Osborn, A.J.; Mathias, J.L.; Fairweather-Schmidt, A.K.
Depression following adult, non-penetrating traumatic brain injury: A meta-analysis examining methodological variables and sample characteristics, *Neuroscience and Biobehavioral Reviews*, 2014; 47:1-15

Originally Published at:
http://dx.doi.org/10.1016/j.neubiorev.2014.07.007

Neuroscience and Biobehavioral Reviews 47 (2014) 1–15

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

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

Review

Depression following adult, non-penetrating traumatic brain injury: A meta-analysis examining methodological variables and sample characteristics

A.J. Osborn[a], J.L. Mathias[a,*], A.K. Fairweather-Schmidt[a,b]

[a] School of Psychology, University of Adelaide, Adelaide, South Australia, Australia
[b] School of Psychology, Flinders University, Bedford Park, South Australia, Australia

ARTICLE INFO

Article history:
Received 6 November 2013
Received in revised form 16 June 2014
Accepted 8 July 2014
Available online 16 July 2014

Keywords:
Traumatic brain injury
Major depression
Dysthymia
Prevalence
Meta-analysis
Adult
Self-report measures
Injury severity
Post-injury interval

ABSTRACT

Background: Depression is one of the most frequently reported psychological problems following TBI, however prevalence estimates vary widely. Methodological and sampling differences may explain some of this variability, but it is not known to what extent.
Methods: Data from 99 studies examining the prevalence of clinically diagnosed depression (MDD/dysthymia) and self-reports of depression (clinically significant cases or depression scale scores) following adult, non-penetrating TBI were analysed, taking into consideration diagnostic criteria, measure, post-injury interval, and injury severity.
Results: Overall, 27% of people were diagnosed with MDD/dysthymia following TBI and 38% reported clinically significant levels of depression when assessed with self-report scales. Estimates of MDD/dysthymia varied according to diagnostic criteria (ICD-10: 14%; DSM-IV: 25%; DSM-III: 47%) and injury severity (mild: 16%; severe: 30%). When self-report measures were used, the prevalence of clinically significant cases of depression differed between scales (HADS: 32%; CES-D: 48%) method of administration (phone: 26%; mail 46%), post-injury interval (range: 33–42%), and injury severity (mild: 64%; severe: 39%).
Conclusion: Depression is very common after TBI and has the potential to impact on recovery and quality of life. However, the diagnostic criteria, measure, time post-injury and injury severity, all impact on prevalence rates and must therefore be considered for benchmarking purposes.

Contents

* Corresponding author at: School of Psychology, University of Adelaide, Adelaide, South Australia 5005, Australia. Tel.: +61 8 8313 5266; fax: +61 8 8313 3770.
E-mail address: jane.mathias@adelaide.edu.au (J.L. Mathias).

http://dx.doi.org/10.1016/j.neubiorev.2014.07.007

1. Introduction

Traumatic brain injuries (TBIs) can cause a variety of changes in cognitive, physical and psychological functioning, which may impact on all areas of a person's life. Cognitive changes include problems with memory and attention, poorer executive functioning and slowed information processing (Bay et al., 2012; Belmont et al., 2009; Konrad et al., 2011; Rochat et al., 2009), with the physical consequences including headaches, sleep problems and fatigue (Cantor et al., 2012; Chaput et al., 2009; Mathias and Alvaro, 2012). Psychological problems are also very common following TBI (for a review see Kim et al., 2007), with the most widely recognised and researched of these being depression (Hart et al., 2012; Rapoport, 2012).

The prevailing biopsychosocial model of health provides one framework for understanding some of the variables that may contribute to the development of these problems – including depression – following TBI (Helmchen, 2013). Specifically, this model posits that illnesses are caused by a complex interaction between a range of biological, psychological and social factors, with a person's vulnerability to illness changing over time (Molina, 1983). In the context of TBI, there are a variety of neuroanatomical changes that may provide a biological basis for the development of depression. For example, the shear, tensile and compressive strains experienced during a TBI can lead to diffuse axonal injury in the frontal and temporal lobes, disrupting the neural circuitry between the prefrontal cortex, amygdala, hippocampus, basal ganglia and thalamus (Jorge and Robinson, 2002; Kumar and Cook, 2002; Morris, 2010; Silver et al., 2009). This neuronal damage and cell loss can occur for weeks to months after an injury, and may provide the neurological substrate for many of the cognitive and psychological changes that occur after a TBI (Jorge and Starkstein, 2005; Sherin and Nemeroff, 2011). Neurochemical changes, such as cholinergic and serotonergic deficits, neuroendocrinal abnormalities and compromised hypothalamic–pituitary–adrenal axis function, also occur in the acute period post-TBI; potentially also causing depression (Jorge and Starkstein, 2005; Rosenthal et al., 1998). Psychological variables – such as a diminished tolerance to frustration, impaired self-awareness, low self-esteem, and poor coping strategies – may additionally lead to depression after a TBI (Kelley et al., 2012; Malec et al., 2007; Molina, 1983). Lastly, a variety of social factors – including a lack of social support, the loss of personal relationships/friendships, unrealistic expectations and involvement in litigation – may independently contribute to the development of depression following TBI or exacerbate symptoms that arise from any of the aforementioned causes (Dikmen et al., 2003; Gunstad and Suhr, 2001; Iverson et al., 2010b; Waljas et al., 2014). Thus, there are a large number of variables that may explain why depression is a common problem after TBI.

Estimates of the prevalence of depression following TBI vary considerably – ranging from 6% to 77% (Rutherford et al., 1977; Varney et al., 1987). This variability not only seriously limits the clinical utility of these findings, but also raises questions about its source. Differences in how depression is conceptualised (diagnosed disorder vs self-reported symptoms), the diagnostic criteria and/or measures that are used to assess depression and a number of patient characteristics (e.g., injury severity), may explain a significant amount of this variance; however, we do not currently know to what extent these variables impact on estimates of the prevalence of depression following TBI.

The two most commonly diagnosed depressive disorders following TBI are major depressive disorder (MDD) and dysthymia (Gomez-Hernandez et al., 1997; Hibbard et al., 1998; Meares et al., 2011; Whelan-Goodinson et al., 2009a), which are generally determined using one of two criteria, namely the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, DSM-IV, DSM-5; American Psychiatric Association [APA], 1987, 2001, 2013) or the International Classification of Diseases (ICD-9, ICD-10; World Health Organisation, 1977, 1992). These disorders have overlapping symptoms, including depressed mood, disturbed sleep, low energy and poor concentration. Whereas a diagnosis of MDD requires the presence of five or more symptoms during a 2-week period, dysthymia (also known as persistent depressive disorder) requires the presence of two symptoms for a minimum of 2 years (APA, 2013).

MDD and dysthymia are frequently diagnosed using one of a number of structured clinical interviews to determine whether their patients meet DSM or ICD criteria (e.g., Structured Clinical Interview for DSM-IV Axis I Disorders [SCID-I]; First et al., 1997). However, these interviews examine symptoms over different time periods – ranging from the previous week (e.g., the Clinical Interview Schedule [CIS]; Lewis et al., 1992) to the previous month (e.g., SCID, Schedules for Clinical Assessment in Neuropsychiatry [SCAN]; Wing et al., 1990) or preceding 6 months/year (e.g., Composite International Diagnostic Interview [CIDI]; Robins et al., 1988); which may have a significant impact on the resulting prevalence rates. Not only can the symptoms vary between these time frames, but also memory and other cognitive problems following TBI may affect the accuracy of the information that is reported (Hilsabeck et al., 1998). Therefore, differences in the diagnostic criteria and/or interview schedules that are used may be impacting on estimates of the prevalence of MDD and dysthymia.

Prevalence rates may additionally be affected by a number of sample characteristics, including the time interval between the injury and when depression is assessed. Some studies have found that MDD is more prevalent in the early stages after an injury (Bombardier et al., 2010; Lin et al., 2010), possibly reflecting neuroanatomical abnormalities or the cascade of neurochemical changes that occur in the acute post-TBI period (Jorge et al., 1993a). Conversely, others have found MDD and/or dysthymia are more common in the long-term (Al-Adawi et al., 2007; Rao et al., 2010), which may be more indicative of the psychosocial challenges faced by individuals as they adjust to their altered life circumstances (e.g., lack of social support, reduced social functioning) (Jorge et al., 1993b). Similarly, the severity of an injury can range from minor to severe, with some studies examining mixed samples (mild, moderate and severe), others targeting specific injuries (e.g., mild or severe), and still others examining less common categories, such as minor (Van Der Horn et al., 2013) or complicated mild TBI (Bombardier et al., 2010; Fann et al., 2005; Juengst et al., 2013).

In addition, control groups are often also recruited to examine the base-rates of depression because depression is not unique to TBI, but the samples chosen for this purpose can vary. Typically, medical patients (Brown et al., 2004; Jorge et al., 2004), people from the general community (Belmont et al., 2009; Konrad et al., 2011; Ponsford and Ziino, 2003), or family and friends of the TBI group (Perlesz et al., 2000; Ponsford et al., 2003) are used for this purpose. Each one attempts to control for the potential impact of different

variables (e.g., illness-related stress) on the prevalence of depression and, therefore, the base-rates are likely to differ between these groups. Once again, it is not known whether or how the choice of control groups impacts on the conclusions that are drawn regarding post-TBI rates of depression when they are used for comparative purposes.

Also important is the distinction between clinical diagnoses of MDD/dysthymia and assessments that use self-report questionnaires (e.g., Beck Depression Inventory [BDI]; Beck et al., 1996) to assess depression on a continuous scale (minimal to severe); often with the additional ability to identify clinically-significant levels of depression ('cases') using designated cut-off scores (dichotomous scale). Self-report scales are frequently used in clinical settings to screen for depression and in research settings to examine the prevalence and severity of depression following TBI. However, many of these scales were not specifically designed for use with TBI groups or in medical settings; instead being intended for use with the general population (e.g., Center for Epidemiological Scale – Depression [CES-D]; Radloff, 1977) or psychiatric patients (e.g., Hamilton Depression Rating Scale [HAM-D]; Hamilton, 1960). Consequently, they may contain items that can be affected by the physical consequences of a TBI (e.g., poor sleep, fatigue), which may inflate the prevalence of depression.

Furthermore, the way that clinicians or researchers administer questionnaires may affect the depression scores obtained on self-report scales. Various administration methods have been used, with some participants completing them at the research site (Hudak et al., 2011; Konrad et al., 2011), in their own home (Bushnik et al., 2008; King and Kirwilliam, 2011), over the phone (Bombardier et al., 2010; Hart et al., 2011) or by using a combination of these methods (Hawthorne et al., 2009; Smith, 1992). Specifically, certain situations may elicit socially-desirable responses, provide fewer opportunities to reflect on and revise answers (e.g., telephone interviews), or be subject to other unidentified influences (e.g., mailed: when completed and whether completed alone/with others). These variables are known to have an impact on people's responses (for a review see Richman et al., 1999) and, consequently, should be considered in the current context.

Any one or more of these variables has the capacity to influence estimates of the prevalence of depression and may help to explain why the aforementioned statistics vary so widely. However, as yet, their impact has not been assessed. A systematic analysis of the prevalence of depression following TBI is needed to evaluate the impact of these variables and to assist clinicians in selecting the most appropriate benchmark(s) for their particular circumstance. The current study therefore meta-analysed existing research that has examined: (1) the prevalence of clinical diagnoses of MDD and dysthymia following TBI or (2) used self-report scales to assess the prevalence of clinically significant symptoms and/or the severity of depression. To this end, the impact of diagnostic criteria, interview schedule, post-injury interval and injury severity on the prevalence of MDD/dysthymia was evaluated, as was the type of control group. In addition, the impact of questionnaire, method of administration, post-injury interval, injury severity and type of control group on self-reported measures of depression was examined.

2. Methods

2.1. Literature search, inclusion and exclusion criteria

Comprehensive searches of the PsycINFO, Pubmed, Scopus, and ISI Web of Knowledge electronic databases, from January 1980 to June 2013, were undertaken to identify studies that examined depression following TBI using search terms that were tailored for each database (see Supplementary data: Table A). In addition, the reference lists of all studies that were included in the final analysis were examined to identify any other relevant research.

For a study to be included in the current meta-analysis, it had to meet the following criteria: (1) it examined depression following non-penetrating TBI; (2) participants were 18 years or older (where age range was not provided: mean age minus 1 SD $\geq$18); (3) it reported the prevalence of current MDD or dysthymia, which was formally diagnosed using DSM or ICD criteria, and/or 'cases' (clinically significant levels of depression), or depression scale scores from a common self-report depression scale (excludes general quality of life and mood-state measures, and study-specific or modified scales) (see Supplementary data: Table B for a list of eligible measures); (4) data were provided for a TBI sample (single-sample) or both a TBI and control group (independent samples); (5) the data (prevalence rates, cases or depression scores) were reported in a way that enabled the calculation of an effect size; (6) the data were published in a journal in English and contained original data (excludes reviews); and (7) the sample size was greater than 15 (excludes very small samples and case studies).

Studies were excluded if participants were drawn exclusively from very specific or at-risk TBI populations – such as war veterans, prison inmates, victims of large-scale trauma/terrorism, or psychiatric populations – as their exposure to other traumatic events/situations may have increased their vulnerability to depression, rendering them less comparable to the broader TBI population. In addition, control groups were excluded if the group was very specific (e.g., depressed controls), depression was not assessed, or different depression scales were administered to the TBI and control groups. Moreover, if a study examined the efficacy of some form of treatment, only the pre-treatment data were analysed.

The literature search initially identified 8,399 potentially relevant articles, 2,217 of which were duplicates. The titles and abstracts of the remaining 6,182 articles were screened by the first author (AJO) using the aforementioned inclusion and exclusion criteria, after which the full-text versions of 459 studies were retrieved for detailed screening. Re-application of the inclusion criteria to these papers reduced the number of eligible studies to 99 (see Supplementary data: Table C for an overview of the study review and selection process). In ambiguous cases, papers were independently assessed by AJO and JLM, and eligibility determined following discussion.

Data that are meta-analysed must be obtained from independent samples (Rosenthal, 1995); consequently all studies were checked to establish independence. Six samples were followed longitudinally (two articles each); the data from these articles were combined, resulting in six independent studies; further reducing the final number of eligible studies to 93. Moreover, the data for the control groups from five studies were unsuitable for present purposes: only the TBI data from these studies were extracted (Capizzano et al., 2010; Hawthorne et al., 2009; Reza et al., 2007; Schnabel and Kydd, 2012; Wood and Williams, 2008).

2.2. Data preparation

Some basic data preparation was needed in order to render it suitable for analysis. Specifically, where demographic details were reported for TBI subgroups that were not relevant to the current analyses (e.g., fatigued vs non-fatigued TBI patients), the data were combined. If median and range were reported (e.g., age), the mean and SD were estimated using the methods recommended by Hozo et al. (2005). In addition, where necessary, standard errors were transformed to standard deviations and descriptive data transformed to a common scale of measurement (e.g., time-since-injury: months).

The post-injury interval for studies varied widely – ranging from a few days to over 30 years – necessitating the classification of these intervals into four broad groups: the first included studies that examined mean post-injury intervals of <6 months (acute to post-acute period); the second included intervals of ≥6 months to <2 years (short term); the third spanned ≥2 years to <5 years (medium term); and the fourth ≥5 years (long-term). Unfortunately, very few studies reported separate prevalence rates for their mild, moderate and severe participants. Thus, the data from studies that examined mild-moderate and moderate–severe TBI samples were combined with those that assessed all three categories (mild, moderate and severe) for present purposes. Further, where studies assessed depression in a control group, the type of control was classified into one of three groups: 'medical controls' (spinal cord, orthopaedic or general trauma patients), 'general community', or 'significant other' (family/friends/caregivers).

2.3. Data collection and effect size calculation

Demographic and injury information (e.g., age, gender, time-since-injury, injury severity data), the method by which depression was assessed (e.g., clinical diagnosis of MDD/dysthymia or self-report measure), the criteria used to diagnose MDD/dysthymia (e.g., DSM-IV, ICD-10), the measure used (clinical diagnoses: SCID-I, SCAN, etc., self-report: BDI, Hospital Anxiety and Depression Scale [HADS], etc.), the method by which self-report scales were administered (research centre, phone, mail, or combination of methods), sample details (i.e., recruitment source, pre-injury history of mental health problems and TBIs, current medication use, type of control group [medical, community, significant other]), and statistical data necessary for the calculation of effect sizes were extracted from each study for analysis. This information was then entered into Comprehensive Meta-Analysis Software version 2 (CMA; ©2006, Biostat, Inc., Englewood, NJ, USA).

Three types of effect size were calculated in the current study. Firstly, proportions were used to summarise the prevalence of (1) clinically diagnosed cases of MDD and dysthymia, and (2) clinically significant levels of depression, based on self-report data ('cases'), in studies that used single (TBI) or independent (TBI and controls) samples designs. Weighted mean prevalence rates were calculated using sample size as the weighting variable. Secondly, odds ratios were calculated to measure any increase (OR >1) or decrease (OR <1) in the likelihood of experiencing depression following TBI for those studies that used self-report measures to identify clinically significant levels of depression (cases) in TBI and control groups. Thirdly, weighted standardised mean differences (Hedges *g*) were used to estimate the magnitude of the difference between the depression scale scores (means, SDs) of TBI and Control groups (independent samples study design). A positive Hedges *g* indicates that the TBI group reported higher levels of depression than the controls, with a small effect defined as ≥.2, a moderate effect as ≥.5, and a large effect as ≥.8 (Cohen, 1992). As a guide, a Hedges *g* of .5 (medium effect) indicates that the means of the two groups differ by half of a pooled standard deviation.

The current study used a conservative random-effects model to calculate effect sizes, which assumes that effect sizes can vary due to sampling error and differences in study design. Importantly, when a study reported multiple scores that were eligible to be included in the same analysis, a mean effect was calculated to ensure that each study only contributed one effect size to any given analysis (Lipsey and Wilson, 2001). Forest plots were generated to examine the effect size distributions and assist in identifying outliers (Boyles et al., 2011), and ninety-five percent confidence intervals (95% CIs) were calculated to provide the upper and lower bounds within which we can be 95% confident that the actual population prevalence rate for depression following TBI lies. In the case of Hedges *g*, 95% CIs do that do not include zero, indicate that there is a significant difference between the depression scores of the TBI and control groups.

One problem that meta-analyses face is that the research literature may be biased towards publishing studies that report significant findings (publication bias/file-drawer problem) and, consequently, the resultant analyses tend to exclude non-significant findings; thereby inflating the effect sizes (Rosenthal, 1979). Publication bias was assessed using Orwin's (1983) Fail-safe *N* statistic (N_{fs}), which estimates the number of unpublished studies that would be required to draw a finding into question. Orwin's formula requires three values to compute a N_{fs}: the number of studies contributing to a mean effect, the resulting weighted mean effect size, and an alternative mean effect size, below which a result would be considered inconsequential/of minor clinical significance. For current purposes, TBI prevalence rates of less than 7.5%, odds ratios of <1.0, and Hedges *g* values of <0.15 were deemed to be of minor clinical significance. These figures were chosen on the basis of (a) a population-based survey of the 12-month prevalence of depression in Australian adults (Australian Bureau of Statistics, 2008), (b) Hopkin's (2002) guidelines for a trivial effect when using odds ratios, and (c) Cohen's definition of a small standardised mean difference. The resulting N_{fs} indicates the number of unpublished studies, with non-significant findings, that would be required to render the current findings inconsequential. Therefore, the larger the N_{fs}, the more confidence we can have in a finding.

2.4. Statistical analyses

Consistent with recommendations made by the Meta-Analysis of Observational Studies in Epidemiology (MOOSE) group Stroup et al. (2000), the impact of a variety of methodological and sampling variables on findings were examined in order to address the fact that findings from different studies were heterogeneous. This approach is also suggested by Borenstein et al. (2009) who note that a random-effects model and subgroup analyses can be used to identify sources of variability in the data.

The overall prevalence of MDD and dysthymia was calculated on the basis of data extracted from studies that used a single-sample (TBI group) or independent samples (TBI + controls) design, after which the impact of a number of moderator variables on the prevalence of MDD and dysthymia was examined, namely the: diagnostic criteria (DSM-III, DSM-IV, ICD-10); clinical interview (SCID-I, SCAN, etc.); time post-injury (<6 months, ≥6 months to <2 years, ≥2 years to <5 years, ≥5 years); and injury severity (i.e., mild, mild–moderate–severe, severe). In addition, the prevalence of MDD and dysthymia, relative to controls, was examined using ORs; both overall and based on the type of control group (medical, community, significant other).

Next, studies that used self-report scales to identify clinically significant levels of depression were examined. The overall prevalence of depression (cases) was calculated using data from single and independent samples designs, and the following moderator variables examined: the self-report scale (BDI, HADS, etc.); the method of administration (research centre, phone, mail, or combination of methods); time post-injury; and injury severity. The prevalence of clinically significant levels of depression (cases), relative to controls, was examined using ORs – both overall and by type of control group. Finally, the depression scale scores of TBI and control groups (level/severity of depression) were compared using Hedges *g* – both overall and by type of control group.

Table 1
Summary demographic and injury characteristics for the studies ($N = 93$).

Variable	$N_{studies}$	$N_{participants}$	%	Mean	SD
Sample size	93	11,926		131	209
Age (years)	93	11,926		37.1	6.8
Gender (males)	93	8,176	68.6		
Time-since-injury (months)	92	11,898		33.7	51.7
Glasgow Coma Scale score (GCS)	32	4,037		9.7	2.2
Injury severity					
Mild	12	1,134	9.5		
Mild, moderate	8	912	7.6		
Mild, moderate, severe	46	6,779	56.8		
Moderate, severe	17	2,569	21.5		
Severe	8	415	3.8		
Details not specified	2	117	0.9		
Recruitment source					
Outpatients	84	10,815	90.7		
Inpatients	6	936	7.8		
Both	3	175	1.5		
Pre-injury history of depression or anxiety					
Participants with history included	38	5,784	48.5		
Participants with history excluded	15	1,881	15.8		
Not specified	40	4,261	35.7		
Pre-injury history of TBI					
History of prior TBI	12	1,146	9.6		
No history of prior TBI	21	2,051	17.2		
Not specified	60	8,729	73.2		
Treatments					
Depression/anxiety medication	11	1,180	9.9		
Anti-epileptic medication	2	138	1.2		
Medication or counselling	1	100	0.8		
Participants excluded if using medication	2	73	0.6		
Medications used, no further detail	2	152	1.3		
Not specified	75	10,283	86.2		

	$N_{Studies}$	N_{TBI}	%	$N_{Control}$	%
Type of control group					
Medical	11	1,077	42.5	1,067	44.6
General community	13	809	31.9	691	28.9
Significant others	7	647	25.5	633	26.5
Total	31	2,533		2,391	

Note: $N_{studies}$ and $N_{participants}$ refer to the total number of studies and participants for which data were available. One study used two different control groups: community and medical (Clarke et al., 2012).

3. Results

3.1. Participant details

The 93 studies included in this meta-analysis provided data for a total of 11,926 participants. The background demographic and injury data for these studies are summarised in Table 1, where it can be seen that the majority of participants were young to middle-aged males.

Most studies reported the mean time between injury and assessment ($N_{studies} = 92$), with the average interval being just under 3 years (see Table 1). In contrast, only a limited number of studies reported mean Glasgow Coma Scale scores (GCS) ($N_{studies} = 32$), although most provided categorical information relating to injury severity. The majority of studies examined mixed samples of mild, moderate and severe TBI ($N_{studies} = 46$), however most did not report separate outcomes for these sub-groups.

Participants were largely recruited from outpatient settings ($N_{studies} = 84$; see Table 1). Six studies recruited from inpatient settings, all of which examined depression 1–6 months after severe TBI. Thirty-eight studies included participants who had a pre-injury diagnosis of depression or anxiety (779 out of 5,784 participants), 15 excluded participants on this basis, and 40 did not specify. Although the majority of studies failed to report whether participants had previously sustained a TBI ($N_{studies} = 60$) or excluded participants with such a history ($N_{studies} = 21$), 12 studies reported that 167 of their 1,146 participants had previously sustained a TBI. Similarly, most studies ($N_{studies} = 75$) failed to report medication use, however 11 reported that 314 (out of 1,180) participants were taking medications for depression or anxiety. Finally, 30 studies recruited one or more control groups (see Table 1), with the majority using medical ($N_{studies} = 11$; primarily general trauma) or community ($N_{studies} = 13$) controls, and a further seven recruiting significant others (family, friends, caregivers of the TBI group).

3.2. Prevalence of formally diagnosed depression following TBI

The data from all studies that reported the prevalence of MDD and/or dysthymia following TBI ($N_{studies} = 31$, $N_{participants} = 5{,}678$) were combined in order to calculate an overall prevalence rate. Fig. 1a

provides a forest plot of the prevalence rates for each of the individual studies (rank-ordered by size), together with the overall weighted mean, which indicates that, on average, 27% were diagnosed with MDD or dysthymia after a TBI. The associated N_{fs} was very large ($N_{fs} = 81$), suggesting that publication bias is unlikely to be a problem. Importantly, there was substantial variation in the prevalence estimates of individual studies (range: 9–67%), highlighting the need to undertake additional analyses to examine some of the variables that may have contributed to this variability.

Studies were first partitioned according to the diagnostic criteria that were used and, as seen in Fig. 1b, most used DSM criteria.

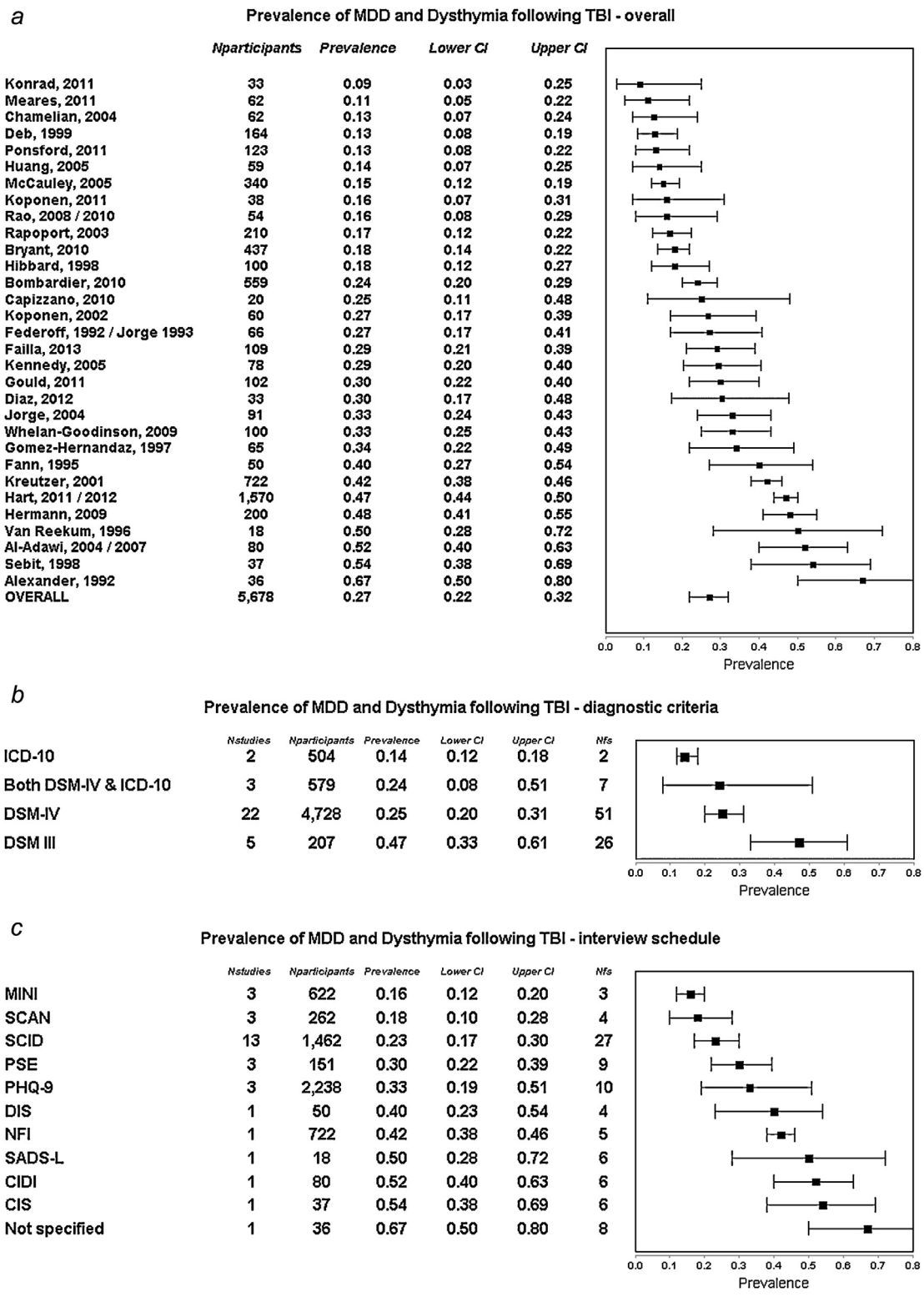


Fig. 1. Prevalence of formally diagnosed MDD and dysthymia: (a) overall, (b) diagnostic criteria, (c) interview schedule, (d) time post-injury, (e) injury severity, (f) overall, relative to controls, and (g) according to the type of control group. *Note*: MDD, major depressive disorder; CI, confidence interval; N_{fs}, fail-safe *N*; ICD, International Classification of Diseases; DSM, Diagnostic and Statistical Manual; MINI, Mini-International Neuropsychiatric Interview; SCAN, Schedules Clinical Assessment in Neuropsychiatry; SCID, Structured Clinical Interview; PSE, Present State Examination; PHQ-9, Patient Health Questionnaire-9; CIDI, Composite International Diagnostic Interview; SADS-L, Schedule for Affective Disorders and Schizophrenia (lifetime); DIS, Diagnostic Interview Schedule; NFI, Neurobehavioral Functioning Inventory; CIS, Clinical Interview Schedule. Refer online Supplementary data: Table D for details of studies contributing to the summary analyses in this figure; Kreutzer et al., 2001 used the NFI to identify and quantify depressive symptoms of MDD as specified in the DSM-IV.

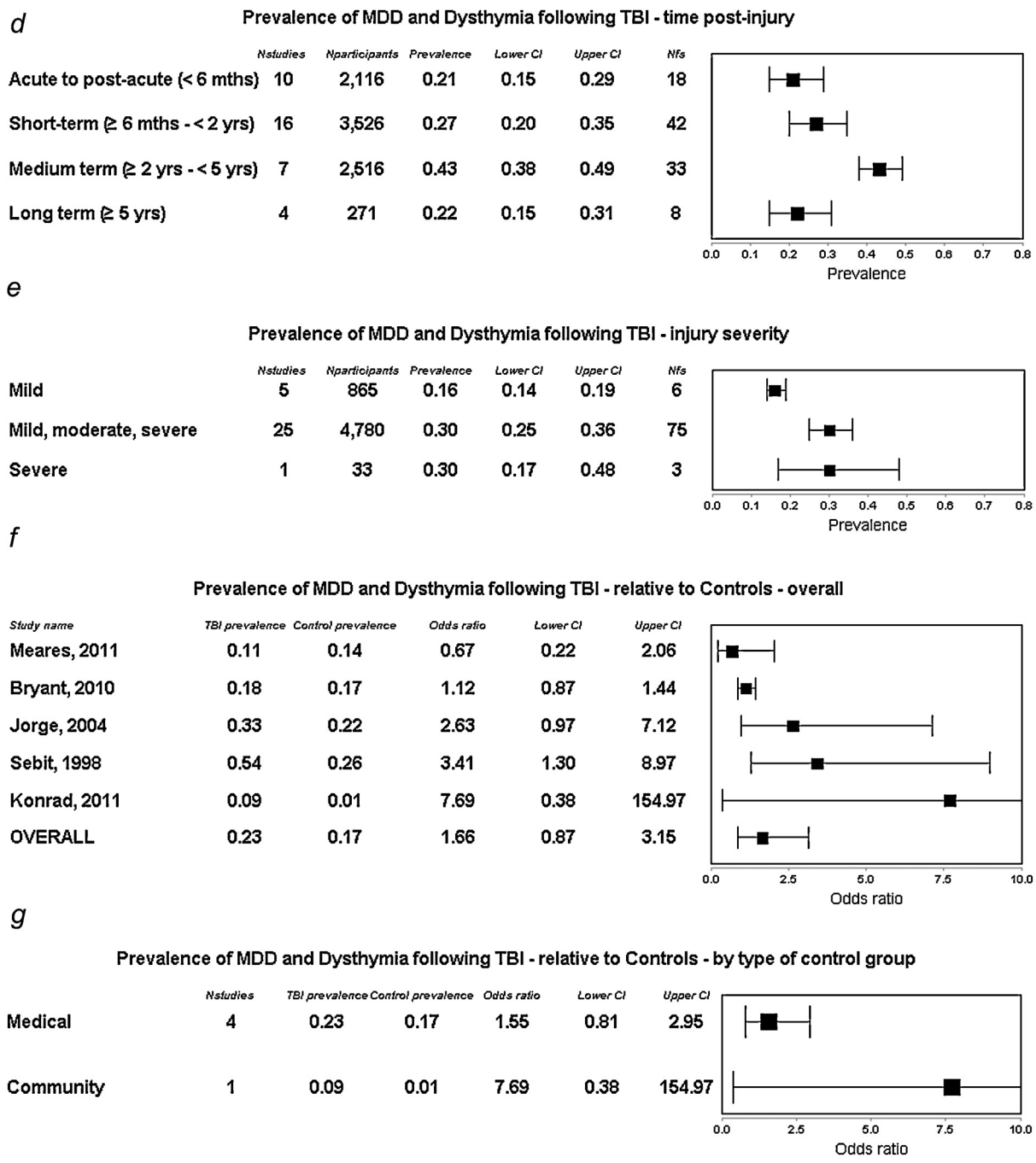


Fig. 1. (*Continued*)

The lowest prevalence rate (14%) was obtained using ICD-10 criteria and the highest (47%) using DSM-III criteria. Moreover, the CIs for ICD-10, DSM-IV and DSM-III prevalence rates did not overlap, indicating that they yielded significantly different rates.

As seen in Fig. 1c, a total of 10 different interview schedules were used to diagnose MDD/dysthymia, with the SCID-I ($N_{studies}$ = 13) being the most commonly used, followed by the SCAN, Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998), Present State Examination (PSE; Wing et al., 1974) and Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001), which were each used by three studies. The prevalence rates obtained using these measures varied between 16% and 33%; although, with the exception of the MINI and PSE, these differences were not significant. In contrast, the interviews used by single studies (e.g., CIDI, CIS, clinical diagnosis based on the Neurobehavioral Functioning Inventory [NFI]; Kreutzer et al., 1999) yielded significantly higher prevalence rates (range: 42–54%) than the more commonly used measures (i.e., SCID-I, SCAN and MINI).

Next, post-injury interval was examined to determine whether this impacted on prevalence rates (see Fig. 1d). The mean time intervals for the acute/post-acute, short, medium and long-term studies were 2.4 months (SD = 1.4), 11.5 months (SD = 5), 3.1 years (SD = 0.8) and 10.5 years (SD = 6.5), respectively. Notably, the mean prevalence of MDD/dysthymia appears to increase in the first 5 years after a TBI (21–43%), after which it declines to acute/post-acute levels (22%). Moreover, the medium-term prevalence rate was significantly higher than any other period.

Although injury severity may impact on the prevalence of depression after a TBI, only a coarse-grained analysis of this variable was possible because many studies used mixed samples (e.g., mild, moderate and severe) and only provided data for the whole sample. As seen in Fig. 1e, mild TBI was associated with a significantly lower prevalence of MDD and dysthymia (16%), compared with the mixed sample category of mild, moderate and severe TBI (30%). Severe TBI was only examined by one small-scale study with a wide CI that overlapped with the other categories, indicating that it did not differ from them in terms of the prevalence of MDD/dysthymia.

Finally, data from five studies that compared the prevalence of MDD/dysthymia in TBI and control groups were examined ($N_{participants}$: TBI = 600; controls = 712) (see Fig. 1f). Overall, there was a higher prevalence of MDD/dysthymia following TBI (23%) than in the controls (17%), with the associated OR indicating that a person is 1.66 times more likely than controls to develop MDD or dysthymia after a TBI. There was considerable variation in the ORs for individual studies (.67–7.69), some of which may have resulted from the choice of control groups. Of the five studies, four used medical controls and only one used community controls. Importantly, after sustaining a TBI, a person is nearly eight times more likely to develop MDD or dysthymia (OR = 7.69) than someone from the general community but only one and a half times more likely than medical controls (OR = 1.55) (see Fig. 1g).

3.3. Prevalence of clinically significant levels of depression ('cases') following TBI

When the data from the 57 studies that used self-report scales to identify clinically significant cases of depression following TBI were combined, it was found that the overall prevalence was 38% (refer to Fig. 2a). The associated N_{fs} statistic was very large (N_{fs} = 232), indicating that this is a very robust finding. As with diagnoses of MDD and dysthymia, there was considerable variability in the number of cases reported by individual studies (range: 2–74%), again highlighting the importance of examining some of the variables that may impact on these findings.

In terms of methodology, the specific self-report scale may impact on the prevalence of clinically significant cases of depression (see Fig. 2b). Indeed, there was considerable variability in the mean prevalence rates that were obtained using these different scales, ranging from 2% for the Montgomery–Asberg Depression Rating Scale [MADRS] (Montgomery and Asberg, 1979) to 48% for the CES-D. However, as is evident from the CIs, the prevalence rates reported by studies using the same measure were also highly variable (e.g., BDI-II; Zung Self-rating Depression Scale [ZSDS]; Zung, 1965), so much so that after excluding the MADRS, which was only used by one small study, none of the other measures differed significantly; although the HADS and CES-D approached significance. Similarly, when studies were grouped on the basis of how they administered the self-report scale – by phone, in person (research centre), by mail or using a combination of methods – there was substantial variability in the number of cases of depression reported by studies using the same method. Interestingly, although only the 'combination' and 'mailed' groups differed significantly, there was a trend towards fewer cases when questionnaires were administered by phone, compared to mailed questionnaires (see Fig. 2c).

Next, the prevalence of clinically significant cases of depression was found to steadily increase, albeit not significantly, as the post-injury interval increased (see Fig. 2d), with estimates ranging from 33% in the acute/post-acute period, 35% in the short-term, 41% in the medium term, and 42% in the longer term. Moreover, mild TBIs were associated with significantly more cases of depression (64%) than the mixed (mild/moderate/severe: 36%) and severe (39%) (see Fig. 2e) TBI samples.

Finally, the data from the 16 studies that used self-report measures to identify cases of depression in TBI and control samples were examined ($N_{participants}$: TBI = 1,055; controls = 1,000) (see Fig. 2f). The overall mean prevalence of depression following TBI (44%) was substantially higher than that of control groups (19%), with the associated OR of 3.41 indicating that a person is nearly three and a half times more likely to report clinically significant depression after a TBI, compared to controls. Once again, the ORs for individual studies varied substantially (range: 1–49), raising the possibility that the type of control group impacted on these findings. As seen in Fig. 2g, controls from the general community reported the lowest rates of depression (9%), followed by significant others (23%) and medical controls (36%). This was reflected in the ORs, which indicated that, following a TBI, people are nearly six times more likely than those in the general community, three times more likely than their significant others (family, friends), and over twice as likely as those with other medical conditions to experience clinically significant levels of depression. Nevertheless, all CIs overlapped, indicating the aforementioned differences were not significant.

3.4. Self-reported levels of depression: TBI vs controls

Twenty studies provided mean depression scale scores (continuous data) for TBI and Control groups, which were examined ($N_{participants}$: TBI = 1,563; controls = 4,017) using Hedges g (weighted standardised mean difference). Overall, there was a moderate and significant difference in the depression scores of the TBI and control groups (Hedges g = 0.63), together with a large N_{fs} statistic (N_{fs} = 43) (see Fig. 3a). When these studies were grouped according to type of control group (medical/community/significant other), there was a large and significant difference between the depression scores of the TBI and community controls (see Fig. 3b). Medical and 'significant other' controls also had significantly lower scores than the TBI group, but these differences equated to small to low-moderate effects.

4. Discussion

Estimates of the prevalence of depression following TBI vary widely, limiting the clinical utility of this research. The present study analysed the data from research that has examined the prevalence of MDD/dysthymia or used self-report scales to assess the severity of depression following TBI. A variety of methodological (diagnostic criteria, interview schedule/self-report scale, method of administering self-report scales, control group) and patient (time post-injury, injury severity) variables were examined to determine whether, and to what extent, they impacted on the available findings.

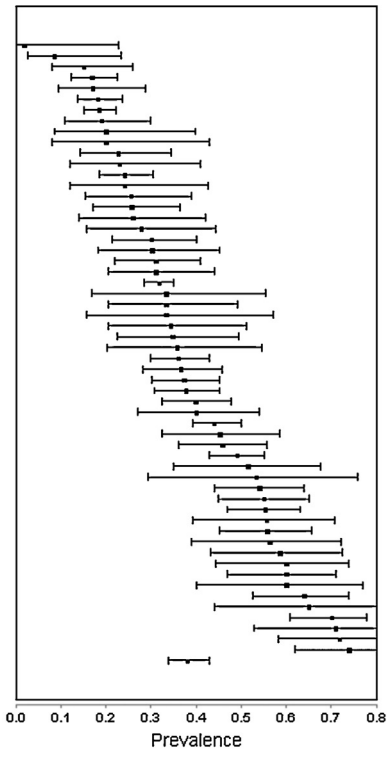
Overall, the findings indicate that depression is extremely common after a TBI, with 27% of people receiving a formal diagnosis of MDD or dysthymia and 38% reporting clinically significant symptoms on self-report scales. The lower prevalence of clinical diagnoses is not surprising because, in addition to using more stringent criteria, they provide a detailed assessment of the aetiology and chronology of symptoms, and greater opportunities for clarification (APA, 2000). In contrast, self-report scales measure the presence and severity of symptoms, applying a threshold to identify clinically significant cases; and do not provide an opportunity to clarify whether symptoms are the result of pre-existing (e.g., prior psychiatric history) or co-morbid conditions (e.g., physical/cognitive consequences of a TBI), which may inflate the prevalence rates (Green et al., 2001; Schwarzbold et al., 2008).

Moreover, people report more symptoms when prompted with specific questions than when asked to freely recall them

a

Prevalence of self-reported 'cases' of depression following TBI - overall

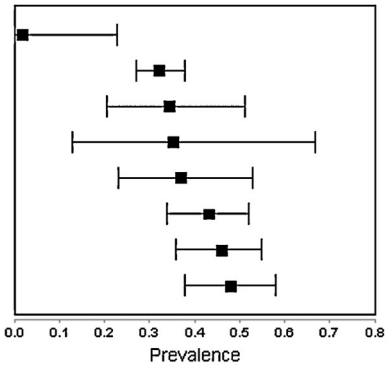
	Nparticipants	Prevalence	Lower CI	Upper CI
Belmont, 2009	27	0.02	0.00	0.23
Powell, 1998	35	0.03	0.03	0.22
Baumann, 2007 / Kempf, 2010	65	0.15	0.08	0.26
Skilbeck, 2013	214	0.17	0.12	0.22
Huang, 2005	59	0.17	0.09	0.29
van der Horn, 2013	242	0.18	0.14	0.24
Hudak, 2012	471	0.18	0.15	0.22
Al-Adawi, 2007	68	0.19	0.11	0.30
Hudak, 2011	25	0.20	0.09	0.40
Smith, 1992	19	0.20	0.08	0.43
Hawthorne, 2009	65	0.23	0.14	0.34
Gagnon, 2006	30	0.23	0.12	0.41
Chiu, 2006	199	0.24	0.19	0.31
Carroll, 2011	29	0.24	0.12	0.43
Rowland, 2005	51	0.25	0.15	0.39
Weddell, 2010	78	0.26	0.17	0.36
Bushnik, 2008	38	0.26	0.14	0.42
O'Carroll, 1991	36	0.28	0.16	0.44
Schnieders, 2012	90	0.30	0.21	0.40
Salmond, 2006	43	0.30	0.18	0.45
Forslund, 2013	91	0.31	0.22	0.41
Andelic, 2009	58	0.31	0.21	0.44
von Steinbuchel, 2010	795	0.32	0.29	0.35
Chalton, 2009	21	0.33	0.17	0.55
Kinsella, 1998	39	0.33	0.20	0.49
Williams, 1998	18	0.33	0.16	0.57
Goldstein, 2001	35	0.34	0.21	0.51
Powell, 2002	46	0.35	0.23	0.49
Ponsford, 2003	28	0.36	0.20	0.55
Dikmen, 2004	283	0.36	0.30	0.43
Senathi-Raja, 2010	112	0.37	0.28	0.46
Lin, 2010	158	0.37	0.30	0.45
Seel, 2003	172	0.38	0.31	0.45
Malec, 2010	158	0.40	0.33	0.48
Wallace, 2000	50	0.40	0.27	0.54
Ponsford, 2010	301	0.44	0.39	0.50
Draper, 2007	53	0.45	0.32	0.59
Bryant, 2001	96	0.46	0.36	0.56
Bush, 2004	253	0.49	0.43	0.55
Anson, 2006	33	0.52	0.35	0.68
Whalley Hammell, 1994	15	0.53	0.29	0.76
Evans, 2006	96	0.54	0.44	0.64
Wood, 2008	89	0.55	0.45	0.65
Ponsford, 2003a	143	0.55	0.47	0.63
de Almeida Lima, 2008	36	0.56	0.39	0.71
Curran, 2000	88	0.56	0.45	0.66
Alfano, 1993	32	0.56	0.39	0.72
Glenn, 2001	41	0.59	0.43	0.72
Chen, 2008	40	0.60	0.44	0.74
Perlesz, 2000	62	0.60	0.47	0.71
Tyerman, 1994	25	0.60	0.40	0.77
Bay, 2007	75	0.64	0.53	0.74
King, 2011	24	0.65	0.44	0.82
Englander, 2010	119	0.70	0.61	0.78
Reza, 2007	31	0.71	0.53	0.84
Alfano, 2006	53	0.72	0.58	0.82
Peleg, 2009	65	0.74	0.62	0.83
OVERALL	5,719	0.38	0.34	0.43



b

Prevalence of self-reported 'cases' of depression following TBI - self-report scale

	Nstudies	Nparticipants	Prevalence	Lower CI	Upper CI	Nfs
MADRS	1	27	0.02	0.00	0.23	0
HADS	19	2,247	0.32	0.27	0.38	62
GDS	1	35	0.34	0.21	0.51	6
ZSDS	3	168	0.35	0.13	0.67	11
BDI-II	9	973	0.37	0.23	0.53	35
BDI-I	11	879	0.43	0.34	0.52	52
Leeds	6	542	0.46	0.36	0.55	31
CES-D	8	1,149	0.48	0.38	0.58	43



c

Prevalence of self-reported 'cases' of depression following TBI - method of administration

	Nstudies	Nparticipants	Prevalence	Lower CI	Upper CI	Nfs
Phone	3	828	0.26	0.16	0.38	7
Combination	3	880	0.28	0.21	0.36	8
Research centre	43	3,558	0.39	0.34	0.44	181
Mailed	8	453	0.46	0.37	0.56	41

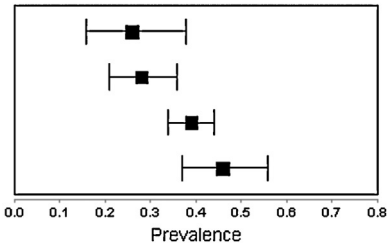
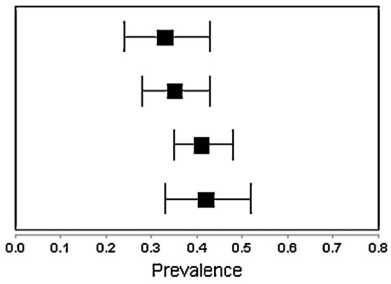


Fig. 2. Prevalence of clinically significant levels of depression identified using self-report scales: (a) overall, (b) self-report scale, (c) method of administration, (d) time post-injury, (e) injury severity, (f) overall, relative to controls, and (g) according to the type of control group. *Note*: CI, confidence interval; N_{fs}, fail-safe *N*; MADRS, Montgomery–Asberg Depression Rating Scale; HADS, Hospital Anxiety and Depression Scale; GDS, Geriatric Depression Scale; ZSDS, Zung Self-rating Depression Scale; BDI, Beck Depression Inventory; Leeds, The Leeds Scale for the Self-assessment of Anxiety and Depression; CES-D, Center for Epidemiological Scale – Depression. Refer online Supplementary data: Table D for details of studies contributing to the summary analyses in this figure.

d

Prevalence of self-reported 'cases' of depression following TBI - time post-injury

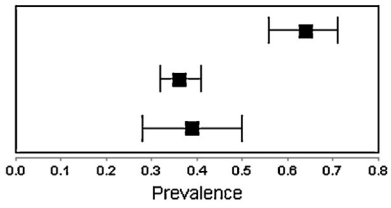
	Nstudies	Nparticipants	Prevalence	Lower CI	Upper CI	Nfs
Acute to post-acute (< 6 mths)	8	1,030	0.33	0.24	0.43	27
Short-term (≥ 6 mths - < 2 yrs)	25	2,422	0.35	0.28	0.43	92
Medium term (≥ 2 yrs - < 5 yrs)	19	2,225	0.41	0.35	0.48	85
Long term (≥ 5 yrs)	10	820	0.42	0.33	0.52	46



e

Prevalence of self-reported 'cases' of depression following TBI - injury severity

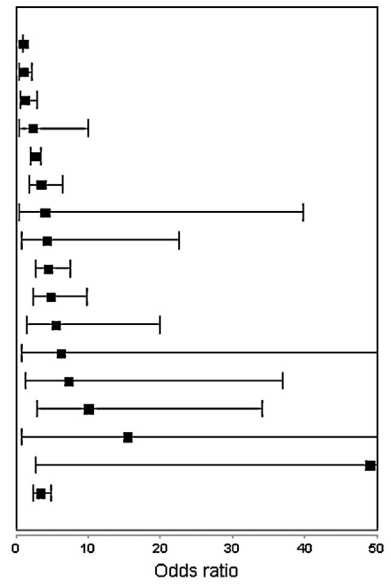
	Nstudies	Nparticipants	Prevalence	Lower CI	Upper CI	Nfs
Mild	4	153	0.64	0.56	0.71	30
Mild, moderate, severe	44	5,161	0.36	0.32	0.41	167
Severe	9	405	0.39	0.28	0.50	38



f

Prevalence of self-reported 'cases' of depression following TBI - relative to Controls - overall

Study name	TBI prevalence	Control prevalence	Odds ratio	Lower CI	Upper CI
Belmont, 2009	0.02	0.02	1.00	1.00	1.00
Curran, 2000	0.56	0.55	1.03	0.48	2.18
Wallace, 2000	0.40	0.34	1.29	0.57	2.92
Whalley Hammell, 1994	0.53	0.33	2.29	0.52	10.01
Ponsford, 2010	0.44	0.23	2.63	2.04	3.40
Perlesz, 2000	0.60	0.30	3.48	1.86	6.52
Smith, 1992	0.20	0.06	4.00	0.40	39.83
Gagnon, 2006	0.23	0.07	4.26	0.81	22.53
Ponsford, 2003a	0.55	0.22	4.46	2.66	7.48
Senathi-Raja, 2010	0.37	0.11	4.81	2.36	9.80
Alfano, 1993	0.56	0.19	5.46	1.50	19.93
O'Carroll, 1991	0.28	0.06	6.15	0.72	52.72
Ponsford, 2003	0.36	0.07	7.22	1.41	36.96
de Almeida Lima, 2008	0.56	0.11	10.00	2.92	34.21
Goldstein, 2001	0.34	0.03	15.43	0.85	280.76
Chen, 2008	0.60	0.03	49.00	2.75	874.56
OVERALL	0.44	0.19	3.41	2.40	4.84



g

Prevalence of self-reported 'cases' of depression following TBI - relative to Controls - by type of control group

	Nstudies	TBI prevalence	Control prevalence	Odds ratio	Lower CI	Upper CI
Medical	3	0.56	0.36	2.10	0.74	6.01
Community	6	0.35	0.09	5.75	3.21	10.30
Significant Other	7	0.46	0.23	3.25	2.20	4.81

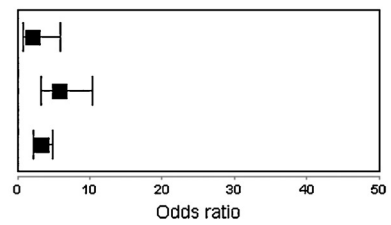


Fig. 2. (*Continued*)

(Iverson et al., 2010a), also leading to higher prevalence rates. The cognitive changes associated with TBIs (e.g., poorer memory, impaired insight) can, however, lead to fewer reports of depression (Wallace and Bogner, 2000); the impact of which may be offset by using questionnaires. Regardless of the method of assessment, individuals may exaggerate their symptoms if they are seeking financial compensation, highlighting the importance of assessing symptom validity when disingenuous performance may be an issue (Whiteside et al., 2012). Once compensation claims have been settled, the motivation to exaggerate symptoms is likely to reduce, suggesting that longer-term prevalence rates are less likely to be affected by this.

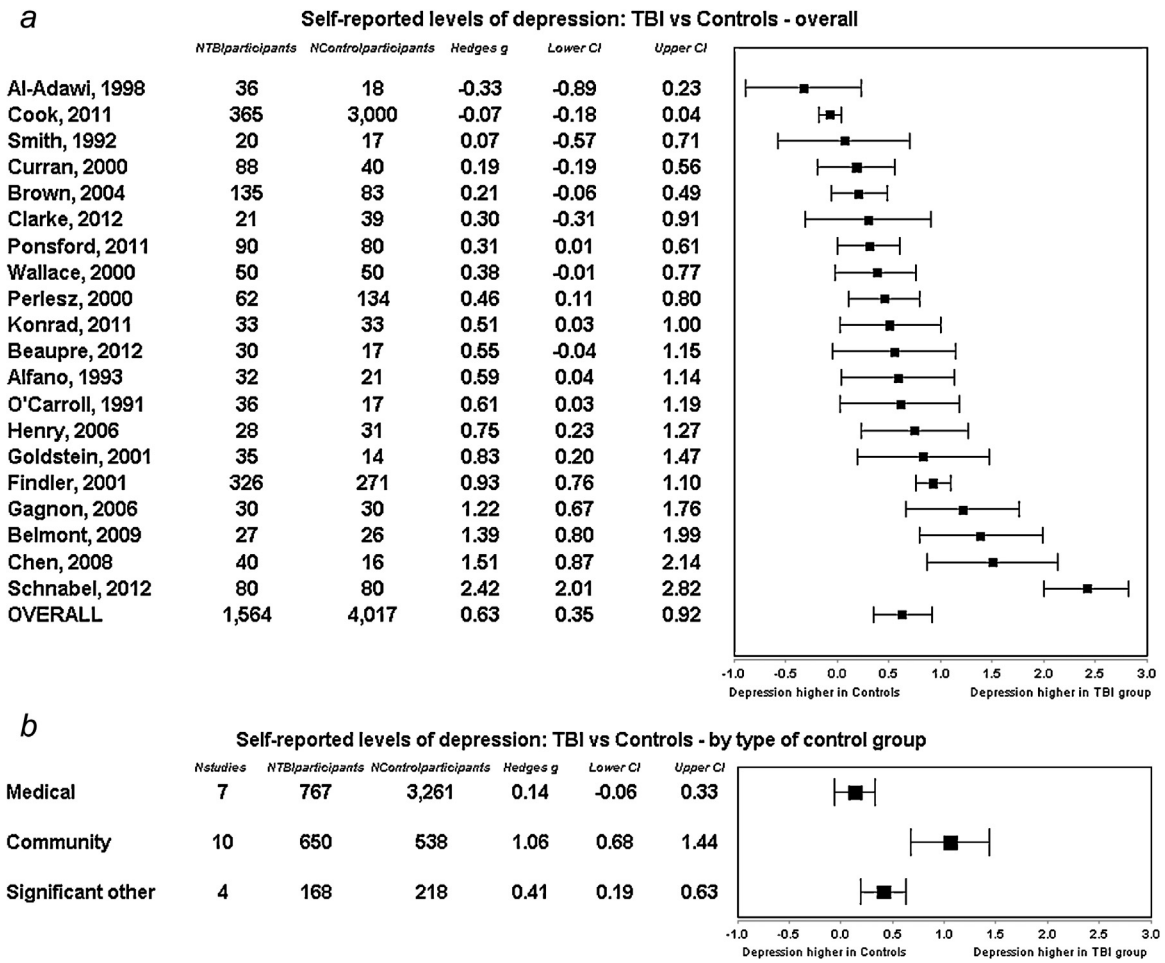


Fig. 3. Differences in the depression scores of TBI and Control groups, as assessed by self-report scales: (a) overall and (b) according to the type of control group. *Note*: CI, confidence interval.

Estimates of the prevalence of MDD and/or dysthymia differed when different diagnostic criteria were used, with the highest rates noted for the DSM-III (47%) and DSM-IV (25%) criteria, decreasing to 14% for the two studies that used ICD-10 criteria. These differences are surprising, given the overlap between these criteria and the fact that the DSM-IV revisions were relatively conservative (First, 2010). However, unlike the DSM, the ICD-10 categorises symptoms into two groups, each of which has a diagnostic threshold; potentially resulting in cases that meet one criterion, but not the other and leading to fewer diagnoses (First, 2009).

Prevalence rates were also affected by the specific interview that was used to diagnose MDD and dysthymia. The most commonly used schedule – the SCID-I – yielded a prevalence rate of 23% ($N_{studies}$ = 13), with the others ranging from 16% (MINI: $N_{studies}$ = 3) to 54% (CIS; $N_{studies}$ = 1). Some of this variability may result from the different time frames that are assessed. Indeed, the CIS and Schedule for Affective Disorders and Schizophrenia (SADS; Endicott and Spitzer, 1978), which focus on the preceding week, had the highest rates (54% and 50%, respectively). Longer time frames (previous 2 weeks to month) yielded substantially lower rates (e.g., MINI = 16%; SCAN = 18%; SCID-I = 23%). Other scales allow clinicians to select the time-frame (e.g., Diagnostic Interview Schedule; Robins et al., 1981), but this was often not reported.

There was also considerable variability in the prevalence of clinically significant cases of depression, identified on the basis of self-report scales; with estimates ranging from 2% for the MADRS ($N_{studies}$ = 1) to 48% for the CES-D ($N_{studies}$ = 8). Notably, the CES-D was designed for use in the general population and incorporates items that may be indicative of physical or cognitive TBI symptoms (e.g., sleep difficulties, fatigue, attentional problems), possibly inflating the number of 'cases'. Interestingly, the rate obtained from the most frequently used scale – the HADS ($N_{studies}$ = 19; 32%) – was substantially lower than the finding for the CES-D (48%). The HADS was specifically designed for use in medical settings and, consequently, does not include items that may reflect the physical, rather than psychological, consequences of a TBI (Zigmond and Snaith, 1983). This measure may therefore provide the best estimate of self-reported cases of depression.

The prevalence of self-reported 'cases' of depression also differed according to how the scale was administered. Specifically, more cases were identified when people completed questionnaires at home and returned them by mail (46%), than when completed by phone interview (26%). Phone administration may encourage people to respond in a socially-desirable manner, possibly causing them to down-play their symptoms and/or provide limited opportunity to reflect on and revise their answers (Aziz and Kenford, 2004; Fairweather-Schmidt and Anstey, 2012). However, at home, a person may be influenced by others, even when there are explicit instructions stating that all responses must be their own and/or no discussion with others is permitted (Alfano et al., 1993; O'Carroll et al., 1991).

In addition, prevalence rates varied according to the post-injury interval, with the prevalence of MDD/dysthymia steadily increasing in the first 5 years post-TBI (21–27–43%) and subsequently declining to a level similar to that seen in the early post-injury period (22%). In contrast, the number of 'cases' of depression identified using questionnaires steadily increased – although not significantly – from the acute/post-acute period until the medium-term (33–41%), when it plateaued. These findings highlight temporal changes to the risk of developing depression, possibly reflecting the changing influence of a number of different variables (e.g., neuronal/neurochemical, psychological, social). They also underscore the importance of monitoring individuals over an extended period of time and providing ongoing access to mental health support services.

Unfortunately, injury severity could only be examined in a basic way, due to the limited availability of data. These analyses revealed that mild TBI was associated with the lowest prevalence of MDD and dysthymia (16%); a rate that was significantly lower than that seen in mixed samples (30%). Although lower than the rate for severe TBI (30%), this difference was not significant, possibly due to the small sample size. These findings contrasted with those from self-report measures, which revealed significantly more cases of depression following mild TBI (64%) (mixed samples = 36%; severe TBI = 39%). There are a number of factors that may contribute to the latter finding. For example, severe TBIs are more frequently associated with memory problems and impaired self-awareness (Evans et al., 2005), which may reduce the number of symptoms that are endorsed on questionnaires (Malec et al., 2007). Alternatively, persons with mild TBI may be exaggerating their symptoms for financial gain (Kurtz et al., 2007). However, it is also possible that people do not receive adequate psycho-educational support following a mild TBI, which may increase their distress or, in the absence of significant physical injuries, they may focus on other problems (Malec et al., 2007). Therefore, even following mild TBI, individuals should be monitored to ensure that these symptoms do not interfere with their recovery or quality of life.

Relative to others, people are more likely to be diagnosed with MDD/dysthymia (OR = 1.66), or experience clinically significant levels of depression (OR = 3.41) following a TBI. Even when contributing factors, such as pain and hospital/medical procedures, are taken into account, a TBI provides an additional, unique source of psychological distress. Similarly, TBI groups were nearly eight times more likely to be diagnosed with MDD/dysthymia, and over five times more likely to be classified as having clinically significant levels of depression, than members of the general community. Lastly, the family/friends/caregivers of those who have sustained a TBI reported suffering from high levels of depression (23%), indicating that they are also at considerable risk of developing depression, and may require monitoring and treatment to optimise their outcomes (Ergh et al., 2002; Ponsford et al., 2010).

Finally, when the full spectrum of self-reported depressive symptoms was examined – ranging from normal to severe depression – it was found that individuals experienced moderately higher levels of depression following a TBI than their peers. This finding was impacted by the source of the controls, with the largest difference associated with people residing in the community, followed by significant others and then medical controls. This highlights the importance of selecting the appropriate norms or controls, based on the clinical or research question, to enable depression to be examined independently of a range of confounding variables. Specifically, medical controls endeavour to control for pain, other injuries and hospital routines/procedures (Ponsford et al., 2011); significant others control for the increased levels of stress and emotional distress related to a family member's TBI (Ponsford and Schönberger, 2010); and community controls enable an assessment of depression relative to people who are residing in the general community (Wacholder et al., 1992).

4.1. Limitations and recommendations for future research

There are a number of limitations that warrant consideration. First, although prior TBIs and psychiatric history may contribute to the development of depression (Anstey et al., 2004; Fann et al., 2002), these data were often not reported, or provided in a form that could not be compared (e.g., no major psychiatric illness, no prior hospitalisation, not currently using medication); precluding an analysis of these variables. Second, data were often combined across different injury categories (mild, moderate, severe), which meant that it was only possible to undertake a coarse-grained examination of TBI severity. Third, it was not possible to examine the impact of medications on the prevalence of depression because very few studies reported these data. Anti-depressant medications are likely to reduce symptoms and result in lower prevalence estimates, making this an important variable to consider. Fourth, it is possible that gender may have impacted on the prevalence of depression following TBI because females have a higher risk of developing depression (Kessler et al., 1993), although males are more likely to sustain a TBI (Anstey et al., 2004). Of the studies that reported the prevalence of MDD/dysthymia, only six provided gender-based data. While there was a trend for females to have higher rates of MDD/dysthymia in these studies (47% vs 34%), the difference was not significant.

It is recommended that researchers report participants' history of TBIs/psychiatric diagnoses and, ideally, provide subgroup data (mild/moderate/severe TBI; medicated vs unmedicated; males vs females), so that these variables can be examined in greater detail. Multivariate analyses of the data were not possible due to the variability in the research designs that have been used to examine the prevalence of depression following TBI. A large-scale study that evaluates the impact of these variables is now needed.

5. Conclusions

There is now a substantial body of research that has examined the prevalence of depression following TBI, but the estimates from individual studies vary widely. The challenges involved in interpreting these disparate findings are well-known, with researchers repeatedly noting that numerous methodological differences have made it difficult to compare findings and draw definitive conclusions (Koponen et al., 2011; Tsaousides et al., 2013; Whelan-Goodinson et al., 2009a).

Overall, the prevalence of formally diagnosed MDD and dysthymia was 27%, although this varied depending on whether ICD-10 (14%), DSM-IV (25%) or DSM-III (47%) diagnostic criteria were used. The different interview schedules also yielded variable prevalence rates, ranging from 16% to 54%. MDD/dysthymia was more prevalent between 2 and 5 years post-injury (43%), compared with the acute/post-acute period (<6 months; 21%), short-term (≥6 months to <2 years; 27%), and long-term (≥5 years; 22%). In addition, the prevalence of MDD/dysthymia was substantially higher following severe TBI (30%) than mild TBI (16%), although this difference was not significant. Moreover, MDD/dysthymia is more common following TBI than it is after other injuries (OR = 1.55) and in the general community (OR = 7.69).

The overall prevalence of clinically significant 'cases' of depression, assessed using questionnaires, was 38%; although this rate varied considerably depending on the measure that was used (2–48%) and the method of administration (phone: 26%; mail: 46%). Unlike MDD and dysthymia, self-reported depression continued to increase over time (from 33% to 42%) and injury severity had a paradoxical effect, with more cases of depression following mild

TBI (64%, severe TBI = 39%). The odds of developing depression after a TBI are more than five, three and two times higher than those living in the general community, the family and friends of the person who sustained the TBI, and other medical patients, respectively.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.neubiorev.2014.07.007.

References*

*Al-Adawi, S., Dorvlo, A.S., Al-Naamani, A., Glenn, M.B., Karamouz, N., Chae, H., Zaidan, Z.A., Burke, D.T., 2007. The ineffectiveness of the Hospital Anxiety and Depression Scale for diagnosis in an Omani traumatic brain injured population. Brain Inj. 21 (4), 385–393.

*Al-Adawi, S., Dorvlo, A.S.S., Burke, D.T., Huynh, C.C., Jacob, L., Knight, R., Shah, M.K., Al-Hussaini, A.A., 2004. Apathy and depression in cross-cultural survivors of traumatic brain injury. J. Neuropsychiatry Clin. Neurosci. 16 (4), 435–442.

*Al-Adawi, S., Powell, J.H., Greenwood, R.J., 1998. Motivational deficits after brain injury: a neuropsychological approach using new assessment techniques. Neuropsychology 12 (1), 115–124.

*Alexander, M.P., 1992. Neuropsychiatric correlates of persistent postconcussive syndrome. J. Head Trauma Rehabil. 7 (2), 60–69.

*Alfano, D.P., 2006. Emotional and pain-related factors in neuropsychological assessment following mild traumatic brain injury. Brain Cogn. 60 (2), 194–196.

*Alfano, D.P., Neilson, P.M., Fink, M.P., 1993. Long-term psychosocial adjustment following head or spinal cord injury. Neuropsychiatry Neuropsychol. Behav. Neurol. 6 (2), 117–125.

American Psychiatric Association, 1987. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. American Psychiatric Association, Washington, DC.

American Psychiatric Association, 2000. Handbook of Psychiatric Measures. American Psychiatric Publishing, Washington, DC.

American Psychiatric Association, 2001. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), 4th ed. American Psychiatric Publishing, Washington, DC.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Publishing, Arlington, VA.

*Andelic, N., Hammergren, N., Bautz-Holter, E., Sveen, U., Brunborg, C., Roe, C., 2009. Functional outcome and health-related quality of life 10 years after moderate-to-severe traumatic brain injury. Acta Neurol. Scand. 120 (1), 16–23.

*Anson, K., Ponsford, J., 2006. Coping and emotional adjustment following traumatic brain injury. J. Head Trauma Rehabil. 21 (3), 248–259.

Anstey, K.J., Butterworth, P., Jorm, A.F., Christensen, H., Rodgers, B., Windsor, T.D., 2004. A population survey found an association between self-reports of traumatic brain injury and increased psychiatric symptoms. J. Clin. Epidemiol. 57 (11), 1202–1209.

Australian Bureau of Statistics, 2008. 4326.0: National Health Survey: Summary of Results. ABS, Canberra.

Aziz, M., Kenford, S., 2004. Comparability of telephone and face-to-face interviews in assessing patients with posttraumatic stress disorder. J. Psychiatr. Pract. 10 (5), 307–313.

*Baumann, C.R., Werth, E., Stocker, R., Ludwig, S., Bassetti, C.L., 2007. Sleep–wake disturbances 6 months after traumatic brain injury: a prospective study. Brain 130 (7), 1873–1883.

*Bay, E., Hagerty, B.M., Williams, R.A., 2007. Depressive symptomatology after mild-to-moderate traumatic brain injury: a comparison of three measures. Arch. Psychiatr. Nurs. 21 (1), 2–11.

Bay, E., Kalpakjian, C., Giordani, B., 2012. Determinants of subjective memory complaints in community-dwelling adults with mild-to-moderate traumatic brain injury. Brain Inj. 26 (7/8), 941–949.

*Beaupre, M., De Guise, E., McKerral, M., 2012. The Association between pain-related variables, emotional factors, and attentional functioning following mild traumatic brain injury. Rehabil. Res. Pract. 2012, 1–10.

Beck, A.T., Steer, R.A., Brown, G.K., 1996. Manual for the Beck Depression Inventory. The Psychological Corporation, San Antonio, TX.

*Belmont, A., Agar, N., Azouvi, P., 2009. Subjective fatigue, mental effort, and attention deficits after severe traumatic brain injury. Neurorehabil. Neural Repair 23 (9), 939–944.

*Bombardier, C.H., Fann, J.R., Temkin, N.R., Esselman, P.C., Barber, J., Dikmen, S.S., 2010. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. J. Am. Med. Assoc. 303 (19), 1938–1945.

Borenstein, M., Hedges, L.V., Higgins, J.P.T., 2009. Introduction to Meta-analysis. Wiley, Hoboken, NJ, USA.

Boyles, A.L., Harris, S.F., Rooney, A.A., Thayer, K.A., 2011. Forest Plot Viewer: a new graphing tool. Epidemiology 22 (5), 746–747.

*Brown, S.A., McCauley, S.R., Levin, H.S., Contant, C., Boake, C., 2004. Perception of health and quality of life in minorities after mild-to-moderate traumatic brain injury. Appl. Neuropsychol. 11 (1), 54–64.

*Bryant, R.A., Marosszeky, J.E., Crooks, J., Baguley, I.J., Gurka, J.A., 2001. Posttraumatic stress disorder and psychosocial functioning after severe traumatic brain injury. J. Nerv. Ment. Dis. 189 (2), 109–113.

*Bryant, R.A., O'Donnell, M.L., Creamer, M., McFarlane, A.C., Clark, C.R., Silove, D., 2010. The psychiatric sequelae of traumatic injury. Am. J. Psychiatry 167 (3), 312–320.

*Bush, B.A., Novack, T.A., Schneider, J.J., Madan, A., 2004. Depression following traumatic brain injury: the validity of the CES-D as a brief screening device. J. Clin. Psychol. Med. Settings 11 (3), 195–201.

*Bushnik, T., Englander, J., Wright, J., 2008. The experience of fatigue in the first 2 years after moderate-to-severe traumatic brain injury: a preliminary report. J. Head Trauma Rehabil. 23 (1), 17–24.

Cantor, J.B., Bushnik, T.A., Cicerone, K., Dijkers, M.P., Gordon, W., Hammond, F.M., Kolakowsky-Hayner, S.A., Lequerica, A., Nguyen, M., Spielman, L.A., 2012. Insomnia, fatigue, and sleepiness in the first 2 years after traumatic brain injury: an NIDRR TBI model system module study. J. Head Trauma Rehabil. 27 (6), E1–E14.

*Capizzano, A.A., Jorge, R.E., Robinson, R.G., 2010. Limbic metabolic abnormalities in remote traumatic brain injury and correlation with psychiatric morbidity and social functioning. J. Neuropsychiatry Clin. Neurosci. 22 (4), 370–377.

*Carroll, E., Coetzer, R., 2011. Identity, grief and self-awareness after traumatic brain injury. Neuropsychol. Rehabil. 21 (3), 289–305.

*Chalton, L.D., McMillan, T.M., 2009. Can 'partial' PTSD explain differences in diagnosis of PTSD by questionnaire self-report and interview after head injury? Brain Inj. 23 (2), 77–82.

*Chamelian, L., Reis, M., Feinstein, A., 2004. Six-month recovery from mild to moderate traumatic brain injury: the role of APOE-ε4 allele. Brain 127 (12), 2621–2628.

Chaput, G., Giguère, J.-F., Chauny, J.-M., Denis, R., Lavigne, G., 2009. Relationship among subjective sleep complaints, headaches, and mood alterations following a mild traumatic brain injury. Sleep Med. 10 (7), 713–716.

*Chen, J.-K., Johnston, K.M., Petrides, M., Ptito, A., 2008. Neural substrates of symptoms of depression following concussion in male athletes with persisting postconcussion symptoms. Arch. Gen. Psychiatry 65 (1), 81–89.

*Chiu, W.T., Huang, S.J., Hwang, H.F., Tsauo, J.Y., Chen, C.F., Tsai, S.H., Lin, M.R., 2006. Use of the WHOQOL-BREF for evaluating persons with traumatic brain injury. J. Neurotrauma 23 (11), 1609–1620.

*Clarke, L.A., Genat, R.C., Anderson, J.F.I., 2012. Long-term cognitive complaint and post-concussive symptoms following mild traumatic brain injury: the role of cognitive and affective factors. Brain Inj. 26 (3), 298–307.

Cohen, J., 1992. A power primer. Psychol. Bull. 112 (1), 155–159.

*Cook, K.F., Bombardier, C.H., Bamer, A.M., Choi, S.W., Kroenke, K., Fann, J.R., 2011. Do somatic and cognitive symptoms of traumatic brain injury confound depression screening? Arch. Phys. Med. Rehabil. 92, 818–823.

*Curran, C.A., Ponsford, J., Crowe, S., 2000. Coping strategies and emotional outcome following traumatic brain injury: a comparison with orthopedic patients. J. Head Trauma Rehabil. 15 (6), 1256–1274.

*de Almeida Lima, D.P., Simao Filho, C., Abib Sde, C., de Figueiredo, L.F., 2008. Quality of life and neuropsychological changes in mild head trauma. Late analysis and correlation with S100B protein and cranial CT scan performed at hospital admission. Injury 39 (5), 604–611.

*Deb, S., Lyons, I., Koutzoukis, C., Ali, I., McCarthy, G., 1999. Rate of psychiatric illness 1 year after traumatic brain injury. Am. J. Psychiatry 156 (3), 374–378.

*Diaz, A.P., Schwarzbold, M.L., Thais, M.E., Hohl, A., Bertotti, M.M., Schmoeller, R., Nunes, J.C., Prediger, R., Linhares, M.N., Guarnieri, R., Walz, R., 2012. Psychiatric disorders and health-related quality of life after severe traumatic brain injury: a prospective study. J. Neurotrauma 29 (6), 1029–1037.

*Dikmen, S.S., Bombardier, C.H., MacHamer, J.E., Fann, J.R., Temkin, N.R., 2004. Natural history of depression in traumatic brain injury. Arch. Phys. Med. Rehabil. 85 (9), 1457–1464.

Dikmen, S.S., Machamer, J.E., Powell, J.M., Temkin, N.R., 2003. Outcome 3 to 5 years after moderate to severe traumatic brain injury. Arch. Phys. Med. Rehabil. 84 (10), 1449–1457.

*Draper, K., Ponsford, J., Schönberger, M., 2007. Psychosocial and emotional outcomes 10 years following traumatic brain injury. J. Head Trauma Rehabil. 22 (5), 278–287.

Endicott, J., Spitzer, R.L., 1978. A diagnostic interview: the schedule for affective disorders and schizophrenia. Arch. Gen. Psychiatry 35 (7), 837–844.

*Englander, J., Bushnik, T., Oggins, J., Katznelson, L., 2010. Fatigue after traumatic brain injury: association with neuroendocrine, sleep, depression and other factors. Brain Inj. 24 (12), 1379–1388.

Ergh, T.C., Rapport, L.J., Coleman, R.D., Hanks, R.A., 2002. Predictors of caregiver and family functioning following traumatic brain injury: social support moderates caregiver distress. J. Head Trauma Rehabil. 17 (2), 155–174.

*Evans, C.C., Sherer, M., Nick, T.G., Nakase-Richardson, R., Yablon, S.A., 2005. Early impaired self-awareness, depression, and subjective well-being following traumatic brain injury. J. Head Trauma Rehabil. 20 (6), 488–500.

*Failla, M.D., Burkhardt, J.N., Miller, M.A., Scanlon, J.M., Conley, Y.P., Ferrell, R.E., Wagner, A.K., 2013. Variants of SLC6A4 in depression risk following severe TBI. Brain Inj. 27 (6), 696–706.

Fairweather-Schmidt, A.K., Anstey, K.J., 2012. Prevalence of suicidal behaviours in two Australian general population surveys: methodological considerations when comparing across studies. Soc. Psychiatry Psychiatr. Epidemiol. 47, 515–522.

* References marked with an asterisk indicate studies included in the meta-analyses.

Fann, J.R., Leonetti, A., Jaffe, K., Katon, W.J., Cumming, P., Thompson, R.S., 2002. Psychiatric illness and subsequent traumatic brain injury: a case control study. J. Neurol. Neurosurg. Psychiatry 72, 615–620.

Fann, J.R., Bombardier, C.H., Dikmen, S., Esselman, P., Warms, C.A., Pelzer, E., Rau, H., Temkin, N., 2005. Validity of the patient health questionnaire-9 in assessing depression following traumatic brain injury. J. Head Trauma Rehabil. 20 (6), 501–511.

*Fann, J.R., Katon, W.J., Uomoto, J.M., Esselman, P.C., 1995. Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. Am. J. Psychiatry 152 (10), 1493–1499.

*Fedoroff, J.P., Starkstein, S.E., Forrester, A.W., Geisler, F.H., 1992. Depression in patients with acute traumatic brain injury. Am. J. Psychiatry 149 (7), 918–923.

*Findler, M., Cantor, J., Haddad, L., Gordon, W., Ashman, T., 2001. The reliability and validity of the SF-36 health survey questionnaire for use with individuals with traumatic brain injury. Brain Inj. 15 (8), 715–723.

First, M.B., 2010. Paradigm shifts and the development of the Diagnostic and Statistical Manual of Mental Disorder: past experience and future aspirations. Can. J. Psychiatry 55 (11), 692–700.

First, M.B., 2009. Harmonisation of ICD-11 and DSM-V: opportunities and challenges. Br. J. Psychiatry 195 (5), 382–390.

First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B., 1997. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV). American Psychiatric Association, Washington, DC.

*Forslund, M.V., Roe, C., Sigurdardottir, S., Andelic, N., 2013. Predicting health-related quality of life 2 years after moderate-to-severe traumatic brain injury. Acta Neurol. Scand. 128, 220–227.

*Gagnon, J., Bouchard, M.-A., Rainville, C., Lecours, S., St-Amand, J., 2006. Inhibition and object relations in borderline personality traits after traumatic brain injury. Brain Inj. 20 (1), 67–81.

*Glenn, M.B., O'Neil-Pirozzi, T., Goldstein, R., Burke, D., Jacob, L., 2001. Depression amongst outpatients with traumatic brain injury. Brain Inj. 15 (9), 811–818.

*Goldstein, F.C., Levin, H.S., Goldman, W.P., Clark, A.N., Altonen, T.K., 2001. Cognitive and neurobehavioral functioning after mild versus moderate traumatic brain injury in older adults. J. Int. Neuropsychol. Soc. 7 (3), 373–383.

*Gomez-Hernandez, R., Max, J.E., Kosier, T., Paradiso, S., Robinson, R.G., 1997. Social impairment and depression after traumatic brain injury. Arch. Phys. Med. Rehabil. 78 (12), 1321–1326.

*Gould, K.R., Ponsford, J., Johnston, L., Schönberger, M., 2011. The nature, frequency and course of psychiatric disorders in the first year after traumatic brain injury: a prospective study. Psychol. Med., 1–11.

Green, A., Felmingham, K., Baguley, I.J., Slewa-Younan, S., Simpson, S., 2001. The clinical utility of the Beck Depression Inventory after traumatic brain injury. Brain Inj. 15 (12), 1021–1028.

Gunstad, J., Suhr, J.A., 2001. "Expectation as etiology" Versus "the good old days": postconcussion syndrome symptom reporting in athletes, headache sufferers, and depressed individuals. J. Int. Neuropsychol. Soc. 7 (3), 323–333.

Hamilton, M., 1960. A rating scale for depression. J. Neurol. Neurosurg. Psychiatry 23 (1), 56.

*Hart, T., Brenner, L., Clark, A.N., Bogner, J.A., Novack, T.A., Chervoneva, I., Nakase-Richardson, R., Arango-Lasprilla, J.C., 2011. Major and minor depression after traumatic brain injury. Arch. Phys. Med. Rehabil. 92 (8), 1211–1219.

*Hart, T., Hoffman, J.M., Pretz, C., Kennedy, R., Clark, A.N., Brenner, L.A., 2012. A longitudinal study of major and minor depression following traumatic brain injury. Arch. Phys. Med. Rehabil. 93 (8), 1343–1349.

*Hawthorne, G., Gruen, R.L., Kaye, A.H., 2009. Traumatic brain injury and long-term quality of life: findings from an Australian study. J. Neurotrauma 26 (10), 1623–1633.

Helmchen, H., 2013. Different conceptions of mental illness: consequences for the association with patients. Front. Psychol. 4 (269), 1–5.

*Henry, J.D., Phillips, L.H., Crawford, J.R., Theodorou, G., Summers, F., 2006. Cognitive and psychosocial correlates of alexithymia following traumatic brain injury. Neuropsychologia 44 (1), 62–72.

*Herrmann, N., Rapoport, M.J., Rajaram, R.D., Chan, F., Kiss, A., Ma, A.K., Feinstein, A., McCullagh, S., Lanctot, K.L., 2009. Factor analysis of the Rivermead Post-Concussion Symptoms Questionnaire in mild-to-moderate traumatic brain injury patients. J. Neuropsychiatry Clin. Neurosci. 21 (2), 181–188.

Hibbard, M.R., Uysal, S., Kepler, K., Bogdany, J., Silver, J., 1998. Axis I psychopathology in individuals with traumatic brain injury. J. Head Trauma Rehabil. 13 (4), 24–39.

Hilsabeck, R.C., Gouvier, D.Wm., Bolter, J.F., 1998. Reconstructive memory bias in recall of neuropsychological symptomatology. J. Clin. Exp. Neuropsychol. 20 (3), 328–338.

Hopkins, W., 2002. A new view of statistics: a scale of magnitudes for effect statistics, Retrieved 08.03.14.

Hozo, S.P., Djulbegovic, B., Hozo, I., 2005. Estimating the mean and variance from the median, range, and the size of a sample. BMC Med. Res. Methodol. 5.

*Huang, D.B., Spiga, R., Koo, H., 2005. Use of the Zung depression scale in patients with traumatic brain injury: 1 year post-injury. Brain Inj. 19 (11), 903–908.

*Hudak, A., Hynan, L.S., Harper, C.R., Diaz-Arrastia, R., 2012. Association of depressive symptoms with functional outcome after traumatic brain injury. J. Head Trauma Rehabil. 27 (2), 87–98.

*Hudak, A., Warner, M., Marquez de la Plata, C., Moore, C., Harper, C., Diaz-Arrastia, R., 2011. Brain morphometry changes and depressive symptoms after traumatic brain injury. Psychiatry Res. 191 (3), 160–165.

Iverson, G.L., Brooks, B.L., Ashton, V.L., Lange, R.T., 2010a. Interview versus questionnaire symptom reporting in people with the postconcussion syndrome. J. Head Trauma Rehabil. 25 (1), 23–30.

Iverson, G.L., Lange, R.T., Brooks, B.L., Rennison, V.L., 2010b. "Good old days" bias following mild traumatic brain injury. Clin. Neuropsychol. 24 (1), 17–37.

*Jorge, R.E., Robinson, R.G., Arndt, S., 1993a. Depression following traumatic brain injury: a 1 year longitudinal study. J. Affect. Disord. 27, 233–243.

Jorge, R.E., Robinson, R.G., Arndt, S.V., Forrester, A.W., Geisler, F., Starkstein, S.E., 1993b. Comparison between acute- and delayed-onset depression following traumatic brain injury. J. Neuropsychiatry Clin. Neurosci. 5, 43–49.

Jorge, R.E., Robinson, R.G., 2002. Mood disorders following traumatic brain injury. NeuroRehabilitation 17, 311–324.

*Jorge, R.E., Robinson, R.G., Moser, D., Tateno, A., Crespo-Facorro, B., Arndt, S., 2004. Major depression following traumatic brain injury. Arch. Gen. Psychiatry 61 (1), 42–50.

Jorge, R.E., Starkstein, S.E., 2005. Pathophysiologic aspects of major depression following traumatic brain injury. J. Head Trauma Rehabil. 6, 475–487.

Juengst, S., Skidmore, E., Arenth, P.M., Niyonkuru, C., Raina, K.D., 2013. Unique contribution of fatigue to disability in community-dwelling adults with traumatic brain injury. Arch. Phys. Med. Rehabil. 94 (1), 74–79.

Kelley, E., Sullivan, C., Loughlin, J.K., Hutson, L., Dahdah, M.N., Long, M.K., Schwab, K.A., Poole, J.H., 2012. Self-awareness and neurobehavioral outcomes, 5 years or more after moderate to severe brain injury. J. Head Trauma Rehabil., http://dx.doi.org/10.1097/HTR.0b013e31826db6 b9 (advance online publication).

*Kempf, J., Werth, E., Kaiser, P.R., Bassetti, C.L., Baumann, C.R., 2010. Sleep–wake disturbances 3 years after traumatic brain injury. J. Neurol. Neurosurg. Psychiatry 81 (12), 1402–1405.

*Kennedy, R.E., Livingston, L., Riddick, A., Marwitz, J.H., Kreutzer, J.S., Zasler, N.D., 2005. Evaluation of the neurobehavioral functioning inventory as a depression screening tool after traumatic brain injury. J. Head Trauma Rehabil. 20 (6), 512–526.

Kessler, R.C., McGonagle, K.A., Swartz, M., Blazer, D.G., Nelson, C.B., 1993. Sex and depression in the National Comorbidity Survey. I. Lifetime prevalence, chronicity and recurrence. J. Affect. Disord. 29 (2/3), 85–96.

Kim, E., Lauterbach, E.C., Reeve, A., Arciniegas, D.B., Coburn, K.L., Mendez, M.F., Rummans, T.A., Coffey, E.C., 2007. Neuropsychiatric complications of traumatic brain injury: a critical review of the literature (a report by the ANPA committee on research). J. Neuropsychiatry Clin. Neurosci. 19 (2), 106–127.

*King, N.S., Kirwilliam, S., 2011. Permanent post-concussion symptoms after mild head injury. Brain Inj. 25 (5), 462–470.

*Kinsella, G., Moran, C., Ford, B., Ponsford, J., 1988. Emotional disorder and its assessment within the severe head injured population. Psychol. Med. 18 (1), 57–63.

*Konrad, C., Geburek, A.J., Rist, F., Blumenroth, H., Fischer, B., Husstedt, I., Arolt, V., Schiffbauer, H., Lohmann, H., 2011. Long-term cognitive and emotional consequences of mild traumatic brain injury. Psychol. Med. 41 (6), 1197–1211.

*Koponen, S., Taiminen, T., Hiekkanen, H., Tenovuo, O., 2011. Axis I and II psychiatric disorders in patients with traumatic brain injury: a 12-month follow-up study. Brain Inj. 25 (11), 1029–1034.

*Koponen, S., Taiminen, T., Portin, R., Himanen, L., Isoniemi, H., Heinonen, H., Hinkka, S., Tenovuo, O., 2002. Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. Am. J. Psychiatry 159 (8), 1315–1321.

*Kreutzer, J.S., Seel, R.T., Gourley, E., 2001. The prevalence and symptom rates of depression after traumatic brain injury: a comprehensive examination. Brain Inj. 15 (7), 563–576.

Kreutzer, J.S., Seel, R.T., Marwitz, J.H., 1999. Neurobehavioral Functioning Inventory: NFI. Psychological Corporation, San Antonio, Texas.

Kroenke, K., Spitzer, R.L., Williams, J.B.W., 2001. The PHQ-9. Validity of a brief depression severity measure. J. Gen. Intern. Med. 16, 606–613.

Kumar, A., Cook, I.A., 2002. White matter injury, neural connectivity and the pathophysiology of psychiatric disorders. Dev. Neurosci. 24, 255–261.

Kurtz, J.E., Shealy, S.E., Putnam, S.H., 2007. Another look at the paradoxical severity effects in head injury with the personality assessment inventory. J. Pers. Assess. 88 (1), 66–73.

Lewis, G., Pelosi, A.J., Araya, R., Dunn, G., 1992. Measuring psychiatric disorder in the community: a standardized assessment for use by lay interviewers. Psychol. Med. 22, 465–486.

*Lin, M.R., Chiu, W.T., Chen, Y.J., Yu, W.Y., Huang, S.J., Tsai, M.D., 2010. Longitudinal changes in the health-related quality of life during the first year after traumatic brain injury. Arch. Phys. Med. Rehabil. 91 (3), 474–480.

Lipsey, M.W., Wilson, D.B., 2001. Practical Meta Analysis. Applied Social Research Methods Series, vol. 49. Sage, Thousand Oaks, CA.

*Malec, J.F., Brown, A.W., Moessner, A.M., Stump, T.E., Monahan, P., 2010. A preliminary model for posttraumatic brain injury depression. Arch. Phys. Med. Rehabil. 91 (7), 1087–1097.

Malec, J.F., Testa, J.A., Rush, B.K., Brown, A.W., Moessner, A.M., 2007. Self-assessment of impairment, impaired self-awareness, and depression after traumatic brain injury. J. Head Trauma Rehabil. 22 (3), 156–166.

Mathias, J.L., Alvaro, P.K., 2012. Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: a meta-analysis. Sleep Med. 13 (7), 898–905.

*McCauley, S.R., Boake, C., Pedroza, C., Brown, S.A., Levin, H.S., Goodman, H.S., Merritt, S.G., 2005. Postconcussional disorder: are the DSM-IV criteria an improvement over the ICD-10? J. Nerv. Ment. Dis. 193 (8), 540–550.

*Meares, S., Shores, E.A., Taylor, A.J., Batchelor, J., Bryant, R.A., Baguley, I.J., Chapman, J., Gurka, J., Marosszeky, J.E., 2011. The prospective course of postconcussion syndrome: the role of mild traumatic brain injury. Neuropsychology 25 (4), 454–465.

Molina, J.A., 1983. Understanding the biopsychosocial model. Int. J. Psychiatry Med. 13 (1), 29–36.

Montgomery, S.A., Asberg, M., 1979. A new depression scale designed to be sensitive to change. Br. J. Psychiatry 134, 382–389.

Morris, T., 2010. Traumatic brain injury. In: Armstrong, C.L., Morrow, L. (Eds.), Handbook of Medical Neuropsychology. Springer, New York, pp. 17–32.

*O'Carroll, R.E., Woodrow, J., Maroun, F., 1991. Psychosexual and psychosocial sequelae of closed head injury. Brain Inj. 5 (3), 303–313.

Orwin, R.G., 1983. A fail-safe *N* for effect size in meta-analysis. J. Educ. Behav. Stat. 8 (2), 157–159.

*Peleg, G., Barak, O., Harel, Y., Rochberg, J., Hoofien, D., 2009. Hope, dispositional optimism and severity of depression following traumatic brain injury. Brain Inj. 23 (10), 800–808.

*Perlesz, A., Kinsella, G., Crowe, S., 2000. Psychological distress and family satisfaction following traumatic brain injury: injured individuals and their primary, secondary, and tertiary carers. J. Head Trauma Rehabil. 15 (3), 909–929.

*Ponsford, J., Cameron, P., Fitzgerald, M., Grant, M., Mikocka-Walus, A., 2011. Long-term outcomes after uncomplicated mild traumatic brain injury: a comparison with trauma controls. J. Neurotrauma 28 (1), 1–10.

*Ponsford, J., Olver, J., Ponsford, M., Nelms, R., 2003. Long-term adjustment of families following traumatic brain injury where comprehensive rehabilitation has been provided. Brain Inj. 17 (6), 453–468.

Ponsford, J., Olver, J., Ponsford, M., Schönberger, M., 2010. Two-year outcome following traumatic brain injury and rehabilitation: a comparison of patients from metropolitan Melbourne and those residing in regional Victoria. Brain Impair. 11 (3), 253–261.

*Ponsford, J., Schönberger, M., 2010. Family functioning and emotional state two and five years after traumatic brain injury. J. Int. Neuropsychol. Soc. 16 (2), 306–317.

*Ponsford, J., Ziino, C., 2003. Fatigue and attention following traumatic brain injury. Z. Neuropsychol. 14 (3), 155–163.

*Powell, J., Heslin, J., Greenwood, R., 2002. Community based rehabilitation after severe traumatic brain injury: a randomised controlled trial. J. Neurol. Neurosurg. Psychiatry 72 (2), 193–202.

*Powell, T.J., Collin, C., Sutton, K., 1996. A follow-up study of patients hospitalized after minor head injury. Disabil. Rehabil. 18 (5), 231–237.

Radloff, L.S., 1977. The CES-D scale: a self-report depression scale for research in the general population. Appl. Psychol. Meas. 1 (3), 385–401.

*Rao, V., Bertrand, M., Rosenberg, P., Makley, M., Schretlen, D.J., Brandt, J., Mielke, M.M., 2010. Predictors of new-onset depression after mild traumatic brain injury. J. Neuropsychiatry Clin. Neurosci. 22 (1), 100–104.

*Rao, V., Spiro, J., Vaishnavi, S., Rastogi, P., Mielke, M., Noll, K., Cornwell, E., Schretlen, D., Makley, M., 2008. Prevalence and types of sleep disturbances acutely after traumatic brain injury. Brain Inj. 22 (5), 381–386.

*Rapoport, M.J., McCullagh, S., Streiner, D., Feinstein, A., 2003. Age and major depression after mild traumatic brain injury. Am. J. Geriatr. Psychiatry 11 (3), 365–369.

Rapoport, M., 2012. Depression following traumatic brain injury. CNS Drugs 26 (2), 111–121.

*Reza, M., Ikoma, K., Ito, T., Ogawa, T., Mano, Y., 2007. N200 latency and P300 amplitude in depressed mood post-traumatic brain injury patients. Neuropsychol. Rehabil. 17 (6), 723–734.

Richman, W.L., Weisband, S., Kiesler, S., Drasgow, F., 1999. A meta-analytic study of social desirability distortion in computer administered questionnaires, traditional questionnaires, and interviews. J. Appl. Psychol., 754–775.

Robins, L.N., Helzer, J.E., Croughan, J., Ratcliff, K.S., 1981. National Institute of Mental Health Diagnostic Interview Schedule. Its history, characteristics, and validity. Arch. Gen. Psychiatry 38 (4), 381–389.

Robins, L.N., Wing, J., Wittchen, H., et al., 1988. The composite international diagnostic interview: an epidemiologic instrument suitable for use in conjunction with different diagnostic systems and in different cultures. Arch. Gen. Psychiatry 45 (12), 1069–1077.

Rochat, L., Ammann, J., Mayer, E., Annoni, J.M., Van Der Linden, M., 2009. Executive disorders and perceived socio-emotional changes after traumatic brain injury. J. Neuropsychol. 3 (2), 213–227.

Rosenthal, R., 1979. The file drawer problem and tolerance for null results. Psychol. Bull. 86 (3), 638–641.

Rosenthal, R., 1995. Writing meta-analytic reviews. Psychol. Bull. 118 (2), 183–192.

Rosenthal, M., Christensen, B.K., Ross, P.T., 1998. Depression following traumatic brain injury. Arch. Phys. Med. Rehabil. 79, 90–103.

*Rowland, S.M., Lam, C.S., Leahy, B., 2005. Use of the beck depression inventory-II (BDI-II) with persons with traumatic brain injury: analysis of factorial structure. Brain Inj. 19 (2), 77–83.

Rutherford, W.H., Merrett, J.D., McDonald, J.R., 1977. Sequelae of concussion caused by minor head injuries. Injury 9 (2), 171.

*Salmond, C.H., Menon, D.K., Chatfield, D.A., Pickard, J.D., Sahakian, B.J., 2006. Cognitive reserve as a resilience factor against depression after moderate/severe head injury. J. Neurotrauma 23 (7), 1049–1058.

*Schnabel, R., Kydd, R., 2012. Neuropsychological assessment of distractibililty in mild traumatic brain injury and depression. Clin. Neuropsychol. 26 (5), 769–789.

*Schnieders, J., Willemsen, D., De Boer, H., 2012. Factors contributing to chronic fatigue after traumatic brain injury. J. Head Trauma Rehabil. 27 (6), 404–412.

Schwarzbold, M., Diaz, A., Martins, E.T., Rufino, A., Amante, L.N., Thais, M.E., Quevedo, J., Hohl, A., Linhares, M.N., Walz, R., 2008. Psychiatric disorders and traumatic brain injury. Neuropsychiatr. Dis. Treat. 4 (4), 797–816.

*Sebit, M.B., Siziya, S., Ndetei, D.M., Sande, G.M., 1998. Study of psychiatric consequences of closed head injury at the Kenyatta National Hospital. East Afr. Med. J. 75 (6), 332–335.

*Seel, R.T., Kreutzer, J.S., 2003. Depression assessment after traumatic brain injury: an empirically based classification method. Arch. Phys. Med. Rehabil. 84 (11), 1621–1628.

*Senathi-Raja, D., Ponsford, J., Schönberger, M., 2010. The association of age and time postinjury with long-term emotional outcome following traumatic brain injury. J. Head Trauma Rehabil. 25 (5), 330–338.

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., Hergueta, T., Baker, R., Dunbar, G.C., 1998. The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J. Clin. Psychiatry 59, 22–33.

Sherin, J.E., Nemeroff, C.B., 2011. Post-traumatic stress disorder: the neurobiological impact of psychological trauma. Dialogues Clin. Neurosci. 13, 263–278.

Silver, J.M., McAllister, T.W., Arciniegas, D.B., 2009. Depression and cognitive complaints following mild traumatic brain injury. Am. J. Psychiatry 166 (6), 653–661.

*Skilbeck, C., Dean, T., Thomas, M., Slatyer, M., 2013. Impaired National Adult Reading Test (NART) performance in traumatic brain injury. Neuropsychol. Rehabil. 23 (2), 234–255.

*Smith, H.E., 1992. Head injury follow-up: is the Sickness Impact Profile a useful clinical tool? Clin. Rehabil. 6 (1), 31–39.

Stroup, D.F., Berlin, J.A., Morton, S.C., Olkin, I., Williamson, G.D., Rennie, D., Moher, D., Becker, B.J., Sipe, T.A., Thacker, S.B., 2000. Meta-analysis of observational studies in epidemiology; a proposal for reporting. J. Am. Med. Assoc. 283 (15), 2008–2012.

Tsaousides, T., Ashman, T.A., Gordon, W.A., 2013. Diagnosis and treatment of depression following traumatic brain injury. Brain Impair. 14 (1), 63–76.

*Tyerman, A., Humphrey, M., 1984. Changes in self-concept following severe head injury. Int. J. Rehabil. Res. 7 (1), 11–23.

*Van Der Horn, H.J., Spikman, J.M., Jacobs, B., Van Der Naalt, J., 2013. Postconcussive complaints, anxiety, and depression related to vocational outcome in minor to severe traumatic brain injury. Arch. Phys. Med. Rehabil. 94 (5), 867–874.

*van Reekum, R., Bolago, I., Finlayson, M.A.J., Garner, S., 1996. Psychiatric disorders after traumatic brain injury. Brain Inj. 10 (5), 319–327.

Varney, N.R., Martzke, J.S., Roberts, R.J., 1987. Major depression in patients with closed head injury. Neuropsychology 1 (1), 7–9.

*von Steinbuchel, N., Wilson, L., Gibbons, H., Hawthorne, G., Hofer, S., Schmidt, S., Bullinger, M., Maas, A., Neugebauer, E., Powell, J., von Wild, K., Zitnay, G., Bakx, W., Christensen, A.L., Koskinen, S., Formisano, R., Saarajuri, J., Sasse, N., Truelle, J.L., 2010. Quality of Life after Brain Injury (QOLIBRI): scale validity and correlates of quality of life. J. Neurotrauma 27 (7), 1157–1165.

Wacholder, S., Silverman, D.T., McLaughlin, J.K., Mandel, J.S., 1992. Selection of controls in case-control studies. II. Types of controls. Am. J. Epidemiol. 135 (9), 1029–1041.

Waljas, M., Lange, R.T., Hakulinen, U., Huhtala, H., Dastidar, P., Hartikainen, K., Ohman, J., Iverson, G.L., 2014. Biopsychosocial outcome after uncomplicated mild traumatic brain injury. J. Neurotrauma 31, 108–124.

*Wallace, C.A., Bogner, J., 2000. Awareness of deficits: emotional implications for persons with brain injury and their significant others. Brain Inj. 14 (6), 549–562.

*Weddell, R.A., 2010. Relatives' criticism influences adjustment and outcome after traumatic brain injury. Arch. Phys. Med. Rehabil. 91 (6), 897–904.

*Whalley-Hammell, K.R., 1994. Psychosocial outcome following severe closed head injury. Int. J. Rehabil. Res. 17 (4), 319–332.

*Whelan-Goodinson, R., Ponsford, J., Johnston, L., Grant, F., 2009. Psychiatric disorders following traumatic brain injury: their nature and frequency. J. Head Trauma Rehabil. 24 (5), 324–332.

Whelan-Goodinson, R., Ponsford, J., Schönberger, M., 2009a. Validity of the Hospital Anxiety and Depression Scale to assess depression and anxiety following traumatic brain injury as compared with the Structured Clinical Interview for DSM-IV. J. Affect. Disord. 114 (1–3), 94–102.

Whiteside, D.M., Galbreath, J., Brown, M., Turnbull, J., 2012. Differential response patterns on the personality assessment inventory (PAI) in compensation-seeking and non-compensation-seeking mild traumatic brain injury patients. J. Clin. Exp. Neuropsychol. 34 (2), 172–182, http://dx.doi.org/10.1080/13803395.2011.630648.

*Williams, W.H., Williams, J.M.G., Ghadiali, E.J., 1998. Autobiographical memory in traumatic brain injury: neuropsychological and mood predictors of recall. Neuropsychol. Rehabil. 8 (1), 43–60.

Wing, J.K., Babor, T., Brugha, T., Burke, J., Cooper, J.E., Giel, R., Jablenski, A., Regier, D., Sartorius, N., 1990. SCAN: schedules for clinical assessment in neuropsychiatry. Arch. Gen. Psychiatry 47 (6), 589–593.

Wing, J.K., Cooper, J.E., Sartorius, N., 1974. The Description and Classification of Psychiatric Symptoms: An Instruction Manual for the PSE and CATEGO System. Cambridge University, London.

*Wood, R.L., Williams, C., 2008. Inability to empathize following traumatic brain injury. J. Int. Neuropsychol. Soc. 14 (2), 289–296.

World Health Organisation, 1977. International Classification of Diseases: Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death (ICD-9). World Health Organization, Geneva.

World Health Organisation, 1992. ICD-10 Classifications of Mental and Behavioural Disorder: Clinical Descriptions and Diagnostic Guidelines. World Health Organisation, Geneva.

Zigmond, A.S., Snaith, R.P., 1983. The hospital anxiety and depression scale. Acta Psychiatr. Scand. 67 (6), 361–370, http://dx.doi.org/10.1111/j.1600-0447.1983.tb09716.x.

Zung, W., 1965. A self-rating depression scale. Arch. Gen. Psychiatry 12, 63–70.