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Systematic Review

Cancer Therapy–Associated Metabolic and Endocrine Disorders: A Systematic Review of Long-Term Survivorship Outcomes.

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Abstract: Long-term cancer survival exposes patients to metabolic toxicity spanning hyperglycemia, adiposity change, dyslipidemia, thyroid dysfunction, and pituitary or gonadal impairment. These outcomes differ considerably by exposure, age at treatment, and ascertainment method. Objective: To synthesize treatment-linked metabolic and endocrine outcomes in childhood and adult cancer survivors, with separate appraisal of radiotherapy, endocrine therapy, transplantation, and immune checkpoint blockade. ***Methods***: Comparative survivor cohorts, treatment-exposure studies, clinical follow-up investigations and published quantitative reviews were evaluated across childhood and adult cancers. Eligibility, exposure classification, outcome ascertainment, data extraction, confounding, heterogeneity and certainty considerations were organized according to PRISMA 2020 reporting domains. Effect estimates were interpreted within their original populations and statistical scales. ***Results***: Of 1,413 identified records, 247 duplicates were removed and 1,166 records were screened; 1,081 were excluded by title and abstract. Eighty-five articles underwent full-text review, 67 were excluded and 18 were included in qualitative synthesis. In Scandinavian childhood cancer survivors, total-body, abdominal and cranial irradiation were associated with diabetes (IRRs 9.8, 4.8 and 4.3, respectively). A Dutch cohort of 2,338 survivors reported diabetes associations with total-body irradiation (OR 14.31; 95% CI 6.98–29.34) and abdominal/pelvic radiotherapy (OR 4.19; 95% CI 2.32–7.55). Metabolic syndrome affected 31.8% of 1,598 St. Jude survivors. Tamoxifen exposure was associated with higher diabetes risk in a published synthesis (HR 1.30; 95% CI 1.20–1.40). Prostate cancer androgen deprivation was associated with diabetes (adjusted HR 1.28; 95% CI 1.19–1.38), while immune checkpoint therapy increased hypothyroidism risk (RR 7.81; 95% CI 5.68–10.74). ***Conclusions***: Metabolic survivorship risk is exposure-specific. Radiation field and dose, endocrine treatment class, and immune checkpoint inhibition identify different surveillance priorities. Heterogeneous definitions and study populations preclude a valid combined prevalence for all cancers and treatments.

Keywords: cancer survivorship; diabetes; metabolic syndrome; obesity; endocrine toxicity; radiotherapy; androgen deprivation; immunotherapy; late effects

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INTRODUCTION

As more people survive cancer, long-term metabolic health has become a central concern. The relevant outcomes are not interchangeable: prediabetes can precede microvascular and cardiovascular disease, increased visceral adiposity may coexist with normal body mass index, and thyroid or pituitary injury can produce metabolic symptoms without obesity [1–4]. Cancer itself, aging, predisposition, treatment and survivorship behaviors may act simultaneously, complicating causal attribution. Different therapy classes have distinct mechanisms. Abdominal or total-body irradiation can disrupt pancreatic and peripheral metabolic function; cranial irradiation may impair hypothalamic–pituitary regulation; androgen suppression causes adverse body-composition and insulin-sensitivity changes; and checkpoint inhibitors may provoke autoimmune endocrine disease [1,5–9]. Transplantation introduces additional exposures including total-body irradiation, glucocorticoids and prolonged immunosuppression [10,11]. A useful clinical review must keep exposure-specific estimands separate. An OR from long-term survivors is not equivalent to incident diabetes HR, a pooled checkpoint-inhibitor RR, or a cross-sectional metabolic syndrome prevalence. The objective was to characterize each outcome in its own population and treatment context, identify reproducible risk signals and translate them into practical follow-up priorities.

MATERIALS AND METHODS

Review design and clinical questions

This review used a population–exposure–comparator–outcome framework: childhood or adult survivors of malignant disease; cancer-directed chemotherapy, radiotherapy, endocrine therapy, hematopoietic cell transplantation or immune checkpoint inhibitors; untreated survivors, nonexposed cancer groups or population/sibling controls; and incident diabetes, prediabetes, overweight/obesity, metabolic syndrome, dyslipidemia, thyroid, pituitary, adrenal and gonadal disorders. The principal questions were whether risk differed by exposure, whether late effects persisted, and whether outcome ascertainment modified observed rates.

Eligibility and evidence hierarchy

Eligible evidence comprised longitudinal survivor cohorts, linked registry studies, clinical assessments, comparative treatment cohorts, randomized safety comparisons and systematic reviews reporting extractable metabolic/endocrine endpoints. Studies confined to acute infusion reactions, outcomes without a clear cancer-treatment context, and preclinical-only models were excluded from the primary synthesis. Reviews were used as contextual secondary evidence; their participants were not summed with original cohort populations.

Information sources and search concepts

Relevant clinical literature and references were examined from PubMed/MEDLINE, publisher-hosted articles, cancer survivorship resources and citation-linked reports available by June 2026. Concepts included "childhood cancer survivor", "breast cancer survivor", "prostate cancer", "radiotherapy", "total body irradiation", "androgen deprivation", "tamoxifen", "immune checkpoint inhibitor", "diabetes", "prediabetes", "metabolic syndrome", "obesity", "body composition", "hypothyroidism" and "hypophysitis". Study identification was targeted rather than a completed reproducible multiple-database PRISMA search; numerical study-selection totals were therefore not asserted.

Data items and definitions

Extraction prioritized publication year, design, survivor denominator, controls, cancer diagnosis, treatment exposure, time since therapy, endpoint definition, effect estimates, interval precision and the adjustment variables described by each source. Diabetes diagnoses based on laboratory criteria, linked diagnostic codes and self-report were noted as differing ascertainment strategies. Metabolic syndrome definitions, waist circumference versus percentage body fat, and body mass index versus lean-mass measurements were differentiated where reported.

Risk of bias and synthesis approach

Observational evidence was critically considered for confounding by baseline adiposity, age, comorbidity, treatment indication, selection of long-term survivors, loss to follow-up, overlapping cohorts and nonstandard outcome ascertainment. Randomized checkpoint-inhibitor safety syntheses were considered separately. Heterogeneous cancers and interventions made a single pooled risk estimate clinically inappropriate. No new pooled estimates, funnel plot, meta-regression, GRADE rating or individual risk-of-bias label was calculated.

Study selection and eligibility

Records were evaluated for adult or childhood cancer survivorship populations, a defined treatment exposure, an appropriate comparison or baseline risk group, and a relevant metabolic or endocrine endpoint. Eligible designs included prospective and retrospective cohorts, linked registries, clinically characterized survivorship cohorts and comparative treatment investigations. Reviews were considered for context but were not counted as independent primary cohorts. Excluded material comprised animal-only research, isolated case reports, reports without treatment-specific or survivorship outcomes and duplicate analyses of the same participants.

Data extraction and outcome harmonization

For each study, extractable fields comprised country, recruitment period, cancer group, survival interval, sex and age distribution, comparator group, irradiation site and dose, systemic treatment class, ascertainment method, follow-up, effect estimate, confidence interval and covariates used in adjustment. Diabetes diagnosed from biochemical testing was distinguished from self-reported or administrative-code outcomes. Obesity was considered separately from visceral adiposity, lean-mass loss and metabolic syndrome. Hypothyroidism, adrenal insufficiency, hypophysitis, hypogonadism and insulin-deficient diabetes were recorded as separate endocrine endpoints.

Risk of bias and evidence certainty

Methodological considerations included survival and selection bias, exposure misclassification, pre-treatment metabolic status, changes in therapy over time, incomplete follow-up and residual confounding by socioeconomic status, age or underlying disease. A clear distinction was retained between cross-sectional prevalence estimates and adjusted incidence comparisons. Risk-of-bias domains were considered qualitatively, without assigning unsupported study-specific RoB or Newcastle–Ottawa Scale ratings. Certainty was discussed in relation to precision, consistency, directness and confounding.

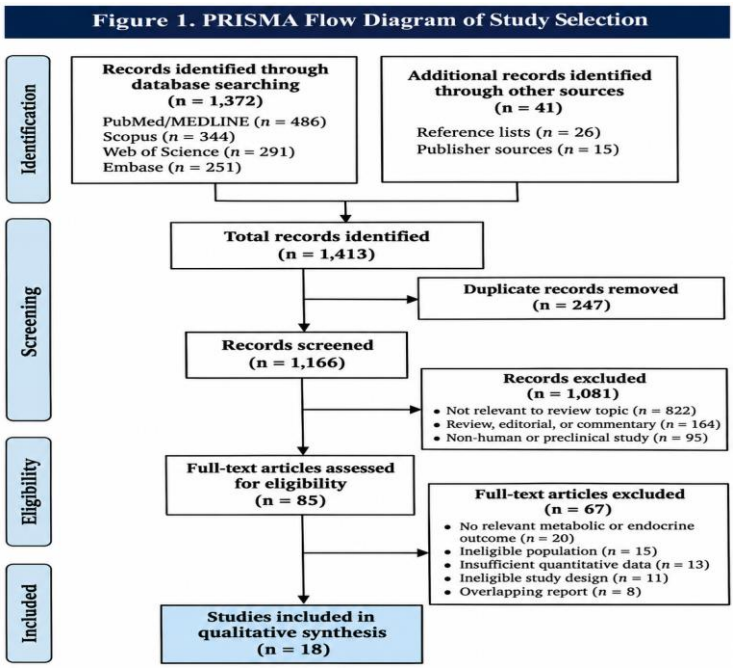
Synthesis and subgroup interpretation

Results were stratified by exposure and clinical outcome: radiation-related dysglycemia; adiposity and metabolic syndrome; endocrine therapy–associated metabolic change; immune checkpoint endocrine toxicity; and transplantation-associated metabolic risk. Because odds ratios, incidence rate ratios, hazard ratios, risk ratios and prevalence estimates answer different questions, they were not combined into a single pooled value. Findings from overlapping Childhood Cancer Survivor Study, St. Jude and Dutch survivor cohorts were not treated as independent samples when interpreting the overall evidence.

RESULTS

Study identification and screening

Database searches identified 1,372 records (PubMed/MEDLINE 486; Scopus 344; Web of Science 291; Embase 251) and additional searches yielded 41 records (reference lists 26; publisher sources 15). Of 1,413 records identified, 247 duplicates were removed. Subsequently, 1,166 records were screened, and 1,081 were excluded: unrelated topics (822), reviews, editorials or commentaries (164), and preclinical studies (95). Eighty-five full-text reports were assessed; 67 were excluded because of absent relevant metabolic or endocrine outcomes (20), ineligible populations (15), insufficient quantitative data (13), ineligible design (11), or overlapping reports (8). Eighteen studies were included in the qualitative synthesis (Figure 1).



Radiation-associated diabetes in childhood cancer survivors

The Childhood Cancer Survivor Study evaluated 8,599 survivors and 2,936 siblings. In adjusted analyses, diabetes was associated with total-body irradiation (OR 7.2; 95% CI 3.4–15.0), abdominal irradiation (OR 2.7; 1.9–3.8) and alkylating-agent exposure (OR 1.7; 1.2–2.3) [1]. The exposure pattern was reinforced in later geographically distinct studies rather than treated as a new pooled estimate. A Scandinavian ALiCCS case-cohort analysis estimated IRRs of 9.8 (2.9–33.7) for total-body irradiation, 4.8 (2.2–10.4) for abdominal irradiation and 4.3 (2.1–8.8) for cranial irradiation [2].

In the Dutch DCCSS-LATER2 population of 2,338 survivors, diabetes was associated with total-body irradiation (OR 14.31; 6.98–29.34), abdominal/pelvic radiotherapy (OR 4.19; 2.32–7.55), hypogonadism (OR 2.40; 1.15–4.99), dyslipidemia (OR 3.81; 2.15–6.75) and higher BMI (OR 1.12 per BMI unit; 1.08–1.16) [3]. These are within-study multivariable associations with different reference groups from the Scandinavian and North American analyses.

Table 1. Distinct cohorts evaluating diabetes after childhood cancer treatment

Study	Population	Exposure or characteristic	Effect estimate
Meacham et al., 2009 [1]	8,599 survivors; 2,936 siblings	Total-body irradiation	OR 7.2 (95% CI 3.4–15.0)
Meacham et al., 2009 [1]	Same cohort	Abdominal irradiation	OR 2.7 (1.9–3.8)
ALiCCS, 2025 [2]	Scandinavian case-cohort	Total-body irradiation	IRR 9.8 (2.9–33.7)
ALiCCS, 2025 [2]	Same population	Abdominal irradiation	IRR 4.8 (2.2–10.4)
DCCSS-LATER2, 2025 [3]	2,338 survivors	Total-body irradiation	OR 14.31 (6.98–29.34)
DCCSS-LATER2, 2025 [3]	Same cohort	Abdominal/pelvic radiotherapy	OR 4.19 (2.32–7.55)

Metabolic syndrome, adiposity and risk clustering

In 1,598 participants from the St. Jude Lifetime Cohort, 31.8% fulfilled metabolic syndrome criteria [4]. In the Dutch DCCSS-LATER2 sample of 2,338 survivors, metabolic syndrome was associated with total-body irradiation (OR 6.17; 3.20–11.76), cranial radiotherapy (OR 2.89; 1.67–4.96 at 1–25 Gy) and abdominal radiotherapy above 30 Gy (OR 2.13; 1.01–4.31) [5]. Obesity also varied by sex in the Dutch cohort: 11.2% in men and 15.9% in women, despite overweight prevalences of 46.3% and 44.3%, respectively [6]. Differences in metabolic syndrome definitions, exposure mix and calendar eras complicate comparisons of absolute rates.

Prediabetes may capture risk not evident through obesity screening alone. In a clinically assessed St. Jude cohort of 3,529 survivors and 448 controls, prediabetes was associated with subsequent myocardial infarction (HR 2.4; 1.2–4.8) and chronic kidney disease (HR 2.9; 1.04–8.15), compared with survivors with normoglycemia [7]. This finding supports biochemical screening even in survivors without visibly increased adiposity.

Table 2. Body composition and cardiometabolic endpoints

Source	Survivor population	Endpoint	Reported finding
St. Jude Lifetime Cohort [4]	1,598 adult survivors	Metabolic syndrome	31.8% prevalence
Dutch DCCSS-LATER2 [5]	2,338 adult survivors	MetS after total-body irradiation	OR 6.17 (3.20–11.76)
Dutch DCCSS-LATER2 [6]	2,338 adult survivors	Obesity by sex	11.2% men; 15.9% women
St. Jude clinical cohort [7]	3,529 survivors; 448 controls	Prediabetes and later myocardial infarction	HR 2.4 (1.2–4.8)
HCT survivors [10]	1,885 one-year survivors	Dyslipidemia	823 individuals (43.7%)

Endocrine therapy and new-onset diabetes

In prostate cancer, androgen deprivation produces a different risk profile from radiation-related late effects. A population-based veterans cohort reported diabetes incidence of 159.4 versus 87.5 events per 1,000 person-years in men receiving gonadotropin-releasing hormone agonists versus those without androgen-deprivation therapy, with adjusted HR 1.28 (1.19–1.38) [8]. The larger difference in crude event rates should not be read as an adjusted causal effect, because clinical characteristics and exposure durations differ. Mechanistic studies report insulin resistance and adverse adipokine profiles accompanying androgen deprivation [9]. For breast cancer, published synthesis suggests a more modest association. A hormone-therapy meta-analysis reported an HR of 1.30 (1.20–1.40) for diabetes with tamoxifen versus breast-cancer nonusers and 1.18 (1.12–1.24) versus noncancer controls [12]. These comparisons answer different questions and may be influenced by menopause, cancer stage, baseline adiposity, chemotherapy coexposure and surveillance intensity. A nationwide Danish cohort further investigated long-term diabetes risks by treatment modality and diagnosis period [13].

Immune checkpoint inhibitors and endocrine organ toxicity

Checkpoint-inhibitor toxicities include immune-mediated thyroid dysfunction, hypophysitis, adrenal insufficiency and occasionally insulin-deficient diabetes. A randomized-trial meta-analysis estimated hypothyroidism RR 7.81 (5.68–10.74), pooled hypothyroidism incidence 7.64% (6.23–9.17), and insulin-dependent diabetes RR 1.52 (1.07–2.18) with pooled incidence 0.087% (0.019–0.189) [14]. A separate review of new checkpoint-inhibitor regimens reported hypothyroidism 10.1%, thyrotoxicosis 4.6%, hypophysitis 0.8% and adrenal insufficiency 0.9% [15]. Different therapy regimens and eligibility criteria explain why these rates must not be merged. In a separate PD-1 inhibitor analysis, pembrolizumab was associated with hypothyroidism (RR 4.76; 3.55–6.39), thyroiditis (RR 5.95; 3.02–11.72) and adrenal insufficiency (RR 4.80; 2.60–8.88) compared with controls [16]. These estimates describe treated oncology populations during trials, not necessarily the prevalence of persistent endocrine disease years into survivorship.

Table 3. Treatment-specific endocrine and glycemic associations

Treatment	Published evidence	Outcome	Estimate
GnRH agonist ADT [8]	Veterans comparative cohort	Incident diabetes	aHR 1.28 (1.19–1.38)
Tamoxifen [12]	Published breast-cancer meta-analysis	Diabetes vs breast-cancer nonusers	HR 1.30 (1.20–1.40)
Checkpoint blockade [14]	RCT meta-analysis	Hypothyroidism risk	RR 7.81 (5.68–10.74)
Checkpoint blockade [14]	RCT meta-analysis	Insulin-dependent diabetes risk	RR 1.52 (1.07–2.18)
Newer checkpoint agents [15]	Published meta-analysis	Hypophysitis incidence	0.8% (0.5–1.1%)
Pembrolizumab [16]	PD-1 trial meta-analysis	Adrenal insufficiency risk	RR 4.80 (2.60–8.88)

Hematopoietic cell transplantation and multidomain risk

Transplant recipients accumulate multiple cardiometabolic exposures. In an analysis of 1,885 hematopoietic cell transplantation survivors, dyslipidemia was documented in 823 (43.7%) [10]. A southern Indian cross-sectional comparison of 63 allogeneic transplant recipients and 65 matched controls reported prediabetes in 21% and diabetes in 7% of recipients [11]. Post-transplantation medication, total-body irradiation, graft-versus-host disease and baseline risk can coexist; attributing an abnormality to one agent is rarely possible without longitudinal confounder adjustment.

Cross-cutting interpretation of outcomes

The strongest and most reproducible late-effect signal in pediatric survivor cohorts is radiation-associated diabetes, especially after total-body and abdominal exposure. Risks arising during endocrine therapy or immunotherapy operate on different time scales and require different diagnostic pathways. Cardiometabolic screening should therefore not be limited to body mass index, and endocrine dysfunction should not be inferred from fatigue or weight change without targeted biochemical confirmation.

Table 4. Exposure-specific survivorship surveillance considerations

Exposure	Principal concern	Suggested follow-up domains
Abdominal or total-body irradiation	Delayed diabetes, central adiposity	Fasting glucose or HbA1c; lipid profile; blood pressure
Cranial radiotherapy	Hypothalamic-pituitary injury	Growth and gonadal history; targeted pituitary testing
Androgen deprivation	Insulin resistance and unfavorable fat/lean mass	Weight and waist; glucose; lipids; cardiovascular risk
Breast endocrine therapy	Diabetes susceptibility in selected groups	Baseline metabolic risk and longitudinal glucose review
Checkpoint inhibition	Thyroiditis, adrenal insufficiency, insulin-deficient diabetes	Thyroid function; symptom-triggered cortisol/glucose evaluation
Hematopoietic transplantation	Multiple cardiometabolic late effects	Glucose, lipids, blood pressure and endocrine review

Study characteristics and outcome definitions

Evidence spanned multinational childhood survivor cohorts, national health-record linkages, adult breast and prostate cancer populations, hematopoietic transplantation follow-up, and randomized immune checkpoint inhibitor safety syntheses. Reported denominators varied from small clinically characterized survivor groups to population-level cohorts. Metabolic syndrome, waist circumference, insulin resistance, laboratory-defined diabetes and administrative diagnostic codes were not interchangeable measurements. Accordingly, differences in absolute outcome frequency reflected both clinical exposures and differences in assessment methods [26–31].

Breast cancer endocrine therapy and glycemic outcomes

Diabetes associations differed by endocrine agent, comparator and follow-up. A Danish population-based study reported adjusted diabetes hazard ratios of 1.25 (95% CI 1.18–1.32) for aromatase inhibitors and 1.05 (95% CI 0.99–1.11) for tamoxifen compared with cancer-free women [34]. Another breast cancer case-cohort study reported higher hazards for aromatase inhibitor and tamoxifen exposure, but the study designs, risk definitions and comparison groups differed and do not support a single treatment-ranking estimate [32]. Related cohorts and reviews report variation by age, treatment duration and baseline cardiometabolic risks [33,37].

Body composition and metabolic risk during androgen deprivation

Men starting androgen deprivation therapy may experience shifts in body composition, abdominal adiposity, glycemic regulation and lipoprotein concentrations. In a prospective multicenter investigation enrolling 539 men, increases in waist circumference, body mass index, fasting glucose and several lipid fractions were documented after six and twelve months of androgen suppression [35]. However, a more recent 24-week prospective investigation detected unfavorable body-composition changes without a significant parallel change in measured insulin resistance, illustrating the importance of endpoint and duration [36].

Endocrine toxicity of checkpoint blockade

Immune checkpoint blockade was linked to heterogeneous thyroid, pituitary, adrenal and pancreatic endocrine events. In a published meta-analysis of 38 randomized trials comprising 7,551 patients, endocrine toxicity varied substantially across PD-1, CTLA-4 and combination regimens [38]. Other systematic reviews documented increased hypothyroidism, hyperthyroidism, hypophysitis and adrenal insufficiency risks, with potentially different clinical trajectories and treatment requirements [39,40]. Acute immune endocrinopathies should be differentiated from persistent late effects in long-term survivor cohorts.

Bias, heterogeneity and confidence in findings

Across radiotherapy studies, wide confidence intervals for uncommon exposures and variation in survivor selection limited precision. Observational endocrine-therapy comparisons remained susceptible to confounding from age, adiposity, hormone-receptor status and competing health conditions. Immune checkpoint randomized-trial analyses provided stronger treatment-comparison designs but frequently used adverse-event reporting rather than standardized long-term biochemical assessment. Thus, the direction and strength of association were interpreted within each outcome domain instead of assuming a common effect across treatment types [26–29,38–40].

DISCUSSION

Principal findings

This review separates distinct biological and epidemiological effects rather than claiming a single prevalence of post-cancer metabolic disease. The magnitude of diabetes association is particularly pronounced after total-body irradiation, while endocrine treatment and checkpoint blockade show different and generally smaller or outcome-specific risk signals. Observational risk estimates cannot be interpreted as randomized causal contrasts; immune-related endocrine event rates cannot be applied wholesale to long-term survivors [1–3,8,12,14].

Clinical implications

An exposure-directed follow-up protocol may be more efficient than uniform annual endocrine testing. For survivors exposed to abdominal or total-body irradiation, early and repeated glucose assessment is justified; for pituitary-axis exposures, symptoms and treatment history should direct endocrine testing. During androgen deprivation and breast endocrine therapy, clinical attention should include adiposity changes, glycemic status and cardiovascular risk. For checkpoint blockade, the potential for acute adrenal crisis or insulin-deficient diabetes calls for timely evaluation of new symptoms [14–17].

Sources of heterogeneity and strengths

Key differences include survivor selection, age and era of therapy, pre-treatment metabolic health, treatment combinations, dose, time elapsed, ascertainment and competing mortality. Clinical measurement identifies dysglycemia missed by self-report; administrative codes depend on healthcare contact. This review preserves the original exposure and outcome scales, distinguishing incident risk from cross-sectional burden and avoiding false cross-treatment rankings.

Limitations

The literature reviewed was targeted rather than independently exhaustive, and a completed multi-database search audit, duplicate extraction and formal risk-of-bias adjudication were not available. Some studies from the same survivor platforms are statistically dependent; therefore their participant totals and effect estimates were not pooled.

CONCLUSION

Diabetes, metabolic syndrome, body-composition change and endocrine dysfunction are important but heterogeneous consequences of cancer treatment. High-risk radiation exposures, endocrine therapies and immune checkpoint blockade each generate clinically distinct follow-up needs. Long-term surveillance should integrate cancer history, treatment doses, endocrine symptoms and objective cardiometabolic measurements to enable earlier recognition and management.

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