

**Visual strategies underpinning social cognition in  
traumatic brain injury**

GREENE, Leanne

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Visual strategies underpinning social cognition in traumatic  
brain injury

Leanne Greene

A thesis submitted in partial fulfilment of the requirements of Sheffield Hallam  
University for the degree of Doctor of Philosophy

October 2019

Candidate's statement

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2. None of the material contained in the thesis has been used in any other submission for an academic award.
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## **Acknowledgements**

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Firstly, I would like to thank my supervisor, Professor John Reidy, and my director of studies, Dr Lynne Barker, for their continued support, feedback and advice over the many years.

This thesis would not have been possible without the constant encouragement from my family. Mum and dad, thank you for providing me with all the opportunities that led me to complete this thesis and for your unconditional love and support, it means the world to me.

Thank you to my granny and granddad who consistently cheered me on during our Sunday evening telephone calls. To my siblings, Laura, Liam and Erin, thank you for always being there and helping me out, I would be lost without you all. And Matthew, for helping with stats and being a general sounding board.

To all my friends, thank you for putting up with my PhD rants and moans. In particular to The Sarah's (Johnston and Nolan), I feel truly grateful to call you my best friends and your support and encouragement has made this possible. To Lisa, Lucy, Gary and Miranda, I really value your friendships and hope to see more of you now this is finally over!

To Jacob, my best pal and partner in crime. This thesis was bigger than we thought so my deepest thanks for your help, patience, encouragement, support and cooking, these last few months would have been impossible without you.

*'But if only you could see yourself in my eyes,*

*You'd see you shine, you shine'*

Finally, my sincere gratitude to all of my participants, without you this research would not have been possible-thank you.

## **Publications and conference presentations**

---

Greene, L. (2018). The Forgotten Impairment. *The Psychologist*.

<https://thepsychologist.bps.org.uk/forgotten-impairment>.

Greene, L., Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). Exploring fixation patterns and social cognition after TBI. EuroSciCon Conference on Neurology & Neurological Disorders, Paris. DOI: 10.21767/2171-6625-C1-009

Greene, L., Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). (2018). Investigating visual strategies during the recognition of static and dynamic facial affect in TBI and control cohorts. EuroSciCon Conference on Neurology & Neurological Disorders, Paris. DOI: 10.21767/2171-6625-C1-008

Greene, L., Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). (2018). Investigating eye scan patterns in TBI and control groups when viewing static facial expressions. BPS Conference, Nottingham.

Greene, L., Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). Are you looking at me? Exploring eye scan patterns of TBI and control groups when viewing static and dynamic facial affect. *Frontiers in TBI, London and Creating Knowledge Conference, Sheffield*.

Greene, L., Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). Investigating male and female fixation patterns and social responses on a dynamic task: An eye-tracking study . In preparation for submission.

Greene, L. Barker, L. A., Reidy, J., Morton, N. & Atherton, A. (2018). Static and dynamic facial expression recognition after TBI: An eye-tracking study. In preparation for submission to *Cognitive, Affective, & Behavioral Neuroscience*.

## **Abstract**

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Impairments in social cognition after traumatic brain injury (TBI) are well documented but poorly understood (McDonald, 2013). Deficits in emotion perception, particularly facial affect recognition, are frequently reported in the literature (Babbage et al., 2011; Knox & Douglas, 2009), as well as mentalizing impairments and difficulty in understanding sincere and sarcastic exchanges (Channon, Pellijeff & Rule, 2005). To fully understand social impairments, both low-level and high-level processes must be explored. Few studies have focused on low-level perceptual processes in regards to facial affect recognition after TBI, and those that do typically use static social stimuli which lack ecological validity (Alves, 2013). This thesis employed eye-tracking technology to explore the visual strategies underpinning the processing of contemporary static and dynamic social cognition tasks in a group of 18 TBI participants and 18 age, gender and education matched controls.

The group affected by TBI scored significantly lower on the Movie for the Assessment of Social Cognition (MASC; Dziobek, et al., 2006), the Amsterdam Dynamic Facial Expression Set (ADFES; van der Schalk, Hawk, Fischer & Doosje, 2009), and The Assessment of Social Inference Test (McDonald et al., 2003). These findings suggest that, across a range of reliable assessments, individuals with TBI displayed significant social cognition deficits, including emotion perception and theory of mind, thus presenting strong evidence that social cognition is altered post-TBI. Impairments were not related to low-level visual processing as measured through eye-tracking metrics. This important insight suggests that social cognition changes post-TBI is likely associated with impairments in higher-level cognitive functioning. Interestingly, the group with TBI did display some aberrant fixation patterns in response to one static and one dynamic task but gaze patterns were similar between the groups on the remaining tasks. These non-uniform results warrant further exploration of low-level alterations post-TBI. Findings are discussed in reference to academic and clinical implications.

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## **List of acronyms**

TBI	Traumatic brain injury
DLPFC	Dorsolateral prefrontal cortex
VLPFC	Ventrolateral prefrontal cortex
OFC	Orbital frontal cortex
FEF	Frontal eye fields
SEF	Supplementary eye fields
PEF	Parietal eye fields
ADFES	Amsterdam Dynamic Facial Expression Set
MASC	Movie for the Assessment of Social Cognition
TASIT	The Awareness of Social Inference Test
EET	Emotion Evaluation Test
SI-M	Social Inference-Minimal
SI-E	The Social Inference-Enriched
IAPS	International Affective Picture System
GCS	Glasgow Coma Scale
ToM	Theory of Mind
PTA	Post Traumatic Amnesia
LOC	Loss of consciousness
FPC	Frontopolar cortex
MFC	Medial frontal cortex
DAI	Diffuse axonal injury
NICE	National Institute for Health and Care Excellence
CT	Computed tomography
MRI	Magnetic resonance imaging

# **1. Chapter one: Traumatic brain injury, brain anatomy and divisions of the frontal lobes**

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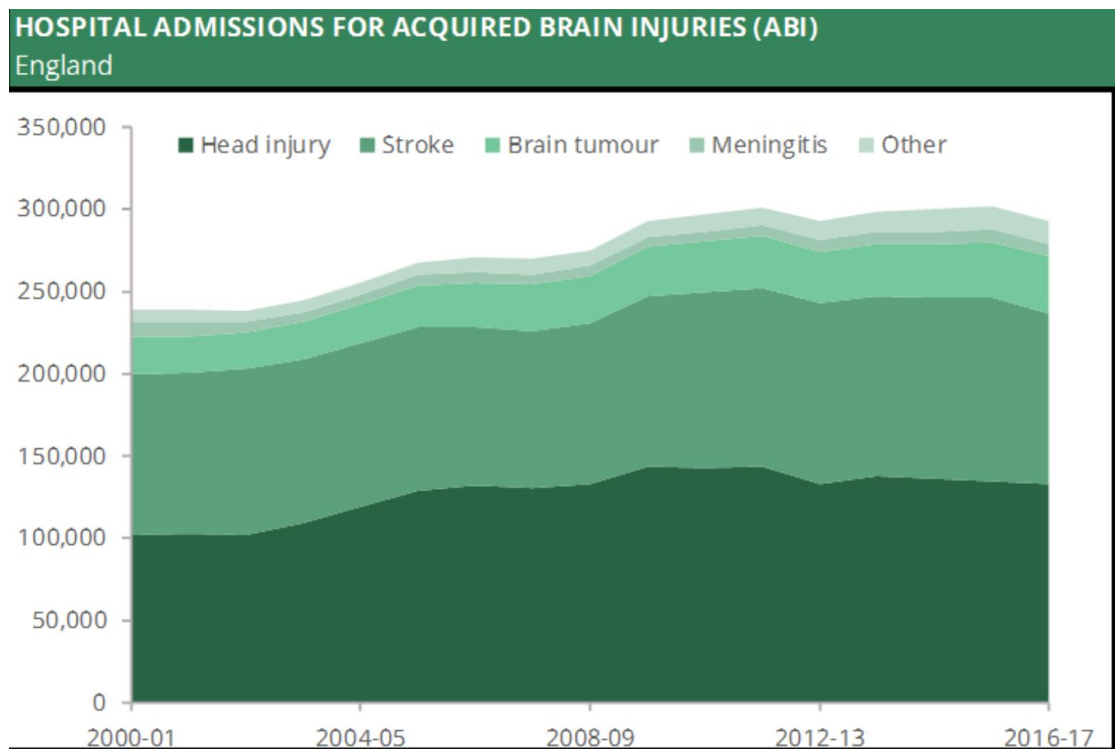
## **1.1. Chapter overview**

The first section of this chapter evaluates the prevalence and sequelae of traumatic brain injury (TBI). A brief outline of human brain anatomy is presented, with particular focus on the structure and function of the frontal cortex, an area susceptible to damage during TBI and associated with social cognition functions (Adolfi et al., 2017; Andreasen, Calarge, Calage, & O'Leary, 2008; Rushworth, Mars & Sallet, 2013).

## **1.2. Prevalence and sequelae of brain injury**

TBI is defined as a traumatic event which causes the brain to move rapidly within the skull, leading to damaged brain matter (Prins, Greco, Alexander & Giza, 2013). Precise brain injury statistics are currently unavailable, most likely due to; discrepancies in the diagnosis and classification of brain injury, variable definitions of acquired brain injury (ABI), the lack of distinction between ABI and TBI in the extant literature and socioeconomic divides (Dewan et al., 2018; Faul & Coronado, 2015; Roozenbeek, Maas & Menon, 2013). It is estimated that in 2015/16 between 293,000 and 301,500 individuals sustained an ABI in the UK (Figure 1), and although the majority of ABI's are classified as mild (Peeters et al., 2015; Yates, Williams, Harris, Round & Jenkins, 2006), up to approximately 1.4 million individuals are living with the long-term effects of brain injury with a cost to the UK economy of £15 billion per annum, equalling 10% of the National Health Service's (NHS) annual budget (Barber et al., 2018; Davis & Ings, 2015; Dinsmore, 2013). Worldwide, it is thought that approximately 69 million (95% CI 64-74 million) people per year experience a TBI, with research suggesting that it is a major if not leading cause of physical and mental disability and mortality in young adults living in western society (Dewan et al., 2018; Johnson & Griswold, 2017; Li, Zhao, Yu & Zhang,

2016). TBI has been coined by researchers as a ‘silent epidemic’ (Dewan et al., 2018) and an ‘overwhelming burden’ (Johnson & Griswold, 2017). It is estimated that by the year 2020, TBI will be the leading cause of death and disability worldwide for the general public (Humphreys, Wood, Phillips & Macey, 2013).



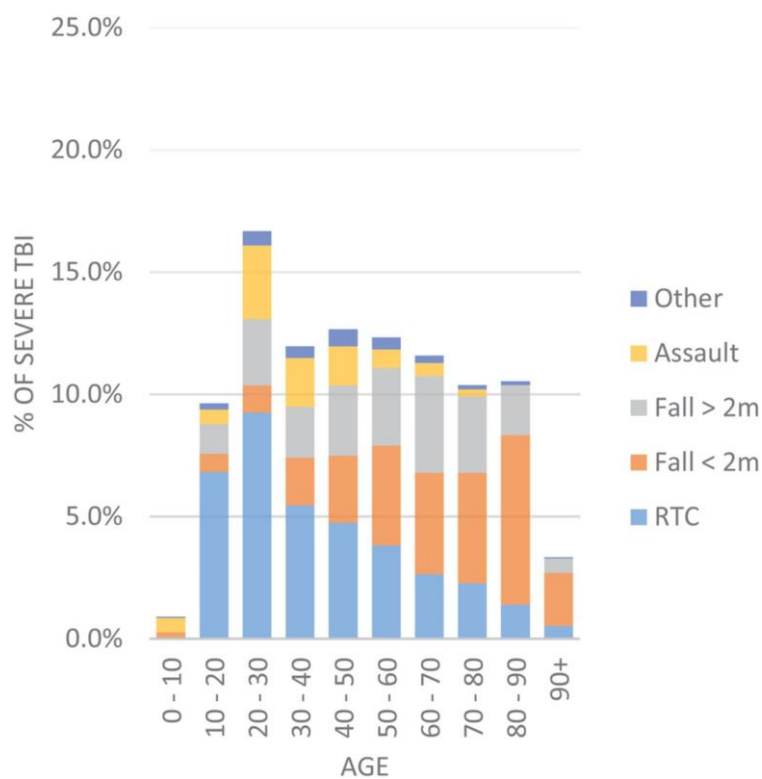
**Figure 1. Chart illustrating hospital admissions for acquired brain injuries in England between 2000 - 2017 (reproduced from Barber et al., 2018, p.5).**

Worldwide, the most common causes of TBI include falls (22-43%), assaults (30-50%) and road traffic accidents (25%) (Bruns & Hauser, 2003; Roozenbeek et al., 2013). Lawrence et al., (2016) conducted a prospective audit of brain injury epidemiology in England and Wales and reported that the most frequent mechanisms of injury were falls in the elderly and road traffic accidents in younger adults. Marshman, Jakabek, Hennessy, Quirk and Guazzo (2013) reported that the majority of TBI cases occur in males aged between 16-24 years as a result of motor vehicle accidents, assaults, falls, and sporting accidents. Indeed, the authors proposed that males are up to three times more likely to sustain a brain injury compared to females and this

increases to five times more likely if males are aged between 15-29 (Greenwald, Burnett & Miller, 2003; Williams, 2012). However, this gender bias reverses with regards to elderly individuals over 65 where females are more likely to be admitted to the hospital after a head injury. This gender effect has been reported from several countries including the UK, USA, Australia and Finland (Fletcher, Khalid & Mallonee, 2007; Harvey and Close, 2012; Kannus, Palvanen, Niemi & Parkkari, 2007; Shinoda-Tagawa & Clark, 2003; Shivaji, Lee, Dougall, McMillan & Stark, 2014). Research on gender reversal with age is scant but several theories as to why elderly females acquire more brain injuries than males include the hypotheses that females tend to outlive males on average by 5.8 years longer (Rochelle, Yeung, Bond & Li, 2015), they tend to combine multiple medications, and often live alone (Ebrahim & Kalache, 1996). There is a rising prevalence of TBI in the elderly population as a result of falls, likely due to an increase in the ageing population (Gaastra et al., 2016; Korhonen et al., 2013). UK research infers that elderly trauma may soon be on par with young male statistics (Kehoe, Smith, Edwards, Yates & Lecky, 2015). Lawrence et al., (2016) reported that across all TBI severities in England and Wales, there was a unimodal age distribution, which peaked between the ages of 80 and 90 and represented one in five of TBI's, usually because of falls from under two metres high. Severe TBI's accumulated a smaller peak (estimated to be around 15% of all TBI cases) and is more common in younger cohorts (age 20-30), usually as a result of road traffic accidents and assaults. From the total cohort (15,820) reviewed by the authors, Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974, discussed in detail in the latter part of this chapter) scores indicated that 68% of cases were mild, 6% moderate, and 26% severe (Figure 2).

Due to developments in medicine, diagnostic evaluation, and acute care pathways, patient outcome is more positive now post-TBI than in the past (Gerber, Chiu, Carney, Härtl & Ghajar, 2013). The majority of individuals with TBI will go on to have relatively normal lifespan expectancy (Harrison-Felix et al., 2015; Harrison-Felix, Whiteneck, DeVivo, Hammond & Jha,

2004), although this does depend on extraneous factors such as gender and age at injury (Brooks, Shavelle, Strauss, Hammond & Harrison-Felix, 2015; Ziebell et al., 2017). Nevertheless, many TBI survivors will need some form of support, either professional or family orientated after injury which can be costly to public health services (Fountain, Kolas, Laing & Hutchinson, 2017; Humphreys et al., 2013), as well as creating a strain on social relationships (Backhaus et al., 2016; Humphreys et al., 2013; Temkin, Corrigan, Dikmen & Machamer, 2009).



**Figure 1. Graph outlining the number of individuals with TBI by age and cause of injury from the Lawrence et al., (2016) audit of England and Wales (reproduced from Lawrence et al., 2016, p.3). Abbreviations: Road traffic accident (RTA), traumatic brain injury (TBI).**

Many TBI survivors experience some form of physical impairment post-injury, for example, epilepsy (Lowenstein, 2009), dizzy spells (Chandrasekhar, 2013), and headaches (Lucas, Hoffman, Bell & Dikmen, 2014) which often require life-long and costly pharmaceutical

treatments as well as limiting the individual's physical abilities (e.g. the ability to drive). TBI survivors may also experience a range of cognitive, social and emotional impairments, which restrict independent living in the community, even if those impairments are mild (Drake, Gray, Yoder, Pramuka & Llewellyn, 2000; Vikane et al., 2016). Ponsford et al., (2014) undertook a longitudinal outcome follow-up of 141 individuals with TBI at two, five and ten years post-injury. Fatigue, balance, cognitive, communication, behavioural and emotional problems were all reported over the ten years with over 40 per cent of the cohort requiring more support than they did pre-injury. Merely 50 per cent had returned to participating in leisure activities, less than half were employed, and 30 per cent admitted difficulties in personal relationships. Reports from survivors of TBI and their families suggested that it was the social and emotional impairments after TBI which had the largest and most adverse impact on life quality (Fleminger & Ponsford, 2005; Humphrey et al., 2013; McDonald, Flanagan, Rollins & Kinch, 2003; Ubukata et al., 2014). It is often the case that TBI survivors live with their families and do not return to school or work after the injury, creating physical, psychological and financial burdens on the relatives who care for the patient (Humphrey et al., 2013; Ponsford et al., 2014). Social relationships are often negatively affected after a TBI and many survivors lose close friendships and experience marital breakdowns (Bodley-Scott & Riley, 2015; Carlozzi et al., 2015). This, in turn, leads to social isolation and risk of developing mood disorders, such as depression and anxiety, with an increased risk of suicide (Bornhofen & McDonald, 2008; Fralick, Thiruchelvam, Tien & Redelmeier, 2016).

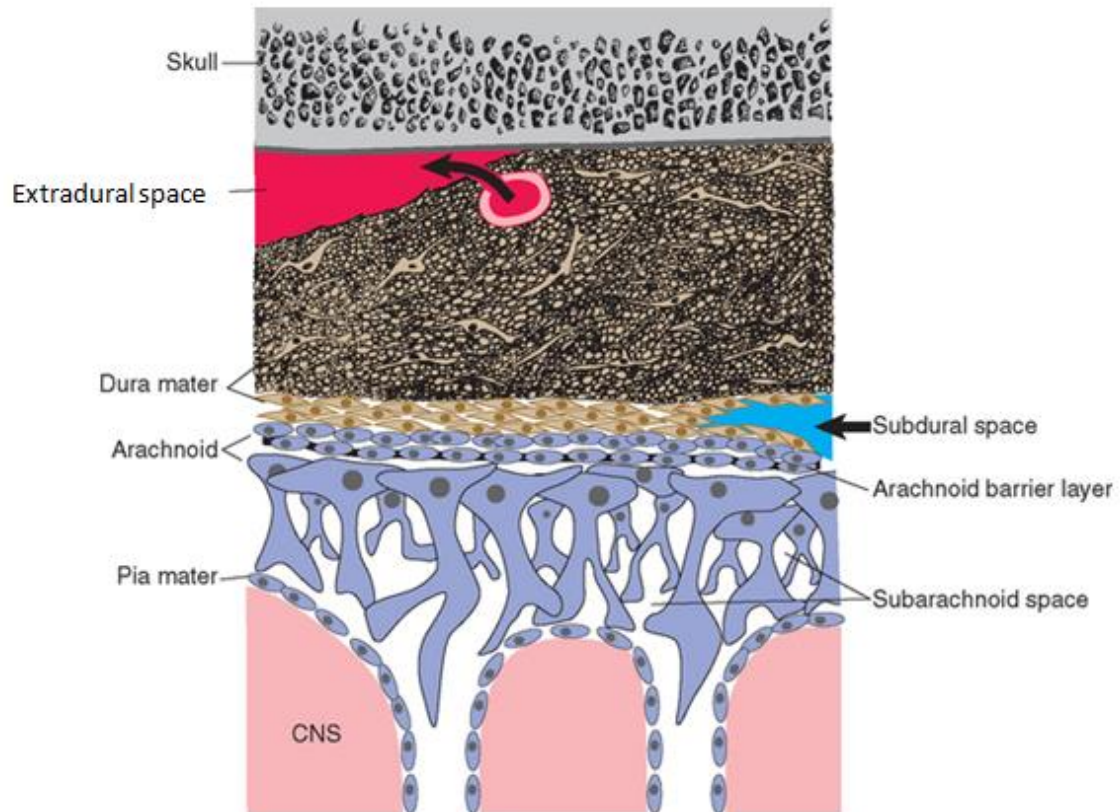
In summary, TBI is a major economic, social, medical and public health concern (Roosenbeek et al., 2013; Humphrey et al., 2013). Mechanisms underpinning the social and emotional deficits after TBI are not fully understood and it is not known whether these impairments are due to a disruption of complex higher-level cognitive functions or lower-level functions, such as visual processing. Research investigating low-level visual processing after TBI is scant and

little is known about how these visual processes relate to social functioning. If low-level visual processes are disrupted after TBI then this could lead to the incapacity to capture, monitor and evaluate dynamic social stimuli, as well as having a simultaneous effect on higher-order cognitive functions. Future research might focus on the fundamental automatic and controlled elements of visual processing to disentangle at which level of function the primary deficits of TBI occur.

### **1.3. Introduction to brain anatomy**

The human brain is encased within the skull to protect it from outside forces. The brain does not touch the overlying bone of the skull since it is protected by three meninges (layers of protective tissue) which ensure the brain is stabilised during head and body movements. The three meninges are the dura mater, the arachnoid membrane, and the pia mater (Figure 3). The dura mater is tough and adheres to the inner skull and the arachnoid. The arachnoid and pia are both thin and merge so subtly that it is difficult to distinguish them. The arachnoid membrane and the pia mater are separated by cerebrospinal fluid (CSF), a layer of salty liquid which provides a buoyant environment that helps decrease the distortion of the brain from various forces (Ivancevic, 2009). Despite this mechanical protection, pathology to brain matter still occurs as a result of rotational forces and the forwards and backwards motion of the brain during impact, leading to coup-contrecoup injury and shearing of dura mater and neural tissue (Bigler, 2013; Drew & Drew, 2004; Fujiwara, Schwartz, Gao, Black and Levine, 2008; Kolb & Wishaw, 1990; McDonald, Togher & Code, 2013; Taylor & Ford, 2009; Zappala, de Schotten & Eslinger, 2012). It should be noted that two 'potential spaces' (defined as space which can appear between two structures that are normally pressed together) are affiliated with the dura as a result of pathology, the extradural space (separation from the cranial bones and

the endosteal layer of the dura mater) and the subdural space (separation of the arachnoid mater from the dura mater).



**Figure 2. Separate levels of the cranial meninges (reproduced from Nolte, 2008, p.92).**

At a simple anatomical level, the central nervous system (CNS) contains two categories of cells; neurons which mediate information processing and signalling, and glial cells which provide a supportive function. Many articles and books state that the human brain contains more than 100 billion neurons and up to 10 times that many glial cells yet no original reference supports this fact, leading to the erroneous assumption that the cellular composition of the human brain has been determined. Evidence from Azevedo et al., (2009) and Herculano-Houzel (2009) challenged this popular fact and suggested that 100 billion neurons are an overestimation and that it is probable that glial cells constitute approximately 50% of human brain cells. The CNS can be further divided into grey and white matter. Grey brain matter is



rich in nerve cell bodies which consist of unmyelinated axons, dendrites, and glial cells (Swanson & Bota, 2010). White brain matter consists mainly of long myelinated axons.

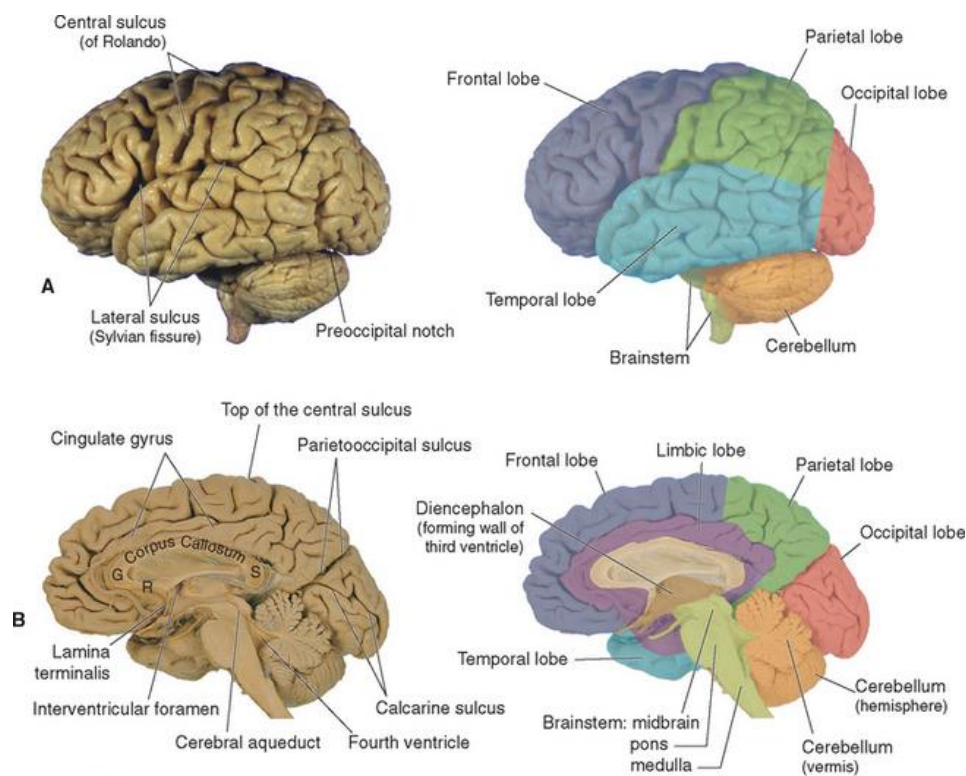
The brain can be divided into three main structures or vesicles; rhombencephalon (hindbrain), mesencephalon (midbrain) and prosencephalon (forebrain) (Blumenfeld, 2010). The rhombencephalon can be further divided into the myelencephalon (medulla oblongata), metencephalon (pons) and the cerebellum (Figure 4). Collectively, the medulla oblongata, pons and cerebellum are referred to as the brainstem. The prosencephalon can be subdivided into the diencephalon and the telencephalon. The diencephalon section is concealed from view by the cerebral hemispheres and contains the thalamus, hypothalamus, and other smaller structures such as the epithalamus and subthalamus. The telencephalon comprises the cerebral hemispheres containing fissures, sulci, and gyri and can be subdivided into four distinct lobes; the occipital, temporal, parietal and frontal lobes. The furrows on the surface of the hemispheres are divided into two; fissures which are prominent and deep, and sulci which are shallower (Standring, 2015). The longitudinal fissure separates the two cerebral hemispheres while cerebral commissures provide connecting tracts for the two hemispheres to communicate. The frontal and parietal lobes are separated by the rolandic or central fissure. The frontal and parietal lobes are separated from the temporal lobes by the Sylvian or lateral fissure. The parieto-occipital fissure or notch, on the medial surface of the cortex, separates the parietal and the occipital lobes. The different brain structures are selectively vulnerable to TBI, for example, the brainstem is postulated to be less susceptible to injury due to its location, providing a degree of protection against trauma-induced forces (Adams, Graham & Jennett, 2000; Valko et al., 2016). Moreover, the tissue property of the brainstem, being rich in sturdy white matter, could allow the structure to flex during TBI, thus reducing damage (Valko et al., 2016). In contrast, the frontal lobes are predisposed to damage during TBI as they are subjected to compressive pressure when they make contact with the bony protuberances of the skull (Bigler, 2013). The

different structures of the brain mediate diverse and distinct cognitive functions which may be diminished or abolished by brain trauma (Rabinowitz & Levin, 2014).

The cerebral cortex is divided into two, the allocortex consisting of three to four cellular levels that include structures such as the hippocampus and neocortex (isocortex). These consist of six cellular levels that encompass the four lobes of the human brain. The neocortex is the most recently evolved region of the brain comprising of approximately 90% of the human cerebral cortex (Coqueugnot, Hublin, Vellon, Houët & Jacob, 2004; Northcutt and Kaas, 1995). The region displays a substantial level of individual macroscopic structural variability concerning secondary structures (e.g. operculum; Idowu, Soyemi & Atobatele, 2014), posing a challenge when trying to ascertain functional and structural relationships from neuroimaging experiments. The continuous development of superior imaging techniques is allowing scientists to study the macro-structure of the human brain and large scale neural data projects (e.g. the Human Connectome Project) could potentially lead to changes in the way the human brain is mapped. For example, Kruggel (2018) applied a data-driven analytic approach to assess structural magnetic resonance imaging (MRI) data, proposing that subdividing the neocortex into communities, rather than lobes, could provide a more meaningful overview of vascular, anatomical, and functional brain connectivity.

The human evolution of the neocortex is associated with the development and advancement in higher cognitive function (Lui, Hansen & Kriegstein, 2011), particularly social cognition (Dunbar, 2009) and executive function (Morton & Barker, 2010; Taylor, Barker, Reidy & McHale, 2013). Executive function is a term used to refer to a multifaceted psychological construct that incorporates the formation, maintenance and shifting of mental sets to generate, motivate and alter goals or plans in response to changing contingencies (Suchy, 2009). Research findings have reported strong associations between frontal lobe regions and uniquely

human social-cognitive behaviours (Adolfi et al., 2017; Forbes & Grafman, 2010; Rushworth et al., 2013), including speech production and language comprehension associated with Broca's area (Skeide & Friederici, 2016), moral judgement (Cameron, Reber, Spring & Tranel, 2018; Rowley, Rogish, Alexander & Riggs, 2017), and theory of mind (ToM; Powell, Grossi, Corcoran, Gobet & Garcia-Fiñana, 2017; Saxe & Baron-Cohen, 2006; Yeh, Tsai, Tsai, Lo & Wang, 2017) which is the ability to attribute and understand the mental states of others (Premack & Woodruff, 1978).



**Figure 3. The localisation of the four human lobes (frontal, parietal, temporal and occipital), brainstem and cerebellum and some of the main sulci and gyri (A) lateral view, (B) medial view (reproduced from Nolte, 2008, p.92).**

Social behaviours are thought to be vital underpinnings of human social achievement and culture (Cummings and Miller, 2007; Frith and Frith, 2010; Simpson & Beckes, 2010). One theory proposes that a relative and disproportionate enlargement of the frontal lobes during

evolution led to superior social abilities in humans over other species (Powell, Lewis, Roberts, Garcia-Finana & Dunbar, 2012). Although the frontal lobes are relatively large in humans, recent research has challenged this concept (Barton & Venditti, 2013a, 2013b; Semendeferi, Lu, Schenker & Damasio, 2002). These objections are commonly based on the fact that prior research had less precise methodologies to measure the delineation of brain region boundaries compared to modern technologies today (Risberg & Grafman, 2006). Barton and Venditti (2013a) analysed five data sets with rigorous scaling and phylogenetic techniques and reported that the frontal lobes were an expected size relative to the size of other brain structures. Barton and Venditti's (2013a) research supports Semendeferi et al., (2002) who also concluded, using MRI, that the frontal lobes in humans were not overly large and that superior human social abilities may be due to alterations in cortical areas as well as a higher density of cortical interconnectivity compared to other species.

The evolutionary enlargement of white brain matter volume compared to grey matter in humans has also been proposed to underpin human social cognition (Schenker, Desgouttes & Semendeferi, 2005). Increased white matter is caused by an expansion of axons, dendrites, and myelination which might expedite neuronal transmission. In turn, this may facilitate the fast learning needed for superior human, compared to other species, social cognition, particularly in rapidly changing dynamic social contexts (Schenker et al., 2005). This theory is supported by adolescent research which reports a surge of myelination during puberty (Arain et al., 2013), and this stage of development is also associated with increased social cognitive ability (Burnett & Blakemore, 2009; Choudhury, Blakemore & Charman, 2006; Taylor et al., 2013, Taylor, Barker, Reidy & McHale, 2015). Although brain matter expansion theories are well supported in the literature, at present there is conflicting data regarding which areas undergo expansion. For example, Smaers et al., (2011) postulated that there has not been a disproportionate expansion of overall white or grey matter in the human frontal cortex, proposing that a specific

white and grey matter increase in the left prefrontal region has led to the evolutionary increase in social skills in humans. Cellular research is focusing on Von Economo neurons (VENs) which are located within layer five of the anterior cingulate and frontoinsula cortex. VENs are unique to hominoid primates (Butti, Santos, Uppal & Hof, 2013), and are less dense in apes compared to humans (Nimchinsky et al., 1999). VENs are thought to be a specialised projection neuron for linking cortical regions and possibly relaying social information (Stimpson et al., 2011). VENs are depleted in frontotemporal dementia, a neurodegenerative disease associated with social cognition deficits, particularly the inability to identify emotional states and demonstrate empathy (Waldö, Santillo, Gustafson, Englund, & Passant, 2014; Seeley et al., 2007). The opposite effect is reported for autism, a developmental disorder associated with poor communication and other social deficits. Santos et al., (2011) reported that post-mortem results indicated that autistic groups had a significantly higher number of VENs compared to control individuals. This finding concurs with Frith's (2004) 'under-pruning' hypothesis for autism which proposes that connections are not effectively pruned during infancy resulting in abnormal connectivity. This excessive preservation of VENs may be linked with the common autistic symptom of muted interoception, the inability to moderate bodily sensations which, in turn, has a significant effect on social functioning (Schauder, Mash, Bryant & Cascio, 2015). Conversely, Thomas, Davis, Karmiloff-Smith, Knowland and Cahrman (2016) challenge the under-pruning theory and present conflicting evidence for an 'over-pruning' hypothesis for the neurophysiological basis of autism. Although there is contention regarding the cellular basis of human social cognition, the research area is broadening the empirical evidence base investigating the underlying neural networks of social cognition in humans.

There is still a longstanding controversy concerning the association between the disproportionate evolution of the frontal lobes and high-order human abilities. A recent tissue volume analysis conducted by Donahue, Glasser, Preuss, Rilling and Van Essen (2018)

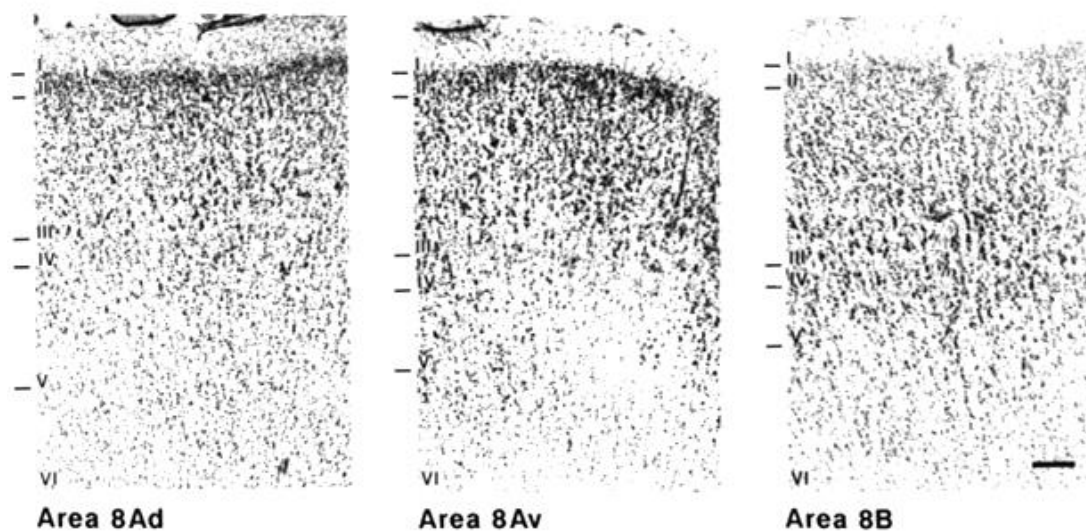
reported a disproportionately large volume of grey and white matter corresponding to the prefrontal cortex (PFC) in humans compared to non-human primates. Smaers, Gómez-Robles, Parks and Sherwood (2017) explored PFC scaling across anthropoid primates and reported that the human PFC expansion does not follow typical allometric growth patterns (e.g. not associated with scale and body size), aligning with evidence for a developmental heterochronic shift in the human prefrontal expansion (Somel et al., 2011). In his review of the evolution of the human brain, Preuss (2017) summarised that evolutionary changes in the human brain were not restricted to one specific region, with this cortical change running parallel with developments in molecular, cellular physiology and network connectivity systems. The majority of human brain enlargement appears to be in the association cortex, an area highly related to higher-cognitive functions (Jung, Cloutman, Binney & Ralph, 2017). The expansion of the association cortex was accompanied by changes of the systems connecting cortical areas to functional networks, for example, language and tool-manipulation systems (Rakic, 2009).

In summary, the specific evolutionary basis for human social cognition remains unclear. However, there is a strong evidence base indicating that this may be related to a natural adaptation and enlargement of specific distributed neural network connectivity throughout cortical and non-cortical brain regions (Barton & Venditti, 2013b; Risberg & Grafman, 2006; Schenker et al., 2005; Semendeferi, Armstrong, Schleicher, Zilles & Van Hoesen, 2001). The next section discusses neuroanatomy and connectivity of the frontal lobes as this brain region is frequently associated with social cognition (Amodio & Frith, 2006; Bzdok et al., 2013; Forbes & Grafman, 2010; Wood, 2003). Evidence has suggested a specialised social neural circuit within the frontal lobes which undergoes alterations during different stages of human development, particularly during adolescence (Kilford, Garrett & Blakemore, 2016; Taylor et al., 2012, 2015). This social neural substrate is sensitive to trauma, developmental, and degenerative abnormalities resulting in abnormal social functioning (Baez, García & Ibanez,

2017; Eack, Wojtalik, Keshavan & Minshew, 2017; Rigon, Voss, Turkstra, Mutlu & Duff, 2016).

#### 1.4. Delineations of the frontal lobes

The frontal lobes are located at the anterior of the cerebral cortex, directly in front of the parietal lobes with the central sulcus separating the two lobes (Figure 4). The frontal lobes are not structurally homogeneous but comprised of distinct regions, which can be characterised by cellular structure or by functionality (Cummings & Miller, 2007). Cytoarchitectonic studies subdivide the cortex based on differential neuronal patterns in the cortical layers of the brain and thus indicate specialised functional units (Figure 5).



**Figure 4. Photomicrographs representing Brodmann areas 8Ad, 8Av, and 8B located in the frontal cortex of humans (reproduced from Petrides & Pandya, 2012, p.996). The roman numerals illustrate cortical layers and the black line in 8B represents 1mm.**

One of the most well-known cytoarchitectonic maps was produced by Brodmann who labelled individual cortical areas with numbers, commonly referred to as Brodmann areas (BA; Brodmann, 1908; Figure 6). Several alternative cytoarchitectonic maps of the human cortex have since been produced (Bailey & von Bonin, 1951; von Economo & Koskinas, 1925;

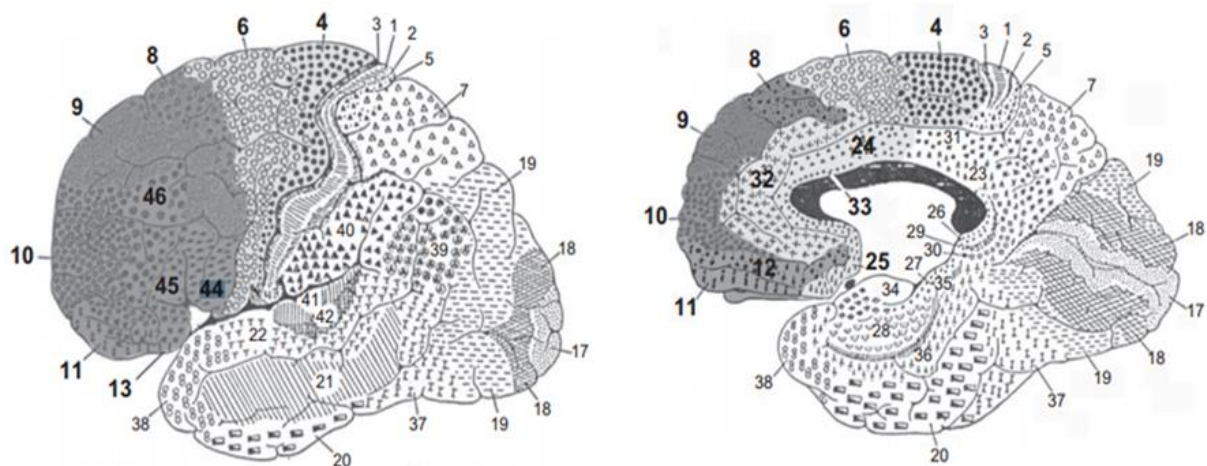
Sarkissov et al., 1955; Vogt, 1919), one of the most prominent being Walker's (1940) cytoarchitectural study of the prefrontal area of the macaque monkey. However, with advancements in modern technology, discrepancies in the architectonic parcellation of the human cortex have emerged (Ardila, Bernal & Rosselli, 2016; Zilles & Amunts, 2010).

Petrides, Tomaiuolo, Yeterian and Pandya (2012) and Petrides and Pandya (2012) re-examined the Brodmann cytoarchitectonic map of the human PFC to resolve previous discrepancies. This thesis will draw upon the seminal Brodmann maps when referring to frontal lobe delineations but will also reference Petrides et al., (2012) cytoarchitectonic map of the human PFC to ensure that the most contemporary data is being presented (Figure 7 and 8). Petrides et al.'s (2012) work highlights advancements in the field of cytoarchitectonic mapping and illustrates the evolution of the research field. Commonly the frontal lobes are divided into the motor, premotor and prefrontal regions (Cummings & Miller, 2007; Penfield & Jasper, 1954). The primary motor (area 4) and premotor regions (area 6) are distinct, while the prefrontal region may be further divided (Rae, Hughes, Anderson & Rowe, 2015). The motor regions mediate motor functions and bodily movements in conjunction with the basal ganglia, cerebellum and brain stem pathways (Leisman, Moustafa & Shafir, 2016). Penfield and Boldrey (1937) proposed the cortical homunculus which depicts the anatomical delineations of the motor strip in a pictorial form, where the size of each body part is proportional to the allocated size in the motor strip (Figure 9).

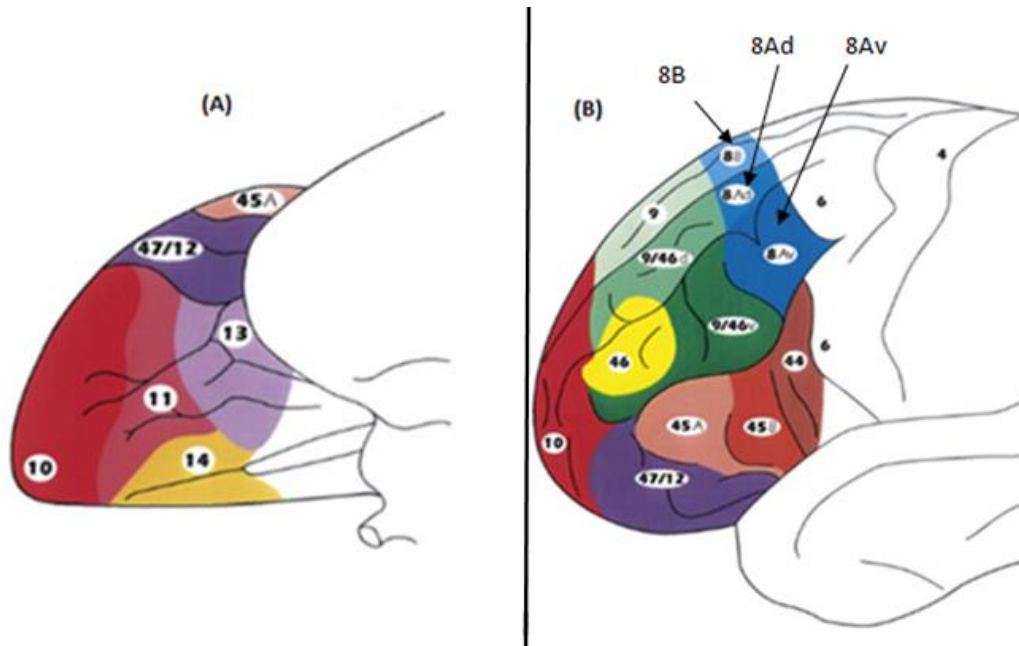
The current work will use the terms dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC) and orbital frontal cortex (OFC) for the subdivisions of the prefrontal cortex following the delineations of Petrides et al., (2012). Although the PFC is frequently described as homogeneous, it is evidentially heterogeneous. For instance, prefrontal neurons are thought to be highly diverse in their; developmental lineages, molecular phenotypes, projection targets, afferent inputs, synaptic connectivity, physiological properties,



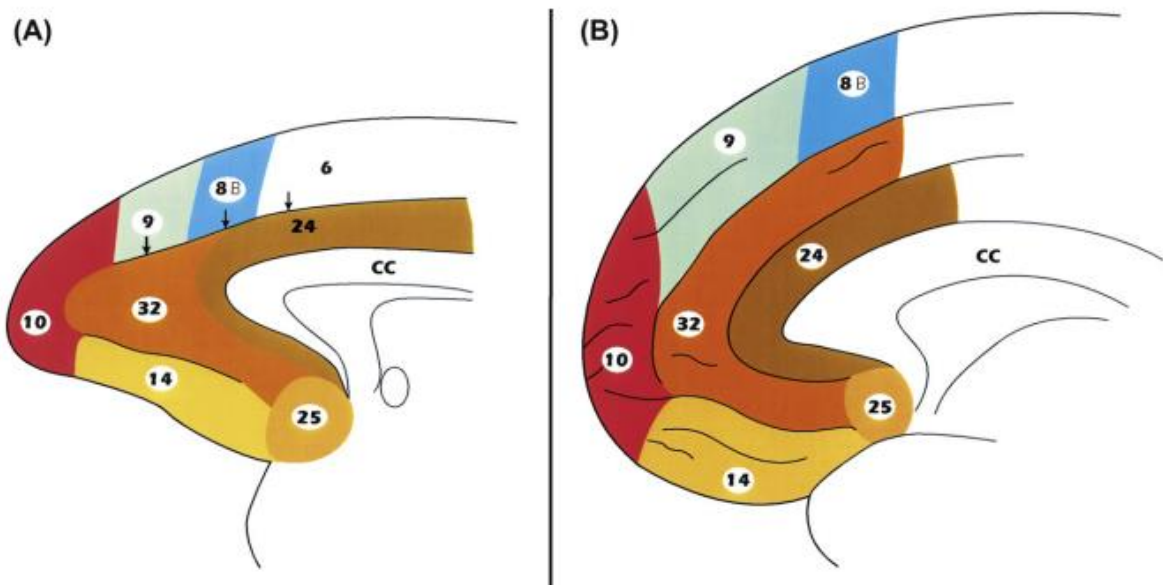
and behavioural functions (Chandler, Gao & Waterhouse, 2014). The diverse characteristics of prefrontal neurons could play a role in the dissociable cognitive functions associated with the PFC (Chandler, Lamperski & Waterhouse, 2013). Research findings have shown that the PFC undergoes a diverse and protracted maturation including the expansion of synaptic neuronal connections as well as the pruning and reorganisation of these networks (Taylor et al., 2015). This extended maturation is related to the longitudinal development of executive and social functions into early adulthood (Taylor et al, 2015). Prefrontal architecture is reviewed in the next section.



**Figure 5. Brodmann area map of the brain (reproduced from Teffer & Semendeferi, 2012, p. 192). The frontal lobe is shaded in grey with the prefrontal lobe depicted by the darker shading of grey. Brodmann denoted frontal regions to include areas 8, 9, 10, 11, 12, 44, 45, 46, and 47. Precentral regions included areas 4 and 6. Anterior cingulate cortex included areas 24, 25 32 and 33. The orbital prefrontal region includes area 11 and 13. Prefrontal cortex generally refers to all shaded areas in the diagram bar the motor and premotor areas (4, 6, 24 and 44) and the dorsolateral frontal lobe (9, 10 and 46).**



**Figure 6. Modified cytoarchitectonic map of the lateral and orbital surfaces of the (A) monkey lobe and (B) human lobe (reproduced from Petrides & Pandya, 2012, p.995).**



**Figure 7. Modified cytoarchitectonic map of the medial surfaces of the (A) monkey and (B) human frontal lobe (reproduced from Petrides & Pandya, 2012, p.996).**

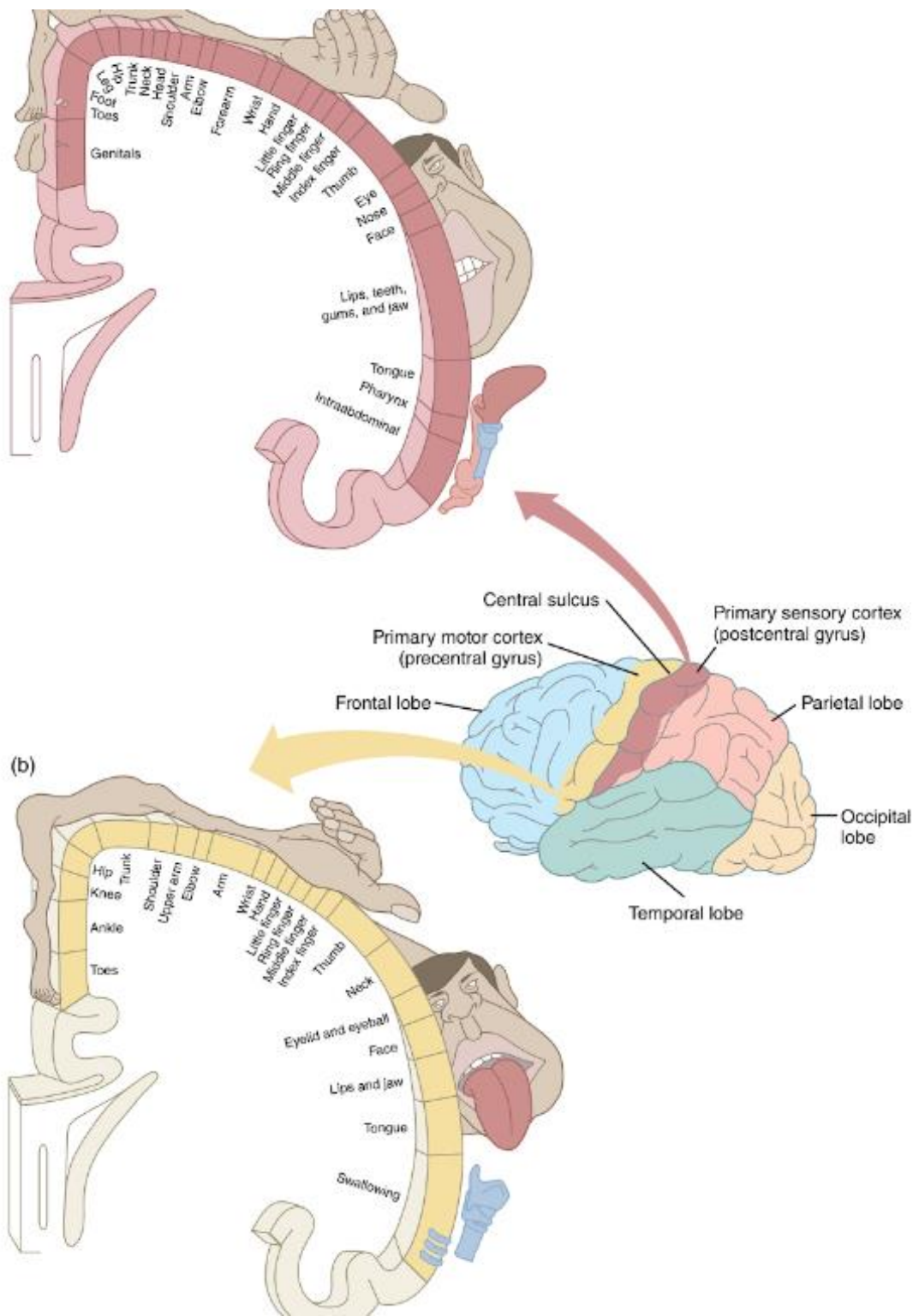


Figure 8. A diagram depicting the motor homunculus (reproduced from Manasco, 2016, p.34).

#### **1.4.1. Dorsolateral prefrontal cortex (DLPFC)**

In terms of cytoarchitectonics, the DLPFC encompasses area 8, 9 and 46 (Brodmann, 1908; Cieslik et al., 2013; Petrides et al., 2012; Petrides & Pandya, 2012; Pierrot-Deseilligny, Rivaud, Gaymard & Agid, 1991). Area 8 is located on the posterior side of the superior and middle frontal gyri, extending medially to the paracingulate sulcus (Figure 6-8). To the posterior of area 8 is rostral area 6 and anteriorly it abuts area 9 on the superior frontal gyrus and area 9/46 on the middle frontal gyrus. Area 8 can be divided into 8A and 8B (Figure 7). Area 8A is located on the middle frontal gyrus and can be further divided into ventral (area 8Av) and dorsal (area 8Ad) regions (Figure 7) (Petrides et al., 2012). Area (8Av) is located in a narrow section of the caudal-most middle frontal gyrus while the area 8Ad extends into the superior frontal sulcus.

On Brodmann's (1908) map, areas 9 and 46 are located in the dorsolateral frontal area at the midsection of the superior and middle frontal gyri. Area 9 extends across the superior frontal gyrus and the middle frontal gyrus and, therefore, area 46 occupies the middle frontal gyrus, is abutted by area 9, both dorsally and caudally. Petrides et al., (2012) reported that the architecture of the superior frontal gyrus, labelled as area 9 on their map, differed from Brodmann (1908), with area 9 being more analogous with 46. Petrides et al., (2012) have consequently labelled the area occupying the middle frontal gyrus 9/46 (Figure7). According to Petrides et al., (2012), area 9 is located in the superior frontal gyrus, extending medially to the paracingulate sulcus. Area 46 is located in the anterior section of the middle frontal gyrus with a significant area concealed in the intermediate frontal sulcus.

The DLPFC has complex interconnections and subcortical circuits which allow communication across the cerebral cortex and other brain regions (Cummings & Miller, 2007). Of relevance to the current research, area 46 projects to area 8 where the frontal eye fields (FEF) are located

(Barbas, Ghashghaei, Rempel-Clower & Xiao, 2002). The FEF plays a role in covert spatial attention where, without moving the eyes, a location is selected for cognitive processing (Brooks & List, 2006). The FEF's also mediate voluntary control over saccades, the rapid shift in eye movements, which allow an individual to shift attention to the most salient information in the visual scene (Ghasia & Shaikh, 2013). When engaging in social interaction, an individual must constantly adjust their attention to the most pertinent information, such as facial expressions or following eye gaze (Auyeung et al., 2015; Frischen, Bayliss & Tipper, 2007; Tanaka & Sung, 2016). Mundy and Newell (2007) suggested that FEF's are an important network for social cognition as they mediate voluntary saccades, an integral mechanism for encoding other's behaviour. Furthermore, the FEF region is postulated to be part of an attention system that mediates volitional and goal-directed behaviour, processes vital for appropriate social behaviour i.e. joint attention (Mundy & Newell, 2007; Mundy, Sullivan & Mastergeorge, 2009) where two people consciously coordinate attention to a common focus (e.g. another person, object, or situation). Joint attention can be divided into (1), response joint behaviour; the ability to follow gaze, head turn and gestures, and (2), initiating joint behaviour where an individual consciously creates a new point of reference with the use of gaze, head turn or gesture (Mundy et al., 2009). If joint attention is not followed and responded to, then this will have a negative effect on social behaviour and may lead to communication breakdown (Hahn, 2016). Additionally, if eye gaze synchrony is not initiated or sustained, then this will also lead to poor communication and may be seen as a deviation from the typical social convention (Adamson, Bakeman, Suma & Robins, 2017). Impairments in eye gaze are frequently associated with autism; a developmental disorder characterised by communication and relationship difficulties (Tanaka & Sung, 2016) and schizophrenia; a neuropsychiatric disorder characterised by abnormal social behaviour (Seymour et al., 2017). Emerging data has also indicated that there may be a relationship between aberrant eye scan patterns and social

cognition impairments after TBI (Vassallo, Douglas & White, 2010). As frontal brain areas are particularly vulnerable to head trauma (Rabinowitz & Levin, 2014), it could be postulated that damage to the visual networks within the frontal lobes and visual network connectivity between frontal, parietal and temporal lobes will have a detrimental effect on social functioning, although more data to support this theory is needed.

#### **1.4.2. The ventrolateral prefrontal cortex (VLPFC)**

The VLPFC encompasses areas 44, 45, 47/12 and 14 (Figure 7). Area 44 is reported to encompass Broca's speech area and is located rostral to the inferior precentral sulcus in the pars opercularis of the inferior frontal gyrus (Figure 7). Area 45 is located in the pars triangularis of the inferior frontal gyrus and can be subdivided into rostral and caudal sections labelled as area 45A and area 45B in Figure 7. Brodmann (1908) labelled the area rostroventral to region area 45 as area 47 (Figure 6). Area 47 is located in the most rostral region of the inferior frontal gyrus and extends onto the caudal half of the orbitofrontal cortex. Petrides et al., (2012) noted that a section of area 47 is located to the rostral and ventral region of area 45, extending to the lateral orbital sulcus. Area (47) shares cytoarchitecture similarities with Walker's (1940) map of the monkey brain and hence Petrides et al., (2012) have labelled the area 47/12. The remaining area 47 is located on the caudal orbitofrontal cortex.

Area 44 is thought to have visual, motor, language, and working memory functions (Ardila et al., 2016; Bernal, Ardila & Rosselli, 2015; Morin & Grèzes, 2008). Neural activation in area 44 during movement observation and production has indicated a possible connection with the mirror neuron system (Iacoboni et al., 2005; Press, Weiskopf & Kilne, 2012). A mirror neuron fires when an animal or primate performs a behaviour and also when the animal or primate observes the same behaviour performed by another. The mirror neuron system is of great significance to social cognition as understanding the actions of others is necessary for survival,

social organisation, human culture, and imitation learning, for example, the development of language (Rizzolatti & Craighero, 2004). Area 44 has also been associated with intentional facial expressions, a complex human communication tool which is socially learned and used to convey emotional states. Facial expressions are also used to mask underlying intentions and facilitate traits such as deception, manipulation and empathy (Gola et al., 2017). Intentional facial expressions are vital for modulating social functioning as they express an individual's emotional state as well as influencing the mood state of others. Gola et al., (2017) reported a correlation between intentional expression imitation and cortical volume loss in the speech production network, a prime region of the mirror neuron system, in individuals with prefrontal atrophy who displayed socioemotional impairments. Gola et al., (2017) concluded that imitation of facial expressions was associated with a right lateralised cortical network, coined the 'emotional apraxia' network which was homologous to the left-sided speech production network. It should be noted that research on area 44 is tentative and there are discrepancies regarding the exact location and function of this area (Morin & Grèzes, 2008). Functions associated with area 45 are similar to that of area 44 in the mediation of language, including lexical search during reading (Heim, Eickhoff & Amunts, 2009) and encoding of word meaning (Zaccarella, Meyer, Makuuchi & Friederici, 2017). Area 45 is also thought to play a role in the perception of affective prosody, the ability to understand emotion expressed through speech (Belyk & Brown, 2014; Witteman, van Ijzendoorn, van der Velde, van Heuven & Schiller, 2011). This is important for social interactions as it enables emotional valence to be understood (Brazo, Beaucousin, Lecardeur, Razafimandimby & Dollfus, 2014).

Neuroimaging data have shown that area 47 and the ventral section of area 6 are part of the language module of the brain (Bookheimer, 2002; Devlin, Matthews & Rushworth, 2003; Hagoort, Hald, Bastiaansen & Petersson, 2004). Area 47 may also mediate certain emotion-related processes, such as the successful control of emotions, particularly the modulation of

negative affect through the use of cognitive strategies (Phan et al., 2005) and executive functions, such as working memory (Collette, Hogge, Salmon & Van der Linden, 2006; Wager & Smith, 2003). There is debate surrounding whether emotional mimicry is an implicit or explicit process (Kirkham, Hayes, Pawling & Tipper, 2015). The phenomenon occurs rapidly (Chechko, Pagel, Otte, Koch & Habel, 2016; Riehle, Kempkensteffen & Lincoln, 2017) and subconsciously when it is not the main focus of the task (Kaiser, Davey, Parkhouse, Meeres & Scott, 2016), as well as during tasks where participants are asked to inhibit facial mimicry (Kirkham et al., 2015). Nevertheless, humans can inhibit emotional mimicry to avoid inappropriate responses during social interactions, for example, not smiling at an offensive joke. Consequently, emotional mimicry may be a moderated automatic process (Kirkham et al., 2015; Murata, Saito, Schung, Ogawa & Kameda, 2016) that only occurs if there is a neutral or positive rapport between individuals (Murata et al., 2016). Reductions in cell density in area 47 have been documented in individuals with schizophrenia, Alzheimer's disease and depression (Bralet, Buchsbaum, DeCastro, Shihabuddin & Mitelman, 2016; Underwood et al., 2012), conditions associated with impairments in intentional facial mimicry (Gola et al., 2017; Varcin, Bailey & Henry, 2010; Zwick & Wolkenstein, 2017). Pathology to the VLPFC often causes disturbances within the frontal-subcortical signalling circuitry which, in turn, disrupts higher-order regulatory functions, such as speech and the modulation of facial expressions, negatively impacting socioemotional functioning (Lee, Josephs, Dolan & Critchley, 2006).

### **1.4.3. Orbital frontal cortex (OFC)**

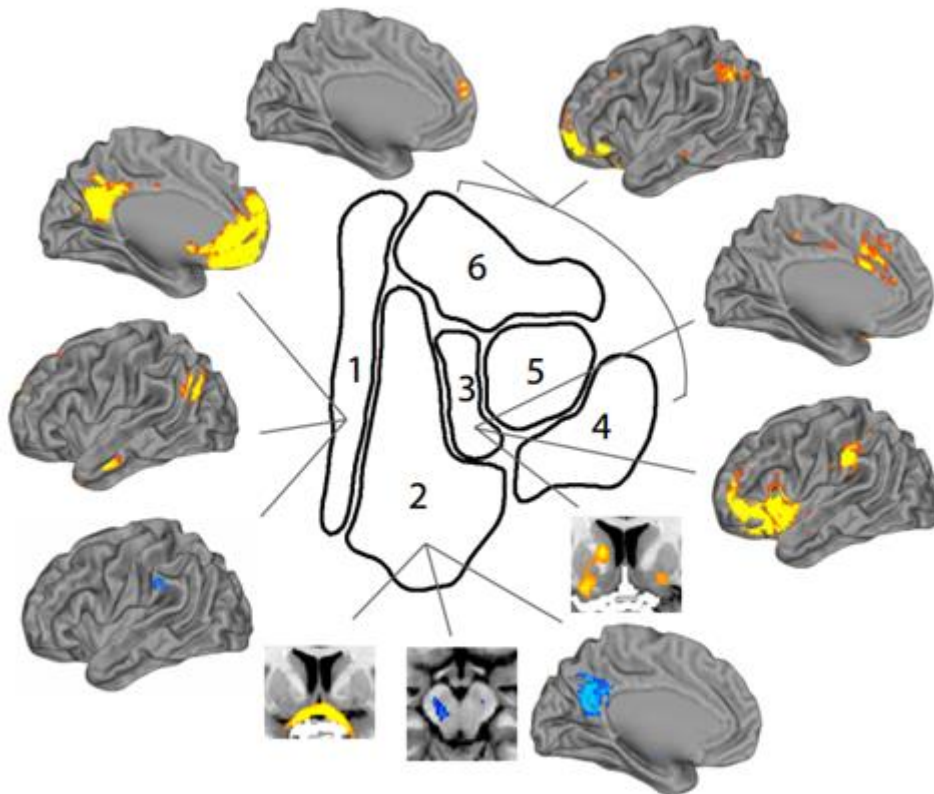
Brodmann (1908) labelled a significant area of the caudal OFC as area 47 and then labelled the remaining OFC as area 11. Brodmann (1908) acknowledged that area 47 and area 11 were heterogeneous and could be subdivided. Petrides et al., (2012) drew upon this distinction and incorporated it into their cytoarchitecture model, recognising that a significant part of the



ventrolateral frontal region was labelled as area 12. Medial to area 12 on the orbital frontal region was area 13 caudally and area 11 rostrally. Using their architectonic analysis of monkey and human brains, Petrides et al., (2012) reported that Walker's (1940) area 12 only slightly abutted with area 47 on the ventral-most section of the ventrolateral frontal area and extended as far as the lateral orbital sulcus. Petrides et al., (2012) referred to this area as 47/12. The caudomedial section of area 47 shares cytoarchitecture similarities with Walker's (1940) area 13 and therefore Petrides et al., (2012) labelled the area as 13 (Figure 7). This division differentiates area 13 from more lateral orbital extensions of area 47/12, rostral area 11 and medial area 14 (Figure 7).

In the monkey brain, the OFC has connections with higher sensory areas, medial and lateral PFC, temporal lobe, midbrain, and striatum (Cavada, Compañy, Tejedor, Crux-Rizzolo & Reinoso-Suárez, 2000). Ongür and Price (2000) presented a functional division of the monkey OFC; the medial network comprised the medial and posterior-lateral section of the OFC and the orbital network which extends over the ventral surface. Zald et al., (2012) used meta-analytic connectivity modelling (MACM) to support Ongür and Price's (2000) findings, suggesting clear segregation between medial and lateral regions of the OFC. The human medial OFC is understood to be an output system, mediating stimuli associated with reward as well as playing a role in the expression of emotion and action (Neubert, Mars, Sallet & Rushworth, 2015; O'Doherty, Kringelbach, Rolls, Hornak & Andrews, 2001; Price & Drevets, 2010; Xiu, Geiger & Klaver, 2015). The lateral OFC is thought to be a sensory-related system combining multi-modal stimuli as well as being vital in mediating stimuli related to punishment and behaviour alteration signals (O'Doherty et al., 2001; Price & Drevets, 2010; Rolls, 2017). Contemporary research has advocated a more detailed delineation of the OFC rather than a simple medial and lateral division. Kahnt, Chang, Park, Heinzle and Hayne (2012) used brain imaging methodology to parcellate the human OFC. Findings suggested six functionally

distinct sub-regions; medial, posterior-central, central and three clusters in the lateral OFC (Figure 10).



**Figure 9. OFC human subdivisions (reproduced from Kahnt et al., 2012 p.6247). The different numbers refer to the specific units of the OFC as identified by Kahnt et al., (2012). (1) medial, (2) post-central, (3) central, (4-6) lateral.**

Indeed, Kahnt et al.'s, (2012) division of the human OFC is convergent with diffusion-weighted MRI (Neubert et al., 2015), cytoarchitectonic (Uylings et al.; 2010), functional activation (Berridge & Krigelbach, 2015), and resting-state functional connectivity research (Rolls, Joliot & Tzourio-Mazoyer, 2015). Hierarchical clustering suggests that the medial cluster of the OFC is activated during reward and decision tasks (Kahnt, Heinzle, Park & Haynes, 2011). The medial cluster has connections with the medial PFC and the posterior cingulate cortex which are thought to regulate episodic memory (Cavanna and Trimble, 2006) and self-related processes (Sajonz et al., 2010). The posterior-central cluster is connected to

the midbrain, possibly the substantia nigra, which provides the striatum with dopaminergic neurons and is related to the experience of pleasure and reward learning (Li et al., 2015; Zhang, Harris, Split, Troiani & Olson, 2016). The central cluster has connections with ventrolateral and dorsal striatum and is reported to mediate value learning (Kahnt et al., 2010), while the dorsomedial frontal cortex is associated with action-outcome values (Gläscher, Hampton & O'Doherty, 2009). The lateral clusters displayed connections with the temporal and parietal cortices involved in high-level visual processing (Delgado, Li, Schiller & Phelps, 2008) as well as the DLPFC which mediates optimal decision making and self-control (Hare, Camerer & Rangel, 2009).

Importantly, two distinct functional regions appear evident in the OFC. Pegors, Kable, Chatterjee & Epstein (2015) scanned human participants with functional MRI (fMRI) while they judged the attractiveness of faces and places. The first region appeared to process category-unique responses to face attractiveness, and the other region responded strongly to faces but exhibited no preference for attractiveness versus non-attractive faces. The authors concluded that the OFC may modulate basic reward processing that is specific to certain stimulus categories, possibly socially relevant stimuli. Troiani, Dougherty, Michael and Olson (2016) termed these regions 'OFC face patches' and reported a prevalence of OFC face-selective regions in control participants. Furthermore, they described a correlation between the OFC face patches and social motivation assessed via the Broad Autism Phenotype Questionnaire (Hurley, Piven & Parlier, 2007). The authors proposed that abnormal activation in OFC face patches during the viewing of faces could reflect developmental disorder symptoms. For instance, it is well established that autistic individuals display altered reward sensitivity during behavioural tasks (Kohls et al., 2014), and there is evidence that OFC activation is reduced in autistic groups (Lynn et al., 2018). Troiani et al., (2016) advised that future research should investigate the role OFC face patches play in social learning. Retrograde

tracing determined that the face patches have complex connectivity with both feed-forward and feedback projections (Grimaldi, Saleem & Tsao, 2016). Grimaldi et al., (2016) recorded low inputs from other prefrontal regions while distinct face patches received strong inputs from the extrastriate cortex (in the visual cortex), the medial temporal lobe, and three subcortical structures including the pulvinar, claustrum, and amygdala. These findings suggest a highly specialised system incorporating prefrontal face patches and critical connections to subcortical regions for the processing of face-related information. Also, the face patch network overlaps with the social brain network including the supplementary eye fields (SEF) which are thought to aid attention direction by guiding saccades to relevant facial features during emotion identification. Hence, pathology to this region of frontal cortices could have a devastating effect on the recognition, evaluation and generation of social signals (Schwiedrzik, Zarco, Everling & Freiwald, 2015).

Social interactions are often complex and rapid and frequently require an individual to engage in decision making and adapt behavioural responses, as well as modulating moral behaviour and empathy which are critical for human social functioning (Kringelbach & Rolls, 2003). If an individual is unable to assess and utilise reward and punishment or they are impaired at alternating behaviour then this would have an adverse effect on social communication and integration (Hornak, Rolls, & Wade, 1996; Rolls, Hornak, Wade & McGrath, 1994). Damage to the OFC can lead to inhibition impairments which can have a detrimental effect on interpersonal behaviours such as the inability to regulate interpersonal space (Peters & D'Esposito, 2016), self-disclosed information (Beer, John, Scabini & Knight, 2006) or emotionally-driven outrages (Mimura, 2010). Abnormalities in the medial OFC is also correlated with mood disorders such as depression (Rolls et al., 2018), and obsessive-compulsive disorder (Ducharme, Dougherty & Drevets, 2016). Social interactions can promote physical and psychological benefits (Holt-Lunstad et al., 2015), therefore, disinhibited

behaviours, which lead to social isolation, could have a significant effect on wellbeing in groups that are affected by abnormal OFC functions (Lefebvre, Cloutier & Levert, 2008).

#### **1.4.4. Frontopolar cortex (FPC)**

The Frontopolar cortex (FPC) is one of the largest cytoarchitectural areas of the human cortex, situated at the most anterior point of the PFC and corresponding to area 10 (Boschin, Piekema & Buckley, 2015) (Figures 6 and 7). On the lateral surface, it caudally abuts areas 9, 46 and 47/12, on the orbital surface it abuts areas 11 and 14 and on the medial surface, it abuts areas 9, 32 and 14. The functionality of area 10 is relatively unknown as the area is methodologically difficult to investigate due to structural differences between humans and animals, hindering the application of animal to human models. At present, electrophysiological and imaging techniques are not able to reliably isolate area 10 and focal lesions to this region are rare (Burgess, Simons, Dumontheil & Gilbert, 2007).

In a review of area 10, Burgess et al., (2007) outlined four theories of function which included; episodic memory, meta-cognition (the ability to reflect on one's thoughts), higher-order cognitive processes, for example holding numerous goals in mind or selecting between goals and the default mode hypothesis. The default mode hypothesis relates to decreases in neural activity during a task compared to a previously defined baseline state (Greicius, Krasnow, Reiss & Nenon, 2003). Burgess et al., (2005) questioned all four theories, mainly on the grounds of; incomplete accounts, discrepancies in definitions of brain regions and the different tasks/stimuli being used. Due to the anterior location of area 10, the well-established complexity of the prefrontal cortex, and the association the area has with executive functions (Koechlin, 2016), it may be surmised that damage to area 10 could result in severe cognitive dysfunction but this is not so. Case study N.M (Metzler & Parkin, 2000) underwent virtually complete removal of the rostral PFC but presented with good social skills and performed within the superior range on IQ, memory and executive function tasks. N.M did display impairments

in multitasking which had a detrimental effect on social functioning, such as frequent tardiness and disorganisation, meaning he was unable to maintain his premorbid occupational role. Conclusions based on case-studies must be treated with caution and are hard to generalise yet research by Burgess et al., (2000) also reported that 60 individuals with neurological damage to area 10 displayed impairments on the Greenwich multi-tasking task. Similarly, Boschini et al., (2015) research findings supported the theory that damage to area 10 does not necessarily produce marked cognitive deficits but, may instead, produce subtle deficits which lead to significant social deficits. Boschini et al., (2015) reported that localised lesions to non-human primate frontopolar cortex led to deficits in rapid novel learning, particularly during uncertain contexts. Rapid novel learning is integral to human-level intelligence and impairments would lead to abnormalities in everyday functioning, such as not being able to learn new technologies or adapt behaviour according to verbal instructions (Cole, Laurent & Stocco, 2013). Boschini et al., (2015) postulated that the prefrontal cortex is vital for rapid learning regarding the relative value of alternatives, including stimuli and rules. It should be noted that animal research may be a poor predictor of human physiology and experience, and caution must be taken when trying to apply animal findings to humans (Akhtar, 2015; Bracken, 2009). Hyafil and Koechlin (2016) described area 10 as the apex underlying executive control. The authors proposed that the area mediates a basic process known as cognitive branching, the ability to maintain a pending task whilst completing an alternative task and then reverting to the original pending task. Hyafil and Koechlin (2016) suggested that their cognitive branching theory also explained why the area is associated with so many other cognitive functions, such as prospective memory and attentional shift. This theory aligns with previous research reporting activation of area 10 during tasks such as the standard Tower of London. The Tower of London draws on executive functions including planning and prospective memory (Christoff & Gabrieli, 2000).

The current models exploring the anatomical and functional basis of the FPC require more investigation, particularly around the role of area 10. Needless to say, advancements in neuroimaging may inform new human models. Currently, it appears that the FPC mediates a range of social and cognitive processes. Damage to the area may result in impairments in novel learning and the ability to switch in and out of multiple tasks without the aid of external cues or sequential learning. These types of impairments would have a negative effect on planning and multi-tasking in the social world (Boschin et al., 2015; Hyafil and Koechlin, 2016).

#### **1.4.5. Medial frontal cortex (MFC)**

The cingulate and paracingulate areas on the medial surface of the frontal lobes are respectively referred to as the medial prefrontal cortex (MFC) labelled as areas 24, 25 and 32 (Figure 7). On the medial surface of the superior frontal gyrus, dorsal to areas 24 and 32, is the medial extensions of areas 8, 9 and 10. Area 32 ventrally abuts the medial section of area 14 and caudally area 25.

de la Vega, Chang, Banich, Wager and Yarkoni (2016) published a meta-analysis across nearly 10,000 human studies to create a topological map of cognitive, social and emotional functions within the MFC. The authors applied cluster-based analysis (grouping contiguous brain regions activated during fMRI-Heller, Stanley, Yekutieli, Rubin & Benjamin, 2006) to create zones, which were small and consisted of few voxels, and sub-regions which were larger and contained more voxels. At a basic level, the MFC was divided into three bilateral clusters spanning across the rostrocaudal axis. The posterior zone had two clusters, with de la Vega et al., (2016) proposing that there was a high likelihood that it contained parts of the supplementary motor area. In the middle zone, four clusters were identified with two anterior and two posterior clusters located anterior to the anterior vertical commissure. The dorsal clusters were homogenous to the paracingulate gyrus and the two ventral clusters to the cingulate gyrus. In the anterior zone, a dorsal cluster was homogenous to the medial frontal

pole and superior frontal gyrus with no aspects of the anterior cingulate gyrus. Two ventral clusters were reported; the first was within pregenual aspects of the anterior cingulate gyrus and included pregenual portions of paracingulate gyrus while the second more ventral cluster was associated with pregenual aspects of the ACC and medial OFC. The research team then contrasted co-activation patterns of the three zones and determined that the posterior zone demonstrated bilateral co-activation with the primary motor cortex, superior parietal cortex, anterior cerebellum, posterior insula and subcortical regions, including the thalamus and dorsal striatum. This activity configuration is comparable to motor processing. The middle zone co-activated with anterior aspects of the thalamus as well as regions in the frontoparietal control network, such as dorsolateral prefrontal cortex, anterior insula and superior parietal cortex. The anterior zone co-activated with angular gyrus, hippocampus, posterior cingulate cortex, amygdala and ventral striatum. de la Vega et al., (2016) used a data-driven multivariate classifier to ascertain whether clusters were correlated to specific cognitive functions. The posterior zone was related to motor function, including gaze, the middle zone was linked to negative affect recognition, pain, fear and decision making and the anterior zone was associated with affective functions including fear, reward, and decision making, in addition to internally orientated functions, for example, episodic memory and social processing. While de la Vega et al., (2016) meta-analysis results provided insight into the functional diversity of the MPFC, the authors note that the apparent heterogeneity of the region must be interpreted with caution as it was noted that no single region was solely activated by a single function. It is probable that there are overlapping neuronal populations within the region (Kvitsiani et al., 2013), suggesting that pathology to the area could result in the inability to perceive and process social stimuli causing impairments in mentalizing and affective states.

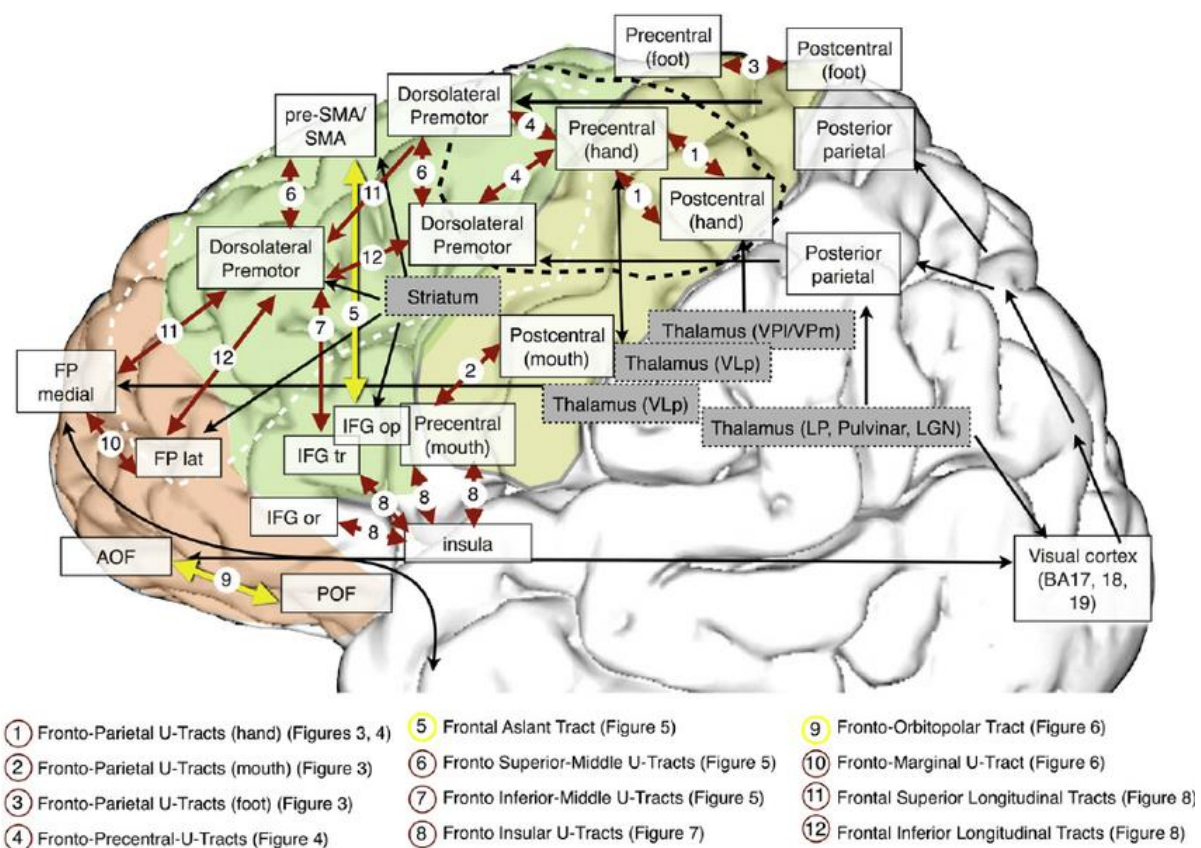


#### **1.4.6. Cortical connection patterns of the frontal lobes**

The frontal lobes are associated with higher-order cognitive and social-emotional functions. Nevertheless, specialised regions within the frontal lobes, such as the motor cortices, supplementary motor areas, and FEF, mediate more basic processes. These low-level processes are just as important, if not more so, as high-level cognition. If initial processes or functions are aberrant then this is likely to have an adverse influence on upstream cognitive functions. The complex range of basic and higher cognitive abilities mediated by the frontal lobes is dependent upon integrated and intricate networks of fibrous connections (Bartolomeo, Thiebaut de Schotten & Chica, 2012; Yeterian, Pandya, Tomaiuolo & Petrides, 2012) (Figure 11). Long-range projections and association fibres convey sensory knowledge from subcortical nuclei and sensory cortices and then facilitate frontal input to this information (Catani et al., 2012). Short-range projections are responsible for local connectivity of the frontal lobes and consist of U-shaped projections among gyri and longer intralobar fibres (Yeterian et al., 2012). Also, critical feed-forward and feedback circuits form connections from the PFC to the rest of the frontal lobe region. The extensive PFC connections are widely dispersed and include circuits with the association and limbic cortices and brainstem areas, more than likely mediating specific elements of social cognitive processes (Siddiqui, Chatterjee, Kumar, Siddigui & Goyal, 2008).

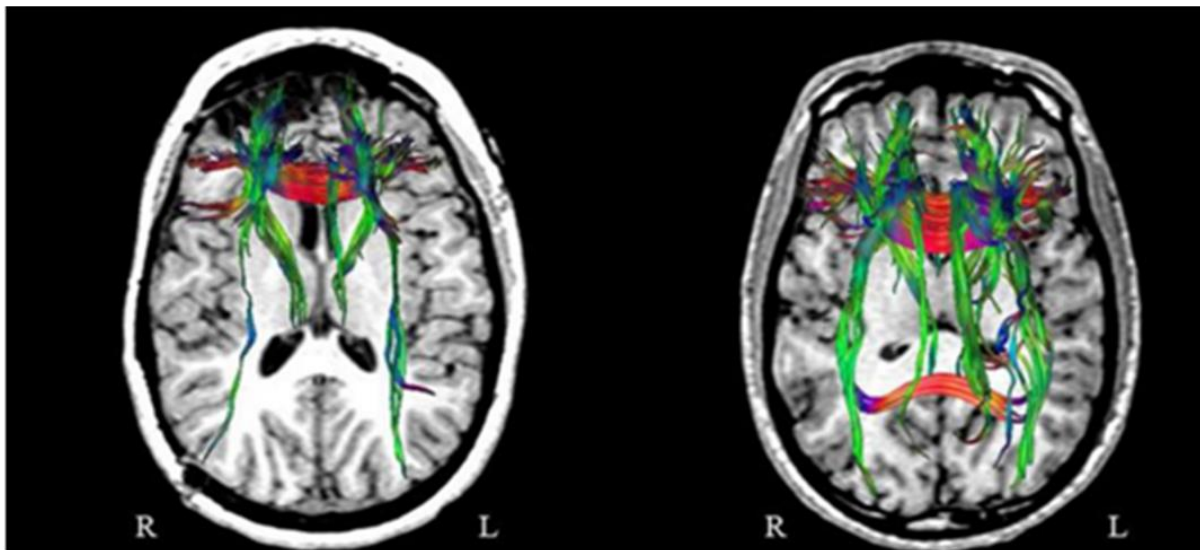
The vulnerability of the frontal lobes, due to their position within the skull and back and forth shearing motions during impact, often leads to pathology which can sever or damage these complex and intricate connections (Schiff, 2010; Shenton et al., 2012). This effect, combined with Wallerian degeneration (neuroinflammation caused by a cascade of cellular and molecular events distal to injury of nerve fibres-Stoll, Jander & Myers, 2002) causes serious disruption to cognitive function after pathology (Bigler, 2007) (Figure 12).

Figure 11 illustrates the intricate connectivity of the frontal lobes while Figure 12 outlines the diminishment of these connections after pathology. Oni et al., (2010) did not present a neuropsychological assessment of the patient in Figure 12 but, with the extent of neuronal pathway loss, it is almost certain that cognitive impairments, specifically social and emotional impairments, would have occurred due to the intensity of damage to the frontal lobes and the association this region has with social cognition (Adolphs, 2001; Brothers, 1990; Satpute & Lieberman, 2006).



**Figure 11. Diagram of frontal lobe connections. (reproduced from Catani, Dell’Acqua, Vergani & Thiebaut de Schotten, 2012, p.15). U-tracts are red, intralobar frontal tracts are yellow and long-range association and projection connections are black. The different functional divisions are; central sulcus connections (yellow area), hand-knob connections (dashed black line area), premotor connections (green area), prefrontal and orbito-polar**

(light red area), dorsolateral longitudinal connections (dashed white line area). AOF, anterior orbitofrontal; FP, frontal pole; IFGop, pars opercularis; IFGtr, inferior frontal gyrus pars triangularis; IFGor, inferior frontal gyrus pars orbitalis; LGN, lateral geniculate nucleus; LP, lateroposterior nucleus; POF, posterior orbitofrontal; SMA, supplementary motor area; VLp, ventral lateral posterior nucleus; VPl, ventral posterior lateral; VPm, ventral posterior medial.

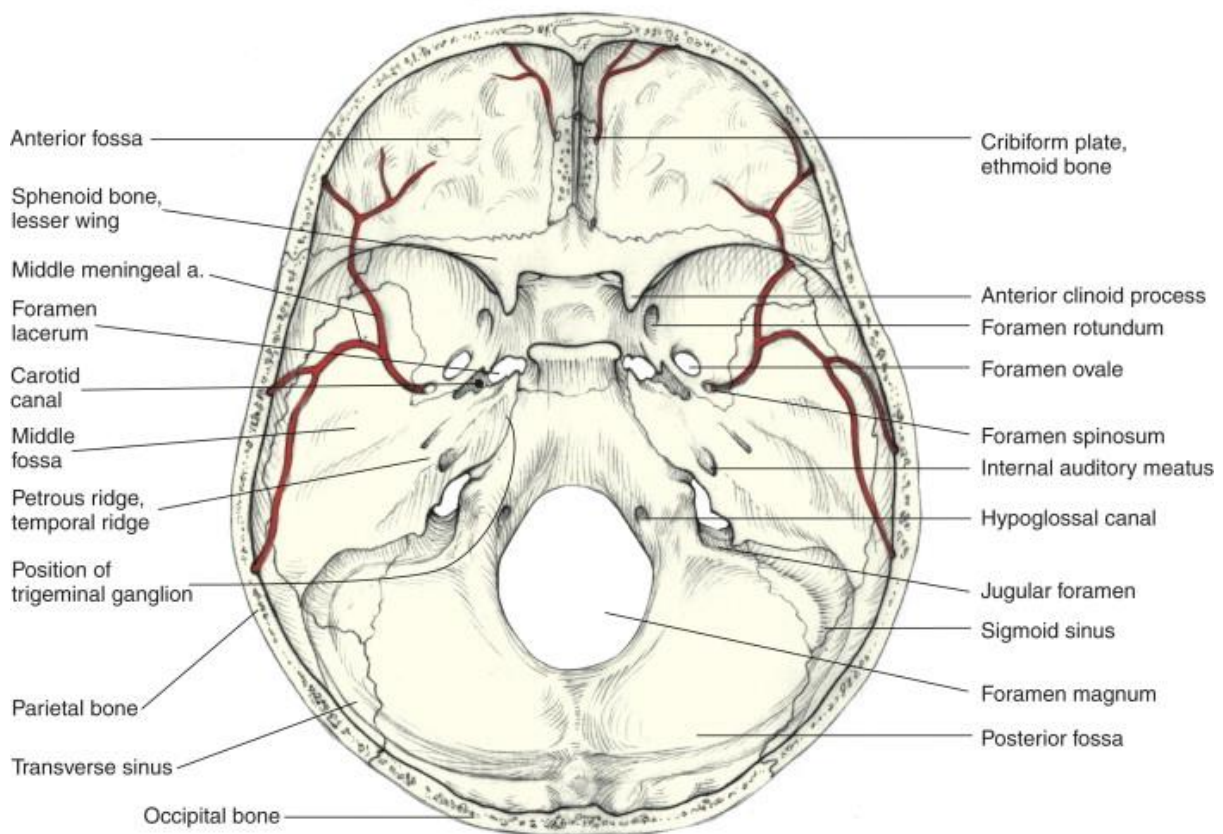


**Figure 10. Diffusion tensor imaging (DTI) data after TBI (reproduced from Oni et al., 2010, p.13). DTI data from a 12-year-old girl (left) who had sustained a severe TBI after hitting the back of her head leading to frontal pathology compared with an age and sex-matched control (right). In the right image, tracts from the frontal region are displayed posteriorly via central white matter pathways and the cingulum as well as interhemispheric connections across the anterior corpus callosum including bi-frontal pathways. In the left image, there is extensive frontal encephalomalacia and all of the pathways are damaged and reduced including an absence of callosal fibres across the posterior corpus callosum.**

## 1.5. Pathophysiology of TBI

An impact to the head produces an array of mechanical and chemical processes which cause pathology to brain matter. Any impact to the head or acceleration, deceleration, velocity alterations/forces or torsion and tension (twisting and stretching) motions can cause neural damage. Brain injury can be categorised as *impact* if the head has direct contact with an object or *non-impact* if the force is a result of a blast or rapid acceleration-deceleration such as whiplash (Prins et al., 2013). Head movements are restricted by the physiology of the neck and can only make limited forward movement before a rebound action occurs. This rebound action produces a momentum shift and leads to pathology during non-impact injuries. When the head and neck are violently forced to extend and flex, brain matter moves within the skull. This movement is due to the soft nature of brain matter (Meaney, Morrison & Bass, 2014). In TBI, brain matter and meningeal membranes impinge upon the irregular and bony surfaces of the skull, stretching and tearing blood vessels and creating lesions (Figure 12 and 13). During impact, the brain collides with the skull which creates a large positive pressure at the coup site (direct site of impact), while the skull opposite the impact briefly enlarges, thus generating negative pressure at the contrecoup site (remote sites of the direct impact –Payne & Payne, 2019) (Figure 14). The rebound motion of the brain produces spikes in pressure as well as shearing and strains at the contrecoup area (Moss, King & Blackman, 2009). The expansion of the skull and the rebound action after this is associated with the tearing of blood vessels crossing from the skull to the dura, leading to extradural haematoma cavities (Chen et al., 2012). Pathology to orbital and ventral surfaces of frontal and ventromedial regions of the temporal lobes are common as they are subjected to compressive pressure when they make contact with the bony protuberances of the skull, particularly the anterior cranial fossa, ethmoid bone, the sphenoid bone and the crista galli located on the cribriform plate (Bigler, 2013; Fujiwara et al.,

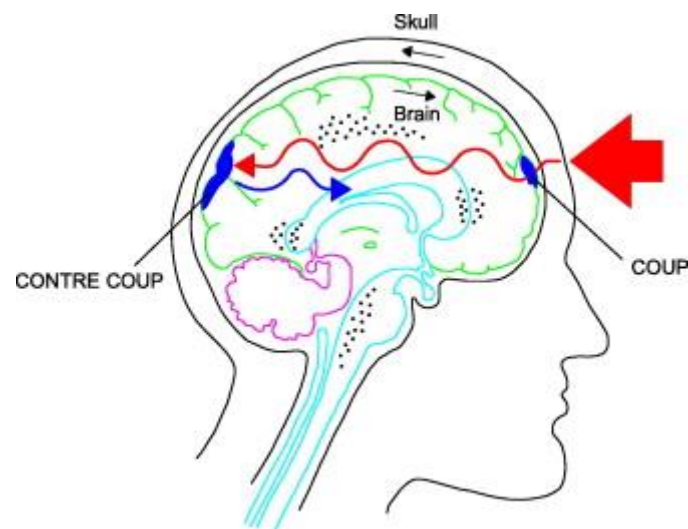
2008; Kolb & Wishaw, 1990; Maas, Stocchetti & Bullock, 2008; McDonald et al., 2013; Taylor & Ford, 2009; Zappala et al., 2012 see Figure 1.13).



**Figure 13. Interior skull surface showing rigid and bony structures of the skull floor (reproduced from Remington & Goodwin, 2012 p.146).**

During impact or acceleration/deceleration forces, the underside of the frontal and temporal lobes slide or 'slap' along these rough skull bones. This effect is due to the brain momentarily lifting away from the skull and then slapping back when the momentum moves in another direction (Bigler, 2013; Gurdjian, 1975). The falx and tentorium often create a region of strain during the impact that can lead to the shearing and stretching of white matter, causing diffuse axonal injury (DAI; Strich, 1956). DAI is related to mechanical disruption of a large number of axons in cerebral hemispheres and subcortical white matter. DAI can induce deep white matter damage, damage to membranes connecting grey and white matter, and damage to

capillary blood vessels known as petechial haemorrhages (Li and Feng, 2009). DAI is theorised to play a critical role in cortical network dysfunction after TBI, even if the injury is mild (Vascak, Jin, Jacobs & Povlishock, 2018). The damage to white matter after TBI is associated with the severity of the injury as well as future outcome (Kraus et al., 2007).



**Figure 14. Coup-contrecoup injury (reproduced from El Sayed, Mota, Fraternali & Ortiz, 2008, p. 4693).**

Evidence indicates that TBI initiates a complex cascade of events leading to primary and secondary damage that varies according to the mechanics and severity of the injury. The primary effects of TBI are immediate and occur as a direct result of the injury. Primary damage includes coup-contrecoup injury, possible skull fracture, focal and diffuse contusions, hematoma, lacerations, DAI, and concussion (Drew & Drew, 2004; Werner & Engelhard, 2007). During the acute stage after TBI (< 1 hour) there is a complex alteration in neurochemical processes including, but not restricted to, the release of glutamate, potassium and calcium within neurons (Prins et al., 2013).

Primary injuries can induce a series of biochemical, physiological and pathological processes, including cellular inflammatory responses and ionic and neurotransmitter alterations, these propagate secondary effects of TBI and lead to abnormal cellular functioning (Prins et al., 2013; Werner & Engelhard, 2007). Typically, apoptosis (a naturally programmed cell death important for neuronal regeneration and repair; Sabirzhanov et al., 2016) and necrosis (a pathological and non-programmed cell death; Fujikawa, 2015) both occur after brain trauma. Subsequent secondary effects, which can develop over hours or even days, may also include infection, hypoxia, oedema and increased intracranial pressure. The main aim of acute management of TBI is to limit secondary effects (Murthy, Bhatia, Sandhu, Prabhakar & Gogna, 2005).

#### **1.6. The diagnosis and classification of TBI**

TBI's are classified as mild, moderate or severe based on the level of primary and secondary injuries. This can be assessed via a range of clinical indices and outcome measures (Friedland & Huthchinson, 2013). TBI is usually classified using a combination of neurological measures including; the length of loss of consciousness (LOC), physical and neurological alertness measured by the GCS, and the length of post-traumatic amnesia (PTA). Less than 30 minutes of LOC indicates a mild TBI, 1-24 hours as moderate TBI, and more than 24 hours as severe TBI. The GCS is used to measure the severity of brain injury using eye, motor and verbal responses and classifies brain injury on a scale as mild (13-15), moderate (9-12) or severe (0-8). The GCS is a quick and simple bedside measure to administer. It is widely used and, therefore, the majority of health professionals know how to administer and interpret the scores. Reviews on the reliability of the GCS are mixed with some research indicating good reliability while others highlighted issues regarding inexperience of administrator (Bledsoe et al., 2015; Mattar, Liaw & Chan, 2015; Reith, Van den Brande, Synnot, Gruen & Mass, 2016). In addition,

there is evidence of inconsistencies between staff members at accident and emergency wards regarding when the GCS should be administered. Marion and Carlier (1994) reported that neurosurgical staff members administered the GCS one hour following admission after hypotension and hypoxia are stabilised, while non-surgical staff members administered the GCS immediately on arrival of the patient, regardless of hypotension and hypoxia. Reith, Brennan, Maas and Teasdale (2016) conducted an online questionnaire with 616 health professionals from different disciplines over 48 countries. Significant discrepancies were documented for the type of stimulus applied when patients do not obey verbal commands (e.g. nail bed pressure, pectoral pinch), with 25% of participants reporting that no stimulus was used. Techniques for reporting GCS scores have major variations with 35% of health professionals only reporting a summary score. There were also large inconsistencies with methods to deal with issues completing certain components of the test, such as sedation. Within the UK, an increased burden on NHS resources might exacerbate this variability of measurement (Robertson, Wenzel, Thompson & Charles, 2017).

PTA can also be used as an indicator of brain injury severity. A definition of PTA was provided in Russell and Smith's (1961) seminal research where they described the event as the period between head injury and the ability of the individual to create continuous memories of daily events. Although PTA is a well-established phenomenon, little is known about the underlying pathophysiological processes, with no standardised practices as to when PTA should be measured. In a review, McMillan (2015) outlined variabilities between clinician's measurement of PTA, highlighting how some clinicians included time spent in coma and others documented it from the point when the patient emerged from a coma. National Institute for Health and Care Excellence (NICE) guidelines have advised that PTA for more than five minutes indicates a need for hospital admission and a head CT scan (NICE, 2019), but no clear guidance is given on when to start measuring amnesia. This ambiguity is disadvantageous when



trying to standardise and index TBI severity. PTA generally encompasses a global memory disturbance in encoding, storage, and retrieval of information. Anterograde amnesia, the inability to maintain new memories, is the most common memory disturbance, although retrograde amnesia, the inability to recall previously stored memories, as well as a tendency to forget information, can also be present (De Simoni et al, 2016; Luoto et al, 2015). PTA lasting 24 hours is supposedly indicative of a severe brain injury, PTA lasting 30 minutes to 24 hours is indicative of moderate injury, and finally, PTA lasting 30 minutes or less is indicative of mild injury (Isaac et al, 2016; Rao and Lyketsos, 2000). Friedland (2013) noted issues with misclassification of PTA, particularly mistaking other factors, such as sedation, intoxication, or memory alternating drug effects (e.g. morphine) for PTA symptoms (Kemp, Agostinis, House & Coughlan, 2010; Ruff, Iverson, Barth, Bush, & Broshek, 2009).

In summary, LOC, the GCS, and PTA are the most commonly used measures to determine the severity of TBI. With improvements in medical treatment, neuroimaging and other physiological biomarker techniques, as well as advancements in published reviews and empirical research, the reliability and validity of the TBI classification system have been brought into question. Indeed, the literature is proposing that a revision of the current system is needed, including more accurate measures and further education and teaching on how to administer the tests (Bledsoe et al., 2015; Levin & Diaz-Arrastia, 2015; Mattar et al., 2015; Reith et al., 2015). Furthermore, LOC, the GCS, and PTA scores can dramatically fluctuate when an individual emerges from a coma, responds to treatment, endures secondary stroke or complicating medical problems. There appears to be a need for a more holistic and linear TBI classification system. Improvement of this system will progress evidence-based research and interventions which will, in turn, mitigate morbidity rates, patient outcomes, and socioeconomic strain. The current research followed guidance from the literature and classified TBI as mild if; LOC and PTA were less than 30 minutes and GCS was 13-15, moderate if LOC

was 1-24 hours, PTA was 30 minutes-24 hours and GCS was 9-12, and severe if LOC and PTA were greater than 24 hours and GCS was 0-9.

## **1.7. Summary**

In summary, TBI is a major cause of death and disability with statistical evidence suggesting that worldwide, approximately 69 million people per year sustain a documented TBI (Dewan et al., 2018; Johnson & Griswold, 2017; Li et al, 2016). TBI can have a devastating effect on the patient and their family, with the high prevalence of TBI creating a serious burden on public health services (Fineberg et al., 2013), creating a need for improved neurorehabilitation programmes (Wiltshire & Ehrlich, 2014). Due to the brains position within the skull and the biomechanical and pathophysiological factors associated with TBI, the frontal lobes and their intricate connections often become damaged (Bigler, 2007). Of note, damage to the frontal lobes is associated with impaired social cognition (Molesworth, Sheu, Cohen, Gianaros & Verstynen, 2015). The prevailing nature of this impairment has been documented as the most challenging and debilitating of all TBI sequelae, for both the TBI patient and their family or caregivers. Consequently, abnormal social skills have a direct impact on functional daily living and quality of life (Brooks, Campsie, Symington, Beattie & McKinlay, 1986; Kinsella Packer & Olver, 1991; Ubukata et al., 2014) yet is often overlooked by clinicians (Kelly, McDonald & Frith, 2017a). The next chapter will discuss and review theories of social cognition and the effect that TBI can have on the psychological processing of social stimuli.

## **2. Chapter two: Social cognition and the effects of TBI**

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### **2.1. Chapter overview**

Social cognition is a complex skill which enables people to understand the social world (Cassel, McDonald, Kelly & Togher, 2019) and likely gives rise to uniquely human constructs such as civilisation and culture (Adolphs, 2009). Despite its importance in everyday functioning, the cognitive and neural underpinnings of social cognition are still poorly understood. Over the past decade, social cognition research has gained traction and there have been several influential papers which have underscored the importance of continued investigation into this field (Cassel et al., 2019; Cotter et al., 2018). Developing a better understanding of social cognition is paramount for the amelioration of disorders associated with poor social interaction and cognition (Happé, Cook & Bird, 2017), particularly after brain injury since those most affected are likely to be young with normal life expectancy (Harrison-Felix et al., 2015; Lawrence et al., 2016).

Individuals with TBI frequently exhibit impaired social cognition including, but not limited to; emotion identification deficits, problems inferring people's thoughts and intentions, difficulties maintaining social relationships, and social inappropriateness (Babbage et al., 2011; McDonald, 2013; McDonald, Rushby, Dalton, Allen & Parks, 2018; Spell & Frank, 2000). However, social cognition is sometimes overlooked in clinical practice and post-injury rehabilitation is not routinely offered (Kelly, McDonald & Frith, 2017a, 2017b; McDonald, 2017). This is surprising as evidence suggests a negative relationship between social cognition deficits and functional outcome post-TBI (Ubukata et al., 2014). For example, reduced subjective emotional experience after TBI, particularly of sadness and fear, has been correlated with poor emotion matching (Crocker & McDonald, 2005), inappropriate behaviour, and poor community re-integration (May et al., 2017).

This chapter will define social cognition and discuss the current challenges related to delineating a standardized factor structure. It will also review the lower and higher-level processing stages of social cognition and the basic neural underpinnings of the specialised ‘social brain’. A specific objective of the current thesis was to explore facial affect recognition post-TBI, which is the processing of others’ emotional expressions. Justification for this decision, as well as an overview of extant facial expression identification research and paradigms in normative and clinical samples, will also be presented. This review will also highlight gaps in the literature, with subsequent chapters detailing how the present research addressed these.

## **2.2. Social cognition: Definitions**

Humans are thought to be among the most social of all primates with the success of the human species possibly associated with superior social cognition abilities compared to other species (Adolphs, 1999; Frith & Frith, 2010). Social cognition is a broad term which can refer to any cognitive process which mediates or facilitates the ability to understand the self, other conspecifics (a member of the same species), or the relationship between the self and other conspecifics (Forbes & Grafman, 2010). There are various definitions of social cognition from very basic explanations such as ‘simply thinking about people’ (Fiske & Taylor, 1995, p.151) to more advanced definitions, for example, ‘psychological processes that are involved in the perception, encoding, storage, retrieval and regulation of information about other people and ourselves. These processes include social cue perception, experience sharing, inferring other people’s thoughts and emotions, and managing emotional reactions to others’ (Green, Horan and Lee, 2015, p.620). This present research adopts Green et al.’s., (2015) definition of social cognition and analogous theoretical framework which recognises the various processing stages,

including lower (perception) and higher (encoding and regulation) levels of cognition, whilst acknowledging the multifaceted nature of social cognition.

### **2.3. Conceptual challenges for social cognition research**

An important challenge for the current research was how to standardise the conceptualisation of the multi-componential processes constituting social cognition. There are several different approaches to social cognition including; developmental (Bandura, 2001), psychoanalytical (Fonagy, 1991), and neuroscientific perspectives (Adolphs, 2009; Frith & Frith, 2007). Each perspective uses a different vocabulary which has more than likely slowed the progress of the research field (Cassel et al., 2019). There is also dispute within the literature regarding the core processes of social cognition. Happé et al., (2017) reviewed the factor structure of social cognition and highlighted the disparity between different authors' division of social cognition abilities. For instance, Green et al., (2015) deduced four social cognitive processes, including; perception, experience sharing, mentalizing, and experiencing/regulating emotion. Fiske and Taylor (2013) proposed 14 domains of social cognition that ranged from low-level processes, such as perception and attention of social stimuli, to higher-level processes, such as decision-making and social attitudes. Happé and Frith (2014) suggested ten components and presented a specific division of the processes, such as agent detection (a human preference for presuming a purposeful involvement of another agent (human or animal) in a social interaction that may or may not involve one), and social policing (taking accountability and mitigating behaviour in terms of moral principles/justice). Besides, there is a lack of consensus within the field regarding which processes should be considered core and which processes are interrelated with these core factors. Happé et al., (2017) argued the need for a standardized taxonomy and vocabulary within the discipline and highlighted the negative effect that loose definitions can have on the replication of studies and mapping of social processes to brain regions. One

example of this disunity is the inconsistency in the definition of social perception and facial affect recognition. Some authors combine the processes, for instance, Adolphs (2010) presented social perception as an overarching principle and classified facial affect recognition as a subcomponent along with auditory and olfactory processing. Other authors distinguish between the two processes and assess them as distinct functions (Barbato et al., 2015).

Mitchell and Phillips (2015) have commented on the lack of agreement in terminology and conceptual frameworks between low-level emotion perception and higher-level theory of mind (ToM: the ability to attribute and understand the mental states of others (Premack & Woodruff, 1978)). Reviewing the existing evidence base, Mitchell and Phillips (2015) explored the similarities and distinctions of emotion perception and ToM at a conceptual, theoretical, and neuroanatomical level. They concluded that the two functions were distinct yet overlapped on multiple levels. Mitchell and Phillips (2015) recommended that when both processes are discussed in a single study, the authors should be explicit regarding the distinctions between the two concepts, including definitions and the extent to which the experimental tasks make emotional and cognitive demands. This position contrasts with work from Njomboro, Deb and Humphrey (2008) who proposed that emotion perception was a sub-component of ToM. Presently, these conceptual and methodological issues remain unresolved and further clarity is warranted to better inform socio-cognitive models and methodological designs.

A further challenge arises when researchers consider how to measure such complex multifaceted processes. Broadly, social cognition depends upon social and non-social cognitive processes and behaviours, for example, perception, attention, memory, reward/punishment learning, and cognitive flexibility. Whether these processes are related or distinct is not yet clear. Evidence suggests a dissociation between classic cognitive (e.g. executive functions: a set of higher-order cognitive processes which allow humans to monitor and control behaviour),

and social-cognitive processes in individuals with autism (Miranda, Berenguer, Roselló, Baixauli & Colomer, 2017), Alzheimer's disease (Cosentino et al., 2014), schizophrenia (Green et al., 2015), and frontotemporal dementia (Lough, Gregory, Hodges, 2001). Furthermore, individuals with frontal pathology from trauma frequently present with intact or mildly impaired intellect but chronic impairments in social functioning (Blair & Cipolotti, 2000; Tranel, Bechara & Denburg, 2002). These findings suggest that social and non-social cognitive processes are dissociable in certain pathological conditions, although this may partly reflect the functions measured. There is likely to be crosstalk between social and non-social cognitive processes for adaptive social functioning. For example, the decoding of facial expressions relies on several non-social processes such as vision, attention, and memory. Indeed, perceptual processes are pivotal to social functioning, with evidence suggesting that humans have evolved specialised brain areas for social perception, for instance, the fusiform face area (FFA, Schultz et al., 2003) and amygdala (Rutishauser, Mamelak & Adolphs, 2015). Aberrations in these systems are related to disorders such as autism (Schultz, 2005) and schizophrenia (Mukherjee et al., 2013), both of which are clinically characterised by impairments in social cognition (Gall & Iakimova, 2018).

In a systematic review of meta-analyses across 30 clinical conditions, Cotter et al., (2018) reported that social cognition impairments were a core phenotype or marker. In many of these conditions, social cognition deficits were similar in magnitude to 'classic' cognitive functions, such as memory or executive functions. The conditions were diverse, including; psychiatric disorders (e.g. bipolar disorder and anorexia nervosa), neurological disorders (e.g. Alzheimer's disease and Huntington's disease), and developmental disorders (e.g. autism and attention deficit hyperactivity disorder). Cotter et al., (2018) theorised that social cognition abilities can be distinguished from non-social abilities that could potentially be used as trait markers or endophenotypes for many neurological or psychiatric conditions, particularly autism and

schizophrenia (Derntl & Habel, 2011). However, Dodell-Feder and Germine (2018) cautioned against making generalised inferences across conditions as the cause and nature of social impairments usually differ according to the condition and other inter-individual factors. For example, autistic children display social cognition deficits from a young age, whereas an individual with a brain injury may have developed typical social cognition, yet this ability has been selectively diminished as a result of pathology. The neurological underpinnings of the two conditions are, therefore, disparate, and this needs to be taken into account when assessing social cognition and offering intervention or therapy.

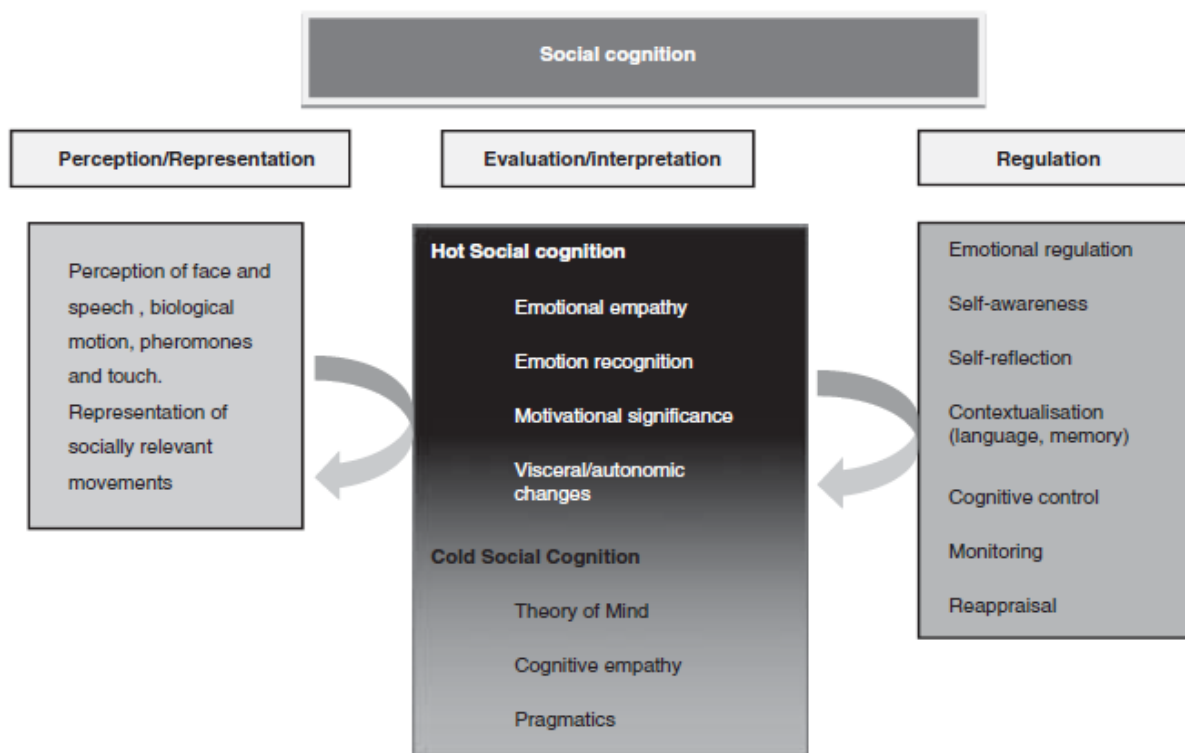
Beer and Ochsner (2006) define social cognition through an interdisciplinary approach, bridging psychology and cognitive neuroscience. The authors advised that a comprehensive account of social cognition should take into consideration; the processing of other people, the self, social norms and procedures of the social world. More than likely, these processes will occur on both an implicit and explicit level and will be subjective to; motivational biases, the context of the situation, previous experience, and declarative (e.g. social scripts and schemas) and procedural (social rules and skills) knowledge. Beer and Ochsner (2006) highlighted that understanding the social world, including the self, encompasses multiple stages of processing, as well as the use of information from all of the sensory channels to process both verbal and nonverbal cues. Beer and Ochsner (2006) underscored that the interconnected and overlapping nature of the psychological processes involved in social cognition would be supported by nearly every neural network (e.g. vision, language, hearing, memory), leaving social cognition module theories open to scrutiny on the basis that they are biologically implausible as they do not correspond to neural substrates. Later work by Ochsner (2008) proposed a framework based on the premise that human social and emotional behaviours are highly intertwined, identifying that emotions arise from appraisals of the goal relevance of a stimulus, and people are typically the most goal-relevant stimuli during daily lives. To this end, Ochsner proposed the *social-*



*emotional processing stream* which denoted a collection of psychological and neural processes that encoded social and emotional inputs, represented their meaning, and guided responses. The stream consisted of five core constructs; (i) acquisition of social-affective values and responses, (ii) recognition of and response to social-affective stimuli, (iii) embodied simulation or low-level mental state inference, (iv) high-level mental state or trait inference, and (v) context-sensitive regulation. Ochsner (2008) then considered translational examples of how each construct could aid in the understanding of negative symptoms in schizophrenia (e.g. pervasive lack of emotional expressivity, abnormal emotional experience, lack of motivation, and asociality) and other clinical samples, such as TBI. For example, with regards to constructing (ii), Ochsner proposed that the decoding of subtle nonverbal social cues could be challenging for individuals with schizophrenia, particularly cues which convey the intentions of conspecifics. One illustration provided by the author was that individuals with schizophrenia may exhibit heightened amygdala activation to patterns of eye gaze that normally are not considered threatening to non-schizophrenic individuals. The above work provides a basic framework for exploring the relationship between social cognition and social functioning and supports the proposal that social cognition impairments in clinical populations may result in difficulties with social behaviour and poor social outcome.

A third challenge stems from reports of dissociations between certain social cognition processes. For example, some researchers make distinctions between ‘hot’ social cognition (e.g. emotion processing and empathy) and ‘cold’ social cognition (e.g. ToM) (McDonald, 2013; Figure 15). Another example comes from reports that several neurological disorders are associated with social cognition impairments but differ in terms of social deficits and their developmental courses. For example, the clinical diagnosis for both autism (Hoffman, Brück, Kreifelts, Ethofer & Wildgruber, 2016; Rosenblau, Kliemann, Heekeren & Dziobek, 2015), and schizophrenia (Schilbach et al., 2016; White, Borgan, Ralley & Shergill, 2016) include

problems identifying social cues and mentalizing. Impairments in autism develop early in life compared to impairments in schizophrenia that typically develop during adolescence or adulthood. Frontotemporal dementia often occurs later in life and is also associated with mentalizing problems (Caminiti et al., 2015), while conduct disorder, which can be diagnosed from a young age, is associated with diminished empathy, disinhibited behaviour, and emotion sharing problems, yet mentalizing abilities remain intact (Winter, Spengler, Bempohl, Singer & Kanske, 2017).



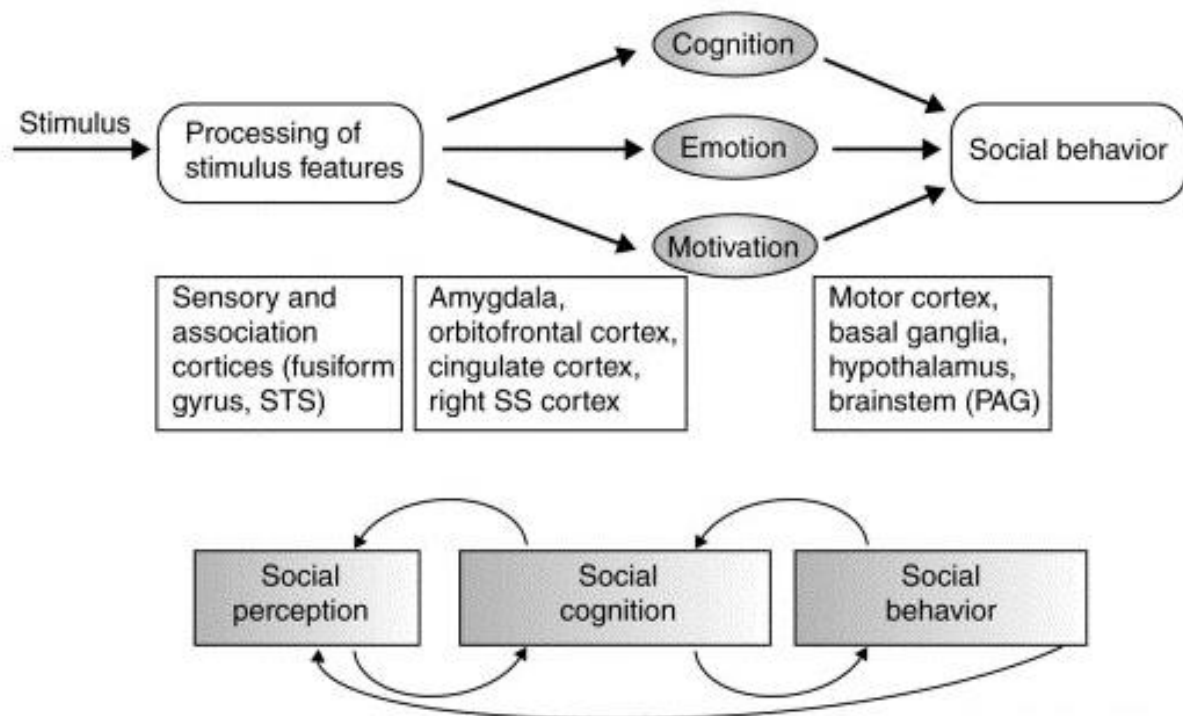
**Figure 15. Processes during social cognition (reproduced from McDonald, 2013, p. 232).**

Shamay-Tsoory, Aharon-Peretz and Perry (2009) reported a double dissociation between affective empathy (the implicit process of sharing others' emotional state) and cognitive empathy (the explicit process of trying to understand others' emotional state). They suggested a two-system basis for empathy, with increased activation in the inferior frontal gyrus during affective empathy compared to increased activation in ventromedial regions during cognitive

empathy. However, the concept that specialised functions (e.g. facial affect recognition, memory, empathy) can be modularised to specific brain areas is contentious (Dias, 2009; Shettleworth, 2012) as the brain is a connectome. Dissociations between social-cognitive processes are seen in abnormal and pathological conditions, but there are also multiple inter-relationships between cognitive processes. For instance, Neumann, Zupan, Malec and Hammond (2014) suggested that alexithymia (difficulty in identifying and describing emotions) is associated with impaired affect recognition and cognitive empathy but not affective empathy. Emerging reports appear to consider social cognition as a construct which relies on several different components working in unison and, therefore, a primary concern is how to operationalise these functions for empirical study.

One approach involves isolating a specific social process (e.g. facial affect recognition) and investigating this process using different experimental paradigms (Figure 16). This reductionist approach (i.e. investigating part of the process thoroughly compared to the whole phenomenon) facilitates the development of operational variables and makes a complex process easier to test. Isolating the constituent parts of social cognition and gaining a comprehensive understanding of a specific process should, in theory, aid the understanding of social cognition as a whole. Nevertheless, the reductionist approach has an obvious limitation in that it reduces behaviour down to, for example, brain structures or neurotransmitters, and omits other influential factors, for instance, culture or the combination of social processes (e.g. the effect of context, tone of voice, and facial expressions on the behavioural response). This produces a simplistic overview of a multifaceted process and generating theoretical frameworks which may lack validity (van Kleef, Heerdink & Homan, 2017). However, adopting a holistic approach to the investigation of social cognition would be challenging as these functions span low-level social perception to complex and higher-level ToM. At present, it is common practice in affective neuroscience to

employ the reductionist approach, with the collation of findings is providing an insightful overview of social cognition.



**Figure 16. Diagram illustrating the processing of social information (reproduced from Adolphs, 2001, p.232). At the input stage, social cognition involves neural networks associated with perception and the recognition and evaluation of stimuli, subsequently supplying the information which is necessary to construct central representations of the social world. Several brain regions and structures are involved in this process including, but not limited to, the fusiform gyrus and the superior temporal sulcus working together with a network of structures. This includes; the amygdala, orbitofrontal cortex, anterior and posterior cingulate cortices, right somatosensory-related cortices, motor and premotor cortices, basal ganglia, hypothalamus, periaqueductal grey (PAG), and visual system. Critically, social perception, social cognition, and social behaviour are interconnected as depicted by the bottom of the diagram.**

#### 2.4. Stages of social cognition

Adolphs (2010) proposed three stages of social cognition; perception of social information, social understanding, and social behaviour (Figure 15 and 16). Adolphs' (2010) framework

was developed from earlier schizophrenia models of social cognition proposing; perception of social cues, interpretation of social information, and processing of social information (Ostrom, 1984; Penn, Corrigan, Bentall, Racenstein & Newman, 1997). Social perception refers to the initial stage of social cognition where visual cues, such as eye gaze (Allison, Puce & McCarthy, 2000), auditory cues (Blake et al., 2015) and olfactory cues (Lahera et al., 2016) are encoded. During social interaction, an individual must extract relevant information whilst inhibiting irrelevant information from the environment. Visual cognition is restricted by computational capacity as the human brain can only process a finite amount of information. Two mechanisms moderate this constraint: attention prioritizes stimuli based on motivational relevance, and expectations limit visual interpretation based on prior likelihood (Summerfield & Egner, 2009). Less salient information must be ignored and relevant information must then be interpreted (processed) to formulate appropriate responsive behaviour. Penn et al., (1997) proposed that the amalgamation of these stages is the basic underpinning of social cognition. A strength of this particular model is that it incorporates early attentional and visual processing. These initial low-level functions influence upstream cognitive processes, yet many studies fail to acknowledge, assess, or even consider the effect they may have on higher-order cognitive abilities (Pinkham, Penn, Perkins and Lieberman, 2003). Nevertheless, frameworks which present cognitive processes as linear and sequential pathways are likely to be over-simplistic. Perception alone is a dynamic interactive process, and the combination of social cognition functions are likely to proceed in parallel and through constant iterative feedback loops.

## **2.5. Implicit and explicit social cognition**

Another important consideration in social cognition research is the distinction between low level, implicit processes that largely occur in the absence of awareness and higher-level explicit processes (Frith & Frith, 2008). Most social cognitive processes are thought to be implicit and

processed automatically without the influence of conscious processes, allowing people to respond swiftly in social situations (Bargh & Williams, 2006; Forbes & Grafman, 2010). Unconscious processes may underpin several social cognitive functions, including but not confined to, emotion recognition and gesture interpretation (Barker, Andrade, Morton Romanowski & Bowles, 2010; Nosek, Hawkins & Frazier, 2011). Implicit processes are crucial for increasing the success of group behaviour, for example, the generation of good rapport through unconscious mirroring of other's behaviour (Chartrand & Bargh, 1999).

Explicit processes depend upon conscious thought and are generally slower but more dynamic than implicit processes (Frith & Frith, 2008). These processes are thought to contribute to personal adaptability and goal-directed behaviours, such as collective responses to danger (e.g. raising the alarm to a fire) and acts of persuasion and deception (Frith & Frith, 2012). Satpute and Lieberman (2006) proposed a working model that distinguished between the reflexive implicit system (X-system) and a reflective explicit system (C-system). The brain regions associated with the X-system were thought to be slow learning, rapid operating, bi-directional and able to parallel-process information. These implicit systems included the amygdala, basal ganglia, ventromedial prefrontal cortex, dorsal anterior cingulate cortex and lateral temporal cortex, including the superior temporal sulcus (STS); essentially, midbrain areas and associated networks. C-system areas were proposed to incorporate rapid learning, slow operating functions mediating symbolic or propositional functions, including the lateral prefrontal cortex, posterior parietal cortex, rostral anterior cingulate, medial temporal, and medial prefrontal cortices.

A strength of Satpute and Lieberman's (2006) model is that it recognises the distinction between cognitive and social processes and implicit and explicit processes. The model amalgamates the two by identifying shared and distinct brain regions emphasising the

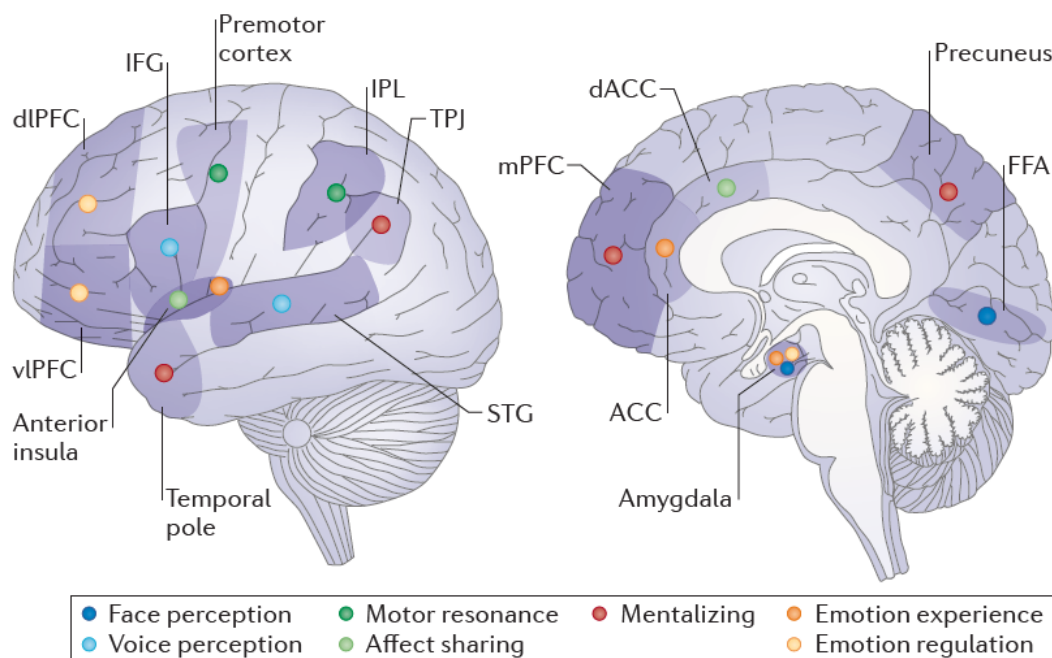
importance of parallel connectivity. In sum, Satpute and Lieberman (2006) presented a biologically plausible model of social cognition and their framework is mirrored by other scholars (e.g. Forbes & Grafman, 2013). Nonetheless, the existence of discreet X- or C-systems in the brain may be oversimplified with recent research reporting a complex interaction between regional activations and connectivity changes when different tasks are performed, for example, recognising emotion compared to a language task (Di & Biswal, 2019). Similarly, there is debate within the literature regarding dissociations between implicit and explicit processes. Some researchers advocate an independent role for the two processes (Frith & Frith, 2008), while others support a more complex synergistic relationship (Forbes, Cox, Schmader & Ryan, 2011; Forbes & Grafman, 2013).

## **2.6. The social brain**

### **2.6.1. Brain networks**

The neurological underpinnings of social cognition are complex and appear to be dependent upon a variety of cortical and subcortical areas and connective circuitry (Van Overwalle, 2009). The term ‘social brain’ was coined by Gazzaniga (1985) as a result of research investigating social deficits after right hemisphere damage. The term is now used to refer to social brain functions (Glozman & Krukow, 2013). Brothers’ (1990) seminal research with primates advanced the social brain theory and identified multiple brain networks and structures including; the amygdala, prefrontal cortex, and the temporal poles, as the neural basis of the social brain. The evidence for Brothers’ (1990) model was mainly based on monkey lesion studies; for example, amygdala lesions resulted in social isolation (Kling & Brothers, 1992), and orbitofrontal lesions negatively impacted social behaviour, such as reduced grooming and huddling (Raleigh & Steklis, 1981). However, Brothers (1990) work is limited as it was based on animal data which does not always translate well to humans. For instance, although primates

and humans both tend to have social and hierarchical groups, humans have additional complex factors, such as finances, culture, and language, which impacts upon social constructs. Nevertheless, the underlying principle of Brothers (1990) hypothesis has been partially supported by neuroimaging and human lesion studies that have consistently associated a strong involvement of frontal and limbic micro-circuitry (Amodio & Frith, 2006; Bicks, Koike, Akbarian & Morishita, 2015; Wittmann, Lockwood & Rushworth, 2018; Xiao, Jacobsen, Chen & Wang, 2017). It should be noted that although the existing evidence base suggests a specialised role of the frontal and limbic systems during social cognition, the social brain network is thought to vary depending on task demands and social context and likely draws on a widely distributed brain network (Frith & Frith, 2006; Fujiwara & Bartholomeusz, 2010; Figure 16).



**Figure 17. Brain areas related to social cognition (reproduced from Green et al., 2015, p.625). Social perception can be divided into facial perception, reported to rely on amygdala and FFA, and voice perception, related to superior temporal gyrus (STG) and**



**inferior frontal gyrus (IFG) activation. The ability to share experiences with others may be related to motor resonance (activation of observer's motor system when they perceive a conspecific performing an action) which is associated with inferior parietal lobule (IPL) and premotor cortex, and affect sharing which appears to rely on the dorsal anterior cingulate cortex (dACC) and anterior insula. Mentalizing (understanding the mental state of ourselves and others) is reported to rely on several brain regions including the temporoparietal junction (TPJ), temporal pole, precuneus and medial prefrontal cortex (mPFC). Experiencing emotions activates the amygdala, anterior hippocampus (not shown), ACC and anterior insula, while the regulation of emotions is associated with the dorsolateral PFC (dlPFC), ventrolateral PFC (vlPFC) and amygdala. It is evident that several brain structures are associated with several social processes (e.g. the amygdala and anterior insula) and those social processes, such as empathy and emotion recognition, may be inter-related.**

### **2.6.2. Neuropeptides**

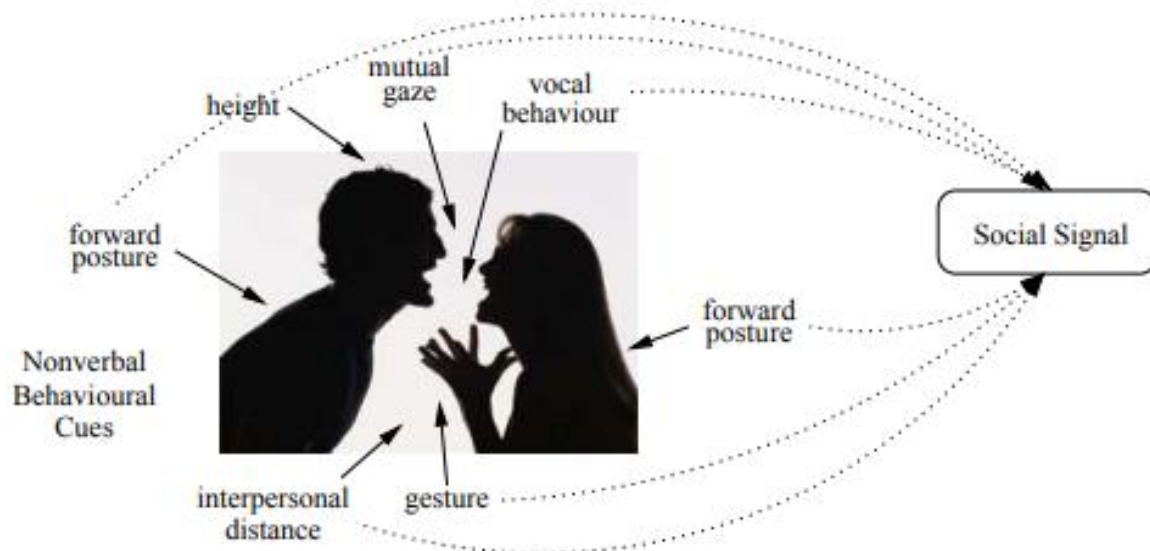
The role of neuropeptides in human social cognition is critical to understanding the biological basis of human social cognition. Two of the most well researched human social neuropeptides are oxytocin and vasopressin. Oxytocin is produced in the hypothalamus and released by the posterior pituitary gland (Fineberg & Ross, 2017). The key pathways through which oxytocin modulate social functioning include; communication with serotonergic systems in the nucleus accumbens, interaction with the amygdala and other limbic structures, and direct projections to brainstem nuclei (Fineberg & Ross, 2017). Oxytocin plays a pro-social role in human behaviour by diminishing behavioural and neuroendocrine responses to social stress, therefore, facilitating approach behaviour and decreasing avoidance behaviour (Heinrichs & Domes, 2008). Oxytocin increases the formation of trusting behaviour (Kosfeld, Heinrichs, Zak, Fishbacher & Fehr, 2005), cooperation (Declerck, Boone & Kiyonari, 2010), social bonds (Bosch & Young, 2017), and the perception and processing of faces (Theodoridou, Rowe, Penton-Voak & Rogers, 2009). Additionally, oxytocin appears to increase empathy levels (Geng et al., 2018), in-group compared to out-group preferences (Ten Velden, Daughters & De Dreu, 2017), and social motivation and reward (Gordon, Martin, Feldman & Leckman, 2011).

Vasopressin is synthesized in the hypothalamus and is released to several brain regions including; the amygdala, cingulate gyrus, dorsal hippocampus, and caudate nucleus (Cuzzo & Lappin, 2019; Dumais & Veenema, 2016). The role of vasopressin on human social behaviour is less researched compared to oxytocin, but the neuropeptide appears to produce an anxiogenic effect (causing anxiety). Males produce more vasopressin than females (De Vries & Panzica, 2006) and has been implicated in male-typical social behaviours, particularly aggression during pair bonding (Caldwell, Lee, Macbeth & Young, 2008; Goodson & Bass, 2001). It has been hypothesised that there may be an association between higher levels of vasopressin and a bias to respond to neutral social stimuli as threatening (Thompson, Gupta, Miller, Mills & Orr, 2004).

The specific mechanisms underpinning the effects of neuropeptides on social cognition are still under investigation but it is evident that both oxytocin and vasopressin modulate vital social behaviours, including prosocial behaviour, attachment, social recognition, and defence behaviours (Geng et al., 2018; Heinrichs & Domes, 2008). Interestingly, oxytocin is being considered as a psychopharmacological enhancement for social cognition disorders and has been reported to increase empathy levels in individuals with schizophrenia (Davis et al., 2014) and emotional ratings of faces in autistic individuals (Quintana et al., 2017). Despite these positive effects, current ethical considerations and methodologies are problematic in human cohorts and present findings are equivocal (Bradley & Woollet, 2017).

## **2.7. Facial affect recognition**

Humans rely heavily on visual cues, particularly non-verbal visual communication to negotiate social contexts (Beattie & Ellis, 2014). Birdwhistell (2010) postulated that during a dyadic conversation, only one-third of the social meaning is conveyed by verbal components, the remaining two-thirds are conveyed by non-verbal components (Figure 18).



**Figure 18. Combined verbal and non-verbal social signals during a dyadic interaction (reproduced from Vinciarelli, Salamin & Pantic, 2009, p.43). Verbal cues include language, tone of voice, and intonation. Non-verbal cues include posture, personal space, expressions and gestures. The combination of these signals allows humans to decipher a social situation, for example, with the above image, hostility, aggressiveness and disagreement.**

Indeed, Mehrabian (1971) proposed that up to 93% of a conversational message is conveyed by non-verbal components. These non-verbal components are usually visually processed and include aspects of human behaviour such as eye contact, facial expressions, body language and gestures (Knapp & Hall, 2002). According to Argyle (2013), there are five main functions of non-verbal communication; to express emotion, facilitate interpersonal relationships, accompanying speech to create a feeling of synchronisation, self-presentation, and to uphold cultural rituals, for example, handshaking. The ability to correctly identify, assimilate and engage paralinguistic features, non-verbal and contextual cues from the social environment is vital in behaving appropriately during social situations (Mah, Arnold & Grafman, 2004).

The present research was interested in exploring the visual non-verbal communicators of mood state, specifically emotion perception through facial expressions. The human face is one of the

most communicative modes utilised during social interactions, and correct identification of facial expressions is central to adaptive social functioning (Hinojosa, Mercado & Carretié, 2015; Jack & Schyns, 2015). Human faces display visible signals of social intentions, motivations, and communicate internal emotional states (Hess & Hareli, 2015; Schmidt & Cohn, 2001). It is generally accepted that there are six basic facial expressions/emotions which are recognisable across cultures; anger, fear, disgust, happiness, sadness and surprise (Ekman & Keltner, 1997). Nevertheless, there is still a lack of consensus between theorists regarding a common taxonomy of emotions (Izard, 2009, 2010; Turner & Stets, 2006), mainly because they are investigating phenomena from different theoretical positions.

As well as emotions, human faces also display intention gestures and signals, such as the eyebrow flash which indicates a desire to communicate (Frith, 2009), yawning, which is theorised to promote emotional contagion (the transfer of emotion and mood to other individuals (Ferrari & Coudé, 2018), and eye gaze (Slonimska, Campisi & Ozyurek, 2015), which is the basis for *joint attention*; the ability of two or more individuals to concurrently utilise gestures or eye gaze to focus attention on salient objects or events in the environment (Jones & Carr, 2004). These gestures are critical for adaptive social behaviour as they allow humans to perceive and interpret other people's intentions, thoughts, and behaviours during social interactions, as well as mediating the generation of contextually appropriate social responses (Green et al., 2008). Interestingly, Elder (2018) explored the use of emojis (ideograms displaying facial expressions) in textual computer-mediated communication. The research concluded that the use of emojis had a positive effect on individual well-being and interpersonal communication, directed attention, expressed or acknowledged difficult emotions, and increased altruistic tendencies, highlighting the human reliance on facial expressions for communication.

Facial expressions can also be used to build rapport consciously through friendly displays (Grahe & Bernieri, 1999), and unconsciously through a process of mirroring (the chameleon effect which is the unconscious imitation of facial expression, gestures or speech patterns; Lakin, Jefferis, Cheng & Chartrand, 2003; Tramacere & Ferrari, 2016). The evolutionary benefit of mirroring is that it drives humans to unconsciously produce survival behaviours. For instance, a fearful face is indicative of a threat and the physiological facial adaptations associated with fear (e.g. widening of the eyes and nostrils) enlarge the visual field, inspiratory capacity, and sense of smell (Susskind et al, 2008).

Humans exhibit an innate preference for faces and evidence has proposed that the human visual system rapidly identifies human faces compared to other visual stimuli (Frank, Vul & Johnson, 2009). Eye-tracking research has reported that human saccades (quick movements of the eye in preparation for a fixation) are exhibited as early as 100 milliseconds after stimulus onset for happy faces (Crouzet, Kirchner & Thorpe, 2010). When faces are paired with other objects (e.g. vehicles), initial saccades are biased towards faces, even when the other object is the target, suggesting that these quick saccades are not under conscious control (Crouzet et al., 2010). This unconscious bias appears to extend to social cues from facial expressions (e.g. eye gaze). Deaner and Platt (2003) reported that when participating in a peripheral visual target detection task, macaque and human performance levels were both facilitated when eye gaze was consistent with the target location. These findings indicated that primates displayed innate covert attention to face stimuli. Abnormalities within this social perception system, for example avoiding looking at the eye area of a face, have been related to poor social functioning in autistic cohorts (Tanaka & Sung, 2016).

The neural networks underpinning facial affect recognition are extensive and beyond the scope of detailed discussion in the current thesis. The recognition of facial expressions relies on both

low and high-level processes, activating early sensory cortices (discussed in detail in chapter three), subcortical structures, and widespread cortical regions. In brief, Phillips, Drevets, Rauch and Lane's (2003) neurobiological model of emotion perception suggests two emotion perception neural systems. Firstly, a ventral system, predominantly important for identifying emotion valence and producing appropriate affective responses, and included the amygdala, insula, ventral striatum, and ventral regions of the anterior cingulate gyrus and prefrontal cortex. Secondly, a dorsal system, necessary for the regulation of affective states, and included the hippocampus and dorsal regions of the anterior cingulate gyrus and prefrontal cortex. TBI, even mild, has been associated with structural and neurochemical brain changes, including DAI, in the aforementioned regions (Eierud et al., 2014; Mayer, Mannell, Ling, Gasparovic & Yeo, 2011), and could account for the frequently reported emotion perception impairments post-TBI (Babbage et al., 2011).

In summary, facial expressions are arguably the most important conveyor of human mood state and aid social behaviours such as emotional contagion and joint attention. Even in the 'digital age', where there has been a reduction in face to face, humans have perceived the need to develop a facial expression system to facilitate the message they are trying to convey, highlighting human dependency on this intrinsic ability (Elder, 2018). Facial affect recognition draws on a diverse set of brain networks including; early sensory cortices, subcortical structures, and widespread cortical regions, notably the frontal cortex and the motion detection regions of the parietal cortex (Adolphs, 2009; Phillips et al., 2003). Facial affect recognition was identified as the primary focus for the current thesis because facial expressions are considered to be the most important non-verbal conveyor of mood state in humans (Croker & McDonald, 2005; Frith, 2009), and impairments in understanding facial expressions post-TBI are well-established but poorly understood (Babbage et al., 2011; May et

al., 2017). The next section critically reviews the effects of TBI on social cognition with a specific focus on facial affect identification.

## **2.8. Social functioning post-TBI**

Social cognition impairments after TBI are well documented and can include behaviours such as self-focused conversations, the inability to express, inhibit or regulate emotions, lack of empathy, inability to detect nuanced expression such as sarcasm, and lack of social etiquette (Cattran, Oddy, da Silva Ramos, Channon, PelliJeff & Rule, 2005; McDonald, 2000; McDonald & Pearce, 1996). Some authors have proposed that social cognition impairments, compared to other cognitive deficits, are the greatest barrier to adjustment and rehabilitation after brain injury (Grattan & Ghahramanlou, 2002; Ponsford & Schönberger, 2010; Yates, 2003). Indeed, research exploring the functional outcomes of TBI survivors has typically focused on classical cognitive dysfunction, such as memory and information processing speed deficits, but correlations are usually weak and poor predictors of post-TBI social outcome (Ownsworth & McKenna, 2004; Wood & Rutterford, 2006). Social cognition impairments may be a better predictor of community reintegration and measure of assisted living needs post-TBI (Ubukata et al, 2014). For example, May et al., (2017) found that poor facial affect recognition was related to lower community outcome and social functioning post-TBI. In their study, May et al., (2017) reported that 40 mixed aetiology TBI participants, aged 19-60, performed significantly poorer on two emotion recognition tasks (dynamic facial expressions and morphed facial expressions), ToM tasks (faux pas and hinting test), and cognitive flexibility tasks (stoop and go/no-go) compared to controls. The authors also administered three socio-emotional behaviour questionnaires to measure self-report and proxy ratings of post-TBI behaviour. Findings suggested that the group affected by TBI had developed behaviour problems post-injury, including poor interpersonal behaviour, as well as lower levels of

community integration compared to before the injury and control group. Emotion recognition was related to post-injury behaviour, measured through a subset of the Dysexecutive Questionnaire proxy ratings (DEX; Burgess, Alderman, Wilson, Evans & Emslie, 1996) with higher emotion recognition being related to better community outcome and social functioning.

Ubukata et al., (2014) also investigated social cognition and functional outcomes in a post-TBI group using the Revised Craig Handicap Assessment and Reporting Technique (R-CHART: Mellick, 1999). The study focused on facial emotion perception (The Japanese and Caucasian facial expressions of emotion set: JACFEE; Matsumoto & Ekman, 1988), social emotion perception (Eyes Test: Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001), and ToM (Faux-Pas test: Stone, Baron-Cohen & Knight 1998). An original factor in Ubukata and colleagues (2014) research was that it was one of the first studies to explore multiple domains of social cognition concerning social functioning in a moderate-severe TBI cohort. This multi-domain approach is important for collecting ecologically valid results and developing efficient rehabilitation programmes post-TBI. Findings from the study revealed that, compared to control data (extracted from the author's data set and not matched for age and gender), the group with TBI were impaired on all social cognition measures, as well as having lower scores in social participation compared to controls. As participants were not matched for age or gender, care should be taken when drawing conclusions from the research. Nevertheless, there was a negative relationship between nonverbal ToM (reading the eyes test) and cognitive independence (ability to sustain an effective independent existence) measured through the R-CHART. The non-significant correlation between the facial affect task and the R-CHART could be attributed to the static nature of the facial expression task as it provided the perceiver with an apex emotion for a sustained period. It should be noted that Knox and Douglas (2009) employed a similar methodology to Ubukata et al., (2014) and reported that TBI participants had significantly lower scores on the JACFEE (Matsumoto & Ekman, 1988), and on a matching



emotions to social situations task compared to matched controls. Furthermore, Knox and Douglas (2009) reported a significant correlation between the R-CHART and scores in the JACFEE (Matsumoto & Ekman, 1988). May et al., (2017) and Ubukata et al., (2014) results emphasise the importance of investigating the factors which contribute to impairments in facial expression recognition after TBI as they seem to adversely affect functional outcomes. As TBI is most common in young adults (Marshman et al., 2013), who will go on to have a relatively normal lifespan expectancy (Harrison-Felix et al., 2015), the need to understand social problems and functional outcomes post-TBI is of paramount importance.

Abnormalities in social functioning can lead to reductions in friendship group size (Hoofien, Gilboa, Vakil & Donovan, 2001; Salas, Casassus, Rowlands, Pimm & Flanagan, 2018), increased strain on family relationships (Schönberger, Ponsford, Olver and Ponsford, 2010), and breakdown of a marriage or long-term relationships (Wood, Lioffi & Wood, 2005). Hoofien et al., (2001) reported that in their sample of 60 moderate-severe TBI participants, 30% had very few friends and rarely took part in social activities 10-20 years post-injury. These findings are replicated by other research which has demonstrated that individuals with TBI find it hard to maintain social relationships (Salas et al., 2018; Struchen, Pappadis, Sander, Burrows & Myszka, 2011), feel isolated, lonely, depressed, and overall rate a low quality of life (Ditchman, Keegan, Batchos, Haak & Johnson, 2017; Lefebvre et al., 2008). Families of TBI survivors have described increased daily hardships, such as changes to relationships and losing contact with social networks (Arango-Lasprilla et al., 2008; Carozzi et al., 2015; Wood & Rutterford, 2006), with many reporting that changes in emotional and social abilities were more disabling and difficult to deal with than cognitive or physical changes (Kelly, Brown, Todd & Kremer, 2008; Koskinen, 1998; Marsh, Kersel, Havill & Sleight, 2002). Individuals with poor social skills frequently do not return to work (Douglas, Bracy & Snow, 2016), and when they do it is usually in a lower position within the company (Temkin et al., 2009). There are

considerable benefits of employment including financial independence, a sense of ‘social cohesion’, as well as economic benefits as a result of reduced use of health and welfare services (Douglas et al., 2016). Negative correlations between a sense of social belonging and mental (Hagerty, Lynch-Sauer, Patusky, Bouwsema & Collier, 1992; Hagerty, Williams, Coyne & Early, 1996) and physical health (Hale, Hannum & Espelage, 2005; Mushtaq, Shoib, Shah & Mushtaq, 2014) have been reported in the literature, making the post-TBI person, and their families, vulnerable to further mental and physical health problems. The current literature may underestimate the long-term socioemotional effects of TBI. One possible limitation of TBI longitudinal studies is that severely depressed, anxious, disinhibited or cognitively impaired individuals are unlikely to participate.

Despite the high prevalence of social cognition deficits after TBI and the negative interaction between social cognition and functional outcome post-TBI, the underlying mechanisms of these impairments are not well understood. Several theories have intended to explain social problems post-TBI, including; impaired executive functioning (Bechara & Van Der Linden, 2005), self-regulatory behaviour problems (Ganesalingam, Sanson, Anderson & Yeates, 2007), and self-appraisal deficits (Kervick & Kaemingk, 2005). One prominent hypothesis proposes that aberrant facial affect recognition may underpin abnormalities in social cognition after TBI.

Biszak and Babbage (2014) reported that 51% of 45 individuals referred to TBI rehabilitation programmes displayed clinically significant impairments in facial affect recognition. A meta-analysis of 13 studies (296 individuals with TBI) conducted by Babbage et al., (2011) provided evidence that individuals with TBI were significantly poorer at identifying facial expressions compared to control participants, and that the severity of emotion perception deficits was variable. The analysis estimated that up to 39% of individuals with a moderate-severe TBI could present with significant facial affect identification impairments. Babbage and colleagues

(2011) meta-analysis provided an informative overview of facial affect recognition after TBI, addressing the frequent criticism that TBI studies usually only include small sample sizes, as well as highlighting the magnitude of an often untreated sequela of TBI. However, the authors restricted their review to static facial affect due to the paucity of available dynamic methodologies and, therefore, estimations are based on partial data and not entirely representative of general facial affect impairments post-TBI. Regardless of specific percentages, there is a consensus that facial affect recognition impairments are often evident post-TBI. Furthermore, they appear to be associated with other debilitating post-TBI deficits such as alexithymia and lower levels of cognitive empathy (Neumann et al., 2016) which, in turn, weaken social attachments and interpersonal relationships (Grynberg et al., 2012; Williams, Wood & Howe, 2019).

Other researchers have investigated the effects that emotion perception impairments can have on social functioning. In early work, Hornak et al., (1996) explored a putative correlation between facial affect recognition and social behaviour. The authors established that individuals affected by TBI, particularly those with ventral frontal lesions, were poor at identifying facial expressions. Additionally, individuals with TBI and their relatives reported a rise in inappropriate behaviour, apathy, withdrawal and a reduction in communication post-injury. Hornak et al., (1996) reported a significant negative correlation between facial affect recognition and subjective emotional experience, for example, an inability or reduction in the ability to experience emotions. The researchers suggested that impairments in facial affect recognition may be part of a wider disturbance of emotional experience. This theory was supported by Croker and McDonald (2005) who also reported significant correlations between facial affect recognition and subjective emotional experience post-TBI. Facial affect recognition research, including Hornak et al., (1996), and Croker and McDonald (2005) has tended to use forced-choice paradigms with isolated static faces which do not capture the

naturalistic emotion perception of everyday life (Turkstra et al., 2017). Similarly, empirical research may underestimate the severity of facial affect identification impairments post-TBI as matching and labelling tasks arguably provide cues and prompts. However, it is hard to strike a balance between the confounding variables of providing emotional labels and controlling for verbal expression impairments during TBI research. Contemporary research needs to develop improved tools and methodologies which capture social impairments in real-world settings (Sasson, Pinkham, Weittenhiller, Faso & Simpson, 2015).

In contrast to Hornak et al.'s, (1996) findings, Milders, Fuchs and Crawford (2003) reported that TBI participants scored significantly lower on naming and matching facial expressions compared to controls. However, there was no correlation between facial affect recognition and changes in emotional and social behaviour. Milders et al., (2003) proposed that their findings do not support the assumption that facial affect recognition impairments are related to poor social behaviour. This view is disputable as an inability to perceive emotions could logically lead to abnormal social behaviour as emotion perception is essential for the engagement of successful social functioning (Phillips et al., 2003) and maintenance of relationships (Clark, Von Culin, Clark-Polner & Lemay, 2017). Osborne-Crowley and McDonald (2018) proposed that, if an individual with TBI is unable to identify the emotion of a person they are interacting with, then they will be unable to experience 'social punishment' which usually curtails inappropriate behaviour. A lack of significant correlation in the Milders et al., (2003) study may be related to the design of their methodology, (e.g. lengthy questionnaires-219 questions), or stringent statistical adjustments (e.g. adjusting the  $\alpha$  level for multiple comparisons).

Spikman et al., (2013) implemented a self-report and proxy version of the DEX to measure behavioural problems after TBI. Although the DEX is widely used in research, the inter-rater reliability of the test has been questioned (Barker, Morton, Morrison & McGuire, 2011;

McGuire et al, 2014). Both the self-report and proxy DEX scores indicated that TBI participants had significantly more behavioural problems compared to controls. Furthermore, proxy ratings for the DEX tended to be higher than the self-report scores, suggesting that TBI participants underestimated their behavioural problems, a condition known as anosognosia or lack of insight. The extent of anosognosia in the group affected by TBI was related to the severity of facial affect recognition deficits. A further negative relationship was reported between deficits in facial affect recognition and behavioural changes reported by significant others. The authors proposed that impairments in aspects of social cognition, particularly facial affect recognition, may underlie, at least in part, behavioural disturbances after TBI. Spikman et al., (2013) postulated that discrepancies in the extant literature may stem from variation in participant samples. For instance, Milders et al., (2003) research included participants who were one-year post-injury while Spikman et al.'s., (2013) participants were, on average, more than six years post-injury. In early work, McKinlay, Brooks, Bond, Martinage & Marshall (1981) reported that adverse psychosocial changes increased after the first 12 months post-injury. These psychosocial changes probably became more apparent after 12 months, possibly because this is around the time that medical and rehabilitation support decreases and the individual with TBI are expected to resume daily activities (e.g. returning to work) and reintegrate into society (e.g. participate in social activities such as hobbies or meeting friends). It may be the case that Spikman et al.'s (2013) self-report and proxy ratings indexed chronic problems while Milder's et al.'s (2003) research failed to capture enduring deficits. Guidelines for distinguishing between acute and chronic impairments are lacking and there are inconsistencies between studies regarding the length of time post-TBI and recruitment into research. For instance, some studies have recruited individuals as early as three months post-TBI (Neumann, McDonlad, West, Keiski & Wang, 2016) while others only included individuals at least one-year post-TBI (McDonald, Flanagan, Martin & Saunders, 2004).

Therefore, caution must be taken when interpreting results in regards to whether temporary acute effects or long-term intractable deficits were being measured.

Nevertheless, facial affect recognition impairments have been evidenced during the acute phase of TBI and appear to continue into the chronic phase. Ietswaart, Milders, Crawford, Currie & Scott (2008) reported that TBI participants had significantly lower accuracy scores on a facial affect recognition task compared to controls, both shortly after their injury (mean of 2.1 months) and one-year post-injury. They also reported that individuals with TBI had slower reaction times during the task, a finding which has been supported by subsequent research (Celeghin, Galetto, Tamietto & Zettin, 2019). The group affected by TBI emotion identification scores had increased slightly after one year, for example, labelling expressions rose from 73 % shortly after injury to 79.8 % at the one-year follow-up. Correlational analyses revealed that injury severity, measured by the GCS and PTA, negatively correlated with affect recognition scores. Importantly, Ietswaart et al., (2008) showed that 63% of the TBI cohort had mild or moderate injuries but still displayed significant facial affect recognition impairments. Mild TBI is often underrepresented in the literature, even though it is associated with long-term complaints including a decline in social cognition (van der Horn, Liemburg, Aleman, Spikman, van der Naalt, 2016).

Similarly, Green, Turner and Thompson (2004) employed a longitudinal design to assess acute (two months post-injury) and chronic (one-year post-injury) emotion perception impairments in a group of 30 TBI participants. TBI participants had differing injury severities and were divided into sub-groups consisting of; (i), individuals who had sustained right posterior focal lesions, including pathology to the basal ganglia and the amygdala, both of which have been implicated in facial affect recognition (+RPF;  $n=16$ ) and (ii), individuals who exhibited no right posterior focal lesions (-RPF;  $n= 6$ ). Overall, the combined group with TBI were less accurate

on a facial affect identification task compared to controls at both time points. Importantly, there was no difference between the TBI and control groups on a non-affect face perception task, implying that TBI generates impairments in the ability to make fine-grained face analysis (e.g. processing facial expressions and subtle facial movements) compared to coarse-grained judgements of facial features (e.g. identifying sex). Further analysis revealed that both subgroups were impaired on the emotion perception task compared to the control group. Green et al., (2004) proposed that the dramatic shearing forces associated with DAI could be the underlying mechanism of poor facial expression recognition post-TBI, compared to focal lesions to brain regions frequently implicated in emotion perception. This theory is supported by subsequent research from Philippi, Mehta, Grabowski, Adolphs and Rudrauf (2009) and Genova et al., (2015) who reported an interaction between reduced white and grey matter integrity in relation to DAI and emotion perception deficits post-TBI.

Advancements in neuroscientific techniques are helping to elucidate the relationship between network functionality and emotion perception deficits post-TBI. McDonald, Dalton, Rushby and Landin-Romero (2018) used diffusion tensor imaging (DTI; a technique which measures water diffusion at specific points along white matter tracts) to measure fractional anisotropy (FA; indexes the variability of diffusion in different directions with high values indicating intact white matter and low values indicating abnormalities) and mean diffusivity (MD; a scalar measure of the total diffusion within a brain voxel) using tract-based spatial statistics (TBSS; measures white matter of the whole brain). McDonald et al., (2018) explored the white matter integrity in 17 adults with severe TBI. Compared to a matched control group, individuals affected by TBI displayed abnormal FA and MD in numerous tracts including the thalamus, external capsule and cerebellum tracts, all which are necessary for multimodal processing. There were also abnormalities in the orbitofrontal, frontopolar and right temporal cortices which have strong associations with social cognition Amodio & Frith, 2006; Bzdok et al., 2013;

Forbes & Grafman, 2010; Wood, 2003. There was also a significant correlation between decreased FA in widespread structures and The Assessment of Social Inference Task (TASIT; McDonald et al., 2003) which provided a systematic examination of social perception. McDonald et al.'s (2019) findings highlight that more research is warranted to understand the effects of DAI.

## **2.9. The nature and extent of emotion identification impairments post-TBI**

The previous section outlined why social cognition deficits are important to explore post-TBI. For example, it is generally accepted that TBI is a risk factor for negative social outcomes across the adult lifespan, including poor social integration (Knox & Douglas, 2009), loss of close friendships/marital breakdowns (Bodley-Scott & Riley, 2015), and loss of occupation (Humphrey et al., 2013). Previous outcome studies in TBI have focused on cognitive functions, such as information processing, memory and attention impairments, as predictors for poor social reintegration, but findings have been limited (Spikman et al., 2011). The focus on cognitive deficits after TBI is more than likely related to the fact that cognitive sequelae can be objectively and reliably measured through neuropsychological assessments, while there is a paucity of suitable social cognitive assessments (Spikman et al., 2012). Contemporary research has started to explore the relationships between general cognitive deficits, social cognition impairments and social outcomes.

Rosenberg et al., (2018) reported that in a group of 32 individuals affected by TBI emotion recognition impairments could not be accounted for by deficits in working memory or processing speed, although, the authors did acknowledge that they employed a short battery of tests to assess executive function which may not have tapped into the necessary difficulties which equate to poor social functioning post-TBI. Interestingly, Rosenberg and colleagues did hypothesise that subtle cognitive impairments, such as aberrant visual strategies, may have



impacted on social performance, although this was not tested. However, the authors did find that more severe injuries did contribute somewhat to emotion identification impairments. These results mirror previous work by the research team which had also documented that severity of the injury was a solitary predictor of performance on an emotion perception task (Rosenberg et al., 2015; Rosenberg et al., 2014). Although previous work has indicated that more severe TBI is related to more severe social cognition deficits (Spikman et al., 2012), some studies have found that controlling for injury severity does not account for the correlation between social cognition and post-injury behaviour (De Sousa et al., 2012; Knox and Douglas, 2009; May et al., 2017). Consequently, it is improbable that injury severity could fully account for the association between social cognition and social behaviour, but it is likely a contributing factor.

As general cognitive impairments are common after TBI, an important research question is to what extent do these account for social cognition deficits post-TBI? Spikman et al., (2012) explored social cognition impairments in relation to general cognitive impairments (memory, mental speed, attention, executive function) in individuals with moderate to severe TBI. The authors reported that the individuals with TBI scored significantly lower on general cognitive assessments (mental speed, attention, memory and executive function) compared to matched controls. However, the authors reported that lower performance on the social cognition tasks (emotion recognition, ToM, empathy) was not related to impairments on the general cognitive assessments. At present, it is generally accepted that the interaction between general cognitive impairments and social outcome post-TBI is weak (Ownsworth & McKenna, 2004; Wood & Rutterford, 2006), and it is more likely that social cognition impairments (e.g. social cue perception, empathy and ToM) are related to social behaviour and outcome. Unfortunately, correlational analyses do not provide details of the direction of the relationship, so it is unknown whether (1) impairments in social cognition lead to poor social functioning or (2)

post-TBI social behaviour changes are a result of a reduction of social interactions over time and, therefore, the individual with TBI had fewer opportunities to utilise social skills (3) it is a combination of both. However, the second theory is unlikely as research has indicated that post-TBI social impairments remain stable over time. Indeed, the literature documents that social cognition impairments post-TBI are persistent, they are present in the acute phase of the injury and extend into the chronic stages (at least one-year post-injury) of the condition (Blair & Cipolotti, 2000; Hoofien, Gilboa, Vakil, & Donovick, 2001; Ietswaart, Milders, Crawford, Currie & Scott, 2008; Tranel, Bechara & Denburg, 2002). However, that does not mean that other factors, such as time since injury, are not contributing to intractable social cognition deficits post-TBI.

This chronicity of social impairments post-TBI is apparent across the lifespan with extant evidence suggesting that emotion identification impairments occur in both younger and older adults. However, there is discord regarding whether age might mediate aspects of emotion recognition abilities following TBI. The literature suggests that older adults may be less accurate at identifying facial expressions compared to younger adults (Gonçalves et al., 2018; Ruffman et al., 2008). However, Byom, Duff, Mutlu and Turkstra (2019) found that their combined TBI sample (older and younger participants) were less accurate on both subtle and intense facial expression recognition conditions compared to a control group, but there was no relationship between age and intensity level. This is in line with findings from Rigon, Turkstra, Mutlu and Duff (2016) who also found no significant effect of age on an emotion recognition task post-TBI. Rigon et al., (2016) also investigated sex as a possible protective factor against emotion perception deficits following TBI. As it is predicted that up to 39% of individuals who have sustained a TBI experience impairments in emotion recognition (Babbage et al., 2011), uncovering protective or risk factors would be a clinically significant finding. Rigon et al., (2016) reported that in a group of individuals with TBI, females, compared to males, were

significantly better at identifying dynamic, but not static, emotions. Furthermore, males, but not females, performed significantly poorer compared to control participants on the dynamic task. This sex difference could not be accounted for by pathology location, injury severity or other neuropsychological variables. Zupan, Babbage, Neumann and Willer (2017) explored whether males and females with TBI differed in their emotion recognition and emotional inferencing abilities. The research team also investigated whether differences were related to target emotion or high and low-intensity facial and vocal emotion expressions. The female individuals with TBI were significantly more accurate during the vocal expression and emotional inferencing tasks compared to males. Interestingly, females were also better at identifying fearful facial expressions and also facial expressions in the high-intensity stimuli set. The author's proposed that the purported female advantage, often found in emotion perception studies with non-TBI participants, is at least partially maintained following a TBI (Collignon et al., 2010; Hall & Matsumoto, 2004). They also acknowledged that their findings may have differed from Rigon et al., (2016) because their study only employed static stimuli while Rigon and colleagues also employed dynamic stimuli. A pertinent finding from Zupan et al., (2017) is that 32% of their female participants with TBI exhibited impairments in facial expression recognition while only 19% displayed vocal affect recognition deficits. This pattern was not expected as it is typically thought that vocal cues are harder to decipher (Johnstone & Scherer, 2000). This is interesting as it could suggest that emotion perception deficits post-TBI may be related to a specific abnormality in visually scanning faces, compared to a general inability to process all emotional cues. However, this effect was only apparent in females, with a similar proportion of males being impaired on faces (42%) and voices (41%). Babbage, Zupan, Neumann & Willer (2018) reported a significant sex treatment effect for a 'Stories Intervention' (a programme which taught individuals living with TBI to infer emotions based on the contextual information that was provided during a short story). The authors reported that

the group of women affected by TBI significantly improved during the intervention, exhibiting improved emotion perception, compared to males with TBI. Contrasting findings were reported by Zupan, Neumann, Babbage and Willer (2017) who found that there were no significant sex differences in general emotion perception, although the authors acknowledged that this may have been due to the prolonged stimulus exposure of their task. Interestingly, females were more accurate than males at recognising fearful faces while there were no differences for happy or angry stimuli.

Sex differences on one measure do not exclude the possibility of sex differences on another measure and vice-versa. For instance, Zupan, Babbage, Neumann and Willer (2018) investigated sex differences in affective and cognitive empathy post-TBI. The study required participants to complete a self-report affective and cognitive empathy questionnaire and the scores were then compared against normative data. Pre-existing normative data has indicated that females have higher levels of both affective and cognitive empathy than men. The females in the study rated themselves significantly lower than the normative means on both measures whereas males only rated themselves as lower on the cognitive empathy measures. Further analysis indicated that significantly more females with TBI exhibited deficits in affective empathy compared to male participants, suggesting that diminished empathy levels, particularly affective empathy, may be more marked for females post-TBI compared to males. These results contrast with Wood and Willams (2008) who reported that a group of males with TBI were more impaired than females on an affective empathy task. Again, these discrepancies may be related to differences in methodologies or cohort demographics. Interestingly, Zupan et al., (2018) reported that there were no statistical differences between males and females for self-reported and proxy levels for empathetic concern or perspective-taking. This finding suggested that sex differences in empathy levels following TBI are minimal, but females may experience more relative difficulties compared to males for affective empathy. Zupan and

colleagues' results show that females do experience social cognition impairments post-TBI despite the underlying assumption that females are less vulnerable to socioemotional changes following TBI (Rigo et al., 2016). With the variability of stimuli sets used within the TBI literature, researchers need to continue to explore sex differences, and other possible protective factors post-TBI, on both pre-existing and new social cognition assessments. It is unlikely that age and sex are driving factors for social cognition deficits post-TBI. However, it may be the case that they may mediate some aspects of the impairments, but the evidence base is univocal and needs extending.

There is a trend in the literature documenting that individuals affected by TBI may exhibit an 'emotion-specific' deficit. Extant evidence has indicated that negative emotions are particularly impaired post-TBI (Green et al., 2004; Ietswaart et al., 2008; McDonald et al., 2003; Spikman et al., 2013). For example, Croker and McDonald (2005) reported that both individuals with and without TBI scored higher when identifying positive (happy, surprised) compared to negative (sad, fearful, angry) facial expressions. However, individuals with TBI scored lower overall for both positive and negative emotions. The reason for this effect is debated, one hypothesis is that the frontal brain regions associated with the processing of negative emotional stimuli are vulnerable to damage during TBI (Adolphs, 2001; McDonald et al., 2013). However, given the heterogeneous nature of TBI and the finding that other clinical cohorts (e.g. schizophrenic and autistic populations), and control groups also score lower when deciphering negative compared to positive emotions (Teh, Yap & Liow, 2018), it seems unlikely that anatomical damage can sufficiently account for the valence effect. A parsimonious explanation for the valence effect is the frequent imbalance between positive and negative emotions in most studies, typically with four negative possibilities (anger, disgust, sadness and fear) and only two positive options (happy and surprise, although surprise is sometimes not considered a true representation of positive emotion and can be either positive

or negative - Kreibig, 2010). High scores on positive affect measures may change if research included alternative, non-basic positive emotions such as amusement or excitement. Indeed, this was the case in the Rosenberg et al., (2019) study, as they reported a decrease in control participant accuracy identifying dynamic happy faces from 85.42% to 41% when the group were provided with multiple positive labels. Recent research has highlighted the importance of studying emotion perception in contexts that more clearly represent real-life perception of emotional displays, including moving away from forced-choice paradigms (Turkstra et al., 2017). Accordingly, disputes about valence-based differences in emotion identification within the TBI literature are confounded by methodological limitations, with floor and ceiling effects often being reported for fearful and happy expressions respectively (Rosenberg, Dethier, Kessels, Westbrook, & McDonald, 2015; Rosenberg, McDonald, Dethier, Kessels, & Westbrook, 2014).

The social cognition research field is rapidly developing and, therefore, innovative tools to assess emotion perception are being created. The complex audio-visual emotion assessment task (CAVEAT; Rosenberg, McDonald, Rosenberg & Westbrook, 2019) incorporates non-posed, audio-visual aspects of social interactions and includes subtle emotions such as pleasant and unpleasant surprise. The task is thought to eliminate floor effects often associated with fear conditions by directing actors to intensely display fear as if they were terrified, although this may decrease the ecological validity of the assessment as fear varies in intensity during real world interactions. The assessment was also designed to reduce the ceiling effects of happiness by presenting a list of 11 positive emotions for participants to choose from. Rosenberg et al., (2016) assessed 32 moderate-severe TBI participants and matched controls on the CAVEAT. Compared to the control group, the TBI group displayed poor emotion identification across all emotions. This finding is of significance as it is frequently reported that both TBI and control groups find it more difficult to identify negative compared to positive emotions (Crocker &

McDonald, 2005; Green et al., 2004; Ietswaart et al., 2008; McDonald et al., 2003; Spikman et al., 2013), although findings are mixed (Rosenberg et al., 2014). To further this work, Rosenberg et al., (2018) explored emotion identification of 22 different emotions (11 positive and 11 negative emotions) in a group of individuals affected by TBI and matched controls. The study also investigated basic (innate emotions from birth e.g. fear) versus complex social emotions (learned through cultural interactions and social ‘display rules’, involving mentalistic construction of emotions as internal states e.g. embarrassment). The researchers reported that the group with TBI exhibited a general deficit in emotion recognition, rather than displaying a selective deficit in recognising negative compared to positive emotions or basic versus complex emotions. This finding is in line with previous research (e.g. Ietswaart et al., 2008; Rosenberg et al., 2014).

Emotion recognition is not only mediated by emotion valence but also by expression intensity (Spell & Frank, 2000; Rigon et al., 2016). Rosenberg et al., (2014) and Rigon et al., (2016) reported that individuals with TBI scored significantly lower than controls on both low and high-intensity emotion recognition tasks. However, Spell and Frank (2000) reported that their group of individuals with TBI only displayed deficits in recognising low-intensity emotions, with no group differences between the TBI and control group on high-intensity expressions. Indeed, individuals with TBI appeared to be poorer at recognising subtle compared to intense emotions, even during happiness conditions. However, findings from Rosenberg et al., (2014) highlighted that this interaction is complex as they reported that, for some emotions (fear, surprise), the group TBI only displayed deficits on low-intensity expressions while for other emotions (anger, disgust) recognition was impaired regardless of intensity.

Emotion perception impairments post-TBI are apparent regardless of the stimuli mode. That is, individuals affected by TBI frequently display impairments on both static and dynamic emotion

perception tasks (Babbage et al., 2011; McDonald et al., 2003; Rosenberg et al., 2019). Recently, the use of static stimuli has been criticized on the grounds of poor ecological validity and mundane realism (Alves, 2013), with many researchers now attempting to employ dynamic stimuli in their research designs. The processing of static and dynamic stimuli are likely to depend upon different neurological underpinnings (Trautmann-Lengsfeld, Dominguez-Borrás, Escera, Herrmann & Fehr, 2013; Zinchenko, Yaple & Arsalidou, 2018), and may induce different underlying physiological responses (Blais, Fiset, Roy, Saumure-Régimbald & Gosselin, 2017; Rymarczyk, Żurawski, Jankowiak-Siuda & Szatkowska, 2016). Furthermore, static stimuli often eliminate the contextual effects of social interactions. Social context influences our mental and behavioural processes and plays a major role in social cognition (Baez, García & Ibanez, 2018). Although the exploration of the relationship between context effects and social cognition is still in its infancy, the existing literature suggests that variables such as; faces within a natural scene, tone of voice, body language, cultural orientation, and the nature of the relationship (e.g. romantic/strangers) shape how emotions are perceived (Barrett, Mesquita & Gendron, 2011; Ibañez & Manes, 2012; Turkstra et al., 2017). Importantly, Croker and McDonald (2005) and Zupan et al., (2014) reported that participants with TBI improved on a label and matching facial expression task when they were provided with context compared to when no context was provided. McDonald and Saunders (2005) assessed emotion recognition in individuals with and without TBI using four types of stimuli (static images, video only, audio-only, and audiovisual media). Individuals with TBI scored significantly lower on the audio-only and the audiovisual media conditions compared to non-TBI individuals. A deeper exploration of the data revealed that eight of the individuals with TBI were significantly impaired (two standard deviations below the control group mean) on the static images, in contrast to one participant on the video only condition. This led the authors to conclude that audiovisual media is easier for individuals with TBI to decipher, probably due to the additional



contextual cues. However, Williams and Wood (2009) pointed out that this conclusion appears counterintuitive as the audiovisual condition would involve processing semantic and affective content of the conversation as well as visual cues, constituting to dual processing, an ability which is well known to be impaired post-TBI (Azouvi et al., 2004).

Williams and Wood (2009) reported that a group of 64 individuals affected by moderate-severe TBI performed significantly poorer compared to a control group on a static and dynamic emotion recognition task. In line with similar research, the group with TBI exhibited significantly greater difficulty during the static compared to the dynamic task, suggesting that the media of stimuli plays an integral role in emotion perception (Hornak et al., (1996); Humphrey, Donnelly & Riddoch, 1993; McDonald & Saunders, 2005). These results also support the hypothesis that static and dynamic displays of emotion may draw upon dispersed and dissociated neural networks (Adolphs, 2003). This theory was also supported by Wood and William's (2009) finding that information processing, measured through a variety of neuropsychological measures, was not associated with emotion recognition ability. Although there was a significant correlation between vocabulary and performance on the static facial expression recognition task, regression analysis indicated that verbal ability only accounted for 8.7 % of the variance on the static task. The facial movement during the dynamic task may have aided emotion identification, regardless of the greater demands which would have been placed on the information processing system. In line with an earlier discussion point, the group with TBI in the Wood and Williams study also exhibited impairments when recognising negative versus positive emotions, suggesting that emotion valence may influence emotion perception performance. The authors did point out that there were a greater number of negative emotions on both the static and dynamic tasks, that negative emotions share similar features, and both the TBI and control groups found negative emotions harder to identify during the static task. Consequently, these preceding factors may account for the valence findings rather

than there being a true specific deficit in negative emotion perception post-TBI. A further finding was that both groups performed poorly on the neutral items of the dynamic task, but the group with TBI were significantly poorer than controls. The authors proposed that group differences could be related to a wider emotion dysfunction, rather than an impairment in specific emotions, suggesting that TBI induces a global emotion recognition impairment, regardless of the nature of the assessment.

To address the criticisms of static stimuli, researchers have developed dynamic emotion perception assessments. For example, McDonald et al., (2003) developed TASIT, a dynamic audio-visual assessment of social perception. The authors reported that 12 TBI participants scored significantly lower on the test compared to matched controls, with specific difficulties interpreting neutral, fear and disgust items. These results were mirrored by Knox and Douglas (2009) who measured the ability of individuals with severe TBI to match facial expressions to social situations using static photographs (JACFEE; Matsumoto & Ekman, 1988) and TASIT (McDonald et al., 2003). Knox and Douglas (2009) reported that the group with TBI performed significantly lower compared to matched controls on both the static and dynamic tasks. The control group scored similarly across both tasks but the group with TBI scored significantly lower on the dynamic task, suggesting that static images were easier to decode. Knox and Douglas (2009) theorised that inappropriate behaviour after TBI may stem from a misinterpretation or a failure to understand how another person is feeling, rather than disinhibited responses. Knox and Douglas (2009) observed that control participants mirrored facial expressions of the photographs and actors in TASIT and added context-specific spontaneous scripts to the muted video clips, this appeared to aid emotion detection. Only one out of 13 TBI participants appeared to display similar behaviour. Knox and Douglas (2009) did not provide further comments on this case but it would have been interesting to explore the

nature and location of pathology to determine if this may have accounted for the spared mirroring ability.

In summary, impairments in emotion recognition have been found in groups of individuals affected by TBI, irrespective of cognitive deficits, time since injury, age, sex, valence, intensity and mode (e.g. dynamic or static) of the emotions. This suggests that the nature of emotion recognition deficits post-TBI are significant and long-term and the extent of the deficits do not appear to be wholly related to cognitive changes, individual characteristics or stimuli presentation. For that reason, other factors must mediate the persistent emotion identification impairments frequently reported in the TBI literature. One possibility is that TBI leads to aberrant visual strategies which are insufficient during the initial stages of social cognition. These abnormal gaze patterns could mean that individuals with TBI miss the vital social cues necessary to decipher another person's mood state.

## **2.10. Multifaceted components of social cognition post-TBI**

To understand the nature of emotion recognition impairments post-TBI, it is important to understand these in context with other social cognition components which work simultaneously and in synergy with emotion recognition. Wearne, Osborne-Crowley, Rosenberg, Dethier and McDonald (2019) examined the relationship between affective expressivity, the subjective experience of emotion, and emotional face recognition in both individuals with and without TBI. The authors wanted to test the unique contribution of these factors after controlling for demographic and cognitive variables related to emotion recognition. They found that subjective emotional experience significantly predicted the ability to recognize facial expressions. This is in line with previous research by Croker and McDonald (2005) who found that poor matching of facial expressions was related to a reduction in the experience of sadness and fear in a group of individuals who had sustained TBI. Hornak et al., (1996) also discovered that a group of

individuals affected by TBI who were poor at identifying mood states, as identified by nursing staff, were also poor on a test of emotion recognition. These combined findings support simulation models of emotion recognition which theorise that the ability to identify and understand emotions in conspecifics is reliant on individuals simulating a similar emotional state in themselves as a means to understand it (Niedenthal, Mermillod, Maringer & Hess, 2010). Consequently, individuals with TBI may find it challenging to simulate the emotions of others or to attribute the meaning to the experience they feel, linking tightly with theoretical models of alexithymia (the inability to identify, process or describe personal feelings; Williams, Wood & Howe, 2019; Wood & Williams, 2012; Wood, Williams & Kalyani, 2009). It should be noted that alexithymia is a broad term and it is not clear whether individuals with TBI have difficulty experiencing emotions or whether they are incapable of assigning meaning or salience to these emotions. Individuals with TBI may also find it difficult to utilise emotional empathy with research reporting significantly higher frequencies of alexithymia and lower levels of emotional empathy compared to control groups (Williams & Wood, 2010; Wood & Williams, 2008). Therefore, individuals with TBI may have generalized difficulty in decoding emotional stimuli and experiences, which include emotion perception deficits. If this is the case, post-TBI interventions could target emotional experience as opposed to the perceptual analysis of facial expressions.

Other abilities which contribute to effective social behaviour are ToM, that is, the ability to understand and infer other people's thoughts, intentions and beliefs (Premack & Woodruff, 1978) and mentalizing, that is, the reflection of affective mental states (Wyl, 2014). It should be noted that although some authors (e.g. Wyl, 2014) acknowledge a distinction between ToM and mentalizing, others do not and the terms are often used interchangeably in the literature. First-order ToM mental states include thoughts such as 'he thinks' while second-order mental states include thoughts such as 'he thinks she thinks' (Coull, Leekam & Bennett, 2006). Both

ToM and mentalizing deficits are often reported in the TBI literature (Bibby & McDonald, 2005; Havet-Thomassin, Allain, Etcharry-Bouyx & Le Gall, 2006; McDonald & Flanagan, 2004), and research has suggested a relationship between deficits in ToM, mentalizing and maladaptive social and emotional behaviour post-TBI (Milders et al., 2003).

In a recent review by Bivona et al., (2018), the authors outlined the two main theoretical models explaining ToM. *Theory Theory* suggests that humans infer other people's intentions and beliefs by deploying general principles, such as cultural rules and laws, with the involvement of executive functions, abstract reasoning and working memory (Carruthers & Smith, 1996). On the other hand, *Simulation Theory* suggests that autobiographical experiences allow an individual to 'put themselves in someone else's shoes', remodelling the other person's experience to make predictions about their behaviour. The *Simulation Model* is largely based on the discovery of mirror neurons which have briefly been discussed in the preceding chapter. To support the *Theory Theory*, research has documented a correlation between working memory, processing speed, inhibition and ToM (Bibby & McDonald, 2005) while others have reported no interactions (Tranel, Bechara & Denburg, 2002). The *Simulation Theory* also has support from studies reporting associations between low self-awareness, emotional disturbances and low ToM scores (Bivona et al., 2014; de Sousa, McDonald, Rushby, Dimoska & James, 2010). Bivona et al., (2018) concluded that the two models are more than likely not mutually exclusive in explaining ToM and mentalizing impairments post-TBI and more research is needed in the area to fully understand these univocal findings.

Executive functioning, particularly impairments inflexible response selection, could also affect the social outcome of individuals who have sustained TBI. Indeed, inflexibility would interfere with Penn et al's (1997) third stage of social cognition, response selection, as individuals would be unable to adjust their social behaviour in response to dynamic environments (Milders et al.,

2008). Vilkki et al., (1994) and Nybo et al., (2004) demonstrated that there was a relationship between cognitive flexibility and return to work/level of social activity. Milders et al., (2003) investigated emotion recognition, ToM and cognitive flexibility and related test performance to self and proxy scores of post-injury behaviour in a group of individuals affected by TBI shortly after injury and one-year post-injury. Compared to orthopaedic control participants, individuals with TBI exhibited impairments on an emotion identification, ToM and cognitive flexibility task both soon after injury and one-year post-injury. The proxy ratings of the group with TBI demonstrated an increase in behavioural problems one year following TBI but there was no significant relationship between test performance, both shortly after and one-year post-injury, and poor social behaviour. Conflicting results between studies such as Vilkki et al., (1994), Nybo et al., (2004), Milders et al., (2003) and Milders et al., (2008) have highlighted the challenges of measuring executive functioning, particularly the features relevant to social cognition outcome post-TBI. Another factor to consider is that measurements of behaviour changes post-TBI are complex as individuals affected can provide inaccurate reports of their functioning due to lack of insight (Spikman et al., 2013). However, focusing on proxy data disempowers the individual affected by TBI and raises issues such as time of testing (e.g. family members or care staff need time to develop an accurate perspective of the new behaviours; Bennett, Ong and Ponsford, 2005). Interestingly, Milders et al., (2008) proposed that their findings presented little reason to conclude that deficits in emotion recognition or ToM were associated with changes in social behaviour post-TBI. Knox and Douglas (2009) and Struchen et al., (2008) both found a significant interaction between facial expression identification and social integration, thus supporting the assumption that poor social reintegration following TBI is more related to poor emotion perception rather than executive function impairments. However, it should be noted that although extant evidence suggests that executive dysfunction is not the main driving force behind poor social reintegration post-TBI,

and emotion perception may play a leading role, the identification of facial expressions will draw upon executive function abilities and impairments in this domain will have an impact on social cognition. Spikman et al., (2013) proposed that poor social behaviour might pertain to passivity or apathy associated with fatigue or disorganised behaviour, both of which can be related to executive dysfunction.

While social outcomes after TBI are influenced by many factors (injury severity, pathology location, pre-morbid personality, economic status; Ponsford, 2013), evidence suggests that social cognitive abilities are key contributors, particularly the ability to identify facial expressions. Further nuanced research is needed to understand the nature and extent of emotion identification impairments post-TBI, particularly the deleterious effect it has on functional outcomes. The proportion of variability in aberrant behaviour explained by a specific impairment (e.g. emotion perception, executive function) will more than likely be small if the tools employed to measure post-TBI behaviour include a wide range of behaviours (e.g. the DEX). The implication for these findings is that to better understand the impairments underpinning post-TBI changes in social behaviour other mechanisms need to be explored, such as initial visual strategies to social stimuli.

As detailed earlier in the chapter, measuring social cognition is a complicated task as the ability taps into numerous cognitive, social and emotional functions. It is impossible to fully separate emotion perception from other social abilities, such as ToM, empathy and simulation, as well as neuropsychological functions. However, it is nearly almost impossible to study all of these factors in-depth during a single study. Therefore, many researchers usually choose one area of interest to focus on. Based on evidence reporting that emotion perception impairments post-TBI are consistent and persistent, as well as findings that effect sizes were more substantial and larger compared to effect sizes in ToM and empathy studies (Milders, 2018; Spikman et

al., 2012), this thesis' main focus was on emotion perception. Furthermore, although emotion perception deficits post-TBI are well documented, they are poorly understood. A gap in the current literature is that there is a paucity of research exploring low-level social cognition, particularly with regards to facial affect recognition after TBI, specifically in response to dynamic stimuli.

## **2.11. Summary**

In summary, this chapter has defined social cognition as the ability to perceive, encode, store, retrieve and regulate social information about self and others (Green et al., 2015). It is generally accepted that there are three stages to social cognition; the perception of social cues, interpretation and processing of social information and response selection (Adolphs, 2010; Ostrom, 1984; Penn et al., 1997). The 'social brain' is orchestrated by an extensive neural network which is heavily dependent on a frontal and limbic micro-circuitry (Amodio & Frith, 2006; Bicks et al., 2015; Wittmann et al., 2018; Xiao et al., 2017), and is influenced by social neuropeptides, particularly oxytocin and vasopressin. Research suggests that humans rely heavily on non-verbal visual cues (Beattie & Ellis, 2014), and the 'social brain' appears to have an innate preference for faces (Deaner & Platt, 2003). This preference is likely associated with the fact that facial expressions provide signals of social intentions, motivations and are communicative of internal emotional states (Hess & Hareli, 2015; Schmidt & Cohn, 2001). The processing of facial expressions relies on an extensive brain network, notably the frontal cortex and the motion detection regions of the parietal cortex (Adolphs, 2009; Phillips et al., 2003). Unfortunately, these areas are particularly vulnerable to damage during TBI due to movement of the brain in the skull and internal skull structures (Bigler, 2013). Pathology to these brain areas is frequently related to difficulty interpreting facial expressions and poor social functioning (Crocker & McDonald, 2005; Hornack et al., 1996; Tanaka & Sung, 2016).



The mechanisms underpinning these facial affect recognition impairments are poorly understood and are complicated by the complex interplay between classic cognitive deficits (e.g. memory and executive function), co-morbid effects (e.g. anxiety/depression and substance abuse) and pre-morbid personality traits. These confounding variables compound the research field and make it extremely challenging to disentangle the underlying mechanisms of facial affect recognition deficits post-TBI. To fully comprehend these impairments, researchers must explore both low-level and higher-level processes. Few studies have focused on low-level perceptual processes in regards to facial affect recognition after TBI, particularly in response to dynamic stimuli. Individuals *perceive* what they *attend* to, or fixate on, so crucially these data can provide objective indices of whether TBI and control cohorts differ in this respect when viewing naturalistic social stimuli. If there is variability in basic visual processing, then this might underpin upstream facial affect recognition impairments frequently reported after TBI. One method to investigate early social perception is through the implementation of eye-tracking technology which offers a non-invasive and quantitative assessment of social attention. However, the investigation of visual strategies, particularly in response to dynamic and naturalistic stimuli, post-TBI is scant. The next chapter will review typical visual processing and changes associated with TBI with a primary focus on facial affect identification.

## **3. Chapter three: Visual processing and visual attention after brain injury**

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### **3.1. Chapter overview**

This thesis investigated fixation patterns during social cognition task performance in TBI and control cohorts. Therefore, the present chapter will provide a brief overview of human visual processing. The neurological underpinnings and functionality of several eye movements are discussed.

Eye movements provide a powerful measure of the workings of the brain, with abnormalities within eye movement systems providing diagnostic clues and theoretical insight into disorders such as autism (Flack-Ytter et al., 2018), and schizophrenia (Benson et al., 2012). There is evidence that brain injury, even mild in nature, can damage neural network activation in brain areas associated with eye movements (Rockswold et al., 2019) and effect aspects of vision including; saccades, pursuit tracking, and convergence (the simultaneous inward movement of both eyes toward each other, typically to maintain single binocular vision; Ventura et al., 2014). Indeed, contemporary research, particularly in the field of sports science, is exploring visual scanning behaviour as a diagnostic biomarker of post-concussion syndrome (Chung et al., 2017; Childs, Barker, Gage & Loosemore, 2018; Maruta, Spielman, Rajashekar & Ghajar, 2018). Oculomotor testing offers a non-invasive and quantitative assessment method to investigate the neural underpinnings of brain networks often damaged during TBI, particularly frontal-striatal circuitry (Kraus, Little, Wojtowicz & Sweeney, 2010), which is strongly associated with social cognition (Bicks et al., 2015; Lee, Walker, Hale & Chen, 2017). Pathology and neurological disorders which disrupt the connectivity of the frontal lobes are associated with changes to eye scan patterns and social cognition (Klin, Jones, Schultz, Volkmar & Cohen, 2002), but the cause and effect of this relationship are unknown. This

chapter reviews the literature on the neurological underpinnings of human eye movements. This evaluation is not exhaustive due to space limitations and will, therefore, focus on the critical gaze-control networks including; the frontal eye fields (FEF), the supplementary eye fields (SEF), the dorsolateral prefrontal cortex (DLPFC) and the parietal eye fields (PEF) (Vernet, Quentin, Chanes, Mitsumasu & Valero-Cabr , 2014).

### 3.2. Human oculomotor and visual systems

Visual processing begins in the photoreceptor cells, specialised neurons of the retina (back part of the eye). These cells are responsible for visual phototransduction, the conversion of light into electrical signals. There are three known photoreceptor cells in the human eye; rods, cones and photosensitive retinal ganglion cells (Figure 19).

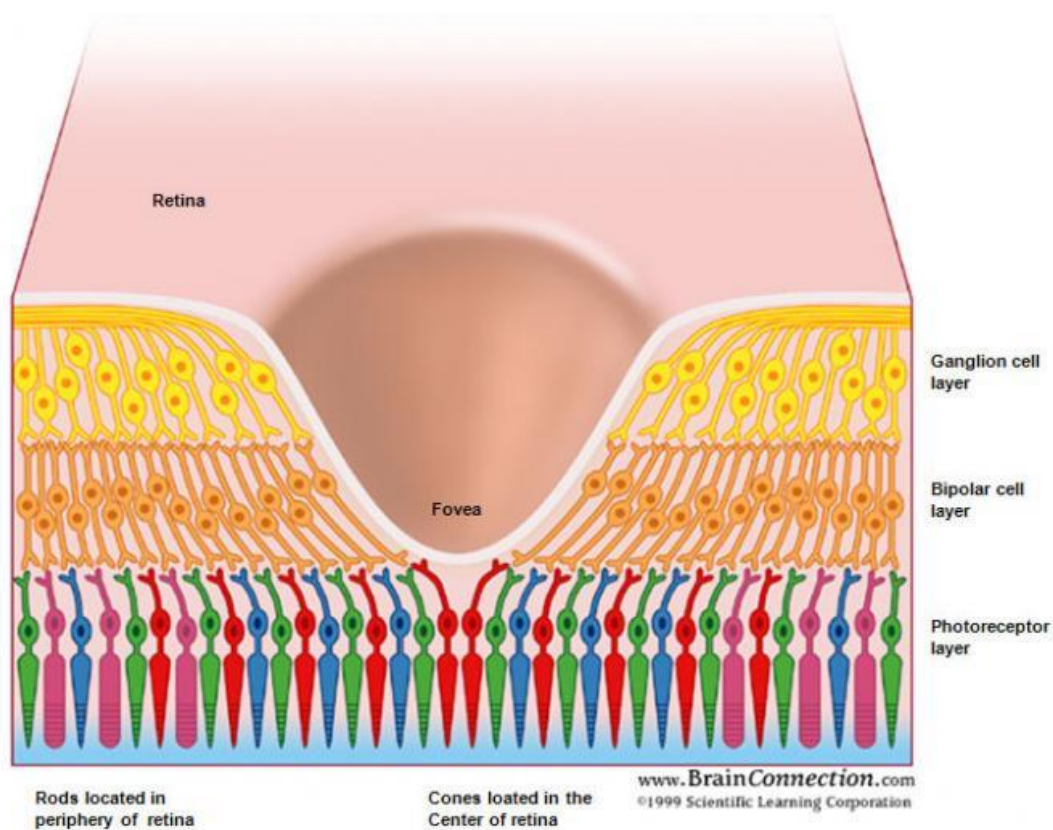
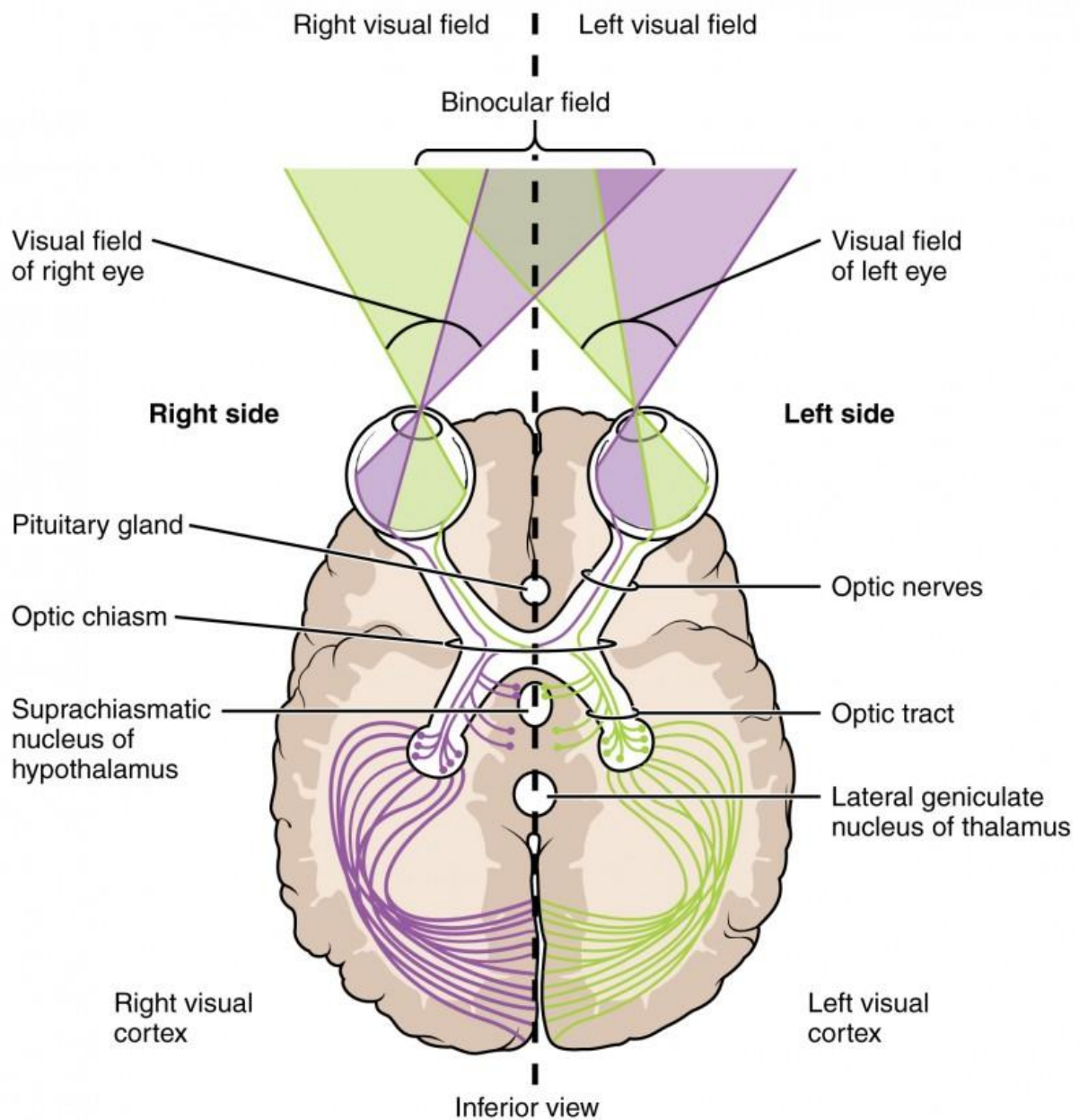


Figure 19. Layers of human retinal cells (reproduced from Dynamic Brain, 2019).

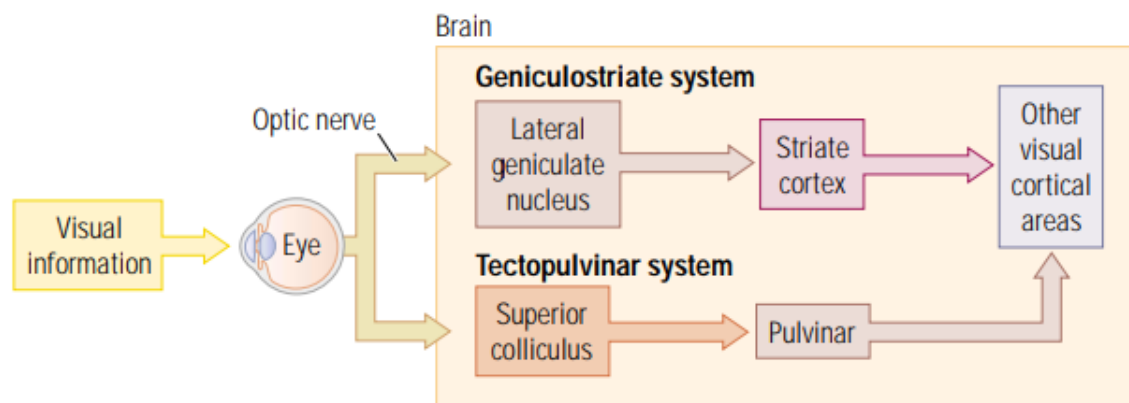
The visual process begins at the retina and progresses to the optic nerves, optic chiasm and optic tracts. The ipsilateral and decussating pathways are demonstrated in Figure 20 as well as the retinotopic visual scene projections to the primary visual areas (striate) in the occipital cortex. The optic chiasm is formed by the crossing of the optic nerves, with axons from the medial (nasal) retina decussating and continuing as the contralateral optic tract. Lateral (temporal) retina axons remain ipsilateral (do not cross at the chiasm) and feed into the ipsilateral optic tract. During primate vision, the majority of fibres from the medial half of the retina cross while all the fibres from the lateral half of the retina remain ipsilateral, enabling binocular vision (Reamington, 2012). Therefore, signals from the left visual field are passed to the right visual primary cortex ipsilaterally, via the temporal hemiretina of the right eye, or contralaterally, via the nasal hemiretina of the left eye, and vice versa for the right visual field.



**Figure 20. Segregation of the human visual field (reproduced from Betts et al., 2013).**

There are two major pathways within the human brain which process visual information. The largest pathway is the retina-geniculate-striate pathway which passes information from the retinal neurons in the eye to the optic nerve and then onto the primary visual cortex in the occipital lobe via the lateral geniculate nuclei (LGN) of the thalamus (Burman & Wurtz, 2008). The LGN is part of thalamic structures and is a relay centre and proposed ‘early gatekeeper’ in the wider visual pathways network (Kastner, Schneider & Wunderlich, 2006; Weyand, 2016).

Additionally, the LGN receives feedback connections from the primary visual and striate cortices (Briggs & Usrey, 2011). The second pathway is the tectopulvinar pathway. This pathway passes information from the optic nerve onto the superior colliculus (SC), a midbrain structure, next to the pulvinar nucleus in the thalamus, and lastly the visual cortex with primary projections to the parietal and temporal lobes (Kolb & Whishaw, 2016) (Figure 21).



**Figure 21. Diagram of the flow of visual information from the eye into the brain (reproduced from Kolb and Whishaw, 2016, p.234). The optic nerve consists of two branches. The first branch projects to the LGN in the thalamus (geniculostriate system) and the second branch projects to the SC (tectopulvinar system). The LGN pathway enters the occipital cortex and the information is passed to other visual regions in the temporal and parietal cortices. The SC pathway leads to the pulvinar and, again, onto the temporal and parietal cortices.**

After initial visual processing in the primary visual cortex, information is transmitted to secondary and association visual areas in the occipital lobes, collectively termed the striate cortex (Standring, 2015). The majority of pathways which mediate transference between the primary and secondary association areas are part of two main streams; the dorsal and the ventral stream (Goodale & Milner, 1992). In the most simple form, the dorsal stream (occipitoparietal pathway) projects visual information to the parietal cortex and has been coined the 'where is it' or 'how' pathway as it is responsible for the processing of spatial location and motion

processing. The ventral stream (occipitotemporal pathway) projects visual information to the temporal cortex and has been coined the 'what is it' pathway as it is responsible for colour, form, and identity of an object (Goodale & Milner, 1992). The occipitoparietal and occipitotemporal pathways are output pathways from the visual cortex and receive numerous top-down input from distributed brain regions (Kravitz, Saleem, Baker, Ungerleider & Mishkin, 2013). Goodale and Milner (1992) proposed that the main difference between the two streams was the function in output systems. Both the dorsal and ventral streams process information about the structure and location of an item but the two separate streams process and disseminate the information differently. The ventral stream generates perceptual representations from visual inputs concerning an object's structure and location (vision for perception). The dorsal stream mediates real-time visual actions, such as reaching and grasping, by constantly processing visual information about an object and creating and updating the egocentric coordinates of the object within the environment (vision for action). Goodale and Milner (1992) therefore proposed a division between the vision for perception and vision for conducting actions.

Empirical support for the 'two visual system hypothesis' is robust and heavily based on neuropsychological dissociations for object and spatial vision. For example, while some brain-injured individuals present with visual agnosia, the inability to recognise everyday objects, others present with optic ataxia, the inability to control visually orientated hand movements (Goodale, Milner, Jakobson & Carey, 1991; Martinaud et al., 2012; McIntosh, Mulroue, Blangero, Pisella & Rossetti, 2011). However, evidence supporting this apparent double dissociation has been challenged. For instance, Pisella, Binkofski, Lasek, Toni and Rossetti (2006) proposed that individuals in case studies were not administered an extensive visual perception assessment which raises questions concerning the validity of Goodale and Milner's (1992) conclusions. Milner and Goodale (2008) rebutted these challenges and suggested that some controversies around the 'two visual system hypothesis' were based on an imprecise

reading of some of the more subtle details of the model. The authors clarified the terms ‘vision for perception’ and ‘vision for action’ and provided a fuller account of the processing characteristics for these two kinds of vision. Nevertheless, there is still uncertainty surrounding the ‘two visual system hypothesis’. For example, Michel and Henaff (2004) argued against the dichotomy of the model, contending that the functional double dissociation, demonstrated by brain-injured individuals, could stem from aberrant central or peripheral vision rather than impairments in the vision for perception and vision for conducting actions. Rossetti, Pisella and McIntosh (2017) suggested that it is no longer tenable to assume independence between visuomotor and visual perception functions based solely on the double dissociation between optic ataxia and visual agnosia in brain-injured individuals. The authors highlighted the importance of taking into account the perception-action circle as a functional system. Compelling experimental and tractography research mapping white matter connectivity has shown a myriad of interconnected pathways, opposing the supposed isolated dual visual system theory. More than likely, the two systems approach is an oversimplification and there is cross-talk between the pathways (Budisavljevic, Dell’Acqua & Castiello, 2018). Indeed, a large contemporary fMRI study utilising data from the Human Connectome Project suggested that the functional human visual system may incorporate three, not two, cortical pathways (Haak & Beckmann, 2018), highlighting the complexity of the system and the reality that there are, more than likely, multiple visual circuits.

### **3.3. Human eye movements**

At the most basic level, human eye movements are vital for survival (Shaikh & Zee, 2018) but are also pivotal for more complex functions such as social interactions, as these functions are underpinned by the information acquired during eye movements (Emery, 2000). As humans have front-facing eyes, each eye captures different information. The brain has to integrate these



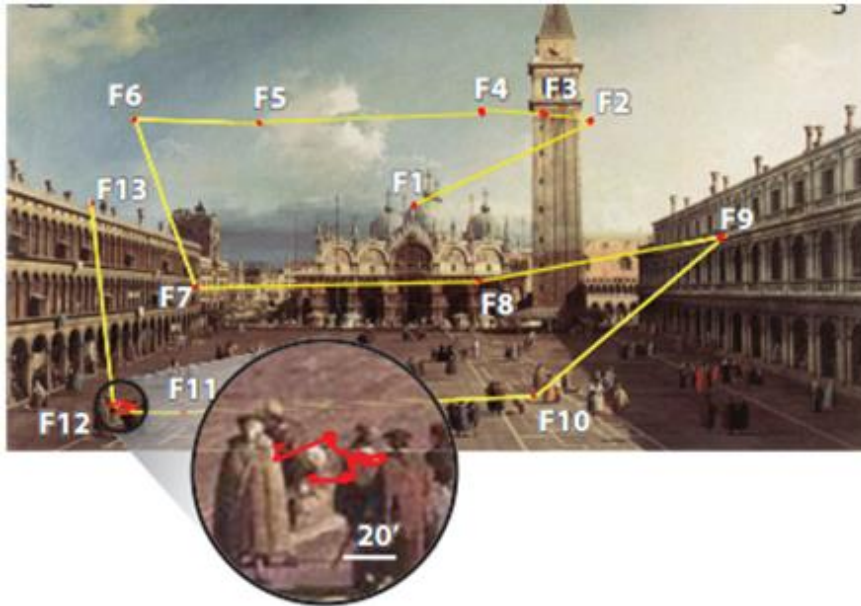
separate neural signals (binocular vision) to create a single 3D image with a sensation of depth (stereopsis). A seminal theory proposed by Hering (1977) suggested that both eyes were innately mediated by a single neural network and that the left and right eye muscles were always equally innervated. An opposing theory was proposed by Helmholtz and Atkinson (1873) who argued that each eye was controlled individually and that there was an element of conscious learning involved in visual orientation. Although both Hering's and Helmholtz's theories hold merit, developments in research techniques have revealed that binocular vision is a multi-component process, involving at least five neuro-visual systems which originate in the retina and are distributed throughout the brain (Coubard, 2015). During human visual processing, both eyes move together several times a second to hold areas of interest on the fovea (Krauzlis, Goffart & Hafed, 2017). High visual acuity in humans is restricted to the fovea, a very small pit in the centre of the retina composed of cone photoreceptors (Mccamy, Macknik & Matinez-Conde, 2014). Eye movements precipitate foveation, the process where areas of interest within the environment become concentrated on the fovea (Purves et al., 2001). On a basic level, the decision to move the eyes is dependent upon target-related signals from the peripheral visual field and signals from the fixated target at the fovea (Krauzlis et al., 2017). The most studied eye movements in humans are saccades, smooth pursuit and fixations; discussed below. The control of eye movements is navigated by several subsystems, described below, and is well-documented in animal and human research.

### **3.3.1. Saccades**

Saccades are high-velocity ballistic movements where both eyes move toward a point of visual space to redirect the point of fixation (Krauzlis et al., 2017). On average, humans generate a saccade two to three times per second (Pèlisson, Alahyane, Panouillères & Tilikete, 2010), and during this process, the eyes are moving so rapidly that no new visual information is acquired

(Berman, Cavanaugh, McAlonan & Wurtz, 2017; Bridgeman, Hendry & Stark, 1975; Gegenfurtner, 2016).

The saccadic system mediates information about the distance and direction of visual stimuli to ensure that an area of interest is projected onto the fovea (Sparks, 2002). The system also controls for gaze errors and integrates information to ensure that the visual scene is perceived as a unified whole (McCamy et al., 2014). Although all saccades require a degree of attentional processing, they are generally separated into conscious (voluntary) or unconscious (reflexive) movements (Ross, Morrone, Goldberg & Burr, 2001; Terao, Fukuda & Hikosaka, 2017). Reflexive saccades are rapid, automatic eye movements generated in response to a novel peripheral stimulus, while voluntary saccades are consciously controlled and usually made based on a symbolic cue or instruction (Walker, Walker, Husain & Kennard, 2000). Evidence indicates that reflexive and voluntary saccades rely on different neural networks (Cieslik, Seidler, Laird, Fox & Eickhoff, 2016; McDowell, Dyckman, Austin & Clementz, 2008), but they also rely on mutual neural networks including; the FEF, DLPFC, PEF, middle temporal regions, supplementary motor regions, occipital lobes, thalamus, SC, cerebellum, and brain stem structures (Berman et al., 1999; Cieslik et al., 2016; Kraus, 2013).



**Figure 22.** A diagram to illustrate saccades and fixations during natural vision (reproduced from Rucci & Poletti, 2015, p.500). This image outlines the eye scan pattern of one participant. The yellow lines represent saccades while fixations are represented by the red areas. F1 represents the first fixation, F2 the second fixation and so forth. As demonstrated in the enlarged circle, the eyes continue to elicit small movements during fixations.

### 3.3.2. Smooth pursuit

Smooth pursuit eye movements are slower than saccades and require continuous rotation of the eyes to avoid retinal slip (when the amplitude and/or velocity of eye movements are inaccurate, resulting in a gaze shift that induces the appearance of a stationary item as moving). A retinal slip would result in a blurring effect and therefore smooth pursuit ensures that moving targets (e.g. cars, animals, or people) can be tracked with high acuity (Krauzlis et al., 2017; Orban de Xivry & Lefevre, 2007).

### 3.3.3. Fixations

In the lapses between saccades and smooth pursuit, the eyes are held relatively still and fixation is maintained (Krauzlis et al., 2017). Martinez-Conde, Macknik, Troncoso and Hubel (2009)

defined fixations as ‘microscopic and unnoticed motions of the eyes made when fixating the gaze between larger eye movements’ (p. 463). It should be noted, while many researchers refer to fixations as the period when the eyes are stationary, this is somewhat of a misnomer (Rayner, 1998). The eyes are never completely static (Figure 23), and during fixations in humans, there are three categories of these types of constant eye movements; microsaccades, tremor, and drift (Martinez-Conde, 2005; see Figure 23).



**Figure 23. A diagram to illustrate fixational eye movements (reproduced from Martinez-Conde and Macknik, 2008, p.5). Microsaccades are quick and straight movements, drifts are slow and curved, and tremors are oscillations which overlap drifts.**

Fixational eye movements counteract visual neural adaptation (adjustment of the sensory system over time in response to a constant stimulus) so that a stimulus remains visible at fixation. They also inhibit visual fading (also known as Troxler fading where fixating on one element of the visual field results in fading or disappearance of surrounding visual information; Figure 24).



**Figure 24. An example of visual fading in normal vision (reproduced from Martinez-Conde, Macknik & Hubel, 2004, p.231). Precisely fixate on the red dot, without blinking, but pay attention to the light blue circle. After about ten seconds, the blue circle will disappear and it will appear that the red dot is surrounded by white. When you move your fixation the blue circle will reappear.**

The human visual system, therefore, presents as a paradox, humans must fixate on an object to process the finer details but if the fixation is maintained in a strictly static fashion then the visual scene would fade away. Microsaccades, tremors, and drifts counteract this paradox.

During human visual processing, the most useful visual information is collected during fixations (Morris, 2015). Human eye fixation data can be analysed to explore saliency in images or videos owing to the direct association between eye movements and visual attention (Duc, Bays & Husain, 2008). Distinguishing between areas of interest of a visual scene, as indicated by fixations, aids the understanding of the behavioural underpinnings of human visual attention (Atkinson, Simpson & Cole, 2017; Edwards, Stepenson, Dalmaso & Bayliss, 2015).

### **3.3.3.1. Microsaccades**

Microsaccades are small, rapid, straight eye movements which arise during fixations (Martinez-Conde, 2005; Figure 23). The specific functionality of microsaccades is still under

investigation, although the relationships between microsaccades, perception, and cognition have been subject to much research over the past decade. Martinez-Conde, et al., (2009) proposed that regardless of minor discrepancies between research groups, there is a consensus that microsaccades modulate neural activity in early visual areas through retinal motion. Microsaccades are reported to enhance edge sensitivity and spatial resolution (Donner & Hemilä, 2007), and to render latency and contrast in perception (Martinez-Conde et al., 2004). It is probable that they also play a role in correcting fixation errors (Engbert & Kliegl, 2004). Also, microsaccades are theorised to be critical in overcoming neural adaptation (Costela et al., 2017; Martinez-Conde, Macknik, Troncoso & Dyar, 2006; Troncoso, Macknik & Martinez-Conde, 2008), as well as being vital for several other aspects of attention, such as; spatial cueing, visual exploration, and visual search (Meyberg, Sinn, Engbert & Sommer, 2017; Otero-Millan, Troncoso, Macknik, Serrano-Pedraza & Martinez-Conde, 2008). Microsaccades and saccades have similar functional and physical characteristics, implying that both eye movements may share common oculomotor origins (Otero-Millan et al., 2008; Rolfs, Kliegl & Engbert, 2008). Animal models and fMRI studies have revealed neuron activation in response to microsaccades in the LGN, area V1 of the occipital cortex, and extrastriate regions (Donner & Hemilä, 2007; Peter, Tse, Baumgartner & Greenlee, 2010). Similar to saccades, the SC is thought to be pivotal for the generation of microsaccades (Chen & Hafed, 2017; Chen, Ignashchenkova, Thier & Hafed, 2015). Microsaccades and saccades activate burst neurons in the pontomedullary reticular formation, which is downstream from the SC, and putative motor neurons in the nearby abducens nucleus (eye motor cranial nerve; Van Gisbergen, Robinson & Gielen, 1981).

### **3.3.3.2. Tremors**

The aperiodic wave-like movement of the eyes is referred to as a tremor and is the smallest of all eye movements (Figure 23). The role of tremor in vision is still unknown but it is theorised

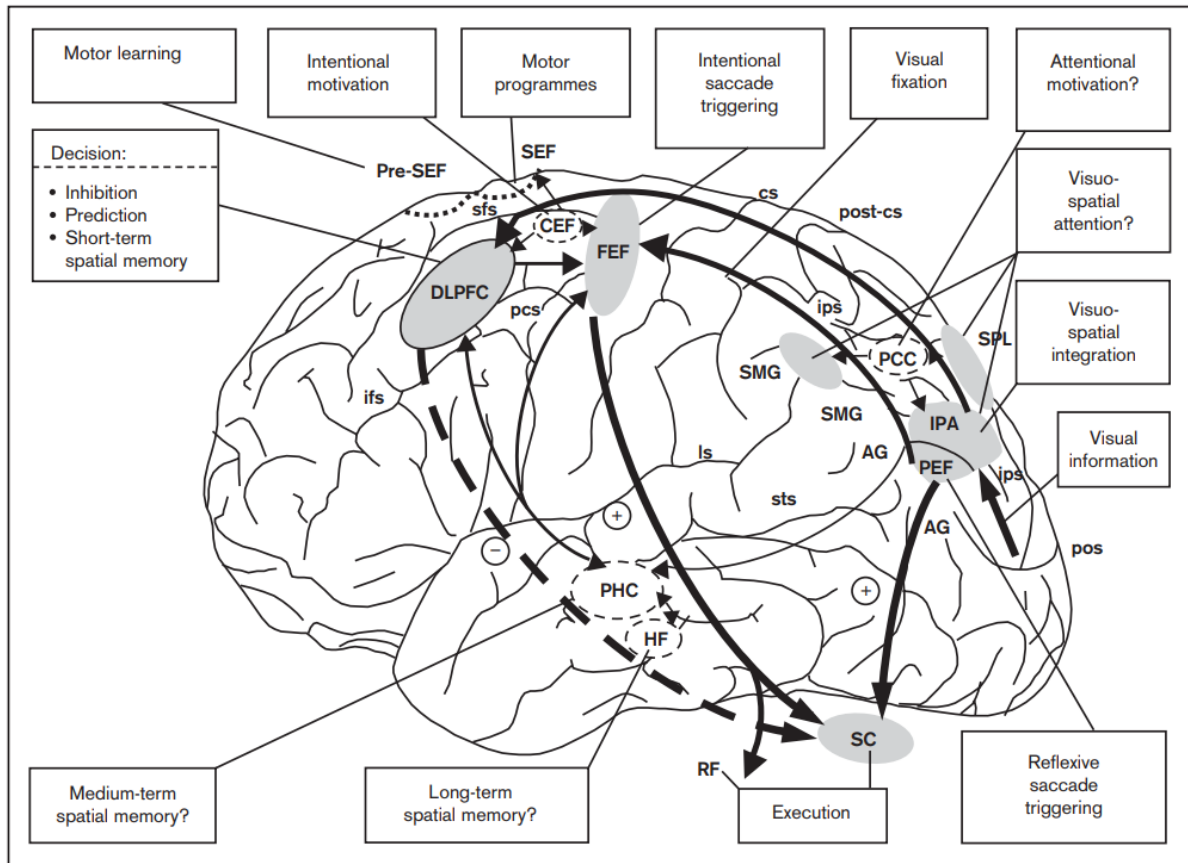
that the movement may be associated with ensuring there is a continuous source of image motion to offset visual fading (Martinez-Conde et al., 2004).

### **3.3.3.3. Drifts**

Drifts emerge in synchrony with tremor movements and are slow curved eye movements which occur during the period's in-between microsaccades (Figure 23). Drifts are hypothesised to correct fixation position and disparity (Cyr & Fender, 1969; Steinman, Cunitz, Timberlake & Herman, 1967), and drifts and tremors work in unison during high spatial frequency processing (Kuang, Poletti, Victor & Rucci, 2012).

## **3.4. Neural control centres for eye movements**

The neural basis of human oculomotor function is extensive and a detailed account of these processes is beyond the scope of this thesis. Instead, there is a specific focus on the FEF, SEF, DLPFC and PEF as these are documented to be significant oculomotor structures (Pierrot-Deseilligny, Milea & Müri, 2004; Figure 24), and are vulnerable to damage during head trauma (Bigler, 2013; Jenkins, Mehta & Sharp, 2016; McAllister, 2011). Intricate connections between the frontal and parietal cortices form neural networks which modulate oculomotor functions, such as spatial attention (Ptak & Müri, 2013) and oculomotor sequence learning (Gonzalez, Billington & Burke, 2016); vital for successful social interactions (Gobel, Tufft & Richardson, 2017). TBI is evidenced to disrupt this brain connectivity (Hayes, Bigler & Verfaellie, 2016; Kinnunen et al., 2010) and, therefore, may impede social reintegration after TBI, although research on this hypothesis is scant.



**Figure 25. Major cortical brain regions and pathways involved in eye movement generation and control (reproduced from Pierrot-Deseilligny et al., 2004, p.19). Abbreviations: supplementary eye fields (SEF), superior frontal sulcus (SFS), cingulate eye field (CEF), central sulcus (CS), dorsolateral prefrontal cortex (DLPFC), precentral sulcus (PCS), frontal eye fields (FEF), intraparietal sulcus (IPS), inferior frontal sulcus (IFS), supramarginal gyrus (SMG), posterior cingulate cortex (PCC), superior parietal lobule (SPL), intraparietal areas (IPA), lateral sulcus (ls), angular gyrus (AG), posterior eye fields (PEF), superior temporal sulcus (sts), parieto-occipital sulcus (pos), parahippocampal cortex (PHC), hippocampal formation (HF), superior colliculus (SC), reticular formations (RF).**

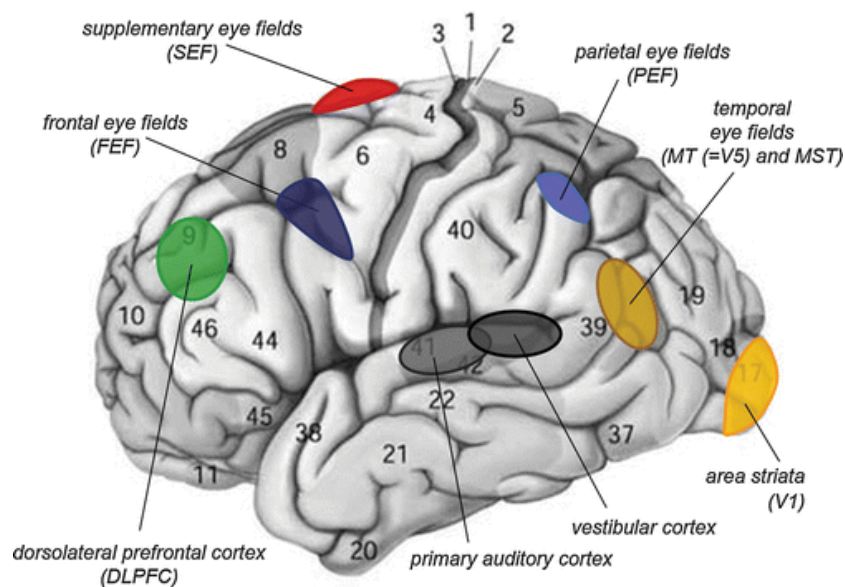
### **3.4.1. Frontal eye fields (FEF)**

The FEF are a vital part of the cortical network controlling eye movements (Figure 25). Ferrier's (1874) seminal work exploring the oculomotor effect of cortical stimulation in monkeys revealed that eye movements could be evoked from the frontal, temporal, and parietal



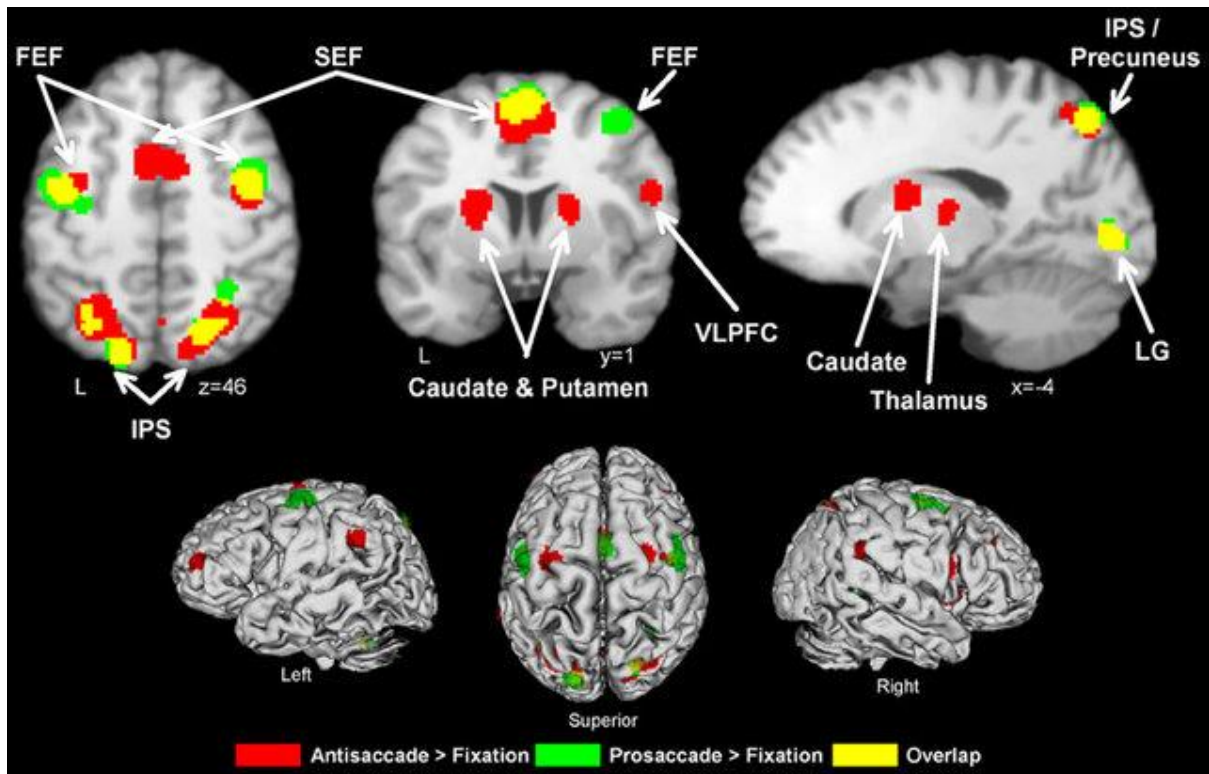
lobes, as well as the SC and cerebellum. Ferrier (1874) proposed that the monkey frontal lobe eye field was quite large and extended from the arcuate sulcus to the midline. The monkey FEF is thought to correspond to Brodmann area (BA) 8 (Brodmann, 1908; Watanabe, 2017). The location of the human FEF is less certain and is still debated within the literature (Percheron, François & Pouget, 2015; Vernet et al., 2014) with different studies showing activation in contrasting brain areas (Amiez & Petrides, 2009).

Paus (1996) reviewed eight studies investigating the location of the human FEF in the brain using cerebral blood-flow levels measured through positron emission tomography (PET) scans. Findings indicated that an area of the precentral sulcus and superior frontal sulcus (BA 6) showed increased cerebral blood-flow levels during a variety of oculomotor tasks (e.g. anti-saccade and pro-saccade tasks; Figure 26). Electrostimulation studies have supported these findings and have localised the human FEF anterior to the superior precentral sulcus (Blanke et al., 2000; Yamamoto et al., 2004).



**Figure 26. The proposed location of human cortical eye fields with corresponding BA's (reproduced from Gulyás, 2016, p.525).**

In other work, Grosbras, Laird & Paus (2005) conducted a quantitative meta-analysis of 59 PET and fMRI experiments investigating voluntary and involuntary triggered eye movements. The authors computed activation likelihood estimation maps based on the study data. Findings supported spatial localisation of the FEF in humans to the precentral sulcus, close to the junction of the superior frontal sulcus. However, both of the above meta-analyses had some limitations in that several of the studies included did not image the whole brain. This could lead to confirmation bias, with findings based on incomplete evidence when selecting areas of activation. It could be argued that it is hard to draw inferences from meta-analyses which have aggregated data from diverse visual paradigms, as they involve different cognitive processes and different neural networks. For example, during an anti-saccade task, the participant is required to exhibit a pre-emptive top-down inhibition of a saccade and then a voluntary anti-saccade must override a reflexive saccade (Coe & Munoz, 2017). During a pro-saccade task, the participant must encode the location of a target, remember this information during a delay period, and then generate an accurate saccade to the previous location (Srimal & Curtis, 2011; Figure 27). Hence, these eye movements are quite distinct neural mechanisms governed by different brain circuitry. Interestingly, FEF's are not confined to a single brain region with evidence suggesting that different FEF neurons mediate motor, visual, and visuomotor information (Crapse & Sommer, 2009; Lawrence & Snyder, 2009; Schall, Purcell, Heitz, Logan & Palmeri, 2011).



**Figure 27. Activation likelihood maps displaying the FEF and SEF during an anti-saccade and pro-saccade task in humans (reproduced from Jamadar, Fielding & Egan, 2013, p.6). The yellow regions represent overlap, the red represents activation during an anti-saccade and green represents activation during a pro-saccade. Abbreviations: frontal eye field (FEF); supplementary eye field (SEF); superior parietal lobule (SPL); intraparietal sulcus (IPS); inferior frontal gyrus (IFG); lingual gyrus (LG).**

In human work, Amiez and Petrides (2012) used fMRI methodology to assess eye movements and reported that basic eye movements (following a dot) caused activation in the ventral branch of the superior precentral sulcus, while high-level eye movements (performing appropriate eye movements to previously learned conditional rules) caused activation in the superior frontal sulcus. These findings illustrated functional sub-areas within the FEF in humans. Tseng, Wang, Lo and Juan (2018) used transcranial direct current stimulation (tDCS) to apply anodal (positively charged electrode) and cathodal (negatively charged electrode) currents over the right FEF while 53 control participants performed pro and anti-saccade tasks. The researchers recorded facilitation in pro-saccades towards low-probability locations compared to high-

probability locations when anodal tDCS was administered, and facilitation of anti-saccades away from the high-probability location when cathodal tDCS was applied. Based on these findings, the authors proposed an overlapping spatial endpoint-selective mechanism between pro-saccades and anti-saccades within human FEF. However, similar research by Reteig, Knapen, Roelofs, Ridderinkhof and Slagter (2018) reported contradictory findings to Tseng et al., (2018). They reported null effects for latency and accuracy of pro-saccades during tDCS administration over the FEF in 26 control participants. These contrasting findings might reflect sample size differences or methodological inconsistencies, as Tseng et al., (2018) only included data where participants showed a positive probability effect (i.e. they were sensitive and responsive during a practice tDCS) whereas Reteig et al., (2018) did not discriminate between participants included in the study on this basis.

Hutchison et al., (2012) investigated the functional connectivity of the FEF in humans using resting-state fMRI. They used Petrides and Pandya's (2012) comparative cytoarchitectonic analysis to assign corticocortical patterns to the human brain (see chapter one for more details of Petrides and Pandya's analysis). Findings revealed frontal areas that shared functional connectivity with the FEF. The study also revealed strong functional connectivity between the FEF and posterior cortical areas including; the IPS, posterior central sulcus, parieto-occipital sulcus, upper superior temporal sulcus, and cingulate sulcus marginal ramus. These findings likely represent spatial and time-based inputs to the FEF.

It is well established that the FEF are involved in the control and initiation of voluntary saccades (Cameron, Riddle & D'Esposito, 2015; Tseng et al., 2018), including predictive memory-guided and anti-saccades (Pierrot-Deseilligny, Müri, Ploner, Gaymard & Rivaud-Péchéux, 2003), and for transforming visual signals into saccade commands (Schall & Thompson, 1999). It is generally accepted that the FEF have relatively little control over

reflexive saccades (Pierrot-Deseilligny et al., 2004). To test this assumption, van der Stigchel, Koningsbruggen, Nijboer, List and Rafal (2012) recruited four individuals with lesions to the FEF (three stroke patients and one schizencephally patient, a developmental birth defect which causes abnormal clefts in the cerebral cortex) and asked them to complete an anti-saccade task and an oculomotor capture task (a visual search task where the target is endogenous and no reflexive eye movements should be activated). They compared performance in the contralesional compared to ipsilesional visual fields of the four individuals, therefore acting as their controls. The participants displayed more errors on both tasks for contralesional compared to ipsilesional stimuli. While the study supported the role of the FEF in suppressing voluntary saccades, as demonstrated through patient's impairments on the anti-saccade task, the findings also suggested that the FEF may mediate aspects of reflexive inhibition, as participants also had impaired performance on the oculomotor capture task. Subsequent research by Peel, Hafed, Dash, Lomber and Corneil (2016) has supported the theory that the FEF may modulate facets of reflexive saccades. The research group administered unilateral and bilateral pharmacological reversible inactivation of the FEF in three macaque monkeys in an attempt to identify a cortical location responsible for generating reflexive microsaccades. The authors discovered that inactivation of the FEF leads to impairments in microsaccade metrics, kinematics (the study of motion without consideration of mass or other forces), and generation to peripheral cues. Peel et al., (2016) concluded that the FEF supplies top-down information for the generation of reflexive microsaccades. However, there are studies which have reported no error increases on anti-saccade tasks when transcranial magnetic stimulation (TMS) is applied to the human FEF (Nagel et al., 2008; Olk, Chang, Kingstone & Ro, 2005), although response times increased when TMS was applied during an anti-saccade task.

In summary, the FEF is an oculomotor region involved in many visual processes. It is well established that the FEF is vital for successful voluntary saccades (Cameron et al., 2015; Tsang

et al., 2018), with mounting evidence that this region may also play a role in reflexive saccades (van der Stigchel et al., 2012; Peel et al., 2016). Furthermore, the region appears to modulate the direction of covert spatial attention (Schall, 2004) as well as possibly mediating aspects of smooth pursuit (Missal & Heinen, 2017), vergence (Searle & Rowe, 2016), and fixations (Krauzlis et al., 2017).

### **3.4.2. Supplementary eye fields (SEF)**

The SEF constitute an integral part of the network controlling eye movements. In their pioneering research, Penfield and Rasmussen (1950) noticed that electrical stimulation, during craniotomy, of the human rostral supplementary motor area (SMA), induced gaze shifts. Schlag and Schlag-Rey (1985, 1987) recorded gaze and head movements when single-unit activity was administered to the dorsomedial edge of the frontal lobe, above the superior arcuate sulcus, in three trained monkeys. The authors proposed that this was a supplementary eye field, separate from the FEF, and termed the area the SEF. The human SEF is thought to correlate with the dorsal bank of the cingulate sulcus in the monkey brain. Hutchison et al., (2012) proposed that the SEF boundary was located along the anterior-posterior axis within the medial wall, dorsal to the cingulate sulcus. The connective network of the dorsomedial region is extensive and complex and innervates the FEF and the SC in monkeys (Huerta & Kaas, 1990). The SEF also receives reciprocal projections from the motor cortex (Lu, Preston & Strick, 1994) and projects to the brainstem and spinal cord nuclei involved in eye, head, and limb movement (Huerta & Kaas, 1990). Visual centres, including the lateral IPS area and the medial superior temporal area, innervate the region, while reciprocal connections to the prefrontal cortex form pathways to thalamic and striatal areas associated with an oculomotor function (Huerta & Kaas, 1990). Evidence suggests that SEF may contain a topographic map (Savaki, Gregoriou, Bakola & Moschovakis, 2015; Stuphorn, 2015), and single-unit recording

experiments have demonstrated that eye movements can be evoked when the rostral area is activated, compared to when stimulation is applied to caudal or between rostral and caudal areas, which evoked hind-leg and forelimb movement.

Neural recordings from monkey electro-stimulation studies and human imaging studies have documented activity in the SEF during a range of saccadic tasks including, but not limited to; distinguishing between successful and unsuccessful cancellation of saccades (Curtis, Cole, Rao, & D'Esposito, 2005), smooth pursuit (Missal & Heinen, 2017), object-centred spatial awareness (Olson & Gettner, 1995, 1996, 1999), change-of-plan saccades (Nachev, Rees, Parton, Kennard, & Husain, 2005), memory-guided saccade sequences (Lu, Matsuzawa, & Hikosaka, 2002), and serial decision-making (Abzug & Sommer, 2018).

Several studies have investigated the functional role of the SEF in oculomotor behaviour after brain injury in humans (Gaymard, Pierrot-Deseilligny, & Rivaud, 1990; Heide, Kurzidim, & Kompf, 1996), reporting impairments in sequential eye movements and saccades anticipating predictable target motion. However, these experiments had small sample sizes, and lesions were not localized specifically to SEF, therefore, findings cannot be attributed to discrete SEF damage but probably reflect disruption to multiple systems.

Parton et al., (2007) reported the case study of JR, a 55-year-old male who suffered a small left medial frontal venous stroke eight months prior. Using high-resolution structural MRI, the research team precisely localized the bilateral lesion to SEF in both axial and sagittal planes and confirmed that pathology did not extend beyond this specific area. JR completed anti-saccade, pro-saccade, and memory-guided saccade tasks, as well as a task that involved learning arbitrary oculomotor stimulus-response mappings through trial and error. JR's performance on the anti and pro-saccade tasks exceeded that of age-matched controls and he made fewer errors overall. Overall, JR was slower at generating saccades and this may explain

his high accuracy scores; a speed/accuracy trade-off. However, he showed impaired performance switching from anti to pro-saccades on a conflict task compared to age-matched controls. He also showed prolonged latencies on the arbitrary stimulus-response task when selecting the correct saccade compared to controls. These findings suggest that the SEF may not be necessary for instigating saccades, but mediates the implementation of control when there is a conflict between competing saccadic responses, but not when saccades need to be made accurately in sequence. Indeed, it seems likely that that SEF does not directly control saccades but rather mediate higher-order cognitive variables during saccades, such as monitoring error, conflict, and reward (Husain, Parton, Hodgson, Mort & Rees, 2003; Stuphorn, Brown, & Schall, 2009; Roesch & Olson, 2003).

In summary, the role of the SEF in human oculomotor function is still under investigation. It is generally agreed that the SEF is not directly involved in the generation of eye movements but instead mediates higher-order cognitive functions during saccades (Husain et al., 2002; Stuphorn et al., 2010; Roesch & Olson, 2003).

### **3.4.3. Dorsolateral prefrontal cortex (DLPFC)**

The location and connections of the DLPFC are discussed in detail in chapter one but, in brief, the area encompasses area 8, 9 and 46 and has complex interconnections with subcortical circuits including the FEF, SEF, and PEF (Cummings & Miller, 2007; Pierrot-Deseillingny, Müri, Nyffeler & Milea, 2005; Figure 26). Lesion, tDCS, and neuroimaging studies have reported that the human DLPFC pathways mediate aspects of executive function control, the capability to undertake goal-directed behaviour using complex mental processes (Manes et al., 2002; Nejati, Salehinejad & Nitsche, 2018; Yuan & Raz, 2014), and monkey and human DLPFC have both been associated with executive control of anti-saccades (Coe & Munoz, 2017; Johnston & Everling, 2006; Munoz & Everling, 2004).



The anti-saccade movement requires two mechanisms; firstly executive resources (Tarnowski, 2013) suppressing reflexive saccades (DLPFC inhibits reflexive saccade generated by the PEF), and secondly, generating the correct anti-saccade (generated by the FEF; Ploner, Gaymard, Rivaud-Péchoux & Pierrot-Deseilligny, 2005). Human DLPFC lesions have resulted in an increased percentage of errors in the anti-saccade task, while FEF lesions can cause an increase in the latency of correct anti-saccades (Gaymard, Ploner, Rivaud-Pechoux & Pierrot-Deseilligny, 1999; Pierrot-Deseilligny et al., 1991; Rivaud, Müri, Gaymard, Vermersch & Pierrot-Deseilligny, 1994). This dissociation suggests an inhibitory role of the DLPFC.

Ploner et al., (2005) tested 15 participants with acute unilateral ischemic lesions of the prefrontal cortex and 20 controls on an anti-saccade task to determine if there was a distinct sub-region of the human prefrontal cortex responsible for reflexive saccades. They found that lesions affecting a region in the mid-DLPFC, or the white matter between this region and the anterior portions of the internal capsule, resulted in increased anti-saccade errors. Lesions outside of these areas did not appear to affect anti-saccade error rates. These results indicated that the DLPFC inhibit reflexive saccades. However, the authors excluded participants who had sustained 'unusual' strokes such as vasculitis and venous thrombosis, only including individuals who had sustained unilateral atherothrombotic or embolic infarction. Although researchers control for differences in lesions, each human brain damage case is essentially unique. For instance, evidence suggests that different subtypes of typical strokes (e.g. atherothrombotic or embolic) still produce differences in pathogenesis (Arboix, Oliveres, Massons, Pujades & Garcia-Eroles, 1997; Kozuka et al., 2002), making it difficult to ascertain the reliability and validity of human lesion studies and to generalise the results.

It is theorised that there may be a 'task-set' neuronal organisation in the DLPFC with different sets of neurons exhibiting activation for anti and pro-saccade instructions (Everling & DeSouza,

2005). The DLPFC is active during the preparatory phase of anti-saccades and may also mediate inhibitory signals to saccade-related regions, such as the SC. Johnston and Everling (2006) recorded DLPFC activity in monkeys when they performed alternating pro and anti-saccade trials. The researchers discovered that the DLPFC appeared to generate task-selective signals (e.g. stimulus location and saccade direction) to the SC, suggesting that the region is involved in controlling the activity of other brain regions depending on the goal of the task. Although Johnston and Everling's (2006) research provides evidence of single neuron behaviour in the monkey DLPFC, it is still not clear how this very specific neuronal activity functions in the human oculomotor system (Johnston, DeSouza & Everling, 2009), and therefore it remains a hypothetical model until direct human data are established.

TMS pulse activation over the DLPFC of control participants during the preparatory phase of anti-saccades increased errors in anti-saccades, while the application of TMS after the preparatory phase did not affect anti-saccades (Nyffeler et al., 2007). These findings suggest a critical time frame where the DLPFC can inhibit reflexive saccades during the anti-saccade task, and this time interval is before the onset of the stimulus. While TMS can provide causal brain-behaviour information in humans, the method does have some disadvantages. For example, Nyffeler et al.'s (2007) localisation procedure for positioning the TMS coil over the DLPFC relied upon functionally localising TMS 5cm anteriorly from the motor hand area. However, there is no guarantee that this TMS placement specifically targeted the DLPFC in each of the 15 participants, as genetic and environmental effects may contribute to individual differences in adult human brain structure (Gu & Kanai, 2014).

In other work, human lesion (Pierrot-Deseilligny et al., 2003; Pierrot-Deseilligny et al., 1991; Ploner et al., 1999) and TMS studies (Müri et al., 2000) have reported that the DLPFC may be involved in memory-guided saccades. However, a recent study by Mackey, Devinsky, Doyle,

Meager and Curtis (2016) reported that individuals who had sustained damage to the DLPFC, which did not encroach on the precentral sulcus, performed equally well on a memory-guided saccade task compared to controls. Mackey et al.'s., (2016) findings may contrast with other studies conducting similar research because much of the earlier work was based on patient populations who had sustained strokes affecting large areas of the DLPFC.

In summary, the DLPFC exerts executive control over the programming and preparation of anti-saccades (Cameron et al., 2015). The region inhibits unnecessary reflexive saccades, triggered by the PEF, by mediating bias signals to saccade-related areas (Pierrot-Deseillingny et al., 2005). Therefore, pathology to this region may produce selective deficits in these visual mechanisms depending on lesion location and severity, thus impacting the processing of visual stimuli.

#### **3.4.4. Parietal eye fields (PEF)**

The posterior parietal cortex (PPC) provides an interface for visuospatial attention (Yang, Jacobson & Burwell, 2017; Wu et al., 2016) and accurate oculomotor movement plans; a critical process for the integration of multiple sensory modalities to construct a representation of the body in the external world (Ptak & Müri, 2013). The intraparietal sulcus separates the PPC into superior and inferior lobules. The superior regions primary inputs are somatosensory, while the inferior regions are visual-based (Paré and Dorris, 2011). In non-human primates, there is a specialised region in the IPS, known as the lateral intraparietal area (LIP), located in area 7a. The LIP is anatomically embedded between visual brain areas (occipital lobes) and saccade executive centres (frontal lobes; Thomas & Paré, 2006) so goal-directed attentional activity can be co-ordinated (Bisley, Mirpour, Arcizet & Ong, 2011; Freedman & Assad, 2006). Certain neurons in the LIP respond to visual (Gottlieb, Kusunoki & Goldberg, 2004) and visuomotor stimuli (Colby & Duhamel, 1991), with others active during fixations

(Constantinidis & Steinmetz, 2001). The human homologue of the LIP is termed the PEF and is theorised to be located along the IPS within the sulcus, in its posterior half, adjacent laterally to the anterior part of the angular gyrus (area 39) and medially to the posterior part of the superior parietal lobule (area 7) (Pierrot-Deseilligny et al., 2004; Pouget, 2015) (Figures 25 and 26).

The role of the PEF is still being researched, but extant literature suggests that the region may modulate aspects of visual-spatial attention (Goldberg, Bisley, Powell et al., 2006) and the intention to make a saccade (Andersen & Cui, 2009). The PEF are thought to be an integral system for visual working memory, involving eye movements to remembered locations. The ability to temporarily remember information is necessary for goal-driven behaviour and has been investigated with delayed-oculomotor-response tasks in humans and monkeys (Mirpour, Ong & Bisley, 2010). Seminal research by Gnadt and Anderson (1988) demonstrated that monkey LIP neurons were activated during the delay period of saccade tasks, with many studies supporting the author's early findings. Goldberg et al., (2006) reported that monkey LIP neurons responded to the appearance of a flashed distracter even when the monkey was required to generate a memory-guided delayed saccade elsewhere. This data suggested that the monkey's attention was aligned with the goal of a memory-guided saccade unless a distracter appeared, in which case attention was directed to the distracter and then back to the memory-guided saccade. Goldberg et al., (2006) postulated that LIP neurons generated a salience map which mediated saccade goals and locus of attention. Mackey et al., (2016) compared the performance of three human participants with lesions to PEF (from surgical resections of cortical tissue for a tumour or focal epilepsy) to 12 controls on a memory-guided saccade task (measuring working memory), and a visually guided saccade task (to control for impairments in spatial or visuomotor processing). The individuals who had been affected by brain injury exhibited slower and less accurate memory-guided saccades compared to controls, while no

differences were found on the visual-guided saccade task. Mackey et al.'s., (2016) work is innovative in paralleling human and monkey PEF/LIP control over memory-guided saccades. However, there were large differences in age (35 years), lesion size (36.7 mm), and time since lesion (10.5 years) between the three participants. Furthermore, there were differences in hemispheric location, aetiology and specific PEF region of the lesion. Mackey and Curtis (2017) used TMS over the parietal cortex of two human control participants in a robust experimental study including fMRI and nonlinear population receptive field mapping to locate stimulation sites. The results indicated that TMS administration caused memory-guided saccade errors, supporting the research team's earlier lesion study. It appeared that PEF neurons control attention (Snyder, Batista, & Andersen, 2000) and provide a saliency map as so attention and saccades can be regulated (Goldberg et al., 2006).

In summary, the primary oculomotor function of the PEF appears to be the integration of visual and goal-directed information (Mirpour et al., 2010). This includes the transformation of visual information into a saliency map (Goldberg et al., 2006; Gottlieb, 2007), where a representation of spatial locations can be created and selected to guide saccade generation (Colby & Goldberg, 1999).

In conclusion, it is generally accepted that the FEF mediates intentional saccades, the SEF modulates the generation of motor programs saccades, the DLPFC mediates the remembering, inhibition, and prediction of saccades and the PEF informs saccade generation through saliency maps. These circuits form the basis of visual processing of complex social stimuli.

### **3.5. Typical human visual attention and the effects of TBI**

There have been many studies focussing on emotional and social deficits after neuropathology (May et al., 2017; McDonald et al., 2018; McDonald et al., 2017). These have been based on the assumption that impairments are due to disrupted impulse control and/or emotion regulation

i.e. failure in executive functions (van der Horn et al., 2016; Wood & Worthington, 2017) or impaired facial affect recognition (Rigon et al., 2016). One key consideration, however, is that all higher-level visuospatial functions require accurate visual data extraction, and this is the start point for subsequent higher socio-cognitive functions. Few studies have investigated whether these fundamental processes remain intact post-TBI. Research that does explore this often has small sample sizes and implements static stimuli, which lacks ecological validity (Douglas, Vassallo & White, 2010; Vassallo, Douglas & White, 2010; Vassallo et al., 2011).

The importance of low-level visual processes to normal social functioning cannot be underestimated. During everyday functions, dyadic relationships and social interactions rely on constant and diverse visual processing, including action observation and the ability to read others' feelings and intentions through facial affect (Matsumoto, Takahashi, Murai & Takahashi, 2015). Normally functioning eye movement mechanisms are critical for these functions (Gobel, Kim & Richardson, 2015; Freeth, Foulsham & Kingstone, 2013).

The visual system has developed across evolution to detect biological motion (Nishimoto, Huth, Bilenko & Gallant, 2017; Yovel & O'Toole, 2016), focus attention on the most salient area of the visual scene (Schurgin et al., 2014; Vabalas & Freeth, 2016), and to participate in joint attention (Cazzato, Mazzeo, Spagnolo & Distanto, 2015; Emery, 2000). These processes are thought to be innate (Bardi, Regolin & Simion, 2014; Leppänen, 2016) and largely automatic, for instance, human adults evoke rapid responses during facial expression recognition tasks, with happiness being recognised as quickly as 23 milliseconds after stimuli onset (Martinez & Du, 2010).

Similarly, Hsiao and Cottrell (2008) reported that, on average, it takes adults just two fixations to recognise a face. However, these findings were based on data collected in an experimental context and eye scanning patterns may differ in naturalistic settings. Typically, during natural

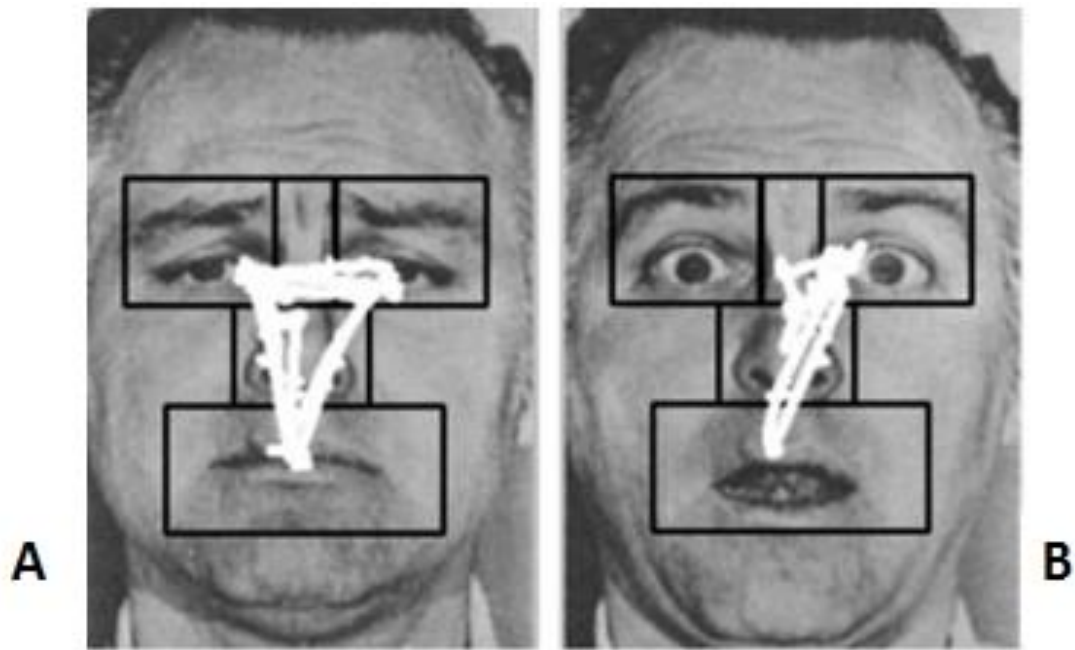
vision, eye movements are only directed to salient information and it is seldom that random saccades or fixations are made (Hayhoe, Shrivastave & Nrucek, 2003; Peterson & Eckstein, 2012). Everyday tasks vary greatly (e.g. participating in a conversation compared to making a cup of tea) and, therefore, what is considered salient information will also vary. Hayhoe and Ballard (2005) reported that the majority of fixations will be directed towards the object which is paramount for the current goal, coining the ‘just in time’ selection strategy (the acquisition of specific information at the point it is required and no sooner). So, for instance, when making a cup of tea, fixations would be generated towards the cup rather than areas around it (Figure 28). Gaze positioning also informs the musculoskeletal system indicating that gaze monitoring and motor output have rapid communication and reciprocal circuitry (Cesqui, Mezzetti, Lacquaniti & d’Avella, 2015; Koike, Sumiya, Nakagawa, Okazaki & Sadato, 2019; Oullier, de Guzman, Jantzen, Lagarde & Kelso, 2008).



**Figure 28. A participant’s point of gaze (circle) while picking up a mug (reproduced from Foulsham, 2015, p. 197). The participant fixates immediately on the handle, about 0.5 s before reaching for the object. Once lifted, gaze moves to a different location. Fixations are directed on the most pertinent part of the visual scene (i.e. the handle for picking the cup up and the rim for going to take a drink).**

Concerning face processing, salient facial features are well-known. Research suggests that during social interactions, internal facial features (eyes, nose, mouth) are more attended to compared to external features (hair, face, forehead, ears; Mehoudar, Arizpe, Maker & Yovek, 2014; Schurgin et al., 2014; Vassallo et al., 2011). More specifically, human eye-tracking research investigating the identification of emotional facial expressions showed that the eye region is the most fixated area of the face, due to the amount of social information which can be extracted from the area, including mood, attention, and identity (Eisenbarth & Alpers, 2011; Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells, Gillespie & Rotshtein, 2016). An interesting paper by Keil (2009) suggested that the eyes are the most informative feature for person identification, as the area provides the least ‘noisy’ information, that is, the eyes contain more reliable information compared to the nose and mouth. It has been documented that humans frequently display five isostatic fixation patterns of oculomotor activity when viewing the human face (Ananyeva et al., 2010). During the perception of basic facial expressions, humans tend to display a triangular model of eye movements, known as a ‘T pattern’ or a ‘Y’ pattern where they alternate between fixating between the eyes and the mouth when they perceive basic emotions (Barabanschikov, 2015; Galambos et al., 2018; William & Henderson, 2007; see Figure 29).

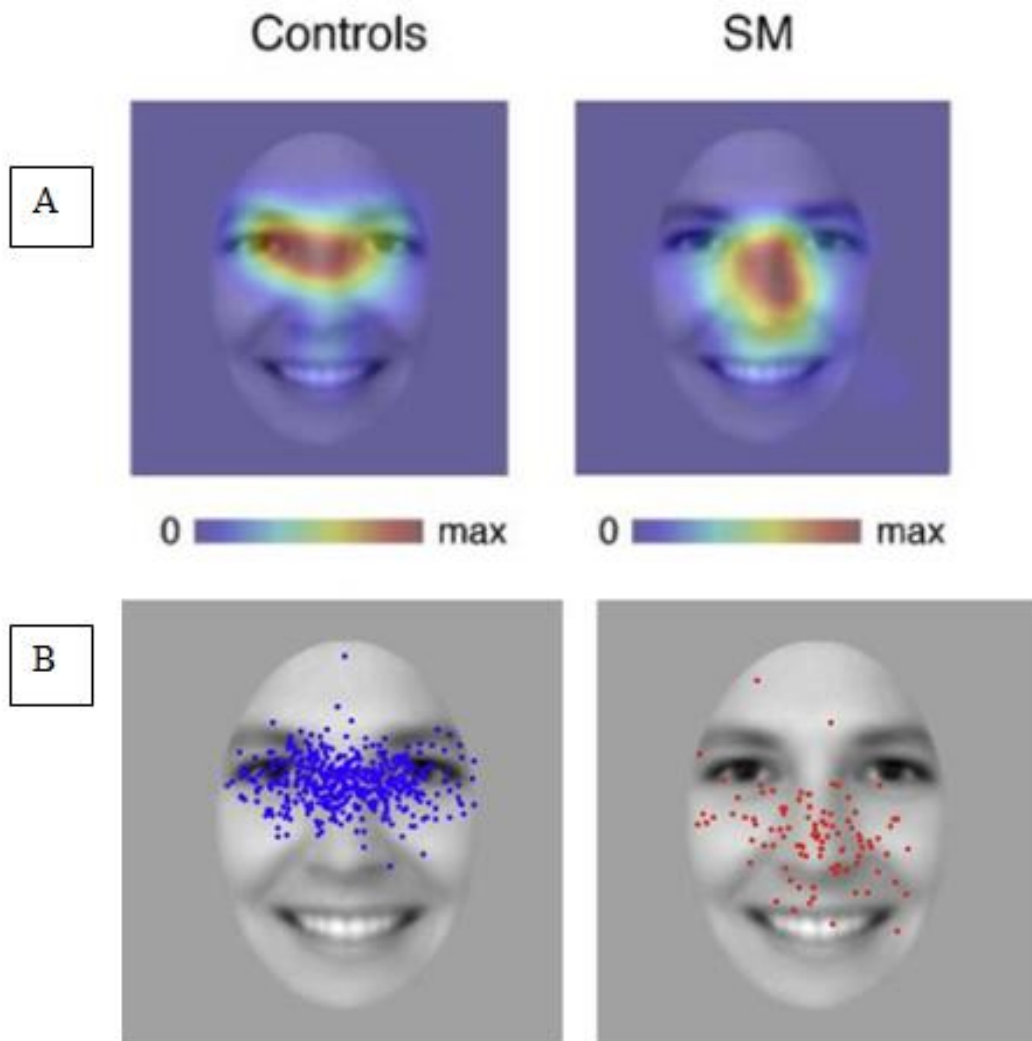




**Figure 29. Example of the typical ‘T’ (image A) and ‘Y’ (image B) fixation patterns displayed by humans when deciphering mood state (reproduced from Barabanschikov, 2015, p.105).**

Ocular fixation patterns appear to differ in response to the six basic emotions (Barabanschikov, 2015; Pérez-Moreno et al., 2016; Schurgin et al., 2014; Smith, Cottrell, Gosselin & Schyns, 2005). For example, Calvo, Fernández-Marín, Gutiérrez-García and Lundqvist (2018) explored the visual strategies of observers during the identification of dynamic facial expressions. Findings showed that specific visual scan-path profiles characterised the different facial expressions. Angry and sad faces drew more attention to the eyes; happy faces produced more fixations to the mouth, while the nose and cheek regions were fixated upon during the viewing of disgusted faces. Fearful and surprised faces elicited a more equal pattern of fixations across eye and mouth regions. Calvo et al., (2018) concluded that different ocular fixation patterns represented a selective attention pattern to expression-specific diagnostic face regions. Further research is needed to explore whether the six basic emotions do produce different ocular scanning patterns.

In line with Hayhoe and Ballard's (2005) 'just in time' theory, typical adults fixate on the eye region of a static image during a passive viewing task, whereas damage to the brain, either through injury, abnormal development, or disease, appears to disrupt this pattern (Figure 30). Aberrant gaze processing has been associated with neuro-social-developmental disorders such as autism and schizophrenia. For example, autistic individual's saccadic exploration of faces is reported as disorganised, incoherent and variable, with less fixation on the eye region and more on peripheral (and uninformative) aspects of the visual scene compared to controls (Black et al., 2017; Dalton et al., 2005; Klin et al., 2002; Wang et al., 2015). Individuals with schizophrenia have been reported to display fewer and longer fixations compared to matched controls (Bortolon, Capdevielle, Salesse & Raffard, 2016; Delerue, Laprévotte, Verfaillie & Boucart, 2010; Simpson, Pinkham, Kelsven & Sasson, 2013) as well as reduced attention to salient facial features and more attention to context irrelevant areas (e.g. forehead) during passive viewing, as compared to non-schizophrenic participants (Bekele, Bian, Peterman, Park & Sarkar, 2017; Bortolon et al., 2016; Delerue et al., 2010).



**Figure 30. Heat map (A) and first fixation count bee swarm (B) during a gender identification task and free passive viewing for controls and SM, a brain-damaged patient (reproduced from Kennedy & Adolphs, 2011, p. 11-12). (A) Mean fixation count heat map displaying fixation location for controls and SM, a brain-damaged participant, on a gender identification task and a passive viewing task. Zero fixations = blue and maximal fixations = red. While controls fixate on the eyes more, SM exhibits more fixations on the nose and appears to avoid the eyes. (B) First fixation count locations for controls ( $n= 500$ ) and SM, (100 trials), during a gender identification task and a passive viewing task. Each dot signifies a single fixation. While controls exhibit attention to the eye region, SM's appears to display less attention to the eye region and fixations are disparate and more focused on the nose region.**

Antoniades and Kennard (2015) reviewed the literature on abnormal eye scan patterns often associated with numerous neurodegenerative diseases, including Alzheimer's disease (prolonged saccade latencies, reduced peak velocities, disorganized visual scanning), frontotemporal dementia (reduced inhibition on the anti-saccade task), Huntington's disease (impairments in vertical and horizontal saccades, slowed saccades), and Parkinson's disease (saccadic hypometria, when the saccade is not accurate and misses the target). One explanation for these eye movement abnormalities is that all of the disorders mentioned above involve some component of frontal lobe dysfunction (Cummings & Miller, 2007). It could be that these conditions produce motor deficits as they usually affect motor regions in the brain.

Furthermore, as discussed in chapter one and two, research from animal models, imaging data, and behavioural findings from studies with brain-damaged participant establish the integral role of the frontal lobes for human social cognition (Adolfi et al., 2017; Wittmann et al., 2018). Consequently, contemporary research suggests an integrative relationship between eye movements, social cognition, and the frontal lobes (Funahashi, 2014; Wolf, Philippi, Motzkin, Baskaya & Koenigs, 2014). Abnormal eye scan patterns are associated with low social functioning and high social disability in schizophrenic and autistic individuals (Bo, Li, Jiang, Wang & Wang, 2018; Klin et al., 2002), as well as impairments in deciphering mental states (Grynszpan & Nadel, 2015; Itier & Batty, 2009). Although caution must be taken as the majority of evidence is from correlational analyses and, therefore, cause and effect cannot be directly inferred, these effects are more than likely due to the abnormality of eye scan patterns, more precisely; decreased fixation numbers in areas of interest pertinent for the task at hand.

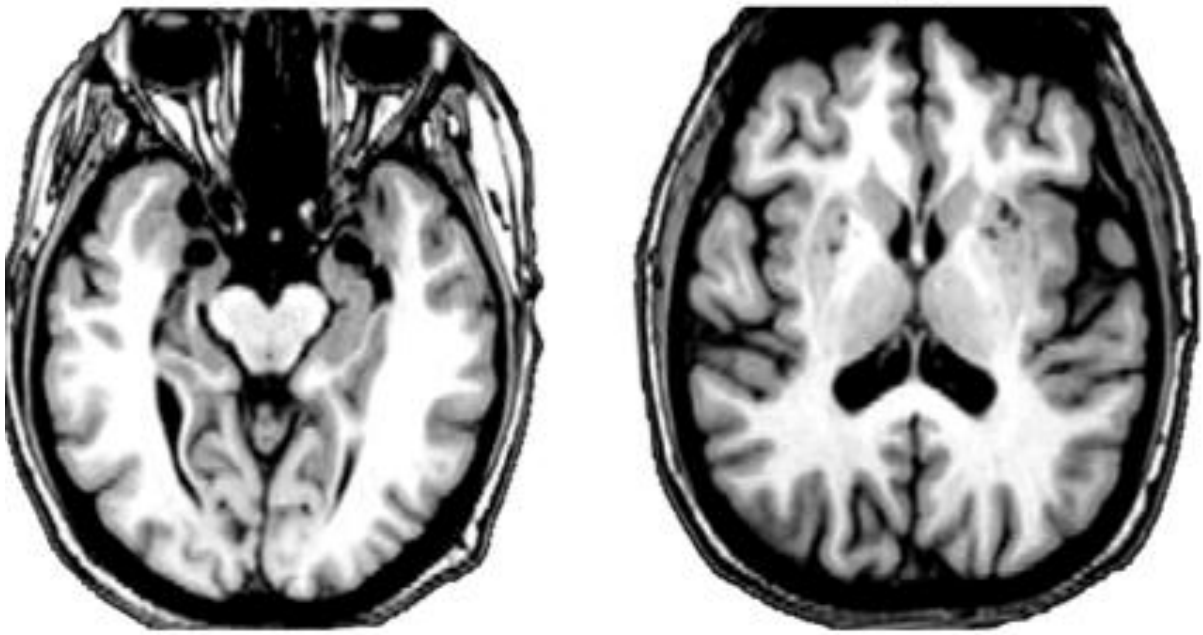
The effect of TBI on basic human vision is a well-studied area. Recently, Armstrong (2018) published a review paper on post-TBI visual problems including disruptions in; visual acuity, photophobia, colour vision, stereopsis, visual field loss, papillary response, and eye movements.

Eye movement impairments, recorded via eye-tracking technology, are documented in approximately 90% of concussion or blast injury cases with conjugate eye movements appearing to be the most affected function (Samadani et al., 2015). Data from fMRI exploring binocular saccades and vergence eye movements during a computerised circle/cross-task revealed that control participants displayed bilateral activation of the SC and oculomotor/abducens nuclei, while signals from TBI participants was significantly reduced in comparison (Tyler, Likova, Mineff & Nicholas, 2015).

In animal work, Evanson, Guillaume-Correa, Herman and Goodman (2018) investigated why some adult mice displayed motor and memory impairments after TBI without any visible brain damage on MRI and diffusion tensor imaging. Fluoro-Jade B and silver degeneration staining showed axonal neurodegeneration in the optic tract, the LGN, and the SC. Interestingly, this damage was not detectable 24 hours after the injury but was present seven days after the injury. These brain regions are critical for normal eye movement function (Erskine & Herrera, 2014). The mouse-model suggested that TBI causes axonal degeneration in the optic tract, LGN, and SC, with related neuroinflammation and astrogliosis that develops several days after the injury, potentially impacting on eye movement generation and control. Despite the extensive body of research documenting damage to the oculomotor systems and visual impairments after TBI, there is a paucity of research investigating the initial visual attention and visual strategies underpinning facial affect recognition (see review below).

Mancuso et al., (2015) investigated the theory that emotion identification impairments experienced by TBI and schizophrenic individuals may be secondary to impairments in attention and/or executive function. The authors used eye-tracking technology to explore the visual strategies of 24 severe TBI participants, 21 schizophrenic participants, and 38 control participants when viewing different facial expressions. The group of individuals with schizophrenia displayed abnormal eye scan patterns compared to controls, but no differences

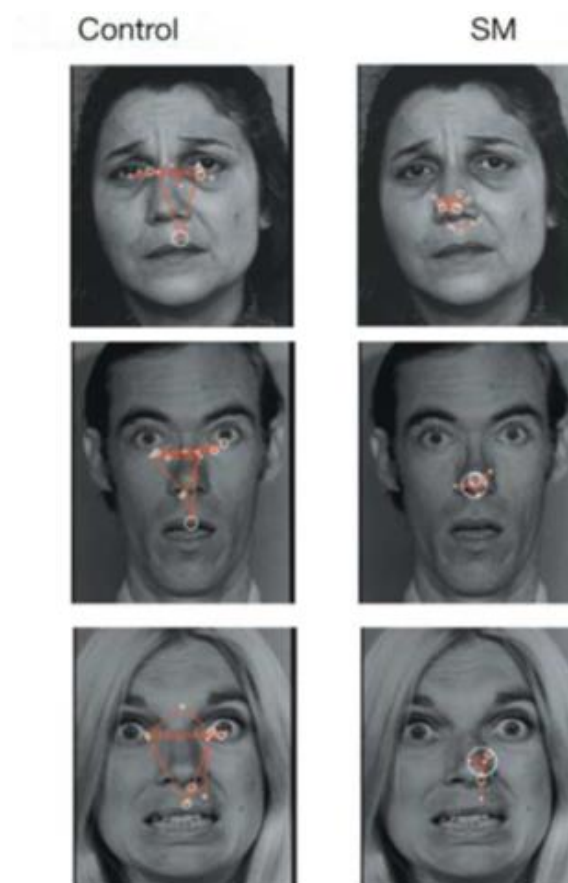
were found between the TBI and control group. There was a significant relationship for the group affected by TBI between emotion recognition and attentional function (measured with the Trail Making task part-B; Army Individual Test Battery, 1944). The authors did not include details of effect sizes so the magnitude of the difference between the TBI and control group could not be assessed. Exploring effect sizes, rather than alpha levels, can sometimes yield interesting findings as they describe ‘how much the groups are affected’ rather than simply ‘are the groups different’ (Sullivan & Feinn, 2012). The findings led the authors to propose a tentative hypothesis that attention deficits after TBI may impede emotion recognition, while low-level visual abnormalities appear to account for the emotion recognition deficits in the group with schizophrenia. One possible confounding variable in the Mancuso et al., (2015) study was the effect of the prescribed antipsychotic medication. All 21 individuals in the schizophrenic cohort were taking antipsychotic medications which are known to sedate the central nervous system (Miller, 2004). It would be difficult to disentangle the disease and medication effects on oculomotor differences between the schizophrenic and control group and raises the caveat of ascribing the performance differences between the groups to illness effects only (Reilly, Lencer, Bishop, Keedy & Sweeney, 2008). Furthermore, the design of Mancuso et al.’s., study (2015) included a non-speeded task (i.e. five-second fixed presentation of facial expressions), meaning their findings cannot be used to explain deficits in dynamic processing. In other work, Adolphs et al., (2005) reported the case of SM, a 38-year-old female who suffered from Urbach–Wiethe disease, a rare autosomal recessive disorder associated with calcification of brain tissue (Hortensius et al., 2017). SM had bilateral lesions to the nuclei of the amygdala, including a small section of the adjacent entorhinal cortex, but no damage to other subcortical and cortical structures (Figure 31).



**Figure 31. An MRI scan of SM's brain (reproduced from Feinstein, Adolphs, Damasio & Tranel, 2011, online supplementary material, p.5). Axial slices depicting focal bilateral amygdala lesions caused by the calcium deposits due to lipoid proteinosis associated with Urbach-Wiethe disease as well as circumscribed damage to white matter around the amygdala and the anterior entorhinal cortex.**

SM displayed impairments in processing emotional and social information, particularly in terms of fearful facial expressions compared to control participants. Interestingly, SM could recognise fear from visual scenes and tone of voice, indicating that the deficit was selectively visual. Using eye-tracking technology, Adolphs et al., (2005) identified that SM had abnormal eye scan patterns compared to controls (Figure 32). Adolphs et al., (2005) used the bubbles technique (a method designed to solve the problem of finding the most informative region in a complex search; Gosselin & Schyns, 2001) and interactively adjusted the number of bubbles on the face while participants tried to identify if the face was displaying fear or happiness. On average, the control group needed 16.5 bubbles to be removed before identifying the emotion while SM required, on average, 30.8 bubbles. Eye-tracking data indicated that while control participants used the eye region of the face to discriminate between happy and fearful faces,

SM failed to make spontaneous fixations to the eye region during the free viewing of the face. The selective impairment in identifying fear indicated that the eyes are the most critical facial feature for recognising fear. SM was able to identify happy faces, possibly because she showed normal eye scan patterns for the mouth region. SM may have used compensatory information from other areas of the face, outside of the eye region, when processing the majority of emotions, but this was unsuccessful or insufficient during the fear condition. Interestingly, SM's recognition of fearful faces matched controls when directed to look at the eyes. When the cues ceased, SM reverted to excluding the eye region during her natural eye scanning pattern, potentially indicating dissociation between voluntary and reflexive saccades, with voluntary saccades remaining intact post-amygdala damage.



**Figure 32. Saccades (red lines) and fixations (white circles) exhibited by one control participant and SM during an emotion identification task (reproduced from Adolphs et al., 2005, p.70). SM displayed no fixations to the eyes during a free-view emotion recognition task.**



Adolphs et al., (2005) also administered a control task using the same faces and the bubbles technique in which participants were asked to name the sex of the face rather than the emotion. During this task, SM's performance was similar to controls; requiring a similar number of bubbles to be removed as well as exhibiting similar eye scanning patterns to controls, including frequent fixations to the eyes. These findings suggest that SM was capable of spontaneous fixations to the eyes during a sex discrimination task but not during an emotion identification task. To further explore the direct importance of the eye region during emotion recognition, the researchers completed a further study with two sets of stimuli; whole faces displaying the six basic emotions and faces with the eyes digitally removed displaying the same emotions. Control participants were significantly poorer at identifying fear during the erased eye condition, while SM showed no change in accuracy. These results suggest that SM failed to make typical use of the eye region when identifying facial expressions. Adolphs et al., (2005) concluded that SM's fear recognition impairment stemmed from a loss of amygdala function guiding the visual system to focus attention on salient information for decoding emotions. It is well documented that the amygdala mediates emotional responses (Diano, Celeghin, Bagnis & Tamietto, 2017; Murray, 2007), but it is also hypothesised that these structures also facilitate aspects of attention (Davis & Whalen, 2001; Peck & Salzman, 2014). The amygdala has multiple feedback projections to the visual cortex (Ousdal, Andreassen, Server & Jensen, 2014). Nevertheless, Adolphs et al., (2005) also conducted the same fear/happiness emotion recognition experiment with 13 unilateral amygdala damaged participants who displayed normal fear identification scores compared to controls. All 13 individuals also showed typical fixation patterns to the eye area of the face compared to controls. These findings seem to imply that SM's impairment cannot be attributed to amygdala damage alone. Researchers have not explored the possibility that Urbach–Wiethe disease produces pathology that is not easily detectable through neuroimaging scans. If Urbach–Wiethe disease causes pathology to

oculomotor regions, then this could induce the abnormal eye scan patterns shown by SM. Little is known about Urbach–Wiethe disease, with less than 500 documented cases since its discovery in 1929, and current literature relies mostly on anecdotal reports and case studies, none of which have employed diffusion tensor imaging to investigate diffuse axonal injury (DAI).

A follow-up study with SM analysed the time course of fixations during a facial expression identification task and reported that SM's first fixation was abnormal compared to control participants (Kennedy & Adolphs, 2010). SM's subsequent fixations were more typical and mirrored that of the control group. In a second experiment, Kennedy and Adolphs (2010) combined eye-tracking and a gaze-contingent face task where only a small area of the face is visible at the centre point of the participants gaze. This task (i.e. only allowing the participant to see the area of the face they are fixating on) eliminates all bottom-up processing (automatic amygdala processing). During this study, SM's results showed typical fixation patterns to the eye region. Taking all of SM's findings into account, it appears that SM's top-down processing was still intact (i.e. that the eyes are the most salient facial feature and the allocation of visual attention under voluntary control is independent of the amygdala), and that abnormalities must be present with the bottom-up attentional system. Interestingly, Spezio, Huang, Castelli & Adolphs (2007) reported that when viewing static stimuli, SM's fixations were typically on the centre of the face (nose) but when SM was part of a dyadic interaction, the majority of fixations were directed around the mouth, even if the other person was not talking. Kennedy and Adolphs (2010) proposed that this may be the result of an expectation of speech which is associated with dynamic social interactions and which is removed from static stimuli.

Wolf et al., (2014) investigated the human ventromedial prefrontal cortex's (VMPFC) role in controlling basic aspects of attention, in particular, the orientation of attention to social and emotional stimuli. The VMPFC has vital connections to the amygdala (Pessoa & Adolphs, 2010), with damage to the amygdala disrupting visual mechanisms underlying facial expression recognition (Adolphs et al., 2005). It is well documented that damage to the VMPFC results in emotion identification impairments (Hiser & Koenigs, 2018; McCormick, Ciaramelli, De Luca & Maguire, 2017). Wolf et al., (2014) recruited three participants who had undergone surgery to resection large anterior cranial fossa meningiomas with vasogenic oedema largely confined to the VMPFC, defined as areas 11, 12, 25 and 32 and the medial portion of area 10 below the level of the genu of the corpus callosum. After surgery (at least three months), participants' eye movements were tracked whilst they identified emotions from the Karolinska Directed Emotional Faces set (Lundqvist, Flykt & Öhman, 1998) and compared against control participants. The findings revealed that VMPFC damage impaired visual attention during the facial expression recognition task and that damage to this region disrupted attention to the eyes, particularly during fear conditions. Interestingly, there were no group differences in total fixations, the distance between fixations, or maximum eccentricity of fixation (the position of the centre of the fovea relative to the point of fixation). The authors concluded that the VMPFC and amygdala may be part of a neural circuit that controls aspects of visual attention to the eye region of the face, possibly by processing the socio-emotional salience of a stimulus. The VMPFC and the amygdala also have connective pathways to the orbital cortex, which projects to the FEF, further supporting this assumption (Barbas, 2000). Wolf et al., (2014) found that the most significant visual strategy abnormality arose during the first second (i.e. first fixation) of face viewing. This signifies the importance of exploring first fixation count and duration, mirroring the findings of Kennedy and Adolphs (2010).

In other work, Oatley, Torsein, Sadeghi and Green (2014) recruited seven male participants with moderate-severe TBI and nine male control participants, administering a simple static facial expression recognition task in two blocks (self-paced vs. fixed-timed) whilst eye movements were recorded. Oatley et al., (2014) reported that proportional data indicated a significant correlation between emotion and task (self-paced vs. fixed-timed) for the eyes and the mouth and a significant interaction between group (TBI vs. controls) and task, with the TBI group fixating on the eyes significantly less than the controls, especially on the self-paced task. These findings demonstrated that controls fixated more on the eye region of fearful faces compared to other emotions, indicating that there may be differences in emotion/task-specific scanning patterns for both control and TBI populations.

Kenrick, Neumann and Conner (2017) also explored the hypothesis that TBI could result in suboptimal visual processing during facial expression recognition. The authors investigated possible visual scanning differences between nine controls and nine moderate to severe TBI participants whilst they viewed the adult faces subset of the Diagnostic Analysis of Nonverbal Accuracy (DANVA; Nowicki & Duke, 1994). Findings showed that the individuals with TBI made fewer fixation counts to the faces compared to controls, with errors on the facial expression task negatively correlated with the total number of fixations overall, as well as fixations specifically on the face (compared to visual background). Errors on the facial expression task were also positively correlated with the per cent of fixation counts and the per cent of fixation duration on the nostrils. This finding suggests that the nose does not provide enough cues to decipher a facial expression. The authors proposed that the group with TBI may have been distracted by information outside of the designated face AOI. The authors also highlighted the need for further investigation into why the group affected by TBI fixated more on the lower nose, a feature which is generally considered minimally relevant for emotion identification.

Turkstra (2005) compared gaze patterns of adults and adolescents with TBI with matched controls whilst engaging in a three-minute conversation. Turkstra (2005) reported that matched controls looked at the face of their conversation partner more compared to the group with TBI, but these results were not statistically significant. However, the within-group variability was significantly greater in the group affected by TBI, suggesting that gaze patterns may be qualitatively rather than quantitatively altered post-TBI.

Douglas et al., (2010) investigated the interpretation of facial expressions and the role of visual scanning with individuals who had sustained TBI. Ten individuals with a severe TBI, at least two years post-injury, and ten age and gender-matched control participants were recruited. All participants viewed static images depicting the six basic emotions and static images of objects. Douglas et al., (2010) reported that TBI participants scored significantly lower on the emotion identification task compared to control participants. Furthermore, TBI participants displayed abnormal scan paths compared to controls, including increased number, duration, and dispersion of fixations when viewing faces. An interesting finding from this study was that TBI participant scan paths were typical for object stimuli, but no further work exists exploring this discrepancy. The authors concluded that impaired emotion identification after TBI may be related to abnormal eye scan patterns. In other research, Vassallo et al., (2010) conducted a case study with patient LY, a male (age unknown) with a severe TBI who displayed significant deficits in emotion identification. Three male control participants were recruited and test stimuli consisted of 18 static images depicting the six basic emotions. Vassallo et al., (2010) reported that control participants tended to fixate on internal facial regions, particularly the eyes, nose, and mouth, while LY displayed a dispersed (hyper scanning) eye scan pattern with significantly more fixations on the peripheral regions of the face, including the hair, ears, forehead, and background of the scene; similar to the findings of Kenrick et al., (2017). Again, the authors concluded that impairments in eye scan patterns may be associated with lower

emotion recognition scores. Vassallo et al., (2011) also investigated the visual scan paths of four male participants with severe TBI and four age and gender-matched controls. A static facial affect recognition task, including the six basic emotions, was used while visual scan paths were recorded. TBI participants displayed significantly poorer accuracy and response times compared to the control group during the facial affect task. With regards to visual scan paths, groups were compared on the number and duration of fixations to internal (eye, nose and mouth) and external (all the remaining) areas of the image. There was no significant difference in the overall number or duration of fixations to internal versus external facial areas between the two groups, but the control group displayed more frequent and longer fixations on the internal facial areas than the group with TBI.

Although there are some differences across different eye-tracking studies, the initial results indicate that neuropathology may alter the generation and/or control of eye scan patterns, warranting further investigation of visual attention and fixational patterns in response to social stimuli. Individuals *perceive* what they *attend* to, or fixate on, so crucially this low-level visual attention data can provide objective indices of whether TBI and control cohorts differ in this respect when viewing social stimuli. Research investigating facial affect impairments after a brain injury usually focus on higher-level functions, such as executive control, yet these functions require accurate visual data extraction, and this is the start point for all subsequent socio-cognitive functions. Few studies have investigated whether these fundamental processes remain intact post-TBI, particularly when viewing naturalistic/dynamic social stimuli.

The above eye-tracking studies are innovative in that they explore visual attention post-TBI but they rely heavily on static stimuli. There are concerns that the majority of social-affective neuroscience studies lack ecological validity as static stimuli (Knox & Douglas, 2009), posed expressions (Juslin, Laukka & Bänziger, 2018; Namba et al., 2018), and experimental settings (Barrett et al., 2011; Ibañez & Manes, 2012) do not correspond to real-world interactions.

Important differences between laboratory experiments and experience of real-world emotions include; lack of dynamics, lack of motion, and absence of depth cues (Alves, 2013; McCamy et al., 2014; McDonald, et al, 2003), as well as the absence of contextual cues (Barrett et al., 2011; Ibañez & Manes, 2012). For over a decade, research has suggested that static and dynamic stimuli are processed by different brain networks (Kilts, Egan, Gideon, Ely & Hoffmann, 2002; McDonald & Saunders, 2005) so this is an important distinction when trying to map structure to function in eye-tracking research.

A compelling study by Blais et al., (2017) explored control participants visual strategies when processing static and dynamic facial expressions using eye-tracking and the bubbles technique. The authors reported that the same facial features (eyes, nose and mouth) were utilised during both conditions to identify emotions, but participants exhibited more fixations to the left eye and mouth during the static condition and more fixations to the centre of the face during the dynamic condition. The authors proposed that one possible explanation for this difference is that humans can process biological motion outside of the fovea and peripherally to the fixation location (Gurnsey, Roddy, Ouhnana, & Troje, 2008; Thompson, Hansen, Hess, & Troje, 2007). Therefore, more fixations to the centre of the face during dynamic conditions may allow an individual to process motion cues from the eyes and mouth without directly fixating on the features, whereas this visual strategy would be insufficient for static stimuli. Blais et al., (2017) findings mirror that of Stoesz and Jakobson (2013) who had previously investigated processing differences between static and dynamic speeded identity and emotion expression judgements. The authors reported disparate processing patterns between static and dynamic displays, with the latter yielding longer responses. Stoesz and Jakobson (2013) stressed the importance of using dynamic displays when assessing face-processing mechanisms.

### **3.6. Summary**

In conclusion, the human oculomotor and visual systems are a complex collection of densely connected brain networks, originating in the photoreceptor cells of the retina and evolving to cortical areas, which work in synergy to direct attention to, and process, salient information in the dynamic world (Goodale & Milner, 1992; Willermain et al., 2014). This process relies on numerous neural processes, such as binocular vision, and is modulated by several eye movements, notably saccades, smooth pursuit, and fixations. The neurological underpinnings of human oculomotor function are extensive, with evidence from animal and human models suggesting that the FEF, SEF, DLPFC, and PEF are paramount for the generation and control of appropriate eye movements (Pierrot-Deseilligny et al., 2004). Damage to these brain areas or associated neural pathways are related to aberrant gaze patterns with marked reductions in fixations to areas of saliency, in the case of facial affect recognition, a noticeable curtailment of fixations to the eyes, nose and mouth (Adolphs et al., 2005, Vassallo et al., 2010). However, the majority of these findings are based on static stimuli which lack ecological validity and create challenges for generalisability. Additionally, recent evidence proposes that static and dynamic stimuli rely on different visual strategies (Blais et al., 2017). Nevertheless, these initial data warrant further exploration of fixation patterns in TBI and control cohorts to determine whether neuropathology produces low-level visual attention impairments which impact on higher-level functions, such as facial affect recognition. The present thesis will investigate fixation patterns about social stimuli, particularly facial expressions, in a group of individuals with TBI compared to matched controls. The next chapter presents the rationale for conducting the current research.



## **4. Chapter four: Rationale for the current research**

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### **4.1. The rationale for the current research**

As previously outlined, TBI is a major cause of death and disability (Dinsmore, 2013; Maas et al., 2008). The high prevalence of TBI creates a serious burden on public health services (Fineberg et al., 2013), as well as physical, emotional, and financial strains on the survivor and their families. Indeed, some authors have documented that social cognition impairments are the greatest barrier to life adjustment and rehabilitation after brain injury (Grattan & Ghahramanlou, 2002; Ponsford & Schönberger, 2010; Yates, 2003). The extant literature illustrates that many TBI survivors and their families report that changes in emotional and social abilities were more disabling and difficult to deal with than cognitive or physical changes (Kelly et al., 2008; Koskinen, 1998; Marsh et al., 2002). Individuals who have sustained a TBI often do not return to full-time employment (Grauwmeijer, Heijenbrok-Kal, Haitisma & Ribbers, 2017) and experience decreased social relationships and material difficulties (Ponsford et al., 2014). This deleterious sequela more than likely exacerbates pre-existing mental health conditions that include depression, anxiety, and substance abuse (van der Horn, Spikman, Jacobs & van der Naalt, 2013; Kreutzer, Seel & Gourley, 2001).

As human beings are social creatures, interpersonal relationships are important for both physical and mental health (Heinrich & Gullone, 2006). Exclusion from or inadequate participation in the social community through core social roles (e.g. having a job, being part of a family or hobbies) can lead to poor self-regulation and poor self-esteem (Siegrist, 2000). Human loneliness is a risk factor for the decline in cognitive function, depression, and social anxiety (Cacioppo & Hawkley, 2009). Social cognition, the skill which enables people to understand the social world (Cassel et al., 2019), is a prerequisite of social integration (Brother, 1990). As outlined in the previous chapters, social cognition impairments after TBI are well

documented (May et al., 2017; McDonald, 2013). It has been acknowledged that the prevailing nature of socioemotional impairments post-TBI makes them one of the most challenging and deliberating of all TBI sequelae (Brooks et al., 1986; Kinsella et al., 1991; Ubukata et al., 2014). It has been noted that social cognition impairments occur on an implicit and automatic level (Barker & Andrade, 2006; Morton & Barker 2010, Barker, Andrade & Romanowski 2004), strongly correlating with frontal lobe pathology (Barker, et al., 2004; Frith and Frith 2001; Siegal and Varley, 2002). Due to the brains position within the skull and the biomechanical and pathophysiological factors associated with TBI, the frontal lobes and the connecting pathways often sustain an injury during trauma (Bigler, 2007). One of the most prevalent impairments associated with frontal lobe damage is facial affect recognition (Drapeau, Gosselin, Peretz & McKerral, 2017; Neumann et al., 2016; Spikman et al, 2013), yet the underlying mechanisms of this deficit are not well understood.

It is estimated that up to 39% of individuals with a moderate-severe TBI could present with significant facial affect identification impairments (Babbage et al., 2011), with experts suggesting that this particular impairment could be one of the main barriers to successful community re-integration (Croker & McDonald, 2005). Nevertheless, formal assessments of facial expression recognition post-TBI are often overlooked in clinical practice (Kelly et al., 2017a, 2017b). Current experimental treatment-outcome research has indicated that rehabilitation programmes can be successful in improving social behaviour post-TBI (Barman, Chatterjee & Bhide, 2016; Flanagan, McDonald and Togher, 1995; Williamson & Isaki, 2015). Indeed, there are positive reports of facial affect recognition interventions post-TBI (Bruhns, 2017; Neumann, Babbage, Zupan & Willer, 2015; Williamson & Isaki, 2015), but more success could be gained if the underlying impairment mechanisms were fully understood, potentially allowing clinicians to target specific deficits rather than resorting to a global intervention approach (Yeates, 2014).

Social perception, that is the ability to detect social stimuli or social cues, is pivotal for social functioning. Social interactions rely on several visual processes, including action observation and the ability to read others' feeling and intentions through body language and facial expressions (Matsumoto et al., 2015). A range of brain networks orchestrates social perception; central to this is the perception network (Bickart, Dickerson & Barrett, 2014). This network is thought to process information rapidly and automatically (Whalen, 2007). Appropriate eye movements are critical for social perception (Gobel et al., 2015; Freeth et al., 2013) and aberrant eye scan patterns are associated with low social adaptation and high social disability (Klin et al., 2002), as well as impairments in deciphering mental states (Grynszpan & Nadel, 2015; Itier & Batty, 2009).

Residing within the frontal lobes are critical eye movement regions that mediate saccades and fixations, namely the FEF, SEF, DLPFC. Intracortical white matter pathways also connect the frontal lobes to other critical eye movement centres within the brain, such as the PEF and the occipital cortex (Stanton, Bruce & Goldberg, 1995; Vernet et al., 2014). These networks often incur some degree of pathology during trauma due to the compressive forces of the skull on soft brain tissue (Bigler, 2013), yet there is a dearth of research exploring the impact this has on social cognition. There are a limited number of studies which have investigated eye scan patterns in response to social stimuli after TBI (Adolphs et al., 2005; Douglas et al., 2010; Mancuso et al., 2015; Vassallo et al., 2010; Vassallo et al., 2011; Wolf et al., 2014), but the methodologies are constrained by small sample sizes and static stimuli. Nevertheless, what these findings underscore is that impairments in social perception after TBI may be associated with the inability to direct attentional resources to salient social information, rather than damage to specific areas (e.g. the face perception network) or aberrant upstream cognitive processing.

Eye movement abnormalities are well established in several disorders associated with frontal lobe dysfunction, notably schizophrenia (Bortolon et al., 2016) and autism (Wang et al., 2015). In fact, aberrant eye scan patterns are used as a classifier and trait marker in both schizophrenia (Benson et al., 2012) and autism (Carette et al., 2019). This information is pertinent for the TBI research field as common behavioural symptoms shared by the conditions (e.g. apathy, lack of motivation, lack of spontaneity, and disinterest in social interactions) might be related with atypical eye movements. For example, Schwab, Würmle, Razavi, Müri and Altorfer (2013) developed a visual peripheral recognition task that required participants to coordinate both eye and head movements. A visual target was first presented in the centre of the screen and then in the periphery and the participant had to decide if the two targets were equal or not. There were two different tasks, a simple colour recognition task and a more challenging Landolt-C orientation task. Two tasks were implemented to explore the hypothesis that tasks of varying difficulty may induce differences between participants with schizophrenia and controls concerning eye-head coordination. There were differences between individuals with schizophrenia and controls where the control group displayed long latencies during the colour task and short latencies during the Landolt task while individuals with schizophrenia had similar latencies across both tasks. The individuals with schizophrenia also displayed more head movements and increased eye-head offsets. As head movements and visual strategies are both used during social interactions to pick up social cues and to display engagement (e.g. joint attention), abnormalities within these systems are bound to have a negative effect of social integration. The researchers suggested that the individuals with schizophrenia may have displayed a specific oculomotoric attentional dysfunction as they demonstrated difficulty in adapting to the two different tasks, compared to the control group. Furthermore, the uneconomic over-performance of head-movements may have been due to poor inhibition, possibly related to alterations in the frontal executive function system. This may indicate that

individuals with schizophrenia have difficulty determining the relevance of stimuli which could extend to social interactions. This hypothesis is supported by a published literature review which concluded that restricted scanning behaviour, often documented in schizophrenic studies, could be linked with the negative symptoms of the condition, specifically in terms of face viewing (Beedie, Clair & Benson, 2011). Research has demonstrated that individuals with TBI frequently display very similar behaviours to the negative symptoms of schizophrenia (e.g. apathy, anhedonia difficulty in determining the relevance of social stimuli – Damasio, Tranel & Damasio, 1990; Worthington & Wood, 2018), so it may be the case that aberrant visual strategies lead to difficulty determining the relevance of social stimuli, but this hypothesis is currently untested. An emerging theory within the autism literature is that abnormal social reward processing may underlie social impairments in autism (Bottini, 2018), and this has been linked to pupillary responses to faces. Sepeta et al., (2012) reported that children with autism did not show typical pupillary sensitivity to gaze direction in response to happy faces compared to typically developed children. The authors proposed that the findings supported the hypothesis that individuals with autism displayed reduced sensitivity to the reward value of social stimuli. This theory has also been supported by subsequent research (Gale, Eikeseth & Klintwall, 2019; Ruta et al., 2017) and could potentially explain why individuals with TBI often appear apathetic and disinterested in the social environment (Worthington & Wood, 2018) (i.e. pupillary sensitivity indicates appeal/attention/preference but if visual strategies are aberrant after TBI then the individuals affected may not be picking up relevant cues/appropriately reacting to cues). Again, this hypothesis is currently untested.

Therefore, the negative symptoms of schizophrenia (e.g. apathy, anhedonia) and the principal characteristics of autism (e.g. finding it hard to understand others, finding it hard to make friends or preferring to be on their own, seeming blunt, rude or not interested in others), are all behaviours which have been mirrored by individuals with TBI. These behaviours may be

mediated by aberrant low-level visual processing abilities, but eye-tracking research within the TBI field is significantly lacking.

From reviewing the existing literature base, it is clear that the mechanisms underpinning social cognition post-TBI are poorly understood. Research suggests that upstream cognitive processes, as well as co-morbid effects and pre-morbid personality traits, could affect social cognition impairments post-TBI. Correlational analyses have reported that poor performance on social cognition tasks is generally not related with cognitive deficits post-TBI (e.g. memory, executive function and attention – Spikman et al., 2012), suggesting that other factors must mediate the social cognition deficits consistently reported in the literature. TBI is often associated with co-morbid conditions such as post-traumatic stress disorder (PTSD; Tanev, Pentel, Kredlow & Charney, 2012), substance abuse (Weil, Corrigan & Karerlina, 2016), psychiatric disorders (Rogers & Read, 2007) and mood disorders (Spitz, Always, Gould & Ponsford, 2017). These post-TBI comorbidities interplay with social cognition deficits, for example, a general impairment in memory may lead to an individual with TBI forgetting key dates resulting in relationship breakdowns. Furthermore, PTSD, mood disorders and other psychiatric disorders may stop an individual with a TBI from leaving their home, harming social relationships and general mental health. Premorbid personality characteristics have also been proposed as a moderator for ‘reserved’ social cognition following TBI. Sela-Kaufman, Rassovsky, Agranov, Levi and Vakil (2013) reported that premorbid personality characteristics provided the most robust moderator of occupational outcome (which could be considered as a good measure of social reintegration). Frank (2011) also proposed that certain premorbid personality traits may have implications regarding the way the individual copes with post-TBI changes. High levels of neuroticism, ineffective problem solving, and avoidance coping have been associated with poor adaption, while characteristics such as optimism and positivity are related to better adaption. Alexithymia is a personality trait found in up to 12% of the general

public (Salminen, Saarijärvi, Aärelä, Toikka & Kauhanen, 1999) and is hypothesised to be a risk factor for several medical and psychiatric disorders associated with affect regulation (Luminet, Bagby & Taylor, 2018). Individuals who experience this ‘organic alexithymia’ are likely to have a lower social reserve if they experienced a TBI compared to individuals who do not have alexithymia. Although the literature suggests that these comorbid factors are not the underpinning mechanisms driving social cognition impairments post-TBI, the factors mentioned above, plus other factors (e.g. employment status, social/culture/economic status), are likely to exacerbate the already deleterious emotional and social sequelae of TBI and partially determine psychosocial outcome post-TBI (Williams et al., 2020).

Existing attempts to elucidate the underlying mechanisms of social cognition post-TBI have, so far, been unsuccessful. Existing research tends to employ imaging techniques with findings indicating a general brain dysfunction, with particular abnormalities within the frontal circuits. However, the specific mechanisms underpinning these impairments are still unknown and findings are univocal. For instance, Neumann, McDonald, West, Keiski and Wand (2016) proposed that post-TBI brain dysfunction may lead to impaired holistic face processing while Rigon, Voss, Turkstra, Mutlu and Duff (2019) hypothesised that facial affect recognition was comprised of subcomponents that depend on distinct neural substrates and that individuals with TBI were impaired in some, but not all, subcomponents. Other schools of thought for explaining the potential mechanisms underpinning impaired social cognition include impaired simulation (McDonald et al., 2011), impaired physiological responsivity and dulled emotional experience (Crocker & McDonald, 2005; Hornak et al., 1996). However, none of these theories presents a complete account for the underlying mechanisms of social cognition deficits post-TBI. One downfall of the pre-existing explanations is that they tend to focus on mid to high-level processing and rarely assess the impact of automatic, low-level visual processing.

Individuals perceive what they attend to, or fixate on, so recording fixations in response to social stimuli can offer objective indices of typical or aberrant visual scanning patterns. If there is variability between the TBI and control groups, then this might underpin the reported upstream social cognition deficits in the TBI literature. Few studies have explored these basic visual processes and the experiments which do have only implemented very basic static stimuli which lack ecological validity. It appears that one of the reasons for this deficiency in understanding the low-level social cognition processing of individuals with TBI is due to a lack of published research. For example, when a targeted literature search was conducted using Scopus, an abstract and citation database, with the terms ‘eye-tracking’ and ‘traumatic brain injury’ only 86 documents were found. When ‘social cognition’ was added to the search terms, only three papers were found. These figures are extremely low in comparison to a similar search when the terms ‘eye-tracking’ and ‘autism’ were inputted, generating 761 documents or ‘eye-tracking’, ‘autism’ and ‘social cognition’ which generated 122 articles. Indeed, while the research fields of autism and schizophrenia have explored, in-depth, visual strategies in response to social stimuli, the TBI research field is lagging. One reason for this may be that higher-level ‘classic’ cognitive impairments have been the focus of investigation following TBI. This is supported by a Scopus review using the terms ‘cognitive impairment’ and ‘traumatic brain injury’ which generated a list of 3,087 articles. It has been established that social cognition is not well understood by experts in the TBI field and that social cognition deficits are rarely ever assessed during clinical practice (Kelly et al., 2017a, 2017b). Commonly, practice-based evidence feeds into research, for instance, clinicians consistently feedback that ‘classic’ cognitive functions are impaired post-TBI. However, if clinicians are not assessing social cognition post-TBI, probably due to a lack of education, assessments and rehabilitation interventions, then this cross-talk between clinicians and researchers is naturally going to hinder the research field.



When referring back to the three stages of social cognition (Ostrom, 1984; Penn et al., 1997), there is a wealth of evidence focusing on stages two and/or three (interpretation of social information and processing of social information) while very little exploration is given to the very initial perception of social cues. This dearth could be associated with the fact that, in the past, eye-tracking technology was limited (not many models available, difficult to use, i.e. chin rest and head restrictions, and varying accuracy) and costly or because other areas of post-TBI impairments appeared more pertinent. As the research field of eye-tracking and social cognition post-TBI is scant, very few efforts have been made to create reliable and valid assessments. Indeed, to the best of the researcher's knowledge, only two dynamic tasks have been validated with TBI populations; TASIT (McDonald et al., 2002) and CAVEAT (Rosenberg et al., 2016), neither of which have been implemented with eye-tracking technology. This also supports the fact that the deficiency in understanding the role of low-level processing in social cognition post-TBI is due to a general paucity of investigation. Hence, the current research, and similar projects, are needed to fill the gap in the literature and to shed light on the deficient understanding of the role of low-level processing in social cognition post TBI.

The innovative aspect of the current research is the implementation of 'real world' dynamic and contextual stimuli. The denouncement of static stimuli in the neuropsychology literature is gaining traction, with scholars arguing that static stimuli lack ecological validity and mundane realism (Blais et al., 2017; Feldman-Barrett, 2011; Ibañez & Manes, 2012; Juslin et al., 2018; Knox & Douglas, 2009; Leigh & Zee, 2016; Namba et al., 2018; Recio, Sommer and Schacht, 2011). There are clear disparities between static laboratory-based social cognition tasks and everyday social interactions. For instance, experimental paradigms often lack motion and depth cues (Alves, 2013; Mccamy et al., 2014; McDonald et al., 2003) as well as the absence of contextual cues (Feldman-Barrett et al., 2011; Ibañez & Manes, 2012). Furthermore, evidence proposes that static and dynamic stimuli rely on different visual strategies (Blais et al., 2017;

Stoesz and Jakobson, 2013). Social interactions demand the tracking of emotions and mental states through complex cues, such as facial expressions, body language, and eye gaze. This dynamic process requires a constant shift in attentional processing. The currently available dynamic social cognition assessments are still flawed in that they include posed expressions (Juslin et al., 2018; Namba, et al., 2018) and are administered in experimental settings (Feldman-Barrett et al., 2011; Ibañez & Manes, 2012). Nevertheless, they offer a vast improvement on static stimuli yet are not as widely implemented in the existing evidence base, particularly within the TBI research field.

#### **4.2. Aims of the thesis**

Comprehensive objectives and hypotheses for each experiment are presented at the beginning of each chapter. In brief, the focus of this thesis was to explore how mechanisms of attention, specifically early visual processes, may modulate social behaviour in a TBI and age and sex-matched control group. Data from typically developed samples are vital for the development of evidence-based interventions and the understanding of social cognition. Consequently, the opening study of this thesis investigated sex differences in social cognition with non-patient groups. One of the central aims of the research was to determine if TBI causes impairments during the first stage of social cognition, that is, the perception of social cues. Few studies have explored these basic visual processes and the experiments which do have only implemented static stimuli. Consequently, this thesis employed a combination of static and dynamic socioemotional assessments. Moreover, it is scarce that research studies explore more than one construct of social cognition. Therefore, a second aim of the current project was to investigate visual scanning differences in response to distinct domains of social cognition (e.g. thoughts, intentions, and emotions).

A further aim of the current research programme was to conduct a fine-grained analysis of the Movie for the Assessment of Social Cognition (MASC – Dziobek et al., 2006) using its multidimensional subcategories (e.g. emotions, thought, and intentions). The designers of the MASC had eluded to these subcategories in their original publication but no further details were provided (e.g. which construct each clip measured). Through personal email communication with the test author (Dziobek), the present research was able to divide the different social constructs and assign the relevant video clips. This process also involved two researchers separately validating the categories and associated videos. To test the process and to determine if the subcategories were sensitive enough to discriminate the separate social constructs, the normative MASC study was conducted. To understand social cognition deficits following TBI, the research field must first have a sound comprehension of social cognition in non-TBI individuals, including sex differences. For that reason, the MASC normative study was conducted to (i) allow the research team to explore the sensitivity of the MASC subcategories and (ii) to feed into the knowledge base of normative social cognition and to explore behavioural and cognitive (eye-tracking) sex differences in response to the MASC.

One aspiration of this research was to disseminate research findings to health care professionals in the hope that it will raise awareness of social cognition impairments after TBI. Recent large scale online studies have demonstrated that clinicians and other health care professionals rarely or sometimes never formally assess social cognition deficits after TBI (Kelly et al., 2017a, 2017b). This finding is concerning taking into account the major personal, social, and economic repercussions social cognition impairments give rise to post-TBI.

## **5. Chapter five: Methodology review**

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### **5.1. Chapter overview**

This chapter reviews the specific neuropsychological tests selected to assess social cognition in the current study and a detailed description of the eye-tracking equipment, eye-tracking data preparation and the designation of areas of interest (AOI).

### **5.2. Methodology**

The present programme of research included a neuropsychological test battery and eye-tracking technology to investigate social cognition after TBI. Standardized neuropsychological assessments were used to compare expected levels of functioning on behavioural and demographic tests between the TBI and matched control groups. Eye-tracking offers a non-invasive and quantitative method to investigate how attention is distributed across a visual scene. The rich data collected during eye-tracking tasks allows researchers to evaluate continuous attentional processes that cannot be measured through standard neuropsychological assessments. Atypical eye movements can often provide insight and diagnostic clues into neurological disorders and the method is frequently used with autistic and schizophrenic cohorts (Murias et al., 2018; Porffy et al., 2018; Raffard et al., 2016; Wang et al., 2015). Despite this knowledge, there is a paucity of research implementing eye-tracking technology with TBI populations, despite evidence that eye movements are disrupted after TBI (Caplan et al., 2015; Samadani et al., 2015; Ventura et al., 2014).

Some studies have measured visual fixations after TBI using static stimuli that have been criticised on the grounds of poor ecological validity and mundane realism (e.g. Knox & Douglas, 2009; Leigh & Zee, 2016). Mindful of this, the current research project used more recently developed static stimuli as well as innovative dynamic stimuli to address some of the

criticisms of current emotion perception studies. This research explored the underpinning mechanisms of social cognition by integrating behavioural and cognitive measures (neuropsychological tests) of static and dynamic social processing, in conjunction with eye-tracking with TBI and control participants to contribute to the knowledge of why social deficits commonly occur after brain injury (Spikman, Timmerman, Milders, Veenstra & van der Naalt, 2012).

### **5.3. Selection of test measures and materials**

Figure 33 outlines the selected test battery for the current research with each test being discussed in more detail in the chapter.

### Screening Measures

- Health of the Nation Outcome Scales (HoNOS; Wing, Curtis & Beevor, 1996)  
Risk assessment.
- Hospital Anxiety Depression Scale (HASS; Zigmond & Snaith, 1983)  
Measure of anxiety and depression levels.
- Michigan Alcohol Screening Test (Selzer, 1971)  
Measure of alcohol usage.
- Drugs Abuse Screening Test (DAST; Skinner 1982)  
Measure of drug dependence.
- Visual Object Spatial Perception battery (VOSP; Warrington & James, 1991)  
Assessment of object and space perception whilst curtailing involvement of alternative cognitive abilities
- Cortical Visual Screening Test (CORVIST; Warrington, Plant & James, 2001)  
Tests the early visual processing in the higher visual areas of the brain

### Behavioural Data

- MASC (Dziobek et al., 2006)
- TASIT (McDonald et al., 2003)
- ADFES (van der Schalk et al., 2012)
- IAPS (Lang et al., 2008)

### Demographic Information

- Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999)
- Measure of intelligence.

### Eye-Tracking Data

- Movie for the Assessment of Social Cognition (MASC; Dziobek et al., 2006)
- Dynamic task to assess various aspects of social cognition.
- The Assessment of Social Inference Test (TASIT; McDonald, Flanagan & Kinch, 2003)
- Dynamic task to assess various aspects of social cognition
- Amsterdam Dynamic Facial Expression Set (ADFES; van der Schalk et al., 2013)
- Static and dynamic tests to evaluate facial expression recognition.
- International Affective Picture System (IAPS; Lang, Bradley & Cuthber, 2008)
- Recognition of humans, animals and objects.

**Figure 33. Summary of the current test battery.**

## **5.4. Screening measures**

The present research included eye-tracking methodology so participants needed to display no basic visual or perceptual impairments. Both TBI and control participants were administered the Visual Object Spatial Perception Battery (VOSP; Warrington & James, 1991) and the Cortical Vision Screening Test (CORVIST; James, Plant & Warrington, 2001) to measure low-level visual perception and acuity. The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was included as a mood state measure as high levels of anxiety and depression have been linked to abnormal eye scanning patterns (Carvalho et al., 2015; Liang, Tsai & Hsu, 2017). The Michigan Alcoholism Screening Test (MAST; Selzer, 1971), and the Drug Abuse Screening Test (DAST; Skinner, 1982) were also administered as alcohol and other drugs have been related to dysfunctional eye scan patterns (Costa & Bauer, 1998; Peragallo, Biousse & Newman, 2013). Additionally, the collaborating consultant psychologists conducted a risk assessment to measure the TBI participant's suitability for inclusion in the study.

### **5.4.1. The Health of the Nation Outcome Scales**

Participants with TBI had to present as no or extremely low risk to others and themselves to be recruited to the study. The justification for this choice was based on the fact that the PhD researcher would be lone working in an isolated laboratory. The two collaborating consultants screened potential participants for risk, on the researcher's behalf, using the Health of the Nation Outcome Scale (HoNOS; Wing, Curtis and Beevor, 1996). Clinicians, rather than the researcher, assessed risk as they were considered an expert in this field, having received professional training and utilising this skill on a day-by-day basis. The PhD researcher had no experience or training in assessing risk. The HoNOS measures the health and social competence of individuals with suspected mental health conditions. The measure includes 12

items which evaluate behaviour, deficits, symptoms and social competence. The HoNOS is well known for its easy administration, sensitivity, reliability and validity (Pirkis et al., 2005).

The 12 items assess a dynamic range of behaviour and are outlined below:

- 1) Overactive, aggressive, disruptive or agitated behaviour
- 2) Non-accidental self-injury
- 3) Problem drinking or drug-taking
- 4) Cognitive problems
- 5) Physical illness or disability problems
- 6) Problems associated with hallucinations and delusions
- 7) Problems with depressed mood
- 8) Other mental and behavioural problems
- 9) Problems with relationships
- 10) Problems with activities of daily living
- 11) Problems with living conditions
- 12) Problems with occupation and activities

Each item is scored on a five-point scale; zero for no problems, one for a minor problem requiring no action, two for a mild problem but present, three for a moderately-severe problem and four for a severe-very severe problem.

#### **5.4.2. Hospital Anxiety and Depression Scale**

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is a self-administered questionnaire and includes questions such as 'I feel tense or wound up' and 'I still enjoy the things I used to enjoy' (Appendix 1). The HADS is quick and easy to administer and takes approximately five minutes to complete. Participants are asked to read each question



carefully and to underline the response which comes closest to how they have been feeling in the past week. There are four responses to each question and each response is assigned a number from 0-3. So for example, the question 'I feel tense or wound up' can be answered as; (0) not at all, (1) from time to time, occasionally, (2) a lot of the time, (3) most of the time. There are a total of 14 questions, seven of which relate to anxiety and seven to depression. The questionnaire is scored using a separate point system for anxiety and depression with a maximum score of 21 for each condition or a total overall maximum score of 42. The HADS classification system is based on the overall total score for each of the subscales and a score of 0-7 indicates normal mood, 8-10 indicates mild mood disorder, 11-15 indicates moderate mood disorder and 16-21 indicates severe mood disorder (Snaith, 2003).

A wide range of research findings has shown that the HADS is a reliable and valid tool for the screening of depression and anxiety. Martin and Thompson (1999) investigated the internal reliability of the HADS and concluded that the measure has good internal reliability ( $\alpha = 0.85$ ). This result was supported by a meta-analysis conducted by Bjellanda, Dahlb, Haugc and Neckelmannnd (2002) who reported a high mean internal consistency for both the HADS anxiety scale ( $\alpha = 0.83$ ) and the HADS depression scale ( $\alpha = 0.82$ ) and good concurrent validity with correlations between 0.60-0.80 for both anxiety and depression subscales. The HADS has been widely used in TBI research and is a popular instrument to measure mood state (Dahm, Wong & Ponsford, 2013; Gregório, Gould, Spitz, van Heugten & Ponsford, 2013; McKenzie, Downing & Ponsford, 2018).

It is well documented that mood disorders negatively impact on emotion perception and that individuals with diagnosed depressive, anxiety or aggression syndromes often exhibit a negative bias towards emotive stimuli (Kaletsch et al., 2014; Niedenthal, Brauer, Halberstadt & Innes-Ker, 2001; Penton-Voak, Munafó & Looi, 2017; Vaskinn et al., 2017). Mood disorders

are also associated with abnormal eye scan patterns (Gamble & Rapee, 2010; Hills, Marquardt, Young & Goodenough, 2017; Horley, Williams, Gonsalvez & Gordon, 2003; Kellough, Beevers, Ellis & Wells, 2008; Mogg, Millar and Bradley, 2000; Wu, Pu, Allen & Pauli, 2012), including a bias towards threatening stimuli (e.g. angry compared to neutral faces (Gamble et al., 2010; Mogg et al., 2000), and avoidance of looking at facial features (Horley et al., 2003). In the present study, TBI and control participants completed the HADs to control for possible confounding variables related to anxiety and depression. As suggested by Snaith (2003) sub-scale scores of 16 or above indicated severe anxiety or depressive disorder and scores above this would have been accounted for during analyses, however, none of the TBI nor control group exceeded the established screening criteria for the HADs.

#### **5.4.3. Michigan Alcoholism Screening Test**

The Michigan Alcoholism Screening Test (MAST; Selzer, 1971) is a self-administered questionnaire and includes 24 questions such as ‘Do you feel you are a normal drinker? (“Normal” is defined as drinking as much or less than most other people)’, ‘Can you stop drinking without a struggle after one or two drinks?’ and ‘Do you ever feel guilty about your drinking?’ (Appendix 2). Participants answered yes or no for all 24 questions. The questionnaire is scored using a point system across the 24 questions (Appendix 2). Originally, Selzer (1971) suggested a total score of 5 or above indicated alcoholism. However, the cut-off point has been adjusted so a score of 0-4 indicates no alcohol dependency; 4-6 may suggest some alcohol dependency and a score of 7 signals alcohol dependency (Hedlund & Vieweg, 1984; Selzer, Yinokur, & Rooijen, 1975). The present study adopted the more conservative cut-off score of  $\geq 7$  in line with Hedlund et al., (1984) and Selzer et al., (1975). The MAST questionnaire takes approximately ten minutes to complete.

The MAST is considered the 'gold standard' of alcohol screening tests in medical and research settings (Laux, Newman & Brown, 2004). The measure is widely deemed as valid (Hirata, Almeida, Funari & Klein, 2001; Storgaard, Nielsen & Gluud, 1994), and reliable, with high internal consistency (Gibbs, 1983;  $\alpha = 0.87$ ; Laux et al., 2004  $\alpha = 0.88$ ), and high test-retest reliability (Zung, 1982; 0.86-0.97). A recent systematic review confirmed the robust psychometric properties of the MAST including good internal consistency, test-retest reliability, and internal and external validity (Minnich, Erford, Bardhoshi & Atalay, 2018). The test is widely implemented in various psychological and medical research settings, for instance, with PTSD (Dahlgren et al., 2018), and ADHD individuals (Giordano et al., 2017) and TBI cohorts (McDonald et al., 2017; McDonald et al., 2019).

Alcohol dependency has been associated with emotion perception abnormalities (Kopera et al., 2015; Monnot, Nixon, Lovallo & Ross, 2001; Townshend & Duka, 2003) as well as affecting eye movements (Roche & King, 2010). Harvey (2014) found that participants under the influence of alcohol displayed reduced visual scanning during face encoding and also fixated more on the nose compared to the eyes. Taking these findings into consideration, as well as evidence indicating that TBI might be linked with an increased risk of developing alcohol dependency (Bjork & Grant, 2009; Pagulayan, Temkin, Machamer & Dikmen, 2016; Weil & Karelina, 2017), all TBI participants were asked to complete the MAST. None of the individuals with TBI exceeded the MAST cut-off limit.

#### **5.4.4. Drug Abuse Screening Test**

The Drug Abuse Screening Test (DAST; Skinner, 1982) is a self-administered questionnaire and includes 28 questions such as 'Have you used drugs other than those required for medical reasons?' (Appendix 3). Participants either answer yes or no to each question. One point is assigned for each 'yes' answer excluding question numbers 4, 5 and 7 when a 'no' answer is

assigned 1 point. A score of 12 or over indicates drug dependence. The questionnaire takes approximately ten minutes to complete.

The DAST is a reliable measure of substance abuse and a review of the DAST by Yudko, Lozhkina and Fouts (2007) reported that internal consistency scores ranged from  $\alpha=0.92 - 0.94$ . This supports Skinner's (1982) high internal consistency score of  $\alpha=0.92$  with 223 participants who had previously been treated for drug and alcohol abuse problems. El-Bassel, et al., (1997) measured the test-retest reliability of the DAST over two weeks and reported a high correlation coefficient of 0.85. Staley and El-Guebaly (1990) tested the DAST with 250 clinical participants and reported that the measure had significantly high diagnostic validity (89%) in classifying participants who matched the DSM-III substance abuse diagnoses. Also, Giguère and Potvin (2016) administered the DAST to 912 psychiatric patients in an emergency setting and reported an internal consistency  $\alpha= 0.88$  and a test-retest reliability correlation coefficient of 0.86, concluding that the DAST has excellent psychometric properties in psychiatric populations.

Drug abuse has been linked to impairments in social cognition, including deficits in emotion perception, emotional empathy, mental perspective taking, increased criminal behaviour and reduced social networks (Kemmis, Hall, Kingston & Morgan, 2007; Preller et al., 2014). This study screened all TBI participants for high levels of drug dependency ( $\geq 12$ ) as research has documented a high incidence of illicit drug taking pre and post-TBI (Bombardier, Rimmele & Zintel, 2002; Corrigan, Lamb-Heart & Rust, 1995). None of the individuals with TBI exceeded the cut-off limit for the DAST.

#### **5.4.5. Visual Object and Space Perception Battery**

The Visual Object and Space Perception Battery (VOSP; Warrington and James, 1991) was administered to all TBI and control participants to measure possible low-level visual

impairments. The measure comprises eight sub-tests assessing object and space perception (Table 1). Four of the sub-tests measure visual object perception abilities and include incomplete letters, silhouettes, object decision and progressive silhouettes. The remaining four sub-sets measure visual space perception abilities and include dot counting, position discrimination, number location and cube analysis. The VOSP includes a measure of visual sensory efficiency, the shape detection test, to ensure that participants have efficient visual ability to undertake the VOSP. During this task, the participant is required to detect if there is an 'X' embedded within 20 separate pattern designs. One point is awarded for each correct answer. If participants fail to detect 15 or more of the 'X' Figures then they are considered ineligible to complete the rest of the test. All sub-tests are untimed and administered at a pace suitable for the participant. The VOSP was developed to assess the group affected by TBI and it has been successfully implemented within the TBI population (Miotto, et al., 2010; Summers, 2006). To date, it appears that no published studies have explored the psychometric properties of the VOSP so the reliability and validity of the assessment is unknown. The test usually takes approximately 15-20 minutes to complete. None of the participants, either the group of individuals with TBI or matched controls, exceeded the VOSP cut-off limit.

#### **5.4.6. Cortical Vision Screening Test**

The Cortical Vision Screening Test (CORVIST; Warrington, Plant and James, 2001) was used in conjunction with the VOSP to assess visual acuity. The CORVIST assesses early visual processing in higher visual brain areas. When used in conjunction with the VOSP, the CORVIST offers a comprehensive measure of visual perception (James et al., 2001; Yong, Warren, Warrington & Crutch, 2013). There are ten sub-tests including symbol acuity, shape discrimination, size discrimination, shape detection, hue discrimination, scattered dot counting, fragmented numbers, word reading, crowding and face perception (Table 2). The ten sub-tests

assess different features of early visual processing controlled by various cortical regions. The CORVIST has been successfully implemented with individuals who have brain damage as a consequence of degenerative diseases although reliability and validity indices are not known (Adlington, Laws and Gale, 2009; Charles & Hillis, 2005). None of the participants, either the group of individuals with TBI or matched controls, exceeded the CORVIST cut-off limit.

Table 1. Descriptions of the VOSP subtests.

<b>VOSP</b>	<b>Description</b>
<b><i>Object Perception Subtest</i></b>	
Incomplete Letters	During this test, the participant is required to view and identify 20 letters which are incomplete. One point is awarded for each correct answer.
Silhouettes	The silhouettes task involves participants viewing and identifying shadows of fifteen animals and fifteen everyday objects. The test ascends in the level of difficulty with five consecutive errors resulting in a cessation of the test.
Object Decision	Participants are presented with four images; one of these represents a real object while the remaining are distracters. The participant is required to select the real image and to also name the image. There are 20 trials and one point is awarded for each correct response.
Progressive Silhouettes	This task required the participant to view a series of boards which outline the silhouette of an object. The first board shows very little of the picture with the following successive boards providing a more complete picture of the object with the final board presenting a complete image of the object. The participant has to identify the object using the least amount of boards possible. There are two objects, a gun and a trumpet; both objects are displayed over ten boards.
<b><i>Space Perception Subtest</i></b>	
Dot Count	This test requires the participant to count the number of black dots on a white piece of card. There are ten trials and each correct trial is awarded one point.
Position Discrimination	Participants are asked to view two squares presented on a white piece of card. Each square has a black dot either in the exact centre of the square or just off-centre. The participant must select which square contains the exact centre dot. One point is allocated to each correct answer over the ten trials.
Number Location	A white piece of card is split horizontally. In the top half of the page numbers are scattered in a random order. In the bottom half of the page is one single black dot. The black dot corresponds in location to one of the random numbers in the top half of the page. The participant is asked to say which number is represented by the black dot for all ten trials.
Cube Analysis	Depicts a solid structure which has been devised using 3D cubes. The participant needs to count how many cubes have been used to create the solid structure. There are ten trails which consecutively increase in difficulty.

Table 2. Descriptions of the CORVIST subtests.

<b>CORVIST</b>	<b>Description</b>
Symbol Acuity	A simplified acuity test for individuals who have deficits in scanning visual arrays.
Shape Discrimination	To determine if an individual with normal acuity has impairments in shape discrimination.
Size Discrimination	This test complements and extends the Shape Discrimination test and again tests individuals who display normal acuity but may suffer alternative visual deficits.
Shape Detection	This is a more complex shape discrimination task using stimuli which covers a wider area of the visual field.
Hue Discrimination	Participants had to identify the odd hue square out of nine coloured squares.
Scattered Dot Counting	This determines impairments in the localisation of a single point or spatial scanning of the visual field.
Fragmented Numbers	A test to investigate deficits in perceptual identification.
Word Reading	Assesses reading ability and includes words such as scare and biscuit.
Crowding	Determines if there are excessive deficits of acuity when symbols are placed closely together.
Face perception	Although individuals may display normal acuity and discrimination they may still be impaired with more complex stimuli such as processing faces. During this sub-test participants need to determine which face is the oldest and which face is the youngest out of three possible choices.



## 5.5. Demographic measures

### 5.5.1. Wechsler Abbreviated Scale of Intelligence

The effects of participant differences between the TBI and control groups were mitigated through matching on participant variables including IQ, gender and age. The Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999) *was* administered to all participants to gain an estimate of general intellectual (IQ) ability. The WASI contains four subsets; vocabulary, similarities, block design, and matrix reasoning. The vocabulary subset assesses word knowledge, verbal comprehension, and verbal retrieval, with questions such as ‘Tell me what intermittent means’. The similarities subset measures verbal reasoning and concept formation and requires the participant to explain how two words are similar, for instance, ‘In what way is smooth and rough alike?’ The combination of scores from the subsets contributes to the calculation of verbal IQ. The block design subset assesses perception, organisational skills, ability to analyse and complete patterns following a tacit rule, nonverbal concept formation, parallel processing, visuomotor coordination, and the ability to learn from mistakes. This subtest requires participants to create pre-specified patterns by matching several blocks within a specified time-limit with the tasks getting progressively harder. The matrix reasoning subset provides a measure of visual information processing and abstract reasoning by presenting the participant with an incomplete pattern and asking them to select the missing segment from a choice of four. The combination of block design and matrix-reasoning subset scores allows for the calculation of performance IQ. The combination of verbal and performance scores estimates the full-scale IQ score. The test takes approximately 45 minutes to administer.

The WASI has been frequently used with TBI participants (Biederman et al., 2015; McDonald et al., 2017) as it takes less time to administer than alternative IQ measures and saves patient

and clinician time. Van Duijvenbode, Didden, van den Hazel and Engels (2014) reported that the WASI has good reliability ( $r = 0.96$ ) and a strong, positive correlation ( $r = 0.89$ ) with the full the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1981) offering a reliable and valid alternative IQ assessment for clinical and research settings. Furthermore, Canivez, Konold, Collins and Wilson (2009) and Ryan et al., (2003) reported that the WASI also had good construct validity.

## **5.6. Social cognition tasks**

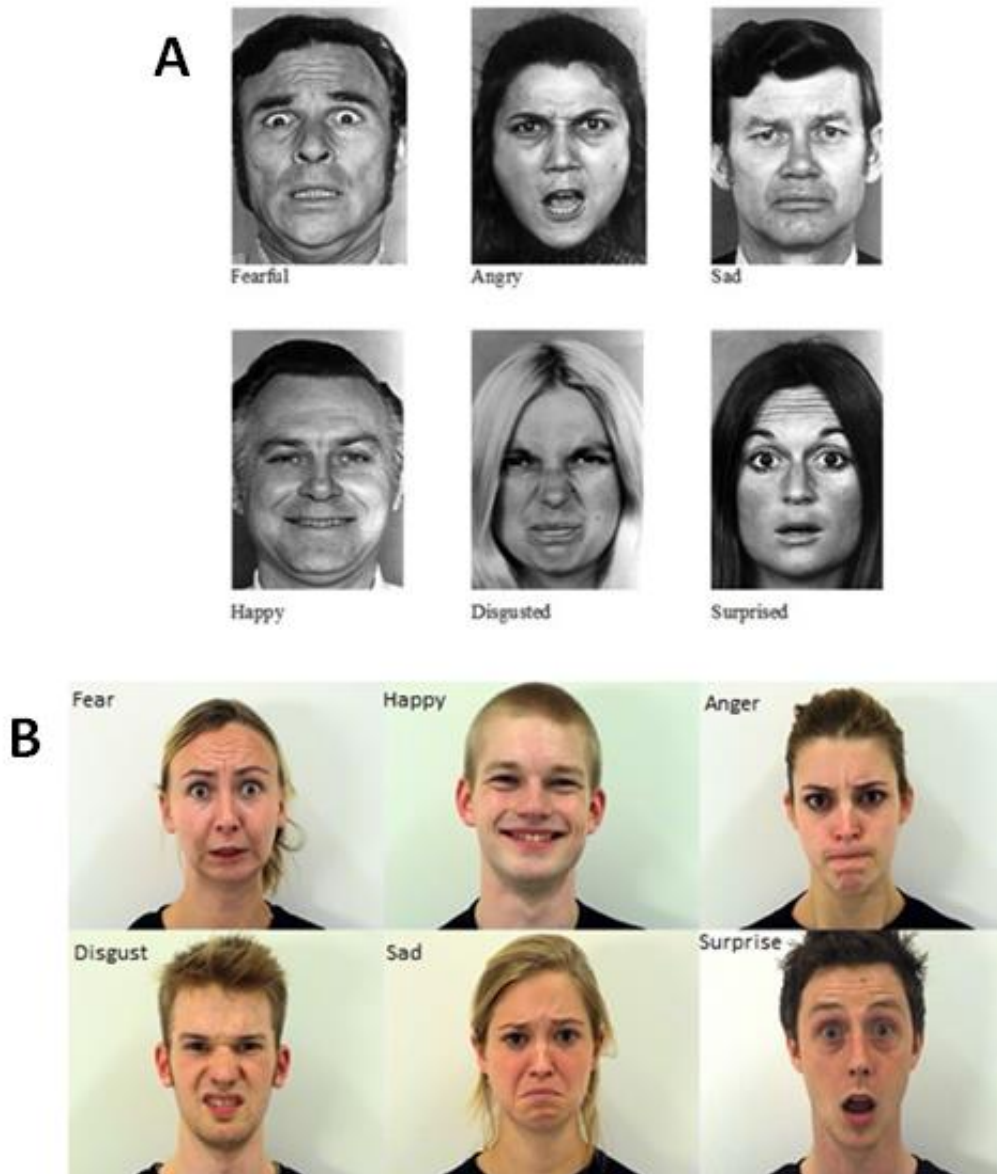
One aim of the current research was to contribute to the TBI literature through the use of comprehensive and innovative methodology. To address this, both modern static and novel dynamic stimuli were used to address growing criticism that static displays of facial expressions do not capture the complex unfolding of emotion and lack ecological validity (Alves, 2013; Knox & Douglas, 2009; McDonald et al., 2003).

From an evolutionary perspective, humans are far less accustomed to viewing static compared to active expressions. Static facial displays do not capture the rapid facial muscle contractions that unfold over short periods (Recio et al., 2011), they exclude the perception of implicit emotional sources such as gaze direction and pattern, tears, as well as autonomic responses such as pupil-dilation, eye blinks, and blushing (Kret, 2015). Static displays of emotion also exclude tone of voice, body language, and contextual cues, all features which aid emotion recognition (Barrett et al., 2011; Bassili, 1978; Ibañez & Manes, 2012; Scherer, Banse, Wallbott & Goldbeck, 1991). Static stimuli usually allow the participant unlimited or prolonged exposure to a fixed apex expression which has very little resemblance to real-life emotions (McDonald et al., 2003; Knox & Douglas, 2009). When emotions are displayed through facial expressions, and there is no need for them to be concealed, the expression duration usually lasts between 0.5-4 seconds and activates the majority of facial muscles

(Ekman, 2003). These expressions are known as macro expressions. Humans also produce microexpressions, which are more rapid than macro expressions, only lasting a fraction of a second and are thought to be a by-product of when an individual tries to conceal or control an emotion (Ekman & Friesen, 1974; Haggard & Isaacs, 1966). Microexpressions can also appear during the act of deception (Porter & Ten-Brinke, 2008). The use of static stimuli, and possibly also the use of staged/posed dynamic stimuli, neglect these fleeting processes (Juslin et al., 2018; Namba et al., 2018).

For more than a decade, scientific findings have reported differences in accuracy, brain areas activated, mimicry levels, and visual strategies when participants view static and dynamic facial expressions (Blais et al., 2017; Rymarczyk et al., 2016; Wehrle, Kaiser, Schmidt & Scherer, 2000; Zinchenko et al., 2018). Trautmann-Lengsfeld et al., (2013) reported that viewing dynamic facial expressions activated a more complex and enhanced neural network during an fMRI study, particularly within the frontal regions, compared to static stimuli. This finding is not surprising as dynamic stimuli require additional attention, working memory and more rapid information processing than static stimuli (McDonald & Saunders, 2005). Visual strategies are also different for the processing of static and dynamic stimuli, with fewer fixations on the eye and mouth area during the recognition of dynamic than static expressions (Blais et al., 2017).

The current research also included newer facial affect datasets, thus improving on research using older datasets, for instance, the black and white images from the Ekman and Friesen's (1976) Facial Expressions of Emotion Stimuli Tests (FEEST) are still frequently employed in research (Connolly, Lefevre, Young & Lewis, 2018; Pohl et al., 2017). However, images from the FEEST are visually outdated (e.g. hairstyles) and raises concerns regarding confounding variables (Figure 34 A).



**Figure 34. Examples of static facial affect datasets. The top set of images (A) are examples taken from the Ekman and Friesen (1976) Facial Expressions of Emotion Stimuli Tests (FEEST). The bottom set of images (B) are from the Amsterdam Dynamic Facial Expression Set (ADFES; van der Schalk, Hawk, Fischer & Doosje, 2009).**

Contemporary static and dynamic facial affect data sets are frequently generated with the majority free to download for research purposes (face-rec.org; Krumhuber, Skora, Küster & Fou, 2016). Despite free accessibility to new facial rating stimuli, researchers frequently adopt black and white static images of facial expressions and often address this as a limitation during discussion sections (Spikman et al., 2013). This is likely because these tests have been widely

used and many studies have demonstrated that the stimulus has strong psychometric properties (Adolphs, Jansari & Tranel, 2001; Young, Perrett, Calder, Sprengelmeyer & Ekman, 2002). The use of static datasets in research has advanced understanding of human emotion perception and it is understandable that researchers still use these datasets in their research as they have strong reliability, are easy to administer and allow comparison across studies. Nevertheless, for the field of social cognitive and affective neuroscience to advance, it seems necessary to move away from static datasets and use more ecologically valid stimuli.

The present research included contemporary static and dynamic social cognition tasks to address some of the limitations of older static stimuli, within the restrictions of university resources and funds. Although the aforementioned section has outlined the criticisms of static stimuli, one of the aims of the current research was to investigate whether there were differences in the visual strategies underpinning static and dynamic facial affect recognition using eye-tracking to augment current findings in this area.

### **5.6.1. The Amsterdam Dynamic Facial Expression Set**

The Amsterdam Dynamic Facial Expression Set (ADFES, van der Schalk et al., 2009) contains dynamic MPEG-2 video clips and static photographs of ten emotions (anger, contempt, disgust, embarrassment, fear, joy, pride, sadness, surprise, and neutral). The data set is comprised of 22 models, both male and female aged 18-25. The video clips begin with a model displaying a neutral face for 0.5 seconds before the onset of the emotion begins. Once the emotion reaches the apex, the pose is held for five seconds (Appendix 4). Static photographs consist of video clips which have been frozen at the apex of an unfolding emotion (Figure 34 B). Faces produced for the ADFES are founded upon the Facial Action Coding System (FACS; Ekman, Friesen, & Hagar, 2002). Models were trained by two coaches specialised in FACS to elicit the

appropriate facial expressions during a variation of the Directed Facial Action Task (DFAT; Ekman, 2007).

The ADFES was selected as the data set includes modern static and dynamic facial affect stimuli. However, the assessment only includes a narrow age-range of models that were trained to elicit facial expressions, and these caveats should be noted. Van der Schalk et al., (2009) showed that the ADFES had good recognition scores and the measure is gaining traction in emotion research (Grainger, Henry, Phillips, Vanman & Allen, 2015; Widen, Pochedly & Russell, 2015; Wingenbach, Ashwin & Brosnan, 2016; Żurowska, Kalwa, Rymarczyk & Habrat, 2018).

Six emotions were chosen for the present research; happy, sad, anger, surprise, fear and disgust. The rationale for this decision was based on Ekman's (1984, 1992) pioneering research identifying the six basic and universal emotions. Furthermore, the six chosen emotions are frequently used in facial expression recognition studies with individuals who have sustained a TBI (Croker & McDonald, 2005; McDonald, Bornhofen & Hunt, 2009; Spikman et al., 2013).

Appendix 5 and 6 detail the procedure of the static and dynamic experiment. There was one practise trial for each experiment to ensure that the participant understood the task. Each of the six basic emotions was presented six times, three times by a male model and three times by a female model, equalling 36 images in both static and dynamic conditions. An online number generator was utilised to randomly determine the order of the stimuli. Reaction time data was collected during the static experiment by participants pressing a corresponding keyboard button. This keypress documented whether the response was correct or incorrect and also moved the experiment on to the next image. Reaction time data was not collected during the dynamic experiment and participants verbally stated their answer to the researcher who would then press the space bar to move onto the next video.

### **5.6.2. Movie for the Assessment of Social Cognition**

The Movie for the Assessment of Social Cognition (MASC; Dziobek, et al., 2006) is a dynamic measure of social mindreading which assesses participant's social cognition abilities such as emotion identification and understanding of false belief, deception, faux pas, metaphors, and irony. Participants are presented with instructions on a screen explaining that they will be watching a short 15-minute film and that they are required to try and understand what the characters are feeling and thinking. The short film depicts four characters meeting for a dinner party. The characters engage in small talk before making and eating dinner and then playing a board game. Some of the characters are friends and some are strangers to each other, thus creating different social reference systems. The main themes throughout the video are friendship and dating. All four characters have distinct personalities and exhibit different personality traits (e.g. timid, arrogant, and outgoing). Different scenarios during the video elicit a range of emotions and mental states in each of the characters (e.g. jealousy, embarrassment, and anger). During the MASC each character is introduced individually with a photograph and their name.

During administration, the video is paused 45 times and participants are asked questions about the characters feelings, thoughts, and intentions. Overall, the task takes approximately 45 minutes to administer. A description of one scene with its subsequent question is given in Appendix 7. An example of a question is 'What is Betty feeling/thinking/intending to do?' There are four possible responses to the MASC, a correct response and three 'distracter' responses (Appendix 8). The three distracter responses are as follows: (1) hypermentalizing relating to the tendency of making complex or excessive mental state inferences (i.e. over-attribution or exaggeration of mental state content) (2), undermentalizing which refers to overly simplistic inferences (i.e. insufficient or reduced mental states) or (3), no mentalizing in which

no mindreading inferences occur (i.e. focus on physical causation and no mental state attribution).

In the literature, the term hypermentalizing is used to define the excessive theory of mind (ToM; Dziobek et al., 2006), colloquially known as ‘reading too much into something’. Hypermentalizing is characterised by individuals making excessively complex assumptions about others people’s mental states beyond observable data or evidence, with a clear lack of connection between thinking and feeling (Luyten & Fonagy, 2015; Sharp et al., 2013). Undermentalizing refers to reduced ToM, meaning that a person is capable of mentalizing, but does it incorrectly (Vaskinn et al., 2015). No Theory of Mind inferences occurs when individuals focus on physical causation showing little mental state attribution.

#### **5.6.2.1. MASC subcategories**

MASC test authors referred to several multidimensional subcategories (e.g. emotions, thought, and intentions) measured by the MASC in the original publication (Dziobek et al., 2006), although video clips measuring each factor were not provided. The present research aimed to conduct fine-grained analyses of social cognition across TBI and control groups and, therefore, wanted to isolate these subcategories during analyses.

MASC subcategories include positive, negative, and neutral emotions. The MASC includes items that focus on verbal (19 displays) and non-verbal (16 displays) aspects of social cognition. Verbal items are divided into literal and sincere scenes (10 displays) and then non-literal scenes where figurative speech or other paralinguistic features are employed (9 displays). Non-verbal items include body language and gestures (10 displays) and facial expressions (6 displays). Each scene may depict more than one domain, for example, scene five requires the participant to understand an intention and figurative speech. Participants have to be aware of verbal and non-verbal cues to perform well on the test. Also, the MASC aims to assess three different



mental state modalities; emotions, thoughts, and intentions. The emotion category contains 17 items, the thought category 7 items, and the intention category 18 items.

The original MASC paper did not provide a template of the specific clips that contributed to each subcategory. Consequently, the current research included two independent raters to assess the proposed MASC subcategories. The test author (Professor Isabel Dziobek) provided preliminary clip categories (through email correspondence), and the two independent raters established an agreement on which clips measured the different subcategory constructs. The subcategories are presented in Table 3 and included emotions, thoughts, intentions, warm (emotional items), cold (non-emotional items), perceptive, and cognitive items. Perceptive items refer to when the mental state of a character can be inferred by perceptive cues, such as tone of voice, facial expressions, and body posture while being relatively independent of the context of the measure. In contrast, cognitive items are those where a mental state inference demands the interpretation of context, for instance, language (literal and non-literal).

Table 3. MASC scenes (clips 1-45) ascribed to corresponding subcategories after inter-rater reliability procedure.

<b>MASC Subcategory</b>	<b>Current<sup>a</sup></b>	<b>Original<sup>b</sup></b>	<b>Number of video clip/scenario 1-45</b>
Emotions	18	17	1, 4, 8, 9, 10, 12, 13, 14, 20, 22, 25, 26, 30, 33, 34, 38, 40, 45
Thoughts	9	7	6, 15, 18, 24, 28, 29, 35, 36, 39
Intentions	15	18	2, 3, 5, 7, 11, 17, 19, 21, 27, 31, 32, 41, 42, 43, 44
Warm (emotional)	13	Unknown	9, 10, 14, 18, 22, 25, 26, 27, 30, 33, 38, 39, 40
Cold (non-emotional)	12	Unknown	2, 6, 7, 19, 20, 21, 28, 32, 36, 37, 43, 44
Perceptive (non-verbal)	14	16	3, 8, 9, 14, 15, 18, 24, 25, 26, 33, 40, 41, 42, 45
Cognitive (verbal)	17	19	1, 2, 3, 5, 6, 7, 11, 12, 16, 17, 21, 23, 28, 36, 37, 43, 44

<sup>a</sup>Number of scenes categorised from the present research by independent raters.

<sup>b</sup>Number of scenes categorised from the original Dziobek et al., (2006) paper.

An advantage of the MASC's properties is that it assesses three main social input modes; visual information (facial expression recognition, gaze), auditory information (prosody), and verbal information (sincerity, sarcasm, the content of speech). The majority of social cognition tasks do not incorporate this multidimensional approach. Another positive aspect of the MASC is that the test allows the quantification of mindreading errors. Erickson (1995) suggested that error responses can often provide useful insight into an individual's ability, highlighting the fact that many neuropsychological measures rarely analyse errors, although some contemporary tests do include this (e.g. Delis-Kaplan Executive Function System; D-KEFS; Delis, Kaplan & Kramer, 2001).

As the MASC was originally developed to assess social cognition abilities in individuals with Asperger's syndrome, the measure has varying difficulty levels to challenge the participant and detect subtle impairments in social cognition. Research has reported that individuals who fall within the autistic spectrum can often perform similarly to control participants on certain social cognition tasks (Brook & Bowler, 1992; Happé, 1994; Jolliffe & Baron-Cohen, 1999; Ozonoff, Rogers, & Pennington, 1991), even though they may show apparent social cognition deficits in everyday life. Dziobek et al., (2006) used receiver operating characteristic curves (a graphical plot which outlines the diagnostic ability of a task) that suggested the MASC was significantly more accurate in discriminating the Asperger's syndrome group from the control group compared to the Strange Stories (Happé, 1994), Reading the Mind in the Eyes (Baron-Cohen, Jolliffe, Mortimore & Robertson, 1997), and a basic emotion recognition task (Ekman & Friesen, 1971).

The MASC is thought to be an ecologically valid measure of social cognition (Dziobek et al., 2006; Lahera et al., 2013) as it relies on real-life settings; it has also been used with several clinical populations including schizophrenic, personality disorder, and depressed cohorts

(Dziobek et al., 2006; Martinez et al., 2017; Preißler, Dziobek, Ritter, Heekeren & Roepke, 2010; Wolkenstein, Schönenberg, Schirm & Hautzinger, 2011).

A Cronbach  $\alpha$  level of 0.84 was reported for the overall MASC scale by Dziobek et al., (2006) which is satisfactory according to Nunnally's (1978) criteria. This finding was supported by Preißler et al., (2013) who also reported high internal consistency (Cronbach's  $\alpha = 0.86$ ), and Fossati, Borroni, Dziobek, Fonagy and Somma (2018) who reported  $\alpha$  levels between 0.70-0.78. Preißler et al., (2013) also reported satisfactory internal consistency scores for the individual subscales of the MASC (emotions: Cronbach's  $\alpha = 0.62$ ; thoughts: Cronbach's  $\alpha = 0.55$ ; and intentions: Cronbach's  $\alpha = 0.71$ ). Dziobek et al., (2006) further reported a correlation coefficient of 0.97 suggesting that the MASC has high test re-test reliability. Subsequently, this measure was selected as a component of the test battery for the current study.

### **5.6.3. The Awareness of Social Inference Test**

The Awareness of Social Inference Test (TASIT; McDonald, Flanagan & Kinch, 2003) was adopted for the present research to also assess participant's social perception skills. The test comprises of video vignettes of individuals engaged in social interactions. Professional actors were employed to use a 'method style' of acting where they were required to demonstrate an emotion relevant to the situation. This method provided a realistic, spontaneous, and natural test context. The TASIT includes three sub-component measures; the emotion evaluation test (EET), the social inference-minimal test (SI-M), and the social inference-enriched (SI-E) test. The task is moderately lengthy (approximately one hour to complete with control participants) and potentially longer with TBI participants.

#### **5.6.3.1. The Emotion Evaluation Test (EET)**

The EET subtest of the TASIT assesses social communication components that include facial expressions, tone of voice, and gestures. As part of this subtest, participants are required to

identify the emotion expressed by the actor. Participants are provided seven emotions to choose from; anger, anxiety, surprise, sadness, happy, revulsion, and neutral. These emotions are then grouped into positive (surprise & happy) and negative (anger, anxiety, sadness & revulsion) emotions for analysis. The EET consists of 28 short videos (15-60 seconds) displaying actors interacting with each other in everyday situations. There are four videos for each of the seven emotions which are presented in a quasi-randomised fashion. For some videos, there is one actor, either on the telephone or directly talking to the camera. For the remaining videos, two actors are interacting with each other. See Appendix 9 for an example of the EET design.

### **5.6.3.2. The Social Inference-Minimal (SI-M)**

The SI-M assesses the ability to read paralinguistic features, facial expressions, and tone of voice in either sincere or sarcastic situations. Participants are required to pay close attention to the tone of voice, facial expressions, gestures, and body language to correctly identify whether the speech is sincere or sarcastic. There are 15 short videos (20-60 seconds) in the SI-M displaying conversational exchanges between actors. Five of the videos have sincere exchanges, another five have sarcastic exchanges, and the remaining five videos have paradoxical scripts, that is, the scripts are nonsensical unless it is understood that one actor is being sarcastic (Appendix 10).

After each video, the participant is required to answer four questions about the target actor. The first question indexes what the speaker was trying to 'do' (their intentions, for example, to insult or to reassure), what they were trying to say (what they meant), what they were thinking (what the participant thinks the target actor knows), and lastly, what the target actor was feeling in a given context. Please see Appendix 11 for the design of the study.

### 5.6.3.3. The Social Inference-Enriched test (SI-E)

The SI-E part of the TASIT included 16 short (15-60 seconds) videos of everyday conversations (Appendix 10). The SI-E assesses the participants' ability to use contextual cues (tone of voice and facial expressions) to determine if the conversation in the video is deceptive or if the meaning is opposite to what the actor is saying (i.e. the actor is being sarcastic).

The sarcasm and deception videos have identical scripts but during the sarcastic videos, actors do not try to obscure the truth but instead seek to emphasise it. The deception exchanges include *white* lies and *sympathetic* lies. White lies include situations such as a mother lying to a father about their child finishing dinner, with sympathetic lies including situations where a friend tells another friend that they do not look fat in a new outfit. Again, participants must use paralinguistic cues to make the correct answer. Furthermore, participants are provided with additional, enriched contextual cues during this subtest. Half of the video clips have camera edits to help participants establish the correct category of the video clip, for example, in the deceptive mother scenario, participants are provided with a camera shot of the child's unfinished dinner.

The additional camera shots providing supplementary information can only be seen by the main actor during the deception videos (mother); the other actor (father) cannot see the extra visual cue. During the sarcastic video clips, both actors can see the visual cues highlighting the correct subtest category. The remaining halves of the videos use a prologue or an epilogue scene where the main actor discusses their thoughts with a third actor. For example, during the new outfit scenario, Ruth tells Gary that she does *not* think he has put on weight. Nevertheless, a prologue scene features the sympathetic friend (Ruth) telling another friend (Keith) that she does think Gary has put on weight. These additional cues should aid participants in understanding the video. The SI-E has a similar answer format to the SI-M in that it asks the participant's four

questions about what the actor was doing, saying, thinking, and feeling. See Appendix 12 for the study's design.

The advantage of the TASIT, compared to other social inference tasks, is that it includes naturally occurring visual cues during social situations (demeanour of the speaker and reaction of the listener) which many other experimental inference tasks do not. The MASC also includes dynamic social cues but does not explore the processing of sincere and sarcastic exchanges in as much depth as the TASIT. In conjunction with each other, the MASC and the TASIT provide a comprehensive assessment of social cognition. It has been reported that the TASIT has good reliability and high test-retest reliability levels ( $r = 0.74-0.88$ ) as well as good construct validity, low practice effects, and high ecological validity (McDonald, 2012; McDonald et al., 2003).

### **5.7. Control test (International Affective Picture System)**

The International Affective Picture System (IAPS; Lang, Bradley & Cuthbert, 2008) is a set of colour photographs depicting a wide range of categories aimed at assessing various normative emotion ratings that include pleasure, arousal, and dominance. The IAPS has been used in research with clinical populations (Jayaro, de la Vega, Diaz-Marsa, Montes & Carrasco, 2008) as well as in research investigating eye movements in reaction to emotional scenes (Bradley, Houbova, Miccoli, Costa & Lang, 2011; Nummenmaa, Hyönä & Calvo, 2009; Simola, Tornainen, Moisala, Kivikangas & Krause, 2013).

In the current study, IAPS stimuli were included to investigate general eye scan patterns in response to different types of emotive stimuli (humans, mammals, non-mammals, and objects).

A study by Douglas et al., (2010) documented that a group of TBI participants exhibited abnormal eye scan patterns compared to control participants when viewing faces but typical fixation patterns when viewing objects. The current study aimed to further explore this dissociation. Forty-eight images were selected from the IAPS, 12 for each category (Appendix

13). Images were chosen because the focus of the picture was a human, animal, or object. To try and minimise attentional demands, images with little additional information in the background were selected by the researcher. A random online number generator selected the order of the images.

In this task, participants viewed an image for eight seconds, similar to eye-tracking studies exploring visual strategies in response to different stimuli (McPartland, Webb, Keehn & Dawson, 2011), and were asked to determine if the image depicted a human, animal, or object. Once an answer had been verbally provided the next image would be presented. This particular study aimed to replicate the work of Douglas et al., (2010) by exploring the visual strategies underpinning the perception of humans and objects. As well as comparing the gaze patterns for faces and objects, the current PhD research programme also wanted to extend Douglas and colleagues work by exploring visual strategies underpinning the perception of animals, namely mammals and non-mammals. Current theory suggests that there is a positive correlation between the degree of biological/behavioural similarity between a given species and human beings, and an increase in attitude level towards the species (Batt, 2009; Prokop & Randler, 2018; Wilson, 1984). Therefore, it was hypothesised that participants would elicit more and longer fixations to mammals, compared to non-mammals, because they felt a sense of similarity towards them. As humans have a natural disposition to attend to human faces (Pascalis & Kelly, 2009), it was hypothesised that human stimuli would elicit more and longer fixations, followed by mammals (based on research by Batt, 2009; Prokop & Randler, 2018; Wilson, 1984), non-mammals and then everyday objects, which are usually mundane and elicit little emotion. Findings could elucidate whether aberrant visual strategies are specific to humans or whether they extend to other living creates as well as everyday objects.



**Figure 35. Example stimuli from the International Affective Picture System (IAPS; Lang, Bradley & Cuthbert, 2008) control task. Example of an (A) human, (B) object, (C) mammal and (D) a non-mammalian animal included in the control task to explore natural eye scanning patterns in response to different stimuli.**

## **5.8. Equipment**

### **5.8.1. Tobii T120 eye tracker**

Participants in the present research viewed static and dynamic social cognition tasks. Fixation patterns were recorded using a Tobii T120 Eye Tracker (Tobii Technologies, Stockholm, Sweden). The recording of eye movements and the analysis of gaze allocation quantified the degree of social information and emotional valence that influenced low-level attentional and visual resources. The T120 eye-tracker has a 17-inch thin-film-transistor screen (1280x1024 pixels) with embedded near-infrared light-emitting diodes and optical sensors (Figure 36). The cameras capture pupil location and size with an accuracy of  $0.5^\circ$  at a sampled data rate of 120 Hz. As the T120 has two cameras, producing two images of the eyes, as well as pupil and corneal reflective technology, the equipment computed information from two sources concerning eye position. Thus Tobii software calculates robust fixation positions regardless of moderate head movements, negating the need for restrictive head constraints.

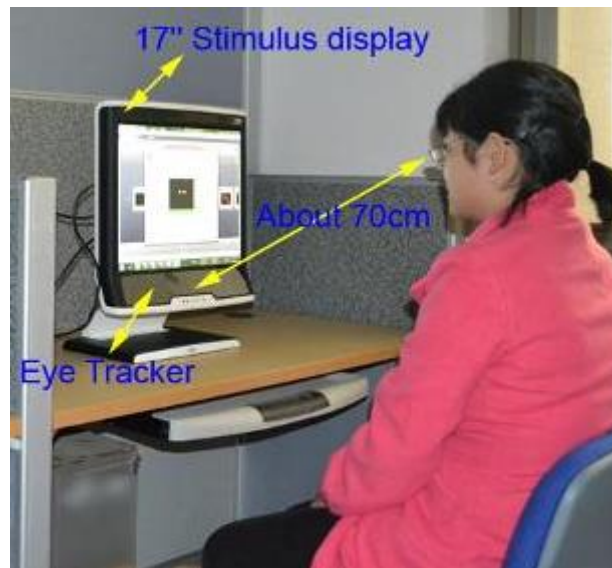


Both static and dynamic areas of interest (AOI) were constructed using Tobii Studio Analysis Software 3.1.6 through the oval, rectangular and freehand drawing tools, similar to pre-existing research using Tobii studio (Müller, Baumeister, Dziobek, Banaschewski & Poustka, 2016). Each stimulus typically consisted of an eye, nose, and mouth AOI (Figure 37), except for the control task demonstrated in Figure 38 To create the AOI for the static stimuli, a boundary was drawn around a feature (e.g. the eyes) (Figure 37). The creation of AOI for the dynamic stimuli was more complex due to the movement of actors (e.g. natural movement of facial features associated with a change in mood) and also changes in scenario or scene (e.g. due to TASIT and MASC's film-like presentation, different camera angles may mean that an actor is in the first part of the clip, absent for the second part of the clip, and then re-enters for the third part – see Appendix 7 as an example). Therefore, to account for a character, scenario, scene, zoom or perspective changes across different clips, dynamic AOI were individually generated, again using oval, rectangular and freehand drawing tools, and were manually moved and resized to track the targets of interest. This manual drawing and moving were carried out on a frame by frame basis by using the playback controls (play/pause, frame forward/backwards) and increasing the zoom so smaller facial features could be drawn around. So, for example, an AOI would be drawn around the eye area and doing so would create a key-frame marker onto the AOI timeline. Using the playback controls, the researcher would move the movie forward until the eyes had moved such that it was outside the boundaries of the AOI as defined in the first key-frame. The AOI would then be dragged and re-sized if necessary, to encompass the eye region fully again. These steps were repeated until the last frame where the AOI was present.

The Tobii T120 allowance for head movement is  $30 \times 22 \times 30$  at 70 cm. During a recording, the Tobii eye tracker collects eye movement data points every 8.3 ms and assigns a timestamp and an 'X, Y' coordinate. This information was analysed using Tobii Studio 3.1.6 eye movement recording software. For the current research, raw data was organised into mean

fixation durations and fixation counts using the default Tobii fixation filter in Tobii Studio. The Tobii fixation filter uses the classification algorithm proposed by Olsson (2007) and detects rapid changes in gaze points using a sliding averaging window. Fixation filter algorithms rely on mathematical and statistical processes which capture eye movement. The default Tobii fixation filter algorithm sets a fixation threshold at 35 pixels for velocity and 35 pixels for distance per sample. If the velocity of the eye movement was above the  $0.5^\circ$  per the second threshold then the eye movement was classified as a saccade and if below this parameter, it was classified as a fixation.

The eye tracker was calibrated for each participant using the regular five-point calibration of each eye and was set to the calibration speed of medium. The foreground colour was set to red and the background colour was set to grey. The calibration area was set to 100%. Calibration took place at the beginning of each eye-tracking experiment and also halfway through the presentation of the MASC as the task is lengthy. Participants were seated approximately 60-65cm away from the screen in a stationary chair. Fixation crosses were not utilised during the research programme as the research team wanted to explore natural gaze behaviour in response to the social stimuli. As one of the research questions was to see if there were differences between the individuals with TBI and matched controls for the first fixation, it did not seem appropriate to include a fixation cross as this would have contaminated the findings. Furthermore, research has demonstrated that forced fixation conditions during emotion perception paradigms produced detrimental effects on emotion perception performance (Peterson & Eckstein, 2012).



**Figure 36. Image of the Tobii T120 Eye Tracker (reproduced from Nudt, 2007).**

### **5.9. Ethical procedures**

All procedures were approved by the Leeds East Research Ethics Committee, Rotherham, Doncaster and South Humber NHS Trust, Sheffield Health and Social Care NHS Trust, and Sheffield Hallam University Research Ethics Committee.

### **5.10. Recruitment**

Consultants based within two NHS Trusts referred TBI participants, who met inclusion criteria, to the study. Collaborating clinicians identified potential participants based on the individual's TBI history and risk factor. Clinicians initially approached potential participants and verbally explained the research. If the individual expressed an interest, they were provided with a participant information sheet that they were encouraged to take home, read, and show family or friends. With the potential participant's verbal consent, the clinician provided the chief investigator with the participant's contact details so they could further discuss the research and obtain consent.

## 5.11. Participants

Eighteen TBI participants and 18 age and sex-matched controls were recruited for the research. There was no attrition throughout the study and all individuals with TBI and matched control participants completed all sections of the research programme.

### *Inclusion criteria*

1. An acquired adult TBI which had been verified with neuropsychological measures on hospital admission or brain pathology evident through imaging scans.
2. One year post-injury to warrant that chronic effects, rather than acute effects, of TBI, were being measured.
3. Eighteen years old or over.

### *Exclusion Criteria*

1. A history of psychiatric illness that is documented to disrupt eye scan patterns (e.g. schizophrenia/psychosis).
2. Severe current drug and alcohol abuse.
3. Did not have the capacity to provide fully informed consent.
4. Individuals over 65 were excluded to try to mitigate natural ageing effects.

Variables used to match control participants to TBI participants included gender, age, and years of education. Matched control participants were recruited using stratified opportunity and snowballing sampling. All participants were screened for depression, anxiety and visual deficits. It should be noted that none of the participants, from either the group with TBI or matched controls, scored above the HADS score of 16 which indicates abnormal levels of anxiety and depression (group with TBI (Anxiety  $M = 8.11$ ,  $SD = 4.61$ , depression  $M = 6.00$ ,  $SD = 3.31$ , overall HAD's  $M = 14.11$ ,  $SD = 6.28$ ); matched control group (Anxiety  $M = 4.94$ ,  $SD = 2.04$ , depression  $M = 2.67$ ,  $SD = 1.94$ , overall HAD's  $M = 7.61$ ,  $SD = 3.48$ )). All

participants exhibited sufficient visual and perceptual abilities according to the VOSP and CORVIST criteria. Males are over-represented in the TBI population so it was expected to recruit more males than females (Marshman et al., 2013). Demographic information and psychometric measure scores can be found in Table 4. Information regarding injury pathology, severity, and cause were obtained from medical records, including scan data and A&E admission notes (Table 5 and supplementary information). Collaborating clinicians who referred the participants to the research project classified the severity of the injury. This detail was provided to the researcher via telephone recruitment meetings, post-consent, and was based on injury indices and expert clinical judgement. In line with most TBI research, injuries were heterogeneous and included brain haemorrhages, skull fractures and contusions. Detailed case notes for each TBI participants can be found in Appendix 13.

Table 4. Demographic information for TBI and control group.

Descriptive statistic	Gender	Current Age	Age at injury (years)	Post-injury years	Years of education	Verbal IQ	Performance IQ	Overall IQ
<u>Number</u>								
<i>TBI</i>	m= 15, f= 3	-	-	-	-	-	-	-
<i>Control</i>	m= 15, f=3							
<u>Mean</u>								
<i>TBI</i>	-	44.94	36.44	8.50	14.83	84.06	91.00	90.25
<i>Control</i>		43.83	-	-	15.56	95.33	104.72	100.06
<u>SD</u>								
<i>TBI</i>	-	11.69	13.79	8.68	4.25	18.71	17.50	17.66
<i>Control</i>		12.26	-	-	3.65	8.66	11.64	10.44
<u>Range</u>								
<i>TBI</i>	-	26-62	17-59	1-28	10-23	57-135	55-111	55-120
<i>Control</i>		25-63	-	-	11-23	82-113	79-121	81-116
<u>Mann-Whitney U test</u>		$p =$			$p = .389$	$p =$	$p = .015^{**}$	$p = .025^{**}$
		.696				.007**		

\* Two TBI participants opted out of the WASI (IQ test)

\*\* Significant at  $\alpha = .05$

Table 5. Demographic and clinical characteristics of TBI participants ( $n=18$ ).

Participant Code	Gender	Age	Injury Location	Injury Severity	Years Post-Injury	Mechanism of Injury
1	Male	26	O	Severe	7	Assault
2	Male	40	FLP	Severe	21	RTA (car)
3	Male	53	FLP	Severe	15	Fall
4	Male	54	O	Severe	30	RTA (car)
5	Female	33	O	Mild	10	Fall from horse
6	Female	60	O	Severe	29	RTA (car)
7	Male	47	O	Moderate	16	RTA (motorbike)
8	Male	28	FLP	Severe	11	Assault
9	Male	50	FLP	Severe	5	Fall (unconfirmed)
10	Male	31	FLP	Severe	1	Assault
11	Male	63	FLP	Severe	4	RTA (pedestrian)
12	Male	43	FLP	Severe	3	Fall from scaffold
13	Male	33	O	Severe	16	Fall from seizure
14	Male	60	FLP	Severe	3	Fall downstairs
15	Male	53	FLP	Severe	2	Fall
16	Female	47	FLP	Severe	1	Fall
17	Male	39	FLP	Severe	4	Assault
18	Male	59	FLP	Severe	1	Cyclist (collision with car)

*Key:* Frontal lobe pathology (FLP); pathology which does not encroach on either frontal lobes, or occipital cortex, but may be present in other cortical or subcortical brain regions (O); road traffic accident (RTA).

### 5.12. Procedure

Test sessions took place either in the university laboratory or in the hospital clinic. The length and number of sessions were determined by the participant. Typically, control participants completed testing in one session over a three to four hour period (with breaks), although a minority of control participants decided to divide testing into two sessions that lasted

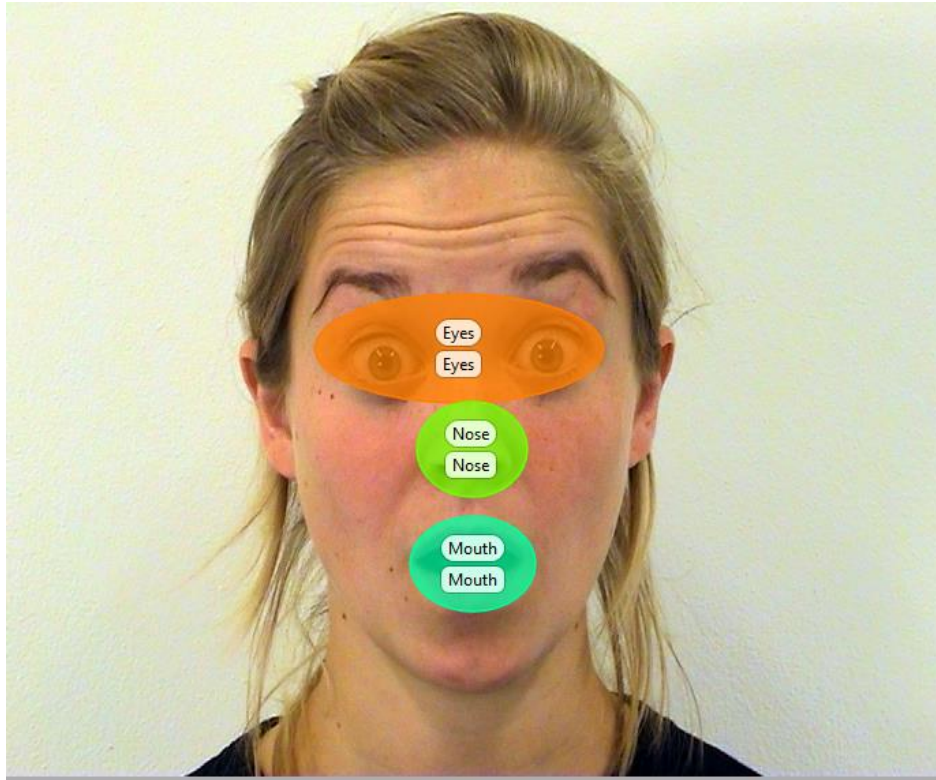
approximately two hours each. The number and length of testing sessions for TBI participants varied, with some participants completing the testing in two sessions and others taking up to six sessions. Participants completed the screening measures first, followed by the demographic tasks, and then social cognition/eye-tracking tasks. Counterbalancing was implemented to minimise order effects and a set A and set B procedure was used (Appendix 14).

### **5.13. Study design and analyses**

The current research had a cross-sectional design with two cohorts; individuals who had sustained a TBI, and controls matched for age, gender and years of education. Similarly, cross-sectional designs have been used in previous research studies investigating social cognition after TBI (e.g. Neumann et al., 2015; Spikman et al., 2013; Rosenberg, McDonald, Dethier, Kessels & Westbrook, 2014).

Eye-tracking data included time to first fixation, first fixation duration, total fixation duration, and total fixation count to AOI (see Appendix 15 for more detailed definitions of the Tobii descriptive statistics).





**Figure 37.** An example of AOI for a female face during the ADFES static task. For the IAPS study, AOI incorporated the whole animal, object or human.



**Figure 38.** An example of AOI for the IAPS control task. An example of an animal (Image A) and an object (Image B) condition during the IAPS control task. Image numbers from left to right (1080 and 7175).

The human images included head and body shots and, therefore, AOI sizes varied across clips (Figure 39).



**Figure 39.** An example of human images used during the IAPS control task. IAPS image numbers from left to right (top: 2495, 2030, bottom: 2005, and 2830).

High acuity visual information is collected during fixations and so by investigating this process, the underpinnings of visual attention and cognitive engagement can be understood (Macknik & Martinez-Conde, 2007). Fixation metrics were selected for analyses following other research that suggests the majority of visual processing occurs during a fixation, with very little visual processing during saccades (Auyeung et al., 2015; Helo, Pannasch, Sirri & Rämä, 2014; Leigh & Zee, 2015; Vassallo et al., 2010). Saccades were not analysed in the present research as Mancuso et al., (2015) proposed that saccades were less impacted by TBI than fixations. These temporal parameters (number, duration and sequence of fixations) offer an objective measure of visual processing and visual attention.

The interpretation of the time to the first fixation is important as it provides information about attentional resources and saliency during visual scanning (Pérez-Moreno et al., 2016). Calvo, Averó and Lundqvist (2006) suggested that while time to the first fixation may not be the most

accurate measure of attention, the first fixation duration could provide a more rigorous measure of attention, as gaze duration quantifies the amount of attention that is directed to an AOI. With this in mind, the present research included both time to first fixation and first fixation duration for the ADFES static and dynamic tasks and the IAPS control task. These eye-tracking metrics were not utilised during the MASC or the TASIT as the tasks were lengthy with multiple facial stimuli and, thus, many potential AOIs during each scene. The inclusion of numerous AOIs across different times during each scene meant time to the first fixation for a particular AOI was not as meaningful as the total number of fixations and total fixation duration to AOI types. The traditional fixation duration and fixation count metrics were collected for all social cognition tasks. Preliminary data analysis indicated that there were missing data values for the majority of tasks, although this was considerably lower compared to the first fixation and first fixation duration.

There are often missing values associated with dynamic eye-tracking data due to participants not fixating on AOI (Scheiter & Eitel, 2017). This issue can be addressed in two ways. Firstly, group means can be calculated based on the differing number of raw scores (i.e. fixation duration or count); however, this can lead to heterogeneity of variance. The second option is to use missing data imputation in which was the method utilised for the current research. For time to the first fixation, missing scores were replaced with the total video or image display time. For example, missing data for the ADFES dynamic task were replaced by the length of a video (e.g. eight seconds), indicating that the participant did not fixate on that particular AOI. For the first fixation duration, missing scores were replaced with a zero to indicate that no fixations were made in the AOI. Missing data was not an issue for total fixation duration or fixation count as Tobii studio has an option to include zero scores for these two metrics.

The analysis differed for each experiment and specific details are presented at the beginning of each analysis chapter.

#### **5.14. Considerations for reporting inferential statistics**

To determine the general quality of data and parametric assumptions were checked per group using histograms, box plots and the z-test for skew and kurtosis. The z-test method calculates a z-score and then divides the skew and kurtosis values by their standard errors (Dancey & Reidy, 2007; Field, 2009; Kim, 2013). If sample sizes are small ( $n < 50$ ), then an absolute z-score larger than 1.96, which corresponds to an alpha level of 0.05, indicates that the sample is not normally distributed. Outliers were identified using z-scores with a limit of  $\pm 3.29$  (Dawson, 2011; Field, 2009).

There are contrasting opinions regarding the transformation of non-normal data (Feng et al., 2014; Games & Lucas, 1966; Glass, Peckham & Sanders, 1972; Levine & Dunlap, 1982). After much consideration, traditional transformation methods were not deemed appropriate for the current data for several reasons. Firstly, a central concern for transforming data during hypothesis testing is that the newly transformed data may not be testing the null hypothesis, as developed for the original data. That is to say, the parameters of the equation used to obtain the observed difference between groups with a normal distribution are not the same as the equation associated with various types of transformation. For instance, normal distribution methods compare means on an arithmetic basis while, take, for example, log transformation generates geometric means. Consequently, the two null hypotheses are not equivalent and different empirical constructs are therefore being tested. As a result, any outcomes from transformed data must be interpreted with caution (Feng et al., 2014; Grayson, 2004). Secondly, the fundamental purpose of transforming data is to alter the nature of the variable and this also makes understanding findings more difficult, particularly if the variable is meant to be interpretable e.g. reaction time (Osborne, 2002). Take for example square root transformation, this method eliminates the equal intervals between successive values and thus reduces interval and ratio data to ordinal data. While this may not pose a problem for some data, there are certain

statistical analyses, such as ANOVA, which assume interval or ratio scales (Osborne, 2002). Thirdly, there is no guarantee that using transformations will make the data conform to a normal distribution and even after transformations have been applied, data can still display skew and kurtosis (Feng et al., 2014). Transforming data can lead to an increase in the variability of data, such as outliers (Feng et al., 2014).

Statisticians frequently question the appropriateness of transforming data to fit statistical analyses (Field, 2009). Seminal research by Games and Lucas (1966) reported that ANOVA's performed adequately on skewed data sets and that transforming the data sometimes created more disadvantages. Although it is commonly believed that non-parametric tests are unaffected by violations of normality this is not the case. When homogeneity of variance is violated, both parametric and non-parametric tests can lead to a distortion of power and a rise in the likelihood of a type I error with non-parametric equivalents sometimes having a greater degree of bias (Zimmerman, 1998). Violations of homogeneity of variance are less severe if sample sizes are equal, which was the case for this research, but this point is important as it highlights that non-parametric tests are also affected by non-normal distributions.

It was decided that transforming data, although suitable in some experimental designs, was more of a limitation for the current research. To keep the integrity of data, ANOVA was conducted on untransformed scores, even if there were violations of normality as ANOVA is robust to moderate violations of normality (Donaldson, 1968; Glass et al., 1972; Kim, 2013). As there is no objective rule as to what constitutes a 'moderate' violation of ANOVA (Glass et al., 1972, Hoekstra, Kiers & Johnson, 2012; Cottingham, Lennon & Brown, 2005) rational judgement was used to determine the boundaries of mild, moderate and severe. This approach is parallel with research advocating flexibility around knowing when to draw 'violation' lines when designing more informative ecological experiments (Cottingham et al., 2005). In general, if sample sizes are equal then this lessens the likelihood of normality assumptions being

violated (Finch, 2005). It was fully acknowledged that this decision could have consequences regarding type I errors if violations went beyond moderate but this risk was conceived as less severe compared to transforming the data. Greenhouse-Geisser correction for degrees of freedom was applied if Mauchly's test was violated (Dancey & Reidy, 2017).

With regards to post-hoc tests, while stringent corrections to  $\alpha$  levels for multiple comparisons are advocated by some statisticians (Shaffer, 1995), this can increase the risk of type II errors and loss of power, particularly if there are numerous comparisons (Field, 2009; Kim, 2015; Krzywinski & Altman, 2014; Perneger, 1998; Rothman, 1990). Furthermore, it has been suggested that type II errors can be more pernicious compared to type I errors as these results are often not reported in psychological research. Lieberman and Cunningham (2009) maintain that as social and affective neuroscience are still relatively primitive research areas, and most studies are exploratory, it is more pertinent to not reject possible true effects compared to accepting false effects. Additionally, they highlight the fact that false effects will not be replicated in future studies through data aggregation and can, therefore, be approached as 'self-erasing' whereas unreported true effects will rarely ever be the subject of replication. Nevertheless, it was acknowledged that applying no corrections to multiple comparisons increased the likelihood of making a type I error (Cormier & Pagano, 1999; Moyé, 1998). Adopting researcher judgement and adhering to Goodman's (1993) advice that the p-value is used as a flexible tool within the context of a given problem, the  $\alpha$  level for multiple comparisons was set at 0.01 to balance between type I and type II error avoidance. If the omnibus ANOVA was significant but variables violated the assumptions of normality then t-tests were still conducted.

The current research used the traditional approach of following significant ANOVA with separate ANOVA's for each of the dependent variables. Some regard this approach as

controversial (Grice & Iwasaki, 2007) as it defeats one of the underlying strengths of ANOVA, which is to reduce the likelihood of type I errors by running multiple analyses (Share, 1984). However, the follow-up ANOVAs are deemed ‘protected’ by the omnibus ANOVA (Bock, 1975). This ‘protection’ stems from the idea that if the initial multivariate test is non-significant, then follow-up tests will not be conducted. Nevertheless, this view is somewhat contested (Huberty & Morris, 1989) as a significant ANOVA may reflect a significant difference for one dependent variable only; the other dependent variables may be non-significant. The original ANOVA only protects the significant dependent variable (Bray & Maxwell, 1985). That being the case, the current research applied a post-hoc correction by adjusting the  $\alpha$  level to .01 to control the family-wise error rate (Stevens, 2001).

### **5.15. Summary**

This chapter provides a rationale of tasks selected and a summary of research design, data preparation and choice of statistical approaches. The next chapter outlines findings from the MASC.

## **6. Chapter six: The International Affective Picture System (IAPS)**

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### **6.1. Objectives and hypothesis**

Humans have a preference for faces that is exhibited early in life with young infants generating more attention to faces and face-like stimuli compared to distracters (Frank et al., 2009; Powell, Kosakowski & Saxe, 2018). This preference extends into adulthood and is also demonstrated through face pareidolia, the illusory perception of non-existent faces in common objects (Liu et al., 2014). One illustration of this is from Windhager et al.'s (2010) experiment having discovered that participants still generated the typical 'T' fixation pattern (looking at the eyes and mouth more) associated with face processing when viewing inanimate objects. Their participants developed analogies between faces and cars (e.g. viewing the headlights as the eyes and the grill as the nose) and exhibited the characteristic preference for eyes (i.e. they would look at the headlights first even if the task was for them to focus on the grill). Indeed, the human brain appears to display a high degree of specialization for social stimuli that may be modulated by a 'social brain' network (Brothers, 1990; Dunbar, 2009). Current evidence theorises that the visual cortex contains distinct and specialized neural pathways for processing faces (Kanwisher, McDermott & Chun, 1997; Leibo, Mutch & Poggio, 2011). For instance, human brains have evolved a specialised face-processing region, coined the fusiform face area, which is activated significantly more during the perception of faces compared to other stimuli (Kanwisher & Yovel, 2006). The hypothesis that humans have developed distinct face processing neural pathways is further supported by data reporting differences in developmental trajectories of the visual system for faces and everyday objects, with the former extending into young adulthood (Meinhardt-Injac, Persike & Meinhardt, 2014).



Face-specificity theories argue that faces and objects are processed differently (Galambos et al., 2018; Kanwisher, 2000; McKone, Kanwisher & Dunchaine, 2007). As everyday objects vary in their configuration it is harder to distinguish fixation patterns, although fixations would still be directed to the most salient parts of the object. As was mentioned in the previous chapters, aberrant fixation patterns in response to faces are documented in several clinical populations. It is less clear whether these abnormal gaze-patterns extend to other stimuli but the extant literature suggests that this may be the case. For example, Wang, Chawarska, Zucker, Scassellati and Shic (2014) reported that typically-developing toddlers displayed different basic attentional strategies when processing human faces compared to block designs. However, a group of toddlers with autism employed a similar visual strategy for processing both stimuli. A similar result was found by McPartland et al., (2011) who reported that a group of autistic children exhibited greater attentional resources to the upper, relative to lower, portions of upright faces, three-dimensional objects, and geometric patterns compared to a control group.

There is a limited evidence-base suggesting that TBI can alter eye scan patterns (Adolphs et al., 2005; Douglas et al., 2010; Oatley et al., 2014; Wolf et al., 2014). Yet, this restricted research has mainly explored gaze-patterns in response to faces and has not fully investigated whether aberrant eye scan patterns extend to other stimuli, such as everyday objects. However, Douglas et al., (2010) have addressed this hypothesis when they asked ten TBI participants and gender and age-matched controls to view 18 images of facial expressions and 15 images of objects. During this task, eye movements were recorded. The group affected by TBI generated increased number, duration and dispersion of fixations to the facial stimuli compared to the control group. In contrast to the clinical findings aforementioned, this abnormal visual scan path was not evident for the object condition, suggesting that aberrant fixation patterns post-TBI may be specific to faces. This difference highlighted the concern that, although TBI, autistic and schizophrenic cohorts share similar symptoms and biological mechanisms (Singh

et al., 2016), the conditions are fundamentally different in aetiology and so likely to produce contrasts.

Therefore, this particular study aimed to build on the work of Douglas et al., (2010) by exploring the visual strategies underpinning the perception of humans and objects. Douglas et al's., (2010) findings are pertinent for the TBI research field and replication of their results would support the hypothesis that TBI can induce specific impairments to the processing of social stimuli (e.g. humans) compared to non-social items. The experiment also wanted to extend the work of Douglas and colleagues and, therefore, included images of other living stimuli; mammals and non-mammals. The research team were interested to investigate whether these four sets of different stimuli would evoke different gaze patterns and whether these patterns would be different in the individuals with TBI compared to the matched control group. As outlined in the methodology, the current experiment ranked humans as the most emotive stimuli followed by mammals, non-mammals, and objects.

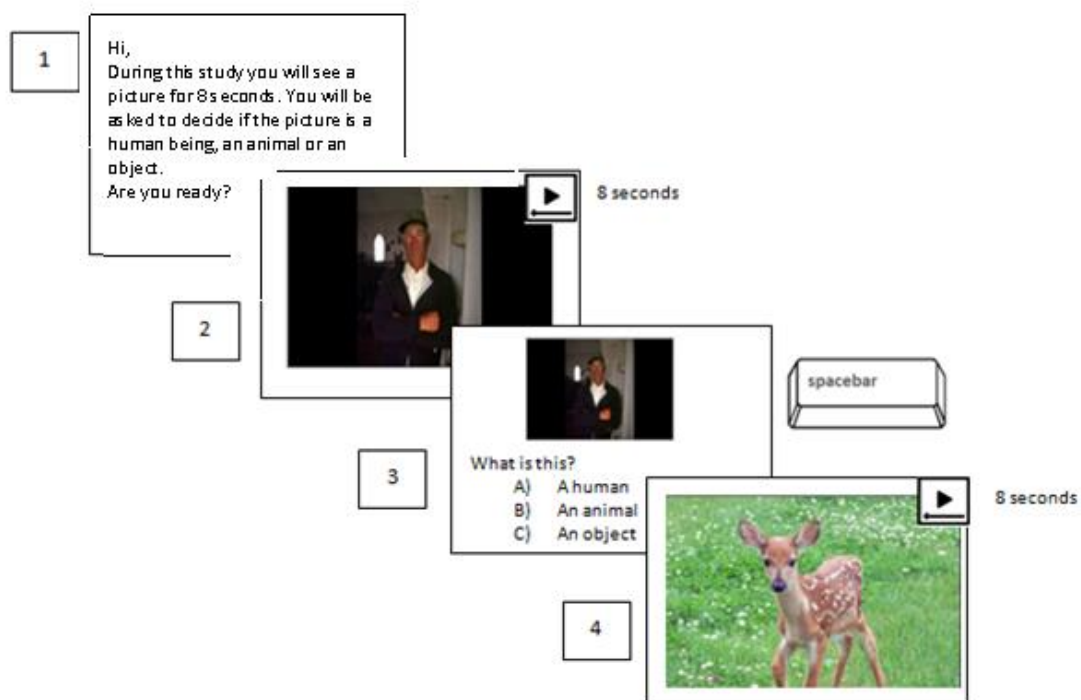
**Hypothesis 1.** The group affected by TBI would display different fixation patterns to control participants when viewing the human and animal stimuli, but there would be no difference between the groups when they viewed everyday objects.

**Hypothesis 2.** Participants would generate a quicker first fixation and would have more and longer fixations to emotive human and animal stimuli compared to everyday objects.

## **6.2. Procedure**

The International Affective Picture System (IAPS; Lang et al., 2008) images were presented on the Tobii T120 Eye Tracker. Participants were seated approximately 60-65cm away from the eye tracker screen in a stationary chair. Participants viewed an instruction screen and were

given time to ask any questions. The images remained on the screen for the whole eight seconds and the task could not be moved on, even if the participant named the category. The answer screen automatically appeared after eight seconds and the researcher noted down the participant's answer and moved the task on by pressing the space bar. Figure 40 outlines the procedure of the experiment.



**Figure 40.** A diagram to illustrate the sequence of events for the IAPS study.

### 6.3. Exploration of data

Only eye-tracking data collected during the main image viewing (e.g. boxes 2 and 4 in Figure 40) was analysed. To explore data trends and to check parametric assumption criteria, distribution plots and statistics were examined. Histograms illustrated skew and kurtosis for all four eye-tracking metrics (time to first fixation, first fixation duration, the total number of

fixations and total fixation duration), which was further confirmed by  $z$ -test statistics. There were two outliers in the time to first fixation dataset ( $z = 3.77$ ,  $z = 3.38$ ) but no other outliers for the other three eye-tracking metrics. Levene's test indicated that there were minor violations of homogeneity of variance. It should be noted that behavioural data was not explored as all participants scored full marks, as predicted.

#### **6.4. Design**

A 4 within (category: humans, mammals, non-mammals and objects) x 2 between (group: TBI and control) repeated measures design was utilised to investigate potential differences in the four eye-tracking metrics; time to the first fixation, first fixation duration, total fixation duration and total fixation count.

#### **6.5. Results**

The descriptive statistics, outlined in Table 6, seemed to indicate that when the participant groups were combined, the quickest first fixation was generated for the human stimuli followed by mammals, non-mammals and objects. The same pattern was present when the groups were split, but the control group appeared to exhibit a quicker first fixation to the non-mammal compared to the mammal stimuli. The group with TBI appeared to display slower first fixations to all stimuli compared to the control group. Overall, the longest first fixation duration was for non-mammals followed by objects, humans and mammals. Individuals in the group with TBI seemed to display longer first fixation durations compared to individuals in the control group. Total fixation duration descriptive statistics indicated that both the TBI and control group fixated for longest on human stimuli followed by mammals, non-mammals and objects. However, fixation count indicated that both groups produced more fixations to the mammal stimuli followed by humans, non-mammals and objects. Overall, the group affected by TBI appeared to produce fewer and shorter fixation counts compared to the control group. The

descriptive statistics were followed up by inferential statistics to determine if any of the above observations were statistically significant.

Table 6. Descriptive statistics for eye-tracking metrics for the four stimuli categories; non-mammals, mammals, objects and humans.

Note: The ‘overall’ columns are the TBI and control groups combined.

		Time to First Fixation			First Fixation Duration			Total Fixation Duration			Fixation Count		
		<i>Overall</i>	<i>TBI</i>	<i>Control</i>	<i>Overall</i>	<i>TBI</i>	<i>Control</i>	<i>Overall</i>	<i>TBI</i>	<i>Control</i>	<i>Overall</i>	<i>TBI</i>	<i>Control</i>
<b>Stimuli</b>	<b>Non-Mammals</b>												
	<i>Mean</i>	0.55	0.71	0.39	0.27	0.29	0.25	4.83	4.54	5.14	15.17	14.10	16.31
	<i>SD</i>	0.69	0.85	0.43	0.16	0.21	0.07	1.45	1.74	1.04	4.37	4.82	3.64
	<b>Mammals</b>												
	<i>Mean</i>	0.41	0.41	0.41	0.21	0.22	0.19	5.72	5.32	6.13	17.03	15.16	19.02
	<i>SD</i>	0.61	0.67	0.56	0.14	0.19	0.07	1.76	2.20	1.06	5.66	5.68	5.05
	<b>Objects</b>												
	<i>Mean</i>	0.68	0.72	0.63	0.24	0.24	0.24	4.42	4.27	4.58	13.68	12.85	14.56
	<i>SD</i>	0.80	0.96	0.60	0.05	0.06	0.04	1.49	1.75	1.19	3.95	4.19	3.59
	<b>Humans</b>												
	<i>Mean</i>	0.29	0.36	0.21	0.22	0.22	0.22	5.64	5.29	6.02	16.48	14.94	18.11
	<i>SD</i>	0.41	0.51	0.26	0.07	0.05	0.09	1.48	1.80	0.98	4.65	4.73	4.08

### 6.5.1. Time to first fixation

There was a significant main effect of stimuli category, ( $F(2.07, 68.20) = 6.17, p = .003, \eta_p^2 = 0.16$ ) but the interaction between stimuli category and group was not significant, ( $F(2.07, 68.20) = 0.98, p = .384, \eta_p^2 = 0.03$ ). The test of between-subjects effects was also not significant, ( $F(1, 33) = 0.53, p = .471, \eta_p^2 = 0.02$ ). Post-hoc comparisons for the significant main effect of stimuli category were explored by conducting paired samples  $t$  tests (two-tailed). When the post-hoc correction was applied, participants generated significantly quicker first fixations to humans compared to non-mammals, ( $t(34), 3.63, p = .001, d = 0.61, CI = -0.12-0.41$ ) and to humans compared to objects, ( $t(34), 3.91, p \leq .001, d = 0.65, CI = 0.19-0.59$ ).

### 6.5.2. First fixation duration

There was no significant main effect of stimuli category, ( $F(1.68, 55.34) = 2.60, p = .092, \eta_p^2 = 0.07$ ) or group, ( $F(1, 33) = 0.26, p = .614, \eta_p^2 = 0.01$ ) and no significant interaction between stimuli category and group, ( $F(1.68, 55.34) = 0.55, p = .551, \eta_p^2 = 0.02$ ).

### 6.5.3. Total fixation duration

There was a significant main effect of stimuli category, ( $F(2.38, 78.39) = 56.00, p \leq .001, \eta_p^2 = 0.93$ ) but the interaction between stimuli category and group, ( $F(2.38, 78.39) = 1.74, p = .177, \eta_p^2 = 0.05$ ) and the main effect of group were not significant, ( $F(1, 33) = 1.50, p = .229, \eta_p^2 = 0.04$ ). Post-hoc comparisons for the significant main effect of stimuli category were explored by conducting paired samples  $t$  tests (two-tailed). When the post-hoc correction was applied, participants generated significantly longer fixations to humans compared to non-mammals, ( $t(34), -7.04, p \leq .001, d = 1.17, CI = -1.05 - -0.58$ ), humans compared to objects, ( $t(34), -11.54, p \leq .001, d = 1.91, CI = -1.44 - -1.01$ ), mammals compared to non-mammals, ( $t(34), -6.66, p = .001, d = 1.11, CI = -1.16 - -0.62$ ), mammals compared to objects, ( $t(34), 8.48, p \leq .001,$

$d=1.41$ ,  $CI= 0.98-1.60$ ), and finally to non-mammals compared to objects, ( $t(34)$ ,  $3.46$ ,  $p= .001$ ,  $d=0.58$ ,  $CI= 0.17-0.65$ ).

#### **6.5.4. Fixation count**

There was a significant main effect of stimuli category, ( $F(3, 99) = 24.00$ ,  $p \leq .001$ ,  $\eta_p^2 = 0.42$ ) but the interaction between stimuli category and group, ( $F(3, 99) = 2.44$ ,  $p = .069$ ,  $\eta_p^2 = 0.07$ ) and the main effect of group were not significant, ( $F(1, 33) = 3.63$ ,  $p = .065$ ,  $\eta_p^2 = 0.10$ ). Post-hoc comparisons for the significant main effect of stimuli category were explored by conducting paired samples  $t$  tests (2 tailed). When the post-hoc correction was applied, participants generated significantly more fixations to humans compared to non-mammals, ( $t(34)$ ,  $-3.07$ ,  $p= .004$ ,  $d=0.51$ ,  $CI= -2.18 - 0.44$ ) and objects, ( $t(34)$ ,  $-6.94$ ,  $p \leq .001$ ,  $d=1.16$ ,  $CI= -3.62 - -1.98$ ), mammals compared to objects, ( $t(34)$ ,  $6.11$ ,  $p \leq .001$ ,  $d=1.02$ ,  $CI= 2.24-4.47$ ), and non-mammals, ( $t(34)$ ,  $-4.09$ ,  $p \leq .001$ ,  $d=0.68$ ,  $CI=-2.79 - -0.94$ ), and to non-mammals compared to objects, ( $t(34)$ ,  $3.79$ ,  $p= .001$ ,  $d=0.63$ ,  $CI= 0.69-2.29$ ).

#### **6.6. Discussion**

The first aim of the current experiment was to explore whether TBI alters fixation patterns in comparison to controls when viewing static images of humans, animals and objects. The group variable had no effect on fixation patterns and TBI and control participants exhibited similar eye scan patterns across all the stimuli. The current findings support Douglas et al.'s (2010) research as there were no differences in fixation patterns between the two groups when viewing objects. However, a conflict in findings also occurred as Douglas et al., (2010) reported that their group of individuals with TBI generated increased number, duration and dispersion of fixations to faces compared to the control group, whereas the present group with TBI generated similar fixation patterns to matched-controls. This discrepancy may be related to methodological differences. The Douglas et al., (2010) study was centred on an emotion



identification task and the stimuli included human faces only. The current research employed more general human images that included photo shots of head and shoulders and images with whole bodies. Furthermore, participants only had to identify the stimuli category which is easier than emotion identification. A notable contrast between Douglas et al., (2010) and current published evidence exploring fixation patterns post-TBI is that Douglas et al.'s group with TBI exhibited 'hyperscanning'. The extant literature frequently documents that TBI cohorts display reduced fixations or a similar ratio of fixations but with altered fixation patterns compared to controls (Adolphs, 2005; Kenrick et al., 2017; Oatley et al., 2014; Wolf et al., 2014). However, the current experiment found no differences between the two groups. It is unclear why this distinction occurred but the central concern is that TBI may induce changes in the visual systems which, subsequently, may activate aberrant gaze patterns with marked reductions in fixations to areas of saliency (Adolphs et al., 2005, Vassallo et al., 2010). This study emphasises the need for further work into this research field as, at present, the findings are inconclusive.

The second aim of the current study was to investigate if static images of humans, mammals, non-mammals and objects evoked disparate fixation patterns. The findings indicated that the four different categories had a significant effect on time to the first fixation, with participants generating quicker first fixations to human images compared to non-mammals and objects. Furthermore, participants exhibited significantly more and longer fixations to humans and mammals compared to non-mammals and objects. These results are in line with the theory that attentional resources are directed to social stimuli (humans and mammals) compared to neutral (objects) or less-emotive stimuli (non-mammals) (Ono & Taniguchi, 2016; Simola et al., 2013).

One limitation of the present study is the heterogeneous nature of the IAPS. For example, there were other items in several of the images, which may have acted as a distracter from the stimuli

(e.g. man holding a newspaper, gecko on a leaf, spoon on the table). However, the images which were selected were based on the inclusion criteria that the target (e.g. human) was the main focus of the image. It could be argued that these types of visual scenes, i.e. non-isolated real-world images, are more representative of everyday scenarios and provide context to the stimuli. Indeed, previous research has administered images from the IAPS database to investigate the valence effect on eye movements (Acunzo & Henderson, 2011; Calvo & Lang, 2004; Nummenmaa et al., 2006).

The current study predicted that participants would produce more and longer fixations to the human images, compared to other stimuli, based on the findings that humans frequently display an innate natural preference for human faces (Frank, Vul & Johnson, 2009). It was further hypothesised that the next highest and longest fixations would be elicited by images of mammals. This prediction was based on evidence reporting a positive correlation between the degree of biological/behavioural similarity between a given species and human beings, and an increase in attitude level towards the species (Batt, 2009; Prokop & Randler, 2018; Wilson, 1984). Non-mammals were expected to produce the next highest and longest fixations followed by objects. Objects were assigned as the final category as they were thought of as mundane and lacking strong emotional reaction by the researcher. However, average scores for valence and for arousal for each of the categories (Appendix 13), calculated after the testing had taken place, indicated that human images had the highest scores followed by objects, non-mammals and finally mammals. Interestingly, objects scored slightly higher for mean dominance compared to humans (mean difference = 2.23). This may present as a confounding variable as there was high variability between the conditions (e.g. humans generated a dominance score 75% higher than mammals). However, it should be noted that the main aim of the study was to capture the natural gaze patterns of participants when they viewed different types of stimuli. In the natural world, it would be typical for individuals to encounter stimuli of differing valence that would,

in turn, elicit different levels of arousal and dominance. In this sense, the difference between the average scores for the valence, arousal and dominance levels could be seen as representing more ‘real world’ experience, rather than being seen as a confounding variable. Valence, dominance and arousal may be some of the factors that help explain natural eye-gaze differences across different categories (humans, animals, objects) but there are also other factors, such as familiarity and social importance, which may also play a role. Indeed, it might be the case that social salience is a prominent predictor for arousal values, for example. However, the exact interplay of these variables was beyond the scope of this thesis, although, it should be noted that an important point from the current findings is that human stimuli attract more attention than non-human stimuli.

This section has reviewed the literature concerning differences between ocular fixation patterns in response to faces and objects. The processing of humans appears to be more rapid and demands additional attentional resources, quantified by fixation count and duration, compared to objects (Ono & Taniguchi, 2017; Simola et al., 2013); with this effect also extending to mammals (Ono & Taniguchi, 2016; Simola et al., 2013). There were no differences between the group with TBI and matched controls fixation patterns, indicating that the individuals with TBI generated a typical number and length of fixations when viewing all of the stimuli. This finding varies from the literature which often reports that individuals with TBI display aberrant fixation patterns in comparison with control groups when viewing faces (Adolphs, 2005; Kenrick et al., 2017; Oatley et al., 2014; Wolf et al., 2014). In terms of object perception, the current results are in line with Douglas et al., (2010) who also reported that TBI and control participants generated similar fixation patterns when viewing everyday objects. Although this contrasts with autism and schizophrenia research, which documents atypical scanning patterns for both faces and objects, it appears that TBI does not impair visual strategies underpinning object perception.

Although the IAPS study explored visual strategies underpinning the basic perception of humans, animals and objects, the current research programme was also interested in more complex social cognition and the fixation patterns associated with this ability. It may be the case that, although global face recognition appears intact post-TBI, finer-grained processing of early social cues may be aberrant following TBI. Therefore, the next chapters explore mentalizing abilities and emotion and mood perception in a group of individuals who have sustained TBI and a group of individuals who have no TBI.

## **7. Chapter seven: Movie for the Assessment of Social Cognition (MASC)**

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### **7.1. Overview**

This chapter details two experiments using the MASC (Dziobek et al., 2006); a normative study and a clinical study with TBI participants. To gain a better understanding of the visual scanning mechanisms underpinning social cognition, it is vital to explore these processes in non-clinical samples. This method allows the comparison of an individual with a representative group to determine whether there are deficits in cognitive functions (Lezak, Howieson & Loring, 2004; Strauss et al., 2006). Although there is research exploring isolable constructs of social cognition (e.g. face perception, understanding emotions, thoughts and intentions) in normative groups, a comprehensive understanding of the visual strategies utilised during these different but interconnected domains is lacking (Schurgin et al., 2014; Vaidya et al., 2014). Moreover, the majority of existing evidence is based on static social stimuli so this study employed the MASC to investigate dynamic social cognition.

### **7.2. MASC normative study**

A female advantage on social cognition tasks is documented to be apparent early in life (Herlitz & Lovén, 2013), and may be underpinned by biological factors (Adenzato et al., 2017). For example, 12-month-old female infants are reported to display significantly more eye contact than age-matched males (Lutchmaya, Baron-Cohen & Raggatt, 2002) as well as more joint attention (Mundy et al., 2007). Joint attention refers to when two or more people simultaneously focus their attention on social cues, such as gestures, eye gaze or salient objects in the environment (Jones & Carr, 2004). This purported female advantage is thought to extend into adulthood (Olderbak, Wilhelm, Hildebrandt & Quidback, 2018), and is expressed as better ability on emotion recognition (Saylik, Ramen & Szameitat, 2018; Wingenbach, Ashwin

& Brosnan, 2018), theory of mind (ToM) (Adenzato et al., 2017; Wacker, Bölte & Dziobek, 2017), and empathy tasks (Christov-Moore et al., 2014; Tracy & Giummarra, 2017) compared to males. One theoretical explanation for this purported female superiority is the primary caretaker hypothesis; that females have evolved a proficiency in emotion recognition to increase offspring survival (Babchuk, Hames & Thompson, 1985). Hampson, van Anders and Mullin (2006) found that women were faster at recognising both positive and negative emotions compared to men. Hampson et al., (2006) found no evidence to suggest that the female advantage was learned through previous childcare experience or perceptual speed. The authors concluded that the observed sex differences in facial expression recognition were rooted in evolution and were not associated with domain-general learning. In contrast to Hampson et al's conclusions, Wright, Riedel, Sechrest, Lane and Smith (2018) reported that individual differences for emotion recognition accuracy was mediated by sex but also by trait emotional awareness; a trait variable associated with early learning. It is logical to presume that social cognition has evolutionary, developmental and cultural precursors. It is more than likely that these factors influence the ongoing debate and current equivocal findings regarding the supposed female advantage on social cognition tasks compared to males (Geary, 2006).

In a meta-analytic review, Thompson and Voyer (2014) investigated the purported sex differences in non-verbal emotion recognition tasks and reported a small but consistent female advantage (effect size  $d = 0.19$ ; according to Cohen's 1977 original distinction) across a range of studies. Thompson & Voyer (2014) noted that their findings might fit well with Hyde's (2005) gender similarities hypothesis; that most sex differences on psychological tasks are small to non-existent. However, the authors emphasised that care must be taken that findings are neither under or over-interpreted and that other potential moderators, such as age and type of emotion, are also considered. In other work, Grimshaw, Bulman-Fleming and Ngo (2004) reported no significant difference between males and females in emotion identification on a

signal-detection task. Calvo and Lundqvist (2008) found similar results for males and females on an emotion identification task under varied display-duration conditions. Interestingly, Russell, Tchanturia, Rahman and Schmidt (2007) reported a male advantage compared to females on the Happé ToM task (Happé, Brownell & Winner, 1999). Males were more accurate than females on both the physical and mental state ToM conditions with large and medium effect sizes (Cohen's *d*) respectively. In general, it appears that the female advantage on social tasks is small, variable, and lacking specificity (Wright, Riedel, Sechrest, Lane & Smith, 2018). What is less studied are other factors related to these proposed sex differences.

Individuals *perceive* what they *attend* to, or fixate on, so recording fixations in response to social stimuli can offer objective indices of male and female visual scanning patterns and can distinguish any sex differences. If there is variability, then this might underpin some of the reported sex differences in upstream social cognition abilities reported in the literature. Few studies have explored these basic visual processes in males and females, those that have generally support the female superiority assumption. Hall, Hutton and Morgan (2010) used eye-tracking technology to investigate emotion decoding and found that females identified facial expressions faster and more accurately than males. Fixation duration and fixation count to the eyes correlated with accuracy and reaction time, indicating that eye scan patterns informed behavioural responses similarly for both sexes. Vassallo, Cooper and Douglas (2009) recorded eye scan patterns in response to male and female faces. Both sexes exhibited similar accuracy scores across the task although females exhibited more rapid reaction times when identifying facial expressions compared to males. Both sexes fixated more on the eyes compared to other facial regions but males also fixated longer on nose and mouth regions compared to females. Vassallo et al., (2009) theorised that this affinity for less salient facial features might impede male efficiency during emotion identification.

Stoesz and Jakobson (2013) explored the processing differences between static and dynamic speeded identity and emotion expression judgements in male and female groups. The authors found disparate processing patterns between static and dynamic displays with the latter yielding longer responses in both sexes. Blais, Fiset, Roy, Saumure-Régimbald and Gosselin (2017) also compared male and female visual strategies underpinning static and dynamic facial affect identification. Blais et al., (2017) reported that participants displayed different visual strategies for the two stimuli. The dynamic condition induced fewer fixation counts to the eyes and mouth compared to the static condition in both sexes. Both Stoesz and Jakobson (2013) and Blais et al., (2017) findings suggest that there are differences in the visual strategies underpinning the processing of static and dynamic social stimuli. This finding also underlines the importance of using dynamic stimuli when exploring sex differences in emotion perception, as static stimuli might not be valid measures of ‘real-world’ vision. The subsequent study explored emotion perception as well as other mindreading abilities in a normative sample of males and females.

In conclusion, the evidence is equivocal regarding a potential female advantage on social cognition tasks. Possible reasons are task-specific demands, and/or use of non-naturalistic static stimuli and varied methodologies across studies.

### **7.2.1. Objectives and hypotheses**

The following research investigated whether males and females differed on accuracy scores on the MASC, a task that taps into several constructs of social cognition. Visual strategies in response to dynamic social interactions were also explored.

**Hypothesis 1.** Females would have higher accuracy scores on the MASC compared to males.



**Hypothesis 2.** All participants would exhibit more and longer fixations to the eyes followed by the mouth and then the nose.

**Hypothesis 3.** Males and females would exhibit different eye fixation patterns in response to the MASC stimuli.

**Hypothesis 4.** There would be fixation patterns for the different subcategories of the MASC.

### **7.2.2. Participants**

The MASC normative study was a standalone study based on a separate normative sample from the group with TBI and matched controls in the rest of the thesis. Forty-four participants, 16 male (age  $M = 22.36$ ,  $SD = 5.73$ ), and 28 female (age  $M = 20.60$ ,  $SD = 4.07$ ) (overall age  $M = 21.25$ ,  $SD = 4.75$ , range = 18-37) took part in the study. The majority of the cohort were undergraduate students ( $N = 35$ ), the remainder were recruited through a snowballing technique ( $N = 9$ ). Males and females did not differ in age ( $t(42) = 1.19$ ,  $p = .239$ ,  $d = 0.36$ ). All participants had normal or corrected to normal vision which was assessed by asking the participant. Sheffield Hallam University ethics board approved the research. All participants provided written informed consent before taking part in the research.

### **7.2.3. Procedure**

The MASC was presented on the Tobii T120 Eye Tracker. Participants were seated approximately 60-65cm away from the eye tracker screen in a stationary chair. The video was paused 45 times (after each different clip) and participants were asked questions about the characters' feelings, thoughts or intentions using a multiple-choice format (see chapter five for further details regarding the MASC design and procedure).

#### **7.2.4. Exploration of data**

Exploring histograms, box-plots and absolute z-scores for skewness and kurtosis, it was determined that the MASC behavioural accuracy data were relatively normally distributed. There were no outliers when raw scores were converted into z-scores with a cut-off point of  $\pm 3.29$ . Levene's test of homogeneity of variance was also non-significant for correct scores and the three error categories of the MASC (hypermentalizing, undermentalizing and no mentalizing). The eye-tracking data were examined with the same plots and tests. There were no outliers but there were mild to moderate violations of normality regarding skew, kurtosis and homogeneity of variance.

#### **7.2.5. Design**

A 2\*(4) ANOVA was conducted to explore any differences between male and female responses for behavioural accuracy data of the MASC. Two 2\*(3) repeated measures ANOVAs were conducted to investigate potential differences in fixation duration and count between males and females. Two further 2\*(3)\*(7) ANOVAs were conducted to test for potential differences in fixation duration and count on the subcategories of the MASC. Post-hoc corrections were applied to follow-up tests ( $\alpha = .01$ ).

#### **7.2.6. Behavioural data**

The descriptive statistics appeared to indicate that males and females exhibited similar accuracy and error scores on the MASC (Table 7).

Table 7. Descriptive statistics for males and females for the four response options during the MASC.

<b>MASC Score</b>	<b>Male Mean (SD)</b>	<b>Female Mean (SD)</b>
Correct	34.81 (3.94)	35.54 (3.27)
Hypermentalizing	4.88 (2.63)	4.57 (2.25)
Undermentalizing	3.63 (1.71)	3.75 (2.50)
No mentalizing	1.69 (1.82)	1.14 (1.18)

Results of the ANOVA showed no significant effect of sex on response selection (correct response, hypermentalizing, undermentalizing and no mentalizing), ( $F(3, 40) = 0.51, p = .677, \eta_p^2 = 0.04$ ).

### 7.2.7. Eye-tracking data

#### 7.2.7.1. Fixation duration across the MASC in seconds

Females tended to look more at the eyes compared to males whilst males appeared to display longer fixation durations to the nose and mouth (Table 8).

Table 8. Descriptive statistics for male and female fixation duration (seconds) across the three AOI set during the MASC.

<b>AOI</b>	<b>Male Mean (SD)</b>	<b>Female Mean (SD)</b>
Eyes	0.71 (0.41)	0.78 (0.49)
Nose	0.77 (0.36)	0.69 (0.23)
Mouth	0.81 (0.33)	0.70 (0.33)

There was no significant effect of AOI, ( $F(1.46, 61.13) = 0.55, p = .898, \eta_p^2 = 0.001$ ) on fixation duration, no significant interaction between AOI and sex, ( $F(1.46, 61.13) = 0.59, p$

= .506,  $\eta_p^2 = 0.01$ ) and the between-subjects test for sex was also non-significant, ( $F(1, 42) = 0.69, p = .411, \eta_p^2 = 0.02$ ).

### 7.2.7.2. Fixation duration subcategories of the MASC in seconds

Mean fixation duration scores for each subcategory of clips were obtained by collapsing together eye-tracking data for each clip tapping that construct (Table 3). Males and females performed similarly across all subcategories (Table 9).

Table 9. Descriptive statistics for male and female fixation durations (seconds) across the subcategories of the MASC.

<b>MASC Score</b>	<b>Groups Combined Mean (SD)</b>	<b>Male Mean (SD)</b>	<b>Female Mean (SD)</b>
Emotions	1.56 (0.43)	1.56 (0.36)	1.56 (0.47)
Thoughts	0.95 (0.30)	0.94 (0.29)	0.96 (0.31)
Intentions	1.27 (0.40)	1.29 (0.32)	1.25 (0.43)
Warm	1.24 (0.47)	1.20 (0.39)	1.27 (0.52)
Cold	1.03 (0.31)	1.05 (0.27)	1.01 (0.34)
Perceptive	2.59 (0.86)	2.60 (0.78)	2.58 (0.92)
Cognitive	2.54 (0.78)	2.63 (0.63)	2.49 (0.86)

There was a significant main effect of MASC subcategory, ( $F(1.54, 64.46) = 292.15, p \leq .001, \eta_p^2 = 0.87$ ) and a significant interaction between subcategory and AOI, ( $F(1.88, 79.05) = 3.94, p = .026, \eta_p^2 = 0.09$ ). There were no main effects of AOI, ( $F(1.46, 61.31) = 0.10, p = .842, \eta_p^2 = 0.002$ ) and the interactions between subcategory and sex, ( $F(1.54, 64.46) = 0.65, p = .485, \eta_p^2 = 0.02$ ), AOI and sex, ( $F(1.46, 61.31) = 0.24, p = .720, \eta_p^2 = 0.01$ ), and subcategory, AOI and sex, ( $F(1.88, 79.05) = 0.36, p = .688, \eta_p^2 = 0.01$ ) were all non-significant. The test of between-subjects effects was also not significant ( $F(1, 42) = 0.02, p = .896, \eta_p^2 \leq 0.001$ ).

Results of post-hoc comparisons of subcategory were conducted with paired-samples *t* tests (2 tailed) and are presented in Table 10. In brief, participants generated longer fixations to videos displaying emotions compared to intentions and thoughts, and to emotional compared to non-emotional visual scenes. Simple effects tests exploring the interaction between subcategory and AOI found a significant difference on the emotion subcategory between fixations to eyes ( $M = 0.74, SD = 0.40$ ) and nose ( $M = 0.41, SD = 0.24$ ) ( $p \leq .001$ ), and the eyes and mouth ( $M = 0.41, SD = 0.29$ ) ( $p = .003$ ).

Table 10. Significant post-hoc paired-samples *t* tests exploring the significant effect of subcategory in the omnibus ANOVA exploring fixation duration in seconds.

<b>Subcategory Comparison</b>	<b>Mean difference (SD) (seconds)</b>	<b><i>t</i></b>	<b>95% CI</b>	<b>Sig</b>
Emotions – Thoughts	0.61 (0.20)	20.08	0.55 – 0.67	≤ .001
Emotions – Intentions	0.30 (0.17)	11.90	0.25 - 0.35	≤ .001
Emotions – Warm	0.32 (0.14)	15.52	0.28-0.36	≤ .001
Emotions - Cold	0.54 (0.19)	18.43	0.48-0.60	≤ .001
Emotions – Perceptive	-1.02 (0.47)	-14.33	-1.17 - -0.88	≤ .001
Emotions - Cognitive	-0.97 (0.44)	-14.82	-1.11 - -0.84	≤ .001
Thoughts – Intentions	-0.32 (0.16)	-13.52	-0.36 - -0.27	≤ .001
Thoughts - Warm	-0.29 (0.23)	-8.30	-0.36 - -0.22	≤ .001
Thoughts – Cold	-0.07 (0.10)	-4.83	-0.11 - -0.04	≤ .001
Thoughts – Perceptive	-1.64 (0.59)	-18.43	-1.81- -1.46	≤ .001
Thoughts – Cognitive	-1.59 (0.51)	-20.44	-1.74 - -1.43	≤ .001
Intentions – Cold	0.24 (0.12)	13.47	0.21-0.28	≤ .001
Intentions - Perceptive	-1.32 (0.49)	-17.72	-1.47 - -1.17	≤ .001
Intentions - Cognitive	-1.27 (0.40)	-20.85	-1.40 - -1.15	≤ .001
Warm – Cold	0.22 (0.23)	6.40	0.15-0.29	≤ .001
Warm – Perceptive	-1.34 (0.44)	-20.17	-1.48 - -1.21	≤ .001
Warm – Cognitive	-1.29 (0.43)	-19.74	-1.43 - -1.16	≤ .001
Cold – Perceptive	-1.56 (0.58)	-17.80	-1.74 - -1.38	≤ .001
Cold – Cognitive	-1.51 (0.49)	-20.62	-1.66 - -1.36	≤ .001

### 7.2.7.3. Fixation count across the MASC

Females appeared to make more fixations to the eyes, nose and mouth compared to males (Table 11). From the descriptive statistics, males and females seemed to exhibit different fixations patterns, with females generating more fixations to the eyes than the nose and mouth while males made more fixation counts to the nose followed by the mouth and eyes.

Table 11. Descriptive statistics for male and female fixation counts across the three AOI set during the MASC.

AOI	Male Mean (SD)	Female Mean (SD)
Eyes	0.93 (0.75)	1.21 (1.00)
Nose	1.13 (0.55)	1.16 (0.49)
Mouth	0.95 (0.49)	1.01 (0.64)

There was no significant effect of AOI, ( $F(1.31, 55.00) = 0.47, p = .546, \eta_p^2 = 0.01$ ), no significant interaction between AOI and sex, ( $F(1.31, 55.00) = 0.34, p = .623, \eta_p^2 = 0.01$ ) and no significant between-subjects test for sex, ( $F(1, 42) = 1.56, p = .219, \eta_p^2 = 0.04$ ).

### 7.2.7.4. Fixation count subcategories

The fixation count means for each subcategory was obtained by collapsing together the eye-tracking data for each clip tapping that subcategory (Table 3). Separate analyses by sex indicated that females exhibited more fixations to all subcategories of the MASC compared to males (Table 12).

Table 12. Descriptive statistics for male and female fixation counts across the subcategories of the MASC.

<b>MASC Score</b>	<b>Groups Combined Mean (SD)</b>	<b>Male Mean (SD)</b>	<b>Female Mean (SD)</b>
Emotions	3.24 (0.95)	3.01 (0.82)	3.38 (1.01)
Thoughts	3.01 (0.84)	2.67 (0.72)	3.20 (0.87)
Intentions	3.81 (1.15)	3.42 (1.02)	4.04 (1.17)
Warm	3.93 (1.32)	3.54 (1.04)	4.16 (1.43)
Cold	3.17 (0.93)	2.86 (0.80)	3.34 (0.97)
Perceptive	7.60 (2.41)	6.76 (2.11)	8.07 (2.47)
Cognitive	7.69 (2.24)	7.00 (2.06)	8.09 (2.28)

Results of the analyses showed a significant main effect of subcategory, ( $F(1.35, 56.60) = 357.64, p \leq .001, \eta_p^2 = 0.90$ ). The main effects of AOI was non-significant, ( $F(1.27, 53.47) = 0.18, p = .735, \eta_p^2 = 0.004$ ), and the interactions between subcategory and AOI, ( $F(1.65, 69.15) = 0.56, p = .540, \eta_p^2 = 0.01$ ), subcategory and sex, ( $F(1.59, 66.96) = 2.09, p = .0141, \eta_p^2 = 0.05$ ), AOI and sex, ( $F(1.27, 53.47) = 0.29, p = .646, \eta_p^2 = 0.01$ ), and subcategory, AOI and sex, ( $F(1.65, 69.15) = 0.31, p = .690, \eta_p^2 = 0.01$ ) were all non-significant. The test of between-subjects effects was also not significant, ( $F(1, 42) = 0.02, p = .896, \eta_p^2 \leq 0.001$ ). Paired samples  $t$  tests (2 tailed) were conducted to explore the significant effect of subcategory and are presented in Table 13. In brief, participants made more fixation counts to videos displaying actors intentions compared to emotions or thoughts. Participants also elicited more fixations to warm (emotional) versus cold (non-emotional) scenes.

Table 13. Significant post-hoc paired-samples *t* tests exploring the significant effect of MASC subcategory in the omnibus fixation count ANOVA.

<b>Subcategory Comparison</b>	<b>Mean difference (SD)</b>	<b><i>t</i></b>	<b>95% CI</b>	<b>Sig</b>
Emotions – Thoughts	0.24 (0.34)	4.54	0.13-0.34	≤ .001
Emotions – Intentions	-0.57 (0.57)	-6.71	-0.74 - -0.40	≤ .001
Emotions – Warm	-0.69 (0.51)	-8.92	-0.85 - -0.53	≤ .001
Emotions – Perceptive	-4.35 (1.58)	-18.30	-4.83 - -3.87	≤ .001
Emotions - Cognitive	-4.45 (1.53)	-19.30	-4.91 - -3.98	≤ .001
Thoughts – Intentions	-0.81 (0.50)	-10.63	-0.96 - -0.65	≤ .001
Thoughts - Warm	-0.93 (0.59)	-10.38	-1.11 - -0.75	≤ .001
Thoughts – Cold	-0.16 (0.33)	-3.24	-0.26 - -0.06	.002
Thoughts –Perceptive	-4.59 (1.62)	-18.81	-5.08 - -4.10	≤ .001
Thoughts – Cognitive	-4.68 (1.53)	-20.35	-5.15 - -4.22	≤ .001
Intentions – Cold	0.65 (0.36)	12.00	0.54-0.76	≤ .001
Intentions – Perceptive	-3.78 (1.35)	-18.64	-4.19 - -3.37	≤ .001
Intentions –Cognitive	-3.87 (1.14)	-22.59	-4.22 - -3.53	≤ .001
Warm –Cold	0.77 (0.67)	7.62	0.56-0.97	≤ .001
Warm –Perceptive	-3.66 (1.24)	-19.61	-4.04 - -3.29	≤ .001
Warm – Cognitive	-3.76 (1.34)	-18.55	-4.16 - -3.35	≤ .001
Cold – Perceptive	-4.43 (1.57)	-18.73	-4.90 - -3.95	≤ .001
Cold – Cognitive	-4.52 (1.38)	-21.74	-4.94 - -4.10	≤ .001

In summary, findings showed no significant effect of sex on response selection on the MASC. The eye-tracking data indicated that there were no sex differences for the number or duration of fixations exhibited by males and females. There were significant interactions between the subcategories of the MASC and AOI on the emotion category, with both males and females displaying longer fixations on the eyes compared to the nose and mouth. Furthermore, the significant effect of subcategory signifies that clips incorporating different elements of social cognition produced different fixation durations and counts, suggesting that the task may be



sensitive to these additional socio-cognitive distinctions (although these were not different as an effect of sex).

### **7.2.8. Discussion**

This normative study investigated early visual processing in young males and females using an eye-tracking methodology in conjunction with the MASC. Research exploring potential sex differences in early attentional and visual processing in response to social stimuli is limited. Understanding the initial stage of social cognition, i.e. social perception, is imperative as disparities during this stage of the processing could explain sex differences in upstream cognitive processes that are often reported in the literature.

Males and females generated similar accuracy scores on the MASC and the tendency to hypermentalize, undermentalize, or show an absence of ToM inferences was not statistically different between the two groups. These findings do not support the theory that females possess superior mindreading and emotion perception compared to males (Wacker et al., 2017; Wingenbach et al., 2018). Moreover, the current research established no significant differences between male and female visual strategies when watching the MASC. This finding remained true even when the MASC data were segregated into the seven subcategories and behavioural errors were quantified into the separate response options.

Interestingly, there was no main effect of AOI and descriptive data revealed equivalent fixation duration for the eyes, nose and mouth with all data collapsed across the entire MASC. This finding was unexpected as the majority of eye-tracking evidence suggests that humans exhibit significantly longer dwell times on the eyes compared to other facial features (Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells, Gillespie & Rotshtein, 2016). This result may have occurred as previous research is heavily based on static stimuli which are documented to elicit differential fixation patterns compared to dynamic displays of emotions (Stoesz & Jakobson,

2013; Blais et al., 2017). It should be noted that the descriptive statistics indicated a potential difference in fixation duration patterns between the two groups. Males spent longer looking at the mouth, nose then eyes while females had longer dwell times on the eyes than the mouth and nose, although these differences were not large enough to reach statistical significance. Nevertheless, this pattern mirrors that of Vassallo et al., (2009) who reported that males spent significantly more time viewing the nose and mouth.

The subcategory analyses indicated that there were disparate fixation durations across the subcategories, although this was not related to sex differences. Fixation duration latency was longer for both groups on combined clips thought to measure perceptive (non-verbal) and cognitive (verbal) components of social cognition during the MASC. The shortest fixation durations were recorded for clips measuring thoughts about others and on cold (non-emotional) clips. All participants displayed longer fixation durations on the eyes compared to the nose and mouth on clips thought to index the emotion subcategory. Social judgements and behaviours are complex and dynamic and research exploring the visual strategies underpinning the different dimensions of social cognition (e.g. recognising emotions vs. interpreting thoughts) is limited. Conceivably, social perception and cognition rely on an amalgamation of bottom-up and top-down processes which, in turn, could result in disparate visual strategies. Bottom-up processing is theorised to be rapid and unconscious, driven by saliency to sensory input in the environment (Theeuwes, 2010). For example, emotion perception is believed to be largely automatic and driven by bottom-up processing (Carretié, Hinojosa, Martín -Loeches, Mercado & Tapia, 2004). Top-down processing is theorised to be slower compared to bottom-up processing and relies on previous experiences, learned expectations and social context, which, consecutively, shape future behaviour/decisions. Top-down processes are primarily conscious, under the control of the individual (Theeuwes, 2010), and mediate higher-order social cognition abilities, such as ToM judgements, as well as potentially playing a role in the

modulation of social perception (Cook, Barbalat & Blakemore, 2012). One possible explanation for the current finding is that the human visual-attention system is innately drawn to emotive stimuli, which is usually portrayed by facial expressions (Crocker & McDonald, 2005; Frith, 2009; Jack & Schyns, 2015). Rapidly decoding and reacting to mood states more than likely has evolutionary benefits (e.g. self-preservation).

There was no significant effect of AOI for fixation count with participants generating similar counts to the eyes, nose and mouth, although the mouth AOI was viewed least compared to the eyes and nose. When the analysis included the subcategories of the MASC, all participants displayed greater fixation counts during *perceptive* (non-verbal) and *cognitive* (verbal) clips. Similar to fixation duration, results of post-hoc analyses demonstrated that subcategory of clips could be distinguished based on fixation counts.

Essentially, the data from this initial normative study demonstrated that all participants, regardless of sex, exhibited different visual strategies for most of the MASC subcategories. Of fundamental importance is the current data demonstrated that the MASC reliably differentiates between socio-cognitive variables and induces contrasting visual strategies for fixation count and duration to facial features. This result underscores the benefits of utilising eye-tracking methodologies during the exploration of the subtle boundaries between emotion and cognition. Furthermore, the findings highlight the need for future research to take into consideration these bottom-up and top-down processes when exploring social cognition in normative and clinical cohorts.

A potential limitation of this introductory study is the ability of the analyses to detect small, possibly subtle, behavioural and/or eye-tracking differences between males and females. Whilst the study may have been powered to detect large or moderately sized differences, it may not have been powered to detect small differences. If subtle differences exist between males

and females, which may impact on social behaviour, then this needs to be addressed in future research.

In summary, there appeared to be no significant sex differences between males and females on the MASC in terms of ToM judgements and fixation patterns. Participants did elicit contrasting gaze patterns in response to the subcategories of the MASC suggesting diversity in visual attention during distinct social cognition demands/domains. These findings emphasise the necessity of evaluating each stage of social cognition processing. Russell et al., (2007) recommended that task-specific demands need to be considered when drawing conclusions about sex differences on social cognition tasks. The MASC was chosen for the current research as the task-specific demands were considered more ecologically valid compared to other social cognition assessments (Dziobek et al., 2006). Nevertheless, the current normative study investigated both low-level (visual processing) and higher-level (ToM) abilities and found no sex differences. This raises the question as to why some studies have found sex differences on social cognition assessments and whether these differences are generalisable to real-world settings. The ability to perceive, process and respond to verbal and non-verbal aspects of social cognition is vital for everyday functioning and is often impaired after brain injury (Barker, Andrade, Romanowski, Morton & Wasti, 2005). The normative study data informed the main patient by supporting the hypothesis that the MASC assessment was sensitive to different emotional and social contexts as shown by disparate gaze patterns in response to the MASC subcategories.

### **7.3. MASC TBI Study**

There is some evidence from eye-tracking research of correlations between TBI and abnormal gaze patterns in response to social stimuli (Adolphs et al., 2005, Vassallo et al., 2010; Wolf et al., 2015), although most of this evidence comes from the use of static stimuli which lacks

ecological validity. The present study compared fixation patterns and mindreading abilities of a TBI and matched-control group on the MASC. The MASC has been deemed a valuable and ecologically valid measure of social cognition (Dziobek et al., 2006; Lahera et al., 2013) and is now widely used to assess social cognition in a range of clinical populations including individuals with Asperger's syndrome (Dziobek et al., 2006; Lahera et al., 2013), borderline personality disorder (Preißler, Dziobek, Ritter, Heekeren & Roepke, 2013; Sharp et al., 2011), and schizophrenic and bi-polar populations (Neuhaus, 2013). Despite its reliability and prevalence in other research fields, the MASC has not been used in the TBI literature hence the normative study undertaken to establish task parameters, reliability and sensitivity.

### **7.3.1. Objectives and hypothesis**

The experiment investigated the differences in visual scanning behaviour between a TBI and matched-control cohort in response to the MASC.

**Hypothesis 1.** The group with TBI would exhibit significantly fewer and shorter fixation to AOI, particularly the eyes, compared to the control group.

**Hypothesis 2.** TBI participants would produce significantly fewer correct answers on the MASC compared to controls and also score higher on the three error categories.

**Hypothesis 3.** It was hypothesised that different subcategories of the MASC would elicit different fixation patterns in both the TBI and control groups.

### **7.3.2. Exploration of data**

Exploring histograms, box-plots and absolute z-scores for skewness and kurtosis, the MASC behavioural data was relatively normally distributed with one outlier ( $z$  score=3.34) on the hypermentalizing condition. Levene's test of homogeneity of variance was non-significant for

the three error categories of the MASC. Eye-tracking data were examined following the same procedure. There was one outlier in the following conditions; fixation duration mouth across the whole MASC ( $z$  score= -3.37), intentions fixation duration mouth ( $z$  score= 3.36), warm fixation duration eye ( $z$  score= 3.39), cold fixation duration eye ( $z$  score= 3.78), and cold fixation count eye ( $z$  score= 3.45). There were mild to moderate violations of normality regarding skew, kurtosis and homogeneity of variance for all eye-tracking indices.

### **7.3.3. Design**

A 2\*(4) ANOVA was conducted to explore putative differences between TBI and control responses for behavioural data of the MASC. Two 2\*(3) repeated measures ANOVAs were conducted to investigate potential differences in fixation duration and count between the TBI and control groups. Two further 2\*(7)\*3 ANOVAs were conducted to test for differences in fixation duration and count on the subcategories of the MASC. pots-hoc corrections were applied to all follow-up tests ( $\alpha = .01$ ).

### **7.3.4. Behavioural data**

As demonstrated in Table 14, the descriptive statistics indicated that the group with individuals affected by TBI had lower accuracy scores and higher error scores on the MASC compared to the control group. Statistical analyses were conducted to determine if these differences were significant.

Table 14. Descriptive statistics for the MASC accuracy scores for TBI and control groups, including the three error categories.

<b>MASC Score</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Correct	28.11 (5.66)	34.28 (4.38)
Hypermentalizing	7.39 (4.09)	4.89 (2.42)
Undermentalizing	6.94 (2.60)	3.56 (1.95)
No mentalizing	2.56 (1.95)	2.22 (1.48)

Results of the ANOVA showed a significant effect of group on response selection (correct response, hypermentalizing, undermentalizing and no mentalizing) ( $F(4, 31) = 7.23, p \leq .001, \eta_p^2 = 0.48$ ). Separate univariate ANOVAs on the response variables revealed significant differences between the TBI and control groups on the MASC correct, ( $F(1, 34) = 13.39, p = .001, \eta_p^2 = 0.28$ ), and undermentalizing responses, ( $F(1, 34) = 19.59, p \leq .001, \eta_p^2 = 0.37$ ). When post-hoc correction was applied, the hypermentalizing response was non-significant, ( $F(1, 34) = 4.98, p = .032, \eta_p^2 = 0.13$ ). The no mentalizing condition was also non-significant ( $F(1, 34) = 0.34, p = .567, \eta_p^2 = 0.01$ ).

### 7.3.5. Eye-tracking data

#### 7.3.5.1. Fixation Duration across the MASC in seconds

The group with TBI appeared to have shorter fixation durations to the eyes and nose compared to controls while both groups displayed similar fixation durations to the mouth (Table 15).

Table 15. Descriptive statistics for the TBI and control group for fixation duration across the MASC in seconds.

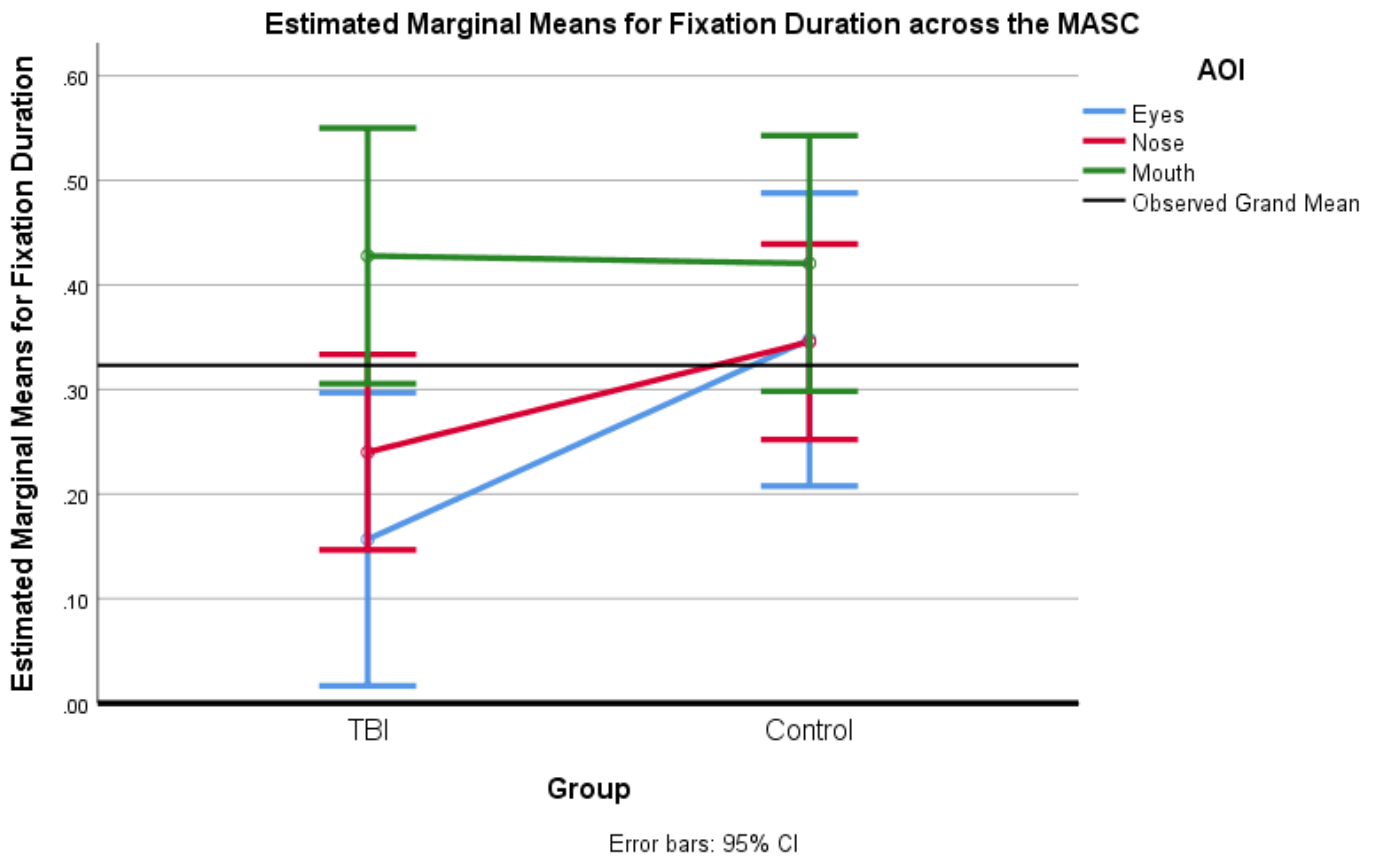
AOI	TBI Mean (SD)	Control Mean (SD)
Eyes	0.16 (0.20)	0.35 (0.36)
Nose	0.24 (0.15)	0.35 (0.23)
Mouth	0.43 (0.22)	0.42 (0.29)

The analysis showed a significant main effect of AOI, ( $F(1.51, 51.16) = 4.04, p = .034, \eta_p^2 = 0.11$ ) but the interaction between AOI and group was not significant, ( $F(1.51, 51.16) = 1.24, p = .290, \eta_p^2 = 0.04$ ). The tests of between-subjects effects was also significant, ( $F(1, 34) = 5.61, p = .024, \eta_p^2 = 0.14$ ) with the control group exhibiting significantly longer fixation durations ( $M = 1.11, SD = 0.40$ ) compared to the TBI group ( $M = 0.82, SD = 0.33$ ).

To explore the significant main effect of AOI, three paired samples  $t$  tests were conducted. When the post-hoc correction was applied, the difference between the eyes and the nose, ( $t(35), -0.80, p = .43, d = 0.36, CI = -0.14-0.06$ ), the eyes and mouth, ( $t(35), -2.15, p = .038, d = 0.13, CI = -0.33- -0.01$ ) and the nose and mouth, ( $t(35), -2.35, p = .025, d = 0.39, CI = -0.24- -0.02$ ), were all non-significant.



Graph 1. Estimated marginal means for fixation duration across the three AOI of the MASC



### 7.3.5.2. Fixation duration subcategories

The fixation duration means for each subcategory was obtained by collapsing together eye-tracking data for each clip tapping that subcategory (Table 3). Descriptive statistics in Table 16 indicated that the group affected by TBI generated shorter fixation durations to all three AOI across all of the subcategories. Inferential statistics were conducted to determine if this difference was significant.

The analysis showed a significant main effect of subcategory, ( $F(1.83, 62.08) = 15.16, p \leq .001, \eta_p^2 = 0.31$ ) but no significant effect for AOI, ( $F(1.62, 55.05) = 3.35, p = .052, \eta_p^2 = 0.09$ ). The interaction between subcategory and AOI was significant, ( $F(2.38, 80.75) = 4.18, p = .014, \eta_p^2 = 0.11$ ) and was investigated further by evaluating the simple main effects of AOI

for each of the subcategories. When the post-hoc correction was applied, the only significant differences were between the eyes ( $M= 0.18, SE= 0.35$ ) and the mouth ( $M= 0.36, SE= 0.35$ ) on the cold condition ( $p= .004$ ) and the nose ( $M= 0.22, SE= 0.03$ ) and the mouth on the cold condition ( $p= .003$ ). The interactions between subcategory and group, ( $F (1.83, 62.08) = 1.44, p= .245, \eta_p^2 = 0.04$ ), AOI and group, ( $F (1.62, 55.05) = 1.09, p= .333, \eta_p^2 = 0.03$ ) and subcategory, AOI and group, ( $F (2.38, 80.75) = 1.21, p= .309, \eta_p^2 = 0.03$ ) were not significant. The tests of between-subjects effects was significant, ( $F (1, 34) = 5.95, p= .020, \eta_p^2 = 0.15$ ) with the control participants initiating longer fixation durations ( $M= 7.10, SD= 2.94$ ) compared to the TBI participants ( $M= 5.00, SD= 2.20$ ). Post-hoc comparisons of subcategory were conducted with paired samples  $t$  tests (2 tailed) and are presented in Table 17. In sum, participants made longer fixation durations to videos displaying emotions compared to thoughts or intentions. They also made longer fixations to videos displaying warm (emotional items) compared to cold (non-emotional items) and perceptive compared to cognitive interactions.

Table 16. Descriptive statistics for the TBI and control group for fixation duration across AOI and subcategory of the MASC.

MASC Score	Groups combined Mean (SD)	TBI Mean (SD)	Control Mean (SD)
Emotions	1.00 (0.45)		
Overall		0.82 (0.36)	1.19 (0.45)
Eyes		0.16 (0.22)	0.35 (0.39)
Nose		0.24 (0.15)	0.39 (0.28)
Mouth		0.42 (0.21)	0.45 (0.32)
Thoughts	0.78 (0.30)		
Overall		0.66 (0.27)	0.91 (0.28)
Eyes		0.12 (0.12)	0.30 (0.30)
Nose		0.19 (0.12)	0.28 (0.18)
Mouth		0.35 (0.21)	0.33 (0.23)
Intentions	0.82 (0.44)		
Overall		0.69 (0.34)	0.96 (0.49)
Eyes		0.15 (0.18)	0.31 (0.33)
Nose		0.25 (0.16)	0.32 (0.22)
Mouth		0.30 (0.13)	0.34 (0.12)
Warm	0.99 (0.43)		
Overall		0.81 (0.34)	1.17 (0.44)
Eyes		0.15 (0.18)	0.38 (0.38)
Nose		0.26 (0.16)	0.38 (0.25)
Mouth		0.40 (0.20)	0.40 (0.28)
Cold	0.76 (0.30)		
Overall		0.65 (0.27)	0.87 (0.30)
Eyes		0.12 (0.12)	0.24 (0.27)
Nose		0.18 (0.13)	0.26 (0.18)
Mouth		0.35 (0.18)	0.37 (0.24)
Perceptive	0.89 (0.56)		
Overall		0.71 (0.43)	1.07 (0.62)
Eyes		0.16 (0.23)	0.34 (0.36)
Nose		0.26 (0.19)	0.36 (0.26)
Mouth		0.28 (0.12)	0.37 (0.14)
Cognitive	0.79 (0.43)		
Overall		0.65 (0.34)	0.93 (0.47)
Eyes		0.15 (0.20)	0.29 (0.32)
Nose		0.22 (0.15)	0.31 (0.22)
Mouth		0.28 (0.13)	0.32 (0.16)

Table 17. Significant post-hoc paired-samples t tests exploring the significant effect of subcategory in the omnibus ANOVA exploring fixation duration in seconds.

<b>Subcategory Comparison</b>	<b>Mean difference (SD)</b>	<b>t value</b>	<b>95% CI</b>	<b>Sig</b>
Emotions – Thoughts	0.22 (0.19)	6.89	0.16-0.29	≤ .001
Emotions – Intentions	0.18 (0.23)	4.84	0.11-0.26	≤ .001
Emotions - Cold	0.24 (0.21)	6.99	0.17-0.31	≤ .001
Emotions - Cognitive	0.22 (0.21)	6.21	0.15-0.29	≤ .001
Thoughts - Warm	-0.21 (0.18)	-6.99	-0.27- -0.15	≤ .001
Intentions – Warm	-0.17 (0.21)	-4.73	-0.24- -0.10	≤ .001
Intentions –Cognitive	0.03 (0.04)	4.84	0.02-0.05	≤ .001
Warm –Cold	0.23 (0.20)	6.77	0.16-0.30	≤ .001
Warm – Cognitive	0.200 (0.20)	6.06	0.13-0.27	≤ .001
Perceptive – Cognitive	0.10 (0.17)	3.63	0.04-0.16	≤.001

Note: See Table 16 for means and SD.

### 7.3.5.3. Fixation count across the MASC

The group with TBI appeared to generate fewer fixations to the eyes and nose compared to controls while both groups displayed similar numbers of fixations to the mouth (Table 18).

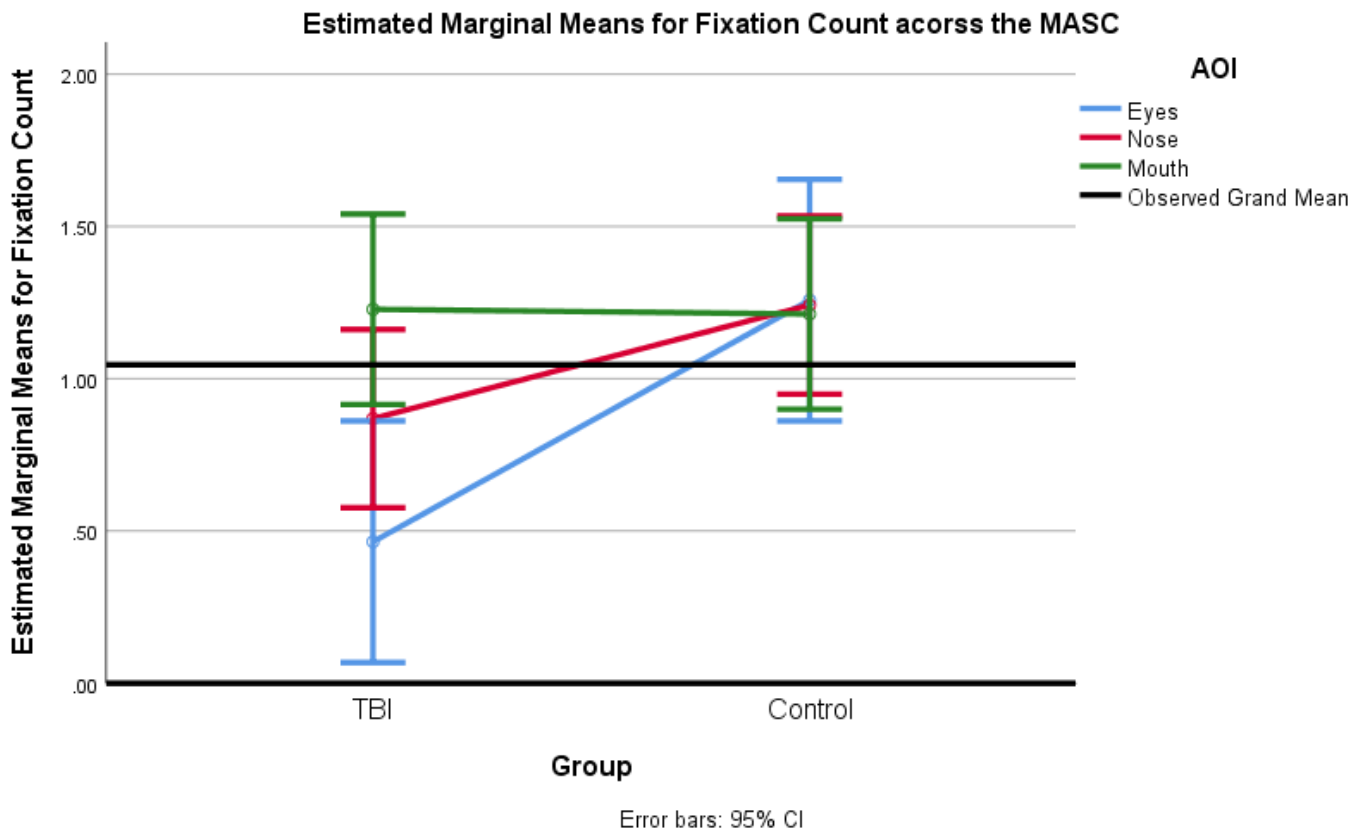
Table 18. Descriptive statistics for fixation count across the MASC.

<b>AOI</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Eyes	0.46 (0.40)	1.26 (1.10)
Nose	0.87 (0.46)	1.24 (0.73)
Mouth	1.23 (0.63)	1.21 (0.68)

The analysis showed a non-significant main effect of AOI, ( $F(1.43, 48.60) = 2.41, p = .115, \eta_p^2 = 0.07$ ) and no interaction between AOI and group, ( $F(1.43, 48.60) = 3.06, p = .072, \eta_p^2 = 0.08$ ). The tests of between-subjects effects was significant, ( $F(1, 34) = 7.60, p = .009, \eta_p^2 =$

0.18) with the control group exhibiting significantly more fixations ( $M= 3.71, SD= 1.48$ ) compared to the TBI group ( $M= 2.56, SD= 0.97$ ).

Graph 2. Estimated marginal means for fixation count across the three AOI of the MASC.



#### 7.3.5.4. Fixation count subcategories

The fixation count means for each subcategory was obtained by collapsing together eye-tracking data for each clip tapping that subcategory (Table 3). Descriptive statistics in Table 19 seemed to indicate that control participants exhibited more fixations to the eyes and nose compared to the TBI participants but both groups made similar numbers of fixations to the mouth. All participants appeared to generate a greater number of fixations to the mouth, then the nose and eyes.

The analysis showed a significant main effect of subcategory, ( $F(1.86, 63.11) = 17.68, p \leq .001, \eta_p^2 = 0.34$ ). The effect for AOI was not significant, ( $F(1.53, 51.98) = 3.04, p = .070, \eta_p^2 = 0.08$ ). The interaction between subcategory and AOI was significant, ( $F(2.44, 83.06) = 2.90, p = .050, \eta_p^2 = 0.08$ ) and was investigated further by evaluating the simple main effects of subcategory separately for the three AOI. When the post-hoc correction was applied, none of the pairwise comparisons were significant, but the difference between the eyes ( $M = 0.62, SE = 0.10$ ) and the mouth ( $M = 1.03, SE = 0.09$ ) on the cold condition was reaching significance ( $p = .012$ ). The interactions between subcategory and group, ( $F(1.86, 63.11) = 2.35, p = .108, \eta_p^2 = 0.07$ ), AOI and group, ( $F(1.53, 51.98) = 3.10, p = .066, \eta_p^2 = 0.08$ ) and subcategory, AOI and group, ( $F(2.44, 83.06) = 2.19, p = .108, \eta_p^2 = 0.06$ ) were not significant. The tests of between-subjects effects was significant, ( $F(1, 34) = 8.02, p = .008, \eta_p^2 = 0.19$ ) with control participants initiating more fixation counts ( $M = 23.53, SD = 10.55$ ) compared to the TBI participants ( $M = 15.40, SD = 6.09$ ). Post-hoc comparisons of subcategory were conducted with paired samples  $t$  tests (2 tailed) and are presented in Table 20. In brief, participants made more fixations to videos displaying emotions compared to thoughts or intentions, and to emotional compared to non-emotional displays.

Table 19. Descriptive statistics for fixation count across AOI and subcategory.

MASC Score	Groups Combined	TBI Mean (SD)	Control Mean (SD)
	Mean (SD)		
Emotions	3.08 (1.46)		
Overall		2.49 (1.03)	3.66 (1.62)
Eyes		0.42 (0.38)	1.21 (1.09)
Nose		0.86 (0.51)	1.30 (0.85)
Mouth		1.22 (0.59)	1.16 (0.68)
Thoughts	2.51 (1.00)		
Overall		2.04 (0.75)	2.97 (1.03)
Eyes		0.37 (0.30)	0.98 (0.83)
Nose		0.72 (0.34)	0.98 (0.55)
Mouth		0.96 (0.59)	1.00 (0.55)
Intentions	2.66 (1.46)		
Overall		2.07 (0.92)	3.26 (1.67)
Eyes		0.39 (0.34)	1.09 (0.99)
Nose		0.81 (0.44)	1.12 (0.65)
Mouth		0.87 (0.36)	1.05 (0.40)
Warm	3.26 (1.51)		
Overall		2.64 (1.07)	3.88 (1.65)
Eyes		0.49 (0.40)	1.31 (1.12)
Nose		0.93 (0.52)	1.37 (0.85)
Mouth		1.22 (0.60)	1.21 (0.65)
Cold	2.43 (1.01)		
Overall		2.01 (0.71)	2.85 (1.11)
Eyes		0.35 (0.26)	0.89 (0.78)
Nose		0.64 (0.33)	0.91 (0.52)
Mouth		1.01 (0.52)	1.05 (0.53)
Perceptive	2.84 (1.77)		
Overall		2.09 (1.06)	3.58 (2.04)
Eyes		0.41 (0.36)	1.19 (1.08)
Nose		0.88 (0.53)	1.23 (0.75)
Mouth		0.82 (0.34)	1.17 (0.51)
Cognitive	2.69 (1.47)		
Overall		2.06 (0.92)	3.33 (1.66)
Eyes		0.39 (0.36)	1.12 (1.00)
Nose		0.79 (0.43)	1.14 (0.66)
Mouth		0.88 (0.36)	1.06 (0.41)

Note: Overall refers to the three AOI (eyes, nose, mouth) combined.

Table 20. Significant post-hoc paired-samples t tests exploring the significant effect of subcategory in the omnibus ANOVA exploring fixation count.

Subcategory Comparison	Mean difference (SD)	<i>t</i>	95% CI	Sig
Emotions – Thoughts	0.57 (0.62)	5.51	0.36-0.78	≤ .001
Emotions – Intentions	0.41 (0.54)	4.58	0.23-0.60	≤ .001
Emotions – Warm	-0.18 (0.16)	-7.01	-0.23- -0.13	≤ .001
Emotions - Cold	0.65 (0.58)	6.63	0.45-0.84	≤ .001
Emotions - Cognitive	0.39 (0.50)	4.59	0.21-0.56	≤ .001
Thoughts - Warm	-0.75 (0.62)	-7.30	-0.96- -0.54	≤ .001
Intentions – Warm	-0.59 (0.56)	-6.43	-0.78- -0.41	≤ .001
Warm –Cold	0.83 (0.59)	8.40	0.63-1.03	≤ .001
Warm –Perceptive	0.42 (0.63)	3.98	0.21-0.63	≤ .001
Warm – Cognitive	0.57 (0.53)	6.40	0.39-0.75	≤ .001

### 7.3.5.5. Correlations

When the groups were combined, Spearman’s rank-order correlations suggested that there were no significant correlations between the behavioural scores (correct and errors) on the MASC and eye-tracking metrics ( $r$ 's  $\leq 0.32$ ,  $p \geq .055$ ). When the groups were separated, Spearman’s rank-order correlations suggested that there were no significant correlations between the behavioural scores (correct and errors) on the MASC and eye-tracking metrics ( $r$ 's  $\leq 0.43$ ,  $p \geq .072$ ) for the group with TBI but there were significant correlation between MASC ‘no mentalizing’ errors and fixation duration ( $r = .74$ ,  $p \geq .001$ ) and counts ( $r = .67$ ,  $p = .002$ ) to the nose.

### 7.3.6. Discussion

Individuals affected by TBI scored significantly lower on the MASC correct responses and significantly higher on the undermentalizing responses compared to the control group. This



finding is consistent with previous research that has reported mindreading impairments in groups living with the effects of TBI (Bibby & McDonald, 2005; Fazaeli, Yazdi, Sharifi, Sobhani-Rad & Ehsaei, 2018). It further suggests that individuals with TBI tend to undermentalize, that is, they produce insufficient mental state attributions compared to excessive or no mental state attributions. This finding is important and indicates that it may be over-simplistic to report that TBI can result in general mindreading deficits. Indeed, the group with TBI did not differ from the control group on the no mentalizing condition. This implied that the group with TBI could understand the characters internal mental states and used this knowledge to interpret behaviour but they had difficulty using this knowledge to activate relational-emotional internal representations. The current results suggest that TBI attenuates or blunts mindreading abilities rather than destroying them or exaggerating them.

Dissociation between hypermentalizing and undermentalizing is also reported in other clinical populations and may be related to the conditions symptomology. One illustration is the disparity on mindreading tasks between schizophrenic and borderline personality disorder (BPD) groups. While individuals with schizophrenia are reported to generate more undermentalizing errors, individuals with BPD generate more hypermentalizing errors (Andreou et al., 2015; Sharp et al., 2013; Vaskinn et al., 2015). These findings are in line with the typical characteristics of the conditions. For instance, schizophrenic cohorts frequently exhibit below-average social cognition abilities, e.g. facial affect recognition impairments (Grave, Soares, Martins & Madeira, 2017; Yildirim, Yalinçetin, Sevilmiş, Kutay & Alptekin, 2018) while BPD cohorts are documented to exhibit excessive social responses, e.g. often evaluating others as malevolent (Scott, Levy, Adams & Stevenson, 2011) and presenting as hypervigilant (Sieswerda, Arntz, Mertens & Vertommen, 2007).

Peyroux et al., (2019) reported that schizophrenic individuals who were experiencing positive symptoms (e.g. hallucinations, delusions) were more likely to hypermentalize while negative symptoms (e.g. apathy, blunted emotions) corresponded to undermentalizing errors. The authors theorised that the different categories of ToM errors are dependent on the clinical characteristics of the condition and stressed the necessity of measuring symptoms concerning cognitive functioning. Naturally, the distinction between a lack of mentalizing, hypermentalizing and undermentalizing could have potential ramifications for appropriate post-TBI mindreading assessments and interventions and calls for the development of more sophisticated resources.

One ambiguity of the MASC concerns the interchangeable use of the terms social cognition, ToM, mentalizing and mindreading. Although some of the core concepts are similar for the aforementioned terms, it has been argued that at least ToM and mentalizing are distinct psychological abilities in terms of theoretical conceptualisations, research findings and clinical presentation (Górska & Marszał, 2014). The original test authors refer to the MASC as a mindreading tool and a video-based instrument for the assessment of social cognition (Dziobek et al., 2006). During the Spanish validation of the MASC, the authors refer to the error differentiations as; excessive ToM, reduced ToM and total absence of mental inference (Lahera, de Salut Mar, Boada & Nozaleda, 2014) but the terms undermentalizing and overmentalizing were also included. Indeed, ToM and mindreading appear to be synonymous in some articles, for instance, Turner and Felisberti (2017) recently reported that mindreading could also be referred to as ToM.

ToM has been defined as the capability to accurately attribute mental states to others to understand and predict social behaviour (Baron-Cohen, Golan, Chakrabarti & Belmonte, 2008). ToM requires the understanding that beliefs can influence behaviour and that this

understanding or perspective must be adopted to predict what will happen. First-order mental states include thoughts such as ‘he thinks’ while second-order mental states include thoughts such as ‘he thinks she thinks’(Coull, Leekam & Bennett, 2006). ToM is most traditionally measured through false belief paradigms where participants are required to understand that others may have different mental states from themselves and may, therefore, hold a false belief (Wimmer & Perner, 1983). One example is the Sally-Anne test (Baron-Cohen, Leslie & Frith, 1985) where a participant is shown a cartoon of Sally hiding a marble in a basket. Sally then leaves the room and while she is out Anne moves Sally marble into another box. Sally then comes back into the room and the participant is asked where they think Sally will look for the marble. Fonagy and Gergely (2007) criticised the traditional definition of ToM as being too narrow, specifically in regards to the relational and emotional components of understanding others behaviour and, therefore, attempts have been made to distinguish affective and cognitive ToM (Kalbe et al., 2007, 2010).

In their recent paper, Wacker et al., (2017) defined mindreading as the social-cognitive capacity to infer other people’s emotions, cognitions and mental states. Within the mindreading umbrella, they included ‘affective processes’ such as facial affect recognition and empathy and ‘cognitive processes’ such as the attribution of thoughts and intentions, referred to as ToM, and understanding faux pas. Turner and Felisberti (2017) referred to mindreading as the ability to attribute mental states, including thoughts, intentions and emotions, to oneself and other people. They too divided mindreading into cognitive and affective components, outlined how these were dissociable at a neural level, and reported that they could be implicit (e.g. eye gaze) or explicit (e.g. mental state reasoning). As well as understanding the mental states of others, mentalizing also involves the regulation and transference of one’s emotions (Górska & Marszał, 2014). Mentalizing can be defined as a reflection on imaginative affective mental states, allowing humans to understand the behaviour of other people concerning their intentions, goals

and desires (Fonagy & Luyten, 2009; Wyl, 2014). It is thought that by understanding other people's intentions, feelings and beliefs, one can regulate their own emotions, possibly through the mirror neuron system (Sperduti, Guionnet, Fossati & Nadel, 2014). Inconsistencies also exist with mindreading assessments. For example, the Reading the Mind in the Eyes Test (RMET) is often cited as a traditional mindreading task where, in fact, Oakley, Brewer, Bird and Catmur (2016) convincingly argued that it is an emotion perception task. This distinction is significant as it could be theorised that individuals who perform poorly on the RMET could potentially be able to understand others mental states, and on the contrary, individuals who perform well could potentially display impairments in understanding mental states. Although it could be argued that emotion recognition is moderately related to mindreading tasks there are clinical dissociations between the two processes lending support to the proposal that emotion recognition and mental state inference are discrete cognitive processes (Mitchell & Phillips, 2015).

The group affected by TBI exhibited significantly fewer and shorter fixation durations to the three AOI compared to the control group across the entire MASC. This demonstrated that the individuals living with TBI generated less attention to the salient areas of the visual scene compared to the control group. Nevertheless, there did not appear to be a relationship between eye scanning patterns and the number of errors on the MASC for the group with TBI, thus suggesting that abnormal social perception (i.e. fixation patterns) does not account for impairments in mindreading abilities post-TBI. Interestingly, there were correlations between no mentalizing errors and fixation duration and counts to the nose in the control group. As the nose is argued to provide fewer diagnostic cues for how or what someone is feeling, compared to the eyes and mouth (Calvo et al., 2018; Eisenbarth & Alpers, 2011; Schurgin et al., 2014), it is unsurprising that more and longer fixations to the nose are related to increased errors. It

appears that if control participants were unable to gather the necessary cues to determine mood state, they defaulted to non-mentalizing, rather than hyper or undermentalizing.

Exploration of the descriptive statistics revealed that both groups seemed to generate longer fixation durations on the mouth compared to the eyes and the nose. Although the follow-up tests for fixation duration on AOI were non-significant when the post-hoc correction was applied, this is worth noting as the pattern is aberrant compared to typical face scanning patterns (Guo & Shaw, 2015; Keil, 2009; Laidlaw & Kingstone, 2017; Wells, Gillespie & Rotshtein, 2016). This raises concerns regarding the confounding variable of the MASC being dubbed into English and the reliability of employing the assessment in conjunction with eye-tracking technology.

The subcategories of the MASC elicited disparate fixation durations for the three AOI (eyes, nose, mouth), although this was not related to group differences. When the groups were combined, participants had more and longer fixation durations to the mouth compared to the eyes and nose during the cold condition. This could be associated with the fact that cold items rely on verbal compared to emotional cues and thus attention is drawn to the mouth to process speech. However, it could also be the result of dubbing. Fixation duration and count was longer for both groups on combined clips thought to measure emotions and warm processes during the MASC. The fewest and shortest fixation durations were recorded for clips measuring thoughts about others and on cold (non-emotional) clips. These findings mirror data from the normative study and may result from the evolution of the human visual system to implicitly generate more and longer fixation counts to emotive stimuli for self-preservation.

#### **7.4. Overall Discussion**

Originally, the MASC was made in German and has subsequently been dubbed into English. Although Dziobek et al., (2006) reported that dubbing did not interfere with participant's task

focus, and Koolstra et al., (2002) proposed that dubbing does not affect information processing, this artefact could still be considered a confounding variable. Several participants from both the normative and clinical studies commented on the dubbing and how the video and sound were discordant. While the dubbing may not have a considerable effect on behavioural data (Perego, Orrego-Carmona & Bottiroli, 2016) eye-tracking data may be more sensitive to this incongruence and may have altered the viewer's visual and cognitive processing strategies. There is an interaction between auditory and visual information during speech production and interference with this process can cause confusion. A prime example of this is the McGurk effect where a voice pronounces a consonant and a dubbed face mouths another consonant leading to the perception of a third sound (McGurk and Macdonald, 1976). Poorly synchronised audio and video can also create a 'pop-out' effect where the viewer's attention is drawn to the speaker's mouth (Smith, Batten & Bedford, 2014). One concern with employing the MASC was that participant's visual attention may have been implicitly drawn to the mouth AOI due to the dubbing. During the normative study, participants generated fewer fixations to the mouth compared to the eyes and nose. However, fixation durations to the mouth were longer compared to the eyes and nose. The clinical findings revealed that participants elicited more and longer fixations to the mouth. Therefore, it is difficult to disentangle whether results are based on typical visual strategies or whether the confounding variable of dubbing altered gaze patterns. This could explain why the typical triangular model of eye movements, known as a 'T pattern' (William & Henderson, 2007), was not documented during either of the MASC studies. Future English research wanting to employ dynamic social cognition assessments in conjunction with eye-tracking technology may consider employing non-dubbed stimuli such as the TASIT or the Edinburgh Social Cognition Test (ESCoT, Baksh, Abraham, Auyeung & MacPherson, 2018).

The MASC appears to be a reliable assessment for measuring the different dimensions of social cognition. No ceiling effects were produced during either the normative or clinical study which can sometimes be an issue when social cognition is explored in normative samples. The finding that the subcategories of the MASC generated different eye fixation patterns in both the normative and clinical studies highlights the need for research to explore the various domains of social cognition. Furthermore, the finding that the group affected by TBI were more likely to provide over-simplistic mental state attributions compared to excessive or no mental state attributions underscores the need for future research to assess error categories and to not generalise or over-simplify impairments.

While the MASC is comparatively more ecologically valid to static social cognition tasks, dynamic assessments of social cognition have been criticised for not being ‘pure’ as they draw on other cognitive processes such as executive functions. Heavey, Phillips, Baron-Cohen and Rutter (2000) comment on how naturalistic stimuli, such as videos, necessitate the participant to perceive salient social cues, retain information from across the different scenes and then integrate this information to derive a conclusion regarding mental states. This argument must be taken into consideration when working with TBI cohorts as executive functioning is often reported as impaired in the TBI literature (Barker, Andrade, Morton, Romanowski & Bowles, 2010; Barker, Andrade, Romanowski, Morton & Wasti, 2006). Heavey et al., (2000) point out this ‘contamination’ of other cognitive processes is concomitant with real-life social interactions. To reduce this confounding variable, Dziobek et al., (2006) avoided distracting (e.g. music) or prompting (e.g. direct camerawork) stimuli.

In summary, there were no sex differences between a normative group of males and females when they interpreted the MASC. This null finding included both the behavioural accuracy scores and eye scan patterns. This result is in contrast to the theory that females display a natural

superiority deciphering social interactions compared to males (Olderbak et al., 2018). However, the different subcategories of the MASC appeared to elicit contrasting gaze patterns suggesting diversity in visual attention during distinct social cognition demands/domains. In regards to the clinical MASC study, the group with TBI scored significantly lower on the MASC correct responses and significantly higher on the undermentalizing responses compared to the control group. This could indicate that individuals affected by TBI understood the characters internal mental states but they had difficulty choosing the correct response, typically displaying an attenuated or blunted selection pattern. The group with TBI made significantly fewer and shorter fixation durations to the three AOI compared to the control group across the entire MASC. Nevertheless, there was no relationship between eye scanning patterns and the number of errors on the MASC, demonstrating that abnormal social perception (i.e. fixation patterns) does not account for impairments in mindreading abilities post-TBI. Mirroring the findings from the normative study, the subcategories of the MASC elicited disparate fixation durations for the three AOI, highlighting the necessity of evaluating each stage of social cognition processing. The next chapter goes on to compare and contrast static and dynamic emotion perception in a TBI and matched control group.



## **8. Chapter eight: The Amsterdam Dynamic Facial Expression Set (ADFES)**

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### **8.1. Objectives and hypothesis**

The previous chapter explored different domains of social cognition (e.g. thoughts, feelings and intentions) during dynamic interactions. The current chapter builds upon chapter six by using simple facial expression stimuli to collect a comprehensive overview of fixation patterns in response to the six basic emotions. The ADFES (Van der Schalk et al., 2009) experiments explored and compared the visual strategies underpinning the identification of static and dynamic facial expressions in a TBI and control group. Comparisons on emotion labelling accuracy and fixation patterns were analysed, as well as reaction time during the static condition. Further investigations examined variations in the fixation patterns for the six basic emotions to determine if attention was directed to different diagnostic facial regions. The study of specific visual scan-path profiles characterised by different emotion expressions is sparse, with existing evidence proposing that eye movements may be stimulus-driven, that is, the salient properties of the emotional face (e.g. smile expressing happiness) capture attention, thereby producing differential emotion-driven fixation patterns (Schurgin et al., 2014; Vaidya, Jin & Fellows, 2014). This chapter aimed to augment the existing evidence relating to fixation profiles for the six basic emotions. Interactions between the visual scanning data, labelling accuracy and response time were also explored.

**Hypothesis 1.** The control group would be more accurate at recognising static and dynamic emotions compared to the group affected by TBI.

**Hypothesis 2.** The group with TBI would display different visual scanning patterns compared to the control group on both the static and dynamic conditions.

**Hypothesis 3.** The six basic emotions would elicit different visual scan-path profiles.

**Hypothesis 4.** The individuals with TBI would be slower at identifying emotions compared to control participants.

**Hypothesis 5.** More and/or longer fixations to AOI, particularly the eyes, would be associated with higher levels of accuracy for identifying emotions as well as quicker response times.

## 8.2. ADFES static

### 8.2.1. Participants

Due to a technical issue with the eye-tracker, one TBI participant's data was not available for the static condition. To keep sample sizes equal, the control participant for the missing TBI data was also excluded from the ADFES static analysis. Remaining participant demographic details are presented in Table 21.

Table 21. Participant demographics for the ADFES.

	<b>TBI (n = 17)</b> <i>M (SD)</i>	<b>Control (n = 17)</b> <i>M (SD)</i>	<b>Mann-Whitney Test</b>
Gender	m = 15, f = 2	m = 15, f = 2	
Current Age	44.18 (11.58)	43.00 (12.10)	$p = .708$
Age at Injury (years)	36.71 (14.17)	-	
Post Injury Years	7.47 (7.73)	-	
Years of Education	14.82 (4.38)	15.47 (3.74)	$p = .454$
*Verbal IQ	83.13 (19.08)	95.06 (8.85)	$p = .013$
*Performance IQ	92.93 (14.73)	104.29 (11.85)	$p = .027$
*Overall IQ	84.73 (17.73)	99.65 (10.61)	$p = .008$

\*Two TBI participants were unable to complete all components of the WASI and, therefore, Mann-Whitney  $U$  calculations are based on partial data.

### **8.2.2. Procedure**

The ADFES static task required participants to identify 36 static facial expressions, displayed for eight seconds, as quickly but as accurately as possible (Appendix 5). Reaction time data was collected during the static experiment by participants pressing a corresponding keyboard button. This keypress documented whether the response was correct or incorrect and it also moved the experiment on to the next image. The ADFES static experiment was administered on the Tobii T120 eye tracker. Participants were seated approximately 60-65cm away from the eye tracker screen (see chapter five for methodological details).

### **8.2.3. Exploration of data**

Exploring histograms, box-plots and absolute  $z$ -scores for skewness and kurtosis, the emotional labelling accuracy scores for the individuals with TBI was slightly skewed ( $z = 1.98$ ) but the kurtosis score was within the set parameters (see chapter five for specific details on parametric assumption checks) with no outliers. The emotion accuracy data for the control group violated the skewness ( $z = -3.58$ ) and kurtosis ( $z = 4.93$ ) parameters but this is likely related to ceiling effects. There were no outliers for the control group. Levene's statistic was significant ( $p \leq .001$ ) indicating that the data violated the assumption of homogeneity of variance. The reaction time data indicated that some conditions had skewness and kurtosis (e.g. TBI group in response to sad videos; skewness= 4.80, kurtosis= 7.60), there were no outliers in the data set but Levene's test was significant for the disgusted ( $p = .003$ ), fearful ( $p = .013$ ) and sad ( $p = .009$ ) conditions.

The data sets for the four eye-tracking metrics, that is time to first fixation, first fixation duration, total fixation duration and fixation count to the AOI (eyes, nose and mouth) were examined with the same procedure as the behavioural data. There were generally mild to moderate violations of ANOVA assumptions concerning the distribution of the data. A

minority of conditions generated greater violations, for example, the control groups first fixation duration to the mouth for videos depicting anger generated a skewness statistic of 6.81 and a kurtosis statistic of 13.85. There were no outliers but several of the conditions violated the assumption of homogeneity of variance, as indicated by significant Levene's statistics.

#### **8.2.4. Analysis**

Although the data sets violated some parametric assumptions, ANOVA was still used to analyse the data and justification for this decision can be found in the methodology section under 'considerations for reporting inferential statistics'. Two 2\*(6) ANOVAs were conducted to explore group differences for emotion labelling accuracy and reaction time on the ADFES static task. Four 2\*(6) mixed-design ANOVAs were conducted for the four eye-tracking metrics (time to first fixation, first fixation duration, total fixation duration and total fixation count). Two further 2\*(6)\*(2) mixed-design ANOVAs were conducted to explore whether participants fixated more and for longer on internal facial features compared to other areas of the visual scene. Spearman's Rho correlational analyses were conducted to explore relationships between behavioural data and eye-tracking metrics.

##### **8.2.4.1. Emotion labelling accuracy**

From the mean scores (Table 22), it appeared that the group with TBI had fewer correct answers compared to the control group during the ADFES static task. Specifically, it appears that the group affected by TBI scored lower than the control group when identifying negative emotions. The differences between the two cohorts were less pronounced during the surprised and happy conditions. There was also considerably greater variability in the performance of the individuals with TBI compared to controls as indicated by the larger standard deviations, predominately during the identification of fear and disgust. It is also unusual that a ceiling effect was not found for the group with TBI for happy faces as the ability to recognise happiness

usually remains intact after TBI. Nevertheless, variability for the happy condition is low, indicating the majority of TBI participants did score close to full marks during the recognition of happy faces.

Table 22. Mean and SD for correct scores on the ADFES static test for the TBI and control group.

<b>ADFES Score</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Overall	28.35 (6.62)	34.65 (1.50)
Anger	4.00 (1.73)	5.65 (0.86)
Disgust	4.24 (2.14)	6.00 (0.00)
Fear	3.76 (2.05)	5.41 (0.71)
Happy	5.94 (0.24)	6.00 (0.00)
Sad	4.94 (1.25)	5.71 (0.59)
Surprise	5.47 (1.23)	5.88 (0.33)

The analysis showed a significant main effect of emotion, ( $F(10.26, 110.15) = 10.26, p \leq .001, \eta_p^2 = 0.24$ ) and a significant interaction between emotion and group, ( $F(3.44, 110.15) = 5.14, p = .001, \eta_p^2 = 0.14$ ). Pairwise comparisons using simple effects test revealed that the TBI group scored fewer correct answers when identifying angry ( $p = .001$ ), disgusted ( $p = .002$ ) and fearful ( $p = .004$ ) faces compared to controls. The tests of between-subjects effect was also significant, ( $F(1, 32) = 14.61, p = .001, \eta_p^2 = 0.31$ ) with the estimated marginal means indicating that the group with TBI had significantly lower accuracy scores ( $M = 4.73, SE = 0.19$ ) compared to the control group ( $M = 5.78, SE = 0.19$ ).

#### **8.2.4.2. Reaction times**

It should be noted that only correct scores were included in the reaction time analysis. Table 23 suggests that the group affected by TBI had slower reaction times for correct answers across all six emotions as well as larger SDs compared to the control group. As expected, both groups

recognised happy faces the most rapidly with angry, fearful and sad faces producing prolonged response times.

Table 23. Descriptive statistics for reaction time for correct responses on the ADFES static test for the TBI and control group (milliseconds).

ADFES Score	TBI Mean (SD)	Control Mean (SD)
Overall	4743.61 (3080.25)	2785.24 (1206.44)
Anger	4500.35 (2673.73)	3265.03 (1704.20)
Disgust	4720.33 (4338.31)	2372.87 (851.21)
Fear	5350.31 (3322.14)	3198.37 (1772.11)
Happy	2879.02 (1122.40)	2439.28 (973.57)
Sad	5142.48 (4998.24)	2849.91 (1009.19)
Surprise	3649.15 (2026.67)	2585.99 (928.38)

There was a significant main effect of emotion, ( $F(2.41, 77.25) = 5.14, p = .005, \eta_p^2 = 0.14$ ) with pairwise comparisons, indicating that participants had quicker response times to happy faces compared to angry and fearful faces ( $p \leq .001$ ) and to surprised faces compared to fearful faces ( $p = .001$ ). There was a significant main effect of group for correct response reaction time, ( $F(1, 32) = 4.86, p = .035, \eta_p^2 = 0.13$ ) with the marginal means revealing that the group with TBI exhibited significantly longer response times compared to the control group. There was no significant interaction between emotion and group, ( $F(2.41, 77.25) = 2.21, p = .107, \eta_p^2 = 0.07$ ).

#### 8.2.4.3. Eye-tracking analyses

Overall, the TBI and control participants displayed different eye scan patterns when viewing static faces. Table 24 suggests that the control participants typically generated the first fixation on the eyes whereas individuals affected by TBI tended to fixate first on the nose. TBI participants typically displayed longer first fixation durations across all emotions suggesting

that they had a more delayed eye scan pattern compared to control participants. The control group consistently had the longest overall fixation duration for the eyes followed by the mouth and then the nose. The group with TBI displayed slightly shorter total fixation durations across the whole task, although this effect appeared minimal, and the fixation duration pattern was variable with TBI participants fixating on the eyes and the mouth for the longest periods. Control participants displayed the highest number of fixations to the eye region whereas TBI participants exhibited the most fixations to the nose region.

Table 24. Descriptive statistics for eye-tracking metrics across the six emotion conditions.

		Anger		Disgust		Fear		Happy		Sad		Surprised	
		<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>
<b>Time to first Fixation (Seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	2.98	1.43	4.14	1.79	2.99	1.24	4.17	1.58	3.70	1.22	3.33	1.25
	<i>SD</i>	2.48	1.03	2.38	1.75	2.37	1.43	2.58	1.30	2.85	1.12	2.49	1.44
	<b>Nose</b>												
	<i>Mean</i>	2.00	2.41	1.77	2.12	1.74	2.93	2.44	2.94	1.61	2.37	2.24	2.99
	<i>SD</i>	1.51	1.27	1.53	1.33	1.47	1.55	1.83	1.60	1.08	1.51	1.57	1.56
	<b>Mouth</b>												
	<i>Mean</i>	4.05	4.06	2.30	2.20	3.22	2.58	2.90	2.69	3.65	3.61	3.62	3.06
	<i>SD</i>	2.58	2.36	2.28	1.56	2.15	2.05	2.73	2.08	2.81	2.54	2.69	2.03
<b>First Fixation Duration (Seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	0.30	0.31	0.37	0.25	0.25	0.33	0.32	0.29	0.31	0.30	0.38	0.30
	<i>SD</i>	0.23	0.13	0.42	0.07	0.17	0.11	0.30	0.23	0.24	0.09	0.39	0.13
	<b>Nose</b>												
	<i>Mean</i>	0.31	0.23	0.36	0.27	0.31	0.22	0.28	0.19	0.29	0.27	0.29	0.21
	<i>SD</i>	0.34	0.08	0.21	0.08	0.12	0.09	0.19	0.09	0.12	0.14	0.15	0.07
	<b>Mouth</b>												
	<i>Mean</i>	0.27	0.22	0.30	0.32	0.29	0.29	0.37	0.32	0.29	0.23	0.27	0.25
	<i>SD</i>	0.18	0.14	0.17	0.15	0.17	0.13	0.30	0.18	0.21	0.14	0.20	0.13
<b>Total Fixation Duration (Seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	1.64	2.64	1.00	2.05	1.45	2.67	1.00	1.92	1.33	2.76	1.71	2.81
	<i>SD</i>	1.56	1.25	1.04	1.28	1.31	1.38	1.06	1.20	1.29	1.38	1.49	1.35
	<b>Nose</b>												
	<i>Mean</i>	1.14	0.67	1.19	0.82	1.32	0.77	0.91	0.54	1.31	0.79	1.06	0.55
	<i>SD</i>	0.70	0.37	0.52	0.33	0.66	0.58	0.65	0.29	0.57	0.54	0.80	0.27
	<b>Mouth</b>												
	<i>Mean</i>	0.67	0.69	1.49	1.34	0.87	1.17	1.42	1.23	0.98	0.90	0.93	1.03
	<i>SD</i>	0.63	0.84	1.07	1.05	0.67	1.02	0.97	0.85	0.91	0.92	0.88	1.02
<b>Fixation Count</b>	<b>Eyes</b>												
	<i>Mean</i>	3.29	7.54	2.16	6.35	3.42	8.14	1.97	5.90	3.08	8.16	3.40	8.19
	<i>SD</i>	2.18	3.95	1.85	3.78	2.33	4.69	1.80	3.34	2.59	4.21	2.60	4.67
	<b>Nose</b>												
	<i>Mean</i>	3.57	2.49	3.59	2.76	4.17	2.58	2.58	2.16	3.89	2.71	3.32	2.12
	<i>SD</i>	2.09	0.92	2.29	0.88	2.04	1.28	1.55	1.01	2.05	1.14	2.17	1.00
	<b>Mouth</b>												
	<i>Mean</i>	1.71	1.79	3.04	3.31	2.26	2.83	2.84	3.03	2.11	2.22	2.27	2.59
	<i>SD</i>	1.39	1.79	1.94	1.96	1.62	1.95	2.00	2.11	1.68	2.03	1.78	1.88



#### 8.2.4.3.1. Time to first fixation

The omnibus analysis showed a significant main effect of emotion, ( $F(3.51, 112.28) = 3.36, p = .007, \eta_p^2 = 0.10$ ) and significant interactions between AOI and group, ( $F(1.47, 47.16) = 6.07, p = .009, \eta_p^2 = 0.16$ ) and between emotion and AOI, ( $F(10, 320) = 8.61, p \leq .001, \eta_p^2 = 0.21$ ). There were no significant main effects of AOI, ( $F(1.47, 47.16) = 2.48, p = .109, \eta_p^2 = 0.07$ ), or of group, ( $F(1, 32) = 2.38, p = .133, \eta_p^2 = 0.07$ ) and no significant interactions between emotion and group, ( $F(3.51, 112.28) = 0.61, p = .691, \eta_p^2 = 0.02$ ) and AOI, emotion and group, ( $F(6.83, 218.57) = 1.08, p = .375, \eta_p^2 = 0.03$ ).

Simple effects tests<sup>1</sup> were conducted to investigate the significant interaction between AOI and group. The output indicated that the group with TBI took significantly longer ( $p = .002$ ) to instigate the first fixation to the eyes ( $M = 3.55$ ) compared to the control group ( $M = 1.42$ ).

To analyse the significant interaction between emotion and AOI, pairwise simple effects tests were again conducted. Participants took significantly longer to instigate a first fixation to the mouth ( $M = 4.06$ ) compared to the eyes ( $M = 2.21$ ) ( $p = .002$ ) and to the mouth compared to the nose ( $M = 2.21$ ) ( $p \leq .001$ ) when identifying angry faces. Moreover, participants exhibited a delayed first fixation to the mouth ( $M = 3.63$ ) compared to the nose ( $M = 1.99$ ) ( $p \leq .001$ ) during the recognition of sad faces.

#### 8.2.4.3.2. First fixation duration

There was no significant effect of group, ( $F(1, 32) = 1.27, p = .268, \eta_p^2 = 0.04$ ), AOI, ( $F(1.63, 52.45) = 0.97, p = .371, \eta_p^2 = 0.03$ ) or emotion, ( $F(2.62, 83.86) = 0.85, p = .0455, \eta_p^2 = 0.03$ ). No significant interactions were found between AOI and emotion, ( $F(4.22, 134.88) = 1.91, p$

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<sup>1</sup> Only significant simple effects tests are reported throughout this chapter.

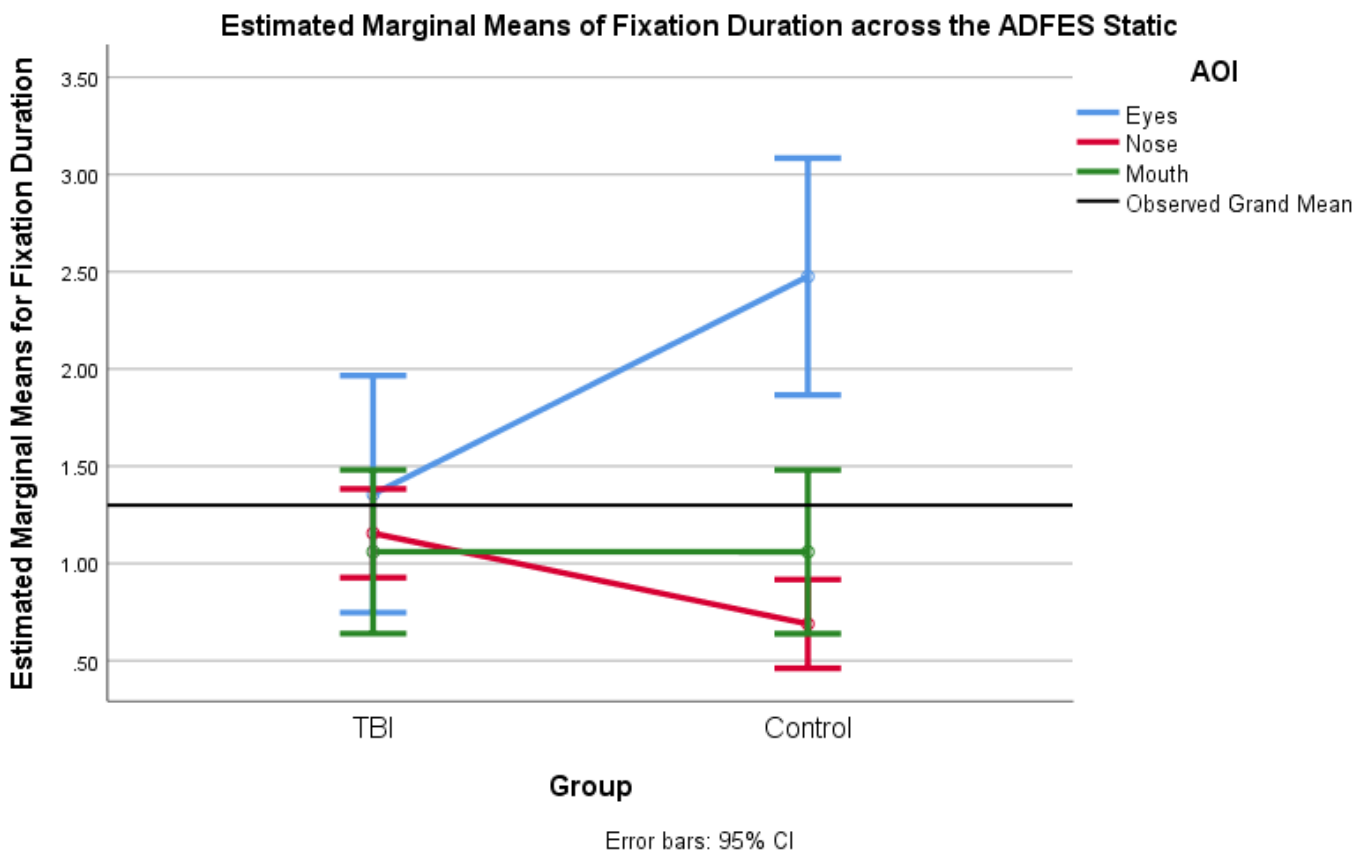
= .109,  $\eta_p^2 = 0.06$ ), AOI and group, ( $F(1.63, 52.25) = 0.50, p = .571, \eta_p^2 = 0.02$ ), emotion and group, ( $F(2.62, 83.86) = 0.55, p = .625, \eta_p^2 = 0.02$ ) or AOI, emotion and group, ( $F(4.22, 134.88) = 1.16, p = .331, \eta_p^2 = 0.04$ ).

#### **8.2.4.3.3. Total fixation duration**

There was a significant effect of AOI, ( $F(2.22, 70.93) = 9.54, p = .0001, \eta_p^2 = 0.230$ ) and emotion, ( $F(3.90, 124.71) = 4.31, p = .0003, \eta_p^2 = 0.12$ ) as well a significant interaction between AOI and emotion, ( $F(8.54, 273.26) = 12.63, p \leq .001, \eta_p^2 = 0.28$ ) and between AOI and group, ( $F(2.22, 70.93) = 4.76, p = .004, \eta_p^2 = 0.13$ ). The interactions between emotion and group, ( $F(3.90, 124.71) = 0.46, p = .761, \eta_p^2 = 0.01$ ) and AOI, emotion and group, ( $F(8.54, 273.26) = 1.68, p = .096, \eta_p^2 = 0.05$ ) were not significant. Additionally, no significant main effect of group was found, ( $F(1, 32) = 0.15, p = .703, \eta_p^2 = 0.005$ ).

The significant interaction between AOI and group was further analysed using simple effects tests. Pairwise comparisons suggested that there was a significant difference between the TBI ( $M=1.16$ ) and control ( $M=0.69$ ) group on the nose AOI ( $p = .006$ ), signifying that TBI participants exhibited significantly longer fixation durations to the nose compared to controls. There was no significant difference for the fixation duration on the eyes between the TBI ( $M=1.36$ ) and control ( $M = 2.48$ ) groups ( $p = .013$ ).

Graph 3. Estimated marginal means for Fixation Duration across the ADFES Static.



The interaction between AOI and emotion was also investigated with simple effects analyses and the results indicated that for angry, fearful, sad and surprised faces there were significantly longer fixations to the eyes compared to the nose and mouth (all  $p$ 's  $\leq .003$ ). Happy faces produced longer fixation durations to the eyes ( $M = 1.46$ ) compared to the nose ( $M = 0.72$ ) ( $p = .005$ ) and longer durations to the mouth ( $M = 1.33$ ) compared to the nose ( $p = .001$ ).

#### 8.2.4.3.4. Fixation duration for internal facial areas vs. external areas

Internal facial areas included the eyes, nose and mouth while external areas included all other areas of the face (i.e. the chin, forehead and cheeks) and the rest of the visual scene (i.e. the shoulders and chest of the actor and the background area of the image).

There was a significant effect of AOI, ( $F(1, 32) = 36.85, p \leq .001, \eta_p^2 = 0.54$ ) and emotion, ( $F(3.90, 124.71) = 4.31, p = .003, \eta_p^2 = 0.12$ ) as well a significant interaction between AOI and emotion, ( $F(4.41, 141.19) = 5.88, p \leq .001, \eta_p^2 = 0.16$ ). There was no significant interaction between emotion and group, ( $F(3.90, 124.71) = 0.46, p = .761, \eta_p^2 = 0.01$ ), AOI and group, ( $F(1, 32) = 3.02, p = .092, \eta_p^2 = 0.09$ ) or emotion, AOI and group, ( $F(4.412, 141.193) = 2.13, p = .074, \eta_p^2 = 0.06$ ). There was also no significant main effect of group, ( $F(1, 32) = 0.15, p = .703, \eta_p^2 = 0.01$ ). Table 25 indicated that participants generated shorter fixation durations to internal facial features in response to happy faces compared to negative and surprised faces. It was also evident that participants displayed longer fixation durations to external areas compared to internal areas when viewing angry faces.

Table 25. Significant simple effects analysis exploring the interaction between AOI and emotion (fixation duration in seconds).

<b>Emotion</b>	<b>Mean difference (SE)</b>	<b>Sig.</b>
Internal		
Fearful-Angry	-0.40	.009
Disgusted-Happy	0.44	.008
Fearful-Happy	0.62	.001
Sad-Happy	0.52	.001
Surprised-Happy	0.11	.001
External		
Angry-Disgusted	0.51	.001
Angry-Sad	0.46	.001
Angry-Surprised	0.41	.001

Note: The emotion listed first in the table generated the longest fixation duration.

#### 8.2.4.3.5. Fixation count

There was a significant effect of AOI, ( $F(2.04, 65.24) = 17.53, p \leq .001, \eta_p^2 = 0.35$ ) and emotion, ( $F(5, 160) = 9.60, p \leq .001, \eta_p^2 = 0.23$ ) but the between subjects factor of group was

not significant, ( $F(1, 32) = 4.00, p = .054, \eta_p^2 = 0.11$ ). There was a significant interaction between AOI and group, ( $F(2.04, 65.24) = 9.13, p \leq .001, \text{partial } \eta_p^2 = 0.22$ ), AOI and emotion, ( $F(8.65, 276.77) = 10.51, p \leq .001, \eta_p^2 = 0.25$ ) and between AOI, emotion and group, ( $F(8.65, 276.77) = 2.09, p = .033, \eta_p^2 = 0.06$ ). No significant interaction was found between emotion and group, ( $F(5, 160) = 1.37, p = .0240, \eta_p^2 = .04$ ).

To explore the significant interaction between AOI, emotion and group two 6 within (emotion: happy, surprise, fear, anger, sad, and disgust) x 3 within (AOI: eyes, nose and mouth) repeated-measures ANOVAs were conducted with the groups split. The ANOVA for the TBI group indicated a significant effect of emotion, ( $F(5, 80) = 5.54, p \leq .001, \eta_p^2 = 0.26$ ) and a significant interaction between emotion and AOI, ( $F(6.43, 102.86) = 7.61, p \leq .001, \eta_p^2 = 0.32$ ). The effect of AOI was not significant, ( $F(1.40, 22.44) = 1.53, p = .237, \eta_p^2 = 0.09$ ). To further investigate the interaction between AOI and emotion, paired-samples comparisons through simple effects analysis were examined. The data in Table 26 suggested that the TBI participants made more fixations to the nose compared to the mouth during the viewing of angry, fearful and sad faces.

Table 26. Significant post-hoc tests exploring the interaction between AOI and emotion for the TBI group.

<b>Emotion</b>	<b>TBI Group Mean difference (SE)</b>	<b>Sig</b>
Anger		
Nose-Mouth	1.86 (0.51)	.002
Fear		
Nose-Mouth	1.90 (0.54)	.003
Sad		
Nose-Mouth	1.78 (0.48)	.002

Note: The AOI listed first in the table received the most fixation counts.

The ANOVA for the control group indicated a significant effect of emotion, ( $F(5, 80) = 7.94, p \leq .001, \eta_p^2 = 0.33$ ) and AOI, ( $F(1.17, 18.66) = 9.60, p \leq .001, \eta_p^2 = 0.53$ ) and a significant

interaction between emotion and AOI, ( $F(4.53, 72.50) = 10.89, p \leq .001, \eta_p^2 = 0.41$ ). To further investigate this interaction, parried-samples comparisons through simple effects analysis were examined, with significant results presented in Table 27. The results suggested that control participants made more fixations to the eyes compared to the nose and mouth across all six emotions.

Graph 4. Estimated Marginal Means for Emotion\*AOI\*Group for Fixation Count for the Group with TBI

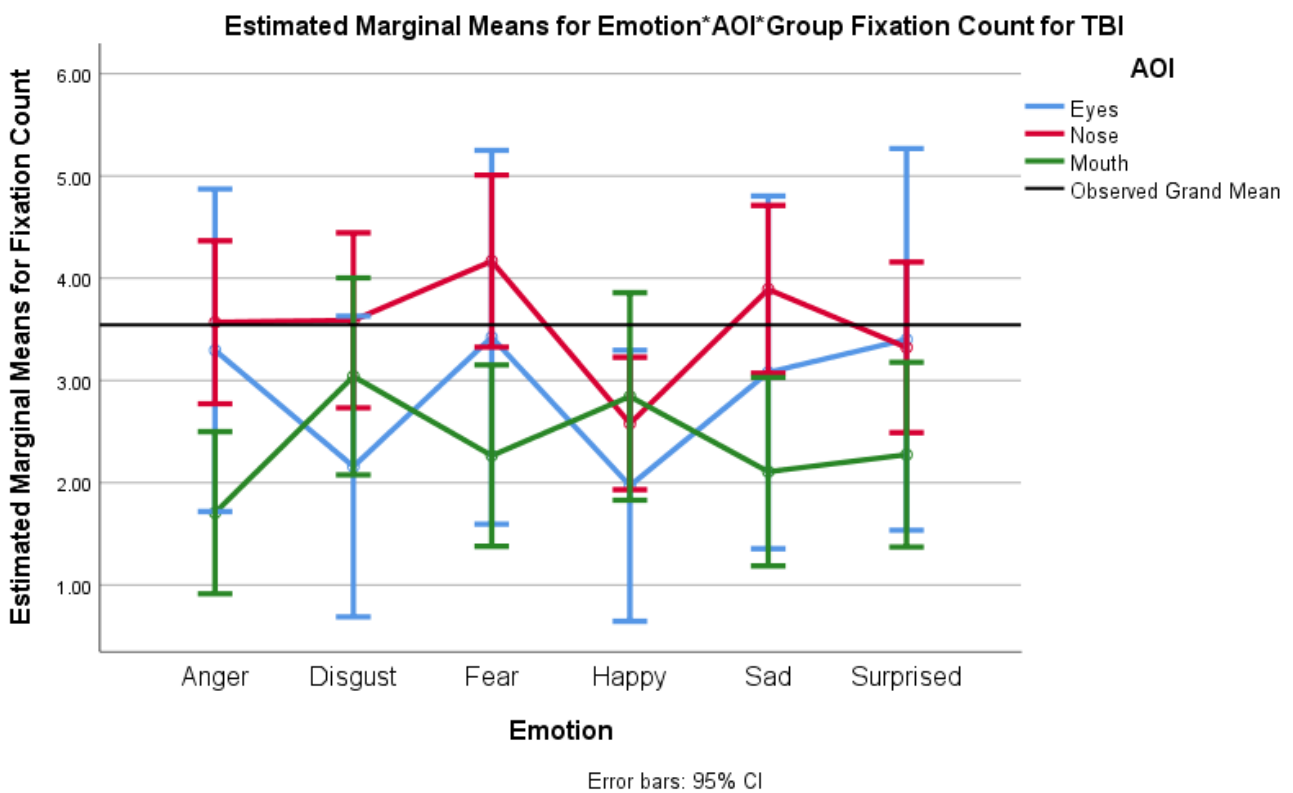
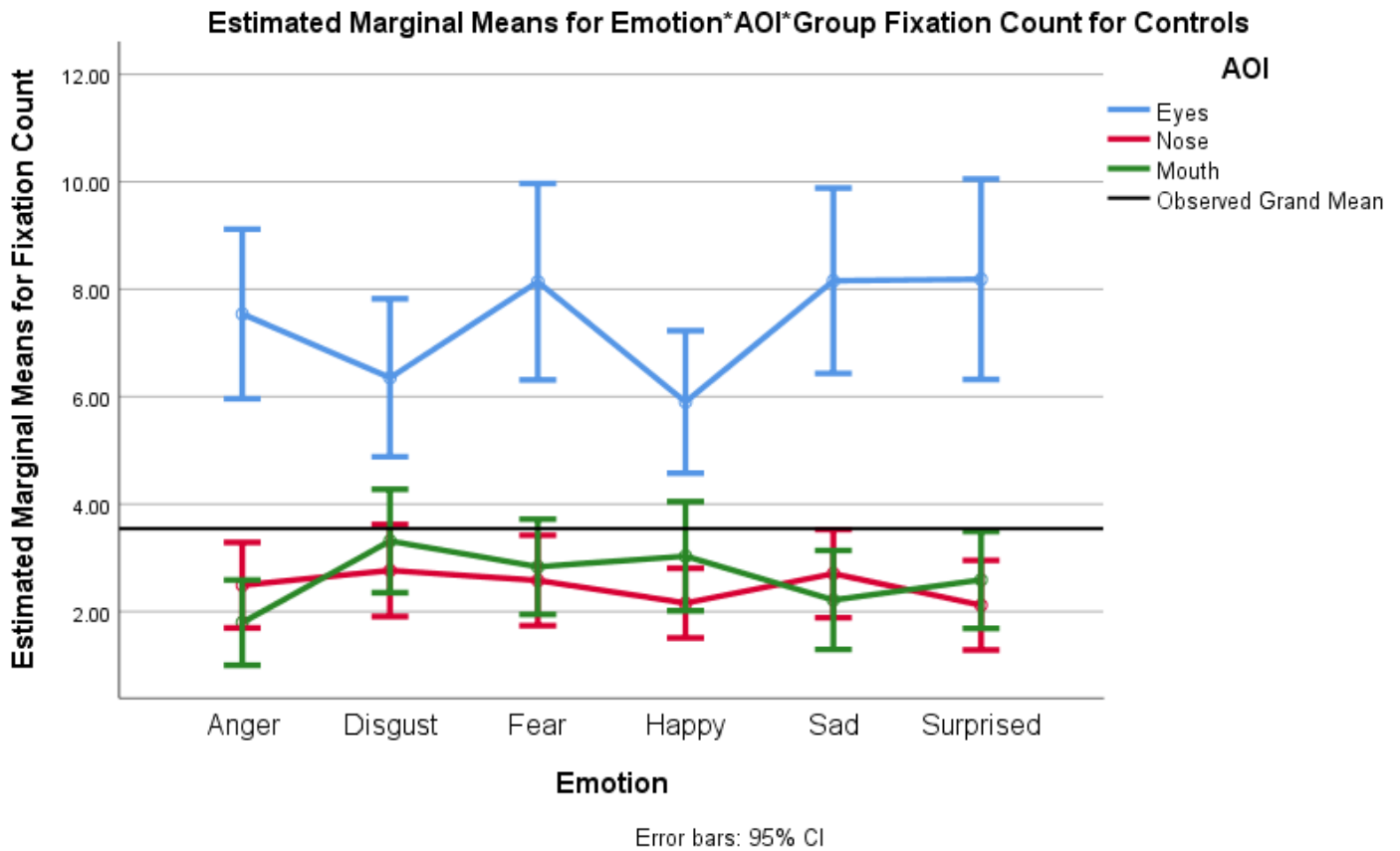


Table 27. Significant post-hoc tests exploring the interaction between AOI and emotion for the control group (fixation count).

<b>Emotion</b>	<b>Control Group Mean difference (SE)</b>	<b>Sig.</b>
Anger		
Eyes-Nose	5.05 (1.06)	.001
Eyes-Mouth	5.75 (1.16)	.001
Disgust		
Eyes-Nose	3.59 (0.93)	.001
Fear		
Eyes-Nose	5.56 (1.32)	.001
Eyes-Mouth	5.31 (1.35)	.001
Happy		
Eyes-Nose	3.74 (0.81)	.001
Eyes-Mouth	2.87 (0.97)	.009
Sad		
Eyes-Nose	5.45 (1.16)	.001
Eyes-Mouth	5.94 (1.29)	.001
Surprise		
Eyes-Nose	6.07 (1.19)	.001
Eyes-Mouth	5.60 (1.33)	.001

Graph 5. Estimated Marginal Means for Emotion\*AOI\*Group for Fixation Count for the Group with TBI



#### 8.2.4.3.6. Fixation count for internal facial areas vs. external areas

There was a significant effect of AOI, ( $F(1, 32) = 29.81, p = .001, \eta_p^2 = 0.48$ ) and emotion, ( $F(5, 160) = 9.60, p = .001, \eta_p^2 = 0.23$ ) as well a significant interaction between AOI and emotion, ( $F(5, 160) = 14.69, p \leq .001, \eta_p^2 = 0.20$ ) and between AOI and group, ( $F(1, 32) = 7.73, p = .009, \eta_p^2 = 0.13$ ). The interactions between emotion and group, ( $F(5, 160) = 1.37, p = .24, \eta_p^2 = 0.04$ ) and AOI, emotion and group, ( $F(5, 160) = 1.61, p = .161, \eta_p^2 = 0.05$ ) were not significant. The main effect of group was not significant, ( $F(1, 32) = 4.00, p = .054, \eta_p^2 = 0.11$ ).

Simple effects analysis exploring the AOI by emotion interaction indicated that participants, as a whole, exhibited significantly more fixations to the internal facial features compared to



external areas across all six emotions ( $p$ 's  $\leq .001$ ). To explore the significant interaction between AOI and group simple effects tests were again conducted and demonstrated that the control group ( $M = 12.48$ ) made significantly more fixation to the internal AOI compared to the TBI group ( $M = 8.78$ ) ( $p = .004$ ).

#### **8.2.4.4. Correlations**

To explore potential relationships between visual fixation patterns and behavioural data, correlational analyses were conducted. Spearman's Rho correlations were generated by combining both TBI and control participants into one group to explore the overall relationships of the data. The post-hoc correction  $\alpha$  level of .01 was applied to correlational analyses. The only significant relationship was between ADFES emotion labelling accuracy and response time ( $r = -0.44$ ,  $p = .004$ ). See Appendix 17 for correlational matrix. When the groups were separated out, there were no significant correlations for either the group with TBI ( $r = -0.56$ ,  $p = .019$ ) or the control group ( $r = -.27$ ,  $p = .298$ ).

### **8.3. Discussion**

The visual strategies underpinning facial affect recognition have been extensively investigated in normative (Schurgin et al., 2014; Vassallo et al., 2009) and clinical cohorts, particularly in autistic (Black et al., 2017; Klin et al., 2002) and schizophrenic (Asgharpour, Tehrani-Doost, Ahmadi & Moshki, 2015; Bortolon et al., 2016; Sasson et al., 2015) groups. However, research exploring fixation patterns in response to static facial affect post-TBI is scant. The present research investigated the visual scanning patterns of TBI and control participants when they viewed photographs of facial expressions displaying six different emotions (anger, disgust, fear, happy, sad and surprise).

Overall, the TBI participants were less accurate and slower at identifying static facial expressions compared to control participants. This finding is consistent with existing empirical work documenting that TBI is associated with impairments in facial affect recognition (Babbage et al., 2011; Biszak & Babbage, 2014; May et al., 2017) and delayed response times (Celeghin et al., 2019; Ietswaart et al., 2008). Slower reaction times may be equated to secondary effects of impaired neuropsychological functioning such as slowed processing speed or attention deficits, which are well documented after TBI (Miotto et al., 2010; Dymowski, Owens, Ponsford & Willmott, 2015). Alternatively, delayed reaction times could be associated with aberrant eye scan patterns. For instance, if the TBI participants were failing to fixate on the most salient AOI, namely the eyes and the mouth, then it would lead to protracted reaction times (Wells et al., 2016). However, this assumption was not supported by the correlational analysis, although the non-significant findings could be equated to the stringent post-hoc correction which was applied. Curtailed  $\alpha$  levels can increase the risk of Type II errors and loss of power, particularly if there are numerous comparisons (Field, 2009; Kim, 2015; Krzywinski & Altman, 2014; Perneger, 1998; Rothman, 1990), and this should be taken into consideration when evaluating the current findings. It should be noted that measuring TBI participant's reaction times has been deemed problematic as their responses are often delayed and, therefore, reliability of the task could be affected (Celeghin et al., 2019). Although the current study intended to circumvent delayed reactions by adopting a simple and consistent methodology, findings should still be interpreted with caution.

The control group consistently exhibited a rapid first fixation to the eyes across all emotions while the group affected by TBI took significantly longer to instigate this first fixation to the eyes. Laidlaw and Kingstone (2017) proposed that the eyes are the most important facial feature due to the amount of social and emotional information which can be extracted from the area. The finding that control participants displayed natural gravitation towards the eyes corroborates

with research suggesting that human beings naturally focus attention on the most salient area of the visual scene (Wells et al., 2016). The group affected by TBI displayed an irregular gaze pattern compared to controls with quicker first fixations to the nose and subsequent sporadic fixations to the mouth and eye regions. The nose provides fewer emotive cues during facial expression identification compared to the eyes and mouth (Wells et al., 2016). It is not clear why the TBI participants routinely made the first fixation to the nose. One explanation could be that the group with TBI displayed screen centre bias (Bindemann, 2010; Tatler, 2007) which would roughly correspond to the position of the nose (Barabanschikov, 2015; Scheller, Büchel & Gamer, 2012). Alternatively, it could be argued that individuals with TBI were exhibiting similar markers to autistic individuals by implicitly avoiding eye contact; this theory is known as the Eye Avoidance Hypothesis (Madipakkam, Rothkirch, Dziobek & Sterzer, 2017). Tanaka and Sung (2016) suggested that atypical eye contact displayed by autistic individuals could be an adaptive avoidance response as the eyes are viewed as socially threatening. Research by Singh et al., (2016) highlighted the pathophysiological similarities between paediatric TBI and autistic symptoms, including poor facial affect recognition. The authors outlined the resemblance between subcortical and sensory cortical disruption in both cohorts as well as decreased white matter in similar brain areas. The aberrant eye scan patterns of the group living with TBI could result from disrupted cortical pathways underpinning saccade or fixation generation, thus leading to an inability to appropriately evaluate the social threat.

There were no significant differences between the TBI and control groups for first fixation duration. This is an insightful finding as it indicated that fixations for the group with TBI were not impaired per se but rather the pattern of the fixations was aberrant and deviated from the natural eye scan pattern of the control group. Turkstra (2005) likewise reported that there were no significant quantitative differences between TBI and control participants gaze times and discussed the possibility of a qualitative distinction between visual strategies among the groups.

However, Turkstra's (2005) interpretation must be taken with caution as she did not employ standardised eye-tracking techniques in her research and instead visually calculated the percentage of time that each participant displayed eye to face gaze towards a partner. Furthermore, Turkstra's (2005) cohort consisted of adolescents and young adults who may display different eye scan patterns compared to adults (Ebner, He & Johnson, 2011). Additionally, current formulations make distinctions between impairments associated with brain injuries acquired during childhood, adolescence and adulthood (Barker et al., 2010), therefore, the generalisability of Turkstra's (2005) findings to adult TBI populations are debatable.

Participants exhibited the longest fixation durations on the eyes followed by the mouth and then the nose. This finding is in line with previous eye-tracking research which has reported that the eye region of the face is generally the most fixated area (Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells et al., 2016). Humans typically display a triangular model of eye movements, known as a 'T pattern', where they alternate between fixating on the eyes and the mouth (Smith et al., 2005; William & Henderson, 2007). This 'T pattern' was established in the present research when the control data was explored using heat maps (Appendix 16), but the same pattern was non-existent in the TBI data. The group affected by TBI did display longer fixation durations to the eyes, although the total duration was almost half compared to controls, followed by the nose and then the mouth. Additionally, while the control group made more fixations to the eyes, the group with TBI tended to fixate more on the nose. This pattern is inconsistent with the 'T pattern' and is more in line with Peterson and Eckstein's (2012) downward shift theory of saccadic eye movement. Fixations to the eye region have both a social (joint attention) and perceptual function (identification of mood state). Decreased fixation duration to the eye region could have potentially adverse effects on social functioning. Firstly, emotion perception would be slower or emotion state could go unnoticed, and secondly,

maintaining sufficient eye contact preserves social value and precipitates social relationships (Peterson & Eckstein, 2012). Peterson and Eckstein (2012) included a free viewing and a forced viewing condition during their normative face-scanning research. During the free viewing condition, participants viewed a human face with no instructions, therefore allowing them to exhibit their natural visual scanning pattern. During the forced fixation condition, participants were instructed to fixate on an ill-favoured facial feature (e.g. forehead). Peterson and Eckstein (2012) reported that the forced condition produced a detrimental effect on emotion perception performance, leading the researchers to conclude that human gaze behaviour is primed to optimize evolutionary performance and deviations from this are disadvantageous. As the eyes are frequently reported to be the most salient feature for identifying mood state (Wells et al., 2016), the group affected by TBI aberrant fixation duration pattern to the nose compared to controls could explain the group with TBI lower accuracy scores.

Two eye-tracking studies (Vassallo et al., 2010, 2011) reported that individuals with TBI exhibited a more widespread scanning strategy and made similar fixation durations to both internal and external facial areas compared to controls. However, in the current research, both the TBI and control group spent significantly longer fixating on internal facial features compared to external areas. This finding conforms with existing research documenting that internal facial features (eyes, nose, mouth) are more attended to compared to external features (hair, face, forehead, ears) during social interactions (Mehouard et al., 2014; Schurgin et al., 2014). Although the current group of individuals with TBI fixated for longer on internal facial features, they did not appear to be fixating on the most salient internal features (the eyes), but instead displayed longer fixation durations on less important features (the nose), indicating that TBI disrupts fixation patterns. Moreover, the group affected by TBI made significantly fewer fixations to internal facial features compared to controls across all six emotions.

In conclusion, the group with TBI performed significantly poorer compared to the control group on the facial expression identification task. Furthermore, the group with TBI also displayed slower reaction times for correct responses compared to the control group. There was a significant difference between the TBI and control participants' fixation patterns concerning the number, duration and pattern of fixations. In terms of fixation patterns, the group affected by TBI took significantly longer to instigate the first fixation to the eyes compared to the control group. This finding is pertinent as it is theorised that the eye region reveals the most detail about mood state (Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells et al., 2016) and, therefore, delayed fixation to that regions could arguably lead to delayed or erroneous emotion recognition. Interestingly, there were no group differences for the first fixation duration, indicating that this particular eye-tracking metric remained intact and appropriate post-TBI. However, there was a significant difference between the two groups for total fixation duration and fixation count, with the TBI participants generating more and longer fixation durations to the nose compared to controls. The results also signified that the control group exhibited more and longer fixations to the eyes compared to the group with TBI. This finding links in with research from Adolphs et al., (2005) and Oatley et al., (2014) who reported that individuals with TBI appeared to naturally avoid the eye region of the face when participating in facial affect recognition tasks. The correlations between the behavioural data and the eye-tracking metrics were all non-significant once the multiple comparisons correction was applied. This suggests that the group affected by TBI poor emotion labelling accuracy and slower response times were not related to aberrant fixation patterns.

## **8.4. ADFES dynamic**

### **8.4.1. Participants**

The ADFES dynamic analysis was based on the full cohort of participants recruited for the present research (TBI group  $n = 18$ , control group  $n = 18$ ). Participant demographics can be found in chapter five.

### **8.4.2. Procedure**

The ADFES dynamic task, as described in more detail in chapter five, required participants to identify 36 facial expressions by watching video clips of an actor displaying a neutral face for 0.5 seconds before the onset of the emotion began. Once the emotion reached the apex, the pose was held for five seconds and then the answer screen would appear. The participant would verbally tell the researcher which emotion they believed the actor was displaying and then the researcher would move the experiment on by pressing the spacebar (Appendix 6). Parallel to the ADFES static experiment, the dynamic experiment was administered on the Tobii T120 eye tracker. Participants were seated approximately 60-65cm away from the eye tracker screen. No reaction time data was recorded for the dynamic task.

### **8.4.3. Exploration of dynamic data**

To explore data trends and to check parametric assumption criteria, distribution plots and statistics were examined. For the dynamic condition, plots indicated that data for correct scores was relatively normal with no significant scores on the  $z$ -test and no outliers. The assumption of homogeneity of variance was violated but as sample sizes were equal the effect was mitigated. Plots indicated several eye-tracking variables which had moderate and severe skew and kurtosis which was confirmed by several  $z$ -test scores over 1.96. There were no outliers in the time to first fixation or fixation count conditions but there were four outliers in the first

fixation duration condition and four in the total fixation duration conditions. A minority of variables violated the assumption of homogeneity of variance. Parallel to the static ADFES analysis, ANOVA was employed to compare eye-tracking metrics as the analysis is documented to be robust to moderate violations of normality (Donaldson, 1968; Glass et al., 1972; Kim, 2013).

#### 8.4.4. Analysis

The same design was used to analyse both the ADFES static and dynamic tasks.

##### 8.4.4.1. Emotion labelling accuracy

The descriptive statistics in Table 28 appeared to indicate that the group with TBI scored lower on the emotion labelling task compared to the control group, with particularly low scores across the negative emotions. Furthermore, the group affected by TBI displayed greater performance variability compared to controls, as indicated by the larger standard deviations, predominately during the identification of anger, fear and disgust.

Table 28. Mean and SD for correct scores on the ADFES dynamic test for the TBI and control group.

<b>ADFES Score</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Overall	28.39 (4.50)	32.89 (2.22)
Anger	4.28 (2.02)	5.72 (0.57)
Disgust	3.94 (1.55)	5.39 (1.20)
Fear	3.44 (1.62)	4.78 (0.94)
Happy	5.94 (0.24)	6.00 (0.00)
Sad	4.78 (0.81)	5.06 (0.73)
Surprise	5.72 (0.67)	6.00 (0.00)



The analysis showed a significant main effect of emotion, ( $F(3.58, 121.87) = 18.85, p \leq .001, \eta_p^2 = 0.36$ ) and a significant interaction between emotion and group, ( $F(3.58, 121.87) = 4.10, p = .005, \eta_p^2 = 0.11$ ). The tests of between-subjects effect was also significant, ( $F(1, 34) = 18.88, p \leq .001, \eta_p^2 = 0.36$ ) with the estimated marginal means indicating that the group with TBI had significantly lower accuracy scores ( $M = 4.69, SE = 0.13$ ) compared to the control group ( $M = 5.49, SE = 0.13$ ).

Pairwise comparisons using simple effects test revealed that the group with TBI scored fewer correct answers when identifying angry ( $p = .006$ ), disgusted ( $p = .004$ ) and fearful ( $p = .005$ ) faces compared to controls.

#### **8.4.4.2. Eye-tracking data**

The statistics in Table 29 depict that overall, control participants consistently fixated on the eyes first and then moved onto the nose or mouth. The TBI group tended to fixate on the mouth first, except during the disgusted condition where the first fixation was directed to the eyes, followed by a variable pattern between the eyes and nose. There was a clear pattern concerning total fixation duration where the control group had longer fixations on the eyes and mouth across all emotions, while the group affected by TBI displayed a more variable pattern, mainly fixating for the longest on the eyes but fluctuating between the nose and mouth. This pattern was mirrored for fixation count where the control participants made more fixations on the eyes and the mouth across all emotion categories, while TBI participants made more overall fixations to the eyes but exhibited a mixed fixation pattern for the nose and mouth. Taken as a whole, the descriptive statistics suggest that the TBI and control groups have different eye scanning patterns when viewing dynamic facial expressions.

Table 29. Descriptive statistics for eye-tracking metrics across the six emotion conditions.

		Anger		Disgust		Fear		Happy		Sad		Surprised	
		<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>	<i>TBI</i>	<i>Control</i>
<b>Time to first Fixation (seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	1.58	1.25	2.35	1.41	1.98	1.35	1.96	1.57	2.23	1.70	1.68	1.32
	<i>SD</i>	1.32	1.19	1.66	1.29	1.19	1.26	1.22	1.35	1.23	1.62	1.39	1.40
	<b>Nose</b>												
	<i>Mean</i>	1.94	1.89	2.04	1.80	1.81	2.22	2.18	2.40	2.03	2.04	1.62	2.13
	<i>SD</i>	1.54	1.15	1.45	0.84	1.31	1.12	1.22	0.77	1.26	0.83	1.16	1.25
	<b>Mouth</b>												
	<i>Mean</i>	3.16	2.46	2.32	1.77	2.75	2.34	2.30	1.83	2.80	2.33	2.64	2.17
	<i>SD</i>	1.67	1.55	1.42	1.17	1.69	1.52	1.39	1.22	1.59	1.67	1.67	1.47
<b>First Fixation Duration (seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	0.12	0.28	0.20	0.21	0.30	0.27	0.24	0.22	0.24	0.22	0.20	0.22
	<i>SD</i>	0.20	0.22	0.16	0.11	0.19	0.20	0.16	0.18	0.20	0.13	0.14	0.16
	<b>Nose</b>												
	<i>Mean</i>	0.36	0.35	0.22	0.36	0.28	0.39	0.28	0.27	0.25	0.37	0.36	0.26
	<i>SD</i>	0.25	0.57	0.10	0.48	0.20	0.75	0.19	0.17	0.13	0.51	0.23	0.37
	<b>Mouth</b>												
	<i>Mean</i>	0.25	0.33	0.28	0.39	0.26	0.28	0.51	0.42	0.30	0.36	0.28	0.29
	<i>SD</i>	0.25	0.24	0.20	0.23	0.19	0.19	0.45	0.28	0.23	0.26	0.22	0.21
<b>Total Fixation Duration (seconds)</b>	<b>Eyes</b>												
	<i>Mean</i>	1.61	2.14	1.73	2.17	1.29	1.90	1.21	1.82	1.13	1.55	1.53	1.74
	<i>SD</i>	1.18	1.59	1.38	1.63	0.95	1.48	1.03	1.66	1.02	1.36	1.21	1.49
	<b>Nose</b>												
	<i>Mean</i>	1.14	0.85	0.99	0.93	0.95	0.77	0.89	0.73	0.89	0.81	0.77	0.96
	<i>SD</i>	0.88	0.88	0.72	0.55	0.66	0.81	0.56	0.52	0.56	0.61	0.55	0.83
	<b>Mouth</b>												
	<i>Mean</i>	0.59	1.10	1.15	1.44	0.72	1.19	1.46	1.72	0.91	1.22	0.95	1.02
	<i>SD</i>	0.66	1.10	0.89	0.98	0.66	1.12	1.05	1.15	0.92	1.08	0.10	0.90
<b>Fixation Count</b>	<b>Eyes</b>												
	<i>Mean</i>	4.00	6.36	2.36	4.60	3.33	6.36	3.59	5.79	2.86	4.85	4.01	6.14
	<i>SD</i>	2.01	4.91	2.11	3.75	2.55	5.29	3.15	5.21	2.56	4.22	2.01	5.11
	<b>Nose</b>												
	<i>Mean</i>	3.00	2.38	2.97	2.56	2.93	2.14	2.73	2.12	2.66	2.32	3.09	2.15
	<i>SD</i>	2.44	1.19	2.08	1.23	1.94	1.03	1.75	1.24	1.72	1.00	1.81	1.38
	<b>Mouth</b>												
	<i>Mean</i>	1.41	2.44	2.41	3.09	1.87	2.81	2.82	3.59	1.85	2.57	1.89	2.93
	<i>SD</i>	1.35	1.90	1.57	1.84	1.64	2.23	1.79	2.42	1.45	2.02	1.62	2.19

#### 8.4.4.2.1. Time to first fixation

There were no significant main effects of emotion, ( $F(5, 170) = 1.98, p = .084, \eta_p^2 = 0.05$ ), AOI, ( $F(1.41, 47.87) = 3.11, p = .070, \eta_p^2 = 0.08$ ) and group, ( $F(1, 34) = 1.51, p = .227, \eta_p^2 = 0.04$ ). There were also no significant interactions between emotion and group, ( $F(5, 170) = 1.50, p = .193, \eta_p^2 = 0.04$ ), AOI and group, ( $F(1.41, 47.87) = 0.91, p = .377, \eta_p^2 = 0.03$ ) or between AOI, emotion and group, ( $F(7.98, 271.43) = 8.10, p = .594, \eta_p^2 = 0.02$ ). There was a significant interaction between emotion and AOI, ( $F(7.98, 271.43) = 6.50, p \leq .001, \eta_p^2 = 0.160$ ) which suggests that the time to first fixation for the different AOI were different across the six emotions. To further explore this, simple effects tests were conducted and the results indicated that participants took significantly longer to instigate a first fixation to the mouth compared to the eyes ( $p = .001$ ) and to the mouth compared to the nose ( $p = .002$ ) when viewing angry faces.

#### 8.4.4.2.2. First fixation duration

There were no significant main effects for emotion, ( $F(2.19, 74.28) = 0.93, p = .404, \eta_p^2 = 0.02$ ), AOI ( $F(1.73, 58.85) = 1.97, p = .154, \eta_p^2 = 0.06$ ) or group, ( $F(1, 34) = 0.19, p = .664, \eta_p^2 = 0.006$ ). There were no significant interactions between emotion and group, ( $F(2.19, 74.28) = 1.24, p = .298, \eta_p^2 = 0.04$ ), AOI and group, ( $F(1.73, 58.85) = 0.23, p = .768, \eta_p^2 = 0.007$ ), or AOI and group or emotion, ( $F(2.80, 95.29) = 0.84, p = .471, \eta_p^2 = 0.02$ ). The interaction between emotion and AOI was not significant, ( $F(2.80, 95.29) = 2.55, p = .064, \eta_p^2 = 0.07$ ).

#### 8.4.4.2.3. Total fixation duration

There was a significant main effect of emotion, ( $F(2.19, 74.47) = 11.69, p \leq .001, \eta_p^2 = 0.26$ ) and AOI, ( $F(2.37, 80.62) = 5.21, p = .005, \eta_p^2 = 0.13$ ) but no significant effect of group, ( $F(1, 34) = 2.93, p = .096, \eta_p^2 = 0.08$ ) and also all interactions were not significant ( $p \geq .464$ ) bar emotion and AOI, ( $F(3.70, 125.93) = 4.89, p \leq .001, \eta_p^2 = 0.126$ ). To further investigate the interaction between AOI and emotion, paired-samples comparisons through simple effects analysis were examined and the significant results are presented in Table 30. The findings suggested that participants fixated for longer on the eyes compared to the nose and mouth across the majority of emotions.

Table 30. Significant post-hoc tests exploring the interaction between AOI and emotion (total fixation duration).

Emotion	Mean difference (SE)	Sig.
Anger		
Eyes-Nose	0.88 (0.32)	.009
Eyes-Mouth	1.32 (0.34)	.004
Disgust		
Eyes-Nose	0.99 (0.27)	.001
Fear		
Eyes-Nose	0.73 (0.27)	.010
Happy		
Mouth-Nose	0.78 (0.18)	.001

Note: The AOI listed first in the table received the longest fixation duration.

#### 8.4.4.2.4. Fixation duration for internal facial areas vs. external areas

Mirroring the static condition, internal facial areas included the eyes, nose and mouth while external areas include all other areas of the face (i.e. the chin, forehead and cheeks) and the rest

of the visual scene (i.e. the shoulders and chest of the model and the background area of the photo).

There was a significant effect of AOI, ( $F(1, 34) = 38.89, p \leq .001, \eta_p^2 = 0.53$ ) and emotion, ( $F(2.19, 74.47) = 11.69, p \leq .001, \eta_p^2 = 0.26$ ) as well a significant interaction between AOI and emotion, ( $F(2.11, 71.67) = 5.87, p = .004, \eta_p^2 = 0.15$ ). There was no significant interaction between emotion and group, ( $F(2.19, 74.47) = 0.83, p = .451, \eta_p^2 = 0.02$ ), AOI and group, ( $F(1, 34) = 1.69, p = .202, \eta_p^2 = 0.05$ ) or emotion, AOI and group, ( $F(2.11, 71.67) = 0.16, p = .867, \eta_p^2 = 0.005$ ). There was also no significant main effect of group, ( $F(1, 34) = 2.93, p = .096, \eta_p^2 = 0.08$ ).

The significant interaction between AOI and emotion was analysed with paired-sample contrasts through simple effects analysis. Participants spent significantly longer fixating on internal facial areas across all emotions compared to external areas of the visual scene ( $p \leq .001$ ).

#### **8.4.4.2.5. Fixation count**

There was a significant main effect of emotion, ( $F(5, 170) = 9.14, p \leq .001, \eta_p^2 = 0.212$ ) and AOI, ( $F(2.03, 69.17) = 8.24, p \leq .001, \eta_p^2 = 0.20$ ) but no significant tests of between-subjects effects for group, ( $F(1.13, 34.71) = 1.13, p = .295, \eta_p^2 = 0.03$ ). Only the emotion and AOI interaction was significant, ( $F(7.81, 265.44) = 8.16, p \leq .001, \eta_p^2 = 0.19$ ), and the emotion and group, ( $F(5, 170) = 1.45, p = .21, \eta_p^2 = 0.04$ ), AOI and group, ( $F(2.03, 69.17) = 2.42, p = .10, \eta_p^2 = 0.07$ ), and emotion, AOI and group, ( $F(7.81, 265.44) = 1.85, p = .99, \eta_p^2 = 0.03$ ) were not significant.

To further investigate the interaction between AOI and emotion, parried-samples comparisons through simple effects analysis were examined and the significant results are presented in Table

31. Participants made more fixations to the eyes compared to the nose and mouth when looking at angry, fearful, happy and surprised faces.

Table 31. Significant post-hoc tests exploring the interaction between AOI and emotion (fixation count).

Emotion	Mean difference (SE)	Sig.
Anger		
Eyes-Nose	2.49 (0.78)	.003
Eyes-Mouth	3.26 (0.83)	.001
Fear		
Eyes-Nose	2.31 (0.76)	.005
Eyes-Mouth	2.51 (0.85)	.006
Happy		
Eyes-Nose	2.26 (0.73)	.004
Surprise		
Eyes-Nose	2.45 (0.78)	.003
Eyes-Mouth	2.67 (0.86)	.004

Note: The AOI listed first in the Table received the most fixation counts.

#### 8.4.4.2.6. Fixation count for internal facial areas vs. external areas

There was a significant effect of AOI, ( $F(1, 34) = 22.25, p \leq .0001, \eta_p^2 = 0.40$ ) and emotion, ( $F(5, 170) = 9.14, p \leq .0001, \eta_p^2 = 0.21$ ) as well a significant interaction between AOI and emotion, ( $F(5, 170) = 5.30, p \leq .001, \eta_p^2 = 0.14$ ). The interactions between AOI and group, ( $F(1, 1) = 3.37, p = .075, \eta_p^2 = 0.09$ ), emotion and group, ( $F(5, 170) = 1.45, p = .210, \eta_p^2 = 0.04$ ) and AOI, emotion and group, ( $F(5, 170) = 0.69, p = .633, \eta_p^2 = 0.02$ ) were not significant. The main effect of group was also not significant, ( $F(1, 34) = 1.13, p = .295, \eta_p^2 = 0.03$ ). Significant pairwise comparisons investigating the interaction between emotion and AOI indicated that angry faces generated more fixations to internal compared to external areas while fearful faces evoked more fixations to external compared to internal areas (Table 32).

Table 32. Significant post-hoc tests exploring the interaction between AOI and emotion (fixation count).

<b>Emotion</b>	<b>Mean difference (SE)</b>	<b>Sig.</b>
Fear		
External-Internal	-1.54 (0.25)	.001

Note: The AOI listed first in the table received the longest fixation duration.

#### **8.4.4.3. Correlations**

When the groups were combined, there were no significant correlations between the ADFES dynamic emotion labelling accuracy scores and the four eye-tracking metrics (all  $r$ 's  $\leq 0.003$ ,  $p \geq .493$ ). When the groups were separated, there were no significant correlations for the group with TBI (all  $r$ 's  $\leq 0.26$ ,  $p \geq .304$ ) or controls (all  $r$ 's  $\leq 0.47$ ,  $p \geq .047$ ).

### **8.5. Discussion**

Overall, the individuals with TBI were less accurate at identifying dynamic facial expressions compared to the control group. This data corroborates with existing research documenting facial affect recognition impairments post-TBI (Knox & Douglas, 2009; McDonald et al., 2003) and the findings from the static task. Although dynamic facial expressions provide additional cues through facial motion, which is documented to enhance emotion recognition (Alves, 2013; McCamy et al., 2014), the current group affected by TBI still displayed deficits when trying to decipher dynamic facial expressions. These findings supplement the limited extant evidence base exploring dynamic facial affect recognition post-TBI. Furthermore, the results have higher generalisability compared to static stimuli which are often criticised for poor ecological validity (Knox & Douglas, 2009).

Interestingly, McDonald and Saunders (2005) reported that only one individual from a group of 34 severe TBI participants displayed impairments in recognising dynamic facial expressions.

This finding could be equated with the task which McDonald and Saunders (2005) used as they employed the Emotion Evaluation Test (EET) from The Assessment of Social Inference Test (McDonald et al., 2003). Although the authors omitted the sound, contextual cues through body language could have been perceived, potentially influencing performance levels. It could be argued that the TASIT offers a more realistic account of social interactions compared to the ADFES, which presents viewers with a posed headshot of an actor displaying an apex emotion, making McDonald and Saunders (2005) results more ecologically valid. Nevertheless, Knox and Douglas (2009) also used the EET (with sound omitted) in their research, reporting that individuals with TBI scored significantly lower on the EET compared to the control group. It is unclear why these differences occurred, one explanation could be that TBI is heterogeneous and each research cohort will present with a variety of injury aetiology and pathology. Another explanation could be that McDonald and Saunders (2005) recruited a higher percentage of females. It is frequently documented that women exhibit superior emotion recognition skills compared to males (Olderbak et al., 2019; Wingenbach et al., 2018) so cognitive reserve, that is the ability for the brain to use pre-existing cognitive processing approaches to aid compensatory approaches (Stern, 2012), could act as a moderator between pathology and clinical impairments. Cognitive reserve could potentially account for the discrepancies between findings. The aforementioned points underscore some of the challenges of mapping brain and behaviour changes post-TBI and highlight the importance of controlling for variables such as sex and pathology location.

Although there were significant differences between the two groups for emotion labelling accuracy, the TBI and control group exhibited similar eye scan patterns throughout the ADFES dynamic task. There were also no significant correlations between the accuracy data and the eye-tracking data suggesting that the lower emotion recognition scores exhibited by the group affected by TBI were not related to aberrant fixation patterns. These findings conflict with the



literature reporting abnormalities in visual strategies in response to faces post-TBI (Adolphs et al., 2005; Douglas et al., 2010; Kenrick et al., 2017; Wolf et al., 2014), although the majority of this research is based on static stimuli. As everyday social interactions are dynamic, dynamic research stimuli are likely to elicit more naturalistic eye scan patterns compared to static stimuli. Furthermore, the use of these dynamic tools has the potential to advance the understanding of typical and non-typical visual strategies underpinning emotion perception.

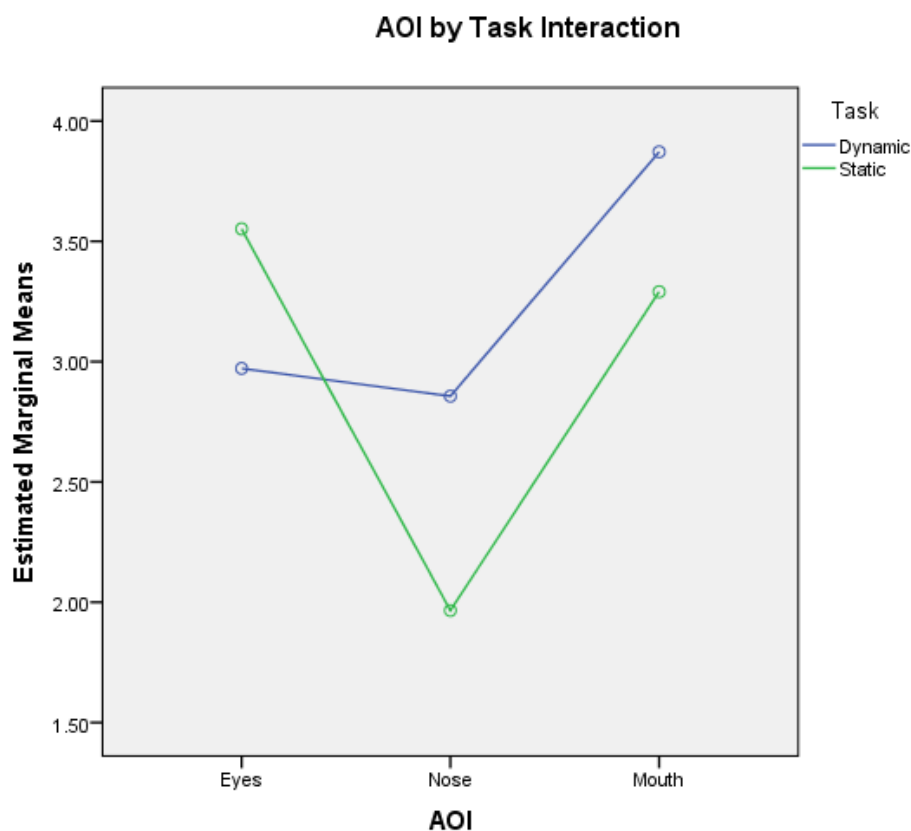
In sum, the group living with TBI exhibited lower emotion recognition accuracy scores when identifying dynamic facial expressions compared to a matched control group. Both the TBI and control groups displayed similar eye scanning patterns when viewing faces. Lower behavioural scores were not correlated with eye-tracking data, suggesting that non-typical eye scan patterns did not account for group discrepancies in emotion recognition accuracy.

## **8.6. Comparison of static versus dynamic stimuli**

As the static task presented faces for 8 seconds and the dynamic task presented them for 5.5 seconds, a straightforward comparison was not possible. To account for this variation in time, the data was made equivalent across the two tasks by dividing the dynamic task first fixation duration, total fixation duration and fixation count by 0.6875 (this is the ratio of 5.5/8 seconds for the task duration). This adjustment was not made to the time to first fixation analysis because these were already equivalent metrics. Several 2(task)\*(6-emotion)\*(3-AOI)\*(2-group) ANOVA were conducted and it was decided that only interactions involving both group and task would be reported. The only significant interaction involving group and task was the time to first fixation analysis ( $F(1.5, 47.3) = 4.25, p = .030, \eta_p^2 = 0.12$ ). This was the task by AOI by group interaction. To explore this further, separate ANOVAs for each group were conducted and revealed that there was only a significant effect for the group with TBI ( $F(1.5, 23.5) = 4.99, p = .024, \eta_p^2 = 2.38$ ) (please see graph 6). To explore this task by AOI interaction

for the group with TBI, the main effects of AOI for each task were explored. This showed that only the static task produced a significant main effect of AOI ( $F(2,15) = 8.52, p = .003, \eta_p^2 = 0.53$ ). Breaking this main effect down further with pairwise comparisons, with the post-hoc adjustment applied, showed that the time to first fixation for the nose was significantly slower than the mouth during the static task ( $p = .005$ ).

Graph 6. Graph to illustrate the interaction between AOI and task for ADFES comparison.



### 8.7. Overall discussion

The group with TBI displayed impairments in recognising both static and dynamic facial expressions compared to a group of matched controls, supporting the view that facial expression recognition is disrupted post-TBI. One hypothesis of the current research was that aberrant fixation patterns contributed to poor facial expression identification scores. While the group affected by TBI exhibited aberrant eye fixation patterns compared to controls when viewing static faces, there were no significant differences in visual strategies between the TBI

and control groups when viewing dynamic faces. These findings appear to demonstrate that dynamic and static stimuli evoke disparate visual strategies and there may be a dissociation between static and dynamic processing systems, an approach which is advocated by others (Kilts et al., 2003; McDonald & Saunders, 2005). fMRI and electroencephalogram evidence suggests that static and dynamic facial expressions elicit different brain activation patterns (Foley, Rippon, Thai, Longe & Senior, 2011; Garrido-Vásquez, Pell, Paulmann & Kotz, 2018; Schultz & Pilz, 2009; Trautmann-Lengsfeld et al., 2013; Zinchenko et al., 2018) so it is not surprising that they also produce different gaze patterns.

Blais et al., (2017) explored visual strategies underpinning static and dynamic facial expression recognition in a normative sample using eye-tracking and the bubbles technique. The authors reported that the same facial features (eyes, nose and mouth) were utilised during both conditions when identifying emotions but, participants exhibited more fixations to the left eye and mouth during static conditions and more fixations to the centre of the face during dynamic conditions. The authors proposed that one possible explanation for this difference is that humans can process biological motion outside of the fovea and peripheral to the fixation location (Gurnsey et al., 2008; Thompson et al., 2007). Therefore, more fixations to the centre of the face during dynamic conditions may allow an individual to process motion cues from the eyes and mouth without directly fixating on the features. As discussed in chapter three, visual resolution and sensitivity are decreased outside of the fovea. Direct fixations on the eyes during dynamic conditions would result in the nose, and particularly the mouth, to be in the periphery of vision, where high-acuity vision is attenuated. Fixating on the centre of the face allows for a diffuse visual scanning strategy with less visual information falling in the periphery of the visual field. However, the present findings indicated that the control participants displayed more and longer fixations on the eyes during the static and dynamic conditions while the group with TBI fixated more on the nose only when viewing static faces. Therefore, the

results of the present research corroborate with Blais et al., (2017) static findings, that non-clinical groups exhibit more fixations on the eyes and mouth, but contradict their dynamic findings, that the nose received the most fixations.

A second explanation to account for the lack of disparity between the gaze patterns of the two groups on the dynamic condition is the role of biological motion. The natural movement of facial muscles during emotion expression causes certain facial features to appear more salient, for instance, the mouth turning upwards during a smile or the enlarged sclera's (white of eyes) during fear, therefore drawing attention to them (Horstmann & Ansorge, 2009; Tobin, Favelle, & Palermo, 2016). The present findings illustrated that the individuals affected by TBI fixation pattern differed between static and dynamic stimuli, with a shift to attending to the eyes more in the dynamic condition. It could be suggested that the movement associated with dynamic expressions implicitly captured the attention and eliminated the differences in gaze patterns observed during the static condition. Indeed, Calvo et al., (2018) highlighted that research with static faces had produced non-conclusive findings and that motion benefits facial affect recognition. In their research, Calvo et al., (2018) reported that different dynamic facial expressions elicited different ocular fixation patterns. The researchers proposed that this represented a selective attention pattern to expression-specific diagnostic face regions, possibly related to contrasting facial muscle patterns associated with different emotions. The premise that dynamic and static facial expressions could potentially evoke different eye scan patterns raises questions regarding the validity of existing research which have based their findings on static stimuli. It also highlights the fact that individuals with TBI are observing these motion cues during the dynamic task, and possibly during everyday social interactions, yet are still failing to correctly identify facial expressions. It appears that facial affect recognition impairments post-TBI do not fully stem from aberrant gaze patterns, suggesting that there is a

disruption to later social cognitive processing, either during the social knowledge retrieval or response selection stages Penn et al., 2007).

A limitation of the present study is that it did not collect reaction time data for the dynamic task. Evidence suggests a distinction between speeded and non-speeded face viewing which appears to affect fixation patterns (Hsiao & Cottrell, 2008). It would be interesting to compare speeded and non-speeded static and dynamic tasks to explore if the time element of the design had an effect of fixation patterns. Another limitation of the static condition is that the stimuli were presented for a relatively long time (eight seconds) allowing for a high number of saccades and fixations. As research reports that face identification can be achieved in as few as two fixations (Hsiao & Cottrell, 2008), the visual data after initial fixations may not be directly relevant to the task. Alternating stimulus display times, particularly introducing very short or fleeting facial expressions, could yield interesting results. Although the present research is innovative, being the first to explore eye scan patterns of TBI and control groups when viewing dynamic and static facial expressions, one limitation, similar to the Blais et al., (2017) study, was the use of simulated rather than natural facial expressions. There are qualitative differences between simulated and spontaneous emotions (Cohn & Schmidt, 2011; Ross & Pulusu, 2013), and future research could take this into account. Furthermore, laboratory experiments often eliminate the contextual effects of social interactions. Although the exploration of the relationship between context effects and social cognition is still in its infancy, emerging research surmises a positive interaction, with participants scoring higher on context, included conditions compared to context removed conditions (Barrett et al., 2011; Ibañez & Manes, 2012). To address this concern, the next two chapters will outline findings from the Movie for the Assessment of Social Cognition (Dziobek et al., 2006) and The Awareness of Social Inference Test (McDonald et al., 2003), two dynamic social cognition assessments which introduce context into the experimental design.

When a direct comparison was made between the static and dynamic tasks, there was minimal difference between them, gaze patterns for both the group with TBI and controls were similar across both tasks. The group with TBI did exhibit a significantly slower first fixation to the nose compared to the mouth during the static task. These findings do not fully support the hypothesis that visual strategies underpinning the recognition of static and dynamic facial expressions are different (Blais et al., 2017). In fact, the findings point more towards a similarity in gaze patterns between static and dynamic conditions. This may have implications for scholars who have reprimanded the use of static stimuli (e.g. Knox & Douglas, 2009) although, common sense would prevail that the use of dynamic stimuli does increase ecological validity. Moreover, this study was exploratory in nature and further research is needed to ensure that this was not a result of a type I error.

In conclusion, the group with TBI performed significantly lower compared to controls when tasked with correctly identifying six basic static and dynamic emotions. Furthermore, the TBI group were significantly slower at identifying static expressions. The current findings indicated that the group affected by TBI exhibited abnormal visual strategies when viewing static facial expressions compared to the control group. There were no group differences for the dynamic facial expressions which suggested that the group with TBI displayed similar eye scanning patterns to the control group. There were also no significant correlations between facial expression recognition scores and eye-tracking metrics for either the static or dynamic conditions. This result implies that impairments in emotion perception post-TBI may not stem from low-level visual or spatial frequency disruptions but may be related to higher-level cognitive processes. The contrasting visual strategies elicited by the group with TBI for static and dynamic facial expressions demonstrate modality-specific processing differences. It could be postulated that the visual processing of dynamic facial expressions remains intact as the biological motion associated with the unfolding of emotion draws the perceiver's attention to

the salient facial features (Horstmann & Ansorge, 2009; Tobin et al., 2016). Static expressions could require a degree of explicit processing, as the stimuli do not have the natural cues of biological motion. Referring back to Penn et al., (1997) social cognition model, it seems plausible to suggest that the group affected by TBI displayed impairments during the very initial stages of social cognition, the perception of social cues, during the static condition, and thus the remaining two stages (retrieval of social knowledge and response selection) were negatively affected. However, during the dynamic task, the group affected by TBI perception of social cues was similar to controls, indicating that there were no low-level visual disruptions; yet, they still scored lower on the emotion identification part of the task compared to controls. Consequently, poor performance on the recognition of dynamic facial expressions may result from the disruption of higher-level upstream cognitive processes compared to low-level visual deficits. The amalgamation of static and dynamic stimuli has subsequently exposed disparities, warranting further investigation. It has also highlighted the processing differences between the two modes and underscored the necessity for research to utilise more ecologically valid social measures.

## 9. Chapter nine: The Assessment of Social Inference Test (TASIT)

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### 9.1. Objectives and hypothesis

The present chapter will outline and discuss the findings from The Assessment of Social Inference Test (TASIT; McDonald et al., 2003). All three sub-components of the TASIT were administered to thoroughly explore social cognition; the Emotion Evaluation Test (EET), the Social Inference-Minimal (SI-M) test, and the Social Inference-Enriched (SI-E) test. Through the EET, the understanding of communication components such as facial expressions, tone of voice, and gestures were investigated. The SI-M provided a measure of the participants' ability to read paralinguistic features, facial expressions and tone of voice in either sincere or sarcastic situations. Finally, the SI-E assessed the participants' ability to use contextual cues (i.e. tone of voice and facial expressions) to determine if everyday conversations were deceptive or sarcastic.

Given the literature discussed previously, differences in behavioural and eye-tracking responses on the various sub-tasks of the TASIT were expected between the TBI and control group. Three hypotheses were constructed:

**Hypothesis 1.** The group affected by TBI would have significantly lower accuracy scores across the TASIT compared to the control group.

**Hypothesis 2.** TBI Participants would exhibit different fixation patterns compared to the control group.

**Hypothesis 3.** The seven different emotions of the EET would generate different fixation patterns.



**Hypothesis 4.** There will be positive interactions between fixation duration and counts to the AOI and accuracy scores across the TASIT.

## **9.2. Participants**

Due to a malfunction with the eye-tracking equipment, one of the control participant's data was corrupted for the SI-M and SI-E tests. Therefore, the sample size for these two sub-tasks consisted of 18 TBI participants and 17 age and sex-matched controls. See Table 33 for revised demographic data for the SI-M and SI-E tests. Please note that the EET data is based on the full cohort described in the methodology (chapter five).

## **9.3. Procedure**

The TASIT, as described in detail in chapter five, was administered on the Tobii T120 eye tracker. Participants were seated approximately 60-65cm away from the eye tracker screen. Participants watched each sub-task independently with breaks in between. The EET consists of 28 short videos, the SI-M has 15 videos and the SI-E has 16 videos, all lasting between 15-60 seconds. Each sub-task took approximately 20 minutes to administer and would begin with written instructions on the screen. Participants watched the videos and answered subsequent questions about them. The EET has a multiple-choice answer format where participants were required to choose from seven emotions. The SI-M and SI-E asked four questions about what the actor was doing, saying, thinking and feeling. The researcher moved the experiment on by pressing the space bar after the participant had confirmed they had read and understood the instructions on the screen. Please see Appendix 9, 11 and 12 for the design and protocol of the EET, SI-M and SI-E.

Table 33. Participant demographics for the SI-M and SI-E.

<b>Descriptive statistic</b>	<b>Gender</b>	<b>Current Age</b>	<b>Age at injury (years)</b>	<b>Post-injury years</b>	<b>Years of education</b>	<b>Verbal IQ</b>	<b>Performance IQ</b>	<b>Overall IQ</b>
Number								
TBI	m= 15, f= 3	-	-	-	-	-	-	-
Control	m= 14, f=3							
Mean								
TBI	-	44.94	36.44	8.50	14.83	84.06	91.00	90.25
Control		44.71	-	-	15.53	96.06	105.41	100.88
SD								
TBI	-	11.69	13.79	8.68	4.25	18.71	17.50	17.66
Control		12.05	-	-	3.76	8.35	11.61	10.14
Range								
TBI	-	26-62	17-59	1-28	10-23	57-135	55-111	55-120
Control		25-63	-	-	11-23	82-113	79-121	81-116
Mann-Whitney U test		$p = .858$			$p = .443$	$p=.005^{**}$	$p = .009^{**}$	$p = .014^{**}$

\* Two TBI participants opted out of the WASI (IQ test)

\*\* Significant at  $\alpha = .05$

#### **9.4. Exploration of data**

The behavioural and eye-tracking metrics for all sub-tasks were explored using histograms, box-plots, and absolute  $z$ -scores for skewness and kurtosis. The behavioural data was relatively normally distributed with mild violations. The eye-tracking data had moderate violations of normality regarding skewness and kurtosis, homogeneity of variance, and outliers. However, as violations were moderate and not severe, and because the study had approximately equal sample sizes, the effects of violation would have been mitigated (Finch, 2005). Therefore, parametric analyses were used to analyse the TASIT data with post-hoc corrections applied to all follow-up analyses ( $\alpha = .01$ ).

#### **9.5. EET**

For the EET, a  $2*(7)$  ANOVA was conducted to explore potential differences in emotion perception. A further  $2*(7)*(3)$  repeated measures ANOVA was conducted to investigate potential differences in fixation duration and count between the TBI and control groups.

##### **9.5.1. Behavioural data**

As demonstrated in Table 34, the descriptive statistics indicated that the group with TBI had lower overall accuracy scores on the EET compared to the control group.

Table 34. Descriptive statistics for the number of correct responses for the TBI and control groups for the EET.

<b>TASIT EET Score</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Overall Correct	19.67 (3.99)	24.28 (1.60)
Happy	3.11 (1.13)	3.28 (0.67)
Surprised	3.22 (0.81)	3.67 (0.49)
Neutral	2.00 (0.91)	2.72 (0.83)
Sad	2.72 (1.32)	3.50 (0.71)
Angry	3.00 (1.08)	3.56 (0.51)
Anxious	2.89 (1.32)	3.94 (0.24)
Revolted	2.72 (1.02)	3.56 (0.62)

The analysis showed a significant main effect of emotion, ( $F(6, 204) = 6.85, p \leq .001, \eta_p^2 = 0.17$ ), but the interaction between emotion and group was non-significant, ( $F(6, 204) = 1.08, p = .375, \eta_p^2 = 0.03$ ). The test of between-subjects effects was also significant, ( $F(1, 34) = 20.28, p \leq .001, \eta_p^2 = 0.37$ ) with the group means indicating that the TBI participants had significantly fewer correct responses across the EET compared to the control group. When comparing the descriptive statistics, it appeared that the TBI group scored lower on negative emotions compared to positive, particularly interpreting sad, anxious and revolted displays of emotion, compared to the control group.

## **9.5.2. Eye-tracking data**

### **9.5.2.1. Fixation duration across the EET in seconds**

The group with TBI had shorter fixation durations to the eyes and nose compared to controls, while both groups displayed similar fixation durations to the mouth (Table 35).

Table 35. Descriptive statistics for the TBI and control group for fixation duration across the EET in seconds.

<b>EET Emotions</b>	<b>Groups combined Mean (SD)</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Angry	3.97 (3.01)	2.92 (2.12)	5.03 (3.43)
Eyes	1.15 (1.25)	0.92 (1.08)	1.37 (1.40)
Nose	1.59 (1.80)	0.93 (1.16)	2.26 (2.10)
Mouth	1.23 (0.90)	1.07 (0.90)	1.40 (0.90)
Revolted	3.76 (3.06)	3.01 (1.99)	4.50 (3.76)
Eyes	0.78 (0.97)	0.72 (0.97)	0.85 (0.99)
Nose	1.43 (2.05)	0.82 (1.04)	2.05 (2.60)
Mouth	1.54 (1.56)	1.48 (1.35)	1.61 (1.78)
Anxious	2.44 (2.74)	1.53 (2.10)	3.35 (3.05)
Eyes	0.41 (0.61)	0.51 (0.74)	0.32 (0.44)
Nose	1.43 (1.99)	0.60 (1.06)	2.27 (2.36)
Mouth	0.60 (0.87)	0.42 (0.76)	0.77 (0.96)
Happy	4.41 (2.35)	4.04 (2.32)	4.79 (2.38)
Eyes	0.93 (1.13)	0.62 (0.96)	1.24 (1.23)
Nose	0.86 (1.02)	0.69 (0.68)	1.02 (1.28)
Mouth	2.62 (1.70)	2.73 (1.74)	2.53 (1.71)
Sad	8.56 (6.68)	6.92 (5.48)	10.21 (7.50)
Eyes	3.84 (3.53)	3.40 (3.91)	4.28 (3.16)
Nose	2.16 (3.16)	1.05 (1.13)	2.37 (3.99)
Mouth	2.56 (2.60)	2.47 (2.59)	2.65 (2.67)
Surprised	2.18 (1.95)	1.64 (1.77)	2.72 (2.01)
Eyes	0.63 (1.05)	0.70 (1.28)	0.55 (0.79)
Nose	0.94 (1.22)	0.51 (0.66)	1.38 (1.49)
Mouth	0.61 (0.68)	0.43 (0.46)	0.79 (0.82)
Neutral	4.84 (2.92)	4.36 (2.76)	5.32 (3.08)
Eyes	1.92 (2.22)	1.85 (2.36)	1.98 (2.14)
Nose	1.59 (1.49)	1.13 (1.23)	2.06 (1.61)
Mouth	1.33 (1.23)	1.38 (1.51)	1.27 (0.90)
Overall (emotions combined)	3.82 (2.82)	2.93 (2.32)	4.69 (3.05)
Eyes	0.98 (1.21)	0.87 (1.29)	1.57 (1.58)
Nose	1.37 (1.65)	0.72 (0.89)	1.57 (1.56)
Mouth	1.47 (1.16)	1.35 (1.16)	2.68 (1.88)

The analysis showed a significant main effect of emotion, ( $F(1.83, 62.17) = 38.99, p \leq .001, \eta_p^2 = 0.53$ ), and a significant interaction between emotion and AOI, ( $F(4.19, 142.35) = 12.29, p \leq .001, \eta_p^2 = 0.27$ ). The main effect of AOI, ( $F(1.70, 57.87) = 0.09, p = .884, \eta_p^2 = 0.003$ ), and the interactions between emotion and group, ( $F(1.83, 62.17) = 1.67, p = .198, \eta_p^2 = 0.05$ ), AOI and group, ( $F(1.70, 57.87) = 2.17, p = .130, \eta_p^2 = 0.06$ ), and emotion, AOI and group, ( $F(4.19, 142.35) = 1.26, p = .289, \eta_p^2 = 0.04$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 34) = 2.84, p = .101, \eta_p^2 = 0.08$ ).

The significant interaction between emotion and AOI were explored through three one-way ANOVAs, with emotion as the independent variable (IV) and fixation duration as the dependent variable (DV). There was a significant effect of eyes, ( $F(1.40, 48.90) = 36.35, p \leq .001, \eta_p^2 = 0.51$ ), nose, ( $F(2.53, 88.41) = 5.99, p = .002, \eta_p^2 = 0.15$ ) and mouth, ( $F(1.40, 48.90) = 36.35, p \leq .001, \eta_p^2 = 0.51$ ) on fixation duration in response to different facial expressions, with follow-up paired-samples  $t$  tests in Appendix 15. In brief, the post-hoc tests indicated that participants made longer fixations to the eyes when viewing sad faces compared to all other emotions. Participants also spent significantly longer looking at the eyes of neutral faces compared to the other emotions (bar sad). The group elicited longer fixations to the nose when viewing angry, sad and neutral faces and more fixations to the mouth when looking at happy and sad faces.

### **9.5.2.2. Fixation count across the EET**

The group affected by TBI had fewer fixations compared to controls across all of the emotions and most of the AOIs within the emotions (Table 36).

Table 36. Descriptive statistics for the TBI and control group for fixation count across the EET.

<b>EET Emotions</b>	<b>Groups combined Mean (SD)</b>	<b>TBI Mean (SD)</b>	<b>Control Mean (SD)</b>
Angry	8.80 (5.52)	6.54 (3.75)	11.06 (6.15)
Eyes	2.67 (2.76)	1.93 (2.14)	3.42 (3.16)
Nose	2.98 (2.74)	1.69 (1.70)	4.26 (3.01)
Mouth	3.14 (1.98)	2.92 (1.99)	3.38 (1.99)
Revolted	7.12 (4.93)	6.36 (4.07)	7.89 (5.68)
Eyes	1.68 (1.66)	1.66 (1.68)	1.70 (1.69)
Nose	2.75 (3.00)	2.02 (2.35)	3.48 (3.45)
Mouth	2.69 (2.13)	2.68 (2.02)	2.71 (2.29)
Anxious	4.21 (4.25)	3.00 (3.51)	5.41 (4.68)
Eyes	1.02 (1.24)	1.12 (1.39)	0.91 (1.11)
Nose	2.02 (2.52)	0.92 (1.13)	3.11 (3.05)
Mouth	1.17 (1.65)	0.96 (1.65)	1.39 (1.65)
Happy	9.22 (4.19)	8.79 (3.99)	9.64 (4.45)
Eyes	2.17 (2.12)	1.42 (1.38)	2.92 (2.49)
Nose	2.25 (1.77)	2.07 (1.65)	2.43 (1.92)
Mouth	4.79 (2.60)	5.30 (2.88)	4.29 (2.26)
Sad	12.81 (8.24)	11.15 (7.00)	14.47 (9.21)
Eyes	5.22 (3.77)	4.96 (4.08)	5.49 (3.53)
Nose	3.44 (3.98)	2.21 (2.51)	4.67 (4.81)
Mouth	4.15 (3.26)	3.99 (3.09)	4.32 (3.49)
Surprised	4.29 (3.28)	3.37 (2.89)	5.21 (3.47)
Eyes	1.08 (1.16)	1.02 (1.19)	1.13 (1.16)
Nose	1.87 (2.15)	1.19 (1.32)	2.54 (2.60)
Mouth	1.34 (1.29)	1.16 (1.15)	1.53 (1.42)
Neutral	9.56 (5.42)	7.81 (4.34)	11.31 (5.93)
Eyes	3.75 (3.72)	2.98 (3.08)	4.52 (4.21)
Nose	3.03 (2.46)	2.12 (2.10)	3.94 (2.50)
Mouth	2.78 (2.03)	2.71 (2.29)	2.84 (1.78)
Overall	7.38 (4.61)	5.82 (3.72)	8.94 (4.97)
Eyes	2.02 (1.84)	1.57 (1.58)	2.48 (2.01)
Nose	2.54 (2.44)	1.57 (1.56)	3.51 (2.80)
Mouth	2.82 (1.80)	2.68 (1.88)	2.96 (1.76)

The analysis showed a significant main effect of emotion, ( $F(3.40, 115.65) = 41.78, p \leq .001, \eta_p^2 = 0.55$ ) and a significant interaction between emotion and AOI, ( $F(5.85, 198.88) = 10.77, p \leq .001, \eta_p^2 = 0.24$ ). The main effect of AOI, ( $F(1.60, 54.41) = 0.44, p = .602, \eta_p^2 = 0.01$ ) and the interactions between emotion and group, ( $F(3.40, 115.65) = 1.79, p = .146, \eta_p^2 = 0.05$ ), AOI and group, ( $F(1.60, 54.41) = 2.26, p = .124, \eta_p^2 = 0.06$ ), and emotion, AOI and group, ( $F(5.85, 198.88) = 1.88, p = .088, \eta_p^2 = 0.05$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 34) = 2.96, p = .094, \eta_p^2 = 0.08$ ).

The significant interaction between emotion and AOI were explored through three one-way ANOVAs, with emotion as the IV and fixation count as the DV. There was a significant effect of eyes, ( $F(1.40, 48.90) = 36.35, p \leq .001, \eta_p^2 = 0.44$ ), nose, ( $F(4.17, 146.02) = 5.87, p \leq .001, \eta_p^2 = 0.14$ ) and mouth, ( $F(3.94, 138.06) = 30.97, p \leq .001, \eta_p^2 = 0.47$ ) on fixation count in response to different facial expressions, with follow-up paired-samples  $t$  tests presented in Appendix 16. In summary, the group made more fixations to the eyes and nose when viewing neutral and sad faces. The mouth was looked at more when participants viewed happy and sad faces.

### **9.5.3. Correlations**

One-tailed Spearman's Rho correlations were run to explore relationships between the behavioural data (accuracy for emotion recognition) and eye-tracking data (fixation duration and fixation count). When the groups were combined and the  $\alpha$ -level had been adjusted to account for multiple comparisons ( $\alpha = .01$ ) there were no significant correlations (all,  $r$ 's  $\leq 0.29$ , all  $p$ 's  $\geq .043$ ). When the groups were separated there were also no significant correlations for the group with TBI (all  $r$ 's  $\leq -0.23, p \geq .352$ ) or controls (all  $r$ 's  $\leq 0.58, p \geq .013$ ).



## 9.6. SI-M

A 2\*(3) ANOVA was conducted to investigate group differences during the understanding of conversational meanings. A further 2\*(4) ANOVA was conducted to investigate the difference between the groups in understanding different facets of social interactions. With eye-tracking data, a 2\*(3)\*(3) repeated measures ANOVA was conducted to investigate potential differences in fixation duration and count between the TBI and control groups. 8.6.1

Behavioural data

### 9.6.1. Accuracy for conversation style

As demonstrated in Table 37, the descriptive statistics appeared to indicate that the TBI group had lower overall accuracy scores across the conversational styles (simple-sarcasm, paradoxical-sarcasm and sincere) compared to the control group.

Table 37. Descriptive statistics for the accuracy of the TBI and control groups for the different conversational constructs during the SI-M.

SI-M Score	TBI Mean (SD)	Control Mean (SD)
Simple-sarcasm	15.28 (3.63)	17.71 (2.11)
Paradoxical-sarcasm	15.94 (4.01)	18.18 (2.24)
Sincere	15.39 (3.31)	19.76 (1.48)

There was no significant effect of conversation style, ( $F(1.59, 52.47) = 1.37, p = .260, \eta_p^2 = 0.04$ ) and no interaction between conversation style and group, ( $F(1.59, 52.47) = 1.63, p = .203, \eta_p^2 = 0.05$ ). The tests of between-subjects effects was significant, ( $F(1, 33) = 21.03, p \leq .001, \eta_p^2 = 0.39$ ) and referring to the estimated marginal means, the group with TBI scored significantly lower across the three conversation styles of the SI-M ( $M = 15.54, SD = 3.65$ ) compared to the control group ( $M = 18.55, SD = 1.94$ ).

### 9.6.2. Accuracy for comprehension probes

The descriptive statistics for the four different comprehension probes (beliefs, meanings, intentions, feelings) suggested that the group affected by TBI were less accurate at understanding what the actor was trying to do, what they were trying to say, what they were thinking, and what they were feeling (Table 38).

Table 38. Descriptive statistics for the accuracy of the TBI and control groups for the four comprehension probes during the SI-M.

SI-M Score	TBI Mean (SD)	Control Mean (SD)
Intentions	11.39 (2.73)	13.82 (1.07)
Meaning	11.78 (2.21)	13.82 (0.95)
Beliefs	11.11 (2.47)	13.71 (1.05)
Feelings	12.89 (1.75)	14.00 (0.94)

There was a significant effect of comprehension probe, ( $F(2.38, 78.36) = 3.19, p = .038, \eta_p^2 = 0.09$ ) but there was no interaction between comprehension probe and group, ( $F(2.38, 78.36) = 1.37, p = .261, \eta_p^2 = 0.04$ ). To further explore the significant effect of comprehension probe, paired-samples  $t$  tests were conducted. When the post-hoc correction was applied, none of the follow-up tests were significant. The tests of between-subjects effects was significant, ( $F(1, 33) = 17.67, p \leq .001, \eta_p^2 = 0.35$ ) and referring to the estimated marginal means, the group with TBI scored significantly lower across the four comprehension probes of the SI-M ( $M = 11.67, SD = 2.29$ ) compared to the control group ( $M = 13.84, SD = 1.00$ ).

### 9.6.3. Eye-tracking data

#### 9.6.3.1. Fixation duration across the SI-M in seconds

The descriptive statistics seemed to indicate that participants tended to spend more time fixated on the mouth than the nose and eyes during all three conversation styles (Table 39). The sincere

videos appeared to elicit the longest fixations, followed by the sarcastic and then paradoxical videos.

Table 39. Fixation duration for SI-M across the conversation styles.

<b>Conversational Style</b>	<b>Overall mean (SD)</b>	<b>TBI mean (SD)</b>	<b>Control mean (SD)</b>
Simple-sarcasm	1.70 (1.54)	1.77 (1.47)	1.63 (1.65)
Eyes	0.34 (0.54)	0.44 (0.65)	0.23 (0.37)
Nose	0.55 (0.67)	0.51 (0.56)	0.58 (0.78)
Mouth	0.81 (0.79)	0.82 (0.75)	0.81 (0.86)
Paradoxical-sarcasm	0.74 (0.66)	0.80 (0.62)	0.68 (0.72)
Eyes	0.21 (0.26)	0.26 (0.30)	0.15 (0.19)
Nose	0.29 (0.39)	0.24 (0.30)	0.35 (0.48)
Mouth	0.24 (0.28)	0.30 (0.33)	0.18 (0.20)
Sincere	2.79 (1.82)	3.02 (2.24)	2.53 (1.26)
Eyes	0.59 (0.82)	0.68 (1.00)	0.49 (0.59)
Nose	0.95 (0.92)	1.01 (0.98)	0.89 (0.88)
Mouth	1.25 (1.23)	1.34 (1.51)	1.15 (0.88)

The analysis showed a significant main effect of conversational style, ( $F(1.34, 44.16) = 23.59$ ,  $p \leq .001$ ,  $\eta_p^2 = 0.42$ ) and AOI, ( $F(2, 66) = 6.29$ ,  $p = .003$ ,  $\eta_p^2 = 0.16$ ). The interactions between conversation style and group, ( $F(1.34, 44.16) = 0.25$ ,  $p = .78$ ,  $\eta_p^2 = 0.01$ ), AOI and group, ( $F(1.78, 58.82) = 0.39$ ,  $p = 0.657$ ,  $\eta_p^2 = 0.01$ ), conversation style and AOI, ( $F(2.39, 78.98) = 2.62$ ,  $p = .070$ ,  $\eta_p^2 = 0.07$ ), and conversation style, AOI and group, ( $F(2.39, 78.98) = 0.15$ ,  $p = .894$ ,  $\eta_p^2 = 0.01$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 33) = 0.52$ ,  $p = .476$ ,  $\eta_p^2 = 0.02$ ).

To explore the significant main effect of conversation style, two-tailed paired samples  $t$  tests were conducted and the post-hoc correction was applied. Participants generated significantly longer fixation durations during the sarcastic videos compared to paradoxical videos, ( $t(34) = 5.67$ ,  $p \leq .001$ ,  $d = 0.96$ ,  $CI = 0.61-1.30$ ), sincere videos compared to sarcastic videos, ( $t(34) =$

-3.00,  $p = .005$ ,  $d = 0.51$ ,  $CI = -1.82 - -0.35$ ), and sincere videos compared to paradoxical videos, ( $t(34) = -6.50$ ,  $p \leq .001$ ,  $d = 1.09$ ,  $CI = -2.68 - -1.41$ ).

Additional two-tailed paired-samples  $t$  tests were conducted to investigate the significant main effect of AOI. When the post-hoc correction was applied, participants exhibited significantly longer fixations to the mouth ( $M = 2.30$ ,  $SD = 1.69$ ) compared to the eyes ( $M = 1.13$ ,  $SD = 1.07$ ), ( $t(34) = -3.24$ ,  $p = .003$ ,  $d = 0.55$ ,  $CI = -1.90 - -0.44$ ).

### 9.6.3.2. Fixation count across the SI-M

From the descriptive statistics presented in Table 40, there appeared to be a similar pattern for both the TBI and control participants, with both groups having higher fixation counts for the sarcastic and sincere compared to paradoxical videos.

Table 40. Descriptive statistics for the AOI for the TBI and control groups across the three conversational styles (fixation count).

Conversational Style	Overall mean (SD)	TBI mean (SD)	Control mean (SD)
Sarcasm	4.52 (3.21)	4.62 (2.74)	4.41 (3.72)
Eyes	0.95 (1.27)	1.22 (1.49)	0.67 (0.96)
Nose	1.55 (1.46)	1.48 (1.76)	1.61 (1.75)
Mouth	2.02 (1.58)	1.92 (1.43)	2.13 (1.76)
Paradoxical	1.97 (1.48)	2.12 (1.44)	1.82 (1.56)
Eyes	0.62 (0.63)	0.76 (0.71)	0.46 (0.52)
Nose	0.75 (0.78)	0.73 (0.69)	0.76 (0.89)
Mouth	0.61 (0.50)	0.63 (0.56)	0.59 (0.46)
Sincere	5.73 (3.41)	5.44 (3.71)	6.04 (3.16)
Eyes	1.67 (2.06)	1.55 (1.62)	1.79 (2.49)
Nose	1.94 (1.56)	1.91 (1.67)	1.98 (1.49)
Mouth	2.12 (1.69)	1.98 (1.90)	2.27 (1.48)

The analysis showed a significant main effect of conversational style, ( $F(1.39, 45.91) = 22.40, p \leq .001, \eta_p^2 = 0.40$ ). The other main effect of AOI, ( $F(1.69, 55.77) = 2.96, p = .068, \eta_p^2 = 0.08$ ), and the interactions between conversation style and group, ( $F(1.39, 45.91) = 0.37, p = .617, \eta_p^2 = 0.01$ ), AOI and group, ( $F(1.69, 55.77) = 0.38, p = .648, \eta_p^2 = 0.01$ ), conversation style and AOI, ( $F(2.26, 74.71) = 2.40, p = .091, \eta_p^2 = 0.07$ ), and conversation style, AOI and group, ( $F(2.26, 74.71) = 0.44, p = .671, \eta_p^2 = 0.01$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 33) = 0.002, p = .965, \eta_p^2 \leq 0.001$ ).

To explore the significant main effect of conversation style, two-tailed paired-samples  $t$  tests were conducted with post-hoc corrections applied. Participants generated significantly more fixations during the sarcastic videos compared to paradoxical videos, ( $t(34) = 7.24, p \leq .001, d = 1.22, CI = 1.83-3.26$ ), and to sincere ( $M = 5.73, SD = 3.41$ ) videos compared to paradoxical videos, ( $t(34) = -6.30, p \leq .001, d = 1.06, CI = -4.97- -2.54$ ).

#### **9.6.4. Correlations**

One-tailed Spearman's Rho correlations explored the relationships between the behavioural data (accuracy for conversation style and comprehension probe) and eye-tracking data (fixation duration and fixation count). When the groups were combined and once the  $\alpha$ -level had been adjusted to account for multiple comparisons ( $\alpha = .01$ ), there were no significant correlations (all,  $r$ 's  $\leq 0.001$ , all  $p$ 's  $\geq .110$ ). When the groups were separated, there were still no correlations for the group with TBI (all  $r$ 's  $\leq 0.51, p \geq .032$ ) but there were significant correlations for the control group between fixation duration to the eyes and intentions ( $r = 0.74, p = .001$ ), fixation count to the eyes and intentions ( $r = 0.73, p = .001$ ) and between fixation duration to the eyes and simple sarcasm ( $r = 0.69, p = .002$ ).

## 9.7. SI-E

A 2\*(2) ANOVA was conducted to investigate group differences during the understanding of conversational meanings. A further 2\*(4) ANOVA was conducted. Again, this analysis investigated the difference between the groups in understanding different facets of social interactions. A 2\*(2)\*(3) repeated measures ANOVA was conducted to investigate potential differences in fixation duration and count between the TBI and control groups.

### 9.7.1. Behavioural data

#### 9.7.1.1. Accuracy for conversational style

As demonstrated in Table 41, the descriptive statistics appeared to indicate that the group affected by TBI had lower overall accuracy scores across the two conversational styles (sarcastic and lie) compared to the control group.

Table 41. Descriptive statistics for the behavioural data of the TBI and control groups during the sarcastic and lie conditions of the SI-E.

SI-E Score	TBI Mean (SD)	Control Mean (SD)
Sarcastic	22.56 (4.77)	29.41 (3.37)
Lie	25.33 (4.38)	27.76 (3.11)

The effect of conversation style was non-significant, ( $F(1.00, 33.00) = 0.64, p = .428, \eta_p^2 = 0.02$ ) but there was a significant interaction between conversation style and group, ( $F(1.00, 33.00) = 9.86, p = .004, \eta_p^2 = 0.23$ ). Simple effects analysis revealed that the group with TBI scored significantly lower on the sarcastic vignettes ( $M = 22.56, SD = 4.77$ ) compared to controls ( $M = 29.41, SD = 3.37$ ) ( $p \leq .001$ ). The tests of between-subjects effects was also significant, ( $F(1, 33) = 16.31, p \leq .001, \eta_p^2 = 0.33$ ). Referring to the estimated marginal means, the group affected by TBI scored significantly lower across the two conversation styles of the SI-E ( $M = 23.94, SD = 4.78$ ) compared to the control group ( $M = 28.58, SD = 3.24$ ).

Although the  $t$  tests were non-significant once the post-hoc correction was applied, the follow-up tests indicated that the control group had higher recognition scores for the sarcastic videos compared to the videos depicting lies ( $t(16) = -2.83, p = .012, d = 0.69, CI = [-2.88, -0.41]$ ) while the group with TBI exhibited the reverse pattern, ( $t(17) = 2.22, p = .040, d = 0.52, CI = [0.13, 5.42]$ ).

### 9.7.1.2. Accuracy for comprehension probes

The descriptive statistics for the four different comprehension probes (intentions, meanings, beliefs, and feelings) suggested that the group living with TBI were less accurate at understanding what the actor was trying to do, what they were trying to say, what they were thinking, and what they were feeling (Table 42).

Table 42. Descriptive statistics for the behavioural data of the TBI and control groups for the four comprehension probes during the SI-E.

SI-E Score	TBI Mean (SD)	Control Mean (SD)
Intentions	11.28 (2.49)	14.82 (1.63)
Meaning	11.17 (2.60)	14.47 (1.70)
Beliefs	13.33 (1.81)	14.47 (0.72)
Feelings	12.11 (2.47)	14.12 (2.45)

There was a significant effect of comprehension probe, ( $F(2.46, 81.19) = 3.25, p = .034, \eta_p^2 = 0.09$ ) and a significant interaction between comprehension probe and group, ( $F(2.46, 81.19) = 4.69, p = .008, \eta_p^2 = 0.12$ ). Simple effects analysis revealed that the group with TBI scored significantly lower on the ‘intention’ probes ( $M = 11.28, SD = 2.49$ ) compared to controls ( $M = 14.82, SD = 1.63$ ) ( $p \leq .001$ ). The same effect was found for the ‘meaning’ probes, with the group with TBI scoring significantly lower ( $M = 11.17, SD = 2.60$ ) compared to the control group ( $M = 14.47, SD = 1.70$ ) ( $p \leq .001$ ). The tests of between-subjects effects was also

significant, ( $F(1, 33) = 21.41, p \leq .001, \eta_p^2 = 0.39$ ). Referring to the estimated marginal means, the group affected by TBI scored significantly lower across the four comprehension probes of the SI-E ( $M = 11.97, SD = 2.34$ ) compared to the control group ( $M = 14.47, SD = 1.63$ ).

## 9.7.2. Eye-tracking data

### 9.7.2.1. Fixation duration across the SI-E in seconds

Table 43 appears to suggest that individuals with TBI had shorter fixation durations to the eyes, nose and mouth compared to controls (Table 43).

Table 43. Descriptive statistics for fixation duration to the AOI for the TBI and control groups across the two conversational styles of the SI-E.

Conversational Style	Overall mean (SD)	TBI mean (SD)	Control mean (SD)
Sarcasm	0.98 (0.92)	0.85 (0.70)	1.11 (1.10)
Eyes	0.37 (0.45)	0.31 (0.37)	0.43 (0.53)
Nose	0.35 (0.39)	0.29 (0.30)	0.41 (0.47)
Mouth	0.26 (0.25)	0.25 (0.19)	0.28 (0.30)
Lie	0.38 (0.57)	0.25 (0.30)	0.51 (0.75)
Eyes	0.14 (0.24)	0.12 (0.19)	0.17 (0.30)
Nose	0.11 (0.20)	0.07 (0.11)	0.14 (0.25)
Mouth	0.13 (0.22)	0.06 (0.08)	0.20 (0.29)

The analysis showed a significant main effect of conversational style, ( $F(1, 33) = 44.18, p \leq .001, \eta_p^2 = 0.57$ ) with participants exhibiting longer fixations for sarcastic videos compared to lie videos. The main effect of AOI, ( $F(1.22, 40.16) = 0.92, p = .405, \eta_p^2 = 0.03$ ) and the interactions between conversation style and group, ( $F(1.00, 33.00) = 0.002, p = .969, \eta_p^2 \leq 0.01$ ), AOI and group, ( $F(1.22, 40.16) = 0.01, p = .991, \eta_p^2 \leq 0.01$ ), conversation style and AOI, ( $F(1.61, 53.26) = 2.54, p = .099, \eta_p^2 = 0.07$ ), and conversation style, AOI and group, ( $F(1.61, 53.26) = 1.42, p = .249, \eta_p^2 = 0.04$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 33) = 1.13, p = .296, \eta_p^2 = 0.03$ ).



### 9.7.2.2. Fixation count across the SI-E

From the descriptive statistics presented in Table 44, the TBI cohort appeared to produce fewer fixation counts to all AOI across the sarcastic and lie conditions compared to control participants. Both the TBI and control participants generated fewer fixation counts during the videos depicting lies compared to videos where the actors were being sarcastic. Inferential statistics were conducted to determine if these apparent differences were significant.

Table 44. Descriptive statistics for fixation counts to the AOI for the TBI and control groups across the two conversational styles of the SI-E.

Conversational Style	Overall mean (SD)	TBI mean (SD)	Control mean (SD)
Sarcasm	3.90 (2.74)	3.62 (2.04)	4.20 (3.37)
Eyes	1.33 (1.25)	1.21 (1.10)	1.45 (1.42)
Nose	1.39 (1.22)	1.24 (0.83)	1.55 (1.55)
Mouth	1.18 (0.82)	1.16 (0.72)	1.20 (0.95)
Lie	1.48 (1.94)	1.12 (0.98)	1.86 (2.58)
Eyes	0.47 (0.63)	0.44 (0.53)	0.49 (0.74)
Nose	0.49 (0.74)	0.35 (0.39)	0.65 (0.98)
Mouth	0.52 (0.77)	0.33 (0.31)	0.72 (1.03)

The analysis showed a significant main effect of conversational style, ( $F(1, 33) = 75.77, p \leq .001, \eta_p^2 = 0.70$ ) with participants displaying more fixations during the sarcastic vignettes compared to vignettes depicting lies. The main effect of AOI, ( $F(1.36, 44.95) = 0.28, p = .673, \eta_p^2 = 0.01$ ) and the interactions between conversation style and group, ( $F(1, 33) = 0.08, p = .780, \eta_p^2 = 0.002$ ), AOI and group, ( $F(1.36, 44.95) = 0.20, p = .732, \eta_p^2 = 0.01$ ), conversation style and AOI, ( $F(1.58, 52.23) = 1.29, p = .279, \eta_p^2 = 0.04$ ), and conversation style, AOI and group, ( $F(1.58, 52.23) = 1.49, p = .236, \eta_p^2 = 0.04$ ) were all non-significant. The tests of between-subjects effects was also not significant, ( $F(1, 33) = 0.76, p = .390, \eta_p^2 = 0.02$ ).

### **9.7.3. Correlations**

One-tailed Spearman's Rho correlations explored the relationships between the behavioural data (accuracy for conversation style and comprehension probe) and eye-tracking data (fixation duration and fixation count). When the groups were combined and once the  $\alpha$ -level had been adjusted to account for multiple comparisons ( $\alpha = .01$ ), there were no significant correlations ( $r \leq 0.39, p \geq .022$ ). When the groups were separated, there were still no significant correlations for the group with TBI (all,  $r$ 's  $\leq 0.56$ , all  $p$ 's  $\geq .016$ ) or controls (all,  $r$ 's  $\leq 0.56$ , all  $p$ 's  $\geq .019$ ).

### **9.8. Discussion**

The EET was administered to measure the participants understanding of social communication components such as facial expressions, tone of voice and gestures. The analysis indicated that the group with TBI were significantly less accurate identifying emotions compared to the control group. This is in line with similar research reporting that individuals with moderate-severe TBI displayed poor emotion identification across all emotions on the TASIT (McDonald et al., 2003; Rosenberg et al., 2019). These findings support the suggestion that TBI disrupts emotion perception. However, there were no differences between the two groups on the eye-tracking analysis, suggesting that TBI and control participants exhibited similar fixation patterns across the EET. There were also no significant correlations between eye-tracking metrics and accuracy scores for the group with TBI. This result indicated that poor emotion perception post-TBI is not related to aberrant gaze patterns. There were significant correlations for the control group which indicated that more and longer fixations to the eyes were related to increased understanding of intentions and simple sarcasm. Understanding other people's intentions and sarcasm are complex social processes and focusing more on the eyes would allow the individual to gather as many diagnostic cues as possible.

Recently, investigators have examined if certain regions of the face may be more diagnostic when identifying specific emotions. One objective from the current study was to explore

whether participants produced different fixation patterns to AOI when judging different emotions. The significant interaction between AOI and emotion for fixation duration and fixation count during the EET indicated that distinct fixation patterns emerged for the seven basic emotions. Similar to Schurgin et al., (2014), Pérez-Moreno et al., (2016) and Calvo et al., (2018), the current findings point to a goal-driven influence on eye gaze patterns, as distinct patterns of fixations were observed for both emotional and neutral faces. Moreover, visual strategies appeared to reflect attention to the most diagnostic area of the face for each emotion (e.g. mouth for identifying happiness and nose for recognising disgusted faces) suggesting that gaze patterns may also be stimulus-driven (Calvo et al., 2018; Eisenbarth & Alpers, 2011; Schurgin et al., 2014).

The results from the EET indicated that individuals with TBI fail to capture and evaluate verbal and non-verbal social cues in comparison to control participants. In line with Eisenbarth and Alpers (2011) and Schurgin et al., (2014), the current findings illustrated that ocular fixation patterns may follow both stimulus-driven and goal-driven perceptual strategies when identifying facial affect. However, group did not have an effect on fixation patterns and there were no significant differences between the TBI and control participants in terms of fixation duration or count to the predetermined AOI. This signified that the TBI participant's lower scores on the EET were not associated with deficits in low-level social perception but must be related to impairments in the higher-level stages of social processing.

The SI-M measured the participant's ability to read paralinguistic features, facial expressions and tone of voice in either sincere, simple-sarcastic and paradoxical-sarcastic exchanges. The group with TBI cored significantly lower across all three of the conversation styles of the SI-M, in comparison to the control group, with simple-sarcasm vignettes being the hardest for the group affected by TBI to decipher, followed by sincere, and then paradoxical-sarcastic

exchanges. There was also a significant difference between the TBI and control groups on the comprehension probes of the SI-M, with the group with TBI exhibiting difficulty in understanding the actor's intentions, feelings and the meaning of the conversation compared to the control group. The current findings are similar to McDonald et al., (2003) who reported that their TBI group were inferior to controls when asked to identify simple and paradoxical-sarcastic exchanges. Unexpectedly, McDonald et al., (2003) reported that individuals with TBI performed better than controls on the sincere items, suggesting that this finding reflected a failure in the group affected by TBI to be distracted by innuendo. Nevertheless, this effect was not replicated in a follow-up study by McDonald et al., (2004) when they compared SI-M scores between a group of severe TBI participants and matched controls. Furthermore, the present research found that the group with TBI exhibited lower accuracy scores when recognising sincere exchanges compared to controls. It could be the case that McDonald et al.'s (2003) findings were related to a type I error.

With regards to the SI-M eye-tracking data, all participants generated significantly longer fixation durations to the simple-sarcastic and sincere exchanges compared to paradoxical-sarcastic exchanges. Additionally, both groups elicited more and longer fixations during the simple-sarcastic videos compared to paradoxical-sarcastic videos, and to the sincere videos compared to paradoxical-sarcastic videos. These findings appear to suggest that the simple-sarcastic exchanges were the most challenging to decipher, as evidenced through increased attentional resources measured through ocular fixations. This suggestion is corroborated with the behavioural data which indicated that both groups scored lowest on the simple-sarcasm conditions compared to the other two conditions. It is logical to assume that simple-sarcastic exchanges would be more difficult to understand compared to paradoxical exchanges where you have the additional aid of nonsensical verbal utterances (Appendix 10). Similar to the findings of the MASC, it appears that different domains of socio-cognitive processing (i.e.

different social communication styles: sincere, simple-sarcasm, and paradoxical-sarcasm) elicited different visual strategies. This more than likely reflects a contrast in the magnitude of processing necessary for deciphering the social interaction.

The eye-tracking literature frequently reports that participants typically fixate for longer on the eyes compared to other facial features (Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells et al., 2016). However, during the SI-M, participants tended to fixate for longer on the mouth compared to the eyes. This finding may be related to the dynamic nature of the study and the frequent movements of the mouth during speech, highlighting the essentiality of including more ecologically valid measures during social cognition research.

There was no difference between the TBI and the control groups visual strategies when viewing the social exchanges of the SI-M. This suggests that both groups elicited a similar number of fixations with comparable durations to the eyes, nose and mouths of the actors. Correlational analyses revealed that there were no significant relationships between fixation duration or fixation count and accuracy scores for identifying the conversation style (sarcastic or sincere) or comprehension probes (what the actor was intending to do or say and what they were thinking and feeling). Accordingly, it appears that individuals with TBI difficulty in accurately deciphering sincere and sarcastic social exchanges may not be attributed to aberrant eye scan patterns.

The SI-E assessed the participant's ability to use contextual cues (i.e. tone of voice and facial expressions) to determine if everyday conversations were deceptive (the actor was lying) or sarcastic. Again, the group with TBI were significantly poorer at understanding the actor's intentions, feelings and beliefs and the overall meaning of the social exchange compared to the control group. The group affected by TBI were significantly poorer at recognising sarcastic conversations compared to the control group which is in line with previous research (Channon,

Pellijeff & Rule, 2005; McDonald & Pearce, 1996). Interestingly, there was no significant difference between the two groups during the lie vignettes. These findings are consistent with McDonald et al., (2003) who also found that their TBI participants displayed impairments when trying to decipher sarcastic exchanges, but scored in a typical range during the lie conditions when compared with matched controls. These results imply that TBI diminishes the ability to comprehend sarcasm but the aptitude to recognise lies remains intact, a model that McDonald (2000) previously outlined in a review paper of neuropsychological studies of sarcasm. There are conceptual differences between sarcasm and lying; when an individual is being sarcastic they want their conspecifics to identify that they are speaking insincerely, while someone who is telling a lie aims to hide their insincerity. It has been argued that understanding sarcasm requires more cognitive effort compared to understanding sincere and untruthful exchanges (Deliens, Antoniou, Clin & Kissine, 2017; Miron-Spektor, Efrat-Treister, Rafaeli & Schwarz-Cohen, 2011), mainly because understanding sarcasm necessitates first-order theory of mind (ToM; understanding what someone is thinking or feeling) and second-order ToM (predicting what one person thinks or feels about what another person is thinking or feeling). Further research exploring the intricacies of sarcasm and deception comprehension post-TBI is warranted as there is insufficient evidence at present. This research area is important as sarcasm permeates modern society and individuals who fail to understand this social construct are usually viewed as socially inept (Haiman, 1998).

All participants exhibited more and longer fixations during the sarcastic vignettes compared to vignettes depicting lies. This suggests that participants directed more attentional resources to the sarcastic videos compared to the lie videos. Similar to the EET and the SI-M, group did not have an effect on fixation patterns and there were no significant differences between the TBI and control participants in terms of fixation duration or count to the predetermined AOI. These findings appear to indicate that individuals with TBI have an intact ‘visual’ social perception,

as their ocular fixation patterns were similar to the matched control group. Nevertheless, the individuals in the TBI group still scored significantly lower compared to the control group when identifying mood states and deducing whether a conversational exchange was sincere or sarcastic. This suggests that post-TBI deficits may lie in the second or third stage of social cognitive processing, which is the retrieval of social knowledge or response selection.

In conclusion, the group affected by TBI found it harder to recognise emotions compared to a group of matched control participants. The present research also found distinct fixation patterns in response to seven different emotions and between sincere, sarcastic and deceptive social exchanges. Overall, the group living with TBI displayed lower accuracy scores when identifying simple-sarcastic, paradoxical-sarcastic and sincere exchanges, although there was a more pronounced impairment for the deciphering of simple-sarcastic conversations. Interestingly, the group affected by TBI displayed no deficits when interpreting lies when compared with the control group. Sarcastic remarks often transpire during every day social interactions and impairments in understanding these exchanges are reported in several clinical populations including autistic (Deliens, Papastamou, Ruytenbeek, Geelhand & Kissine, 2018) and schizophrenic cohorts (Kantrowitz, Hoptman, Leitman, Silipo & Javitt, 2014). During human relations, literal and non-literal meanings of speech unfold simultaneously with numerous communication devices such as facial expressions and body language. Unfortunately, the majority of existing research has employed context-void and uni-modal stimuli, focusing solely on visual or vocal components of social processing. The present study builds upon the current evidence base exploring indirect forms of speech post-TBI. Eye-tracking data during the interpretation of these different conversational exchanges revealed that differences in foveal fixation patterns did not appear to explain the low accuracy scores exhibited by the individuals in the group with TBI for the identification of mood state and conversational meaning. The previous sections have attempted to explore if there were differences between a TBI and

matched-control group when viewing static and dynamic social stimuli. The chapter which follows moves on to consider whether some of the atypical visual strategies, reported in the previous studies of this thesis, extend to common day objects and animals.



## **10. Chapter ten: Overall discussion**

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### **10.1. Chapter overview**

This chapter will summarise the main results of the research programme. Findings will be discussed in relation to previous studies and theories. Following this, implications of the research, including social cognition assessment and TBI rehabilitation, will be discussed. The limitations of the current research and potential future research directions will be also be outlined.

### **10.2. Research review**

The overarching aim of this thesis was to explore the visual strategies underpinning social cognition, including emotion perception, ToM and social concepts such as lies and sarcasm. This was achieved by using static and dynamic social stimuli with individuals with TBI and a matched control cohort. TBI is a major public health concern (Roozenbeek et al., 2013; Humphreys et al., 2013), and is documented as a primary cause of chronic psychological impairments (Johnson & Griswold, 2017; Li et al., 2016). Reports from individuals affected by TBI and their families frequently indicate that social and emotional impairments after injury have the most adverse effect on the quality of life (Fleminger & Ponsford, 2005; Humphreys et al., 2013; McDonald et al., 2002; Ubukata et al., 2014). TBI survivors usually experience a breakdown in familial and social relationships (Bodley-Scott & Riley, 2015; Carlozzi et al., 2015), which subsequently leads to social isolation and the increased risk of developing mental health conditions (Bjork & Grant, 2009; Bornhofen & McDonald, 2008; Fralick et al., 2016).

The mechanisms underpinning these social and emotional changes post-TBI are not fully understood; despite frequent documentation that TBI disrupts social cognition, particularly facial affect recognition (Babbage et al., 2011; McDonald, 2013; McDonald et al., 2018; Spell

& Frank, 2000). It is unknown whether facial affect recognition impairments post-TBI are due to a disruption of low-level functions, such as visual processing, or more complex high-level cognitive functions. Research exploring low-level visual processes post-TBI is sparse, and the relationship between visual processes and social functioning is not sufficiently explored. If low-level visual processes are disrupted by TBI then this could impair the ability to capture, monitor and evaluate social stimuli, consequently having an adverse effect on upstream higher-order cognitive functions, such as emotion perception and ToM.

The current research utilised a range of static and dynamic social cognition assessments, in combination with eye-tracking technology, to examine the recognition of the six basic emotions (anger, disgust, fear, happiness, sadness and surprise) as well as other bi-modal social cues such as tone of voice, body language and gestures. Together, the four studies present an empirical exploration of social cognition in TBI and control cohorts and the overall findings are summarised below.

### **10.3. Summary of overall findings**

The clinical MASC data indicated that the group affected by TBI had lower accuracy scores compared to the control group, supporting the evidence base documenting that social cognition is disrupted post-TBI (Babbage et al., 2011; Ubukata et al., 2014). The individuals in the TBI group also scored significantly higher on the undermentalizing condition compared to the control group. Undermentalizing refers to reduced ToM, meaning that a person is capable of mentalizing, but does it incorrectly (Vaskinn et al., 2015). In the context of brain injury, this is an insightful finding as it suggests that individuals with TBI may still be able to execute ToM if they receive psychosocial rehabilitation. Indeed, ToM training post-TBI, although in its infancy, has proved beneficial (Lundgren & Brownell, 2015; Zettin & Galetto, 2016).

The group with TBI also displayed differences in gaze patterns compared to the control group, eliciting significantly fewer and shorter fixations to the three AOI. Typically, during natural vision, eye movements are only directed to salient information and it is seldom that random fixations are made (Hayhoe, Shrivastave & Nrucek, 2003; Peterson & Eckstein, 2012). In the case of the MASC, the most salient areas would be the faces of the characters, namely the eyes but also the nose and mouth, as these features express mood state (Eisenbarth & Alpers, 2011; Guo & Shaw, 2015; Laidlaw & Kingstone, 2017; Wells, Gillespie & Rotshtein, 2016). Arguably, if the group affected by TBI were eliciting fewer and shorter fixations to facial features then they were going to find it more challenging to decipher the social situation. Research has proposed a relationship between fixation patterns and social cognition (Funahashi, 2014; Wolf, Philippi, Motzkin, Baskaya & Koenigs, 2014). Indeed, aberrant visual strategies have been associated with low social functioning and high social disability in schizophrenic and autistic individuals (Bo, Li, Jiang, Wang & Wang, 2018; Klin et al., 2002), as well as impairments in deciphering mental states (Grynszpan & Nadel, 2015; Itier & Batty, 2009). However, the current research failed to find a relationship between fixation patterns and accuracy scores on the MASC. This finding indicated that abnormal social perception (i.e. fixation patterns) does not account for impairments in mindreading abilities post-TBI.

An interesting finding from the MASC study was that both the TBI and control groups generated disparate fixation patterns for the three AOI in response to the subcategories of the MASC. Although it is generally well-established that ocular fixation patterns appear to differ in response to the six basic emotions (Barabanschikov, 2015; Calvo et al., 2018; Pérez-Moreno et al., 2016; Schurgin et al., 2014), there does not appear to be any published data exploring visual strategies in response to different domains of social cognition (i.e. deciphering thoughts, emotions and intentions). The hypothesis that different domains of social cognition produce disparate attentional orienting and time course of fixations on diagnostic regions of the social

scene was also supported by the non-clinical study. This finding suggested that different domains of social cognition activate different attentional resources, as evidenced through contrasting gaze patterns. This finding highlights the possibility of studying the initial stages of social processes in isolation to gain a deeper understanding of social cognition overall. This might prove fruitful for future research wanting to index social cognition in clinical and non-clinical groups.

The data from the MASC normative study indicated that there were no significant differences between males and females on the MASC in terms of ToM judgements and fixation patterns. This is an important finding as there is currently discourse in the literature regarding a female advantage on social cognition tasks (Olderbak et al., 2018). The findings are in line with previous research documenting non-significant differences between male and female gaze patterns (Hall et al., 2010), although, there is evidence contesting these findings (Vassallo et al., 2009). It appears that sex differences on social tasks and fixation patterns are small, variable and surrounded by controversy regarding specificity (Wright, Riedel, Sechrest, Lane & Smith, 2018). Thus, the current findings expand the knowledge base surrounding this controversial research field, as well as presenting results which indicated that there are no significant differences between male and female processing abilities for social stimuli.

Fixation duration scores for the control group in the clinical MASC study were half the value of those for the two groups (males and females) in the normative study. One explanation for this anomaly could relate to age as the MASC normative participants mean age ( $M = 21.25$ ) was half compared to the MASC clinical control group ( $M = 43.83$ ). It is documented that there are age-related changes in visual exploratory behaviour towards faces. Indeed, Chasby, Hupont, Avril, Luherne-du Boullay and Chetouani (2017) reported that older adults exhibited longer fixation durations over the lower-face area compared to younger adults. Alternatively, age-

related socio-emotional differences may account for discrepancies. Hamel et al., (2013) found that age-related video game-experienced participants displayed larger saccade amplitudes and a broader distribution of fixations across the screen. The younger adults reacted faster to peripheral objects compared to older adults, with the authors concluding that the younger participants displayed superiority in general detection compared to the older adults. Although these findings highlighted the importance of accounting for age in social cognition and eye-tracking research, the area is generally underexplored. Moreover, existing research typically has wide-ranging groups (e.g. young adults in their 20's and older adults 70+) making it difficult to distinguish when these age-related changes might appear. The implications for age-related differences could be significant, especially in clinical research where age could potentially be a confounding variable.

In line with the MASC data, the ADFES static task also suggested that the group with TBI visual strategies differed from the control group concerning the number and duration of fixations, again, supporting preceding research documenting aberrant fixation patterns in TBI samples (Douglas et al., 2010; Kenrick et al., 2017; Oatley et al., 2014; Vassallo et al., 2011; Wolf et al., 2014). Individuals with TBI took significantly longer to instigate the first fixation to the eyes compared to the control group, as well as exhibiting more and longer fixations to the nose. In contrast, there were no differences in gaze patterns between the TBI and control group during the dynamic ADFES task. This is an interesting finding and is in line with research reporting that static and dynamic stimuli elicit different visual strategies. Blais et al., (2017) found that during the viewing of both static and dynamic facial expressions, the same facial features (the eyes, nose and mouth) were fixated on the most but there was a significant difference in the underlying visual strategy patterns. Participants exhibited more fixations to the left eye and mouth during the static condition and more fixations to the centre of the face during the dynamic condition. Blais et al., (2017) suggested that one possible explanation for

this difference is that humans can process biological motion outside of the fovea (Gurnsey et al., 2008; Thompson et al., 2007). Consequently, more fixations to the centre of the face during dynamic conditions may allow an individual to process motion cues from the eyes and mouth without directly fixating on the features, whereas this visual strategy would be insufficient for static stimuli. It could be the case that the biological motion during the dynamic task acted as a natural attention-cue for TBI participants (Horstmann & Ansorge, 2009; Tobin et al., 2016), thus, eliminating the gaze pattern differences observed in the static task. Nevertheless, the biological motion did not appear to guide the group with TBI during the MASC and, therefore, this theory is not fully supported and more research would be needed to disentangle the inconsistencies. One explanation for this variation in results could be that the MASC is film-like, with numerous fast-paced scene changes and multiple characters, whereas the ADFES dynamic task depicts one actor in isolation and, consequently, is less attention-demanding. It could be argued that the MASC has more ecological validity compared to the ADFES dynamic, thus, findings from the MASC may be more representative of real-world visual strategies.

The group affected by TBI were significantly poorer at correctly identifying static and dynamic facial expressions compared to the control group, again, corroborating previous research documenting facial expression recognition impairments post-TBI (Babbage et al., 2011; McDonald et al., 2003). Similar to the MASC findings, the correlational analyses for the ADFES static and dynamic data were non-significant, suggesting that the group affected by TBI's poor emotion labelling accuracy and slower response times on the static task were not related with aberrant fixation patterns. The ADFES studies inform the evidence base that dynamic and static stimuli evoke disparate visual strategies in TBI populations, pointing to dissociation between static and dynamic processing systems. The comparison of the two experiments calls attention to the reliability of static stimuli in social cognition research.

The ADFES findings also highlighted a relationship between AOI and emotion. These results suggested that gaze patterns were goal-driven, as distinct visual strategies appeared to reflect attention to the most diagnostic area of the face for each emotion (e.g. mouth for identifying happiness and nose for recognising disgusted faces). This theory corroborates with pre-existing research documenting differences in gaze patterns in response to different emotions (Calvo et al., 2018; Eisenbarth & Alpers, 2011; Pérez-Moreno et al., 2016; Schurgin et al., 2014). The findings are also in line with the MASC normative and clinical data which found that participants displayed different gaze patterns in response to the seven subcategories of the MASC. It appeared to be the case that low-level visual processes, such as fixation duration and count, are stimulus-driven, and that attentional resources are automatically directed towards the most diagnostic and salient feature of the social scene (Schurgin et al., 2014; Vabalas & Freeth, 2016).

The TASIT findings indicated that the group living with TBI consistently scored lower on all three subtests of the TASIT, suggesting that, compared to controls, they found it difficult to understand emotions and mood states. During the EET, the group with TBI scored lower across all emotions compared to the control group, showing diminished recognition of spontaneous emotional expressions. This is similar to other studies specifically using the EET (McDonald et al., 2003; McDonald et al., 2018), and research with similar designs (Hornak et al., 1996; Knox & Douglas, 2009; Ubukata et al., 2014). In line with similar research (McDonald, Fisher, Flanagan & Honan, 2017; McDonald et al., 2003), the group affected by TBI had lower scores across the sincere, simple-sarcastic and paradoxical-sarcastic conversational exchanges compared to the control group. TBI participants also displayed difficulties in understanding the actor's beliefs, meanings, intentions, and feelings compared to controls. The SI-E results indicated that the group with TBI had significantly lower accuracy scores for the sarcastic but, interestingly, there was no significant difference between the two groups during the lie

vignettes. These findings are consistent with McDonald et al., (2003) who also found that individuals with TBI displayed impairments when trying to decipher sarcastic exchanges, but scored in a typical range during the lie conditions when compared with matched controls. It appears to be the case that the ability to comprehend sarcasm is diminished post-TBI but the ability to recognise lies remains intact. This hypothesis has previously been presented by McDonald (2000). This specific finding highlights the conceptual differences between sarcasm and lying and suggests that these processes may be mediated by disparate neural networks. Another finding from the TASIT was that the groups performed similarly across videos depicting emotions and thoughts, but the group affected by TBI had significantly lower accuracy scores on the intention and meaning vignettes compared to controls. These combined findings support the hypothesis that global social cognition impairments are present post-TBI, spanning across verbal and non-verbal processes (Bird & Parente, 2013; McDonald et al., 2014).

The TASIT presents complex audio-visual social stimuli where verbal and non-verbal social cues need to be integrated to correctly identify mental or emotional states. It was hypothesised that abnormal low-level visual processing would have a relationship with the TASIT performance. However, there were no group differences in visual strategies across the three sub-tests of the TASIT and no significant correlations between accuracy scores and eye-tracking metrics. This finding suggests that the individuals affected by TBI's poor performance on the TASIT was not related to social perception deficits (i.e. disrupted fixation patterns) but must be reliant on impairments in upstream cognitive processes, such as the retrieval of social knowledge or response selection. Furthermore, this finding supports the null findings of the ADFES dynamic task but conflicts with the MASC and ADFES static results which indicated that the group with TBI did display abnormal gaze patterns compared to controls.



An interesting finding from the TASIT eye-tracking analysis was that all participants displayed distinct fixation patterns in response to the seven basic emotions of the EET. This result supports findings from the ADFES and MASC where visual strategies reflected the attention to the most diagnostic area of the social scene, as well as previous research documenting similar findings (Calvo et al., 2018; Eisenbarth & Alpers, 2011; Schurgin et al., 2014). The implication of this finding could extend to post-TBI interventions where individuals are coached to redirect their gaze to the most salient visual area. However, the current research findings are not clear-cut, and the group affected by TBI did not consistently display aberrant gaze patterns in response to all social stimuli. Therefore, this approach may prove unsuccessful and further work is needed to see if development is warranted.

Finally, the IAPS study indicated that there were no differences between the TBI and control group's fixation patterns when viewing humans, animals and objects. This finding is in contrast with Douglas et al., (2010) who reported that a group of individuals affected by TBI generated abnormal gaze patterns (i.e. increased number, duration and dispersion of fixations) in response to faces, but typical gaze patterns in response to objects when compared to matched controls. Differences in gaze patterns were observed across the IAPS in response to stimuli category indicating that emotional displays (e.g. humans and mammals) generated more rapid and sustained attentional demands compared to non-emotional displays (e.g. objects). This result was expected as previous research has reported similar findings (Crouzet et al., 2010; Frank et al., 2009; Powell, Kosakowski & Saxe, 2018). Overall, the IAPS findings suggest that, on a very basic level (i.e. static displays), individuals with TBI appear to generate typical gaze patterns in response to social and non-social stimuli compared to matched controls.

In sum, the behavioural data from the current research programme lends support to the proposal that social cognition is disrupted post-TBI, particularly facial affect recognition. From the

MASC and ADFES static experiments, it appeared that TBI may have visual strategies compared to controls. However, results uncovered in the TASIT, ADFES dynamic and IAPS studies suggest the antithesis, with the TBI and control group generating similar eye scan patterns. It is not evident why the results are non-uniform. One explanation could be that the stimuli used were so variable that is difficult to directly compare them, particularly the MASC which was dubbed from German into English. Nevertheless, the overall analyses of eye-tracking data suggested that differences in fixation patterns did not appear to explain the low accuracy scores exhibited by the group affected by TBI for the identification of facial expressions, mood state and conversational meaning. There were also no significant correlations between facial expression recognition scores and eye-tracking metrics for either the static or dynamic ADFES conditions. This result implies that impairments in emotion perception post-TBI may not stem from low-level visual or spatial frequency disruptions, but may be related to higher-level cognitive processes. These inconsistent results highlight that gaze patterns are highly complex and, although they may be disrupted post-TBI, the visual system may still collect the necessary information it needs to process social interactions (i.e. this may be demonstrated through the non-significant correlations between eye-tracking metrics and accuracy scores). Combined, the findings of the four studies enhance the understanding of the nature of social cognition in both normative and TBI cohorts and suggest that further research is needed to understand the mechanisms underpinning social cognition impairments post-TBI.

#### **10.4. Evaluation of research**

This research aimed to fill a gap in knowledge about fixation strategies in response to static and dynamic social stimuli post-TBI. Eye-tracking offers an experimental strategy to unravel the low-level visual processes underpinning social and emotion perception. Not only is this

exploration constructive from a social cognition theoretical perspective, but it could also facilitate the development of emotion perception rehabilitation programmes post-TBI. The research also tapped into the different verbal and non-verbal domains of social cognition (e.g. deciphering thoughts, emotions and intentions), providing a fine-grained analysis of the low-level social process in normative and TBI samples.

A second strength of the research was the inclusion of dynamic stimuli. The majority of extant studies have employed static stimuli which lack ecological validity and mundane realism (Alves, 2013; Knox & Douglas, 2009), making it hard to generalise results. Therefore, the current research builds upon the small evidence base using dynamic research (McDonald et al., 2003; Rosenberg et al., 2019). Furthermore, the current research was also able to directly compare low-level processes in response to static and dynamic stimuli, with findings indicating differences in underlying visual strategies, highlighting the methodological concern that static stimuli are not a true representative of real-world social cognition (Juslin et al., 2018; Namba et al., 2018).

Although the current research tried to utilise the most contemporary social cognition assessments, the tools could still be criticised on the basis that they are limited (e.g. still frequently using the six basic Ekman (1992) emotions) and posed expressions (Juslin et al., 2018; Namba et al., 2018). Furthermore, the social stimuli were not designed to be used in conjunction with eye-tracking. At present, there are no specifically designed social cognition tools which have been developed to be used with eye-tracking and, therefore, the reliability and validity of this methodology are not certain. The MASC has previously been used in combination with eye-tracking in the autism literature (Bast, Baumeister, Dziobek, Banaschewski & Poustka, 2016) but this does not detract from the fact that the assessment was not originally designed for use with eye-tracking. One of the issues with using non-specific

eye-tracking stimulus is that the design of AOIs, upon which the analysis depends, will not have been considered. The size and placement of AOIs are important and the design of the stimulus should balance selectivity and sensitivity. Although including more ecologically valid tools, such as the MASC and TASIT, provides a potentially more realistic insight into social cognition functioning post-TBI, there were limitations to their inclusion. Both the MASC and TASIT include small AOIs, due to the movie-like feel of the tools, while the ADFES displays quite large AOIs. Too small AOIs are at risk of excluding valid gaze data while larger AOIs increase sensitivity and runs the risk of collecting extraneous gaze data. Tobii Pro has also highlighted that the space between AOIs should be factored into the design of eye-tracking stimuli, advising researchers to implement a one-degree measure between AOIs to ensure sufficient spacing, a factor not considered during the MASC or TASIT. The present research aimed to utilize the most contemporary pre-existing social cognition tools to offer insightful findings regarding low-level visual strategies underpinning social cognition post-TBI. Future research could focus on designing social cognition tools which are specifically designed to be used in conjunction with eye-tracking, thus eliminating the confounding variables of selectivity and sensitivity encountered during the current programme of research.

The present research only analysed visual strategies in terms of fixation count and duration. This decision was theoretically-driven by research documenting that the majority of visual processing occurs during a fixation, with very little visual processing during saccades (Auyeung et al., 2015; Helo, Pannasch, Sirri & Rämä, 2014; Leigh & Zee, 2015; Vassallo et al., 2010), as well as research indicating that saccades were less impacted by TBI than fixations (Mancuso et al., 2015). With this in mind, the current thesis only provides a partial exploration of gaze patterns underpinning social cognition. Indeed, there is evidence documenting that TBI alters saccades (Antoniades & Kennard, 2015) so future research could investigate potential differences in saccade patterns in TBI and matched control groups. It may be the case that,

although fixation count and fixation duration appear similar between TBI and controls on certain social cognition tasks, the pattern of these fixations (i.e. where the individual looks first, second, third and so on) could be different. Aberrant saccade patterns could potentially lead to delayed behavioural reactions which, in a dynamic and fast-paced social environment, could cause social difficulties. On a similar note, it should also be highlighted that there are limitations in terms of precision of the Tobii T120 eye-tracking equipment, and more fine-grained analysis may be possible with upgraded Tobii products.

Screening for risk, whilst necessary for the safety of the researcher, the participants themselves (e.g. ensuring the participant was deemed well enough to undertake the research), and others (e.g. the research was carried out on a University campus), this exclusion criteria could have restricted the representativeness of the TBI sample and, therefore, affected the external validity of the research findings. In a narrative literature review of studies exploring treatments for neurological disorders, Trivedi and Humphreys (2015) reported that exclusion criteria's prevent approximately 3 in 4 individuals from participating in neurological research, including TBI studies. This raises concerns regarding the generalizability of findings, as well as ethical questions around excluding individuals from vulnerable or marginalised populations (e.g. individuals who do not have mental capacity) and, thus, creating a research knowledge shadow. Indeed, there tends to be a 'protection by exclusion' mindset when it comes to research with individuals lacking mental capacity (Shepherd, Wood, Griffith, Sheehan & Hood, 2019) but leading research organisations, such as the UK's National Institute for Health Research, are seeking to identify under-represented groups to develop innovative clinical trials. Trivedi and Humphreys (2015) recommended that exclusion criteria should only be implemented when there is a strong rationale. However, this recommendation is somewhat idealistic when applied to real-world studies where research groups must go through rigorous ethical procedures. Furthermore, concomitant effects of TBI will also limit or skew the representativeness of

recruited samples as individuals with low motivation or mood disorders are unlikely to be able to participate in research, particularly lengthy repeated measures designs. More work is needed around the field of inclusion and exclusion criteria in TBI research and specific recommendations on how to overcome some of the barriers often incurred during ethics and recruitment. One limitation of the current research was that clinicians did not document how many potential participants were excluded during the current screening process. Future research could capture this data to determine the proportion of individuals with TBI who are excluded from potential research projects and the reasons for these exclusions.

The current programme of research utilised consecutive recruitment, which means that every individual meeting the inclusion criteria were approached until the overall sample size was achieved. It was hoped that this approach might counterbalance some of the deleterious effects of the restrictive exclusion criteria as well as reducing the risk of inducing bias by seeking specific impairments relevant to the research (e.g. only recruiting individuals who displayed social cognition impairments daily). However, one of the limitations of consecutive recruitment in the present research is that it resulted in a wide variation of TBI severity and causes.

Indeed, time since injury was very wide-ranging in the current sample of TBI individuals. The present research methodology was based on previously published research which had also recruited samples with large differences in the time since injury. To ensure that the research was exploring chronic impairments, rather than acute impairments, the current programme employed the inclusion criterion that individuals had to be one-year post-TBI to be recruited. It could be argued that social cognition deficits may improve over time with brain plasticity or that individuals who had sustained their injury many years ago may have developed coping strategies. However, social cognition impairments post-TBI are documented to be enduring (from around one year) and do not appear to improve (Blair & Cipolotti, 2000; Tranel et al.,

2002; Ietswaart et al., 2008; Milder's et al., 2003). Indeed, it appears that social cognition, particularly emotion perception and ToM deficits, remain stable over time. There are accounts that socioemotional impairments post-TBI may worsen over time. This may be related to reduced social feedback as individuals with TBI often experience relationship breakdowns years after the injury (Wood & Yurdakul, 1997), and frequently do not return to work or hobbies (Humphrey et al., 2013). Therefore, the individual's social network reduces and they have fewer opportunities to have interpersonal interactions and social skills may weaken even further. This social isolation may even lead to frustration and aggressive behaviours which are also reported to increase over time (Williams et al., 2020). What is unclear is whether this deterioration is associated with a decline in social cognition (e.g. emotion recognition worsening over time) or whether it is related more to proxy ratings. There are reports that it takes proxy's (e.g. family/friends) a significant amount of time to come to terms with socioemotional changes post-TBI (e.g. at least a year but possibly more – Spikman et al., 2013). Future research could explore this area to determine whether social cognition deficits remain stable over time but environmental factors exacerbate them, or whether there is a deterioration in social skills (e.g. poorer emotion recognition 10 years post-injury compared to five years post-injury) and the factors for this (e.g. brain cell death, age). Indeed, controlling for time since injury may shed light on the persistence of social cognition deficits post-TBI.

TBI is heterogeneous in cause, pathology, and severity and poses as a methodological issue during the design and analysis of most TBI research. However, this is mainly the case for clinical trials which contain treatment and non-treatment groups where there could be an imbalance of baseline characteristics (Roozenbeek, Lingsma & Maas, 2012). As the current research programme was a non-clinical trial, and the individuals with TBI were matched to controls, heterogeneity was considered to be a low confounding variable. However, this minimal attention to heterogeneity may be a key factor contributing to the high percentage of

TBI research which reports inconsistent findings. It could be suggested that studies should recruit more homogeneous samples (e.g. only include individuals with severe TBI who have had a fall) which should, technically, eliminate this confounding variable. However, from a practical point of view, strict inclusion criteria would increase the screening and recruitment timeframe, it would lower the percentage of individuals eligible for the study (creating a barrier to taking part in research), and the results would also not be generalizable to the TBI population. It may be the case that research with larger sample sizes could control for heterogeneity during analyses, although for instance, determining cut-offs for severity is not standardized across the research field (Stein, 2015). Stein put it nicely when he wrote: “Complaining about patient heterogeneity and the experimental variance caused by it needs to be replaced with more proactive management and less ambitious, more carefully focused enrolment criteria for TBI trials”. This is something which the researcher will keep in mind for future research.

One potential confounding variable in the current research programme is the diversity of the individuals with TBI in terms of verbal IQ (ranging from the mid-50s to more than 135). However, despite this heterogeneity, overall group differences in social cognitive functioning were still found. Nevertheless, it could be argued that this variance may have masked results from the individual research studies. The researcher decided not to exclude participants with the lowest/highest performance from analysis for several reasons. Firstly, IQ was employed as a third matching criterion, not a screening measure. While gender and age were the primary matching criteria, it was hoped that the introduction of the third criteria would reduce the risk of confounding variables between the group with TBI and the control group. Existing eye-tracking studies with TBI populations only employ age and gender as the matching criterion (Douglas et al., 2010; Vassallo et al., 2011), while many emotion perception studies with TBI participants use age, gender and years of education to match participants (Croker & McDonald, 2005; Milders et al., 2003; Spikman et al., 2013), not IQ. With many TBI studies not screening



for IQ (e.g. Milders et al., 2003; Spell & Frank, 2000; Spikman et al., 2013) it did not seem necessary to exclude participants based on this optional factor. This point was even more pertinent as it is well known that recruitment for TBI studies is protracted and often sample sizes, and thus power, are small and low. Secondly, when considering the nature of the high and low scores, it was decided by the researcher that they were legitimate ‘real world’ observations. TBI is documented to negatively affect IQ (Königs, Engenhorst & Oosterlaan, 2015; Parker & Rosenblum, 1996), particularly in severe TBI, which the majority of participants had sustained. Indeed, with the heterogeneous nature of TBI (Maas, 2016) combined with the observation that the recruitment streams were from low socioeconomic areas, it did not seem surprising to the researcher that there was large variability within the IQ scores of individuals with TBI. Moreover, as discussed in detail during chapter five, the current research programme was opposed to transforming the data and felt strongly about maintaining the integrity of the raw data. Thirdly, research has demonstrated that there is no correlation between social cognition and IQ (Baksh, Abrahams, Auyeung & MacPherson, 2018; Mohn, Sundet & Rund, 2014), consequently, as this thesis was interested in social cognition post-TBI, it did not seem appropriate to exclude participants or control for low IQ levels during statistical analysis. Fourthly, to account for potential verbal deficits, all of the studies employed a forced-choice format, mitigating communication impairments. The researcher found the WASI time consuming to administer, particularly with individuals who had sustained TBI. As the WASI was part of a larger neuropsychological test battery which often fatigued both individuals with and without TBI, combined with the points above, on reflection, future research studies might consider removing this as a matching criterion.

The present sample size was based on previous research which implemented similar methodology. Although the sample size was moderate, it is acknowledged that the study may not have had sufficient power to detect small differences between the groups. As *p* values are

influenced by sample sizes, and to ensure that misleading conclusions were not presented, effect sizes were included to try and mitigate any uncertainties (Cabella et al., 2017). One of the challenges of the research project was the slow and low rate of referrals for individuals with TBI. This may have been a result of too stringent inclusion/exclusion criterion or time constraints experienced by both the referring consultants. One way to combat both of these barriers would be to collaborate with several NHS Trusts, thus increasing referral streams.

As discussed earlier in the thesis, it is likely that social cognition impairments are influenced by pre and post-TBI variables, for example, pre-morbid personality traits, pre and post-TBI mood disorders, psychiatric conditions, and/or substance abuse use. Factors such as mood or substance abuse would more than likely exacerbate social cognition problems post-TBI. If the individual experienced more than one of these factors (e.g. an individual started taking recreational drugs to alleviate low mood) then this is likely to significantly impact on social functioning. These factors are also likely to have an impact on motivation and, therefore, the chances of an individual taking part in research is decreased, particularly if the design is repeated measures/ longitudinal. Individuals with mood or substance abuse might also be excluded from research to control for confounding variables. On the other hand, individuals may also display 'reserved' social functioning which may benefit them during the adjustment period post-TBI (Frank, 2011; Sela-Kaufman et al., 2013) and social cognition may appear less impacted. Extant literature on social reserve is mainly focused on neurodegenerative diseases and while the evidence base is slowly growing for TBI it is still insufficient (Nunnari, Bramanti & Marino, 2014). Future research could explore the role of cognitive reserve in brain injury psychosocial outcomes.

## 10.5. Implications

Normative data is particularly important in clinical neuropsychology as it allows the comparison of an individual with a representative group to determine whether there are deficits in cognitive functions (Lezak, Howieson & Loring, 2004; Strauss et al., 2006). The normative MASC data demonstrated that there were differences in fixation patterns in response to different domains of social cognition, which was further replicated in the clinical MASC data. The MASC is a popular social cognition tool which is frequently cited in the literature (Lahera et al., 2014; Martinez et al., 2017; Preißler et al., 2010). If the subcategories of the MASC were further validated then future research could include them in their analysis, providing a finer-grained analysis of mindreading abilities compared to the current scoring system. Despite its reliability and prevalence in other research fields, the MASC has not been used in the TBI literature; hence, the clinical study undertaken in the current programme of research has preliminarily established task parameters, reliability and sensitivity with a TBI cohort.

The MASC normative study also found that there were no sex differences in either eye-tracking metrics or accuracy scores on the MASC. This finding has implications for the extant evidence base documenting that there is a female superiority on social cognition tasks (Olderbak et al., 2018). Males performed on par with females across the different subcategories of the MASC and they displayed similar gaze patterns in terms of fixation counts and fixation durations. It appears that the purported female advantage in non-verbal emotion recognition tasks are small and it might be the case that the introduction of dynamic stimuli eliminates these sex differences. Indeed, recent research has suggested that there are significant differences in visual strategies underpinning static and dynamic social processing (Blais et al., 2017; Stoesz and Jakobson, 2013). These findings highlight the need for future research to employ dynamic, rather than static, stimuli. Moreover, findings from normative sex research have implications

for clinical studies as some scholars theorise that sex may be a protective factor for social cognition abilities post-TBI. Unfortunately, normative and clinical evidence is equivocal regarding a potential female advantage on social cognition tasks and findings are complex (Rigon et al. 2008; Zupan et al., 2017). Possible reasons are task-specific demands, and/or use of non-naturalistic static stimuli and varied methodologies across studies. Future research could aim to disentangle these inconsistent findings with ecologically valid stimuli.

Although the current gaze data findings were non-uniform, there were significant differences between the TBI and control groups fixation patterns on two social cognition assessments (one static and one dynamic). These findings should not go unnoticed and, although there were no significant correlations between eye-tracking metrics and emotion accuracy scores, the fact that TBI appears to disrupt gaze patterns, although inconsistently, warrants that low-level visual processes be taken into consideration when assessing psychosocial processing post-TBI, both in clinical and research settings. These findings also suggested that eye-tracking might be a useful tool to measure post-TBI changes, potentially as a diagnostic tool, even for mild TBI which is notoriously difficult to detect. Indeed, recent reports have suggested that eye-tracking can be used as a potential diagnostic biomarker and outcome measure for brain injury (Samadani, 2016).

As previously discussed, social cognition is an area which is often not assessed nor rehabilitated post-TBI. Social cognition functioning after brain injury can vary greatly depending on a range of injury-related (e.g. severity, pathology location, mechanism of trauma), personal factors (e.g. pre-morbid personality factors and mood/psychiatric conditions) and social influences (e.g. employment status, social network size, culture). All of these elements would affect cognitive and behavioural changes post-TBI and would need to be factored into psychosocial rehabilitation or intervention treatments. Psychosocial assessment and rehabilitation post-TBI

is still in its infancy (Kelly et al., 2017) but there is evidence which suggests that visual scanning training may be beneficial (Cicerone et al., 2011; Neumann, Zupan, Tomita & Willer, 2009). However, the paucity of eye-tracking studies with TBI populations, particularly those focusing on social cognition, combined with the lack of consistency between findings in the current research programme, might challenge the efficacy underpinning the incorporation of visual scanning training in post-TBI interventions. With the lack of evidence-based research supporting the theory that low-level visual processes are disrupted after TBI, and that these low-level processes can be successfully rehabilitated, it would be inappropriate for public health services to offer visual scanning training post-TBI. However, it is important to note that although the present findings were inconsistent, a number of the current studies did find aberrant gaze patterns in the group affected by TBI. This is also supported by pre-existing research which has also documented aberrant gaze patterns in groups of individuals affected by TBI (Vassallo et al., 2010, 2011). Therefore, gaze patterns following TBI need to be investigated further, particularly as visual scanning training could offer a clinically practical and low-cost intervention. Indeed, one of the novel aspects of the current programme of research was that no other studies had explored gaze-patterns in a sample of TBI individuals when they viewed static and dynamic social stimuli. When searching the autism or schizophrenia eye-tracking literature, both have a plethora of research and this is what the TBI evidence-base is lacking. Future research needs to replicate the basic design of the current thesis, i.e. exploring the low-level visual processes underpinning social cognition post-TBI. This knowledge will provide the evidence-base with a clearer picture of whether low-level processes are disrupted as a result of TBI and whether they are related to social cognition impairments. Future research should consider implementing dynamic, 'real world' social stimuli which are specifically designed to be used with eye-tracking technology. A superior method would be to record gaze patterns during non-posed dyadic interactions and this is

becoming increasingly possible with the rapid development of eye-tracking technology. For example, Rogers, Speelman, Guidetti and Longmuir (2018) used dual eye-tracking to uncover personal gaze patterns during face-to-face social interactions. This type of study design would also eliminate the limitations of labelling and forced-choice paradigms which the current research, and indeed many studies exploring social cognition post-TBI, have employed. A potential confounding variable in TBI research is communication problems, such as aphasia (Marsh & Hillish, 2006). Forced choice paradigms mitigate these communication challenges and support participants who are reticent to guessing but who might implicitly be able to provide the correct response. Nevertheless, forced-choice paradigms are far removed from real-world social interactions and, therefore, data collected using this method lack mundane realism. Furthermore, there is a large variation in people's vocabularies and they may not resonate with one of the forced labels (e.g. fearful) but they may thinking of a synonym (e.g. scared). To ensure this latter point is alleviated, researchers should ensure that participants understand the meaning of each label and could provide definitions and synonyms, similar to the verbatim instructions of the TASIT.

While the overall findings of the current research do not present a clear picture of the underpinnings of social cognition deficits post-TBI, it appears possible that one or more stages of social processing may be disrupted. This finding has implications regarding the knowledge base concerning the underlying mechanisms of disrupted social cognition post-TBI. As correlations were all non-significant between eye-tracking metrics and accuracy scores on the social cognition assessments, it is unlikely that impairments are due to low-level visual processing disturbances, although this should not be ruled out until further research is explored. It appears more likely that impairments must occur in the subsequent 'higher-order' stages of social cognition; interpretation and processing of social information, and response selection (Adolphs, 2010; Ostrom, 1984; Penn et al., 1997). It may be difficult to disentangle the

underlying mechanisms of social cognition impairments post-TBI as the three stages of the process all heavily rely on interconnected brain regions, particularly frontal circuitry (Amodio & Frith, 2006; Bicks et al., 2015; Wittmann et al., 2018; Xiao et al., 2017), which are vulnerable to damage during TBI (Bigler, 2013). However, future studies could investigate the brain areas which sub-serve these higher-order processes and test these processes directly for impairment. This knowledge could inform post-TBI assessment and rehabilitation. Indeed, Yeates (2014) suggested that more success could be gained in the field of neurorehabilitation if the underlying impairment mechanisms were fully understood. Furthering the knowledge base could potentially allow clinicians to target specific deficits rather than resorting to a global intervention approach (Yeates, 2014).

## **10.6. Conclusion**

The current research presented in this thesis expands on the existing literature pertaining to social cognition in typical cohorts as well as impairments as a direct effect of TBI. There is a plethora of data documenting social cognition impairments post-TBI yet the mechanisms underpinning these are not well understood (Babbage et al., 2011; Knox & Douglas, 2009; McDonald 2013). The current research employed static and dynamic social stimuli, contrasting to previous research, as well as a fine-grained analysis measuring both low-level (visual strategies) and high-level (identifying facial expressions) social cognition in a TBI and control group. In line with existing research, findings revealed that the group affected by TBI consistently scored lower on emotion perception and mindreading tasks compared to the control group. The consistency of these findings strongly supports the theory that TBI disrupts social cognition. The current research found that the group living with TBI scored significantly lower on all of the facial affect recognition assessments, therefore, the overall findings expand on the well-established knowledge base concerning facial affect recognition impairments post-

TBI (Babbage et al., 2011). Of particular interest was the finding that the group with TBI appeared to have diminished abilities to recognise sarcasm but intact abilities to recognise lies. This appears to suggest that these two social processes are modulated by different brain networks and that the underpinnings of sarcasm processing may be more susceptible to damage during TBI. Further research exploring the intricacies of sarcasm and deception comprehension post-TBI is needed as both processes are vital for successful social functioning (Haiman, 1998). The group affected by TBI also exhibited aberrant visual strategies compared to controls on the ADFES static and the MASC but not on the remaining tasks. Referring back to Penn et al., (1997) social cognition model, it may be the case that the individuals in the TBI group displayed selective impairments during the very initial stages of social cognition, the perception of social cues, but this was not consistent across all tasks. Furthermore, correlational analysis exploring the relationship between fixation patterns and emotion perception scores were also non-significant. It might be more probable that TBI disrupts the remaining two stages of social cognition (retrieval of social knowledge and response selection). However, the low-level differences found during the MASC and ADFES static experiments cannot be discounted and highlight the need for further investigation into eye-tracking post-TBI.



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# Appendices

## Appendix 1. Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983)

### Hospital Anxiety and Depression Scale (HADS)

Tick the box beside the reply that is closest to how you have been feeling in the past week.  
Don't take too long over you replies: your immediate is best.

D	A		D	A	
		<b>I feel tense or 'wound up':</b>			<b>I feel as if I am slowed down:</b>
3		Most of the time	3		Nearly all the time
2		A lot of the time	2		Very often
1		From time to time, occasionally	1		Sometimes
0		Not at all	0		Not at all
		<b>I still enjoy the things I used to enjoy:</b>			<b>I get a sort of frightened feeling like 'butterflies' in the stomach:</b>
0		Definitely as much	0		Not at all
1		Not quite so much	1		Occasionally
2		Only a little	2		Quite Often
3		Hardly at all	3		Very Often
		<b>I get a sort of frightened feeling as if something awful is about to happen:</b>			<b>I have lost interest in my appearance:</b>
3		Very definitely and quite badly	3		Definitely
2		Yes, but not too badly	2		I don't take as much care as I should
1		A little, but it doesn't worry me	1		I may not take quite as much care
0		Not at all	0		I take just as much care as ever
		<b>I can laugh and see the funny side of things:</b>			<b>I feel restless as I have to be on the move:</b>
0		As much as I always could	3		Very much indeed
1		Not quite so much now	2		Quite a lot
2		Definitely not so much now	1		Not very much
3		Not at all	0		Not at all
		<b>Worrying thoughts go through my mind:</b>			<b>I look forward with enjoyment to things:</b>
3		A great deal of the time	0		As much as I ever did
2		A lot of the time	1		Rather less than I used to
1		From time to time, but not too often	2		Definitely less than I used to
0		Only occasionally	3		Hardly at all
		<b>I feel cheerful:</b>			<b>I get sudden feelings of panic:</b>
3		Not at all	3		Very often indeed
2		Not often	2		Quite often
1		Sometimes	1		Not very often
0		Most of the time	0		Not at all
		<b>I can sit at ease and feel relaxed:</b>			<b>I can enjoy a good book or radio or TV program:</b>
0		Definitely	0		Often
1		Usually	1		Sometimes
2		Not Often	2		Not often
3		Not at all	3		Very seldom

Please check you have answered all the questions

#### Scoring:

Total score: Depression (D) \_\_\_\_\_ Anxiety (A) \_\_\_\_\_

0-7 = Normal

8-10 = Borderline abnormal (borderline case)

11-21 = Abnormal (case)

## Appendix 2. Michigan Alcoholism Screening Test (MAST; Selzer, 1971)

### The Michigan Alcoholism Screening Test (MAST)

Please circle either Yes or No for each item as it applies to you.

1. Do you feel you are a normal drinker? (By normal we mean you drink less than or as much as most other people.)	Yes	No
2. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening?	Yes	No
3. Does your wife, husband, a parent, or other near relative ever worry or complain about your drinking?	Yes	No
4. Can you stop drinking without a struggle after one or two drinks?	Yes	No
5. Do you ever feel guilty about your drinking?	Yes	No
6. Do friends or relatives think you are a normal drinker?	Yes	No
7. Are you able to stop drinking when you want to?	Yes	No
8. Have you ever attended a meeting of Alcoholics Anonymous (AA)?	Yes	No
9. Have you gotten into physical fights when drinking?	Yes	No
10. Has your drinking ever created problems between you and your wife, husband, a parent, or other relative?	Yes	No
11. Has your wife, husband (or other family members) ever gone to anyone for help about your drinking?	Yes	No
12. Have you ever lost friends because of drinking?	Yes	No
13. Have you ever gotten into trouble at work or school because of drinking?	Yes	No
14. Have you ever lost a job because of drinking?	Yes	No
15. Have you ever neglected your obligations, your family or your work for two or more days in a row because you were drinking?	Yes	No
16. Do you drink before noon fairly often?	Yes	No
17. Have you ever been told you have liver trouble? Cirrhosis?	Yes	No
18. After heavy drinking have you ever had Delirium Tremens (D.T.s) or severe shaking, or heard voices or seen things that really were not there?	Yes	No
19. Have you ever gone to anyone for help about your drinking?	Yes	No
20. Have you ever been in a hospital because of drinking?	Yes	No
21. Have you ever been a patient in a psychiatric hospital or on a psychiatric ward of a general hospital where drinking was part of the problem that resulted in hospitalization?	Yes	No
22. Have you ever been seen at a psychiatric or mental health clinic, or gone to any doctor, social worker, or clergyman for help with an emotional problem, where drinking was part of the problem?	Yes	No
23. Have you ever been arrested for drunk driving, driving while intoxicated, or driving under the influence of alcoholic beverages? (If YES, how many times? _____)	Yes	No
24. Have you ever been arrested, or taken into custody even for a few hours, because of other drunk behavior? If YES, how many times? _____)	Yes	No

## Scoring the MAST

The version of the MAST included on this web site was provided by Professor Selzer, who indicated that the version published in 1971 in the American Journal of Psychiatry was modified in 1980.

In scoring the MAST points are assigned to a response depending upon whether the item is worded positively or negatively.

For items 1, 4, 6 and 7 negative answers are consistent with alcoholic responses.

For items 2, 3, 5, and 9-25 positive responses are consistent with alcoholic responses.

The scale assigns a 1-5 weighting to each of the items, with a rating of 5 being considered diagnostic of alcoholism. Questions that were highly discriminating were given a value of two points and others assigned a one-point value. An alcoholic response to questions 8, 19, or 20 is considered diagnostic and is assigned a value of five points.

A total score is computed as a sum of item values as seen in the table below. Total scores range from 0 to 53.

### MAST Point System

Question	Points Assigned
1. (negative responses are alcoholic)	2
2.	2
3.	1
4. (negative responses are alcoholic)	2
5.	1
6. (negative responses are alcoholic)	2
7. (negative responses are alcoholic)	2
8.	5
9.	1
10.	2
11.	2
12.	2
13.	2
14.	2
15.	2
16.	1
17.	2
18.	2
19.	5
20.	5
21.	2
22.	2
23.	2
24.	2

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Citation: Selzer ML: The Michigan Alcoholism Screening test (MAST): the quest for a new diagnostic instrument. American Journal of Psychiatry 3:176-181, 1975

## Appendix 3. Drug Abuse Screening Test (DAST; Skinner, 1982)

### Substance Abuse Screening Instrument (O4/05)

*The Drug Abuse Screening Test (DAST) was developed in 1982 and is still an excellent screening tool. It is a 28-item self-report scale that consists of items that parallel those of the Michigan Alcoholism Screening Test (MAST). The DAST has "exhibited valid psychometric properties" and has been found to be "a sensitive screening instrument for the abuse of drugs other than alcohol.*

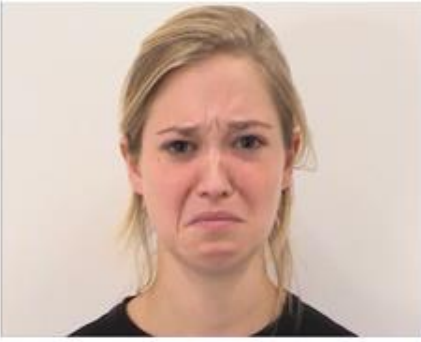
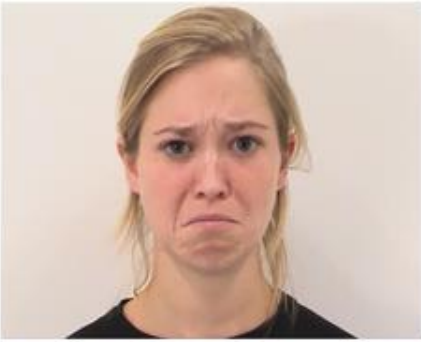
### The Drug Abuse Screening Test (DAST)

**Directions:** The following questions concern information about your involvement with drugs. Drug abuse refers to (1) the use of prescribed or "over-the-counter" drugs in excess of the directions, and (2) any non-medical use of drugs. Consider the past year (12 months) and carefully read each statement. Then decide whether your answer is YES or NO and check the appropriate space. Please be sure to answer every question.

		YES	NO
1.	Have you used drugs other than those required for medical reasons?	___	___
2.	Have you abused prescription drugs?	___	___
3.	Do you abuse more than one drug at a time?	___	___
4.	Can you get through the week without using drugs (other than those required for medical reasons)?	___	___
5.	Are you always able to stop using drugs when you want to?	___	___
6.	Do you abuse drugs on a continuous basis?	___	___
7.	Do you try to limit your drug use to certain situations?	___	___
8.	Have you had "blackouts" or "flashbacks" as a result of drug use?	___	___
9.	Do you ever feel bad about your drug abuse?	___	___
10.	Does your spouse (or parents) ever complain about your involvement with drugs?	___	___
11.	Do your friends or relatives know or suspect you abuse drugs?	___	___
12.	Has drug abuse ever created problems between you and your spouse?	___	___
13.	Has any family member ever sought help for problems related to your drug use?	___	___
14.	Have you ever lost friends because of your use of drugs?	___	___
15.	Have you ever neglected your family or missed work because of your use of drugs?	___	___
16.	Have you ever been in trouble at work because of drug abuse?	___	___
17.	Have you ever lost a job because of drug abuse?	___	___
18.	Have you gotten into fights when under the influence of drugs?	___	___
19.	Have you ever been arrested because of unusual behavior while under the influence of drugs?	___	___
20.	Have you ever been arrested for driving while under the influence of drugs?	___	___
21.	Have you engaged in illegal activities in order to obtain drug?	___	___
22.	Have you ever been arrested for possession of illegal drugs?	___	___
23.	Have you ever experienced withdrawal symptoms as a result of heavy drug intake?	___	___
24.	Have you had medical problems as a result of your drug use (e.g., memory loss, hepatitis, convulsions, bleeding, etc.)?	___	___
25.	Have you ever gone to anyone for help for a drug problem?	___	___
26.	Have you ever been in a hospital for medical problems related to your drug use?	___	___
27.	Have you ever been involved in a treatment program specifically related to drug use?	___	___
28.	Have you been treated as an outpatient for problems related to drug abuse?	___	___

*Scoring and interpretation:* A score of "1" is given for each YES response, except for items 4,5, and 7, for which a NO response is given a score of "1." Based on data from a heterogeneous psychiatric patient population, cutoff scores of 6 through 11 are considered to be optimal for screening for substance use disorders. Using a cutoff score of 6 has been found to provide excellent sensitivity for identifying patients with substance use disorders as well as satisfactory specificity (i.e., identification of patients who do not have substance use disorders). Using a cutoff score of <11 somewhat reduces the sensitivity for identifying patients with substance use disorders, but more accurately identifies the patients who do not have a substance use disorders. Over 12 is definitely a substance abuse problem. In a heterogeneous psychiatric patient population, most items have been shown to correlate at least moderately well with the total scale scores. The items that correlate poorly with the total scale scores appear to be items 4,7,16,20, and 22.

**Appendix 4. Diagram to illustrate an emotion (sad) unfolding for the ADFES dynamic condition**



## Appendix 5. Diagram to illustrate the sequence of events for the static condition

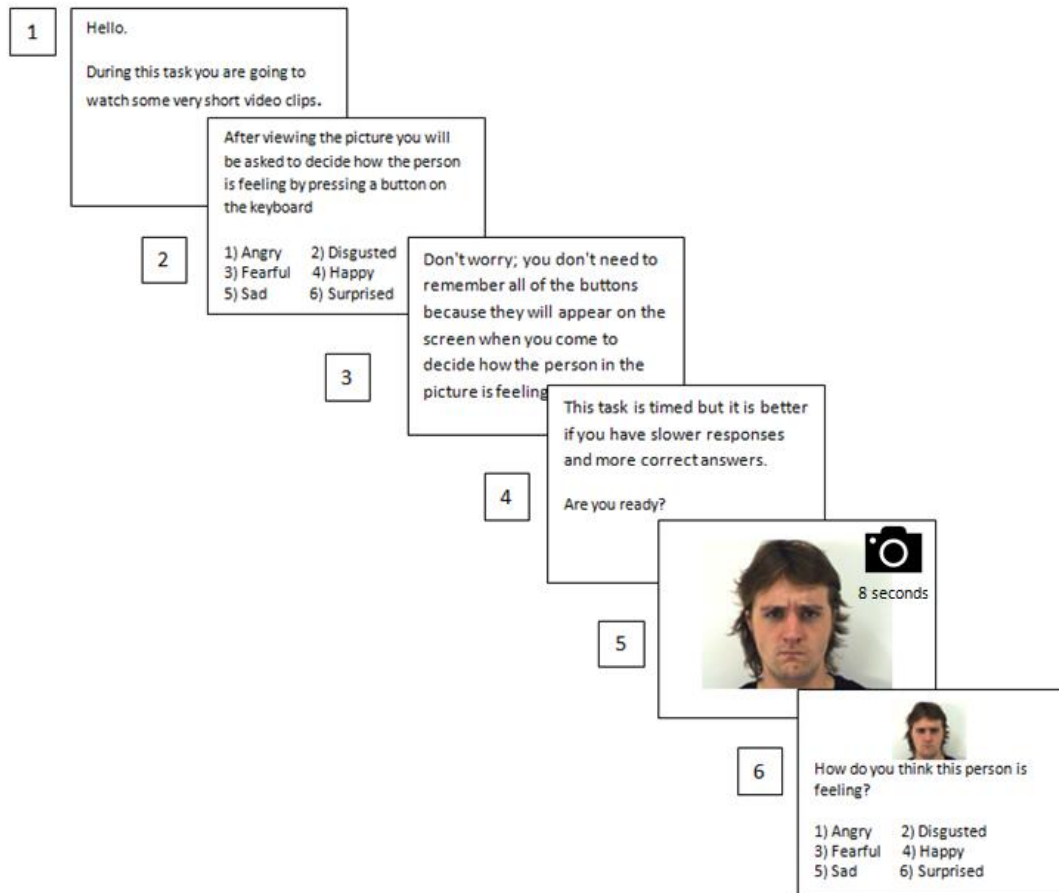
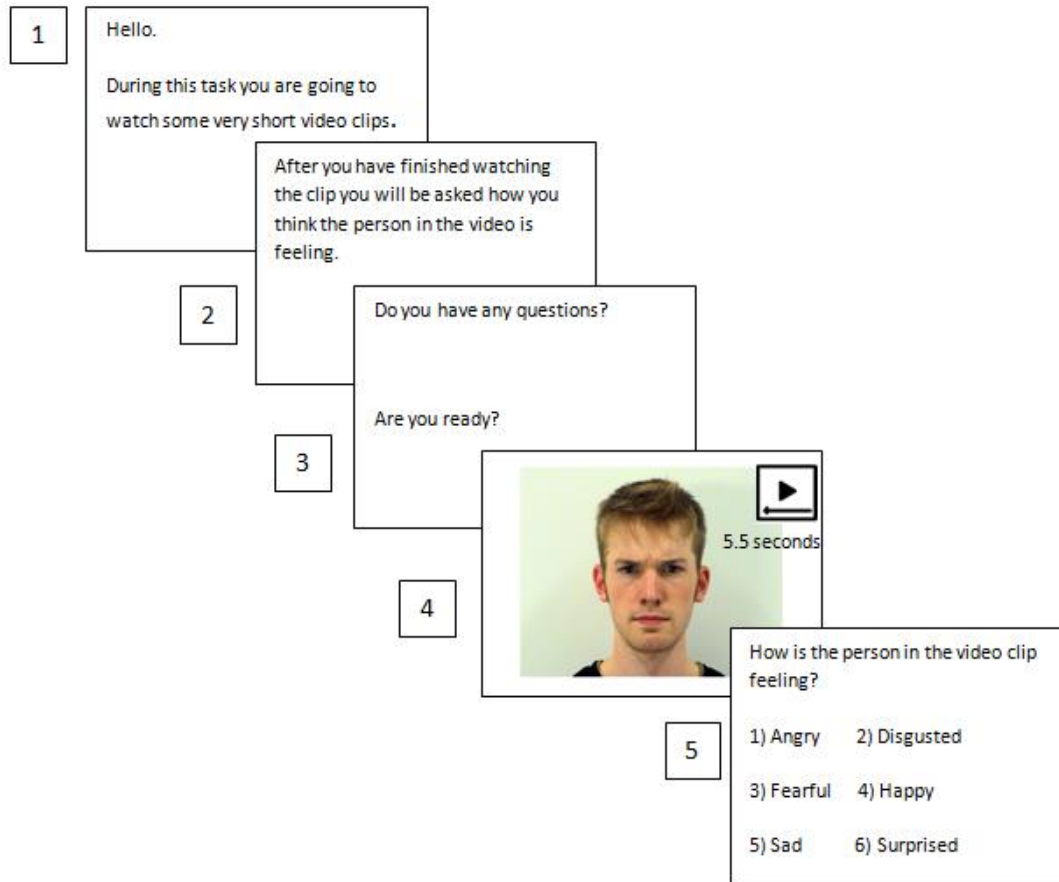


Diagram to illustrate the sequence of events for the static condition. The participants pressed the space bar to move the instructions on at their own pace. The facial expression image appeared for eight seconds before automatically moving onto the answer screen. Only eye tracking data collected during screen five was analysed.

## Appendix 6. Diagram to illustrate the sequence of events for the dynamic condition



The participant would indicate when they had read the instructions and the researcher would move the test on by pressing the space bar. The video lasted for 5.5 seconds before automatically moving onto the answer screen. Only eye tracking data collected during screen four was analysed.

**Appendix 7. An example of a MASC clip and associated question**

**Picture 1.** Cliff is the first one to arrive at Sandra's house for the dinner party. He and Sandra seem to enjoy themselves when Cliff is telling a story about his vacation in Sweden.



**Picture 2.** When Michael arrives, he dominates the conversation, directing his speech to Sandra alone.





**Picture 3.** Slightly annoyed by Michael’s bragging story, Sandra shortly looks in Cliff’s direction and then asks Michael: “Tell me, have you ever been to Sweden?”



**Question 17.** Why is Michael telling this story?

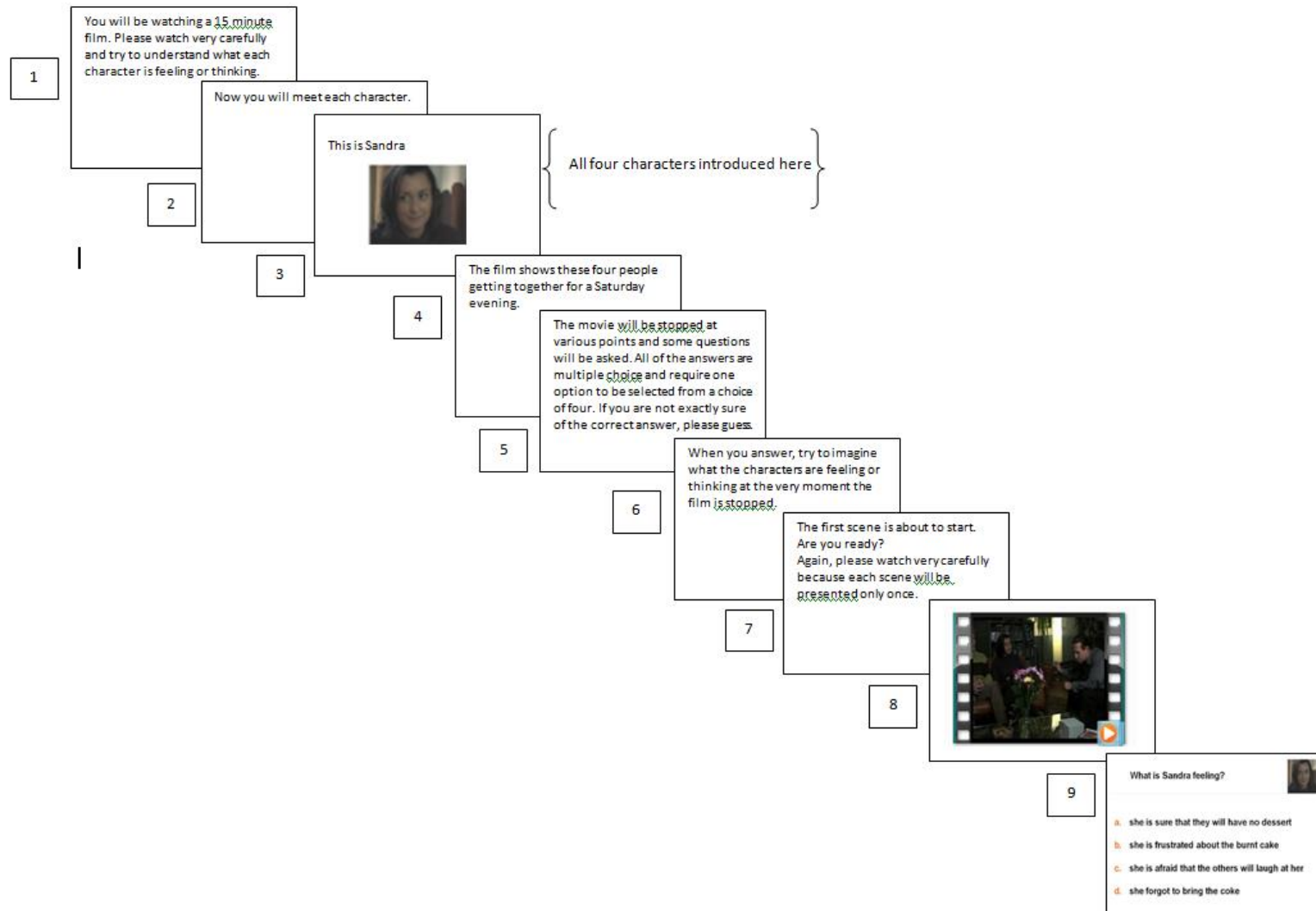
for Sandra to realize that he is the best guy to date

he wants to impress Sandra

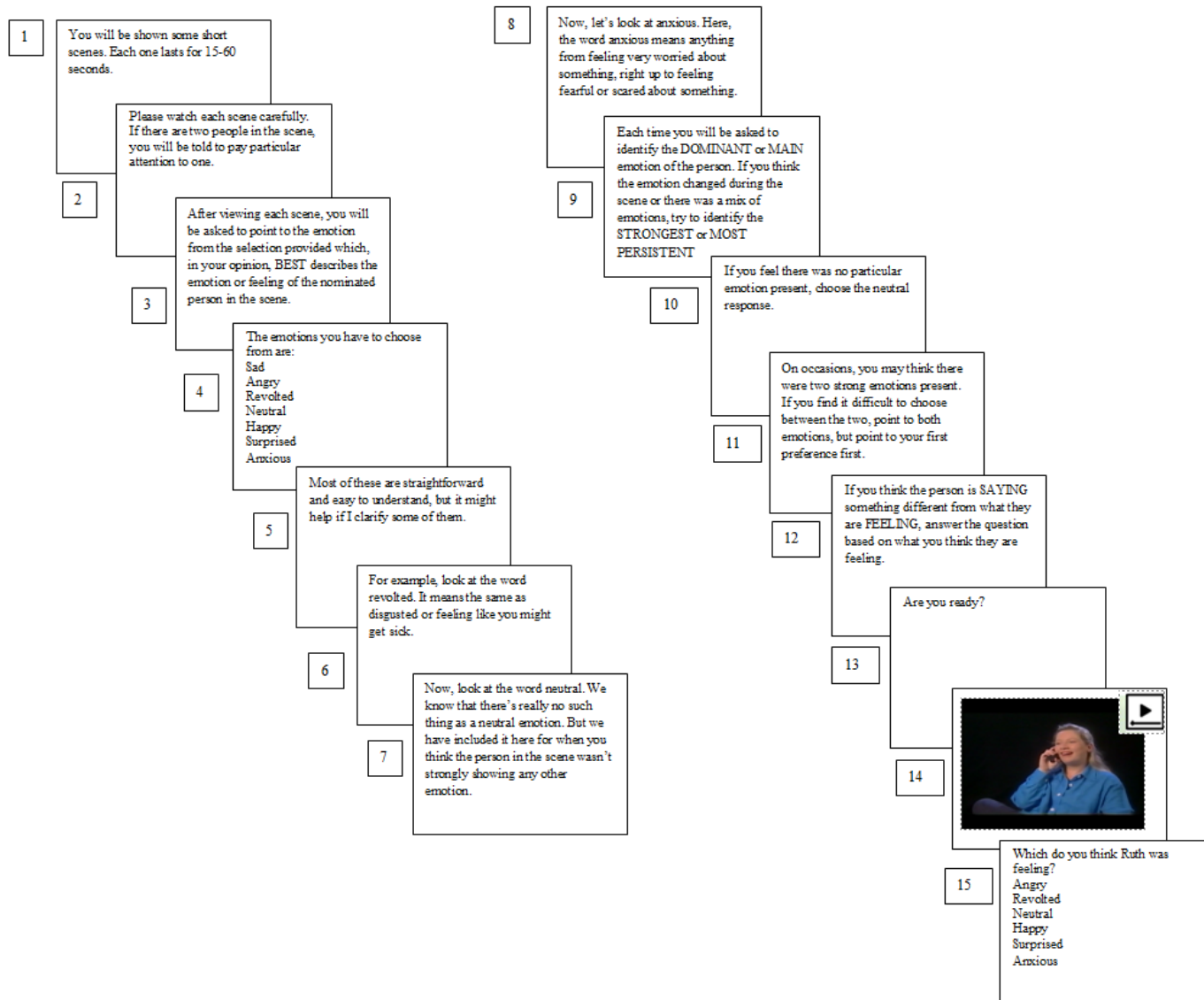
he thinks the story is interesting

the incident just happened today

## Appendix 8. Diagram to illustrate the sequence of events for the MASC



## Appendix 9. Diagram to illustrate the sequence of events for the Emotion Evaluation Test (EET)



**Appendix 10. An example of sample scripts and probe questions used during the Social Inference–Minimal Test Sample and Social Inference–Enriched Test in the TASIT**

Sample Scripts and Probe Questions Used in Social Inference–Minimal Test

**SINCERE OR SIMPLE SARCASTIC ITEMS---PARALINGUISTIC CUES ONLY  
(ENACTED EITHER SARCASTICALLY OR SINCERELY)**

Ruth: Great movie, wasn't it?

Michael: Oh, yeah, great.

Ruth: I thought it was terrific. I was on the edge of my seat.

Michael: Oh, me too, on the edge of my seat

Ruth: Well, weren't you surprised by the ending?

Michael: Oh, yeah, the ending was a huge surprise.

Ruth: I thought the actors were very good. I really liked that main girl.

Michael: She was unbelievable, and the guy opposite her—what a performance!

Ruth: It's a shame it's closing. I'd like to see it again.

Michael: Yeah, what a shame. I feel I could see it another dozen times.

**Probe questions**

Act: Is Michael agreeing with Ruth about the movie?

Say: Does he mean the actors were good?

Think: Does he think the movie was bad?

Feel: Did he enjoy the movie?

**PARADOXICAL SARCASM---SCRIPT IS NONSENSICAL EXCEPT IF ONE  
SPEAKER IS ASSUMED TO BE INSINCERE**

Gary: Have you got your ticket?

Keith: Nope. I tore it up and threw it away.

Gary: Good. And your passport's safe?

Keith: Sure, I threw that in the bin along with my ticket.

Gary: So, you've got everything.

**Probe questions**

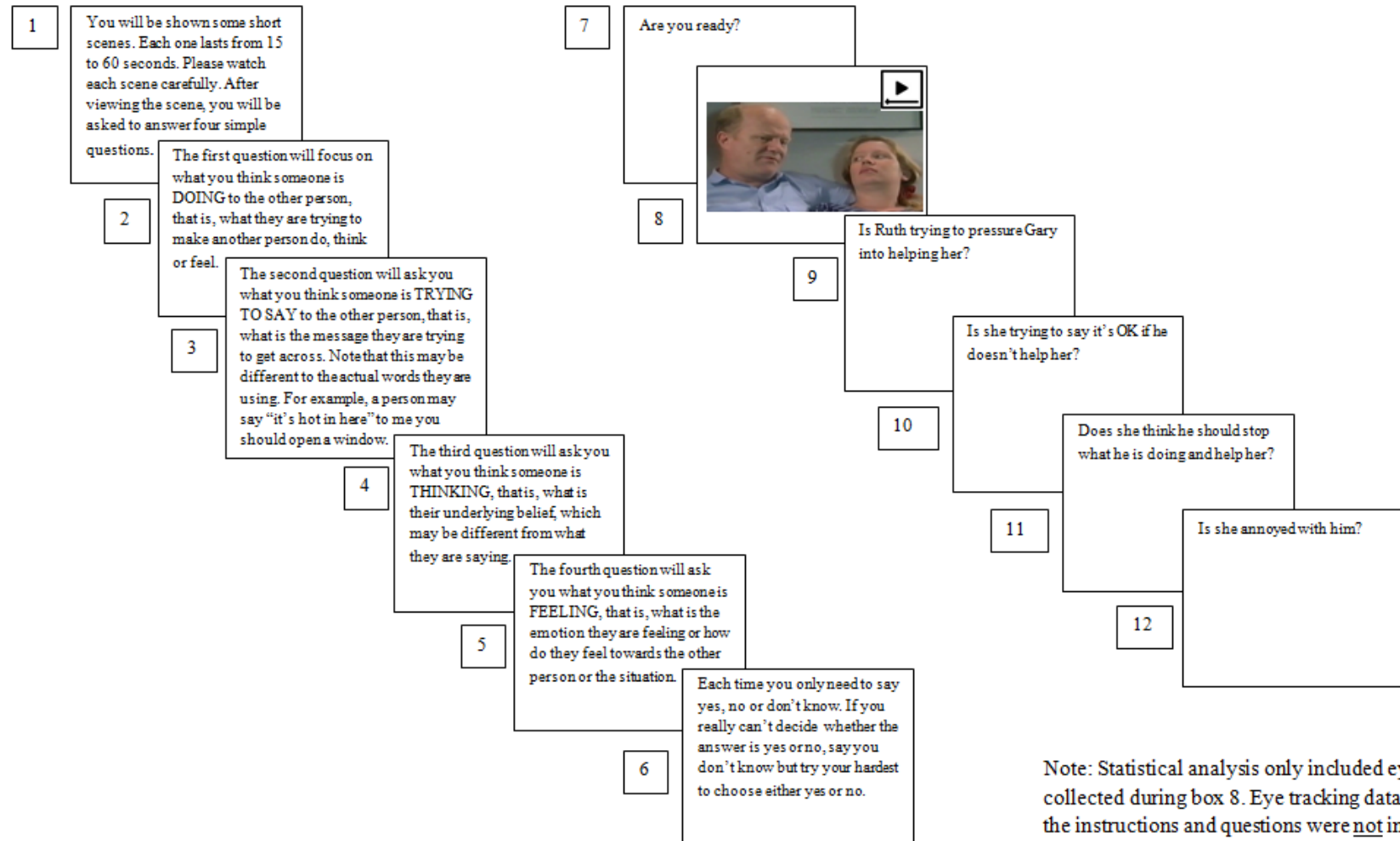
Do: Is Keith seriously trying to make Gary think he's lost his ticket?

Say: Does Keith mean he has got his ticket and passport?

Think: By the end of the scene, does Gary think Keith has his ticket?

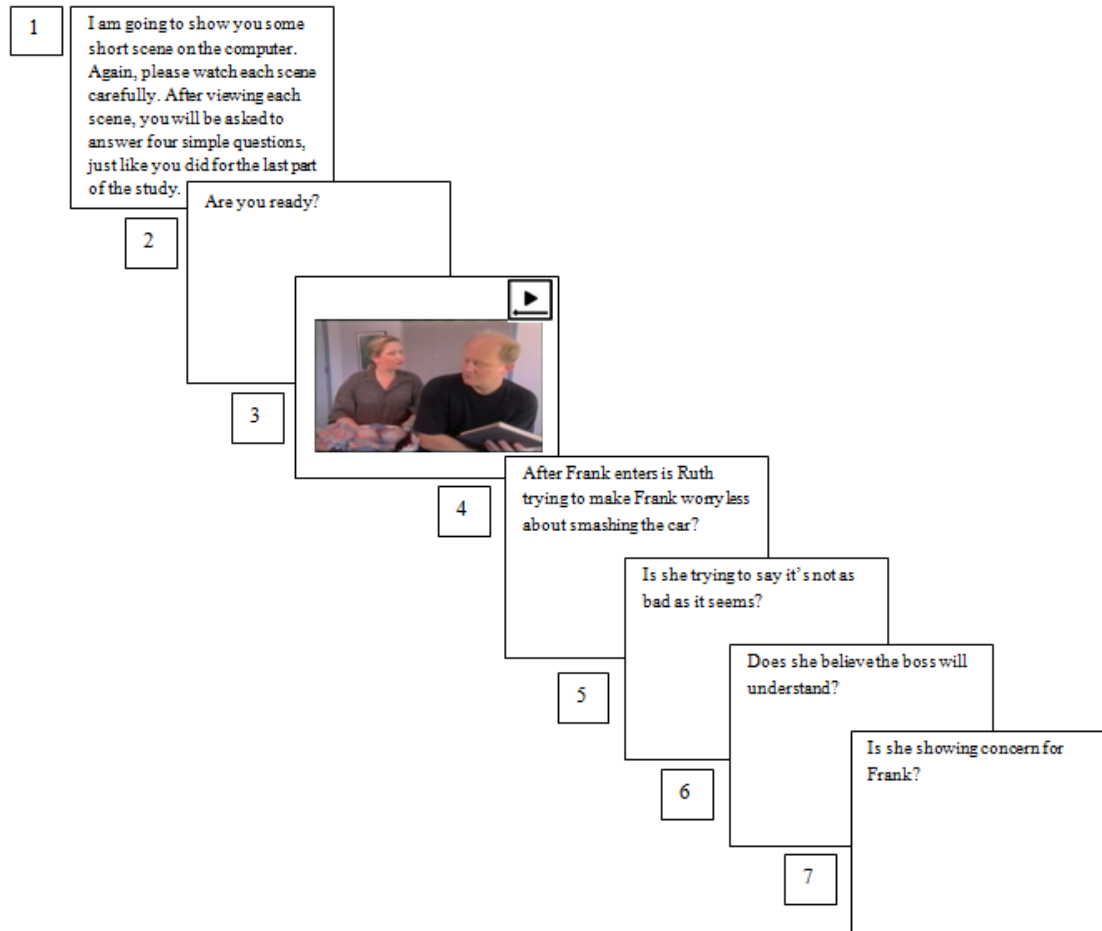
Feel: Is Keith grateful that Gary checked about his ticket?

## Appendix 11. Diagram to illustrate the sequence of events for the Social Inference–Minimal Test



Note: Statistical analysis only included eye tracking data collected during box 8. Eye tracking data collected during the instructions and questions were not included in the analysis.

## Appendix 12. Diagram to illustrate the sequence of events for the Social Inference–Enriched Test



Note: Statistical analysis only included eye tracking data collected during box 3. Eye tracking data collected during the instructions and questions were not included in the analysis.

**Appendix 13. Image numbers, category and category mean valence, arousal and dominance for IAPS images.**

<b>IAPS Number</b>	<b>Category</b>	<b>Mean Valence</b>	<b>Mean Arousal</b>	<b>Mean Dominance</b>
2495	Human	5.22	3.19	5.77
2020	Human	5.68	3.34	5.99
2830	Human	4.73	3.64	5.33
2211	Human	5.19	4.05	5.25
2010	Human	4.38	3.56	5.03
2000	Human	6.51	3.32	6.65
2516	Human	4.90	3.50	5.54
2002	Human	4.95	3.35	5.89
2030	Human	6.71	4.54	5.60
2512	Human	4.86	3.46	4.86
2102	Human	5.16	3.03	5.16
2513	Human	5.80	3.29	5.16
2101	Human	4.49	3.46	5.25
2019	Human	6.07	4.31	5.89
2190	Human	4.83	2.41	5.92
2005	Human	6.00	4.07	6.00
1630	Mammal (deer)	7.26	4.45	6.12
1920	Mammal (dolphin)	7.90	4.27	6.50
1410	Mammal (ferret)	7.00	4.17	6.05
1660	Mammal (gorilla)	6.49	4.57	5.46
1720	Mammal (lion)	6.79	5.32	4.63
1350	Mammal (pig)	5.25	4.37	5.60
1440	Mammal (seal pup)	8.19	4.61	6.05
1820	Non-mammal (alligator)	5.35	5.67	4.66
1603	Non-mammal (butterfly)	6.90	3.37	6.57
1275	Non-mammal (cockroach)	3.30	4.81	5.11
1910	Non-mammal (fish)	6.71	3.29	6.44
1121	Non-mammal (gecko)	5.79	4.83	5.89
1908	Non-mammal (jelly fish)	5.28	4.88	4.75
1903	Non-mammal (prawn)	5.5	4.25	6.01
1931	Non-mammal (shark)	4.00	6.80	3.51
1080	Non-mammal (snake)	4.24	5.69	4.33
1947	Non-mammal (squid)	5.85	4.35	5.84
1390	Non-mammals (wasps)	4.50	5.29	4.75
7010	Object (basket)	4.94	1.76	6.70
7090	Object (book)	5.19	2.61	6.65
7006	Object (bowl)	4.88	2.33	6.18
7057	Object (cup and saucer)	5.35	3.39	5.33
7058	Object (dice)	5.29	3.98	5.29
7043	Object (drill)	5.17	3.68	5.89
7175	Object (lamp)	4.87	1.72	6.47
7009	Object (mug)	4.93	3.01	6.33
7026	Object (picnic table)	5.38	2.63	6.19
7056	Object (pliers)	5.07	3.07	5.07
7000	Object (rolling pin)	5.00	2.42	6.14
7014	Object (scissors)	5.15	3.25	6.21
7004	Object (spoon)	5.04	2.00	6.74
7032	Object (trainers)	4.82	3.18	5.90
7052	Object (washing pegs)	5.33	3.01	6.43



<b>Category</b>	<b>Mean Valence</b>	<b>Mean Arousal</b>	<b>Mean Dominance</b>
<b>Human</b>	85.48	56.52	89.29
<b>Mammals</b>	48.88	31.76	40.41
<b>Non-mammals</b>	57.42	53.23	57.86
<b>Objects</b>	76.41	42.04	91.52

#### Appendix 14. Counterbalancing design

Set A	Set B
HADS	CORVIST
VOSPB	DAST
MAST	VOSPB
CORVIST	WASI
DAST	HADS
WASI	MAST
MASC	ADFES dynamic
ADFES static	Control (IAPS)
TASIT	ADFES static
ADFES dynamic	MASC
Control (IAPS)	TASIT

## **Appendix 15. Definitions of Tobii descriptive statistics**

### **Time to First Fixation (Seconds).**

Time to first fixation is the amount of time it takes until the participant fixates on an active AOI from the start of the media (video or picture). The measuring of time begins when the media containing the AOI is first displayed. For time measurement to start an AOI does not have to be active and the activation state of the AOI (active/inactive) does not affect the time measurement start point. Even if the AOI is inactive time measurement will begin. Time measurement will only stop when the participant fixates on an active AOI.

If media containing the same AOI is displayed several times during the recording but there are also aspects of the media where the AOI is not present then the time to first fixation will be calculated by the addition of each recorded media time displaying the AOI until the active AOI is fixated on by the participant. Media not containing the AOI is excluded from the recording time calculation.

If the participant does not fixate on an AOI by the end of the recording then the time to first fixation value will not be provided and will not be incorporated in the descriptive statistics calculations (e.g. when computing the N value).

### **First Fixation Duration (Seconds)**

This calculation measures the first fixation duration on an active AOI. If the participant does not fixate on an AOI by the end of the recording then the first fixation value will not be provided and will not be incorporated in the descriptive statistics calculations (e.g. when computing the N value). ‘Cut off’ fixations affect the descriptive statistics for this calculation.

### **Total Fixation Duration (Seconds)**

This calculation is the sum of the duration for all fixations within an active AOI. The N value is therefore based on the number of recordings. ‘Cut off’ fixations affect the descriptive statistics for this calculation. If the participant does not fixate on an AOI by the end of the recording then the total fixation duration value will not be provided and will not be incorporated in the descriptive statistics calculations (e.g. when computing the N value).

**Total Fixation Duration (Zeros)**

This is calculated in the same way as Total Fixation Duration, however, if at the end of the recording the participant has not fixated on an active AOI, the total fixation duration will be documented as zero and the recording will be included in the descriptive statistics calculations (e.g. when computing N and means).

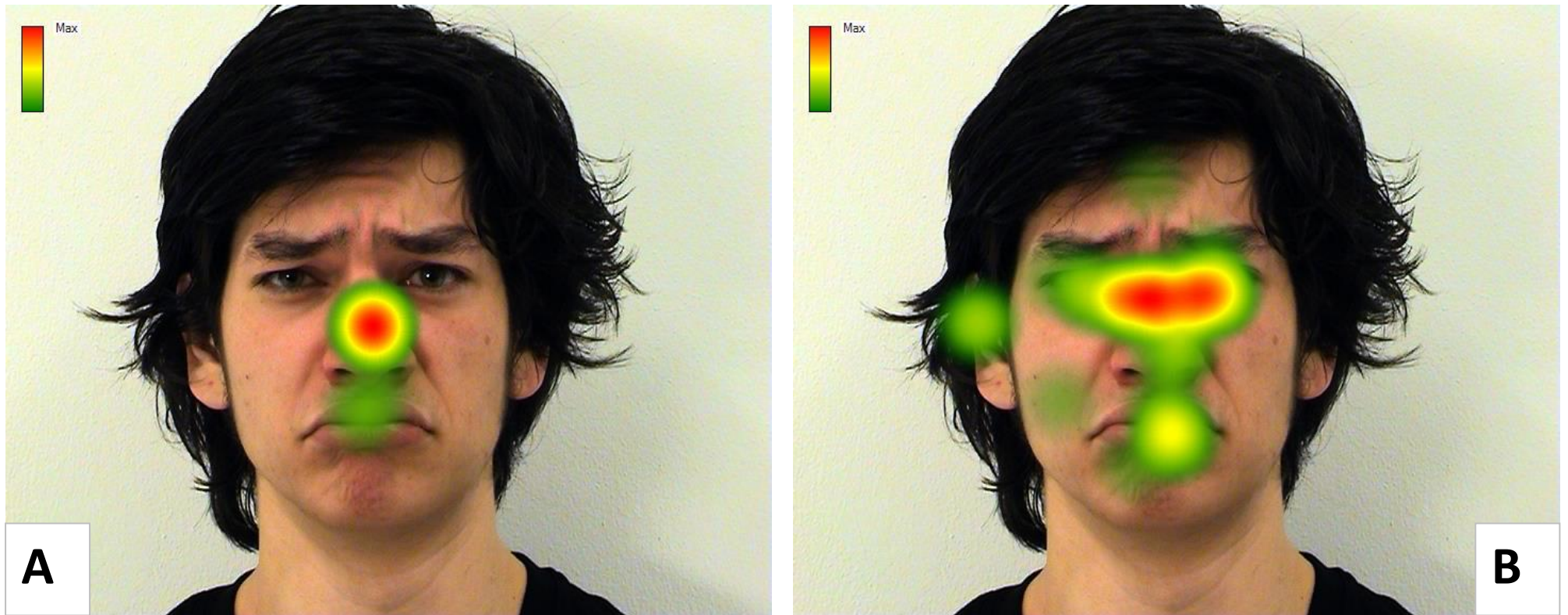
**Fixation Count (Count)**

This calculates the number of times the participant fixates on an active AOI. If the participant stops fixating on an AOI and moves their attention to another area of the media but later fixates again on the AOI then all fixation counts will be amalgamated at the end of the media. If the participant does not fixate on an AOI by the end of the recording then the fixation count value will not be provided and will not be incorporated in the descriptive statistics calculations (e.g. when computing the N value).

**Fixation Count (Zeros)**

This is calculated in the same way as Fixation Count, however, if at the end of the recording the participant has not fixated on an active AOI, the fixation count value will be documented as zero and the recording will be included in the descriptive statistics calculations (e.g. when computing N and means).

## Appendix 16. Heat maps for the static ADFES task



A heat map to illustrate the disparity between eye scan patterns for a male TBI participant (A) and a matched control participant (B) when viewing a static sad facial expression. The heat map displays the number and length of fixations using the different colours with red indicating maximum levels.

### Appendix 17. Post-hoc tests for the TASIT EET (fixation duration)

Post-hoc paired samples t tests exploring the effect of emotion on fixation duration to the eyes following the significant ANOVA interaction between AOI and emotion.

Emotion	Mean difference (SD)	t	95% CI	Sig
Anger-Disgust	0.36 (0.63)	3.46	0.15-0.58	.001*
Anger-Anxious	0.73 (0.98)	4.48	0.40-1.07	≤.001*
Anger-Happy	0.22 (0.82)	1.59	-0.06-0.49	.121
Anger-Sad	-2.70 (2.79)	-5.80	-3.64- -1.75	≤.001*
Anger-Surprise	0.52 (0.96)	3.26	0.20-0.84	.002*
Anger-Neutral	-0.77 (1.42)	-3.25	-1.25- -0.29	.003*
Disgust-Anxious	0.37 (0.73)	3.03	0.12-0.62	.005*
Disgust-Happy	-0.15 (0.76)	-1.16	-0.41-0.11	.253
Disgust-Sad	-3.06 (2.93)	-6.26	-4.05- -2.67	≤.001*
Disgust-Surprise	0.16 (0.81)	1.16	-0.12-0.43	.253
Disgust-Neutral	-1.14 (1.49)	-4.56	-1.64- -0.63	≤.001*
Anxious-Happy	-0.52 (0.83)	-3.76	-0.80- -0.24	.001*
Anxious-Sad	-3.43 (3.08)	-6.69	-4.47- -2.39	≤.001*
Anxious-Surprise	-0.21 (0.62)	-2.08	-0.42- -0.01	.045
Anxious-Neutral	-1.51 (1.80)	-5.03	-2.11- -0.90	≤.001*
Happy-Sad	-2.91 (2.75)	-6.35	-3.84- -1.98	≤.001*
Happy-Surprise	0.30 (0.85)	2.14	0.02-0.59	.039
Happy-Neutral	-0.99 (1.47)	-4.03	-1.49- -0.49	≤.001*
Sad-Surprise	3.22 (2.91)	6.62	2.23-4.20	≤.001*
Sad-Neutral	1.92 (2.39)	4.84	1.12-2.73	≤.001*
Surprise-Neutral	0.36 (0.74)	2.94	0.11-0.61	.006*

\*Significant at  $\alpha = .01$ .

(2) Post-hoc paired samples t tests exploring the effect of emotion on fixation duration to the nose following the significant one-way ANOVA interaction between AOI and emotion.

<b>Emotion</b>	<b>Mean difference (SD)</b>	<b>t</b>	<b>95% CI</b>	<b>Sig</b>
Anger-Disgust	0.16 (1.17)	0.81	-0.24-0.55	.422
Anger-Anxious	0.16 (1.13)	0.85	-0.22-0.54	.404
Anger-Happy	0.73 (1.03)	4.50	0.39-1.08	≤.001*
Anger-Sad	-0.57 (1.86)	-1.83	-1.20-0.06	.076
Anger-Surprise	0.65 (0.91)	4.29	0.34-0.96	≤.001*
Anger-Neutral	-0 ≤.0011 (1.18)	-0.001	-0.40-0.40	.999
Disgust-Anxious	0.001 (1.29)	0.005	-0.43-0.44	.996
Disgust-Happy	0.58 (1.43)	2.42	0.09-1.06	.021
Disgust-Sad	-0.73 (2.04)	-2.14	-1.41- -0.04	.040
Disgust-Surprise	0.49 (1.17)	2.52	0.09-0.89	.017
Disgust-Neutral	-0.16 (1.43)	-0.67	-0.64-0.32	.508
Anxious-Happy	0.57 (1.45)	2.37	0.08-1.07	.023
Anxious-Sad	-0.73 (1.56)	-2.80	-1.26- -0.20	.008*
Anxious-Surprise	0.49 (1.21)	2.43	0.08-0.90	.020
Anxious-Neutral	-0.16 (1.42)	-0.67	-0.64-0.32	.505
Happy-Sad	-1.30 (2.35)	-3.33	-2.10- -0.51	.002*
Happy-Surprise	-0.09 (0.73)	-0.70	-0.33-0.16	.486
Happy-Neutral	-0.73 (1.16)	-3.79	-1.13- -0.34	.001*
Sad-Surprise	1.22 (2.28)	3.20	0.45-1.99	.003*
Sad-Neutral	0.57 (2.46)	1.38	-0.27-1.40	.176
Surprise-Neutral	-0.65 (1.00)	-3.89	-1.00—0.31	≤.001*

\*Significant at  $\alpha = .01$ .

(3) Post-hoc paired samples t tests exploring the effect of emotion on fixation duration to the mouth following the significant one-way ANOVA interaction between AOI and emotion.

<b>Emotion</b>	<b>Mean difference (SD)</b>	<b>t</b>	<b>95% CI</b>	<b>Sig</b>
Anger-Disgust	-0.31 (1.09)	-1.69	-0.68-0.06	.099
Anger-Anxious	0.64 (0.75)	5.12	0.38-0.89	≤.001*
Anger-Happy	-1.40 (1.41)	-5.93	-1.87- -0.92	≤.001*
Anger-Sad	-1.33 (2.08)	-3.84	-2.03- -0.62	.001*
Anger-Surprise	0.63 (0.72)	5.21	0.38-0.87	≤.001*
Anger-Neutral	-0.09 (1.09)	-0.52	-0.46-0.28	.609
Disgust-Anxious	0.95 (1.14)	5.00	0.56-1.33	≤.001*
Disgust-Happy	-1.09 (1.51)	-4.33	-1.60- -0.58	≤.001*
Disgust-Sad	-1.02 (1.60)	-3.83	-1.56- -0.48	.001*
Disgust-Surprise	0.93 (1.18)	4.75	0.53-1.33	≤.001*
Disgust-Neutral	0.21 (1.29)	1.00	-0.22-0.65	.324
Anxious-Happy	-2.03 (1.62)	-7.53	-2.58- -1.49	≤.001*
Anxious-Sad	-1.96 (2.25)	-5.25	-2.72- -1.21	≤.001*
Anxious-Surprise	-0.01 (0.61)	-0.13	-0.22-0.20	.901
Anxious-Neutral	-0.73 (1.25)	-3.52	-1.15- -0.31	.001*
Happy-Sad	0.07 (1.69)	0.24	-0.50-0.64	.808
Happy-Surprise	2.02 (1.37)	8.85	1.56-2.48	≤.001*
Happy-Neutral	1.30 (1.17)	6.68	0.91-1.70	≤.001*
Sad-Surprise	1.95 (2.15)	5.45	1.22-2.68	≤.001*
Sad-Neutral	1.23 (1.77)	4.17	0.63-1.83	≤.001*
Surprise-Neutral	-0.72 (1.03)	-4.19	-1.07- -0.37	≤.001*

\*Significant at  $\alpha = .01$ .



### Appendix 18. Post-hoc tests for the TASIT EET (fixation count)

(1) Post-hoc paired samples t tests exploring the effect of emotion on fixation count to the eyes following the significant one-way ANOVA interaction between AOI and emotion.

Emotion	Mean difference (SD)	T	95% CI	Sig
Anger-Disgust	1.00 (1.74)	3.44	0.41-1.58	.002*
Anger-Anxious	1.66 (2.33)	4.26	0.87-2.45	≤.001*
Anger-Happy	0.50 (1.75)	1.72	-0.09-1.09	.095
Anger-Sad	-2.54 (3.05)	-5.01	-3.58- -1.52	≤.001*
Anger-Surprise	1.60 (2.29)	4.18	0.82-2.37	≤.001*
Anger-Neutral	-1.08 (2.38)	-2.71	-1.88- -0.27	.010*
Disgust-Anxious	0.66 (1.27)	3.11	0.23-1.09	.004*
Disgust-Happy	-0.50 (1.56)	-1.90	-1.02-0.03	.066
Disgust-Sad	-3.54 (2.99)	-7.12	-4.56- -2.53	≤.001*
Disgust-Surprise	0.60 (1.34)	2.69	0.15-1.05	.011
Disgust-Neutral	-2.07 (2.58)	-4.82	-2.95—-1.20	≤.001*
Anxious-Happy	-1.16 (1.88)	-3.68	-1.79- -0.52	.001*
Anxious-Sad	-4.21 (3.18)	-7.94	-5.28- -3.13	≤.001*
Anxious-Surprise	-0.06 (1.00)	-0.37	-0.40-0.27	.714
Anxious-Neutral	-2.73 (3.24)	-5.07	-3.83- -1.64	≤.001*
Happy-Sad	-3.05 (3.20)	-5.71	-4.13- -1.97	≤.001*
Happy-Surprise	1.10 (1.86)	3.53	0.47-1.72	.001*
Happy-Neutral	-1.58 (2.52)	-3.78	-2.43- -0.73	.001*
Sad-Surprise	4.14 (3.39)	7.34	3.00-5.29	≤.001*
Sad-Neutral	1.47 (3.33)	2.65	0.36-2.60	.012
Surprise-Neutral	-2.67 (3.12)	-5.14	-3.73- -1.62	≤.001*

\*Significant at  $\alpha = .01$ .

(2) Post-hoc paired samples t tests exploring the effect of emotion on fixation count to the nose following the significant one-way ANOVA interaction between AOI and emotion.

<b>Emotion</b>	<b>Mean difference (SD)</b>	<b>t</b>	<b>95% CI</b>	<b>Sig</b>
Anger-Disgust	0.23 (1.90)	0.73	-0.41-0.87	.474
Anger-Anxious	0.96 (1.64)	3.52	0.41-1.52	.001*
Anger-Happy	0.73 (1.90)	2.30	0.08-1.37	.028
Anger-Sad	-0.46 (2.42)	-1.14	-1.28-0.36	.264
Anger-Surprise	1.11 (1.50)	4.41	0.60-1.62	≤.001*
Anger-Neutral	-0.05 (1.76)	-0.18	-0.65-0.54	.855
Disgust-Anxious	0.73 (1.94)	2.27	0.08-1.39	.029
Disgust-Happy	0.50 (2.30)	1.30	-0.28-1.28	.201
Disgust-Sad	-0.69 (2.52)	-1.64	-1.54-0.17	.111
Disgust-Surprise	0.88 (1.90)	2.79	0.24-1.53	.009*
Disgust-Neutral	-0.28 (2.17)	-0.78	-1.02-0.45	.439
Anxious-Happy	-0.23 (2.12)	-0.66	-0.95-0.49	.514
Anxious-Sad	-1.42 (2.22)	-3.84	-2.17- -0.67	≤.001*
Anxious-Surprise	0.15 (1.12)	0.80	-0.23-0.53	.429
Anxious-Neutral	-1.02 (1.89)	-3.22	-1.66- -0.38	.003*
Happy-Sad	-1.19 (2.91)	-2.45	-2.17- -0.20	.019
Happy-Surprise	0.38 (1.44)	1.60	-0.10-0.87	.118
Happy-Neutral	-0.78 (1.75)	-2.69	-1.37- -0.19	.011
Sad-Surprise	1.57 (2.35)	4.02	0.78-2.37	≤.001*
Sad-Neutral	0.40 (2.59)	0.94	-0.47-1.28	.355
Surprise-Neutral	-1.17 (1.79)	-3.91	-1.77- -0.56	≤.001*

\*Significant at  $\alpha = .01$ .

(3) Post-hoc paired samples t tests exploring the effect of emotion on fixation count to the mouth following the significant one-way ANOVA interaction between AOI and emotion.

<b>Emotion</b>	<b>Mean difference (SD)</b>	<b>t</b>	<b>95% CI</b>	<b>Sig</b>
Anger-Disgust	0.45 (1.32)	2.05	0.005-0.90	.048
Anger-Anxious	1.97 (1.97)	6.01	1.31-2.64	≤.001*
Anger-Happy	-1.65 (2.26)	-4.39	-2.41- -0.89	≤.001*
Anger-Sad	-1.01 (2.28)	-2.65	-1.78- -0.23	.012
Anger-Surprise	1.80 (1.51)	7.14	1.29-2.31	≤.001*
Anger-Neutral	0.37 (1.61)	1.37	-0.18-0.91	.179
Disgust-Anxious	1.52 (1.77)	5.15	0.92-2.12	≤.001*
Disgust-Happy	-2.10 (2.16)	-5.82	-2.83- -1.37	≤.001*
Disgust-Sad	-1.46 (1.85)	-4.72	-2.09- -0.83	≤.001*
Disgust-Surprise	1.35 (1.44)	5.64	0.86-1.84	≤.001*
Disgust-Neutral	-0.08 (1.82)	-0.27	-0.70-0.53	.786
Anxious-Happy	-3.62 (2.64)	-8.24	-4.51- -2.73	≤.001*
Anxious-Sad	-2.98 (2.75)	-6.50	-3.91- -2.05	≤.001*
Anxious-Surprise	-0.17 (1.11)	-0.93	-0.55-0.20	.357
Anxious-Neutral	-1.61 (2.05)	-4.70	-2.30- -0.91	≤.001*
Happy-Sad	0.64 (2.57)	1.50	-0.23-1.51	.143
Happy-Surprise	3.45 (2.28)	9.09	2.68-4.22	≤.001*
Happy-Neutral	2.02 (2.03)	5.95	1.33-2.71	≤.001*
Sad-Surprise	2.81 (2.38)	7.09	2.00-3.61	≤.001*
Sad-Neutral	1.38 (2.26)	3.65	0.61-2.14	.001*
Surprise-Neutral	-1.43 (1.66)	-5.18	-2.00- -0.87	≤.001*

\*Significant at  $\alpha = .01$ .

## Supplementary Materials

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### Case notes for TBI participants collected from A&E records and medical files

#### Participant A.N.

Participant A.N is a 26 year old male who sustained his brain injury at the age of 19 through an assault when he was punched in the face on a night out and knocked unconscious. He was rushed to A&E and on arrival to hospital had a GCS of 2 (Eyes= 1, Verbal= 2, Motor= 4). In the CT scanner A.N vomited which led to a likely case of pulmonary aspiration due to vomit at the back of the throat which resulted in intubation and ventilation.

A CT scan on the 28/07/2007 revealed a right temporoparietal shift fracture of the skull, a right temporoparietal acute subdural haematoma (SDH) with midline shift, a traumatic subarachnoid hemorrhage, cerebral oedema and massive right side brain swelling with right vesicles effaced. The non-depressed fracture was on the right speno-temporal region of the brain and extended superiorly into the right parietal bone. Beneath the fracture there was an acute sub-dural haematoma having a maximum depth of approximately 10mm. There was subarachnoid blood related to this extending into the Sylvian fissure and superiorly into the central sulcus. Scan interpretation reported that it was difficult to exclude extra dural blood here. Extra axial blood was producing some mass effect with parietal effacement of the right lateral ventricle and approximately 5mm midline shift towards the left but no other intracranial abnormality. It was estimated that there was grade 2 diffuse axonal injury associated with the injury.

A.N was operated on the same day he was admitted to A&E and underwent a decompressive craniectomy for the evacuation of right SDH and temporal clot and a cranioplasty operation to replace a skull defect. A.N then spent 12 days in a coma and demonstrated PTA for 14 days. On the 29/07/2007 GCS dropped to 2 (Eyes= 1, Verbal= 0, Motor= 1) and remained stable until 06/08/2007 where it elevated to 6 (Eyes= 1, Motor= 4, Verbal= 1) and on the 08/08/2007

to 10 (Eyes= 4, Motor= 5, Verbal= 1) and 10/08/2007 11 (Eyes=3, Motor=5, Verbal= 3). LOC, PTA and GCS all indicate that patient A.N sustained a severe brain injury in the temporoparietal region of the brain. A CT scan on 13/08/2007 demonstrated no hydrocephalus or acute haemorrhage but there was some herniation of the brain through the craniectomy site and despite decompression there remained relative asymmetry of the quadrigeminal cistern and suprasellar cistern.

A.N was readmitted to A&E 17/10/2007 11 weeks after his initial accident. A CT scan showed that A.N was suffering from traumatic hydrocephalus and acute haemorrhage as a complication of the former decompressive craniectomy and evacuation surgery. The scan showed a large cranial defect with brain tissue herniated through the cavity. There was a low density area in the parietal region suggesting an infarct together with dilation of the adjacent ventricle but no recurrent haemorrhage was demonstrated. Both lateral, third and fourth ventricles were somewhat dilated which suggests post traumatic hydrocephalus. A.N experienced left hemiplegia and therefore underwent a craniectomy to insert a Ventriculoperitoneal (VP) shunt via a tempo-parietal bone flap (18/10/2007). GCS at this point was 14/15. A.N also had a titanium cranioplasty where surgeons replaced the bone flap removed for the VP shunt.

A further CT scan on the 07/12/2007 demonstrated a large right fronto-temporal craniotomy with no significant midline shift. The ventricles were slightly prominent but there was an intraventricular shunt in the left lateral ventricle which is less dilated than the right side. There was no intracerebral haemorrhage nor obvious increased intracranial pressure nor evidence of recurring hydrocephalus.

After the surgery A.N experienced epileptic drifts and was started on anti-epilepsy medication including Epilim, Phenytoin and Sodium Valprate. The last epileptic seizure experienced by

A.N was in January 2008 and anti-epilepsy medications were withdrawn from September 2008 with no further epileptic seizures occurring.

### **Participant L.J.**

Participant L.J. is a 40 year old male who sustained his brain injury at the age of 19 as a result of a road traffic accident (RTA) (05/08/1993). A CT scan on the day of admission revealed that patient L.J. had a small area of petechial haemorrhage contiguous with the left caudate nucleus. A second small area of the haemorrhage was evident on slice 2 in a more anterior position in the left frontal lobe posteriorly. A follow up CT scan five days after the injury revealed further small petechial haemorrhages on slice 9 in the right frontal region anteriorly with no new major haemorrhage apparent. L.J. spent 14 days in a coma and had PTA for 2 months indicating a severe brain injury.

### **Participant W.Y.**

Participant W.Y. is a 53 year old male who sustained his brain injury at the age of 38 as a result of a fall which caused concussion and a subsequent frontal and bilateral subarachnoid haemorrhage (31/07/1999). He had emergency surgery for internal carotid artery aneurysms where one aneurysm was clipped and another one was coiled. After the surgery W.Y. then suffered a cerebrovascular accident which left him with homonymous hemianopia and residual left side weakness.

### **Participant S.G**

Participant S.G is a 54 year old male who sustained a severe TBI at the age of 24 years old after a RTA where he was a front seat passenger in a car (14/09/1985). Upon arrival to hospital S.G was unconscious and he spent several days in a semi-comatose state. Medical notes reported that S.G displayed PTA for six days after he sustained his injury and that he suffered

a severe parietal-temporal region injury but no scan data was included. S.G received no neurosurgery and remained in hospital for six weeks. S.G presented with a history of alcohol and substance abuse and was treated for depression after the injury. He also presented with parasomnia and post-concussion syndrome.

### **Participant N.W**

Participant N.W is a 43 year old male who sustained a TBI when he fell off a 20 foot scaffolding and landed on a concrete yard when he was 40 years old. Upon arrival to hospital on 08/10/2012 N.W presented with a GCS score of 3, no PTA score was documented but notes indicted that N.W was agitated and had to be tied down to his bed.

A C.T scan on 08/10/2012 revealed a bleed on the left hand side of the brain. There was herniation of brain content through defect and a large intraparenchymal haematoma superiorly in the left frontal lobe. Haemorrhagic bilateral frontal lobe contusions and right temporal lobe contusions were present as well as a decrease in mass effect with no midline shift. There was a linear fracture through the left parietal bone extending to the left lambdoid suture and a large left sided acute subdural haematoma, max depth 13mm, causing significant midline shift and effacement of the basal cisterns. Furthermore, there was associated subarachnoid haemorrhage and further small acute subdural haematoma in the parafalcine region in addition to some acute haemorrhage in the right frontal lobe in association with longstanding encephalomalacia in this region. There was also further encephalomalacia seen in right temporal region. No hydrocephalus was documented. The diagnosis was a severe TBI and with the CT scan indicating that there was a skull fracture and a large left subdural haemorrhage urgent neurosurgical opinion was advised. A cranioplasty was performed and 30-40% of the skull was removed as well as a left decompressive craniectomy, drainage of acute subdural haematoma and insertion of an intracranial pressure monitoring device (ICP). A reconstructive cranioplasty

following previous left fronto-parietal craniectomy was also performed which required a bone flap and N.W was required to wear a protective helmet. N.W was in an induced coma for ten days and received, in total, 96 days of professional medical care.

N.W had previously sustained a brain injury 10 years previously (aged 30) when he was hit over the head with a baseball bat. He was travelling abroad at the time and very limited medical notes are available. The notes imply that N.W may have endured a SDH and a fractured skull leading to a big blood clot on the back of the brain.

### **Participant M.M**

Participant M.M is a 33 year old male who sustained a TBI when he was 30 years of age during an epileptic seizure (10/08/2012). M.M has suffered chronic epileptic seizures from childhood. He sustained a temporal and subdural skull fracture during the fall and was diagnosed with an acute brain injury. He was admitted to hospital on 10/08/2012 at 11.30am and at 14.00 he had a GCS of 14/15. M.M was discharged on 13/08/2012. First viewed as mild TBI but low GOAT scores (14/08/2012: 94, 15/08/2012: 86, 16/08/2012: 73) suggests prolonged PTA.

A C.T scan on 10/08/2012 displayed minimal contusion in right temporal lobe and associated oedema throughout the posterior half of right cerebral hemisphere. There was a localised subarachnoid haemorrhage within right parietal region but no midline shift or hydrocephalus. A right temporal bone fracture breaching the mastoid air cells extending anteriorly to just above the right temporomandibular joint was present but there was no ossicular discontinuity within right ear. There was minimal blood within right mastoid air cells extending into the middle ear cavity. The fracture also appeared to extend into the roof of the right external auditory canal with the fractured skull and right temporal contusion complicated by reduced hearing. The right side also had lower motor neurone and there seemed to be damage to the right seventh nerve palsy.



A C.T scan on 16/08/2012 displayed an area in the posterior right temporal lobe approximately 25x25 mm containing a central high attenuation area. The area represents contusional brain trauma and had matured since the previous scan 10/08/2012. There was no sign of hydrocephalus, subdural haematoma or mass effect and the subarachnoid blood had resolved. Cerebella atrophy was unchanged from previous scan and presumably related to anti-epilepsy medication.

### **Participant D.J**

Participant D.J is a 31 year old male who sustained a TBI when he was intoxicated and assaulted on a night out. D.J was punched in the face resulting in him falling and hitting his head on the floor aged 29. He sustained the injury in the late hours on 30/06/2013 and was admitted to hospital with a GCS score of 9 and severe PTA (GOATS 9/100). D.J was intubated in A&E for a C.T scan and GCS increased to 13 but then dropped to 10 so he was rescanned but no changes were documented.

The CT scan revealed extradural blood and contusions. There were contusional injuries within the right frontal and temporal lobes with small foci of subarachnoid and intraparenchymal haemorrhage. Furthermore, there was diffuse effacement of sulci but no midline shift. A skull fracture extended from parietal bone into temporal bone and there was an account for contrecoup pattern of injury within right frontotemporal region. No other skull fractures were demonstrated from the scan.

A second C.T scan on 01/07/2013 displayed areas of haemorrhagic contusion within the right frontal and temporal lobes. There was a small amount of overlying subarachnoid blood but no significant mass effect present. A fracture on the left side of the skull involving the parietal and temporal bones was recorded. There was no evidence of haemorrhage elsewhere, no midline

shift and no change from the scan on 30/06/2013. D.J displayed an overall PTA and retrograde amnesia of six days.

### **Participant G.M**

Participant J.M is a 60 year old female who sustained a TBI when she was a front seat passenger in a RTA on 30/12/1987 aged 31. J.M was unconscious on the scene of the accident and was hospitalised for two weeks. PTA was documented as more than seven days which constitutes a severe TBI. No scans were performed during her stay in hospital. The consultant at the time made some observational notes stating that J.M displayed patchy PTA for four weeks and she seemed to have reasonable cognitive recovery by 20 months but she had developed fatigability and it seemed likely that she suffered minor concussive injury. He also suggested that she more than likely had significant frontal and temporal contusions with reactive brain swelling.

In 1991, J.M requested an MRI scan which revealed that the brain ventricles were a normal shape, size and disposition for her age. The scan revealed no evidence or abnormal white matter signal change to suggest PT gliosis following TBI. There was prominence of the Sylvain fissures suggesting a minor atrophy involving the tips of temporal lobes which were slightly more marked on the left hand side. Overall, the scan was normal with a suggestion of minor bilateral temporal lobe atrophy. A further scan was conducted on 04/09/2006 and revealed normal intracranial appearances with no sequela of a previous head injury in terms of generalised atrophy, hemosiderin deposits or focal encephalomalacia.

J.M has suffered with significant memory disorders since the accident and with the evidence from the MRI scans it was suggested that this may indicate a bilateral temporal lobe disorder. Moreover, J.M's dyspraxia symptoms might point to possible right posterior frontal lobe area damage and her dysexecutive symptoms suggest widespread bilateral frontal contusions.

However, it should be noted that MRI data does not really support these theories as the scans were relatively normal.

### **Participant J.F**

Participant J.F is a 63 year old male who sustained a TBI as a pedestrian in a RTA on 12/10/2011 aged 59. He received 37 days of acute care and was discharged on 18/11/2011. J.F was hit by a motorcycle going approximately 30mph. On admission, GCS score was 7, LOC approximately 5-10 minutes and PTA was documented for 14 days.

A C.T scan on 12/10/2011 displayed acute subdural blood overlaying the right parietal frontal lobes, extending into intracerebral sulcus with a maximum depth of 4mm. There was sulcal effacement underlying acute blood and slight midline shift to the left. The scan revealed extensive areas of acute intraparenchymal haemorrhage in the frontal lobes bilaterally and a fracture through the midline occipital bone extending from the foramen magnum 5.2cm superiorly. There was also a fracture through the right occipital condyle extending anteriorly through the medial petrous temporal bone and medial wall of right carotid canal. Injury may have extended to the right mastoid air cells which were partially opaque. There were multiple locules of intracerebral air adjacent to right petrous temporal bone in basal cisterns and complex compound occipital and base of skull fractures with possible associated contra-coup injury and acute subdural blood. As a result of the scan, urgent neuro-surgical opinion was advised and a right frontal craniotomy and insertion of intracranial pressure transducer was undertaken.

C.T scan on 19/10/2011 revealed extensive bi-frontal contusions which were more marked on the right than the left side of the brain. Compared to the earlier scan at time of admission, the follow-up scan displayed enlargement of contusions both in terms of haemorrhagic component and adjacent swelling with small amounts of contusional injury to the tip of the right temporal

pole. There was an increase in the amount of swelling associated with frontal contusions with slight increase in effacement of the frontal horns together with a mild shift of midline. Furthermore, there was a very thin subdural collection over the right hemisphere but this was not enlarged and not a major factor with respect to local mass effect. There were no new features but the appearance were those of maturing contusions in both frontal and right temporal lobes which were associated with increased swelling compared to on admission.

A further C.T scan on 21/10/2011 showed maturation of the contusions and mass effect and midline shift towards the left, particularly in anterior cranial fossa, had significantly increased. The basal cisterns were significantly effaced and there was a subdural collection seen on right side which had increased to a max depth of 6mm.

A C.T scan on 24/10/2011 displayed images of the craniotomy. There was progressive contusion and/or ischaemic change in the right frontal lobe with extensive left sided inferior contusional change and damage to anterior right temporal lobe. A midline occipital fracture was clearly demonstrated with a significant midline mass effect and subfalcine shift to the left. Moreover, the scan revealed a haemorrhagic transformation of the contusion and a mass particularly in right frontal lobe.

### **Participant M.O**

Participant M.O is a 47 year old male who sustained a closed TBI during a RTA when he fell off his motor bike on 08/07/2000 aged 31. From his medical notes there is reference to a CT scan which displayed multiple contusions and multiple subarachnoid hematomas with no midline shifts leading the medical team to document his brain injury as severe. There is reference to M.O's father who told neuro-rehabilitation staff that M.O was on a ventilator for six weeks. On 05/09/2000 M.O was still displaying signs of PTA, he was confused and confabulating. M.O was admitted to neuro-rehabilitation on 02/10/2000 and was discharged on

07/05/2001. M.O re-attended neuro-rehabilitation on 24/01/2011 and underwent a period of assessment for decreased mobility; he was discharged on 04/02/2011. A CT scan was conducted on 17/01/2014 which displayed no intra or extra axial haemorrhage. There was established low attenuation in the deep and subcortical white matter of both cerebral hemispheres mainly in parietal lobes but also in right frontal lobes. Given the young age of the patient the changes represent gliosis secondary to intracranial injury related to the RTA in 2000. Small vessel ischemic change of such degree would seem unlikely in someone this age. There was no acute territorial infraction on the scan.

### **Participant K.R**

Participant K.R is a 33 year old female who sustained a minor brain injury when she fell from a horse on 31/05/2006 aged 23. There was a loss of consciousness and her safety hat was fractured and she experienced a graze on her forehead. She had complete amnesia of the event when arriving in A&E, she had a bad headache at the back of her head, nausea and vomiting and scored 14 on the GCS. K.R's CT scan detected no abnormalities. PTA was documented for three to four days after the accident. In October 2007, K.R was still experiencing ongoing memory problems and displayed mild impairments in cognitive function. K.R had previously sustained a head injury at the age of 21 when she again fell off a horse but there are no medical notes available for this fall. K.R does recollect that she had PTA and was only in hospital for one day or maybe just over night. Her husband took two weeks off work to help K.R and to help look after their children during this previous fall. K.R returned to work four weeks after the original injury. An MRI scan was conducted which came back as entirely normal with normal intracranial appearances and no evidence of previous intracranial trauma. The hippocampi and temporal lobes were normal and there was no hemosiderin deposition. The collaborating clinical consultant recommended that K.R cognitive and behavioural presentation

indicated pathology which did not encroach on either frontal lobes, or occipital cortex, but may be present in other cortical or subcortical brain regions.

### **Participant A.H**

Participant A.H is a 60 year old male who sustained a closed TBI following a fall down stairs when a step gave way on 13/08/2013 aged 57. The fall caused a massive extradural bilateral traumatic subarachnoid haemorrhage which needed evacuation via brain surgery. A.H had no memory or recollection of how the accident occurred, what happened during or immediately after the accident and the first thing he can remember is waking up on a ward in the hospital. A.H complained of a constant headache in the left-hand side of his head and displayed memory impairments with PTA of up to two weeks. A.H was admitted to Magnolia neuro-rehabilitation on 03/09/2013 where the notes indicated that he sustained a bilateral small SDH which was treated conservatively and needed intracranial pressure monitoring. A second minor head injury was sustained in 2015 when he was intoxicated and fell in the street. GCS was 15 at the scene (E= 4, V= 5, M = 6) but A.H refused medical treatment and no further medical details were available.

### **Participant J.M**

Participant J.M is a 53 year old male who sustained a traumatic subarachnoid haemorrhage after a fall on 14/05/2014. He was admitted to A&E, scanned and then transferred to a different hospital as an urgent case. A craniotomy was conducted for a subarachnoid haemorrhage.

### **Participant M.W**

Participant M.W is a male participant who sustained a severe TBI on 12/03/2015 at the age of 58 when he fell from his push bike without wearing a helmet. On admission, M.W had a GCS rating of 8. A CT scan on arrival to hospital displayed subdural blood in the left hemisphere,

DAI, severe extensive damage in the left temporal lobe and subarachnoid blood in the left temporal area extending in the lesser degree to the frontal lobe. There was an acute subdural haemorrhage overlying the left cerebral hemispheres extending into the left tentorial margin measuring 1cm at maximum thickness. In addition, there were concussion injuries in the left temporal pole with intraparenchymal haemorrhage. There was mass effect with sulcal effacement and slight midline shift of approximately 5mm and a displaced right parietal fracture extending inferiorly into the squamous-temporal region and furthermore into the skull base near to the carotid canal on the right side. This was a complex fracture with a further transverse component but no obvious bony injury involving the carotid canal. Overlying this fracture was marked soft tissue haematoma in the scalp and there was a small amount of blood adjacent to the fracture in the posterior temporal fossa. There was fluid seen in the sphenoid sinus and the ethmoid air cells with possible un-displaced fractures of the sphenoid posterolaterally on the right side and the right zygomatic arc.

On 14/03/2015 intracranial pressure was initially controlled but then increased leading to a bulging decompressive craniectomy. A CT scan on 16/03/2015 was conducted and a direct comparison was made with the scan on 14/03/2015 after the decompressive craniectomy for the post traumatic left temporal intracerebral haemorrhage and the extra axial haematoma. There was persistent mild mass effect in the left hemisphere with effacement of the left lateral ventricle and very mild subfalcine shift to the right. There was also bulging to the left hemisphere through the craniectomy site, particularly at its superior and temporal regions.

A CT scan on 26/03/2015 was conducted for a left sided decompressive craniectomy. The contusional haemorrhage within the left temporal lobe continued to show evidence of evolution. The degree of cerebral swelling was reducing and the convexity sulci were more clearly visualised. There was some mild herniation of the left cerebral hemisphere through the craniectomy defect and the small subdural haematoma overlying the left frontal lobe was

reduced in size and the free intracranial had been resorbed. The volume of visible subarachnoid haemorrhage within the high right convexity sulci had reduced and no hydrocephalus was present. Grey and white matter differentiation was preserved with no evidence of hypoxic brain injury on CT imaging. Blood was visible within the sphenoid sinus and there was fluid filling both middle ear clefts and the petrous mastoid air cells. A complex base of skull fracture and minimally displaced right squamous-temporal bone fracture extending into the right parietal bone were diagnosed.

A later CT scan on 23/04/2015 with a volumetric study was performed prior to manufacture of the cranioplasty after the craniectomy. The scan displayed some modest herniation of the brain which was reasonably large and overlying the CSF collection that expands the soft tissues of the scalp. There was no hydrocephalus or mass effect present. M.W was discharged from hospital on 29/05/2015.

On 13/08/2015 there was a very brief mention of a cerebral abscess in the medical notes but no other notes were available. A CT scan on 21/10/2015 revealed extreme brain atrophy on the left lobe but that prominent sulcal patterns suggested global rather than focal atrophy. The ventricles of the brain were enlarged, not due to blockages in circulation, but because they had expanded to fill the space left by the atrophy.

### **Participant A.A**

A.A is a 47 year old female who was admitted to A&E on 03/01/2015 after a fall down the stairs resulting in a closed head injury. The fall is thought to have occurred after pulmonary embolus but it was unclear if the fall was mechanical or a syncopal episode. A.A presented with shortness of breath and dizziness. On 04/01/2015 GCS suddenly dropped to 5 (E= 1, V= 1, M= 3) and A.A was intubated and ventilated. A CT scan revealed large acute fronto-parietal SDH measuring 1.6 cm in max depth with significant shift and mass effect. There was right



frontal lobe contusion and intra-parenchymal haemorrhage measuring 1.9 cm with surrounding oedema. Extension of bleed went into the subarachnoid spaces; a 9 mm pericapsular intraparenchymal bleed was also noted on the left. There was significant mass effect with effacement of right lateral ventricle and midline shift to the left with early subfalcine herniation. There was also prominence of the temporal horns of the left lateral ventricle suggesting evolving hydrocephalus. There was a right sided acute subdural haematoma with mass effect and midline shift and uncal herniation. The patient was transferred to a specialist neuro-hospital where she underwent surgical evacuation of the haematoma. A right sided craniotomy for evacuation of acute SDH and insertion of ICP monitor. Surgery included the patient being in a supine position with the head resting on a horse shoe head rest turned to the left. A trauma flap incision was made on the right hand side and burr holes for a craniotomy and bone flap elevated for acute subdural haematoma to be evacuated. There was an active bleed from breaching veins to the dura which could be visualised and had coagulated. There was a washout and haemostasis using SurgiSeal and flow seal and the dura was left opened. An ICP was inserted in the subdural and a bone flap was fixed using Crani fix. A gravity drain was also inserted and secured and closure was attained using vicryl and clips to the skin with a silk for drain and ICP 7. A.A was admitted to the ITU ward with a tracheotomy, NG tube and PICC line. A repeat head CT scan on 16/01/2015 displayed a decrease in the haematoma size and the patient was discharged from ITU where she continued neurological physiotherapy.

On 02/02/2015 A.A developed an intracranial bleed. A MRI head scan displayed previous right sided craniotomy and evacuation of haematoma. Old blood products in the right frontal lobe and prominent perivascular space on the left side in basal ganglia region. No new abnormality but some fluid opacification in right mastoid air cells. Intermittent papillary dilation would be a very unusual sign of intracranial haemorrhage or tumour. Haemorrhage or tumour causing unilateral papillary dilation would have to be within the brain stem or causing compression of

the brain stem. Neither of these pathologies would be likely to cause an intermittent clinical sign. Two days later medical checks revealed a GCS of 14 and although the patient was alert and could follow simple commands she was confused. She was able to explain she was in hospital but she was unsure which one. She was able to detail what month it was but was unaware of the day, date or year. A CT scan on 06/02/2015 revealed a possible infract in the left internal capsule accounting for right hemiparesis in addition to clear contusional SDH.

On 10/04/2016 A.A experienced traumatic optic neuropathy on the right side. This led to new temporal field loss which excluded sellar lesion or aneurysm. A comparison was made with previous scan dated 25/02/2015 and it revealed evidence of previous trauma and neuro-surgery. There were areas of gliosis and old blood products were seen in the right frontal lobe. There was also high T2 signal in the right thalamus and right medial temporal lobe and in the right matter of the right corona radiata and occipital lobe which were consistent with injury due to the intracranial mass effect previously experienced. The sella pituitary and anterior optic pathways were intact. No vascular lesions were visible and no new abnormality was present when compared to the previous scan. It was possible that temporal field loss was related to the original intracranial mass effect and subsequent white matter degeneration in the right hemisphere.

### **Participant I.E**

Participant I.E is a 39 year old male who sustained a SDH following a fall during an assault on New Year's Eve in 2012 where he fell backwards and hit his head on the concrete path. I.E presented with a GCS of 8 (E= 1, V= 1, M= 6). I.E's pupils were equal and reactive to light but he displayed left hemiplegia with GCS dropping to 3 which resulted in sedation and ventilation. A CT scan was conducted and a small right acute subdural haematoma with midline shift was reported. This was initially managed conservatively with ICP but this continued to rise despite

the best medical management and the decision was made to operate. The patient was operated on using a left lateral position on a bean bag and in-line immobilisation with the head on a horse shoe head rest. A large question mark incision was made in the skull and a whole bone flap was elevated revealing a very oozy haemostasis. A bone flap was lifted after four burr holes were joined with craniotomy. Again, this was very oozy. The dura was opened in cruciate fashion and an acute SDH was evacuated under high pressure. There was brain herniation and haemostasis, hitch stitches, surgical patties and floseal required as oozing from various points. A neuropatch was placed over the dural flaps and some surgical tape over the area around key burr holes. A gravity drain was inserted and closure was completed in layers. Post-operatively, I.E developed staphylococcus aureus pneumonia and a haemophilus infection. After the injury, I.E experienced on-going headaches and weakness in the left had side of his body.

### **Participant P.P**

Participant P.P is a 50 year old male who sustained a closed head injury which may have occurred after falling off some ladders on 28/01/2011, although this is not confirmed in the medical notes. He was treated in hospital after the accident and there was a brief medical extract stating a severe frontal brain injury with a subdural haematoma and spinal damage. P.P received neuro-rehabilitation after the injury but no further medical details could be found.

### **Participant A.D**

Participant A.D is a 28-year-old male who is thought to have sustained a brain injury after an assault. The clinical consultant was able to document that A.D sustained a severe frontal brain injury and he was in a coma for three weeks. No other medical notes could be located.

