

Investigating the Relationship Between Mild Head Injury, Physiological Arousal, and Neuropsychological Performance: Is there Potential for Residual Orbitofrontal Cortex Dysfunction with Respect to Processing Social and Emotional Information?

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STUDY 1

Background

Head injuries introduce sequelae of affective, behavioural, cognitive, and social dysfunction. Physical and cognitive deficits can often be compensated, but difficulties in social and emotional contexts tend to be more residual.

The ventral prefrontal cortex (vPFC) is involved in processing socio-emotional information and is highly susceptible to impact trauma during a closed head injury (CHI)¹¹. Moderate to severe vPFC injury relates to limitations in interpreting emotional signals from others (particularly negative affect)^{12,3,4} without gravely disrupting other cognitive abilities (e.g., reasoning).¹⁵

For milder injuries, asymptomatic university students' reports of head injuries resulting in an altered state of consciousness (ASC) have been shown to relate to subtle differences in neuropsychological performance and patterns of brain activation.^{16,71}

These findings, along with evidence from the animal literature⁸, suggest that metabolic disruption in the brain may be sufficient to produce performance problems in persons with no loss of consciousness (LOC) or contusions with DTI evidence demonstrating disruption to the ventral surface of the brain (limbic structures)⁹, the frontal poles¹⁰, and splenium of the corpus callosum.¹¹

Given that emotional awareness is particularly sensitive to impairment following head trauma and studies have found evidence of negative consequences in milder injuries, research is needed to clarify the role of MHI in emotion recognition capacity.

Purpose:

To investigate whether asymptomatic MHI in high functioning individuals is associated with differences in discriminating facial emotional expressions and if a history of MHI contributes to performance in affect recognition, independent of abstract and social reasoning.

Hypotheses:

(H1) Individuals self-reporting a history of MHI will be less successful in discriminating facial expressions, having particular difficulty interpreting affective displays of negative valence.

(H2) A history of MHI is expected to uniquely contribute to affect recognition performance, independent of abstract and social reasoning.

Methods

- 40 Brock University students (25 females, 15 males)
- 40% (n = 16) self-reported a history of MHI

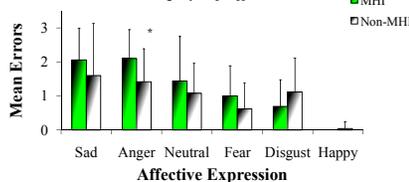
Measures

Neuropsychological measures included (i) Cognitive Flexibility¹², (ii) Abstract Reasoning¹³, (iii) Social Reasoning¹⁴, and (iv) Affect Recognition¹⁵.

Demographic Questionnaire to assess history of MHI: **Have you ever had a head injury resulting in an altered state of consciousness (including: vomiting, dizziness, seeing stars, confusion)?**

Results

Average Errors for Discriminating Facial Displays of Affect



(H1) The MHI group was generally worse at discriminating facial displays of affect ($F(1, 35) = 3.26, p = .08$; Interaction: $F(4, 156) = 2.39, p = .05$). Emotional displays of anger are most difficult for the MHI group to discriminate compared to the non-MHI group ($*p < .05$).

Variable	B	SE B	β	sr^2
Step 1				
Abstract Reasoning	0.14	0.07	0.31	0.12
Social Reasoning	0.06	0.07	0.14	0.04
Step 2				
MHI	-0.58	0.29	0.30*	0.11

Note: $\Delta R^2 = .13$ for Step 1; $\Delta R^2 = .08$ for Step 2
 * $p = .05$

(H2) History of MHI positively predicted the extent to which individuals incorrectly identify expressions of anger, over and above the influence of abstract and social reasoning.

General Discussion

Individuals self-reporting a history of MHI had more difficulty discriminating emotional signals from others, particularly when discerning facial displays of anger.

These results align with findings from the traumatic brain injury literature indicating that a dysfunctional vPFC is associated with limitations in discriminating negative affect and modulating affective responses, particularly in social/provocative environments.

MHI predicted one's proficiency in discriminating facial expressions of anger, over and above the capacity for abstract and social reasoning, suggesting that MHI uniquely contributes to emotional awareness, independent of other executive reasoning abilities.

STUDY 2

Background

Annual hospitalizations due to head trauma exceed 57 million cases worldwide, with over 80% of injuries receiving a classification of "mild"^{1,2}

The orbitofrontal cortex (OFC), a region subsumed by the vPFC, has been implicated as an important neural substrate for successful socio-emotional decision-making, modulating emotional arousal in response to anticipation of outcomes, and adjusting behaviours to adhere to contextual demands.^{3,4} Individuals with injury to the vPFC/OFC, despite being intellectually intact, have difficulty making decisions which have affective and/or social consequences.^{5,6}

While these persons have the capacity to emotionally respond to the feedback about their choices, they have attenuated emotional responses when anticipating potential consequences to an upcoming decision.^{13,5}

While the neuropsychological and physiological consequences of moderate to severe injuries to the vPFC/OFC are well researched, minimal investigation has been directed toward understanding the ramifications of MHI.

Purpose:

To extend previous findings of impaired emotion recognition in persons with MHI and investigate whether asymptomatic MHI in high functioning individuals is associated with differences in sympathetic arousal during decision-making, and if a history of MHI contributes to decision-making success.

Hypotheses:

(H1) It is expected that both groups will exhibit similar cognitive performance.

(H2) Self-reported history of MHI will be associated with poorer decision-making performance.

(H3) Individuals with a history of MHI will respond to environmental feedback (i.e., rewards and punishments) at comparable levels to their non-MHI counterparts; however, the MHI group will be underaroused relative to the non-MHI group when anticipating the potential consequences of future decisions.

Methods

- 44 Brock University students (33 females, 11 males)
- 41% (n = 18) self-reported a history of MHI

Measures:

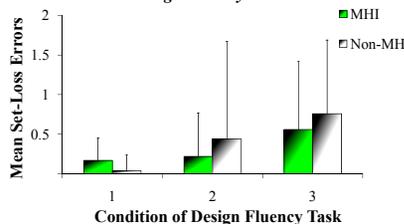
Neuropsychological measures included (i) Cognitive Performance – Design-Fluency⁷ and (ii) Decision-making – Iowa Gambling Task⁸.

Psychophysiological (sympathetic) arousal measured by Electrodermal Activity (EDA)⁹.

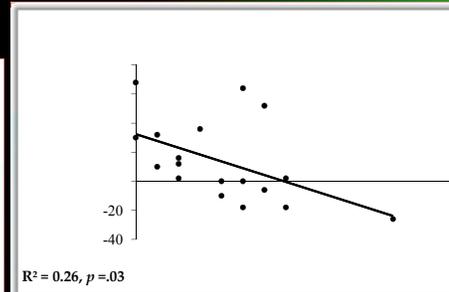
Demographic Questionnaire to assess history of MHI was the same as Study 1.

Results

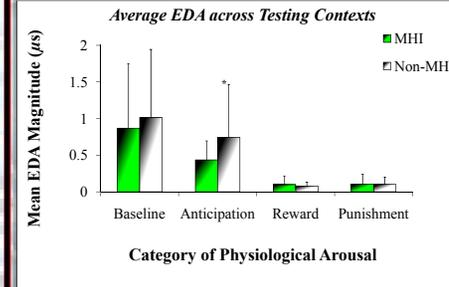
Errors Committed Across Conditions of the Design Fluency Task



(H1) Both groups responded similarly to the DF task demands. The number of errors made between the MHI and non-MHI groups did not differ and individuals in both groups made increasingly more errors as task difficulty increased (Condition 3 compared to Condition 1: $F(2, 82) = 6.17, p < .01$).



(H2) Overall IGT performance was similar between groups. However, a composite variable reflecting the objective severity of the trauma event indicates that injury severity was inversely related to decision-making success – as injury status increased, decision-making efficacy decreased.



(H3) EDA magnitude only differed between groups during the anticipatory stages of decision-making during which the MHI exhibited significantly less arousal than the non-MHI group ($*p < .05$).

General Discussion

Objective markers of injury severity indicated that increasingly more severe injuries were associated with the tendency to make fewer advantageous choices. Self-reported injury severity is, nominally, a marker of underlying neural trauma since this metric was related to performance on an objective neuropsychological test known to assess OFC function.

The MHI group was also emotionally underaroused prior to a decision/choice event, indicating that limitations in emotional processing can be observed both in terms of physiological arousal [Study 2] and appropriate processing of non-verbal affective cues from other humans [Study 1]. This dysregulation of sympathetic arousal could compromise an individual's sensitivity to impending consequences because, in some cases, these emotional signals serve to guide/bias choices and behaviour.

Conclusion and Implications

Injuries to the head resulting in an ASC are far from inconsequential as they are capable of producing detectable changes in physiological and neuropsychological functioning, well beyond the "concussive" phase.

Overall, the neurophysiological and neuropsychological profile of MHI can emulate features of more traumatic cases, even in those with high functional capacity.

Together, these findings support the argument that a history of MHI can differentially impact physiological and psychological mechanisms which sustain adaptive social decision-making, and more research needs to be carried out to better elucidate this profile.

References [Study 1]
 1)Hicklin et al. (2007). *Medicine*.
 2)Hicklin et al. (2002). *Brain Injury*, 16(3), 245-257.
 3)Hicklin et al. (2008). *J. of the International Neuropsychological Society*, 14, 111-125.
 4)Hicklin et al. (2008). *J. of Cognitive Neuroscience*, 20(4), 721-733.
 5)Hicklin et al. (2008). *Neuropsychology of Rehabilitation*, 19(2), 236-250.
 6)Hicklin et al. (1999). *Brain Injury*, 13(3), 151-172.
 7)Kaplan et al. (2001). *Brain and Cognition*, 43(1), 142-156.
 8)Coca et al. (2001). *J. of Anxiety Disorders*, 16, 228-232.
 9)Pollock et al. (2005). *J. of Head Trauma and Rehabilitation*.
 10)Rieder et al. (2010). *NARS 7th Annual Congress on Brain Injury*.
 11)Everson et al. (2010). *IBRA 9th World Congress on Brain Injury*.
 12)Ochs et al. (2010). *IAKEFS 5th Annual Congress on Brain Injury*.
 13)Everson et al. (1996). *Cerebral Cortex*, 6, 213-225.
 14)Everson et al. (2007). *Social and Cognitive Affective Neuroscience*, 2, 84-92.
 15)Everson et al. (1996). *Cerebral Cortex*, 6, 213-225.
 16)Everson et al. (2007). *IAKEFS 5th Annual Congress on Brain Injury*.
 17)Woolcher (1997). *IAKEFS 5th Annual Congress on Brain Injury*.
 18)Woolcher (1997). *IAKEFS 5th Annual Congress on Brain Injury*.
 19)Woolcher (1997). *IAKEFS 5th Annual Congress on Brain Injury*.
 20)Woolcher (1997). *IAKEFS 5th Annual Congress on Brain Injury*.
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