Seventeen-Year Associations between Diet Quality Defined by the Health Star Rating and Mortality in Australians: The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)

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ABSTRACT

Background: The Health Star Rating (HSR) is the government-endorsed front-of-pack labeling system in Australia and New Zealand.

Objectives: We aimed to examine prospective associations of a dietary index (DI) based on the HSR, as an indicator of overall diet quality, with all-cause and cardiovascular disease (CVD) mortality.

Methods: We utilized data from the national population-based Australian Diabetes, Obesity and Lifestyle Study. The HSR-DI at baseline (1999–2000) was constructed by *1*) calculation of the HSR points for individual foods in the baseline FFQ, and *2*) calculation of the HSR-DI for each participant based on pooled HSR points across foods, weighted by the proportion of energy contributed by each food. Vital status was ascertained by linkage to the Australian National Death Index. Associations of HSR-DI with mortality risk were assessed by Cox proportional hazards regression.

Results: Among 10,025 eligible participants [baseline age: 51.6 ± 14.3 y (mean $\pm$ standard deviation)] at entry, higher HSR-DI (healthier) was associated with higher consumption of healthy foods such as fruits, vegetables, and nuts, and lower consumption of discretionary foods such as processed meats and confectionery (*P*-trend < 0.001 for each). During a median follow-up of 16.9 y, 1682 deaths occurred with 507 CVD deaths. In multivariable models adjusted for demographic characteristics, lifestyle factors, and medical conditions, higher HSR-DI was associated with lower risk of all-cause mortality, with a hazard ratio (95% confidence interval) of 0.80 (0.69, 0.94; *P*-trend < 0.001) comparing the fifth with the first HSR-DI quintile. A corresponding inverse association was observed for CVD mortality (0.71; 0.54, 0.94; *P*-trend = 0.008).

Conclusions: Better diet quality as defined by the HSR-DI was associated with lower risk of all-cause and CVD mortality among Australian adults. Our findings support the use of the HSR nutrient profiling algorithm as a valid tool for guiding consumer food choices. *Curr Dev Nutr* 2020;4:nzaa157.

Introduction

An unhealthy diet characterized by excessive salt, refined sugars and carbohydrates, "energy dense and nutrient poor" discretionary foods, and insufficient intake of healthy foods such as fruit and vegetables is one of the most important modifiable risk factors for cardiovascular disease (CVD) and other noncommunicable diseases (NCDs). Globally it accounted for nearly 12 million (28% of all) premature deaths from NCDs and 274 million (18% of all) disability-adjusted life years in 2017 according to the Global Burden of Disease Study (1). Thus, strategies to improve population diet quality have become a major public health focus in an effort to reduce the health and economic burden of NCDs around the world.

An important tool that has been developed to help improve population diets is nutrient profiling. Nutrient profiling classifies or ranks foods according to their nutritional composition for reasons related to health, and has been used as the basis of a range of important public health policies such as regulation of food marketing to children, front-of-pack nutrition labeling, and food procurement standards for public institutions (2). The Health Star Rating (HSR) nutrient profiling system was introduced in 2014 by the Australian and New Zealand governments in partnership with the food industry, consumers, and public health groups as a voluntary front-of-pack labeling scheme for packaged, manufactured, and processed foods (3). As an interpretive scheme, it rates packaged foods from 0.5 (least healthy) to 5.0 (most healthy) stars on the front-of-pack to assist consumers in identifying healthier foods in the same category (4). It was derived from the Nutrient Profiling Scoring Criterion (NPSC) that was developed by Food Standards Australia New Zealand to determine whether a food is eligible to make a health claim. Scoring algorithms underlying both the HSR and NPSC originated from the UK Food Standards Agency nutrient profiling system (FSA-NPS), a tool to determine which foods are permitted to be advertised during children's television programming (5). Surveys indicate that the HSR is gradually being adopted by the food industry and trusted by consumers in Australia (6, 7) and that consumers prefer the HSR over traditional nutrition information panels on the back of the pack (8).

Although the HSR classification overall aligns with Australian food-based dietary guidelines (9, 10) and the NPSC (11), no prior studies to our knowledge have assessed if regular consumption of foods classified as healthier by the HSR system is associated with better health outcomes. The aim of the current research was therefore to examine the prospective associations of a dietary index (DI) based on the HSR, as an indicator of the overall quality of an individual's diet, with all-cause and CVD mortality. To conduct these analyses, we utilized data from the Australian Diabetes, Obesity and Lifestyle Study (AusDiab), a national population-based study of adults (12).

Methods

Study population

AusDiab is a national population-based longitudinal study established to examine the prevalence of diabetes and related risk factors in Australia (12). Using a stratified cluster sampling method, it enrolled 11,247 adults (aged ≥25 y) from 42 randomly selected census collector districts across Australia between May 1999 and December 2000. The majority of the participants (87%) were classified as Europids, and included those born in Australia, Northern Europe, Canada, the USA, and New Zealand. The AusDiab study was approved by the Human Research Ethics Committee of the International Diabetes Institute and Australian Institute of Health and Welfare, and all participants provided written informed consent. The current HSR-DI study was approved by the University of New South Wales Human Research Ethics Committee.

Baseline data collection

Validated questionnaires were administered by trained interviewers to collect information on demographic characteristics (age, sex, education, and income), medical and family history of disease (previous diagnosis of diabetes and CVD, and family history of diabetes), lifestyle factors (alcohol drinking, tobacco smoking, physical activity, and diet), and health-related behaviors. The area-level socioeconomic disadvantage was estimated using the Index of Relative Disadvantage code from the Socioeconomic Indexes for Areas (13). Physical activity was assessed using the validated Active Australia Survey that measures frequency and duration of walking, moderate activity, and vigorous activity in the last week (14). According to the guidelines for the Active Australia Survey, total physical activity time was calculated as the sum of the time spent walking (if continuous and for ≥10 min) or performing moderate activity, plus double the time spent in vigorous activity. The time spent in vigorous activity is doubled because vigorous activity is more intense and confers greater health benefits (15).

Weight was assessed using beam balance scales, whereas height was assessed using a stadiometer. Waist and hip circumferences were measured by tape measures. Blood pressure was measured using a Dinamap oscillometric blood pressure recorder (GE Healthcare) or a standard mercury sphygmomanometer with appropriate adjustments (16). Fasting plasma glucose (FPG) and serum total cholesterol were measured using an Olympus AU600 analyzer (Olympus Optical). All participants except for those currently receiving diabetes treatment or who were pregnant underwent a standard 2-h oral-glucose-tolerance test for FPG and 2-h plasma glucose (2h-PG). The 1999 WHO criteria for diabetes and hyperglycemia were used to define impaired fasting glucose (FPG ≥6.1 and <7.0 mmol/L with 2h-PG <7.8 mmol/L), impaired glucose tolerance (2h-PG ≥7.8 and <11.1 mmol/L with FPG <7.0 mmol/L), and diabetes mellitus (FPG ≥7.0 mmol/L or 2h-PG ≥11.1 mmol/L) (17). Participants who reported having doctor-diagnosed diabetes, were taking hypoglycemic medications, or met the aforementioned criteria for diabetes at baseline were classified as having prevalent diabetes. Prevalent hypertension was defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg or self-reporting of blood pressure–lowering medication. High cholesterol was defined as plasma total cholesterol >5.5 mmol/L (18).

Assessment of diet and calculation of the HSR-DI

Although HSR is currently only implemented for packaged foods in Australia, for the purpose of the current analyses we applied the HSR system to all foods (packaged and nonpackaged) in the AusDiab baseline survey to comprehensively evaluate diet quality and construct the HSR-DI. Dietary information was collected using a semiquantitative FFQ investigating food consumption over the past 12 mo, developed and validated by the Anti-Cancer Council of Victoria (19). The FFQ

contains 74 items with 10 frequency options, and additional questions on food habits, portion size, and alcoholic beverages. For each food, the 10 frequency options ranged from never to $\geq$3 times/d, whereas 7 different portion sizes could be selected from less than, equivalent to, between, or more than the 3 portion sizes represented by 3 photos. The HSR-DI was constructed using a 2-step approach: *1*) calculation of HSR points for individual foods in the FFQ, and *2*) calculation of the HSR-DI for each participant based on pooled HSR points across foods, weighted by the proportion of energy contributed by each food.

The HSR points for all 74 packaged and nonpackaged foods in the FFQ were determined according to government guidelines (4). In brief, each food was first categorized into 1 of 6 possible categories (Categories 1–3 and 1D–3D; see **Supplemental Table 1**). Then, "HSR baseline points" were assigned depending on the amounts of total energy, saturated fat, total sugars, and sodium content (per 100 g or mL) for each food, with varying baseline point values depending on the food category. Total HSR baseline points were calculated as the sum of the baseline points for each nutrient. In accordance with government guidelines, HSR modifying points were then assigned for each food, based on the percentage content of fruits, vegetables, nuts, and legumes (FVNL%) for 100 g or mL. Similarly, additional modifying points were then assigned to some foods based on the amount of protein and dietary fiber (4). HSR modifying points for protein were only awarded if a food scored <13 total baseline points, or if a food scored $\geq$13 total baseline points and $\geq$5 FVNL% points, whereas the HSR modifying points for fiber could only be scored for Category 2, 2D, 3, and 3D foods. The final HSR points were calculated by subtracting the total HSR modifying points from the total HSR baseline points.

The energy and nutrient values (saturated fat, total sugars, sodium, protein, and fiber) for each food used for the HSR points calculation were based on the NUTTAB95 food composition data (20). The FVNL% of foods were estimated through linkage to the 2017 Australian FoodSwitch Monitoring Database designed for analyses of the Australian food supply (6, 21). It contains nutrient information and ingredients lists (obtained directly from the Nutrition Information Panel) for 23,859 packaged foods in 714 categories, which were collected by trained data collectors from 1 store of each of the 4 major supermarkets (Coles, Woolworths, Aldi, and IGA) in Australia (21, 22). The FVNL% contents of foods in the FoodSwitch Monitoring Database were estimated using the ingredients list. Each food in the baseline FFQ was matched to the most similar category of food products in the FoodSwitch Monitoring Database and assigned an FVNL% by calculating the mean FVNL% across all similar products in that food category. For example, we matched "Potato fried or roasted" to "frozen potato chips" (69%), "frozen potato wedges" (69%), and "hash browns" (70%) in the "Fruit and vegetables" food category in the FoodSwitch Monitoring Database, and imputed a mean FVNL% of 69.3% for fried or roasted potatoes.

Because the range of final HSR points differs for products that fall into different food categories (i.e., −23 to 93 for Category 1 and 1D foods; −38 to 93 for Category 2 and 2D foods; and −38 to 81 for Category 3 and 3D Foods), the final HSR points were transformed to standardize their distribution into a 0–100 scale using the equation: standardized HSR points = (final HSR points − lowest category-specific final HSR points possible)/range of category-specific HSR points × 100

(8). The HSR-DI was then constructed as the weighted average (with weights determined by the percentage of energy contributed by each food) of the standardized HSR points across all foods consumed by the participant using the following formula:

$$\mathrm{HSR - DI} \ = \ \frac{\sum_{i=1}^{n} \mathrm{HSRP}_i \, \mathrm{E}_i}{\sum_{i=1}^{n} \mathrm{E}_i} \qquad (1)$$

where HSRP$_i$ is the standardized HSR points for each individual food consumed and E$_i$ is the energy intake from this food. A higher HSR-DI was indicative of a healthier diet quality. Because the HSR is not used to rate alcoholic drinks, all alcoholic beverages were excluded from estimation of the HSR-DI.

Outcome ascertainment

Vital status was ascertained by linking all participants to the Australian National Death Index (NDI), which is managed by the Australian Institute of Health and Welfare. Linkage to the NDI was performed first in May 2004 and thereafter annually up to 17 April, 2017. CVD was defined by using diagnosis codes for underlying cause of death from the WHO International Classification of Diseases 10th revision. CVD codes included I10–I25, I46.1, I48, I50–I99, or R96. When the underlying cause of death was uncomplicated diabetes (E109, E119, or E149) or unspecified hyperlipidemia (E785), CVD was considered to be the cause of death if any of the aforementioned CVD codes were the first listed cause on the death certificate, resulting in 23 such deaths. The accuracy of the NDI has been established for ascertainment of vital status and CVD deaths in a previous comparison with adjudicated fatal health outcomes (23).

Statistical analysis

We excluded 233 participants where information was missing on hypertension, high cholesterol, or diabetes at baseline. In addition, we excluded 467 participants who did not provide information on key covariates including socioeconomic status, physical activity, smoking, alcohol drinking, weight, and waist and hip circumference, or who had not fasted for $\geq$8 h before diabetes diagnosis or cholesterol testing. We also excluded 506 participants with reported implausible extreme energy intakes (<500 or >3500 kcal for women; <800 or >4000 kcal for men) (24) or who were pregnant and thus may not have been following their usual diet. We further excluded 16 participants whose mortality status could not be confirmed by the NDI. A total of 10,025 participants were included in the main analyses (**Supplemental Figure 1**).

To assess the relation between the HSR-DI and consumption of different food groups and nutrients, we examined daily intakes of foods and nutrients across HSR-DI quintiles using linear regression, adjusted for sex and age. Linear trends of consumption of food groups and nutrients across HSR-DI quintile groups were assessed by assigning participants the median value for HSR-DI in each quintile and modeling it as a continuous variable.

Person-years of follow-up were estimated from the date of enrolment to the date of death or 17 April, 2017, whichever came first. Cox proportional hazards regression models with age as the time scale were applied to assess associations of the HSR-DI with all-cause and CVD mortality and determine HRs and 95% CIs. The HSR-DI was modeled

in quintiles as indicator variables and as a continuous variable. We also examined the linear trend across HSR-DI quintiles by assigning participants the median value for HSR-DI in each quintile and modeling it as a continuous variable. Nonlinear dose response was explored nonparametrically using restricted cubic splines (25), and tests for nonlinear relation were done through the likelihood ratio test comparing the model with only the linear term to the model with the linear and cubic spline terms. There was no evidence of departure from the proportional hazards assumption as determined by the Schoenfeld residual–based test. To minimize potential confounding, covariates that are well-established risk factors for CVD and mortality were selected. Two final multivariable models were fitted with adjustment for *1*) key demographic risk factors including sex, age (time scale), and area-level socioeconomic disadvantage; and *2*) in addition BMI, waist-to-hip ratio, total energy consumption, physical activity, smoking status, alcohol drinking, prevalent hypertension, high total cholesterol, prevalent glucose tolerance status (normal glucose tolerance, impaired fasting glucose, impaired glucose tolerance, and diabetes mellitus), prevalent ischemic heart disease (IHD), and prevalent stroke. All covariates were collected from the baseline survey.

In exploratory analyses, potential effect modification (interaction) was examined for age (<65 and ≥65 y), sex (female and male), and BMI (<25.0 and ≥25.0 kg/m^2) by adding a product term between the stratified variables and HSR-DI, and statistical significance was assessed by the likelihood ratio test. We also conducted several sensitivity analyses: *1*) excluding cases of baseline IHD or stroke; *2*) excluding participants who died within the first 2 y of follow-up to minimize reverse causation; *3*) censoring at 10 y of follow-up given possible change of dietary behaviors in the long-term follow-up; and *4*) adjusting for systolic blood pressure, total cholesterol, LDL:HDL cholesterol ratio, and FPG at baseline which may be potential mediators for the relation between HSR-DI and mortality. In another sensitivity analysis for CVD mortality, competing risk modeling was applied to account for competing risk of death due to other causes (26). Statistical analyses were performed using Stata 14.0 (StataCorp LLC). All *P* values were 2-sided and statistical significance was defined as *P* < 0.05.

Results

Baseline characteristics of study participants

Of 10,025 participants included in the analysis, 4458 (44.5%) were men, and the baseline age was 51.6 ± 14.3 y [mean ± standard deviation (SD)]. The range of HSR-DI for study participants was 57.9–76.5 (mean ± SD: 67.3 ± 2.4) with an approximately normal distribution (**Supplemental Figure 2**). Compared with those in the lower quintiles, participants in the top HSR-DI quintile (indicating higher overall dietary quality) were more likely to be women, older, and to have never been smokers or drinkers (**Table 1**). They were also more likely to be from the least disadvantaged socioeconomic areas, achieve sufficient physical activity, and have lower daily energy intakes. The lower energy intake was likely due to the significantly higher proportion of women and persons of older age in this quintile than in other quintiles. However, they were also more likely to have diabetes, IHD, or stroke at baseline.

Consumption of food groups and nutrients across quintiles of HSR-DI

The consumption of core food groups as defined by the Australian Dietary Guidelines such as cereals, fruits, vegetables, nuts, legumes, fish and seafood, and milk and yogurt was higher across the quintiles of HSR-DI (*P*-trend < 0.001 for all) (**Table 2**). For example, the mean ± SD intake of total fruits, vegetables, nuts, and legumes was 268.4 ± 133.8 and 436.9 ± 205.0 g/d in the lowest and highest quintiles of HSR-DI, respectively. Conversely, discretionary food groups (energy dense and nutrient poor) such as processed meat, ice cream, and snacks were consumed less by participants in the higher HSR-DI quintiles (*P*-trend < 0.001 for all). With regards to macro- and micronutrients, HSR-DI was positively associated with energy intakes from carbohydrates and protein, consistent with higher intakes of core food groups, and also positively related to intakes of fiber, potassium, calcium, and vitamins and carotenes such as vitamin C, folate, α-carotene, and β-carotene (*P*-trend < 0.001 for all) (**Supplemental Table 2**). On the other hand, HSR-DI was inversely associated with intakes of total energy, all major classes of fats (saturated, monounsaturated, and polyunsaturated), total sugars, and sodium (*P*-trend < 0.001 for all).

Prospective associations between the HSR-DI and all-cause and CVD mortality

During a median follow-up of 16.9 y, there were 1682 deaths, of which 507 were deemed due to CVD. After adjustment for major potential confounders, higher HSR-DI was significantly associated with lower risk of overall mortality, with 20% lower risk (HR: 0.80; 95% CI: 0.69, 0.94; *P*-trend < 0.001) comparing the fifth with the first HSR-DI quintile (**Table 3**). Each 1-SD higher HSR-DI was associated with an 8% lower risk of all-cause mortality (HR: 0.92; 95% CI: 0.88, 0.97). A stronger inverse association was observed for CVD mortality, with 29% lower risk comparing the fifth with the first HSR-DI quintile (HR: 0.71; 95% CI: 0.54, 0.94; *P*-trend = 0.008). There was little evidence that the associations were nonlinear for either all-cause or CVD mortality (**Figure 1**) (*P* for nonlinearity ≥ 0.10 for both). We note that in these analyses our estimates have wide CIs toward the tails of the HSR-DI distribution, likely owing to the limited statistical power because there are few participants with these values of HSR-DI.

There was little evidence that sex, age, or BMI modified the associations of HSR-DI with all-cause mortality (*P*-interaction ≥ 0.20 for each) or CVD mortality (*P*-interaction ≥ 0.06 for each) (**Supplemental Figure 3**). The magnitude and direction of associations between HSR-DI and all-cause and CVD mortality were not materially affected in each of the following sensitivity analyses conducted: *1*) excluding baseline CVD; *2*) excluding deaths that occurred within the first 2 y of follow-up; *3*) censoring at 10 y of follow-up; and *4*) adjusting for systolic blood pressure, total cholesterol, LDL:HDL cholesterol ratio, and FPG at baseline as continuous variables (**Supplemental Tables 3–6**). Furthermore, results for CVD mortality were largely similar using competing risk modeling (**Supplemental Table 7**).

Discussion

In this large prospective cohort study of Australian adults, a higher-quality diet as defined by the HSR nutrient profiling system was

TABLE 1 Baseline characteristics of the AusDiab study participants according to quintiles of HSR-DI[1]

| | HSR-DI[2] | | | | |
	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Participants, n	2092	2038	1998	1905	1992
HSR-DI, range	57.9–65.3	65.4–66.6	66.7–67.8	67.9–69.3	69.4–76.5
Men	1096 (52.4)	1018 (50.0)	917 (45.9)	748 (39.3)	679 (34.1)
Age, y	50.7 ± 14.7	50.6 ± 14.4	51.7 ± 14.4	52.4 ± 14.2	52.8 ± 13.6
Participants in the least disadvantaged socioeconomic quintile[3]	363 (17.4)	387 (19.0)	374 (18.7)	398 (20.9)	434 (21.8)
Smoking[4]					
Never	1031 (49.3)	1101 (54.0)	1118 (56.0)	1092 (57.3)	1206 (60.5)
Former	602 (28.8)	590 (29.0)	582 (29.1)	570 (29.9)	591 (29.7)
Current	459 (21.9)	347 (17.0)	298 (14.9)	243 (12.8)	195 (9.8)
Alcohol[5]					
Never	158 (7.6)	162 (8.0)	172 (8.6)	201 (10.6)	230 (11.6)
Former	166 (7.9)	154 (7.6)	133 (6.7)	142 (7.5)	150 (7.5)
Low intake	1213 (58.0)	1184 (58.1)	1167 (58.4)	1091 (57.3)	1202 (60.3)
Moderate intake	513 (24.5)	501 (24.6)	500 (25.0)	444 (23.3)	384 (19.3)
High intake	42 (2.0)	37 (1.8)	26 (1.3)	27 (1.4)	26 (1.3)
Physical activity[6]					
Inactive	433 (20.7)	369 (18.1)	342 (17.1)	326 (17.1)	254 (12.8)
Insufficient	725 (34.7)	730 (35.8)	689 (34.5)	601 (31.6)	604 (30.3)
Sufficient	934 (44.7)	939 (46.1)	967 (48.4)	978 (51.3)	1134 (56.9)
Daily energy consumption, kcal	2107.9 ± 685.3	2087.9 ± 654.8	1994.7 ± 655.0	1890.3 ± 613.7	1679.9 ± 596.2
BMI,[7] kg/m^2	26.5 ± 4.8	27.1 ± 5.0	27.2 ± 4.8	27.1 ± 4.9	26.8 ± 5.1
Waist-to-hip ratio	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	0.8 ± 0.1
High total cholesterol (≥5.5 mmol/L)	1169 (55.9)	1153 (56.6)	1129 (56.5)	1064 (55.9)	1053 (52.9)
Prevalent hypertension	658 (31.5)	627 (30.8)	666 (33.3)	660 (34.7)	659 (33.1)
Prevalent diabetes	126 (6.0)	144 (7.1)	171 (8.6)	199 (10.5)	195 (9.8)
Prevalent IHD	71 (3.4)	56 (2.8)	76 (3.8)	85 (4.5)	96 (4.8)
Prevalent stroke	51 (2.4)	44 (2.2)	45 (2.3)	47 (2.5)	53 (2.7)

[1]Values are presented as n (%) or mean ± standard deviation unless otherwise indicated. AusDiab, Australian Diabetes, Obesity and Lifestyle Study; IHD, ischemic heart disease; HSR-DI, Health Star Rating-Dietary Index.
[2]Quintile 1 indicates the lowest dietary quality, whereas Quintile 5 indicates the highest dietary quality.
[3]Area-level socioeconomic disadvantage was estimated using the Index of Relative Disadvantage code of the Socioeconomic Indexes for Areas, which was categorized into quintiles in the final sample.
[4]Never smokers were defined as not currently smoking, and reporting smoking <100 cigarettes over their lifetime; former smokers as not currently smoking, and reporting smoking >100 cigarettes over their lifetime or smoking daily in the past; and current smokers as currently smoking daily.
[5]Alcohol drinking was categorized into never drinker, former drinker, low intake (<10 g/d), moderate intake (≥10 and <30 g/d), and high intake (≥30 g/d).
[6]Total physical activity time (exercise) was calculated as the sum of the time spent performing moderate activity (including walking) plus double the time spent in vigorous activity, and categorized into inactive (0 min/wk), insufficient (≤150 min/wk), and sufficient levels (>150 min/wk).
[7]BMI was calculated at baseline.

associated with lower risk of all-cause and CVD mortality. Associations appeared particularly strong for CVD mortality, with those in the top quintile of the HSR-DI having ∼30% lower risk. Our findings were robust to sensitivity analyses, and associations of the HSR-DI with all-cause and CVD mortality were generally consistent across subgroups defined by age, sex, and BMI.

As expected, given the underlying nutrient profiling algorithm, there were generally consistent strong associations between higher HSR-DI and lower consumption of discretionary foods and nutrients that are considered to contribute to NCD risk, and higher consumption of core foods and nutrients whose intakes are recommended by dietary guidelines (27). Given that the HSR-DI was constructed to incorporate weighting by the percentage of energy contributed from different foods, our findings suggest that both more frequent consumption and consuming more of foods that score high on the HSR (and conversely for low-HSR foods) could relate to lower all-cause and CVD mortality. These findings support the validity of the underlying nutrient profiling system used by the HSR to discriminate between healthier and less

healthy foods and beverages. The observed associations of the HSR-DI with foods and nutrients also provide biological plausibility for the observed protective associations with lower all-cause and CVD mortality risk. For instance, reducing sodium (28), increasing fruits and vegetables (29, 30), and increasing nut intake (31, 32) have been shown in trials to improve CVD risk factors such as blood pressure, endothelial function, lipid risk factors, weight gain, and markers of glucose homeostasis.

Our findings have important implications for the HSR front-of-pack label and its implementation in Australia. At present, the application of HSR on packaged foods is voluntary, i.e., at the food manufacturers' discretion. Recent monitoring evidence suggested that many food manufacturers remained reluctant to introduce the HSR on their products, such that in 2017—3 y after the initial launch of the HSR—<30% of packaged food products in Australia displayed the HSR (6). Furthermore, HSR labels were much more likely to be applied by food manufacturers to healthier foods (those that qualify for higher HSR ratings). The low prevalence of HSR labels and

TABLE 2 Food group consumption across quintiles of the HSR-DI among 10,025 Australian adults[1]

Food types	HSR-DI					P-trend[2]
	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	
Cereals and potatoes	255.5 ± 113.8	276.3 ± 126.0	284.6 ± 134.9	291.1 ± 149.4	294.8 ± 167.5	<0.001
Cereals	63.6 ± 56.4	82.3 ± 72.4	87.6 ± 78.0	97.7 ± 100.2	107.9 ± 113.4	<0.001
Breakfast cereals	41.5 ± 64.1	53.1 ± 67.8	63.5 ± 80.1	69.2 ± 85.4	71.3 ± 93.6	<0.001
Bread	114.0 ± 50.8	100.4 ± 44.4	91.0 ± 41.7	82.4 ± 42.1	78.8 ± 46.9	<0.001
Potatoes	36.4 ± 35.2	40.5 ± 36.3	42.4 ± 37.4	41.8 ± 38.7	36.8 ± 38.0	0.12
Fats and sauces	31.3 ± 13.7	27.7 ± 12.5	24.0 ± 12.0	19.3 ± 12.1	9.1 ± 9.5	<0.001
Fats	26.5 ± 12.0	22.9 ± 10.8	19.8 ± 10.6	15.4 ± 10.9	5.7 ± 8.5	<0.001
Dressings and sauce	4.8 ± 4.8	4.8 ± 4.7	4.3 ± 4.3	3.9 ± 4.1	3.4 ± 4.0	<0.001
Fruit juices	69.2 ± 105.5	82.8 ± 107.4	89.8 ± 119.9	93.7 ± 127.1	84.4 ± 136.9	<0.001
Fruits, vegetables, nuts, and legumes	268.4 ± 133.8	316.9 ± 143.9	354.7 ± 162.4	383.3 ± 179.8	436.9 ± 205.0	<0.001
Fruits	147.3 ± 108.9	183.7 ± 123.0	211.7 ± 138.8	235.5 ± 151.8	280.0 ± 167.5	<0.001
Vegetables	110.4 ± 55.2	121.2 ± 53.8	129.7 ± 56.2	133.5 ± 60.4	139.6 ± 67.2	<0.001
Nuts	3.8 ± 5.8	4.2 ± 5.9	4.6 ± 6.7	5.0 ± 7.4	5.5 ± 9.2	<0.001
Legumes	6.9 ± 10.0	7.9 ± 10.2	8.6 ± 11.3	9.3 ± 11.6	11.9 ± 15.2	<0.001
Meat, fish/seafood, and eggs	167.7 ± 93.0	184.2 ± 96.9	186.1 ± 106.4	181.2 ± 109.4	170.0 ± 124.5	<0.001
Meat	94.9 ± 63.7	108.8 ± 67.7	110.8 ± 75.7	109.5 ± 80.9	99.2 ± 90.5	<0.001
Processed meat	32.0 ± 29.5	28.1 ± 24.0	24.3 ± 22.5	20.0 ± 20.0	13.6 ± 15.3	<0.001
Fish and seafood	26.1 ± 24.3	32.1 ± 32.3	36.2 ± 35.1	37.9 ± 43.6	44.5 ± 58.2	<0.001
Eggs	14.7 ± 11.9	15.2 ± 11.7	14.7 ± 11.4	13.8 ± 11.0	12.6 ± 10.7	<0.001
Milk and dairy products	305.4 ± 154.3	363.1 ± 169.6	378.8 ± 186.4	408.6 ± 200.3	418.2 ± 219.2	<0.001
Milk and yogurt	272.6 ± 147.2	333.6 ± 162.9	353.6 ± 179.9	386.5 ± 195.9	401.9 ± 216.6	<0.001
Cheese	16.6 ± 14.5	14.2 ± 12.7	12.2 ± 10.8	10.7 ± 10.0	9.3 ± 10.0	<0.001
Ice cream	16.1 ± 25.3	15.2 ± 22.9	13.1 ± 20.3	11.3 ± 19.4	7.0 ± 14.1	<0.001
Mixed dishes	54.1 ± 52.0	51.6 ± 47.8	40.9 ± 37.9	34.2 ± 32.2	23.6 ± 24.1	<0.001
Pizza, pies, and quiche	44.5 ± 44.3	42.4 ± 41.6	33.4 ± 31.0	28.0 ± 28.2	19.4 ± 20.7	<0.001
Sandwiches	9.6 ± 18.0	9.2 ± 14.6	7.5 ± 14.2	6.2 ± 9.9	4.2 ± 8.5	<0.001
Snacks	120.0 ± 73.2	97.5 ± 57.7	82.0 ± 50.6	66.5 ± 43.9	44.7 ± 32.3	<0.001
Biscuits	20.2 ± 22.9	13.8 ± 16.8	10.1 ± 12.9	7.6 ± 10.4	4.6 ± 8.1	<0.001
Cakes	28.0 ± 35.0	22.2 ± 28.6	17.1 ± 22.3	13.9 ± 20.6	8.4 ± 12.5	<0.001
Salty snacks	33.8 ± 30.3	32.7 ± 28.1	31.0 ± 29.5	26.6 ± 25.0	19.2 ± 19.5	<0.001
Sweets	26.1 ± 20.6	21.1 ± 17.8	17.6 ± 15.7	13.7 ± 13.4	9.7 ± 11.3	<0.001
Chocolate products	11.9 ± 17.7	7.7 ± 10.4	6.3 ± 8.9	4.7 ± 7.2	2.8 ± 4.5	<0.001

[1]Values are presented as mean ± standard deviation (g/d). HSR-DI, Health Star Rating-Dietary Index.
[2]P values for trend were obtained by linear regression through assigning participants the median value for HSR-DI in each quintile and modeling it as a continuous variable, adjusted for sex and age.

selective labeling likely undermine the consumers' ability to distinguish between products and effectively use the HSR to guide their dietary choices. The observed beneficial associations of the HSR-DI with risk of CVD and all-cause mortality in our study provide important evidence supporting the validity of the HSR system and highlight the need for improved implementation, for example, requiring mandatory HSR labeling.

Some of the current criticisms of the HSR nutrient profiling system have focused on specific foods for which the HSR may misclassify their level of healthiness (33–36). Such criticisms highlight the valid concern about and difficulty in designing a "perfect" nutrient profiling system, given the complexity of the food supply and the sheer diversity of food products available. Therefore, although our results support the overall performance of the HSR system, additional studies on how changing the underlying nutrient profiling could strengthen or weaken the association of the HSR-DI with disease outcomes should be an area of future investigation, which may inform further updates to the HSR. For example, robust evidence from large observational studies and randomized controlled trials supports the cardiometabolic benefits of foods high in polyunsaturated fats (37–40). However, the current HSR nutrient pro-

filing algorithm only penalizes saturated fat, and our study found that higher HSR-DI was related to lower intake of all types of fats, indicating that further differentiating fat types in HSR scoring algorithms could further strengthen the system.

Only a handful of studies have assessed the associations of specific nutrient profiling system–defined DIs with risk of incident CVD and all-cause mortality (41–45). Our current study in Australia is most directly comparable with prior studies that have been carried out in France and the United Kingdom (43–45), because all 3 countries used similar, but not identical, nutrient profiling systems that were originally developed by the UK FSA (5). Our results are generally consistent with those from previous studies in France, including 2 large, long-term prospective cohorts (43, 44), both of which found that a higher-quality diet based on an FSA-NPS DI was associated with lower risk of incident CVD. In the NutriNet-Santé cohort, a higher diet quality as defined by the FSA-NPS DI was associated with lower risk of incident CVD, with an HR of 0.71 (95% CI: 0.54, 0.94) comparing participants in the highest with those in the lowest quartile (44). Recently, another study conducted among British adults in the EPIC (The European Prospective Investigation of Cancer)-Norfolk cohort focused only on the

TABLE 3 Associations between the HSR-DI and mortality risk among 10,025 Australian adults[1]

	Deaths	Person-years	Model 1: HR (95% CI)[2]	Model 2: HR (95% CI)[3]	P-trend[4]
All-cause mortality					
Per SD increase	1682	159,606	0.91 (0.87, 0.96)	0.92 (0.88, 0.97)	<0.001
Quintile 1	393	32,881	1	1	
Quintile 2	342	32,518	0.90 (0.78, 1.04)	0.93 (0.81, 1.08)	
Quintile 3	333	31,836	0.81 (0.70, 0.94)	0.82 (0.70, 0.95)	
Quintile 4	314	30,345	0.80 (0.69, 0.93)	0.78 (0.67, 0.91)	
Quintile 5	300	32,027	0.77 (0.66, 0.90)	0.80 (0.69, 0.94)	
CVD mortality					
Per SD increase	507	159,606	0.91 (0.83, 0.99)	0.90 (0.82, 0.99)	0.008
Quintile 1	137	32,881	1	1	
Quintile 2	93	32,518	0.72 (0.55, 0.94)	0.74 (0.57, 0.97)	
Quintile 3	90	31,836	0.63 (0.48, 0.82)	0.61 (0.47, 0.80)	
Quintile 4	96	30,345	0.71 (0.54, 0.92)	0.65 (0.49, 0.85)	
Quintile 5	91	32,027	0.71 (0.54, 0.92)	0.71 (0.54, 0.94)	

[1]CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; HSR-DI, Health Star Rating-Dietary Index; SD, standard deviation.
[2]Adjusted for sex (men and women), age (continuous, time scale), and area-level socioeconomic disadvantage (quintiles).
[3]Adjusted for sex (men and women), age (continuous, time scale), area-level socioeconomic disadvantage (quintiles), BMI (continuous; kg/m^2), waist-to-hip ratio (continuous), total energy consumption (continuous, kcal), physical activity (inactive, insufficient, and sufficient), smoking status (never, former, and current), alcohol drinking (never, former, low intake, moderate intake, and high intake), prevalent hypertension (yes and no), high total cholesterol (yes and no), prevalent glucose tolerance status (diabetes, impaired glucose tolerance, impaired fasting glucose, and normal glycemia), prevalent ischemic heart disease (yes, no, and not sure), and prevalent stroke (yes, no, and not sure).
[4]The linear trend was examined by using the median value for each quintile and fitting it as a continuous variable in the model.

association of intake of less healthy foods, as defined by the FSA-NPS, with CVD and all-cause mortality and reported generally null associations for incident CVD or CVD mortality (45). The discrepancy between our results and the EPIC-Norfolk study could be attributable to a number of factors, in particular differences in dietary assessments, i.e., calculation of a total dietary index compared with focusing only on unhealthy foods and beverages, and differences in inherent scoring algorithms between the HSR and FSA-NPS (4, 45). Overall, our study builds on and significantly adds to these previous studies by assessing the HSR-DI and mortality risk for the first time in an Australian population.

Our study has major strengths. The prospective cohort design minimized selection and recall bias. The large number of all-cause mortality outcomes provided robust statistical power. We had detailed and standardized collection of key demographic and anthropometric factors and baseline medical conditions that enabled statistical adjustment to reduce confounding. The likelihood of missed or misclassified death and CVD outcomes was reduced through linkage to the NDI to ascertain vital status. The AusDiab cohort recruited participants across diverse areas in Australia, which enables characterization of diverse dietary intakes and enhances generalizability. Conducting the study in an Australian population also contributed to the policy relevance and applicability of our findings, because the HSR has already been endorsed by the federal government and other relevant stakeholders.

Potential limitations should also be acknowledged. The HSR-DI was constructed based on an baseline FFQ in 1999–2000 that included a limited number of foods. However, the FFQ had been validated previously against other dietary instruments (19) and covered major food groups likely consumed by the Australian population. The FFQ also appears to underestimate mean energy intake by ∼6% compared with a 7-d weighted food record and estimate retinol intake poorly (19). However, retinol was not used in the calculation of the HSR-DI, and therefore

the FFQ's measurement error related to retinol is unlikely to have influenced our findings. In addition, expected variability in dietary behavior over time likely caused measurement error during follow-up, although we expect such measurement error was likely nondifferential with respect to the outcome, thus likely resulting in biases toward the null (i.e., regression dilution) (46). This should be addressed in future analyses, using repeated measures of food intakes at multiple time points, where repeat data are available. Residual confounding due to unmeasured or imprecisely measured factors cannot be excluded, and the direction in which the resulting bias would have occurred is not clear (47). For foods that were generic or mixed dishes in the FFQ, we imputed and assigned to them mean FVNL% values from similar foods in the Australian FoodSwitch Database. Although the imputation was based on a contemporary Australian database with large numbers of food items, such an approach is imperfect, and may have resulted in random errors in the estimation of the FVNL% that may contribute to attenuation of the results toward the null. There were relatively smaller number of cases of CVD subtypes in the AusDiab than of all-cause mortality, so we did not assess associations of the HSR-DI with subtypes of CVD. Finally, we were unable to conduct sensitivity analyses restricting to only packaged foods (the current intended targets of the HSR system), because the FFQ on which our HSR dietary index was calculated does not distinguish between packaged and unpackaged foods. Nevertheless, our finding, by applying the HSR to all foods, would appear to provide evidence to support recent government deliberations to also apply the HSR to minimally processed foods. Similarly in support of broadening the application of the HSR, our previous analyses using data for 1529 fast-food products from 17 major food categories suggested that the HSR appeared to perform well in distinguishing healthier from less healthy fast-food products (48).

In conclusion, a healthier diet as defined by the HSR-DI was related to a lower risk of both all-cause and CVD mortality among Australian
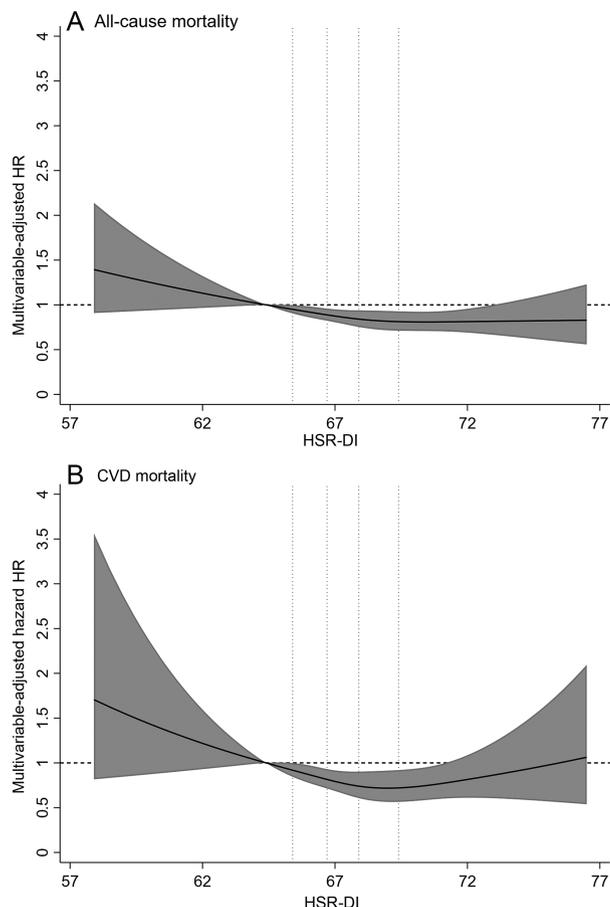
FIGURE 1 Multivariable HRs of HSR-DI for mortality, evaluated by restricted cubic splines. (A) HSR-DI was inversely associated with risk of all-cause mortality ($P = 0.006$), with little evidence for nonlinearity (P for nonlinearity = 0.34). (B) HSR-DI was inversely associated with risk of mortality from CVD ($P = 0.02$), with little evidence for nonlinearity (P for nonlinearity = 0.10). The solid lines and shaded areas represent the central risk estimates and 95% CIs, respectively, relative to the reference level (10th percentile). The dotted vertical lines correspond to the 20th, 40th, 60th, and 80th percentiles of HSR-DI. CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; HSR-DI, Health Star Rating-Dietary Index.

adults. Our findings support the overall validity of the HSR nutrient profiling algorithm in ranking the overall healthiness of foods and drinks, and corroborate calls to further strengthen its implementation in Australia as a valid tool for guiding consumer food choices.

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