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RESEARCH ARTICLE

Mediating effects of impulsivity and alexithymia in the association between traumatic brain injury and aggression in incarcerated males

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Abstract

Studies suggest both alexithymia and impulsivity (partially) explain aggressive behavior in traumatic brain injury (TBI) patients, but none of these studies use both questionnaire and performance-based measures as recommended, nor simultaneously investigate both impulsivity and alexithymia. The available studies therefore likely miss part of the constructs of alexithymia and impulsivity, and do not comprehensively assess the mediating effects of both constructs in the relationship between TBI and aggression. A sample of $N = 281$ incarcerated individuals were recruited from Dutch penitentiary institutions, and completed the Buss Perry Aggression Questionnaire (aggression), BIS-11 (impulsivity) and Toronto Alexithymia Scale-20 (alexithymia) questionnaires, as well as a stop-signal task and an emotion recognition paradigm. Several multiple mediation analyses were conducted using structural equation modelling, to assess the viability of a causal theoretical model of aggression. The final planned models were the original models with a good fit with the data (comparative fit index > 0.95 , root mean square error of approximation and Standardized root mean square residual < 0.05), and results indicate that only questionnaire-based impulsivity mediated the relationship between TBI and aggression. TBI was unrelated to alexithymia, stop-signal or emotion recognition performance. Aggression was predicted by both alexithymia and impulsivity, but not by the performance measures. Post hoc analyses shows that alexithymia moderates the relationship between impulsivity and aggression. These results imply that aggressive incarcerated individuals showing impulsive behavior should be screened for TBI, since TBI is often overlooked or misdiagnosed, and indicate that both impulsivity and alexithymia are potential focus points for aggression reduction treatment in TBI patients.

KEYWORDS

aggression, alexithymia, emotion recognition, impulsivity, response inhibition, traumatic brain injury

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1 | INTRODUCTION

1.1 | Aggression

Aggression is considered a natural ability which may serve both adaptive and maladaptive purposes and is most often defined as "... any behavior directed toward another individual that is carried out with the proximate (immediate) intent to cause harm. In addition, the perpetrator must believe that the behavior will harm the target, and that the target is motivated to avoid the behavior" (Anderson & Bushman, 2002). The maladaptive manifestations of aggression are often explained by malfunctioning processes which usually channel and regulate aggression toward adaptive goals (Garofalo et al., 2018). These processes originate from an interplay of social, cognitive, developmental, and biological factors (Allen et al., 2018), and theories on aggression suggest it is more likely to occur when individuals feel threatened or irritated and are unable to exert sufficient inhibitory control (Bertsch et al., 2020).

Aggression assessment is often conducted through questionnaire assessment, and the Buss-Perry Aggression Questionnaire (AQ) (short form) is among the most popular measures. The instrument includes subscales for verbal and physical aggression as well as anger and hostility (Bryant & Smith, 2001). It's total score therefore reflects aggressive acts, as well as the degree to which individuals experience anger (e.g., a "negative" emotional reaction to perceived provocation (Novaco, 2011)) and exhibit hostility (e.g., a "negative" interpretation or evaluation of events or people) which are considered important contributing factors for aggression (Berkout et al., 2019; Robinson et al., 2020).

1.2 | Traumatic brain injury (TBI)

TBI involves an insult to the brain from an external mechanical force, leading to lacerations and bruising of the brain, often causing long-term behavioral changes (Pöttker et al., 2017). Individuals who have suffered TBI often show altered social behavior and higher levels of aggression and (violent) crime (Williams et al., 2018), anger and irritability (Hart et al., 2015). Nevertheless, TBI is often overlooked and/or misdiagnosed in general (Vaishnavi et al., 2009), and as an underlying cause of behavioral problems in forensic patients specifically (Williams et al., 2018). Mechanisms explaining the underlying relationship between TBI and aggression remain unclear and are in need of further study (see [Williams et al., 2018]). Especially, because a better understanding of these mechanisms could improve the diagnostic process, create additional treatment options and could be an additional focus for risk assessment instruments.

Recent studies investigated such possible mechanisms, identifying adverse psychological effects (Silver & Nedelec, 2020) and impaired executive functioning (Ryan et al., 2021)—especially inhibition and emotional control (Trajtenberg et al., 2023)—as mediating factors in the association between TBI and aggression. Research shows that TBI may

have a substantial negative impact on affective processing abilities (Fynn et al., 2021), increase irritability (Wood & Thomas, 2013) and reduce inhibitory control (Wood & Worthington, 2017). In fact, TBI patients may have particular problems exerting self-control in emotional situations (Wood & Worthington, 2017). This is in line with (reviews of) neuroimaging studies which show that frontotemporal limbic areas of the brain are specifically vulnerable for TBI (Bigler, 2013; McAllister, 2011), and reduced white matter integrity in TBI patients was associated with higher levels of aggression (Dailey et al., 2018). Sequelae of TBI, including aberrant emotional processing and/or a lack of control over certain impulses, may therefore increase aggression. Two characteristics closely related to aberrant emotional processing and/or a lack of control that have gained attention both because of TBI and as a precursor for aggression are alexithymia and impulsivity.

1.3 | Alexithymia

Alexithymia is a personality trait characterized by difficulties experiencing, identifying and describing emotions (Bermond et al., 2006), and impairments in this ability may lead to frustration and aggression due to the inability to communicate about feelings (Fonagy, 2003). There is ample evidence that individuals with TBI show higher levels of alexithymia (see (Fynn et al., 2021) for a recent meta-analysis), and that individuals with higher levels of alexithymia show higher levels of aggression (Hemming et al., 2019). Elements of alexithymia, such as identifying and describing emotions have been implicated in hostility biases and aggression (Smeijers et al., 2019) and previous studies in patients with acquired brain injury show that impairments in emotion recognition are associated with higher levels of concerning behavior, including aggression (Jorna et al., 2021). Based on these studies one would expect that alexithymia mediates the association between TBI and aggression, but there are no available studies assessing such mediating effects.

Although alexithymia is prevalent in offending populations, studies also show high prevalence rates of other psychiatric problems associated with impairments in emotion processing, including antisocial personality disorder (Bulten et al., 2009) and psychopathy (Kiehl & Hoffman, 2011). A recent meta-analysis indeed shows that higher levels of psychopathy are associated with higher levels of alexithymia (Burghart & Mier, 2022), although the association appeared stronger when *both* constructs were measured using self-report measures – indicating that the results could (partially and potentially) be explained by shared method variance. Other studies suggests that alexithymia is a transdiagnostic factor in affective disorders (Preece et al., 2022) and disorders characterized by empathy abnormalities (Valdespino et al., 2017). It is therefore not entirely clear how alexithymia relates to TBI and aggression in offending populations.

Despite the fact that alexithymia is characterized by problems with introspection (Brewer et al., 2016), most studies assessing alexithymia only use self-report questionnaires instead of additionally using performance-based tests (Rosch et al., 2022). Difference in

assessment is coined the primary cause of the debate on the definition and factor structure of alexithymia (Preece et al., 2017; Rösch et al., 2022). The Toronto Alexithymia Scale-20 (TAS-20) is most often used in questionnaire assessment (Schroeders et al., 2022), whereas facial recognition paradigms are often used as performance measures to assess difficulties in identifying feelings in TBI (Milders, 2019) and alexithymia (Grynberg et al., 2012). A review of such paradigms concludes that alexithymia is associated with the need for more time and/or information to make decisions about emotional facial expressions, and that difficulties arise when there is limited perceptual information. This appears to be a general deficit, which is not more pronounced in one emotion compared to another and results suggest that alexithymia is linked to impairments in facial recognition paradigms independently of clinical diagnosis (Grynberg et al., 2012). It is therefore suggested to use morphed facial expressions (blending two emotions) to decrease perceptual information, to facilitate the study of alexithymia through facial recognition paradigms.

1.4 | Impulsivity

Another line of inquiry suggests that TBI decreases inhibitory control and (thereby) increases impulsivity (Rochat et al., 2013), and that the increased impulsivity leads to aggression (Mosti & Coccato, 2018). Impulsivity definitions generally include tendencies relating to acting rapidly and/or with diminished forethought or consideration of negative consequences to oneself or others (Hamilton et al., 2015). TBI is one of the primary causes of impulsivity in forensic populations (Alford et al., 2020), and research suggest that impulsivity partially mediates the relationship between TBI and delinquency (Schwartz et al., 2017).

There is considerable debate about the conceptualization and measurement of impulsivity (Cyders & Coskunpinar, 2011; Strickland & Johnson, 2021). A well conducted meta-analysis indicates that self-report and performance based measures of impulsivity show a low to very low correlation, but that both measures correlate moderately with daily-life impulsive behavior (Sharma et al., 2014). In other words, both performance and questionnaire assessments independently predict problematic daily-life impulsive behaviors, making a strong case for using both types of measurements when assessing impulsive behavior (Sharma et al., 2014; Votruba et al., 2008).

Questionnaire assessment of impulsivity is often conducted using the Barratt Impulsiveness Scale (Stanford et al., 2009), whereas performance based assessment of impulsivity may either focus on rapid response inhibition (Hamilton et al., 2015), or choice impulsivity (Hamilton et al., 2015). A recent review shows that response inhibition is most consistently impaired in offending participants (Vedelago et al., 2019). Rapid response inhibition reflects a tendency towards immediate actions—without forethought—which are not in line with current situational demands (Hamilton et al., 2015). Impairments in rapid response inhibition likely lead to aggression through frustration, especially when failing to overpower emotional

impulses (Puiu et al., 2018). Response inhibition is often measured with stop-signal or go/no-go paradigms (Hamilton et al., 2015). These paradigms focus on either of two (partially) neurobiologically distinct types of rapid response inhibition; refraining from action initiation (go/no-go task) and stopping an ongoing action (stop-signal task) (see (Hamilton et al., 2015) for a review). However, the performance of these processes appears to be equally impaired in offenders relative to control groups (Vedelago et al., 2019).

1.5 | Self-report and behavioral assessment

In recent years studies indicate that both alexithymia (Preece et al., 2017; Rösch et al., 2022) and impulsivity (Cyders & Coskunpinar, 2011) are constructs which can be measured as relatively stable personality characteristics (e.g., traits)—often assessed through questionnaires—and as context dependent characteristics (e.g., states), which are often assessed through behavioral assessment and performance-based tests. Combining both types of measurement may increase the conceptualization of these constructs (Cyders & Coskunpinar, 2011), and other studies indicate that behavioral assessments are especially well suited for forensic populations since they: (1) are more engaging, (2) provide possibilities to assess low effort, (3) assess implicit and unconscious processes, and are (4) thereby less prone to malingering or socially desirable answers (Vedelago et al., 2019). Several researchers have therefore suggested using more behavioral assessments within a criminal justice setting, and such use is indeed increasing (de Ruigh et al., 2021; Haarsma et al., 2020; Norman et al., 2023).

1.6 | Alexithymia and impulsivity mediating the TBI aggression link

The mechanism underlying the association between TBI and aggression is poorly understood (Williams et al., 2018), but the above suggests that both alexithymia and impulsivity are often reported consequences of TBI which may mediate the relationship between TBI and aggression. A small body of research supports this assumption, for example, a longitudinal study using structural equation modelling (SEM) of questionnaire data indicates that TBI leads to aggressive offending through (interrelated) adverse psychological effects like increased impulsivity, anxiety and moral disengagement (Silver & Nedelec, 2020). A study in incarcerated adolescents showed that effortful control and negative emotionality mediate the relationship between TBI and violent behavior (Veeh et al., 2018), although the conceptualization was based on a temperamental theory of antisocial behavior, instead of impulsivity and alexithymia. Other studies in TBI patients show significantly higher levels of aggression and alexithymia, with difficulties describing feelings as a consistent predictor of aggression (Williams et al., 2019). Together, these studies suggest that alexithymia and impulsivity (partially) explain aggressive behavior in TBI patients, but

none of these studies use both questionnaire and performance-based measures as recommended, nor simultaneously investigate both impulsivity and alexithymia. Since alexithymia and impulsivity are likely correlated (Garofalo et al., 2018), it is important to simultaneously assess their effects to identify their unique contributions. The available studies likely miss part of the constructs of alexithymia and impulsivity, and do not comprehensively assess the mediating effects of both constructs in the relationship between TBI and aggression.

Importantly, the high prevalence of emotion and cognitive control difficulties in offending populations complicates the study of the TBI aggression relationship. They are core characteristics of psychiatric problems which are commonly found in antisocial populations, including antisocial personality disorder, psychopathy, and attention deficit hyperactivity disorder (Bulten et al., 2009; Fazel et al., 2016; Kiehl & Hoffman, 2011). Additionally, there are few studies with an appropriate (prospective) methodology to discern whether TBI was cause or consequence of aggression and/or an antisocial lifestyle (Williams et al., 2018). Preliminary results do indicate that aggression increases from pre- to post-injury (Cole et al., 2008) and that TBI is still related to aggression when correcting for violence as the cause of injury (Jansen, 2020).

1.7 | The current study

TBI is often overlooked as underlying cause of behavioral problems in forensic patients and the mechanisms through which TBI might cause TBI are poorly understood (Williams et al., 2018). This likely leads to suboptimal guidance and/or treatment and subsequently higher levels of recidivism (risk). A better understanding of these mechanisms could open possible avenues to tailor diagnostic procedures, treatment or supervision to these needs.

In the current study the mediating effects of alexithymia and impulsivity—measured through questionnaire and performance-based assessment—on the association between TBI and self-reported aggression are assessed in a sample of Dutch incarcerated individuals. A previous study already confirmed high prevalence of TBI within these incarcerated individuals and the association with aggression, but did not assess possible mechanisms underlying this relationship (Jansen, 2020). Based on the literature discussed above, three mediation analyses will be conducted using SEM with cross-sectional questionnaire and performance-based assessments of alexithymia and impulsivity as mediators for the relationship between TBI and aggression. Separate mediation analyses will be conducted for alexithymia and impulsivity, and one mediation analysis will include both constructs as simultaneous mediators.

The aim of the current study is to assess the viability of a causal theoretical model using cross-sectional data. Based on the literature outlined above, it is expected that those with a TBI history show higher levels of aggression, impulsivity, alexithymia, but lower levels of response inhibition (i.e., higher stop signal reaction time [SSRT]) and emotion recognition (i.e., lower accuracy). In turn, aggression is

predicted by higher levels of impulsivity and alexithymia, slower response inhibition and lower emotion recognition accuracy. The indirect effects of impulsivity, alexithymia, response inhibition and emotion recognition will (partially) mediate the association between TBI and aggression (see Supporting Information: 1 for a specification of models and path directions). Respecification is planned when the original model is rejected based on the goodness of fit indicators.

2 | METHODS AND MATERIALS

2.1 | Participants

A total of 281 incarcerated individuals were recruited from six different penitentiaries in the Netherlands and were eligible for participation when currently housed in the general population units or houses of custody. No participants were included from psychiatric wards or maximum-security units. All participants were residents for at least 3 weeks, because stress related to entering the prison system could otherwise have confounded the psychophysiological measurements (heart rate and skin conductance) which were also conducted (see [Den Bak et al., 2018]). Participants were recruited through poster advertisement, newsletter, and personal communication.

The study was approved by the Ethical Commission of the psychology department of the University of Leiden and participants signed an informed consent form after a thorough explanation of the research procedure—consistent with the declaration of Helsinki—before participating in the study. All participants were remunerated for their participation; a total of €10 was added to their in-prison bank-account.

2.2 | Sample size estimation

Associations investigated in this article are generally characterized by medium to large effects (see (Dimoska-Di Marco et al., 2011; Fynn et al., 2021; Grynberg et al., 2012)). Based on this assumption of medium effects, a total of $n = 118$ participants would need to be included to obtain $\alpha = .8$ power in the mediation analyses (Fritz & MacKinnon, 2007). Sample size determination for the SEM was set at a minimum of 15 cases per measured variable or indicator (Siddiqui, 2013). Since five measured variables are included in the analysis, a minimum sample size of $n = 75$ would be required for SEM in order obtain sufficient power.

2.3 | Instruments

2.3.1 | TBI history and demographic information

All participants were asked whether they had ever experienced “severe trauma to the head.” After receiving an affirmative response, participants could indicate how often this had occurred and what the

cause of the injuries was. These questions were part of a demographic questionnaire that was administered, which also assessed current age and education level (low, medium, and high).

2.3.2 | Aggression

The Dutch version of the AQ was administered, consisting of 12 items, each of which was answered on a 5-point Likert scale (Buss & Perry, 1992; Hornsveld et al., 2009). The questionnaire provides information on the general level of aggression (total score), and four subscales on physical and verbal aggression, as well as anger and hostility. Total score was used as dependent (exogenous) variable in the analysis, and showed good reliability (Cronbach $\alpha = .89$, 95% confidence interval [CI]: 0.86–0.91).

2.3.3 | Impulsivity

Barret impulsivity scale–11

To assess self-reported impulsivity, a Dutch version of the Barret Impulsivity Scale was administered. The Barrett Impulsivity Scale is a 30-item questionnaire to assess impulsive behaviors and preferences. All items are scored on a 4-point scale, ranging from rarely/never to almost always/always. No participants were excluded, and total score will be used as a continuous mediator the analyses. Total score of the BIS-11 showed good reliability (Cronbach $\alpha = .88$, 95% CI: 0.84–0.91).

Stop-signal task

To assess response inhibition, the freely available STOP-IT task was used (Verbruggen & Logan, 2008). During this response inhibition task, participants are instructed to press a corresponding key as fast as possible after either a square or circle is presented on the screen. In 25% of trials the symbol (square/circle) is followed by an auditory stop signal. The initial delay between presenting the symbol and this auditory signal is 250 ms, which is either decreased or increased by 50 ms after correctly or incorrectly inhibiting the response, respectively. Participants first completed a practice run, consisting of 32 trials, before three identical separate blocks of 64 trials were completed (see Supporting Information: 2). The SSRT is calculated across three blocks by subtracting the mean stop signal delay from the mean reaction time, and indicates inhibition performance (stopping an ongoing action) by using analysis software ANALYZE-IT (Verbruggen & Logan, 2008). A higher SSRT reflects lower behavioral inhibition performance. Participants were excluded when their chance of responding to no-go trial was more than 70%, and they responded to go trials less than 65% of the trials and missed 25% or more on no-signal trials ($n = 2$), additionally participants with an SSRT < 100 ms were excluded ($n = 3$) as this indicates poor understanding and/or compliance with task instructions. SSRT was included as a continuous moderator in the analyses.

2.3.4 | Alexithymia

TAS-20

The TAS-20 is a self-report questionnaire consisting of 20 items, which can be rated on a 5-point likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). Scores may range from 20 to a maximum of 200. Although, three subscales may be calculated from the individual items; (1) difficulty identifying feelings (DIF), difficulty describing feelings, and externally oriented thinking the total score was used as a continuous mediator in the analyses. Research shows excellent discriminant validity and high agreement with observer ratings of alexithymia (Bagby et al., 1994). No participants were excluded. Total score for the TAS-20 showed good reliability (Cronbach $\alpha = .79$, 95% CI: 0.74–0.83).

Emotion recognition task

To assess emotion recognition ability, a computerized facial recognition paradigm was used. Participants were shown morphed grey-scaled images of people showing happy, surprised, fearful, sad, disgusted or angry facial expressions (Fairchild et al., 2009). Each image morphed two different emotions together in varying intensities (90%/10%, 70%/30%, 50%/50%, 30%/70%, 10%/90%), and emotion combinations were limited to; angry-happy, disgust-angry, sadness-disgust, fear-sadness, surprise-fearful, and happy-surprised (see Supporting Information: 3). The task consisted of six blocks—including one practice block—and the complete stimulus set consisted of 30 different images (6 continua \times 5 morphed faces) was presented once in each block. Each image was presented for 5 s, and participants were then asked to indicate the dominant emotion by clicking on that emotion on a computer screen without any time limit and without feedback on their performance. Participants performing under chance level (17% correct) were removed ($n = 0$). Test-retest reliability was assessed in a small sample of $n = 10$ incarcerated individuals, with measurement moment separated by at least 2 weeks and showed high intra class correlation ($icc = 0.918$). Since a recent review concludes that alexithymia is associated with general deficit in emotion recognition, and did not find any specific emotion to be more affected (Grynberg et al., 2012), mean percentage of correct responses (across all emotions and intensities, excluding the morphed faces at 50%/50%) was used as a continuous mediator in the analyses.

2.4 | Analysis

2.4.1 | Data checks & imputation

Univariate and multivariate outliers were defined as deviating three standard deviations from the mean, and having a cook's distance of four times the mean. No univariate outliers were found, but four cases were identified as possible multivariate outliers. After inspection these were not removed. A total of $n = 5$ datapoints were removed due to poor response inhibition task compliance. The

assumption of multivariate normality was violated—as evidenced by significant Shapiro-Wilks test for univariate normality for aggression, emotion recognition, response inhibition and impulsivity. Linearity between variables was assessed and confirmed using scatterplots. Since our model also includes discrete variables, the lavaan “WLSMV” estimated was used (Rosseel, 2012).

A total of 13.4% of our data was missing, ranging from 0% to 52.6% between variables, see Supporting Information: 4. Missing data was imputed using default settings in the Mice package in RStudio, using $m = 50$ multiple imputed data sets. Multiple imputation is currently regarded as a state-of-the-art technique because it improves accuracy and statistical power relative to other missing data techniques (Buuren & Groothuis-Oudshoorn, 2011). Parameters of substantive interest were estimated in each imputed data set separately, and then combined into a single data set for further analysis. Analyses are performed on both original (nonimputed) data, and the imputed data set. Results from the planned analyses conducted on the original (nonimputed) data are presented in Supporting Information: 7.

2.4.2 | Bivariate statistics

To assess group characteristics for the total group, and separately for those with and without TBI, several wilcoxon rank-sum tests and χ^2 tests were performed for age, education, aggression, alexithymia, impulsivity, response inhibition and emotion recognition. Additionally, zero-order correlation analyses were performed between all variables of interest.

2.4.3 | Mediation analysis using SEM

To generate a theoretical model for the association between TBI and aggression, mediation analyses were performed using SEM with the lavaan package in Rstudio (Rosseel, 2012). Three separate mediation analyses were conducted, including (1) impulsivity and response inhibition, (2) alexithymia and emotion recognition and (3) impulsivity, response inhibition, alexithymia and emotion recognition as mediators for the association between TBI and aggression. Model selection procedures were based on Grace (2020). All variances were scaled, setting mean to 0 and variance to 1 and all endogenous variables are considered observed variables. The first models in each mediation analysis were created using theoretical assumptions, outlined in the introduction and formalised in Supporting Information: Table 1. χ^2 test, root mean square error of approximation (RMSEA), comparative fit index (CFI) and standardized root mean square residual (SRMR) were used to assess model fit. CFI values of 0.95 or higher show very good fit, and both RMSEA and SRMR values lower than 0.08 indicate adequate fit, whereas lower than 0.05 indicate a good fit (Little, 2013). Additionally, the expected cross-validation index (ECVI) was used to compare alternative models. A smaller ECVI value indicates better model fit (Browne & Cudeck, 1992).

For the model including impulsivity and response inhibition as mediators, TBI was an (discrete) exogenous variable with paths specified to all endogenous variables (i.e., aggression, impulsivity and response inhibition), aggression further received paths from mediator variables (impulsivity and response inhibition). Indirect effects were specified to assess mediating effects of each mediator. Age and education were included as exogenous control variables sending paths to all endogenous and exogenous variables (see Supporting Information: Table 1).

For the model including alexithymia and emotion recognition as mediators, TBI was an (discrete) exogenous variable with paths specified to all endogenous variables (i.e., aggression, alexithymia and emotion recognition), aggression further received paths from mediator variables (alexithymia and emotion recognition). Indirect effects were specified to assess mediating effects of each mediator. Age and education were included as exogenous control variables sending paths to all endogenous and exogenous variables (see Supporting Information: Table 1).

For the model including all mediator variables TBI was an (discrete) exogenous variable with paths specified to all endogenous variables (i.e., aggression, impulsivity, alexithymia, response inhibition and emotion recognition), aggression further received paths from mediator variables (impulsivity, alexithymia, response inhibition and emotion recognition). Impulsivity and alexithymia scores were allowed to covary. Indirect effects were specified to assess mediating effects of each mediator. Finally, age and education were included as exogenous control variables sending paths to all endogenous and exogenous variables (see Supporting Information: Table 1).

2.4.4 | Exploratory models

Two exploratory models were analysed: (1) one post hoc moderated mediation model was analysed, using the model with impulsivity and response inhibition as mediators (see above), but adding the moderation effect of alexithymia on the association between impulsivity and aggression. Alexithymia and impulsivity were again allowed to covary. Additionally (2) an alternative model including all mediator variables was conducted, while using aggression as an exogenous (continuous) variable and TBI as exogenous outcome variable. This alternative model was tested because previous studies indicate that aggression may also lead to TBI due to higher risk of injury when engaging in aggressive behavior.

3 | RESULTS

3.1 | Participant characteristics

Several wilcoxon rank-sum tests and χ^2 tests were performed for age, education, aggression, alexithymia, impulsivity, response

inhibition and emotion recognition. Results show that participants with TBI are generally more aggressive, and more impulsive at trait level, but no differences were found for response inhibition, emotion recognition, alexithymia, age or education, (see Table 1). Correlation analyses show significant correlation between TBI and aggression ($r = 0.30$ $p < .01$) and impulsivity ($r = 0.12$, $p < .05$), between aggression and alexithymia ($r = 0.40$ $p < .01$) and between impulsivity and aggression ($r = 0.57$, $p < .05$), alexithymia ($r = 0.40$ $p < .01$) and age ($r = -0.17$ $p < .01$) but no other correlations reached significance (see Table 2).

3.2 | Mediation results

The final model for impulsivity—including impulsivity (BIS-11) and response inhibition as mediators—was the original model (CFI = 1.0, RMSEA = 0.00 (90% CI: 0.00–0.16), SRMR = 0.02), and shows TBI is a significant predictor of aggression (total effect: $\beta = .48$, $b = 4.17$, $z(6) = 7.51$, $p < .001$), indicating that those incarcerated individuals with TBI show higher levels of aggression compared to those without (see Figure 1a). This effect is partially mediated by impulsivity (indirect effect: $\beta = .13$, $b = 0.97$, $z(6) = 3.87$, $p < .001$), but not by

TABLE 1 Characteristics table.

Characteristic	N	Overall, N = 281 ^a	no TBI, N = 154 ^a	TBI, N = 127 ^a	p Value ^b
Aggression	281	28 (19–37)	26 (18–32)	31 (23–40)	<.001
Emotion recognition	281	74 (65–84)	76 (65–84)	72 (63–84)	.50
Alexithymia	281	50 (44–59)	50 (43–59)	50 (44–58)	>.90
Response inhibition	281	278 (244–313)	278 (245–309)	277 (243–313)	>.90
Impulsivity	281	64 (56–70)	63 (56–69)	64 (57–72)	.14
Age	281	33 (26–45)	32 (25–44)	37 (27–45)	.13
Education	281				.10
High		31 (11%)	17 (11%)	14 (11%)	
Low		101 (36%)	47 (31%)	54 (43%)	
Middle		149 (53%)	90 (58%)	59 (46%)	

^aMedian (IQR); n (%).

^bWilcoxon rank sum test; Pearson's χ^2 test.

TABLE 2 Means, standard deviations, and correlations with confidence intervals.

Variable	M	SD	1	2	3	4	5	6
1. TBI	1.45	.50						
2. Aggression	28.52	10.59	.30**					
			[.19–.040]					
3. Emotion recognition	72.84	13.99	–0.03	–.04				
			[–.14 to .09]	[–.16 to .08]				
4. Alexithymia	49.91	10.99	0.02	.40**	–.03			
			[–.10 to .13]	[.30–.49]	[–.14 to .09]			
5. Response inhibition	284.73	72.25	–.03	–.03	.01	.09		
			[–.15 to .08]	[–.14 to .09]	[–.11 to .13]	[–.03 to .20]		
6. Impulsivity	63.72	11.86	.12*	.57**	–.03	.56**	–.05	
			[.01–.24]	[.48–.64]	[–.15 to .09]	[.47–.63]	[–.16 to .07]	
7. Age	36.15	12.25	.07	–.17**	–.02	.01	.05	–.15*
			[–.05 to .18]	[–.28 to –.05]	[–.14 to .10]	[–.10 to .13]	[–.07 to .16]	[–.26 to –.03]

Note. M and SD are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. Cumming (2014).

*indicates $p < .05$.

**indicates $p < .01$.

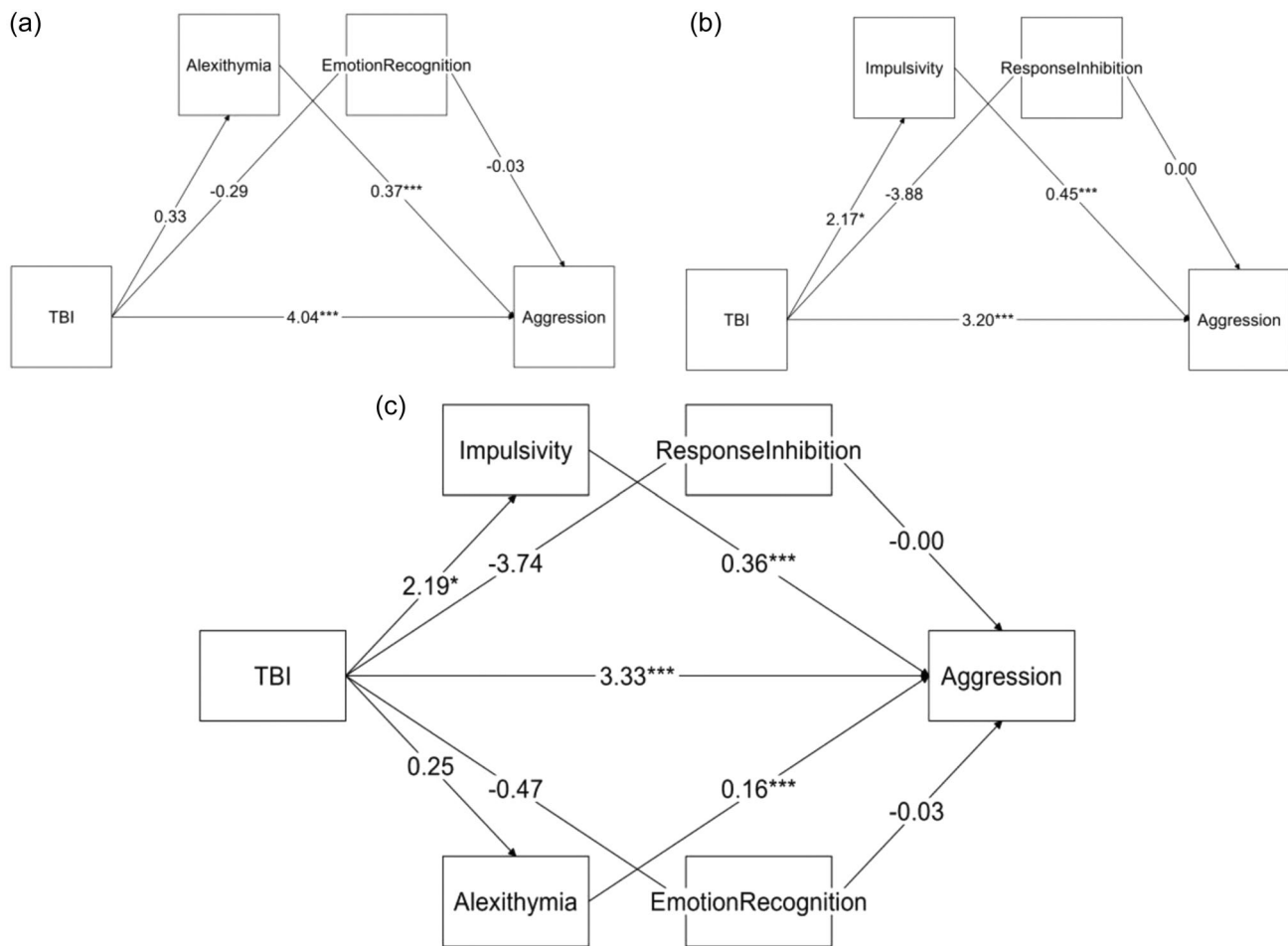


FIGURE 1 Planned SEM multiple mediation results. This figure shows the SEM multiple mediation results for (a) alexithymia and emotion recognition, (b) impulsivity and response inhibition and (c) alexithymia, emotion recognition, impulsivity and response inhibition. Path coefficients are unstandardized, and covariates age and education are omitted from the graphs. * $p < .05$, *** $p < .001$.

reponse inhibition (indirect effect: $\beta = .00$, $b = 0.00$, $z(6) = -0.19$, $p = .96$). TBI remained a significant predictor for aggression ($\beta = .36$, $b = 3.21$, $z(6) = 6.21$, $p < .001$). When assessing the different mediators in the model, results show that TBI is related to impulsivity ($\beta = .26$, $b = 2.17$, $z(6) = 3.67$, $p < .001$), and impulsivity is related to aggression ($\beta = .49$, $b = 0.45$, $z(6) = 11.15$, $p < .001$). TBI is not related to reponse inhibition ($\beta = -.05$, $b = -3.88$, $z(6) = -0.74$, $p = .46$), and response inhibition is not related to aggression ($\beta = .02$, $b = 0.00$, $z(6) = 0.01$, $p = .99$). A table with standardised and unstandardised path coefficients may be found in Supporting Information: 5. The final model therefore implies that the association between TBI and aggression is partially mediated through self-reported impulsivity, but not through response inhibition.

The final model for alexithymia—including alexithymia (TAS-20) and emotion recognition as mediators—was the original model (CFI = 1.00, RMSEA = 0.00 (90% CI: 0.00–0.12), SRMR = 0.01), and shows TBI is a significant predictor of aggression (total effect: $\beta = .39$, $b = 4.17$, $z(6) = 5.95$, $p < .001$), indicating that those incarcerated individuals with TBI show higher levels of aggression compared to those without (see Figure 1b). This effect is not mediated by

alexithymia (indirect effect: $\beta = .06$, $b = 0.12$, $z(6) = 0.40$, $p < .69$), nor by emotion recognition (indirect effect: $\beta = .00$, $b = 0.01$, $z(6) = 0.29$, $p = .78$). TBI remained a significant predictor for aggression ($\beta = .39$, $b = 4.04$, $z(6) = 6.18$, $p < .001$). When assessing the different mediators in the model, results show that TBI is not related to alexithymia ($\beta = .03$, $b = 0.33$, $z(6) = 0.39$, $p = .69$), but that alexithymia is related to aggression ($\beta = .39$, $b = 0.37$, $z(6) = 8.84$, $p < .001$). TBI is not related to emotion recognition ($\beta = -.03$, $z(6) = -0.34$, $p = .73$), and emotion recognition is not related to aggression ($\beta = -.02$, $b = -0.29$, $z(6) = -0.29$, $p = .78$). A table with standardised and unstandardised path coefficients may be found in Supporting Information: 5. The final model therefore implies that the association between TBI and aggression is not mediated through alexithymia nor through emotion recognition, but also that alexithymia is associated with aggression.

The final full model including all moderators was the original model (CFI = 1.00, RMSEA = 0.00 (90% CI: 0.00–0.06), SRMR = 0.02), and shows that TBI is a significant predictor of aggression (total effect: $\beta = .40$, $b = 0.4.17$, $z(21) = 5.95$, $p < .001$), indicating that those incarcerated individuals with TBI show higher levels of aggression compared to those without (see Figure 1c). This effect is partially

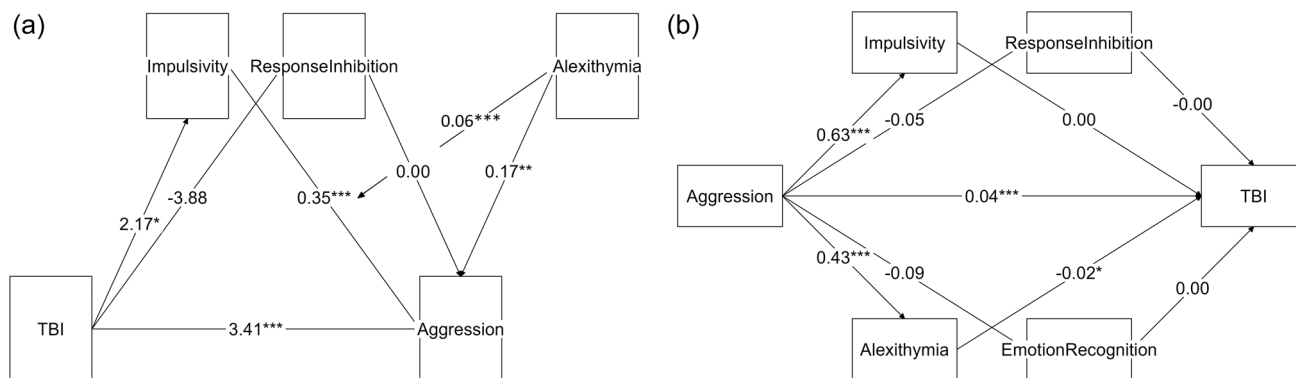


FIGURE 2 Exploratory SEM multiple mediation results. This figure shows the SEM multiple mediation results for the exploratory models: (a) including the moderating effect of alexithymia on the association between impulsivity and aggression, and (b) an alternative model where aggression is modelled as cause of TBI. Path coefficients are unstandardized, and covariates age and education are omitted from the graphs. * $p < .05$, *** $p < .001$. SEM, structural equation modelling; TBI, traumatic brain injury.

mediated by impulsivity (indirect effect: $\beta = .08$, $b = 0.78$, $z(15) = 2.67$, $p < .01$), but not by alexithymia (indirect effect: $\beta = .00$, $b = 0.04$, $z(15) = 0.31$, $p = .76$), response inhibition (indirect effect: $\beta = .00$, $b = 0.00$, $z(15) = 0.00$, $p = .99$) or emotion recognition (indirect effect: $\beta = .00$, $b = 0.01$, $z(15) = 0.42$, $p = .68$), see Figure 1. When assessing the different mediators in the model, results show that TBI is related to impulsivity ($\beta = .19$, $b = 2.19$, $z(15) = 2.60$, $p < .01$), but not to alexithymia ($\beta = .02$, $b = 0.25$, $z(15) = 0.31$, $p = .76$), response inhibition ($\beta = -.05$, $b = -3.74$, $z(15) = -0.73$, $p = .47$) or emotion recognition ($\beta = -.03$, $b = -0.47$, $z(15) = -0.47$, $p = .64$). Only alexithymia ($\beta = .17$, $b = 0.16$, $z(15) = 3.37$, $p = .001$) and impulsivity ($\beta = .40$, $b = 0.36$, $z(15) = 7.45$, $p < .001$) are associated with higher levels of aggression, whereas response inhibition ($\beta = .00$, $b = -0.00$, $z(15) = -0.00$, $p = .99$) and emotion recognition ($\beta = -.04$, $b = -0.03$, $z(15) = -0.74$, $p = .46$) were not. A table with standardised and unstandardised path coefficients may be found in Supporting Information: 5.

3.3 | Exploratory models

As post hoc addition, self-reported alexithymia was added as a moderator on the relationship between impulsivity and aggression (see Figure 2a). The final model including impulsivity and response inhibition mediators and alexithymia as moderator on the association between impulsivity and aggression was the original model (CFI = 0.99, RMSEA = 0.05 (90% CI: 0.00–0.12), SRMR = 0.04), and shows that TBI is a significant predictor of aggression (total effect: $\beta = .47$, $b = 4.24$, $z(10) = 6.56$, $p < .001$). This effect is partially mediated by impulsivity (indirect effect: $\beta = .07$, $b = 0.76$, $z(10) = 4.16$, $p < .01$), but not by response inhibition (indirect effect: $\beta = .00$, $b = -0.00$, $z(10) = -0.01$, $p = .99$). Alexithymia significantly moderated the association between impulsivity and aggression (moderating effect: $\beta = .07$, $b = 0.06$, $z(10) = 4.16$, $p < .001$). These results indicate that aggression in TBI patients is partially explained by increased impulsivity, and is more likely to occur when these individuals

exhibit higher levels of alexithymia. A table with standardised and unstandardised path coefficients may be found in Supporting Information: 6. When comparing the post hoc model with the impulsivity model, the fit indexes indicate that the post-hoc model was a slightly worse fit to the data ($\Delta CFI = 0.00$, $\Delta RMSEA = 0.0$, $\Delta SRMR = 0.014$, $\Delta ECVI = 0.051$).

Finally, an exploratory alternative model was analysed switching aggression and TBI. The original model was the final model (CFI = 1.00, RMSEA = 0.00 (90% CI: 0.00–0.06), SRMR = 0.02), and results indicate aggression is a significant predictor of TBI (total effect: $\beta = .39$, $b = 0.04$, $z(15) = 5.97$, $p < .001$), indicating that those incarcerated individuals with higher levels of aggression more often report TBI. This effect is partially mediated by alexithymia (indirect effect: $\beta = -.07$, $b = -0.01$, $z(15) = -2.01$, $p < .05$), but not by impulsivity (indirect effect: $\beta = .01$, $b = 0.00$, $z(15) = 0.25$, $p = .81$), response inhibition (indirect effect: $\beta = .00$, $b = 0.00$, $z(15) = 0.11$, $p = .91$) or emotion recognition (indirect effect: $\beta = -0.00$, $b = -0.00$, $z(15) = -0.09$, $p = .93$), see Figure 2b. When assessing the different mediators in the model, higher levels of aggression were associated with higher levels of alexithymia ($\beta = .41$, $b = 0.43$, $z(15) = 8.59$, $p < .001$), but those with higher levels of alexithymia were significantly less likely to report TBI ($\beta = -0.17$, $b = -0.02$, $z(15) = -2.01$, $p = .04$). A table with standardised and unstandardised path coefficients may be found in Supporting Information: 6. When comparing the alternative model with the full original model, the fit indexes indicate that the post-hoc model fitted the data equally well ($\Delta CFI = 0.00$, $\Delta RMSEA = 0.0$, $\Delta SRMR = 0.00$, $\Delta ECVI = 0.00$).

Together these results imply that only (self-reported) impulsivity mediated the relationship between TBI and aggression, that (self-reported) alexithymia is associated with aggression independent of TBI history, and that alexithymia moderated the association between impulsivity and aggression. Additionally, response inhibition nor emotion recognition were predicted by TBI or predictive of aggression. Analyses on non-imputed data are reported in Supporting Information: 7, but are generally similar to the results of the imputed data.

4 | DISCUSSION

In the current study we assess the mediating effects of alexithymia and impulsivity—measured through questionnaire and performance-based assessment—on the association between TBI and aggression in a sample of Dutch incarcerated individuals to assess the viability of a causal theoretical model. A previous study already confirmed high prevalence of TBI within these incarcerated individuals and the association with aggression, but did not assess possible mechanisms underlying this relationship (Jansen, 2020). It was expected that TBI history predicted higher levels of aggression, and that the indirect effects of impulsivity, alexithymia, response inhibition and emotion recognition would (partially) mediate the association between TBI and aggression.

The results of the different mediation models show that all originally planned models had a good fit with the data, and that impulsivity but not alexithymia partially mediated the relationship between TBI and aggression. Alexithymia was not associated with TBI but did predict aggression. These results were consistent when assessing impulsivity and alexithymia in separate models, and when combining both constructs in a single model. A post-hoc analysis revealed that alexithymia is a possible moderator in the association between impulsivity and aggression, suggesting that aggression is more likely to occur in individuals with higher levels of both impulsivity and alexithymia. This moderating effect is in line with studies suggesting that aggression is more likely to occur when individuals fail to exert control in emotionally challenging situations (Bertsch et al., 2020), and that TBI patients experience difficulties exerting self-control in emotional situations (Wood & Worthington, 2017). Since these results are based on cross-sectional data they cannot yield strong causal conclusions but do provide information on the viability of a causal theoretical model. These results are therefore in need of replication and further investigation in longitudinal research.

These findings fit well with previous studies showing that TBI is related to aggression (Williams et al., 2018) and impulsivity (Alford et al., 2020), and that impulsivity partially mediates the relationship between TBI and aggression (Schwartz et al., 2017). It was expected that performance based assessment of response inhibition would also be a significant mediator because (1) previous studies show impaired response inhibition in TBI patients (Dimoska-Di Marco et al., 2011), and that response inhibition is (2) often impaired in offenders (Vedelago et al., 2019) and (3) associated with (self-reported) aggression (Sun et al., 2020). Aggression is most likely to occur when individuals feel threatened or irritated and fail to exert inhibitory control (Bertsch et al., 2020), it is therefore possible that using neutral stimuli in our response inhibition task explains why no associations were found because this provides a measure of response inhibition in neutral conditions. Indeed, studies have suggested that reacting to such emotional stimuli results in increased difficulties and is important in assessing inhibitory control (Eben et al., 2020). A recent study showed that incarcerated individuals show impaired performance on an emotional go/no-go

task, although performance on this task was not associated with higher levels of aggression (Jansen et al., 2022). In conclusion, the added value of using emotion stimuli is proposed but its added value is currently unclear. Future studies might include response inhibition paradigms with emotional or threatening stimuli to assess inhibitory control over emotional stimuli.

Another possible explanation could be that shared variance within self-report (TAS-20, BIS-11 and AQ) and within performance-based measures (response inhibition and emotion recognition) explains these null results. This interpretation is partially supported by the zero-order correlations (Table 2) showing that self-report questionnaires correlate with one another, although performance-based measures do not correlate with each other (nor with the questionnaire data). On the contrary, alexithymia and impulsivity were allowed to covary within the final and exploratory models, which would account for shared method variance between these constructs. Studies on impulsive behavior show that both self-report and performance measures correlate moderately with daily-life impulsive behavior (Sharma et al., 2014), suggesting unique contributions of each in explaining daily life behavior. It is therefore not clear why response inhibition was unrelated to aggression or TBI. This question could potentially be answered by including a behavioral assessment of aggression, but this unfortunately was not conducted. Nevertheless, self-reported aggression has quite consistently been associated with response inhibition (Madole et al., 2020; Pawliczek et al., 2013; Sun et al., 2020), and response inhibition is a stronger predictor of reactive aggressive behavior than other cognitive processes (Tonnaer et al., 2016). In conclusion, shared variance is likely to only partially explain why self-report measures were associated with one another, whereas performance-based measures did not show any significant results.

Also counter to previous studies we did not show an association between TBI and emotion recognition, or between emotion recognition and aggression. The above mentioned argument on shared method variance could also explain these null results, although—again—the performance based measures did not correlate and previous studies do show that emotion recognition deficits are related to self-reported aggression (for a review, see [Smeijers et al., 2019]). Studies have even assessed the potential beneficial effects of training emotion recognition or response inhibition (Hubble et al., 2015), their results suggest that performance does improve but that it does not always result in less aggressive behavior (Kuin et al., 2020). In line with recommendations made by Grynberg et al. (2012), an emotion recognition paradigm with morphed faces was used to limit perceptual information available for emotion identification. Research shows that emotion recognition deficits are more likely to present themselves when perceptual information is limited. Within the analyses the mean percentage of correct responses (across all emotions and intensities) was used. This information could be further reduced by presenting morphed faces with higher degrees of uncertainty (i.e., also presenting faces morphed at 40%/60% and 20%/80%). It is possible that the emotion recognition problems are more likely to manifest in situations with a

higher degree of uncertainty, and future studies should include a wider range of uncertainty to assess these effects.

Although the final model does suggest that alexithymia is related to aggression, TBI does not predict alexithymia and therefore alexithymia does not explain why TBI leads to aggression the theoretical model. This does not fit well with a previous study implication emotion recognition as possible mechanism for problematic behavior in TBI patients (Jorna et al., 2021) nor with a recent meta-analysis which showed higher levels of alexithymia (measured with the TAS-20) in TBI patients (Fynn et al., 2021). Nevertheless, another recent review and meta-analysis indicates that basic social skills (including emotion recognition) are generally intact in children and adolescents with TBI, but more complicated processes (including theory of mind and pragmatic language) were significantly impaired (On et al., 2022). This would indicate that future studies on the association between TBI and aggression should also assess higher order aspects of social cognition.

Finally, an alternative model tested aggression as exogenous variable with a path towards TBI, and this model also showed a good fit with the data. Those incarcerated individual with higher levels of aggression are more likely to report TBI, and this is well in line with long-standing discussions on whether TBI should be seen as cause or consequence of aggressive behavior (Williams et al., 2018). Interestingly, this alternative model indicated that alexithymia partially mediated the association between aggression and TBI, showing that higher levels of aggression were associated with higher levels of alexithymia, but also that higher levels of alexithymia were associated with lower levels of TBI. This is counter to studies showing that patients with TBI generally show higher levels of alexithymia (Fynn et al., 2021), additionally studies show that aggression generally worsens after TBI (Cole et al., 2008), and that TBI is still a significant predictor of aggression after correcting for violence as the cause of TBI (Jansen, 2020). Although this alternative model fitted the data well and previous research indeed indicates that aggression may predate TBI, the literature provides a stronger theoretical basis for a model where TBI increases aggression.

4.1 | Limitations and future research

The current study assessed the viability of a causal theoretical model using cross-sectional data, but to establish causal relationships between the different variables these results should be replicated by both larger samples and by longitudinal research. Our final model showed partial mediation through self-reported impulsivity, therefore suggesting other mechanisms besides impulsivity are involved as well. The etiology of aggression is complex (Bertsch et al., 2020), multifaceted and dependent on social interactions (Smeijers et al., 2019). It is therefore likely our final model does not represent a complete model for explaining how TBI is related to aggression. Future studies should expand the model, and include other factors explaining aggression and TBI, like—for example—childhood adverse events (Madole et al., 2020).

There are several options to expand and deepen the current research. Currently only the total scores on the questionnaires were used, thereby omitting the subscales which could potentially provide more detailed information on specific processes. We also did not include a behavioral measure of aggression, which would have been a good addition to the study to assess whether shared method-variance introduced bias in the analysis. Future studies could investigate these processes in more detail.

TBI assessment was conducted through self-report, which is often thought to be problematic especially in forensic populations; however, previous studies show that TBI self-report in detainees is fairly reliable (Schofield et al., 2011), and the method is often used for TBI research in forensic populations (Williams et al., 2018). Nevertheless, a neuropsychological assessment of TBI status was not conducted and would likely have resulted in a more reliable indicator of TBI history. It is also possible that relatively less severe cases of TBI were included in the current study, because the applied TBI assessment did not—for example—systematically consider loss of consciousness which is considered a proxy for TBI severity. Participants did provide some additional information on how TBI occurred (see (Jansen, 2020)), indicating more severe TBI was likely present in our current sample (i.e., high-speed motor accidents, shot/stabbed in the head, etc.).

The planned SEM mediation analyses were run on both the original (non-imputed) data and the imputed data, and results are fairly consistent across all models and across both data sets. Some minor differences were found, for example, the indirect mediating effect of impulsivity did not reach significance in the original data ($p = .09$). This is likely the result of increased power in the analysis based on the imputed data set.

4.2 | Clinical implications

TBI is often overlooked as underlying cause of behavioral problems in forensic patients (Williams et al., 2018), which likely leads to suboptimal guidance and/or treatment and subsequently higher levels of recidivism (risk). The current study suggests that TBI is a likely (partial) explanation for aggressive behavior in incarcerated individuals exhibiting aggressive and impulsive behavior, especially when these individuals also show higher levels of alexithymia. This information could be used to identify aggressive individuals for TBI screening and assessment, since TBI is often overlooked or misdiagnosed. Additionally, the results indicate that both impulsivity and alexithymia are crucial for aggression in TBI patients and therefore open possible avenues to tailor treatment or supervision to these needs.

5 | CONCLUSION

In the current study we assess the mediating effects of alexithymia and impulsivity—measured through questionnaire and performance-based assessment—on the association between TBI and aggression in

a sample of Dutch incarcerated individuals. Only questionnaire-based assessment of impulsivity partially mediated the relationship between TBI and aggression. Alexithymia was not associated with TBI but did predict aggression. A post-hoc analysis revealed that alexithymia is a possible moderator in the association between impulsivity and aggression, suggesting that aggression is more likely to occur in TBI patients with higher levels of both impulsivity and alexithymia. These results imply that aggressive incarcerated individuals showing impulsive behavior should be screened for TBI, since TBI is often overlooked or misdiagnosed, and indicate that both impulsivity and alexithymia are potential focus points for aggression reduction treatment in TBI patients.

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CONFLICT OF INTEREST STATEMENT

The author declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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