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Trodelvy® (sacituzumab govitecan-hziy) Pharmacokinetics

This document is in response to your request for information regarding the pharmacokinetics (PK) of Trodelvy® (sacituzumab govitecan-hziy [SG]).

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Summary

Relevant Product Labeling[1]

Based on popPK analysis, steady state volume of distribution of SG is 3.6 L, and the estimated mean (%CV) clearance of SG is 0.13 L/h (12%). The median elimination $t_{½}$ of SG and free SN-38 in patients with mTNBC was 23.4 and 17.6 hours, respectively. SN-38 is metabolized via *UGT1A1*.

SG PK Data

In the IMMU-132-01 study among patients who received SG 8 mg/kg or 10 mg/kg, ~90% of SN-38 (the active metabolite of irinotecan) was released from the ADC over 3 days.[2]

Analyses of the IMMU-132-01, TROPHY-U-01, and ASCENT studies showed no accumulation of SG or free SN-38 after multiple treatment cycles, and exposures of SG and free SN-38 were comparable across the studies.[3] There was no effect on the PK of SG or free SN-38 when SG was administered concomitantly with UGT1A1 inhibitors or inducers; *UGT1A1* GT was not identified as a significant covariate for AUC or C_{max}.[4]

Analyses of the IMMU-132-01, ASCENT and TROPiCS-02 studies demonstrated no clinically relevant changes in SG exposure in the first treatment cycle, with covariates that included mild to moderate renal impairment, mild hepatic impairment, age, sex, albumin level, race, ECOG PS, tumor type, *UGT1A1* GT and Trop-2 expression.[5] Increased $CAVG_{SG}$ ohwas associated with an increased probability of developing any-grade diarrhea, neutropenia, nausea, vomiting, and hypersensitivity.[6]

Exposure-response analyses of patients with mBC in IMMU-132-01, ASCENT and TROPiCS-02 demonstrated that higher $CAVG_{tAB}$ values were associated with longer PFS and OS; higher $CAVG_{SG}$ values were associated with an increased probability of CBR[6], CR, and ORR.[6,7] In patients with mTNBC (N=277), the probability of any-grade vomiting, diarrhea, nausea and neutropenia significantly increased with increasing $CAVG_{SG}$; neutropenia was the only evaluated AE for which $CAVG_{SG}$ was significantly associated with a Grade ≥3 event.[7] In patients with mBC (N=569), the probability of any-grade diarrhea, neutropenia, nausea, vomiting, and hypersensitivity was significantly increased with

increasing $CAVG_{SG}$. Increased $CAVG_{SG}$ was also significantly associated with increased probability of Grade ≥3 neutropenia and febrile neutropenia.[6]

Relevant Product Labelling[1]

PK

The serum PK of SG and SN-38 were evaluated in patients with mBC who received SG as a single agent at a dose of 10 mg/kg. The PK parameters of SG and free SN-38 are presented in Table 1.

Table 1. Summary of Mean PK Parameters of SG and Free SN-38[1a]

PK Parameter	SG (N=693)	Free SN-38 (N=681)
C_{max}, mean (CV%), ng/mL	239,000 (11)	98 (45)
$AUC_{0\text{-}168h}$, mean (CV%), ng·h/mL	5,640,000 (22)	3696 (56)

[a]Parameters estimated based on popPK analyses.

Distribution

Based on popPK analysis, steady state volume of distribution of SG is 3.6 L.

Elimination

The median elimination $t_{½}$ of SG and free SN-38 in patients with mTNBC was 23.4 and 17.6 hours, respectively. Based on popPK analysis, the estimated mean (%CV) clearance of SG is 0.13 L/h (12%).

Metabolism

No metabolism studies with SG have been conducted. SN-38 is metabolized via *UGT1A1*. The glucuronide metabolite of SN-38 was detectable in the serum of patients.

Specific populations

PK analyses in patients treated with SG did not identify an effect of age (27–88 years), race (White, Black, or Asian), or mild renal impairment to moderate renal impairment (CrCl 30–89 mL/min) on the PK of SG. Renal elimination is known to contribute minimally to the excretion of SN-38, the small molecule moiety of SG. There are no data on the PK of SG in patients with severe renal impairment (CrCl 15–29 mL/min) or end-stage renal disease (CrCl <15 mL/min).

Patients with hepatic impairment

The exposure of SG is similar in patients with mild hepatic impairment (total bilirubin ≤ULN with AST >ULN, or bilirubin >1 to <1.5 × ULN with any AST; n=257) to patients with normal hepatic function (total bilirubin or AST <ULN; n=526).

SG and free SN-38 exposures are unknown in patients with moderate (total bilirubin >1.5 to 3 × ULN) or severe (total bilirubin >3 × ULN) hepatic impairment.

Drug interaction studies

No drug-drug interaction studies were conducted with SG or its components. Inhibitors or inducers of UGT1A1 may increase or decrease SN-38 exposure, respectively.

SG PK Data

PK Profile of SG After Multiple Cycles of SG 8 or 10 mg/kg[2]

In IMMU-132-01, the PK profile was analyzed with SG 8 mg/kg (n=81) and 10 mg/kg (n=97) in patients with metastatic epithelial cancers (including HR+/HER2- mBC, mTNBC, and mUC). Median total SN-38 levels in the serum of the 10 mg/kg group were 4234 ng/mL at 30 min and 1334 ng/mL at Day 1. Free serum SN-38 levels were 95.3 ng/mL at 30 min and 56.9 ng/mL at Day 1. The AUC for free SN-38 comprised ~2.5% of the total SN-38, which indicated that most of the serum SN-38 was bound to IgG. Approximately 90% of SN-38 was gradually released from the ADC over 3 days. The $t_{1/2}$ of SG was ~11 to 14 h, which reflected the release of SN-38 from the conjugate. The monoclonal antibody was cleared more slowly ($t_{1/2}$: ~103 to 114 h).

PK Profile of SG Using Concentration-Time Data

Concentration-time profiles of SG, free SN-38, tAB, total SN-38, and the glucuronide metabolite of free SN-38, were evaluated in IMMU-132-01 (N=275), TROPHY-U-01 (N=131), and ASCENT (N=252) in patients with various metastatic epithelial cancers, mUC and mTNBC, respectively; SG 10 mg/kg was administered on Days 1 and 8 of a 21-day treatment cycle. After multiple treatment cycles, no accumulation of SG or free SN-38 was seen and the exposures of SG and free SN-38 were comparable (Table 2). Mean clearance of SG was 0.14 to 0.16 L/h; steady-state volume of distribution was 2.45 to 2.82 L.[3]

Table 2. PK Parameters After the First Dose of SG 10 mg/kg[3,8]

Study (n)		PK Parameter					
		C_{max}, mean (%CV), ng/mL	T_{max}, median (range), h	$AUC_{0–168\ h}$, mean (%CV), ng·h/mL	$t_{½}$, median (range), h	V_{ss},[a] L	C_L,[a] L/h
IMMU-132-01	SG (120)	227,000 (24)	3.03 (1.1–23.3)	5,190,000 (24)	14.7 (12.1–27.5)	2820	149
	Free SN-38 (95)	120 (82)	3.58 (1.5–6.7)	3620 (72)	16.9 (11.5–30.3)	6900	270,000
	tAB (5)	252,000 (27)	3.42 (2.5–24.9)	21,200,000 (21.4)	63.1 (57.4–83.4)	2.41	26.7
TROPHY-U-01	SG (9)	224,000 (23)	3.52 (1.6–4.6)	5,270,000 (31)	14 (12–15.4)	–	–
	Free SN-38 (8)	67.3 (44)	4.6 (3–7.2)	1970 (37)	16.2 (13.6–23.4)	–	–
	tAB (9)	228,000 (23.8)	4.67 (3–21.3)	21,000,000 (32.2)	122 (56.1–279)	–	–
ASCENT	SG (28)	240,000 (22)	3.09 (1.2–5.4)	5,340,000 (24)	14.7 (8.83–24.7)	–	–
	Free SN-38 (27)	90.6 (65)	3.25 (1.2–6.3)	2730 (41)	17.6 (11.1–44)	–	–
	tAB (7)	281,000(39.1)	3.07 (2.6–6.1)	18,100,000 (20.5)	60.1 (9.05–97.6)	–	–

Abbreviations: C_L=total body clearance; CV=coefficient of variation; T_{max}=time to maximum concentration; V_{ss}=apparent volume of distribution at steady state following IV administration.
[a]Measure (eg, mean, median) was not specified in the source.

Effect of *UGT1A1* Polymorphisms on the PK of SG[4]

Among patients (N=558) with available *UGT1A1* GT and exposure data in IMMU-132-01, TROPHY-U-01, and ASCENT, 42% were homozygous for the wild type allele (**1/*1*), 32% were heterozygous (**1/*28*), and 14% were homozygous (**28/*28*). No effects on the PK

parameters of SG or free SN-38 were detected when SG was administered concomitantly with UGT1A1 inhibitors or inducers.

In the popPK model, *UGT1A1* GT was not a significant covariate for AUC or C_{max} at the first treatment cycle for SG or free SN-38 (Table 3); the median values of $AUC_{0\text{-}168\ h}$ for SG and free SN-38 were also similar across different *UGT1A1* GTs.

Table 3. Estimated Exposure Across *UGT1A1* GTs in the PopPK Model (Cycle 1)[4]

GT	SG, Cycle 1		Free SN-38, Cycle 1	
	AUC, mean (SD), μg·h/mL	C_{max}, mean (SD), μg/mL	AUC, mean (SD), μg·h/mL	C_{max}, mean (SD), μg/mL
**1/*1*	9790 (2110)	230 (41.7)	5.39 (2.78)	0.0874 (0.0348)
**1/*28*	9480 (2300)	223 (41.2)	5.25 (2.54)	0.0891 (0.0291)
**28/*28*	9370 (2250)	235 (42.9)	4.82 (2.63)	0.088 (0.0388)
Other	9640 (2290)	223 (39.3)	4.43 (2.35)	0.0747 (0.0275)

Effect of Covariates on the PopPK of SG[5]

In IMMU-132-01, ASCENT, and TROPiCS-02, no clinically relevant impact on SG exposure in the first treatment cycle was observed in 789 patients with mBC or other epithelial cancers and mild to moderate renal impairment, mild hepatic impairment, tumor type, UGT1A1 GT, use of UGT1A1 inducers or inhibitors, or Trop 2 expression (Tables 4 and 5).

Table 4. Categorical Covariate Relationships to SG Exposure Relative to Reference[a5]

Covariate (Reference [n])	Covariate (n)	Predicted SG Exposure (90% CI)	
		AUC	C_{max}
Sex (female [670])	Male (117)	1.03 (0.998–1.05)	1.08 (1.07–1.1)
Race (White [618])	Black or African American (45)	1.03 (0.987–1.07)	1.03 (0.997–1.06)
	Asian (22)	0.974 (0.911–1.04)	0.937 (0.893–0.981)
	Other (34)	0.983 (0.932–1.03)	1 (0.965–1.04)
Prior treatment (PLT-based [335])	PLT-based and CPI (89)	0.973 (0.942–1)	0.987 (0.965–1.01)
	CPI (41)	0.986 (0.94–1.03)	0.972 (0.94–1)
	Other (322)	0.998 (0.982–1.01)	0.976 (0.965–0.988)
Number of prior lines of therapy,(≥5 [417])	4 (120)	1.02 (0.991–1.04)	1.03 (1.02–1.05)
	3 (121)	1 (0.978–1.03)	1.01 (0.995–1.03)
	2 (85)	1 (0.968–1.03)	1.03 (1–1.05)
	1 (44)	1.01 (0.969–1.06)	1.04 (1.01–1.07)
ECOG PS (0 [299])	1 (482)	0.97 (0.957–0.983)	0.997 (0.988–1.01)
Tumor type (HR+/HER2- mBC [292])	mUC (36)	1.01 (0.961–1.06)	1.09 (1.06–1.13)
	mTNBC (275)	0.991 (0.973–1.01)	1.01 (1–1.03)
	Other (184)	1 (0.979–1.02)	1.04 (1.02–1.05)
UGT1A1 GT (*1/*1 [302])	**1/*28* (308)	0.98 (0.963–0.996)	0.991 (0.980–1)
	**28/*28* (89)	0.976 (0.945–1.01)	0.998 (0.976–1.02)
	Missing (78)	0.973 (0.941–1.01)	0.997 (0.973–1.02)
	Other (10)	0.97 (0.878–1.06)	1.04 (0.971–1.1)
UGT1A1 inducer use (no [782])	Yes (5)	1.05 (0.918–1.18)	1.02 (0.932–1.12)
UGT1A1 inhibitor use (no [771])	Yes (16)	1.01 (0.939–1.09)	1.03 (0.977–1.08)
Hepatic function (normal function, total bilirubin and AST ≤ULN [525])	Mild impairment, bilirubin >ULN–1.5× ULN or AST >ULN (257)	0.976 (0.958–0.994)	0.983 (0.97–0.996)

Covariate (Reference [n])	Covariate (n)	Predicted SG Exposure (90% CI)	
		AUC	C_{max}
Renal function (normal function, ≥90 mL/min [404])	Mild impairment, 60 to <90 mL/min (301)	0.916 (0.901–0.931)	0.925 (0.915–0.936)
	Moderate impairment, 30 to <60 mL/min (78)	0.87 (0.84–0.9)	0.894 (0.874–0.915)

Abbreviations: CPI=checkpoint inhibitor; PLT=platinum.
a. Categorical covariate relationships to SG exposure in the first cycle relative to reference in IMMU-132-01, ASCENT, and TROPiCS-02.

Table 5. Continuous Covariate Relationships to SG Exposure Relative to Reference[a5]

Covariate (Reference)	Percentile	Predicted SG Exposure (90% CI)	
		AUC	C_{max}
Body weight at baseline (68.7 kg)	95th (105 kg)	1.24 (1.22–1.26)	1.22 (1.22–1.22)
	5th (49 kg)	0.87 (0.859–0.882)	0.88 (0.878–0.883)
Age (58 years)	95th (76 years)	0.979 (0.96–0.999)	0.99 (0.976–1)
	5th (38 years)	1.02 (1–1.04)	1.01 (0.997–1.03)
Albumin level (39 g/L)	95th (45 g/L)	1.06 (1.04–1.08)	1 (0.988–1.01)
	5th (30 g/L)	0.912 (0.892–0.932)	0.998 (0.983–1.01)
AST level (27.5 IU/L)	95th (124 IU/L)	0.951 (0.925–0.977)	0.984 (0.965–1)
	5th (14 IU/L)	1.01 (0.994–1.02)	1 (0.993–1.01)
ALT level (21.6 IU/L)	95th (88.3 IU/L)	0.985 (0.963–1.01)	0.996 (0.981–1.01)
	5th (8.84 IU/L)	1 (0.99–1.02)	1 (0.991–1.01)
Alkaline phosphatase level (99 IU/L)	95th (339 IU/L)	0.959 (0.939–0.979)	0.986 (0.971–1)
	5th (51.4 IU/L)	1.01 (0.995–1.02)	1 (0.994–1.01)
Bilirubin level (0.4 mg/dL)	95th (1 mg/dL)	0.986 (0.965–1.01)	1 (0.984–1.02)
	5th (0.2 mg/dL)	1 (0.989–1.02)	1 (0.989–1.01)
CrCl (91 mL/min)	95th (167 mL/min)	1.13 (1.11–1.15)	1.12 (1.1–1.13)
	5th (51.7 mL/min)	0.935 (0.919–0.95)	0.939 (0.929–0.95)
Baseline Trop-2 level (H score: 170)[b]	95th (290)	0.997 (0.972–1.02)	1 (0.986–1.02)
	5th (7.1)	1 (0.975–1.03)	0.994 (0.974–1.01)

Abbreviation: H score=histochemical score.
a. Continuous covariate relationships to SG exposure in the first cycle relative to reference in IMMU-132-01, ASCENT, and TROPiCS-02.
b. Patients in ASCENT and TROPiCS-02 only.

Exposure-Response Analyses in mBC

Exposure analysis of efficacy endpoints

The relationship between exposure and efficacy of SG, free SN-38, and tAB has been assessed in patients with mTNBC (N=277 [ASCENT, n=253 and IMMU-132-01, n=24]) who received SG 8 or 10mg/kg[7], and in patients with HR+/HER2- mBC (N=260 [TROPiCS-02]) who received SG 10 mg/kg[6]; SG was administered IV on days 1 and 8 of a 21-day cycle.

$CAVG_{tAB}$ was the most statistically significant (P<0.001 [p-value not reported in the HR+/HER2- mBC analysis]) exposure metric correlated with OS and PFS. Within the exposure range, higher $CAVG_{tAB}$ values were associated with longer mPFS and OS in patients with mTNBC and HR+/HER2- mBC (Table 6).[6]

Table 6. Median Survival Times by Quartiles of Exposure for PFS and OS[6,7]

Endpoint	Quartile	HR+/HER2- mBC (N=260)		mTNBC (N=277[a])	
		$CAVG_{tAB}$, median (range), µg/mL	Duration, median (95% CI), mo	$CAVG_{tAB}$, median (range), µg/mL	Duration, median (95% CI), mo
PFS	1	95 (55–116)	3.25 (2.79–4.27)	95.1 (50.9–112)	2.79 (1.87–3.35)
	2	135 (117–150)	2.86 (2.56–5.45)	130 (112–142)	4.01 (2.76–5.68)
	3	170 (150–190)	5.58 (4.17–10.3)	158 (143–174)	5.55 (4.24–6.73)
	4	232 (190–568)	9 (8.51–12.5)	205 (175–450)	7.91 (6.9–10.4)
OS	1	88 (48–110)	9.06 (6.67–11.6)	88.7 (50.6–107)	6.57 (5.12–9.03)
	2	127 (110–143)	12 (9.82–14.4)	125 (109–139)	10.8 (9–14.5)
	3	169 (145–187)	16.9 (13.9–22.7)	153 (140–173)	13.3 (10.9–15.9)
	4	226 (187–568)	26.8 (21.9–NA)	204 (174–451)	19.7 (17.6–NA)

Abbreviation: NA=not available.

a. Data from 275 patients were used for PFS analysis. Of the 277 patients, 24 were from IMMU-132-01 (starting dose 8mg/kg for 16 patients, 10mg/kg for five patients, and 12mg/kg for three patients) and 253 were from ASCENT (starting dose 8mg/kg for four patients and 10mg/kg for 249 patients).

Exposure response analyses for CR and ORR showed that $CAVG_{SG}$ was the most statistically significant metric (P<0.001 [p-value not reported in the HR+/HER2- mBC analysis]) correlated with these endpoints; higher values of $CAVG_{SG}$ were associated with an increased probability of CBR[6], CR and ORR.[6,7] See Table 7 for model-predicted probabilities of patients with CR, ORR, and CBR with SG 10 mg/kg.

Table 7. Model-Predicted Probabilities for Efficacy Endpoints (SG Starting Dose 10 mg/kg)[6,7]

	Probability (95% CI)		
	CR	ORR	CBR
mTNBC (n=258)	0.0426 (0.0194–0.0659)	0.326 (0.275–0.372)	NR
HR+/HER2- mBC (N=260)	0.008 (0–0.016)	0.204 (0.162–0.250)	0.331 (0.285–0.381)

Abbreviation: NR=not reported.

Exposure analysis of adverse events

For patients with mTNBC or HR+/HER2- mBC, there was a statistically significant relationship with increasing $CAVG_{SG}$ and certain AEs (*P*=0.001 for the mTNBC analysis, no significance value was specified for the mTNBC and HR+/HER2- mBC analysis [Table 8]).[6,7]

Table 8. Model-predicted OR for AEs Associated With an Increase in SG Exposure[6,7]

Any-grade AE	OR (95% CI) for a 1 µg/mL increase in $CAVG_{SG}$	
	mTNBC (N=277)[a]	mTNBC and HR+/HER2- mBC (N=569)[b]
Vomiting	1.29 (1.18–1.39)	1.29 (1.22–1.37)
Diarrhea	1.45 (1.3–1.63)	1.4 (1.32–1.5)
Nausea	1.55 (1.36–1.8)	1.37 (1.28–1.46)
Neutropenia	1.37 (1.26–1.5)	1.39 (1.33-1.45)
Hypersensitivity	NR	1.28 (1.21–1.35)

a. Patients in IMMU-132-01 and ASCENT.

b. Patients in IMMU-132-01, ASCENT and TROPiCS-02.

In patients with mTNBC, neutropenia was the only evaluated AE for which $CAVG_{SG}$ was significantly associated with a Grade ≥3 event (OR 1.09 [95% CI 1.05–1.14], *P*-value not provided). There was no significant association between $CAVG_{SG}$ and the probability of Grade 4 AEs for the four AEs listed in Table 7. An increase in $CAVG_{SG}$ was associated with

a statistically significant (P<0.001) increase in the risk of first dose reduction and first dose delay.[7]

In patients with HR+/HER2- mBC or mTNBC (N=569), a 10% increase in $CAVG_{SG}$ was predicted to increase the risk of experiencing Grade ≥3 neutropenia (OR 1.35 [95% CI 1.3–1.41]) and Grade ≥3 febrile neutropenia (OR 2.21 [95% CI 1.86–2.64]).[6]

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Abbreviations

ADC=antibody drug conjugate
AE=adverse event
AUC=area under the concentration-time curve
$AUC_{0–168\ h}$=area under serum concentration curve from 0 to 168 hours
$CAVG_{SG}$=average SG concentration
$CAVG_{tAB}$=total antibody average concentration
CBR=clinical benefit rate
C_{max}=maximum concentration
CR=complete response
ECOG PS=Eastern Cooperative Oncology Group Performance Status
GT=genotype
HER2=human epidermal growth factor receptor 2
HR=hormone receptor
mBC=metastatic breast cancer
mTNBC=metastatic triple-negative breast cancer
mUC=metastatic urothelial cancer
OR=odds ratio
ORR=objective response rate
OS=overall survival
PFS=progression-free survival
PK=pharmacokinetics
PopPK=population pharmacokinetics
SG=sacituzumab govitecan-hziy
$t_{1/2}$=half-life
tAB=total antibody
Trop-2=trophoblast cell surface antigen-2
UGT1A1=uridine diphosphate glucuronosyl transferase family 1 member A1

ULN=upper limit of normal

Product Label

For the full indication, important safety information, and boxed warning(s), please refer to the Trodelvy US Prescribing Information available at: www.gilead.com/-/media/files/pdfs/medicines/oncology/trodelvy/trodelvy_pi.

Follow-Up

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