Postprogression Outcomes in Patients With Ovarian Carcinoma Associated With a Mutation in a Non-*BRCA* Homologous Recombination Repair Gene Receiving Rucaparib Maintenance Treatment: Results From the Phase 3 Study ARIEL3

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Presenting Author Disclosures

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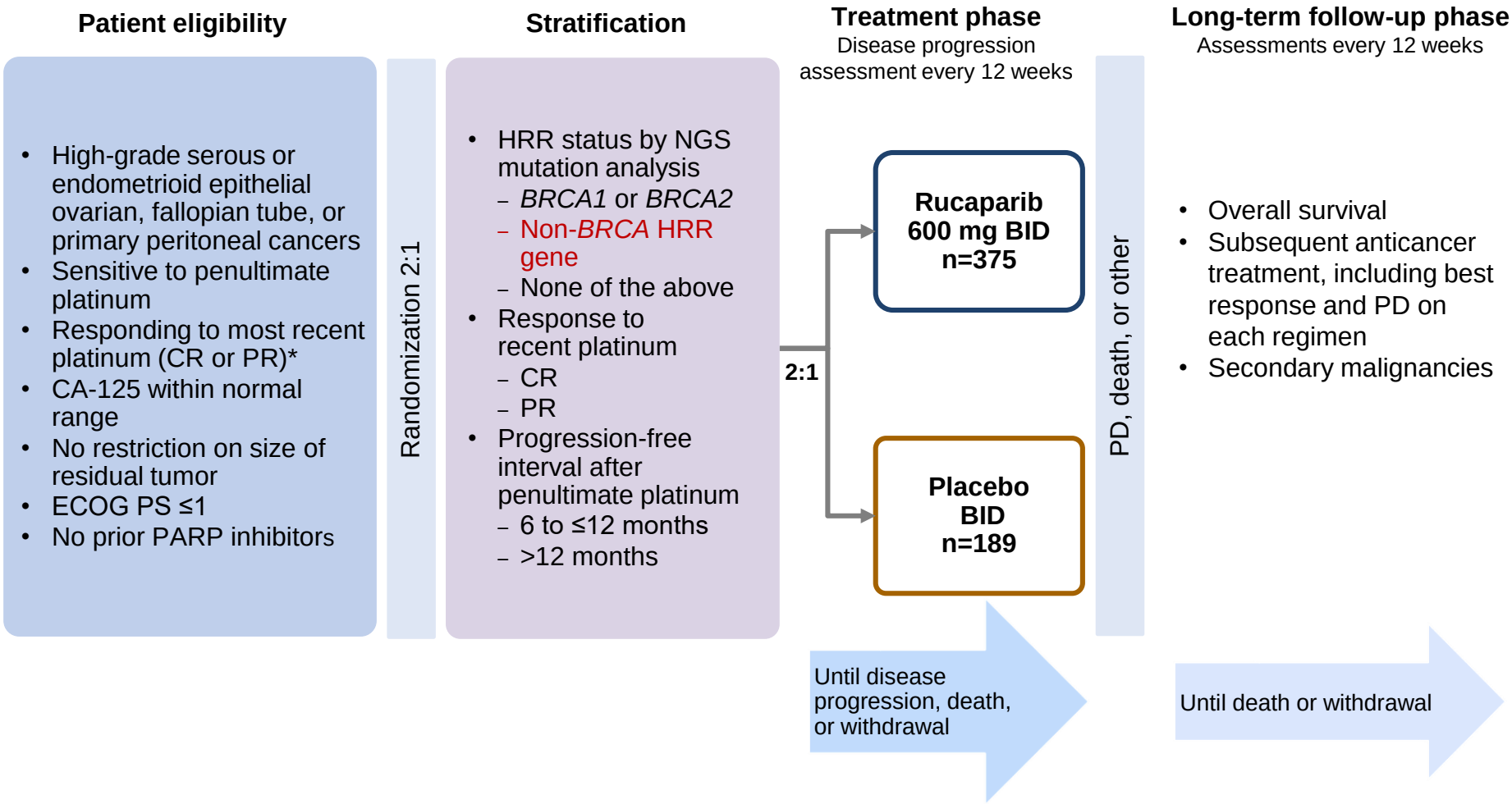
Introduction

- Maintenance therapy for patients with recurrent ovarian cancer is intended to extend PFS without compromising postprogression survival
- In the phase 3 ARIEL3 study (CO-338-014; NCT01968213), rucaparib maintenance treatment significantly improved PFS vs placebo in all predefined patient cohorts[1]
  - Greatest effects were seen in carcinomas deficient in HRR (eg, a mutation in *BRCA* or other HRR pathway gene, or high genomic LOH)[1]
- Here, we analyzed postprogression outcomes to evaluate the durability of the clinical benefit of rucaparib maintenance treatment following disease progression in the subgroup of patients with tumors associated with a mutation in a prespecified, non-*BRCA* HRR gene

HRR, homologous recombination repair; LOH, loss of heterozygosity; PFS, progression-free survival.

1. Coleman et al. *Lancet.* 2017;390:1949–1961.

ARIEL3 Study Design



*CR (defined by RECIST) or PR (defined by RECIST and/or a GCIG CA-125 response [CA-125 within normal range]) maintained until entry to ARIEL3 (≤8 weeks of last dose of chemotherapy).
BID, twice daily; CA-125, cancer antigen 125; CR, complete response; ECOG PS, Eastern Cooperative Oncology Group performance status; GCIG, Gynecological Cancer InterGroup; HRR, homologous recombination repair; NGS, next-generation sequencing; PARP, poly(ADP-ribose) polymerase; PD, progressive disease; PR, partial response; RECIST, Response Evaluation Criteria in Solid Tumors version 1.1.

1. Norquist et al. *JAMA Oncol.* 2016;2:482–90; 2. Domchek. *Cancer Discov.* 2017;7:937–39.

ARIEL3 Study Design

Patient eligibility

- High-grade serous or endometrioid epithelial ovarian, fallopian tube, or primary peritoneal cancers
- Sensitive to penultimate platinum
- Responding to r platinum (CR or
- CA-125 within n range
- No restriction or residual tumor
- ECOG PS ≤1
- No prior PARP

n 2:1

Stratification

- HRR status by NGS mutation analysis
  - *BRCA1* or *BRCA2*
  - Non-*BRCA* HRR gene
  - None of the above

Treatment phase
Disease progression assessment every 12 weeks

Rucaparib 600 mg BID n=375

other

Long-term follow-up phase
Assessments every 12 weeks

- Overall survival
- Subsequent anticancer treatment, including best response and PD on

alignancies

drawal

Prespecified Non-*BRCA* HRR Genes

ATM	*ATR*	*ATRX*	***BARD1***	*BLM*	***BRIP1***	*CHEK1*
CHEK2	*FANCA*	*FANCC*	*FANCD2*	*FANCE*	*FANCF*	*FANCG*
FANCI	*FANCL*	*FANCM*	*MRE11A*	*NBN*	***PALB2***	*RAD50*
RAD51	*RAD51B*	***RAD51C***	***RAD51D***	*RAD52*	*RAD54L*	*RPA1*

- Mutations in *BARD1*, *BRIP1*, *PALB2*, *RAD51C*, and *RAD51D* are significantly associated with hereditary ovarian cancer[1]

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FANCI	*FANCL*	*FANCM*	*MRE11A*	*NBN*	***PALB2***	*RAD50*
RAD51	*RAD51B*	***RAD51C***	***RAD51D***	*RAD52*	*RAD54L*	*RPA1*

- Mutations in *BARD1*, *BRIP1*, *PALB2*, *RAD51C*, and *RAD51D* are significantly associated with hereditary ovarian cancer[1]
- Mutations in *PALB2*, *RAD51C*, and *RAD51D* are causally associated with clinical sensitivity to PARP inhibitors[2]

*CR (defined by RECIST) or PR (defined by RECIST and/or a GCIG CA-125 response [CA-125 within normal range]) maintained until entry to ARIEL3 (≤8 weeks of last dose of chemotherapy).
BID, twice daily; CA-125, cancer antigen 125; CR, complete response; ECOG PS, Eastern Cooperative Oncology Group performance status; GCIG, Gynecological Cancer InterGroup; HRR, homologous recombination repair; NGS, next-generation sequencing; PARP, poly(ADP-ribose) polymerase; PD, progressive disease; PR, partial response; RECIST, Response Evaluation Criteria in Solid Tumors version 1.1.
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Genomic Characteristics of Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation in ARIEL3

Rucaparib-treated patients (n=28)

Pt	HRR gene	Mutation type	Mutation zygosity in tumor[a]	Genomic LOH status
1	*ATM*	Frameshift	Heterozygous	LOH low
2	*ATM*	Nonsense	NA	Unknown
3	*ATR*	Frameshift	Heterozygous	LOH low
4	*ATR*	Splice site	NA	LOH low
5	*BARD1*	Nonsense	Homozygous	LOH high
6	*CHEK2*	Splice site	Homozygous	LOH high
7	*CHEK2*	Deletion	Homozygous	LOH high
8	*FANCD2*	Nonsense	Heterozygous	LOH low
9	*FANCD2*	Splice site	NA	LOH low
10	*FANCI*	Frameshift	NA	LOH low
11	*FANCL*	Frameshift	Heterozygous	LOH high
12	*FANCL*	Frameshift	NA	LOH high
13	*FANCM*	Frameshift	NA	LOH low
14	*RAD50*	Frameshift	Heterozygous	LOH low
15	*RAD50*	Frameshift	NA	Unknown
16	*RAD51C*	Splice site	Homozygous	LOH high
17	*RAD51C*	Nonsense	Homozygous	LOH high
18	*RAD51C*	Frameshift	Homozygous	LOH high
19	*RAD51C*	Splice site	Homozygous	LOH high
20	*RAD51C*	Splice site	Homozygous	LOH high
21	*RAD51C*	Frameshift	Homozygous	LOH high
22	*RAD51D*	Nonsense	Homozygous	LOH high
23	*RAD51D*	Nonsense	Homozygous	LOH high
24	*RAD51D*	Frameshift	Homozygous	LOH high
25	*RAD51D*	Frameshift	Homozygous	LOH high
26	*RAD54L*	Frameshift	Heterozygous	LOH high
27	*RAD54L*	Nonsense	Heterozygous	LOH high
28	*RAD54L*	Frameshift	NA	Unknown

Placebo-treated patients (n=15)

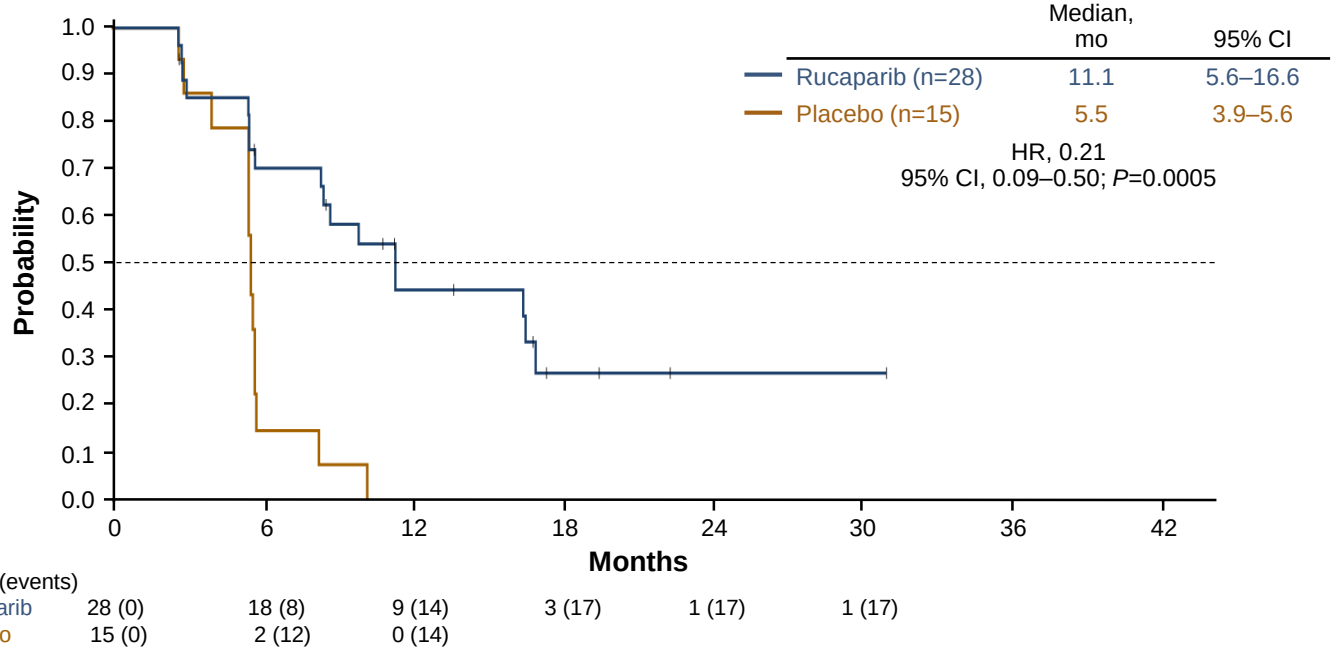
Pt	HRR gene	Mutation type	Mutation zygosity in tumor[a]	Genomic LOH status
29	*BRIP1*	Nonsense	Homozygous	LOH low
30	*BRIP1*	Nonsense	Homozygous	LOH high
31	*BRIP1*	Nonsense	Homozygous	LOH high
32	*BRIP1*	Nonsense	Homozygous	LOH low
33	*BRIP1*	Frameshift	Homozygous	LOH low
34	*FANCA*	Splice site	NA	LOH high
35	*FANCC*	Frameshift	Heterozygous	LOH low
36	*FANCD2*	Frameshift	Heterozygous	LOH low
37	*FANCE*	Frameshift	Homozygous	LOH low
38	*FANCF*	Frameshift	NA	Unknown
39	*RAD50*	Splice site	NA	LOH low
40	*RAD51C*	Nonsense	Homozygous	LOH high
41	*RAD51C*	Deletion	Homozygous	LOH low
42	*RAD51D*	Nonsense	Homozygous	LOH high
43	*RAD54L*	Splice site	Heterozygous	LOH low

Color code

RAD51C/D	Homozygous	LOH high

[a]Based on Foundation Medicine sequencing results, in which a tumor is classified as homozygous if both copies in the tumor carry the mutant allele and heterozygous if both the wild-type and mutant alleles are present.
HRR, homologous recombination repair; LOH, loss of heterozygosity; NA, not available; Pt, patient.

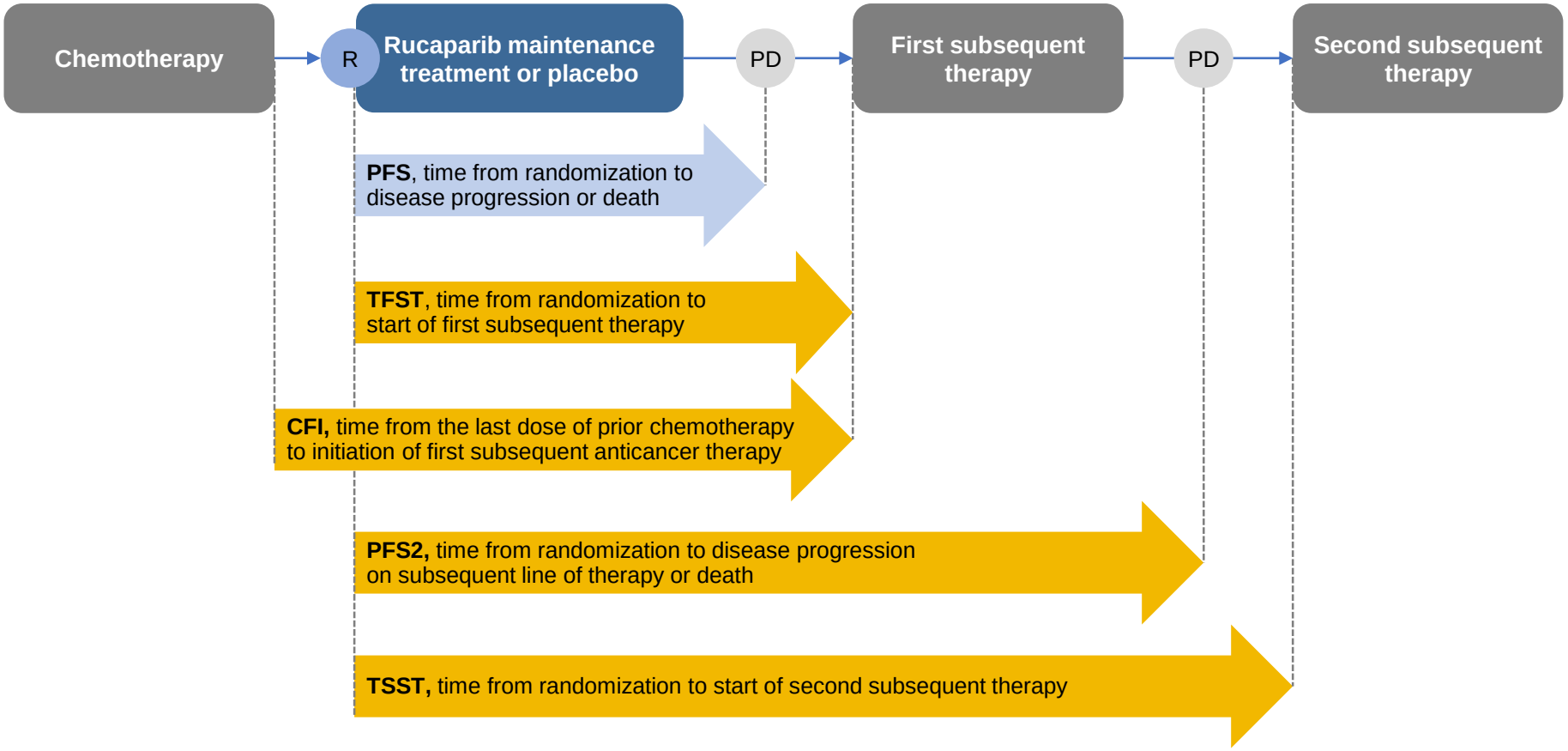
Initial Results: PFS and Safety from ARIEL3 in Patients With Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation



- The safety profile of rucaparib in patients with a carcinoma associated with a non-*BRCA* HRR gene mutation was consistent with the overall safety population
  - In the non-*BRCA* HRR gene mutation subgroup vs the overall population, incidence of grade ≥3 AEs and AEs leading to dose reduction and/or treatment interruption of rucaparib were 55.6% vs 59.7% and 66.7% vs 71.8%, respectively

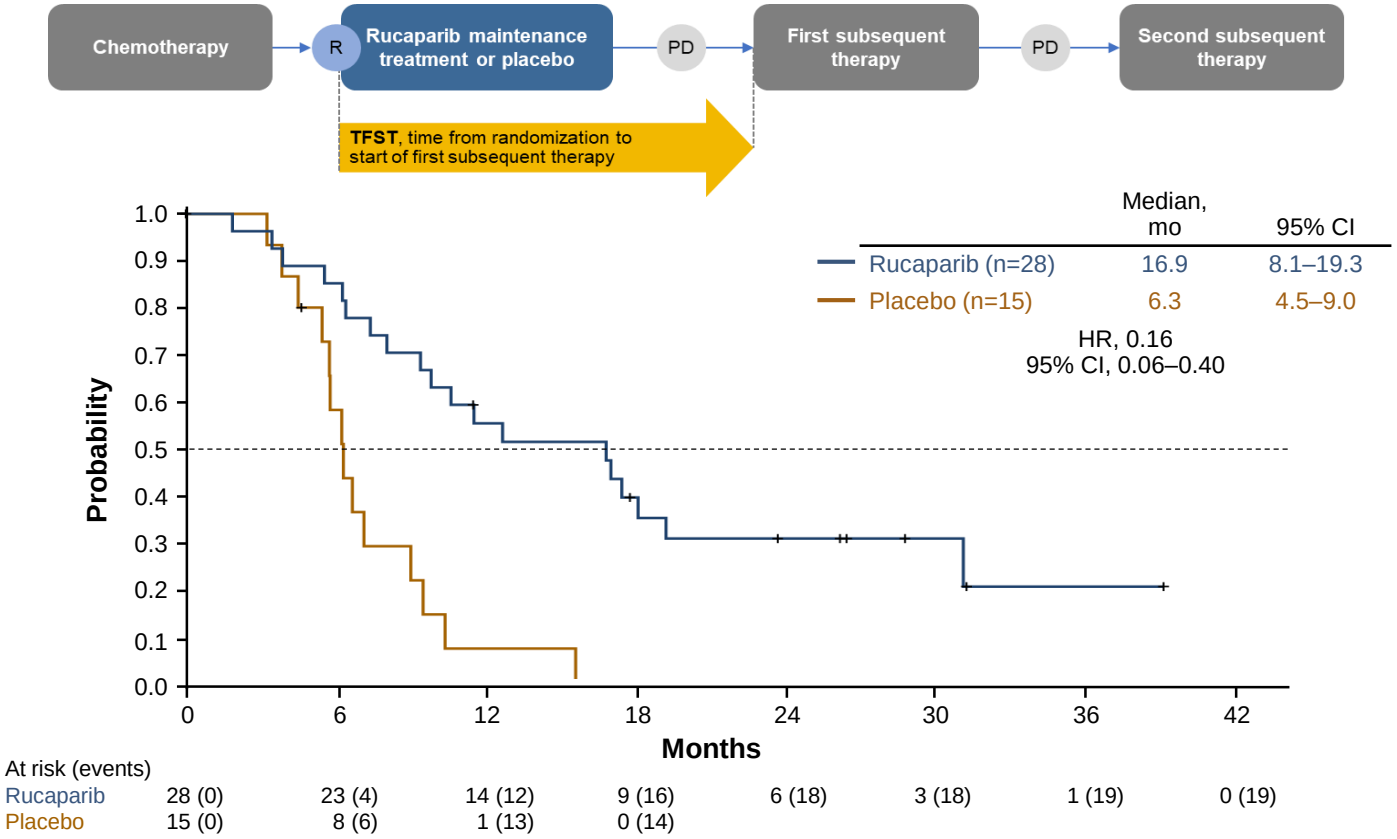
O'Malley et al. *Mol Cancer Ther.* 2018;17(1 suppl):abst LB-A12.
Visit cutoff date for PFS April 15, 2017; visit cutoff date for safety December 31, 2017.
AE, adverse event; HR, hazard ratio; HRR, homologous recombination repair; PFS, progression-free survival.

Schema Comparing Different Efficacy Endpoints



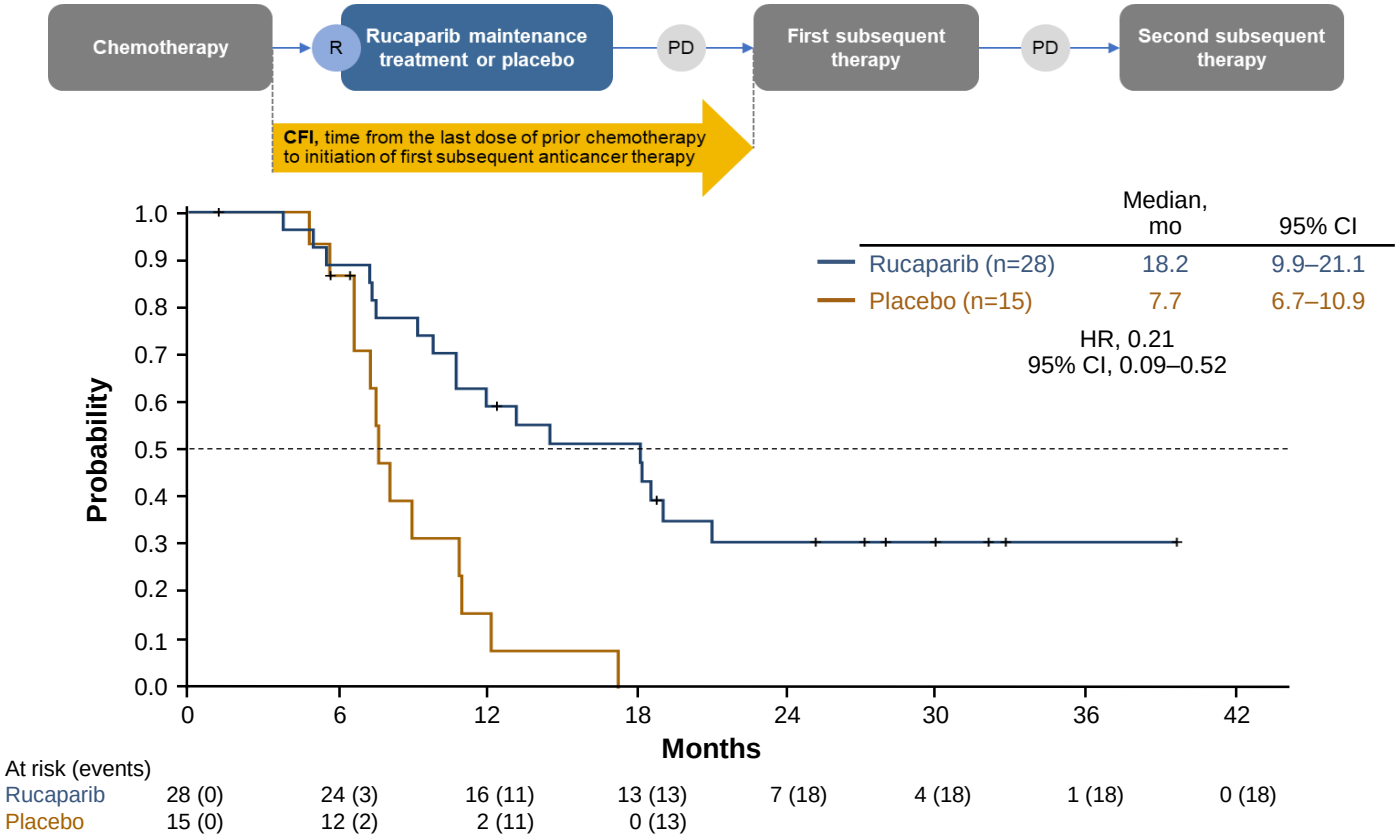
PD, progressive disease; R, randomization.

Patients With Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation: Time to First Subsequent Therapy



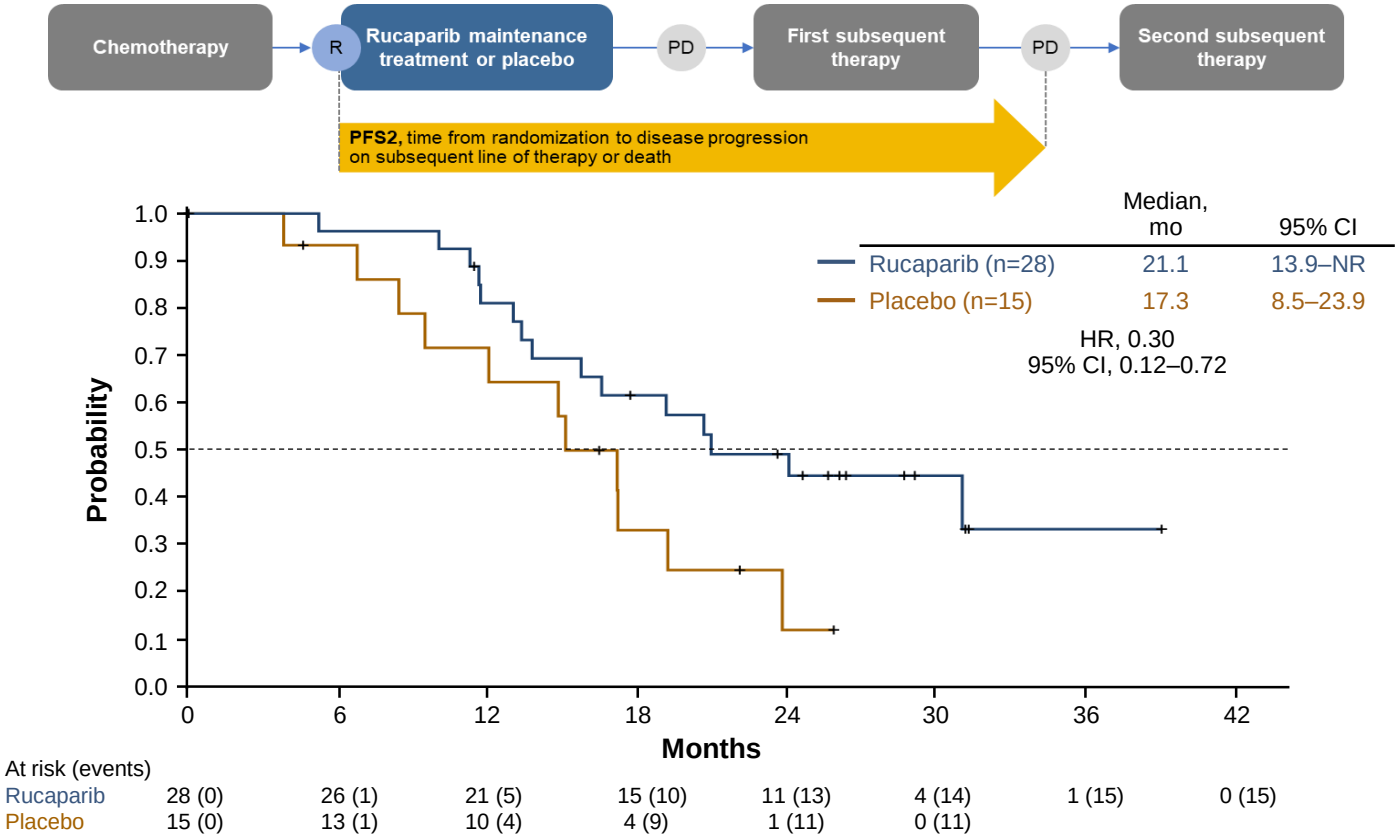
Visit cutoff date December 31, 2017.
HR, hazard ratio; PD, progressive disease; R, randomization; TFST, time to first subsequent therapy.

Patients With Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation: Chemotherapy-Free Interval



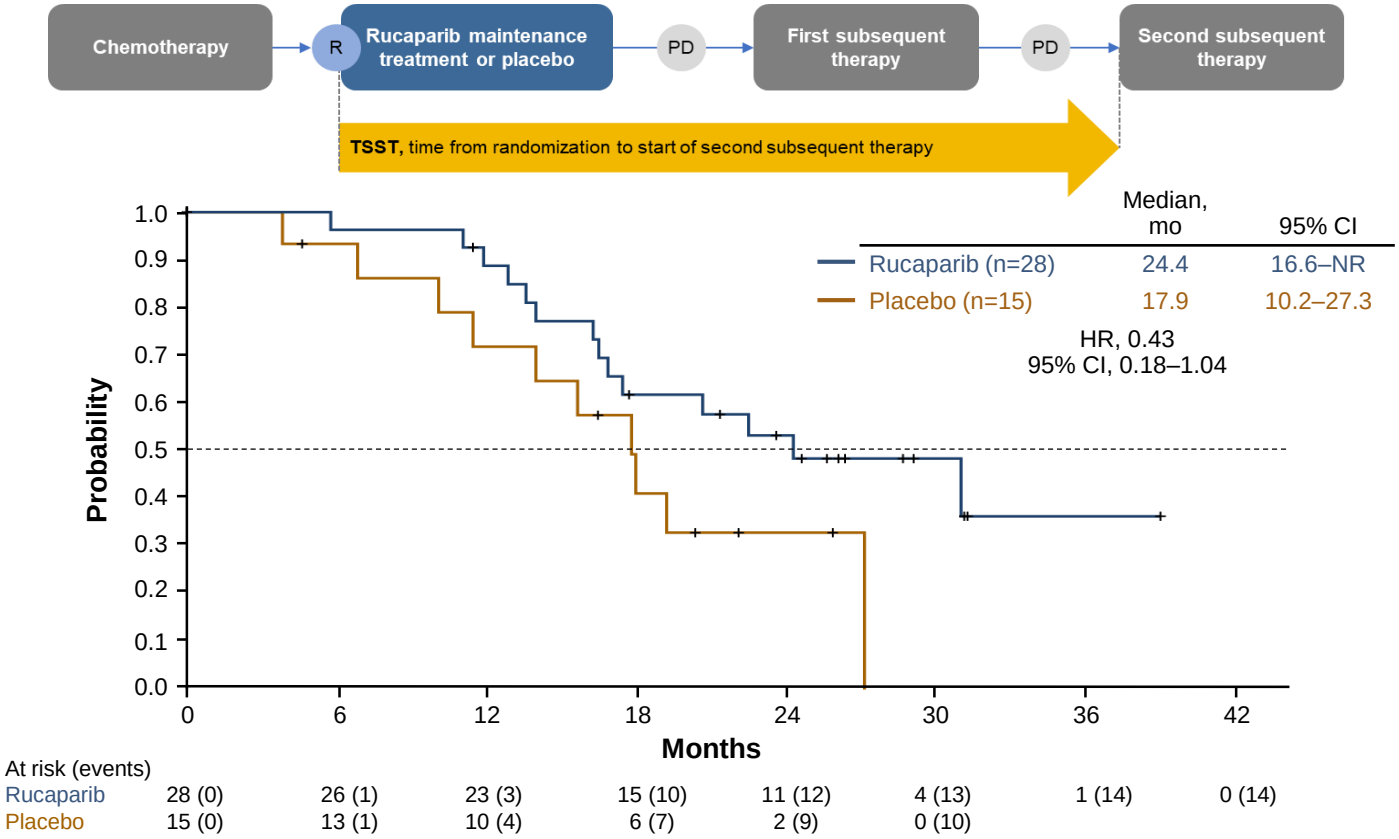
Visit cutoff date December 31, 2017.
CFI, chemotherapy-free interval; HR, hazard ratio; PD, progressive disease; R, randomization.

Patients With Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation: Time to Disease Progression on Subsequent Therapy or Death



Visit cutoff date December 31, 2017.
HR, hazard ratio; HRR, homologous recombination repair; NR, not reached; PD, progressive disease; PFS2, time to disease progression on subsequent therapy or death; R, randomization.

Patients With Carcinomas Associated With a Non-*BRCA* HRR Gene Mutation: Time to Second Subsequent Therapy



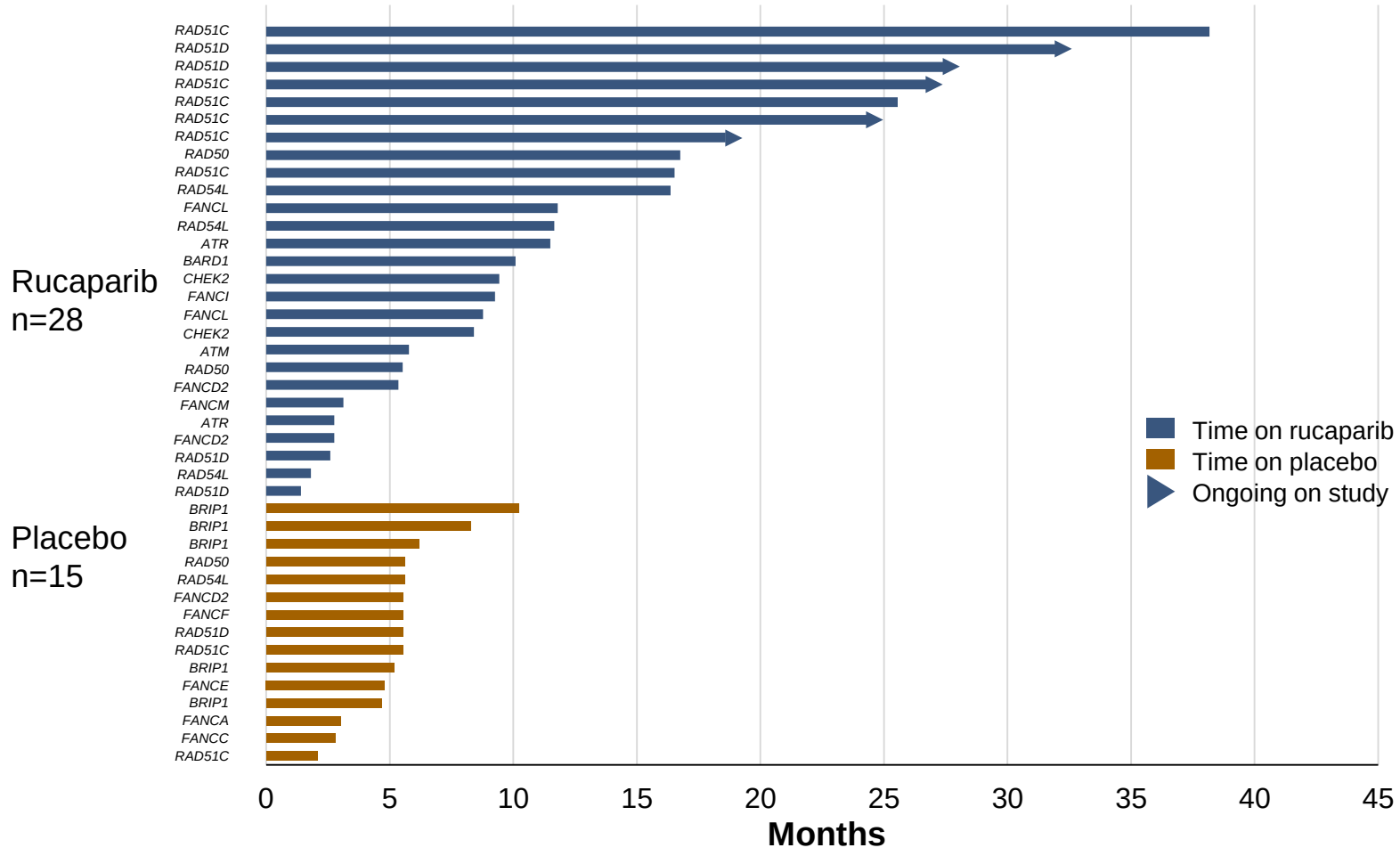
Visit cutoff date December 31, 2017.
HR, hazard ratio; HRR, homologous recombination repair; NR, not reached; PD, progressive disease; R, randomization; TSST, time to second subsequent therapy.

Summary of Postprogression Outcomes in Patients With Carcinomas Associated With a *BRCA* or Non-*BRCA* HRR Gene Mutation

	Non-*BRCA* HRR mutation		*BRCA* mutation	
	Rucaparib (n=28)	Placebo (n=15)	Rucaparib (n=130)	Placebo (n=66)
TFST				
Median, mo	16.9	6.3	18.9	7.2
HR (95% CI)	0.16 (0.06–0.40)		0.28 (0.20–0.41)	
CFI				
Median, mo	18.2	7.7	20.8	8.7
HR (95% CI)	0.21 (0.09–0.52)		0.28 (0.19–0.41)	
PFS2				
Median, mo	21.1	17.3	26.8	18.4
HR (95% CI)	0.30 (0.12–0.72)		0.56 (0.38–0.83)	
TSST				
Median, mo	24.4	17.9	28.8	17.7
HR (95% CI)	0.43 (0.18–1.04)		0.53 (0.36–0.80)	

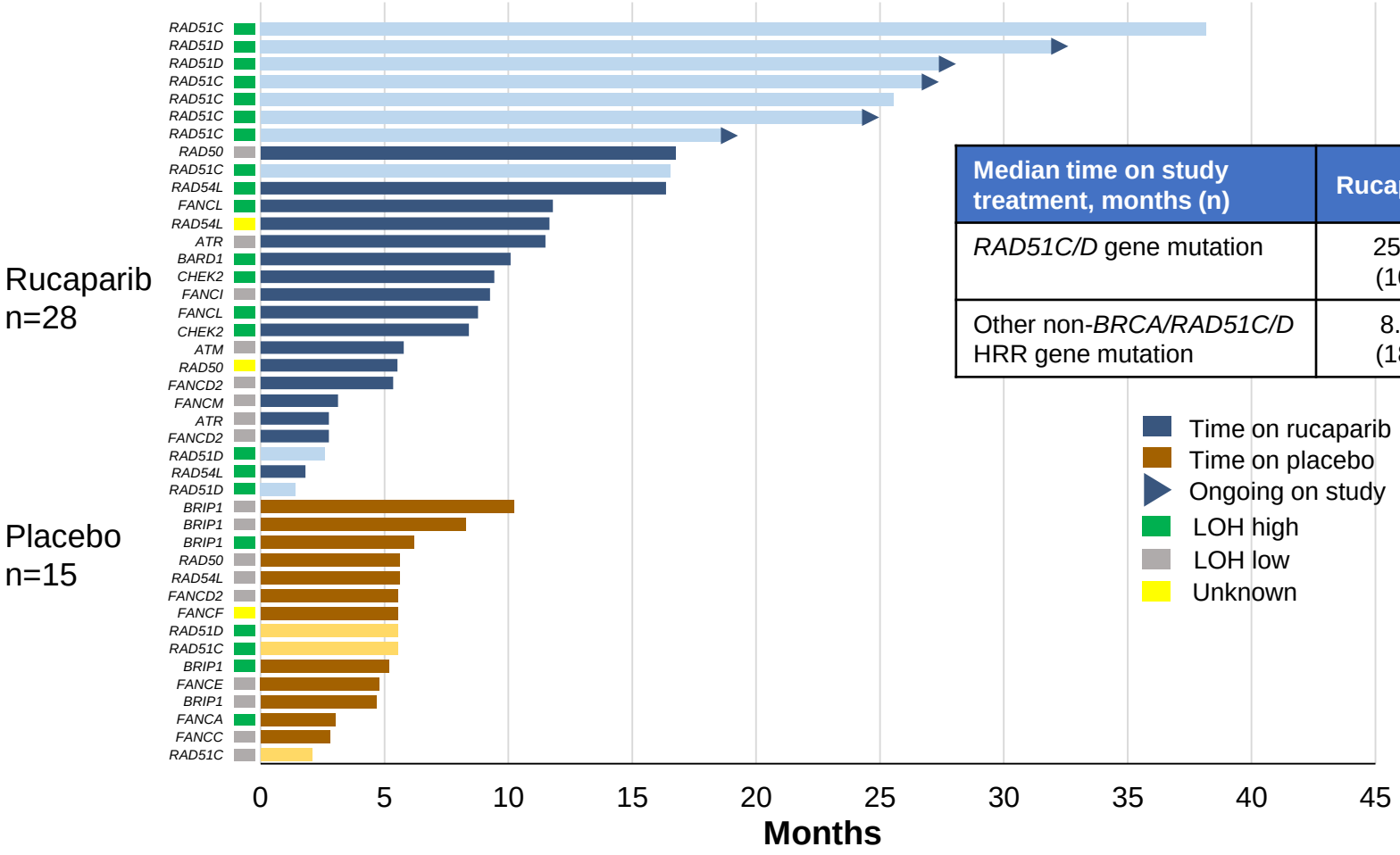
Visit cutoff date December 31, 2017.
HRs estimated with a Cox proportional hazards model.
CFI, chemotherapy-free interval; HR, hazard ratio; HRR, homologous recombination repair; PFS2, time to disease progression on subsequent therapy or death; TFST, time to first subsequent therapy; TSST, time to second subsequent therapy.

Time on Study Treatment: Patients With Carcinomas Associated With a Non-*BRCA* HRR Mutation



Visit cutoff date December 31, 2017.
HRR, homologous recombination repair.

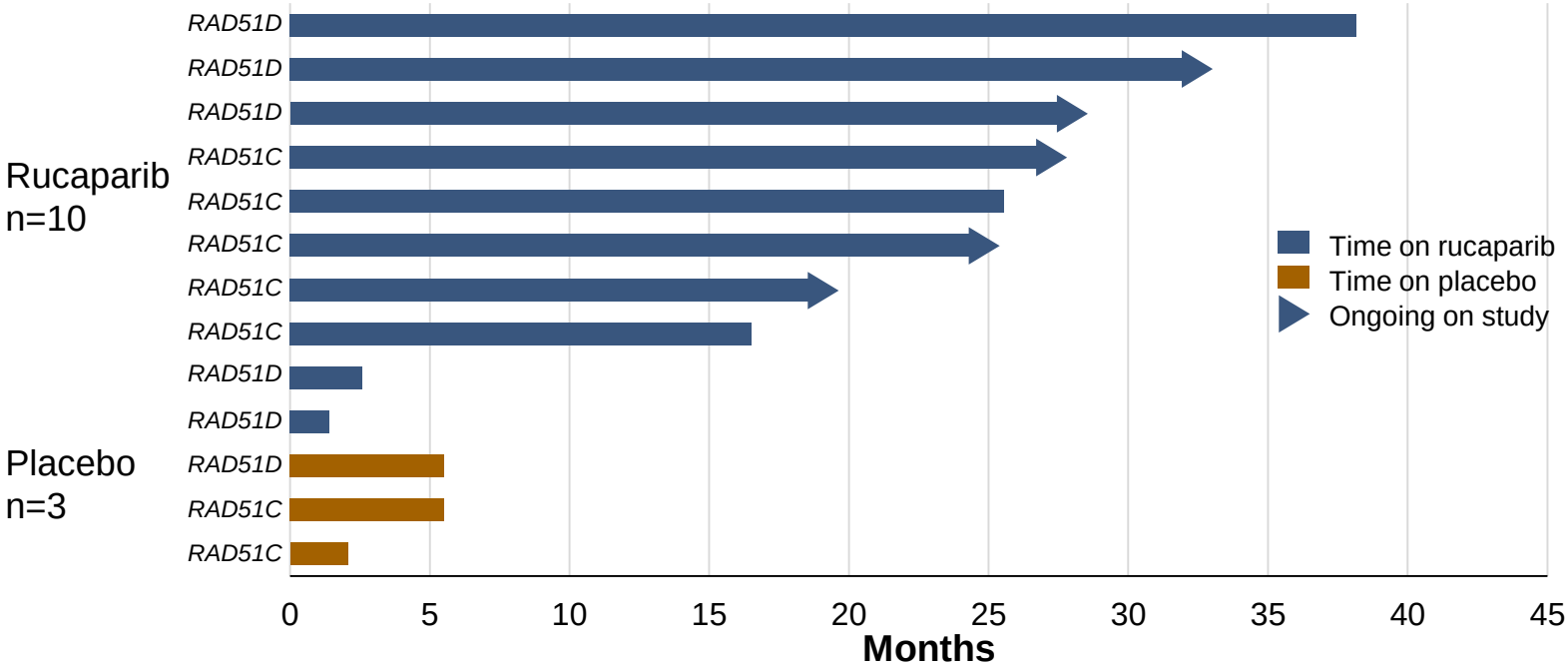
Time on Study Treatment: Patients With Carcinomas Associated With a Non-*BRCA* HRR Mutation



Median time on study treatment, months (n)	Rucaparib	Placebo
RAD51C/D gene mutation	25.0 (10)	5.5 (3)
Other non-*BRCA/RAD51C/D* HRR gene mutation	8.6 (18)	5.5 (12)

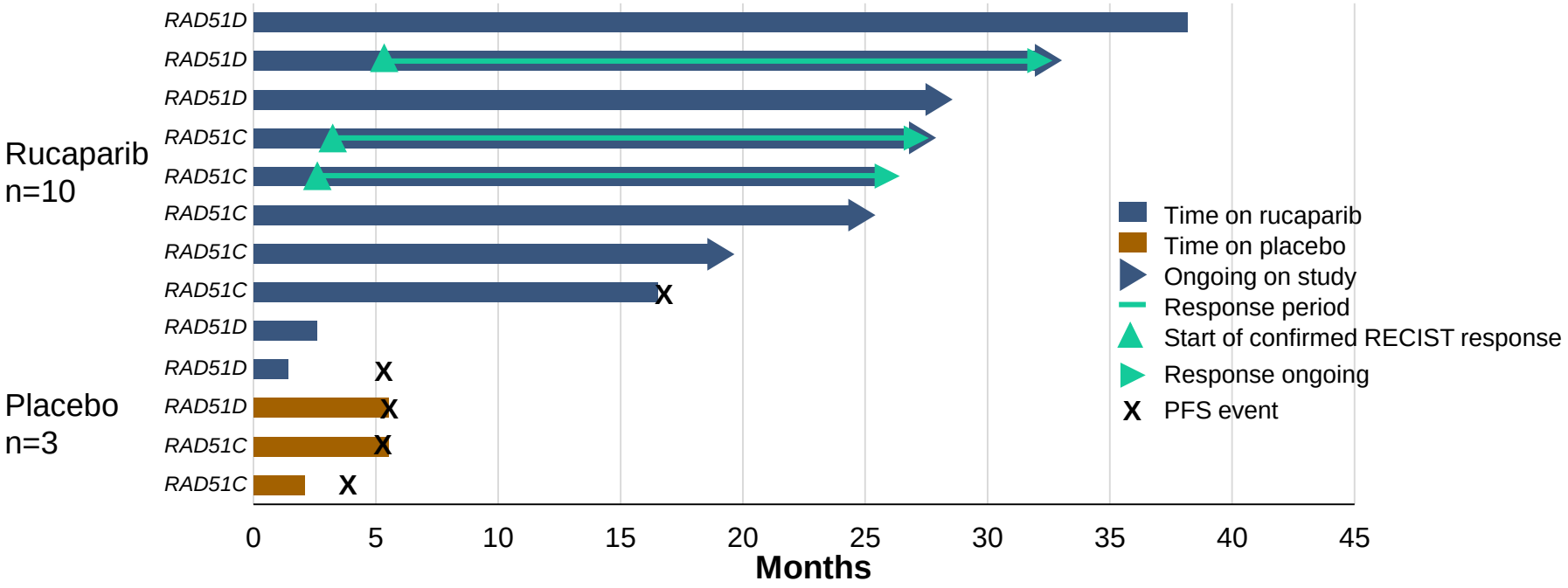
Visit cutoff date December 31, 2017.
HRR, homologous recombination repair; LOH, loss of heterozygosity.

Time on Study Treatment: Patients With Carcinomas Associated With a *RAD51C/D* Mutation



Visit cutoff date December 31, 2017.
HRR, homologous recombination repair; PFS, progression-free survival.

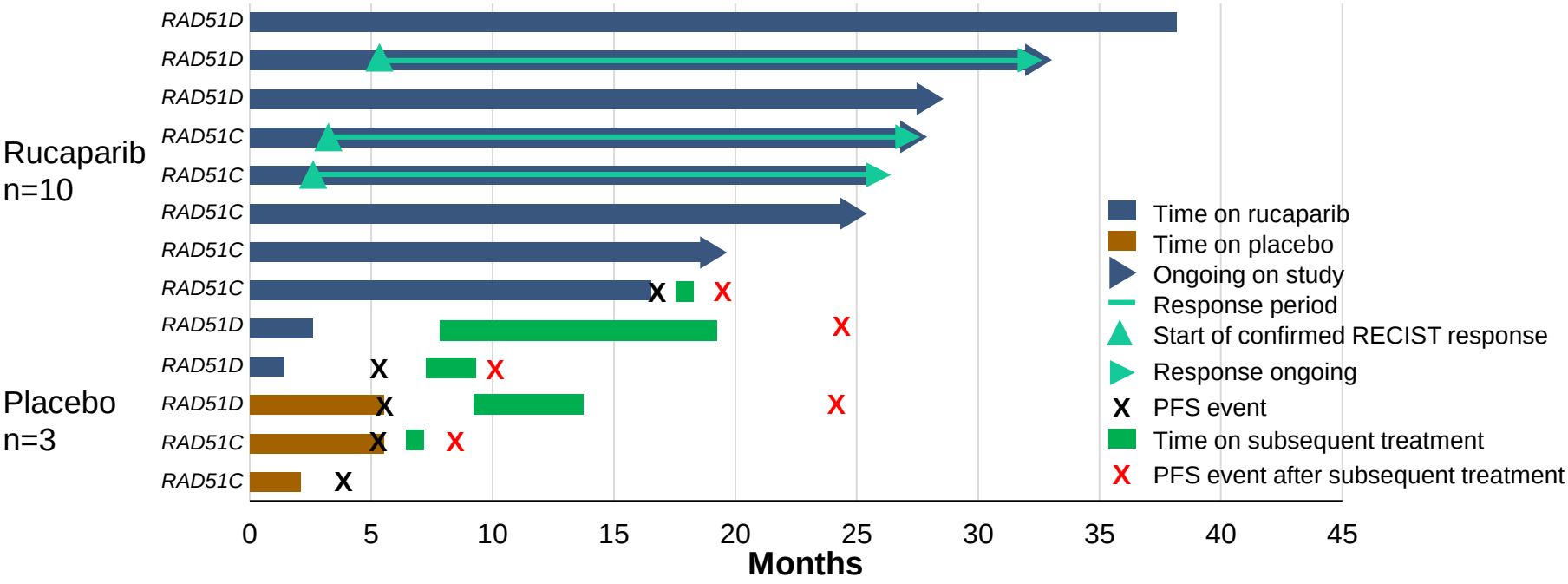
Time on Study Treatment: Patients With Carcinomas Associated With a *RAD51C/D* Mutation



- Three patients with a *RAD51C/D* mutation had measurable disease at baseline, and all 3 achieved a confirmed response with rucaparib treatment (1 complete response and 2 partial responses)

Visit cutoff date December 31, 2017.
PFS, progression-free survival; RECIST, Response evaluation criteria in solid tumors version 1.1.

Time on Study Treatment: Patients With Carcinomas Associated With a *RAD51C/D* Mutation



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Conclusions

- Although the number of patients in this subgroup was small, rucaparib improved the clinically meaningful postprogression endpoints TFST, CFI, PFS2, and TSST vs placebo in patients with platinum-sensitive, recurrent ovarian cancer harboring a non-*BRCA* HRR gene mutation
    - Prior rucaparib treatment did not adversely impact the possibility for patients in this subgroup to benefit from subsequent therapy
- Mutations in a subset of HRR genes, such as *RAD51C/D*, may confer greater sensitivity to PARP inhibitor treatment

CFI, time from the last dose of prior chemotherapy to initiation of first subsequent anticancer therapy; HRR, homologous recombination repair; PARP, poly(ADP-ribose) polymerase; PFS2, time from randomization to disease progression on subsequent line of therapy or death; TFST, time from randomization to start of first subsequent therapy; TSST, time from randomization to start of second subsequent therapy.

Acknowledgments

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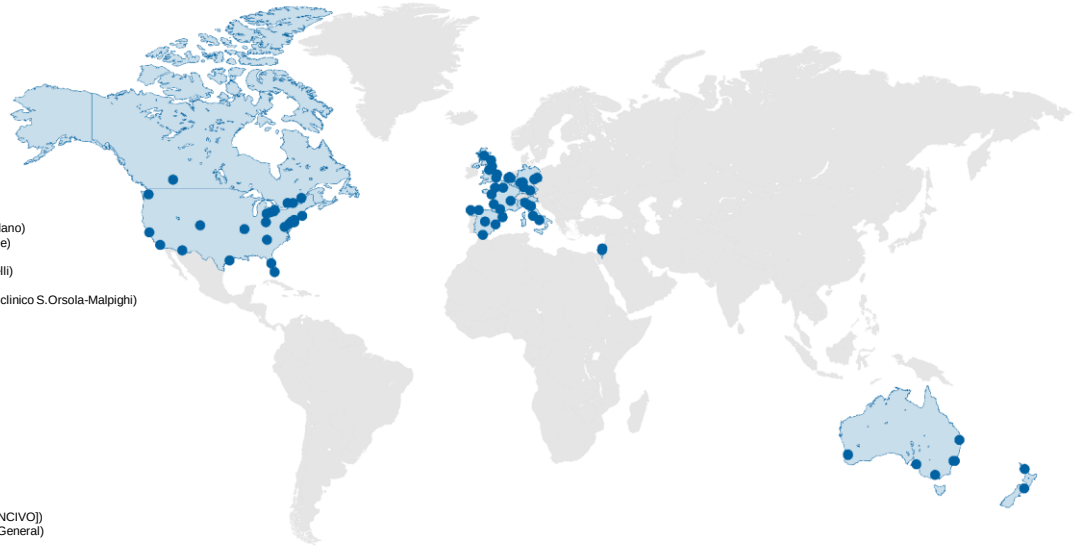

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