Examination of the association between mental health, morbidity and mortality in late life:
Findings from longitudinal community surveys

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Abstract

Background: Physical health has been demonstrated to mediate the mental health and mortality-risk association. The current study examines an alternative hypothesis; that mental health mediates the effect of physical health on mortality risk.

Methods: Participants (N = 14, 019; Female = 91%), including eventual decedents (n = 3,752), were aged 70 years and older, and drawn from the Dynamic Analyses to Optimise Ageing (DYNOPTA) project. Participants were observed on between two to four occasions, over a 10-year period. Mediation analysis compared the converse mediation of physical and mental health on mortality risk.

Results: For men, neither physical nor mental health were associated with mortality risk. For women, poor mental health reported only a small effect on mortality risk (HR = 1.01; $p < .001$); more substantive was the risk of low physical health (HR = 1.04; $p < .001$). No mediation effects were observed.

Conclusion: Mental health effects on mortality were fully attenuated by physical health in men, and partially so in women. Neither mental nor physical health mediated the effect of each other on mortality risk for either sex. We conclude that physical health is a stronger predictor of mortality risk than mental health.

Keywords: Death and Dying; Depression; Epidemiology; Gender Differences; Longitudinal Studies

Running Title: Late-life mental health mortality risk

Introduction

Mental health has been cited as a risk factor for all-cause mortality. In clinical populations, there is considerable evidence for depression and depressive symptomology as risk factors for mortality, particularly for those individuals who present with cancer (Pinquart and Duberstein 2010), coronary heart disease (Barth, Schumacher et al. 2004), and stroke (Bartoli, Lillia et al. 2013). Depression has also been implicated as a risk for mortality in large population studies (Harris and Barraclough 1998, Saz and Dewey 2001, Cuijpers and Schoevers 2004). However, there are a number of limitations that have not been adequately addressed in the literature. It has been suggested that the mental health-mortality relationship could be attenuated by measurement and design issues (Wulsin, Vaillant et al. 1999). It has been suggested that many studies also fail to control for the confounding effects of physical health and functioning (Batterham, Christensen et al. 2012). Recently, the association between mental health and mortality has been accounted for by intra-individual variation in physical health (Burns, Butterworth et al. 2013, Burns, Mitchell et al. 2014). That is, as individuals increasingly develop poor physical health in the years preceding death, so they decline in mental health and wellbeing.

Batterham and colleagues (Batterham, Christensen et al. 2012) assessed one possible mechanism to explain the mental health-mortality risk association, proposing that physical health mediates the relationship between depression and mortality. They found that mortality risk for mental health was attenuated by an indirect pathway through physical health. Specifically, depression led to poorer physical health which was associated with increased mortality risk. Closer examination of the physical health indicators in that study however revealed that physical health was operationalised across four domains including history of heart attack and stroke. Given that past history of physical health was regressed on current depressive symptomology, we believe there is a need to re-examine this proposed depression-

mortality pathway. Also, we believe that an equally plausible hypothesis is that current mental health is a consequence of physical health. Furthermore, we examine the concurrent intra-individual changes in both mental and physical health in the years preceding death. A limitation of findings into the mental health-mortality risk include the use of time-invariant risk factors. That is, mortality risk is typically regressed on mental and physical health assessed at some distal point. This is a significant limitation given the episodic nature of depression and related mental disorders. The current study recognises the findings of prior research which emphasise the need to clearly delineate gender differences in the epidemiology of late-life depression. Whilst women report higher levels of depression across most of their adult lifespan (Bebbington 1998), the extent of gender differences in late-life, and their associations with physical health and mortality, is less clear. There is evidence that gender differences converge (van't Veer-Tazelaar, van Marwijk et al. 2008, Pachana, McLaughlin et al. 2012), persist (Osborn, Fletcher et al. 2002, Zunzunegui, Minicuci et al. 2007) or even reverse, such that men are at greater risk of depression with increasing age (Anstey and Luszcz 2002) and in the years preceding death (Burns, Butterworth et al. 2013). In the current study, we control for these issues by stratifying our analyses by sex.

For the current paper, we first replicate the Batterham study to consider the mortality risk with multiple observations of depression and physical health, utilising data from a large community sample. Second, we compare Batterham and colleagues' (Batterham, Christensen et al. 2012) hypothesis, whereby physical health mediates the relationship between mental health and mortality, with an alternative hypothesis in which mental health mediates the association between physical health and mortality. Given the lack of consistency in findings related to gender differences in the course of late-life depression, we stratify our analyses by gender in order to examine the extent of gender differences in the relationships between mental health, physical health and mortality.

Method

Participants

Data were drawn from the DYNOPTA project (Anstey, Byles et al. 2010). DYNOPTA involves harmonizing data from nine Australian longitudinal studies of ageing. The harmonization of existing studies, by pooling data or parallel analysis, is increasingly recognized as an important method that contributes to, and addresses the limitations of, individual longitudinal studies (Noale, Minicuci et al. 2005). Ethical approval was obtained for all individual studies from relevant supervisory bodies (see acknowledgement), while ethical approval for the DYNOPTA project was approved by the Australian National University Human Research Ethics Committee in accordance with National guidelines and the ethical standards laid down in the 1964 Declaration of Helsinki. Baseline observations were made between 1990 and 2001, yielding 50,652 respondents in the pooled DYNOPTA dataset at baseline. As we are specifically interested in the course of late-life depression and mortality risk and intend comparing our results with other samples of older adults (Batterham, Christensen et al. 2012), we restrict our sample to participants aged 70 years and older (range: 70 – 98 years). So, for this study, participants ($N = 14,019$) were mostly women (91%) and aged 70-95 years ($M = 73$ years; $SD = 3$ years) at baseline. Over the course of the study, approximately 80% were aged 70-79 years of age, 18% were aged 80-89 years of age, and 2% were aged 90+. Participants were observed on at least two occasions up to a maximum of 4 occasions for up to 10 years ($M = 9$ years; $SD = 2$ years) from baseline to the final census date December 2006. The timing of measurement occasions occurred on average 3 years ($SD = 1.5$), 6 years ($SD = .43$) and 8 years ($SD = .46$) from baseline. All eventual decedents ($n = 3,752$, 26% of current sample) were observed between 8 and 3,837 days ($M = 2,054$ days; $SD = 1,065$ days) from death. All decedents completed a minimum of

two observations; 898 (24% of the decedent sample) completed a total of three observations, and 313 (8% of the decedent sample) completed a total of four observations.

Measures

Mortality status: Mortality status was provided by the contributing studies to the DYNOPTA project. Methods of collecting mortality data included collection of death data from the National Death Index, from death notices, and informants. Time to death was calculated from baseline observation until date of death or 31st of December 2006 for right-censored participants (survivors).

Mental Health: We derived wellbeing variables from the Short-Form Health Survey-36 (SF-36) (Ware, Kosinski et al. 1998). The mental health index partly contributes to the SF-36 Mental Health Component Score. The mental health subscale has been validated against clinical measures of depression (Rumpf, Meyer et al. 2001) and has been used in epidemiological studies worldwide as an indicator psychological distress (Skapinakis, Lewis et al. 2005, Gill, Butterworth et al. 2006). The mental health subscale comprises five items including, ‘Been a nervous person’, ‘Felt so down in the dumps nothing could cheer you up’, ‘Felt calm and peaceful’, ‘Felt down’, ‘Been a happy person’. Participants indicated the extent to which they experienced each statement on a 6-point Likert-type scale, ranging from ‘0’ (*None of the Time*) to ‘6’ (*All of the Time*) over the preceding four weeks.

Physical Health: Physical health was assessed with the SF-36 Physical Component Score (Ware, Kosinski et al. 1998), which comprises measures of physical functioning, general health, role-physical and bodily pain. Physical functioning rates the extent to which participants’ health limits their performance in a range of physical activities (e.g. to carry groceries, walk several blocks, bathe oneself). General health reflects how well participants rate their current health status, their susceptibility to disease, and their expectations for their future health. Role-physical reflects the extent to which participants’ health impacts on their

capacity to perform physical activities. Bodily pain rates the extent to which participants' health interferes with their day-to-day activities. Each of these scales has been validated against objective health indicators (Kravitz, Greenfield et al. 1992), including cardiac disease (Zhang, Pozuelo et al. 2010), diabetes mellitus (Davidson 2005), stroke (Kappelle, Adams et al. 1994), low back pain (Garratt, Ruta et al. 1993), lung disease (Viramontes and O'Brien 1994), and renal disease (Osthus, Preljevic et al. 2012).

Covariates: Socio-demographic and health variables.

Time invariant covariates included baseline age, which was centered at 70 years as the reference for the youngest participant, Partner Status, which was defined as Partnered (the reference category; coded as 0) and Not Partnered (coded as 1), and Education status, which was coded as Secondary School or Less (the reference category; coded as 0;), Non-Tertiary Study (coded as 1) and Tertiary Study (coded as 2).

Statistical Analysis

Mental health and the Physical Health Component Scores were *T*-scored ($M = 50$; $SD = 10$) and standardized to baseline observation and coded so that high scores reflected negative/poor health status in order that positive coefficients (and Hazard Ratios > 1) could be interpreted as increased mortality-risk. First we undertook a series of mediation models to replicate the findings of Batterham et al (Batterham, Christensen et al. 2012). Three models were tested: Model B1 tested the unadjusted effect of mental health on mortality-risk; Model B2 re-estimated Model B1, adjusting for age, education and partner status; Model B3 re-estimated Model B2 and included a mediation model whereby physical health mediated the effect of mental health on mortality risk. Then, our multi-level survival analyses were undertaken within a Structural Equation Modelling framework in Mplus v.7.1. Due to the complex nature of the survey design and nested nature of the data (ie. non-independence of repeated observations within the same individual), maximum likelihood (MLR) estimates,

standard errors and model fit statistic were estimated and robust to non-normality and non-independence of observations. MLR standard errors were computed with a sandwich estimator. A series of models were estimated to: i) test the unadjusted univariate effects of mental health (Model 1) and physical health (Model 2) on survival; ii) test the adjusted univariate effects of mental health (Model 3) and physical health (Model 4) on survival (adjusting for partner status, education and baseline age); and iii) compare two mediation models whereby either a) physical health mediated the effect of mental health on mortality-risk (Model 5) or b) mental health mediated physical health on mortality-risk (Model 6). Analyses are stratified by sex whereby the subscript _a in Model 1_a, for example, denotes analysis undertaken on women only and Model 1 without a subscript denotes analysis undertaken on men only.

Results

Mortality Risk of Mental Health

We initially attempted to replicate the analyses of Batterham et al (Batterham, Christensen et al. 2012) by examining the baseline mortality risk for mental health. Results in Table 1 report the HR for mental health and model fit for three models tested by Batterham et al (Batterham, Christensen et al. 2012), stratified by sex. Overall, our results confirm their findings. For males, the inclusion of both covariates and physical health accounted for the mortality risk of mental health (HR = 1.00; $p = .120$), whilst for females the mental health effect (HR = 1.01; $p < .001$) was highly significant, but reflected only a very small effect. Comparison of model fit indicated that the mediation model, whereby physical health mediated the effect of mental health on mortality, was a better fitting model for both men and women than in unadjusted or direct effect-only models. We therefore concluded that our sample produced comparable

results to prior findings and we therefore extended our analyses to consider our alternative hypothesis that mental health is more likely to mediate the effect of physical health.

INSERT TABLE 1

For men, unadjusted univariate analyses indicated small mortality risk for poor mental (Model 1: HR = 1.01 (95%CI = 1.00; 1.02); $p = .012$) and physical health (Model 2: HR = 1.01 (95%CI = 1.00; 1.02); $p = .007$). GFI indicated comparable model fit in these unadjusted analyses for both mental (-LL = 3173; BIC = 6351) and physical health (-LL = 3172; BIC = 6349). Results from adjusted analyses are reported in Table 2. These results suggest that mental health was only weakly associated with mortality risk ($p < .05$). Any effect for mental health needs to be balanced by the non-substantive effect size and the sample size. Indeed, in the mediation models, neither mental health nor physical health revealed direct effects on mortality risk (Models 5 and 6). Whilst the coefficient for physical health regressed on mental health (Model 5) was larger than that estimate with mental health regressed on physical health (Model 6), no significant difference in these effect sizes was observed ($Z = 0.933$; $p = .351$), suggesting that mental health and physical health yield comparably-sized reciprocal effects. In the non-mediation models (Models 3 and 4), model fit was comparable. However, in the mediation models (Model 5 and 6) GFI indicated better fit for the model whereby mental health mediated the effect of physical health on mortality risk (Model 6). However, since neither physical nor mental health was associated with mortality risk in adjusted models, model comparisons are immaterial.

INSERT TABLE 2

Similar to our findings for men, unadjusted analyses for women indicated only small mortality risk for poor mental (Model 1_a: HR = 1.02 (95%CI = 1.02; 1.03); $p < .001$) and physical health (Model 2_a: HR = 1.04 (95%CI = 1.04; 1.04); $p < .001$). In these unadjusted analyses, GFI indicated slightly better model fit for physical health as a risk factor for mortality (-LL = 55608; BIC = 111224) in comparison with mental health (-LL = 55964; BIC = 111936). Across adjusted and mediation models (Table 3), physical health consistently reported stronger direct effects on mortality-risk than mental health. For the mediation analyses, the effect of mental health on physical health (Model 5_a), was larger than the effect of physical health on mental health (Model 6_a). In contrast to men, these effects were significantly different ($Z = 6.90$; $p < .001$), suggesting that for women, their mental health is a stronger driver of physical health. However, despite this stronger effect, and similar to men, GFI indicated superior fit for the model whereby mental health was regressed on physical health (Model 6_a).

INSERT TABLE 3

Examination of the mediation of Physical and Mental Health on Mortality Risk

Since significant effects for physical and mental health were reported in the mediation models for women only, we examined the extent of these mediation effects further in just the women. Specifically, we examined whether the direct effects for mental or physical health on mortality risk was attenuated when they functioned as mediators. We did this by testing the significance of any change in the direct effect for the mediators on mortality in comparison with the earlier non-mediation models that tested the adjusted association between health and mortality risk. For example, the direct effect of mental health on mortality declined significantly in the mediation model (Model 3_a ($\beta = .021$; SE = .002) vs. Model 6_a ($\beta = .013$;

SE = .002): $Z = 2.82$; $p = .004$), suggesting that around 38% of the mental health effect on mortality is accounted for by physical health. A similar decline in the direct effect of physical health on mortality when it functioned as a mediator was also reported although this failed to reach statistical significance (Model 4_a: $\beta = .038$ (SE = .002) vs. Model 5_a: $\beta = .036$ (SE = .002); $Z = 0.701$; $p = .478$). This suggests that despite the stronger effect for mental health on physical health, this does not attenuate the physical health-mortality risk relationship. Overall, the mortality-risk for physical health appears more robust and substantive, approximately 4 times stronger than the effect reported by mental health.

Discussion

In this study we sought to extend prior research into the association between mental health and mortality risk. First, we extended the current research base by including longitudinal data for a large sample of older adults on the assumption that increased information in the years leading to death will provide more detailed mortality-related information for deceased participants especially. Given the episodic nature of mental illnesses, such as depression, increased coverage for decedents, particular in the years immediately preceding death, are important to efficiently estimate 'true' population parameters. Second, we re-evaluated the association between mental and physical health in mortality risk. In contrast to prior research (Batterham, Christensen et al. 2012), we postulated two main hypotheses: First, does mental health mediate the effect of physical health on mortality risk, or second, does physical health mediate the effect of mental health on mortality risk? For men we can conclude that there are no substantive associations for mental and physical health on mortality risk in adjusted or mediation analyses. A very small effect for mental health only just reached statistical significance in the adjusted model only and is likely a consequence of sample size; its effect size is non-substantive. Although no mediation effects could be identified, GFI did indicate

support for a model whereby mental health is regressed on physical health. In contrast, for women, associations between mental health and physical health with mortality were reported in adjusted and mediation analyses with stronger effect sizes suggesting this gender difference is not an artefact of greater statistical power. Physical health revealed stronger effects on mortality risk and accounted for half of the mental health-related mortality-risk. Although mental health was a significantly stronger driver of physical health, as for the analyses of men, GFI indicated stronger support for a model whereby mental health was regressed on physical health. Of particular note, no indirect effects were reported; physical and mental health revealed only direct effects on mortality risk.

The failure to identify any mediation effects contrasts with other findings (Batterham, Christensen et al. 2012). These differences cannot be attributed to a different sampling frame as our initial replication of Batterham et al (Batterham, Christensen et al. 2012) confirmed their results for baseline mortality-risk. For both males and females, mortality-risk data were best reflected by a model in which physical health mediates the effect of mental health on mortality risk. Given our replication of these earlier findings in our sample, this provides more robust support for our findings and more defensible hypothesis that it is mental health that mediates the association of physical health on mortality-risk. The findings from prior studies are not without other caveats. Much of the research into the depression-mortality link has failed to control for confounding physical health states (Harris and Barraclough 1998, Saz and Dewey 2001, Cuijpers and Schoevers 2004) and where physical health has been controlled for, operationalization has limited the extent to which meaningful conclusions can be drawn (Batterham, Christensen et al. 2012). Importantly we have utilised longitudinal data which has examined mental and physical health repeatedly at the at multiple measurement occasions. Our findings demonstrate that the effect of mental health on mortality in a large observational study is weak at best, especially in contrast to the effect for physical health.

However, even the effect for physical health was small in contrast to increasing age and the other socio-demographic ‘control’ variables. In line with the existing literature (Anstey and Luszcz 2002, Burns, Butterworth et al. 2013), we have also identified gender differences in the way mental health and physical health are related to mortality. Clearly, the association between of mental health and mortality-risk was more pronounced in women. Given the increasing heterogeneity of older populations, it is important to examine assumptions of generalisation across different samples population and socio-demographic populations, particularly with the reported gender differences identified in this study.

Despite reporting a negligible effect on survival, for women, mental health actually produced a stronger effect on physical health than did physical health on mental health. This could be explained by the limitation of our study in which the analyses are based on self-report measures. It could be that current physical health assessment are based on self-assessments and that concurrently poor mental health is driving down women’s assessment of their physical health and conversely, good physical health assessment is buoyed by positive mental health states. This effect can only be quantified by the inclusion of objective physical health states which are not available in DYNOPTA

The current study has compared two pathways by which mental health may place individuals at increased mortality risk. First, we examined the extent to which mental health mediates the effect of physical health on mortality. Second, we examined the extent to which physical health mediates the effect of mental health on mortality. We found little support for either hypothesis. Instead, we found the mental health risk for mortality was fully attenuated by physical health in men and partially so for women. Physical health was implicated as a substantive mortality risk for women.

Conflicts of Interest:

No conflict of interest to declare.

Description of Authors' Roles

All authors have read and approved the final version of this manuscript. Specifically, RAB: Formulated the research question, determined the analytical methods and undertook the statistical analysis, and led the writing of the manuscript; PB contributed to the data collection, analytical method and analyses, and contributed to writing the manuscript; CB JB ML PM JS KJA: Supervised the data collection, contributed to writing the manuscript

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Table 1. Mortality Risk for Poor Mental Health following Batterham et al (Batterham, Christensen et al. 2012).

	Women (n = 12, 461)			Men (n = 1, 558)		
	Model B1 _a	Model B2 _a	Model B3 _a	Model B1	Model B2	Model B3
	HR	HR	HR	HR	HR	HR
	(95% CI)	(95% CI)	(95% CI)	(95% CI)	(95% CI)	(95% CI)
Mental Health	1.02	1.02	1.01	1.02	1.01	1.01
	(1.02; 1.02)***	(1.02; 1.02)***	(1.01; 1.02)***	(1.01; 1.03)**	(1.00; 1.02)*	(0.99; 1.02)
Goodness of Fit Indices						
LL	-216528	-216425	-215864	-17108	-17078	-17031
AIC	433066	432869	431751	34227	34174	34084
BIC	433087	432907	431797	34235	34188	34102

***p < .001; **p < .01; * p< .05. Model B1: Mortality regressed on MH; Model B2: = Model B1 + adjusted for age, partner status and education; Model B3: = Model B2 + PH mediates regression of Mortality on MH. LL: Log likelihood; AIC: Akaike Information Criteria; BIC: Bayesian Information Criteria.

Table 2 Cox proportional hazards regression models predicting time to death for men (n = 1, 558)

	Model 3	Model 4	Model 5	Model 6
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Mental Health	1.01 (1.00; 1.02) [*]		1.01 (0.99; 1.02)	1.01 (0.99; 1.02)
Physical Health		1.01 (0.99; 1.02)	1.01 (0.99; 1.01)	1.01 (0.99; 1.01)
Age	1.09 (1.07; 1.11) ^{***}	1.09 (1.07; 1.11) ^{***}	1.09 (1.07; 1.11) ^{***}	1.09 (1.07; 1.11) ^{***}
Not Partnered ^a	1.49 (1.14; 1.95) ^{**}	1.48 (1.13; 1.94) ^{**}	1.49 (1.14; 1.94) ^{**}	1.49 (1.14; 1.94) ^{**}
Non-Tertiary ^b	0.89 (0.69; 1.16)	0.84 (0.65; 1.08)	0.90 (0.69; 1.61)	0.90 (0.69; 1.61)
Tertiary ^b	1.07 (0.72; 1.59)	1.08 (0.73; 1.61)	1.08 (0.73; 1.61)	1.08 (0.73; 1.61)
Mental Health →			.30 (.24; .37) ^{***}	
Physical Health ⁺				
Physical Health →				.26 (.20; .32) ^{***}
Mental Health ⁺				
-LL	-3137	-3138	-8019	-7922
BIC	6294	6296	16074	15880

⁺ effects for mediation paths reflect unstandardized estimates; *** $p < .001$, ** $p < .01$, * $p < .05$. ^a Partner Status Reference = Partnered; ^b

Education Status Reference = Secondary Schooling. LL: Log likelihood; AIC: Akaike Information Criteria; BIC: Bayesian Information Criteria.

Table 3 Cox proportional hazards regression models predicting time to death for women (n = 12, 461)

	Model 3 _a	Model 4 _a	Model 5 _a	Model 6 _a
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Mental Health	1.02 (1.02; 1.03) ^{***}		1.01 (1.01; 1.02) ^{***}	1.01 (1.01; 1.02) ^{***}
Physical Health		1.04 (1.03; 1.04) ^{***}	1.04 (1.03; 1.04) ^{***}	1.04 (1.03; 1.04) ^{***}
Age	1.10 (1.09; 1.03) ^{***}	1.09 (1.08; 1.11) ^{***}	1.09 (1.08; 1.11) ^{***}	1.09 (1.08; 1.11) ^{***}
Not Partnered ^a	1.15 (1.06; 1.25) ^{**}	1.13 (1.04; 1.23) ^{**}	1.13 (1.04; 1.23) ^{**}	1.13 (1.04; 1.23) ^{**}
Non-Tertiary ^b	1.03 (0.90; 1.16)	1.02 (0.90; 1.15)	1.04 (0.92; 1.18)	1.04 (0.92; 1.18)
Tertiary ^b	0.77 (0.60; 1.00) [*]	0.82 (0.63; 1.06)	0.84 (0.64; 1.08)	0.84 (0.64; 1.08)
Mental Health →			.29 (.27; .30) ^{***}	
Physical Health				
Physical Health →				.22 (.21; .23) ^{***}
Mental Health				
-LL	-55821	-55492	-186268	-181492
BIC	111680	111021	372602	363051

⁺ effects for mediation paths reflect unstandardized estimates; *** $p < .001$, ** $p < .01$, * $p < .05$. ^a Partner Status Reference = Partnered; ^b

Education Status Reference = Secondary Schooling. LL: Log likelihood; AIC: Akaike Information Criteria; BIC: Bayesian Information Criteria