Gilead Sciences, Inc. is providing this document to you, a US Healthcare Professional, in response to your unsolicited request for medical information.

GILEAD | Medical Information

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]).

Some data may be outside of the US FDA-approved prescribing information. In providing this data, Gilead Sciences, Inc. is not making any representation as to its clinical relevance or to the use of any Gilead product(s). For information about the approved conditions of use of any Gilead drug product, please consult the FDA-approved prescribing information.

The full indication, important safety information, and boxed warnings for neutropenia and diarrhea are available at: www.gilead.com/-/media/files/pdfs/medicines/oncology/trodelvy/trodelvy_pi.

Summary

Relevant Product Labeling[1]

The serum PK of SG and SN-38 were estimated in patients with mBC who received SG at the approved recommended dosage as a single agent or in combination with pembrolizumab and are presented as mean (%CV) unless otherwise specified. The following PK parameters were estimated based on population PK analyses as a single agent: C_{max} for SG and free SN-38 were 257107 ng/mL (18%) and 108 ng/mL (39%), respectively; $AUC_{0–168h}$ for SG and free SN-38 were 12,049,500 ng·h/mL (18%) and 3510 ng·h/mL (63%). No clinically significant differences in the PK of SG or SN-38 were observed when coadministered with pembrolizumab.

SG steady state volume of distribution is 4.6 L. The median elimination half-life ($t_{1/2}$) of SG is 6.5 days and free SN-38 is 22 hours. The mean (%CV) terminal half-life ($t_{1/2}$) of SG is 6.7 days (23%) and the apparent half-life ($t_{1/2}$) of free SN-38 is 24 hours (50%). The mean (%CV) clearance of SG is 0.13 L/h (20%). Renal elimination is known to contribute minimally to the excretion of SN-38, the small molecule moiety of SG.

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

SG PK Data

In the IMMU-132-01 study (in metastatic epithelial cancer) 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 (in mUC), and ASCENT (in mTNBC) 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* genotype was not identified as a significant covariate for AUC or C_{max}.[4]

Analyses of the IMMU-132-01, ASCENT and TROPiCS-02 (in HR+/HER2- mBC) 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, baseline albumin level, race, ECOG PS status, tumor type, *UGT1A1* genotype and Trop-2 expression.[5]

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]

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 1). 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 1. 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	$T_{1/2}$, Median (Range), h	V_{ss},[a] L	CL,[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

Study (n)		PK Parameter					
		C_{max}, Mean (%CV), ng/mL	T_{max}, Median (Range), h	$AUC_{0–168\ h}$, Mean (%CV), ng·h/mL	$T_{1/2}$, Median (Range), h	V_{ss},[a] L	CL,[a] L/h
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: CL=total body clearance; 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* genotype 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* genotype was not a significant covariate for AUC or C_{max} at the first treatment cycle for SG or free SN-38 (Table 2); the median values of $AUC_{0\text{-}168h}$ for SG and free SN-38 were also similar across different *UGT1A1* GTs.

Table 2. Estimated Exposure Across *UGT1A1* GTs in the PopPK Model (Cycle 1)[4]

Genotype	SG, Cycle 1		Free SN-38, Cycle 1	
	AUC, Mean (SD), mcg·h/mL	C_{max}, Mean (SD), mcg/mL	AUC, Mean (SD), mcg·h/mL	C_{max},Mean (SD), mcg/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 for the following covariates: mild to moderate renal impairment, mild hepatic impairment, tumor type, age, sex, baseline albumin level, race, ECOG status, UGT1A1 genotype, use of UGT1A1 inducers or inhibitors, or Trop 2 expression (Table 3 and Table 4).

Table 3. Categorical Covariate Relationships to SG Exposure Relative to Reference[5a]

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)
	Missing (68)	0.979 (0.943–1.01)	0.963 (0.938–0.988)

Intended for U.S. Healthcare Professionals only
Last Updated: 25 Jun 2026

Gilead Sciences, Inc. is providing this document to you, a US Healthcare Professional, in response to your unsolicited request for medical information.

Covariate (Reference [n])	Covariate (n)	Predicted SG Exposure (90% CI)	
		AUC	C_{max}
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])	mTNBC (275)	0.991 (0.973–1.01)	1.01 (1–1.03)
	mUC (36)	1.01 (0.961–1.06)	1.09 (1.06–1.13)
	Other (184)	1 (0.979–1.02)	1.04 (1.02–1.05)
UGT1A1 genotype (*1/*1 [302])	**1/*28* (308)	0.98 (0.963–0.996)	0.991 (0.98–1)
	**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)
Renal impairment (normal, ≥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; ULN=upper limit of normal.
[a]Categorical covariate relationships to SG exposure in the first cycle relative to reference in IMMU-132-01, ASCENT, and TROPiCS-02.

Table 4. Continuous Covariate Relationships to SG Exposure Relative to Reference[5a]

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 (95 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 (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)

Abbreviations: 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.

A bootstrap analysis confirmed final model estimates for SG, free SN-38, and tAB. Similar results noted for exposures to SG were also observed for free SN-38 and tAB (data not presented).

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 5).[6]

Table 5. 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), mcg/mL	Duration, Median (95% CI), mo	$CAVG_{tAB}$, Median (Range), mcg/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: 8 mg/kg for 16 patients, 10 mg/kg for 5 patients, and 12 mg/kg for 3 patients) and 253 were from ASCENT (starting dose: 8 mg/kg for 4 patients and 10 mg/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); higher values of $CAVG_{SG}$ were associated with an increased probability of CBR,[6] CR, and ORR.[6,7] See Table 6 for model-predicted probabilities of patients with CR, ORR, and CBR with SG 10 mg/kg.

Table 6. 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 (0.162–0.25)	0.331 (0.285–0.381)

Exposure analysis of AEs

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 7).[6,7]

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

Any-Grade AE	OR (95% CI) Per 1 mcg/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 4 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]

References

1. TRODELVY® Gilead Sciences Inc. Trodelvy (sacituzumab govitecan-hziy) for injection, for intravenous use. U.S. Prescribing Information. Foster City, CA.
2. Ocean AJ, Starodub AN, Bardia A, et al. Sacituzumab govitecan (IMMU-132), an anti-Trop-2-SN-38 antibody-drug conjugate for the treatment of diverse epithelial cancers: Safety and pharmacokinetics. *Cancer*. 2017;123(19):3843-3854.
3. Singh I, Sathe AG, Singh P, et al. Pharmacokinetics of sacituzumab govitecan, a Trop-2-directed antibody-topoisomerase I inhibitor SN-38 drug conjugate, in patients with advanced solid tumors [Poster Abstract P-161]. Presented at: American Society for Clinical Pharmacology and Therapeutics (ASCPT) Annual Meeting; 16-18 March, 2022; Virtual.
4. Sathe AG, Singh P, Singh I, et al. Impact of UGT1A1 polymorphisms on the pharmacokinetics of sacituzumab govitecan [Poster Abstract P-151]. Presented at: American Society for Clinical Pharmacology and Therapeutics (ASCPT) Annual Meeting; 16-18 March, 2022; Virtual.
5. Sathe AG, Jones AK, Diderichsen PM, et al. Sacituzumab govitecan population pharmacokinetics: updated analyses using HR+/HER2− metastatic breast cancer data from the phase 3 TROPiCS-02 trial. *Clin Transl Sci*. 2025;18(7):e70291.
6. Singh I, Sathe GK, Diderichsen PM, et al. Exposure-response analyses of sacituzumab govitecan efficacy and safety in patients with metastatic breast cancer [Poster PO1-04-06]. Presented at: San Antonio Breast Cancer Symposium (SABCS); December 5-9, 2023; San Antonio, TX, USA.
7. Sathe AG, Diderichsen PM, Fauchet F, Phan SC, Girish S, AA O. Exposure-response analyses of sacituzumab govitecan efficacy and safety in patients with metastatic triple-negative breast cancer. *Clin Pharmacol Ther*. 2024 Nov 14;doi: 10.1002/cpt.3495. Epub ahead of print.
8. Bardia A, Messersmith WA, Kio EA, et al. Sacituzumab govitecan, a Trop-2-directed antibody-drug conjugate, for patients with epithelial cancer: final safety and efficacy results from the phase I/II IMMU-132-01 basket trial [Supplementary Appendix]. *Ann Oncol*. 2021;32(6):746-756.

Abbreviations

ADC=antibody drug conjugate
AE=adverse event
AUC=area under the concentration-time curve
$AUC_{0–168h}$=area under serum concentration-time 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
CV=coefficient of variation
ECOG PS=Eastern Cooperative Oncology Group Performance Status
HER2=human epidermal growth factor receptor 2
HR=hormone receptor
mBC=metastatic breast cancer
mTNBC=metastatic triple-negative breast cancer
mUC=metastatic urothelial cancer
NR=not reported
OR=odds ratio
ORR=objective response rate
OS=overall survival
PFS=progression-free survival
PK=pharmacokinetic(s)
PopPK=population pharmacokinetics
SG=sacituzumab govitecan-hziy
SN-38=active metabolite of irinotecan
$t_{1/2}$=terminal half-life
tAB=total antibody
Trop-2=trophoblast cell surface antigen-2
UGT1A1=uridine diphosphate glucuronosyl transferase family 1 member A1

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

For any additional questions, please contact Trodelvy Medical Information at:

☎1-888-983-4668 or 🖱 www.askgileadmedical.com

Adverse Event Reporting

Please report all adverse events to:

Gilead Global Patient Safety ☎ 1-800-445-3235, option 3 or 🖱 www.gilead.com/utility/contact/report-an-adverse-event

FDA MedWatch Program by ☎ 1-800-FDA-1088 or ✉MedWatch, FDA, 5600 Fishers Ln, Rockville, MD 20852 or 🖱 www.accessdata.fda.gov/scripts/medwatch

Data Privacy

The Medical Information service at Gilead Sciences may collect, store, and use your personal information to provide a response to your medical request. We may share your information with other Gilead Sciences colleagues to ensure that your request is addressed appropriately. If you report an adverse event or concern about the quality of a Gilead or Kite product, we will need to use the information you have given us in order to meet our regulatory requirements in relation to the safety of our medicines.

It may be necessary for us to share your information with Gilead's affiliates, business partners, service providers, and regulatory authorities located in countries other than your own. Gilead Sciences has implemented measures to protect the personal information you provide. Please see the Gilead Privacy Statement (www.gilead.com/privacy-statements) for more information about how Gilead handles your personal information and your rights. If you have any further questions about the use of your personal information, please contact gilead.privacy@gilead.com.

TRODELVY, GILEAD, and the GILEAD logo are registered trademarks of Gilead Sciences, Inc., or its related companies.
© 2026 Gilead Sciences, Inc.