

Youth diet quality and hazard of mood disorder in adolescence and adulthood among an Australian cohort.

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Abstract

Background

Prospective studies on youth diet and mood disorders outcomes are limited. We examined if youth diet quality was associated with mood disorder onset over a 25-year follow-up period.

Methods

In 1985, Australian participants (aged 10-15 years) completed a 24-hour food record. A validated 100-point Dietary Guidelines Index (DGI) assessed diet quality. In 2009-11, 1005 participants (aged 33-41 years) completed the lifetime Composite International Diagnostic Interview for age of first DSM-IV defined mood disorder (depression or dysthymia). Cox proportional hazards regression estimated hazard of mood disorder during the 25-year follow-up according to baseline DGI score. Sensitivity analyses censored the study at 5, 10, and 15 years after baseline and used log binomial regression to estimate relative risk (RR). Covariates included baseline negative affect, BMI, academic performance, smoking, breakfast eating, physical activity, and socioeconomic status.

Results

The mean(SD) youth DGI score was 45.0(11.5). A 10-point higher DGI was not associated with hazard of mood disorder onset over the 25-year follow-up (Hazard Ratio (HR):1.00; 95% Confidence Interval (CI):0.89-1.13). The only indication that higher DGI might be associated with lower risk of mood disorder was within the first 5 years after baseline and this was not statistically significant (RR=0.85; 95% CI:0.60-1.18).

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39 Limitations

40 Loss-to-follow-up. A single 24-hour food record may not represent usual diet.

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42 Conclusion

43 Youth diet did not predict mood disorders in adulthood. The suggestions of a lower risk of
44 mood disorder during late adolescence highlights that further prospective studies are needed.

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46 **Key words:** child; adolescent; DSM-IV; depressive disorder; affective disorder; nutrition

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Introduction

The mood disorders depression and dysthymia are highly prevalent conditions with complex aetiologies that may involve a combination of genetic, environmental, lifestyle and situational factors. Diet is thought to influence mental health via nutritional effects on biochemical pathways related to hormones, neurological signalling, inflammation, and microbiota and the gut-brain axis (Lang et al., 2015). If there is a true association, modification of diet and nutrition could be important for prevention or treatment of depressive symptomology. Mood disorders often have their first onset in adolescence or early adulthood (World Health Organization, 2013), meaning youth may be a crucial time for prevention or early intervention. Several systematic reviews have supported the likelihood of a cross-sectional association between healthier dietary intake and lower prevalence of depressive symptoms among adults (Quirk et al., 2013; Lai et al., 2014; Rahe et al., 2014; Liu et al., 2016), and to a limited extent among children and adolescents (O'Neil et al., 2014; Khalid et al., 2016), but there is less consistent evidence of a prospective relationship. The diet-mood relationship is likely to be somewhat bi-directional due to mood influencing food choices, which means that longitudinal studies are required to explore directionality of this relationship (Lopresti et al., 2013; Jacka et al., 2015).

Prospective studies among children and adolescents (youth) often have short follow-up periods, use general questions about dietary practice rather than assess overall diet, or look at

Abbreviations: ASHFS, Australian Schools Health and Fitness Survey; BMI, body mass index; CDAH, Childhood Determinants of Adult Health study; CI, confidence interval; CIDI, Composite International Diagnostic Interview; DGI, Dietary Guidelines Index; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; FFQ, food frequency questionnaire; FHQ, food habits questionnaire; HR, hazard ratio; NDSS, National Dietary Survey of Schoolchildren; RR, relative risk; SES, socioeconomic status.

diverse mental or behavioural disorders under broad categories rather than formal diagnoses (O'Neil et al., 2014; Khalid et al., 2016). For example, outcomes of interest in prior studies have included emotional functioning, internalising problems (e.g. depression, anxiety), depressive symptoms (Winpenny et al., 2018) and psychological distress (Jacka et al., 2013; Wu et al., 2016). A Canadian study used administrative health data to determine clinically diagnosed mood disorders, but grouped them with neurotic or general anxiety disorders, severe stress reaction, and child-onset emotional disorders as internalising disorders (McMartin et al., 2012; Wu et al., 2017). To our knowledge, there are only four studies (from three cohorts) among the general population, not related to eating disorders or specific medical conditions, that have tracked the effects of youth overall diet on depressive symptoms for longer than three years, and none have examined effects beyond late adolescence (McMartin et al., 2012; Wu et al., 2016; Wu et al., 2017; Cong et al., 2020). Only three prospective studies have reported associations between aspects of diet or dietary behaviours (but not overall diet) and mental disorders that were robust to covariate adjustment (Jacka et al., 2011; McMartin et al., 2012; Wu et al., 2016). The relationship between diet and mood disorders is worth investigating further due to the high prevalence and widespread burden of the disorders on individuals and society.

There are several mechanisms by which diet in youth may influence long-term mental health outcomes. Firstly, poor diet may have long-term biological effects due to nutrient deficiencies in the case of undernutrition (such as iron or omega-3 fatty acids required to support neurological functioning), or inflammation or metabolic derangement in the case of overnutrition and excess energy intake (Hale et al., 2015). Secondly, food preferences and dietary behaviours are developed in youth, within the context of socioeconomic and

sociocultural circumstance and shape long-term dietary preferences into adulthood, which may affect or mediate adult health (Mikkila et al., 2005).

The Australian Childhood Determinants of Adult Health (CDAH) cohort study facilitates examination of associations between youth diet at 10-15 years of age, and adult mood disorders during a 25-year follow-up period. Our objective was to examine if diet quality in youth was associated with first onset of mood disorders, determined according to diagnostic criteria, using survival analysis. We hypothesised that better diet quality in youth would be associated with reduced hazard of mood disorder during the follow-up period.

Methods

Participants

The 1985 Australian Schools Health and Fitness Survey (ASHFS) was a nationwide survey of schoolchildren aged 7-15 years by the Australian Department of Community Services and Health. To achieve a nationally representative sample, a two-stage probability design was used. There was a 90.1% school response rate, with 109 of the 121 approached schools participating. The survey aimed for 500 students of each sex at ages 7 to 15 years, to permit estimates from the questionnaire data that would be within 10% of the population means. The overall student response rate was 67.6% ($n = 8498$ out of 12578 students approached) [16]. ASHFS participants aged 10-15 years ($N = 5589$) were invited to participate in the 1985 National Dietary Survey of Schoolchildren (NDSS).

During 2001-02, ASHFS participants were traced and invited to participate in the CDAH study. This resulted in 5170 participants enrolling in the CDAH study (61.0%) (Gall et al., 2009), including 3188 participants who had participated in the NDSS. The first follow-up was held in 2004-06 (CDAH-1, data not used for this analysis). During the second follow-up in 2009-11 (CDAH-2), 1144 of the NDSS participants, then aged 33-41 years, completed postal questionnaires and a lifetime mental health diagnostic interview over the telephone.

The State Directors General of Education approved the ASHFS. Participation required signed parental consent. The CDAH study protocol was approved by the Southern Tasmanian Health and Medical Ethics Committee. All participants gave informed written consent.

Dietary measures

During the 1985 NDSS, the participants completed a 24-hour food and drink record. Trained data collectors showed students in groups of four or five how to measure and record their intake in a record booklet with the aid of circles, rulers, and metric cups and spoons that the students were given to keep. The 24-hour recording period started immediately after a practice exercise. When the record books were collected, each student was interviewed to check the entries. The Department of Community Services and Health coordinated the survey design, collected and processed the food record data with assistance from the Dietitians Association of Australia (English et al., 1988).

Food intakes from the 1985 NDSS food record were converted to equivalent proportions of standard daily serves of food and beverage items. For example, 250ml of milk is one standard

serving of dairy, therefore, a report of 125ml of milk in the 24-hour food record was equivalent to 0.5 standard servings of dairy. Diet quality was calculated using a validated Dietary Guidelines Index (DGI) (Wilson et al., 2019) that reflected the age- and sex-specific recommended food group servings in the 2013 Australian Dietary Guidelines (National Health and Medical Research Council, 2013). The Australian Dietary Guidelines are food-based guidelines that share fundamental similarities with other guidelines worldwide to achieve at least minimum intakes of a variety of whole foods, and limit sugar, fat and salt (Herforth et al., 2019). The DGI scores overall diet using seven scoring indicators that align with the guidelines (dietary variety and recommended intakes of vegetables, fruit, grains, lean meats and alternatives, dairy and alternatives, water) and two indicators that reflect guidelines to limit intake (saturated fat, and discretionary items (food and drinks high in saturated fat, added salt and sugar, and alcohol)) (Wilson et al., 2019). A higher score on the range of 0-100 indicates better alignment with the 2013 Australian Dietary Guidelines and therefore better diet quality.

Mood disorder

Mental health was assessed at CDAH-2 using the lifetime version of the Composite International Diagnostic Interview (CIDI) (World Health Organization, 1997). Trained interviewers administered the computerised CIDI over the telephone to collect data on depressive symptoms and age of first onset. Symptoms were scored using Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria (American Psychiatric Association, 2000) to determine experiences of dysthymia or major depressive disorder. Participants were categorised as having or not having a mood disorder. The date of first onset was calculated by the participant's date of birth plus the age of onset.

Covariates

All covariates were measured at baseline when participants were aged 10-15 years. The ASHFS questionnaire, completed concurrently with the NDSS, included questions on demographics, lifestyle, health attitudes, and sport and exercise history. Data collectors administered the questionnaires to groups of four students at a time and assisted with explaining questions as needed. The following data were used in this analysis: age in years; ever smoked (never, <10 cigarettes, ≥ 10 cigarettes); breakfast eating (usually eat something before school on 4 or more days per week, don't usually eat), frequency of alcohol drinking (never, less than weekly, weekly or more), and total hours of physical activity per week calculated from physical activity to, from, outside and during school over the previous week. Negative affect was measured using four Bradburn Scale items (McDowell and Praught, 1982), which asked if during the past few weeks the participants felt: lonely, depressed or unhappy, bored, or upset due to criticism, with response options of "often", "sometimes" or "never". Possible scores range from 0 (low negative affect) to 8 (high negative affect).

Socioeconomic status (SES) quartiles (high, medium-high, medium-low, low) were determined according to postcode of residence by the Australian Bureau of Statistics Socio-Economic Index for Areas and 1981 census data (Australian Bureau of Statistics, 2013). School type (public, Catholic, or independent) was used as an additional indicator of SES. Public schools in Australia are primarily government funded, whereas Catholic and independent schools rely on comparatively more private funding. Academic performance was reported by the student's school (excellent, above average, average, below average, poor).

Height and weight were measured with participants wearing light clothing and no shoes or socks. KaWe height tape or rigid measuring tape was used to measure height to the nearest 0.1cm. Beam or medical spring scales were used to measure weight to the nearest 0.5kg. BMI was calculated as $\text{weight (kg)} / (\text{height (m)})^2$. Cole's age- and sex-specific cut points were used to classify youth as being non-overweight, overweight or obese (Cole et al., 2000).

Statistical analysis

Statistical analyses were performed using Stata version 15.0 (StataCorp, College Station, Texas, 2017). Means and standard deviation (SD) are reported for continuous variables, and percentages and frequency are reported for categorical variables. The outcome variable was age of first onset of a mood disorder. The effects of DGI score were examined using both the continuous score, and score thirds (Low, Middle, High). The score thirds were defined using score tertiles from the entire baseline NDSS cohort with dietary data so that they reflected low, middle and high scores among the nationally representative sample. For interpretability, the continuous DGI scores were reduced by a factor of 10, meaning that the observed change in the regression coefficient was associated with a 10-point higher DGI score.

Survival analysis was used to consider the effects of diet quality on time-to-event (mood disorder). Participants were considered to be at risk from the date they completed the NDSS, and the timescale was years until onset of first mood disorder or censoring. Participants were right censored at the date of participation in CDAH-2 if no mood event was reported. Survival curves for the three score categories were estimated and plotted using the Kaplan-Meier method (Kaplan and Meier, 1958). Cox proportional hazards regression analysis was

performed to calculate the hazard ratio (HR) and the 95% confidence interval (CI) of mood disorder onset since baseline, predicted by DGI score. Schoenfeld and scaled Schoenfeld residuals were used to check for violation of the proportional hazards assumption by testing for independence between residuals and time (Schoenfeld, 1982). Violation of the proportionality assumption was determined if the tests of proportionality results were significant (P -value <0.05), and by visual examination of the graph of the scaled Schoenfeld residuals. If the proportional hazards were violated, a stratified Cox model was used where the covariate was not included in the model, but used as a stratification variable, allowing the baseline hazards to vary according to each level of the covariate. A stratified Cox model uses the strata variable within the model and produces a single final set of coefficients optimised to fit all strata, rather than executing separate models for each variable level.

Sensitivity analyses were conducted by performing log binomial regression to estimate the relative risk and 95% CIs of mood disorder onset within the first 5, 10, and 15 years after baseline, and for the whole follow-up period to 2009-11. The rationale for this analysis was to determine the cumulative relative risk during these intervals, and if diet was associated with mental health outcomes more proximal in time as previous studies measured mental health outcomes closer in time to the dietary measures (Jacka et al., 2011; McMartin et al., 2012; Jacka et al., 2013; Wu et al., 2016).

Multiple imputation was used, where necessary, to complete the 1985 ASHFS data for missing data on variables that predicted loss-to-follow-up, and then inverse probability weighting using these variables was applied to the regression analyses (motivated by Seaman et al., 2012). Multiple imputation was not used to complete missing covariate data.

Participants were excluded if they were missing covariate data used in the fully adjusted models. Minimally adjusted models (Model 1) were adjusted for sex and baseline age. Covariates for the fully adjusted models (Model 2) were selected based on empirical evidence of associations between mood disorder outcomes and socioeconomic status (Gilman et al., 2002; Elovainio et al., 2012), lifestyle/behavioural factors (Kubik et al., 2003; Lopresti et al., 2013; Schuch et al., 2017), anthropometry (Luppino et al., 2010), and youth negative effect (Fombonne et al., 2001). Model 2 was adjusted for baseline age, area-level SES, school type, BMI category, smoking status, drinking of alcohol, breakfast eating, physical activity, and Bradburn negative affect score. The academic performance variable violated the proportional hazards assumption and therefore this variable was used as a stratification variable rather than a covariate within Model 2 in the Cox regression.

Results

At baseline, 5043 (90.2%) of the eligible 5589 10-15-year old ASHFS participants completed the NDSS (**Figure 1**). Reasons for not participating included: declined to participate, were absent from school on the day of the booklet distribution/collection, or forgot to return or complete the food record. At CDAH-2, 1144 (22.7%) of the 5043 NDSS participants completed the CIDI, with 291 (25.4%) of the 1144 reporting a mood disorder. Participants were excluded if they reported that their first onset of a mood disorder occurred prior to the NDSS ($n = 15$) or were missing covariate data ($n = 124$). The final survival analysis sample was $n = 1005$, with 245 participants (24.4%) reporting mood disorders. Baseline population characteristics of both the original NDSS cohort and the final study sample are shown in **Table 1** for comparison. There was considerably greater loss-to-follow-up of male compared

to female participants, with males comprising 51.2% of the original NDSS cohort, but only 38.4% of the final study sample. The mean (SD) DGI score for the study sample was 45.0 (11.5) compared to 44.6 (12.0) among the NDSS cohort, indicating slightly greater loss to follow-up among those with lower compared to higher DGI scores. Among the NDSS cohort at baseline there were 1681 participants in each DGI score third, whereas among the final study sample there was 306 participants in the lowest third, 339 in the Middle third, and 360 in the High third. The DGI score tertiles 38.8 and 48.6 were used to define the score thirds of the 5043 NDSS participants.

Interaction terms between DGI scores and sex in Cox regression models were not significant (interaction of sex with: continuous DGI $P = 0.861$, Middle DGI third $P = 0.526$, High DGI third $P = 0.703$), so males and females were analysed together. The Kaplan Meier survival curve is shown in **Figure 2**. The survival curve shows that those in the highest third of scores have slightly better probability of no mood disorder onset (“survival”) within the first 10 years than those in the Middle and Low thirds, whereas during the 10-20 years after baseline, there is no difference between the High and Middle thirds, and after 20 years after baseline the Middle third has the highest probability of no mood disorder. The Middle score series crossing the Low and High score series could be an indication of violation of the proportional hazards assumption although the results of the Schoenfeld residual test of proportional hazards do not support this (Middle third $P = 0.978$; High third $P = 0.449$).

Results of the Cox proportional hazards regression shown in **Table 2** indicate that there was no difference in hazard of onset of mood disorder over the 25-year follow-up period associated with a 10-point increase in the continuous DGI score in Model 1 (Hazard Ratio

(HR)=0.99; 95% CI: 0.84, 1.17) or Model 2 (HR=1.00; 95% CI: 0.89, 1.13). Although the regression results by score third reflect the Kaplan-Meier plot by showing hazard ratios <1.0 for the Middle and High score thirds compared to the Low score third, these results did not approach statistical significance in either Model 1 (Middle HR= 0.91, 95% CI:0.65, 1.28; High HR= 0.92, 95% CI:0.67, 1.27) or Model 2 (Middle HR= 0.94, 95% CI: 0.66, 1.33; High HR= 0.96, 95% CI: 0.69, 1.32).

The sensitivity analysis results of relative risk estimates to examine cumulative risk during the periods proximal to baseline, are shown in **Table 3**. None of these results reached statistical significance. There were 28 mood disorder onsets within the first 5 years after baseline, 68 within the first 10 years, and 119 within the first 15 years. A 10-point higher DGI score was associated with a non-significant lower relative risk of mood disorder within the first 5 and 10 years after baseline (5 years: Model 2 RR= 0.85, 95% CI: 0.60, 1.18; 10 years Model 2 RR= 0.95, 95% CI:0.76, 1.18). The first 5-years after baseline was the only period during which there was any indication of a linear trend in the direction of lower risk associated with higher score category, but again, this was not statistically significant (Middle RR= 0.77, 95% CI: 0.30, 1.95; High RR= 0.48, 95% CI: 0.18, 1.31). There was no reduction in relative risk for a 10-point higher DGI score by 15 years after baseline (Model 2 RR= 1.00, 95% CI: 0.86, 1.17) or over the whole follow-up period (Model 2 RR=1.01, 95% CI: 0.92, 1.12).

Discussion

Our results do not support the hypothesis that higher diet quality in childhood is associated with reduced hazard of mood disorder in adulthood. Although visualisation of the data

suggests that those with medium or high diet quality at baseline have lower probability of mood disorder onset during the 25-year follow-up period compared to those with the lowest diet quality, diet did not have an independent or significant effect on the relative hazard or risk of mood disorder. Our results indicate there may be a “ceiling” effect of youth diet quality, where those with scores in the middle range had outcomes equal to or better than those with high scores, but these differences were too small to be statistically significant. These small differences may have also contributed to our inability to detect evidence of violation of the proportional hazards assumption from the Schoenfeld test results. This is despite those in the Middle third appearing to have better outcomes than those in the High third later in the follow-up period whereas during the earlier years they appeared to have poorer probability of survival. The only slight suggestion of a linear effect of youth diet quality was on the most proximal mood disorder outcomes within 5 years after the dietary measure. Although none of the following results were statistically significant and could be due to chance, when the follow-up period was censored at 5 years, the direction of effect indicated a 15% lower risk of mood disorder for a 10-point higher DGI score, and for the categorised scores, a 23% lower risk for those in the Middle score category and 52% lower risk for those in the High category compared to those with lowest scores.

A motivation for our study was consideration that there may be long-term biological effects of poor nutrition that influence mood disorder risk. Diet quality often reflects a social gradient (Vlismas et al., 2009) and socioeconomic factors in youth have been shown to persist into adulthood and be associated with depressive symptoms in adulthood (Gilman et al., 2002; Elovainio et al., 2015). The mechanism of how SES relates to depression risk is not fully understood, but could be due to a combination of the psychological and lifestyle impacts (including diet quality and nutritional deficiencies) of SES level on emotional and

neurocognitive development (Hackman et al., 2010). The mean DGI scores at baseline were quite low, indicating that dietary intake was not a good reflection of a healthy and nutritionally adequate diet as outlined by the 2013 Australian Dietary Guidelines (National Health and Medical Research Council, 2013). The Kaplan-Meier plot and results by score third does appear to indicate that those with the lowest DGI scores (<38.8), which could indicate nutritional deficiencies, had higher risk of mood disorders over the 25-year follow-up period, but these differences were not significant. The generally low DGI scores could be due to the 2013 Guidelines not reflecting the food cultures and food availability of 1985. It is also possible the overall dietary score from a single day's dietary measure is not nuanced enough to indicate nutritional deficiencies predictive of long-term neurological outcomes, or that dietary changes during the 25-year follow-up and later adult diet may have mitigated any risk.

The secondary motivation for our study was the hypothesis that diet in youth is likely to shape diet in adulthood. On the other hand, the transition from childhood and adolescence into adulthood is a period during which diet can alter considerably as the individual becomes more independent from their parents or carers. Changes in diet quality and other factors related to mood disorder outcomes were not able to be assessed within the scope of this current study. However, it was observed that the strength and direction of the effect estimates in the primary and sensitivity analyses remained similar after covariate adjustment and this could be because the baseline covariates were not meaningfully associated with the outcome over the long follow-up period. This highlights the importance of repeat and consistent measures during prospective studies.

The results of the sensitivity analyses that truncated the follow-up period to 5 and 10 years gave slight indications as to the direction of a possible effect (lower risk associated with better diet quality). This direction of effect is plausible given the results of previous studies. Associations, including those attenuated after adjustment, between diet and mental health outcomes among adolescents have been observed in studies with short follow-up periods of 2-4 years (Jacka et al., 2011; McMartin et al., 2012; Jacka et al., 2013; Winpenny et al., 2018). A 10-year Taiwanese study that reported a bidirectional relationship between unhealthy eating and depression symptoms only examined the associations year-to-year and not over the entire period, and highlights the change in eating behaviours and depressive symptoms during adolescence (Wu et al., 2016). In our study, the 5- and 10-year censored results were based on only 28 and 69 cases of mood disorder onset respectively and did not reach statistical significance, but the strength and direction of the effect estimates remained after covariate adjustment. However, we are cautious of overstating any possible reduced risk in the 5- and 10-year periods subsequent to the youth measures. As well as the lack of statistical significance, the strength of effect should be carefully considered when interpreting the findings. For example, if the relative risk estimate in the 5-year sensitivity analysis was the true effect and not due to chance or residual confounding, the 15% reduction in risk was for a 10-point increase in DGI score. As the mean baseline DGI score was quite low at 45 points, a 10-point increase is a considerable improvement in diet quality. Furthermore, translating observed effects from epidemiological studies into meaningful prevention and intervention strategies is a considerable challenge that must be overcome to see real changes in population health outcomes.

The lack of statistical significance of our results are not unexpected. Systematic reviews of associations between diet and mental health in youth have reported that there is some

consistency in evidence of cross-sectional associations but evidence of a prospective association is limited (O'Neil et al., 2014; Khalid et al., 2016). It is possible that null and weak results in longitudinal studies are due to the influence of later or long-term diet. This is perhaps also why there have been stronger results of a relationship between diet and depressive risk from prospective studies involving adults whose lifestyle and environmental context may be more static, and dietary measures more indicative of long-term food and nutrient intake (Quirk et al., 2013; Lai et al., 2014; Rahe et al., 2014). Studies reporting higher depression risk associated with a long-term diet high in processed foods and saturated fat suggest that this may be due to chronic inflammation and poor immune functioning (Berk et al., 2013; Jacka et al., 2014). Youth have had less time to develop and experience these chronic states, and improving diet quality or reducing BMI before adulthood may also mitigate future risk. Among the CDAH cohort for example, females who were overweight in childhood and non-obese in adulthood did not have a different risk of mood or anxiety disorder compared to women who had been non-obese at both timepoints, whereas being overweight or obese in youth and adulthood was associated with mood disorder (Sanderson et al., 2011). Young adult dietary factors have also been shown to be associated with mood disorders. Women who reported eating fish more than twice a week at CDAH-1 had lower risk of a mood disorder episode over the 5-year follow-up to CDAH-2 compared to women who ate fish less than twice per week (Smith et al., 2014). The risks of mood disorder associated with physical health conditions such as obesity or poor nutrition may be tempered by extent and duration of the condition or dietary practices.

This study has several limitations. The baseline dietary survey was a 24-hour food record intended as a 'snapshot' of Australian schoolchildrens' diet in 1985 and may not be an adequate measure of participants' usual diet in youth. Diet can vary over time and is not a

single event to mark the starting point of time to event (e.g. in traditional survival analysis, disease diagnosis is often the starting point of the period at risk). This poses difficulties for prospective studies where diet is measured intermittently, particularly for this study where there was a 25-year gap between baseline and the measure of lifetime mood disorders. Many other lifestyle and external events could occur during the follow-up period and contribute to the development of depressive symptoms. Significant loss-to-follow-up, and greater loss of participants with lower baseline diet quality and particular characteristics (e.g. male, lower SES) may have also biased results, although the final sample retained participants with a similar distribution of most characteristics as shown in Table 1. Furthermore, the effects of loss-to-follow-up was mitigated by inverse probability weighting in the regression analyses, using the baseline cohort and variables that predicted participant drop-out. The lifetime mood disorder prevalence of 24% was higher than the latest available (2007) national Australian estimates of lifetime affective disorders among 18-65 year olds of 15% (Australian Bureau of Statistics, 2008), which although is not for a directly comparable age group, could indicate sample bias and limit generalisation of results. Retrospective self-reporting of lifetime mood by our participants in their late 30s and early 40s may also have introduced some bias. However, the diagnostic classification and age of onset information from the CIDI has been validated and found to be reliable (Wittchen, 1994).

Strengths of the study include the use of the CIDI as it is a high quality ‘gold standard’ instrument for retrospective measurement of mental disorders in epidemiological studies (Steel et al., 2014). It is a diagnostic interview identifying mood disorder according to globally used and recognised clinical diagnostic criteria from the DSM-IV. Exclusion of participants with first mood onset prior to baseline, and inclusion of baseline negative affect and other potential confounders such as BMI and SES in the analysis is also a strength and

addresses a common recommendation from systematic reviews (Quirk et al., 2013; Lai et al., 2014; Rahe et al., 2014; Molendijk et al., 2018). Another strength was that we used an overall measure of diet, which is important as discrete measures such as intake of individual nutrients or individual practices such as take-away food consumption do not account for the influence of other aspects of the diet or the non-nutritional qualities of food such as dietary fibre which may have synergistic effects on health (Ocké, 2013). Furthermore, the DGI is based on core recommendations that are nearly universal among dietary guidelines internationally (Herforth et al., 2019), which enhances the generalisability of results outside of Australia. Although the study was a secondary analysis of data, which contributed to several of the limitations outlined above, the range of measures and longitudinal structure enabled analysis of an important research question among a unique cohort.

In conclusion, diet quality in youth was not associated longitudinally with mood disorder outcomes 25 years later. There were indications that higher diet quality in youth may be associated with lower risk of mood disorder at more proximal times in later adolescence and very early adulthood, but our results were not statistically significant and therefore may be due to chance. Further prospective research to better assess associations with long-term diet quality beginning in youth and over follow-up periods extending beyond adolescence, would benefit from regular and repeat dietary and covariate measures, diagnostic mood disorder measures, and analysis of the proximity of dietary measures to future or past mood events.

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Conflict of interest

The Authors declare that there is no conflict of interest.

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Table 1. Characteristics of participants at baseline in 1985.

Variable	Study sample (<i>n</i> = 1005)		Baseline cohort (<i>N</i> = 5589)	
	Mean or %	(SD) or (n/N)	Mean or %	(SD) or (n/N)
Sex				
Female	61.6	(619/1005)	48.8	(2729/5589)
Male	38.4	(386/1005)	51.2	(2860/5589)
Age (years)	12.5	(1.7)	12.4	(1.7)
DGI score ^a	45.0	(11.5)	44.6	(12.0) ^e
Bradburn negative affect ^b	2.8	(1.7)	2.9	(1.7) ^e
Physical activity (hrs/wk) ^c	7.4	(6.6)	7.6	(7.1) ^e
BMI category				
Non-overweight	91.6	(921/1005)	88.7	(4952/5584)
Overweight	7.2	(72/1005)	9.8	(545/5584)
Obese	1.2	(12/1005)	1.6	(87/5584)
Area-level SES				
High	26.6	(267/1005)	23.2	(1242/5365)
Medium-High	27.5	(276/1005)	29.0	(1555/5365)
Medium-Low	39.5	(397/1005)	38.6	(2071/5365)
Low	6.5	(65/1005)	9.3	(497/5365)
School type				
Public	74.4	(748/1005)	74.9	(4184/5589)
Catholic	20.1	(202/1005)	19.8	(1109/5589)
Independent	5.5	(55/1005)	5.3	(296/5589)
Academic performance				
Excellent	15.5	(156/1005)	9.3	(487/5224)
Above average	35.7	(359/1005)	27.3	(1428/5224)
Average	37.5	(377/1005)	41.4	(2165/5224)
Below average	10.5	(105/1005)	16.7	(874/5224)
Poor	0.8	(8/1005)	5.2	(270/5224)
Ever smoked				
Never	52.5	(528/1005)	51.3	(2791/5440)
<10 cigarettes	34.9	(351/1005)	32.8	(1782/5440)
≥ 10 cigarettes	12.5	(126/1005)	15.9	(867/5440)
Drink alcohol				
Never	65.7	(660/1005)	65.2	(3547/5442)
Less than weekly	26.7	(268/1005)	26.7	(1452/5442)
Weekly or more	7.7	(77/1005)	8.1	(443/5442)
Usually eat breakfast ^d				
Usually eat	86.1	(865/1005)	84.5	(4590/5435)

Don't usually eat	13.9 (140/1005)	15.6 (845/5435)
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^aDietary Guidelines Index: higher score on range of 0-100 indicates higher diet quality.

^bBradburn negative affect scale, higher score on range of 0-8 indicates higher negative affect.

^cPhysical activity to, from, during, and outside of school ($n = 1005$).

^dUsually eat breakfast defined as eating breakfast four or more days per week.

^eDGI: $n = 5043$; Bradburn negative affect: $n = 5323$; Physical activity: $n = 5400$.

Table 2. Hazard ratios (HRs) and 95% confidence intervals (CI) of mood disorder^a onset from 1985 baseline (10-15 years of age) to 2009-11 follow-up (33-41 years of age), for baseline Dietary Guidelines Index (DGI) score.

	Percentage with mood disorder (n/N)	Model 1 ^b		Model 2 ^c	
		HR (95% CI)	<i>P</i>	HR (95% CI)	<i>P</i>
DGI ^d	24.4% (245/1005)	0.99 (0.84, 1.17)	0.917	1.00 (0.89, 1.13)	0.940
Score third					
Low	27.1% 83/306	Reference		Reference	
Middle	21.8% 74/339	0.91 (0.65, 1.28)	0.607	0.94 (0.66, 1.33)	0.712
High	24.4% 88/360	0.92 (0.67, 1.27)	0.625	0.96 (0.69, 1.32)	0.783

^aMood disorder defined as first onset of dysthymia or major depressive disorder.

^bAdjusted for sex and age.

^cAllowing baseline hazards to vary by academic performance category, and adjusted for sex and baseline age, BMI, smoking, breakfast eating, alcohol drinking, area level SES, school type, and physical activity.

^dContinuous DGI score, reduced by factor of 10. Hazard ratio is for a ten-point higher DGI score (range 0-100) where higher score indicates better diet quality.

Table 3. Log binomial regression risk ratios and 95% confidence intervals (CI) of mood disorder^a onset at 5, 10, and 15 years after the 1985 baseline and at CDAH-2 follow-up (2009-11), for baseline Dietary Guidelines Index (DGI) score.

	Percentage with mood disorder	Model 1 ^b		Model 2 ^c	
	(n/N)	RR (95% CI)	<i>P</i>	RR (95% CI)	<i>P</i>
To 5 years after baseline (1990)					
DGI ^d	2.7% (27/1005)	0.84 (0.59, 1.19)	0.331	0.85 (0.60, 1.18)	0.329
Score third					
Low	3.3% (10/306)	Reference		Reference	
Middle	2.9% (10/339)	0.78 (0.30, 2.00)	0.602	0.77 (0.30, 1.95)	0.581
High	1.9% (7/360)	0.47 (0.17, 1.33)	0.157	0.48 (0.18, 1.31)	0.153
To 10 years after baseline (1995)					
DGI ^d	6.8% (68/1005)	0.92 (0.73, 1.16)	0.506	0.95 (0.76, 1.18)	0.626
Score third					
Low	8.2% (25/306)	Reference		Reference	
Middle	6.2% (21/339)	0.76 (0.42, 1.38)	0.369	0.81 (0.44, 1.50)	0.506
High	6.1% (22/360)	0.75 (0.42, 1.36)	0.348	0.79 (0.44, 1.41)	0.430
To 15 years after baseline (2000)					
DGI ^d	11.8% (119/1005)	1.00 (0.85, 1.16)	0.952	1.00 (0.86, 1.17)	0.967
Score third					
Low	13.4% (41/306)	Reference		Reference	
Middle	11.5% (39/339)	0.98 (0.63, 1.52)	0.932	1.01 (0.63, 1.60)	0.976
High	10.8% (39/360)	0.83 (0.54, 1.29)	0.408	0.84 (0.54, 1.32)	0.458
To CDAH-2 follow-up (2009-11)					
DGI ^d	24.4% (245/1005)	1.00 (0.90, 1.10)	0.939	1.01 (0.92, 1.12)	0.781
Score third					
Low	27.1% (83/306)	Reference		Reference	
Middle	21.8% (74/339)	0.93 (0.70, 1.23)	0.595	0.97 (0.72, 1.31)	0.840

High	24.4% (88/360)	0.96 (0.73, 1.26)	0.746	1.00 (0.75, 1.33)	0.991
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^aMood disorder defined as first onset of dysthymia or major depressive disorder.

^bAdjusted for sex and age.

^c Adjusted for sex and baseline age, BMI, smoking, breakfast eating, alcohol drinking, area level SES, school type, academic performance, and physical activity.

^d Continuous DGI score, reduced by factor of 10. Risk ratio reflects a ten-point change in DGI score (range of 0-100) where higher score indicates better diet quality.

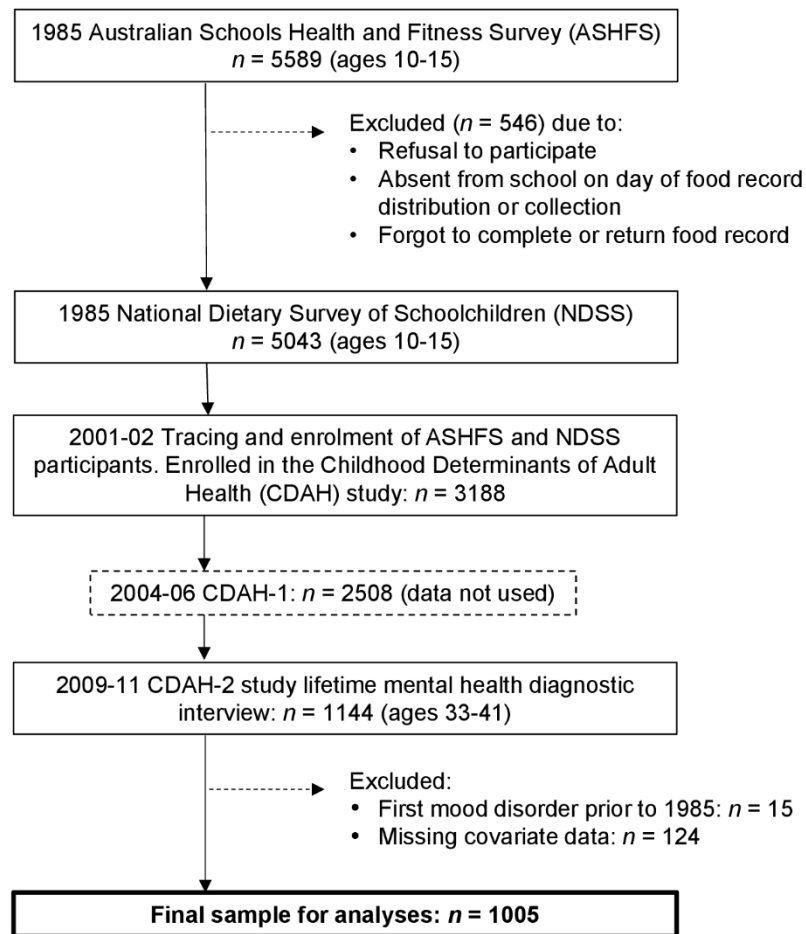


Figure 1. Flow diagram of the Childhood Determinants of Adult Health (CDAH) cohort participants from 1985 baseline to follow-up in 2009-11.

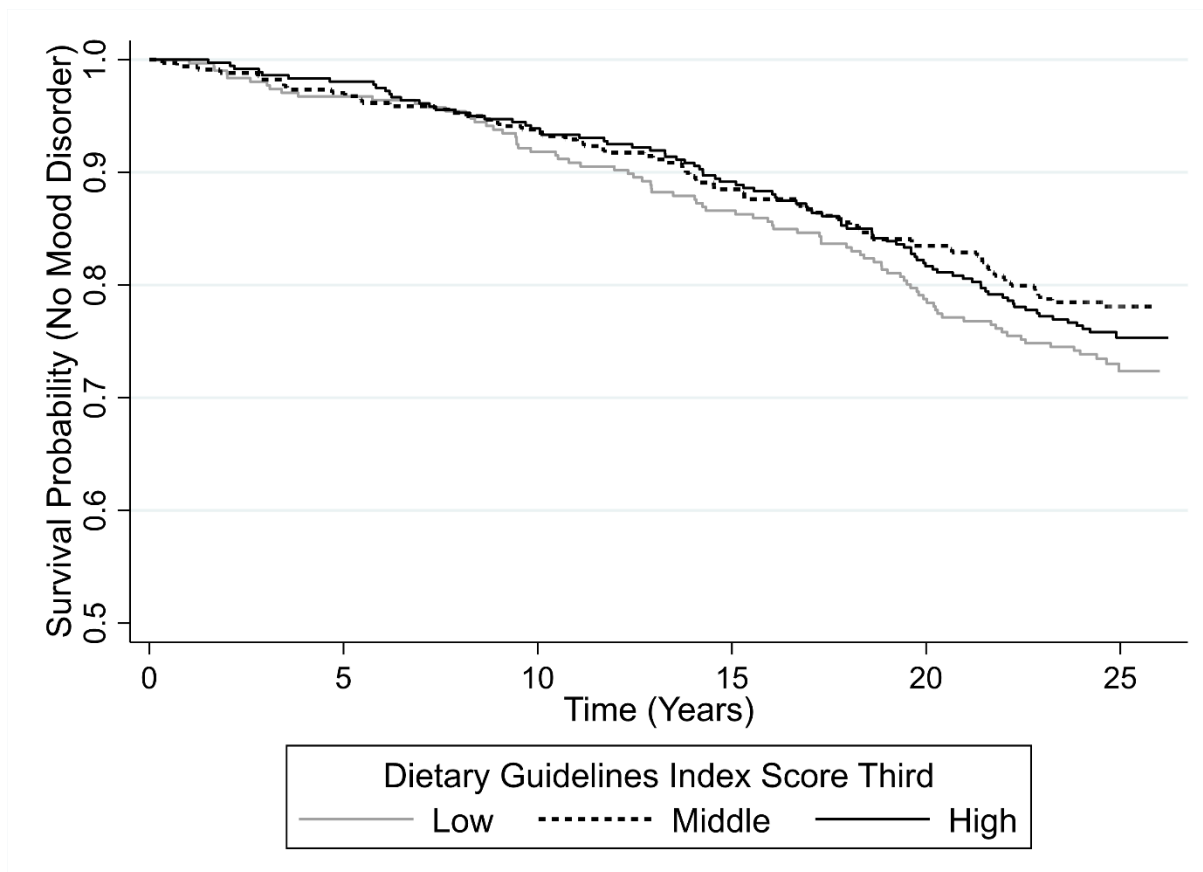


Figure 2. Kaplan-Meier survival estimates of not having mood disorder onset by Dietary Guidelines Index score third (Low, Middle, High).