Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Journal of Psychosomatic Research 136 (2020) 110176

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Suboptimal baseline mental health associated with 4-month premature all-cause mortality: Findings from 18 years of follow-up of the Canadian National Population Health Survey

Esme Fuller-Thomson[a,*], Yu Lung[a], Keri J. West[a], Corey L.M. Keyes[b], Philip Baiden[c]

[a] Factor-Inwentash Faculty of Social Work, University of Toronto, Toronto, Ontario, Canada
[b] Department of Sociology, Emory University, Atlanta, GA, United States
[c] School of Social Work, The University of Texas at Arlington, Arlington, TX, United States

ARTICLE INFO

Keywords:
Flourishing
Mental health
Positive mental health
Mortality
Risk factors

ABSTRACT

Objective: To investigate: 1) whether baseline non-flourishing mental health is associated with a higher probability of all-cause mortality over 18-year follow-up after controlling for many risk factors for premature mortality; and 2) what other factors, independent of mental health status, are associated with all-cause mortality after adjustment for known risk factors.

Methods: Data were derived from waves 1 and 9 (1994/1995; 2010/2011) of the Canadian National Population Health Survey. An analytic sample of 12,424 participants 18 years and above was selected. Baseline information on flourishing and predictors of all-cause mortality was from wave 1 and mortality data was ascertained by the Canadian Vital Statistics-Death Database in wave 9. Mean time to all-cause mortality was estimated using Kaplan-Meir procedure. Cox proportional hazards models were used to assess the association of baseline non-flourishing mental health and potential predictors with time to all-cause mortality.

Results: About one in five participants was classified as non-flourishing at baseline. At the end of the study period 2317 deaths were observed. Baseline non-flourishing mental health was associated with a 19% higher probability of all-cause mortality during 18-year follow-up (HR = 1.19; 95% CI 1.08–1.32), corresponding to a 4.7-month shorter survival time. After controlling for baseline chronic health conditions, past-year depression, sociodemographics, health behaviors, social support, pain and functioning, baseline non-flourishing mental health status was associated with a 14% higher probability of death (HR = 1.14; 95% CI 1.02–1.27).

Conclusions: Suboptimal mental health is associated with premature mortality even after accounting for many risk factors for early death. Future research should explore the physiological pathways through which non-flourishing influences mortality.

1. Introduction

Although the World Health Organization (WHO) has endorsed a positive definition of health and mental health since the adoption of its Constitution in 1946, characterizing health as “a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity” (p. 1) [1], Psychology, as a discipline – both clinically and in research – has tended since the Second World War to be oriented toward a pathology model of human functioning [2]. The emergence of positive psychology in the past three decades has elevated mental health as an empirical concept distinct from mental illness [3]. Keyes' [4] dual continua model of mental health and illness provides a useful theoretical alternative to the conventional pathological conceptual framework for mental health research. This model, which has strong empirical support, posits that mental illness and mental health constitute correlated but distinct axes - one indicates an individual's level of mental health (i.e., languishing, moderate, and flourishing); the other, their level of mental illness [4]. According to Keyes [5], an individual with a high level of subjective well-being (i.e., positive emotions and satisfaction with one's life), and optimal psychological functioning, is considered to have flourishing mental health.

A burgeoning literature has aimed to conceptualize, operationalize, and measure human flourishing and explore the etiology and sequelae of flourishing mental health. Previous research has provided convergent evidence of the favourable association between flourishing mental health and physical health [6]. Indicators of flourishing mental health

* Corresponding author at: Factor-Inwentash Faculty of Social Work, University of Toronto, 246 Bloor Street West, Toronto, Ontario M5S 1V4, Canada.
E-mail address: esme.fuller.thomson@utoronto.ca (E. Fuller-Thomson).

https://doi.org/10.1016/j.jpsychores.2020.110176
Received 28 October 2019; Received in revised form 14 June 2020; Accepted 19 June 2020

have shown advantageous effects on a range of health outcomes, including diabetes [7], cardiovascular disease [8], cancer [9], stroke [10], chronic lung disease [11], inflammatory conditions [12], and risk factors for coronary heart disease, such as high blood pressure [13].

A growing body of literature has also link flourishing mental health to longevity [14]. Cohen and colleagues [15] found a significant association between higher purpose in life and reduced all-cause mortality in a meta-analysis of prospective observational studies. Sub-group analyses by study country of origin, purpose in life instrument used, and inclusion/exclusion of participants with baseline cardiovascular disease all produced similar results. Another meta-analysis of prospective observational studies by Chida and Steptoe [16] found that positive psychological well-being (i.e., positive affect or positive trait-like disposition) was associated with reduced mortality in initially healthy populations as well as among those with baseline chronic disease. Pressman and Cohen's[17] findings likewise suggest that among those who are healthy at baseline, longevity is greater among individuals with positive affect; however, they found equivocal evidence of positive affect predicting survival among those with serious illness. The authors hypothesized that positive affect may confer increased risk of death among those with serious or end-stage disease, in part, because it may signal maladaptive coping, over-optimism, or denial, leading to inadequate disease management [17].

Others have found differential effects of various indicators of subjective well-being on mortality in different populations. For instance, Wiest et al. [18] found that positive affect, as a measure of emotional well-being, was predictive of mortality over a 13-year period in older adults (aged 65+) after controlling for sociodemographic characteristics, self-rated health, and physical activity; whereas the relationship between life satisfaction and mortality was attenuated by self-rated health and physical activity. Interestingly, for middle-aged participants, neither life satisfaction nor positive affect predicted mortality when controlling for covariates [18]. Martín-María and colleagues' [19] meta-analytic findings indicate that although subjective well-being conferred protection against mortality in both men and women, the effect was slightly stronger for men.

While the mechanisms through which flourishing mental health exerts positive effects on health and mortality are not fully understood, there are several hypothesized pathways through which flourishing mental health could affect mortality. For instance, Pressman and Cohen [17] propose two models linking positive affect and health. In the *direct (main) effects model*, positive affect is posited to have salutogenic effects on health by directly influencing health practices and social behaviors, decreasing autonomic nervous system activation, and regulating hypothalamic–pituitary–adrenal axis activity, endogenous opioid activity, and immune function. The *stress-buffering model*, in contrast, suggests that positive affect may mitigate the potentially pathogenic effects of stressful life events by generating psychological resources that enhance resiliency, coping, and creative problem solving [17].

An important limitation of the existing evidence base is the overwhelming focus on emotional well-being. In a 10-year longitudinal study of mid-life adults, Keyes and Simoes [14] offered a more holistic model of positive mental health that included psychological well-being. In models adjusting for known causes of mortality, the absence of positive mental health, assessed categorically, was associated with 62% higher odds of mortality. Interestingly, in analyses in which the components of positive mental health were disaggregated, emotional well-being was not a predictor of mortality after taking psychological well-being into account [14].

The purpose of the present study is to examine the relationship between non-flourishing mental health and all-cause mortality. Using a nationally representative Canadian data, we aim to address the following research questions:

1. Is non-flourishing (i.e., suboptimal) mental health at baseline associated with a higher probability of all-cause mortality during the 18-year follow-up period, after controlling for many known risk factors for mortality?
2. What other factors, independent of baseline mental health status, are significantly associated with all-cause mortality during the 18-year follow-up period in the fully adjusted Cox proportional hazard regression analysis?

Building upon previous work by Keyes and Simoes [14], this study will add to the existing evidence base and provide a more holistic understanding of the effect of suboptimal mental health on mortality by using a nationally representative Canadian data with a large sample size and a long follow-up period. We controlled for a range of known risk factors for mortality and correlates of mental health that were in the baseline dataset, including: chronic diseases, health behaviors (i.e., drinking, smoking, physical activity, obesity) [20,21], social support [22,23], marital status [24,25], pain [26], functional limitations [27,28], socioeconomic status (i.e., educational attainment, income) [29,30], and demographic characteristics (i.e., age, ethnicity, gender) [29,31–33].

We hypothesized that: 1) non-flourishing mental health at baseline will be associated with higher all-cause mortality during the 18-year follow-up period after adjusting for sociodemographic characteristics and known risk factors for mortality; 2) adverse health behaviors, chronic illnesses, and lack of social support at baseline will be associated with a higher probability of all-cause mortality after controlling for baseline mental health status and other characteristics.

2. Methods

2.1. Data

This study was based on data from the National Population Health Survey (NPHS), a longitudinal panel study conducted by Statistics Canada every two years starting in 1994/1995. The objective of the nationally representative survey was to investigate the dynamic changes in health, illness, and health care utilization with the purpose of understanding the determinants of health and to improve the health status of the population in Canada [34]. The NPHS used a stratified two-stage (clusters, dwellings) sampling design, comprised of 17,276 participants over 12 years of age living in the ten provinces in the first wave (1994/1995). For a detailed description of the NPHS, we refer the reader to the NPHS Data Documentation Guide [34]. The present study used data from wave 1 (1994/1995) through wave 9 (2010/2011). The baseline response rate was 83.6% which was based on the 20,095 individuals who were initially selected to form the longitudinal panel. The response rates for the subsequent waves ranged between 69.7% and 92.8%, which were based on the final sample of 17,276 individuals [34].

2.2. Sample

A flowchart of inclusion and exclusion of the participants is shown in Fig. 1. We first excluded respondents who were under the age of 18 and had missing data on the flourishing scale, which gave us a sample of 13,009 respondents. We then excluded respondents who had missing data on key variables at baseline except for income and major depressive episode (MDE). This resulted in a sample of 12,459 respondents. We further excluded respondents whose year of death was unknown and obtained a final sample of 12,424 participants. Thus, the final analytic sample (n = 12,424) included 95.5% of the original respondents who were 18 years and over at baseline (n = 13,009).

Among the 12,424 participants, 2317 were deceased at the end of the observation period with year and month of death information provided. Two different cases of right-censoring occurred: In wave 9, 5329 individuals were alive and interviewed. For these individuals, time to censor was calculated as the number of months between each

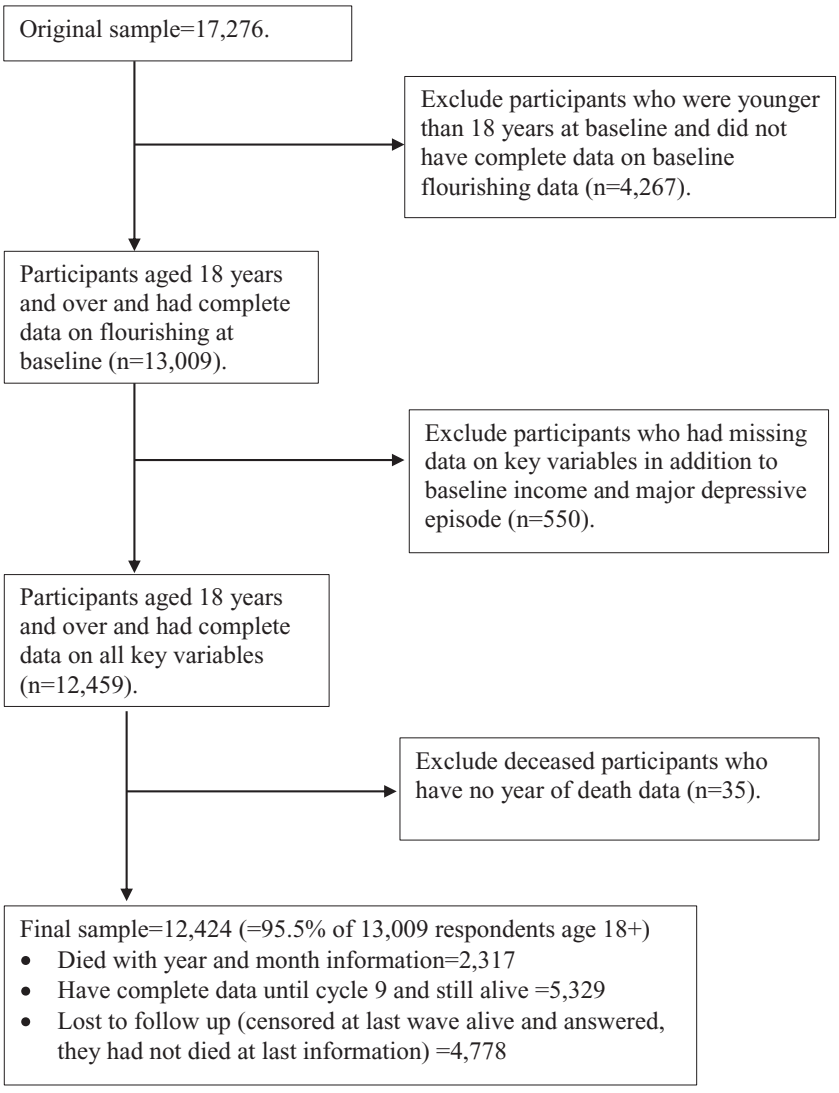

Fig. 1. Flowchart for the inclusion and exclusion of NPHS cohort to obtain final analytical sample.

person's wave 1 interview and wave 9 interview. For those who dropped out of the study before the wave 9 interview (n = 4778 individuals), time to censor was determined as the number of months between each respondent's wave 1 interview and the last interview in which they participated.

2.3. Measures

Flourishing. Under the advisement of Keyes, the flourishing scale was constructed of two parts: (1) happiness and satisfaction in life; and (2) positive psychological functioning.

The first part of the flourishing scale was measured by an affirmative response to either of the questions. The first question was "would you describe yourself as being usually happy and interested in life" with five response options (happy and interested in life, somewhat happy, somewhat unhappy, unhappy with little interest in life, and so unhappy that life is not worthwhile). Respondents who indicated "happy and interested in life" were recoded as 1; otherwise they were recoded as 0. The second question "Is doing the things you do every day a source of great pleasure and satisfaction or a source of pain and boredom?" was originally measured as an ordinal variable on a scale of one to seven, where one indicated "a great deal of pleasure and satisfaction" and seven indicated "a source of pain and boredom". We recoded scores of one or two to "1 = pleasure and satisfaction" and scores of three to seven to "0 = not a source of pleasure and satisfaction". The second part of the flourishing scale was measured by affirmative responses to at least five out of eight of the following questions. The first question "Until now your life has had no clear goals or purpose, or has it had very clear goals and purpose?" had a seven-point response scale anchored at 1 "no clear goals or no purpose" and 7 "very clear goals and purpose". The second question "When something happens, you generally find that you overestimate or underestimate its importance or you see things in the right proportion?" had a seven-point scale with 1 anchored at "overestimate or underestimate its importance" and 7 "see things in the right proportion". People who scored six or seven were recoded as "1 = positive psychological functioning", while people who scored one to five were recoded as "0 = negative psychological functioning". The remaining six questions: "You feel that you have a number of good qualities", "You feel that you're a person of worth at least equal to others", "You are able to do things as well as most other people", "You take a positive attitude toward yourself", "On the whole you are satisfied with yourself" and "You can do just about anything you really set your mind to", were originally measured on a five-point Likert scale ranging from "strongly agree" to "strongly disagree". For each question, we recoded scores of 1 or 2 as "1 = positive psychological functioning" and scores of 3 to 5 as "0 = negative psychological functioning".

The Cronbach's alpha reliability coefficient of the flourishing scale, based on the 10 items, was 0.7, indicating acceptable internal

consistency. Confirmatory factor analysis was conducted to examine the factor structure of the flourishing scale using Mplus 8. A single-factor model showed acceptable fit with the data (RMSEA = 0.054; TLI = 0.914; CFI = 0.933). The chi-square test was statistically significant (χ^2 = 1353, p < .001), however, the chi-square test is sensitive to sample size and tends to be significant for models with more than 200 cases. Regarding discriminant validity, we found at baseline that respondents without flourishing mental health had a higher percentage of MDE (16.8%) compared to those who had flourishing mental health (3.4%). A dichotomous flourishing indicator was created to identify people who were happy and satisfied in life and had good psychological functioning at baseline (1994–1995), in contrast to those with suboptimal mental health.

All-cause mortality. Mortality was ascertained by the Canadian Vital Statistics-Death Database in the NPHS. We subtracted the month and year of death from the month of interview in 1994 or 1995 to generate the time in months to mortality. **Predictors of all-cause mortality**. Drawing on previous empirical studies, the following variables were used as potential risk factors that may affect the probability of mortality in later life. All the predictors included in the study were measured at baseline.

Sociodemographic characteristics. Gender (male vs. female) and ethnicity (white vs. non-white) were coded as dichotomous variables. Age was coded into six groups: 18–29 years, 30–39 years, 40–49 years, 50–59 years, 60–69 years, 70 years or older. Marital status was measured as a categorical variable, into single, widowed/divorced/separated, and married/common-law/partner. Education was measured as an ordinal variable with four levels: less than secondary, secondary, some post-secondary, and post-secondary graduation. Income was measured based on the ranking of household income, which was categorized into five quintiles: highest quintile, fourth quintile, middle quintile, second quintile, and lowest quintile.

Pain and functioning. Pain was measured as a dichotomous variable (yes/no) based on response to the question, “Are you usually free of pain or discomfort?” Functional limitations (yes/no) was measured by an affirmative response to a derived variable “Restriction of activity excluding long-term disabilities or handicaps” provided by the NPHS survey.

Health behaviors. Obesity (normal, underweight, overweight, or obese), smoking status (never smoker, former smoker, or current smoker), and level of physical activity (regular, occasional, or infrequent) were included. Heavy drinking was measure as a dichotomous variable with “1 = women who have eight or more drinks per week and men who have 15 or more drinks weekly” versus “0 = those who drink less (not heavy drinker)”.

Social support. Social support was measured as a dichotomous variable based the question, “Do you have someone you can confide in or talk to about your private feelings or concerns?”

Chronic illness. Eight types of physical chronic illness were included in this study: asthma, high blood pressure, chronic bronchitis, diabetes, heart disease, cancer, stroke, and other long-term condition. Each chronic illness was measured by an affirmative answer to the question, “Do you have any of the following long-term conditions that have been diagnosed by a health professional?”. We also included a dichotomous measure of previous 12-month MDE at baseline by assessing the Composite International Diagnostic Interview-Short Form (CIDI-SF) in the NPHS. MDE was defined as a 90% predictive probability of the CIDI-SF algorithm in NPHS. The 90% cut-point has been validated against the DSM-III-R diagnosis for MDE [35].

2.4. Statistical analysis

Survival analyses were conducted to examine the relationship between non-flourishing (i.e., suboptimal) mental health and other characteristics assessed during the 1994/95 baseline wave of data collection and all-cause mortality over an 18-year period. First, descriptive statistics were provided to determine the distribution of the baseline sociodemographic and health characteristics of the 12,424 participants. We further compared the distribution of baseline characteristics among the full sample, the subsample of people who died, and the subsample of people who dropped out of the study before wave 9 (i.e., right-censored respondents). Second, the mean time to all-cause mortality was calculated using the Kaplan-Meier procedure and comparisons were made for the baseline flourishing status and potential predictors using the Log-Rank test. Lastly, a series of Cox proportional hazard models using the block selection method were performed to assess the association of baseline non-flourishing mental health and potential predictors with the time (month) to all-cause mortality. The Cox proportional hazard assumption was assessed by visually inspecting the log-log survival curves. Generally, parallel lines of survival curves were observed within each predictor, suggesting that the relationship between hazard functions for each category of a predictor remained constant over the period of observation. The proportional hazard assumption was met.

Model 1 included only the baseline flourishing status. Model 2 and Model 3 included baseline demographic and socioeconomic variables respectively. We then added in different clusters of baseline variables: pain and functioning (Model 4), health behaviors (Model 5), social support (Model 6), and chronic illness (Model 7). Model 8 is the full model which includes all aforementioned variables. Cox proportional Hazard Ratios (HRs) and 95% confidence intervals (CIs) are presented. A p-value less than 0.05, two sided, was used to determine significance for all tests. Analyses were performed using SPSS version 25 (SPSS Inc., Chicago, IL, USA).

3. Results

3.1. Descriptive statistics

Table 1 summarizes the sociodemographic characteristics of the study sample at baseline (1994/1995), indicating that slightly over half of the participants were female (55.2%), under 50 years old (61.3%), and married or living with a partner (57%). The majority were white (94%) and had some post-secondary education (69.7%). One out of five participants (19.1%) were classified as ‘non-flourishing’ at the beginning of the study. At the end of the observation period, roughly 19% of participants were deceased with date information.

The baseline characteristics were quite similar between the total sample included in the study, the individuals who died, and those who dropped out of the study (e.g., those who were-right censored before wave 9) (Table 1). Participants who died during follow up were older, and had a lower education level, lower income, and more chronic illnesses.

Is non-flourishing mental health status at baseline associated with a higher probability of all-cause mortality during the 18-year follow-up period, after controlling for many of the known risk factors?

The all-cause mortality rate for people who were not flourishing in 1994/1995 was significantly higher (20% vs 18%) and time to death was shorter (177 months vs 181.7 months) than for those who were flourishing. Thus, the mean survival time of respondents who did not have flourishing mental health at baseline was 4.7 months less than that of respondents who did have flourishing mental health at baseline (Table 1).

In the Cox regression model with only flourishing status, respondents who had suboptimal mental health at baseline showed a 19% higher probability of all-cause mortality (HR = 1.19; 95% CI, 1.08–1.32; p < .001) compared to people who were flourishing. After controlling for all predictors in Model 8, respondents who had suboptimal mental health at baseline were still 14% more likely to die in the follow-up period than those who had flourishing mental health (HR = 1.14; 95% CI, 1.02–1.27; p = .02) (Table 2). Fig. 2 further

Table 1
Baseline characteristics, mean survival time (month), and mortality rate of the NPHS participants aged 18 years and over (n = 12,424).

Variables	Baseline characteristics % (95% CI)			Mean survival time (month, SE)[b]	Mortality rate (%)
	Analytic sample n = 12,424	Died n = 2317	Dropped out of study before wave 9 (right-censored) n = 4778		
Flourishing					
No	19.1 (18.4–19.8)	20.4 (18.8–22.1)	22.0 (20.9–23.2)	177.0 (1.2)	19.9
Yes	80.9 (80.2–81.6)	79.6 (77.9–81.2)	78.0 (76.8–79.1)	181.7 (0.5)	18.3
Sociodemographics					
Gender					
Male	44.8 (43.9–45.6)	47.2 (45.2–49.3)	46.7 (45.3–48.1)	178.8 (0.7)	19.7
Female	55.2 (54.4–56.1)	52.8 (50.7–54.8)	53.3 (51.9–54.7)	182.4 (0.6)	17.8
Ethnicity					
Non-white	6.0 (5.6–6.4)	2.5 (2.0–3.2)	9.7 (8.9–10.6)	193.3 (1.3)	7.9
White	94.0 (93.6–94.4)	97.5 (96.8–98)	90.3 (89.4–91.1)	180.1 (0.5)	19.3
Age group					
18–29	20.6 (19.9–21.3)	1.4 (1.0–2.0)	19.6 (18.6–20.7)	201.3 (0.3)	1.3
30–39	23.0 (22.2–23.7)	3.0 (2.3–3.7)	27.1 (25.9–28.3)	200.2 (0.4)	2.4
40–49	17.7 (17.1–18.4)	6.0 (5.1–7.1)	22.2 (21.1–23.3)	195.8 (0.7)	6.4
50–59	13.2 (12.6–13.8)	11.4 (10.1–12.7)	16.0 (15.0–17.0)	185.6 (1.1)	16.1
60–69	11.8 (11.3–12.4)	23.6 (21.9–25.4)	10.8 (10.0–11.6)	163.8 (1.5)	37.2
70 and over	13.7 (13.1–14.3)	54.6 (52.6–56.6)	4.3 (3.8–4.9)	118.8 (1.6)	74.2
Marital status					
Married/Common-law/Partner	57.0 (56.1–57.9)	47.2 (45.2–49.3)	53.4 (52.0–54.8)	185.4 (0.5)	15.4
Single	21.8 (21.1–22.5)	10.4 (9.2–11.6)	29.8 (28.5–31.1)	191.7 (0.7)	8.9
Widowed/Divorced/Separated	21.2 (20.5–21.9)	42.4 (40.4–44.4)	16.8 (15.8–17.9)	158.4 (1.3)	37.3
Education					
Less than secondary	19.0 (18.3–19.7)	42.2 (40.2–44.2)	17.1 (16.1–18.2)	152.9 (1.4)	41.4
Secondary	11.3 (10.8–11.9)	11.5 (10.2–12.8)	12.3 (11.4–13.2)	179.7 (1.4)	18.9
Some post-secondary	26.9 (26.1–27.6)	21.8 (20.1–23.5)	29.3 (28.1–30.6)	185.3 (0.8)	15.1
Post-secondary and higher	42.8 (42.0–43.7)	24.6 (22.8–26.3)	41.3 (39.9–42.7)	190.7 (0.5)	10.7
Income					
Highest	18.1 (17.4–18.8)	8.8 (7.7–10.0)	15.8 (14.7–16.8)	193.4 (0.7)	9.0
4th quintile	18.3 (17.6–19.0)	13.0 (11.7–14.4)	17.2 (16.2–18.3)	187.9 (0.9)	13.3
Middle quintile	17.9 (17.3–18.6)	16.7 (15.2–18.2)	17.4 (16.4–18.5)	182.9 (1.0)	17.3
Second quintiles	19.6 (18.9–20.3)	28.9 (27.1–30.8)	19.1 (18.0–20.2)	169.5 (1.2)	27.5
Lowest	20.3 (19.6–21.0)	27.0 (25.2–28.9)	24.0 (22.8–25.3)	171.4 (1.2)	24.8
Missing data	5.7 (5.3–6.2)	5.6 (4.7–6.6)	6.5 (5.8–7.2)	180.2 (1.9)	18.2
Pain and Functioning					
Pain					
Have pain	18.2 (17.6–18.9)	29.3 (27.4–31.1)	15.9 (14.9–17)	166.6 (1.3)	29.9
Free of pain	81.8 (81.1–82.4)	70.7 (68.9–72.6)	84.1 (83–85.1)	184.0 (0.5)	16.1
Functional limitation					
No	80.5 (79.8–81.2)	61.6 (59.6–63.6)	84.6 (83.6–85.6)	186.8 (0.4)	14.3
Yes	19.5 (18.8–20.2)	38.4 (36.4–40.4)	15.4 (14.4–16.4)	156.9 (1.4)	36.7
Health behaviors					
Obesity					
Overweight	35.7 (34.8–36.5)	36.6 (34.6–38.5)	32.8 (31.5–34.2)	181.7 (0.7)	19.1
Underweight	2.6 (2.3–2.9)	3.9 (3.2–4.8)	2.7 (2.3–3.2)	158.4 (3.9)	28.3
Obese	14.0 (13.4–14.7)	16.1 (14.6–17.6)	12.9 (11.9–13.8)	179.1 (1.2)	21.4
Normal weight	47.7 (46.8–48.6)	43.4 (41.4–45.4)	51.6 (50.2–53.0)	181.9 (0.7)	17.0
Smoking					
Non-smoker	36.1 (35.2–36.9)	31.7 (29.8–33.6)	34.3 (32.9–35.6)	183.9 (0.7)	16.4
Former smoker	31.8 (31.0–32.6)	39.7 (37.7–41.7)	27.2 (26.0–28.5)	175.3 (0.9)	23.3
Current smoker	32.1 (31.3–33.0	28.6 (26.8–30.5)	38.5 (37.1–39.9)	182.8 (0.8)	16.6
Drinking					
Non-heavy drinker	69.7 (68.9–70.5)	53.4 (51.4–55.4)	71.3 (70.0–72.6)	186.4 (0.5)	14.3
Heavy drinker	30.3 (29.5–31.1)	46.6 (44.6–48.6)	28.7 (27.4–30)	168.0 (1.0)	28.7
Physical activity level					
Regular	53.5 (52.7–54.4)	44.0 (42.0–46.0)	55.9 (54.5–57.3)	185.8 (0.5)	15.3
Occasional	21.0 (20.3–21.8)	15.3 (13.9–16.8)	20.9 (19.8–22.1)	187.4 (0.9)	13.6
Infrequent	25.4 (24.7–26.2)	40.7 (38.7–42.7)	23.2 (22.0–24.4)	164.9 (1.1)	29.9
Social support					
No	11.5 (10.9–12.0)	16.4 (14.9–17.9)	11.7 (10.8–12.6)	170.6 (1.6)	26.7
Yes	88.5 (88.0–89.1)	83.6 (82.1–85.1)	88.3 (87.4–89.2)	182.1 (0.5)	17.6
Chronic illness					
Asthma[a]					
No	94.3 (93.9–94.7)	94.3 (93.3–95.2)	94.0 (93.3–94.6)	180.9 (0.5)	18.6
Yes	5.7 (5.3–6.1)	5.7 (4.8–6.7)	6.0 (5.4–6.7)	178.8 (2.1)	18.7
High blood pressure					
No	88.2 (87.6–88.7)	72.2 (70.3–74)	92.9 (92.1–93.6)	184.8 (0.4)	15.3
Yes	11.8 (11.3–12.4)	27.8 (26–29.7)	7.1 (6.4–7.9)	153.1 (1.7)	43.8
Chronic bronchitis					

(*continued on next page*)

Table 1 (*continued*)

Variables	Baseline characteristics % (95% CI)			Mean survival time (month, SE)[b]	Mortality rate (%)
	Analytic sample n = 12,424	Died *n* = 2317	Dropped out of study before wave 9 (right-censored) n = 4778		
No	96.3 (95.9–96.6)	92.1 (90.9–93.1)	96.9 (96.4–97.4)	182.0 (0.5)	17.8
Yes	3.7 (3.4–4.1)	7.9 (6.9–9.1)	3.1 (2.6–3.6)	151.1 (3.2)	39.8
Diabetes					
No	96.3 (96–96.6)	88.0 (86.7–89.3)	98.2 (97.8–98.6)	182.8 (0.4)	17.0
Yes	3.7 (3.4–4.0)	12.0 (10.7–13.3)	1.8 (1.4–2.2)	130.7 (3.2)	60.5
Heart disease					
No	94.5 (94.1–94.9)	81.8 (80.2–83.3)	97.5 (97.0–97.9)	184.1 (0.4)	16.1
Yes	5.5 (5.1–5.9)	18.2 (16.7–19.8)	2.5 (2.1–3)	126.8 (2.7)	62.1
Cancer					
No	98.0 (97.8–98.3)	94.3 (93.4–95.2)	98.8 (98.5–99.1)	181.9 (0.4)	18.0
Yes	2.0 (1.7–2.2)	5.7 (4.8–6.6)	1.2 (0.9–1.5)	127.9 (5)	53.3
Stroke					
No	98.9 (98.7–99.1)	96.2 (95.4–96.9)	99.5 (99.3–99.7)	181.6 (0.5)	18.1
Yes	1.1 (0.9–1.3)	3.8 (3.1–4.6)	0.5 (0.3–0.7)	118.0 (6)	63.8
Major depressive episode					
No	94.0 (93.6–94.4)	95.8 (94.9–96.5)	92.6 (91.9–93.3)	180.5 (0.5)	19.0
Yes	6.0 (5.6–6.4)	4.2 (3.5–5.1)	7.4 (6.7–8.1)	186.7 (1.7)	13.2
Other long-term condition					
No	92.6 (92.1–93.0)	91.2 (90.0–92.3)	93.4 (92.7–94.1)	181.1 (0.5)	18.4
Yes	7.4 (7.0–7.9)	8.8 (7.7–10)	6.6 (5.9–7.3)	178.0 (1.7)	22.1

[a] All the predictors were statistically significantly associated with all-cause mortality ($p < .001$) using Kaplan-Meier bivariate analysis, except for asthma ($p = .6$).

[b] The calculation of mean survival time includes time to death and time to censor.

shows that suboptimal baseline mental health status was consistently associated with higher probability of all-cause mortality across the eight cluster models (adjusted odds ratios range from 1.14 to 1.45), suggesting support for our first hypothesis.

What other factors, independent of flourishing status, are significantly associated with all-cause mortality during the 18-year follow-up period in the fully adjusted Cox proportional hazard regression analysis?

All of the included predictors were statistically significantly associated with all-cause mortality ($p < .001$) using Kaplan-Meier bivariate analyses, except for asthma ($p = .61$). Individuals who were female, non-white, younger, single, and those with higher educational attainment and income, were found to have a longer mean survival time. Individuals who were free from pain, had no functional limitations, were normal weight, non-smokers and not heavy drinkers, and those who had a confidant at baseline, had a longer mean time to all-cause mortality. Our results showed that individuals with occasional physical activity had a longer mean time to all-cause mortality than individuals with regular or infrequent physical activity (Table 1).

In order to address whether these factors were *independently* associated with all-cause mortality after flourishing status and the other characteristics were taken into account, we conducted Cox proportional hazard analysis (Table 2). After controlling for sociodemographic variables, our second hypothesis was partially supported. The final model showed that respondents who had functional limitations, were underweight, smokers, heavy drinkers, and had infrequent physical activity, had a higher probability of all-cause mortality. Respondents who, at baseline, had high blood pressure, chronic bronchitis, diabetes, heart disease or cancer, also had a higher probability of all-cause mortality.

However, several of the factors we had hypothesized would be associated with all-cause mortality did not come through in the multivariable analyses (e.g., MDE and having a confidant), although they had in the bivariate analyses. Perplexingly, one factor was associated with all-cause mortality in the opposite direction than we had anticipated: the findings indicate that respondents who were overweight or obese at baseline had a *lower probability* of all-cause mortality when compared to people with normal weight (Overweight: HR = 0.82; 95% CI, 0.75–0.90; $p < .001$; Obese: HR = 0.85; 95% CI, 0.75–0.96; $p = .01$).

4. Discussion

The objective of the present study was to examine the relationship between baseline suboptimal mental health and all-cause mortality during the 18-year follow-up period, after controlling for many known risk factors for mortality. We found that the nearly one in five participants (19.1%) in the present study was classified as having non-flourishing mental health at baseline. This proportion is comparable to previous Canadian nationally representative data. For instance, in the 2012 Canadian Community Health Survey-Mental Health (CCHS-MH), 23.1% of respondents were found to be not flourishing [36]. A higher prevalence of flourishing mental health has been reported in Canadian studies than in those conducted in the U.S. [3], the Netherlands [37], Denmark [38], South Africa [39], France [38], and Korea [40], where the prevalence of flourishing ranges from 8% to 40.6%. In the present study, those who did not have flourishing mental health at baseline died, on average, 4.7 months prematurely compared to those who had flourishing mental health at baseline. Even after full adjustment for sociodemographic characteristics, pain, health behaviors, social support, and chronic illness, those who had suboptimal mental health at baseline were 14% more likely to die during the 18-year follow-up period.

It is conceivable that physiological processes may mediate the observed relationship between baseline suboptimal mental health and premature death. Previous research has found reduced neuroendocrine, inflammatory, and cardiovascular activity among those with positive affect [41]. Positive psychological well-being is associated with lower levels of cortisol and inflammatory factors (e.g., pro-inflammatory cytokines, fibrinogen), which may be implicated in the pathogenesis of a range of chronic diseases [16]. Behavioral cascades may also play a role in this relationship. Those with high levels of mental well-being and positive affect are more likely to consume nutritious foods, adhere to treatment regimens, maintain strong social ties, and have better sleep quality, which may contribute to longevity [17,42].

Our finding that those who were not flourishing at baseline died

Table 2
Cox proportional hazards regression predicting time to mortality.

	Model: Full model	
	Hazard ratio (95% CI)	p-value
No Flourishing (ref. Flourishing)	1.14 (1.02–1.27)	0.02
Sociodemographics		
Female	0.57 (0.52–0.63)	< 0.001
Ethnicity (ref. non-white)	1.07 (0.82–1.39)	0.61
Age group (ref. 18-29yrs)		
30–39	1.99 (1.31–3.03)	< 0.001
40–49	5.09 (3.45–7.50)	< 0.001
50–59	12.44 (8.56–18.09)	< 0.001
60–69	28.67 (19.90–41.30)	< 0.001
70 and over	75.63 (52.53–108.9)	< 0.001
Marital status (ref. married)		
Single	1.30 (1.12–1.50)	< 0.001
Widowed/Divorced/Separated	1.19 (1.08–1.31)	< 0.001
Education (ref. post-secondary)		
Less than college/university	1.26 (1.12–1.42)	< 0.001
Secondary school	1.19 (1.02–1.38)	0.02
Some post-secondary	1.12 (0.99–1.27)	0.06
Income (ref. highest)		
4th quintile	1.25 (1.05–1.50)	0.01
Middle quintile	1.32 (1.11–1.57)	< 0.001
Second quintiles	1.37 (1.16–1.63)	< 0.001
Lowest	1.47 (1.24–1.76)	< 0.001
Missing data	1.22 (0.97–1.53)	0.08
Pain and Functioning		
Free of pain (ref. has pain)	1.08 (0.97–1.20)	0.15
Functional limitation (ref. no)	1.39 (1.25–1.54)	< 0.001
Health behaviors		
Obesity (ref. normal weight)		
Overweight	0.82 (0.75–0.90)	< 0.001
Underweight	1.62 (1.30–2.03)	< 0.001
Obese	0.85 (0.75–0.96)	0.01
Smoking (ref. non-smoker)		
Former smoker	1.21 (1.09–1.35)	< 0.001
Current smoker	1.84 (1.64–2.06)	< 0.001
Heavy drinker	1.21 (1.11–1.32)	< 0.001
Physical activity level (ref. regular)		
Occasional	1.07 (0.95–1.21)	0.27
Infrequent	1.40 (1.27–1.53)	< 0.001
Social Support (ref. no)	1.02 (0.91–1.14)	0.72
Chronic Illness (ref. no)		
Asthma	1.03 (0.85–1.24)	0.75
High blood pressure	1.15 (1.05–1.27)	< 0.001
Chronic bronchitis	1.28 (1.09–1.50)	< 0.001
Diabetes	1.75 (1.53–2.00)	< 0.001
Heart disease	1.39 (1.24–1.55)	< 0.001
Cancer	1.67 (1.39–2.00)	< 0.001
Stroke	1.21 (0.97–1.51)	0.09
Major depressive episode	0.81 (0.66–1.00)	0.05
Other long-term condition	1.03 (0.89–1.19)	0.69

significantly earlier than those who were flourishing at baseline is in keeping with Keyes and Simoes' [14] earlier study. However, the magnitude of the effect of baseline suboptimal mental health was much lower in the current study (14%) in comparison to Keyes and Simoes' [19] study (62%). The discrepancy in our findings may be attributable to methodological differences in our analyses and characteristics of our samples. We had a longer follow-up period (18 years versus 10 years) and larger age range of respondents in the present study. We also adjusted for a greater number of baseline chronic health conditions, and controlled for additional relevant factors, including income and baseline chronic pain. Further, the national context (Canadian versus American data) of the studies is likely to have impacted the results. Canada's universal, publicly funded healthcare system may provide stronger primary and preventive care than the U.S. multi-payer, heavily private system [43], where a substantial proportion of the population is uninsured or underinsured, and among the insured, high deductibles and copayments can be prohibitive [44]. Future research exploring the association between baseline non-flourishing mental health and all-cause mortality in other nations with universal health care would be illuminating.

Our findings lend further support to the dual-continua mental health and illness theory. A subsample of respondents in the present study (3.4%) had both flourishing mental health and past-year MDE at baseline. This is highly concordant with previous research. For instance, in the 2012 CCHS-MH, 4.5% of respondents had flourishing mental health in the presence of a past-year mental illness [36].

In the fully adjusted model, those who were overweight had an 18% lower likelihood of death during the follow-up period than those who were normal weight. This finding is convergent with meta-analytic research showing a significantly lower association between overweight and all-cause mortality relative to normal weight [45]. It has been speculated that the ostensive protection against mortality conferred by overweight may be due to increased secretion of adiponectin - a protein hormone involved in the modulation of glucose and lipids - which has anti-inflammatory, anti-oxidant, and atherogenic-inhibiting effects [46]. Alternatively, those who are overweight may have greater metabolic reserves to meet the demands of chronic disease [47].

Surprisingly, in contrast to previous studies [48], obesity was associated with *later* mortality compared to normal weight in the present multivariable analyses. Our puzzling findings with respect to obesity may be due to the long latency period between obesity onset and the pathogenesis of chronic disease [49]. In 1994, the prevalence of obesity among Canadian adults (excluding those residing in the Territories) was 13.1%, comparable to the 13.4% at baseline in our sample. In 2004, 23.1% of Canadians were obese [50]. It is plausible that the follow-up period in the present study was not sufficiently long to capture the lagged effects of obesity on the pathogenesis of chronic disease and mortality.

Further, the NPHS derives body mass index (BMI) from self-reported height and weight. BMI based on self-report tends to be underestimated, particularly among overweight and obese participants [51]. Such bias would likely render our findings more conservative. In addition, obesity classes I, II, and III were combined in the present study due to power issues. Our broad obesity category may, thus, not have been adequately differentiated to detect distinct effects on all-cause mortality. In previous research, effects on all-cause mortality have frequently been shown for those in class II and III obesity categories, but not for those in class I [52]. The majority of the subsample of obese respondents in our study were in class I. Finally, some previous research has found that weight cycling [53] or weight history (i.e., maximum BMI) [48] may be more important predictors of all-cause mortality than baseline weight. Future research should examine the relationship between flourishing mental health and all-cause mortality using measures of weight fluctuation rather than BMI at a single point in time.

Although social support is robustly inversely associated with all-cause mortality in meta-analytic research [23], and in our bivariate analyses, we did not find a significant relationship in our multivariable analyses. Our measure of social support may not have been sufficiently nuanced. Complex measures of social integration have shown stronger effects on all-cause mortality than single-item measures of social isolation in previous research [23]. Furthermore, adjustments for potential reasons for social isolation (e.g., chronic pain, chronic illness, major depressive disorders) may have minimized the independent direct effect linking social support to all-cause mortality.

Despite the fact that many previous studies have shown that people with depression generally have an increased risk of mortality [54–56], we did not find an effect of MDE on all-cause mortality after full adjustment. One possible explanation is that our study was based on a relatively long follow-up period. Previous studies using longitudinal data have indicated that the relative risk of dying among people with depression decreases with the length of the follow-up period when compared to those without depression [56–58]. For example, one Canadian study found that while there was more than a threefold

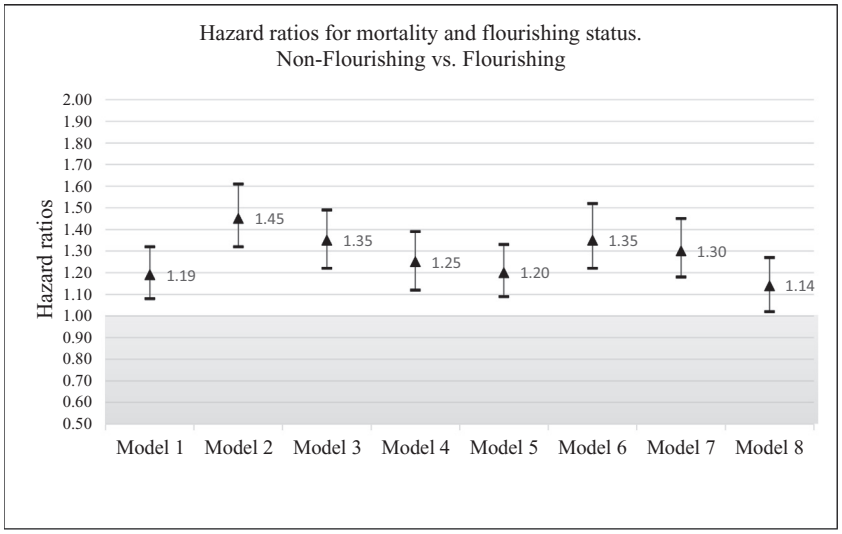

Fig. 2. Hazard ratios for mortality and flourishing status.
Model 1 - Flourishing only.
Model 2 - Demographics: Gender, ethnicity, age groups +FLOURISHING.
Model 3 - Sociodemographics: Model 2, marital status, education, income +FLOURISHING.
Model 4 – Pain and Functioning: Model 3, pain, functional limitation +FLOURISHING.
Model 5 – Health behaviors: Model 3, obesity, smoking, drinking, physical activities +FLOURISHING.
Model 6 – Social support: Model 3, social support +FLOURISHING.
Model 7 – Chronic illness: Model 3, asthma, high blood pressure, chronic bronchitis, diabetes, heart disease, cancer, stroke, major depressive episode, other long-term condition +FLOURISHING.
Model 8 - Full Model: All variables in models 1 to 7.

increased hazard of dying for people with major depression within two years of follow-up after adjusting for age and sex, the effect attenuated dramatically after more than two years of follow-up [57]. Given that MDE is an episodic and treatable illness, it is likely that people who recover from depressive symptoms may have a mortality risk that is similar to the general population. As a previous Canadian study demonstrated, most community members (77%) with MDE remitted from depression after two years [59]. Moreover, our findings are consistent with at least one previous epidemiologic study examining the relationship between MDE and all-cause mortality using the NPHS data, which similarly failed to identify an independent effect after adjustment for other risk factors [35]. The authors of that study suggested that other risk factors for all-cause mortality may be associated with MDE, which may confound the results. Alternatively, some of these variables may mediate the relationship between MDE and all-cause mortality. It is not possible with the NPHS data to disentangle confounding and mediating effects. Nevertheless, the magnitude of the association between non-flourishing and premature mortality in the current study is a relatively small effect and markedly smaller than the associations observed in studies of other mental disorders, such as schizophrenia [60] and bipolar disorder [61] – conditions which respectively represent upwards of a decade of life years lost compared to the general population. Unfortunately, the NPHS dataset did not contain information on these other forms of mental illness. Future research would benefit from the inclusion of a wide range of mental disorders.

As expected, modifiable risk factors, including smoking, heavy drinking, and infrequent physical activity, were associated with a higher probability of all-cause mortality. Likewise, cancer, heart disease, diabetes, chronic obstructive pulmonary disease, and high blood pressure were associated with a higher probability of all-cause mortality. Stroke did not come through in the fully adjusted model, likely due to power issues. Only 1.1% of the sample had stroke. However, as the bivariate analysis indicates, 63.8% of those who had stroke died during the follow-up period. Asthma was not significantly associated with the probability of all-cause mortality in our multivariable analysis. Asthma-related mortality has decreased substantially over the past several decades. Asthma deaths reportedly declined at a rate of 5.6% per year from 2.1 per 10,000 persons with asthma in 2001 to 1.4 per 10,000 in 2009, reflecting improvements in asthma management [62].

4.1. Limitations

The findings of the present study should be interpreted in light of several limitations. The flourishing scale was constructed for the purposes of this study using relevant questions from the baseline NPHS items. Although imperfect, the scale had good reliability and our findings were highly congruent with previous research by Keyes and Simoes [14]. The use of self-report of chronic health conditions may have biased the results. However, previous studies have shown high agreement between self-reported medical history and medical records or physician-reported medical history for a range of conditions, including diabetes, stroke, myocardial infarction, and hypertension [63,64], whereas cancer diagnosis, particularly among non-white and older respondents, tends to be underreported [65], and asthma and chronic obstructive pulmonary disease overreported, compared to medical records [66].

We chose to dichotomize the flourishing variable in order to enable comparison of our findings to those reported in previous studies, the majority of which likewise dichotomize the measure. As a large proportion of our sample (81%) were flourishing at baseline, secondarily examining flourishing as a continuous measure may have been informative. However, this study was conducted using embargoed data at Statistics Canada's Toronto Research Data Centre, which due to COVID-19, was closed, and thus, we were unable to undertake supplementary analyses.

Because all predictors used in the present study were measured at baseline, it is not possible to draw conclusions regarding temporal ordering or mediation. The potential biological mechanisms and social and behavioral cascades through which flourishing mental health may influence longevity and mortality should be further investigated in future prospective studies. Finally, as this study was based on data derived within the Western cultural context, findings may not be generalizable to non-Western populations and other cultural settings. The evidence base would be enriched by future research focusing on flourishing mental health and all-cause mortality in non-Western populations. In addition, our findings are not exempt from potential residual confounding due to the way several variables were measured and modeled. There may also be other unknown factors that we did not include in our analyses.

Despite these limitations, this study provides, to our knowledge, the longest follow-up period in a nationally representative study to examine the effect of baseline suboptimal mental health on all-cause mortality, taking into account a wide range of known risk factors for premature all-cause mortality. The findings suggest that suboptimal mental health is strongly associated with dying 4.7 months prematurely over an 18-year follow-up period. The negative association between baseline flourishing mental health and premature death is a robust relationship that is independent of comorbid diseases, pain and functional limitations, health behaviors, and social support at baseline. Our findings underline the importance of considering the mind and body as a true

continuum.

Funding

This work was supported by the Sandra Rotman Endowed Chair in Social Work at the University of Toronto (EFT).

Declaration of Competing Interest

The authors have no conflict of interest.

Acknowledgements

Esme Fuller-Thomson would like to gratefully acknowledge support received from the Sandra Rotman Endowed Chair in Social Work at the University of Toronto. The analysis presented in this paper was conducted at the Toronto Research Data Centre (RDC), which is part of the Canadian Research Data Centre Network (CRDCN). The services and activities provided by the Toronto RDC are made possible by the financial or in-kind support of the SSHRC, the CIHR, the CFI, Statistics Canada, and University of Toronto. The opinions expressed do not necessarily represent the views of Statistics Canada, the CRDCN's or that of its partners. The authors would like to thank the staff at the Toronto RDC for their kind assistance.

References

[1] World Health Organization. Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19–22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948. 1948;(Generic).
[2] M.E. Seligman, M. Csikszentmihalyi, Positive Psychology: An Introduction, in: M. Csikszentmihalyi (Ed.), Flow and the Foundations of Positive Psychology, Springer, 2014, pp. 279–298.
[3] C.L. Keyes, The mental health continuum: From languishing to flourishing in life, JHealth SocBehav (2002) 207–222 Journal Article.
[4] C.L.M. Keyes, Mental illness and/or mental health? Investigating axioms of the complete state model of health, J. Consult. Clin. Psychol. 73 (3) (2005) 539–548, https://doi.org/10.1037/0022-006X.73.3.539.
[5] C.L. Keyes, Promoting and protecting mental health as flourishing: a complementary strategy for improving national mental health, Am Psychol 62 (2) (2007) 95.
[6] R.T. Howell, M.L. Kern, S. Lyubomirsky, Health benefits: meta-analytically determining the impact of well-being on objective health outcomes, Health Psychol. Rev. 1 (1) (2007) 83–136.
[7] J.T. Moskowitz, E.S. Epel, M. Acree, Positive affect uniquely predicts lower risk of mortality in people with diabetes, Health Psychol. 27 (1S) (2008) S73.
[8] J.K. Boehm, L.D. Kubzansky, The heart's content: the association between positive psychological well-being and cardiovascular health, Psychol. Bull. 138 (4) (2012) 655.
[9] R.B. Williams, N. Schneiderman, Resolved: psychosocial interventions can improve clinical outcomes in organic disease (pro), Psychosom. Med. 64 (4) (2002) 552–557.
[10] G.V. Ostir, K.S. Markides, M.K. Peek, J.S. Goodwin, The association between emotional well-being and the incidence of stroke in older adults, Psychosom. Med. 63 (2) (2001) 210–215.
[11] J.A. Okely, S.O. Shaheen, A. Weiss, C.R. Gale, Wellbeing and chronic lung disease incidence: the survey of health, ageing and retirement in Europe, PLoS One 12 (7) (2017) e0181320.
[12] J.E. Stellar, N. John-Henderson, C.L. Anderson, A.M. Gordon, G.D. McNeil, D. Keltner, Positive affect and markers of inflammation: discrete positive emotions predict lower levels of inflammatory cytokines, Emotion. 15 (2) (2015) 129.
[13] J.K. Boehm, Y. Chen, D.R. Williams, C.D. Ryff, L.D. Kubzansky, Subjective well-being and cardiometabolic health: an 8–11 year study of midlife adults, J. Psychosom. Res. 85 (2016) 1–8.
[14] C.L. Keyes, E.J. Simoes, To flourish or not: positive mental health and all-cause mortality, Am J Public Health. 102 (11) (2012) 2164–2172.
[15] R. Cohen, C. Bavishi, A. Rozanski, Purpose in life and its relationship to all-cause mortality and cardiovascular events: a meta-analysis, Psychosom. Med. 78 (2) (2016) 122–133, https://doi.org/10.1097/PSY.0000000000000274.
[16] Y. Chida, A. Steptoe, Positive psychological well-being and mortality: a quantitative review of prospective observational studies, Psychosom. Med. 70 (7) (2008) 741–756, https://doi.org/10.1097/PSY.0b013e31818105ba.
[17] S.D. Pressman, S. Cohen, Does positive affect influence health? Psychol. Bull. 131 (6) (2005) 925.
[18] M. Wiest, B. Schüz, N. Webster, S. Wurm, Subjective well-being and mortality revisited: differential effects of cognitive and emotional facets of well-being on mortality, Health Psychol. 30 (6) (2011) 728.
[19] N. Martín-María, M. Miret, F.F. Caballero, et al., The Impact of Subjective Well-being on Mortality: A Meta-Analysis of Longitudinal Studies in the General Population. Psychosomatic Medicine, Published online (2017), https://doi.org/10.1097/PSY.0000000000000444.
[20] N. Grant, J. Wardle, A. Steptoe, The relationship between life satisfaction and health behavior: a cross-cultural analysis of young adults, IntJBehavMed. 16 (3) (2009) 259–268.
[21] E. Kvaavik, G.D. Batty, G. Ursin, R. Huxley, C.R. Gale, Influence of individual and combined health behaviors on total and cause-specific mortality in men and women: the United Kingdom health and lifestyle survey, Arch. Intern. Med. 170 (8) (2010) 711–718.
[22] K.L. Siedlecki, T.A. Salthouse, S. Oishi, S. Jeswani, The relationship between social support and subjective well-being across age, Soc. Indic. Res. 117 (2) (2014) 561–576.
[23] J. Holt-Lunstad, T.B. Smith, J.B. Layton, Social relationships and mortality risk: a meta-analytic review, PLoS Med. 7 (7) (2010) e1000316.
[24] C.M. Kamp Dush, P.R. Amato, Consequences of relationship status and quality for subjective well-being, J. Soc. Pers. Relat. 22 (5) (2005) 607–627.
[25] N.J. Johnson, E. Backlund, P.D. Sorlie, C.A. Loveless, Marital status and mortality: the national longitudinal mortality study, Ann. Epidemiol. 10 (4) (2000) 224–238.
[26] P.H. Finan, E.L. Garland, The role of positive affect in pain and its treatment, Clin. J. Pain 31 (2) (2015) 177–187, https://doi.org/10.1097/AJP.0000000000000092.
[27] J. Millán-Calenti, J. Tubío, S. Pita-Fernández, et al., Prevalence of functional disability in activities of daily living (ADL), instrumental activities of daily living (IADL) and associated factors, as predictors of morbidity and mortality, Arch. Gerontol. Geriat. 50 (3) (2010) 306–310. Accessed June 17, 2019 https://www.google.com/search?client=safari&rls=en&q=%5BPDF%5D+udc.es+Prevalence+of+functional+disability+in+activities+of+daily+living+(ADL),+instrumental+activities+of+daily+living+(IADL)+and+associated+factors,+as+predictors+of+morbidity+and+%E2%80%A6&ie=UTF-8&oe=UTF-8.
[28] R.E. Lucas, Long-term disability is associated with lasting changes in subjective well-being: evidence from two nationally representative longitudinal studies, J. Pers. Soc. Psychol. 92 (4) (2007) 717–730, https://doi.org/10.1037/0022-3514.92.4.717.
[29] S.D. Barger, C.J. Donoho, H.A. Wayment, The relative contributions of race/ethnicity, socioeconomic status, health, and social relationships to life satisfaction in the United States, Qual. Life Res. 18 (2) (2009) 179–189.
[30] A. Rawshani, A.-M. Svensson, B. Zethelius, B. Eliasson, A. Rosengren, S. Gudbjörnsdottir, Association between socioeconomic status and mortality, cardiovascular disease, and cancer in patients with type 2 diabetes, JAMA Intern. Med. 176 (8) (2016) 1146–1154.
[31] L.K. George, Still happy after all these years: research Frontiers on subjective well-being in later life, J Gerontol B Psychol Sci Soc Sci. 65B (3) (2010) 331–339, https://doi.org/10.1093/geronb/gbq006.
[32] M. Pinquart, S. Sörensen, Gender differences in self-concept and psychological well-being in old Age: A meta-analysis, J Gerontol B Psychol Sci Soc Sci. 56 (4) (2001) P195–P213, https://doi.org/10.1093/geronb/56.4.P195.
[33] M. Noale, N. Minicuci, C. Bardage, et al., Predictors of mortality: an international comparison of socio-demographic and health characteristics from six longitudinal studies on aging: the CLESA project, Exp. Gerontol. 40 (1) (2005) 89–99, https://doi.org/10.1016/j.exger.2004.09.003.
[34] Statistics Canada. National Population Health Survey: Household Component, Longitudinal (NPHS). 2012. Accessed June 19, 2019. http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=3225.
[35] S.B. Patten, J.V.A. Williams, D. Lavorato, J. Li Wang, S. Khaled, A.G.M. Bulloch, Mortality associated with major depression in a Canadian community cohort, Can. J. Psychiatr. 56 (11) (2011) 658–666, https://doi.org/10.1177/070674371105601104.
[36] H. Gilmour, Positive mental health and mental illness, Health Rep. 25 (9) (2014) 3.
[37] M. Schotanus-Dijkstra, M.E. Pieterse, C.H.C. Drossaert, et al., What factors are associated with flourishing? Results from a large representative National Sample, J. Happiness Stud. 17 (4) (2016) 1351–1370, https://doi.org/10.1007/s10902-015-9647-3.
[38] F.A. Huppert, T.T.C. So, Flourishing across Europe: application of a new conceptual framework for defining well-being, Soc. Indic. Res. 110 (3) (2013) 837–861, https://doi.org/10.1007/s11205-011-9966-7.
[39] C.L.M. Keyes, M. Wissing, J.P. Potgieter, M. Temane, A. Kruger, S. van Rooy, Evaluation of the mental health continuum-short form (MHC-SF) in setswana-speaking South Africans, Clin Psychol Psychother. 15 (3) (2008) 181–192, https://doi.org/10.1002/cpp.572.
[40] Y.-J. Lim, Y.-G. Ko, H.-C. Shin, Y. Cho, Prevalence and Correlates of Complete Mental Health in the South Korean Adult Population | SpringerLink, in: C. Keyes (Ed.), Mental Well-Being, Springer, 2013, pp. 91–109. Accessed February 28, 2020 https://doi.org/10.1007/978-94-007-5195-8_5.
[41] A. Steptoe, K. O'Donnell, E. Badrick, M. Kumari, M. Marmot, Neuroendocrine and inflammatory factors associated with positive affect in healthy men and women: the Whitehall II study, Am. J. Epidemiol. 167 (1) (2008) 96–102, https://doi.org/10.1093/aje/kwm252.
[42] S. Stranges, P.C. Samaraweera, F. Taggart, N.-B. Kandala, S. Stewart-Brown, Major health-related behaviours and mental well-being in the general population: the Health Survey for England, BMJ Open 4 (9) (2014), https://doi.org/10.1136/bmjopen-2014-005878.
[43] R. Moreno-Serra, P.C. Smith, Does progress towards universal health coverage improve population health? Lancet 380 (9845) (2012) 917–923, https://doi.org/

10.1016/S0140-6736(12)61039-3.
[44] D.U. Himmelstein, S. Woolhandler, M. Almberg, C. Fauke, The U.S. Health care crisis continues: a data snapshot, Int. J. Health Serv. 48 (1) (2018) 28–41, https://doi.org/10.1177/0020731417741779.
[45] K.M. Flegal, B.K. Kit, H. Orpana, B.I. Graubard, Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis, JAMA. 309 (1) (2013) 71–82, https://doi.org/10.1001/jama.2012.113905.
[46] L.B. Tankó, C. Christiansen, Can the obesity paradox be explained by the protective effects of peripheral adiposity? Arch. Intern. Med. 165 (15) (2005) 1796–1797, https://doi.org/10.1001/archinte.165.15.1796-b.
[47] W. Doehner, A. Clark, S.D. Anker, The obesity paradox: weighing the benefit, Eur. Heart J. 31 (2) (2010) 146–148, https://doi.org/10.1093/eurheartj/ehp339.
[48] H. Xu, L.A. Cupples, A. Stokes, C.-T. Liu, Association of Obesity with Mortality over 24 years of weight history: findings from the Framingham heart study, JAMA Netw. Open 1 (7) (2018) e184587, , https://doi.org/10.1001/jamanetworkopen.2018.4587.
[49] H.L. Walls, K. Backholer, J. Proietto, J.J. McNeil, Obesity and trends in life expectancy, J. Obes. 2012 (2012) 107989, https://doi.org/10.1155/2012/107989.
[50] M. Tjepkema, M. Shields, Measured Obesity: Adult Obesity in Canada, Health Statistics Division, Statistics Canada, 2005, http://publications.gc.ca/collections/collection_2016/statcan/CS82-620-1-2005-1-eng.pdf.
[51] M. Maukonen, S. Männistö, H. Tolonen, A comparison of measured versus self-reported anthropometrics for assessing obesity in adults: a literature review, Scand J Public Health. 46 (5) (2018) 565–579, https://doi.org/10.1177/1403494818761971.
[52] H.M. Orpana, J.-M. Berthelot, M.S. Kaplan, D.H. Feeny, B. McFarland, N.A. Ross, BMI and mortality: results from a national longitudinal study of Canadian adults, Obesity (Silver Spring) 18 (1) (2010) 214–218, https://doi.org/10.1038/oby.2009.191.
[53] J. Cologne, I. Takahashi, B. French, et al., Association of Weight Fluctuation With Mortality in Japanese Adults, JAMA Netw Open 2 (3) (2019), https://doi.org/10.1001/jamanetworkopen.2019.0731.
[54] M. Pinquart, P.R. Duberstein, Depression and cancer mortality: a meta-analysis, Psychol. Med. 40 (11) (2010) 1797–1810, https://doi.org/10.1017/S0033291709992285.
[55] F. Farrokhi, N. Abedi, J. Beyene, P. Kurdyak, S.V. Jassal, Association between depression and mortality in patients receiving long-term dialysis: a systematic review and meta-analysis, Am. J. Kidney Dis. 63 (4) (2014) 623–635, https://doi.org/10.1053/j.ajkd.2013.08.024.
[56] P. Cuijpers, N. Vogelzangs, J. Twisk, A. Kleiboer, J. Li, B.W. Penninx, Comprehensive meta-analysis of excess mortality in depression in the general community versus patients with specific illnesses, Am. J. Psychiatry 171 (4) (2014) 453–462, https://doi.org/10.1176/appi.ajp.2013.13030325.
[57] M. Chiu, S. Vigod, F. Rahman, A.S. Wilton, M. Lebenbaum, P. Kurdyak, Mortality risk associated with psychological distress and major depression: a population-based cohort study, J. Affect. Disord. 234 (2018) 117–123, https://doi.org/10.1016/j.jad.2018.02.075.
[58] S.E. Gilman, E. Sucha, M. Kingsbury, N.J. Horton, J.M. Murphy, I. Colman, Depression and mortality in a longitudinal study: 1952-2011, CMAJ. 189 (42) (2017) E1304–E1310, https://doi.org/10.1503/cmaj.170125.
[59] E. Fuller-Thomson, M. Battiston, T.M. Gadalla, S. Brennenstuhl, Bouncing back: remission from depression in a 12-year panel study of a representative Canadian community sample, Soc. Psychiatry Psychiatr. Epidemiol. 49 (6) (2014) 903–910, https://doi.org/10.1007/s00127-013-0814-8.
[60] T.M. Laursen, O. Plana-Ripoll, P.K. Andersen, et al., Cause-specific life years lost among persons diagnosed with schizophrenia: is it getting better or worse? Schizophr. Res. 206 (2019) 284–290, https://doi.org/10.1016/j.schres.2018.11.003.
[61] L.V. Kessing, E. Vradi, R.S. McIntyre, P.K. Andersen, Causes of decreased life expectancy over the life span in bipolar disorder, J. Affect. Disord. 180 (2015) 142–147, https://doi.org/10.1016/j.jad.2015.03.027.
[62] J. Moorman, L. Akinbami, C. Bailey, et al., National Surveillance of Asthma: United States, 2001–2010, National Center for Health Statistics, 2012, https://www.cdc.gov/nchs/data/series/sr_03/sr03_035.pdf.
[63] Y. Okura, L.H. Urban, D.W. Mahoney, S.J. Jacobsen, R.J. Rodeheffer, Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure, J. Clin. Epidemiol. 57 (10) (2004) 1096–1103.
[64] K. White, M. Avendaño, B.D. Capistrant, J. Robin Moon, S.Y. Liu, Glymour M. Maria, Self-reported and measured hypertension among older US- and foreign-born adults, J. Immigr. Minor. Health 14 (4) (2012) 721–726, https://doi.org/10.1007/s10903-011-9549-3.
[65] M.M. Desai, M.L. Bruce, R.A. Desai, B.G. Druss, Validity of self-reported cancer history: a comparison of health interview data and cancer registry records, AmJEpidemiol. 153 (3) (2001) 299–306.
[66] A.D. Mohangoo, M.W. van der Linden, F.G. Schellevis, H. Raat, Prevalence estimates of asthma or COPD from a health interview survey and from general practitioner registration: what's the difference? Eur. J. Pub. Health 16 (1) (2006) 101–105, https://doi.org/10.1093/eurpub/cki043.