Table 1 (*continued*)

Item	Description	Factor loadings					
		Brazilian ACME			English ACME		
		COG	RES	DIS	COG	RES	DIS
10r	When my friends are having a good time I often get angry / *Frequentemente me sinto irritado(a) quando meus amigos estão se divertindo*	--	--	0.30	--	--	0.41
11r	People who are cheery disgust me / *Sinto desprezo por pessoas "alegrinhas"*	--	--	0.47	--	--	0.72
18r	I like making other people uncomfortable / *Gosto de deixar os outros desconfortáveis*	--	--	0.52	--	--	0.50
19r	I get a kick out of making other people feel stupid / *Sinto prazer em fazer com que os outros se sintam bobos*	--	--	0.66	--	--	0.57
20r	When my friends get angry I often feel like laughing / *Quando meus amigos ficam com raiva, muitas vezes sinto vontade de rir*	--	--	0.54	--	--	0.55
21r	Sometimes I enjoy seeing people cry / *Às vezes, sinto prazer em ver pessoas chorando*	--	--	0.53	--	--	0.69
26r	Sometimes it's funny to see people get humiliated / *Às vezes, é engraçado ver pessoas sendo humilhadas*	--	--	0.52	--	--	0.45
27r	If I could get away with it, there are some people I would enjoy hurting / *Se eu pudesse sair impune, há algumas pessoas que eu sentiria prazer em machucar*	--	--	0.64	--	--	0.45
36r	I admit that I enjoy irritating other people / *Admito que sinto prazer em irritar outras pessoas*	--	--	0.73	--	--	0.49

Cognitive empathy (COG) = 1r, 3, 4, 9, 14r, 15, 16, 25, 31r, 33, 34, 35r.
Affective resonance (RES) = 7, 8, 12r, 13r, 17r, 22r, 23, 24r, 28, 29, 30, 32r.
Affective dissonance (DIS) = 2r, 5r, 6r, 10r, 11r, 18r, 19r, 20r, 21r, 26r, 27r, 36r.
r = reverse scored item: items are administered on 5-point Likert scale ranging from strongly disagree (1) to strongly agree (5). On all three scales (including DIS), high scores indicate greater empathy.

concurrent validity. It is worth noting that the sample was disproportionately young (20% of participants older than 38), limiting the generalizability of the results. Future work should evaluate the ACME in older Brazilian samples and in clinical samples. The ACME-BP is a short, useful measure of empathy that shows evidence of internal consistency, test-retest reliability, and structural generalizability in a Brazilian sample.

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Return to work after severe traumatic brain injury: further investigation of the role of personality changes

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Although behavioral changes after traumatic brain injury (TBI) have been linked to work disability, research in the area is limited, especially in developing countries.[1] In severe TBI, behavioral sequelae mainly include depression and personality changes. The latter is marked by varying levels of apathy, disinhibition, aggression, and affective lability.[2] In a study published in the Brazilian Journal of Psychiatry,[3] we found that a diagnosis of

personality change due to severe TBI was associated with non-return to work independently from a range of sociodemographic, clinical and psychiatric variables. In the present study (Plataforma Brasil ethical approval 02832612.6.1001.0121), we expanded this investigation by examining the role of specific personality change symptoms in a new sample recruited three years later.

Forty-one survivors of severe TBI, who were working at the time of injury, were recruited from two intensive care units in the metropolitan area of Florianópolis, Brazil, and underwent psychiatric assessment during the chronic phase of the disease. Except for three cases, a close relative was available to describe the participants' behavior. Major depressive episode and substance abuse or dependence were diagnosed using the Mini International Neuropsychiatric Interview. Personality change was diagnosed according to DSM-5 criteria. Using the Overt Aggression Scale, aggression was defined as any incident of verbal or physical aggression in the last month. Disinhibition and general neuropsychiatric symptoms were defined using the Neuropsychiatric Inventory-Questionnaire. Apathy was defined using Robert's criteria and

Table 1 - Sociodemographic and psychiatric variables and return to work after severe traumatic brain injury

Variable	All participants n=41	Return to work		p-value
		No n=21 (51.2)	Yes n=20 (48.8)	
Sex				
Female	7 (17.1)	3 (14.3)	4 (20.0)	
Male	34 (82.9)	18 (85.7)	16 (80.0)	0.697
Age	32.0 (25.0-43.5)	35.0 (27.0-51.5)	29.5 (23.3-40.8)	0.140
Education				
Lower than high school	19 (46.3)	13 (61.9)	6 (30.0)	
High school or higher	22 (53.7)	8 (38.1)	14 (70.0)	**0.041**
Married or live-in partner				
No	31 (75.6)	15 (71.4)	16 (80.0)	
Yes	10 (24.4)	6 (28.6)	4 (20.0)	0.719
Glasgow Coma Scale*	6.0 (3.3-8.0)	6.0 (4.0-8.0)	6.0 (3.0-8.0)	0.739
Months since injury	28.0 (26.0-31.5)	28.0 (26.0-30.5)	28.0 (25.3-32.8)	0.865
Major depressive episode				
No	34 (82.9)	16 (76.2)	18 (90.0)	
Yes	7 (17.1)	5 (23.8)	2 (10.0)	0.410
Substance abuse or dependence				
No	35 (85.4)	18 (85.7)	17 (85.0)	
Yes	6 (14.6)	3 (14.3)	3 (15.0)	1.000
NPI-Q total†	5.5 (3.5-11.3)	8.0 (4.0-13.0)	4.5 (1.0-7.0)	0.062
Personality change				
No	26 (63.4)	10 (47.6)	16 (80.0)	
Yes	15 (36.6)	11 (52.4)	4 (20.0)	**0.031**
Aggression†				
No	23 (60.5)	9 (45.0)	14 (77.8)	
Yes	15 (39.5)	11 (55.0)	4 (22.2)	**0.039**
Disinhibition†				
No	31 (81.6)	16 (80.0)	15 (83.3)	
Yes	7 (18;4)	4 (20.0)	3 (16.7)	1.00
Apathy (Robert criteria)				
No	29 (70.7)	11 (52.4)	18 (90.0)	
Yes	12 (29.3)	10 (47.6)	2 (10.0)	**0.008**
Starkstein Apathy Scale†	11.5 (8.0-19.3)	13.5 (11.0-28.3)	8.5 (4.0-13.5)	**0.006**

Categorical variables are shown as frequency (percent) and were analyzed using a chi-square or Fisher's test. Numerical variables were non-normally distributed; they are shown as median (1st to 3rd quartiles) and were analyzed using a Mann-Whitney test.
Bold type denotes significance.
NPI-Q = Neuropsychiatric Inventory-Questionnaire.
* One missing in the group that returned to work.
† Two missing in the group that returned to work and one missing in the group that did not return to work.

the Starkstein Apathy Scale. The participants had not engaged in vocational rehabilitation prior to assessment.

Table 1 compares participants who did and did not return to work. Lower education, aggression, apathy, and a diagnosis of personality change were associated with non-return to work. These behavioral variables were significant predictors of non-return to work in separate multiple logistic regressions, including education as a covariate (personality change: adjusted odds ratio [AOR] = 4.4, p = 0.047, Nagelkerke R^2 [NR^2] = 0.25; aggression: AOR = 5.0, p = 0.037, NR^2 = 0.24; apathy according to Robert criteria: AOR = 7.9, p = 0.021, NR^2 = 0.31; Starkstein Apathy Scale score: AOR = 1.1, p = 0.024, NR^2 = 0.31).

Study limitations include the relatively small sample and the fact that work was not characterized (e.g., according to complexity level). We also did not perform a neuropsychological assessment of the participants, although it has been proposed that personality changes and cognitive impairment represent two aspects of the same phenomenon.[4]

Our results add to the literature by linking aggression and apathy to work disability in severe TBI. Given the importance of reproducibility in psychological research, it is also noteworthy that our previous findings were replicated. Apathy and aggression could serve as timely markers of attention for vocational rehabilitation after severe TBI, since they emerge early in the course of the disease and are easily observed by relatives and clinicians.[2,5] Larger longitudinal studies that comprehensively evaluate vocational and cognitive functioning and characterize distinct presentations of personality change may help identify accurate predictors of work disability, in addition to potential targets for interventions.

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Ditching candidate gene association studies: lessons from psychiatric genetics

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For decades, candidate association studies represented a cost-effective approach to investigate the genetic factors underlying heritable human traits, including psychiatric disorders. This methodology entailed testing variants in genes hypothetically related to phenotypes of interest, based on gene function and historical relevance. However, the genes selected for candidate association studies were typically gleaned from rare, familial instances of disease that do not necessarily translate to the wider population, or were based on weak empirical data that yielded false associations, often due to the presence of hidden confounders in the small samples analyzed. Another issue was that we currently do not know the full repertoire of functional variants regulating human gene expression and, therefore, candidate studies were unlikely to have selected the correct regulatory variants to test for association in the first place. Ultimately, this approach contributed to a publication bias in the literature and hindered the identification of the true biological risk mechanisms underlying psychiatric disorders. A lack of consistency and trust in the results arising from these studies led to replication studies and meta-analyses that altogether discredited most candidate gene associations in psychiatry.[1-3]