



Research paper

The association between dietary exposures and anxiety symptoms: A prospective analysis of the Australian Longitudinal Study on Women's Health cohort

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ARTICLE INFO

Keywords:

Diet quality
Anxiety
Depression
ALSWH
GLAD
Nutritional psychiatry

ABSTRACT

Background: Nutrition is a potentially modifiable risk factor for mental disorders. Since anxiety is the most common mental disorder globally and most commonly impacts women, understanding its relationship with dietary exposures may elucidate important prevention and treatment approaches. We aimed to explore the association between dietary exposures and incident anxiety in women.

Methods: We used prospective longitudinal dietary data from 20,307 women enrolled in the Australian Longitudinal Study on Women's Health from 1973 to 1978 (young cohort between waves 3–5) and 1946–1951 (mid cohort between waves 3–7). Dietary exposures were defined according to the Global Burden of Disease framework (from wave 3). The primary outcome, anxiety and secondary outcome, depression were measured using subscales of the Goldberg Anxiety and Depression Scale (from wave 4). Generalized estimating equation Poisson regression models estimated risk ratios with 95 % confidence intervals, adjusting for age, socioeconomic status, and energy intake.

Results: Higher intakes of vegetables (adjusted risk ratio [aRR]: 0.94, 95%CI: 0.93–0.96 per serve increase), fruits (aRR: 0.93, 95%CI: 0.92–0.94), nuts and seeds (aRR: 0.93, 95%CI: 0.89–0.98 per serve), milk (aRR: 0.98, 95%CI: 0.96 to 0.99 per serve), fiber (aRR: 0.76, 95%CI: 0.72 to 0.81 per 30-g) and calcium (aRR: 0.88, 95%CI: 0.84 to 0.91 per gram) were associated with lower risk of incident anxiety. Conversely, higher intake of processed meat (aRR: 1.02, 95%CI: 1.00–1.05 per serve) and sodium (aRR: 1.15, 95 % CI: 1.09–1.22 per 2-g) was associated with higher anxiety risk.

Conclusions: Higher intake of plant-based foods and lower intake of processed foods may help reduce the risk of anxiety. Further prospective and intervention studies should confirm these associations and underlying biological mechanisms.

The role of dietary risk in the prevention and treatment of chronic lifestyle diseases such as cardiovascular disease (Pörschmann et al., 2024), Type II diabetes (Jannasch et al., 2017), some cancers (Srouf et al., 2019), metabolic syndrome and obesity (Rush and Yan, 2017) is well-documented. But only in the last decade has research turned its sights to the association between dietary exposures and mental health (Jacka, 2017). There is a growing body of epidemiological, longitudinal and randomized control trial (RCT) literature providing consistent

support for an association between dietary exposures and depression (Bayes et al., 2021; Lassale et al., 2018; Lee et al., 2025; Lee et al., 2023a; Lee et al., 2021; Walsh et al., 2023), while in the largest umbrella review yet conducted, higher ultra-processed food intake was associated with an increased risk of prevalent depression and combined common mental disorder outcomes (Lane et al., 2024). The mechanisms linking diet to mental health outcomes involve several interacting biological pathways. These include the modulation of pathways related to inflammation,

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<https://doi.org/10.1016/j.jad.2025.119651>

Received 13 March 2025; Received in revised form 26 May 2025; Accepted 7 June 2025

Available online 9 June 2025

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oxidative stress, epigenetics, mitochondrial dysfunction, gut microbiota, tryptophan-kynurenine metabolism, the hypothalamic-pituitary-adrenal (HPA) axis, neurogenesis, and brain-derived neurotrophic factor (BDNF) (Marx et al., 2021). Achieving a healthy diet is now included as a foundational aspect of treatment for depression in clinical practice guidelines (Malhi et al., 2015), but the association between diet quality and anxiety is under-researched (Eliby et al., 2023).

Studies have found an association between diets high in plant foods such as fruits, vegetables, seeds, nuts, whole grains, legumes, spices, herbs and ferments and a reduced risk of anxiety symptoms and an association between diets high in refined, ultra-processed and sugary snacks and beverages and increased risk of anxiety symptoms (Jacka et al., 2015; Lu et al., 2024; Opie et al., 2015; Staudacher et al., 2023). The majority of the current evidence on dietary exposures and anxiety comes from animal or cross-sectional studies (Aucoin et al., 2021). Given that anxiety is the most common mental disorder worldwide, intervention and prospective studies are needed to better infer causation, directionality, and temporality, addressing this gap in the literature.

According to the Global Burden of Disease (GBD) study - the largest epidemiological study in the world - there are 15 dietary exposures associated with physical health conditions (Murray et al., 2020) such as cardiovascular disease (Pörschmann et al., 2024) and type two diabetes (Forray et al., 2023) often comorbid with common mental health disorders such as depression and anxiety (Ferrari et al., 2022). These dietary exposures range from a low intake of fruits, vegetables, legumes, whole grains, nuts, seeds, fiber, Omega-3 polyunsaturated fatty acids, calcium and milk to a high intake of sodium, red and processed meat, sugar-sweetened beverages and trans fatty acids (Collaborators Global Burden of Disease and Årnlöv, 2020). These dietary exposures are the fifth leading cause of disability and disease burden, globally (Global Burden of Disease, 2020). GBD dietary risk data are used by governments to prioritize public health strategies. Thus using the GBD lens not only adds to the body of evidence linking diet with mental disorders but could provide evidence for global government policies to inform which diet components might be a good investment strategy for improving population-level mental health (Collaborators Global Burden of Disease and Årnlöv, 2020). Given the collective burden of dietary exposures and common mental disorders, combined with the growing evidence linking dietary exposures with depression, understanding the prospective and potentially causal role of dietary exposures with anxiety through a GBD risk factor lens is essential.

While there are several studies that assess whole-of-diet patterns and anxiety symptoms (Aucoin et al., 2021) and in women (Jacka et al., 2010), it is the aim of this study to examine the association of dietary exposures and anxiety in line with the GBD framework.

Using data from the Australian Longitudinal Study on Women's Health (ALSWH), we examined the prospective association between dietary exposures, as defined according to the GBD risk factors, and the incidence of anxiety (primary outcome) and depression (secondary outcome) in two cohorts of Australian women born between 1973 and 1978 and between 1946 and 1951.

1. Methods and materials

1.1. Participants

The Australian Longitudinal Study on Women's Health (ALSWH, 2023) is a prospective, longitudinal study of >50,000 Australian women, surveyed every three to four years since 1996. The ALSWH is separated into age cohorts - women born between (i) 1921 and 1926, (ii) 1946 and 1951, (iii) 1973 and 1978, and (iv) 1989 and 1995. Women were randomly chosen from Medicare, an Australian public health insurance database. Estimated response rates for each cohort were 37 %–40 % (1921 to 1926), 53 %–56 % (1946 to 1951), 41 %–42 % (1973 to 1978), and 70 % (1989 to 1995). The analysis for this specific study

targets the 1973 to 1978 cohort (henceforth, the “young” cohort) and the 1946 to 1951 cohort (the “mid” cohort) where anxiety, dietary intake and all covarying factors were measured. The follow-up period was 2003–2020 for the young cohort and 2001–2020 for the mid-cohort (follow-ups for each wave can be found in the supplementary material). Participants who reported a diagnosis of anxiety or depression in the three years prior to and including the year of dietary assessment were excluded from the analyses to ensure we were capturing incident cases.

1.2. Ethics and data access

The ALSWH protocol aligned with guidelines from the Declaration of Helsinki. Human Research Ethics Committees from the University of Queensland and the University of Newcastle provided formal approval for the project. All participants provided informed consent to be involved in the project. This specific analysis was approved by ALSWH (project number: A1453) and approved in March 2024 by the Deakin University Human Research Ethics Committee for exemption from ethical review (project number: 2024–085).

1.3. Exposures

Dietary intake was measured via the Dietary Questionnaire for Epidemiological Studies version 2 (DQES v2) for the young cohort at waves 3 and 5 and for the mid cohort at waves 3 and 7. The DQES v2 is a self-reported food frequency questionnaire developed by the Cancer Council Victoria to measure dietary intake in epidemiological studies (Cancer Council Victoria, 2024). The DQES v2 has been validated against weighed food records (Hebden et al., 2013). Using the DQES v2, participants self-reported dietary consumption of 74 food items over the past year, including the frequency of consuming fruit, vegetables, dairy products, meat, fish, snacks and sweets, grains, and legumes.

Our variables of interest were dietary exposures (in grams per day), as defined by the GBD study, measured at wave 3 (Murray et al., 2020). Based on the food items reported by participants in the ALSWH, 12 dietary exposures were included in these analyses – intake of fruit, vegetables, legumes, nuts and seeds, milk, red meat, processed meat, fiber, calcium, omega-3, polyunsaturated fatty acid, sodium and ultra-processed foods (Supplementary Table 1). Ultra-processed foods are not currently included in the GBD dietary exposures. However, due to increasing evidence linking intake of ultra-processed foods to detrimental mental health outcomes (Lane et al., 2024), we have included it as an additional dietary risk in this study. Ultra-processed food intake was defined following the Nova food classification system (Monteiro et al., 2019), whereby food items corresponding to Nova category four (“Ultra-processed Foods”) were classified as ultra-processed foods. The food items included in this category and their relative contribution to ultra-processed foods can be found in Supplementary Table 2 and Supplementary Fig. 1.

1.4. Outcomes

Participants completed a variety of mental health assessments at each time-point (waves 3–9), including the Goldberg Anxiety and Depression Scale (GADS), Center for Epidemiologic Studies Depression Scale - Shortened Version (CESD-10), and the Short Form Survey (SF-36).

Our primary outcome measure was anxiety (waves 4–9), as determined by the anxiety subscale of the GADS (Goldberg et al., 1988). Participants answered the first nine questions from the GADS. Each question was given a score of 0 (“No”) and 1 (“Yes”), and scores from all nine questions were summed to provide a total score on the GADS anxiety subscale. Higher scores on this tool indicate a higher level of distress. We used a binary outcome variable in these analyses, based on a cut-off score of four or more to indicate likely anxiety diagnosis (Reivan-Ortiz et al., 2019).

We included depression (waves 4–9), as identified by the CESD-10, as a secondary outcome. The CESD-10 is a commonly used tool for self-reporting depressive symptoms (Andresen et al., 1994). The CESD-10 includes eight negative mood items (including “I was bothered by things that don't usually bother me”) which are scored from 0 (“Rarely or none of the time”) to 3 (“Most or all of the time”). There are two additional positive mood items (including “I felt hopeful about the future”) where the scoring scale is reversed (i.e. 3 for “Rarely or none of the time” and 0 for “Most or all of the time”). Scores from each question are then summed to provide a total score on the CESD-10, where higher scores indicate worse symptoms. We used a cut-off score of 10 or more to indicate likely depression diagnosis (Australian Longitudinal Study on Women's Health, 2002).

In addition to CESD-10 indicated depression, we included an additional depression variable, as identified by the mental health subscale of the SF-36 (waves 4–9), in a sensitivity model. The SF-36 is a 36-item survey measuring health-related quality of life (Ware et al., 1993); however, the mental health subscale has also been evaluated as a screening tool for depression. As such, we used the cut-off score of 52 or below (where lower scores indicate poorer health-related quality of life) to categorize participants with and without likely depression (Silveira et al., 2005).

1.5. Covariates

Participants self-reported demographic information at each wave of follow-up, including age, whether they received a diagnosis of depression or anxiety in the previous three years, and various measures of socioeconomic status. Different socioeconomic variables were measured at each follow-up, such as educational attainment and household income. Given that these questions were not asked in all surveys, we included a variable outlining how well participants manage on their available income as a proxy measure for individual socioeconomic position, as recommended by the ASLWH (Egan et al., 2020). However, this may not accurately capture individual socioeconomic status, so we included an additional, area-level socioeconomic variable. Area-level socioeconomic status is often used as a proxy measure for individual socioeconomic status, but both individual- and area-level socioeconomic status independently affect health (Hastert et al., 2015; Moss et al., 2021). We used the Socio-Economic Indexes for Areas (SEIFA) in these analyses, specifically deciles of the Index of Relative Socioeconomic Advantage and Disadvantage. SEIFA scores are calculated by the Australian Bureau of Statistics based on postcodes to capture relative socioeconomic advantage and disadvantage of a given geographical area (Australian Bureau of Statistics, 2023). The two socioeconomic variables of interest were first assessed for collinearity before including both in the statistical models.

Additionally, in order to remove the potential confounding effect of energy intake, our dietary exposures were adjusted for energy using Willett's residual method and used to model our exposures (Willett et al., 1997).

1.6. Participant characteristics

Demographic characteristics were reported as mean (standard deviation) or median [quartile 1 to quartile 3] for continuous variables or n (%) for categorical variables. We also reported the percentage of participants at risk of each dietary risk, based on the GBD diet risk definitions, or the mid-point of the definitions as follows: fruit risk <325 g/day, vegetable risk <300 g/day, legume risk <95 g/day, nuts/seeds risk <14.5 g/day, milk risk <430 g/day, red meat risk >0 g/day, processed meat risk >0 g/day, fiber risk <21.5 g/day, calcium risk <1.08 g/day, omega-3 risk <450 mg/day, polyunsaturated fatty acid risk <8 % energy/day, and sodium risk >3 g/day. Ultra-processed food was not included in the analyses reporting percentage of participants at risk of the dietary variables as the GBD does not currently include definitions

for what is considered ‘at risk’.

1.7. Statistical analyses

In order to capture dietary exposures as defined by the GBD, the following analyses were conducted in line with the Global Burden of Disease Lifestyle And Mental Disorder (GLAD) project (Ashtree et al., 2025). The methods were prospectively registered on the Open Science Framework (<https://doi.org/10.17605/OSF.IO/ZBG6X>). All statistical analyses were conducted using Stata statistical analysis software, version 17.0.

We fitted a complete case model to assess the association of each dietary exposure at wave 3 with the risk of incident anxiety (primary outcome) and depression (secondary outcome) at subsequent waves. We used generalized estimating equations Poisson regression models with robust standard errors to estimate the average risk ratios across all time points (waves 4–9), accounting for the longitudinal data. Participants with a self-reported history of anxiety (when anxiety was the outcome) or depression (when depression was the outcome), including comorbid depression and anxiety, in the three years up to and including the year of their first dietary measurement were excluded from the analyses.

All model assumptions were assessed prior to analysis, then we fitted three main models for each exposure-outcome combination: 1) unadjusted, 2) adjusted for age in years, and socioeconomic status (income management and SEIFA scores as time-varying covariates, and 3) as in 2 but with additional adjustment for energy intake using Willett's residual method. The GBD definitions do not include an adjustment for energy intake, however, this is an important step in nutrition analyses (Willett et al., 1997), and so we consider model 3 to be our main model. We modelled each dietary variable continuously (per mg/day increase for omega-3, per percentage energy/day increase for polyunsaturated fatty acids, and per gram/day increase for all other dietary variables). However, to make the interpretation of the results more meaningful, we rescaled the units so that one unit in our models is approximately equivalent to one recommended Australian serving size (for food groups) or recommended daily intake (for nutrients). Where Australian recommendations were not available, we used peer-reviewed articles reporting average serving sizes or recommendations from other high-income countries. These are 150 g for fruit, 75 g for vegetables, 150 g for legumes, 30 g for nuts/seeds, 250 g for milk, 65 g for red meat (National Health and Medical Research Council (Australia), 2024b), 50 g for processed meat (National Health and Medical Research Council (Australia), 2024a), 90 g for ultra-processed foods (Clapp et al., 2018), 30 g for fiber, 1 g for calcium, 2 g for sodium (United States Department of Agriculture, 2020), 10 % energy for polyunsaturated fatty acids (National Research Council (US) Committee on Technological Options to Improve the Nutritional Attributes of Animal Products, 1988), and 1 g for omega-3 (National Institute of Health, 2024).

1.8. Sensitivity and subgroup analyses

In order to rigorously understand the associations of dietary exposures with anxiety and depression, we conducted a number of sensitivity and subgroup analyses. Results from these analyses are presented in Supplementary Material.

Firstly, to understand whether the association of dietary exposures with anxiety and depression differed by age, we include two subgroup models: (i) the two cohorts (young and mid); and (ii) GBD-defined age groups: <30, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, and 70–74 (age was assessed as a continuous variable initially). We also included a subgroup model to explore whether social functioning, based on a cut-off of 62.5 on the SF-36 social functioning subscale (Jason et al., 2011), was influencing this association.

Sensitivity models included: (i) additionally adjusting for BMI, physical activity, smoking status and marital status, (ii) additionally adjusted for self-reported diagnosis of depression or anxiety throughout

the follow-up period, (iii) excluding participants with extreme energy intake (<1st and > 99th percentile), (iv) participants with anxiety or depression at waves 3 and 4 excluded, (v) participants with anxiety or depression (respectively, not comorbidly) excluded; (vi) diet measured at all available time points as a longitudinal exposure to capture dietary change throughout the follow-up period; (vii) analyses for each wave separately to determine whether diet at wave 3 was prospectively and sustainably associated with anxiety and depression at waves 4–9; (viii) multiple imputation chained equations to account for missing data; and (ix) removing potentially influential observations, as determined by Cook's distance values greater than $(4/n-p)$; [David Sam Jayakumar and Sulthan, 2015](#)).

Finally, given the number of exposure-outcome combinations and the number of models, we included an adjustment for multiple testing based on the Simes method for p -value adjustment, which are interpreted in the same way as p -values (i.e. the standard <0.05 inference criteria applies) ([Simes, 1986](#)). Adjusted p -values were calculated separately for anxiety and depression and for the following models: 1) main models, including all levels of adjustment; 2) subgroup models - cohort, age, social functioning, and wave; and 3) sensitivity models - longitudinal diet, multiple imputation, influence diagnostics and SF-36 indicated depression.

2. Results

2.1. Demographics

At baseline (wave 3), the average age of participants was 41.4 years (27.6 years for the young cohort and 52.5 years for the mid cohort; [Table 1](#)). Overall, 11.9 % and 6.4 % of participants reported a diagnosis of anxiety or depression, respectively, in the three years prior to wave 3. Based on GBD definitions, most participants were at dietary risk ([Figs. 1 and 2](#)); for example, only 19.3 % of participants ate sufficient fruit and 95.1 % ate too much red meat according to the GBD definitions.

2.2. Diet and anxiety (primary outcome)

Eight dietary exposures at wave 3 were associated with risk of incident anxiety across waves 4–9 ([Fig. 3](#); Supplementary Table 3). After adjusting for age, socioeconomic status and energy intake, each recommended serving increment of vegetables (adjusted risk ratio [aRR]: 0.94, 95%CI: 0.93 to 0.96 per 75-g increase), fruits (aRR: 0.93, 95%CI: 0.92 to 0.94 per 150-g increase), nuts and seeds (aRR: 0.93, 95%CI: 0.89 to 0.98 per 30-g increase), milk (aRR: 0.98, 95%CI: 0.96 to 0.99 per 250-g increase), each recommended daily intake increment of fiber (aRR: 0.76, 95%CI: 0.72 to 0.81 per 30-g increase) and calcium (aRR: 0.88, 95%CI: 0.84 to 0.91 per gram increase) were associated with lower risk of anxiety. Conversely, each recommended serving and daily intake increment of processed meat (aRR: 1.02, 95%CI: 1.00 to 1.05 per 50-g increase) and sodium (aRR: 1.15, 95%CI: 1.09 to 1.22 per 2-g increase) were associated with increased risk of anxiety. We found no association of legume intake, red meat intake, polyunsaturated fat intake, omega-3 intake, or ultra-processed food intake with risk of anxiety after adjusting for age, socioeconomic status or energy.

Results were consistent when missing data were imputed, with the exception of legume and red meat intake. After imputing missing data, higher legume intake (aRR: 0.93, 95%CI: 0.87 to 0.99 per 150-g increase) and red meat intake (aRR: 0.99, 95%CI: 0.98 to 1.00 per 65-g increase) were associated with lower risk of anxiety (Supplementary Table 5). Inferences were consistent for all other sensitivity models, including when excluding participants with anxiety not co-morbid with depression (Supplementary Table 4) and when using diet as a time-varying exposure (Supplementary Table 5). The magnitude and direction of point estimates were consistent in all subgroup models (Supplementary Tables 6–8), including when analyzing anxiety separately by wave (Supplementary Table 9). However, the strength of evidence was

Table 1

Participant demographic characteristics at wave 3 (used as the baseline for these analyses).

	Whole Cohort (n = 20,307)	Young Cohort (n = 9081)	Mid Cohort (n = 11,226)
Participant age (years)	41.4 (12.5)	27.6 (1.5)	52.5 (1.5)
SEIFA index of Socio-economic Advantage & Disadvantage (decile)	5.5 (2.9)	5.5 (2.9)	No observations
Able to manage on income available			
It is impossible	277 (1.4 %)	110 (1.2 %)	167 (1.5 %)
It is difficult all the time	2059 (10.2 %)	972 (10.8 %)	1087 (9.8 %)
It is difficult some of the time	5743 (28.5 %)	2720 (30.1 %)	3023 (27.3 %)
It is not too bad	8288 (41.2 %)	3468 (38.4 %)	4820 (43.5 %)
It is easy	3757 (18.7 %)	1764 (19.5 %)	1993 (18.0 %)
Current depression at wave 3	2388 (11.9 %)	1125 (12.6 %)	1263 (11.4 %)
Current anxiety at wave 3	1290 (6.4 %)	545 (6.1 %)	745 (6.7 %)
Co-morbid depression and anxiety at wave 3	729 (3.6 %)	334 (3.7 %)	395 (3.5 %)
SF36 mental health subscale score of 52 or below	72.3 (17.8)	70.3 (17.3)	73.8 (18.0)
GADS anxiety subscale score of four or more	4.8 (2.7)	4.8 (2.7)	No observations
CESD-10 score of ten or more	6.5 (5.3)	7.0 (5.3)	6.0 (5.3)
BMI (kg/m ²)	25.96 (5.59)	24.80 (5.51)	26.85 (5.48)
Physical Activity (MET minutes/week)	599.4 [199.8–1265.4]	699.3 [266.4–1398.6]	499.5 [133.2–1198.8]
Marital Status			
Currently or Formerly Married	14,916 (73.5 %)	4082 (45.0 %)	10,834 (96.5 %)
Still Married	–	–	9104 (81.1 %)
Separated/Divorced	–	–	1391 (12.4 %)
Widowed	–	–	339 (3.0 %)
Never married	5284 (26.0 %)	4945 (54.5 %)	339 (3.0 %)
Missing	107 (0.5 %)	54 (0.6 %)	53 (0.5 %)
Smoking Status			
Never Smoked	12,023 (59.2 %)	5171 (56.9 %)	6852 (61.0 %)
Former Smoker	4383 (21.6 %)	1674 (18.4 %)	2709 (24.1 %)
Current Smoker	3804 (18.7 %)	2210 (24.3 %)	1594 (5.3 %)
Missing	97 (0.5 %)	26 (0.3 %)	71 (0.6 %)
Fruit (g/day)			
GBD risk definition <310-340 g/day	153.2 [86.9–272.1]	127.8 [72.0–242.6]	194.4 [103.4–297.9]
Vegetable (g/day)			
GBD risk definition <280-320 g/day	93.2 [66.6–127.7]	88.4 [61.0–122.4]	97.3 [71.4–131.8]
Legumes (g/day)			
GBD risk definition <90-1000 g/day	22.4 [12.1–36.0]	20.1 [9.9–34.2]	24.2 [14.2–37.3]
Nuts and Seeds (g/day)			
GBD risk definition <10-19 g/day	1.7 [0.5–4.6]	1.5 [0.5–3.9]	1.7 [0.5–5.3]
Milk (g/day)			
GBD risk definition <360-500 g/day	200.0 [200.0–375.0]	200.0 [200.0–375.0]	200.0 [200.0–375.0]
Red Meat (g/day)			
GBD risk	49.4 [25.1–83.0]	46.2 [22.4–79.3]	51.3 [27.9–86.0]

(continued on next page)

Table 1 (continued)

	Whole Cohort (n = 20,307)	Young Cohort (n = 9081)	Mid Cohort (n = 11,226)
definition >0 g/day			
Processed Meat (g/day)			
GBD risk definition >0 g/day	13.7 [5.7–26.1]	15.4 [6.4–29.4]	12.5 [5.4–23.2]
Fiber (g/day)			
GBD risk definition <21–22 g/day	18.2 [14.0–23.5]	17.5 [13.5–22.6]	18.9 [14.5–24.2]
Calcium (g/day)			
GBD risk definition <1.06–1.1 g/day	0.8 [0.6–1.0]	0.8 [0.6–1.0]	0.8 [0.6–1.0]
Omega 3 Fatty Acids (mg/day)			
GBD risk definition <430–470 mg/day	0.3 [0.2–0.5]	0.3 [0.1–0.5]	0.3 [0.2–0.5]
Polyunsaturated Fatty Acids (% energy)			
GBD risk definition <7–9 % energy//day	5.1 [3.8–6.6]	4.8 [3.7–6.2]	5.4 [4.0–6.9]
Sodium (g/day)			
GBD risk definition >1–5 g/day	2.1 [1.6–2.7]	2.2 [1.7–2.8]	2.0 [1.6–2.5]
Ultra-processed food (g/day)	323.3 [227.2–454.8]	341.6 [238.2–477.2]	310.8 [219.7–434.7]
Energy (excluding alcohol; kJ/day)	6397.1 [5065.1–8036.1]	6513.4 [5102.2–8315.8]	6295.4 [5032.8–7811.9]

altered for many of these subgroup models, where reduced sample sizes led to confidence intervals that contained null values.

2.3. Diet and depression (secondary outcome)

After adjusting for age, socioeconomic status and energy intake, each recommended serving increment of vegetables (aRR: 0.82, 95%CI: 0.79 to 0.86 per 75-g increase), fruits (aRR: 0.86, 95%CI: 0.83 to 0.89 per 150-g increase), nuts and seeds (aRR: 0.76, 95%CI: 0.65 to 0.89 per 30-g increase), each recommended daily intake increment of fiber (aRR: 0.43, 95%CI: 0.37 to 0.49 per 30-g increase) and calcium (aRR: 0.68, 95%CI: 0.62, 0.75 per 1-g increase) were associated with lower risk of depression throughout the follow-up period. In contrast, each recommended serving and daily intake increment of processed meat (aRR: 1.11, 95%CI: 1.06 to 1.16 per 50-g increase) and sodium (aRR: 1.42, 95%CI: 1.24 to 1.63 per 2-g increase) were associated with an increased risk of depression (Fig. 4; full results Supplementary Table 10). Inferences were consistent in all sensitivity models (Supplementary Tables 11 and 12). Magnitudes and directions were consistent in all subgroup models, but subgroup models were often underpowered to detect associations (Supplementary Tables 13–16).

3. Discussion

Higher intake of vegetables, nuts, seeds, legumes, fiber, milk, and calcium were associated with a reduced risk of incident anxiety and depression, whereas higher intake of processed meat and sodium, was linked to an increased risk of these conditions. This study examined the association between dietary exposures and anxiety and depression in two cohorts of Australian women born between 1973 and 1978 and between 1946 and 1951. According to the 2019 GBD-defined dietary exposures, overall, both cohorts were eating higher than recommended red and processed meat, and lower than recommended fruit, vegetables,

legumes or fiber. This aligns with other prospective research that found only 4 % of women were consuming the recommended dietary allowance of two fruits and five vegetables daily and that lower consumption was associated with increased risk of depressive symptoms (Kalmpourtzidou et al., 2020; Lee et al., 2023b).

The prospective nature of this study is a notable strength due to the largely cross-sectional nature of current evidence relating to diet and anxiety. This is particularly important with anxiety due to the potential for reverse causality. People with generalized anxiety might be more health-conscious and attentive to their diet (Musa, 2022) and because of this, cross-sectional epidemiological studies might not find clear associations between diet and anxiety as the findings may be clouded by individuals who eat well due to their symptoms or obscure the effects of poor diet in those who have recently developed symptoms of anxiety. This inconsistent pattern was observed in a study of 267 mothers in the United States (Trude et al., 2020), which found that diet quality and anxiety scores varied over time ($b = 0.28$, $p = .03$; Trude et al., 2020). It also found evidence for an inverse association of lower diet quality and anxiety at time one ($b = -0.71$, 95 % CI $[-1.09, -0.34]$), and time two ($b = -0.51$, 95 % CI $[-0.97, -0.05]$), but not at time three ($b = -0.14$, 95 % CI $[-0.54, 0.26]$). In contrast, when using a prospective design, 126,819 participants were studied from the UK Biobank between 2006 and 2010 and follow-up between 2011 and 2012. They found that participants with a high intake of chocolate, candy, added sugars, high-fat cheese, and butter and a low intake of fruits and vegetables had increased odds of anxiety symptoms (Chen et al., 2023). Although our results and the results from the UK Biobank are consistent, and RCTs (Jacka et al., 2017) indicate the potential causal role of diet with anxiety, further prospective and intervention studies are needed to confirm these findings and to elucidate the mechanisms of action, specifically through the lens of GBD risk factors.

The relationship between diet and anxiety may be explained by biological mechanisms of action, such as micro-nutrient deficiencies that may affect brain health, anti-inflammatory properties of diet, and modulation of the gut-microbiome-brain axis (Marx et al., 2021). Since chronic low-grade inflammation and oxidative stress have been implicated in several chronic diseases, including common mental disorders, reducing inflammation and oxidative stress through diet may also improve anxiety (Marx et al., 2017). Diets rich in plant foods are also high in polyphenols (secondary metabolites of plants) that have been shown to protect against the development of many health conditions via their anti-inflammatory properties and may, therefore, help reduce inflammation and decrease anxiety (Norwitz and Naidoo, 2021).

This is also true for our findings on dietary exposures and depression. Research indicates that higher intakes of fiber-rich plant foods (and lower intakes of meat and sodium) are also known to reduce risk of depression through biological mechanisms of action such as the gut microbiome/brain connection (Winter et al., 2018), brain health, anti-inflammatory properties (Jacka, 2017) and micro-nutrient deficiencies (Stevens et al., 2018). A growing body of evidence indicates that the gut microbiome likely influences the development of mental disorders (McGuinness et al., 2024) via the gut-brain axis (Cryan et al., 2019). Foods high in fiber, such as plant-based foods, have been shown to influence gut-microbiome composition (McDonald et al., 2018), which has in turn been shown to reduce symptoms of anxiety and depression (Deans, 2017). This is of particular interest in the context of our results, as we identified that fruit and vegetable intake reduced risk of anxiety and that fiber, specifically, had a large effect in lowering risk of anxiety. This may support the role of high-fiber diets in improving mental health via the gut microbiome pathway; however, further research is needed to elucidate this as a potential biological pathway.

To our knowledge, this was the first prospective study examining dietary risk factors, as defined by the GBD, and anxiety with access to a large, representative study of Australian women. However, several limitations must be considered. Firstly, though our study used a representative sample of Australian women, the Australian-only and female-

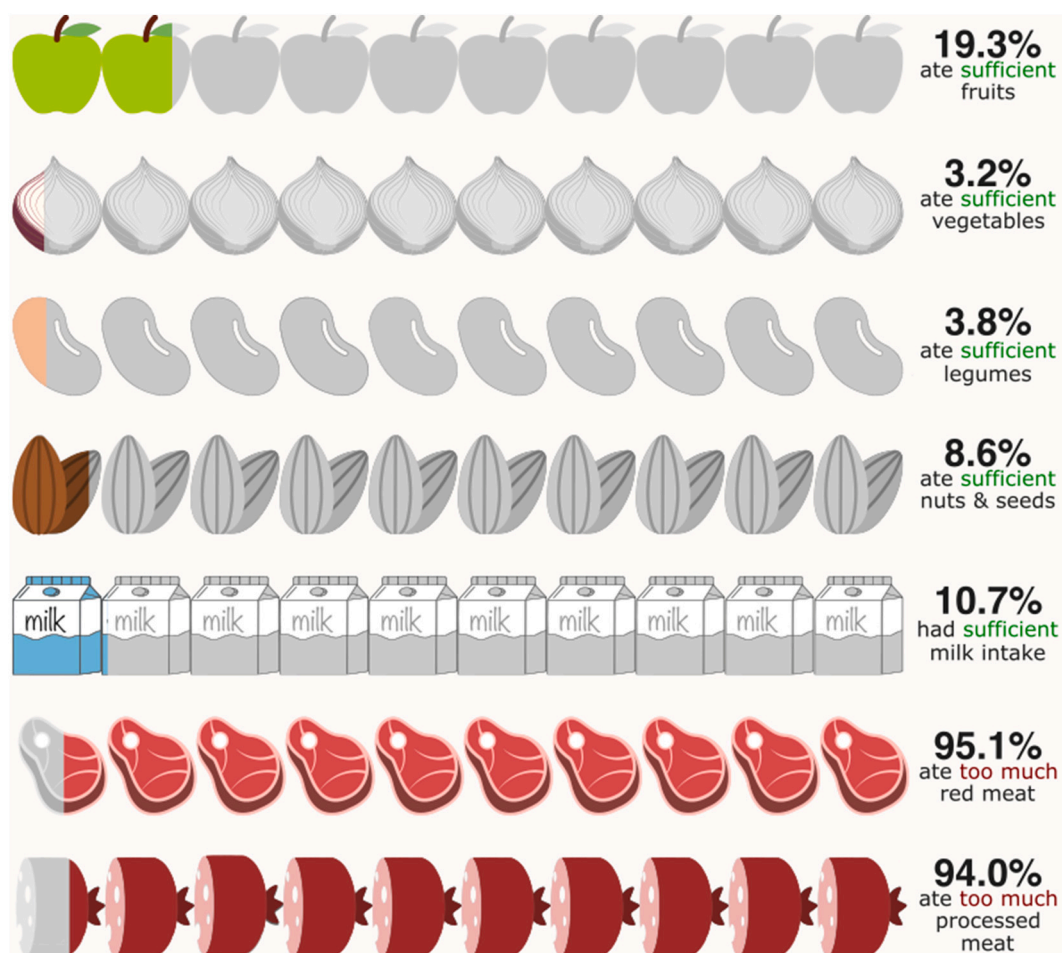


Fig. 1. Percentage of people with reported dietary intake within or above each GBD-defined dietary risk factor for food groups (UPF was not included, as the GBD do not currently define this as a risk factor). Where the risk factor definition included a range, we used the midpoint of the range as the cut-off. For example, diet low in fruit is defined as intake below 310–340 g per day, and so the percentages are based on a dietary intake below 325 g per day.

only population is also a limitation of this study, as findings may not be representative of other countries or genders. The study relied on self-reported nutrition and mental health data, so memory recall and social desirability bias (under or over-reporting un/healthy behaviors to appear more acceptable) may have influenced the results. The potential confounding effect of severe food allergies, leading to heightened anxiety and subsequent food avoidance, could not be accounted for and may have influenced the observed associations. Future studies should consider assessing clinically diagnosed food allergies and related avoidance behaviors to better disentangle their potential role in the relationship between diet and anxiety. Estimating nutrient intakes, particularly sodium, via FFQs is known to be challenging due to day-to-day variability and difficulty recalling discretionary salt use. Although the FFQ used in this study (DQES v2) has shown acceptable validity for sodium intake compared to weighed food records and other FFQs (Hodge et al., 2000), measurement error remains a possibility and may have obscured observed associations. Finally, the current GBD risk factors focus on food components rather than a whole-of-diet approach, which may not represent how dietary patterns are consumed (Pollan, 2008). Although our analyses focused on individual foods and nutrients, the collective findings may reflect broader dietary patterns, which were not formally assessed in this study. While examining individual components may underestimate the broader implications of whole-of-diet patterns, identifying specific dietary exposures most strongly associated with mental health can help pinpoint priority areas for improving population-level outcomes.

The findings of this study have several other important implications.

Firstly, future researchers could focus on the impact of whole-of-diet patterns on anxiety symptoms, particularly RCT and longitudinal study designs that could assess causality, directionality and temporality, as well as unpacking the relationship between dietary behaviors and anxiety. Secondly, studies can use these results to guide further investigations into the mechanistic actions of micro- and macronutrients in inflammation, oxidative stress and gut-brain communication.

This study highlights the association between GBD dietary exposures and incident anxiety and incident depression in Australian women. Higher intake of vegetables, fruits, nuts and seeds, milk, fiber, and calcium were associated with decreased anxiety and depression while higher intake of processed meats and sodium were associated with an increased risk. These findings underscore the importance of dietary intervention in mental healthcare and suggest that improving diet risk factors could be a safe and cost-effective strategy for reducing anxiety and depression symptoms.

Ethical standards disclosure

This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all the procedures involving research study participants were approved by the Human Research Ethics Committees of The University of Newcastle and The University of Queensland. Written informed consent was obtained from all participants.



Fig. 2. Percentage of people with reported nutrient intake within or above each GBD-defined nutrient risk factor. Where the risk factor definition included a range, we used the midpoint of the range as the cut-off. For example, diet low in fiber is defined as intake below 21–22 g per day, and so the percentages are based on a dietary intake below 21.5 g per day.

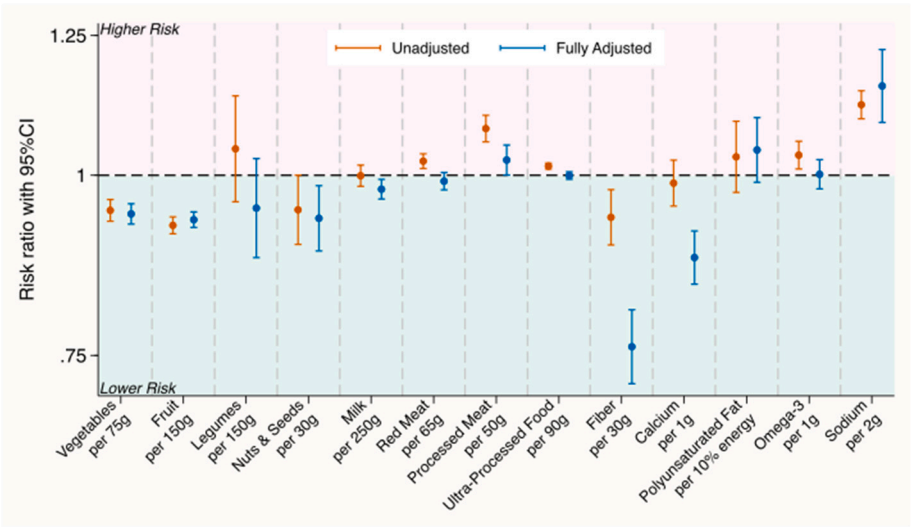


Fig. 3. Association of each dietary variable with risk of anxiety in the whole cohort in the unadjusted model (orange) and after adjusting for age, SES and energy (blue). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

CRedit authorship contribution statement

Megan F. Lee: Writing – review & editing, Writing – original draft, Software, Resources, Project administration, Methodology, Investigation, Conceptualization. **Rebecca Orr:** Writing – review & editing, Investigation, Conceptualization. **Wolfgang Marx:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Felice N. Jacka:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Adrienne O’Neil:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Melissa M. Lane:** Writing – review & editing, Validation, Software, Resources, Project

administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Deborah N. Ashtree:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of Generative AI and AI-assisted technologies in the writing process

Generative AI was not used to write or reproduce any section of this manuscript.

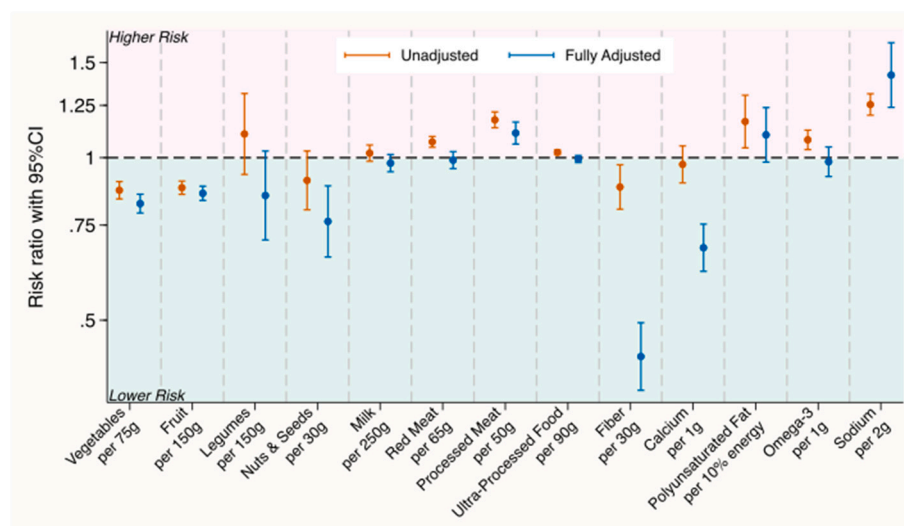


Fig. 4. Association of each dietary variable with risk of depression in the whole cohort in the unadjusted model (orange) and after adjusting for age, SES and energy (blue). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Declaration of competing interest

This work was supported by a National Health and Medical Research Council Emerging Leader 2 Fellowship (grant #2009295 to AON). MFL is the Secretary of the International Society of Nutritional Psychiatry Research. FNJ is supported by a National Health and Medical Research Council Leader 1 Fellowship (grant #1194982). DNA is supported by a National Health and Medical Research Council Fellowship (#2009295). MML is supported by a Deakin University Alfred Deakin Postgraduate Fellowship and is secretary for the Melbourne Branch Committee of the Nutrition Society of Australia (unpaid). MML has received travel funding support from the International Society for Nutritional Psychiatry Research, the Nutrition Society of Australia, the Australasian Society of Lifestyle Medicine, and the Gut Brain Congress and is an associate investigator for the MicroFit Study, an investigator-led randomized controlled trial exploring the effect of diets with varying levels of industrial processing on gut microbiome composition and partially funded by Be Fit Food (payment received by the Food and Mood Centre, Deakin University). RO is supported by a Deakin University Postgraduate Research Scholarship. WM is currently funded by an NHMRC Investigator Grant (#2008971) and is the President of the International Society of Nutritional Psychiatry Research.

The opinions, methods, and conclusions reported in this paper are those of the authors and are independent from the funding sources. This manuscript has been prepared in accordance with the requirements of the GLAD Taskforce, as part of a global collaborative project to inform the Global Burden of Diseases, Injuries, and Risk Factors Study.

Acknowledgments

The research on which this paper is based was conducted as part of the Australian Longitudinal Study on Women's Health by the University of Queensland and the University of Newcastle. We are grateful to the Australian Government Department of Health and Aged Care for funding and to the women who provided the survey data. The authors would like to acknowledge the Australian Longitudinal Study on Women's Health for access to their data sets; the support of Professor Gita Mishra the ALSWH liaison person for our study and Professor Graham Giles and Professor Roger Milne of the Cancer Epidemiology Centre of Cancer Council Victoria, for permission to use the Dietary Questionnaire for Epidemiological Studies (Version 2), Melbourne: Cancer Council Victoria, 1996. We would like to acknowledge all GLAD project members, including the GLAD Project Team based at Deakin University, the GLAD

Advisory Group, and the GLAD Working Group comprised of all Member Studies. Open access publishing facilitated by Bond University, as part of read and publish agreement via the Council of Australian University Librarians.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jad.2025.119651>.

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