



## Use of the Personality Assessment Inventory (PAI) in individuals with traumatic brain injury

Christine Till, Bruce K. Christensen & Robin E. Green

To cite this article: Christine Till, Bruce K. Christensen & Robin E. Green (2009) Use of the Personality Assessment Inventory (PAI) in individuals with traumatic brain injury, *Brain Injury*, 23:7-8, 655-665, DOI: [10.1080/02699050902970794](https://doi.org/10.1080/02699050902970794)

To link to this article: <https://doi.org/10.1080/02699050902970794>



Published online: 21 Jul 2009.



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



Article views: 622



View related articles [↗](#)



Citing articles: 5 View citing articles [↗](#)

## Use of the Personality Assessment Inventory (PAI) in individuals with traumatic brain injury

CHRISTINE TILL<sup>1,2</sup>, BRUCE K. CHRISTENSEN<sup>3,4</sup>, & ROBIN E. GREEN<sup>2,4,5</sup>

<sup>1</sup>Department of Psychology, York University, Toronto, Ontario, Canada, <sup>2</sup>University Centre, Toronto Rehabilitation Institute, Toronto, Ontario, Canada, <sup>3</sup>Department of Psychiatry & Behavioural Neurosciences, McMaster University, Toronto, Ontario, Canada, <sup>4</sup>Department of Psychiatry, and <sup>5</sup>Department of Rehabilitation Sciences, University of Toronto, Toronto, Ontario, Canada

(Received 14 January 2009; revised 14 April 2009; accepted 15 April 2009)

### Abstract

**Primary objective:** To evaluate the extent to which the Personality Assessment Inventory (PAI) is confounded by symptoms that are transdiagnostic between psychopathology and neurological sequelae.

**Methods:** Sixty-one adults with moderate-to-severe traumatic brain injury (TBI) completed the PAI over the first year post-injury. Items that discriminated brain-injured individuals from a normative sample were identified using effect size analyses and were then subjected to principal components analysis (PCA) with varimax rotation. To explore whether the items identified in the PCA may be considered transdiagnostic in nature, an expert rating task and correlations with objective outcome measures were employed.

**Results:** Effect sizes analyses identified 21 items that discriminated adults with TBI from the normative sample. Eight items associated with component 1 of the PCA were considered to be transdiagnostic. These items reflected health concerns and thinking problems from the Somatic Complaints, Depression and Schizophrenia scales. Items belonging to the other components reflected behaviours that are commonly associated with TBI, but not considered transdiagnostic.

**Conclusion:** Using a comprehensive and multi-modal approach, results demonstrated good convergent validity for a small sub-set of items as being transdiagnostic. Overall, the findings support the PAI as a useful measure of psychiatric and emotional disturbances among persons with TBI.

**Keywords:** Personality Assessment Inventory (PAI), validity, traumatic brain injury, self-report, neurologic symptoms

### Introduction

The assessment of personality and psychopathology following traumatic brain injury (TBI) is an important aspect of clinical care and rehabilitation. Although the most overt symptoms of moderate-to-severe TBI are cognitive [1–5] and physical [6], personality and psychiatric disturbance also constitute prevalent and consequential sequelae [5, 7–9], which often persist after cognitive and physical symptoms resolve [10, 11]. The most common psychiatric sequelae following TBI are those

associated with Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD) [9, 12, 13]. These common psychiatric sequelae of TBI can affect a number of domains, including increasing suicide risk [14–18], disrupting family life [19] and psychosocial function [11] and impeding successful reintegration to work and community [9, 20]. Consequently, measurement of psychiatric sequelae in individuals with TBI is critical for diagnostic assessment and for treatment planning and rehabilitation care. The Personality Assessment

Inventory (PAI) [21] is widely used for these purposes, ranking among the most frequently used objective tests of personality and psychopathology in clinical settings [22].

The PAI offers practical advantages for use with brain-injured populations because of its low literacy level requirement, which requires only a grade four reading level, and its brevity relative to other personality inventories, such as the Minnesota Multiphasic Personality Inventory (MMPI) and the Millon Clinical Multiaxial Inventory (MCMI). In addition to its focus on a broad range of diagnostic concepts, the PAI provides breadth in its coverage of clinical management issues by including scales that inquire directly about risk factors that are particularly pertinent to individuals with TBI, including alcohol and drug use, aggressive and suicide potential and lack of treatment amenability, any of which may directly affect rehabilitation progress. The clinical importance of this instrument within the field of clinical psychology and neurorehabilitation is therefore exemplified by its comprehensive and multi-purpose role.

The issue of whether neurological consequences of brain injury confound psychopathologic symptoms has received much attention in studies concerning the use of the MMPI [23] and its successor the MMPI-2 [24–31]. While this issue is also controversial with the PAI, little research has examined the validity of the items on this questionnaire when used with individuals with TBI. Most studies on the PAI have focused on the efficacy of the Validity scales and index scores in detecting various kinds of response distortion and in understanding the phenomenology of this distortion [32].

Interpretation of the PAI in patients with TBI may be complicated by the observation that some of the items on this instrument query symptoms that are characteristic of both psychopathology and neuropathology. That is, several psychological symptoms are common to both psychiatric and neurological illness and, as a consequence, can signal the presence of more than one condition. For example, endorsement of item 78 from the PAI, ‘*My thoughts get scrambled sometimes*’, may constitute one to three raw score points on the Schizophrenia scale according to the four-choice response format of the PAI. Elevations on other clinical scales and in particular the Somatic Complaints (SOM) and Depression (DEP) scales, which include items such as ‘*difficulty with memory and concentration*’ and ‘*headaches and dizziness*’, may also be confounded by the typical symptoms of brain injury. Indeed, clinical elevations on these two scales with mean *T*-scores greater than 60 have been demonstrated in brain injury studies that have conducted PAI profile analyses [33, 34]. Similarly, in a study of chronic pain patients [35],

endorsement of items on the SOM and DEP scales, particularly items related to physiological manifestations of depression, was elevated relative to the normative sample. The authors explained that the psychological factors observed in this sample may play a role in maintaining and exacerbating chronic pain. While this relationship is probable, an alternate explanation is that elevated scores on the DEP scale were inflated given the influence of neurological-related endorsements on the Physiological Depression sub-scale that could be attributed to genuine physical (rather than psychological) symptoms in chronic pain patients.

The current study was conducted in order to better understand the validity of the PAI when used with a TBI population and whether items on this questionnaire are confounded by symptoms that are transdiagnostic between psychopathology and neurological sequelae. The term ‘transdiagnostic’ is used to address items on the PAI that, in addition to their intended content, may also be associated with physical (e.g. orthopaedic injury) and/or neurological sequelae related to TBI (e.g. cognitive complaints secondary to the TBI). This objective was achieved using three convergent approaches: empirical identification of transdiagnostic items, expert rating of these items and correlational analyses between these items and an independent TBI-related outcome measure (i.e. Functional Independence Measure (FIM)) [36]. The hypothesis was that two distinct factors would be endorsed by individuals with TBI relative to the normative sample. The first factor, referred to as ‘transdiagnostic’, would consist of items that are considered to be a veridical reflection of the *bonafide* physical and cognitive symptoms related to the injury and/or psychopathological features common to this population, including elevations on the SOM and DEP scales; the second factor, referred to as ‘non-transdiagnostic’, would consist of items considered to reflect behavioural disorders that are commonly elevated in brain-injured individuals [33], but do not appear to be confounded by neurological symptoms, such as elevations on the Antisocial (ANT) and Alcohol (ALC) scales.

## Methods

### *Participants*

Sixty-one adults with moderate-to-severe TBI (53 men, eight women) met inclusion criteria for the study. All patients were recruited from the in-patient programme of the Neurorehabilitation Programme at the Toronto Rehabilitation Institute, Canada as part of a larger study on cognitive recovery following TBI. Patients were included in

the study if they had a TBI that was confirmed by the presence of post-traumatic amnesia (PTA) of 1 hour or more (but no more than 6 weeks) and/or Glasgow Coma Scale (GCS) score of 12 or less, either on arrival in the emergency room or at the scene of the accident and/or positive CT or MRI findings. Where information related to PTA was not recorded in the medical record, questioning of the patient and caregivers was undertaken during a structured interview. Exclusion criteria included the following: a pre-existing neurological diagnosis (e.g. Huntington's Disease, dementia); TBI secondary to another brain injury (e.g. fall due to stroke); elevations on any of the four validity scales of the PAI; current diagnosis or history of psychotic or psychiatric disorder, including depression and schizophrenia; alexia or English proficiency precluding comprehension of the PAI, as judged by the treating Speech Language Pathologist and/or Psychometrist. Trans-diagnostic items in brain injured populations have been identified on the MMPI/MMPI-2 (termed 'neurologically-relevant items') using similar subject selection criteria and sample sizes [23, 26–28].

The mean age of the sample was 38.1 years ( $SD = 15.3$ , range 17–79) and mean years of formal education was 12.3 years ( $SD = 3.5$ ). Average time since injury was 119.0 days ( $SD = 122.2$ , range 32–496) at time of PAI completion. Injury was most commonly due to motor vehicle accidents (61.7%), followed by falls (30.0%), assaults (6.7%) and sports injury (1.7%). The mean GCS score (lowest of recorded scores taken; data available for 52 (85%) participants) was 7.11 ( $SD = 3.7$ ), denoting a severe injury on average. Across the sample, the length of PTA was classified as being very mild (<5 minutes) for 4.9%; moderate (1–24 hours) for 3.3%; severe (1–7 days) for 18.0%; very severe (1–4 weeks) for 39.3%; and extremely severe (>4 weeks) for 13.1%. Length of PTA was not available for 13 (21.3%) participants. The mean acute care length of stay (in days), calculated based on admission and discharge dates from the acute care hospital, was 36.0 ( $SD = 15.3$ ; range = 12–68). Twenty-one per cent of the sample (13/61) was actively involved in litigation at the time of PAI completion.

Participants in this study were judged to represent a valid sample of TBI patients for the following reasons: All patients received a clinically confirmed diagnosis of TBI; no evidence of symptom exaggeration was observed by the clinical team during any of the patients' acute care stay, which was 2 months on average; no individuals were identified as exaggerating cognitive dysfunction on the Test of Memory Malingering (TOMM) [37]; and, finally, participants were not included in the study if they showed elevations greater than the cut-off on any

of the four validity scales using the recommended guidelines [21].

### Measures

Individuals with TBI were administered the PAI according to standard test instructions, which are provided in the item booklet. The importance of answering all items was emphasized, as was the necessity for choosing only one response per item. If the respondent did not understand an item, he or she was asked to circle it; the examiner would then provide simple definitions of words or clarification of questions when appropriate.

The PAI requires roughly 45 minutes to complete and consists of 344 statements rated on a four-point scale with the labels: 'false, not at all true', 'slightly true', 'mainly true' or 'very true'. This self-report instrument consists of 22 non-overlapping full scales: four validity (Inconsistency, Infrequency, Negative Impression and Positive Impression), 11 clinical (Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression, Mania, Paranoia, Schizophrenia, Borderline Features, Antisocial Features, Alcohol Problems and Drug Problems), five treatment consideration (Aggression, Suicidal Ideation, Stress, Non-support and Treatment Rejection) and two interpersonal (Dominance and Warmth). The 11 clinical scales are further organized into clinically meaningful sub-scales designed to facilitate interpretation of multifaceted clinical constructs [21]. Raw scores on the PAI are standardized with reference to a national, census-matched sample of 1000 community adults and transformed to linear *T*-scores that have a mean of 50 and a standard deviation of 10.

### Procedure

All patients were assessed by a clinical neuropsychologist. Following a clinical interview and neuropsychological assessment, the PAI was administered only to patients deemed capable of reading and understanding the item content and as having the attention span to complete the questionnaire in one sitting (as deemed by the patient's level of functioning and stamina during the neuropsychological assessment).

The following three techniques were executed to discriminate items that represented a blending of psychopathology and sequelae of TBI, which are referred to here as transdiagnostic items. The first technique, following Gass [25], entailed a statistical comparison of endorsements made by the TBI sample to those of the normative sample published in the PAI manual, followed by principal components analysis (PCA) on the items that discriminated the two groups. Here, Cohen's *d* effect sizes [38]

were calculated using the mean score and standard deviation for each item on the PAI as endorsed by the TBI sample compared with the census-matched community standardization sample of 1000 subjects on which the PAI is normed. Items were evaluated according to the criteria outlined by Gass and Russell [23]: (1) degree of discriminative power (i.e. medium effect size of at least 0.3) and (2) the frequency of endorsement within the TBI group (i.e. item endorsed by at least 25% of sample). Items selected via these criteria were then subjected to a PCA and the resulting component matrix was submitted to varimax rotation. The number of factors to retain was determined using parallel analysis [39]. Only items with a factor loading of 0.40 or greater were retained in the component solution [40].

In the second technique, expert ratings were used to establish the content validity of the two-component model. Nine raters with extensive experience working with TBI clients (including physiatrists, neuropsychologists and occupational therapists who were not authors on the paper) were asked to decide whether the 21 items that empirically discriminated controls from TBI patients may be considered to be trans-diagnostic (i.e. defined as the possibility of signalling a symptom of brain injury in addition to the construct it is designed to assess). Raters were asked independently to indicate 'yes' if the item may be considered to be transdiagnostic, and 'no' if it was not. The raters were unaware of the components that were identified using PCA. Percentage agreement across raters and the average measure inter-class correlation were examined for all 21 items in this task.

The third technique involved a series of bivariate correlations between factor 1 and 2 items with (i) admission total score on the Functional Independence Measure (FIM) [36], where a higher score reflects a greater degree of independent functioning and (ii) an overall measure of psychopathology (Mean Clinical Elevation (MCE) index score) on the PAI, where a higher score is reflective of a greater degree of psychopathology. The FIM was designed to assess areas of dysfunction relevant to the assessment of persons with TBI and is characterized by two dimensions labelled motor and cognitive. Since the MCE index score is not an independent measure of psychopathology as it may be impacted by elevations tapped by items in components 1 and 2, the MCE score was recalculated without inclusion of the items (i) comprising factor 1 (for the correlation with the factor 1 score)

and (ii) comprising factor 2 (for the correlation with the factor 2 score). In order to examine the face validity of factors 1 and 2, factor score coefficients were estimated using the regression method and were then correlated with these independent outcome measures (i.e. FIM and MCE scores). A dissociation between the factor scores and these two outcome measures would provide support for the idea of two distinct constructs, i.e. a transdiagnostic and a non-transdiagnostic construct. Correlation analysis were also used between factor 1 and 2 scores and total score on the Beck Depression Inventory (BDI) [41] to examine the strength of association between symptoms of depression and endorsement of transdiagnostic items. It was reasoned that factor 1 scores would correlate with the BDI score given that somatic complaints represents a cluster of symptoms that also may arise from or be direct manifestations of pathology and neurologic impairment [42].

As a final step, the impact of the identified transdiagnostic items on the relevant clinical subscale *T*-scores was evaluated by deleting these eight items from their respective sub-scales and then re-scoring the PAI for each individual. This approach is equivalent to re-scoring the PAI with the transdiagnostic items left in and assigning each item the lowest score (i.e. 'false') regardless of the patient's actual endorsement for the specific item.

## Results

Of the 61 completed PAIs, eight were excluded from further analysis because of elevations on the validity scales: Infrequency,  $T > 74$  ( $n = 1$ ), Inconsistency,  $T > 73$  ( $n = 1$ ), Negative Impression Management,  $T > 84$  ( $n = 2$ ) and Positive Impression Management,  $T > 68$  ( $n = 4$ ). Table I presents the means and standard deviations for the relevant PAI validity indices obtained in the TBI sample compared with the normative population mean. Mean differences on the Infrequency, Inconsistency and Negative Impression Management validity scales were not statistically different and reflected small-to-medium effect sizes (Cohen's  $d < 0.30$ ) [38]. The TBI group showed a significantly higher score on the PIM scale ( $t(52) = 4.16$ ,  $p < 0.01$ ) relative to the normative group.

Of the 336 clinical scale items, an elevated endorsement in the TBI sample relative to the normative sample with an effect size of 0.30 or greater was found in 21 of the comparisons.† Differential responses on these 21 items showed an

†Endorsements on all PAI items were transformed such that a higher score reflected increased symptomatology or phenomenology of the clinical construct under investigation. The items for which the normal control sample produced a higher endorsement were not included in the analyses as these items would not increase the probability of over-interpreting psychopathology on the PAI.

Table I. Mean Personality Assessment Inventory validity scales in individuals with moderate-to-severe traumatic brain injury ( $n = 53$ ) relative to the normative value.

Scale	<i>M</i>	SD	<i>t</i> -test	Cohen's <i>d</i>
ICN	51.60	7.98	1.46	0.18
INF	52.40	9.07	1.92	0.25
NIM	49.85	8.06	-0.14	-0.02
PIM	54.64	8.11	4.16*	0.51

ICN = Inconsistency; INF = Infrequency; NIM = Negative Impression Management; PIM = Positive Impression Management. \* $p < 0.01$ .

endorsement frequency above the criterion of 25% and thus were submitted to a PCA. Seven components with eigenvalues greater than one and accounting for 67.6% of matrix variance were extracted from these items. However, results of a parallel analysis [43] suggested a four component solution, beyond which only minimal additional incremental variance would be explained. A solution of four components yielded a distinctive transdiagnostic component, two further components with conceptual coherence to items (i.e. set of inter-related items that are applied to a given concept) and a fourth conceptually ambiguous component. Items loaded on these components in a non-overlapping manner that together accounted for 50.8% of the matrix variance (see Table II). No differences in item endorsement patterns were revealed according to the interval between TBI and PAI assessment (4 months and less vs. greater than 4 months post-injury) and litigation status and thus these patients were combined in subsequent analyses.

Component 1 (19.53% of the variance) consisted of eight items. The content of the component 1 items was considered to reflect trans-diagnostic items, including complaints about physical health, confused thinking and difficulties with concentration. Five of the eight items in component 1 are from the Somatic Complaints scale, specifically the Conversion (item 3), Somatization (item 112) and Health Concern sub-scales (items 52, 92 and 252); two items are from the Schizophrenia scale (items 38 and 318, Thoughts Disorder sub-scale); and one item is from the Depression scale (item 155, Physiological sub-scale). Component 2 (13.40% of the variance) consisted of five items seemingly related to a history of negative behaviours, most notably in the area of substance abuse (items 95 and 334, Alcohol scale) and unlawful behaviour (item 251, Antisocial Behaviours sub-scale), as well as uncertainty about life issues (item 217, Identity Problems sub-scale) and perceived stress (item 326, Stress scale). Component 3 (9.65% of the variance) consisted of four items related to psychiatric

Table II. Component loadings of the PAI items that significantly differentiated the TBI sample from the census-matched standardization sample ( $n = 1000$ ).

Item no.	Clinical scale**	Component*			
		1	2	3	4
318	SCZ-T	<i>-0.707</i>	0.256	0.270	-0.042
3	SOM-C	<i>0.657</i>	0.318	0.088	-0.289
92	SOM-H	<i>0.667</i>	-0.189	-0.074	-0.048
38	SCZ-T	<i>0.652</i>	0.080	0.292	-0.087
155	DEP-P	<i>0.694</i>	-0.130	0.097	0.322
112	SOM-S	<i>-0.597</i>	0.393	0.361	0.114
52	SOM-H	<i>0.550</i>	0.023	0.158	0.033
252	SOM-H	<i>-0.485</i>	0.469	0.196	0.006
334	ALC	<i>-0.001</i>	<i>0.748</i>	-0.029	-0.070
251	ANT-A	0.070	<i>0.730</i>	-0.139	0.155
95	ALC	0.074	<i>-0.553</i>	0.350	0.478
217	BOR-I	<i>-0.244</i>	<i>0.408</i>	-0.064	0.095
326	STR	<i>-0.225</i>	<i>0.405</i>	-0.234	-0.012
159	ANT-S	<i>-0.033</i>	-0.044	<i>0.752</i>	-0.151
42	RXR	0.000	-0.123	<i>0.712</i>	-0.003
288	PAR-H	0.000	-0.123	<i>0.712</i>	-0.003
205	ARD-O	-0.008	0.219	<i>0.538</i>	0.412
211	ANT-A	0.032	0.311	-0.045	<i>0.648</i>
128	PAR-H	0.202	-0.046	-0.231	<i>0.647</i>
323	STR	0.329	-0.225	0.320	<i>0.520</i>
274 <sup>§</sup>	ARD-T	0.225	-0.278	0.337	0.199

\*Italicized loadings indicate assignment of items to components. \*\* ALC = Alcohol; ANT-A = Anti-social-Anti-social Behaviours; ANT-S = Anti-social-Social; ARD-O = Anxiety Related Disorder-Obsessive Compulsive; ARD-T = Anxiety Related Disorder-Traumatic Stress; DEP-P = Depression-Physiological; SCZ-T = Schizophrenia-Thought Disorder; SOM-C = Somatic-Conversion; SOM-H = Somatic-Health Concerns; SOM-S = Somatic-Somatization; PAR-H = Paranoid-Hypervigilance; RXR = Treatment Rejection; STR = Perceived Stress. <sup>§</sup> Item not included in 4-factor model because item loading less than 0.40.

problems of an anxiety-related (item 205, Obsessive-Compulsive sub-scale) and paranoid nature (item 288, Hypervigilance sub-scale), a desire to change (item 42, Treatment Rejection scale) and be stimulated (item 159, Stimulus Seeking sub-scale). Finally, component 4 (8.20% of the variance) consisted of the following three items: 'I usually assume people are telling the truth' (item 128, Hypervigilance sub-scale), 'I was never expelled or suspended from school when I was young' (item 211, Antisocial Behaviour sub-scale) and 'There have been many changes in my life recently' (item 323, Stress scale). Item 274 (Traumatic Stress sub-scale), 'Since I had a very bad experience, I am no longer interested in some things that I used to enjoy', was excluded from the four-factor model because its loading was less than 0.40

A two-component solution was also generated to determine whether component 1 (suggestive of transdiagnostic items) would be preserved based on *a priori* expectations that transdiagnostic items would be identified among non-transdiagnostic items.

All eight items comprising component 1 in the four-component solution were preserved and identical in the two-component solution, which accounted for 32.9% of the total variance. The items and respective scales comprising each component and their respective component loadings are listed in Table III. In the compressed two-component solution, three of the non-transdiagnostic items (items 128, 211 and 217) had component loadings less than 0.40 and were therefore dropped from the solution. These three items also showed the lowest communalities, which provides further support that they were poor items for inclusion in the matrix solution.

The content validity of the transdiagnostic component was examined using an expert-rating task. As shown in Table III, raters identified all eight trans-diagnostic items from the entire set of 18 items presented to them, consistent with the results of the PCA. Agreement was above 70% for all items, except for one (item 323, 'There have been many changes in my life recently'), for which 66.3% of raters identified it as a non-transdiagnostic item. The inter-class correlation coefficient averaged across all nine experts and 21 items was 0.95.

The content validity of the two components was further examined by correlating each component with an objective measure of neurological sequelae

(i.e. Functional Independence Measure (FIM)) and an index of general psychopathology (i.e. Mean Clinical Elevation (MCE) on the PAI). As shown in Table IV, results revealed a significant negative correlation between FIM total score and the factor score coefficient for component 1 (i.e. transdiagnostic factor) ( $r = -0.25, p < 0.05$ ), but not component 2 (i.e. non-transdiagnostic factor) ( $r = 0.09, p = 0.27$ ). With respect to correlations with the MCE score, both factors were significantly correlated, though the relationship was slightly stronger for factor 2 ( $r = 0.55, p < 0.001$ ) than factor 1 ( $r = 0.43, p = 0.001$ ). Finally, the association was significant between the BDI score and factor 1 ( $r = 0.55, p < 0.001$ ), but not between the BDI score and factor 2 ( $r = 0.14, p = 0.17$ ).

Table V presents the original and adjusted composite *T*-scores for each clinical scale and sub-scale on the PAI affected by the identified transdiagnostic items. Average *T*-score reductions that resulted from adjustment were as follows: the Somatic Complaints scale declined by 4.96 *T*-scores, with average sub-scale reductions of 5.27 on the Health Concerns sub-scale, 4.45 on the Conversion sub-scale and 3.15 on the Somatization sub-scale. The Depression scale declined by 1.38 *T*-scores, which directly reflected the reduction of 2.70 *T*-scores on the

Table III. Component loadings of the PAI items that significantly differentiated the TBI sample from the census-matched standardization sample ( $n = 1000$ ) using a two-component model. Expert rating agreement for each item is shown.

PAI Scale*	Component loading	Expert Rating	
		Item	Agreement
Component 1: Trans-diagnostic items			
SCZ-T	0.785	I can concentrate now as well as I ever could.	78%
SOM-S	0.765	I am in good health.	78%
SOM-H	-0.692	It's a struggle for me to get things done with the medical problems I have.	100%
SOM-H	0.642	For my age, my health is pretty good.	78%
DEP-P	-0.623	I've been moving more slowly than usual.	100%
SCZ-T	-0.512	My thinking has become confused.	100%
SOM-C	0.502	My health condition has restricted my activities.	78%
SOM-H	-0.456	My health problems are very complicated.	100%
Component 2: Non-transdiagnostic items			
ALC	0.694	There have been times when I've had to cut down on my drinking.	89%
RXR	0.665	I need to make some important changes in my life.	100%
PAR-H	0.643	People have to earn my trust.	100%
ANT-S	0.594	If I get tired of a place, I just pick up and leave.	89%
STR	0.533	There have been many changes in my life recently.	67%
ARD-T	0.480	Since I had a very bad experience, I am no longer interested in some things that I used to enjoy.	78%
ARD-O	0.444	I'm usually aware of objects that have a lot of germs.	100%
ANT-A	-0.434	I've never been in trouble with the law.	100%
ALC	-0.419	My drinking has never gotten me into trouble.	100%
STR	-0.407	I'm happy with my job situation.	100%

\*ALC = Alcohol; ANT-A = Anti-social-Anti-social Behaviours; ANT-S = Anti-social-Social; ARD-O = Anxiety Related Disorder-Obsessive Compulsive; ARD-T = Anxiety Related Disorder-Traumatic Stress; DEP-P = Depression-Physiological; SCZ-T = Schizophrenia-Thought Disorder; SOM-C = Somatic-Conversion; SOM-H = Somatic-Health Concerns; SOM-S = Somatic-Somatization; PAR-H = Paranoid-Hypervigilance; RXR = Treatment Rejection; STR = Perceived Stress (item number associated with each statement on the PAI is removed).

Depression–Physiological sub-scale. Finally, the Schizophrenia scale declined by 2.47 *T*-scores, which reflected the statistically significant reduction on the Schizophrenia–Thoughts Disorder sub-scale of 5.68 *T*-scores. A multivariate analysis of variance conducted between the adjusted and unadjusted *T*-scores for the scales and sub-scales revealed a significant omnibus difference,  $F(8, 97) = 3.19$ ,  $p < 0.01$ . Univariate tests for differences in *T*-scores on each adjusted vs. unadjusted clinical scale and sub-scale were significant for all Somatic Complaints sub-scales (Conversion, Somatization, Health

Concern) as well as the Thoughts Disorder sub-scale from the Schizophrenia scale.

## Discussion

This study attempted to ascertain whether certain items from the PAI are sensitive to non-psycho-pathological effects of moderate-to-severe TBI (e.g. the physical and cognitive sequelae of brain injury). To this end, 21 items were identified on which clients with TBI scored statistically higher

Table IV. Pearson correlation between component 1 and 2 items and FIM total score and mean clinical elevation score on the PAI.

Item	FIM total score		Mean clinical elevation	
	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value
Component 1: Trans-diagnostic items				
My health condition has restricted my activities.	−0.38	0.003**	0.09	0.24
My thinking has become confused.	−0.12	0.20	0.42	0.001**
My health problems are very complicated.	−0.09	0.26	0.33	0.006*
It's a struggle for me to get things done with the medical problems I have.	−0.12	0.20	0.36	0.003**
I am in good health.	0.22	0.06	−0.30	0.01*
I've been moving more slowly than usual.	−0.21	0.07	0.19	0.08
For my age, my health is pretty good.	0.21	0.07	−0.30	0.01*
I can concentrate now as well as I ever could.	0.13	0.18	−0.25	0.03*
Factor score coefficient (Component 1)	−0.25	0.047*	0.43	0.001**
Component 2: Non-transdiagnostic				
There have been times when I've had to cut down on my drinking.	0.05	0.38	0.18	0.09
I need to make some important changes in my life.	−0.90	0.27	0.32	0.007**
People have to earn my trust.	0.01	0.47	0.36	0.003**
If I get tired of a place, I just pick up and leave.	−0.28	0.02*	0.33	0.005**
There have been many changes in my life recently.	−0.07	0.31	0.26	0.025*
Since I had a very bad experience, I have lost interest in things that I used to enjoy.	−0.23	0.06	0.38	0.002**
I'm usually aware of objects that have a lot of germs.	0.01	0.48	0.09	0.25
I've never been in trouble with the law.	0.07	0.31	−0.40	0.001**
My drinking has never gotten me into trouble.	−0.16	0.14	−0.20	0.06
I'm happy with my job situation.	0.12	0.20	−0.34	0.004**
Factor score coefficient (Component 2)	0.09	0.27	0.55	< 0.001**

Mean Clinical Elevation score is adjusted for items in component 1 and 2 in order to decrease artificial correlation with factor scores and individual items\* $p < 0.05$ ; \*\* $p < 0.005$ .

Table V. Means, standard deviations and *F*-ratios for the PAI *T*-scores in the original and adjusted profiles (i.e. deletion of TBI-contaminated items).

PAI clinical and sub-clinical scale	Original profile		Adjusted profile		<i>F</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Somatic complaints	51.68	7.39	46.72	5.20	15.99**
Conversion	52.53	9.35	48.08	7.90	7.04*
Somatization	48.83	7.40	45.68	6.52	5.41*
Health Concern	52.77	7.86	47.51	5.30	16.36**
Depression	50.42	9.49	49.04	9.07	0.58
Physiological	50.70	10.45	48.00	9.42	1.95
Schizophrenia	48.43	8.83	45.96	8.57	2.14
Thought disorder	49.87	9.39	44.19	7.42	11.93**

\*\*  $p < 0.001$ ; \*  $p < 0.025$ .

than healthy controls, which were then subjected to a principal components analysis (PCA). As predicted, results revealed a set of discriminatory items, which appeared to have a four-dimensional structure that accounted for 50.8% of the total variance. All eight items that loaded on the first component had face validity consistent with being transdiagnostic in nature or reflecting a blending of neuropathology and psychopathology. A similar dimensional structure with evidence of a division between transdiagnostic and non-transdiagnostic items was also revealed in a two-factor solution, suggesting that the transdiagnostic component is preserved using a compressed PCA.

The items that were considered to be transdiagnostic between psychopathology and neurological sequelae belonged to one of three clinical scales: Depression (DEP), Somatic Complaints (SOM) and Schizophrenia (SCZ). Elevations in these domains are consistent with reports in the literature in both mild and moderate-to-severe TBI injury groups [10, 33, 44], as assessed using different instruments. Of note, the discriminating items in the current study were identified using a conservative criterion (effect size of 0.3) intended to reduce the likelihood of a Type I error. While this conservative approach provides confidence that the identified items were indeed discriminating items, it may have resulted in a failure to identify items that can discriminate between the groups, but less robustly so. For example, item 313, 'I have very steady hands' or item 166, 'I've lost interest in things that I used to enjoy' would logically seem to discriminate between the groups, but did not reach the 0.3 cut-off.

To explore whether the transdiagnostic items identified in component 1 are associated with physical and cognitive neurologic symptoms, a stepwise empirical method was employed that included an

expert rating task and correlations with objective outcome measures. The strong inter-rater agreement in the rating task provided construct validity supporting interpretation of this component as a TBI-relevant one: here, experts discriminated all eight transdiagnostic items from the non-transdiagnostic items with levels of agreement greater than 70%. In other words, these were the items that raters identified as being the most difficult to interpret clinically as representing organic vs psychological changes in diagnostic assessment.

To further substantiate the notion of a transdiagnostic component, the items in component 1 were correlated with the admission score on the FIM. Results revealed a negative and significant association between the FIM score and the factor score of the eight identified transdiagnostic items, but not for the non-transdiagnostic items. In other words, the higher the FIM score, which is reflective of a greater degree of functional independence in both cognitive and motor domains, the less likely items in component 1, which were reflective of somatic complaints, thought disorders and physiological depression, would be endorsed. This dissociation provides support for the items in component 1 as reflecting TBI-related sequelae in addition to their intended construct.

While both factors correlated with a general index of psychopathology (i.e. Mean Clinical Elevation (MCE) index score on the PAI) and did not differ significantly between each other, results were in the expected direction with the non-transdiagnostic component 2 being more strongly correlated with the MCE score relative to the transdiagnostic component 1. The lack of a clear dissociation between the components and the MCE score may reflect the fact that the items in component 1 do not solely reflect TBI-related symptoms, but also the possibility of comorbid *bonafide* psychopathology, which is expected given the significant prevalence of psychopathology among TBI groups [9, 10, 12, 13]. Secondly, the MCE may not be a sensitive measure of general psychopathology, particularly in cases where the psychiatric problems are specific to a particular clinical domain, such as depression. Finally, the use of the BDI in the current study as a measure of depression may also be impacted by neurological symptoms [42, 45] and thus was not expected to differentiate the transdiagnostic items from psychopathology. Future studies should use an independent criterion variable to assess psychopathology and its relationship to items identified in the non-transdiagnostic component.

Inspection of the individual items that loaded on the non-transdiagnostic factor (component 2) also

made sense conceptually for a TBI population. These items related to anti-social behaviours, history of substance abuse and psychiatric problems of an anxiety-related and paranoid nature, all of which have been associated with TBI [10, 44, 46] and would be expected to discriminate patients with TBI from a normative population. However, whether these traits—in addition to those identified as being transdiagnostic—reflect post-injury changes in personality and psychopathology or whether they pre-date the injury or have become exacerbated as a result of the TBI is difficult to establish given the temporal constraints of addressing this question. Indeed, previous longitudinal studies have reported evidence of pre-existing elevations in somatoform symptomatology amongst persons who later sustained head injuries [47, 48].

One of the limitations of the current study is that only moderate-to-severe TBI patients who were physically and cognitively able to complete the PAI were included. It is possible that the nature and intensity of symptom reporting would differ in a mild TBI group given the 'severity effect' that has been well documented in the literature [23, 49] showing that patients with mild head injuries tend to self-report greater symptomatology than do patients with more severe injuries. Thus, future studies should include a comparison between mild and more severe TBI groups to examine whether severity of injury or other factors (e.g. involvement in litigation) might moderate the frequency of endorsement of neurologic or transdiagnostic symptoms. In addition, the impact of neurologic symptom reporting may become less pronounced as TBI-related symptoms resolve over time as shown in a study of patients with mild TBI who completed the MMPI-2 immediately upon hospitalization and again at least 3 Months later [30]. Current findings, however, did not suggest any differences in item endorsement patterns as a function of time since injury. It should also be noted that the current TBI sample reported a significantly higher Positive Impression Management score relative to the normative sample, though not above that of a clinically significant cut-off for an invalid response set. This result implies that the sample of TBI patients presented themselves in a more favourable light compared with the normative sample. Similar findings were reported by Ruocco et al. [46] using the MCMI-III in a large outpatient sample of TBI patients who were compared with a psychiatric sample, despite the majority of the TBI patients being involved in litigation or in seeking a disability claim. Thus, cross-validation is warranted extending to outpatient settings, as well as to other neurodiagnostic

populations, such as stroke or multiple sclerosis, with whom the PAI is used.

#### *Validity of using the PAI in persons with moderate-to-severe TBI*

The current findings raise the question of how neurologic symptom reporting can impact scores on the PAI and its subsequent interpretation. Not surprising, a significant change in the SOM clinical scale score was observed after correction for the five items identified as being transdiagnostic. The need for careful interpretation of this particular scale is echoed in the PAI interpretive guide where it is cautioned that elevations on the SOM scale 'cannot distinguish between functional and organic features' ([21], p. 23). The SCZ scale also showed an overall significant change after correction, supporting Morey's [50] claim that elevations on the SCZ Thought Disorders sub-scale may be indicative of cognitive problems of non-schizophrenic origin, including 'the sequelae of brain injury or disease' (p. 105). In contrast, the DEP scale was shown to be relatively immune to the effects of neurological symptom reporting with no significant change in the overall *T*-score observed after correction. Taken together, these data suggest that clinicians can be confident in applying a standard interpretation to scores on the DEP clinical scale in a moderate-to-severe TBI population whereas greater interpretive uncertainty remains with an elevated score on the SOM and SCZ-Thought Disorder scales. Clearly, a complete clinical picture describing neurologic, psychological and financial/motivational factors is always warranted to determine whether reported elevations are due to *bonafide* psychopathology, an attempt to feign psychopathology for secondary gain or possibly attributed and/or confounded by cerebral dysfunction.

In conclusion, individuals with TBI often exhibit high levels of psychopathology and personality disturbance that may, in part, be attributed to the cognitive and physical sequelae of the injury, rather than solely representing psychopathology, mood disorders or psychological maladjustment. Consequently, clinicians and researchers are faced with the challenge of understanding how sequelae of the injury may impact the assessment of psychological disturbance. Using an empirical analysis and convergent approaches, the results of the current study identified eight transdiagnostic items on the PAI amongst individuals with moderate-to-severe TBI. The potential for an inflated clinical score due to the endorsement of these transdiagnostic items was confined to only three scales. Correction for these specific items had a significant impact on the

Somatic Complaints scale and Schizophrenia–Thoughts Disorders sub-scale, but not the Depression scale. These results conform with and underscore Morey’s [50] caution for a careful interpretation for the Somatic Complaints scale and the Schizophrenia–Thought Disorders sub-scale when using the PAI with brain-injured populations. Importantly, the current findings do not suggest evidence of neurologic symptom contamination on other scales on the PAI and therefore support the applicability of the PAI as an assessment tool for individuals who have sustained TBI. Additional research is needed to confirm and extend the current findings on the PAI in the assessment of other neurologic populations.

**Declaration of interest:** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

## References

- Dikmen SS, Machamer JE, Powell JM, Temkin NR. Outcome 3 to 5 years after moderate to severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation* 2003;84:1449–1457.
- Millis SR, Rosenthal M, Novack TA, Sherer M, Nick TG, Kreutzer JS, High Jr WM, Ricker JH. Long-term neuropsychological outcome after traumatic brain injury. *Journal of Head Trauma Rehabilitation* 2001;16:343–355.
- Novack TA, Alderson AL, Bush BA, Meythaler JM, Canupp K. Cognitive and functional recovery at 6 and 12 months post-traumatic brain injury. *Brain Injury* 2000;14:987–996.
- Novack TA, Bush BA, Meythaler JM, Canupp K. Outcome after traumatic brain injury: Pathway analysis of contributions from premorbid, injury severity, and recovery variables. *Archives of Physical Medicine and Rehabilitation* 2001;82:300–305.
- Prigatano GP. Personality disturbances associated with traumatic brain injury. *Journal of Consulting and Clinical Psychology* 1992;60:360–368.
- Swaine BR, Sullivan SJ. Longitudinal profile of early motor recovery following severe traumatic brain injury. *Brain Injury* 1996;10:347–366.
- Bowen A, Chamberlain MA, Tennant A, Neumann V, Conner M. The persistence of mood disorders following traumatic brain injury: A 1 year follow-up. *Brain Injury* 1999;13:547–553.
- Fann JR, Burington B, Leonetti A, Jaffe K, Katon WJ, Thompson RS. Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. *Archives of General Psychiatry* 2004;61:53–61.
- Fann JR, Katon WJ, Uomoto JM, Esselman PC. Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *American Journal of Psychiatry* 1995;152:1493–1499.
- Koponen S, Taiminen T, Portin R, Himanen L, Isoniemi H, Heinonen H, Hinkka S, Tenovuo O. Axis I and II psychiatric disorders after traumatic brain injury: A 30-year follow-up study. *American Journal of Psychiatry* 2002;159:1315–1321.
- Malia K, Powell G, Torode S. Personality and psychosocial function after brain injury. *Brain Injury* 1995;9:697–712.
- Hibbard MR, Uysal S, Kepler K, Bogdany J, Silver J. Axis I psychopathology in individuals with traumatic brain injury. *Journal of Head Trauma Rehabilitation* 1998;13:24–39.
- Bryant RA. Posttraumatic stress disorder and mild brain injury: Controversies, causes and consequences. *Journal of Clinical and Experimental Neuropsychology* 2001;23:718–728.
- Anstey KJ, Butterworth P, Jorm AF, Christensen H, Rodgers B, Windsor TD. A population survey found an association between self-reports of traumatic brain injury and increased psychiatric symptoms. *Journal of Clinical Epidemiology* 2004;57:1202–1209.
- Leon-Carrion J, De Serdio-Arias ML, Cabezas FM, Roldan JM, Dominguez-Morales R, Martin JM, Sanchez MA. Neurobehavioural and cognitive profile of traumatic brain injury patients at risk for depression and suicide. *Brain Injury* 2001;15:175–181.
- Oquendo MA, Friedman JH, Grunebaum MF, Burke A, Silver JM, Mann JJ. Suicidal behavior and mild traumatic brain injury in major depression. *Journal of Nervous and Mental Disease* 2004;192:430–434.
- Simpson G, Tate R. Suicidality after traumatic brain injury: Demographic, injury and clinical correlates. *Psychology and Medicine* 2002;32:687–697.
- Simpson G, Tate R. Clinical features of suicide attempts after traumatic brain injury. *Journal of Nervous and Mental Disease* 2005;193:680–685.
- Prigatano GP, Borgaro S, Baker J, Wethe J. Awareness and distress after traumatic brain injury: A relative’s perspective. *Journal of Head Trauma and Rehabilitation* 2005;20:359–367.
- Wagner AK, Hammond FM, Sasser HC, Wiercisiewski D. Return to productive activity after traumatic brain injury: Relationship with measures of disability, handicap, and community integration. *Archives of Physical Medicine and Rehabilitation* 2002;83:107–114.
- Morey LC. *Personality Assessment Inventory professional manual*. Odessa, FL: Psychological Assessment Resources; 1991.
- Piotrowski C. How popular is the Personality Assessment Inventory in practice and training? *Psychological Reports* 2000;86:65–66.
- Gass CS, Russell EW. MMPI profiles of closed head trauma patients: Impact of neurologic complaints. *Journal of Clinical Psychology* 1991;47:253–260.
- Burke JM, Smith SA, Imhoff CL. The response styles of post-acute traumatic brain-injured patients on the MMPI. *Brain Injury* 1989;3:35–40.
- Gass CS. MMPI-2 interpretation and closed head injury: A correction factor. *Psychological Assessment: A Journal of Consulting and Clinical Psychology* 1991;3:27–31.
- Gass CS. MMPI-2 interpretation and stroke: Cross-validation of a correction factor. *Journal of Clinical Psychology* 1996;52:569–572.
- Gass CS, Wald HS. MMPI-2 interpretation and closed-head trauma: Cross-validation of a correction factor. *Archives of Clinical Neuropsychology* 1997;12:199–205.
- Lachapelle DL, Alfano DP. Revised neurobehavioral scales of the MMPI: Sensitivity and specificity in traumatic brain injury. *Applied Neuropsychology* 2005;12:143–150.

29. Nelson LD, Elder JT, Tehrani P, Groot J. Measuring personality and emotional functioning in multiple sclerosis: A cautionary note. *Archives of Clinical Neuropsychology* 2003;18:419–429.
30. Rayls KR, Mittenberg W, Burns WJ, Theroux S. Prospective study of the MMPI-2 correction factor after mild head injury. *Clinical Neuropsychology* 2000;14:546–550.
31. Van Balen HG, de Mey HR, van Limbeek J. A neurocorrective approach for MMPI-2 use with brain-damaged patients. *International Journal of Rehabilitation Research* 1999;22:249–259.
32. Bagby RM, Nicholson RA, Bacchocchi JR, Ryder AG, Bury AS. The predictive capacity of the MMPI-2 and PAI validity scales and indexes to detect coached and uncoached feigning. *Journal of Personality Assessment* 2002;78:69–86.
33. Kurtz JE, Shealy SE, Putnam SH. Another look at paradoxical severity effects in head injury with the personality assessment inventory. *Journal of Personality Assessment* 2007;88:66–73.
34. Demakis GJ, Hammond F, Knotts A, Cooper DB, Clement P, Kennedy J, Sawyer T. The Personality Assessment Inventory in individuals with traumatic brain injury. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists* 2007;22:123–130.
35. Karlin BE, Creech SK, Grimes JS, Clark TS, Meagher MW, Morey LC. The Personality Assessment Inventory with chronic pain patients: Psychometric properties and clinical utility. *Journal of Clinical Psychology* 2005;61:1571–1585.
36. Forer S. Functional assessment instruments in medical rehabilitation. *Journal of Organization and Rehabilitation Evaluators* 1982;2:29–41.
37. Tombough TN. *Test of Memory Malinger (TOMM)*. Toronto: Multi-Health Systems; 1996.
38. Cohen J, Cohen P. *Applied multiple regression/correlation analyses for the behavioral sciences*. Hillsdale, NJ: Erlbaum; 1983.
39. O'Connor BP. SPSS and SAS programs for determining the number of components using parallel analysis and velicer's MAP test. *Behavioural Research Methods, Instruments, and Computers* 2000;32:396–402.
40. Tabachnick BG, Fidell LS. *Using multivariate statistics*. Northridge: HarpersCollins Publisher; 1989.
41. Beck AT, Steer RA, Brown GK. *Manual for the Beck Depression Inventory-II*. 2nd ed. San Antonio, TX: Psychological Corporation, Harcourt Brace & Company; 1996.
42. Sliwinski M, Gordon WA, Bogdany J. The Beck Depression Inventory: Is it a suitable measure of depression for individuals with traumatic brain injury? *Journal of Head Trauma Rehabilitation* 1998;13:40–46.
43. O'Connor P. Symptom management. *Advances in Neurology* 2006;98:227–255.
44. Tuokko H, Vernon-Wilkinson R, Robinson E. The use of the MCMI in the personality assessment of head-injured adults. *Brain Injury* 1991;5:287–293.
45. Christensen BK, Ross TP, Kotesek RS. Factor structure of the Beck Depression Inventory in a sample of persons with traumatic brain injury. *Journal of International Neuropsychological Society* 1995;1:121–186.
46. Ruocco AC, Swirsky-Sacchetti T, Choca JP. Assessing personality and psychopathology after traumatic brain injury with the millon clinical multi-axial inventory-III. *Brain Injury* 2007;21:1233–1244.
47. Greiffenstein FM, Baker JW. Comparison of premorbid and postinjury mmpi-2 profiles in late postconcussion claimants. *The Clinical Neuropsychologist* 2001;15:162–170.
48. Fann JR, Leonetti A, Jaffe K, Katon WJ, Cummings P, Thompson RS. Psychiatric illness and subsequent traumatic brain injury: A case control study. *Journal of Neurology, Neurosurgery, and Psychiatry* 2002;72:615–620.
49. Miller LJ, Donders J. Subjective symptomatology after traumatic head injury. *Brain Injury* 2001;15:297–304.
50. Morey LC. *Essentials of PAI interpretation*. New York: Wiley: John & Sons, Incorporated; 2003.