



# Healthy minds live in healthy bodies – effect of physical health on mental health: Evidence from Australian longitudinal data

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Accepted: 25 March 2022 / Published online: 4 April 2022  
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## Abstract

It is well known that physical and mental health are closely related, with growing evidence for biological and behavioural pathways. Mostly the research has focussed on mental health as the key driver of this inter-connection; the extent physical health shapes mental health has received less attention. We aim to derive robust estimates of the unique role physical health may play in shaping mental health outcomes. To do so we use a novel approach, incorporating longitudinal and instrumental variable methods which can address the reciprocal relationship between physical and mental health, and the endogeneity of physical health, before estimating the physical to mental health pathway. A sample of 209,442 observations (or 24,966 unique individuals) aged 15 and over spanning 18 years (2002–2019) was extracted from the Household Income and Labour Dynamics in Australian Survey (HILDA). We find that physical activity and health shocks erode mental health via their impact on physical health with a one point improvement (or worsening) in physical health scores (0–100) resulting in a rise (or decline) of 0.43 points (or 43%) in mental health score.

**Keywords** physical health · mental health · instrumental variable · Australia

## Introduction

Mental and physical health goes hand in hand, and it is widely agreed that they can be interrelated. However, most studies of their interplay have focused on effects of mental health on physical health, with less attention paid to the reverse (i.e., impact of physical health on mental health). This mental to physical health pathway was highlighted in Prince et al. (2007) ‘*No health without mental health*’, and a range of studies demonstrated how shocks to mental health from job loss, loss of family or relatives, family violence, abuse and bullying would change health behaviours (sleep, eating, and exercise) and hence underlying physical health (e.g., increased blood pressure, heart rate, decreased mobility, pain, migraines etc.). Such studies trace the pathways and associations, often starting from broader social and economic circumstances, which through their impact on mental

health generate physical illness (Fadlon & Nielsen, 2019; Hoang et al., 2019; Margolis, 2013; Saxena et al., 2005; Averina et al., 2005; Lasser et al., 2000; Spiegel & Giese-Davis, 2003; De Groot et al., 2001).

## Mental and physical health pathways

There are, however, multiple linkages and potential pathways between mental and physical health. For example, positive psychological well-being can help reduce the risk of serious physical problems including heart attacks and strokes (Vázquez et al., 2009). In contrast, poor mental health can lead to damaging behaviors or inhibit health behaviors (substance abuse, physical inactivity for instance), leading to poor physical health. For instance, people with mental illness are much more likely to suffer from sleeping disorders (Averina et al., 2005), and they are more likely to smoke than people without a mental illness (Lasser et al., 2000). This in turn increases risks for health concerns such as cancer or cardiovascular diseases (Fadlon & Nielsen, 2019; Hoang et al., 2019; Margolis, 2013; Saxena et al., 2005). Depression is also found to be linked to some chronic diseases, such as cancer (Spiegel & Giese-Davis, 2003), diabetes (De Groot et al., 2001) and asthma (Di Marco et al.,

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2010) and has been considered by some to have a causal role (e.g., Maghout-Juratli et al., 2010). It is also argued that people with mental illness may find it difficult to access adequate healthcare (Eisenberg et al., 2007), compounding their physical health status. Vaillant (1979) showed that better mental health in the past was strongly linked to good current physical health. This relationship was still strongly positive even after controlling for alcohol and tobacco use, and obesity. Poor mental health increase risk for communicable and non-communicable diseases, unintentional and intentional injuries (Prince et al., 2007).

In many studies the direction of influence is assumed to be from mental to physical health. However, some findings point to a complex and possibly reciprocal relationship. According to a 20-year follow-up study conducted by Paffenbarger et al. (1994) on 21,569 Harvard alumni, the depression rate was lower among participants with regular physical activity such as sports players and higher among those who are smokers or heavy drinkers, who are classified as poor physical health. Similarly, Camacho et al. (1991), using three waves of data of the Alameda County during 1965–1983, found a lower risk of depression among those with better physical health and regular physical activity. An investigation in the middle-age population in the Biobank, UK found that people who had more than one physical health condition (multi-morbidity), tended to have worse mental health than their physically healthier counterparts (Ronaldson et al., 2021). Further, Ohrnberger et al. (2017a) investigated 11,203 older adults from 2002 to 2013 and found that better physical health in the past has a strong association with current mental health, and the relationship was even stronger than that from education or physical activity.

Another potential pathway is via adverse shocks or events such as accidents or traumatic injuries, which might worsen physical and mental health simultaneously or through the distress caused by the physical injuries or conditions and functional impairments (Kessler et al., 1997; Flett et al., 2002; Holman et al., 2008; Koren et al., 2001; Ullman & Siegel, 1996). Trauma is also the cause of some chronic and acute physical disorders including cardiovascular illness (Geerse et al., 2006) or immune functioning (McEwen, 2005) that may in turn affect mental health in the long term. Olafson et al. (2021) showed that ICU hospitalized patients used more medications for their mental health than before their hospitalization, and also more than the general population or non-ICU hospitalized cohorts.

Very few studies address the possibility of reciprocal causality between mental and physical health, despite this being integral to theories such as allostatic load. Chronic stresses, or physical shocks and trauma can alter body physiology including the endocrine system (McEwen & Wingfield, 2003). For example, Miller et al. (2007) found that cortisol levels for trauma exposed individuals were related to timing

after the events, and that trauma events create both too much and too little cortisol, and then health outcomes related to both too much and too little cortisol. Trauma events also result in rising heart rate, and rising blood pressure and even lead to long-term high blood pressure (Pole, 2007; Buckley & Kaloupek, 2001), affecting the immune system (McEwen, 2005), increasing risk of musculoskeletal disorders after health shocks such as accidents (Dorn et al., 2008). Acute trauma affects adversely physical health as well as mental health, and this is widely evidenced in the literature (D'Andrea et al., 2011).

### Methodology to address reciprocal pathways and endogeneity

Both theory and evidence point to complex, reciprocal linkages between mental and physical health. However few psychological or population health studies explicitly model them, and current estimates of the direction and strength of influence are likely biased. Ohrnberger et al. (2017b) looking at the relationship between physical and mental health, would be the most comprehensive study to date. They analysed the direct and indirect effects of past mental health on present physical health, and past physical health on present mental health using lifestyle choices and social capital in a mediation framework. However, their model (simultaneous equation modelling-SEM) could only provide estimates of relationships between two health measures but not the direction of influence because the SEM design does not allow for causal inference (Dilalla, 2000). The SEM methodology is not able to address bias caused by potential reverse causality between mental health and physical health variables, a gap this current study seeks to address. We therefore incorporate time-differenced estimator and longitudinal instrumental variable (IV) methods into our modelling of physical and mental health associations, using the Australian nationally representative longitudinal data.

The longitudinal instrumental variable (IV) method can correct for biases caused by endogeneity of physical health, and the reverse relationship between mental health and physical health. These methods are widely used in economic research, and are potentially valuable techniques for psychology and population health research, where reciprocal and reverse relationships are likely important for testing assumptions of causality. Most existing public health studies use Randomised Control Trials (RCTs) or experiments to estimate directions of influence. Unlike RCTs, the IV method is a quasi or natural experimental method using available secondary datasets (see Angrist & Krueger, 2001; Wooldridge, 2010) and can address the bias caused by endogeneity of physical health and reverse causality between mental health and physical health before modelling the effect of physical health on mental health. Unlike RCT, which is often

expensive to implement, the quasi experiment approach enables effective use of existing data sets, a less costly design option. Using this method we seek to robustly estimate the unbiased or pure effects of physical health on mental health, adjusting for reverse and reciprocal relationships to test the following hypotheses:

### Research hypotheses

- H<sub>1</sub>. Physical exercise and health shocks e.g., accidents strongly affect physical health
- H<sub>2</sub>. Changes in physical health status strongly impact mental health

## Data and method

### Data and analysis sample

The data used in current analysis is from the Household, Income and Labour Dynamics in Australia (HILDA) survey. The HILDA survey is a household-based longitudinal study of a nationally representative sample of Australians, surveying more than 7000 households and 17,000 individuals each year since 2001. The survey combines detailed information on household and individual circumstances with measures of health, employment, income, health shock events. Attrition rates are low; with more than 90% of respondents in any wave responding in the next wave. For a detailed discussion of the survey design, see Watson and Wooden (2012) and for user manuals, technical papers and related publications, see <https://melbourneinstitute.unimelb.edu.au/hilda>.

Because some of our variables used in the analysis are not available in wave 2001 (e.g., smoking status), we restricted our sample to waves 2–19 (2002–2019). Our analysis was restricted to individuals aged 15 and over. The final data set has 209,442 observations (from 24,966 unique individuals).

### Measures of mental health and physical health

Our outcome of interest is *mental health* (MH), which was constructed from five questions from the Short Form 36 (SF-36) (Ware et al., 2000), which is included in the HILDA survey. Three items assess nervousness and depression ('Have you been a very nervous person?', 'Have you felt so down in the dumps that nothing could cheer you up?' and 'Have you felt downhearted and blue?') and the other two happiness and calm ('Have you felt calm and peaceful?' and 'Have you been a happy person?'). The score for mental health was calculated by summing scores of these above 5 questions and then transforming to a 0–100 scale, with a higher score representing better mental health. The primary variable of interest is *physical health component score* (PCS) from the

SF-36, which covers 21 questions divided into several components including *physical functioning* (constructed from 10 questions about activities such as lifting, carrying, climbing, bending, kneeling, walking, bathing...), *physical limitations* (or role physical, constructed from 4 questions about physical limitations that affect work or activities: 'Cut down the amount of time spent on work or other activities'; 'Accomplished less than would like'; 'Were limited in the kind of work'; 'Had difficulty performing work or other activities'), *bodily pain* (2 questions about bodily pain and how it interferes with normal work), and *general health* (5 questions about self-assessed health and one's own health relative to others' health such as 'Get sick a little easier than other people'; 'As healthy as anybody I know'; 'Expect my health to get worse'; 'My health is excellent'). PCS is obtained by averaging over the component scores. These component scores were computed using the same method applied for mental health above.

### Instrumental variables

One of our models was an instrumental variable (IV) estimator (we will provide detailed discussion in Section 3.3 below) in which we used lag of physical activity level, lag of long term health condition, and length of health shock events (a duration between year the health shock occurred to contemporary year) as instruments to predict physical health (PCS) before estimating the effect of PCS on MH. *Physical activity* was frequency of physical activity per week. Participants responded to a six-category question "In general, how often do you participate in moderate or intensive physical activity for at least 30 min?": (1) Not at all; (2) Less than once a week; (3) One to two times a week; (4) Three times a week; (5) More than 3 times a week (but not every day); and (6) Every day. We regrouped these responses into 4 categories: sedentary (combining 1 and 2), low (combining 3 and 4), moderate (5), and high activity (6). We used 4 categories to capture physical health-relevant variation in activity, and used lag of physical activity to avoid the reciprocal relationship between current physical activity and PCS. The lag of the response to a question on *long-term health condition* (yes/no; 'do you have any long-term health condition, impairment or disability that restricts you in your everyday activities, and has lasted or is likely to last, for 6 months or more?') to predict physical health was also included. The lag was used to address the reciprocal relationship between the long term health condition and physical health. The last instrument was the length from shock events to physical health. We used the length (years) to explore the lasting effect of the shock events to physical health. For people who had no physical health shocks, the length was set to null (year). These events include conditions affecting the use of feet and legs, arms and fingers, restricting physical activity and work, causing back pain, migraines, chronic or

recurring pains, developing long-term effect as result of head injuries, stroke or brain damage. We tested the IV relevance and exclusion assumptions, and the results showed that these IVs are valid and are very strong predictors of physical health (see more in the Result section below).

Table 1 presents basic statistics for key variables for the analysis sample. Because we used different levels of variables’ lags in a series of model specifications, only the contemporary value for these variables is presented.

### Estimation methods

The determinants of mental health have been well explored in the existing literature (Allen et al., 2014; Maunder & Hunter, 2008). They include physical activity (Ohrnberger et al., 2017b), social attachment (Maunder & Hunter, 2008), education (Ohrnberger et al., 2017a), and income and employment (Stuckler et al., 2009). The relationship between physical health and mental health can be explained by two main underlying mechanisms: namely psychological channels and biological channels (Gaudlitz et al., 2013). From the psychological point of view, physical health can boost individual self-esteem by the appreciation of other people or the society on the level of fitness or physical abilities, physical self-concept, etc. that helps to protect them from anxiety and negative feelings (Knapen et al., 2005). Moreover, good physical functioning is commonly associated with social engagement that is shown to be protective from mental health problems, and high levels of leisure-time activities such as physical exercise can help mitigate the risk of depression or anxiety (Pasco et al., 2011). From the biological perspective, good physical health is also believed to increase the availability of the enzyme tryptophan in the brain, which stimulates serotonin synthesis and helps to mitigate anxiety or mental disorders (Gaudlitz et al., 2013).

Mental health (MH) disorders such as psychological distress, depression or anxiety are also affected by numerous factors apart from physical health status including individual and household demographic and economic profiles such as age, gender, education, race, language barrier, marital status, employment status, income, surrounding environment such as socio-economic disadvantage as well as regional-specific factors such as regional unemployment rate (Andrade et al., 2014; Cohen et al., 2009; Caldwell et al., 2004; Norstrand et al., 2013; Nieuwenhuijsen et al., 2010; Warr, 1984).

Our mental health prediction model is based on individual and household demographic and socio-economic characteristics and physical health as discussed above. Accordingly, the model for MH prediction is as follow:

$$MH_{it} = \alpha + \beta_1.PCS_{it} + \beta_2.X_{1it} + \beta_3.X_{2it} + \epsilon_{it} \tag{1}$$

Where  $MH_{it}$  is mental health score of individual  $i$  in year  $t$ .  $PCS_{it}$  is physical health component score ( $PCS$ ),  $X_{1t}$  is a

**Table 1** Descriptive statistics aged 15–101, 2002–2019.

| Variable   | Mean   | Std Dev |
|--|--------|---------|
| Mental health score (MH)                                   | 73.69  | 17.39   |
| Physical health component score (PCS)                      | 75.31  | 22.03   |
| <i>Migration background (%)</i>                            | 100%   |         |
| Native-born  | 71.94  | 44.93   |
| English speaking countries                                 | 10.14  | 30.19   |
| Non-English speaking                                       | 17.92  | 38.35   |
| Age  | 44.02  | 18.82   |
| Sex (men = 1/women = 0)                                    | 0.49   | 0.50    |
| Marital status (married/de facto = 1)                      | 0.61   | 0.48    |
| <i>Education (%)</i>                                       | 100%   |         |
| Post graduate (masters/doctors)                            | 4.83   | 21.44   |
| Graduate diploma/graduate certificate                      | 4.98   | 21.76   |
| Bachelor/honours   | 14.02  | 34.72   |
| Advance diploma, diploma                                   | 9.01   | 28.63   |
| Cert III/IV  | 20.17  | 40.13   |
| Year 12  | 15.92  | 36.59   |
| Year 11 and below  | 31.07  | 46.28   |
| <i>Labour force status (%)</i>                             | 100%   |         |
| Employed   | 63.01  | 48.28   |
| Unemployed   | 3.38   | 18.08   |
| Not in labour force  | 33.61  | 47.24   |
| Home ownership (owned = 1/rented and other = 0)            | 0.71   | 0.45    |
| Socio-economic index SEIFA (1–10)                          | 5.65   | 2.87    |
| Regional unemployment rate (%)                             | 5.11   | 0.96    |
| <i>Equivalentized household disposable income (\$1000)</i> |        |         |
| Quintile 1   | 18.81  | 18.32   |
| Quintile 2   | 32.91  | 4.52    |
| Quintile 3   | 45.10  | 5.27    |
| Quintile 4   | 60.02  | 7.55    |
| Quintile 5   | 109.51 | 64.78   |
| Urban (yes = 1)  | 0.88   | 0.32    |
| <i>Alcohol drinking (%)</i>                                | 100%   |         |
| Never drink  | 13.30  | 33.96   |
| Very rare/no longer drink                                  | 29.87  | 45.77   |
| Moderate drinker   | 47.33  | 49.93   |
| Heavy drinker  | 9.49   | 29.31   |
| <i>Smoking (%)</i>   | 100%   |         |
| Never smoke  | 55.27  | 49.72   |
| Past smoker  | 26.66  | 44.22   |
| Current/but not daily                                      | 3.50   | 18.38   |
| Daily smoker   | 14.56  | 35.27   |
| <i>Instruments</i>   |        |         |
| <i>Physical activity (%)</i>                               | 100%   |         |
| Sedentary  | 27.88  | 44.84   |
| Low  | 39.31  | 48.84   |
| Moderate   | 20.86  | 40.63   |
| High   | 11.95  | 32.43   |
| Long-term health conditions (yes = 1)                      | 0.28   | 0.45    |
| Length of physical health shock (years)                    | 2.62   | 7.86    |

Authors’ estimation from HILDA 2002–2019 sample of 274,104 observations. Estimates were adjusted for sample weights. Our models also controlled for state and year dummy variables, but are omitted for succinctness.

vector of the individual and household characteristics (age, age squared, gender, marital status, migration background, education, smoking, alcohol drinking, labour force status, home ownership, household equivalized disposable income, household socioeconomic disadvantage deciles).  $X_2$  is a vector of extra control variables including regional unemployment rate, urbanity, and state and year dummy variables.

There is a potential mutual or reciprocal relationship between PCS and MH. The endogeneity of PCS suggests a non-zero correlation between PCS and error terms in the Ordinary Least Squares (OLS) estimation of eq. (1), resulting in bias in the estimated coefficient of the effect of PCS on MH.

To overcome this bias in the OLS estimate, we employed a series of estimation techniques. *First*, we used the first and second lags of PCS (prior physical health) in place of PCS in eq. (1). This approach ensures PCS is pre-determined or exogenous to contemporary mental health (MH) status. *Second*, longitudinal models (e.g. fixed effect (FE)) were used to estimate the effect of PCS variation within individuals on MH variation over time. The longitudinal methods can address the bias resulting from unobserved heterogeneity. However, longitudinal strategies cannot address reverse causality (Lakdawalla & Philipson, 2007). *Third*, a similar approach to the FE model is the time-differenced estimator that estimates the effect of change in PCS on change in MH, but this approach also fails to address reverse causality. We modified this approach by adding the first and second lag of the variation (time-differences) in PCS. This approach would capture the lasting effect of physical health on mental health. Further, longer differenced estimators (in this paper, we used one-year and then two-year differences)<sup>1</sup> can reduce the bias from measurement error (Greene, 1993; Griliches & Hausman, 1986). *Finally*, we applied an instrumental variable (IV) estimator to identify the causal relationship. In this method, physical health (PCS) is instrumented by physical activity level (lag), long-term health conditions (lag),<sup>2</sup> and length of shocks to physical health and its squared term (to capture PCS worsening and then recovering after the health shocks) in the first stage of estimation.<sup>3</sup> These IVs are more likely to affect physical health conditions (relevance assumption), but the residuals/error terms in the model of MH are unlikely to be correlated (directly) with these factors (exclusion assumption). These three IVs passed the tests for the relevance and exclusion assumptions so that they are valid

instruments (see more details in Section 4). In the second stage, the predicted PCS was included in estimation equation to estimate the causal effect of PCS on MH. Because the standard IV estimator assumes the error terms are homoscedastic and non-autocorrelated, this can lead to inconsistent estimates of standard errors. We thus applied GMM (generalized method of moment) IV regression to obtain consistent estimates of coefficients and asymptotic standard errors. The advantage of GMM IV over standard IV estimator is that if heteroskedasticity is present, the GMM estimator is more efficient than the standard IV estimator, whereas if heteroskedasticity is not present, the GMM estimator is no worse asymptotically than the IV estimator (Baum et al., 2003).

## Results

We *first* started with a simple Ordinary Least Squares (OLS) estimator in which we controlled for a full range of observed risk factors associated with mental health. *Second*, we modified the OLS model to include lags of PCS to address reverse causality between PCS and MH (mutual effect). *Third*, we ran Fixed Effect (FE) and time-differenced estimators (first and second time-differenced). *Fourth*, we employed the IV estimator which we believed to provide the most reliable estimates of the causal effects in our data.

Although we used various approaches, we expected estimates to provide consistent findings (although magnitudes of estimates from different models may vary, as each model captures different levels of health variation, as well as having different sample sizes).

### OLS estimates

The first column in Table 2, tells us that MH is associated with PCS, each score increase in PCS is associated with about 0.4 score points increase in MH. In columns 2 and 3, each score rise in the range (0–100) leads to an increase of 0.3173–0.3309 score points. When we added both first and second lag of PCS in the last model (column 4), the total effect of each score rise in PCS on MH is about 0.38 score points (0.2206 + 0.1627). All of the estimates are highly statistically significant at 1% confidence level (or  $p < 0.0001$ ). The OLS estimates especially in column 1 are likely biased due to endogeneity of PCS, that is, correlation between PCS and the error term of eq. 1 (due to unobserved confounding factors).

### Longitudinal approach and time-differenced estimates

Fixed effect and time-differenced estimates using one-year and two-year differences show a very consistent finding

<sup>1</sup> One may want to use longer time differences but it reduces sample size significantly.

<sup>2</sup> We also ran a model with contemporary physical activity and long-term health conditions, producing consistent effects of these factors on PCS. Details are available upon request.

<sup>3</sup> We also ran a model using a dummy variable for *prior* physical health shocks, with modest changes to the results.

**Table 2** OLS estimates with contemporary and lags of physical health.

| Dependent variable: Mental health (MH) | (1)                  | (2)                  | (3)                  | (4)                  |
|--|----------------------|----------------------|----------------------|----------------------|
| PCS                                    | 0.3996**<br>(0.0019) |                      |                      |                      |
| PCS (lag)                              |                      | 0.3309**<br>(0.0021) |                      | 0.2206**<br>(0.0030) |
| PCS (second lag)                       |                      |                      | 0.3173**<br>(0.0022) | 0.1627**<br>(0.0030) |
| Observations                           | 209,442              | 199,248              | 175,227              | 169,646              |
| R-squared                              | 0.2700               | 0.2061               | 0.1950               | 0.2262               |
| Prob > F                               | 0.0000               | 0.0000               | 0.0000               | 0.0000               |

Robust standard errors in parentheses, significant \*\*  $p < 0.01$ , \*  $p < 0.05$ , +  $p < 0.1$ . All models controlled further for migration background, marital status, age, sex, education, smoking, alcohol drinking, employment status, household income level, house ownership, socio-economic disadvantage, regional unemployment rate, urbanity, state and year dummy variables.

that physical health significantly affects mental health. The estimates of the effect of PCS on MH on columns 1, 2 and 4 are very consistent both in terms of magnitude and sign (a small variation in coefficients is due to changes in sample size). However, FE or time-differenced models may

still give biased estimates due to a reciprocal relationship between change in MH and change in PCS. We are not able to definitively attribute the change in MH to the change in PCS. To overcome this, in columns 3 and 5, we allowed changes in PCS *prior* to changes in MH by adding lags of change in PCS. This enables us to more confidently make inferences that a physical health change leads to a change in mental health. The estimates show that changes in PCS result in changes in MH, although the effect is smaller. This is expected because the lags of time-difference in PCS create larger time gaps between when physical health is observed and when mental health is observed. The effect of PCS on MH may therefore fade out (Table 3).

**Instrumental variable (IV) estimates**

We employed the GMM IV estimator in a joint estimation procedure to estimate the effect of physical health (PCS) on mental health (MH) and the results are presented in Table 4.

In IV model, the population is divided into subgroups ( $g$ ) who share the same values for unobserved attributes. Suppose an intervention leads to a change in mean PCS of group  $g$  ( $\Delta PCS_g$ ), and let  $\beta_{1g}$  is the effect of PCS on MH of group  $g$  when there is no intervention. Suppose the intervention (the instruments) affects only treatment group who are

**Table 3** Fixed effect and time-differenced estimator.

| Variable                     | (1)<br>FE            | (2)<br>One-year difference ( $\Delta_1$ .MH) | (3)                  | (4)<br>Two-year difference ( $\Delta_2$ .MH) | (5)                  |
|------------------------------|----------------------|--|----------------------|--|----------------------|
| PCS                          | 0.2625**<br>(0.0032) |  |                      |  |                      |
| $\Delta_1$ .PCS              |                      | 0.2182**<br>(0.0025)                         | 0.2185**<br>(0.0033) |  |                      |
| $\Delta_1$ .PCS (first lag)  |                      |  | 0.0144**<br>(0.0035) |  |                      |
| $\Delta_1$ .PCS (second lag) |                      |  | 0.0007<br>(0.0031)   |  |                      |
| $\Delta_2$ .PCS              |                      |  |                      | 0.2386**<br>(0.0027)                         | 0.2320**<br>(0.0034) |
| $\Delta_2$ .PCS (first lag)  |                      |  |                      |  | 0.0093**<br>(0.0029) |
| $\Delta_2$ .PCS (second lag) |                      |  |                      |  | 0.0088**<br>(0.0032) |
| Observations                 | 209,442              | 198,343                                      | 144,809              | 174,452                                      | 124,287              |
| R-squared                    | 0.1958               | 0.0543                                       | 0.0531               | 0.0658                                       | 0.0634               |
| Unique observations          | 24,966               |  |                      |  |                      |
| Prob > F                     | 0.0000               | 0.0000                                       | 0.0000               | 0.0000                                       | 0.0000               |

Robust standard errors in parentheses, significant \*\*  $p < 0.01$ , \*  $p < 0.05$ , +  $p < 0.1$ . Dependent variable is mental health. All models controlled further for migration background, marital status, age, sex, education, smoking, alcohol drinking, employment status, household income level, house ownership, socio-economic disadvantage, regional unemployment rate, urbanity, state and year dummy variables.

**Table 4** GMM instrumental variable estimates of effect of PCS on MH.

| Variable  | (1)<br>1st stage       | (2)<br>2nd stage     |
|---|------------------------|----------------------|
| PCS   |                        | 0.4315**<br>(0.0038) |
| <i>Excluded instruments</i>   |                        |                      |
| Physical activity (lag) - Sedentary ( <i>reference group</i> )  |                        |                      |
| Low   | 4.8320**<br>(0.1005)   |                      |
| Moderate  | 7.2610**<br>(0.1127)   |                      |
| High  | 8.3306**<br>(0.1343)   |                      |
| Long-term health condition (yes = 1) (lag)  | -15.3568**<br>(0.1175) |                      |
| Length of PCS health shock  | -0.9692**<br>(0.0143)  |                      |
| Length of PCS health shock-squared  | 0.0129**<br>(0.0003)   |                      |
| Observations  | 198,346                | 198,346              |
| Centered R-squared  |                        | 0.2684               |
| Uncentered R-squared  |                        | 0.9625               |
| Prob > F  | 0.0000                 | 0.0000               |
| IV tests  |                        |                      |
| Endogeneity test of the endogenous regressor (PCS), GMM C statistics and [P value]  | 93.80** [0.0000]       |                      |
| Test for all included and excluded IVs jointly equal zero in the first stage F-value and [P- value]   | 1939.9** [0.0000]      |                      |
| Test for excluded IVs jointly equal zero in the first stage F-value and [P value]   | 7835.9** [0.0000]      |                      |
| Weak instrument test, Anderson-Rubin Wald test F-statistics and [P value]   | 1887.9** [0.0000]      |                      |
| LM test of redundancy of excluded IVs, chi-squared(6) and [P- value]  | 2.7e+04** [0.0000]     |                      |
| Weak identification test (equation is weakly identified), Kleibergen-Paap rank Wald F-statistic [Stock-<br>Yogo weak ID test critical values-5% maximal IV relative bias] | 7835.9** [19.28]       |                      |
| Sargan and Basmann tests for overidentification Hansen's J chi-squared and [P value]  | 637.4** [0.0000]       |                      |

Robust standard errors in parentheses, significant \*\*  $p < 0.01$ , \*  $p < 0.05$ , +  $p < 0.1$ . Dependent variable in the first stage is PCS, in the second stage is MH. is mental health. All models controlled further for migration background, marital status, age, sex, education, smoking, alcohol drinking, employment status, household income level, house ownership, socio-economic disadvantage, regional unemployment rate, urbanity, state and year dummy variables.

identical to those in comparison group, that is, both groups those have the same unobserved attributes would have the same PCS level and MH outcome in the absence of the intervention. The instrument e.g., shocks to health play a role like intervention that is why IV method is named as a quasi or natural experiment.

The validity of the IV estimator relies heavily on an assumption that the instruments  $Z_s$  (intervention) are uncorrelated with unobserved attributes of individuals that affects MH, that is,  $cov(Z_i, \varepsilon_i) = 0$ . In the case of experiment of  $Z_i$  (random assignment of the intervention), the difference in mean MH between treatment/intervention and control/comparison group will not be exacerbated by IV estimator, but this is not completely the case of quasi or natural experiments (Card, 1999). This problem is the limitation of IV

estimator. The IV estimates in the presence of weak instruments (weakly correlated with PCS, but possibly correlated with the residual of the MH equation), the estimates would be very imprecise and seriously inconsistent (Belzil, 2007). Thus, the weak IV test is needed to ensure that IV estimation does not provide imprecise estimates of effect of physical health on mental health.

We conducted a series of the IV tests and the results are presented in the bottom panel of Table 4. The first test was an endogeneity test of the endogenous regressor (PCS), and its null hypothesis is that PCS is exogenous. The test result rejected the null hypothesis ( $p < 0.0001$ ) suggesting that physical health PCS is endogenous and the OLS estimator for eq. (1) produces a biased estimate of the effect of PCS on MH. The next test is to test the power

of prediction of the three excluded instruments  $Z$  in the first stage of estimation. The hypothesis of the instruments jointly equalling zero in the first stage was rejected (with a very high  $F$ -value of 7835.9,  $p < 0.0001$ ), and the test for weak instruments (Anderson-Rubin Wald test) rejected the hypothesis of weak instruments ( $p < 0.0001$ ). Further, all coefficients of excluded instruments in the first stage estimation (column 1, Table 4) are significant at the 1% level. We also conducted a test for IV redundancy to see if any of the excluded IVs are redundant, and the test showed that the IVs were not redundant ( $p < 0.0001$ ). Another important test was to test if the equation is weakly identified. The Kleibergen-Paap rank Wald  $F$ -statistic of 7835.5 was much higher than the Stock-Yogo weak identification test critical values at 5% maximal IV relative bias of 19.28. This suggested that our model equation is not weakly identified. Finally, the Sargan and Basman test for over-identification shows that our model is over-identified (that is, we have more valid IVs than number of equations, in our case two equations). In sum, all of the tests that were conducted to determine the validity of the IV model indicate that we have identified strong instruments that enable us to be confident in our IV estimates presented in Table 4.

Our IV estimates show that higher levels of *prior* physical activity were a strong predictor of PCS. For example, comparing to those having a sedentary physical activity level, people who engaged in moderate or intensive physical activity one to three times a week (low physical activity) were about 4.8 points higher in PCS, which increased to 7.3 points for those who exercised more than 3 times a week (moderate physical activity), and 8.3 points for those who exercised every day (high physical activity). Having *prior* long-term health conditions was associated with a decline of 15.4 points in their PCS. For people who had physical health shocks their PCS declined by 0.97 points per year since the shock occurred. However, PCS decline flattened and recovered after some time as the squared term of the time since shock is positive and statistically significant. In a model using a dummy variable for lag of physical health shocks, it showed that following one year after the shock, PCS declined by 11.3 points (we did not report the full estimates here but will be available upon request).

After adjusting for the endogeneity of physical health using the IV method, our estimate showed that physical health strongly affects mental health. Each point improvement in PCS led to an increase of 0.43 points in MH. In contrast, worsening physical health by one point resulted in a decline of 0.43 points in mental health. Any shocks that are harmful to people's physical health appear to cause significant deterioration to their mental health.

## Conclusion and discussion

The main body of mental health research focuses on individual, household and socioeconomic risk factors as predictors of mental health, or on the impact of mental health on physical health. Another body of the literature looks at the simple relationship or correlation between physical and mental health. Much less is known about the causal influence of physical health on mental health. In this paper, we used comprehensive estimation methods to address the reciprocal relationship between physical health and mental health, and endogeneity of physical health.

There has been a paradigm shift on how health is conceived: mental and physical health are now viewed as interlinked and mutually influential. However, research evidence and approaches have lagged behind this shift, especially research on the extent physical health shocks and conditions are determinants of mental health conditions and symptoms. A key barrier has been the lack of feasible methods that can robustly test for influence by modelling these complex endogenous and reciprocal pathways. In this paper, we used comprehensive estimation methods (time-differenced and IV estimators) drawing from econometric approaches and applied them to this public health problem. These methods help address the reciprocal relationship between physical health and mental health, and endogeneity of physical health. We found that factors such as physical exercise, long-term health conditions and shocks to physical health strongly affect physical health. Any improvement (or worsening) in physical health then strongly affects mental health afterward. Based on responses to health questions from the SF-36, we found that a one-point improvement (or worsening) in physical health scores (0–100) will result in a rise (or decline) of 0.43 points (or 43%) in mental health score.

Our findings, based on modelling systems of influence, provide estimates of likely causal paths and shed new light on the significance of physical health-related factors as drivers of mental health. For example, Ohrnberger et al. (2017b), Roberts et al. (2018), and Ronaldson et al. (2021) found the relationship between poor physical health and mental health illness, depression, anxiety or risk for mental health disorders. Our finding delivers a precise estimate of this causal effect of physical health on mental health, consistent with Olafson et al. (2021) evidence that that worsening physical health due to trauma events or hospitalization leads to higher mental health medication. Their study was on a cohort of ICU-hospitalized patients, while our analysis was conducted on a nationally representative sample of Australian population aged 15 and over.

The instruments in the IV model such as *prior* (or lag of) physical exercise and shocks to physical health



were found to have strong effects on individual physical health status. These instruments play a role as mediators between physical health and mental health. The effect of these instruments on physical health is consistent with the existing literature. For instance, D'Andrea et al. (2011) found that shocks to health such as trauma affected adversely physical health. Acute trauma exposure leads to negative physical health consequences such as significant disruption to gastrointestinal functioning, the cardiovascular system, immunological functioning, the reproductive system, the musculoskeletal system, neuroendocrine functioning, brain structure and functioning. Camacho et al. (1991), Gaudlitz et al. (2013) and Ohrnberger et al. (2017b) showed that physical activity helped reduce risks for physical health and hence mental health.

Our analysis using IV models (quasi or natural experiment) goes beyond just looking at simple association between physical health and mental health. It offers causal relationship inference for policy on mental health that could be both feasible and (relatively) immediate where there is existing data. Adapting this analytic strategy would benefit other areas where random controlled trials (RCT) and experimental methods may be practically impossible or prohibitively expensive, and may take many years to undertake.

In Australia, a recent study (Young et al., 2017) found a significant correlation between physical health and mental health status. The study indicated that the majority of participants reported the awareness of the importance of physical health to their mental health and wellbeing. An earlier study by Creamer et al. (2001) on 10,641 participants in 1997 found that post-traumatic stress disorders lead to poorer physical state and caused a higher level of depression and anxiety among Australian households. Improving the physical health for general population in particular, for those with mental health issues is important in order to improve their wellbeing in Australia. People diagnosed with mental illness have relatively poor physical health and often die earlier than their counterparts in the general population. People living with mental illness in rural Australia have three times the risk of premature death than the total population (Roberts et al., 2018).

Strategies aiming to help improve physical health in the population ultimately will improve mental health. Morgan et al. (2021) showed that most strategies out of 61 programs and initiatives in Australia, which were reviewed in their study, aiming at improving mental health have primarily addressed stigma and discrimination towards people with mental illness and their families, or for culturally and linguistically diverse communities with limited evidence of effectiveness. Other studies in Australia or elsewhere focused on improving service use accessibility, treatment and social supports (Allen et al., 2014; Andrade et al., 2014; Cohen et al., 2009; Caldwell et al., 2004), but missed the

role of physical health and physical health-related risk factors/health behaviour. Intervention and preventative strategies therefore should also focus on promoting a healthy life via physical exercise, and safer environments at work and outside work to reduce risks to physical health, as our study showed that health behaviours such as exercise and shocks (i.e., risk) to health strongly affect physical health, and worsening physical health leads to significant declines in mental health.

One limitation may be that the HILDA mental health measure used in the current study is simplistic, and the gold standard is diagnosis-validated data. However, the health measures in the SF-36 were validated with Australian data. For example, Sanson-Fisher and Perkins (1998) showed that physical functioning, role-physical, general health, and bodily pain scales were found to be strongly correlated with the physical dimension, and mental health measure was strongly correlated with the mental health dimension in psychometric testing, using the International Quality of Life Assessment (IQOLA) methods (Ware & Gandek, 1998b). In addition, Butterworth and Crosier (2004) demonstrated and provided evidence for the validity of the SF-36 data in the HILDA Survey, and supported its use as a general outcome measure of physical and mental health status in Australia. To further validate the HILDA mental health measure used in the current analysis, we checked the correlation between Kessler psychological distress (K10)<sup>4</sup> (also collected in HILDA) and the mental health variable for the years with available K10 psychological distress data, the correlation was found to be very high (0.82, statistically significant,  $p < 0.0001$ ) suggesting that using either the K10 score or mental health variable captures well individual's mental health status. Unfortunately, the K10 data in the HILDA were collected in a few years with time gaps (in waves 2007, 2009, 2011, 2013, 2015, 2017, 2019). The K10 psychological distress data with time gaps thus were not suitable for our longitudinal data modelling.

Our study underscores the importance of redesigning health systems to truly address the interplay between physical and mental health. Usually services are designed to offer separate, specialist teams and treatment for physical and mental health. Our study shows that physical health promotion, health problems, risks and shocks are important drivers of physical health changes and ultimately mental health, as is the reverse, and these dual impacts need to be assessed, monitored and addressed as 'business as usual'.

<sup>4</sup> Kessler psychological distress scale (K10) score was constructed from 10 questions about mental health: *depressed; everything was an effort; so nervous that nothing could calm you down; so restless that you could not sit still; hopeless; nervous; restless or fidgety; so sad that nothing could cheer you up; tired out for no good reasons; worthless.*

Our study supports calls for embedding dual mental and physical health supports and treatment in all health services as a system and policy reform.

**Acknowledgments** This paper uses unit record data from the Household, Income and Labour Dynamics in Australia (HILDA) Survey 2002–2019. The HILDA Project was initiated and is funded by the Australian Government Department of Social Services (DSS) and is managed by the Melbourne Institute of Applied Economic and Social Research (Melbourne Institute). The findings and views reported in this paper, however, are those of the authors and should not be attributed to the universities, DSS or the Melbourne Institute. The authors are responsible for all findings and views expressed in this paper.

**Funding** Open Access funding enabled and organized by CAUL and its Member Institutions

## Declarations

**Conflict of interest** We declare that there is no conflict of interest in this study.

**Ethical approval** This paper uses secondary data (HILDA) and there is no requirement for ethical review.

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