Associations between diet and mood disorders during youth and young- to mid-adulthood

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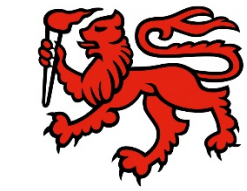

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Declaration of originality

This thesis contains no material which has been accepted for a degree or diploma by the University or any other institution, except by way of background information and duly acknowledged in the thesis, and to the best of my knowledge and belief no material previously published or written by another person except where due acknowledgement is made in the text of the thesis, nor does the thesis contain any material that infringes copyright.

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Statement of co-authorship

This thesis uses data from the Childhood Determinants of Adult Health (CDAH) study, which comprises the baseline 1985 Australian Schools Health and Fitness Survey (ASHFS), 1985 National Dietary Survey of Schoolchildren (NDSS), and three follow-ups during 2004-06, 2009-11, and 2014-19. Johanna Wilson (the Candidate) was not involved in the study design but designed the research questions covered in this thesis based on the data available. The Candidate had sole responsibility for cleaning the 1985 NDSS dietary data, merging it with the ASHFS data, identifying and categorising the NDSS food codes. The Candidate also assisted with data collection at study clinics in Perth, Western Australia in 2018 during the third CDAH follow-up.

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Statement of ethical conduct

The research associated with this thesis abides by the international and Australian codes on human and animal experimentation, the guidelines by the Australian Government's Office of the Gene Technology Regulator and the rulings of the Safety, Ethics and Institutional Biosafety Committees of the University. Ethics Approval Numbers for the Childhood Determinants of Adult Health study: H6020, H8152, H0010454 and H0013826

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Abstract

Background: The mood disorders depression and dysthymia are highly prevalent conditions that affect an individual's capacity to function and participate in society. Dietary intake supports neurobiological functions and may influence mental health outcomes. There is consistent evidence of cross-sectional associations between better diet quality and lower prevalence of depressive symptoms, but there are few prospective studies, particularly among child or adolescent (herein referred to as youth) cohorts. Moreover, some dietary behaviours related to timing of meals (e.g., skipping breakfast) are understood to be associated with diet quality and may influence hormones and neurobiology. However, there are few longitudinal studies on associations between timing of eating occasions and mood disorders. There are also few longitudinal studies that have examined associations between overall diet or dietary behaviours and mood disorder outcomes defined by diagnostic criteria. Due to the high prevalence of mood disorders and in some cases, limited efficacy of or access to clinical and pharmaceutical treatments, it is important to consider the lifestyle factors such as diet that could help prevent or modulate mood disorders.

Aims: To develop a consistently structured measure of diet quality for use among both children and adults, and examine whether diet quality and time-of-day eating patterns were associated with mood disorder outcomes that met diagnostic criteria, during youth and adulthood among an Australian cohort.

Methods: The research aims were examined through four original studies using data from the Childhood Determinants of Adult Health (CDAH) study. The CDAH study comprises data from a nationwide health, fitness and dietary survey of 8,498 Australian schoolchildren aged 7-15 years in 1985, and three follow-ups in adulthood between 2004 and 2019. Key measures include: a baseline 24-hour food record in youth; food frequency and food habit questionnaires in adulthood; structured diagnostic interviews in adulthood to identify 12-month (at all three follow-ups) and lifetime prevalence (at second and third follow-up) of DSM-IV major depressive disorder and dysthymic disorder; and a range of demographic, socioeconomic, psychosocial, and physical covariates at baseline and follow-up.

Results: The first study involved development and validation of a sex- and age-specific Dietary Guidelines Index (DGI) that measured adherence to the Australian Dietary Guidelines. The DGI was found to be an adequate measure of diet quality among youth and young adults and the DGI scores highlighted that, on average, diet quality among the cohort was poor.

In the second study it was found that diet quality in youth was not associated longitudinally with onset of mood disorders in adulthood. Similarly, the third study found that diet quality in adulthood was not associated cross-sectionally or longitudinally with mood disorders. However, there was a non-significant association between better diet quality in youth and a lower risk of mood disorder in late adolescence/early adulthood in the second study, and significant cross-sectional associations in the minimally adjusted analyses in mid-adulthood in the third study, suggesting further research is needed.

The fourth study found that in adulthood, higher adherence to a “Late” time-of-day eating pattern, characterised by delayed or skipped breakfast, was associated with a higher prevalence of mood disorder, and the relationship may be bidirectional.

Conclusion: There was very limited evidence of longitudinal associations between diet and risk of mood disorders. Continuing work on public health campaigns and regulation of the food industry to support individuals to make healthy food choices, is required to improve diet quality and meal habits among the Australian population. This could help lower mood disorder risks due to improvements in physical and psychosocial health, but further prospective research is needed to determine if dietary intake is independently and causally associated with mood disorder outcomes.

List of Abbreviations

ACHPER - Australian Council for Health, Physical Education and Recreation Inc.

APA – American Psychiatric Association

ASHFS – Australian Schools Health and Fitness Survey

BDNF - brain derived neurotrophic factor

BMI – body mass index

CDAH – Childhood Determinants of Adult Health

CI – confidence interval

CIDI – Composite International Diagnostic Instrument

CRP – C-reactive protein

DGI – Dietary Guidelines Index

DSM – Diagnostic Statistical Manual

FFQ – food frequency questionnaire

FHQ – food habits questionnaire

HDL – high density lipoprotein

HOMA-IR - Homeostasis Model Assessment of Insulin Resistance

HPA - hypothalamic-pituitary-adrenal

HR – hazard ratio

IL-6 - interleukin-6

ICD – International Classification of Diseases

LDL – low density lipoprotein

NDSS – National Dietary Survey of Schoolchildren

OR – odds ratio

PCA – principal component analysis

PR – prevalence ratio

RR – risk ratio

SES – socioeconomic status

UK – United Kingdom

US – United States

WHO – World Health Organization

Chapter 1: Introduction

The emerging field of nutritional psychiatry, which examines the relationship between diet and psychiatric (mental) disorders, has gained considerable academic, clinical and media attention in recent years. This thesis aims to help advance the field by examining associations between diet and mood disorders among community-living individuals during different life stages from youth to adulthood.

1.1 Aims and objectives

The aim of this thesis was to determine associations between diet and mood disorder outcomes during childhood/adolescence (youth) and young- to mid-adulthood among an Australian cohort. The high prevalence of chronic mental disorders, is recognised as a global issue requiring coordinated action (1). Identifying contributing factors to poor mental health is therefore crucial and is the rationale for this thesis. Data from the longitudinal Childhood Determinants of Adult Health (CDAH) cohort study provided an opportunity to examine dietary intake and dietary practices in relation to mood disorder outcomes over time.

In support of the primary aim, the thesis objectives were:

- Review the literature pertaining to the influence of diet on mood disorders.
- Use CDAH data to:
    - Examine cross-sectional diet quality of the cohort during youth and adulthood.
    - Determine if diet quality in youth is longitudinally associated with subsequent onset of mood disorders during adulthood.
    - Examine associations between diet quality and prevalence (cross-sectional analysis) and risk (longitudinal analysis) of mood disorders during young- to mid-adulthood.
    - Examine bidirectional associations between timing of daily food intake and mood disorders during young- to mid-adulthood.

The resulting original studies contribute novel perspectives and results to the emerging nutritional psychiatry field, which has an important role in shaping the diet-disease narrative and providing evidence to support public health policy to maximise wellbeing of populations.

1.2 Background, definitions, and scope

1.2.1 Mood disorders

Mood disorders, also commonly known as affective or depressive disorders, can be unipolar and involve only depression, or bipolar and involve alternated episodes of depression and mania. The focus of this thesis is on the unipolar disorders major depressive disorder and dysthymia due to the high prevalence and burden of these disorders outlined later in this section. As well as lower prevalence, bipolar disorders have characteristics associated with mania phases that are distinct from unipolar depressive conditions and may impact differently on behaviour and lifestyle choices, and entail different pharmaceutical treatments and associated effects (2, 3). The lower prevalence of bipolar disorders and thus possible low statistical power in later analyses combined with the necessitation to limit the number of measures collected from participants, meant that only unipolar depressive disorders were assessed at the CDAH follow-ups.

Major depressive disorder can take the form of single or recurrent episodes and be mild, moderate or severe, while dysthymic disorder is a chronic form of mild depression (4). Clinical diagnosis of a mood disorder is often achieved using criteria outlined in the American Psychiatry Association's (APA) Diagnostic and Statistical Manual of Mental Disorders, currently in its fifth edition (DSM-V) (5). The DSM-V defines depressive disorders as disorders that have "…the presence of sad, empty, or irritable mood, accompanied by somatic and cognitive changes that significantly affect the individual's capacity to function" (6). The DSM criteria for mental disorders are commonly used worldwide. An alternate classification system is the World Health Organization (WHO) International Classification of Diseases (ICD), currently in its 11th revision, in which definitions of mental disorders are now broadly aligned to the DSM-V (7). In addition to clinical use, the standard criteria of these diagnostic manuals enable consistent identification and measurement of

mental disorders for epidemiological and health management purposes (8). This thesis uses DSM-IV (9) criteria (Table 1.1) to identify mood disorders among the study cohort.

Table 1.1 Diagnostic Statistical Manual, fourth edition (DSM-IV) criteria for major depressive disorder and dysthymic disorder in adults

Disorder	Symptoms
Major Depressive Disorder Five or more symptoms must have been present for 2 weeks or more and represent a change in the person's functioning. At least one symptom is either 1. depressed mood or 2. decreased interest or pleasure.	1. Depressed mood most of the day, nearly every day. Either subjective report (e.g. feels sad or empty) or observation made by others (e.g. appears tearful). 2. Decreased interest or pleasure in all, or almost all, activities most of the day, nearly every day. 3. Significant weight gain or loss (when not dieting) of 5% of body weight in a month, or change in appetite. 4. Change in sleep: Insomnia or hypersomnia nearly every day. 5. Change in activity: Psychomotor agitation or retardation nearly every day. 6. Fatigue or loss of energy nearly every day. 7. Feelings of worthlessness or excessive or inappropriate guilt nearly every day. 8. Diminished ability to think or concentrate nearly every day, or increased indecisiveness. 9. Recurrent thoughts of death or suicidal ideation.
Dysthymic Disorder Depressed mood for most of the time for at least 2 years, with presence of 2 or more symptoms. Never without symptoms for 2 months or more over a 2-year period, and symptoms must cause clinically significant distress or impairment. There has been no manic, hypomanic, or mixed episodes, and is not better accounted for by major depressive disorder.	1. Poor appetite/weight loss, or weight gain. 2. Insomnia or hypersomnia. 3. Fatigue or loss of energy. 4. Low self-esteem. 5. Diminished ability to think, concentrate, or make decisions. 6. Feelings of hopelessness.

Note: Adapted from Table 1, Primary DSM-IV depression disorders, criteria for adults by O'Connor EA, Whitlock EP, Gaynes B, et al. Screening for Depression in Adults and Older Adults in Primary Care: An Updated Systematic Review [Internet]. Rockville (MD): Agency for Healthcare Research and Quality (US); 2009 Dec. (Evidence Syntheses, No. 75.) Available from: https://www.ncbi.nlm.nih.gov/books/NBK36406/table/ch1.t1/

The DSM-IV was the edition current at the time of the first CDAH follow-up and diagnostic mental health interview. However, some studies referenced in this thesis have used other editions of the DSM, or the ICD and therefore the reader should be aware that there are some differences. For example, whereas in the DSM-IV dysthymic disorder is named as such, in the DSM-V, dysthymia is named as "persistent depressive disorder", which includes what was previously known as dysthymia as well as chronic major depressive disorder (11).

Depressive disorders are the third leading cause of disability worldwide after low back pain and headache disorders (12). In Australia, depressive disorders are the third leading non-fatal condition contributing to years lived with a disability, following back pain and anxiety disorders (13). There is greater proportional impact earlier in life, with mental and substance abuse disorders being the leading non-fatal disease burden among Australian adolescents and adults under 45 years of age (13). The most recent national Australian survey of adults that used diagnostic instruments to determine mental ill health was the National Survey of Mental Health and Wellbeing 2007 (14). This study estimated that among the Australian population aged 16-85 years, the 12-month prevalence of mild, moderate or severe depressive episodes was 5.1% among females and 3.1% among males, and the lifetime estimate (those who reported ever having a depressive episode) was 14.5% among females and 8.8% among males (Figure 1.1) (14). There is evidence that these conditions have early onset. The second national Australian Child and Adolescent Survey of Mental Health and Wellbeing was conducted during 2013-14 (15). The results highlighted the high prevalence of depressive disorders among adolescents, particularly among older adolescents, with 19.6% of females and 8.2% of males aged 16-17 years reporting a depressive episode during the past 12 months (Figure 1.1) (15).

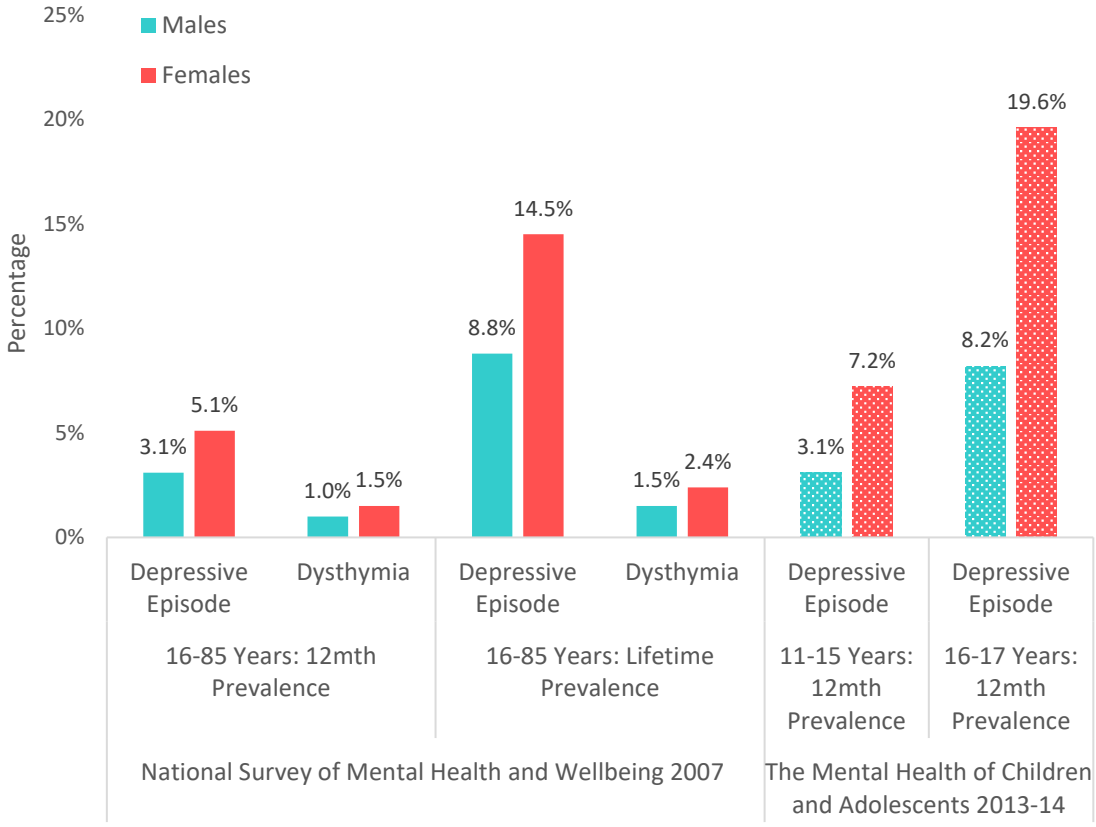


Figure 1.1 Prevalence of mood disorders among Australian populations from the National Survey of Mental Health and Wellbeing 2007, and the Mental Health of Children and Adolescents 2015

These disorders have a range of negative impacts on the individual and their social and family relationships, have economic costs due to lost productivity, and in extreme cases, may lead to suicide. Therefore, it is crucial that potential causal factors such as diet are thoroughly investigated, particularly among youth cohorts prior to condition onset, and over time, as they may identify opportunities for prevention or intervention. The scope of this thesis focuses on associations between dietary intake and mood disorders in terms of depressive symptomology of clinical significance. This thesis does not explore associations between food intake and effects on short-term mood or vice versa (e.g. clinical studies on whether certain food or nutrients influence immediate mood).

1.2.2 Diet quality

Diet refers to the food and drinks that a person habitually consumes. Diet quality is commonly used in terms of how well an individual's dietary intake reflects notions of

a ‘healthy’ diet – often in relation to adherence to national dietary guidelines or other evidence-based concepts of nutrition (16). A healthy diet is usually defined as one with high intakes of minimally processed food that are nutrient dense but not energy dense (e.g. vegetables, fruits, wholegrains, lean proteins), and low in processed food that are energy dense but low in nutrients, have added salt, or are high in added sugars or saturated fat (17). Studies focusing on individual foods or nutrients and associated mental health outcomes may be helpful in understanding specific neurobiological mechanisms, but singular foods or nutrients are not eaten in isolation, and food or drink commonly eaten in combination may have synergistic effects on health (18). Therefore, diet quality, derived from measures of overall dietary intake of foods and food groups, is used as the primary dietary variable of interest in the original studies in Chapter 4, Chapter 5, and Chapter 6 in this thesis. A review of methods for measuring and assessing overall diet is provided in the Chapter 2 Literature Review, Section 2.3.

1.2.3 Timing of daily food intake

Food choices and when and how frequently food is consumed during the day are determined by complex interactions of social, cultural, physical, environmental, economic, and psychological influences (19). Beyond the nutritional composition of foods that determines diet quality, dietary behaviours around the timing and frequency of intake (sometimes referred to as chrononutrition) may also impact on physical and mental health due to metabolic and hormonal effects (e.g., insulin sensitivity) (20). Timing of daily food intake is often studied in relation to discrete behaviours such as skipping meals (particularly skipping breakfast), snacking, eating late at night, or adhering to an intermittent fasting regime. This thesis examines timing of daily food intake and associations with mood disorders to gain a better understanding of how dimensions of diet separate to nutritional intake could also influence health outcomes. Further background information on the health effects of timing of food intake, methods, and literature pertaining to food timing and mental health outcomes, is outlined in the Chapter 2 Literature Review, Section 2.7.

1.2.4 Nutritional psychiatry

Although the influence of nutrition and diet in relation to physical health is well-established, the influence in relation to mental health is less well understood. Mental ill-health has a neurological basis and complex aetiology from a variety of contributing factors which may be biological, psychological, social, or environmental (21, 22). During the past few decades, the field of nutritional psychiatry has emerged, focused on determining how nutrition relates to mental health outcomes, and providing evidence on whether diet may assist with prevention and treatment of mental disorders at both the clinical and public health level (23). Although there are pharmacological treatments for depressive disorders, they can have limited efficacy (particularly for milder cases), are for treatment after diagnosis rather than prevention of first onset, and may have negative side effects (24). Evidence of associations between diet and mental disorders and plausible pathways of effect suggest that diet is a modifiable lifestyle factor that could provide opportunities to modify risk of poor mental health (25). The Chapter 2 Literature Review addresses key aspects of the nutritional psychiatry literature, including the foundational work on how dietary components of foods (e.g., micro and macronutrients) may influence neurobiology pertaining to mental health, methods for assessing diet and mood disorder, and a review of the literature examining overall diet and mood disorders among both youth and adults, focusing on longitudinal studies.

1.2.5 Nutritional epidemiology

Epidemiology is the study of the distribution and determinants of health or disease within a population. Nutritional epidemiology is a subdiscipline of epidemiology specifically focused on the relationship between diet (nutritional exposures) and disease within a population (26). Key, long-standing challenges in nutritional epidemiology include the reliable measurement of diet as a continuous exposure, and controlling for confounding factors that could obscure the diet-disease relationship (27).

This thesis is primarily concerned with observational nutritional epidemiological studies, with a focus on longitudinal cohort studies of community-living individuals, rather than clinical or experimental (intervention) studies that study modification of

diet for prevention or treatment of mental disorders. Intervention studies, ideally high quality randomised control trials, can entail careful sample selection to reduce confounding and allow greater control or measures of diet for hypothesis testing and to infer causality, but often use small samples, examine single exposures and are short-term due to costs and onus on participants, which limits generalisability of results to broader populations and to the public health setting (28). The majority of nutritional epidemiology research and the building blocks of the nutritional psychiatry field are observational studies. Observational studies can provide good evidence of associations in 'real world' settings from large samples and over long timeframes, but also have many challenges. These challenges include balancing the accuracy and efficiency of measuring diet and confounding factors among larger participant populations, and for longitudinal studies, loss of participants over time - limitations that are outlined in the discussion of each of the original studies in Chapters 4-7, and the Chapter 8 Summary.

As highlighted in the Chapter 2 Literature Review, there remain gaps in the literature concerning consistent evidence of prospective associations between diet quality and mood disorders, particularly disorders that meet diagnostic criteria. The original studies of this thesis aim to address that gap and contribute to the epidemiological understanding of the diet-mood disorder relationship that informs public health messaging.

1.3 Thesis Outline

Chapter 2 outlines literature on the relationship between diet and mood disorders, including background on the influence of nutrition on neurobiological processes, and common methodologies for measuring dietary intake and mental disorders in observational studies. The review of epidemiological research focuses on results of longitudinal cohort studies among youth and participants in young- to mid-adulthood. The methods of the CDAH study and measures used in this thesis are outlined in Chapter 3. Chapters 4-7 report on the following original studies:

- Chapter 4: An age- and sex-specific dietary guidelines index is a valid measure of diet quality in an Australian cohort during youth and adulthood.
- Chapter 5: Youth diet quality and hazard of mood disorder in adolescence and adulthood among an Australian cohort.
- Chapter 6: Associations between diet quality and DSM-IV mood disorders among an Australian cohort during young- to mid-adulthood.
- Chapter 7: An eating pattern characterised by skipped or delayed breakfast is associated with mood disorders among an Australian adult cohort.

Chapter 8 summarises the key outcomes, future directions and conclusions arising from the literature review and original studies.

Chapter 2: Literature Review

2.1 Introduction

Nutritional epidemiology studies may examine health outcomes in relation to intake of individual nutrients or foods, or in relation to overall dietary intake. Individual nutrients can influence specific biological functions so it is important to study their effects, but assessing overall diet is now common practice due to recognition that foods and combinations of foods have variable potential for digestion, absorption, metabolism of nutrients, and impact on the gut microbiome (18, 29). This chapter:

- Provides an overview of nutrients and foods related to the neurobiology of mental health.
- Outlines common dietary and mental disorder measurement and evaluation methods.
- Reviews studies that have examined overall dietary intake and mood disorder outcomes among youth and adults.
- Reviews studies on mood disorder outcomes associated with timing of daily food intake.

The type of studies reviewed are largely observational, with a focus on prospective cohort studies. Although randomised control trials are generally considered the 'gold standard' for testing hypotheses, as outlined in Chapter 1, they are more expensive, usually involve small samples over short periods of time, and have difficulty ensuring dietary compliance among community living individuals due to the complex nature of dietary behaviour (28). Whereas observational studies can assess a wider range of measures and outcomes among larger populations over extended periods of time in more natural (real world) settings. Moreover, this review is directed at identifying gaps in the observational study literature that this thesis, with the CDAH cohort data, may be able to address.

2.2 Nutrients, biochemical pathways and mental health

This section of the review outlines the mechanisms whereby nutritional intake influences neurobiological processes that are thought to be related to mental health outcomes. Although this thesis focuses on the relationship between overall diet quality and mood disorders, it is important to understand how components of overall diet may influence mental health. Micro or macronutrient deficiencies (or excess, e.g., sugars) due to poor diet quality may mean that certain neurobiological functions are not adequately supported (30). Specific nutrients of interest include omega-3 fatty acids, folate, B-vitamins, zinc, magnesium, vitamin D, and selenium (31). Research in recent years has also highlighted pro- and anti-inflammatory effects of particular nutrients and their possible role in supporting mental health (32, 33). There is also emerging evidence around the role of the gut microbiome in metabolic and inflammatory responses related to mental disorders, and how diet can alter populations of microorganisms for positive or detrimental effects (34).

2.2.1 Micronutrients

Micronutrients are vitamins and minerals that are required by the body in very small amounts to support biological processes and good health. Although many essential and non-essential vitamins and minerals are required in conjunction to support proper biological functions, cell growth and signalling, the following are a few of the more commonly highlighted nutrients thought to influence mental health outcomes through neurobiological pathways.

B-vitamins

B-vitamins including B6, B12 and folate are thought to protect against elevated homocysteine levels in the blood which can lead to inflammation of the blood vessels, a risk factor for cardiovascular disease which is often a comorbidity of mood disorders, particularly among older adults (35). Folate and B12 are also required for synthesis of monoamine neurotransmitters (dopamine, noradrenaline and serotonin), which are important bioactive substances required for many physiological and homeostatic functions, including regulating mood (36). Foods that are good sources of both folate and B6 include green leafy vegetables, legumes, poultry, fortified grains, and some fish. B12 which is synthesised by certain bacteria,

is found in animal products such as poultry, meat, fish, eggs and dairy products, as well as some fortified grain and non-dairy milk products.

Magnesium

Magnesium is necessary for energy metabolism, is a cofactor for many enzymatic reactions, and may be associated with mental health outcomes by helping block calcium from entering neurons, thus protecting against cell death (37). Food sources rich in magnesium are green leafy vegetables, legumes, nuts and seeds, wholegrains, and some fish.

Zinc

Zinc is thought to influence mental health due to its role in neurotransmitter activity, as an antioxidant, and in proliferation of neuronal precursor cells (38). Foods high in zinc include seafood, meat and poultry, spinach, some nuts and seeds, and egg yolk.

Vitamin D

Vitamin D plays a crucial role in several hormonal and neurological processes. However, it is widely acknowledged that food sources (such as oily fish), are not sufficient to meet the body's needs, and adequate sunlight exposure is required for conversion of compounds in the skin to Vitamin D (39).

Selenium

Selenium is an antioxidant that limits damage to cells by free-radicals (particularly damage to cell lipids by peroxidation), and supports neurotransmitter function, immune function, and regulation of thyroid hormones (40). Foods high in selenium include brazil nuts, fish and seafood, meat, poultry, eggs, and fortified wheat products.

2.2.2 Macronutrients

Macronutrients are components of food (carbohydrates, proteins, and fats) required in large amounts in the diet. Discerning the influence of a macronutrient is difficult as other qualities of food (e.g. dietary fibre), quantity, and the foods they are consumed alongside, may be confounding factors. For example, among a cohort of post-menopausal women, diets higher on the glycaemic index (the higher the

glycaemic index, the faster the rise in blood glucose), were associated with increased risk of depressive symptoms over a three year period (41). There was higher risk associated with added sugars, but not total sugars or total carbohydrates, which could indicate that the association is due specifically to the refined carbohydrates and sugars found in high-glycaemic index foods and added sugar foods which may contribute to inflammation (41).

Several essential amino acids (building blocks for proteins) have been linked to mood regulation, such as tryptophan which is used to make serotonin, but the few existing studies on dietary protein intake and associations with mood disorder outcomes have shown inconsistent results (42).

Fats (fatty acids), particularly polyunsaturated fatty acids (PUFA), are the most widely researched macronutrient in relation to mental health. Omega-3 and omega-6 fatty acids are PUFA with a first double bond three (omega-3) or six (omega-6) carbons from the end of the fatty acid chain. Omega-3 is found as short chain alpha-linoleic acid in some nuts and seeds (e.g. walnuts, flax seeds), and long chain eicosapentaenoic acid and docosahexaenoic acid in marine sources (e.g. algae and fish). Omega-3 has potential to be converted in the human body to anti-inflammatory compounds and has been found to affect gut microbiota populations that support immune functioning and production of anti-inflammatory compounds (43). Omega-6 such as linoleic acid, primarily consumed via plant and seed oils have potential to be converted in the body mainly to pro-inflammatory compounds, and therefore a low ratio of omega-6 to omega-3 is thought to be important in controlling inflammation (44, 45). Omega-3 is also understood to have positive effects on serotonergic neurotransmission, and regulation of the hypothalamic-pituitary-adrenal (HPA) axis involved in stress response (46). Brain derived neurotrophic factor (BDNF) is a type of protein that assists neural cell growth and survival. Increased levels of BDNF have been associated with higher omega-3 intake and the Mediterranean diet (high in fish and only moderate intake of omega-6), while imbalances in micronutrients with a high fat or high carbohydrate diet have been associated with reduced BDNF levels (47).

2.2.3 Inflammation

Inflammation is a protective and complex response of bodily tissues to an irritant or pathogen, but chronic inflammation can be detrimental to healthy cells. Chronic inflammation has been associated with a range of poor health outcomes including cardiovascular disease, type 2 diabetes, cancer, and mental and cognitive disorders (48, 49). Elevated pro-inflammatory cytokines (small proteins used for cell signalling) including interleukin-6 (IL-6) and C-reactive protein (CRP), have been associated with depressive symptoms, and the relationship may be bidirectional (49). Cytokines are thought to influence mental disorders by affecting synthesis and release of neurotransmitters, adding to oxidative stress which affects neural cell growth, and impact on normal functioning of the HPA axis (49).

Cross-sectional observational studies have reported associations between "Western" style/meat-based diets high in processed foods and inflammatory markers, and an inverse association between plant-based/whole foods patterns and inflammation (50-52). Adherence to a Mediterranean-style diet has been longitudinally associated with reduced inflammatory markers (53, 54). There are several aspects of the Mediterranean diet which may be protective against inflammation including high intakes of fibre from plant foods, and a lower ratio of omega-6 to omega-3 fatty acids due to olive oil and fish consumption.

2.2.4 Gut microbiome

The bodily and gut microbiotic populations comprise trillions of microorganisms that confer gene expression and functional capacity additional to the human genome. The gut-brain axis is the biochemical communication between the gastrointestinal tract, the bodily nervous systems, and the immune system, which may influence inflammation, stress response, fat storage, and behaviour (55). Although evidence is still emerging and many existing studies are on animal models, there are indications that the microbiome influences stress reactivity, which is implicated in mental disorders such as depression and anxiety (34). Other factors associated with mental disorders that may be influenced by microbiota include dysregulation of the HPA axis due to an undeveloped immune system, activation of stress circuits via the vagal

nerve pathway due to pathogenic bacteria, inflammation due to bacterial imbalances, and influence on BDNF and serotonergic signalling (55).

Changes to diet can lead to shifts in the bacterial populations in as little as 24 hours (56, 57). Diversity in the bacterial community may be important for health, and long-term dietary patterns such as "Western"/animal fat versus plant-based/carbohydrate, have been associated with different bacterial communities and microbial gene expression and metabolic products (57, 58). Certain dietary practices are recognised as beneficial for building communities of beneficial bacteria in the lower gastrointestinal tract, such as consuming soluble fibre and resistant starches (found in plant-based foods) that are prebiotic and provide substrate for fermentation by gut microorganisms to produce fatty acids (59). Emerging evidence regarding the influence of the microbiome highlights the importance of studying overall diet of foods rather than nutrients, to establish optimum diets for good mental health.

2.3 Dietary measurement and assessment methods

This section of the literature review outlines common methods used to measure and assess dietary intake and mood disorders. This will provide background context with which to review the literature on diet-mood disorder relationships.

2.3.1 Dietary data collection

Common methods of dietary measurement used in studies on community-living individuals involve participants self-reporting dietary intake via:

- Dietary recall: Reporting dietary intake over the previous 24-hours or a specified period to a trained interviewer or online.
- Dietary record: Keeping a food diary over a set period such as 1-3 days, possibly repeating several times.
- Food frequency questionnaire (FFQ): non-quantitative (frequency only) or semi-quantitative (also collects portion size information) questionnaire on frequency of usual consumption of a set number of different food and beverage items over a specified period e.g. previous 6 or 12 months.

Each method entails different levels of time and resources for data collection that must be balanced against the risk of reporting bias. Dietary recall methods can capture a large amount of information through open-ended or semi-structured interviews, but rely on participant memory and are very resource intensive, particularly in terms of interviewer training, administration of the interviews, and processing of the data (60). The advent of mobile and web-based dietary reporting tools can help reduce the onus on researchers, particularly in the collation of data, and there are currently several validated applications available, including ones for use among children and adolescents (61). Dietary records kept by participants, particularly where food is weighed, may have lower (but not eliminate) reporting error from misrepresentation of intake amounts, but are also very resource intensive, rely on participants maintaining accurate record keeping (often over several days), and may result in participants modifying what they eat to simplify reporting (62, 63). Both recall and record methods can be repeated to use average values and give a more representative measure of usual diet, but the repetition may also influence diet or reporting to simplify the process (60). FFQs are less resource intensive for researchers and participants, but may introduce greater measurement bias as they cover a set number of food and beverage items or groupings (often around 100 items), rely on participant recall and averaging of intake over lengthy periods, and reporting is often influenced by current dietary intake and may not account for dietary change or seasonal variation if they cover long periods (e.g. up to 12 months) (64). FFQs are the most efficient and often most feasible data collection method for epidemiological studies but should be validated against more intensive methods such as recall or dietary record methods. If FFQs are the primary method of data collection, it has been recommended that validation and calibration sub-studies using recall or dietary record methods are performed (60, 62). However, seasonal variation in dietary intake or usual intake during the past 6-12 months captured by an FFQ, may not be reflected in dietary reporting from a limited number of days.

All three dietary data collection methods rely on accuracy of participant recall or reporting. The prevalence of mis-reporting, particularly under-reporting, is a widely recognised problem (65). Social desirability and social approval bias may underpin some mis-reporting due to participants wanting to present what they consider to be

a socially acceptable diet. The effects may differ among participants by gender or other characteristics, such as females being more likely to under-report intake (66). The potential for measurement error is a common criticism of nutritional epidemiology and therefore it is important that the limitations of the method used are acknowledged, and inform the analysis and interpretation of findings (62). An objective measure of nutritional status not influenced by recall or reporting bias is the use of biomarkers such as serum levels of lipids. However, collection of samples is physically invasive, expensive (particularly if analysing several biomarkers), can be impractical for large studies, and variation in absorption and metabolism between individuals means that it can be difficult to equate these levels to dietary intake and translate them to public health recommendations (67, 68).

2.3.2 Assessment of overall dietary intake

Common methods to assess overall dietary intake include scoring dietary intake against a pre-determined dietary index, or by identifying dietary patterns based on the foods consumed among the study population.

Dietary indices

A dietary index is an à priori measure of how well dietary intake complies with a type of diet (e.g. Mediterranean diet), or a dietary model such as national dietary guidelines. National dietary guidelines are usually devised based on empirical evidence of food and nutrient intakes required for good health and disease prevention, while particular dietary types such as the Mediterranean diet (high in plant foods, legumes, nuts, wholegrains, olive oil and fish) is recognised as a healthy diet model due to consistent associations with reduced risk of overall mortality and disease, specifically cardiovascular disease, cancer and neurodegenerative diseases (69). A dietary index comprises multiple components that assess different aspects of dietary intake (e.g. vegetable intake, grain/cereals intake, type of grains etc), which are then summed to calculate an overall score (70).

Dietary patterns

An à posteriori approach uses dietary intake data to identify patterns of foods that are commonly eaten together within the study population, and score an individual

on how well they adhere to each pattern (commonly two or three main patterns are identified) (71). Derivation of dietary patterns is usually achieved through the data reduction methods of exploratory factor analysis or principal component analysis (PCA). Factor analysis identifies underlying structures in the data and measures latent variables, whereas PCA, which is more commonly used in nutritional epidemiology, determines linear combinations of foods consumed by the study population that explain variation in the reported intakes. Commonly identified dietary patterns using PCA methods include “Western”/”Unhealthy” patterns characterised by high intakes of processed food, red meat, sugars and fats, “Prudent”/”Healthy” patterns characterised by high intakes of whole grains, vegetables and fish, and “Traditional” patterns that vary according to the food culture of the study population (72, 73). Although pattern analysis is an empirical approach, factor analysis and PCA involve evaluation using current literature and subjective interpretation in determining how foods should be grouped for analysis, which patterns should be retained, and how they should be named (73).

Reduced rank regression

An emerging statistical method that combines à priori and à posteriori approaches is reduced rank regression. This method uses nutrients or inflammatory biomarkers thought to be related to the health condition of interest as proxy response variables and applies factor analysis to derive dietary patterns that explain the most variation in the nutrient intakes or biomarkers. The response variables chosen should be hypothesised using à priori knowledge to be on the pathway to the outcome of interest rather than the outcome measures themselves, to avoid forcing correlations between the dietary patterns and outcome measures (74).

2.3.3 Australian Dietary Guidelines Indices

Although empirical methods can be valuable in determining dietary food combinations that are associated with certain health outcomes, an advantage of dietary indices over dietary patterns is that an index is a standard measure that compares what participants are eating to what is recommended for good health, and allows comparison of diet quality over time and between populations (75).

An Australian Dietary Guidelines Index (DGI), comprising 15 components and scoring indicators with a total score range of 0-150, was validated in 2008 as a measure of diet quality by showing associations with sociodemographic characteristics and disease risk factors among an Australian adult population (76). This original DGI was based on the 2003 Dietary Guidelines for Australian Adults (76, 77). A revised DGI, updated to the 2013 Australian Dietary Guidelines and comprising 13 components (with 15 scoring indicators and a total score range of 0-130), was shown in a 2016 study to be associated cross-sectionally with sociodemographic and health behaviour characteristics and body mass index (BMI) among an older adult population (aged 55-65 years) (78). In 2011, a Dietary Guidelines Index for Children and Adolescents (DGI-CA), comprising 11 components and scoring indicators (with a total score range of 0-100) was developed using the same approach to reflect the 2003 Australian Dietary Guidelines for Children and Adolescents in Australia (79, 80). The DGI-CA was validated cross-sectionally among participants aged 4-16 years, using two 24-hour food recalls as the dietary measure, with associations observed between socioeconomic factors and nutritional intakes (79). The differing structures (number of scoring indicators, score range) of these indices, outlined in Table 2.1, highlight the need for an instrument to assess diet in both childhood and adulthood for prospective cohort studies spanning these different life stages. Revising the index for consistency in the scoring approach for use among different age groups, is an important step towards a better understanding of diet quality over time, associated factors, and health outcomes related to diet.

Table 2.1 Summary of Australian Dietary Guidelines Indices

			Scoring Indicator and Maximum Points																
DGI, author	Dietary Guidelines	Age groups (years)[a]	Food variety	Vegetables	Fruit	Cereals	Whole-grain cereals	Meat and meat alternatives	Lean protein	Dairy foods and alternatives	Low fat/reduced fat dairy	Fluids	Limit saturated fat	Small amount of unsaturated fat	Salt use	Limit alcoholic beverages	Limit added sugars	Limit extra foods	Total maximum score
Adults																			
DGI, McNaughton et al (2008) (76)	2003 Dietary Guidelines for Australian Adults (77)	19-60, >60	10	10	10	10	10	10	10	10	10	10	10	N/A	10	10	10	10	150
DGI-2013, Thorpe et al (2016) (78)	2013 Eat for Health: Australian Dietary Guidelines (82)	19-50, 51-70, >70	10	10	10	5	5	5	5	10	5	10	5	10	10	10	10	10	130
Children and adolescents																			
DGI-CA, Golley et al (2016) (79)	2003 Dietary Guidelines for Children and Adolescents in Australia (80)	4-7, 8-11, 12-18	10	10	10	5	5	10	N/A	5	5	10	10	N/A	N/A	N/A	N/A	20	100

DGI: Dietary Guideline Index; DGI-CA: Dietary Guideline Index for Children and Adolescents; N/A: not applicable (indicator was not included in the DGI).
[a] Number of recommended food serves vary by age and sex for some dietary guidelines.

2.4 Mood disorder definition and measurement

The definition and measurement of mood disorders often varies between studies. Many observational studies rely on self-report measures of depressive symptoms, psychological distress, or emotional problems and use different criteria and thresholds to categorise participants as having symptoms or meeting diagnostic criteria of a disorder.

2.4.1 Measures of depressive symptomology

Depression scale instruments comprise a brief set of usually around 10 to 30 items, often based on clinical diagnostic criteria for mood disorders, that are used to calculate an overall score. Two of the more commonly used instruments are the Center for Epidemiologic Study Depression Scale and Beck Depression Inventory II (83). Benefits of scale scores are that the instruments have been widely validated, are time-efficient for researchers and participants, and may allow examination of health impacts on sub-clinical symptoms of depression as well as more severe manifestations (83, 84). However, these non-diagnostic instruments have notable limitations that can limit generalisability of results and consolidation of evidence, including the use of different cut-offs to indicate symptom severity, varying interpretation of scale scores, and different underlying constructs of different instruments (83, 84).

2.4.2 Measures of mood disorders that meet clinical diagnostic criteria

Assessment of depressive symptoms according to widely used and recognised clinical diagnostic criteria for mood disorders such as the DSM, are considered higher quality measures and include structured diagnostic interviews such as the Diagnostic Interview Schedule or Composite International Diagnostic Interview (CIDI) (85, 86). The CIDI is designed to be administered by lay-interviewers or as a computerised interview. Identification of mood disorders using the CIDI has shown good concordance with diagnoses by clinicians, and time related questions required to determine age-of-onset of disorders or prevalence of symptoms within the past 12 months, have shown good reliability (87, 88). Structured interviews take more time to complete and non-computerised versions are more resource intensive for

researchers, but enable categorisation of mental disorder outcomes according to standardised and widely used definitions of the DSM or ICD, and therefore ensure better generalisability and qualification of study results. Use of diagnostic measures in epidemiological studies is regarded as the "gold standard" as it supports linkage of results to fields that focus on specific diagnoses, including biological research studies, clinical psychiatric practice, and health services (89, 90).

Self-report of clinician diagnoses or use of prescription medicine for mood disorders is also used as a measure of disorder diagnosis and has shown reasonable reliability (91). However, not all participants with mood disorders will have sought clinical help, and receipt of a diagnosis could vary according to clinical practice of treating physicians (91). Linkage with administrative health data such as clinical records, prescriptions, or hospital discharges to identify clinical diagnoses is another validated method of identifying depressive disorders, but also risks bias due to varying clinical practices, and not identifying participants who did not engage with health services (92).

2.4.3 Terminology: depressive symptoms versus depressive disorder

Where formal diagnoses of mood disorders such as major depressive disorder or dysthymia are used as the outcome in the following reviewed studies, the outcome will be identified as a "disorder" e.g. depressive disorder. Whereas measures from depression scales or questionnaire items (that may encompass other mental disorders such as anxiety disorders) will be described as depressive symptoms, or other psychological states/conditions such as "negative affect" or "internalising disorder" as appropriate.

2.5 Prospective studies on overall diet quality and mood disorders among youth

The complexities of diet and dietary effects described in Section 2.2 above, highlights the importance of using overall measures of dietary intake to examine associations with health outcomes. Depressive symptomology and mood disorders often have first onset in adolescence or early adulthood (93, 94), and high quality cohort studies could help determine the influence of lifestyle factors such as diet, on disorder onset and outcomes to inform public health prevention and intervention.

Youth is also often a time when dietary habits and behaviours are established and can inform adult diet (95, 96). Therefore, measuring diet in youth and understanding how it tracks into adulthood may help identify associations between diet quality over time and mood disorders.

2.5.1 Prospective studies among youth

There are few longitudinal studies on associations between overall diet quality and mental health among children and adolescents in the general population. Systematic narrative literature reviews from 2014 (97) and 2016 (98) were predominantly of cross-sectional studies but included the same three prospective studies. These reviews examined diet or dietary components and broad mental health outcomes within childhood and adolescence and reported consistent cross-sectional associations between unhealthy dietary patterns and poorer mental health, and healthy dietary patterns or high diet quality with better mental health (97, 98). However, when applying best evidence synthesis and looking at the studies with a quality rating above the mean, evidence was deemed limited (97).

This current review aimed to identify longitudinal studies involving children and adolescents, that examined overall, or multiple aspects of diet, and association with mood disorder outcomes. Studies were identified via repeat searches of medical and psychiatry journal databases (e.g., PubMed, PsycINFO) throughout the PhD candidature period and from reference lists of review and original studies. Studies were excluded if they focused on intake of one food type or nutrient, eating disorders, specific diets due to medical conditions (e.g., coeliac disease), food insecurity, or were among very young children (under 10 years of age at both baseline and follow-up with dietary intake reported by a parent/caregiver). After these exclusions, to date there are only 10 observational prospective studies examining associations between multiple aspects of diet or overall diet and mood disorder outcomes among children and adolescents in the general population (99-108). An overview of these studies is provided in Table 2.1. There are several additional longitudinal studies among the general population that reported on associations between dietary factors in youth and mood disorder outcomes. However, they are not included in this review as they either analysed specific dietary

components (e.g. fruit and vegetable intake) rather than deriving an overall measure of diet quality (109, 110), or were entirely retrospective with no measures taken in youth (111).

Table 2.2 Longitudinal studies on diet quality and mood disorder outcomes among children and adolescents (ordered by country and year)

Author, Date	Country and Cohort Follow-up years	Participants (sex %) Baseline age	Dietary assessment	Mood disorder measure	Longitudinal analysis results[a]
Jacka et al., 2011 (101)	Australia, It's Your Move (IYM). 2 years	2,996 (54% male). 11-18 years.	Healthy diet score calculated from 7 questions on food habits (final score range 1-8). Unhealthy diet score calculated from 7 questions on food habits (total score range 9-53).	Emotional functioning subscale of the Pediatric Quality of Life Inventory (PedsQL) comprising 5 questions with a 5-point Likert type response: "I feel afraid or scared"; "I feel sad"; "I feel angry"; "I have trouble sleeping"; "I worry about what will happen to me". Higher score (range 0-100) indicates higher quality of life.	Compared to those with the lowest Healthy diet scores at baseline, those with middle and high scores had higher PedsQL scores at follow-up after covariate adjustment: middle: β= 0.11, 95% CI: 0.01, 0.21); high: β= 0.14, 95% CI: 0.02, 0.27).
Trapp et al., 2016 (103)	Australia, Western Australian Pregnancy Cohort (Raine) Study. 3 years.	746 (49.2% male). 14 years.	212 item semi-quantitative FFQ. Two dietary patterns identified: Healthy: whole grains, fruit, vegetables, legumes, fish. Western: take-away foods, meat, full-fat dairy, fried potatoes, refined cereals, cakes, biscuits, confectionary, soft drinks, crisps, sauces, and dressings.	118-item Youth Self-Report (YSR), an adolescent self-report version of the Child Behaviour Checklist for Ages 4–18 years. The internalising problem score describes depressive and anxiety symptoms and withdrawn behaviour.	There were no statistically significant associations between Western or Healthy dietary pattern z-scores at 14 years and internalising behaviours at 17 years after adjustment.
Oddy et al., 2018 (105)	Australia, Raine Study. 3 years.	843 (49.1% male). 14 years.	As above.	YSR internalising disorders as above. Also, Beck Depression Inventory for Youth (BDI-Y) - 20 items relating to feelings experienced over the previous two weeks. Higher scores on the BDI-Y (range 0-60), reflects the presence of more depressive symptoms.	Higher pattern scores at 14 years of age was associated positively (Western pattern) and inversely (Healthy pattern) with BMI and inflammation markers at 17 years. Structural equation models demonstrate plausible pathways linking dietary patterns to depressive symptoms via BMI and inflammation.

Author, Date	Country and Cohort Follow-up years	Participants (sex %) Baseline age	Dietary assessment	Mood disorder measure	Longitudinal analysis results[a]
McMartin et al., 2012 (100)	Canada, Children's Lifestyle and School Performance study (CLASS). 3 years.	3,757 (48% male). 10-11 years.	Harvard Youth/Adolescent Food Frequency Questionnaire (YAQ), modified to include Canadian food and product names[b]. Diet Quality Index–International comprising four components: (i) variety; (ii) adequacy; (iii) moderation; and (iv) balance. Higher scores on range 0-100, indicates higher diet quality.	ICD-9 or ICD-10 diagnosis of an internalising disorder (depressive episode, recurrent or persistent mood disorder, neurotic or general anxiety disorder, acute reaction to severe stress, or emotional disorder with childhood onset), identified from medical insurance and hospital admission databases.	Overall diet quality was not associated with internalising disorder. For diet variety, relative to children in the lowest tertile, children in the highest tertile had around half as many health-care contacts with a diagnosis of internalising disorder (incidence rate ratio: 0.45; 95% CI: 0.25, 0.82) after model adjustment.
Wu et al., 2017 (104)	Canada, CLASS. 7 years.	4,875 (49.2% male). 10-11 years.	As above.	As above.	Overall diet quality was not associated with internalising disorder. Relative to participants in the lowest score third for diet variety, those in the middle third had fewer diagnoses of internalising disorder (incidence rate ratio: 0.77; 95% CI 0.60, 0.99) after model adjustment.
Esteban-Gonzalo et al., 2019 (107)	Spain, UP&DOWN study. 2 years.	1,197 (51.5% male). 6-16, years.	The Mediterranean Diet Quality Index for children and adolescents (KIDMED): 16 yes-or-no question to determine adherence to the Mediterranean dietary (MD) pattern. Four questions were scored –1, and 12 questions were scored 1. Higher score (range –4 to 12) indicates higher adherence to a MD.	The Positive and Negative Affect Schedule (PANAS) for children and adolescents. The negative affect component comprises 10 items with 5-point Likert-type responses. The score range is 10-30, with higher scores indicating higher negative affect.	There was no prospective relationship between adherence to a MD and negative affect scores 2 years later. At the 2-year follow-up, MD adherence was cross-sectionally associated with lower negative affect in adolescent girls and boys (β = –0·15, SE 0.07, p = 0.047; β = –0·16, SE 0.06, p = 0.019, respectively).

Author, Date	Country and Cohort Follow-up years	Participants (sex %) Baseline age	Dietary assessment	Mood disorder measure	Longitudinal analysis results[a]
Wu et al., 2016 (102)	Taiwan, Child and Adolescent Behaviors in Long-Term Evolution (CABLE) study. 10 years (follow-ups every year).	2,630 participated in at least 2 waves of the study (50.0% male). 8 years.	Unhealthy eating behaviours score calculated from 3 questions on how often during the past week the participant consumed take-away foods, packaged snacks/ confectionary, sweetened beverages. Fruit and vegetable intake (> 3 days per week), was included as a covariate.	Scale developed from Kovacs' Children's Depression Inventory and the Center for Epidemiological Studies Depression Scale for Children. Scale comprises 7 items about feelings and behaviour during previous 2 weeks. Higher score (range: 7-21) indicates greater number of depressive symptoms.	The frequency of unhealthy eating behaviours in the previous year and the increases in frequency between the previous and successive year were positively associated with the initiation and growth rate of depressive symptoms, and vice-versa.
Jacka et al., 2013 (99)	United Kingdom, Research with East London Adolescents: Community Health Survey (RELACHS) 2 years.	2,093 (% male not reported for longitudinal analysis - baseline sample of 2,789 was 48.8% male). 11-14 years.	Healthy diet score calculated from 3 questions (breakfast eating, daily serves of fruit, daily serves of vegetables). Unhealthy score calculated on 5 questions: frequency of eating savoury snacks, sweets, biscuits, fried foods, and soft drinks.	Short Moods and Feelings Questionnaire (SMFQ) – 13 items about emotions and behaviours during past 2 weeks. Participants with a score of 8 or above (score range 0-26) were categorised as experiencing depressive symptoms.	Weak inverse relationships between Healthy diet scores by quintiles and depressive symptoms at follow-up [Q1 = reference (Q2: OR=0.82, 95% CI: 0.60, 1.11; Q3: OR=0.65, 95% CI: 0.46, 0.92; Q4: OR=0.70, 95% CI: 0.52, 0.94; Q5: OR=0.75, 95% CI: 0.55, 1.04)], were attenuated after covariate adjustment.
Winpenny et al., 2018 (106)	United Kingdom, ROOTS. 3 years.	603 (40.0% male). 14 years.	Four-day diet diary. Mediterranean diet score (MDS) calculated based on nine items (vegetables, legumes, fruit, nuts, whole grains, red and processed meat, fish, ratio of mono-unsaturated to saturated fat, ethanol). Score range not reported.	Moods and Feelings Questionnaire (MFQ), a 33-item self-report measure of depressive symptoms. For each item, participants reported their mood over the previous 2 weeks on a 3-point scale (mostly/sometimes/never), giving an overall score ranging from 0 to 66.	There were no prospective associations between MDS and depressive symptoms.

Author, Date	Country and Cohort Follow-up years	Participants (sex %) Baseline age	Dietary assessment	Mood disorder measure	Longitudinal analysis results[a]
Cong et al., 2020 (108)	United Kingdom, Avon Longitudinal Study of Parents and Children (ALSPAC). 9 years.	6,939 (50.3 % male). 9 years.	Parent reported dietary intake On a 90 item FFQ. Reduced rank regression and child biomarkers of CRP and IL-6 were used to determine an Inflammatory Dietary Pattern (IDP) score.	Computerised version of the Clinical Interview Schedule-Revised (CIS-R). The CIS-R incorporates core symptoms of depression from ICD-10 to produce a composite depression score (range: 0 to 21). A CIS-R score of 9 or higher was used in combination with the ICD-10 diagnosis to identify probable cases of depression.	Compared to lowest third of baseline IDP scores, the highest third had 1.34 times higher odds (95% CI: 1.08, 1.66) of developing depression over follow-up. The association was attenuated by covariate adjustment to 1.2 times higher odds (95% CI: 0.96, 1.51).

β: the difference in the outcome variable calculated from linear regression of the outcome on the predictor; OR: odds ratio; 95% CI: 95% confidence interval

[a] Only the effect sizes and 95% confidence intervals for statistically significant longitudinal associations have been reported. Cross-sectional analysis results and null effects are not reported.

[b] The Harvard Youth/Adolescent Food Frequency Questionnaire includes 151 items, however, the number of food and beverage items in the modified FFQ was not specified.

2.5.2 Study Design

All the studies outlined in Table 2.1 were derived from cohort studies designed to determine the influence of various factors on a range of health, academic, or social outcomes. That is, none of the studies were designed with the primary aim of examining the effects of diet on mental health or mental disorder outcomes. Some of these studies relate to the same cohorts: two studies were based on the Western Australian Pregnancy Cohort (Raine) (103, 105) and two were based on the Canadian Children's Lifestyle and School Performance (CLASS) cohort (100, 104). The Avon Longitudinal Study of Parents and Children (ALSPAC) and Raine studies involved participants who were the offspring of pregnant mothers, with ALSPAC recruiting additional children at seven years of age (103, 108). The remaining studies recruited child or adolescent participants through schools (99-102, 104, 106-108). All studies were based on observational study cohorts except for the 2011 Australian It's Your Move (IYM) study which was originally designed as a quasi-experimental obesity prevention program comprising a group that received a healthy lifestyle intervention and a comparison group (101). The combined population analysis was justified by no effect modification of the outcome variables by group type (intervention group, comparison group) (101).

The follow-up periods ranged between two and 10 years, and none of the 10 studies extended follow-up beyond late adolescence. The 2011 IYM study reported that students up to 18 years of age participated in the baseline cross-sectional study, but did not report the ages of participants at the follow-up two years later when it is noted that questionnaires were completed during school class time (101).

2.5.3 Dietary assessment

The brevity and heterogeneity of dietary assessment is a limitation of existing studies on children and adolescents. Three of the studies calculated healthy or unhealthy diet scores based on a small number of questions (between three and seven questions) about aspects of usual dietary intake (99, 101, 102). A 2019 Spanish study used 16 yes/no questions to determine a Mediterranean diet score (107). Several studies used more comprehensive measures of usual diet. The Raine, CLASS, and ALSPAC studies used food frequency questionnaires incorporating

between 90 and 212 food and beverage items (100, 103-105, 108). Diet quality for these studies was assessed by three different methods. The Raine studies used principal component analysis to empirically determine two major dietary patterns (Healthy and Western) (103, 105), the CLASS studies applied the Diet Quality Index – International (100, 104), and the ALSPAC study used reduced rank regression on inflammatory biomarkers to determine an Inflammatory Dietary Pattern score (108). The ROOTS study in the United Kingdom was the only study to collect dietary data with a food diary (106). Data from the four day diet diary that incorporated two weekend days, was used to calculate a Mediterranean diet index score (106).

2.5.4 Mood disorder measures

The weakest measure of depressive symptoms was the PedsQL emotional functioning subscale that was used in the 2011 Australian IYM study (101). The study rationalised the use of the subscale as it is a validated measure of depressive symptoms, but it only comprises the following five questions that also could be interpreted as symptoms of anxiety or externalising disorders: “I feel afraid or scared”; “I feel sad”; “I feel angry”; “I have trouble sleeping”; “I worry about what will happen to me” (101).

Six of the studies used more extensive self-report instruments capturing varied concepts around depressive symptoms conceived as negative effect, moods and feelings, internalising problems, or depression scales (99, 102, 103, 105-107). Although these were validated instruments, or adapted from validated instruments, they varied widely in their structure, ranging from seven items for the 2016 Taiwan study scale developed from the Kovacs’ Children’s Depression Inventory and the Center for Epidemiological Studies Depression Scale for Children (102), to the 118 item Youth Self-Report questionnaire used in the Raine studies, which is a self-report version of the Child Behavior Checklist for ages 4–18 years (103, 105). The score ranges or methods used to categorise participants as experiencing depressive symptoms also varied.

Only two studies from the Canadian CLASS cohort (100, 104) and the one from the United Kingdom ALSPAC cohort (108) used clinically defined mood disorder as the outcome measure. The CLASS study linked participant data to clinical diagnosis of

internalising disorders (any ICD-9 or ICD-10 diagnosis of a mood or depressive disorder, neurotic or general anxiety disorder, and severe stress reaction or emotional disorder) in administrative health care databases (100). The ALSPAC study used the Clinical Interview Schedule-Revised structured diagnostic interview to identify ICD-10 defined depression disorder at age 18, but also used a cut-off from the continuous composite depression score to identify probable cases (108).

2.5.5 Evidence of associations between diet and mood disorder outcomes

Five studies from the CLASS, Raine, ROOTS, and Up&Down cohorts reported no longitudinal associations between baseline diet quality or dietary patterns and follow-up depression measures in either the unadjusted or covariate adjusted models (100, 103, 104, 106, 107). Although the two CLASS studies did not find any association between overall diet quality and internalising disorders, they did report significant associations in the fully adjusted models between higher scores on the dietary variety component of the Diet Quality Index – International and lower diagnosis of internalising disorder during the four-year (100) and eight-year follow-ups (104). A limitation of the CLASS studies is that they were not able to capture outcomes for participants who did not seek clinical help and did not account for baseline internalising disorders or mental health (100). Adjusting for baseline negative affect is important as depression in adolescence has been associated with greater risk of depression in adulthood (112).

The two studies from the UK RELACHS and ALSPAC cohorts reported longitudinal associations that were attenuated after covariate adjustment (99, 108). The 2013 RELACHS study reported that a weak relationship between higher Healthy diet scores and lower odds of depressive symptoms at follow-up among East London adolescents was fully attenuated after adjustment for gender, dieting, parental conflict and family social support (99). The ALSPAC study reported that those with the highest (most inflammatory) compared to lowest (least inflammatory) third of inflammatory dietary pattern scores had 1.3 times higher odds of depressive disorder at follow-up, but this was attenuated to 1.2 times higher odds and not statistically significant after adjustment for sex, race, parental education, parental social class (professional, maternal age at delivery, birthweight, maternal smoking

status, maternal anxiety, maternal depression, physical activity level, and weight status (108).

Only two of the studies reported associations that were robust to covariate adjustment. The first was the 2011 IYM study, which reported that baseline healthy diet scores (based on seven questions) were linearly associated with higher PedsQL scores (higher quality of life) at follow-up after adjustment for gender, age, dieting behaviours, BMI, socioeconomic status and physical activity and baselined PedsQL score. However, the brief dietary measures and general mental health outcome means that the study provides little evidence of a diet-mood disorder relationship (101). The second study was a Taiwanese study with annual follow-ups for 10 years between the ages of eight and 17 (102). This study had brief dietary measures (three questions on unhealthy dietary practices) and reported that frequency of unhealthy eating in the previous year and increased frequency between the previous and current year, was positively associated in with depressive symptoms in the current year and vice versa, indicating bi-directionality of the relationship (102). The analysis was adjusted for time-fixed variables (sex, parental education, monthly household income) and time-varying variables (parents' marital status, family activity score, body weight, consumption of fruit or vegetables, exercise participation, smoking) (102).

The remaining 2018 study on the Raine cohort did not examine the direct relationship between dietary patterns and depressive symptoms (shown to be no direct association in the 2016 study (103)), and instead used structural equation models to demonstrate associations between a Western dietary pattern at 14 years of age and biomarkers of inflammation linked to depressive symptoms at 17 years of age (105). The aim of this study was to demonstrate the plausible pathways linking diet to depressive symptoms via BMI and inflammation (105). This study and the results from the 2020 ALSPAC study (108) outlined above, highlight how poor diet and inflammation in youth could have long term effects that influence mood disorder outcomes.

2.5.6 Summary

This review indicates that there is a need for further prospective studies of youth diet and mood disorder outcomes, with follow-ups that extend beyond adolescence and involve quality diet, mood and covariate measures.

2.6 Prospective studies on overall diet quality and mood disorders among adults

There have been more studies on overall diet and associations with mood disorders among adults than among children and adolescents, although many were cross-sectional analyses. Due to the large number of studies, what follows is an outline of systematic reviews, focusing on review of overall diet and highlighting results around prospective studies.

2.6.1 Systematic reviews of studies examining effects of overall diet

Systematic reviews of observational studies among adults have found limited to moderate evidence of relationships between overall diet quality or dietary patterns, and mental health (72, 113-119). A 2013 systematic review of 25 studies (including five cohort studies) on community-dwelling adults applied best evidence synthesis and reported only limited evidence for associations between dietary patterns and depression (113). The review highlight differences in measurement tools for diet and mental health, variation in study population characteristics, and inconsistency in adjustment for covariates between studies as contributing factors to the inconsistent results and weakening of evidence (113). A systematic review from the same year, focused only on cohort studies, but only two of the 11 included studies examined overall diet, while the others focused on specific nutrients such as omega-3 or folate, or intake of individual foods (fish, olive oil, seed oil) (116). The narrative review concluded that diet may influence risk of depression but due to possible selection, measurement and publication bias, further robust prospective research is required (116). A 2014 systematic review of dietary patterns and depression risk was also reluctant to perform a meta-analysis due to heterogeneity of the 16 studies (nine cross-sectional, seven cohort studies), but the descriptive review concluded that healthier dietary patterns may be associated with reduced odds of depressive symptoms, while unhealthy patterns may be associated with higher odds of

symptoms (72). Another 2014 review, also on studies that examined overall diet, performed a meta-analysis of pooled results from 13 studies (including four cohort studies) and found that a healthy dietary pattern was inversely associated with depressive symptoms (OR=0.84; 95% CI: 0.76, 0.92) (114).

There have been stronger results reported in recent years from meta-analyses. A 2017 meta-analysis of 21 studies (11 cohort, six cross-sectional, and four case-control studies) also found that a higher adherence to a healthy dietary pattern was associated with lower odds of depressive symptoms (OR=0.64; 95% CI: 0.57, 0.72), while higher adherence to a Western/unhealthy pattern was associated with higher odds of depressive symptoms (OR=1.18; 95% CI: 1.05, 1.34) (119). A 2018 meta-analysis by Lassale et al. of healthy dietary indices and depression, examined pooled results of five longitudinal studies that focused on the effects of inflammation using the Dietary Inflammatory Index, and reported that the lowest compared to the highest adherence to a pro-inflammatory diet was associated with lower risk of depressive symptoms (RR=0.76; 95% CI: 0.63, 0.92) (118). This finding was similar to another review published the same year that used four of the same studies, but used lowest adherence as the reference category (117). This review reported that the highest compared to lowest adherence to an inflammatory diet was associated with increased risk of depressive symptoms (RR=1.25; 95% CI: 1.12, 1.40) (117). A further 2019 meta-analysis reviewed the same five longitudinal studies as Lassale et al., but with the addition of two cohort studies where inflammatory potential of diet was measured by blood cytokine biomarkers, and lowest adherence to an inflammatory diet was used as the reference category (120). This review also reported that highest compared to lowest adherence was associated with increased odds of depressive symptoms (OR=1.31; 95% CI: 1.20, 1.44) (120). All three reviews on the inflammatory effects of diet highlighted the heterogeneity of the studies as a limitation, particularly in regards to the measurement and definition of depressive symptoms.

A further 2018 meta-analysis (of 24 cohort studies) found that the highest category of adherence to a high quality dietary pattern (e.g. Mediterranean, healthy, prudent), was associated with lower risk of depressive symptoms (OR=0.77; 95% CI:

0.69, 0.84) and there was a dose response association (115). However, it was noted that diet was not associated with depression risk in the studies that had clinical diagnosis of depression as the outcome or that had controlled for depression severity at baseline (115). The authors were also cautionary and quantified that even including the studies with non-clinical depression measures, to prevent one case of depression, 47 people needed to make considerable dietary improvements to change from the lowest to highest category of dietary pattern adherence (115). Interpretation of findings and translation into real-world or clinical effects is rare but is beneficial to the nutritional psychiatry narrative.

To evaluate the growing interest and body of literature on diet and mood disorders, similar to what has been done in this current review but extended to other lifestyle factors, a 2020 meta-review summarised the evidence from meta-analyses on associations between depressive symptoms and diet, physical activity, smoking, and sleep (121). The review included 10 meta-analyses focused on diet, of which five examined associations of overall dietary measures and five examined associations of singular food types or nutrients. The meta-review concluded that although there was some evidence of prospective inverse associations between healthy dietary patterns and depressive symptoms, prospective associations with diagnosed depressive disorders were less clear (121).

2.6.2 Evidence related to the Mediterranean diet

In a 2018 meta-analysis, pooled results of five longitudinal studies from four cohorts showed that the highest compared to lowest adherence to the Mediterranean diet was associated with lower risk of depressive symptoms (RR=0.67; 95% CI: 0.55, 0.82) (118). A 2019 narrative review of studies that included analysis on the Mediterranean diet or Mediterranean type diets, indicated that 85% of the 20 observational studies reviewed (including 13 cohort studies), reported that higher adherence to the Mediterranean diet was associated with lower risk of depressive symptoms (122). Another 2019 review that conducted a meta-analysis of seven cohort studies reported no significant association between adherence to a Mediterranean diet and risk of depressive symptoms, but there was an association for the nine cross-sectional studies between higher adherence and lower odds of

symptoms (OR=0.72; 95% CI: 0.60, 0.87). A 2020 meta-analysis of three cohort studies reported that a one standard deviation higher baseline Mediterranean diet score was associated with lower odds of incidence of high depressive symptoms (OR=0.88; 95% CI: 0.81, 0.94) (123). Although this meta-analysis had some strengths such as excluding participants with high depression symptom scores at baseline (cut-offs varied between the studies), the selection criteria for the included studies appeared to be based on data availability, rather than a systematic review (123).

2.6.3 Studies with diagnostic mood disorder measures

The systematic reviews highlighted variation in the measurement and criteria used to determine depressive symptoms as a common limitation, with most studies using questionnaires and depression scales rather than clinical diagnostic interview and assessment or self-report of clinical diagnoses. Of the individual studies included in the reviews outlined above that did use diagnostic criteria, seven focused on the effects of select foods or dietary nutrients rather than overall diet (124-130), and four studies used measures of overall diet but were cross-sectional studies (131-134). The longitudinal studies that did use diagnostic measures all used self-report of physician diagnoses and anti-depressant use. These included four prospective studies arising from the Seguimiento Universidad de Navarra (SUN) Spanish cohort of university graduates (135-138). The self-report measure was found to be an adequate measure of mood disorder through validation of a sub-sample of the cohort against diagnosis by a psychiatrist or clinical psychologist using the Structured Clinical Interview for DSM-IV (139). The SUN studies had follow-up periods of between four and eight years, and reported that higher scores on Mediterranean dietary patterns and healthy eating indices were associated with lower risk of depressive disorders, whereas a pro-inflammatory dietary pattern was associated with higher risk of depressive disorders (135-138). In contrast, a study using data from the United States Nurses' Health Study that identified depression cases from self-reports of clinical diagnosis, reported no independent association between dietary patterns and risk of depressive disorder during the 12-year follow-up (140).

2.6.4 Other associations between dietary intake and mood disorders

Examining only one facet of diet such as fruit and vegetable intake allows ease of measurement and synthesis of data across different cohorts and potential for inclusion of large numbers of participants. However, although a dietary aspect such as fruit and vegetable intake may be representative of certain health behaviours, it does not describe or account for possible confounding effects of other foods that may be commonly consumed. A 2016 meta-analysis of cohort studies with measures of fruit (four studies) and vegetable (four studies) intake found that participants with the highest consumption of fruit or vegetables had reduced risk of depression compared to those with the lowest intake (fruit: RR=0.83; 95% CI: 0.77,0.91; vegetables: RR=0.88; 95% CI: 0.79, 0.96) (141). A further 2018 meta-analysis reported lower risk of depressive symptoms associated with the highest compared to lowest intake of fruit (five cohort studies, overall RR=0.83; 95% CI: 0.71, 0.98), and the highest compared to lowest intake of vegetables (seven cohort studies, overall RR=0.86; 95% CI: 0.75, 0.98) (142). A 2019 narrative review with a narrower literature search that included five cohort studies with depressive symptoms as the outcome measure, found little evidence of prospective associations between fruit and vegetable intake and depressive symptoms, and noted that regular consumption may be linked to other positive health behaviours that also act to reduce risk of depression (143).

Although this thesis focuses on observational studies as a means of examining associations among community-living populations and identifying how diets may have preventative effects on mood disorders, other supporting evidence of the diet-mood disorder relationship comes from case-control and intervention studies. A 2015 review of randomised control trials involving dietary intervention determined there was moderate evidence of a relationship between diet and depression, with nearly half of the 17 studies reviewed observing reduced risk of depressive symptoms in the treatment groups (144). Around 75% of the trials that reported significant improvements had recommended a high fibre diet or increase in fruit and vegetables, while the dietary interventions that involved eating more fish, reducing red meat, increasing lean meat, reducing cholesterol, or were focused on weight loss were more likely to result in no difference between the treatment and control

groups (144). A 2019 meta-analysis of randomised control trials on dietary interventions highlighted heterogeneity of the 16 included studies and that only one study used diagnosis of clinical depression as the outcome measure, but reported a small positive effect of interventions to improve diet on reduced depressive symptoms (Hedge's *g* = 0.275; 95% CI: 0.10, 0.45) (145). The 2019 narrative review of Mediterranean dietary patterns and depression risk, detailed in Section 2.6.2, included results of six randomised control trials, and reported that all six trials showed that intervention groups prescribed Mediterranean style diets had fewer depressive symptoms during the trial period than the control groups (122).

2.6.5 Summary

It is apparent from the existing literature that further research efforts in the field should be focused on longitudinal studies with high-quality outcome measures, to determine whether diet could have a protective or preventative role in the development of mood disorders.

2.7 Time-of-day eating and mood disorders

There is evidence that timing of daily food intake, sometimes referred to as chrononutrition, and habitual practices such as skipping meals or frequency of eating occasions can affect nutritional intake and diet quality (146). However, timing of eating occasions may also affect biological processes, including neurobiology (20, 147), and the microbiome (148). The timing of eating occasions and the duration between meals is thought to influence circadian rhythms and gene regulation, affecting metabolism and markers of cardiometabolic health (148, 149). Some of the understanding on the impact of meal timing comes from research on animal models, which has indicated neurobiological effects such as enhanced levels of BDNF associated with intermittent fasting, and increased serotonin signalling in response to caloric restriction (which intermittent fasting is thought to mimic) (20). The time of eating during the day or night may also affect hormone levels such as leptin which suppresses hunger, ghrelin which stimulates hunger, melatonin which promotes sleep, and cortisol which is involved in stress response (150).

2.7.1 Prospective observational studies

Much of the existing research on time-of-day eating focuses on discrete practices such as meal skipping, snacking, meal frequency, or intermittent fasting in relation to physical health outcomes such as obesity and cardiometabolic disease. There is a considerable body of research on the effects of skipping breakfast on obesity and cardiovascular health (151) and child and adolescent cognitive function and academic performance (152), but studies examining mood disorder outcomes or other dietary practices are limited. Longitudinal studies on skipping breakfast and mood disorder outcomes are particularly limited, although results indicate that further research may be warranted. A Japanese study among an adult occupational cohort reported that compared to participants who ate breakfast every day, regularly skipping breakfast was associated with higher risk of depressive symptoms during the three year follow-up period, and this risk increased as the frequency of skipping breakfast increased (153). A study among young adult Chinese college students, found a higher weekly frequency of skipping breakfast was associated with increasing risk of depressive symptoms during a one year follow-up period (154).

There have also been limited longitudinal studies on other aspects of meal timing such as meal frequency and mood disorder outcomes. A 2019 study among Bangladeshi university students examined daily meal frequency in relation to depression and anxiety outcomes at 15 month follow-up (155). The study reported associations between both high (five or more) and low (no more than two) daily meals and higher risk of anxiety but not depression, compared to students who consumed 3-4 meals per day (155).

The only prospective study that has analysed more than one aspect of the timing of daily food intake, was a 2016 Japanese study that examined associations between skipping breakfast, eating dinner shortly before bedtime, and snacking after dinner on mood disorder outcomes at a two year follow-up among an adult cohort (aged 24-83 years) (156). It was reported that snacking after dinner, or having two or more of these dietary practices, was associated with higher risk of depressive symptoms at follow-up (156).

2.7.2 Time-of-day eating occasions measures and methods

Variations in the definition of eating occasions such as “meals” and “snacks” and whether to include beverage intake affects the interpretation and generalisation of results from different studies (146, 149). This variation can be due to participant perception, or the definitions used between studies. For example, the definition of breakfast may be a meal of a certain size eaten at a particular time e.g. between 6:00am and 9:00am, or refer to the first meal or any food or caloric beverage consumed after waking.

An approach that may be of use in understanding chrononutrition is empirical pattern analysis of eating occasions, similar to how methods such as principal component analysis and exploratory factor analysis have been used to determine dietary patterns of foods and food types commonly eaten together among community-living populations. Empirical analysis could help derive common patterns of time-of-day eating that people may habitually adhere to, allowing exploration of associated health outcomes.

2.7.3 Summary

Studies on time-of-day eating practices tend to focus on discrete practices such as skipping breakfast, rather than examine multiple dimensions of timing of daily food intake. There is also a gap in the literature on prospective studies examining the relationship between timing of daily food intake and mood disorder outcomes.

2.8 Review summary and implications

It is apparent in the Section 2.2 overview of nutrients associated with mental health, that the dietary sources of beneficial nutrients are primarily unprocessed foods, from a variety of food groups. The physical growth and development period from childhood to adolescence to adulthood is when there is an increase in the recommended dietary intake of many nutrients (157). As nutrients have multiple roles in biological functions and a wide range of nutrients are required for effective neurological functioning, it follows that diets rich in a variety of whole foods, many of which are good sources of multiple nutrients, would be associated with better mental health. This supports the importance of measuring overall diet via methods

outlined in Section 2.3.2 such as a diet quality index, to examine the relationship with mood disorder outcomes.

The review of existing studies examining the overall dietary patterns and diet quality of community-living cohorts and mental health outcomes (Section 2.5 and Section 2.6) indicates that further evidence is required to determine if there is an independent effect of dietary intake. Ideally this would be from high-quality longitudinal studies to establish the nature of the relationship between diet and mood disorders, or the extent that it is confounded or mediated by other lifestyle, economic, social, or physiological factors. This literature review has highlighted that a recurring limitation is the non-clinical measures of depression. This particularly gap in the literature would be partly due to the greater cost and resources for the researchers and time and onus on the participants of using structured interviews to identify disorders, or concerns around only identifying participants who have sought clinical help by using self-report of clinical diagnoses or anti-depressant use.

The CDAH study is a valuable resource in addressing some of these limitations and enabling high quality original studies. The following chapter outlines the measures and methods involved in the CDAH study. In summary, the measures include validated dietary measures in youth and adulthood, a “gold standard” instrument for determining clinical diagnoses of mental disorders in observational studies, and a wide range of repeat socioeconomic, demographic, lifestyle, wellbeing, and physical measurements. This thesis therefore aims to use the available data to build on the existing literature and contribute to understanding directionality of the diet-mood disorder relationship via longitudinal analyses.

Chapter 3: Methods

3.1 Preface

The aim of this thesis was to explore associations between diet and mood disorders using data from the Childhood Determinants of Adult Health (CDAH) cohort study. The CDAH study followed up participants of the 1985 Australian Schools Health and Fitness Survey (ASHFS) and concurrent National Dietary Survey of Schoolchildren (NDSS). The CDAH study has so far involved three follow-ups during adulthood: CDAH-1 during 2004-06, CDAH-2 during 2009-11, and CDAH-3 during 2014-19. The primary aim of CDAH is to determine how childhood factors influence cardiovascular and cardiometabolic disease risk in later life. A wide range of data has been collected including demographic, lifestyle (including dietary), physical, metabolic, and mental health measures to enable tracking of the health and lifestyle of the cohort through adulthood. Data from the 1985 baseline and three follow-ups in adulthood are used in the four original studies that comprise this thesis (Figure 3.1). This chapter describes the baseline ASHFS and NDSS studies, and the three CDAH follow-ups, to provide an overview of participants and the methods used to collect data. Methods and measures specific to each of the four original studies of this thesis are outlined in their respective chapter (Chapters 4-7).

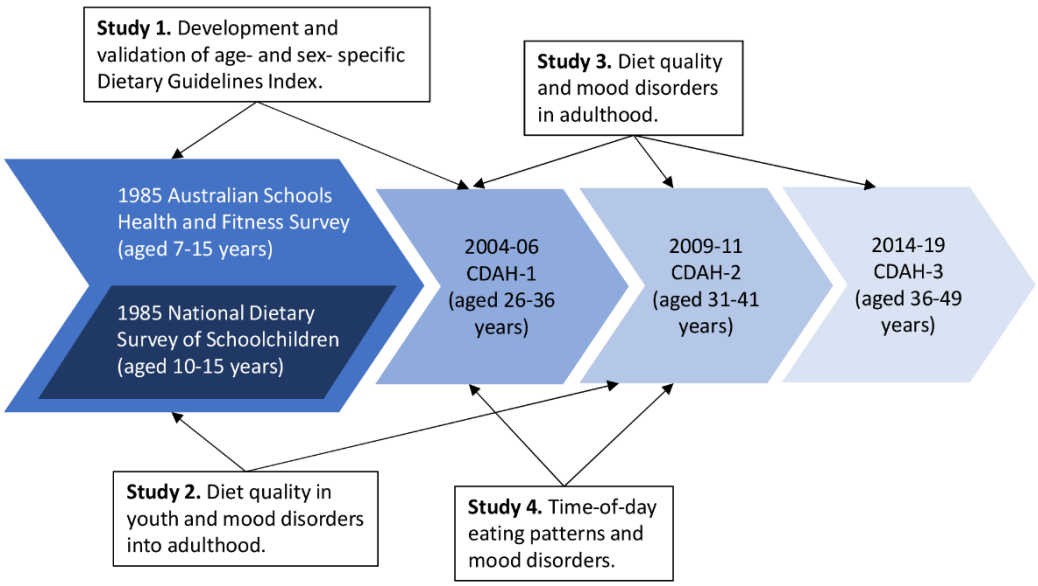


Figure 3.1 Childhood Determinants of Adult Health data used in Chapters 4-7

3.2 Australian Schools Health and Fitness Survey (ASHFS)

3.2.1 Study rationale

The ASHFS study was designed and conducted by the Australian Council for Health, Physical Education and Recreation Inc. (ACHPER) to provide a benchmark of the physical health and fitness of Australian primary and secondary schoolchildren with which to compare later surveys to. Other objectives of the study were to establish percentile norms for fitness and performance tests of youth aged 7-15 years, raise awareness of health and fitness through the publication of survey results, and establish a National Fitness Award Scheme to encourage participation in regular physical activity (158, 159).

The ASHFS was an extension of the 1971 Australian Youth Fitness Survey, with additional health measures and inclusion of primary schoolchildren. The study comprised field tests for all age levels 7-15 years (including additional technical tests for ages 9, 12 and 15 years), a physical activity and lifestyles questionnaire for participants aged 9-15 years, a 24-hour food record for those aged 10-15 years, and blood samples for those aged 9, 12, and 15 years. Younger children were not asked to complete the questionnaire due to concerns they may be too young to complete it reliably. The blood sampling was restricted to participants aged 9, 12, and 15 years

due to time and resource considerations and because these test results were not planned to be used to establish percentile norms.

3.2.2 Design and participants

The ASHFS was designed, coordinated, and administered by ACHPER with assistance from the National Heart Foundation of Australia, the Australian Government Commonwealth Department of Sport, Recreation and Tourism, Commonwealth Schools Commission, and the Commonwealth Department of Health.

The sample size was determined to allow establishment of fitness norms, with the estimates of the means for the fitness field and technical tests to be within 5% of the true mean. Sample size calculations indicated that a standard sample of 500 participants of each age level and gender would allow estimation of the mean within 5% of the true mean for most tests, with the exception of technical tests (skinfold thickness and blood lipids) which would be within 7% of the true mean, and the questionnaire, which would be within 10% of the true mean.

Students aged 7 to 15 years were selected as the target population due to differing enrolment requirements by state that affected numbers of children enrolled at school outside this age range. The sampling frame included state, Catholic and independent schools across all Australian states and territories with at least 10 students in each age and sex category. These minimum size of enrolment requirements resulted in exclusion of 9.9% of primary students (excluded schools were mainly small primary schools in regional areas) and 3.1% of secondary students.

A two-stage probability sample design was used to first select primary and secondary schools based on postcode distribution to ensure geographical distribution, and the probability proportional to enrolment numbers of primary school students aged 10 years, and secondary school students aged 14 years. The second stage, independent from the first sample of schools, was to randomly select 15 girls and 15 boys at each age level from the school enrolment. Five additional students of each age/sex category were selected to allow for factors such as absence from school at the time of the survey, or refusal to participate.

In total, 121 schools were invited to participate, with 109 schools agreeing, giving a school response rate of 90.1%. A total of 12,578 students were approached, with 8,498 participating in at least one component of the study, giving an overall response rate of 67.6%. The response rate was highest among the younger age groups, and lowest among girls aged 13-15 years. The numbers of participants of each age and sex category differed according to the study component and are outlined in Table 3.1.

Table 3.1 Number of Australian Schools Health and Fitness Survey participants for each survey component, by age and sex

Age (years)	Field and Technical[a]		Questionnaire[b]		Blood Sample[c]	
	N	%	*N*	%	*N*	%
Females						
7	478	11.4				
8	496	11.8				
9	488	11.6	481	15.2	339	38.3
10	497	11.9	489	15.5		
11	484	11.5	475	15.0		
12	489	11.7	479	15.2	301	34.0
13	438	10.5	426	13.5		
14	408	9.7	400	12.7		
15	413	9.9	407	12.9	246	27.8
Sub total	4191	100.0	3157	100.0	886	100.0
Males						
7	475	11.0				
8	490	11.4				
9	482	11.2	476	14.6	359	36.4
10	493	11.4	478	14.7		
11	489	11.4	473	14.5		
12	494	11.5	473	14.5	341	34.6
13	466	10.8	454	14.0		
14	467	10.8	452	13.9		
15	451	10.5	448	13.8	285	28.9
Sub total	4307	100.0	3254	100.0	985	100.0
Total	8498		6411		1871	

[a] Field tests included fitness tests such as push-ups and 50 metre run, and height and weight measurements. Technical tests (e.g., skinfolds, muscular strength, blood pressure) required specialised equipment and were time consuming so were only administered to students aged 9, 12 and 15 years who participated in the field tests.
[b] Students aged 7 and 8 years were not given the questionnaire due to concerns they may be too young to complete it reliably.
[c] Restricted to three ages 9, 12, and 15 years due to time and resource considerations and because these test results were not planned to be used to establish percentile norms.

3.2.3 Measurements

The ASHFS physical testing comprised field tests (including fitness tests such as push-ups and 50 metre run, and height and weight measurements), technical (e.g. skinfolds, blood pressure, lung function), and blood tests. The only ASHFS physical measures used in this thesis are the height and weight, which were used to calculate BMI as weight in kilograms, divided by squared height in metres (kg/m^2). Height was measured for students with bare feet to the nearest 0.1cm using KaWe Height tape

or rigid measuring tape. Weight was measured for students in light clothing (i.e. barefoot in. t-shirt/shirt and shorts/skirt only) to the nearest 0.5 kg, using beam or medical scales. The questionnaire collected data on demographics, lifestyle, health behaviours and attitudes, and physical activity participation in and outside of school. Specific information on the ASHFS variables used as covariates in this thesis are detailed in the methods sections of the relevant studies in Chapter 4 and Chapter 5.

3.2.4 Ethics

Participation in the ASHFS and the NDSS required signed parental consent. Parents could choose which components of the ASHFS and the NDSS their child participated in by selecting “YES” or “NO” to indicate consent for each of the following:

- Fitness and physical performance testing
- Questionnaire
- Food record
- Blood fat measure
- One-year follow-up of height and weight.

There was no information provided with the ASHFS data or survey results to indicate that the one-year follow-up was completed (159).

3.2.5 Funding

Funding for ASHFS was provided by the Commonwealth Department of Sport, Recreation and Tourism, the National Heart Foundation of Australia, the Commonwealth Schools Commission, and the Commonwealth Department of Health.

3.3 National Dietary Survey of Schoolchildren (NDSS)

3.3.1 Study rationale

Prior to the NDSS, data on the dietary intake of Australian children and adolescents were last collected in 1938 and 1944 in studies on household diets. In 1983 the Commonwealth Department of Community Services and Health conducted a National Dietary Survey of Adults. The 1985 NDSS was the first national study on the dietary intake of youth as a separate cohort. Whereas rationale for the surveys in 1938 and 1944 had been around identifying nutritional deficiencies and

undernutrition, the surveys in the 1980s were concerned with emerging problems of overnutrition and excess intakes of fat, added sugars and salt. The primary objectives of the NDSS were to determine food and nutrient intake of urban and rural schoolchildren across Australia, correlate the intake data with ASHFS health and fitness information, and to use the data to develop nutrition policy and education relevant to the population group.

3.3.2 Design and participants

The Australian Commonwealth Department of Health conducted the NDSS concurrently with the ASHFS, in collaboration with ACHPER. The survey design, training of data collectors, data entry and processing, and analysis of nutritional content of intake was headed by officers within the Nutrition Section of the Commonwealth Department of Health, with assistance from the Dietitians Association of Australia and nutrition consultants. ASHFS participants aged 10-15 years were invited to participate in the survey, which comprised a record of all food and beverages consumed during a 24-hour period. ASHFS participants aged 7-9 years were not included in the survey due to time and resource constraints and because this younger age group may have had difficulty completing the food record. Students were also excluded if they:

- Had not agreed to participate in the ASHFS physical testing.
- Were absent on the day of food record distribution or collection (this included suspension from school, on holidays, on work experience, sitting an examination, or being on a school camp or excursion).
- No longer attended the school.
- Left the food record at home or did not return the food record on the day of collection.
- Refused to participate.
- Forgot to complete the food record.

In total, 7,976 students aged 10-15 years were approached to participate in the ASHFS physical testing, which was completed by 5,589 students (70.1% response rate). Of the 5,589 students, 5,043 completed the NDSS, meaning that the NDSS response rate of eligible ASHFS participants aged 10-15 years was 90.2%. The

number of NDSS participants by age and sex is shown in Table 3.2. Participation was lower among the older age groups, with the lowest participation among females aged 14-15 years.

Table 3.2 National Dietary Survey of Schoolchildren participants by age and sex

Age (years)	Females		Males		Total	
	N	%	*N*	%	*N*	%
10	452	18.0	443	17.5	895	17.7
11	448	17.9	446	17.6	894	17.7
12	459	18.3	426	16.8	885	17.5
13	399	15.9	407	16.0	806	16.0
14	372	14.8	412	16.2	784	15.5
15	377	15.0	402	15.9	779	15.4
Total	2507	100.0	2536	100.0	5043	100.0

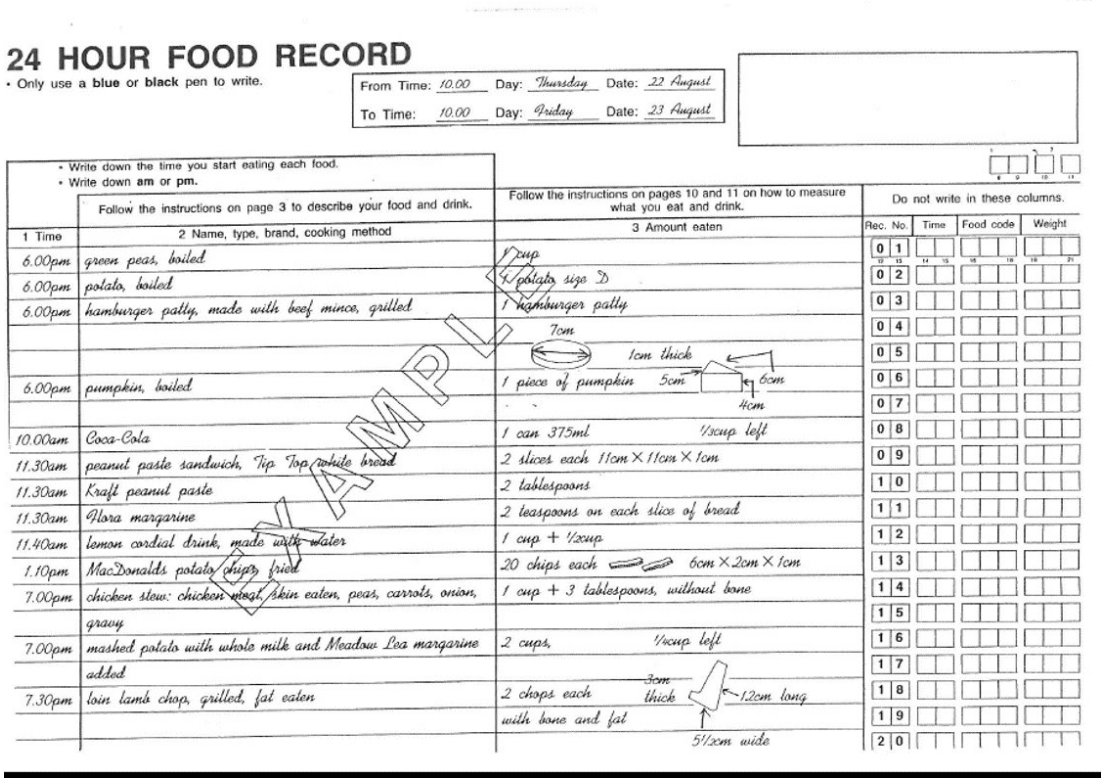
3.3.3 24-hour food record

The Department of Health selected a 24-hour food record as the method of data collection for the following reasons:

- Presence of the participants at schools meant that contact could be easily arranged with the participant for the instruction session before the recording period, and later collection/checking of records.
- The single recording period would minimise risk of participants changing their food intake to simplify the recording process, which is an issue associated with longer or repeat recording periods.
- Participants could fill out the record throughout the day and would not have to rely on memory.
- Quantities of food and beverages could be measured directly by the participants with the assistance of measuring aids provided.

The food record booklet included detailed instructions, space for recording detailed information on the amount, brand, cooking methods and type of food or beverage, a field for recording the time of intake, and circles and rulers to help estimate portion sizes. The example guide provided in the food record booklet is shown in Figure 3.2,

and the full record booklet is provided in Appendix A. Participants were given metric measuring cups and spoons to use for the measurements, that they could keep.



24 HOUR FOOD RECORD

• Only use a **blue** or **black** pen to write.

From Time: 10.00 Day: Thursday Date: 22 August

To Time: 10.00 Day: Friday Date: 23 August

• Write down the time you start eating each food.
• Write down **am** or **pm**.

Follow the instructions on page 3 to describe your food and drink.

Follow the instructions on pages 10 and 11 on how to measure what you eat and drink.

Do not write in these columns.

1 Time	2 Name, type, brand, cooking method	3 Amount eaten	Rec. No.	Time	Food code	Weight
6.00pm	green peas, boiled	1 cup	01			
6.00pm	potato, boiled	1 potato size 'D'	02			
6.00pm	hamburger patty, made with beef mince, grilled	1 hamburger patty 7cm 1cm thick	03			
			04			
			05			
6.00pm	pumpkin, boiled	1 piece of pumpkin 5cm 6cm 4cm	06			
			07			
10.00am	Coca-Cola	1 can 375ml ½cup left	08			
11.30am	peanut paste sandwich, Tip Top white bread	2 slices each 11cm × 11cm × 1cm	09			
11.30am	Kraft peanut paste	2 tablespoons	10			
11.30am	Flora margarine	2 teaspoons on each slice of bread	11			
11.40am	lemon cordial drink, made with water	1 cup + ½cup	12			
1.10pm	MacDonalds potato chips, fried	20 chips each 6cm × 2cm × 1cm	13			
7.00pm	chicken stew: chicken meat, skin eaten, peas, carrots, onion, gravy	1 cup + 3 tablespoons, without bone	14			
			15			
7.00pm	mashed potato with whole milk and Meadow Lea margarine added	2 cups, ½cup left	16			
			17			
7.30pm	loin lamb chop, grilled, fat eaten	2 chops each 3cm thick 12cm long with bone and fat 5½cm wide	18			
			19			
			20			

SAMPLE

Figure 3.2 Example 24-hour food record provided for the 1985 National Dietary Survey of Schoolchildren.

The trained data collectors attended the schools and showed students in groups of four or five how to fill out the food record booklet. The students were taken through a practice exercise of reporting that morning's breakfast, and the records were checked to ensure they included adequate detail to allow coding of the food or beverage and derive nutrient intake. The 24-hour recording period started immediately after the practice session. When the data collectors returned to collect the booklets, at least 24-hours later, each student was interviewed to check and clarify the entries for legibility and completeness.

The data was manually processed by Department of Health officers. A total of 775 Food Codes were used to describe the food and beverage items consumed. These Food Codes were categorised further into Food Subgroups of food and then into 15

major Food Groups (Table 3.3). For example, cheddar cheese was categorised under the "Cheese" Subgroup of the "Milk and milk products" Food Group. Most mixed dishes (e.g. quiche, pizza, beef stew) had their own codes and assigned nutrient compositions. In a small number of cases, mixed foods were separated into their main components. An example was a cream filled sponge cake being separated into sponge (recorded under the Subgroup "Cakes and cake type puddings"), jam (recorded under "Jams and lemon spreads"), and cream (recorded under "Creams"). The gram weight of each food item was calculated from the dimensions or measurements provided (participants had been instructed not to weigh their food). Only the edible weight of a food item was calculated. For example, the consumed quantity of an orange was weight without the peel.

Table 3.3 National Dietary Survey of Schoolchildren major Food Groups and total number of Food Subgroups identified from the coding frame and data

Food Group	Description	Number of Subgroups
A	Cereal and cereal products	22
B	Vegetables	11
C	Fruits	8
D	Meat and meat products	9
E	Fish, seafood and seafood products	3
F	Eggs	1
G	Nuts and seeds	2
H	Milk and milk products	9
I	Fats	7
J	Sugars, jams, honey and syrups	3
K	Confectionary	2
L	Snack foods	2
M	Beverages, non-alcoholic	9
N	Beverages, alcoholic	6
O	Condiments, flavourings and soups.	8

The nutrient composition of the consumed foods and beverages were determined using a specially compiled coding manual relevant to the Australian food context. The manual was built on "McCance and Widdowson's The composition of foods" (160), with adjustment for differences between British and Australian foods and

known ingredients of Australian processed foods. There were further adjustments from the partially compiled "Tables of composition of Australian foods" which would later be published in 1989 as the "Composition of Foods Australia" and produced as the NUTTAB electronic database (161). The following nutrients were calculated for each consumed item:

- Total energy (kJ)
- Protein (g)
- Total carbohydrates (g)
- Starch (g)
- Sugars (g)
- Dietary fibre (g)
- Total fat (g)
- Polyunsaturated fat (g)
- Monounsaturated fat (g)
- Saturated fat (g)
- Cholesterol (mg)
- Vitamin C (mg)
- Calcium (mg)
- Iron (mg)
- Zinc (mg)
- Magnesium (mg)
- Thiamin (mg)
- Riboflavin (mg)
- Niacin (mg)
- Retinol (µg)
- β-carotene (µg)

3.3.4 Ethics

Participation in ASHFS and the NDSS required signed parental consent as outlined in Section 3.2.4.

3.3.5 Funding

Funding for ASHFS is outlined in Section 3.2.5. The NDSS component was funded by the National Health and Medical Research Council and supported by Kellogg Australia Pty. Ltd.

3.4 Childhood Determinants of Adult Health (CDAH) study

3.4.1 Study rationale

In the year 2000, Professor Terence Dwyer, the director of the Menzies Centre for Population Health Research in Hobart, Tasmania (now the Menzies Institute for Medical Research), identified the ASHFS as a possible baseline study to examine the influence of childhood factors on adult health. A successful pilot study to trace and

follow-up the ASHFS participants was conducted in 2001, and ASHFS data was incorporated into what is now the Childhood Determinants of Adult Health (CDAH) study. The primary objective of the CDAH study is to determine if childhood factors are associated with adult cardiometabolic outcomes, and to determine the strength of these associations after taking into account adult measures (162). Secondary objectives include examining the associations of childhood factors with adult mental health and lifestyle. Multiple follow-ups in adulthood have also allowed examination of associations between factors in early adulthood with outcomes in later adulthood.

3.4.2 Design and participants

As ASHFS was not intended as a follow-up study there was limited identifying information available for tracing participants. First name, last name, school, and post-code of residence were used to trace participants during 2001-02. The information from 1985 was matched to historical and current electoral rolls and participants were sought through school networks, resulting in the location of 6,840 (80%) of ASHFS participants. Of those traced, 61% (n = 5,170) agreed to complete a brief enrolment questionnaire. A total of 3,992 (45.8% male) participants took part in the first follow-up during 2004-06 which comprised questionnaires and study clinics in major cities and regional centres nation-wide. A second follow-up (CDAH-2) was conducted during 2009-11 and comprised postal and online questionnaires and telephone interviews without study clinics. A total of 3,038 participants aged 31-41 years (42.3% male) took part. The third follow-up (CDAH-3), commenced with a pilot study in 2014 (220 participants), followed by Australia-wide study clinics during 2017-19. CDAH-3 included 2,074 participants overall (44.4% male), in the age range of 36-49 years old. A flow chart of study participation is shown in Figure 3.3.

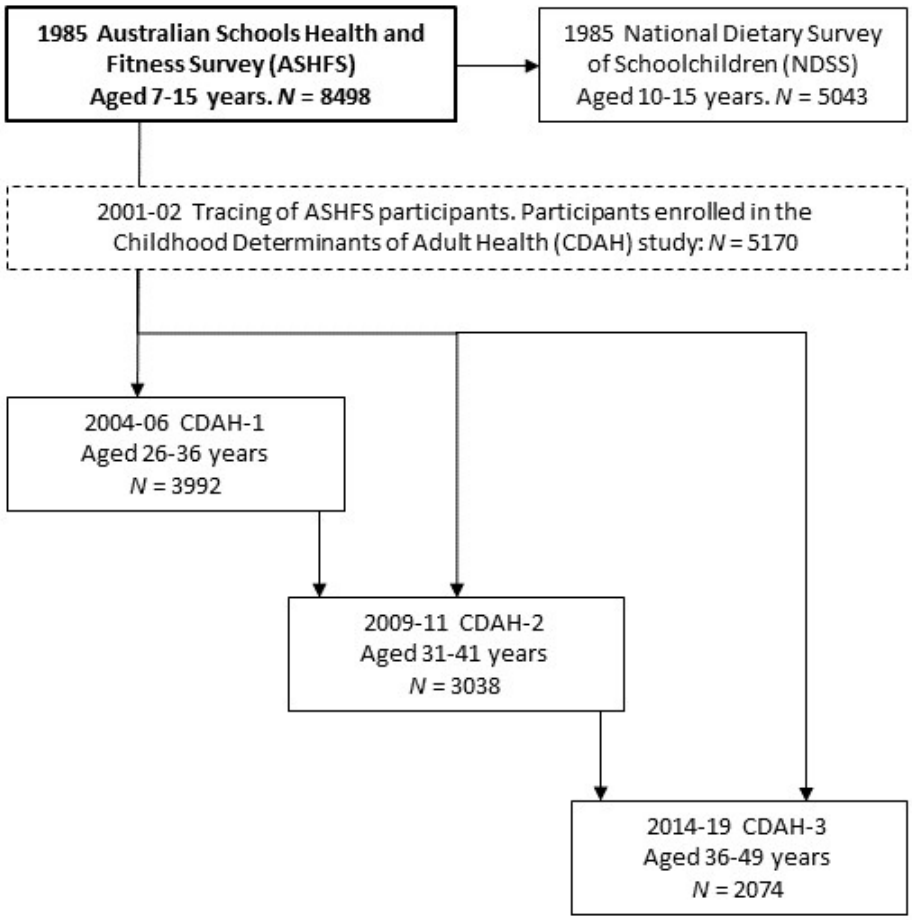


Figure 3.3 Childhood Determinants of Adult Health (CDAH) study participant flow chart

The overall number of participants and those who participated in the main study components used in this thesis are shown in Table 3.4. This thesis uses data from the general questionnaires, study clinics, dietary questionnaires, and mental health interviews. The general questionnaire included sections on demographics, medical conditions, family background, lifestyle practices, and social support. At CDAH-1 and CDAH-2 the general and dietary questionnaires were mailed to participants who returned them by post or for CDAH-1, at study clinics. The questionnaires at CDAH-3 were completed by participants online. The study clinics at CDAH-1 and CDAH-3 were held in major cities and regional centres across Australia. The clinics took participants around three hours to complete and rotated them through stations to collect data on physical measures including height, weight, waist circumference, blood pressure, fasting blood samples, ultrasound of carotid arteries, and physical fitness such as grip strength and cardiorespiratory fitness (measured on a stationary

bicycle). At CDAH-1 and CDAH-3, participants completed the computerised mental health interview on laptop computers during the clinics (see Section 3.4.4 below). At CDAH-2, trained interviewers administered the interviews over the telephone.

Table 3.4 Childhood Determinants of Adult Health study participants by sex and follow-up component at CDAH-1 (2004-06), CDAH-2 (2009-11), and CDAH-3 (2014-19)

	Female		Male		
	N[a]	%	*N*	%	Total
CDAH-1 total	2164	54.2	1828	45.8	3992
Study clinic	1259	52.3	1150	47.7	2409
General questionnaire	2154	54.3	1813	45.7	3967
Dietary questionnaires	1595	55.3	1287	44.7	2882
Mental health interview	1205	52.1	1109	47.9	2314
CDAH-2 total	1753	57.7	1285	42.3	3038
General questionnaire	1750	57.7	1285	42.3	3035
Dietary questionnaires	1128	63.4	652	36.6	1780
Mental health interview	1103	63.1	645	36.9	1748
CDAH-3 total	1154	55.6	920	44.4	2074
Study clinic	843	53.8	724	46.2	1567
General questionnaire	1142	55.7	910	44.3	2052
Dietary questionnaires	1125	55.5	902	44.5	2027
Mental health interview	801	53.5	697	46.5	1498

[a] This table details total participants of the study component, including responses that were later deemed incomplete, or were excluded for other reasons (e.g. pregnancy). The individual studies in Chapters 4-7 outline the number of participants with complete components and number excluded.

3.4.3 Dietary questionnaires

At the three CDAH follow-ups, the dietary questionnaires included a food frequency questionnaire (FFQ) and food habits questionnaire (FHQ) which collected information on usual dietary intake and dietary practices over the past 12 months, and a 24-hour meal chart that collected information on consumption of snacks, meals and beverages during the previous day. The full CDAH-1 dietary questionnaire is provided in Appendix B. The FFQ were based on the Anti-Cancer Council of Victoria FFQ, which had been adapted for Australian populations from questionnaires used in the US Nurses' Health Study and validated for use within Australian populations (163-165). The FHQ collected information on dietary behaviours and was developed for the CDAH study by colleagues from The Centre for Physical Activity and Nutrition Research at Deakin University, Melbourne.

The FFQ at CDAH-1 contained 127-items. To reflect changes in foods commonly consumed in the Australian population over time, the FFQ was expanded to 128 items at CDAH-2 (fried chicken added to differentiate from non-fried poultry products) and 132 items at CDAH-3 (kiwi fruit, avocado, beetroot and energy drinks added). The food and beverage items in the FFQ are grouped into eight categories (Table 3.5).

Table 3.5 Food groups and number of food items in the Childhood Determinants of Adult Health (CDAH) study food frequency questionnaires at CDAH-1, CDAH-2, and CDAH-3

Food Group	CDAH-1 food items	CDAH-2 food items	CDAH-3 food items
Dairy foods	10	10	10
Bread and cereal foods	10	10	10
Meat, fish, eggs	22	23	23
Sweets, baked goods, and snacks	18	18	18
Dressings and spreads	6	6	6
Non-dairy beverages	23	23	24
Vegetables (including frozen and canned)	27	27	28
Fruits	11	11	13
Total	127	128	132

The FFQ asked for frequency of consumption without specifying what a serving of each food or beverage type was. Participants could select one of nine responses for each item, ranging from “Never or less than once a month” to “6+ times per day”. As data on frequencies and not quantity of food intake was collected, energy and nutrient intakes were not calculated from the CDAH FFQ data.

Two questions in the FHQ asked about usual fruit and vegetable consumption and specified serving sizes. The first question was “How many serves of vegetables (excluding potatoes) do you usually eat each day (one serve = ½ cup cooked vegetables or 1 cup salad vegetables)?”. There were five response options: “One serve or less”, “2-3 serves”, “4-5 serves”, “6 or more serves”, “I don’t eat vegetables”. The second question was “How many serves of fruit do you usually eat each day (one serve = 1 medium piece of fruit or 1 cup of diced pieces)?”. There were also five response options: “One serve or less”, “2-3 serves”, “4-5 serves”, “6 or

more serves", "I don't eat fruit". Similar short questions developed for Australian populations regarding vegetable and fruit intake have been validated against weighed food records (166).

This thesis also used data from three other multiple-choice questions in the FHQs:

- What type of milk do you usually consume? Nine options: "Whole milk"; "Low/reduced fat milk"; "Skimmed milk"; "Evaporated or sweetened condensed milk"; "Soy milk"; "Vitamin/calcium enriched milk"; "Other milk"; "None of the above"; "Don't know".
- How often is the meat you eat trimmed of fat either before or after cooking? Four options: "Never/rarely"; "Sometimes"; "Usually"; "I don't eat meat".
- What type of spreads do you usually use on bread, savoury biscuits etc? Twelve options: "I do not use any spread"; "Butter"; "Poly-unsaturated margarine"; "Canola"; "Table margarine"; "Eta™ or light margarine"; "Omega 3 or phytosterol margarine"; "An oil (e.g. olive oil)"; "Cream cheese"; "Nut butter (e.g. peanut butter)"; "Another kind of spread"; "Don't know".

Packaged with the FFQ and FHQ was a previous-day 24-hour meal chart (Figure 3.4). The 24-hour meal chart asked participants whether they ate and/or drank anything during each hourly interval from 6:00 the previous morning to 23:00 the night before (e.g. 6am-7am, 7am-8am etc), and over the night period 23:00 to 6:00 that morning. Food options were "No", "A snack", "A small meal", "A large meal". Drink options were "No", "Alcohol", "Water", "Something else". Participants were asked what day of the week it was the day before (which they were completing the chart for), and were given examples of the food options:

- "Snacks include things like a biscuit or a piece of fruit"
- "A small meal would be something like beans on toast, boiled egg and bread, breakfast cereal, a pie or a pastie"
- "A large meal would be something like meat and three veg, or a large serving of fish and chips"

SECTION C: When, and what, did you eat and drink yesterday?

1. What day was it yesterday?

Monday ○ Tuesday ○ Wednesday ○ Thursday ○ Friday ○ Saturday ○ Sunday ○

Think back to **yesterday**. In the chart below fill in the circle to indicate the sorts of meals and drinks you had at each time of the day.
(Please remember to fill in a response for both food AND drink for each time period, even if you have not consumed anything)

Snacks *include things like a biscuit or a piece of fruit.*
A small meal *would be something like beans on toast, boiled egg and bread, breakfast cereal, a pie or a pastie.*
A large meal *would be something like meat and three veg, or a large serving of fish and chips.*
You may specify ***more than one*** *type of drink for each time period, e.g. alcohol and water*

TIME	Did you eat anything?				Did you drink anything?			
	No	A snack	A small meal	A large meal	No	Alcohol	Water	Something else
6-7am	○	○	○	○	○	○	○	○
7-8am	○	○	○	○	○	○	○	○
8-9am	○	○	○	○	○	○	○	○

Figure 3.4 Excerpt of the CDAH dietary questionnaire 24-hour meal chart

3.4.4 Mental health interview

The Composite International Diagnostic Interview (CIDI) (167) was used to assess mental disorders of the CDAH adult participants. The CIDI is a structured interview designed for epidemiological studies to assess mental disorders according to the American Psychiatric Association Diagnostic Statistical Manual of Mental Disorders, fourth edition (DSM-IV) criteria (9). The DSM fifth edition (DSM-V) was released in 2013, but for consistency DSM-IV criteria were used for all three CDAH follow-ups. The disorders of interest for this thesis were major depressive disorder (mild, moderate, or severe), and dysthymic disorder. The computerised CIDI Auto Version 2.1 was completed by CDAH-1 and CDAH-3 clinic participants on laptop computers at study clinics. At CDAH-2, trained telephone interviewers administered the CIDI. At CDAH-1, the 12-month version of the CIDI was used, to identify mood, anxiety, and substance or alcohol abuse/dependence disorders experienced during the 12 months prior to the interview. At CDAH-2 and CDAH-3, lifetime versions of the CIDI assessed mood and anxiety disorders experienced during the participant's lifetime.

The CIDI also collects data on the age (in years) of onset of disorders when respondents first experienced symptoms, the age of most recent recurrence, and when symptoms had last been experienced (within last 2 weeks, 2 weeks – 1 month ago, 1-6 months ago, 6 months – 1 year ago, in last 12 months, or more than 1 year ago). This meant that it could be determined when an individual had their first onset of a mood disorder and when they experienced their most recent recurrence of the disorder, but the number and frequency of recurrent episodes in between was not captured. A summary of the CIDI delivery methods, time-period of mood disorder diagnosis, and the time-related data captured at each CDAH follow-up, are outlined in Table 3.6.

Table 3.6 Twelve-month and lifetime mood disorder measures captured with the Composite International Diagnostic Interview (CIDI) at each Childhood Determinants of Adult Health (CDAH) follow-up

Follow-up	Delivery method	Diagnosis (mood disorder/no mood disorder)[a]			Time-related measures		
		12-month/ lifetime CIDI	12-month mood disorder	Lifetime mood disorder	Age of first onset	Age of most recent symptoms	Recency of symptoms[b]
CDAH-1 (2004-06)	Computers at study clinics	12-month	Disorder during 12 months prior to interview	Not measured	Participants with a 12-month disorder reported the age they first experienced the disorder	Participants with a 12-month disorder reported the age that they last experienced symptoms	Participants with a 12-month disorder reported how long ago they last experienced symptoms
CDAH-2 (2009-11)	Phone interview	Lifetime	Disorder during 12 months prior to interview - derived from lifetime diagnosis and recency of symptoms.	Disorder at any time up to interview	Participants with a lifetime disorder reported the age they first experienced the disorder	Participants with a lifetime disorder reported the age that they last experienced symptoms	Participants with a lifetime disorder reported how long ago they last experienced symptoms
CDAH-3 (2014-19)	Computers at study clinics	Lifetime	Disorder during 12 months prior to interview - derived from lifetime diagnosis and recency of symptoms.	Disorder at any time up to interview	Participants with a lifetime disorder reported the age they first experienced the disorder	Participants with a lifetime disorder reported the age that they last experienced symptoms	Participants with a lifetime disorder reported how long ago they last experienced symptoms

[b] Mood disorder includes major depressive disorder (mild, moderate or severe) and dysthymic disorder.

[b] Recency response categories for both the 12-month and lifetime CIDI were: within last 2 weeks; 2 weeks – 1 month ago; 1-6 months ago; 6 months – 1 year ago; In last 12 months; More than 1 year ago.

3.4.5 Covariate measurements

A range of sociodemographic and lifestyle measures were collected at each follow-up using questionnaires. The methods section of Chapters 4-7 provide details on the relevant questionnaire data that were used in each original study. Several physical and fasting blood biochemistry measures were also used and are outlined in the relevant Chapter, but detailed methodology is provided below for completeness.

Physical measurements

At CDAH-1 and CDAH-3 study clinics, height and weight were measured at the study clinics by staff who had been trained by a qualified anthropometrist. Participants wore light clothing, without shoes. Height was measured to the nearest 0.1cm using a Leicester stadiometer (Invicta, Leicester, UK), and weight was measured to the nearest 0.1kg using a Heine portable scale (Heine, Dover, NH, US). Waist circumference (used in Chapter 4), was measured to the nearest 0.1cm with non-stretch tape at the narrowest point between the lower costal border and iliac crest. For CDAH-1 participants who did not attend study clinic, self-reported height and weight from questionnaires had a correction factor applied to account for mis-reporting (168). The correction factor was derived from linear regression of the self-reported height and weight and measured height and weight of a subset of 1,185 CDAH-1 participants (168) The correction factor was also applied the height and weight self-reported in questionnaires at CDAH-2, which did not involve study clinics. Body mass index (BMI) was calculated as weight in kilograms divided by squared height in metres (kg/m^2).

Adult blood pressure measures were used in the Chapter 4 study. Before blood pressure was taken, participants had a blood pressure arm cuff of appropriate size fitted and were instructed to sit quietly for five minutes. Three measures of systolic and diastolic blood pressure were taken from the right brachial artery using the Omron HEM907 digital automatic monitor (Omron Healthcare Co, Ltd, Kyoto, Japan). There was a one-minute interval between each reading. The mean of the three readings was used.

Fasting blood biochemistry

Fasting blood biochemistry measures from samples collected at the CDAH-1 study clinics were used in Chapter 4. Participants had been instructed to not have any food or drinks apart from water and regular medications during the 12 hours overnight prior to their clinic appointment. A qualified nurse took a 30ml blood sample from the antecubital vein into two tubes (one for lipid and insulin analysis and one for glucose). The samples were centrifuged to separate the serum for the insulin and lipids analysis and plasma for the glucose analysis and then stored upright at 4° Celsius. Samples were sent in insulated containers with cold packs by overnight courier to the laboratory (Medvet, Adelaide, South Australia). An Olympus AU5400 automated analyser (Olympus Optical, Tokyo, Japan) was used to enzymatically determine concentrations of glucose, total and HDL cholesterol, and triglycerides (169). Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation (170). Fasting insulin concentrations were measured by a microparticle enzyme immunoassay kit (AxSYM; Abbot Laboratories, Abbot Park, IL, US) or an electrochemiluminescence immunoassay (Elecsys Modular Analytic E170; Roche Diagnostics, Mannheim, Switzerland). The two methods were due to the contracted laboratory changing their assay part way through the study clinic data collection (171). A correction factor of 0.81 was applied to the insulin values assessed with the microparticle-enzyme immunoassay so that the levels were consistent (171). Insulin resistance was estimated using the homoeostatic model assessment index using fasting glucose and fasting insulin (172). In 2009, serum samples from female participants only, which had been stored at -70°C since 2004-06, were sent to the Institute of Medical and Veterinary Sciences in Adelaide, South Australia. The samples were analysed for serum folate using a chemiluminescent microparticle folate binding protein assay on an Abbott Architect analyser (Abbott, IL, US) (173).

3.4.6 Ethics

The CDAH study was approved by the Southern Tasmania Health and Medical Research Ethics Committee operating within the guidelines of the National Health

and Medical Research Council. All participants gave informed written consent at each CDAH follow-up.

3.4.7 Funding

The CDAH study was funded with grants from the National Health and Medical Research Council, the National Heart Foundation of Australia, the Tasmanian Community Fund, and Veolia Environmental Services. Additional support for CDAH-1 was provided by Sanitarium Health Food Company, ASICS Oceania, and Target Australia. No funding organisation had any role in the design or conduct of the study or the collection, management, analysis, and interpretation of the data.

3.5 Processing of dietary data

3.5.1 Identifying NDSS food items and matching to CDAH FFQ items

The NDSS dietary data contained multiple records for each participant, detailing the time of the eating or drinking occasion, the coded item (each with a Food Group, Food Subgroup, and Food Group code) and the composition of nutrients outlined in Section 3.3.3. To allow comparisons and consistency in the assessment of food and beverage intake between the youth and adult data, the NDSS Food Groups, Food Subgroups and Food Codes of the youth data were used to categorise items consumed to the food groupings used in the CDAH FFQs. The definition of the Food Groups, Subgroups, and Codes were outlined in the NDSS "User's Guide for the Machine-Readable Data File (SSDA Study no. 617)" (174) and the government report "The Bridging Study - comparing results from the 1983, 1985 and 1995 Australian national nutrition surveys" (the Bridging Study) (161). For example, an item with Food Code = 490 is defined as "Strawberries, raw", and has Food Group = C ("Fruit"), and Food Subgroup = 6 ("Berry fruits"). The user guide had definitions for all Food Groups A-O, and Food Subgroups (e.g. Food Group C had Subgroups 1-8) (see Table 3.3. in Section 3.3.3). For unknown reasons, not all 775 Food Codes that appeared in the NDSS dietary data were defined in the user guide – only 627 codes with definitions are listed. No further information was able to be obtained from the Australian Data Archive at the Australian National University. However, the Bridging Study report included information on some of the missing 1985 Food Codes (161). The study outlined and defined codes used in the 1985 studies that were mapped to

codes for an updated food composition database (NUTTAB 91 /92) (161). For any Food Codes that were not defined in either document, these items were categorised based on their Food Groups and Food Subgroup. An example is shown in Table 3.6. Food Code 12 is listed as “Pasta, boiled” and Food Code 24 is not listed. Because the Food Group and Subgroup are known (Food Group A = “Cereal and cereal products”; Subgroup = “Pasta”), the observations for Food Code 24 can be categorised to the CDAH food group: “Bread and Cereal Foods”, food type: “Pasta (including filled), noodles”. The full list of categorised codes is outlined in Appendix C, Table C.1.

Table 3.7 Example of matching non-missing and missing National Dietary Survey of Schoolchildren Food Codes to Childhood Determinant of Adult Health food items

NDSS					Match to CDAH	
Food Group	Food Subgroup	Food Code	Food Code Defined	Food Code Name	Food Group	Item
A	5	12	Yes	Pasta, boiled	Bread & Cereal Foods	Pasta
A	5	24	No	-		

In some cases, there were no suitable FFQ categories for the 24-hour food record item. In these cases, they were grouped with foods of similar nutritional quality or usage. For example, radishes, swedes, celeriac and parsnips were grouped with carrots as they were all root vegetables. There were 41 Food Codes that were unable to be re-categorised into the CDAH FFQ food groups. These included sauces, spices, flavourings, stocks, oils, yeast and thickeners used in cooking and are outlined in Appendix C, Table C.2.

3.5.2 Calculating standard servings of food and beverages

To convert the dietary data from the NDSS into number of daily standard serves, the standard serve sizes outlined in the 2013 Australian Dietary Guidelines (the Guidelines) were used (81). The Guidelines define standard serve sizes for the five core food groups that people are encouraged to eat a variety of (vegetables, fruit, grains (cereals), lean meats or alternatives, and dairy or alternatives) and the discretionary foods that people are encouraged to limit (processed foods, foods high in saturated fats and foods with added sugar and/or salt) (81).

NDSS dietary data

For every participant, the NDSS data included gram weight and kilojoules of energy for each food or beverage item consumed. These amounts were used to identify the number (or proportion) of standard serves for each reported item. The Guidelines give some standard serves as a range of weight or energy content due to differing density of foods. For example, a standard serve of a grain food ranges from 30-120g in weight but has one energy value specified of 500kJ. Gram weight was used to calculate serves for vegetables, fruits, meat or alternatives, and dairy or alternatives, whereas energy in kilojoules was used to calculate serves of grains and discretionary foods. The standard serve sizes are outlined in Table 3.7. Where the Guidelines specify a volume, this was equated to grams in weight e.g. 125 grams of 100% orange juice was equated to one serve (125ml) of 100% fruit juice. For mixed dishes (e.g. cottage pie), the value of 600kJ was used to calculate a standard serve. The Guidelines specify one set of standard serve sizes applicable for all age groups, but the number of serves recommended for each food group differ by age and sex (81).

Table 3.8 Standard serve sizes of foods in grams (g) and kilojoules (kJ), outlined in the Australian Dietary Guidelines 2013

Food Group	Weight (g)	Energy (kJ)
Vegetables[a]		
Cooked vegetables	75	100-350
Salad vegetables	75	100
Fruit		
Fresh, cooked or canned	150	350
Dried fruit	30	350
100% fruit juice	125	350
Grains and cereals[b]	30-120	500
Lean meats or alternatives		
Cooked lean red meats	65	500-600
Cooked poultry	80	500-600
Cooked or canned fish	100	500-600
Eggs	120	500-600
Cooked or canned legumes or beans	150	500-600
Tofu	170	500-600
Nuts and seeds	30	500-600
Dairy or alternatives		
Fresh, long-life, or reconstituted powdered milk	250	500-600
Evaporated milk	120	500-600
Hard cheese	40	500-600
Ricotta	120	500-600
Yoghurt	200	500-600
Soy or other milks with added calcium	250	500-600
Discretionary foods[c]	-	600

The highlighted cells indicate the measurement used to calculate serving sizes for items within that food group from the National Dietary Survey of Schoolchildren data.
[a] Vegetables include: cooked brassica, cruciferous or orange vegetables; dried or canned beans, chickpeas, or lentils; raw green leafy vegetables; starchy vegetables (e.g. potato, sweet potato, taro, sweet corn or cassava); other (e.g. tomato).
[b] Grains and cereals include bread, flat bread, crispbread, crumpets, English muffins, scones, flour, cereal flakes, muesli, porridge, and cooked grains (e.g. rice, pasta, noodles, barley, semolina, polenta, quinoa, bulgur).
[c] Discretionary foods include alcohol, cakes, biscuits, pastries, desserts, confectionary, sugar-sweetened drinks, savoury processed snack foods, processed meats. Due to the wide variation of composition of items in this group, no gram weight was specified for a standard serve.

CDAH dietary data

The frequency of consumption of foods in the CDAH FFQs were used to calculate daily equivalent standard servings as per accepted methods of assuming one reported frequency was equivalent to one standard serve (76, 175). For example, if a food item was reported as consumed once per week, this was equated to 0.143 daily serves (one serve divided by seven days). Where the response option was a range,

the mid-point was used in the calculation (e.g. 1-3 times per month was calculated as 2/30), except for "never or less than once a month" where a lower amount was used (1/80 = 0.0125) to account for the likelihood of respondents never consuming the item. The following conversions to equivalent daily serves were used:

- Never or less than once a month: 1/80 = 0.0125 serves
- 1-3 times per month: 2/30 = 0.067 serves
- Once per week: 1/7 = 0.143 serves
- 2-4 times per week: 3/7 = 0.429 serves
- 5-6 times per week: 5.5/7 = 0.786 serves
- Once per day: 1/1 = 1 serve
- 2-3 times per day: 2.5/1 = 2.5 serves
- 4-5 times per day: 4.5/1 = 4.5 serves
- 6+ times per day: 6/1 = 6 serves.

Responses to FHQ questions about fruit and vegetable consumption were also converted to a daily equivalent as follows:

- One serve or less = 1 serve
- 2-3 serves = 2.5 serves
- 4-5 serves = 4.5 serves
- 6 or more serves = 6 serves
- I don't eat vegetables = 0 serves.

The resulting daily equivalent serves of food and beverage items in youth and adulthood were used to calculate measures of diet quality used in the original studies comprising this thesis. Further details are outlined in the method sections of the relevant chapters.

3.6 Statistical analyses

3.6.1 Study specific analyses

The statistical methods used in the four original studies comprising this thesis are outlined in the method sections of Chapters 4-7. Additional detail on identification of over or under reporters of dietary intake from the NDSS, and inverse probability weighting to account for loss to follow-up in regression analyses, are outlined below.

3.6.2 Identifying under- and over-reporters of energy intake in the NDSS

The Goldberg method using the Schofield equations for basal metabolic rate by age and sex, was used to identify NDSS under- and over-reporters of energy intake (176-178). Calculation of the Goldberg cut-offs used the suggested values for within subject energy intake variation (23%), within subject variation in estimated BMR (8.5%), and between subject variation in Physical Activity Level (PAL) (15%) (176). The cut offs were calculated using the conservative PAL value of 1.55, which is deemed "light activity" (179). NDSS participants were classified as under-reporters (*n* = 445, 8.8%), plausible reporters (*n* = 4,514, 89.5%), or over-reporters (*n* = 84, 1.7%). No participants were excluded from the analyses used in this thesis based on their Goldberg values, due to the variation that could occur from measuring food intake over a single 24-hour period. As show in Table 3.8, under-reporters were more likely to be female and older (14-15 years of age), which is in line with previously reported trends in other adolescent cohorts (180, 181).

Table 3.9 Under and over reporting of energy intake in the National Dietary Survey of Schoolchildren according to the Goldberg method

Age (years)	Under-reporter		Plausible reporter		Over-reporter		Total	
	n	%	*n*	%	*n*	%	*n*	%
Females								
10	32	12.0	417	18.8	3	14.3	452	18.0
11	30	11.3	417	18.8	1	4.8	448	17.9
12	41	15.4	410	18.5	8	38.1	459	18.3
13	48	18.0	347	15.6	4	19.0	399	15.9
14	53	19.9	314	14.1	5	23.8	372	14.8
15	62	23.3	315	14.2	0	0.0	377	15.0
Sub total	266	100.0	2220	100.0	21	100.0	2507	100.0
Males								
10	23	12.8	414	18.0	6	9.5	443	17.5
11	31	17.3	406	17.7	9	14.3	446	17.6
12	35	19.6	383	16.7	8	12.7	426	16.8
13	36	20.1	357	15.6	14	22.2	407	16.0
14	33	18.4	369	16.1	10	15.9	412	16.2
15	21	11.7	365	15.9	16	25.4	402	15.9
Sub total	179	100.0	2294	100.0	63	100.0	2536	100.0
Total	445		4514		84		5043	

This method was not applied to the CDAH follow-ups as the dietary data were based on frequency of consumption, not amount, and energy intake was not calculated.

3.6.3 Multiple imputation and inverse probability weighting

The ASHFS was a nationally representative sample, however there was considerable loss to follow-up in the CDAH study. To reduce the possibility of bias due to loss to follow-up, a two-step approach was used, motivated by the work of Seaman et al. 2012 (182). For relevant regression analyses, multiple imputation was performed using up to 50 chained equations to replace missing data on ASHFS covariates, where necessary. The covariates were those that were thought to predict loss to follow-up: age, sex, area-level socioeconomic status (high, medium-high, medium-low, low), BMI, usual breakfast eating before school (usually eat (four or more times per week), don't usually eat), school type (state, Catholic, independent), smoking status (never, less than 10 cigarettes, 10 or more cigarettes), and school-reported academic ability (excellent, above average, average, below average, poor). Inverse probability weighting on these variables was then applied in the regression analyses to help account for those from the baseline sample who were under-represented in the follow-up data.

3.7 Summary

The CDAH study is a longitudinal cohort study that has so far conducted three follow-ups with participants of the nationally representative 1985 Australian Schools Health and Fitness Survey of youth aged 7-15 years (*N* = 8,498). Among the ASHFS participants, 5,043 participants aged 10-15 years also participated in the 1985 National Dietary Survey of Schoolchildren. The primary aim of the CDAH study is to examine the influence of childhood factors on adult health outcomes. Data used in this thesis includes dietary data from a 24-hour food record in youth, food frequency questionnaires in adulthood, diagnostic mood disorder measures in adulthood, and a range of baseline and follow-up covariate measures.

Chapter 4: An age- and sex-specific dietary guidelines index is a valid measure of diet quality in an Australian cohort during youth and adulthood

4.1 Preface

Measuring diet quality over time is important due to health impacts, but to our knowledge, a Dietary Guideline Index (DGI) with consistent scoring across childhood/adolescence (youth) and adulthood has not been validated. The evidenced based 2013 Australian Dietary Guidelines were used determine diet quality. Although the Mediterranean diet is regarded as a dietary model that promotes good health and as outlined in the Chapter 2 literature review, has been associated with lower risk of depressive symptoms, there were low intakes of core components of the Mediterranean diet among the youth cohort (legumes, fish, wine). The low intakes would affect the validity of these components and result in a narrow distribution of scores and therefore a dietary guidelines index was deemed more appropriate. We hypothesised that a DGI that reflected age- and sex-specific guidelines would be a valid measure of diet quality in youth and adulthood. A validated index provides the foundation to examine associations between diet quality and mental health outcomes in the subsequent chapters. The text from this chapter was accepted and published in Nutrition Research in January 2019 (183).

4.2 Introduction

Understanding diet quality over the life course is important due to associations between diet and highly prevalent health conditions including obesity related illness and mental disorders (116, 184). Diet in observational studies is often assessed using a dietary index, which applies pre-conceived concepts of diet quality such as evidence-based dietary guidelines, to calculate an overall score from food and drink intake (70). An advantage over methods such as dietary pattern analysis, is that an index is a standard measure that compares what participants are eating to what is

recommended for good health, and allows comparison of diet quality over time and between populations (75).

Dietary guidelines differ between countries according to food cultures, but commonly aim to achieve adequate nutrition and reduce risk of diet related disease (185). Similar to other western countries such as the US and UK, the Australian Dietary Guidelines are food-based guidelines encouraging intakes of core food groups ("eat more": vegetables, fruit, wholegrains, reduced fat dairy, and lean meat), while recommending limited intake of discretionary foods ("eat less": alcohol and foods high in saturated fat, added sugar or salt) (81). The Australian "eat more" and "eat less" guidelines differ in the number of recommended servings by sex and age group but are otherwise consistent from the age of two years onwards. However, existing validated Australian dietary guideline indices for children/adolescents (youth) and adults are not directly comparable due to their different composition (76, 78, 79, 186). Validation of a consistent index for application among both youth and adults is lacking in Australia and internationally (187, 188). Furthermore, although analyses of data collected several years or even decades ago is common in epidemiology, there has been little focus on the challenges posed in applying dietary indices retrospectively. Whereas other data types are objective (e.g. weight), dietary measures require interpretation if researchers wish to examine effects of overall diet quality rather than single food groups or nutrients.

The aim of this study was to revise a previously validated Australian dietary guidelines index and evaluate its appropriateness as a measure of diet quality in both youth and adulthood in an Australian cohort. Our index, hereafter referred to as the DGI, reflects the evidence-based 2013 Australian Dietary Guidelines to assess food intake against a current understanding of a healthy and nutritionally adequate diet. Appropriateness of the index was determined by evaluating measures of construct validity as to whether the DGI adequately reflects variation in diet quality according to measured intake, and is associated with dietary and population characteristics as would be predicted based on existing literature (189, 190). We hypothesised that our DGI, which reflected age- and sex-specific guidelines, would

be a valid measure of diet quality in both childhood/adolescence (youth) and adulthood. To test the hypothesis, we used several validation methods recognised within nutritional epidemiology (189). Construct validity was evaluated by distribution of index scores, index dimensionality (whether variation in dietary intake among the sample is explained by more than one linear combination of components), and in youth, associations between DGI and nutritional quality of diet. A further type of construct validity, concurrent criterion validity, was evaluated by assessing discrimination between population characteristics including sociodemographic variables, and cardio-metabolic risk factors by DGI scores according to known differences, such as the social gradient of diet in which lower diet quality is often associated with lower socioeconomic status (SES) (189-191). Internal consistency of the index was also examined.

4.3 Methods

4.3.1 Study design and sample

In 1985, the Australian Department of Community Services and Health conducted the Australian Schools Health and Fitness Survey (ASHFS) of schoolchildren aged 7-15 years. A two-stage probability design was used to achieve a nationally representative sample across all Australian states and territories, with 109 schools participating (90.1% response rate) (159). A sample size target of 500 students of each sex at each of the age levels 7 to 15 years was determined to be able to permit estimates from the questionnaire data that were within 10% of the population means. The overall student response rate from the 12,578 students approached was 67.6% (*N* = 8,498) (159). Students aged 10-15 years (*N* = 5,589) were invited to participate in the concurrent National Dietary Survey of Schoolchildren (NDSS).

During 2001-02, ASHFS participants were traced and invited to take part in the Childhood Determinants of Adult Health (CDAH) study, resulting in enrolment of 5,170 (61.0%) participants (162). During 2004-06, the first CDAH follow-up was conducted comprising questionnaires (*n* = 3,967) and study clinics for physical measurements (*n* = 2,410).

The State Directors General of Education approved the ASHFS, and signed parental consent was required. The CDAH study protocol was approved by the Southern Tasmanian Health and Medical Ethics Committee, operating in accordance with National Health and Medical Research Council requirements, and all participants gave informed written consent. All protocols conformed to the ethical guidelines of the 1975 Declaration of Helsinki.

4.3.2 Dietary measures in youth

In 1985, NDSS participants recorded the time and estimated amount of each food or drink item consumed during a 24-hour period. Trained data collectors showed students in groups of four or five how to measure and record their intake in the food record booklet with the aid of circles, rulers, and metric cups and spoons. The students did a practice exercise and the 24-hour recording period started immediately after the session. When the food records were collected, each student was interviewed to check and clarify the entries.

Survey design, collection and processing of NDSS data was coordinated by the Department of Community Services and Health with assistance from the Dietitians Association of Australia (192). Energy, and macro and micronutrient compositions were determined in 1985 according to the edible weight of each item (e.g. peeled orange), from a specially compiled database based on the British "McCance and Widdowson's The composition of foods", and adjusted for differences in Australian foods with "Metric tables of composition of Australian foods" (160, 161, 193).

To score the DGI, all items consumed were converted to a proportion of a standard serving as defined in the 2013 Australian Dietary Guidelines (81). Servings were determined by grams for the core food groups (fruit, vegetables, grains, lean meats/fish or alternatives, and dairy or alternatives), with the exception of cooked grains which were calculated by kilojoules (450kJ) due to variations in weight (81). For example, if a participant reported consuming 60g of toast at breakfast, this was equated to 1.5 standard 40g servings of bread. For mixed dishes (e.g. casserole, stir-fry) or discretionary items (high in saturated fat, or added sugars or salt, e.g. processed meats, cakes, potato chips, sugar-sweetened drinks), one serving was

defined as a portion equivalent to 600kJ (81). For example, a 1,200kJ serving of hot potato chips equated to two servings of discretionary food.

Total daily energy density of food intake was calculated by a participant's total daily energy in kJ, divided by their total daily grams of food consumed. Energy adjusted daily macro and micronutrient density intakes for each participant were calculated as total daily nutrient values divided by total daily energy intake, reported as units per 1MJ (1,000kJ).

4.3.3 Dietary measures in adulthood

Participants completed a 127-item food frequency questionnaire (FFQ), and a food habits questionnaire (FHQ) which were modified versions of those previously used in the 1995 National Nutrition Survey and based on a validated FFQ and FHQ developed for Australian populations (163, 164). The multiple choice FFQ asked for the average number of times each food or beverage was consumed daily, weekly, or monthly during the previous 12 months. Nine response options ranged from "Never or less than once a month" to "6+ times per day". Daily equivalents were calculated for each FFQ item, assuming one standard serving was consumed at each eating occasion (76, 175). For example, if a participant responded that they ate fresh fish "Once per week", their daily serving of fresh fish was calculated as one seventh of a 100g standard serving of fish (14.3g). Average daily servings of fruit and vegetables were estimated from two multiple-choice questions in the FHQ that asked participants to report how many servings of fruit and vegetables they usually consumed each day and provided examples of serving sizes. The FHQ also asked about type of milk usually consumed (e.g. full fat, reduced fat), type of spread usually used, and how often fat was trimmed from meat. Energy intake was not calculated as the FFQ only collected data on the frequency of consumption, not the amount consumed.

4.3.4 Dietary Guidelines Index composition

The DGI comprises nine indicators reflecting the 2013 Australian Dietary Guidelines. Current guidelines were used for interpretability of results, as they reflect current understanding of food-based nutritional intakes required for good health. The indicators and scoring criteria are described in detail in Table 4.1**.** Seven indicators,

worth 10 points each, related to recommended minimum intakes (dietary variety, vegetables, fruit, grains, lean meats and alternatives, low fat dairy and alternatives, water). Two indicators were for limiting intake of discretionary foods (worth 20 points) and replacing saturated fats with unsaturated fats (10 points). Previous versions of the adult DGI included separate indicators for alcohol, added sugar, and added salt (76, 78). However, in our DGI, these were combined into the single discretionary food indicator, consistent with the youth index, as processed foods may be high in combinations of these ingredients. The maximum possible DGI score was 100, with a higher score indicating higher diet quality. As outlined in Table 4.1, cut-offs for achieving maximum and minimum scores for each indicator were determined according to the age- and sex-specific serving recommendations, or the nutritional quality of the food (e.g. proportion of wholegrains to all grains) (81, 157, 194). Proportionate scores were given for partially meeting recommendations, for example if a participant reported one daily serving of fruit rather than the recommended minimum of two serves, they received five of the potential 10 points.

Table 4.1 Age and sex specific Dietary Guidelines Index scoring matrix, based on the 2013 Australian Dietary Guidelines

Dietary Guideline	Indicator and Description	Max score	Criteria for maximum score by sex and age in years: Boys 9-11	Boys 12-13	Boys 14-18	Girls 9-11	Girls 12-13	Girls 14-18	Men 19-50	Women 19-50	Criteria for minimum score
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 serves from any of the core food groups
2. Vegetables, including legumes/beans.	2. Servings of vegetables per day including legumes/beans.	10	≥ 5	≥ 5.5	≥ 5.5	≥ 5	≥ 5	≥ 5	≥ 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	≥ 2	≥ 2	≥ 2	≥ 2	≥ 2	≥ 2	0 servings
4. Grains, mostly whole-grain and/or high fibre.	4a. Servings of breads/cereals per day.	5	≥ 5	≥ 6	≥ 7	≥ 4	≥ 5	≥ 7	≥ 6	≥ 6	0 servings
	4b. Wholegrains as a proportion of total grains[a].	5	100%	100%	100%	100%	100%	100%	100%	100%	0%
5. Lean meat and poultry, fish, eggs, tofu, nuts/seeds, and legumes/beans.	5a. Servings of meats or alternatives per day (excluding processed meats).	5	≥ 2.5	≥ 2.5	≥ 2.5	≥ 2.5	≥ 2.5	≥ 2.5	≥ 3	≥ 2.5	0 servings
	5b. Lean meats/ alternatives to total meat/ alternatives.	5	100%	100%	100%	100%	100%	100%	100%	100%	0%
6. Dairy and/or alternatives, mostly reduced fat.	6a. Servings per day of total dairy or alternatives.	5	≥ 2.5	≥ 3.5	≥ 3.5	≥ 3	≥ 3.5	≥ 3.5	≥ 2.5	≥ 2.5	0 servings
	6b. Reduced fat dairy or alternatives to total dairy or alternatives.	5	100%	100%	100%	100%	100%	100%	Skim, low, or reduced fat milk or alternatives		Child: 0% / Adult: Whole milk

7. Drink plenty of water[b]	7a. Servings per day of fluids, excluding alcohol[c].		5	≥ 6	≥ 6	≥ 7	≥ 5	≥ 5	≥ 6	≥ 10	≥ 8	0 servings	
	7b. Proportion of water to total fluid intake per day, excluding alcohol[c].		5	≥5 0%	≥ 50%	≥ 50%	≥ 50%	≥ 50%	≥ 50%	≥ 50%	≥ 50%	0%	
Limit intake													
8. Limit intake of saturated fat, alcohol and added salt and sugars.	8. Servings per day of foods high in saturated fat, added sugars or salt[d]. Alcohol was included for adults.		20	≤ 1.5	≤ 1.5	≤ 2.5	≤ 1.5	≤ 1.25	≤ 1.25	≤ 1.5	≤ 1.25	Males 9-13/19-50: > 3; Males 14-18: > 5; Females 9-11: > 3; Females 12-50: > 2.5	
9. Replace saturated fats with unsaturated fats.	Child: 9. kJ from healthy fats/oils as proportion of total fats/oils.	Adult: 9a. Trimming fat from meat.	5	80%[e]	80%	80%	80%	80%	80%	Usually		Child: No un-saturated fats eaten	Adult: Never/ rarely
		Adult: 9b. Type of spread usually used.	5							Spreads low in saturated fat			Spreads high in saturated fats
	Total:		100										

[a] Childhood: included bread and breakfast cereals. Adulthood: calculated for bread only. This was due to the different data from different measurement methods.
[b] The water intake cut-offs are based on Nutrient Reference Values for Australia and New Zealand and the proportion of water to total fluids was derived by McNaughton et al. (2008) from the US Beverage Guideline Panel recommendations (76, 157, 194).
[c] Childhood: tea and coffee excluded from overall fluid intake as not recommended for children.
[d] Additional servings of discretionary choices are only recommended for active, taller children or adults, or older children in the age range, and where possible extra foods should be eaten from the five core food groups (81). 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.
[e] 80% is used as the maximum, in recognition that eggs and cheese which contain saturated fat are included in recommended food groups (81). Nutrient Reference Values for Australia and New Zealand recommended that saturated and trans fat comprise no more than 10% of daily energy intake (157).

4.3.5 Covariates in youth

The ASHFS questionnaire included questions on demographics, lifestyle, health attitudes, and sport and exercise history. Trained data collectors administered the questionnaires to small groups of four or five students and assisted with reading or explaining questions as needed. The following data were used in this analysis: age in years; ever smoked (never, less than 10 cigarettes, 10 or more cigarettes); breakfast eating (usually eat, don't usually eat), self-reported health status (very good, good, average, poor, very poor), and total hours of physical activity per week calculated from physical activity to, from, outside and during school over the previous week.

Socioeconomic status (SES) quarters (high, medium-high, medium-low, low) were determined by postal area code of place of residence according to the Australian Bureau of Statistics Socio-Economic Index for Areas and 1981 census data (195). Scholastic level was reported by each 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. Height was measured to the nearest 0.1cm using KaWe height tape or rigid measuring tape. Weight was measured to the nearest 0.5 kg using beam or medical spring scales. BMI was calculated as weight (kg)/ (height (m))^2.

4.3.6 Covariates in adulthood

Participants were mailed questionnaires, which were returned at study clinics or by post. Collected data included age in years, marital status (married/living as married, single/separated/divorced), highest level of education (university, vocational, school), occupational status (professional, non-manual (e.g. office work), manual, not working), smoking status (never/ex, current smoker), and self-reported health status (very good, good, average, poor, very poor). Total hours of physical activity per week were measured using the validated International Physical Activity Questionnaire long form (196).

Weight was measured to the nearest 0.1kg in light clothing using Heine portable digital scales, and height was measured to the nearest 0.1cm with a Leicester stadiometer. Waist circumference was measured to the nearest 0.1cm with non-

stretch tape at the narrowest point between the lower costal border and iliac crest. BMI was calculated as above. For those who did not attend clinics, BMI was calculated from self-reported height and weight and a correction factor applied (197).

Systolic and diastolic blood pressure (mmHG) were measured from the right brachial artery using the Omron HEM907 digital automatic monitor (Omron Healthcare Co, Ltd, Kyoto, Japan), after the participant had been sitting quietly for five minutes. The mean of three readings was used. Insulin, glucose, total cholesterol, high density lipoprotein (HDL) cholesterol, and triglycerides were measured from blood samples collected after an overnight fast (169). An Olympus AU5400 automated analyser (Olympus Optical, Tokyo, Japan) was used to measure fasting insulin (mIU/l); glucose (mmol/l); total and HDL cholesterol (mmol/l) and triglycerides (mmol/l) (169). LDL cholesterol was calculated using the Friedewald equation (170). Insulin resistance was estimated using the homoeostasis model assessment (HOMA-IR) index (172). In 2009, samples from female participants that had been stored at -70°C since 2004-06, were analysed for serum folate using a chemiluminescent microparticle folate binding protein assay on an Abbott Architect analyser (Abbott, IL, US) (173).

4.3.7 Statistical analyses

Analysis was stratified by sex. To assess construct validity and distribution of DGI scores, at each time point, means and standard deviations (*SD*) and percentiles were calculated for the overall population and each sex. Also at each time point, principal component analysis (PCA) on the nine index indicators assessed underlying dimension that explain variation in DGI score (189, 190). Orthogonal varimax rotation was applied for interpretability of uncorrelated components. The number of components were selected based on visual examination of the scree plot, and eigenvalues > 1. Cronbach's coefficient α assessed internal consistency between index indicators, which were standardised due to the difference in scale for the discretionary foods indicator (20 points compared to 10 points for the other indicators) (198).

Linear regression was used to determine significance ($p < 0.05$) of cross-sectional associations at each time-point between DGI and energy and nutrient density of intakes (construct validity) and cohort characteristics (criterion validity). Multiple imputation and inverse probability weighting were used to account for missingness of data at baseline and loss to follow-up (182). Where necessary, transformations (e.g. logarithmic) of the response variable were used to remove skewness. Beta coefficients (β) and 95% confidence intervals (CI) are reported for associations between DGI and population characteristics, adjusted for all other characteristics relevant to each time-point to minimise potential confounding. Among youth, the covariates were age, SES quarter, scholastic level, breakfast eating, smoking, self-reported health, BMI, physical activity, and total energy intake. Among adults, the covariates were age, education level, occupational status, marital status, smoking, self-reported health, BMI and physical activity. Correlation coefficients (*r*) and level of significance are reported for the associations between DGI and nutrient density of intakes among youth, and cardio-metabolic risk factors among adults.

Analysis was performed with Stata Version 15.0 (StataCorp, College Station, Texas, 2017).

4.4 Results

4.4.1 Participants

Of the 5,589 10-15 year-old ASHFS participants, *N* = 5,043 (90.2%) completed the NDSS 24-hour food record. Reasons for non-completion included refusal to participate, absence from school on the day of food record distribution or collection, or did not return the food record. No participants were excluded based on reported energy intake due to the variation that could occur during a single 24-hour period.

In adulthood 2,868 participants returned the CDAH dietary questionnaires, however 82 females were excluded due to pregnancy and a further 97 participants were excluded due to missing > 10% of FFQ item responses or key responses in the FHQ e.g. type of milk usually consumed. DGI was calculated for the remaining *N* = 2,689 participants. Of the adults with a DGI score, *n* = 2,135 attended CDAH clinics for physical and fasting blood measurements.

Participants missing covariate measures were excluded from the population characteristics linear regression analyses (youth: *n* = 508; adulthood: *n* = 275). Therefore, the samples used for the linear regression of DGI scores with population characteristics was *n* = 4,535 in youth, and *n* = 2,414 in adulthood.

4.4.2 Construct validity

The evaluation of associations with score distribution, nutrient densities (youth only), PCA and Cronbach's coefficient analysis was performed on the full DGI sample (youth, *n* = 5,043; adulthood, *n* = 2,689).

DGI was normally distributed at both time points, although with a slight right skew, more apparent in youth (youth skewness: 0.43; adult skewness: 0.20). DGI means and percentiles are shown in Table 4.2. Among youth, the mean (standard deviation(*SD*)) DGI was 43.9(11.9) for females and 45.3(12.2) for males. Among adults, the mean(*SD)* DGI was 58.5(11.3) for females and 51.3(11.0) for males.

Table 4.2 Dietary Guideline Index means and percentiles for the youth (1985) and adult (2004-06) populations

			Percentile[b]								
DGI	*N*	Mean(*SD*)[a]	1st	5th	10th	25th	50th	75th	90th	95th	99th
Youth	5043	44.6 (12.0)	19.9	26.2	30.2	36.3	43.6	51.6	61.0	66.9	75.9
Female	2507	43.9(11.9)	19.2	26.3	30.0	35.8	42.6	50.5	60.3	66.4	75.8
Male	2536	45.3(12.2)	20.9	26.1	30.9	36.9	44.3	52.9	61.7	67.3	76.7
Adulthood	2689	55.2(11.7)	29.7	36.5	40.3	47.0	54.8	62.8	69.9	75.6	85.7
Female	1462	58.5(11.3)	34.3	40.3	44.1	50.8	58.4	65.6	72.9	78.8	87.0
Male	1227	51.3(11.0)	27.4	34.4	37.7	43.7	50.8	58.0	65.7	70.0	78.7

[a] Means(standard deviation) of Dietary Guidelines Index (DGI) score with possible range 0-100.

[b] Percentiles of the Dietary Guidelines Index (DGI) score with possible range 0-100.

Regression correlation coefficients between youth DGI and nutrient density intakes are reported in Table 4.3. Total energy density (total kJ/total grams of food), sugar, total fat, saturated fat, monounsaturated fat and carbohydrate intake densities were negatively correlated with DGI, while protein, fibre, polyunsaturated fat and cholesterol were positively correlated. All measured micronutrients, except retinol among females, were positively correlated with DGI.

Table 4.3 Correlations between Dietary Guideline Index scores and daily nutrient density intakes among youth

	Male (*n* = 2536)	Female (*n* = 2507)
	r[a]	*r*[a]
Total energy density (kJ/g)	–0.346***	–0.332***
Nutrient densities		
Carbohydrates (g/MJ)	–0.042*	–0.079***
Sugars (g/MJ)	–0.062**	–0.061**
Starch (g/MJ)	0.026	–0.014
Fibre (g/MJ)	0.417***	0.403***
Protein (g/MJ)	0.366***	0.404***
Total fats (g/MJ)	–0.150***	–0.142***
Saturated fat (g/MJ)	–0.260***	–0.241***
Monounsaturated fat (g/MJ)	–0.161***	–0.169***
Polyunsaturated fat (g/MJ)	0.140***	0.125***
Cholesterol (mg/MJ)	0.146***	0.124**
Vitamin C (mg/MJ)	0.171***	0.122***
Calcium (mg/MJ)	0.179***	0.229***
Iron (mg/MJ)	0.354***	0.355***
Zinc (mg/MJ)	0.320***	0.349***
Thiamin (mg/MJ)	0.188*	0.266***
Riboflavin (mg/MJ)	0.186***	0.241***
Niacin (mg/MJ)	0.237***	0.236***
Magnesium (mg/MJ)	0.421***	0.453***
Beta–carotene (μg/MJ)	0.252***	0.244***
Retinol (μg/MJ)	0.039*	0.064

*$p < 0.05$ **$p < 0.01$ ***$p < 0.001$

[a] Correlation coefficient from univariate linear regression.

The PCA indicated there were four components underlying the youth DGI, cumulatively explaining 61.2% of the variation, while in adulthood there were three components cumulatively explaining 53.6% of the variation. The eigenvalues,

variance explained, and factor loadings > |2| for these components are shown in Table 4.4. The Cronbach's coefficient α was 0.47 for youth and 0.68 for adults.

Table 4.4 Results of Principal Component Analysis on the Youth (1985) and Adult (2004-06) Dietary Guidelines Index scores

Dietary Guidelines Index (DGI) indicator	Youth (*n* = 5043) principal components[a]				Adult (*n* = 2689) principal components[a]		
	1	2	3	4	1	2	3
Dietary variety	0.63[b]				0.53		–0.25
Vegetables	0.52		–0.26		0.38		0.26
Fruit	0.31			–0.51	0.43		
Grains and cereals			0.72		0.38		
Lean meats/alternatives	0.48				0.33	0.29	
Dairy/alternatives		–0.60	0.44			0.53	
Drink plenty of water				0.77	0.34	–0.21	0.24
Limit discretionary foods		0.34	0.37	-0.20			0.87
Limit saturated fat		0.69				0.76	
Eigenvalue[c]	2.02	1.35	1.09	1.05	2.69	1.12	1.01
Variance explained (%)[d]	21.0	14.7	13.5	12.0	27.8	13.9	12.0

[a] Only components with eigenvalues > 1 were extracted. The principal components are linear combinations of the nine DGI indicator variables that explain the most variance in the data.
[b] Only factor loadings > |0.2| are shown to highlight the strongest associations between the indicator variable and principal component.
[c] An eigenvalue > 1 indicates that that the component explains at least as much variance as a single indicator variable.
[d] Percentage of common variance explained by the corresponding component.

4.4.3 Concurrent criterion validity

Associations between DGI and sociodemographic, lifestyle, and weight status characteristics, adjusted for all other reported variables are shown for youth (1985) in Table 4.5. In the adjusted analyses, significant linear trends were observed between lower DGI and lower SES, lower scholastic achievement and smoking status among both males and females. A lower DGI was also associated with being younger, lower self-reported health and not usually eating breakfast among males, and slightly fewer hours of weekly physical activity among females. There was no association between DGI and BMI or total energy intake.

Fully adjusted associations, adjusted for all other reported variables, between DGI and population characteristics are reported for adults (2004-06) in Table 4.6. Linear trends were observed between lower DGI and lower level of education and self-reported health. A lower DGI was associated with smoking among females but not males. There was no significant association between DGI and age group, marital status, or weekly physical activity for either sex. In the unadjusted analysis, a lower DGI was associated with lower occupational status (linear trend males: β=-1.43, 95% CI: -2.13, -0.72; females: β= -1.14, 95% CI: -1.71, -0.57). This association was attenuated in the adjusted analysis and the only significant difference remaining was a lower DGI score among males employed in manual work compared with those in professional roles (Table 4.6). Among females, a higher DGI was associated with being in the overweight compared to non-overweight BMI category.

Table 4.5 Youth participant population characteristics (1985) and multivariate associations with Dietary Guidelines Index scores

	Male			Female		
Variable	*n*	Mean(*SD*)[a]	β[b] (95% CI)	*n*	Mean(*SD*)[a]	β[b] (95% CI)
Age						
10-11 years	797	45.4(11.5)	Reference	818	45.0(12.0)	Reference
12-13 years	747	43.3(11.8)	-1.93 (-3.16, -0.70)*	780	42.8(11.5)	-2.04 (-3.27, -0.80)*
14-15 years	720	46.7(12.7)	1.97 (0.52, 3.42)*	673	43.7(12.0)	-0.63 (-2.15, 0.89)
Linear trend			$p = 0.105$			$p = 0.156$
Socioeconomic status[c]						
High	519	46.4(12.2)	Reference	569	45.1(12.1)	Reference
Medium high	640	44.4(11.7)	–2.15 (–3.56, –0.75)*	655	43.0(11.8)	–1.77 (-3.16, –0.38)*
Medium low	891	45.7(12.2)	–1.16 (-2.51, 0.19)	863	44.1(11.5)	–0.41 (-1.75, 0.92)
Low	214	41.7(11.9)	–4.69 (–6.65, –2.74)*	184	41.5(12.0)	–3.75 (–5.91, –1.58)*
Linear trend			$p = 0.001$			$p = 0.048$
Body mass index[d]						
Not overweight	2005	45.2(12.1)	Reference	2011	43.9(11.9)	Reference
Overweight	214	44.8(11.5)	0.55 (-1.10, 2.20)	228	43.3(11.2)	0.50 (-1.22, 2.22)
Obese	45	41.4(13.5)	-2.71 (-6.91, 1.49)	32	45.1(13.4)	3.08 (-2.39, 8.54)
Linear trend			$p = 0.550$			$p = 0.233$
Scholastic level						
Excellent	153	48.3(11.4)	Reference	280	46.4(11.6)	Reference
Above average	556	46.9(12.3)	–1.19 (–3.25, 0.88)	730	44.7(11.8)	–1.26 (–2.97, 0.45)
Average	1015	44.5(11.8)	–2.83 (–4.78, –0.89)*	874	43.3(11.8)	–2.39 (–4.09, –0.69)*
Below average	416	43.2(12.1)	–3.80 (–6.00, –1.59)*	306	42.0(11.7)	–3.69 (–5.70, –1.68)*
Poor	124	44.5(13.0)	–2.94 (–5.90, 0.03)	81	40.5(12.1)	–4.58 (–7.91, –1.24)*
Linear trend			$p < 0.001$			$p < 0.001$

Usually eat breakfast						
Yes	1973	45.4(12.1)	Reference	1855	44.2(11.7)	Reference
No	291	43.0(11.6)	–2.30 (–3.76, –0.83)*	416	42.1(12.4)	–1.21 (–2.64, 0.21)
Self-reported health						
Very good	791	46.5(12.2)	Reference	787	45.0(11.7)	Reference
Good	1023	44.9(12.1)	–1.17 (–2.32, –0.01)*	974	43.5(11.8)	–0.95 (–2.13, 0.23)
Average	412	43.4(11.7)	–2.11 (–3.58, –0.63)*	483	42.6(12.0)	–1.43 (–2.88, 0.01)
Poor	31	38.3(9.4)	–5.13 (–9.60, –0.66)*	23	41.4(11.0)	–0.04 (–5.44, 5.36)
Very poor	7	44.2(15.6)	–2.54 (–11.26, 6.18)	4	46.7(10.6)	1.61 (–8.98, 12.21)
Linear trend			*p* = 0.001			*p* = 0.070
Ever smoked						
Never	1110	45.8(12.3)	Reference	1228	44.7(11.8)	Reference
< 10 cigarettes	793	44.6(11.4)	–1.11 (–2.22, 0.00)	706	43.1(11.6)	–1.38 (–2.58, –0.19)*
≥ 10 cigarettes	361	44.0(12.7)	–2.39 (–4.03, –0.75)*	337	42.2(12.1)	–2.29 (–4.05, –0.53)*
Linear trend			*p* = 0.003			*p* = 0.003
Physical Activity - hours per week						
Lowest third	634	44.6(12.1)	Reference	812	43.6(11.8)	Reference
Middle third	752	45.2(12.1)	0.17 (-1.13, 1.48)	782	43.2(11.6)	-0.52 (-1.77, 0.73)
Highest third	878	45.4(12.1)	-0.03 (-1.31, 1.24)	677	44.9(12.1)	1.24 (-0.08, 2.55)
Linear trend			*p* = 0.947			*p* = 0.091
Total Energy (Mj)						
Lowest third	519	45.0(14.0)	Reference	987	44.3(13.3)	Reference
Middle third	705	44.8(11.9)	0.10 (-1.42, 1.61)	815	43.1(11.0)	-1.07 (-2.27, 0.13)
Highest third	1040	45.4(11.1)	-0.25 (-1.67, 1.17)	469	44.2(9.6)	0.39 (-0.89, 1.68)
Linear trend			*p* = 0.693			*p* = 0.952

*$p < 0.05$.

[a] Unadjusted mean(*SD)* of DGI score with possible range 0-100.

[b] β: the difference in Dietary Guidelines Index calculated from linear regression of the index score as the outcome against the variable characteristic as the predictor, adjusted for all other variables in the table.

[c] Area-level socioeconomic status.

[d] BMI calculated as kg/m^2. Age- and sex-specific cut points determined by Cole et al (2000) were used to classify youth as being not overweight, overweight or obese.

Table 4.6 Adult participant population characteristics (2004-06) and multivariate associations with Dietary Guidelines Index scores

	Male			Female		
Variable	*n*	Mean(*SD*)[a]	β[b] (95% CI)	*n*	Mean(*SD*)[a]	β[b] (95% CI)
Age						
26-31 years	571	51.6(11.1)	Reference	740	58.2(10.8)	Reference
32-36 years	516	50.9(10.8)	-0.38 (-1.76, 1.00)	587	58.7(11.7)	0.86 (-0.40, 2.13)
Highest education						
University	425	54.7(10.9)	Reference	614	61.2(10.2)	Reference
Vocational	384	49.8(10.2)	–4.33 (–6.12, –2.55)*	335	57.8(11.4)	–3.03 (–4.66, –1.39)*
School	278	48.1(10.6)	–6.13 (–8.02, –4.25)*	378	54.5(11.3)	–6.08 (–7.76, –4.40)*
Linear trend			$p < 0.001$			$p < 0.001$
Occupation						
Professional	639	52.7(10.9)	Reference	654	60.1(10.1)	Reference
Non-manual	83	49.3(9.8)	–1.14 (–3.58, 1.30)	713	56.6(11.7)	0.41 (–1.33, 2.15)
Manual	323	48.7(10.8)	–2.00 (–3.72, –0.29)*	936	55.9(13.1)	–0.21 (–3.70, 3.27)
Not in workforce	42	54.6(10.1)	3.30 (–0.09, 6.69)	198	57.3(11.9)	0.14 (–1.69, 1.98)
Linear trend			$p = 0.304$			$p = 0.962$
Marital status						
Living as single	348	51.9(11.7)	Reference	401	59.0(11.4)	Reference
Living as married	739	51.0(10.6)	–0.76 (–2.28, 0.76)	926	58.2(11.1)	–1.12 (–2.51, 0.27)
Self-reported health						
Very good	182	54.7(10.7)	Reference	213	63.1(10.0)	Reference
Good	436	51.7(10.9)	–2.29 (–4.28, –0.31)*	576	59.1(10.5)	–3.69 (–5.39, –1.98)*
Average	382	49.9(10.6)	–3.58 (–5.69, –1.46)*	445	56.6(11.6)	–5.58 (–7.47, –3.70)*
Poor	74	48.3(10.5)	–5.50 (–8.71, –2.28)*	81	53.3(11.6)	–7.80 (–11.09, –4.52)*
Very poor	13	48.7(13.8)	–5.12 (–10.9, 0.66)	12	49.0(13.7)	–12.69 (–21.11, –4.26)*
Linear trend			$p < 0.001$			$p < 0.001$

Smoking status						
Never/Ex	826	51.9(10.9)	Reference	1038	59.7(11.0)	Reference
Current smoker	261	49.5(10.7)	–0.71 (–2.42, 1.00)	289	53.9(10.7)	–3.37 (–4.91, –1.84)*
Body mass index (kg/m^2)						
Not overweight (BMI < 25)	434	52.2(10.9)	Reference	818	58.7(11.2)	Reference
Overweight (25 ≤ BMI < 30)	478	51.0(11.0)	-0.77 (-2.28, 0.74)	317	59.3(10.7)	2.03 (0.61, 3.45)*
Obese (BMI ≥ 30)	175	49.8(10.7)	-0.15 (-2.19, 1.89)	192	55.9(11.4)	-0.37 (-2.29, 1.56)
Linear trend			p = 0.669			p = 0.607
Physical Activity – total hours/week						
Lowest third	352	51.2(10.6)		456	58.4(10.9)	
Middle third	361	51.7(10.7)	1.36 (-0.33, 3.05)	435	58.3(11.1)	-0.28 (-1.70, 1.15)
Highest third	374	51.0(11.4)	1.17 (-0.60, 2.95)	436	58.6(11.6)	0.53 (-0.97, 2.02)
Linear trend			p = 0.207			p = 0.207

*p < 0.05.

[a] Mean(*SD)* of DGI score with possible range 0-100.

[b] β: the difference in Dietary Guidelines Index score calculated from linear regression of the index score as the outcome against the variable characteristic as the predictor, adjusted for all other variables in the table.

Cross-sectional linear regression results between adult DGI and cardio-metabolic risk factors are reported for CDAH clinic participants in Table 4.7. For both sexes, DGI was negatively correlated with waist circumference and fasting total cholesterol, LDL cholesterol, insulin and HOMA-IR score. Among males, DGI was also negatively correlated with diastolic blood pressure, while among females DGI was negatively correlated with triglycerides, and positively correlated with HDL cholesterol and serum folate.

Table 4.7 Correlations between Dietary Guideline Index score and cardio-metabolic risk factors among adult participants 26-36 years old

	Male		Female	
Variable	*n*	r[a]	*n*	r[a]
Waist circumference (cm)[b]	1023	–0.080*	1100	–0.079*
Blood pressure (mmHG)[b]				
Systolic	1025	–0.010	1099	–0.046
Diastolic	1025	–0.087*	1099	0.040
Fasting blood				
Triglycerides (mmol/L)	1029	–0.033	1106	–0.075*
Total cholesterol (mmol/L)	1029	–0.147***	1106	–0.097**
HDL-C[c] (mmol/L)	1029	0.046	1106	0.067*
LDL-C[d] (mmol/L)[b]	1016	–0.166***	1102	–0.118***
Insulin (mIU/L)[b]	1025	–0.095**	1096	–0.145***
Glucose (mmol/L)[b]	1027	–0.010	1105	–0.036
HOMA-IR[b,e]	1024	–0.093**	1095	–0.141***
Folate (nmol/L)[b,f]	–	–	904	0.258***

*$p < 0.05$ **$p < 0.01$ ***$p < 0.001$

[a] Correlation coefficient from univariate linear regression.

[b] Some participants were missing data for these measures, therefore the total is not 1,029 for males and 1,106 for females.

[c] High-density lipoprotein cholesterol.

[d] Low-density lipoprotein cholesterol.

[e] Homeostasis Model Assessment of Insulin Resistance index: (fasting glucose x fasting insulin)/22.5.

[f] Serum folate was only measured among females.

4.5 Discussion

This study demonstrates that retrospective application of the DGI is a valid measure of diet quality among youth and adults in this cohort. The correlations with nutrient density of intakes in youth and distribution of DGI scores affirms the hypothesis that our age- and sex-specific index reflects variation in dietary intake and importantly,

reflects higher scores for nutrient dense, not energy dense dietary intake. The DGI also appropriately discriminates between groups based on sociodemographic and lifestyle characteristics in both youth and adulthood. For example, associations between DGI and SES, smoking, scholastic level in youth, and education in adulthood, reflect known differences based on existing empirical research, particularly the "social gradient" of diet where higher diet quality is associated with indicators of higher SES (189, 191, 199). Among adults, higher diet quality was negatively correlated with several cardio-metabolic risk factors (total cholesterol, LDL cholesterol, insulin, waist circumference, triglycerides (women only), and diastolic blood pressure (men only)), reflecting health outcomes the dietary guidelines are designed to achieve (81).

Evidence of construct validity supports the use of the DGI as a diet quality measure. In both youth and adulthood, DGI was normally distributed and of a sufficient range to allow meaningful differences in scores. The PCA demonstrates that the index is multidimensional and variation in the data is explained by more than one linear combination of index indicators. Four components in youth, and three components in adulthood have eigenvalues greater than one, meaning they account for at least the same amount of variance as a single variable and therefore have structure. This suggests that variations in scores may arise from different combinations of DGI indicators. For example, among youth, one component had high loadings for the fruit, vegetables and lean meat/alternatives indicators, while another had high loadings for grains, discretionary foods, and dairy/alternatives. The Cronbach's coefficient α for internal consistency of scores was low for the youth DGI indicators, but approached the generally recognised level of adequacy (0.7) for the adult DGI indicators and was consistent with other dietary indices such as the Healthy Eating Index (189). The multidimensional aspect of diet explored through the PCA could contribute to the low α, as a participant would not necessarily score at consistent levels across all indicators due to individual dietary preferences. The particularly low α in childhood may be due to the single 24-hour food record and high amount of variation between indicators at the individual level, as well as the food context of 1985 affecting scores in some indicators (e.g. use of reduced fat milk was less common).

Characterisation of diet on nutritional quality was consistent with a previous cross-sectional study among children where a DGI based on the 2003 Australian dietary guidelines was positively associated with fibre and protein intake, and negatively associated with sugar and saturated fat intake (79). The negative correlations between DGI and carbohydrates, sugars, total fat, saturated fat, and monounsaturated fat reflects the indicators for reduced fat dairy, lean meat, and limiting discretionary foods. The slight positive correlation of DGI with cholesterol may be due to points for overall servings of dairy, meat and eggs irrespective of fat composition. The strong positive correlation between DGI and fibre intakes reflects the emphasis on high-fibre foods in the fruit, vegetables and wholegrains indicators. The inverse correlation with energy density, and lack of association between DGI and overall energy intake, indicates that the index appropriately distinguishes between energy-dense and nutrient-dense intake (79).

Although nutrient and energy composition were not calculated in adulthood due to the non-quantitative aspects of the FFQ, serum folate in women was associated with higher DGI. Folate is found in whole foods such as legumes and dark leafy greens, which contribute positively to the DGI. High serum folate cannot be attributed to folic acid fortification of bread flour as this became mandatory in Australia in 2009 and was not in place at the time of the 2004-06 data collection.

Means of the DGI were low in both youth and adulthood. This is consistent with previous studies that have highlighted low intakes of fruit, vegetables, and water as areas of concern, particularly among children (79, 200-203). Different food contexts in 1985 compared to 2013, may have contributed to low youth scores. However, this study evaluates quality of the reported diet as number of servings deemed appropriate for good health, not cognisant adherence to guidelines on the part of participants. Therefore, retrospective application is no more problematic than using a Mediterranean dietary index in non-Mediterranean populations. Temporal context would influence associations with population characteristics such as SES if concepts around healthy diets had changed considerably or if there was insufficient variation in diet (e.g., if processed and convenience foods were not in use at the time). However, the adequate intake of the core food groups and limiting saturated fat and

added sugars were key components of the 1982 Dietary Guidelines for Australians (204). We would therefore expect that groups previously demonstrated to exhibit healthier diets of wider variety and whole foods (e.g. higher SES, non-smokers, breakfast eaters), would have higher DGI, as we saw in our analysis.

Our observed associations with population characteristics showed good agreement with previous studies. In cross-sectional analyses among children, the 2003 DGI for Children and Adolescents was positively associated with markers of SES (79), while poorer overall diet quality has been associated with poorer academic achievement (205), and among adolescents, smoking has been cross-sectionally associated with unhealthy dietary habits (206, 207). Among adults, higher index scores arising from the 2003 and 2013 versions of the DGI were associated with higher education and SES (208), higher health-related quality of life (209), not smoking, and lower obesity and cardio-metabolic risk (76, 78, 210, 211). In our study, the social gradient of diet was reflected in the strong associations between DGI and education level, with adults who had school as their highest education level having a DGI score ~6 points lower than university educated adults. Correlations between higher DGI and lower waist circumference and lower diastolic blood pressure (males only), was consistent with prior applications of Australian DGIs (210, 211). In adulthood, women had a significantly higher mean score than men, which accords with other Australian and international dietary guideline studies (76, 187, 189, 211, 212). This could be influenced by gender differences in education, occupation, social factors and health consciousness. In this particular cohort, women have been shown to be more likely to meet healthy lifestyle guidelines than men (168).

Limitations of this study were as follows. Firstly, the methods of dietary measurement may have introduced bias. Dietary data from free-living individuals in the community is often biased, mainly towards under-reporting of intakes (213). Also, the FFQ used in adulthood only collected data on the frequency of consumption, not serving sizes. The difference in mean scores by sex may reflect women having greater dietary variety or instances of eating partial servings of foods, as opposed to more standard servings. However, frequency alone has been shown to explain the major variance in food intake, and having participants estimate

portion sizes can be problematic and introduce measurement error (214). In youth, only a single 24-hour food record was taken, whereas repeat or multi-day food records are the preferred approach (215), as they may be more reflective of usual diet and strengthen discrimination between group characteristics. Instead, as with the premise of the original "snapshot" study, we have relied on the large sample size to represent average intakes in the population. For the purposes of assessing construct validity of the index using the nutritional composition of the food and beverage items actually consumed, the data is fit for purpose.

A second limitation was that although the sample was nationally representative of Australian schoolchildren at baseline, the large loss to follow-up may have had some effect on the associations with population characteristics among adults in the concurrent criterion validity analysis. However, despite the adult sample being of higher SES than the general population, our sample remained diverse with a range of characteristics. Bias was partially mitigated with inverse probability weighting. Furthermore, as those lost to follow-up are more likely to have lower SES (216), observed associations may be more conservative than if the full cohort was included.

A possible third limitation was the structure of the DGI and point allocation to each of the indicators. To an extent, this was an arbitrary process, with a maximum of 10 points allocated to each indicator apart from the discretionary foods indicators which had a maximum of 20 points. It may be that some food groups or dietary practices warrant higher weighting than others, but this would require further extensive empirical research and sensitivity analyses which is beyond the scope of this study.

Our study also had several strengths. Although the differing dietary measurement methods used in youth and adulthood may limit inferences of diet quality over time for this particular cohort, our results indicate that the DGI is adaptable to different dietary data and collection methods. Another strength is validation of a food-based index that captures important nutrient and non-nutrient qualities of diet, which may have synergistic health effects (185). The study sample is quite large, and the unique dataset includes a range of sociodemographic, lifestyle, and objective physical

measures, facilitating evaluation of the DGI in the same cohort of individuals in two distinct life phases. To our knowledge, this is the first study to assess the appropriateness of a uniformly structured diet quality index for use among children, adolescents and adults.

4.6 Conclusion

In conclusion, this study provides evidence that our DGI, aligned to age- and sex-specific dietary guidelines, is a nuanced and appropriate measure of diet quality in youth and adulthood as higher scores reflect nutrient-dense, rather than energy-dense intake, and discriminate between population characteristics consistent with the literature. Furthermore, our results indicate that retrospective application of the DGI to our data collected prior to the release of the current Australian Dietary Guidelines is appropriate, with the DGI reflecting variations in the cohort's diet according to current evidence-based understanding of diet quality. The DGI provides an interpretable measure of overall diet with which to assess associated factors over time. Further research using dietary indices in cohorts from youth into adulthood is needed, particularly longitudinal studies using consistent and repeat dietary measurement methods at each follow-up. This would help determine associations between diet quality, sociodemographic factors, and health outcomes over the life course, and work to support and inform future dietary guidelines.

4.7 Postscript

This study provides an important tool for researchers in the validated age- and sex-specific dietary guidelines index to assess diet quality among children, adolescents, and adults. In the following two chapters, the index is used as the predictor variable in analyses to examine mood disorder outcomes among the CDAH cohort.

Chapter 5: Youth diet quality and hazard of mood disorder in adolescence and adulthood among an Australian cohort

5.1 Preface

The literature review in Chapter 2 highlighted that there are a limited number of prospective studies on diet quality in youth and mood disorder outcomes and no studies that extend follow-up beyond late adolescence. This study uses the Dietary Guidelines Index developed in Chapter 4 to determine diet quality of CDAH participants in youth, and then examine mood disorder outcomes during a 25-year follow-up period. The text from this chapter was accepted and published in the Journal of Affective Disorders in July 2020 (217).

5.2 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 (31). 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 (1), 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 (72, 113, 114, 141), and to a limited extent among children and adolescents (97, 98), 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 (47, 218).

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 diverse mental or behavioural disorders under broad categories rather than formal diagnoses (97, 98). For example, outcomes of interest in prior studies have included emotional functioning, internalising problems (e.g. depression, anxiety), depressive symptoms (106) and psychological distress (99, 102). 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 (100, 104). 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 (100, 102, 104, 108). 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 (100-102). 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 (219). 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 (96).

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.

5.3 Methods

5.3.1 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* = 8,498 out of 12,578 students approached) [16]. ASHFS participants aged 10-15 years (*N* = 5,589) 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 5,170 participants enrolling in the CDAH study (61.0%) (162), including 3,188 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), 1,144 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.

5.3.2 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 (192).

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) (183) that reflected the age- and sex-specific recommended food group servings in the 2013 Australian Dietary Guidelines (81). 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 (220). 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)) (183). A higher score on the range of 0-100 indicates better alignment with the 2013 Australian Dietary Guidelines and therefore better diet quality.

5.3.3 Mood disorder measure

Mental health was assessed at CDAH-2 using the lifetime version of the Composite International Diagnostic Interview (CIDI) (167). 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 (9) 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.

5.3.4 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 (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 (221), 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 (195). School type (state, Catholic, or independent) was used as an additional indicator of SES. State 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 weight (kg)/(height (m))^2. Cole's age- and sex-specific cut points were used to classify youth as being non-overweight, overweight or obese (222).

5.3.5 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 (223). 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 (224). Violation of the proportionality assumption was determined if the tests of proportionality results were significant ($p < 0.05$), 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 (99-102).

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 (225, 226), lifestyle/behavioural factors (47, 227, 228), anthropometry (229), and youth negative effect (230). 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.

5.4 Results

At baseline, 5,043 (90.2%) of the eligible 5,589 10-15-year old ASHFS participants completed the NDSS (Figure 5.1). 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, 1,144 (22.7%) of the 5,043 NDSS participants completed the CIDI, with 291 (25.4%) of the 1,144 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* = 1,005, with 245 participants (24.4%) reporting mood disorders.

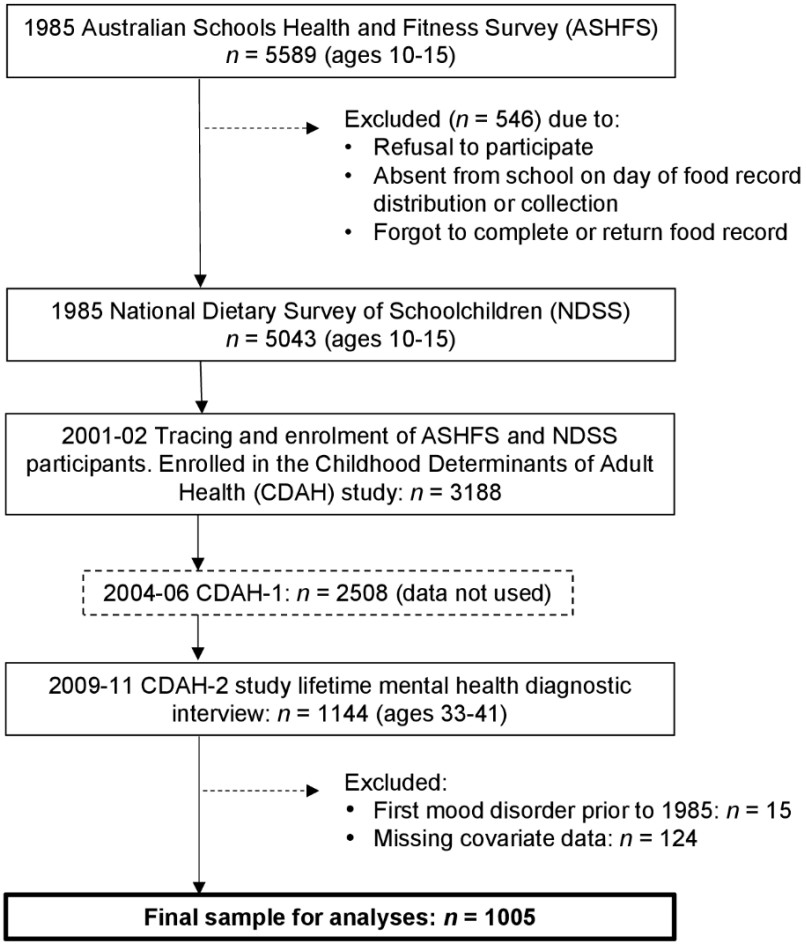


Figure 5.1 Flow diagram of the Childhood Determinants of Adult Health (CDAH) cohort participants from 1985 baseline to follow-up in 2009-11

Baseline population characteristics of both the original NDSS cohort and the final study sample are shown in Table 5.1 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 1,681 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 5,043 NDSS participants.

Table 5.1 Characteristics of participants at baseline in 1985

	Study sample (*n* = 1005)		Baseline cohort (*N* = 5589)	
Variable	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)[f]
Bradburn negative affect[b]	2.8	(1.7)	2.9	(1.7)[f]
Physical activity (hrs/wk)[c]	7.4	(6.6)	7.6	(7.1)[f]
BMI category[d]				
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				
State	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[e]				
Usually eat	86.1	(865/1005)	84.5	(4590/5435)
Don't usually eat	13.9	(140/1005)	15.6	(845/5435)

[a] Dietary Guidelines Index: higher score on range of 0-100 indicates higher diet quality.
[b] Bradburn negative affect scale, higher score on range of 0-8 indicates higher negative affect.
[c] Physical activity to, from, during, and outside of school (*n* = 1,005).
[d] BMI calculated as kg/m^2. Age- and sex-specific cut points determined by Cole et al (2000) were used to classify youth as being non-overweight, overweight or obese.
[e] Usually eat breakfast defined as eating something before school four or more days per week.
[f] DGI: *n* = 5,043; Bradburn negative affect: *n* = 5,323; Physical activity: *n* = 5,400.

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 5.2. 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 $p = 0.449$).

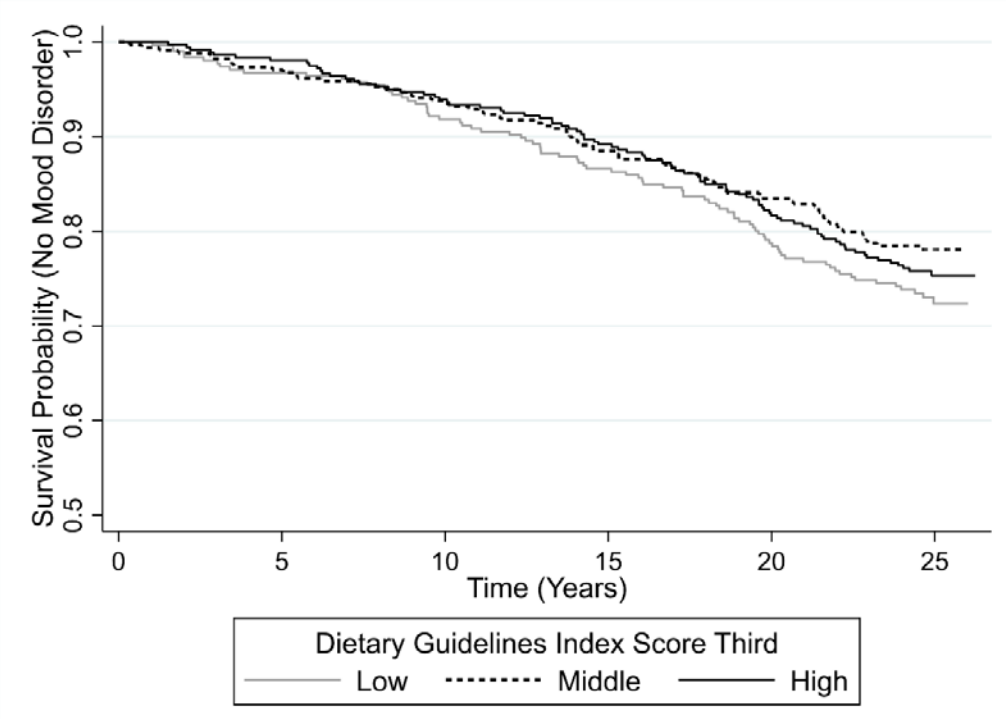


Figure 5.2 Kaplan-Meier survival estimates of not having mood disorder onset by Dietary Guidelines Index score third (Low, Middle, High)

Results of the Cox proportional hazards regression shown in Table 5.2 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).

Table 5.2 Hazard ratios and 95% confidence intervals of mood disorder 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[a]			Model 2[b]		
		HR	(95% CI)	*P*	HR	(95% CI)	*P*
DGI[c]	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

Mood disorder: first onset of dysthymia or major depressive disorder; HR: hazard ratio; CI: confidence interval.
[a] Adjusted for sex and age.
[b] Allowing 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.
[c] Continuous 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.

The sensitivity analysis results of relative risk estimates to examine cumulative risk during the periods proximal to baseline, are shown in Table 5.3. 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).

Table 5.3 Log binomial regression risk ratios and 95% confidence intervals of mood disorder 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 (*n*/*N*)	Model 1[a]		Model 2[b]	
		RR (95% CI)	*P*	RR (95% CI)	*P*
To 5 years after baseline (1990)					
DGI[c]	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	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	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	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

Mood disorder: first onset of dysthymia or major depressive disorder; RR: risk ratio; CI: confidence interval.

[a] Adjusted for sex and age.

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

[c] 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.

5.5 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 (191) and socioeconomic factors in youth have been shown to persist into adulthood and be associated with depressive symptoms in adulthood (225, 231). 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 (232). 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 (81). 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 (99-101, 106). 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 (102). 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 (97, 98). 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 (72, 113, 114). 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 (233, 234). 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 (235). 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 (129). 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 schoolchildren's 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 5.1. 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% (14), 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 (87).

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 (89). 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 (72, 113-115).

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 (18). Furthermore, the DGI is based on core recommendations that are nearly universal among dietary guidelines internationally (220), 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.

5.6 Conclusion

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.

5.7 Postscript

Although the research presented in this chapter has not provided evidence of associations between youth diet quality and adult mood disorders, it has highlighted the high prevalence of mood disorders among the CDAH cohort. The following

chapter examines associations between diet quality and mood disorders among the cohort during early- to mid-adulthood.

5.8 Supplementary material

Chi-square tests of association were performed on key baseline demographic variables and score thirds of the baseline Dietary Guidelines Index (DGI) (Table 5.4). The table is not included in the published version but is included here as further descriptive statistics of the study sample.

Table 5.4. Distribution of study sample (*n* = 1005) by Dietary Guidelines Index score third

Variable	DGI Score Third			Pearson chi-squared *p*-value[a]
	Low	Middle	High	
n	306	339	360	
Sex, %				
Female	66.7	61.7	57.2	0.044
Male	33.3	38.3	42.8	
Age (years), %				
10	14.4	15.3	18.6	0.042
11	14.7	20.6	17.5	
12	21.6	15.0	14.2	
13	16.3	11.8	16.9	
14	19.3	18.0	16.4	
15	13.7	19.2	16.4	
Bradburn negative affect[b], %				
No negative affect (<=4)	250	292	311	0.177
Negative affect (>4)	56	47	49	
BMI category[c], %				
Non-overweight	91.8	91.2	91.9	0.578
Overweight	6.2	8.0	7.2	
Obese	2.0	0.9	0.8	
Area-level SES, %				
High	20.6	26.3	31.9	0.002
Medium-High	30.7	29.5	22.8	
Medium-Low	38.9	38.6	40.8	
Low	9.8	5.6	4.4	
School type, %				
State	74.2	72.9	76.1	0.166
Catholic	22.2	21.5	16.9	
Independent	3.6	5.6	6.9	
Academic performance, %				
Excellent	12.1	18.9	15.3	0.011
Above average	32.7	35.7	38.3	
Average	39.9	34.5	38.3	
Below average	13.7	10.9	7.2	
Poor	1.6	0.0	0.8	
Ever smoked, %				
Never	47.1	51.9	57.8	0.009
< 10 cigarettes	35.6	37.8	31.7	
≥ 10 cigarettes	17.3	10.3	10.6	
Drink alcohol, %				
Never	61.4	61.4	73.3	0.004
Less than weekly	29.1	30.4	21.1	
Weekly or more	9.5	8.3	5.6	

Physical Activity (minutes/week)[d], %				
Lowest third	32.0	30.4	29.7	0.290
Middle third	39.2	33.6	34.7	
Highest third	28.8	36.0	35.6	
Usually eat breakfast[e], %				
Usually eat	83.3	87.6	86.9	0.245
Don't usually eat	16.7	12.4	13.1	

DGI: Dietary Guidelines Index, higher score on range of 0-100 indicates higher diet quality.
[a] *p*-value for Pearson chi-squared test of independence
[b] Bradburn negative affect scale, higher score on range of 0-8 indicates higher negative affect.
[c] BMI calculated as kg/m^2. Age- and sex-specific cut points determined by Cole et al (2000) were used to classify youth as being non-overweight, overweight or obese.
[d] Physical activity to, from, during, and outside of school.
[e] Usually eat breakfast defined as eating something before school four or more days per week.

Chapter 6: Associations between diet quality and DSM-IV mood disorders during young- to mid-adulthood among an Australian cohort

6.1 Preface

The high prevalence of mood disorders and potential negative impact this can have on an individual's health, relationships, social and work life, mean it is an important area of study. Although the previous study in Chapter 5 did not find evidence of associations among the cohort between youth diet and subsequent mood disorder onset, associations should be examined among this later life stage of early to mid-adulthood, when mental disorders comprise the highest proportional non-fatal burden of disease (13). Moreover, the Chapter 2 literature review identified that there are limited prospective studies among adults that have used clinical diagnostic criteria to determine mood disorder outcomes. The CDAH study provides an opportunity to address this gap. The text from this chapter was accepted for publication by Social Psychiatry and Psychiatric Epidemiology in April 2021 (236).

6.2 Introduction

Numerous studies have examined associations between diet and mood disorders. Recent systematic reviews consolidating evidence from both cross-sectional and longitudinal studies (114, 119), and longitudinal studies only (115, 118), 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 (114, 237).

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 (114, 115, 118, 119). 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 (83, 90). 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 (135, 140).

The Australian Childhood Determinants of Adult Health (CDAH) study (162) 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 (12). 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.

6.3 Methods

6.3.1 Participants

The study sample comprised participants of the CDAH cohort study (Figure 6.1). 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.

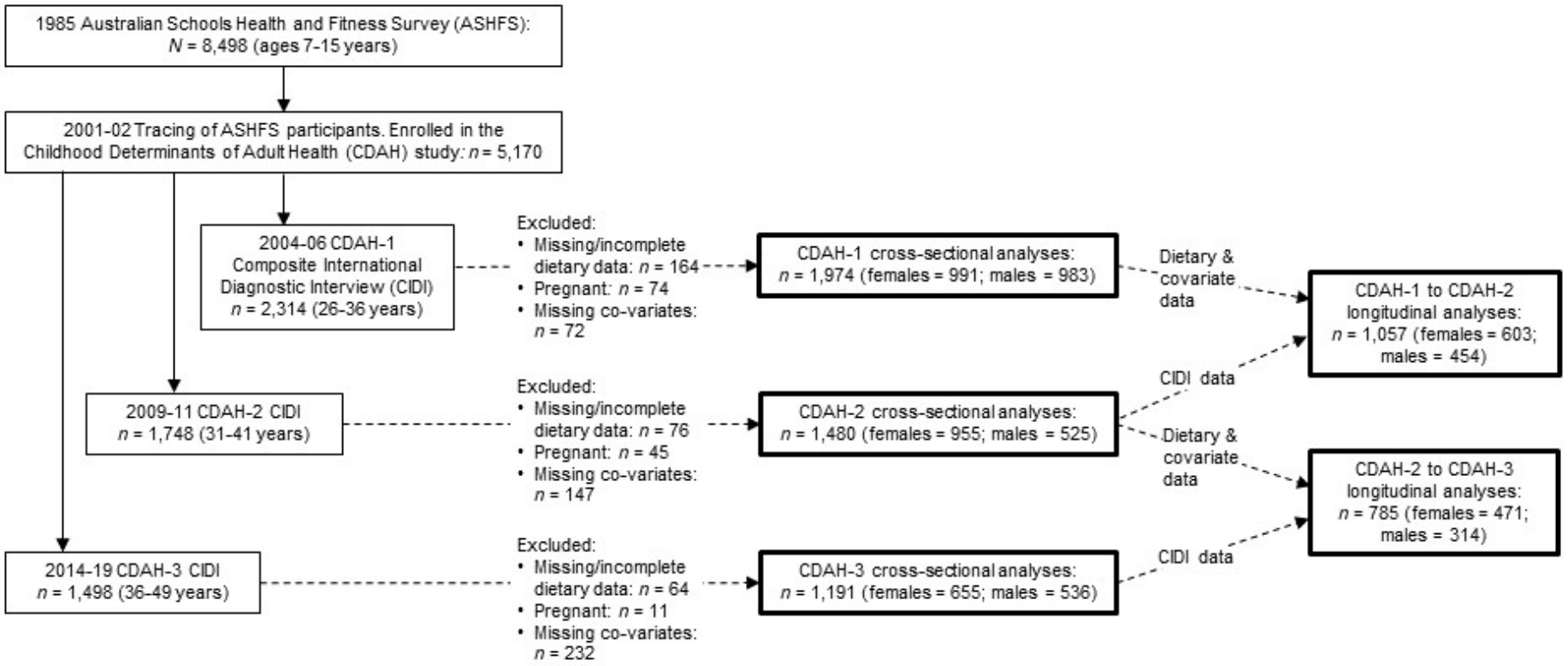


Figure 6.1 Childhood Determinants of Adult Health (CDAH) study participation flow chart

6.3.2 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.

6.3.3 Dietary Guidelines Index

Dietary data were collected using food frequency questionnaires (FFQ) and food habits questionnaires (FHQ) based on a validated FFQ designed for Australian populations (163, 164). 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 (76). 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 a100-point Dietary Guidelines Index (DGI) that reflected the nutritional recommendations in the 2013 Australian Dietary Guidelines (81, 183). 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 (183). 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 Section 6.8 Supplementary material, Table 6.5. A higher score on the range 0-100 indicated better diet quality.

6.3.4 Mood Disorder

The World Health Organization's Composite International Diagnostic Interview (CIDI-Auto) (167) was used to determine prevalence of DSM-IV dysthymic disorder and major depressive disorder (mild, moderate or severe) (9) 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 (any episode – 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 during follow-up, a second longitudinal outcome variable excluded participants who had experienced a mood disorder prior to baseline (CDAH-1 or CDAH-2 as relevant), leaving only participants who had never had a prior mood disorder. To examine reverse causality, age of most recent mood disorder from the CDAH-2 and CDAH-3 lifetime CIDIs 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.

6.3.5 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 (238). 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 (196, 239).

At CDAH-1 and CDAH-3, BMI (kg/m2) 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 (BMI < 25), overweight (25 ≤ BMI < 30), or obese (BMI ≥ 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 (168).

6.3.6 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, White (182). 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 (217). 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% (240). 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 (105) so it was not used in the model building process where mood disorder was the outcome due to the risk of over-adjustment bias (241), 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 (198), with α=0.86 at CDAH-1, and α=0.87 at both CDAH-2 and CDAH-3.

6.4 Results

The number of participants at each follow-up, participant exclusions, and final samples for analysis are detailed in Figure 6.1. 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 6.1. 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.

Table 6.1 Characteristics of the Childhood Determinants of Adult Health (CDAH) study participants at 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	
	n	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)
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
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
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
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.

Results of the cross-sectional analyses of mood disorder and DGI are shown in Table 6.2. 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 (Section 6.8 Supplementary material, Table 6.6).

Table 6.2 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.

Longitudinal analysis results are shown in Table 6.3. 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 6.4. 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).

Table 6.3 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)
First onset or recurrent mood disorder								
CDAH-1 to CDAH-2								
Females	19.1	(115/603)	1.03	(0.86, 1.24)	1.07	(0.9, 1.28)	1.07	(0.88, 1.3)
Males	13.2	(60/454)	1.06	(0.91, 1.23)	1.06	(0.87, 1.28)	1.08	(0.97, 1.2)
CDAH-2 to CDAH-3								
Females	17.2	(81/471)	1.08	(0.87, 1.33)	1.08	(0.9, 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 6.4 Linear regression of Dietary Guidelines Index score on mood disorder status at CDAH-2 (2009-11) and CDAH-3 (2014-19)

			Model 1[a]		Model 2[b]	
	%	(*n*/*N*)	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 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 (Section 6.8 Supplementary material, Table 6.7). 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) (Section 6.8 Supplementary material, Table 6.8). 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.

6.5 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 (137-139). 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 (140). 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 (115). Previous research has also highlighted the importance of controlling for time-varying covariates (115, 242). The mediation of current circumstance on mood disorders is consistent with stronger results reported from cross-sectional studies (118, 119).

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 (218), whereas a 2020 study reported that participants with mood disorders prior to (but not at) baseline had lower diet quality at follow-up (243). 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 (244), and mood disorders and a time-of-day eating pattern characterised by skipped or delayed breakfast (245).

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 (246). 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 (247, 248), friendship quality, support scales, or social activity (106, 244, 249-251). 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 behaviour change (252). There may also be a synergistic effect of self-efficacy and social support for improving and maintaining better diet quality (253). Women may have better internalised strategies for healthy eating compared to men (254), but also be more susceptible to mood disorders in

the face of low social support (255). 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 (163) and the assumption that one reported frequency of an item is equal to one serve has been used for validated diet quality measures (76, 183). 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 (225) and lower adult diet quality (256, 257), 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 some mis-identification of cases, primarily due to under-diagnosis (87), the CIDI is regarded as the 'gold standard' for epidemiological studies and suitable for retrospective measurement of mental disorders (89). 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.

6.6 Conclusion

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

6.7 Postscript

This study did not identify any longitudinal relationships between diet quality and mood disorders, among the CDAH cohort. The inclusion of diagnostic mood disorder measures and the longitudinal analysis provides important results that could potentially contribute to future meta-analyses. The study also identified that self-perceived social support may be an important confounder or mediator in the relationship. There are many inter-related aspects of diet that complicate nutritional epidemiology and the determination of diet-disease relationships. One of these aspects is timing of eating occasions, which is examined in the following chapter.

6.8 Supplementary material

6.8.1 Supplementary material submitted to the journal

The following tables were submitted to the journal as online supplementary material.

Table 6.5 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			

[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 6.6 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 6.7 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

	Model 1[a]		Model 2[b]		Model 3[c]	
Sex and DGI component	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 6.8 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)

		Outcome Measure[a]			
		Dietary Guidelines Index[b]		Mood disorder	
	n	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

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

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

6.8.2 Additional supplementary material not submitted to the journal

The following results by DGI score third category were not submitted to the journal but are included here for reference.

Distribution of participant characteristic by index score thirds

Chi-square tests of association were performed on key demographic variables and score thirds of the baseline Dietary Guidelines Index (DGI) at each follow-up. The DGI score thirds were defined by score tertiles from all participants with DGI scores at each time-point, before participants were excluded from the study sample for missing outcome or covariate measures. The DGI score tertiles used to determine the score thirds were 49.7 and 59.9 at CDAH-1 (2004-06), 52.3 and 62.0 at CDAH-2 (2009-11), and 50.4 and 59.8 at CDAH-3 (2014-19). The distribution of participant characteristics at each follow-up are outlined in the Table 6.9 (CDAH-1), Table 6.10 (CDAH-2), and Table 6.11 (CDAH-3).

Table 6.9 Distribution of characteristics of the study sample at CDAH-1 (2004-06) by Dietary Guidelines Index (DGI) score third

	Females – DGI score third				Males – DGI score third			
Variable	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]
n[b]	216	339	436		427	337	219	
Mood disorder in past 12 months, %								
No mood disorder	86.1	85.5	92.2	0.007	94.8	93.5	92.7	0.514
Mood disorder	13.9	14.5	7.8		5.2	6.5	7.3	
Age (years), %								
26-31	59.7	57.2	54.8	0.479	51.3	55.2	59.4	0.141
32-36	40.3	42.8	45.2		48.7	44.8	40.6	
BMI category, %								
Non-overweight (BMI < 25)	63.4	57.8	65.1	0.007	37.7	35.9	44.3	0.102
Overweight (25 ≤ BMI < 30)	17.1	27.7	23.2		43.6	48.1	43.8	
Obese (BMI ≥ 30)	19.4	14.5	11.7		18.7	16.0	11.9	
Smoking status, %								
Never	41.7	56.0	63.8	<0.001	54.8	55.5	63.9	0.072
Ex-smoker	23.6	23.9	22.2		18.3	17.8	18.7	
Smoker	34.7	20.1	14.0		26.9	26.7	17.4	
Social support index, %								
Lowest third	41.7	33.6	26.1	<0.001	38.4	35.9	29.2	0.109
Middle third	37.5	36.3	35.1		33.7	34.4	33.8	
Highest third	20.8	30.1	38.8		27.9	29.7	37.0	
Marital status, %								
Living as single	31.0	31.6	33.0	0.847	31.4	32.3	32.0	0.960
Living as married	69.0	68.4	67.0		68.6	67.7	68.0	

Parental status, %								
No children	39.0	48.3	52.7	0.006	53.5	65.2	68.0	0.002
Children	61.0	51.7	47.3		46.5	34.8	32.0	
Education, %								
University	28.2	48.7	58.7	<0.001	27.8	41.4	54.3	<0.001
Vocational	30.6	23.6	22.9		40.5	35.7	28.8	
School	41.2	27.7	18.3		31.8	22.9	16.9	
Occupation, %								
Manager/Professional	38.4	53.1	57.6	<0.001	49.8	61.4	71.0	<0.001
Non-manual	37.0	24.2	23.6		9.8	6.9	4.6	
Manual	6.9	4.4	2.8		38.5	27.5	20.7	
Not in workforce	17.6	18.3	16.1		1.9	4.2	3.7	
Leisure time physical activity (hrs/wk), %								
Lowest third	47.2	35.1	23.4	<0.001	41.8	33.0	18.8	<0.001
Middle third	35.2	34.5	36.2		28.4	35.3	32.5	
Highest third	17.6	30.4	40.4		29.9	31.7	48.7	

[a] *p*-value for Pearson chi-squared test of independence.

[b] *n* for each score third of all variables except for the following variables that were not used in the fully adjusted regression analysis models after purposeful model building, and therefore were not used to exclude participants due to missing data: parental status (females - low: 205, middle: 321, high: 406; males – low: 314, middle: 244, high: 169), and among males only: occupation (low: 418, middle: 334, high: 217), leisure time physical activity (low: 395, middle: 309, high: 197).

Table 6.10 Distribution of characteristics of the study sample at CDAH-2 (2009-11) by Dietary Guidelines Index (DGI) score third

	Females – DGI score third				Males – DGI score third			
Variable	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]
n[b]	245	335	375		233	172	120	
Mood disorder in past 12 months, %								
No mood disorder	89.4	91.0	91.7	0.607	93.6	92.4	94.2	0.830
Mood disorder	10.6	9.0	8.3		6.4	7.6	5.8	
Age (years), %								
31-36	49.0	55.5	59.5	0.037	52.8	45.3	51.7	0.310
37-41	51.0	44.5	40.5		47.2	54.7	48.3	
BMI category, %								
Non-overweight (BMI < 25)	55.9	54.0	65.1	0.021	33.9	40.7	40.8	0.052
Overweight (25 ≤ BMI < 30)	23.7	27.5	21.3		45.9	43.6	50.8	
Obese (BMI ≥ 30)	20.4	18.5	13.6		20.2	15.7	8.3	
Smoking status, %								
Never	50.0	60.6	61.3	<0.001	58.4	64.0	70.8	0.002
Ex-smoker	29.1	29.3	29.3		19.3	25.6	20.0	
Smoker	20.9	10.1	9.3		22.3	10.5	9.2	
Social support index, %								
Lowest third	41.2	32.8	29.6	0.057	39.9	45.9	40.0	0.326
Middle third	30.6	34.9	37.1		35.6	29.1	28.3	
Highest third	28.2	32.2	33.3		24.5	25.0	31.7	
Marital status, %								
Living as single	18.8	16.4	21.3	0.248	17.6	14.5	12.5	0.420
Living as married	81.2	83.6	78.7		82.4	85.5	87.5	

Parental status, %								
No children	22.3	25.3	31.5	0.031	31.3	34.3	35.0	0.727
Children	77.7	74.7	68.5		68.7	65.7	65.0	
Education, %								
University	33.9	54.3	62.7	<0.001	38.6	46.5	68.3	<0.001
Vocational	31.4	27.2	22.1		37.8	40.1	24.2	
School	34.7	18.5	15.2		23.6	13.4	7.5	
Occupation, %								
Manager/Professional	38.8	51.3	57.9	0.001	66.1	68.0	79.2	0.159
Non-manual	31.0	25.1	20.8		7.3	4.1	5.0	
Manual	8.2	5.7	3.7		23.6	23.8	13.3	
Not in workforce	22.0	17.9	17.6		3.0	4.1	2.5	
Leisure physical activity (hrs/wk)[c], %								
Lowest third	47.8	31.3	21.8	<0.001	41.2	31.4	20.8	0.002
Middle third	29.0	36.4	32.5		31.3	34.3	35.8	
Highest third	23.2	32.3	45.7		27.5	34.3	43.3	
Usual Sleep hours								
≤7.5 hours	47.3	49.7	46.0	0.612	61.4	73.8	65.0	0.030
> 7.5 hours	52.7	50.3	54.0		38.6	26.2	35.0	

[a] *p*-value for Pearson chi-squared test of independence.

[b] *n* for each score third of all variables except for the following variables for females that were not used in the fully adjusted regression analysis models after purposeful model building, and therefore were not used to exclude participants due to missing data: parental status (low: 242, middle: 332, high: 365), leisure time physical activity (low: 224, middle: 316, high: 357), usual sleep hours (low: 241, middle: 332, high: 374).

Table 6.11 Distribution of characteristics of the study sample at CDAH-3 (2014-19) by Dietary Guidelines Index score third

	Females – DGI score third				Males – DGI score third			
Variable	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]
n[b]	148	232	275		230	166	140	
Mood disorder in past 12 months, %								
No mood disorder	81.8	90.1	91.3	0.009	90.4	93.4	95.7	0.154
Mood disorder	18.2	9.9	8.7		9.6	6.6	4.3	
Age (years), %								
36-43	51.4	49.6	47.3	0.710	47.4	42.2	53.6	0.138
44-49	48.6	50.4	52.7		52.6	57.8	46.4	
BMI category, %								
Non-overweight (BMI < 25)	28.4	44.4	56.0	<0.001	25.7	33.7	40.0	0.007
Overweight (25 ≤ BMI < 30)	35.8	31.0	28.4		45.7	41.0	45.7	
Obese (BMI ≥ 30)	35.8	24.6	15.6		28.7	25.3	14.3	
Smoking status, %								
Never	54.7	62.9	68.7	0.007	60.4	66.9	68.6	0.094
Ex-smoker	31.8	31.0	25.8		24.8	22.3	25.7	
Smoker	13.5	6.0	5.5		14.8	10.8	5.7	
Social support index, %								
Lowest third	43.2	30.2	25.5	0.004	44.3	34.9	33.6	0.219
Middle third	25.7	30.6	29.8		31.7	36.1	36.4	
Highest third	31.1	39.2	44.7		23.9	28.9	30.0	
Marital status, %								
Living as single	20.9	19.4	18.2	0.787	17.0	13.3	15.7	0.601
Living as married	79.1	80.6	81.8		83.0	86.7	84.3	

Parental status, %								
No children	18.2	20.7	15.6	0.336	16.2	16.3	17.3	0.962
Children	81.8	79.3	84.4		83.8	83.7	82.7	
Education, %								
University	44.6	57.3	67.3	<0.001	42.2	56.0	66.4	<0.001
Vocational	39.2	31.9	22.2		41.3	30.7	27.1	
School	16.2	10.8	10.5		16.5	13.3	6.4	
Occupation, %								
Manager/Professional	53.4	55.6	64.4	0.093	65.7	75.3	80.0	0.060
Non-manual	29.7	28.0	20.7		6.5	4.8	6.4	
Manual	5.4	3.9	1.8		23.5	16.3	10.7	
Not in workforce	11.5	12.5	13.1		4.3	3.6	2.9	
Leisure physical activity (hrs/wk)[c], %								
Lowest third	52.7	34.5	27.3	<0.001	43.5	33.7	18.6	<0.001
Middle third	27.7	34.1	30.2		27.8	26.5	37.9	
Highest third	19.6	31.5	42.5		28.7	39.8	43.6	
Usual Sleep hours								
≤7.5 hours	73.6	68.1	69.1	0.491	75.2	77.1	87.1	0.019
> 7.5 hours	26.4	31.9	30.9		24.8	22.9	12.9	

[a] *p*-value for Pearson chi-squared test of independence.

[b] *n* for each score third of all variables except for parental status among males (low: 228, middle: 166, high: 139), which was not used in the fully adjusted regression analysis models after purposeful model building, and therefore was not used to exclude participants due to missing data.

Regression results by index score thirds

The cross-sectional regression results by DGI score third category are presented in Table 6.12 for additional illustration of the associations. Although there is a statistically significant association ($p < 0.05$) for higher prevalence of mood disorder associated with high compared to low DGI score among males at CDAH-1 following covariate adjustment, the number of comparisons, the low number of prevalent cases, and the wide confidence interval indicates that the results should be interpreted with caution. Furthermore, the opposite effect is observed among males in the high compared to low DGI score categories at CDAH-2 and CDAH-3. For interpretability, the results were presented in the primary analysis by continuous DGI score (10-point increase) rather than score third, due to the differing tertiles used to categorise scores at each time-point.

Additional longitudinal analysis using generalised estimating equations (GEE) was also performed to calculate the population averaged effects for first onset mood disorder for participants from the two follow-up periods CDAH-1 to CDAH-2 and CDAH-2 to CDAH-3 (Table 6.13). Only first onset mood disorder (excluding all participants with reported onset of a mood disorder prior to or at the baseline follow-up), was performed as recurring mood disorder episodes were not independent of each other. As with the primary results presented in Table 6.3, there were no significant longitudinal associations between diet quality and risk of mood disorder onset during follow-up.

Multiple imputation and inverse probability weighting, as described in the methods in Section 6.3.6 was applied to the cross-sectional log binomial regression analyses. The GEE analysis was performed in Stata 16.1 (StataCorp, College Station, Texas, 2017) using a binomial family and log link. Multiple imputation and inverse probability weighting were not applied to the longitudinal GEE analysis due to current limitations of the combined imputation and weighting program to apply these within GEE models, and as the analyses by follow-up period for any mood disorder and first onset disorder only, shown in Table 6.3 was developed and presented in the submitted paper instead.

Table 6.12 Prevalence ratios of mood disorder within 12 months prior to follow-up for middle and high compared to low Dietary Guidelines Index (DGI) score third among participants at Childhood Determinants of Adult Health (CDAH) study follow-ups at CDAH-1 (2004-06

	% with mood disorder	(*n*/*N*)	Model 1[a]		Model 2[b]		Model 3[c]		Model 4[d]	
			PR	(95% CI)	PR	(95% CI)	PR	(95% CI)	PR	(95% CI)
CDAH-1 DGI score third										
Females										
Low	13.9	(30/216)	1.00		1.00		1.00		1.00	
Middle	14.5	(49/339)	1.12	(0.72, 1.74)	1.24	(0.80, 1.93)	1.13	(0.74, 1.72)	1.09	(0.71, 1.67)
High	7.8	(34/436)	0.59	(0.36, 0.96)*	0.75	(0.43, 1.29)	0.78	(0.47, 1.29)	0.75	(0.44, 1.30)
Males										
Low	5.2	(22/427)	1.00		1.00		1.00		1.00	
Middle	6.5	(22/337)	1.16	(0.64, 2.08)	1.16	(0.65, 2.08)	1.36	(0.77, 2.43)	1.36	(0.77, 2.43)
High	7.3	(16/219)	1.34	(0.71, 2.54)	1.45	(0.77, 2.72)	1.83	(1.13, 2.97)*	1.85	(1.10, 3.13)*
CDAH-2 DGI score third										
Females										
Low	10.6	(26/245)	1.00		1.00		1.00		1.00	
Middle	9.0	(30/335)	0.87	(0.52, 1.46)	0.92	(0.55, 1.54)	0.89	(0.54, 1.49)	0.89	(0.53, 1.48)
High	8.3	(31/375)	0.86	(0.51, 1.44)	0.87	(0.51, 1.46)	0.88	(0.53, 1.45)	0.83	(0.50, 1.39)
Males										
Low	6.4	(15/233)	1.00		1.00		1.00		1.00	
Middle	7.6	(13/172)	1.14	(0.55, 2.34)	1.12	(0.51, 2.45)	1.15	(0.52, 2.54)	1.14	(0.50, 2.55)
High	5.8	(7/120)	0.85	(0.34, 2.12)	0.86	(0.32, 2.26)	0.87	(0.33, 2.25)	0.84	(0.31, 2.27)
CDAH-3 DGI score third										
Females										
Low	18.2	(27/148)	1.00		1.00		1.00		1.00	
Middle	9.9	(23/232)	0.51	(0.29, 0.89)*	0.61	(0.34, 1.10)	0.58	(0.30, 1.11)	0.56	(0.28, 1.10)

High	8.7	(24/275)	0.45	(0.26, 0.77)*	0.59	(0.34, 1.03)	0.69	(0.38, 1.24)	0.70	(0.39, 1.26)
Males										
Low	9.6	(22/230)	1.00		1.00		1.00		1.00	
Middle	6.6	(11/166)	0.61	(0.29, 1.28)	0.58	(0.25, 1.35)	0.76	(0.31, 1.83)	0.77	(0.31, 1.90)
High	4.3	(6/140)	0.38	(0.14, 0.99)*	0.51	(0.20, 1.29)	0.68	(0.26, 1.77)	0.69	(0.27, 1.79)

*$p < 0.05$

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.

Table 6.13 Generalised estimating equations calculations of relative risk of first onset mood disorder during the follow-up periods between CDAH-1 (2004-06) to CDAH-2 (2009-11), and CDAH-2 to CDAH-3 (2014-19), for a 10-point higher Dietary Guidelines Index (DGI) score, and for middle and high compared to low DGI score third.

	% with mood disorder	(*n*/*N*)	Model 1[a]		Model 2[b]		Model 3[c]	
			RR[d]	(95% CI)	RR	(95% CI)	RR	(95% CI)
10-point higher DGI score								
Females	7.0	(60/858)	0.93	(0.74, 1.17)	1.04	(0.81, 1.33)	1.02	(0.79, 1.31)
Males	5.0	(34/674)	0.94	(0.70, 1.26)	0.91	(0.68, 1.23)	0.92	(0.68, 1.25)
DGI score thirds								
Females								
Low	6.7	(12/180)	1.00		1.00		1.00	
Middle	7.4	(22/297)	1.08	(0.55, 2.12)	1.37	(0.69, 2.72)	1.32	(0.67, 2.62)
High	6.8	(26/381)	0.97	(0.50, 1.86)	1.34	(0.67, 2.66)	1.25	(0.62, 2.50)
Males								
Low	4.1	(12/290)	1.00		1.00		1.00	
Middle	7.4	(17/229)	1.82	(0.88, 3.74)	1.77	(0.86, 3.66)	1.78	(0.86, 3.67)
High	3.2	(5/155)	0.77	(0.27, 2.16)	0.73	(0.25, 2.10)	0.75	(0.26, 2.18)

CDAH: Childhood Determinants of Adult Health study; RR: risk ratio; CI: confidence interval

[a] Model 1: adjusted for baseline age and time between follow-ups.

[b] Model 2: as per Model 1 plus additional adjustment for covariates as follows: females: smoking status, parental status, marital status, education, occupation, social support; males: marital status, parental status, education, social support.

[c] Model 3: as per Model 2, plus BMI.

[d] The RR value for the continuous DGI score is for a 10-point higher Dietary Guidelines Index (DGI) score (range 0-100). A higher DGI score indicates better diet quality. The RR for the DGI score thirds is for the middle or high third of Dietary Guidelines Index (DGI) score, compared to the lowest third of scores.

Chapter 7: An eating pattern characterised by skipped or delayed breakfast is associated with mood disorders among an Australian adult cohort

7.1 Preface

Although only very limited evidence of associations between diet quality and mood disorders was established in Chapters 5 and Chapter 6, there are other dietary factors additional to the nutritional composition of intake, that could be related to mood disorders. For example, meal timing may influence food choices, neurobiology and psychological states. To provide a more comprehensive examination of diet and dietary behaviours, this exploratory study examined if time-of-day eating patterns were associated with mood disorders among the CDAH cohort in adulthood. The study contained in this chapter was accepted and published by Psychological Medicine in September 2019 (245).

7.2 Introduction

Mood disorders, primarily depressive disorders, contribute more to worldwide disability than any other health condition (4). Diet may influence mood disorders due to the physiological effects of nutrients on biochemical processes involved in mental health, such as hormones, neurotransmitter activity, and the gut-brain axis (31). However, the frequency and timing of meals can also have hormonal, neurobiological and microbiome effects, thought to be related to circadian rhythms (258, 259). Physical effects include possible influence on cardiometabolic conditions such as diabetes and obesity that are often comorbid with mood disorders (20, 260, 261).

Existing research on the relationship between food timing and mood has largely involved à priori defined dietary behaviours and cross-sectional analyses. For example, skipping breakfast has been consistently cross-sectionally associated with depressive symptoms and poorer mental well-being among both youth (262-265),

and adults (266-269). These associations are often clinically significant, and robust to potential confounders including socioeconomic factors (267) and lifestyle practices such as diet quality, smoking, or alcohol consumption (264, 266, 269). Other eating behaviours are less well studied, but snacking between meals has been associated with depressive symptoms among adults (270), while snacking and meal skipping has been associated with higher levels of psychological problems in female adolescents (271). To our knowledge only one prospective study has examined multiple eating behaviours. That study reported that having at least two out of three unhealthy eating practices of skipping breakfast, snacking after dinner, or eating dinner shortly before bed was associated with a higher incidence of depressive symptoms (156).

Limitations of previous studies include examining discrete eating behaviours, using non-clinical measures of depression, or only considering concurrent mood. Cross-sectional analyses are unable to identify directionality of the relationship. Both high and low emotional states have been found to influence food consumption (272) meaning bidirectionality should be considered. Furthermore, despite the popularity of methods such as principal component analysis (PCA) to examine patterns of nutritional intake, it is rare for data-driven approaches to be used to determine time-of-day eating patterns. Two time-of-day eating patterns (a conventional pattern of three main meals, and a snack-dominant pattern), were derived using PCA in a 2011 cross-sectional study (273). However, the outcome in that study was sleep duration, not mood.

There were two important rationales for this study. Firstly, empirical analysis of eating and drinking occasions would allow us to determine common eating patterns that explain variation in timing of food intake over the day. The term “eating patterns” refers to patterns related to the timing and relative size of meals/snacks as a proportion of daily intake, not the foods, nutrients, or energy consumed. Secondly, examining bidirectional associations between eating patterns and clinical diagnosis of depressive episodes over time could help us understand the relationship between eating patterns and mood disorders if one exists. In this study we aimed to determine if time-of-day eating patterns were longitudinally associated with mood

disorders (dysthymia or depression) among an Australian cohort of young to middle-aged adults. We examined if eating pattern score predicted subsequent mood disorders, if tracking of pattern scores were associated with mood disorder over time, and if mood disorders predicted eating pattern scores.

7.3 Methods

7.3.1 Participants

In 1985, the Australian Department of Community Services and Health conducted the Australian Schools Health and Fitness Survey (ASHFS) of schoolchildren aged 7-15 years. A two-stage probability design derived a nationally representative sample. Of 121 schools approached, 109 schools participated (90.1% response rate). The student response rate was 67.6% (*N* = 8,498).

During 2001-02, ASHFS participants were traced and invited to participate in the Childhood Determinants of Adult Health (CDAH) study, resulting in enrolment of 5,170 participants (61.0%) (162). For the first follow-up 2004-06 (CDAH-1), *n* = 2,410 participants (aged 26-36 years) attended study clinics for physical measurements and completed questionnaires including a food frequency questionnaire (FFQ) and food habits questionnaire (FHQ). At the second follow-up 2009-11 (CDAH-2), *n* = 1,749 participants (aged 31-41 years) completed a mental health diagnostic interview, questionnaires, and the same FFQ and FHQ used in CDAH-1.

7.3.2 Ethical standards

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.

7.3.3 Eating occasions

At CDAH-1 and CDAH-2, participants were mailed questionnaires that were returned by post or collected at the CDAH-1 clinics. The FHQ included a meal pattern chart,

which collected information on the types of meals and drinks consumed from 6am the previous day to 6am that morning (274). The 24-hour period was divided into hourly periods (e.g. 6-7am) from 6am to 11pm, and an overnight period of 11pm-6am. For each time period, respondents were asked “Did you eat anything?” with responses of “No”, “A snack”, “A small meal” or “A large meal”, and “Did you drink anything”, with responses of “No”, “Alcohol”, “Water”, or “Something else”. Examples of meal types were given: snacks: a biscuit or piece of fruit; small meal: beans on toast, boiled egg and bread, breakfast cereal, a pie; large meal: meat and three vegetables or a large serving of fish and chips. Participants were instructed that they could fill in more than one type of drink for each period.

Seven time intervals were defined based on commonly-understood Australian meal windows to aid interpretability of results (146): early (6am-9am), late morning (9am-12pm), midday (12pm-3pm), afternoon (3pm-6pm), evening (6pm-9pm), night (9pm-11pm), overnight (11pm-6am). To estimate the proportion of daily food intake consumed during each interval, 1 point was awarded for a snack, 3 points for a small meal, and 5 points for a large meal. Water was not awarded any points, but drinks of “Alcohol” or “Something else” were awarded one point, according to accepted methods of including beverages as eating occasions (146, 273). The number of points consumed during each interval by a participant were divided by their total points consumed that day to calculate the percentage distribution of daily intake across the seven time intervals. This distribution therefore reflected temporal distribution of daily intake, not nutritional or energy intake.

Participants reported the day of the week they completed the meal pattern chart for. Participants were categorised as weekday (Monday to Friday) or weekend (Saturday or Sunday) reporters.

7.3.4 Mood disorder

Mental health was assessed at CDAH-2 using the lifetime version of the Composite International Diagnostic Interview (CIDI) (167). The computerised CIDI was administered by trained telephone interviewers to collect data on the lifetime prevalence of depressive symptoms, age of onset, and age of most recent recurrence. Symptoms were scored using DSM-IV criteria (9) to determine

depressive episodes, or dysthymia. Participants, including those who had experienced a mood disorder prior to CDAH-1, were categorised as having a mood disorder only if they had experienced any episodes (first or recurrent) between CDAH-1 and CDAH-2. Sensitivity analyses excluded all participants who had their first mood disorder prior to CDAH-1.

7.3.5 Covariates

At CDAH-1 and CDAH-2, questionnaires collected data on age, marital status (married/living as married, single/separated/divorced), highest education (university, vocational, school), occupational status (professional, non-manual, manual, not in workforce), and current smoking status (never, ex-smoker, smoker). Total weekly minutes of leisure-time physical activity were measured using the validated International Physical Activity Questionnaire long form (196), and converted to hours per week for interpretability. Parenting status (no children, have children) was determined using date of birth data for biological children reported at CDAH-2. Social support at CDAH-1 and CDAH-2 was measured using the Henderson Index of Perceived Social Support (potential range 15-75), with a higher score indicating higher self-perceived social support (238). At CDAH-2 only, participants reported the hours and minutes of usual sleep duration and the preferred amount of sleep they need to feel they have rested properly. Discrepancy of sleep preference was calculated as preferred minus usual sleep duration. At CDAH-2, participants reported their usual type of work schedule (regular daytime, evening/night/rotating, irregular (e.g. split shift, on call), not employed).

Dietary data were collected using a 127-item FFQ based on a validated FFQ developed for Australian populations (163, 164). Diet quality was calculated using a validated Dietary Guidelines Index (DGI) that reflects the 2013 Australian Dietary Guidelines (183). A higher score on the scale 0-100 indicated higher diet quality. At CDAH-2, participants were asked how many days per week they usually ate breakfast (range 0-7). Participants were categorised as never skip breakfast, sometimes skip (skip 1-3 days/week), or regularly skip (skip 4-7 days/week).

For CDAH-1 clinic participants, weight was measured to the nearest 0.1kg in light clothing using Heine portable digital scales (Heine, Dover, NH, US), and height to the

nearest 0.1cm with a Leicester stadiometer (Invicta, Leicester, UK). BMI was calculated as weight in kilograms divided by squared height in metres (kg/m^2). For CDAH-1 participants who did not attend clinics, and at CDAH-2, BMI was calculated from self-reported height and weight with a correction factor applied. The correction factor was determined based on discrepancies between the self-reported and measured height and weight of CDAH-1 clinic participants (168).

Transition variables reflect change in circumstance between CDAH-1 and CDAH-2: parenting status (no children, first child born since CDAH-1, additional children born since CDAH-1, same number of children as CDAH-1); marital status (stayed living as married, became living as married, stayed living as single, became living as single); smoking (non-smoker, stopped smoking, started smoking, continued smoking); change in education level (advanced education, same level of education); and change in employment (remained employed, became employed, became unemployed, remained unemployed). For continuous variables (BMI, social support, DGI, and leisure-time physical activity), the transition variable was calculated by the value at CDAH-2 minus the value at CDAH-1.

7.3.6 Statistical analyses

All analyses were performed in Stata Version 15 (StataCorp, College Station, Texas, 2017). Time-of-day eating patterns were determined by PCA of the percentages of daily food intake consumed during each time interval (6-9am, 9am-12pm, 12-3pm, 3-6pm, 6-9pm, 9-11pm, 11pm-6am). The number of components were selected based on visual examination of the scree plot, and size of the eigenvalues. Orthogonal varimax rotation was applied to improve interpretability of the identified components. Bartlett's test of sphericity was used to test whether the variables were unrelated and therefore unsuitable for PCA. The Kaiser-Mayer-Olkin statistic for sampling adequacy was not generated due to singular correlation matrices arising from standardisation of the eating interval variables to sum to one for each participant.

Every participant received a score for each pattern and scores were categorised by tertiles into low, middle and high thirds. A tracking variable for change in pattern scores from CDAH-1 to CDAH-2 was created: consistently low (lowest third of

pattern scores at both time-points), decreased (decrease from high or middle to a lower third), consistently middle (middle third at both time-points), increased (increase from low or middle to a higher third), or consistently high (highest third at both time-points). Tracking of pattern scores was determined by examining percent agreement of the categories and Cohen's Kappa coefficient for inter-rater reliability (275). At CDAH-2, percent agreement was also used to assess concordance of eating pattern score categories with reported frequency of eating breakfast.

Multiple imputation was performed to complete the 1985 ASHFS data for missing variables that predicted loss to follow-up. Inverse probability weighting on these variables was used in the regression analyses (motivated by Seaman *et al*., 2012). Firstly, we examined if eating patterns at CDAH-1 predicted risk of mood disorder during the follow-up period using log binomial regression to calculate relative risks (RR). Secondly, we examined if tracking of eating pattern scores from CDAH-1 to CDAH-2 was associated with prevalence of mood disorder during the intervening period using binomial logistic regression to calculate prevalence ratios (PR). Thirdly, to explore bidirectionality, we examined whether experiencing a mood disorder during follow-up predicted eating pattern category at CDAH-2. We used adjacent categories ordered log-link regression to calculate the relative risk (RR) for being in a higher adjacent score category for those who experienced a mood disorder during the follow-up period compared to those who did not (276). Males and females were analysed together as there was no evidence of differences by sex in the estimates.

Minimally adjusted models (Model 1) adjusted for sex and age. Purposeful model building procedures were used to determine the fully adjusted models (Model 2) with adjustment for variables thought to be causally associated with the outcome and that changed the coefficient of the principal study factor by at least 10% (240). Model 2 for the prediction of mood disorder based on CDAH-1 eating pattern adjusted for sex, age, social support, BMI, and smoking at CDAH-1. Model 2 for the tracking analyses adjusted for sex, CDAH-2 age and work schedule, and transitions between CDAH-1 and CDAH-2 in social support, marital status, smoking, and BMI. Model 2, for the analysis of mood disorder predicting eating pattern at CDAH-2, adjusted for sex and CDAH-2 age, education, BMI, work schedule, parental status,

smoking status, and self-perceived social support. Model 3 further adjusted for eating pattern category at CDAH-1. Statistical significance was deemed if $p < 0.05$.

Two PCA sensitivity analyses were conducted to check the robustness of the patterns by stratifying separately by: 1) sex, and 2) weekday/weekend. Two separate log binomial regression sensitivity analyses were conducted: 1) excluding weekend reporters; 2) excluding all participants who had experienced a mood disorder prior to CDAH-1.

7.4 Results

The meal pattern chart at CDAH-1 was completed by 2,853 participants, however 78 were excluded due to pregnancy. Of the remaining 2,775 participants, 1,435 completed the meal pattern chart at CDAH-2, with 39 participants excluded for pregnancy. Of the 1,396 participants with meal data at both time points, 1,374 also completed the CDAH-2 CIDI. PCA was performed separately on the CDAH-1 and CDAH-2 time-of-eating data for this group. After exclusion of 70 participants missing covariate data, the final sample for regression analyses was n = 1,304 (Figure 7.1). Participant characteristics are shown in Table 7.1**.**

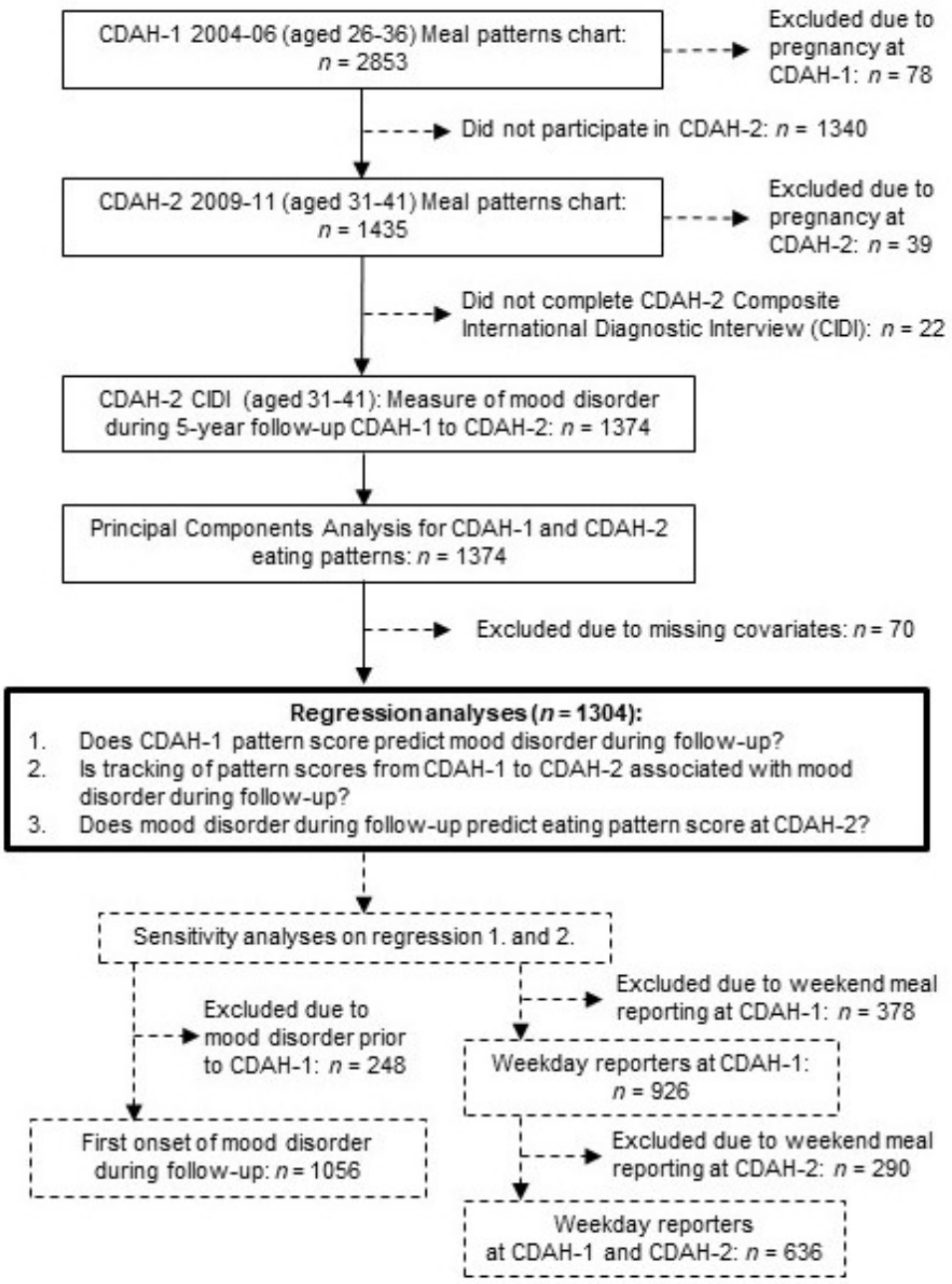


Figure 7.1 Childhood Determinants of Adult Health (CDAH) study participant flow chart and related analyses

Table 7.1 Participant characteristics by experience of mood disorder during follow-up (CDAH-1 to CDAH-2)

	CDAH-1 (2004-06)				CDAH-2 (2009-11)			
	No mood disorder		Mood disorder		No mood disorder		Mood disorder	
	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*	% or mean(*SD*)	*n*
Sex[a]								
Female	58.4	631	70.4	157	–	–	–	–
Male	41.6	450	29.6	66	–	–	–	–
Age (years)	31.6(2.7)	1081	31.5(2.6)	223	36.6(2.6)	1081	36.5(2.5)	223
Living as married								
No	27.3	295	34.1	76	16.0	173	31.4	70
Yes	72.7	786	65.9	147	84.0	908	68.6	153
Parental status								
No children	51.9	561	54.3	121	27.9	302	39.5	88
≥ 1 child	48.1	520	45.7	102	72.1	779	60.5	135
Smoking status								
Never	60.6	655	50.2	112	62.3	674	52.0	116
Ex-Smoker	20.3	219	20.6	46	24.5	265	28.7	64
Current smoker	19.1	207	29.1	65	13.1	142	19.3	43
Highest education								
University	47.6	515	47.1	105	49.7	537	52.5	117
Vocational	28.0	303	24.2	54	30.3	328	26.9	60
School	24.3	263	28.7	64	20.0	216	20.6	46
Occupation								
Professional	55.9	596	53.4	117	58.6	631	55.7	123
Non-manual	18.1	193	20.5	45	17.7	191	19.0	42
Manual	13.3	142	11.0	24	13.1	141	11.3	25
Not working	12.7	136	15.1	33	10.6	114	14.0	31

BMI (kg/m^2)	25.2(4.7) 1081	26.0(5.4) 223	25.7(5.0) 1081	27.0(6.1) 223
Leisure-time physical activity (hrs/wk)	2.8(3.3) 1016	2.4(3.2) 204	2.8(3.1) 1004	2.4(3.1) 208
Social support[b]	62.5(7.1) 1081	59.0(8.2) 223	62.1(7.6) 1081	57.0(9.9) 223
Dietary Guidelines Index score[c]	56.0(11.1) 1049	56.7(11.5) 222	56.9(11.2) 1008	57.5(11.2) 211
Usual sleep (hrs:mins)	– –	– –	7:25(1:00) 1076	7:22(1:06) 221
Sleep discrepancy (hrs:mins)[d]	– –	– –	0:33 (1:04) 1068	0:46(1:17) 219
Work schedule				
Regular day	– –	– –	64.1 693	56.4 127
Irregular hours	– –	– –	21.0 227	22.7 51
Night/Evening/ Rotating	– –	– –	5.2 56	6.2 14
Not employed	– –	– –	9.7 105	13.8 31

CDAH: Childhood Determinants of Adult Health study; *SD*, Standard deviation; BMI, body mass index

[a] The distribution of mood disorder status during follow-up is presented once here under the CDAH-1 columns as the sex-distribution of the cohort was the same at CDAH-1 and CDAH-2.

[b] Henderson Index of Perceived Social Support, possible score range 15-75. A higher score indicates higher self-perceived social support.

[c] Possible score range 0-100 A higher score indicates better compliance with the 2013 Australian Dietary Guidelines.

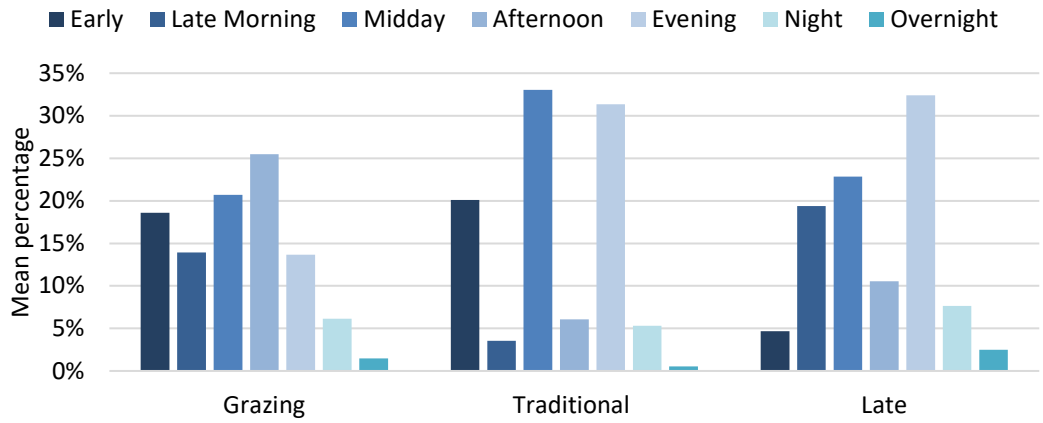
[d] Discrepancy between preferred and usual minutes of sleep per night.

7.4.1 Time-of-day eating patterns

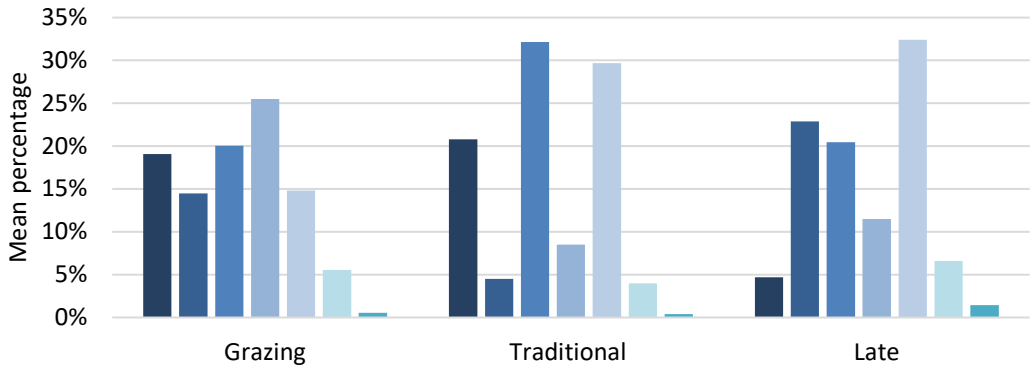
Three similar patterns were obtained at both time-points, cumulatively explaining 65% (CDAH-1) and 64% (CDAH-2) of the variation in timing of daily food intake. Factor loadings, which indicate the strength of association between the variable and component, and scree plots are shown in Section 7.8 Supplementary material Table 7.4 and Figure 7.3 respectively.

Bartlett test of sphericity results for CDAH-1 and CDAH-2 were $p < 0.001$. Sensitivity analyses of PCA on subgroups male, female, weekday, and weekend day produced the same three dominant patterns, with similar loadings to whole-of-group patterns (data not shown).

The mean percentages of daily intake consumed at each of the seven time intervals by those in the highest third of pattern scores, were examined to further describe and name the patterns as Grazing, Traditional, and Late (Figure 7.2). Those high on the Grazing pattern had intake spread across the day from 6am-6pm and consumed the highest average percentage of their daily food intake during the afternoon 3-6pm. The Traditional pattern was characterised as three main intakes, with the largest mean percentages reflecting breakfast, lunch and dinner times. The Late pattern was characterised by low intake during 6-9am, with slightly higher mean percentages of intake during the night and overnight periods than the other patterns.



A.Time-of-day Eating Patterns at CDAH-1 (2004-06)



B. Time-of-day Eating Patterns at CDAH-2 (2009-11)

CDAH: Childhood Determinants of Adult Health study
Early: 6-9am; Late Morning: 9am-12pm; Midday: 12-3pm; Afternoon: 3-6pm; Evening 6-9pm; Night: 9-11pm; Overnight: 11pm-6am.

Figure 7.2 Mean percentage of daily intake by eating interval among participants scoring in highest third of each time-of-day eating pattern at CDAH-1 and CDAH-2

There was evidence of tracking of participant scores for all patterns from CDAH-1 to CDAH-2, with participants more likely to be in the same score category at CDAH-2 than the two other score categories (Section 7.8 Supplementary material, Table 7.5). For example, of the 33.4% of participants who were in the highest third of the Late pattern at CDAH-1, 16.0% were also in the highest third at CDAH-2, 8.6% in the middle third, and 8.8% in the lowest third.

Only the Late pattern was associated with skipping breakfast. Of the 426 participants in highest third of the Late pattern who had breakfast frequency data, 239 (56.1%) reported skipping breakfast at least once per week (Section 7.8 Supplementary material, Table 7.6).

7.4.2 Associations between eating patterns and mood disorder

Time-of-day eating patterns at CDAH-1 were not significantly associated with mood disorder outcomes during the 5-year follow-up (Table 7.2). A borderline significant increased risk for those in the highest compared to the lowest third of the Late pattern (RR= 1.33; 95% CI: 0.97, 1.83) was attenuated in Model 2 (RR= 1.13; 95% CI: 0.82, 1.55).

Associations between pattern score tracking categories from CDAH-1 to CDAH-2 and mood disorder during follow-up are also shown in Table 7.2. After adjustment, compared to those in the consistently low category of the Late pattern, there was a higher prevalence of mood disorder among those in the increased (PR=1.85; 95% CI: 1.11, 3.09) and consistently high (PR=2.04; 95% CI: 1.20, 3.48) categories. A significant trend for the Late pattern was observed, with higher pattern category associated with higher prevalence of mood disorder. Indications of higher prevalence of mood disorder among those in the consistently high category of the Grazing pattern and lower prevalence among those in the consistently high category of the Traditional pattern, were not statistically significant.

Table 7.2 Associations between time-of-day eating pattern category at CDAH-1 or tracking of eating pattern category from CDAH-1 to CDAH-2, with mood disorder during follow-up between CDAH-1 and CDAH-2

	Mood events		Model 1[a,b]		Model 2[c,d]	
	%	(*n*/*N*)	RR/PR	(95% CI)	RR/PR	(95% CI)
CDAH-1 patterns predicting mood disorder risk during follow-up						
Grazing						
Low	16.5	(72/437)	Reference		Reference	
Middle	16.7	(73/437)	0.94	(0.69, 1.29)	0.92	(0.67, 1.24)
High	18.1	(78/430)	1.03	(0.75, 1.40)	0.92	(0.68, 1.25)
Trend				$p = 0.862$		$p = 0.612$
Traditional						
Low	18.7	(83/444)	Reference		Reference	
Middle	17.0	(73/429)	0.89	(0.66, 1.21)	0.98	(0.71, 1.35)
High	15.5	(67/431)	0.84	(0.61, 1.15)	1.01	(0.72, 1.41)
Trend				$p = 0.262$		$p = 0.969$
Late						
Low	15.0	(65/434)	Reference		Reference	
Middle	17.1	(74/434)	1.11	(0.81, 1.54)	1.11	(0.81, 1.53)
High	19.3	(84/436)	1.33	(0.97, 1.83)	1.13	(0.82, 1.55)
Trend				$p = 0.076$		$p = 0.473$
Tracking category CDAH-1 to CDAH-2 and association with prevalence of mood disorder during follow-up						
Grazing						
Consistently low	15.1	(29/192)	Reference		Reference	
Decreased	18.0	(72/400)	1.12	(0.73, 1.71)	1.22	(0.81, 1.83)
Consistently middle	17.2	(27/157)	1.18	(0.71, 1.99)	1.35	(0.82, 2.23)
Increased	17.5	(67/383)	1.22	(0.79, 1.89)	1.38	(0.92, 2.08)
Consistently high	16.3	(28/172)	1.11	(0.66, 1.86)	1.14	(0.70, 1.86)
Trend				$p = 0.535$		$p = 0.321$
Traditional						
Consistently low	21.1	(37/175)	Reference		Reference	
Decreased	16.1	(63/392)	0.72	(0.49, 1.06)	0.76	(0.52, 1.10)
Consistently middle	16.7	(28/168)	0.77	(0.48, 1.25)	0.95	(0.59, 1.54)
Increased	17.9	(73/407)	0.79	(0.54, 1.15)	0.83	(0.57, 1.19)
Consistently high	13.6	(22/162)	0.61	(0.37, 1.01)	0.64	(0.39, 1.06)
Trend				$p = 0.209$		$p = 0.284$
Late						
Consistently low	10.6	(19/180)	Reference		Reference	
Decreased	15.2	(56/369)	1.51	(0.89, 2.56)	1.28	(0.75, 2.21)
Consistently middle	12.3	(21/171)	1.27	(0.67, 2.38)	1.20	(0.64, 2.24)
Increased	20.0	(75/375)	2.13	(1.28, 3.53)*	1.85	(1.11, 3.09)*
Consistently high	24.9	(52/209)	2.69	(1.60, 4.55)*	2.04	(1.20, 3.48)*
Trend				$p < 0.001$		$p < 0.001$

*$p < 0.05$.

[a] Prediction analysis models adjusted for sex and age at CDAH-1.

[b] Tracking analysis models adjusted for sex and age at CDAH-2.

[c] Prediction analysis models adjusted for sex and CDAH-1 age, BMI, social support, and smoking status.
[d] Tracking analysis models adjusted for sex, age, and work schedule at CDAH-2, and change from CDAH-1 to CDAH-2 in social support, smoking, marital status, and BMI.

Results for the analysis of mood disorder predicting eating pattern scores are presented in Table 7.3. After adjustment for covariates, participants who experienced a mood disorder during the follow-up period had a 7% increased risk (RR=1.07; 95% CI: 1.00, 1.14) of being in a higher adjacent score category (e.g. high rather than middle, or middle rather than low), compared to participants who had not experienced a mood disorder during follow-up. Having a mood disorder during follow-up was not associated with the Grazing or Traditional patterns at CDAH-2.

Table 7.3 Relative risk of being in a higher score category of CDAH-2 eating pattern for participants who experienced a mood disorder during follow-up between CDAH-1 and CDAH-2, compared to participants who did not experience a mood disorder during follow-up

	Model 1[a]		Model 2[b]		Model 3[c]	
CDAH-2 pattern	RR	(95% CI)	RR	(95% CI)	RR	(95% CI)
Grazing	1.04	(0.98, 1.12)	1.02	(0.95, 1.10)	1.03	(0.97, 1.10)
Traditional	0.95	(0.88, 1.03)	0.99	(0.91, 1.07)	0.99	(0.92, 1.07)
Late	1.12	(1.06, 1.19)*	1.08	(1.01, 1.15)*	1.07	(1.00, 1.14)*

CDAH: Childhood Determinants of Adult Health study; RR: relative risk; CI: confidence interval.
*$p < 0.05$.
[a] Model 1: Adjusted for sex and CDAH-2 age.
[b] Model 2: Adjusted for sex and CDAH-2 age, BMI, education level, work schedule, parenting status, smoking status, and social support.
[c] Model 3: Model 2 plus additional adjustment for eating pattern category at CDAH-1.

Results of the sensitivity analyses are presented in Section 7.8 Supplementary material, Table 7.7 and Table 7.8. Among participants who experienced their first mood disorder between CDAH-1 and CDAH-2, those in the consistently high category of the Late pattern had higher prevalence of mood disorder compared to those in the consistently low category (PR=2.84; 95% CI: 1.06, 7.58). For the Traditional pattern, compared to those in the lowest category at both time points, a lower prevalence of mood disorders during the follow-up period was observed among those in the consistently middle category (PR=0.34; 95% CI: 0.12, 0.99), and

the consistently high category (PR=0.31; 95% CI: 0.11, 0.87). After excluding weekend reporters, compared to those in the lowest category of the Late pattern at both time-points, those in the increasing (PR=2.30; 95% CI: 1.01, 5.24) and consistently high categories (PR=3.46; 95% CI: 1.47, 8.14) had an increased prevalence of mood disorder during follow-up. Those who increased their Grazing pattern score category between follow-ups also had a higher prevalence of mood disorders during follow-up (PR=2.67; 95% CI: 1.19, 5.99) compared to those in the consistently low category.

7.5 Discussion

Three distinct time-of-day eating patterns were identified. The Traditional pattern described a conventional eating schedule of breakfast, lunch, and dinner, and the Grazing pattern had intake spread more evenly across the daytime hours. The Late pattern was characterised by low intake in the early morning (6-9am) but higher intakes late morning, indicating skipped or delayed breakfast, and proportionally more food consumed during the evening and night than the other patterns. High compared to low scores on the Late pattern at both time-points were associated with a higher likelihood of experiencing a mood disorder, and a nearly three times higher prevalence of first ever onset of a disorder during the intervening 5-year period. However, there was also weak evidence of bidirectionality, with mood disorder during follow-up associated with slightly increased risk of being in a higher Late pattern score category at CDAH-2. Participants who consistently scored in the middle or highest third of the Traditional pattern had a lower prevalence of first onset of mood disorder during the follow-up period. These results suggest that a more traditionally structured pattern of eating may be associated with better mental health.

Preference for a later-in-the-day style of eating could be a biological or social trait that is implicated in, or predisposes an individual to, poorer mental health. Chronotype characteristics relating to difference in preference for morning or evening activity may contribute to the observed associations. Evening chronotypes are more likely to skip or delay breakfast, consume higher intakes of food later in the day compared to morning types (277, 278), and have a higher risk of major

depressive disorder (279, 280). It is suggested that preference for evening activity may be a pre-existing trait of the individual rather than symptom of mood disorders (281, 282). A later pattern of eating may precede onset of mood disorders, and contribute to "social jetlag" which has been associated with depressive symptoms (283). Social jetlag refers to a discrepancy between biological and social or work schedules, where evening chronotypes are unable to fulfil their sleep timing preferences (284). In our cohort, a larger mean discrepancy between preferred sleep and actual sleep times at CDAH-2 was reported by participants who experienced a mood disorder (46 minutes) than those with no mood disorder (33 minutes). However, the amount of reported usual sleep was very similar at 7 hours 22 minutes for those who had experienced a mood disorder compared to 7 hours 25 minutes for those who had not. Usual sleep duration and sleep preference were not included in our adjusted models as they did not have sufficient effect on the prevalence estimates after inclusion of other covariates.

There were indications of bidirectionality, as participants with mood disorders during follow-up were slightly more likely to be in a higher score category of the Late pattern at CDAH-2 compared to participants who had not experienced a mood disorder. Mood disorders may influence lifestyle and dietary behaviours, but this does not preclude the influence of chronobiology. Mood disorders and emotional stress may reduce capacity to adhere to morning or daytime work/life schedules, or what are considered favourable behaviours such as making healthy food choices (47). Therefore, bidirectionality and the concept of social jetlag and chronobiology should be considered when exploring the nexus between diet, time-of-day eating patterns, and mood disorders.

Our results concerning the Late pattern complement existing literature reporting cross-sectional associations between skipping breakfast and depressive symptoms (262-267). However, "breakfast" has often been poorly defined or not defined at all (285) making it difficult to determine whether associations are due to not eating a morning meal, or delaying first consumption until later in the morning. In the current study, the Late pattern is likely to reflect both skipped and delayed breakfast. Participants who scored highly on the Late pattern had greater intake

during late morning (9am-12pm) compared to other patterns, and more than half of these participants reported they usually skipped breakfast at least once per week. Although this demonstrates the need for clarification around what constitutes breakfast, previous studies examining various concepts of "skipping breakfast" have highlighted the physiological and hormonal mechanisms that could explain the associations between omitting or delaying breakfast and mood disorders. Skipping breakfast has been shown to be associated with poorer diet quality and obesity which may affect mood due to long-term nutritional imbalance as well as metabolic co-morbidities (274, 285, 286). Eating breakfast lowers cortisol levels so skipping or delaying this meal may affect mood due to higher levels of cortisol and immune system dysregulation (267, 287). Lower appetite for breakfast first thing in the morning could also indicate reduced levels of the appetite regulating hormone ghrelin. Ghrelin has been shown to have an anti-depressant effect in mice (288) and affect plasma cortisol (289). Proximity of the last eating occasion can influence the amount of food consumed at the following eating occasion, so higher intake at night may result in less subsequent hormonal drive to eat early the next day. People with night eating syndrome (NES), typified by > 50% of daily calorie intake during the evening and waking at night to eat, have been shown to have lower ghrelin levels than controls during the early morning period to 9am (290). We do not suggest that participants who scored high on the Late pattern meet criteria for NES, but later eating combined with skipping breakfast could be eating practices that warrant further attention.

Associations between the Grazing pattern and mood disorder only reached statistical significance in the sensitivity analyses excluding weekend reporters, with those who increased their score category between CDAH-1 and CDAH-2 having a 2.7 times higher prevalence of mood disorder during the follow-up period. The Grazing pattern's spread of food intake across daytime hours, could represent snacking type behaviour and varied eating schedules. Irregular meal schedules, including skipped meals, snacking, and delayed lunch, have been associated with unfavourable health outcomes including obesity, depressed mood, and hypertension (270, 291, 292).

Consistently high scores on the Traditional pattern, characterised by distinct mealtimes, was associated with a non-statistically significant lower prevalence of mood disorder during follow-up. Furthermore, in the sensitivity analyses, high scores on the Traditional pattern at CDAH-1 was associated with a lower risk of first ever onset of mood disorder during follow-up. Structured and regular mealtimes may indicate healthier behaviours. In a previous study, healthier lifestyle behaviours were protective against mood disorders among the CDAH cohort (244).

Limitations of this study include potential bias as the meal pattern chart was reliant on recall and only covered a single 24-hour period at each time-point which may not reflect usual eating patterns. However, there was evidence the pattern scores tracked from CDAH-1 to CDAH-2, indicating possible habituality of time-of-day eating. There was no guidance given to participants about entering multiple meal types in the same hourly period, or which time-period they should use when entering food or drink consumed on the hour (e.g. whether a drink at 7am should be entered as 6-7am or 7-8am). The 11pm-6am period meant there was no differentiation between overnight eating and an early breakfast. Bias from loss to follow-up between the nationally representative baseline youth sample and the adult surveys may limit the generalisability of our results. However, there was wide variation in the characteristics of participants in the adult sample and loss to follow-up was mitigated by inverse probability weighting. There is also the possibility of bias from misreporting of covariate measures, such as self-reported weight (mitigated by using a correction factor) and physical activity; or unmeasured confounding such as lifestyle (e.g. work schedule or sleep hours at CDAH-1) or psychological factors.

Strengths of the study include the use of the CIDI, which is considered the “gold-standard” measure for retrospective assessment of history of mental disorders in epidemiological studies (89). Participant recollection may have resulted in some misreporting. However, the time-related questions in the CIDI around the first and last occurrence of a disorder have shown good reliability (87). Although misreporting of snack and beverage intake is common in dietary surveys, primarily as under-reporting (293), converting each individual’s eating occasion to a proportion of their

total intake may have helped address systematic misreporting by individuals, or variation in concepts of snack or meal sizes between participants. The assessment of BMI, overall diet quality, and physical activity as covariates in our models considered potential confounding or mediation from energy and nutritional aspects of diet. Diet quality and physical activity did not change the coefficients sufficiently to be included in our models, indicating they were not confounding measures. The sensitivity analyses on the PCA and regression analyses confirmed that the patterns and associations were robust to influence of factors such as sex, prior mood disorder, and differences between weekday and weekend eating practices. Another strength is the novel application of PCA to derive patterns that capture dietary behaviours, and in the case of the Late pattern, multiple behaviours of skipping breakfast and eating later into the evening. Furthermore, the longitudinal design builds on existing cross-sectional research.

Longitudinal studies that replicate the eating patterns observed in this study, or specifically examine clustering of several habits, may be useful in determining lifestyle and chronobiological influences on mood disorders. Repeat measures and more detailed information about timing and size of meals would help determine the nature of the relationship between eating patterns and mental health outcomes.

7.6 Conclusion

In conclusion, delaying or skipping breakfast and eating higher proportions of intake later in the day may be an unhealthy behaviour associated with higher likelihood of mood disorder among adults. Whereas more traditional eating patterns of main meals at breakfast, lunch and dinner may be associated with lower likelihood of mood disorder over time. These relationships may be bidirectional, and a pre-existing preference for certain eating patterns due to chronobiological traits of the individual should be considered.

7.7 Postscript

This study has demonstrated that analysis of time-of-day eating occasions may be an understudied but important aspect of diet related to mood disorder outcomes. In addition to the effects of social support shown in the cross-sectional analysis

between diet quality and mood disorder in Chapter 6, this study highlights that other lifestyle or dietary aspects such as habitual timing of eating occasions may also need to be considered as potential confounding variables.

7.8 Supplementary material

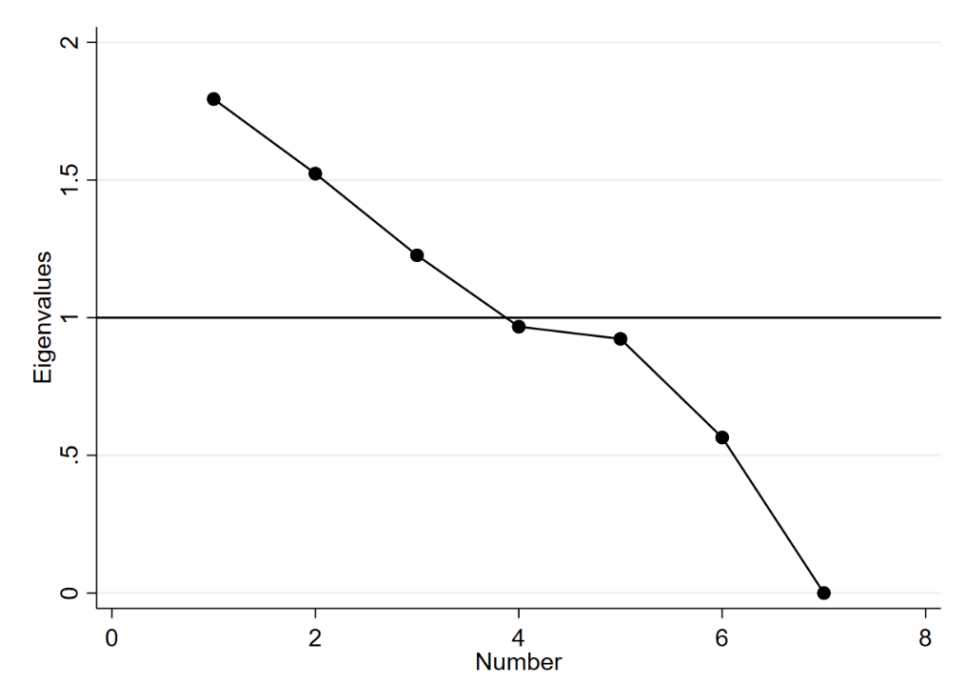
7.8.1 Supplementary material for the published paper

The following results were submitted to the journal and published as online supplementary material.

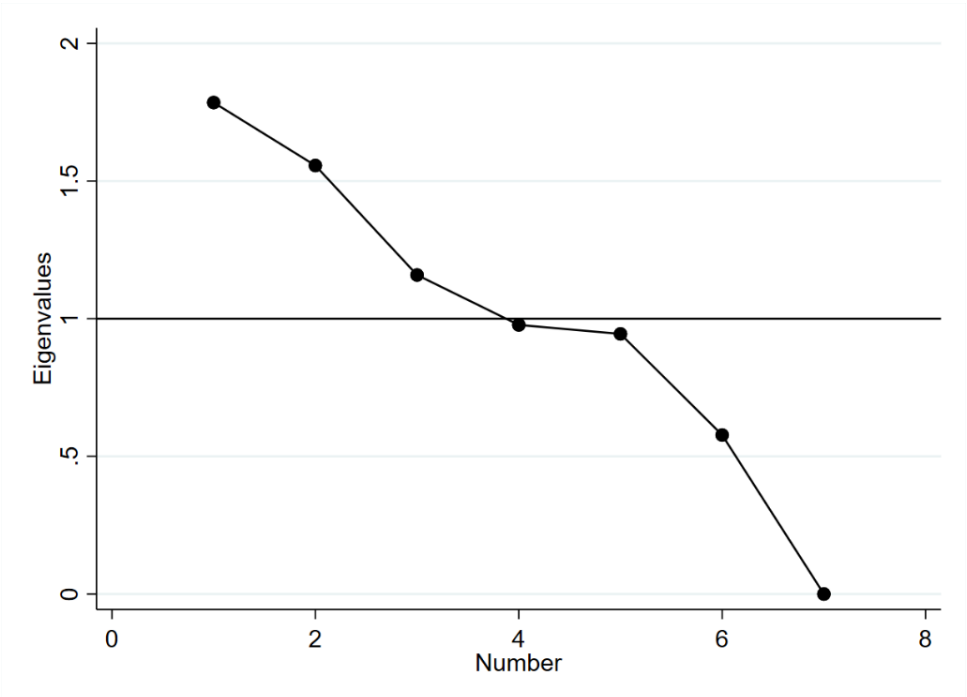
Table 7.4 Time-of-day eating pattern factor loadings generated by principal component analyses of percentage of daily food consumed during each interval at CDAH-1 and CDAH-2

	CDAH-1[a]			CDAH-2[a]		
Eating interval	Grazing	Traditional	Late	Grazing	Traditional	Late
Early 6-9am	–	–	–0.69[b]	–	–	–0.74
Late morning 9am-12pm	–	–0.67	–	–	–0.61	–
Midday 12-3pm	–	0.69	–	–	0.75	–
Afternoon 3-6pm	0.65	–	–	0.67	–	–
Evening 6-9pm	–0.73	–	–	–0.72	–	–
Night 9-11pm	–	–	0.49	–	–	0.51
Overnight 11pm-6am	–	–	0.47	–	–	0.32
Eigenvalue	1.68	1.53	1.33	1.68	1.46	1.37
Variance explained[c]	0.24	0.22	0.19	0.24	0.21	0.20

CDAH: Childhood Determinants of Adult Health study.
[a] *N* = 1,374.
[b] Only factor loadings > |0.3| are shown for clarity. Loadings are for varimax rotated components.
[c] Proportion of common variance (total of 1.00), explained by component.



7.3A. Scree plot of eigenvalues after PCA on eating-time intervals at CDAH-1 (*n* = 1,374)



7.3B. Scree plot of eigenvalues after PCA on eating-time intervals at CDAH-2 (*n* = 1,374)

Figure 7.3 Principal components analysis (PCA) scree plots for time-of-day eating patterns at CDAH-1 (2004-06) and CDAH-2 (2009-11)

Table 7.5 Percent agreement of low, middle and high score categories of time-of-day eating patterns at CDAH-1 (2004-06) and CDAH-2 (2009-11)

Pattern and score category at CDAH-1	Pattern score category at CDAH-2						Cohen's Kappa[a]
	Lowest		Middle		Highest		
	%	(*n*)	%	(*n*)	%	(*n*)	
Grazing[b]							0.099
Lowest	14.7	(192)	9.9	(129)	8.9	(116)	
Middle	10.9	(142)	12.0	(157)	10.6	(138)	
Highest	8.2	(107)	11.6	(151)	13.2	(172)	
Traditional[c]							0.081
Lowest	13.4	(175)	10.1	(131)	10.6	(138)	
Middle	9.4	(123)	12.9	(168)	10.6	(138)	
Highest	10.1	(132)	10.5	(137)	12.4	(162)	
Late[d]							0.144
Lowest	13.8	(180)	11.6	(151)	7.9	(103)	
Middle	10.9	(142)	13.1	(171)	9.3	(121)	
Highest	8.8	(115)	8.6	(112)	16.0	(209)	

CDAH: Childhood Determinants of Adult Health study.

[a] Possible range -1 to +1. < 0: no agreement; 0–0.20: slight; 0.21–0.40: fair; 0.41–0.60: moderate; 0.61–0.80: substantial; 0.81–1: almost perfect agreement.

[b] Grazing pattern: intake spread across the day, highest in the afternoon.

[c] Traditional pattern: highest proportions of intake reflect breakfast, lunch and dinner times.

[d] Late pattern: skipped/delayed breakfast and higher intakes during the evening.

Table 7.6 Percent agreement of low, middle and high score categories of time-of-day eating patterns and weekly frequency of skipping breakfast at CDAH-2 (2009-11)

Pattern and score category at CDAH-2	Usual breakfast skipping frequency per week at CDAH-2[a]						Cohen's Kappa[b]
	Never (*n* = 875)		1-3 days (*n* = 235)		4-7 days (*n* = 174)		
	%	(*n*)	%	(*n*)	%	(*n*)	
Grazing[c]							-0.110
Lowest	21.9	(281)	5.8	(74)	5.8	(75)	
Middle	24.2	(311)	6.4	(82)	3.3	(42)	
Highest	22.0	(283)	6.2	(79)	4.4	(57)	
Traditional[d]							-0.032
Lowest	20.6	(265)	6.1	(78)	6.1	(78)	
Middle	24.8	(319)	5.8	(74)	3.0	(38)	
Highest	22.7	(291)	6.5	(83)	4.5	(58)	
Late[e]							0.144
Lowest	27.7	(356)	5.1	(65)	0.8	(10)	
Middle	25.9	(332)	5.0	(64)	2.4	(31)	
Highest	14.6	(187)	8.3	(106)	10.4	(133)	

CDAH: Childhood Determinants of Adult Health study.

[a] *N* = 1,284.

[b] Possible range -1 to +1. < 0: no agreement; 0–0.20: slight; 0.21–0.40: fair; 0.41–0.60: moderate; 0.61–0.80: substantial; 0.81–1: almost perfect agreement.

[c] Grazing pattern: intake spread across the day, highest in the afternoon.

[d] Traditional pattern: highest proportions of intake reflect breakfast, lunch and dinner times.

[e] Late pattern: skipped/delayed breakfast and higher intakes during the evening.

Table 7.7 Sensitivity analyses: Associations between time-of-day eating pattern category at CDAH-1 or tracking of eating pattern category from CDAH-1 to CDAH-2, with first onset of mood disorder during follow-up between CDAH-1 and CDAH-2

	Mood events		Model 1[a,b]		Model 2[c,d]	
	%	(*n*/*N*)[e]	RR/PR	(95% CI)	RR/PR	(95% CI)
CDAH-1 patterns predicting mood disorders[f] during follow-up						
Grazing						
Low	7.4	(26/353)		Reference		Reference
Middle	6.6	(24/362)	0.73	(0.42, 1.26)	0.74	(0.43, 1.27)
High	7.0	(24/341)	0.89	(0.50, 1.56)	0.86	(0.49, 1.52)
Trend				$p = 0.683$		$p = 0.615$
Traditional						
Low	8.5	(30/353)		Reference		Reference
Middle	6.5	(22/340)	0.76	(0.44, 1.33)	0.81	(0.46, 1.43)
High	6.1	(22/363)	0.79	(0.45, 1.38)	0.90	(0.50, 1.62)
Trend				$p = 0.398$		$p = 0.698$
Late						
Low	7.0	(26/372)		Reference		Reference
Middle	6.7	(23/344)	1.00	(0.56, 1.77)	1.01	(0.57, 1.78)
High	7.4	(25/340)	1.21	(0.69, 2.13)	1.06	(0.61, 1.85)
Trend				$p = 0.523$		$p = 0.845$
Tracking categories CDAH-1 to CDAH-2 and association with mood disorder[f] during follow-up						
Grazing						
Consistently low	6.3	(10/159)		Reference		Reference
Decreased	8.0	(26/325)	1.14	(0.55, 2.37)	1.15	(0.55, 2.40)
Consistently middle	6.2	(8/130)	0.80	(0.31, 2.05)	0.78	(0.30, 2.04)
Increased	8.1	(25/307)	1.16	(0.55, 2.44)	1.19	(0.56, 2.53)
Consistently high	3.7	(5/135)	0.55	(0.18, 1.73)	0.54	(0.18, 1.60)
Trend				$p = 0.457$		$p = 0.451$
Traditional						
Consistently low	11.6	(16/138)		Reference		Reference
Decreased	6.8	(22/323)	0.58	(0.31, 1.11)	0.62	(0.32, 1.23)
Consistently middle	3.8	(5/131)	0.28	(0.10, 0.83)*	0.34	(0.12, 0.99)*
Increased	8.0	(26/326)	0.62	(0.33, 1.17)	0.63	(0.34, 1.16)
Consistently high	3.6	(5/138)	0.30	(0.11, 0.83)*	0.31	(0.11, 0.87)*
Trend				$p = 0.068$		$p = 0.054$
Late						
Consistently low	3.8	(6/159)		Reference		Reference
Decreased	6.6	(20/304)	1.80	(0.66, 4.86)	1.60	(0.59, 4.32)
Consistently middle	3.6	(5/138)	1.23	(0.35, 4.26)	1.15	(0.33, 3.98)
Increased	8.6	(26/301)	2.61	(1.00, 6.82)*	2.32	(0.89, 6.07)
Consistently high	11.0	(17/154)	3.73	(1.37, 10.15)*	2.84	(1.06, 7.58)*
Trend				$p = 0.002$		$p = 0.011$

CDAH: Childhood Determinants of Adult Health study; RR, relative risk; PR, prevalence ratio; CI, confidence interval.

*$p < 0.05$.

[a] Prediction analysis models adjusted for sex and age at CDAH-1.

[b] Tracking analysis models adjusted for sex and age at CDAH-2.
[c] Prediction analysis models adjusted for sex and CDAH-1 age, BMI, social support, and smoking status.
[d] Tracking analysis models adjusted for sex, age, and work schedule at CDAH-2, and change from CDAH-1 to CDAH-2 in social support, smoking, marital status, and BMI.
[e] Total participants *N* = 1,056.
[f] Only includes those who experienced their first mood disorder during the follow-up period. Participants were excluded from this analysis if they reported experiencing a mood disorder with age of onset at or prior to their age at CDAH-1.

Table 7.8 Sensitivity analyses: associations between time-of-day eating pattern category for weekday reporters only at CDAH-1 or tracking of pattern categories from CDAH-1 to CDAH-2, and mood disorder during follow-up between CDAH-1 and CDAH-2

	Mood events		Model 1[a,b]		Model 2[c,d]	
	%	(*n*/*N*)	RR/PR	(95% CI)	RR/PR	(95% CI)
CDAH-1 patterns and relative risk of mood disorders during follow-up (*n* = 926)						
Grazing						
Low	17.1	(55/321)		Reference		Reference
Middle	17.5	(58/331)	0.93	(0.65, 1.33)	0.88	(0.62, 1.25)
High	16.4	(45/274)	0.89	(0.61, 1.30)	0.78	(0.53, 1.15)
Trend				*p* = 0.545		*p* = 0.206
Traditional						
Low	20.2	(58/287)		Reference		Reference
Middle	15.0	(51/341)	0.71	(0.49, 1.03)	0.77	(0.52, 1.14)
High	16.4	(49/298)	0.82	(0.57, 1.19)	0.97	(0.66, 1.43)
Trend				*p* = 0.292		*p* = 0.798
Late						
Low	15.3	(50/327)		Reference		Reference
Middle	16.9	(54/320)	1.03	(0.71, 1.51)	1.01	(0.70, 1.46)
High	19.4	(54/279)	1.25	(0.85, 1.84)	1.06	(0.72, 1.56)
Trend				*p* = 0.256		*p* = 0.764
Tracking category CDAH-1 to CDAH-2 and mood disorder prevalence during follow-up (*n* = 636)						
Grazing						
Consistently low	7.7	(8/104)		Reference		Reference
Decreased	14.4	(25/174)	1.81	(0.80, 4.09)	1.86	(0.82, 4.23)
Consistently middle	13.8	(13/94)	1.92	(0.78, 4.71)	2.21	(0.88, 5.53)
Increased	17.5	(33/189)	2.65	(1.18, 5.96)*	2.67	(1.19, 5.99)*
Consistently high	10.7	(8/75)	1.45	(0.52, 4.04)	1.42	(0.51, 3.92)
Trend				*p* = 0.096		*p* = 0.083
Traditional						
Consistently low	12.5	(10/80)		Reference		Reference
Decreased	15.1	(28/186)	1.23	(0.60, 2.52)	1.24	(0.60, 2.55)
Consistently middle	11.9	(12/101)	1.05	(0.44, 2.52)	1.19	(0.49, 2.91)
Increased	14.0	(26/186)	1.27	(0.61, 2.64)	1.13	(0.51, 2.52)
Consistently high	13.3	(11/83)	1.20	(0.53, 2.75)	1.23	(0.54, 2.79)
Trend				*p* = 0.682		*p* = 0.888
Late						
Consistently low	8.3	(8/96)		Reference		Reference
Decreased	13.7	(24/175)	2.14	(0.95, 4.84)	1.86	(0.81, 4.31)
Consistently middle	8.6	(8/93)	1.15	(0.41, 3.19)	1.09	(0.39, 3.05)
Increased	14.2	(26/183)	2.53	(1.12, 5.67)*	2.30	(1.01, 5.24)*
Consistently high	23.6	(21/89)	4.34	(1.94, 9.72)*	3.46	(1.47, 8.14)*
Trend				*p* = 0.001		*p* = 0.002

CDAH: Childhood Determinants of Adult Health study; RR: relative risk; PR: prevalence ratio; CI: confidence interval.
*$p < 0.05$.

[a] Prediction analysis models adjusted for sex and age at CDAH-1.
[b] Tracking analysis models adjusted for sex and age at CDAH-2.
[c] Prediction analysis models adjusted for sex and CDAH-1 age, BMI, social support, and smoking status.
[d] Tracking analysis models adjusted for sex, age, and work schedule at CDAH-2, and change from CDAH-1 to CDAH-2 in social support, smoking, marital status, and BMI.

7.8.2 Additional supplementary material not submitted to the journal

Chi-square tests of association were performed on key demographic variables and score thirds of the time-of-day eating patterns at both follow-up. The pattern score thirds were defined using score tertiles from all participants with time-of-day eating pattern scores at each time-point, before participants were excluded from the study sample for missing outcome or covariate measures. The following Table 7.9 (CDAH-1) and Table 7.10 (CDAH-2) were not submitted to the journal, but are included here as further descriptive statistics of the study sample.

Table 7.9 Distribution of characteristics of the study sample at CDAH-1 (2004-06) by time-of-day eating pattern score third

	Afternoon Pattern				Traditional Pattern				Late Pattern			
Variable	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]
n[b]	437	437	430		444	429	431		434	434	436	
Sex, %												
Female	56.5	64.8	60.0	0.044	60.8	65.7	54.8	0.004	63.4	63.8	54.1	0.004
Male	43.5	35.2	40.0		39.2	34.3	45.2		36.6	36.2	45.9	
Age (years), %												
26-31	57.7	51.5	51.2	0.095	48.2	54.3	58.0	0.013	49.8	56.0	54.6	0.156
32-36	42.3	48.5	48.8		51.8	45.7	42.0		50.2	44.0	45.4	
DGI score[c]												
Lowest third	32.6	23.3	30.8	0.004	32.1	27.1	27.3	0.174	23.6	26.6	36.4	<0.001
Middle third	36.5	34.8	32.0		34.2	32.4	36.8		33.8	37.6	31.9	
Highest third	30.9	41.9	37.2		33.7	40.5	35.9		42.6	35.8	31.7	
BMI category, %												
Non-overweight (BMI < 25)	52.2	55.8	54.7	0.678	51.6	57.6	53.6	0.296	55.5	56.5	50.7	0.355
Overweight (25 ≤ BMI < 30)	34.8	30.7	30.9		34.7	28.2	33.4		31.3	31.6	33.5	
Obese (BMI ≥ 30)	13.0	13.5	14.4		13.7	14.2	13.0		13.1	12.0	15.8	
Smoking status, %												
Never	59.0	61.1	56.3	0.125	53.6	56.6	66.4	0.002	59.9	62.0	54.6	<0.001
Ex-smoker	17.8	21.7	21.4		22.5	22.6	15.8		25.6	19.8	15.6	
Smoker	23.1	17.2	22.3		23.9	20.7	17.9		14.5	18.2	29.8	
Marital status, %												
Living as single	28.8	27.5	29.1	0.851	29.1	27.5	28.8	0.865	21.7	25.6	38.1	<0.001
Living as married	71.2	72.5	70.9		70.9	72.5	71.2		78.3	74.4	61.9	

Social support index, %												
Lowest third	30.4	33.6	40.0	0.027	38.3	31.9	33.6	0.288	32.9	32.9	38.1	0.258
Middle third	38.4	33.4	30.2		33.6	34.7	33.9		33.4	34.3	34.4	
Highest third	31.1	33.0	29.8		28.2	33.3	32.5		33.6	32.7	27.5	
Parental status, %												
No children	58.8	49.0	49.1	0.004	50.2	50.3	56.4	0.117	44.9	51.8	60.1	<0.001
Children	41.2	51.0	50.9		49.8	49.7	43.6		55.1	48.2	39.9	
Education, %												
University	47.4	51.9	43.3	0.059	42.1	48.0	52.7	0.037	50.9	50.2	41.5	0.026
Vocational	25.4	25.4	31.4		30.9	27.3	23.9		24.4	27.6	30.0	
School	27.2	22.7	25.3		27.0	24.7	23.4		24.7	22.1	28.4	
Occupation, %												
Manager/Professional	57.6	58.2	50.4	0.251	48.1	53.5	65.1	0.000	56.5	56.2	53.6	0.072
Non-manual	18.2	17.2	20.2		21.2	19.3	14.9		18.2	16.6	20.7	
Manual	12.9	11.4	14.5		18.1	12.6	7.8		10.0	13.3	15.4	
Not in workforce	11.3	13.2	15.0		12.6	14.6	12.3		15.2	14.0	10.3	
Leisure physical activity (hrs/wk), %												
Lowest third	32.4	32.8	34.7	0.358	33.2	34.5	32.2	0.353	32.3	34.7	32.8	0.903
Middle third	34.4	31.8	36.4		36.1	30.3	36.2		34.5	34.5	33.6	
Highest third	33.2	35.4	28.9		30.8	35.2	31.7		33.3	30.8	33.6	
Work type												
Regular day	64.5	66.8	57.2	0.109	62.6	64.1	61.9	0.411	68.7	61.5	58.5	0.004
Irregular hours	19.5	19.9	24.7		21.2	21.2	21.6		18.7	22.6	22.7	
Night/Evening/Rotating	5.7	4.1	6.3		7.0	3.5	5.6		2.3	6.0	7.8	
Not employed	10.3	9.2	11.9		9.2	11.2	10.9		10.4	9.9	11.0	

[a] *p*-value for Pearson chi-squared test of independence.

[b] *n* for each score third of all variables except for the following variables that were not used in the fully adjusted regression analysis models after purposeful model building, and therefore were not used to exclude participants due to missing data: DGI (Afternoon - low: 427, middle: 425, high: 419; Traditional – low: 433, middle: 420, high: 418; Late – low: 420, middle: 425, high: 426), Occupation (Afternoon - low: 434, middle: 431, high: 421; Traditional – low: 443, middle: 419, high: 424; Late – low: 428, middle: 428, high: 429), and leisure time physical activity (Afternoon - low: 410, middle: 412, high: 398; Traditional – low: 413, middle: 406, high: 401; Late – low: 403, middle: 409, high: 408).

[c] Dietary Guidelines Index: higher score on range of 0-100 indicates higher diet quality.

Table 7.10 Distribution of characteristics of the study sample at CDAH-2 (2009-11) by time-of-day eating pattern score third

	Afternoon Pattern				Traditional Pattern				Late Pattern			
Variable	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]	Low	Middle	High	*p*-value[a]
n[b]	441	437	426		430	436	438		437	434	433	
Sex, %												
Female	56.5	60.4	64.6	0.051	59.8	67.0	54.6	0.001	62.0	63.1	56.1	0.076
Male	43.5	39.6	35.4		40.2	33.0	45.4		38.0	36.9	43.9	
Age (years), %												
31-36	58.3	49.9	54.0	0.045	54.0	52.5	55.7	0.639	52.2	54.4	55.7	0.580
37-41	41.7	50.1	46.0		46.0	47.5	44.3		47.8	45.6	44.3	
DGI score[b]												
Lowest third	37.5	32.9	31.3	0.119	34.7	30.5	36.7	0.102	32.0	27.3	42.7	<0.001
Middle third	35.1	33.2	33.3		35.1	32.3	34.2		33.0	37.3	31.3	
Highest third	27.4	33.9	35.4		30.2	37.2	29.1		35.0	35.4	26.1	
BMI category, %												
Non-overweight (BMI < 25)	45.8	52.2	49.1	0.146	44.9	53.9	48.2	0.121	49.9	50.0	47.1	0.218
Overweight (25 ≤ BMI < 30)	37.6	32.3	31.5		36.7	30.7	34.0		35.9	32.7	32.8	
Obese (BMI ≥ 30)	16.6	15.6	19.5		18.4	15.4	17.8		14.2	17.3	20.1	
Smoking status, %												
Never	60.1	59.3	62.4	0.792	55.1	62.4	64.2	0.029	65.0	61.3	55.4	<0.001
Ex-smoker	26.5	25.6	23.5		27.9	25.9	21.9		25.9	25.6	24.2	
Smoker	13.4	15.1	14.1		17.0	11.7	13.9		9.2	13.1	20.3	
Marital status, %												
Living as single	17.7	17.6	20.7	0.426	22.1	14.9	18.9	0.025	14.9	15.7	25.4	<0.001
Living as married	82.3	82.4	79.3		77.9	85.1	81.1		85.1	84.3	74.6	

Social support index, %												
Lowest third	37.2	38.2	39.7	0.918	42.6	34.2	38.4	0.080	37.1	34.8	43.2	0.072
Middle third	32.7	30.9	31.7		30.2	35.3	29.7		32.5	31.8	30.9	
Highest third	30.2	30.9	28.6		27.2	30.5	32.0		30.4	33.4	25.9	
Parental status, %												
No children	32.2	30.0	27.5	0.314	31.6	28.2	29.9	0.547	24.3	25.3	40.2	<0.001
Children	67.8	70.0	72.5		68.4	71.8	70.1		75.7	74.7	59.8	
Education, %												
University	47.6	51.9	50.9	0.600	45.8	53.7	50.9	0.145	52.2	53.2	45.0	<0.001
Vocational	30.8	30.0	28.4		31.9	26.6	30.8		32.7	28.1	28.4	
School	21.5	18.1	20.7		22.3	19.7	18.3		15.1	18.7	26.6	
Occupation, %												
Manager/Professional	60.5	60.8	52.8	0.001	54.5	57.9	61.8	0.193	58.4	58.5	57.4	0.609
Non-manual	19.8	17.7	16.3		17.4	18.6	17.8		19.3	17.9	16.7	
Manual	12.5	9.9	16.0		15.0	11.7	11.7		11.5	11.6	15.3	
Not in workforce	7.3	11.5	14.9		13.1	11.7	8.7		10.8	12.1	10.6	
Leisure physical activity (hrs/wk), %												
Lowest third	32.7	29.7	38.4	0.080	37.4	28.6	34.7	0.084	30.3	30.7	39.5	0.023
Middle third	35.3	34.1	29.6		32.3	34.0	32.9		33.3	33.8	32.1	
Highest third	32.0	36.3	31.9		30.3	37.4	32.4		36.4	35.5	28.4	
Usual sleep hours												
≤ 7.5 hours	52.7	56.5	53.4	0.504	57.8	50.6	54.3	0.105	47.4	57.2	58.1	0.002
> 7.5 hours	47.3	43.5	46.6		42.2	49.4	45.7		52.6	42.8	41.9	
Work type												
Regular day	67.8	64.1	56.6	<0.001	59.3	65.1	64.2	0.209	64.4	64.8	60.5	0.026
Irregular hours	21.5	21.5	20.9		23.0	18.8	22.1		23.7	18.5	22.1	
Night/Evening/Rotating	4.1	4.3	7.7		6.0	4.4	5.7		2.5	6.3	7.4	

Not employed	6.6	10.1	14.8		11.6	11.7	8.0		9.9	10.9	10.7

[a] *p*-value for Pearson chi-squared test of independence.

[b] *n* for each score third of all variables except for the following variables that were not used in the fully adjusted regression analysis models after purposeful model building, and therefore were not used to exclude participants due to missing data: DGI (Afternoon - low: 416, middle: 413, high: 390; Traditional – low: 404, middle: 403, high: 412; Late – low: 409, middle: 407, high: 403, Occupation (Afternoon - low: 440, middle: 434, high: 424; Traditional – low: 426, middle: 435, high: 437; Late – low: 435, middle: 431, high: 432), leisure time physical activity (Afternoon - low: 416, middle: 411, high: 385; Traditional – low: 396, middle: 412, high: 404; Late – low: 396, middle: 411, high: 405), and usual sleep hours (Afternoon - low: 438, middle: 434, high: 425; Traditional – low: 424, middle: 435, high: 438; Late – low: 435, middle: 432, high: 430).

[c] Dietary Guidelines Index: higher score on range of 0-100 indicates higher diet quality.

Chapter 8: Summary, future directions and conclusions

8.1 Aims of the thesis

Mood disorders are highly prevalent worldwide and can have severe negative impacts on the individual, society, and the economy. The field of nutritional psychiatry looks to how diet quality and dietary practices may support neurobiological functioning and influence mood disorders. The aim of this thesis was to determine associations between diet and mood disorder outcomes during youth and young to mid-adulthood among an Australian cohort.

In support of the primary aim, the thesis objectives were:

- Review the literature pertaining to the influence of diet on mood disorders.
- Use CDAH data to:
    - Examine cross-sectional diet quality of the cohort during youth and adulthood.
    - Determine if diet quality in youth is longitudinally associated with subsequent onset of mood disorders during adulthood.
    - Examine associations between diet quality and prevalence (cross-sectional analysis) and risk (longitudinal analysis) of mood disorders during young- to mid-adulthood.
    - Examine bidirectional associations between timing of daily food intake and mood disorders during young- to mid-adulthood.

8.2 Summary of results

The literature review identified several crucial gaps concerning longitudinal studies that have examined the influence of diet quality on mood disorder outcomes. There was limited and inconsistent evidence of a causal relationship between dietary intake and mood disorder outcomes, and very few longitudinal studies used

diagnostic measures of mood disorders. This limits the generalisability of results and consolidation of evidence due to variable definitions and severity of depressive symptoms. Only five longitudinal studies among adults (four of which were from the same study cohort) that examined overall dietary intake as opposed to individual foods or nutrients, used mood disorders that met clinical diagnostic criteria as outcome measures (135-138, 140). Of the 10 prospective studies identified involving children and adolescents that used overall measures of diet, none had a follow-up period beyond late adolescence, and only three studies (two of which were from the same cohort), used clinical diagnostic mood disorder measures (100, 104, 108).

The first original study for this thesis examined diet quality of the CDAH study cohort by developing an age- and sex- specific Dietary Guidelines Index (DGI) and validating it against a range of nutritional, physical, and sociodemographic measures among participants in youth (aged 10-15 years) and young adulthood (aged 26-36 years). The index was shown to be an adequate measure of diet quality to reflect the recommendations of the 2013 Australian Dietary Guidelines (81). Mean index scores highlighted that the dietary intake of the cohort was fairly poor at both time-points and on average did not reflect the recommended dietary intakes required to support good health (81).

The second original study examined the association between diet quality as determined by the DGI scores of participants in 1985 when they were 10-15 years of age, and the onset of mood disorders up until 2009-11 (33-41 years of age). Although there was no association between youth diet quality and mood disorders during the 25-year follow-up, there was a weak indication that participants with the poorest diet quality in youth (lowest third of DGI scores) may have had slightly worse mood disorder outcomes over the period compared to those in the middle or high third of DGI scores. There was also an indication that better diet quality may be protective against mood disorder onset in late adolescence in a dose-response relationship, although this association was not statistically significant. These results highlight the need for further prospective studies among youth, including studies that have repeat measures into young adulthood.

The third original study examined cross-sectional and longitudinal associations between diet quality and mood disorder during young- to mid-adulthood, using data from three follow-ups. In the cross-sectional analyses in mid-adulthood (CDAH-3, aged 36-49 years), better diet quality was associated with a lower prevalence of mood disorders during the 12-months prior to the follow-up ("current" mood disorder), however, the association was attenuated after adjustment for sociodemographic and lifestyle factors. The measure of self-perceived social support had a particularly strong attenuating effect. Social support is not commonly measured or adjusted for in observational studies examining associations between diet and mood disorders, but may be an important confounder or mediator. In the longitudinal analyses, diet quality was not associated with mood disorder outcomes during the follow-up period. There was also no evidence of reverse causation. Compared to participants who had never had a mood disorder, those who had a prior but not current mood disorder did not have significantly different DGI scores, while those who had current mood disorder had lower DGI scores in mid-adulthood, but the association was attenuated after covariate adjustment (reflecting the cross-sectional analysis results).

The fourth and final original study examined the relationship between the timing of daily food intake and mood disorder outcomes. This study determined three time-of-day eating patterns ("Grazing", "Traditional", and "Late") from participants' reporting of snack, meal and beverage intake during a 24-hour period on a meal chart. Three similar patterns were derived at both CDAH-1 (aged 26-36 years) and CDAH-2 five years later. Participants who had high scores at both time-points on a "Late" pattern characterised by skipped or delayed breakfast and comparatively higher intakes in the evening and at night, had twice the prevalence of mood disorder during the five year follow-up period compared to participants with low scores on the pattern at both time-points. There was evidence of reverse-causality, with mood disorder during follow-up associated with a slightly higher risk of being in a higher score third of the Late pattern compared to having no mood disorder during follow-up. Chronotype preference for evening or morning activity, influenced by genetics and circadian rhythms, was discussed as a possible trait that may shape an

individual's time-of-day eating patterns and contribute to their propensity for mood disorders.

8.3 Limitations

There are several limitations of the studies that should be considered when interpreting the results. These limitations were outlined in the relevant chapters but will be summarised here.

The dietary measures used for the determination of diet quality and time-of-day eating may have introduced error. The NDSS 24-hour food record in 1985 was intended as a snapshot of the dietary intake of Australian schoolchildren rather than representative of an individual's usual dietary intake. The large sample size reduces the margin of error in estimation of mean intakes among the population, but a more accurate measure would have been derived from taking averages of repeated 24-hour food records to remove between-day or between-season variation (60). There is also the possibility of reporting bias or modification of eating behaviours during the 24-hour period arising from participants wanting to present what they perceive to be healthier or more socially desirable diets, or to simplify the reporting process (63, 294). Social desirability reporting bias may also have biased the CDAH adult FFQ data, while the non-quantitative design of the questionnaire and difficulties for participants in averaging their usual intake of each item during the previous 12 months may have introduced further error. However, the FFQ is based on an FFQ that has been validated for use within Australian populations (163, 165). Error may also have been introduced in calculation of the DGI by the assumption that one frequency equalled one "standard serving" outlined in the 2013 Australian Dietary Guidelines, although this method has been used in previous validation of diet quality instruments (76). Moreover, the different methods of dietary measurement in the NDSS and CDAH follow-ups meant it was not possible to examine tracking of diet or dietary change from youth to adulthood as the data were not directly comparable.

Social desirability bias and lack of clarity in the layout of the 24-hour meal chart questionnaire used to determine time-of-day eating patterns could also have introduced some error. The chart lacked instructions to participants around whether they could enter multiple meals/snacks within an hourly interval, and the single

11pm-6am time interval meant that it was not possible to discriminate between a late meal or an early breakfast. Response options and meal size examples were brief, but error from between-person differences in concepts of meal or snack size were mitigated by standardising intake for each participant.

There are also several aspects around the study samples and loss-to-follow up that must be addressed. The ASHFS was designed to provide a benchmark with which to compare later studies on children and adolescents. It was not created to be a longitudinal cohort study. Although the tracing of participants in 2001-02 successfully located and enrolled 5,170 participants, varying numbers of these participants went on to participate in CDAH follow-ups (3,992 at CDAH-1; 3,038 at CDAH-2; 2,074 at CDAH-3). Moreover, not all participants completed all components at each follow-up, meaning samples for the individual studies are smaller still and vary according to which measures were used. Although the samples for the original studies comprising this thesis include participants with a range of characteristics, there was greater loss to follow-up among males, and among those who at baseline were overweight or obese, regular smokers, had poorer academic achievement reported by the school, lower area-level SES, and who were less likely to regularly eat breakfast. This participant bias is evident, for example, in the comparison in Chapter 5 of the baseline characteristics of the participants who completed the CDAH-2 mental health interview with those in the original NDSS cohort (Table 5.1). This greater loss to follow-up among participants with indicators of lower SES and less healthy lifestyle behaviours is a recognised issue for cohort studies (295). However, the samples retained participants with a wide range of characteristics. Furthermore, relevant confounding factors were adjusted for in the analyses, and a combined statistical approach of multiple imputation and inverse probability weighting was used in each of the studies (182). These delimitations would have gone some way to estimating associations to reflect the original nationally representative sample. An additional issue, however, is that the sociocultural profile of the original youth population is not representative of the Australian adult population in 2020, largely due to migration. Among the 6,586 ASHFS questionnaire participants aged 9-15 years, 13% (*n* = 850) spoke a language other than English at home and the most frequently spoken languages were Italian and then Greek (159).

Whereas results of the 2016 Census indicate that 22% of the Australian population spoke a language other than English at home and the most frequent languages were Mandarin, followed by Arabic (296). Therefore, generalisability of the study results to the broader adult Australian population is limited, and results should be interpreted as more relevant to Anglo- and European-Australian populations.

A further limitation is the time between baseline in 1985 and the first follow-up in 2004-06, meaning there was approximately a 20-year gap when no measurements were taken. This period includes the transition from childhood and adolescence into young adulthood, which was when many participants later reported their first onset of a mood disorder, as outlined in Chapter 5. There is evidence that dietary patterns can track from youth to adulthood (96), but dietary intake of participants may have undergone considerable variation after baseline due to availability of different foods and changed social and economic contexts as the participants left school, started work or tertiary study, and moved out of home (297). In terms of the retrospective mood disorder measures, although the CIDI has been shown to have good reliability as to the time-related questions (such as age of first onset, and age of most recent recurrence of symptoms) (87), the risk of recall bias may have been reduced by having an earlier measure of lifetime mood disorder than at CDAH-2 when participants were between 31-41 years of age.

As with all epidemiological studies, there is the possibility of residual or unmeasured confounding where variables that have not been considered, measured or adjusted for could explain or obscure the true association between predictor and outcome variables. The wide range of measures collected for the ASHFS and CDAH follow-ups allowed good consideration of potential confounders and model adjustment, but there are some unmeasured lifestyle and socioeconomic factors that may have been important in the analyses. For example, at CDAH-1, parental status for male participants was not collected (female participants were asked about live births), and therefore could only be imputed for CDAH-1 males who participated in CDAH-2. Possibly relevant to the time-of-day eating patterns study in Chapter 7, usual nightly sleep duration and work schedule (e.g. regular hours, overnight shifts, casual work) were also not captured at CDAH-1. Pertaining to the development of mood

disorders, stressful life events such as death of close friends or family, relationship breakdown, or financial difficulties that have physical and psychological impacts on the individual may have been positive confounders that further attenuated the few observed associations, but these were only measured at CDAH-2 and only for events during the past five years. The primary focus of the CDAH study on cardiometabolic health outcomes and subsequent design and inclusion of measures as covariates to support those primary analyses, means that the data available for secondary analyses may not be ideal.

8.4 Public health implications

The findings of this thesis have several important public health implications outlined below.

8.4.1 Improving diet quality

The development and application of the DGI highlighted poor diet quality among the cohort participants in both youth and adulthood. This is consistent with other studies in Australia and internationally that have reported poor adherence to dietary guidelines (298-300). Scores were particularly low in youth, although this could reflect food cultures at the time and the single 24-hour snapshot measure, as outlined in Section 8.3 above.

Australian Government messaging around what constitutes a healthy and nutritious diet has been broadly consistent in Australia over the last 30-40 years. The first edition of the Australian Dietary Guidelines was issued in 1982 and outlined a nutritious diet as one with a variety of foods from the five food groups of breads/cereals, vegetables and fruits, meat and meat alternatives (e.g. lentils, nuts), milk and milk products, and butter/table margarine (to be limited to one tablespoon per day) (204). People were also advised to control their weight via exercise and limit (but not necessarily avoid) discretionary foods such as jams, biscuits, cake, confectionary, and potato crisps (204). At the time of the youth study in 1985, increases in obesity and over-consumption of energy dense foods of low micronutrient value were an emerging concern, as evidenced by the rationale for the ASHFS and NDSS (192). During subsequent decades there was further

development of the Australian Dietary Guidelines based on empirical scientific evidence (81). However, despite government mass media campaigns regarding healthy diets and physical activity in different states and territories since the 1980s (301, 302), rates of obesity and cardiometabolic disease in Australia have become increasingly pressing public health issues (301).

In recent years there have been several initiatives to make consumers more cognisant of the nutritional value of the foods they choose. The Australian Government's food health star rating system, implemented on a voluntary basis since June 2014, provides a rating out of five stars (calculated on the nutritional and fibre content of the item) on the front of packaged food. However, the system remains voluntary and has been subject to considerable resistance from some sectors of the food industry (303, 304). Meanwhile, New South Wales, Victoria, Queensland, South Australia and the Australian Capital Territory have regulated point-of-sale nutritional labelling for fast food restaurant chains, but there is no nationwide consistency yet despite recommendations (305). Nutritional menu labelling has been regulated nationally in the United States and has been estimated to have significant population health gains and healthcare savings due to consumer response and the reformulation of menu items to healthier options (306).

The results in Chapter 5 provided weak indications of poorer mood disorder outcomes over the follow-up period for participants in the lowest third of baseline youth DGI scores. Prior studies have suggested that poor diet in youth could have long lasting effects due to inadequate nutrition or prolonged inflammatory responses that impact on the neurobiological processes that support good brain functioning and mental health (25, 219). Poor eating practices may also carry over into adulthood (96). The Australian Government has formalised food and nutrition content in the national school curriculum (302), and developed guidelines to encourage a nationally consistent approach to promoting healthy foods in school canteens (302, 307). These are positive steps, but teaching of content and associated programs such as school gardens and developing cooking skills, have been found to be ad hoc and comprise a low proportion of the curriculum (308). Furthermore, educational programs need to engage families and the broader

community. The home food environment and parental values and behaviour is crucial to complement and provide consistency around healthy eating messaging to children and adolescents (309, 310). There is also continuing advocacy by concerned health organisations for further actions such as government controls on junk food advertising to children, including via social media, sponsorship of sports, and school fundraising (311).

Education and public health campaigns can increase understanding of nutritious foods or how to prepare them, but there are a range of other factors that contribute to poor quality diets. These include time pressures, living environments, economic factors, confusing media messages from different sources (exacerbated in the past decade due to social media), and the increasingly market-driven availability of processed and packaged snack and convenience foods that are both tasty and cheap (302, 312). The effects of socioeconomic disadvantage also need to be considered as diet quality often follows a social gradient - better diet quality has been consistently associated with better markers of socioeconomic status and education (208, 299). To support education and information campaigns so that individuals can make healthy food choices, action is needed on multiple aspects of the food environment, including greater government and industry regulation and controls around the food, retail, and advertising industries. Significant barriers to achieving food environment regulation include political and ideological climates that prioritise the free market, focus on short term over long term gains, effectiveness of the bureaucratic administration responsible for implementation, resourcing, and industry pressure from national and international bodies who's interests may lie more with financial return than population health (313).

8.4.2 Time-of-day eating, circadian rhythms, and food choices.

The derivation of three time-of-day eating patterns in Chapter 7 was a novel finding that could provide insight into how and when people eat. The association between the "Late" pattern and mood disorder prevalence has public health implications as it provides further evidence of poor health outcomes associated with skipping or delaying breakfast, additional to previously reported associations with cardiometabolic health risks (314, 315). However, the study suggests that the

relationship between a Late eating pattern and mood disorder outcomes may involve reverse causality or be bidirectional. A possible mechanism behind this could be chronobiology influenced by genes and environmental cues, manifesting as chronobiological traits (being a “morning” or “evening” person) (316). There is consistent evidence that compared to morning chronotypes, evening chronotypes tend to have poorer diet quality and higher consumption of kilojoules (particularly at later hours) (146, 317). The evening chronotype has also been linked to poorer health outcomes including mood disorders, substance abuse, obesity, reduced insulin sensitivity, and difficulties related to sleep disturbances (318). Although evidence from human rather than animal studies is limited, the timing of food intake is understood to be a time cue that affects the circadian system and may be an important for good sleep habits to help support good health (319, 320). The circadian disturbance experienced by evening chronotypes in trying to fit into regular social and business hours, could also affect shift-workers with irregular work hours. In Australia, the number of employees (not including owner operators) engaged in shift work (regular shifts, rotating shifts, split shifts, irregular hours) has increased in recent years, from 1.7 million Australians in 2015 to 1.9 million in 2019 (321). Shift work has been associated with low quality diet, irregular eating patterns, poor lifestyle behaviours, and increased health risks (322).

As more evidence becomes available as to the negative health effects of disrupted circadian cycles, workplaces may have more of a responsibility to educate and support their employees on the health risks of shift work. The Safe Work Australia and state government work safety bodies have extensive information on reducing risks of fatigue related to shift work, but information on other associated health risks is generally limited. For example, the Worksafe Victoria guide to preventing fatigue in the workplace acknowledges the long term health risks associated with shift work, but provides no guidance on these risks apart from stating that workplace training or information should be given on “nutrition, fitness and health issues relating to fatigue”, and the only nutritional references relate to use of stimulants, such as “Do not consume coffee or tea before going to bed” (323). A notable exception is Workplace Health and Safety Queensland who provide detailed information and advice regarding healthy lifestyle choices for shift workers, including a pamphlet for

employers about the importance of good nutrition in shift work environments (324, 325).

Alongside the increase in shift work, societal change to a more global 24-hour society means that adhering to traditional western notions of breakfast, lunch, and dinner occurring at set times and comprising certain types of food may also change. The move away from the centralised 9:00am to 5:00pm office or retail environment to a more dispersed and time-flexible business environment may have ramifications for time-of-day eating patterns in the future. Therefore, to separate the effects of timing of eating occasions from quality of intake, the focus on healthy eating (whether by governments, health bodies, or workplaces), should be on planning ahead and encouraging healthy food choices for all meals of the day.

8.4.3 Health promotion to reduce mood disorder risk

The aim of this thesis was to determine whether there were independent effects of the quality and timing of dietary intake on mood disorder outcomes. However, food choices and dietary behaviours are shaped and influenced within a social and economic context. There are numerous environmental, physical, and social factors that could contribute to the development of mood disorders and trajectories of the conditions. As shown in Chapter 6, the covariates important to the model differed by sex and at each follow-up, and self-perceived social support had a particularly strong effect on the prevalence ratio estimate. The previous two sections have highlighted that education and information to better equip individuals with health knowledge needs to be accompanied by the social and economic environment that will support them in making better lifestyle choices based on that knowledge. Therefore, public health initiatives to address mood disorders must enact change at both the individual and societal level.

A health promotion model that focuses on strengthening the individual to cope with challenges is the salutogenic model (“salus” meaning health, and “genesis” meaning origin). This contrasts to a biomedically defensive health promotion model, which focuses on preventative measures and highlights risks related to the individual to motivate change (326). The salutogenic model of health promotes good health habits and learning instead of focusing on health risks, and these improved health

behaviours move the individual towards good health (327). The initiatives for improving diet quality of populations outlined in the previous sections in terms of education, consistent messaging, and reducing environmental barriers to make healthy food choices, falls under this salutogenic approach. Underpinning the salutogenic model is the concept of sense of coherence (resilience) - the ability of an individual to understand, manage, and make sense of a situation to deal with health issues or challenges to move towards good health. For example, sense of coherence has been associated with healthier eating patterns among a Swedish adult cohort (328), greater intake of fruit, vegetables and fibre among a UK cohort (329), and has been suggested as a possible link between healthier diet and lower likelihood of mental disorders among an Australian population (249). There are indications that sense of coherence is shaped by socioeconomic status and therefore health interventions that are targeted to remove socioeconomic barriers and reduce socioeconomic vulnerabilities, particularly in early life, are important for developing good health and good health behaviours (330).

Enacting broader societal change to support the individual, entails the ecological framework where health outcomes are determined by interactions between factors at different levels (331, 332):

- The individual - e.g. biology, personality, behaviour, personal history.
- Interpersonal relationships - e.g. families, friends, social networks.
- Community and institutions - e.g. school, work, neighbourhood.
- Society – e.g. economic and social policy, cultural norms.

This ecological framework entails a biopsychosocial model of health as an intersect of biological, psychological, and social factors, rather than a strictly biologic outcome (333). Even if diet quality is determined to have an independent effect on mood disorders through biological pathways, this must still be understood within the context of the environment and decisions that shape dietary intake. The social gradient of diet quality, and associations of socioeconomic and psychosocial factors with mood disorders (191, 334, 335), mean that the ecological approach is critical to address these confounding and mediating factors. In Australia, the ecological

approach has informed various public health reports and policies in which social determinants of health are acknowledged, but not always addressed via action (336). The Australian Productivity Commission's 2019 review of mental health programs in Australia identified that a key factor driving poor outcomes in Australia was "…a focus on clinical services which often overlooks other determinants of, and contributors to, mental health", and that investment in services beyond health e.g. housing, is a required area of reform (337).

Strengthening individuals and communities, including targeted social support strategies to encourage healthy lifestyles, and reducing social and environmental barriers to good mental health has been highlighted by the WHO as crucial to public health initiatives around mental health (338). To reduce the prevalence and impact of mood disorders in Australia, a holistic approach is needed to build resilience among individuals, and identify and improve on a nationwide level the broader social, environmental and economic structures that contribute to unhealthy lifestyle choices, lack of social resources, and mood disorder risk.

8.5 Future directions

Consistent evidence of associations between dietary intake and mood disorders is required to support further development of dietary guidelines, public health messaging and government regulation and policy. This could include prospective observational studies, ideally beginning in childhood prior to first onset of mood disorder and extending over adequate durations to capture data during several life stages. The studies should involve:

- Regular follow-ups and repeat measures to capture dietary, sociodemographic, physical, and psychosocial change, particularly relevant for different life stages and periods of change such as the transition from adolescence into adulthood.
- Consistent dietary measurement methods to allow examination of dietary change between follow-ups, and long-term diet. Repeat dietary recall or dietary record methods would capture versatile data for determining both quality of usual dietary intake, and timing of daily eating occasions.

- Assessment of diet quality against commonly used and recommended pillars of a healthy diet such as adequate intake of whole foods (fruits, vegetables, wholegrains, lean proteins, dairy or alternatives) and limited consumption of processed foods with added sugars, salt and saturated fats. This will aid in interpretation of results, generalisability to other populations, and contribution to meta-analyses.
- Structured diagnostic interviews and standard definitions of mood disorders (e.g. DSM or ICD) to identify cases at baseline and examine subsequent first onsets and recurrences.

Additional to observational cohort studies, high-quality prevention and intervention randomised control trials are also required to determine the feasibility of firstly improving dietary intake, and secondly, determining if this has positive effects on risk of mood disorder onset or recurrence. For example, the 2017 Australian "SMILES" trial, a 12 week dietary intervention, reported a reduction in depressive symptoms among the 56 adult participants who completed the trial, but acknowledged the preliminary nature of the study and that more extensive and larger trials are required (339). A 2019 three-week randomised control trial that gave instructions on healthy eating and provided basic foods and some financial reimbursement for groceries to the 38 young adults in the intervention group (not provided to the control group), also showed promising results in reducing depressive symptoms (340). However, this trial was brief with a small sample, and although follow-up extended to three months, follow-up was among the intervention group only (340). Future trials should be designed with post-trial follow-ups to examine whether dietary change is sustained after regular trial contact and support ends, and what the health outcomes are over the long-term.

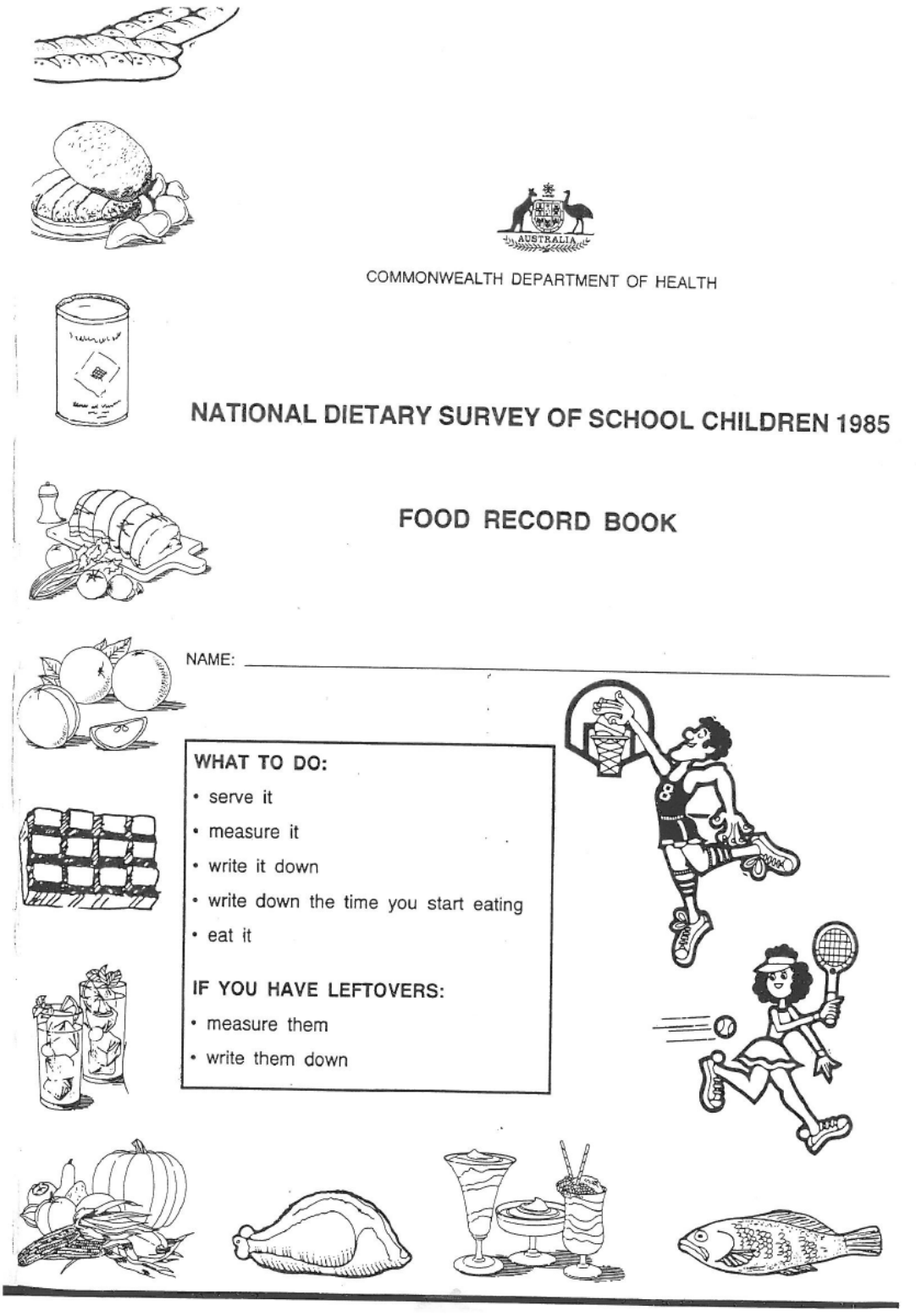
For both observational and trial studies, the study design must carefully consider the inclusion of potential confounding and covariate measures based on up-to-date evidence. This is crucial to determine if diet has an independent effect. For example, social support, as highlighted in Chapter 6, may be a potential confounder or mediator but has not been measured or adjusted for in many previous studies. Discerning the physical and neurobiological effects of diet and nutrition from the

influence of environmental, social context and life events remains a key challenge in nutritional psychiatry. The technique of Mendelian randomisation in which genetic variants are used as proxies for environmental exposure, offer alternatives to randomised control trials to determine causality of the diet-disease relationship and can mitigate issues around residual confounding and bias (341). Although there has been some initial studies on the application of Mendelian randomisation in nutritional epidemiology in relation to mental health outcomes (342), there are several limitations that mean it may not be suitable for use in diet-depression studies. These limitations include that the genetic marker must be reliably associated with the outcome of interest, the effects of the marker acts only via the intermediate exposure (i.e. diet), and that there is no genetic confounding (341, 343). Whatever the approach used, it is also crucial that studies of sufficient quality with negative results where no association is observed are reported, as these can provide important insights into populations, methods, and confounders.

8.6 Conclusion

The findings of this thesis indicate that on average, diet quality among the study cohort was poor and highlights the need for continued work to encourage and support healthier food choices, beginning in youth. This thesis provides an important tool for researchers in the validated age- and sex-specific Dietary Guidelines Index for use among children and adults. The index could support investigations between diet quality and health over the life-course. Although there was no clear evidence of an association between diet quality and mood disorder outcomes among the cohort, weak indications of a relationship between better diet quality and lower likelihood of mood disorders, and the associations between time-of-day eating patterns and mood disorders, highlight the need for continued research on the topic.

Appendix A: National Dietary Survey of Schoolchildren 24-hour food record



COMMONWEALTH DEPARTMENT OF HEALTH

NATIONAL DIETARY SURVEY OF SCHOOL CHILDREN 1985

FOOD RECORD BOOK

NAME: _____

WHAT TO DO:

- serve it
- measure it
- write it down
- write down the time you start eating
- eat it

IF YOU HAVE LEFTOVERS:

- measure them
- write them down

HOW TO USE THIS BOOK

You have been given this book to measure and write down everything you eat and drink, including water, for the next 24 hours. Special pages are provided in the centre of this book for this purpose.

Do not record medicines or tablets, vitamins or mineral pills.
Do not record salt, pepper, herbs and spices.

Keep this book with you at all times so you can measure and write down foods and drink as soon as you have them.

Read the instructions before you begin and follow them carefully.

Remember to bring your food record book back on the next school day. The interviewer will check that the description of all foods and drink you have had is complete.

HOW TO FILL IN YOUR RECORD

- Use a blue or black pen when writing down everything you eat and drink.
- You will need to fill in three columns.

Column 1: Time

- Every time you have something to eat or drink, write down the time you started.
- Write down a.m. for morning and p.m. for afternoon or evening.

Column 2: Name and full description of all food and drink

- For each food or drink use a new line.
- You may use more than one line to describe your food.
- Write down the type and name of foods and when possible, the brand name. For example,
  Arnotts milk arrowroot biscuit
  Tip Top white bread
  Lamb, loin chop
  Whole milk
- If the food was coated, write down if it was coated in flour, crumbs or batter.
- If the food was cooked, write down how it was cooked. For example grilled, fried, boiled.
- Write down if the fat on meat was eaten or not eaten.
- Write down if the skin on poultry was eaten or not eaten.
- If the dish is a mixture of several foods, name the dish, and all the separate ingredients you can see in it or know are there. Examples of food mixtures include stew, soup, coleslaw and mashed potato. (See chicken stew and mashed potato on the example page).
- For sandwiches and rolls, measure each ingredient as it is being made, if you can. If the sandwich is already prepared, name each ingredient and the number of sandwiches or rolls eaten (1 sandwich has 2 slices of bread).
- Remember to record foods added to other foods such as milk or sugar in tea and coffee; gravy, sauce or pickles on meat or in sandwiches; cream or icecream on dessert; flavourings such as icecream, topping or Milo in milk drinks.

Column 3: Amount eaten

- Measure and write down the amount you eat and drink.
- Measure the amount using the special cups and spoons, your ruler and the circles on pages 10 and 11.
- Do not weigh your food.
- For a dish that is a mixture of several foods, write down the total amount of the mixed dish eaten. There is no need to record the amount of each separate ingredient.
- For meat and chicken, write down if the measurements taken are with bone or without bone and with or without fat.

24 HOUR FOOD RECORD

• Only use a **blue** or **black** pen to write.

From Time: 10.00 Day: Thursday Date: 22 August

To Time: 10.00 Day: Friday Date: 23 August

• Write down the time you start eating each food.
• Write down **am** or **pm**.

1 Time	2 Name, type, brand, cooking method (Follow the instructions on page 3 to describe your food and drink.)	3 Amount eaten (Follow the instructions on pages 10 and 11 on how to measure what you eat and drink.)
6.00pm	green peas, boiled	1 cup
6.00pm	potato, boiled	1 potato, size D
6.00pm	hamburger patty, made with beef mince, grilled	1 hamburger patty
		7cm, 1cm thick
6.00pm	pumpkin, boiled	1 piece of pumpkin 5cm × 6cm × 4cm
10.00am	Coca-Cola	1 can 375ml ½cup left
11.30am	peanut paste sandwich, Tip Top white bread	2 slices each 11cm × 11cm × 1cm
11.30am	Kraft peanut paste	2 tablespoons
11.30am	Flora margarine	2 teaspoons on each slice of bread
11.40am	lemon cordial drink, made with water	1 cup + ½cup
1.10pm	MacDonalds potato chips, fried	20 chips each 6cm × 2cm × 1cm
7.00pm	chicken stew: chicken meat, skin eaten, peas, carrots, onion,	1 cup + 3 tablespoons, without bone
	gravy	
7.00pm	mashed potato with whole milk and Meadow Lea margarine	2 cups, ½cup left
	added	
7.30pm	loin lamb chop, grilled, fat eaten	2 chops each 3cm thick, 12cm long, with bone and fat, 5½cm wide

Do not write in these columns.

Rec. No.	Time	Food code	Weight
01			
02			
03			
04			
05			
06			
07			
08			
09			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

24 HOUR FOOD RECORD

• Only use a **blue** or **black** pen to write.

From Time: ____ Day: ____ Date: ____

To Time: ____ Day: ____ Date: ____

• Write down the time you start eating each food.
• Write down **am** or **pm**.

Follow the instructions on page 3 to describe your food and drink.

Follow the instructions on pages 10 and 11 on how to measure what you eat and drink.

Do not write in these columns.

1 Time	2 Name, type, brand, cooking method	3 Amount eaten	Rec. No.	Time	Food code	Weight
			01			
			02			
			03			
			04			
			05			
			06			
			07			
			08			
			09			
			10			
			11			
			12			
			13			
			14			

24 HOUR FOOD RECORD

• Only use a **blue** or **black** pen to write.

From Time: _____ Day: _____ Date: _____

To Time: _____ Day: _____ Date: _____

☐ ☐

• Write down the time you start eating each food. • Write down **am** or **pm**.	Follow the instructions on page 3 to describe your food and drink.	Follow the instructions on pages 10 and 11 on how to measure what you eat and drink.	Do not write in these columns.			
1 Time	2 Name, type, brand, cooking method	3 Amount eaten	Rec. No.	Time	Food code	Weight
			01			
			02			
			03			
			04			
			05			
			06			
			07			
			08			
			09			
			10			
			11			
			12			
			13			
			14			

HOW TO MEASURE YOUR FOOD

- If your food is cooked, measure it cooked, not raw.
- Measure the food after it is served.
- If you do not eat all the food you first served out, measure and write down how much of each food is left.
- There is no need to measure left over cores, peel or fish bones.

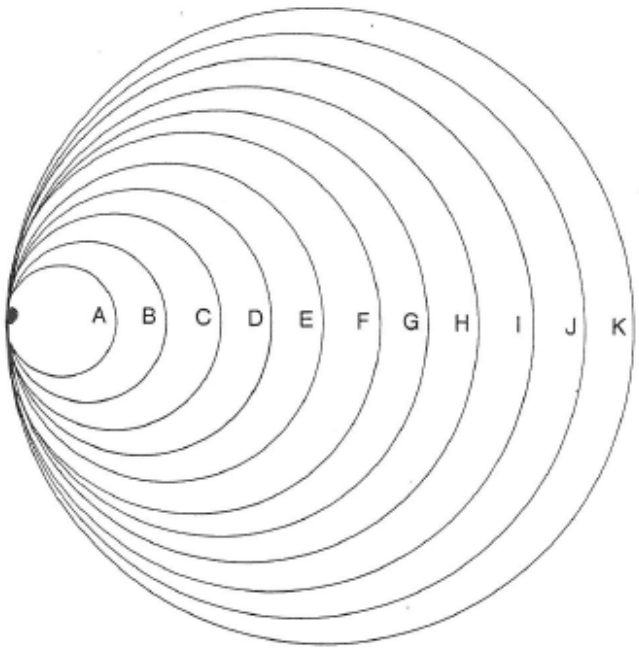
Measure your food using:

Cups and spoons

- Use the special cups and spoons to measure liquid foods and foods like cereal, sugar, stews, rice and vegetables.
- Only use the special cups and spoons provided.
- Measure as level, never heaped.

Circles

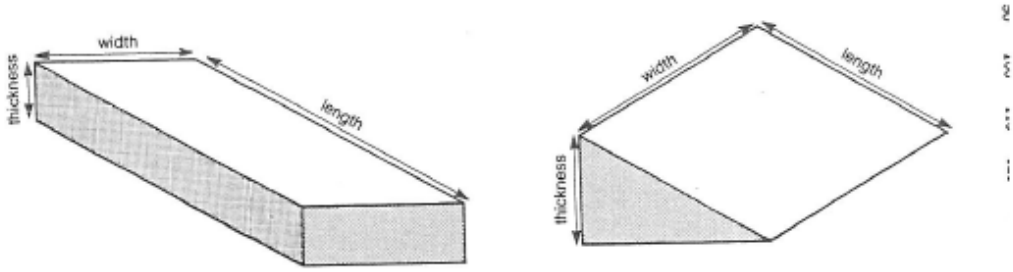
- Use the circles to measure foods which are shaped like a ball for example apples, tomatoes, oranges, round bread rolls.
- Put one edge of the food on the black dot, look down from the top and choose which is the closest fit.
- Round flat foods like hamburgers and biscuits may also be measured using the circles or by measuring how wide it is across with your ruler. You also need to measure the thickness of these foods with your ruler.

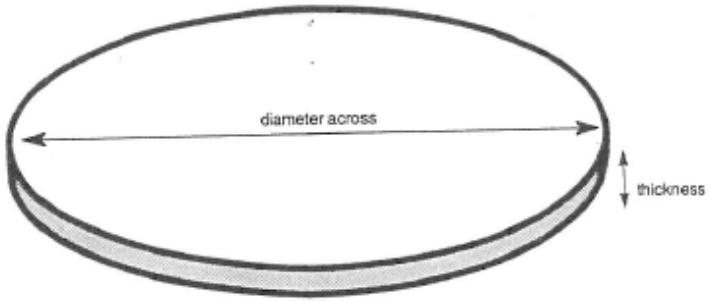


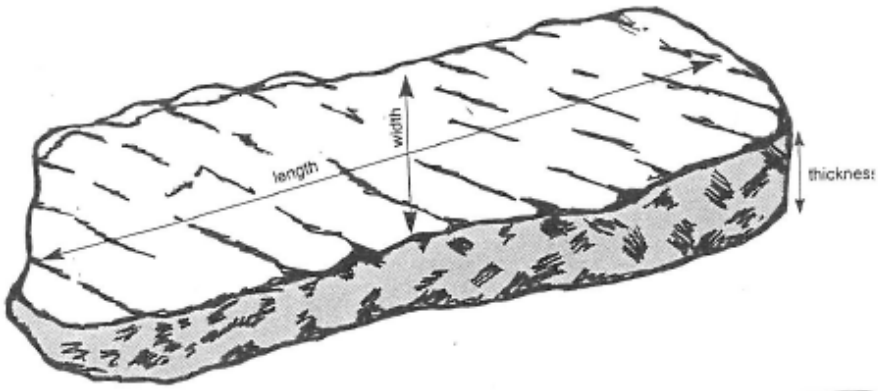
Ruler

Where cups, spoons and circles won't work, use a ruler.
Examples of foods to measure this way are pieces of meat, cake, bread.

- For round, flat foods, measure how wide it is across and the thickness.
- For foods of other shapes, measure how **long** by how **wide** by how **thick** it is.
- Draw a small picture to help you describe the shape of food and show where the food was measured.
- Use your school ruler to measure your food. If you do not have it with you use the ruler given on this page.







Appendix B: Childhood Determinants of Adult Health dietary questionnaire

The following example questionnaire was used during the first Childhood Determinants of Adult Health follow-up in 2004-06. The questionnaires administered at the 2009-11 and 2014-19 follow-ups had minor changes:

- Additional food items were included in Section A (as detailed in Chapter 3, Section 3.4.3).
- In Section B:
    - Free text fields were added for respondents to specify further detail when they selected “Other” for Question 7 (usual way of eating) and Question 9 (type of spread usually used).
    - Questions 16 and 17 were removed and a question about eating breakfast was added – “In the last 7 days, on how many days did you eat breakfast?”, with seven response options (1-7) provided.
- In Section C, respondents were additionally asked “If you currently have a paid job, did you work yesterday?”, with response options of “Yes”, “No”, “Not applicable (no current paid job)”. Those who answered “Yes” were asked “What time did you start work yesterday?” and “What time did you finish work yesterday?”, and instructed to fill out the time in hours and minutes and specify if each time was AM or PM.

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(Clinic staff attach bar code here)

HOW TO COMPLETE THIS SECTION:

For each food item listed, circle the box that best represents your average pattern of consumption of that food over the previous 12 months.

For example:

If you usually eat baked beans for breakfast every day, fill in *"Once per day"* (see example below). If, however, you never eat baked beans, fill in "*Never, or less than once a month*"

FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
Baked beans	○	○	○	○	○	●	○	○	○

Mixed foods

Some commonly consumed mixed foods, such as salads, stir-fried vegetables etc have been included as distinct items. Other foods such as sandwiches are not listed as distinct items as their composition varies depending on how they are made up. For these foods think about the separate ingredients and answer accordingly.

For example:

- If you usually eat a ham and mixed salad sandwich once a week and you usually eat no other ham or mixed salad during the week, fill in the "*once per week*" box for **ham** and the *"once per week"* box for **green/mixed salad**. Don't forget to include the bread/roll in your breads total either!

Seasonal foods

There are some foods that you eat only when they are in season. For very seasonal fruits such as stone fruits, melons etc you should estimate your average consumption when the fruits are in season.

For example:

- If you eat fresh plums once a week during summer, and eat no plums for the rest of the year you should fill in the box for *"once per week"*

- If you eat fresh plums once a week during summer, and eat canned plums once a week for the rest of the year you should also fill in the box for *"once per week"*

Important points to remember

- - Please fill in one response for every food listed.
  - If you never eat a particular food, fill in the "*Never, or less than once a month*" box.
- - Think about how often you eat take away or restaurant prepared foods.
- - If you make a mistake, put a cross X through the wrong answer, and fill in the correct answer

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2

SECTION A : This section of the questionnaire is designed to estimate your usual pattern of food intake by providing us with information on your average consumption of certain foods and beverages during the last 12 months

BEFORE STARTING THIS QUESTIONNAIRE, PLEASE MAKE SURE THAT YOU HAVE READ THE INSTRUCTIONS ON THE PRECEEDING PAGES

For each food item listed, indicate how often **on average** you consumed that food in the **past 12 months**. Please fill in one circle for each food listed, even if you **never** eat it.

FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
1. DAIRY FOODS									
Flavoured milk drink (e.g milkshake, iced coffee, hot chocolate)	○	○	○	○	○	○	○	○	○
Milk as a drink	○	○	○	○	○	○	○	○	○
Milk in hot beverages (e.g. in coffee, tea)	○	○	○	○	○	○	○	○	○
Milk added to breakfast cereal	○	○	○	○	○	○	○	○	○
Cream or Sour Cream	○	○	○	○	○	○	○	○	○
Ice-cream	○	○	○	○	○	○	○	○	○
Yoghurt, plain or flavoured (including fromage frais)	○	○	○	○	○	○	○	○	○
Cottage or ricotta cheese	○	○	○	○	○	○	○	○	○
Cream cheese (e.g. Philadelphia™)	○	○	○	○	○	○	○	○	○
Cheddar and other cheeses	○	○	○	○	○	○	○	○	○
2. BREAD & CEREAL FOODS									
White bread,toast or rolls	○	○	○	○	○	○	○	○	○
Wholemeal/mixed grain bread, toast or rolls	○	○	○	○	○	○	○	○	○
English muffin, bagel or crumpet	○	○	○	○	○	○	○	○	○
Flat bread (e.g. pita,chapatti)	○	○	○	○	○	○	○	○	○
Dry or savoury biscuits, crispbread, crackers	○	○	○	○	○	○	○	○	○
Muesli	○	○	○	○	○	○	○	○	○
Cooked porridge	○	○	○	○	○	○	○	○	○
Breakfast cereal	○	○	○	○	○	○	○	○	○
Rice (white or brown)	○	○	○	○	○	○	○	○	○
Pasta (including filled), noodles	○	○	○	○	○	○	○	○	○

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
3. MEAT,FISH,EGGS									
Mince dishes (e.g. rissoles, meatloaf)	O	O	O	O	O	O	O	O	O
Mixed dishes with beef, veal, lamb, pork (e.g. casserole,stir fry)	O	O	O	O	O	O	O	O	O
Beef, veal- roast,chop or steak	O	O	O	O	O	O	O	O	O
Lamb-roast,chop	O	O	O	O	O	O	O	O	O
Pork-roast,chop	O	O	O	O	O	O	O	O	O
Ham/bacon	O	O	O	O	O	O	O	O	O
Luncheon meats,salami	O	O	O	O	O	O	O	O	O
Sausages (beef,pork,other)	O	O	O	O	O	O	O	O	O
Liver (including pate)	O	O	O	O	O	O	O	O	O
Other offal (e.g. kidneys)	O	O	O	O	O	O	O	O	O
Mixed dishes with chicken, duck, turkey (e.g. casserole,stir-fry)	O	O	O	O	O	O	O	O	O
Chicken,turkey,duck- roast,steamed or barbequed	O	O	O	O	O	O	O	O	O
Canned fish (e.g. tuna, salmon, sardines)	O	O	O	O	O	O	O	O	O
Fresh fish- steamed,baked,grilled	O	O	O	O	O	O	O	O	O
Frozen fish- steamed,baked,grilled	O	O	O	O	O	O	O	O	O
Fish,fried	O	O	O	O	O	O	O	O	O
Mussels/oysters	O	O	O	O	O	O	O	O	O
Lobster/crayfish/yabbies	O	O	O	O	O	O	O	O	O
Calamari/squid	O	O	O	O	O	O	O	O	O
Prawns	O	O	O	O	O	O	O	O	O
Other seafood	O	O	O	O	O	O	O	O	O
Egg	O	O	O	O	O	O	O	O	O

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months: Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
4. SWEETS, BAKED GOODS AND SNACKS									
Cakes, sweet muffins, scones or pikelets	○	○	○	○	○	○	○	○	○
Sweet pies or sweet pastries	○	○	○	○	○	○	○	○	○
Other pudding or desserts	○	○	○	○	○	○	○	○	○
Plain, sweet biscuits	○	○	○	○	○	○	○	○	○
Cream, chocolate biscuits	○	○	○	○	○	○	○	○	○
Meat pie, sausage roll or other savoury pastries	○	○	○	○	○	○	○	○	○
Pizza	○	○	○	○	○	○	○	○	○
Hamburger	○	○	○	○	○	○	○	○	○
Chocolate (including chocolate bars e.g. Mars bar™)	○	○	○	○	○	○	○	○	○
Other confectionary	○	○	○	○	○	○	○	○	○
Potato chips, corn chips, Twisties™ etc.	○	○	○	○	○	○	○	○	○
Almonds, walnuts, hazelnuts	○	○	○	○	○	○	○	○	○
Cashews	○	○	○	○	○	○	○	○	○
Coconuts	○	○	○	○	○	○	○	○	○
Peanuts	○	○	○	○	○	○	○	○	○
Pistachio	○	○	○	○	○	○	○	○	○
Seeds-pumpkin, sesame, pine nuts, tahini	○	○	○	○	○	○	○	○	○
Other nuts, seeds	○	○	○	○	○	○	○	○	○
5. DRESSINGS AND SPREADS									
Oil and vinegar dressing	○	○	○	○	○	○	○	○	○
Mayonnaise or other creamy dressings	○	○	○	○	○	○	○	○	○
Jam, marmalade, syrup or honey	○	○	○	○	○	○	○	○	○
Peanut butter, other nut spreads	○	○	○	○	○	○	○	○	○
Vegemite™, Marmite™ or Promite™	○	○	○	○	○	○	○	○	○
Creamy dips and spreads	○	○	○	○	○	○	○	○	○

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
6. NON-DAIRY									
Fruit juice (100%)	O	O	O	O	O	O	O	O	O
Vegetable,tomato juices	O	O	O	O	O	O	O	O	O
Fruit juice drink or fruit cordial	O	O	O	O	O	O	O	O	O
Low-joule cordial	O	O	O	O	O	O	O	O	O
Cordial	O	O	O	O	O	O	O	O	O
Low-joule soft drink	O	O	O	O	O	O	O	O	O
Soft drinks (including flavoured mineral water)	O	O	O	O	O	O	O	O	O
Water (including unflavoured mineral water, soda water,tap water)	O	O	O	O	O	O	O	O	O
Coffee-full strength	O	O	O	O	O	O	O	O	O
Decaffeinated coffee	O	O	O	O	O	O	O	O	O
Tea- green or black (ordinary tea)	O	O	O	O	O	O	O	O	O
Herbal tea	O	O	O	O	O	O	O	O	O
Soy beverages	O	O	O	O	O	O	O	O	O
Light beer	O	O	O	O	O	O	O	O	O
Medium strength beer	O	O	O	O	O	O	O	O	O
Full strength beer	O	O	O	O	O	O	O	O	O
Red wine	O	O	O	O	O	O	O	O	O
White wine or champagne/sparkling wine	O	O	O	O	O	O	O	O	O
Wine cooler	O	O	O	O	O	O	O	O	O
Spirit-based mixed drinks (eg Lemon Ruski™)	O	O	O	O	O	O	O	O	O
Sherry/port/ fortified wines	O	O	O	O	O	O	O	O	O
Spirits, liquers	O	O	O	O	O	O	O	O	O
Other alcoholic drinks (e.g cider)	O	O	O	O	O	O	O	O	O

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
7. VEGETABLES (Including frozen and canned)									
Green/mixed salad (including lettuce, tomato etc.) in a sandwich	○	○	○	○	○	○	○	○	○
Green/mixed salad (including lettuce, tomato etc.) as a side-salad with a main meal	○	○	○	○	○	○	○	○	○
Stir-fried or mixed vegetables	○	○	○	○	○	○	○	○	○
Vegetable casserole	○	○	○	○	○	○	○	○	○
Potato- boiled, mashed or baked	○	○	○	○	○	○	○	○	○
Hot chips/roast potatoes/potato wedges	○	○	○	○	○	○	○	○	○
Sweet potato	○	○	○	○	○	○	○	○	○
Pumpkin	○	○	○	○	○	○	○	○	○
Peas (including snow peas)	○	○	○	○	○	○	○	○	○
Green beans	○	○	○	○	○	○	○	○	○
Silverbeet / spinach	○	○	○	○	○	○	○	○	○
Broccoli	○	○	○	○	○	○	○	○	○
Cauliflower	○	○	○	○	○	○	○	○	○
Brussel sprouts, cabbage, coleslaw	○	○	○	○	○	○	○	○	○
Carrots	○	○	○	○	○	○	○	○	○
Mushrooms	○	○	○	○	○	○	○	○	○
Capsicum	○	○	○	○	○	○	○	○	○
Sweetcorn, corn on the cob	○	○	○	○	○	○	○	○	○
Zucchini, eggplant, squash	○	○	○	○	○	○	○	○	○
Cucumber	○	○	○	○	○	○	○	○	○
Tomatoes (except when in mixed salad)	○	○	○	○	○	○	○	○	○

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months								
	Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
7. VEGETABLES (Including frozen and canned) contd.									
Lettuce (except when in a 'mixed salad')	○	○	○	○	○	○	○	○	○
Celery (except when in a 'mixed salad')	○	○	○	○	○	○	○	○	○
Onion or leek	○	○	○	○	○	○	○	○	○
Soy beans, tofu	○	○	○	○	○	○	○	○	○
Baked beans	○	○	○	○	○	○	○	○	○
Other beans, lentils	○	○	○	○	○	○	○	○	○
8. FRUITS (*Remember instructions for seasonal fruits*)									
Fruits - dried,frozen, canned	○	○	○	○	○	○	○	○	○
Fresh fruit salad	○	○	○	○	○	○	○	○	○
Apple or pear	○	○	○	○	○	○	○	○	○
Orange, mandarin, grapefruit	○	○	○	○	○	○	○	○	○
Banana	○	○	○	○	○	○	○	○	○
Peach, nectarine, plum or apricot	○	○	○	○	○	○	○	○	○
Mango or paw paw	○	○	○	○	○	○	○	○	○
Pineapple	○	○	○	○	○	○	○	○	○
Grapes or berries	○	○	○	○	○	○	○	○	○
Melon (watermelon, rockmelon or honeydew melon)	○	○	○	○	○	○	○	○	○
Other fruit not listed	○	○	○	○	○	○	○	○	○

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FOOD PRODUCT LIST	Average number of times consumed in the last 12 months: Never or less than once a month	1-3 times per month	Once per week	2 - 4 times per week	5 - 6 times per week	Once per day	2 - 3 times per day	4 - 5 times per day	6+ times per day
9. VITAMIN and MINERAL SUPPLEMENTS									
Multivitamin with iron or other minerals	○	○	○	○	○	○	○	○	○
Multivitamin	○	○	○	○	○	○	○	○	○
Vitamin A	○	○	○	○	○	○	○	○	○
Vitamin B	○	○	○	○	○	○	○	○	○
Vitamin C	○	○	○	○	○	○	○	○	○
Vitamin E	○	○	○	○	○	○	○	○	○
B- carotene	○	○	○	○	○	○	○	○	○
Calcium	○	○	○	○	○	○	○	○	○
Folic acid / folate	○	○	○	○	○	○	○	○	○
Iron	○	○	○	○	○	○	○	○	○
Zinc	○	○	○	○	○	○	○	○	○
Oatbran or wheatbran	○	○	○	○	○	○	○	○	○
Evening primrose oil / fish oil	○	○	○	○	○	○	○	○	○
Herbal remedies e.g Echinacae	○	○	○	○	○	○	○	○	○
Other nutrients	○	○	○	○	○	○	○	○	○

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SECTION B: This section asks about some of your food habits
(please select ONE answer only)

1. What type of milk do you usually consume?

- O Whole milk
- O Low / reduced fat milk
- O Skimmed milk
- O Evaporated or sweetened condensed milk
- O Soy milk
- O Vitamin/calcium enriched milk
- O Other milk
- O None of the above
- O Don't know

2. When you eat the following products, how often do you eat a low/reduced fat variety?

Product	Never/Rarely	Sometimes	Usually	I don't eat this food
Cream	O	O	O	O
Ice-cream	O	O	O	O
Cheddar- type cheeses	O	O	O	O
Oily salad dressing	O	O	O	O
Spreads (e.g. margarine, peanut butter, mayonnaise)	O	O	O	O

3. How often is the meat you eat trimmed of fat either before or after cooking?

- O Never/Rarely
- O Sometimes
- O Usually
- O I don't eat meat

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4. **How many serves of vegetables (excluding potatoes) do you <u>usually</u> eat each day?** *(one serve= ½ cup cooked vegetables or 1 cup of salad vegetables)*

O 1 serve or less

O 2-3 serves

O 4-5 serves

O 6 or more serves

O I don't eat vegetables

5. **How many serves of fruit do you <u>usually</u> eat each day?** *(one serve= 1 medium piece of fruit or 1 cup of diced pieces)*

O 1 serve or less

O 2-3 serves

O 4-5 serves

O 6 or more serves

O I don't eat fruit

6. **When cooking, how often do you or the person who cooks your food use the following?**

Product	Never/Rarely	Sometimes	Usually	Don't know
Olive oil	O	O	O	O
Canola or sunflower oil	O	O	O	O
Vegetable oil	O	O	O	O
Nut oil (eg peanut oil)	O	O	O	O
Butter	O	O	O	O
Margarine	O	O	O	O
Omega 3 or phyto sterol margarine	O	O	O	O
Dairy blend	O	O	O	O
Lard or dripping	O	O	O	O
Other oil: (please specify)	O	O	O	O

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7. Which one of the following best describes your usual way of eating?

- O Vegetarian
- O Weight reduction diet
- O Diabetic diet
- O Fat modified diet
- O Other (eg vegan, salt free)
- O No special way of eating

8. How many slices of bread do you normally eat in one day? Count a large bread roll as equal to 2 slices and a small bread roll as equal to 1 slice. *(Write 0 if you do not eat bread or rolls.)*

☐☐ Slices

9. What type of spread do you usually use on bread, savoury biscuits etc?

- O I do not use any spread
- O Butter
- O Poly unsaturated margarine
- O Canola
- O Table margarine
- O Era ™ or light margarine
- O Omega 3 or phytosterol margarine
- O An oil (e.g.olive oil)
- O Cream cheese
- O Nut butter (e.g. peanut butter)
- O Another kind of spread
- O Don't know

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10. What type of butter or margarine do you normally use?

- O I do not use butter or margarine
- O Unsalted
- O Low-salt (less than 1% salt)
- O Normally salted (1-1.5%salt)
- O Strongly salted (more than 2.5% salt)
- O Don't know

11. How many lumps or teaspoonfuls of sugar do you usually add to one cup of coffee, tea or chocolate? Write "0" if you do not add any sugar.

	Teaspoons, or lumps of sugar	I don't drink this beverage
Coffee	☐	O
Tea	☐	O
Chocolate	☐	O

12. How often do you salt your food after it is cooked?

- O Never/rarely
- O Sometimes
- O Usually
- O Don't know

13. How often do you salt your food during cooking?

- O Never/rarely
- O Sometimes
- O Usually
- O Don't know

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14. **How many times per week would you usually eat hot takeaway meals** (*e.g. pizza, burgers, fried or roast chicken, Chinese/Indian/Thai takeaway*)

- O I don't eat takeaway
- O 1 meal or less per week
- O 2-3 meals per week
- O 4-5 meals per week
- O 6-7 meals per week

15. **How many times per week would you usually eat out for dinner** (*restaraunt meals or friends/relatives houses*)

- O I don't eat out
- O 1 meal or less per week
- O 2-3 meals per week
- O 4-5 meals per week
- O 6-7 meals per week

16. **Where do you normally eat your lunch? (meal between 10 am and 3 pm)**

- O I do not normally have lunch
- O At home
- O In a student/school canteen
- O in a cafeteria, restaurant, bar, fast food restaurant
- O Elsewhere (please specify) ☐

17. **Where do you normally eat your dinner? (meal between 3pm and 8pm)**

- O I do not normally have dinner
- O At home
- O In a student/school canteen
- O in a cafeteria, restaurant, bar, fast food restaurant
- O Elsewhere (please specify) ☐

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18. Who normally prepares your main meal at home on working days?

- O I do not have meals at home on working days
- O Myself
- O My partner
- O My mother or father
- O Someone else (please specify) _____ (e.g. housemate)
- O Myself, together with..... _____ (e.g partner)

19. Who normally buys the groceries for your household?

- O Nobody
- O Myself
- O My partner
- O My mother or father
- O Someone else (please specify) _____ (e.g. housemate)
- O Myself, together with..... _____ (e.g. partner)

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SECTION C: When, and what, did you eat and drink yesterday?

1. What day was it yesterday?

Monday ○ Tuesday ○ Wednesday ○ Thursday ○ Friday ○ Saturday ○ Sunday ○

Think back to **yesterday**. In the chart below fill in the circle to indicate the sorts of meals and drinks you had at each time of the day.
(Please remember to fill in a response for both food AND drink for each time period, even if you have not consumed anything)

Snacks include things like a biscuit or a piece of fruit.
A small meal would be something like beans on toast, boiled egg and bread, breakfast cereal, a pie or a pastie.
A large meal would be something like meat and three veg, or a large serving of fish and chips.
You may specify more than one type of drink for each time period, e.g. alcohol and water

TIME	Did you eat anything?				Did you drink anything?			
	No	A snack	A small meal	A large meal	No	Alcohol	Water	Something else
6-7am	○	○	○	○	○	○	○	○
7-8am	○	○	○	○	○	○	○	○
8-9am	○	○	○	○	○	○	○	○
9-10am	○	○	○	○	○	○	○	○
10-11am	○	○	○	○	○	○	○	○
11-12noon	○	○	○	○	○	○	○	○
12-1pm	○	○	○	○	○	○	○	○
1-2pm	○	○	○	○	○	○	○	○
2-3pm	○	○	○	○	○	○	○	○
3-4pm	○	○	○	○	○	○	○	○
4-5pm	○	○	○	○	○	○	○	○
5-6pm	○	○	○	○	○	○	○	○
6-7pm	○	○	○	○	○	○	○	○
7-8pm	○	○	○	○	○	○	○	○
8-9pm	○	○	○	○	○	○	○	○
9-10pm	○	○	○	○	○	○	○	○
10-11pm	○	○	○	○	○	○	○	○
11pm-6am	○	○	○	○	○	○	○	○

Appendix C: National Dietary Survey of Schoolchildren food record codes

The following tables outline how the National Dietary Survey of Schoolchildren (NDSS) Food Codes were categorised to match the Childhood Determinants of Adult Health (CDAH) food frequency questionnaire items.

Key

Food category/item	Group	Sub-Group	Code
CDAH FOOD GROUP e.g. 1. DAIRY FOODS			
CDAH Food item e.g. Flavoured milk drink			
NDSS food item[a] e.g. Ovaltine	O[b]	7	598

[a] If the NDSS Food Code description was missing the NDSS food is recorded as "Not specified" and has been categorised according to the Group and Subgroup. For example, NDSS Food Code 213 is missing a description, but has Food Group = H (Milk and milk products), and Subgroup = 7 (Ice-creams and ice confections), so has been grouped under the "Ice-cream" CDAH food item. Where it was not clear from the NDSS Food Group and Food Subgroup what CDAH grouping the item should be categorised into, the nutrient values of the items were examined. For example, Food Codes 416 and 378 were both missing descriptions but have Food Group= B (Vegetables), Subgroup = 1 (Potatoes) to identify them as potato foods. There are two relevant CDAH categories: "Potato - boiled, mashed or baked", and "Hot chips/roast potatoes/potato wedges". For items recorded as 416, the gram weight of total fat was around 3% of the total food gram weight, so these items were categorised as "Potato - boiled, mashed or baked". Whereas for items recorded as 378, the fat gram weight was around 22% of the total food weight so these items were categorised as "Hot chips/roast potatoes/potato wedges".

[b] Food Group codes:
A - Cereal and cereal products
B - Vegetables
C - Fruits
D - Meat and meat products
E - Fish, seafood and seafood products
F - Eggs
G - Nuts and seeds
H - Milk and milk products
I - Fats
J - Sugars, jams, honey and syrups
K - Confectionary
L - Snack foods
M - Beverages, non-alcoholic
N - Beverages, alcoholic
O - Condiments, flavourings and soups.

Table C.1 NDSS food items grouped into CDAH FFQ categories

Food category/item	Group	Sub-Group	Code
1. DAIRY FOODS			
Flavoured milk drink			
Milk shake/thickshake	H	9	200
Not specified	H	9	208
Not specified	H	9	211
Drinking choc	O	7	595
Cocoa powder (drinking)	O	7	596
Horlicks	O	7	597
Ovaltine	O	7	598
Whole Milk, as drink, in beverages			
Milk, blended	H	1	184
Condensed (whole)	H	3	191
Evaporated milk (whole)	H	3	193
Whole milk (cow)	H	1	194
Whole milk (goat)	H	1	197
Whole milk powder	H	4	198
Whole milk in coffee	H	1	834
Reduced fat milk, as drink, in beverages			
Evaporated milk (reduced fat)	H	3	190
Condensed milk (reduced fat)	H	3	192
Skim milk	H	2	195
Reduced fat milk	H	2	196
Skim milk powder	H	4	199
Reduced fat milk powder	H	3	206
Not specified	H	4	707
Not specified	H	4	756
Skim milk in coffee	H	2	835
Reduced fat milk in coffee	H	2	836
Cream or sour cream			
Cream, imitation	H	8	185
Light cream, including sour	H	8	186
Thickened cream, including sour	H	8	188

Food category/item	Group	Sub-Group	Code
Canned cream	H	8	189
Cream dessert topping	H	8	703
Ice-cream			
Not specified	H	7	182
Not specified	H	7	183
Ice-cream	H	7	205
Frozen confection	H	7	212
Not specified	H	7	213
Not specified	H	7	214
Not specified	H	7	215
Frozen confection	H	7	216
Ice-cream, homemade, plain	H	7	265
Not specified	H	7	266
Not specified	H	7	550
Not specified	H	7	552
Iceblock, sugar only	H	7	553
Ice-cream sundae, McDonalds	H	7	590
Yoghurt - full fat			
Yoghurt, plain, full fat	H	6	201
Yoghurt, flavoured/fruit, full fat	H	6	202
Not specified	H	6	218
Yoghurt - reduced fat			
Yoghurt flavoured/fruit, low fat	H	6	203
Yoghurt, plain, low fat	H	6	204
Yoghurt, flavoured/fruit, reduced fat	H	6	210
Cottage or ricotta cheeses			
Cottage cheese, creamed	H	5	173
Cottage cheese, low fat	H	5	174
Cottage cheese, skim milk	H	5	227
Cream cheese			
Cream cheese	H	6	175
Cheddar and other cheeses			
Camembert	H	5	171
Cheddar	H	5	172
Blue cheese	H	5	176
Edam	H	5	177
Parmesan	H	5	178
Processed cheese	H	5	179
Cheese spread	H	5	180
Stilton	H	5	181
2. BREAD & CEREAL FOODS			
White bread, toast or rolls			
White flour, plain	A	4	6
White flour, self-raising	A	4	7
Damper	A	1	32
White bread	A	1	36
White bread rolls	A	1	40
Breadcrumbs	A	1	42
Garlic bread	A	1	49
Wholemeal/mixed grain bread, toast or rolls			
Soy flour, low fat	A	7	4
Soy flour, full fat	A	7	5
Wholemeal flour, plain	A	4	8
Light rye bread	A	1	33
Dark rye bread	A	1	34
Wholemeal bread	A	2	37
Brown bread	A	2	38
Brown bread roll	A	2	41
Wholemeal bread roll	A	2	48
English muffin, bagel or crumpet			
Flour in coated fried foods	A	4	20
Not specified	A	3	31
Muffins, all types	A	3	43
Crumpets	A	3	44
Fruit bread	A	3	45
Fruit bun	A	3	46
Not specified	A	3	52
Flat bread			
Lebanese bread	A	1	39
Dry or savoury biscuits, crispbread, crackers			
Cracker, high fat	A	16	78
Biscuit cracker, medium fat	A	16	82
Dry cracker plain	A	16	83
Cracker, rice/prawn	A	16	540
Pretzels	A	16	541
Muesli			
Muesli, untoasted	A	10	64
Muesli, toasted	A	10	65
Muesli flake	A	10	66
Porridge			
Oatmeal/oats, rolled, boiled	A	11	10
Oatmeal/oats, rolled, raw	A	11	11
Breakfast cereal			
Bran, unprocessed	A	8	2
Wheatgerm, wheat hearts	A	7	17
All Bran	A	8	61
Cornflakes	A	9	62
Special K	A	12	63
Sugar Puffs	A	21	67
Weetbix	A	13	68
Puffed wheat	A	13	69
Rice (white or brown)			
Rice, brown	A	6	14

Food category/item	Group	Sub-Group	Code
Rice, white	A	6	15
Pasta (including filled), noodles			
Pearl barley	A	7	1
Pasta, boiled	A	5	12
Pasta, spaghetti, canned	A	5	13
Semolina/polenta	A	7	16
Not specified	A	5	24
Lasagne (pasta, cheese and Bolognese sauce)	D	8	811
Macaroni cheese, home-made	D	8	812
Macaroni cheese, commercial	D	8	813
3. Meat, Fish, Eggs			
Mince dishes (e.g. rissoles, meatloaf)			
Beef burger, beef rissoles, fried	D	1	912
Mixed dishes with beef, veal, lamb, pork (e.g. stir fry, casserole)			
Not specified	D	8	738
Not specified	D	8	739
Not specified	D	8	740
Not specified	D	8	741
Not specified	D	8	743
Not specified	D	8	744
Not specified	D	8	745
Not specified	D	8	746
Not specified	D	8	747
Not specified	D	8	748
Not specified	D	8	750
Not specified	D	8	752
Beef mince stew, undrained	D	8	793
Beef mince and vegetable stew	D	8	794
Not specified	D	8	795
Cottage pie/shepherd's pie	D	9	798
Chilli Con Carne	D	8	800
Beef and gravy stew, canned	D	8	808
Sauce, Bolognese	D	8	815
Not specified	D	8	818
Not specified	D	8	820
Not specified	D	8	821
Not specified	D	8	822
Not specified	D	8	823
Not specified	D	8	824
Not specified	D	8	825
Not specified	D	8	826
Not specified	D	8	843
Not specified	D	8	844
Not specified	D	8	845
Not specified	D	8	846
Not specified	D	8	847
Not specified	D	8	848
Not specified	D	8	849
Not specified	D	8	850
Shepherd's pie	D	8	895
Beef mince, stewed	D	1	915
Rabbit, stewed	D	3	962
Not specified	D	8	987
Not specified	D	8	988
Not specified	D	8	990
Beef, veal - roast, chop or steak			
Beef, ribeye steak, grilled, untrimmed	D	1	782
Beef, ribeye steak, grilled, trimmed	D	1	783
Beef, ribeye steak, grilled, trimming unknown	D	1	784
Beef fillet, grilled/fried, trimming unknown	D	1	797
Beef, rump steak, untrimmed	D	1	829
Beef, rump steak, trimmed	D	1	830
Beef, rump steak, trimming unknown	D	1	831
Beef filled, grill/fried, trimmed	D	1	832
Beef fillet, grill/fried, untrimmed	D	1	833
Not specified	D	1	867
Beef, sirloin, grilled, untrimmed	D	1	874
Beef, sirloin, grilled, trimmed	D	1	875
Beef, sirloin, grilled, trimming unknown	D	1	876
Beef, brisket, untrimmed	D	1	892
Beef, brisket, trimmed	D	1	893
Beef brisket, trimming unknown	D	1	894
Beef stewing cuts, unknown if trimmed	D	1	898
Beef sirloin, roast, trimming unknown	D	1	899
Beef, topside roast, trimming unknown	D	1	900
Beef silverside corned, trimming unknown	D	1	901
Beef steak, grilled, trimming unknown	D	1	902
Beef steak all types, trimming/cooking method unknown	D	1	903
Chuck steak, trimmed, stewed	D	1	913
Chuck steak, untrimmed, stewed	D	1	914

Food category/item	Group	Sub-Group	Code
Beef, sirloin, roast, untrimmed	D	1	916
Beef sirloin, roast, trimmed	D	1	917
Beef, topside, roast, untrimmed	D	1	918
Beef, topside, roast, trimmed	D	1	919
Beef, silverside, corned, untrimmed, boiled	D	1	920
Beef, silverside, corned, trimmed, boiled	D	1	921
Beef, steak, grilled, untrimmed	D	1	922
Beef, steak, grilled, trimmed	D	1	923
Beef, steak, fried, untrimmed	D	1	924
Beef, steak, fried, trimmed	D	1	925
Oxtail, stewed	D	1	955
Veal, crumbed and fried	D	1	973
Veal, roast	D	1	974
Not specified	D	1	983
Beef, steak, blade, untrimmed	D	1	992
Beef steak, blade, trimmed	D	1	993
Beef steak, blade, trimming unknown	D	1	994
Veal schnitzel, frozen, fried	D	8	995
Lamb - roast, chop			
Lamb shank, untrimmed	D	2	827
Lamb shank, trimmed	D	2	828
Lamb, chump chop, untrimmed	D	2	864
Lamp, chump chop, trimmed	D	2	865
Lamb, chump chop, trimming unknown	D	2	866
Lamb, leg, baked, unknown if trimmed	D	2	868
Lamb, leg, baked, untrimmed	D	2	869
Lamb, leg, baked, trimmed	D	2	870
Lamb, cutlets, untrimmed	D	2	880
Lamb, cutlets, trimmed	D	2	881
Lamb, cutlets, trimming unknown	D	2	882
Lamb, chops, loin, untrimmed	D	2	883
Lamb, chops, loin, trimmed	D	2	884
Lamb, chops, loin, trimming unknown	D	2	885
Lamb, chops, unknown type, trimming unknown	D	2	886
Lamb chops, forequarter, stewed, untrimmed	D	2	887
Lamb, unknown type, roast, trimming unknown	D	2	888
Lamb shoulder, roast, trimming unknown	D	2	889
Lamb chops, loin, untrimmed, grilled	D	2	943
Lamb chops, loin, trimmed, grilled	D	2	944
Lamb, chops, F/quarter, untrimmed, stewed	D	2	945
Lamb, chops, F/quarter, trimmed, stewed	D	2	946
Lamb, leg, roast, untrimmed	D	2	947
Lamb, leg, roast, trimmed	D	2	948
Lamb, shoulder, roast, untrimmed	D	2	949
Lamb, shoulder, roast, trimmed	D	2	950
Pork - roast, chop			
Pork chop, loin, untrimmed	D	4	853
Pork chop, loin, trimmed	D	4	854
Pork chop, forequarter, untrimmed	D	4	855
Pork chop, forequarter, trimmed	D	4	856
Pork chop, forequarter, trimming unknown	D	4	857
Pork chop, unspecified, untrimmed	D	4	858
Pork chop, unknown type, trimmed	D	4	859
Pork chop, unknown type, untrimmed	D	4	860
Pork steak, unspecified, untrimmed	D	4	861
Pork steak, unknown type, trimmed	D	4	862
Pork steak, unspecified, trimming unknown	D	4	863
Pork, roast, unspecified, untrimmed	D	4	871
Pork, roast, unspecified, trimmed	D	4	872
Pork roast, unspecified, trimming unknown	D	4	873
Pork leg roast, trimming unknown	D	4	891
Pork, belly rashers, untrimmed, grilled	D	4	957
Pork, chops, mid-loin, untrimmed, grilled	D	4	958
Pork, leg, untrimmed	D	4	960
Pork, leg, trimmed	D	4	961
Ham/bacon			
Bacon rashers, grilled, trimming unknown	D	4	896
Bacon rashers, fried, trimming unknown	D	4	897
Bacon, rashers, grilled, untrimmed	D	4	908
Bacon, rashers, grilled, trimmed	D	4	909

Food category/item	Group	Sub-Group	Code
Bacon, rashers, fried, untrimmed	D	4	910
Bacon, rashers, fried, trimmed	D	4	911
Ham steak, grilled	D	4	936
Pork, crackling	D	4	977
Luncheon meats, salami			
Brawn	D	7	905
Cured ham	D	4	937
Luncheon meat	D	7	938
Corned beef, canned	D	7	939
Meat paste	O	5	954
Salami	D	7	964
Chicken, pressed (roll, loaf)	D	7	986
Sausages (beef, pork, other)			
Scotch egg	F	1	224
Frankfurter, canned	D	6	934
Sausage, beef	D	6	965
Sausage, pork	D	6	966
Not specified	D	6	991
Liver (including pate)			
Liver, cooked, coated in flour	D	5	951
Liver sausage	D	5	953
Liver, cooked, not flour coated	D	5	979
Liver, cooked, chicken, not flour coated	D	5	981
Other offal (e.g. kidneys)			
Kidney, cooked	D	5	942
Tongue, pickled, canned, lamb	D	5	968
Tripe, stewed	D	5	976
Brains, lamb, boiled	D	5	980
Not specified	D	5	984
Mixed dishes with chicken, duck, turkey (e.g. casserole, stir fry)			
No equivalent NDSS food item code.			
Chicken, turkey, duck - roast, steamed or barbequed			
Not specified	D	3	878
Chicken, meat only, roast	D	3	928
Chicken, meat and skin, roast	D	3	929
Chicken, light meat only, roast	D	3	930
Chicken, dark meat only, roast	D	3	931
Duck, roast, meat only	D	3	932
Duck, roast, meat and skin	D	3	933
Turkey, roast, meat only	D	3	969
Turkey, roast, meat and skin	D	3	970
Turkey, roast, light meat only	D	3	972

Food category/item	Group	Sub-Group	Code
Chicken – fried (ONLY A FOOT ITEM AT CDAH-2 AND CDAH-3)			
Chicken croquette	D	9	721
Chicken, K.F.C, barbecue	D	9	722
Chicken, K.F.C, fried, thigh, wing	D	9	723
Chicken, crumbed and fried	D	3	985
Canned fish (e.g. tuna, salmon, sardines)			
Tuna, canned in oil, drained	E	3	289
Tuna, canned in brine, drained	E	3	290
Salmon, canned, drained	E	3	291
Crab, canned	E	2	299
Salmon, canned	E	3	312
Sardines, canned in oil	E	3	315
Sardines, canned in sauce	E	3	316
Tuna, canned in water or brine	E	3	320
Tuna, canned in oil	E	3	321
Sardines, canned in oil, drained	E	3	322
Fresh fish - steamed, baked, grilled			
Fish, steamed	E	1	304
Herring, grilled	E	3	305
Trout, steamed, fresh water	E	1	329
Frozen fish - steamed, baked, grilled			
No equivalent NDSS food item code.			
Fish, fried			
Salmon rissole	E	3	288
Calamari, fried in batter/crumbs	E	3	294
Fishburger, McDonalds	D	9	295
Fish cakes, fried	E	3	300
Fish fingers, fried	E	3	301
Fish, coated and fried	E	1	302
Fish, crumbed, fried, homemade	E	1	303
Fish, oven fried, coated	E	1	330
Prawns, crumbed or battered	E	2	331
Mussels/oysters			
Mussels, boiled	E	2	308
Oysters, raw	E	2	309
Lobster/crayfish/yabbies			
Lobster, boiled or canned	E	2	307
Calamari/squid			
Calamari, boiled, steamed	E	3	293
Prawns			
Prawns, boiled	E	2	311
Shrimp	E	2	318
Other seafood			

Food category/item	Group	Sub-Group	Code
Not specified	E	3	287
Not specified	E	3	292
Fish paste	E	3	296
Anchovy	E	3	297
Crab, boiled	E	2	298
Scallops, steamed	E	2	317
Fish paste	E	3	877
Egg			
Fried egg	F	1	219
Egg, duck, whole cooked	F	1	220
Eggs, whole, cooked	F	1	221
Eggs, white, raw	F	1	222
Eggs, yolk	F	1	223
Plain omelette	F	1	225
Scrambled eggs	F	1	226
4. Sweets, baked goods and snacks			
Cakes, sweet muffins, scones or pikelets			
Fruit cake	A	15	91
Not specified	A	15	93
Lamington	A	15	94
Cake, plain	A	15	95
Not specified	A	15	97
Cake, Victoria sponge	A	15	98
Cake, Swiss roll	A	15	99
Cake, sponge, plain	A	15	100
Rock cake	A	15	104
Pancake	A	3	115
Pikelet	A	3	117
Scone	A	3	119
Doughnuts, waffles	A	3	121
Dumplings (bread)	A	3	122
Not specified	A	3	125
Sweet pies or sweet pastries			
Croissant	A	3	50
Flaky/Puff pastry	A	14	107
Shortcrust pastry	A	14	109
Choux pastry	A	14	111
Pastry, wholemeal, shortcrust	A	14	114
Apple pie, family size, homemade	A	19	129
Vanilla slice	A	19	131
Fat in cheesecakes	A	19	140
Cheesecake	A	19	143
Custard tart	A	19	148
Fruit crumble	A	19	150
Lemon meringue pie, home made	A	19	152
Lemon meringue pie, commercial	A	19	153
Fruit pie	A	19	155
Apple pie, McDonalds	A	19	156
Other puddings or desserts			
Sponge pudding, steamed, homemade	A	17	89
Sponge pudding, steamed, commercial	A	15	90
Puddings, self-saucing	A	15	101
Not specified	A	19	128
Not specified	A	19	130
Not specified	A	20	136
Not specified	A	20	137
Custard, homemade, from powder	A	19	138
Not specified	A	19	139
Bread and butter pudding	A	19	142
Custard, no egg	A	19	144
Custard, egg or baked, home-made	A	19	145
Custard, egg or baked, commercial	A	20	146
Jelly	A	20	151
Trifle	A	19	154
Not specified	A	20	217
Meringue	A	20	567
Not specified	A	20	568
Not specified	A	20	569
Not specified	A	20	570
Not specified	A	20	571
Jelly, low energy	A	21	633
Plain, sweet biscuits			
Muesli bar, commercial	A	22	70
Not specified	A	22	71
Not specified	A	17	75
Not specified	A	17	76
Anzac biscuit	A	17	77
Sweet plain biscuit	A	17	86
Homemade biscuit	A	17	88
Biscuit, homemade, fruit	A	17	133
Cream, chocolate biscuits			
Not specified	A	18	55
Not specified	A	18	56
Not specified	A	18	57
Biscuit, homemade, cream sandwich	A	18	58
Not specified	A	18	59
Not specified	A	18	72
Not specified	A	18	73

Food category/item	Group	Sub-Group	Code
Biscuit, homemade, choc-chip	A	18	74
Fat in biscuits/slices	A	18	79
Chocolate biscuit	A	18	81
Shortbread biscuit	A	18	84
Sweet fancy biscuit	A	18	85
Limmits biscuit	A	18	87
Choc-coated fancy biscuit, high fat	A	18	281
Choc-coated plain biscuit	A	18	285
Choc-coated fancy biscuit	A	18	286
Meat pie, sausage roll or other savoury pastries			
Filo pastry	A	14	123
Not specified	D	9	708
Chiko/spring roll, home-made	D	9	709
Saveloy, battered	D	6	817
Pie, meat, individual	D	9	711
Pie, meat, party	D	9	712
Pasty, individual	D	9	713
Chiko/spring roll, commercial	D	9	714
Pie, pork	D	9	715
Sausage roll, individual	D	9	716
Sausage roll, party	D	9	717
Meat pie, homemade	D	9	732
Quiche, ham and cheese, commercial	D	8	785
Pasty	D	9	786
Pie meat, family, commercial	D	9	802
Quiche, home-made	D	8	806
Quiche, commercial	D	8	807
Cheese souffle, homemade	D	9	809
Pie, spinach	D	8	814
Pizza			
Pizza, home-made	D	9	710
Pizza, non-Pizza Hut	D	9	718
Pizza, Pizza Hut	D	9	719
Hamburger			
Hamburger, McDonalds, Big Mac	D	9	725
Hamburger, McDonalds, Cheese	D	9	726
Hamburger, McDonalds, Junior	D	9	727
Hamburger, McDonalds, McFeast	D	9	728
Hamburgers, plain	D	9	729
Hamburgers, cheese, cheeseburger	D	9	730
Not specified	D	9	731
Hamburger, with bacon	D	8	989

Food category/item	Group	Sub-Group	Code
Chocolate (including chocolate bars)			
Milk chocolate	K	1	575
Dark chocolate	K	1	576
Chocolate, filled	K	1	577
Mars Bar	K	1	578
Bounty Bar	K	1	580
Other confectionary			
Not specified	K	2	572
Not specified	K	2	573
Not specified	K	2	574
Not specified	K	2	589
Not specified	K	2	591
Confectionary, sweets	K	2	579
Carob bars	K	2	581
Oilseed/nut bars	K	2	582
Fruit bars	K	2	583
Liquorice	K	2	584
Toffees	K	2	585
Pastilles/jellies, gums, marshmallows	K	2	586
Carob button, light	K	2	587
Potato chips, corn chips, Twisties etc			
Snacks, crackling	L	1	542
Potato crisps, straws	L	2	543
Snacks, extruded, non-cheese	L	1	544
Snacks, extruded, cheese flavour	L	1	545
Corn chips	L	1	546
Popcorn, plain	L	1	588
Almonds, walnuts, hazelnuts			
Almonds, pistachio nuts	G	2	231
Hazelnuts	G	2	237
Walnuts	G	2	240
Cashews			
Cashews	G	2	230
Coconuts			
Coconut, desiccated	G	2	234
Coconut, meat, fresh	G	2	235
Peanuts			
No equivalent NDSS food item code.			
Pistachio			
No equivalent NDSS food item code.			
Seeds - pumpkin, sesame, pine nuts, tahini			
Sesame seeds	G	2	229
Sunflower seeds	G	2	244
Pine nuts	G	2	246
Pumpkin seeds	G	2	247
Caraway seeds	G	2	332

Food category/item	Group	Sub-Group	Code
Other nuts, seeds			
Brazil nuts	G	2	232
Chestnuts	G	2	233
Nuts, roasted, salted	G	1	238
Macadamias	G	2	241
5. Dressings and spreads			
Oil and vinegar dressing			
Dressing, French, commercial	O	4	679
Dressing, Italian, commercial	O	4	681
Mayonnaise or other creamy dressings			
Mayonnaise, rich	O	4	664
White sauce, sweet	O	1	675
White sauce, savoury	O	1	678
Dressing, coleslaw	O	4	683
Dressing, mayonnaise, light	O	4	684
Cheese sauce	O	1	693
White savoury sauce, homemade	O	1	780
Jam, marmalade, syrup, or honey			
Honey	J	3	556
Marmalade	J	2	557
Jam	J	2	558
Sugar	J	1	559
Glucose, liquid, glucose powder	J	3	560
Golden syrup	J	3	561
Lemon flavoured spread, commercial	J	2	562
Lemon curd, homemade	J	2	563
Peanut butter, other nut spreads			
Peanut paste, peanut butter	G	1	239
Vegemite, Marmite, or Promite			
Marmite/Promite	O	5	697
Vegemite	O	5	698
Creamy dips and spreads			
Fat spreads, unknown, on sandwich or roll	I	4	158
Margarine - Polyunsaturated, used as spread	I	2	159
Butter	I	1	160
Not specified	I	3	161
Table margarine	I	3	162
Fat spreads, unknown, in sandwich	I	4	163
Not specified	I	4	165
Cooking margarine used as spread	I	3	257
6. Non-dairy			
Fruit juice (100%)			
Not specified	M	2	27
Not specified	M	2	28
Lemon juice, fresh	M	2	449
Apple juice	M	2	608
Apricot nectar	M	2	609
Grape, pear juice	M	2	610
Grapefruit juice, unsweetened	M	2	611
Orange juice	M	1	613
Pineapple juice	M	2	614
Vegetable, tomato juices			
Tomato juice	M	2	615
Vegetable juice	M	2	616
Fruit juice drink or fruit cordial			
Fruit juice drink	M	3	607
Tang powder	O	7	635
Fruit drink, all types	M	3	636
Fruit juice drink, except orange or tropical	M	3	637
Low-joule cordial			
Cordial, low energy	M	5	631
Cordial			
Cordial, lime, other	M	6	604
Cordial, Ribena	M	6	605
Low-joule soft drink			
Cola soft drink, low energy	M	5	593
Soft drink, low energy	M	5	630
Soft drinks (including flavoured mineral water)			
Not specified	M	4	551
Cider, non-alcoholic	M	2	600
Cola drinks	M	4	617
Lemonade	M	4	618
Mineral water with fruit juice	M	4	619
Tonic water	M	4	624
Soft drink, soda stream	M	4	638
Water (including unflavoured mineral water, soda water, tap water)			
Water	M	11	622
Mineral water	M	5	629
Soda water	M	5	632
Mineral water, low energy	M	5	634
Water, as diluting agent	M	6	639
Water, in coffee	M	8	839
Coffee - full strength			
Not specified	M	8	592
Coffee, instant, dry	M	8	601
Coffee, instant, made up, black	M	8	602
Coffee and chicory, concentrate	M	8	603

Food category/item	Group	Sub-Group	Code
Decaffeinated coffee			
Coffee, made up, decaffeinated	M	8	625
Coffee substitute, made up	M	8	626
Carob powder beverage flavouring	M	7	627
Coffee substitute, dry powder	M	8	628
Tea - green or black (ordinary tea)			
Tea	M	7	621
Herbal tea			
Herbal tea	M	7	620
Soy beverages			
Soy milk	B	3	594
Light beer			
No equivalent NDSS food item code.			
Medium-strength beer			
No equivalent NDSS food item code.			
Full-strength beer			
Beer, extra stout, stout	N	1	641
Beer	N	1	644
Red wine			
Red wine	N	1	657
White wine or champagne/sparkling			
Rose	N	1	658
White wine, dry	N	1	659
White wine, sparkling	N	1	660
Wine cooler			
No equivalent NDSS food item code.			
Spirit-based mixed drinks (e.g. Lemon Ruski)			
No equivalent NDSS food item code.			
Sherry/port/fortified wines			
Port	N	6	653
Sherry	N	6	654
Vermouth, dry	N	6	655
Spirits, liqueurs			
Spirits	N	6	648
Liqueurs	N	6	652
Other alcoholic drinks (e.g. cider)			
No equivalent NDSS food item code.			
Energy drinks (ONLY A FOOD ITEM AT CDAH-3)			
No equivalent NDSS food item code.			
7.Vegetables			
Green/mixed salad (including lettuce, tomato etc) in a sandwich			
No equivalent NDSS food item code.			
Green/mixed salad (including lettuce, tomato etc) as a side salad with a main meal			
No equivalent NDSS food item code.			
Stir-fried or mixed vegetables			
No equivalent NDSS food item code.			
Vegetable casserole			
Cauliflower cheese	B	7	363
Ratatouille	B	8	407
Soup, light or vegetable	O	6	687
Soup, medium	O	6	688
Not specified	B	11	764
Not specified	B	11	775
Not specified	B	8	792
Potato - boiled, mashed or baked			
Potato, baked	B	1	385
Potato, boiled	B	1	386
Potato, canned	B	1	387
Potato, instant	B	1	391
Not specified	B	1	402
Potato, Kentucky Fried, mashed	B	1	410
Not specified	B	1	416
Not specified	B	1	417
Not specified	B	1	767
Not specified	B	1	768
Not specified	B	1	769
Not specified	B	1	770
Not specified	B	1	773
Hot chips/roast potatoes/potato wedges			
Not specified	B	1	378
Potato chips/french fries	B	1	388
Potato chips, home-made	B	1	389
Potato chips, oven fries	B	1	390
Not specified	B	1	760
Not specified	B	1	771
Potato gem/royal	B	1	772
Sweet potato			
Sweet potato, baked	B	1	763
Pumpkin			
Pumpkin, boiled	B	8	392
Peas (including snow peas)			
Peas, snow peas, raw	B	2	383
Peas, boiled	B	2	384
Peas, canned	B	2	394
Green beans			
Green beans, raw	B	2	340
Green beans, boiled	B	2	341
Silverbeet/spinach			
Parsley	B	6	381
Spinach, boiled	B	6	395

Food category/item	Group	Sub-Group	Code
Mustard greens and cress, raw	B	6	415
Watercress, boiled	B	6	761
Fennel, raw	B	6	765
Broccoli			
Broccoli boiled	B	7	351
Broccoli, raw	B	7	766
Cauliflower			
Cauliflower, raw	B	7	360
Cauliflower, boiled	B	7	361
Brussel sprouts, cabbage, coleslaw			
Artichoke globe, boiled	B	11	336
Asparagus, cooked	B	11	337
Bean sprouts, canned	B	6	349
Brussel sprouts, boiled	B	7	352
Cabbage, raw	B	7	353
Cabbage, boiled	B	7	354
Coleslaw, Kentucky Fried	B	7	406
Carrots			
Carrots, raw	B	5	357
Carrots, cooked	B	5	358
Carrots, canned	B	5	359
Parsnips, boiled	B	10	382
Radishes	B	10	393
Swede, boiled	B	10	396
Turnip, boiled	B	10	404
Celeriac, boiled	B	10	413
Mushrooms			
Mushrooms, raw	B	11	376
Mushrooms, boiled or canned	B	11	377
Mushroom, canned in sauce	B	11	414
Capsicum			
Capsicum, raw	B	8	355
Capsicum, cooked	B	8	356
Sweetcorn, corn on the cob			
Corn, boiled	B	11	366
Zucchini, eggplant, squash			
Eggplant	B	8	368
Marrow, raw	B	8	374
Marrow, boiled	B	8	375
Cucumber			
Cucumber	B	8	367
Tomatoes			
Tomato, canned	B	4	399
Tomato, fried	B	4	400
Tomato, raw	B	4	401
Tomato paste	B	4	403
Tomato soup, canned/homemade	O	6	689
Lettuce, except when in a mixed salad			
Lettuce	B	6	373
Alfalfa sprouts	B	6	409
Celery, except when in a mixed salad			
Celery, raw	B	6	364
Celery, boiled	B	6	365
Onion or leek			
Garlic, garlic powder	B	9	334
Leeks, boiled	B	9	370
Onions, raw	B	9	379
Onions, boiled	B	9	380
Onions, fried	B	9	408
Soy beans, tofu			
No equivalent NDSS food item code.			
Baked beans			
Baked beans, canned in sauce	B	3	338
Other beans, lentils			
Not specified	B	3	270
Butter beans, boiled	B	3	339
Broad beans	B	2	342
Not specified	B	3	344
Not specified	B	3	345
Chickpeas, raw	B	3	347
Red kidney beans	B	3	348
Lentils, boiled	B	3	371
Lentils, as dahl	B	3	372
Soup, pea/bean or lentil, homemade	O	6	690
Not specified	B	3	774
Beetroot (ONLY A FOOD ITEM AT CDAH-3)			
Beetroots, boiled	B	10	350
Beetroot, raw	B	10	412
8. Fruits			
Fruits - canned, natural juice or unsweetened			
Apricots, fresh, canned, unsweetened	C	5	424
Fruit salad, canned in juice	C	8	445
Cherries, stewed/canned/ unsweetened	C	5	454
Peaches, stewed, canned, unsweetened	C	5	469
Pears, canned without sugar	C	2	471
Mandarin, oranges, canned	C	1	494
Apples, stewed/canned, unsweetened	C	2	498
Fruit salad, canned	C	8	525
Apricots canned in pear juice	C	5	528

Food category/item	Group	Sub-Group	Code
Apricots canned in unknown type of juice	C	5	529
Apricots, canned, drained	C	5	530
Pears, canned, drained	C	2	531
Pineapple, canned, drained	C	4	532
Peaches, canned, drained	C	5	533
Peaches, canned in pear juice	C	5	534
Peaches, canned, unknown type of juice	C	5	535
Pineapple, canned in juice	C	4	536
Pineapple, canned in unknown juice	C	4	537
Pears, canned in pear juice	C	2	538
Fruit - stewed or canned, sweetened			
Apricots, canned, sweetened	C	5	422
Apricots, dried, stewed, sweetened	C	5	423
Apricots, fresh, stewed, sweetened	C	5	425
Blackberries, stewed, sweetened	C	6	429
Fruit pie filling, canned	C	8	439
Fruit salad, canned, sweetened	C	8	440
Mango, canned, sweetened	C	4	456
Peaches, stewed, canned, sweetened	C	5	468
Pears, canned, sweetened	C	2	472
Pineapple, canned, sweetened	C	4	473
Plums, stewed, canned, sweetened	C	5	477
Prunes, cooked, sweetened	C	5	481
Raspberries, stewed, canned, sweetened	C	6	485
Rhubarb, cooked, sweetened	C	8	488
Strawberries, canned in syrup	C	6	489
Apples, stewed/canned, sweetened	C	2	499
Pears, stewed, sweetened	C	4	519
Pears, canned in syrup	C	2	539
Dried fruit			
Apricots, dried	C	7	421
Currants, dried	C	7	432
Dates, dried	C	7	434
Figs, dried	C	7	435
Fruit mince, sweetened	C	7	438
Peaches, dried	C	7	467
Prune, dried	C	7	479
Raisins	C	7	483
Sultanas	C	7	491
Cherries, glace	C	7	517
Not specified	C	7	520
Not specified	C	7	521
Not specified	C	7	522
Fresh fruit salad			
Fruit salad, fresh	C	8	441
Fruit salad in juice	C	8	526
Fruit salad in heavy syrup	C	8	527
Apple or Pear			
Pears, raw	C	2	470
Apples, raw	C	2	496
Apples, baked without sugar	C	2	497
Orange, mandarin, grapefruit			
Grapefruit, raw	C	1	447
Oranges, raw	C	1	463
Tangerines, raw	C	1	492
Banana			
Banana	C	3	427
Not specified	C	3	523
Peach, nectarine, plum or apricot			
Peaches, raw	C	5	466
Plums, raw	C	5	475
Mango or pawpaw			
Mango, raw	C	4	457
Pawpaw, raw	C	4	465
Pineapple			
Pineapple, raw	C	4	474
Grapes or berries			
Mulberry, raw	C	6	482
Strawberries, raw	C	6	490
Grapes	C	8	493
Blueberries, raw	C	6	500
Cranberries, raw	C	6	512
Melon (watermelon, rockmelon or honeydew)			
Rockmelon/cantaloupe, raw	C	4	458
Watermelon, raw	C	4	460
Kiwi fruit ((ONLY A FOOD ITEM AT CDAH-3)			
Kiwi fruit, raw	C	8	419
Avocado (ONLY A FOOD ITEM AT CDAH-3)			
Avocado	C	4	426
Other fruit not listed			
Lemons, fresh	C	1	448
Olives	C	8	462
Passionfruit	C	4	464
Not specified	C	8	478
Loquat, raw	C	4	506

Table C.2 NDSS food items not grouped into CDAH FFQ categories

Food category/item	Group	Sub-Group	Code
Sauces, condiments and flavourings, not categorised			
Gravy powder	A	7	22
Sweetener, artificial, no kJ	O	8	554
Sweetener Lite & Low type	O	8	555
Not specified	O	1	662
Gravy, unknown recipe	O	1	663
Chilli sauce	O	1	665
Curry powder	O	1	666
Soy sauce	O	1	667
Horseradish, raw	O	1	668
Worcestershire sauce	O	1	670
Pickles in brine, sweetened	O	3	672
Chutney/relish, vegetable	O	3	673
Chutney, fruit	O	3	674
Tomato sauce, commercial	O	2	676
Tomato sauce	O	2	677
Broth soup, canned/homemade	O	6	686
HP sauce	O	1	692
Onion sauce	O	1	694
Not specified	O	1	699
Stock cubes	O	5	700
Coffee whitener	O	7	704
Not specified	O	1	841
Cornflour	A	7	3
Soy Lecithin	O	5	18
Gelatin, dry powder	O	5	696
Yeast, dried	O	5	702
Fats and oils			
Copha, hard	I	7	157
Oil, unknown type, used voluntarily	I	6	164
Not specified	I	7	166
Not specified	I	7	167
Not specified	I	7	168
Cooking oil, unknown type	I	6	169
Not specified	I	7	170
Coconut oil	I	6	258
Cottonseed oil	I	6	259
Peanut oil	I	6	260
Not specified	I	5	261
Olive oil	I	6	262
Not specified	I	6	263
Fat in sauces	O	6	695
Fat in savoury dishes	D	9	799

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