Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

GLP-1 Receptor Agonists and Cancer: Interpreting a Wave of Real-World Signals From the 2026 ASCO Annual Meeting

Natallia Starasvetskaya
Sylvester Comprehensive Cancer Center
Fernando Gerchman
Hospital de Clinicas de Porto Alegre
Gilberto de Lima Lopes Junior
glopes@med.miami.edu

Sylvester Comprehensive Cancer Center https://orcid.org/0000-0002-1151-9903

Systematic Review

Keywords: cancer, prevention, detection, treatment, GLP1 agonists, glucagon-like peptide-1

Posted Date: July 16th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10368790/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

Additional Declarations: The authors declare no competing interests.

Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and dual GIP and GLP-1 receptor/co-agonists have become central to the debate in metabolism and oncology. Evidence based on randomized clinical trials has established that these agents do not increase cancer risk, thereby dispelling the early concern regarding their potential association with cancer. Attention has since shifted to the converse hypothesis that GLP-1 RAs and dual GIP and GLP-1 receptor/co-agonists may prevent or slow cancer, advanced by large observational cohorts and, at the 2026 ASCO Annual Meeting, by an unusual concentration of abstracts. We performed a structured review and critical appraisal of that meeting, identifying 28 analyzable abstracts and presentations addressing prevention, metastatic progression, survival, drug interactions, and safety across at least twelve tumor types. With two exceptions, every abstract favored GLP-1 exposure, frequently with large effect sizes. All were observational and retrospective, and most were derived from the same federated electronic-health-record networks; the uniformity of apparent benefit is therefore at least as consistent with shared confounding (healthy-user selection, immortal-time bias, and confounding by indication) as with a genuine antineoplastic effect. The most rigorously designed studies, which used active antidiabetic comparators or quantified residual confounding, reduce but do not eliminate this concern. We interpret these preliminary not fully published data in the context of the published randomized and cohort literature, consider candidate mechanisms relevant to both obesity-related and non-obesity-related malignancies, and review the registered-trial landscape, which presently contains no adequately powered cancer-outcome study. The evidence is sufficient to prioritize randomized trials and insufficient to alter clinical practice. GLP-1 RAs should not be prescribed to prevent or treat cancer outside a clinical trial.

Plain Language Summary

GLP-1 receptor agonists and dual GIP and GLP-1 receptor/co-agonists, which include liraglutide, semaglutide and tirzepatide and are widely used for diabetes and obesity, have been linked in many recent studies to lower rates of cancer and to better survival. At the 2026 ASCO annual meeting, 28 such studies were presented, and nearly all reported benefit. This article reviews those studies for cancer specialists and primary care physicians. We explain why the results are encouraging but do not yet constitute proof. Each study observed patients who happened to be taking these drugs, and such patients tend to be healthier to begin with, which can make a medication look more effective than it is. The studies with a more solid methodological approach reduce this problem but do not remove it. We conclude that GLP-1 receptor agonists should not be used to prevent or treat cancer outside a clinical trial, and that suitably designed trials, which do not yet exist, are needed. Their utilization by oncologists in patients living with cancer, obesity and diabetes is a new frontier that need to be reached in order to define clear indications for their utility in clinical practice.

Introduction

The relationship between incretin mimetics (GLP-1 receptor and dual GIP and GLP-1 receptor agonists) and cancer has been reinterpreted twice within a decade, and on each occasion the pivotal evidence was randomized or otherwise peer reviewed. An early safety concern arose when the liraglutide development program recorded a numerical excess of breast cancer events. That concern was not borne out. A meta-analysis of 52 randomized controlled trials (1) enrolling approximately 89,000 participants found no increase in breast cancer with GLP-1 RA therapy (relative risk, 0.98; 95% CI, 0.76 to 1.26). Attention subsequently turned to the opposite hypothesis, that these agents might reduce cancer risk. A 2025 retrospective cohort study using a target-trial emulation design with 86,632 matched adults reported an approximately 17% lower incidence of obesity-related cancers (hazard ratio, 0.83) (2). A 2026 meta-analysis restricted to placebo-controlled randomized trials again identified no excess across malignancy types, largely reaffirming overall safety (3).

It is against this published background that the 2026 ASCO Annual Meeting presented an unusually large number of reports concerning GLP-1 RAs and cancer. These reports are conference abstracts. They are preliminary and most have not undergone full peer review or publication. While their results may change with longer follow-up and more complete analysis, the sheer volume of abstracts merit a systematic summary to inform clinicians and researchers. We have treated them not as established findings but as a body of work to be appraised, interpreted considering the published randomized and observational literature and of the registered trial record. Our objective is to provide oncologists, endocrinologists and primary care physicians with a structured basis for evaluating data whose internal consistency is considerable, but whose methodological foundations are still weak.

Methods: structured survey and critical appraisal of the meeting

We searched the 2026 ASCO Annual Meeting library using three terms. “GLP-1” returned 42 records, “GLP1” returned 8, and “Glucagon Like Peptide 1” returned 11, for 61 records identified. After removal of 12 duplicates, 49 unique abstracts remained. We excluded 21: publication-only abstracts that were confirmatory or redundant, and abstracts in which a GLP-1 RA was not the primary exposure. We retained two publication-only abstracts for specific evidentiary value: one reporting the only signal of possible harm, namely a GLP-1-associated delay in the stage at which pancreatic cancer was diagnosed (22), and one reporting a pre-specified safety hypothesis for small-intestinal neoplasia that returned a null result (21). This process yielded 28 abstracts: 4 oral, 1 rapid-oral, 21 posters, and 2 publication-only. The full classified dataset is available if requested. Because all included items are non-fully-peer-reviewed abstracts, the effect estimates reported here should be regarded as provisional.

The evidence in aggregate

The table lists all 28 abstracts by disease site, setting, design, headline effect estimate, and direction, with the corresponding reference number. The predominance of favorable findings is apparent.

Abs.	Disease	Setting	Design	Key result	Direction	Ref
10506	Breast	Prevention	TriNetX PSM	OR 0.695 (~ 30% lower incidence)	Favorable	4
10520	Breast	Prevention (high-risk)	TriNetX PSM	HR 0.844 (0.78–0.92)	Favorable	5
10548	Breast	Incidence + survival	TriNetX PSM	incidence RR 0.88; OS HR 0.75	Favorable	6
639	Breast	Survival	Single-center, UNMATCHED	5-yr OS 98.5% vs 85.5%	Favorable*	7
629	Breast	Survival	MarketScan PSM	5-yr OS adj HR 0.48	Favorable	8
1070	Breast (mBC)	Concurrent CDK4/6i	TriNetX PSM	mOS HR 0.70 (0.56–0.89)	Favorable	9
1100	Breast (mBC)	CDK4/6i - safety	Matched case-control	no excess ADR; PFS preserved	Favorable	10
628	Breast (support.)	Lymphedema prevention	TriNetX PSM	HR 0.37 (0.32–0.43)	Favorable	11
10559	Lung	Prevention	TriNetX PSM	HR 0.69 (0.62–0.76)	Favorable	12
8636	NSCLC	Concurrent TKI	TriNetX PSM	5-yr OS HR 0.46 (ALK 0.22)	Favorable	13
8598	Lung	Concurrent ICI	TriNetX PSM	mortality HR 0.65 (0.59–0.71)	Favorable	14
3636	Colorectal	Recurrence/survival	TriNetX PSM	RFS HR 0.37; OS HR 0.45	Favorable	15
3661	Colorectal	Concurrent chemo	Blue Diamond PSM	OS HR 0.82 (0.76–0.89)	Favorable	16
3605	CRC (mCRC)	Concurrent ICI	TriNetX PSM	1-yr mortality HR 0.67	Favorable	17
5543	Endometrial	Survival/mets	TriNetX PSM	mortality HR 0.45 (0.39–0.51)	Favorable	18
5625	Endometrial	Biomarker (GLP-1R)	Tissue microarray	GLP-1R + 32% (NSMP 42%)	n/a	19
4620	Bladder (NMIBC)	Concurrent BCG	TriNetX PSM	mortality RR 0.65; prog. RR 0.43	Favorable	20
e16499	Small intestine	Safety/incidence	TriNetX PSM	ARR 3.2%; no excess risk	Favorable (safety)	21
e16462	Pancreatic	Stage at diagnosis	Matched cohort 2:1	later-stage dx (+ 21.7 pts unadjusted; 18.6 pts NS adjusted)	Unfavorable	22
6592	Hematologic	Incidence	Medicare PSM vs DPP-4i	lower MDS/lymphoma/leukemia	Favorable	23
10507	Leukemia	Prevention	TriNetX PSM	AML incidence ~ -51%	Favorable	24
7548	Myeloma	Survival	TriNetX PSM	mortality HR 0.42 (0.35–0.51)	Favorable	25

Abs.	Disease	Setting	Design	Key result	Direction	Ref
3143	Multi (7)	Progression to stage IV	TriNetX PSM vs DPP-4i	NSCLC 10% vs 22%; +TCGA	Favorable	26
10508	Multi (13)	Incidence/mortality	TriNetX PSM vs insulin/metf.	HRs 0.04–0.46 (implausibly low)	Favorable*	27
10583	Multi (CKD)	Incidence/mortality	TriNetX PSM	ACM RR 0.70; malignancy RR 0.57	Favorable	28
10590	Multi	Incidence (vs SGLT2i)	MarketScan TTE	GLP-1 ~ SGLT2i, HR 0.97 (NS)	Null	29
11000	Multi (on ICI)	Survival + irAEs	TriNetX PSM	OS HR 0.69–0.71; lower pyrexia	Favorable	30
11017	Multi (6)	Survival	iKnowMed PSM	OS adj HR 0.66; E-value 2.4	Favorable	31

** Methodologically weakest designs: reference 7 (unmatched) and reference 27 (insulin comparator yielding implausibly low hazard ratios). PSM, propensity-score matched; TTE, target-trial emulation; ACM, all-cause mortality; ARR, absolute risk reduction; OS, overall survival; RFS, recurrence-free survival. All data derive from non-fully-peer-reviewed 2026 ASCO abstracts.*

We summarize the findings by disease cluster and by cross-cutting setting rather than by individual abstract.

Breast cancer

Prevention generated the most reproducible signal. In an analysis of more than 100,000 screening-eligible women, GLP-1 use was associated with approximately 30% lower breast cancer incidence (odds ratio, 0.695) (4), a finding echoed in a high-risk cohort (hazard ratio, 0.844) (5) and in a cohort of subjecs without diabetes (incidence relative risk, 0.88; overall-survival hazard ratio, 0.75) (6). Survival analyses were consistently favorable but varied in methodological quality. A propensity-matched cohort reported an adjusted 5-year overall-survival hazard ratio of 0.48 (8), whereas a single-institution series without matching reported 5-year survival of 98.5% compared with 85.5% (7). In metastatic disease, concurrent GLP-1 use was not associated with diminished CDK4/6-inhibitor efficacy and was associated with improved outcomes in one analysis (overall-survival hazard ratio, 0.70) (9), with progression-free survival preserved and no excess toxicity (10). In supportive care, GLP-1 use was associated with a substantial reduction in post-mastectomy lymphedema (hazard ratio, 0.37) (11). The mechanistic rationale is most direct for breast cancer: after menopause, adipose aromatase is the principal source of circulating estrogen, and weight loss would be expected to reduce estrogenic drive, with additional contributions from lower insulin and IGF-1 concentrations and reduced adipose inflammation.

Lung and colorectal cancer

In lung cancer, GLP-1 exposure was associated with lower incidence among high-risk patients (hazard ratio, 0.69) (12) and with improved survival in combination with tyrosine kinase inhibitors (5-year overall-survival hazard ratio, 0.46; ALK subgroup, 0.22) (13) and with immune checkpoint inhibitors (mortality hazard ratio, 0.65) (14). In colorectal cancer, a stage I to III cohort reported lower recurrence (recurrence-free survival hazard ratio, 0.37) and improved survival (0.45) (15); a cohort receiving chemotherapy reported a survival hazard ratio of 0.82 with reduced toxicity (16); and a metastatic cohort treated with checkpoint inhibitors reported a 1-year mortality hazard ratio of 0.67 (17). The repeated

co-occurrence of GLP-1 exposure with improved checkpoint-inhibitor and TKI outcomes raises the possibility of an immuno-metabolic interaction that weight loss alone would not account for.

Gynecologic, hematologic, and genitourinary cancers

In endometrial cancer complicated by obesity, GLP-1 use was associated with a mortality hazard ratio of 0.45 (18). A companion tissue study identified GLP-1 receptor expression in 32% of endometrial tumors, rising to 42% in the no-specific-molecular-profile subtype (19), which provides a candidate direct target. In hematologic malignancy, GLP-1 RAs were associated with lower incidence of myelodysplastic syndrome, lymphoma, and leukemia relative to an active DPP-4 inhibitor comparator (23); a large emulation analysis (or large target trial emaulation?) reported an approximately 51% reduction in acute myeloid leukemia incidence (24); and a myeloma cohort reported a mortality hazard ratio of 0.42 (25). In high-risk non-muscle-invasive bladder cancer treated with BCG, GLP-1 exposure was associated with lower mortality (relative risk, 0.65) and reduced progression to muscle-invasive disease (relative risk, 0.43) (20). Benefits in malignancies outside the obesity-estrogen axis, if substantiated, would implicate direct receptor signaling, immunomodulation, or systemic anti-inflammatory effects rather than weight loss.

Pan-cancer and safety analyses

Multi-tumor analyses were prominent at the meeting. A propensity-matched comparison against DPP-4 inhibitors across seven solid tumors reported reduced progression to stage IV disease in lung, breast, colorectal, and liver cancers, supported by a correlation between GLP-1 receptor expression and survival in The Cancer Genome Atlas (26). GLP-1 RAs use compared against insulin and metformin reported uniformly lower incidence in 13-cancer types, although several hazard ratios near 0.04 are not biologically credible or clinically relevant (27). A separate analysis restricted to patients with chronic kidney disease reported lower all-cause mortality (relative risk, 0.70) and lower overall malignancy (relative risk, 0.57) (28). A community-oncology cohort reported an adjusted survival hazard ratio of 0.66 accompanied by an E-value sensitivity analysis (31). Two analyses serve as counterweight: one found GLP-1 RAs no better than SGLT2 inhibitors for cancer incidence (29), and a pre-specified analysis found no excess of small-intestinal neoplasia (21). An analysis of patients receiving immune checkpoint inhibitors reported improved survival (overall-survival hazard ratio, 0.69 to 0.71) with fewer immune-related adverse events (30).

Not all studies were created equally

The 28 abstracts are not of equal evidentiary value and should not be weighted equally. Four are designed more rigorously than the remainder. The progression analysis used an active comparator, DPP-4 inhibitors rather than non-users, so that both groups comprised patients with diabetes who were under treatment, and it incorporated an independent biomarker correlate from The Cancer Genome Atlas (26). The hematologic and DPP IV-inhibitor analyses employed active antidiabetic comparators (23, 29). The community-oncology cohort reported an E-value of 2.4, indicating that an unmeasured confounder would need to be associated with both exposure and survival by a factor of 2.4 to account for the observed association (31). Such features mitigate the two biases most likely to distort this literature. Healthy-user bias arises because patients who initiate and adhere to a weight-loss or glucose-lowering medication tend to be healthier and more engaged in their care; immortal-time bias arises when the interval preceding treatment is misclassified as exposed. An active comparator, in which both groups are unwell enough to warrant a glucose-lowering agent, reduces the former, and careful definition of the index date reduces the latter.

These designs nonetheless cannot establish causation. An active comparator attenuates but does not remove confounding by indication, because GLP-1 RAs may be preferentially prescribed to patients with better functional status

and fewer competing risks. A biomarker correlation is associative. An E-value characterizes the strength of a confounder and would require without excluding its existence. Residual confounding, the short interval between exposure and ascertainment, and reverse causation remain plausible throughout. The defensible conclusion is correspondingly limited. The evidence is sufficient to justify prioritizing randomized trials and insufficient to alter clinical practice or to establish a new standard of care.

Interpreting the predominance of positive findings

Among the 28 abstracts, 25 favored GLP-1 exposure, one reported harm, one was null, and one assessed a biomarker without a clinical outcome. Uniform benefit across more than a dozen tumor types is uncommon and warrants scrutiny. Three explanations merit consideration. The first, and the most parsimonious, is confounding. Two of the included studies illustrate the problem. The unmatched single-institution breast series reported 5-year survival approaching 99% across all subtypes, including triple-negative and stage III disease (7), a result more consistent with a selected, lower-risk population than with a treatment effect. The 13-cancer analysis reported hazard ratios near 0.04 against an insulin comparator (27); a reduction of that magnitude reflects the status of insulin as a marker of advanced metabolic disease and high competing mortality rather than antineoplastic effect. The second explanation concerns method and reporting. The studies are retrospective and uncontrolled, conference abstracts are subject to selective submission of positive findings, the examination of many tumor types and outcomes invites false-positive associations, and the brief interval between exposure and ascertainment is incompatible with the latency of carcinogenesis. The third explanation, a genuine biological effect, cannot be dismissed. The mechanism is plausible, receptor expression correlates with survival in independent data, the active-comparator studies did not revert to the null, benefit was observed in both obesity-related and non-obesity-related malignancies, and a pre-specified safety analysis returned a null rather than a spuriously positive result. The available data do not permit these explanations to be disentangled. A randomized trial with a cancer endpoint and an active comparator would do so. Future clinical trials should adopt novel methodological approaches specifically designed to evaluate how incretin mimetics and new classes of co-agonists in development affects patients living with cancer, individuals at high risk for cancer, and those living with obesity and/or diabetes. These studies should incorporate rigorous and prespecified cancer adjudication procedures to accurately assess how or if these medications changes cancer incidence and/or protects against cancer progression, and cancer-specific mortality. Such approaches are particularly important because previous randomized trials of anti-diabetic and anti-obesity therapies, which were not designed to evaluate their effects on cancer outcomes and were conducted in populations with obesity and/or diabetes—groups inherently at increased risk of cancer—have generally failed to demonstrate reductions in cancer mortality among participants without pre-existing cancer. Clinical trials and research actively investigating how diabetes and anti-obesity medications can be repurposed to prevent and treat cancer are under way and will answer their impact on cancer remission and/or mortality.

Mechanistic considerations

Two mechanistic questions should be considered separately. For obesity-related cancers, the relevant pathways are established: weight loss reduces aromatase-derived estrogen in hormone-receptor-positive disease, lowers circulating insulin and IGF-1, and diminishes adipose-associated inflammation. The more difficult question concerns malignancies not strongly associated with obesity, such as leukemia and urothelial carcinoma, for which weight loss provides an insufficient explanation. Several alternative mechanisms are biologically plausible: direct GLP-1 receptor signaling on tumor and stromal cells, immunomodulation consistent with the observed association between GLP-1 use and improved checkpoint-inhibitor outcomes, systemic anti-inflammatory effects, incretin- and microbiome-mediated effects, and metabolic reprogramming of the tumor microenvironment. The receptor itself connects association to mechanism. Its expression in approximately one-third of endometrial tumors, together with the correlation between

expression and survival in pan-cancer data, is consistent with an effect exerted on the tumor directly rather than solely through changes in the patient's weight. This hypothesis remains untested.

Where are the prospective trials?

If the observational evidence is sufficient to warrant randomized trials, the relevant question is what trials are in progress. The current landscape is limited. ClinicalTrials.gov lists 26 ongoing interventional studies of GLP-1 RAs and the dual GIP/GLP-1 receptor agonist tirzepatide in cancer, none of which is powered to address the question definitively (32). They are small, with enrollment between 20 and 80 participants, Phase 2 or earlier, and oriented toward weight-loss or window-of-opportunity endpoints rather than cancer incidence, survival, or progression, and they are concentrated in endometrial and breast cancer. Representative examples include an adjuvant tirzepatide study in HR-positive early breast cancer with a circulating-tumor-DNA endpoint (NCT06517212), a fertility-sparing endometrial trial (NCT06073184), a metabolic-protection trial during androgen-deprivation therapy in prostate cancer (NCT07202247), and an early-phase study adding semaglutide to first-line therapy in metastatic gastrointestinal cancers (NCT07627191). The large GLP-1 outcome programs, including SELECT and the STEP and SURMOUNT trials, capture malignancy only as exploratory adverse events. No adequately powered randomized trial with a cancer endpoint currently exists, and this deficiency is the principal conclusion of the present review.

Implications for practice

For oncologists. Medication reconciliation at each encounter is warranted, given the rising prevalence of GLP-1 RA, dual GIP/GLP-1 receptor agonist and their crescent application in patients living with cancer.

Additionally, the anticipated approval of novel potent weight loss medications that act on different hormone pathways, particularly those used in dual or triple combination regimens, may broaden therapeutic options and offer new perspectives in cancer care. This is especially relevant when considering obesity and diabetes as key comorbidities that significantly affect quality of life and influences the prognosis of patients living with cancer. The progression and survival signals should be noted but not overinterpreted, and these agents should not be initiated or continued for an oncologic indication. The pancreatic data support one specific caution: GLP-1-induced satiety and weight loss may resemble and obscure the early symptoms of pancreatic cancer, and symptoms disproportionate to the expected pharmacologic effect should prompt evaluation rather than attribution to the medication. In that analysis, GLP-1 users were 21.7 percentage points more likely than non-users to be diagnosed with borderline-resectable rather than resectable disease ($p = .035$), but the difference attenuated to 18.6 points and lost significance after adjustment for diabetes ($p = .09$), so the signal is also hypothesis-generating rather than established.

For primary care physicians. GLP-1 RAs and the other incretin mimetics are agents for obesity and diabetes management that may confer an incidental oncologic benefit; they are not cancer therapeutics. The association is sufficiently substantiated to justify investigation and insufficiently substantiated to influence the decision to start, continue, or discontinue treatment. These agents should be prescribed for their approved indications and should not be initiated or stopped for cancer-related reasons.

Limitations

This review is based principally on conference abstracts that have not undergone extensive peer review and whose results may change with longer follow-up; we have therefore anchored our conclusions to the peer-reviewed randomized, and cohort literature and to the registered trial record rather than to the abstracts themselves. The included studies are uniformly observational and retrospective, and most derive from the same federated electronic-health-record networks, so they may share, rather than independently corroborate, the same biases. Effect estimates were extracted from abstracts, posters, and slides, several from figures rather than text, and should be verified against the

final publications. Our search was confined to a single meeting and three search terms and may have omitted abstracts that named specific agents without the class term. We did not undertake a quantitative synthesis, which would be inappropriate given the heterogeneity and probable confounding of the source data.

Conclusion

The 2026 ASCO Annual Meeting transformed an incidental observation in metabolic oncology into an active area of inquiry, with 28 abstracts pointing, with few exceptions, in the same direction. The consistency of these findings is their most notable feature and the principal reason for caution. A plausible mechanism and demonstrable receptor expression in tumors lends the pattern credibility. At the same time, uniform benefit across many tumor types, derived almost entirely from retrospective records that share common sources of bias, is also the expected product of confounding. The trajectory recalls that of metformin, whose apparent anticancer benefit attenuated as methodological biases were addressed. Two conclusions should be maintained concurrently: that GLP-1 receptor agonists and other incretin mimetics are safe with respect to cancer, based on RCTs and metanalysis of RCTs, and that their putative capacity to prevent or slow cancer remains an unproven hypothesis. Pending an adequately powered randomized trial, GLP-1 receptor agonists should not be used to prevent or treat cancer outside the setting of a clinical trial.

References

1. Piccoli GF, Mesquita LA, Stein C, Lopes G, Gerchman F et al (2021) Do GLP-1 receptor agonists increase the risk of breast cancer? A systematic review and meta-analysis. J Clin Endocrinol Metab 106(3):912–921
2. Dai H et al (2025) GLP-1 receptor agonists and cancer risk in adults with obesity. JAMA Oncol 11(10):1186–1193
3. Ko A, Chang YC, Bahar F et al (2026) Risk for cancer with GLP-1 receptor agonists and dual agonists: a systematic review and meta-analysis of randomized trials. Ann Intern Med 179:216–229
4. McDonald ES et al (2026) Association of GLP-1 agonists with breast cancer incidence in women. J Clin Oncol 44(suppl 16):abstr10506
5. Gotera NP, Jones C et al (2026) GLP-1 receptor agonists for primary prevention of breast cancer in high-risk women with obesity: a real-world analysis of efficacy and safety. J Clin Oncol 44(suppl 16):abstr10520
6. Shah Z et al (2026) GLP-1 therapy and hormone receptor-positive breast cancer risk and survival: a real-world analysis. J Clin Oncol 44(suppl 16):abstr10548
7. Prior JT et al (2026) The impact of GLP-1 receptor agonists on the survival of breast cancer patients. J Clin Oncol 44(suppl 16):abstr639
8. Sukumar J, Jackson I et al (2026) Real-world treatment patterns and mortality associated with GLP-1 receptor agonists in breast cancer patients. J Clin Oncol 44(suppl 16):abstr629
9. Palleschi M et al (2026) Real-world survival benefit of GLP-1 receptor agonists concomitant with CDK4/6 inhibitors and endocrine therapy in HR-positive/HER2-negative metastatic breast cancer. J Clin Oncol 44(suppl 16):abstr1070
10. Patino K et al (2026) Impact of GLP-1 receptor agonists in hormone receptor-positive breast cancer patients receiving CDK4/6 inhibitors. J Clin Oncol 44(suppl 16):abstr1100
11. Yesho DH et al (2026) GLP-1 receptor agonist for primary prevention of post-mastectomy lymphedema in breast cancer patients: a multicenter real-world analysis. J Clin Oncol 44(suppl 16):abstr628
12. Neely AN et al (2026) Clinical impact of GLP-1 receptor agonist use on lung cancer incidence in high-risk patients: a real-world analysis. J Clin Oncol 44(suppl 16):abstr10559
13. Shaverdashvili K et al (2026) The effect of GLP-1 receptor agonists on outcomes in metastatic NSCLC patients treated with tyrosine kinase inhibitors: a real-world retrospective analysis. J Clin Oncol 44(suppl 16):abstr8636

14. Ng WL et al (2026) Glucagon-like peptide-1 receptor agonist use in lung cancer patients receiving immune checkpoint inhibitors: a real-world study. J Clin Oncol 44(suppl 16):abstr8598
15. Kinsey EN et al (2026) Relationship between GLP-1 receptor agonists and cancer recurrence and survival in patients with stage I-III colorectal cancer: a retrospective cohort study using the TriNetX database. J Clin Oncol 44(suppl 16):abstr3636
16. Krishnan A et al (2026) Impact of concomitant GLP-1 receptor agonist use on overall survival and chemotherapy-related adverse events in colorectal cancer: a real-world cohort study. J Clin Oncol 44(suppl 16):abstr3661
17. Rawal S et al (2026) Association of concurrent GLP-1 receptor agonist use with survival outcomes in patients with metastatic colorectal cancer receiving immune checkpoint inhibitors: a multicenter real-world study. J Clin Oncol 44(suppl 16):abstr3605
18. Arya Y et al (2026) Real-world outcomes comparing the use of GLP-1 agonists in patients with endometrial cancer and comorbid obesity. J Clin Oncol 44(suppl 16):abstr5543
19. Gonzalez-Merino C et al (2026) GLP-1R expression in a molecularly classified cohort of endometrial carcinoma: GEICO 163-T/SPECTRUM study. J Clin Oncol 44(suppl 16):abstr5625
20. Yaniv B, Trivedi C, Mittal K (2026) Association of GLP-1 receptor agonist exposure with BCG response in high-risk non-muscle-invasive bladder cancer. J Clin Oncol 44(suppl 16):abstr4620
21. Kherajani PA et al (2026) GLP-1 receptor agonist therapy and risk of small intestinal neoplasms: real-world evidence from a global network. J Clin Oncol. ;44(suppl 16):abstr e16499
22. O'Connor S et al (2026) Evaluation of the impact of GLP-1 receptor agonists on stage of pancreatic ductal adenocarcinoma diagnosis: a retrospective matched cohort study. J Clin Oncol. ;44(suppl 16):abstr e16462
23. Chen WH et al (2026) Glucagon-like peptide-1 receptor agonist and hematologic cancer risk among older adults with type 2 diabetes. J Clin Oncol 44(suppl 16):abstr6592
24. Jones CF et al (2026) Glucagon-like peptide-1 receptor agonists for the primary prevention of leukemias in high-risk patients: a global, propensity-matched real-world analysis. J Clin Oncol 44(suppl 16):abstr10507
25. Syal A et al (2026) Real-world outcomes comparing the use of GLP-1 agonists in patients with multiple myeloma and comorbid obesity: a propensity score-matched analysis. J Clin Oncol 44(suppl 16):abstr7548
26. Orland M et al (2026) Can GLP-1 receptor agonists mitigate cancer progression? A propensity-matched analysis across seven solid tumors. J Clin Oncol 44(suppl 16):abstr3143
27. Shah VR et al (2026) Comparative analysis of tirzepatide versus metformin and insulin in type 2 diabetes patients across the 13 most common cancers. J Clin Oncol 44(suppl 16):abstr10508
28. Asad M, Durmishi A et al (2026) GLP-1 receptor agonist therapy and cancer risk and mortality in patients with moderate chronic kidney disease: a national matched cohort study. J Clin Oncol 44(suppl 16):abstr10583
29. Vobugari N et al (2026) Association of SGLT2 inhibitors and GLP-1 receptor agonists or DPP-4 inhibitors with incident cancer in patients with type 2 diabetes. J Clin Oncol 44(suppl 16):abstr10590
30. Jajja SA et al (2026) Association of GLP-1 receptor agonist use with survival and immune-related adverse events in patients receiving immune checkpoint inhibitors: a multi-institutional real-world analysis. J Clin Oncol 44(suppl 16):abstr11000
31. Paulus JK et al (2026) GLP-1 receptor agonist use and survival outcomes in a large US community oncology cohort. J Clin Oncol 44(suppl 16):abstr11017
32. ClinicalTrials.gov. US National Library of Medicine. Accessed (2026) June Cited registrations: NCT06517212, NCT06073184, NCT07202247, NCT07627191, and related GLP-1 oncology trials

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- GLP1ASCO2026master.xlsx