



Social Anxiety Following Traumatic Brain Injury: An Exploration of Associated Factors

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Social Anxiety Following Traumatic Brain Injury: An Exploration of Associated Factors

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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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Abstract

Social anxiety (SA) following traumatic brain injury (TBI) has the potential to affect an individual's general psychological wellbeing and social functioning, however little research has explored factors associated with its development. The present study used hierarchical multiple regression to investigate the demographic, clinical and psychological factors associated with SA following TBI. A sample of 85 people who experienced TBI were recruited through social media websites and brain injury services across the NorthWest of England. The overall combined biopsychosocial model was significant, explaining 52-54.3% of the variance in SA (across five imputations of missing data). The addition of psychological variables (self-esteem, locus of control, self-efficacy) made a significant contribution to the overall model, accounting for an additional 12.2-13% of variance in SA above that explained by demographic and clinical variables. Perceived stigma was the only significant independent predictor of SA ($B = .274, p = .005$). The findings suggest that psychological variables are important in the development of SA following TBI and must be considered alongside clinical factors. Furthermore, the significant role of stigma highlights the need for intervention at both an individualised and societal level.

Keywords: traumatic brain injury, social anxiety, stigma, psychological

Social Anxiety Following Traumatic Brain Injury: An Exploration of Associated Factors

People who have experienced a traumatic brain injury (TBI) are at increased risk of developing psychological difficulties such as depression and anxiety (Scholten et al., 2016; Osborn, Mathias, & Fairweather-Schmidt, 2014; Gould, Ponsford, Johnston and Schönberger, 2011; Whelan-Goodinson, Ponsford, Schönberger & Johnston, 2010; Bryant et al., 2010). However, recognising psychological problems after TBI can be challenging, given the complex interactions between the neurological and emotional sequelae of TBI and the difficulties in identifying symptoms of psychological problems in the context of other factors (e.g., cognitive impairment, physical disability) associated with TBI (Kim et al., 2007; Scheutzwow & Wiercisiewski, 1999). Nonetheless, as psychological problems following TBI can be longstanding (Konrad et al., 2011) and may affect wellbeing and inhibit recovery (Osborn et al., 2014), it is imperative to improve understanding and management of these difficulties during assessment and rehabilitation (Williams, Evans & Fleminger, 2003).

Furthermore, it is vital to understand the social context in which TBI rehabilitation occurs. Social functioning is commonly affected by TBI and this can have a significant impact on life satisfaction (Pierce & Hanks, 2006; Truelle, Fayol, Montreuil, & Chevignard, 2010; Jones et al., 2010). Qualitative research highlights the importance of social activity following TBI in making sense of oneself (Yeates, Gracey, & Mcgrath, 2008), and social support is predictive of lower levels of post-traumatic stress (Jones et al., 2012). However, declines in activity, social contact, independence, functional status and employment opportunities are often reported following TBI (Antonak, Livneh, & Antonak, 1993; Temkin, Corrigan, Dikmen, & Machamer, 2009). Severity of injury fails to account fully for differences in psychosocial functioning (Antonak et al., 1993) and life satisfaction post-TBI (Jones et al., 2010), with the latter study finding that social support mediated the relationship between well-being and injury severity.

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Moreover, following TBI people may feel embarrassed or self-conscious in social situations given the frequency of physical consequences (e.g., physical impairment, hemiparesis, skull depressions, scarring, tremors, motor/speech problems) and often unseen cognitive problems with word finding, attention, memory, executive functioning and processing speed (Rochat, Ammann, Mayer, Annoni, Van Der Linden, 2009; Hiott & Labbate, 2002; Moore, Terryberry-Spohr & Hope, 2006). Therefore, social interaction can be negatively impacted following TBI if a person is less able to follow or engage in conversation (Morris et al., 2005). Consequently, problems following TBI may result in people becoming particularly anxious in social situations (Moore et al., 2006; Wright & Telford, 1996).

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However, despite the importance of social reintegration, social anxiety in people with a TBI has been the subject of very little research. Social anxiety (SA) is characterised by a marked fear of situations in which a person might face scrutiny from others and subsequent avoidance of common triggers (e.g., social interactions, meeting new people, public speaking) which can result in significant distress and impairments in functioning (National Institute for Health and Care Excellence [NICE], 2013; American Psychiatric Association [APA], 2013). While both anxiety (Rao & Lyketsos, 2002) and declines in psychosocial functioning (Ponsford et al., 2014; Antonak et al., 1993) following TBI are well documented, the available research examining SA following TBI is limited and of poor quality. Only two studies have been identified which have assessed social anxiety in this population. A prospective cohort study of people who had experienced traumatic injuries found that 6.1% of people with mild-TBI met criteria for SA three months post-injury, rising to 9% after 12 months (Bryant et al., 2010). Conversely, Newton and Johnson (1985) found that SA was lower in participants with a TBI compared to those without. However, the TBI group comprised only eleven participants who exhibited a broad range of scores on a measure of

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3 SA. The authors concluded that although the mean score was lower than the control group, a
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5 high level of SA was observed in the majority of the TBI group ($n = 8$).
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8 This lack of research interest may be a consequence of the complex interaction and
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10 overlap between psychological and neurological problems as discussed above. It may also
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12 result from the criteria within the Diagnostic and Statistical Manual of Mental Disorders,
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14 Fifth Edition (DSM-5; APA, 2013) for SA which state that, if a medical condition is present,
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16 anxiety or avoidance must be unrelated or out of proportion to it. This suggests that a
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18 diagnostic label of social anxiety disorder may not be appropriate for people experiencing
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20 anxiety in social situations after TBI. This may result in social anxiety not being considered
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22 in this population, or such difficulties being attributed to the cognitive or neurological
23
24 consequences of TBI. However, this is not in keeping with recommendations for a broad and
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26 biopsychosocial approach to providing support and rehabilitation following TBI (Gracey,
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28 Evans & Malley, 2009; Wilson & Gracey, 2009).
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32 No guidance is available specific to the management of SA after TBI, but empirically-
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34 based guidance for generic SA interventions in the UK (NICE, 2013) recommends cognitive
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36 behavioural therapy (CBT) as a first-line intervention (i.e., before pharmacological
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38 interventions), underpinned by a specifically developed theoretical model (e.g., Clark &
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40 Wells, 1995). However, a randomised controlled trial of a CBT programme for SA after
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42 acquired brain injury (ABI) found that although SA did reduce, treatment effects were not
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44 statistically significant (Hodgson, McDonald, Tate, & Gertler, 2012). However, a small
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46 sample size ($n = 12$) and variability in the ABI group (people who had experienced stroke,
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48 hypoxic brain injury and cerebral oedema were included alongside those who had
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50 experienced TBI) limits the usefulness of this study in understanding management of SA
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52 after TBI.
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Despite the lack of research or guidance around SA after TBI, a literature review exploring anxiety following mild TBI (Moore et al., 2006) highlighted the potential for SA to be a significant problem in this population. Furthermore, Soo, Tate and Rapee (2012) present a theoretical rationale for high levels of SA in children and adolescents who have experienced TBI. They draw on Kendall and Terry's (1996) model for understanding individual differences and predicting psychosocial adjustment outcomes following TBI, acknowledging a role for direct (neurological and cognitive impairment) and indirect (situational and environmental) antecedent factors, but also emphasising the importance of an individual's psychological resources such as appraisal style and coping responses. This is consistent with cognitive theories of SA (e.g., Clark & Wells, 1995; Wells, 2013) and approaches to management of other anxiety problems following TBI (Williams et al., 2003; Soo & Tate, 2009). Consequently, an understanding of SA following TBI in adults must be guided by research which explores the role of potentially relevant neurological, cognitive, situational and psychological factors to guide assessment, formulation and intervention during acute and long-term rehabilitation.

A broad range of psychological variables may be important in SA following TBI (Soo et al., 2012). Locus of control (LoC), the beliefs a person holds about how the behaviour of themselves and others influences their health (Wallston, Stein, & Smith, 1994), has been associated with SA (Cloitre, Heimberg, Liebowitz, & Gitow, 1992; Kennedy, Lynch, & Schwab, 1998) and emotional problems in people who have experienced TBI (Moore & Stambrook, 1992). Self-efficacy, the beliefs people hold about their capabilities is also associated with SA (Leary & Atherton, 1986) and is predictive of global life satisfaction following TBI (Cicerone & Azulay, 2007). Low self-esteem is also linked to SA (Ritter, Ertel, Beil, Steffens, & Stangier, 2013). Though debate continues around the consistency of the construct, self-esteem is generally defined as the global, subjective and emotional

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3 judgements one holds about the self (Guindon, 2002), which are activated and reinforced in
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5 social situations and contribute to fear of negative evaluation (Wells, 2013; Clark & Wells,
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7 1995; Rapee & Spence, 2004). People who have experienced TBI have been found to have
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9 lower self-esteem (Ponsford, Kelly, & Couchman, 2014) and self-esteem has been shown to
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11 predict psychosocial outcomes following TBI (Tate & Broe, 1999).

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14 Furthermore, fear of negative evaluation may mean that people with SA perceive or
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16 experience higher levels of stigma (Anderson, Jeon, Blenner, Wiener, & Hope, 2015; Clark
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18 & Wells, 1995). People who are socially anxious may be rejected or perceived negatively,
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20 particularly if anxiety related behaviours (e.g., gripping hands together, avoiding eye contact)
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22 compound the anxiety symptoms or impair social performance (Wells, 2013; Rapee &
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24 Spence, 2004). As highlighted above, the physical and cognitive consequences of TBI may
25
26 add further challenges to social interactions. Qualitative research has suggested stigma may
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28 be a potential factor affecting wellbeing following TBI, with participants highlighting the
29
30 lack of public understanding about the consequences of TBI and how this impacts on their
31
32 social engagement (Morris et al., 2005; Nochi, 1998). Furthermore, perceived stigma is
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34 strongly associated with anxiety in people with chronic physical conditions (Alonso et al.,
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36 2008) and epilepsy (Beyenburg, Mitchell, Schmidt, Elger, & Reuber, 2005).

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41 In conclusion, despite the theoretical rationale for SA following TBI presented by Soo
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43 et al. (2012) and Moore et al., (2006), present understanding of SA following TBI is limited
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45 given the limited available research. No research to date has explored psychological factors
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47 which might contribute to the development of SA following TBI to provide guidance for
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49 assessment and intervention. While it is recognised that psychological problems may predate
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51 a brain injury (Williams et al., 2003), people who have experienced TBI may be at greater
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53 risk of developing SA due to the nature of the factors described above. Consequently, the
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55 present study aimed to investigate psychological factors associated with SA following TBI,
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3 alongside clinical and demographic variables. It was hypothesised that psychological
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5 variables such as LoC, self-efficacy, self-esteem and perceived stigma would account for an
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7 additional and significant amount of variance in SA, above that explained by demographic
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9 and clinical variables.
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11 **Methods**

12 **Design**

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15 The study employed a quantitative, cross-sectional within-subjects design to explore
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17 factors predicting SA after TBI. Self-report questionnaires were used as the data collection
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19 method. If required, participants were given support from the lead researcher to complete the
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21 questionnaires.
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25 **Participants**

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27 Participants were required to have sustained a TBI, defined as an injury caused by an
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29 external or mechanical force (Morton & Wehman, 1995), to differentiate from the broader
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31 categorisation of ABI. Participants in the study were required to be aged over 18 and able to
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33 read English (due to lack of the validated measures in other languages). Participants were
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35 required to have sustained a TBI after the age of 16 to allow for specific examination of
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37 factors in relation to adults, as other developmental factors are likely to influence cognitive
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39 and psychological outcomes following TBI experienced in childhood or adolescence
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41 (Anderson et al., 2006; Catroppa, Anderson, Morse, Hariou, & Rosenfeld, 2008). Given the
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43 focus on social functioning, participants were required to be living in the community (either
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45 at home or in long-term supported accommodation) rather than a medical ward or residential
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47 rehabilitation unit. Participants were also required to have capacity to consent to participation
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49 in the study.
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54 An a priori power calculation for multiple regression analysis, assuming a medium
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56 effect size of 0.15, 80% power and an alpha level set at $p = .05$, suggested that a sample of
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3 between 92 and 139 would be required. A total of 98 participants were recruited, with 54
4 participants completing the questionnaires online and 44 submitting paper copies provided
5 via National Health Service (NHS) or third sector services. Five participants who completed
6 the study online were excluded from the analysis as they described their injury as an ABI
7 (e.g., subarachnoid haemorrhage) rather than a TBI and therefore did not meet all the
8 inclusion criteria. A further eight participants were excluded as a significant amount of
9 questionnaire data (more than 10%, as recommended by Bennett, 2001) were missing.

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11 Therefore, a total of 85 participants provided data for the analyses. Participants
12 ranged in age from 19 to 81 years ($M = 42.4$, $SD = 13.335$). The final sample included 63.5%
13 ($n = 54$) males and 32.9% ($n = 28$) females, with 3.5% ($n = 3$) reporting “Other / Prefer not to
14 say”. Further demographic information is shown in Table 1.

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29 Due to ethical and resource constraints, medical data regarding severity of injury were
30 not available. Participants were asked to report the length of time they were in hospital for
31 after their injury ($M = 16.529$ weeks, $SD = 32.120$) and time since injury ($M = 7.719$ years,
32 $SD = 8.733$).

33 34 35 36 37 38 39 **Measures**

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41 **Outcome variable.** The Social Phobia Inventory (SPIN; Connor et al., 2000) was
42 used as the outcome measure for the study. The SPIN is a 17-item self-report measure of
43 three domains of SA; fear, avoidance and physiological discomfort. Responses are scored
44 from 0 (not at all) to 4 (extremely), with a maximum total score of 68 indicating high levels
45 of SA. A cut-off score of 19 is recommended by the authors to distinguish those with SA.
46 High levels of internal consistency ($\alpha = .95$) and test-retest reliability ($r = .86$) have been
47 demonstrated (Antony, Coons, McCabe, Ashbaugh, & Swinson, 2006; Connor et al., 2000).
48 Although the measure has not been used in a TBI population in any published research to
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3 date, it has been utilised with patients with multiple sclerosis (Poder et al., 2009) and is
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5 recommended by guidance provided by NICE (2013) for use in NHS services within the UK.
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7 The SPIN's face validity and brevity make it the most appropriate measure from available
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9 measures of SA.
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11 **Predictor variables.** The Applied Cognition measure (Neuro-QOL, 2012) was used
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13 to assess subjective severity of cognitive problems. This 18-item measure assesses perceived
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15 difficulties in everyday cognitive domains including memory, attention, and decision-
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17 making. Responses range from never (1) to very often (5), with a maximum score of 90. High
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19 levels of internal consistency ($\alpha = .95$) and test-retest reliability ($r = .82$) have been
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21 demonstrated in samples of patients with a range of neurological problems (e.g., stroke,
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23 epilepsy, Parkinson's disease) but data are not available for a TBI sample (Neuro-QOL,
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25 2010).
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29 Form C of the Multidimensional Health Locus of Control (MHLoC, Wallston, Stein,
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31 & Smith, 1994) assesses belief in one's ability to control health outcomes, in relation to a
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33 specific illness or disease. The measure encompasses four subscales of LoC: internal; chance;
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35 powerful others (doctors) and powerful others (other people). Responses are scored from 1
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37 (strongly disagree) to 6 (strongly agree), with a higher subscale score indicating higher LoC
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39 (no total score is calculated). Wallston et al. (1994) demonstrated acceptable levels of internal
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41 consistency and test-retest reliability for each subscale; internal ($\alpha = .79 - .87$; $r = .80$),
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43 chance ($\alpha = .79 - .82$; $r = .72$), doctors ($\alpha = .71$; $r = .58$) and other people ($\alpha = .70 - .71$; $r =$
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45 $.40$). Despite its focus on control over one's specific illness or disease (Wallston, 2005), no
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47 published research has used Form C with a TBI population. However, Forms A and B of the
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49 MHLoC have been used in previous TBI research (Bedard et al., 2005; Moore & Stambrook,
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51 1992), and Form C has been used to assess LoC following spinal cord injury (Waldron et al.,
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53 2010).
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3 The Rosenberg Self-Esteem Scale (RSES, 1965) is a 10-item measure, with responses
4 recorded on a 0 to 3 scale (reverse coded on some items) so that a low score on the RSES
5 indicates low self-esteem. The RSE demonstrates high internal consistency ($\alpha = .92$), and
6 test-retest reliability ($r = .85$) after two weeks (Rosenberg, 1979). This measure has been used
7 to examine self-esteem in people who have experienced a TBI (e.g., Anson & Ponsford,
8 2006a; Anson & Ponsford, 2006b; Ponsford et al., 2014).

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16 The Self-Efficacy for Symptom Management Scale (Cicerone & Azulay, 2007)
17 assesses confidence in managing common challenges and seeking support after TBI. The 13-
18 items measure is scored 1 (not at all confident) to 10 (totally confident), with a maximum
19 total score of 130 indicating high self-efficacy. High levels of internal consistency ($\alpha = .93$)
20 and test-retest reliability ($r = .93$) have been demonstrated (Cicerone & Azulay, 2007).

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28 The Stigma scale published by Neuro-QOL (2012) is a 24-item measure which
29 examines a person's perceptions of self and publically enacted prejudice and discrimination
30 experienced as a result of neurological problems. Responses are scored from 1 (never) to 5
31 (always), with a maximum score of 120 indicating high levels of perceived stigma. High
32 levels of internal reliability ($\alpha = .91$) and test-retest reliability ($r = .82$) have been
33 demonstrated in samples of patients with a range of neurological problems (e.g., stroke,
34 epilepsy, Parkinson's disease) but no data are available for a TBI sample (Rao et al., 2009).
35 For the purposes of the study, the word 'illness' was replaced with the term 'brain injury' on
36 each item of the questionnaire.

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48 The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was
49 designed for use with people with physical health problems and assesses anxiety and
50 depression without relying on somatic symptoms of illness (e.g., fatigue, insomnia). The 14-
51 item measure is scored on a 0 to 3 scale, appropriately coded so that a higher score on each
52 subscale indicates a more severe problem with anxiety or depression. A review of its
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3 psychometric properties reports good levels of internal consistency on the anxiety ($\alpha = .68 -$
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.93) and depression ($\alpha = .67 - .9$) subscales across a variety of settings (Bjelland, Dahl, Haug, & Neckelmann, 2002), with similar findings reported by Whelan-Goodinson, Ponsford and Schönberger (2009) with a TBI sample (depression $\alpha = .88$; anxiety $\alpha = .92$). The HADS has been used to measure depression and anxiety after TBI in a number of published studies (e.g., Anson & Ponsford, 2006a; Anson & Ponsford, 2006b; Downing, Stolwyk, & Ponsford, 2013).

Procedure

Potential participants were identified and recruited through professionals working in neuropsychology teams across nine NHS Trusts in the North-West of England and third sector organisations relevant to TBI. Participants were also able to self-refer into the study and could opt to complete an online version of the study made using Qualtrics Survey Software (Qualtrics, 2013), which provided security and encryption for online information. The study was advertised via social networking websites and posters displayed in NHS neuropsychology services and third sector organisations.

Prior to completing the questionnaires, participants were required to complete a screening and consent form based on the inclusion and exclusion criteria outlined above. On the online version of the study, participants were only able to progress onto the questionnaires if they answered each item of the consent form. Capacity to consent and participate in the study was assumed in line with the UK Mental Capacity Act (2005). Participants had the option of completing the questionnaires online or on paper posting them to the lead researcher. To reduce bias, the online study was set to present questionnaires in a random order.

Ethical Approval

The study received ethical approval from the UK NHS National Research Ethics Service, followed by local approval from the Research and Development Departments of each NHS Trust involved in recruitment. This approval also covered participants recruited through third sector organisations and online.

Data Analysis Strategy

Data were analysed using IBM SPSS Statistics version 20. All questionnaires were scored in accordance with scale instructions and reverse coded as required. Relationship status was recoded to a binary variable (i.e., yes / no). Due to its descriptive nature, cause of injury was not entered into the regression model. Anxiety (measured by HADS) was not entered into the regression model as it correlated too highly with the outcome variable ($r = .726, p < .001$) and, as it is conceptually similar, would have reduced the variance available to other variables. Additionally, depression was considered a clinical variable rather than a psychological one, due to the focus of the HADS on measuring clinical difficulties associated with depression.

Throughout the study, a p value of .05 was used as a threshold for statistical significance in line with convention (Field, 2013). Furthermore, the decision was taken not to use Bonferroni corrections to counteract multiple comparisons as this would have resulted in a very low p value and significantly reduced statistical power.

Hierarchical multiple regression analysis was used to explore the study hypothesis. Variables were entered into the model in three blocks; demographic, clinical, psychological. Consistent with the available theoretical rationale for SA following TBI discussed above, this allowed for examination of the amount of variance in SA which could be explained by psychological variables, above that explained by demographic and clinical variables.

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3 In determining what variables were entered into the regression model, decisions for
4 subset selection were made based on effect size instead of p values. While use of p values is
5 common, effect sizes are less reliant on sample size (Coe, 2002). Given the relatively low
6 sample size in this study ($n = 85$), variables were included in the multiple regression analysis
7 if a small effect size was observed (i.e., $r > .1$; Cohen, 1988). This threshold was chosen to
8 allow an inclusive, exploratory approach which minimised the risk of overlooking emerging
9 effects of small magnitude (Hemphill, 2003).
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19 Results

20 Data Preparation and Analysis

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22 It did not appear that there were any systematic biases or patterns to the missing data
23 as defined by Graham (2009), with 34 cases (40% of the sample) having incomplete data
24 across 42 (34.43%) of the variables. Little's (1988) Missing Completely At Random
25 (MCAR) test was not significant ($X^2 = 1921.880$, $df = 3105$, $p = 1.000$), suggesting that the
26 null hypothesis of data being missing randomly could be assumed.
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34 Even after removing the eight cases missing more than 10% of data, the number of
35 other cases missing smaller amounts of data was high. Listwise or pairwise deletion methods
36 were not considered appropriate as this would have seen a large proportion of cases deleted,
37 thereby reducing sample size and power in addition to potentially introducing bias into the
38 multiple regression model. Consequently, multiple imputation was conducted with the data
39 provided by 85 participants to analyse missing data and input substituted values (Rubin,
40 1987; Schaffer, 1997). Five iterations of imputation were performed (Schaffer, 1997).
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50 Clinical Characteristics of Sample

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52 Descriptive statistics for all self-report measures used in the study are provided in
53 Table 2. As can be seen in Table 2, all measures demonstrated acceptable levels of internal
54 consistency ($\alpha > .6$; Hair, Anderson, Tatham & Black, 2006).
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[INSERT TABLE 2 HERE]

Independent samples t-test showed no significant difference on SPIN scores between participants who completed the questionnaire online compared to those who did not ($t(91) = .635, p = .527$). Using the cut-off scores for social anxiety as recommended by the authors of the SPIN (Connor et al, 2000), most participant scores (47.1%) lay in the 'None' category (> 20). A further 15 participants (17.6%) scored within the 'Mild' category, 13 (15.3%) scored within the 'Moderate' category, 10 (11.8%) scored in the 'Severe' category, and 7 (8.2%) participants were categorised as 'Very Severe'. Using the cut-offs provided by the scale authors (Zigmond & Snaith, 1983), 70.6% of the sample showed clinically significant levels of anxiety (with 21.2% in the severe category) while 63.5% of the sample showed clinically significant levels of depression (with 20% in the severe category).

Correlational Analysis

Correlational analysis (Pearson's r) was conducted on the pooled dataset comprising of all iterations of the multiple imputation process (Rubin, 1987). Correlations are shown in Tables 3 and 4.

[INSERT TABLE 3 & 4 HERE]

The following variables correlated significantly ($p < .05$) with higher SA scores on the SPIN: not being employed ($r = .239, p = .028$); higher levels of cognitive problems ($r = .476, p < .001$); higher levels of internal ($r = .248, p = .022$) and chance ($r = .217, p = .046$) LOC; lower self-esteem ($r = -.441, p < .001$); lower self-efficacy ($r = -.472, p < .001$); higher perceived stigma ($r = .654, p < .001$); higher levels of anxiety ($r = .726, p < .001$) and higher levels of depression ($r = .516, p < .001$). Age, gender, time since TBI, time in hospital, living alone, relationship status and the two Powerful Others subscales of the MHLoC (Doctors and Others) did not significantly correlate with SA scores.

Hierarchical Multiple Regression Analysis

Hierarchical multiple regression analysis was conducted to examine if the predictor variables were able to explain the variance in SA scores. Predictor variables which correlated with SA demonstrating a small effect size or above (Pearson's $r > 0.1$) were entered into the regression model. Predictor variables were entered into the regression model in three blocks: (a) demographic variables (gender, employment status); (b) clinical variables (time since TBI, perceived cognitive problems, depression); (c) psychological variables (MHLoC internal, MHLoC chance, self-esteem, self-efficacy, perceived stigma).

The overall model was significant, both with the original dataset ($F(2, 63) = 5.918, p < .001$, explaining 51.8% ($R^2 = .518, R^2_{adj} = .431$) of the variance in SA scores and across all five imputations of missing data, with $F(2, 82)$ values ranging from 8.006 to 8.799, with all values of $p < .001$. The amount of variance in SA scores explained ranged from 52% ($R^2 = .520, R^2_{adj} = .455$) to 54.3% ($R^2 = .543, R^2_{adj} = .481$) of the variance in SA scores.

The Durbin-Watson values across the imputations ranged from 1.962 to 2.000 compared to the value from the original data of 1.846, and therefore it was assumed there was no autocorrelation of residuals (Field, 2013). Examination of the VIF, tolerance and eigenvalues confirmed that there was no evidence of collinearity within the dataset (Bowerman & O'Connell, 1990; Menard, 1995; Field, 2013). Graphical representation of the data suggested that assumptions of homoscedasticity and normally distributed residuals could be upheld.

Block one (demographic variables) accounted for 10.3% ($R^2 = .103, R^2_{adj} = .074, p = .033$) of the variance in SA scores in the original dataset, rising to between 11.9% ($R^2 = .119, R^2_{adj} = .097, p = .006$) and 14.7% ($R^2 = .147, R^2_{adj} = .126, p = .001$) following imputation. The addition of block two (clinical variables) made a significant contribution to the model, increasing the total variance explained to 36.1% ($\Delta R^2 = .259, p < .001$) for the original

dataset and between 39.8% ($\Delta R^2 = .279, p < .001$) and 41.3% ($\Delta R^2 = .280, p < .001$) following imputation, with significant changes in F ($p < .001$) for both original and imputed data. Within this block of variables, standardised beta values across imputations indicated that higher levels of perceived cognitive problems ($\beta = .249$ to $.253, p = .012$) and depression ($\beta = .348$ to $.367, p < .001$) were significant independent predictors of higher reported SA, with time since injury not statistically significant ($\beta = .055$ to $.064, p = .516$).

The addition of block three (psychological variables) also made a significant contribution to the overall model, explaining an additional 15.7% ($\Delta R^2 = .157, p < .001$) of the total variance for the original dataset and between 12.2% ($\Delta R^2 = .122, p < .001$) and 13% ($\Delta R^2 = .130, p < .001$) for each imputation. The change in F associated with the addition of block three was statistically significant for both original ($p = .007$) and imputed data ($p = .002$ to $.004$).

For individual predictors of SA, the overall model including all three blocks (and based on data pooled from all imputations) indicated that only higher levels of perceived stigma significantly predicted higher levels of SA ($B = .274, \beta = .334$ to $.341, t = 2.789, p = .005$). In the final model, reported cognitive problems and depression ceased to meet criteria for statistical significance. In terms of the amount of variance explained by the other psychological variables, standardised beta values across imputations suggested that the internal subscale of the MHLc ($\beta = .116$ to $.123$) and self-esteem ($\beta = -.090$ to $-.124$) predicted more variance in SA than self-efficacy ($\beta = -.050$ to $-.070$) and the chance subscale of the MHLc ($\beta = .047$ to $.061$). However, internal LoC and self-esteem were not statistically significant independent predictors of SA.

Discussion

Key findings

The present study examined psychological variables associated with SA following TBI. The overall regression model was significant, and the hypothesis that psychological variables would account for a significant proportion of the variance in SA was supported. Over half the sample (52.9%) showed clinically significant levels of SA, as defined using the cut-off provided by the scale author (Connor et al., 2000). This is substantially higher than both the estimated prevalence rate of 12% observed in the general population (NICE, 2013) and the rate of 30.6% found with a sample of people diagnosed with another chronic neurological condition, multiple sclerosis (Poder et al., 2013).

Before psychological variables were added to the regression model, severity of perceived cognitive problems and depression were significant predictors of greater levels of SA. Depression is often comorbid with SA in the general population (Ohayon, Schatzberg, 2010), with negative beliefs about the self and others central to cognitive understandings of both presentations. Additionally, it is understandable that people who perceive more severe levels of cognitive impairment might have more negative evaluations of themselves as social objects, thereby experiencing higher levels of social anxiety. This has been highlighted in qualitative research with people who have experienced TBI (Morris et al., 2005; Nochi, 1998); worry that other people will think they are slow or stupid has the potential to increase anxiety in social situations. Anxiety may also further reduce available attentional and cognitive processing capacity (which may already be decreased following TBI), thereby heightening and maintaining the problems experienced and the development of avoidance patterns. In this respect, perception of cognitive problems and low mood are clearly important clinical factors to consider in understanding the development and maintenance of SA.

The addition of psychological variables (MHLoC internal, MHLoC chance, self-esteem, self-efficacy, perceived stigma) made a significant additional contribution to the

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2
3 amount of variance explained, suggesting that psychological variables are important factors
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5 in the development of SA following TBI in addition to demographic and clinical variables.
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7 In the overall model (i.e., where the available variance was shared across a greater number of
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9 predictor variables), only perceived stigma was a significant independent predictor of SA. All
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11 other psychological variables explained some variance in SA, with internal LoC and self-
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13 esteem predicting a greater amount of variance than self-efficacy and chance LoC. Although
14
15 internal LoC and self-esteem did not reach statistical significance as independent predictors,
16
17 this may be due to the relatively small sample size employed in the study and further
18
19 examination is warranted. Nevertheless, when self-esteem, self-efficacy and LoC are
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21 combined with perceived stigma they explain a significant amount of variance in SA, above
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23 and beyond that explained by demographic and clinical factors such as depression and
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25 perceived cognitive problems. It should also be noted that adding these variables as the final
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27 block in the regression model provides a particularly rigorous and robust test of their
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29 predictive power.
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34 As outlined above, there is no previous research directly examining the role of
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36 psychological variables in the development of SA following TBI. However, the results are in
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38 keeping with theoretical and empirical understandings of psychological and psychosocial
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40 functioning following TBI. Indeed, there is growing consensus that psychological wellbeing
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42 and psychosocial functioning following TBI is influenced by a broad range of factors, with
43
44 psychological variables playing a key role alongside cognitive, neurological and demographic
45
46 factors (Soo et al., 2012; Moore et al., 2006; Kendall & Terry, 1996).
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49 Furthermore, the emergence of perceived stigma as a significant independent
50
51 predictor is a key finding. This offers support for Kendall and Terry's (1996) model of
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53 psychosocial functioning after TBI, in which perceived stigma is proposed as a key factor
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 affecting primary appraisal (i.e., how events are appraised), which subsequently affects
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5 secondary appraisal (i.e., a person's beliefs around how well they can cope with an event).
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7 This finding is also consistent with theoretical models highlighting how aversive social
8
9 experiences are a key factor in the development of SA (Rapee & Spence, 2004). The
10
11 cognitive model of SA, proposed by Clark and Wells (1995) and updated by Wells (2013),
12
13 proposes that social situations activate negative automatic thoughts based on assumptions
14
15 around perceived danger in social situations. Negative evaluations of how the self is
16
17 processed as a social object (i.e., how the person thinks they appear to others) are often
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19 inaccurate or exaggerated and can lead to safety behaviours (e.g., avoidance), which serve to
20
21 reinforce the beliefs (Wells, 2013). Safety behaviours maintain and exacerbate the problems
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23 by perpetuating the beliefs that social interactions will lead to negative outcomes (Clark &
24
25 Wells, 1995; Wells, 2013; Banerjee & Henderson, 2001). Since social experiences are key to
26
27 the development and maintenance of SA, it is consistent that perceived stigma would play a
28
29 key role in the development of SA. As discussed above, greater levels of perceived cognitive
30
31 problems and reduced mood are also likely to be important factors in the development of
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33 such problem cycles.
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39 These findings are also consistent with social models of disability, which highlight the
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41 need to focus on the societal context of impairment (Oliver, 1983; 2004). Instead of focusing
42
43 on the functional impairments of the individual, the social model considers disability to be
44
45 caused by the economic, cultural and environmental barriers which are faced by people with
46
47 physical or cognitive impairments. Consistent with the findings of the present study, Oliver
48
49 (2004) discusses how cultural norms around disability, which view impairment as
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51 unattractive and unwanted, negatively impact people by creating stigmatising, discriminatory
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53 environments which devalue and actively disable people with impairments, thereby causing
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55 psychological distress. Individualistic psychiatric or psychological approaches often fail to
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3 take this into account, instead conceptualising psychological problems as a consequence of
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5 the impairment itself and focusing on the need for people to seek treatment or adapt to the
6
7 disabling environment (Simpson & Thomas, 2014; Simpson, McMillan & Reeve, 2013).
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10 Moreover, people who develop impairments throughout their lives have been raised
11
12 within these cultural norms (Oliver, 2004). The term psychoemotional disablism refers to
13
14 how negative social interactions can lead to negative societal stereotypes about what it means
15
16 to have an impairment being internalised, which can limit the coping resources people have
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18 to draw on and lead to reduced participation in society (Reeve, 2012; Simpson et al., 2013).
19
20 Research has highlighted how stigma and poor understanding are key problems in relation to
21
22 TBI (e.g., Linden & Boylan, 2010; McClure, 2011; Guilmette & Paglia, 2004). In
23
24 emphasising the role of stigma in the development of SA following TBI, this study highlights
25
26 the importance of considering the societal and cultural factors influencing a person's
27
28 experience of impairment following TBI, guiding intervention at both an individual and
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30 social level.
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33 34 **Clinical implications**

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36 These findings have various implications for health professionals. It appears that SA
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38 is a problem following TBI and the application of cognitive models of SA to therapeutic
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40 work may be a useful way to conceptualise problems with psychosocial functioning
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42 following TBI. The clear role for psychological factors such as self-esteem, self-efficacy and
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44 LoC in the development of SA following TBI suggests a need to consider these variables
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46 during physical and cognitive assessment and rehabilitation, supporting the development of
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48 an individual's psychological resilience during the complex process of recovery from TBI.
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52 In particular, the significant role which stigma plays in the development of SA
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54 following TBI highlights the importance of developing contextually inclusive formulations
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56 (BPS, 2011) which explore the reactions people experience from others, in addition to the
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individual psychological factors which affect how responses of other people are perceived.

By considering the ways in which disability is constructed by the discriminatory social context faced by people who have experienced TBI and not focusing solely on the individual, interventions which challenge the lack of knowledge and negative attitudes around TBI (Linden & Boylan, 2010; McClure, 2011; Guilmette & Paglia, 2004) can begin to address the barriers, discrimination and stigma which are often imposed through entrenched societal and cultural norms (Simpson & Thomas, 2014; Oliver, 2004). Indeed, the precise nature of the stigma being experienced is important. For example, this study highlights the importance of perceived cognitive impairment; specific cognitive impairments following TBI may be misunderstood as a reduction in overall intellectual ability and functional independence. Educational programmes could highlight the difference between general intellectual ability and the types of cognitive problems that can be experienced after TBI, along with ways in which the individual and the people around them can reduce the impact these problems might have on their life.

Limitations and Implications for Future Research

It is recognised that the use of a self-selecting sample may have introduced some bias to the sample. The study also focused exclusively on people living in the community. A different pattern of results may be evident with a sample in the earlier stages of recovery and future research may be useful in exploring how different kinds of interactions with professionals at an early stage affect the development of SA. Moreover, this study focused on TBI to explore specific issues relating to this population. Further research which widens the scope of the study to include people with other kinds of acquired brain injuries may increase the generalisability of these findings to clinical practice.

Furthermore, the cross-sectional nature of the study limits the potential for understanding how SA and the other variables under examination may change over time.

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3 Consequently, future research which utilises a longitudinal or prospective design would be of
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5 value. In addition, the use of multiple regression in the current study assumes a linear
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7 relationship between variables. However, as psychological variables have been shown to play
8
9 a significant role in the development of SA, use of more advanced statistical techniques (e.g.,
10
11 structural equation modelling) would be useful next step following this study. For example,
12
13 the regression model suggests that perceived stigma is predictive of SA, however it is
14
15 possible that this is a bi-directional relationship and that people who are more anxious in
16
17 social situations are likely to be hyper-vigilant to threat, thereby perceiving higher levels of
18
19 stigmatising behaviour from others. Further research analysing hypothesised pathways
20
21 between factors will allow for a more detailed understanding of the complex bi-directional
22
23 interactions between predictor and outcome variables. This will be useful in guiding
24
25 intervention, in that targeting particular variables (e.g., self-esteem) in therapy may help to
26
27 reduce the amount of stigma which is perceived, mitigating its effect on SA.
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32 Furthermore, the lack of characterisation of the sample in terms of objective severity
33
34 of injury and cognitive impairment is a limitation of the study. Perceived severity of
35
36 cognitive problems may not be accurate and the self-selected sample may potentially result in
37
38 a less impaired group. However, injury variables and degree of cognitive impairment do not
39
40 fully account for variance in psychosocial adjustment following TBI (Antonak et al., 1993)
41
42 and appraisal of cognitive limitations has been shown to moderate the relationship between
43
44 injury severity and psychosocial function (Kervick & Kaemingk, 2005). Therefore, by
45
46 assessing subjective severity of cognitive problems using a self-report measure, the degree to
47
48 which an individual's appraisal of their cognitive problems can contribute to SA can be
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50 explored. Future research employing other methods of assessing neurological and cognitive
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52 variables would be useful, for example using neuropsychological assessments to assess
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54 impairments in specific cognitive domains, or consulting medical records to obtain specific
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3 details of TBI severity. Further examination of other relevant psychological variables such as
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5 appraisal and coping style would also be of value, given the relevance of such factors in
6
7 relation to wellbeing following TBI (Anson & Ponsford, 2006a; Kendall & Terry, 1996).
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9
10 The present study also did not explore situational factors in detail. Although living
11
12 alone and being in a relationship did not significantly correlate with SA in this study, future
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14 research might address environmental factors hypothesised to be of importance for
15
16 psychosocial wellbeing following TBI (Kendall & Terry, 1996). For example, social contact,
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18 family dynamics and perceptions of support from others might be important variables to
19
20 consider in the development of SA following TBI, particularly as social learning theories of
21
22 SA suggest that experience of aversive situations and lack of modelling of adaptive coping
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24 strategies for managing social situations are key to the development of SA (Rapee & Spence,
25
26 2004). Longitudinal research examining relationships post-TBI may be extremely useful in
27
28 understanding SA and psychosocial wellbeing more broadly.
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32 Even considering the limitations discussed above, the present study is the first to
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34 examine factors associated with SA following TBI. The findings of this study highlight the
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36 importance of considering SA in this population, particularly when considering rehabilitation
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38 adjustment following TBI. The significance of perceived stigma as a predictor of SA is an
39
40 important finding in this context, highlighting a clear role for clinical psychologists and other
41
42 rehabilitation professionals to integrate social models of disability into their practice and
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44 make a valued contribution to the psychological wellbeing of people who have experienced
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46 TBI.
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Conclusion

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50 The current study explored factors predicting SA following TBI. Hierarchical
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52 multiple regression was used to examine the extent to which demographic, clinical and
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54 psychological variables predicted scores on a measure of SA. Psychological variables,
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3 particularly perceived stigma, explained a significant proportion of the variance in SA.

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5 Therefore it is proposed that psychological variables are important factors affecting the
6
7 development of SA following TBI, above and beyond demographic and clinical variables.

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9 The study provides empirical support to the theoretical rationale for SA following TBI
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11 proposed by Soo et al. (2012) and Moore et al. (2006), highlighting the potential application
12
13 of Kendall and Terry's (1996) model for psychosocial adjustment. Further research is
14
15 required to examine the complex relationships between such variables using a more stable
16
17 regression model, and to explore in more detail other variables which may have an influence
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19 on SA using more advanced statistical techniques which allow for the examination of non-
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21 linear relationships.
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Social Anxiety Following Traumatic Brain Injury: An Exploration of Associated Factors

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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2

Abstract

Social anxiety (SA) following traumatic brain injury (TBI) has the potential to affect an individual's general psychological wellbeing and social functioning, however little research has explored factors associated with its development. The present study used hierarchical multiple regression to investigate the demographic, clinical and psychological factors associated with SA following TBI. A sample of 85 people who experienced TBI were recruited through social media websites and brain injury services across the NorthWest of England. The overall combined biopsychosocial model was significant, explaining 52-54.3% of the variance in SA (across five imputations of missing data). The addition of psychological variables (self-esteem, locus of control, self-efficacy) made a significant contribution to the overall model, accounting for an additional 12.2-13% of variance in SA above that explained by demographic and clinical variables. Perceived stigma was the only significant independent predictor of SA ($B = .274, p = .005$). The findings suggest that psychological variables are important in the development of SA following TBI and must be considered alongside clinical factors. Furthermore, the significant role of stigma highlights the need for intervention at both an individualised and societal level.

Keywords: traumatic brain injury, social anxiety, stigma, psychological

Social Anxiety Following Traumatic Brain Injury: An Exploration of Associated Factors

~~Traumatic brain injury (TBI), generally defined as a non-degenerative insult to the brain caused by an external mechanical force (e.g., from a road traffic accident or a fall), can lead to temporary or permanent impairment of brain function, affecting cognitive and physical abilities (World Health Organisation [WHO], 2006; Menon, Schwab, Wright, & Maas, 2010). Head injuries are the most common cause of death and impairment in people under 40 (National Institute for Health and Care Excellence [NICE], 2014; WHO, 2006).~~

People who have experienced a traumatic brain injury (TBI) are at increased risk of developing psychological difficulties such as depression and anxiety (Scholten et al., 2016; Osborn, Mathias, & Fairweather-Schmidt, 2014; Gould, Ponsford, Johnston and Schönberger, 2011; Whelan-Goodinson, Ponsford, Schönberger & Johnston, 2010; Bryant et al., 2010).

However, recognising psychological problems after TBI can be challenging, given the complex interactions between the neurological and emotional sequelae of TBI and the difficulties in identifying symptoms of psychological problems in the context of other factors (e.g., cognitive impairment, physical disability) associated with TBI (Kim et al., 2007; Scheutzwow & Wiercisiewski, 1999). Nonetheless, as psychological problems following TBI can be longstanding (Konrad et al., 2011) and may affect wellbeing and inhibit recovery (Osborn et al., 2014), it is imperative to improve understanding and management of these difficulties during assessment and rehabilitation (Williams, Evans & Fleminger, 2003).

Furthermore, it is vital to understand the social context in which TBI rehabilitation occurs. Social functioning is commonly affected by TBI and this can have a significant impact on life satisfaction (Pierce & Hanks, 2006; Truelle, Fayol, Montreuil, & Chevignard, 2010; Jones et al., 2010). Qualitative research highlights the importance of social activity following TBI in making sense of oneself (Yeates, Gracey, & Mcgrath, 2008), and social support is predictive of lower levels of post-traumatic stress (Jones et al., 2012). However,

SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 declines in activity, social contact, independence, functional status and employment
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5 opportunities are often reported following TBI (Antonak, Livneh, & Antonak, 1993; Temkin,
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7 Corrigan, Dikmen, & Machamer, 2009). Severity of injury fails to account fully for
8
9 differences in psychosocial functioning (Antonak et al., 1993) and life satisfaction post-TBI
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11 (Jones et al., 2010), with the latter study finding that social support mediated the relationship
12
13 between well-being and injury severity.
14

15
16 Moreover, following TBI people may feel embarrassed or self-conscious in social
17
18 situations given the frequency of physical consequences (e.g., physical impairment,
19
20 hemiparesis, skull depressions, scarring, tremors, motor/speech problems) and often unseen
21
22 cognitive problems with word finding, attention, memory, executive functioning and
23
24 processing speed (Rochat, Ammann, Mayer, Annoni, Van Der Linden, 2009; Hiott &
25
26 Labbate, 2002; Moore, Terryberry-Spohr & Hope, 2006). Therefore, social interaction can be
27
28 negatively impacted following TBI if a person is less able to follow or engage in conversation
29
30 (Morris et al., 2005). Consequently, problems following TBI may result in people becoming
31
32 particularly anxious in social situations (Moore et al., 2006; Wright & Telford, 1996).
33
34

35
36 However, despite the importance of social reintegration, social anxiety in people with
37
38 a TBI has been the subject of very little research. Social anxiety (SA) is characterised by a
39
40 marked fear of situations in which a person might face scrutiny from others and subsequent
41
42 avoidance of common triggers (e.g., social interactions, meeting new people, public
43
44 speaking) which can result in significant distress and impairments in functioning (National
45
46 Institute for Health and Care Excellence [NICE], 2013; American Psychiatric Association
47
48 [APA], 2013). While both anxiety (Rao & Lyketsos, 2002) and declines in psychosocial
49
50 functioning (Ponsford et al., 2014; Antonak et al., 1993) following TBI are well documented,
51
52 the available research examining SA following TBI is limited and of poor quality. Only two
53
54 studies have been identified which have assessed social anxiety in this population. A
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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1
2
3 prospective cohort study of people who had experienced traumatic injuries found that 6.1%
4
5 of people with mild-TBI met criteria for SA three months post-injury, rising to 9% after 12
6
7 months (Bryant et al., 2010). Conversely, Newton and Johnson (1985) found that SA was
8
9 lower in participants with a TBI compared to those without. However, the TBI group
10
11 comprised only eleven participants who exhibited a broad range of scores on a measure of
12
13 SA. The authors concluded that although the mean score was lower than the control group, a
14
15 high level of SA was observed in the majority of the TBI group ($n = 8$).
16
17

18
19 This lack of research interest may be a consequence of the complex interaction and
20
21 overlap between psychological and neurological problems as discussed above. It may also
22
23 result from the criteria within the Diagnostic and Statistical Manual of Mental Disorders,
24
25 Fifth Edition (DSM-5; APA, 2013) for SA which state that, if a medical condition is present,
26
27 anxiety or avoidance must be unrelated or out of proportion to it. This suggests that a
28
29 diagnostic label of social anxiety disorder may not be appropriate for people experiencing
30
31 anxiety in social situations after TBI. This may result in social anxiety not being considered
32
33 in this population, or such difficulties being attributed to the cognitive or neurological
34
35 consequences of TBI. However, this is not in keeping with recommendations for a broad and
36
37 biopsychosocial approach to providing support and rehabilitation following TBI (Gracey,
38
39 Evans & Malley, 2009; Wilson & Gracey, 2009).
40
41
42

43
44 No guidance is available specific to the management of SA after TBI, but empirically-
45
46 based guidance for generic SA interventions in the UK (NICE, 2013) recommends cognitive
47
48 behavioural therapy (CBT) as a first-line intervention (i.e., before pharmacological
49
50 interventions), underpinned by a specifically developed theoretical model (e.g., Clark &
51
52 Wells, 1995). However, a randomised controlled trial of a CBT programme for SA after
53
54 acquired brain injury (ABI) found that although SA did reduce, treatment effects were not
55
56 statistically significant (Hodgson, McDonald, Tate, & Gertler, 2012). However, a small
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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1
2
3 sample size ($n = 12$) and variability in the ABI group (people who had experienced stroke,
4
5 hypoxic brain injury and cerebral oedema were included alongside those who had
6
7 experienced TBI) limits the usefulness of this study in understanding management of SA
8
9 after TBI.

10
11
12 Despite the lack of research or guidance around SA after TBI, a literature review
13
14 exploring anxiety following mild TBI (Moore et al., 2006) highlighted the potential for SA to
15
16 be a significant problem in this population. Furthermore, Soo, Tate and Rapee (2012) present
17
18 a theoretical rationale for high levels of SA in children and adolescents who have
19
20 experienced TBI. They draw on Kendall and Terry's (1996) model for understanding
21
22 individual differences and predicting psychosocial adjustment outcomes following TBI,
23
24 acknowledging a role for direct (neurological and cognitive impairment) and indirect
25
26 (situational and environmental) antecedent factors, but also emphasising the importance of an
27
28 individual's psychological resources such as appraisal style and coping responses. This is
29
30 consistent with cognitive theories of SA (e.g., Clark & Wells, 1995; Wells, 2013) and
31
32 approaches to management of other anxiety problems following TBI (Williams et al., 2003;
33
34 Soo & Tate, 2009). Consequently, an understanding of SA following TBI in adults must be
35
36 guided by research which explores the role of potentially relevant neurological, cognitive,
37
38 situational and psychological factors to guide assessment, formulation and intervention
39
40 during acute and long-term rehabilitation.

41
42
43
44
45 A broad range of psychological variables may be important in SA following TBI (Soo
46
47 et al., 2012). Locus of control (LoC), the beliefs a person holds about how the behaviour of
48
49 themselves and others influences their health (Wallston, Stein, & Smith, 1994), has been
50
51 associated with SA (Cloutre, Heimberg, Liebowitz, & Gitow, 1992; Kennedy, Lynch, &
52
53 Schwab, 1998) and emotional problems in people who have experienced TBI (Moore &
54
55 Stambrook, 1992). Self-efficacy, the beliefs people hold about their capabilities is also
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1
2
3 associated with SA (Leary & Atherton, 1986) and is predictive of global life satisfaction
4
5 following TBI (Cicerone & Azulay, 2007). Low self-esteem is also linked to SA (Ritter,
6
7 Ertel, Beil, Steffens, & Stangier, 2013). Though debate continues around the consistency of
8
9 the construct, self-esteem is generally defined as the global, subjective and emotional
10
11 judgements one holds about the self (Guindon, 2002), which are activated and reinforced in
12
13 social situations and contribute to fear of negative evaluation (Wells, 2013; Clark & Wells,
14
15 1995; Rapee & Spence, 2004). People who have experienced TBI have been found to have
16
17 lower self-esteem (Ponsford, Kelly, & Couchman, 2014) and self-esteem has been shown to
18
19 predict psychosocial outcomes following TBI (Tate & Broe, 1999).
20
21

22
23 Furthermore, fear of negative evaluation may mean that people with SA perceive or
24
25 experience higher levels of stigma (Anderson, Jeon, Blenner, Wiener, & Hope, 2015; Clark
26
27 & Wells, 1995). People who are socially anxious may be rejected or perceived negatively,
28
29 particularly if anxiety related behaviours (e.g., gripping hands together, avoiding eye contact)
30
31 compound the anxiety symptoms or impair social performance (Wells, 2013; Rapee &
32
33 Spence, 2004). As highlighted above, the physical and cognitive consequences of TBI may
34
35 add further challenges to social interactions. Qualitative research has suggested stigma may
36
37 be a potential factor affecting wellbeing following TBI, with participants highlighting the
38
39 lack of public understanding about the consequences of TBI and how this impacts on their
40
41 social engagement (Morris et al., 2005; Nochi, 1998). Furthermore, perceived stigma is
42
43 strongly associated with anxiety in people with chronic physical conditions (Alonso et al.,
44
45 2008) and epilepsy (Beyenburg, Mitchell, Schmidt, Elger, & Reuber, 2005).
46
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48

49
50 In conclusion, despite the theoretical rationale for SA following TBI presented by Soo
51
52 et al. (2012) and Moore et al., (2006), present understanding of SA following TBI is limited
53
54 given the limited available research. No research to date has explored psychological factors
55
56 which might contribute to the development of SA following TBI to provide guidance for
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8

assessment and intervention. While it is recognised that psychological problems may predate a brain injury (Williams et al., 2003), people who have experienced TBI may be at greater risk of developing SA due to the nature of the factors described above. Consequently, the present study aimed to investigate psychological factors associated with SA following TBI, alongside clinical and demographic variables. It was hypothesised that psychological variables such as LoC, self-efficacy, self-esteem and perceived stigma would account for an additional and significant amount of variance in SA, above that explained by demographic and clinical variables.

Methods

Design

The study employed a quantitative, cross-sectional within-subjects design to explore factors predicting SA after TBI. Self-report questionnaires were used as the data collection method. If required, participants were given support from the lead researcher to complete the questionnaires.

Participants

Participants were required to have sustained a TBI, defined as an injury caused by an external or mechanical force (Morton & Wehman, 1995), to differentiate from the broader categorisation of ABI. Participants in the study were required to be aged over 18 and able to read English (due to lack of the validated measures in other languages). ~~As the research~~

~~literature regarding the developmental impact of TBI in childhood is scarce (Barlow, Thompson, Johnson, & Minns, 2004);~~

~~participants~~ Participants were required to have sustained a TBI after the age of 16 **to allow for specific examination of factors in relation to adults, as other developmental factors are likely to influence cognitive and psychological outcomes following TBI experienced in childhood or adolescence (Anderson et al., 2006; Catroppa, Anderson, Morse, Hariou, & Rosenfeld,**

2008). Given the focus on social functioning, participants were required to be living in the community (either at home or in long-term supported accommodation) rather than a medical ward or residential rehabilitation unit. Participants were also required to have capacity to consent to participation in the study.

An a priori power calculation for multiple regression analysis, assuming a medium effect size of 0.15, 80% power and an alpha level set at $p = .05$, suggested that a sample of between 92 and 139 would be required. A total of 98 participants were recruited, with 54 participants completing the questionnaires online and 44 submitting paper copies provided via National Health Service (NHS) or third sector services. Five participants who completed the study online were excluded from the analysis as they described their injury as an ABI (e.g., subarachnoid haemorrhage) rather than a TBI and therefore did not meet all the inclusion criteria. A further eight participants were excluded as a significant amount of questionnaire data (more than 10%, as recommended by Bennett, 2001) were missing.

Therefore, a total of 85 participants provided data for the analyses. Participants ranged in age from 19 to 81 years ($M = 42.4$, $SD = 13.335$). The final sample included 63.5% ($n = 54$) males and 32.9% ($n = 28$) females, with 3.5% ($n = 3$) reporting “Other / Prefer not to say”. Further demographic information is shown in Table 1.

[INSERT TABLE 1 HERE]

Due to ethical and resource constraints, medical data regarding severity of injury were not available. Participants were asked to report the length of time they were in hospital for after their injury ($M = 16.529$ weeks, $SD = 32.120$) and time since injury ($M = 7.719$ years, $SD = 8.733$).

Measures

Outcome variable. The Social Phobia Inventory (SPIN; Connor et al., 2000) was used as the outcome measure for the study. The SPIN is a 17-item self-report measure of

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1
2
3 three domains of SA; fear, avoidance and physiological discomfort. Responses are scored
4
5 from 0 (not at all) to 4 (extremely), with a maximum total score of 68 indicating high levels
6
7 of SA. A cut-off score of 19 is recommended by the authors to distinguish those with SA.
8
9 High levels of internal consistency ($\alpha = .95$) and test-retest reliability ($r = .86$) have been
10
11 demonstrated (Antony, Coons, McCabe, Ashbaugh, & Swinson, 2006; Connor et al., 2000).
12
13 Although the measure has not been used in a TBI population in any published research to
14
15 date, it has been utilised with patients with multiple sclerosis (Poder et al., 2009) and is
16
17 recommended by guidance provided by NICE (2013) for use in NHS services within the UK.
18
19 The SPIN's face validity and brevity make it the most appropriate measure from available
20
21 measures of SA.
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24

25
26 **Predictor variables.** The Applied Cognition measure (Neuro-QOL, 2012) was used
27
28 to assess subjective severity of cognitive problems. This 18-item measure assesses perceived
29
30 difficulties in everyday cognitive domains including memory, attention, and decision-
31
32 making. Responses range from never (1) to very often (5), with a maximum score of 90. High
33
34 levels of internal consistency ($\alpha = .95$) and test-retest reliability ($r = .82$) have been
35
36 demonstrated in samples of patients with a range of neurological problems (e.g., stroke,
37
38 epilepsy, Parkinson's disease) but data are not available for a TBI sample (Neuro-QOL,
39
40 2010).
41
42

43 Form C of the Multidimensional Health Locus of Control (MHLc, Wallston, Stein,
44
45 & Smith, 1994) assesses belief in one's ability to control health outcomes, in relation to a
46
47 specific illness or disease. The measure encompasses four subscales of LoC: internal; chance;
48
49 powerful others (doctors) and powerful others (other people). Responses are scored from 1
50
51 (strongly disagree) to 6 (strongly agree), with a higher subscale score indicating higher LoC
52
53 (no total score is calculated). Wallston et al. (1994) demonstrated acceptable levels of internal
54
55 consistency and test-retest reliability for each subscale; internal ($\alpha = .79 - .87$; $r = .80$),
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1
2
3 chance ($\alpha = .79 - .82$; $r = .72$), doctors ($\alpha = .71$; $r = .58$) and other people ($\alpha = .70 - .71$; $r =$
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.40). Despite its focus on control over one's specific illness or disease (Wallston, 2005), no published research has used Form C with a TBI population. However, Forms A and B of the MHLc have been used in previous TBI research (Bedard et al., 2005; Moore & Stambrook, 1992), and Form C has been used to assess LoC following spinal cord injury (Waldron et al., 2010).

The Rosenberg Self-Esteem Scale (RSES, 1965) is a 10-item measure, with responses recorded on a 0 to 3 scale (reverse coded on some items) so that a low score on the RSES indicates low self-esteem. The RSE demonstrates high internal consistency ($\alpha = .92$), and test-retest reliability ($r = .85$) after two weeks (Rosenberg, 1979). This measure has been used to examine self-esteem in people who have experienced a TBI (e.g., Anson & Ponsford, 2006a; Anson & Ponsford, 2006b; Ponsford et al., 2014).

The Self-Efficacy for Symptom Management Scale (Cicerone & Azulay, 2007) assesses confidence in managing common challenges and seeking support after TBI. The 13-items measure is scored 1 (not at all confident) to 10 (totally confident), with a maximum total score of 130 indicating high self-efficacy. High levels of internal consistency ($\alpha = .93$) and test-retest reliability ($r = .93$) have been demonstrated (Cicerone & Azulay, 2007).

The Stigma scale published by Neuro-QOL (2012) is a 24-item measure which examines a person's perceptions of self and publically enacted prejudice and discrimination experienced as a result of neurological problems. Responses are scored from 1 (never) to 5 (always), with a maximum score of 120 indicating high levels of perceived stigma. High levels of internal reliability ($\alpha = .91$) and test-retest reliability ($r = .82$) have been demonstrated in samples of patients with a range of neurological problems (e.g., stroke, epilepsy, Parkinson's disease) but no data are available for a TBI sample (Rao et al., 2009).

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1
2
3 For the purposes of the study, the word 'illness' was replaced with the term 'brain injury' on
4
5 each item of the questionnaire.
6

7
8 The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was
9
10 designed for use with people with physical health problems and assesses anxiety and
11
12 depression without relying on somatic symptoms of illness (e.g., fatigue, insomnia). The 14-
13
14 item measure is scored on a 0 to 3 scale, appropriately coded so that a higher score on each
15
16 subscale indicates a more severe problem with anxiety or depression. A review of its
17
18 psychometric properties reports good levels of internal consistency on the anxiety ($\alpha = .68 -$
19
20 $.93$) and depression ($\alpha = .67 - .9$) subscales across a variety of settings (Bjelland, Dahl, Haug,
21
22 & Neckelmann, 2002), with similar findings reported by Whelan-Goodinson, Ponsford and
23
24 Schönberger (2009) with a TBI sample (depression $\alpha = .88$; anxiety $\alpha = .92$). The HADS has
25
26 been used to measure depression and anxiety after TBI in a number of published studies (e.g.,
27
28 Anson & Ponsford, 2006a; Anson & Ponsford, 2006b; Downing, Stolwyk, & Ponsford,
29
30 2013).
31
32

Procedure

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36 Potential participants were identified and recruited through professionals working in
37
38 neuropsychology teams across nine NHS Trusts in the North-West of England and third
39
40 sector organisations relevant to TBI. Participants were also able to self-refer into the study
41
42 and could opt to complete an online version of the study made using Qualtrics Survey
43
44 Software (Qualtrics, 2013), which provided security and encryption for online information.
45
46 The study was advertised via social networking websites and posters displayed in NHS
47
48 neuropsychology services and third sector organisations.
49
50

51
52 Prior to completing the questionnaires, participants were required to complete a
53
54 screening and consent form based on the inclusion and exclusion criteria outlined above. On
55
56 the online version of the study, participants were only able to progress onto the
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1
2
3 questionnaires if they answered each item of the consent form. Capacity to consent and
4
5 participate in the study was assumed in line with the UK Mental Capacity Act (2005).
6
7 Participants had the option of completing the questionnaires online or on paper posting them
8
9 to the lead researcher. To reduce bias, the online study was set to present questionnaires in a
10
11 random order.
12

13 14 **Ethical Approval**

15
16 The study received ethical approval from the UK NHS National Research Ethics
17
18 Service, followed by local approval from the Research and Development Departments of
19
20 each NHS Trust involved in recruitment. This approval also covered participants recruited
21
22 through third sector organisations and online.
23

24 25 **Data Analysis Strategy**

26
27 Data were analysed using IBM SPSS Statistics version 20. All questionnaires were
28
29 scored in accordance with scale instructions and reverse coded as required. Relationship
30
31 status was recoded to a binary variable (i.e., yes / no). Due to its descriptive nature, cause of
32
33 injury was not entered into the regression model. Anxiety (measured by HADS) was not
34
35 entered into the regression model as it correlated too highly with the outcome variable ($r =$
36
37 $.726, p < .001$) and, as it is conceptually similar, would have reduced the variance available to
38
39 other variables. Additionally, depression was considered a clinical variable rather than a
40
41 psychological one, due to the focus of the HADS on measuring clinical difficulties associated
42
43 with depression.
44
45

46
47 Throughout the study, a p value of .05 was used as a threshold for statistical
48
49 significance in line with convention (Field, 2013). Furthermore, the decision was taken not to
50
51 use Bonferroni corrections to counteract multiple comparisons as this would have resulted in
52
53 a very low p value and significantly reduced statistical power.
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Hierarchical multiple regression analysis was used to explore the study hypothesis. Variables were entered into the model in three blocks; demographic, clinical, psychological. Consistent with the available theoretical rationale for SA following TBI discussed above, this allowed for examination of the amount of variance in SA which could be explained by psychological variables, above that explained by demographic and clinical variables.

In determining what variables were entered into the regression model, decisions for subset selection were made based on effect size instead of p values. While use of p values is common, effect sizes are less reliant on sample size (Coe, 2002). Given the relatively low sample size in this study ($n = 85$), variables were included in the multiple regression analysis if a small effect size was observed (i.e., $r > .1$; Cohen, 1988). This threshold was chosen to allow an inclusive, exploratory approach which minimised the risk of overlooking emerging effects of small magnitude (Hemphill, 2003).

Results

Data Preparation and Analysis

It did not appear that there were any systematic biases or patterns to the missing data as defined by Graham (2009), with 34 cases (40% of the sample) having incomplete data across 42 (34.43%) of the variables. Little's (1988) Missing Completely At Random (MCAR) test was not significant ($X^2 = 1921.880$, $df = 3105$, $p = 1.000$), suggesting that the null hypothesis of data being missing randomly could be assumed.

Even after removing the eight cases missing more than 10% of data, the number of other cases missing smaller amounts of data was high. Listwise or pairwise deletion methods were not considered appropriate as this would have seen a large proportion of cases deleted, thereby reducing sample size and power in addition to potentially introducing bias into the multiple regression model. Consequently, multiple imputation was conducted with the data

provided by 85 participants to analyse missing data and input substituted values (Rubin, 1987; Schaffer, 1997). Five iterations of imputation were performed (Schaffer, 1997).

Clinical Characteristics of Sample

Descriptive statistics for all self-report measures used in the study are provided in Table 2. As can be seen in Table 2, all measures demonstrated acceptable levels of internal consistency ($\alpha > .6$; Hair, Anderson, Tatham & Black, 2006).

[INSERT TABLE 2 HERE]

Independent samples t-test showed no significant difference on SPIN scores between participants who completed the questionnaire online compared to those who did not ($t(91) = .635, p = .527$). Using the cut-off scores for social anxiety as recommended by the authors of the SPIN (Connor et al, 2000), most participant scores (47.1%) lay in the 'None' category (> 20). A further 15 participants (17.6%) scored within the 'Mild' category, 13 (15.3%) scored within the 'Moderate' category, 10 (11.8%) scored in the 'Severe' category, and 7 (8.2%) participants were categorised as 'Very Severe'. Using the cut-offs provided by the scale authors (Zigmond & Snaith, 1983), 70.6% of the sample showed clinically significant levels of anxiety (with 21.2% in the severe category) while 63.5% of the sample showed clinically significant levels of depression (with 20% in the severe category).

Correlational Analysis

Correlational analysis (Pearson's r) was conducted on the pooled dataset comprising of all iterations of the multiple imputation process (Rubin, 1987). Correlations are shown in Tables 3 and 4.

[INSERT TABLE 3 & 4 HERE]

The following variables correlated significantly ($p < .05$) with higher SA scores on the SPIN: not being employed ($r = .239, p = .028$); higher levels of cognitive problems ($r = .476, p < .001$); higher levels of internal ($r = .248, p = .022$) and chance ($r = .217, p = .046$)

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3 LOC; lower self-esteem ($r = -.441, p < .001$); lower self-efficacy ($r = -.472, p < .001$); higher
4
5 perceived stigma ($r = .654, p < .001$); higher levels of anxiety ($r = .726, p < .001$) and higher
6
7 levels of depression ($r = .516, p < .001$). Age, gender, time since TBI, time in hospital, living
8
9 alone, relationship status and the two Powerful Others subscales of the MHLoC (Doctors and
10
11 Others) did not significantly correlate with SA scores.

Hierarchical Multiple Regression Analysis

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14
15
16 Hierarchical multiple regression analysis was conducted to examine if the predictor
17
18 variables were able to explain the variance in SA scores. Predictor variables which correlated
19
20 with SA demonstrating a small effect size or above (Pearson's $r > 0.1$) were entered into the
21
22 regression model. Predictor variables were entered into the regression model in three blocks:
23
24 (a) demographic variables (gender, employment status); (b) clinical variables (time since
25
26 TBI, perceived cognitive problems, depression); (c) psychological variables (MHLoC
27
28 internal, MHLoC chance, self-esteem, self-efficacy, perceived stigma).
29
30

31
32 The overall model was significant, both with the original dataset ($F(2, 63) = 5.918, p$
33
34 $< .001$, explaining 51.8% ($R^2 = .518, R^2_{adj} = .431$) of the variance in SA scores and across all
35
36 five imputations of missing data, with $F(2, 82)$ values ranging from 8.006 to 8.799, with all
37
38 values of $p < .001$. The amount of variance in SA scores explained ranged from 52% ($R^2 =$
39
40 $.520, R^2_{adj} = .455$) to 54.3% ($R^2 = .543, R^2_{adj} = .481$) of the variance in SA scores.
41
42

43 The Durbin-Watson values across the imputations ranged from 1.962 to 2.000
44
45 compared to the value from the original data of 1.846, and therefore it was assumed there was
46
47 no autocorrelation of residuals (Field, 2013). Examination of the VIF, tolerance and
48
49 eigenvalues confirmed that there was no evidence of collinearity within the dataset
50
51 (Bowerman & O'Connell, 1990; Menard, 1995; Field, 2013). Graphical representation of the
52
53 data suggested that assumptions of homoscedasticity and normally distributed residuals could
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55 be upheld.
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 Block one (demographic variables) accounted for 10.3% ($R^2 = .103$, $R^2_{adj} = .074$, $p =$
4
5 .033) of the variance in SA scores in the original dataset, rising to between 11.9% ($R^2 = .119$,
6
7 $R^2_{adj} = .097$, $p = .006$) and 14.7% ($R^2 = .147$, $R^2_{adj} = .126$, $p = .001$) following imputation. The
8
9 addition of block two (clinical variables) made a significant contribution to the model,
10
11 increasing the total variance explained to 36.1% ($\Delta R^2 = .259$, $p < .001$) for the original
12
13 dataset and between 39.8% ($\Delta R^2 = .279$, $p < .001$) and 41.3% ($\Delta R^2 = .280$, $p < .001$)
14
15 following imputation, with significant changes in F ($p < .001$) for both original and imputed
16
17 data. Within this block of variables, standardised beta values across imputations indicated
18
19 that higher levels of perceived cognitive problems ($\beta = .249$ to $.253$, $p = .012$) and depression
20
21 ($\beta = .348$ to $.367$, $p < .001$) were significant independent predictors of higher reported SA,
22
23 with time since injury not statistically significant ($\beta = .055$ to $.064$, $p = .516$).
24
25
26
27

28 The addition of block three (psychological variables) also made a significant
29
30 contribution to the overall model, explaining an additional 15.7% ($\Delta R^2 = .157$, $p < .001$) of
31
32 the total variance for the original dataset and between 12.2% ($\Delta R^2 = .122$, $p < .001$) and 13%
33
34 ($\Delta R^2 = .130$, $p < .001$) for each imputation. The change in F associated with the addition of
35
36 block three was statistically significant for both original ($p = .007$) and imputed data ($p =$
37
38 .002 to .004).
39
40
41

42 For individual predictors of SA, the overall model including all three blocks (and
43
44 based on data pooled from all imputations) indicated that only higher levels of perceived
45
46 stigma significantly predicted higher levels of SA ($B = .274$, $\beta = .334$ to $.341$, $t = 2.789$, $p =$
47
48 .005). In the final model, reported cognitive problems and depression ceased to meet criteria
49
50 for statistical significance. In terms of the amount of variance explained by the other
51
52 psychological variables, standardised beta values across imputations suggested that the
53
54 internal subscale of the MHLc ($\beta = .116$ to $.123$) and self-esteem ($\beta = -.090$ to $-.124$)
55
56 predicted more variance in SA than self-efficacy ($\beta = -.050$ to $-.070$) and the chance subscale
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59
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of the MHLoC ($\beta = .047$ to $.061$). However, internal LoC and self-esteem were not statistically significant independent predictors of SA.

Discussion

Key findings

The present study examined psychological variables associated with SA following TBI. The overall regression model was significant, and the hypothesis that psychological variables would account for a significant proportion of the variance in SA was supported. ~~The overall regression model was significant and the addition of psychological variables (MHLoC internal, MHLoC chance, self-esteem, self-efficacy, perceived stigma) made a significant additional contribution to the amount of variance explained, suggesting that psychological variables are important factors in the development of SA following TBI in addition to demographic and clinical variables.~~ Over half the sample (52.9%) showed clinically significant levels of SA, as defined using the cut-off provided by the scale author (Connor et al., 2000). This is substantially higher than both the estimated prevalence rate of 12% observed in the general population (NICE, 2013) and the rate of 30.6% found with a sample of people diagnosed with another chronic neurological condition, multiple sclerosis (Poder et al., 2013).

Before psychological variables were added to the regression model, severity of perceived cognitive problems and depression were significant predictors of greater levels of SA. Depression is often comorbid with SA in the general population (Ohayon, Schatzberg, 2010), with negative beliefs about the self and others central to cognitive understandings of both presentations. Additionally, it is understandable that people who perceive more severe levels of cognitive impairment might have more negative evaluations of themselves as social objects, thereby experiencing higher levels of social anxiety. This has been highlighted in qualitative research with people who have experienced TBI (Morris et al., 2005; Nochi,

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3 1998); worry that other people will think they are slow or stupid has the potential to increase
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5 anxiety in social situations. Anxiety may also further reduce available attentional and
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7 cognitive processing capacity (which may already be decreased following TBI), thereby
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9 heightening and maintaining the problems experienced and the development of avoidance
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11 patterns. In this respect, perception of cognitive problems and low mood are clearly important
12
13 clinical factors to consider in understanding the development and maintenance of SA.
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16 The addition of psychological variables (MHL0C internal, MHL0C chance, self-
17 esteem, self-efficacy, perceived stigma) made a significant additional contribution to the
18 amount of variance explained, suggesting that psychological variables are important factors
19 in the development of SA following TBI in addition to demographic and clinical variables.
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23 In the overall model (i.e., where the available variance was shared across a greater number of
24
25 predictor variables), only perceived stigma was a significant independent predictor of SA. All
26
27 other psychological variables explained some variance in SA, with internal LoC and self-
28
29 esteem predicting a greater amount of variance than self-efficacy and chance LoC. Although
30
31 internal LoC and self-esteem did not reach statistical significance as independent predictors,
32
33 this may be due to the relatively small sample size employed in the study and further
34
35 examination is warranted. Nevertheless, when self-esteem, self-efficacy and LoC are
36
37 combined with perceived stigma they explain a significant amount of variance in SA, above
38
39 and beyond that explained by demographic and clinical factors such as depression and
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41 perceived cognitive problems. It should also be noted that adding these variables as the final
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43 block in the regression model provides a particularly rigorous and robust test of their
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45 predictive power.
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52 As outlined above, there is no previous research directly examining the role of
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54 psychological variables in the development of SA following TBI. However, the results are in
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56 keeping with theoretical and empirical understandings of psychological and psychosocial
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 functioning following TBI. Indeed, there is growing consensus that psychological wellbeing
4
5 and psychosocial functioning following TBI is influenced by a broad range of factors, with
6
7 psychological variables playing a key role alongside cognitive, neurological and demographic
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9 factors (Soo et al., 2012; Moore et al., 2006; Kendall & Terry, 1996).
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11
12 Furthermore, the emergence of perceived stigma as a significant independent
13
14 predictor is a key finding. This offers support for Kendall and Terry's (1996) model of
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16 psychosocial functioning after TBI, in which perceived stigma is proposed as a key factor
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18 affecting primary appraisal (i.e., how events are appraised), which subsequently affects
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20 secondary appraisal (i.e., a person's beliefs around how well they can cope with an event).
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22 This finding is also consistent with theoretical models highlighting how aversive social
23
24 experiences are a key factor in the development of SA (Rapee & Spence, 2004). The
25
26 cognitive model of SA, proposed by Clark and Wells (1995) and updated by Wells (2013),
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28 proposes that social situations activate negative automatic thoughts based on assumptions
29
30 around perceived danger in social situations. Negative evaluations of how the self is
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32 processed as a social object (i.e., how the person thinks they appear to others) are often
33
34 inaccurate or exaggerated and can lead to safety behaviours (e.g., avoidance), which serve to
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36 reinforce the beliefs (Wells, 2013). Safety behaviours maintain and exacerbate the problems
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38 by perpetuating the beliefs that social interactions will lead to negative outcomes (Clark &
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40 Wells, 1995; Wells, 2013; Banerjee & Henderson, 2001). Since social experiences are key to
41
42 the development and maintenance of SA, it is consistent that perceived stigma would play a
43
44 key role in the development of SA. As discussed above, greater levels of perceived cognitive
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46 problems and reduced mood are also likely to be important factors in the development of
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48 such problem cycles.
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54 These findings are also consistent with social models of disability, which highlight the
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56 need to focus on the societal context of impairment (Oliver, 1983; 2004). Instead of focusing
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3 on the functional impairments of the individual, the social model considers disability to be
4 caused by the economic, cultural and environmental barriers which are faced by people with
5 physical or cognitive impairments. Consistent with the findings of the present study, Oliver
6 (2004) discusses how cultural norms around disability, which view impairment as
7 unattractive and unwanted, negatively impact people by creating stigmatising, discriminatory
8 environments which devalue and actively disable people with impairments, thereby causing
9 psychological distress. Individualistic psychiatric or psychological approaches often fail to
10 take this into account, instead conceptualising psychological problems as a consequence of
11 the impairment itself and focusing on the need for people to seek treatment or adapt to the
12 disabling environment (Simpson & Thomas, 2014; Simpson, McMillan & Reeve, 2013).

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25 Moreover, people who develop impairments throughout their lives have been raised
26 within these cultural norms (Oliver, 2004). The term psychoemotional disablism refers to
27 how negative social interactions can lead to negative societal stereotypes about what it means
28 to have an impairment being internalised, which can limit the coping resources people have
29 to draw on and lead to reduced participation in society (Reeve, 2012; Simpson et al., 2013).
30 Research has highlighted how stigma and poor understanding are key problems in relation to
31 TBI (e.g., Linden & Boylan, 2010; McClure, 2011; Guilmette & Paglia, 2004). In
32 emphasising the role of stigma in the development of SA following TBI, this study highlights
33 the importance of considering the societal and cultural factors influencing a person's
34 experience of impairment following TBI, guiding intervention at both an individual and
35 social level.
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49 **Clinical implications**

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52 These findings have various implications for health professionals. It appears that SA
53 is a problem following TBI and the application of cognitive models of SA to therapeutic
54 work may be a useful way to conceptualise problems with psychosocial functioning
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 following TBI. The clear role for psychological factors such as self-esteem, self-efficacy and
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5 LoC in the development of SA following TBI suggests a need to consider these variables
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7 during physical and cognitive assessment and rehabilitation, supporting the development of
8
9 an individual's psychological resilience during the complex process of recovery from TBI.
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12 In particular, the significant role which stigma plays in the development of SA
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14 following TBI highlights the importance of developing contextually inclusive formulations
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16 (BPS, 2011) which explore the reactions people experience from others, in addition to the
17
18 individual psychological factors which affect how responses of other people are perceived.
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20 By considering the ways in which disability is constructed by the discriminatory social
21
22 context faced by people who have experienced TBI and not focusing solely on the individual,
23
24 interventions which challenge the lack of knowledge and negative attitudes around TBI
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26 (Linden & Boylan, 2010; McClure, 2011; Guilmette & Paglia, 2004) can begin to address the
27
28 barriers, discrimination and stigma which are often imposed through entrenched societal and
29
30 cultural norms (Simpson & Thomas, 2014; Oliver, 2004). Indeed, the precise nature of the
31
32 stigma being experienced is important. For example, this study highlights the importance of
33
34 perceived cognitive impairment; specific cognitive impairments following TBI may be
35
36 misunderstood as a reduction in overall intellectual ability and functional independence.
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38 Educational programmes could highlight the difference between general intellectual ability
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40 and the types of cognitive problems that can be experienced after TBI, along with ways in
41
42 which the individual and the people around them can reduce the impact these problems might
43
44 have on their life.
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49 Limitations and Implications for Future Research

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51 It is recognised that the use of a self-selecting sample may have introduced some bias
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53 to the sample. The study also focused exclusively on people living in the community. A
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55 different pattern of results may be evident with a sample in the earlier stages of recovery and
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3 future research may be useful in exploring how different kinds of interactions with
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5 professionals at an early stage affect the development of SA. Moreover, this study focused on
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7 TBI to explore specific issues relating to this population. Further research which widens the
8
9 scope of the study to include people with other kinds of acquired brain injuries may increase
10
11 the generalisability of these findings to clinical practice.
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14 Furthermore, the cross-sectional nature of the study limits the potential for
15
16 understanding how SA and the other variables under examination may change over time.
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18 Consequently, future research which utilises a longitudinal or prospective design would be of
19
20 value. In addition, the use of multiple regression in the current study assumes a linear
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22 relationship between variables. However, as psychological variables have been shown to play
23
24 a significant role in the development of SA, use of more advanced statistical techniques (e.g.,
25
26 structural equation modelling) would be useful next step following this study. For example,
27
28 the regression model suggests that perceived stigma is predictive of SA, however it is
29
30 possible that this is a bi-directional relationship and that people who are more anxious in
31
32 social situations are likely to be hyper-vigilant to threat, thereby perceiving higher levels of
33
34 stigmatising behaviour from others. Further research analysing hypothesised pathways
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36 between factors will allow for a more detailed understanding of the complex bi-directional
37
38 interactions between predictor and outcome variables. This will be useful in guiding
39
40 intervention, in that targeting particular variables (e.g., self-esteem) in therapy may help to
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42 reduce the amount of stigma which is perceived, mitigating its effect on SA.
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48 Furthermore, the lack of characterisation of the sample in terms of objective severity
49
50 of injury and cognitive impairment is a limitation of the study. Perceived severity of
51
52 cognitive problems may not be accurate and the self-selected sample may potentially result in
53
54 a less impaired group. However, injury variables and degree of cognitive impairment do not
55
56 fully account for variance in psychosocial adjustment following TBI (Antonak et al., 1993)
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SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 and appraisal of cognitive limitations has been shown to moderate the relationship between
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5 injury severity and psychosocial function (Kervick & Kaemingk, 2005). Therefore, by
6
7 assessing subjective severity of cognitive problems using a self-report measure, the degree to
8
9 which an individual's appraisal of their cognitive problems can contribute to SA can be
10
11 explored. Future research employing other methods of assessing neurological and cognitive
12
13 variables would be useful, for example using neuropsychological assessments to assess
14
15 impairments in specific cognitive domains, or consulting medical records to obtain specific
16
17 details of TBI severity. Further examination of other relevant psychological variables such as
18
19 appraisal and coping style would also be of value, given the relevance of such factors in
20
21 relation to wellbeing following TBI (Anson & Ponsford, 2006a; Kendall & Terry, 1996).
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25 The present study also did not explore situational factors in detail. Although living
26
27 alone and being in a relationship did not significantly correlate with SA in this study, future
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29 research might address environmental factors hypothesised to be of importance for
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31 psychosocial wellbeing following TBI (Kendall & Terry, 1996). For example, social contact,
32
33 family dynamics and perceptions of support from others might be important variables to
34
35 consider in the development of SA following TBI, particularly as social learning theories of
36
37 SA suggest that experience of aversive situations and lack of modelling of adaptive coping
38
39 strategies for managing social situations are key to the development of SA (Rapee & Spence,
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41 2004). Longitudinal research examining relationships post-TBI may be extremely useful in
42
43 understanding SA and psychosocial wellbeing more broadly.
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47 Even considering the limitations discussed above, the present study is the first to
48
49 examine factors associated with SA following TBI. The findings of this study highlight the
50
51 importance of considering SA in this population, particularly when considering rehabilitation
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53 adjustment following TBI. The significance of perceived stigma as a predictor of SA is an
54
55 important finding in this context, highlighting a clear role for clinical psychologists and other
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3 rehabilitation professionals to integrate social models of disability into their practice and
4
5 make a valued contribution to the psychological wellbeing of people who have experienced
6
7 TBI.
8

9 10 **Conclusion**

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12 The current study explored factors predicting SA following TBI. Hierarchical
13
14 multiple regression was used to examine the extent to which demographic, clinical and
15
16 psychological variables predicted scores on a measure of SA. Psychological variables,
17
18 particularly perceived stigma, explained a significant proportion of the variance in SA.
19
20 Therefore it is proposed that psychological variables are important factors affecting the
21
22 development of SA following TBI, above and beyond demographic and clinical variables.
23
24 The study provides empirical support to the theoretical rationale for SA following TBI
25
26 proposed by Soo et al. (2012) and Moore et al. (2006), highlighting the potential application
27
28 of Kendall and Terry's (1996) model for psychosocial adjustment. Further research is
29
30 required to examine the complex relationships between such variables using a more stable
31
32 regression model, and to explore in more detail other variables which may have an influence
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34 on SA using more advanced statistical techniques which allow for the examination of non-
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36 linear relationships.
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Table 1.

Demographic characteristics (N = 85)

	n	%	Mean (SD)	Range
Gender				
Male	54	63.5%		
Female	28	32.9%		
Other / prefer not to say	3	3.5%		
Age			42.4 (13.34)	19 - 81
Cause of injury				
Road traffic accident	36	42.4%		
Assault	11	12.9%		
Sport injury	4	4.7%		
Work injury	6	7.1%		
Trip / fall	23	27.1%		
Other	3	3.5%		
Prefer not to say	2	2.4%		
Time since injury			7.72 years (8.73)	0.37 - 33
Time spent in hospital			16.53 weeks (32.12)	0 - 208
Employed				
Yes	27	31.8%		
No	57	67.1%		
Prefer not to say	1	1.2%		
Live alone				
Yes	25	29.4%		
No	59	69.4%		
Prefer not to say	1	1.2%		
Relationship status				
Single	28	32.9%		
In a relationship	44	51.8%		
Separated / divorced	12	14.1%		
Other / prefer not to say	1	1.2%		
Recruitment method				
Online	54	55.1%		

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NHS / third sector	44	44.9%
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Note. All data were collected via self-report.

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2-41

Table 2.

Clinical characteristics of sample

	Mean (SD)	Range	n (%)	α
<u>Social Phobia Inventory (SPIN)</u>				
Total	25.67 (16.88)	0 - 68	85 (100%)	.944
None (< 20)			40 (47.1%)	
Mild social anxiety (21 – 30)			15 (17.6%)	
Moderate social anxiety (31 – 40)			13 (15.3%)	
Severe social anxiety (41 – 50)			10 (11.8%)	
Very severe social anxiety (> 51)			7 (8.2%)	
<u>Applied Cognition*</u>	67.62 (17.41)	28 - 90	85 (100%)	.960
<u>Multidimensional Health Locus of Control (MHLc)*</u>				
Internal subscale	21.61 (6.72)	6 – 36	85 (100%)	.783
Chance subscale	20.22 (7.24)	6 – 36	85 (100%)	.788
Doctors subscale	10.88 (3.92)	3 – 18	85 (100%)	.696
Others subscale	10.87 (4.13)	3 - 18	85 (100%)	.764
<u>Rosenberg Self-Esteem Scale (RSES)*</u>	15.73 (5.97)	2 – 28	85 (100%)	.849
<u>Self Efficacy</u>				
Total	65.96 (30.83)	13 - 130	85 (100%)	.953
Low (13-59)			41 (48.2%)	
Moderate (60 – 114)			41 (48.2%)	
High (115 – 130)			3 (3.5%)	
<u>Stigma*</u>	65.50 (20.80)	24 – 120	85 (100%)	.953
<u>Hospital Anxiety and Depression Scale (HADS):Anxiety</u>				
Total	10.64 (4.72)	2 – 21	85 (100%)	.812
Normal (0 – 7)			25 (29.4%)	
Mild (8 – 10)			17 (20%)	
Moderate (11 – 14)			25 (29.4%)	
Severe (15 – 21)			18 (21.2%)	
<u>HADS: Depression</u>				

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Total	9.24 (4.92)	0 - 21	.830
Normal (0 – 7)		31 (36.5%)	
Mild (8 – 10)		25 (29.4%)	
Moderate (11 – 14)		12 (14.1%)	
Severe (15 – 21)		17 (20%)	

Note. All data in this table was calculated using pooled scores, following multiple imputation of missing data items. * indicates measures where valid cut-off scores for categorisation within a TBI population are not provided by the scale authors or subsequent published research.

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Table 3.

Correlation matrix for pooled demographic data following multiple imputation

	SPIN	Age	Gender	Time since TBI	Time in hospital	Employed	Live alone	In a relationship
SPIN	1							
Age	-.082	1						
Gender	.207	-.241*	1					
Time since TBI	.153	.274*	-.207	1				
Time in hospital	.037	.067	-.178	.482**	1			
Employed	.239*	.040	-.232*	.164	.125	1		
Live alone	-.090	-.308**	.002	-.175	-.120	-.167	1	
In a relationship	.065	-.008	-.172	.121	.276*	.398**	-.470**	1

Note. SPIN = Social Phobia Inventory; TBI = Traumatic brain injury.

* $p < .05$, ** $p < .01$, two-tailed

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Table 4.

Correlation matrix for pooled questionnaire data following multiple imputation

	SPIN	Applied cognition	MHLLoC Internal	MHLLoC Chance	MHLLoC Doctors	MHLLoC Other	RSES	Self Efficacy	Stigma	HADS Anxiety	HADS Depression
SPIN	1										
Applied cognition	.476**	1									
MHLLoC Internal	.248*	-.018	1								
MHLLoC Chance	.217*	.025	.324**	1							
MHLLoC Doctors	.033	-.083	.185	.167	1						
MHLLoC Other	.035	.073	.026	.151	.379**	1					
RSES	-.441**	-.345**	-.013	-.085	.101	-.012	1				
Self Efficacy	-.472**	-.398**	.022	-.087	.237*	.222*	.611**	1			
Stigma	.654**	.568**	.245*	.207	-.104	.079	-.481**	-.523**	1		
HADS anxiety	.726**	.384**	.199	.088	-.018	-.110*	-.492**	-.562**	.614**	1	
HADS depression	.516**	.433**	-.027	.174	-.170	.040	-.550**	-.677**	.582**	.505**	1

Note. HADS = Hospital Anxiety and Depression Scale; MHLLoC = Multidimensional Health Locus of Control (Form C); RSES = Rosenberg Self-Esteem Scale; SPIN = Social Phobia Inventory. * $p < .05$, ** $p < .01$, two-tailed