



## 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*

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## 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  
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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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33 No guidance is available specific to the management of SA after TBI, but empirically-  
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35 based guidance for generic SA interventions in the UK (NICE, 2013) recommends cognitive  
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37 behavioural therapy (CBT) as a first-line intervention (i.e., before pharmacological  
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39 interventions), underpinned by a specifically developed theoretical model (e.g., Clark &  
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41 Wells, 1995). However, a randomised controlled trial of a CBT programme for SA after  
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43 acquired brain injury (ABI) found that although SA did reduce, treatment effects were not  
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45 statistically significant (Hodgson, McDonald, Tate, & Gertler, 2012). However, a small  
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47 sample size ( $n = 12$ ) and variability in the ABI group (people who had experienced stroke,  
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49 hypoxic brain injury and cerebral oedema were included alongside those who had  
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51 experienced TBI) limits the usefulness of this study in understanding management of SA  
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53 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.

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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  
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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  
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30 lack of public understanding about the consequences of TBI and how this impacts on their  
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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 MHLc (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  
10  
11 other psychological variables explained some variance in SA, with internal LoC and self-  
12  
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,  
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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  
22  
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  
37  
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  
41  
42 and psychosocial functioning following TBI is influenced by a broad range of factors, with  
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44 psychological variables playing a key role alongside cognitive, neurological and demographic  
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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  
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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  
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9 experiences are a key factor in the development of SA (Rapee & Spence, 2004). The  
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11 cognitive model of SA, proposed by Clark and Wells (1995) and updated by Wells (2013),  
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13 proposes that social situations activate negative automatic thoughts based on assumptions  
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15 around perceived danger in social situations. Negative evaluations of how the self is  
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17 processed as a social object (i.e., how the person thinks they appear to others) are often  
18  
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  
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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  
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43 on the functional impairments of the individual, the social model considers disability to be  
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45 caused by the economic, cultural and environmental barriers which are faced by people with  
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47 physical or cognitive impairments. Consistent with the findings of the present study, Oliver  
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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  
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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  
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14 how negative social interactions can lead to negative societal stereotypes about what it means  
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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  
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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  
43  
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  
47  
48 an individual's psychological resilience during the complex process of recovery from TBI.  
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51  
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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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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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  
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9 a significant role in the development of SA, use of more advanced statistical techniques (e.g.,  
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11 structural equation modelling) would be useful next step following this study. For example,  
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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  
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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  
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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  
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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  
49  
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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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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2  
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  
13  
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  
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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  
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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  
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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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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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2 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  
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7 development of SA following TBI, above and beyond demographic and clinical variables.

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10 The study provides empirical support to the theoretical rationale for SA following TBI  
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12 proposed by Soo et al. (2012) and Moore et al. (2006), highlighting the potential application  
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14 of Kendall and Terry's (1996) model for psychosocial adjustment. Further research is  
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16 required to examine the complex relationships between such variables using a more stable  
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18 regression model, and to explore in more detail other variables which may have an influence  
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20 on SA using more advanced statistical techniques which allow for the examination of non-  
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22 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,

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2  
3 declines in activity, social contact, independence, functional status and employment  
4  
5 opportunities are often reported following TBI (Antonak, Livneh, & Antonak, 1993; Temkin,  
6  
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  
10  
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).  
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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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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 ( $n = 8$ ).  
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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).  
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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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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 Despite the lack of research or guidance around SA after TBI, a literature review  
12  
13 exploring anxiety following mild TBI (Moore et al., 2006) highlighted the potential for SA to  
14  
15 be a significant problem in this population. Furthermore, Soo, Tate and Rapee (2012) present  
16  
17 a theoretical rationale for high levels of SA in children and adolescents who have  
18  
19 experienced TBI. They draw on Kendall and Terry's (1996) model for understanding  
20  
21 individual differences and predicting psychosocial adjustment outcomes following TBI,  
22  
23 acknowledging a role for direct (neurological and cognitive impairment) and indirect  
24  
25 (situational and environmental) antecedent factors, but also emphasising the importance of an  
26  
27 individual's psychological resources such as appraisal style and coping responses. This is  
28  
29 consistent with cognitive theories of SA (e.g., Clark & Wells, 1995; Wells, 2013) and  
30  
31 approaches to management of other anxiety problems following TBI (Williams et al., 2003;  
32  
33 Soo & Tate, 2009). Consequently, an understanding of SA following TBI in adults must be  
34  
35 guided by research which explores the role of potentially relevant neurological, cognitive,  
36  
37 situational and psychological factors to guide assessment, formulation and intervention  
38  
39 during acute and long-term rehabilitation.

40  
41 A broad range of psychological variables may be important in SA following TBI (Soo  
42  
43 et al., 2012). Locus of control (LoC), the beliefs a person holds about how the behaviour of  
44  
45 themselves and others influences their health (Wallston, Stein, & Smith, 1994), has been  
46  
47 associated with SA (Cloutre, Heimberg, Liebowitz, & Gitow, 1992; Kennedy, Lynch, &  
48  
49 Schwab, 1998) and emotional problems in people who have experienced TBI (Moore &  
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51 Stambrook, 1992). Self-efficacy, the beliefs people hold about their capabilities is also  
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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).  
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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).  
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49  
50 In conclusion, despite the theoretical rationale for SA following TBI presented by Soo  
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52 et al. (2012) and Moore et al., (2006), present understanding of SA following TBI is limited  
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54 given the limited available research. No research to date has explored psychological factors  
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56 which might contribute to the development of SA following TBI to provide guidance for  
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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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25 **Predictor variables.** The Applied Cognition measure (Neuro-QOL, 2012) was used  
26  
27 to assess subjective severity of cognitive problems. This 18-item measure assesses perceived  
28  
29 difficulties in everyday cognitive domains including memory, attention, and decision-  
30  
31 making. Responses range from never (1) to very often (5), with a maximum score of 90. High  
32  
33 levels of internal consistency ( $\alpha = .95$ ) and test-retest reliability ( $r = .82$ ) have been  
34  
35 demonstrated in samples of patients with a range of neurological problems (e.g., stroke,  
36  
37 epilepsy, Parkinson's disease) but data are not available for a TBI sample (Neuro-QOL,  
38  
39 2010).  
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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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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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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  
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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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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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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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3 Hierarchical multiple regression analysis was used to explore the study hypothesis.  
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5 Variables were entered into the model in three blocks; demographic, clinical, psychological.  
6  
7 Consistent with the available theoretical rationale for SA following TBI discussed above, this  
8  
9 allowed for examination of the amount of variance in SA which could be explained by  
10  
11 psychological variables, above that explained by demographic and clinical variables.  
12  
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14 In determining what variables were entered into the regression model, decisions for  
15  
16 subset selection were made based on effect size instead of  $p$  values. While use of  $p$  values is  
17  
18 common, effect sizes are less reliant on sample size (Coe, 2002). Given the relatively low  
19  
20 sample size in this study ( $n = 85$ ), variables were included in the multiple regression analysis  
21  
22 if a small effect size was observed (i.e.,  $r > .1$ ; Cohen, 1988). This threshold was chosen to  
23  
24 allow an inclusive, exploratory approach which minimised the risk of overlooking emerging  
25  
26 effects of small magnitude (Hemphill, 2003).  
27  
28

## 29 Results

### 30 Data Preparation and Analysis

31  
32 It did not appear that there were any systematic biases or patterns to the missing data  
33  
34 as defined by Graham (2009), with 34 cases (40% of the sample) having incomplete data  
35  
36 across 42 (34.43%) of the variables. Little's (1988) Missing Completely At Random  
37  
38 (MCAR) test was not significant ( $X^2 = 1921.880$ ,  $df = 3105$ ,  $p = 1.000$ ), suggesting that the  
39  
40 null hypothesis of data being missing randomly could be assumed.  
41  
42  
43  
44

45 Even after removing the eight cases missing more than 10% of data, the number of  
46  
47 other cases missing smaller amounts of data was high. Listwise or pairwise deletion methods  
48  
49 were not considered appropriate as this would have seen a large proportion of cases deleted,  
50  
51 thereby reducing sample size and power in addition to potentially introducing bias into the  
52  
53 multiple regression model. Consequently, multiple imputation was conducted with the data  
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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**

12  
13  
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  
54  
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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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,

1  
2  
3 1998); worry that other people will think they are slow or stupid has the potential to increase  
4  
5 anxiety in social situations. Anxiety may also further reduce available attentional and  
6  
7 cognitive processing capacity (which may already be decreased following TBI), thereby  
8  
9 heightening and maintaining the problems experienced and the development of avoidance  
10  
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  
17 The addition of psychological variables (MHLc internal, MHLc chance, self-  
18  
19 esteem, self-efficacy, perceived stigma) made a significant additional contribution to the  
20  
21 amount of variance explained, suggesting that psychological variables are important factors  
22  
23 in the development of SA following TBI in addition to demographic and clinical variables.  
24

25 In the overall model (i.e., where the available variance was shared across a greater number of  
26  
27 predictor variables), only perceived stigma was a significant independent predictor of SA. All  
28  
29 other psychological variables explained some variance in SA, with internal LoC and self-  
30  
31 esteem predicting a greater amount of variance than self-efficacy and chance LoC. Although  
32  
33 internal LoC and self-esteem did not reach statistical significance as independent predictors,  
34  
35 this may be due to the relatively small sample size employed in the study and further  
36  
37 examination is warranted. Nevertheless, when self-esteem, self-efficacy and LoC are  
38  
39 combined with perceived stigma they explain a significant amount of variance in SA, above  
40  
41 and beyond that explained by demographic and clinical factors such as depression and  
42  
43 perceived cognitive problems. It should also be noted that adding these variables as the final  
44  
45 block in the regression model provides a particularly rigorous and robust test of their  
46  
47 predictive power.  
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51  
52 As outlined above, there is no previous research directly examining the role of  
53  
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  
8  
9 factors (Soo et al., 2012; Moore et al., 2006; Kendall & Terry, 1996).  
10

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  
15  
16 psychosocial functioning after TBI, in which perceived stigma is proposed as a key factor  
17  
18 affecting primary appraisal (i.e., how events are appraised), which subsequently affects  
19  
20 secondary appraisal (i.e., a person's beliefs around how well they can cope with an event).  
21  
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),  
27  
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  
31  
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  
35  
36 reinforce the beliefs (Wells, 2013). Safety behaviours maintain and exacerbate the problems  
37  
38 by perpetuating the beliefs that social interactions will lead to negative outcomes (Clark &  
39  
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  
45  
46 problems and reduced mood are also likely to be important factors in the development of  
47  
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.

### 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 **Clinical implications**

51  
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  
4  
5 LoC in the development of SA following TBI suggests a need to consider these variables  
6  
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.  
10

11  
12 In particular, the significant role which stigma plays in the development of SA  
13  
14 following TBI highlights the importance of developing contextually inclusive formulations  
15  
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.  
19  
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  
25  
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.  
37  
38 Educational programmes could highlight the difference between general intellectual ability  
39  
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

50  
51 It is recognised that the use of a self-selecting sample may have introduced some bias  
52  
53 to the sample. The study also focused exclusively on people living in the community. A  
54  
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  
6  
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.  
17  
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  
21  
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  
35  
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  
41  
42 reduce the amount of stigma which is perceived, mitigating its effect on SA.

43  
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45 Furthermore, the lack of characterisation of the sample in terms of objective severity  
46  
47 of injury and cognitive impairment is a limitation of the study. Perceived severity of  
48  
49 cognitive problems may not be accurate and the self-selected sample may potentially result in  
50  
51 a less impaired group. However, injury variables and degree of cognitive impairment do not  
52  
53 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  
4  
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).  
22  
23  
24

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  
28  
29 research might address environmental factors hypothesised to be of importance for  
30  
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,  
40  
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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46

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  
52  
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**

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

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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2-39

Table 1.

*Demographic characteristics (N = 85)*

	n	%	Mean (SD)	Range
<b>Gender</b>				
Male	54	63.5%		
Female	28	32.9%		
Other / prefer not to say	3	3.5%		
Age			42.4 (13.34)	19 - 81
<b>Cause of injury</b>				
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
<b>Employed</b>				
Yes	27	31.8%		
No	57	67.1%		
Prefer not to say	1	1.2%		
<b>Live alone</b>				
Yes	25	29.4%		
No	59	69.4%		
Prefer not to say	1	1.2%		
<b>Relationship status</b>				
Single	28	32.9%		
In a relationship	44	51.8%		
Separated / divorced	12	14.1%		
Other / prefer not to say	1	1.2%		
<b>Recruitment method</b>				
Online	54	55.1%		

## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2-40

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

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## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2-41

Table 2.

*Clinical characteristics of sample*

	Mean (SD)	Range	n (%)	$\alpha$
<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>				



## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2-42

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.

## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

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

## SOCIAL ANXIETY FOLLOWING TRAUMATIC BRAIN INJURY

2-44

Table 4.

*Correlation matrix for pooled questionnaire data following multiple imputation*

	SPIN	Applied cognition	MHL0C Internal	MHL0C Chance	MHL0C Doctors	MHL0C Other	RSES	Self Efficacy	Stigma	HADS Anxiety	HADS Depression
SPIN	1										
Applied cognition	.476**	1									
MHL0C Internal	.248*	-.018	1								
MHL0C Chance	.217*	.025	.324**	1							
MHL0C Doctors	.033	-.083	.185	.167	1						
MHL0C 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; MHL0C = Multidimensional Health Locus of Control (Form C); RSES = Rosenberg Self-Esteem Scale; SPIN = Social Phobia Inventory. \*  $p < .05$ , \*\*  $p < .01$ , two-tailed