FGFR inhibitor erdafitinib in patients with platinum-refractory advanced UC selected on the basis of *FGFR* DNA alterations, median OS and PFS were 13.8 and 5.5 months, respectively.[5] Our findings show that patient selection on the basis of *FGFR1* or *FGFR3* mRNA positivity alone does not lead to improved outcomes with rogaratinib versus chemotherapy.

Following a higher-than-expected proportion of patients having tumors with high *FGFR1* and *FGFR3* mRNA expression (69% in this study *v* 50% in the phase I study[17]), we performed a retrospective exploratory rescoring of tumor samples from randomly assigned patients; efficacy and safety were not significantly affected by this rescoring (Data Supplement). However, we cannot rule out the possibility that patients without high *FGFR1* and *FGFR3* mRNA expression were included in the study, potentially contributing to the limited objective responses with rogaratinib.

Because of the small proportion of patients with confirmed *PIK3CA/RAS* mutations, it was not possible to robustly test if wild-type *PIK3CA/RAS* was associated with improved responses to FGFR inhibition compared with patients with activating mutations. Previous observations linking *PIK3CA*- and *RAS*-activating mutations with resistance to FGFR inhibition in solid tumors, including UC,[13-16] indicate that this hypothesis may be worthy of investigation.

Retrospective exploratory analysis of patients positive for *FGFR3* mRNA and with *FGFR3* DNA alterations revealed a higher ORR with rogaratinib compared with the full analysis set (52.4% *v* 20.7%). An ORR of 40% was observed with erdafitinib in patients with advanced UC selected on the basis of *FGFR* DNA alterations.[5] In our study, median OS was not reached in either group in patients with *FGFR3* DNA alterations, but survival was slightly lower with rogaratinib compared with chemotherapy. However, these ORR and OS results should be interpreted with caution because of the small sample size and retrospective exploratory nature. Overall, the improved ORR with rogaratinib seen in this study in *FGFR* mRNA-positive patients with *FGFR* genetic DNA alterations is of interest and may warrant further evaluation.

GI toxicities were among the most commonly observed TEAEs, in line with the phase I study of rogaratinib[17] and other studies of pan-FGFR inhibitors in patients with advanced UC.[5,19,20] Hyperphosphatemia was the second most common TEAE with rogaratinib and is considered an on-target effect of FGFR inhibition related to FGFR2/3 signaling[21,22]; no grade ≥ 3 events were reported, and all cases of hyperphosphatemia resolved with treatment interruption and were without clinically relevant symptoms. Grade ≥ 2 retinal disorders were considered TEAEs of special interest and were reported in 7.0% of patients receiving rogaratinib compared with zero receiving chemotherapy; 30.2% of patients receiving rogaratinib and 3.7% receiving chemotherapy experienced retinal disorders of any grade. An exploratory analysis showed no significant relationships between rogaratinib exposure and TEAEs. Dose modifications were more frequent with rogaratinib than with chemotherapy, partly because of protocol-mandated modifications for hyperphosphatemia.

Grade 5 events were more common with rogaratinib than with chemotherapy (16.3% *v* 6.1%). Most grade 5 events across both groups were in patients with stage IV B disease at study entry, suggesting that these patients may have been at greater risk of TEAEs leading to death, irrespective of treatment group; the higher percentage of patients with stage IV B disease receiving rogaratinib may explain the slight imbalance in grade 5 events observed between groups. No grade 5 events were considered related to rogaratinib, and one case of grade 5 respiratory tract infection was attributed to chemotherapy. Potentially severe

TABLE 4. Summary of Most Common Any-Grade TEAEs Occurring in ≥ 10% of Patients in Either Treatment Group (safety analysis set[a])

	Rogaratinib (n = 86)			Chemotherapy (n = 82)			Total (N = 168)		
Adverse Event Category	**All Grade**	**Grade 3**	**Grade 4**	**All Grade**	**Grade 3**	**Grade 4**	**All Grade**	**Grade 3**	**Grade 4**
Any TEAE, No. (%)	86 (100)	37 (43.0)	4 (4.7)	82 (100)	32 (39.0)	15 (18.3)	168 (100)	69 (41.1)	19 (11.3)
System organ class MedDRA preferred term, No. (%)									
GI disorders									
Diarrhea	48 (55.8)	4 (4.7)	0	19 (23.2)	2 (2.4)	0	67 (39.9)	6 (3.6)	0
Constipation	25 (29.1)	1 (1.2)	0	29 (35.4)	0	0	54 (32.1)	1 (0.6)	0
Nausea	28 (32.6)	2 (2.3)	0	19 (23.2)	0	0	47 (28.0)	2 (1.2)	0
Vomiting	15 (17.4)	1 (1.2)	0	18 (22.0)	0	0	33 (19.6)	1 (0.6)	0
Abdominal pain	16 (18.6)	4 (4.7)	0	13 (15.9)	1 (1.2)	0	29 (17.3)	5 (3.0)	0
Stomatitis	10 (11.6)	1 (1.2)	0	10 (12.2)	0	0	20 (11.9)	1 (0.6)	0
Dry mouth	10 (11.6)	0	0	2 (2.4)	0	0	12 (7.1)	0	0
Metabolism and nutrition disorders									
Decreased appetite	35 (40.7)	2 (2.3)	0	20 (24.4)	1 (1.2)	0	55 (32.7)	3 (1.8)	0
Hyperphosphatemia	39 (45.3)	0	0	0	0	0	39 (23.2)	0	0
General disorders and administration site conditions									
Fatigue	21 (24.4)	2 (2.3)	0	28 (34.1)	5 (6.1)	0	49 (29.2)	7 (4.2)	0
Asthenia	25 (29.1)	8 (9.3)	0	19 (23.2)	1 (1.2)	0	44 (26.2)	9 (5.4)	0
Pyrexia	12 (14.0)	0	0	11 (13.4)	1 (1.2)	0	23 (13.7)	1 (0.6)	0
Peripheral edema	8 (9.3)	1 (1.2)	0	11 (13.4)	2 (2.4)	0	19 (11.3)	3 (1.8)	0
Skin and subcutaneous tissue disorders									
Alopecia	20 (23.3)	0	0	24 (29.3)	0	0	44 (26.2)	0	0
Blood and lymphatic system disorders									
Anemia	11 (12.8)	3 (3.5)	0	28 (34.1)	12 (14.6)	0	39 (23.2)	15 (8.9)	0
Neutropenia/decreased neutrophil count	3 (3.5)	0	1 (1.2)	35 (42.7)	14 (17.1)	8 (9.8)	38 (22.6)	14 (8.3)	9 (5.4)
Infections and infestations									
Urinary tract infection	11 (12.8)	2 (2.3)	0	10 (12.2)	4 (4.9)	1 (1.2)	21 (12.5)	6 (3.6)	1 (0.6)
Nervous system disorders									
Dysgeusia	13 (15.1)	0	0	5 (6.1)	0	0	18 (10.7)	0	0
Peripheral neuropathy	3 (3.5)	0	0	10 (12.2)	0	0	13 (7.7)	0	0
Peripheral sensory neuropathy	1 (1.2)	0	0	10 (12.2)	3 (3.7)	0	11 (6.5)	3 (1.8)	0
Investigations									
Increased blood creatinine	13 (15.1)	0	2 (2.3)	3 (3.7)	0	0	16 (9.5)	0	2 (1.2)
Increased lipase	9 (10.5)	6 (7.0)	1 (1.2)	3 (3.7)	2 (2.4)	0	12 (7.1)	8 (4.8)	1 (0.6)
Increased ALT	11 (12.8)	0	0	2 (2.4)	0	0	13 (7.7)	0	0
Increased AST	10 (11.6)	1 (1.2)	0	2 (2.4)	0	0	12 (7.1)	1 (0.6)	0
Increased blood alkaline phosphatase	10 (11.6)	0	0	2 (2.4)	0	0	12 (7.1)	0	0
Decreased weight	9 (10.5)	0	0	6 (7.3)	0	0	15 (8.9)	0	0
Musculoskeletal and connective tissue disorders									
Back pain	10 (11.6)	2 (2.3)	0	8 (9.8)	0	0	18 (10.7)	2 (1.2)	0
Myalgia	5 (5.8)	0	0	10 (12.2)	0	0	15 (8.9)	0	0
Respiratory, thoracic, and mediastinal disorders									
Epistaxis	10 (11.6)	0	0	1 (1.2)	0	0	11 (6.5)	0	0

NOTE. Grade 5 TEAEs are not included as they did not occur in > 10% of patients for any MedDRA preferred term; TEAEs leading to death (grade 5) are shown in the Data Supplement.

Abbreviations: MedDRA, Medical Dictionary for Regulatory Activities; TEAE, treatment-emergent adverse event.

[a]Excludes one patient assigned to rogaratinib and six patients assigned to chemotherapy who did not receive study treatment.

TABLE 5. Summary of Most Common Drug-Related Any-Grade TEAEs Occurring in ≥ 10% of Patients in Either Treatment Group (safety analysis set[a])

	Rogaratinib (n = 86)			Chemotherapy (n = 82)			Total (N = 168)		
Adverse Event Category	**All Grade**	**Grade 3**	**Grade 4**	**All Grade**	**Grade 3**	**Grade 4**	**All Grade**	**Grade 3**	**Grade 4**
Any TEAE, No. (%)	81 (94.2)	28 (32.6)	4 (4.7)	76 (92.7)	26 (31.7)	8 (9.8)	157 (93.5)	54 (32.1)	12 (7.1)
System organ class MedDRA preferred term, No. (%)									
GI disorders									
Diarrhea	41 (47.7)	2 (2.3)	0	13 (15.9)	1 (1.2)	0	54 (32.1)	3 (1.8)	0
Constipation	5 (5.8)	0	0	18 (22.0)	0	0	23 (13.7)	0	0
Nausea	20 (23.3)	2 (2.3)	0	17 (20.7)	0	0	37 (22.0)	2 (1.2)	0
Vomiting	8 (9.3)	1 (1.2)	0	14 (17.1)	0	0	22 (13.1)	1 (0.6)	0
Stomatitis	9 (10.5)	1 (1.2)	0	10 (12.2)	0	0	19 (11.3)	1 (0.6)	0
Skin and subcutaneous tissue disorders									
Alopecia	19 (22.1)	0	0	23 (28.0)	0	0	42 (25.0)	0	0
General disorders and administration site conditions									
Fatigue	13 (15.1)	1 (1.2)	0	25 (30.5)	3 (3.7)	0	38 (22.6)	4 (2.4)	0
Asthenia	16 (18.6)	6 (7.0)	0	16 (19.5)	0	0	32 (19.0)	6 (3.6)	0
Metabolism and nutrition disorders									
Hyperphosphatemia	37 (43.0)	0	0	0	0	0	37 (22.0)	0	0
Decreased appetite	20 (23.3)	1 (1.2)	0	13 (15.9)	0	0	33 (19.6)	1 (0.6)	0
Blood and lymphatic system disorders									
Anemia	5 (5.8)	2 (2.3)	0	20 (24.4)	7 (8.5)	0	25 (14.9)	9 (5.4)	0
Neutropenia/decreased neutrophil count	2 (2.3)	0	1 (1.2)	34 (41.5)	13 (15.9)	7 (8.5)	36 (21.4)	13 (7.7)	8 (4.8)
Nervous system disorders									
Dysgeusia	12 (14.0)	0	0	5 (6.1)	0	0	17 (10.1)	0	0
Peripheral neuropathy	2 (2.3)	0	0	9 (11.0)	0	0	11 (6.5)	0	0
Peripheral sensory neuropathy	0	0	0	10 (12.2)	3 (3.7)	0	10 (6.0)	3 (1.8)	0
Musculoskeletal and connective tissue disorders									
Myalgia	3 (3.5)	0	0	10 (12.2)	0	0	13 (7.7)	0	0

NOTE. Drug-related retinopathy was reported in three patients (2.3%) receiving rogaratinib (two events of grade 1 and one event of grade 3) and in zero patients receiving chemotherapy. Grade 5 TEAEs are not included as they did not occur in > 10% of patients for any MedDRA preferred term; TEAEs leading to death (grade 5) are shown in the Data Supplement.

Abbreviations: MedDRA, Medical Dictionary for Regulatory Activities; TEAE, treatment-emergent adverse event.

[a]Excludes one patient assigned to rogaratinib and six patients assigned to chemotherapy who did not receive study treatment.

lung infection as a result of immune consequences is a known side effect of chemotherapy.[23]

In conclusion, to our knowledge, these are the first reported data comparing FGFR-directed therapy with chemotherapy in patients with *FGFR*-altered UC. Rogaratinib demonstrated efficacy comparable with standard chemotherapy and a manageable safety profile. Prespecified efficacy criteria were not met for continuation to phase III in this population. An exploratory analysis suggested that rogaratinib may have greater antitumor benefit in patients with both *FGFR3* mRNA overexpression and an *FGFR* DNA alteration, which warrants further investigation.

AFFILIATIONS

[1]Englander Institute for Precision Medicine, Weill Cornell Medicine, Sandra and Edward Meyer Cancer Center, New York, NY
[2]Yale Cancer Center, New Haven, CT
[3]Beth Israel Deaconess Medical Center and PSMAR-IMIM Lab, Boston, MA
[4]Harvard Medical School, Boston, MA
[5]Department of Urology, University of Tsukuba, Tsukuba, Japan
[6]Vita-Salute San Raffaele University and IRCCS San Raffaele Hospital and Scientific Institute, Milan, Italy
[7]Clinical Trials Unit FMHS, Macquarie University, Sydney, New South Wales, Australia
[8]University of Ulsan College of Medicine, Asan Medical Center, Seoul, Republic of Korea
[9]Medical Oncology, the Netherlands Cancer Institute, Antoni van Leeuwenhoek Hospital, Amsterdam, the Netherlands

[10]Davidoff Cancer Center, Rabin Medical Center, Petah Tikva, Israel
[11]Lille University and Department of Medical Oncology, Centre Oscar Lambret, Lille, France
[12]Division of Urology, Department of Surgery, Linkou Chang Gung Memorial Hospital, Linkou, Taiwan
[13]Division of Urology, Department of Surgery, Taichung Veterans General Hospital, Taichung, Taiwan
[14]Department of Medical Oncology, University of Barcelona, Idibell Institute of Research, Institut Català d'Oncologia Hospitalet, Barcelona, Spain
[15]Clinical Research Department, Centre François Baclesse, Caen, France
[16]Oncology Department, Medical Centre, Hungarian Defence Forces, Budapest, Hungary
[17]Bayer HealthCare Pharmaceuticals, Inc, Whippany, NJ
[18]Bayer AG, Wuppertal, Germany
[19]Bayer AG, Diegem, Belgium
[20]Division of Oncology, Department of Medicine, USC Norris Comprehensive Cancer Center, Los Angeles, CA

CORRESPONDING AUTHOR

David I. Quinn, MBBS, PhD, Division of Oncology, Department of Medicine, USC Norris Comprehensive Cancer Center, 1441 Eastlake Ave, Ste 3440, Los Angeles, CA 90033; e-mail: diquinn@usc.edu.

PRIOR PRESENTATION

Presented in part at the 2020 ASCO Genitourinary Cancers Symposium, San Francisco, CA, February 13-15, 2020.

SUPPORT

Supported by research funding from Bayer AG.

CLINICAL TRIAL INFORMATION

NCT03410693

AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

Disclosures provided by the authors are available with this article at DOI https://doi.org/10.1200/JCO.21.02303.

DATA SHARING STATEMENT

Availability of the data underlying this publication will be determined according to Bayer's commitment to the EFPIA/PhRMA Principles for Responsible Clinical Trial Data Sharing. This pertains to scope, time point, and process of data access.
As such, Bayer commits to sharing upon request from qualified scientific and medical researchers patient-level clinical trial data, study-level clinical trial data, and protocols from clinical trials in patients for medicines and indications approved in the United States and European Union as necessary for conducting legitimate research. This applies to data on new medicines and indications that have been approved by the EU and US regulatory agencies on or after January 1, 2014.
Interested researchers can use www.clinicalstudydatarequest.com to request access to anonymized patient-level data and supporting documents from clinical studies to conduct further research that can help advance medical science or improve patient care. Information on the Bayer criteria for listing studies and other relevant information is provided in the study sponsor's section of the portal.
Data access will be granted to anonymized patient-level data, protocols, and clinical study reports after approval by an independent scientific review panel. Bayer is not involved in the decisions made by the independent review panel. Bayer will take all necessary measures to ensure that patient privacy is safeguarded.

AUTHOR CONTRIBUTIONS

Conception and design: Cora N. Sternberg, Daniel P. Petrylak, Joaquim Bellmunt, Weichao Bao, Peter Ellinghaus, Chengxing Lu, Sabine Coppieters, Keiko Nakajima, Tatiane Cristine Ishida, David I. Quinn
Administrative support: Tatiane Cristine Ishida
Provision of study materials or patients: Cora N. Sternberg, Joaquim Bellmunt, Howard Gurney, Jae-Lyun Lee, Michiel S. van der Heijden, Nicolas Penel, See-Tong Pang, Jian-Ri Li, Xavier García del Muro, Florence Joly, David I. Quinn
Collection and assembly of data: Cora N. Sternberg, Joaquim Bellmunt, Hiroyuki Nishiyama, Howard Gurney, Jae-Lyun Lee, Michiel S. van der Heijden, See-Tong Pang, Jian-Ri Li, Xavier García del Muro, Peter Ellinghaus, Chengxing Lu, Sabine Coppieters, Keiko Nakajima, Tatiane Cristine Ishida, David I. Quinn
Data analysis and interpretation: Cora N. Sternberg, Joaquim Bellmunt, Hiroyuki Nishiyama, Andrea Necchi, Howard Gurney, Jae-Lyun Lee, Michiel S. van der Heijden, Eli Rosenbaum, Nicolas Penel, Xavier García del Muro, Florence Joly, Weichao Bao, Peter Ellinghaus, Chengxing Lu, Mitchell Sierecki, Sabine Coppieters, Keiko Nakajima, Tatiane Cristine Ishida, David I. Quinn
Manuscript writing: All authors
Final approval of manuscript: All authors
Accountable for all aspects of the work: All authors

ACKNOWLEDGMENT

The authors wish to thank the patients and their families, coinvestigators, and referring physicians who participated in this study. The authors thank Ashraf Yassen and Xiang Qing Yu of Bayer AG, and Jon Moss and Adam Lloyd of BAST Inc Limited, Kington, United Kingdom, for their expertise and assistance with pharmacometric evaluations, and Bingyan Wu of Bayer AG for her assistance with statistical analyses and outputs. Laura Valenzo, PhD, and Jake Stoddart, MRes, of Complete HealthVizion, McCann Health Medical Communications, provided medical writing support with this manuscript, on the basis of detailed discussion and feedback from all the authors; this assistance was funded by Bayer AG. RNAscope assay for *FGFR* expression was developed for use in this study by Leica Biosystems (Newcastle upon Tyne, United Kingdom).
The list of FORT-1 trial investigators who contributed to the success of the trial is available in Appendix Table A1 (online only).

REFERENCES

1. Dietrich B, Siefker-Radtke AO, Srinivas S, et al: Systemic therapy for advanced urothelial carcinoma: Current standards and treatment considerations. Am Soc Clin Oncol Ed Book 38:342-353, 2018
2. Nadal R, Bellmunt J: Management of metastatic bladder cancer. Cancer Treat Rev 76:10-21, 2019
3. AstraZeneca: Voluntary Withdrawal of Imfinzi Indication in Advanced Bladder Cancer in the US, 2021. https://www.astrazeneca.com/content/astraz/media-centre/press-releases/2021/voluntary-withdrawal-imfinzi-us-bladder-indication.html
4. Roche: Roche Provides Update on Tecentriq US Indication in Prior-Platinum Treated Metastatic Bladder Cancer, 2021. https://www.roche.com/media/releases/med-cor-2021-03-08.htm
5. Loriot Y, Necchi A, Park SH, et al: Erdafitinib in locally advanced or metastatic urothelial carcinoma. N Engl J Med 381:338-348, 2019

6. US Food and Drug Administration: FDA Grants Accelerated Approval to Erdafitinib for Metastatic Urothelial Carcinoma, 2019. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-grants-accelerated-approval-erdafitinib-metastatic-urothelial-carcinoma
7. Ahmad I, Iwata T, Leung HY: Mechanisms of FGFR-mediated carcinogenesis. Biochim Biophys Acta 1823:850-860, 2012
8. Guancial EA, Werner L, Bellmunt J, et al: FGFR3 expression in primary and metastatic urothelial carcinoma of the bladder. Cancer Med 3:835-844, 2014
9. Helsten T, Elkin S, Arthur E, et al: The FGFR landscape in cancer: Analysis of 4,853 tumors by next-generation sequencing. Clin Cancer Res 22:259-267, 2016
10. Robertson AG, Kim J, Al-Ahmadie H, et al: Comprehensive molecular characterization of muscle-invasive bladder cancer. Cell 171:540-556.e25, 2017
11. Tomlinson DC, Baldo O, Harnden P, et al: FGFR3 protein expression and its relationship to mutation status and prognostic variables in bladder cancer. J Pathol 213:91-98, 2007
12. Knowles MA, Hurst CD: Molecular biology of bladder cancer: New insights into pathogenesis and clinical diversity. Nat Rev Cancer 15:25-41, 2015
13. Wang L, Hu H, Pan Y, et al: *PIK3CA* mutations frequently coexist with *EGFR*/*KRAS* mutations in non-small cell lung cancer and suggest poor prognosis in *EGFR*/*KRAS* wildtype subgroup. PLoS One 9:e88291, 2014
14. De Roock W, Claes B, Bernasconi D, et al: Effects of *KRAS, BRAF, NRAS*, and *PIK3CA* mutations on the efficacy of cetuximab plus chemotherapy in chemotherapy-refractory metastatic colorectal cancer: A retrospective consortium analysis. Lancet Oncol 11:753-762, 2010
15. Nagano T, Tachihara M, Nishimura Y: Mechanism of resistance to epidermal growth factor receptor-tyrosine kinase inhibitors and a potential treatment strategy. Cells 7:212, 2018
16. Kompier LC, Lurkin I, van der Aa MN, et al: *FGFR3, HRAS, KRAS, NRAS* and *PIK3CA* mutations in bladder cancer and their potential as biomarkers for surveillance and therapy. PLoS One 5:e13821, 2010
17. Schuler M, Cho BC, Sayehli CM, et al: Rogaratinib in patients with advanced cancers selected by *FGFR* mRNA expression: A phase 1 dose-escalation and dose-expansion study. Lancet Oncol 20:1454-1466, 2019
18. Bellmunt J, Choueiri TK, Fougeray R, et al: Prognostic factors in patients with advanced transitional cell carcinoma of the urothelial tract experiencing treatment failure with platinum-containing regimens. J Clin Oncol 28:1850-1855, 2010
19. Pal SK, Rosenberg JE, Hoffman-Censits JH, et al: Efficacy of BGJ398, a fibroblast growth factor receptor 1-3 inhibitor, in patients with previously treated advanced urothelial carcinoma with *FGFR3* alterations. Cancer Discov 8:812-821, 2018
20. Necchi A, Pouessel D, Leibowitz-Amit R, et al: Interim results of fight-201, a phase II, open-label, multicenter study of INCB054828 in patients (pts) with metastatic or surgically unresectable urothelial carcinoma (UC) harboring fibroblast growth factor (FGF)/FGF receptor (FGFR) genetic alterations (GA). Ann Oncol 29:900P, 2018 (suppl 8)
21. Hierro C, Rodon J, Tabernero J: Fibroblast growth factor (FGF) receptor/FGF inhibitors: Novel targets and strategies for optimization of response of solid tumors. Semin Oncol 42:801-819, 2015
22. Chae YK, Ranganath K, Hammerman PS, et al: Inhibition of the fibroblast growth factor receptor (FGFR) pathway: The current landscape and barriers to clinical application. Oncotarget 8:16052-16074, 2017
23. Vento S, Cainelli F, Temesgen Z: Lung infections after cancer chemotherapy. Lancet Oncol 9:982-992, 2008

AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

FORT-1: Phase II/III Study of Rogaratinib Versus Chemotherapy in Patients With Locally Advanced or Metastatic Urothelial Carcinoma Selected Based on *FGFR1/3* mRNA Expression

The following represents disclosure information provided by authors of this manuscript. All relationships are considered compensated unless otherwise noted. Relationships are self-held unless noted. I = Immediate Family Member, Inst = My Institution. Relationships may not relate to the subject matter of this manuscript. For more information about ASCO's conflict of interest policy, please refer to www.asco.org/rwc or ascopubs.org/jco/authors/author-center.

Open Payments is a public database containing information reported by companies about payments made to US-licensed physicians (Open Payments).

Jae-Lyun Lee
Stock and Other Ownership Interests: Myovant Sciences, Johnson & Johnson/Janssen, Amgen, Merck, BeiGene, Innovent Biologics, Black Diamond Therapeutics, Karyopharm Therapeutics, Zymeworks
Honoraria: Bristol Myers Squibb, Astellas Pharma, Pfizer, AstraZeneca, MSD
Consulting or Advisory Role: Pfizer, BMS Korea, GI Innovation, MSD, Merck, AstraZeneca, Sanofi, Oscotec
Research Funding: Pfizer (Inst), Janssen (Inst), Novartis (Inst), Bristol Myers Squibb (Inst), Roche/Genentech (Inst), AstraZeneca/MedImmune (Inst), MSD (Inst), Bayer Schering Pharma (Inst), Seattle Genetics (Inst), GI Innovation (Inst), Amgen (Inst)

Tatiane Cristine Ishida
Employment: Bayer

Hiroyuki Nishiyama
Consulting or Advisory Role: MSD, Chugai Pharma, Bayer Yakuhin, Janssen, Lilly
Speakers' Bureau: MSD, Chugai Pharma, Astellas Pharma
Research Funding: Astellas Pharma (Inst), Ono Pharmaceutical (Inst), Takeda (Inst), Bayer Yakuhin (Inst)

Sabine Coppieters
Employment: Bayer, Argenx
Stock and Other Ownership Interests: Bayer, Argenx

Michiel S. van der Heijden
Stock and Other Ownership Interests: Gilead Sciences
Consulting or Advisory Role: Roche/Genentech (Inst), Astellas Pharma (Inst), AstraZeneca/MedImmune (Inst), Bristol Myers Squibb (Inst), MSD Oncology (Inst), Seattle Genetics (Inst), Janssen (Inst), Pfizer (Inst)
Research Funding: Astellas Pharma (Inst), Bristol Myers Squibb (Inst), Roche (Inst), AstraZeneca (Inst), Seattle Genetics (Inst), 4SC (Inst)
Travel, Accommodations, Expenses: Novartis, Astellas Pharma, MSD Oncology, Roche

Weichao Bao
Stock and Other Ownership Interests: Bayer (Inst)

David I. Quinn
Employment: AbbVie
Honoraria: Bayer, Pfizer, Genentech/Roche, Merck Sharp & Dohme, Bristol Myers Squibb, Exelixis, Seattle Genetics, Myovant Sciences, AVEO, Clinigen Group
Consulting or Advisory Role: Pfizer, Bristol Myers Squibb, Genentech/Roche, Merck Sharp & Dohme, Bayer, Exelixis, Eisai, US Biotest, Seattle Genetics, Myovant Sciences, AVEO, Clinigen Group
Research Funding: Genentech/Roche (Inst), Merck (Inst), Pfizer (Inst)
Travel, Accommodations, Expenses: Bayer, Exelixis
Uncompensated Relationships: Eisai, US Biotest

Howard Gurney
Consulting or Advisory Role: Bristol Myers Squibb, Ipsen, Merck Sharp & Dohme, AstraZeneca, Janssen-Cilag, Pfizer, Roche, Merck Serono, Astellas Pharma
Speakers' Bureau: Merck Serono
Travel, Accommodations, Expenses: AstraZeneca

Florence Joly
Consulting or Advisory Role: AstraZeneca, Janssen, Ipsen, Pfizer, MSD Oncology, Bristol Myers Squibb, GlaxoSmithKline, Astellas Pharma, Clovis Oncology, Amgen, Seattle Genetics, Bayer
Travel, Accommodations, Expenses: Janssen, AstraZeneca, Ipsen, GlaxoSmithKline, BMS

Keiko Nakajima
Employment: Bayer, Daiichi Sankyo/Astra Zeneca
Stock and Other Ownership Interests: Bayer, Daiichi Sankyo/Astra Zeneca

Joaquim Bellmunt
Stock and Other Ownership Interests: Rainier Therapeutics
Honoraria: UpToDate
Consulting or Advisory Role: Pierre Fabre, Astellas Pharma, Pfizer, Merck, Genentech, Novartis, AstraZeneca/MedImmune, Bristol Myers Squibb
Research Funding: Millennium (Inst), Sanofi (Inst), Pfizer/EMD Serono (Inst)
Travel, Accommodations, Expenses: Pfizer, MSD Oncology, Ipsen

Mitchell Sierecki
Employment: Gilead Sciences, Bayer HealthCare Pharmaceuticals
Leadership: Gilead Sciences, Bayer HealthCare Pharmaceuticals
Travel, Accommodations, Expenses: Gilead Sciences

Andrea Necchi
Employment: Bayer
Stock and Other Ownership Interests: Bayer
Honoraria: Roche, Merck, AstraZeneca, Janssen, Foundation Medicine, Bristol Myers Squibb
Consulting or Advisory Role: Merck Sharp & Dohme, Roche, Bayer, AstraZeneca, Clovis Oncology, Janssen, Incyte, Seattle Genetics/Astellas, Bristol Myers Squibb, Rainier Therapeutics, GlaxoSmithKline, Ferring
Research Funding: Merck Sharp & Dohme (Inst), AstraZeneca (Inst), Ipsen, Seattle Genetics (Inst)
Travel, Accommodations, Expenses: Roche, Merck Sharp & Dohme, AstraZeneca, Janssen, Rainier Therapeutics
Other Relationship: Bayer

Xavier García del Muro
Consulting or Advisory Role: Pfizer, Bristol Myers Squibb, Ipsen, Roche, Lilly, PharmaMar, EUSA Pharma, GlaxoSmithKline, Merck, Eisai
Speakers' Bureau: Pfizer, Bristol Myers Squibb, Astellas Pharma, Eisai
Research Funding: AstraZeneca
Travel, Accommodations, Expenses: Pfizer, Roche

Cora N. Sternberg
Consulting or Advisory Role: Bayer, MSD, Pfizer, Roche, Incyte, AstraZeneca, Merck, Medscape, UroToday, Astellas Pharma, Genzyme, Immunomedics, Foundation Medicine, Bristol Myers Squibb/Medarex, IMPAC Medical Systems

Eli Rosenbaum
Stock and Other Ownership Interests: Brainsway, Conergent
Consulting or Advisory Role: MSD Oncology, Teva, Astellas Pharma, Bayer, Janssen
Speakers' Bureau: MSD Oncology

Peter Ellinghaus
Employment: Bayer
Leadership: Bayer
Stock and Other Ownership Interests: Bayer

Daniel P. Petrylak
Stock and Other Ownership Interests: Bellicum Pharmaceuticals, TYME
Consulting or Advisory Role: Bayer, Exelixis, Pfizer, Roche, Astellas Pharma, AstraZeneca, Lilly, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Clovis Oncology, Incyte, Janssen, Pharmacyclics, Seattle Genetics, Urogen pharma, Advanced Accelerator Applications, Ipsen, Bicycle Therapeutics, Mirati Therapeutics, Monopteros Therapeutics, Regeneron, Gilead Sciences
Research Funding: Progenics (Inst), Sanofi (Inst), Endocyte (Inst), Genentech (Inst), Merck (Inst), Astellas Medivation (Inst), Novartis (Inst), AstraZeneca (Inst), Bayer (Inst), Lilly (Inst), Innocrin Pharma (Inst), MedImmune (Inst), Pfizer (Inst), Roche (Inst), Seattle Genetics (Inst), Clovis Oncology (Inst), Bristol Myers Squibb (Inst), Advanced Accelerator Applications (Inst), Agensys (Inst), BioXCel Therapeutics (Inst), Eisai (Inst), Mirati Therapeutics (Inst), Replimune (Inst), Medivation (Inst), Gilead Sciences (Inst)
Expert Testimony: Celgene, Sanofi

Nicolas Penel
Research Funding: Bayer (Inst)
Travel, Accommodations, Expenses: Astellas Pharma, Janssen-Cilag
Other Relationship: PharmaMar

Chengxing Lu
Employment: Bayer, Biogen, AstraZeneca
Stock and Other Ownership Interests: Bayer, Biogen, AstraZeneca

No other potential conflicts of interest were reported.

APPENDIX

TABLE A1. FORT-1 Principal Investigators

Principal Investigator	Site Name	City/State/Region	Country
Renuka Chittajallu	Riverina Cancer Care Center	Wagga Wagga, NSW	Australia
William Fox	Mid North Coast Cancer Institute	Coffs Harbour, NSW	Australia
Howard Gurney	Macquarie University Hospital	Sydney, NSW	Australia
Laurence Krieger	Northern Cancer Institute	Sydney, NSW	Australia
Gavin Marx	Sydney Adventist Hospital	Sydney, NSW	Australia
Marco Matos	Pindara Private Hospital	Gold Coast, QLD	Australia
David Pook	Monash Medical Center	Melbourne, VIC	Australia
Kilian Gust	Universitätsklinikum AKH Wien	Vienna	Austria
Wolfgang Loidl	Ordensklinikum Linz GmbH Elisabethinen	Linz	Austria
Dora Niedersuess-Beke	Klinik Ottakring - Wilhelminenspital	Vienna	Austria
Sonia Vallet	Landesklinikum Krems	Krems	Austria
Sabine Weibrecht	Krankenhaus der Barmherzigen Brüder	Vienna	Austria
Herlinde Dumez	UZ Leuven Gasthuisberg	Leuven	Belgium
Sylvie Rottey	UZ Gent	Ghent	Belgium
Nicolas Whenham	Clinique Saint-Pierre	Ottignies	Belgium
Cristiano Ferrario	Sir Mortimer B. Davis Jewish General Hospital	Montreal, QC	Canada
Michael Ong	Ottawa Hospital-General Campus	Ottawa, ON	Canada
Srikala Sridhar	Princess Margaret Hospital-University Health Network	Toronto, ON	Canada
Lijun Chen	Fifth Medical Center, General Hospital of the Chinese People	Beijing	China
Cheng Fu	Liaoning Cancer Hospital and Institute	Shengyang	China
Hongqian Guo	Nanjing Drum Tower Hospital, The Affiliated Hospital of Nanjing University Medical School	Nanjing	China
Yongda Liu	First Affiliated Hospital of Guangzhou Medical University	Guangzhou	China
Zhongquan Sun	Huadong Hospital, Affiliated to Fudan University	Shanghai	China
Shaozhong Wei	Hubei Cancer Hospital	Wuhan	China
Dingwei Ye	Fudan University Shanghai Cancer Center	Shanghai	China
Song Zheng	Fujian Medical University Union Hospital	Fuzhou	China
Fangjian Zhou	Sun Yat-sen University Cancer Center	Guangzhou	China
Qing Zou	Jiangsu Cancer Hospital	Nanjing	China
Jan Dvorak	Fakultni nemocnice Kralovske Vinohrady	Prague	Czech Republic
Jaroslav Hajek	Fakultni nemocnice Ostrava	Ostrava	Czech Republic
Milan Kohoutek	Bata Hospital	Zlín	Czech Republic
Michaela Matouskova	Fakultni Thomayerova Nemocnice	Prague	Czech Republic
Mads Agerbaek	Aarhus Universitetshospital, Skejby	Aarhus	Denmark
Line Dohn	Herlev Hospital - Oncology Research Dept.	Herlev	Denmark
Henriette Lindberg	Herlev Hospital - Oncology Research Dept.	Herlev	Denmark
Helle Pappot	Rigshospitalet	Copenhagen	Denmark
Tuomo Alanko	Docrates Klinikka	Helsinki	Finland
Katriina Peltola	Docrates Klinikka	Helsinki	Finland
Philippe Beuzeboc	Center Médico-Chirurgical Foch	Suresnes	France
Anne Escande	Clinique Saint Anne	Strasbourg	France

(continued on following page)

TABLE A1. FORT-1 Principal Investigators (continued)

Principal Investigator	Site Name	City/State/Region	Country
François-Régis Ferrand	Hôpital d'Instruction des Armées Begin	Saint Mandé	France
Aude Flechon	Center Léon Bérard	Lyon	France
Gwenaelle Gravis	Institut Paoli-Calmettes	Marseille	France
Marine Gross-Goupil	Hôpital Saint André	Bordeaux	France
Olivier Huillard	Hôpital Cochin	Paris	France
Florence Joly	Center de Lutte Contre le Cancer François Baclesse	Caen	France
Fredrik Laestadius	Center Oscar Lambret	Lille	France
Hakim Mahammedi	Center Jean Perrin	Clermont-Ferrand	France
Nicolas Penel	Center Oscar Lambret	Lille	France
Antoine Thiery-Vuillemin	Hopital Jean Minjoz	Besançon	France
Georg Bartsch	Universitätsmedizin der Johannes Gutenberg Universität Mainz	Mainz	Germany
Günter Niegisch	Heinrich-Heine-Universität Düsseldorf	Düsseldorf	Germany
Arnulf Stenzl	Eberhard-Karls-Universität Tübingen	Tübingen	Germany
Darren Ming-Chun Poon	Prince of Wales Hospital Hong Kong	Hong Kong	Hong Kong
Lajos Geczi	Orszagos Onkologiai Intezet	Budapest	Hungary
Laszlo Mangel	Pecsi Tudomanyegyetem Klinikai Kozpont	Pécs	Hungary
Zsuzsanna Papai	MH Egeszsegugyi Kozpont	Budapest	Hungary
Richard Bambury	Cork University Hospital	Cork	Ireland
Ray McDermott	AMNCH	Dublin	Ireland
Raanan Berger	Chaim Sheba Medical Center	Ramat Gan	Israel
Stephen Frank	Hadassah Hebrew University Hospital Ein Kerem	Jerusalem	Israel
Daniel Kejzman	Meir Medical Center	Kfar Saba	Israel
Raya Leibowitz-Amit	Chaim Sheba Medical Center	Ramat Gan	Israel
Avivit Peer	Rambam Health Corporation	Haifa	Israel
Eli Rosenbaum	Clalit Health Services Rabin Medical Center-Beilinson Campus	Petah Tikva	Israel
Fabio Calabro	AO San Camillo-Forlanini	Rome	Italy
Ugo Federico Francesco De Giorgi	IRST Istituto Scientifico Romagnolo per studio e cura tumori	Meldola	Italy
Luca Galli	AOU Pisana	Pisa	Italy
Roberto Iacovelli	AOUI Verona	Verona	Italy
Michele Milella	AOUI Verona	Verona	Italy
Claudia Mucciarini	AUSL Modena	Modena	Italy
Andrea Necchi	Fondazione IRCCS Istituto Nazionale dei Tumori	Milan	Italy
Franco Nole	IRCCS Istituto Europeo di Oncologia s.r.l. (IEO)	Milan	Italy
Roberto Sabbatini	AOU di Modena - Policlinico	Modena	Italy
Giorgio Vittorio Scagliotti	AOU San Luigi Gonzaga	Torino	Italy
Giovanni Schinzari	Fondazione Policlinico Universitario Agostino Gemelli IRCCS	Rome	Italy
Salvatore Siena	ASST Grande Ospedale Metropolitano Niguarda	Milan	Italy
Cora Sternberg	AO San Camillo-Forlanini	Rome	Italy
Takashige Abe	Hokkaido University Hospital	Sapporo	Japan
Shin Ebara	Hiroshima City Hiroshima Citizens Hospital	Hiroshima	Japan
Masatoshi Eto	Kyushu University Hospital	Fukuoka	Japan
Hiroyuki Fujimoto	National Cancer Center Hospital	Chūō, Tokyo	Japan
Kenichi Kakimoto	Osaka International Cancer Institute	Osaka	Japan

(continued on following page)

TABLE A1. FORT-1 Principal Investigators (continued)

Principal Investigator	Site Name	City/State/Region	Country
Mutsushi Kawakita	Kobe City Medical Center General Hospital	Kobe	Japan
Hiroshi Kitamura	Toyama University Hospital	Toyama	Japan
Takahiro Kojima	University of Tsukuba Hospital	Tsukuba	Japan
Yukihiro Kondo	Nippon Medical School Hospital	Bunkyō, Tokyo	Japan
Naoya Masumori	Sapporo Medical University Hospital	Sapporo	Japan
Noboru Nakaigawa	Yokohama City University Hospital	Yokohama	Japan
Shintaro Narita	Akita University Hospital	Akita	Japan
Koshiro Nishimoto	Saitama Medical University International Medical Center	Hidaka	Japan
Wataru Obara	Iwate Medical University Hospital	Morioka	Japan
Mototsugu Oya	Keio University Hospital	Shinjuku, Tokyo	Japan
Chikara Oyama	Hirosaki University Hospital	Hirosaki	Japan
Naoto Sassa	Nagoya University Hospital	Nagoya	Japan
Nobuaki Shimizu	Gunma Prefectural Cancer Center	Ōta	Japan
Kazuhiro Suzuki	Gunma University Hospital	Maebashi	Japan
Kosuke Tochigi	Nagoya University Hospital	Nagoya	Japan
Yoshihiko Tomita	Niigata University Medical and Dental Hospital	Niigata	Japan
Hirotsugu Uemura	Kindai University Hospital	Osakasayama	Japan
Takahiro Yamaguchi	Kumamoto University Hospital	Kumamoto	Japan
Junji Yonese	The Cancer Institute Hospital of JFCR	Kōtō, Tokyo	Japan
Jinsoo Chung	National Cancer Center	Goyang	Republic of Korea
Jae Lyun Lee	Asan Medical Center	Seoul	Republic of Korea
Se Hoon Park	Samsung Medical Center	Seoul	Republic of Korea
Sun Young Rha	Severance Hospital, Yonsei University Health System	Seoul	Republic of Korea
Michiel van der Heijden	Nederlands Kanker Instituut	Amsterdam	the Netherlands
R. de Wit	Erasmus Medisch Centrum	Rotterdam	the Netherlands
Boguslawa Karaszewska	Przychodnia Lekarska KOMED	Konin	Poland
Jaroslaw Kolb-Sielecki	Samodzielny Publiczny Zespol Gruzlicy i Chorob Pluc	Olsztyn	Poland
Anna Kolodziej	Uniwersytecki Szpital Kliniczny UM we Wroclawiu	Wrocław	Poland
Dariusz Kucharczyk	Swietokrzyskie Centrum Onkologii	Kielce	Poland
Anna Lowczak	Samodzielny Publiczny Zespol Gruzlicy i Chorob Pluc	Olsztyn	Poland
Piotr Tomczak	Szpital Kliniczny Przemienienia Panskiego	Poznań	Poland
Bogdan Zurawski	Centrum Onkologii im. Prof. Franciszka Lukaszczyka	Bydgoszcz	Poland
Ana Castro	Centro Hospitalar Universitario do Porto	Porto	Portugal
Joana Febra	Centro Hospitalar Universitario do Porto	Porto	Portugal
Fabio Lopes	Hospital Beatriz Angelo	Loures	Portugal
Antonio Quintela	CHULN - Hospital Santa Maria	Lisbon	Portugal
Ana Raimundo	Hospital CUF Infante Santo	Lisbon	Portugal
Gabriela Sousa	IPO Coimbra	Coimbra	Portugal
Boris Alekseev	Moscow Scient. Res. Institute of Oncology n.a P.A. Hertzen	Moscow	Russia
Vagif Atduev	Volga District Med Center FMBA	Nizhny Novgorod	Russia
Adel Izmailov	Bashkir State Medical University	Ufa	Russia
Evgeny Kopyltsov	Clinical Oncological Dispensary of Omsk Region	Omsk	Russia
Ruslan Zukov	Krasnoyarsk Regional Clinical Oncology Dispensary	Krasnoyarsk	Russia

(continued on following page)

TABLE A1. FORT-1 Principal Investigators (continued)

Principal Investigator	Site Name	City/State/Region	Country
Chee Keong Toh	National Cancer Center Singapore	Singapore	Singapore
Alvin Seng Cheong Wong	National University Hospital	Singapore	Singapore
Marek Brezovsky	UROEXAM, spol. s r.o.	Nitra	Slovakia
Patrik Palacka	Národný onkologický ústav	Bratislava	Slovakia
Maria Reckova	POKO Poprad s.r.o.	Poprad	Slovakia
Teresa Alonso Gordoa	Hospital Ramón y Cajal	Madrid	Spain
Cristina Caballero Díaz	Hospital General Universitario de Valencia	Valencia	Spain
Joan Carles Galcerán	Ciutat Sanitària i Universitaria de la Vall d'Hebron	Barcelona	Spain
Daniel Ernesto Castellano Gauna	Hospital Universitario 12 de Octubre	Madrid	Spain
Ricardo Collado Martín	Hospital San Pedro de Alcántara	Cáceres	Spain
Albert Font	Institut Català d'Oncologia Badalona	Badalona	Spain
Xavier García del Muro Solans	Institut Català d'Oncologia Hospitalet	L'Hospitalet de Llobregat	Spain
María del Carmen Garcías de Espana	Hospital Universitari Son Espases	Palma	Spain
Aránzazu González del Alba	Hospital Universitari Son Espases	Palma	Spain
María José Juan Fita	Instituto Valenciano de Oncología	Valencia	Spain
María Méndez Vidal	Hospital Reina Sofía	Cordova	Spain
Rafael Morales-Barrera	Ciutat Sanitària i Universitaria de la Vall d'Hebron	Barcelona	Spain
Alejo Rodríguez-Vida	Hospital del Mar	Barcelona	Spain
María Sáez Medina	Hospital Virgen de la Victoria	Málaga	Spain
Agneta Holm	Södersjukhuset	Stockholm	Sweden
Serafeim Theodoroglou	Södersjukhuset	Stockholm	Sweden
Anders Ullén	Karolinska Institutet	Stockholm	Sweden
Richard Cathomas	Kantonsspital Graubünden	Chur	Switzerland
Pirmin Haeuptle	Universitätsspital Basel	Basel	Switzerland
Aurelius Omlin	Kantonsspital St Gallen	St Gallen	Switzerland
Sabine Schmid	Kantonsspital St Gallen	St Gallen	Switzerland
Frank Stenner	Universitätsspital Basel	Basel	Switzerland
Hsiao-Jen Chung	Taipei Veterans General Hospital	Taipei	Taiwan
Jian-Ri Li	Taichung Veterans General Hospital	Taichung	Taiwan
See-Tong Pang	Chang Gung Memorial Hospital at Linkou	Taoyuan	Taiwan
Wen-Pin Su	National Cheng Kung University Hospital	Tainan	Taiwan
Yu-Chieh Tsai	National Taiwan University Hospital	Taipei	Taiwan
Vincent Khoo	Royal Marsden Hospital (London)	London	United Kingdom
Isabel Syndikus	Clatterbridge Center for Oncology	Bebington	United Kingdom
Leonard Appleman	University of Pittsburgh	Pittsburgh, PA	United States
Hani Babiker	University of Arizona Cancer Center	Tucson, AZ	United States
Manojkumar Bupathi	Rocky Mountain Cancer Centers	Littleton, CO	United States
Arvind Chaudhry	Summit Cancer Center	Spokane, WA	United States
William Clark	Alaska Clinical Research Center, LLC	Anchorage, AK	United States
Jorge Darcourt	Houston Methodist Hospital	Houston, TX	United States
Stephen Dyar	Bon Secours St Francis Hospital	Greenville, SC	United States
Johnpaul Flores	Virginia Mason Medical Center	Seattle, WA	United States
Mukul Gupta	Sansum Clinic	Santa Barbara, CA	United States

(continued on following page)

TABLE A1. FORT-1 Principal Investigators (continued)

Principal Investigator	Site Name	City/State/Region	Country
Sharad Jain	Texas Oncology-Denton South	Denton, TX	United States
Daniel Landau	UF Cancer Center at Orlando Health	Orlando, FL	United States
Chong-xian Pan	UC Davis Comprehensive Cancer Center	Sacramento, CA	United States
Mamta Parikh	UC Davis Comprehensive Cancer Center	Sacramento, CA	United States
Rahul Parikh	University of Kansas Medical Center	Westwood, KS	United States
Anthony Pham	Compass Oncology	Tigard, OR	United States
David Quinn	University of Southern California	Los Angeles, CA	United States
Josh Simmons	Lewis Hall Singletary Oncology Center	Thomasville, GA	United States
Nicholas Vogelzang	Comprehensive Cancer Centers of Nevada	Las Vegas, NV	United States