Bi-directional associations between healthy lifestyles and mood disorders in young adults: The Childhood Determinants of Adult Health Study

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Background. Healthy lifestyles prevent cardiovascular disease and are increasingly recognized in relation to mental health but longitudinal studies are limited. We examined bi-directional associations between mood disorders and healthy lifestyles in a cohort followed for 5 years.

Method. Participants were aged 26–36 years at baseline (2004–2006) and 31–41 years at follow-up (2009–2011). At follow-up, lifetime mood disorders (depression or dysthymia) were retrospectively diagnosed with the Composite International Diagnostic Interview. A five-item lifestyle score (comprising body mass index, non-smoking, alcohol consumption, leisure time physical activity and healthy diet) was measured at both time points. Linear and log multinomial regression determined if mood disorder before baseline predicted changes in lifestyle (n = 1041). Log binomial regression estimated whether lifestyle at baseline predicted new episodes of mood disorder (n = 1233). Covariates included age, sex, socio-economic position, parental and marital status, social support, major life events, cardiovascular disease history, and self-rated physical and mental health.

Results. A history of mood disorder before baseline predicted unfavourable trajectories of lifestyle over follow-up, including somewhat lower risk of improvement [relative risk (RR) 0.76, 95% confidence interval (CI) 0.56–1.03] and greater risk of worsening (RR 1.46, 95% CI 0.99–2.15) of lifestyle independent of confounding factors. Higher lifestyle scores at baseline were associated with a 22% (RR 0.76, 95% CI 0.61–0.95) reduced risk of first episodes of mood disorder, independent of confounding factors.

Conclusions. Healthy lifestyles and mood disorders are closely related. Our results suggest that healthy lifestyles may not only reduce cardiovascular disease but also promote mental health.

Key words: Adults, longitudinal studies, mood disorder, risk reduction behaviour.

Introduction

The link between mood disorders, including depression and dysthymia (American Psychiatric Association, 2000), and cardiovascular disease have long been recognized (Seligman & Nemeroff, 2015). Recent evidence suggests that the elevated risk of cardiovascular disease in people with mood disorders can be accounted for by their higher prevalence of risk behaviours, such as smoking and physical inactivity (Ye et al. 2013). Of interest is that several risk behaviours have bi-directional associations with mood disorders. For example, smoking is associated with an increased risk of developing depression and having depression is associated with an increased likelihood of taking up smoking (Chaiton et al. 2009). Similar findings have been reported for physical activity (McKercher et al. 2014) and weight (De Wit et al. 2010; Sanderson et al. 2011).

Studying individual risk factors and their relationship with mood disorders ignores the fact that risk factors often cluster as unhealthy lifestyles in younger (Raitakari et al. 1995; Gall et al. 2009) and older (Van Dam et al. 2008) adults. Unhealthy lifestyles predict all-cause mortality, cardiovascular disease and type 2 diabetes (Spencer et al. 2005; Khaw et al. 2008; Van Dam et al. 2008) but there has been little investigation of whether such lifestyles predict mood disorders. Understanding the associations between healthy lifestyles and mood disorders has implications for reducing cardiovascular risk in those with mood disorders and potentially reducing the burden of mood disorders. There is also a desire to find non-pharmacological ways
to manage mood disorders given the modest effects of pharmacological agents in many people (Undurraga & Baldessarini, 2012; Seligman & Nemeroff, 2015).

The rationale for this research question comes from the known biological links between unhealthy behaviours and mood disorders. Such behaviours are associated with inflammatory and immune pathways (Giugliano et al. 2006; Walsh et al. 2011) that affect the neurobiological pathways associated with mood disorders (Berk et al. 2011). Conversely, those with a mood disorder may engage in unhealthy behaviours in an attempt to control their mood. For example, nicotine and alcohol affect neurotransmitter systems related to the symptoms of mood disorders (Markou et al. 1998).

Our aim was to examine the bi-directional associations between healthy lifestyles and mood disorders in a cohort of young adults followed for 5 years. Based on the associations between mood disorders and individual risk factors, we hypothesized that those with a history of mood disorder would have unfavourable trajectories of their lifestyle and that healthier lifestyles would protect against mood disorder.

Method

Participants

This study was part of the Childhood Determinants of Adult Health (CDAH) study that began in 1985 with a nationally representative study (response proportion 64%) of 8498 children between the ages of 7 to 15 years (Fig. 1) (Venn et al. 2007). Full details are provided in the online Supplementary material. In brief, in 2004–2006 (herein ‘baseline’), participants completed assessments of their lifestyle (n = 2407). In 2009–2011 (herein ‘follow-up’), participants were re-contacted, with 1233 completing assessments of lifetime mood disorder. Of these, 1041 also had complete lifestyle information at follow-up.

Measures

Mood disorder

The lifetime version of the Composite International Diagnostic Interview (CIDI-Auto 2.1 version; World Health Organization, 1997) was administered at follow-up providing retrospective lifetime diagnoses of major depression and dysthymia. Using dates of onset for the first and most recent episodes we classified people as having suffered from an episode of mood disorders before baseline, herein ‘history of mood disorder’, and those that experienced an episode between baseline and follow-up, herein ‘new episode of mood disorder’. This latter category could be separated into ‘recurrent’ and ‘first’ episodes of mood disorder.

Lifestyle risk factors

We calculated a Healthy Lifestyle Score at baseline and follow-up that is associated with biomedical cardiovascular risk factors in this cohort (Gall et al. 2009) and is similar to other scores (Khaw et al. 2008; Lloyd-Jones et al. 2010). Our score comprised five ‘healthy’ items assigned one point each: body mass index (BMI) <25 kg/m², never smoker or ex-smoker ≥12 months, ≥3 h of moderate to vigorous leisure time physical activity per week, ≤20 g alcohol per day and for indicating a ‘healthy’ diet, scoring in the 75th percentile of a validated Dietary Guideline Index that assessed adherence to Australian dietary guidelines from a food frequency questionnaire (Sanjoti et al. 2009). Details of item measurement are given in the online Supplementary material. The total score ranged from zero (no healthy behaviours) to five (all healthy behaviours).

Data analysis

History of mood disorder at baseline predicting changes in the Healthy Lifestyle Score between baseline and follow-up

We investigated whether a history of mood disorder before baseline predicted changes in the Healthy Lifestyle Score during follow-up (calculated as Healthy Lifestyle Scorefollow-up – Healthy Lifestyle Scorebaseline) using linear regression. Log multinomial regression was used to estimate the relative risk [RR ± 95% confidence interval (CI)] of changing category of the Healthy Lifestyle Score over follow-up by history of mood disorder before baseline. Healthy Lifestyle Scores at baseline and follow-up were categorized as low (zero to 2) or high (3 to 5) to make the following variable: highbaseline/highfollow-up (reference group); lowbaseline/highfollow-up; highbaseline/lowfollow-up; lowbaseline/lowfollow-up.

Sensitivity analyses were conducted excluding participants that developed an episode of mood disorder over follow-up, to explore potential reverse causation.

Healthy Lifestyle Score at baseline predicting episodes of mood disorder between baseline and follow-up

We estimated the RR (±95% CI) of having an episode of mood disorder between baseline and follow-up according to baseline Healthy Lifestyle Score using log binomial regression adjusted for covariates. The outcome was classified in several ways: (1) any new episode of mood disorder (i.e. first and recurrent) v. no new episode (this reference category included those with a history of mood disorder before baseline); (2) first v. no new episode (this reference category
included those with a history of mood disorder before baseline; and (3) first v. no lifetime episode of mood disorder (this reference category excluded those with a history of mood disorder before baseline).

Sensitivity analyses included adjusting for baseline mental health [Short Form-12 (SF-12) mental component score; Ware et al. 1996] and excluding those with subthreshold depressive symptoms in the 12 months before baseline (classified with CIDI 12-month version from baseline). The results are presented in the online Supplementary material. These were to explore whether mood disorders present at or before baseline, but below the threshold for Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) diagnoses in the lifetime CIDI at follow-up, could explain any associations seen between baseline lifestyle and mood disorders during follow-up.

We also examined if the findings were influenced by particular health behaviours in the Healthy Lifestyle Score by re-running analyses with the score excluding BMI, physical activity, smoking and diet items. As an alternative way of considering the role of individual healthy lifestyle items, the association between each item in the Healthy Lifestyle Score and the risk of mood disorder was examined by entering each item alone into a model including confounders and then in a model mutually adjusted for all other items and confounders (see online Supplementary material).

**Covariates**

Covariates were included in accordance with purposeful model-building procedures, including the putative covariate being associated with the exposure and
outcome (e.g., Healthy Lifestyle Score and mood disorders) and that the inclusion of the covariate in a model caused a change in the effect estimate of at least 10% (Greenland, 1989). The following potential covariates from baseline were considered: sex, age, highest attained education, area-level disadvantage, marital status, parental status, social support (Henderson Social Support Index; Henderson et al. 1978), personality (NEO five-factor inventory; Costa & McCrae, 1992), history of cardiovascular disease or diabetes (self-report or medication use), use of oral contraceptives (women only), self-rated physical and mental health-related quality of life (SF-12) (Ware et al. 1996) and time between follow-ups. A 12-item life events inventory was administered at follow-up covering the 5 years since baseline (Brugha & Cragg, 1990).

Multiple imputation using chained equations with 30 estimations was used to replace missing data on covariates. This method was used to replace missing data on the covariates listed above using the following variables from a previous follow-up of the cohort between 2001 and 2004 on sex, age, smoking status, education, BMI, state of residence, marital status, self-rated health and one variable from 1985 on scholastic ability.

We examined the effect of loss to follow-up on our results using inverse probability weighting, with weights based on the inverse of the probability of providing follow-up data given variables from a previous adult follow-up in 2001–2004 (sex, age, education, self-rated health, smoking and BMI) or, in a separate analysis, variables from childhood (age, sex, BMI, state of residence and three measures of cardiorespiratory fitness) (Seaman & White, 2013). Unweighted and weighted models, which did not have missing covariates imputed, were then compared. The results of these analyses are presented in the online Supplementary material. To examine the generalizability of our sample, we also compared our included participants with those not included using data from the 1985 study and the general Australian population of a similar age with data from the Australian Bureau of Statistics (Australian Bureau of Statistics, 2007; Australian Bureau of Statistics, 2011). These results are presented in the online Supplementary material.

There was no evidence of effect modification by sex (see online Supplementary material), so results for men and women are presented together. Analyses were conducted in Stata 12.0 (USA).

**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. Participants provided written informed consent and the study was approved by the Tasmanian Health and Medical Human Research Ethics Committee.

**Results**

The characteristics of participants are shown in Table 1. In general, participants were of higher socio-economic status and had healthier lifestyles than those lost to follow-up (see online Supplementary Results) or the Australian population of a similar age, with the exception of lifetime prevalence of mood disorders that were more common in the CDAH study (see online Supplementary Table S1).

**Mood disorders as a predictor of the Healthy Lifestyle Score**

Among the 1041 participants with data for these analyses, those with a history of mood disorder before baseline tended to have unfavourable trajectories of lifestyle (Fig. 2). Those with a history were less likely to improve their lifestyle (RR 0.76, 95% CI 0.49–1.18), more likely to worsen (RR 1.46, 95% CI 0.99–2.15) or stay in the low lifestyle score group (RR 1.30, 95% CI 1.01–1.66) than those without a history (Fig. 2, black squares). Models were adjusted for age, education, history of cardiovascular disease or diabetes, oral contraceptive use in females, area-level socio-economic status, social support and parental status.

Sensitivity analyses excluding those that had a new episode of mood disorder over follow-up (n = 107, Fig. 2, grey squares) mostly strengthened the associations. For example, those with a history were less likely to improve their lifestyle (RR 0.46, 95% CI 0.21–1.01) and more likely to worsen (RR 1.95, 95% CI 1.18–3.22) than those without a history. Applying inverse probability weights did not appreciably change these results (see online Supplementary Results and Supplementary Fig. S1).

The findings using change in the continuous Healthy Lifestyle Score supported the categorical findings. Having a history of mood disorder before baseline was associated with worsening Healthy Lifestyle Score during follow-up, although the changes were small (β = −0.18, 95% CI −0.35 to −0.01, p = 0.040) (online Supplementary Table S2). Sensitivity analyses excluding participants who developed a new episode of mood disorder over follow-up (n = 107) strengthened the association (β = −0.28, 95% CI −0.52 to −0.04, p = 0.024). Applying inverse probability weights made small differences to these results (see online Supplementary Results and Supplementary Table S2).
Table 1. Characteristic of participants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mood disorder predicting lifestyle (n = 1041)</th>
<th>Lifestyle predicting mood disorder (n = 1233)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>% or Mean (S.D.)</td>
<td>% or Mean (S.D.)</td>
</tr>
<tr>
<td>Age, years</td>
<td>1041 31.6 (2.6)</td>
<td>1233 31.65 (2.6)</td>
</tr>
<tr>
<td>Female</td>
<td>657 63</td>
<td>769 62</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertiary</td>
<td>539 52</td>
<td>614 50</td>
</tr>
<tr>
<td>Vocational</td>
<td>258 25</td>
<td>324 26</td>
</tr>
<tr>
<td>School only</td>
<td>244 23</td>
<td>295 24</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>272 26</td>
<td>331 27</td>
</tr>
<tr>
<td>Married/living as married</td>
<td>742 71</td>
<td>866 70</td>
</tr>
<tr>
<td>Divorced/separated/widowed</td>
<td>27 3</td>
<td>36 3</td>
</tr>
<tr>
<td>Parental status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No children</td>
<td>551 53</td>
<td>648 53</td>
</tr>
<tr>
<td>One or more children</td>
<td>488 47</td>
<td>581 47</td>
</tr>
<tr>
<td>Oral contraceptive usea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>276 42</td>
<td>314 41</td>
</tr>
<tr>
<td>Yes</td>
<td>381 58</td>
<td>455 59</td>
</tr>
<tr>
<td>Henderson social support index</td>
<td>1038 62 (7)</td>
<td>1229 62 (7)</td>
</tr>
<tr>
<td>Area-level socio-economic disadvantage</td>
<td>1039 1029 (78)</td>
<td>1231 1028 (78)</td>
</tr>
<tr>
<td>Number of major life events</td>
<td>1041 2.1 (1.7)</td>
<td>1232 2.1 (1.7)</td>
</tr>
<tr>
<td>Personality factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td>965 19.6 (3.7)</td>
<td>1228 19.7 (3.8)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>965 26.7 (3.6)</td>
<td>1228 26.9 (3.6)</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>965 23.7 (4.1)</td>
<td>1228 23.7 (4.1)</td>
</tr>
<tr>
<td>Openness</td>
<td>962 24.3 (3.2)</td>
<td>1228 24.4 (3.2)</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>965 28.7 (2.9)</td>
<td>1228 28.7 (2.9)</td>
</tr>
<tr>
<td>History of cardiovascular disease or diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>868 88</td>
<td>1029 88</td>
</tr>
<tr>
<td>Yes</td>
<td>119 12</td>
<td>143 12</td>
</tr>
<tr>
<td>Time between follow-ups, years</td>
<td>1041 4.9 (0.3)</td>
<td>1233 4.9 (0.3)</td>
</tr>
<tr>
<td>Physical component score on SF-12</td>
<td>1023 54.5 (6.3)</td>
<td>1208 54.5 (6.4)</td>
</tr>
<tr>
<td>Mental component score on SF-12</td>
<td>1023 49.9 (8.1)</td>
<td>1208 49.8 (8.3)</td>
</tr>
<tr>
<td>Healthy Lifestyle Score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline score</td>
<td>1041 2.9 (1.1)</td>
<td>1233 2.9 (1.1)</td>
</tr>
<tr>
<td>Follow-up score</td>
<td>1041 2.9 (1.1)</td>
<td>– –</td>
</tr>
<tr>
<td>Change in score</td>
<td>1041 0.1 (1.0)</td>
<td>– –</td>
</tr>
<tr>
<td>Categorical change in score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highbaseline/highfollow-up</td>
<td>177 35</td>
<td>– –</td>
</tr>
<tr>
<td>Lowbaseline/highfollow-up</td>
<td>145 24</td>
<td>– –</td>
</tr>
<tr>
<td>Highbaseline/lowfollow-up</td>
<td>123 9</td>
<td>– –</td>
</tr>
<tr>
<td>Lowbaseline/lowfollow-up</td>
<td>592 33</td>
<td>– –</td>
</tr>
<tr>
<td>Mood disorder before baseline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>852 82</td>
<td>– –</td>
</tr>
<tr>
<td>History prior to baseline</td>
<td>189 18</td>
<td>– –</td>
</tr>
<tr>
<td>No episode over follow-up</td>
<td>82 8</td>
<td>– –</td>
</tr>
<tr>
<td>New episode over follow-up</td>
<td>107 10</td>
<td>– –</td>
</tr>
<tr>
<td>Mood disorder over lifetime</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>– –</td>
<td>931 76</td>
</tr>
<tr>
<td>History prior to baseline</td>
<td>– –</td>
<td>99 8</td>
</tr>
<tr>
<td>Episode between baseline and follow-up</td>
<td>– –</td>
<td>203 16</td>
</tr>
<tr>
<td>Recurrent episode</td>
<td>– –</td>
<td>128 10</td>
</tr>
<tr>
<td>First episode</td>
<td>– –</td>
<td>75 6</td>
</tr>
</tbody>
</table>

s.d., Standard deviation; SF-12, Short Form-12.

*a Women only.
Healthy Lifestyle Score as a predictor of mood disorder

Among the 1233 participants with data for these analyses, higher Healthy Lifestyle Scores at baseline protected against episodes of mood disorder over follow-up, particularly first episodes (Fig. 3). These analyses were adjusted for age, sex, education level, area-level disadvantage, time between follow-ups, history of cardiovascular disease, social support, having children, major life events and physical health-related quality of life. The relationship was such that each unit increase in the Healthy Lifestyle Score at baseline was associated with a 24% (RR 0.76, 95% CI 0.61–0.95) reduction in the risk of developing a first episode of mood disorder (n = 75) compared with a reference category of no new episode of mood disorder (n = 1158, Fig. 3, middle panel, black circle). The results were similar when the outcome compared first episodes (n = 75) with no lifetime history of mood disorder (n = 931, Fig. 3, final panel, RR 0.76, 95% CI 0.62–0.96).

Sensitivity analyses explored if these findings were influenced by mental health at baseline. Adjusting the models for baseline mental health-related quality of life made no appreciable difference to the magnitude of the results, irrespective of the outcome classification used (Fig. 3, grey circles). Further, using the subset (n = 986) with data on the 12-month version of the CIDI administered at baseline we excluded those with threshold (n = 83) or subthreshold (n = 122) depression, which somewhat strengthened the results. As shown in Fig. 3 (white circles), each unit increase in the Healthy Lifestyle Score at baseline was associated with a significantly reduced risk of any new (i.e. first or recurrent) mood disorder episode (RR 0.79, 95% CI 0.65–0.97), or a first-ever episode (RR 0.67, 95% CI 0.51–0.89) or a first-ever episode compared with no lifetime history (RR 0.70, 95% 0.52–0.92).

Recalculating the Healthy Lifestyle Score to include only certain items (Fig. 4, grey circles) did not affect any of the results, suggesting that no single item was driving these associations. We also examined the association between individual items and the risk of mood disorder (see online Supplementary Table S3). All items were associated with a reduced risk of new episodes of mood disorder, particularly first-ever episodes, but only non-smoking appeared protective when all other items were included in a mutually adjusted model along with confounding factors.

Applying inverse probability weights to account for loss to follow-up did not change the results for these analyses (online Supplementary Fig. S2).
Discussion

We found bi-directional associations between healthy lifestyles and mood disorders in young adults. People with healthier lifestyles at baseline were significantly less likely to develop a first episode of mood disorder over 5 years of follow-up. There was also a tendency for those with a history of mood disorder to have an unfavourable trajectory of their lifestyle over time.

This is the first study to consider the association between the number of health behaviours and risk of developing mood disorder over time. The association between healthy lifestyles and mood disorder was somewhat stronger for first than recurrent episodes. One potential explanation is the younger age of onset (around 10 years) between those with first and recurrent episodes in this study. There is some evidence that different risk factors are associated with development of mood disorders at different ages. For example, mood disorders with a younger age of onset are often associated with early childhood factors, such as perinatal insults and parental factors (Jaffee et al. 2002). Our data suggest that a constellation of healthy behaviours is associated with a lower risk of depression in adulthood. This supports the limited randomized controlled trial evidence showing that lifestyle modification reduces depressive symptoms in older people at high risk for cardiovascular disease (Rubin et al. 2014). Targeting the health behaviours of young adults before the peak onset of mood disorders could have benefits in terms of reducing mood disorders, along with reducing the burden of cardiovascular disease, but this requires testing in well-designed intervention studies at either the population or individual level.

One possible explanation for our finding was that mood disorders before baseline that were subthreshold, therefore not identified using the standard diagnostic criteria, may have already resulted in poorer lifestyles, in a sense ‘reverse causation’. Our analyses suggested that this was not the case, as excluding those who reported subthreshold or threshold mood disorders in the 12 months prior to baseline, or adjusting for baseline mental health-related quality of life, still showed that those with better lifestyles had a lower risk of mood disorder over follow-up. The robustness of the results was further evident from the similar effect sizes when only some health behaviours were included in the score. This suggests that no individual health behaviour or cluster of health behaviours was responsible for the associations. With that said, analyses of the effects of individual items did suggest that non-smoking was particularly protective. This reflects the complex inter-relationship between smoking and mood disorders, noting the ongoing controversy regarding whether this is a causal relationship (Chaiton et al. 2009) or the result of confounding (Bjorngaard et al. 2013). The similarity in effect sizes with items removed provides evidence that it is the

![Fig. 3. Healthy Lifestyle Score at baseline as a predictor of episodes of mood disorder between baseline and follow-up with adjustment for confounding factors and different measures of baseline mental health. Values are relative risk [95% confidence interval (CI)]. HRQoL, Health-related quality of life.](image-url)
Fig. 4. Sensitivity analyses examining Healthy Lifestyle Score (HLS) at baseline as a predictor of episodes of mood disorder between baseline and follow-up with exclusion of specific items from the HLS. Values are relative risk [95% confidence interval (CI)]. excl., Excluding; BMI, body mass index; LTPA, leisure time physical activity.
Bi-directional associations between healthy lifestyles and mood disorders in young adults

latent construct of a ‘healthy lifestyle’ that is important for mental health. Bearing in mind the observational nature of these data, our results suggest that a healthier lifestyle may protect against new-onset mood disorder. Importantly, the magnitude of the reduction in risk of mood disorder per healthy behaviour was similar to the effects of psychological interventions in a meta-analysis of trials for the primary prevention of mood disorders (Cuijpers et al. 2008). The findings are therefore relevant to those managing the physical or mental health of younger adults.

The potential mechanisms explaining why those with healthy lifestyles might be protected against mood disorder are governed by the contents of the score, which contains items known to protect against depression; for example, being healthy weight (De Wit et al. 2010), not smoking (Patton et al. 1998; Taylor et al. 2014), dietary factors (Smith et al. 2014; Jacka et al. 2015) and physical activity (McKercher et al. 2014). The mechanisms common across the healthy behaviours are the links with lower levels of inflammation, better immune system functioning and lower oxidative stress (Lopez-Garcia et al. 2004; Paul et al. 2004; Raison et al. 2006; Berk et al. 2011). These mechanisms, in turn, may affect pathways implicated in the development and progression of mood disorders such as the monoaminergic system (Chaouloff, 1997; Dani & De Biasi, 2001; Berk et al. 2011). The development of mood disorders and uptake and maintenance of health behaviours are complex and influenced by a range of genetic (Nabeshima & Kim, 2013; De Geus et al. 2014; Ware & Munafo, 2015) and environmental factors (Baler & Volkow, 2011; Nabeshima & Kim, 2013) that were not measured in this study. It is therefore possible that the results seen here are the result of residual confounding by shared genetic or environmental factors. This might mean that the associations seen here are not causal. However, with confirmation of our findings in other longitudinal studies or randomized controlled trials it is possible that we could consider including the potential mental health benefits of adopting health behaviours that prevent cardiovascular disease in health promotion messages.

We acknowledge the behaviour change can be difficult but note that in this cohort, albeit relatively young and socio-economically advantaged, about 60% of people either maintained or gained a high Healthy Lifestyle Score over 5 years. There is also some evidence that interventions to change multiple behaviours in younger adults can be successful (An et al. 2013, Valve et al. 2013) which is somewhat contrary to studies that include older people (Butler et al. 2013). Ideally, people would have healthy lifestyles from childhood and throughout adulthood with there being some evidence that multifaceted programmes (i.e. child and family) can have very long-term influences on lifestyle (Pahkala et al. 2013).

There was also evidence that a history of mood disorder before baseline was associated with unfavourable changes in lifestyle over 5 years. This novel finding in a cohort of generally healthy young adults supports the literature on the bi-directional associations between mental health and single risk factors. It also reinforces recent analyses demonstrating that health behaviours accounted for much of the association between depression and cardiovascular disease albeit in older people (Ye et al. 2013). There are many potential mechanisms linking a history of mood disorder with worsening lifestyle, including self-medication with alcohol (Dixit & Crum, 2000) and cigarettes (Patton et al. 1998) in an attempt to manage symptoms. The symptoms associated with mood disorder such as psychomotor retardation or alterations in appetite could also result in worse scores on items for physical activity and diet (Wenzel et al. 2005; Simon et al. 2008). This finding highlights the need for more recognition of the close link between lifestyle and mental health and the need for a ‘whole of person’ approach to these conditions (Baird & Clarke, 2011).

As noted by others (Ye et al. 2013), addressing the lifestyles of people with mood disorders is imperative for reducing their risk of physical illness and may, indeed, ameliorate their symptoms of mood disorder if our findings regarding lifestyle predicting depression are confirmed in intervention studies.

This study has limitations that should be acknowledged. There was substantial loss to follow-up. We found that those included were of higher socio-economic status and had healthier lifestyles than those not included and compared with the general Australian population of a similar age. We addressed this issue by using inverse probability weights and showed this loss to follow-up appeared to only have a minor impact on our findings. Further, the validity of associative analyses is not reliant on a generalizable sample; rather, what is important is that the cohort has well-characterized participants; adequate sample size; and heterogeneity of determinants, modifiers and confounders, as is the case for our cohort (Miettinen, 1985). Nonetheless, if the methods we used have not adequately accounted for the attrition in the study then it is possible that our findings are only applicable to relatively healthy, higher socio-economic-status individuals. Further, the study was based on younger individuals, which may be a strength in terms of less confounding by chronic conditions, but this may mean results are not generalizable to older people. Assessments of mood disorders were retrospective using the lifetime version of the CIDI at follow-up. Repeat assessments over shorter time periods may
Bi-directional associations between the number of healthy behaviours and mood disorders were found in our cohort of young adults followed for 5 years. This highlights the need for holistic management of young adults in terms of their mental and physical health including health behaviours. Our results suggest that achieving and maintaining a healthy lifestyle will not only reduce cardiovascular disease but also promote good mental health.

**Supplementary material**

For supplementary material accompanying this paper visit http://dx.doi.org/10.1017/S0033291716000738

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S.L.G. designed, conducted and interpreted the analyses, drafted the manuscript and contributed to the acquisition of data. K.S., K.J.S., G.P., T.D. and A.V. interpreted analyses, contributed to the acquisition of data, provided intellectual content and contributed to the drafting of the manuscript. All authors approved the final version of the manuscript.

**Declaration of Interest**

None.

**References**


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