PERSPECTIVE

Perspective: Whole and Refined Grains and Health—Evidence Supporting "Make Half Your Grains Whole"

Julie Miller Jones,[1] **Carlos Guzmán García,**[2] **and Hans J Braun**[3]

[1] Department of Nutrition, St. Catherine University, St. Paul, MN, USA; [2] Department of Genetics, Advanced Technical College of Agricultural Engineering and Forestry, University of Córdoba, Córdoba, Spain; and [3] Global Wheat Program, Centro Internacional de Mejoramiento de Maiz y Trigo, El Batan, near Texcoco, Mexico

ABSTRACT

Research-based dietary guidelines suggest that consumers "make half their grains whole." Yet some advocate ingesting only whole-grain foods (WGFs) and avoiding all refined-grain foods (RGFs). Some even recommend avoiding all grain-based foods (GBFs). This article will provide arguments to counter negative deductions about GBFs and RGFs, especially staple ones, and to support dietary guidance recommending a balance of GBFs—achieved through the right mix, type, and quantity of WGFs and RGFs. Studies looking at early mortality, body weight, and glucose tolerance and diabetes will be used as examples to characterize the literature about GBFs. The following issues are highlighted: *1*) inconsistent findings between epidemiological and interventional studies and impacts of GBFs on health outcomes, and the underreporting of findings showing RGFs neither raise nor lower health risks; *2*) multiple confounding and potential interactions make adequate statistical adjustment difficult; *3*) nonuniform WGF definitions among studies make comparison of results challenging, especially because some WGFs may contain 49–74% refined grain (RG); *4*) binary categorization of GBFs creates bias because nearly all categories of WGFs are recommended, but nearly half the RGF categories are not; *5*) ingestion of >5 (30-g) servings RGFs/d and <1 serving WFGs/d creates dietary imbalance; *6*) pattern names (e.g., "white bread") may impugn RGFs, when names such as "unbalanced" or "few fruits and vegetables" may more fairly characterize the dietary imbalance; *7*) avoidance of all enriched RGs may not only impair status of folate and other B vitamins and certain minerals such as iron and zinc but also decrease acceptability of WGFs; *8*) extrapolation beyond median documented intakes in high-WGF consumers (∼48 g whole grain/d) in most cohorts is speculative; *9*) recommended dietary patterns such as the Mediterranean diet demonstrate that the right mix of WGFs and RGFs contributes to positive health outcomes. *Adv Nutr* 2020;11:492–506.

Keywords: whole grains, refined grains, indulgent grains, bias and confounding, dietary recommendations, chronic disease risk, grain enrichment and fortification, balanced dietary patterns (DASH and Mediterranean diets), "Make half your grains whole", carbohydrate and grain avoidance

Introduction: The Need for Whole and Refined Grains in the Diet

Grain-based foods (GBFs) supply much of the world's energy (E) and nutrient needs, providing 25–50% of E in Western diets and over half the world's calories (1, 2). Moreover, they are important sources of carbohydrates and dietary fiber (DF). Dietary guidance around the world recommends 40–65% of E as carbohydrates, with 65% regarded as high by some (1–3).

GBFs also are important sources of micronutrients and plant-based protein. The protein, although incomplete, provides amino acids that complement those found in other plant foods (e.g., nuts and legumes) to make complete proteins, in foods that are affordable, shelf-stable, portable, versatile, and popular. GBFs will play a key role as the world transitions to plant-based diets to meet future food supply needs (1, 2).

Health promotion bodies worldwide recognize GBFs' critical role in their food group–based recommendations. In pictorial food-based guidance such as USDA MyPlate or the Japanese Pagoda, GBFs are prominent in these culturally specific icons (1–10). The importance of GBFs, especially of whole-grain foods (WGFs), is being undermined by popular diets claiming these foods are unnecessary or unhealthful. Yet, diets that omit all GBFs will lack cereal fiber and may have inadequate intakes of micronutrients.

Dietary guidance since 2005 has underscored the importance of WGFs by specifically suggesting that "half of the

 Adv Nutr 2020;11:492–506; doi: https://doi.org/10.1093/advances/nmz114.

grains should be whole grains" (WGs) (4). The American Association of Cereal Chemists defined WGs as follows: "Whole grains shall consist of the intact, ground, cracked or flaked caryopsis, whose principal anatomical components—the starchy endosperm, germ and bran—are present in the same relative proportions as they exist in the intact caryopsis" (11). This definition was adopted by regulatory and health promotion bodies in order to encourage consumers to replace some refined-grain foods (RGFs) with WGFs (7–10, 12–14).

Refined grains (RGs) differ from WGs in that some or all of the outer bran layers are removed by milling, pearling, polishing, or de-germing. These processes reduce micronutrients, decrease DF by ≤75%, and lower some antinutritional components held in the bran (2). Refined, enriched grains (REGs) have micronutrients added to replace some of these losses. In some jurisdictions, RGs may be subject to mandatory or voluntary fortification of folate and other micronutrients.

Health impacts of WG ingestion have been the focus of epidemiological analyses, randomized controlled trials (RCTs), reviews, and meta-analyses for the last 20 y (15–101). Epidemiological studies and their meta-analyses consistently show those in the quintile ingesting the highest quantity of WGFs, compared with those in the quintile eating the lowest, are associated with lower risks of overall or cause-specific early mortality and chronic diseases (17–32). Specifically, WGFs are associated with reduced risks of obesity or overweight (33–62); abnormal glucose tolerance, prediabetes, and type 2 diabetes (T2D) (41, 50, 62–77); elevated inflammatory markers (41, 77–79), blood pressure (80, 81), or blood lipids; coronary and cardiovascular disease (32, 44, 50, 82–87); and certain cancers (88–94). Such consistency is not observed in RCTs (51–61, 71, 79, 95–101).

The health impacts associated with RGFs are often reported in the same studies looking at WGFs; however, the results are less widely promulgated. Whereas numerous reviews focus on WGFs, few focus on RGFs. Those that do show a null effect or associations with slightly lower health risks for many health outcomes (102, 103). Such findings support recommendations for "half the grains to be whole" (4–6).

Although some research shows increased risks associated with RGFs and metabolic syndrome, T2D, and biomarkers such as blood glucose or lipids (56–67, 75), the adverse impacts are not seen consistently. They are more likely in the following scenarios: *1*) total calories or number of grain servings exceed E needs; *2*) total carbohydrate intakes are >60% of E; *3*) RGFs are mostly nonrecommended, indulgent ones; and *4*) persons at risk of certain conditions may benefit from lower carbohydrate intakes (40–45% of E) (3).

USDA data covering the period 1970–2010 document increasing intakes of GBFs and percentage of E from carbohydrate (104). These increases correlate with increasing rates of obesity over the same time period and are used by some to posit that GBFs and carbohydrates are the cause of obesity and chronic disease. However, the correlations do not prove causation, and during that same time period available E from most food groups increased, a more likely cause of weight gain and associated chronic disease risks (104). In addition, the correlation was no longer relevant after 2010 because GBF intake decreased, but obesity continued to rise (105).

Negativity concerning and blaming carbohydrates and GBFs, especially RGFs, continue (106–114). Many deduce that positive impacts for ingestion of WGFs imply negative ones for RGFs and recommend limitation or elimination of RGFs by promoting Paleo, ketogenic, very-low-carbohydrate diets or advocating for ingestion of WGFs only (106–114). RGFs were denounced by one researcher as "the single most harmful influence in the American diet today" (107).

The popular press and public health professionals tout positive findings about WGFs but ignore or fail to fully report null findings on RGFs. Such pronouncements color perceptions of RGFs, as demonstrated in a 2018 survey (115): 80% of respondents believed WGFs were "healthy," compared with 40% for RGFs, and 15% named RGFs "unhealthy."

In-depth discussion of data for 3 health endpoints will serve as examples for 9 arguments to support the current dietary recommendations "to make half your grains whole" (4, 5, 6). Further, the documented benefits associated with ingestion of WGFs will be discussed together with a number of issues that inadvertently bias findings against RGFs.

WGFs are consistently associated with improved health outcomes in epidemiological studies, but not in RCTs; RGFs are not associated with risks in many of the same studies

Ingestion of 48 g WGs/d [∼2.7 one-ounce (30-g) WGF servings/d] is consistently associated with health benefits in prospective cohort and case-control studies and their meta-analyses. No such consistency is observed in results from RCTs and their meta-analyses, even when all WGFs are fed (15–101). RGFs, in many of the same epidemiological studies

Supported by the CRP-Wheat program of the CGIAR consortium (to JMJ). CGG gratefully acknowledges the European Social Fund and the Spanish Ministry of Science, Innovation and Universities for financial funding through the Ramon y Cajal Program (RYC-2017-21891).
Author disclosures: JMJ reports conflicts of interest relating to American Association of Cereal Chemists International, Bay State Milling, Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), General Mills, Inc., Grains Foods Foundation, and Healthy Grains Institute, Quaker Oats Company. CGG and HJB report conflicts of interest relating to CIMMYT and CGIAR.
Perspective articles allow authors to take a position on a topic of current major importance or controversy in the field of nutrition. As such, these articles could include statements based on author opinions or point of view. Opinions expressed in Perspective articles are those of the author and are not attributable to the funder(s) or the sponsor(s) or the publisher, Editor, or Editorial Board of *Advances in Nutrition*. Individuals with different positions on the topic of a Perspective are invited to submit their comments in the form of a Perspectives article or in a Letter to the Editor.
Address correspondence to JMJ (e-mail: jmjones@stkate.edu).
Abbreviations used: DASH, Dietary Approaches to Stop Hypertension; DF, dietary fiber; E, energy; GBF, grain-based food; HEI, Healthy Eating Index; iAUC, integrated AUC; IWHS, Iowa Women's Health Study; MEDiet, Mediterranean diet; RCT, randomized controlled trial; REG, refined, enriched grain; RG, refined grain; RGF, refined-grain food; RTEC, ready-to-eat cereal; SSB, sugar-sweetened beverage; SUN, Seguimiento Universidad de Navarra; T2D, type 2 diabetes; VAT, visceral adipose tissue; WC, waist circumference; WG, whole grain; WGF, whole-grain food; WHI, Women's Health Initiative; WMD, weighted mean difference.

finding benefits of WGFs, often show a null effect, or even a risk-lowering effect. Such findings may not be emphasized or communicated (116–119). Three health outcomes—risk of early mortality, risk of elevated body weight, and risk of elevated glycemia and T2D—will show how epidemiological findings on GBFs differ from those observed with RCTs and are indicative of a variability observed for most health outcomes.

Studies of WGs and RGs have multiple sources of confounding

Better health outcomes and lower disease risks are associated with WGF ingestion, but clear attribution to WGFs is difficult because high-WG eaters have lifestyles and diets deemed healthier, and vice versa (32, 37, 44, 49). The multiple confounding and potential interactions call into question the adequacy of multiple statistical adjustments (118).

WGF and RGF definitions are inconsistently applied and may be a source of error

Nonuniform definitions of and criteria for determining WGFs, together with imprecise terms for data collection (e.g., dark bread), or mis-categorization of foods, such as dark bread, bran, couscous, or crackers, can affect results and make comparisons among studies difficult (31, 32, 37, 44, 65, 120–124).

Binary categorization of GBFs into RGFs and WGFs

Early epidemiological studies classed foods as either WG or RG (31, 32). This was continued in most subsequent studies. Inadvertent bias against RGFs may have occurred because nearly all WGFs are recommended, staple GBFs, but over half of RGFs' calories come from nonrecommended, indulgent GBFs. No study has yet been published that compares health outcomes of staple and nonstaple WGFs and RGFs.

Intakes of GBFs are unbalanced

Consumers worldwide fail to meet the recommended balance of GBFs, e.g., for a 2000-kcal diet, six 30-g servings of GBFs, with half being WGFs. WGFs are underconsumed, and RGFs are overconsumed through large portions, more servings than recommended, and frequent ingestion of indulgent, nonrecommended RGFs (4, 6, 39, 124–131). When carbohydrate intakes approach 65% of E, overconsumption of RGFs, especially when diet quality is marginal or a person has certain metabolic conditions, may increase health risks (3, 69, 70).

Named patterns (e.g., "white bread") may be a source of inadvertent bias

Named dietary patterns, such as "white bread" (38), may promote deductions that the named food, not the pattern, is associated with health risks.

Recommendations for all WGFs may lower WG consumption and nutrient status

Foods formulated with both RGs (especially REGs) and WGs not only improve acceptability of WGFs, especially for children (132, 133), but also improve micronutrient content and/or availability and reduce intake of antinutritional components found in bran (134–138).

Recommendations for only WGFs extrapolate beyond existing data

Most epidemiological studies show health benefits of three 30-g WGF servings/d (48 g WG/d). Higher intakes are documented in regions such as Scandinavia, where the mix of grains includes a significant proportion of wheat, rye, barley, and oats. However, extrapolations using these data may have limited applicability in other regions.

Dietary pattern research supports the right mix of RGs and WGs

Recommended dietary patterns, such as the Mediterranean Diet (MEDiet), are associated with multiple positive health outcomes. These diet patterns demonstrate the health-promoting role of staple GBFs eaten in the recommended balance—half WGFs and half RGFs.

These points will be discussed to support current recommendations of dietary guidance for "half the grains to be WGs." Further, this discussion might spur reanalysis of existing studies, in which health impacts of indulgent GBFs are compared with staple GBFs. Such an analysis would have 4, not 2, GBF categories: WG staples, RG staples, WG indulgent foods, and RG indulgent foods.

Results and Discussion

WGFs are consistently associated with improved health outcomes in epidemiological, but not intervention, studies; RGFs may not be associated with increased health risks for some outcomes

High intakes of WGFs by consumers from different populations with diverse dietary intakes in epidemiological studies are consistently associated with lower total and disease-specific risk of early mortality (indicating a lower risk of dying during the follow-up period in prospective studies), improved measures of body weight, and lower risks of many chronic conditions. Despite a range of search strategies, databases, and inclusion criteria in meta-analyses of epidemiological studies, risk reductions consistently range from 10% to 25% for most health endpoints (15–50, 62–94). However, no such consistency was observed in RCTs and their meta-analyses (51–61, 95–101).

Mortality and GBFs.

Those ingesting the most WGFs were associated in prospective studies with lower RRs of mortality from all causes. Among studies, risk reductions of ≥15% were observed for those in the quintile ingesting the most WGFs (∼3 servings/d) compared with the least (<1 serving/d).

RRs ranged from 0.78 (95% CI: 0.67, 0.91) to 0.85 (95% CI: 0.80, 0.91) (17–32). There were 17–30% decreases in risks across studies for death from cardiovascular or coronary disease (RR: 0.70; 95% CI: 0.61, 0.79 to RR: 0.83; 95% CI: 0.79, 0.86). There was a 6–18% range in reduction in cancer risks for high-WGF consumers (RR: 0.82; 95% CI: 0.69, 0.96 and RR: 0.94; 95% CI: 0.87, 1.01 per 90 g WG/d; *P*-heterogeneity < 0.001) (17–27).

Often the same studies that show inverse associations between early mortality and intake of WGs also assessed health impacts of RG intake (18, 24–26, 29–32). Daily consumption of 6–7 servings (180–210 g/d) of RGFs did not raise mortality risk (RR: 1.02; 95% CI: 0.26, 1.45; *P*-heterogeneity = 0.64) (31). The same is true of GBFs, e.g., total bread (RR: 0.77; 95% CI: 0.72, 0.81; *P*-heterogeneity = 0.42) and breakfast cereals (RR: 0.87; 95% CI: 0.81, 0.93; *P*-heterogeneity = 0.17) (31, 102, 103). There was a wide range of RRs: 0.82 (95% CI: 0.48, 1.40) to 1.46 (95% CI: 0.80, 2.67). One meta-analysis (18) showed half the studies on RGFs had RRs $<$1.02 and 16 of 19 had RRs $<$1.11. Thus, RGFs were not associated with increased risk of early mortality from heart disease (RR: 1.16; 95% CI: 0.84, 1.59; *P*-heterogeneity = 0.12). Neither were white bread (RR: 1.07; 95% CI: 0.86, 1.34; *P*-heterogeneity = 0.160) nor total rice (RR: 0.98; 95% CI: 0.90, 1.07; *P*-heterogeneity = 0.44) (18). However, there was a possible trend toward increased risk for refined breakfast cereals (RR: 1.15; 95% CI: 0.79, 1.67; *P*-heterogeneity = 0.07) (18). Williams (103) and Williams et al. (47) reported similar findings for RGs but did not document an increased risk associated with breakfast cereals. Differences in background diets, total RG intakes, definitions of WGFs, and adjustment for various confounders may all contribute to variability of outcomes.

WGs and body weight: epidemiological studies.

WG consumption, in a variety of cross-sectional and prospective studies (33–50), is associated with lower risks of obesity measures such as high waist circumference (WC), visceral adipose tissue (VAT), BMI (in kg/m^2), and fat mass. In most studies the risk was ∼15% lower for high-WG consumers. Weight gain, over 8-y follow-up, was significantly lower (0.75 kg compared with 1.24 ± 0.23 kg, *P*-trend < 0.0001) for men ingesting a mean of three 30-g WGF servings/d (42.7 g WG/d) than for those eating very little WG ($<$5 g/d) (34). A meta-analysis of 13 prospective studies found that WG intake was associated with lower weight gain, ranging from 0.4 to 1.5 kg, during 8–13 y of follow-up (50).

In the Framingham Heart Study (35), there was an inverse relation of WGF intake with VAT and WC (*P*-trend < 0.001). Although increased RGF consumption was associated with increased VAT and WC (*P*-trends < 0.001) (35), the lowest VAT was observed with 2 servings of RGFs and 3 of WGFs, a balance approximating "half the grains as whole grains."

Cross-sectional data from NHANES 2001–2012 also showed a significant inverse relation of WG intake with BMI and overweight or obesity (39, 40). For example, the mean BMI ± SD for adults who ate no WGFs was 28 ± 0.1 compared with 27.6 ± 0.1 ($P < 0.0001$) for those who ate ≥1 WGF serving (39).

Low grades were assigned to the strength of the epidemiological evidence. Cho et al. (44) gave a grade of "C/D" owing to inconsistent results or methodological flaws. Further, reanalysis showed that only the inclusion of bran foods as WGFs resulted in a significant association. Schlesinger et al. (49) observed a weak inverse association between WGF intake and overweight/obesity (RR: 0.93; 95% CI: 0.89, 0.96) but graded the dose–response as "very low to low quality."

Results from RCTs studying WGFs and measures of weight are variable (50–61). Compared with RGFs, WGFs did not result in significant differences in weight or weight loss in a meta-analysis of 26 studies (weighted difference: 0.06 kg; 95% CI: −0.09, 0.20 kg; $P = 0.45$) (51). Similarly, no significant differences in BMIs, fat mass, or fat-free mass were observed in 21 RCTs, even 1 with large numbers of subjects (52, 53). Nor were there any significant effects with regard to body fat percentage [weighted mean difference (WMD): 0.27%; 95% CI: −0.05%, 0.58%; $P = 0.09$], fat mass (WMD: 0.45 kg; 95% CI: −0.12, 1.02 kg; $P = 0.12$), or WC (WMD: 0.06 cm; 95% CI: −0.50, 0.63 cm; $P = 0.82$) (53). Several studies showed that weight was lost with both WGs and RGs (54–56); a slightly lower body fat percentage (weighted difference: −0.48%; 95% CI: −0.95%, 0.01%; $P = 0.04$) was observed with WGFs in 1 study (51). In another study where 33 low-WG consumers substituted WGFs for RGFs, there was a trend toward lower body weights but differences did not reach statistical significance (Δ intervention 2-sample *t* test, $P = 0.10$), as for BMI (Δ intervention 2-sample *t* test, $P = 0.08$) (55).

Greater consistency was seen among studies where subjects had high intakes of WGs, especially when WG rye or oats were eaten (57–60). In a 1-y study of 298 subjects with T2D, a healthy diet plus 100 g/d of WG oats promoted −0.89 kg (95% CI: −1.56, 0.22 kg) greater mean weight loss than occurred with the usual-care high-fiber, low-fat diet (57). In another study, addition of ready-to-eat cereal (RTEC) WG oat cereal with 3 g β-glucan to intervention diets did not result in a significant difference in weight loss from the control (−2.2 ± 0.3 compared with −1.7 ± 0.3 kg, $P = 0.325$), but did significantly decrease WC (−0.3 ± 0.4 compared with −1.9 ± 0.4 cm, $P = 0.012$) (58). In an 8-wk crossover study, 59 Danes ate either 179 ± 50 g WGFs/d or $<$13 g WGFs/d. The very high WG intake resulted in −0.2 kg loss compared with a 0.9-kg gain ($P < 0.001$) for those eating mostly RGs (59). Fat-free mass was lower ($P = 0.010$), and there was a tendency for lower WC ($P = 0.097$) (59). A 6-wk intervention comparing wheat and rye found that, compared with RG wheat, WG rye caused greater mean loss of body weight (+0.15 ± 1.28 and −1.06 ± 1.60 kg, respectively; $P < 0.01$) and fat mass (−0.04 ± 0.82 and −0.75 ± 1.29 kg, respectively; $P < 0.05$) (60).

Many RCTs replaced RGFs with WGFs in diets of consumers with habitually low WGF intake. However, many showed little effect on weight. Calorie restriction had larger impact than WG or RG (61). A few RCTs, usually with grains such as rye or oats, showed trends toward reductions in some measures of weight. Although mechanisms of action have been suggested, the losses of <5% body weight found in most studies raise questions about clinical or health relevance (139).

RGs and body weight.

Findings on RGFs and body weight are also not consistent. Some large cohort studies show that intake of RGFs is associated with increased risk of weight gain or higher BMIs. However, this usually occurs with intakes of RGFs higher than recommended. For example, analysis of food frequencies of middle-aged female nurses shows RG median intakes for the highest and lowest quintiles were 2.27 servings $\cdot$ 1000 $kcal^{-1} \cdot d^{-1}$ and 0.40 servings $\cdot$ 1000 $kcal^{-1} \cdot d^{-1}$, respectively. Thus, at 2000 kcal/d those at the highest intakes would be ingesting >5 grain servings daily. Thus, the chances of BMI > 30 were elevated (adjusted OR: 1.18; 95% CI: 1.01, 1.35; *P*-trend < 0.0001) (45). However, the ORs for quintiles 2–4 hovered around 1.0 (OR: 0.96; 95% CI: 0.88, 1.05; OR: 0.94; 95% CI: 0.86, 1.00; and OR: 1.03; 95% CI: 0.95, 1.13, respectively; *P*-trend < 0.0001) (45). In contrast, dietary records from the Baltimore Longitudinal Study on Aging showed median RG intake of 39 g/d in the lowest quintile and 102 g/d in the highest quintile. Neither mean baseline BMIs $\pm$ SD for those in the lowest quintile of refined grain intake compared to the highest quintile of intake (24.9 $\pm$ 0.2 vs 25.2 $\pm$ 0.2, $P = 0.51$, respectively) nor body weights $\pm$ SD (73.5 $\pm$ 0.7 vs 73.9 $\pm$ 0.7 kg, $P = 0.78$, respectively) were significantly different (38). In a meta-analysis of food groups (49), a J-shaped risk curve with increasing intakes of RGFs was observed (*P*-nonlinearity < 0.001). The RR of elevated body weight was <1 for RGF intakes between 0 and 90 g/d but increased with intakes of >100 g/d. At intakes of 200 g/d, the RR increased to 1.43 (95% CI: 1.26, 1.63).

Daily ingestion of breakfast cereals, in any combination of WG and RG, compared with infrequent ingestion (≤1/wk) by men in the Physicians' Health Study was associated with greater likelihood of having a BMI < 25 (RR: 0.81; 95% CI: 0.64, 1.03; *P*-trend < 0.03) (43). This association was assigned a "grade of B" (140).

The effect of RGF intake on weight gain lacks consistency. White bread consumption for those students in the Seguimiento Universidad de Navarra (SUN) cohort in the highest quartile of intake (median $\pm$ SD) (171 $\pm$ 62 g/d; 6 slices per day), compared with the quartile with the lowest intake (<3 $\pm$ 4 g/d; <3 slices per week), was associated with an elevated risk of becoming overweight or obese (adjusted OR: 1.40; 95% CI: 1.08, 1.81; *P*-trend = 0.008) (46). While a significantly elevated risk was observed for those with the highest intakes, the ORs for the 2 middle quartiles were only slightly elevated. For those in the second and third quartiles, white bread intakes were 36 $\pm$ 11 g/wk and 60 $\pm$ 0 g/d (420 $\pm$ 0 g/wk), but the adjusted ORs were similar (OR: 1.14; 95% CI: 0.90, 1.50 and OR: 1.12; 95% CI: 0.90, 1.50, respectively). If white bread is associated with elevated body weight, it is illogical that a mean consumption of 380 g/wk more white bread caused no difference in the risk of overweight in the middle quartiles.

Such data may be explained by significant confounding. Students in the SUN quartile with the highest white bread intake also had higher baseline BMIs and body weights and significant differences in the 21 dietary qualities measured, with most varying in ways considered less healthful ($P < 0.0001$). With significant dietary differences including a mean of 446 more calories per day for white-bread consumers ($P < 0.001$), adverse health outcomes ascribed to white bread intake may be misleading (46).

The impact of RGFs on weight depends on overall diet and total grain intake. Chinese adults eating >401 g white rice/d, compared with <200 g/d, were associated with less weight gain in 5 y (−2.08 kg; 95% CI: −2.75, −1.41 kg; $P < 0.001$) (141). These findings should not be interpreted to mean that high rice consumption does not provide more calories, but rather to emphasize that total calories and other food choices, not rice alone, determine weight gain.

Low grades were assigned to the quality of the data on RGFs and weight. In a meta-analysis of food groups, those eating the highest compared with the lowest number of RGF servings per day had a greater risk of weight gain (RR: 1.18; 95% CI: 0.95, 1.50), but the NutriGrade quality of evidence was graded "very low" (49). Further, risk of weight gain was flat until consumers ate ≥3 servings/d (49). The German Nutrition Society's evidence-based review on carbohydrates and other reviews concluded there was insufficient evidence to link RGFs to weight, obesity, and most measures of body fatness (142). In cases where evidence exists, it was deemed weak and effects null or small and significantly confounded (49–51, 53, 102, 103).

WGs, glycemia, and diabetes.

Ingestion of WGFs (∼3 servings/d) was associated, in a number of prospective cohort studies and systematic reviews, with decreased risks of incidence of and mortality from T2D (15–28, 62–72). In meta-analyses Aune et al. (18, 68) found that 3 WGF servings/ d were associated with a reduced risk of T2D and death from T2D (summary RR: 0.68; 95% CI: 0.58, 0.81; $I^2 = 82\%$; and RR: 0.49; 95% CI: 0.23, 1.05; *P*-nonlinearity < 0.0001, respectively). Similar risk reductions were reported in other studies for T2D and associated biomarkers such as glucose tolerance or inflammation and for intakes of specific WGFs including breads, cereals, rye, oats, and rice (50, 63–77).

In a meta-analysis assessing the impact of food groups on T2D risk, WGFs reduced the risk (RR: 0.77; 95% CI: 0.71, 0.84) (76). The strength of association was graded "high," with the greatest risk reduction occurring with WG intakes between 0 to 40 g/d (76). Similarly, Aune et al. (68) drew a U-shaped curve, suggesting the lowest risk occurs with ∼3 WG servings/d (50 g/d).

Confounding by DF and by other variables known to lower diabetes risk makes attribution of risk to WGs alone challenging. Those in the Women's Health Initiative (WHI) who ingested >2 WGF servings/d had a reduced risk of

T2D (HR: 0.75; 95% CI: 0.63, 0.89; $P < 0.0139$) (62). When adjusted for lifestyle and most dietary factors, the association remained; however, adjustment for DF attenuated it. A similar attenuation was observed when bran foods were not counted as part of WGFs (44, 65).

Lower incidence of T2D is associated in epidemiological studies with habitual consumption of ~3 WGF servings/d (50 g WG/d). Calls for higher WGF intake are speculative because only a few cohorts consume higher amounts. In Scandinavia the highest quintile of WGF intake reaches 170 g/d and includes a variety of WGFs: oats, barley, and rye. Each is associated with lowering inflammation and blood sugar parameters (64). Extrapolations for intakes beyond 50 g/d WG may not apply to all populations (67) where lower intakes are documented and WG mixtures differ.

RGs, glycemia, and diabetes.

Many studies showing that WGFs reduce T2D risk also find that RGFs, especially in Western cohorts, do not increase, and may even lower, risk (18, 65–77). In a meta-analysis of 15 prospective studies assessing RGFs and T2D, the range of RGF intake was 0–700 g/d and the risk of T2D for the highest compared with the lowest quintile was not elevated (RR: 1.01; 95% CI: 0.92, 1.10) (76). Similarly, a recent meta-analysis of 16 cohort studies found RGF intake was not associated with risk of T2D (RR: 0.95; 95% CI: 0.88, 1.04) (68). Women in the WHI eating ≥6 RGF servings/d compared with <1 serving/d had a lower risk of T2D (adjusted HR: 0.73; 95% CI: 0.60, 0.87; $P < 0.0519$) (62). However, RGFs were positively associated with fasting insulin (P-trend = 0.002) in women in the Baltimore Longitudinal Study of Aging (37).

RGFs were associated with increased risk of T2D in regions with high intakes of carbohydrate and/or rice (>200 g/d) (141, 143–152). In parts of India where carbohydrate intakes for the highest quartiles are above recommendations at >72.8% of E and DF intakes low and mean intake ± SD of RGFs was 516.5 ± 137.1 g/d, the risk of T2D for those in the highest quartiles of intake was markedly increased (adjusted OR: 4.98; 95% CI: 2.69, 9.19; $P < 0.001$) (143, 144). The quartile ingesting median white rice intakes of 587 g/d, compared with those ingesting less, had elevated T2D risk (OR: 5.31; 95% CI: 2.98, 9.45; $P < 0.001$) (143, 144). For Chinese middle-aged women with high carbohydrate and white rice (≥200 g/d) intakes, T2D risks were raised but not nearly as much as in India (RR carbohydrate: 1.28; 95% CI: 1.09, 1.50; RR rice: 1.78; 95% CI: 1.48, 2.15) (141). In the Japan Public Health Center-based Prospective Study, white rice intake was associated with T2D in women (OR: 1.65; 95% CI: 1.06, 2.57; P-trend < 0.005, for the highest compared with the lowest quartile) (145, 146). There was a nonsignificant trend in sedentary men (P-trend = 0.08) (146). However, these same data analyzed by dietary patterns showed that in those eating the "westernized breakfast pattern" with more bread and less rice, RG intake was inversely related to A1C concentrations (P-trend = 0.02) (147). In the Chinese Jiangsu study, hyperglycemia risk increased linearly as rice intakes increased across tertiles from <200 to 201–400 and ≥401 g/d (RR: 1; 1.96; 95% CI: 1.07, 3.60 and RR: 2.50; 95% CI: 1.37, 4.57, respectively ; P-trend < 0.005) (147).

Carbohydrates provided 57.7% of E in urban Tehran and 56.9% of E in rural Iran (Golestan). However, mean total calorie intake was also roughly 200 calories/d more in Tehran than rural Iran. Rice intake impacted T2D risk not only because of differences in caloric intake, but also perhaps because the mean rice intake is 250 g/d and 15.7% of E in urban Tehran and 120 g/d (77–210 g/d) and 8% of E in rural Iranians. In Tehran those eating >250 g/d of rice were associated with increased risk (adjusted OR: 2.08; 95% CI: 1.08, 3.91) compared with those eating less. In the the rural cohort where white rice intakes are lower, there was a slightly increased risk for those in the tertil ingesting >210g/d of white rice (adjusted OR: 1.05; 95% CI: 0.85, 1.30; $P < 0.001$) (148). Differences in lifestyle and diet in rural Iran including more activity, ingestion of more bread including soluble fiber and magnesium and less meat are postulated as attenuating T2D risk (148).

High rice consumption in other cohorts is not always associated with increased T2D risk. In the Singapore Chinese Cohort Study, there was no elevation of T2D risk due to high rice consumption.The median rice intakes for the high and low quintiles varied from 236.5 to 649.3 g/d with the adjusted HR: 0.98; CI: 0.90, 1.08) (149). However, this study differs from others because risk calculations were adjusted for many factors and shows that other foods such as meat or Asian noodles substituted for white rice increases the HR.

In a country where rice consumption is lower, its consumption was associated with reducing T2D risk. In a prospective cohort in southern Spain, "frequent" rice consumption (>3 times/wk) versus less than once a week was associated with a lower risk of developing T2D over a 6-y period (adjusted OR: 0.43; $P = 0.04$) (150).

Meta-analyses do not help clarify matters. One comparing 3 Asian and 4 Western cohorts done in 2012 found an association between high rice intake and increased T2D risk (RR: 1.55; 95% CI: 1.20, 2.01; and RR: 1.12; 95% CI: 0.94, 1.33, respectively) (151). However, a 2017 meta-analysis of 11 studies found white rice consumption was not associated with increased risk of developing T2D (pooled RR: 1.08; 95% CI: 0.87, 1.33; $P = 0.33$), even when stratified by region for rice consumption (152).

Heterogeneity among studies is contributed by differences in genetics, lifestyle, and diet including rice intakes. For example, rice intakes in 3 US cohorts varied from 5.3 to 112.9 g/d across the quintiles (151). In Japanese cohorts they ranged from 278 to 560 g/d, but in Shanghai women from 500 to 750 g/d with 67.5% of E from carbohydrate (82, 141, 145, 146).

Assessment of RGFs' impact on T2D risk in RCTs requires the use of biomarkers such as blood glucose and insulin resistance (50–61, 79, 96–103), and they also show variability. Neither RGs nor WGs in a 6-wk crossover study (n = 33) caused any difference in blood glucose values (ΔGlucose from baseline with RG intervention 0.0 ± 0.1 mmol/L and WG intervention: –0.1 ± 0.1 mmol/L; NS)

(55). Another study in 50 overweight subjects showed that WG ingestion caused greater decreases in blood glucose than RG (−4.3 ± 1.15 compared with −1.0 ± 1.1 mg/dL, $P = 0.02$) (56). Glycemia after oral-glucose-tolerance tests trended lower when middle-aged adults were eating WGs compared with RGs ($P = 0.10$) (58).

Meta-analyses of RCTs reflect the variation observed. One study of 21 RCTs showed significantly lower mean weighted fasting glucose differences (−0.93 mmol/L; 95% CI: −1.65, −0.21 mmol/L; P-heterogeneity < 0.05) (50). Another showed that WGFs reduced the postprandial values of the glucose integrated AUC (iAUC) (0–120 min) by −29.71 mmol/L · min (95% CI: −43.57, −15.85 mmol/L · min) in acute studies, but not in 14 RCTs lasting longer than 2 mo (95). There were trends toward lower plasma glucose with WG compared with control meals, but heterogeneity among the studies was large ($I^2 = 80\%$; $P < 0.001$).

The degree of grinding/milling had an impact on blood glucose. Consumption of foods from either WG or RG wheat flour did not cause a significant reduction in blood glucose (iAUC: −6.7 mmol/L · min; 95% CI: −25.1, 11.7 mmol/L · min; $P = 0.477$) in a meta-analysis of 20 studies (96). Similarly, ground WG and RG rye flours caused no difference in blood glucose curves iAUC (−5.5 mmol/L · min; 95% CI: −24.8, 13.8 mmol/L · min; $P = 0.576$). However, brown rice kernels, compared with white rice, resulted in a significant reduction in blood glucose response (iAUC: −40.5 mmol/L · min; 95% CI: −59.6, −21.3 mmol/L · min; $P < 0.001$) (96).

Data on body weight and T2D show the variability seen with most health endpoints. WG ingestion may be associated with reduced risks in epidemiological studies, but findings are not consistent among studies. Confounding is a huge concern.

Because changes in biomarkers are measured, and not disease incidence, comparison of findings from RCTs and epidemiological studies is difficult. It is further complicated by varying designs, small numbers of subjects and their selection criteria, and the short duration of studies (<4 mo). Many factors appear to affect observed health outcomes in both types of studies including design, power, subject characteristics, compliance, background diet, grain type, WG definition used, and the type and mix of WGs.

Studies of WGs and RGs have multiple sources of confounding

Better health outcomes and lower disease risks are associated with ingestion of WGFs. In nearly all studies WGF consumers are documented as having health-promoting diets and lifestyles and vice versa (28, 31, 33–41, 44–46, 49). Specifically, WG consumers compared with RG consumers have higher Healthy Eating Indexes (HEIs), ingest less total E, sugar, alcohol, fat, red meat, and indulgent GBFs, and ingest more fruits, vegetables, and fish (41, 46, 91, 153).

Dietary variables covary. High intakes of RGFs, especially at amounts higher than dietary guidance, result in lower consumption of other recommended foods. For example, in the SUN cohort, as white bread consumption increased, vegetable and fruit consumption decreased (46, 153). This forces the question of whether observed associations are due to higher white bread intake, lower intake of fruits and vegetables, or their interaction.

Dietary choices also vary with lifestyle (153–161). High-WG consumers are more likely to have normal BMIs, higher educational attainment, and socioeconomic status, and to be physically active and nonsmokers, and vice versa (31, 32, 37, 46, 152–159, 162). Models attempt to adjust for many factors: calories, red and processed meat, fruits, vegetables, dairy, fat, protein, sugar-sweetened beverages (SSBs), antioxidants, nutrients, DF, measures of diet quality (e.g., HEI), relevant health history, baseline values (e.g., blood glucose or BMI), race-ethnicity, physical activity, smoking status, education, supplement and alcohol use, and menopausal status and hormone use. Because few adjust for all of the possible known confounders, the likelihood of residual confounding is substantial. The myriad of potential interactions may muddle accurate statistical adjustment, especially as factors tend to cluster, thus amplifying observed positive or negative effects (118).

Definitions of WGFs and RGFs are inconsistent and may be a source of error or bias

There is concurrence regarding the definition of WGs as an ingredient or single food (e.g., brown rice) (11–14), but little agreement regarding the definition of WGFs or ways to report WG intake (120–123). The varying definitions and criteria among studies make comparing results difficult. A few studies report grams of WG, which recognizes foods contributing any amount of WG. Most studies use ≥25% WGs/serving as initiated by Jacobs et al. (31, 32). With this criterion WGFs could contain 74% RG. A few studies use the FDA Health Claim of >51% WG by weight per serving (37, 123), which means WGFs could have 49% RG. These sharp cutoffs mean that foods with 24% or 50% WGs, respectively, are counted as RGFs. Because many WGFs may contain between 49% and 74% RG, the benefits attributed to WGFs may more accurately be associated with the mix of WGs and RGs.

Imprecise terms used in food frequencies coupled with consumers' inability to identify WGFs or RGFs create error (160, 161, 163–165). For example, respondents are asked how frequently they eat "dark bread." Caramel-colored or multigrain bread may be identified by consumers as "dark" despite it being mostly RGs. Conversely, light-colored RGFs made with white WG wheat may be classed as RG despite their WG content.

Mis-categorization errors also occur. Couscous, bulgur, farro, and barley were counted as WGFs, despite many marketplace forms being RG (31, 32, 164). Foods with 25% bran were deemed WG in some studies. Reanalyses excluding bran foods erased the significant risk reductions attributed to WGs (44, 65).

Foods with positive health images, e.g. WGFs, are more likely to be reported, whereas those with negative health images, e.g., indulgent RGFs such as sweets and cakes, are

TABLE 1 Percentages by food category of weekly servings of foods classed as WGs and RGs in most epidemiological studies[1]

	Percentage of weekly servings[2]	Recommended staple (core) food
WG food categories		
Dark bread	60.5	Yes
WG ready-to-eat cereal	17.6	Yes
Popcorn	13.4	At times[3]
Oatmeal	6.8	Yes
Wheat germ	1.5	Yes
Brown rice/rice mixes	1.3	Yes
Bran	0.6	Yes
Other[4]	0.3	Yes
RG food categories		
White bread and pita	29.9	Yes
Pasta	5.0	Yes
English muffins, bagels, rolls	4.7	Yes
RG breakfast cereal	3.9	Yes
White rice and mixes	3.4	Yes
Pizza crust	2.5	At times
Sweets/desserts	45.2	No
Muffins or biscuits	3.2	At times
Sweet rolls	2.6	No
Pancakes or waffles	22.6	At times
Crackers[5]	Not included in initial study	At times

[1]RG, refined grain; WG, whole grain.
[2]Percentages of weekly WG servings for WG foods; percentages of weekly RG servings for RG foods. Data are from the Iowa Women's Health Study (31, 32). Proportions of intakes from indulgent grains, RGs, and staple ones were similar in subsequent studies, such as references 33–37.
[3]An "at times" designation suggests the food in question could be a recommended staple if prepared with little sugar or saturated or total fat.
[4]Bulgur, kasha, couscous, barley.
[5]All crackers were deemed as RG regardless of their WG content.

underreported, especially by overweight individuals (166, 167). This could create errors for both WG and RG intake data.

Binary categorization into RGFs and WGFs

Categorizing GBFs as either WGFs or RGFs (31, 32) inadvertently biases in favor of WGFs. Seven of 8 WGF subcategories are staple (core) foods recommended in dietary guidance (**Table 1**). Only the "popcorn" category might be indulgent. Assuming all popcorn is indulgent, then 87% of weekly intake WGFs in the Iowa Women's Health Study (IWHS) are foods that contribute little sugar or fat and are recommended in dietary guidance.

In contrast, RGF categories contain a mix of recommended and nonrecommended foods (5–8). Breads comprise 30% of the IWHS weekly RGF servings (31, 32). The addition of rolls, bagels, English muffins, pasta, pizza crust, rice, and RG breakfast cereals means that 46.0% of weekly RGF servings are recommended in dietary guidance. In contrast, 45.2% of the weekly RGF servings were "sweets and desserts." With the addition of biscuits, muffins, pancakes, and doughnuts, over half the weekly intake of RG servings are foods that dietary guidance suggests "to limit" owing to contributions of added sugar, fat, and calories (Table 1) (31, 32).

Intakes of GBFs are unbalanced

Consumers worldwide under-consume WGFs and overconsume RGFs (5–10, 39, 124–129). Many consumers ingest more total grain servings than recommended for E needs (e.g., six 30-g servings per 2000 kcal) (5, 6). Many ingest little if any WG, creating an imbalance. Large portions and energy-dense, nonrecommended RGFs contribute to imbalance (131, 132). In the IWHS, those with the highest intake of RGFs had a median intake of all GBFs of 30 servings/wk (range: 23.0–155.5 servings/wk) including "grain-based sweets and desserts" (range: 11.5–143 servings/wk) (31, 32). In contrast, those with the highest intake of WGFs had a median intake of all GBFs of 22.5 servings/wk (range: 18.5–84.5 servings/wk). Such data confirm those eating the most RGFs ate more total GBFs with many being nonrecommended. High-RGF consumers had higher mean sugar intakes (47.8 g/d compared with 38.8 g/d) and more total E (9.7 MJ/d compared with 8.6 MJ/d) than high-WGF consumers ($P < 0.001$) (31, 32).

As demonstrated in previous sections, health risks associated with GBFs are exacerbated when diet quality is marginal, RG intake is high, WG and DF intakes are low, and carbohydrate intakes are near or above the recommended 65% of E (3, 141, 143, 144, 147, 148).

Named patterns (e.g., "white bread pattern") may be a source of inadvertent bias

Dietary pattern research can yield insights beyond studies of single foods or components, partly because of the ability to assess dietary components that vary together (153–160). Patterns such as "white bread" or "RG and red meat" in epidemiological research are often associated with adverse health outcomes. Those not understanding pattern research as "a way of conceptualizing numerous diet exposures" (168, 169, may deduce that foods in the pattern name, not the pattern, are associated with increased risk (38). In reality, the pattern name describes diets not meeting the food group distribution prescribed in dietary guidance. A pattern name such as "low fruit and vegetable" or "unbalanced" might be less misleading.

Recommendations for only WGFs may impair nutrient status and lower WG consumption

RGFs, especially enriched or fortified staples, provide nutrients which are potentially at risk if RGF consumption is limited. REGs have reduced the percentage of the US population failing to meet the estimated average requirement as follows: iron, from 22% of the population to 7%; thiamin, from 51% to 4%; vitamin B-6, from 22% to 12%; riboflavin, from 11% to 1.7%; and folate, from 88% to 11% (134).

Near-daily RTEC (WG or RG) consumption was associated with increased milk, yogurt, and fruit consumption and lower risks of inadequate intakes of vitamin A, calcium, folate, vitamin B-6, magnesium, and zinc (136–138, 168).

Despite contributing to higher sugars, both RG and WG RTECs were associated, in a systematic review of 64 studies, with dietary patterns containing more DF and micronutrients (135). Nutrient availability may be enhanced from REGs because of specific fortificants used and fewer bran components that impair absorption (136, 137, 168, 170).

Mandatory (and voluntary) folate fortification of RG flour in many jurisdictions deserves mention as an initiative that is helping to decrease the incidence of spina bifida, anencephaly, and other birth defects (171, 172). The importance of REGs or fortified grains is demonstrated in a study of US women. In the year before conception, women adhering to dietary regimens avoiding grains or carbohydrates were associated with having a 30% increase in risk of folate-related birth defects in their offspring (172,173).

DF also deserves a mention, because only 4% of the US population ingests the recommended amount of DF (174–177). Although WGFs contribute 15.3% of the total US DF, and would contribute more if more were eaten, recommendations for exclusive WGF intake fail to consider that RGFs provide 54.5% of the DF consumed (175–177).

GBFs are the only source of cereal fiber, and various fibers are needed to meet all the fibers' functions (178). Because DF is "a nutrient of concern," research is needed to show that recommendations for no GBFs or no RGFs lower neither total DF nor cereal fiber intakes.

Recommendations for ingestion of only WGFs extrapolate beyond existing data

In the highest quintiles of most cohorts, median WGF consumption is ∼3 servings/d (∼80–90 g WGF/d or 48 g WG/d). Although regions such as Scandinavia have higher WGF consumption, the foods include WG rye, barley, and oats (67). Thus, extrapolating from these data to other populations may not be predictive. Further, intervention data (52–61, 71, 95–101), even where all WGFs were substituted for RGFs, yield inconsistent results.

In the US NHANES (2009–2012; 2012–2014) and the Canadian Community Health Survey (2015), the right mix of WGFs and RGFs is supported by cross-sectional and epidemiological data from both children and adults (176, 177, 179–180) and is associated with better nutrient and body weight measures than intake of all WGFs or no grains (135). Data from the Framingham Heart cohort showed the lowest VAT was associated with the ingestion of 3 WGF and 2 RGF servings (35). Because WGFs may contain 49–74% RGs, positive health outcomes associated with WGFs tacitly argue for the mix of WGs and RGs recommended in dietary guidance.

Dietary pattern research supports the right mix of RGs and WGs

Vetted, balanced patterns [e.g., MyPlate, Dietary Approaches to Stop Hypertension (DASH), or MEDiet] demonstrate the health impact of diets with 50% carbohydrate and the right mix of foods including GBFs (5, 6, 10, 181–184). Analyses of the DASH diet or MEDiet (182–184) show a mix of 35% RGFs and 23% WGFs. Thus, cross-sectional, epidemiological, and dietary pattern research suggests associations with positive health outcomes when total grain consumption meets recommendations and is comprised of roughly half RGs and WGs

Conclusions

A dietary balance of GBFs and carbohydrates is associated with better nutrient intake and health measures. A recent meta-analysis showed that the lowest all-cause mortality risk was associated with 50% of E coming from carbohydrate (185). Epidemiological findings consistently show that ingestion of three 30-g WGF servings/d (48 g WGs/d) is associated with reduced chronic disease risk. However, findings from RCTs that replace all RGFs with WGFs yield inconsistent results. Many of the same studies that show WGFs are associated with better health outcomes also show that RGFs, especially staples, eaten in recommended amounts, are not independently associated with increased risk. Very high intakes of specific RGFs such as rice are associated with increased risks in certain populations. However, the variability and potential residual confounding among studies make attribution of risks to rice or RGFs problematic.

Outcomes of epidemiological studies skew toward WGs for the following reasons. *1*) Multiple confounding occurs because WG eaters have healthy behaviors (and vice versa), thus amplifying the health benefits of WGF consumption. *2*) Varying WGF definitions make comparison of study outcomes challenging and mean that WGFs may contain between 49% and 74% RGs, which argues for a mix of WGs and RGs. *3*) Binary categorization of GBFs creates inadvertent bias in favor of WGFs because the WGF categories are comprised almost exclusively of recommended GBFs, whereas the RGF category consists of half recommended and half indulgent GBFs. Reanalysis of food intake data separating indulgent GBFs from staple ones could be useful. *4*) GBF intake is unbalanced. Some consumers overconsume GBFs, mostly as RGFs and often indulgent RGFs. Nearly all populations underconsume WGFs (186–194). This imbalance coupled with overconsumption of RGFs amplifies negative impacts of RGFs. *5*) Food pattern names such as "white bread" may subtly direct thoughts to the named food (e.g., white bread), not to the dietary imbalance or foods missing from the pattern.

Some advocate that all GBFs should be WGFs, despite evidence showing that RGFs, especially staples eaten in recommended amounts, are not independently associated with increased risk. Nevertheless, some health professionals and groups such as the 2019 EAT–*Lancet* Commission deem RGFs as "unhealthful plant-based foods," categorize them with other dietary pariahs—processed meats, sweets, and SSBs—and suggest their reduction or elimination (195–198).

Recommendations that GBFs should be all WGFs extrapolate beyond the existing data. In most cohorts the quintile eating the most WGFs ingests ∼3 servings/d or 90 g/d (∼48 g/d WG). Extrapolation using data from populations with higher consumption may not be applicable to cohorts where lifestyle, diet, and mix of WGFs (wheat, rye, barley, and oats) differ.

Important nutritional contributions of RGs, especially REGs, to iron, folic acid, and B-vitamin intakes may be disregarded by those zealously recommending all WGFs. Further, findings from vetted patterns, such as the MEDiet where the mix of WGFs and RGFs contributes to healthy outcomes, may be overlooked. RGs can make WGFs more acceptable and, thereby, improve WG intake. Inclusion of GBFs can aid long-term dietary compliance and create sustainable dietary change by contributing plant-based protein, dietary fiber, and nutrients in forms that are acceptable, familiar, affordable, and shelf-stable. Recommendations for GBFs need to be crafted so that consumers aim to ingest the recommended number of servings of GBFs (mostly staples) to meet E needs, and so that consumers replace half the RGF servings with WGFs. These points affirm the role of GBFs in the diet and support recommendations that consumers "make at least half their grains whole."

Acknowledgments

The authors' responsibilities were as follows—JMJ: did most of the writing; CGG and HJB: were involved in the inception of the initial articles that led to this article and offered suggestions as the paper progressed, on the first submission and the revised versions; and all authors: read and approved the final manuscript.

References

1. FAO. Staple foods: what do people eat? [Internet]. Rome, Italy: FAO; 1995 [cited 10 August, 2018]. Available from: http://www.fao.org/3/u8480e/u8480e07.htm.
2. Jones JM, Peña RJ, Korczak R, Braun HJ. CIMMYT series on carbohydrates, wheat, grains, and health. Carbohydrates, grains, and wheat in nutrition and health: an overview. Part I. Role of carbohydrates in health. Cereal Fds Wld 2015;60:224–33, 10.1094/CFW-60-6-0260.
3. Mohan V, Unnikrishnan R, Shobana S, Malavika M, Anjana RM, Sudha V. Are excess carbohydrates the main link to diabetes & its complications in Asians? Indian J Med Res 2018;148(5):531–8.
4. US Department of Agriculture. Nutrition and your health: Dietary Guidelines for Americans, 2005: part D: science base: section 5: carbohydrates [Internet]. Washington (DC): US Department of Health and Human Services and USDA; 2015 [cited 4 May, 2018]. Available from: https://health.gov/dietaryguidelines/dga2005/report/html/d5_carbs.htm#top.
5. US Department of Agriculture. ChooseMyPlate: make half your grains whole grains [Internet]. Alexandria, VA: USDA Center for Nutrition Policy & Promotion; 2016 [cited 10 August, 2018]. Available from: https://www.choosemyplate.gov/eathealthy/make-half-your-grains-whole-grains.
6. US Department of Agriculture and Department of Health and Human Services. Dietary Guidelines for Americans, Shifts Needed To Align With Healthy Eating Patterns; Current Eating Patterns in the United States [Internet]. 8th ed. Washington (DC): US Department of Health and Human Services and USDA; 2016 [cited 4 May, 2018]. Available from: https://health.gov/dietaryguidelines/2015/guidelines/chapter-2/current-eating-patterns-in-the-united-states/
7. WHO/FAO. Food-based dietary guidelines – 2019 [Internet]. Rome, Italy: FAO [accessed 17 October 2019]. Available from: http://www.fao.org/nutrition/education/food-dietary-guidelines/home/en.
8. Health Canada. Eating Well with Canada's Food Guide [Internet]. Ottawa, ON: Health Canada; 2007 [cited 10 August, 2018]. Available from: https://www.canada.ca/en/sr/srb.html?q=make+half+your+grains+whole+grains&wb-srch-sub=wb-land.
9. Public Health England, Scientific Advisory Committee on Nutrition. Carbohydrates and health [Internet]. London: TSO; 2015 [cited 10 August, 2018]. Available from: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/445503/SACN_Carbohydrates_and_Health.pdf.
10. Gray A, Threlkeld RJ. Nutritional recommendations for individuals with diabetes. In: Feingold KR, Anawalt B, Boyce A, Chrousos G, Dungan K , Hershman JM, Kaltsas G, Koch C, Kopp P, et al., editors. Endotext [Internet]. South Dartmouth, MA: MDText.com, Inc.; 2015 [cited 10 August, 2018]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279012/.
11. AACC Committee to Define Whole Grain. AACC members agree on definition of whole grain [Internet]. Cereal Fds Wld 2000;45:79 [cited 3 May, 2019]. Available from: https://www.aaccnet.org/initiatives/definitions/Documents/WholeGrains/wgflyer.pdf.
12. US FDA Food Labeling and Standards Staff (HFS-820), Office of Nutrition, Labeling and Dietary Supplements, Center for Food Safety and Applied Nutrition. Draft guidance for industry and FDA staff: whole grain label statements [Internet]. Rockville, MD: Food and Drug Administration; 2006 [cited 10 August, 2018]. Available from: https://www.fda.gov/regulatory-information/search-fda-guidance-documents/draft-guidance-industry-and-fda-staff-whole-grain-label-statements.
13. Food Standards Australia New Zealand. Wholegrain food [Internet]. Kingston, ACT: Food Standards Australia New Zealand; 2016 [cited 10 August, 2018]. Available from: http://www.foodstandards.gov.au/consumer/nutrition/wholegrain/Pages/default.aspx.
14. van der Kamp JW. Whole grain definition: new perspectives for inclusion of grains and processing but not for analysis [Internet]. Cer Foods Wld Plexus 2012;15–16 [cited 10 August, 2018]. Available from: https://www.aaccnet.org/publications/plexus/cfwplexus/library/books/Documents/WholeGrainsSummit2012/CPLEX-2013-1001-08B.pdf.
15. McRae MP. Health benefits of dietary whole grains: an umbrella review of meta-analyses. J Chiropr Med 2017;16(1):10–18.
16. Seal CJ, Brownlee IA. Whole-grain foods and chronic disease: evidence from epidemiological and intervention studies. Proc Nutr Soc 2015;74(3):313–19.
17. Chen GC, Tong X, Xu JY, Han SF, Wan ZX, Qin JB, Qin LQ. Whole-grain intake and total, cardiovascular, and cancer mortality: a systematic review and meta-analysis of prospective studies. Am J Clin Nutr 2016;104:164–72.
18. Aune D, Keum N, Giovannucci E, Fadnes LT, Boffetta P, Greenwood DC, Tonstad S, Vatten LJ, Riboli E, Norat T. 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.
19. Huang T, Xu M, Lee A, Cho S, Qi L. Consumption of whole grains and cereal fiber and total and cause-specific mortality: prospective analysis of 367,442 individuals. BMC Med 2015;13:59.
20. Zhang B, Zhao Q, Guo W, Bao W, Wang X. Association of whole grain intake with all-cause, cardiovascular, and cancer mortality: a systematic review and dose-response meta-analysis from prospective cohort studies. Eur J Clin Nutr 2018;72:57–65.
21. Benisi-Kohansal S, Saneei P, Salehi-Marzijarani M, Larijani B, Esmaillzadeh A. Whole-grain intake and mortality from all causes, cardiovascular disease, and cancer: a systematic review and dose-response meta-analysis of prospective cohort studies. Adv Nutr 2016;7:1052–65.
22. Li B, Zhang G, Tan M, Zhao L, Jin L, Tang X, Jiang G, Zhong K. Consumption of whole grains in relation to mortality from all causes, cardiovascular disease, and diabetes: dose–response meta-analysis of prospective cohort studies. Medicine (Baltimore) 2016;95:e4229.

23. Zong G, Gao A, Hu FB, Sun Q. Whole grain intake and mortality from all causes, cardiovascular disease, and cancer: a meta-analysis of prospective cohort studies. Circulation 2016;133(24):2370–80.
24. Wei H, Gao Z, Liang R, Li Z, Hao H, Liu X. Whole-grain consumption and the risk of all-cause, CVD and cancer mortality: a meta-analysis of prospective cohort studies. Br J Nutr 2016;116:514–25.
25. Johnsen NF, Frederiksen K, Christensen J, Skeie G, Lund E, Landberg R, Johansson I, Nilsson LM, Halkjær J, Olsen A, et al. Whole-grain products and whole-grain types are associated with lower all-cause and cause-specific mortality in the Scandinavian HELGA cohort. Br J Nutr 2015;114:608–23.
26. Schwingshackl L, Schwedhelm C, Hoffmann G, Lampousi AM, Knüppel S, Iqbal K, Bechthold A, Schlesinger S, Boeing H. Food groups and risk of all-cause mortality: a systematic review and meta-analysis of prospective studies. Am J Clin Nutr 2017;105:1462–73.
27. Ma X, Tang WG, Yang Y, Zhang QL, Zheng JL, Xiang YB. Association between whole grain intake and all-cause mortality: a meta-analysis of cohort studies. Oncotarget 2016;7(38):61996–2005.
28. Wu H, Flint AJ, Qi Q, van Dam RM, Sampson LA, Rimm EB, Holmes MD, Willett WC, Hu FB, Qi S. Association between dietary whole grain intake and risk of mortality: two large prospective studies in US men and women. JAMA Intern Med 2015;175:373–84.
29. Liu S, Sesso HD, Manson JE, Willett WC, Buring JE. Is intake of breakfast cereals related to total and cause-specific mortality in men? Am J Clin Nutr 2003;77:594–9.
30. Steffen LM, Jacobs DR, Jr, Stevens J, Shahar E, Carithers T, Folsom AR. Associations of whole-grain, refined-grain, and fruit and vegetable consumption with risks of all-cause mortality and incident coronary artery disease and ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) study. Am J Clin Nutr 2003;78:383–90.
31. Jacobs DR, Meyer KA, Kushi LH, Folsom AR. Is whole grain intake associated with reduced total and cause-specific death rates in older women? The Iowa Women's Health Study. Am J Public Health 1999;89:322–9.
32. Jacobs DR, Jr, Meyer KA, Kushi LH, Folsom AR. Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. Am J Clin Nutr 1998;68: 248–57.
33. Jones JM, Peña RJ, Korczak R, Braun HJ. CIMMYT series on carbohydrates, wheat, grains, and health: carbohydrates, grains, and whole grains and disease prevention. Part I. Body weight and obesity. Cereal Fds Wld 2016;61:96–105.
34. Koh-Banerjee P, Franz M, Sampson L, Liu S, Jacobs DR, Jr, Spiegelman D, Willett W, Rimm E. Changes in whole-grain, bran, and cereal fiber consumption in relation to 8-y weight gain among men. Am J Clin Nutr 2004;80:1237–45.
35. McKeown NM, Troy LM, Jacques PF, Hoffmann U, O'Donnell CJ, Fox CS. Whole- and refined-grain intakes are differentially associated with abdominal visceral and subcutaneous adiposity in healthy adults: the Framingham Heart Study. Am J Clin Nutr 2010;92:1165–71.
36. Koh-Banerjee P, Rimm EB. Whole grain consumption and weight gain: a review of the epidemiological evidence, potential mechanisms and opportunities for future research. Proc Nutr Soc 2003;62:25–9.
37. Newby PK, Maras J, Bakun P, Muller D, Ferrucci L, Tucker KL. Intake of whole grains, refined grains, and cereal fiber measured with 7-d diet records and associations with risk factors for chronic disease. Am J Clin Nutr 2007;86(6):1745–53.
38. Newby P, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and changes in body mass index and waist circumference in adults. Am J Clin Nutr 2003;77:1417–25.
39. Albertson AM, Reicks M, Joshi N, Gugger CK. Whole grain consumption trends and associations with body weight and measures in the United States: results from the cross sectional National Health and Nutrition Examination Survey 2001–2012. Nutr J 2016;15:8.
40. O'Neil CE, Zanovec M, Cho SS, Nicklas TA. Whole grain and fiber consumption are associated with lower body weight measures in US adults: National Health and Nutrition Examination Survey 1999–2004. Nutr Res 2010;30:815–22.
41. Montonen J, Boeing H, Fritsche A, Schleicher E, Joost HG, Schulze MB, Steffen A, Pischon T. Consumption of red meat and whole-grain bread in relation to biomarkers of obesity, inflammation, glucose metabolism and oxidative stress. Eur J Nutr 2013;52: 337–45.
42. Thielecke F, Jonnalagadda SS. Can whole grain help in weight management? J Clin Gastroenterol 2014;48(Suppl 1):S70–7.
43. Bazzano LA, Song Y, Bubes V, Good CK, Manson JE, Liu S. Dietary intake of whole and refined grain breakfast cereals and weight gain in men. Obes Res 2005;13:1952–60.
44. Cho SS, Qi L, Fahey GC, Jr, Klurfeld DM. Consumption of cereal fiber, mixtures of whole grains and bran, and whole grains and risk reduction in type 2 diabetes, obesity, and cardiovascular disease. Am J Clin Nutr 2013;98:594–619.
45. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz G. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. Am J Clin Nutr 2003;78:920–7.
46. de la Fuente-Arrillaga C, Martinez-Gonzalez MA, Zazpe I, Vazquez-Ruiz Z, Benito-Corchon S, Bes-Rastrollo M. Glycemic load, glycemic index, bread and incidence of overweight/obesity in a Mediterranean cohort: the SUN project. BMC Public Health 2014;14: 1091.
47. Williams PG, Grafenauer SJ, O'Shea JE. Cereal grains, legumes, and weight management: a comprehensive review of the scientific evidence. Nutr Rev 2008;66:171–82.
48. Giacco R, Della Pepa G, Luongo D, Riccardi G. Whole grain intake in relation to body weight: from epidemiological evidence to clinical trials. Nutr Metab Cardiovasc Dis 2011;21:901–8.
49. Schlesinger S, Neuenschwander M, Schwedhelm C, Hoffmann G, Bechthold A, Boeing H, Schwingshackl L. Food groups and risk of overweight, obesity, and weight gain: a systematic review and dose–response meta-analysis of prospective studies. Adv Nutr 2019;10: 205–18.
50. Ye EQ, Chacko SA, Chou EL, Kugizaki M, Liu S. Greater whole-grain intake is associated with lower risk of type 2 diabetes, cardiovascular disease, and weight gain. J Nutr 2012;142:1304–13.
51. Pol K, Christensen R, Bartels EM, Raben A, Tetens I, Kristensen M. Whole grain and body weight changes in apparently healthy adults: a systematic review and meta-analysis of randomized controlled studies. Am J Clin Nutr 2013;98:872–84.
52. Brownlee IA, Moore C, Chatfield M, Richardson DP, Ashby P, Kuznesof SA, Jebb SA, Seal CJ. Markers of cardiovascular risk are not changed by increased whole-grain intake: the WHOLEheart study, a randomised, controlled dietary intervention. Br J Nutr 2010;104: 125–34.
53. Sadeghi O, Sadeghian M, Rahmani S, Maleki V, Larijani B, Esmaillzadeh A. Whole-grain consumption does not affect obesity measures: an updated systematic review and meta-analysis of randomized clinical trials. Adv Nutr 2020;11(2):280–92.
54. Kirwan JP, Malin SK, Scelsi AR, Kullman EL, Navaneethan SD, Pagadala MR, Haus JM, Filion J, Godin JP, Kochhar S, et al. A whole-grain diet reduces cardiovascular risk factors in overweight and obese adults: a randomized controlled trial. J Nutr 2016;146: 2244–51.
55. Ampatzoglou A, Atwal KK, Maidens CM, Williams CL, Ross AB, Thielecke F, Jonnalagadda SS, Kennedy OB, Yaqoob P. Increased whole grain consumption does not affect blood biochemistry, body composition, or gut microbiology in healthy, low-habitual whole grain consumers. J Nutr 2015;145:215–21.
56. Harris Jackson K, West SG, Vanden Heuvel JP, Jonnalagadda SS, Ross AB, Hill AM, Grieger JA, Lemieux SK, Kris-Etherton PM. Effects of whole and refined grains in a weight-loss diet on markers of metabolic syndrome in individuals with increased waist circumference: a randomized controlled-feeding trial. Am J Clin Nutr 2014;100: 577–86.
57. Li X, Cai X, Ma X, Jing L, Gu J, Bao L, Li J, Xu M, Zhang Z, Li Y. Short- and long-term effects of wholegrain oat intake on

weight management and glucolipid metabolism in overweight type-2 diabetics: a randomized control trial. Nutrients 2016;8(9):549.
58. Maki KC, Beiseigel JM, Jonnalagadda SS, Gugger CK, Reeves MS, Farmer MV, Kaden VN, Rains TM. Whole-grain ready-to-eat oat cereal, as part of a dietary program for weight loss, reduces low-density lipoprotein cholesterol in adults with overweight and obesity more than a dietary program including low-fiber control foods. J Am Diet Assoc 2010;110:205–14.
59. Roager HM, Vogt JK, Kristensen M, Hansen LBS, Ibrügger S, Mærkedahl RB, Bahl MI, Lind MV, Nielsen RL, Frøkiær H, et al. Whole grain-rich diet reduces body weight and systemic low-grade inflammation without inducing major changes of the gut microbiome: a randomised cross-over trial. Gut 2019;68:83–93.
60. Suhr J, Vuholm S, Iversen KN, Landberg R, Kristensen M. Wholegrain rye, but not wholegrain wheat, lowers body weight and fat mass compared with refined wheat: a 6-week randomized study. Eur J Clin Nutr 2017;71:959–67.
61. Katcher HI, Legro RS, Kunselman AR, Gillies PJ, Demers LM, Bagshaw DM, Kris-Etherton PM. The effects of a whole grain-enriched hypocaloric diet on cardiovascular disease risk factors in men and women with metabolic syndrome. Am J Clin Nutr 2008;87: 79–90.
62. Parker ED, Liu S, Van Horn L, Tinker LF, Shikany JM, Eaton CB, Margolis KL. The association of whole grain consumption with incident type 2 diabetes: the Women's Health Initiative Observational Study. Ann Epidemiol 2013;23:321–7.
63. Jones JM, Peña RJ, Korczak R, Braun HJ. CIMMYT series on carbohydrates, wheat, grains, and health: carbohydrates, grains, and whole grains and disease prevention. Part II. Blood pressure, metabolic syndrome, and diabetes. Cereal Fds Wld 2016;61:106–27, 10.1094/CFW-61-3-0096.
64. Kyrø C, Tjønneland C, Overvad K, Olsen A, Landberg R. Higher whole-grain intake is associated with lower risk of type 2 diabetes among middle-aged men and women: the Danish Diet, Cancer, and Health Cohort. J Nutr 2018;148:1434–44.
65. de Munter JSL, Hu FB, Spiegelman D, Franz M, van Dam RM. Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. PLoS Med 2007;4(8):e261.
66. Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, Willett WC. Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. Am J Clin Nutr 2002;76:535–40.
67. Chanson-Rolle A, Meynier A, Aubin F, Lappi J, Poutanen K, Vinoy S, Braesco V. Systematic review and meta-analysis of human studies to support a quantitative recommendation for whole grain intake in relation to type 2 diabetes. PLoS One 2015;10(6):e0131377.
68. Aune D, Norat T, Romundstad P, Vatten LJ. Whole grain and refined grain consumption and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of cohort studies. Eur J Epidemiol 2013;28:845–58.
69. Sun Q, Spiegelman D, van Dam RM, Holmes MD, Malik VS, Willett WC, Hu FB. White rice, brown rice, and risk of type 2 diabetes in US men and women. Arch Intern Med 2010;170:961–9.
70. Montonen J, Knekt P, Jarvinen R, Aromaa A, Reunanen A. Whole-grain and fiber intake and the incidence of type 2 diabetes. Am J Clin Nutr 2003;77(3):622–9.
71. Della Pepa G, Vetrani C, Vitale M, Riccardi G. Wholegrain intake and risk of type 2 diabetes: evidence from epidemiological and intervention studies. Nutrients 2018;10:1288.
72. Murtaugh MA, Jacobs DR, Jr, Jacob B, Steffen LM, Marquart L. Epidemiological support for the protection of whole grains against diabetes. Proc Nutr Soc 2003;62:143–9.
73. Jensen MK, Koh-Banerjee P, Franz M, Sampson L, Grønbæk M, Rimm EB. Whole grains, bran, and germ in relation to homocysteine and markers of glycemic control, lipids, and inflammation. Am J Clin Nutr 2006;83:275–83.
74. Qi L, van Dam RM, Liu S, Franz M, Mantzoros C, Hu FB. Whole-grain, bran, and cereal fiber intakes and markers of systemic inflammation in diabetic women. Diabetes Care 2006;29:207–11.
75. Reynolds A, Mann J, Cummings J, Winter N, Mete E, Te Morenga L. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. Lancet 2019;393(10170):434–45.
76. Schwingshackl L, Hoffmann G, Lampousi A-M, Knüppel S, Iqbal K, Schwedhelm C, Bechthold A, Schlesinger S, Boeing H. Food groups and risk of type 2 diabetes mellitus: a systematic review and meta-analysis of prospective studies. Eur J Epidemiol 2017;32:363–75.
77. Jones JM, Peña RJ, Korczak R, Braun HJ. CIMMYT series on carbohydrates, wheat, grains, and health. Carbohydrates, grains, and wheat in nutrition and health: their relation to digestion, digestive disorders, blood glucose, and inflammation. Cereal Fds Wld 2016;61:4–17, 10.1094/CFW-61-1-0004.
78. Lefevre M, Jonnalagadda S. Effect of whole grains on markers of subclinical inflammation. Nutr Rev 2012;70:387–96.
79. Buyken AE, Goletzke J, Joslowski G, Felbick A, Cheng G, Herder C, Brand-Miller JC. Association between carbohydrate quality and inflammatory markers: systematic review of observational and interventional studies. Am J Clin Nutr 2014;99:813–33.
80. Wang L, Gaziano JM, Liu S, Manson JE, Buring JE, Sesso HD. Whole- and refined-grain intakes and the risk of hypertension in women. Am J Clin Nutr 2007;86:472–9.
81. Schwingshackl L, Schwedhelm C, Hoffmann G, Knüppel S, Iqbal K, Andriolo V, Bechthold A, Schlesinger S, Boeing H. Food groups and risk of hypertension: a systematic review and dose-response meta-analysis of prospective studies. Adv Nutr 2017;8:793–803.
82. Yu D, Shu X-O, Li H, Xiang Y-B, Yang G, Gao Y-T, Zheng W, Zhang X. Dietary carbohydrates, refined grains, glycemic load, and risk of coronary heart disease in Chinese adults. Am J Epidemiol 2013;178:1542–9.
83. Juan J, Liu G, Willett WC, Hu FB, Rexrode KM, Sun Q. Whole grain consumption and risk of ischemic stroke: results from 2 prospective cohort studies. Stroke 2017;48(12):3203–9.
84. Chen J, Huang Q, Shi W, Yang L, Chen J, Lan Q. Meta-analysis of the association between whole and refined grain consumption and stroke risk based on prospective cohort studies. Asia Pac J Public Health 2016;28:563–75.
85. Wu D, Guan Y, Lv S, Wang H, Li J. No evidence of increased risk of stroke with consumption of refined grains: a meta-analysis of prospective cohort studies. J Stroke Cerebrovasc Dis 2015;24: 2738–46.
86. Deng C, Lu Q, Gong B, Li L, Chang L, Fu L, Zhao Y. Stroke and food groups: an overview of systematic reviews and meta-analyses. Public Health Nutr 2018;21:766–76.
87. Bechthold A, Boeing H, Schwedhelm C, Hoffmann G, Knüppel S, Iqbal K, De Henauw S, Michels N, Devleesschauwer B, Schlesinger S, et al. Food groups and risk of coronary heart disease, stroke and heart failure: a systematic review and dose–response meta-analysis of prospective studies. Crit Rev Food Sci Nutr 2019;59(7):1071–90.
88. Jones JM, Peña RJ, Korczak R, Braun HJ. Carbohydrates, grains, and whole grains and disease prevention. Part III. Cancer risk: overview, breast, and colorectal. Cereal Fds Wld 2016;61:228–42, 10.1094/CFW-61-6-0228.
89. Jones JM, Peña RJ, Korczak R, Braun HJ. Carbohydrates, grains, and whole grains and disease prevention. Part IV. Cancer risk: lung, prostate, and stomach. Cereal Fds Wld 2017;62:12–22, 10.1094/CFW-61-3-0096.
90. Skeie G, Braaten T, Olsen A, Kyrø C, Tjønneland A, Landberg R, Nilsson LM, Wennberg M, Overvad K, Åsli LA, et al. Intake of whole grains and incidence of oesophageal cancer in the HELGA cohort. Eur J Epidemiol 2016;31:405–14.
91. Makarem N, Nicholson JM, Bandera EV, McKeown NM, Parekh N. Consumption of whole grains and cereal fiber in relation to cancer risk: a systematic review of longitudinal studies. Nutr Rev 2016;74: 353–73.
92. Makarem N, Bandera EV, Lin Y, McKeown NM, Hayes RB, Parekh N. Associations of whole and refined grain intakes with adiposity-related cancer risk in the Framingham Offspring Cohort (1991–2013). Nutr Cancer 2018;70:776–86.

93. Xu Y, Yang J, Du L, Li K, Zhou Y. Association of whole grain, refined grain, and cereal consumption with gastric cancer risk: a meta-analysis of observational studies. Food Sci Nutr 2018;7:256–65.
94. Bonequi P, Meneses-González F, Correa P, Rabkin CS, Camargo MC. Risk factors for gastric cancer in Latin America: a meta-analysis. Cancer Causes Control 2012;24:217–31.
95. Marventano S, Vetrani C, Vitale M, Godos J, Riccardi G, Grosso G. Whole grain intake and glycaemic control in healthy subjects: a systematic review and meta-analysis of randomized controlled trials. Nutrients 2017;9(7):769.
96. Musa-Veloso K, Poon T, Harkness LS, O'Shea M, Chu Y. The effects of whole-grain compared with refined wheat, rice, and rye on the postprandial blood glucose response: a systematic review and meta-analysis of randomized controlled trials. Am J Clin Nutr 2018;108: 759–74.
97. Tighe P, Duthie G, Vaughan N, Brittenden J, Simpson WG, Duthie S, Mutch W, Wahle K, Horgan G, Thies F. Effect of increased consumption of whole-grain foods on blood pressure and other cardiovascular risk markers in healthy middle-aged persons: a randomized controlled trial. Am J Clin Nutr 2010;92:733–40.
98. Ampatzoglou A, Williams CL, Atwal KK, Maidens CM, Ross AB, Thielecke F, Jonnalagadda SS, Kennedy OB, Yaqoob P. Effects of increased wholegrain consumption on immune and inflammatory markers in healthy low habitual wholegrain consumers. Eur J Nutr 2016;55:183–95.
99. Hajihashemi P, Haghighatdoost F. Effects of whole-grain consumption on selected biomarkers of systematic inflammation: a systematic review and meta-analysis of randomized controlled trials. J Am Coll Nutr 2019;38:275–85.
100. Kazemzadeh M, Safavi SM, Nematollahi S, Nourieh Z. Effect of brown rice consumption on inflammatory marker and cardiovascular risk factors among overweight and obese non-menopausal female adults. Int J Prev Med 2014;5:478–88.
101. Hollænder PL, Ross AB, Kristensen M. Whole-grain and blood lipid changes in apparently healthy adults: a systematic review and meta-analysis of randomized controlled studies. Am J Clin Nutr 2015;102:556–72.
102. Gaesser GA. Perspective: refined grains and health: genuine risk, or guilt by association? Adv Nutr 2019;10:361–71.
103. Williams PG. Evaluation of the evidence between consumption of refined grains and health outcomes. Nutr Rev 2012;70:80–99.
104. Norén L. Loss adjusted US per capita caloric intake, 1970–2008 (data from USDA Economic Research Service) [Internet]. Minneapolis, MN: The Society Pages; 2011 [cited 10 August, 2018]. Available from: https://thesocietypages.org/graphicsociology/2011/04/11/nutrition-circles/.
105. FAO/WHO. Global and regional food consumption patterns and trends [Internet]. Rome, Italy: FAO; 2002 [cited 10 August, 2018]. Available from: http://www.fao.org/3/ac911e/ac911e05.htm.
106. Willett W. Off the cuff: Walter Willett [Internet]. Harvard School of Public Health Newsletter 2011;(Spring/Summer) [cited 10 August, 2018]. Available from: https://www.hsph.harvard.edu/news/magazine/off-the-cuff-walter-willett/.
107. Ludwig D. Always hungry? Conquer cravings, retrain your fat cells, and lose weight permanently. New York, NY; Hachette Book Group; 2016.
108. L'Chef LLC - NutriMill, The problem with processed, "refined" grain [Internet]. St. George , Utah, L'Chef [cited 10 August, 2018]. Available from: http://www.lchef.com/refined-grain/.
109. 5 Reasons why we don't recommend bread [Internet]. Mankato, Minnesota. Healthy Simple Life [cited 15 May, 2018]. Available from: https://www.healthysimplelife.com/5-reasons-why-we-dont-recommend-bread/.
110. Bjork C, 7 negative effects of refined flour, 2014 [Internet].Mankato, Minnesota, Katethehealthysimplelife [cited 10 August, 2018]. Available from: https://www.care2.com/greenliving/7-negative-effects-of-refined-flour.html.
111. Knight C. "Most people are simply not designed to eat pasta": evolutionary explanations for obesity in the low-carbohydrate diet movement. Public Underst Sci 2011;20:706–19.
112. Lindeberg S. Paleolithic diets as a model for prevention and treatment of Western disease. Am J Hum Biol 2012;24:110–15.
113. Stoler DR. Are grains destroying your health? Why grains can affect us badly and what you should know [Internet]. New York, NY: Psychology Today; 26 March, 2018 [cited 10 May, 2019]. Available from: https://www.psychologytoday.com/us/blog/the-resilient-brain/201803/are-grains-destroying-your-health.
114. Giugliano D, Maiorino MI, Bellastella G, Esposito K. More sugar? No, thank you! The elusive nature of low carbohydrate diets. Endocrine 2018;61:383–7.
115. International Food Information Council. 2018 Food and Health Survey [Internet]. Washington (DC): International Food Information Council; 2018 [cited 10 May, 2019]. Available from: https://foodinsight.org/wp-content/uploads/2018/05/2018-FHS-Report-FINAL.pdf.
116. Brown AW, Ioannidis JP, Cope MB, Bier DM, Allison DB. Unscientific beliefs about scientific topics in nutrition. Adv Nutr 2014;5(5): 563–5.
117. Cofield SS, Corona RV, Allison DB. Use of causal language in observational studies of obesity and nutrition. Obes Facts 2010;3: 353–6.
118. Ioannidis JPA. The challenge of reforming nutritional epidemiologic research. JAMA 2018;320(10):969–70.
119. Koretz RL. *JPEN* Journal Club. Association vs causation. J Parenter Enteral Nutr 2014;38:269–70.
120. Ross AB, Kristensen M, Seal CJ, Jacques P, McKeown NM. Recommendations for reporting whole-grain intake in observational and intervention studies. Am J Clin Nutr 2015;101: 903–7.
121. Ferruzzi MG, Jonnalagadda SS, Liu S, Marquart L, McKeown N, Reicks M, Riccardi G, Seal C, Slavin J, Thielecke F, et al. Developing a standard definition of whole-grain foods for dietary recommendations: summary report of a multidisciplinary expert roundtable discussion. Adv Nutr 2014;5:164–76.
122. Sawicki CM, Livingston KA, Ross AB, Jacques PF, Koecher K, McKeown NM. Evaluating whole grain intervention study designs and reporting practices using evidence mapping methodology. Nutrients 2018;10:1052.
123. US Food and Drug Administration. Health claim notification for whole grain foods: guidance for industry [Internet]. Washington (DC): US FDA; 2014 [cited 10 August, 2018]. Available from: https://www.fda.gov/food/labelingnutrition/ucm073639.htm.
124. Reedy J, Krebs-Smith SM. Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. J Am Diet Assoc 2010;110:1477–84.
125. Bachman JL, Reedy J, Subar AF, Krebs-Smith SM. Sources of food group intakes among the US population, 2001–2002. J Am Diet Assoc 2008;108:804–14.
126. Slining MM, Mathias KC, Popkin BM. Trends in food and beverage sources among US children and adolescents: 1989–2010. J Acad Nutr Diet 2013;113:1683–94.
127. Krebs-Smith SM, Guenther PM, Subar AF, Kirkpatrick SI, Dodd KW. Americans do not meet federal dietary recommendations. J Nutr 2010;140:1832–8.
128. Tester JM, Leung CW, Leak TM, Laraia BA. Recent uptrend in whole-grain intake is absent for low-income adolescents, National Health and Nutrition Examination Survey, 2005–2012. Prev Chronic Dis 2017;14:E55.
129. Whole Grains Council. Whole grain consumption worldwide [Internet]. Boston, MA: Whole Grains Council; 2017 [cited 10 August, 2018]. Available from: https://wholegrainscouncil.org/blog/2017/07/whole-grain-consumption-worldwide.
130. Livingstone MB, Pourshahidi LK. Portion size and obesity. Adv Nutr 2014;5:829–34.

131. Hetherington MM, Blundell-Birtill P, Caton SJ, Cecil JE, Evans CE, Rolls BJ, Tang T. Understanding the science of portion control and the art of downsizing. Proc Nutr Soc 2018;77(3):347–55.
132. Burgess-Champoux T, Marquart L, Vickers Z, Reicks M. Perceptions of children, parents, and teachers regarding whole-grain foods, and implications for a school-based intervention. J Nutr Educ Behav 2006;38:230–7.
133. Chu YL, Warren CA, Sceets CE, Murano P, Marquart L, Reicks M. Acceptance of two US Department of Agriculture commodity whole-grain products: a school-based study in Texas and Minnesota. J Am Diet Assoc 2011;111:1380–4.
134. Fulgoni VL, 3rd, Keast DR, Bailey RL, Dwyer J. Foods, fortificants, and supplements: where do Americans get their nutrients? J Nutr 2011;141:1847–54.
135. Hosseini SH, Papanikolaou Y, Islam N, Rashmi P, Shamloo A, Vatanparast H. Consumption patterns of grain-based foods among adults in Canada: evidence from Canadian Community Health Survey—Nutrition 2015. Nutrients 2019;11(4):784.
136. Priebe MG, McMonagle JR. Effects of ready-to-eat-cereals on key nutritional and health outcomes: a systematic review. PLoS One 2016;11:e0164931.
137. Sandstead HH, Prasad AS. Dietary whole grains and zinc nutriture. Am J Clin Nutr 2017;106:955–6.
138. Thielecke F, Nugent AP. Contaminants in grain—a major risk for whole grain safety? Nutrients 2018;10:1213.
139. Williamson DA, Bray GA, Ryan DH. Is 5% weight loss a satisfactory criterion to define clinically significant weight loss? Obesity (Silver Spring) 2015;23:2319–20.
140. Williams PG. The benefits of breakfast cereal consumption: a systematic review of the evidence base. Adv Nutr 2014;5(5): 636S–73S.
141. Villegas R, Liu S, Gao YT, Yang G, Li H, Zheng W, Shu XO. Prospective study of dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Arch Intern Med 2007;167:2310–16.
142. Hauner H, Bechthold A, Boeing H, Brönstrup A, Buyken A, Leschik-Bonnet E, Linseisen J, Schulze M, Strohm D, Wolfram G, et al. Evidence-based guideline of the German Nutrition Society: carbohydrate intake and prevention of nutrition-related diseases. Ann Nutr Metab 2012;60(Suppl 1):1–58.
143. Mohan V, Radhika G, Sathya RM, Tamil SR, Ganesan A, Sudha V. Dietary carbohydrates, glycaemic load, food groups and newly detected type 2 diabetes among urban Asian Indian population in Chennai, India (Chennai Urban Rural Epidemiology Study 59). Br J Nutr 2009;102:1498–506.
144. Mohan V, Radhika G, Vijayalakshmi P, Sudha V. Can the diabetes/cardiovascular disease epidemic in India be explained, at least in part, by excess refined grain (rice) intake? Indian J Med Res 2010;131:369–72.
145. Nanri A, Mizoue T, Noda M, Takahashi Y, Kato M, Inoue M, Tsugane S; Japan Public Health Center-based Prospective Study Group. Rice intake and type 2 diabetes in Japanese men and women: the Japan Public Health Center-based Prospective Study. Am J Clin Nutr 2010;92(6):1468–77.
146. Nanri A, Mizoue T, Yoshida D, Takahashi R, Takayanagi R. Dietary patterns and A1C in Japanese men and women. Diabetes Care 2008;31(8):1568–73.
147. Shi Z, Taylor AW, Hu G, Gill T, Wittert GA. Rice intake, weight change and risk of the metabolic syndrome development among Chinese adults: the Jiangsu Nutrition Study (JIN). Asia Pac J Clin Nutr 2012;21(1):35–43.
148. Golozar A, Khalili D, Etemadi A, Poustchi H, Fazeltabar A, Hosseini F, Kamangar F, Khoshnia M, Islami F, Hadaegh F, et al. White rice intake and incidence of type-2 diabetes: analysis of two prospective cohort studies from Iran. BMC Public Health 2017;17:133.
149. Seah JYH, Koh WP, Yuan JM, van Dam RM. Rice intake and risk of type 2 diabetes: the Singapore Chinese Health Study. Eur J Nutr 2018:1–12.
150. Soriguer F, Colomo N, Olveira G, García-Fuentes E, Esteva I, Ruiz de Adana MS, Morcillo S, Porras N, Valdés S, Rojo-Martínez G. White rice consumption and risk of type 2 diabetes. Clin Nutr 2013;32(3): 481–4.
151. Hu EA, Pan A, Malik V, Sun Q. White rice consumption and risk of type 2 diabetes: meta-analysis and systematic review. BMJ 2012;344:e1454.
152. Krittanawong C, Tunhasiriwet A, Zhang H, Prokop LJ, Chirapongsathorn S, Sun T, Wang Z. Is white rice consumption a risk for metabolic and cardiovascular outcomes? A systematic review and meta-analysis. Heart Asia 2017;9:e010909.
153. Martínez-González MÁ, de la Fuente-Arrillaga C, López-Del-Burgo C, Vázquez-Ruiz Z, Benito S, Ruiz-Canela M. Low consumption of fruit and vegetables and risk of chronic disease: a review of the epidemiological evidence and temporal trends among Spanish graduates. Public Health Nutr 2011;14(12A):2309–15.
154. Enget Jensen TM, Braaten T, Jacobsen BK, Barnung RB, Olsen A, Skeie G. Adherence to the Healthy Nordic Food Index in the Norwegian Women and Cancer (NOWAC) cohort. Food Nutr Res 2018;62:1339.
155. Kyrø C, Skeie G, Dragsted LO, Christensen J, Overvad K, Hallmans G, Johansson I, Lund E, Slimani N, Johnsen NF, et al. Intake of whole grains in Scandinavia is associated with healthy lifestyle, socio-economic and dietary factors. Public Health Nutr 2011;14:1787–95.
156. Giskes K, Turrell G, van Lenthe FJ, Brug J, Mackenbach JP. A multilevel study of socio-economic inequalities in food choice behaviour and dietary intake among the Dutch population: the GLOBE study. Public Health Nutr 2006;9:75–83.
157. Gissing SC, Pradeilles R, Osei-Kwasi HA, Cohen E, Holdsworth M. Drivers of dietary behaviours in women living in urban Africa: a systematic mapping review. Public Health Nutr 2017;20: 2104–13.
158. Moy FM, Hoe VC, Hairi NN, Buckley B, Wark PA, Koh D, Bueno-de-Mesquita HB, Bulgiba AM. Cohort study on clustering of lifestyle risk factors and understanding its association with stress on health and wellbeing among school teachers in Malaysia (CLUSTer)—a study protocol. BMC Public Health 2014;14:611.
159. Bakken T, Braaten T, Olsen A, Lund E, Skeie G. Characterization of Norwegian women eating wholegrain bread. Public Health Nutr 2015;18:2836–45.
160. Cereal Partners Worldwide (CPW). Consumers confused about how much is enough when it comes to whole grain in their diets, Switzerland, Lausanne[Internet]. CPW; 2017 [cited 15 August, 2018]. Available from: https://www.nestle.com/asset-library/documents/media/news-feed/cpw-whole-grain-press-release-nov-2017.pdf.
161. Marquart L, Wiemer KL, Jones JM, Jacob B. Whole grains health claims in the USA and other efforts to increase whole-grain consumption. Proc Nutr Soc 2003;62:151–60.
162. Mann KD, Pearce MS, McKevith B, Thielecke F, Seal CJ. Whole grain intake and its association with intakes of other foods, nutrients and markers of health in the National Diet and Nutrition Survey rolling programme 2008–11. Br J Nutr 2015;113:1595–602.
163. Chea M, Mobley AR. Factors associated with identification and consumption of whole-grain foods in a low-income population. Curr Dev Nutr 2019;3(7):nzz064.
164. Abécassis J, Cuq B, Boggini G, Namoune H. Other traditional durum-derived products. In: Sissons M, Abécassis J, Marchylo B, Carcea M, editors. Durum wheat chemistry and technology. 2nd ed. Eagan, MN: AACCI Press; 2012. p. 177–99.
165. Deschamps V, de Lauzon-Guillain B, Lafay L, Borys JM, Charles MA, Romon M. Reproducibility and relative validity of a food-frequency questionnaire among French adults and adolescents. Eur J Clin Nutr 2009;63:282–91.
166. Gottschald M, Knüppel S, Boeing H, Buijsse B. The influence of adjustment for energy misreporting on relations of cake and cookie intake with cardiometabolic disease risk factors. Eur J Clin Nutr 2016;70:1318–24.
167. Macdiarmid J, Blundell J. Assessing dietary intake: who, what and why of under-reporting. Nutr Res Rev 1998;11:231–53.

168. Michels N, De Henauw S, Beghin L, Cuenca- García M, Gonzalez-Gross M, Hallstrom L, Kafatos A, Kersting M, Manios Y, Marcos A, et al. Ready-to-eat cereals improve nutrient, milk and fruit intake at breakfast in European adolescents. Eur J Nutr 2016;55:771–9.
169. Reedy J, Krebs-Smith SM, Hammond RA, Hennessy E. Advancing the science of dietary patterns research to leverage a complex systems approach. J Acad Nutr Diet 2017;117(7):1019–22.
170. Gupta RK, Gangoliya SS, Singh NK. Reduction of phytic acid and enhancement of bioavailable micronutrients in food grains. J Food Sci Technol 2015;52:676–84.
171. Wahengbam ED, Das AJ, Green BD, Shooter J, Hazarika MK. Effect of iron and folic acid fortification on in vitro bioavailability and starch hydrolysis in ready-to-eat parboiled rice. Food Chem 2019;292: 39–46.
172. Kancherla V. Countries with an immediate potential for primary prevention of spina bifida and anencephaly: mandatory fortification of wheat flour with folic acid. Birth Defects Res 2018;110:956–65.
173. Desrosiers TA, Siega-Riz AM, Mosley BS, Meyer RE; National Birth Defects Prevention Study. Low carbohydrate diets may increase risk of neural tube defects. Birth Defects Res 2018;110:901–9.
174. US Department of Agriculture, Agricultural Research Service. Nutrient intakes from food: mean amounts consumed per individual, by gender and age. What We Eat in America, NHANES 2009–10 [Internet]. Beltsville, MD: USDA Agricultural Research Service; 2012 [cited 10 May, 2019]. Available from: http://www.ars.usda.gov/Sp2userfiles/Place/12355000/Pdf/0910/Table_1_Nin_Gen_09.Pdf.
175. Kranz S, Dodd KW, Juan WY, Johnson LK, Jahns L. Whole grains contribute only a small proportion of dietary fiber to the U.S. diet. Nutrients 2017;9(2):153.
176. Papanikolaou Y, Fulgoni VL. Grain foods are contributors of nutrient density for American adults and help close nutrient recommendation gaps: data from the National Health and Nutrition Examination Survey, 2009–2012. Nutrients 2017;9(8):873.
177. Papanikolaou Y, Fulgoni VL. Grains contribute shortfall nutrients and nutrient density to older US adults: data from the National Health and Nutrition Examination Survey, 2011–2014. Nutrients 2018;10(5):534.
178. Jones JM. CODEX-aligned dietary fiber definitions help to bridge the ‘fiber gap’. Nutr J 2014;13:34.
179. Hosseini SH, Papanikolaou Y, Isalm N, Rashmi P, Shamloo A, Vatanparast H. Consumption patterns of grain-based foods among children and adolescents in Canada: evidence from Canadian Community Health Survey-Nutrition 2015. Nutrients 2019;11(3):623.
180. Papanikolaou Y, Jones JM, Fulgoni VL, 3rd. Several grain dietary patterns are associated with better diet quality and improved shortfall nutrient intakes in US children and adolescents: a study focusing on the 2015–2020 Dietary Guidelines for Americans. Nutr J 2017;16:13.
181. Lin PH, Aickin M, Champagne C, Craddick S, Sacks FM, McCarron P, Most-Windhauser MM, Rukenbrod F, Haworth L; Dash-Sodium Collaborative Research Group. Food group sources of nutrients in the dietary patterns of the DASH-Sodium trial. J Am Diet Assoc 2003;103:488–96.
182. Castro-Quezada I, Román-Viñas B, Serra-Majem L. The Mediterranean diet and nutritional adequacy: a review. Nutrients 2014;6:231–48.
183. Schwingshackl L, Bogensberger B, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, Alternate Healthy Eating Index, Dietary Approaches to Stop Hypertension score, and health outcomes: an updated systematic review and meta-analysis of cohort studies. J Acad Nutr Diet 2018;118(1):74–100.e11.
184. Schwingshackl L, Schwedhelm C, Galbete C, Hoffmann G. Adherence to Mediterranean diet and risk of cancer: an updated systematic review and meta-analysis. Nutrients 2017;9:1063.
185. Mann KD, Pearce MS, Seal CJ. Providing evidence to support the development of whole grain dietary recommendations in the United Kingdom. Proc Nutr Soc 2017;76:369–77.
186. Seidelmann SB, Claggett B, Cheng S, Henglin M, Shah A, Steffen LM, Folsom AR, Rimm EB, Willett WC, Solomon SD. Dietary carbohydrate intake and mortality: a prospective cohort study and meta-analysis. Lancet Public Health 2018;3:e419–28.
187. Adriouch S, Lelong H, Kesse-Guyot E, Baudry J, Lampuré A, Galan P, Hercberg S, Touvier M, Fezeu LK. Compliance with nutritional and lifestyle recommendations in 13,000 patients with a cardiometabolic disease from the Nutrinet-Santé Study. Nutrients 2017;9(6):546.
188. Kovalskys I, Fisberg M, Gómez G, Pareja RG, Yépez García MC, Cortés Sanabria LY, Herrera-Cuenca M, Rigotti A, Guajardo V, Zimberg IZ, et al. Energy intake and food sources of eight Latin American countries: results from the Latin American Study of Nutrition and Health (ELANS). Public Health Nutr 2018;21:2535–47.
189. Alexy U, Zorn C, Kersting M. Whole grain in children’s diet: intake, food sources and trends. Eur J Clin Nutr 2010;64:745–51.
190. Brownlee IA, Durukan E, Masset G, Hopkins S, Tee ES. An overview of whole grain regulations, recommendations and research across Southeast Asia. Nutrients 2018;10(6):752.
191. Bellisle F, Hébel P, Colin J, Reyé B, Hopkins S. Consumption of whole grains in French children, adolescents and adults. Br J Nutr 2014;112:1674–84.
192. Sette S, D’Addezio L, Piccinelli R, Hopkins S, Le Donne C, Ferrari M, Mistura L, Turrini A. Intakes of whole grain in an Italian sample of children, adolescents and adults. Eur J Nutr 2017;56: 521–33.
193. Galea LM, Beck EJ, Probst YC, Cashman CJ. Whole grain intake of Australians estimated from a cross-sectional analysis of dietary intake data from the 2011–13 Australian Health Survey. Public Health Nutr 2017;20:2166–72.
194. Neo JE, Brownlee IA. Wholegrain food acceptance in young Singaporean adults. Nutrients 2017;9:371.
195. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition, and physical activity and the prevention of cancer: a global perspective. Washington (DC): American Institute for Cancer Research; 2007.
196. Satija A, Bhupathiraju SN, Spiegelman D, Chiuve SE, Manson JE, Willett W, Rexrode KM, Rimm EB, Hu FB. Healthful and unhealthful plant-based diets and the risk of coronary heart disease in U.S. adults. J Am Coll Cardiol 2017;70:411–22.
197. Mozaffarian D. Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. Circulation 2016;133:187–225.
198. Willett W, Rockström J, Loken B, Springmann M, Lang T, Vermeulen S, Garnett T, Tilman D, DeClerk F, Wood A, et al. Food in the Anthropocene: the EAT–Lancet Commission on healthy diets from sustainable food systems. Lancet, 2019; 393(10170): 447–92.