

Associations between diet quality and DSM-IV mood disorders during young- to mid-adulthood among an Australian cohort

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Abstract

Purpose: Many studies have reported associations between diet and depression, but few have used formal diagnoses of mood disorder as the outcome measure. We examined if overall diet quality was associated cross-sectionally or longitudinally with DSM-IV mood disorders among an adult cohort.

Methods: Participants from the Australian Childhood Determinants of Adult Health study were followed up during 2004-06 (n=1974, age: 26-36 years), 2009-11 (n=1480, 31-41 years), and 2014-19 (n=1191, 36-49 years). Dietary Guidelines Index (DGI) scores were calculated from food frequency questionnaires at each time-point (higher DGI reflects better diet quality). DSM-IV mood disorders (dysthymia or depression) during the periods between, and 12 months prior to each follow-up were determined using the Composite International Diagnostic Interview. Sex-stratified risk and prevalence ratios (PR) and 95% confidence intervals (CI) were estimated using log binomial regression. Covariates included age, self-perceived social support index score, marital status, parenting status, education, occupation, physical activity, BMI, and usual sleep duration.

Results: A 10-point higher DGI was cross-sectionally associated with lower prevalence of mood disorders at the third follow-up only (females: PR=0.73, 95% CI=0.56,0.95; males: PR=0.72, 95% CI=0.53,0.97), but was attenuated after covariate adjustment (females: PR=0.92, 95% CI=0.73,1.16; males: PR=0.92, 95% CI=0.69,1.22). Adjustment for social support in the final model had attenuated the association for both sexes from 18% reduced prevalence to 8%. DGI scores were not longitudinally associated with mood disorder risk.

Conclusions: Crude cross-sectional associations between diet quality and mood disorders at ages 36-49 years were explained by sociodemographic and lifestyle factors, particularly social support.

Introduction

Numerous studies have examined associations between diet and mood disorders. Recent systematic reviews consolidating evidence from both cross-sectional and longitudinal studies [1,2], and longitudinal studies only [3,4], have reported that better adherence to a healthy dietary pattern (generally a diet high in minimally processed foods) was associated with lower risk of depressive symptoms among adults. These reviews primarily focused on studies that assessed overall diet quality using patterns or dietary indices, rather than individual nutrients. Composite score methods are now commonly used to assess overall diet as foods and their different nutritional components may have complex and synergistic effects on the bodily and microbiotic processes that support good health, including neurobiological functions required for good mental health [1,5].

The reviews were cautious in their findings and highlighted limitations arising from the heterogeneity of the outcome measures used (mainly scales of depressive symptoms with few studies using outcomes determined by diagnostic criteria), and likely bias and residual confounding [1-4]. Diagnostic mood disorder measures such as structured diagnostic interviews, provide a standardised measure of the potential impact of the condition on the individual and are generally considered higher quality measures compared to depression symptom scales, which can vary in interpretation, have different underlying constructs, and can limit generalisability of results and evidence consolidation [6,7]. We have identified only two cohorts that have been used to study the prospective association among adults in the general population between overall diet and mood disorders, and in both cases the measure was self-report of physician diagnosis or use of anti-depressant medication [8,9].

The Australian Childhood Determinants of Adult Health (CDAH) study [10] offers a unique opportunity to explore the relationship between diet and DSM-IV mood disorders determined via structured diagnostic interviews. Our primary aim was to examine if diet quality was cross-

sectionally and longitudinally associated with mood disorders in adulthood. Three follow-ups during young to mid-adulthood allowed examination of the relationship between these measures over time, during the age range when mental disorders comprise a particularly high proportion of the non-fatal burden of disease worldwide [11]. We hypothesised that better diet quality would be associated with a lower risk of mood disorder. Secondary aims were to explore reverse causality and examine the contribution of dietary components and covariates to any observed associations.

Methods

Participants

The study sample comprised participants of the CDAH cohort study (**Figure 1**). In 1985, the Australian Schools Health and Fitness Survey (ASHFS) was conducted by the Australian Council for Health, Physical Education and Recreation Inc. to provide a snapshot of the health and fitness of schoolchildren. A two-stage probability design aimed for a nationally representative sample of 500 students of each sex at ages 7-15 years to allow estimates from the physical tests and questionnaire data that would be at least within 10% of the population means. The school response rate was 90% (109 of the 121 approached schools), and the student response rate was 68% ($N = 8,498$ out of 12,578 approached students).

During 2001-02, 6840 ASHFS participants (80%) were traced and invited to participate in the CDAH study. During 2004-06, 3967 (47%) of the original ASHFS participants took part in the first follow-up (CDAH-1), with 2410 attending face-to-face study clinics. During 2009-11, the CDAH-2 follow-up involved 3036 participants (36% of ASHFS) and comprised postal questionnaires and telephone interviews. In 2014, a pilot study for CDAH-3 was held, followed

in 2017-19 by CDAH-3, with a total (including pilot participants) of 2074 participants (24% of ASHFS). At CDAH-3, 1567 participants attended study clinics.

Ethical approval

All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. The State Directors General of Education approved the ASHFS, and signed parental consent was required for all participants. The Southern Tasmanian Health and Medical Ethics Committee approved the CDAH study protocol, and all participants gave informed written consent.

Dietary Guidelines Index (DGI)

Dietary data were collected using food frequency questionnaires (FFQ) and food habits questionnaires (FHQ) based on a validated FFQ designed for Australian populations [12,13]. The FFQ included 127 items at CDAH-1 and was expanded to 128 items at CDAH-2 and 131 items at CDAH-3 to capture commonly consumed foods. The multiple choice FFQ asked respondents about their average daily, weekly or monthly consumption of food or drink items during the previous 12 months. For seasonal foods, participants were instructed to estimate intake frequency when the food was in season. Nine response options were given, ranging from “Never or less than once a month” to “6+ times per day”. It was assumed one frequency equalled one serve, and frequencies for each food item were converted into daily servings [14]. For example, if fresh fish was reported as consumed once per week, then the participant was recorded as consuming a 1/7 serve of fish per day. Usual daily servings of fruits and vegetables were derived from the FHQ which provided examples of serving sizes, with options ranging

from “I don’t eat vegetables” or “I don’t eat fruit”, to “6 or more serves”. The FHQ asked about the usual type of milk consumed (e.g. reduced fat, soy), whether the participant usually trimmed fat from meat, and the usual type of spread (e.g. polyunsaturated margarine, butter). Diet quality was calculated at each time-point using a 100-point Dietary Guidelines Index (DGI) that reflected the nutritional recommendations in the 2013 Australian Dietary Guidelines [15,16]. The DGI has been validated among the CDAH cohort by examining construct validity, including concurrent criterion validity, and was found to be an appropriate measure of diet quality [15]. The DGI comprises nine components. Seven components, each worth 10 points, reflected guidelines for having a varied diet, drinking plenty of water, and achieving minimum intakes of vegetables, fruit, grains, lean meats or alternatives, and dairy or alternatives. Two components reflected guidelines to limit intake of discretionary items (processed items high in saturated fat, added sugars, salt, or alcohol, equal to 20 points), and to replace saturated fat with unsaturated fat (10 points). Component scores were calculated from usual daily servings derived from the FFQ and FHQ responses. The DGI scoring matrix is provided in the supplementary material, **Table S1**. A higher score on the range 0-100 indicated better diet quality.

Mood Disorder

The World Health Organization’s Composite International Diagnostic Interview (CIDI-Auto) [17] was used to determine prevalence of DSM-IV dysthymic disorder and major depressive disorder (mild, moderate or severe) [18] during the 12 months prior to CDAH-1, and during the lifetime and the 12 months prior to CDAH-2 and CDAH-3. At CDAH-1 and CDAH-3, participants completed the CIDI on computers at study clinics. At CDAH-2 the interview was administered by telephone interviewers trained by the Director of the WHO CIDI Australasian

Training and Resource Centre, who provided oversight of the CIDI data collection and application of the scoring syntax. Data included age of first onset of symptoms (for mood disorders 12-months prior to CDAH-1 and lifetime mood disorder at CDAH-2 and CDAH-3), and age and recency of the last recurrence. For cross-sectional analyses, participants were categorised as having or not having a mood disorder during the 12 months prior to follow-up. For the longitudinal analyses, participants were categorised as either having a mood disorder (first onset or recurrence), or not having a mood disorder during the follow-up period. To determine if diet was associated with first onset of mood disorder, a second longitudinal outcome variable excluded participants who had experienced a prior mood disorder, leaving only participants who had never had a mood disorder, or had their first mood disorder during the follow-up period. To examine reverse causality, age of most recent mood disorder from the CDAH-2 and CDAH-3 lifetime CIDs was used to categorise participants into three groups: never had a mood disorder; had a prior mood disorder but not during the previous 12 months, or; had a mood disorder during the previous 12 months.

Other Covariates

Questionnaires collected data on: age (in years); current smoking status (never, ex-smoker, smoker); marital status (living as married, not living as married); highest education (university, vocational, school); and occupational status (manager/professional, non-manual, manual, not in workforce). Questions on usual duration (hours and minutes) of nightly sleep were only included at CDAH-2 and CDAH-3. Biological parenting status was defined as have children or don't have children. At CDAH-1, biological parenting status was only measured for females (reporting of live births) but was retrospectively imputed for males using birth dates of children reported at CDAH-2. At CDAH-2 and CDAH-3 participants were asked how many biological children they had. At each follow-up, social support was measured using the Henderson Index

of Perceived Social Support comprising 15 questions (for example, “I seem to have a lot of friends”, and “I have no-one to lean on in times of trouble”), with answers on a 5-point Likert scale from strongly agree to strongly disagree [19]. A higher score (range 15-75) indicated higher self-perceived social support. The validated International Physical Activity Questionnaire long form measured total weekly minutes of leisure-time physical activity, which was converted to hours/week for interpretability [20,21].

At CDAH-1 and CDAH-3, BMI (kg/m^2) was calculated from height and weight measured at clinics. A Heine portable digital scale (Heine, Dover, NH, US) was used to measure weight to the nearest 0.1kg. A Leicester stadiometer (Invicta, Leicester, UK) was used to measure height to the nearest 0.1cm. BMI categories were defined as non-overweight ($\text{BMI} < 25$), overweight ($25 \leq \text{BMI} < 30$), or obese ($\text{BMI} \geq 30$). At CDAH-2, BMI was calculated from self-reported height and weight with a correction factor based on discrepancies between self-reported and measured height and weight of CDAH-1 clinic participants [22].

Statistical analyses

Statistical analyses were undertaken using Stata version 16.1 (StataCorp, College Station, Texas, 2017). Summary statistics are reported as means and standard deviation (*SD*) for continuous variables, and percentages and frequency for categorical variables.

The cross-sectional samples at each time-point included all participants with relevant measures. The longitudinal samples included participants with baseline DGI and covariate data, and follow-up mood disorder data. A sensitivity analysis restricted the cross-sectional analyses to participants with data at all three time-points. For interpretability of results, the cross-sectional and longitudinal log-binomial regression analyses used DGI scores reduced by a factor of 10 so that difference in effect was for a 10-point change in DGI. Supplementary analysis used log-

binomial regression to examine the contribution of each DGI component to the cross-sectional associations between DGI score and mood disorder prevalence. Each regression adjusted for the total DGI score minus the component score.

Reverse causality analyses on the CDAH-2 and CDAH-3 cross-sectional samples used linear regression to examine if having a prior or current mood disorder, compared to never having a mood disorder, was associated with the outcome of DGI score. Transformations (e.g. logarithmic) of the DGI were used to remove skewness. The reverse causality analysis was not performed on the CDAH-1 sample as only 12-month, rather than lifetime mood disorders, had been measured.

Analyses were stratified by sex due to an interaction between DGI and sex at CDAH-1 ($p = 0.070$). Participants were excluded if they were missing covariate measures used in the final models or if they were pregnant (as their FFQ responses may not reflect usual diet). To mitigate bias from loss-to-follow-up, we applied an approach motivated by Seaman et al. [23]. Multiple imputation was used, where necessary, to complete the 1985 ASHFS data for missing data on variables that predicted loss-to-follow-up: sex and baseline BMI, smoking status, area-level socioeconomic status (SES), school type (government, Catholic, independent), academic performance, and if breakfast was usually eaten. These variables and collection methods are described in detail elsewhere [24]. Inverse probability weighting with weights based on the probability of participating in the follow-up using these variables, was then applied to the regression analyses.

Purposeful model building techniques used covariates plausibly associated causally with the outcome and that changed the coefficient of the principal study factor by at least 10% [25]. Model 1 adjusted for age in the cross-sectional and reverse causality analyses, and for age and time between follow-ups (follow-up age in years minus age at baseline) for the longitudinal analyses. The longitudinal analyses that included all mood disorders (first onset or recurrence)

was additionally adjusted for mood disorder during the 12 months prior to baseline. Fully adjusted models were built separately for males and females. Covariates considered for adjustment were smoking status, marital status, education, occupation; usual nightly sleep hours, parental status, leisure-time physical activity, and BMI. Total daily energy intake was not calculated as the FFQ measured usual frequency of consumption, not quantities. The inflammatory effects of obesity plausibly places BMI on the pathway between diet quality and mood disorders [26] so it was not used in the model building process where mood disorder was the outcome due to the risk of over-adjustment bias [27], but was added separately in an additional model.

Due to the strong effect of social support in the regression models, we examined the relationship between social support and DGI (using linear regression), and social support and mood disorders (using log binomial regression). The Henderson Index of Perceived Social Support was determined to have good internal consistency by calculating Cronbach's coefficient α [28], with $\alpha=0.86$ at CDAH-1, and $\alpha=0.87$ at both CDAH-2 and CDAH-3.

Results

The number of participants at each follow-up, participant exclusions, and final samples for analysis are detailed in **Figure 1**. Participants were missing dietary data if they did not complete the questionnaires or were missing responses to more than 10% of the FFQ items or key questions in the FHQ. Following exclusions for pregnancy and missing dietary and covariate data, the final samples for the cross-sectional analyses were: 1,974 CDAH-1 participants, 1,480 CDAH-2 participants, and 1,191 participants at CDAH-3. There were 1,057 participants with data for the CDAH-1 to CDAH-2 longitudinal analyses, and 785 participants for the CDAH-2 to CDAH-3 analyses.

Participant characteristics at each time-point are shown in **Table 1**. Compared to males, at each follow-up females had higher mean DGI and social support scores, lower mean hours of physical activity, lower percentages classified as overweight/obese or smokers, and a higher percentage with university education. Among both sexes, at CDAH-2 and CDAH-3 the percentage of participants who were overweight/obese, living as married, had biological children, and who were university educated was higher than the previous follow-up, while the percentage of smokers was lower.

Results of the cross-sectional analyses of mood disorder and DGI are shown in **Table 2**.

Females had higher prevalence of mood disorders at each time-point (CDAH-1: 11%; CDAH-2: 9%; CDAH-3: 11%) compared to males (CDAH-1: 6%; CDAH-2: 7%; CDAH-3: 7%).

Adjusting for social support had the largest effect on the estimated coefficient of the DGI at each time-point and therefore the results are presented without (Model 2) and with (Model 3) adjustment for social support. A 10-point higher DGI score was cross-sectionally associated with lower prevalence of mood disorders among females at all three time-points but was only statistically significant in Model 1 at CDAH-3 (PR=0.73, 95% CI: 0.56, 0.95). The association was attenuated after covariate adjustment in Model 2 (PR=0.82, 95% CI: 0.65, 1.05), and reduced further with inclusion of social support in Model 3 (PR=0.92, 95% CI: 0.73, 1.16).

Among males, better diet quality was associated with higher prevalence of mood disorder at CDAH-1 but not CDAH-2, and neither association was statistically significant. At CDAH-3, better diet quality was associated with a statistically significant lower prevalence of mood disorder among males in Model 1 (PR=0.72, 95% CI: 0.53, 0.97), but this was attenuated in Model 2 (PR=0.82, 95% CI: 0.61, 1.09) and Model 3 (PR=0.92, 95% CI: 0.69, 1.22). Further adjustment for BMI in Model 4 had little effect on the prevalence estimates for either sex (e.g. at CDAH-3: female PR=0.92, 95% CI: 0.75, 1.13; male PR=0.94, 95% CI: 0.69, 1.27). A complete-case sensitivity analysis among participants with data at all three time-points.

reduced the samples to 288 females and 214 males and there were no significant results, although associations remained broadly in the same directions (supplementary material **Table S2**).

Longitudinal analysis results are shown in **Table 3**. The mean (*SD*) follow-up intervals were 5.0 (0.3) years CDAH-1 to CDAH-2, and 7.5 (1.2) years CDAH-2 to CDAH-3. During each follow-up period, the percentage of females who had first or recurrent mood disorder (CDAH-1 to CDAH-2: 19%, CDAH-2 to CDAH-3: 17%) was higher than males (CDAH-1 to CDAH-2: 13%, CDAH-2 to CDAH-3: 12%). There was no longitudinal effect of DGI score among either sex when including both first or recurrent mood disorders during follow-up, or when examining first onset of mood disorder only. A higher DGI score at CDAH-2 was associated with an increased risk of first mood disorder between the CDAH-2 to CDAH-3 follow-ups among females in Model 1 (RR=1.25, 95% CI: 0.75, 2.06) and males in Model 3 (RR=1.26, 95% CI: 0.83, 1.91), but were not statistically significant and there was no association among females after covariate adjustment. Adjustment for social support in the longitudinal models did not have a similar strength of effect to that observed in the cross-sectional analyses, so separate models with and without social support are not presented.

Reverse causality analysis results are shown in **Table 4**. For both sexes at CDAH-2 and CDAH-3, the estimated DGI of participants with prior mood disorder but no symptoms within the past 12 months was not significantly different from participants who reported never having a mood disorder. Among both sexes at CDAH-3 a mood disorder within the past 12 months was significantly associated with a lower DGI score in Model 1 (females: β =-4.18, 95% CI: -7.76, -0.60; males β =-4.42, 95% CI: -8.26, -0.57), but the associations were attenuated in Model 2 (females: β =-0.93, 95% CI: -4.23, 2.36; males β =-1.31, 95% CI: -4.97, 2.35).

Supplementary analyses examined the contribution of individual DGI components to the CDAH-3 statistically significant cross-sectional association between total DGI score and mood

disorder prevalence (**Table S3**). After adjustment for age and total DGI score minus the component score, among females, lower mood disorder prevalence was associated with higher scores on the dietary variety, vegetables, fruit, grain, dairy and water components, but was only statistically significant for vegetable and grains. Among males, lower mood disorder prevalence was associated with higher scores on the variety, vegetable, fruit, protein, and water components, but was only statistically significant for protein. The associations were attenuated after further covariate adjustment but mainly remained in the same direction or reduced to null association.

In univariable linear regression analyses for each time-point, the relationship between social support and DGI was weakly positive (e.g. a one-point higher social support score was associated with a 0.28 higher DGI score among females at CDAH-3) (supplementary material **Table S4**). All results were significant ($p < 0.05$) except among males at CDAH-2. There was an inverse relationship between higher social support and mood disorder. A one-point higher social support score was associated with at least 7% lower prevalence of mood disorder among both sexes at all three time-points and was significant ($p < 0.05$) in all analyses.

Discussion

Diet quality was not longitudinally associated with risk of DSM-IV mood disorders among our cohort of young- to mid-adulthood participants, and there was no evidence of reverse causality. Significant cross-sectional associations between better diet quality and lower prevalence of mood disorder among both sexes in mid-adulthood were attenuated after covariate adjustment. The Henderson Index of Perceived Social Support score was noted as having the largest covariate effect on the cross-sectional regression coefficient at each time-point. For example,

adding social support in Model 3 at CDAH-3 attenuated the association for both sexes by 56%, from 18% reduced prevalence to 8%.

To our knowledge, only a few longitudinal studies among adults have examined overall diet quality in relation to mood disorder outcomes that met diagnostic criteria, and none have used mood disorder outcomes from structured diagnostic interviews. Therefore, it is difficult to say whether our null results are consistent with the existing literature. Several previous studies have used self-report of physician diagnosis of mood disorders. Prospective studies from the Seguimiento Universidad de Navarra cohort of Spanish university graduates reported associations between healthier dietary patterns and lower risk of depressive disorder over the eight-year follow-up, measured by self-report of physician diagnosis and anti-depressant use and validated with clinical interviews among a sub-sample [29-31]. In contrast, a 2013 study from the United States, the Nurses' Health Study, reported that there was no prospective association during the 12-year follow-up among middle-aged and older women between healthier dietary intake and self-reported clinical diagnosis of depression or use of anti-depressants [9]. A 2018 meta-analysis (including studies of individual foods (e.g. fish) and one study among adolescents), found that although better diet quality may be associated with lower risk of depressive symptoms, results were weaker and non-significant for studies that used a formal diagnosis as the outcome or controlled for depressive symptoms at baseline [3].

Previous research has also highlighted the importance of controlling for time-varying covariates [3,32]. The mediation of current circumstance on mood disorders is consistent with stronger results reported from cross-sectional studies [2,4].

In the reverse causality analysis, at CDAH-3 only, associations between current mood disorder and lower DGI scores was attenuated after covariate adjustment. Participants with a prior (not within past 12 months) mood disorder did not have significantly different diet quality scores from those who had never had a mood disorder. There is limited and inconsistent evidence of

reverse causality in existing literature. For example, a 2015 study reported that prior and treated mood disorders were associated with better diet quality [33], whereas a 2020 study reported that participants with mood disorders prior to (but not at) baseline had lower diet quality at follow-up [34]. A bidirectional relationship is possible. Previous CDAH studies have reported bidirectional associations from CDAH-1 to CDAH-2 between mood disorders and healthy lifestyle scores [35], and mood disorders and a time-of-day eating pattern characterised by skipped or delayed breakfast [36].

Female participants had markers of healthier lifestyles (e.g. lower BMI, higher DGI, lower smoking rates) compared to males, but higher prevalence of mood disorders. The higher mood disorder prevalence among women is consistent with numerous studies and may arise from social factors, biological and hormonal influences, and women being more likely to remember and report more extreme negative or positive affect than men, therefore reaching diagnostic criteria [37]. The direction of the cross-sectional relationship between better diet quality and lower prevalence of recent mood disorder among females at all three follow-ups was in line with our hypothesis, as was the association among males at CDAH-3.

Supplementary analysis indicated that the DGI components that contributed to the overall DGI-mood disorder association at CDAH-3 were those that promote good health (quality and minimum intakes of foods from the core food groups, water, and dietary variety), rather than components that measured limiting of discretionary foods and saturated fats. However, these effects were lessened after covariate adjustment. Higher intake of healthy foods and dietary variety rather than unhealthy food intake, appear to underpin the crude relationship between better overall diet and lower mood disorder prevalence, but the apparent effects of these core food groups were largely explained by sociodemographic and lifestyle factors.

Social support was cross-sectionally associated with DGI score (positively) and mood disorder (inversely) and may be an important confounder or mediator not commonly accounted for in

diet-mood disorder studies. Few studies in recent decades have taken into account dimensions of support other than marital status, such as living alone [38,39], friendship quality, support scales, or social activity [35,40-43]. Moreover, various dimensions of social support may play different roles at different life stages or according to gender or cultural context. For example, dietary related social support such as level of encouragement to eat healthily from family and friends, has been effective in improving success of nutritional interventions or dietary behavior change [44]. There may also be a synergistic effect of self-efficacy and social support for improving and maintaining better diet quality [45]. Women may have better internalised strategies for healthy eating compared to men [46], but also be more susceptible to mood disorders in the face of low social support [47]. These factors highlight that social support could be an important variable to consider in future research on diet and mood disorders.

A limitation of this study includes the non-quantitative FFQ, which may have introduced error as guidance on serve sizes was only given for fruit and vegetables. However, this type of FFQ has been found to be a valid method of data collection [13] and the assumption that one reported frequency of an item is equal to one serve has been used for validated diet quality measures [14,15]. The non-quantitative FFQ also prevented total energy intake calculation, but the main determinants of energy intake were accounted for by using the sex-specific scoring for the DGI, stratifying analyses by sex, considering physical activity as a potential confounder, and adjusting for BMI. Another limitation is that the participant samples in the primary analyses were different at each follow-up as the number of participants who completed three follow-ups was low. The longitudinal analyses examining first onset mood disorders and the complete-case sensitivity analyses involve particularly small samples and low statistical power. The different methods of data collection at CDAH-2 (postal/online surveys and telephone CIDI interviews) compared to collection of data at clinics may have introduced bias and could account for smaller effects observed in the CDAH-2 results. These factors limit

interpretation of the influence of life stage. There was also large loss-to-follow-up from the original nationally representative ASHFS sample, which was greater for males and those with markers of lower childhood SES and poorer dietary habits (overweight/obese and less likely to eat breakfast before school). There is evidence that factors such as low childhood SES and poor childhood dietary practices are related to greater risk of adult mood disorder [48] and lower adult diet quality [49,50], which means that participants with these characteristics may be under-represented in our sample. Loss-to-follow-up was mitigated by inverse probability weighting in the regression analyses, but caution should be taken in generalising the results to other populations.

Strengths of the study include: longitudinal and reverse causality analyses to examine direction of associations and effects over time; accounting for prior mood in the longitudinal analyses; the range of covariate measures, and; using DSM-IV diagnoses of mood disorder. Although CIDI validation studies have indicated that there may be some mis-identification of cases, primarily due to under-diagnosis [51], the CIDI is regarded as the ‘gold standard’ for epidemiological studies and suitable for retrospective measurement of mental disorders [52]. The use of the structured diagnostic interview and DSM-IV criteria is important as they identify outcomes that are likely to be more clinically relevant and impact on the participant’s everyday life than non-standardised measures of depressive symptoms. The influence of covariates and potential confounders were carefully considered. Purposeful model building highlighted that the covariates included in the regression models differed by sex and life stage. For example, educational attainment and marital status had insufficient effect on the coefficients to be included in the final models at CDAH-1 but were important at CDAH-2 and CDAH-3.

In summary, among our young- to mid-adulthood cohort, we found no evidence to support the hypothesis that diet quality had an independent effect on mood disorders. Significant cross-

408 sectional associations observed between diet quality and mood among participants in mid-
409 adulthood were explained by demographic, lifestyle, and psycho-social factors, particularly the
410 level of self-perceived social support.

411

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417 **Declarations**

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425

426 **Conflicts of interest**

427 The authors declare that they have no conflict of interest.

428

429 **Ethics approval**

430 All procedures contributing to this work comply with the ethical standards of the relevant
431 national and institutional committees on human experimentation and with the Helsinki
432 Declaration of 1975, as revised in 2008. The State Directors General of Education approved the
433 ASHFS and the Southern Tasmanian Health and Medical Ethics Committee approved the
434 CDAH study protocol.

435

436 **Consent to participate**

437 Signed parental consent was required for all ASFHS participants. All CDAH follow-up
438 participants gave informed written consent.

439 **Consent for publication**

440 Patients signed informed consent regarding publishing their data, provided that individuals
441 could not be identified as a subject.

442

443 **Availability of data and material (data transparency)**

444 The data that support the findings of this study are available from the corresponding author
445 upon reasonable request.

446

447 **Code availability (software application or custom code)**

448 The custom Stata code used for the study analyses is available from the corresponding author
449 upon reasonable request.

450

451 **Authors' contributions**

452 Johanna Wilson conceived and designed the study, understood all data analyses, composed the
453 draft manuscript and coordinated revisions of the manuscript. Leigh Blizzard provided
454 statistical expertise. Kylie Smith provided subject matter expertise and editing. Alison Venn
455 and Terence Dwyer were involved in conceptualization of the CDAH study and data
456 acquisition. All authors reviewed and commented on the draft manuscript and approved the
457 final manuscript.

References

1. Lai JS, Hiles S, Bisquera A, Hure AJ, McEvoy M and Attia J (2014) A systematic review and meta-analysis of dietary patterns and depression in community-dwelling adults. *Am J Clin Nutr* 99: 181-197. <https://doi.org/10.3945/ajcn.113.069880>
2. Li Y, Lv MR, Wei YJ, Sun L, Zhang JX, Zhang HG and Li B (2017) Dietary patterns and depression risk: A meta-analysis. *Psychiatry Res* 253: 373-382. <https://doi.org/10.1016/j.psychres.2017.04.020>
3. Molendijk M, Molero P, Sanchez-Pedreno FO, Van der Does W and Martinez-Gonzalez MA (2018) Diet quality and depression risk: A systematic review and dose-response meta-analysis of prospective studies. *J Affect Disord* 226: 346-354. <https://doi.org/10.1016/j.jad.2017.09.022>
4. Lassale C, Batty GD, Baghdadli A, Jacka F, Sánchez-Villegas A, Kivimäki M and Akbaraly T (2018) Healthy dietary indices and risk of depressive outcomes: a systematic review and meta-analysis of observational studies. *Mol Psychiatry* 24: 965-986. <https://doi.org/10.1038/s41380-018-0237-8>
5. Cespedes EM and Hu FB (2015) Dietary patterns: from nutritional epidemiologic analysis to national guidelines. *Am J Clin Nutr* 101: 899-900. <https://doi.org/10.3945/ajcn.115.110213>
6. Smarr KL and Keefer AL (2011) Measures of depression and depressive symptoms: Beck Depression Inventory-II (BDI-II), Center for Epidemiologic Studies Depression Scale (CES-D), Geriatric Depression Scale (GDS), Hospital Anxiety and Depression Scale (HADS), and Patient Health Questionnaire-9 (PHQ-9). *Arthritis Care Res (Hoboken)* 63: S454-S466. <https://doi.org/10.1002/acr.20556>
7. Eaton WW, Hall ALF, Macdonald R and McKibben J (2007) Case identification in psychiatric epidemiology: A review. *Int Rev Psychiatry* 19: 497-507. <https://doi.org/10.1080/09540260701564906>

- 482 8. Sánchez-Villegas A, Delgado-Rodríguez M, Alonso A, Schlatter J, Lahortiga F, Majem LS
483 and Martínez-González MA (2009) Association of the Mediterranean dietary pattern with the
484 incidence of depression: the Seguimiento Universidad de Navarra/University of Navarra
485 follow-up (SUN) cohort. Arch Gen Psychiatry 66: 1090-1098.
486 <https://doi.org/10.1001/archgenpsychiatry.2009.129>
- 487 9. Chocano-Bedoya PO, O'Reilly EJ, Lucas M, Mirzaei F, Okereke OI, Fung TT, Hu FB and
488 Ascherio A (2013) Prospective study on long-term dietary patterns and incident depression in
489 middle-aged and older women. Am J Clin Nutr 98: 813-820.
490 <https://doi.org/10.3945/ajcn.112.052761>
- 491 10. Gall SL, Jose K, Smith K, Dwyer T and Venn A (2009) The Childhood Determinants of
492 Adult Health Study: a profile of a cohort study to examine the childhood influences on adult
493 cardiovascular health. Australas Epidemiol 16: 35-39.
- 494 11. Institute for Health Metrics and Evaluation (2018) Findings from the Global Burden of
495 Disease Study 2017. Seattle, WA, 25.
- 496 12. McLennan W and Podger AS (1998) National Nutrition Survey Users' Guide, 1995.
497 Canberra: Australian Bureau of Statistics, 162.
- 498 13. Hodge A, Patterson AJ, Brown WJ, Ireland P and Giles G (2000) The Anti Cancer Council
499 of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in
500 young to middle-aged women in a study of iron supplementation. Aust N Z J Public Health 24:
501 576-583. <https://doi.org/10.1111/j.1467-842X.2000.tb00520.x>
- 502 14. McNaughton SA, Ball K, Crawford D and Mishra GD (2008) An index of diet and eating
503 patterns is a valid measure of diet quality in an Australian population. J Nutr 138: 86-93.
- 504 15. Wilson JE, Blizzard L, Gall SL, Magnussen CG, Oddy WH, Dwyer T, Venn AJ and Smith
505 KJ (2019) An age-and sex-specific dietary guidelines index is a valid measure of diet quality in

506 an Australian cohort during youth and adulthood. *Nutr Res* 65: 43-53.

507 <https://doi.org/10.1016/j.nutres.2019.01.007>

508 16. National Health and Medical Research Council (2013) *Eat for Health: Australian Dietary*

509 *Guidelines*. Canberra: National Health and Medical Research Council, 210.

510 17. World Health Organization (1997) *Composite International diagnostic Interview, CIDI-*

511 *Auto 2.1 : Administrator's guide and reference*, Geneva: World Health Organization.

512 18. American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental*

513 *Disorders*, Washington: American Psychiatric Association.

514 19. Henderson S, Duncan-Jones P, McAuley H and Ritchie K (1978) The patient's primary

515 group. *Br J Psychiatry* 132: 74-86. <https://doi.org/10.1192/S0007125000283001>

516 20. Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, Pratt M,

517 Ekelund U, Yngve A and Sallis JF (2003) International physical activity questionnaire: 12-

518 country reliability and validity. *Med Sci Sports Exerc* 35: 1381-1395.

519 <https://doi.org/10.1249/01.MSS.0000078924.61453.FB>

520 21. McKercher CM, Schmidt MD, Sanderson KA, Patton GC, Dwyer T and Venn AJ (2009)

521 *Physical Activity and Depression in Young Adults*. *Am J Prev Med* 36: 161-164.

522 <https://doi.org/10.1016/j.amepre.2008.09.036>

523 22. Smith KJ, Gall SL, McNaughton SA, Cleland VJ, Otahal P, Dwyer T and Venn AJ (2017)

524 *Lifestyle behaviours associated with 5-year weight gain in a prospective cohort of Australian*

525 *adults aged 26-36 years at baseline*. *BMC Public Health* 17: 54.

526 <https://doi.org/10.1186/s12889-016-3931-y>

527 23. Seaman SR, White IR, Copas AJ and Li L (2012) Combining Multiple Imputation and

528 Inverse-Probability Weighting. *Biometrics* 68: 129-137. [https://doi.org/10.1111/j.1541-](https://doi.org/10.1111/j.1541-0420.2011.01666.x)

529 [0420.2011.01666.x](https://doi.org/10.1111/j.1541-0420.2011.01666.x)

530 24. Wilson JE, Blizzard L, Gall SL, Magnussen CG, Oddy WH, Dwyer T, Venn AJ and Smith
531 KJ (2020) Youth diet quality and hazard of mood disorder in adolescence and adulthood
532 among an Australian cohort. *J Affect Disord* 276: 511-518.
533 <https://doi.org/10.1016/j.jad.2020.07.048>

534 25. Greenland S (1989) Modeling and variable selection in epidemiologic analysis. *Am J*
535 *Public Health* 79: 340-349. <https://doi.org/10.2105/AJPH.79.3.340>

536 26. Oddy WH, Allen KL, Trapp GS, Ambrosini GL, Black LJ, Huang R-C, Rzehak P, Runions
537 KC, Pan F and Beilin LJ (2018) Dietary patterns, body mass index and inflammation:
538 pathways to depression and mental health problems in adolescents. *Brain Behav Immun* 69:
539 428-439. <https://doi.org/10.1016/j.bbi.2018.01.002>

540 27. Schisterman EF, Cole SR and Platt RW (2009) Overadjustment bias and unnecessary
541 adjustment in epidemiologic studies. *Epidemiology* 20: 488.
542 <https://doi.org/10.1097/EDE.0b013e3181a819a1>

543 28. Cronbach LJ (1951) Coefficient alpha and the internal structure of tests. *Psychometrika* 16:
544 297-334. <https://doi.org/10.1007/BF02310555>

545 29. Perez-Cornago A, Sanchez-Villegas A, Bes-Rastrollo M, Gea A, Molero P, Lahortiga-
546 Ramos F and Martinez-Gonzalez MÁ (2017) Relationship between adherence to Dietary
547 Approaches to Stop Hypertension (DASH) diet indices and incidence of depression during up
548 to 8 years of follow-up. *Public Health Nutr* 20: 2383-2392.
549 <https://doi.org/10.1017/S1368980016001531>

550 30. Sanchez-Villegas A, Schlatter J, Ortuno F, Lahortiga F, Pla J, Benito S and Martinez-
551 Gonzalez MA (2008) Validity of a self-reported diagnosis of depression among participants in
552 a cohort study using the Structured Clinical Interview for DSM-IV (SCID-I). *BMC Psychiatry*
553 8: 43. <https://doi.org/10.1186/1471-244X-8-43>

- 554 31. Sánchez-Villegas A, Henríquez-Sánchez P, Ruiz-Canela M, Lahortiga F, Molero P, Toledo
555 E and Martínez-González MA (2015) A longitudinal analysis of diet quality scores and the risk
556 of incident depression in the SUN Project. BMC Med 13: 197. [https://doi.org/10.1186/s12916-](https://doi.org/10.1186/s12916-015-0428-y)
557 [015-0428-y](https://doi.org/10.1186/s12916-015-0428-y)
- 558 32. Lai JS, Oldmeadow C, Hure AJ, McEvoy M, Byles J and Attia J (2016) Longitudinal diet
559 quality is not associated with depressive symptoms in a cohort of middle-aged Australian
560 women. Br J Nutr 115: 842-850. <https://doi.org/10.1017/s000711451500519x>
- 561 33. Jacka FN, Cherbuin N, Anstey KJ and Butterworth P (2015) Does reverse causality explain
562 the relationship between diet and depression? J Affect Disord 175: 248-250.
563 <https://doi.org/10.1016/j.jad.2015.01.007>
- 564 34. Ofstedal S, Glozier N, Holliday EG and Duncan MJ (2020) Diet quality and depressive
565 symptoms. Assessing the direction of the association in a population-based cohort study. J
566 Affect Disord 274: 347-353. <https://doi.org/10.1016/j.jad.2020.05.046>
- 567 35. Gall SL, Sanderson K, Smith KJ, Patton G, Dwyer T and Venn A (2016) Bi-directional
568 associations between healthy lifestyles and mood disorders in young adults: The Childhood
569 Determinants of Adult Health Study. Psychol Med 46: 2535-2548.
570 <https://doi.org/10.1017/S0033291716000738>
- 571 36. Wilson JE, Blizzard L, Gall SL, Magnussen CG, Oddy WH, Dwyer T, Sanderson K, Venn
572 AJ and Smith KJ (2019) An eating pattern characterised by skipped or delayed breakfast is
573 associated with mood disorders among an Australian adult cohort. Psychol Med: 1-11.
574 <https://doi.org/10.1017/S0033291719002800>
- 575 37. Parker G and Brotchie H (2010) Gender differences in depression. Int Rev Psychiatry 22:
576 429-436. <https://doi.org/10.3109/09540261.2010.492391>

577 38. Akbaraly TN, Sabia S, Shipley MJ, Batty GD and Kivimaki M (2013) Adherence to
578 healthy dietary guidelines and future depressive symptoms: evidence for sex differentials in the
579 Whitehall II study. *Am J Clin Nutr* 97: 419-427. <https://doi.org/10.3945/ajcn.112.041582>

580 39. Okubo R, Matsuoka YJ, Sawada N, Mimura M, Kurotani K, Nozaki S, Shikimoto R and
581 Tsugane S (2019) Diet quality and depression risk in a Japanese population: the Japan Public
582 Health Center (JPHC)-based Prospective Study. *Sci Rep* 9: 7150.
583 <https://doi.org/10.1038/s41598-019-43085-x>

584 40. Hodge A, Almeida OP, English DR, Giles GG and Flicker L (2013) Patterns of dietary
585 intake and psychological distress in older Australians: benefits not just from a Mediterranean
586 diet. *Int Psychogeriatr* 25: 456-466. <https://doi.org/10.1017/s1041610212001986>

587 41. Gangwisch JE, Hale L, Garcia L, Malaspina D, Opler MG, Payne ME, Rossom RC and
588 Lane D (2015) High glycemic index diet as a risk factor for depression: analyses from the
589 Women's Health Initiative. *Am J Clin Nutr* 102: 454-463.
590 <https://doi.org/10.3945/ajcn.114.103846>

591 42. Winpenny EM, van Harmelen A-L, White M, van Sluijs EM and Goodyer IM (2018) Diet
592 quality and depressive symptoms in adolescence: no cross-sectional or prospective associations
593 following adjustment for covariates. *Public Health Nutr* 21: 2376-2384.
594 <https://doi.org/10.1017/S1368980018001179>

595 43. Sánchez-Villegas A, Ruíz-Canela M, Gea A, Lahortiga F and Martínez-González MA
596 (2016) The association between the Mediterranean lifestyle and depression. *Clin Psychol Sci* 4:
597 1085-1093. <https://doi.org/10.1177/2167702616638651>

598 44. Greaves CJ, Sheppard KE, Abraham C, Hardeman W, Roden M, Evans PH and Schwarz P
599 (2011) Systematic review of reviews of intervention components associated with increased
600 effectiveness in dietary and physical activity interventions. *BMC Public Health* 11: 119.
601 <https://doi.org/10.1186/1471-2458-11-119>

- 602 45. Anderson ES, Winett RA and Wojcik JR (2007) Self-regulation, self-efficacy, outcome
603 expectations, and social support: social cognitive theory and nutrition behavior. *Ann Behav*
604 *Med* 34: 304-312. <https://doi.org/10.1007/BF02874555>
- 605 46. Lange D, Corbett J, Knoll N, Schwarzer R and Lippke S (2018) Fruit and Vegetable Intake:
606 the Interplay of Planning, Social Support, and Sex. *Int J Behav Med* 25: 421-430.
607 <https://doi.org/10.1007/s12529-018-9718-z>
- 608 47. Kendler KS, Myers J and Prescott CA (2005) Sex differences in the relationship between
609 social support and risk for major depression: a longitudinal study of opposite-sex twin pairs.
610 *Am J Psychiatry* 162: 250-256. <https://doi.org/10.1176/appi.ajp.162.2.250>
- 611 48. Gilman SE, Kawachi I, Fitzmaurice GM and Buka SL (2002) Socioeconomic status in
612 childhood and the lifetime risk of major depression. *Int J Epidemiol* 31: 359-367.
613 <https://doi.org/10.1093/ije/31.2.359>
- 614 49. Mikkila V, Rasanen L, Raitakari OT, Pietinen P and Viikari J (2004) Longitudinal changes
615 in diet from childhood into adulthood with respect to risk of cardiovascular diseases: The
616 Cardiovascular Risk in Young Finns Study. *Eur J Clin Nutr* 58: 1038-1045.
617 <https://doi.org/10.1038/sj.ejcn.1601929>
- 618 50. Hare-Bruun H, Togo P, Andersen LB and Heitmann BL (2011) Adult Food Intake Patterns
619 Are Related to Adult and Childhood Socioeconomic Status. *J Nutr* 141: 928-934.
620 <https://doi.org/10.3945/jn.110.133413>
- 621 51. Wittchen HU (1994) Reliability and validity studies of the WHO-Composite International
622 Diagnostic Interview (CIDI): A critical review. *J Psychiatr Res* 28: 57-84.
623 [https://doi.org/10.1016/0022-3956\(94\)90036-1](https://doi.org/10.1016/0022-3956(94)90036-1)
- 624 52. Steel Z, Marnane C, Iranpour C, Chey T, Jackson JW, Patel V and Silove D (2014) The
625 global prevalence of common mental disorders: a systematic review and meta-analysis 1980–
626 2013. *Int J Epidemiol* 43: 476-493. <https://doi.org/10.1093/ije/dyu038>

Fig. 1 Childhood Determinants of Adult Health (CDAH) study participation flow chart

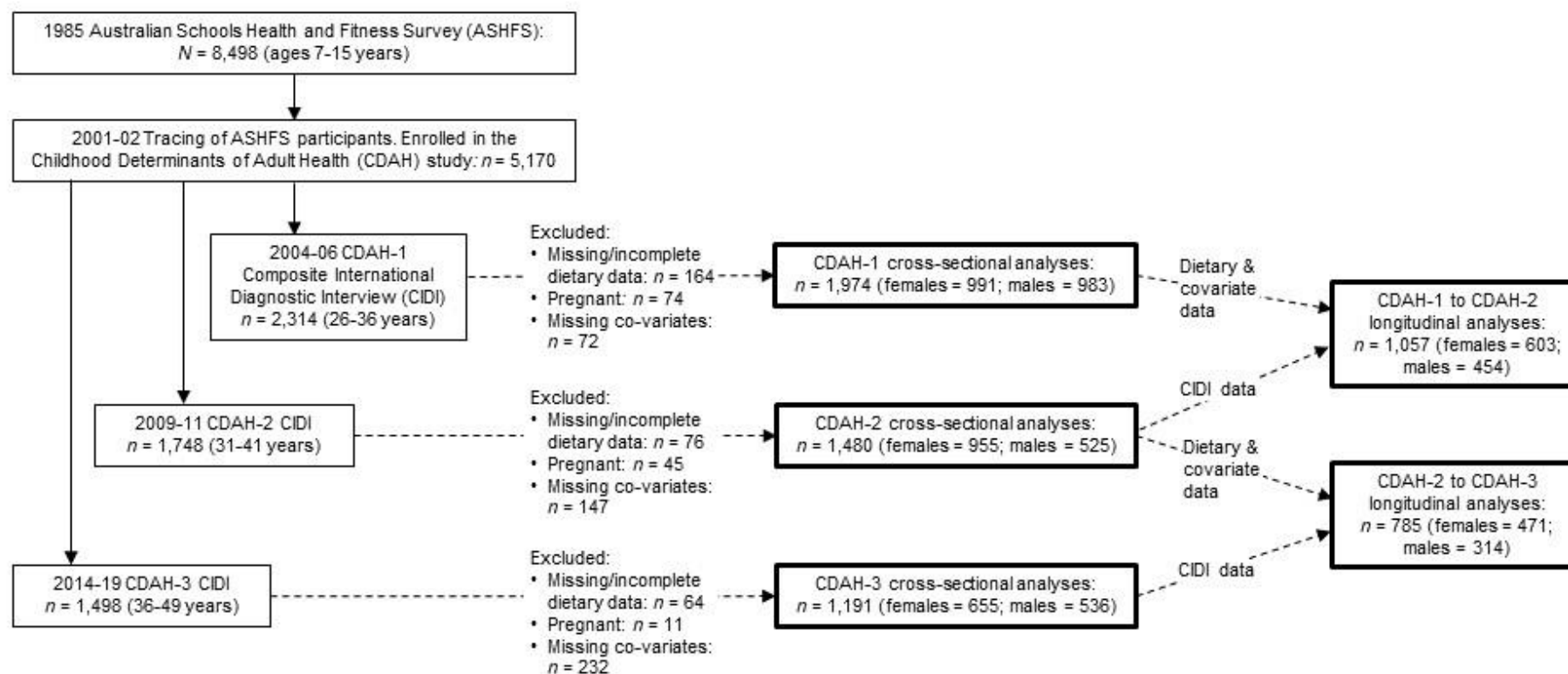


Table 1 Characteristics of the Childhood Determinants of Adult Health (CDAH) study participants at follow-ups: CDAH-1 (2004-06), CDAH-2 (2009-11), and CDAH-3 (2014-19)

	Females						Males					
	CDAH-1		CDAH-2		CDAH-3		CDAH-1		CDAH-2		CDAH-3	
	<i>n</i>	% or mean(<i>SD</i>)	<i>n</i>	% or mean(<i>SD</i>)	<i>n</i>	% or mean(<i>SD</i>)	<i>n</i>	% or mean(<i>SD</i>)	<i>n</i>	% or mean(<i>SD</i>)	<i>n</i>	% or mean(<i>SD</i>)
Age (years)	991	31.4(2.6)	955	36.4(2.6)	655	43.9(2.9)	983	31.6(2.6)	525	36.8(2.6)	536	44(2.8)
Social Support Index ^a	991	62.2(7.4)	955	61.9(7.9)	655	63.1(7.9)	983	61.7(7.6)	525	60.6(8.2)	536	61.0(8.3)
Dietary Guidelines Index ^b	991	58.3(11.0)	955	59.3(10.9)	655	58.4(11.2)	983	51.6(11.0)	525	53.9(11.3)	536	52.8(11.3)
Leisure-time Physical Activity	991	2.6(3.0)	897	2.8(3.0)	655	3.1(3.6)	901	2.9(3.6)	525	2.9(3.4)	536	3.3(3.6)
Usual sleep hours ^c	–	–	947	7.5(1.0)	655	7.1(1.0)	–	–	525	7.2(1.0)	536	6.9(1.0)
BMI Category												
Non-overweight (BMI < 25)	617	62.3	562	58.9	299	45.7	379	38.6	198	37.7	171	31.9
Overweight (25 ≤ BMI < 30)	232	23.4	230	24.1	203	31.0	444	45.2	243	46.3	237	44.2
Obese (BMI ≥ 30)	142	14.3	163	17.1	153	23.4	160	16.3	84	16.0	128	23.9
Smoking												
Never	558	56.3	555	58.2	416	63.5	561	57.1	331	63.1	346	64.6
Ex-Smoker	229	23.1	279	29.3	190	29.0	179	18.2	113	21.5	130	24.3
Smoker	204	20.6	120	12.6	49	7.5	243	24.7	81	15.4	60	11.2
Marital Status												
Not living as married	318	32.1	181	19.0	126	19.2	313	31.8	81	15.4	83	15.5
Living as married	673	67.9	774	81.1	529	80.8	670	68.2	444	84.6	453	84.5
Have Children												
No	449	48.2	253	26.9	118	18.0	442	60.8	174	33.1	88	16.5
Yes	483	51.8	686	73.1	537	82.0	285	39.2	351	66.9	445	83.5
Highest Education												
University	482	48.6	500	52.4	384	58.6	376	38.4	252	48.0	283	52.8
Vocational	246	24.8	251	26.3	193	29.5	355	36.2	186	35.4	184	34.3
School	263	26.5	204	21.4	78	11.9	249	25.4	87	16.6	69	12.9

Occupation												
Manager/Professional	514	51.9	484	50.7	385	58.8	567	58.5	366	69.7	388	72.4
Non-manual	265	26.7	238	24.9	166	25.3	74	7.6	30	5.7	32	6.0
Manual	42	4.2	53	5.6	22	3.4	298	30.8	112	21.3	96	17.9
Not in workforce	170	17.2	180	18.9	82	12.5	30	3.1	17	3.2	20	3.7

SD: standard deviation; BMI: body mass index = (weight (kg))/(height (m))²

^a Henderson Index of Perceived Social Support: range 15-75. A higher score indicates higher self-perceived social support

^b Dietary Guidelines Index: range 0-100. A higher score indicates better diet quality

^c Usual nightly sleep hours were not measured at CDAH-1

Table 2 Prevalence ratios of mood disorder within 12 months prior to follow-up for a 10-point higher Dietary Guidelines Index score among participants at Childhood Determinants of Adult Health (CDAH) study follow-ups at CDAH-1 (2004-06), CDAH-2 (2009-11), and CDAH-3 (2014-19)

	% with mood disorder	(n/N)	Model 1 ^a		Model 2 ^b		Model 3 ^c		Model 4 ^d	
			PR ^e	(95% CI)	PR	(95% CI)	PR	(95% CI)	PR	(95% CI)
CDAH-1										
Females	11.4	(113/991)	0.84	(0.70, 1.00)	0.92	(0.76, 1.12)	0.96	(0.80, 1.16)	0.95	(0.77, 1.16)
Males	6.1	(60/983)	1.04	(0.80, 1.36)	1.06	(0.82, 1.38)	1.21	(0.98, 1.49)	1.21	(0.99, 1.49)
CDAH-2										
Females	9.1	(87/955)	0.90	(0.73, 1.11)	0.90	(0.75, 1.09)	0.92	(0.78, 1.08)	0.89	(0.76, 1.06)
Males	6.7	(35/525)	0.99	(0.79, 1.24)	1.01	(0.77, 1.31)	0.98	(0.74, 1.32)	0.98	(0.72, 1.33)
CDAH-3										
Females	11.3	(74/655)	0.73	(0.56, 0.95)*	0.82	(0.65, 1.05)	0.92	(0.73, 1.16)	0.92	(0.75, 1.13)
Males	7.3	(39/536)	0.72	(0.53, 0.97)*	0.82	(0.61, 1.09)	0.92	(0.69, 1.22)	0.94	(0.69, 1.27)

* $p < 0.05$

PR: prevalence ratio; CI: confidence interval

^a Model 1: adjusted for age

^b Model 2: CDAH-1: females adjusted for age, smoking status, occupation, leisure-time physical activity; males adjusted for age, smoking status

CDAH-2: females adjusted for age, occupation, education, marital status; males adjusted for age, occupation, education, marital status, smoking status, parental status, sleep hours, and leisure-time physical activity

CDAH-3: females adjusted for age, occupation, education, marital status, smoking status, parental status, sleep hours, and leisure-time physical activity; males adjusted for age, occupation, education, marital status, smoking status, sleep hours, and leisure-time physical activity

^c Model 3: as per Model 2, plus Social Support

^d Model 4: as per Model 3, plus BMI.

^e The PR value is for a 10-point higher Dietary Guidelines Index (DGI) score (range 0-100). A higher DGI score indicates better diet quality

Table 3 Relative risk of mood disorder during follow-up, for a 10-point higher Dietary Guidelines Index score at baseline among participants of the Childhood Determinants of Adult Health (CDAH) study follow-ups CDAH-1 (2004-06) to CDAH-2 (2009-11), and CDAH-2 to CDAH-3 (2014-19)

	% with mood disorder	(n/N)	Model 1 ^a		Model 2 ^b		Model 3 ^c	
			RR ^d	(95% CI)	RR	(95% CI)	RR	(95% CI)
Any mood disorder during follow-up (first onset or recurrence)								
CDAH-1 to CDAH-2								
Females	19.1	(115/603)	1.03	(0.86, 1.24)	1.07	(0.90, 1.28)	1.07	(0.88, 1.30)
Males	13.2	(60/454)	1.06	(0.91, 1.23)	1.06	(0.87, 1.28)	1.08	(0.97, 1.20)
CDAH-2 to CDAH-3								
Females	17.2	(81/471)	1.08	(0.87, 1.33)	1.08	(0.90, 1.29)	1.04	(0.85, 1.28)
Males	12.1	(38/314)	1.09	(0.79, 1.49)	1.15	(0.85, 1.56)	1.12	(0.83, 1.51)
First onset mood disorder only ^e								
CDAH-1 to CDAH-2								
Females	9.1	(43/474)	0.86	(0.62, 1.19)	0.96	(0.66, 1.40)	0.95	(0.64, 1.41)
Males	4.6	(18/390)	1.01	(0.69, 1.48)	0.91	(0.63, 1.32)	0.91	(0.64, 1.30)
CDAH-2 to CDAH-3								
Females	4.4	(17/384)	1.25	(0.75, 2.06)	1.05	(0.71, 1.55)	0.98	(0.60, 1.62)
Males	5.6	(16/284)	1.07	(0.76, 1.51)	1.17	(0.80, 1.70)	1.26	(0.83, 1.91)

RR: risk ratio; CI: confidence interval

^a Model 1: adjusted for baseline age and time between follow-ups. Analyses for “First onset or recurrent mood disorder” were also adjusted for experience of mood disorder during 12 months prior to baseline at CDAH-1 or CDAH-2

^b Model 2: as per Model 1 plus additional adjustment for covariates as follows:

First onset or recurrent mood disorder: CDAH-1 to CDAH-2 females: smoking status, parental status, education, social support; CDAH-1 to CDAH-2 males: marital status, occupation, education, social support; CDAH-2 to CDAH-3 females: marital status, smoking status, occupation, usual sleep hours, leisure-time physical activity; CDAH-2 to CDAH-3 males: occupation, leisure-time physical activity, social support

First onset mood disorder only: CDAH-1 to CDAH-2 females: smoking status, parental status, marital status, occupation, education, leisure-time physical activity, social support; CDAH-1 to CDAH-2 males: parenting status, education; CDAH-2 to CDAH-3 females: smoking status, parental status, marital status, occupation, education, leisure-time physical activity, social support, usual sleep hours; CDAH-2 to CDAH-3 males: smoking status, parenting status

^c Model 3: as per Model 2, plus BMI

^d The RR value is for a 10-point higher Dietary Guidelines Index (DGI) score (range 0-100). A higher DGI score indicates better diet quality

^e The analyses examining risk of “First onset mood disorder only” excluded participants who reported experiencing a mood disorder prior to baseline at CDAH-1 or CDAH-2 as relevant

Table 4 Linear regression of Dietary Guidelines Index score on mood disorder status at CDAH-2 (2009-11) and CDAH-3 (2014-19)

	%	(n/N)	Model 1 ^a		Model 2 ^b	
			β	(95% CI)	β	(95% CI)
CDAH-2						
Females						
Never had mood disorder	68.4	(653/955)	Reference		Reference	
Mood disorder > 12 months previous	22.5	(215/955)	0.05	(-1.74, 1.83)	0.33	(-1.43, 2.09)
Mood disorder ≤ 12 months previous	9.1	(87/955)	-1.37	(-4.11, 1.37)	-0.76	(-3.61, 2.10)
Males						
Never had mood disorder	80.0	(420/525)	Reference		Reference	
Mood disorder > 12 months previous	13.3	(70/525)	1.28	(-2.09, 4.65)	1.47	(-1.57, 4.50)
Mood disorder ≤ 12 months previous	6.7	(35/525)	0.21	(-3.08, 3.49)	0.45	(-3.20, 4.10)
CDAH-3						
Females						
Never had mood disorder	68.1	(446/655)	Reference		Reference	
Mood disorder > 12 months previous	20.6	(135/655)	0.09	(-2.20, 2.37)	1.45	(-0.94, 3.84)
Mood disorder ≤ 12 months previous	11.3	(74/655)	-4.18	(-7.76, -0.60)*	-0.93	(-4.23, 2.36)
Males						
Never had mood disorder	79.1	(421/536)	Reference		Reference	
Mood disorder > 12 months previous	14.3	(76/536)	0.19	(-2.64, 3.02)	0.76	(-1.96, 3.49)
Mood disorder ≤ 12 months previous	7.3	(39/536)	-4.42	(-8.26, -0.57)*	-1.31	(-4.97, 2.35)

* $p < 0.05$

CDAH: Childhood Determinants of Adult Health study; CI: confidence interval; β: The estimated change in DGI score (range 0-100) among those with previous or 12-month current mood disorder compared to participants with no reported mood disorder

^a Model 1: adjusted for age^b Model 2 CDAH-2: females adjusted for age, social support occupation, education, marital status, and BMI; males adjusted for age, social support, occupation, education, marital status, smoking status, parental status, sleep hours, leisure-time physical activity, and BMI

CDAH-3: females adjusted for age, social support, occupation, education, marital status, smoking status, parental status, sleep hours, and leisure-time physical activity, and BMI; males adjusted for age, social support, occupation, education, marital status, smoking status, sleep hours, leisure-time physical activity, and BMI

Supplementary Material for the article “Associations between diet quality and DSM-IV mood disorders during young- to mid-adulthood among an Australian cohort”

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Table S1 Dietary Guidelines Index scoring matrix for men and women aged 19-50 years based on the 2013 Australian Dietary Guidelines

Dietary Guideline	Indicator and Description	Max score	Criteria for maximum score		Criteria for minimum score
			Men	Women	
Adequate intake					
1. Variety of nutritious foods.	1. Intake of foods from each of the five core food groups	10	Two points for a serving from each of the five core food groups. <1 serving receives appropriate proportion of the 2 points.		0 servings from any of the core food groups
2. Vegetables, including legumes/beans.	2. Servings of vegetables per day including legumes/beans, excluding fried potato.	10	≥6	≥5	0 servings
3. Fruit.	3. Servings of fruit per day (max 125ml 100% fruit juice, one serving of dried/sweetened fruit)	10	≥2	≥2	0 servings
4. Grain (cereal) foods, mostly wholegrain and/or high fibre.	4a. Servings of breads and cereals per day.	5	≥6	≥6	0 servings
	4b. Servings of wholegrains as a proportion of total grains. ^a	5	100%	100%	0%
5. Protein foods. Lean meat and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans.	5a. Servings of meats or alternatives per day	5	≥3	≥2.5	0 servings
	5b. Proportion of lean meats/ alternatives to total meat and alternatives	5	100%	100%	0%
6. Dairy or alternatives. Milk, yoghurt, cheese or alternatives, mostly reduced fat.	6a. Servings per day of total dairy or alternatives.	5	≥2.5	≥2.5	0 servings
	6b. Proportion of reduced fat dairy or alternatives to total dairy or alternatives	5	Skim, low, or reduced fat milk or alternatives		Whole milk
7. Drink plenty of water	7a. Servings per day of fluids, excluding alcohol.	5	≥10	≥8	0 servings
	7b. Proportion of water to total fluid intake per day, excluding alcohol.	5	≥50%	≥50%	0%

Limit intake					
8. Limit intake of saturated fat, added salt, added sugars and alcohol.	8. Servings per day of alcohol, or foods high in saturated fat, added sugars or salt ^b .	20	≤1.5	≤1.25	Men: >3, Women: >2.5
9. Replace saturated fats with unsaturated fats	Trimming fat from meat	5	Usually		Never/ rarely
	Type of spread usually used for bread/crackers	5	Spreads low in saturated fat		Spreads high in saturated fats
Total:		100			

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^a Calculated for bread only as other cereal foods in the food frequency questionnaire (e.g. rice, pasta) did not specify wholegrain quality

^b Servings of discretionary choices are only recommended for active, taller adults, or older children, and therefore, the number of servings for the maximum score for discretionary items is less than or equal to half the recommended servings for the age group and sex

Table S2 Prevalence ratios of mood disorder within 12 months prior to follow-up for a 10-point higher Dietary Guidelines Index score among complete-case participants at Childhood Determinants of Adult Health (CDAH) study follow-ups at CDAH-1 (2004-06), CDAH-2 (2009-11), and CDAH-3 (2014-19)

	% with mood disorder	(n/N)	Model 1 ^a		Model 2 ^b		Model 3 ^c		Model 4 ^d	
			PR ^e	(95% CI)	PR	(95% CI)	PR	(95% CI)	PR	(95% CI)
CDAH-1										
Females	11.5	(33/288)	0.96	(0.65, 1.44)	1.00	(0.67, 1.50)	1.02	(0.70, 1.49)	1.00	(0.65, 1.53)
Males	11.2	(24/214)	1.20	(0.66, 2.18)	1.19	(0.67, 2.13)	1.31	(0.76, 2.26)	1.35	(0.77, 2.36)
CDAH-2										
Females	11.1	(32/288)	1.02	(0.68, 1.53)	0.99	(0.65, 1.51)	1.01	(0.67, 1.52)	1.00	(0.67, 1.47)
Males	5.6	(12/214)	1.16	(0.88, 1.54)	1.14	(0.85, 1.52)	1.14	(0.81, 1.59)	1.23	(0.83, 1.83)
CDAH-3										
Females	5.6	(16/288)	0.88	(0.65, 1.19)	0.96	(0.69, 1.33)	1.02	(0.72, 1.45)	1.02	(0.73, 1.44)
Males	6.5	(14/214)	0.91	(0.68, 1.23)	0.97	(0.65, 1.44)	1.03	(0.70, 1.52)	1.25	(0.78, 2.01)

* $p < 0.05$

PR: prevalence ratio; CI: confidence interval

^a Model 1: adjusted for age

^b Model 2: CDAH-1: females adjusted for age, smoking status, occupation, leisure-time physical activity; males adjusted for age, smoking status

CDAH-2: females adjusted for age, occupation, education, marital status; males adjusted for age, occupation, education, marital status, smoking status, parental status, sleep hours, and leisure-time physical activity

CDAH-3: females adjusted for age, occupation, education, marital status, smoking status, parental status, sleep hours, and leisure-time physical activity; males adjusted for age, occupation, education, marital status, smoking status, sleep hours, and leisure-time physical activity

^c Model 3: as per Model 2, plus Social Support

^d Model 4: as per Model 3, plus BMI

^e The PR value is for a 10-point higher Dietary Guidelines Index (DGI) score (range 0-100). A higher DGI score indicates better diet quality

Table S3 Prevalence ratios of mood disorder within 12 months prior to follow-up by a 1-point higher Dietary Guideline Index (DGI) component score, estimated by log-binomial regression at the third Childhood Determinants of Adult Health (CDAH-3) study in 2014-19

Sex and DGI component	Model 1 ^a		Model 2 ^b		Model 3 ^c	
	PR ^d	(95% CI)	PR	(95% CI)	PR	(95% CI)
Females						
Variety	0.87	(0.74, 1.01)	0.95	(0.82, 1.11)	0.96	(0.82, 1.12)
Vegetables	0.87	(0.78, 0.98)*	0.92	(0.84, 1.01)	0.92	(0.83, 1.01)
Fruit	0.94	(0.87, 1.01)	0.98	(0.92, 1.06)	0.99	(0.92, 1.06)
Grain	0.86	(0.78, 0.96)*	0.90	(0.80, 1.02)	0.91	(0.80, 1.03)
Protein	1.02	(0.82, 1.26)	1.12	(0.90, 1.40)	0.99	(0.91, 1.07)
Dairy or alternatives	0.96	(0.87, 1.05)	0.99	(0.89, 1.09)	1.10	(0.91, 1.33)
Water	0.96	(0.87, 1.07)	0.99	(0.88, 1.12)	0.99	(0.88, 1.10)
Limit discretionary foods	1.00	(0.97, 1.04)	1.02	(0.98, 1.05)	1.02	(0.99, 1.05)
Limit saturated fat	0.98	(0.92, 1.05)	0.97	(0.90, 1.04)	0.96	(0.89, 1.03)
Males						
Variety	0.92	(0.73, 1.17)	1.00	(0.77, 1.31)	1.02	(0.77, 1.35)
Vegetables	0.81	(0.64, 1.03)	0.84	(0.67, 1.06)	0.84	(0.66, 1.07)
Fruit	0.93	(0.84, 1.03)	0.93	(0.82, 1.06)	0.94	(0.83, 1.08)
Grain	1.03	(0.89, 1.20)	0.99	(0.85, 1.15)	1.02	(0.86, 1.20)
Protein	0.76	(0.60, 0.95)*	0.83	(0.65, 1.06)	0.84	(0.65, 1.10)
Dairy or alternatives	1.03	(0.92, 1.14)	1.09	(0.97, 1.23)	1.09	(0.97, 1.22)
Water	0.88	(0.77, 1.00)	0.96	(0.82, 1.12)	0.96	(0.83, 1.12)
Limit discretionary foods	0.99	(0.94, 1.05)	1.02	(0.96, 1.08)	1.01	(0.96, 1.08)
Limit saturated fat	0.99	(0.90, 1.10)	0.99	(0.89, 1.10)	0.99	(0.89, 1.09)

* $p < 0.05$

PR: prevalence ratio; CI: confidence interval

^a Model 1: adjusted for age, and total DGI score minus component score

^b Model 2: females adjusted for age, total DGI score minus component score, occupation, education, marital status, smoking status, parental status, sleep hours, leisure-time physical activity, social support; males adjusted for age, total DGI score minus component score, occupation, education, marital status, smoking status, sleep hours, leisure-time physical activity, social support

^c Model 3: as per Model 2, plus BMI

^d The PR value is for a 1-point higher component score. All components have possible scores of 0-10 points except “Limit discretionary foods” which has a possible score of 0-20 points. Discretionary foods include processed items such as cakes and biscuits, packaged snack foods, processed meats, fast-food, and sugar-sweetened and alcoholic beverages

Table S4 Univariable cross-sectional regression of the Dietary Guidelines Index score and log-binomial regression of mood disorder on Social Support Index score at follow-up: CDAH-1 (2004-06), CDAH-2 (2009-11), and CDAH-3 (2014-19)

	<i>n</i>	Outcome Measure ^a			
		Dietary Guidelines Index ^b		Mood disorder	
		β^c	95% CI	PR ^b	95% CI
CDAH-1					
Females	991	0.21	(0.12, 0.31)*	0.93	(0.92, 0.93)*
Males	983	0.14	(0.03, 0.25)*	0.92	(0.91, 0.94)*
CDAH-2					
Females	955	0.15	(0.06, 0.24)*	0.93	(0.93, 0.94)*
Males	525	0.04	(-0.08, 0.16)	0.93	(0.91, 0.95)*
CDAH-3					
Females	655	0.28	(0.15, 0.41)*	0.93	(0.91, 0.95)*
Males	536	0.25	(0.13, 0.36)*	0.93	(0.91, 0.94)*

* $p < 0.05$

CDAH: Childhood Determinants of Adult Health study; PR: prevalence ratio; CI: confidence interval

^a Predictor variable: Henderson Index of Perceived Social Support: range 15-75. A higher score indicates higher self-perceived social support

^b Dietary Guidelines Index: range 0-100. A higher score indicates better diet quality

^c β : change in Dietary Guidelines Index score for every 1-unit of change in the Social Support score