# Adults imitate to send a social signal

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I, Sujatha Krishnan-Barman, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signed

Sujatha Krishnan-Barman

Date: 28 April 2021

உருவாய் அருவாய், உளதாய் இலதாய்
மருவாய் மலராய், மணியாய் ஒளியாய்க்
கருவாய் உயிராய்க், கதியாய் விதியாய்க்
குருவாய் வருவாய், அருள்வாய் குகனே.

## Abstract

Humans are prolific imitators, even when copying may not be efficient. A variety of explanations have been advanced for this phenomenon, including that it is a side-effect of learning, that it arises from a lack of understanding of causality, to imitation being a mechanism to boost affiliation. This thesis systematically outlines the hypothesis that imitation is a social signal sent between interacting partners, which rests on testing whether our propensity to imitate is modulated by the social availability of the interaction partner (i.e., whether our interaction partner is watching us or not). I developed a dyadic block-moving paradigm that allowed us to test this hypothesis in a naturalistic manner in four behavioural and neuroimaging studies using functional near-infrared spectroscopy (fNIRS). I found that imitative fidelity was modulated by whether the interaction partner was watching the participant make their move or not, and this effect replicated across all four studies, in both neurotypicals and autistic participants. I also examined the neural correlates of responding to irrational actions, and of being watched. I found that being watched led to a robust deactivation in the right parietal cortex across both neurotypicals (in two studies) and autistic participants (one study). Among autistic participants we also found strong engagement in the left superior temporal sulcus (STS) when being watched. For responding to irrational actions, in one study of neurotypicals we found greater deactivation in the right superior parietal lobule (SPL) when making more irrational responses. In another study of autistic and neurotypical participants we found deactivation in the bilateral inferior parietal cortex (IPL) in neurotypicals when responding to irrational actions, while this deactivation appeared confined to the left IPL for autistic participants. Autistic participants also showed differentially higher engagement in the left occipitotemporal regions when responding to irrational actions. This thesis supports the social-signalling hypothesis of imitation and is accompanied by suggestions for future directions to explore this theory in more detail.

## Impact statement

We copy others extensively, from very early in our development, and copying is thought to help us learn as well as connect with others. There has been long-standing debate on the purpose or reasons behind copying irrational actions, and whether this is indeed a feature or a bug in human behaviour. Copying behaviour among autistic individuals has also been a hotly debated topic, with mixed evidence on the prevalence and extent of imitative deficits in that population.

Investigating a potential socially useful explanation for copying behaviour and exploring the differences in how this behaviour is manifested in autistic and neurotypical individuals could help us understand both how neurotypical brains work, and whether there are differences in those with autism, particularly if they can help explain the social challenges associated with autism.

Studying naturalistic behaviours such as copying in a social context is challenging, and the paradigm developed in this thesis attempted to bridge the gap between the real world and the laboratory by using a simple block-moving task that people are likely to be familiar with and deploying subtle rational and irrational features such as the demonstrated trajectories. Moving beyond traditional neuroimaging modalities such as functional magnetic resonance imaging (fMRI), I used a relatively novel wearable neuroimaging technique (fNIRS) to allow us to capture cortical activation while subjects were able to move and interact in a relatively natural fashion.

This thesis reported three main findings. First, the fidelity with which participants imitated their interaction partner was related to the social availability of the interaction partner, specifically whether the interaction partner was watching the participant or not. This was true for both neurotypicals and those with autism. Second, we found a robust deactivation in the right parietal cortex when participants were being watched by their interaction partner when compared with trials where they were not being watched. This effect was found in both neurotypicals (across two studies) and those with autism (one study). However, we also found that autistic participants showed strong activation in left STS when being watched. Third, we found deactivation of the bilateral IPL in neurotypicals when responding to irrational actions; among autistic participants we found deactivation only in the left IPL, and we also found greater engagement in the left occipitotemporal regions among this group. Together these findings support the view that imitation can function as a social signal. Also, these findings suggest that imitative deficits in autism are not universal, and that in many circumstances people with autism

may behave the same as neurotypicals, despite engaging different brain regions when doing so.

These findings make a significant contribution to the study of imitation in a social context, enabling us to pursue the view that imitation is not a side-effect or epiphenomenon, but may in fact serve a valuable social goal in dyadic interactions. This thesis also advances our knowledge of social cognition in autism showcasing that behaviourally people with autism can and do match neurotypicals in some cases even when the underlying brain mechanisms utilised may be different. In the longer term this will enable us to better understand autism as well.

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## List of Abbreviations

| Term | Definition |
|---|---|
| ADHD | Attention-Deficit Hyperactivity Disorder |
| ANOVA | Analysis of Variance |
| AQ | Autism Spectrum Quotient |
| ASC | Autism Spectrum Condition |
| ASD | Autism Spectrum Disorder |
| CBSI | Correlation-Based Signal Improvement Method |
| dlPFC | Dorsolateral Prefrontal Cortex |
| DSM-5 | Diagnostic and Statistical Manual |
| EEG | Electroencephalography |
| EP-M | Emulation, Planning and Mimicry Model |
| fMRI | Functional Magnetic Resonance Imaging |
| fNIRS | Functional Near-Infrared Spectroscopy |
| GLM | General Linear Model |
| HbO | Oxygenated Haemoglobin |
| HbR | Deoxygenated Haemoglobin |
| IFG | Inferior Frontal Gyrus |
| IPL | Inferior Parietal Lobule |
| MEG | Magnetoencephalography |
| MNI | Montreal Neurological Institute |
| MNS | Mirror Neuron System |
| mPFC | Medial Prefrontal Cortex |
| MTG | Middle Temporal Gyrus |
| NT | Neurotypical |
| PET | Positron Emission Tomography |
| SPL | Superior Parietal Lobule |
| STORM | Social Top-Down Response Modulation Model |
| STS | Superior Temporal Sulcus |
| tDCS | Transcranial Direct-Current Stimulation |
| TMS | Transcranial Magnetic Stimulation |
| TPJ | Temporal-Parietal Junction |
| WHO | World Health Organisation |
| WTC | Wavelet Transform Coherence |

## Publications

Krishnan-Barman, S., Hakim, U., Smith, M., Pinti, P., & Hamilton, A. F. de C. (in prep). Imitation as a social signal in autism.

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## Presentations

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# Chapter 1. Introduction

This introductory chapter sets out the definition of imitation and introduces the social-signalling hypothesis of imitation which is rigorously tested in this thesis. We examine existing explanations for imitation and review what we currently know about the brain mechanisms underpinning imitation, before finally reviewing the study of imitation through deficits in imitative abilities among those with autism.

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## 1.1. Imitation

On an unseasonably warm spring day in London my pre-schooler wants to have another go on the swing in our garden. Before climbing on she picks up a dust cloth hanging on the side of the swing's frame and carefully wipes the seat down. I had done the same earlier in the day to get rid of the cobwebs that had accumulated. There are no cobwebs now, but the desire to do exactly as mummy does remains strong. Broadening our horizons from my personal sample size of one, we know that imitation is a ubiquitous, and often uniquely human phenomenon. While there is growing evidence in recent decades that non-human primates imitate too (Whiten & van Schaik, 2007), the breadth of human imitation is far greater (Gergely & Csibra, 2020).

Imitation encompasses a wide range of behaviours, from the very simple "monkey-see monkey-do" formulation involving just mirroring a basic motor movement, to more complex copying of norms ("when in Rome, do as Romans do") that incorporate social context and learning. It has been studied for centuries, starting from ancient theories of *mimesis* (the Greek word for imitation) put forward by Plato, to more contemporary accounts. Yet, we still do not fully understand what purpose imitation serves, or how it is instantiated and modulated in the brain.

Before exploring the theoretical accounts of imitation, it is useful to define the term explicitly. While this may seem trivial, debates over what constitutes imitation have occupied scientists for decades (Hamilton, 2015; Heyes, 2021). In the simplest terms, imitation refers to copying or reproducing the actions of another individual (Heyes, 2011). However, there is ambiguity on whether this involves the end goal, the means by which it is achieved, or both, and whether or not this should include action features that are clearly identifiable as being irrational or irrelevant to the goal. In line with the taxonomy outlined by Whiten and colleagues (2004) we define ***true imitation*** as copying both the end of a goal-directed action and the means used to achieve them. ***Emulation***, in contrast, involves copying only the end state and does not require employing the same means (Tomasello, 1990). ***Mimicry***, or automatic imitation, involves the unintentional copying of means or features of an action, whether goal-directed or not (Chartrand & Bargh, 1999; Heyes, 2011)[1]. Finally, ***overimitation*** involves copying the irrelevant features of an action

[1] While both mimicry and automatic imitation involve the involuntary copying of action features or means, automatic imitation is used to describe behaviours in a laboratory such as the stimulus response compatibility effect where observing an action makes it easier to perform it; mimicry in contrast is usually used to refer to naturalistic behaviours.

sequence even if they are not helpful to reaching the goal (Hamilton, 2015; Horner & Whiten, 2005). An overview of this taxonomy is presented below in Table 1.1.

**Table 1.1. A taxonomy of different types of imitation**

| Type of copying | Definition | Example |
|---|---|---|
| True imitation | Reproducing both the end goal as well as the means of a goal-directed action | A student learning to play the piano watches an instructor and presses the keys in the same sequence using the same fingers as the instructor |
| Emulation | Copying only the end state of a goal-directed action, without necessarily copying the means | A child watches an adult who has their hands full use their elbow to press a switch, but the child uses her hands to press the switch |
| Mimicry / automatic imitation | Copying the means or features of an action | Two friends who are chatting unconsciously cross their legs in the same manner |
| Overimitation | Copying the irrelevant features of a goal-directed action sequence | A child learning to serve from a tennis instructor carefully watches the instructor bounce the ball three times before throwing it up to serve; they replicate the entire action sequence including three bounces before serving |

Reflecting on the varied definitions of imitation, we can see why there continues to be enduring debate on the reasons why we imitate, and the processes underpinning it. In this thesis we are specifically interested in whether we copy the irrelevant or irrational features of a goal-directed action sequence—i.e., overimitation.

In this chapter we will first review theories of why we imitate. Second, we will outline our theory of imitation as a social signal, what this means, and sketch out some hypotheses that will be tested in this thesis. Third, we review what we know so far about the brain mechanisms of imitation. Fourth, we will examine how imitation is studied via imitative impairments in autism, and how this has contributed to our understanding of the phenomenon. Finally, we will present an overview of the structure of the thesis.

## 1.2. Theories of why we imitate

Biologists in the 19th century suggested that imitation was a mechanism that facilitated the transfer of various behaviours in a species across generations, with the young learning from the old via copying (Darwin, 1871; Romanes, 1884; Wallace, 1870). In humans, the

various explanations advanced as to why we imitate broadly centre on imitation as a way to learn, and as a way to communicate "mutuality" (Uzgiris, 1981). In this section we review what we know so far about why we imitate.

### *1.2.1. Imitation emerges early in humans…but how early?*

Developmental studies have typically suggested that infants begin imitation very early although the precise age at which imitation begins has been disputed. The first formal study of imitation in very young infants (Meltzoff & Moore, 1977) reported evidence of imitation in children as young as two and three weeks old, which has since been replicated (Meltzoff, 2005). However, other studies conducted on infants aged 9-30 days (McKenzie & Over, 1983) and one conducted on infants aged 4-21 weeks (Abravanel & Sigafoos, 1984) failed to find evidence of imitation in very young children. A review of 35 studies of neonates suggested that only tongue protrusion was reliably matched by neonates (Anisfeld, 1996). In very young infants, at least, there remains controversy on whether this is true imitation or the behavioural matching that arises from arousal or some other explanation (S. S. Jones, 2009). A large longitudinal study (N = 64 infants) of neonates aged 1-9 weeks found no evidence that infants copied any of the nine social gestures that were displayed to them; this suggests that imitation may not be an inbuilt feature that children are born with, but may emerge later in development (Oostenbroek et al., 2016). Nevertheless, when we turn to slightly older children, we find stronger evidence of clear imitation. Seminal work by Meltzoff (1995) showed that children who were 18 months old were able to re-enact what adults demonstrated. Meanwhile Barr and colleagues (1996) showed that children aged 12 months and older showed evidence of imitation when tested after a delay (of a day), while children as young as six months imitated when tested immediately after an action was demonstrated.

This distinction between whether imitation emerges from birth or a few months later may seem trivial, but it seeks to answer an important question: is imitation an innate genetic mechanism or is it a skill that emerges postnatally via associative learning? Farmer and colleagues (2018) characterise this as the nature-nurture divide in theory. The origins of imitation are closely linked to the existence of mirror neurons[2] which fire both when an action is observed and when it is performed (Rizzolotti & Craighero, 2004). Mirror neurons are thought to be central to imitation, and the origins of mirror neurons have been hotly debated for decades. At one end of the spectrum is the view that we have a

[2] Reviewed in detail in Section 1.4 below

"genetically predetermined" propensity to develop mirror neurons (Gallese et al., 2009), and this is thought to be reflected in the propensity of neonates to imitate. At the other end of the spectrum, there has been growing evidence that mirror neurons develop postnatally via associative learning in response to the rich sensorimotor environment surrounding neonates; here it is argued that mirror neurons acquire their matching properties via domain-general learning processes, similar to the mechanisms that produce Pavlovian conditioning (Cook et al., 2014; Heyes, 2001; Heyes & Catmur, 2021). A novel study implemented "counter-mirror" training and showed that it led to the reversal of mirror responses; in this study adult participants moved their index finger while viewing little finger movements and vice versa, and later showed greater activation of the index finger muscle when observing little finger movement than when viewing the same finger movement (Catmur et al., 2011). Studies on seven-month-old infants also showed that their mirror responses to certain movements increased with the amount of experience they had in making those movements during an earlier training session (de Klerk et al., 2015). When it comes to imitation as well, the most recent evidence from Oostenbroek and colleagues (2016) appears to suggest that imitation arises after birth, further supporting the associative learning account. This goes some way towards answering the question of how imitation arises in humans; in the next subsection we review existing explanations of what purpose it may serve. In line with the focus of this thesis, we are specifically interested in what drives the copying of irrelevant features of a goal-directed action sequence, which can be considered inefficient.

### *1.2.2. Imitation as an efficient way to learn*

Studying imitation in children has been a productive way to explore theories of why we imitate. Tomasello (1999) suggests that imitation serves a clear evolutionary purpose, allowing children to learn without needing to make potentially dangerous mistakes.. Along similar lines, it has been argued that imitation allows humans to transmit culture across generations (Boyd et al., 2011; Tennie et al., 2009). Here, it has been argued that overimitation, which involves copying even the irrelevant features of an action, may be evolutionarily efficient. One important paradigm that has been used to study imitation involves the use of an "artificial fruit" (Whiten et al., 1996), a Perspex box containing an edible item at its core; the fruit can typically be opened in one of two ways (using a bolt latch or a barrel latch for example), and a demonstrator would showcase one method often alongside some irrelevant action such as stroking the box to test which actions were copied by the subjects (see Section 2.2.1). Using a version of this paradigm Whiten and colleagues (2009) suggest that children adopt a "copy-all, refine-later" strategy that

enables them to acquire vast amounts of cultural knowledge quickly. This ***causal-confusion account of imitation*** essentially suggests that we imitate because we are confused about the exact features of an action sequence that contribute causally to achieving the goal. While this explains high-fidelity copying among young children, three strands of evidence stand counter to this view, which we review in detail below.

First, a number of studies have shown that that neurotypical children are able to identify the features of an action as "silly" and yet persist in overimitating them (Lyons et al., 2007; Marsh et al., 2013). We should note that Lyons (2007) argues that the tendency of children to encode all of an adult's actions as causally-relevant is so strong that it overrides countervailing task demands, time pressures, and explicit warnings to not copy the irrelevant actions. Regardless, we can conclude that overimitation does not extinguish with greater knowledge of the causal mechanisms driving an action sequence.

Second, studies have also shown that overimitation does not disappear as development proceeds; indeed, several studies have shown that even older children overimitate. Horner and Whiten (2005) showed that children, unlike chimpanzees, copied causally-irrelevant actions with high fidelity. This finding was replicated with three-year olds (McGuigan et al., 2007), and this same study showed that the propensity to overimitate *increased* when the study was performed with five-year olds; this suggests that overimitation is not merely a side-effect of immature cognitive capabilities. There is growing evidence that adults overimitate as well (Flynn & Smith, 2012; Whiten et al., 2016). Flynn and Smith (2012) also showed that overimitation does not diminish in the face of time pressure or the inclusion of a monetary reward for performing the most efficient action (and discarding the irrational features of the action sequence). The only situation in their experiment where overimitation is reduced was when the task was demonstrated by a naïve co-participant, suggesting that social context modulates overimitation.

Finally, turning to the question of selectivity in overimitation: an influential study by Meltzoff (Meltzoff, 1988) showed 14-month-old infants a novel action involving a demonstrator sitting at a table and bending forward from the waist to touch a panel in front of them with their forehead; pressing the panel illuminated a light bulb. When tested a week later, infants who were shown the irrational action showed a high propensity to imitate it. However, a subsequent study (Gergely et al., 2002) showed that infants can be selective: in this study the authors showed that 14-month old infants only copied the irrational action if it was demonstrated by a demonstrator whose hands were

free to press the panel, but they still chose to use their forehead. In cases where the demonstrator's hands were constrained in a sheet and they used their forehead, the children themselves used their hands to press the panel. This suggests that even very young children can be highly selective, choosing either a rational or irrational response based on context.

In contrast to the causal-confusion account, the ***rational-normative account of imitation*** instead suggests that the causally-irrelevant actions are viewed as part of the normative conventional whole (Keupp et al., 2013, 2015). Let us take a scenario where participants view an action sequence of say, a causally relevant action (A) followed by a causally irrelevant action (B) which brings about an effect (E); according to this account , participants who overimitate A+B = E view the task not as "bring about E", but as "perform A and B in order to bring about E". In an interesting study, Keupp et al (2015) showed children an action sequence known as "daxing" which involved both relevant and irrelevant actions. The children were then told to either "bring about E" or "dax" (both of which had the same end goal of E). When children were asked to "dax", they copied with high fidelity, whereas when the task context was changed, a significant portion of children did not overimitate. However, while this offers an account of specific situations where there is no causal confusion but perhaps there is opacity about the exact end-goal, it does not allow for a more generalisable theory of overimitation.

### *1.2.3. Imitation serves an affiliative purpose*

An alternate strand of explanation of imitative behaviour centres on communicating mutuality (Uzgiris, 1981), often termed ***the affiliative account of imitation***. In this account, people may be aware that an action is irrelevant, and not see it as necessary (or part of the normative whole), but nevertheless copy it for some affiliative purpose. Under this view we can conceive of imitation as a social signal that is sent to achieve some social aim. If this were true, then we should show that imitation is modulated by social context, and that being imitated has some positive social effect. We use the term social throughout this thesis to refer to thought, feeling or behaviour of an individual that is influenced by the actual, imagined or implied presence of others (Allport, 1954). In this section we first review the evidence on whether imitation is socially-modulated before examining what we know about the effects of being copied. The affiliative account of imitation accounts for the flexibility seen in empirical studies of overimitation, where children appear to choose to overimitate (or not) based on a variety of socially motivated factors such as the presence of a demonstrator, whether the demonstration involved a video or a live person,

and whether the participant was being watched. A study by DiYanni, Nini and Rheel (2011) involved an adult model on video rejecting a more efficient tool for a task in favour of a less efficient tool; children who watched these videos were more likely to subsequently copy the model's choice when the model was present when the children were making their choice than when the model was not present during the child's turn. A similar result was found by Nielsen and Blank (2011) in a study involving pre-schoolers who watched two adult demonstrators show them how to get a toy out of an apparatus; one adult used only causally-relevant actions while the other included irrelevant actions in her demonstration. Subsequently, one of the adults remained in the room while the other left, and it was the child's turn. They found that children copied the irrelevant actions in their turn but only when the adult who demonstrated the irrelevant actions was the one remaining in the room with them.

With regards to how the demonstration is delivered, studies have spanned from using video demonstrations, to humanoid robots, and live demonstrations from humans. The research suggests that the degree of imitation is modulated by the nature of the demonstration: Marsh, Ropar and Hamilton (2014) found that children overimitated irrational actions to a greater extent when watching a live demonstrator than when watching a video demonstration, and this effect was more pronounced among older children. This is similar to a result found among younger children (24-month olds) in a study that showed that children were likely to copy live models, or models they could communicate with via CCTV, to a greater extent than videotaped models who could not interact with them (Nielsen et al., 2008). Finally, it has also been shown that children are likely to copy humanoid robots but to a lesser degree than human demonstrators (Sommer et al., 2020). In contrast to these results, however, a study by Lyons (2007) showed that children overimitate even when the demonstrator is absent. To explain this apparent contradiction we consider a newer study by Marsh, Ropar and Hamilton (2019) which showed that children overimitate when the demonstrator watched them *and* when the demonstrator left the room, but not when she turned away from them in the same room. This suggests that perhaps in an experimental context, the demonstrator exiting the room still leaves open the possibility that we may be being watched from outside, while clear disengagement from a demonstrator in the same room reduces the propensity to overimitate. Taken together these studies suggest that the extent to which an intended interaction partner is available to view or interpret our actions influences the extent to which we imitate. In this thesis we term this availability as specifically the ***social availability*** of the interaction partner.

This social availability of the interaction partner in this thesis specifically refers to whether the interaction partner is watching the participant. Having reviewed studies on the presence or absence of a demonstrator when it comes to children, we now turn to studies of adults where the effect of eye contact and gaze can be specifically studied. In an innovative study, Bavelas and colleagues (1986) showed that the extent to which participants winced when an apparent victim was injured was related to whether the victim was looking at the participant at the time of the injury, what they term the ***visual availability*** of the victim. A more recent study has also shown that eye contact modulated the mimicry of rapid hand actions (Wang, Newport, et al., 2011). This effect of being watched has also been shown to persist across cultures: a recent study testing children between the ages of three and eight from two rural populations in Namibia and an urban German population found that across cultures children tended to imitate adult actions to a greater extent when the adult model was present rather than absent (Stengelin et al., 2019).

In this section we have focused mainly on whether an interaction partner is available to receive an imitative signal or not. There are other social factors that may also influence the extent of imitation, such as group membership; however the evidence here is considerably more mixed (see Marsh et al., 2016 for a review of these effects). As we have noted above, one study showed that the extent of overimitation reduced when the demonstrator was replaced by a naïve co-participant (Flynn & Smith, 2012). Other studies have showed that we tend to imitate those we share group membership, political views, or interests with to a greater extent (Bourgeois & Hess, 2008; Yabar et al., 2006); however a follow-up experiment by Yabar and colleagues (2006) failed to replicate their initial finding that group membership boosted imitation. When it comes to race, the evidence has been similarly mixed with studies showing no effect (Bourgeois & Hess, 2008), or showing that some races are more likely to demonstrate an increase in imitation of same-race members than others (Mondillon et al., 2007). Other studies have also shown that a demonstrator's race modulated the brain activity seen in a participant during an imitation task (Losin et al., 2012). As Marsh and colleagues (2016) note, frequently in the literature the same mechanism of affiliative intent is used to explain effects that run in opposite directions: i.e., we want to affiliate more with outgroup members and therefore copy them more; or we are compelled to affiliate more with ingroup members and therefore copy them more. Multiple social variables may also throw up interaction effects: one study showed that we copy ingroup members with greater fidelity than outgroup members in a cooperative context, but this effect disappears in a competitive context (Gleibs et al., 2016). While these dynamics are interesting, the evidence thus far does not

lend itself to robust theory-building. In this thesis, we are focused only on the extent to which an interaction partner is available to view the imitative behaviour, and not on the characteristics of the partner themselves.

Thus far in this section we have reviewed evidence of why individuals imitate others. We now turn to the question of what being copied does to the other partner in an interaction. When Alice copies Betty, what effect does it have on Betty, and what benefits accrue to Alice as a result? At a very basic level, being imitated has been shown to foster faster motoric responses (Pfister et al., 2013). We are more interested in the social outcomes, and here a seminal study by Chartrand and Bargh (1999) showing that interacting with a confederate who mimicked one's posture and movements increased the liking between interaction partners; this study also showed that the effect ran both ways—we feel more affiliation to people we copy, and we feel more affiliation to others who copy us. Other studies have built on this to show that participants who are given an affiliation goal increase their propensity to mimic their partner (Lakin & Chartrand, 2003). This has been termed the chameleon effect, and under this theory imitation is conceptualised as a ***social glue*** that increases our social advantage (Lakin et al., 2003; Wang & Hamilton, 2012).

In addition to increasing affiliation, researchers have also found other downstream consequences of being mimicked (Chartrand & Lakin, 2013). When interacting with someone who copies us with greater fidelity, we view them as more knowledgeable (van Swol, 2003); we also enjoy a salesperson's product more if they mimic us (Tanner et al., 2008). Being mimicked also led customers to leave larger tips for waitstaff (van Baaren et al., 2003), to people being more likely to make charitable donations (Stel et al., 2008) or to provide money to a stranded stranger (Fischer-Lokou et al., 2011). People who were mimicked were also more likely to be helpful, by picking up a pen for someone (van Baaren et al., 2004) or even engage in more costly helpful behaviour such as accompanying someone on a 15-20 minute walk (Müller et al., 2012). This effect is not limited to humans: Suzuki and colleagues (2003) found that we liked an animated character to a greater extent when it mimicked the prosodic features of our own voice; Bailenson and Yee (2005) similarly showed that we liked virtual characters who imitated our head movements to a greater extent than those who showed no mimicry[3]. This effect is also not limited to adults, with even very young children (18-

[3] We should note that Hale and Hamilton (2016b) found that being mimicked by a virtual avatar did not always increase rapport or trust, finding instead a null effect in a pre-registered study.

month olds) being more likely to help adults who had mimicked them (Carpenter et al., 2013). In contrast, encountering an interaction partner who exhibits very low levels of mimicry has been seen to increase cortisol levels, which are associated with increased stress (Kouzakova et al., 2010).

### *1.2.4. Summary*

While the causal-confusion and rational-normative accounts of imitation can explain certain types of overimitation in specific contexts, particularly when it comes to object-learning imitation, they fail to explain a wide variety of other factors and situations in which overimitation occurs. In contrast, the affiliative account of overimitation appears to hold promise in generating new theories on overimitation. The affiliative account posits that we copy in order to achieve certain social goals, and imitation thus functions as social glue; however there is no clear theory on how imitation performs this function. In this thesis we have developed and tested the social-signalling theory of imitation, which builds on the affiliative account to generate testable hypotheses on how imitation functions as a social signal. In the next section we review this in greater detail.

## 1.3. Imitation as a social signal

As reviewed in the previous section, the literature on copying appears to suggest that being copied leads to positive social outcomes, while not being copied is associated with more negative social outcomes. We now turn to the question of whether this is a happy side-effect, or one of the aims of imitation. The ***social top-down response modulation (STORM)*** model (Wang & Hamilton, 2012) reviews a wide range of existing neurocognitive evidence to suggest that imitation is socially modulated and incorporates a "Machiavellian goal of increasing one's social standing". The STORM model thus makes a highly specific claim that we imitate in order to affiliate (in the same vein as the argument advanced in Farmer et al., 2018; Over & Carpenter, 2013). The model suggests that the decision to copy or not in a specific situation is modulated by whether your interaction partner is available to receive your signal (i.e., that you are imitating them) (Wang & Hamilton, 2012). This claim has not been rigorously tested, however, and that is what this thesis is attempting to do. We know from the literature above that imitation is modulated by social context, but we do not know if it is specifically a signal that is modulated by whether we are being watched or not.

### *1.3.1. What constitutes a signal?*

In the literature so far, we know that some social factors influence the degree of imitation. However, we do not know if imitation is explicitly a social signal. Here, it would be useful to define what a signal comprises; building on the definitions used in animal behaviour (Stegmann, 2013) we see a ***signal*** as an action that is sent by one individual, received by another, and benefits both parties in an interaction. In this respect it stands in contrast to a ***cue***, which is an action that only benefits one party in an interaction. A classic example of a cue is when a mosquito detects carbon dioxide in the air and finds a mammal to bite; the mammal is not intentionally sending this out as a signal to derive any benefit. A signal in contrast is sent to benefit both parties in an interaction. Here, as we have seen from the literature reviewed in the previous section, we know that imitation increases prosociality and has a range of positive outcomes. We also know that not being copied in some cases leads to an increase in stress. This offers support to the hypothesis that imitation may be a social signal.

### *1.3.2. Can we engender a change in behaviour (imitation) by changing the social context?*

The other building block of this theory rests on us being able to manipulate the extent to which an imitative signal is sent by manipulating the social context. As we noted above, a variety of social factors relating to the interaction partner (including availability, group membership, status) may influence the degree of imitation. In this thesis we are exclusively focused on whether the interaction partner is available to receive the imitative signal, and whether manipulating this availability can engender a change in the degree of imitation.

We will now review the evidence that being watched by an interaction partner can influence our behaviour. Early animal studies conducted on rats, monkeys, and even cockroaches have shown evidence of ***social facilitation*** or changes in behaviour in the presence of a conspecific (Zajonc, 1965); these effects have been thought to arise from an increase in arousal, and occur regardless of whether the conspecific is actually watching them or not (Zajonc & Sales, 1966). When it comes to the specific effect of being watched, in humans this is termed the ***audience effect***, a phenomenon that has been studied for a long time (Triplett, 1898). As we have seen in Section 1.2.3 there is growing evidence that the presence of a demonstrator influences the degree of imitation. In other realms too, we see that being watched leads to changes in behaviour. Cañigueral & Hamilton (2019b, 2019a) have shown that participants tend to gaze less at a live interaction partner (when

compared with a video), and behave in a more prosocial manner when they believe they are being watched. Other studies have also shown that people's behaviour changes even when the feeling of being watched is manipulated at an abstract level, such as when people are told their cognitive capacity is being evaluated (Bengtsson et al., 2009), or when people make disclosures about themselves in the presence of others (Izuma et al., 2010).

Taken together these studies suggest that being watched will change behaviour, but the evidence on the direction of behavioural change is mixed and may depend on the task (both in terms of novelty and complexity) and social context. In this thesis, we are seeking to test whether imitation is a social signal. If Alice and Betty are engaged in a pairwise interaction, and Alice performs an action that includes irrational steps, then we are interested in whether Betty copies Alice's action, and whether this copying constitutes a social signal. If it is a social signal, then it should be influenced by the extent to which Alice is ***socially-available*** to receive a signal. That is, Betty should copy the irrational steps when she knows Alice is watching her, and not bother with copying the irrational steps when she knows that Alice is not watching her.

In this thesis we manipulate the social availability of an interaction partner by designing a paradigm where in some trials the interaction partner will be viewing the participant make a response and in other trials the interaction partner will not be viewing the participant make a response. In the case of Alice and Betty, in half the trials Alice will watch Betty make her response, while in the other half of the trials Alice will have her eyes closed. The full paradigm design is discussed in detail in Chapter 2. If imitation is a social signal sent by Betty to Alice when Alice performs an irrational action, then the following should occur:

(a) Betty should recognise (either consciously or unconsciously) that Alice has performed an irrational action
(b) Betty should recognise on a trial-by-trial basis whether Alice is socially-available or not (i.e., whether Alice is watching Betty or not)
(c) Betty should copy Alice's actions, including potentially Alice's irrational actions
(d) The extent to which Betty copies Alice's irrational actions will be modulated by Alice's social availability

Of these hypotheses, (a) and (b) will be tested by measuring brain activity (in the experiments in Chapter 4 and Chapter 5), while (c) and (d) will be tested behaviourally across all four experiments (outlined in Chapters 3-5) in this thesis.

## 1.4. Brain mechanisms of imitation

From addressing why we copy, and delineating our theory of imitation as a social signal, we now turn to the brain mechanisms underpinning imitation. We start by reviewing the basics of imitation, which involve observing and producing actions. We then incorporate evidence that imitation is selective and highlight cognitive models that account for this, as well as brain regions thought to be implicated in this selectivity and control of imitation. We turn then to the question of how we assess and process irrationality before finally reviewing what we know of the neural correlates of being watched.

### *1.4.1. Understanding actions, and controlling imitation*

On a basic level imitation involves observing and producing an action. The ***direct-matching hypothesis*** (Rizzolotti et al., 2001) posits that we understand actions by mapping the visual representation of the observed actions onto our motor representation of the same action, implying some sort of "resonance" between the observational and motor systems. The genesis of this idea comes from the discovery several decades ago of neurons in the F5 area (the ventral premotor cortex) of the macaque monkey that showed activation both when the monkey observed and executed actions (di Pellegrino et al., 1992; Rizzolotti et al., 1996). These neurons in macaque monkeys have been termed ***mirror neurons***. While single neuron recordings have rarely been recorded in humans (although see Mukamel et al., 2010 for an account of single neuron responses in the medial frontal and temporal cortices), there has been detailed evidence from functional magnetic resonance imaging (fMRI) studies that show that there exists a ***mirror neuron system*** (MNS) in humans which comprises cortical areas active during action observation and action production (Buccino et al., 2001; Kilner et al., 2009).

The human MNS comprises the frontal gyrus (IFG), or Broca's area, the inferior parietal lobule (IPL) and the superior temporal sulcus and middle temporal gyrus (STS/MTG). The MNS is thought to enable imitation by directly mapping observed actions onto one's motor system (Iacoboni, 1999, 2005; Rizzolotti & Craighero, 2004). A large meta-analysis by Caspers et al. (2010) reviewed 139 experiments that used fMRI as well as positron emission tomography (PET) to identify cortical areas that were consistently involved in action observation and imitation. This meta-analysis found consistent activations in a broader MNS including the IFG, the IPL, the premotor cortex and adjacent superior frontal gyrus, supplementary motor area, and visual area V5 during action observation and imitation tasks. Thus, the MNS is part of the visuo-motor system, responding both when we perform actions and observe them. A number of studies have

shown that parts of the MNS such as Broca's area (Molnar-Szakacs et al., 2005; Nishitani & Hari, 2000) are active when observing and imitating a wide range of actions ranging from hand movements (Molenberghs et al., 2009) to lip forms (Nishitani & Hari, 2002). The direct-matching hypothesis suggests that the MNS is the primary mechanism through which we can understand others actions and imitate them (Rizzolotti & Craighero, 2004; Rizzolotti & Sinigaglia, 2010). However, this assertion remains controversial. Two key issues to address here are what we mean by action understanding, and how the brain decides to control imitation and select which actions to imitate. We examine these in turn below.

Imagine we are watching someone put away toys in a playroom. Viewing her we can describe what she is doing as (variously): 'housekeeping', 'tidying a playroom', 'putting the Duplo™ away', or 'utilising a precision grip to move blocks from the floor to a box using a relatively straight-line trajectory'. Although the actions appear identical from a kinematic point of view, the brain areas implicated in the lower-level processing are different from the brain areas involved in synthesising and higher-order processing of actions. Early MNS research claimed that the MNS is involved in "understanding from within" suggesting it plays a key role in high-level processes such as inferring intentions from observed actions (Rizzolotti & Sinigaglia, 2010). However, this assertion is unsupported by evidence: as Heyes and Catmur (2021) note if we can understand intentions only by matching motor movements, then those who cannot match movements, such as individuals born without upper limbs, would not be able to undertake action-recognition, and here the evidence is decidedly mixed. Evidence from neuroimaging studies also show that the MNS is involved in encoding low-level representations of observed actions ('using a precision grip to move blocks') rather than abstract representations ('tidying up') (Wurm & Caramazza, 2019; Wurm & Lingnau, 2015). Instead, other brain regions have been implicated in instantiating this higher-order processing. Csibra (1993) argues that empirical evidence suggests that rather than understanding action via direct mirroring or matching, we instead interpret actions outside of the motor system. Brass and colleagues (2007) showed that understanding actions in plausible and implausible contexts (a demonstrator used their knee to press a light switch when their hands were occupied versus when their hands were unoccupied) involved activations in the STS and the dorsolateral prefrontal cortex (dlPFC) rather than traditional MNS areas. This suggests that the MNS is involved in low-level processing rather than making higher-order inferences about actions (Heyes & Catmur, 2021; Thompson et al., 2019).

We now turn to the question of imitation control and selectivity. As we have seen in the section on theories of imitation (see Section 1.2.2) humans do not imitate indiscriminately. Imitation is a sophisticated and dynamic process, and for each encounter, we choose whether to copy or not, and whether to copy only the end goal, the kinematics of the movement, or both. This is dealt with in theoretical models by incorporating a control mechanism; below we review two models that address this question.

The Emulation, Planning and Mimicry (EP-M) model envisages two routes by which imitation may occur (Hamilton, 2008). This model suggests that there are three main nodes in the brain involved in supporting the observation and imitation of an action: the IFG, the IPL and the MTG. All action is initially observed and processed in the MTG where key kinematic features are extracted. Following this, if the action is goal-directed then it follows an emulation pathway to the IPL, where the goal of the action is processed. Next the signal is sent to IFG via a planning pathway where the kinematic plan for the action execution is formulated. This is termed the emulation-planning pathway for goal-directed actions. For actions where there is no goal, a direct mimicry pathway is posited from the MTG to the IFG.

The STORM model (Wang & Hamilton, 2012) in contrast suggests that imitation is controlled in a top-down manner rather than within the MNS. Here, it is suggested that the MNS performs the action-observation and execution functions as expected, but this is modulated by the medial prefrontal cortex (mPFC) which imposes a social meaning for the actions onto the MNS. This allows us to distinguish between someone raising their hand to wave to a friend across the hall versus raising their hand to hail a cab for example.

A recent review of the mechanisms of imitation reviewed the empirical evidence for the direct-matching hypothesis as well as the EP-M and STORM models and found that there was evidence to support an integrated EP-M and STORM model (Yates & Hobson, 2020). Indeed in the original formulation of the EP-M model it was suggested that the dysfunction in the M-pathway could arise from a disruption to the top-down modulation of the signal (Hamilton, 2008).

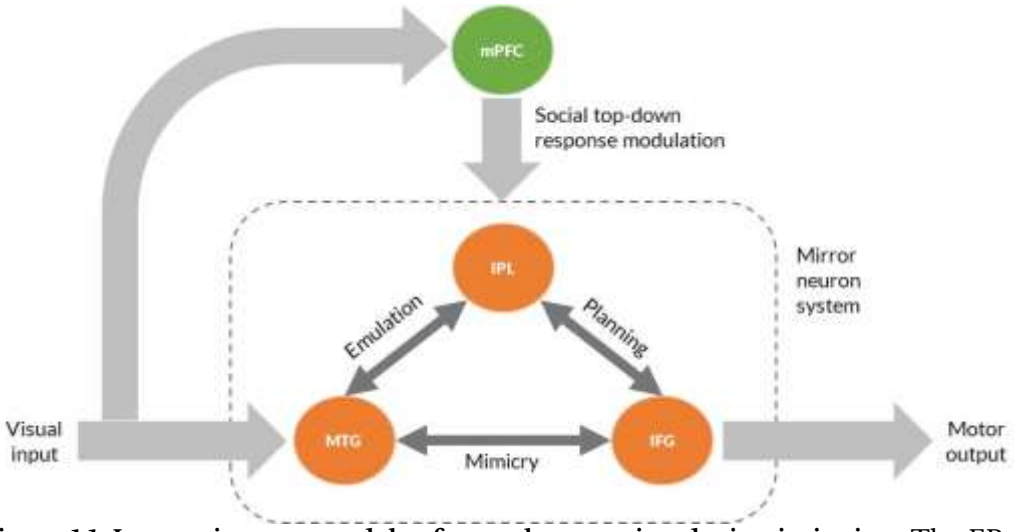


**Figure 1.1. Integrating two models of neural processing during imitation**. The EP-M model is depicted within the dotted lines comprising the MNS. This delineates two paths for information processing within the MNS: the Emulation-Planning (EP) pathway which allows for processing of goals of an action before generating a motor plan; the Mimicry (M) pathway in contrast allows for quick, direct copying, eschewing higher-order cognitive processing (Hamilton, 2008). The STORM model (Wang & Hamilton, 2012) incorporates a top-down socially-modulated control of this process as well. Figure adapted from Wang & Hamilton (2012)

*Brain regions involved in control of imitation*

We now turn to reviewing the most recent empirical evidence from brain imaging studies showing the regions involved in the control of imitation. Inhibiting imitation appears to involve the mPFC and the temporal-parietal junction (TPJ), as shown in a pair of studies by Brass and colleagues (2001, 2005). Another pair of studies by Brass and colleagues (2003, 2005) also showed that the networks involved in inhibiting imitation are both anatomically and functionally distinct from those involved in inhibiting other types of prepotent responses such as those engaged in the Stroop task. This suggests that this is domain-specific to imitation, rather than part of a more domain-general inhibitory mechanism. The mPFC and the TPJ also form the core of the mentalising network (Amodio & Frith, 2006; Frith & Frith, 2003) leading to suggestions that imitation inhibition and mentalising may be linked; Brass et al. (2009) suggests that both require good self-other distinction. In a series of studies they established that mentalising ability and the ability to inhibit imitative responses are positively related in those with lesions in the prefrontal cortex or the TPJ (Spengler, von Cramon, et al., 2010), in those with autism (Spengler, Bird, et al., 2010), and in healthy neurotypicals whose self-other distinction was manipulated experimentally (Spengler, Brass, et al., 2010). Several stimulation studies have shown that applying excitatory stimulation via transcranial direct-current stimulation (tDCS) to the TPJ improves imitative control (Hogeveen et al., 2015;

Santiesteban et al., 2012, 2015); in a similar vein disruptive stimulation targeting the TPJ via transcranial magnetic stimulation (TMS) has been shown to interfere with performance on imitative-control tasks (Sowden & Catmur, 2015). The mPFC meanwhile is implicated in modulating imitative behaviour, particularly in mediating the effect of social priming (Wang & Hamilton, 2015) and eye-gaze (Wang, Ramsey, et al., 2011) on imitation. It has been suggested that the TPJ may be involved in inferring temporary states of mind, while the mPFC integrates these over time into broader traits (Van Overwalle, 2009). In summary the evidence suggests that mentalising networks in the brain are also involved in imitation.

### *1.4.2. Assessing rationality*

Beyond observing and reproducing actions imitation also involves examining the rationality of actions (i.e., whether it is congruent with expectations given the implicit goal of the action and the context). In a seminal study Gergely and colleagues developed a paradigm showing a ball moving from point A to point B with a curved, high trajectory; in the rational version of the experiment, a large rectangular obstacle is shown placed between points A and B, while in the nonrational version, the obstacle is shown placed before point A (Gergely et al., 1995). In this thesis this is the conception of ***rationality*** that we are focused on: a rational agent is assumed to perform an action that will lead to achieving his goal in the most efficient manner (Gergely et al., 1995). In this context, irrational actions are those that are inefficient kinematically. Merely moving a ball from A to B with a high trajectory is hard to interpret: if we see the obstacle between the points then it is congruent with expectations; if however, there is no obstacle, then the high trajectory violates our expectations (Gergely & Csibra, 2003) and can be considered irrational. Several studies have shown that these violations of expectations are accompanied by activations in the STS (Grèzes et al., 2004; Pelphrey et al., 2003; Saxe et al., 2004). Activation in an adjacent region, the MTG has been found to be positively correlated with the degree of (ir)rationality of an action (Jastorff et al., 2011; Marsh, Mullett, et al., 2014). Several studies have also shown increased activation in the TPJ and the IPL when observing irrational actions (Brass et al., 2007; Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011; Oliver et al., 2017). One brain region where the evidence is mixed is the mPFC: two studies showed deactivation in the mPFC for irrational actions (Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011), while others have found an increase in activation in this region when it comes to observing novel irrational actions (Brass et al., 2007) or when there was a mismatch between the content of a narration by an actor and their facial affect (Decety & Chaminade, 2003). It should be noted that all the studies

reviewed above are observational studies; for practical reasons it is difficult to study imitation within the constraints of a scanner. In using a novel wearable neuroimaging modality (covered in detail in Chapter 2) we hope to advance our understanding of the neural correlates of processing irrational actions in the context of choosing to imitate them or not.

We should also explicitly note that irrational and rational actions as envisaged in this thesis, and in this literature on overimitation in general, can also be described as efficient and inefficient ways of achieving a goal, or involving a violation of expectations on how a goal should be reached. In line with how irrationality is envisaged as a behaviour in this field, this is how the terms have been used in this thesis.

### *1.4.3. Being watched*

As we set out to test out our social-signalling hypothesis of imitation it is important to also understand brain regions that may encode the social availability of an interaction partner. The key question here is whether there is a brain region that responds to being watched. The answer is tricky to investigate from a technological standpoint within typically used neuroimaging modalities since they usually study one subject in isolation in a scanner. However, there are several studies that have attempted to overcome these barriers using innovative solutions. One fMRI study allowed a participant inside the scanner to interact face-to-face with a partner in the scanner room via a mirror-array mechanism; it was shown that direct gaze by the partner was associated with increased activation in the IFG, premotor cortex and the supplementary motor area, while increased activation in the mPFC was seen in the mutual-gaze condition (Cavallo et al., 2015). A hyperscanning fMRI study—where two participants were scanned simultaneously side-by-side—measured neural activation when pairs of participants exchanged eye signals found increased activation in the anterior cingulate cortex (ACC) and the cerebellum during live eye contact when compared to a condition where previously-recorded eye contact was replayed (Koike et al., 2019). Another hyperscanning study using functional near-infrared spectroscopy (fNIRS) and eye-tracking found that participants who were engaged in a social joint attention task (cued by eye gaze) showed greater activation in the right TPJ (Dravida et al., 2020). Other studies have also shown that mentalising networks are reliably engaged when we encounter direct gaze (Wang, Ramsey, et al., 2011) and when people believe they can be seen (Somerville et al., 2013). Some studies that have manipulated the feeling of being watched at a more abstract level by telling people that they are being evaluated in some way: Bengtsson and colleagues

(2009) found that when a group of participants were told that their cognitive abilities were being evaluated, they showed greater activation in the anterior paracingulate cortex, which is part of the mentalising network. Another study induced feelings of embarrassment among participants in a scanner and found increased activation in the mentalising networks (Müller-Pinzler et al., 2015). Similarly, subjects making self-disclosures showed strong activation in the mentalising networks including the mPFC and the striatum during self-referential processing (Izuma et al., 2010).

### *1.4.4. Summary*

In summary, observing and producing actions appears to involve the mirror-neuron system, or MNS, while the inferential and control processes around imitation implicate the mentalising network. Processing the rationality of actions appears to involve both parts of the MNS and the mentalising network. Finally, based on the evidence of gaze studies and audience effects, it appears that the mentalising network is also likely to be involved in encoding the effect of being watched, or the social availability of one's interaction partner.

## 1.5. Studying imitation through deficits: Imitation in autism

When considering the mechanisms underlying copying, it is considered useful to compare the behaviour of neurotypicals with those who exhibit imitative deficits; one large clinical population that has been thought to have imitative deficits is autistic people[4]. These deficits along with the key role that the MNS is thought to play in imitation under the direct-matching hypothesis has led to strong claims that autism is associated with dysfunction in the MNS (Ramachandran & Oberman, 2006), known as the ***broken-mirror hypothesis***. In the next section we first outline what autism involves, before considering the evidence regarding imitative deficits in autistic people and the related evidence surrounding the broken-mirror hypothesis.

### *1.5.1. What is autism?*

Autism is a highly heritable, heterogenous, and lifelong neurodevelopmental condition that affects one's ability to relate to and communicate with others. According to the most

[4] There is ongoing debate within the autism community on the usage of 'people-first' (ie. Adults with autism) or 'identity-first' (i.e. autistic adults) language to describe autism; while many autistic adults prefer the identity-first formulation, this is not universal, and many professionals continue to endorse people-first language (Kenny et al., 2016). This thesis uses both terms interchangeably.

recent edition of the Diagnostic and Statistical Manual (DSM-5) used by clinicians autism is characterised by "persistent difficulties with social communication and social interaction" and "restricted and repetitive patterns of behaviours, activities or interests"(American Psychiatric Association, 2013). To receive a clinical diagnosis these impairments must be present from early childhood, and to such an extent as to "limit and impair everyday functioning". While prevalence estimates vary widely worldwide (largely owing to methodological issues in diagnosis), a recent review commissioned by the World Health Organization (WHO) estimates that around one in every 160 children has autism (Elsabbagh et al., 2012). The condition is heterogenous and the term autism has been used both as an umbrella term for a variety of presentations as well as a specific diagnosis within a category previously known as pervasive developmental disorders (Lord et al., 2020). To clarify diagnostic criteria, the DSM-5 uses autism spectrum disorder (ASD), often also referred to as autism spectrum condition (ASC), as term for the broader presentation, and uses various clinical modifiers to differentiate subgroups. This diagnosis of ASC replaces previously used diagnoses of autistic disorder, Asperger's disorder, or pervasive developmental disorder. In this thesis the terms ASC and autism are used interchangeably in line with these diagnostic criteria.

Autism has a strong genetic component (Le Couteur et al., 1995; Rutter & Thapar, 2014; Steffenburg et al., 1989), and co-occurs more frequently in people with some other genetic syndromes, such as Fragile X Syndrome (Hatton et al., 2006). Autism is also associated with a range of other conditions that frequently co-occur including depression (Stewart et al., 2006), epilepsy (Clarke et al., 2005; Viscidi et al., 2013), anxiety (Kim et al., 2000; Simonoff et al., 2008; White et al., 2009), or attention-deficit hyperactivity disorder, or ADHD (Jang et al., 2013; Johnson et al., 2015). Another common co-occurring diagnosis is of intellectual disability, with rates of overlap ranging from 30-50% (Chakrabarti & Fombonne, 2005; Matson & Shoemaker, 2009).

### *1.5.2. Imitation in autism*

Despite decades of research into the issue (DeMyer et al., 1972) the question of whether people with autism experience imitative impairments remains controversial (for a detailed review see Vivanti & Hamilton, 2014). Impairments in imitation were not initially observed by the diagnosticians who first described the condition observed (Asperger, 1944; Kanner, 1943), but both noted that those with autism often failed to learn from others (Vivanti & Hamilton, 2014). Rogers and Pennington (1991) suggested that autism involved impaired self-other representations that first manifested as imitative

impairments, which then cascaded down into deficits in theory of mind. While the official diagnostic criteria (namely the DSM-5) for autism does not refer specifically to imitative impairments, diagnostic and screening tools do involve evaluating spontaneous imitation (Rutter et al., 2003). To some extent, this may have been justified given early findings. Jones and Prior (1985) for example found that children with autism performed worse than matched neurotypical children on motor imitation tasks, and suggested this may explain the failure of children with autism to learn to use gestures. Another study showed that children with ASC imitate tasks that had high motor demands more poorly compared to low motor demand tasks, while this discrepancy was not seen in neurotypical children (Chetcuti et al., 2019). One recent study showed that individuals with autism imitate intentional as well as accidental actions, while neurotypical individuals imitate only the intentional actions (D'Entremont & Yazbek, 2007). This stands in contrast with a study by Marsh and colleagues (2013) which tested neurotypical children and children with autism on a puzzle-box task (for a detailed overview of paradigms used see Section 2.1) where a demonstrator showed the children how to retrieve a toy from a box using a mix of necessary and unnecessary actions; here, while neurotypical children copied the unnecessary actions much more frequently than autistic children (despite both groups being able to understand these actions as "silly").

Some larger reviews have supported this view that those with autism demonstrate imitative impairments, with one review of 21 studies showing that children with ASC performed worse in imitative tasks (Williams et al., 2004), and suggesting that ASC may be associated with delayed development of imitative abilities. Another review of 53 studies on imitation found impairments among those with ASC and showed that the severity of autistic symptoms was correlated with increasing imitative impairments, but was not correlated with intellectual impairments measured via IQ (Edwards, 2014); however this review also found great heterogeneity in imitative deficits, and found that when only the ability to emulate was tested, those with ASC showed no impairments when compared with neurotypicals. The broken-mirror hypothesis (Ramachandran & Oberman, 2006) emerged as a way to explain the imitative deficits seen in autistic people. This theory suggests that autistic people have dysfunctional MNS which leads to global impairments in imitation.

However, people with autism show great heterogeneity in imitative impairments. A number of studies have shown little or no difference in imitative abilities between neurotypicals and those with autism. One study from two decades ago, for example, showed that children with autism performed better than neurotypical children and

children with Down syndrome at imitating pretend play acts (Libby et al., 1997). Dapretto and colleagues (2006) tested high-functioning children with autism and matched controls on imitating and observing emotional expressions while recording their brain signals, and found that both groups performed equally well, but had different patterns of neural activity; children with autism showed reduced response in the IFG (part of the MNS) when compared with neurotypical children, and the extent to which their response in this region was reduced was inversely related to the severity of their autism symptoms. Another study that similarly tested automatic imitation of facial actions showed no difference between those with autism and neurotypicals (Press et al., 2010). Sowden and colleagues (2016) tested 60 autistic adults with matched controls on an automatic imitation task and showed that both groups showed similar significant imitation effects, wherein they executed an action faster when it was preceded by viewing the same action rather than an alternate action. They also showed that the tendency to imitate was correlated with symptom severity among autistic adults. These results stand in contrast to the broken-mirror hypothesis since according to that theory the imitative abilities of those with autism should be impaired relative to neurotypicals.

Overall, there appears to be significant heterogeneity in imitative impairments, both depending on the exact nature of the task and the severity of autistic symptoms. The empirical evidence here also ties in with the alternate theoretical accounts of imitation described in the previous section, namely the EP-M and the STORM model. The EP-M model suggests that the EP route is intact in those with autism while the M route is impaired affecting their ability to mimic actions. The STORM model meanwhile claims that imitative impairments in autistic people arise from the way the MNS is regulated top-down rather than from within the MNS itself. These explanations appear to be borne out when we consider some of the evidence. Hobson and Lee (1999) showed that autistic people can emulate the end result of an action sequence even when not copying the style of the action. A later replication showed that those with autism copy the style of an action when it is necessary to achieve the goal, but not when it is incidental, while neurotypical participants copy the style of an action in both conditions (Hobson & Hobson, 2008). This suggest that autistic individuals may tend to imitate more rationally, copying the goal and copying the style only when it is necessary, while neurotypicals may tend to copy irrational elements as well.

Other studies have suggested that the imitative deficits in autism may arise from impairments in imitative control rather than global impairments in imitation (Schunke et al., 2016): the authors in this study tested autistic adults and controls using simple

finger-lifting movements in response to biological and non-biological stimuli, and to compatible and incompatible stimuli; while they found that both groups processed biological stimuli faster than non-biological stimuli, they found that autistic adults responded very slowly to incompatible stimuli. Another study testing autistic children and adolescents and matched controls on automatic facial mimicry showed that both groups executed a facial expression faster if a congruent facial expression was observed (Schulte-Rüther et al., 2017); however this compatibility effect was positively related to empathy and emotion processing in the neurotypical controls, while it was negatively related to only age in those with autism. This suggest that basic motor mimicry may be intact in those with autism, but not linked to higher-order social cognitive abilities such as understanding emotions and empathy.

### *1.5.3. Summary*

The existing evidence paints a picture of great heterogeneity in imitative abilities and deficits in those with autism. Some of these results may be attributable to the diverse methodologies used in the studies, involving novel or routine tasks, automatic or voluntary imitation and varying social contexts (Sevlever & Gillis, 2010). Nevertheless this mixed evidence offers a direct challenge to theories of autism which suggests that these imitative impairments in those with ASC arise from MNS dysfunction. The studies reviewed in this section challenge the view that there is a global dysfunction of imitative abilities (and by extension a global dysfunction of the MNS) in those with autism.

When it comes to evaluating the social-signalling hypothesis of imitation, we do not know whether people with autism will identify actions as irrational to the same extent as neurotypicals, whether they will imitate to a lesser, same or greater extent than neurotypicals. Most notably we do not know if the extent of their imitation will be modulated by the social availability of their interaction partner in a manner similar to neurotypicals. But studying this will provide useful answers to enable us to build the theory. In Chapter 5 we will evaluate the behaviour and neural correlates of those with autism in comparison with matched neurotypicals.

## 1.6. Overview of this thesis

This thesis seeks to test the specific claim that imitation is a social signal that is selectively sent, depending on whether the interaction partner is available to receive it or not.

**Chapter 2** outlines methodological considerations including the design of paradigms used to study imitation, before reviewing the need for second-person neuroscience. We outline the usage of fNIRS for naturalistic experiments. Finally, this chapter details how we designed the dyadic block-moving paradigm that has been adapted for use in all the experiments in this thesis.

**Chapter 3** investigates the behavioural response of whether participants imitate an irrational action demonstrated by a naïve co-participant using our dyadic block-moving paradigm. We used motion-capture to track the movements of a Leader who is secretly instructed to make irrationally high trajectories in certain trials, and a Follower who is only told to move the blocks in the same order without being instructed on trajectory. This chapter incorporates the results from a pilot study (N = 22) as well as a pre-registered replication (N = 30) that investigates whether imitative fidelity is modulated by social context.

**Chapter 4** incorporates neuroimaging in the same paradigm to investigate whether participants encode the rationality of their interaction partner's actions as well as their partners' social availability. In a study using ***hyperscanning*** where both participants in the dyad are scanned simultaneously (N = 20) we look for the neural correlates of identifying irrational actions as well as the correlates of being watched by an interaction partner. This chapter also includes some exploratory analysis on improving the standard general linear model by including brain activity from an interaction partner in addition to the usual behavioural regressors.

**Chapter 5** extends the social-signalling hypothesis of imitation by comparing the imitative behaviour and associated neural correlates of both neurotypicals and those with ASC. This chapter seeks to examine whether there are behavioural and/or neural differences between the two groups.

**Chapter 6** summarises the findings from the empirical chapters (Chapters 3-5), as well as discusses the broader implications of this work, and highlights directions for future research.

# Chapter 2. Methodological considerations

This chapter reviews the typical paradigms used to study imitation and evaluates their strengths and weaknesses. We examine the need for genuine second-person neuroscience, and a wearable functional imaging technology (fNIRS) that enables us to design naturalistic paradigms. We detail the design of the dyadic block-moving paradigm which has been adapted for all the experiments conducted in this thesis.

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## 2.1. How do we study imitation

In the previous chapter we reviewed the theoretical underpinnings of imitation and the empirical behavioural and neural evidence available. We will now consider the question of how to evaluate imitative behaviour in the lab, critically analysing existing paradigms before outlining the considerations that drove our paradigm design. In this chapter we will first outline the main types of paradigms used to study imitation, and ideas we can incorporate from the study of irrational actions. Second, we will review the need for a fresh approach to paradigm design incorporating naturalistic social interaction. Third, we will outline the usage of a novel wearable imaging technology—fNIRS—that was deployed for all the neuroimaging experiments in this thesis. Finally, we will outline the design of our ***dyadic block-moving*** paradigm that has been used throughout this thesis.

## 2.2. Typical paradigms used to study imitation

In this section we review the main paradigms that have been used to study imitation including puzzle boxes and isolation paradigms.

### *2.2.1. Puzzle boxes*

When it comes to studying object-learning imitation in particular there is a long tradition of using puzzle boxes to evaluate imitative behaviour (Horner & Whiten, 2005; Lyons et al., 2007, 2011; McGuigan et al., 2007). These are typically transparent or opaque boxes that require a specific set of actions to be undertaken to open them, such as pulling a latch and then lifting a lid. Once opened the central receptacle usually holds a toy or a reward. The box often includes some superfluous mechanism (such as a bolt that does not open), and in an overimitation task a demonstrator would usually perform a series of relevant and irrelevant steps to retrieve the reward before turning it over to the study participant.

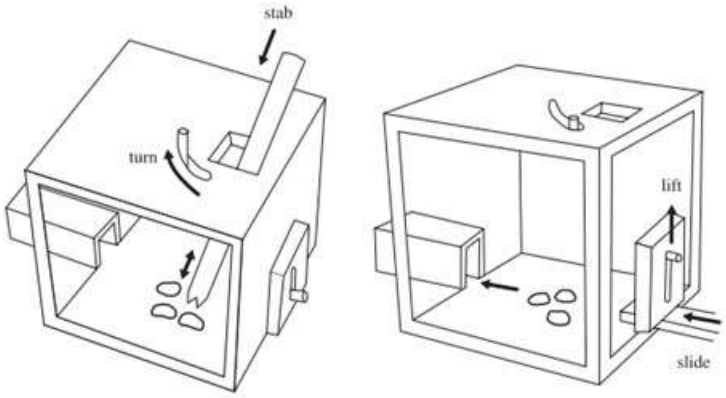


**Figure 2.1. Artificial fruit.** A typical puzzle box, or artificial fruit, used in studying imitation. The box can be manipulated in two ways to access the central core and can be combined with superfluous or irrational movements to study overimitation. Taken from Whiten et. al (2007)

This has spawned a rich vein of research in both humans—adults and children—as well as non-human primates (Custance et al., 2006; Whiten et al., 1996). These boxes have also been called artificial fruit, particularly in the context of testing non-human primates. However, it is unclear whether this novel task is ecologically valid, in the sense of being generalisable to real-world events (Lewkowicz, 2001) when it comes to testing adults. One study with adults using these puzzle boxes by McGuigan, Makison and Whiten (2011) showed that only 25% of the participants thought the purpose of the study was to retrieve the toy from within the box, showcasing that they did not understand the goal of the demonstrated action! This suggests that amid goal-ambiguity, people may well be following a "copy when uncertain" heuristic as suggested by Flynn and Smith (2012). Further, these boxes lend themselves almost exclusively to one-shot trials given their trivial nature once solved. This poses challenges for repeated measurements as well as for generalisability to contexts outside the lab.

### *2.2.2. Isolation paradigms*

When it comes to studies that have attempted to measure brain activity relating to imitation, much of the research has relied on PET (Decety et al., 1997, 2002; Grafton et al., 1996; Grezes, 1998; Krams et al., 1998), Magnetoencephalography, or MEG (Nishitani & Hari, 2000, 2002) or fMRI (see Caspers et al., 2010 for a review). The earliest studies focused on experiments where participants were asked to watch videos of actions in order to be able to recognise or imitate them later, and found differences in activations based on whether the actions were meaningful, and whether they were watching them with an intent to imitate them (Decety et al., 1997). Later studies often also incorporated small hand movements, particularly finger-tapping, that subjects could perform while in the

scanner (Brass et al., 2000, 2001; Decety et al., 2002; Iacoboni, 1999; Koski et al., 2002; Krams et al., 1998; Mengotti et al., 2012; Tanaka & Inui, 2002). Some experiments have also used grasping tasks or tasks involving various hand configurations (Buccino et al., 2004; Grafton et al., 1996). While these studies have provided valuable insights into the neural correlates of observing and reproducing actions, they are limited by the small range of movement possible within the scanner.

A second limitation of these paradigms is that they necessarily use 'isolation paradigms' (Becchio et al., 2010) where individual minds are studied in isolation. Again, these studies have unearthed rich findings, and are very useful when analysing an individual's actions on their own (a first-person account). However, when deployed to study social interactions such as imitation, these paradigms invite us to imagine other people's behaviour or mental states to generate a third-person account of the interaction. Implicit in this formulation is a view that imagining what we would do in the presence of another person is equivalent to actually engaging in a social interaction. Typically, neuroimaging studies of imitation have almost exclusively used pre-recorded video stimuli (Caspers et al., 2010). Some studies have attempted to circumvent the physical limitations of the testing environment by asking participants to interact in real-time with a real (or sometimes fictitious) partner outside the scanner (Decety et al., 2004; Gallagher et al., 2002).

### *2.2.3. Summary*

The existing paradigms used to study imitation have significantly aided our understanding of both behavioural and neural correlates of imitation. However, there are some limitations that arise from the design of existing paradigms, including a lack of ecological validity, difficulties in engendering genuine social interaction or a social context that is manipulable across repeated trials. Next, we turn to an area where there is growing focus, namely second-person neuroscience.

## 2.3. On the need for second-person neuroscience

As we have seen in the previous section, using one-shot paradigms and isolation paradigms are limiting when it comes to studying genuine social interaction. Isolation paradigms are based on the premise that thinking about a social interaction engenders the same neural processes as actually engaging in one, while one-shot paradigms do not allow for repeated trials. Existing paradigms also implicitly incorporate a "fourth-wall"—a barrier that exists between participants and the stimuli (Risko et al., 2016). Lab studies

frequently present participants with stimuli on a screen from which participants glean information; however, this is not a two-way process and participants do not expect to send information back to the stimuli. While this allows for good experimental control, it poses problems for studying genuine social interactions. In our real life we typically interact with other people, in two-way or multi-way exchanges of cues and signals, and there are crucial differences between genuine dyadic (or multi-person) interaction and interacting with a computer. Specifically, when we believe we are interacting with a rational agent we adopt an ***intentional stance,*** interpreting and predicting their behaviour in the context of what we believe about their beliefs, desires and intentions (Dennett, 1971). In the realm of neuroscience, it has been shown that parts of the mentalising network (specifically, the anterior paracingulate cortex) are active when participants believe they are playing against a real person when compared with playing against a computer, although in reality they were playing "rock, paper, scissors" against a randomly-generated sequence in both cases (Gallagher et al., 2002). In response, in recent years there has been an increasing focus on ***second-person neuroscience***, where subjects are able to interact in real-time with others (Schilbach et al., 2013). This approach incorporates the view that a social interaction is more than the sum of its parts, with interactive processes that can both supplement and replace individual processes (De Jaegher et al., 2010). This is of course a lot more difficult to implement, designing paradigms involving two real subjects and measuring brain activity in one or both participants using mobile technologies that allow them to continue to interact naturally.

In a similar vein it has also been argued that capturing the interaction of two brains by simultaneously measuring activity in both—termed ***hyperscanning*** (Montague et al., 2002)—allows us to study complex joint behaviours that may not emerge in isolation (Hasson et al., 2012). The earliest mentions of hyperscanning come from a study where electroencephalography (EEG) signals were simultaneously recorded in pairs of twins (Duane & Behrendt, 1965). However, the idea was not widely pursued, and it is only in recent decades that it has been used again. Many hyperscanning studies use fMRI, EEG or a combination of both (Babiloni & Astolfi, 2014; Dumas et al., 2011; Koike et al., 2015). More recently, using fNIRS for hyperscanning is gaining popularity. A number of studies have used simultaneous recording of fNIRS signals to study inter-brain synchrony (Cheng et al., 2015; Cui et al., 2012; Dommer et al., 2012; Holper et al., 2012; Jiang et al., 2012; Liu et al., 2016; Osaka et al., 2014). A more detailed review of hyperscanning is presented later in this thesis (see Section 4.2.1).

### *2.3.1. Summary*

Without engendering a genuine social interaction, it would not be possible for us to manipulate the social availability of an interaction partner (i.e., whether a participant was watched or not on a trial-by-trial basis) in a meaningful and repeatable manner. Thus, designing a paradigm that incorporates genuine dyadic interaction is a key focus for this thesis.

## 2.4. Using fNIRS for social experiments

When it comes to studying social interactions, one key constraint is the kind of neuroimaging technology that is used and the constraints it places on the experiment. Here, fNIRS is very useful in allowing for much more free-flowing natural interactions between dyads. fNIRS is a relatively novel brain-imaging technique that measures changes in the concentration of oxygenated and deoxygenated haemoglobin in the cortical surface (Boas et al., 2014; Ferrari & Quaresima, 2012; Pinti, Tachtsidis, et al., 2020; Scholkmann et al., 2014). fNIRS was initially deployed with much success to study developing brains (particularly neonates in intensive care units), enabling us to research both typical and atypical development, particularly of behaviours that are hard to study within the constraints of a scanner (Vanderwert & Nelson, 2014). Similarly it has also been used to study various psychiatric and neurodevelopmental conditions including schizophrenia, ADHD and autism (Ehlis et al., 2014). In this section we will first review the mechanics of the technology, before delineating its advantages and limitations.

### *2.4.1. Mechanics of fNIRS*

fNIRS works by taking advantage of the relative transparency of biological tissue to light within the near-infrared optical window. Light within these wavelengths—650nm-950nm—can penetrate through skin, skull, and cerebrospinal fluid to reach brain tissue. This discovery was first made when Jöbsis found that red light penetrated through bone when he was holding it against a visible light (Jöbsis, 1977). As shown in Figure 2.1 when light is transmitted from a source, we can measure the amount of backscattered light available at detectors placed at a suitable distance. The amount of backscattered light will depend on the amount of light absorption and scattering by the various layers that the light passes through. Within the near-infrared window the key chromophore that affects the amount of light absorption is haemoglobin (Scholkmann et al., 2014). Here, the level of absorption owing to oxygenated haemoglobin (HbO) is higher for light of wavelengths above 800nm, while absorption of light owing to deoxygenated haemoglobin (HbR) is

greater for light of wavelengths below 800nm. Most fNIRS systems use two or three different wavelengths and this allows them to measure the changes in light attenuation, which can then be expressed as a change in the concentrations of HbO and HbR. This change in attenuation will be dependent on HbO and HbR since other factors such as absorption and scattering owing to water or other biological tissue is unlikely to change over the course of a study. The relative amounts of HbO and HbR in a brain area are determined by the regional cerebral blood flow which is related to the amount of neuronal activity in that area. Thus, an increase in HbO (and a decrease in HbR) is associated with increased brain activity in a region.

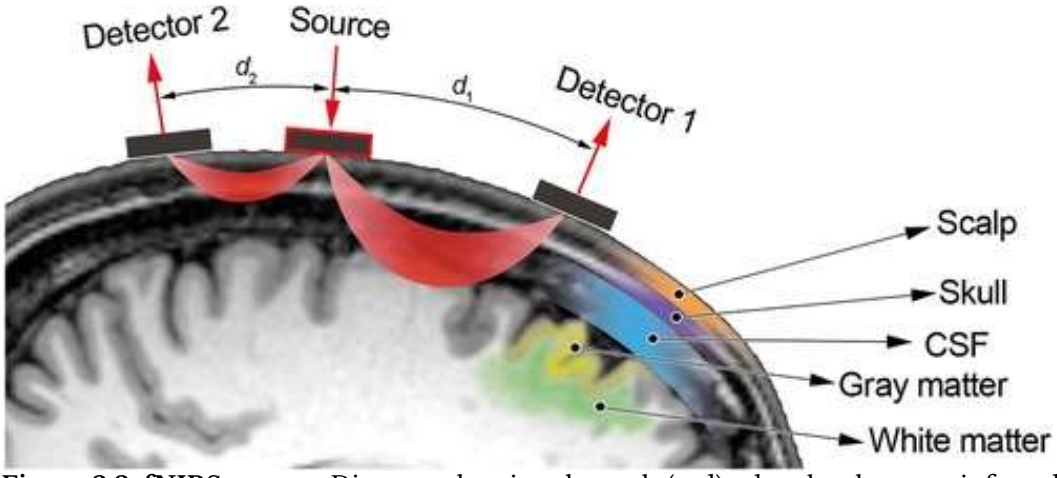


**Figure 2.2. fNIRS system.** Diagram showing the path (red) taken by the near-infrared signal from the light source to the detector through different layers in the brain. The depth to which light penetrates is proportional to the distance between the source-detector. Here, d1 is a deeper channel, while d2 is a shallower channel. (Pinti, Tachtsidis, et al., 2020)

To analyse the signals, first the amount of light detected is converted to optical density. Second, this optical density is converted to concentrations of HbO and HbR using the modified Beer-Lambert Law. Thus signals obtained from fNIRS are similar to the BOLD signal used in fMRI and the method yields comparable results (Noah et al., 2015).

This technique measures the neuronal activity (via estimating blood flow) at a point midway between the source and the detector at a depth of around half the separation between the source and the detector, and this point of measurement is called the channel (Patil et al., 2011; Pinti, Tachtsidis, et al., 2020). Thus, the technology can be used to measure activity on the cortical surface and not activity in deeper brain structures. The depth of the channel can be varied by varying the source-detector distance, but there is a trade-off between channel depth and quality of the signal, with deeper channel depths

leading to greater deterioration of the signal (Pinti, Tachtsidis, et al., 2020). Typically, source-detector separations of 30-35mm are used for studies involving adults while separations of 20-25mm are used for children. The fNIRS system is usually set up with source and detector optodes distributed throughout the region to be studied with a fixed separation between sources and detectors.

### *2.4.2. Advantages and limitations of fNIRS*

fNIRs is low-cost, and non-invasive, and relatively easy to adapt to wider non-clinical use. fNIRS systems are also less susceptible to motion artefacts and can be made portable which greatly expands the range of experiments that can be carried out using the technology (Pinti, Tachtsidis, et al., 2020). Further, fNIRS offers better spatial resolution when compared with EEG, and better temporal resolution when compared with fMRI. In the context of social experiments it is also useful that fNIRS is silent, and compatible with other magnetic and electrical equipment such as motion trackers and physiological monitors (Pinti, Tachtsidis, et al., 2020). However, it does not provide any structural information, and can only measure activity at a depth of 1.5-2cm.

### *2.4.3. Summary*

fNIRS is a wearable brain-imaging technology that allows us to measure neural activity. However, it only provides functional data on neural activity over a time period, rather than any structural information, and is limited to measuring activity only on the cortical surface. These limitations mean that it is worthwhile thinking about which brain regions we want to study before deciding on a technology and adapting our paradigm design to take advantage of the technology to the fullest extent possible.

## 2.5. Paradigm design

In this final section we review our thinking regarding designing the dyadic block-moving paradigm that is used throughout the thesis. Our objective in this thesis is to test the social-signalling hypothesis of imitation as outlined in Section 1.3. To evaluate this, we needed to design a task that would fulfil the following criteria:

a. An easy-to-understand task that allows two naïve participants to interact in a lab setting
b. A believable cover story that would allow us to hide the true behaviour being tested

c. A task that incorporates both rational and irrational elements
d. Allow for easily identifying whether participants copied the irrational elements or not
e. Allow for repeated trials
f. Allow for the social availability of the interaction partner to be varied on a trial-by-trial basis in a simple manner

### *2.5.1. Developing the dyadic block-moving task*

The inspiration for this task comes from different veins of research including studies of mimicry and studies on evaluating rationality. A seminal study by Gergely and colleagues (1995) developed a simple task to test whether 12-month old infants can evaluate the rationality of an agent's movements in attempting to move a ball from point A to point B. In the rational version of the experiment, a large rectangular obstacle is shown placed between points A and B, while in the nonrational version, the obstacle is shown placed before point A (Gergely et al., 1995). This is shown below in Figure 2.3.A. A later study built on this paradigm to evaluate how neurotypical and autistic adults processed action rationality by using different action trajectories (straight and curved) which were rational or irrational depending on the presence or absence of obstacles (Marsh et al., 2015). This is shown below in Figure 2.3.B.

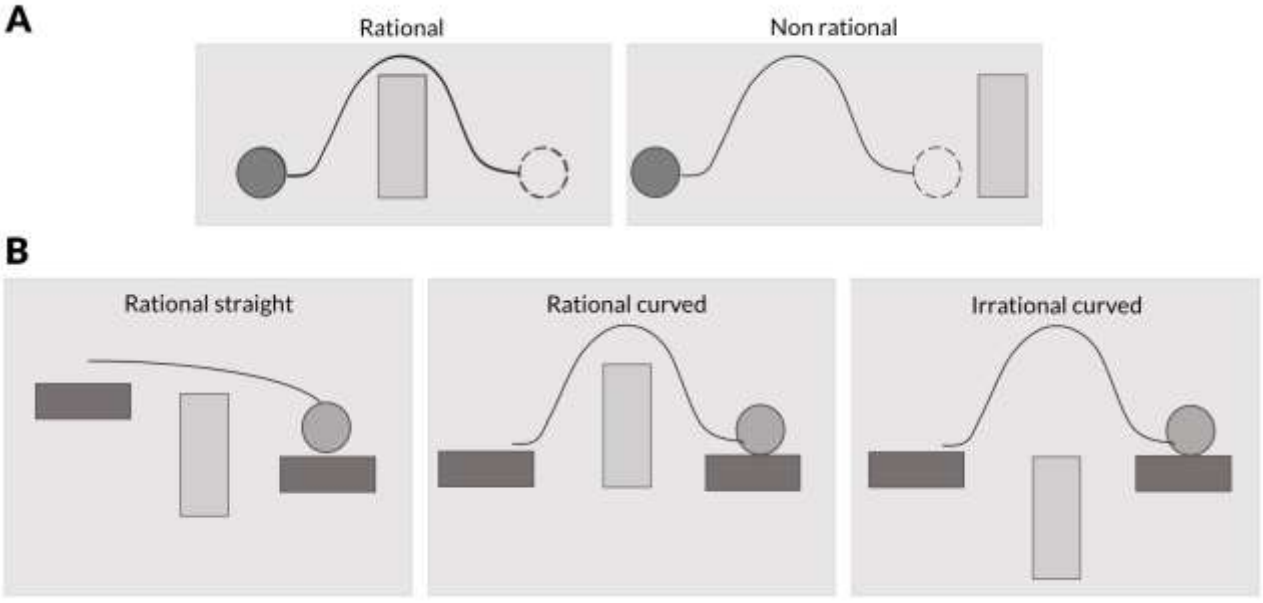


**Figure 2.3. Rational and irrational habituation tasks. A. R**epresentation of the paradigm used in Gergely et. al. (1995) which tested whether 12-month-old infants can adopt an intentional stance when evaluating an agent's movements. **B.** Stylised representation of the trajectories used in the Marsh et. al. (2015) study to evaluate how neurotypical and autistic adults understood action rationality.

The task also draws inspiration from pointing paradigms used in other studies (Forbes & Hamilton, 2017; Wild et al., 2012) that allow us to easily track the kinematics of the movement, and the fidelity with which the trajectory was copied (Krishnan-Barman et al., 2017). The study by Wild and colleagues (2012) evaluated movement kinematics and eye movements when participants watched goal-directed and non-goal-directed hand movements. Building on this, a study tested participants on a goal-directed imitation task without any obstacles but where participants viewed demonstrations with one of three trajectories: low, high, and super high (Forbes & Hamilton, 2017).

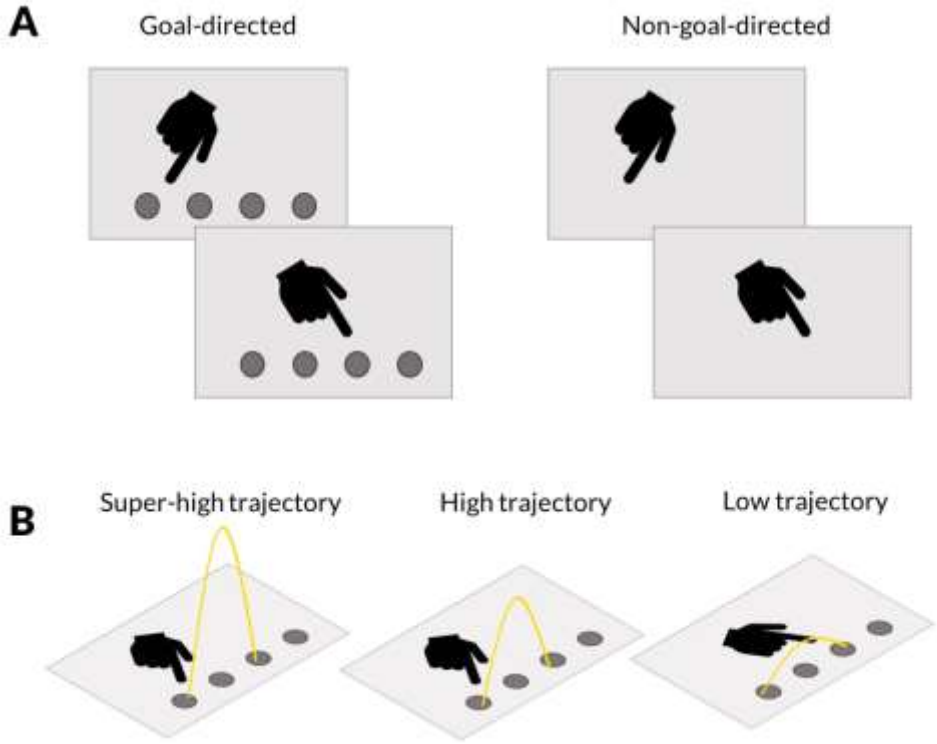


**Figure 2.4. Pointing paradigms. A.** Representation of the paradigm used in Wild et. al. (2012) which evaluated how people with autism are affected by the presence or absence of goals during an imitative task. **B.** Depiction of the task used in Forbes and Hamilton (2017) that recorded participants movements on a goal-directed imitation task after watching demonstrations with one of three trajectories.

Building on these studies we designed a dyadic block-moving task where participants were required to move blocks from one table to another. We combined this with the augmented-reality approach used in Pan and Hamilton (2015) to implement this in a virtual environment wherein participants controlled the movements of the blocks via motion trackers attached to their fingers. This allowed us to minimise set up times and run repeated trials easily; this augmented reality setup also meant that participants were making whole arm movements rather than moving a mouse. This latter approach had previously been used in a simple block-moving task that found only limited social

engagement (Oliver et al., 2017), driving our decision to use an augmented-reality approach instead[5].

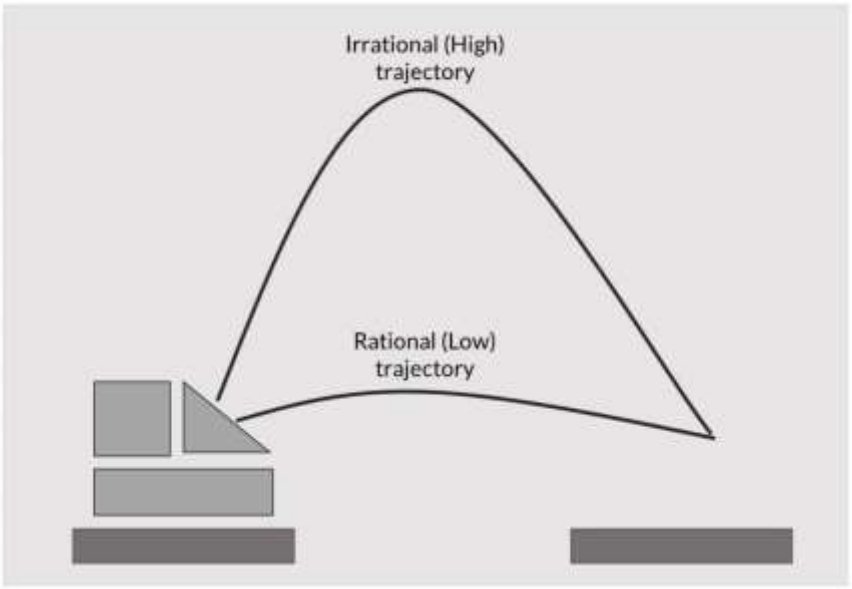


**Figure 2.5. A stylised representation of the dyadic block-moving paradigm.** Participants are asked to move the blocks from one table to another. One participant (appointed as the Leader) is secretly told to move using either irrational or rational trajectories. The other participant (the Follower) is then only asked to move the blocks in the same order as the Leader. We measure the extent to which the Follower imitates the Leader's trajectory on each trial. The Leader is also instructed to close their eyes (and not watch the Follower) during one half of the trials allowing us to measure whether the Leader watching or not watching the Follower has an impact on the Follower's imitative fidelity.

In a dyadic interaction we appointed one participant as the Leader and one as the Follower. Participants were told that they were participating in an experiment designed to assess "information loss". We told them the game they would play would be similar to the game "Telephone" that children play, where they sit in a circle, and a simple message (say, "My aunt likes banana cake") is relayed from child to child by whispering. In the game there is usually some slight mangling of the phrase by each child, and by the end it is often completely different leading to great hilarity among the pre-teen crowd. Many participants in our experiment were aware of the game and understood how it worked. In this vein we told them that they would play a version of this game where the computer would give information in private to the Leader, who would then pass this information

[5] We used this augmented reality approach in the experiments described in Chapters 3 and 4. For the final study involving autistic participants other constraints of where we could position the fNIRS system meant we could not use the augmented reality setup and instead used physical blocks (Duplo™ blocks). The setup of that experiment is described in detail in Chapter 5.

to the Follower, and we would assess how much information was lost between the stages. This was done to make the whole setup slightly more believable and try to hide the fact that we were studying imitation of irrational actions. Participants were told that they would take turns to move the blocks from one location to another, with the Leader going first and the Follower going second. They were told that the Follower would need to close their eyes while the computer gave the Leader instructions and the Leader would then transmit this "information" (on block order) to the Follower.

After this cover story was explained in detail to participants, they were also told that they would receive a score for each trial, which depended on how fast and how accurately they moved, and that they were a team competing against other teams. A range of team scores from previous participants (including some fictional scores) were displayed on a leaderboard in the room where the experiment was being conducted; participants were also asked to choose a team name which was added to the leaderboard at the start of the study, to draw their attention to other teams' scores.

In the actual experiment, the Leader was secretly told to make irrationally high trajectories when moving the blocks in some trials, while in others they were told to make rational straight-line trajectories. We then measured the height demonstrated by the Leader in each trial and the height subsequently reached by the Follower in each trial when it was their turn.

In this paradigm we varied the rationality or irrationality of the Leader's movements by varying the height of the trajectory used. We built on the paradigm used in Forbes and Hamilton (2017) in this regard. The paradigm was initially used in two behavioural studies (Chapter 3) and then extended to a neuroimaging study with neurotypical participants (Chapter 4) and finally to a neuroimaging study with autistic and neurotypical participants (Chapter 5). In extending the paradigm we were keen to keep the main elements the same as in our pilot study to ensure we could test whether the behavioural results replicated across different studies and different populations. However, we should note that as a result rationality is always confounded with the kinematics of the movement in our paradigm. That is, making an irrational move always involves moving higher. We address this in greater detail in the section on future directions in Chapter 6, but in developing future paradigms to test this behaviour it would be useful to also test situations where rationality is manipulated by the presence or absence of obstacles, for example, while keeping the movement kinematics the same.

Depending on the constraints of the experiment the Leader was either a naïve co-participant, or a trained confederate. In particular, the confederate was used in the autism study described in Chapter 5, where the constraints of testing participants who had made special arrangements to arrive for the experiment meant we could not risk disruptions to the testing schedule. There are both advantages and disadvantages to using a trained confederate rather than a naïve participant. There is a risk that the confederate may modify their behaviour especially between groups such as neurotypicals and autistic participants since the study is not conducted in a double-blind manner. Second, if subjects became aware that they were doing the task alongside a trained confederate they could start performing differently to potentially meet what they perceive as the experimenter's expectations, a phenomenon known as experimenter effect (Gilder & Heerey, 2018; Kuhlen & Brennan, 2013; Rosenthal, 1966). However, the confederate can be trained to perform exactly the same across all the trials and takes away the need to familiarise them with the secret instructions on the different trajectories to be demonstrated unlike naïve participants who needed more time to be specifically instructed on this in secret.

Another challenge in the study was to generate a genuine feeling of being observed in one-half of the trials. As we have noted earlier, even very young children show a greater propensity to imitate live models rather than videotaped models (Nielsen et al., 2008), while other studies show that participants viewing video know that they are not really being watched and this compromises the ecological validity (Risko et al., 2012). This issue is also addressed by the usage of live interaction partners (the Leaders) in our studies. In one-half of the trials in each experiment the Leader was instructed via computerised voice cues to close their eyes while the Follower made their movement. This allowed us to manipulate the cognitive effect of being watched on a trial-by-trial basis.

It is important to note that being watched can also lead to social- facilitation effects (Zajonc, 1965), changes in anxiety due to direct gaze (Senju & Johnson, 2009), or changes in attention. In a recent review Heyes (2017) contends that several results that show modulation of imitation by social context instead arise from differences in attention or anxiety and are not related to social-signalling. This has been difficult to test in much of the existing research because the social context varies widely between the watched and the unwatched conditions.

In this thesis we sought to explicitly address this by ensuring the watched and unwatched conditions were as closely matched as possible: specifically, the interaction

partner stood side-by-side with the participant (precluding any gaze effects) and was present throughout the experiment (rather than leaving the room in some trials which may instead have led to a difference in social facilitation or attention). Given the nature of the task participants were encouraged to move quickly and accurately (which would promote a straight-line trajectory over an irrationally curved one). Further, if participants were feeling more anxious in the trials where they were watched, the hypothesised effect (of moving faster, straighter and lower as a result of anxiety) would run counter to our hypothesis of imitation being boosted by being watched. Finally, the demonstration of the block-moving task by the Leader was identical in both the watched and unwatched trials, allowing us to rule out attentional explanations.

### *2.5.2. Summary*

The dyadic block-moving task used in this thesis was designed to allow us to measure imitation in a social context while easily manipulating the rationality of demonstrated actions and the social availability of the interaction partner. To measure brain activity in these studies, we have utilised fNIRS, which is a novel wearable imaging technology that enables the recording of signals from a single brain as well as hyperscanning, or simultaneously recording from both participants in a social interaction.

# Chapter 3. Adults imitate to send a social signal

In a pilot study and a pre-registered replication, we examine the fidelity with which naïve participants copy the exaggerated trajectories demonstrated by a fellow participant in a simple block-moving task. The studies in this chapter use an augmented-reality paradigm and pairs of naïve participants who are randomly assigned the roles of Follower and Leader. Imitation fidelity is measured when the Follower knows that the Leader is watching them, and when the Follower knows that they are not being watched, allowing us to test the social-signalling hypothesis of imitation.

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*One of the studies described in this paper was preregistered at Open Science Framework:*

Krishnan-Barman, S., & Hamilton, A. F. de C. (2017). Overimitation: Examining the effect of social context. Retrieved June 10, 2020 from https://osf.io/ezj8g/

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## 3.1. Abstract

Humans frequently imitate each others' actions and often do so with high fidelity. A variety of reasons have been proposed for why this behaviour occurs. Here we test the hypothesis that imitation can serve as a social signal, occurring with greater fidelity when the participant knows that they are being watched by their interaction partner. A pilot study with 22 pairs of participants and a pre-registered replication with 30 pairs of participants were conducted. Participants were assigned the role of Leader and Follower and participated in our dyadic block-moving task in an augmented reality environment. The Leaders were privately told to move the blocks using specific trajectories, including exaggerated trajectories in some trials. We measured the extent to which Followers imitated the trajectory height demonstrated by the Leader, both in trials where the Leader watched the Follower's actions, and trials where the Leader did not. Followers imitated the Leader's trajectories, and critically, the strength of this correlation was greater in trials where the Follower knew they were being watched by the Leader. This suggests that Followers used imitation fidelity as a social signal in a nonverbal task, supporting the social-signalling hypothesis of imitation outlined in Chapter 1.

## 3.2. Introduction

Humans imitate prolifically, from early childhood through to adulthood, and even when imitation is not strictly necessary (Nadel, 2002; Whiten et al., 2016), but we are yet to fully understand why. A variety of explanations have been advanced to explain imitation, including as a mechanism to learn new skills (Flynn & Smith, 2012), as a by-product of domain-general learning (Heyes, 2017), or as a way to boost social affiliation (Over & Carpenter, 2013; Uzgiris, 1981). This latter theory, also known as the 'social glue hypothesis' (Lakin et al., 2003), suggests that imitation is a social signal which can influence an interaction (Wang & Hamilton, 2012). The aim of the current paper is to test this social-signalling hypothesis of imitation, in a robust fashion.

The genesis of this idea comes from examining how it is possible for imitation to create affiliations between people. Such affiliation could emerge as a lucky side-effect of imitation, but the STORM model (Wang & Hamilton, 2012) makes the more specific claim that imitation is performed in order to affiliate (Farmer, Ciaunica, & Hamilton, 2018). We illustrate this with a scenario in which Alice imitates an action performed by Ben. If imitation influences affiliation, Ben should receive the signal 'I am imitating you' and change his attitude or behaviour towards Alice in response. This is supported by evidence that being imitated leads to an increase in liking (Lakin & Chartrand, 2003). Further, if Alice imitates in order to send a signal to Ben, she should imitate him with greater fidelity when she knows he is watching her, compared with when she knows he is not watching her. Here we consider this latter prediction – that imitation should be produced with greater fidelity when the interaction partner is watching and can therefore receive the social signal being transmitted.

Previous work testing if imitation increases when a participant is being watched (and can send a social signal) has yielded mixed results. Studies using video stimuli have shown that imitation is enhanced when a direct gaze cue is present at the time of responding (Wang, Newport, et al., 2011; Wang & Hamilton, 2015). A study of facial mimicry[6] found stronger imitation of a wince following eye contact, supporting the social-signalling hypothesis (Bavelas et al., 1986). In some studies, children imitate the irrelevant actions performed by a demonstrator only when the demonstrator is present

[6] Mimicry is a subset of imitation referring to copying of actions that are not goal directed. Overimitation, meanwhile, involves copying unnecessary or causally-irrelevant features of a goal-directed action (Hamilton, 2015). In this chapter we use the neutral term imitation to refer to all copying behaviour, whether explicitly goal-directed or not.

during the child's turn (DiYanni et al., 2011; Nielsen & Blank, 2011). However, other studies suggest that children overimitate even when the demonstrator is absent (Lyons et al., 2007), and that both children and adults overimitate when they are not aware of being watched (Whiten et al., 2016). These latter studies argue against imitative behaviour serving as a social signal. However, these may be due to other confounding factors, which overshadow the social-signalling effect. Several of these studies use puzzle-box tasks where learning about a novel object may dominate the response. In the case of the study by Lyons and colleagues (2007) we suggest there may have been ambiguity over whether the demonstrator leaving the room actually meant the participant was not being watched (see Section 1.2.3). In contrast, a study by Marsh, Ropar and Hamilton (2019) showed that children overimitate when the demonstrator watched them *and* when the demonstrator left the room, but not when she turned away from them in the same room. This suggests that perhaps in an experimental context, the demonstrator exiting the room still leaves open the possibility that we may be being watched from outside, while clear disengagement from a demonstrator in the same room reduces the propensity to overimitate. Many studies use confederates to demonstrate the to-be-imitated actions, which could lead to an experimenter effect (Gilder & Heerey, 2018; Kuhlen & Brennan, 2013). Other studies use video stimuli where participants know they are not really being watched, compromising ecological validity (Risko et al., 2012). Finally, the situations where someone is being watched versus one in which no one is watching can engender several possible cognitive changes (Bond, 1982), including social-facilitation effects, changes in anxiety due to direct gaze and changes in attention. One recent review paper has suggested that several results showing modulation of imitation by social context arise due to effects of attention or anxiety and are not related to social-signalling (Heyes, 2017). Given that in several extant studies the social context differs significantly between the watched condition and the unwatched condition, it has not so far been possible to explicitly test whether social signalling drives imitative behaviour in adults.

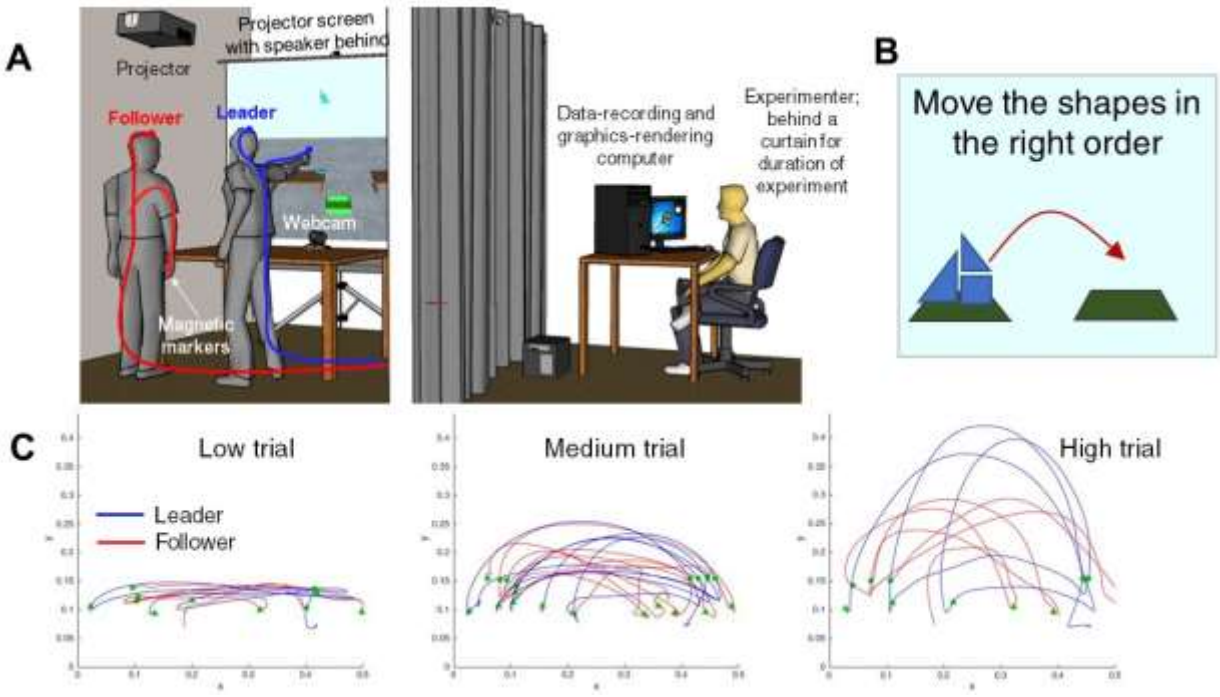


**Figure 3.1. An overview of the experimental setup. A.** Configuration of the augmented reality (AR) lab. Two participants standing side-by-side can see the AR space and are motion tracked. A curtain separates the participants from the experimenter. **B.** The task was to move blocks from one table to another in a demonstrated order. **C.** Sample trajectories from leader (blue) and follower (red) for trials with different demonstration heights. From Krishnan-Barman & Hamilton (2019).

The current study aims to test the social-signalling hypothesis of imitation in a rigorous manner, avoiding confounding factors that have affected previous studies. In this study pairs of naïve adult participants were asked to move blocks from one location to another in a specified order, as part of our dyadic block-moving task. The inspiration and design of this task is detailed in Chapter 2 (see Section 2.5.1) and builds on a range of paradigms used in the study of mimicry and on evaluating rationality (Forbes & Hamilton, 2017; Gergely et al., 1995; Krishnan-Barman et al., 2017; Marsh et al., 2015; Oliver et al., 2017; Wild et al., 2012) (Figure 3.1). This augmented-reality setup provided a rich interactive context while avoiding experimenter effects.

Two independent variables were manipulated: the height of trajectory demonstrated by the Leader, and whether the Leader's eyes were open or closed during the Follower's turn. As the two participants stand side by side throughout there are no changes in eye contact or social facilitation between the two conditions. This is akin to studies of visual perspective taking using 'goggles' (Teufel et al., 2010) which have been accepted as a definitive test of 'social' information processing (Heyes, 2015). Finally, we resolve issues of variance in participant performance by using a simple task with clear rules for excluding non-compliant participants. The study was pre-registered to support

a rigorous analysis scheme. This chapter presents the results of an exploratory pilot study, and a preregistered replication.

Based on the STORM model, we predict that

(a) Followers will copy the heights of the trajectories demonstrated by Leaders without being explicitly instructed to do so
(b) The fidelity with which Followers copy the heights demonstrated by the Leaders will be greater when the Leaders are watching the Followers, compared with trials where the Leaders are not watching the Followers

## 3.3. Pilot experiment

### *3.3.1. Materials and Methods*

*Participants*

22 pairs of participants were tested in the exploratory pilot study. In eight of these dyads the role of Leader was taken by a confederate because one of the participants failed to arrive on time for the booked slot. Excluding the confederate, a total of 36 participants were tested in 22 pairs (17 males, 19 females; mean age = 24.92 years; Std. Dev. = 5.95 years). All were right-handed, had normal or corrected-to-normal vision and hearing, had no history of neurological or psychiatric disorders, and had not participated in this experiment previously.

All participants were recruited for this study using the subject pools of the UCL Department of Psychology and the Institute of Cognitive Neuroscience. Participants were reimbursed financially for their time (£7.50 for one hour) and provided informed written consent prior to participating. All procedures were approved by the UCL Research Ethics Committee (ICN-AH-PWB-3-3-2016c).

*Procedure*

Pairs of participants arrived at the same time and were asked to introduce themselves to each other and choose a 'team name' together; they were told they would be competing against other teams who had previously participated in the experiment. This was done to introduce a prosocial collaborative mind-set during the task. One participant was assigned the role of Leader and the other of the Follower; they stood side-by-side facing the screen (Leader/Follower locations were counterbalanced). Magnetic motion-trackers

(Polhemus Liberty, Colchester Vermont) were fixed to the right hand and forehead of each participant. The hand markers allowed participants to control a hand icon in the augmented-reality environment and move objects (akin to a 3D mouse-pointer) (Figure 3.1A). Participants were instructed to move blocks from one table to another in a specified order (Figure 3.1B). The augmented-reality environment and experimental sequence were implemented in Vizard (WorldViz, Santa Barbara, CA). The study had three phases: familiarisation, experimental trials and the final check trials. The details of these phases are summarised in Table 3.1 below.

**Table 3.1.** Phases of the experiment

| | Explicitly instructed to copy trajectory of demonstration? | | | | | |
|---|---|---|---|---|---|---|
| Phase | Leader | Follower | Follower's eyes during demonstration | Leader's eyes during Follower's movement | Trajectory heights | Number of trials |
| Familiarisation | No | No | Closed | Open | Low / Med / High | 3 |
| Leader practice | Yes | N/A | N/A | N/A | Low / Med / High | 3 |
| Experimental | Yes | No | Closed | Open / Closed | Low / Med / High | 18 |

In the familiarisation phase, participants practiced moving blocks in the augmented-reality environment. At the start of each trial a (computerised) voice command instructed the Follower to close his/her eyes. The Leader then saw a demonstration of three or five blocks being moved in a specific order from one table to another (Figure 3.2A). Then the Follower heard a voice command to open their eyes, and the Leader demonstrated the block-movement task to the Follower (Figure 3.2B). Finally, the Follower was asked to move the blocks in the same order to the final table (Figure 3.2C). Both participants then saw a joint score based on accuracy (moving blocks in the right order) and timing (moving quickly) (Figure 3.2D).

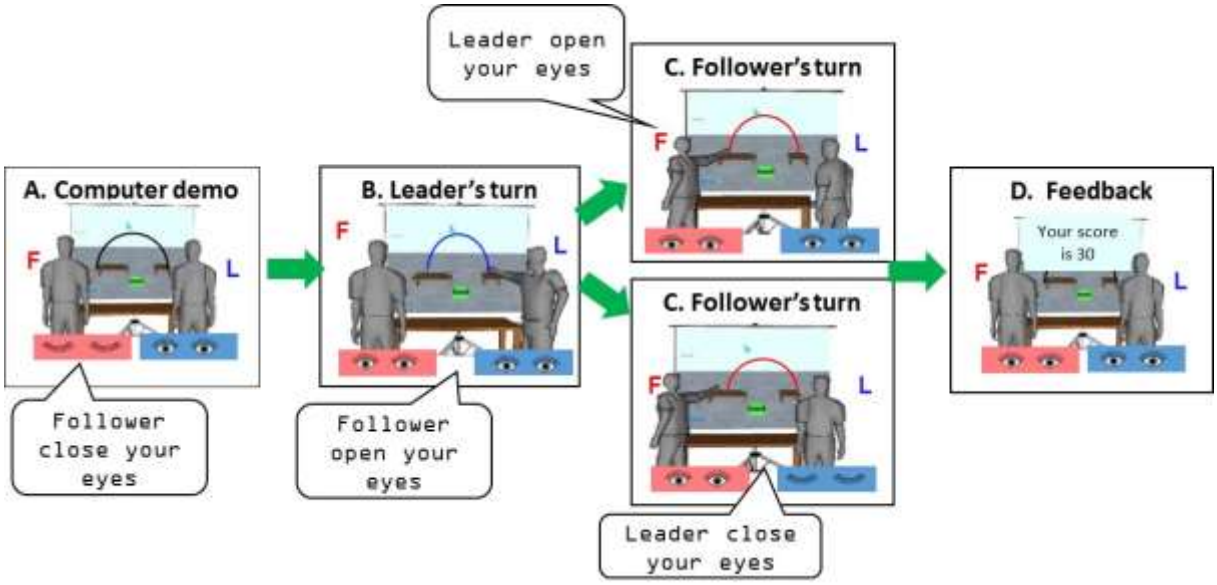


**Figure 3.2. Trial timeline.** **A.** The Follower closes their eyes while the Leader watches the computer demonstration. **B.** The Leader demonstrates, while the Follower watches. **C.** The Follower moves the blocks (The Leader's eyes can be open or closed). **D.** Participants see a joint score which rewards accuracy and speed. Speech bubbles throughout show computerised voice commands.

After three familiarisation trials, the experimental phase started. On the first trial, after the Follower closed their eyes, the Leader read an on-screen message with an additional 'secret' instruction to explicitly follow the trajectory demonstrated by the computer[7]. The computer demonstration then showed the blocks moving using a low, medium, or high trajectory, and the Leader was instructed to copy both the block order and the trajectory when demonstrating to the Follower. Leaders who failed to follow these instructions or shared this secret information with the Follower were excluded (see Data Analysis below). On half the trials, prior to the Follower's turn the Leader was instructed to close their eyes. On the other half the Leader was instructed to keep their eyes open. Thus, the ability of the Leader to monitor the Follower's movement was manipulated. All pairs completed 18 experimental trials (with three movement heights and the watched/unwatched conditions, each repeated thrice). The Leader's eyes were open or closed in blocks of three trials (with the order of watched blocks vs unwatched blocks counterbalanced across pairs). Participants then completed the final phase of six trials where both the Leader and Follower were explicitly told to follow the trajectory to

[7] The exact wording of the instruction was as follows: "You should follow the same PATH as the demonstration. This means you should move the pieces along the same path (reaching the same height) as the demonstration! However, you should NOT share this instruction with your teammate".

enable us to check that they understood this idea. Following the block-moving task, participants individually completed the following questionnaires:

(i) Rapport questionnaire: Six-item survey of their feelings of rapport towards each other
(ii) Autism Spectrum Quotient (AQ) questionnaire (Baron-Cohen et al., 2001) which measures the extent of autistic traits in adults
(iii) Interaction Anxiousness Scale (Leary, 1987) which measures social anxiety, and
(iv) Rosenberg Self-Esteem Scale (Rosenberg, 1965) which measures self-esteem.

Participants also completed a written debrief in which they were asked what they thought the purpose of the experiment was, and whether they noticed the differences in the trajectory heights prior to being explicitly told about them. Further, they were asked to self-report their ethnicity and their level of familiarity with their partner and the experimenter. In addition to tracking motion using a motion-capture system, we also recorded videos of the participants performing the task. This allowed us to verify that they kept their eyes closed and refrained from talking when required to do so, ensuring that the social manipulation worked.

*Data Analysis*

Our primary analysis focused on a single parameter: the maximum heights reached by the Leader and the Follower in each trial in the Full phase. Since each trial involved moving three or five blocks, we believe peak height is the most salient measure of whether movement trajectory was copied. To normalise for individual differences between participants, the Pearson correlation coefficient (R) was calculated between the computer demonstration and the height reached by the Leader, and between the heights reached by the Leader and the Follower in each trial. These were used to estimate the imitation fidelity across trial types.

*Exclusion criteria for the pilot experiment*

Dyads were excluded for the following reasons:

(i) Data was not recorded owing to equipment failure or failure in the task software

(ii) In the Full trials if the Leader failed to copy the computer demonstration with high fidelity (defined as having an R value of 0.5 or higher) after having been explicitly instructed to do so

(iii) In the Final check trials if the Follower failed to copy the Leader with high fidelity (R of 0.5 or higher) after having been explicitly instructed to do so

(iv) The Leader revealed the secret instruction (to copy the trajectory demonstrated by the computer) to the Follower during the full trials

(v) The Follower specifically asked the Leader about the path or trajectories they were demonstrating

(vi) Either the Leader or the Follower failed to follow instructions to close their eyes at various points in the trial.

### *3.3.2. Results*

*Overall peak height*

Followers tended to imitate the Leaders' trajectory with high fidelity during the Experimental trials, despite not being asked to do so explicitly (Figure 2.3A). A one-sample t-test showed a statistically-significant correlation between the heights reached by the Leader and the Follower across all trials [N = 22, Mean R value = 0.30, Std. Dev. = 0.43, t(21) = 3.24, p = 0.04].

*Imitation fidelity when being watched vs when not being watched*

Our second analysis tested the core experimental question: do participants imitate with more fidelity when they know they are being watched, compared to when they are not watched? Figure 3.3C shows the peak heights reached by the Leader and the Follower for one sample dyad. Across all participants, we compared the correlation between the peak heights reached by the Leader and the Follower in trials where the Leader was watching the Follower make their movements and the trials where the Leader was not watching. A paired-sample t-test [N = 22] showed that these R-values were higher when the Leader was watching [Mean R value = 0.43, Std. Dev. = 0.36] than when the Leader was not watching [Mean R value = 0.19, Std. Dev = 0.55] and that this effect was statistically significant [t(21) = 2.96, p = 0.008]. The overall effect size was 'medium', with Cohen's *d* =

0.631 (Cohen, 1992)[8]. This supports our hypothesis that participants will imitate to a greater extent when they know they are being watched by their partner when compared with a situation where they know their partner cannot see them.

[8] This was calculated for the paired-samples t-test using the formula

$$d = \frac{|m_1 - m_2|}{\sqrt{(s_1^2) + s_2^2 - (2rs_1s_2)}}$$

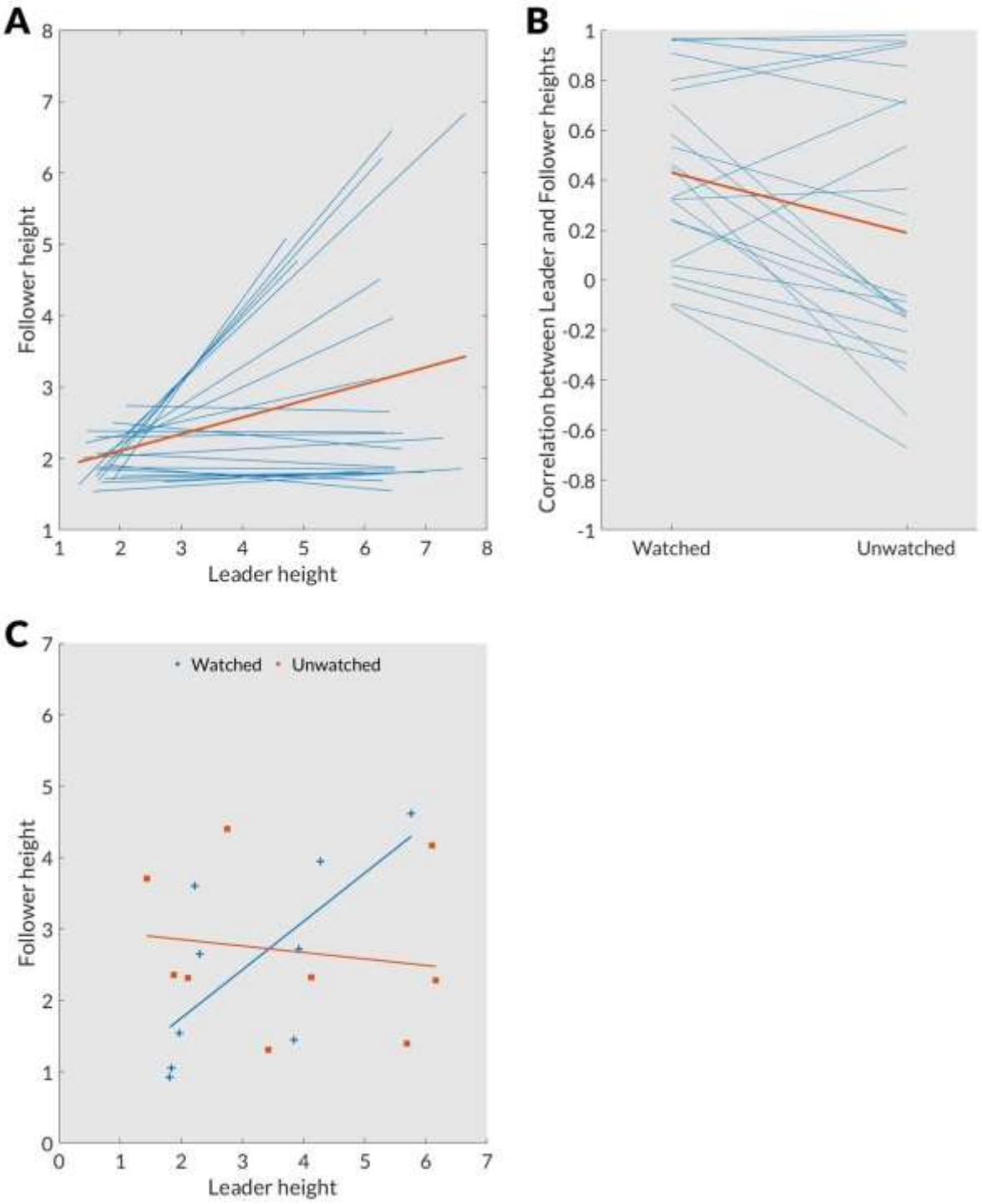


**Figure 3.3. Pilot Experiment (N = 22) A. Overall imitation pattern** Line of best-fit for the correlation between Leader heights and Follower heights for each dyad (blue lines) and the group as a whole (heavy orange line). **B. Correlations:** The R values representing the correlation between the Leader and Follower heights are shown for each dyad and for the whole group (heavy orange line) for trials where the Leader watches the Follower make their movements and trials where the Leader does not watch. **C. Results for one sample dyad:** The heights reached by the Leader and Follower in one dyad (#23) and respective trendlines across both the Watched and Unwatched conditions.

*Exploratory analysis of movement duration*

We also conducted an exploratory analysis of the relationship between how quickly the Follower moved and how quickly the Leader moved. In each trial the participants moved either three or five blocks. For each trial we calculated the average time taken to move one block for both the Leader and the Follower; for each dyad we used this to calculate the correlation between average movement duration of the Leader and the Follower. A one-sample t-test showed a statistically significant correlation between the average movement duration of the Leader and the Follower [N = 22, Mean R value = 0.24, Std. Dev. = 0.31, t(21) = 3.69, p = 0.001]. However, the correlation in movement durations in the Watched trials [N = 22, Mean R value = 0.23, Std. Dev. = 0.40] and the Unwatched trials [N = 22, Mean R value = 0.29, Std. Dev. = 0.30] were not statistically significantly different from each other according to a paired-sample t-test [t(21) = -0.65, p = 0.522].

This suggests that Followers tended to move more slowly when Leaders moved slowly, and this effect persisted in both the Watched and the Unwatched conditions.

*Multi-level regression analysis of Follower height*

In addition to the analysis above, a more detailed exploration of the factors that predict the height reached by the Follower was performed, using a multi-level regression analysis. It is important to note that the previous results in this chapter used the Pearson correlation coefficient (R) as the dependent variable; the R value captures the relationship between the Leader's and the Follower's heights. In this exploratory analysis we used the Follower's height as the dependent variable, which is less subtle and does not account for variations in individual Leader's demonstrations. Having undertaken the analysis, however, it is presented below for the sake of completeness. The factors considered in this multi-level regression included:

(a) The dyad number, to account for differences between pairs
(b) The trial number, to account for order effects in the trials
(c) The height demonstrated by the Leader
(d) The average time taken by the Leader to move one block in each trial
(e) Whether the Leader watched the Follower when they were making their moves

We also considered two interaction effects, both between whether the Follower was watched during their move and

(f) The height demonstrated by the Leader (c) x whether the Follower was watched or not during their turn (e)

(g) The average time taken by the Leader to move one block (d) x whether the Follower was watched or not during their turn (e)

Using the Enter method we found that a model incorporating only the main effects (a-e) accounted for 12.6% of the variance in the Follower's height [$F(5,390) = 11.26$; $p<0.001$]. A model incorporating the main effects as well as the two interaction effects (f-g) explained 15.3% of the variance in the Follower's height and this $R^2$ change was significant [$\Delta F(2,388) = 6.13$; $p = 0.002$]. The following table (Table 2.2) summarises the two models. The table also includes the analysis of variance (ANOVA) and the regression coefficients for the second model.

**Table 3.2. Pilot experiment: Regression model to predict Follower height**

**Summary of Models**

| Model | R | R Square | Adjusted R Square | Std. Error of the Estimate | Change Statistics | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | R Square Change | F Change | df1 | df2 | Sig. F Change |
| 1 | .355[a] | 0.126 | 0.115 | 1.10175 | 0.126 | 11.257 | 5 | 390 | 0.000 |
| 2 | .391[b] | 0.153 | 0.138 | 1.08753 | 0.027 | 6.134 | 2 | 388 | 0.002 |

a. Predictors: (Constant), Leader time per block, Watched/Unwatched, Trial ID, Leader height, Dyad number

b. Predictors: (Constant), Leader time per block, Watched/Unwatched, Trial ID, Leader height, Dyad number, Leader height x Watched / Unwatched, Leader time x Watched/Unwatched

**Analysis of Variance (ANOVA)[a]**

| | Sum of Squares | df | Mean Square | F | |
|---|---|---|---|---|---|
| Regression | 82.831 | 7 | 11.833 | 10.005 | .000[b] |
| Residual | 458.897 | 388 | 1.183 | | |
| Total | 541.728 | 395 | | | |

a. Dependent Variable: Follower height

b. Predictors: (Constant), Leader time x Watched/Unwatched, Dyad number, Trial ID, Leader height, Leader time per block, Leader height x Watched / Unwatched, Watched/Unwatched

**Regression Coefficients[a]**

| | Unstandardized Coefficients | | Standardized Coefficients | | |
|---|---|---|---|---|---|
| | B | Std. Error | Beta | t | Sig. |
| (Constant) | 1.288 | 0.290 | | 4.447 | 0.000 |
| Dyad number | -0.006 | 0.006 | -0.053 | -1.113 | 0.266 |
| Trial ID | -0.008 | 0.011 | -0.034 | -0.719 | 0.473 |
| Watched/Unwatched | 0.685 | 0.219 | 0.586 | 3.129 | 0.002 |
| Leader height | 0.211 | 0.035 | 0.287 | 6.002 | 0.000 |
| Leader time per block | 0.146 | 0.043 | 0.171 | 3.407 | 0.001 |
| Leader height x Watched / Unwatched | -0.059 | 0.035 | -0.203 | -1.667 | 0.096 |
| Leader time per block x Watched/Unwatched | -0.117 | 0.042 | -0.469 | -2.795 | 0.005 |

a. Dependent Variable: Follower height

There was no main effect of dyad or trial number. However, the height reached by the Leader, the time taken by the Leader, and whether the Follower was Watched or Unwatched were all statistically significant predictors of the height reached by the Follower. The correlation coefficients suggest that, holding all other factors constant:

(a) Follower's height *increases* in Watched trials over Unwatched trials
(b) Follower's height *increases* as Leader's demonstrated height increases
(c) Follower's height *increases* as Leader's movements become slower
(d) A slow movement by the Leader *increases* the Follower's height to a greater extent in the Unwatched trials, when compared with the Watched trials

*Exploratory analysis of questionnaire data*

As outlined above (Section 3.3.1) above, our questionnaires measured the following individual traits: AQ, social anxiety, self-esteem. In addition, we also collected data on level of education, and self-reported ethnicity for each participant, feelings of rapport of each participant towards their interaction partner, and information on how familiar they

were (if at all) with each other prior to the experiment. These showed that none of the participants were outliers on these traits.

We attempted to ascertain whether the degree to which Follower's movements correlated with Leader's movements (as measured by the Pearson's correlation coefficient, R) could be predicted by these individual traits or dyad features. Using the Enter method we found that a multi-level regression model incorporating these factors was not a significant predictor of overall correlation [$F(7,14) = 1.31$; $p = 0.316$]. There is therefore no evidence (in this study) of a relationship between these traits and a participant's propensity to imitate. However, our study was not designed to examine this issue and thus the lack of an effect should not be taken to mean there is no relationship.

## 3.4. Preregistered replication

### *3.4.1. Materials and Methods*

The pre-registered replication closely followed the procedures outlined for the pilot study above. A power-analysis in G*Power (Faul et al., 2007) showed that with a Cohen's *d* of 0.63, it would be sufficient to test 29 dyads to detect such an effect with a power of 90%. We planned to test until we collected data from 30 valid dyads based on the pre-analysis exclusion criteria.

The exclusion criteria used were the same as in the previous pilot study (Section 3.3.1), with one addition: participants were also excluded if they were familiar with each other prior to the study. The pre-analysis checks were done using demographic and self-reported data from participants, checking the independent variables in the data, and checking the data from the Final Check trials during the data collection phase, to ensure we were able to count the number of valid dyads tested. The data from the Experimental phase was otherwise untouched during the data collection phase, and the analysis strategy was pre-registered prior to data analysis (https://osf.io/ezj8g/).

*Participants*

A total of 80 naïve participants were tested in 40 pairs to collect data from 30 valid dyads (42 females, 18 males; mean age = 24.17 years; SD = 6.58 years). The same criteria were used as in the previous study, with the addition of the requirement that participants be unfamiliar with each other prior to the commencement of the study. No confederates were used during this study.

*Procedure*

The procedures for this study were identical to the pilot study reported above, except that each trial had three or four blocks per trial (instead of three or five blocks per trial in the pilot study). The data analysis strategy was outlined in the preregistration and is identical to the pilot study in Section 3.3.

### *3.4.2. Results*

*Overall peak height*

As in the pilot study, we found that Followers tended to imitate the Leader's trajectories with high fidelity during the Experimental trials, without having been asked to do so (Figure 3.4A). A one-sample t-test showed that the correlation between the Leader heights and the Follower heights across all trials is statistically significant [N = 30, Mean R value = 0.38, Std. Dev. = 0.46, $t(29) = 4.52$, $p < 0.001$].

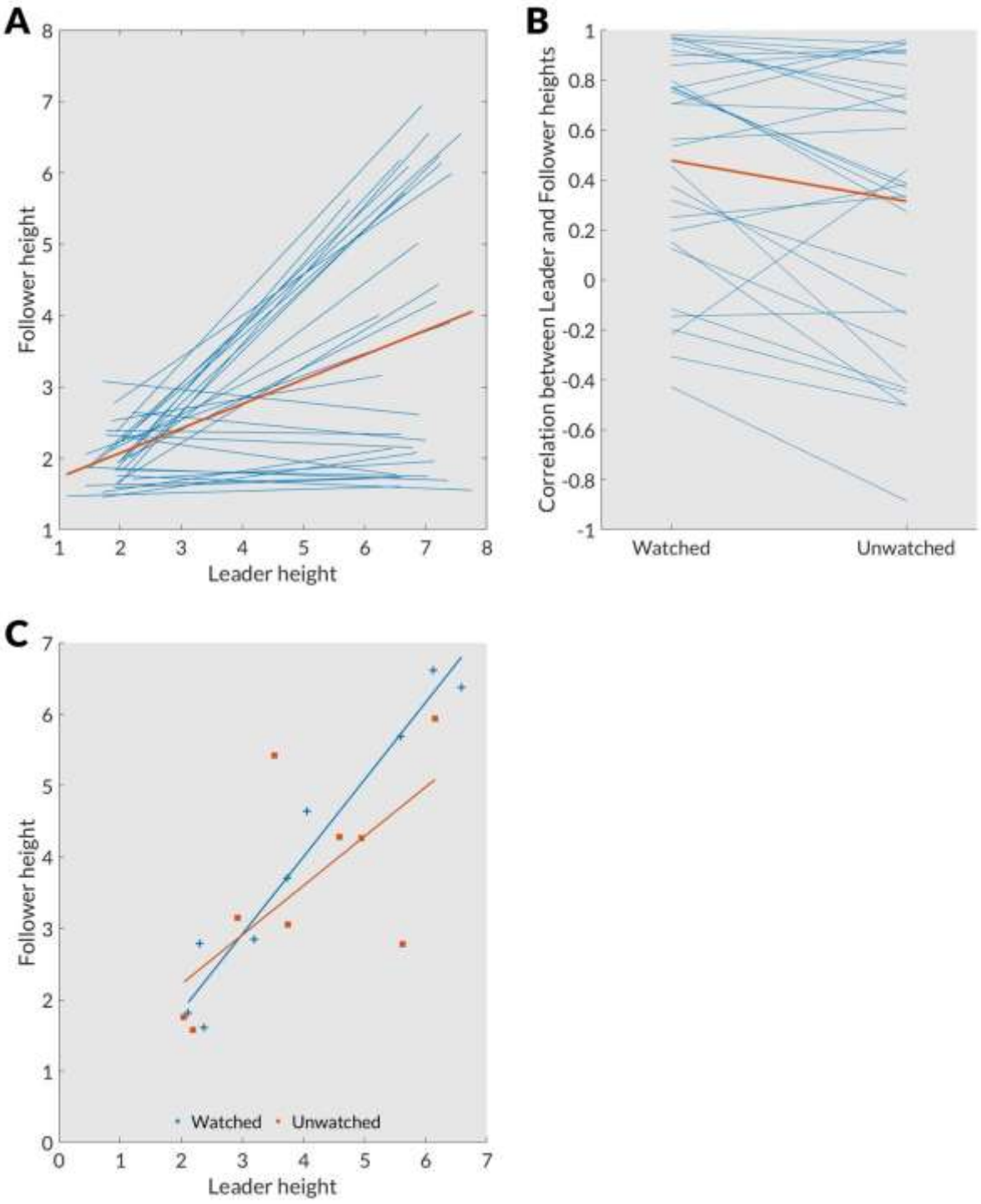


**Figure 3.4. Preregistered Replication (N = 30) A. Overall imitation pattern:** Line of best-fit for the correlation between Leader heights and Follower heights for each dyad (blue lines) and the group as a whole (heavy orange line). **B. Correlations:** The R values representing the correlation between the Leader and Follower heights are shown for each dyad and for the whole group (heavy orange line) for trials where the Leader watches the Follower make their movements and trials where the Leader does not watch. **C. Results for one sample dyad:** The heights reached by the Leader and Follower in one dyad (#20) and respective trendlines across both the Watched and Unwatched conditions.

*Imitation fidelity when being watched vs when not being watched*

When it comes to the core experimental question of whether participants imitate with greater fidelity when being watched, we find results similar to those in the pilot study. Figure 3.4C shows the peak heights reached by the Leader and the Follower in each of the 18 trials for one sample dyad. Across all participants we compared the correlation between the Leader and the Follower heights in the Watched trials and the Unwatched trials. A paired-sample t-test [N = 30] showed that these R values were higher when the Leader watched the Follower [Mean R value = 0.48, Std. Dev. = 0.45] than when the Leader did not watch the Follower [Mean R value = 0.32, Std. Dev. = 0.55] and that this difference was statistically significant [$t(29) = 2.84$, $p = 0.008$].

This supports the social-signalling hypothesis of imitation, with participants imitating a naïve teammate with greater fidelity when they know they are being watched by their interaction partner, when compared with a situation where they know their partner is not watching them.

*Multi-level regression analysis of Follower height*

As outlined in the preregistration, we also conducted a multi-level regression analysis on whether Follower height could be predicted based on several factors, including:

(a) The dyad number, to account for differences between pairs
(b) The trial number, to account for order effects in the trials
(c) The height demonstrated by the Leader
(d) The average time taken by the Leader to move one block in each trial
(e) Whether the Leader watched the Follower when they were making their moves

We also considered two interaction effects, both between whether the Follower was watched during their move and

(h) The height demonstrated by the Leader (c) x whether the Follower was watched or not during their turn (e)
(i) The average time taken by the Leader to move one block (d) x whether the Follower was watched or not during their turn (e)

Using the Enter method we found that a model incorporating only the main effects (a-e) accounted for 19.3% of the variance in the Follower's height [$F(5,534) = 25.49$;

$p < 0.001$]. Incorporating the two interaction effects (f-g) did not significantly improve the performance of the model [$\Delta F(2,532) = 0.276$; $p = 0.759$] and therefore only the model with the main effects is considered below. The following table (Table 2.3) summarises the models, along with the ANOVA and the regression coefficients of the first model. This model suggests that the height reached by the Leader was predictive of the Follower's height. The main effect of trial number suggests that the Follower's heights increased as the experiment progressed, while the main effect of dyad number is indicative of individual differences between Followers in various dyads. There was no main effect of being Watched on the Follower's height. It is important to note that this analysis focuses on the height reached by the Follower as the dependent variable, since it is done on a trial-by-trial basis; however, our key variable of interest in this chapter is the relationship between the Leader's height and the Follower's height, rather than the Follower's trajectory alone. Nevertheless, this analysis is presented below for the sake of completeness.

**Table 3.3. Pilot experiment: Regression model to predict Follower height**

**Summary of Models**

| Model | R | R Square | Adjusted R Square | Std. Error of the Estimate | Change Statistics | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | R Square Change | F Change | df1 | df2 | Sig. F Change |
| 1 | .439a | 0.193 | 0.185 | 1.31996 | 0.193 | 25.493 | 5 | 534 | 0.000 |
| 2 | .440b | 0.194 | 0.183 | 1.32175 | 0.001 | 0.276 | 2 | 532 | 0.759 |

a. Predictors: (Constant), Leader time per block, Watched /Unwatched, Dyad number, Leader height, Trial ID

b. Predictors: (Constant), Leader time per block, Watched /Unwatched, Dyad number, Leader height, Trial ID, Leader height x Watched / Unwatched, Leader time per block x Watched / Unwatched

**Analysis of Variance (ANOVA)[a]**

| | Sum of Squares | df | Mean Square | F | |
|---|---|---|---|---|---|
| Regression | 222.077 | 5 | 44.415 | 25.493 | .000b |
| Residual | 930.383 | 534 | 1.742 | | |
| Total | 1152.460 | 539 | | | |

a. Dependent Variable: Follower height

b. Predictors: (Constant), Leader time per block, Watched /Unwatched, Dyad number, Leader height, Trial ID

**Regression Coefficients[a]**

| | Unstandardized Coefficients | | Standardized Coefficients | | |
|---|---|---|---|---|---|
| | B | Std. Error | Beta | t | Sig. |
| (Constant) | 0.918 | 0.294 | | 3.120 | 0.002 |
| Dyad number | 0.020 | 0.005 | 0.143 | 3.681 | 0.000 |
| Trial ID | 0.027 | 0.011 | 0.096 | 2.359 | 0.019 |
| Watched /Unwatched | 0.000 | 0.057 | 0.000 | -0.007 | 0.995 |
| Leader height | 0.339 | 0.034 | 0.397 | 9.929 | 0.000 |
| Leader time per block | -0.021 | 0.041 | -0.021 | -0.509 | 0.611 |

a. Dependent Variable: Follower height

*Exploratory analysis of movement duration*

In addition to the preregistered analyses above, we also conducted an exploratory analysis of the relationship between how quickly the Leader and the Follower moved in each dyad. As in the pilot experiment, we calculated the correlation between the average time taken to move one block by the Leader and the Follower in each dyad, across all trials, and in only the Watched and Unwatched trials separately. A one-sample t-test showed a statistically significant correlation between the average movement duration of

the Leader and the Follower [N = 30, Mean R value = 0.21, Std. Dev. = 0.33, t(29) = 3.38, p = 0.021]. However, the correlation in movement durations in the Watched trials [N = 30, Mean R value = 0.22, Std. Dev. = 0.48] and the Unwatched trials [N = 30, Mean R value = 0.23, Std. Dev. = 0.35] were not statistically significantly different from each other according to a paired-sample t-test [t(29) = -0.10, p = 0.918]. This suggests that Followers tended to move more slowly when Leaders moved slowly, and this effect persisted in both the Watched and the Unwatched conditions.

*Exploratory analysis of questionnaire data*

Similar to the analysis done in the pilot experiment (Section 3.3.3) we conducted an exploratory analysis of whether the correlation between the Leader and the Follower's movements could be predicted by traits and demographic data collected on each Follower. A multi-level regression model incorporating the Follower's self-reported social anxiety, self-esteem, the average rapport between the Leader and the Follower, the Follower's AQ, level of education, and whether the Leader and the Follower belonged to the same ethnic group was not found to be a significant predictor of the degree to which Follower's movements correlated with Leader's movements [F(6,23) = 1.19; p = 0.346]. However, as discussed earlier, this was not the primary purpose of our study, and thus a lack of an effect could be due to the study being underpowered, rather than a lack of an underlying relationship.

## 3.5. Overall discussion

The two studies in this chapter aim to test the social-signalling hypothesis of imitation, which posits that one of the reasons that humans imitate others is to send a social signal. In this study we found clear evidence that adults imitate with greater fidelity when they know they are being watched by an interaction partner.

These two studies advance previous work on imitation in several important ways. By undertaking an exploratory pilot study, and pre-registering the analysis for the replication study, we can be confident that our results are robust, and not the outcome of testing multiple hypotheses until results were found (or p-hacking). In the replication, by using two naïve participants (rather than having a confederate or experimenter demonstrate the action sequence) we can avoid experimenter effects. Third, using augmented reality allowed for precise capture of motion kinematics. Fourth, the 'feeling of being watched' was manipulated at an abstract level by voice signals instructing the Leader to open or close their eyes. This allows us to rule out several alternative

interpretations of differences in imitative behaviour being due to arousal from direct gaze (Senju & Johnson, 2009), due to social facilitation (Zajonc, 1965), or due to varying levels of anxiety or attention (Heyes, 2017). In our study both participants stood side-by-side throughout the experiment and therefore differences in eye-gaze and social-facilitation cannot explain the present results. Participants were asked to move quickly and accurately, implying that a straight trajectory was more efficient than using a curved one. Further, if participants felt more anxious during trials when they know they are being watched (Zajonc, 1965), we would expect them to move faster, straighter and lower. Yet, participants' actual movements were higher and did not differ in speed. Finally, since the demonstration phases were identical in both the watched and unwatched conditions there cannot be systematic differences in attention during the demonstration phase, allowing us to rule out attentional explanations.

Overall, our experimental design suggests that the 'being watched' effect does not arise from differences in arousal, social facilitation, anxiety, or attention. The remaining explanation is that participants imitate with greater fidelity to send a signal to their interaction partner. That is, these results support the claim that imitation can serve as a *social signal* (Farmer et al., 2018) and suggests that this signal is enhanced when senders know the recipient can receive it. This is compatible with the STORM model which posits that basic mechanisms for observing and performing actions can be modulated according to the scope and need to strengthen a social connection (Wang & Hamilton, 2012). The current data is also consistent with earlier work on emotion mimicry (Bavelas et al., 1986) and studies using video stimuli (Wang, Newport, et al., 2011). Note that the claim that imitation can be a social signal does not rule out the possibility that, in other contexts, imitation can also be used for social learning (Lyons et al., 2011), as many functions can coincide in this behaviour (Over & Carpenter, 2012).

The question of what participants are signalling remains open. If imitation is a social signal, then what is the content of the signal participants are sending? According to the affiliative account of imitation (see Section 1.2.3) we copy for some affiliative purpose, tending to imitate more when we are in the presence of a demonstrator (DiYanni et al., 2011; Nielsen & Blank, 2011). In this particular experiment an alternate explanation could be that Followers copy the Leader's movements in order to secure a higher score. At the start of the experiment participants are told that their score depends on how quickly and accurately they make their movements. There is a possibility that Followers may misinterpret this to think they should copy the Leaders trajectory to get a higher score—however, if this were true then the fidelity with which they imitate the Leader should not

vary based on whether they are watched or not. However, given the difference in imitative fidelity between the Watched and the Unwatched condition, this is less likely. What is possible, however, is that the Follower may be signalling to the Leader (in the Watched trials) that they are a good learner, or are working hard to raise the team's score. Our current paradigm was not designed to parse the content of the signal being sent, but in future work it would be interesting to test this by changing the cover story (by removing the score element for example) to see if the imitative behaviour persists when this specific sub-goal is eliminated.

There are also some limitations to our results. We cannot determine if Followers became consciously aware of the Leaders unusual trajectories at some point prior to being explicitly told about the trajectories (in the final phase)[9]. This study, therefore, does not distinguish between conscious and unconscious copying. Future studies could measure when (if ever) Followers become aware of the unusual trajectories and test if awareness modulates imitation fidelity. Second, this study set social imitation of kinematics within the context of a block-order learning task; a potential manipulation for future experiments would be to generate a paradigm without a learning objective, such as a task involving only natural conversation. Third, the rationality of the task was varied by manipulating the height of the demonstrated trajectory. That is, moving higher was always associated with making a more irrational move. In future studies it may be fruitful to consider paradigms where the rationality is manipulated by either including or excluding an obstacle, for example, to allow us to have the same movements for rational and irrational actions.

Our study also generates several possible directions for future research. First, if imitation is being used as a social signal, what message is being sent? Previous work has suggested that imitation signals a desire to affiliate (Chartrand & Lakin, 2013) but positive effects of being imitated are not always seen (Hale & Hamilton, 2016a; Verberne et al., 2015). Kinematic patterns can also signal informative intentions (McEllin et al., 2018) or confidence (Patel et al., 2012) which could be important here. It would also be interesting to understand the neural mechanisms of imitation as a social signal. The STORM model suggests that the interaction between gaze and imitation arises due to influence of medial prefrontal cortex on mirror neuron regions (Wang, Ramsey, et al., 2011; Wang & Hamilton, 2013). Combining this paradigm with brain imaging techniques such as

[9] We could not explicitly ask about this since we did not want to prime participants with questions about trajectories until the end of the experiment.

functional near-infrared spectroscopy (fNIRS) (Pinti, Tachtsidis, et al., 2020), will allow us to test the brain mechanisms involved while preserving the believability of the dyadic interaction[10]. Finally, our results suggest that some Followers imitated with greater fidelity than others, although we did not find links between the self-reported traits and propensity to imitate. Examining these individual differences could also be a productive avenue for further study.

## 3.6. Conclusions

We hypothesised that imitation functions as a social signal and would be modulated in line with its expected communicative capacity. A preregistered study of 30 pairs of naïve participants shows that participants tend to imitate the causally irrelevant kinematic features of their partner's movements, and imitate more when they know their partner can see them. This provides evidence for top-down social modulation of imitation (Wang & Hamilton, 2012) and for the use of imitation behaviour as a social signal to others.

[10] See Section 2.5 for a detailed discussion of the cover story used in the paradigm

## 3.7. Appendix: Questionnaires

*Rapport questionnaire*

This is a six-item questionnaire used to measure the participant's feeling of rapport towards each other, with participants rating their degree of agreement or disagreement with each statement on a seven-point Likert scale.

Please read each statement carefully, and rate how strongly you agree or disagree with the statement.

1. The interaction with my partner was very smooth

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

2. I felt rapport with my partner

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

3. I felt that the communication flow with my partner was easy

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

4. I felt very comfortable during the interaction

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

5. During the interaction I found it easy to express what I wanted to say

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

6. I felt that my partner could easily understand what I was thinking

   Strongly disagree 1 2 3 4 5 6 7 Strongly agree

*Familiarity questionnaire*

How well did you know your teammate before the game?

- Unfamiliar (did not know him/her at all)
- Somewhat familiar (casual acquaintance)
- Familiar (friend)
- Very familiar (long-time friend, roommate, partner, family member etc)

If you knew your teammate before the game, how long have you known each other?

[Text box]

If you knew your teammate before the game, how often have you met in the past week?

- Not met in the past week
- Met once
- Met more than once but not everyday
- Met everyday

How well did you know the experimenter before the game?

- Unfamiliar (did not know him/her at all)
- Somewhat familiar (casual acquaintance)
- Familiar (friend)
- Very familiar (long-time friend, roommate, partner, family member etc)

# Chapter 4. Neural correlates of imitation as a social signal

This study seeks to examine the neural correlates of imitative behaviour, in the context of it being deployed as a social signal. We examine whether participants recognise an action as irrational, the fidelity with which they imitate it, whether they encode the social availability of their interaction partner, and whether this social availability modulates their imitative fidelity.

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## 4.1. Abstract

The social-signalling hypothesis of imitation is supported by previous research showing that imitation fidelity is positively modulated by the social availability of an interaction partner (Krishnan-Barman & Hamilton, 2019). This study examines the neural correlates of this imitative behaviour, building on two previous studies detailed in Chapter 3. With simultaneous neuroimaging (hyperscanning) using fNIRS we obtained brain signals from the right hemisphere, centred on the right TPJ, and extending into the right IPL, in 20 pairs of naïve participants, randomly assigned the roles of Leader and Follower using our dyadic block-moving task (see Section 2.5.1). Leaders demonstrated the movement of blocks in a simple task to Followers who then moved the blocks in the same order. Two independent variables were manipulated: the height of trajectory demonstrated by the Leader, and whether the Leader's eyes were open or closed during the Follower's turn. Replicating previous results, we found that Followers imitated the Leader's irrational movements with greater fidelity when they knew they were watching them. When watching irrational actions, Leaders showed greater activation in the right TPJ when watching Followers make high trajectories; Followers meanwhile showed activation in the right IPL that was parametrically modulated by the rationality of viewed movements. Followers encoded the social availability of the Leader via deactivation in the right TPJ and right IPL. Further, general linear models (GLMs) that incorporated an interaction partner's neural activity (made possible by hyperscanning) in addition to the behavioural signals from both participants were found to be a better fit at predicting neural activity in an individual. In addition to showcasing the utility of hyperscanning, this study offers support for the social-signalling hypothesis of imitation.

## 4.2. Introduction

Copying another person's behaviour is often inefficient, particularly when their actions are irrational or irrelevant. Yet, humans copy irrational actions prolifically while our nearest primate relatives do not (Subiaul, 2016; Whiten, 2011). Several explanations have been advanced for copying behaviour, including that it is a developmental side-effect of domain-general learning (Darda & Ramsey, 2019; Heyes, 2017) or that it enables us to build new skills (Flynn & Smith, 2012). It has also been observed that being copied builds rapport and increases our liking of others (Chartrand & Bargh, 1999; Lakin & Chartrand, 2003; Stel & Vonk, 2010), and this effect persists even when the mimicking agent is a computer or a virtual avatar (Bailenson & Yee, 2005; Suzuki et al., 2003). Imitation is also seen to increase pro-social behaviour such as helping others (Müller et al., 2012) or increasing the tips that waitresses receive (van Baaren et al., 2003).

We are particularly interested in the question of whether the affiliative effect of imitation is an epiphenomenon, or one of its aims. If someone liking us more when we imitate them is merely a happy side effect, then our imitative behaviour should not be modulated by social context. That is, we should imitate all people relatively equally regardless of whether they are observing us or not. Here the evidence is mixed, with some studies suggesting that imitative behaviour is not influenced by whether we are being watched or not (Lyons et al., 2007; Whiten et al., 2016), while others have found that we tend to imitate a demonstrator with greater fidelity when they are watching us (DiYanni et al., 2011; Marsh et al., 2019; Nielsen & Blank, 2011). The STORM model (Wang & Hamilton, 2012) makes the specific claim that we imitate in order to affiliate building on the hypothesis that imitation functions as a social glue (Farmer et al., 2018; Lakin et al., 2003).

In our previous work we tested this social-signalling hypothesis of imitation by examining whether social availability, that is the degree to which the recipient is available to process a social signal, would influence imitation (Chapter 3). We found that people tended to imitate their interaction partner with greater fidelity when they know they are being watched by their partner. In this chapter we seek to extend our previous work by examining the neural correlates of imitation in a social context. If the social-signalling hypothesis of imitation is valid, then in a dyadic interaction where a demonstrator makes an irrational movement and a responder then acts following the demonstration, we would expect the following to occur:

(a) The responder should recognise (either consciously or subconsciously) that the action demonstrated is irrational; that is, brain activity should be different in the rational and irrational conditions
(b) The responder should recognise the social availability of the demonstrator, as reflected in differential brain activity between the watched and unwatched conditions
(c) The responder should copy the demonstrators' actions, including potentially the demonstrator's irrational actions
(d) The degree to which the responder copies the irrational action should be related to the social availability of the demonstrator; that is, they should copy the irrational action more closely when the demonstrator is watching them

Here, (c) and (d) are behavioural responses that we have systematically tested in two previous studies using pairs of naïve participants (detailed in Chapter 3). We used our dyadic block-moving task (see Section 2.5.1) where in each pair one participant was randomly assigned the role of Leader and demonstrated the order in which blocks were to be moved to a Follower, using irrationally high trajectories in some trials. This task builds on a range of paradigms used in the study of mimicry and on evaluating rationality (Forbes & Hamilton, 2017; Gergely et al., 1995; Krishnan-Barman et al., 2017; Marsh et al., 2015; Oliver et al., 2017; Wild et al., 2012). In the pair of studies detailed in Chapter 3 we found that Followers reliably imitated the Leader's high trajectories and did so with greater fidelity in trials where they knew the Leader was watching them, versus trials where they knew the Leader had their eyes closed. In the current study we will seek to replicate these results a third time. Testing (a) and (b) requires examining neural activity during the study, to see if brain activation differs based on the rationality of the demonstrated action, and between the watched and unwatched conditions.

### *4.2.1. Dyadic experiments using fNIRS*

Functional near-infrared spectroscopy (fNIRS) is a relatively novel brain-imaging technique that measure changes in concentration of oxygenated and deoxygenated haemoglobin in the cortical surface (Scholkmann et al., 2014). As detailed in Chapter 2 (see Section 2.4) fNIRS is a safe, low-cost, non-invasive imaging technology that was originally developed to study neonates in intensive care units and has since been adapted for wider non-clinical use. The signals obtained from fNIRS are similar to the BOLD signal used in fMRI and the method yields comparable results (Noah et al., 2015).

Much of the existing research on the neural correlates of imitation has relied on PET (Decety et al., 1997, 2002; Grafton et al., 1996; Grezes, 1998; Krams et al., 1998), MEG (Nishitani & Hari, 2000, 2002) or fMRI (see Caspers et al., 2010 for a review), which are sensitive to motion artefacts and have physical limits on the range of possible movement. Consequently these have been limited to using finger-tapping tasks (Brass et al., 2001; Iacoboni, 1999; Koski et al., 2002; Mengotti et al., 2012; Tanaka et al., 2001), or grasping and hand configuration tasks (Buccino et al., 2004; Grafton et al., 1996). In contrast fNIRS allows for a much wider range of movement (Pinti, Tachtsidis, et al., 2020).

Another limitation of many of the finger-tapping or grasping experiments is that they use 'isolation paradigms' (Becchio et al., 2010) which focus on individual actions on their own (a first-person account), or more typically, invite us to imagine other people's behaviour or mental states (a third-person account). Implicit in studies using third-person neuroscience is the view that imagining a social interaction generates the same behaviour and neural activation as actually engaging in one. Neuroimaging studies of imitation, for example, almost exclusively use video stimuli (Caspers et al., 2010) while neuroimaging studies of other types of social interaction study participants who are physically isolated in the scanner but are asked to interact with a real or fictitious partner outside the scanner (Decety et al., 2004; Gallagher et al., 2002) . However, a growing body of research in social neuroscience is focused on a second-person approach, where participants engage in real-time social interaction with partners (Schilbach et al., 2013). While more complex to implement, this approach adheres to the idea that social interaction is more than the sum of its parts (De Jaegher et al., 2010; Hasson et al., 2012); that is, the neural activations engendered by social interaction is more than that generated during similar activities undertaken on one's own.

### *Hyperscanning*

As paradigms are developed to allow two people to engage in social interactions, scientists have become interested in simultaneously recording neural data from both subjects (Konvalinka & Roepstorff, 2012). This technique, known as hyperscanning (Montague et al., 2002), allows us to investigate the inter-brain links during social interaction as well. The first mentions of hyperscanning come from a study by Duane and Behrendt (1965) who recorded EEG simultaneously in pairs of twins; however the idea fell out of vogue and it was only in the last two decades that it has been employed again. Many hyperscanning studies use EEG, fMRI or a combination of both (Babiloni & Astolfi, 2014; Dumas et al., 2011; Koike et al., 2015), while the usage of fNIRS for hyperscanning is only

just beginning to gain popularity (Cheng et al., 2015; Cui et al., 2012; Dommer et al., 2012; Funane et al., 2011; Holper et al., 2012; Jiang et al., 2012; Nozawa et al., 2016; Osaka et al., 2014; Scholkmann et al., 2013).

Development of this new technique has also led to the development of a range of new analytical techniques that extend our analysis of single brains to dyadic or group interactions. Broadly, these analyses focus on estimating inter-brain synchrony via measuring the correlation between two signals (their similarity in the temporal domain) or the coherence (similarity in the frequency domain); Czeszumski et al. (2020) provide a detailed review of analysis techniques for hyperscanning used in different modalities. One frequently used technique is wavelet transform coherence (WTC). This was initially developed to analyse geophysical time series (Grinsted et al., 2004), but was used in one of the earliest fNIRS hyperscanning studies (Cui et al., 2012); since then it has been used widely in this field (Cheng et al., 2015; Dommer et al., 2012; Holper et al., 2012; Jiang et al., 2012; Liu et al., 2016; Osaka et al., 2014). These analyses effectively tell us how similar the signals from two interacting brains are to each other. Similarities could arise because of the social interaction between the two participants, but it could also simply be because both are receiving the same stimuli in the same environment simultaneously, known as the 'problem of common input' (Burgess, 2013; Hamilton, 2020). These measures of inter-brain coherence also do not provide information on how this coherence is related to participant's behaviour during the task.

*Mutual prediction hypothesis*

An alternative analytical approach has been advanced by Kingsbury and their colleagues (2019) who modelled neural activation in pairs of interacting mice using a mutual prediction framework. They recorded neural activations in the prefrontal cortex in mice who were either exploring the same space or interacting competitively and found an increase in correlation of brain signals in the exploring condition when compared with the competitive condition. To analyse this further, they used GLMs which modelled neural activation in each mouse by including the behavioural signals from both animals, as well as the brain signals from their interaction partner. These extended GLMs outperformed the traditional GLMs (which include only behavioural signals from both animals) at predicting neural activation. The traditional GLM captures the moment-to-moment synchronisation of the animals' behaviours; however, the existence of inter-brain coherence over and above what is captured in the traditional GLM suggests that the mice are mutually predicting each other's behaviour too.

Our experiment was initially designed only to allow us to observe activations in both the Leader and the Follower simultaneously while interacting. However, the experiment design allowed us to also test this mutual prediction hypothesis using an exploratory analysis technique. These are two distinct ideas—whether imitation is a social signal, and whether two people interacting mutually predict each other's neural activations as well—where one does not depend on another. In terms of analysing the inter-brain coupling, we have followed a similar analytical approach to Kingsbury et. al. For each pair of participants we built a traditional GLM and compared this with an extended GLM that incorporates neural activations from an interacting partner. This allows us to test whether there is inter-brain coupling over and above what is captured in the behavioural measurement (if the extended GLM outperforms the traditional GLM).

### *4.2.2. Neural correlates of imitation and social availability*

In this section we review what we know so far about the brain regions involved in imitation, including the observation and production of actions, interpreting the rationality of actions, and processing social context.

*Observing and producing actions*

At its heart, imitation involves observing and producing actions. The ***direct-matching hypothesis*** (Rizzolotti et al., 2001) argues that both action-observation and production arise in the human mirror-neuron system, or MNS. The MNS comprises the IFG, or Broca's area, the IPL, and the STS/MTG. The MNS is thought to enable imitation by directly mapping observed actions onto one's motor system (Iacoboni, 1999, 2005; Rizzolotti & Craighero, 2004); indeed, a large meta-analysis by Caspers et al. (2010) identified consistent activations in the IFG, the IPL, the premotor cortex and adjacent superior frontal gyrus, supplementary motor area, and visual area V5 during action imitation tasks. These support the view that the MNS is implicated in imitative behaviour.

*Rationality*

In addition to observing and producing actions faithfully, imitation also involves examining whether an action is congruent with expectations (see Section 1.4.2). For example, if we move a ball with a high trajectory in order to avoid an obstacle, it is congruent with expectations; a high trajectory that is undertaken when there is no obstacle, however, violates our expectations (Gergely & Csibra, 2003). Here, a number of studies have shown that there is activation in the STS when we view actions that are

incongruent with expectations (Grèzes et al., 2004; Pelphrey et al., 2003; Saxe et al., 2004). Activation in an adjacent region, the MTG has been found to be positively correlated with the degree of (ir)rationality of an action (Jastorff et al., 2011; Marsh, Mullett, et al., 2014). More dorsally, a number of studies have found increased activation in the right TPJ and right IPL when observing irrational actions (Brass et al., 2007; Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011; Oliver et al., 2017). One brain region where the evidence is mixed is the mPFC: two studies showed deactivation in the mPFC for irrational actions (Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011), while others have found an increase in activation in this region when it comes to observing novel irrational actions (Brass et al., 2007) or when there was a mismatch between the content of a narration by an actor and their facial affect (Decety & Chaminade, 2003). Nevertheless, it appears that imitation involves a sophisticated view of rationality that incorporates the physical constraints and goals of an actor in interpreting actions.

*Control of imitation*

Humans do not imitate indiscriminately, and imitation is a sophisticated and dynamic process that requires other inferential and control processes (Brass et al., 2007; Csibra, 1993; Southgate & Hamilton, 2008; Uddin et al., 2007). We now turn to the question of how we interpret actions in the mind. Several studies have shown that the inhibition of imitation in adults involves mPFC and the TPJ (Brass et al., 2001, 2005). The networks engaged in this control are anatomically and functionally distinct from mechanisms involved in controlling other types of prepotent responses such as those engaged in the Stroop task, suggesting this is domain-specific to imitation (Brass et al., 2003, 2005). These networks, the mPFC and TPJ, also form the core of the mentalising network (Amodio & Frith, 2006; Frith & Frith, 2003). Brass et al. (2009) suggest that the process of controlling imitation and mentalising are linked, with both requiring good self-other distinction. In a series of studies they showed that mentalising ability is positively related to the ability to control prepotent imitative responses in those with prefrontal or TPJ lesions (Spengler, von Cramon, et al., 2010), in those with autism (Spengler, Bird, et al., 2010), and in healthy neurotypicals whose self-other distinction was manipulated experimentally (Spengler, Brass, et al., 2010). Stimulation studies have also shown that the right TPJ is implicated in the control of imitation (Hogeveen et al., 2015; Santiesteban et al., 2012; Sowden & Catmur, 2015). The mPFC meanwhile has been found to be involved in modulating imitative behaviour; in particular, the mPFC mediates the effect of eye-gaze (Wang, Ramsey, et al., 2011) and social priming (Wang & Hamilton, 2015) on imitation. Van Overwalle (2009) suggests that the TPJ is involved in inferring temporary

states of mind, while the mPFC integrates these over time into broader traits. Taken together these suggest that in adults mentalising networks are also implicated in imitation.

*Social availability*

Finally, we turn to the question of social availability: is there a brain region that encodes the effect of being watched? This is technologically challenging to study within existing neuroimaging modalities given that they usually study one subject in isolation. However, there is reason to suggest that mentalising is likely to be a key component of this phenomenon (Hamilton & Lind, 2016). Mentalising networks in the brain are reliably engaged in the presence of direct gaze (Wang, Ramsey, et al., 2011) and when people believe they can be seen (Somerville et al., 2013). This effect persists even when the feeling of being watched occurs at a more abstract level, such as when people are told their cognitive capacity is being evaluated (Bengtsson et al., 2009) and when people make self-disclosures (Izuma et al., 2010) or are embarrassed (Müller-Pinzler et al., 2015).

In summary, observing and producing actions appears to involve the mirror-neuron system, or MNS, while the inferential and control processes around imitation implicate the mentalising network. Based on the evidence on gaze and audience effects described above, we expect that the mentalising network is also likely to be involved in encoding social availability, or the effect of being watched. Finally, processing action rationality appears to involve both parts of the MNS and the mentalising network.

### *4.2.3. Current study*

In our study we wished to use one fNIRS system to study two subjects simultaneously. This necessitated splitting the fibres to cover one hemisphere for each participant. Further, using fNIRS requires us to limit ourselves to studying structures on the cortical surface. As discussed in the previous section the observation and production of actions, as well as processing action rationality involve parts of the MNS. Specifically, processing action rationality appears to involve the right IPL, as well as a part of the mentalising network, namely the right TPJ. The right TPJ is also implicated in the inhibition of imitation and the mentalising network more broadly is thought to be involved in encoding the effect of being watched. Given these prior results and the physical constraints of our equipment we focused our study on the right TPJ, right IPL and adjacent areas.

We used a single fNIRS system (Hitachi ETG-4000 Optical Tomography system) and divided the optodes between both participants as shown in Figure 4.1. Dividing the cables from a single system allowed us to overcome the issues of variable sensitivities and synchronisation across multiple devices. As introduced earlier, this study investigates the neural correlates of imitation in an ecologically valid fashion. Pairs of naïve participants are assigned the role of Leader and Follower and asked to undertake a sequential block-moving task in an augmented-reality environment. Privately, Leaders are instructed to make irrationally high trajectories when demonstrating the movement of the blocks to the Follower in some trials. The Follower then makes their move; in half the trials the Follower is watched by the Leader when it is their turn. To avoid confounding our study with the effect of gaze (Senju & Johnson, 2009) and social facilitation (Zajonc, 1965), the feeling of being watched is manipulated at an abstract level through sound cues. The participants stand side-by-side throughout the experimental phase of the study and do not look at each other's faces, therefore there are no differences in eye-gaze and social facilitation throughout.

Based on the social-signalling hypothesis of imitation, our four main hypotheses for this study are:

(a) The Follower will recognise (consciously or subconsciously) that a demonstrated high trajectory is irrational; based on the existing research highlighted above we expect this will be encoded in the right TPJ and right IPL.
(b) The Follower will recognise the social availability of the Leader; that is, they will be aware of when the Leader is watching them. This effect of being watched is expected to be encoded in the mentalising networks of the brain.
(c) The degree to which the Follower imitates the Leader's trajectory will be modulated by whether the Leader is watching the Follower make their movement.
(d) Finally, we expect that incorporating the neural activation of the interaction partner will improve the model fit, supporting the mutual-prediction hypothesis.

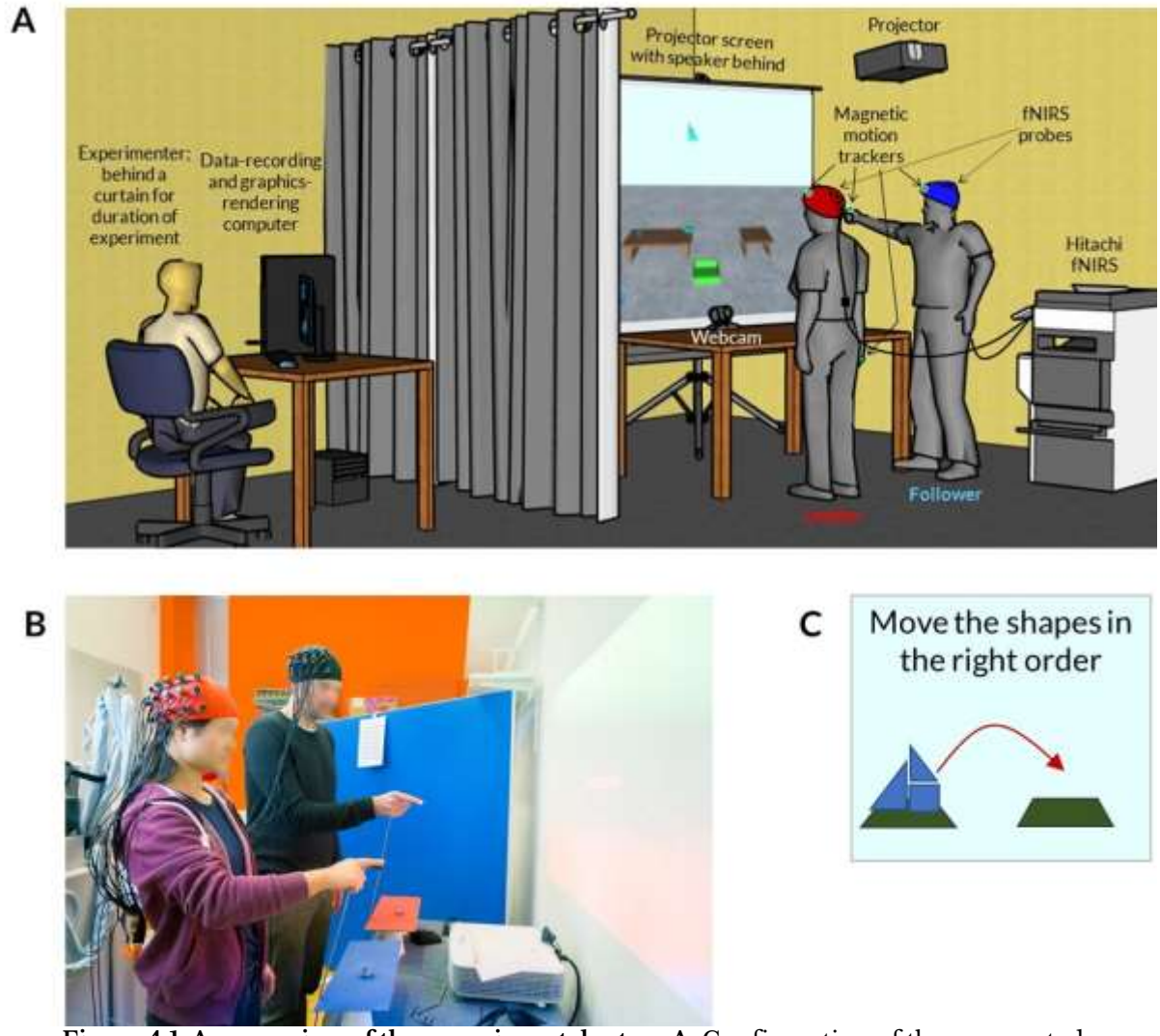


**Figure 4.1. An overview of the experimental setup. A**. Configuration of the augmented reality (AR) lab with the functional near-infrared spectroscopy (fNIRS) equipment. **B.** Two participants stand side-by-side and can see the AR space. The fNIRS probes are positioned over the right temporal-parietal junction (right TPJ) of both participants as shown. **C.** The task involved moving blocks from one table to another in the AR space in the demonstrated order.

## 4.3. Materials and Methods

### *4.3.1. Participants*

A total of 22 pairs of participants were tested in the study. Two pairs of participants were excluded based on the behavioural exclusion criteria (outlined below), leaving us with 20 pairs of participants in the final analysis (13 males, 27 females; mean age = 27.45 years; Std. Dev. = 9.01 years). All were right-handed, had normal or corrected-to-normal vision and hearing, had no history of neurological or psychiatric disorders, and had not participated in this experiment previously.

All participants were recruited for this study using the subject pools of the UCL Department of Psychology and the Institute of Cognitive Neuroscience. Participants were reimbursed financially for their time (£10 for the experiment) and provided informed written consent prior to participating. All procedures were approved by the UCL Research Ethics Committee (ICN-AH-PWB-3-3-2016c).

### *4.3.2. Block-moving task in augmented-reality environment*

Pairs of participants arrived together for the study and were randomly assigned the role of Leader and Follower. After they introduced themselves to each other they were asked to choose a name for their team together and were told they would be competing against other teams whose (partly fictional) scores were on a visible leader board. This was done to induce a prosocial collaborative frame of mind within the dyad, rather than have them view each other as competitors (also see Section 2.5 for details on the cover story that was provided to participants on what the experiment was designed to study). The participants stood side by side (see Figure 4.1) facing the screen; the Leader was always on the left-hand side and the Follower was on the right-hand side (it was not possible to counterbalance this owing to the orientation of the fNIRS equipment). Magnetic motion-trackers (Polhemus Liberty, Colchester Vermont) were fixed to the right hand and the forehead of each participant. These hand markers allowed participants to control a hand icon in the augmented-reality environment. The augmented-reality environment and the experiment were implemented in Vizard (WorldViz, Santa Barbara, CA).

*Timeline of one trial*

The timeline of a typical trial is shown in Figure 4.2 below. At the start of each trial the Follower was instructed via a computerised voice command to close their eyes. The Leader saw a demonstration of three or four blocks being moved from one table to another in a particular order within the AR environment (Figure 4.2A). The Follower was then asked to open their eyes, and the Leader demonstrated the order in which the blocks are to be moved to the Follower (Figure 4.2B). The blocks were then reset to their starting position. The Follower was then asked to move the blocks in the same order to the right-hand side table (Figure 4.2C). Both participants then saw a joint score based on accuracy (moving the blocks in the right order) and timing (moving quickly) (Figure 4.2D).

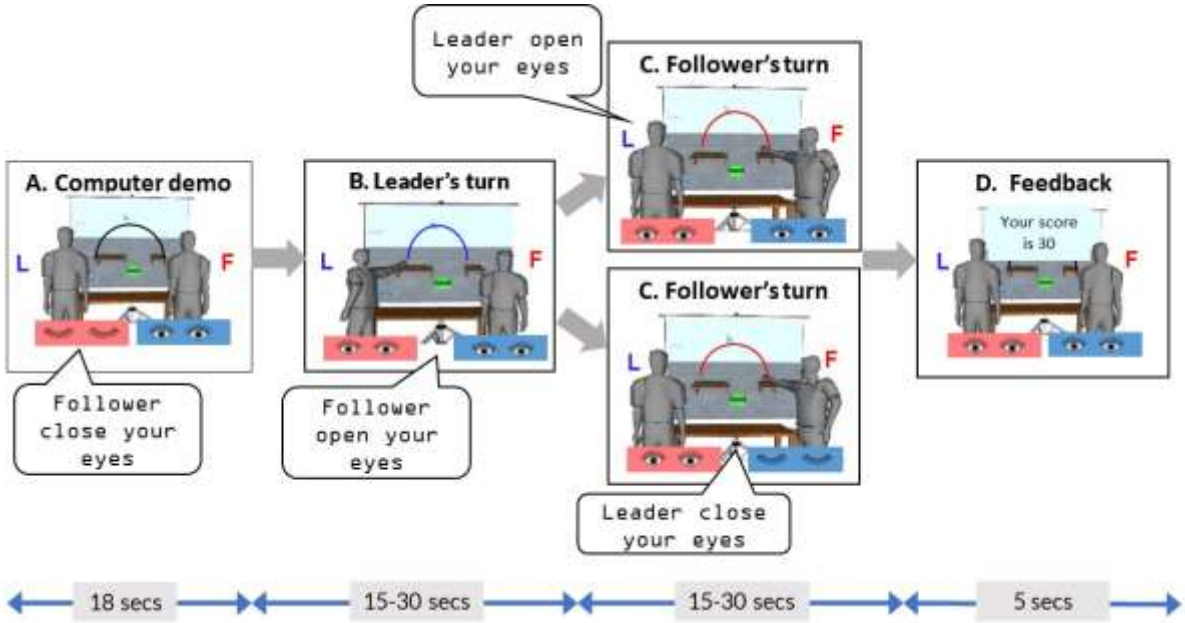


**Figure 4.2. Trial timeline.** **A.** The Follower closes their eyes while the Leader watches the computer demonstration. **B.** The Leader demonstrates, while the Follower watches. **C.** The Follower moves the blocks (The Leader's eyes can be open or closed). **D.** Participants see a joint score which rewards accuracy and speed. Speech bubbles throughout show computerised voice commands.

## *Phases of the experiment*

The study had three phases: familiarisation (both Leader and Follower), Leader practice (with only the Leader), and full experimental trials. These phases are summarised in Table 4.1 and explained in detail below.

**Table 4.1 Phases of the experiment**

| Phase | Explicitly instructed to copy trajectory of demonstration? | | Followers' eyes during demonstration | Leaders' eyes during Follower's movement | Trajectory heights | Number of trials |
|---|---|---|---|---|---|---|
| | Leader | Follower | | | | |
| Familiarisation | No | No | Closed | Open | Low / Med / High | 3 |
| Leader practice | Yes | N/A | N/A | N/A | Low / Med / High | 3 |
| Experimental | Yes | No | Closed | Open / Closed | Low / Med / High | 18 |

In the *familiarisation phase* participants hand movements were calibrated within the augmented-reality environment and they practiced using their hands to move blocks. After three familiarisation trials the Follower was told that they would now have the fNIRS cap placed on them, and that the Leader would have additional practice trials during this time. The Follower was then asked to turn and sit in a way that they were unable to see the screen during the Leader's practice trials.

During the *Leader's practice phase* the Leader was given explicit instructions to copy the trajectory demonstrated by the computer and asked to keep this secret. The instructions displayed for the Leader are shown below in Figure 4.3A. These instructions were left onscreen for as long as it took the Leader to read and absorb the instructions; they signalled with a head nod once they had finished reading it, and then proceeded to the Leader practice trials. The Leader was shown the three different trajectories (low, medium and high) in a randomised order over three trials and asked to copy the trajectory. The Leader was then given positive or negative feedback on how accurately they copied the path demonstrated (Figure 4.3B).

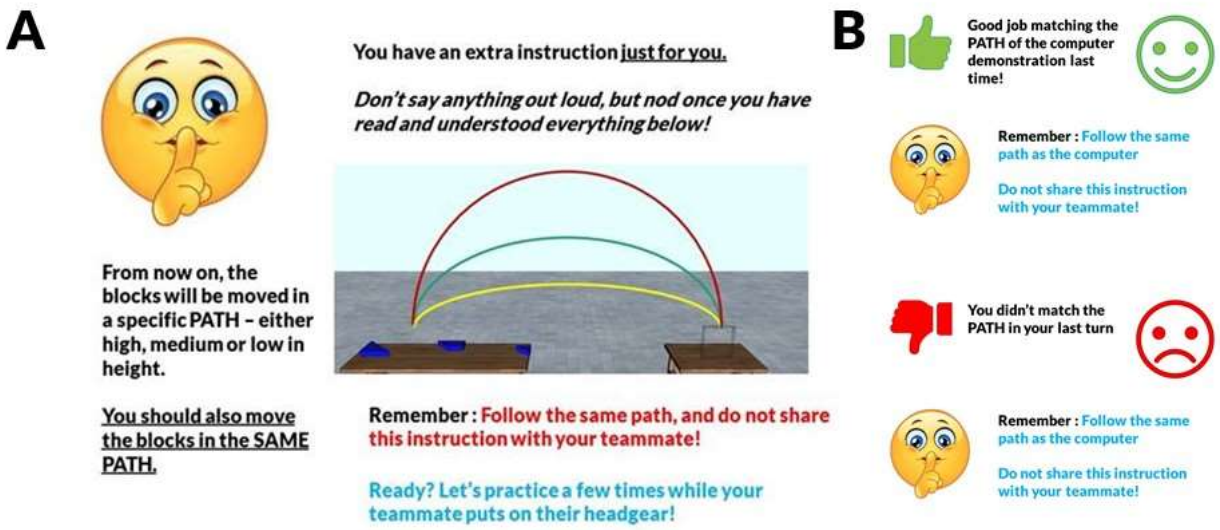


**Figure 4.3. Leader's secret instructions. A.** At the start of the Leader practice trials the Leader is told to copy the trajectory demonstrated by the computer and to keep this secret from their teammate (the Follower). **B.** Feedback screens shown to the Leader at the start of each trial in the Leader practice trials and the Experimental trials, showing whether they followed the path demonstrated by the computer in the previous trial or not.

While the Leader completed their practice trials, the fNIRS optodes were placed on the Follower. Following this, the Leader also had the fNIRS cap placed on their head (the procedure is described below), before moving on to the *experimental phase.* In this phase we used a 3 x 2 factorial design with three different trajectory heights (low, medium and high) crossed with whether the Leader had their eyes open or closed during the Follower's turn (Watched and Unwatched). The six conditions were repeated thrice over the 18 experimental trials, with the trajectories presented in a randomised order. The Watched and Unwatched trials occurred in blocks of three trials; the initial block (Watched or Unwatched) was randomised.

Following the Experimental Trials, the fNIRS probes and the Polhemus magnetic motion trackers were removed from participants, and both separately filled out a series of questionnaires including:

(i) Rapport questionnaire: Six-item survey of their feelings of rapport towards each other
(ii) AQ (Baron-Cohen et al., 2001) which measures the extent of autistic traits in adults
(iii) Interaction Anxiousness Scale (Leary, 1987) which measures social anxiety, and
(iv) Rosenberg Self-Esteem Scale (Rosenberg, 1965) which measures self-esteem.

Participants also completed a written debrief in which they were asked what they thought the purpose of the experiment was, and whether they noticed the differences in the trajectory heights prior to being explicitly told about them. Further, they were asked to self-report their ethnicity and their level of familiarity with their partner and the experimenter. In addition to tracking motion using a motion-capture system, we also recorded videos of the participants performing the task. This allowed us to verify that they kept their eyes closed and refrained from talking when required to do so, ensuring that the social manipulation worked.

### *4.3.3. Behavioural analysis*

The behavioural analysis follows the same procedure as in the two studies described in Chapter 3. The analysis centred on a single parameter: the maximum heights reached by the Leader and the Follower in each trial in the Experimental phase. Given that each trial involved moving three or four blocks, in our view peak height is the most salient measure of the fidelity with which a movement trajectory was copied. To account for individual differences between Leaders, the Pearson correlation coefficient (R) was calculated between the Leader's height and the Follower's height in each trial and used to evaluate imitation fidelity across the Watched and Unwatched trials.

*Behavioural exclusion criteria*

Dyads were excluded for the following reasons:

(i) Data was not recorded owing to equipment failure or failure in the task software;

(ii) In the Full trials if the Leader failed to copy the computer demonstration with high fidelity (defined as having an R value of 0.5 or higher) after having been explicitly instructed to do so;

(iii) The Leader revealed the secret instruction (to copy the trajectory demonstrated by the computer) to the Follower during the full trials;

(iv) The Follower specifically asked the Leader about the path or trajectories they were demonstrating;

(v) Either the Leader or the Follower failed to follow instructions to close their eyes at various points in the trial.

### *4.3.4. fNIRS data acquisition*

The NIRS signals were recorded using an ETG-4000 (Hitachi, Japan) Optical Topography system. A 3 x 5 probe holder was attached to a regular swimming cap for each participant and positioned over the right hemisphere, centred on the right TPJ as shown in Figure 4.1B. A total of 15 optodes, comprising eight dual-wavelength laser diodes (695/830nm) and seven photo detectors, allowed for measurement of 22 channels as shown in Figure 4.4A. The distance between source-detector pairs was 3 cm. The signals were acquired at a frequency of 10 Hz and the signals were downsampled to 1 Hz to reduce temporal autocorrelation.

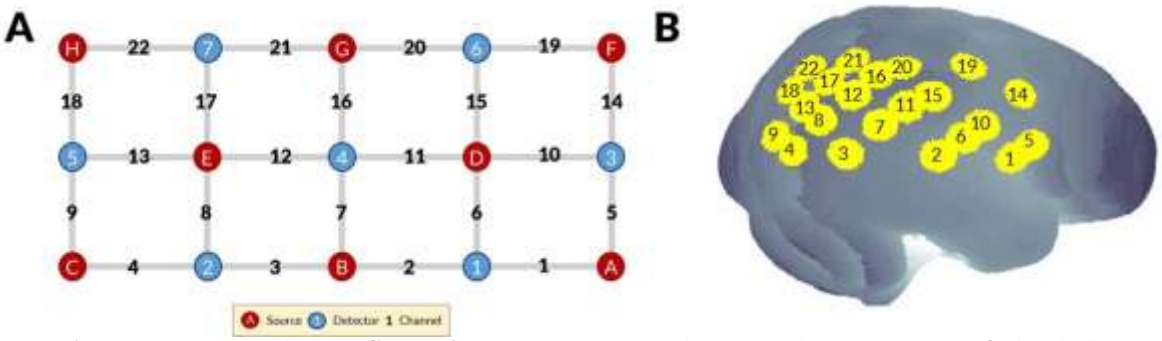


**Figure 4.4. Optode configuration. A.** Diagram showing the positions of the light sources (red) and receivers (blue) and the 22 channels created by the 15 probes. **B.** Locations of the 22 channels on the right hemisphere for both participants.

*fNIRS optode localisation*

The locations of the optodes and five canonical head locations (inion, nasion, right preauricular, left preauricular and vertex) were recorded for one participant and the corresponding Montreal Neurological Institute (MNI) coordinates (Mazziotta et al., 2001)

for each channel was obtained using the NIRS-SPM software (Ye et al., 2009) with MATLAB® and the corresponding anatomical locations of each channel were derived from the included atlas (Rorden & Brett, 2000). Table 4.2 lists the MNI coordinates and the anatomical regions, Brodmann Areas (BA) and the probability that each region is included in the channel. Only regions with a probability greater than 0.1 are included in the table below. The equipment used to digitise the optode locations was very sensitive to magnetic interference in the testing environment, preventing us from obtaining optode locations for each individual participant.

**Table 4.2. Channel coordinates and anatomical regions.** Channels are listed below alongside their Montreal Neurological Institute (MNI) coordinates (Mazziotta et al., 2001). The corresponding anatomical region labels are obtained from the NIRS-SPM software (Ye et al., 2009) based on the included atlas (Rorden & Brett, 2000). Alongside each anatomical region the corresponding Brodmann Area (BA) and the probability that this region is included in the channel are shown.

| Channel number | MNI coordinates | | | Anatomical region | BA | Probability of inclusion |
|---|---|---|---|---|---|---|
| | X | Y | Z | | | |
| 1 | 66.0 | 3.3 | 9.7 | Retrosubicular area | 48 | 0.50 |
| | | | | Pre-Motor and Supplementary Motor Cortex | 6 | 0.31 |
| | | | | Subcentral area | 43 | 0.13 |
| 2 | 72.3 | -29.7 | 11.7 | Superior Temporal Gyrus | 22 | 0.88 |
| | | | | Middle Temporal gyrus | 21 | 0.10 |
| 3 | 60.0 | -67.3 | 12.3 | Fusiform gyrus | 37 | 0.67 |
| | | | | Angular gyrus, part of Wernicke's area | 39 | 0.27 |
| 4 | 45.3 | -87.7 | 12.7 | V3 | 19 | 0.82 |
| | | | | Visual Association Cortex (V2) | 18 | 0.18 |
| 5 | 65.0 | 11.7 | 15.7 | Pre-Motor and Supplementary Motor Cortex | 6 | 0.63 |
| | | | | pars opercularis, part of Broca's area | 44 | 0.29 |
| 6 | 70.0 | -18.3 | 20.7 | Superior Temporal Gyrus | 22 | 0.43 |
| | | | | Primary Somatosensory Cortex | 2 | 0.32 |
| | | | | Subcentral area | 43 | 0.25 |
| 7 | 65.3 | -53.7 | 27.3 | Superior Temporal Gyrus | 22 | 0.60 |
| | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.19 |
| | | | | Angular gyrus, part of Wernicke's area | 39 | 0.14 |
| 8 | 50.7 | -76.7 | 28.7 | Angular gyrus, part of Wernicke's area | 39 | 0.84 |
| | | | | V3 | 19 | 0.16 |
| 9 | 35.3 | -93.0 | 19.3 | V3 | 19 | 0.49 |
| | | | | Visual Association Cortex (V2) | 18 | 0.46 |
| 10 | 70.0 | -10.3 | 26.7 | Subcentral area | 43 | 0.78 |
| | | | | Primary Somatosensory Cortex | 2 | 0.17 |
| 11 | 67.0 | -42.7 | 36.7 | Supramarginal gyrus part of Wernicke's area | 40 | 0.88 |
| | | | | Retrosubicular area | 48 | 0.10 |
| 12 | 56.3 | -63.3 | 42.0 | Angular gyrus, part of Wernicke's area | 39 | 0.96 |
| | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.04 |
| 13 | 42.3 | -82.3 | 35.3 | V3 | 19 | 0.76 |
| | | | | Angular gyrus, part of Wernicke's area | 39 | 0.24 |
| 14 | 58.7 | 8.3 | 41.7 | Pre-Motor and Supplementary Motor Cortex | 6 | 0.74 |
| | | | | pars opercularis, part of Broca's area | 44 | 0.18 |
| 15 | 68.0 | -31.3 | 42.3 | Supramarginal gyrus part of Wernicke's area | 40 | 0.56 |
| | | | | Primary Somatosensory Cortex | 2 | 0.26 |
| | | | | Primary Somatosensory Cortex | 1 | 0.18 |

| Channel number | MNI coordinates X | Y | Z | Anatomical region | BA | Probability of inclusion |
|---|---|---|---|---|---|---|
| 16 | 56.3 | -53.3 | 52.3 | Supramarginal gyrus part of Wernicke's area | 40 | 0.79 |
| | | | | Angular gyrus, part of Wernicke's area | 39 | 0.21 |
| 17 | 45.3 | -72.3 | 48.3 | Angular gyrus, part of Wernicke's area | 39 | 0.58 |
| | | | | Somatosensory Association Cortex | 7 | 0.31 |
| | | | | V3 | 19 | 0.11 |
| 18 | 29.3 | -86.0 | 42.3 | V3 | 19 | 0.88 |
| | | | | Somatosensory Association Cortex | 7 | 0.11 |
| 19 | 56.3 | -13.7 | 56.3 | Primary Motor Cortex | 4 | 0.48 |
| | | | | Primary Somatosensory Cortex | 3 | 0.31 |
| | | | | Pre-Motor and Supplementary Motor Cortex | 6 | 0.20 |
| 20 | 56.7 | -42.3 | 56.7 | Supramarginal gyrus part of Wernicke's area | 40 | 0.81 |
| | | | | Primary Somatosensory Cortex | 2 | 0.15 |
| 21 | 43.7 | -61.7 | 58.0 | Somatosensory Association Cortex | 7 | 0.40 |
| | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.34 |
| | | | | Angular gyrus, part of Wernicke's area | 39 | 0.27 |
| 22 | 29.7 | -77.7 | 53.3 | Somatosensory Association Cortex | 7 | 0.90 |
| | | | | V3 | 19 | 0.10 |

### *4.3.5. fNIRS analysis*

The raw fNIRS data (showing absorption of the two wavelengths) was converted into concentration changes of HbO, HbR, and total-Hb by the modified Beer-Lambert law using custom MATLAB® scripts and the preprocessing of the data was performed in HomER (Huppert et al., 2009). In line with the recommendations made by Pinti et al. (2019) we applied a bandpass filter (0.01 Hz to 0.3 Hz) to denoise the data.

#### *fNIRS exclusion criteria*

The data for each of the 22 channels across all 40 participants (from the 20 valid dyads) was visually inspected in MATLAB to correct for motion artefacts, and channels with large motion artefacts were excluded from the analysis. The HbO and HbR data for each channel were also visually inspected to see if they were positively correlated with each other; this would be an indication that the signal was being driven by other physiological changes and not by neural activity (Oliver et al., 2017; Tachtsidis & Scholkmann, 2016). Channels where both signals were positively correlated were also removed from the analysis. Following these exclusions, participants for whom there were fewer than nine usable channels (representing 40% of the channels) were excluded (1 Leader and 2 Followers). For the remaining 37 participants, individual channels were excluded if they were subject to motion artefacts or physiological noise.

*First-level analysis*

The first-level GLM analysis was performed using the fNIRS toolbox in SPM12 (Friston et al., 1994; Tak et al., 2016) implemented in MATLAB®. For this analysis, the HbO and HbR data were combined using the correlation-based signal improvement method (Cui et al., 2010), or CBSI, to generate a corrected activation signal. This is a noise-reduction algorithm that is built on maintaining the negative correlation between HbO and HbR and this combined CBSI signal is used in the remainder of this analysis.

The design matrix for the Leader included one regressor for the Leader's action, and one for the Follower's action in the Watched trials, as well as additional regressors for the demonstration and scoring portions of each trial. Two parametric regressors were generated for the Leader's action and the Follower's action in the Watched trials; the weightings in these parametric regressors were determined by the height demonstrated by the Leader and the height reached by the Follower, respectively. The Leader's design matrix thus had seven regressors.

Two parametric contrasts were created by placing a +1 over the column for each parametric regressor, and with zeros placed over all other columns. This allowed us to calculate activation when the Leader performed an irrational action (C1, based on the parametric regressor using the height demonstrated by the Leader), and activation when the Leader watched the Follower perform an irrational action (C2, based on the parametric regressor using the height demonstrated by the Follower). The relevant columns and contrasts are shown below in Table 4.3.

**Table 4.3. Contrasts.** The structure of the individual design matrices is shown below for the Leader and the Follower showing the included regressors for each. The five contrasts of interest are highlighted showing how they are constructed, with +1 over each relevant column for forward contrasts, and a -1 over the relevant columns for reverse contrasts.

| Leader design matrix | Demo | Leader action | Leader action (Parametric based on Leader height) | Follower action - Watched | Follower action - Watched (Parametric based on Follower height) | Score | Const |
|---|---|---|---|---|---|---|---|
| C1 Performing irrational actions | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| C2 Watching Follower perform irrational actions | 0 | 0 | 0 | 0 | 1 | 0 | 0 |

| Follower design matrix | Demo | Leader action | Leader action (Parametric based on Leader height) | Follower action - Watched | Follower action - Watched (Parametric based on Follower height) | Follower action - Unwatched | Follower action - Unwatched (Parametric based on Follower height) | Score | Const |
|---|---|---|---|---|---|---|---|---|---|
| C3 Watching Leader perform irrational actions | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| C4 Performing actions when Watched vs Unwatched | 0 | 0 | 0 | 1 | 0 | -1 | 0 | 0 | 0 |
| C5 Performing irrational actions (Watched and Unwatched) | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 |

For the Follower, the design matrix included regressors for the Leader's action, the Follower's action in the Watched condition and the Follower's action in the Unwatched condition, as well as additional regressors for the demonstration and scoring portions of each trial. As above, three parametric regressors were generated: one for the Leader's action, based on the height demonstrated by the Leader, and one each for the Follower's action in the Watched and in the Unwatched conditions, both based on the height reached by the Follower. Together, the Follower's design matrix had nine regressors.

Three different contrasts were calculated by placing a +1 over the column for forward contrasts and a -1 over the column for reverse contrasts. C3 calculated activation in the Follower when watching the Leader perform an irrational action; this was calculated using the parametric regressor of the Leader's action based on height demonstrated by the Leader. C4 evaluated activation in the Follower when being watched; this was calculated by placing a +1 over the column for the Follower action in the Watched condition and a -1 over the column for the Follower action in the Unwatched condition. Finally, C5 evaluated activation in the Follower in both the Watched and Unwatched condition when performing an irrational action; this was calculated by placing a +1 over both the parametric regressors of the Follower's action (Watched and Unwatched columns) which are based on the height reached by the Follower. The relevant columns and contrasts are shown in Table 4.3. These analyses were all performed at the individual level before being combined at the group level as outlined below.

*Group-level analysis*

At the group level, t-tests were conducted on the individual parameter values (β values) for each contrast across all valid channels. We did not undertake a voxelwise analysis approach owing to the high likelihood of false positive findings given multiple voxel comparisons (Hirsch et al., 2017). Instead, we used individual channels as the unit of analysis. Given the limited number of planned comparisons, the uncorrected p-values are presented in the results below. However, where two or more adjacent channels show a significant effect for $p<0.05$ we consider this as a corrected significant result (Pinti, Tachtsidis, et al., 2020; Southgate et al., 2014).

*Extending the GLM*

In addition to this traditional GLM, we undertook an exploratory analysis of the inter-brain links within each dyad. Building on the approach used by Kingsbury et al. (2019) we created two additional design matrices for the Leader and the Follower. The traditional GLM incorporated the task-related regressors (seven regressors in the case of the Leader, and nine regressors for the Follower) as outlined in Table 4.3. In addition, the extended GLM included a further 22 additional regressors comprising the neural activation in the channels of the interaction partner. Channels which had to be excluded for failing quality checks (outlined in the fNIRS exclusion criteria in Section 4.3.5 above) were also excluded here. For each channel of each participant, we compared the fit of the traditional GLM to the extended GLM using the inbuilt MATLAB® likelihood ratio test to determine whether the inclusion of the additional regressors improved the model fit; a threshold p-value of 0.05 was used for this comparison.

The traditional GLM models activity in each participant (Leader and Follower) separately as a function of both their behaviours as described above. The extended GLM models activity for each participant as a function of both participants' behaviours, as well as the brain activity of their interaction partner. As Kingsbury et al. (2019) argue, if the extended model is a better fit this would suggest activations in one subject contained information about activation in the other subject over and above what can be explained by moment-to-moment behaviour during the task.

## 4.4. Results

### *4.4.1. Overall peak height*

The Pearson correlation coefficient (R) was calculated between the maximum height reached by the Leader and the Follower across all 18 trials in the Experimental phase. Figure 4.5A shows the trendlines for the relationship between the Leader's height and the Follower's height for all dyads, and we can see that there is a positive relationship between the heights reached by the Leader and the Follower. However, a one-sample t-test showed that while this was in line with previous studies (see Chapter 3) the effect was not statistically significant [N = 20, Mean R value = 0.13, Std. Dev. = 0.35, t(19) = 1.66, p = 0.113].

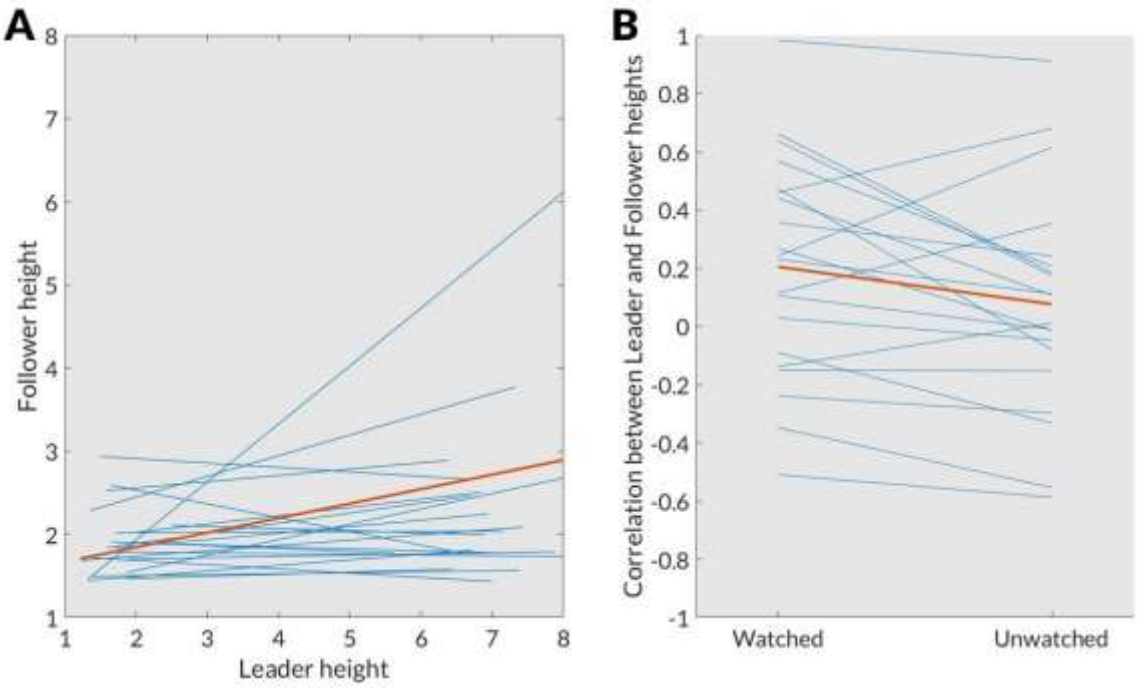


**Figure 4.5. Behavioural Results of fNIRS study (N = 20). A. Overall imitation pattern:** Line of best-fit for the correlation between Leader heights and Follower heights for each dyad (blue lines) and the group as a whole (heavy orange line). **B. Correlations:** The R values representing the correlation between the Leader and Follower heights are shown for each dyad and for the whole group (heavy orange line) for trials where the Leader watches the Follower make their movements and trials where the Leader does not watch.

### *4.4.2. Imitation fidelity in watched vs unwatched condition*

Turning to the main experimental question of whether the fidelity with which Follower's imitated Leader's trajectories was modulated by whether the Followers were being watched by the Leader or not, our results are in line with previous work (Chapter 3, and Krishnan-Barman & Hamilton, 2019). Across all participants, we compared the correlation between the Leader and the Follower heights in the Watched condition and

in the Unwatched condition (Figure 4.5B). A paired-sample t-test [N = 20] showed that the correlation coefficients (R values) were higher when the Follower was being watched by the Leader [Mean R value = 0.21, Std. Dev. = 0.38] than when the Follower was not being watched by the Leader [Mean R value = 0.08, Std. Dev. = 0.38] and that this effect was statistically significant [t(19) = 2.32, p = 0.031]. This offers further support to the social-signalling hypothesis of imitation, with Followers imitating their teammate with greater fidelity when they know the teammate is watching them, compared with trials where they know that the interaction partner is not watching them.

### *4.4.3. Brain areas parametrically modulated by rationality when observing action*

In both the Leader and the Follower channels in the right TPJ and the right IPL were parametrically modulated by the rationality of their interaction partner's movement (namely the height of the trajectory). In the Leader (C2 in Table 3.3), activation in channel 7 (right TPJ) was found to be parametrically modulated by the height performed by the Follower (Figure 4.6A). In the Follower (C3), activation in channel 12 (right angular gyrus) was found to be parametrically modulated by the height demonstrated by the Leader (Figure 4.6B).

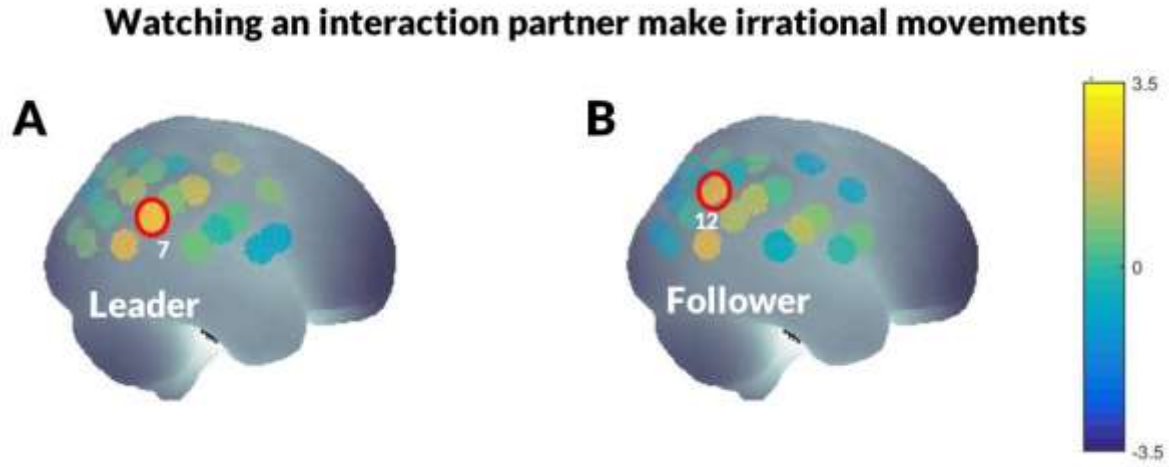


**Figure 4.6. Channel-wise activation when watching irrational actions.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ are highlighted with a red circle.

### *4.4.4. Brain areas parametrically modulated by rationality when performing action*

When it comes to performing irrational actions, no channels in the Leader showed significant activation in this parametric contrast (C1). It is important to note that the Leader performed high movements when specifically instructed to do so by the

computer. As a result, the high movement performed is not strictly "irrational" since the Leader is aware of why they are making this movement.

When it comes to the Follower we see that activation in channel 22 is supressed parametrically based on the height performed by the Follower (C5 in Table 4.3 and Figure 4.7B). This means that activation in channel 22, part of the right superior parietal lobe (right SPL), is greater when the Follower performs a rational action (of moving with a low trajectory) than when the Follower performs an irrational action (moving with a high trajectory). While the SPL has been implicated in other studies in the copying of arm postures (Tanaka & Inui, 2002), the direction of the effect here appears to run counter to what we would expect if it was an effect of moving higher.

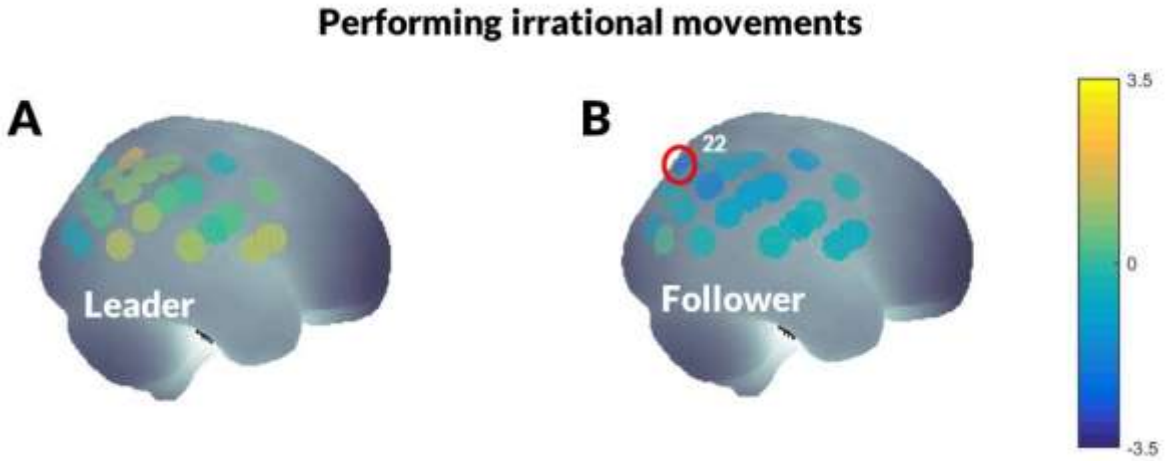


**Figure 4.7. Channel-wise activation when performing irrational actions.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ are highlighted with a red circle.

### *4.4.5. Brain response to being watched*

The Follower's brain responses (C4 in Table 4.3) when being watched by the Leader (Watched) were contrasted with responses when not being watched (Unwatched). Six channels around the right TPJ extending into the right IPL showed greater deactivation in the Watched condition. This suggests that the abstract effect of being watched could be encoded here.

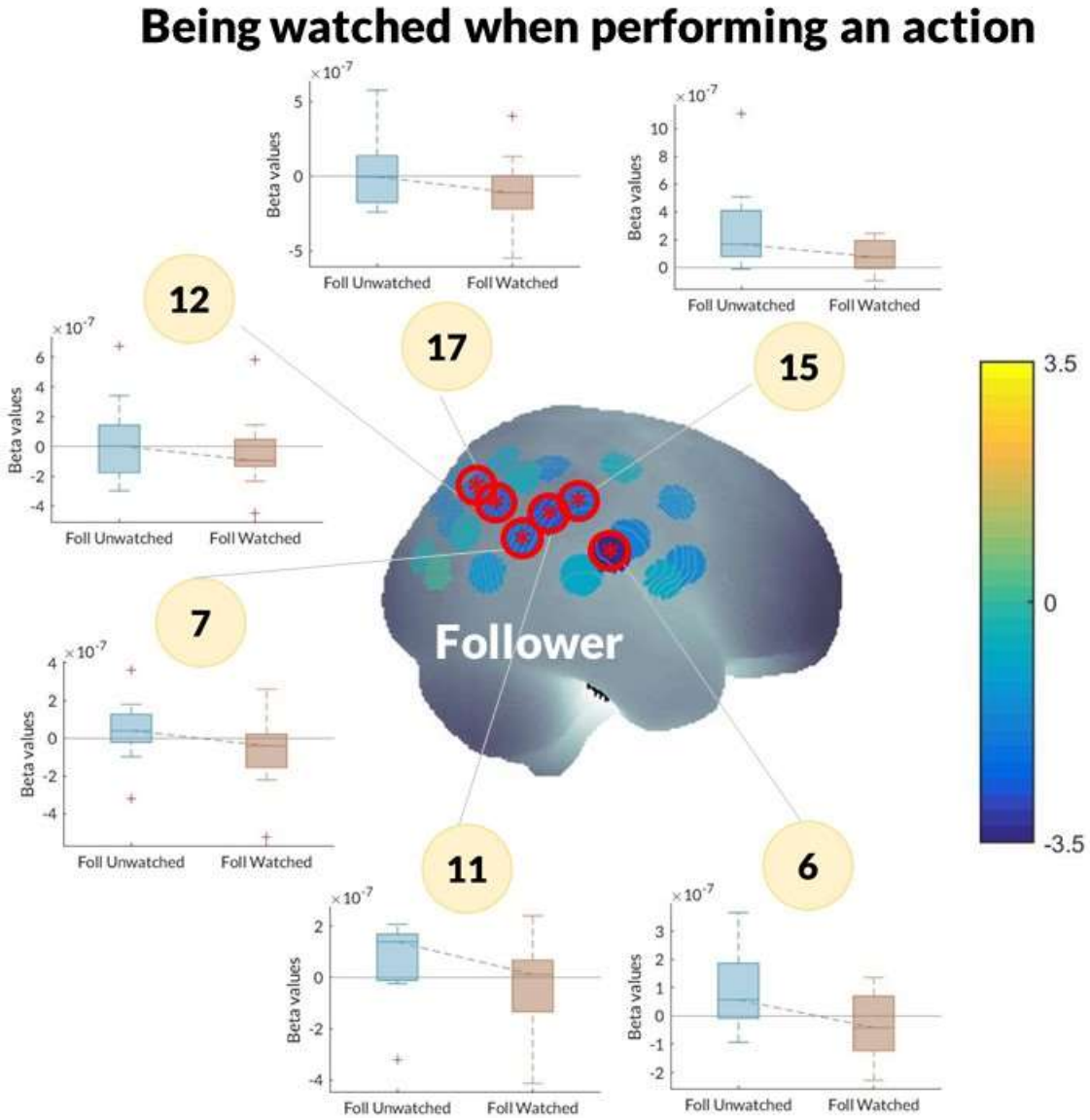


**Figure 4.8. Channel-wise activation when being watched.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Significant channels (uncorrected $p<0.05$) surviving the correction for multiple comparisons (where two or more adjacent channels are also significant) are marked with asterisks and a red circle (Southgate et al., 2014).

The figure above (Figure 4.8) shows the six channels that showed a unique effect of being watched. Since in each case two or more adjacent channels show a significant effect for $p<0.05$ this is considered corrected for multiple comparisons (Pinti, Tachtsidis, et al., 2020; Southgate et al., 2014). In all six channels the activation in the Unwatched condition was greater than in the Watched condition.

### *4.4.6. Summary of brain activation results*

Table 4.4 below shows the details of channels where unique statistically significant responses were found for each of the four significant contrasts. C1 was excluded from this

since none of the channels in the Leader were parametrically modulated by the height performed by the Follower.

**Table 4.4. Channels showing significant activations in GLM analysis**. The channels are listed below alongside the corresponding anatomical region labels are obtained from the NIRS-SPM software (Ye et al., 2009) based on the included atlas (Rorden & Brett, 2000). The most likely anatomical region is included in this table; for full details please refer to Table 4.2. There were no channels with significant activations for C1; this has been excluded from the table below. For contrasts C2 – C5 the T values and the uncorrected p-values are listed for each channel showing significant activations. Significant channels which survive the correction for multiple comparisons by virtue of having two or more adjacent channels with an uncorrected p<0.05 are highlighted in bold.

| | | C2 Leader watching Follower perform irrational actions | | C3 Follower watching Leader perform irrational actions | | C4 Follower performing actions Watched >> Unwatched | | C5 Follower performing irrational actions | |
|---|---|---|---|---|---|---|---|---|---|
| **Channel number** | **Anatomical label** | **T** | **p (uncorr)** | **T** | **p (uncorr)** | **T** | **p (uncorr)** | **T** | **p (uncorr)** |
| 6 | Superior Temporal Gyrus | | | | | **-3.32** | **0.01** | | |
| 7 | Superior Temporal Gyrus | 2.56 | 0.02 | | | **-2.22** | **0.04** | | |
| 11 | Supramarginal gyrus part of Wernicke's area | | | | | **-2.68** | **0.03** | | |
| 12 | Angular gyrus, part of Wernicke's area | | | 2.50 | 0.03 | **-2.66** | **0.02** | | |
| 15 | Supramarginal gyrus part of Wernicke's area | | | | | **-2.27** | **0.04** | | |
| 17 | Angular gyrus, part of Wernicke's area | | | | | **-2.64** | **0.02** | | |
| 22 | Somatosensory Association Cortex | | | | | | | -2.38 | 0.04 |

### *4.4.7. Comparing the fit of the traditional GLM with an extended GLM*

The traditional GLM (incorporating only the behavioural data from both participants) was compared with the extended GLM (which additionally incorporated neural activation from all 22 channels of the interaction partner). Figure 4.9 shows this comparison for one channel (channel number 6): for each Leader, the brain activation was modelled using the traditional GLM (with seven regressors), and using the extended GLM (with the seven regressors plus an additional 22 regressors incorporating activation from each channel of the corresponding Follower).

The traditional model and the extended model were compared using the inbuilt MATLAB© function `lmecompare`. This compares the two models using the likelihood ratio test to generate the maximised log likelihood for each model, and a p-value for the likelihood ratio test comparing the two models. While the traditional model has far fewer

parameters than the extended model, the comparison corrects for this, generating both Akaike and Bayesian information criteria for each model. In Figure 4.9 the traditional model is highlighted in blue if it is the better model (null hypothesis), while the extended model is highlighted in orange if the p-value shows that it is a statistically significant improvement over the traditional model.

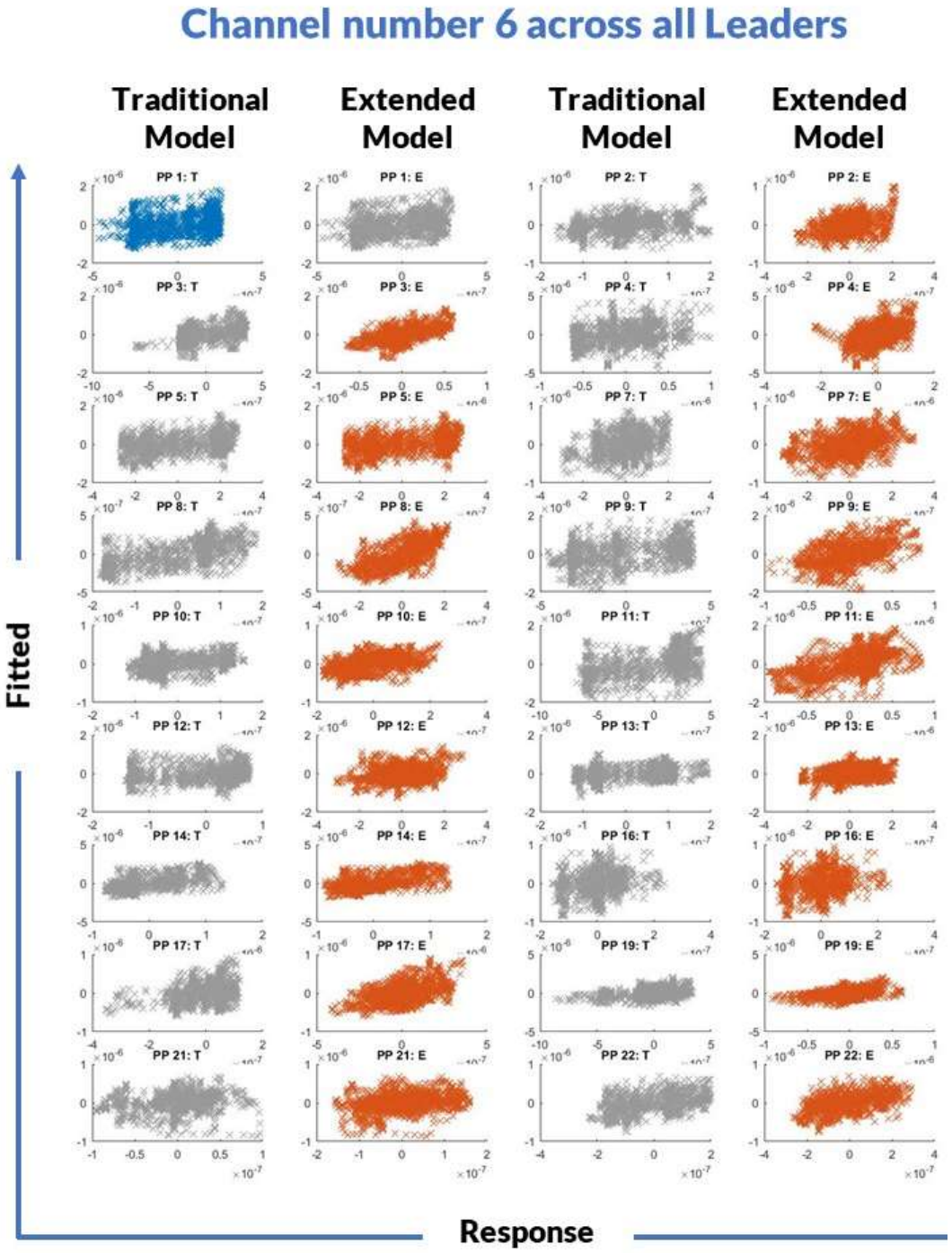


**Figure 4.9. Comparison of the traditional GLM and extended GLM for channel 6 for all Leaders.** The traditional GLM incorporated task-related regressors, while the extended GLM also included neural activation from the interacting partner, in this case

the Follower. The models were compared using the inbuilt MATLAB® likelihood ratio test (**`lmecompare`**) to determine whether inclusion of the additional regressors improved the model fit. Participants for whom the extended GLM met the threshold p-value of 0.05 are highlighted in orange; participants for whom this threshold was not met have the traditional GLM highlighted in blue.

The log likelihood of the extended and traditional GLMs across all Leaders (Figure 4.10 A) and all Followers (Figure 4.10 B) for each channel is shown below. Adding the neural activation of the interaction partner improved the model fit supporting the view that there is inter-brain coupling over and above what is captured by their behaviour. This would suggest that both the Leader and the Follower are mutually predicting each other's brain activation patterns.

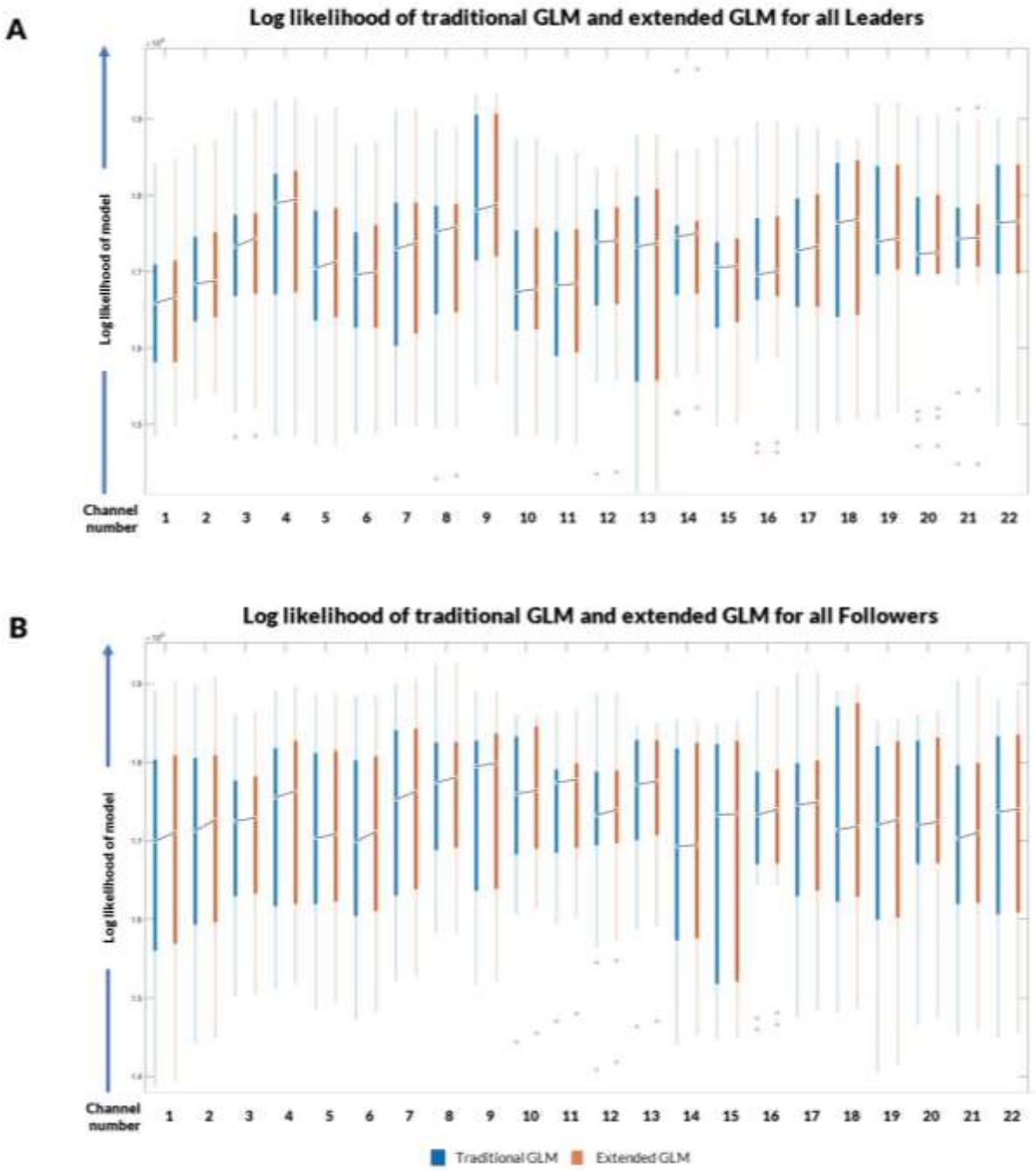


**Figure 4.10. Log likelihood comparison of traditional and extended GLMs. A.** This shows the comparison of the log likelihoods of the traditional GLM to the extended GLM for all Leaders. **B.** This shows the comparison of the log likelihoods of the traditional GLM to the extended GLM for all Followers.

## 4.5. Discussion

In this study we sought to extend the social-signalling hypothesis of imitation by evaluating its neural correlates using the same simple block-moving task in an augmented-reality environment as outlined in Chapter 3 (Krishnan-Barman & Hamilton, 2019). In addition to replicating our behavioural results we found a robust effect of being

watched on brain activity in the parietal cortex. Our results also suggest that incorporating neural activation from an interaction partner improved the model fit, supporting the mutual-prediction account. In this discussion we will first review the behavioural results, followed by the brain responses to watching and performing irrational actions, brain responses to being watched, and the benefits of incorporating an interaction partner's neural signals in a model of neural activity. Finally, we will discuss some limitations of this study and directions for future work.

### *4.5.1. Imitative fidelity when watched by an interaction partner*

In line with our previous work (Krishnan-Barman & Hamilton, 2019) we found that adults imitated irrational movements with greater fidelity when they knew they were being watched by an interaction partner. This is in line with other recent studies that have shown that our imitative fidelity is modulated by the social availability of the demonstrator (DiYanni et al., 2011; Marsh et al., 2019; Nielsen & Blank, 2011; Sommer et al., 2020).

Studies of the audience effect have shown that differences in behaviour may arise owing to differences in anxiety and attention when we are being watched (Heyes, 2017), differences in arousal from direct gaze (Senju & Johnson, 2009), or mere social facilitation (Zajonc, 1965). However, our study was designed to minimise the differences in social context between the watched and unwatched condition: participants stood side-by-side throughout rather than face to face. This ensured there were no differences in social facilitation or eye-gaze between the two conditions. If an increase in anxiety in the Watched condition affected behaviour, we would expect the effect to be reversed as typical hand movements tend to proceed in a straight line (Abend et al., 1982) and participants' scores were based on speed, encouraging them to move as quickly as possible. Instead, people imitate their partners' irrational trajectories with greater fidelity in the Watched condition, precluding the anxiety explanation. Finally, the demonstration phases in both Watched and Unwatched trials were identical ruling out explanations involving a difference in attention. This suggests that the behaviour arises from a (subconscious or conscious) intent to imitate more in the Watched condition, supporting the hypothesis that imitation is a social signal.

### *4.5.2. Brain responses to watching and performing irrational actions*

In our study pairs of naïve participants were assigned the role of Leader and Follower randomly, and the Leader was explicitly and secretly instructed to make irrational

movements, while the Follower was not given this instruction. For Leaders observing their interaction partners' irrational movements, activation in the right TPJ was parametrically modulated by action rationality. In the Followers activation in the right angular gyrus (part of the right IPL) was parametrically modulated by action rationality.

Previous studies have shown increased activation in the right TPJ or adjacent areas when observing irrational actions (Brass et al., 2007; Marsh, Mullett, et al., 2014 reported activation in the right IPL extending to the right TPJ) or when observing actions that are incongruent with expectations (Brass et al., 2009; Saxe et al., 2004). Marsh and Hamilton (Marsh & Hamilton, 2011), meanwhile, reported greater activation in the right IPL when viewing irrational actions. These studies all involved watching irrational actions passively. However, a number of studies have implicated the right IPL in preparing to move. Sirigu et al. (2004) report that the angular gyrus (part of the IPL) generates internal representations of action before performance, while another study showed that activation in the right IPL predicted subsequent imitative fidelity (Frey & Gerry, 2006). This may explain the difference in activation patterns seen in Leaders and Followers: in our paradigm, Leaders observed Followers make their movements at the end of each trial; Followers meanwhile were observing Leaders with the expectation of making their own movement immediately after. This interpretation is supported by a study by Oliver et al. (2017) who used a similar block-moving task with rational and irrational trajectories, and showed an increase in activation in right IPL when observing irrational actions prior to performing movements. The TPJ is part of the human mentalising network (Amodio & Frith, 2006) and is active when people contemplate others' intentions and beliefs. The IPL meanwhile forms a core of the human mirror-neuron system (MNS) which is part of the action observation and production network in the brain. These results support the view that both the action observation and mentalising networks are involved in processing and preparing to imitate irrational actions.

Performing irrational actions (moving with a higher trajectory) led to deactivation in the right SPL in the Followers; no regions in the Leader were parametrically modulated by the height performed. While the SPL has been found to be involved in the copying of whole-arm postures (Tanaka & Inui, 2002), the direction of the effect here runs counter to what we would expect if it was related to moving the arm higher since here higher arm movements lead to greater deactivation in the right SPL. This suggests that it may be an effect of having to perform an irrational action, rather than merely a motor response to making a higher movement. Given the design of our study it is not possible to disambiguate the effect of moving higher from making an irrational movement.

Subsequent studies could be designed to separate these two effects by having both the rational and irrational movements have identical kinematics, with rationality solely manipulated by the placement of barriers in the path. While a number of studies have utilised this obstacle-priming paradigm (Forbes & Hamilton, 2017; Griffiths & Tipper, 2009), none so far to our knowledge have combined this with neuroimaging.

### *4.5.3. Brain responses to being watched*

Audience effects are challenging to study using neuroimaging, but this study sought to overcome this using a relatively believable paradigm (see Section 2.5) and using fNIRS to capture neural signals. We found significant deactivation in neural signals around the right TPJ and right IPL in participants in the Watched condition compared with the Unwatched condition. This was our most robust result with multiple adjacent channels showing significant activations, thereby passing the test for multiple comparisons (Pinti, Devoto, et al., 2020; Southgate et al., 2014).

This deactivation stands somewhat in contrast with other papers that have shown that the mentalising network is engaged in the presence of direct gaze (Wang, Ramsey, et al., 2011), when the belief that we are being watched is induced indirectly (Somerville et al., 2013) or manipulated at an abstract level, such as when we feel we are being evaluated in some way (Bengtsson et al., 2009; Izuma et al., 2010; Müller-Pinzler et al., 2015). However, in all these cases, the activation in the mentalising network is found more rostrally, in the mPFC rather than the right TPJ.

Here we review some potential explanations for this intriguing result. In our experiment the Follower was not passive in either the Watched or Unwatched condition; rather in both cases the Follower was executing movements. Based on this, several possible explanations arise for the deactivation seen in the Watched condition versus the Unwatched condition.

First, we know that in the Watched condition Followers tended to copy the Leader's movements with greater fidelity. While the trajectories in the rational condition are similar across both Watched and Unwatched conditions, Followers tended to move higher in the irrational condition in the Watched condition rather than the Unwatched condition. Thus, the deactivation in the parietal cortex in the Watched condition could be linked to the effect of moving higher in the Watched condition. However, the GLM included a parametric regressor of the height of the Follower's movements, making this less likely.

Second, it could relate to the degree of motor planning required. If we assume that simply following what the Leader does (and not generating one's own motor plan) is less cognitively demanding, then Followers would be able to follow the Leader's motor plan in the Watched-rational and Watched-irrational conditions as well as the Unwatched-rational conditions. It is only in the Unwatched-irrational condition where the Leader would make a high movement, and the Follower would (as seen in the behavioural results) be more likely to respond without copying the Leader's trajectory with great fidelity. Potentially, this could involve the motor cortex, explaining the greater activation in this region in the Unwatched-irrational condition. However, we note that this would be an interaction effect rather than a main effect of being watched per se. Our study was not designed to capture this interaction effect in our planned comparisons. In future studies it would be worthwhile to plan these comparisons in advance.

A third strand of explanation centres on the possibility that being watched could be cognitively more demanding, having an impact on neural activity. On one hand, being watched may require more cognitive resources as we attempt to take an intentional stance or monitor our partner's attention. This may take resources away from controlling or inhibiting our tendency to imitate, leading to greater imitation in the Watched condition. This may also fit in with the reduction in neural activity in the TPJ seen in the Watched condition, since there is evidence that the TPJ is involved in controlling imitation (Brass et al., 2001, 2005; Hogeveen et al., 2015; Santiesteban et al., 2012, 2015). Previous studies that have involved just being watched (see Section 1.4.3) have usually shown that mentalising regions, including the mPFC and the TPJ, are engaged when we encounter direct gaze or are being watched (Cavallo et al., 2015; Dravida et al., 2020; Wang, Ramsey, et al., 2011). This would be an interesting avenue to explore in future work, particularly if we could scan the entire brain to see what is happening elsewhere during the Watched and Unwatched condition.

A final explanation is that not being watched may be the more atypical condition[11]. Remembering that our interaction partner cannot see us may require more mentalising since it is an unusual condition in a typical pair-wise interaction. The uniqueness of the situation may make it more demanding in terms of perspective-taking or mentalising. This is another idea worth exploring more deeply. In existing research on being watched versus not being watched, we have not seen mention of this dynamic (see Section 1.4.3) and instead have seen mentalising networks more engaged when being watched. But

[11] We would like to thank Gergely Csibra for this valuable suggestion.

again, whole brain scans will give us more information on what is going on elsewhere in the brain while participants are doing this task, and inform these tentative hypotheses.

### *4.5.4. Extending the GLM*

In simultaneously recording signals from both members of the dyad, we also found that a GLM that incorporated the neural signals of an interaction partner outperformed a GLM that only included behavioural signals from both participants in an interaction even after correcting for the increase in the number of parameters. This supports the view that neural signals in both participants are mutually predictive beyond what is captured in their instantaneous behavioural measurements. This builds on an innovative analytical approach advanced by Kingsbury et. al. (2019) when analysing neural signals in pairs of interacting mice. However, it remains to be seen whether the improvement in the GLM seen is specific to the partner, or whether incorporating the neural activations from anyone doing the task would outperform a GLM without neural data from an interaction partner. That is, we need to mitigate the problem of brain signals being synchronised owing merely to receiving the same stimuli in the same environment, or what is known as the 'problem of common input' (Burgess, 2013; Hamilton, 2020). In our experimental design different pairs saw trials in randomised order, so it was not possible to synchronise the neural data and compare real pairs with pseudo pairs, but in future work designed to test this hypothesis, we could present trials in the same order for different dyads and then compare the improvement seen in the GLM for genuine and pseudo pairs to parse this hypothesis.

### *4.5.5. Limitations and future directions*

These results have some limitations, both relating to the paradigm and to technical challenges. First, our study assumes that consistent placement of the cap on all subjects allowed us to record from the same brain regions throughout. However, Oliver et al. (2017) suggest that this may not always be valid. The limitations of our testing environment meant that we were unable to undertake a spatial registration of the optode positions for each participant. Future studies should attempt to do so to test this assumption rigorously and exclude channels that deviate too far from the group mean.

Second, the design of the experiment means that we cannot differentiate between the effect of moving higher versus making an irrational movement. On one hand this allowed us to design an experiment that did not explicitly call attention to the trajectory, and to test the replicability of the results found in the studies described in Chapter 3.

However, this meant that the brain response to irrationality is confounded with the brain response to moving higher. One mitigant here is that we found that performing irrational actions, which involve moving with a higher trajectory, led to a deactivation in the SPL in Followers. As we note above (in section 4.5.2) the SPL is implicated in the imitation of whole-arm movements (Tanaka & Inui, 2002), but the direction of this effect runs counter to what we would expect if it was related to the height rather than the rationality of the movement. Nevertheless, replicating these effects with a different kind of paradigm, such as one where the movements are same throughout with rationality manipulated by the presence of an obstacle in the path would increase the robustness of our findings. Finally, our design does not distinguish between conscious and unconscious copying or allow us to ascertain whether Followers became aware of the Leader's trajectory during the experiment.

The sample size of the present study was not large enough to test for fine individual differences. However, we highlight two avenues where further investigation of these differences may be productive. First, some Followers exhibited greater imitative fidelity than others; we found no meaningful links between their self-reported traits measured via the questionnaires and their propensity to imitate, but larger studies may be better equipped to study these. Second, the mutual prediction account advanced by Kingsbury et al. (2019) found that the degree to which including the interacting partner's brain signals improved the predictive capability of the GLM was related to the subordinate-dominant relationship between the two animals. Our study was setup between two naïve participants who were treated as equals, and this is also seen in how including the interaction partners brain signals improved the predictive capacity of the GLM for both Leaders and Followers. However, in future research, setting up a more hierarchical experiment with a clear dominant-subordinate dynamic would enable us to study whether this would have an impact on both behaviour and mutual prediction using neural signals.

This study offers behavioural support for the hypothesis that imitation is a social signal and sketches out the neural mechanisms in the mentalising and action observation networks that are involved in sending this signal. The content of this signal is yet to be fully parsed, however. Extant research suggests that imitation increases affiliation and pro-social behaviour (Chartrand & Bargh, 1999; Lakin & Chartrand, 2003; Müller et al., 2012; Stel & Vonk, 2010; van Baaren et al., 2003). Examining whether the neural mechanisms are modulated by affiliative goals, whether they change for example based

on the group membership or status of the target, is another rich vein worthy of exploration.

## 4.6. Conclusions

Overall, this research supports the social-signalling hypothesis of imitation by showing that participants encode the rationality of observed actions in the mentalising and action-observation networks, and the social availability of the interaction partner in the mentalising networks of the brain. This study also replicates our previous research (Krishnan-Barman & Hamilton, 2019) showing that people imitate an interaction partner with greater fidelity when they know that their partner is watching them. A joint analysis of hyperscanning data supports a mutual prediction account of neural signals that go beyond the matching of behavioural responses.

# Chapter 5. Imitation as a social signal in autism

This study extends the social-signalling hypothesis of imitation by comparing the behaviour and neural correlates of imitative behaviour among neurotypicals and autistic adults. Existing research paints a picture of great heterogeneity in imitative abilities and deficits among those with autism, and this study seeks to improve our understanding of whether autistic participants also imitate to send a social signal. We examine the fidelity with which participants imitate irrational actions and whether this is modulated by the social availability of their interaction partner. We also evaluate the neural correlates of observing irrational actions, encoding whether they are watched or not, as well as how we process being copied ourselves.

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## 5.1. Abstract

This thesis has focused on evaluating the social-signalling hypothesis of imitation which posits that imitation can serve as a social signal. The evidence from previous studies (in Chapters 3 and 4) conducted on neurotypical participants shows that they tend to imitate the irrational features of actions demonstrated by an interaction partner and do so with greater fidelity when they know their interaction partner can watch them. In this study we sought to extend this hypothesis by examining the differences between neurotypical and autistic participants. Autism is thought to be accompanied by imitative deficits, although the evidence on their extent is mixed. Here we examined differences in behaviour as well as in neural correlates across both groups in a modified version of our dyadic block-moving task and found that both groups imitated the irrational trajectories shown, and the imitative fidelity in both groups was modulated by the social availability of their interaction partner. Despite similar behavioural outcomes we saw some differences in the neural correlates of responding to irrational actions, being watched, and being copied between neurotypicals and autistic adults. This suggests that the same behaviour (of using imitation as a social signal) may arise from different brain mechanisms between neurotypical and autistic participants.

## 5.2. Introduction

A variety of explanations have been advanced for imitative behaviour including that it is a side-effect of domain-general processes (Darda & Ramsey, 2019; Heyes, 2017), that it enables us to build new skills (Flynn & Smith, 2012), or that it serves as a social-glue (Chartrand & Lakin, 2013; Lakin et al., 2003; Lakin & Chartrand, 2003). This thesis has focused on testing the social-signalling hypothesis of imitation which builds on the social-glue explanation and the STORM model (Wang & Hamilton, 2012) to claim that imitation is a social signal sent to an interaction partner. The test of whether this hypothesis is true rests on whether imitation in a pair-wise interaction is then modulated by the social availability of the interaction partner, namely whether the partner is watching the potential imitator make their movements, or not. Extant research in this regard has been mixed, with some studies showing that the degree of imitation is modulated by the social context (DiYanni et al., 2011; Marsh et al., 2019; Nielsen & Blank, 2011); other studies meanwhile have shown that that children overimitate even when the demonstrator is absent (Lyons et al., 2007), and that both children and adults overimitate when they are not aware of being watched (Whiten et al., 2016). While these latter studies argue against imitation being a social signal, they also involve novel tasks or ambiguity over whether the absence of the demonstrator implies the participant is not being watched. A more recent study showed that children overimitate when the demonstrator watched them *and* when the demonstrator left the room, but not when she turned away from them in the same room, suggesting that active disengagement was the variable that modulated imitative fidelity (Marsh et al., 2019).

The social-signalling hypothesis of imitation (outlined in Section 1.3.2) is concerned with whether people imitate an interaction partner's irrational actions, and whether this is modulated by the social availability of the interaction partner. In the context of a pairwise interaction between a demonstrator and a responder (who acts following a demonstration), the social-signalling hypothesis would posit that the following would occur:

(a) The responder should recognise (either consciously or subconsciously) that the action demonstrated is irrational, as reflected in differential brain activity between the rational and irrational conditions
(b) The responder should recognise the social availability of the demonstrator; that is, brain activity in the watched and unwatched conditions should be different
(c) The responder should copy the demonstrators' actions, including potentially the demonstrator's irrational actions

(d) The degree to which the responder copies the irrational action should be related to the social availability of the demonstrator; that is, they should copy the irrational action more closely when the demonstrator is watching them
(e) Further, if imitation is a social signal, then the neural correlates of being copied should differ from the neural correlates of not being copied

In a series of studies (Chapter 3 and 4) we tested the behavioural and neural correlates of this using pairs of naïve participants in our dyadic block-moving task (see Section 2.5.1). In each pair one participant was randomly assigned the role of the Leader and asked to demonstrate the order in which to move blocks to a Follower, using both rational and irrational trajectories. The Follower was unaware of the manipulation of the trajectories and was merely asked to move the blocks in the same order as the Leader. We also manipulated whether the Leader watched the Follower make their moves. Across three studies we found that Followers reliably imitated the Leader's irrational trajectories to a greater extent when the Leader was watching them rather than in trials where they knew the Leader had their eyes closed. This is in line with the behavioural response expected in (c) above. In Chapter 4 we also tested (a) and (b), looking at neural correlates in the right hemisphere fNIRS. Here we found that watching irrational actions led to an increase in activation in right IPL among naïve participants. This supports the view that participants identify actions as irrational. We also found a robust effect of being watched on the right parietal cortex, supporting the hypothesis that participants identify whether their interaction partner watches them. Taken together this offered support for the social-signalling hypothesis of imitation.

In the present study we sought to extend this by comparing neurotypical (NT) and autistic adults. Autistic people show some imitative impairments when compared with neurotypicals although the evidence on the universality and extent of these deficits has been disputed (see Section 1.5.2). When it comes to the social-signalling hypothesis of imitation we do not know if people with autism will identify actions as irrational to the same extent as neurotypicals, whether they will encode the feeling of being watched in the same way as neurotypicals, whether they will imitate irrationally high trajectories, and whether the extent of their imitation will be modulated by the social availability of their interaction partners. We also do not know if both neurotypical and autistic people will show similar responses to being copied by an interaction partner. These are the main questions that this study sought to answer.

### *5.2.1. Imitation of irrational actions in a social context: the evidence from autism*

In this section we review the existing evidence on how people with autism process social interaction, potential imitative impairments in autistic people, and how those with autism respond to rational and irrational actions. Impairments in social interaction are one of the defining features of autism; individuals with ASC often have trouble understanding social norms or socially-relevant cues (Frith, 2003; Schilbach et al., 2012). One reason for this could be that autistic people fail to orient attention towards relevant social cues (Klin et al., 2002; Pelphrey et al., 2002). Indeed Marsh and colleagues (2014) show that individuals with ASC show reduced attention to features of an action and simultaneously an impairment in their ability to predict goals; however looking only at trials where ASC participants paid attention to the action features, their performance matched that of neurotypical controls. The behaviour of those with ASC also does not show the same extent of modulation based on social context that is seen in neurotypicals: studies have shown that gaze and social engagement do not modulate copying or learning in those with ASC to the same extent as in matched controls (Vivanti et al., 2016; Vivanti & Dissanayake, 2014). Other studies have shown that typically developing children show a susceptibility to an audience effect that is not exhibited by autistic children (Chevallier et al., 2014); here typically-developing children showed an improvement in performance on a theory-of-mind task when they were watched which was not replicated in those with ASC. These results suggest that those with ASC may be less prone to modulate their behaviour based on whether their interaction partner is watching them or not.

Although impairments in imitation were not initially observed (Asperger, 1944; Kanner, 1943), both diagnosticians who first identified the condition noted that those with autism frequently failed to learn from others (Vivanti & Hamilton, 2014). In the subsequent decades imitation in autistic people has been extensively studied, but the evidence on imitative impairments is mixed (see Section 1.5.2). Two extensive reviews have supported the view that those with ASC exhibit imitative impairments (Edwards, 2014; Williams et al., 2004). However, other studies have shown little or no difference in imitative abilities between neurotypical and autistic people (Dapretto et al., 2006; Libby et al., 1997; Press et al., 2010; Sowden et al., 2016). Nevertheless, as Vivanti and Hamilton (2014) note the idea of imitative impairments among autistics is deeply ingrained, and these impairments are often used for diagnosing ASC despite it not being one of the core measures recommended in the DSM-5 (American Psychiatric Association, 2013).

It has been suggested that some of these contradictory results could be attributed to the diverse methodologies used in the studies, involving novel or routine tasks, automatic or voluntary imitation and varying social contexts (Sevlever & Gillis, 2010). Indeed Edwards (2014) notes that there appears to be great heterogeneity in imitative deficits, although the degree of impairment appeared to be related to the severity of ASC symptoms. While the broken-mirror hypothesis (Iacoboni & Dapretto, 2006; Ramachandran & Oberman, 2006; see Section 1.5) suggests that these imitative impairments in those with autism arises from a dysfunction in their MNS, this this has been challenged by later work that has shown that not all forms of imitation are equally impaired in those with autism (Hamilton, 2008; Southgate & Hamilton, 2008). This empirical evidence also ties in with alternate theoretical accounts of imitation, namely the EP-M and the STORM model (see Section 1.4.1) which suggests that rather than a global impairment in imitation, specific mechanisms are differentially impaired in those with autism. These specific mechanisms include the M-route, or mimicry route, in the EP-M model, while the STORM model claims that imitative impairments in autistic people arise from atypicality in the top-down regulation of the MNS. These dual-route and top-down regulation explanations are not mutually exclusive, and are supported by evidence showing that those with autism copy the style of an action when it is necessary to achieve a goal, but not when it is incidental, unlike neurotypicals who copy style in both scenarios (Hobson & Hobson, 2008; Hobson & Lee, 1999)

An alternate explanation is that differences in imitation task performance between neurotypical and autistic individuals rest on the fact that imitation requires good self-other distinction (Brass et al., 2009), a phenomenon that may be compromised in those with autism. Studies have shown that imitative abilities are impaired in those with prefrontal or TPJ lesions (Spengler, von Cramon, et al., 2010) or others where their self-other distinction was experimentally manipulated (Spengler, Brass, et al., 2010). These studies also found that mentalising ability was positively correlated with the ability to control imitative responses, supporting the view that mentalising and imitation are linked. Mentalising impairments have been frequently reported in those with ASC (Baron-Cohen et al., 1985; Frith, 2001). Several studies have shown that autistic people have difficulties comprehending stories involving others' mental states (Jolliffe & Baron-Cohen, 1999) or describing sequences where animated shapes behave with implied intentionality (Castelli, 2002).

An intriguing result from Dapretto and colleagues (2006) showed that both neurotypicals and those with ASC imitate equally well but found differences in the

associated neural correlates, with ASC participants showing reduced response in the IFG when compared with neurotypicals. Taken together these studies and reviews suggest that imitative deficits in autistic people are heterogenous and vary based on both symptom severity as well as the demands of the task. For our task we expect those with ASC to imitate their interaction partners with less fidelity when compared with neurotypicals and exhibit a different pattern of neural activation when doing so.

Finally, we turn to the question of action rationality, which comprises a key step in the kind of imitation engendered in our task. As noted in Chapter 1 (Section 1.4.2) observing and reproducing actions involves considering whether the actions demonstrated are congruent with expectations given the goals and the context. Studies have shown that violations of expectations are accompanied by activations in the STS (Grèzes et al., 2004; Pelphrey et al., 2003; Saxe et al., 2004) and the MTG (Jastorff et al., 2011; Marsh, Mullett, et al., 2014), with activation in the latter region found to be positively correlated with the (ir)rationality of the action. Irrational actions have also been shown to lead to increased activations in the TPJ and IPL (Brass et al., 2007; Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011; Oliver et al., 2017), while evidence on the mPFC has been mixed with some studies showing deactivations in the mPFC for irrational actions (Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011), while others have shown an increase in activation when viewing irrational actions (Brass et al., 2007; Decety & Chaminade, 2003). Broadly these regions comprise the action-observation and mentalising regions of the brain, and several studies have shown that autistic people exhibit impaired functioning in both action-observation (Iacoboni & Dapretto, 2006; Ramachandran & Oberman, 2006) and in mentalising (Baron-Cohen et al., 1985; Castelli, 2002; Frith, 2001; Frith & Frith, 2003; Jolliffe & Baron-Cohen, 1999). However, evidence on whether those with autism interpret action rationality in a manner similar to neurotypicals is mixed. Some studies have shown that both neurotypical and autistic children tend to copy rational movements more than irrational movements, suggesting both groups are able to discriminate between rational and irrational actions equally well, while others have reported differences in the copying behaviour of autistic children and adults when compared with neurotypicals (D'Entremont & Yazbek, 2007; Hobson & Hobson, 2008; Hobson & Lee, 1999). Even the studies showing differences have demonstrated effects that run in opposite directions: the study by D'Entremont and Yazbek (2007) showed that neurotypical children only copied the rational movements of a demonstrator, while children with autism copied both the rational and irrational movements. However, the studies by Hobson and Hobson (2008) showed that neurotypicals copied the style of an action used to reach a goal even when the style was

not relevant to achieving the goal (i.e., the style was an irrational feature), while autistic people only copied the style of an action when it was relevant to achieving the goal. This is partially explained by differences between what neurotypicals and those with autism focus on. A study by Marsh and colleagues (2015) showed both rational and irrational actions to autistic individuals as well as neurotypicals: they found that those with autism showed reduced attention to features of the action such as the hand performing the action; however, in trials where participants with autism did focus on these features, their performance was similar to the neurotypicals. This suggests that the basic mechanisms of understanding and interpreting actions are intact in those with autism, but there are potential impairments in top-down processes that impact where they direct their focus. In terms of neural correlates, it has been found that both autistic and neurotypical participants showed activation in the right IPL when viewing irrational actions; neurotypicals also showed reduced activation in the mPFC, while autistic participants did not (Marsh & Hamilton, 2011). In line with this evidence, we would expect autistic participants in our study to encode irrational actions differently in their brains when compared with neurotypicals.

### *5.2.2. Current study*

As outlined above, this study is focused on four specific questions. First, whether autistic and neurotypical people identify irrational actions in similar ways; we expect that autistic participants will encode irrational actions differently to neurotypicals, and be less likely to show activation in the mentalising networks of the brain in response to viewing irrational actions. Second, we are interested in whether the two groups encode the effect of being watched in the same manner; in light of existing research outlined in the previous section, we expect neural activations of being watched to be different in neurotypicals and those with ASC. Third, when it comes to behaviour we expect that imitation in those with autism should be less susceptible to the social availability of their interaction partner; i.e., we expect that neurotypicals will modify their imitative behaviour when watched to a greater extent than autistic people. Finally, we expect neurotypicals to show a greater neural response to being copied, while we expect no significant effect in those with ASC.

However, it is not a foregone conclusion that there will be differences in behaviour and in neural correlates. There is a possibility that autistic participants may consciously or unconsciously behave in ways similar to neurotypicals which may be underpinned by similar or very different brain mechanisms. If the move to generate the same behaviours

as neurotypicals is conscious and effortful, it is often called camouflaging (Hull et al., 2017). However, our task design would not allow us to make strong claims in this regard. If we did find that neurotypicals and autistic individuals behaved in similar ways with different underlying brain mechanisms it would suggest that some form of ***cognitive compensation*** is taking place, wherein autistic participants adjust their behaviour to more closely match what neurotypicals would do.

To test these hypotheses this study is designed based on our dyadic block-moving task (see Section 2.5.1) and adapted to the constraints of the testing environment and the population being tested. Similar to the study described in Chapter 4, this study uses fNIRS to study neural correlates among those with ASC and neurotypicals. In addition to being safe and economical, fNIRS allows for much freer movement than other similar imaging technologies such as PET or fMRI (Pinti, Tachtsidis, et al., 2020). In our study design we are constrained to examining neural correlates on the cortical surface and given our areas of interest the optodes are centred on the bilateral TPJ, extending into the bilateral IPL. This will enable us to examine neural correlates in the MNS as well as the mentalising network, namely the bilateral TPJ.

## 5.3. Materials and Methods

### *5.3.1. Participants*

A total of 25 neurotypicals and 26 participants with ASC were recruited using the UCL Institute of Cognitive Neuroscience's autism@icn participant database. Seven participants (three neurotypicals and four with ASC) were excluded from the analysis based on the exclusion criteria detailed below in Section 4.3.3. We aimed for a sample size of 20 or more participants in each group and the final analysis was conducted on 22 neurotypicals and 22 participants with ASC. The final sample size was determined by the availability of participants from the autism@icn database during the testing period. Both groups were matched on gender, handedness, and on intelligence quotient (IQ) using the Wechsler Adult Intelligence Scale versions III and IV (WAIS-III, Wechsler, 1997, WAIS-IV, Wechsler 2008) but differed on AQ (Baron-Cohen et al., 2001). The WAIS-III had been administered to participants who joined the database in previous years; while newer participants who joined in 2019 were asked to complete the WAIS-IV. To ensure comparability across the two scales they were matched on full-scale IQ and verbal IQ as outlined in Table 4.1 below. Both groups had high IQ (higher than 80) on average since the ASC group was high functioning. ASC participants had a diagnosis of Asperger's syndrome (11), autism (5), or autism spectrum disorder (6), from an independent clinician.

The ASC participants were also tested on module 4 of the Autism Diagnostic Observation Schedule (ADOS-G, Lord et al., 2000, ADOS-2, Lord et al., 2012) by a trained researcher. 11 participants met the ADOS classification for autism, four for autism spectrum, while seven did not meet the classification for either autism or autism spectrum. However, all seven had a clear diagnostic history from an independent clinician. As outlined in Table 4.1 the groups were slightly imperfectly matched in age, with the neurotypical group slightly younger than the ASC group. We were constrained by the availability of participants who matched on the other criteria and accepted the slight mismatch in age based on the groups having similar ranges and standard deviations (see Table 5.1). All participants had normal or corrected-to-normal vision and hearing and had not participated in this experiment previously. Participants were reimbursed financially and provided informed written consent prior to participating. All procedures were approved by the UCL Research Ethics Committee (Approval ID: 5975/003).

**Table 5.1. Comparison of the Neurotypical (NT) and Autism Spectrum Condition (ASC) groups**

| | ASC ($n$ = 22) | | NT ($n$ = 22) | | $t$ test $p$ value |
|---|---|---|---|---|---|
| | Mean (SD) | Range | Mean (SD) | Range | |
| Age (years) | 33.8 (6.2) | 21-45 | 30.2 (5.9) | 19-39 | 0.06 |
| Fullscale IQ | 115 (13.9) | 81-138 | 112.3 (14.4) | 89-133 | 0.54 |
| Verbal IQ | 115.5 (14.7) | 92-153 | 114.5 (12.3) | 83-132 | 0.81 |
| Autism Quotient (AQ) | 32.2 (10.9) | 10-47 | 16.1 (7.2) | 7-33 | 0.00 |
| ADOS: total | 9 (4.3) | 1-17 | | | |
| ADOS: communication | 2.6 (1.6) | 0-6 | | | |
| ADOS: social interaction | 6.3 (3.1) | 0-13 | | | |
| Gender | 5 F; 17 M | | 6 F; 16 M | | |
| Handedness | 1 L; 21 R | | 2 L; 20 R | | |

### *5.3.2. Procedure*

When participants arrived for the experiment, they were told they would be participating in a team challenge with another participant who was a student at UCL. The participant was introduced to Experimenter A who was conducting the study, and Experimenter B who was operating the fNIRS equipment. The confederate arrived after the participant and introduced herself to the experimenters and the participant, to create the illusion that she was unknown to the experimenters. Experimenter A then asked both the participant and the confederate some demographic information (including how to spell their name and their age) and initiated some conversation on what they were studying or how their commute into the lab was today. Experimenter A then explained that the confederate was assigned the role of the Leader, and the participant that of the Follower. She then explained that the study was designed to look at how information is lost when transmitted from the computer to the Leader, and the Leader to the Follower (a fiction designed to distract participants from the true purpose of the study). The Leader was told they would have to move blocks in an order demonstrated by the computer, and the Follower was told they would have to move the blocks in an order demonstrated by the Leader. They were asked to select a team name together and told that they would be competing against other teams over the duration of the study. The participants were informed that their score for each trial would depend on how quickly and accurately they moved the blocks from one board to another. Prior to the experiment commencing, participants were fitted with equipment to measure physiological signals, track motion, and record neural signals. These are described in detail below in the sections on data acquisition.

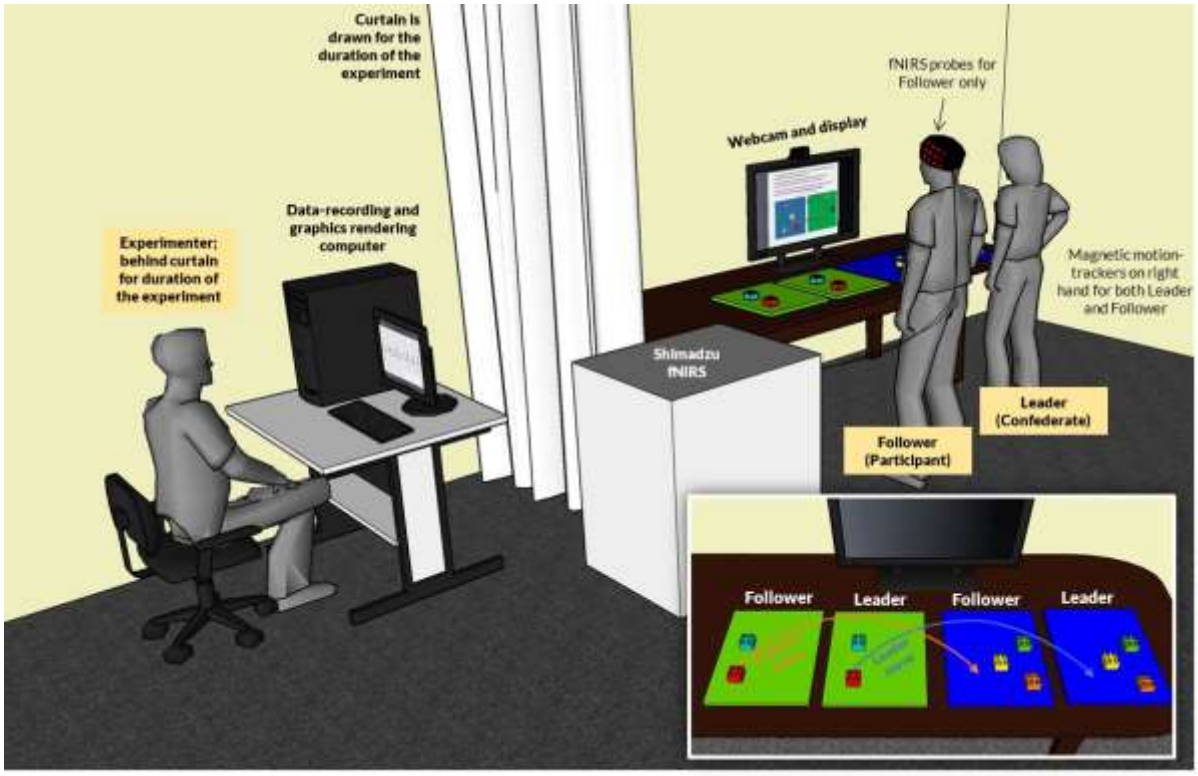


**Figure 5.1. An overview of the experimental setup.** Figure shows the lab with the functional near-infrared spectroscopy (fNIRS) equipment. The Experimenter remained behind a curtain throughout the experiment. The Leader was a trained confederate while the Follower was a neurotypical or ASC participant. The Follower's brain activity was measured using the fNIRS equipment bilaterally centred on the temporal-parietal junction in each hemisphere. In addition to a magnetic motion tracker, a webcam was also used to capture the social interaction. Followers also wore a belt tracking heart rate, breathing rate and galvanic skin response.

*Main and Switch trials*

At the start of each trial in the Main phase the Follower was instructed (via a computerised voice command) to close their eyes. The Leader (the trained confederate) was told which three blocks to move, and in what order, as well as whether they should be moved with a baseline or exaggerated trajectory. The Follower was then asked to open their eyes, and the Leader demonstrated by moving the blocks in a specified order, with either a baseline trajectory or an exaggerated trajectory. After moving, the Leader then covered their two boards (using two cloths) and pressed the button to move the trial forward. The Follower then had to move their blocks in the same order from one board to another. During the Follower's turn in half the trials the Leader was allowed to watch them, and they were allowed to speak to each other. In the other half of the trials, during the Follower's turn, the Leader was told to close their eyes. After the Follower finished their move, the Leader then uncovered their boards. The Leader and the Follower then rearranged blocks in case there were any errors (for example, if the Follower had moved the wrong block over). After this "rearrange" phase, the team received a score for one trial

based on their speed and accuracy (measured by how long the rearranging took) before moving on to the next trial. The Main trials thus had a 2x2 factorial design with two different trajectories (baseline and exaggerated) and whether the Leader had their eyes open or closed during the Follower's turn (Watched and Unwatched). The four conditions were repeated four times over the 16 experimental trials, with trajectories presented in a randomised order. The Watched and Unwatched trials occurred in blocks of four trials; the initial block was randomised.

After sixteen trials in the Main phase, participants were told they would switch roles. The Follower was then informed of the baseline and exaggerated trajectories and was explicitly told to imitate the trajectory demonstrated by the computer when showing the Leader which blocks the move. Each of these Switch trials began with the Leader now being asked to close their eyes, and the Follower being told which blocks to move and what trajectory to use (baseline or exaggerated) when moving. The Leader then opened their eyes, and the Follower demonstrated the block movement. The Follower then covered their boards. During the Leader's turn the Follower always watched them move the blocks. The Leader was secretly instructed to copy the Follower's trajectory or not copy the Follower's trajectory. As before, this was followed by a rearrange phase to correct for any mistakes and to generate a score for that trial. The Switch trials thus had a 2x2 factorial design with two different trajectories (baseline and exaggerated) and whether the Leader copied the Follower or not (Copy / Not Copy). The four conditions were repeated twice over the 8 experimental trials, with trajectories presented in a randomised order. The Copy and Not Copy trials occurred in blocks of four trials; the initial block was randomised.

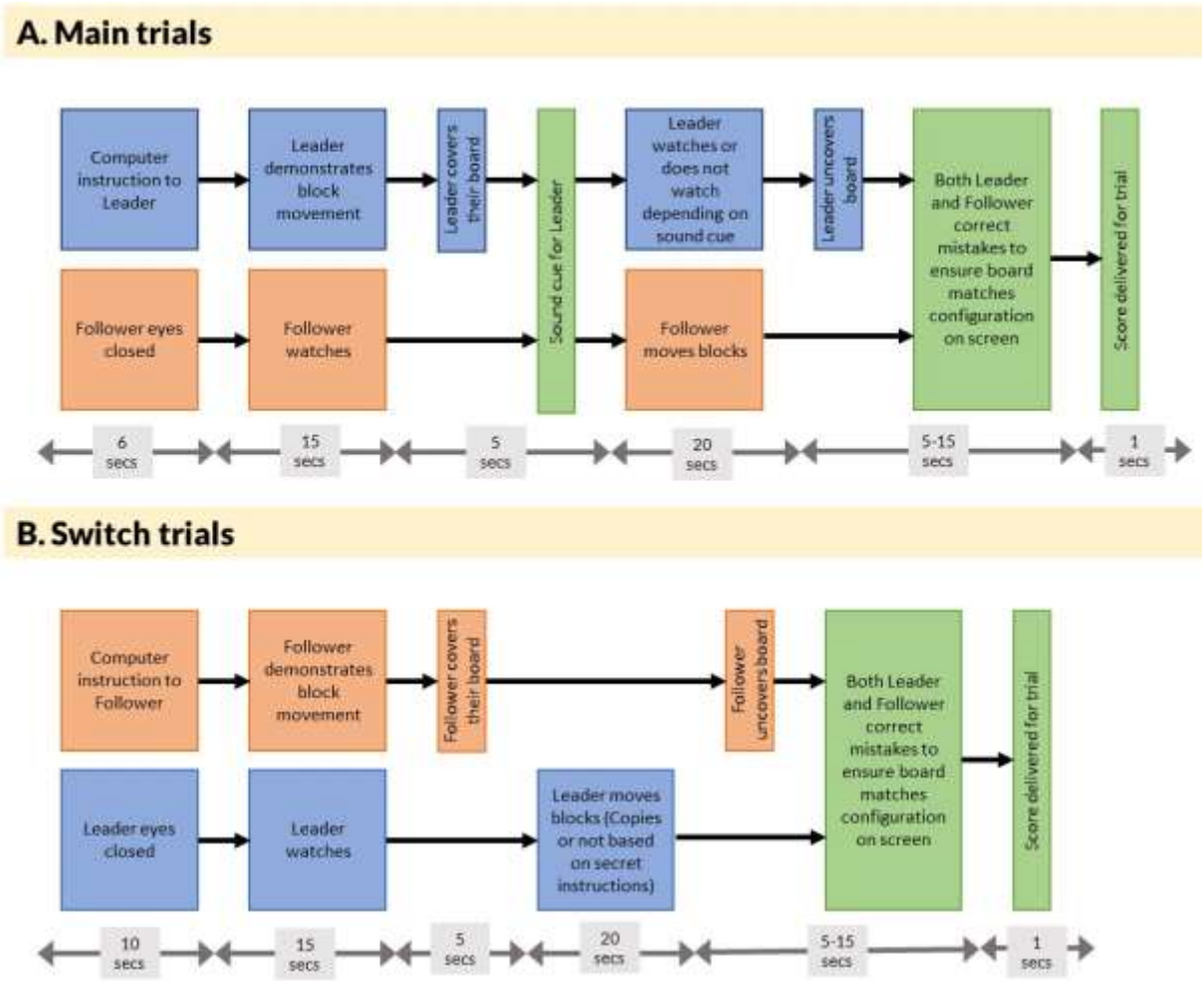


**Figure 5.2. Timeline of Main trials (A) and Switch trials (B).**Figure shows the Leader and Follower roles in the Main and Switch trials along with typical length of each stage in seconds.

As noted in Section 5.3.1 participants also completed an AQ questionnaire (Baron-Cohen et al., 2001) and intelligence tests ((WAIS-III, Wechsler, 1997, WAIS-IV, Wechsler 2008). Participants with ASC were also tested on module 4 of the Autism Diagnostic Observation Schedule (ADOS-G, Lord et al., 2000, ADOS-2, Lord et al., 2012) by a trained researcher. These questionnaires were administered at a separate time and not in conjunction with the experiment itself.

### *5.3.3. Behavioural analysis*

The main parameter used to evaluate imitative behaviour was the height reached by the Follower in each trial. The confederate was trained to reach a uniformly high peak height for the exaggerated condition and a uniformly low peak height for the baseline condition. Based on this, if the Follower reached a high peak height in a trial where an exaggerated trajectory had been demonstrated, this suggests that the Follower imitated the exaggerated trajectory. Given that each trial involved moving three blocks, in our view

peak height is the most salient measure of the fidelity with which a movement trajectory was copied. The peak height was then used to evaluate imitative fidelity across the Watched and Unwatched trials. The peak height was also measured in the Switch trials to ensure the Follower understood the principle of exaggerated and baseline trajectories and showed a difference in the heights they reached for each trial type.

*Behavioural exclusion criteria*

Dyads were excluded for the following reasons:

(i) Data was not recorded owing to equipment failure or failure in the task software;
(ii) The Follower specifically asked the Leader about the trajectories they were demonstrating in the Main trials;
(iii) The Follower failed to follow instructions to close their eyes at various points in the trial;
(iv) In the Switch trials if Followers failed to move higher in the trials where they were asked to demonstrate an exaggerated trajectory when compared with trials where they were asked to demonstrate a baseline trajectory.

Out of the 51 participants who were tested, seven participants had to be excluded (all for failing criteria iv), leaving 44 participants for whom the behavioural data could be analysed.

### *5.3.4. Acquisition and analysis of physiological signals*

Prior to the experiment commencing, Followers were asked to wear a device that allowed us to record physiological signals (heart rate, breathing rate and galvanic skin response). Since this belt was required to be worn under their clothes (and would necessitate them changing in privacy) participants were offered an option of not wearing the belt if they felt uncomfortable doing so. Participants who agreed to wear the belt were then measured and fitted with a suite of equipment based around the Equivital Belt (ADInstruments, Dunedin, New Zealand). This recorded both heart rate and breathing rate. The heart rate is measured using three electrodes placed in contact with the skin after being moistened, and the signal is measured at 256Hz. The breathing rate was measured based on the expansion of the belt, and the signal is measured at 25.6Hz. In addition, the galvanic skin response was measured using a wrist-mounted auxillary device, which uses two electrodes placed on the middle and index fingers of the non-dominant hand. The signal

is recorded at 12.8Hz. The belt includes a sensor electronics module (SEM) that stores all three signals and is synchronised with the experimental computer at the start of each experiment. The physiological data was synchronised with the fNIRS data and processed using MATLAB®. The processed data was then synchronised with the trials and for each trial a mean signal was generated by averaging the signal throughout the trial.


**Figure 5.3. The Equivital Belt system with the Sensor Electronics Module.**

### *5.3.5. fNIRS data acquisition*

Neural signals were acquired using the LABNIRS system (Shimadzu Corporation, Kyoto, Japan). The LABNIRS system uses multiple wavelengths (780/805/830nm) at a sampling frequency of 7.4Hz. At the analysis stage this was downsampled to 1Hz to reduce temporal autocorrelation. A spandex cap fitted with a rigid probe holder was used to hold the probes, allowing us to fit participants with varying head sizes, while keeping a source-detector distance of 3cm. For each participant we measured the distance from the nasion to the inion and marked the centre point of this. Similarly, the centre point was marked between the left and right auricular points. Taken together this allowed us to locate the mid-point of the head, and line the cap up appropriately.

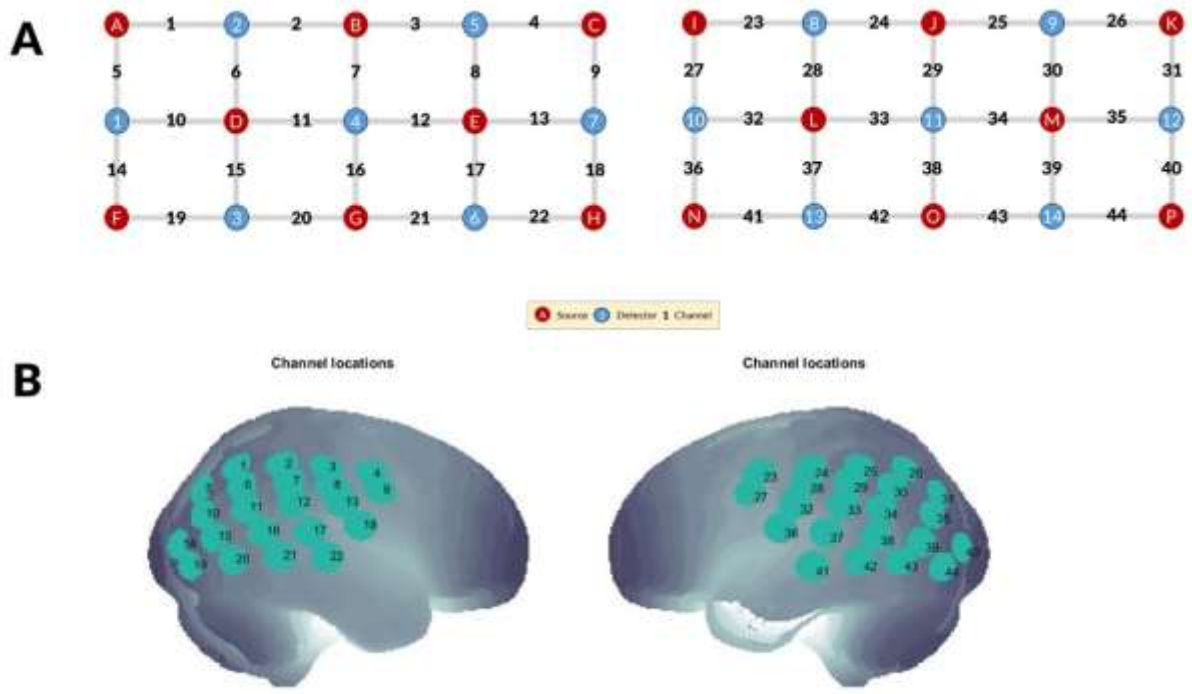


**Figure 5.4. Optode Configuration. A.** Diagram showing the positions of light sources (red), detectors (blue) and the 44 channels created by the 30 probes. **B.** Locations of the 44 channels on the left and right hemisphere for each participant.

## *fNIRS optode localisation*

The location of each optode and five canonical head locations (inion, nasion, right preauricular, left preauricular and vertex) were recorded for each participant using the Polhemus Liberty (Colchester Vermont) magnetic motion tracker. Here, the metallic stylus was used to mark the specific points in a magnetic field relative to the origin, enabling us to digitise the positioning of each optode. This was done for each participant once the cap was positioned; however, if after three attempts we were not successful (typically owing to magnetic interference in the testing environment) in capturing the positions the digitization was skipped and a reference digitization was used.

The channel locations for each participant were averaged to generate canonical channel locations for all valid participants, and the corresponding MNI coordinates (Mazziotta et al., 2001) for each channel were obtained using the NIRS-SPM software (Ye et al., 2009) with MATLAB® and the corresponding anatomical locations of each channel were derived from the included atlas (Rorden & Brett, 2000). Table 4.2 lists the MNI coordinates, the anatomical regions, Brodmann Areas (BA) and the probability that each region is included in the channel. Only regions with a probability greater than 0.05 are included in the table below.

**Table 5.2 Channel coordinates and anatomical regions.** Channels are listed below alongside their Montreal Neurological Institute (MNI) coordinates (Mazziotta et al., 2001). The corresponding anatomical region labels are obtained from the NIRS-SPM software (Ye et al., 2009) based on the included atlas (Rorden & Brett, 2000). Alongside each anatomical region the corresponding Brodmann Area (BA) and the probability that this region is included in the channel are shown.

| Channel number | MNI coordinates | | | Anatomical region | BA | Probability of inclusion |
|---|---|---|---|---|---|---|
| | X | Y | Z | | | |
| 1 | 48.3 | -66.9 | 49.3 | Angular gyrus, part of Wernicke's area | 39 | 0.85 |
| 1 | | | | Somatosensory Association Cortex | 7 | 0.13 |
| 2 | 60.5 | -44.7 | 50.3 | Supramarginal gyrus part of Wernicke's area | 40 | 1.00 |
| 3 | 63.4 | -23.4 | 48.8 | Primary Somatosensory Cortex | 1 | 0.55 |
| 3 | | | | Primary Somatosensory Cortex | 3 | 0.21 |
| 3 | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.12 |
| 3 | | | | Primary Somatosensory Cortex | 2 | 0.06 |
| 3 | | | | Primary Motor Cortex | 4 | 0.06 |
| 4 | 59.0 | 1.0 | 45.3 | Pre-motor and Supplementary Motor cortex | 6 | 0.80 |
| 4 | | | | Primary Motor Cortex | 4 | 0.18 |
| 5 | 37.6 | -84.2 | 36.4 | V3 | 19 | 0.93 |
| 5 | | | | Angular gyrus, part of Wernicke's area | 39 | 0.05 |
| 6 | 56.2 | -65.1 | 38.3 | Angular gyrus, part of Wernicke's area | 39 | 1.00 |
| 7 | 65.7 | -41.9 | 40.8 | Supramarginal gyrus part of Wernicke's area | 40 | 0.98 |
| 8 | 67.5 | -19.3 | 38.8 | Primary Somatosensory Cortex | 1 | 0.44 |
| 8 | | | | Primary Somatosensory Cortex | 2 | 0.40 |
| 8 | | | | Subcentral area | 43 | 0.09 |
| 8 | | | | Primary Somatosensory Cortex | 3 | 0.07 |
| 9 | 62.6 | 4.5 | 36.1 | Pre-motor and Supplementary Motor cortex | 6 | 0.67 |
| 9 | | | | Primary Motor Cortex | 4 | 0.16 |
| 9 | | | | Subcentral area | 43 | 0.12 |
| 9 | | | | pars opercularis, part of Broca's area | 44 | 0.05 |
| 10 | 46.5 | -83.2 | 23.0 | V3 | 19 | 0.74 |
| 10 | | | | Angular gyrus, part of Wernicke's area | 39 | 0.26 |
| 11 | 61.5 | -61.4 | 27.0 | Angular gyrus, part of Wernicke's area | 39 | 0.60 |
| 11 | | | | Superior Temporal Gyrus | 22 | 0.35 |
| 12 | 69.0 | -39.0 | 29.2 | Supramarginal gyrus part of Wernicke's area | 40 | 0.50 |
| 12 | | | | Superior Temporal Gyrus | 22 | 0.23 |
| 12 | | | | Retrosubicular area | 48 | 0.22 |
| 12 | | | | Primary Somatosensory Cortex | 2 | 0.06 |
| 13 | 69.3 | -14.7 | 29.7 | Subcentral area | 43 | 0.49 |
| 13 | | | | Primary Somatosensory Cortex | 2 | 0.40 |
| 13 | | | | Primary Somatosensory Cortex | 1 | 0.10 |
| 14 | 35.3 | -96.8 | 6.2 | Visual Association Cortex (V2) | 18 | 0.70 |
| 14 | | | | Primary Visual Cortex (V1) | 17 | 0.26 |
| 15 | 53.9 | -77.4 | 10.4 | V3 | 19 | 0.66 |
| 15 | | | | Fusiform gyrus | 37 | 0.22 |
| 15 | | | | Angular gyrus, part of Wernicke's area | 39 | 0.12 |
| 16 | 67.0 | -53.9 | 13.6 | Superior Temporal Gyrus | 22 | 0.35 |
| 16 | | | | Middle Temporal gyrus | 21 | 0.34 |
| 16 | | | | Fusiform gyrus | 37 | 0.31 |
| 17 | 71.6 | -31.3 | 13.8 | Superior Temporal Gyrus | 22 | 0.89 |

| Channel number | MNI coordinates X | Y | Z | Anatomical region | BA | Probability of inclusion |
|---|---|---|---|---|---|---|
| 18 | 69.4 | -6.7 | 17.0 | Subcentral area | 43 | 0.60 |
| 18 | | | | Superior Temporal Gyrus | 22 | 0.31 |
| 18 | | | | Retrosubicular area | 48 | 0.08 |
| 19 | 44.6 | -89.4 | -5.1 | V3 | 19 | 0.61 |
| 19 | | | | Visual Association Cortex (V2) | 18 | 0.39 |
| 20 | 59.8 | -68.2 | -2.1 | Fusiform gyrus | 37 | 0.90 |
| 20 | | | | V3 | 19 | 0.10 |
| 21 | 70.7 | -45.6 | -0.2 | Middle Temporal gyrus | 21 | 0.43 |
| 21 | | | | Superior Temporal Gyrus | 22 | 0.26 |
| 21 | | | | Fusiform gyrus | 37 | 0.18 |
| 21 | | | | Inferior Temporal gyrus | 20 | 0.14 |
| 22 | 72.6 | -22.9 | -0.8 | Middle Temporal gyrus | 21 | 0.71 |
| 22 | | | | Superior Temporal Gyrus | 22 | 0.29 |
| 23 | -55.0 | 1.8 | 46.3 | Pre-motor and Supplementary Motor cortex | 6 | 0.87 |
| 23 | | | | Primary Motor Cortex | 4 | 0.11 |
| 24 | -60.5 | -23.0 | 48.7 | Primary Somatosensory Cortex | 3 | 0.40 |
| 24 | | | | Primary Somatosensory Cortex | 1 | 0.30 |
| 24 | | | | Primary Somatosensory Cortex | 2 | 0.17 |
| 24 | | | | Primary Motor Cortex | 4 | 0.07 |
| 24 | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.07 |
| 25 | -58.6 | -46.6 | 49.3 | Supramarginal gyrus part of Wernicke's area | 40 | 0.99 |
| 26 | -47.8 | -68.6 | 48.2 | Angular gyrus, part of Wernicke's area | 39 | 0.90 |
| 26 | | | | Somatosensory Association Cortex | 7 | 0.08 |
| 27 | -59.0 | 6.6 | 35.5 | Pre-motor and Supplementary Motor cortex | 6 | 0.68 |
| 27 | | | | pars opercularis, part of Broca's area | 44 | 0.18 |
| 27 | | | | Primary Motor Cortex | 4 | 0.11 |
| 28 | -64.6 | -18.2 | 39.1 | Primary Somatosensory Cortex | 1 | 0.38 |
| 28 | | | | Primary Somatosensory Cortex | 2 | 0.33 |
| 28 | | | | Primary Somatosensory Cortex | 3 | 0.17 |
| 28 | | | | Subcentral area | 43 | 0.13 |
| 29 | -64.3 | -42.8 | 39.3 | Supramarginal gyrus part of Wernicke's area | 40 | 0.92 |
| 29 | | | | Retrosubicular area | 48 | 0.08 |
| 30 | -56.0 | -66.0 | 36.6 | Angular gyrus, part of Wernicke's area | 39 | 1.00 |
| 31 | -39.2 | -85.5 | 33.8 | V3 | 19 | 0.92 |
| 31 | | | | Angular gyrus, part of Wernicke's area | 39 | 0.08 |
| 32 | -67.2 | -14.7 | 27.9 | Subcentral area | 43 | 0.48 |
| 32 | | | | Primary Somatosensory Cortex | 2 | 0.31 |
| 32 | | | | Retrosubicular area | 48 | 0.11 |
| 32 | | | | Primary Somatosensory Cortex | 1 | 0.07 |
| 33 | -67.4 | -39.5 | 27.5 | Retrosubicular area | 48 | 0.34 |
| 33 | | | | Superior Temporal Gyrus | 22 | 0.31 |
| 33 | | | | Supramarginal gyrus part of Wernicke's area | 40 | 0.28 |
| 33 | | | | Primary Somatosensory Cortex | 2 | 0.06 |
| 34 | -61.6 | -60.7 | 24.8 | Angular gyrus, part of Wernicke's area | 39 | 0.50 |
| 34 | | | | Superior Temporal Gyrus | 22 | 0.31 |
| 34 | | | | Fusiform gyrus | 37 | 0.08 |
| 34 | | | | Middle Temporal gyrus | 21 | 0.06 |
| 35 | -47.5 | -83.3 | 22.4 | V3 | 19 | 0.77 |

| Channel number | MNI coordinates X | Y | Z | Anatomical region | BA | Probability of inclusion |
|---|---|---|---|---|---|---|
| 35 | | | | Angular gyrus, part of Wernicke's area | 39 | 0.23 |
| 36 | -66.6 | -6.9 | 14.3 | Subcentral area | 43 | 0.41 |
| 36 | | | | Superior Temporal Gyrus | 22 | 0.40 |
| 36 | | | | Retrosubicular area | 48 | 0.18 |
| 37 | -69.3 | -31.2 | 12.2 | Superior Temporal Gyrus | 22 | 0.85 |
| 37 | | | | Middle Temporal gyrus | 21 | 0.06 |
| 37 | | | | Primary and Auditory Association Cortex | 42 | 0.06 |
| 38 | -66.1 | -54.2 | 10.9 | Fusiform gyrus | 37 | 0.38 |
| 38 | | | | Middle Temporal gyrus | 21 | 0.37 |
| 38 | | | | Superior Temporal Gyrus | 22 | 0.26 |
| 39 | -54.8 | -76.4 | 7.7 | V3 | 19 | 0.68 |
| 39 | | | | Fusiform gyrus | 37 | 0.29 |
| 40 | -37.1 | -96.1 | 4.9 | Visual Association Cortex (V2) | 18 | 0.85 |
| 40 | | | | Primary Visual Cortex (V1) | 17 | 0.11 |
| 40 | | | | V3 | 19 | 0.05 |
| 41 | -70.4 | -23.7 | -5.7 | Middle Temporal gyrus | 21 | 0.94 |
| 41 | | | | Superior Temporal Gyrus | 22 | 0.06 |
| 42 | -68.6 | -46.1 | -3.0 | Middle Temporal gyrus | 21 | 0.41 |
| 42 | | | | Fusiform gyrus | 37 | 0.24 |
| 42 | | | | Inferior Temporal gyrus | 20 | 0.24 |
| 42 | | | | Superior Temporal Gyrus | 22 | 0.11 |
| 43 | -59.7 | -67.7 | -3.8 | Fusiform gyrus | 37 | 0.95 |
| 43 | | | | V3 | 19 | 0.05 |
| 44 | -45.1 | -88.5 | -6.3 | V3 | 19 | 0.78 |
| 44 | | | | Visual Association Cortex (V2) | 18 | 0.22 |

### *5.3.6. fNIRS analysis*

The raw light intensity data from the LABNIRS system for all three wavelengths was converted to concentration changes of HbO, HbR and total-Hb by the modified Beer-Lambert law using custom MATLAB® scripts and the preprocessing of data was done in HomER (Huppert et al., 2009). In line with the recommendations made by Pinti et al. (2019) we applied a bandpass filter (0.01 Hz to 0.3 Hz) to denoise the data.

#### *fNIRS exclusion criteria*

The data for each of the 44 channels across 44 valid participants was examined as follows. First, we visually inspected the Power Spectral Density (PSD) for each participant at each channel to look for a peak between 1-2Hz corresponding to the heartbeat oscillation. A lack of a heartbeat oscillation could suggest that the coupling between the optode and the scalp was poor. Second, the raw intensity data was inspected for saturation and artefacts. Third, the HbO and HbR signals were inspected to see if they were positively correlated (the signals should be negatively correlated with HbR having a much smaller magnitude than HbO). Based on these criteria individual channels were excluded. If any participant

had more than 50% of the channels in one hemisphere excluded, that hemisphere alone was excluded from the analysis. A total of six participants were excluded entirely, largely owing to them having very thick hair or fixed hairstyles which prevented optimal optode-skin coupling.

In the initial stage of the experiment, we used a slightly different configuration for the optodes, including the PFC and two 3x4 arrays of optodes over the bilateral TPJ. However, two out of the three participants who had this configuration were unable to complete the experiment due to discomfort in the forehead. We therefore dropped the PFC and used a configuration involving two 3x5 arrays as described above. These three participants were also excluded from the fNIRS analysis, bring the total number of participants whose neural signals were analysed to 35.

*Channel thresholding*

Since we had digitised the optode locations for participants wherever possible we were able to plot the actual positions of the channels for all participants as well as the canonical average channel centres. This is shown below in Figure 5.5A. The average distance between the canonical channel centres was 20.8mm. Based on this, we set a threshold of 10mm and excluded any channels that were more than 10mm away from their corresponding channel centre. Out of 1,284 valid channels available for analysis, 909 channels survived this thresholding (Figure 5.5 B.) and these are analysed below.

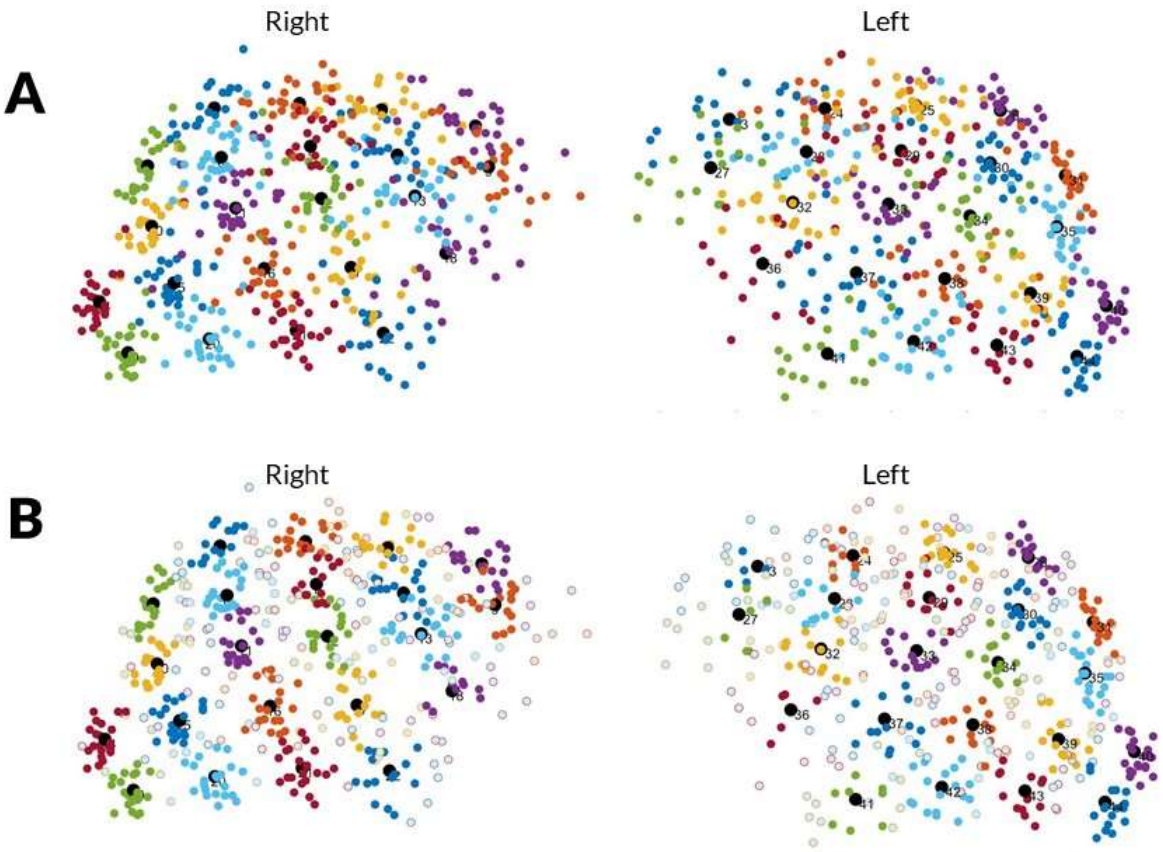


**Figure 5.5. Channel Thresholding. A**. Figure shows the canonical channel location (black) for each channel as well as the recorded position of that channel for each participant, in a different colour for each channel. **B.** Figure shows the channels that are more than 10mm away from the canonical channel location excluded (those with only the outline visible).

## *First-level analysis*

The first-level GLM analysis was performed using the fNIRS toolbox in SPM12 (Friston et al., 1994; Tak et al., 2016) implemented in MATLAB®. For this analysis, the HbO and HbR data were combined using the correlation-based signal improvement method (Cui et al., 2010), or CBSI, to generate a corrected activation signal. This is a noise-reduction algorithm that is built on maintaining the negative correlation between HbO and HbR and this combined CBSI signal is used in the remainder of this analysis. The data was recorded and analysed as two separate runs with the main trials analysed using a separate design matrix to the switch trials.

**Table 5.3. Contrasts.** The structure of the design matrix is shown below for the Main trials (A) and the Switch trials (B). The five contrasts of interest are highlighted showing how they are constructed, with +1 over each relevant column for forward contrasts, and a -1 over the relevant columns for reverse contrasts.

## A. Main Trials

Phases of Main trials modelled in design matrix

| Main Trials | Instruct | Leader Action | | Cover | Follower Action | | | | Rearrange | Score | Constant |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Watched | | Unwatched | | | | |
| | | Base | Exag | | Base | Exag | Base | Exag | | | |
| C1 Watching Leader perform irrational actions | | -1 | 1 | | | | | | | | |
| C2 Performing actions when watched vs unwatched | | | | | 1 | 1 | -1 | -1 | | | |
| C3 Responding to an irrational action | | | | | -1 | 1 | -1 | 1 | | | |

## B. Switch Trials

Phases of Switch trials modelled in design matrix

| Switch Trials | Instruct | Follower Action | | Cover | Leader Action | | | | Rearrange | Score | Constant |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Base | | Exag | | | | |
| | | Base | Exag | | Copy | Not Copy | Copy | Not Copy | | | |
| C4 Explicit demand for irrational action | | -1 | 1 | | | | | | | | |
| C5 Being imitated vs not being imitated | | | | | 1 | -1 | 1 | -1 | | | |

The design matrix for the Follower in the Main trials included two regressors for the Leader's action (Baseline and Exaggerated), and four regressors for the Follower's action depending on whether it was Watched or Unwatched, and whether it occurred after a Baseline demonstration or an Exaggerated demonstration by the Leader. Additional regressors for the instruction, cover, rearrange, score and constant phases of each trial were also included. Contrast 1 (C1) thus evaluates activation in the Follower when the Leader demonstrated an Exaggerated trajectory when compared with a Baseline trajectory. C2 meanwhile calculates activation when the Follower was watched by the Leader during their turn versus when the Follower was not watched by the Leader. Finally, C3 evaluates the Follower's activation during their own turn right after watching the Leader make an Exaggerated demonstration versus having watched the Leader make a Baseline demonstration. While C1 measures activation when watching an irrational action, C3 measures activation when responding to an irrational action (whether that is by copying an irrational action or ignoring it and performing a rational action instead).

When it comes to the Switch trials, the Follower design matrix included two regressors for the Follower's action (Baseline and Exaggerated), as well as four regressors for the Leader's action depending on whether the Leader's action followed a Baseline or Exaggerated demonstration by the Follower, and whether the Leader responded by copying or not copying the Follower's trajectory. Additional regressors were also

included for the instruct, cover, rearrange, score and constant phases. Using these regressors C4 calculates activation in the Follower when they are asked to make an exaggerated movement when compared with a baseline movement. C5 seeks to measure activation in the Follower when they are copied by the Leader versus when the Leader fails to copy their trajectory.

Both sets of analyses of the Main and the Switch trials were performed at the individual level before being combined at the group level as outlined below.

*Group-level analysis*

T-tests were conducted on the individual parameter values (β values) for each contrast in each valid channel separately for the neurotypical (NT) and the ASC groups. A t-test was also conducted to look for group differences between the two groups for each contrast. We used the individual channels as a unit of analysis given the high likelihood of false positive findings given multiple voxel comparisons (Hirsch et al., 2017). Given the limited number of planned comparisons and the relatively small sample size, the uncorrected p-values are presented below. However, where two or more adjacent channels show a significant effect for $p<0.05$ we consider this as a corrected significant result (Pinti, Tachtsidis, et al., 2020; Southgate et al., 2014).

## 5.4. Results

### *5.4.1. Follower height*

We ran a two-way mixed ANOVA to compare the effect of being watched, and the trajectory demonstrated (baseline or exaggerated) on the height performed by the Follower in both the NT and ASC groups. We found a significant effect of being watched on the height performed by the Follower [$F(1,42) = 7.29$, $p = 0.01$]. We found no main effect of trajectory or group, and no significant two- or three-way interaction effects.

**Table 5.4. Two-way Mixed ANOVA of Follower Height.**

**Tests of Within-Subjects Contrasts**

| Source | df | F | Sig. |
|---|---|---|---|
| Watch_UnW | 1 | 7.291 | 0.010 |
| Watch_UnW * Group | 1 | 0.089 | 0.767 |
| Error(Watch_UnW) | 42 | | |
| Base_Exag | 1 | 0.266 | 0.609 |
| Base_Exag * Group | 1 | 1.017 | 0.319 |
| Error(Base_Exag) | 42 | | |
| Watch_UnW * Base_Exag | 1 | 1.550 | 0.220 |
| Watch_UnW * Base_Exag * Group | 1 | 0.025 | 0.874 |
| Error(Watch_UnW*Base_Exag) | 42 | | |

**Tests of Between-Subjects Effects**

| Source | df | F | Sig. |
|---|---|---|---|
| Intercept | 1 | 2967.865 | 0.000 |
| Group | 1 | 0.282 | 0.598 |
| Error | 42 | | |

Subsequent paired-sample t-tests showed that participants in both NT and ASC groups reached a greater height in the Watched trials with Exaggerated trajectory than in the Unwatched trials with Exaggerated trajectory [NT: $t(21) = 2.32$, $p = 0.03$; ASC: $t(21) = 2.86$, $p = 0.009$]. This suggests that participants in both the NT and ASC groups moved with a higher trajectory in the Exaggerated trials when being watched when compared with not being watched.

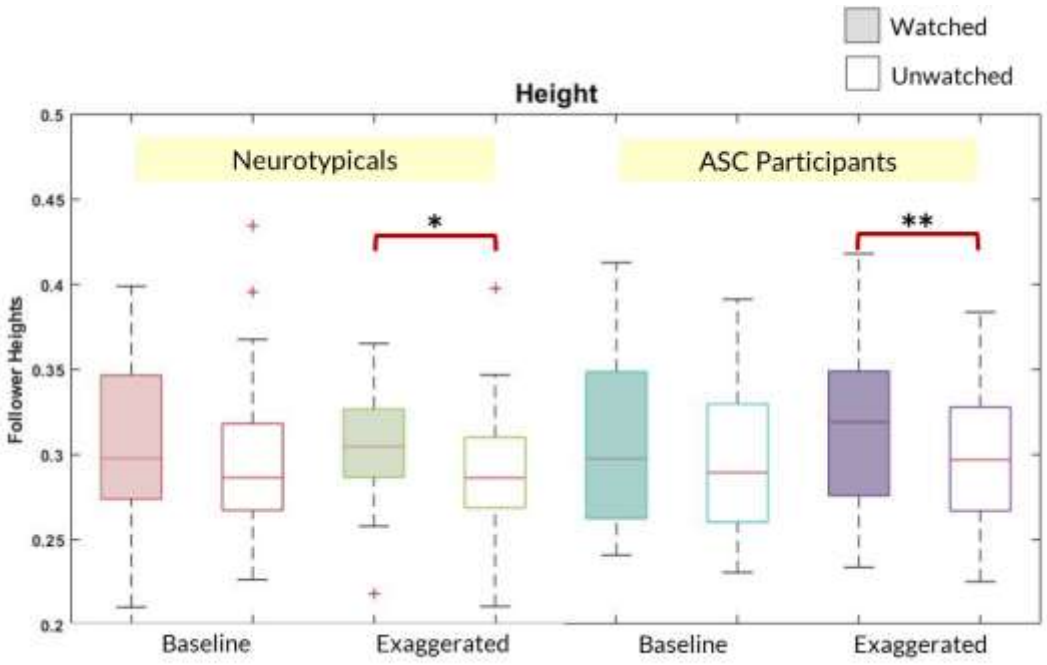


**Figure 5.6. Two-way mixed ANOVA of Follower Height.** This shows the height reached by the Followers in both NT and ASC groups in the Baseline and Exaggerated

trajectory conditions, when being watched and unwatched. Paired-sample t-tests showcase the sources of significant differences in heights. * $p < 0.05$; ** $p < 0.01$.

### *5.4.2. Other behavioural and physiological signals*

In addition to looking at the height reached by Followers we also looked at the Time taken per trial, and the three physiological signals recorded by the Equivital belt, namely heart rate, breathing rate, and galvanic skin response. In each case, we conducted a two-way mixed ANOVA to compare the effect of trajectory, and being Watched or Unwatched on the dependent variable, in both NT and ASC groups. These results are presented in detail below.

*Time taken by the Follower*

There was a significant main effect of being Watched on the time taken by the Follower [$F(1,42) = 17.72$, $p < 0.001$]. There was no main effect of Group, or of Trajectory. There was a significant interaction effect between being Watched or Unwatched and Group [$F(1,42) = 6.97$, $p = 0.012$]. A paired sample t-test showed that there was a difference in the Time taken for the ASC group to move in the Watched and Unwatched conditions [M-Watched = 22.02, SD = 7.06, M-Unwatched = 17.26, SD = 5.26; $t(21) = 4.22$, $p < 0.001$].

These results suggest that participants with ASC moved more slowly in the Watched condition. Although motor control difficulties do not form part of the diagnostic criteria for autism, motor control difficulties have been shown to be prevalent in those with ASC (Licari et al., 2020). We would expect in this situation that we should find a main effect of Group, rather than an interaction effect. However, if ASC participants tended to make high movements only in the Watched condition (when they copied Leaders), then this may explain why their movements were slower in the Watched condition. An alternate explanation could be that in the Watched condition participants had more conversations with the Leader and this lengthened trial times. We manually coded the videos of each trial to evaluate the amount of conversation between the Leader and the Follower during the Follower's turn. This was coded on a scale ranging from 1 for no conversation, to 5 where both the Leader and the Follower spoke sentences longer than two words to each other during the Follower's turn. A Pearson's product-moment correlation coefficient was calculated to assess the relationship between the time taken by the Follower and the amount of conversation. This showed that for both NT and ASC Followers there was a positive correlation between the time taken per trial and the overall conversation [$r(NT) = 0.396$, $p<0.001$; $r(ASC) = 0.604$, $p<0.001$]. This suggests that the

amount of conversation is a likely explanation for why trials took longer in the Watched condition when compared with the Unwatched condition.

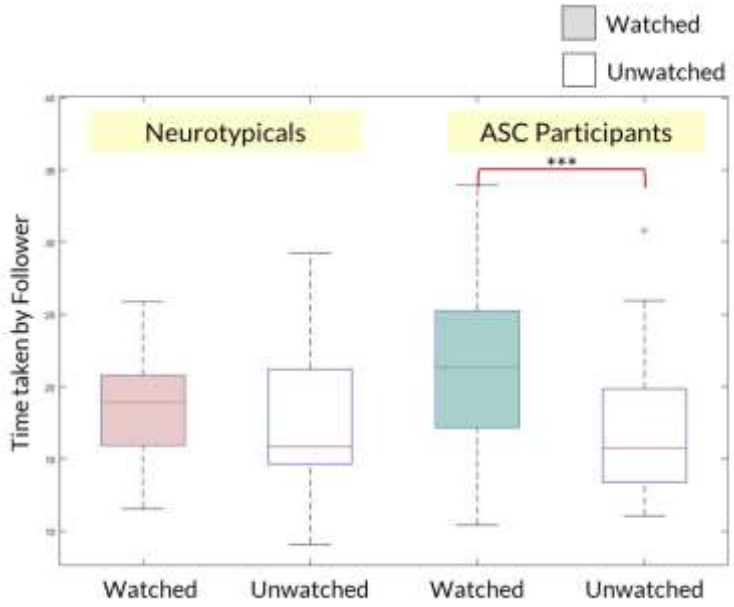


**Figure 5.7. Two-way mixed ANOVA of Time taken by Follower.** This shows the time taken by both NT and ASC groups in the Watched and Unwatched conditions.

*Heart rate*

We found no significant main or interaction effects of the independent variables on the heart rate of subjects.

*Breathing rate*

There was a significant main effect of being Watched on the Breathing rate of the Follower [$F(1,17) = 4.92$, $p = 0.041$]. There was no main effect of Group, or of Trajectory. There were no significant two-way interactions between the independent variables, but there was a significant three-way interaction effect between being Watched, Trajectory and Group [$F(1,17) = 6.28$, $p = 0.023$]. Detailed pair-wise comparisons show that both groups breathe faster in the Unwatched condition over the Watched condition, but this increase occurred in the Baseline trials for NT participants, and in the Exaggerated trials for the ASC participants. Speech patterns are once again a likely cause for the difference in breathing rates since participants are likely to breathe more rapidly when not speaking.

Again, a Pearson's product-moment correlation coefficient was calculated to assess the relationship between Follower breathing rate and the amount the Follower spoke during their turn (coded manually from the video). For the NT group we found no

significant correlation, but for the ASC group we found a negative correlation between breathing rate and the amount of Follower conversation [r(ASC) = -0.223, p = 0.003] which supports our view that breathing rate was lower when the Follower was speaking more.

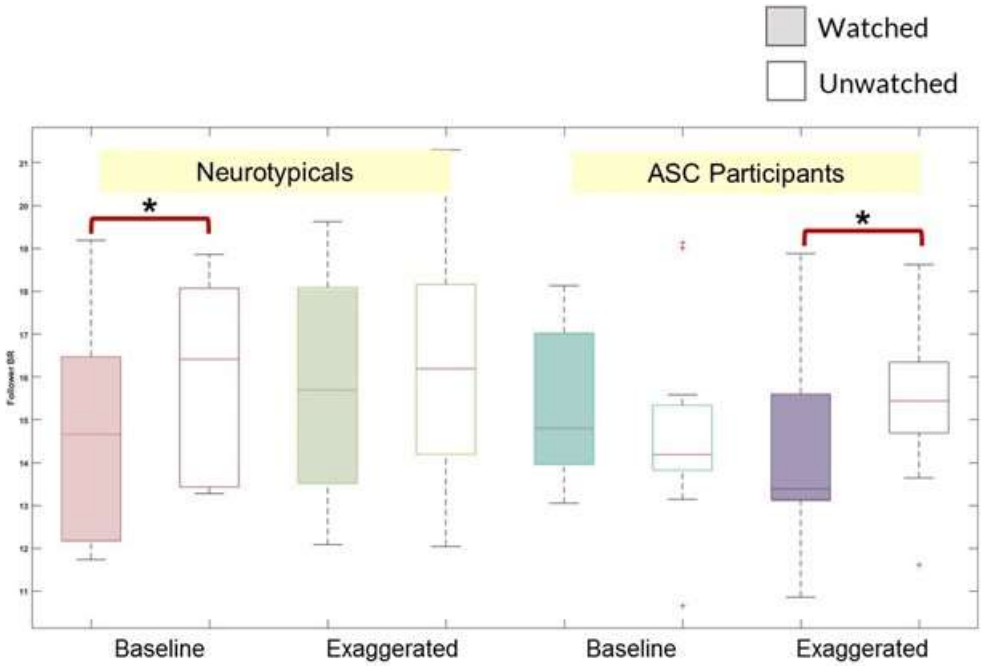


**Figure 5.8. Two-way mixed ANOVA of Follower Breathing rate.** This shows the breathing rate of both NT and ASC groups in the Baseline and Exaggerated conditions.

*Galvanic skin response*

There was no significant main effect of Group, Trajectory, or being Watched on the galvanic skin response of the Follower. There was a two-way interaction effect between being Watched and trajectory [F(1,27) = 5.62, p = 0.03]. There was no three-way interaction effect.

**Table *5.5.* Two-way Mixed ANOVA of Follower Galvanic Skin Response.**

**Tests of Within-Subjects Contrasts**

| Source | df | F | Sig. |
|---|---|---|---|
| Watch_UnW | 1 | 0.59 | 0.449 |
| Watch_UnW * Group | 1 | 3.33 | 0.079 |
| Error(Watch_UnW) | 27 | | |
| Base_Exag | 1 | 0.79 | 0.382 |
| Base_Exag * Group | 1 | 1.78 | 0.193 |
| Error(Base_Exag) | 27 | | |
| Watch_UnW * Base_Exag | 1 | 5.62 | 0.025 |
| Watch_UnW * Base_Exag * Group | 1 | 0.23 | 0.637 |
| Error(Watch_UnW*Base_Exag) | 27 | | |

**Tests of Between-Subjects Effects**

| Source | df | F | Sig. |
|---|---|---|---|
| Intercept | 1 | 103.53 | 0.000 |
| Group | 1 | 2.49 | 0.126 |
| Error | 27 | | |

Despite the two-way interaction, however, detailed pair-wise t-tests do not offer any support for a difference in galvanic skin response between conditions. A paired-sample t-test of the galvanic skin response in the watched and unwatched condition for baseline trials [$t(28) = 0.73$, $p = 0.47$] and for exaggerated trials [$t(28) = -0.172$, $p = 0.10$] showed no statistical significance. Perhaps with a larger sample size in future studies this would be worthy of exploring further. At present there is no support here for a difference in arousal between conditions.

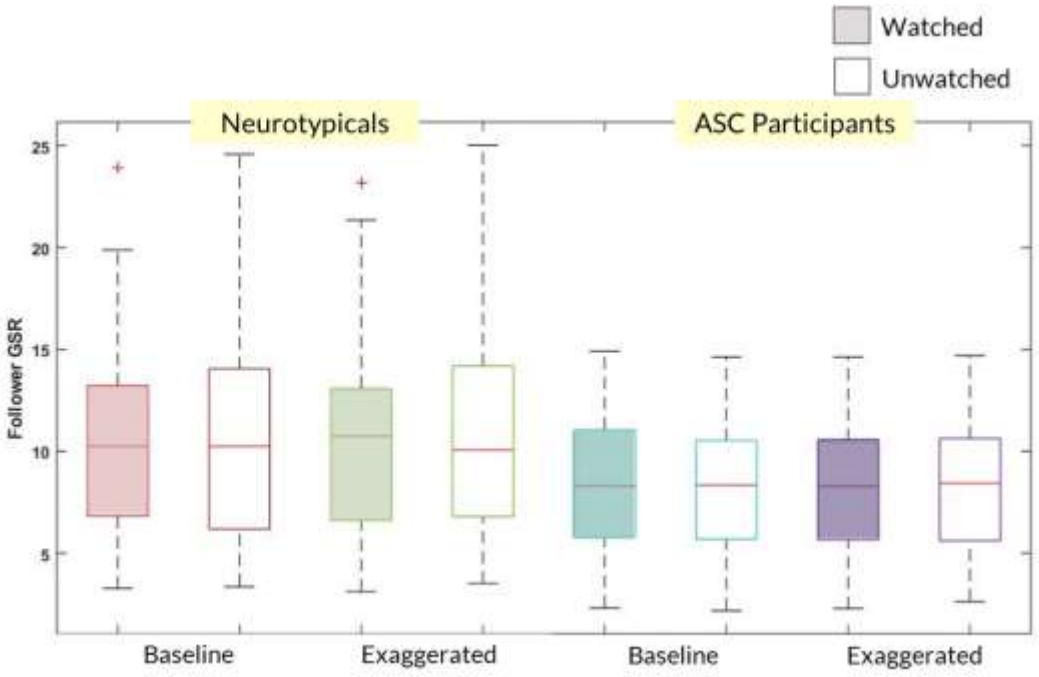


**Figure 5.9. Two-way mixed ANOVA of Follower Galvanic skin response.** This shows the galvanic skin response of both NT and ASC groups in the Baseline and Exaggerated conditions.

### *5.4.3. Neural correlates*

We now turn to the various neural correlates analysed in this study, namely the five contrasts of interest highlighted in Table 5.3. These are also summarised below in Table 5.6.

**Table 5.6. Summary of Neural activations for key contrasts of interest.** Blue refers to channels that were statistically significant for the NT group, orange is for channels that were statistically significant for the ASC group. Channels that showed group similarity (i.e., channels that were significant for both groups combined) are shown with a green background. Significant channels which survive the correction for multiple comparisons by virtue of having two or more adjacent channels with an uncorrected $p<0.05$ are highlighted in bold. The most likely anatomical region is included in this table; for full details please refer to Table 5.2.

| Channel number | Anatomical region | C1: Watching Leader perform irrational actions | | C2: Performing actions when watched vs unwatched | | C3: Responding to an irrational action | | C4: Explicit demand for irrational action | | C5: Being imitated vs not being imitated | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | T | p(uncorr) | T | p(uncorr) | T | p(uncorr) | T | p(uncorr) | T | p(uncorr) |
| 2 | Right Inferior Parietal Lobule | | | **-2.29** | **0.03** | -2.47 | 0.04 | | | | |
| 3 | Right Inferior Parietal Lobule | | | **-2.69** | **0.01** | | | | | | |
| 5 | Right Inferior Parietal Lobule | | | | | | | | | 2.78 | 0.02 |
| 7 | Right Inferior Parietal Lobule | | | -3.93 | 0.00 | | | | | | |
| 16 | Right Temporal Lobe | | | | | | | 2.19 | 0.05 | | |
| 18 | Right Temporal Lobe | | | | | | | | | -4.55 | 0.00 |
| 21 | Right Temporal Lobe | | | | | | | -2.06 | 0.05 | | |
| 24 | Left Parietal Lobe | | | | | **-4.07** | **0.00** | | | | |
| | | | | | | -4.87 | 0.00 | | | | |
| 25 | Left Inferior Parietal Lobule | | | | | **-2.17** | **0.04** | | | | |
| 26 | Left Inferior Parietal Lobule | | | -2.64 | 0.02 | | | | | | |
| 28 | Left Parietal Lobe | 3.81 | 0.01 | | | | | | | | |
| 31 | Left Inferior Parietal Lobule | | | -2.95 | 0.02 | | | | | | |
| 37 | Left Temporal Lobe | | | **2.93** | **0.01** | | | | | | |
| 39 | Left Inferior Parietal Lobule | -2.69 | 0.02 | | | | | | | | |
| 41 | Left Temporal Lobe | | | **2.71** | **0.04** | | | | | | |
| 44 | Left Occipital Lobe | | | | | -2.73 | 0.02 | | | | |

Given the large number of contrasts and the exploratory nature of this study, we will focus on discussing only the results that survive the correction for multiple comparisons in the discussion section. The other results will be briefly discussed in the results section alongside the reporting of the results for that contrast. The significant results that will be reviewed in detail in the discussion section include C2 and C3 in the table above.

*C1: Watching Leader perform irrational actions*

Here the neural activation when the Follower was watching the Leader make Exaggerated movements was contrasted with the activation when the Follower was watching the Leader make Baseline movements. We found suppression in channel 39 (IPL/V3) for exaggerated actions vs base actions in the NT group, and activation in channel 28 (primary somatosensory cortex) for exaggerated actions vs baseline actions in the ASC group. We also found group differences between the NT and ASC groups in channel 39. This suggests that the irrational action (an exaggerated trajectory) is processed differently in NT and ASC groups, perhaps with the ASC group more focused on integrating sensory input in preparation for making a motor response (Borich et al., 2015).

Previous studies among neurotypicals have shown activation in the TPJ and the IPL when watching irrational actions (Brass et al., 2007, 2009; Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011; Saxe et al., 2004), although many of these studies have identified the activation in the right hemisphere rather than the left as we found. When it comes to those with ASC the question of whether they are equally able to distinguish rational from irrational actions remains unanswered. While some studies have shown that children with ASC perform as well as TD children in distinguishing rational and irrational actions (Hamilton et al., 2007), others have highlighted impairment (D'Entremont & Yazbek, 2007). Marsh and Hamilton (2011) found that viewing irrational actions led to different patterns of activation among NTs and those with ASC. In our previous study (Chapter 4) we found significant activation in the right IPL amongst Followers when viewing the Leader making irrational movements. While we find a similar pattern of activation in this study in the right IPL amongst neurotypicals, none of these channels reach significance.

Given the different neural activation patterns seen in the NT and ASC groups, it could suggest that the ASC participants may not be identifying the exaggerated trajectory as irrational. However, if this were true and they were merely copying the higher trajectories throughout then we should not see the difference in the heights performed by ASC participants between the watched and unwatched conditions. Instead we found ASC participants also made exaggerated trajectories in response to exaggerated demonstrations to a greater extent in the watched condition rather than the unwatched condition. This suggests that both NT and ASC participants identified the exaggerated trajectories as irrational, and responded by copying them to a greater extent in the watched condition rather than the unwatched condition.

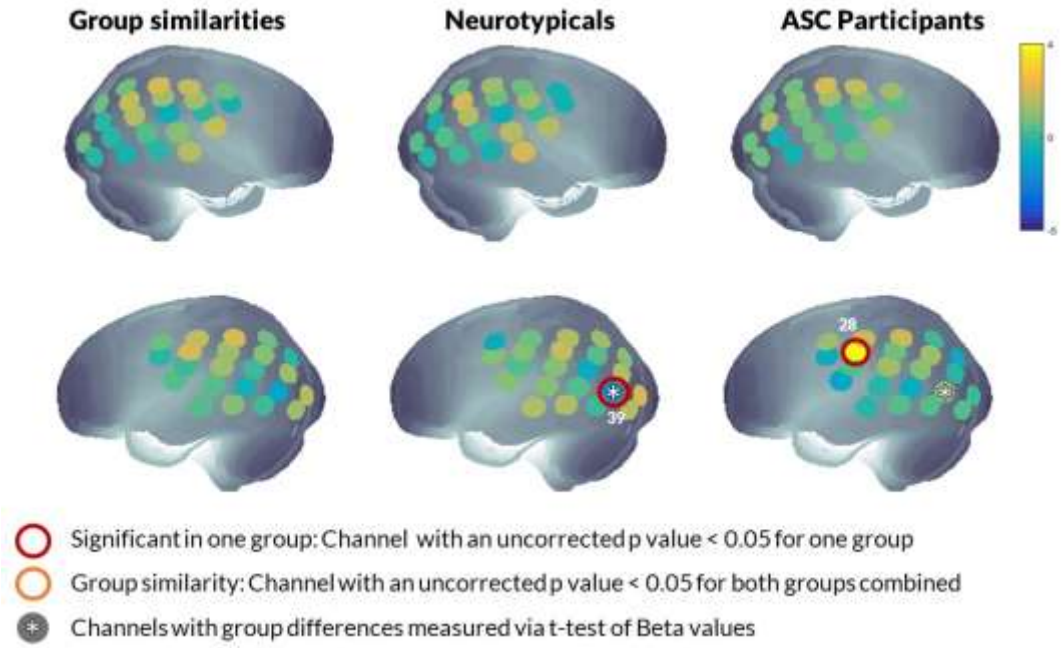


**Figure 5.10. Channel-wise activation in Follower in Main phase when watching Leader perform exaggerated irrational actions vs baseline rational actions.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ in any group are highlighted with a red circle. Channels with an overall uncorrected p-value of $p<0.05$ in both groups combines are highlighted with an orange circle. Channels where there is a statistically significant group difference (uncorrected p-value $p<0.05$) between the NT and ASC groups are highlighted with an asterisk.

*C2: Being watched*

The Follower's neural activation when being watched by the Leader was contrasted with the activation when the Leader was not watching them on their turn. Both NT and ASC participants (when analysed together) showed suppression in and around the bilateral inferior parietal lobule (IPL) namely in channels 2, 3, and 26. In ASC participants this suppression extended deeper into the right IPL (channel 7), while NT participants showed an extension of this suppression in the left hemisphere (channel 31) extending into V3, adjoining the left IPL. This is similar to the suppression in right IPL and right TPJ seen when being watched as opposed to not being watched in our previous study (see Section 4.4.5). Another key result in this contrast is the strong activation seen in ASC participants around the left STS, namely channels 37 and 41. Channel 37 also showed group differences, suggesting that this activation is specific to how ASC participants reacted to being watched. Statistically significant activations in two adjacent channels are considered corrected for multiple comparisons (Pinti, Tachtsidis, et al., 2020; Southgate et al., 2014). As a robust result, this is reviewed in detail in the discussion section that follows.

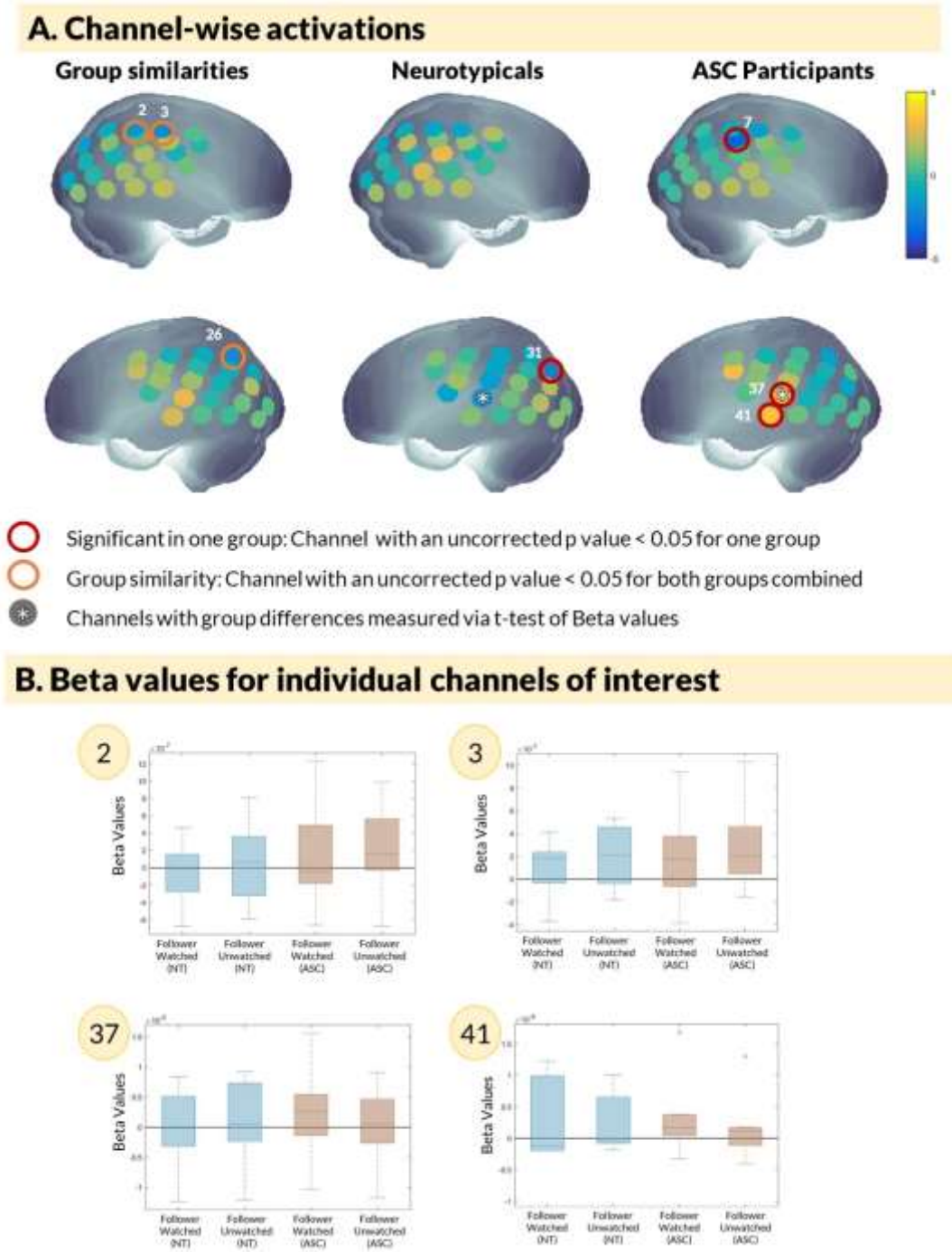


**Figure 5.11. Channel-wise activation in Follower in Main phase when being Watched vs when not being Watched.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ in any group are highlighted with a red circle. Channels with an overall uncorrected p-value of $p<0.05$ in both groups combines are highlighted with an orange circle. Channels where there is a statistically significant group difference (uncorrected p-value $p<0.05$) between the NT and ASC groups are highlighted with an asterisk. Channels 2,3 and channels 37,41 are pairs of two adjacent channels that are significant; these are considered to survive the correction of multiple comparisons (Southgate et al., 2014). **B. Beta values for individual channels of interest.** The Beta values are shown for the two pairs of channels in contrast 2 (Watched > Unwatched) where adjacent channels have p-values of $<0.05$.

*C3: Responding to an irrational action*

This contrast evaluates the Follower's response during the Follower's turn following an exaggerated demonstration by the Leader, versus following a baseline demonstration by the Follower. Here we found suppression in the left parietal lobule extending into the left IPL for both groups (channels 24 and 25) and suppression in the bilateral IPL for NT participants (channel 2). We also found group differences in the left V3 extending into the left IPL. Adjacent channels here show both group similarities and group differences, and these results can be considered to be corrected for multiple comparisons. These results are discussed in detail in the discussion section that follows.

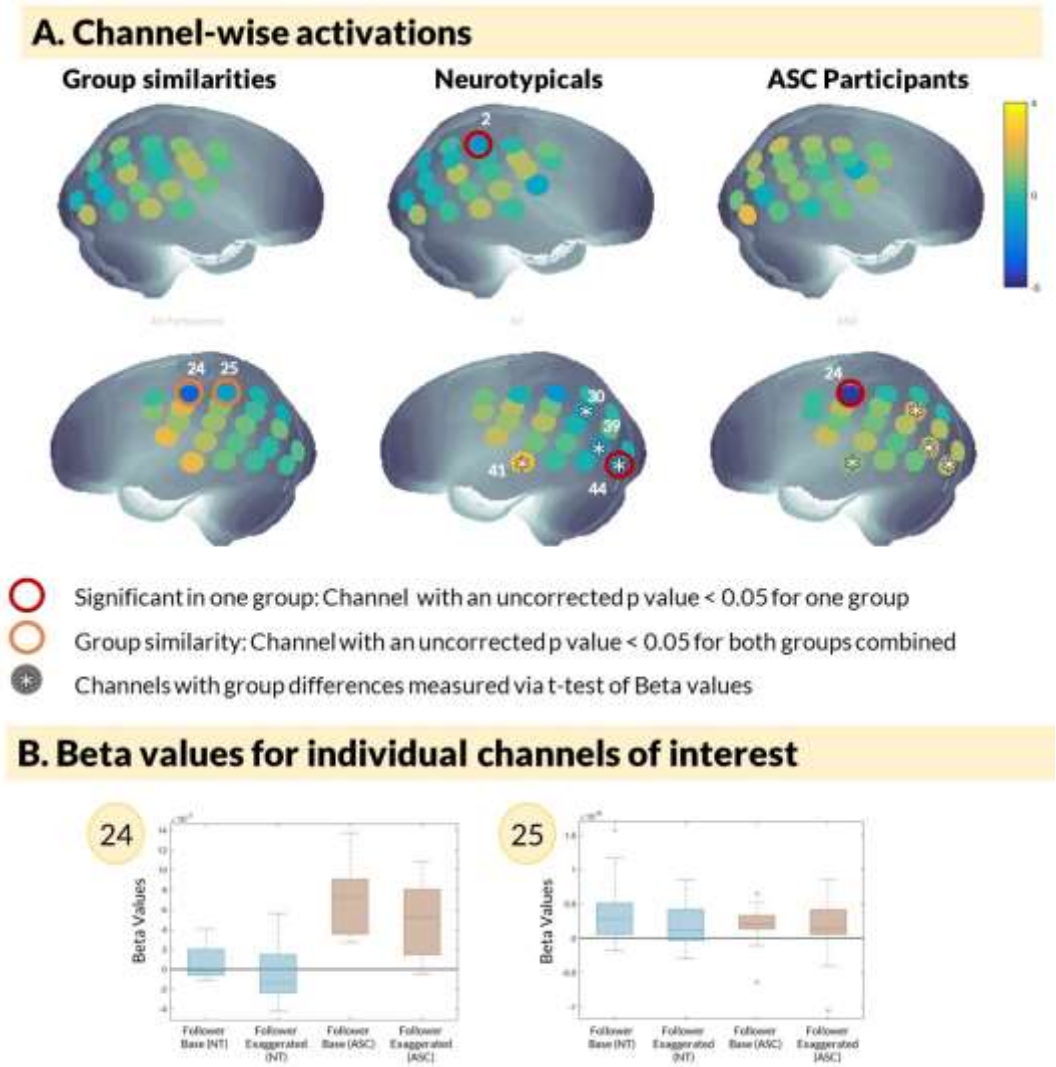


**Figure 5.12. A. Channel-wise activation in Follower in Main phase during Follower turn after an exaggerated demo by the Leader, when compared with activation after a baseline demo by the Leader.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ in any group are highlighted with a red circle. Channels with an overall uncorrected p-value of $p<0.05$ in both groups combines are highlighted with an orange circle. Channels where there is a statistically significant group difference (uncorrected p-value $p<0.05$) between the NT and ASC groups are highlighted with an asterisk.

Channels 24 and 25 are two adjacent channels that are significant; these are considered to survive the correction of multiple comparisons (Southgate et al., 2014). **B. Beta values for individual channels of interest.** The Beta values are shown for the two pairs of channels in contrast 2 (Exaggerated>Base) where adjacent channels have p-values of <0.05.

## *C4: Explicit demand for an irrational action*

Moving to the Switch trials, we look at the neural activation when the Follower is explicitly instructed to make an exaggerated movement vs when they are asked to make a baseline movement. Here we found suppression in the right temporal lobe (channel 21) in both NT and ASC participants (when analysed together). In ASC participants this was accompanied by a strong activation in the adjoining right STS (channel 16). We also found group differences in the left V3 region. Behaviourally both groups were able to meet the explicit demands for irrational action, but this result suggests that the demand is processed differently in ASC and NT groups.

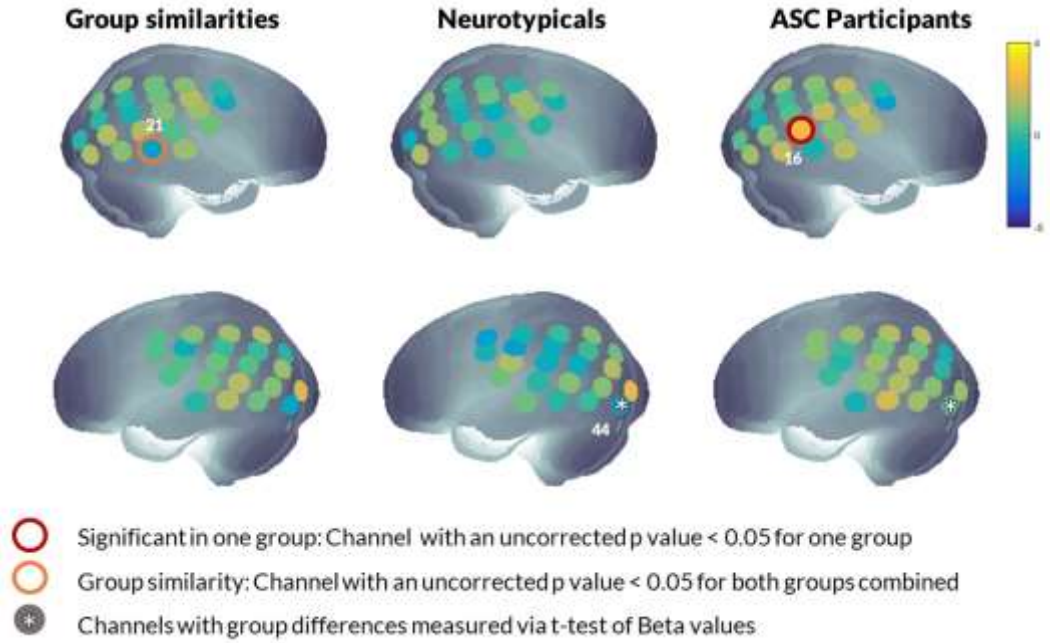


**Figure 5.13. Channel-wise activation in Follower in the Switch trials when asked to make an exaggerated trajectory versus when asked to make a baseline trajectory.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ in any group are highlighted with a red circle. Channels with an overall uncorrected p-value of $p<0.05$ in both groups combines are highlighted with an orange circle. Channels where there is a statistically significant group difference (uncorrected p-value $p<0.05$) between the NT and ASC groups are highlighted with an asterisk.

*C5: Being copied*

Finally, we turn to how ASC and NT participants processed being copied. We looked at neural activation when the Follower was copied by the Leader vs when the Leader did not copy the Follower's trajectory. Here we found activation in the NT group in V3 bordering the right IPL. In the ASC group we found strong suppression in the right TPJ.

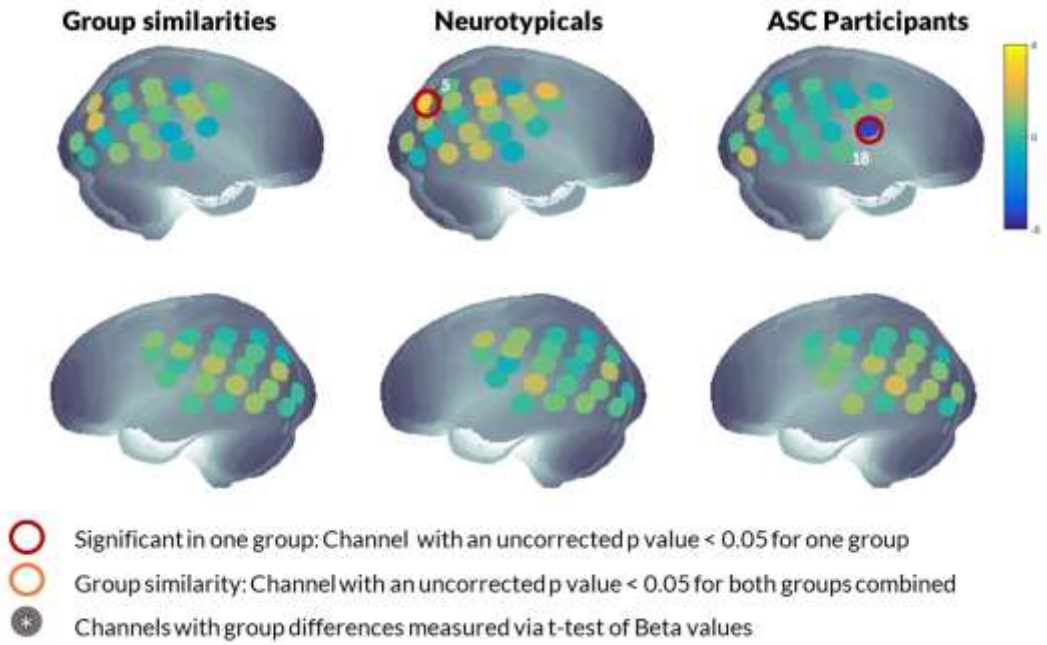


**Figure 5.14. Channel-wise activation in Follower in the Switch trials when copied by the Leader versus when not copied by the Leader.** The T-values for the specified contrast are plotted at the channel locations on the canonical brain. Channels with an uncorrected p-value of $p<0.05$ in any group are highlighted with a red circle. Channels with an overall uncorrected p-value of $p<0.05$ in both groups combines are highlighted with an orange circle. Channels where there is a statistically significant group difference (uncorrected p-value $p<0.05$) between the NT and ASC groups are highlighted with an asterisk.

On a behavioural level, studies have shown that being copied is rewarding: it builds rapport and how much we like others (Chartrand & Bargh, 1999; Lakin & Chartrand, 2003; Stel & Vonk, 2010), and being copied has also been shown to increase our propensity to help others (Müller et al., 2012) or increase the tips that waitresses receive (van Baaren et al., 2003). Our study did not have any behavioural correlates to being copied, and the neural activations when copied do not show any group differences between NT and ASC participants.

## 5.5. Discussion

In this study we sought to extend the social-signalling hypothesis of imitation by comparing neurotypicals with those with ASC, given the differences in perceptions of action rationality, imitative behaviour and social interaction seen in those with ASC when compared with neurotypicals. Using a variation of our dyadic block-moving task (detailed in Section 2.5.1), we evaluated the imitative fidelity of participants when their interaction partner produced irrationally high trajectories both in situations where the partner was watching the participant make their movement and when the partner had their eyes closed during the participant's turn. In addition to replicating our behavioural results with the neurotypical group we found that those with ASC also imitated their interaction partners and did so with greater fidelity when they knew they were being watched. Further, we found a robust effect of being watched in the parietal cortex in both groups, and differences in neural activation in other scenarios between the two groups. In this discussion we will first review the behavioural results, followed by the neural correlates for key results centring on the neural correlates of performing actions when watched versus when not being watched (C2 in the table below), and in responding to irrational actions (C3). Finally, we will discuss some limitations to this study and directions for future work.

### *5.5.1. Imitative fidelity when watched by an interaction partner*

Replicating our previous work (Krishnan-Barman & Hamilton, 2019 and Chapter 4) we found that neurotypical (NT) adults imitated irrational movements with greater fidelity in trials where they knew their interaction partner was watching them, than in trials where they knew they were not being watched. This result is in line with other recent work that has shown that whether an interaction partner is watching us or not has an impact on the fidelity with which we imitate (DiYanni et al., 2011; Marsh et al., 2019; Nielsen & Blank, 2011; Sommer et al., 2020). We also found that this effect is preserved in autistic participants who also copied their partners with greater fidelity when being watched. This evidence runs counter to our initial hypothesis (Section 5.2.2) where we suggested that those with autism were less likely to deploy imitation as a social signal. Based on our study design it is not possible to parse whether autistic participants engaged in this social imitation consciously or unconsciously, since we could not explicitly question them on it until the end of the experiment, when they had already been exposed to the trajectory manipulations as part of the Switch trials.

Other studies of audience effects have suggested that differences in behaviour may result from differences in arousal from direct gaze (Senju & Johnson, 2009), differences in anxiety (Heyes, 2017) or simply as a result of social facilitation (Zajonc, 1965). Our study was designed to minimise the differences between the watched and unwatched condition: participants were side-by-side throughout rather than watching each other ensuring there were no differences in eye-gaze or in social facilitation between conditions. Further, the null result for galvanic skin response between the watched and unwatched condition (Section 5.4.2) supports the view that there was no difference in arousal between the two conditions. This buttresses our view that the behavioural differences seen between the watched and the unwatched condition arise from the cognitive effect of being watched, rather than explanations rooted in arousal or social facilitation.

### *5.5.2. Neural correlates of responding to irrational actions*

In this study Followers watched the Leader (a trained confederate) perform both rational and irrational trajectories before responding with their own move. This contrast specifically evaluates the Follower's response during the Follower's turn after an exaggerated demonstration by the Leader, versus following a baseline demonstration by the Leader. In both autistic and neurotypical participants we found suppression in the left IPL in adjacent channels. This can be considered a robust result since two adjacent signals show statistically significant effects, allowing us to consider them corrected for multiple comparisons (Southgate et al., 2014). In a similar vein, a study by Marsh and Hamilton (2011) found that both neurotypical and autistic adults exhibited similar activation patterns in the left IPL when viewing rational and irrational goal-directed actions.

We also found that this deactivation extends bilaterally into the right IPL for neurotypicals. This is similar to the deactivation in the right IPL seen in the study in Chapter 4 when performing irrational movements. It is important to note that the contrast in Chapter 4 was slightly different, measuring brain activity based on the height performed by the Follower as a parametric regressor. In that study, it was found that activity in the right SPL was parametrically modulated by the height performed by the Follower, with greater deactivation the higher (or the more irrationally) the Follower moved. It is useful to consider both the above results in the current chapter in conjunction with behavioural results. We know that there was no main effect of trajectory on the Follower's height suggesting that participants were not merely primed to respond with a

high trajectory right after viewing one. Thus, the neural results above are not the result of simply moving higher in response to viewing a high trajectory.

Finally, we also found group differences between the autistic and neurotypical participants in the left occipitotemporal regions, with neurotypical participants showing suppression in this area, while autistic participants do not. This signposts interesting similarities and differences in how neurotypical and autistic participants processed their response to an irrational action, despite the fact that their actual behavioural responses were not significantly different.

### *5.5.3. Brain responses to being watched*

In behavioural terms we saw that both neurotypicals and autistic participants moved higher in the watched condition following an exaggerated trial than in the unwatched condition following exaggerated trials. With regards to the neural correlates we found that ASC participants showed strong activation in the left STS when being watched when compared to the unwatched condition. We also found that both neurotypical and autistic participants showed suppression around the bilateral IPL, extending slightly deeper into the right for autistic participants. For the neurotypical participants we found instead that this suppression extended slightly deeper into the left IPL The headline result here is similar to the result found in Chapter 4 where brain activity was compared between the watched and unwatched condition and we found a robust, significant deactivation around the right TPJ or right IPL.

In Chapter 4 we reviewed several potential explanations for this deactivation in activity in the parietal cortex in the Watched condition (see Section 4.5.3). Broadly the deactivation in the parietal cortex could be linked to Followers making different movements in the Watched condition versus the Unwatched condition. Specifically, the behavioural results suggest that Followers make relatively straight-line movements in the Unwatched condition and in the Watched-rational condition, moving higher only in the Watched-irrational condition. On a similar vein, Followers also tend to copy the Leader directly in the Watched condition throughout and in the Unwatched-rational condition. Together, this would suggest that Followers only move higher in the Watched-irrational condition, or make their own decision on how to move in the Unwatched-irrational condition. Our study was not designed to capture the interaction effects in our planned comparisons. In future studies, it would be useful to plan these comparisons in advance.

Another strand of explanations could be differences in the cognitive demands arising from being watched or not. As we note in Chapter 4 (see Section 4.5.3), being watched could be more cognitively demanding taking resources away from brain regions that are typically involved in controlling imitation. Or alternatively, not being watched may be the more unusual situation; keeping in mind that our interaction partner cannot view us may necessitate greater mentalising, given the atypical-ness of the situation. In both cases it would be informative to undertake whole-brain scans to analyse neural activation in other regions while being watched and develop a better picture of what else is happening while participants undertake this task.

### *5.5.4. Limitations and future directions*

These results have some limitations relating to the paradigm used. While allowing for a social interaction without explicitly calling attention to the trajectory, our design does not allow us to distinguish between the effect of making an irrational movement and moving higher. It would be useful to attempt to replicate these results by manipulating rationality through the presence or absence of an obstacle, while keeping the movement kinematics the same between the rational and irrational condition. Our design also did not allow us to differentiate between conscious and unconscious copying or allow us to determine at what point Followers became aware of the Leader's trajectory changes during the experiment.

When it comes to ASC participants in particular, we do not know the extent to which motor control difficulties may have influenced the results. Both ASC and neurotypical participants showed similar behavioural results when it came to imitative fidelity, however ASC participants moved slower than NT participants in the Watched condition. We have reviewed potential explanations for this above (see Section 5.4.2), but in future experiments separating out the effect of making an irrational movement from having to move higher would be useful to parse these effects. In future studies it would also be useful to include a non-social motor-based task such as just moving blocks based on computer instructions to develop a baseline of motor performance for all participants.

Another fruitful avenue for future exploration would be to incorporate recording of eye gaze into this study. Previous studies have shown that NT and ASC participants orient their gaze differently in goal-directed action sequences. NT participants have been shown to engage in predictive gaze, moving their gaze proactively in line with an expected trajectory (Flanagan & Johansson, 2003). When it comes to those with ASC the evidence is mixed, with some studies showing that they engage in predictive gaze (Falck-

Ytter, 2010) while others suggest that they do not (Senju et al., 2009). Marsh and colleagues (2015) showed that ASC participants tended to pay less attention to features such as the action goal and the hand performing the action, but if we look only at trials where they do pay attention, they are able to engage in predictive gaze on par with NTs. Evaluating gaze patterns in both groups while engaging in this task may provide more insight into this.

## 5.6. Conclusions

Overall, this study supports the social-signalling hypothesis of imitation by showing that both NT and ASC participants can identify rational actions, and the social availability of an interaction partner; both groups also respond in similar ways by imitating irrational actions with greater fidelity when they know their interaction partner is watching them. The differing patterns in neural activation between NT and ASC groups suggest that although their behaviours are similar, the brain mechanisms underpinning them may be different.

# Chapter 6. Discussion

This concluding chapter summarises the experimental work carried out in this thesis and outlines how it supports the social-signalling hypothesis of imitation. We also discuss future directions that could advance our understanding of imitation as a social signal.

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## 6.1. Imitation as a social signal

Over a series of behavioural and neuroimaging studies this thesis has sought to test the social-signalling hypothesis of imitation. This affiliative account of imitation theorises that we imitate in order to achieve certain social goals, such as increasing rapport or communicating mutuality (Uzgiris, 1981). Under this, imitation is thought to function as a social glue that increases our social advantage (Lakin et al., 2003; Wang & Hamilton, 2012). The STORM model (Wang & Hamilton, 2012) in particular reviews a wide range of existing neurocognitive evidence to suggest that imitation is socially modulated and incorporates a "Machiavellian goal of increasing one's social standing". The STORM model thus makes a highly specific claim that we imitate in order to affiliate (in the same vein as the argument advanced in Farmer et al., 2018; Over & Carpenter, 2013). This thesis has sought to explicitly delineate the mechanism by which this may function, and test the individual components of this mechanism, both in terms of behaviour and to understand the brain mechanisms underpinning these behaviours.

Specifically, the social-signalling hypothesis says that imitation is a social signal selectively sent between interaction partners depending on the social availability of the recipient. Unlike a cue which only benefits one party in an interaction, a signal is conceptualised as benefiting both the sender and the recipient (Stegmann, 2013). In an interaction between the copier (sender) and the person being copied (recipient), we know from the existing evidence that not being imitated can increase cortisol levels in the signal recipient (Kouzakova et al., 2010). A wide range of studies have also shown that copying benefits the sender as well; the recipient of the signal is more likely to view the sender as more knowledgeable, tip them more in the case of waitstaff, be more inclined to buy the sender's product, and be more likely to engage in helpful and charitable behaviour when copied (Chartrand & Lakin, 2013; Fischer-Lokou et al., 2011; Stel et al., 2008; Tanner et al., 2008; van Baaren et al., 2003, 2004; van Swol, 2003).

In this thesis we systematically tested this hypothesis using our dyadic block-moving paradigm (see Section 2.5.1), which involves two participants—a Leader and a Follower. The Leader first demonstrates moving blocks from point A to point B using either a rational (straight-line) trajectory or an irrational (high and curved) trajectory. The Follower is subsequently told to move the blocks in the same order as the Leader, while no explicit mention is made of the trajectories; however, the Follower is told that their score on each trial depends on moving quickly, incentivising them to move as fast as possible. This task builds on a range of a range of paradigms used in the study of mimicry and on evaluating rationality (Forbes & Hamilton, 2017; Gergely et al., 1995; Krishnan-

Barman et al., 2017; Marsh et al., 2015; Oliver et al., 2017; Wild et al., 2012), and was implemented in an augmented-reality environment to maintain experimental consistency and reduce setup times. In addition to the height of the demonstrated trajectory, the other independent variable manipulated was the social availability of the Leader when the Follower was making their move. In one-half of the trials, the Leader was explicitly instructed (via voice commands) to close their eyes during the Follower's turn, while in the other trials the Leader could watch the Follower make their move.

In manipulating the social availability, we were keen to engender a cognitive effect of being watched, rather than merely increased arousal or anxiety from the presence of another person, or attentional explanations. It has been argued that several results showcasing the modulation of imitation by social context may occur instead owing to differences in attention or anxiety and be unrelated to social-signalling (Heyes, 2017). We sought to address this concern in our paradigm design by ensuring the watched and unwatched conditions were as closely matched as possible. First, the interaction partners stood side-by-side precluding any effects of direct gaze. Second, both partners remained in situ throughout the experiment avoiding any changes in social facilitation or attention between trials. Third, the actual demonstration by the Leader was identical in the watched and unwatched conditions, ruling out any differences in attention between the two conditions. Finally, if anxiety in the watched condition was driving behaviour, it should cause the Follower to respond (when watched) by moving faster and in a more straight-line trajectory, which is counter to the social-signalling hypothesis that would suggest that Followers would imitate the high, curved trajectories of Leaders with greater fidelity in the watched conditions.

We now turn to the specific testable hypotheses arising from our theoretical framework, and how these were tested in each chapter. According to the social-signalling hypothesis of imitation in a dyadic interaction between Alice and Betty, where Alice occasionally demonstrates irrationally high trajectories, and where Alice is only socially available (i.e., watching Betty) in one half of the trials, we would expect the following to occur:

(a) Betty should recognise (either consciously or unconsciously) that Alice is making irrational movements in some trials
(b) Betty should recognise on a trial-by-trial basis whether Alice is socially available to receive a potential imitative signal
(c) Betty should copy Alice's actions including the irrational trajectories in some trials

(d) The degree to which Betty copies Alice's actions should be modulated by whether Alice was socially available in that trial

### *6.1.1. Summary of experimental chapters*

Below we first briefly summarise the experimental chapters before considering the evidence from the experiments in this thesis for each of the hypotheses above.

***Chapter 3*** described a pilot behavioural study and a pre-registered replication that examined the fidelity with which Followers copied irrational movements made by Leaders. I found that Followers imitated the Leaders, and critically, they did so with greater fidelity in trials where they knew the Leader was watching them. This supports (c) and (d) suggesting that imitation does function as a social signal.

***Chapter 4*** examined the neural correlates of imitation in a social context by simultaneously capturing neural activations in the right hemisphere, centred on the right TPJ, for both Leaders and Followers using the same dyadic block-moving task as described earlier. I found again that Follower imitated the Leader with greater fidelity in the watched trials when compared with the unwatched trials. I also found that when watching irrational actions Leaders showed greater activation in the right TPJ, while Followers showed greater activation in the right IPL when watching Leaders make irrational actions. When it came to being watched, Followers showed strong, robust deactivation in the right TPJ and the right IPL. These provide neural correlates to inform (a) and (b) showing that both action rationality and the cognitive effect of being watched are robustly encoded in the TPJ and IPL.

**Chapter *5*** sought to extend this hypothesis by additionally testing the behaviour and neural correlates of autistic individuals using a similar dyadic block-moving task. Here we found that behaviourally both neurotypicals and those with autism were matched, with both copying the Leader with greater fidelity in trials where they knew the Leader was watching them. When it came to neural correlates the picture was more mixed. When responding to irrational actions both neurotypicals and those with autism showed suppression of activation in the left IPL, while in neurotypicals this extended bilaterally into the right IPL as well. However, only autistic individuals engaged left occipitotemporal regions after viewing irrational actions. In line with the results seen in Chapter 4, I found that being watched was accompanied by a robust deactivation in the right parietal cortex across both neurotypicals and autistic participants. However, only autistic participants engaged left STS when being watched.

### *6.1.2. Imitation fidelity in watched and unwatched trials*

The key behavioural component of our theory is whether the extent to which people copy irrational actions is modulated by the social availability of their interaction partner (i.e., whether the interaction partner can watch them make their movement or not). Early evidence from animal studies have supported the view that merely being the presence of a conspecific can change our behaviour owing to changes in arousal regardless of whether the conspecific is watching them or not (Zajonc, 1965; Zajonc & Sales, 1966). However, we are interested in a more specific phenomenon of the audience effect, where behaviour is influenced by being watched (Triplett, 1898). On this there has been growing evidence that being in the presence of a demonstrator influences our imitative behaviour, with a number of studies showing that children tended to imitate a demonstrator who was watching them to a greater extent (DiYanni et al., 2011; Marsh, Ropar, et al., 2014; Nielsen & Blank, 2011). Evidence from other fields of study has also suggested that being watched modulates behaviour: Cañigueral & Hamilton (2019b, 2019a) have shown that participants tend to gaze less at a live interaction partner (when compared with a video), and behave in a more prosocial manner when they believe they are being watched. Other studies have also shown that people's behaviour changes even when the feeling of being watched is manipulated at an abstract level, such as when people are told their cognitive capacity is being evaluated (Bengtsson et al., 2009), or when people make disclosures about themselves in the presence of others (Izuma et al., 2010).

In this study across all four experiments, we found that Followers moved higher or copied Leaders with greater fidelity when they knew the Leader was socially available to receive a signal from the Follower. This occurred despite participants being instructed to move as quickly as possible, which would incentivise the use of a straight-line, fast, trajectory. Interestingly, and in contrast to what we initially expected, the same behavioural effect was seen in autistic participants in the experiment in Chapter 5. Our results do not allow us to distinguish whether autistic participants made a conscious choice to copy Leaders to a greater extent in the watched trials (in an effort to behave in what may be considered a socially appropriate way) or whether this was unconscious. Thus, we cannot parse whether they were engaging in some kind of cognitive compensation, which is effortful, or whether their behaviour on this simple task matched neurotypicals owing to some other unconscious mechanism.

All these results form the crux of support for our social-signalling hypothesis of imitation, showing that imitation is modulated by whether we are being watched by an interaction partner or not. As we highlighted above, the experiments were carefully

designed to ensure there were no differences in anxiety or arousal between watched and unwatched conditions to ensure we were measuring changes that arose from the cognitive effect of being watched. This is further supported by the finding in the experiment in Chapter 5 that found no significant difference in galvanic skin response between watched and unwatched trials further supporting the view that our paradigm engendered a genuine cognitive feeling of being watched, rather than differences arising from differences in anxiety.

### *6.1.3. Recognising social availability*

We now turn to the brain mechanisms that underpin our ability to understand the social availability of an interaction partner. The key question here is whether there is a brain region that encodes whether we are being watched. Understandably it is tricky to engender a feeling of being watched when inside an fMRI scanner. However, innovative attempts involving mirror-arrays, scanning people simultaneously while exchanging eye signals, or using wearable imaging technologies such as fNIRS have shown the engagement of mentalising networks when we are being watched (Cavallo et al., 2015; Dravida et al., 2020; Koike et al., 2019).

Across Chapters 4 and 5, it was found that being watched led to strong, robust deactivation in the right parietal cortex across both neurotypicals and autistic individuals. This is a robust result with multiple adjacent channels showing statistical significance, which can be considered a result that is corrected for multiple comparisons (Pinti, Tachtsidis, et al., 2020; Southgate et al., 2014). However, this result stands in contrast to the activations in the mentalising networks reported in other studies above. In the experimental chapters we reviewed several potential explanations for this intriguing result. First, it could be that Followers moved higher in the Watched condition. We might expect greater activation in the parietal cortex rather than deactivation, but studying what is happening elsewhere in the brain through a whole brain scan would help address this question. Second, in the rational condition (Watched and Unwatched) and in the Watched-irrational condition Followers may be just copying what they see the Leader doing. It may only be in the Unwatched-irrational condition where Followers have to expend cognitive resources deciding what movement they want to make. Again, our study was not designed to capture this interaction effect in our planned comparisons and this is something that could be addressed in future work. Again, extending the neuroimaging to the whole brain would also be useful to understand what else is happening while the Follower is making their move. Finally, we can speculate that both

being watched or not being watched may exact different cognitive demands being atypical depending on the context: it may be that being watched may require us to monitor our partner's attention, taking resources away from areas such as the mentalising networks that are often implicated in controlling imitation. On a similar vein, it may also be that not being watched is more atypical and require more mentalising, leading to a relative deactivation in the watched condition. Both these ideas are worth exploring further. As we note in Chapter 4, we have not seen any existing research that touches on these dynamics (see Section 1.4.3), but repeating these studies adapted to fMRI could shed some light on neural activations in other parts of the brain when being watched versus not being watched.

### *6.1.4. Responding to irrational actions*

Another key hypothesis tested as part of the social-signalling hypothesis of imitation was whether participants recognise (consciously or unconsciously) actions as irrational. Amongst neurotypicals, previous studies have shown that viewing videos of irrational actions in fMRI studies have been accompanied by activations in the STS as well as in the adjacent MTG (Grèzes et al., 2004; Jastorff et al., 2011; Marsh, Mullett, et al., 2014; Pelphrey et al., 2003; Saxe et al., 2004). Previous observational studies have shown increased activation in the right TPJ and right IPL when observing irrational actions (Brass et al., 2007; Marsh, Mullett, et al., 2014; Marsh & Hamilton, 2011; Oliver et al., 2017). Thus, these studies broadly implicate the action observation and mentalising regions of the brain in identifying irrational actions. Both these phenomena are also thought to be impaired in autism, with studies showing deficits in in both action-observation (Iacoboni & Dapretto, 2006; Ramachandran & Oberman, 2006) and in mentalising (Baron-Cohen et al., 1985; Castelli, 2002; Frith, 2001; Frith & Frith, 2003; Jolliffe & Baron-Cohen, 1999) among autistic individuals. However, the evidence on how autistic individuals interpret action rationality is mixed. Hamilton and colleagues (2007) have shown that both neurotypical and autistic children are able to distinguish between rational and irrational actions equally well.

Nevertheless, there are differences between neurotypicals and autistic individuals in how they respond to irrational actions. A study by Hobson and Hobson (2008) showed that when viewing an action sequence with irrational elements that achieves a goal, autistic individuals only copied the style of an action insofar as it was relevant to achieving the goal while neurotypicals copied the style of action used to achieve the goal regardless of its irrationality. This raises the question of whether it is because autistic individuals

recognise an action as irrational and make a (conscious or unconscious) choice to discard the irrational elements in favour of efficiency, while neurotypicals copy the style regardless to achieve social goals. An alternate explanation is that autistic individuals just do not pay any attention to the style unless it is germane to the goal. A study by Marsh and colleagues (2015) showed both rational and irrational actions to autistic individuals as well as neurotypicals: they found that those with autism showed reduced attention to features of the action such as the hand performing the action; however, in trials where participants with autism did focus on these features, their performance was similar to the neurotypicals.

In the studies in this thesis, we found that neurotypicals showed deactivation in the right IPL when responding immediately after an irrational demonstration. In Chapter 5 we also saw that this extended into the left IPL for neurotypicals. However, autistic participants showed activations only in the left IPL (not the right IPL) and showed engagement in the left occipitotemporal regions when responding to irrational actions. Our results suggest that there is engagement in the action-observation network when viewing or responding to irrational actions, but there are differences between neurotypicals and autistic individuals in how they perceive and process irrational actions. This is particularly intriguing given that there were no significant differences in behaviour between autistic and neurotypical individuals (see 6.1.2). This suggests that autistic individuals may be undertaking some form of cognitive compensation, adjusting their behaviour either consciously or unconsciously to match their understanding of what is expected of neurotypicals.

## 6.2. General limitations

In this thesis one of our priorities was to design a paradigm that would enable us to study imitative behaviour among interacting partners in a social context. While this paradigm generated useful insights, there are some questions that we were not able to answer using this paradigm.

First, given that we used multiple trials, we cannot determine if and when Followers became consciously aware of the Leader's unusual trajectories. In the studies in Chapter 3 and the study in Chapter 5, there was a final phase in the experiments where Followers were explicitly told about the irrationally high trajectories and required to copy them faithfully. Thus, in the debrief at the end there was no reliable way to test when they become aware of the irrationally high trajectories in a systematic manner. We know from the neural correlates that there was increased activation for both neurotypicals and

autistic participants in the left IPL when viewing or responding to irrational actions. However, we do not know whether this was something processed consciously or subconsciously, and similarly whether their subsequent (copying) behaviour was conscious or unconscious.

Second, the design of the experiment does not allow us to distinguish between moving higher and making an irrational movement. It would be useful to attempt to replicate these results by manipulating rationality through the presence or absence of an obstacle, while keeping the movement kinematics the same between the rational and irrational condition (similar to the habituation paradigm used in Gergely et al., 1995). For example, we could design a study where in one-half of the trials there is an obstacle between the source and destination of the blocks. In the other half of the trials the obstacle is placed either before the source or after the destination. In all trials the Leader would make a high trajectory to move the blocks from the source to the destination. But in the obstacle-in-between trials this would be a rational movement, while in the no-obstacle-in-between trials this would be an irrational movement. We could then maintain the same split of Watched and Unwatched trials, and see if the Followers copy the Leader's trajectories in various trials. This would us to generalise our results further.

Finally, we found differences in the neural correlates between the watched and unwatched conditions, but as outlined above (see Section 6.1.3) we cannot separate out the drivers of this difference such as whether the unwatched condition is more atypical, forces participants to generate their own motor plan, or this is due to some other explanation we have not yet considered. As above, manipulating the rationality of the movements without changing the kinematics of the movement required (i.e., leave the movement the same, but make it rational or irrational by the presence or absence of obstacles in the way) would enable us to parse these differences.

## 6.3. Future directions

The studies described in this thesis support the social-signalling hypothesis of imitation and generate several possible directions for future research. First, if imitation is a social signal, what is the message being sent? One suggestion is Followers may be copying the high trajectory demonstrated by the Leader in the irrational condition to show the Leader that they are committed to gaining points. Also, in our exploratory pilot study (Chapter 2) we found that Followers imitated some Leaders with greater fidelity than others, although we did not find any links between any self-reported traits and imitative fidelity. Exploring whether we imitate people we already feel a kinship with to a greater extent,

or imitate people we want to affiliate with more to a greater extent would be an interesting avenue to explore. The mutual prediction account advanced by Kingsbury et al. (2019) found that extent to which incorporating an interaction partner's brain signals improved the GLM was dependent on the subordinate-dominant relationship between the two animals. While there were no explicit power differentials between Leaders and Followers in our studies, this could also be a worthwhile variable to manipulate. This would enable us to understand whether the increase in imitative fidelity when we are being watched is a conscious "Machiavellian" instinct, or a more unconscious process.

Incorporating eye gaze measurements into the paradigm used in these studies could also prove fruitful. Neurotypical and autistic participants have been shown to orient their gaze differently, paying attention to different aspects of an action sequence such as goal or kinematic features such as the trajectory or hand used to make a movement (Falck-Ytter, 2010; Flanagan & Johansson, 2003; Senju et al., 2009). We know that the neural signals recorded were different for autistic and neurotypical participants, and we know that their behaviours were similar. Evaluating eye gaze may help us separate out where the differences arise in the underlying mechanisms between the two groups.

Finally, it would also be worthwhile exploring the downstream consequences of being imitated in a more systematic manner. The evidence on being copied and the increase in prosociality showcase a wide range of positive outcomes (Chartrand & Lakin, 2013; Fischer-Lokou et al., 2011; Stel et al., 2008; Tanner et al., 2008; van Baaren et al., 2003, 2004; van Swol, 2003). However, it is unclear if this is driven by a specific kinship to an interaction partner who copies us, or a more general boost to one's ego or sense of wellbeing from being copied. Do we feel a specific social contract come into play when we are copied by an individual? Or do we just feel better about ourselves as someone worthy of being copied? This would be an interesting avenue to study in the future.

## 6.4. Closing summary

In this thesis I have outlined the social-signalling hypothesis of imitation and developed a paradigm that enables us to test individual steps of this theory in a systematic manner. Across four behavioural and neuroimaging studies we found evidence to support this hypothesis. The evidence also raised intriguing questions about how we process being watched and not being watched, as well as around the performance of autistic participants who matched neurotypicals on behavioural measures but showed some differences in neural activations. These findings support the view that imitation can function as a social

signal. This social-signalling theory of imitation is a useful construct through which to understand imitative behaviour amongst people operating in a social context.

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