

SPECIAL REPORT

Post-diagnosis dietary factors, supplement use and colorectal cancer prognosis: A Global Cancer Update Programme (CUP Global) systematic literature review and meta-analysis

Doris S. M. Chan¹  | Margarita Cariolou¹ | Georgios Markozannes^{1,2}  |
 Katia Balducci¹ | Rita Vieira¹ | Sonia Kiss¹ | Nerea Becerra-Tomás¹ |
 Dagfinn Aune^{1,3,4}  | Darren C. Greenwood⁵ | Esther M. González-Gil⁶ |
 Ellen Copson⁷ | Andrew G. Renehan⁸ | Martijn Bours⁹ |
 Wendy Demark-Wahnefried¹⁰  | Melissa M. Hudson¹¹ | Anne M. May¹² |
 Folakemi T. Odedina¹³ | Roderick Skinner¹⁴ | Karen Steindorf¹⁵ |
 Anne Tjønneland^{16,17} | Galina Velikova¹⁸ | Monica L. Baskin¹⁹ |
 Rajiv Chowdhury²⁰ | Lynette Hill²¹ | Sarah J. Lewis²² | Jaap Seidell²³ |
 Matty P. Weijenberg⁹ | John Krebs²⁴ | Amanda J. Cross¹ |
 Konstantinos K. Tsilidis^{1,2} 

Correspondence

Doris S. M. Chan, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, St. Mary's Campus, Norfolk Place, London, W2 1PG, UK.
 Email: d.chan@imperial.ac.uk

Funding information

World Cancer Research Fund; Wereld Kanker Onderzoek Fonds; American Institute for Cancer Research

Abstract

The role of diet in colorectal cancer prognosis is not well understood and specific life-style recommendations are lacking. We searched for randomised controlled trials (RCTs) and longitudinal observational studies on post-diagnosis dietary factors, supplement use and colorectal cancer survival outcomes in PubMed and Embase from inception until 28th February 2022. Random-effects dose-response meta-analyses were conducted when at least three studies had sufficient information. The evidence was interpreted and graded by the CUP Global independent Expert Committee on Cancer Survivorship and Expert Panel. Five RCTs and 35 observational studies were included (30,242 cases, over 8700 all-cause and 2100 colorectal cancer deaths, 3700 progression, recurrence, or disease-free events). Meta-analyses, including 3–10 observational studies each, were conducted for: whole grains, nuts/peanuts, red and processed meat, dairy products, sugary drinks, artificially sweetened beverages, coffee, alcohol, dietary glycaemic load/index, insulin load/index, marine omega-3

Doris S. M. Chan and Margarita Cariolou contributed equally and share first authorship.

Where authors are identified as personnel of the International Agency for Research on Cancer/World Health Organization, the authors alone are responsible for the views expressed in this article and they do not necessarily represent the decisions, policy, or views of the International Agency for Research on Cancer/World Health Organization.

For affiliations refer to page 464

This is an open access article under the terms of the [Creative Commons Attribution](https://creativecommons.org/licenses/by/4.0/) License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

© 2024 The Authors. *International Journal of Cancer* published by John Wiley & Sons Ltd on behalf of UICC.

polyunsaturated fatty acids, supplemental calcium, circulating 25-hydroxyvitamin D (25[OH]D) and all-cause mortality; for alcohol, supplemental calcium, circulating 25(OH)D and colorectal cancer-specific mortality; and for circulating 25(OH)D and recurrence/disease-free survival. The overall evidence was graded as 'limited'. The inverse associations between healthy dietary and/or lifestyle patterns (including diets that comprised plant-based foods), whole grains, total, caffeinated, or decaffeinated coffee and all-cause mortality and the positive associations between unhealthy dietary patterns, sugary drinks and all-cause mortality provided 'limited—suggestive' evidence. All other exposure-outcome associations provided 'limited—no conclusion' evidence. Additional, well-conducted cohort studies and carefully designed RCTs are needed to develop specific lifestyle recommendations for colorectal cancer survivors.

KEYWORDS

colorectal cancer survival, diet, evidence grading, food, systematic review

What's new?

The role of diet in colorectal cancer prognosis is not well understood. As part of CUP Global, here the authors systematically reviewed, meta-analysed, and independently graded the quality of evidence on the associations of post-diagnosis dietary intake, dietary patterns, and supplement use with colorectal cancer prognosis. Whilst the overall evidence was graded as 'limited', it suggested that consuming a healthy diet, including diet patterns with plant-based foods, and avoiding sugary drinks may be associated with improved overall survival after colorectal cancer diagnosis. The study calls for well-designed cohort and intervention studies to help develop lifestyle recommendations for colorectal cancer survivors.

1 | INTRODUCTION

Colorectal cancer is the third most diagnosed malignancy after lung and breast cancer,^{1,2} and ranks second in terms of cancer-related deaths in men and women worldwide.¹ Globally, in 2020, there were more than 1.9 million colorectal cancer cases and more than 0.9 million colorectal cancer deaths.³ By 2040, incident colorectal cancer cases and colorectal cancer-related deaths are expected to reach 3.2 and 1.6 million, respectively.¹ Possible reasons for the rise in cases include adoption of a westernised diet and lifestyle, population growth and extended lifespan.^{4,5}

Colorectal cancer has an overall 5-year relative survival rate of at least ~60% in higher income countries,⁶ and a lower rate, on average ~40%, in lower-income settings.^{7,8} Advancements in detection^{9,10} and treatment services¹¹ have led to greater numbers of colorectal cancer survivors, especially in most developed countries.^{6,12} There were an estimated 5.2 million colorectal cancer survivors living within 5 years of diagnosis in 2020.³ Extended survival, however, co-exists with increased co-morbidities,¹³ including cardiovascular disease, the most common cause of non-cancer deaths.¹⁴ Colorectal cancer survivors are at risk of recurrence, metastasis^{15,16} or second primary cancers.^{17,18} Colorectal cancer will continue posing an enormous global health burden and financial challenge across health systems.^{4,5}

Prevention strategies through lifestyle modifications may improve cancer survivorship. Numerous lifestyle factors have been identified for their involvement in colorectal cancer development. The World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) Third Expert Report identified 'strong—probable' evidence that consuming red meat increases, and consuming whole grains, dietary fibre, dairy products, and calcium supplements reduces the risk of colorectal cancer and 'strong—convincing' evidence that consumption of processed meat and alcohol increases risk. Evidence on other dietary factors was limited.¹⁹ A recent umbrella review also reported strong meta-analytic evidence for alcohol intake and higher risk of colorectal cancer and for calcium, whole grain, and dairy product intake and lower risk of colorectal cancer.²⁰ However, evidence on how dietary factors could influence survival is currently limited. Such knowledge is essential to develop targeted dietary guidance/recommendations for colorectal cancer survivors. Currently, cancer survivors are advised to follow general cancer and chronic disease prevention guidelines.^{19,21} Various recent meta-analyses of observational studies have investigated associations between post-diagnosis dietary exposures and colorectal cancer survival but included a generally low number of studies.^{22–25} Categorical meta-analyses showed inverse associations between post-diagnosis whole grain²³ and coffee²³ intake, the American Cancer Society recommendations (ACS-score)²³ and all-cause mortality, between a prudent dietary

pattern,²³ calcium supplementation²⁵ and colorectal cancer-specific mortality. Positive associations were observed for an unhealthy dietary pattern and all-cause and colorectal cancer-specific mortality as well as for the Dietary Inflammatory Index²³ and all-cause mortality. The most recent meta-analysis²⁶ of circulating 25(OH)D concentrations and colorectal cancer outcomes reported inverse associations for all-cause and colorectal cancer-specific mortality but included a mixture of studies with pre- or post-diagnosis 25(OH)D assessment. A meta-analysis of two randomised controlled trials (RCTs) on vitamin D3 supplementation reported a favourable effect on colorectal cancer progression or death.²⁷ Most of the meta-analyses on dietary factors to date focused on categorical analyses and did not explore non-linearity. As part of the WCRF Global Cancer Update Programme (CUP Global), formerly known as the WCRF/AICR Continuous Update Project, we conducted comprehensive systematic literature reviews (SLRs) and meta-analyses to evaluate the magnitude and the shape of the associations of interest. The evidence was subsequently independently interpreted and graded by the CUP Global Expert Committee and Expert Panel.²⁸ This paper presents the evidence on post-diagnosis dietary factors, supplement use and colorectal cancer outcomes. Evidence on body fatness, physical activity, and the overall summary is presented in the accompanying papers.^{29–31}

2 | METHODS

The present systematic review was conducted following a standard pre-published CUP Global protocol.³² Details on the methods and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist are available in Supplementary Text S1 and Table S1.

2.1 | Search strategy, selection criteria and data extraction

We searched in PubMed and Embase from inception to 28th February 2022, and screened through relevant reference lists, for publications that met the following inclusion criteria: (1) RCTs with at least 6 months duration, longitudinal observational studies, or pooled analyses of the aforementioned designs that (2) included at least 100 participants; (3) examined dietary exposures which were assessed at/after diagnosis (in a few studies diet was recalled shortly after diagnosis in some of their participants and the assessment may have included the pre-diagnosis period^{33–37}) that is, dietary patterns, foods, circulating 25(OH)D, beverages, macro- and micronutrients and dietary supplements (any vitamins, minerals, herbs or other botanicals, amino acids, or other dietary substances or their constituents³⁸). All exposures are being referred to as 'post-diagnosis' in the current manuscript for brevity. The examined colorectal cancer outcomes were all-cause mortality, cause-specific mortality, progression/recurrence/disease-free survival, any second primary cancers. In the case of multiple publications by the same study on the same exposure-outcome association, the publication with the greater number of

outcome events was used. Studies that examined solely nutrient-based patterns or were lacking information on the foods and beverages contributing to the dietary pattern were excluded because the information could not be used towards the development of food-based dietary recommendations. Study and participants' characteristics and the results for each exposure-outcome association were extracted into the CUP Global database.

2.2 | Systematic literature review

RCTs and observational studies were reviewed separately. Relevant publications were meta-analysed when at least three studies were available for a given exposure, or descriptively synthesised for the studies that did not report sufficient information for a meta-analysis.

The RCTs and the publications on dietary exposures related to the WCRF/AICR Cancer Prevention Recommendations¹⁹ were descriptively synthesised even when there were fewer than three available studies. For descriptive synthesis, the results for each exposure-outcome association reported in the individual studies were summarised in text and the relative risks (RRs) comparing extreme exposure categories were presented in forest plots. The various dietary and/or lifestyle patterns were grouped into 'healthy' and 'unhealthy' to explore if there was any tendency of associations.

2.3 | Statistical methods for meta-analysis

Linear dose-response meta-analyses were conducted to calculate summary RRs and 95% confidence intervals (CIs) from multivariable adjusted results for a continuous exposure or for exposures with at least three categories reported in the individual studies. A Der Simonian-Laird random-effects model was used.³⁹ Heterogeneity was assessed using the estimate of between study variance (τ^2)⁴⁰ and reflected by the range of effect estimates provided in the forest plots. The proportion of total variability in effect estimates due to between-study heterogeneity was assessed using the I^2 statistic.⁴¹ The 95% predictive intervals (PIs) were used to estimate the range of values that may contain the value of a new study.⁴² Sources of heterogeneity were explored when there were at least three studies in pre-defined subgroups (cancer subsite and subtype, exposure assessment time-frame relative to cancer treatment, and risk of bias domains). Small-study effects, a reason of which is publication bias, were examined using the Egger's test⁴³ and by visually inspecting funnel plots with 10 or more studies. One-stage non-linear dose-response meta-analysis was conducted using restricted cubic splines with three knots placed at 10th, 50th, and 90th percentiles of the overall dose distribution (based on the estimated or reported category midpoints of the included studies).⁴⁴ This was done when at least five studies with three or more exposure categories were available. The likelihood ratio test was used to compare the linear and non-linear models.⁴⁵ Sensitivity meta-analyses, including leave-one-out analysis to examine the influence from each study on the summary estimate⁴⁶ and analyses

excluding locally advanced and metastatic patients were conducted when appropriate. Hypothesis testing and *p*-values reported are two-tailed, unless otherwise mentioned.

2.4 | Risk of bias assessment

Version 2 of the Cochrane risk-of-bias tool for randomised trials (RoB 2) was used to assess the risk of bias in individual RCTs,⁴⁷ whereas a modified version of the Risk of Bias for Nutrition Observational Studies (RoB-Nobs) tool⁴⁸ was used to assess the risk of bias in observational studies of dietary and/or lifestyle patterns and in other observational studies included in the meta-analyses. The RoB-Nobs tool was originally developed by the U.S. Department of Agriculture (USDA) Nutrition Evidence Systematic Review after modifications to the Cochrane's collaboration Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I).⁴⁹ The Imperial College London (ICL) review team further refined and tested the tool to ensure its suitability for investigating exposure-outcome associations in cancer survivorship studies. This involved adapting the tool's prompting questions and providing additional guidance to encompass adiposity, physical activity, and dietary/nutritional exposures (working document version dated 11/07/2023 can be found in Supplementary Table S2). The tool consists of seven domains, including confounding, participant selection, exposure classification, departures from intended exposures, missing data, outcome measurement, and selective reporting. The studies not included in the meta-analyses were assessed descriptively considering the most likely influential sources of potential bias in survival studies⁵⁰ (selection bias, information bias of exposure and outcome assessment, and residual confounding).

2.5 | Quality control

Study selection, data extraction, and risk of bias assessment were checked by a second reviewer. Any disagreements were resolved by consensus with a third reviewer.

2.6 | Evidence grading criteria

The findings from the systematic review were interpreted by the CUP Global independent Expert Committee on Cancer Survivorship and the Expert Panel convened by WCRF International. The Expert Committee provided preliminary conclusions on the evidence, and these were finalised by the Expert Panel independently of the ICL review team. Pre-defined evidence grading criteria, covering the quantity, consistency, magnitude and precision of the summary estimates, existence of a dose-response relationship, risk of bias, study design, generalisability and mechanistic plausibility of the results, were used to assess the quality of the evidence evaluating likelihood of causality into 'strong (subgrades: convincing, probable, substantial effect on risk unlikely)' or 'limited (subgrades: limited-suggestive or limited-no conclusion)' (Supplementary Table S3).

3 | RESULTS

3.1 | Literature search and study characteristics

Figure 1 shows the study selection process. There were 92 potentially eligible publications investigating dietary factors and colorectal cancer outcomes, 23 of which were excluded for not meeting inclusion criteria^{51–73} (Supplementary Table S4). The present systematic review included 69 publications, from five RCTs (6 publications)^{74–79} and 35 observational studies (63 publications),^{33–37,80–137} comprising 30,242 colorectal cancer cases and over 8700 all-cause deaths, 2100 colorectal cancer deaths, and 3700 progression, recurrence, or disease-free events.

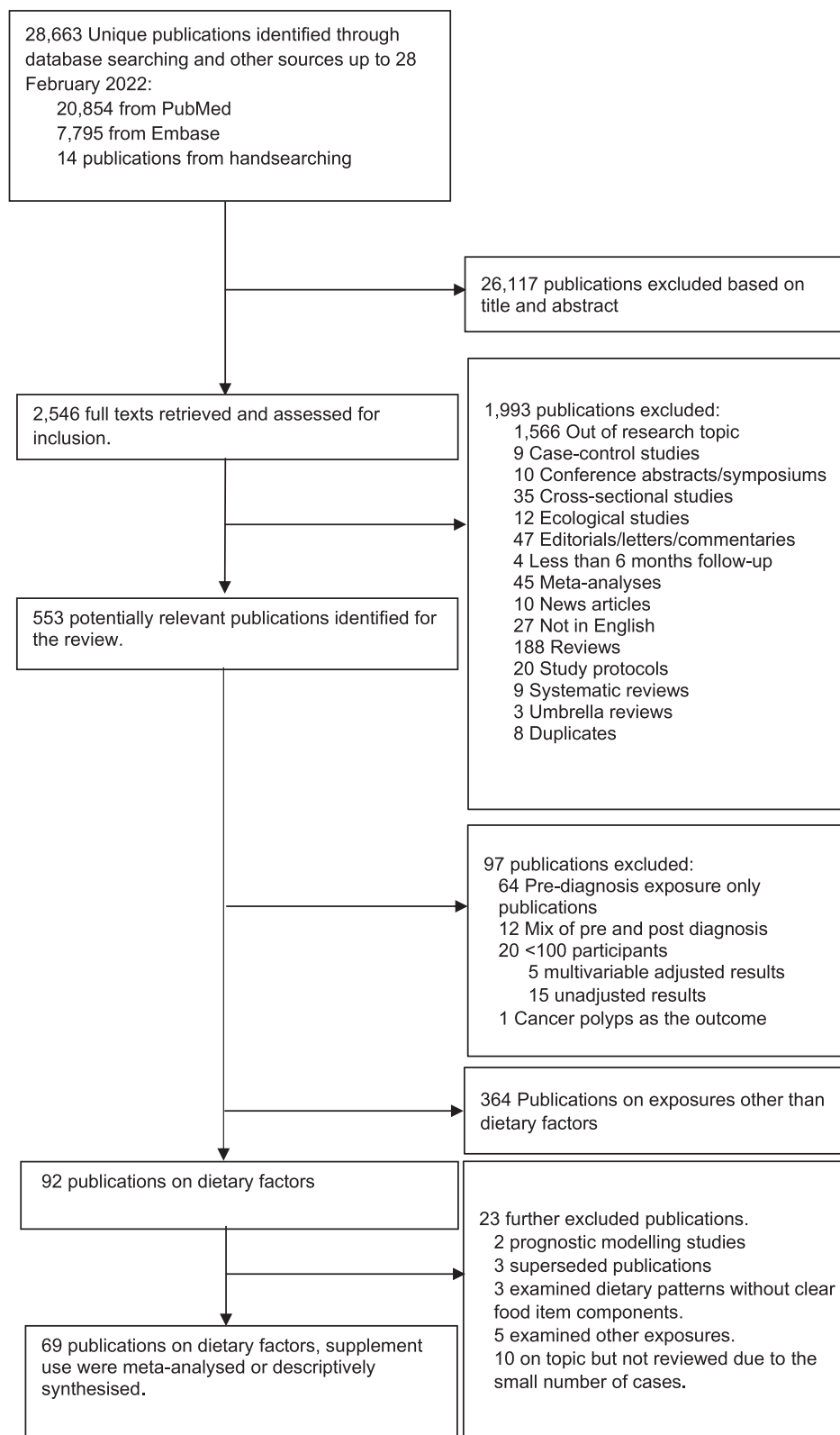
Fifteen studies (38%) (47 publications) were from North America^{33,36,75–77,80,82–87,89–91,93,94,96–111,114–121,123,124,127,131,135,136} and eleven (26%) (13 publications) were from Europe,^{35,37,74,79,81,88,92,125,128,130,132–134} which included cancer survivors of mostly white race or ethnicity. Six studies (15%) (6 publications) were from South-East Asia,^{78,95,113,126,129,137} one (3%) from East Africa (Ethiopia)¹¹² and seven (18%) (2 publications) were international studies.^{34,122} Thirty-six studies (90%) (52 publications) investigated colorectal cancer^{34–37,74–81,83–85,87–90,92,94,95,98,101,102,104,106–108,110–115,117,120–122,124–126,128–137} and four (10%) (17 publications) only colon cancer.^{33,82,86,91,93,96,97,99,100,103,105,109,116,118,119,123,127}

Twenty-seven studies (67%) (42 publications) included individuals with colorectal cancer at various stages,^{34,35,37,74,78–81,83,85,87–90,92,94,95,98,101,102,104,106,108,110–115,117,120,121,124–126,128–130,132,134,135,137} of which 17 publications included stage IV cancer survivors^{37,74,79,87,88,90,92,95,112–114,121,125,130,132,135,137} (median percentage [range] of stage IV: 16% [3.5%–29%]; five of the 17 publications did not report % metastatic^{74,79,87,90,113}).

One (2%) study included stage II patients only,³³ three (8%) (16 publications) stage III only,^{82,86,91,93,96,97,99,100,103,105,109,116,118,119,123,127} two (5%) (5 publications) locally advanced or metastatic stage only,^{76,77,84,107,131} and seven (18%) (5 publications)^{36,75,122,133,136} stage IV only. Study size ranged from 100 to 2910 participants (median 611 participants). Most studies conducted dietary or blood measurement once at various times up to a median of 6 years after cancer diagnosis.^{88,92}

3.2 | Randomised controlled trials

Five RCTs (6 publications) were identified, one of each on a nutritional behavioural intervention and high-protein supplementation,⁷⁹ omega-3 fatty acid supplementation,⁷⁴ and vitamin C supplementation,⁷⁵ and two (3 publications) on vitamin D3 supplementation^{76–78} (Table 1, Supplementary Figures S1 and S2). Two RCTs included unresectable advanced/metastatic patients,^{75–77} two included stage I–IV patients before resection,^{74,79} and one included stage I–III resected patients.⁷⁸ In the SUNSHINE trial,^{76,77} high-dose (*n* = 69; 4000 IU/day) versus regular-dose (*n* = 70; 400 IU/day) vitamin D3 supplementation did not affect overall survival (median 24.3 months for both) but suggested improved progression-free survival (hazard ratio [HR] = 0.64,

FIGURE 1 Flowchart of study selection process.

one-sided 95% CI = 0.00–0.90). A similar but less precise estimate was observed in the AMATERASU trial⁷⁸ that compared high-dose ($n = 251$; 2000 IU/day) versus placebo ($n = 166$) (HR = 0.69, 95% CI = 0.39–1.24). Vitamin C supplementation ($n = 51$; 10 g/day) versus placebo ($n = 49$) worsened both overall (HR = 1.25, one-sided

$p = .017$) and progression-free survival (median 2.9 vs. 4.1 months, one-sided $p = .01$).⁷⁵ Use of omega-3 fatty acid enriched supplements ($n = 74$; 2 g eicosapentaenoic acid [EPA]/day and 1 g docosahexaenoic acid [DHA]/day) versus omega-3 fatty acid supplements without fish oil ($n = 74$) resulted in worse 5-year overall survival (HR = 1.73,

TABLE 1 Descriptive table of dietary randomised controlled trials and colorectal cancer outcomes.

Author, year, study name, country	Characteristics of study population	Design, randomisation, blinding	Intervention and timeframe	Follow-up time, compliance	Outcome	RR (95% CI)	Adjustments	Risk of bias
Sørensen 2020 ⁷⁴ NCT00488904, Denmark PMID:32391656	Patients scheduled for elective resection of colorectal cancer (n = 148) Stage: I–IV Age: mean (experimental) 68.3 and (control) 70.6 years Race/Ethnicity: Unknown	Parallel Double-blinded Randomisation ratio: 1:1 (74:74)	Experimental group (n analysed = 65): Omega-3 enriched oral supplement (fish oil, 2 g EPA/day and 1 g DHA/day) Control group (n analysed = 60): Isocaloric, isonitrogenous, same amount of carbohydrate, protein, total fat, omega-6 fatty acids, vitamin, and minerals but no added fish oil 7 days before and 7 days after surgery.	Maximum follow-up: 5 years Loss to follow-up: 0% Adherence with the allocated study groups was 'acceptable', no further info on any changes to regular diet	Primary endpoint Overall survival (5-year) Primary endpoint Recurrence-free survival (local or metastatic colorectal cancer recurrence, 3-year)	Post-hoc analysis 1.73 (1.06–2.83) p = .03 1.66 (0.65–4.26)	Adjuvant chemotherapy, age, stage at diagnosis None (adjustment for adjuvant chemotherapy, stage at diagnosis did not alter the estimate)	Domain 1: Low risk Domain 2: Some concerns Domain 3: Low risk Domain 4: Low risk Domain 5: Low risk Overall: Some concerns
Ng, 2019/Brown, 2020 ^{76,77} The SUNSHINE RCT, USA PMID: 30964527/ 33233566	Patients with unresectable colorectal cancer (n = 139) Stage: Locally advanced or metastatic Age: mean 56 years Race/Ethnicity: White 77%, Black 7.2%, Asian 0.7%, >1 race 2.2%, other 12.9%	Parallel Double-blinded (unblinded statistician and research pharmacist) Computerised block randomisation Randomisation ratio: 1:1 (69:70)	Experimental group (n = 69): Chemotherapy + high dose oral vitamin D3 supplement (8000 IU/day for cycle 1 and 4000 IU/day for the rest) Control group (n = 70): Chemotherapy + standard dose oral vitamin D3 supplement (400 IU/day) From start of chemotherapy until disease progression,	Median follow-up: 22.9 months Loss to follow-up: 2.9% Adherence to vitamin D3 was high, with a median of 98% of expected capsules taken. No further monitoring after cancer progression.	Secondary endpoint Overall survival (high dose: 45 deaths, standard dose: 54 deaths) Primary endpoint Progression-free survival (disease progression or death; high dose: 49 events, standard dose: 62 events)	Median 24.3 months versus 24.3 months Log-rank p = .43 Median 13.0 (10.1–14.7) months versus 11.0 (9.5–14.0) months Log-rank P = 0.07 Supportive analysis 0.64 (0.0–0.90) (one-sided) p = .02	None None Age, ECOG performance, number of metastatic sites, race, sex	Domain 1: Some concerns Domain 2: Low risk Domain 3: Low risk Domain 4: Some concerns Domain 5: Low risk Overall: Some concerns Domain 1: Some concerns Domain 2: High risk Domain 3: Low risk Domain 4: Some concerns Domain 5: Low risk Overall: High risk

TABLE 1 (Continued)

Author, year, study name, country	Characteristics of study population	Design, randomisation, blinding	Intervention and timeframe	Follow-up time, compliance	Outcome	RR (95% CI)	Adjustments	Risk of bias
Urashima 2019, ⁷⁸ The AMATERASU RCT, Japan, PMID: 30964526	Patients with resected colorectal cancer, among other digestive tract cancer survivors (n = 201/417) Stage: I–III Age (overall): median 66 years, range 35–90 years Race/Ethnicity: Unknown	Parallel Double-blinded (unblinded for the Primary investigator and the data monitoring centre staff) Computerised block randomisation Randomisation ratio: 3:2 (251:166)	Experimental group (n = 122): Vitamin D3 oral supplement (2000 IU/day) Control group (n = 79): Placebo After surgery, for 7 years.	Overall median follow-up 3.5 years Loss to follow-up 0.02% Self-reported adherence (overall 9.6% and 11.4% discontinued treatment)	Primary endpoint Relapse-free survival (cancer relapse or death due to any cause)	Post-hoc analysis: 0.69 (0.39–1.24) p = .22	None	Domain 1: High risk Domain 2: Some concerns Domain 3: Low risk Domain 4: High risk Domain 5: Low risk Overall: High risk
Ravasco 2012, ⁷⁹ Portugal PMID: 23134880	Patients eligible for radiotherapy (n = 111) Stage: I–IV Age: mean 64 years Race/Ethnicity: Unknown	Parallel Unblinded Stratified (randomisation (cancer stage) Randomisation ratio: 1:1:1 (37:37:37)	Group 1 (n = 34): Nutrition counselling and education Group 2 (n = 29): High-protein (40 g/day) dietary supplements + usual diet Group 3 (n = 26): Usual diet During the 1.5 months of radiotherapy	Median follow-up 6.5 years Loss to follow-up 19.8% Adherence: not reported	Secondary endpoint Cancer-specific survival (group 1: 3 deaths, group 2: 8 deaths, group 3: 11 deaths) Secondary endpoint Recurrence (group 1: 7 events, group 2: 9 events, group 3: 15 events)	Post-hoc analysis: Median 7.3 years (group 1) versus 6.5 years (group 2) versus 4.9 years (group 3) p < .05 Post-hoc analysis: p < .01	Unclear, may include age, cancer stage, follow-up time, disease recurrence, adjuvant treatments, median survival, and number of patients in each group	Domain 1: High risk Domain 2: Low risk Domain 3: Low risk Domain 4: Low risk Domain 5: Some concerns Overall: High risk Domain 1: High risk Domain 2: Low risk Domain 3: Low risk Domain 4: High risk Domain 5: Some concerns Overall: High risk
Moertel 1985, ⁷⁵ USA PMID: 3880867	Patients with unresectable colorectal cancer who had not received chemotherapy and were beyond hope of curative treatment (n = 101)	Parallel Double-blinded Stratified (randomisation (metastasis location and time from diagnosis to randomisation) Randomisation ratio: 1:1 (51:49)	Experiment group (n = 51): Vitamin C supplement (10 g/day) Control group (n = 49): Placebo (lactose) From one-month post-surgery and continued as long	Maximum follow-up 27 months Loss to follow-up 0% Adherence was > 75% in all but 8 patients	Primary endpoint Overall survival Primary endpoint Progression-free survival (increase > 50% of malignant area; appearance of new malignant areas; worsening symptoms or performance)	1.25 (NA) p = .017 (one-sided) Median 2.9 months versus 4.1 months p = .01 (one-sided)	Adjusted for histological grade, and stratified for metastasis location and time from diagnosis to randomisation	Domain 1: Low risk Domain 2: Some concerns Domain 3: Low risk Domain 4: Low risk Domain 5: Low risk Overall: Some concerns

(Continues)

TABLE 1 (Continued)

Author, year, study name, country	Characteristics of study population	Design, randomisation, blinding	Intervention and timeframe	Follow-up time, compliance	Outcome	RR (95% CI)	Adjustments	Risk of bias
	Stage: Advanced (metastasis to lung, liver, or other sites) Age: Unknown Race/Ethnicity: Unknown		as possible or until evidence of marked colorectal cancer progression		status; >10% body weight loss)			

Abbreviations: CI, confidence interval; DHA, docosahexaenoic acid; ECOG, Eastern Cooperative Oncology Group; EPA, eicosapentaenoic acid; NA, not applicable; P, probability value; PMID, PubMed ID; RCT, randomised controlled trial; RR, relative risk.

95% CI = 1.06–2.83) and 3-year recurrence-free survival of similar size but with a wide 95% CI crossing the null (HR = 1.66; 95% CI = 0.65–4.26).⁷⁴ Colorectal cancer survivors who received individualised nutritional counselling had the longest colorectal cancer-specific survival and lowest number of recurrences compared with those who received high-protein supplements (40 g/day) or usual diet (median 7.3, 6.5, 4.9 years, respectively, $p < 0.05$ and $n = 7/34, 9/29, 15/26$, respectively, $p < 0.01$).⁷⁹

3.3 | Observational studies

Evidence on dietary and/or lifestyle patterns and colorectal cancer outcomes were grouped to observe any tendency of associations (Supplementary Tables S5 and S6). Linear dose–response meta-analyses were possible for 21 dietary exposures–colorectal cancer survival outcome associations investigated (Figure 2). All other associations identified were descriptively reviewed. Subgroup analysis, sensitivity analysis, and test of publication bias was not possible, except for the few occasions as presented. Study and participants' characteristics and the main results of the studies included are provided in Supplementary Tables S7–S41. An overview of the risk of bias assessment of publications from observational studies is provided in Supplementary Figures S3 and S4. A summary of the evidence grading conclusions is provided in Table 2.

3.3.1 | Post-diagnosis dietary and lifestyle patterns combined

Two pooled analyses of two cohorts^{80,81} and two additional studies^{86,90} (4 publications) (1187 deaths) investigated four a priori healthy dietary and lifestyle patterns, defined based on recommendations for cancer prevention or a healthy lifestyle, which included diet, physical activity, and adiposity as components (Supplementary Tables S7–S9, Figures S5 and S6). One pooled analysis investigated three patterns,⁸¹ other studies^{80,86,90} investigated one pattern only, thus the four independent study groups reported six dietary and lifestyle patterns and all-cause mortality point estimates (Supplementary Table S5). The evidence was descriptively reviewed. There was a consistent trend for an inverse association between healthy dietary and lifestyle patterns and all-cause mortality across the studies (HRs for high vs. low scores ranged from 0.58 to 0.80, 3 out of 5 CIs crossing the null), apart from one study that indicated a positive association (HR = 1.19, 95% CI = 0.59–2.43) but with wide CIs crossing the null⁹⁰ (Figure 3).

3.3.2 | Post-diagnosis dietary patterns

Two pooled analyses of two cohorts,^{80,81,83,87} four additional studies,^{82,84,85,88,91,92} and one additional publication from one of the pooled cohorts⁸⁹ (11 publications) investigated 15 food-based dietary patterns, broadly grouped into 11 healthy dietary patterns (dietary

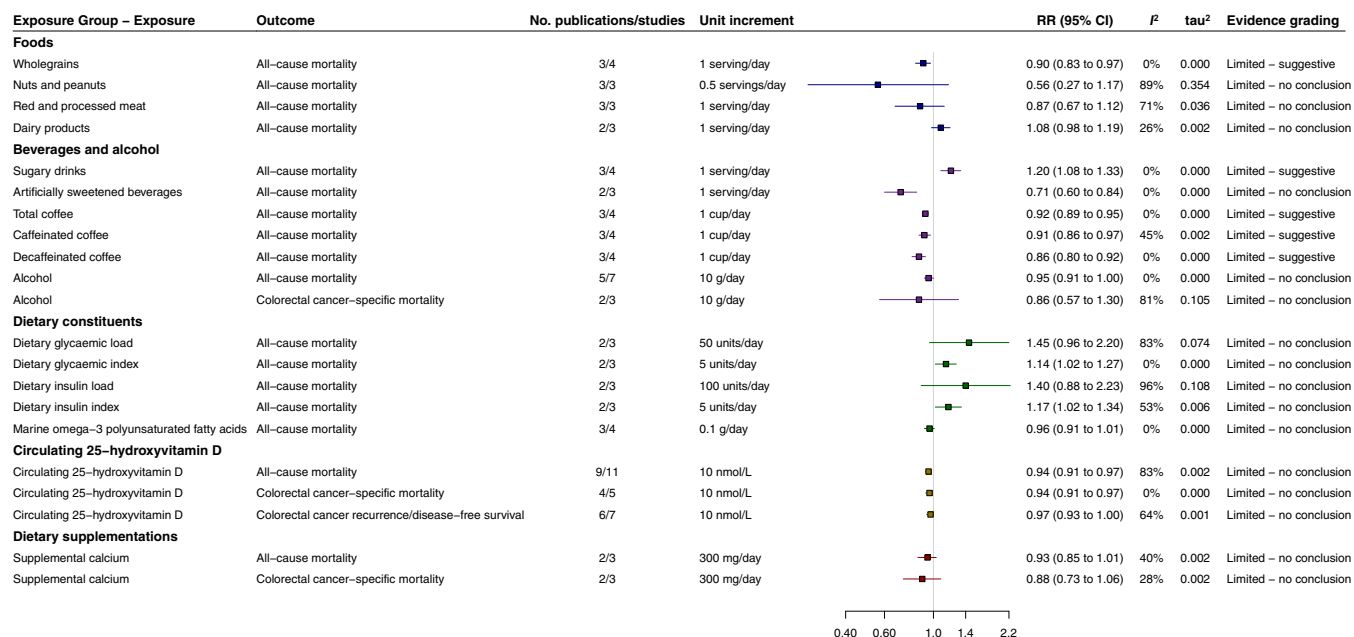


FIGURE 2 An overview of summary relative risks from linear dose-response meta-analyses of observational studies and the corresponding evidence grading.

guidelines or recommendations, namely [modified] Mediterranean diets, [healthy] plant-based foods/diet, prudent dietary patterns) and four unhealthy dietary patterns (i.e., unhealthy plant-based foods/diet, pro-hyperinsulinemia diet, western dietary patterns, pro-inflammatory diet) (Supplementary Tables S10–S21 and Figures S7–S10). Most studies investigated multiple patterns, defined a priori, a posteriori or using hybrid methods (Supplementary Table S6 and Figure S11). The evidence was descriptively reviewed. Six independent study groups reported 20 healthy dietary patterns and all-cause mortality point estimates (8 publications) (3722 deaths),^{80,81,84,85,88,89,91,92} which showed 18 inverse associations (HRs ranged from 0.46 to 0.98, 12 CIs crossing the null), and two positive associations (HRs 1.07 and 1.32, CIs crossing the null) (Figure 3). Five independent study groups reported eight unhealthy dietary patterns and all-cause mortality point estimates (8 publications) (4579 deaths),^{82–85,87,89,91,92} of which six showed positive associations (HRs ranged from 1.23 to 2.32, four CIs crossing the null), and two inverse associations (HRs 0.77 and 0.85, CIs crossing the null) (Figure 3).

3.3.3 | Post-diagnosis exposures with dose-response meta-analysis

Post-diagnosis whole grain intake

Four studies (3 publications)^{92–94} were identified (Supplementary Table S22 and Figures S12 and S13). Linear dose-response meta-analysis showed a lower risk of all-cause mortality with post-diagnosis whole grain intake (summary RR per 1 serving/day = 0.90, 95% CI = 0.83–0.97; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.83–0.94) (1288 deaths) (Figure 4).

Post-diagnosis nut and peanut intake

Three studies (3 publications)^{89,92,96} were identified (Supplementary Table S23, Figures S14 and S15). Linear dose-response meta-analysis showed little evidence of an association between post-diagnosis nut and peanut intake and all-cause mortality, with wide 95% CIs crossing null (summary RR per 0.5 serving of nuts and peanuts/day = 0.56, 95% CI = 0.27–1.17; $I^2 = 89\%$, $\tau^2 = 0.35$, RRs range = 0.36–0.99) (816 deaths) (Supplementary Figure S16).

Post-diagnosis red and processed meat, red meat, and processed meat intake

Three studies (4 publications) were identified investigating post-diagnosis intakes of red and processed meat,^{86,89,98} (unprocessed) red meat⁹⁷ and processed meat⁹⁷ (Supplementary Table S24). Linear dose-response meta-analysis was possible for studies that investigated post-diagnosis red and processed meat intake. No association was observed with all-cause mortality (summary RR per 1 serving/day = 0.87, 95% CI = 0.67–1.12; $I^2 = 71\%$, $\tau^2 = 0.04$, RRs range = 0.65–1.07) (3 studies/publications)^{86,89,98} (1700 deaths), and in the one study reporting results for colorectal cancer-specific mortality (RR per 1 serving/day = 1.22, 95% CI = 0.90–1.67) (162 deaths)⁸⁹ (Supplementary Figures S17–S19).

Post-diagnosis dairy product intake

Four studies (3 publications) were identified^{92,101,102} (Supplementary Table S25). One study lacked sufficient information for inclusion in the meta-analysis¹⁰² (Supplementary Figure S20). Linear dose-response meta-analysis showed little evidence for an association between post-diagnosis dairy intake and risk of all-cause mortality (summary RR per

TABLE 2 Evidence grades and main findings from the meta-analyses and descriptive synthesis of post-diagnosis dietary factors.

Diet and survival in men and women with colorectal cancer				
2023	Exposure	Outcome	Summary of findings RR (95% CI)	Conclusions ^a
Strong evidence	Convincing	-	-	-
Limited evidence	Probable	-	-	-
Decreases risk				
Limited evidence	Healthy dietary and lifestyle patterns combined	All-cause mortality	Descriptive review: 6 HRs from 4 study groups, no meta-analysis.	Evidence from 4 pooled analyses and individual cohort studies showing a trend of inverse associations, that suggested improved survival with adherence to healthful recommendations. The evidence was limited in methodological quality.
	(4 patterns: Recommendations for cancer prevention or a healthy diet and lifestyle [with physical activity and body composition as components])		5 HRs ranged from 0.58 to 0.80 for high versus low scores, 3 CIs crossing the null. 1 HR = 1.19 (0.59–2.43).	
	Healthy dietary patterns (11 patterns: Healthy dietary recommendations, Mediterranean, plant-based, or prudent diets, mostly of high intakes of fruits and vegetables, whole grains, nuts and legumes, and low intakes of red and processed meat)	All-cause mortality	Descriptive review: 20 HRs from 6 study groups, no meta-analysis.	Evidence from 6 pooled analyses and individual cohort studies showing a trend of inverse associations, that suggested better survival with a range of healthy dietary patterns. The evidence was limited in methodological quality.
	Whole grains	All-cause mortality	18 HRs ranged from 0.46 to 0.98 for high versus low scores, 12 CIs crossing the null. 2 HRs = 1.07 and 1.32, CIs crossing the null.	
Limited evidence	Total coffee	All-cause mortality	Summary RR per 1 serving/day = 0.90 (0.83–0.97), $I^2 = 0\%$, $\tau^2 = 0.00$, 4 studies (3 publications), 1288 deaths	Evidence from 4 cohort studies (3 publications) showed on average a lower risk of all-cause mortality with higher consumption. The evidence was limited in methodological quality.
	Caffeinated coffee	All-cause mortality	Summary RRs per 1 cup/day = 0.92 (0.89–0.95), $I^2 = 0\%$, $\tau^2 = 0.00$, 0.91 (0.86–0.97), $I^2 = 45\%$, $\tau^2 = 0.002$, 0.86 (0.80–0.92), $I^2 = 0\%$, $\tau^2 = 0.00$, 4 studies (3 publications), 2078 deaths	Evidence from 4 cohort studies (3 publications) showed on average a lower risk of all-cause mortality with higher consumption. The evidence was limited in methodological quality.
	Decaffeinated coffee	All-cause mortality		Coffee has shown anti-inflammatory and anti-tumourigenic effects, but more is needed to understand the mechanisms underpinning the associations with cancer survival.
	Increases risk			
Limited evidence	Unhealthy dietary patterns (4 patterns: Unhealthy plant-based, pro-hyperinsulinemic, pro-inflammatory, western diets)	All-cause mortality	Descriptive review: 8 HRs from 5 study groups, no meta-analysis.	Evidence from 5 pooled analyses and individual cohort studies generally showed a trend of positive associations, suggesting poorer survival with a range of unhealthy dietary patterns. The evidence was limited in methodological quality.
			6 HRs ranged from 1.23 to 2.32 for high versus low scores, 4 CIs crossing the null. 2 HRs = 0.77 and 0.85, CIs crossing the null.	

TABLE 2 (Continued)

Diet and survival in men and women with colorectal cancer			
2023	Exposure	Outcome	Summary of findings RR (95% CI)
	Sugary drinks	All-cause mortality	Summary RR per 1 serving/day = 1.20 (1.08–1.33), $I^2 = 0\%$, $\tau^2 = 0.00$, 4 studies (3 publications), 1290 deaths
			Conclusions ^a Evidence from 4 cohort studies (3 publications) showed on average a higher risk of all-cause mortality with higher consumption. The evidence was limited in methodological quality.
	Limited—no conclusion Nuts and peanuts	All-cause mortality	Summary RR per 0.5 serving = 0.56 (0.27–1.17), $I^2 = 89\%$, $\tau^2 = 0.35$, 3 studies, 816 deaths
			Evidence from 3 cohort studies showed on average no association. The evidence was limited in methodological quality.
	Red and processed meat	All-cause mortality	Summary RR per 1 serving/day = 0.87 (0.67–1.12), $I^2 = 71\%$, $\tau^2 = 0.04$, 3 studies, 162 deaths
			Evidence from 3 cohort studies showed on average no association. The evidence was limited in methodological quality.
	Dairy products	All-cause mortality	Summary RR per 1 serving/day = 1.08 (0.98–1.19), $I^2 = 26\%$, $\tau^2 = 0.002$, 3 studies (2 publications), 907 deaths
			Evidence from 3 cohort studies (2 publications) showed a summary estimate with 95% CI crossing the null value. The evidence was limited in quantity and methodological quality.
	Artificially sweetened beverages	All-cause mortality	Summary RR per 1 serving/day = 0.71 (0.60–0.84), $I^2 = 0\%$, $\tau^2 = 0.00$, 3 studies (2 publications), 1290 deaths
			Evidence from 3 cohort studies (2 publications) showed on average an inverse association. The evidence was limited in quantity and methodological quality, including potential confounding by other behaviour changes. Better understanding of underpinning biological mechanisms is needed.
	Alcohol	All-cause mortality	Summary RR per 10 g/day = 0.95 (0.91–1.00), $I^2 = 0\%$, $\tau^2 = 0.00$, 7 studies (5 publications), 2122 deaths
			Evidence from 7 cohort studies (5 publications) showed a summary estimate with 95% CI on the null value. The evidence was limited in methodological quality.
		Colorectal cancer-specific mortality	Summary RR per 10 g/day = 0.86 (0.57–1.30), $I^2 = 81\%$, $\tau^2 = 0.11$, 3 studies (2 publications), 479 deaths.
			Evidence from 3 cohort studies (2 publications) showed on average no association. The evidence was limited in quantity and methodological quality.
	Dietary glycaemic index	All-cause mortality	Summary RR per 5 units/day = 1.14 (1.02–1.27), $I^2 = 0\%$, $\tau^2 = 0.00$, 3 studies (2 publications), 1120 deaths.
			Evidence from 3 cohort studies (2 publications) showed on average a positive association. The evidence was limited in quantity and methodological quality, including potential reverse causation.
	Dietary glycaemic load	All-cause mortality	Summary RR per 50 units/day = 1.45 (0.96–2.20), $I^2 = 83\%$, $\tau^2 = 0.07$, 3 studies (2 publications), 1120 deaths.
			Evidence from 3 cohort studies (2 publications) showed a summary estimate with 95% CI crossing the null value. The evidence was limited in quantity and methodological quality, including potential reverse causation.

(Continues)

TABLE 2 (Continued)

Diet and survival in men and women with colorectal cancer				
2023	Exposure	Outcome	Summary of findings RR (95% CI)	Conclusions ^a
	Dietary insulin load	All-cause mortality	Summary RR per 100 units/day = 1.40 (0.88–2.23), $I^2 = 96\%$, $\tau^2 = 0.11$, 3 studies (2 publications), 1134 deaths.	Evidence from 3 cohort studies (2 publications) showed on average no association. The evidence was limited in quantity and methodological quality, including potential reverse causation.
	Dietary insulin index	All-cause mortality	Summary RR per 5 units/day = 1.17 (1.02–1.34), $I^2 = 53\%$, $\tau^2 = 0.01$, 3 studies (2 publications), 1134 deaths.	Evidence from 3 cohort studies (2 publications) showed on average a positive association. The evidence was limited in quantity and methodological quality, including potential reverse causation.
	Marine omega-3 polyunsaturated fatty acids	All-cause mortality	Summary RR per 0.1 g/day = 0.96 (0.91–1.01), $I^2 = 0\%$, $\tau^2 = 0.00$, 4 studies (3 publications), 1258 deaths.	Evidence from 4 cohort studies (3 publications) showed a summary estimate with 95% CI crossing the null value. The evidence was limited in methodological quality.
	Circulating 25-hydroxyvitamin D	All-cause mortality	Summary RR per 10 nmol/L = 0.94 (0.91–0.97), $I^2 = 83\%$, $\tau^2 = 0.002$, 11 studies (9 publications), 3710 deaths.	Evidence from 11 cohort studies (9 publications) and 5 cohort studies (4 publications) showed lower risk of all-cause and colorectal cancer-specific mortality with higher concentrations, respectively. The evidence was limited in methodological quality, including potential reverse causation. Evidence from 2 RCTs provided mixed results. The trials were of some concern to high risk of bias.
		Colorectal cancer-specific mortality	Summary RR per 10 nmol/L = 0.94 (0.91–0.97), $I^2 = 0\%$, $\tau^2 = 0.00$, 5 studies (4 publications), 1293 deaths.	Evidence from 7 cohort studies (6 publications) showed a summary estimate with 95% CI on the null value. The evidence was limited in methodological quality, including potential reverse causation.
		Recurrence/DFS	Summary RR per 10 nmol/L = 0.97 (0.93–1.00), $I^2 = 64\%$, $\tau^2 = 0.001$, 7 studies (6 publications), 2564 events.	Evidence from 3 cohort studies (2 publications) showed a summary estimate with 95% CI crossing the null value for both all-cause and colorectal cancer-specific mortality. The evidence was limited in quantity and methodological quality.
	Supplemental calcium	All-cause mortality	Summary RR per 300 mg/day = 0.93 (0.85–1.01), $I^2 = 40\%$, $\tau^2 = 0.002$, 3 studies (2 publications), 1075 deaths.	Evidence from 3 cohort studies (2 publications) showed a summary estimate with 95% CI crossing the null value for both all-cause and colorectal cancer-specific mortality. The evidence was limited in quantity and methodological quality.
		Colorectal cancer-specific mortality	Summary RR per 300 mg/day = 0.88 (0.73–1.06), $I^2 = 28\%$, $\tau^2 = 0.002$, 3 studies (2 publications), 353 deaths.	
	Circulating folate and folic acid concentrations (ACM, recurrence/DFS)			A pooled analysis of 6 cohort studies showed no associations between circulating folate and folic acid concentrations and risk of overall survival, recurrence, and disease-free survival. The results were limited in methodological quality, including potential reverse causation.

TABLE 2 (Continued)

2023	Diet and survival in men and women with colorectal cancer	Exposure	Outcome	Summary of findings RR (95% CI)	Conclusions ^a
		Dairy products (CCSM), marine omega-3 polyunsaturated fatty acids (recurrence/DFS), calcium supplements (ACM), dietary and supplemental calcium (ACM), dietary calcium (ACM, CCSM)			2-3 cohort studies were identified. Meta-analysis was not possible. The results were inconsistent and limited in methodological quality.
		Healthy dietary and lifestyle patterns combined (CCSM, recurrence/DFS), healthy dietary patterns (CCSM, recurrence/DFS), unhealthy dietary patterns (CCSM), vegetables (ACM), fruits (ACM), milk (CCSM), fruit juice (ACM), multivitamin supplements (ACM), vitamin D supplements (ACM), calcium supplements (CCSM), dietary and supplemental calcium (CCSM), folic acid supplements (ACM)			2-3 cohort studies were identified. Meta-analysis was not possible. There was little evidence of an association, and the results were limited in methodological quality.
		Healthy dietary and lifestyle patterns combined (CVDIM), healthy dietary patterns (CVDIM, other causes of deaths), unhealthy dietary patterns (recurrence/DFS, CVDIM, other causes of deaths), whole grains (CCSM, recurrence/DFS), fruit and vegetables (ACM), fruits (CCSM), vegetables (CCSM), green leafy vegetable (ACM), nuts and peanuts (CCSM, recurrence/DFS), red and processed meat (CCSM), (unprocessed) red meat (ACM, recurrence/DFS), processed meat (ACM, recurrence/DFS), fish, shellfish and other seafoods (ACM, recurrence/DFS), dairy products (recurrence/DFS), high fat dairy, low fat dairy, and other dairy products (ACM, CCSM), milk, high fat, and low fat milk (ACM), sugary drinks (CCSM, recurrence/DFS, non CCSM), artificially sweetened beverages (CCSM, recurrence/DFS, non CCSM), fruit juice (CCSM, non CCSM), coffee, caffeinated coffee, and decaffeinated coffee (CCSM, recurrence/DFS), alcohol (recurrence/DFS, CVDIM, other causes of death), beer, wine, liquor (ACM, CCSM, CVDIM, other causes of death), carbohydrate (ACM, CCSM, recurrence/DFS), simple carbohydrate and complex carbohydrate (ACM, CCSM), dietary fibre, cereal fibre, fruit fibre, and vegetable fibre (ACM, CCSM), dietary glycaemic load and glycaemic index (CCSM, recurrence/DFS), dietary insulin load and insulin index (CCSM, recurrence/DFS), total, mono-unsaturated, poly-unsaturated, saturated, trans fat and all-cause mortality (ACM, CCSM; recurrence/DFS), polyunsaturated to saturated fat ratio (ACM, CCSM), long-chain omega-3 polyunsaturated fatty acids (ACM, CCSM), omega-3 and omega-6 polyunsaturated fatty acids (recurrence/DFS), marine omega-3 polyunsaturated fatty acids (CCSM), animal and plant fat (ACM, CCSM, recurrence/DFS), total protein, animal protein, and plant protein (ACM, CCSM), any mineral or vitamin supplements (ACM), herb or vitamin supplements (ACM, CCSM), multivitamin supplements (CCSM), B-complex supplements (ACM), fish oil supplements (ACM, CCSM), vitamin A, B6, C, E, and beta-carotene supplements (ACM), iron, magnesium, selenium, and zinc supplements (ACM), vitamin D supplements (recurrence/DFS), dietary and supplemental vitamin D (ACM, recurrence/DFS), folic acid supplements (CCSM), dietary and supplemental folate (ACM, CCSM), dietary folate (ACM, CCSM)			Only 1 pooled analysis of 2 cohorts or 1 individual cohort study reported results.

Note: Potential sources of heterogeneity and publication bias were not assessed because of the low number of RCTs and observational studies, except for the analyses of circulating 25-hydroxyvitamin D and all-cause and colorectal cancer-specific mortality, where between-study heterogeneity was partly reduced when advanced/metastatic cancer survivors were excluded, when possible, and for the analysis of circulating 25-hydroxyvitamin D and all-cause mortality, where there was no indication of small-study effects such as publication bias. The observational studies were at risk of bias in various methodological quality, mainly related to residual confounding, differential selection of participants into the study, exposure misclassification, reverse causation, and potential departure from intended exposure. Main concerns that impact the evidence grading were highlighted in the table. Biological mechanisms were not the decisive factor contributing to the present evidence conclusions except otherwise shown.

Abbreviations: ACM, all-cause mortality; CCSM, colorectal cancer-specific mortality; CI, confidence interval; CVDIM, cardiovascular disease mortality; DFS, disease-free survival; HR, hazard ratio; RR, relative risk.

^aOne trial of each on nutritional behavioural intervention and high-protein supplementation, omega-3 fatty acid supplementation, and vitamin C supplementation were identified. The trials conducted planned or post-hoc analyses investigating colorectal outcomes as either primary or secondary outcomes. The trials were of some concerns to high risk of bias and were judged as limited-no conclusion evidence.

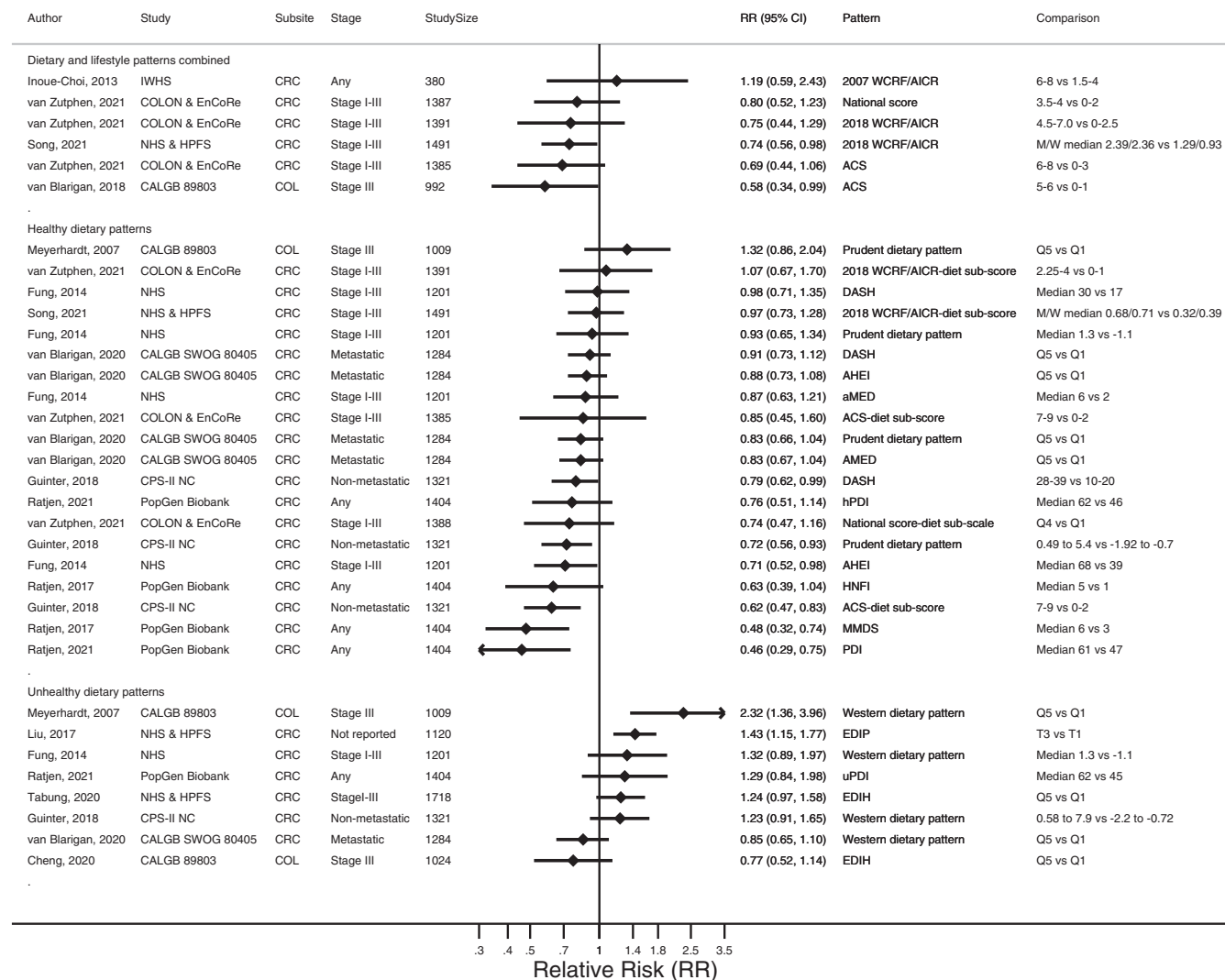


FIGURE 3 Forest plots showing the relative risks (RRs) with the 95% confidence intervals (95% CIs) for all-cause mortality associated with the highest versus lowest scores for dietary and lifestyle patterns combined, healthy dietary patterns, and unhealthy dietary patterns. Forest plot shows the results for the comparison of highest to lowest dietary and/or lifestyle adherence score. Each square and the horizontal line across the square represents the RR estimate of the individual study and its 95% CI. CI, confidence interval; COL, colon cancer; CRC, colorectal cancer; M, men; Q, quartile; RR, relative risk; T, tertile; W, women. Name of dietary and/or lifestyle patterns: 2007/2018 WCRF/AICR, 2007/2018 World Cancer Research Fund/American Institute for Cancer Research Cancer Prevention Recommendations; ACS, American Cancer Society Nutrition and Physical Activity Guideline for Cancer Survivors; AHEI, Alternative Healthy Eating Index; aMED, Alternative Mediterranean Diet; DASH, Dietary Approaches to Stop Hypertension; EDIH, Empirical Dietary Index for Hyperinsulinemia; EDIP, Empirical Dietary Inflammatory Pattern; HNFI, Healthy Nordic Food Index; National score (based on Dutch Healthy Diet index); MMDS, Modified Mediterranean Diet Score; h/uPDI, Healthy/Unhealthy Plant-based Diet Index; Prudent dietary pattern; Western dietary pattern. Study abbreviations: CALGB 89803, Cancer And Leukemia Group B 89803; CALGB SWOG 80405, Cancer And Leukemia Group B (Alliance) Southwest Oncology Group 80405; COLON, COlorectal cancer: Longitudinal, Observational study on Nutritional and lifestyle factors that may influence colorectal tumour recurrence, survival and quality of life; CPS-II NC, Cancer Prevention Study-II Nutrition Cohort; EnCoRe, Energy for life after COloRectal cancer; HPFS, Health Professionals Follow-Up Study; IWHS, Iowa Women's Health Study; NHS, Nurses' Health Study.

1 serving/day = 1.08, 95% CI = 0.98–1.19; $I^2 = 26\%$, $\tau^2 = 0.002$, RRs range = 0.98–1.15) (3 studies, 2 publications) (907 deaths)^{92,101} (Supplementary Figures S21–S23).

Post-diagnosis sugary drink, and artificially sweetened drink intake

The four identified studies on post-diagnosis sugary drinks (3 publications)^{92,103,104} and the three on post-diagnosis artificially sweetened drinks (2 publications)^{104,105} (Supplementary Table S26 and

Figures S24–S27) were included in the linear dose–response meta-analyses.

A higher risk of all-cause mortality (summary RR per 1 serving/day = 1.20, 95% CI = 1.08–1.33; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 1.06–1.32) (1290 deaths) was observed for sugary drinks. (Figure 4). A lower risk of all-cause mortality (summary RR per 1 serving/day = 0.71, 95% CI = 0.60–0.84; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs = 0.64 and 0.76) (1129 deaths) was observed for artificially sweetened drinks (Supplementary Figure S28).

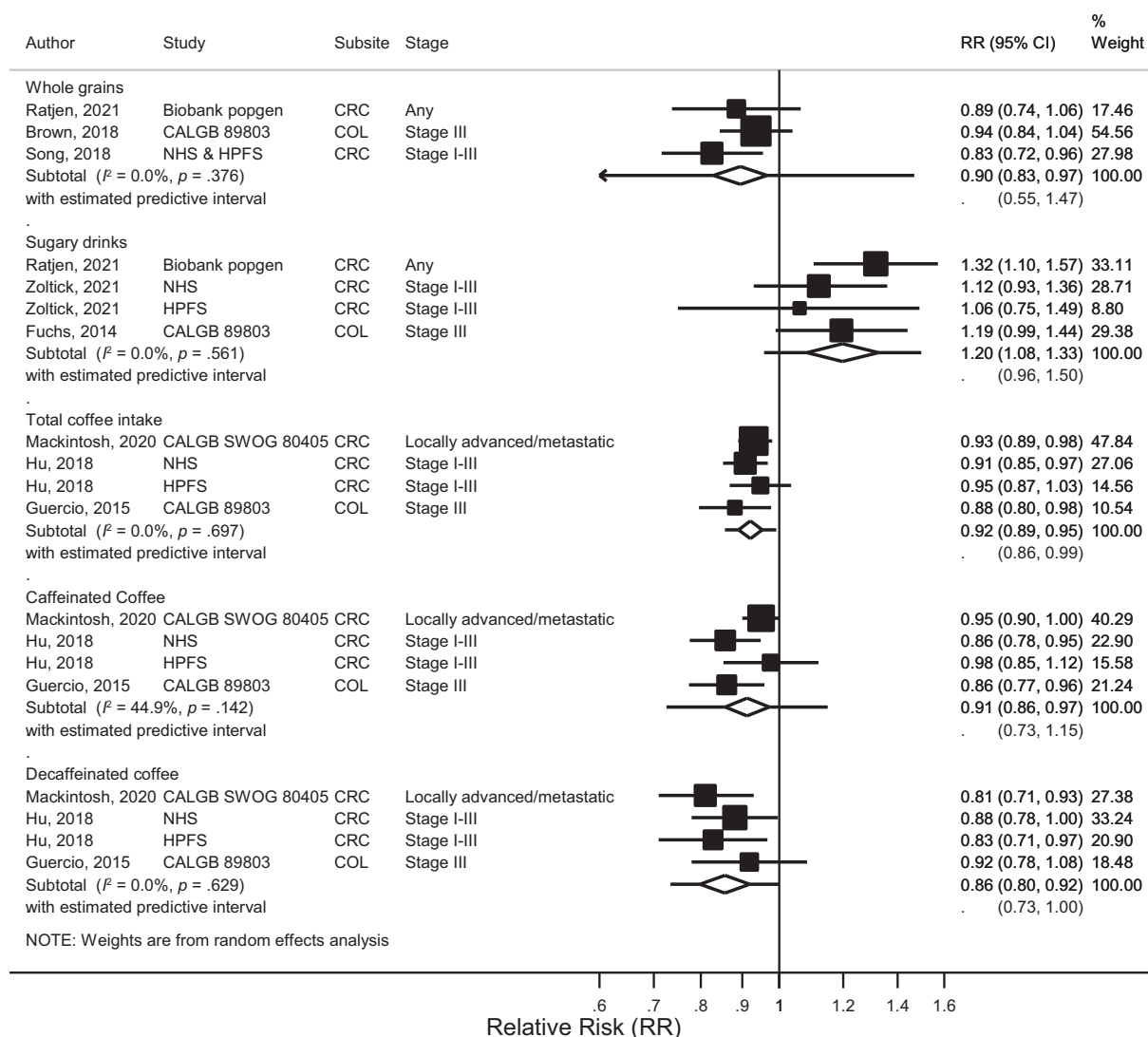


FIGURE 4 Linear dose-response meta-analyses of post-diagnosis intake of whole grains, sugary drinks, and total, caffeinated, and decaffeinated coffee in relation to all-cause mortality (associations presented are those graded as ‘limited–suggestive’). Forest plot shows the linear dose-response results (per 1 serving/day intake of whole grains and sugary drinks; and per 1 cup/day intake of coffee) from the inverse variance DerSimonian–Laird random-effects model. Each diamond represents the summary relative risk (RR) estimate, the diamond's width is the 95% confidence interval (CI), and the diamond's horizontal line is the 95% prediction interval (PI). Each square and the horizontal line across the square represents the RR estimate of the individual study and its 95% CI. CI, confidence interval; COL, colon cancer; CRC, colorectal cancer; RR, relative risk. Study abbreviations: CALGB 89803, Cancer And Leukemia Group B 89803; CALGB SWOG 80405, Cancer And Leukemia Group B (Alliance) Southwest Oncology Group 80405; HPFS, Health Professionals Follow-Up Study; NHS, Nurses' Health Study.

Post-diagnosis total coffee, caffeinated coffee, and decaffeinated coffee intake

Four studies (3 publications)^{107–109} (Supplementary Table S27, Figures S29 and S30) were included in linear dose-response meta-analysis, which showed lower risk of all-cause mortality with total, post-diagnosis caffeinated and decaffeinated coffee intakes (summary RR per 1 cup/day = 0.92, 95% CI = 0.89–0.95, $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.88–0.95; summary RR = 0.91, 95% CI = 0.86–0.97, $I^2 = 45\%$, $\tau^2 = 0.002$, RRs range = 0.86–0.98; and summary RR = 0.86, 95% CI = 0.80–0.92, $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.81–0.92, respectively) (2078 deaths) (Figure 4). The results remained materially unchanged when excluding the study of locally advanced or metastatic colorectal cancer patients,¹⁰⁷ and also the

study of stage III patients¹⁰⁹ in sensitivity analyses (Supplementary Figure S31). The pooled analysis of two cohorts¹⁰⁸ conducted subgroup analyses for total coffee intake and all-cause mortality, observing little evidence of an association among stage I and II (73%) cancer survivors (RRs per 1 cup/day = 0.97, 95% CI = 0.91–1.03), but an inverse association among stage III (27%) cancer survivors (RRs per 1 cup/day = 0.80, 95% CI = 0.69–0.91) ($p_{\text{interaction}} = .02$) (Supplementary Table S27).

Post-diagnosis alcohol intake

Ten studies (8 publications)^{81,86,95,110–114} were identified (Supplementary Table S28). Two studies lacked sufficient information for inclusion in the meta-analysis^{112,114} (Supplementary Figure S32). Seven studies

(5 publications)^{81,86,95,110,111} (Supplementary Figures S33 and S34) were included in the linear and non-linear dose-response meta-analyses of post-diagnosis alcohol intake and all-cause mortality and three (2 publications)^{110,111} in the linear dose-response meta-analysis of colorectal cancer-specific mortality. There was an indication of an inverse association between post-diagnosis alcohol and all-cause mortality (summary RR per 10 g/day = 0.95, 95% CI = 0.91–1.00; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.91–1.06), and some indication of a lower risk of all-cause mortality with alcohol consumption up to ~20 g/day ($P_{\text{non-linearity}} = 0.23$) (2122 deaths), but with the 95% CIs crossing the null value. There was no association between post-diagnosis alcohol and colorectal cancer-specific mortality (summary RR per 10 g/day = 0.86, 95% CI = 0.57–1.30; $I^2 = 81\%$, $\tau^2 = 0.11$, RRs range = 0.60–1.10) (479 deaths) (Supplementary Figures S35 and S36).

Post-diagnosis dietary glycaemic load, glycaemic index, insulin load, and insulin index

The pooled analysis of two cohorts¹¹⁷ and one additional study (2 publications)^{116,118} were included in the linear dose-response meta-analyses of dietary glycaemic load, glycaemic index, insulin load, and insulin index and all-cause mortality (Supplementary Table S29 and Figures S37–S40).

The meta-analyses indicated positive associations, but the 95% CI crossed the null value for glycaemic load and insulin load. The summary RR per 50 glycaemic load units/day was 1.45 (95% CI = 0.96–2.20, $I^2 = 83\%$, $\tau^2 = 0.07$, RRs range = 1.19–1.82) and per 5 glycaemic index units/day was 1.14 (95% CI = 1.02–1.27, $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 1.12–1.14) (1120 deaths).^{116,117} The summary RR per 100 insulin load units/day was 1.40 (95% CI = 0.88–2.23, $I^2 = 96\%$, $\tau^2 = 0.11$, RRs range = 1.11–1.79) and per 5 insulin index units/day was 1.17 (95% CI = 1.02–1.34, $I^2 = 53\%$, $\tau^2 = 0.01$, RRs range = 1.11–1.28) (1134 deaths)^{117,118} (Supplementary Figures S41 and S42).

Post-diagnosis dietary fat intake

Five studies (7 publications) were identified investigating post-diagnosis intakes of total dietary fat,^{115,119} specific dietary fatty acids,^{89,99,100,115,119,120} ratio of polyunsaturated-to-saturated fat,⁸⁹ and fat from animal or plant sources^{92,115,119} (Supplementary Table S30).

Linear dose-response meta-analysis was only possible for marine n-3 PUFA intake and all-cause mortality (4 studies, 3 publications),^{99,100,120} which showed a marginal inverse association (summary RR per 0.1 g/day = 0.96, 95% CI = 0.91–1.01; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.92–1.01) (1258 deaths) (Supplementary Figures S43–S45).

Post-diagnosis circulating 25-hydroxyvitamin D (25(OH)D)

Seventeen studies (17 publications) were identified (Supplementary Table S32). Four studies^{126,127,129,133} lacked sufficient information for inclusion in the meta-analyses (Supplementary Figure S46). One publication⁵⁵ was superseded by a more recent one.¹³⁴ Linear dose-response meta-analyses showed inverse associations with all-cause mortality

(summary RR per 10 nmol/L = 0.94, 95% CI = 0.91–0.97; $I^2 = 83\%$, $\tau^2 = 0.002$, RRs range = 0.69–1.02) (11 studies, 9 publications) (3710 deaths)^{35,36,125,130,131,134–137} and colorectal cancer-specific mortality (0.94, 95% CI = 0.91–0.97; $I^2 = 0\%$, $\tau^2 = 0.00$, RRs range = 0.92–1.00) (5 studies, 4 publications) (1293 deaths),^{125,134,135,137} and marginally improved recurrence/disease-free survival (0.97, 95% CI = 0.93–1.00; $I^2 = 64\%$, $\tau^2 = 0.001$, RRs range = 0.91–1.08) (7 studies, 6 publications) (2564 events)^{35,127,131,132,136,137} (Supplementary Figures S47–S51). Between-study heterogeneity was partly reduced when advanced/metastatic cancer survivors were excluded, when possible, from the analyses (Supplementary Figure S52). There was no indication of small-study effects such as publication bias in the analysis of all-cause mortality (Egger's test $p = .76$) (Supplementary Figure S53). Non-linear dose-response meta-analysis indicated a higher risk of all-cause mortality at lower levels of circulating 25(OH)D and the magnitude of risk gradually decreased with higher levels of 25(OH)D up to ~70 nmol/L and remained constant thereafter ($p_{\text{non-linearity}} = .001$) (9 studies, 7 publications) (3556 deaths)^{35,125,131,134–137}. For recurrence/disease-free survival, there was little evidence of non-linearity, with wide 95% CI and limited data ($p_{\text{non-linearity}} = .14$) (5 studies, 4 publications) (2442 events)^{35,131,132,136} (Supplementary Figures S49 and S51).

Post-diagnosis dietary and supplemental calcium

Four studies (3 publications) investigating post-diagnosis intakes of dietary and supplemental calcium combined,^{102,124} dietary calcium,^{102,124} dairy calcium,¹²⁴ and supplemental calcium^{102,121,124} were identified (Supplementary Table S33). Linear dose-response meta-analyses were possible for supplemental calcium intake and all-cause and colorectal cancer-specific mortality. One study lacked sufficient information for inclusion in the meta-analyses¹²¹ (Supplementary Figure S54). The meta-analyses showed a marginal inverse association between supplemental calcium and all-cause mortality (summary RR per 300 mg/day = 0.93, 95% CI = 0.85–1.01, $I^2 = 40\%$, $\tau^2 = 0.002$, RRs range = 0.87–0.99) (1075 deaths) and colorectal cancer-specific mortality (summary RR = 0.88, 95% CI = 0.73–1.06, $I^2 = 28\%$, $\tau^2 = 0.002$, RRs range = 0.72–1.09) (353 colorectal cancer deaths) (3 studies, 2 publications)^{102,124} (Supplementary Figures S55–S57).

3.3.4 | Other descriptive reviews

The associations for the above exposures in relation with other investigated colorectal cancer outcomes, and for post-diagnosis intakes of fruits and vegetables, (unprocessed) red meat, processed meat, fish and seafoods, specific dairy products and milk, fruit juices, beer, wine, liquor, carbohydrate, dietary fibre, total dietary fat and specific fat types, dietary protein, dietary supplements, dietary and/or supplemental folate, and circulating concentrations of folate or folic acid in relation to all colorectal cancer outcomes were mostly investigated by few studies. These results were descriptively reviewed and are presented in Supplementary Text S2–S24 and Figures S58–S78.

3.4 | Risk of bias assessment

Three^{76,78,79} of the five identified RCTs were overall at high risk of bias, due to unblinded investigator and outcome assessor,^{76,78} and the inability to fully blind for whole food regimens⁷⁹ (Supplementary Figures S1 and S2). Across the 46 observational study publications that investigated dietary and/or lifestyle patterns and other exposures included in the meta-analyses (Supplementary Figures S3 and S4), many had some degree of incomplete adjustment, mainly for critically important confounders such as stage and/or treatment (47% moderate, 9% serious, 45% critical risk of bias). Three publications (6% moderate risk of bias)^{94,108,125} used techniques that partially accounted for selection bias, the rest (94%) had a serious risk of selection bias. Most publications had moderate risk of exposure misclassification (13% low, 70% moderate, 15% serious, 2% critical risk of bias). Only seven publications (15% serious risk of bias)^{82,91,93,105,109,116,118} partially accounted for exposure changes across follow-up, the rest (85%) had critical risk of bias due to departures from intended exposures. About 38% of the publications had no/almost no missing data (low risk of bias); the rest had different degrees of data missingness that could be partially accounted for (19% moderate, 32% serious and 11% with no information on missing data). Most publications had low or moderate risk of bias in outcome measurement (55% low, 30% moderate, 15% no information). Most publications (85%) had moderate risk of selective reporting (85%); (11%) had serious and two (4%)^{84,96} had critical risk of bias due to selective reporting. The few publications at critical risk of bias in any domain (Supplementary Figures S5, S7, S9, S12, S14, S17, S21, S24, S26, S29, S33, S37, S39, S43, S47, and S55) generally showed similar RR estimates and overlapping 95% CIs compared to publications with less than critical risk of bias. For circulating 25(OH)D, it was possible to stratify the studies by moderate or critical risk of bias from confounding. The summary RRs were similar in direction and magnitude (Supplementary Figure S79).

3.5 | Overall evidence grading

Taking together the evidence from RCTs and observational studies, eight associations received 'limited-suggestive' evidence grading in relation to all-cause mortality, namely inverse associations for healthy dietary and lifestyle patterns combined, healthy dietary patterns, whole grains, total, caffeinated, or decaffeinated coffee, and the positive associations for unhealthy dietary patterns, and sugary drinks. All other associations received 'limited-no conclusion' evidence grading. More information on the associations and the reasoning behind the evidence grading is provided in Table 2.

4 | DISCUSSION

Evidence from RCTs and longitudinal observational studies examining the associations between post-diagnosis dietary factors, supplement use and colorectal cancer survival outcomes were systematically

reviewed. Twenty-one of the investigated associations had sufficient information for meta-analysis. Other associations, including those for dietary and/or lifestyle patterns, were descriptively reviewed. The quality of the evidence was then independently graded.

Several studies examined post-diagnosis dietary and/or lifestyle patterns that varied in definitions and derivation methods that is, a priori (hypothesis-driven methods) such as the Alternative Mediterranean Diet (aMed), Dietary Approaches to Stop Hypertension (DASH), or a posteriori (data-driven methods) such as prudent diet, western diet or with hybrid methods such as the Empirical Dietary Inflammatory Pattern (EDIP). We grouped the data broadly into 'healthy' and 'unhealthy' patterns and found generally consistent respective inverse and positive associations with all-cause mortality. 'Healthy' dietary patterns comprise mostly high intakes of fruits and vegetables, whole grains, nuts and legumes, and low intakes of red and processed meat. 'Unhealthy' dietary patterns comprise mostly high intakes of refined grains, red and processed meat, sugary drinks, and low coffee intakes. The beneficial or detrimental association with these respective patterns could be partly explained by the individual, cumulative or synergistic effect of these components, as for other chronic diseases.^{138,139} Studies that included 'healthy body weight' and high physical activity as additional components of healthy dietary and lifestyle patterns, reported inverse associations with all-cause mortality but the point estimates were similar to patterns based solely on dietary components. Studies have suggested that the association between healthy patterns and lower risk of all-cause mortality could be attributed to either the dietary and other lifestyle components^{81,86} or driven primarily by physical activity in the patterns.⁸⁰ The cut-point and the corresponding scoring for 'healthy body weight' may affect the risk estimation.^{80,86} In the present series of reviews, we found an inverted J-shaped relationship between body mass index and all-cause mortality. The lowest risk was observed at 28 kg/m²,²⁹ which is different to the conventional healthy body weight of 18.5–24.9 kg/m². Further studies are needed to differentiate the associations according to the pattern derivation and investigate whether the association could be attributed to individual dietary or lifestyle components within the pattern or potential interactions between dietary and other lifestyle components. Future research on dietary patterns should involve intervention studies that examine both survival outcomes and intermediate omics outcomes (e.g., gut microbiota) that could inform on potential mechanisms and potentially provide biomarkers of effect.

Biological mechanisms underlying the associations between dietary patterns and cancer development, or progression, are poorly understood. Adherence to healthy dietary patterns (e.g., Mediterranean diet or predominantly plant-based diets) has been associated with reduced circulating markers of inflammation, oxidative stress, insulin resistance.^{140–145} Individuals (healthy or with chronic diseases) consuming a 'Western' or unhealthy diet generally have higher levels of inflammation and hyperinsulinemia.^{140,143,146,147} Similarly, in colorectal cancer patients, a Westernised diet and high glycaemic load can lead to chronically elevated insulin levels that could facilitate tumour recurrence, micro-metastasis or development of co-morbidities (e.g., cardiovascular diseases) and higher risk of mortality.¹⁴⁸ Molecular epidemiology

studies are required to elucidate the relevant proposed mechanisms¹⁴⁸ and clarify the influence of what dietary factors or modifications on risk of cancer recurrence and survival.¹⁴⁹ Future studies should examine if associations of diet vary according to disease stage or treatment phase¹⁴⁹ to establish actionable lifestyle factors that can impact treatment response and/or survival.¹⁵⁰ The use of biomarkers and omics approaches could also enable more objective characterisation of dietary intake, considering the diversity of human tumours.^{150–152} Any relevant markers with prognostic value could then be incorporated in trials with surrogate disease end points.¹³⁸ Evidence from Mendelian randomisation (MR) studies could assist in prioritising certain nutritional interventions that are more likely to reduce cancer progression.¹⁵³

Higher intakes of individual food items, such as whole grains and coffee, were associated with a lower risk of all-cause mortality in this review. Whole grain intake was found to lower the risk of colorectal cancer incidence and all-cause mortality in general population studies.^{19,154} Whole grains are high in dietary fibre, which increases faecal bulk and decreases transit time, minimising exposure to intestinal carcinogens.¹⁵⁵ Whole grains may be fermented by gut microbes¹⁵⁶ into short-chain fatty acids that could facilitate normal colonocyte growth and induce tumour cell apoptosis.¹⁵⁷ Clinical intervention studies in healthy individuals or with various chronic diseases found that substituting refined grains with whole grains reduced inflammation¹⁵⁸ and insulin resistance,¹⁵⁹ possibly due to beneficial phytochemical constituents. Future studies should evaluate whole grains as a proportion of total grain consumption or a ratio to refined grain intake, in addition to overall intake. Coffee contains various bioactive phytochemicals that have antioxidant, anti-inflammatory, insulin-sensitising, and anti-tumour properties.^{160,161} The coffee polyphenol, caffeic acid, may inhibit colon cancer metastasis.^{162–164} The association with coffee was unlikely attributed to its caffeine content nor the avoidance of consuming caffeinated drinks after diagnosis and any potential influence from reverse causation, since the present meta-analyses and sensitivity analyses showed consistent inverse associations with all-cause mortality, overall and both for caffeinated and decaffeinated coffee. The pooled analysis of two cohorts observed an inverse association between coffee and all-cause mortality among stage III but not among stage I–II cancer survivors when the analysis was stratified by cancer stage.¹⁰⁸ The authors could not rule out chance findings due to multiple comparisons for the stratified analysis, nevertheless, the result was in line with our findings contributed primarily by studies of advanced/metastatic/stage III disease survivors.^{107,109} Liver metastases are common in colorectal cancer.¹⁶⁵ An inverse association between coffee consumption and liver cancer development has been observed.¹⁹ Coffee consumption could improve survival by reducing risk of liver metastases, but additional studies are required.

Higher sugary drink intake was associated with higher risk of all-cause mortality. The finding is supported by plausible influences of dietary sugar on energy metabolism, insulin resistance, lipid metabolism, inflammation, and immune function that could drive cancer progression.¹⁶⁶ The result was unlikely influenced by whether fruit juice was investigated as part of the sugary drinks^{103,104} or not,⁹² as the evidence of an association with fruit juice intake is limited.^{92,104}

Caution is needed when interpreting the inverse association observed for artificially sweetened beverages and all-cause mortality, as the result was based on few studies. It is possible that the inverse association was partly a result of higher uptake of drinks with sugar alternatives/substitutes in individuals with highest risk for weight gain, a possible indication of general health.¹⁰⁵ Better quantification and characterisation of the drinks, including types of artificial sweeteners which may have different chemical or biological properties and different associations with cancer development¹⁶⁷ are needed in future cancer survival studies.

The marginal inverse associations observed for higher post-diagnosis alcohol consumption and higher at/post-diagnosis circulating 25(OH)D with colorectal cancer outcomes may be partly explained by reverse causation. Individuals who reported no alcohol consumption showed a somewhat higher rate of all-cause mortality compared with alcohol consumers. In the present non-linear analyses, individuals with deficient circulating 25(OH)D levels showed a higher rate of all-cause mortality in comparison to those with normal circulating 25(OH)D levels. Cancer survivors with advanced disease may abstain from drinking alcohol and may be more prone to 25(OH)D deficiency.¹³¹ The included studies were rated as having moderate or serious risk of bias from confounding, since they did not account for cancer treatment and other important variables (such as smoking, adiposity, physical activity). This led to little confidence in reaching a stronger conclusion for the observed associations, despite vitamin D has anticancer and antiproliferative effects.^{168,169}

The present systematic review on post-diagnosis intakes of nuts and peanuts, red and processed meat, dairy products, marine n-3 PUFAs, dietary glycaemic load, glycaemic index, insulin load, and insulin index included only a small number of studies and for most showed null results. Findings on post-diagnosis dietary supplement use, included supplemental calcium use that could lower the risk of colorectal cancer development,^{19,20} were null or inconsistent in the limited studies identified. Future investigations into the types, dosages, duration of use, and potential interactions with cancer treatment¹⁷⁰ are needed to provide more definitive conclusions for cancer survivors who often use dietary supplements.¹⁷¹ Few RCTs, primarily on dietary supplementation, were identified that did not provide substantial supporting data.

Cancer survival studies have inherent methodological limitations,^{50,172} and there are always challenges in their planning and execution. Selection bias is highly likely since participation in studies that investigate outcomes after cancer diagnosis depends on survival time post-diagnosis. Individuals who participate in a survival study could have a different risk profile for the outcome, as compared to those who do not participate.⁵⁰ Reverse causation is also likely in cancer survival studies. Cancer recurrence or undetected cancer progression could be a confounder in studies of mortality outcomes. Associations could be biased if undetected disease progression leads to diet alterations (e.g., malnutrition due to altered nutrient absorption), weight loss¹⁷³ and hence worse outcomes after diagnosis. Potential approaches to reduce such bias in future observational studies include performing lagged analyses or restricting

study participation to recurrence-free individuals, but this information is rarely available.⁵⁰ Exposure measurement error and misclassification is possible, since dietary information is largely self-reported, once at-diagnosis or at a non-clearly specified time after diagnosis, during which diet might have been affected by disease progression and/or cancer treatment. Future studies should report results in a more standardised manner, including making clear statements of when the exposure was assessed with respect to the diagnosis and/or treatment and if possible, conduct subgroup analyses by the timing of exposure assessment. A limited number of studies investigated changes in dietary habits, over time, but these studies reported results that generally agreed with results of studies that performed single time-point assessments.^{81,85,98,120,128} Some studies reported that a substantial number of colorectal cancer survivors (53%–85%) reduced post-diagnosis red meat, hamburger, and other fast-food consumption.^{174,175} It is possible that red and processed meat consumption over time was misclassified, potentially resulting in the null associations with colorectal cancer outcomes observed in this review. A study on nut consumption reported somewhat stronger associations between cumulative nut intake (weighted nut exposure average between two assessments) and improved disease-free and recurrence-free survival, versus a single baseline assessment.⁹⁶ When possible, investigators should capture repeated dietary assessments across the cancer survivorship trajectory and perform time-varying analyses. We were not able to conduct stratified analysis because we lacked information for cancer characteristics, precise exposure timeframe, geographic location,¹⁷⁶ race/ethnicity, socio-demographics.¹⁷⁷

Most meta-analyses performed in this review included studies that looked at all-cause or cancer-specific mortality as the main outcome. In general, such studies are simpler to conduct because information on mortality can be easily captured through death certificates and/or registries of vital status. Information on recurrence is usually only captured in clinical trials or via time-consuming clinical record review. A further limitation is that studies with ‘recurrence’ as outcome have used heterogeneous definitions (such as ‘disease-free survival’, ‘event-free survival’, etc.) making comparisons more difficult and potential errors in outcome assessment more likely.¹⁷⁸ Future studies should use standardised, cancer-specific recurrence definitions to allow more consistent evaluation of this body of evidence and subgroup analyses. Health related quality of life outcomes should also receive more attention in future studies to support the design of suitable survivorship care/plans. An overview of limitations of cancer survival studies and future research recommendations is presented in the summary manuscript, Box 1³¹ of the current manuscript series on colorectal cancer survivors.

This systematic review has enhanced the evidence on post-diagnosis dietary factors and colorectal cancer outcomes through comprehensive collection, synthesis, and evaluation of findings, based to a large extent on observational studies. Such findings will inform the design and execution of carefully designed RCTs that are currently limited but also more challenging to perform particularly when investigating ‘hard’ endpoints including mortality.^{138,179} Certain limitations of

such RCTs in nutritional epidemiology include the challenges of adhering to particular dietary interventions (inclusive of economic burden), and difficulties in recruiting participants for long-term follow-up and the potential recruitment bias and threat to generalise that results. Moreover, identification of appropriate control diets and blinding of dietary interventions is often challenging.^{96,138,179} More personalised, multi-component interventions would be also necessary.^{180–182}

5 | CONCLUSIONS

There was ‘limited-suggestive’ evidence for the associations between post-diagnosis healthy dietary and/or lifestyle patterns, intake of whole grain, or coffee (total, caffeinated, decaffeinated) with lower risk of all-cause mortality, and for the associations between post-diagnosis unhealthy dietary patterns or intake of sugary drinks with higher risk of all-cause mortality. The evidence for other exposure-outcome associations received a ‘limited-no conclusion’ grading.

Conclusions made by the CUP Global independent Expert Committee on Cancer Survivorship and the Expert Panel may contribute towards future formulation of lifestyle guidance/recommendations specific for colorectal cancer survivors. The current evidence is not strong enough for the development of recommendations for cancer survivors following the well-established CUP Global process but a new complementary process, considering evidence which may be more ‘limited’ alongside expert opinion would allow the development of guidance, to provide cancer survivors with sound information based on the best available evidence. To provide conclusions with a higher level of certainty and develop specific lifestyle recommendations, additional evidence is needed from larger, well-designed observational studies in well-characterised populations, with repeated exposure and confounder assessments. Mechanistic studies exploring the biological pathways that underpin potential associations between dietary exposures and colorectal cancer outcomes are crucial to inform recommendations. RCTs,¹⁸⁰ that could possibly, evaluate the effects of specific dietary patterns, or coffee¹⁸³ that have shown survival benefits in this SLR would be informative. MR studies using instrumental exposures to account for confounding and reverse causation¹⁵³ could be used to clarify the results for circulating 25(OH)D or other biomarkers, and examine the role of diet and gut microbiome on colorectal cancer prognosis.¹⁸⁴ Additional studies are also needed in socio-demographically and ethnically diverse survivors, of different cancer stages, and at different phases of the cancer continuum.

AUTHOR CONTRIBUTIONS

Konstantinos K. Tsilidis and Doris S. M. Chan are co-principal investigators of CUP Global at Imperial College London (ICL). Doris S. M. Chan and Konstantinos K. Tsilidis implemented the study according to the protocol reviewed by the CUP Global Protocol Expert Group (PEG). Katia Balducci and Sonia Kiss did the literature search. Katia Balducci, Sonia Kiss, Margarita Cariolou and Rita Vieira did the study selection. Katia Balducci, Sonia Kiss, Margarita Cariolou, Rita Vieira,

Georgios Markozannes and Nerea Becerra-Tomás did the data extraction and checking. Georgios Markozannes, Margarita Cariolou and Nerea Becerra-Tomás did the risk of bias assessment. Doris S. M. Chan, Georgios Markozannes, Margarita Cariolou, Katia Balducci, Sonia Kiss and Rita Vieira analysed and interpreted the data. Konstantinos K. Tsilidis interpreted the data. Dagfinn Aune was a WCRF International CUP Global ICL team member who revised the manuscript. Darren C. Greenwood was a statistical adviser. Amanda J Cross was a CUP Global advisor at Imperial College London. Esther M. González-Gil was a CUP Global collaborator on biological processes and provided input into the biological mechanism citations in the manuscript. Ellen Copson was a PEG member, Chair of CUP Global Expert Committee on Cancer Survivorship, and Expert Panel member. Wendy Demark-Wahnefried and Galina Velikova were PEG, OACD, and CUP Global Expert Committee members. Andrew G. Renehan was a PEG member and Deputy Chair of CUP Global Expert Committee on Cancer Survivorship. John Krebs, Matty P, Weijenberg, Monica L. Baskin, Sarah J. Lewis, Jaap Seidell, Rajiv Chowdhury, and Lynette Hill were CUP Global Expert Panel members. Anne M. May, Anne Tjonneland, Karen Steindorf, Martijn Bours, Melissa M. Hudson, Roderick Skinner, and Folakemi T. Odedina were CUP Global Expert Committee members. All members of the CUP Global Expert Committee and Expert Panel provided input into the judgements on the evidence and advised on the interpretation of the review, the public representative (Lynette Hill) did not contribute to the final decisions made by the Panel. Doris S. M. Chan and Margarita Cariolou drafted the original manuscript. All authors reviewed and provided comments on the manuscript. Doris S. M. Chan is the guarantor and has full access to all the data and takes responsibility for the integrity of the data and the accuracy of the data analysis. The work reported in the paper has been performed by the authors, unless clearly specified in the text.

AFFILIATIONS

- ¹Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, UK
- ²Department of Hygiene and Epidemiology, University of Ioannina Medical School, Ioannina, Greece
- ³Department of Nutrition, Oslo New University College, Oslo, Norway
- ⁴Department of Research, The Cancer Registry of Norway, Oslo, Norway
- ⁵Leeds Institute for Data Analytics, Faculty of Medicine and Health, University of Leeds, Leeds, UK
- ⁶Nutrition and Metabolism Branch, International Agency for Research on Cancer, World Health Organization, Lyon, France
- ⁷Cancer Sciences Academic Unit, Faculty of Medicine, University of Southampton, Southampton, UK
- ⁸The Christie NHS Foundation Trust, Manchester Cancer Research Centre, NIHR Manchester Biomedical Research Centre, Division of Cancer Sciences, School of Medical Sciences, Faculty of Biology, Medicine and Health, University of Manchester, Manchester, UK

- ⁹Department of Epidemiology, GROW School for Oncology and Reproduction, Maastricht University, Maastricht, The Netherlands
- ¹⁰O'Neal Comprehensive Cancer Center, University of Alabama at Birmingham, Birmingham, Alabama, USA
- ¹¹Department of Oncology, St Jude Children's Research Hospital, Memphis, Tennessee, USA
- ¹²Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht University, Utrecht, The Netherlands
- ¹³Mayo Clinic Comprehensive Cancer Center, Jacksonville, Florida, USA
- ¹⁴Department of Paediatric and Adolescent Haematology/Oncology, Great North Children's Hospital and Translational and Clinical Research Institute, and Centre for Cancer, Newcastle University, Newcastle upon Tyne, UK
- ¹⁵Division of Physical Activity, Prevention and Cancer, German Cancer Research Center (DKFZ) and National Center for Tumor Diseases (NCT), Heidelberg, Germany
- ¹⁶Danish Cancer Society Research Center, Diet, Cancer and Health, Copenhagen, Denmark
- ¹⁷Department of Public Health, University of Copenhagen, Copenhagen, Denmark
- ¹⁸School of Medicine, Faculty of Medicine and Health, University of Leeds, Leeds, UK
- ¹⁹UPMC Hillman Cancer Center, Pittsburgh, Pennsylvania, USA
- ²⁰Department of Global Health, Robert Stempel College of Public Health and Social Work, Florida International University, Miami, Florida, USA
- ²¹World Cancer Research Fund International, London, UK
- ²²Department of Population Health Sciences, Bristol Medical School, University of Bristol, Bristol, UK
- ²³Department of Health Sciences, Faculty of Science, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands
- ²⁴Department of Biology, University of Oxford, Oxford, UK

ACKNOWLEDGEMENTS

We thank Teresa Norat for leading the WCRF/AICR Continuous Update Project (CUP) as principal investigator from 2007 to 2020 and for developing the original review protocol. We thank the WCRF International CUP Global ICL team members: Leila Abar, Neesha Nanu, Jakub Sobiecki, Britta Talumaa, and Victoria White, for their contribution to the literature search, study selection, and/or data extraction; and database managers: Rui Vieira, Christophe Stevens, Yusuf O. Anifowoshe, and Lam Teng for implementing and updating the CUP Global database. We also acknowledge the input from the CUP Global Secretariat members: Helen Croker, Panagiota Mitrou, Martin Wiseman, Kate Allen, Nigel T Brockton, Vanessa Gordon-Dseagu (and previous CUP Global Secretariat member Nicole Musuwo), for providing overall coordination for the work and for convening and facilitating discussions with the CUP Global Expert Committee and Expert Panel. We acknowledge the contribution of the CUP Global Protocol Expert Group (PEG) (Annie Anderson, Steven Clinton, John Mathers, Anne McTiernan, Lesley Turner, and Fränzel van Duijnhoven) and the Outcomes After a Cancer Diagnosis (OACD)

CUP Transition workstream (Anne McTiernan, Steven Clinton, Viv Lund) into the development of the original protocol for this work.

FUNDING INFORMATION

This work was funded by the World Cancer Research Fund network of charities (American Institute for Cancer Research [AICR]; World Cancer Research Fund [WCRF]; Wereld Kanker Onderzoek Fonds [WKOF]) (CUP Global Special Grant 2018). The funders of this study had no role in the decisions about the design and conduct of the study; collection, management, analysis, or interpretation of the data; or the preparation, review, or approval of the manuscript. The process used was based on the method developed by WCRF International's Methodology Task Force for the WCRF/AICR Second Expert Report. The views expressed in this review are the opinions of the authors. They may differ from those in future updates of the evidence related to food, nutrition, physical activity, and cancer incidence and survival.

CONFLICT OF INTEREST STATEMENT

Ellen Copson declared research support from Seca. Galina Velikova declared honoraria from Pfizer, Novartis, and Eisai, an institutional grant from Pfizer, and advisory board and consultancy fees from AstraZeneca, Roche, Novartis, Pfizer, Seagene, Eisai, and Sanofi. All other authors have no conflict of interest related to this work.

DATA AVAILABILITY STATEMENT

Only publicly available data were used in our study. Data sources and handling of these data are described in the materials and methods section. Further details are available from the corresponding author upon request.

ORCID

Doris S. M. Chan  <https://orcid.org/0000-0002-0198-1897>

Georgios Markozannes  <https://orcid.org/0000-0001-8481-579X>

Dagfinn Aune  <https://orcid.org/0000-0002-4533-1722>

Wendy Demark-Wahnefried  <https://orcid.org/0000-0001-5241-932X>

Konstantinos K. Tsilidis  <https://orcid.org/0000-0002-8452-8472>

REFERENCES

- Morgan E, Arnold M, Gini A, et al. Global burden of colorectal cancer in 2020 and 2040: incidence and mortality estimates from GLOBOCAN. *Gut*. 2023;72:338-344.
- Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin*. 2021;71:209-249.
- Ferlay JEM, Lam F, Colombet M, et al. *Global Cancer Observatory: Cancer Today*. International Agency for Research on Cancer; 2020.
- Favoriti P, Carbone G, Greco M, Pirozzi F, Pirozzi RE, Corcione F. Worldwide burden of colorectal cancer: a review. *Updates Surg*. 2016;68:7-11.
- Xi Y, Xu P. Global colorectal cancer burden in 2020 and projections to 2040. *Transl Oncol*. 2021;14:101174.
- Allemani C, Matsuda T, Di Carlo V, et al. Global surveillance of trends in cancer survival 2000-14 (CONCORD-3): analysis of individual records for 37 513 025 patients diagnosed with one of 18 cancers from 322 population-based registries in 71 countries. *Lancet*. 2018;391:1023-1075.
- Gullickson C, Goodman M, Joko-Fru YW, et al. Colorectal cancer survival in sub-Saharan Africa by age, stage at diagnosis and human development index: a population-based registry study. *Int J Cancer*. 2021;149:1553-1563.
- Soerjomataram I, Cabasag C, Bardot A, et al. Cancer survival in Africa, central and south America, and Asia (SURVCAN-3): a population-based benchmarking study in 32 countries. *Lancet Oncol*. 2023;24:22-32.
- Lin JS, Perdue LA, Henrikson NB, Bean SI, Blasi PR. Screening for colorectal cancer: updated evidence report and systematic review for the US preventive services task force. *Jama*. 2021;325:1978-1998.
- Lin JS, Piper MA, Perdue LA, et al. Screening for colorectal cancer: updated evidence report and systematic review for the US preventive services task force. *Jama*. 2016;315:2576-2594.
- Dekker E, Tanis PJ, Vleugels JLA, Kasi PM, Wallace MB. Colorectal cancer. *Lancet*. 2019;394:1467-1480.
- Jiang Y, Yuan H, Li Z, et al. Global pattern and trends of colorectal cancer survival: a systematic review of population-based registration data. *Cancer Biol Med*. 2021;19:175-186.
- Anderson AS, Martin RM, Renehan AG, et al. Cancer survivorship, excess body fatness and weight-loss intervention-where are we in 2020? *Br J Cancer*. 2021;124:1057-1065.
- Feng Y, Jin H, Guo K, Wasan HS, Ruan S, Chen C. Causes of death after colorectal cancer diagnosis: a population-based study. *Front Oncol*. 2021;11:647179.
- Fuccio L, Rex D, Ponchon T, et al. New and recurrent colorectal cancers after resection: a systematic review and meta-analysis of endoscopic surveillance studies. *Gastroenterology*. 2019;156:1309-1323.e3.
- Guraya SY. Pattern, stage, and time of recurrent colorectal cancer after curative surgery. *Clin Colorectal Cancer*. 2019;18:e223-e228.
- Du S, Li Y, Sun H, et al. The risk of developing second primary malignancies among colorectal cancer patients. *Aging (Albany NY)*. 2022;14:6756-6779.
- Jia H, Li Q, Yuan J, Sun X, Wu Z. Second primary malignancies in patients with colorectal cancer: a population-based analysis. *Oncologist*. 2020;25:e644-e650.
- World Cancer Research Fund International/American Institute for Cancer Research. Diet, Nutrition, Physical Activity and Cancer: A Global Perspective. Continuous Update Project Expert Report. 2018. Accessed March 2023. <http://dietandcancerreport.org>
- Papadimitriou N, Markozannes G, Kanellopoulou A, et al. An umbrella review of the evidence associating diet and cancer risk at 11 anatomical sites. *Nat Commun*. 2021;12:4579.
- Rock CL, Thomson CA, Sullivan KR, et al. American Cancer Society nutrition and physical activity guideline for cancer survivors. *CA Cancer J Clin*. 2022;72:230-262.
- Zhong Y, Zhu Y, Li Q, et al. Association between Mediterranean diet adherence and colorectal cancer: a dose-response meta-analysis. *Am J Clin Nutr*. 2020;111:1214-1225.
- Hoang T, Kim H, Kim J. Dietary intake in association with all-cause mortality and colorectal cancer mortality among colorectal cancer survivors: a systematic review and meta-analysis of prospective studies. *Cancers (Basel)*. 2020;12:3391.
- Kim Y, Je Y, Giovannucci EL. Association between alcohol consumption and survival in colorectal cancer: a meta-analysis. *Cancer Epidemiol Biomarkers Prev*. 2019;28:1891-1901.
- Kanellopoulou A, Riza E, Samoli E, Benetou V. Dietary supplement use after cancer diagnosis in relation to total mortality, cancer mortality and recurrence: a systematic review and meta-analysis. *Nutr Cancer*. 2021;73:16-30.
- Wu G, Xue M, Zhao Y, et al. Low circulating 25-hydroxyvitamin D level is associated with increased colorectal cancer mortality: a

- systematic review and dose-response meta-analysis. *Biosci Rep*. 2020;40:BSR20201008.
27. Vaughan-Shaw PG, Buijs LF, Blackmur JP, et al. The effect of vitamin D supplementation on survival in patients with colorectal cancer: systematic review and meta-analysis of randomised controlled trials. *Br J Cancer*. 2020;123:1705-1712.
 28. Global Cancer Update Programme (CUP Global). Accessed March 2023. <https://www.wcrf.org/diet-activity-and-cancer/global-cancer-update-programme/about-the-global-cancer-update-programme>
 29. Becerra-Tomás N, Markozannes G, Cariolou M, et al. Post-diagnosis adiposity and colorectal cancer prognosis: a Global Cancer Update Programme (CUP Global) systematic literature review and meta-analysis. *Int J Cancer*. 2024;155(3):400-425.
 30. Markozannes G, Becerra-Tomás N, Cariolou M, et al. Post-diagnosis physical activity and sedentary behaviour and colorectal cancer prognosis: a Global Cancer Update Programme (CUP Global) systematic literature review and meta-analysis. *Int J Cancer*. 2024;155(3):426-444.
 31. Tsilidis KK, Markozannes G, Becerra-Tomás N, et al. Post-diagnosis adiposity, physical activity, sedentary behaviour, dietary factors, supplement use and colorectal cancer prognosis: Global Cancer Update Programme (CUP Global) summary of evidence grading. *Int J Cancer*. 2024;155(3):471-485.
 32. Chan DSM, Tsilidis K, Becerra-Tomas N, et al. Global Cancer Update Programme Protocol for the data collection and systematic literature review on the role of diet, body fatness and physical activity on survival after colorectal cancer. 2022. Accessed March 2023. <https://osf.io/r5ud2/registrations>
 33. Lewis C, Xun P, He K. Vitamin D supplementation and quality of life following diagnosis in stage II colorectal cancer patients: a 24-month prospective study. *Support Care Cancer*. 2016;24:1655-1661.
 34. Geijssen A, Ulvik A, Gigic B, et al. Circulating folate and folic acid concentrations: associations with colorectal cancer recurrence and survival. *JNCI Cancer Spectr*. 2020;4:pkaa051.
 35. Wesselink E, Kok DE, Bours MJL, et al. Vitamin D, magnesium, calcium, and their interaction in relation to colorectal cancer recurrence and all-cause mortality. *Am J Clin Nutr*. 2020;111:1007-1017.
 36. Wesa KM, Segal NH, Cronin AM, et al. Serum 25-hydroxy vitamin D and survival in advanced colorectal cancer: a retrospective analysis. *Nutr Cancer*. 2015;67:424-430.
 37. Abrahamsson H, Meltzer S, Hagen VN, et al. Sex disparities in vitamin D status and the impact on systemic inflammation and survival in rectal cancer. *BMC Cancer*. 2021;21:535.
 38. FDA. *Dietary Supplements*. 2023. Accessed July 2023. <https://www.fda.gov/food/dietary-supplements/dietary-supplement-ingredient-directory>
 39. DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials*. 1986;7:177-188.
 40. Rucker G, Schwarzer G, Carpenter JR, Schumacher M. Undue reliance on I(2) in assessing heterogeneity may mislead. *BMC Med Res Methodol*. 2008;8:79.
 41. Higgins JP, Thompson SG. Quantifying heterogeneity in a meta-analysis. *Stat Med*. 2002;21:1539-1558.
 42. Borenstein M, Higgins JP, Hedges LV, Rothstein HR. Basics of meta-analysis: I(2) is not an absolute measure of heterogeneity. *Res Synth Methods*. 2017;8:5-18.
 43. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ*. 1997;315:629-634.
 44. Orsini N. Weighted mixed-effects dose-response models for tables of correlated contrasts. *Stata J*. 2021;21:320-347.
 45. Royston P. A strategy for modelling the effect of a continuous covariate in medicine and epidemiology. *Stat Med*. 2000;19:1831-1847.
 46. Viechtbauer W, Cheung MW. Outlier and influence diagnostics for meta-analysis. *Res Synth Methods*. 2010;1:112-125.
 47. Sterne JAC, Savovic J, Page MJ, et al. RoB 2: a revised tool for assessing risk of bias in randomised trials. *BMJ*. 2019;366:l4898.
 48. USDA. *Nutrition Evidence Systematic Review*. Risk of Bias for Nutrition Observational Studies (RoB-NObs) Tool. Accessed November 2022. <https://nesr.usda.gov/sites/default/files/2019-07/RiskOfBiasForNutritionObservationalStudies-RoB-NObs.pdf>
 49. Sterne JA, Hernan MA, Reeves BC, et al. ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. *BMJ*. 2016;355:i4919.
 50. Chubak J, Boudreau DM, Wirtz HS, McKnight B, Weiss NS. Threats to validity of nonrandomized studies of postdiagnosis exposures on cancer recurrence and survival. *J Natl Cancer Inst*. 2013;105:1456-1462.
 51. van Zutphen M, van Duijnhoven FJB, Wesselink E, et al. Identification of lifestyle behaviors associated with recurrence and survival in colorectal cancer patients using random survival forests. *Cancers (Basel)*. 2021;13:2442.
 52. Cheng E, Ou FS, Ma C, et al. Diet- and lifestyle-based prediction models to estimate cancer recurrence and death in patients with stage III colon cancer (CALGB 89803/Alliance). *J Clin Oncol*. 2022;40:740-751.
 53. Tretli S, Schwartz GG, Torjesen PA, Røsbahm TE. Serum levels of 25-hydroxyvitamin D and survival in Norwegian patients with cancer of breast, colon, lung, and lymphoma: a population-based study. *Cancer Causes Control*. 2012;23:363-370.
 54. Yuan C, Renfro L, Ambadwar PB, et al. Influence of genetic variation in the vitamin D pathway on plasma 25-hydroxyvitamin D(3) levels and survival among patients with metastatic colorectal cancer. *Cancer Causes Control*. 2019;30:757-765.
 55. Zgaga L, Theodoratou E, Farrington SM, et al. Plasma vitamin D concentration influences survival outcome after a diagnosis of colorectal cancer. *J Clin Oncol*. 2014;32:2430-2439.
 56. Keum N, Yuan C, Nishihara R, et al. Dietary glycemic and insulin scores and colorectal cancer survival by tumor molecular biomarkers. *Int J Cancer*. 2017;140:2648-2656.
 57. Hamada T, Liu L, Nowak JA, et al. Vitamin D status after colorectal cancer diagnosis and patient survival according to immune response to tumour. *Eur J Cancer*. 2018;103:98-107.
 58. Fuchs MA, Yuan C, Sato K, et al. Predicted vitamin D status and colon cancer recurrence and mortality in CALGB 89803 (Alliance). *Ann Oncol*. 2017;28:1359-1367.
 59. Ng K, Wolpin BM, Meyerhardt JA, et al. Prospective study of predictors of vitamin D status and survival in patients with colorectal cancer. *Br J Cancer*. 2009;101:916-923.
 60. Wesselink E, Staritsky LE, van Zutphen M, et al. The association between the adapted dietary inflammatory index and colorectal cancer recurrence and all-cause mortality. *Clin Nutr*. 2021;40:4436-4443.
 61. Zheng J, Tabung FK, Zhang J, et al. Post-cancer diagnosis dietary inflammatory potential is associated with survival among women diagnosed with colorectal cancer in the Women's Health Initiative. *Eur J Nutr*. 2020;59:965-977.
 62. Ratjen I, Shivappa N, Schafmayer C, et al. Association between the dietary inflammatory index and all-cause mortality in colorectal cancer long-term survivors. *Int J Cancer*. 2019;144:1292-1301.
 63. Sawayama H, Miyamoto Y, Mima K, et al. Preoperative iron status is a prognosis factor for stage II and III colorectal cancer. *Int J Clin Oncol*. 2021;26:2037-2045.
 64. Yang M, Zhang Q, Ruan GT, et al. Association between serum creatinine concentrations and overall survival in patients with colorectal cancer: a multi-center cohort study. *Front Oncol*. 2021;11:710423.

65. Long L, Yang W, Liu L, et al. Dietary intake of branched-chain amino acids and survival after colorectal cancer diagnosis. *Int J Cancer*. 2020;148:2471-2480.
66. Jiang R, Poschet G, Owen R, et al. Serum concentration of genistein, luteolin and colorectal cancer prognosis. *Nutrients*. 2019; 11:600.
67. Delphan M, Lin T, Liesenfeld DB, et al. Associations of branched-chain amino acids with parameters of energy balance and survival in colorectal cancer patients: results from the ColoCare study. *Metabolomics*. 2018;2018:22.
68. Maalmi H, Walter V, Jansen L, et al. Dose-response relationship between serum retinol levels and survival in patients with colorectal cancer: results from the DACHS study. *Nutrients*. 2018;10:510.
69. Lee S, Song A, Eo W. Serum ferritin as a prognostic biomarker for survival in relapsed or refractory metastatic colorectal cancer. *J Cancer*. 2016;7:957-964.
70. Psathakis D, Wedemeyer N, Oevermann E, Krug F, Siegers CP, Bruch HP. Blood selenium and glutathione peroxidase status in patients with colorectal cancer. *Dis Colon Rectum*. 1998;41: 328-335.
71. Wang Y, Liu P, Fang Y, et al. The effect of Long-term traditional Chinese medicine treatment on survival time of colorectal cancer based on propensity score matching: a retrospective cohort study. *Evid Based Complement Alternat Med*. 2020;2020:7023420.
72. Zhang M, Zhao QC, Liu YP, Yang L, Zhu HM, Chhetri JK. Prognostic analysis and comparison of colon cancer in Han and Hui patients. *World J Gastroenterol*. 2014;20:5082-5086.
73. Kuo YT, Liao CK, Chen TC, Lai CC, Chiang SF, Chiang JM. A high density of PD-L1-expressing immune cells is significantly correlated with favorable disease free survival in nonmetastatic colorectal cancer. *Medicine (Baltimore)*. 2022;101:e28573.
74. Sorensen LS, Rasmussen SL, Calder PC, Yilmaz MN, Schmidt EB, Thorlacius-Ussing O. Long-term outcomes after perioperative treatment with omega-3 fatty acid supplements in colorectal cancer. *BJS Open*. 2020;4:678-684.
75. Moertel CG, Fleming TR, Creagan ET, Rubin J, O'Connell MJ, Ames MM. High-dose vitamin C versus placebo in the treatment of patients with advanced cancer who have had no prior chemotherapy. A randomized double-blind comparison. *N Engl J Med*. 1985; 312:137-141.
76. Ng K, Nimeiri HS, McCleary NJ, et al. Effect of high-dose vs standard-dose vitamin D3 supplementation on progression-free survival among patients with advanced or metastatic colorectal cancer: the SUNSHINE randomized clinical trial. *Jama*. 2019;321:1370-1379.
77. Brown JC, Rosenthal MH, Ma C, et al. Effect of high-dose vs standard-dose vitamin D(3) supplementation on body composition among patients with advanced or metastatic colorectal cancer: a randomized trial. *Cancers (Basel)*. 2020;12:3451.
78. Urashima M, Ohdaira H, Akutsu T, et al. Effect of vitamin D supplementation on relapse-free survival among patients with digestive tract cancers: the AMATERASU randomized clinical trial. *Jama*. 2019;321:1361-1369.
79. Ravasco P, Monteiro-Grillo I, Camilo M. Individualized nutrition intervention is of major benefit to colorectal cancer patients: long-term follow-up of a randomized controlled trial of nutritional therapy. *Am J Clin Nutr*. 2012;96:1346-1353.
80. Song R, Petimar J, Wang M, et al. Adherence to the World Cancer Research Fund/American Institute for Cancer Research cancer prevention recommendations and colorectal cancer survival. *Cancer Epidemiol Biomarkers Prev*. 2021;30:1816-1825.
81. van Zutphen M, Boshuizen HC, Kenkhuis MF, et al. Lifestyle after colorectal cancer diagnosis in relation to recurrence and all-cause mortality. *Am J Clin Nutr*. 2021;113:1447-1457.
82. Cheng E, Zhang S, Ou FS, et al. The diet of higher insulinemic potential is not associated with worse survival in patients with stage III colon cancer (Alliance). *Cancer Epidemiol Biomarkers Prev*. 2020;29: 1692-1695.
83. Tabung FK, Noonan A, Lee DH, et al. Post-diagnosis dietary insulinemic potential and survival outcomes among colorectal cancer patients. *BMC Cancer*. 2020;20:817.
84. Van Blarigan EL, Zhang S, Ou FS, et al. Association of diet quality with survival among people with metastatic colorectal cancer in the Cancer and Leukemia B and Southwest Oncology Group 80405 Trial. *JAMA Netw Open*. 2020;3:e2023500.
85. Ginter MA, McCullough ML, Gapstur SM, Campbell PT. Associations of pre- and postdiagnosis diet quality with risk of mortality among men and women with colorectal cancer. *J Clin Oncol*. 2018; 36:JCO1800714.
86. Van Blarigan EL, Fuchs CS, Niedzwiecki D, et al. Association of Survival with Adherence to the American Cancer Society nutrition and physical activity guidelines for cancer survivors after colon cancer diagnosis: the CALGB 89803/Alliance trial. *JAMA Oncol*. 2018;4:783-790.
87. Liu L, Nishihara R, Qian ZR, et al. Association between inflammatory diet pattern and risk of colorectal carcinoma subtypes classified by immune responses to tumor. *Gastroenterology*. 2017;153:1517-1530. e14.
88. Ratjen I, Schafmayer C, di Giuseppe R, et al. Postdiagnostic Mediterranean and healthy Nordic dietary patterns are inversely associated with all-cause mortality in long-term colorectal cancer survivors. *J Nutr*. 2017;147:636-644.
89. Fung TT, Kashambwa R, Sato K, et al. Post diagnosis diet quality and colorectal cancer survival in women. *PLoS One*. 2014;9:e115377.
90. Inoue-Choi M, Robien K, Lazovich D. Adherence to the WCRF/AICR guidelines for cancer prevention is associated with lower mortality among older female cancer survivors. *Cancer Epidemiol Biomarkers Prev*. 2013;22:792-802.
91. Meyerhardt JA, Niedzwiecki D, Hollis D, et al. Association of dietary patterns with cancer recurrence and survival in patients with stage III colon cancer. *Jama*. 2007;298:754-764.
92. Ratjen I, Enderle J, Burmeister G, et al. Post-diagnostic reliance on plant-compared with animal-based foods and all-cause mortality in omnivorous long-term colorectal cancer survivors. *Am J Clin Nutr*. 2021;114:441-449.
93. Brown JC, Zhang S, Niedzwiecki D, et al. Grain intake and clinical outcome in stage III colon cancer: results from CALGB 89803 (Alliance). *JNCI Cancer Spectr*. 2018;2:pk017.
94. Song M, Wu K, Meyerhardt JA, et al. Fiber intake and survival after colorectal cancer diagnosis. *JAMA Oncol*. 2018;4:71-79.
95. Tamakoshi A, Nakamura K, Ukawa S, et al. Characteristics and prognosis of Japanese colorectal cancer patients: the BioBank Japan Project. *J Epidemiol*. 2017;27:S36-S42.
96. Fadelu T, Zhang S, Niedzwiecki D, et al. Nut consumption and survival in patients with stage III colon cancer: results from CALGB 89803 (Alliance). *J Clin Oncol*. 2018;36:1112-1120.
97. Van Blarigan EL, Ou FS, Bainter TM, et al. Associations between unprocessed red meat and processed meat with risk of recurrence and mortality in patients with stage III colon cancer. *JAMA Netw Open*. 2022;5:e220145.
98. McCullough ML, Gapstur SM, Shah R, Jacobs EJ, Campbell PT. Association between red and processed meat intake and mortality among colorectal cancer survivors. *J Clin Oncol*. 2013;31:2773-2782.
99. Song M, Ou FS, Zemla TJ, et al. Marine omega-3 fatty acid intake and survival of stage III colon cancer according to tumor molecular markers in NCCTG phase III trial N0147 (Alliance). *Int J Cancer*. 2019;145:380-389.
100. Van Blarigan EL, Fuchs CS, Niedzwiecki D, et al. Marine omega-3 polyunsaturated fatty acid and fish intake after colon cancer

- diagnosis and survival: CALGB 89803 (Alliance). *Cancer Epidemiol Biomarkers Prev.* 2018;27:438-445.
101. Liu X, Yang W, Wu K, et al. Postdiagnostic dairy products intake and colorectal cancer survival in US males and females. *Am J Clin Nutr.* 2021;113:1636-1646.
 102. Yang B, McCullough ML, Gapstur SM, et al. Calcium, vitamin D, dairy products, and mortality among colorectal cancer survivors: the cancer prevention study-II nutrition cohort. *J Clin Oncol.* 2014;32:2335-2343.
 103. Fuchs MA, Sato K, Niedzwiecki D, et al. Sugar-sweetened beverage intake and cancer recurrence and survival in CALGB 89803 (Alliance). *PLoS One.* 2014;9:e99816.
 104. Zoltick ES, Smith-Warner SA, Yuan C, et al. Sugar-sweetened beverage, artificially sweetened beverage and sugar intake and colorectal cancer survival. *Br J Cancer.* 2021;125:1016-1024.
 105. Guercio BJ, Zhang S, Niedzwiecki D, et al. Associations of artificially sweetened beverage intake with disease recurrence and mortality in stage III colon cancer: results from CALGB 89803 (Alliance). *PLoS One.* 2018;13:e0199244.
 106. Ugai T, Haruki K, Vayrynen JP, et al. Coffee intake of colorectal cancer patients and prognosis according to histopathologic lymphocytic reaction and T-cell infiltrates. *Mayo Clin Proc.* 2022;97:124-133.
 107. Mackintosh C, Yuan C, Ou FS, et al. Association of coffee intake with survival in patients with advanced or metastatic colorectal cancer. *JAMA Oncol.* 2020;6:1713-1721.
 108. Hu Y, Ding M, Yuan C, et al. Association between coffee intake after diagnosis of colorectal cancer and reduced mortality. *Gastroenterology.* 2018;154:916-926.e9.
 109. Guercio BJ, Sato K, Niedzwiecki D, et al. Coffee intake, recurrence, and mortality in stage III colon cancer: results from CALGB 89803 (Alliance). *J Clin Oncol.* 2015;33:3598-3607.
 110. Yang B, Gapstur SM, Newton CC, Jacobs EJ, Campbell PT. Alcohol intake and mortality among survivors of colorectal cancer: the cancer prevention study II nutrition cohort. *Cancer.* 2017;123:2006-2013.
 111. Lochhead P, Nishihara R, Qian ZR, et al. Postdiagnostic intake of one-carbon nutrients and alcohol in relation to colorectal cancer survival. *Am J Clin Nutr.* 2015;102:1134-1141.
 112. Atinafu BT, Bulti FA, Demelew TM. Survival status and predictors of mortality among colorectal cancer patients in Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia: a retrospective followup study. *J Cancer Prev.* 2020;25:38-47.
 113. Maeda M, Nagawa H, Maeda T, Koike H, Kasai H. Alcohol consumption enhances liver metastasis in colorectal carcinoma patients. *Cancer.* 1998;83:1483-1488.
 114. Ramjeesingh R, Orr C, Bricks CS, Hopman WM, Hammad N. A retrospective study on the role of diabetes and metformin in colorectal cancer disease survival. *Curr Oncol.* 2016;23:e116-e122.
 115. Song M, Wu K, Meyerhardt JA, et al. Low-carbohydrate diet score and macronutrient intake in relation to survival after colorectal cancer diagnosis. *JNCI Cancer Spectr.* 2018;2:pk077.
 116. Meyerhardt JA, Sato K, Niedzwiecki D, et al. Dietary glycemic load and cancer recurrence and survival in patients with stage III colon cancer: findings from CALGB 89803. *J Natl Cancer Inst.* 2012;104:1702-1711.
 117. Yuan C, Bao Y, Sato K, et al. Influence of dietary insulin scores on survival in colorectal cancer patients. *Br J Cancer.* 2017;117:1079-1087.
 118. Morales-Oyarvide V, Yuan C, Babic A, et al. Dietary insulin load and cancer recurrence and survival in patients with stage III colon cancer: findings from CALGB 89803 (Alliance). *J Natl Cancer Inst.* 2019;111:170-179.
 119. Van Blarigan EL, Ou FS, Niedzwiecki D, et al. Dietary fat intake after colon cancer diagnosis in relation to cancer recurrence and survival: CALGB 89803 (Alliance). *Cancer Epidemiol Biomarkers Prev.* 2018;27:1227-1230.
 120. Song M, Zhang X, Meyerhardt JA, et al. Marine omega-3 polyunsaturated fatty acid intake and survival after colorectal cancer diagnosis. *Gut.* 2017;66:1790-1796.
 121. Inoue-Choi M, Greenlee H, Oppeneer SJ, Robien K. The association between postdiagnosis dietary supplement use and total mortality differs by diet quality among older female cancer survivors. *Cancer Epidemiol Biomarkers Prev.* 2014;23:865-875.
 122. Abdel-Rahman O, Spratlin J, Koski S. Vitamin and herbal supplements' use among patients with advanced gastrointestinal cancers included in eight clinical trials. *J Cancer Res Clin Oncol.* 2020;146:2089-2097.
 123. Ng K, Meyerhardt JA, Chan JA, et al. Multivitamin use is not associated with cancer recurrence or survival in patients with stage III colon cancer: findings from CALGB 89803. *J Clin Oncol.* 2010;28:4354-4363.
 124. Yang W, Ma Y, Smith-Warner S, et al. Calcium intake and survival after colorectal cancer diagnosis. *Clin Cancer Res.* 2019;25:1980-1988.
 125. Boakye D, Jansen L, Schottker B, et al. The association of vitamin D with survival in colorectal cancer patients depends on antioxidant capacity. *Am J Clin Nutr.* 2021;113:1458-1467.
 126. Kim J, Baek DW, Baek JH, et al. Clinical impact of postoperative vitamin D deficiency on the recurrence of colon cancer after curative surgical resection. *Anticancer Res.* 2021;41:3683-3688.
 127. Sinicrope FA, Shi Q, Smyrk TC, et al. Association of adiponectin and vitamin D with tumor infiltrating lymphocytes and survival in stage III colon cancer. *JNCI Cancer Spectr.* 2021;5:pkab070.
 128. Wesselink E, Kok DE, de Wilt JHW, et al. Sufficient 25-hydroxyvitamin D levels 2 years after colorectal cancer diagnosis are associated with a lower risk of all-cause mortality. *Cancer Epidemiol Biomarkers Prev.* 2021;30:765-773.
 129. Bao Y, Li Y, Gong Y, Huang Q, Cai S, Peng J. Vitamin D status and survival in stage II-III colorectal cancer. *Front Oncol.* 2020;10:581597.
 130. Markotic A, Langer S, Kelava T, et al. Higher post-operative serum vitamin D level is associated with better survival outcome in colorectal cancer patients. *Nutr Cancer.* 2019;71:1078-1085.
 131. Yuan C, Sato K, Hollis BW, et al. Plasma 25-hydroxyvitamin D levels and survival in patients with advanced or metastatic colorectal cancer: findings from CALGB/SWOG 80405 (Alliance). *Clin Cancer Res.* 2019;25:7497-7505.
 132. Maalmi H, Walter V, Jansen L, et al. Relationship of very low serum 25-hydroxyvitamin D(3) levels with long-term survival in a large cohort of colorectal cancer patients from Germany. *Eur J Epidemiol.* 2017;32:961-971.
 133. Facciorusso A, Del Prete V, Muscatiello N, Crucinio N, Barone M. Prognostic role of 25-hydroxyvitamin D in patients with liver metastases from colorectal cancer treated with radiofrequency ablation. *J Gastroenterol Hepatol.* 2016;31:1483-1488.
 134. Vaughan-Shaw PG, Zgaga L, Ooi LY, et al. Low plasma vitamin D is associated with adverse colorectal cancer survival after surgical resection, independent of systemic inflammatory response. *Gut.* 2020;69:103-111.
 135. Cooney RV, Chai W, Franke AA, Wilkens LR, Kolonel LN, Le Marchand L. C-reactive protein, lipid-soluble micronutrients, and survival in colorectal cancer patients. *Cancer Epidemiol Biomarkers Prev.* 2013;22:1278-1288.
 136. Ng K, Sargent DJ, Goldberg RM, et al. Vitamin D status in patients with stage IV colorectal cancer: findings from Intergroup Trial N9741. *J Clin Oncol.* 2011;29:1599-1606.
 137. Mezawa H, Sugiura T, Watanabe M, et al. Serum vitamin D levels and survival of patients with colorectal cancer: post-hoc analysis of a prospective cohort study. *BMC Cancer.* 2010;10:347.
 138. Schulze MB, Martinez-Gonzalez MA, Fung TT, Lichtenstein AH, Forouhi NG. Food based dietary patterns and chronic disease prevention. *BMJ.* 2018;361:k2396.

139. Wang P, Song M, Eliassen AH, et al. Optimal dietary patterns for prevention of chronic disease. *Nat Med*. 2023;29:719-728.
140. Aleksandrova K, Koelman L, Rodrigues CE. Dietary patterns and biomarkers of oxidative stress and inflammation: a systematic review of observational and intervention studies. *Redox Biol*. 2021;42:101869.
141. Barbaresco J, Koch M, Schulze MB, Nothlings U. Dietary pattern analysis and biomarkers of low-grade inflammation: a systematic literature review. *Nutr Rev*. 2013;71:511-527.
142. English CJ, Mayr HL, Lohning AE, Reidlinger DP. The association between dietary patterns and the novel inflammatory markers platelet-activating factor and lipoprotein-associated phospholipase A2: a systematic review. *Nutr Rev*. 2022;80:1371-1391.
143. Koelman L, Egea Rodrigues C, Aleksandrova K. Effects of dietary patterns on biomarkers of inflammation and immune responses: a systematic review and meta-analysis of randomized controlled trials. *Adv Nutr*. 2022;13:101-115.
144. Neale EP, Batterham MJ, Tapsell LC. Consumption of a healthy dietary pattern results in significant reductions in C-reactive protein levels in adults: a meta-analysis. *Nutr Res*. 2016;36:391-401.
145. Wu PY, Chen KM, Tsai WC. The Mediterranean dietary pattern and inflammation in older adults: a systematic review and meta-analysis. *Adv Nutr*. 2021;12:363-373.
146. Craddock JC, Neale EP, Peoples GE, Probst YC. Vegetarian-based dietary patterns and their relation with inflammatory and immune biomarkers: a systematic review and meta-analysis. *Adv Nutr*. 2019;10:433-451.
147. Huo R, Du T, Xu Y, et al. Effects of Mediterranean-style diet on glycemic control, weight loss and cardiovascular risk factors among type 2 diabetes individuals: a meta-analysis. *Eur J Clin Nutr*. 2015;69:1200-1208.
148. Lee J, Jeon JY, Meyerhardt JA. Diet and lifestyle in survivors of colorectal cancer. *Hematol Oncol Clin North Am*. 2015;29:1-27.
149. Van Blarigan EL, Meyerhardt JA. Role of physical activity and diet after colorectal cancer diagnosis. *J Clin Oncol*. 2015;33:1825-1834.
150. Schmidt DR, Patel R, Kirsch DG, Lewis CA, Vander Heiden MG, Locasale JW. Metabolomics in cancer research and emerging applications in clinical oncology. *CA Cancer J Clin*. 2021;71:333-358.
151. Mayne ST, Playdon MC, Rock CL. Diet, nutrition, and cancer: past, present and future. *Nat Rev Clin Oncol*. 2016;13:504-515.
152. Steck SE, Murphy EA. Dietary patterns and cancer risk. *Nat Rev Cancer*. 2020;20:125-138.
153. Wade KH, Yarmolinsky J, Giovannucci E, et al. Applying Mendelian randomization to appraise causality in relationships between nutrition and cancer. *Cancer Causes Control*. 2022;33:631-652.
154. Aune D, Keum N, Giovannucci E, et al. Whole grain consumption and risk of cardiovascular disease, cancer, and all cause and cause specific mortality: systematic review and dose-response meta-analysis of prospective studies. *BMJ*. 2016;353:i2716.
155. Burkitt DP. Epidemiology of cancer of the colon and rectum. *Cancer*. 1971;28:3-13.
156. Um CY, Peters BA, Choi HS, et al. Grain, gluten, and dietary fiber intake influence gut microbial diversity: data from the food and microbiome longitudinal investigation. *Cancer Res Commun*. 2023;3:43-53.
157. Gomes SD, Oliveira CS, Azevedo-Silva J, et al. The role of diet related short-chain fatty acids in colorectal cancer metabolism and survival: prevention and therapeutic implications. *Curr Med Chem*. 2020;27:4087-4108.
158. Milesi G, Rangan A, Grafenauer S. Whole grain consumption and inflammatory markers: a systematic literature review of randomized control trials. *Nutrients*. 2022;14:374.
159. Sanders LM, Zhu Y, Wilcox ML, Koecher K, Maki KC. Whole grain intake, compared to refined grain, improves postprandial glycemia and insulinemia: a systematic review and meta-analysis of randomized controlled trials. *Crit Rev Food Sci Nutr*. 2021;5:1-19.
160. Buldak RJ, Hejmo T, Osowski M, et al. The impact of coffee and its selected bioactive compounds on the development and progression of colorectal cancer in vivo and in vitro. *Molecules*. 2018;23:3309.
161. Halvorsen BL, Carlsen MH, Phillips KM, et al. Content of redox-active compounds (ie, antioxidants) in foods consumed in the United States. *Am J Clin Nutr*. 2006;84:95-135.
162. Kang NJ, Lee KW, Kim BH, et al. Coffee phenolic phytochemicals suppress colon cancer metastasis by targeting MEK and TOPK. *Carcinogenesis*. 2011;32:921-928.
163. Choi DW, Lim MS, Lee JW, et al. The cytotoxicity of kahweol in HT-29 human colorectal cancer cells is mediated by apoptosis and suppression of heat shock protein 70 expression. *Biomol Ther (Seoul)*. 2015;23:128-133.
164. Kim HG, Hwang YP, Han EH, et al. The coffee diterpene kahweol inhibits metastasis by modulating expressions of MMPs and VEGF via STAT3 inactivation. *Food Chem*. 2012;133:1521-1529.
165. Riihimäki M, Hemminki A, Sundquist J, Hemminki K. Patterns of metastasis in colon and rectal cancer. *Sci Rep*. 2016;6:29765.
166. Epner M, Yang P, Wagner RW, Cohen L. Understanding the link between sugar and cancer: an examination of the preclinical and clinical evidence. *Cancers (Basel)*. 2022;14:6042.
167. Debras C, Chazelas E, Srour B, et al. Artificial sweeteners and cancer risk: results from the NutriNet-Santé population-based cohort study. *PLoS Med*. 2022;19:e1003950.
168. Krishnan AV, Feldman D. Mechanisms of the anti-cancer and anti-inflammatory actions of vitamin D. *Annu Rev Pharmacol Toxicol*. 2011;51:311-336.
169. van Harten-Gerritsen AS, Balvers MG, Witkamp RF, Kampman E, van Duynhoven FJ. Vitamin D, inflammation, and colorectal cancer progression: a review of mechanistic studies and future directions for epidemiological studies. *Cancer Epidemiol Biomarkers Prev*. 2015;24:1820-1828.
170. Yasueda A, Urushima H, Ito T. Efficacy and interaction of antioxidant supplements as adjuvant therapy in cancer treatment: a systematic review. *Integr Cancer Ther*. 2016;15:17-39.
171. Bours MJ, Beijer S, Winkels RM, et al. Dietary changes and dietary supplement use, and underlying motives for these habits reported by colorectal cancer survivors of the Patient Reported Outcomes Following Initial Treatment and Long-Term Evaluation of Survivorship (PROFILES) registry. *Br J Nutr*. 2015;114:286-296.
172. Savitz DA, Wellenius GA, Trikalinos TA. The problem with mechanistic risk of bias assessments in evidence synthesis of observational studies and a practical alternative: assessing the impact of specific sources of potential bias. *Am J Epidemiol*. 2019;188:1581-1585.
173. Bonet C, Crous-Bou M, Tsilidis KK, et al. The association between body fatness and mortality among breast cancer survivors: results from a prospective cohort study. *Eur J Epidemiol*. 2023;38:545-557.
174. Carr PR, Jansen L, Walter V, et al. Associations of red and processed meat with survival after colorectal cancer and differences according to timing of dietary assessment. *Am J Clin Nutr*. 2016;103:192-200.
175. Lewis CM, Wolf WA, Xun P, Sandler RS, He K. Racial differences in dietary changes and quality of life after a colorectal cancer diagnosis: a follow-up of the study of outcomes in colorectal cancer survivors cohort. *Am J Clin Nutr*. 2016;103:1523-1530.
176. Collaborators GBDCC. Global, regional, and national burden of colorectal cancer and its risk factors, 1990-2019: a systematic analysis for the global burden of disease study 2019. *Lancet Gastroenterol Hepatol*. 2022;7:627-647.
177. Carethers JM, Doubeni CA. Causes of socioeconomic disparities in colorectal cancer and intervention framework and strategies. *Gastroenterology*. 2020;158:354-367.

178. Friedenreich CM, Shaw E, Neilson HK, Brenner DR. Epidemiology and biology of physical activity and cancer recurrence. *J Mol Med (Berl)*. 2017;95:1029-1041.
179. Spei ME, Bellos I, Samoli E, Benetou V. Post-diagnosis dietary patterns among cancer survivors in relation to all-cause mortality and cancer-specific mortality: a systematic review and meta-analysis of cohort studies. *Nutrients*. 2023;15:3860.
180. Demark-Wahnefried W, Rogers LQ, Alfano CM, et al. Practical clinical interventions for diet, physical activity, and weight control in cancer survivors. *CA Cancer J Clin*. 2015;65:167-189.
181. Joseph R, Hart NH, Bradford N, et al. Diet and exercise advice and referrals for cancer survivors: an integrative review of medical and nursing perspectives. *Support Care Cancer*. 2022;30:8429-8439.
182. Orsso CE, Ford KL, Kiss N, et al. Optimizing clinical nutrition research: the role of adaptive and pragmatic trials. *Eur J Clin Nutr*. 2023;77:1130-1142.
183. Cross AJ, Gunter MJ. Coffee and colorectal cancer: grounds for prevention? *Gastroenterology*. 2018;154:790-792.
184. Kim J, Lee HK. Potential role of the gut microbiome in colorectal cancer progression. *Front Immunol*. 2021;12:807648.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Chan DSM, Cariolou M, Markozannes G, et al. Post-diagnosis dietary factors, supplement use and colorectal cancer prognosis: A Global Cancer Update Programme (CUP Global) systematic literature review and meta-analysis. *Int J Cancer*. 2024;155(3):445-470. doi:[10.1002/ijc.34906](https://doi.org/10.1002/ijc.34906)