Role of body mass index and weight change in the risk of cancer: A systematic review and meta-analysis of 66 cohort studies

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Background This study was designed to evaluate the effects of body mass index (BMI) and weight change on the risk of developing cancer overall and cancer at different sites.

Methods We searched PubMed and other databases up to July 2023 using the keywords related to ‘risk’, ‘cancer’, ‘weight’, ‘overweight’, and ‘obesity’. We identified eligible studies, and the inclusion criteria encompassed cohort studies in English that focused on cancer diagnosis and included BMI or weight change as an exposure factor. Multiple authors performed data extraction and quality assessment, and statistical analyses were carried out using RevMan and R software. We used random- or fixed-effects models to calculate the pooled relative risk (RR) or hazard ratio along with 95% confidence intervals (CIs). We used the Newcastle-Ottawa Scale to assess study quality.

Results Analysis included 66 cohort studies. Compared to underweight or normal weight, overweight or obesity was associated with an increased risk of endometrial cancer, kidney cancer, and liver cancer but a decreased risk of prostate cancer and lung cancer. Being underweight was associated with an increased risk of gastric cancer and lung cancer but not that of postmenopausal breast cancer or female reproductive cancer. In addition, weight loss of more than five kg was protective against overall cancer risk.

Conclusions Overweight and obesity increase the risk of most cancers, and weight loss of >5 kg reduces overall cancer risk. These findings provide insights for cancer prevention and help to elucidate the mechanisms underlying cancer development.

Registration Reviewregistry1786.
Cancer is a major global health issue and one of the leading causes of disease-associated mortality [1]. Approximately 19–20 million people worldwide are diagnosed with cancer annually, while approximately 10 million people die from cancer [2]. The five-year survival rate for some malignancies, such as hepatocellular carcinoma, is less than 15%, whereas that for lung cancer is approximately 4–17% [3,4]. Various risk factors for cancer development have been identified, including dietary habits, alcohol consumption, smoking, metabolic syndrome, and diabetes [5–9]. In recent years, research has also indicated a potential link between overweight or obesity and weight change and the risk of certain cancers [10–15].

To date, the effect of overweight or obesity on cancer risk has not been thoroughly studied, and there are controversies among the findings of many studies. While some studies have shown that obesity increases the risk of gastric cancer [16,17], other studies have shown no significant association [18–22]. These discrepancies in findings may be due to inconsistencies in study type (cohort or case-control), differing definitions of obesity, ethnic differences, or sample size. Furthermore, existing meta-analyses have focused mainly on the effect of overweight or obesity on the risk of specific cancer [11,13,23–26]. Limited attention was given to the impact of underweight on cancer risk or the effect of different types of body mass index (BMI) on the risk of cancers in different systems.

Moreover, the current research on the relationship between weight change and cancer risk is also subject to controversy and limitations. For instance, one study concluded that weight gain increases the risk of breast cancer [27], yet another study suggested that the effect of weight gain on breast cancer varies depending on different oestrogen and progesterone receptor statuses [28]. Furthermore, most meta-analyses examining the impact of weight loss on cancer risk have focused on intentional weight loss rather than unintentional [12,29]. These factors may have affected their findings. Moreover, few meta-analyses assessing the effect of weight change on cancer risk have distinguished between different degrees of weight change.

Through a meta-analysis of cohort studies, we explored the risk of cancer in populations with different abnormal BMIs (including underweight, overweight, and obese individuals) compared to that in individuals with a normal BMI, as well as the risk of cancer in populations with different weight changes compared to those with unchanged weight. We specifically included cohort studies and set consistent cutoff values for the BMI categories in the meta-analyses to minimise bias. In addition, we examined the differences in cancer risk according to BMI category or weight change for patients with different cancers and attempted to explain these differences through cancer-related mechanisms. This may have important implications for cancer prevention and further exploration of the mechanisms underlying the relationship between BMI or weight change and cancer risk.

METHODS

We conducted this systematic review and meta-analysis according to the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) 2009 statement (Table S1 in the Online Supplementary Document) [30]. The protocol for this systematic review and meta-analysis was registered on the online registration platform Researchregistry. The study was assigned a unique identification number (Reviewregistry 1786), and can be found by searching the registry’s database of registered systematic reviews and meta-analyses using this number.

Search strategy

The literature search included the search of PubMed, Web of Science, Medline, Scopus, Cochrane, EconLit, Embase, Food Sciences and Technology Abstracts, and PsycINFO databases up to July 2023. The search string was (risk) AND (cancer OR carcinoma) AND (weight OR overweight OR obesity) (Table S2 in the Online Supplementary Document).

Eligibility criteria

Three authors checked all retrieved studies for eligibility; when there was a disagreement, the fourth and fifth authors joined the discussion to determine the final results. We included studies in the meta-analysis if they met the following criteria: involved a cohort study; involved a cancer diagnosis; indicated a BMI or weight change; had a reported headcount ratio or unadjusted relative risk (RR) or hazard ratio (HR); and were published in English. We excluded studies if the full text was not available, relevant data were not reported or were unavailable, or the study was a duplicate.
Data collection process
Two authors extracted the data. The team members independently checked and then cross-checked the data to achieve consistent results. The following variables were recorded for each study: first author's name, year of publication, sample size, sex and age of the baseline population, BMI classification criteria or mode of weight intervention and degree of weight change, raw headcount ratio data or unadjusted RRs or HRs with their 95% confidence intervals (CIs).

Exposure definition
For the classification of BMI, we used the weight classification prescribed by the World Health Organization (WHO). BMI = weight (kg)/height (m^2); underweight (BMI<18.5), normal weight (BMI = 18.5–25), overweight (BMI = 25–30), and obesity (BMI≥30) [31]. We also defined two categories – underweight or normal (BMI<25) and overweight or obesity (BMI≥25).

For the classification of weight change, we grouped the data on weight gain and weight loss provided by the included studies into groups: weight gain >2 kg, weight gain >5 kg, weight gain >10 kg, weight gain >20 kg, weight loss >2 kg and weight loss >5 kg. No weight change was defined as weight gain or weight loss not greater than the threshold value set for each subgroup.

Statistical analysis
We determined the heterogeneity between studies by the $\chi^2$ test and the $I^2$ index. Both random- and fixed-effects models were used to estimate the combined effects. When substantial heterogeneity was observed ($I^2>50\%$), we chose the random-effects model to effectively address the heterogeneity in the meta-analysis. This model accommodates varying true effect sizes between studies, incorporating this variability into the analysis. Conversely, when heterogeneity was minimal ($I^2\leq50\%$), we utilised the fixed-effects model, assuming a single true effect size across all studies. We assessed the potential publication bias by funnel plots and Egger's test [32]. A result with a two-tailed $P$-value ≤0.05 was considered statistically significant. All the statistical analyses and visualisations were completed with the software RevMan, version 5.3, (Nordic Cochrane Centre, Copenhagen, Denmark), R, version 4.1.3 (R Core Team, Vienna, Austria), and R packages ‘grid’ (version 4.2.2), ‘forestploter’ (version 0.2.3), ‘pheatmap’ (version 1.0.12), and ‘meta’ (version 6.0.0).

Study quality score
The quality of the included studies was assessed by the Newcastle-Ottawa Scale (NOS), a validated tool for assessing the quality of non-randomised trials [33]. In the NOS, each study is awarded a maximum score of nine points, and the total score can be divided into three categories – low risk of bias/high quality (0–3 points), medium risk of bias/moderate quality (4–6 points) and high risk of bias/low quality (7–9 points). Team members independently performed the assessment and resolved discrepancies by discussion.

RESULTS
Study selection
The flowchart of our retrieved and selected studies is presented in Figure 1. We obtained 10,014 records during our search, 4,741 records remained after removing duplicates and downloading the full text for filtering. After reviewing the titles and abstracts, 3,821 records were excluded. The remaining 920 articles were independently assessed for eligibility by three researchers. Of the 920 articles, 698 were excluded for the following reasons: full text was unavailable (n = 7), non-English articles (n = 6), non-cohort studies (n = 156), articles not examining the relationship between BMI or weight change and cancer risk (n = 342), relevant data not reported or unavailable (n = 166), and duplicate data sets (n = 21). After the full-text screening of the remaining 222 articles, 129 were excluded for not meeting the predefined requirements for data, and 27 articles were excluded for not meeting the predefined BMI classification thresholds. Finally, we included 66 studies – 56 for the analysis of BMI and cancer risk and 11 for the analysis of weight change and cancer risk [17,19,34–97].

Study characteristics
The studies involved approximately 24 million participants and were published between 2004 and 2023. The populations in the studies were predominantly enrolled at ages 40–70 years or older than 18 years, with
outcome events including the occurrence of common cancers (such as gastric, colorectal, liver, thyroid, endometrial and breast cancers) and rare cancers (such as diffuse large B-cell lymphoma) (Table S3 in the Online Supplementary Document). The proportion of high-quality studies (score ≥7) included in this meta-analysis was approximately 95% (63/66), according to the NOS (Figure S1 in the Online Supplementary Document).

Different BMIs and risk of overall cancer

We analysed the risk of overall cancer estimated by different BMI comparisons. Overweight or obesity was associated with an increased risk of overall cancer compared to underweight or normal weight (RR = 1.16; 95% CI = 1.09–1.24, P < 0.0001). Furthermore, compared to normal weight, overweight, obesity and overweight or obesity were associated with a greater risk of overall cancer (overweight RR = 1.13; 95% CI = 1.05–1.22, P = 0.001; obesity RR = 1.24; 95% CI = 1.11–1.39, P = 0.0002; overweight or obesity RR = 1.17; 95% CI = 1.07–
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1.27, P=0.0003) (Figure 2). However, there was no significant correlation between underweight and the risk of overall cancer. When the HRs from three studies were combined, no significant associations were revealed between overweight or obesity and overall cancer risk compared to underweight or normal weight (Figure S2 in the Online Supplementary Document).

Different BMIs and risks of different cancer sites

We analysed the risk of cancer at different sites with different BMIs. Compared to underweight or normal weight, overweight or obesity was associated with an increased risk of endometrial cancer (RR = 1.76; 95% CI = 1.35–2.30, P< 0.0001), kidney cancer (RR= 1.50; 95% CI= 1.14–1.96, P= 0.004), liver cancer (RR= 1.52; 95% CI= 1.29–1.78, P< 0.0001), colorectal cancer (RR= 1.27; 95% CI= 1.17–1.38, P<0.0001) and postmenopausal breast cancer (RR= 1.13; 95% CI= 1.03–1.24, P= 0.01). Further, obesity was associated with a reduced risk of prostate cancer (RR= 0.95; 95% CI= 0.91–0.99, P= 0.02) and lung cancer (RR= 0.87; 95% CI= 0.75–0.99, P= 0.04), and had no significant association with the risk of oesophageal cancer, ovarian cancer, gastric cancer, premenopausal breast cancer, pancreatic cancer or thyroid cancer (Figure 3, Panel A).

Compared to a normal weight, underweight was associated with an increased risk of stomach cancer (RR = 2.12; 95% CI = 1.24–3.64, P= 0.006) and lung cancer (RR = 1.66; 95% CI = 1.12–2.44, P= 0.01), and a reduced risk of postmenopausal breast cancer (RR= 0.62; 95% CI= 0.47–0.83, P= 0.001) (Figure 3, Panel B). Further, overweight was associated with an increased risk of endometrial cancer (RR= 1.79; 95% CI= 1.47–2.18, P<0.0001), oesophageal cancer (RR= 1.44; 95% CI= 1.13–1.83, P=0.003), colorectal cancer (RR= 1.22; 95% CI= 1.10–1.36, P=0.0003) and postmenopausal breast cancer (RR= 1.11; 95% CI= 1.01–1.20, P= 0.02) (Figure 3, Panel C).

Compared to normal weight, obesity increased the risk of endometrial cancer (RR = 3.80; 95% CI = 3.14–4.61, P<0.0001), oesophageal cancer (RR= 1.61; 95% CI= 1.07–2.43, P= 0.02), colorectal cancer (RR= 1.46; 95% CI= 1.24–1.73, P<0.0001), gastric cancer (RR= 1.43; 95% CI= 1.20–1.70, P<0.0001), and postmenopausal breast cancer (RR= 1.19; 95% CI= 1.00–1.41, P= 0.05). Compared to normal weight, overweight or obesity increased the risk of endometrial cancer (RR= 2.40; 95% CI= 2.04–2.82, P<0.0001), oesophageal cancer (RR= 1.47; 95% CI= 1.03–2.09, P=0.03), gastric cancer (RR= 1.38; 95% CI= 1.05–1.82, P= 0.02), colorectal cancer (RR= 1.27; 95% CI= 1.13–1.43, P<0.0001), and postmenopausal breast cancer (RR= 1.15; 95% CI= 1.01–1.30, P= 0.03) (Figure 3, Panels D and E).

When we analysed cancer of the digestive system and female reproductive system separately, we found an increased risk of digestive system cancer in those with overweight (RR= 1.22; 95% CI= 1.13–1.31, P<0.0001), obesity (RR= 1.39; 95% CI= 1.24–1.55, P<0.0001), and overweight or obesity (RR= 1.27; 95% CI= 1.17–1.37, P<0.0001) compared to normal weight. Notably, the risk of cancer in the female reproductive system cancer was reduced in individuals with underweight (RR= 0.70; 95% CI= 0.55–0.88, P= 0.002) and increased in individuals with overweight (RR= 1.12; 95% CI= 1.00–1.25, P= 0.05), obesity (RR= 1.29; 95% CI= 1.03–1.62, P= 0.03), and obesity or overweight (RR= 1.18; 95% CI= 1.02–1.36, P= 0.02) (Figure S3 in the Online Supplementary Document).

When we further stratified the analysis by sex, we found that underweight, overweight, obesity, and overweight or obesity were associated with an increased risk of colorectal cancer in both men and women, with no significant differences observed between the two groups (Figure S4 in the Online Supplementary Document).

Weight change and risks of overall cancer and different cancer sites

In the weight gain vs non-weight change subgroup, individuals who gained >5 kg (RR= 1.23; 95% CI= 1.06–1.42, P= 0.005), >10 kg (RR= 1.18; 95% CI= 1.05–1.33, P= 0.006), or >20 kg (RR= 1.28; 95% CI= 1.21–1.36, P<0.0001) had a greater risk of developing overall cancer than those who did not experience weight change;
Figure 3. Summary risk estimated by different cancer sites in different BMI comparisons. **Panel A.** Overweight or obesity vs underweight or normal weight. **Panel B.** Underweight vs normal weight. **Panel C.** Overweight vs normal weight. **Panel D.** Obesity vs normal weight. **Panel E.** Overweight or obesity vs normal weight. RR – relative risk, CI – confidence interval.
while no significant difference in cancer risk was observed between those who gained >2 kg and those who gained any degree of weight (total) (Figure 4, Panel A). Regarding cancer type, weight gain was shown to be associated with an increased risk of breast cancer (RR = 1.16; 95% CI = 1.02–1.31, P = 0.02) but had no significant effect on the risk of colon or reproductive cancer.

According to the weight loss vs non-weight change analysis, participants who lost >5 kg had a lower risk of overall cancer than those who did not experience weight change (RR = 0.78; 95% CI = 0.66–0.91, P = 0.002), while there was no significant change in the risk of overall cancer in those who lost any degree of weight. Those who lost >2 kg had an increased risk of overall cancer (RR = 1.11; 95% CI = 1.01–1.21, P = 0.03).

When examining cancer type, weight loss was associated with a decreased risk of breast cancer (RR = 0.90; 95% CI = 0.83–0.97, P = 0.006) and reproductive cancer (RR = 0.92; 95% CI = 0.86–0.99, P = 0.04) but had no significant effect on colon cancer (RR = 1.05; 95% CI = 0.88–1.26, P = 0.57) (Figure 4, Panel B).

Publication bias

In this meta-analysis, we did not find publication bias in the studies on the association between BMI or weight change and the risk of cancer. The funnel plot showed slight symmetry (Figure S5 in the Online Supplementary Document), and Egger’s test showed that the following subgroup comparisons all had P-values >0.05, indicating no significant publication bias (overweight or obesity vs underweight or normal weight P = 0.47; underweight vs normal weight P = 0.56; overweight vs normal weight P = 0.77; obesity vs normal weight P = 0.14; overweight or obesity vs normal weight P = 0.47; weight gain vs non-weight change P = 0.85) and weight loss vs non-weight change P = 0.83).

DISCUSSION

Our meta-analysis was designed to assess the relationship between BMI or weight change and cancer risk. Our findings revealed that compared to underweight or normal weight, overweight or obesity was significantly associated with an increased risk of endometrial, kidney, liver, colorectal and postmenopausal breast cancer. The association was stronger for an increased risk of endometrial cancer and weaker for liver and colorectal cancers. For prostate cancer and lung cancer patients, compared to underweight or normal weight, overweight or obesity was a protective factor. In addition, a weight gain greater than 5 kg was associated with an increased risk of overall cancer, while a weight loss greater than 5 kg was associated with a decreased risk of overall cancer.
Many studies have described the mechanisms that link overweight or obesity to cancer risk. In the vast majority of tumours, inflammation and oestrogen are the shared mechanisms that increase the risk of cancer due to being overweight and obese. However, the specific role of these mechanisms may vary depending on the tumour type (Figure 5). In individuals who are overweight or obese, adipocytes increase in size and release inflammatory factors, leading to chronic low-grade inflammation in the body, which then triggers tumour development and increases cancer risk through a variety of complex mechanisms [98]. In endometrial and colorectal cancers, an increase in inflammatory cytokines can promote the development of tumours by producing damaging reactive oxygen species, causing cell mutation and proliferation, and promoting angiogenesis [98–103]. In the liver, the release of large amounts of inflammatory factors can contribute to the development of non-alcoholic fatty liver disease directly or by increasing the levels of free fatty acids, lipid accumulation, hepatic steatosis and necrosis and causing non-alcoholic fatty liver disease, consequently increasing the risk of liver cancer [98,104]. An obesity-induced proinflammatory environment can cause an imbalance between oestrogen and progesterone, increasing susceptibility to neoplastic processes [105]. Moreover, in adipose tissue, androgens can be converted to oestrogens (estrone and oestradiol) by aromatase enzymes, and the levels of these enzymes increase with increasing BMI [106,107], leading to elevated oestrogen levels in people with overweight or obesity. In endometrial tissue, oestrogen can promote cancer by stimulating cell growth and division and inhibiting apoptosis [108]. Conversely, in the colorectum, oestrogen in women with obesity has been found to reduce the risk of cancer [109]. Increased oestrogen promotes the expression of oestrogen receptor-β, which can promote apoptosis and inhibit carcinogenesis, partially offsetting the increased risk of colorectal cancer caused by obesity [109,110]. Therefore, these differential effects of inflammation and oestrogen in different parts of the body may contribute to the varying associations among the risks of endometrial, liver and colorectal cancer due to being overweight or obese, thus may explain our findings.

Figure 5. Potential mechanisms linking inflammation and oestrogen to obesity and liver, colorectal and endometrial cancers. Created in Biorender (https://biorender.com/). ER – oestrogen receptor, FFAs – free fatty acids, NAFLD – non-alcoholic fatty liver disease, ROS – reactive oxygen species.
The effects of overweight and obesity on cancer risk and their underlying mechanisms differ between tumour types. Our analysis also revealed that, in the case of prostate cancer, being overweight or obese might reduce cancer risk rather than increase it, as observed in other tumour types. This effect may be attributed to the central role of testosterone in the prostate gland. Testosterone is a major androgen in men and is involved in normal prostate growth, and its levels are associated with prostate development and function [111,112]. High testosterone levels can stimulate the proliferation of the prostate tissue [113]. Studies suggest that an increased level of testosterone may be associated with an increased risk of cancer in the prostate [114–116]. In contrast, in individuals with obesity, testosterone levels are typically reduced, which may be detrimental to the growth of tumour cells and tissues in the prostate and thus may lead to a reduced risk of prostate cancer [116–120].

We also found that weight gain over 5 kg was a risk factor for overall cancer, while weight loss over 5 kg was a protective factor. Regarding breast cancer, any degree of weight gain was associated with an increased risk, while weight loss was associated with a decreased risk. The effect of weight change on breast cancer risk may be related to mechanisms such as metabolic dysfunction and oestriadiol levels in the body. The metabolic effects of weight gain on breast tissue during adolescence, pregnancy or menopause may increase a woman's risk of breast cancer [121]. Weight loss has been shown to increase the concentration of sex hormone-binding globulin, a plasma-binding protein with a high specific affinity for oestriadiol. Elevated sex hormone-binding globulin levels generally reduce the proportion of oestriadiol in the body, thereby reducing the stimulation of breast cell proliferation by oestriadiol and thus reducing the risk of cancer [122,123]. In addition, studies have shown that the levels of several cancer biomarkers, including C-reactive protein, tumour necrosis factor-α, interleukin-6, insulin-like growth factor and insulin-like growth factor binding protein, fall rapidly after weight loss [124], suggesting that weight loss can help reduce the risk of cancer.

Our findings point to new directions for weight management in cancer prevention. On the one hand, weight optimisation needs to be tailored for individuals with different BMIs and different cancer risk groups to contribute to cancer risk reduction. For example, in postmenopausal breast cancer patients, underweight was significantly associated with a lower risk, whereas overweight and obesity were associated with a greater risk of occurrence. In contrast, underweight is a risk factor for gastric cancer development. The substantial variability between different BMIs and the risk of cancer development reveals the variability in weight management strategies. On the other hand, since both BMI and weight change were significantly associated with the risk of cancer occurrence, BMI and weight change may contribute to the construction of better cancer risk prediction models.

This study has numerous limitations. First, some results were not significant in the meta-analysis of cancer types, possibly due to the limited size of the data set. Second, the baseline population characteristics were not restricted in this study, and inconsistent baseline population characteristics may also affect the results. Third, due to data limitations, this study did not allow for a comprehensive and systematic analysis of subgroups such as those defined by age and ethnicity. Heterogeneity is often unavoidable in meta-analyses, especially when dealing with diverse baseline population characteristics, such as varying cancer stages among the included patients. Given the difficulty in conducting detailed subgroup analyses due to limited sample sizes and heterogeneous baseline populations, we faced a trade-off between sample size constraints and the potential for extensive subgroup analysis. As such, we endeavoured to investigate scientific outcomes in the presence of certain heterogeneity. Furthermore, to address the issue of heterogeneity and further reduce its impact, obtaining access to raw data for additional analyses would be beneficial. By merging similar patient groups across different studies based on baseline characteristics (e.g. gender, age, ethnicity), we could conduct an in-depth examination of the impact of obesity on cancer risk. However, acquiring such raw data poses considerable challenges and may not be readily achievable. Fourth, this study avoided the inclusion of intentional weight loss-related cohort studies; instead, we opted for studies in which weight change data were obtained via self-reports or third-party institutional measurements. However, there was no way to determine whether the data obtained from self-reports or third-party institutional measures were derived from intentional or unintentional weight loss. Finally, due to the insufficient number of included studies, there was no breakdown of tumour type in the subgroups with a weight gain >5 kg, 10 kg, or 20 kg and weight loss >2 kg or 5 kg, as well as no breakdown of the degree of weight change in the subgroups of breast cancer and reproductive system cancer risk. All of these factors may have contributed to the high heterogeneity of the meta-analysis.

CONCLUSIONS

In conclusion, this meta-analysis showed that BMI and weight change are associated with cancer risk, with overweight and obesity likely associated with an increased risk of most cancers and weight loss >5 kg likely
protective against overall cancer risk. Optimising BMI or weight may be an effective measure for reducing cancer risk. These findings could lead to the development of personalised and more detailed weight management strategies in clinical practice, as well as the optimisation of cancer risk prediction models. More data are needed to investigate the effect of underweight on cancer risk.

Data availability: All data generated or analysed during this study are included in this published article and its supplementary information files.

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Authorship contributions: Peng Luo, Quan Cheng and Jian Zhang conceived, designed and supervised the study. Xiaoye Shi, Gengwen Deng, Haiteng Wen and Anqi Lin collected data and performed the data analysis. Xiaoye Shi and Gengwen Deng drafted the manuscript and drew the figures. Anqi Lin, Xiaoye Shi, Gengwen Deng, Haiteng Wen, Haitao Wang, Lingxuan Zhu, Weiming Mou, Zaoqiu Liu and Xiaohua Li revised the manuscript. All authors reviewed and edited the manuscript, contributed to the article, and approved the submitted version.

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Additional material
Online Supplementary Document

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REFERENCES