



The relationship between cognitive reserve and outcome after controlling for psychological status and sex following mild traumatic brain injury

Jacqueline F. I. Anderson & Laura Martin

To cite this article: Jacqueline F. I. Anderson & Laura Martin (2023): The relationship between cognitive reserve and outcome after controlling for psychological status and sex following mild traumatic brain injury, *Brain Injury*, DOI: [10.1080/02699052.2023.2222642](https://doi.org/10.1080/02699052.2023.2222642)

To link to this article: <https://doi.org/10.1080/02699052.2023.2222642>



Published online: 08 Jun 2023.



Submit your article to this journal [↗](#)



View related articles [↗](#)



View Crossmark data [↗](#)



The relationship between cognitive reserve and outcome after controlling for psychological status and sex following mild traumatic brain injury

Jacqueline F. I. Anderson ^{a,b} and Laura Martin ^a

^aMelbourne School of Psychological Sciences, The University of Melbourne, Melbourne, Victoria, Australia; ^bPsychology Department, The Alfred Hospital, Melbourne, Victoria, Australia

ABSTRACT

Objective: Cognitive reserve is the brain's ability to optimize performance by differentially recruiting brain networks. It is easily measured and is reportedly associated with post-concussion symptom (PCS) reporting in the post-acute period after mild traumatic brain injury (mTBI). Past studies have not examined whether this relationship exists when the influence of psychological status is removed, despite this factor being strongly associated with symptom reporting. This study investigated whether cognitive reserve predicts PCS reporting or cognitive complaint in the post-acute period after mTBI, independently from psychological status and sex.

Method: Ninety-four pre-morbidly healthy adults were assessed on three measures of cognitive reserve, as well as measures of post-concussion symptoms, cognitive complaint, and psychological status.

Results: Bivariate analyses revealed significant relationships between measures of cognitive reserve and both PCS reporting ($p < 0.01$) and cognitive complaint ($< .05$). After removing the influence of psychological distress and sex, however, no measure of cognitive reserve significantly predicted any type of symptom reporting.

Conclusion: These findings indicate that cognitive reserve does not independently predict symptom reporting 9 weeks after mTBI, and clinicians should not incorporate this factor into their decision-making regarding likelihood of ongoing symptom reporting and the consequent need for intervention in the post-acute period after mTBI.

ARTICLE HISTORY

Received 27 July 2022

Revised 21 May 2023

Accepted 2 June 2023

KEYWORDS

Mild traumatic brain injury; cognitive reserve; post-concussion symptoms; cognitive symptoms; psychological distress

Mild traumatic brain injury (mTBI) is common in the general population (excluding professional athletes and war veterans). It occurs in more than 100–300 hospital-treated cases per 100,000 individuals each year (1). It has been well established that at least 20% of these individuals continue to report post-concussion symptoms (PCS) more than 3 months after injury (2), with more recent research reporting elevations in symptoms in more than 50% of individuals, 12 months after injury (3,4). Post-concussion symptoms comprise a range of physical, affective, and cognitive difficulties that are commonly experienced in the acute period after mTBI (5). It is well established that PCS deleteriously impact quality of life, general well-being, and have a significant negative effect on socio-economic status (6–9). Identifying groups at high risk of PCS would facilitate early and targeted intervention for these individuals. Female sex (10–13), as well as older (14) and younger (15) age at injury have been identified as being associated with higher risk of PCS after mTBI. These factors only have limited predictive value in identifying those individuals who will continue to experience PCS in the post-acute period (>6 weeks) after mTBI, however. Investigation of other factors that can predict PCS is therefore warranted.

Despite studies demonstrating a relationship between cognitive reserve and objective cognitive functioning following mTBI (16,17), the relationship between cognitive reserve and the reporting of subjective PCS has received limited attention.

Cognitive reserve is defined as the brain's ability to optimize performance by differentially recruiting brain networks. Considered to be a normal response to increased task demands, it is present in healthy individuals as well as in those with brain damage (18). Cognitive reserve theory posits that individuals vary with respect to the amount of cognitive reserve they can draw on to compensate for the effects of brain damage (18). It has been suggested that this inter-individual variability in cognitive reserve may contribute to inter-individual variability in outcome after mTBI (16,17).

There has been limited literature examining the relationship between cognitive reserve and PCS. The two studies that have investigated this question have reported that individuals with lower levels of cognitive reserve are more likely to experience elevated PCS than those with higher cognitive reserve (10,16). In these studies, cognitive reserve was determined from estimated premorbid IQ and/or education; analyses including occupation as a measure of cognitive reserve approached significance, but sample size was small. Neither of these studies investigated the relationship between cognitive reserve and PCS while controlling for the influence of psychological status, however. This is problematic because it has been demonstrated repeatedly that psychological distress is prevalent after mTBI (19,20) and impacts PCS reporting (21–25).

An aspect of PCS that has been reported to be particularly relevant to individual variations in quality of life and return to

work after mTBI, is subjective cognitive symptoms, or 'cognitive complaint' (26–28). Although a component of PCS, in the post-acute period after mTBI cognitive complaint has been shown to be a distinct and independent factor relative to the broader construct of PCS (29). Therefore, the relationship between cognitive complaint and cognitive reserve warrants further investigation. The limited literature on this topic has reported that individuals with cognitive complaint 6 months after injury have lower educational attainment than those without cognitive complaint (30,31). As with PCS, neither of these studies examined the relationship between cognitive reserve and cognitive complaint while controlling for psychological status. This is problematic because cognitive complaint has also been shown to have a robust association with psychological status (12,32).

It is important to understand the relationship between cognitive reserve and subjective post-concussion and cognitive symptom reporting after mTBI, independently of psychological status. In particular, management and treatment decisions would benefit from clinicians knowing whether variations in cognitive reserve can independently predict variations in the likelihood of ongoing symptom reporting after mTBI.

Measurement of cognitive reserve varies substantially within the literature, with researchers suggesting that it should be conceptualized as a multidimensional construct in individuals with TBI (33). Specifically, cognitive reserve in TBI populations has been modeled to include pre-morbid IQ, socioeconomic status, and pre-injury leisure activities (33). Pre-morbid IQ has been shown to be the most significant predictor of post-TBI outcomes (34), whereas proxies for socioeconomic status, in particular education and occupation, are the most commonly used measures of cognitive reserve in the literature.

The aim of this study was to investigate the relationship between measures of cognitive reserve and PCS and cognitive complaint. On the basis of the limited literature, it was hypothesized that education, occupation, and pre-morbid IQ would significantly predict PCS and cognitive complaint levels, independently of age and sex, in pre-morbidly healthy adults in the post-acute period (>6 weeks) after mTBI.

Method

Participants

Participants comprised 94 adults (71 males, 23 females) who had suffered a mTBI between September 2015 and February 2020 and were consecutively admitted to The Alfred hospital or Royal Melbourne Hospital, Melbourne, Australia, in the preceding 6–12 weeks. Initial hospital admission occurred as a consequence of suffering any traumatic injury (i.e. primarily systemic and/or head). All individuals who had been admitted with traumatic injury were approached to determine if they fulfilled criteria for mTBI. Thus, many individuals were not admitted due to their mTBI, which was diagnosed post-admission. Identical recruitment processes were undertaken at each hospital.

A mTBI event was defined according to World Health Organisation criteria (2). Briefly, individuals needed to

demonstrate one or more of: i) confusion or disorientation, loss of consciousness for 30 min or less, post-traumatic amnesia (PTA) less than 24 h, and/or other transient neurological abnormalities not requiring surgery; ii) Glasgow Coma Scale (GCS) score of 13–15 after 30 min, or later upon presentation for health care. Exclusion criteria were as follows: any previous neurological history, including documented TBI, diabetes, history or current IV or Class A drug (e.g. heroin, cocaine, ecstasy, LSD) or heavy alcohol use (>5 standard drinks/day), history of diagnosis or treatment for any significant psychiatric disorder, current/recent (during previous 12 months) diagnosis or treatment of depression and/or anxiety and/or post-traumatic stress disorder, current TBI as a result of physical assault/attack and lack of conversational English fluency.

Measures

Measures of cognitive reserve

Educational Attainment (Education) was measured as self-reported number of full-time equivalent years of education, with 12 years corresponding to completion of a secondary school education.

Occupation was classified according to the Australian and New Zealand Standard Classification of Occupations (ANZSCO) (35). The highest level of eight occupational hierarchical groupings was used. These eight groups were collapsed into two meaningful groups of approximately equivalent size, on the basis of occupational cognitive demand: Higher ($n = 37$; ANZSCO Major Groups 1 and 2; Managers and Professionals) and Lower ($n = 45$; ANZSCO Major Groups 3–8; Technicians and Trade Workers, Community and Personal Workers, Clerical and Administration Workers; Sales Workers, Machinery Operators and Drivers, and Labourer). Self-reported students ($n = 12$) were re-coded as missing data.

Pre-morbid IQ was determined from the Wechsler Test of Adult Reading (WTAR) (36), a word reading task from which accurate estimates of premorbid intellectual functioning can be derived in individuals with mTBI (37).

Measures of post-concussion symptoms and cognitive complaint

The *Rivermead Post Concussion Symptoms Questionnaire* (RPQ) is a widely used measure of PCS. It assesses physical (10 items), psychological (3 items), and cognitive (3 items) symptoms experienced during the past 24 h (5).

The *Cognitive Complaint After Mild Closed Head Injury* (CCAMCHI) (32), is a 30-item questionnaire that assesses change in subjective cognitive complaints in those domains most commonly affected by mTBI: processing speed, attention, memory, and executive function. Total scores have a possible range of 30 (Much better cognitive functioning than prior to injury) to 150 (Much worse cognitive functioning than prior to injury). A total score of 90 indicates no change in cognitive complaints compared to previously. It has been used previously with mTBI samples (32).

Measures of depression, anxiety, and post-traumatic stress

Three widely used, valid, and reliable questionnaires of psychological status were used. The *Inventory of Depressive*

Symptomatology (IDS) is a 30-item measure of severity of overall depression (38). The *Beck Anxiety Inventory* (BAI) is a 21-item measure of anxiety symptomatology (39). The *PTSD Checklist for the DSM-5* (PCL-5) (40) is a 20-item measure of the symptoms of PTSD defined by DSM-5. To facilitate data reduction, a single variable of Psychological Distress was created from standardized performances on the IDS, BAI, and PCL-5. The IDS and BAI are 4-point scales (range 0–3), whereas the PCL-5 is a 5-point scale (0–4). Consequently, each response on the PCL-5 was multiplied by .75; performance on these measures was then summed together, resulting in a single index of psychological distress, with a possible range of 0–213.

Procedure

Participants with mTBI were initially recruited into the study on the ward within 1–4 days after injury, during their inpatient stay. Written informed consent to participate was given following fulfillment of inclusion/exclusion criteria. Following the discharge, participants were contacted by phone and attended The Alfred hospital to undertake an individual assessment 6–12 weeks after injury. All participants completed the measures in the following order: WTAR, CCAMCHI, RPQ, IDS, BAI, PCL-5.

Data analysis

Data for 26 participants was missing for the CCAMCHI, due to an administration error. There were two missing data points for the WTAR, RPQ, and psychological distress variables. Little's Missing Completely at Random (MCAR) test revealed that the missing data for all variables occurred completely at random ($\chi^2 = 15.47$, $df = 11$, $p = 0.162$). Statistical assumptions were investigated prior to data analysis. A square root transformation was conducted on the RPQ and the Psychological Distress index to correct mild deviations from normality. A Log10 transformation was conducted on the CCAMCHI variable to correct a moderate deviation from normality. The normality of all transformed variables was within acceptable limits (41). No other assumption violations occurred. Pearson correlations were used to investigate linear associations between variables; point-biserial coefficients were interpreted for dichotomous variables. Occupation was measured on an ordinal scale, so Spearman's correlations were used for this variable. Hierarchical linear regression analyses were used to determine if occupation, education, or pre-morbid IQ each significantly predicted symptom reporting, with age and sex (plus litigation for models involving the RPQ) entered in Step 1, psychological distress entered in Step 2 and the relevant cognitive reserve variable entered in Step 3.

Results

Study recruitment pathways have been described previously in detail (21,42). Demographic and injury details are presented in Table 1.

From a possible range of 0–180, the participants with mTBI reported relatively low levels of psychological distress, on

average. After excluding students, there was a broadly even number of participants with higher occupational cognitive demands (39.4%) compared to those with lower occupational cognitive demands (47.8%). The proportion of individuals who were professionals/managers (39%) is consistent with 2021 census data, which indicates 37% of the Australian adult population fell within these categories (43). Litigation strongly trended toward a significant association with RPQ performance ($r_{pb} = .206$, $p = 0.050$), but was not associated with endorsement of cognitive symptoms on the CCAMCHI ($r_{pb} = .045$, $p = 0.726$). Given that being in litigation was strongly trending toward being significantly associated with greater PCS symptom endorsement, litigation status was included in Step 1 of regression analyses that incorporated the RPQ as the dependent variable.

Results of the correlational analyses between the measures of age, sex, psychological distress, cognitive reserve, and symptom reporting are presented in Table 2.

Female sex was associated with increased likelihood of PCS reporting, but not cognitive symptom reporting, whereas higher psychological distress was associated with increased likelihood of all types of symptom reporting. Linear associations between cognitive reserve and symptom reporting were variable; lower years of education were associated with increased PCS and cognitive symptom reporting, but lower cognitive load in occupation was only associated with increased PCS reporting. No linear association was evident between pre-morbid IQ and symptom reporting.

The overall results of the final linear regression models, which regress background variables and cognitive reserve variables onto each symptom reporting measure are presented in Table 3.

All models significantly predicted RPQ and CCAMCHI performance, but none of the models were significantly better predictors of symptom reporting at Step 3, when the cognitive reserve variable had been added, relative to Step 2 of the model, when psychological distress was added. In contrast, for each model, the inclusion of psychological distress at Step 2, significantly improved the predictive ability of the model relative to background variables alone. The parameters of each model, which demonstrate the independent contribution of each variable to the six final linear regression models, are presented in Table 4.

For PCS reporting, psychological distress independently predicted RPQ performance in all models; sex was also an independent predictor in two (Education and Pre-morbid IQ) of the three models. In contrast, for subjective cognitive impairment reporting, sex did not significantly predict CCAMCHI performance in any model. As with the RPQ, psychological distress significantly and independently predicted CCAMCHI performance in all of the models.

Discussion

In contrast to expectations, none of the cognitive reserve variables predicted either PCS reporting or cognitive complaint independently of age, sex, and psychological status. This finding appears to contrast the small number of studies that have examined these relationships previously (10,16). No earlier

Table 1. Background demographic details and injury characteristics.

Background Variable (<i>n</i> = 94)	Frequency (%)
Age (years) Mean (sd)	37.05 (14.01)
Range	18–60
Sex	75.5% Male; 24.5% Female
Education (years) Mean (sd)	13.37 (2.31)
Range	10–19
Premorbid IQ Mean (sd)	105.8 (9.50)
Range	81–124
Psychological Distress Index Mean (sd)	29.24 (22.92)
Range	0.75–95.00
Employment Status	
Full-time work	54.3%
Part-time work	17%
Not working	28.7%
Occupation	
Professionals	22.4%
Managers	17%
Technicians/Trade	20.3%
Community/Personal	5.3%
Clerical/Admin	7.4%
Sales	5.3%
Machinery	2.1%
Labourers	7.4%
Students	12.8%
Involved in litigation	11%
Drug use history	
None	95.7%
Occasional past Class A drug use	3.2%
Current Class B drug use	2.1%
Alcohol use (drinks/week)	
None	22.3%
<2	26.6%
3–10	36.2%
11–20	13.8
>35	1.1%
Injury Descriptor	Frequency (%)
Days since injury Mean (sd)	60.72 (10.61)
Range	37–85
Injury cause	
MVA	19.1%
MBA	12.8%
Cycling	27.7%
Fall	21.3%
Sport	7.4%
Other	11.7%
GCS Mean (sd)	14.55 (.67)
13	9.7%
14	25.8%
15	64.5%
LOC	
None	10.6%
<5 min	75.6%
5–10 min	5.3%
10–30 min	7.4%
PTA	
<1 min	32.9%
1–5 mins	24.6%
5–60 mins	15.9%
1–6 hours	8.5%
6–12 hours	8.5%
12–24 hours	9.6%

Note: MVA: Motor vehicle accident; MBA: Motorbike accident; GCS: Glasgow Coma Scale score; LOC: Loss of consciousness; PTA: Post traumatic amnesia.

study controlled for the effect of psychological status when investigating the association between cognitive reserve and symptom reporting, however, which prevents a meaningful comparison.

The previously reported relationship between cognitive reserve and PCS reporting, prior to controlling for other factors, corresponds to the significant findings of the bivariate analyses in the present study. That is, the bivariate correlations

in the present study demonstrated a linear association between PCS reporting and years of education as well as occupation. It was only with the removal of the significant influence of sex and psychological distress that the purported linear relationship between cognitive reserve and PCS reporting was shown to be spurious. The significant bivariate relationships are consistent with previous studies that have found a relationship between cognitive reserve and PCS reporting (10,16).

Table 2. Bivariate correlations between background variables, cognitive reserve variables, and measures of symptom reporting.

Background and Cognitive Reserve Variables	Measures of Symptom Reporting	
	RPQ	CCAMCHI
Age (years)	-.193	-.138
Sex (0=F; 1=M)	-.357***	-.194
Psychological Distress	.684***	.534***
Education (years)	-.223*	-.256*
Occupation (lower vs. higher)	-.297**	-.181
Pre-morbid IQ	-.194	-.063

Note: Occupation (lower vs. higher): categorized on the basis of occupational cognitive demand; RPQ: Rivermead Post Concussion Symptoms Questionnaire; CCAMCHI: Cognitive Complaint after Mild Closed Head Injury questionnaire; *= $p < .05$; **= $p < .01$; ***= $p < .001$.

Table 3. Model significance of each linear regression model for RPQ and CCAMCHI.

DV	IVs Steps 1 & 2	IV Step 3	F	p	Step 1 Adj R ²	Step 2 Adj R ²	R ² Δ	Step 3 Adj R ²	R ² Δ
RPQ	S1: Age, Sex, Litigation S2: Psych Distress	Education	14.795	<.001	.200	.425	.224***	.437	.018
	S1: Age, Sex, Litigation S2: Psych Distress	Occupation	16.656	<.001	.151	.425	.271***	.418	.001
	S1: Age, Sex, Litigation S2: Psych Distress	Pre-morbid IQ	12.318	<.001	.189	.404	.214***	.397	.000
CCAMCHI	S1: Age, Sex S2: Psych Distress	Education	4.034	.003	.019	.189	.175**	.197	.021
	S1: Age, Sex S2: Psych Distress	Occupation	3.664	.007	-.017	.214	.233***	.201	.003
	S1: Age, Sex S2: Psych Distress	Pre-morbid IQ	3.643	.006	.019	.189	.175**	.176	.001
	S1: Age, Sex S2: Psych Distress								

Note: RPQ: Rivermead Post Concussion Symptoms Questionnaire; CCAMCHI: Cognitive Complaint after Mild Closed Head Injury questionnaire; **= $p < .01$; ***= $p < .001$.

Table 4. Parameters for Model 3 of each hierarchical linear regression model for RPQ and CCAMCHI by cognitive reserve variable.

Dependent variable	Independent variable	b	SE B	b	p-value
RPQ	Age	.006	.009	.059	.509
	Sex	-.737	.298	-.217	.015
	Litigation	.634	.391	.135	.108
	Psychological Distress	.036	.006	.535	<.001
	Education	-.093	.056	-.144	.100
	Age	.008	.011	.073	.446
	Sex	-.345	.336	-.098	.308
	Litigation	.736	.420	.158	.084
	Psychological Distress	.040	.007	.594	<.001
	Occupation	-.120	.293	-.040	.682
	Age	.000	.010	-.004	.969
	Sex	-.652	.306	-.193	.036
	Litigation	.654	.410	.142	.115
	Psychological Distress	.035	.006	.530	<.001
CCAMCHI	Pre-morbid IQ	-.001	.015	-.009	.922
	Age	.000	.000	.046	.726
	Sex	-.010	.007	-.162	.172
	Psychological Distress	.001	.000	.440	.001
	Education	-.002	.001	-.158	.214
	Age	.000	.000	.060	.665
	Sex	-.001	.008	-.022	.865
	Psychological Distress	.001	.000	.514	<.001
	Occupation	-.003	.006	-.059	.660
	Age	.000	.000	-.029	.826
	Sex	-.009	.007	-.145	.225
	Psychological Distress	.001	.000	.452	.001
	Pre-morbid IQ	.000	.000	.040	.755

Note: RPQ: Rivermead Post Concussion Symptoms Questionnaire; CCAMCHI: Cognitive Complaint after Mild Closed Head Injury questionnaire.

Although earlier studies found a relationship between pre-morbid IQ and PCS reporting, which was absent in the current findings, the measure of pre-morbid IQ differed between the studies. Whereas previous studies used a single 'hold' subtest (Vocabulary or Information) of the Wechsler Adult Intelligence Scale (WAIS) as a proxy for pre-morbid IQ, the current study used a formal estimate of pre-morbid IQ derived from the WTAR. This is a more reliable determination of pre-

morbid IQ than the approach taken by earlier studies (44), and provides support for the generalizability of the current findings.

A similar picture emerged from the current study with respect to cognitive complaint. Prior to removing the significant influence of psychological distress, education was significantly and linearly associated with cognitive symptom reporting, which is consistent with previous findings (30,31).

After removing the influence of psychological distress, however, this relationship was no longer evident. This indicates that the current findings of no independent relationship between cognitive reserve and symptom reporting 9 weeks after mTBI cannot be attributed to an anomaly of the current sample as this sample's performances were consistent with previously published data.

Consistent with recent evidence that female sex is associated with an increased likelihood of PCS reporting (10,12,13), the present study showed that sex was a significant independent predictor of PCS reporting in two of the three regression models. Interestingly, however, sex did not independently predict cognitive complaint in any model, suggesting that the relationship between sex and symptom reporting after mTBI may be related to the more generic PCS items (physical and affective symptoms) that are elevated in control participants as well as individuals with mTBI (45–47) rather than cognitive symptoms per se.

In contrast to the variability associated with the impact of sex on symptom reporting, age did not independently predict any symptom reporting in any model. Despite previous findings of age being associated with levels of symptom reporting (14,15), this was not replicated in the current study. Given that earlier studies have variously found that both younger age and older age are associated with increased symptom reporting, the relationship between age and symptom reporting appears to be unclear at this time. Certainly, the current findings of no relationship appear uncontroversial.

These results have implications for clinical decision-making regarding active intervention vs. passive management strategies in the post-acute period after mTBI as they provide facilitative evidence for successfully predicting post-acute symptom reporting. Specifically, the present study indicates that individuals with higher cognitive reserve are equivalently likely to report PCS and cognitive symptoms 9 weeks after injury as those with lower cognitive reserve. In contrast, higher psychological distress and female sex are significant predictors of PCS reporting and/or cognitive complaint in the post-acute period. Therefore, it may be relevant for a clinician to consider the extent of psychological distress and sex when attempting to predict whether symptom reporting is likely to be ongoing in the post-acute period, and therefore require intervention (48). In contrast to the implication of previous studies, however, the level of cognitive reserve appears not to be pertinent to this decision-making process.

The primary limitation of the current study is the modest sample size as it increases the risk of making Type II (false negative) errors and thereby reducing the generalizability of the findings. The consistency of the pattern of findings strengthens the likelihood that the results are generalizable (49,50). Post-hoc analysis also supported the present sample size as adequate; the smallest achieved power for the CCAMCHI linear regression analyses, which were the analyses with the smallest sample size ($n = 68$), was .91. This indicates a high degree of power was present to identify a significant finding for the medium effect size that was evident in this analysis. Thus, there is no evidence to suggest that there were any significant

predictive relationships that remained unrecognized due to the modest sample size.

The inclusion of litigants (11%) could be considered a further limitation of the present study as previous research has shown that these individuals respond differently on measures of symptom endorsement (22). Analysis of the data demonstrated that litigation trended toward having a significant relationship with RPQ performance but no relationship with CCAMCHI responses. Consequently, to conservatively manage a possible relationship, litigation status was included in regression analyses for the RPQ and was found to have no significant independent predictive relationship with PCS reporting. By incorporating litigation status in the analyses, the present study provides evidence that the lack of relationship between cognitive reserve and PCS is independent of any possible impact of litigation status, and supports the generalizability of the present findings.

It might appear that the high proportion of males and selection for pre-morbid healthiness might bias the findings. The high proportion of males (75%) is broadly consistent with the epidemiology of the mTBI population in the community (51), however, and indicates that the sample is appropriately representative of the naturally occurring sex differences that occur in this population. In contrast, pre-morbid healthiness is less common in the broader mTBI population than in the normal population, as those with mTBI more commonly have a history of TBIs, neurological and psychiatric illness and higher drug and alcohol use (52). Consequently, the current pre-morbidly healthy sample does not correspond to the broader mTBI population. All of these pre-morbid health factors can impact symptom reporting after mTBI, however (45), which results in inferential uncertainty regarding the independent role of other factors in predicting symptom reporting. The current study's approach of examining a pre-morbidly healthy sample of individuals enables a direct assessment of the relationship between symptom reporting and a range of variables without needing to control for additional influential factors. Consequently, the present finding that there is no independent predictive relationship between cognitive reserve and PCS or cognitive symptom reporting provides a meaningful insight into the role of cognitive reserve in general outcome after mTBI.

Finally, as with most studies of mTBI, which typically recruit from hospitals and other medical settings, the present findings cannot be considered to fully reflect the broad population of individuals with mTBI, many of whom seek no medical care at all (51). While the current sample was derived from hospitalized individuals, however, participants were admitted for inpatient care following any type of traumatic injury, with recruitment diagnosis of mTBI occurring after admission. This means that many individuals in the sample were not admitted because of a mTBI diagnosis, making it likely that the sample contained the full spectrum of mTBI severity that is present in the general mTBI population. Participant injury details show a range of mTBI symptom severity and duration, with 60% having a Glasgow coma scale (GCS) score = 15, 76% of the sample experiencing loss of consciousness for less than 5 min and 58% of the sample suffering post-traumatic amnesia of less than 5-min duration.

Thus, while the present study did not include individuals who sought no medical care, it seems reasonable to consider that a broad spectrum of mTBI severity was present in this sample. This provides support for the likely generalizability of these findings to a broader population of individuals with mTBI.

Cognitive reserve is an easily measurable factor that has previously been reported to be significantly associated with symptom reporting in the post-acute period after mTBI. The present study indicates that this purported relationship is spurious as no relationship between cognitive reserve and PCS, or cognitive symptom reporting, remains evident when the influence of psychological distress is removed. In contrast, psychological distress is independently predictive of both PCS and cognitive symptom reporting, as is sex for PCS reporting. These findings indicate that clinicians should *not* incorporate cognitive reserve into their decision-making regarding likely continuance, and therefore need for intervention, of symptom reporting in the post-acute period after mTBI; psychological distress and sex may be important factors to consider, however.

Acknowledgments

The authors would like to acknowledge the contribution of post-graduate students and research assistants: Georgia Bolt, Emily Cockle, Nicolette Ingram, Arielle Levy, Courtney Lewis, Joshua Nash, Lucy Oehr, Katie Priestley, Aimee Savage, Nicola Singleton and Patrick Summerell for their assistance in collecting these data. This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Disclosure statement

No conflicts of interest were declared by any author

Funding

This study was supported by grants from Melbourne School of Psychological Sciences at The University of Melbourne.

ORCID

Jacqueline F. I. Anderson  <http://orcid.org/0000-0003-4996-8189>
 Laura Martin  <http://orcid.org/0000-0001-6679-7700>

References

- Carroll LJ, Cassidy JD, Cancelliere C, Cote P, Hincapie CA, Kristman VL, Holm LW, Borg J, Nygren-de Boussard C, Hartvigsen J. Systematic review of the prognosis after mild traumatic brain injury in adults: cognitive, psychiatric, and mortality outcomes: results of the international collaboration on mild traumatic brain injury prognosis. *Arch Phys Med Rehabil*. 2014;95(3):S152–73. doi:10.1016/j.apmr.2013.08.300.
- Carroll LJ, Cassidy JD, Holm L, Kraus J, Coronado VG. Injury WHOCTFoMTB. Methodological issues and research recommendations for mild traumatic brain injury: the WHO collaborating centre task force on mild traumatic brain injury. *J Rehabil Med*. 2004;43(Suppl):113–25. doi:10.1080/16501960410023877.
- Machamer J, Temkin N, Dikmen S, Nelson LD, Barber J, Hwang P, Boase K, Stein MB, Sun X, Giacino J, et al. Symptom frequency and persistence in the first year after traumatic brain injury: a TRACK-TBI study. *J Neurotrauma*. 2022;39(5–6):358–70. doi:10.1089/neu.2021.0348.
- McMahon P, Hricik A, Yue JK, Puccio AM, Inoue T, Lingsma HF, Beers SR, Gordon WA, Valadka AB, Manley GT, et al. Symptomatology and functional outcome in mild traumatic brain injury: results from the prospective TRACK-TBI study. *J Neurotrauma*. 2014;31(1):26–33. doi:10.1089/neu.2013.2984.
- King NS, Crawford S, Wenden FJ, Moss NE, Wade DT. The rivermead post concussion symptoms questionnaire: a measure of symptoms commonly experienced after head injury and its reliability. *J Neurol*. 1995;242(9):587–92. doi:10.1007/BF00868811.
- Emanuelson I, Andersson Holmkvist E, Bjorklund R, Stalhammar D. Quality of life and post-concussion symptoms in adults after mild traumatic brain injury: a population-based study in western Sweden. *Acta Neurol Scand*. 2003;108(5):332–38. doi:10.1034/j.1600-0404.2003.00155.x.
- Losoi H, Silverberg ND, Wäljas M, Turunen S, Rosti-Otajarvi E, Helminen M, Luoto TM, Julkunen J, Ohman J, Iverson GL, et al. Recovery from mild traumatic brain injury in previously healthy adults. *J Neurotrauma*. 2016;33(8):766–76. doi:10.1089/neu.2015.4070.
- Kraus JF, Nourjah P. The epidemiology of mild, uncomplicated brain injury. *J Trauma*. 1988;28(12):1637–43. doi:10.1097/00005373-198812000-00004.
- Binder LM, Rohling ML, Larrabee GJ. A review of mild head trauma. Part I: meta-analytic review of neuropsychological studies. *J Clin Exp Neuropsychol*. 1997;19(3):421–31. doi:10.1080/01688639708403870.
- Oldenburg C, Lundin A, Edman G, Nygren-de Boussard C, Bartfai A. Cognitive reserve and persistent post-concussion symptoms-A prospective mild traumatic brain injury (mTBI) cohort study. *Brain Inj*. 2016;30(2):146–55. doi:10.3109/02699052.2015.1089598.
- Anderson JFI, Jordan AS. Sex predicts post-concussion symptom reporting, independently of fatigue and subjective sleep disturbance, in premorbidly healthy adults after mild traumatic brain injury. *Neuropsychol Rehabil*. 2021;33(1):1–16. doi:10.1080/09602011.2021.1993274.
- Hromas GA, Houck ZM, Asken BM, Svingos AM, Greif SM, Heaton SC, et al. Making a difference: affective distress explains discrepancy between objective and subjective cognitive functioning after mild traumatic brain injury. *J Head Trauma Rehabil*. 2021;36(3):186–95. doi:10.1097/HTR.0000000000000618.
- Levin HS, Temkin NR, Barber J, Nelson LD, Robertson C, Brennan J, Stein MB, Yue JK, Giacino JT, McCrea MA, et al. Association of sex and age with mild traumatic brain injury-related symptoms: a TRACK-TBI Study. *JAMA Netw Open*. 2021;4(4):e213046. doi:10.1001/jamanetworkopen.2021.3046.
- Cassidy JD, Boyle E, Carroll LJ. Population-based, inception cohort study of the incidence, course, and prognosis of mild traumatic brain injury after motor vehicle collisions. *Arch Phys Med Rehabil*. 2014;95(3 Suppl):S278–85. doi:10.1016/j.apmr.2013.08.295.
- Ponsford J, Nguyen S, Downing M, Bosch M, McKenzie JE, Turner S, Chau M, Mortimer D, Gruen R, Knott J, et al. Factors associated with persistent post-concussion symptoms following mild traumatic brain injury in adults. *J Rehabil Med*. 2019;51(1):32–39. doi:10.2340/16501977-2492.
- Stenberg J, Haberg AK, Follstad T, Olsen A, Iverson GL, Terry DP, Karlsen RH, Saksvik SB, Karaliute M, Ek JAN, et al. Cognitive reserve moderates cognitive outcome after mild traumatic brain injury. *Arch Phys Med Rehabil*. 2020;101(1):72–80. doi:10.1016/j.apmr.2019.08.477.
- Steward KA, Kennedy R, Novack TA, Crowe M, Marson DC, Triebel KL. The role of cognitive reserve in recovery from traumatic brain injury. *J Head Trauma Rehabil*. 2018;33(1):E18–E27. doi:10.1097/HTR.0000000000000325.
- Stern Y. What is cognitive reserve? Theory and research application of the reserve concept. *J Int Neuropsychol Soc*. 2002;8(3):448–60. doi:10.1017/S1355617702813248.
- Kim E, Lauterbach EC, Reeve A, Arciniegas DB, Coburn KL, Mendez MF, & Coffey, E. C. Neuropsychiatric complications of

- traumatic brain injury: a critical review of the literature (a report by the ANPA Committee on Research). *J Neuropsychiatry Clin Neurosci.* 2007;19(2):106–27. doi:10.1176/jnp.2007.19.2.106.
20. Rogers JM, Read CA. Psychiatric comorbidity following traumatic brain injury. *Brain Inj.* 2007;21(13–14):1321–33. doi:10.1080/02699050701765700.
 21. Anderson JFI, Fitzgerald P. Associations between coping style, illness perceptions and self-reported symptoms after mild traumatic brain injury in prospectively studied pre-morbidly healthy individuals. *Neuropsychol Rehabil.* 2020;30(6):1115–28. doi:10.1080/09602011.2018.1556706.
 22. Carroll LJ, Cassidy JD, Peloso PM, Borg J, von Holst H, Holm L, Paniak C, Pépin M. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med.* 2004;43:84–105. doi:10.1080/16501960410023859.
 23. Scheenen ME, Spikman JM, de Koning ME, van der Horn HJ, Roks G, Hageman G, van der Naalt J. Patients “At Risk” of suffering from persistent complaints after mild traumatic brain injury: the role of coping, mood disorders, and post-traumatic stress. *J Neurotrauma.* 2017;34(1):31–37. doi:10.1089/neu.2015.4381.
 24. Broshek DK, De Marco AP, Freeman JR. A review of post-concussion syndrome and psychological factors associated with concussion. *Brain Inj.* 2015;29(2):228–37. doi:10.3109/02699052.2014.974674.
 25. Silverberg ND, Iverson GL. Etiology of the post-concussion syndrome: physiogenesis and psychogenesis revisited. *NeuroRehabilitation.* 2011;29(4):317–29. doi:10.3233/NRE-2011-0708.
 26. Theadom A, Barker-Collo S, Jones K, Kahan M, Te Ao B, McPherson K, Starkey N, Feigin V, Feigin V, Theadom A, et al. Work limitations 4 years after mild traumatic brain injury: a cohort study. *Arch Phys Med Rehabil.* 2017;98(8):1560–66. doi:10.1016/j.apmr.2017.01.010.
 27. Voormolen DC, Polinder S, von Steinbuechel N, Vos PE, Cnossen MC, Haagsma JA. The association between post-concussion symptoms and health-related quality of life in patients with mild traumatic brain injury. *Injury.* 2019;50(5):1068–74. doi:10.1016/j.injury.2018.12.002.
 28. Yousefzadeh-Chabok S, Kapourchali FR, Ramezani S. Determinants of long-term health-related quality of life in adult patients with mild traumatic brain injury. *Eur J Trauma Emerg Surg.* 2021;47(3):839–46. doi:10.1007/s00068-019-01252-9.
 29. Levy AM, Saling MM, Anderson JFI. Frequency and extent of cognitive complaint following adult civilian mild traumatic brain injury: a systematic review and meta-analysis. *Brain Impair.* 2022;1–24. doi:10.1017/BrImp.2022.19.
 30. Ngwenya LB, Gardner RC, Yue JK, Burke JF, Ferguson AR, Huang MC, Winkler EA, Pirracchio R, Sattris GG, Yuh EL, et al. Concordance of common data elements for assessment of subjective cognitive complaints after mild-traumatic brain injury: a TRACK-TBI pilot study. *Brain Inj.* 2018;32(9):1071–78. doi:10.1080/02699052.2018.1481527.
 31. Stulemeijer M, Vos PE, Bleijenberg G, van der Werf SP. Cognitive complaints after mild traumatic brain injury: things are not always what they seem. *J Psychosom Res.* 2007;63(6):637–45. doi:10.1016/j.jpsychores.2007.06.023.
 32. Anderson JFI. Cognitive complaint and objective cognition during the post-acute period after mild traumatic brain injury in pre-morbidly healthy adults. *Brain Injury.* 2021;35(1):103–13. doi:10.1080/02699052.2020.1859613.
 33. Levi Y, Rassovsky Y, Agranov E, Sela-Kaufman M, Vakil E. Cognitive reserve components as expressed in traumatic brain injury. *J Int Neuropsychol Soc.* 2013;19(6):664–71. doi:10.1017/S1355617713000192.
 34. Rassovsky Y, Levi Y, Agranov E, Sela-Kaufman M, Sverdlik A, Vakil E. Predicting long-term outcome following traumatic brain injury (TBI). *J Clin Exp Neuropsychol.* 2015;37(4):354–66. doi:10.1080/13803395.2015.1015498.
 35. Australian Bureau of Statistics. Australian and New Zealand standard classification of occupations. 2021.
 36. Wechsler D. The wechsler test of adult reading. New York: The Psychological Corporation; 2001.
 37. Steward KA, Novack TA, Kennedy R, Crowe M, Marson DC, Triebel KL. The wechsler test of adult reading as a measure of premorbid intelligence following traumatic brain injury. *Arch Clin Neuropsychol.* 2017;32(1):98–103. doi:10.1093/arclin/acw081.
 38. Rush AJ, Gullion CM, Basco MR, Jarrett RB, Trivedi MH. The Inventory of Depressive Symptomatology (IDS): psychometric properties. *Psychol Med.* 1996;26(3):477–86. doi:10.1017/S0033291700035558.
 39. Beck AT, Steer RA. Beck anxiety inventory manual. San Antonio, TX: Psychological Corporation; 1993.
 40. Weathers FW, Litz BT, Keane TM, Palmieri PA, Marx BP, Schnurr PP. The PTSD Checklist for DSM-5 (PCL-5). 2013.
 41. Tabachnick BG, Fidell LS. Using multivariate statistics. 6th ed. Boston: Allyn and Bacon; 2013.
 42. Anderson JFI, Jordan AS. An observational study of the association between sleep disturbance, fatigue and cognition in the post-acute period after mild traumatic brain injury in prospectively studied premorbidly healthy adults. *Neuropsychol Rehabil.* 2020;31(9):1–22. doi:10.1080/09602011.2020.1781665.
 43. Australian Bureau of Statistics. Employment in the 2021 Census: analysis of the Australian workforce, key industries, and occupations from the 2021 Census data. ABS website 2022.
 44. Bright P, van der Linde I. Comparison of methods for estimating premorbid intelligence. *Neuropsychol Rehabil.* 2020;30(1):1–14. doi:10.1080/09602011.2018.1445650.
 45. Cassidy JD, Cancelliere C, Carroll LJ, Cote P, Hincapie CA, Holm LW, Hartvigsen J, Donovan J, Nygren-de Boussard C, Kristman VL, et al. Systematic review of self-reported prognosis in adults after mild traumatic brain injury: results of the international collaboration on mild traumatic brain injury prognosis. *Arch Phys Med Rehabil.* 2014;95(3):S132–51. doi:10.1016/j.apmr.2013.08.299.
 46. Dean PJ, O'Neill D, Sterr A. Post-concussion syndrome: prevalence after mild traumatic brain injury in comparison with a sample without head injury. *Brain Inj.* 2012;26(1):14–26. doi:10.3109/02699052.2011.635354.
 47. Meares S, Shores EA, Taylor AJ, Batchelor J, Bryant RA, Baguley IJ, Chapman J, Gurka J, Dawson K, Capon L, et al. Mild traumatic brain injury does not predict acute postconcussion syndrome. *J Neurol Neurosurg Psychiatry.* 2008;79(3):300–06. doi:10.1136/jnnp.2007.126565.
 48. Prince C, Bruhns ME. Evaluation and treatment of mild traumatic brain injury: the role of neuropsychology. *Brain Sci.* 2017;7(12):105. doi:10.3390/brainsci7080105.
 49. Baker M. Is there a reproducibility crisis? *Nature.* 2016;533(7604):452–54. doi:10.1038/533452a.
 50. Open Science Collaboration. Psychologyestimating the reproducibility of psychological science. *Science.* 2015;349(6251):aac4716. doi:10.1126/science.aac4716.
 51. Cassidy JD, Carroll LJ, Peloso PM, Borg J, von Holst H, Holm L, Kraus J, Coronado V. Incidence, risk factors and prevention of mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med.* 2004;2004:28–60. doi:10.1080/16501960410023732.
 52. Dams-O'Connor K, Spielman L, Singh A, Gordon WA, Lingsma HF, Maas AIR, Manley GT, Mukherjee P, Okonkwo DO, Puccio AM, et al. The impact of previous traumatic brain injury on health and functioning: a TRACK-TBI study. *J Neurotrauma.* 2013;30(24):2014–20. doi:10.1089/neu.2013.3049.