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Adiposity and Cancer Survival: A Systematic Review and Meta-analysis

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Abstract

Purpose: The increasing availability of clinical imaging tests (especially CT and MRI) that directly quantify adipose tissue has led to a rapid increase in studies examining the relationship of visceral, subcutaneous, and overall adiposity to cancer survival. To summarize this emerging body of literature, we conducted a systematic review and meta-analysis of imaging-measured as well as anthropometric proxies for adipose tissue distribution and cancer survival across a wide range of cancer types.

Methods: Using keywords related to adiposity, cancer and survival, we conducted a systematic search of the literature in PubMed and MEDLINE, Embase, and Web of Science Core Collection databases from database inception to June 30, 2021. We used a random-effect method to calculate pooled hazard ratios (HR) and corresponding 95% confidence intervals (CI) within each cancer type, and tested for heterogeneity using Cochran's Q test and the I^2 test.

Results: We included 203 records for this review, of which 128 records were utilized for quantitative analysis among 10 cancer types: breast, colorectal, gastroesophageal, head and neck, hepatocellular carcinoma, lung, ovarian, pancreatic, prostate, and renal cancer. We found that imaging-measured visceral, subcutaneous, and total adiposity were not significantly associated with increased risk of overall mortality, death from primary cancer, or cancer progression among patients diagnosed with these 10 cancer types; however, we found significant or high heterogeneity for many cancer types. For example, heterogeneity was similarly high when the pooled HRs (95% CI) for overall mortality associated with visceral adiposity were essentially null as in 1.03 (0.55, 1.92; I^2 = 58%) for breast, 0.99 (0.81, 1.21; I^2 = 71%) for colorectal, versus when they demonstrated a potential increased risk 1.77 (0.85, 1.60; I^2 = 78%) for hepatocellular carcinoma and 1.62 (0.90, 2.95; I^2 = 84%) for renal cancer.

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Author Contributions

All authors contributed to the study conception and design. En Cheng, Jocelyn Kirley, and Elizabeth M. Cespedes Feliciano participated in the literature search and screening. En Cheng and Jocelyn Kirley performed study quality assessment. En Cheng conducted data analysis. En Cheng and Bette Caan drafted the manuscript. All authors critically revised the manuscript for important intellectual content and approved the final manuscript.

Competing Interests: None

Ethics approval

This study is a meta-analysis and does not involve human subjects. IRB review is not required.

Conclusion: Greater adiposity at diagnosis (directly measured by imaging) is not associated with worse survival among cancer survivors. However, heterogeneity and other potential limitations were noted across studies, suggesting differences in study design and adiposity measurement approaches, making interpretation of meta-analyses challenging. Future work to standardize imaging measurements and data analyses will strengthen research on the role of adiposity in cancer survival.

Keywords

Cancer; Survival; Adiposity; Computed Tomography; Body Composition; Waist-Hip Ratio

BACKGROUND

Excessive body weight (overweight or obesity) has been associated with increased incidence of 13 types of cancers [1], which are estimated to account for over 40% of all cancers diagnosed in the United States in 2022 [2]. Excessive body weight is associated with a number of systemic and local changes that are hypothesized to promote cancer initiation and progression, for example, increased circulating levels of insulin and glucose as well as adipose tissue-derived hormones and inflammatory mediators [3–6]. Yet, paradoxically, often overweight (body mass index [BMI]: 25–29.9 kg/m^2) and sometimes obesity (BMI ≥ 30 kg/m^2) are reported to be associated with more favorable outcomes among many of these 13 cancers as well as other cancer types [7, 8]. A potential reason for this is that although BMI is correlated with overall body fatness [9], it does not distinguish muscle from adipose tissue nor quantify specific adipose tissue depots. While, on average, all of these tissues increase in quantity with increasing body size, the relationships of muscle and visceral and subcutaneous adipose tissue to cancer survival often differ [10–12]; and there is substantial variation in body composition between individuals with identical body size, particularly among older patients with chronic conditions such as cancer [10, 11, 13]. Therefore, studies using more precise measures of overall body fatness that can also distinguish specific adipose tissue depots are needed to understand the relationship of adiposity to cancer survival.

The increasing availability of clinical imaging has led to a rapid increase in research using more precise measures of body fatness [14]. Imaging tests such as computed tomography (CT) are frequently performed among cancer patients for diagnostic purposes. Reference methods to directly quantify both total adipose tissue and specific adipose tissue depots from partial fields of view have been developed, and measurements correlate well with whole body volumes on magnetic resonance imaging (MRI) [15]. To summarize this emerging literature on adiposity and cancer survival, we conducted a meta-analysis of imaging-measured adiposity and survival in multiple cancer types. Given that waist-based anthropometric measures are commonly used as surrogates for visceral adiposity and triceps skinfolds for subcutaneous adiposity, we also included these anthropometric measures in our review and meta-analysis.

METHODS

We registered this review (No. CRD42021262968) *a priori* at PROSPERO, an international database of prospectively registered systematic reviews with health-related outcomes [16]. This review followed the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement [17]. Ethical approval was not applicable due to not involving human participants.

Data Sources and Searches

A systematic search of the literature was conducted in PubMed and MEDLINE, Embase, and Web of Science Core Collection databases from database inception to June 30, 2021. Key Medical Subject Headings (MeSH) terms were predefined for adiposity and cancer survival, and details of the full searching strategy are listed in Supplementary Table 1.

Inclusion Criteria

We included prospective studies that estimated associations of adiposity (measured by imaging and anthropometry) and survival after cancer diagnosis among adults (≥18 years). Only studies published in English were included. Case reports, case-control studies, cross-sectional studies, ecologic studies, conference abstracts, reviews, guidelines, perspectives, editorials, letters, and non-human research were excluded. Given that BMI has been the subject of several prior systematic reviews [18–40], and our focus is specifically adiposity and adipose tissue distribution rather than body size, we excluded studies that solely focused on BMI. We also excluded studies that used bioelectrical impedance (BIA) since it is not an imaging technique for estimating total body fat and is based on the assumption of constant hydration status that cancer patients may not meet, which may produce biased estimates of total body fat [41, 42]. Also, studies combining multiple different cancers in analysis were excluded.

Data Extraction and Quality Assessment

Titles and abstracts were screened independently by two reviewers (E. Cheng and J. Kirley) to identify articles related to adiposity and cancer survival. Afterwards, the full texts of those considered eligible were also independently reviewed by the same two authors. Any discrepancies were evaluated by another author (E. M. Cespedes Feliciano) and discussed among these three authors. We extracted the following information from each study: descriptive characteristics of the study population (sample size, country, age, sex, stage, and race and ethnicity); follow-up; adiposity assessment method (for imaging studies) or assessment time (for anthropometry studies); adiposity classification (how adiposity was measured and compared); outcome results; and covariates if multivariable models were applied. For publications using duplicate or overlapping cohorts, we selected the publications with the largest sample size. We also contacted study authors when we needed clarification and additional information not available in the online publications and supplementary materials.

Two reviewers (E. Cheng and J. Kirley) independently assessed the quality of the studies using the Quality Assessment Tool for Observational Cohort and Cross-Sectional

Studies developed by the National Heart, Lung, and Blood Institute (NHLBI) [43]. Any discrepancies were settled after further discussion among these two authors. This tool includes 14 questions focusing on key concepts for evaluating internal validity including considerations regarding the study population, sample size, exposure and outcome assessment, timeframe, loss to follow-up and control for confounding. Rather than creating a list that can simply add up to judge quality, NHLBI encourages investigators to examine the study comprehensively with this tool and then give overall quality rating (good, fair, or poor). If the articles were finally evaluated as poor, we excluded them from systematic review and meta-analysis.

Primary Exposure

In addition to measuring total adipose tissue (TAT), imaging via CT, MRI or Dual X-Ray absorptiometry (DXA) can further separately measure or estimate visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT).

- Visceral adiposity was typically measured via 1) VAT area [cm^2] on a single-slice abdominal scan, 2) VAT area scaled by height squared [cm^2/m^2], 3) VAT-related ratios such as VAT/SAT and VAT/TAT, 4) VAT volume [cm^3] across multiple slices or estimated from three-dimensional imaging models, 5) VAT mass (kg) calculated from estimation formulas [44], and 6) VAT thickness [mm] defined as the imaging distance between abdominal wall and an abdominal organ of interest [45, 46].
- Subcutaneous adiposity was typically measured via 1) SAT area [cm^2] at a single-slice abdominal scan, 2) SAT area scaled by height squared [cm^2/m^2], 3) SAT-related ratios such as SAT/VAT and SAT/TAT, 3) subcutaneous volume [cm^3] across multiple slices or estimated from three-dimensional imaging models, and 4) subcutaneous mass (kg) calculated from estimation formulas [44].
- Total adiposity was generally considered as a sum of SAT and VAT, and was typically measured via 1) TAT area [cm^2] at a single-slice abdominal scan, 2) TAT area scaled by height squared [cm^2/m^2], 3) TAT volume [cm^3] across multiple slices or three-dimensional imaging models, and 4) TAT mass (kg) calculated from estimation formulas [15, 47–49].

As for anthropometric measures, waist-hip ratio (WHR), waist circumference (WC), and waist-height ratio (WHtR) were usually measured as surrogates for visceral adiposity [50, 51], and triceps skinfold thickness has been widely used as an measurement of subcutaneous fat [52, 53]. For meta-analysis in anthropometric measures, when a study investigated multiple measures of visceral adiposity, we would prioritize the findings of WHR over WC and WHtR.

The primary exposure of interest is adiposity, defined as greater quantity of adipose tissue in two main depots (visceral and/or subcutaneous) as well as total body fat (sum of visceral, subcutaneous, and other adipose tissue depots, if measured). For studies categorizing adiposity measures, we used the risk estimate that compared the highest and lowest

quantiles, representing patients with most excessive adipose tissue (highest quantile) and least adipose tissue (lowest quantile). For studies (N = 32) analyzing adiposity measures as continuous variables, their findings were reported in different ways (such as one unit increase and one standard deviation increase) and included in a sensitivity analysis for meta-analysis.

Primary Outcomes

For this analysis, three primary outcomes of interest were defined as follows. Overall survival (OS) was defined as the time from cancer diagnosis until death from any cause. Cancer-specific survival (CSS) was defined as the time from cancer diagnosis until death from the primary cancer. Progression-free survival (PFS) was defined as the time from cancer diagnosis until cancer recurrence, metastasis, or other events (such as new lesions and second primary tumors) suggesting cancer growth.

Statistical Analysis

For each cancer, at least two studies were needed for a meta-analysis [54, 55], and we calculated a pooled hazard ratio (HR) with 95% confidence interval (CI) using the DerSimonian-Laird method for random-effects meta-analysis [56]. Heterogeneity between the studies was calculated both using Cochran's Q test and the I^2 test [57]. A p value <0.05 or I^2 value >75% suggested significant evidence of heterogeneity, whereas for I^2 values, ≤25%, 26–50%, and 51–75% suggested low, moderate, and high heterogeneity [57, 58]. For each cancer, we assessed publication bias with funnel plots if there were ≥10 studies [59, 60], and further examined it with Begg and Mazumdar rank correlation test [61]. All statistical analyses were conducted using R statistical software version 4.1.2 (R Project for Statistical Computing) from November 19, 2021 to December 16, 2021. All p values were 2-sided, and the significance level was set at $p = 0.05$.

RESULTS

The searching and screening process is shown in Figure 1: we included 202 records for this review, of which 128 records were utilized for quantitative analysis. For imaging, there were 10 cancer types with ≥2 eligible studies for at least one outcome (OS, CSS, or PFS) to conduct a meta-analysis, including breast, colorectal, gastroesophageal, head and neck, hepatocellular carcinoma, lung, ovarian, pancreatic, prostate, and renal cancer. In contrast, for anthropometry, there were three cancer types eligible for meta-analysis, including breast, colorectal, and prostate cancer.

IMAGING

Breast

In breast cancer, there were 7 records included for meta-analysis and summary estimates are presented in Figure 2. More details of published records included into the meta-analysis are in Supplementary Table 2 [62–68]. Visceral, subcutaneous, and total adiposity were not significantly associated with OS among breast cancer patients (HRs for visceral: 1.03 [0.55, 1.92]; subcutaneous: 1.36 [0.90, 2.05]; total: 1.14 [0.77, 1.69]), and corresponding

heterogeneity was high for visceral and total adiposity, but low for subcutaneous adiposity. No meta-analysis could be performed for CSS, but one record suggested that visceral adiposity was associated with increased risk of death from breast cancer (HR: 1.18 [1.02, 1.37]) and subcutaneous was not (HR: 0.92 [0.78, 1.08]) [69]. No records in total adiposity and CSS have been published. Visceral, subcutaneous, and total adiposity were not significantly associated with risk of breast cancer progression (HRs for visceral: 1.20 [0.40, 3.57]; subcutaneous: 1.01 [0.53, 1.93]; total: 0.89 [0.48, 1.67]), and corresponding heterogeneity was significant for visceral adiposity, low for subcutaneous, and high for total adiposity.

Colorectal Cancer (CRC)

In CRC, there were 27 records included for meta-analysis and summary estimates are presented in Figure 3. More details of published records included into the meta-analysis are in Supplementary Table 3 [70–96]. Visceral, subcutaneous, and total adiposity were not significantly associated with OS among CRC patients (HRs for visceral: 0.99 [0.81, 1.21]; subcutaneous: 1.01 [0.77, 1.32]; total: 0.89 [0.58, 1.37]), and corresponding heterogeneity were significant for all three adiposity measures. Similarly, visceral, subcutaneous, and total adiposity were not significantly associated with risk of death from CRC (HRs for visceral: 0.92 [0.77, 1.08]; subcutaneous: 0.78 [0.57, 1.07]; total: 1.11 [0.83, 1.49]), and corresponding heterogeneity was low for visceral and subcutaneous adiposity, but high for total adiposity. Visceral, subcutaneous, and total adiposity were not significantly associated with risk of CRC progression (HRs for visceral: 1.07 [0.75, 1.54]; subcutaneous: 0.78 [0.48, 1.25]; total: 0.69 [0.21, 2.30]), and corresponding heterogeneity was significant for visceral adiposity, and high for subcutaneous and total adiposity.

Gastroesophageal

In gastroesophageal cancer, there were 15 records included for meta-analysis and summary estimates are presented in Figure 4. More details of published records included into the meta-analysis are in Supplementary Table 4 [97–111]. Visceral adiposity was not significantly associated with OS among gastroesophageal cancer patients (HR: 0.87 [0.67, 1.14]), whereas subcutaneous adiposity was associated with a decreased risk of mortality (HR: 0.64 [0.46, 0.90]) and no study was done for total adiposity and OS. Corresponding heterogeneity statistics were significant for both visceral and subcutaneous adiposity measures. No records have been published for CSS. Visceral adiposity was not significantly associated with risk of gastroesophageal cancer progression (HR: 0.89 [0.33, 2.42]), and corresponding heterogeneity was significant. No PFS meta-analysis could be performed for subcutaneous and total adiposity, but two studies suggested non-significant associations [112, 113]: HR (1.001 [0.998, 1.004]) for subcutaneous adiposity (measured as continuous SAT index); $p = 0.47$ for total adiposity analyzed using the Kaplan-Meier estimator.

Head and Neck

In head and neck cancer, there were 3 records included for meta-analysis and summary estimates are presented in Figure 5. More details of published records included into the meta-analysis are in Supplementary Table 5 [114–116]. No meta-analysis could be performed for OS, but three studies investigated the associations of visceral, subcutaneous,

and total adiposity, respectively. Visceral adiposity was not significantly associated with risk of mortality (HR: 0.35 [0.09, 1.43]) [114], whereas subcutaneous and total adiposity were associated with a decreased risk of mortality (HRs for subcutaneous: 0.60 [0.48, 0.76]); total: 0.29 [0.10, 0.83]) [116, 117]. No records have been published for CSS. Visceral and subcutaneous adiposity were not significantly associated with risk of head and neck cancer progression (HRs for visceral: 1.36 [0.28, 6.58]; subcutaneous: 1.00 [0.49, 2.03]), and heterogeneity was significant for visceral adiposity and high for subcutaneous adiposity. No PFS meta-analysis could be performed for total adiposity, but one suggested a significant association with lower risk of progression (HR: 0.27 [0.10, 0.71]) [117].

Hepatocellular Carcinoma (HCC)

In HCC, there were 11 records included for meta-analysis and summary estimates are presented in Figure 6. More details of published records included into the meta-analysis are in Supplementary Table 6 [118–128]. Visceral, subcutaneous, and total adiposity were not significantly associated with risk of mortality among HCC patients (HRs for visceral: 1.17 [0.85, 1.60]; subcutaneous: 1.10 [0.55, 2.21]; total: 1.05 [0.44, 2.48]), and corresponding heterogeneity were significant for all three adiposity measures. No records have been published for CSS. Visceral adiposity was not significantly associated with risk of HCC progression (HR: 0.96 [0.73, 1.28]), and heterogeneity was significant for visceral adiposity. No PFS meta-analysis could be performed for subcutaneous and total adiposity. Two studies reported subcutaneous adiposity (measured as continuous SAT index) was inconsistently associated with PFS: one was significant (HR: 1.03 [1.01. 1.05]) whereas the other was not (HR: 1.00 [0.99, 1.01]) [129, 130], and one study reported total adiposity (measured as continuous TAT index) was significantly associated with increased risk of progression (HR: 1.03 [1.01. 1.05]) [129].

Lung

In lung cancer, there were 7 records included for meta-analysis and summary estimates are presented in Figure 7. More details of published records included into the meta-analysis are in Supplementary Table 7 [44, 131–136]. Visceral and subcutaneous adiposity were not significantly associated with OS among lung cancer patients (HRs for visceral: 1.05 [0.90, 1.22]; subcutaneous: 0.74 [0.45, 1.20]), and corresponding heterogeneity was low for visceral and high for subcutaneous adiposity. Only one study investigated total adiposity and OS and reported no association (HR: 0.99 [0.68, 1.46]) [137]. No records have been published for CSS. Visceral adiposity was not significantly associated with risk of lung cancer progression (HR: 1.06 [0.85, 1.33]), and heterogeneity for studies was low. No records have been published for subcutaneous and total adiposity and PFS.

Ovarian

In ovarian cancer, there were 4 records included for meta-analysis and summary estimates are presented in Figure 8. More details of published records included into the meta-analysis are in Supplementary Table 8 [138–141]. Visceral and subcutaneous adiposity were not significantly associated with OS among ovarian patients (HRs for visceral: 0.53 [0.11, 2.66]; subcutaneous: 0.65 [0.19, 2.19]), and corresponding heterogeneity was significant for both visceral and subcutaneous adiposity. Only one study investigated total adiposity (tertiles)

and OS with only an insignificant p value ($p = 0.33$) reported for the Kaplan-Meier estimator [142]. No records have been published for CSS. Visceral adiposity was not significantly associated with risk of ovarian cancer progression (HR: 0.76 [0.44, 1.30]) but subcutaneous adiposity was associated with a borderline decreased risk (HR: 0.76 [0.58, 1.00]), and corresponding heterogeneity was high for visceral adiposity and low for subcutaneous adiposity. No records have been published for total adiposity and PFS.

Pancreatic

In pancreatic cancer, there were 7 records included for meta-analysis and summary estimates are presented in Figure 9. More details of published records included into the meta-analysis are in Supplementary Table 9 [45, 143–148]. Visceral and subcutaneous adiposity were not significantly associated with OS among pancreatic cancer patients (HRs for visceral: 1.05 [0.88, 1.26]; subcutaneous: 0.81 [0.44, 1.49]), and corresponding heterogeneity was low for visceral adiposity and moderate for subcutaneous adiposity. Only one study investigated total adiposity and OS, and reported an insignificant association [149]. No records have been published for CSS. No meta-analysis could be performed for visceral and subcutaneous adiposity and PFS, and no records have been published for total adiposity and PFS. However, four studies investigated visceral adiposity and PFS, and two reported significant, adverse associations (HR: 1.01 [1.00, 1.02] for continuous VAT measures; and $p = 0.04$ for the Kaplan-Meier estimator) whereas the others did not (HR: 1.00 [0.99, 1.01] for continuous VAT measures; and $p > 0.05$ for the Kaplan-Meier estimator) [150–153]. Two studies investigated subcutaneous adiposity and PFS, and both reported no association (HR: 0.98 [0.83, 1.15] for continuous SAT measures; and $p > 0.05$ for the Kaplan-Meier estimator) [150, 153].

Prostate

In prostate cancer, there were 12 records included for meta-analysis and summary estimates are presented in Figure 10. More details of published records included into the meta-analysis are in Supplementary Table 10 [154–165]. Visceral and total adiposity were not significantly associated with OS among prostate cancer patients (HRs for visceral: 1.07 [0.84, 1.35]; total: 0.88 [0.65, 1.20]), but subcutaneous adiposity was associated with a decreased risk: 0.69 [0.57, 0.84]; corresponding heterogeneity was high for visceral adiposity, and low for subcutaneous and total adiposity. Visceral adiposity was not significantly associated with risk of death from prostate cancer (HR: 1.02 [0.76, 1.35]), whereas subcutaneous adiposity was associated with a decreased risk (HR: 0.73 [0.55, 0.98]). Heterogeneity was low for both visceral and subcutaneous adiposity. No records have been published for total adiposity and CSS. Visceral and total adiposity were not significantly associated with prostate cancer progression (HRs for visceral: 1.04 [0.87, 1.24]; total: 0.95 [0.73, 1.24]), and subcutaneous adiposity was significantly associated with decreased prostate cancer progression (HR: 0.81 [0.68, 0.97]). Heterogeneity was moderate for visceral adiposity, and low for subcutaneous and total adiposity.

Renal

In renal cancer, there were 13 records included for meta-analysis and summary estimates are presented in Figure 11. More details of published records included into the meta-analysis are

in Supplementary Table 11 [46, 166–177]. Visceral, subcutaneous, and total adiposity were not significantly associated with OS among renal cancer patients (HRs for visceral: 1.62 [0.90, 2.95]; subcutaneous: 0.90 [0.25, 3.27]; total: 0.87 [0.65, 1.18]), and corresponding heterogeneity was significant for visceral and subcutaneous adiposity, and low for total adiposity. Visceral adiposity was not significantly associated with increased risk of death from renal cancer (HR: 2.47 [0.09, 67.43]) in the two studies examining this association, and heterogeneity was significant. No records have been published for subcutaneous and total adiposity and CSS. Visceral and subcutaneous adiposity were not significantly associated with renal caner progression (HRs for visceral: 0.85 [0.32, 2.27]; subcutaneous: 1.29 [0.31, 5.37]), and heterogeneity was significant for both. No records have been published for total adiposity and PFS.

Other Cancers

There were 18 records in adiposity and survival in 12 cancer types for which no meta-analysis could be conducted. These cancer types were adrenocortical, acute myeloid leukemia (AML), biliary, bladder, cholangiocarcinoma, endometrial, lymphoma, melanoma, multiple myeloma, nasopharyngeal, sarcoma, and urinary tract. More details of these published records are in Supplementary Table 12 [178–195]. Most studies reported insignificant associations or *p* values suggesting that greater adiposity may not be associated with worse survival among most of these cancer types.

Sensitivity Analysis

There were 52 records not included into the above meta-analyses due to 1) analyzing adiposity measure as continuous variables, or 2) only reporting *p* values (most were >0.05). More details are presented in Supplementary Tables 13–22 [69, 112, 113, 117, 129, 130, 137, 142, 149–153, 196–234]. After including studies in meta-analysis that analyzed adiposity measure as continuous variables, the results of the overall pattern remained almost same: imaging-measured adiposity was not significantly associated with risk of mortality or progression (Supplementary Table 23). However, three associations became significant: higher subcutaneous adiposity was significantly associated with worse OS (HR: 1.14 [1.03, 1.27]) and worse PFS (HR: 1.02 [1.01–1.03]) in breast cancer; and higher visceral adiposity was significantly associated with worse OS (HR: 1.14 [1.01–1.29]) in lung cancer. The inverse association of higher subcutaneous adiposity and OS remained significant in prostate cancer, but the magnitude of HR was slightly attenuated from 0.73 (0.55, 0.98) to 0.77 (0.61, 0.96).

Publication Bias

Funnel plots and Begg and Mazumdar rank correlation tests suggested no publication bias, except for visceral adiposity and overall survival in renal cancer ($p = 0.04$). More details are in Supplementary Figure 1 and Supplementary Table 24.

ANTHROPOMETRY

Breast

In breast cancer, there were 14 records for meta-analysis and summary estimates are presented in Figure 12. More details of published records included into the meta-analysis are in Supplementary Table 25 [235–248]. Visceral adiposity assessed by the proxy measure of waist-related anthropometric measures was significantly associated with increased risk of mortality (HR: 1.30 [1.15, 1.46]) and death from breast cancer (HR: 1.26 [1.03, 1.55]), but not with breast cancer progression (HR: 1.17 [0.88, 1.55]). Heterogeneity was moderate for all three estimates. No records have been published on subcutaneous adiposity and breast cancer survival assessed by anthropometric proxies.

Colorectal Cancer (CRC)

In colorectal cancer, there were 5 records for meta-analysis and summary estimates are presented in Figure 13. More details of published records included into the meta-analysis are found in Supplementary Table 26 [249–253]. Waist-related anthropometric measures were significantly associated with increased risk of mortality (HR: 1.24 [1.04, 1.47]) and death from CRC (HR: 1.27 [1.08, 1.49]), and heterogeneity was moderate and high for OS and CSS, respectively. No records have been published for 1) visceral adiposity assessed by waist-related anthropometric measure and PFS, and 2) subcutaneous adiposity assessed by anthropometric measures and CRC survival.

Prostate

In prostate cancer, there were 3 records for meta-analysis and summary estimates are presented in Figure 14. More details of published records included into the meta-analysis are in Supplementary Table 27 [254–256]. Waist-related anthropometric measures were not associated with increased risk of mortality (HR: 1.11 [0.90, 1.36]) or death from prostate cancer (HR: 1.02 [0.81, 1.29]), and heterogeneity was low for both OS and CSS. No records have been published for 1) visceral adiposity assessed by waist-related anthropometric measure and PFS, and 2) subcutaneous adiposity assessed by anthropometric measures and prostate cancer survival.

Other Cancers

There were 4 records in adiposity and survival in 4 cancer types that no meta-analysis could be conducted. These cancer types were gastroesophageal, HCC, lung, and lymphoma. More details of these published records are in Supplementary Table 28 [257–260]. Most studies reported insignificant associations or *p* values suggesting that greater adiposity may not be associated with worse survival among these cancer types.

Publication Bias

Funnel plots and Begg and Mazumdar rank correlation tests for breast cancer suggested no publication bias (Supplementary Figure 1 and Supplementary Table 24).

Comparison of Findings in Imaging Studies vs. Anthropometry Studies

The associations of visceral adiposity with survival for meta-analysis were assessed in both imaging studies and anthropometry studies for breast, colorectal, and prostate cancer. To enable easier comparison, we summarized their findings in Supplementary Table 29.

The Impact of Stage on Findings

Since very few studies reported stage-specific estimates, the exact impact of stage on the meta-analysis of adiposity (imaging-measured or anthropometry-measured adiposity) and cancer survival could not be quantified. However, to provide some insight, we replicated meta-analyses for adiposity and overall survival by restricting to studies with only non-metastatic cancer stage (at least two studies were needed). The findings (Supplementary Table 30) were similar to those without restricting to only non-metastatic stage, except that 1) the association between imaging-measured visceral adiposity and overall survival became significant for pancreatic cancer and 2) the association between anthropometry-measured visceral adiposity and overall survival became not significant for breast cancer. Some potential explanations for such changes may be: 1) for non-metastatic pancreatic cancer patients, visceral adiposity was an indicator for increased pancreatic steatosis that was associated with increased lymphatic invasion, positive lymph nodes, and decreased survival after pancreatoduodenectomy [261]; and 2) for breast cancer, the number of anthropometric studies included for meta-analysis decreased from 13 to 3, after restricting to studies with only non-metastatic stage; however, the magnitude of the HR became larger from 1.30 (1.15, 1.46) to 1.50 (0.76, 2.98) although becoming not significant.

DISCUSSION

In the most comprehensive study to date of imaging measures of adiposity and cancer-related outcomes across 10 cancer sites (breast, colorectal, gastroesophageal, head and neck, HCC, lung, ovarian, pancreatic, prostate, and renal), we found visceral, subcutaneous, and total adiposity were not significantly associated with mortality, death from primary cancer, or cancer progression. For gastroesophageal, head and neck, ovarian and prostate cancer, subcutaneous adiposity appeared protective and was associated with significantly lower mortality risk. Conversely, anthropometric proxies for visceral adiposity were significantly associated with increased risk of overall mortality and death from primary cancer among patients with breast and colorectal cancer.

Several explanations exist for our findings. First, excess adiposity is often associated with higher levels of muscle needed to support extra weight. Patients with low muscle mass often have higher risk of recurrence, overall and cancer-specific mortality [262]. Thus, any risk due to excess adiposity, unless adiposity is extremely high, may be attenuated by the benefits of having adequate muscle which has shown to be protective [72]. In this study, out of 128 records included for meta-analysis, only 11 (8.6%) studies included muscle mass or sarcopenia as covariates for adjusted models and studies without such adjustment may underestimate the unfavorable effects of adiposity. Second, adiposity to a certain extent may provide protective nutritional reserves, especially in the form of SAT, which is considered a more inert storage depot than VAT and has not been linked as strongly to adverse metabolic

sequelae [263]. A moderate amount of SAT may enable patients to survive the catabolic effects of cancer and its treatments, and the resulting weight and muscle loss that can occur [264]. Third, tumor biology may be related to excess adiposity, leading to heterogeneity in diseases previously assumed to be similar [265]. For example, in patients with clear cell renal carcinoma (ccRCC), adverse metabolic oncogene fatty acid synthase (*FASN*) was downregulated in obese patients with ccRCC and upregulated in those who were normal weight [265]. Furthermore, advanced ccRCC tumors of obese patients showed higher angiogenic scores than those of normal-weight patients, which may explain why obese patients survive longer when treated with antiangiogenic treatment [266]. This suggests that either ccRCC in patients with excess adiposity is more indolent, or that the adipose tissue surrounding the tumor may alter its metabolism, resulting in less aggressive disease.

Very few previous reviews or meta-analyses have examined imaging measures of adiposity and cancer survival. One systematic review included 22 prognostic studies and only examined VAT [267]. It concluded that adverse associations between VAT and survival were more frequently observed among patients with colorectal (four of six studies included in that systematic review) and pancreatic (three of five studies included in that systematic review) cancers, compared to higher VAT predicting longer survival in most studies of renal cell carcinoma patients (four of five studies included in that systematic review) [267]. While our study did not see an increased risk between VAT and survival of colorectal and pancreatic cancers, nor a decreased risk associated with renal cancer, their review included fewer studies, a number of which had a small sample size and did not distinguish different survival outcomes such as OS and PFS. As we conducted quantitative analyses by incorporating a larger number of studies, our estimates are more representative of the associations of VAT and other adiposity measures of survival among different cancer types [268].

Bias due to reverse causation or collider stratification bias could also be considered an explanation for our predominantly null findings. In the case of reverse causation, since most imaging measurements are taken during clinical cancer care for diagnostic and surveillance purposes, patients with more advanced cancers may have already lost tissue by time of diagnosis [269]; thus, lower adiposity could be caused by adipose tissue wasting due to more aggressive disease [269, 270]. Collider bias is a specific type of selection bias that occurs when analyses are restricted to a select subgroup (e.g., cancer patients) experiencing a condition that is causally influenced by two or more variables [271]: e.g., if cancer incidence is caused by excess adiposity, which also increases mortality after cancer diagnosis, but among patients with low levels of adiposity cancer incidence is due exclusively to unrelated factors that also sharply increase mortality risk (e.g., genetics predisposing to an aggressive tumor). Thus, when analyses are restricted to cancer patients, an inverse association is artificially induced between excess adiposity and cancer survival [8]. However, previous studies suggest that these biases, while plausible, are unlikely to fully explain associations [272, 273].

In our meta-analysis of studies using anthropometric measures, results align with previous meta-analyses [30, 274] and suggest that excess adiposity was associated with increased risk of mortality for both breast and colorectal cancer patients. The inconsistency of the results from these anthropometric studies that compared to those from imaging studies directly

measuring VAT and SAT could be due to the use of either less precise measures, timing of assessment, or better control for confounding. As a reference standard for measuring body composition [275], CT and MRI provide more direct quantifications of adipose tissue than anthropometric measures and distinguish adipose tissue distribution [7, 8]. Second, patient populations may vary between studies using anthropometry and studies using CT. Anthropometric studies likely include a higher proportion of patients diagnosed with earlier stage disease as these measures are collected as part of research studies for which healthier individuals may volunteer, whereas imaging is most often collected as part of routine clinical care for select stages and cancer sites. For example, in breast cancer, CT scans are typically only available on stage III and IV patients and a small percentage of patients with stage II disease. In contrast, anthropometric studies likely include all stages of disease, of which stage I constitute a large proportion of the patient population. If the effect of adiposity differs by stage, which was observed in a study where BMI increased risk of mortality in CRC patients at stage I/II but not stage III/IV [276], results from anthropometric studies may demonstrate higher risk with adiposity than imaging studies. Additionally, compared to imaging studies, anthropometry studies have overall larger sample size and adjusted for potential confounders more comprehensively. For example, for breast cancer, we observed overall larger sample size and more covariates for adjustment in anthropometry studies (Supplementary Table 25) vs. imaging studies (Supplementary Table 3). In addition, when multiple waist-based measures were available, we prioritized WHR, which may reflect both abdominal adiposity (increased waist circumference) as well as a lack of gluteal muscularity (decreased hip circumference); thus, the harms of excess adiposity may be more apparent after controlling for muscularity. Third, timing of measurement could affect results in several ways. While CT and MRI, if ordered, are requested for cancer diagnostics [277, 278], many of the anthropometric measurements were done several years before cancer diagnosis. For example, in our analysis for waist-based anthropometric measures and survival in colorectal cancer, 4 out of 5 studies included into meta-analysis assessed on average 5–8 years before diagnosis. These pre-diagnosis measurements capture, in part, the increased risk of cancer incidence associated with excess adiposity, and may not accurately reflect the relationship of at-diagnosis adiposity to survival. Since clinicians are confronted with at-diagnosis adiposity when making decisions about cancer treatment and supportive lifestyle interventions, it is the relationship of at-diagnosis adiposity to cancer outcomes that is most relevant to patient care. Given that this systematic review and meta-analysis primarily focuses on single timepoint for adiposity and adiposity is dynamic and can change over time, future studies could focus on the prognostic roles of adiposity change among cancer survivors, which is also of high clinical relevance to clinicians and patients.

Strengths and limitations

To our knowledge, our analysis provides the first and most comprehensive summary of the evidence on adiposity and cancer survival across a wide range of cancer types. In addition to meta-analysis of adiposity among 10 cancer types, we also reviewed imaging-measured and anthropometry-measured adiposity studies among varied cancers, including adrenocortical, AML, biliary, bladder, cholangiocarcinoma, endometrial, lymphoma, melanoma, multiple myeloma, nasopharyngeal, sarcoma, and urinary tract. Although these cancer types did not

have sufficient studies for meta-analysis, most reported null associations of adiposity and cancer survival.

However, our study has several limitations. First, we noticed significant and high heterogeneity in meta-analyses of many cancer types. In imaging-measured studies, methods are not yet standardized, and investigators choose different anatomic landmarks to quantify body composition or apply different Hounsfield unit (HU) ranges to quantify adipose tissue. In addition, adiposity exposures were categorized in the different ways as well as being scaled as continuous variables in different forms across the studies, which may further result in heterogeneity. Second, not all studies fit multivariable models to report adjusted HRs, particularly if adiposity-related variables were not significantly associated with survival in univariate analyses. However, while unadjusted for confounders, these null HRs align with the null findings reported in our analysis. Third, due to the relatively small number of studies in some cancer types (for example, head and neck [N = 3] and ovarian [N = 4]) and potential publication bias for renal cancer, we should cautiously interpret findings in these cancers. Fourth, we did not conduct subgroup analyses, and future studies should further explore the associations between adiposity and cancer survival by demographic and clinicopathologic characteristics such as age, sex, and disease stage, which may contribute to heterogeneity. For example, stage is among key factors impacting survival, and the associations of adiposity with survival may differ by stage due to the increased prevalence of adipose tissue wasting in advanced disease. Since few studies included for this meta-analysis reported stage-specific findings, the exact impact of staging on our findings could not be calculated based on current published reports. However, to provide some insight into the impact of stage on our findings, we conducted meta-analysis for adiposity and overall survival by restricting to studies with only non-metastatic cancer stage, and the findings remained similar. Although no publication bias was observed in our analysis of waist-based measures of adiposity and decreased survival among breast cancer survivors, we note that half of studies reported insignificant associations and exposure assessments occurred pre-, peri-, and post-diagnosis, suggesting some level of heterogeneity. Fifth, only 11 (8.6%) out of 128 studies included in this review controlled for muscularity in multivariable models, but even those which did control for muscle did not find a significant increased risk of cancer-related outcomes. Sixth, there may be selection concerns for imaging-measured adiposity studies among some but not all cancer types. For example, CT scans are not routinely used for breast cancer at stage I and II, but they are commonly ordered for all stages of colorectal cancer at diagnosis and follow-up to determine cancer staging. Thus, such selection bias may affect the generalizability of our findings in some cancer types.

CONCLUSIONS

Imaging-measured visceral, subcutaneous, and total adiposity were not associated with increased risk of overall mortality, death from primary cancer, or cancer progression among patients with breast, colorectal, gastroesophageal, head and neck, HCC, lung, ovarian, pancreatic, prostate, and renal cancer. In some cancers, excess SAT adiposity was associated with better survival. However, given high heterogeneity of studies included and the rapid increase in the use of clinical imaging to examine the relationship of body composition to cancer outcomes, more scientific rigor must be employed before robust comparisons can be

made, including standardized vertebral landmarks, and establishment of relevant cut points to indicate excess adiposity. The ultimate goal of standardization is to enable comparison across studies to understand the role of adiposity in cancer survival, and to provide clinicians with risk stratification tools to identify the most vulnerable patients and design appropriate interventions to enhance survivorship.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability

The datasets analyzed during the current study are publicly available via applying the searching algorithm proposed in this manuscript to PubMed, Embase and Web of Science.

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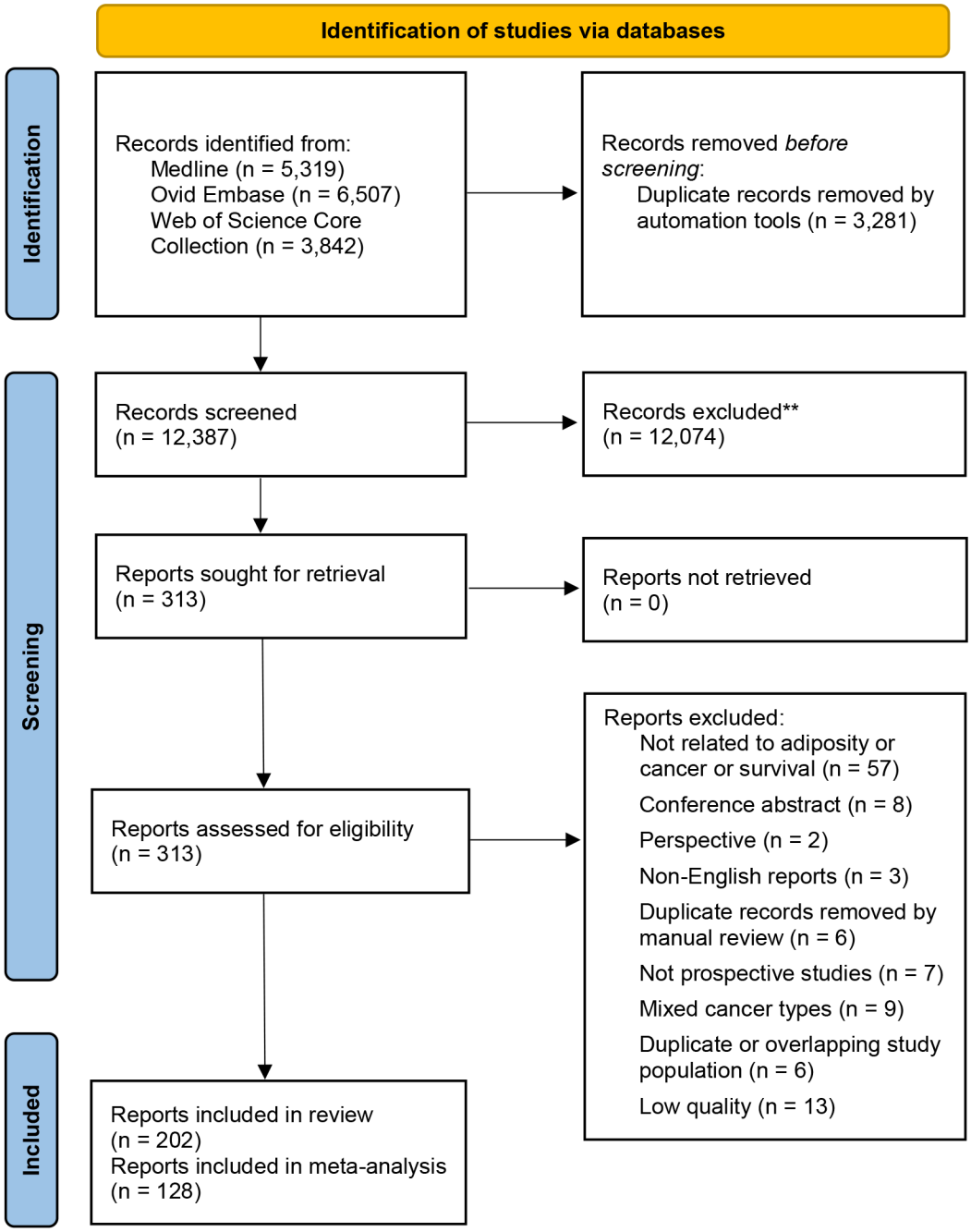


Figure 1.
PRISMA 2020 Flow Diagram for the Systematic Review Which Included Searches of Databases Only

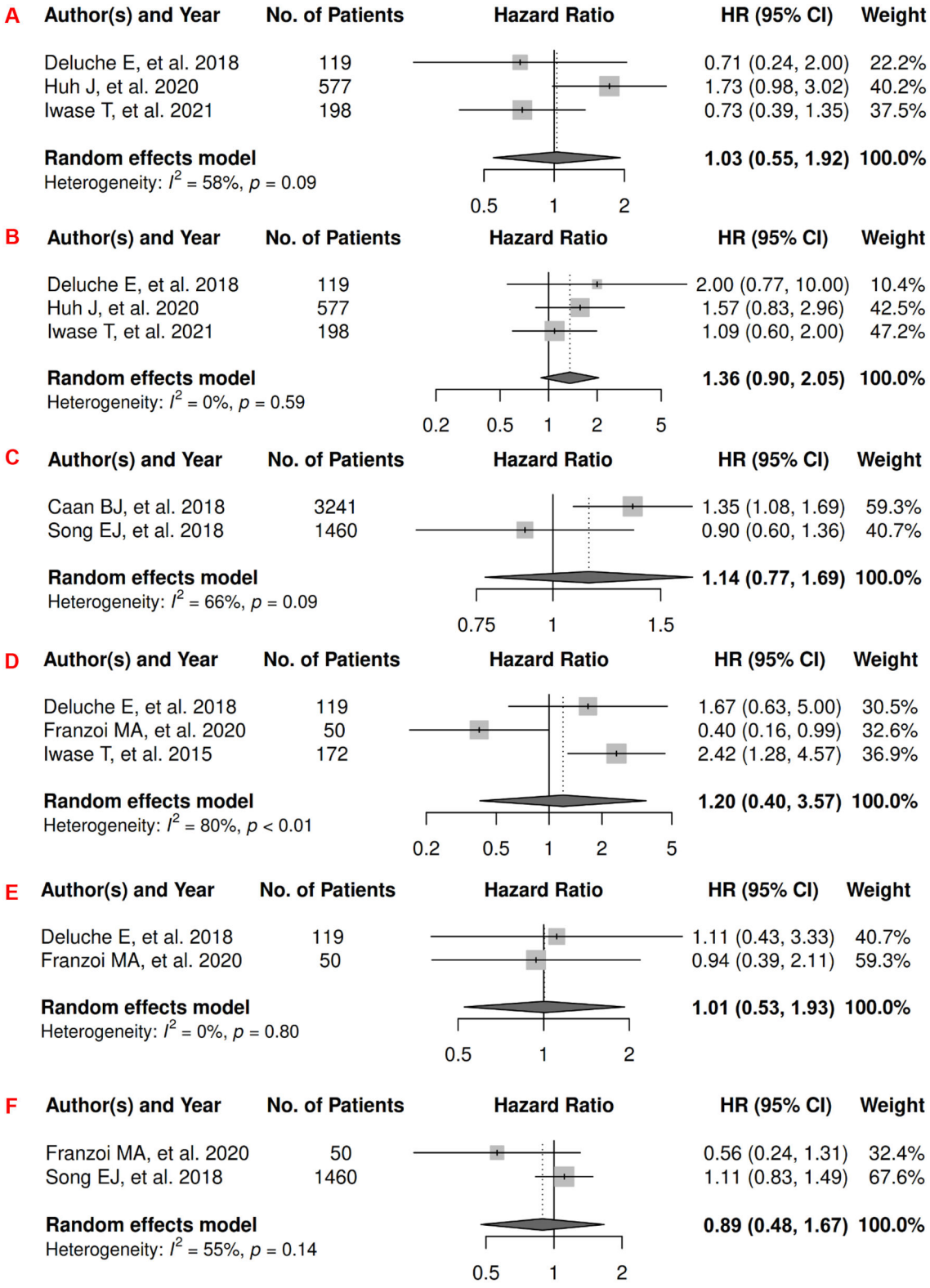


Figure 2.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Breast Cancer

2A: visceral adiposity and overall survival.

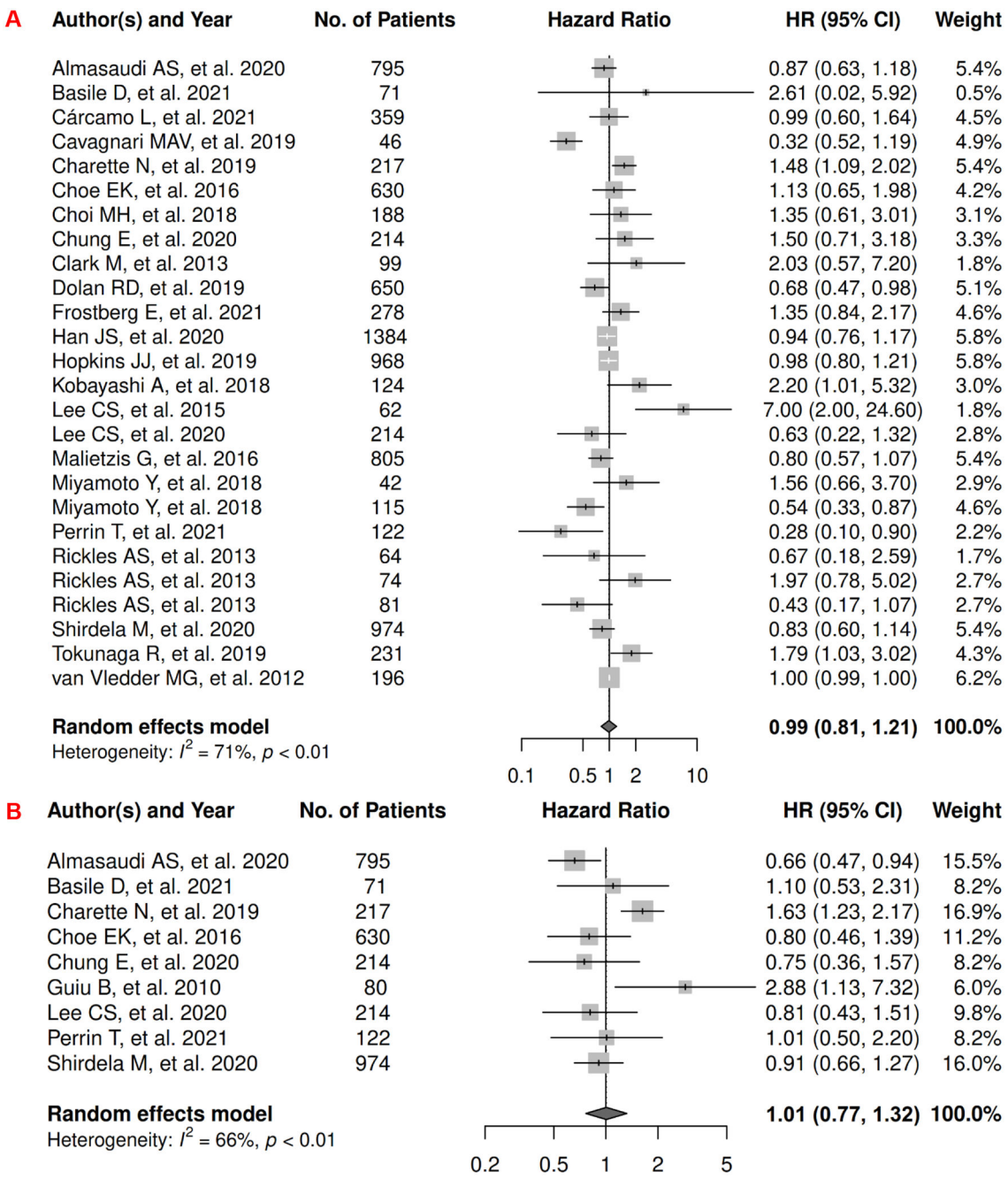
2B: subcutaneous adiposity and overall survival.

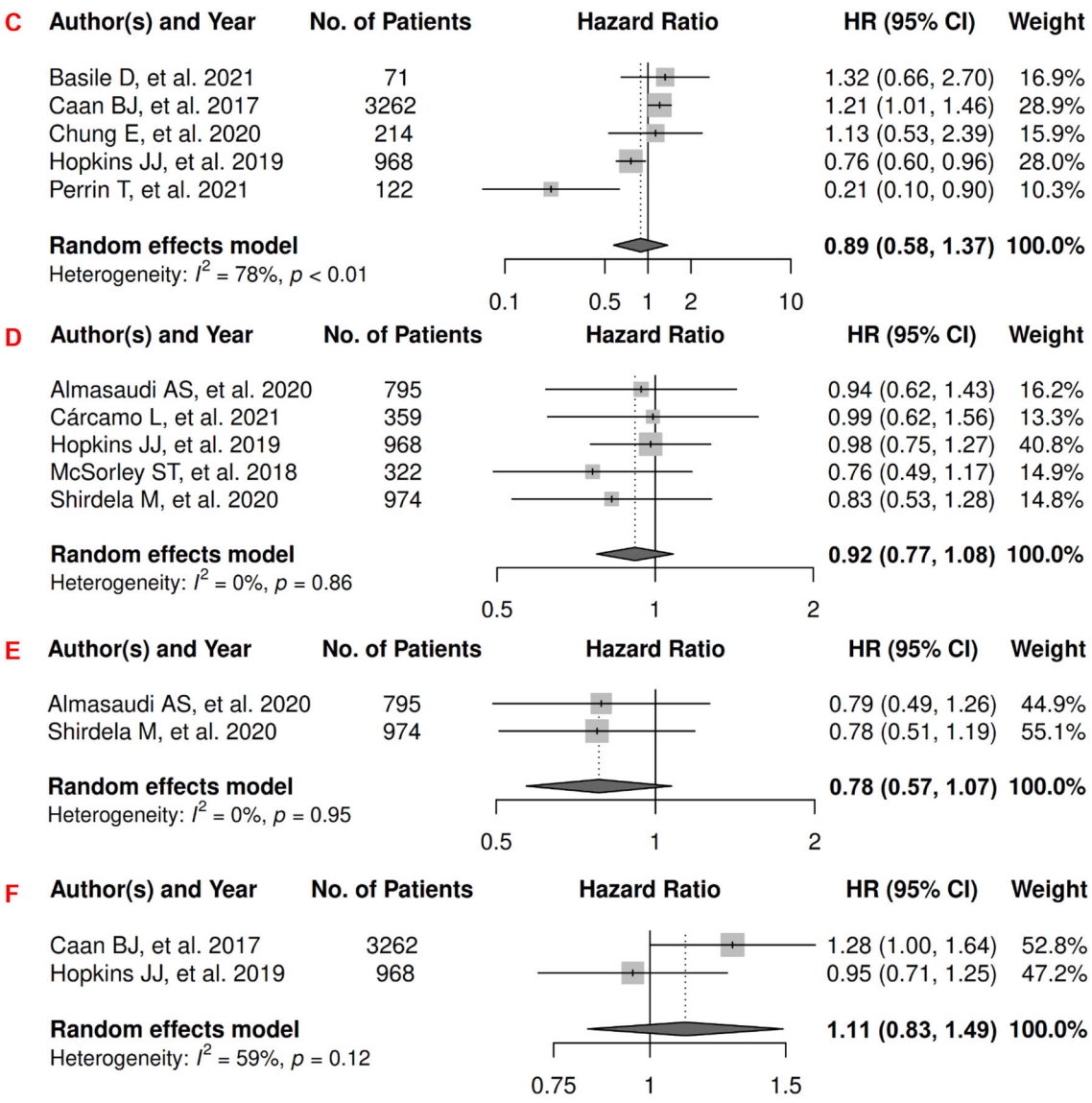
2C: total adiposity and overall survival.

2D: visceral adiposity and progression-free survival.

2E: subcutaneous adiposity and progression-free survival.

2F: total adiposity and progression-free survival.





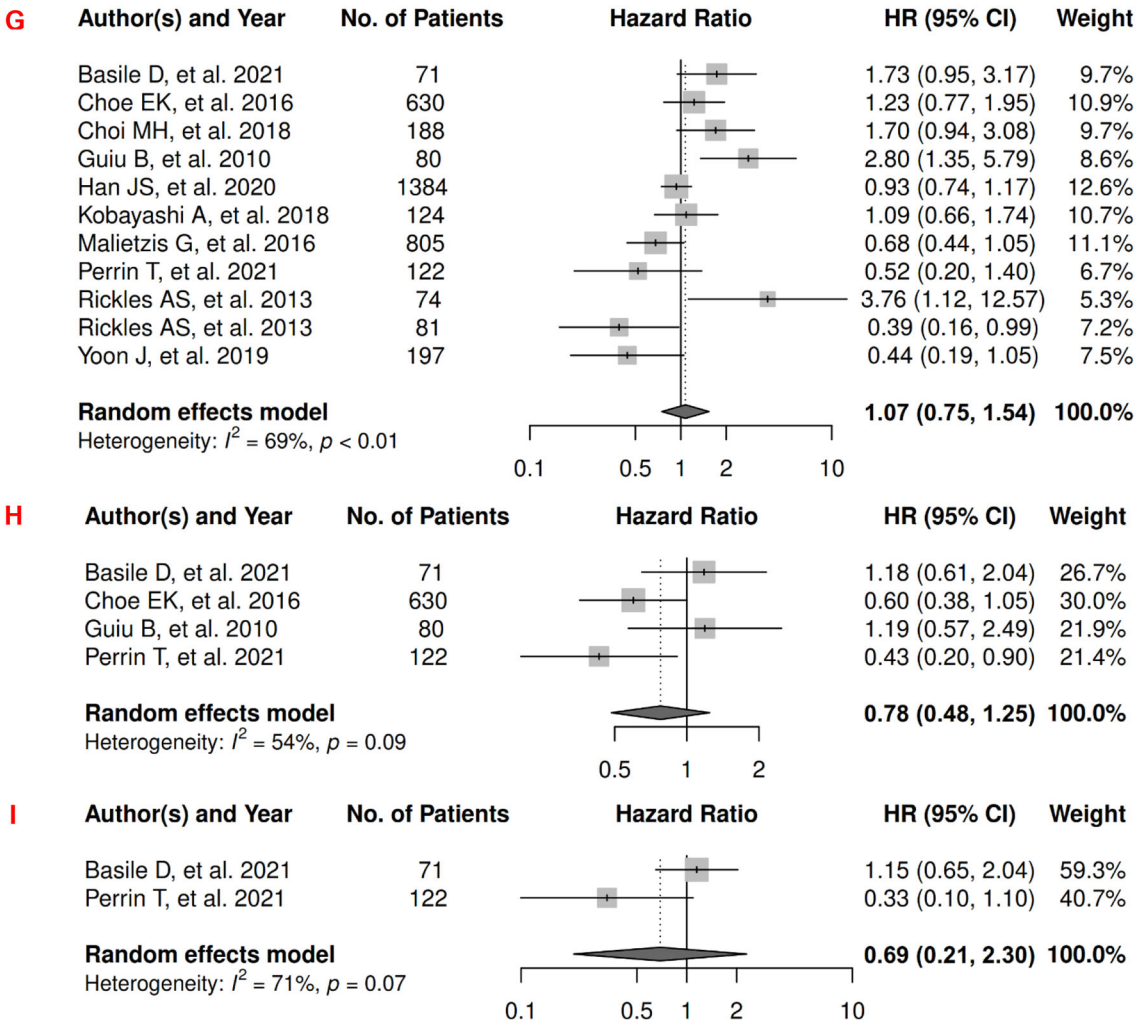


Figure 3.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Colorectal Cancer

3A: visceral adiposity and overall survival.

3B: subcutaneous adiposity and overall survival.

3C: total adiposity and overall survival.

3D: visceral adiposity and cancer-specific survival.

3E: subcutaneous adiposity and cancer-specific survival.

3F: total adiposity and cancer-specific survival.

3G: visceral adiposity and progression-free survival.

3H: subcutaneous adiposity and progression-free survival.

3I: total adiposity and progression-free survival.

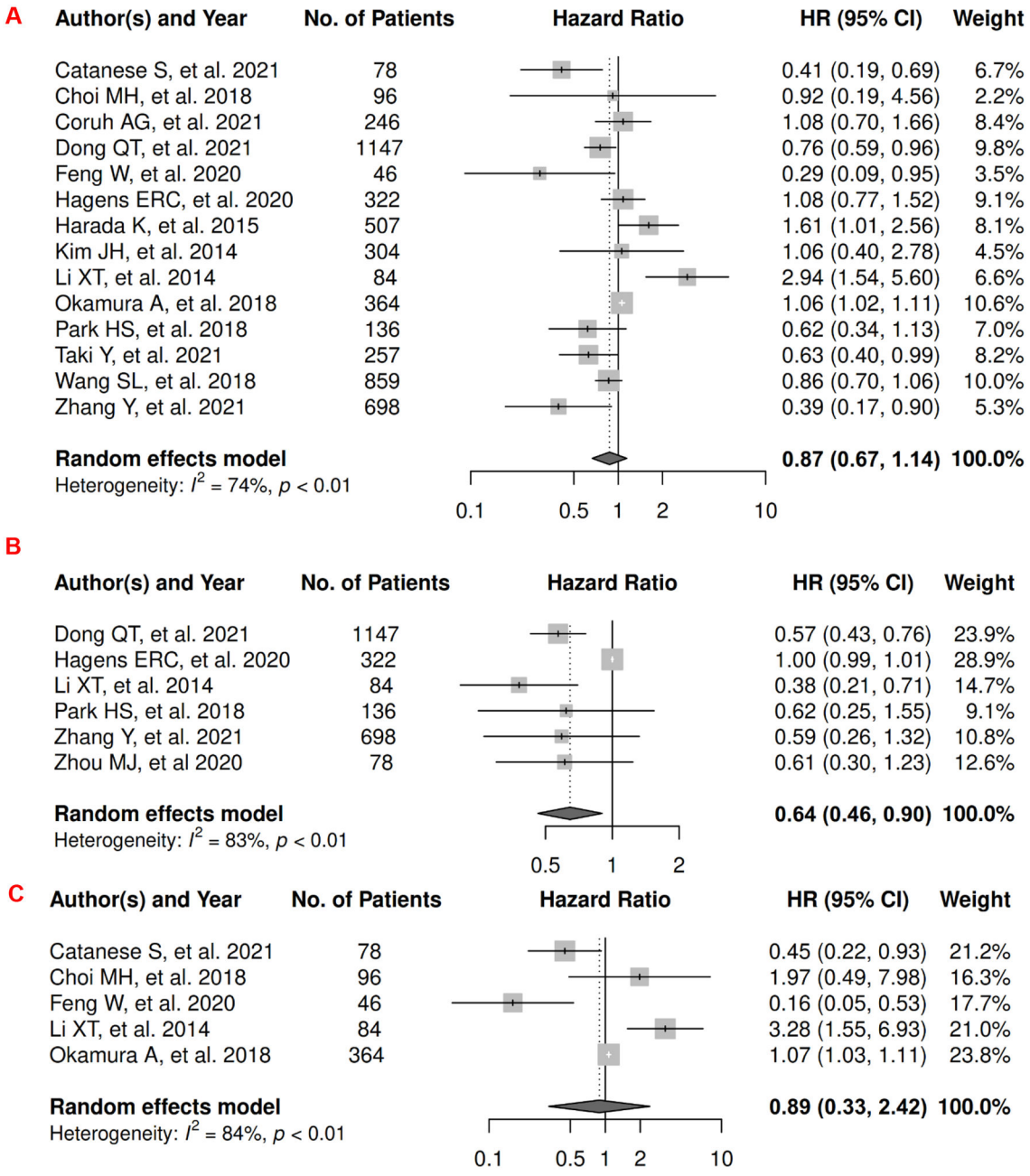


Figure 4.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Gastroesophageal Cancer

4A: visceral adiposity and overall survival.

4B: subcutaneous adiposity and overall survival.

4C: visceral adiposity and progression-free survival.

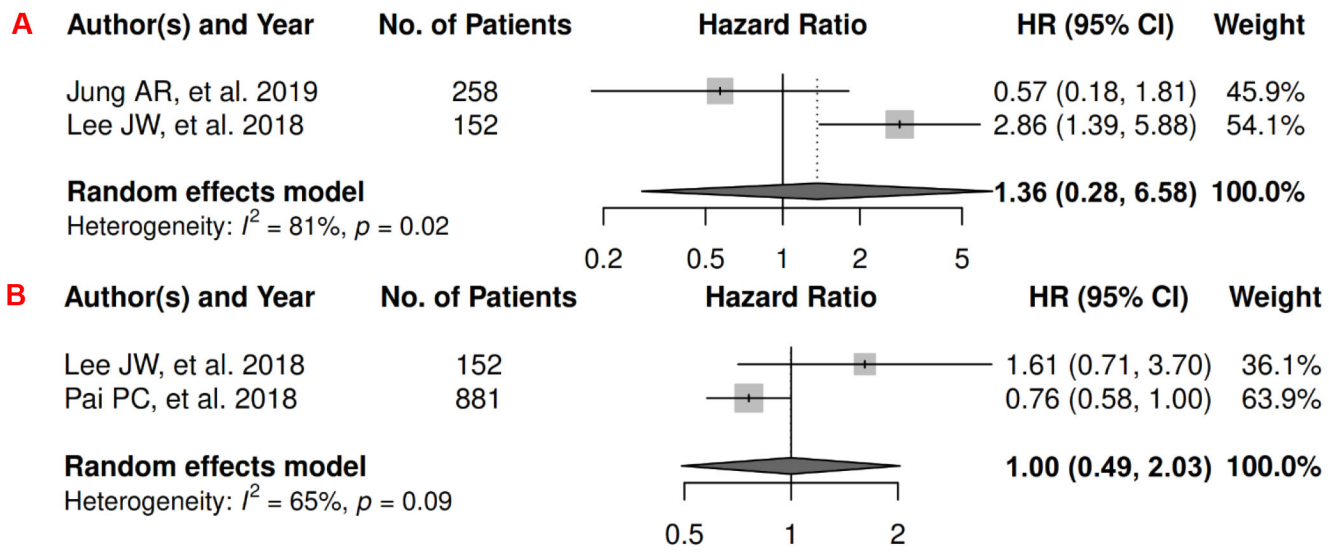


Figure 5.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Head and Neck Cancer

5A: visceral adiposity and progression-free survival.

5B: subcutaneous adiposity and progression-free survival.

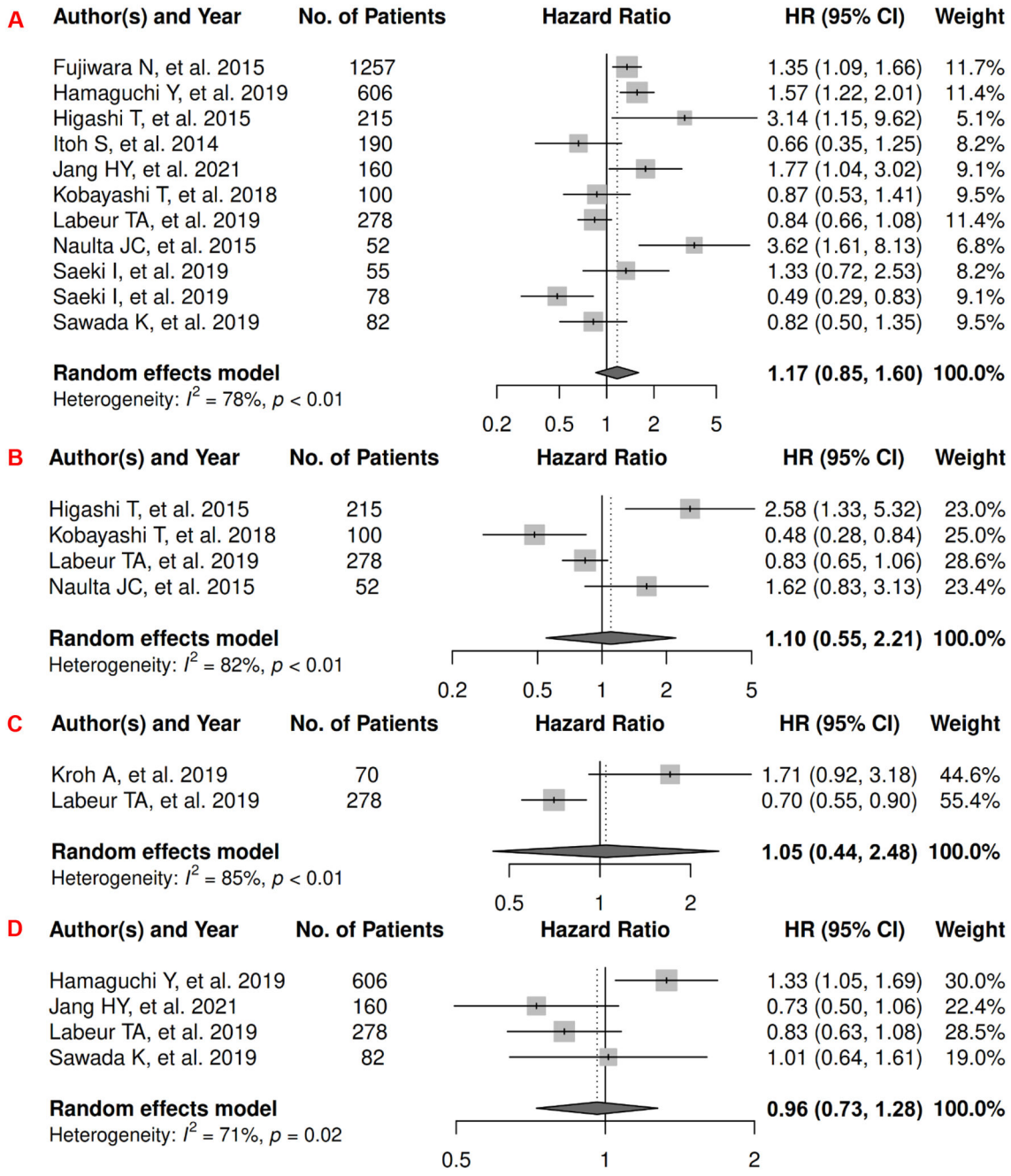


Figure 6.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Hepatocellular Carcinoma

6A: visceral adiposity and overall survival.

6B: subcutaneous adiposity and overall survival.

6C: total adiposity and overall survival.

6D: visceral adiposity and progression-free survival.

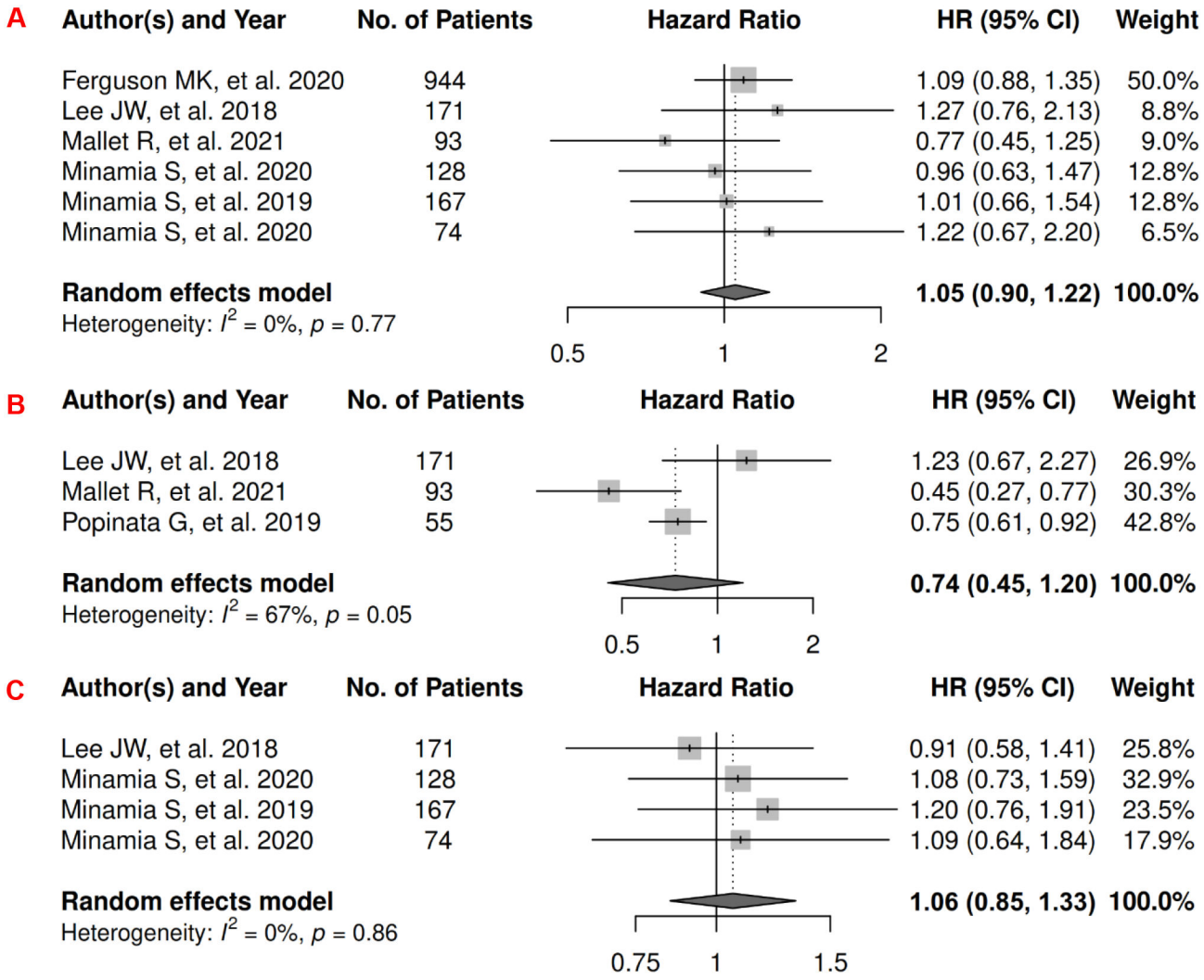


Figure 7.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Lung Cancer

7A: visceral adiposity and overall survival.

7B: subcutaneous adiposity and overall survival.

7C: visceral adiposity and progression-free survival.

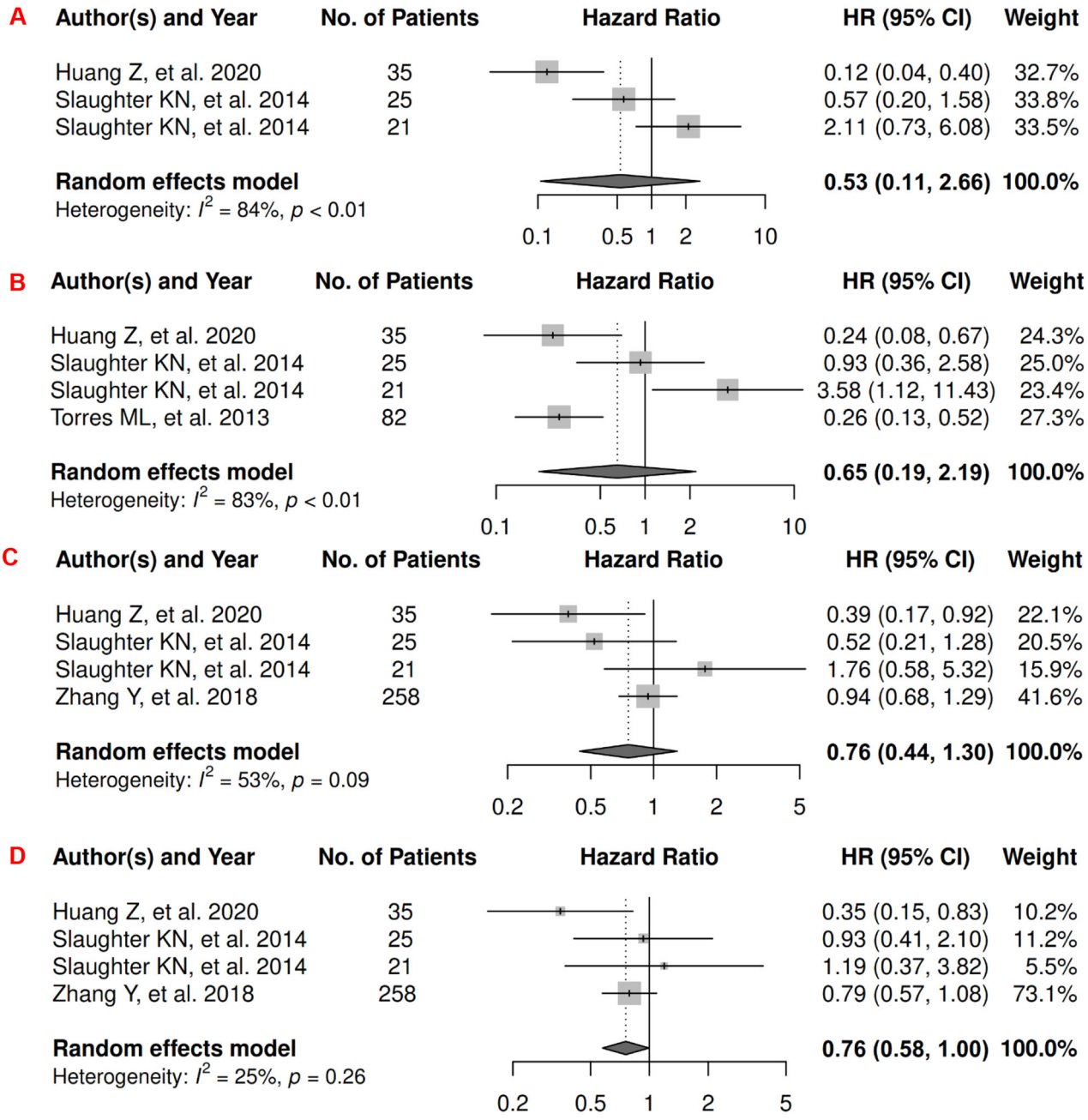


Figure 8.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Ovarian Cancer

8A: visceral adiposity and overall survival.

8B: subcutaneous adiposity and overall survival.

8C: visceral adiposity and progression-free survival.

8D: subcutaneous adiposity and progression-free survival.

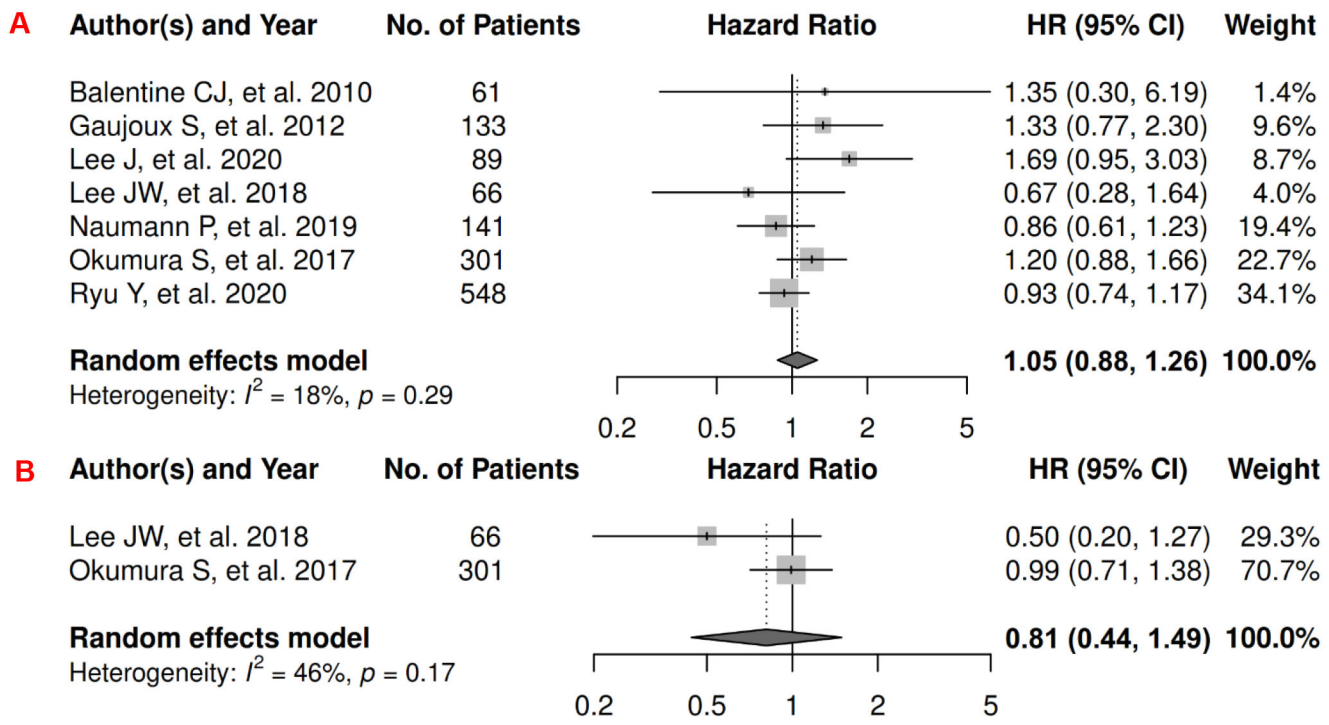
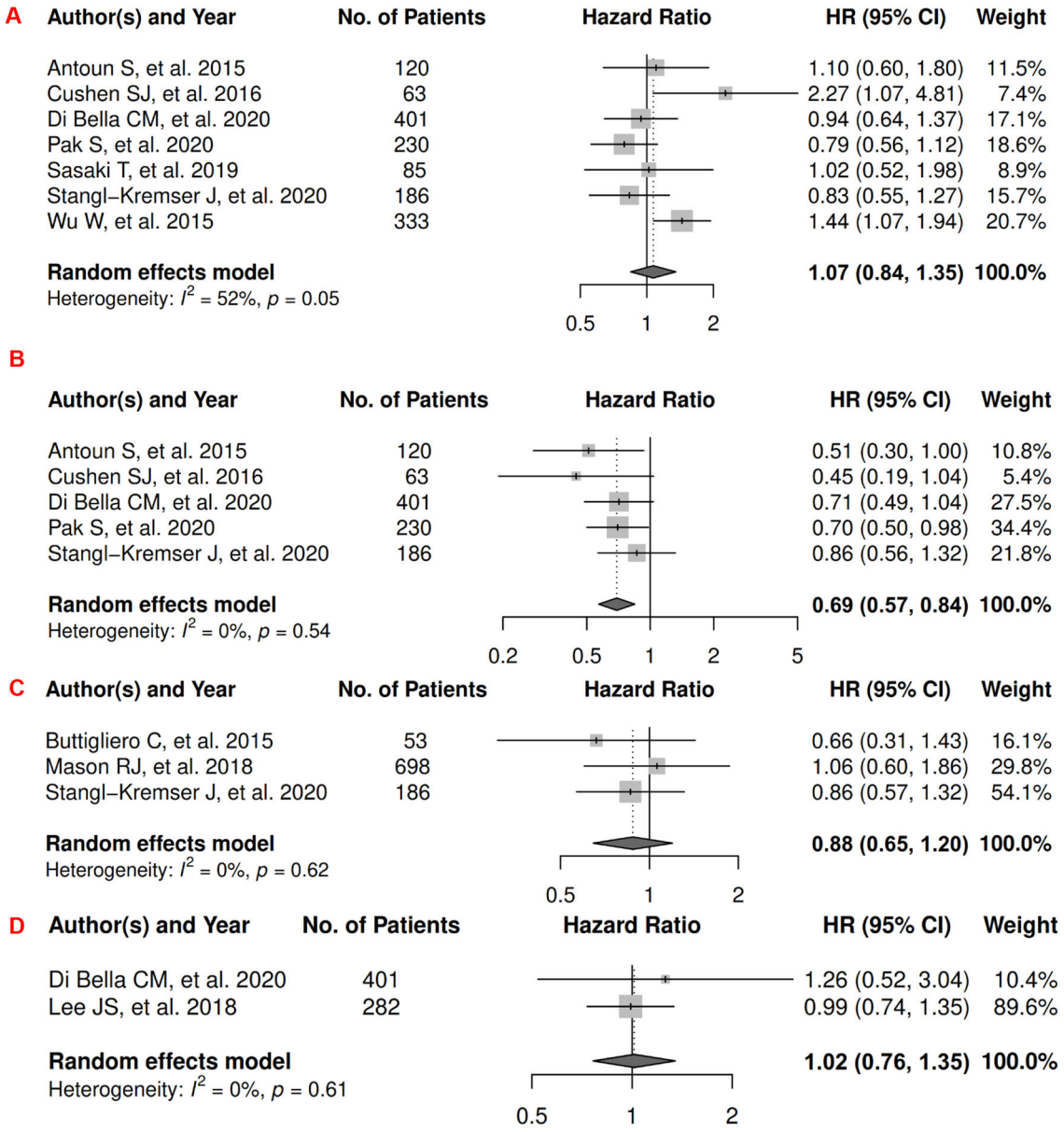


Figure 9.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Pancreatic Cancer

9A: visceral adiposity and overall survival.

9B: subcutaneous adiposity and overall survival.



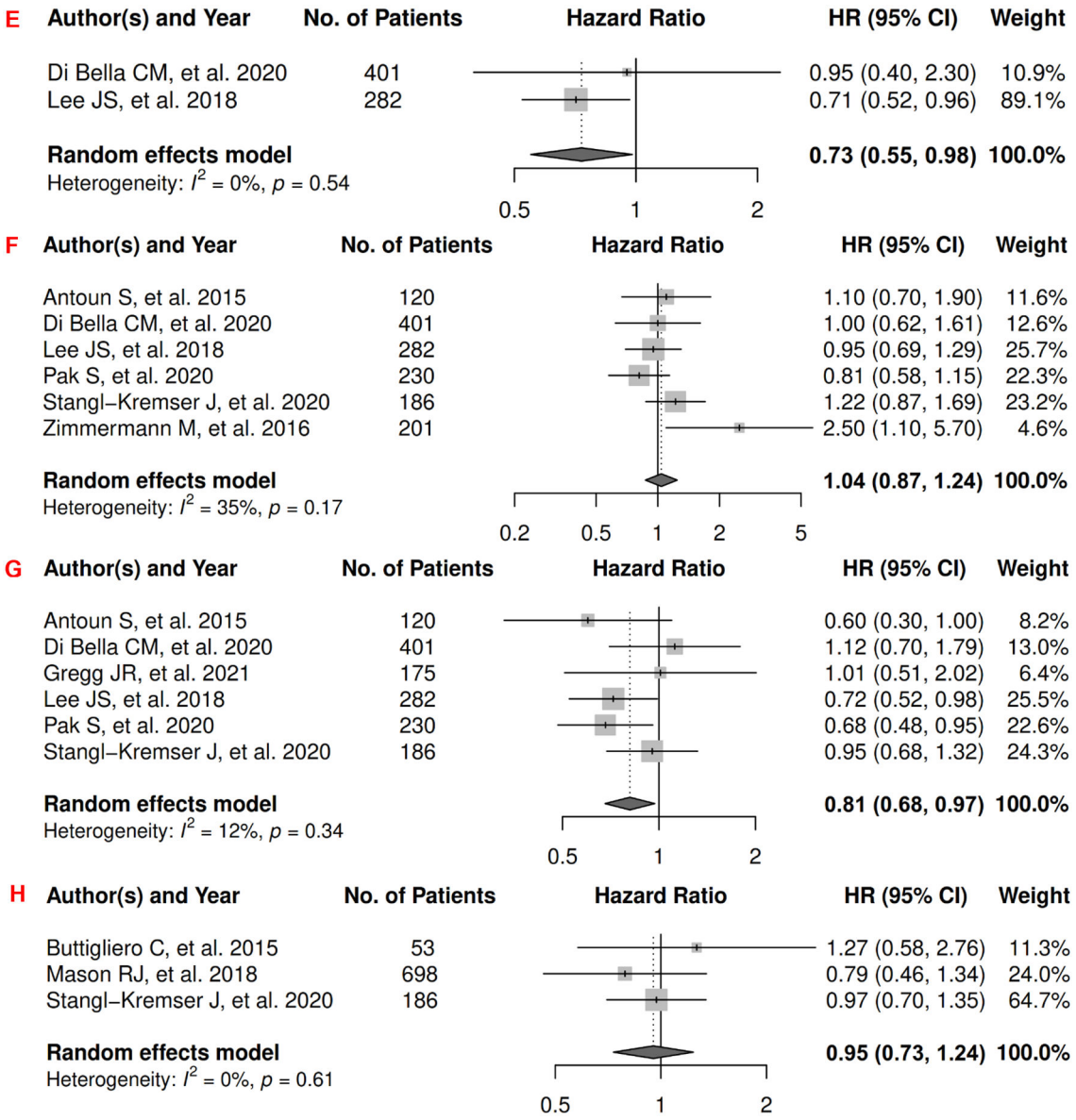


Figure 10.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Prostate Cancer

10A: visceral adiposity and overall survival.

10B: subcutaneous adiposity and overall survival.

10C: total adiposity and overall survival.

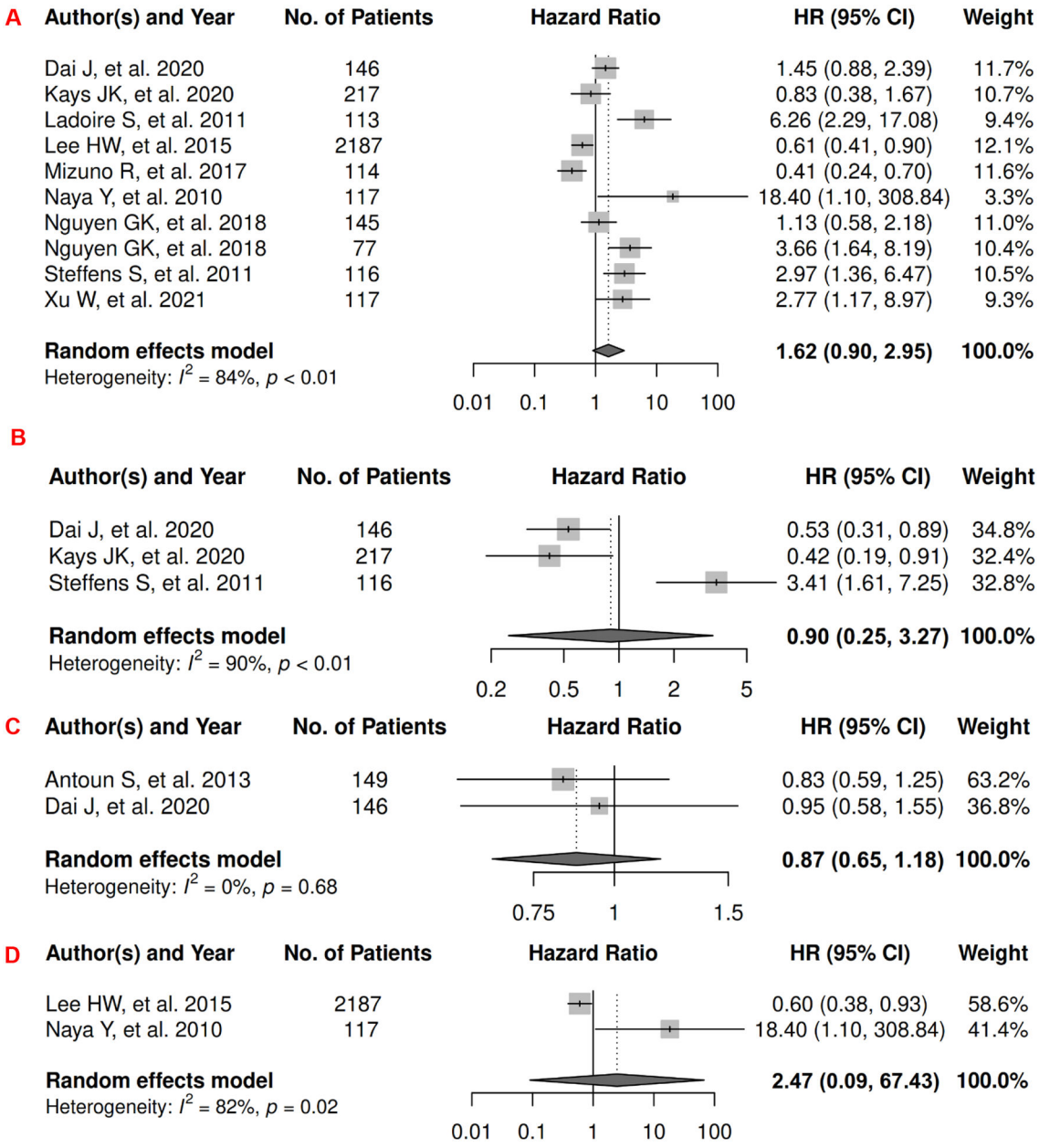
10D: visceral adiposity and cancer-specific survival.

10E: subcutaneous adiposity and cancer-specific survival.

10F: visceral adiposity and progression-free survival.

10G: subcutaneous adiposity and progression-free survival.

10H: total adiposity and progression-free survival.



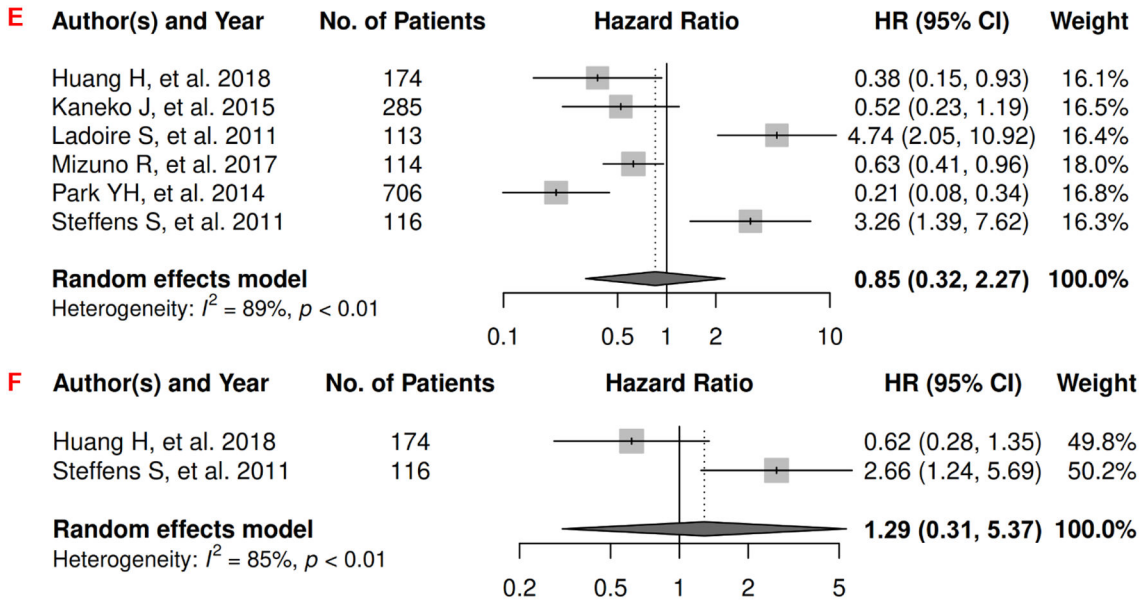


Figure 11.

Forest Plots of Assessing the Associations between Imaging-Measured Adiposity and Survival among Renal Cancer

11A: visceral adiposity and overall survival.

11B: subcutaneous adiposity and overall survival.

11C: total adiposity and overall survival.

11D: visceral adiposity and cancer-specific survival.

11E: visceral adiposity and progression-free survival.

11F: subcutaneous adiposity and progression-free survival.

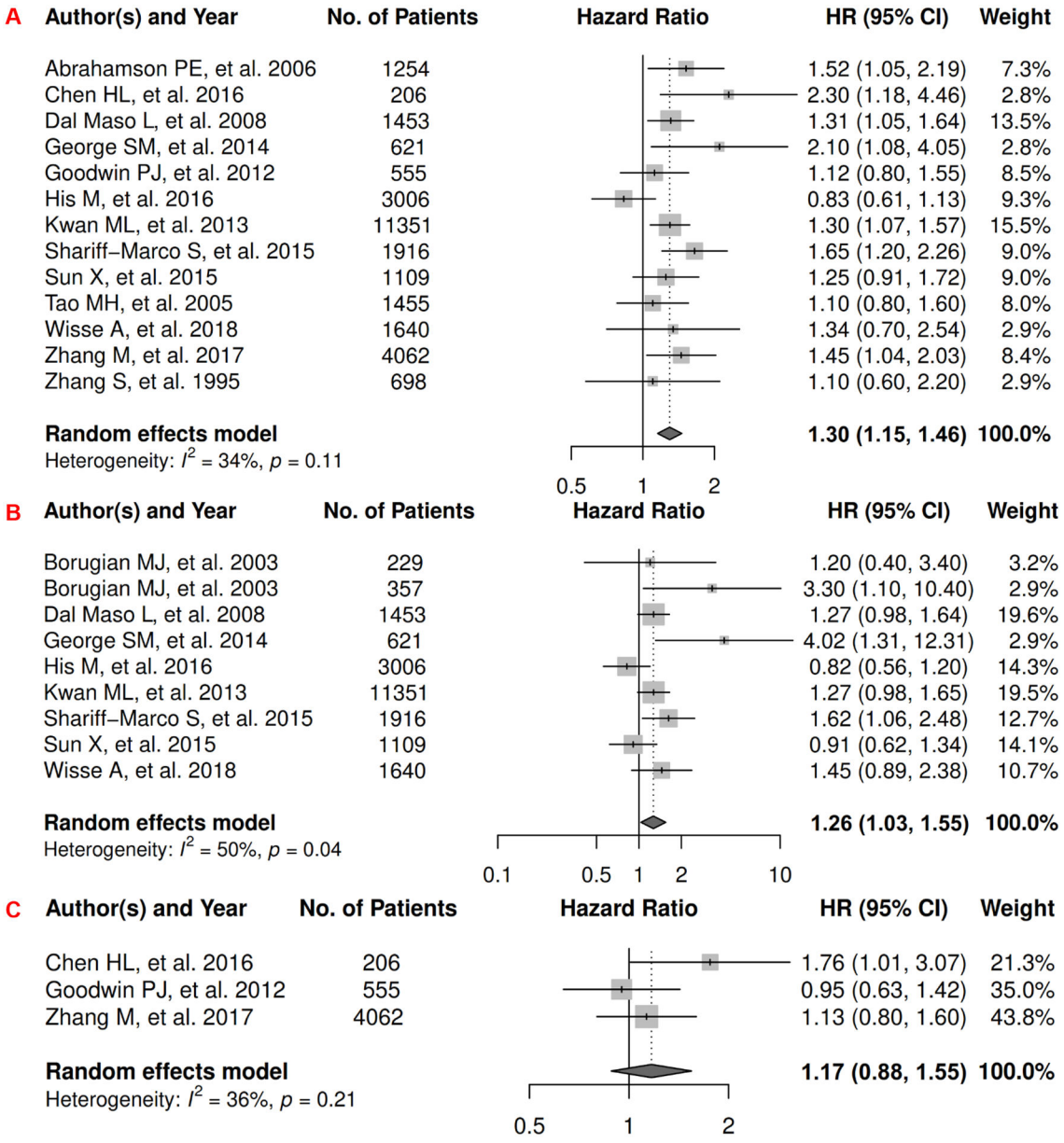


Figure 12.

Forest Plots of Assessing the Associations between Anthropometry-Measured Visceral Adiposity and Survival among Breast Cancer

12A: visceral adiposity and overall survival.

12B: visceral adiposity and cancer-specific survival.

12C: visceral adiposity and progression-free survival.

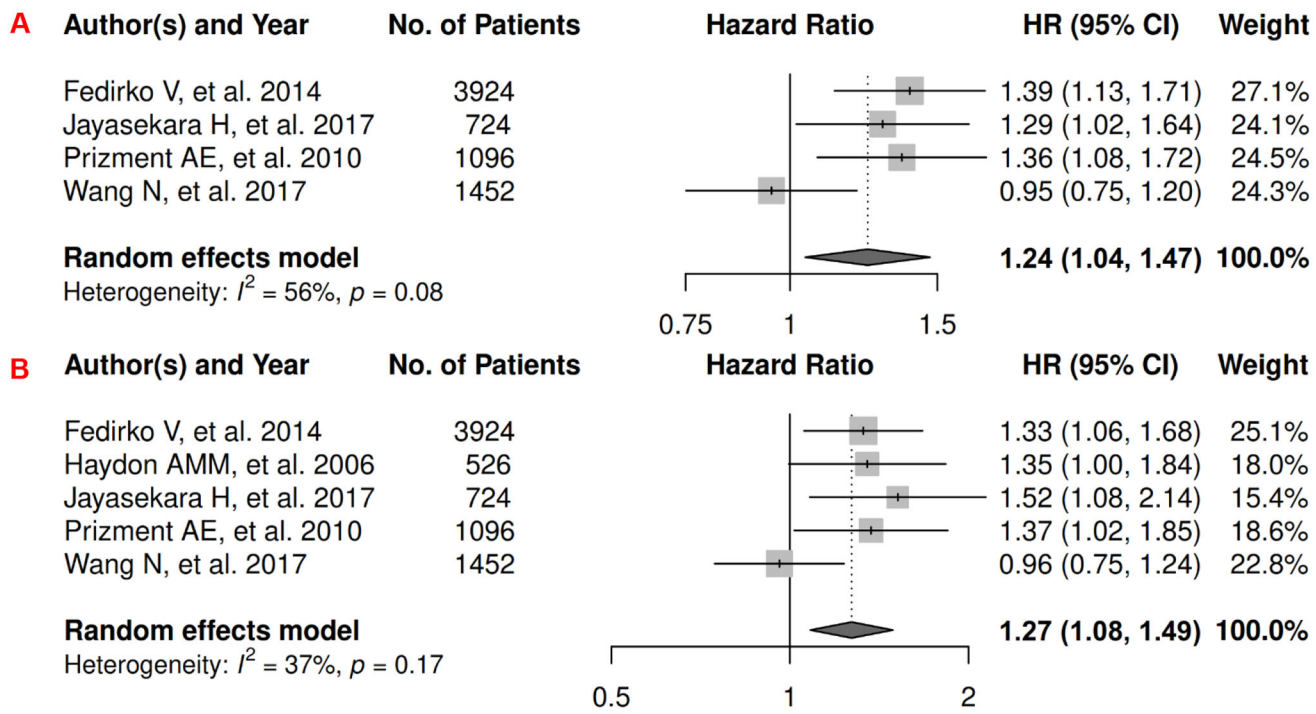


Figure 13.

Forest Plots of Assessing the Associations between Anthropometry-Measured Visceral Adiposity and Survival among Colorectal Cancer

13A: visceral adiposity and overall survival.

13B: visceral adiposity and cancer-specific survival.

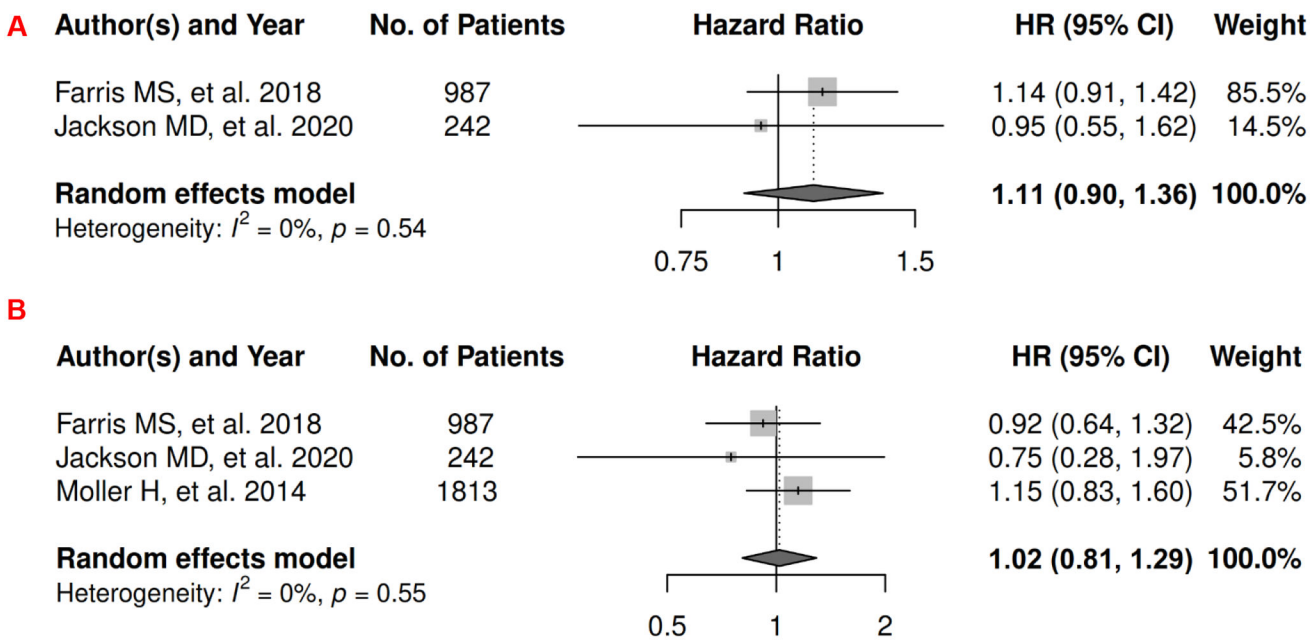


Figure 14.

Forest Plots of Assessing the Associations between Anthropometry-Measured Visceral Adiposity and Survival among Prostate Cancer

14A: visceral adiposity and overall survival.

14B: visceral adiposity and cancer-specific survival.