*Rewards have a transient and task-specific effect on saccade latency and accuracy*

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## Abstract

The focus of this thesis was on investigating the key questions regarding the effectiveness of monetary rewards as a tool for behaviour change in rehabilitation. Firstly, do rewards consistently influence the eye movement behaviour in a neuro-typical human population? Secondly, do these effects persist once rewards are withdrawn? Finally, do these effects transfer to other unrewarded eye movement tasks? Nine experiments investigated the influence of monetary rewards on oculomotor function and attention in humans. Monetary rewards were found to consistently influence human saccadic behaviour such that faster eye movements were generated to rewarded locations compared to unrewarded locations. These effects persisted for a short period of time after rewards were withdrawn before extinguishing quickly. However, these hemifield-specific effects failed to transfer to any secondary unrewarded eye movement task, but instead produced a more general effect of reward in one experiment conducted. The present set of experiments have established a reward paradigm able to consistently produce behaviour change when rewards are present; however these effects were found to be context and task-specific.

The findings of the present set of experiments have highlighted the transient nature of the effects of reward and provide a framework for the future use of monetary rewards as a tool for behaviour change. The findings provided by the present set of experiments can be harnessed in future to guide the effectiveness of monetary reinforcers in a neuro-atypical population.

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## Statement of Copyright

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# Chapter 1 – The effects of reward on the saccadic eye movement system

## 1.1 General Introduction

The relationship between reward and behaviour has become a central theme in psychology (Balleine & Dickinson, 1998), with an increasing number of studies focusing on the link between reward and eye movements. A well-established effect of rewarding eye movements in non-human primates has previously been found, such that primates' eye movements are faster and more accurate to rewarded locations (Bendiksby & Platt, 2006; Takikawa, Kawagoe & Hikosaka, 2002; Kawagoe, Takikawa & Hikosaka, 1998), a finding replicated in healthy humans (Milstein & Dorris, 2007).

More recently, the use of incentives as a tool in the rehabilitation of visual deficits, has been proposed, with monetary rewards found to alleviate the deficits associated with spatial neglect (Lucas et al., 2013; Malhotra, Soto & Russell, 2013). This research suggests that there is scope for the use of monetary rewards in the rehabilitation of visual field deficits. Homonymous hemianopia is a visual field deficit manifesting as blindness in the left or the right visual field. This particular visual field deficit is caused by unilateral damage to the visual cortex or from the nerve fibres with projections in this area often due to stroke in one side of the brain. For the patient, this hemi-blindness is extremely debilitating as everyday activities, such as crossing the road, driving and avoiding obstacles become an extremely difficult or impossible task. Therefore, this type of deficit can leave sufferers isolated and reliant on others. At present there a number of compensatory strategies where patients are trained to make elaborately large eye movements to explore their blind field. However these strategies are not developed spontaneously. Recent research has displayed that with appropriate training, patients can learn strategies that allow for more effective exploration of the blind field, compensating for the area of blindness (Kasten & Sabel, 1995; Kasten et al., 1997). These treatments have limited efficiency however, as they fail to produce persistent behaviour that can be used on untrained tasks. Furthermore, these treatments rely on the patient developing a conscious strategy for exploration; a problematic requirement as the most effective rehabilitation occurs when a patient is able to form a habit not requiring a conscious effort. If rewarding patients is a sustainable way of altering the deficits associated with certain visual field deficits then using monetary rewards in rehabilitation could offer an attractive and cost-effective way of enhancing existing rehabilitation tools (e.g. Aimola et al. 2014; Lane et al., 2010).

Although the empirical evidence from neglect patients points to monetary incentives being a successful tool for rehabilitation of other visual field deficits, this research is still in its infancy. At present, research has failed to answer a number of key questions to assess the effectiveness of rewards in rehabilitation. Firstly, it is unknown whether the effects of reward generalise across a neuro-typical human population, consistently influencing oculomotor function. Secondly, studies have failed to probe the persistence of reward effects once they are withdrawn. Before money can be considered as a potential tool for use in patient rehabilitation, investigation of the persistence of reward learning once incentives are withdrawn is required. In this way the effectiveness of this reinforcer can be gauged. Finally, it is also unknown whether influences of reward are context and task-specific or transfer to other unrewarded tasks. These key questions will be the focus of the present set of experiments.

Studies have also shown that the effects of reward extend to attention. Rewards have been found to affect non-spatial features, such that specific featural aspects of objects associated with monetary rewards can capture the eyes in a stronger fashion than stimulus features without reward associations (Theeuwes & Belopolsky, 2012), consistent with other findings (Della Libera & Chelazzi, 2009; Anderson, Laurent & Yantis, 2011a; Hickey, Chelazzi & Theeuwes, 2010a; Theeuwes, 1991; 1992). However, significantly less is known regarding the specific effects of reward on spatial attention. Only one known study has investigated whether the value associated with a location affected the capture of attention (Camara, Manohar & Husain, 2013). Findings demonstrated that a location associated with a positive outcome were able to bias goal-directed and stimulus-driven deployment of attention, even when these reward locations varied on a trial-by-trial basis. Although this previous empirical research suggests that rewards influence the deployment of attention to specific spatial locations, this question has not been fully explored. Firstly, it is difficult to tease apart the effects of rewards on attention and the effects on oculomotor functioning. Secondly, the key questions remain of whether these effects persist after rewards are withdrawn and whether these effects can transfer to untrained tasks or are specific to tasks featuring rewards.

This thesis aims to evaluate the applicability of monetary incentives as a tool for rehabilitation by creating a reward paradigm able to produce optimal behaviour change in human oculomotor behaviour. Using a neuro-typical human population in an attempt to explore the effects of reward in the human oculomotor system, this thesis aims to investigate three key areas: 1) whether monetary incentives produce changes in the metrics of eye movements; 2) whether any effects

generated persist once rewards are withdrawn; 3) whether any effects transfer to unrewarded eye movement tasks.

The introduction to this thesis will provide the background to the research topic of reinforcement learning, and the distinction between habits and goals. An account of the neural circuitry involved in saccade generation and the encoding and processing of reward information will be outlined. The linked structures involved in both of these processes (reward encoding and saccadic eye movements) will be discussed in detail. The main models of oculomotor control will be described with explanations of how these models account for oculomotor behaviour and phenomena. Finally, a discussion of the development of reward research will be provided, regarding reward theories in psychology, behavioural influences of how incentives influence both primates and humans, and the justification as to why research exploring the transfer and persistence of the effects of reward are necessary.

## 1.2 Learning Theories

### 1.2.1 Reinforcement Learning

Reinforcement learning relates to how organisms can learn to behave so as to maximise the rewards and minimise the punishments they receive. At the core of every organism is the ability to learn to obtain the things they need and want and avoid those that are harmful and undesirable. This is known as the optimisation problem. Reinforcement learning offers formal, mechanistic solutions to this problem. The law of effect (Thorndike, 1911) suggests that all instrumental learning consists of the learning of Stimulus-Reward (S-R) associations. However, animals also learn Action-Outcome (A-O) or more generally, Situation-Action-Outcome (S-A-O) contingencies (Dickinson, 1985, 1994; Tolman, 1932). S-R associations are often called habits (Dickinson, 1994; Packard & Knowlton, 2002) as they are learned and are autonomous from the outcome. Actions guided by the knowledge of A-O and S-A-O contingencies are called goal-directed. The test of whether something is a habit is whether after it is learned it is insensitive to manipulations of the value of the outcome (Dickinson, 1985). For example, if after training, lever pressing for a sucrose reinforcer has become a habit, rats will continue to press the lever even after they have undergone aversion conditioning to sucrose and are no longer interested in consuming it (Adams, 1982). In contrast, goal-directed actions are immediately sensitive to reinforce re-evaluation procedures. For example, if due to limited training, lever pressing for a sucrose reinforcer is still under control of goal-directed systems, rats stop pressing the lever after

they have undergone averse conditioning (Adams, 1982). This difference between habits and goal-directed learning is crucial when considering the use of rewards in rehabilitation. To make persistent behaviour change necessary for rehabilitation of visual field deficits, it is necessary to generate habits rather than goal-directed outcomes in any learning paradigms employed within this thesis. If instead goal-directed outcomes are generated, then after reinforcement is removed any behaviour change will not persist. Therefore, it is important to outline the previous literature regarding reinforcement learning as to guide the creation of reward paradigms and ensure habits are generated that create persistent behaviour instead of goal-directed actions that will extinguish over time.

Habits function almost like reflexes, such that a given stimulus or situation can automatically trigger a response most strongly associated with it (Dickinson, 1985, 1994). An S-R association is between a state and an action. The strength of this association corresponds to the performance for a given action in a given state. Instrumental conditioning can result in either habit or goal-directed actions depending on the parameters of the training procedure. The effectiveness of a reinforcer can be increased or decreased by various factors. For example, the effectiveness of a reinforcer will be reduced if the individual's 'appetite' for the source of stimulation has been satisfied. Satiation is generally only a factor to consider when using primary reinforcers such as food and water. The principle of satiation exists to maintain an individual's homeostasis. Size is also a contributing factor to the efficiency of reinforcement learning (Schneider, 1973; Davison & Baum, 2003). This factor is a cost-benefit determinant of whether a consequence will be effective. If the amount of reinforcer is large, the effort put in to receive it is worthwhile and as such the consequence will have a greater effect upon the behaviour.

Other factors that can influence the effectiveness of a reinforcer exist for neurochemical reasons. Immediacy, for example, suggests that after a response, the immediacy of a consequence directly relates to the effectiveness of a reinforcer; the faster the feedback, the more effective the reinforcer. Furthermore, if a consequence fails to consistently follow the target response, its effectiveness upon the response is reduced. This factor is known as contingency. However, if a consequence follows the response consistently after successive instances, its ability to modify the response is increased. When a consistent schedule of reinforcement is employed, faster learning occurs. However, this can impact upon extinction. Extinction is more difficult when learning occurs during intermittent reinforcement and more easily extinguished when learning occurs during a highly consistent schedule. By using a variable-ratio schedule, where rewards are given based on a predefined probability, the predictability of rewards is significantly reduced, which

also increases the duration of conditioning and resistance to extinction. This has been supported experimentally in a study conducted by Sheffield (1949). Using a population of rats, one group were rewarded continuously on every trial while a second group were rewarded only 50% of the time. The continuously rewarded group showed greater performance during the acquisition phase. However, when extinction occurred the partially reinforced group was slower to stop responding. The non-rewarded trials during acquisition served to make the behaviour more persistent. This is known as the Partial Reinforcement Extinction Effect (PREE). More recently, the sequential theory was proposed as a rival to the PREE. It is a sophisticated version of the idea that behaviour will persist in extinction as long as the stimulus conditions are similar to those persistent in acquisition. Extinction comprises many non-rewarded trials, and partially reinforced subjects have been reinforced by responding after they have received non-rewarded trials. In contrast, continuously reinforced subjects have not. Capaldi (1967, 1994) has proposed that the crucial stimulus is the memory of the previous trials. During the acquisition phase, partially reinforced subjects are reinforced while they remember recent non-rewarded trials, whereas participants who are continuously reinforced are constantly reminded of their recent reinforcement.

To produce consistent and persistent behaviour change it is important to consider these factors which can influence the effectiveness of a reinforcer when building a reward paradigm able to bias the oculomotor system in humans. As the principle of satiation refers to primary reinforcers only (Berridge, 2000), this principle is not applicable to the generation of reward paradigms within this thesis, which will use money; a secondary reinforcer. As discussed previously the size of a reinforcer has an impact upon its effectiveness as a reinforcing agent. Therefore it is crucial to offer a reward that matches or exceeds the participant's expectation of what they deserve. Secondly, considering the principle of immediacy when delivering rewards after performing a task, or in the case of this thesis a saccade, it is imperative for effective reinforcement that reward feedback is delivered rapidly after performance of a task as this creates the most effective reinforcement (Berridge, 2000). Therefore, in any paradigm used, it is important to ensure fast delivery of reward feedback between action and reinforcer. Finally, the principle of contingency has been highlighted as a key factor in the creation of an effective reward paradigm. Previous empirical research discussed has highlighted that rapid behaviour change can occur when rewards are received on successive trials, but this schedule fails to last into extinction (Sheffield, 1949). Alternatively, when reinforcement is intermittent, the schedule is harder to learn, but the effects are more persistent once rewards are removed. This is key when considering that a reward schedule used in the rehabilitation of visual field deficits needs to persist once rewards are

withdrawn in order to be classified as effective. Therefore, a variable-reward schedule where rewards are delivered intermittently should produce the most consistent and persistent effects on oculomotor behaviour.

Within this section the key principles of reinforcement learning have been discussed. Through instrumental conditioning, organisms can learn stimulus-reward associations resulting in the generation of habits. The strength of this formed relationship can be manipulated in a several key ways, including the immediacy of reward feedback, the magnitude of reward and the contingency of reward. As a result, these factors need to be closely considered when designing a reward paradigm able to induce functional changes to the oculomotor system. By considering these principles it is hoped that a reward paradigm able to influence oculomotor control in humans and create a sustainable behaviour change can be generated.

### 1.2.2 Models of Reinforcement Learning

A number of theories have been developed to explain behaviour in relation to reinforcement learning, the most influential of which is the Rescorla-Wagner model (Rescorla & Wagner, 1972). The model postulated that learning only occurs when events violate expectations. For example, in a conditioning trial in which two conditioned stimuli, such as a light and a tone (the conditioned stimuli) are presented, as well as an affective stimulus, such as food (the unconditioned stimulus), the associative strength of each of the conditioned stimuli will change. Learning is driven by the discrepancy between what was predicted and what actually happened. At the basis of this model, two important assumptions exist: 1) learning only happens when events are not predicted; 2) when different stimuli are used within a single trial these stimuli are summated to form the total prediction in a trial. With these two assumptions, the model is able to account for an overwhelming amount of behavioural data. However, the model does suffer from two major shortcomings. Firstly, by treating the conditional and unconditional stimuli as qualitatively different, the model does not explain second-order conditioning; whereby if stimulus B predicts an affective outcome (such as food) and stimulus A predicts stimulus B, then stimulus A also gains reward predictive value. Therefore, this model fails to grant explanation of the effects of monetary rewards on human behaviour, as monetary rewards are a secondary order predictor of a wide range of affectively desirable unconditional stimuli, such as food and shelter. The second shortcoming of this model is that its basic unit of learning is a conditioning trial as a discrete temporal object. This explanation fails to account for the sensitivity of conditioning to the

different temporal relations between the conditional and unconditional stimuli within a trial, such as whether they appeared simultaneously, serially and whether there was a time difference between them.

To overcome these issues, Sutton and Barto (1990) proposed an extension to the Rescorla-Wagner model by including a temporal difference (TD) learning rule in order to account for the timing of different events. In TD learning, the goal of the learning system is to estimate the values of different states or situations in terms of the future outcomes that they predict. Consistent with the Rescorla-Wagner model, learning is driven by discrepancies between available and expected outcomes. However, one difference is that in TD learning, time within a trial is explicitly represented and learning occurs at timepoints within a trial. Furthermore, stimuli within trials create long-lasting memory representations and a separate value is learned for every timepoint of this trace (eg. a stimulus might predict a reward five seconds after its presentation). A second difference is how predictions are constructed in each of the models. In TD learning the associative strength of a stimulus at any given time is taken to predict not only immediate rewards but also future predictions, due to those stimuli that will still be available in the next time-step, discounting future delayed predictions.

The theories put forward by these models are valid when the probabilities of transitioning between different states of the environment are fixed. However, since the environment rewards us for our actions, not predictions, one might argue the ultimate goal of prediction learning is to aid action selection. The correct assignment of credit is crucial for learning to improve by repeating actions leading to reward and avoiding those leading to punishment. Reinforcement learning solves the credit assignment problem (Barto, Sutton & Anderson, 1983; Sutton & Barto, 1998) by basing action selection on both immediate outcomes and future value predictions. In this way optimal action selection occurs.

A third theory, inspired by neural-network models of learning (Barto et al., 1983), proposes a learning system comprised of two neuron-like elements. The Adaptive Critic Element (ACE) constructs an evaluation of different states of the environment through the use of TD learning. This is used to increase the external reinforcement signal and, through a trial and error process, train a second unit defined as the Associative Search Element (ASE), to select the correct action at each state. These two components provide the precursors for the Actor/Critic framework for model-free action selection closely associated with reinforcement learning and action selection in the brain. Within this model, a Critic model uses TD learning to estimate state values from experience with the environment and train the Actor module, which maintains and learns a

policy. Some of the strongest links between reinforcement learning methods and neurobiological data regarding both animal and human decision making have been related to the Actor/Critic framework. Specifically, the methods employed in Actor/Critic models have been extensively linked to instrumental action selection and Pavlovian learning in the basal ganglia (Barto, 1995; Houk, Adams & Barto, 1995; Joel, Niv & Ruppin, 2002). Based on prior experience and patterns of response, the brain expects (or predicts) what will happen with a certain stimulus or situation. When the signal is different from what is expected, a prediction error occurs. This can be used to 'teach' the brain to respond better (Schultz, 2000; Schultz, Dayan & Montague, 1997). In animals, dopamine neurons fire when an animal receives an unexpected reward. However, if the animal has learnt to associate a conditioned stimulus with subsequent reward, dopamine neurons fire to the conditioned stimulus but not the reward. Furthermore, if the conditioned stimulus is presented and then the predicted reward is omitted, dopamine neurons fire below baseline at the time the reward should have been delivered (Schultz, 1998). These findings are consistent with the idea that dopamine neurons report prediction errors (Hollerman & Schultz, 1998). Unpredicted rewards and conditioned stimuli that predict future rewards produce positive prediction errors. Rewards that are fully predicted do not produce prediction error. Omitted rewards produce negative prediction error. Human fMRI findings have reported activation reflecting prediction error in areas richly innervated by dopaminergic afferents (stratum and orbitofrontal cortex) (Bray & O'Doherty, 2007; McClure, Berns & Montague, 2003; O'Doherty, Dayan, Friston, Critchley & Dolan, 2003; Pagnoni, Zink, Montague & Berns, 2002; Pessiglione, Seymour, Flandin, Dolan & Frith, 2006). Presence or absence of activity related to prediction error in the striatum distinguishes participants who learn to perform optimally from those who do not (Schonenberg, Daw, Joel & O'Doherty, 2007). These findings are believed to reflect dopaminergic input.

The theories surrounding reinforcement learning have been used to account for a great deal of behavioural research. The reward paradigm used in the present thesis can be constructed by closely following the principles set out in these theories. Taking into account the TD model of learning it is clear that timing within a trial is a very important factor to consider. The TD learning model suggests that learning is linked to time points within a trial. Therefore, keeping the timing of trials consistent, such as stimulus onset and reward feedback, will allow participants to become familiar with the timing of trials, facilitating learning. It is also important to consider the Actor/Critic model of learning and its implication of the basal ganglia as a significant structure of reinforcement learning. This particular structure will be discussed in more detail in a later section

of this chapter. It is crucial to adhere to the policies implemented by these models of reinforcement learning in order to create the optimal reward learning paradigm.

### 1.2.3 Theories of Monetary Reward and Behaviour

Most experimental findings in reinforcement learning using animals utilise primary rewards, such as food or juice. Experiments with humans sometimes use primary rewards, but more often use money, which is a secondary reward. Secondary rewards are stimuli that acquire rewarding properties by virtue of being paired with primary rewards. There are a number of theories that have aimed to directly address the effects of monetary reward on human behaviour. The following section will outline the two critical and most relevant theories proposed regarding the effects of reward on human behaviour; the Expectancy Theory and the Cognitive Evaluation Theory.

The Expectancy Theory, proposed by Vroom (1964) suggests that humans act to maximise their expected satisfaction in any given situation. Vroom (1964) postulated that an individual's motivation in a particular situation is dependent on two factors: 1) the expectancy between the effort required and the particular outcome; 2) the valence of the outcome itself. Therefore, individuals invest a level of effort that they believe will lead to a desired outcome. Vroom (1964) suggested that the effect of incentives on effort is two-fold. Firstly, the outcome of interest is the financial reward. Money can have valence due to a variety of reasons. Vroom (1964) argued that the valence of money comes from money being instrumental in obtaining things people desire, such as material goods. In addition, money holds symbolic value due to its perceived relationship to prestige, status and other factors (Furnham & Argyle, 1998; Zeliser, 1994). Secondly, expectancies have been found to be higher under monetary incentives than no pay due to stronger links among effort, performance and pay (Jorgenson, Dunnette & Pritchard, 1973; Locke & Latham, 1990). Therefore, according to the expectancy theory, an individual's motivation and subsequent effort are significantly higher when compensation is based on performance due to both an increased expectancy about the effort-outcome relationship and an increased valence in the outcome. Therefore, monetary incentives lead to optimal behaviour.

An alternative model of reward on behaviour is the Cognitive Evaluation Theory (CET) proposed by Ryan and Deci (2000). Motivation is defined as the intensity and direction of effort. Intensity refers to the quantity of effort and direction refers to what individuals are drawn to. There are two clear forms of motivation; intrinsic and extrinsic. Intrinsic motivation exists in the individual

rather than relying on external sources and refers to the motivation driven by interest in completing a task. Extrinsic motivation refers to the performance of an activity in order to attain an outcome, such as monetary reward. The CET focuses on the factors which can influence an individual's intrinsic motivation. The theory proposes that extrinsic rewards have two key properties that can influence intrinsic motivation; information and control. It is these factors that can influence an individual's self-determination and task competency. The informational factor relays information about an individual's competency. The CET suggests that if the informational factor relays an individual's competence, intrinsic motivation is enhanced. Conversely, if a reward fails to relay competence, intrinsic motivation is decreased. The controlling aspect of rewards are able to influence an individual's locus of causality, defined as the degree to which an individual perceives their behaviour to be freely determined or due to an external cause. The CET proposes that if a reward is perceived as controlling, intrinsic motivation will be decreased. However, if a reward is perceived as non-controlling, intrinsic motivation will be high. The theory suggests that offering rewards for completing a task shifts individual's extrinsic motivation, undermining their pre-existing intrinsic motivation. However, removal of rewards leads to decreased interest in the task and the prior intrinsic motivation does not return. The theory suggests that extrinsic rewards must be continuously offered to sustain optimal performance. According to this theory, individuals pay more attention to the external reward offered for an activity than the inherent enjoyment and satisfaction of completing an activity. In this way an external incentive, such as money, decreases an individuals' intrinsic motivation to perform a task; termed the overjustification effect (Greene, Sternberg & Lepper, 1976). The overall effect of offering a reward for a previously unrewarded activity is a shift to extrinsic motivation and the undermining of individual's pre-existing intrinsic motivation. Once rewards are withdrawn, interest in the activity is lost, with the prior intrinsic motivation never returning (Deci & Ryan, 1985). This effect has been found in human behaviour in a number of settings including in education (Lepper, Greene & Nisbett, 1973; Flora & Flora, 1999) and the workplace (Gagne & Deci, 2005).

The two models described in this section have implications for the present set of experiments and offer contrasting views regarding the influence of monetary rewards on human behaviour. Although the Expectancy Theory suggests that monetary incentives result in increased effort and performance on tasks, the overjustification effect within the CET suggests that rewards shift participants' motivation such that when rewards are no longer given, motivation to perform a task is lost. This effect is critical for the use of monetary rewards in rehabilitation, as rewarding eye movements may result in a change in oculomotor behaviour, but the withdrawal of rewards may result in this change being extinguished. As such, the use of financial rewards in

rehabilitation may not be a viable possibility if the behaviour change is not sustainable once rewards are removed.

## 1.3 The Eye Movement System

As the present set of experiments investigated the effects of monetary rewards on oculomotor behaviour it is important to describe the eye movement system in some detail. Therefore, the following section will outline the eye movement system with particular focus on the key areas involved in the generation of saccadic eye movements and the substantial overlap between these areas and reward processing in the brain.

### 1.3.1 Properties of the Eye

The oculomotor plant consists of the eyeball, the extraocular muscles and the surrounding orbital tissue. The eye itself has limited inertia and rotates around a point that is relatively fixed. There is no stretch reflex and activity in each of the three antagonistic muscle pairs is reciprocally related. The precise position of each eye in its orbit is under the control of six extraocular muscles. Extraocular muscles generate the forces necessary to overcome the elasticity and viscosity of the oculomotor plant. Horizontal eye movements are controlled by the medial and lateral rectus muscles, while vertical and torsional movements are controlled by the superior and inferior rectus and oblique muscles. These three pairs of muscles allow the eye to rotate within three degrees of freedom. In most cortical and subcortical visual areas, the fovea has the greatest representation, emphasising the importance of foveal vision in aspects of visual processing and visually guided behaviour (Dow, Snyder, Vautin, & Bauer 1981; Van Essen, Newsome & Maunsell, 1984). To maximise the efficiency of foveal vision we have the ability to align the fovea rapidly to novel targets that could appear unexpectedly in the visual field and keep the fovea aligned upon these targets for a period of time so that the visual system can perform a comprehensive analysis of the image. Therefore, we have the ability to both move the eyes from one target to another and the ability to suppress eye movements to irrelevant stimuli or locations and maintain foveal vision at a specific location as demanded by the task. Thus saccades are used to redirect the fovea from one target of interest to another and a fixation mechanism is used to keep the fovea aligned on the target during subsequent image analysis. This alternating behaviour of saccade-fixate is repeated several hundred thousand times a day and is crucial for completing complex acts including visual search, driving and reading.

### 1.3.2 Brain Mechanisms

A network of cortical and subcortical regions is involved in the generation of saccadic eye movements including the frontal and the parietal cortices, the superior colliculus, the thalamus, the basal ganglia, the cerebellum and the brainstem reticular formation (Schall & Thompson 1999; Munõz, Dorris, Pare, & Everling, 2000; Scudder, Kaneko, & Fuchs, 2002). Figure 1.1 is taken from Munõz (2002) and displays a schematic of the neural circuitry involved in the generation of eye movements.

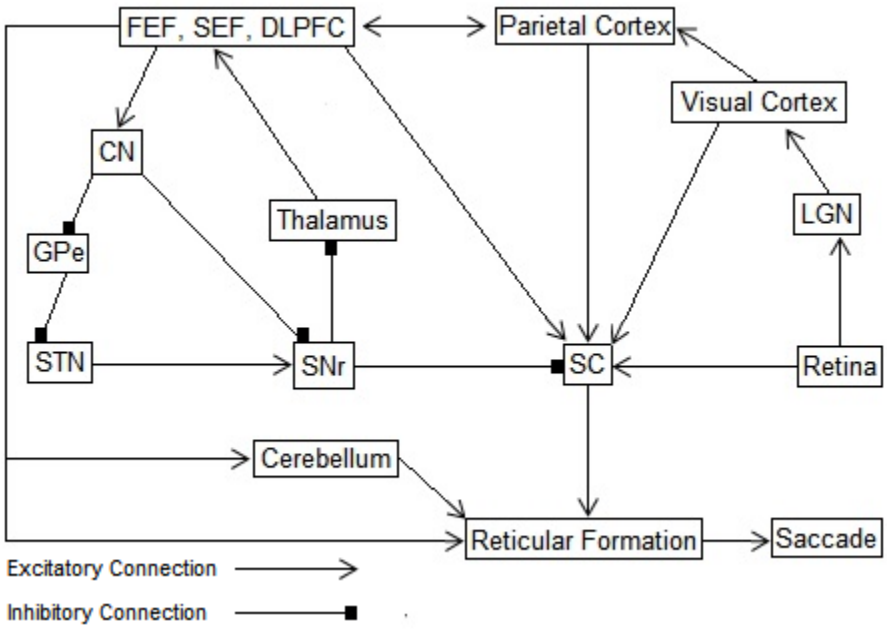


***Figure 1.1:*** *The circuitry connecting the brain areas involved in the generation of saccadic eye movements. Abbreviations: CN, Caudate Nucleus; DLPFC, Dorsolateral Prefrontal Cortex; FEF, Frontal Eye Field; GPe, Globus Pallidus external; LGN, Lateral Geniculate Nucleus; LIP, Lateral Intraparietal Area; SC, Superior Colliculus; SEF, Supplementary Eye Field; SNr, Substantia Nigra Pars Reticulata; STN Subthalamic Nucleus. (Munõz, 2002)*

A variety of recording and experimental techniques in humans and animals has generated a large body of data from which the role of the various brain areas involved in both visual fixations and saccade generation has been explained. Functional imaging techniques in humans, where changes

occur in either the metabolism of oxygen or the blood flow in different brain areas, has been correlated with different aspects of eye movement behaviour. Additionally, since the saccade generation system can be disrupted by psychological and neurological disorders, patients with any of these, and those with discrete lesions to a specific brain area have also been studied. Techniques used in animal studies have additionally used single cell recordings, lesion studies, electromicrostimulation and neuronal activation or deactivation using transmitter substances (Munõz, 2002). It is through these techniques that the generation of saccadic eye movements is a well understood process. The following section will outline what is known about how the eye moves and the role of key structures in the neural circuitry outlined above.

### 1.3.3 Components of the Neuro-Visual System

#### *1.3.3.1 Frontal Eye Field (FEF)*

The frontal eye field (FEF) is reciprocally connected with the occipital, temporal and parietal cortex as well as neighbouring and contralateral areas of the prefrontal cortex (PFC) (Huerta, Krubitzer & Kaas, 1987; Kunzle & Akert, 1977; Maioli et al., 1983). The FEF also receives inputs from the substantia nigra (SNr), the superficial and intermediate layers of the superior colliculus (SC) and the thalamus (Lynch, Hoover & Strick, 1994; Tian & Lynch, 1997). Neurons in the FEF project heavily to the SC (Huerta, Krubitzer & Kaas, 1986; Stanton, Goldberg & Bruce, 1988; Distel & Fries, 1982), the caudate nucleus (CN) and the putamen (Stanton, Goldberg & Bruce, 1998), and regions in the brainstem (Buttner-Ennever & Horn, 1997; Moschovakis & Highstein, 1994).

Neurons within the FEF are thought to play a critical role in transforming visual information into saccade commands (Bruce, Friedman, Kraus & Stanton, 2004). There are several types of FEF neurons including those that respond prior to and during the generation of saccadic eye movements (saccade and motor neurons), neurons that pause during saccades but are active during fixation (fixation neurons) and neurons that respond when a behaviourally relevant stimulus is in its receptive field (visual neurons) (Bruce et al., 2004; Schall, 2002). The most common FEF neuron responds to both visual stimulation and motor plans (visuomotor neurons). Increases in cerebral blood flow in the FEF have been found in a number of oculomotor tasks. FEF activation has been observed during saccadic eye movements made in both darkness and light (Cohen, Heitz, Schall & Woodman, 2009b). Therefore, the only requirement necessary to activate the FEF is that of saccadic eye movement generation irrespective of the presence of visual targets and independent of task context.

Early single unit recording initially raised doubts as to the role of the FEF in saccade generation, as the activity of the FEF neurons followed rather than preceded spontaneous saccades (Bizzi, 1968; Bizzi & Schiller, 1970). Würtz and Mohler (1976) evidenced that approximately half of the neurons in the FEF had visual responses and many neurons selectively enhanced their responses when primates made a saccade towards a target stimulus. Goldberg and Bushnell (1981) extended this finding highlighting that this enhancement was both spatially selective and presaccadic, suggesting that this activity represented the neural correlate for the generation of visually guided eye movements. Strong support for saccade-related activity in the FEF was supplied by single-unit recordings of activity in primate FEF (Bruce & Goldberg, 1985). Through this method, pre-saccadic activity was observed in around 50% of the FEF neurons. This visual activity was further classified as visual movement and anticipatory. Approximately 40% of presaccadic neurons had visual activity, but no movement-related activity. 20% had movement-related activity such that they discharged before purposive saccades with or without a visual target. These neurons were less active or not active at all when the primate spontaneously made saccades in the dark. The remaining 40% of FEF neurons had both visual and movement activity and were classified as visuomovement cells. These neurons responded to visual stimuli but also discharged for saccades made with or without a visual target.

Empirical research has also shown that FEF neurons send a variety of task-related signals directly to the SC (Everling & Munõz, 2000; Seagraves & Goldberg, 1987; Sommer & Würtz, 2000). Seagraves and Goldberg (1987) first found that the majority of corticotectal FEF neurons were movement neurons and to a smaller extent foveal visual neurons. However, subsequent experimentation highlighted that FEF neurons also send visual and cognitive signals to the SC (Sommer & Würtz, 2000; Everling & Munõz, 2000). Recordings from the corticotectal FEF neurons while primates performed delayed and gap saccade tasks found that many corticotectal FEF neurons exhibited tonic delay activity and increases in activity during the gap period, in addition to visual and saccade-related activity (Sommer & Würtz, 2000). These results suggested that the FEF continuously influences the SC during oculomotor tasks.

Differences between FEF activity have also been found for different types of saccade. Everling & Munõz (2000) recorded from corticotectal FEF neurons while primates performed a randomly interleaved prosaccade and antisaccade trial task. The findings displayed that FEF neurons discharged for both types of saccade into their response field. The level of presaccadic activity and the motor burst were lower on antisaccade trials. Furthermore, many FEF neurons had higher levels of activity prior to the presentation of the peripheral stimulus in prosaccade than on

antisaccade trials. These differences in preparatory activity reflected different preparatory sets necessary to perform the two types of saccade (Evarts, Shinoda & Wise, 1984). Everling & Munõz (2000) proposed that the lower preparatory activity of saccade-related neurons and the reduced stimulus-related responses on antisaccade trials reduced the excitation of saccade-related neurons in the SC and therefore reduced the risk of generating a task-inappropriate saccade towards the stimulus (Everling, Dorris & Munõz, 1998b; Everling, Dorris, Klein & Munõz, 1999). The evidence highlighted displays the crucial role that the FEF plays in the generation of saccadic eye movements.

*1.3.3.2 Lateral Intraparietal Area (LIP)*

The lateral intraparietal (LIP) area receives converging inputs from numerous visual areas and sends projections to the two brain regions necessary for saccade production: the FEF and the intermediate layers of the SC (Johnston & Everling, 2006, 2009; Baizer, Ungerleider & Desimone, 1991). Many of these projection neurons have saccade related activity (Ferraina, Paré & Würtz, 2002; Paré & Würtz, 1997, 2001). Furthermore, there are reciprocal connections from the FEF (Ferraina et al., 2002; Lewis & van Essen, 2000b; Schall, Morel, King & Bullier, 1995a; Stanton et al., 1995) and the SC (Clower, West, Lynch & Strick, 2001). These connections previously led to the belief that area LIP was directly involved in the generation of saccadic eye movements (Lynch, Mountcastle, Talbot & Yin, 1977; Yin & Mountcastle, 1977). However, more recent evidence has shown that area LIP is not directly involved in the generation of saccadic eye movements. Firstly, the magnitude of LIP presaccade activity displays visual dependence and is significantly reduced when saccades are made in the absence of a visual stimulus (Ferraina et al., 2002; Pare & Würtz, 1997, 2001) as well as when several stimuli are present (Thomas & Pare, 2007). Furthermore, a large amount of electrical current is required to generate saccadic eye movements when stimulating area LIP (Keating, Gooley, Pratt & Kelsey, 1983; Kurylo & Skavenski, 1991; Shibutani, Sakata & Hyvarinen, 1984; Their & Andersen, 1998). This evidence contrasts with the demonstration of the critical role of the FEF and SC in the generation of saccadic eye movements. Additionally, the presence of presaccadic activity of a neuron cannot be used to conclude that this neuron is involved in the production of saccadic eye movements. For example, it has been shown that neurons in the striate and extrastriate cortex increase their activity prior to a saccade made to a stimulus presented in their visual receptive fields (Moore, 1999; Nakamura & Colby, 2000; Supèr, van der Togt, Spekreijse & Lamme, 2004). However this activity has been interpreted as

guiding saccades. The strong dependence of LIP presaccade activity on visual stimulation is consistent with this interpretation.

More recently the LIP has been implicated in visual saccadic decision making. Anatomically, the LIP area is ideally situated to integrate diverse sources of evidence that are involved in visual decision making and to send guiding signals to saccade generating centres such as the FEF and the SC. It has been argued that area LIP provides a map where evidence supporting the saliency of competing visual items accumulates. Decisional processes can then assist visual attention, visual working memory, saccade preparation and saccade execution if required (Ludwig, Gilchrist, McSorley & Baddeley, 2005). Britten, Shadlen, Newsome and Movshon (1992) used a motion discrimination task where primates viewed a random dot kinetogram in which a minority of the dots moves in a coherent direction amongst the remaining dots moving randomly. Primates were required to indicate the overall perceived direction of the motion by eliciting a saccade to one of two peripheral saccade targets. The location, size, speed and direction of the random dot kinetogram were optimised to best activate the specific LIP neuron under study. Results demonstrated that LIP neuronal activity accumulates for preferred direction motion, the rate of which depends on the quality of the sensory evidence (motion coherence) (Shadlen & Newsome, 1996, 2001). These LIP properties are consistent with bounded accumulator models of simple decision making, which provide a mechanism for integrating incoming sensory information over time.

The LIP has also been found to be crucial in the encoding of economic variables that are learned through experience. Previous experimentation has revealed that LIP neuronal activity is influenced by the probability of a saccade target yielding a reward, the magnitude of reward associated with that option and the degree of confidence in the decision (Churchland, Kiani & Shadlen, 2008; Dorris & Glimcher, 2004; Kiani & Shadlen, 2009; Platt & Glimcher, 1999; Rorie, Gao, McClelland & Newsome, 2010; Yang & Shadlen, 2007). Unlike the FEF and the SC, economic information is not represented in baseline LIP activity but is only revealed immediately after presentation of a target (Basso & Würtz, 1998; Dorris & Munõz, 1998; Ikeda & Hikosaka, 2003; Roesch & Olson, 2003). This finding suggests that the LIP area is not where economic variables are stored, but instead that representations in area LIP are modulated by their potential economic impact from external sources. More recently, it has been suggested that activity in LIP is a function of relative expected value. For example, the expected value of a neuron's preferred target, divided by the sum of the expected values for the other potential visual targets equates to the activity of that neuron (Dorris & Glimcher, 2004; Platt & Glimcher, 1999; Rorie et al., 2010).

This explanation of the normalisation of value across the LIP area allows many multiple potential options to be represented simultaneously and compared across a wide range of values. These decision processes are strongly influenced by the expected timing of environmental events. For example, LIP neurons adjust their activity to reflect whether the duration of sensory events are shorter or longer than a standard time (Leon & Shadlen, 2003). LIP activity can also represent sophisticated probabilistic time distributions of when salient events are likely to occur (Jansen & Shadlen, 2005). Such timing signals are potentially important for initiating voluntary actions especially those constrained by strict deadlines for selection (Churchland et al., 2008; Maimon & Assad, 2006). For the LIP area to be considered to be involved in the decision process its activity must not only be influenced by sensory, economic and timing evidence but also predict the choices that the subjects ultimately make. Increasing LIP activity using electrical micro-stimulation manipulates perceptual decision formation which has been evidenced by decreases in the latency and increases in the proportion of choices in favour of the option associated with the site of stimulation (Hanks, Ditterich & Shadlen, 2006). Similarly, when saccadic choices are based on more economic considerations, LIP activity is influenced by the relative value of the options but also predicts the overall allocation of choices (Coe, Tomihara, Matsuzawa & Hikosaka, 2002; Dorris & Glimcher, 2004; Seo, Barraclough & Lee, 2009; Sugrue, Corrado & Newsome, 2004).

In summary, the evidence provided in this section has highlighted the role that the LIP area plays in contributing to saccade decision processes based on incoming sensory evidence, economic variables and the expected timing of salient events. Therefore, this area is not only crucial for the role it plays in deciding where to saccade to but also its ability to process economic variables.

#### *1.3.3.3 Superior Colliculus (SC)*

As mentioned previously, the LIP sends projections to the SC. The SC forms two rostral bumps on the dorsal aspect of the midbrain. The caudal two bumps are the inferior colliculi. Together the inferior and superior colliculi form the tectum; the roof of the midbrain. The inferior colliculi have been categorised as predominantly an auditory structure (Huffman & Henson, 1990; Casseday, Fremouw & Covey, 2002), whereas the SC is described as purely a visual reflex centre (May, 2005). The SC is defined by a highly laminated structure consisting of seven anatomically distinct layers, grouped into two functional regions: 1) the superficial region (SCs) concerned exclusively with visual processing (Goldberg & Würtz, 1972a); 2) a deeper intermediate region (SCi) concerned with multisensory (Meredith & Stein, 1983, 1985; Stein & Meredith, 1993), motor

(Robinson, 1972; Sparks, 1978; Würtz & Goldberg, 1971) and higher level cognitive processes such as attention (Muller, Philiastides & Newsome, 2005). The superficial layers consist of the three dorsal most laminae; the stratum zonale (SZ) the stratum griseum superficale (SGS) and the stratum opticum (SO). The deeper layers refer to the remaining four lower layers; the stratum griseum intermediale (SGI), the stratum album intermediale (SAI), the stratum griseum profundum (SGP) and the stratum album profundum (SAP).

The SCi receives its projections from a broad range of corticotectal structures encompassing the frontal, parietal, temporal and occipital cortices (Cusick, 1988; Fries, 1984; Kunzle & Akert, 1977; Lock, Baizer & Bender, 2003). These include the LIP (Lynch, Graybiel & Lobeck, 1985), the FEF (Stanton et al., 1995; Stanton et al., 1988), the supplementary eye fields (SEF) (Shook, Schlag-Rey & Schlag, 1990), dorsolateral prefrontal cortex (DLPFC) (Goldman & Nauta, 1976) and the anterior cingulate cortex (ACC) (Leichnetz, Spencer, Hardy & Astruc, 1981). The LIP-SCi projection carries both visual and motor-related information (Paré & Würtz, 1997, 2001) which is critical for the flexible control of oculomotor behaviour (Everling & Munõz, 2000; Hanes & Würtz, 2001). A broad range of response properties have been attributed to the neurons within this layer of the SC due to the vast range of projections to and from this layer. Visuomotor neurons within the SCi discharge a burst of action potentials 50ms after the appearance of a visual stimulus in the neuron's response field and a separate burst of action potentials associated with the occurrence of a saccade (Mohler & Würtz, 1976). Close spatial correspondence between the visual and motor response fields of the neurons exists within this layer (Marino, Rodgers, Levy & Munõz, 2008), ensuring that a visual response is mapped directly onto the appropriate output neurons projecting to the brainstem premotor circuitry to trigger a saccade (Rodgers, Munõz, Scott & Paré, 2006) and for orienting head movement (Corneil, Munõz & Olivier, 2007; Corneil, Olivier & Munõz, 2002, 2004) to the visual stimulus. The most distinctive characteristic of many neurons within this layer is to initiate the build-up activity that precedes a saccade (Glimcher & Sparks, 1992; Munõz & Würtz, 1995a). This low frequency activity can begin well in advance of the movement itself and is associated with motor preparation (Corneil et al., 2007; Dorris, Paré & Munõz, 1997; Dorris & Munõz, 1998; Li & Basso, 2008; Munõz & Würtz, 1995a), as well as various high-level processes, such as covert shifts of attention (Ignaschenkova, Dicke, Haarmeier & Thier, 2004; Kustov & Robinson, 1996), expectation (Basso & Würtz, 1997, 1998; Thevarajah, Mikulic, & Dorris, 2009) and target selection (Basso & Würtz, 1997, 1998; Glimcher & Sparks, 1992; Horwitz & Newsome, 1999, 2001; McPeek & Keller, 2002).

Conversely, the SCs receives visual inputs from two primary sources: 1) a direct projection from the retina (retinotectal pathway) (Cowey & Perry, 1980; Hubel, LeVay & Wiesel, 1975; Pollack & Hickey, 1979); 2) direct projections from the visual cortex, specifically the primary visual cortex, V2, V3, V4 and the middle temporal area (MT) (Cusick, 1988; Fries, 1984; Graham, 1982; Tigges & Tigges, 1981). In turn these project through the pulvinar nucleus of the thalamus to a broad area of the cerebral cortex. The neurons within this layer have been categorised as exclusively visual, eliciting short, high frequency bursts of action potentials as early as 40ms following the appearance of a visual stimulus in their response field (Cynader & Berman, 1972; Goldberg & Würtz, 1972a; Schiller & Koerner, 1971). Other visual neurons found deeper within this layer are categorised as quasi-visual (Mays & Sparks, 1980) and tonic visual neurons (Li & Basso, 2008; McPeek & Keller, 2002; White et al., 2009). These neurons exhibit an initial transient burst of action potentials followed by a lower frequency sustained firing pattern while a stimulus is present in the neurons' response field. It is unclear whether these neurons belong to the lower region of the SCs or the upper region of the SCi but they are typically located above neurons with saccade related activity (Li & Basso, 2008; Mays & Sparks, 1980; McPeek & Keller, 2002). Neurons within this layer are highly sensitive to stimulus intensity (Bell, Meredith, Van Opstal & Munõz, 2006; Li & Basso, 2008), but display little preference for specific visual features or colours (Marrocco & Li, 1977; Schiller & Malpeli, 1977).

It is widely accepted that neurons within the SC are organised into well-defined topographic maps whereby each colliculus contains multisensory (Groh & Sparks, 1996; Jay & Sparks, 1987; Meredith & Stein, 1983, 1985; Stein & Meredith, 1993) and motor (Robinson, 1972; Sparks, 1978; Würtz & Goldberg, 1971) representations of contralateral space. The SCs contains a visual map such that a given neuron at a specific location on the map responds to stimuli presented in a restricted region of the contralateral visual field, which defines a receptive field (Cynader & Berman, 1972). The SCs has been described as a salience map due to projections in this structure from several areas (Fectau & Munõz, 2006). The SCs receives direct projections from visual cortical areas V1, V2, V3 and MT (Fries, 1984; Lock et al., 2003; Tigges & Tigges, 1981) and the projection from earlier to later areas is represented by increasing depth in the SCs layers. The SCs has substantial projections to the pulvinar which then projects to multiple extrastriate visual areas. It has been suggested that a structure coding salience should have extensive feedback to higher levels of visual processing and this has been evidenced by these projections in the SCs.

In contrast to the SCs, the neurons of the SCi reflect the relative importance of a stimulus for the goal of the observer (Fectau & Munõz, 2006); a representation termed priority (Serences & Yantis,

2006, 2007). Based on this evidence the SCi has been functionally described as a priority map (Fectau & Munõz, 2006). Extensive literature has shown that neurons within the SCi have discharges correlating with both exogenous and endogenous shifts of visuospatial attention (Bell et al., 2004; Dorris et al., 2002, 2007; Fectau & Munõz, 2005, 2006; Fecteau, Au, Armstrong, & Munõz, 2004; Gattass & Desimone, 1996; Ignashchenkova et al., 2004; Kustov & Robinson, 1996; Lovejoy & Krauzlis, 2010; Lovejoy, Fowler & Krauzlis, 2009; Muller et al., 2005; Robinson & Kertzman, 1995). Visuomotor neurons within the SCi show enhanced activity during an endogenous shift of attention into their response fields, even in the absence of a visual stimulus (Ignashchenkova et al, 2004). Microstimulation of the neurons within this layer can facilitate visual discrimination performance at the spatially selective location represented by the stimulated site, which is indicative of a covert shift of visual attention (Muller et al., 2005). Furthermore, inactivation of a selective region of the SC caused primates to ignore critical spatial cues that the SC may act as a bottleneck for covert attention (Lovejoy & Krauzlis, 2010). Thus the SCi is modulated by covert shifts of attention, independently of eye movements.

The layers of the SC are not only linked in the processing of visual information but have also been implicated in the encoding and processing of reward. As the SCi receives direct inputs from brain areas that encode reward information, including the PFC and the BG (Ikeda & Hikosaka, 2003), reward responses have been reflected in the activity of neurons within the SCi specifically. When a visual stimulus signals an upcoming reward, both visual and preparatory activity of SCi neurons is enhanced (Ikeda & Hikosaka, 2003). The neurons encoding this enhanced signal have been found to be the build-up neurons, described in detail in a later section (Glimcher & Sparks, 1992; Munõz & Würtz, 1995a). The activity of SCi neurons is modulated by prior expectation that a target will appear in its response field with enhanced activity if the probability is high and suppressed if the probability is low (Basso & Würtz, 1997, 1998; Dorris & Munõz, 1998; Glimcher & Sparks, 1992).

There is evidence however that the SC itself plays an active role in encoding reward information during reinforcement learning via its projection to the SNr. Comoli et al., (2003) demonstrated a previously unreported direct anatomical projection between the SC and the SNr and established that the SC is critical for short-latency visual activation of dopamine-containing regions of the ventral mid-brain. These findings established the retino-tecto-nigral circuit as the most likely source of short latency visual input to the ventral midbrain. This projection carries transient visual activity to the BG dopaminergic system, which is critical for reinforcing the context or actions that immediately precede unpredictable, biologically relevant visual events (Dommett et al., 2005;

Redgrave & Gurney, 2006). Furthermore, sub-threshold stimulation of the SCi can bias choice predictability towards the stimulated site of two equally rewarded stimulus locations, implicating the SCi as an important part of the circuit that actively chooses strategic actions that produce positive rewards (Thevarajah et al., 2009). The layers of the SC are critical for the control and generation of saccadic eye movements but also play a key role in the encoding and processing of reward due to this structure's links with the BG.

*1.3.3.4 Basal Ganglia (BG)*

The BG are a cluster of neurons located at the base of the forebrain, and are strongly associated with purposive motor control, evidenced by the number of movement disorders associated with damage to this area (DeLong & Georgopoulous, 1979). One such structure located within the BG (the SNr) influences oculomotor behaviour through the use of an inhibitory loop involving the SC (Jayaraman, Batton & Carpenter, 1977; Graybiel, 1978; Chevalier, Vacher & Deniau, 1984; May & Hall, 1984). When resting the SNr neurons actively inhibit the SC (Hikosaka & Würtz, 1983). The SNr is under the control of the CN, another structure located within the BG. When a saccade is generated the cortical activity excites caudate neurons which subsequently inhibit the neurons in the SNr (Hikosaka, Sakamoto & Usui, 1989). The SC neurons are subsequently disinhibited triggering downstream premotor activity to drive the appropriate orienting response (Hikosaka & Würtz, 1989). The generation of saccadic eye movements is an inhibitory process with these structures outlined above communicating with each other to generate saccades to an appropriate or designated location.

The BG have also been implicated in the generation of reward oriented behaviour in receiving substantial reward information and subsequently influencing body movements, including saccadic eye movements. Firstly, the motor function of the BG is achieved by output projections of the BG to the brainstem motor areas, such as the SC (Grillner et al., 2005; Takakusaki, Saitoh, Harada & Kashiwayanagi, 2004) and movement related areas in the cerebral cortex through the thalamus (Parent & Hazrati, 1995). Secondly, the reward related information to the BG is derived from inputs from the limbic system to the ventral striatum (Haber & McFarland, 1999; Mogenson, Jones & Yim, 1980) dorsal striatum, such as the caudate nucleus (CN) and the putamen, (Ragsdale & Graybiel, 1988; Uno & Ozawa, 1991) and to dopamine neurons located in the SNr (Fudge & Haber, 2000). It is these dopamine neurons which have been found to carry an essential signal for reward-based learning and project most heavily within the BG (Schultz, 1998). Thirdly, the

inhibitory connections within the BG have been found to be suitable for the selection and learning of optimal behaviour (Hikosaka, Sakamoto & Miyashita, 1993; Mink, 1996). The BG are also thought to be involved in learning sensorimotor procedures and habit formation (Graybiel, 1998; Packard & Knowlton, 2002; Salmon & Butters, 1995). Furthermore, sensorimotor-cognitive signals originating from the cerebral cortex pass through the BG before returning to the cerebral cortex. Therefore, the BG is perfectly placed to control motor behaviours based on reward information.

The relationship between the BG and reward is the product of the ventral striatum and nucleus accumbens inputs from the limbic system (Fudge et al., 2002) and the orbitofrontal cortex (Selemon & Goldman-Rakic, 1985). Lesion studies within the striatum have found deficits in a variety of tasks where reward-predictive cues would usually guide participants' responses (Everitt, Morris, O'Brien & Robbins, 1991; Everitt et al., 1999; Kelley, 2004). The reward related activity is not limited to the ventral striatum, with primate studies revealing the impact of reward to be almost equally as strong in the dorsal striatum. Activity of neurons in the dorsal as well as the ventral striatum is strongly influenced by the effects of appetitive rewards such as food and water (Hollerman, Tremblay & Schultz, 1998). Neurons in the CN and putamen show sustained activity prior to delivery of an expected reward (Hikosaka et al., 1989), similarly to neurons in the ventral striatum (Schultz, 2000). Also found across both the dorsal and ventral striatum are other neurons that respond differentially to sensory stimuli indicating the presence or absence of an upcoming reward (Hollerman et al., 1998; Kawagoe et al., 1998). Such anticipatory and sensory responses are related to the amount of expected reward (Cromwell & Schultz, 2003) or the temporal proximity of reward delivery (Bowman, Aigner & Richmond, 1996). Imaging studies have advanced investigation of these effects in the human brain, typically using monetary rewards finding increased activation of the ventral (Elliott, Friston & Dolan, 2000; Knutson, Adams, Fong & Hommer, 2001; Ullsperger & von Cramon, 2003) and dorsal striatums (Delgado et al., 2000; O'Doherty, Deichmann, Critchley & Dolan, 2002) when a greater magnitude of reward was expected. It is clear from the evidence outlined above that reward related processes are not exclusive to just the ventral striatum but instead are present across the entirety of the striatum. It may be simpler to suggest that the dorsal and ventral striatums have different functions in relation to reward processing. The neurons within the dorsal striatum display sensorimotor or cognitive activities which are modulated by the nature of the expected reward (Cromwell & Schultz, 2003; Kawagoe et al., 1998; Watanabe, Lauwereyns, & Hikosaka, 2003). On the other hand, neurons in the ventral striatum are less selective to sensorimotor events (Schultz, Apicella, Scarnati, & Ljungberg, 1992) and instead activity within these neurons tends to occur prior to the delivery of reward (Hollerman et al., 1998). Therefore, the dorsal striatum, rather than the ventral

striatum, is a place where reward related information is integrated into specific sensorimotor and cognitive information (O'Doherty et al., 2004).

Within the dorsal striatum is the CN. The CN is involved in the generation of saccadic eye movements through its inhibitory projection to the SNr, as outlined previously. However, the CN neurons are also extremely responsive to reward magnitude with greater rewards resulting in a greater level of activity within the CN (Lauwereyns et al., 2002). Due to the inhibitory connection between the CN and the SNr, the increase in CN activity would manifest itself as increased oculomotor readiness at the collicular level. It has been hypothesised that the reward modulation of CN neurons is shaped through dopaminergic inputs into the CN that modulate the synaptic efficacy of the cortical inputs (Hikosaka, Nakamura, & Nakahara, 2006). These dopamine neurons encode a quantity related to the difference between predicted and obtained rewards, whereby a larger obtained reward than expected results in an increased response and a smaller than expected reward results in suppression of a response (Hollerman & Schultz, 1998). As a result these neurons may be regarded as computing a prediction error term that enables learning of the reward structure of any given environment (Nakahara et al., 2004; Schultz, 1998).

It is clear from the evidence outlined above that the BG is a crucial structure in both motor function and reward with its projections to the SC, the SNr and both the dorsal and ventral striatums. The BG is also linked with reinforcement learning due to its connections with the CN, a structure involved in computing prediction error. Therefore, this structure is crucial when considering the aims of the thesis.

#### *1.3.3.5 Brainstem*

A sophisticated control system in the brainstem exists which allows saccadic eye movements to occur, sending coded signals to the oculomotor muscles resulting in a pattern of muscle excitation, consisting of high frequency bursts of activity which serve to reposition the eye. This is followed by tonic activity which keeps the eye in its new position. The extraocular muscles synergistically act to control eye movements. These muscle pairs are innervated by motor neurons (MN) located in the brainstem (Leigh & Zee, 1991). During saccadic eye movements, MNs exhibit a step pulse pattern of discharge; there are bursts of action potentials for the on direction for saccades (the pulse) followed by pauses in the activation for the off direction for saccades. Additionally there is a tonic component of the discharge (the step) following a saccade which keeps the eye in its eccentric orbital position. The types of cell involved in this sequence include

excitatory (EBN) and inhibitory (IBN) burst neurons, which discharge bursts of action potentials for the on direction for saccades, and which are silent during fixations. Omnipause neurons (OPN) pause for saccades in all directions and discharge tonically during fixations. Long-lead burst neurons (LLBN) project to the EBN and IBN to provide the burst input. These neurons have a low frequency build up before the burst and discharge a high frequency burst for saccades directed to the opposite hemifield. The generation of saccadic eye movement therefore requires that the OPN becomes silent whilst the LLBN produces the necessary amount of activity for the EBN and IBN to send a saccade command to the MN. Following the saccade the OPN become tonically active again which inhibits any activity in the EBN or IBN from disrupting fixation. This happens when a saccade is produced.

This section has highlighted the complexity of the eye movement system, with a number of structures communicating with each other to generate saccades. Although the system is complex it is extremely well understood. Furthermore, there is substantial overlap between the structures of the brain involved in the generation of saccadic eye movements and the areas of the brain innervated with dopamine neurons or involved in reward encoding and processing.

### 1.4 Models of Oculomotor Control

In everyday life we make around three saccadic eye movements per second (Rayner, 1998; Becker, 1991). These eye movements are the fastest movement the human body can make and are essential to our interaction with the world, allowing us to attend to stimuli of interest whilst ignoring those that are not of relevance, in a dynamic fashion. We are rarely conscious of these movements and they appear to involve minimal cognitive effort. Previous studies in both primates (Bendiksby & Platt, 2006; Takikawa et al., 2002; Kawagoe et al., 1998) and humans (Milstein & Dorris, 2007) have shown that rewards speed these eye movements. These findings are consistent with accumulator models of oculomotor control. Within this section, two of the most widely accepted and influential accumulator models of saccade generation will be described and a final model that accounts for the generation of eye movements in dynamic natural environments. The first model, proposed by Findlay and Walker (1999), posits two separate processing systems; one for the ‘where’ and ‘when’ pathways of saccade generation. The second model proposes two separate systems sharing the same saccade map, with the possible site of this model recognised as the SCi (Godijn & Theeuwes, 2002; Trappenberg, Dorris, Munõz & Klein, 2001). The final model

proposed by Hayhoe & Ballard (2005) takes into account vision in natural scenarios and the role of fixations in providing task-relevant information that is rewarding.

Findlay and Walker (1999) outline a model of saccade generation based on the principles of parallel processing of saccade timing and metrics in two clear streams and a competitive inhibition through the use of a 'winner takes all' strategy accounting for a variety of oculomotor phenomena. The model distinguishes between a spatial 'where' system and a temporal 'when' system of eye movement control, with saccade generation occurring as a result of competition between these two clear processing streams. 'When' to move the eyes is determined by high level cognitive processes related to the processing of foveal information. 'Where' to move the eyes is determined by low-level visual analysis of peripheral stimuli. In the 'where' pathway, spatially distributed coding and selection of a saccade target is achieved through parallel processing and competitive inhibition within a 2D salience map. The saccade metrics are a direct result of the location of a peak within the salience map. The release of a saccade is determined by conflict resolution between the 'when' pathways 'fixate' centre and the 'where' pathways 'move' centre. This process of conflict resolution described by Findlay and Walker (1999) is time consuming and accounts for the time taken to initiate a saccade. This model is in direct correspondence with previous studies showing that rewards speed eye movements. Based on this model, rewards result in a larger peak on the salience map and subsequently faster release of the saccadic eye movement speeding eye movements to rewarded locations.

More recently, the emphasis of models of saccade generation has been on the competitive integration of endogenous and exogenous saccades occurring in a single saccade map, contrary to Findlay and Walker's (1999) dual stream theory. Trappenberg et al., (2001) presented an idea based on a model by Kopecz (1995) showing that SRTs can be modelled by a mechanism whereby exogenous and endogenous visual signals converge within a dynamic integration layer employing lateral interactions characterised by short-distance excitation and long-distance inhibition. Trappenberg et al., (2001) enhanced this model by placing this structure in the SCi. As previously outlined, the SC is a critical structure as it seems to be a converging point for cortical and subcortical inputs involved in sensory, motor and attentional processing. The model itself is designed to account for how neuronal activity in the SC can produce saccadic behaviour in a range of situations involving the interaction of endogenous and exogenous situations. The model is structured on the neurons in the SCi. The critical feature of the model is the interaction structure within the SCi.

A set of three experiments by Godijn and Theeuwes (2002) support this theory for converging exogenous and endogenous signals in a single saccade map. In the first experiment participants were required to saccade to a target, whilst an abrupt task-irrelevant onset distractor was presented at the same time as the target was defined on some trials. In the second version of the experiment, whilst participants made the first saccade, the position of the target was switched. Finally, the third experiment included a second target switch, in which the target position was repositioned in its original starting position. Stimuli were presented in an around the clock display with possible target locations at 1, 3, 5, 7, 9 and 11 o'clock positions. Participants were required to saccade to a target circle, which changed colour from red to grey 600ms after onset of the display. On some trials a red circle was presented in an empty space on the display which coincided with the colour change of the target from red to grey. These experiments allowed analysis of whether voluntary and involuntary eye movements, and the spatial and temporal aspects of these two types of eye movement, were programmed together in the same system prior to execution of a saccade. The results from these three experiments provided evidence for a competitive integration model, whereby exogenous and endogenous saccades were programmed in the same saccade map. The experiments manipulated the appearance of abrupt onsets with targets in a visual search paradigm finding reduced saccade onset latencies when saccades were directed to the abrupt onset prior to the signalled saccade target.

In Godijn and Theeuwes (2002) model, the competition for saccade programming results from activation at different locations in the saccade map. Although the model is similar to Findlay and Walker's (1999) model, in that it assumes competitive integration of information, it is different in that it does not separate the temporal and spatial aspects of oculomotor control. Godijn and Theeuwes (2002) model supports a competitive integration of spatial and temporal signals rather than an independent signal model. The model assumes that control signals for voluntary and involuntary eye movements converge on a shared saccade map. There is a retinotopic representation whereby the information for both saccade types is integrated. Activation within this map is inhibited for distant locations but spreads to close locations. Lateral inhibition occurs when two distant locations are activated, but when two near locations are activated the resulting combined activation often results in a peak somewhere between the two locations. This competitive integration model is able to account for many oculomotor effects consistently observed in eye movement tasks and can provide an explanation for the outcome when paired saccades are programmed, something Findlay and Walker's (1999) model fails to account for.

The models outlined have one major difference in that Findlay and Walker (1999) advocate separate pathways for the 'when' and 'where' pathways of eye movement control. However, the other models postulate that both the 'when' and 'where' pathways are programmed in the same saccade map, the root of which has been recognised as the SCi. Although there is debate as to whether these two aspects of eye movement programming are separable, it has been recognised that decisions of 'where' and 'when' to have the point of fixation are key aspects of eye movements' controls; understanding the relationship between the two is crucial to fully map the cognitive processes that eye movements reflect (Liversedge & Findlay, 2000).

A final model of oculomotor control with relevance to the present thesis is a model proposed by Hayhoe and Ballard (2005). This model is different form the two previously outlined as it addresses the issue of completing multiple tasks in everyday life by assigning value to different tasks. Reinforcement learning has a central difficulty in that it fails to apply to realistic natural behaviours. However, by factoring complex behaviours into subsets of tasks served by modules that can operate independently, this issue can be addressed and simplified. Each of these modules, defined as a Markov decision process (Bellman, 1957), computes a reward-weighted action recommendation for all the points within its own state space, which is the set of values the process can take. As these modules are all embedded within a single agent, the action space is shared among all modules and the best action is chosen depending on the relative reward weights of the modules. The modules provide separate representations for the information needed by individual tasks and their actions influence state transitions and rewards individually and independently. This modular approach allows fixation choices to be understood in terms of competing modules demands for reward. For example, in the everyday scenario of driving a car, where separate modules address subtasks such as avoiding other cars and staying in lane, specific information is gathered from the visual scene to support the actions required for these tasks. During dynamic everyday scenes a subject acquires a particular piece information for a module, takes an action and then decides which module should get gaze next. When a module is updated with information from gaze, the new sensory information reduces uncertainty about the state of the environment relevant to that module. The next action is chose on the basis of mapping from states to actions, which may be learnt through reinforcement. As a consequence of the action the state of the world is changed and the agent must decide which modules state should nest be updated with gaze. The assumption is that fixation is a serial process where one visual task accesses new information at each time-step and all other tasks must rely on noisy memory estimates. This model provides a more applicable use of fixations and eye movements into natural

everyday scenarios. In decomposing tasks into decision modules which are updated due to task-demands, this model can account for the dynamics if fixations in multiplexed situations.

### 1.5 Saccade Latency

It is argued that the oculomotor system provides a microcosm of the brain (Carpenter, 1994) where sensory input can be precisely controlled and manipulated, and limited motor output can be measured with exceptional accuracy with eye tracking equipment. The eyes have a simple and well defined repertoire of movements and the neural circuitry regulating the production of saccadic eye movements is now understood at a level that is sufficient to link cortical and subcortical areas together. For these reasons the latency of saccadic eye movements is a reliable way of measuring the level of activation in the oculomotor system and linking the effects found with precise regions or structures in the brain.

Saccade latency is described as the time taken to initiate a saccade and is calculated as the duration between the event that is being responded to (such as the peripheral flash or appearance of a visual target) and the onset of the movement. One of the key characteristics of the latency of saccadic eye movements is their extreme variability, with the average saccade latency substantially affected by a number of factors. Latencies can range from as little as 100ms to as much as 1000ms. Even if the stimulus is constant, saccade latency is variable on a trial-by-trial basis. This is an important point to consider when creating a reward paradigm able to consistently alter the eye movement behaviour of sufferers of visual field deficits. Using a paradigm with too many stimuli, whether to use a fixation cross or whether the effects will generalise to different types of saccade will all need to be considered when designing the present set of experiments in order to build a consistent and optimal model of behaviour change for rehabilitation. Furthermore, the pre-existing oculomotor paradigms created in order to test the variable effects of saccade latency and oculomotor behaviour can be used to test the persistence and transfer of any effects of reward found when using a reward paradigm. Therefore, the following section will outline the factors which can affect the latency of saccadic eye movements and the pre-existing paradigms that have investigated competition between and inhibition of saccadic eye movements.

### 1.5.1 Stimulus Properties

The properties of the stimuli that participants are generating saccades to can have a profound effect on the latency of saccadic eye movements. Kalesnykas and Hallett (1994) investigated the influence of eccentricity on saccade latency over a wide range of target eccentricities. Target eccentricity is defined as the distance from the current fixation point to the target. Kalesnykas and Hallett (1994) observed that the size of a saccade to a target had minimal or no effect on saccade latency. Instead the properties of the stimulus had a larger effect on saccade latency than target eccentricity. Saccadic eye movements were found to be slower to a target with decreased intensity. Further investigation has also found saccadic eye movements are slower to targets of decreased contrast and increased spatial frequency (Ludwig, Gilchrist & McSorley, 2004). Therefore the properties of a specific stimulus can drastically alter the speed at which participants generate eye movements.

### 1.5.2 Number of Stimuli

In everyday life saccadic eye movements are made in the context of multiple stimuli. Multiple potential saccade plans are thought to compete with each other, racing to reach threshold and trigger a saccade (Leach & Carpenter, 2001; Theeuwes, Kramer, Hahn & Irwin, 1998). However, an increase in the number of potential targets does not relate to faster saccades. Instead, increasing the number of potential targets results in the slowing of saccadic eye movements. This has been explained in terms of mutual inhibition between competing saccade plans requiring a longer period of time for saccades to become activated in the context of competing saccade plans (Findlay & Walker, 1999; Godijn & Theeuwes, 2002; Kopecz, 1995; Leach & Carpenter, 2001; Trappenberg et al., 2001). Therefore the number of stimuli in a visual presentation can have a profound effect on the latency of participants' saccades.

The remote distractor effect (RDE) is a well-established phenomenon in which saccades are delayed when an irrelevant stimulus appears elsewhere in the visual field (Bompas & Sumner, 2009; Honda, 2005; Lévy-Schoen, 1969; Ludwig, Gilchrist, McSorley & Baddeley, 2005; Walker, Deubel, Schneider & Findlay, 1997). The RDE is automatic, occurring even when the direction of the saccade target is known in advance and distractors appear in the opposite hemifield where they should be easy to ignore (Benson, 2008; Walker, Kentridge & Findlay, 1995; Walker, Mannan, Maurer, Pambakian & Kennard, 2000). Reliable increases of saccadic latencies have been found when distractors are presented simultaneously with, or within 40ms after the target presentation

have been found, with these effects diminishing after this time (Walker et al., 1995). Presenting distractors in the contralateral hemifield to a target has also produced results consistent with presenting distractors bilaterally and simultaneously, increasing saccadic latencies by 20-30ms in a number of published experiments (Walker et al., 1995; Weber & Fischer, 1994).

Recently, understanding of the RDE has increased with revelations regarding the underpinnings of the effect. Reingold and Stampe (2002) suggested that saccadic inhibition is responsible for the slowed saccadic reaction times (SRTs) found in the remote distractor task. Although the RDE is usually elicited by a small localised change in the visual scene, whereas the saccadic inhibition is initiated using a large flash, it is believed that common mechanisms of influence underlie these phenomenon. The "saccadic inhibition hypothesis" (SIH) implies that the key factor governing the RDE is not the temporal relationship between target and distractor, but instead the relationship between the distractor and the planned saccade. Reingold and Stampe (2002) state that if saccadic inhibition is accountable for the slowing of SRTs in response to the remote distractor task, then this effect is dependent on two factors: the latency between target onset and distractor onset; and baseline SRTs when a distractor is not present. Previous research has found differences in the RDE at a number of different target-distractor asynchronies. For example, Ross and Ross (1980) found a distractor effect when the distractor followed the target by 100ms, whereas Walker et al. (1995) found their maximum effect when the target and distractor were presented simultaneously. Although the intricacies of the remote distractor task are still debated, the pervasive finding is that saccadic responses to a target are slowed by the presence of a task-irrelevant stimulus, and this finding has been linked to the misallocation of attention to the location of this distractor (Hickey, McDonald & Theeuwes, 2006).

The increase in latency observed in the remote distractor task has been linked to inhibitory processes known to operate in structures tasked with saccade generation, such as the SCi (Walker, Deubel, Schneider & Findlay, 1997). Therefore, this area is identified as the likely locus of the neurophysiological basis behind these effects. The RDE has been evidenced to involve long-range lateral inhibition between cells within the SC which code for the saccades directed to target and distractor. Signals corresponding to different saccade endpoints (for example a saccade and a distractor) have been found to compete to reach threshold whilst also mutually inhibiting each other (Godijn & Theeuwes, 2002; Trappenberg et al., 2001). The presence of a distractor slows the rise of target activity, delaying saccades to the target location.

From the evidence outlined above it is clear that increasing the number of stimuli results in increased saccadic latency. Therefore to optimise a reward paradigm it is crucial to keep the visual

display as simple as possible to avoid increased SRTs. As such it is important to consider that increasing the number of stimuli could result in slower SRTs. Therefore when designing a reward paradigm using just a single target display should produce consistently fast SRTs with very small variability compared with using a number of different targets of varying values.

### 1.5.3 Presence of Fixation Point (Gap Effect)

The presence or absence of a fixation stimulus can also have an impact on the latency of eye movements. Stimuli at fixation inhibit saccade activation demonstrated by the gap paradigm (Reuter-Lorenz, Hughes & Fendrich, 1991; Saslow, 1967). In this paradigm, saccades are speeded when the fixation stimulus disappears 200ms prior to the appearance of the target stimulus (the gap condition), compared with the overlap condition, where the fixation stimulus does not disappear. Behavioural and neurophysiological studies have demonstrated that there are two components to this effect. Firstly, this speeding is accounted for by the warning signal given by the fixation offset resulting in disinhibition of the oculomotor system. Secondly, the presence of a fixation stimulus activates neurons in the rostral pole of the SC which subsequently inhibit the remainder of the saccade-generating SCi neurons (Dorris & Munõz, 1995; Munõz & Würtz, 1992, 1993a, 1993b). Therefore, the variability that arises from removal of a fixation point needs to be considered when designing a reward paradigm.

### 1.5.4 Goal-Directed or Stimulus-Driven Eye Movements

The antisaccade task is a tool used by psychologists to investigate the competition between reflexive (exogenous) and goal-directed (endogenous) eye movements. In this task a participant is required to inhibit the reflexive prosaccade towards a visual stimulus and instead make an eye movement away from this stimulus. This type of eye movement incurs a larger proportion of errors than with the reflexive prosaccade. Any eye movements made towards the peripheral cue are considered erroneous and as such are classified as a failure of inhibition. Latencies for this type of eye movement are typically 50-100ms slower than prosaccade latencies, representing the additional computations required for this type of eye movement in order to co-ordinate the transformation process (Evdokimidis, Liakopoulos, Constantinidis & Papageorgiou, 1996; McDowell et al., 1999). From this eye movement a number of measures can be analysed including: 1) the error rate, categorised as the number of trials on which the first saccade is generated towards the cue; 2) the latency, categorised as the time in milliseconds from the

appearance of the peripheral cue until the start of the saccade; 3) the accuracy for correct responses and error responses. It has been argued that the key component for this type of eye movement is disengagement from the current locus of fixation prior to the generation of a saccade (Everling, Dorris & Munõz, 1998; Funahashi, Chafee & Goldman-Rakic, 1993; Forbes & Klein, 1996; Schlag-Rey, Amador, Sanchez & Schlag, 1997). This account has often been referred to as the competition account of prosaccade versus antisaccade generation (Kristjánsson, 2007). The competition between pro and antisaccades has often been seen as a race between the two processes during the antisaccade task. When a prosaccade 'wins' an erroneous saccade is made to the peripheral cue. This type of task permits investigation of the de-coupling of the locus of attention from the direction of gaze and as such is used in this thesis to investigate the root of any effects of monetary rewards on human oculomotor behaviour.

### 1.5.5 Cueing

In a cueing paradigm a salient cue is presented to attract attention and perceptual performance is assessed either at the cued or un-cued location with the presentation of a target stimulus. The un-cued location refers to the location where the target is presented but the cue has previously not been presented. Such a paradigm involves three shifts of attention. Firstly attention is drawn to the location of the cue, then back to the central fixation point, then finally to the target stimulus. The robust finding associated with such a paradigm is that when the interval between cue and target presentation is short, performance at the cued location is facilitated (Klein, 2000; Posner & Cohen, 1984; Samuel & Kat, 2003). When longer time intervals occur between the cue and the target, the effect is reversed. This effect has been interpreted as a delay in the re-allocation of attention to the already attended location; a phenomenon termed the Inhibition of Return (IOR) (Klein, 2000; Klein & MacInnes, 1999). IOR is seen to promote efficient exploration of a visual scene, preventing an observer from returning to previously attended locations and instead promoting exploration of novel locations. This inhibition has also been found within saccadic responses (Rafal, Egly & Rhodes, 1994; Taylor & Klein, 2000; Vaughan, 1984) and it has been suggested that the mechanisms underlying IOR may be shared in Inhibition of Saccadic Return (ISR) (Hooge & Frens, 2000). ISR has been found to be a general phenomenon in oculomotor control having been found in visual search tasks (Gilchrist & Harvey, 2000; Hooge, Over, van Wezel & Frens, 2005), saccadic sequence studies (Hooge & Frens, 2000) and visually guided saccade tasks (Carpenter, 2001; Klein & McInnes, 1999). This type of task permits investigation of covert shifts of attention as the salient cue prior to target presentation induces shifts of attention.

As such, in the present set of experiments, this task is utilised to investigate the effects of reward on shifts of attention and oculomotor function.

This section has outlined the different factors that affect the variability of saccade latencies and the pre-existing paradigms used in eye movement research to tease apart oculomotor function and attention. It is important to take these factors into account when considering the aims of the thesis. In aiming to investigate the effects of monetary rewards on eye movements and whether a sustainable, persistent effect of reward can be generated, the factors that can affect saccadic latencies need to be taken into account. When building a reward paradigm and deciding what stimuli to use, the eccentricity, luminance and number of targets needs to be considered. Furthermore, the particular eye movement paradigms outlined above can be utilised to assess what is specifically affected if any effects of reward are found and whether the effects of rewards transfer to these already established eye movement tasks. Therefore, these factors need to be taken into consideration when designing a reward paradigm able to bias human eye movements.

### 1.6 Eye Movements and Attention

One important consideration in regards to the effects of reward on eye movements is the link between eye movements and spatial attention. The overlap between these two systems is somewhat controversial (see Smith & Schenk, 2012 for review). Some evidence suggests that the oculomotor system is involved in attention as the two systems share the same neural networks. Neuroimaging in humans has shown that preparing to move the eyes to a location activates the same network of frontal and parietal cortical regions as covertly attending (Beauchamp et al., 2001; Nobre et al., 2000; Perry & Zeki, 2000). Furthermore, transcranial magnetic stimulation (TMS) over the FEF disrupts the preparation of saccadic eye movements (Beckers et al., 1992; Muri, Hess & Meisenberg, 1991; Muri et al., 1996) and modulates spatial attention during arrow cueing (Smith, Jackson & Rorden, 2005, 2009). Although this data is often interpreted as saccade preparation and attention sharing the same neural regions, FEF data is problematic to interpret. The FEF contains multiple overlapping but independent neuronal populations, of which some are involved in visual selection but not motor control and vice versa (Sato & Schall, 2003; Thompson, Bichot & Schall, 1997; Thompson, Biscoe & Sato, 2005). Therefore, the FEF neurons that drive saccades are separate from those that drive visual selection. Moreover, TMS activates large neuronal populations, so studies showing that TMS over FEF modulates spatial attention (Grosbras & Paus, 2002; O'Shea, Muggleton, Cowey & Walsh, 2004; Smith et al., 2005) cannot be

clear that attentional modulation is driven by the specific activation of the motor system. Therefore, evidence from these studies illustrates that a tight coupling exists between attention and saccade planning but a causal link between the two cannot be demonstrated.

Saccadic attention can be deployed in a covert manner, such that the locus of attention is independent from the direction of gaze (Posner, 1980) seen in the aforementioned IOR task. These shifts of attention can be triggered in response to the goals and desires of the observer (endogenous) or reflexive in response to salient events (exogenous). Endogenous attention is generally slow to deploy and requires conscious cognitive effort but creates sustained enhancement at the attended location. Exogenous attention is rapid and automatic but generally short-lived and suspended by a sustained inhibition at the location of the salient event, known as IOR (Posner, Rafal, Choate & Vaughan, 1985). It has been theorised that these two different types of attention are governed by independent cognitive systems. Klein (1980) proposed that oculomotor preparation was required for endogenous spatial attention prior to conducting a dual task experiment investigating interactions between covert endogenous attention and oculomotor preparation. In one version of the task participants were required to plan a saccade to the left or right. On 70% of trials participants were given a 'go' signal to execute the movement. On 10% of trials a signal was given to execute an eye movement in the direction opposite to which they had prepared. On the final 20% of trials the go signal was withheld and instead a visual probe was presented which participants had to react to as fast as possible. This probe could appear at the saccade goal or the contralateral location. In the second version of the task participants were instructed to attend to either the left or the right. On 70% of trials a target was then presented at an attended location; on 10% of trials it was presented at the unattended location; on 20% of trials no target was presented and participants were instructed to saccade to the attended or unattended location. Klein (1980) argued that if saccade preparation is necessary to orient covert attention, responses should be faster at the goal of the planned saccade then the contralateral location. Secondly, saccades to attended locations should have shorter latencies than saccades to the unattended location. Conversely, Klein (1980) found no attentional facilitation when the probe overlapped with the saccade goal when the primary task was to generate a saccade. Secondly, when participants were required to attend, no SRT facilitation at the attended location was found. These results demonstrate independence of the oculomotor and endogenous attention systems and have been replicated on several occasions (Hunt & Kingstone, 2003b; Klein & Pontefract, 1994).

Further evidence of separate attention and oculomotor systems has been provided from patient studies. Patients with lesions to the FEF have issues with saccadic eye movements but intact covert endogenous attention (Henik, Rafal & Rhodes, 1994). One study has provided evidence that patients who are unable to execute eye movements exhibit deficits of endogenous attention (Craighero, Carta & Fadiga, 2001). However, many other publications consistently report preserved endogenous attention but disrupted exogenous attention in opthalamoplegic patients (Gabay, Henik & Gradstein, 2010; Rafal et al., 1998; Smith, Rorden & Jackson, 2004). Similarly, disruption of saccade preparation by eye-abduction produces a reliable deficit of exogenous attention (Smith & Schenk, 2010, 2012), whereas the effects on endogenous attention are small and unreliable (Craighero et al., 2001). Overall the evidence suggests that endogenous orienting of attention can occur independently of activation of the eye movement system.

## 1.7 Eye Movements and Reward

As previously explored, there is already substantial overlap between the areas of the brain involved in the generation of saccadic eye movements and the areas involved in the encoding and processing of reward (Gold, 2003; Ikeda & Hikosaka, 2003). This link has been evidenced experimentally, predominantly within a primate population, investigating the effects of reward magnitude and probability on saccade metrics. However, more recently human attention has been probed using money, resulting in a small number of experiments suggesting that monetary rewards can modulate spatial neglect. The following section will provide a review of the literature of the influences of rewards in non-human primates, humans and finally the current recent empirical evidence regarding the use of monetary rewards in rehabilitation.

### 1.7.1 Non-Human Primates

Although a number of studies have sought to explore the link between reward and the oculomotor system, the majority have investigated saccade metrics using a primate population. The focus of these studies has been on manipulations of reward magnitude and probability. Bendiksby and Platt (2006) used a peripherally cued saccade task to investigate whether the size of a reward would significantly alter saccade metrics in 2 male adult rhesus monkeys. The purpose of the study was to determine whether changes in reward size would systematically alter saccade metrics. The findings of this study illustrated how increasing the magnitude of rewards available reduced error rates on the task while reaction times decreased. Furthermore, tripling the size of

the reward further reduced SRTs. These findings illustrate that incentives increased attentiveness and motivation in primates, to perform quickly and accurately when a greater magnitude of reward is at stake, consistent with a number of other findings (Takikawa et al., 2002; Hikosaka et al., 2006; Chen, Hung, Quinet & Kosek, 2013). Moreover, Coe et al., (2002) created a 'free-choice task' where two identical stimuli were displayed. Primates were free to make a saccade to either target in order to obtain a reward. In the reward schedule employed primates were encouraged, but not instructed, to choose one target for several trials and then switch to the other target. The monkey was free to select either of the two targets. The findings revealed that primates switched between tasks after consecutive trials. Primates chose a target while reward increased, peaked and began to decrease before switching to the opposing target. Eventually, primates switched to another target, seeking out a greater reward. Furthermore, when presented with cues regarding how they were progressing towards earning a reward, rhesus monkeys' average reaction times and error rates both declined (Bowman et al., 1996). The ability to judge how far from receiving a reward they were and act accordingly displays the alterations that can occur to the saccade metrics and the behaviour of the individual in the pursuit of earning a reward.

Studies have shown a clear, well-established effect of rewarding saccades in non-human primates such that eye movements are faster and more accurate to rewarded locations and result in functional changes to the oculomotor system (Bendiksby & Platt 2006; Kawagoe et al. 1998; Takikawa et al. 2002). It therefore appears reasonable to assume that similar effects can be observed in humans. However, the large majority of the acquired knowledge about the mechanisms underlying visuospatial and visuomotor processing and reward is derived from electrophysiological recordings in macaques and other primates (Takikawa et al., 2002; Kawagoe et al, 1998; Watanabe et al., 2003; Bowman et al., 1996; Bendiksby & Platt, 2006; Sohn & Lee, 2006). Relatively few studies have explicitly focused on these processes in humans. Rather, the main focus of the studies in a human population has been the effect of rewards in relation to attention.

### 1.7.2 Human Population

Only a small number of studies have directly investigated the effects of rewards on human eye movements, with the majority focusing on these effects on human attention. Furthermore, studies that have investigated the effects of rewards and on the oculomotor system or attention have predominantly focussed on these effects in relation to the salience of stimulus and reward-

stimulus associations rather than the association between rewards and a particular location in space. The following section will outline the key publications in this field and evaluate their findings in relation to the aims of the present thesis.

Milstein and Dorris (2007) investigated the influence of relative expected value on reaction times in eleven human participants, by examining the relationship between choice and SRTs under conditions of changing value. Participants were required to make simple saccades to visual targets whose values were manipulated through changing probability and reward magnitude. The proportion of choices and SRTs were measured. The allocation of choices provided an established measure of participant preferences (Samuelson, 1938). This was compared with the latency with which participants responded during the same conditions. Participants were instructed to hold their gaze on a centrally placed fixation point for 800ms. Targets were presented to the left and right of fixation. Participants were required to make a saccade to a target and maintain fixation for 300ms. Two possible target locations were used and three different trial types: 1) two-target trials, where targets were displayed simultaneously (one left and one right) and participants were required to choose a target to saccade to. The purpose of this trial was to assess which of the two targets was preferred; 2) single target trials where only one target was presented on each trial with reward guaranteed if saccades were made. This trial type was used to assess how saccade preparation was allocated across the prospects of different magnitudes of rewards; 3) oculomotor capture trials where an irrelevant green distractor flashed for 70ms between fixation offset and target onset. This trial type probed the level of saccade preparation at specific locations in the visual field.

The authors conducted three experiments varying the proportion of the different types of trial. The findings of these experiments revealed that attention and preparation of saccades is strongly influenced by relative expected value under conditions of uncertainty. When allowed to choose between different rewards, participants opted for the higher expected value, consistent with previous primate research (Coe et al., 2002). The time to initiate saccades, the latency of the saccades, and the spatial allocation of oculomotor captures were all influenced by the expected values, such that expectation of a greater reward resulted in decreased saccadic latency, a shorter initiation time and greater oculomotor capture of the eyes, consistent with primate research (Bendiksby & Platt 2006; Kawagoe et al. 1998; Takikawa et al. 2002; Bowman, Aigner & Richmond, 1996)**.** These findings suggest a weighted combination of probability and reward magnitude influence saccade generation rather than either factor alone. The magnitude of reward exerted a stronger effect than reward probability in influencing choice, revealing that reward magnitude

dominates reward probability across a wide range of saccade target values. This finding further illustrates the importance of reward magnitude when designing a reward paradigm capable of oculomotor behaviour change.

The majority of studies in humans have probed the effects of reward with a specific focus on how rewards can enhance the salience of stimuli or stimulus properties. One such study conducted by Theeuwes and Belopolsky (2012) used an oculomotor task to examine whether a stimulus associated with high monetary reward has a greater ability to capture the eyes than the same stimulus when associated with a low reward. Participants were trained to associate one stimulus (a vertical line segment) with a high monetary reward and another stimulus (a horizontal line segment) with a low monetary reward. During a test phase these stimuli were distractors, while observers searched for a colour singleton. The authors examined whether the eyes would be captured by the distractor line segments and whether this effect was modulated by the learned associated monetary reward. Participants were rewarded after each trial and received visual feedback about how much they had earned (10 cents or 1 cent). The amount of reward received was not related to participant performance, but instead was contingent upon the orientation of the target. Half of the participants received a higher reward when the target was vertical and half when it was horizontal. The reward schedule employed was probabilistic; high reward trial stimuli were followed by a high reward of 10 cents in 80% of the trials and a low reward of 1 cent in the remaining 20% of trials. The probabilities were reversed in low reward stimuli trials. Participants received feedback about their accumulated reward total after each block. Stimuli associated with high monetary reward were found to capture the eyes in a stronger fashion than that associated with low reward. The eyes were more frequently captured by the stimulus during training associated with high monetary reward than by the same stimulus when associated with a low monetary reward. Furthermore, even when the stimulus no longer predicted reward, the learned value of the reward increased exogenous capture of the eyes above and beyond that driven by salience alone. This finding is suggestive of a stimulus-specific persistent effect, such that after rewards were no longer predicted by the stimulus, the associations previously made were still able to affect the eyes. The findings demonstrate that the saccadic eye movement system can indeed be affected by reward, and that there is some persistence to these effects.

Theeuwes and Belopolsky (2012) concluded that learned value increased exogenous oculomotor capture of the eyes above and beyond that driven by salience alone. However, Theeuwes and Belopolsky (2012) failed to probe the extent of this persistence and the time-course of these effects. Furthermore, these associations were attributed to specific stimuli; therefore the

question remains as to whether a location in space can exhibit similar associations. Further to the findings of Theeuwes and Belopolsky (2012), several studies have shown that a specific stimulus or stimulus features associated with reward can change its physical salience in such a way that it becomes more pertinent than that same stimulus or feature when it is not associated with reward, and as such result in an automatic bias of attention (Della Libera & Chelazzi, 2009; Anderson, Laurent & Yantis, 2011a; Hickey, Chelazzi & Theeuwes, 2010a; Theeuwes, 1991, 1992). Incentivising stimuli increases the salience of the stimuli such that a stimulus paired with a high reward is more pertinent and therefore receives attentional priority beyond its physical properties.

In line with other research, Rothkirch, Ostendorf, Sax and Sterzer (2013) investigated whether the initiation of saccades is also influenced by the intrinsic motivational salience of a stimulus. The authors conducted two experiments. The first experiment investigated two key questions: 1) whether voluntary saccades are influenced by intrinsic motivational salience of the target stimulus; 2) whether execution of a saccade can be delayed due to the motivational salience of a distractor stimulus. Two tasks were employed within this particular experiment: A value learning task was used to enable participants to learn the association between face stimuli and assigned values. On each trial, participants were presented with a face pair (a face in the left hemifield and a face in the right hemifield). Participants were required to press a button nominating one of the faces, after which the nominated face was highlighted for 500ms and the signal to inform participants they had been rewarded was presented for 1500ms. Three different face pairs, each with a specific valence of monetary outcome, were used. One pair of faces was associated with monetary reward and classified as the positive valence face pair. One pair of faces was associated with monetary loss and classified as the negative valence face pair. The final face pair had a variable outcome. Within this pair, one face was assigned an 80% probability winning or losing money, whereas the other face had a 20% probability.

After completing this task participants completed a second task; the saccade task. Within this task, participant's eye movements were recorded. After a central fixation, a face appeared in either the left or right hemifield with a scrambled version of the same face presented to the opposite hemifield for 400ms. Participants were asked to make an eye movement to either hemifield. In the value learning task, learning was computed for each pair of faces. For each valenced pair of faces the authors computed a relative probability of optimal stimulus choice. So, for example, trials where participants chose a stimulus associated with a high probability of receiving a reward, or the stimulus with a low probability of losing money, were classified as

optimal choices. This learning was faster for rewarded face pairs, compared to unrewarded or punished stimuli. Participants were able to learn the association between reward and punishment values with the respective face stimuli before the end of the task. In the saccade task, for positive valence stimuli, the latencies of voluntary saccades towards faces with high motivational salience were significantly shorter compared to faces with low motivational salience. This result was not found for negative face values.

A second experiment was conducted to investigate whether the effect of the first experiment relied on object-specific processing or motivational values influence performance of reactive saccades. In this experiment participants completed the same tasks as experiment 1, however in the saccade task only one face was presented so that no discriminations had to be performed. In this experiment, no effects of motivational salience or valence of the stimulus on reactive saccade latencies was found. The authors concluded that initiation of saccade latencies towards visual stimuli are affected by acquired intrinsic motivational salience of stimuli. The findings are extended by showing that the motivational salience of a stimulus guides attentional selection processes underlying saccade initiation. The result of this particular study demonstrates the acquired intrinsic value of a stimulus can affect processes of attentional selection, indexed by the modulation of saccade latencies. Voluntary, not reactive, saccades being modulated indicates some degree of object specific neural processing for target identification is required to allow for an influence of motivational stimulus salience.

Although a number of studies have investigated the effects of rewards in a human population, the main focus has been the effects in relation to particular stimuli or stimulus features and more generally the effects of reward on attention. Only one study has explored the effects of reward on spatial attention. Camara, Manohar and Husain (2013) conducted a recent study that is directly relatable to the aims of the present thesis, investigating how the value associated with a location subsequently affects the involuntary capture of attention, as well as the deployment of goal-directed attention. The study comprised three experiments. Due to the comparisons between this investigation and the present thesis, this study will be examined in detail.

In the first experiment, each trial consisted of two phases. The first phase was a reward encoding phase, where monetary cues were used to associate reward information with spatial locations. Within this phase, six equidistant green circles were presented at 2, 4, 6, 8, 10 and 12 o'clock positions around a fixation point. Four of these circles changed to grey and two adjacent circles changed into a pound and a penny coin. These monetary cues were used to associate reward information with spatial locations. Within this phase participants were instructed to saccade from

the central fixation point towards the pound coin as fast possible. After making a saccade to the pound coin participants were rewarded. A saccade to the penny however, resulted in participants being penalised. The six green circles were then re-presented and participants fixated centrally until the second phase commenced. The second phase was classified as the probe phase, where the influence of reward associations established within the reward-encoding phase was probed in two ways: a 'free-choice' condition, where participants chose where they would look between two possible alternatives and a 'distractor' condition, where participants were shown the target to look at, accompanied by a distractor. The free-choice condition allowed assessment of the influence of reward or punishment in the subsequent phase on independent, goal-directed behaviour, whereas the distractor condition allowed examination of the effect of previous reward or punishment on stimulus-driven capture of behaviour. In the free-choice condition, four of the green circles changed to grey, except for the two circles where the coins were presented which remained green. Participants were required to make a saccade of their own choice to either of the green circles and were rewarded on the basis of their reaction times, classified as goal-directed behaviour. Stimulus-driven behaviour was measured by the influence of distractors in capturing gaze. Within this phase, four of the green circles changed to grey, except for one green target circle and one pink distractor circle at the locations previously occupied by coins. Participants were required to saccade to the green target, resulting in a reward. Generating a saccade to the distractor resulted in a penalty.

The results showed that in the reward-encoding phase participants chose the rewarded location (pound coin) significantly more often than the penalty location (penny coin). However, SRTs were significantly longer towards the rewarded location compared to the penalty location. In the free-choice of the probe phase significantly more saccades were directed to the previously rewarded location, however there was no difference found in participants SRTs. These results suggest that goal-directed action choices show a preference for previously rewarded locations. In the distractor version of the probe phase, visually salient distractors captured gaze on 31% of trials even though they led to a penalty. Further to this, distractors presented at a previously rewarded location captured the eyes significantly more often than distractors presented at the penalty location. Therefore, reward history affected stimulus-driven behaviour even when the visual salience of the distractor was the same.

In a second experiment, participants performed a similar task with a reward-encoding phase and a probe phase with free-choice and distractor trials. However, in this experiment the identity of the coins was not revealed until after the saccade was completed in the reward-encoding phase.

Therefore, initially participants chose freely which one of two brown circles to saccade to after which a pound or penny, selected randomly, was revealed at that location. Reward and penalty feedback was provided based on the cue value and the reaction time. Furthermore, in the distractor version of the probe phase the stimulus onset asynchrony (SOA) of the distractor was varied between 500ms, 150ms and 0ms. Consistent with the previous experiment's results, in the free-choice condition significantly more saccades went to previously rewarded locations. However SRTs did not differ. Furthermore, in the distractor condition, gaze was captured significantly more often if distractors were located at previously rewarded locations. These results indicate that stimulus-driven mechanisms and reward history contribute significantly to attention guidance.

A third and final experiment used a covert attention version of the paradigm employed in Experiment 2, requiring manual responses instead of SRTs. Consistent with the previous results, in the free-choice and distractor condition, reward history modulated manual response choices, whereas no significant differences were found in the RTs. In the free-choice condition participants pressed significantly more often to rewarded locations. In the distractor condition participants committed more errors when distractors were located at a previously rewarded location compared to a penalised one. These findings are consistent with the results of the previous experiments and strongly support the hypothesis that the previous value of a location subsequently affects the deployment of goal-directed attention as well as involuntary capture of attention on a covert attention task.

Although Camara et al., (2013) have demonstrated the effect of previous reward associations on spatial locations, the full extent of this effect has still not been probed. For instance, the time between the two types of experimental phase was two seconds. Therefore, any effect found during the reward-encoding phase persisted into the probe phase over the course of a matter of seconds. The extent to which the effects of reward persist once they are withdrawn and their merit in rehabilitation is yet to be established. A lengthy reward learning period followed by a period of reward withdrawal would be an adequate way to establish the time-course of these effects. Furthermore, the work of Camara et al., (2013) has established that rewards are able to transfer into a secondary task (probe phase), similar to that of the first task (reward-encoding phase). However, these two tasks are extremely similar, using the same experimental array and requiring the same type of eye movement. Therefore, it would be interesting to further probe the transfer of these reward-encoding effects in secondary, unrewarded eye movement tasks employing a different experimental array or different types of eye movement. Overall, this

experiment highlights reward associations to spatial locations can be created, subsequently affecting the deployment of both stimulus-driven and goal-directed deployment of attention.

### 1.7.3 Rewards in Rehabilitation

From the evidence provided within this section it is clear that there is a link between rewards and the oculomotor (Milstein & Dorris, 2007) and attention systems (Theeuwes & Belopolsky, 2012; Camara et al., 2013) in a neuro-typical human population. However, there is a lack of exploration as to the persistence of these effects or the transfer of these effects to other unrelated tasks, once rewards are withdrawn. This research has however led to the suggestion that monetary incentives can be harnessed for use in the rehabilitation of sufferers with certain visual field deficits.

Lucas et al., (2013) investigated the effects of reward on spatial attention in a population of neglect patients and neuro-typical human participants, using a novel gambling task with a spatial choice component and reward reinforcement. This task was derived from cancellation tests typically used in clinical studies. Visual targets were evenly distributed in space among distractors. In this version of the task participants were required to point to select only one of the targets displayed on any given trial; a choice which led to a variable amount of reward. Participants were instructed to guess and find the target with the highest gain. Once chosen, it was rewarded by 0, 5, 10 or 50 points. Participants were told that one target of the highest value (50 points) was always present. The aim was therefore to find the most rewarded target on each trial. Participants were also informed that previous subjects had gained approximately 500 points. Each trial consisted of a central fixation followed by an array of 4, 6 or 8 targets and 12 distractors. Target selection was paired with an audio-visual feedback highlighting the selecting target alone in bright yellow with the number of points gained overlapping it accompanied by a pleasant, melodic tone. Pointing to a distractor resulted in a brief white noise sound and pointing to neither a target nor distractor produced a simple visual tick. This feedback was then replaced by a screen displaying the total amount of points gained so far. In healthy participants, when rewards were distributed evenly across hemifields, no biases were found in the target choices of participants. However, when the highest value reward was presented to only one hemifield (the left), a progressive shift of choices towards this hemifield occurred. Overall, this data shows a significant spatial bias in target choices when the probability of high rewards were present in one hemifield. Patients initially selected targets on their right side more often than left. However, neglect patients

gradually shifted their choices to the left, where the highest rewards were on offer. Overall these findings reveal visual exploration and target selection can be biased by asymmetric reward distribution in space and can occur without any conscious awareness of the reward contingencies.

Furthermore, Malhotra, Soto, Li and Russell, (2013) employed two adapted versions of a standard cancellation task with a search array consisting of 54 targets (27 targets either side of the midline) amongst 52 matched distractors. 10 patients all suffering from neglect due to right hemisphere stroke completed both a rewarded and a baseline unrewarded condition. In the rewarded condition images of pound coins were used as rewarded targets, whereas in the unrewarded condition, brass buttons were the target stimuli (not associated with reward). Each array also contained a number of individual letters and words (e.g. 'ONE', 'FEEL', 'H', 'T'). Prior to testing and in the rewarded condition alone, patients were told they would receive a reward for each target that they found. Patients were reassessed on a second day to examine the effects of reward exposure. After reward exposure, patients were able to find targets for which they were previously unaware and remained unaware of in the unrewarded condition. Malhotra et al., (2013) offered a number of possible explanations for this result. Firstly, the authors postulated that rewards lead to heightened arousal resulting in improved performance in the rewarded condition. Secondly, Malhotra et al., (2013) suggested that rewards lead to increased target salience. Following incentive gain and performance feedback the relative salience of the pound targets may be modulated, enabling patients to find more targets in their blind field and shifting their centre of cancellation towards the neglected space. This particular explanation is consistent with previous findings of reward in attention in healthy human participants (Kristjánsson, Sigurjonsdottir, Driver & Fortune, 2010; Anderson et al., 2011). Therefore it is possible that the effect of reward may be mediated by arousal, salience or a combination of these mechanisms. This work is still in its infancy but the authors suggest that it may be possible to harness rewards in the rehabilitation of sufferers of visual field deficits. However, although some studies have been conducted on the effects of rewards on the oculomotor system there is still a great deal that is unknown. As stated earlier, a limited number of human studies have been conducted; all of which have failed to report the persistence of these effects once rewards are withdrawn, and whether these effects transfer into other unrelated and unrewarded tasks.

## 1.8 Summary

To summarise, work with non-human primates suggests that rewarding spatial locations can create a bias in the oculomotor system, such that eye movements are executed more quickly and accurately to rewarded locations (Bendiksby & Platt, 2006; Takikawa et al., 2002; Coe et al., 2002). These findings have been replicated in human participants (Milstein & Dorris, 2007) drawing conclusions that monetary incentives not only influence saccade metrics but also bias attention to stimulus features (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al., 2010a; Theeuwes, 1991, 1992; Theeuwes & Belopolsky, 2012) and spatial locations (Camara et al., 2013). These findings have prompted the suggestion that rewards can be used as a potential tool for rehabilitation, incentivising eye movements to reduce the deficits associated with spatial neglect (Malhotra et al., 2013; Lucas et al., 2013). However, at present, it is very difficult to outline the benefit of rewards as a tool for rehabilitation due to the lack of knowledge regarding two key factors; 1) the time-course of the reward effects found after withdrawal of incentives; 2) whether these effects transfer to other unrewarded tasks. While the findings of Malhotra et al., (2013) illustrate the potential for the use of rewards as a rehabilitative technique, it is difficult to know how to optimise reward-based therapies without knowing the *persistence* and *transfer* of these effects. Therefore it is important to investigate these effects within a neuro-typical human population prior to reaching any conclusions regarding the therapeutic merit of rewards in rehabilitation. To address these issues this thesis aims to extend the present knowledge held regarding the influence of rewards on the oculomotor system and establish whether monetary incentives can be harnessed as a rehabilitative tool. Using instrumental conditioning three key questions will be addressed: 1) do rewards influence the motor and attentional systems in healthy human participants? 2) how long do these effects persist for once rewards are withdrawn? 3) do these effects transfer to other eye movement tasks not associated with reward?

To create a reward paradigm able to produce consistent and persistent oculomotor behaviour change, it is imperative to take into account a number of different factors outlined in this literature review. One of the critical factors in designing a reward paradigm is the timing and the immediacy of reward feedback. The TD model of learning (Sutton & Barto, 1990) posits that learning occurs at certain time points throughout a trial. Therefore, it is crucial to keep the timing of trials consistent to produce optimal learning across the reward paradigm. Furthermore, the size of a reinforcer is a vital factor in the effectiveness of reward learning, such that the size of a reward should match the expectation of what is deserved for the completion of the task. In non-human primates, increasing the magnitude of reward reduced error rates and decreased SRTs

(Bendiksby & Platt, 2006; Takikawa et al., 2002; Kawagoe et al., 1998), a finding replicated in a human population (Milstein & Dorris, 2007). Therefore, providing a relatively large amount of reward for completing an arbitrary eye movement task should produce behaviour change. Previous studies have varied the amount of money used in their experimental designs, ranging from 10 cents (Theeuwes & Belopolsky, 2012) to £1 (Malhotra et al., 2013; Camara et al., 2013). Therefore, a monetary value between these two would be adequate for reward feedback within a reward paradigm. A further factor to consider is the contingency of reward to employ. The Rescorla-Wagner model of reinforcement (Rescorla & Wagner, 1972) suggests that learning only happens when rewards are not predicted. Therefore, it is important to employ a schedule that does not consistently reward participants for eye movements to the same location. Further to this point, continuous reinforcement in previous experiments has been more susceptible to extinction when compared with variable-rewarded schedules (Sheffield, 1949; Capaldi, 1967, 1994).

The time required for conditioning and the speed at which extinction of the conditioned behaviour occurs is directly related to the value, predictability and delivery characteristics of the reward (Skinner, 1983). The perceived value, predictability and frequency of a reward are usually negatively correlated to the duration of both conditioning and extinction (Zimmerman, 1963). By combining these three features a number of reward schedules, each with their own conditioning and extinction characteristics, can be produced (Kelleher & Gollub, 1962). By using a variable-ratio schedule, where rewards are given based on a predefined probability, the predictability of rewards is significantly reduced which also increases the duration of conditioning and resistance to extinction. Using this knowledge, Chapter 2 presents three reward paradigms, used in three different experiments, each with their own schedule of reward. The aim of employing three different paradigms of reward was to build on the understanding of previous reward research in a human population outlined in this introduction. Primarily, the aim was to replicate the previously obtained primate data of rewards influencing saccade metrics and to verify whether these effects transferred to a healthy human population. The second aim was to investigate whether any effective paradigm could be optimised to produce the greatest effect of reward.

# Chapter 2: The effects of varying reward schedules on the saccadic eye movement system

## 2.1 Introduction

The introductory chapter to this thesis demonstrated that most of the knowledge about the mechanisms underlying visuospatial and visuomotor processing and reward is derived from electrophysiological recordings in macaques and other primates (Takikawa et al., 2002; Kawagoe et al, 1998; Watanabe et al., 2003; Bowman et al., 1996; Bendiksby & Platt, 2006; Sohn & Lee, 2006). Only recently has investigation of these processes been applied to neuro-typical human individuals. For example, expectation of reward is sufficient to significantly influence saccadic behaviour, reducing latencies in 11 healthy human participants (Milstein & Dorris, 2007). Other research, outlined in the introduction has shown that stimuli associated with a high reward have a stronger effect on the oculomotor system than exactly the same stimuli when associated with a low reward (Theeuwes & Belopolsky, 2012; Hickey et al., 2010a). Although this research is not directly comparable to the primate data because the reward was associated with *non-spatial* object properties, rather than a spatial location, it is a relevant finding in shaping a paradigm that will lead to persistent facilitation. This effect of reward has also been associated with locations, with attention found to be directed towards previously rewarded locations even when this interfered with ulterior task demands (Camara et al., 2013). This research, outlined in more detail in the introductory chapter, suggests that eye movements will be biased towards the hemifield with a relatively higher frequency of reward.

To summarise, work with non-human primates suggests that rewarding spatial locations can create a bias in the oculomotor system, such that eye movements are executed more quickly and accurately to rewarded locations. Whilst the few studies which have attempted to replicate this work in human participants show that similar effects can be observed in humans, they did not: 1) use paradigms that are directly comparable to the primate work (Theeuwes & Belopolsky, 2012; Hickey et al., 2010a); 2) investigate the extent to which learning persists beyond the withdrawal of reward (Milstein & Dorris, 2007; Bendiksby & Platt, 2006); 3) investigate the effects of the saccadic system itself, but use the system instead as a tool to learn more about the principles of decision making (Platt & Glimcher, 1999; Dorris & Glimcher, 2004). Establishing these crucial pieces of information in neuro-typical humans can result in guiding future research on the effectiveness of monetary reinforcers in a neuro-atypical human population. To address these issues three tasks were developed to examine the effect of reward in a way that closely followed the experimental design of the primate work. These tasks each consisted of three stages: a

preconditioning phase to obtain baseline data; a conditioning phase where facilitation of saccades by monetary rewards was explored; an extinction phase investigating the time-course of the facilitation effect.

The first task rewarded participants in both hemifields, one to a greater extent than the other. It was expected that participants would elicit significantly faster saccades and be more accurate to the hemifield where a greater number of rewards were received compared to the lower frequency rewarded hemifield. The second task presented participants with rewards to only one hemifield in order to further probe the relationship between reward and the oculomotor system and analyse the effects of rewarding a single spatial location. It was predicted that participants would saccade faster and be more accurate to the rewarded hemifield than the unrewarded hemifield. The final task aimed to maximise the effects of reward by pairing rewards with an auditory tone, as research has suggested that faster saccades occur to bimodal stimuli presented in the same spatial or temporal proximity (Colonius & Diedrich, 2002; Colonius & Arndt, 2001). It was predicted that after associating the auditory tone with receiving a reward participants would elicit significantly faster saccades to this location, maximising the effects of the reward paradigm. These four experiments all used a sample size of 12 participants as most other studies report this as a convincing sample size to produce significant effects and for examining these low-level mechanisms in human saccadic eye movements (Milstein & Dorris, 2007; Camara et al., 2013; Theeuwes & Belopolsky, 2012).

## 2.2 Experiment 1

### 2.2.1 Method

#### *2.2.1.1 Participants*

Twelve participants recruited from the University of Durham volunteered for the experiment. The participants - five male and seven female – had an age range of 19-29 years (mean age 21.58 years). Seven were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

*2.2.1.2 Apparatus*

Participants were required to complete a consent form (see Appendix A) prior to taking part. The experimental stimuli were generated using a Cambridge Research Systems ViSaGe graphics card and displayed on a 17 inch Eizo Flexscan Colour Display monitor with a refresh rate of 100Hz. Responses were collected using a two-button button box. Eye movements were recorded using a Cambridge Research Systems eye tracker with a sampling rate of 160Hz. The experiment was programmed in C++.

*2.2.1.3 Stimuli*

Participants were presented with a black 0.3° x 0.3° fixation cross in the centre of the screen (0°) on a grey background. A target stimulus 0.5° x 0.5° square was presented to the left or right of the fixation cross. The stimuli were presented 6.5° to the left and 3.7° upwards from fixation. After a rewarded trial participants were presented with reward feedback green text with a luminance of 19.61 cm/2 stating '10p'. After an unrewarded trial participants were presented with reward feedback red text with a luminance of 19.69 cm/2 of '0p'.

*2.2.1.4 Procedure*

Eye dominance was assessed for each participant by seating them at a distance of two metres from the experimenter and fixating on the nose of the experimenter. Participants were then asked to extend their arms and bring their hands together in front of their eyes, leaving a small gap through which the participant could see the experimenter's face. Through this gap the experimenter could see only one of the participant's eyes: the visible eye was recorded as dominant.

Participants were seated 57cm away from the display with their head resting on a chinrest. A headband was placed around the top of the head to secure the participant's head, controlling head movements. Participants underwent a 9-point calibration procedure prior to experimentation.

A three-phase experimental paradigm was constructed consisting of a Preconditioning phase (4 blocks, 240 trials), a Conditioning phase (10 blocks, 600 trials) and an Extinction phase (6 blocks, 360 trials). Figure 2.1 displays the experimental array. Each block contained 60 trials with the

entire conditioning task lasting 20 blocks. Participants were instructed to fixate on the central fixation cross prior to the start of each trial. A fixation time period was programmed in which a lower limit of 500ms and an upper limit of 800 ms was computed, followed by a target stimulus square in either the left or right hemifield. After a successful saccade the target stimuli would change colour from black to grey. During the preconditioning phase of the experiment participants received no reward or reward feedback. During the conditioning phase of the experiment participants were rewarded for fast and accurate saccades made only on rewarded trials. A variable-ratio reward schedule was employed. Of the 300 trials to the high rewarded frequency hemifield, 120 were rewarded (40%). Of the 300 trials to the low rewarded frequency hemifield, 60 were rewarded (20%). On a rewarded trial, participants would receive additional information in the form of green text of '10p' presented in Arial font. On an unrewarded trial, red text of '0p' would be displayed below the original target stimuli. During the extinction phase of the experiment, all reward was removed and participants would only receive feedback of red text of '0p', regardless of which hemifield the probe was presented to.

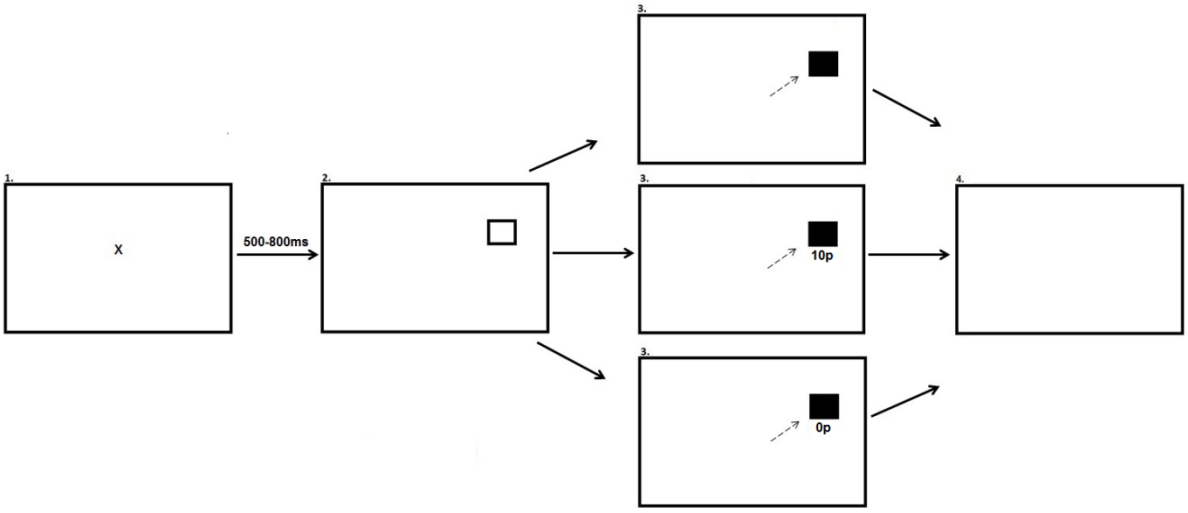


***Figure 2.1:*** *Sequence of events used in Experiment 1 for the conditioning task (not to scale). First a fixation cross appeared (Panel 1). Secondly, the fixation cross disappeared and a target square appeared in either the left or right visual field (Panel 2). Row 1 represents the preconditioning phase. After making a saccade towards the square it would change colour (Row 1, Panel 3). After a button press participants would be presented with a blank screen (Panel 4) indicating the trial had finished and a new trial was about to begin. Row 2 represents the conditioning phase. After a successful saccade the square would change colour and participants would be presented with a green '10p' on a rewarded trial (Row 2, Panel 3), the frequency of these rewards would vary depending on which hemifield the target was presented in. On an unrewarded trial participants would see a red '0p'. Row 3 represents the extinction phase. After a successful saccade the square would change colour and participants would be presented with a red '0p' regardless of the hemifield a trial was presented to as all reward was removed during this phase (Row 3, Panel 3).*

*2.2.1.5 Saccade Analysis*

The analysis was conducted on the mean of each participant's average SRT calculated from each individual block. Data was filtered so that saccadic error and trials over 500 ms were eliminated from the analysis; saccadic error refers to those trials in which saccades left the fixation area but did not land at the designated target location. In total, 1778 trials (12.3% of the data set) were excluded from analysis. Due to the large percentage of rejected trials the analyses were replicated using median saccade reaction times. Median SRTs are robust against outliers and a better measure of central tendency for latency. These analyses are reported alongside the mean SRT analyses.

**2.2.2 Results**

A significance correction level of $p < .05$ was adopted, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*2.2.2.1 Latency*

Using mean SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: High Frequency Reward/Low Frequency Reward) repeated measures ANOVA on mean SRTs revealed a significant effect of Experimental Phase ($F (2, 22) = 4.15$, $p = .03$, $r = .40$) such that saccades were significantly faster in the extinction phase compared to the preconditioning ($t (11) = 2.24$, $p = .04$, $r = .56$) and conditioning ($t (11) = 2.38$, $p = .05$, $r = .58$) phases; however, these did not survive the correction for multiple comparisons.

No effect of Hemifield ($F (1, 11) = .59$, $p = .56$, $r = .23$) and no significant interaction was found between Experimental Phase and Hemifield ($F (2, 22) = .73$, $p = .49$, $r = .18$). Figure 2.2 illustrates these results.

Planned comparisons revealed no significant difference between the latencies of prosaccades to either hemifield in the preconditioning ($t (11) = .11$, $p = >.017$, $r = .03$), conditioning ($t (11) = .50$, $p = >.017$, $r = .21$), or extinction ($t (11) = 1.20$, $p = >.017$, $r = .34$) phases.

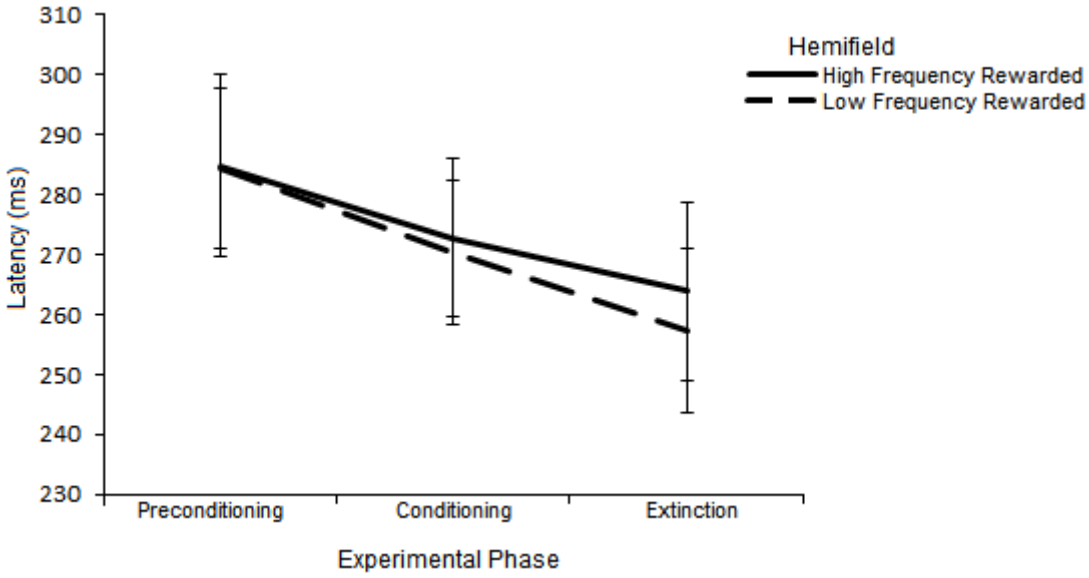


***Figure 2.2:*** *Mean saccadic latency (ms) to the high frequency (black line) and low frequency (black dashed line) rewarded hemifields across experimental phases. Error bars show +/- 1 SEM.*

Median SRT analysis replicated the results found in participants mean SRT analyses. A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: High Frequency Reward/Low Frequency Reward) repeated measures ANOVA revealed a significant effect of Experimental Phase ($F(2, 22) = 3.36$, $p = .05$, $r = .36$) such that saccades were significantly faster in the extinction phase (253 ms) compared to the preconditioning (271 ms) and conditioning phases (262 ms). No effect of Hemifield ($F(1, 11) = 1.68$, $p = .22$, $r = .36$) and no significant interaction was found between Experimental Phase and Hemifield ($F(2, 22) = .35$, $p = .71$, $r = .12$).

*2.2.2.2 Saccadic Error*

For the purpose of this error analysis, saccadic errors previously excluded from the latency analysis were included. Anticipatory trials and trials above the set threshold were excluded.

Error trials occurring in the conditioning and extinction phases were analysed to investigate whether participant errors could be attributed to a conditioning effect on their visual system. The proportion of total errors to each hemifield was calculated for each individual participant. A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: High Frequency Reward/Low Frequency Reward) repeated measures ANOVA on the proportion of saccadic error

revealed no effects of Experimental Phase ($F(2, 22) = 2.128$, $p = .14$, $r = .30$), Hemifield ($F(1, 11) = 1.27$, $p = .28$, $r = .32$) or interaction between the two variables ($F(2, 22) = .03$, $p = .97$, $r = .04$). From these findings it can be concluded that reward had no effect on participant accuracy.

### 2.2.3 Discussion

The aim of this experiment was to generate a sustainable reward schedule that could lead to a change in the exploratory behaviour of the human oculomotor system. The intentions of the study were two-fold. The primary aim was to discover whether a facilitation effect could be generated towards the hemifield being rewarded more frequently than the hemifield being rewarded less often, a result found in primate research (Bendiksby & Platt, 2006). This facilitation effect would be manifested in faster SRTs after the introduction of reward and throughout the conditioning phase of the experiment. The second intention was to investigate the time-course of this facilitation and the extent to which learning persists beyond the withdrawal of reward.

The results of the first experiment indicate that using the particular reward schedule in which both hemifields are rewarded – one to a greater extent than the other – had no hemifield-specific effect on the human oculomotor system. There were no significant differences registered throughout each phase of the experiment. This lack of effect was replicated in participant accuracy. This particular reward schedule failed to produce any hemisphere-specific facilitation effect. Furthermore, as there were non-significant results throughout the experiment, there was no sustained bias on the oculomotor system. However, rewards did generate a significant speeding of SRTs over the course of the experiment. This finding may be attributable to participant's anticipation of a reward producing a non-specific facilitation of participants SRTs (Dorris & Glimcher, 2004).

The results yielded from this experiment are contrary to previous research within a primate population. As stated previously Coe et al., (2002) found that when presented with two targets of varying value, primates will shift their gaze to the target of higher value. Furthermore, in the visual-1DR task employed by Hikosaka et al., (2006), primate latencies were much shorter when saccades were followed by a big reward than when they were followed by a small reward. In the present experiment, one hemifield was rewarded more frequently than the other and found no indication of saccadic facilitation towards the higher value hemifield. It has previously been found that the predicted presence of reward alone facilitates the preceding behaviour (Robbins & Everritt 1996; Schultz, Apicella, Scarnati & Ljungberg, 1992; Bowman et al., 1996; Tremblay &

Schultz, 2000). In rewarding both hemifields an expectation of reward was present for both sides of the visual field, possibly leading to a lack of discrimination between hemifields. This may explain the lack of a significant difference between the latencies towards either hemifield. The reward schedule used may have been insufficient for participants to be able to distinguish between the two hemifields. Therefore, participants came to expect to receive a reward from either hemifield and so neither hemifield received priority.

Further differences in the methodology between the present study and the previous studies might explain the failure to replicate the effects observed in non-human primates. Coe et al., (2002) presented their stimuli of varying values simultaneously, whilst Hikosaka et al., (2006) varied their higher and lower frequency hemifields throughout the duration of the experiment. A further difference lies in the way in which these studies computed their rewards. Coe et al., (2002) rewarded primates through a number of different policies, in which a reward to one hemifield would incrementally increase depending on the primate's success. In this case the primate received an incremental increase to one hemifield. In the present study, the participant saw the same stimulus (10p) when making a correct saccade but the frequency to one hemifield was increased. It is possible that humans are less sensitive to this method of reward delivery and that regardless of whether reward is high or low frequency, so long as it is present, behaviour is facilitated. The same explanation can be applied to the Hikosaka et al., (2006) visual-1DR task, in which primates are receiving a large reward in one direction for 20-60 trials, after which the direction of the large reward would change. It is possible that by changing the direction of the big reward, the primates are constantly readjusting their oculomotor priority and so a difference between the different reward states is clear. However, the difference between reward states in the present study was less obvious.

In summary, no hemifield-specific effect of saccadic facilitation was found when financial incentives were presented to both hemifields at differing frequencies; findings from primate research in a human population were therefore not reproduced (Bendiksby & Platt, 2006; Takikawa et al., 2002; Coe et al., 2002; Hikosaka et al., 2006). However, rewards produced a more general effect of facilitation, speeding saccadic eye movements in conditions subsequent to reward delivery. As the present reward schedule failed to induce a bias among the population tested, a revised reward schedule was employed. Instead of rewarding both hemifields with varying frequency, the amended reward schedule would reward only one hemifield. This new schedule would allow investigation of whether the previous schedule was unable to find an effect due to differences between primate and human sensitivity to reward. It was hypothesised that

exclusively rewarding only one hemifield would be more likely to generate a facilitation effect towards the direction of reward, manifested in participants SRTs and accuracy.

## 2.3 Experiment 2

### 2.3.1 Method

#### *2.3.1.1 Participants*

Twelve participants recruited from the University of Durham volunteered for the experiment. Individuals who had previously participated, and as such had experience of the reward paradigm, were not allowed to participate. The participants – two male and ten female – had an age range of 19-48 years (mean age 24.92 years). Ten were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *2.3.1.2 Apparatus*

The apparatus was the same as described in Experiment 1.

#### *2.3.1.3 Stimuli*

The experimental stimuli and setup was as described in Experiment 1.

#### *2.3.1.4 Procedure*

The procedure employed in Experiment 1 was replicated in the present experiment with some amendments made to the reward paradigm. A three-phase experimental paradigm was again employed with a Preconditioning phase (2 blocks, 120 trials), a Conditioning phase (10 blocks, 600 trials) and an Extinction phase (6 blocks, 360 trials). Each block contained 60 trials with the entire experiment lasting a revised 18 blocks. In this version of the paradigm only one hemifield was rewarded. Of the 300 trials to the rewarded hemifield, 180 were rewarded (60%). All trials to the opposite hemifield were unrewarded.

*2.3.1.5 Saccade Analysis*

The analysis was conducted on the mean of each participant's SRT average, calculated from each individual block. The same inclusion criteria as employed in Experiment 1 were replicated. In total, 1607 trials (12.4% of the entire data set) were excluded from analysis. These analyses were once again replicated using median SRTs.

**2.3.2 Results**

A significance correction level of $p < .05$ was adopted, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*2.3.2.1 Latency*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed a main effect of Experimental Phase ($F(2, 22) = 3.59$, $p = .05$, $r = .37$). Further analysis revealed significantly faster eye movements during the conditioning phase (245 ms) compared to the preconditioning (265 ms) phase ($t(11) = 2.06$, $p = .03$, $r = .53$), although this did not survive the correction for multiple comparisons. A main effect of Hemifield was also revealed ($F(1, 11) = 5.78$, $p = .04$, $r = .59$) such that saccades were significantly faster to the rewarded hemifield (243 ms) compared to the unrewarded hemifield (264 ms). A trend towards an interaction between Experimental Phase and Hemifield was found ($F(2, 22) = 2.64$, $p = .09$, $r = .33$).

Planned comparisons revealed a non-significant difference between the latencies of participant saccades in the rewarded (M = 264 ms, S.D. = 44.17) and unrewarded (M = 267 ms, S.D. = 42.43) hemifields for the preconditioning phase ($t(11) = -.24$, $p = >.017$, $r = .07$). In contrast, a significant difference was found between the saccadic latencies in the rewarded (M = 224 ms, S.D. = 13.48) and unrewarded (M = 265 ms, S.D. = 40.72) hemifields during the conditioning phase ($t(11) = -3.09$, $p = <.017$, $r = .68$). After the removal of reward, no significant differences between the saccadic latencies in the rewarded (M = 241 ms, S.D. = 34.18) and unrewarded (M = 261 ms, S.D. = 37.22) hemifields for the extinction phase was found ($t(11) = -1.44$, $p = >.017$, $r = .40$). Figure 2.3 illustrates the effects.

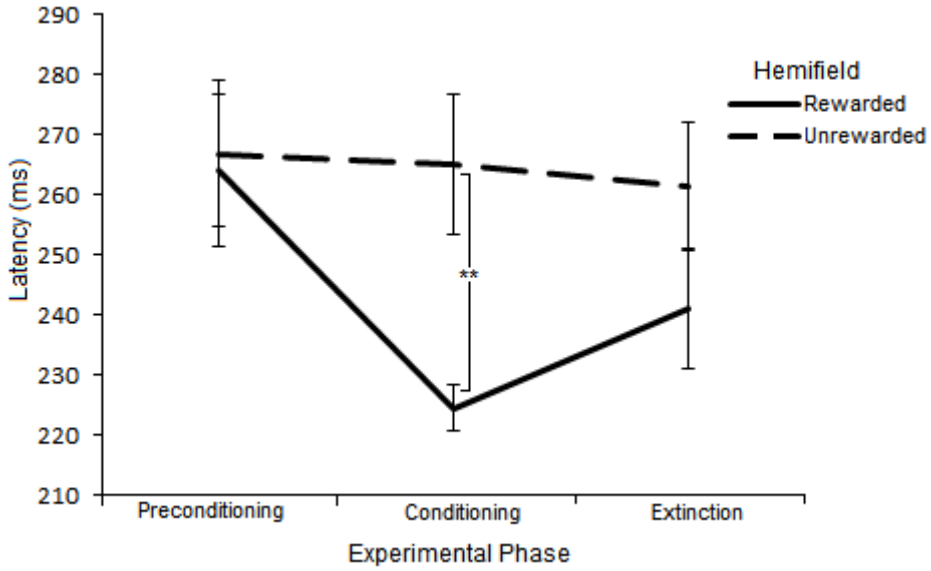


***Figure 2.3:*** *Mean saccadic latency (ms) to the rewarded (black line) and unrewarded (black dashed line) hemifields across experimental phases. Error bars show +/- 1 SEM.*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on median SRTs replicated the main effect of Hemifield ($F(1, 11) = 8.15$, $p = .02$, $r = .65$) such that saccades were significantly faster to the rewarded hemifield (245 ms) compared to the unrewarded hemifield (263 ms). A trend towards an interaction between Experimental Phase and Hemifield was also replicated ($F(2, 22) = 3.26$, $p = .06$, $r = .36$).

Further paired t-tests revealed a non-significant difference between the latencies of participant saccades in the rewarded (264 ms) and unrewarded (M = 267 ms) hemifields for the preconditioning phase ($t(11) = -.24$, $p = >.017$, $r = .58$). In contrast, a significant difference was found between the saccadic latencies in the rewarded (M = 225 ms) and unrewarded (M = 264 ms) hemifields during the conditioning phase ($t(11) = -2.97$, $p = <.017$, $r = .67$). After the removal of reward, no significant differences between the saccadic latencies in the rewarded (M = 247 ms) and unrewarded (M = 257 ms) hemifields for the extinction phase was found ($t(11) = -1.74$, $p = >.017$, $r = .46$). Figure 2.3 illustrates the effects.

*2.3.2.2 Time-course of the Extinction*

The effects of reward found within the conditioning phase permits investigation of the extinction effects of reward learning, a previously unreported finding in studies investigating the effects of reward on the oculomotor system and attention. The first three blocks of the extinction phase were grouped analysed against the final three blocks of the extinction phase.

A 2 (Hemifield: Rewarded/Unrewarded) x 2 (Group: 1/2) repeated measures ANOVA on mean SRTs revealed no effect of Hemifield ($F(1, 11) = 2.08$, $p = .18$, $r = .40$) or Group ($F(1, 11) = .03$, $p = .86$, $r = .05$). However a trend towards an interaction between Hemifield and Group ($F(1, 11) = 4.59$, $p = .06$, $r = .54$) was revealed which was further explored. Paired t-tests revealed a significant difference between the rewarded and unrewarded hemifields in the first three blocks of the extinction phase (Group 1) ($t(11) = -3.32$, $p < .025$, $r = .71$). However no significant difference was found between the final three blocks of the extinction phase (Group 2) ($t(11) = -.30$, $p > .025$, $r = .09$).

Using participants median SRTs a 2 (Hemifield: Rewarded/Unrewarded) x 2 (Group: 1/2) repeated measures ANOVA replicated no effect of Hemifield ($F(1, 11) = 3.01$, $p = .11$, $r = .46$) or Group ($F(1, 11) = .21$, $p = .66$, $r = .14$). The trend towards an interaction between Hemifield and Group ($F(1, 11) = 3.97$, $p = .07$, $r = .51$) was also replicated which was further explored. Paired t-tests revealed a significant difference between the rewarded and unrewarded hemifield in the first three blocks of the extinction phase (Group 1) ($t(11) = -4.51$, $p < .025$, $r = .81$). The non-significant difference found between the final three blocks of the extinction phase (Group 2) was replicated ($t(11) = -.34$, $p > .025$, $r = .10$).

Figure 2.4 illustrates the block-by-block breakdown of this result across the extinction blocks in the extinction phase using mean participants mean SRTs.

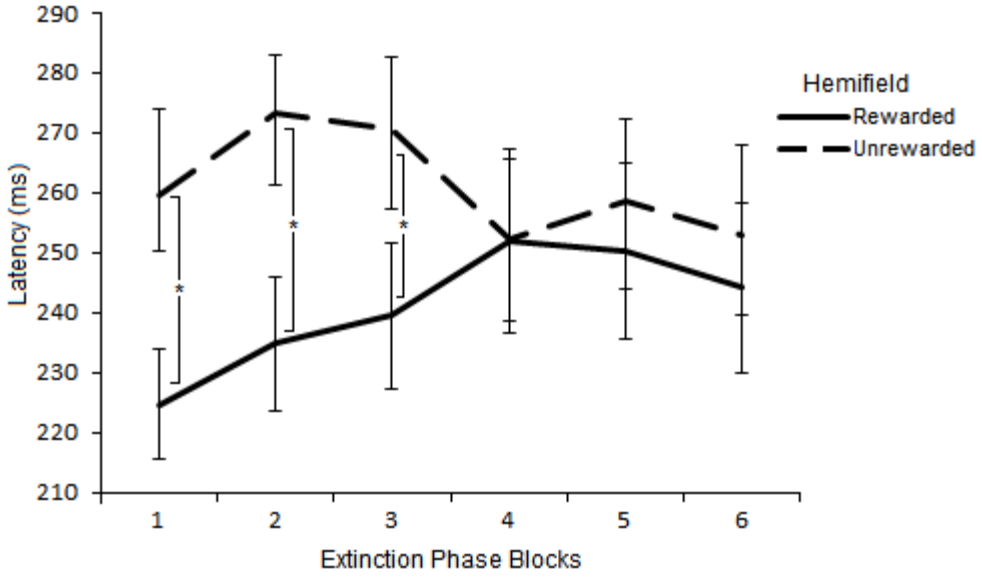


***Figure 2.4:*** *Mean saccadic latency (ms) to the rewarded (black line) and unrewarded (black dashed line) hemifields in the extinction phase. Error bars show +/- 1 SEM.*

*2.3.2.3 Saccadic Error*

Using the total proportion of errors a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed no significant effect of Hemifield ($F(1, 11) = .02$, $p = .90$, $r = .04$) or interaction between Experimental Phase and Hemifield ($F(2, 22) = .38$, $p = .69$, $r = .13$).

**2.3.3 Discussion**

The aim of this experiment was to elaborate on Experiment 1 and generate a sustainable reward schedule leading to a change in the exploratory behaviour of the human oculomotor system. In a revised attempt to discover whether a facilitation effect could be generated, one hemifield was nominated as the rewarded hemifield where participants received all their incentives. This revised schedule found a facilitation effect manifested in faster SRTs after the introduction of reward and throughout the conditioning phase of the experiment. The second intention was to investigate the time-course of this facilitation, finding significant differences into the extinction phase after withdrawal of reward. These effects were not observed in the proportion of saccadic error across the experimental phases.

Rewarding a single hemifield created significant differences in the speed at which saccadic eye movements were elicited to a visual stimulus, consistent with previous research (Milstein & Dorris, 2007). However, this study extended the previous work in one important way: Specifically, the time-course of extinction was examined. Although this effect was found when monetary incentives were present, the effect of facilitation had a limited time-course, with extinction of the effects of reward on SRT occurring after 3 blocks of no-reward trials, approximately ten minutes. Analysing this time-course block-by-block illustrates the facilitation effects of reward appear robust for the initial three blocks once rewards were withdrawn, after which the effects rapidly extinguish (see figure 2.4). This result illustrates the fragility of the facilitation effect and highlights the importance of context, a crucial factor in building effective stimulus-reward associations, in reward learning (Blaukopf & DiGirolamo, 2007).

In summary, the findings of this study demonstrated that the present reward schedule facilitated saccadic eye movement in the direction of reward; an effect sustained for a short period of time after the withdrawal of incentives. Therefore it was concluded that small monetary rewards were able to influence the metrics of the saccadic eye movement system. Recent research has suggested that participants tend to make faster saccades to visual targets when these are paired with stimuli from a different modality presented in the same spatial or temporal proximity (Hershenson, 1962; Simon & Craft, 1970; Colonius & Diedrich, 2002; Colonius & Arndt, 2001). Based on this research and after finding that participants display significantly faster saccades to a rewarded hemifield, further investigation was conducted into whether it was possible to strengthen this effect by associating participants reward with an auditory tone. In second order conditioning, if stimulus B predicts an affective outcome and stimulus A predicts stimulus B, then by association stimulus A gains reward predictive value. The purpose of this investigation was to produce a manipulation of the previously used reward paradigm in order to create the strongest possible bias towards the rewarded hemifield. By pairing the reward with a sound after participants made a saccade to a target stimulus, it was thought that the strength of the conditioning would be increased (Harrington & Peck, 1998; Hughes, Nelson & Aronchick, 1998; Steenken, Colonius, Diederich & Rach, 2008; Corneil et al., 2002).

Based on these findings and using the same paradigm as previously employed, every time participants received a reward they also heard a 1 kHz auditory tone. This experiment used a between-subjects design. Participants were divided into two conditions; a ‘With Sound’ condition and a ‘Without Sound’ condition. During the extinction phase of the experiment, participants in the ‘With Sound’ condition, although receiving no reward, still heard the 1 kHz auditory tone at

the same probability as they received reward (60%), lateralised to the previously rewarded hemifield. In the 'Without Sound' condition, participants experienced the same extinction phase as had been experienced during the previous experiment (Experiment 2), with no auditory feedback. Based on the findings of Experiment 2, it was hypothesised that participants would exhibit significantly faster SRTs to the rewarded hemifield than the unrewarded hemifield. A second hypothesis formulated that effects found in the 'With Sound' condition would persist longer into the extinction phase than effects found in the 'Without Sound' condition, as sound would be a conditioned stimulus still present during the extinction phase in the 'With Sound' condition only, based on the principles of reinforcement learning (Rescorla & Wagner, 1972; Barto, Sutton & Anderson, 1983).

## 2.4 Experiment 3

### 2.4.1 Method

#### *2.4.1.1 Participants*

Twelve participants recruited from the University of Durham volunteered for the experiment. Individuals who had previously participated, and as such had experience of the reward paradigm, were not allowed to participate. Six participants - three male and three female – with an age range of 18-26 (mean age 24.92 years) completed the 'With Sound' condition. Six participants – two male and four female – with an age range of 18-28 (mean 21.83) completed the 'Without Sound' condition. Five participants from each group were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *2.4.1.2 Apparatus*

The apparatus was the same as described in Experiments 1 and 2 with the addition of a set of Dell multimedia speakers, with a 12 volt input, used to generate the auditory tone. These were lateralised according to the hemifield in which the participant would be receiving their rewards. Participants were also required to complete an amended consent form prior to participation (see Appendix B).

*2.4.1.3 Stimuli*

The experimental stimuli and setup was as described in Experiment 1.

*2.4.1.4 Procedure*

The procedure employed in Experiment 2 was replicated in the present experiment. In the 'With Sound' condition the '10p' reward feedback was paired with the 1 kHz tone during the conditioning phase. The tone was played for 400ms. The tone occurred on 30% of trials in each block of the extinction phase also; the same rate as during the conditioning phase. In the 'Without Sound' condition, the auditory tone was coupled with reward during the conditioning phase only with no auditory feedback present in the extinction phase of this condition. The tone did not sound during the extinction phase. No auditory tone was played during the preconditioning phase in either condition.

*2.4.1.5 Saccade Analysis*

The analysis was conducted on the mean of each participant SRT average calculated from each individual block. The exclusion criteria employed in Experiment 1 and 2 was replicated in the present experiment. In total 30.8% of the entire data set was removed prior to analysis. These analyses were once again replicated using median SRTs.

**2.4.2 Results**

A significance correction level of $p < .05$ was adopted, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*2.4.2.1 Latency*

*2.4.2.1.1 'With Sound' during Extinction Phase*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed no effect of

Experimental Phase (F (2, 10) = .19, p = .83, r = .12), Hemifield (F (1, 5) = .31, p = .60, r = .24) or interaction between Experimental Phase and Hemifield (F (2, 10) = .17, p = .85, r = .28).

Planned comparisons revealed no significant differences between the latencies of saccades to the rewarded and unrewarded hemifields in the preconditioning (t (5) = .18, p = >.017, r = .08), conditioning (t (5) = -.68, p = >.017, r = .09) or extinction phases (t (5) = -.45, p = >.017, r = .04). Figure 2.5 illustrates these results.

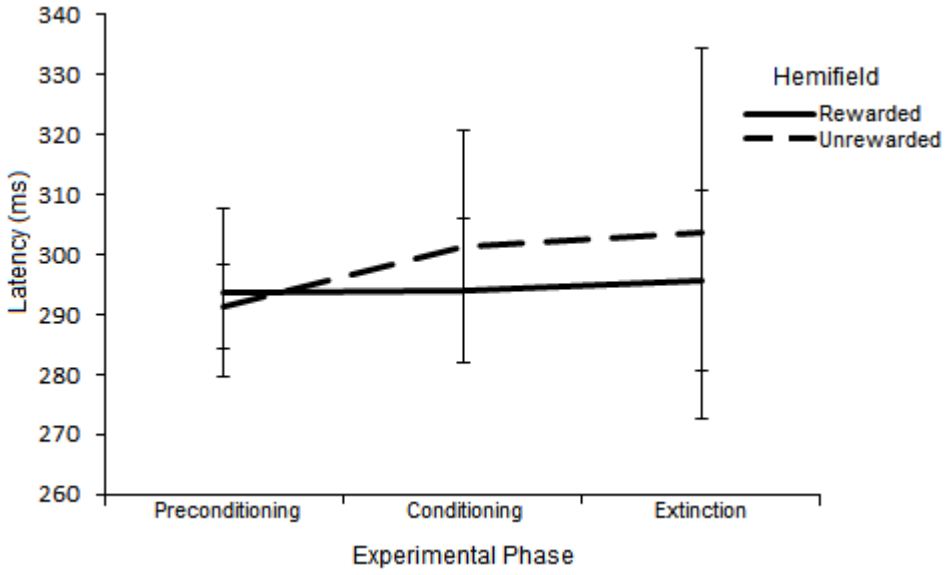


***Figure 2.5:*** *Mean saccadic latency (ms) to the rewarded (black line) and unrewarded (black dashed line) hemifields across experimental phases in the 'With Sound' condition. Error bars show +/- 1 SEM.*

These effects were replicated in participants' median SRTs. A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated no effect of Experimental Phase (F (2, 10) = .72, p = .51, r = .26), Hemifield (F (1, 5) = .01, p = .95, r = .03) or interaction between Experimental Phase and Hemifield (F (2, 10) = 1.20, p = .34, r = .33).

Replication of the planned comparisons revealed no significant differences between the latencies of saccades to the rewarded and unrewarded hemifields in the preconditioning (t (5) = 1.65, p = >.017, r = .45), conditioning (t (5) = -.61, p = >.017, r = .26) or extinction phases (t (5) = -.27, p = >.017, r = .12).

*2.4.2.1.2 'Without Sound' during Extinction Phase*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed no significant effect of Experimental Phase ($F(2, 10) = 1.00$, $p = .40$, $r = .30$) or Hemifield ($F(1, 5) = 1.96$, $p = .22$, $r = .53$). Furthermore no interaction was found between Experimental Phase and Hemifield ($F(2, 10) = .96$, $p = .41$, $r = .30$).

Planned comparisons revealed no significant differences between the latencies of saccades to the rewarded and unrewarded hemifields in the preconditioning ($t(5) = -.18$, $p = >.017$, $r = .08$), conditioning ($t(5) = -2.20$, $p = >.017$, $r = .70$) or extinction phases ($t(5) = -1.81$, $p = >.017$, $r = .63$). Figure 2.6 illustrates these results.

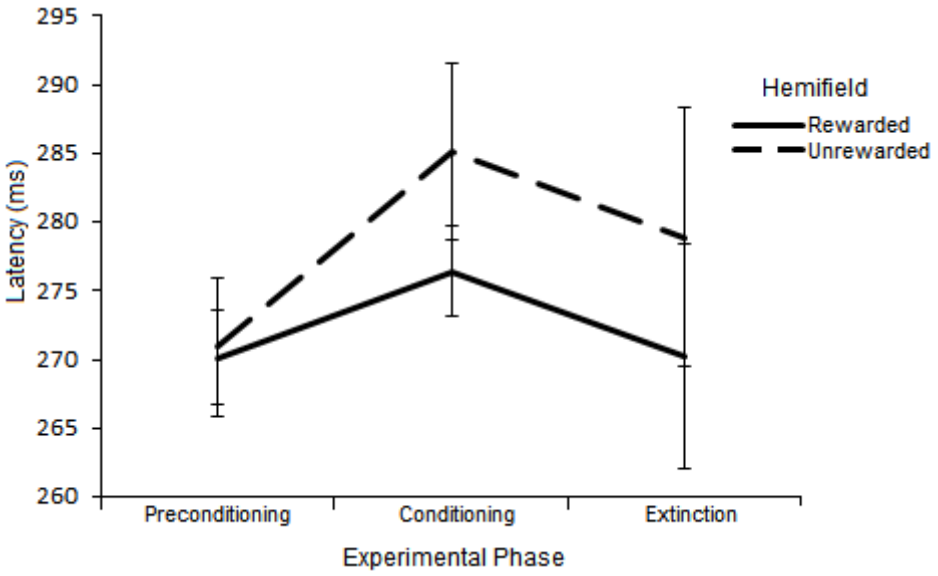


***Figure 2.6:*** *Mean saccadic latency (ms) to the rewarded (black line) and unrewarded (black dashed line) hemifields across experimental phases in the 'Without Sound' condition. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated the null effects of Experimental Phase ($F(2, 10) = 1.19$, $p = .34$, $r = .44$) and Hemifield ($F(1, 5) = .59$, $p = .48$, $r = .32$). Furthermore no interaction was found between Experimental Phase and Hemifield ($F(2, 10) = .04$, $p = .97$, $r = .06$). The planned comparisons were replicated and revealed no significant differences between the latencies of saccades to the rewarded and unrewarded hemifields in the

preconditioning (t (5) = -1.09, p = >.017, r = .44), conditioning (t (5) = -.61, p = >.017, r =.26) or extinction phases (t (5) = -.27, p = >.017, r =.12).

*2.4.2.1.3 Compiled*

Given the lack of any significant differences between conditions, a joint analysis was compiled. A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Sound: With Sound/Without Sound) mixed model repeated measures ANOVA on mean SRTs revealed no effect of Experimental Phase (F (2, 20) = .63, p = .54, r = .18), Hemifield (F (1, 10) = 1.37, p = .27, r = .35) or interaction between Hemifield and Sound (F (1, 10) = .04, p =.85, r = .06) or Experimental Phase and Sound (F (2, 20) = .17, p =.84, r = .09). Furthermore, no interaction between Experimental Phase and Hemifield was revealed (F (2, 20) = .50, p =.62, r = .16). Crucially, a non-significant effect of Experimental Phase, Hemifield and Sound interaction was found (F (2, 20) = .01, p = .99, r = .02).

Planned comparisons revealed no significant differences between the latencies of saccades in the rewarded and unrewarded hemifields for the preconditioning (t (11) = .11, p = >.017, r = .03) conditioning (t (11) = -1.48, p = >.017, r = .41) and extinction (t (11) = -.90, p = >.017, r = .28) phases. Figure 2.7 illustrates these results.

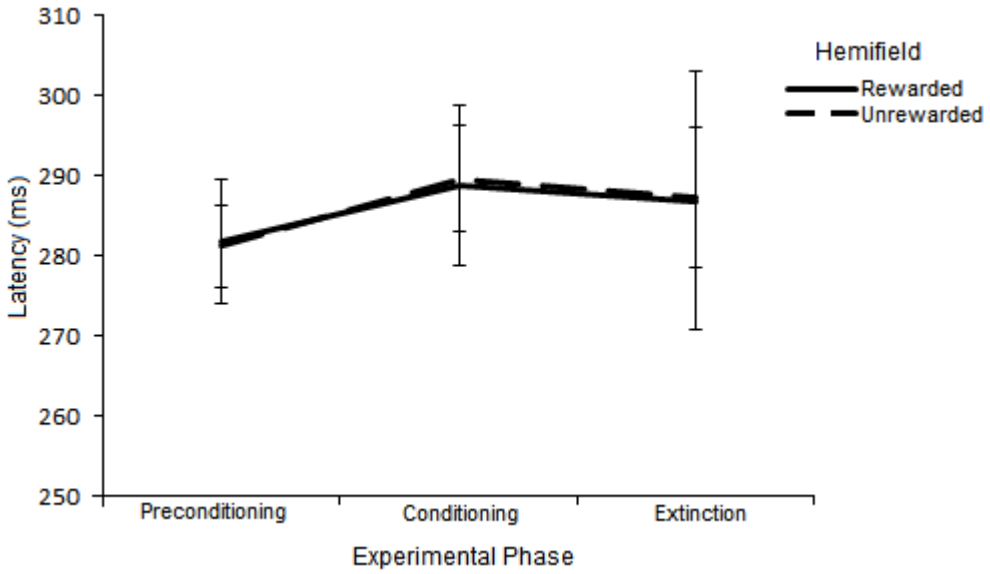


***Figure 2.7:*** *Mean saccadic latency (ms) to the rewarded (black line) and unrewarded (black dashed line) hemifields across experimental phases compiled from both the 'With Sound' and 'Without Sound' conditions. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Sound: With Sound/Without Sound) mixed model repeated measures ANOVA replicated the null effects of Experimental Phase ($F(2, 20) = 1.89$, $p = .18$, $r = .29$), Hemifield ($F(1, 10) = .23$, $p = .64$, $r = .15$) or interaction between Hemifield and Sound ($F(1, 10) = .33$, $p = .58$, $r = .18$) or Experimental Phase and Sound ($F(2, 20) = .21$, $p = .81$, $r = .10$). Furthermore, no interaction between Experimental Phase and Hemifield was revealed ($F(2, 20) = .52$, $p = .60$, $r = .16$). Crucially, the non-significant effect of Experimental Phase, Hemifield and Sound interaction was replicated ($F(2, 20) = .69$, $p = .57$, $r = .18$).

*2.4.2.2 Saccadic Error*

*2.4.2.2.1 'With Sound' during Extinction Phase*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed no effect of Hemifield ($F(1, 5) = .99$, $p = .36$, $r = .41$) or interaction between Experimental Phase and Hemifield ($F(2, 10) = 2.34$, $p = .15$, $r = .44$).

*2.4.2.2.2 'Without Sound' during Extinction Phase*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated the non-significant effect of Hemifield ($F(1, 5) = 3.16$, $p = .14$, $r = .62$) and non-significant interaction between Experimental Phase and Error Location ($F(2, 10) = 1.70$, $p = .23$, $r = .38$).

**2.4.3 Discussion**

The purpose of this investigation was to manipulate the successful pre-existing reward paradigm in order to create the strongest possible facilitation effect towards the rewarded hemifield, by pairing rewarded eye movements with an auditory tone. This experiment had two aims. Firstly, to discover whether the facilitation effect found in Experiment 2 could be replicated and strengthened. Secondly, to investigate whether a conditioned stimulus associated with reward

would lead to a greater facilitation effect to rewarded locations and longer persistence of these facilitation effects.

Contrary to the predictions made prior to the experiment, pairing an auditory tone with incentives did not enhance the facilitation effects observed in Experiment 2. Rather, no significant effects were revealed between hemifields in either the 'With Sound' and 'Without Sound' conditions. One possible explanation for these results comes from the Colavita visual dominance effect. In a natural scene, our senses are overwhelmed with a number of incoming stimuli (Calvert, Spence & Stein, 2004). To allow for coherent behaviour and efficient processing of information, attention must be coordinated across all sensory modalities (Spence & Driver, 2004). However, for a number of years there have been claims that not all senses contribute equally to our perception and that vision is the dominant sense (Posner, Nissen & Klein, 1976). This visual dominance was first demonstrated experimentally by Colavita (1974). In this study, participants were required to respond whenever they detected a light or a tone. On a small number of trials the light and tone were presented simultaneously. On these bimodal trials participants displayed a decreased ability to perceive or respond to the auditory stimulus. Participants often reported being unaware of the tone when asked after experimentation. This visual dominance effect has proven to be robust in a number of different experimental manipulations (Colavita, 1974; Colavita, Tomko & Weisberg, 1976; Colavita & Weisberg, 1979; Koppen & Spence, 2007a, 2007b, 2007c). More recent research investigating this effect has shown that visual stimuli actually have a greater capacity to capture attention exogenously than auditory stimuli (Turatto, Benso, Galfano & Umiltà, 2002). During the present experiment, participants may have prioritised the visual stimuli with very little regard for the auditory tone. Therefore, the auditory tone may have been perceived as more of a distractor than a conditioned stimulus (Parmentier & Andres, 2010) and as such disrupted saccades in the conditioning phase on trials where the 'distractor' was present. Another possible explanation of this effect may regard participants' perception of the auditory tone as an aversive stimulus, rather than a conditioned one, regardless of its presentation coinciding with a visual stimulus signalling a reward. The auditory tone may negate, and even overpower, the reward nullifying the previously found facilitation in the conditioning phase of Experiment 2. It is possible that the unrelated 1 kHz auditory tone is disrupting saccadic execution, rather than facilitating.

In summary, the findings of Experiment 3 demonstrate that rewarding oculomotor behaviour does not always produce consistent results. Saccades made during an experimental phase where rewards were paired with an auditory tone failed to replicate the previous findings in Experiment

2. No effects of reward were found in participants' errors. It is speculated that this occurred because any potential facilitation effects imposed on participants' accuracy would have been countered by the aversive auditory tone. These findings suggest that not all stimuli coupled with a reward can be integrated as conditioned stimuli. Association with a reward alone is not able to replicate facilitation effects seen when reward is presented on its own (Experiment 2).

After finding results where rewards paired with an auditory tone failed to facilitate the latencies of saccadic eye movements from a previously tested reward paradigm in which facilitation was found, a control analysis was employed where reward was removed from the experiment. Participants no longer received the reward feedback ('10p' or '0p') but the auditory tone was still present at the same frequency as those in the 'Without Sound' condition. This experiment was designed to reveal whether a difference would occur between either hemifield, even when reward is not present, and if the difference could be attributed to the presence of the auditory tone.

## 2.5 Experiment 4

### 2.5.1 Method

#### *2.5.1.1 Participants*

Six participants recruited from the University of Durham volunteered for the experiment. The participants - one male and five females – had an age range of 20-30 years (mean age 22.50 years). Five were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *2.5.1.2 Apparatus*

The experimental apparatus was the same as described in Experiment 3. However, participants were required to complete an amended consent form prior to participation (see Appendix C).

#### *2.5.1.3 Stimuli*

The experimental stimuli and setup was as described in Experiment 3.

*2.5.1.4 Procedure*

The procedure employed in Experiment 3 was replicated in the present experiment. However, rewards and reward feedback were removed entirely. Participants completed a three-phase experimental paradigm consisting of a Baseline phase (2 blocks, 240 trials), an Auditory phase (10 blocks, 600 trials) and an Extinction phase (6 blocks, 360 trials). In the Auditory phase one hemifield was designated as the feedback hemifield. Participants would hear a 1 kHz tone after making a successful saccade to a target on 30% of the trials within this hemifield. This hemifield was randomised. The other hemifield received no auditory feedback. In the extinction and baseline phases no auditory feedback was given.

*2.5.1.5 Saccade Analysis*

The analysis was conducted on the mean of each participant's SRT average calculated from each individual block. The exclusion criteria employed in Experiments 1-3 were replicated in the present experiment. In total, 1923 trials of all participant data (14.8% of the entire data set) were excluded from analysis. These analyses were once again replicated using median SRTs.

**2.5.2 Results**

A significance correction level of $p < .05$ was adopted, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*2.5.2.1 Latency*

Using mean SRTs a 3 (Experimental Phase: Baseline/Auditory/Extinction) x 2 (Hemifield: Auditory Feedback/No Auditory Feedback) repeated measures ANOVA revealed no effects of Experimental Phase ($F (2, 10) = .30$, $p = .75$, $r = .17$), Hemifield ($F (1, 5) = 4.00$, $p = .10$, $r = .67$) or an interaction between Experimental Phase and Hemifield ($F (2, 10) = .34$, $p = .72$, $r = .18$).

Planned comparisons revealed non-significant differences between the latencies of saccades in the auditory feedback and no auditory feedback hemifields for the baseline ($t (5) = .07$, $p = >.017$,

r = .03), auditory (t (5) = 1.82, p = >.017, r = .40) and extinction (t (5) = .73, p = >.017, r = .10) phases. Figure 2.8 illustrates this result.

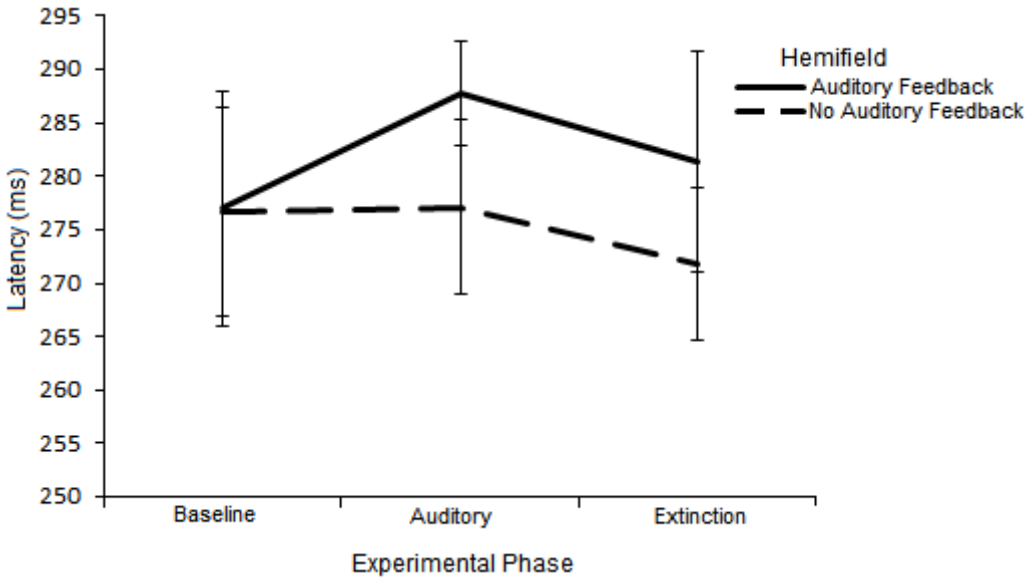


***Figure 2.8:*** *Mean saccadic latency (ms) to the auditory feedback (black line) and no auditory feedback (black dashed line) hemifields across experimental phases in the control condition. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Baseline/Auditory/Extinction) x 2 (Hemifield: Auditory Feedback/No Auditory Feedback) repeated measures ANOVA replicated the non-significant effects of Experimental Phase (F (2, 10) = .29, p = .75, r = .17), Hemifield (F (1, 5) = 3.08, p = .14, r = .62) or an interaction between Experimental Phase and Hemifield (F (2, 10) = .40, p = .68, r = .20).

Planned comparisons revealed non-significant differences between the latencies of saccades in the auditory feedback and no auditory feedback hemifields for the baseline (t (5) = -.73, p = >.017, r = .31), auditory (t (5) = -2.12, p = >.017, r = .69) and extinction (t (5) = -.16, p = >.017, r = .07) phases.

*2.5.2.2 Saccadic Error*

A 3 (Experimental Phase: Baseline/Auditory/Extinction) x 2 (Hemifield: Auditory Feedback/No Auditory Feedback) repeated measures ANOVA on the proportion of participant errors revealed

no effect of Hemifield ($F(1, 5) = <.01$, $p = .98$, $r = .40$) and no interaction between Experimental Phase and Hemifield ($F(2, 10) = 1.37$, $p = .30$, $r = .35$).

### 2.5.3 Discussion

The aim of this experiment was to elaborate on the unexpected results in Experiment 3. The present experiment isolated the auditory tone, removing reward from the successful paradigm entirely, to investigate whether the tone was the reason as to why the facilitation of rewards failed to manifest. This revised paradigm found faster saccadic latencies in the no auditory feedback hemifield during the auditory phase, although this effect was not significant. Furthermore, saccadic error analysis found a significantly larger proportion of errors in the auditory phase overall.

These findings suggest that the auditory tone did play a significant part in altering the metrics of saccades, and stand in contrast to previous findings that an auditory tone paired with reward will lead to faster latencies (Harrington & Peck, 1998; Hughes et al., 1998; Steenken et al., 2008; Corneil et al., 2002). SRTs have previously been found to be considerably faster when they are paired with an auditory stimulus. In an experiment conducted by Nakano (1997) investigating the effects of varying auditory stimulus onset asynchrony (SOA) on a visual stimulus, SRTs were fastest when the stimuli were paired. Based on the opposite being found in the present study it can be speculatively suggested that the auditory tone was attended to as an aversive stimulus, instead resulting in slower eye movements to the hemifield where this tone was present. This result further highlights the fragility of reward learning such that any effect found is abolished with the changes to the original paradigm. Future research should focus on the optimisation of reward effects varying the magnitude of reward, immediacy of the delivery and varying reward schedules. However, this thesis will continue to explore the transfer and persistence of the effects found within the reward paradigm in Experiment 2.

In summary, the findings of this study demonstrate that the auditory tone paired with a visual stimulus can be attributed to the results found in Experiment 3. Saccades made to a hemifield with no auditory tone were faster than when a tone was present. It is speculated that this occurs because participants perceive the auditory tone as aversive. It is possible that the sensitivity of the previously found facilitation effect was negated when another stimuli was added. Although this effect is potentially interesting, the primary goal of this research was to establish a reliable paradigm for using rewards to affect saccade metrics. Therefore, future chapters will use the

paradigm created in Experiment 2 to explore the effects of reward on oculomotor and attentional behaviour.

## 2.6 General Discussion

The purpose of the experiments described in this chapter was to investigate the effect of reward on saccadic behaviour using a simple saccade task. Each paradigm created was directly comparable to the other and comparable to both primate (Bowman et al., 1996; Takikawa et al., 2002; Bendiksby & Platt, 2006) and human studies (Theeuwes, 1991, 1992; Milstein & Dorris, 2007) previously conducted. Experiment 1 used a reward paradigm in which both hemifields were rewarded; one to a greater extent than the other and found no significant differences in the latencies of saccades to the higher or lower frequency rewarded hemifields. Changing the reward schedule in Experiment 2 to create a simpler reward paradigm, where one hemifield was exclusively rewarded leaving the other hemifield unrewarded, led to a facilitation effect in the direction of reward. Experiment 3 was designed to maximise the effectiveness of this reward learning, pairing an auditory tone with the rewarded visual stimulus. The facilitation effect was not replicated. A final control experiment demonstrated that the auditory tone was responsible for the negation of facilitation effects found in Experiment 2, producing slower eye movements to the hemifield where the tone was played. Although a paradigm was created in which rewards facilitated saccadic eye movements, Experiments 1 and 3 failed to replicate extensions of previous primate (Coe et al., 2002) and human (Milstein & Dorris, 2007) research.

Overall, only one reward paradigm was able to generate a facilitation effect on saccadic eye movements. Presenting rewards to one hemifield significantly decreased the latencies of saccadic eye movements to this location when compared with an unrewarded hemifield. The results of Experiment 2 are consistent with accumulator models of oculomotor control described in Chapter 1. The generation of a saccade is a competitive process where each potential saccade is vying to reach threshold. Specifically, the facilitation effect found in both experiments fits the influence of salient items on the WHERE system proposed in Findlay and Walker's (1999) model of parallel processing. Findlay and Walker (1999) suggest that the intrinsic salience of a visual item can impact on the automatic processing of that visual stimulus, and therefore directly impact on the execution threshold. In rewarding eye movements during the reward paradigm, the competitive interaction between the rewarded and the unrewarded hemifield would lead to an equilibrium shift and the ability to reach threshold for rewarded hemifield targets to be faster than those for

the unrewarded hemifield. This would explain the facilitation effect experienced. This account is also consistent with the other models outlined in Chapter 1 (e.g. Trappenberg et al., 2001; Godijn & Theeuwes, 2002). When presented with rewards to one hemifield, as occurred in the reward paradigm of Experiment 2, caudate neurons on the contralateral side to the target are very active. This excitatory activity inhibits the neurons of the ipsilateral Substantia Nigra (SNr) (Sato & Hikosaka, 2002). Decreased activity of the SNr leads to disinhibition of neurons in the Superior Colliculus (SC), thus making it easier for these neurons to reach the threshold for saccade execution (Ikeda & Hikosaka, 2003). In contrast, caudate neurons responding to the unrewarded hemifield are relatively less active. This suppression of activity leads to disinhibition of SNr neurons. Consequently the SC neurons are kept inhibited, thus reducing the likelihood of saccade execution. This hypothesis is also consistent with the activation-orienting hypothesis suggesting that lateralised visual input will produce an activation imbalance in favour of the directly stimulated hemisphere (Reuter-Lorenz, Kinsbourne & Moscovitch, 1990).

The time-course of the extinction of learning was also investigated. The reward schedule used in Experiment 1 found no significant differences between saccades during the conditioning or extinction phases. However, in Experiment 2 the facilitation effect found in the conditioning phase persisted for three blocks prior to extinction. This effect lasted for approximately ten minutes. The sensitivity and fragility of this effect is evidenced in Experiment 3. By altering the paradigm slightly to include an auditory tone coupled with the visual reward stimulus, the effects of facilitation were no longer present. Instead no significant differences between hemifields were found, but saccade latencies were longer when a reward and tone were paired. Experiment 4 demonstrated that the inclusion of an auditory tone was enough to negate the previously found facilitation effect. This result highlights the potential obstructions when dealing with the effects of reward on human behaviour. Previous research had suggested that pairing incentives with an auditory stimulus after participants made a saccade to a target stimulus would enhance the previously obtained facilitation effect (Harrington & Peck, 1998; Hughes et al., 1998; Steenken et al., 2008; Corneil et al., 2002). Instead the opposite result was revealed. Therefore, the effects of the reward paradigm appear to be fragile and can be disrupted easily (Experiment 3). In future chapters, investigation of the effects of reward discovered in Experiment 2 will be explored in secondary unrewarded tasks extending the knowledge previously held regarding the effects of reward and the persistence of these effects on the human visual system.

The data presented in this chapter suggest that instrumental conditioning can modulate saccade metrics. However, previous studies have shown that conditioning can also affect the allocation of

attention (Della Libera & Chelazzi, 2009), which is also known to affect saccade metrics (Van der Stigchel & Theeuwes, 2005; Sheliga, Riggio & Rizzolatti, 1994). Research has highlighted that the mechanisms underlying eye movements and attention are fundamentally interconnected (Goldberg & Würtz, 1972b; Kowler, Anderson, Dosher & Blaser, 1995; Gee et al., 2008) and that motor circuits support the evolution of attentional mechanisms (Rizzolatti et al., 1987; Sheliga, Riggio & Rizzolatti, 1994; Kustov & Robinson, 1996). It is therefore possible that the facilitation effect found in Experiment 2 may be accompanied by a bias in attention. Coupling a visual stimulus or a location with reward can change the salience of that stimulus (Anderson et al., 2011a) or location (Camara et al., 2013), and therefore how it is perceived. The findings of Theeuwes and Belopolsky (2012) and Camara et al., (2013) discussed in Chapter 1 provide an alternative account for the effects found in the reward experiments. In Experiment 2, presenting small monetary rewards consistently to one spatial location may have altered the way in which this location is perceived, increasing its salience relative to the hemifield where no rewards were presented. This attentional priority account can also be applied to Experiment 1 where the rewarding of both hemifields failed to produce a facilitation effect to the higher reward hemifield. As both locations were being rewarded within this task, although one to a greater extent, this may have resulted in both locations receiving equal priority.

At present it is not clear to what extent the effects of instrumental conditioning on saccade metrics were mediated by changes in spatial attention. To address this, Chapter 4 presents two experiments that were conducted to investigate the de-coupling of the locus of attention from the direction of gaze via the use of an antisaccade task. However, prior to this a pilot study was conducted in order to replicate previous well-established oculomotor paradigms.

# Chapter 3 – Antisaccade and Cueing task Pilot Experiment

## 3.1 Introduction

In order to investigate the full extent of the influence of rewards on eye movements and attention, the previously established reward paradigm used in Experiment 2 can be paired with robust eye movement tasks implicated in attention and higher cognitive functioning. However, prior to pairing the reward paradigm with these tasks, a pilot experiment was conducted in order to replicate the robust effects associated with the antisaccade task; such that antisaccades result in significantly slower SRTs than prosaccades (Jazbec et al., 2006; Ross et al., 2011, Munõz & Everling, 2004) and inhibition of return (IOR); such that stimuli presented in previously attended locations results in delayed saccadic responses (Hooge & Frens, 2000; Posner, Rafal, Choate & Vaughan, 1985; Klein, 2000; Klein & MacInnes, 1999). Previous studies have investigated the IOR effect in antisaccades, finding that antisaccades were slowest when both cue and target were congruent, with a saccade required to the opposite location (Rafal, Egly & Rhodes, 1994; Fectau, Bell & Munõz, 2004). This finding follows the principles of inhibition of return such that participants are faster when directed to a novel location, instead of a previously attended location and provides evidence that both antisaccades and prosaccades are equally affected by IOR.

The present experiment used separate blocks of prosaccades and antisaccades to investigate these effects, as employed in the work conducted by Rafal et al., (1994). After using a sample of 12 participants in the previous behavioural experiments and finding significant effects this sample size was replicated in the present experiment. The purpose of this experiment was three-fold. Firstly, this experiment was used to build a paradigm able to effectively generate an antisaccade cost, with slower antisaccades than prosaccades. Secondly, this experiment was used to build an IOR task, able to generate an effect of inhibition. These two reasons are important as these paradigms will be used separately in the thesis to investigate the influence of rewards on the oculomotor system and attention. Finally, the results of this experiment can be directly compared to previous findings that have combined these two paradigms (Rafal et al., 1994; Fecteau et al., 2004).

## 3.2 Experiment 5

### 3.2.1 Method

#### *3.2.1.1 Participants*

Twelve participants recruited from the University of Durham volunteered for the experiment (2 males, 10 females). Seven participants were right eye dominant. Ages ranged from 26-19 (mean 20.58). All participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *3.2.1.2 Apparatus*

The experimental stimuli were generated using a Cambridge Research Systems ViSaGe graphics card and displayed on a 17 inch Eizo Flexscan Colour Display monitor with a refresh rate of 100Hz. Responses were collected using a two-button button box. Eye movements were recorded using a Cambridge Research Systems eye tracker with a sampling rate of 160Hz.

#### *3.2.1.3 Stimuli*

Across the different blocks, participants were presented with the same experimental setup. A black 0.5° x 0.5° fixation cross was in the centre of the screen (0°) on a grey background with a black outlined stimulus 0.5° x 0.5° square present 8.0° to the left and right of the fixation cross. A smaller 0.3° x 0.3° white target square appeared within the larger squares.

#### *3.2.1.4 Procedure*

The pre-experimental procedure of seating and calibration of Experiments 1-4 was repeated in the present experiment. Participants had to complete two different types of block; Prosaccade and Antisaccade blocks. Participants completed each type of block three times. Each block consisted of 60 trials equally split between each type of trial. The order in which participants completed each block was randomised to negate any order effects.

In prosaccade blocks, participants completed three types of trial; Valid, Invalid and No cue. In valid trials, participants were presented with a fixation cross in the centre of the screen and two black-outlined squares to the left and right of fixation. After 700ms one of the black-outlined

squares changed colour from black to white for a brief period of time (100ms) cueing participants attention towards this location. The fixation cross then pulsated to re-orient participants attention back to the centre of the screen. After 500ms the white target square appeared at the same location as the previous colour change for 50ms. On invalid trials, the target square appeared at the opposite location of the previous colour change. In no cue trials, there was no colour change of the black outlined square and participants were required to make an eye movement towards the white target square. The experimental setup of the prosaccade blocks is illustrated in Figure 3.1.

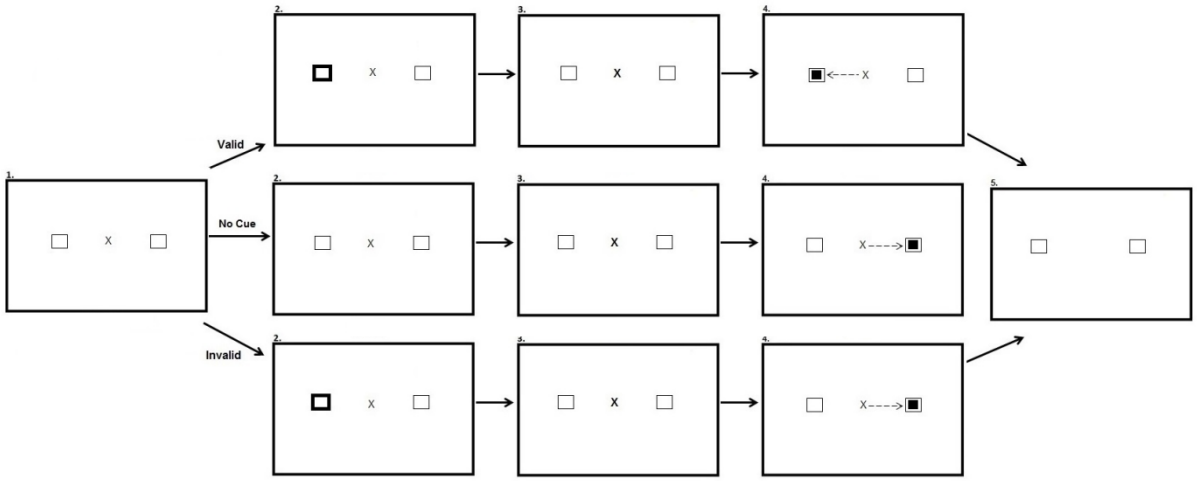


***Figure 3.1:*** *Sequence of events used in Experiment 5 for the prosaccade blocks (not to scale). Participants were presented with a fixation cross and two squares equidistant from the fixation cross in opposing hemifields (Row 1, Panel 1). In valid trials one of the squares changes colour for a period of 100ms, cueing participants to this location (Row 2, Panel 1). Participants are then presented with the same screen as in the first panel for a period of 50ms (Row 3). A smaller target square then appeared in the same location as the cue after a period of 500ms and participants were required to saccade to this location (Row 4, Panel 1). After making a successful saccade the fixation cross disappeared and the screen changed colour requiring a button press to begin the next trial (Row 5, Panel 1). In no cue trials no cue appeared prior to target onset (Row 2, Panel 2). In invalid trials the cue appeared in one location (Row 2, Panel 3) and the target appeared in the opposite location (Row 4, Panel 3).*

Antisaccade blocks consisted of the same 3 types of trial. In valid trials the target and cue location were congruent with participants required to make a saccade to the opposite hemifield. In invalid trials the target and cue location were incongruent with participants required to make a saccade

to the location where the cue had previously been. In no cue trials only the target square appeared and participants had to make an eye movement to the location opposite to where this appeared. The experimental setup of antisaccade blocks is illustrated in Figure 3.2.

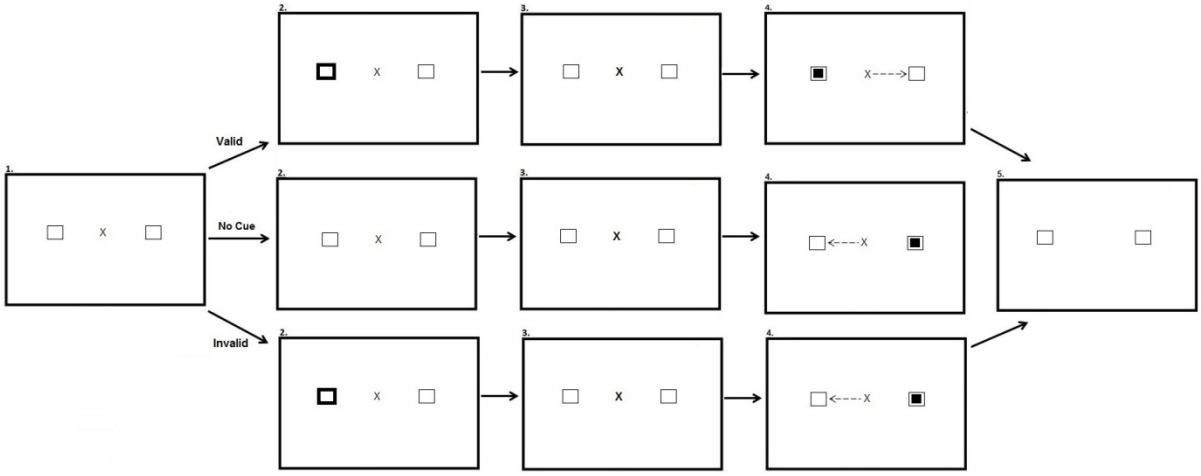


***Figure 3.2:*** *Sequence of events used in Experiment 5 for the antisaccade blocks (not to scale). Participants were presented with a fixation cross and two squares equidistant from the fixation cross in opposing hemifields (Row 1, Panel 1). In valid trials one of the squares changes colour for a period of 100ms, cueing participants to this location (Row 2, Panel 1). Participants are then presented with the same screen as in the first panel for a period of 50ms (Row 3). A smaller target square then appeared in the same location as the cue after a period of 500ms and participants were required to saccade to the opposite location (Row 4, Panel 1). After making a successful saccade the fixation cross disappeared and the screen changed colour requiring a button press to begin the next trial (Row 5, Panel 1). In no cue trials no cue appeared prior to target onset (Row 2, Panel 2). In invalid trials the cue appeared in one location (Row 2, Panel 3) and the target appeared in the opposite location requiring a saccade to the location of the cue (Row 4, Panel 3).*

*3.2.1.5 Saccade Analysis*

The analysis was conducted on the means of each participants mean SRT average calculated from each individual block. Data was filtered so that saccadic error and trials over 500 ms were eliminated from the analysis; saccadic error refers to those trials in which saccades left the fixation area but did not land at the designated target location.

Of the 6,480 trials within this task 4.68% were categorised as saccadic error and removed from the analysis. 2.99% of this error occurred on prosaccade trials. 6.36% of this error occurred on antisaccade trials.

## 3.3 Results

Inferential statistics used a significance correction level of p <.05, except when multiple comparisons were performed, where a Bonferonni correction was applied.

### *3.3.1 Latency*

A 2 (Saccade Type: Prosaccade/Antisaccade) x 3 (Validity: Valid/Invalid/No cue) repeated measures ANOVA on mean SRTs revealed a main effect of Saccade Type (F (1, 11) = 683.37, p = <.01, r = .99) such that prosaccades (260 ms) were significantly faster than antisaccades (343 ms). Figure 3.3 illustrates this result.

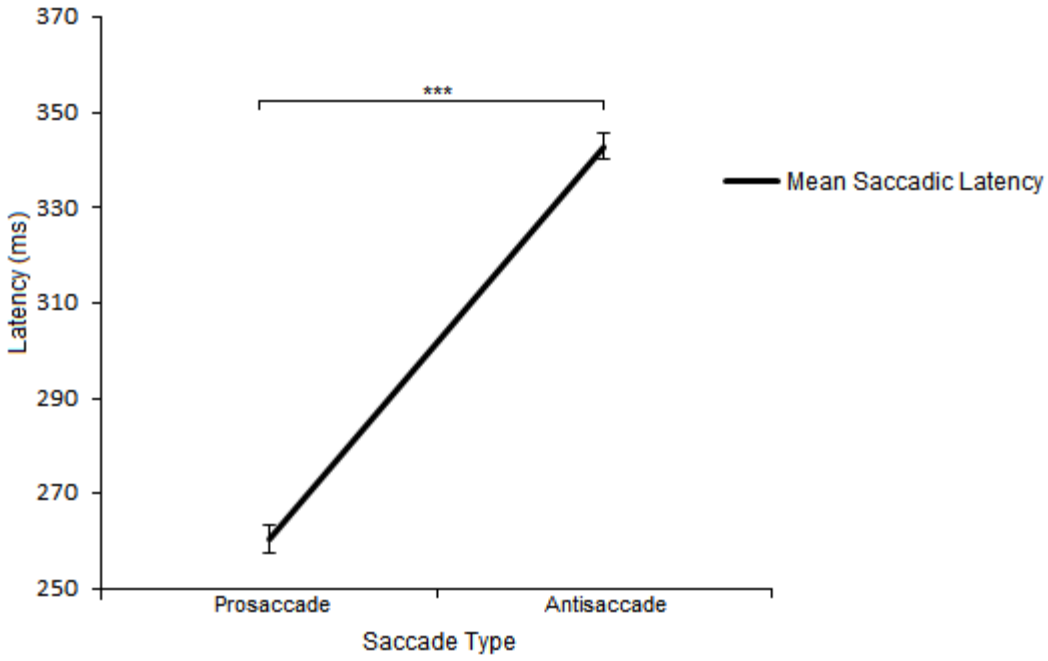


***Figure 3.3:*** *Latency of prosaccades and antisaccades. Error bars show +/- 1 SEM.*

The analysis also revealed a main effect of Validity (F (2, 22) = 16.79, p = <.01, r = .66) such that no cue trials (291 ms) were significantly faster than valid (313 ms) (t (11) = 5.96, p = <.017, r = .68) and invalid (301 ms) (t (11) = 2.58, p = <.017, r = .61) trials. Furthermore, invalid trials were significantly faster than valid trials (t (11) = 3.09, p = <.017, r = .68). Figure 3.4 illustrates these results.

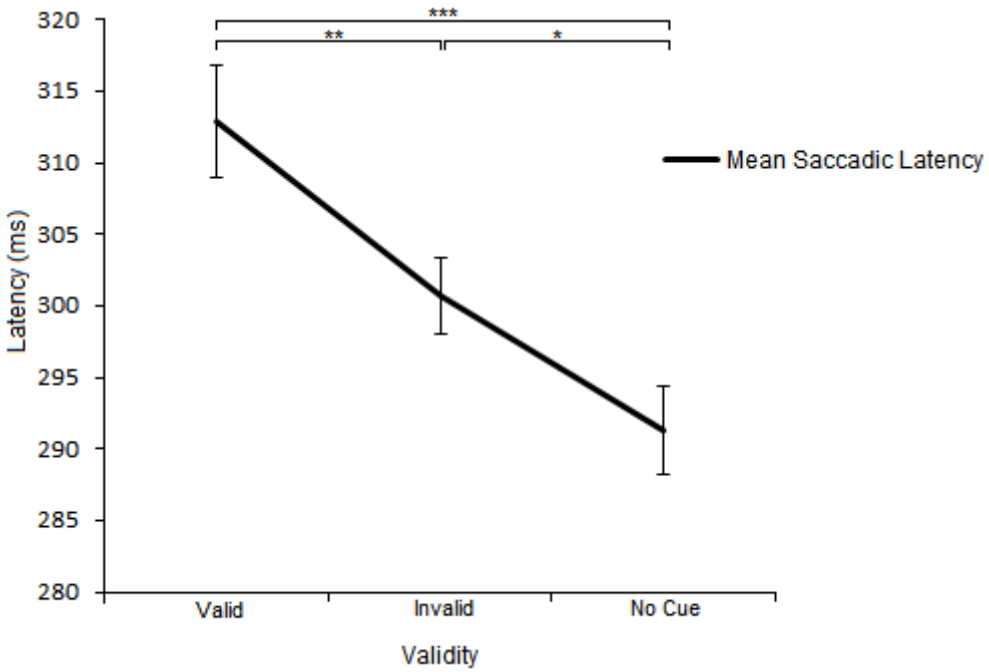


***Figure 3.4:*** *Latency of saccades across the three different types of trial validity. Error bars show +/- 1 SEM.*

These effects were subsumed in a significant interaction between Saccade Type and Validity (F (2, 22) = 5.84, p = .01, r = .46). Two separate one-way ANOVAs were conducted on the latency of pro and antisaccades at each level of validity in order to explore this two-way interaction. In prosaccades, valid trials (278 ms) were significantly slower than invalid (253 ms) (F (2, 35) = 25.35, p = <.01, r = .65) and no cue (250 ms) (F (2, 35) = 28.03, p = <.01, r = .67) trials. In antisaccades, valid trials (348 ms) were significantly slower than no cue trials (333 ms) (F (2, 35) = 15.07, p = <.01, r = .55). No cue trials were significantly faster than invalid trials (349 ms) (F (2, 35) = -15.99, p = <.01, r = .56). However, no significant differences were found between valid and invalid trials (F (2, 35) = -.92, p = 1.00, r = .16). Figure 3.5 illustrates these results.

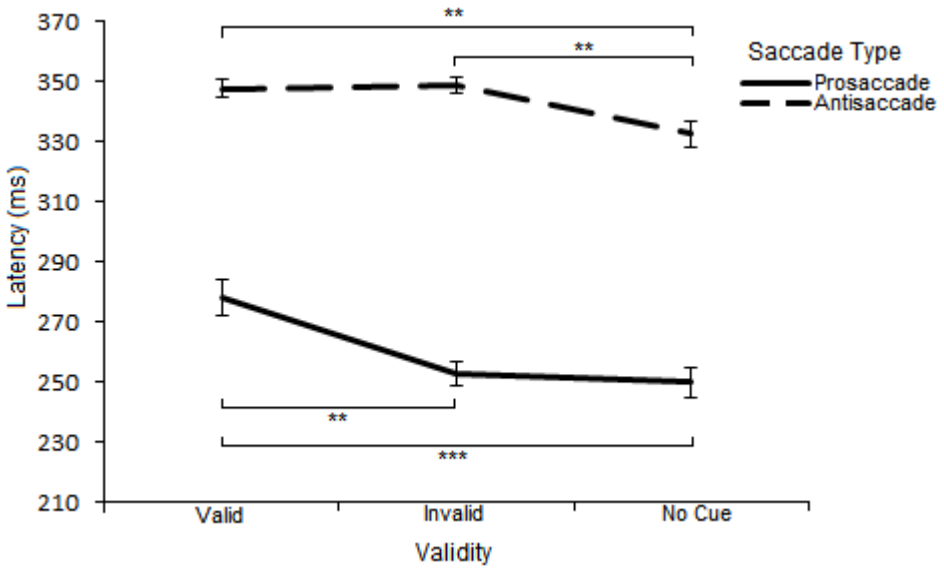


***Figure 3.5:*** *Latency of prosaccades (black line) and antisaccades (black dashed line) across the three types of trial validity. Error bars show +/- 1 SEM.*

Planned comparisons were conducted on no cue blocks to ascertain whether there was any difference between no cue trials in the different saccade types. No cue trials within the antisaccade blocks (333 ms) were significantly slower than no cue trials in the prosaccade blocks (250 ms) (t (11) = -13.16, p = <.017, r = .97). Figure 3.6 illustrates these results.

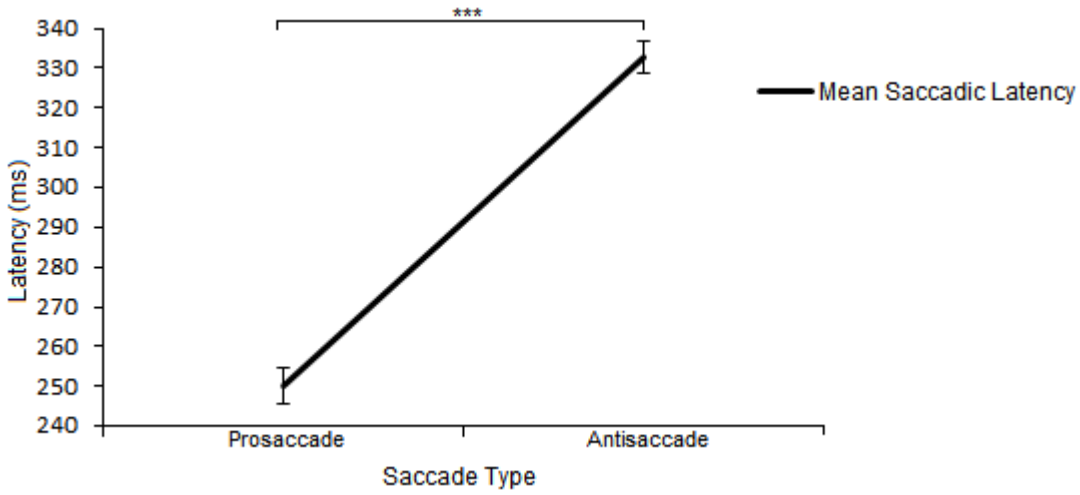


***Figure 3.6:*** *Latency of no cue trials in the prosaccade and antisaccade blocks. Error bars show +/- 1 SEM.*

### 3.4 Discussion

In summary, this pilot experiment was conducted in order to build antisaccade and inhibition of return paradigms that generate effects consistent with previous findings. An antisaccade cost was found such that prosaccades were significantly faster than antisaccades, consistent with previous research (Jazbec et al., 2006; Ross et al., 2011, Munõz & Everling, 2004). Furthermore, an effect of inhibition was found in prosaccades such that when cue and target location were congruent prosaccades were significantly slower, consistent with reports of IOR (Posner & Cohen, 1984; Posner et al., 1985; Klein, 2000; Briand, Larrison & Sereno, 2000)

Interestingly, no significant differences were found between antisaccade trials suggesting inhibition had no effect on this type of saccade within the present paradigm, contrasting previous research (Rafal et al., 1994; Fectau et al., 2004). This result contradicts Experiment 1 in Rafal et al., (1994). In this experiment both antisaccade and prosaccade SRTs were slowest when the target appeared at the cued location, as would be predicted if IOR acted only by inhibiting the detection of targets at the tagged location. Instead the results obtained from Experiment 5 are consistent with Experiment 2 in Rafal et al., (1994). This experiment was used in order to determine whether IOR generated by endogenous saccades had the same effects on antisaccades and prosaccades as IOR generated by a peripheral luminance change. In this experiment participants made a saccade to the pre-cue, returning to the centre of the screen before the appearance of the target. This experiment found that prosaccade latencies were longer when the target appeared at the cued location, compared to the uncued location, while antisaccade latencies did not differ depending on the different trial types; a result consistent with the findings of Experiment 5. The effects of the present experiment may be consistent with the explanation provided by Rafal et al., (1994) that antisaccade trials produce both perceptual IOR which slows processing of the cue and motor IOR which disrupts motor preparation in the invalid condition. One important methodological difference between the present experiment and Rafal et al's., (1994) study is the addition of no cue trials. The lack of this trial type within the experiments of Rafal et al., (1994) means it is unclear how the cue changes performance relative to baseline; a result addressed in Experiment 5. This is an important condition as it demonstrates that peripheral cues generate motor and perceptual IOR when the eye movement system is activated (see Hilchey et al., 2014).

In conclusion, this pilot experiment produced a successful antisaccade cost and effects of IOR within prosaccades. The antisaccade paradigm can therefore be used to investigate the effects of reward on reflexive and voluntary saccades, whereas the IOR paradigm can be used to investigate the effects of reward on exogenous orienting of attention and IOR in future experiments.

# Chapter 4: The effects of reward on stimulus-driven reflexive and voluntary saccades

## 4.1 Introduction

The findings in Chapter 2 have suggested that an association between reward and the oculomotor system exists, with facilitated SRTs to rewarded locations relative to unrewarded locations; an effect that persisted for a short period of time once rewards were withdrawn. The findings of incentive manipulation studies in non-human primates show that saccades to rewarded locations are initiated earlier, have faster peak velocities, and are significantly faster and more accurate, relative to saccades to unrewarded locations (Kawagoe et al., 1998; Bendiksby & Platt, 2006; Takikawa et al., 2002). More recently this relationship has been probed in human participants producing findings consistent with the primate research (Milstein & Dorris, 2007). In Chapter 2 a reward paradigm was constructed that can also produce this facilitation effect on stimulus-driven prosaccadic behaviour (Experiment 2). However, the human oculomotor system mediates both stimulus-driven and voluntary behaviour and it is not clear to what extent the effect of training stimulus-driven eye movements generalises to voluntary eye movements.

One type of eye movement that has been extensively used to investigate voluntary control in the oculomotor system is the antisaccade task, which requires the observer to suppress a stimulus-driven saccade to a sudden onset in favour of a goal-directed saccade to the mirror-symmetrical location (Hallett, 1978). This task has primarily been used as a clinical diagnostic tool for disorders which are known to involve the frontal cortex and the basal ganglia. For example, when tested using the antisaccade task, schizophrenics produce a significantly larger proportion of reflexive saccades, failing to suppress this initial eye movement and generate an antisaccade (Fukushima et al., 1990; Clementz et al., 1994; Everling et al., 1996; McDowell & Clementz, 1996) due to the implication of the frontal cortex in the generation of schizophrenia (Levy, 1996; Weinberger, Berman & Daniel, 1991). As such schizophrenics are unable to successfully perform this task. This task has also been used in the clinical diagnosis of attention deficit hyperactivity disorder (ADHD). Children with ADHD have been found to have greater difficulty in suppressing the reflexive prosaccade during the antisaccade task when compared to controls (Ross et al., 1994; Rothlind et al., 1991). The task has also been utilised in clinical studies to assess the extent to which lesions have affected processing of key structures in eye movement control. For example, Pierrot-Desilligny and colleagues (1996) tested a single patient with a lesion affecting the right SC due to a small haematoma. During the antisaccade task this patient produced a high percentage of reflexive saccades to the right hemifield, interpreted as overactivation of the left SC due to the loss of inhibition from the right SC. This finding highlights the SC as a key structure for oculomotor

control and a necessary structure in the generation of antisaccades. Further use of the antisaccade task comes from its use in the diagnosis of Huntingdon's disease. This disease is characterised by degenerations in the caudate and the substantia nigra pars reticulata (two key structures of the basal ganglia) which leads to deficits in the generation of antisaccades and in the suppression of reflexive prosaccades during the antisaccade task (Lasker et al., 1987; Rothlind et al., 1993). The clinical relevance of the antisaccade task makes it an interesting task to use with the reward paradigm in order to ascertain the effects of rewards on different types of eye movements.

The behavioural effect of incentives on antisaccades has been investigated in both adults (Duka & Lupp, 1997) and adolescents (Jazbec et al., 2006) finding that incentives increase the accuracy of antisaccades. One study has analysed the effects of both positive and negative reinforcement on pro and antisaccades (Ross, Lanyon, Viswanathan, Manoach & Barton, 2011). Participants were presented with a motivational cue indicating a reward, penalty or no consequence (neutral), following which a circular target stimulus would appear. The use of motivational cues was found to reduce the saccadic latency of both prosaccades and antisaccades, more prominently for reward than penalty cues. An interesting result was the difference between penalty and reward cues; the threat of a penalty created more variable effects than the reinforcement of a reward, producing results at times similar to reward trials and at other times similar to neutral trials. The response of the saccadic system to reward was consistent across different saccade types. This effect is interesting in regards to the varying effects of rewards and penalties. From these results, the consistent effects of positive reinforcement, relative to the inconsistent effects when a penalty was employed, suggests that rewards may produce more consistent effects of behaviour change. However, it is important to note that these studies examined the effect of reward on saccades per se, rather than investigating the effect of rewarding a specific spatial location. As a consequence, these data are not directly comparable to the studies of non-human primates (Bendiksby & Platt, 2006; Coe et al., 2002).

To summarise, work with non-human primates suggests that rewarding spatial locations can create a bias in the oculomotor system, such that eye movements are executed more quickly and accurately to rewarded locations, a finding that extends to antisaccades (Ross et al., 2011; Duka & Lupp, 1997; Jazbec et al., 2006). Experiment 2 generated a reward paradigm able to produce a facilitation effect to a single spatial location using monetary incentives. Presently there is little understanding of the transfer of the effects of this paradigm into other unrewarded eye movement tasks. To address this issue a task was developed exploring the effect of a previously

established reward association in an unrewarded task combining both prosaccades and antisaccades, in an attempt to examine whether the effects of monetary incentives generalise to a secondary, unrewarded eye movement task. This task was named the post-reward paradigm eye movement task (PRPEM). Thus the current experiment allowed the study of both the transfer of learning between different tasks and the time-course of extinction effects. Prior to the coupling of the reward paradigm and the PRPEM task, a pilot antisaccade task was employed (see Chapter 3) in order to ensure this task generated results consistent with those previously found in a number of other experiments (Jazbec et al., 2006; Ross et al., 2011, Munõz & Everling, 2004). Using the previously established reward paradigm (Experiment 2) coupled with the PRPEM task, the effects of reward, if any, on attention and behaviour can be established. The experiments within this chapter aim to investigate: 1) whether the facilitation effect found in Experiment 2 can be replicated; 2) the transfer of learning between tasks with the inclusion of an unrewarded post-conditioning eye movement task (PRPEM), combining trials of prosaccades and antisaccades; 3) the time-course of extinction effects when a secondary unrelated eye movement task is introduced. The hypothesis of the present investigation was that in the reward paradigm participants would produce significantly faster saccades to the rewarded hemifield than the unrewarded hemifield, consistent with the use of the reward paradigm in Experiment 2. In the PRPEM task it was predicted that the facilitation effect would transfer to the trained eye movement (prosaccade) and not the untrained eye movement (antisaccade).

## 4.2 Experiment 6

### 4.2.1 General Method

#### *4.2.1.1 Participants*

The sample size of twelve participants used in the previous experiments was replicated. Twelve participants, recruited from the University of Durham, volunteered for the experiment. The participants - one male, eleven females – had an age range of 20-31 years (mean age 23.67 years). Seven were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

*4.2.1.2 Apparatus*

Participants were required to complete a consent form prior to participation (see Appendix D). The experimental stimuli were generated using a Cambridge Research Systems ViSaGe graphics card and displayed on a 17 inch Eizo Flexscan Colour Display monitor with a refresh rate of 100Hz. Responses were collected using a two-button button box. Eye movements were recorded using a Cambridge Research Systems eye tracker with a sampling rate of 160Hz.

*4.2.1.3 Stimuli*

The reward paradigm stimuli employed in Experiment 2 was replicated.

During the PRPEM task participants were presented with a 0.7° x 0.7° fixation cross in the centre of the screen on a grey background. A target stimulus 0.8° x 0.7° square was presented to the left or right of the fixation cross. The stimuli were presented 1.4° to the left and 1.3° upwards from fixation.

*4.2.1.4 Procedure*

The experimental procedure eye dominance tests and calibration employed in Experiment 2 was replicated.

*4.2.1.4.1 Reward Paradigm*

The reward paradigm used in Experiment 2 was replicated.

*4.2.1.4.2 PRPEM Task*

The PRPEM task was run for 6 blocks and consisted of two experimental phases; 1) the Post-Conditioning phase, which ran directly after the conditioning phase of the reward paradigm; 2) the Post-Extinction phase, which ran directly after the extinction phase of the reward paradigm. Figure 4.1 illustrates the experimental array. Each block contained 60 trials evenly split between randomised left antisaccade, right antisaccade, left prosaccade and right prosaccade trials. Participants were instructed to fixate on the central fixation cross prior to the start of each trial. A

blue fixation cross corresponded to a prosaccade trial, whereas a purple cross corresponded to an antisaccade trial. After a successful saccade, the target stimuli would change colour from white to black and the trial ended. A button press was required to start the next trial.

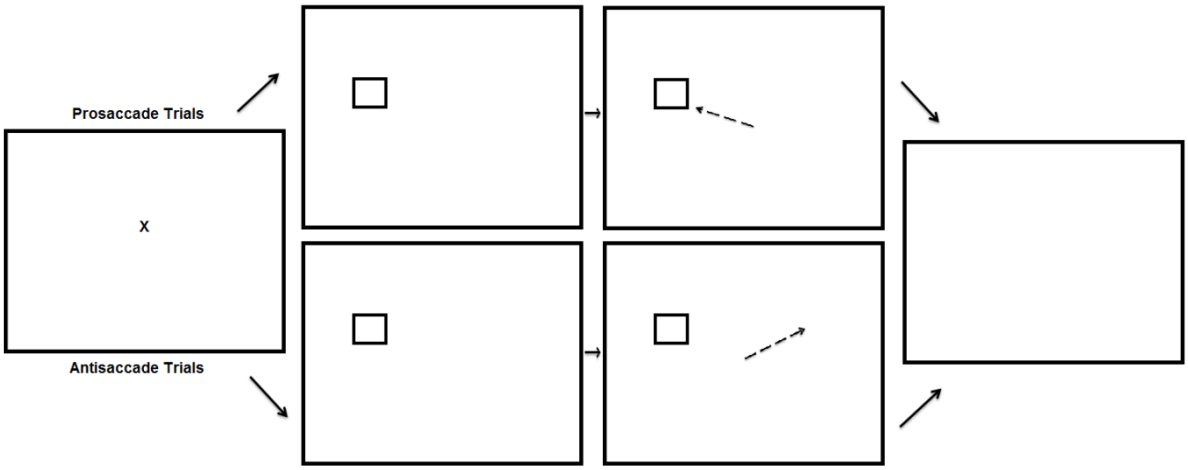


***Figure 4.1:*** *Sequence of events used in Experiment 6 and 7 for the PRPEM task (not to scale). First a fixation cross appeared (first panel). The fixation cross was either blue or purple to inform the participant of whether the trial was a prosaccade or antisaccade trial respectively. Secondly, the fixation cross disappeared and a target square appeared in either the left or right hemifield (second panel). On a prosaccade trial, participants were required to saccade towards the square. On an antisaccade trial participants were required to saccade to the hemifield opposite the square (third panel). After making a saccade participants were presented with a blank screen informing them that the trial was over. A button press was required to begin the next trial.*

The full experiment ran for 30 blocks and lasted around 1 hour. Participants switched between blocks of the two eye movement tasks. Firstly, participants completed the preconditioning and conditioning phases of the reward paradigm, then six blocks of the PRPEM, then the extinction phase of the reward paradigm and a final six blocks of the PRPEM.

*4.2.1.5 Saccade Analysis*

Mean SRTs were calculated for each individual block within an experimental phase. The analysis was then conducted on the mean of these means. Data was filtered so that saccadic error and

trials over 500ms were eliminated from the analysis; saccadic error refers to those trials in which saccades left the fixation area but did not land at the designated target location. These analyses were once again replicated using median SRTs.

*4.2.1.5.1 Reward Paradigm*

Across 12,960 trials, 6.6% were categorised as saccadic errors. 24.7% of trials were above the threshold and also removed from the analysis. In total, 31.3% of conditioning trials were rejected from the analysis.

*4.2.1.5.2 PRPEM Task*

Of the 4,320 prosaccade trials within this experimental phase 19.61% were categorised as saccadic errors and 22.78% of trials were above the threshold. Of the 4,320 antisaccade trials 37.15% were identified as inaccurate and 6.18% were found to be above the threshold and so removed from the analysis.

**4.2.2 Results**

Inferential statistics used a significance correction level of $p < .05$, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*4.2.2.1 Latency*

*4.2.2.1.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed a main effect of Experimental Phase ($F (2, 22) = 10.42$, $p = <.01$, $r = .57$) such that latencies of saccades in the conditioning phase (260 ms) were significantly faster than those in either the preconditioning (291 ms) ($t (11) = 3.95$, $p = <.017$, $r = .77$) or extinction (279 ms) ($t (11) = -2.39$, $p = <.017$, $r = .58$) phases of the experiment. No effect of Hemifield ($F (1, 11) = 2.50$, $p = .14$, $r = .43$) or interaction between Experimental Phase and Hemifield was found ($F (2, 22) = 1.01$, $p = .38$, $r = .21$).

Planned comparisons revealed no significant difference between the latencies of saccades to either hemifield in the preconditioning phase (t (11) = -.09, p = >.017, r = .03). In contrast, a significant difference was found between the latencies of saccades to the rewarded (243 ms) and unrewarded (278 ms) hemifields in the conditioning phase (t (11) = -2.40, p= .04, r = .59), such that participants produced significantly faster SRTs towards the rewarded hemifield. However this did not survive the correction level for multiple comparisons. After the removal of reward, a non-significant difference between the saccadic latencies in the rewarded (272 ms) and unrewarded (286 ms) hemifields for the extinction phase was found (t (11) = -.91, p= >.017, r = .26). Figure 4.2 illustrates these results.

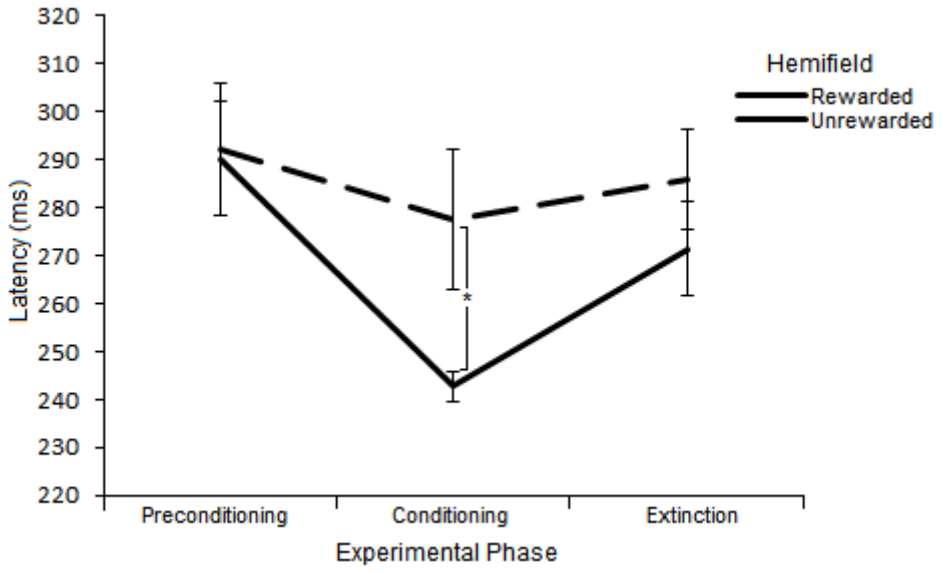


***Figure 4.2:*** *Latency of prosaccades to the rewarded (black line) and unrewarded (black dashed line) hemifields in Experiment 6 across the preconditioning, conditioning and extinction phases of the reward paradigm. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed a main effect of Hemifield (F (1, 11) = 5.40, p = .04, r = .57) such that saccades made to the rewarded hemifield (267 ms) were significantly faster than those made to the unrewarded hemifield (283 ms). Furthermore, a significant interaction between Experimental Phase and Hemifield was found (F (2, 22) = 7.31, p = <.01, r = .50).

Planned comparisons revealed no significant difference between the latencies of saccades to either hemifield in the preconditioning phase (t (11) = -.41, p = >.017, r = .12). In contrast, a significant difference was found between the latencies of saccades to the rewarded (248 ms) and unrewarded (288 ms) hemifields in the conditioning phase (t (11) = -3.17, p= <.017, r = .69), such that participants produced significantly faster SRTs towards the rewarded hemifield. After the removal of reward, a non-significant difference between the saccadic latencies in the rewarded (277 ms) and unrewarded (279 ms) hemifields for the extinction phase was found (t (11) = -.28, p= >.017, r = .08).

*4.2.2.1.2 PRPEM Task*

A 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed a main effect of Saccade Type (F (1, 11) = 201.4, p = <.01, r = .97) such that prosaccades (209 ms) were significantly faster than antisaccades (274 ms). No effect of Experimental Phase (F (1, 11) = .54, p = .48, r = .22) or Hemifield (F (1, 11) = .52, p = .49, r = .21) was found. No significant interactions between Experimental Phase and Saccade Type (F (1, 11) = .14, p = .71, r = .11), Experimental Phase and Hemifield (F (1, 11) = 1.04, p = .33, r = .29), Saccade Type and Hemifield (F (1, 11) = .93, p = .36, r = .28) or Experimental Phase, Saccade Type and Hemifield (F (1, 11) = .07, p = .79, r = .08) were found.

Using median SRTs a 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs replicated the main effect of Saccade Type (F (1, 11) = 185.08, p = <.01, r = .97) such that prosaccades (208 ms) were significantly faster than antisaccades (273 ms). No effect of Experimental Phase (F (1, 11) = .40, p = .54, r = .19) or Hemifield (F (1, 11) = 1.01, p = .34, r = .29) was found.

No significant interactions between Experimental Phase and Saccade Type (F (1, 11) = .01, p = .99, r = .01), Experimental Phase and Hemifield (F (1, 11) = .71, p = .42, r = .25), Saccade Type and Hemifield (F (1, 11) = 1.20, p = .30, r = .31) or Experimental Phase, Saccade Type and Hemifield (F (1, 11) = 1.63, p = .23, r = .36) were found.

*4.2.2.1.3 Time-course of the Extinction*

Based on the findings of Experiment 2, such that the time-course of the reward learning persisted for a period of three blocks, the first three blocks of data obtained from the Post-Conditioning phase of the PRPEM task were compiled into one block named Group 1. The final three blocks of the PRPEM task were compiled into a second group named Group 2. This data was then analysed in order to ascertain whether any transfer of the effects of reward was observed within the PRPEM task.

Using mean SRTs a 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Group: Group 1/Group 2) repeated measures ANOVA revealed a main effect of Saccade Type ($F (1, 11) = 185.49$, $p = <.01$, $r = .97$) such that antisaccades (275 ms) were significantly slower than prosaccades (210 ms). No significant effect of Hemifield ($F (1, 11) = .05$, $p = .83$, $r = .06$), Group ($F (1, 11) = .02$, $p = .90$, $r = .04$), or interaction between Saccade Type and Hemifield ($F (1, 11) = .91$, $p = .36$, $r = .28$), Saccade Type and Group ($F (1, 11) = .45$, $p = .52$, $r = .20$) Hemifield and Group ($F (1, 11) = 1.80$, $p = .21$, $r = .37$) or Saccade Type, Hemifield and Group ($F (1, 11) = .05$, $p = .83$, $r = .07$) was revealed.

Using median SRTs a 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Group: Group 1/Group 2) repeated measures ANOVA replicated a main effect of Saccade Type ($F (1, 11) = 200.76$, $p = <.01$, $r = .97$) such that antisaccades (273 ms) were significantly slower than prosaccades (208 ms). No significant effect of Hemifield ($F (1, 11) = .07$, $p = .79$, $r = .01$), Group ($F (1, 11) = .80$, $p = .39$, $r = .26$), or interaction between Saccade Type and Hemifield ($F (1, 11) = .45$, $p = .52$, $r = .20$), Hemifield and Group ($F (1, 11) = 3.02$, $p = .11$, $r = .46$) or Saccade Type, Hemifield and Group ($F (1, 11) = .48$, $p = .50$, $r = .21$) was revealed. Interestingly a significant interaction was revealed between Saccade Type and Group ($F (1, 11) = 9.03$, $p = .01$, $r = .67$). Using paired t tests this effect was explored. Significantly faster prosaccades were found in Group 1 (205 ms) compared to Group 2 (211 ms) ($t (1, 11) = 2.41$, $p = .03$, $r = .59$), however this did not survive the correction for multiple comparisons. No differences were found between antisaccades in Group 1 (272 ms) and Group 2 (274 ms) ($t (1, 11) = -.92$, $p = .38$, $r = .27$). Figure 4.3 illustrates this result. This finding suggests a more general effect of reward speeding the trained eye movement in the first three blocks of the PRPEM task.

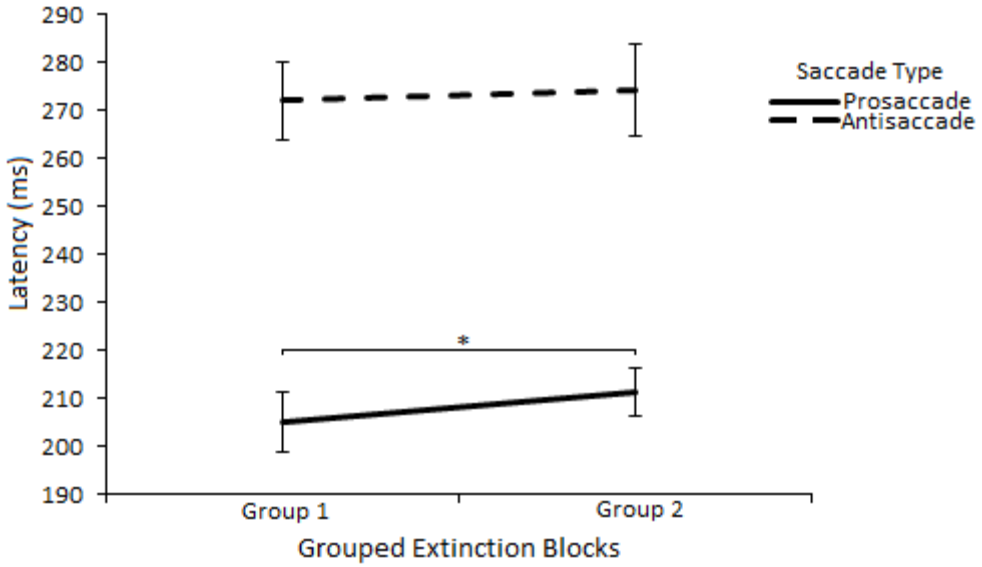


***Figure 4.3:*** *Latency of prosaccades (black line) and antisaccades (dashed line) in the first three blocks of the extinction phase (Group 1) and the second three blocks of the extinction phase (Group 2). Error bars show +/- 1 SEM.*

*4.2.2.2 Saccadic Error*

For the purpose of this error analysis, saccadic errors previously excluded from the latency analysis were included. Trials above the set threshold were still excluded.

*4.2.2.2.1 Reward paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on the proportion of participant errors during the reward paradigm revealed no effect of Hemifield ($F(1, 11) = 2.10$, $p = .18$, $r = .40$), or interaction between Phase and Hemifield ($F(2, 22) = 2.45$, $p = .11$, $r = .32$). These results suggest that reward does not have any effect on participant accuracy.

*4.2.2.2.2 PRPEM Task*

A 2 (Saccade Type: Prosaccade/Antisaccade) x 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on

the proportion of errors within the PRPEM task. This analysis revealed a significant effect of Saccade Type ($F(1, 11) = 9.70$, $p = .01$, $r = .68$) such that a significantly larger proportion of errors occurred in antisaccade trials than prosaccade trials. No other significant effects or interactions were recorded. The lack of significant effects suggests that participant accuracy was not modulated by rewards.

Using antisaccade trial errors, where a prosaccade was made before being corrected to produce a correct antisaccade trial planned comparisons were conducted. A 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the proportion of errors within this trial type. This analysis revealed a main effect of Hemifield ($F(1, 11) = 5.10$, $p = .05$, $r = .56$) with a significantly larger proportion of errors towards the rewarded hemifield (.023) relative to the unrewarded hemifield (.019). No interaction was revealed between Experimental Phase and Hemifield ($F(1, 11) = 1.35$, $p = .27$, $r = .33$).

#### 4.2.3 Discussion

The goals of this experiment were to examine the effect of rewards on the latency of prosaccades, to establish the duration of these effects, and to investigate to what extent the effects of rewarding prosaccades generalised to other types of eye movement.

Consistent with most previous research in human and non-human primates, it was observed that saccades to a rewarded location were relatively facilitated compared to those made to an unrewarded location (Takikawa et al., 2002; Coe et al., 2002; Bowman et al., 1996; Milstein & Dorris, 2007). With respect to the second and third goals of the study, the results extend previous findings in two important ways. Firstly, the effects of facilitation to the rewarded location transferred to the prosaccade data in the PRPEM task only when using median SRTs. Median SRTs are a better This finding illustrates the sensitivity of the facilitation effect. Once rewards are withdrawn, the effects of reward learning fail to transfer consistently to the same type of eye movement in a different task. Secondly, not only do the effects of reward fail to persist convinvingly in the trained eye movement once rewards are withdrawn, the finding that the latencies of antisaccades were not affected by the reward suggests that effects of reward also fail to generalise to untrained eye movements. This finding is noteworthy as it introduces an important constraint for the use of reward-based paradigms in clinical settings. For example, only the specific behaviour being trained seems to be modulated by rewards. Once the task is changed, this behaviour fails to persist into an untrained eye movement task. Therefore, the

context in which reward learning occurs is an important consideration when using rewards to train behaviour.

In the PRPEM task a larger proportion of errors in antisaccades was found, compared to prosaccades, consistent with previous research (Kristjánsson, Vandenbroucke & Driver, 2004; Ross et al., 2011). Importantly, the proportion of antisaccade errors in the PRPEM task was not modulated by the presence of a reward. However, analysis of the initial incorrect prosaccade made in corrected antisaccade trials revealed a larger proportion of saccades were directed towards the rewarded hemifield prior to being corrected. This finding provides evidence that facilitation of participant's reflexive behaviour occurred to the location of reward prior to participants subsequently generating the correct response. This result suggests that rewards modulate the initial, stimulus-driven, automatic, orienting of attention. Although participants were cued to make an antisaccade, the presence of a target in the rewarded hemifield resulted in an incorrect prosaccade towards this hemifield. Rewards have modulated spatial attention to this location, resulting in greater attentional capture for targets presented in this hemifield. This result is consistent with data suggesting that incentives result in attentional priority to stimulus features associated with reward (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al., 2010a; Theeuwes, 1991, 1992; Theeuwes & Belopolsky, 2012). Different interpretations of these data are considered with respect to models of attention and oculomotor controls in the general discussion.

The results from Experiment 6 confirm that the human oculomotor system is susceptible to instrumental conditioning, and demonstrates that this hemifield-specific effect of SRT facilitation fails to transfer beyond the trained task convincingly. However, an effect of reward was found in participants corrected antisaccades. This finding further highlights the transient persistence of these effects such that although an effect of facilitation of SRTs were abolished once the task was changed and rewards were removed, the effect persisted into the accuracy of participants corrected antisaccades when previously it had not been found. This finding further compounds the inconsistency of the reward effects. However, one caveat with this interpretation is that the latency of eye movements in the PRPEM task was not measured prior to the introduction of the reward. To address this issue Experiment 6 was replicated with the addition of a block of PRPEM trials prior to the onset of the conditioning trials.

## 4.3 Experiment 7

### 4.3.1. Method

#### *4.3.1.1 Participants*

The sample size of twelve participants used in the previous experiments was replicated. Twelve participants, recruited from the University of Durham, volunteered for the experiment. The participants - one male, eleven females – had an age range of 18-21 years (mean 19.25). Eleven were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *4.3.1.2 Apparatus*

The experimental stimuli and setup was as described in Experiment 6.

#### *4.3.1.3 Procedure*

The procedure employed in Experiment 6 was replicated in the present experiment with the addition of a further six blocks of the PRPEM task after the preconditioning phase of the reward paradigm. This additional phase was categorised as the Post-Preconditioning Phase.

#### *4.3.1.4 Saccade Analysis*

The saccade analysis undertaken in Experiment 6 was replicated.

##### *4.3.1.4.1 Reward Paradigm*

Across 12,960 saccadic trials, 6.3% were categorised as saccadic errors. 28.4% of trials were above the threshold and also removed from the analysis. In total, 34.8% of conditioning trials were rejected from the analysis.

*4.3.1.4.2 PRPEM Task*

Of the 6,480 prosaccade trials within this experimental phase 15.9% were categorised as saccadic errors. 5.56% of trials were above the threshold. Of the 6,480 antisaccade trials 27.48% were categorised as saccadic errors and 3.53% were found to be above the threshold and so removed from the analysis.

**4.3.2 Results**

Inferential statistics used a significance correction level of p <.05, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*4.3.2.1 Latency*

*4.3.2.1.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed no effect of Experimental Phase ($F(2, 22) = 2.18$, $p = .14$, $r = .30$), Hemifield ($F(1, 11) = 4.01$, $p = .07$, $r = .52$) or interaction between Experimental Phase and Hemifield ($F(2, 22) = 1.63$, $p = .22$, $r = .26$).

Planned comparisons revealed no significant difference between the latencies of saccades to either hemifield in the preconditioning phase ($t(11) = -.29$, $p = >.017$, $r = .09$). In contrast, a significant difference was found between the latencies of saccades to the rewarded (240 ms) and unrewarded (264 ms) hemifields in the conditioning phase ($t(11) = -4.15$, $p = <.01$, $r = .78$), such that participants produced significantly faster SRTs towards the rewarded hemifield. After rewards were withdrawn a non-significant difference between the saccadic latencies in the rewarded (262 ms) and unrewarded (267 ms) hemifields for the extinction phase was found ($t(11) = -.38$, $p = >.017$, $r = .11$). Figure 4.4 illustrates these results. This replicates the finding in Experiment 6 and evidences the consistent effect of this particular reward schedule.

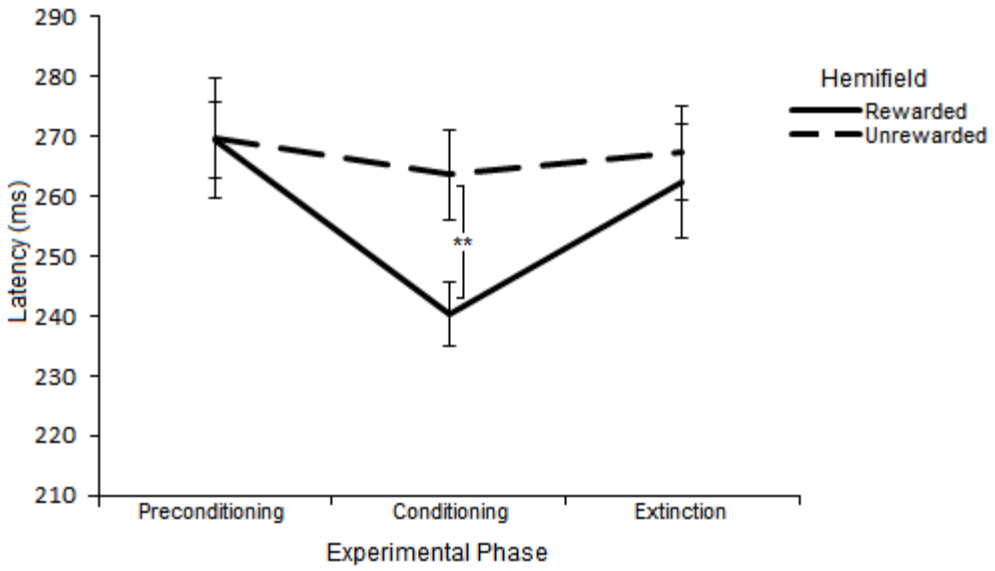


***Figure 4.4:*** *Latency of prosaccades to the rewarded (black line) and unrewarded (black dashed line) hemifields in Experiment 7 across the preconditioning, conditioning and extinction phases of the reward paradigm. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated the null effect of Experimental Phase ($F(2, 22) = 1.74$, $p = .20$, $r = .27$). However a main effect of Hemifield ($F(1, 11) = 7.04$, $p = .02$, $r = .62$) was revealed such that saccades made to the rewarded hemifield (258 ms) were significantly faster than those made to the unrewarded hemifield (265 ms). Furthermore, an interaction between Experimental Phase and Hemifield was found ($F(2, 22) = 3.81$, $p = .04$, $r = .38$).

Further paired t tests explored this interaction revealing no significant difference between the latencies of saccades to either hemifield in the preconditioning phase ($t(11) = -.04$, $p = >.017$, $r = .01$). In contrast, a significant difference was found between the latencies of saccades to the rewarded (238 ms) and unrewarded (265 ms) hemifields in the conditioning phase ($t(11) = -3.74$, $p = <.017$, $r = .75$), such that participants produced significantly faster SRTs towards the rewarded hemifield. After rewards were withdrawn a non-significant difference between the saccadic latencies for the extinction phase was found ($t(11) = -.66$, $p = >.017$, $r = .19$). This replicates the findings of the mean SRT analysis and the results found in Experiment 6 evidencing the consistent effect of this particular reward schedule.

*4.3.2.1.2 PRPEM Task*

A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean participant SRTs revealed a main effect of Saccade ($F (1, 11) = 21.30$, $p = <.01$, $r = .81$) with prosaccades (240 ms) being significantly faster than antisaccades (290 ms) throughout the PRPEM tasks. Consistent with the mean SRT findings of Experiment 6, no main effect of Hemifield ($F (1, 11) = 3.71$, $p = .08$, $r = .50$) or Experimental Phase ($F (2, 22) = .18$, $p = .84$, $r = .09$) were found. Furthermore, no interaction between Experimental Phase and Saccade Type ($F (2, 22) = .69$, $p = .51$, $r = .17$), Saccade Type and Hemifield ($F (1, 11) = 1.35$, $p = .27$, $r = .33$), Experimental Phase and Hemifield ($F (2, 22) = 1.59$, $p = .23$, $r = .26$) or three-way interaction between Experimental Phase, Saccade Type and Hemifield ($F (2, 22) = 1.02$, $p = .38$, $r = .21$) was revealed.

Using median SRTs a 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated the main effect of Saccade ($F (1, 11) = 7.66$, $p = .02$, $r = .64$) with prosaccades (253 ms) being significantly faster than antisaccades (285 ms) throughout the PRPEM task. No main effect of Hemifield ($F (1, 11) = 1.05$, $p = .33$, $r = .30$) or Experimental Phase ($F (2, 22) = .65$, $p = .53$, $r = .17$) were found. Furthermore, no interaction between Experimental Phase and Saccade Type ($F (2, 22) = .13$, $p = .88$, $r = .08$), Saccade Type and Hemifield ($F (1, 11) = .43$, $p = .53$, $r = .19$), Experimental Phase and Hemifield ($F (2, 22) = .34$, $p = .72$, $r = .12$) or three-way interaction between Experimental Phase, Saccade Type and Hemifield ($F (2, 22) = .94$, $p = .41$, $r = .20$) was revealed.

*4.3.2.1.3 Time-course of the Extinction*

As with Experiment 6, the time-course of the extinction was analysed. A 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Group: Group 1/Group 2) repeated measures ANOVA revealed a main effect of Saccade Type ($F (1, 11) = 29.17$, $p = <.01$, $r = .85$) such that antisaccades were significantly slower than prosaccades. No significant effect of Hemifield ($F (1, 11) = .08$, $p = .79$, $r = .08$), Group ($F (1, 11) = 1.30$, $p = .28$, $r = .33$), or interaction between Saccade Type and Hemifield ($F (1, 11) = 2.23$, $p = .16$, $r = .41$), Saccade Type and Group ($F (1, 11) = .07$, $p = .80$, $r = .08$), Hemifield and Group ($F (1, 11) = .65$, $p = .44$, $r = .24$) or Saccade Type, Hemifield and Group ($F (1, 11) = 1.33$, $p = .27$, $r = .33$) was revealed.

Using median SRTs a 2 (Saccade Type: Antisaccade/Prosaccade) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated the main effect of Saccade Type ($F(1, 11) = 17.80$, $p = <.01$, $r = .79$) such that antisaccades were significantly slower than prosaccades. No significant effect of Hemifield ($F(1, 11) = .74$, $p = .41$, $r = .25$), Group ($F(1, 11) = 1.56$, $p = .24$, $r = .35$), or interaction between Saccade Type and Hemifield ($F(1, 11) = 1.06$, $p = .33$, $r = .30$), Hemifield and Group ($F(1, 11) = .30$, $p = .60$, $r = .16$) or Saccade Type, Hemifield and Group ($F(1, 11) = 1.89$, $p = .20$, $r = .38$) was revealed. Interestingly, the previous interaction between Saccade Type and Group failed to replicate ($F(1, 11) = 1.89$, $p = .20$, $r = .38$). This suggests that the effects of reward are trainsient and fail to transfer consistently.

#### *4.3.2.2 Saccadic Error*

As with Experiment 6, inaccurate trials previously excluded from the latency analysis were included. This error analysis was run exclusively using only inaccurate trials. Trials above the set threshold were still excluded.

##### *4.3.2.2.1 Reward paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on the proportion of participant error during the reward paradigm revealed no effect of Hemifield ($F(1, 11) = .95$, $p = .35$, $r = .28$) or interaction between Experimental Phase and Hemifield ($F(2, 22) = 1.33$, $p = .29$, $r = .24$). These results confirm the results of Experiment 6 and suggest that incentivising one hemifield had no effect on the accuracy of participants' saccadic eye movements.

##### *4.3.2.2.2 PRPEM Task*

A 2 (Saccade Type: Prosaccade/Antisaccade) x 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the proportion of participant errors within the PRPEM task. A significant effect of Saccade Type was found ($F(1, 11) = 13.64$, $p = <.01$, $r = .74$) where antisaccades were responsible for a significantly larger amount of the proportion of error than prosaccades,

consistent with previous research (Kristjánsson et al., 2004; Ross et al., 2011). No other significant interactions were found replicating the data obtained from Experiment 6.

Using participants initial prosaccade in corrected antisaccade trials planned comparisons were conducted. A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the proportion of participant errors within this trial type. No effect of Hemifield ($F(1, 11) = .37$, $p = .56$, $r = .18$) was revealed failing to replicate the findings yielded using the same analysis in Experiment 6. Furthermore, no interaction between Experimental Phase and Hemifield was found ($F(2, 22) = .50$, $p = .61$, $r = .15$).

### 4.3.3 Discussion

This experiment was designed to extend the findings of Experiment 6 by including an additional condition in which performance was assessed before reward was introduced. Consistent with the first experiment saccades to a rewarded location displayed facilitation compared to those made to an unrewarded location in the reward paradigm, replicating the results from Experiments 2 and 6. However, there was no evidence that the facilitation of SRTs to rewarded locations transferred to either the trained (prosaccades) or untrained (antisaccades) eye movement when participants performed the PRPEM task. Interestingly, the previous interaction found when using median SRTs failed to replicate in this experiment. This finding further highlights the transient nature of this facilitation. The presence of reward once again failed to elicit any effects on saccadic errors in either of the tasks used. The results of Experiments 6 and 7 are consistent with one another.

## 4.4 General Discussion

The present set of experiments has shown that monetary incentives can modulate the metrics of prosaccades. However, the experiments have also demonstrated the fragility of these effects with respect to their persistence and transference to other types of saccadic eye movement and other tasks. The reward paradigm produced significantly faster prosaccades towards the rewarded hemifield when rewards were introduced during the conditioning phase, an effect that was absent in the preconditioning and extinction phases. This result, observed in Experiments 2, 6 and 7, is consistent with evidence of the effect of reward on the oculomotor system in both primates and humans (Bendiksby & Platt, 2006; Milstein & Dorris, 2007). In an important extension of

previous work, the facilitatory effect has been evidenced as fragile and sensitive to change, such that it did not reliably persist after rewards were withdrawn. Furthermore, no transfer of the effects of rewarding prosaccades to a different form of eye movement was observed. Consistent with previous research and the pilot data (see Chapter 3) antisaccades were significantly slower than prosaccades in the PRPEM task in both Experiments 6 and 7 (Jazbec et al., 2006; Ross et al., 2011; Munõz & Everling, 2004). It is important to be cautious when interpreting this data due to the large rejection rates. However, the replication when using median SRTs highlights the replication of these effects found.

The finding that prosaccade latencies were facilitated when directed to rewarded locations, relative to unrewarded locations, is consistent with threshold models of saccade generation (Findlay & Walker, 1999; Munõz & Schall, 2003), outlined in Chapter 1, in which the generation of a saccade is a competitive process where each potential saccade is vying to reach threshold. In rewarding participants' eye movements during the reward paradigm, the competitive interaction between the rewarded and the unrewarded hemifield would lead to an equilibrium shift and the ability to reach threshold for rewarded hemifield targets to be faster than those for the unrewarded hemifield. This would explain the facilitation effect experienced. This reward learning affect when incentives were present is consistent with neurophysiological models of saccade control (e.g. Trappenberg et al., 2001) and the activation-orienting hypothesis suggesting that lateralised visual input will produce an activation imbalance in favour of the directly stimulated hemisphere (Reuter-Lorenz, Kinsbourne & Moscovitch, 1990). Replication of this result on three separate occasions using the same reward paradigm (Experiments 2, 6 and 7) highlights the consistency of this finding.

An alternative explanation of this reward modulation is that spatial attention was biased towards the rewarded location, resulting in faster processing speed of sensory information from the rewarded location and therefore faster saccade execution. A number of studies discussed in Chapter 1 have demonstrated the capacity for rewards to create attentional biases to colour and forms (Theeuwes, Kramer, Hahn & Irwin, 1998; Della Libera & Chelazzi, 2009). It might therefore be argued that the facilitation effect observed in the reward paradigm of Experiments 2, 6 and 7 stems from alterations in the allocation of spatial attention, rather than modulation of the oculomotor system.

In summary, it has been found that rewarding spatial locations can facilitate the latencies of eye movements to those locations, confirming previous work in humans (Milstein & Dorris, 2007) and non-human primates (Bendiksby & Platt, 2006). However, Experiments 6 and 7 have extended

these findings in two important ways. Firstly, the effects of reward on eye movements were found to be highly context-specific, such that they fail to persist once rewards have been removed. Secondly, the effects of reward learning in a prosaccade task fail to transfer to a separate unrewarded task. These findings permit the conclusion that rewarding eye movements to specific spatial locations is unlikely to induce long-term, systemic changes to the human oculomotor system. The following chapter will further investigate this hypothesis by investigating the effects of reward learning in saccade competition, and whether the competing saccade plans involved in eye movement generation are modulated by rewards.

# Chapter 5: The effects of reward on the remote distractor task

## 5.1 Introduction

The previous chapters (specifically Experiments 2, 6 and 7) have generated a reward paradigm able to create a bias in the processing of eye movements and employed this paradigm to investigate whether rewards can influence the oculomotor and attention systems of human participants. Saccadic eye movements have been found to be susceptible to small monetary rewards, modulating the latency of stimulus-driven eye movements. However it is still unclear to what extent the attention and motor systems are modulated by reward. In everyday life saccades are made in response to multiple stimuli. In this case, multiple potential saccade plans compete with each other in a race to reach threshold first, triggering a saccade (Leach & Carpenter, 2001; Theeuwes et al., 1998). It is believed that mutual inhibition accounts for the longer latencies experienced when saccade plans are in competition with each other, accounted for in the numerous theoretical models of eye movement (see Chapter 1 for review). The remote distractor effect, described in Chapter 1, requires competing saccade plans to be generated as a distractor and target appear together. The present chapter investigates whether the facilitation of eye movements by reward is specific to the motor system through the use of an oculomotor paradigm known to be driven by competing saccade plans, namely the remote distractor paradigm.

There is a direct link between the brain regions involved in the remote distractor effect (RDE) and those implicated in reward learning. The RDE results in increased latencies due to the competing saccade plans prior to saccade generation. This increased latency has been linked with inhibitory processes operating in the SC (Walker et al., 1997) and predominantly the SCi (see Chapter 1 for review). This layer is an area that receives direct inputs from the brain in encoding and processing reward information (Ikeda & Hikosaka, 2003; Basso & Würtz, 1997; Glimcher & Sparks, 1992). Specific to the reward paradigm, evidence has suggested that the SC plays a crucial role in the encoding of reward information during reinforcement learning via its projection to the SNr (Comoli et al., 2003). This projection is responsible for carrying visual activity to the basal ganglia dopaminergic system, critical for reinforcing the behaviour immediately preceding unpredictable, biologically relevant, visual events (Dommett et al., 2005; Redgrave & Guerney, 2006). The SC is also an important part of the circuit that actively chooses strategic actions which will yield positive rewards. This is evidenced by the finding that subthreshold stimulations of the SC can bias choice probability towards the stimulated site of two equally rewarded stimulus locations (Thevarajah et al., 2009). This research implicates the SC as a likely locus for any effects found within the present chapter's experimental findings.

It is possible that both the oculomotor and attention systems are influenced by reward as the mechanisms underlying eye movements and attention are fundamentally interconnected (Goldberg & Würtz, 1972b; Kowler et al., 1995; Gee et al., 2002). However it is also possible that only one of these systems is being affected by reward. Using the remote distractor paradigm, the extent to which the oculomotor system is affected by reward learning will be investigated. Any differences in the latencies of eye movements within distractor trials, when distractors appear in a previously rewarded spatial location, could be a persistent effect of rewards once they are withdrawn. If this effect occurs then we can conclude that the effects of reward are occurring within the oculomotor system, facilitating eye movements in the direction of reward.

Previous studies investigating the effects of reward on attention have highlighted that stimuli or particular stimulus features can be increased in salience when rewarded, such that they can be granted attentional priority over unrewarded stimuli/features (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al., 2010a; Theeuwes, 1991, 1992). Therefore, it is possible that the reward paradigm employed in previous chapters generates a stimulus-reward association rather than a location-reward association. Based on these findings from previous chapters and previous research (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al., 2010a) it seems pertinent to investigate whether a stimulus previously associated with reward feedback during the reward paradigm would result in it being a more salient distractor than a novel stimulus. In this way, stimulus-reward and location-reward associations can be teased apart, extending the findings of the aforementioned literature. Therefore, within the remote distractor task applied in the present chapter, three different trial types were employed: 1) a known distractor trial, where the distractor used was the same stimuli (square) associated with reward feedback in the reward paradigm; 2) a novel distractor trial, where the distractor used was a novel stimulus (triangle); 3) a no distractor trial, where the target (circle) was presented on its own with no distractors. This would enable investigation of whether biases associated with stimuli or stimulus features previously associated with reward feedback persist after rewards are withdrawn, extending the findings of previous investigations in two clear ways. Firstly, the time-course of any stimuli bias associated with a previously rewarded stimulus in the distractor task, relative to a novel stimulus, can be explored. Secondly, this paradigm permits disentanglement of the effects of reward on spatial locations, from the effects of reward on a particular stimulus or stimuli features. The hypothesis of the present investigation was that the remote distractor effect would be greater when distractors appeared at rewarded locations.

## 5.2 Experiment 8

### 5.2.1 Method

#### *5.2.1.1 Participants*

The sample size of twelve participants used in the previous experiments was replicated. Twelve participants recruited from the University of Durham volunteered for the experiment. Individuals who had previously participated, and as such had experience of the reward paradigm, were not allowed to participate. The participants - 4 males and 8 females – had an age range of 19-25 years (mean age 20.83 years). Nine were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *5.2.1.2 Apparatus*

The experimental setup was as described in Experiment 2, Chapter 2. However, participants were also required to complete an amended consent form prior to participation (see Appendix E).

#### *5.2.1.3 Stimuli*

The reward paradigm was as described in Experiment 2, Chapter 2.

In the Remote Distractor (RD) task, participants were presented with a 0.7° x 0.7° fixation cross in the centre of the screen on a grey background. A target stimulus 1.0° x 1.0° circle was presented to the left or right of the fixation cross. A related distractor square and an unrelated distractor triangle were both 1.0° x 1.0° of visual angle. Target and distractor stimuli were presented 6.5° to the left or right and 3.7° upwards from fixation.

#### *5.2.1.4 Procedure*

The procedure employed in Experiment 2 was replicated in the present experiment with the addition of the RD task running for six blocks directly after the conditioning and extinction phases of the reward paradigm. Each block contained 90 trials equally split between each condition type. Trials were also randomised. Participants were instructed to fixate on the central fixation cross prior to the start of each trial which appeared for a random period of time between 500 and

700ms, to avoid anticipatory eye movements. After a successful saccade, the target stimuli would change colour from grey to white, after which the trial ended. A button press was required to start the next trial. A single RD block consisted of three types of distractor trial: 1) a known distractor trial, consisting of a target circle in one hemifield and a distractor square (previously used as the target in the reward paradigm) in the opposite hemifield; 2) a novel distractor trial, consisting of a target circle in one hemifield and a novel stimulus (triangle) in the opposite hemifield; 3) a no distractor trial, where only a target circle appeared in one hemifield, with no other stimuli present. Figure 5.1 displays the experimental array.

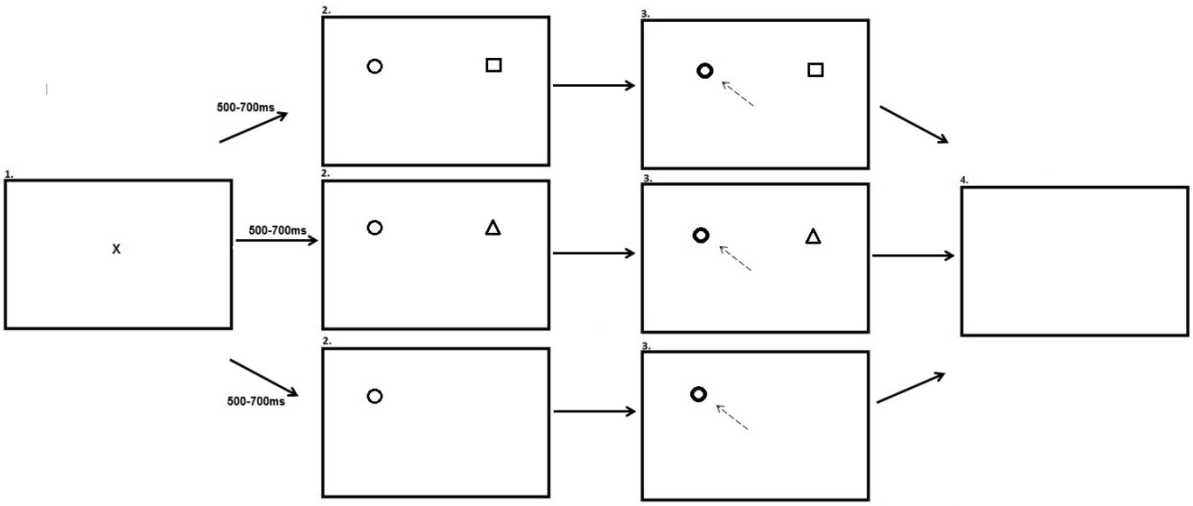


***Figure 5.1*:** Sequence of events used in the remote distractor (RD) task (not to scale). First a fixation cross appeared (Row 1). Secondly, the fixation cross disappeared and a target circle appeared in either the left or right visual field (Row 2). In known distractor trials a distractor square would appear in the hemifield opposite to the target (Row 2, Panel 1). In novel distractor trials a distractor triangle would appear in the hemifield opposite to the target (Row 2, Panel 2). In no distractor trials no distractor was present (Row 2, Panel 3). After a successful saccade to the target circle, this target circle would change colour (Row 3, Panels 1,2 & 3). After a button press participants would be presented with a blank screen (Row 4) indicating the trial had finished and a new trial was about to begin.

*5.2.1.5 Saccade Analysis*

Mean SRTs were calculated for each individual block within an experimental phase. The analysis was then conducted on the mean of these means. These analyses were once again replicated using median SRTs. Data were filtered so that inaccurate trials and trials above the threshold

(>500ms) were eliminated from the analysis. Saccadic error was defined as any saccade that left the fixation area but did not land at the target location.

*5.2.1.5.1 Reward Paradigm*

Across 12,960 saccadic trials, 6% were identified as inaccurate. 30.2% of trials were over the threshold and also removed from the analysis. In total, 36.2% of conditioning trials were rejected from the analysis.

*5.2.1.5.2 RD Task*

Of the 12,960 trials within the remote distractor task, 13.9% were identified as inaccurate with 18.6% of trials over the threshold. In total, 32.6% of remote distractor trials were rejected from the analysis.

**5.2.2 Results**

A significance correction level of $p < .05$ was adopted, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*5.2.2.1 Latency*

*5.2.2.1.1 Reward Paradigm*

Using mean SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed a main effect of Experimental Phase ($F(2, 22) = 4.36$, $p = .03$, $r = .41$) such that saccades were significantly faster during the conditioning phase (256 ms) compared to the extinction phase (285 ms) ($t(11) = -2.93$, $p = >.017$, $r = .66$). No significant effect of Hemifield was found ($F(1, 11) = 3.49$, $p = .09$, $r = .49$).

Crucially, there was a significant interaction found between Experimental Phase and Hemifield ($F(2, 22) = 5.23$, $p = .01$, $r = .44$). Three separate one-way ANOVAs were conducted on the latency of saccades for the rewarded and unrewarded hemifields at each level of Experimental Phase separately in order to explore this two-way interaction. In the preconditioning phase, no

significant difference was found between the rewarded (282 ms) and unrewarded (282 ms) hemifields (F (1, 23) = <.01, p = .99, r = <.01). In the conditioning phase however, a significant difference between the rewarded (242 ms) and unrewarded (271 ms) hemifields was revealed (F (1, 23) = 5.88, p = .02, r = .45). In the extinction phase, where reward was removed, a non-significant difference was found between the rewarded (285 ms) and unrewarded (286 ms) hemifields (F (1, 23) = .01, p = .93, r = .02). Figure 5.2 illustrates these results.

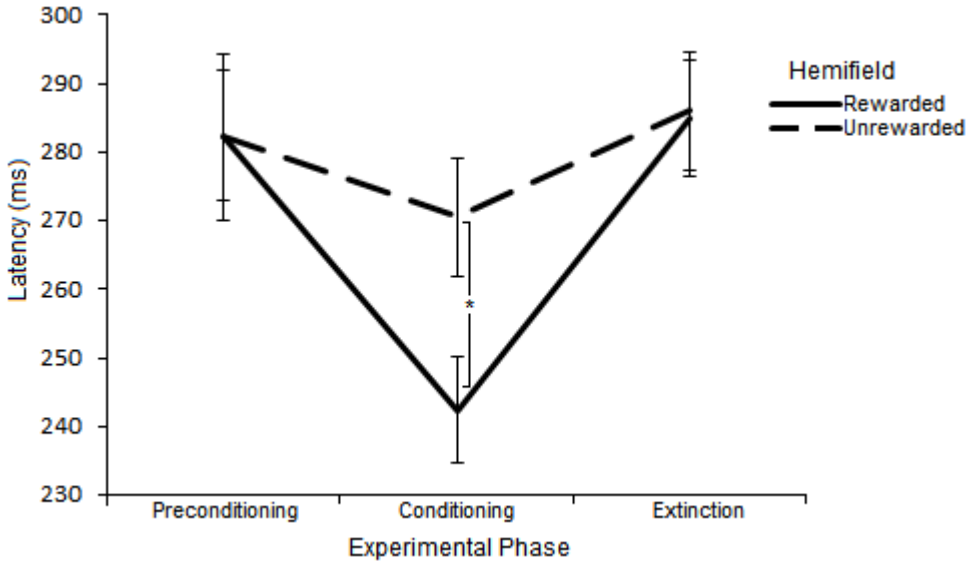


***Figure 5.2:*** *Latency of prosaccades to the rewarded (black line) and unrewarded (black dashed line) hemifields in the reward paradigm across the preconditioning, conditioning and extinction phases of the reward paradigm. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed a main effect of Hemifield (F (1, 11) = 11.74, p = <.01, r = .72) such that saccades to the rewarded hemifield (264 ms) were significantly faster than those to the unrewarded hemifield (283 ms). No interaction between Experimental Phase and Hemifield was revealed (F (2, 22) = 1.58, p = .23, r = .26) contrary to the mean SRT analysis.

Planned comparisons replicated the effects found in mean SRTs. In the preconditioning phase, no significant difference was found between the rewarded and unrewarded hemifields (t (11) = -.46, p = >.017, r = .14). In the conditioning phase however, a significant difference between the rewarded (250 ms) and unrewarded (284 ms) hemifields was revealed (t (11) = -2.47, p = <.017, r

= .60). In the extinction phase, where reward was removed, a non-significant difference was found between the rewarded and unrewarded hemifields (t (11) = -1.95, p = >.017, r = .51).

*5.2.2.1.2 RD task*

A 3 (Distractor Type: Known/Novel/No Distractor) x 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed a main effect of Experimental Phase (F (1, 11) = 9.79, p = .01, r = .69) such that saccades made during the post-extinction phase (235 ms) were significantly faster than saccades made during the post-conditioning phase (245 ms). A significant effect of Distractor Type was revealed (F (2, 22) = 129.47, p = <.01, r = .92) such that saccades in trials where no distractor (212 ms) was present were significantly faster than saccades made on known (254 ms) (t (11) = -14.96, p = >.017, r = .98) and novel (254 ms) (t (11) = -11.86, p = >.017, r = .96) distractor trials.

No other significant effects or interactions were found. Crucially there was no interaction found between Distractor Type and Experimental Phase (F (2, 22) = 1.22, p = .31, r = .23) suggesting no change in the latencies of different distractor trials across the times the trials were delivered. Furthermore, no differences were found between the latencies of saccades to either hemifield across the different types of distractor trial (F (2, 22) = .16, p = .85, r = .09). No three-way interaction between Distractor Type, Hemifield and Experimental Phase existed (F (2, 22) = .36, p = .70, r = .13) suggesting no effect of reward on the saccadic latencies of distractor trials at the two times this task was delivered. Figure 5.3 illustrates these results.

Using median SRTs a 3 (Distractor Type: Known/Novel/No Distractor) x 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated a main effect of Experimental Phase (F (1, 11) = 8.61, p = .01, r = .66) such that saccades made during the post-extinction phase (234 ms) were significantly faster than saccades made during the post-conditioning phase (242 ms). A main effect of Distractor Type was also replicated (F (2, 22) = 122.07, p = <.01, r = .92) where saccades in trials where no distractor (209 ms) was present were significantly faster than saccades made on known (254 ms) or novel (252 ms) distractor trials. Interestingly a main effect of Hemifield was also revealed (F (1, 11) = 4.98, p = .05, r = .56) such that saccades were faster to the rewarded hemifield (236 ms) than the unrewarded hemifield (241 ms). As with the mean SRT analysis no interactions were found.

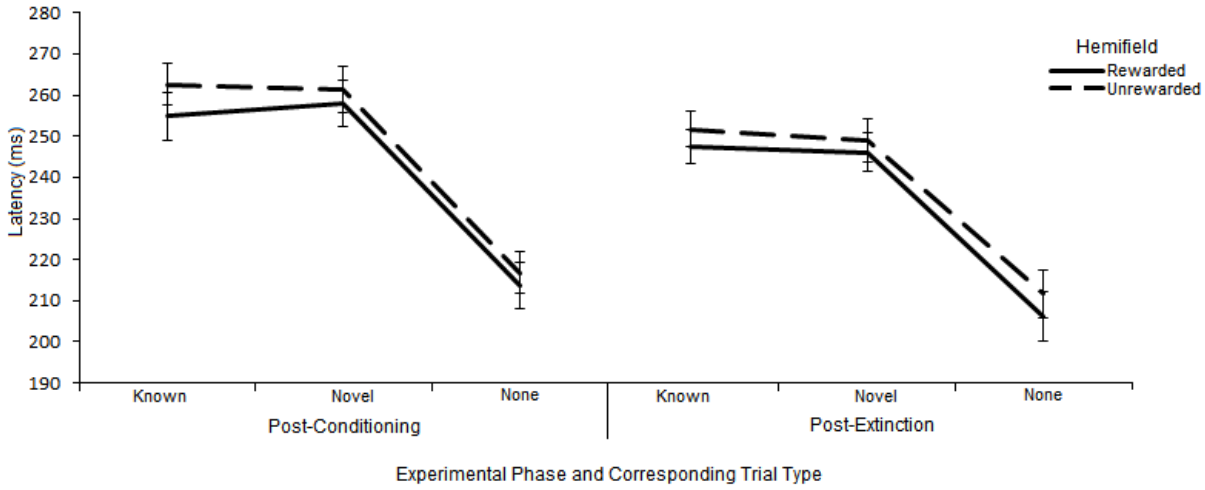


***Figure 5.3****: Latency of prosaccades in the different distractor type trials, to the rewarded (black line) and unrewarded (black dashed line) hemifields, in the post-conditioning and post-extinction phases. Error bars show +/- 1 SEM.*

Planned comparisons were conducted on the latencies of saccades to the rewarded and unrewarded hemifields in the three different types of distractor trials to examine whether the effect of facilitation was evident in the remote distractor task. Using mean SRTs no significant effects were found between the rewarded and unrewarded hemifields in the Post-Conditioning (t (11) = -.80, p = >.017, r = .23) or Post-Extinction (t (11) = -1.08, p = >.017, r = .10) phases in no distractor trials. No significant effects were found between the rewarded and unrewarded hemifields in the Post-Conditioning (t (11) = -1.73, p = >.017, r = .37) or Post-Extinction (t (11) = -.82, p = >.017, r = .26) phases in known distractor trials. No significant effects were found between the rewarded and unrewarded hemifields in the Post-Conditioning (t (11) = -.81, p = >.017, r = .24) or Post-Extinction (t (11) = .91, p = >.017, r = .26) phases in novel distractor trials. These null effects were replicated using median SRTs.

*5.2.2.1.3 Time-course of the Extinction*

Investigation of the time-course of the facilitation effects in Chapter 2 revealed significant differences between the two hemifields in the extinction phase for a period of three blocks after rewards were withdrawn. Therefore, to investigate a time-course for the facilitation effects found, analysis of the RD task administered directly after the post-conditioning phase was

conducted. The first three blocks of the RD task formed one group with the last three blocks forming a second group. Using mean SRTs, a 3 (Distractor Type: Known/Novel/None) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Time-course: Group 1/Group 2) repeated measures ANOVA on saccadic latencies exclusively within the post-conditioning phase of the RD task revealed no effect of Time-course ($F (1, 11) = .01$, $p = .93$, $r = .03$) with no significant differences between the latencies of saccades in Group 1 (235 ms) and Group 2 (235 ms). Furthermore, no interactions were revealed between Distractor Type and Time-course ($F (2, 22) = .57$, $p = .58$, $r = .16$), Hemifield and Time-course ($F (1, 11) = .24$, $p = .63$, $r = .15$), or Distractor Type, Hemifield and Time-course ($F (2, 22) = .15$, $p = .84$, $r = .08$).

Using median SRTs, a 3 (Distractor Type: Known/Novel/None) x 2 (Hemifield: Rewarded/Unrewarded) x 2 (Time-course: Group 1/Group 2) repeated measures ANOVA replicated no effect of Time-course ($F (1, 11) = 2.04$, $p = .18$, $r = .40$) with no significant differences between the latencies of saccades in Group 1 and Group 2. Furthermore, no interactions were revealed between Distractor Type and Time-course ($F (2, 22) = 1.31$, $p = .29$, $r = .24$), Hemifield and Time-course ($F (1, 11) = .13$, $p = .73$, $r = .11$), or Distractor Type, Hemifield and Time-course ($F (2, 22) = .88$, $p = .43$, $r = .20$).

*5.2.2.2 Saccadic Error*

*5.2.2.2.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA revealed a main effect of Experimental Phase ($F (2, 22) = 9.72$, $p = <.01$, $r = .55$) with a significantly larger proportion of errors occurring in the conditioning phase (.024) than the preconditioning (.005) and extinction (.013) phases.

No effect of Hemifield ($F (1, 11) = .03$, $p = .86$, $r = .05$) or interaction between Experimental Phase and Hemifield was revealed ($F (2, 22) = .19$, $p = .83$, $r = .09$) suggesting no effect of the conditioning process on where participant errors were directed.

*5.2.2.2.2 RD Task*

Using only distractor trials, the percentage of incorrect trials was calculated from the total number of trials in each experimental phase. A 2 (Distractor Type: Known/Novel) x 2

(Experimental Phase: Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the calculated percentage of incorrect trials in the RD task. A significant effect of Distractor Type was found ($F(1, 11) = 6.50$, $p = .03$, $r = .61$) such that a larger percentage of incorrect trials occurred on novel distractor trials (.28) compared to known distractor trials (.23). No other significant differences were revealed. No effect of Experimental Phase ($F(1, 11) = 2.54$, $p = .14$, $r = .19$) or Hemifield were revealed ($F(1, 11) = .10$, $p = .76$, $r = .09$). Furthermore, no interaction between Distractor Type and Hemifield ($F(1, 11) = .10$, $p = .76$, $r = .09$) or three-way interaction between Experimental Phase, Distractor Type and Hemifield was found ($F(1, 11) = .27$, $p = .62$, $r = .15$).

Trials in which participants made an erroneous saccade directly towards a distractor were analysed in order to examine the oculomotor capture. A 2 (Experimental Phase: Post-Conditioning/Post-Extinction) x 3 (Distractor Type: Known/Novel/None) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the proportion of errors categorised as oculomotor capture. This analysis revealed a main effect of Distractor Type ($F(2, 22) = 43.97$, $p = <.01$, $r = .82$) such that no distractor trials (.016) produced significantly less oculomotor capture than known (.383) ($t(11) = 5.62$, $p = <.017$, $r = .61$) and novel (.456) ($t(11) = 10.12$, $p = <.017$, $r = .95$) distractor trials. Furthermore, the novel distractor trials produced significantly greater oculomotor capture than the known distractor trials ($t(11) = 2.55$, $p = <.017$, $r = .61$). Figure 5.4 illustrates these results. No effect of Hemifield ($F(1, 11) = .04$, $p = .85$, $r = .06$) or interactions between Experimental Phase and Hemifield ($F(1, 11) = 1.14$, $p = .31$, $r = .31$), Distractor Type and Hemifield ($F(2, 22) = .99$, $p = .83$, $r = .09$) or three-way interaction between Experimental Phase, Distractor Type and Hemifield ($F(2, 22) = .18$, $p = .84$, $r = .09$) was found.

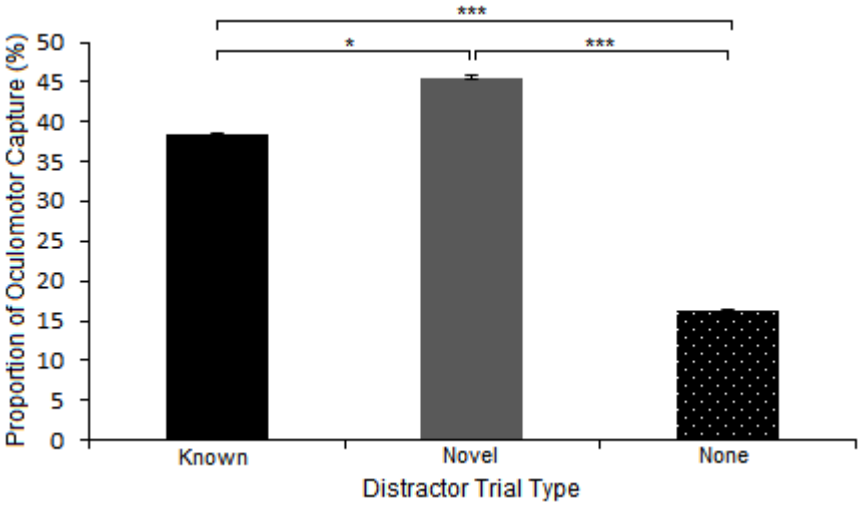


***Figure 5.4:*** *The proportion of oculomotor capture across the three different types of distractor trial within the RD task. The black bar represents the known distractor trials; the grey bar represents the novel distractor trials; the black dotted bar represents the no distractor trials. Error bars show +/- 1 SEM.*

### 5.2.3 Discussion

The primary aim of the present chapter was to determine whether the effects of facilitation found in the reward paradigm occur due to biases in the motor system through the use of the RD task; a paradigm known to involve competing saccade plans. Using the reward paradigm allowed generation of a facilitation effect, whilst reward feedback was present. In the conditioning phase, saccades were facilitated to the rewarded location, relative to the unrewarded hemifield. Although a remote distractor effect was found in the RD task, the magnitude of the RDE was not modulated following the instrumental conditioning of eye movements.

The RD task generated significantly faster saccadic eye movements when no distractors were present compared to trials where distractors coincided with target presentation. This result is consistent with previous findings that the presentation of an irrelevant stimulus in the visual field results in slower SRTs (Bompas & Sumner, 2009b; Honda, 2005; Lévy-Schoen, 1969; Ludwig, Gilchrist, McSorley & Baddeley, 2005a; Walker et al., 1997). In the RD task, two different types of distractor were used: a distractor exactly the same as the target used in the reward paradigm previously associated with reward feedback, and a novel distractor. One interesting finding using these different distractors was the difference in the proportion of error. Distractor trials with a previously known stimulus evoked a significantly smaller proportion of errors and significantly less oculomotor capture than distractor trials with a novel stimulus. Previous studies had suggested that a stimulus or stimulus features associated with rewards are granted attentional priority and as such become more salient (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al.,

2010a; Theeuwes, 1991, 1992). In the present experiment, if this were the case, we would expect to see greater attentional capture of the known distractor. Instead, a significantly smaller proportion of errors occurred on known distractor trials than novel trials. One explanation for this effect is that participants learn to habituate the known distractor. Habituation is a form of learning where an organism decreases or ceases to respond to a stimulus after it has been presented repeatedly (Bouton, 2007). In this experiment, participants habituate to the known distractor after its repeated presentations and are therefore more able to ignore it in search of the target, resulting in less error on this trial type. As the novel stimulus has not been presented as often as the known stimulus, it has greater attentional capture, resulting in the significantly greater proportion of errors on these trials.

The effects of facilitation found in the conditioning phase of the reward paradigm, failed to transfer to the RD task when analysing the time course of the extinction. Direct comparisons can be made between the reward paradigm trials and the no distractor trials in the RD task. The trial types are very similar except for the shape of the target stimulus and the reward feedback available in the reward paradigm. No effects of reward were found in the no distractor trials of the RD task between the two hemifields. The addition of other complex trial types interspersed with the no distractor trials may be a contributing factor, extinguishing any facilitation effects previously found. In previous chapters it has been suggested that this facilitation may be sensitive to a number of factors including alterations in task demands, changing stimuli and different trial types. Therefore, it is possible that the sensitivity of the facilitation effect of reward is susceptible to changes in the context that participants are rewarded in, failing to replicate when the context in which facilitation occurs is altered (Blaukopf & DiGirolamo, 2007). This suggests that the effects seen in the reward paradigm (Experiments 2, 6, 7 and 8) are task-specific.

In summary, replication of the facilitation of saccades to rewarded, relative to unrewarded locations, is evidence of the consistency of the reward paradigm. When reward feedback is available, participants are significantly faster in eliciting saccades to rewarded locations. Similarly to Chapter 3, this specific effect failed to transfer to a secondary unrewarded eye movement task. The data presented shows the limited scope of this effect and that in altering task demands, any effect established is extinguished rapidly. This experiment failed to further the knowledge of the extent to which the motor system is specifically affected by rewards due to the lack of transfer of the facilitation effects found in the reward paradigm. However this result extends the previous knowledge of the longevity of the effects of reward and further highlights the fragility of this effect. Altering the demands of the task or changing the context in which the eye movement is

trained leads to the abolition of the facilitation effects previously found. This result is consistent with the effects found in Chapter 4. Furthermore, differences in the proportion of error were found on distractor trials where the stimulus was previously associated with reward feedback and distractor trials with a novel stimulus. The significantly reduced proportion of errors on known distractor trials can be explained in terms of habituation, directly contrasting previous studies suggesting stimuli previously associated with reward are more salient when reappearing as distractors (Della Libera & Chelazzi, 2009; Anderson et al., 2011a). The next set of experiments will address the effects of reward on covert and overt orienting of attention using the peripheral cueing and inhibition of return paradigms.

# Chapter 6: The effects of reward on exogenous orienting of attention and IOR

## 6.1 Introduction

Chapters 2, 4 and 5 have revealed a robust effect of facilitation in the reward paradigm, such that eye movements are significantly faster to rewarded locations, relative to unrewarded locations, when reward feedback is present. However, a lack of transfer and persistence exists when introducing a different type of task where other targets are involved (the RD task) or other types of eye movement are required (the antisaccade task). Many studies have highlighted that prior to the execution of an eye movement to a novel location, spatial attention is first shifted to the new location (Deubel & Schneider, 1996; Godijn & Theeuwes, 2004; Belopolsky & Theeuwes, 2009). Although we foveate a location in space where we wish to attend we can also direct our attention to a region in our visual field without directing our eyes to that location (Posner, 1980). The selection of information based on its spatial location in the absence of eye movements is referred to as spatial covert attention. Covert spatial attention selectively grants priority in processing to parts of the otherwise overwhelming amount of information at unattended locations (Carrasco, 2011; Lu & Dosher, 1998; Luck et al., 1994; Montagna, Pestilli & Carrasco, 2009; Pestilli & Carrasco, 2005). By trading off processing resources between attended and unattended locations in the visual field, attention allows the optimisation of performance in visual tasks while overcoming the visual system's limited capacity. Spatial covert attention can be deployed exogenously and endogenously. Exogenous attention is stimulus-driven, automatically activated by the sudden onset of a stimulus in the visual field whereas endogenous attention is conceptually driven, voluntarily allocated to a location in the visual field. The present chapter aims to investigate the effects of incentives on covert orienting of attention and on exogenous attention.

In the peripheral cueing paradigm, a salient cue is presented to attract attention and perceptual performance is assessed either at the cued or uncued location with the presentation of a target stimulus. The robust finding associated with this paradigm is that when the interval between cue and target presentation is short, performance at the cued location is facilitated (Klein, 2000; Posner & Cohen, 1984; Samuel & Kat, 2003). When longer time intervals (>300ms) occur between the cue and the target, the effect is reversed, with longer latencies occurring at cued locations. This effect has been interpreted as a delay in the re-allocation of attention to the already attended location; a phenomenon termed the Inhibition of Return (IOR) (Posner et al., 1985; Klein, 2000; Klein & MacInnes, 1999). IOR is seen to promote efficient exploration of a visual scene, preventing an observer from returning to previously attended locations and instead

promoting exploration of novel locations. This inhibition has also been found within saccadic responses (Rafal et al., 1994; Taylor & Klein, 2000; Vaughan, 1984).

Previous research has highlighted the differences that exist between the two types of IOR; the traditional IOR measured in manual reaction times and saccadic IOR. Sumner, Nachev, Vora, Husain and Kennard (2004) explored both types of IOR, investigating the extent to which the SC is involved in the generation of IOR in both response types. Using short-wave sensitive (S) cone stimuli, which are 'invisible' to the SC, they reasoned that if the SC was solely responsible for the generation of IOR, non-informative peripheral events processed via pathways not projecting directly to the SC might fail to generate the IOR effect. Manual responses showed evidence of IOR, whereas saccadic responses failed to show an IOR response when S-cone stimuli cues were used. Sumner et al., (2004) concluded that there must be separate IOR generators; one mediated by the retinotectal pathway generating IOR in response to oculomotor activation and one mediated cortically following attentional capture. The retinotectal pathway is responsible for generating saccadic IOR whereas both generators contribute to the traditional IOR measured by manual responses. Therefore, there are distinct differences between the networks used for perceptual and attention processes in IOR.

The SC is a critical structure for both IOR and attentional capture. In the peripheral cueing task, where delay between cue and target is short, neural activity of the SCi is increased as cue and target combine to produce a greater response. This drives faster SRTs, reflected in greater attentional capture (Fectau & Munõz, 2005). Conversely, when delay is longer between cue and target, visually responsive neurons in the SCs and SCi display an attenuated target-related visual response, correlating with the slower SRTs reflected in IOR (Dorris, Klein, Everling & Munõz, 2002). Both attentional capture seen in the peripheral cueing task and IOR have neural correlates with sensory responses in the SC (Dorris et al., 2002; Fectau & Munõz, 2005).

Patient studies have further highlighted the critical role the SC plays in IOR. Sereno, Briand, Amador and Szapiel, (2006) tested a patient with a thiamine deficiency and a lesion of the SC. The patient had a complete lack of an IOR effect in a covert spatial attention task highlighting the level of collicular involvement in covert orienting in humans. Rafal, Calabresi, Brennan and Sciolto, (1989) demonstrated a visual field asymmetry which they argued was due to the unequal visual field representation occurring in the innervations of the SC. The visual pathways leading to the SC include crossed fibres from the nasal hemiretina of the contralateral eye as well as uncrossed fibres from the temporal hemiretina of the ipsilateral eye. The SC is innervated by more crossed than uncrossed fibres. As a result, visual input from the two nasal hemiretinae (temporal visual

fields) has a stronger representation in the SC than information from the two temporal hemiretinae (nasal visual fields). Rafal et al., (1989) asked participants to complete a standard covert spatial cueing task where they viewed the display whilst wearing an eye-patch over one eye. Participants showed reduced IOR for stimuli in the nasal visual field relative to the temporal visual field. This suggests that the SC plays a role in the generation of IOR; a finding verified by the patient work of Sapir, Soroker, Berger and Henik, (1999). Using a covert spatial orienting task in a patient with a unilateral lesion of the right SC, Sapir et al., (1999) failed to find an IOR effect in the temporal visual field of the left eye or the nasal visual field of the right eye. These visual field deficits correlate with regions of the visual field presumed to be affected by a lesion of the SC. This data provides further evidence for a role of the SC in IOR. This data implicates the SC as a necessary component for IOR and as a critical structure for covert spatial attention in humans.

As has been previously explored, the SC has also been implicated as a significant structure in the processing of reward (see Chapter 1 for review). When a visual stimulus signals an upcoming reward, both visual and preparatory activity of SCi neurons is enhanced (Ikeda & Hikosaka, 2003). The SC has also been implicated in playing an active role in the encoding of reward information during reinforcement learning due to its projection to the SNr (Comoli et al., 2003); a projection which carries transient visual activity to the basal ganglia dopaminergic system, which is critical for reinforcing the context or action that immediately precedes unpredicted biologically relevant visual events (Dommett et al., 2005; Redgrave & Guerney, 2006). Therefore the SC is a key component in the processing of reward and plays a crucial role in both attentional capture and IOR. It is clear that any effects found in the present experiment may be attributable to this structure, or the projections to and from it.

Recently, the investigation of reward-induced motivational effects on exogenous attentional orienting has been explored, measured in manual reaction times. Bucker and Theeuwes (2014) designed an experiment to determine whether motivationally driven influences of reward affect exogenous spatial orienting and IOR. The authors employed a version of the exogenous orienting task in which attention was captured by a peripheral cue non-predictive of target location. Participants could earn a low or high reward dependent upon their performance. Short and long SOAs were varied within blocks. Reorienting of attention was affected by rewards whilst initial orienting of attention was unaffected. These results suggested that cue facilitation effects on initial orienting are not modulated by the manipulation of reward induced motivation. However, reward modulation effects did affect IOR, such that faster reorienting of attention away from the cued location, relative to the validly cued location, was revealed. Crucially, this effect was not

found when small rewards were employed, with no differences between validly and invalidly cued locations found. This finding suggests that IOR, an effect assumed to be stimulus-driven and automatic (Theeuwes, 2013), is affected by reward induced motivational factors. The lack of effect in valid trials at short SOAs is consistent with previous studies (Engelman & Pessoa, 2007). This result is also consistent with Shomstein and Johnson (2013), who have claimed that space-based and not object-based guidance of attention is robust to influences of reward. The findings presented by Bucker and Theeuwes (2014) suggest that immediate spatial exogenous attention is fully automatic, stimuli-driven and not modulated by motivational context.

Bucker and Theeuwes (2014) posited that the reward effects on IOR and lack of effect in initial stimulus-driven orientation could be explained in terms of IOR being influenced in a top-down manner. Separate attentional systems, with distinct underlying neural networks (Fectau & Munõz, 2005) are involved in orienting to a cued location and disengagement of attention from this location (Corebtta & Schulman, 2002). Validly cued targets evoke a single attention guiding process that is mediated by the orienting networks (Thiel, Zilles & Fink, 2004). Conversely, invalidly cued targets evoke several processes including disengagement from the cued location and shifting attention to another location, mediated by the reorienting network (Corbetta, Kincade, Ollinger, McAvoy & Shulman, 2000). This network can be influenced by properties making target stimuli more salient (Downar, Crawley, Mikulis & Davis, 2002). Bucker and Theewues (2014) argue that the reorienting of attention and accompanying IOR are completely stimulus-driven and partially involve top-down processes. It is therefore possible that motivational top-down processes, such as those induced by reward, modulate the reorienting of attention and IOR. Two important methodological differences exist between the present investigation and the work of Bucker and Theewes (2014). Firstly, the present experiment investigates the influence of reward in covert and overt orienting in saccadic, rather than manual, responses. Secondly, reward is processed differently. Bucker and Theewues (2014) presented participants with high and low value monetary rewards for completing the exogenous orienting task after trials. The present investigation will focus on previously learnt spatial-reward associations and their subsequent transfer to unrewarded cueing tasks. If replication of Bucker and Theewues (2014) findings occurs, it can be suggested that the effects of reward persist after withdrawal and continue to influence human attention and the saccadic eye movement system.

This chapter aims to extend the research conducted by Bucker and Theeuwes (2014) investigating the effects of reward learning on covert spatial attention using two exogenous cueing tasks: one employing a short SOA (150 ms) to explore exogenous facilitation and one employing a long SOA

(600 ms) to explore the effects of reward in IOR. Prior to the coupling of the reward paradigm and the spatial cueing task (IOR), a pilot experiment was conducted (see Chapter 3) in order to ensure the task generated results consistent with the inhibition effects previously found in a number of other experiments (Posner et al., 1985; Klein, 2000; Klein & MacInnes, 1999). Monetary rewards modulate saccadic eye movements when they are present, evidenced by the reward paradigm described in Experiments 2, 6, 7 and 8. This modulation may arise due to changes in the levels of activity within the SC. As previously suggested rewarding one hemifield would lead to an equilibrium shift in the SC in the ability to reach threshold for rewarded hemifield targets. It is believed that these effects activate caudate neurons, inhibiting neurons of the ipsilateral SNr, leading to a disinhibition of SC neurons making it easier for saccades to reach threshold for saccade execution to rewarded locations. The aforementioned studies have also evidenced just how critical the SC is as a structure for the generation of IOR. Therefore, it should follow that changes to the SC triggered by financial rewards should also have an effect on IOR. Therefore, it is expected that monetary rewards would lead to increased inhibition of return at the rewarded location. A separate line of argument exists for the exogenous cueing task. Exogenous attentional facilitation is dependent upon activity in the oculomotor system (Smith, Schenk & Rorden, 2012). During the exogenous cueing task, the sudden onset of a cue increases the physical salience of the cued location and triggers the preparation of a saccade to this location. This creates a powerful bias in the visual and oculomotor system in the direction of the cued location. This bias is thought to propagate through the perceptual-motor system facilitating the processing of subsequent visual events at the cued location. Rewards have been found to modulate activity in the oculomotor system (Takikawa et al., 2002). Therefore, it is hypothesised that rewards should also modulate exogenous attentional facilitation.

## 6.2 Experiment 9

### 6.2.1 Method

#### *6.2.1.1 Participants*

The sample size of twelve participants used in the previous experiments was replicated. Twelve participants, recruited from the University of Durham, volunteered for the experiment. The participants – three males, nine females – had an age range of 19-25 years (mean 20.67). Eight were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

*6.2.1.2 Apparatus*

Prior to participation, participants were required to complete a consent form (see Appendix F). The experimental stimuli were generated using a Cambridge Research Systems ViSaGe graphics card and displayed on a 17 inch Eizo Flexscan Colour Display monitor with a refresh rate of 100Hz. Responses were collected using a two-button button box. Eye movements were recorded using a Cambridge Research Systems eye tracker with a sampling rate of 160Hz.

*6.2.1.3 Stimuli*

The reward paradigm was replicated from Experiment 2.

In the peripheral cueing task, participants were presented with a black 0.5° x 0.5° fixation cross in the centre of the screen (0°) on a grey background with a black outlined stimulus 0.5° x 0.5° square present 8.0° to the left and right of the fixation cross. A smaller 0.3° x 0.3° white target square appeared within the larger squares.

*6.2.1.4 Procedure*

The same eye dominance tests and calibration procedure outlined in Experiments 1-8 were replicated in the present experiment.

*6.2.1.4.1 Reward Paradigm*

The reward paradigm was replicated from Experiment 2.

*6.2.1.4.2 Peripheral Cueing task*

The peripheral cueing task occurred directly after the preconditioning, conditioning and extinction phases of the reward paradigm. This task also consisted of three clear experimental phases; the post-preconditioning, post-conditioning and post-extinction phases. Within this task participants completed three types of trial: Valid, Invalid and No cue. In valid trials, participants were

presented with a fixation cross in the centre of the screen and two black-outlined squares, one to the left and one to the right of fixation. After 700ms one of the black-outlined squares changed colour from black to white for a brief period of time (100ms) cueing participants attention towards this location. The fixation cross then pulsated to re-orient participant's attention back to the centre of the screen. 150ms after peripheral cue onset the white target square appeared at the same location as the previous colour change. On invalid trials, the target square appeared at the opposite location of the previous colour change. In no cue trials, there was no initial colour change of the black outlined square and participants had to make an eye movement towards the white target square. Each block consisted of 60 trials equally split between each type of trial. The order in which participants completed each block was randomised to negate any order effects. It is important to state that in investigating exogenous orienting, it is crucial that the cue is non-predictive of the target location. If the cue predicts the location of the target, observers will use the cue to direct their attention and as such conclusions cannot be drawn regarding exogenous, stimulus-driven, bottom-up capture. Therefore, in this paradigm the cues were non-predictive. Figure 6.1 illustrates the experimental array.

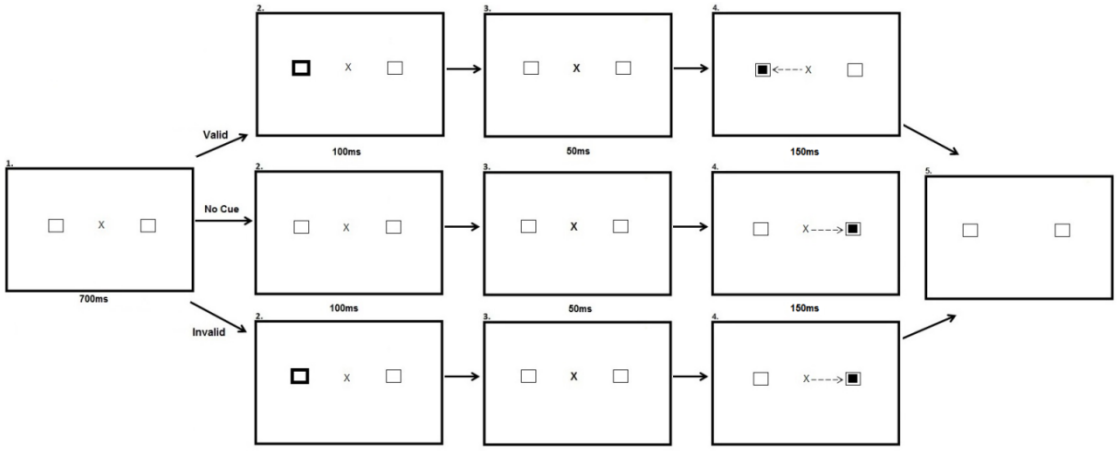


***Figure 6.1:*** *Sequence of events used in Experiment 9 for the attentional capture task (not to scale). Participants were presented with a fixation cross and two squares equidistant from the fixation cross in opposing hemifields (Row 1, Panel 1). In valid trials one of the squares changes colour for a period of 100ms, cueing participants to this location (Row 2, Panel 1). Participants are then presented with the same screen as in the first panel for a period of 50ms (Row 3). A smaller target square then appeared in the same location as the cue and participants were required to saccade to this location (Row 4, Panel 1). After making a successful saccade the fixation cross disappeared and the screen changed colour requiring a button press to begin the next trial (Row 5, Panel 1). In no cue trials no cue appeared prior to target onset (Row 2, Panel 2). In invalid trials the cue appeared in one location (Row 2, Panel 3) and the target appeared in the opposite location (Row 4, Panel 3).*

The full experiment ran for 27 blocks and lasted for approximately one hour. Participants switched between blocks of the two eye movement tasks. Firstly, participants completed the preconditioning phase of the reward paradigm (2 blocks) and then the post-preconditioning phase of the exogenous cueing task (3 blocks). Participants then completed the conditioning phase of the reward paradigm (10 blocks) followed by the post-conditioning phase of the exogenous cueing task (3 blocks). Participants then completed the extinction phase of the reward paradigm (6 blocks) and finally the post-extinction phase of the exogenous cueing task (3 blocks).

#### *6.2.1.5 Saccade Analysis*

The analysis was conducted on the means of each participant's average SRT calculated from each individual block and were replicated using median SRTs. Data was filtered so that saccadic error and trials over 500ms were eliminated from the analysis; saccadic error refers to those trials in which saccades left the fixation area but did not land at the designated target location.

##### *6.2.1.5.1 Reward Paradigm*

Across 12,960 trials, 4.8% were categorised as saccadic errors. 18.5% of trials were above the threshold and also removed from the analysis.

##### *6.2.1.5.2 Peripheral Cueing task*

Of the 6,480 peripheral cueing task trials 4.95% were categorised as saccadic errors and 3.81% of trials were above the threshold and so removed from the analysis.

### 6.2.2 Results

Inferential statistics used a significance correction level of $p < .05$, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*6.2.2.1 Latency*

*6.2.2.1.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed a main effect of Experimental Phase, ($F(2, 22) = 11.55$, $p = <.01$, $r = .59$) such that saccades made during the conditioning phase (248 ms) where rewards were present were significantly faster than saccades made during the preconditioning (265 ms) ($t(11) = 3.65$, $p = <.017$, $r = .74$) and extinction (268 ms) ($t(11) = -5.72$, $p = <.017$, $r = .86$) phases. There was no main effect of Hemifield ($F(1, 11) = 2.46$, $p = .15$, $r = .43$), but there was a trend towards an interaction between Experimental Phase and Hemifield ($F(2, 22) = 3.04$, $p = .07$, $r = .35$).

Planned comparisons found a non-significant difference between saccadic latencies to the rewarded (265 ms) and unrewarded (265 ms) hemifields in the preconditioning phase ($t(11) = .01$, $p = >.017$, $r = <.01$). During the conditioning phase of the experiment, significant differences between saccades to the rewarded (233 ms) and unrewarded (262 ms) hemifields were found ($t(11) = -2.62$, $p = <.017$, $r = .62$). No significant differences were found in the extinction phase ($t(11) = -.69$, $p = >.017$, $r = .20$). Figure 6.2 illustrates this result.

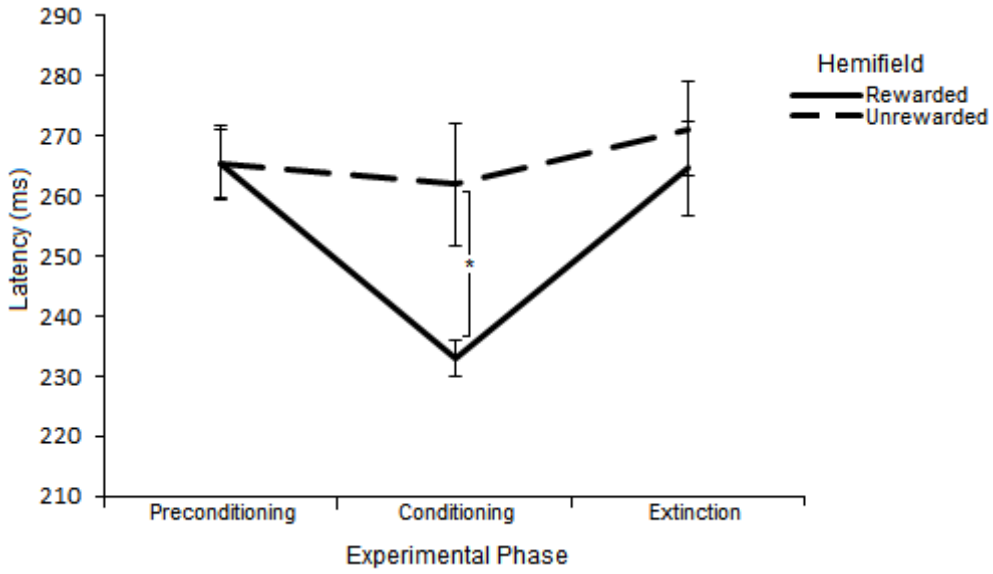


***Figure 6.2:*** *Latency of prosaccades to the rewarded (black line) and unrewarded (black dashed line) hemifields in Experiment 9 across the preconditioning, conditioning and extinction phases of the reward paradigm. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA replicated a main effect of Experimental Phase ($F(2, 22) = 10.53$, $p = <.01$, $r = .57$) such that saccades made during the conditioning phase (247 ms) where rewards were present were significantly faster than saccades made during the preconditioning (265 ms) and extinction (266 ms). There was no main effect of Hemifield ($F(1, 11) = 2.45$, $p = .15$, $r = .43$). An interaction between Experimental Phase and Hemifield was also replicated ($F(2, 22) = 4.42$, $p = .02$, $r = .41$).

Further paired t-tests explored this interaction. In the preconditioning phase a non-significant difference was found between saccadic latencies to the rewarded and unrewarded hemifields in the preconditioning phase ($t(11) = .01$, $p = >.017$, $r = .01$). During the conditioning phase of the experiment, significant differences between saccades to the rewarded (230 ms) and unrewarded (263 ms) hemifields were found ($t(11) = -2.89$, $p = <.017$, $r = .66$). No significant differences were found in the extinction phase ($t(11) = -.41$, $p = >.017$, $r = .12$).

*6.2.2.1.2 Peripheral Cueing Task*

A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 3 (Validity: Valid/Invalid/No Cue) repeated measures ANOVA on mean SRTs revealed a main effect of Validity ($F(2, 22) = 33.35$, $p = <.01$, $r = .78$) where valid trials (308 ms) were significantly faster than invalid trials (327 ms) ($t(11) = -6.82$, $p = <.017$, $r = .90$). No cue trials (301 ms) were found to be significantly faster than invalid trials (327 ms) ($t(11) = 6.89$, $p = <.017$, $r = .90$). Figure 6.3 illustrated this result. No other significant differences were found.

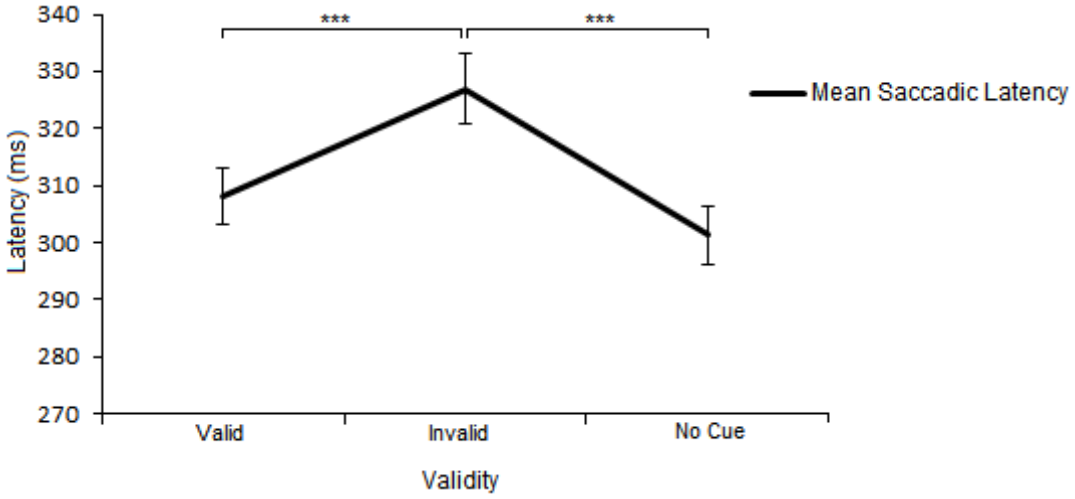


***Figure 6.3:*** *Mean saccadic latency of the three different validity trial types in the attentional capture task employed in Experiment 9. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 3 (Validity: Valid/Invalid/No Cue) repeated measures ANOVA replicated the main effect of Validity ($F(2, 22) = 27.07$, $p = <.01$, $r = .74$) where valid trials (310 ms) were significantly faster than invalid trials (332 ms). No cue trials (301 ms) were found to be significantly faster than invalid trials (332 ms). No other significant differences were found.

### *6.2.2.2 Saccadic Error*

For the purpose of this error analysis, saccadic errors previously excluded from the latency analysis were included. Trials above the set threshold were still excluded.

#### *6.2.2.2.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on the proportion of saccadic errors revealed a significant effect of Experimental Phase ($F(2, 22) = 15.87$, $p = <.01$, $r = .65$). A significantly larger proportion of errors occurred in the conditioning (.022) phase compared to the preconditioning (.004) ($t(11) = -4.17$, $p = <.017$, $r = .78$) and extinction (.015) ($t(11) = 3.15$, $p = <.017$, $r = .69$) phases. Furthermore a significantly larger proportion of errors occurred in the extinction phase

compared to the preconditioning phase (t (11) = -3.99, p = <.017, r = .77). No effect of Hemifield (F (1, 11) = .364, p = .56, r = .18) or interaction between Experimental Phase and Hemifield was revealed (F (2, 22) = .01, p = .99, r = .02).

*6.2.2.2.2 Peripheral Cueing Task*

Using only errors made on invalid trials, where the cue and target are incongruent, the percentage of incorrect trials in each experimental phase was calculated. A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Target Location: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the calculated percentage of incorrect trials in the peripheral cueing task. No effect of Experimental Phase (F (2, 22) = 1.77, p = .19, r = .27), Target Location (F (2, 22) = .96, p = .35, r = .28) or interaction between Experimental Phase and Target Location (F (2, 22) = 2.67, p = .09, r = .44) was revealed.

**6.2.3 Discussion**

The goals of this experiment were to investigate to what extent the effects of rewarding prosaccades influenced exogenous orienting in an unrewarded peripheral cueing task. Facilitation of saccadic eye movements were found in the reward paradigm to rewarded locations, consistent with previous research (Takikawa et al., 2002; Coe et al, 2002; Bowman et al., 1996; Milstein & Dorris, 2007) and the previous reward paradigms utilised in Experiments 2, 6, 7 and 8. Due to the interconnected mechanisms between eye movements and attention it is possible that a combination of attention and motor systems are being biased towards one spatial location due to the presence of reward (Goldberg & Würtz, 1972b; Kowler et al., 1995). However, these effects had limited persistence after rewards were withdrawn. Although a peripheral cueing effect was found in SRTs, with significantly faster saccadic eye movements on valid compared to invalidly cued trials, no effects of rewards were found in saccade latencies in the peripheral cueing task, a result consistent with previous research (Bucker & Theeuwes, 2014; Engelmann & Pessoa, 2007). No effects of reward were revealed in errors in the reward paradigm task or in the invalid trials within the peripheral cueing task.

Even though no effects of reward were found in participant SRTs in the peripheral cueing task, an exogenous facilitation effect was found where validly cued trials were significantly faster than invalidly cued trials, consistent with previous research (Klein, 2000; Posner & Cohen, 1984;

Samuel & Kat, 2003). This effect has previously been explained as the summoning of attention to the location of a target, improving performance at this location, compared to a target presented at an invalidly cued location. Due to the cue appearing in the target location, prior to target presentation, attention has shifted to the locus of the landing point prior to saccade generation, facilitating eye movements to this location (Kristjánsson & Nakayama, 2003; Kristjánsson & Sigurdardottir, 2008; Posner, 1980).

In summary, rewards reproduced the relative facilitation of saccades to the rewarded hemifield in the reward paradigm. However, this effect failed to transfer to the peripheral cueing task further highlighting the task-specific nature of these effects and is consistent with previous experiments (Experiments 6, 7, and 8).

## 6.3 Experiment 10

### 6.3.1 Method

#### *6.3.1.1 Participants*

The sample size of twelve participants used in the previous experiments was replicated. Twelve participants, recruited from the University of Durham, volunteered for the experiment. The participants - five males, seven females – had an age range of 19-25 years (mean age 21.67 years). Nine were right eye dominant: all participants had normal or corrected-to-normal vision and were naive regarding the purpose of the experiment.

#### *6.3.1.2 Apparatus*

Prior to participation participants completed a consent form (see appendix F). The experimental stimuli were generated using a Cambridge Research Systems ViSaGe graphics card and displayed on a 17 inch Eizo Flexscan Colour Display monitor with a refresh rate of 100Hz. Responses were collected using a two-button button box. Eye movements were recorded using a Cambridge Research Systems eye tracker with a sampling rate of 160Hz.

#### *6.3.1.3 Stimuli*

The reward paradigm was replicated from Experiment 2.

In the inhibition task, participants were presented with the same experimental stimuli used in the peripheral cue task.

#### *6.3.1.4 Procedure*

The pre-experimental checks and calibration employed in Experiment 9 were replicated in the present experiment.

##### *6.3.1.4.1 Reward Paradigm*

The reward paradigm was replicated from Experiment 2.

##### *6.3.1.4.2 Inhibition Task*

The inhibition task occurred directly after the preconditioning, conditioning and extinction phases of the reward paradigm. The task itself was unchanged from the peripheral cueing task used in Experiment 9, with the exception of the timings of each trial. After 700ms one of the black-outlined squares changed colour from black to white for a brief period of time (100ms) cueing participants attention towards this location. The fixation cross then pulsated to re-orient participants attention back to the centre of the screen. 600ms after peripheral cue onset the white target square appeared at the same location as the previous colour change for 100ms. On invalid trials, the target square appeared at the opposite location of the previous luminance change. In no cue trials, there was no colour change of the black outlined square and participants had to make an eye movement towards the white target square. Each block consisted of 60 trials equally split between each type of trial. The order in which participants completed each block was randomised to negate any order effects. The cues were non-predictive. Figure 6.4 displays the experimental array.

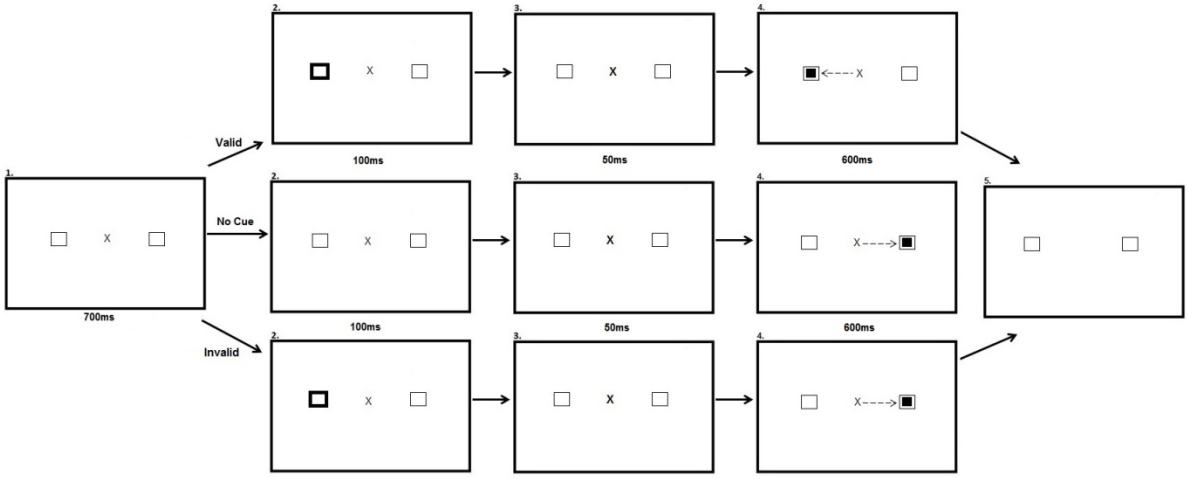


***Figure 6.4:*** *Sequence of events used in Experiment 10 for the inhibition task (not to scale). Participants were presented with a fixation cross and two squares equidistant from the fixation cross in opposing hemifields (Row 1, Panel 1). In valid trials one of the squares changed colour for a period of 100ms, cueing participants to this location (Row 2, Panel 1). Participants are then presented with the same screen as in the first panel for a period of 50ms (Row 3). A smaller target square then appeared in the same location as the cue, 600ms after peripheral cue onset, and participants were required to saccade to this location (Row 4, Panel 1). After making a successful saccade the fixation cross disappeared and the screen changed colour requiring a button press to begin the next trial (Row 5, Panel 1). In no cue trials no cue appeared prior to target onset (Row 2, Panel 2). In invalid trials the cue appeared in one location (Row 2, Panel 3) and the target appeared in the opposite location (Row 4, Panel 3).*

The full experiment ran for 27 blocks and lasted approximately one hour. The running order of the experiment was replicated from Experiment 9.

### *6.3.1.5 Saccade Analysis*

The analysis was conducted on the means of each participant's SRT average calculated from each individual block and were replicated using median SRTs. Data was filtered so that saccadic error and trials over 500ms were eliminated from the analysis; saccadic error refers to those trials in which saccades left the fixation area but did not land at the designated target location.

*6.3.1.5.1 Reward Paradigm*

Across 12,960 trials, 2.35% were categorised as saccadic errors. 15.2% of trials were above the threshold and also removed from the analysis.

*6.3.1.5.2 Inhibition Task*

Of the 6,480 inhibition task trials 13.12% were categorised as saccadic errors and 2.5% of trials were above the threshold and so removed from the analysis.

**6.3.2 Results**

Inferential statistics used a significance correction level of p <.05, except when multiple comparisons were performed, where a Bonferonni correction was applied.

*6.3.2.1 Latency*

*6.3.2.1.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs revealed no effect of Experimental Phase ($F(2, 22) = 2.07$, $p = .15$, $r = .29$). However a main effect of Hemifield was revealed ($F(1, 11) = 5.01$, $p = .04$, $r = .56$) such that saccades made to the rewarded hemifield (239 ms) were significantly faster than those made to the unrewarded hemifield (257 ms). Furthermore, a significant interaction between Experimental Phase and Hemifield was revealed ($F(2, 22) = 4.69$, $p = .02$, $r = .42$). Three separate one-way ANOVAs were conducted on the latency of rewarded and unrewarded hemifields at each level of experimental phase separately in order to explore this two-way interaction. In the preconditioning phase, no significant difference was found between the rewarded (245 ms) and unrewarded (245 ms) hemifields ($F(1, 23) = <.01$, $p = .98$, $r = .01$). In the conditioning phase however, a significant difference between the rewarded (227 ms) and unrewarded (249 ms) hemifields was found ($F(1, 23) = 10.42$, $p = <.01$, $r = .56$). In the extinction phase, a non-significant difference was found between the rewarded (244 ms) and unrewarded (245 ms) hemifields ($F(1, 23) = .02$, $p = .88$, $r = .03$). Figure 6.5 illustrates this result.

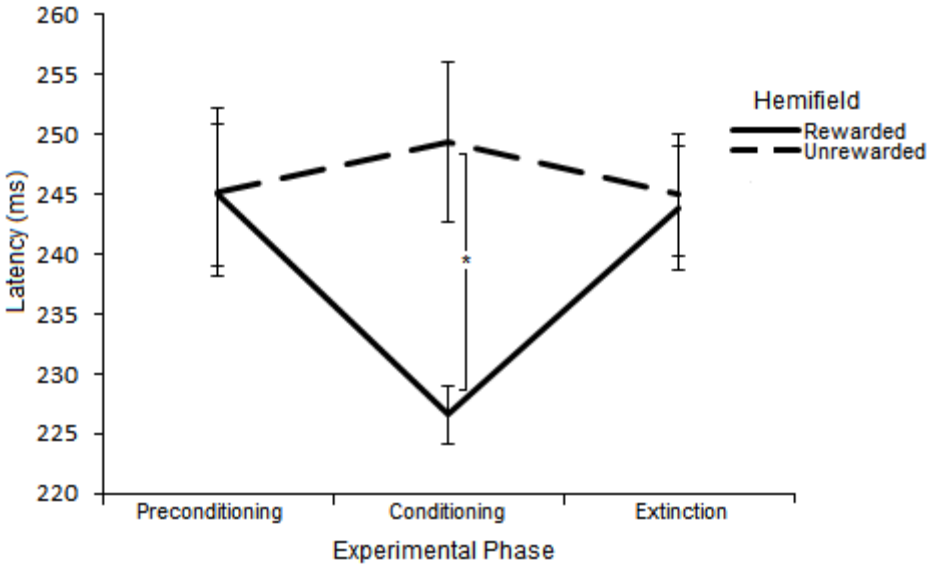


***Figure 6.5:*** *Latency of prosaccades to the rewarded (black line) and unrewarded (black dashed line) hemifields in Experiment 10 across the preconditioning, conditioning and extinction phases of the reward paradigm. Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on mean SRTs replicated no effect of Experimental Phase ($F(2, 22) = 1.97$, $p = .19$, $r = .39$). However the main effect of Hemifield was replicated ($F(1, 11) = 4.16$, $p = .03$, $r = .40$) such that saccades made to the rewarded hemifield were significantly faster than those made to the unrewarded hemifield. Furthermore, the significant interaction between Experimental Phase and Hemifield was replicated ($F(2, 22) = 4.26$, $p = .03$, $r = .40$). Further paired t-tests explored this interaction. In the preconditioning phase, no significant difference was found between the rewarded and unrewarded hemifields ($t(11) = -.05$, $p = .96$, $r = .01$). In the conditioning phase, a significant difference between the rewarded (223 ms) and unrewarded (248 ms) hemifields was found ($t(11) = -2.45$, $p = .03$, $r = .59$). In the extinction phase, a non-significant difference was found between the rewarded and unrewarded hemifields ($t(11) = .47$, $p = .65$, $r = .14$).

*6.3.2.1.2 Inhibition Task*

A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 3 (Validity: Valid/Invalid/No Cue) repeated measures ANOVA on mean

SRTs revealed a main effect of Validity (F (2, 22) = 14.04, p = <.01, r = .62) where saccades made on valid trials (353 ms) were significantly slower than saccades made on invalid (333 ms) (t (11) = 4.19, p = <.017, r = .78) and no cue trials (332 ms) (t (11) = 6.06, p = <.017, r = .88). Figure 6.6 illustrates this result.

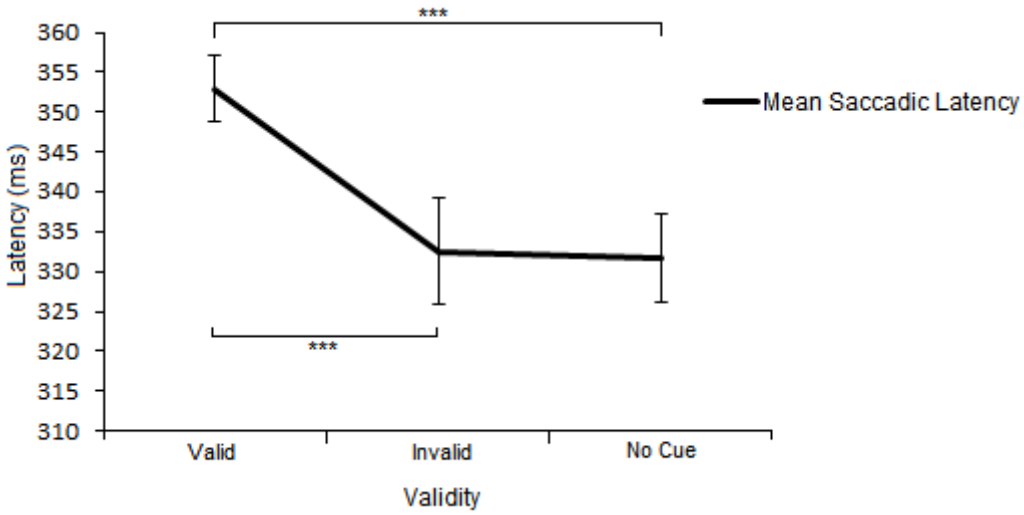


***Figure 6.6:*** *Mean saccadic latency of the three different validity types in the inhibition task employed in Experiment 10. Error bars show +/- 1 SEM.*

A significant interaction between Experimental Phase & Validity was also found (F (4, 44) = 2.66, p = .05, *r* = .24). Three separate one-way ANOVAs were conducted on the latency of valid, invalid and no cue trial types at each level of experimental phase separately in order to explore this two-way interaction. In the post-preconditioning phase no significant difference was found between the valid (346 ms), invalid (338 ms) and no cue (351 ms) trial types (F (2, 35) = .89, p = .42, r = .15). In the post-conditioning phase however, significant differences were found between the different trial types (F (2, 35) = 7.12, p = <.01, r = .77) such that valid trials (362 ms) were significantly slower than invalid (327 ms) (p = <.01) and no cue (333 ms) (p = <.05) trial types. In the post-extinction phase non-significant differences were found between the valid (351 ms), invalid (333 ms) and no cue (327 ms) trials (F (2, 35) = 2.72, p = .08, r = .42). Figure 6.7 illustrates this result.

No other significant interactions were revealed.

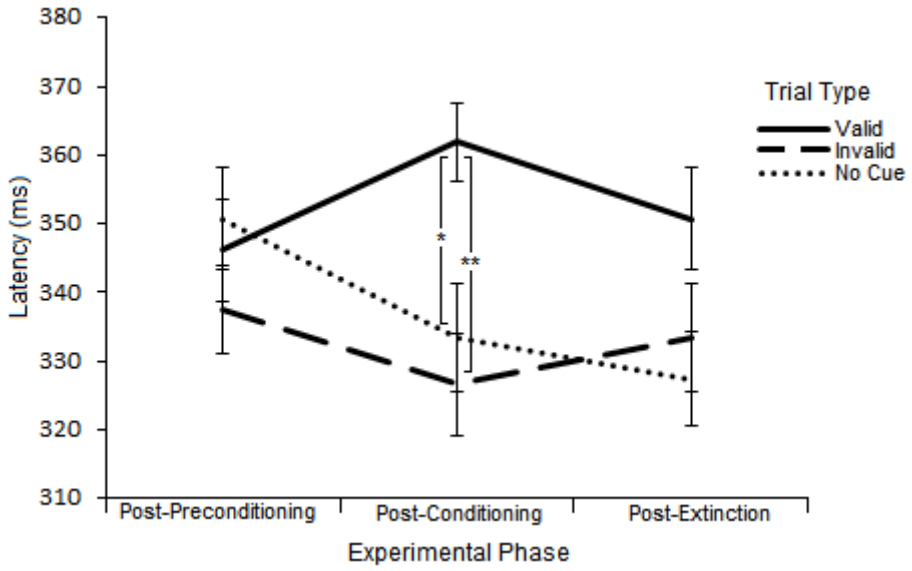


***Figure 6.7:*** *Latency of prosaccades in valid (black line), invalid (black dashed line) and no cue (black dotted line) trials across experimental phases in the inhibition task (IOR). Error bars show +/- 1 SEM.*

Using median SRTs a 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Hemifield: Rewarded/Unrewarded) x 3 (Validity: Valid/Invalid/No Cue) repeated measures ANOVA replicated the main effect of Validity ($F(2, 22) = 12.91$, $p = <.01$, $r = .61$) where saccades made on valid trials (354 ms) were significantly slower than saccades made on invalid (332 ms) and no cue trials (332 ms). Furthermore, the interaction between Experimental Phase and Validity failed to replicate using median SRTs ($F(2, 22) = 2.24$, $p = .08$, $r = .22$).

*6.3.2.2 Saccadic Error*

For the purpose of this error analysis, saccadic errors previously excluded from the latency analysis were included. Trials above the set threshold were still excluded.

*6.3.2.2.1 Reward Paradigm*

A 3 (Experimental Phase: Preconditioning/Conditioning/Extinction) x 2 (Hemifield: Rewarded/Unrewarded) repeated measures ANOVA on the proportion of participant errors replicated previous experiments results revealing no effect of Hemifield ($F(1, 11) = 3.72$, $p = .08$, $r = .50$) and no interaction between Experimental Phase and Hemifield ($F(2, 22) = .92$, $p = .41$, $r = .20$).

*6.3.2.2.2 Inhibition Task*

Using only errors made on invalid trials, where the cue and target were incongruent, the percentage of incorrect trials in each experimental phase was calculated. A 3 (Experimental Phase: Post-Preconditioning/Post-Conditioning/Post-Extinction) x 2 (Target Location: Rewarded/Unrewarded) repeated measures ANOVA was conducted on the calculated percentage of incorrect trials in the spatial cueing (IOR) task. No effect of Experimental Phase ($F(2, 22) = .01$, $p = 1.00$, $r = .02$), Target Location ($F(2, 22) = .23$, $p = .64$, $r = .14$) or interaction between Experimental Phase and Target Location ($F(2, 22) = .19$, $p = .83$, $r = .13$) was revealed.

**6.3.3 Discussion**

In summary, Experiment 10 aimed to investigate the effects of reward on a covert spatial attention task employing the IOR effect. When incentives were present participant SRTs were facilitated to the rewarded location relative to the unrewarded location using the reward paradigm. No hemifield-specific effects transferred to the IOR effect. However, it was evident that after reward feedback the effects of IOR were greater than prior to reward feedback on In mean SRTs, a result consistent with previous research (Bucker & Theeuwes, 2014). Bucker and Theeuwes (2014) employed low and high reward blocks observing cue facilitation for both low and high reward blocks but IOR only in the high reward condition; a finding observed in manual reaction times. The results of Experiment 10 extend Bucker and Theeuwes (2014) findings. In Bucker and Theeues (2014) investigation of motivationally driven influences of reward on spatial orienting in IOR, monetary rewards were presented to participants whilst they completed the exogenous orienting task. In the present experiment, spatial biases induced by reward feedback occurred in a separate task, prior to completing an unrewarded exogenous attention orienting task in participant SRTs. No hemifield-specific effects transferred to the exogenous orienting task. However, a general effect of increased IOR after reward feedback was revealed. This finding extends Bucker and Theewues (2014) research, suggesting that modulation of spatial attention by reward is transient, task-specific and context dependant. After withdrawal of reward, the spatial bias established to rewarded locations extinguished in favour of a more general effect of reward. No effects of reward were found in either task's error data.

## 6.4 General Discussion

The present chapter aimed to extend the effects of reward on covert spatial attention in humans, specifically exogenously cued attention (Experiment 9) and IOR (Experiment 10). In the peripheral cueing task (Experiment 9), SRTs were unaffected by incentives. In contrast, SRTs in the IOR task were affected by rewards, such that IOR was larger after reward feedback was introduced, in the post-conditioning phase. However, this IOR effect was not hemifield-specific.

Although no hemifield-specific effects of reward transferred between tasks, it is evident that inhibition was significantly greater in the post-conditioning phase of the IOR task, compared to the post-preconditioning and post-extinction phases when analysing participants mean SRT responses. As mentioned in the introduction, the IOR effect has been strongly linked with increased activity of the SC (Dorris et al., 2002; Fecatu & Munõz, 2005); a structure also strongly implicated in reward learning (Dommett et al., 2005; Redgrave & Guerney, 2005). It is possible that this structure holds the explanation to this result. Rewards reduce the threshold for saccade generation to occur within the SC. However, after the withdrawal of reward, although no specific effects of reward were present at the spatial location they were previously presented in, it is possible that a more general effect of reward persists whereby the baseline activity of the SC is heightened holistically at the level it has been previously elevated to. This effect was no longer present in the post-extinction phase suggesting that after an extended period of time, the activity within this structure has returned to a baseline level.

This may be due to the recurring use of a target square, used as the target in both the reward paradigm and the IOR task. Previous behavioural studies have identified that targets associated with a reward are more favourable when they reappear again, as learned value increases exogenous oculomotor capture of the eyes (Theeuwes & Belopolsky, 2012). Due to the fact that the cue has become associated with the possibility of reward, it is more salient which would generate a greater IOR effect, irrespective of where it appeared in the visual field. This effect is consistent with Peck, Jangraw, Suzuki, Efem and Gottlieb, (2009) who used a peripheral cueing task where the cue predicted the outcome of the trial rather than the location of the target, in primates. The cue indicated whether the trial would end in a reward or no reward. The authors found that the expectation of reward improved saccadic performance with a higher proportion of completed trials, improved saccadic accuracy and faster SRTs in trials where the cue predicted a reward relative to when it did not. Of most importance however, was the finding that cues predicting a reward spatially biased primate saccades in a valence specific manner such that saccades were facilitated to the location of the cues predicting reward and impaired to the

location of the cues predicting no rewards. Peck et al., (2009) suggested that this result showed that for the rewarded cue, valence specific effects could automatically transfer to a novel context in which the target stimulus did not govern reward expectation. Therefore, long-term training created an intrinsic salience associated with the rewarded cue. The fact that during the IOR task in the present experiment the target is the same stimulus as that previously associated with reward feedback could increase the salience of this particular cue generating a greater IOR effect irrespective of its location in the visual field. However, counter to this argument is the lack of transfer found within participant SRTs in the peripheral cueing task. This finding further highlights the transient nature of the effects of reward.

An alternatve explanation for this increased effect of IOR solely in the post-conditioning phase of the task could be explained by practise effects. Weaver, Lupiáñez and Watson (1998) found that object-based, space-based and static-display IOR all respond to practise effects in the same way; such that IOR decreases in size with relatively small amounts of practise. It is possible that the difference between the post-conditioning and post-extinction phases of the task, such that IOR is significantly greater during the post-conditioning phase and significantly decreased during the post-extinction phase, is the result of participants practising the task and becoming more acquainted with it, reducing the IOR effect in later conditions. However, interpretation of this result should be approached cautiously as the larger effects of IOR were only found in participants mean SRTs and failed to replicate when analysing participants median SRTs.

No reward facilitation effects, specific or general, were found in participants' exogenously cued attention. This result suggests that exogenous attention did not undergo any lasting effects from the reward feedback of the reward paradigm. Furthermore, analysis of invalid trials (when cue and target appeared in opposing hemifields) in both Experiments 9 and 10 revealed no effect of reward on errors in this trial type. If a persistent facilitation effect of reward was evident, this trial type should result in disrupted eye movements when participants were cued to the rewarded location but required to make a saccade to the target in the unrewarded hemifield. The lack of this effect further highlights the short persistence of the effects of reward on eye movements.

In summary, Experiments 9 and 10 have highlighted the effects of reward on covert orienting of attention. No effects of reward were found on the exogenous orienting of attention in either SRTs or error data. In contrast, rewards did affect IOR, exacerbating the effects of IOR in the condition subsequent to reward feedback. These effects generalised across hemifields however, and were not specific to the hemifield in which participants received their rewards. This result suggests that financial incentives do affect eye movements at the level of the SC. The SC is a critical structure

for IOR (Sumner et al., 2004; Dorris et al., 2002; Sereno et al., 2006) and reward (Ikeda & Hikosaka, 2003; Comoli et al., 2003; Dommett et al., 2005; Redgrave & Guerney, 2006) and as such it is likely that this structure is the locus of the effect of reward, subsequently affecting IOR after reward feedback.

# Chapter 7: Summary, Limitations and Future Directions

## 7.1 Introduction

The objective of the experiments presented within this thesis was to address the gaps in the current literature regarding the effects of reward on the human saccadic eye movement system and human attention in the hope that the findings can be used to guide the effectiveness of monetary reinforcers in a neuro-atypical population. The three key questions addressed in the present set of experiments were: 1) do rewards influence the motor and attentional systems in neuro-typical human participants?; 2) how long do these effects persist for when rewards are withdrawn?; 3) do these effects transfer to other eye movement tasks not associated with reward? Within this chapter the findings of experiments 1-10 will be addressed to elaborate on how they have extended previous knowledge regarding the influence of reward learning on the oculomotor and attention system, how this research applies itself within a clinical setting and the potential pitfalls unearthed by the present set of experiments.

## 7.2 Key Findings

Small monetary rewards were found to modulate the latency of saccadic eye movements in neuro-typical human participants consistently when using a reward paradigm where rewards were presented to one hemifield (Experiments 2, 6, 7, 8, 9 and 10). The finding that eye movements were faster to rewarded locations is consistent with evidence in both humans (Milstein & Dorris, 2007) and non-human primates (Bendiksby & Platt, 2006; Takikawa et al., 2002; Kawagoe et al., 1998). However, the present set of experiments has extended these findings by investigating the persistence of these effects once rewards were withdrawn and the transfer of these effects to other unrewarded eye movement tasks. Once rewards were withdrawn, these hemifield-specific effects persisted for a short period of time prior to extinguishing rapidly (Experiment 2). No hemifield-specific effects of reward were found to transfer to SRTs in a secondary, unrewarded eye movement task (Experiments 6-10), extending the findings of previous reward learning research (Milstein & Dorris, 2007; Bendiksby & Platt, 2006; Takikawa et al., 2002; Kawagoe et al., 1998) by showing that the effects of reward are task-specific and short-lived. Only in Experiments 6 and 10 did the influence of monetary incentives extend to the secondary eye movement task, producing faster eye movement to the rewarded hemifield in the trained eye movement and exacerbating the effects of IOR respectively. However these effects manifested in the post-conditioning phase generally and were not hemifield-specific.

In summary, although small monetary rewards were able to consistently produce hemifield-specific effects of reward when incentives were present, these effects were quick to extinguish once rewards were withdrawn and failed to extend to secondary, unrewarded eye movement tasks highlighting the task-specific nature of this finding. Transient transfer of the effects of reward in saccadic error was found in one of the secondary tasks employed (Experiment 6). These results highlight the sensitivity of the effects of reward learning. The following discussion will examine these findings in greater detail.

### 7.3 Rewards modulate saccades when reward feedback is present

The use of the reward paradigm within Experiments 2, 6, 7, 8, 9 and 10 was consistently able to facilitate participants' eye movements to rewarded locations, relative to unrewarded locations when monetary incentives were available. It is well documented that the SC is a structure in the brain heavily linked with the encoding and processing of reward information (Ikeda & Hikosaka, 2003; Comoli et al., 2003) particularly reward learning (Dommett et al., 2005; Redgrave & Guerney, 2006) and with the generation of saccadic eye movements (Li & Basso, 2008; Mays & Sparks, 1980; McPeek & Keller, 2002). Based on this research highlighting this structure as the link between eye movements and reward, it can be speculatively suggested that this structure is the likely locus of the facilitation effects recorded in Experiments 2, 6, 7, 8, 9 and 10.

It can be suggested that rewarding one hemifield alters the state of neurons within certain structures outlined in Chapter 1. For example, rewarding one hemifield during the conditioning phase of the reward paradigm may activate the contralateral caudate neurons, inhibiting neurons of the ipsilateral SNr (Sato & Hikosaka, 2002). Decreased activity within the SNr would lead to a disinhibition of SC neurons, making it easier for these neurons to reach the threshold required for saccadic execution (Ikeda & Hikosaka, 2003). Simply, the small monetary rewards employed in the reward paradigm may alter the equilibrium of activity in the SC, facilitating saccades to rewarded locations. In contrast, the caudate neurons responding to the unrewarded hemifield are relatively less active leading to disinhibition of SNr neurons. The SC neurons are subsequently kept inhibited requiring a relatively longer duration to reach the threshold necessary for saccade execution. This account of the facilitation effect is consistent with threshold models of saccade generation discussed in Chapter 1 (Godijn & Theeuwes, 2002; Trappenberg et al., 2001). This effect can also be explained within Findlay and Walker's (1999) model of saccade generation. Findlay and Walker (1999) proposed that the intrinsic salience of a visual stimulus can impact on the automatic

processing of that stimulus, directly influencing the execution threshold of a saccade. In rewarding participants' eye movements, the competitive interaction between the rewarded and unrewarded hemifield would lead to an equilibrium shift with the ability to reach threshold occurring faster for rewarded, compared to unrewarded, hemifield targets resulting in the finding of the reward paradigm in Experiments 2, 6, 7, 8, 9 and 10. This explanation can also account for the results recorded in Experiment 1. Rewarding both hemifields may have resulted in no equilibrium shift in the activity of the SC and as such both sides of the SC would be excited in relation to the reward feedback being received. Therefore, neither hemifield receives facilitated eye movements. However, the effects of reward have a more general facilitation of eye movements speeding saccades more generally in phases after reward feedback.

Further evidence for the role of the SC in this effect comes from Lucas et al., (2013) who found rewards induced biases in spatial orienting in right brain damaged patients despite their left spatial neglect. This result suggests the effects of reward are at least partially independent of brain systems mediating the attention functions impaired in spatial neglect, such as the fronto-parietal networks normally associated with top-down attention control or exogenous orienting (Corbetta & Schulman, 2001). Therefore, Lucas et al., (2013) suggested that reward reinforcement mechanisms could modulate space representations and their link with exploratory motor output through distinct networks, possibly implicating subcortical pathways in the striatum and crucially the SC (Ding & Hikosaka, 2006; Lauwereyns et al., 2002; Weldon, DiNieri, Silver, Thomas & Wright, 2007). Furthermore, exploratory anatomical analysis of their patient cohort indicated that lesions extending to the basal ganglia and frontal lobe led to weaker reward effects. These lesions could interrupt projections from either the OFC or caudate to more posterior regions in parietal and visual cortices and to subcortical oculomotor circuits in the SC (Sato & Hikosaka, 2002; Hikosaka et al., 2006). This in turn could lead to impaired processing and interruption of motivational signals with representations of space and motor action, abolishing the spatial biases induced by asymmetric reward contingencies during visual exploration (Kawagoe et al., 1998; Lauwereyns et al., 2002; Ding & Hikosaka, 2006; Hikosaka et al., 2006). However, this data was acquired from a small sample and this research is still in its infancy. The findings of Lucas et al., (2013) provide further evidence that the locus of the effects found in the reward paradigm may possibly alter the neuronal states in the SC and its various projections mentioned in detail in the Chapter 1, highlighting this structure as a possible locus of this behaviour change found in Experiments 2, 6, 7, 8, 9 and 10.

### 7.4 The time-course of reward learning

The present set of experiments has extended the knowledge of the time-course of reward learning in the human oculomotor system and attention. When the reward paradigm was employed on its own as a singular task, the results differed when compared with using it in conjunction with another eye movement task. In Experiment 2 using the reward paradigm on its own resulted in a persistence in the effects of facilitation after rewards were withdrawn for three blocks; a period of approximately ten minutes. It is clear that without altering the demands of the task and with participants making the same type of eye movement, facilitation effects persist after rewards are no longer available. This finding suggests that the reward paradigm induces habits, rather than goal-directed changes in the human saccadic eye movement system (see Chapter 1 for review). However, introduction of secondary, unrewarded eye movement tasks extinguishes these hemifield-specific effects. It is possible that altering the task demands results in the lack of transfer and persistence of these hemifield-specific effects subsequently abolishing these previously created habits. This highlights the transient and fragile nature of the effects of reward on the oculomotor system.

Further to this explanation, it is possible that the use of monetary rewards alter the motivational states of participants adversely affecting their motivation to complete tasks once rewards are withdrawn; a phenomenon termed the overjustification effect (Morgan, 1981). The overjustification effect suggests that more attention is paid to the external reward being attained than completing the task at hand, shifting participants' extrinsic motivation and undermining their pre-existing intrinsic motivation. Once rewards are no longer available, interest in the task is lost and the prior intrinsic motivation does not return. The theory suggests that extrinsic rewards must be continuously offered as motivation for the individual to sustain their performance in the activity. Applying this hypothesis to saccadic eye movements grants an explanation of the limited persistence of the facilitation effects into the extinction phase of the reward paradigm and the lack of transfer of these hemifield-specific effects to an unrewarded secondary task.

A further explanation for the lack of hemifield-specific effect after the withdrawal of reward can be attributed to a number of variables, including individual attributes of the participants' and task variables. Individual, or 'person', variables can affect the influence of rewards on behaviour. For example, an individual's motivation or risk preferences will determine how they react to receiving rewards. Furthermore, the complexity of a task can decrease the amount of effort an individual exerts, leading to decreases in performance over a period of time. Although rewards are able to influence and motivate behaviour in one individual, it is not certain this will generalise to the

whole population (Camerer, 1995). Research has previously highlighted the effects of money can be transient in experiments not specific to saccades, with incentives not improving or affecting performance in tasks and not being specific to eye movement. Bonner, Hastie, Sprinkle and Young (2000) presented a review of laboratory studies that used financial rewards to incentivise behaviours. In only half of the studies reviewed monetary incentives led to significant performance improvements. In some studies reviewed, incentives were able to produce positive effects. However, these effects were variable and hard to predict, with other studies failing to find any effects of reward (Lee, Locke & Phan, 1997; Wright, 1989, 1990, 1992). Although this review is not specific to the effects of reward on eye movements, it is important to consider the conflicting evidence as some studies suggest that the effects of reward can influence behaviour whilst others report that these effects do not exist. This is consistent with the findings of the present set of experiments. Although when rewards were present, the effects of facilitation towards the rewarded hemifield were consistent, removing rewards resulted in a transient and inconsistent persistence of these effects.

### 7.5 Transfer of reward modulation to unrewarded eye movements

Although no hemifield-specific transfer of the effects of reward were revealed, a more general effect of incentives was found in Experiments 6 and 10. In Experiment 6, saccades were significantly faster to the rewarded hemifield than the unrewarded hemifield in prosaccade latencies when analysing median SRTs. Although eye movements transferred to the trained eye movement this data failed to replicate when analysing mean SRTs or the results of Experiment 7. In Experiment 10, an exacerbated IOR effect was revealed in conditions subsequent to the availability of reward feedback. Additionally, participant errors displayed specific effects of reward in Experiment 6. These results and what they suggest in relation to the influences of reward on attention will be discussed in the following section.

A general effect of monetary rewards was found in SRTs during the IOR task (Experiment 10) such that a greater IOR effect was found in conditions after reward feedback was available. The root of this result may lie within the orbital frontal cortex (OFC) which has been identified as a crucial structure in the process of reward valuations (Padoa-Schioppa & Assad, 2006). Neurons within the OFC are also able to distinguish between rewards and punishers (Thorpe, Rolls & Maddison, 1983) and the OFC is able to encode stimulus-reward value from secondary rewards, such as money (Elliott, Newman, Longe & Deakin, 2003). It has also been suggested that the OFC mediates IOR based on recent associations between behaviour and reward (Hodgson, Li, Tada & Blow, 2002).

Therefore, based on these two key functions it is possible that this effect is the result of an alteration in activity within this structure leading to increased inhibition in conditions after reward feedback. However, without further experimentation regarding this result it is hard to draw any clear conclusions regarding the OFCs role in this result as this brain region is one of the least understood in the human brain. A further possible explanation for the generally larger inhibition effects arises from previous publications suggesting that rewards alter the salience of stimuli (Della Libera & Chelazzi, 2009; Anderson et al., 2011a; Hickey et al., 2010a; Theeuwes, 1991, 1992; Theeuwes & Belopolsky, 2012), such that when they reappear they increase exogenous oculomotor capture of the eyes across the visual field, generating a greater IOR effect irrespective of where the target appears in the visual field.

In only one experiment rewards were able to influence saccadic accuracy (Experiment 6) resulting in greater capture of prosaccades towards the previously rewarded location, which were subsequently corrected on antisaccade trials. This result is interesting as it suggests that the reflexive prosaccade was influenced by rewards prior to the generation of an antisaccade. Interestingly this result failed to replicate in Experiment 7 despite the same experimental setup. This finding highlights that the effects of rewards are not limited to influencing SRTs but can also lead to greater oculomotor capture of locations or stimuli associated with rewards. However, the fact that this only occurred in one experiment, whilst all other experiments where a facilitation effect of reward was produced resulted in no effects on saccadic errors, further highlights the fragility of this effect.

### 7.6 Implications for the Premotor Theory of Attention (PMT)

The findings of Chapter 6 have implications for the premotor theory of attention (PMT). The PMT is comprised of four key assumptions. Firstly, it is assumed that spatial attention is a consequence of activating neurons located in spatial maps used to plan movements. As such, it is assumed that selective attention and movement planning use the same neural substrates and as such there is no independent attention system. The second assumption is that activation of these neurons depends on the preparation to perform goal-directed spatially-coded movements. Therefore, the theory assumes that spatial attention is the consequence of planning goal-directed actions. The third assumption is that different spatial pragmatic maps become active according to the requirements of the task. Spatial attention can therefore potentially originate from any effector system capable of performing a goal-directed action. The final assumption is that the oculomotor system has a privileged role in selective spatial attention. The presence of rewards bias eye

movements to the rewarded location in the reward paradigm. The mechanisms underlying eye movements and attention are fundamentally interconnected (Goldberg & Würtz, 1972b; Kowler et al., 1995; Gee et al., 2002). One view is that covert attention and eye movement planning are the same thing, sharing the same neural substrates and as such there is no independent attention system; assumed by the PMT. However an alternative view is that exogenous attention is linked to motor control, whereas endogenous attention is not (Smith & Schenk, 2012; Klein, 1980). The PMT suggests that changes in the oculomotor system should affect exogenous attention as the theory assumes that spatial attention is a consequence of activating neurons in spatial maps to plan movements. The assumption that the motor system is necessary and sufficient for spatial attention has been supported behaviourally with a number of studies showing that attention is locked to the goal of eye movements prior to the onset of a voluntary eye or arm movement (Baldauf & Deubel, 2008a, 2008b; Deubel, 2008; Deubel, Schneider & Paprotta, 1998; Dore-Mazars, Pouget & Beauvillain, 2004; Hoffman & Subramaniam, 1995; Shepherd, Findlay & Hockey, 1986). Therefore, if the PMT is correct, changes observed in the oculomotor system should also affect exogenous attention. Instead the findings of Chapter 6 observe that changes in the oculomotor system, evident in the conditioning phase of the reward paradigm, had no effect on exogenous attention (Experiment 9), even though it did produce larger effects of inhibition (Experiment 10). The findings of Chapter 6 are therefore incompatible with a strict version of the PMT.

### 7.7 The transient nature of rewards

The present thesis has found rewards to induce transient transfer of effects through the use of different oculomotor tasks. For example, a general transfer of reward was found in Experiments 6 and 10. However, in Experiments 7, 8 and 9 no transfer of the facilitation effect was found in participant SRTs. Furthermore, altering the reward paradigm to pair rewards with an auditory tone in Experiment 3 resulted in abolition of the facilitation effect entirely. These findings emphasise the transient nature of rewards.

One explanation for these inconsistent effects can be attributed to the nature of the reinforcer itself. Money is an example of a secondary reinforcer and its value is more abstract and cognitive in nature. Previous evidence has highlighted that incentives do not always generate a behaviour change and instead can have variable effects, generating improved performance in tasks and behaviour and at other times failing to generate any effect at all (Lee, Locke & Phan, 1997;

Wright, 1989, 1990, 1992). These results permit explanation of the transient nature of the transfer of rewards. In some cases a general effect of reward did persist in participant SRTs and at other times this was not the case. The evidence provided in this thesis highlights that care needs to be taken when considering monetary rewards as a reinforcer for human behaviour. The one caveat with this particular argument is that although the effects of reward displayed an inconsistent transfer into other oculomotor tasks, the consistent effects of facilitation were present when reward feedback was available. Therefore, when monetary rewards are present, behaviour is consistently altered.

In an effort to enhance the effects of facilitation the reward feedback was paired with an auditory tone in Experiment 3. Previous experiments have highlighted that pairing a reward with a sound after a saccade to a target stimulus increases the strength of the conditioned stimulus (Harrington & Peck, 1998; Hughes et al., 1998; Steenken et al., 2008; Corneil et al., 2002) as the paired stimulus gains value based on its association with the reinforcer. After participants' made a saccade to a rewarded visual target, they would see the reward feedback and hear a 1 kHz auditory tone simultaneously. It was expected that this would produce significantly faster eye movements to rewarded stimuli and to the rewarded hemifield, due to the multisensory nature of the conditioned response. However, Experiment 3 failed to find an effect of reward previously present without an auditory tone. Instead of facilitating, the auditory tone disrupted saccadic performance. A control experiment (Experiment 4) suggested that the 1 kHz auditory tone used was the reason for this result. However, this also displays the transient nature of the effects of reward. Any addition or change to the schedule is able to disrupt it and instead abolish the previously found facilitation effects of reward. Therefore, it is important to recognise the fragility of this effect. Although it is able to be established when rewards are present, alterations in the demands of the task or stimuli used result in this effect not appearing or extinguishing.

A further interpretation of these transient effects could be explained by the differences that occur when rewarding a location compared to rewarding stimulus features. The effects found within this thesis highlight that monetary rewards failed to persist when a spatial location was incentivised. However, previous studies have found that when a feature is rewarded consistently eye movements are significantly speeded to that feature and persist for up to a week afterwards (Della Libera & Chelazzi, 2009). This highlights the possible differences between how reward affects spatial represenations as opposed to feature representations. It is well-established that the processing of features occurs within cortical areas such as the primary visual cortex (Yantis & Serences, 2003; Rossi & Paradiso, 1995; Maunsell & Treue, 2006). Speculatively, these cortical

areas may be more susceptible to the effects of reward and as such instrumental conditioning may result in long-lasting effects when rewarding stimulus features but not spatial locations. This explanation is valid when considering how individuals complete tasks in everyday life. For example, when searching the visual scene for a missing item it is more beneficial for the viewer to be biased by the features of the stimuli to enable faster search. Instead, if the individual is biased by a location in space, this can result in an extended search time or complete inability to find the required item. Furthermore, short-term location-based saliency is beneficial when considering how events occur during natural multiplexed scenarios. When driving a car sudden unexpected events could occur at any time. In this scenario it would be of greater benefit to the viewer to have short-term location saliency in order to attend to these events and act accordingly rather than a longer-term bias towards a particular spatial location that could result in missing the event or have devastating conequences for the driver. In this way, the differences between how reward affects space and feature-based representations can be interpreted with reference to natural scenarios. This research has extended the knowledge of money as a reinforcer in human participants and how these effects persist into untrained tasks and over time.

### 7.8 Summary of the transfer, persistence and time-course of reward modulation

In conclusion, the present set of experiments has extended the previous knowledge held regarding the effects of reward in a human population in three key ways. Firstly, a reward schedule was generated which was able to consistently alter the saccadic eye movement behaviour of neuro-typical human participants. Using a 30% reward schedule produced consistent facilitation to rewarded locations relative to unrewarded locations suggesting that it is possible to reliably induce visual biases to spatial locations in human participants when reward feedback was present. Secondly, the time-course of these effects has been explored within the same context as the reward conditions and also through the use of other oculomotor tasks. When no changes in the demands of the task occur, the facilitation effect persists for a period of approximately ten minutes, prior to extinction. However, alterations in the task demands or changes to task stimuli results in the instant extinction of hemifield-specific effects. Finally, in the majority of experiments the effects of reward previously found when reward feedback was present, failed to transfer to a secondary eye movement task. When this effect did transfer it transferred in only a more general manner (Experiments 6 and 10). The effects of reward are transient and fragile such that any transfer may not generalise across a population.

## 7.9 Limitations

Although the present set of experiments has extended the findings of the current reward literature one important limitation to consider was the large error rates across all experiments. A strict 500 ms threshold was applied to the experiments in order to remove saccades that were not stimulus-elicited (Walker et al., 1995). The tasks were well explained prior to participation by both an information sheet attached to the consent form (see Appendix A-F) and the experimenter. Participants stated they found the tasks straightforward regardless of the number of trials that were omitted. The largest number of errors was found when the reward paradigm was paired with a secondary unrewarded eye movement task. One possible explanation for this limitation may be due to the number of trials participants were required to complete in experiments where a secondary task was employed. Over the course of these experiments, participants made between 1,080-2,160 eye movements depending on which experiment they took part in. It is possible that the large number of erroneous eye movements was the result of oculomotor fatigue (Vienne, Blondé & Doyen, 2012).

While acknowledging this criticism, the use of these experiments has resulted in an extension of the previously held knowledge regarding the influence of rewards on human oculomotor control and attention systems. Further exploration of these particular paradigms, via replication or through a control experiment, would be a viable option for future research to further probe the relationship between rewards and saccade competition and inhibition.

## 7.10 Applications

Recent experimentation with rewards has focussed on the potential uses of money as a viable rehabilitator in visual field deficits. Malhotra et al., (2013) has shown that omissions in a cancellation task were reduced for both left and right targets when patients searched for pictures of coins and were promised monetary rewards for every target found, relative to a no reward condition. However, it is important to note that the effects reported in this study could be explained by more general motivation and arousal factors and may not be necessarily related to stimulus or location-specific associations acquired through reward reinforcement (Robertson, Mattingley, Rorden & Driver, 1998). To alleviate this criticism, Lucas et al., (2013) investigated the specific effects of reward on spatial attention using a novel gambling task in a population of neuro-typical and neglect patients. In the neuro-typical cohort, making rewards available to both

hemifields resulted in no change in oculomotor behaviour; a result consistent with Experiment 1. However, presenting high value rewards to one hemifield resulted in a progressive shift of target choices to that hemifield, correlating with the data presented in Experiment 2. In the patient sample, initial target selection was governed by their deficits. However, patients gradually shifted their target choices to their impaired visual field, where the highest rewards were available. These findings revealed that asymmetric reward distribution in space could bias visual exploration and target selection in both neuro-typical and a patient population alike.

The experiments within the thesis extend the knowledge of the influence of money in a neuro-typical population of humans and provide a clear argument against the potential use of rewards as a rehabilitative tool for sufferers of neglect. The experiments documented within the thesis using the reward paradigm have provided two key pieces of evidence supporting this argument. Firstly, although the facilitation effects of reward feedback persist for a period of approximately 10 minutes in humans, this is without any changes in the demands of the task. This suggests that the hemifield-specific effects of reward are task-specific and as such are bound within the context of the rewarded task. Within a neuro-typical cohort, when reward feedback was available, consistent facilitation of the eyes was recorded to rewarded locations, relative to unrewarded locations (Experiments 2, 6, 7, 8, 9 and 10); an effect that persisted for a short period of time after rewards were withdrawn. However, this result suggests that context is an important factor in the reinforcement of saccadic behaviour. Only under the conditions in which the behaviour was reinforced did hemifield-specific effects persist. This result has implications for the effectiveness of monetary rewards as a rehabilitator as changes in behaviour will fail to persist after the training period. Secondly, the facilitation effects transfer inconsistently into other laboratory based eye movement tasks and when they do transfer, they only do so in a general manner. After rewards are withdrawn, no hemifield-specific effects occur in SRTs and any transfer is inconsistent and may not replicate to other tasks, or tasks outside of the lab. This emphasises the importance of context in reward learning as it is only in the same context in which facilitation effects are found that they persist. Therefore, the use of rewards in rehabilitation may suffer from a lack of applicability to tasks outside of the lab. However, it is important to note that all participants in the present set of experiments were neuro-typical humans so any statements made regarding the effectiveness of incentives in a lab setting are speculative until investigated in a patient cohort. This is true when considering the motivation regarding participation in the experiments within the thesis. The populations tested in the thesis were predominantly undergraduate students required to participate in order to reach the required level of participation for the academic year. The secondary motivation would have been the monetary rewards on offer. It is important to discuss

the differences between this population tested and the population which will benefit from the questions posed within this thesis. Individuals who suffer from visual field deficits, specifically homonymous hemianopia, cope with this extremely debilitating deficit everyday. Simple tasks that can be easily managed and completed by individuals with normal vision, such as crossing the road, driving and navigating obstacles become difficult or impossible depending on the severity of the visual deficit. Therefore, this population can be left isolated, with limited independence and reliant on carers or family members for assistance. Simply, inidividuals with visual field deficits would be motivated to take part in any task if they believed it would increase their visual field and as such their quality of life. Therefore, although the present experiments failed to suggest that monetary rewards would have persistent effects if used in rehabilitation, we can speculate that using a population of individuals who constantly suffer from a debilitating deficit would result in increased motivation to participate and as such, different results.

### 7.11 Future Directions

It is clear that the evidence provided in the present set of experiments has extended the previously held knowledge regarding the influence of rewards on the oculomotor system and its influences on attention. However, the research has also raised further avenues of investigation that should be addressed in order to fully understand the extent to which rewards modulate eye movements and attention and whether adaptations to the present reward schedule can create consistent effects of behaviour change.

Chapter 2 aimed to build a reward schedule able to alter the saccadic behaviour of participants by associating one spatial location with monetary incentives. Three experiments were conducted varying the stimuli and schedule of rewards to create the optimal paradigm. Although a paradigm was created, able to consistently change the oculomotor behaviour of participants, it is possible that further changes to the schedule may result in different, even greater effects. For example, using a greater amount of money than the visual ‘10p’ reward feedback may result in increased effects of reward as a larger value of reward is being attributed to a spatial location. Speculatively, this may also result in faster extinction effects when rewards are withdrawn as eye movements to rewarded locations are more highly rewarded so the effects of withdrawal are more noticeable. In addition, the intrinsic motivation to complete the task may deplete at a faster rate if higher value rewards are employed, as suggested by the overjustification effect (Morgan, 1981). Therefore, experimentation with varying amounts of visual feedback may be beneficial in deciphering the optimal effect of rewards on the oculomotor system.

In addition, the reward paradigm employed in Experiment 3 was created to enhance the previous facilitation generated in Experiment 2. However, the reverse result was found, with abolition of the facilitation effect and instead slower saccades to rewarded locations paired with an auditory tone. Revisiting this experiment and using a different tone, such as the sound of a cashier's till, may generate the expected result suggested when pairing stimuli from different modalities in the same spatial and temporal proximity; faster eye movements to visual targets (Hershenson, 1962; Simon & Craft, 1970; Colonius & Diedrich, 2002; Colonius & Arndt, 2001). With a more favourable sound being used, and one that is often associated with money, may permit the stimuli to be perceived more positively in comparison to the negative perception of the 1 kHz auditory tone (Experiment 4). Therefore, revisiting the reward paradigm should be a target for research in the near future as the effects of an optimal reward paradigm could have great rehabilitative benefits for the future.

Although a consistent effect of rewards was found when reward feedback was available, the individual difference in participants' reaction to reward cannot be overlooked. Monetary rewards are a secondary reinforcer and are a gateway for organisms to obtain primary reinforcers. Their value is subjective depending on the perception of the individual. Therefore, the total sum of £18 offered for the reward paradigm may mean a great deal more to one participant when compared with another. A greater consideration for this factor may be worthwhile when conducting an experiment using monetary rewards. Further to this point, a future direction investigating the individual personality differences and the effectiveness of financial reinforcers may tease apart subtle differences between those more susceptible to monetary reinforcers.

Throughout the thesis the effects of reward found in Experiments 2, 6, 7, 8, 9 and 10 has been discussed in terms of specific neural pathways and structures, with particular emphasis on the SC, and how rewards have affected activity within these areas. Without specifically conducting this research, only speculation can occur regarding what is actually being affected. The reward paradigm is a simple eye movement task and therefore the use of this paradigm in conjunction with an fMRI study would enable specific conclusions to be drawn regarding the structures involved in the facilitation effect found in the reward paradigm employed in Experiments 2, 6, 7, 8, 9 and 10.

A further planned experiment that could not be completed due to time constraints was to examine the effects of the reward paradigm on pseudo-neglect. Pseudo-neglect is a mild asymmetry in spatial attention in neurologically normal individuals, whereby the left hemi-space is favoured, leading to a small leftward bias exhibited in classical tasks of neglect, such as line

bisection (Bowers & Heilman, 1980; Halligan & Marshall, 1989a, 1989b). Using the reward paradigm in conjunction with a paper and pen line bisection task on neuro-typical participants would reveal whether or not the reward paradigm was able to bias participant's pre-existing visual equilibrium, increasing pseudo-neglect or negating it, depending on the direction of the rewarded hemifield. However, based on the experiments presented within the thesis, it is possible that the effects would fail to transfer to the line bisection task. However, this further experimentation may provide evidence of whether the effects transferred to manual tasks and an insight into whether the reward paradigm would alleviate pseudo-neglect allowing further conclusions to be drawn regarding the usefulness of monetary rewards as a tool for behaviour change.

### 7.12 Summary

The present research suggests that monetary rewards can generate a consistent spatial bias towards a spatial location associated with financial gains, relative to an unrewarded spatial location. This finding is consistent with previous studies that have highlighted the influence that rewards can have on saccadic latencies (Milstein & Dorris, 2007; Jazbec et al., 2006) and bias eye movements to certain spatial locations (Camara et al., 2013). However, the experiments within the thesis have extended the previous knowledge held regarding the effects of reward by charting a time-course of these effects and investigating their transfer into other unrewarded eye movement tasks. When no alterations in task demands occur, the hemifield-specific effects of reward are found to persist for a short period of time after rewards are withdrawn before extinguishing rapidly. In this respect, the time-course of the effects of rewards can be clearly seen. However, changing the context under which the behaviour was rewarded led to automatic extinction of the hemifield-specific effects previously observed when rewards were present. In this respect, the factor of context is critical in reward learning. Although no specific effects of reward transferred to secondary eye movement tasks, in one case a general effect of rewards was found, with an increased IOR effect (Experiment 10). It is important to recognise that although an effect of transfer occurred in this experiment, no other experiments found any transfer of the influence of rewards on SRTs. Furthermore, the effects of reward transferred to saccadic error in only one of the experiments (Experiment 6) such that the rewarded hemifield disrupted eye movements in the subsequent eye movement task. This highlights the inconsistency of the transfer of these effects of reward when altering the demands of a task.

The findings of the present set of experiments extend previous knowledge and have wide reaching implications for the future of rehabilitation research in visual field deficits. Although a consistent effect of hemifield bias was present when reward feedback was available, withdrawal of rewards and the addition of a secondary task resulted in inconsistent transfer. Therefore, this research suggests that monetary incentives are not a viable reinforcer in patients with visual field deficits, as suggested in a number of recent publications (Malhotra et al., 2013; Lucas et al., 2013). The inconsistent transfer of hemifield-specific effects and the uncertainty regarding the generalisation of these effects between individuals is suggestive of the need for further research to be conducted and possibly for alternatives to be sought. Future research should address this criticism with further investigation of the reward paradigm. It is possible that altering the value, changing the visual feedback or the addition of a more pleasant auditory tone could optimise the paradigm and result in different and possibly more consistent changes in behaviour. A final consideration is that monetary incentives may not be the most suitable reinforcer in the rehabilitation of visual field deficits. The value of money is subjective, and therefore one individual's perception of a value will be entirely different to another individual. This notion complements a review of studies that have used incentives to change behaviour, with significant performance improvements found in only half of the studies reviewed (Bonner et al., 2000). At times incentives produced behavioural change and improvements in performance but at other times, no changes were revealed (Lee et al., 1997; Wright, 1989, 1990, 1992). Moving forward, it is possible that rewards produce a short-term behaviour change but only in the context in which they are received. Based on the findings of the present research, financial incentives may not be a viable reinforcer for long-term behaviour change unless further investigation of reward schedules exploring variable-reward schedules able to produce optimal behaviour change takes place. Future research should aim to address the questions of whether the facilitation effects found in the present set of experiments can be enhanced using different schedules to increase the duration of these effects prior to extinction and exploration of the neural processes involved in the effects found in order to relate these results to previous neurological findings. However, the present thesis has made headway in identifying an effective reward schedule that produces persistent, short-term behaviour change and extended the present knowledge regarding the influences of reward on saccadic eye movements and as such, human behaviour.

**Appendix A – Experiments 1 & 2 Consent form**

***Participant Consent Form***

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Within this task you will be presented with a single visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. This trial contains three experimental phases:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Reward Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will receive a reward for your eye movement.

3. **No Reward Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no reward is available in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment. The experimenter will tell you which phase you are completing. Measurements will be taken using an eye-tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time
* without having to give a reason for withdrawing
* without any adverse result of any kind? YES / NO

Signed…… Date…….....

**Appendix B – Experiment 3 Consent form**

***Participant Consent Form***

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Within this task you will be presented with a single visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. This trial contains three experimental phases:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Reward Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will receive a reward for your eye movement and hear an auditory tone.

3. **No Reward Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no reward is available in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment. The experimenter will tell you which phase you are completing. Measurements will be taken using an eye-tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time
* without having to give a reason for withdrawing
* without any adverse result of any kind? YES / NO

Signed…… Date…….

**Appendix C – Experiment 4 Consent form**

***Participant Consent Form***

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Within this task you will be presented with a single visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. This trial contains three experimental phases:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Auditory Feedback Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will hear an auditory tone.

3. **No Auditory Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no sound will be played in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment. The experimenter will tell you which phase you are completing. Measurements will be taken using an eye-tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time
* without having to give a reason for withdrawing
* without any adverse result of any kind? YES / NO

**Signed** ..... **Date.....**

**Appendix D – Experiments 6 & 7 Consent Form**

***Participant Consent Form***

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Today you will be required to complete two tasks.

**Task One**

You will be presented with a visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. There are three phases within this task:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Reward Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will receive a reward for your eye movement.

3. **No Reward Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no reward is available in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment.

**Task Two**

In the second task you will be presented with a visual stimulus either on the left or right side of the screen and are required to make an accurate eye movement towards, or away,

from this stimulus depending on what trial you are undergoing. In this task there are two types of trial:-

1. Prosaccade trials: When you see a **BLUE** fixation cross you are required to make an eye movement towards the target
2. Antisaccade trials: When you see a **PURPLE** fixation cross you are required to make an eye movement to the opposite side of the screen that the target is presented to

The experimenter will tell you what task you will be experiencing throughout the experiment. Measurements will be taken using an eye tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time
* without having to give a reason for withdrawing
* without any adverse result of any kind? YES / NO

**Signed** ..... **Date.....**

## Appendix E – Experiment 8 Consent Form

### *Participant Consent Form*

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Today you will be required to complete two tasks.

**Task One**

You will be presented with a visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. There are three phases within this task:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Reward Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will receive a reward for your eye movement.

3. **No Reward Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no reward is available in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment.

**Task Two**

In the second task you will be presented with a target circle either on the left or right side of the screen and are required to make an accurate eye movement towards this stimulus.

On some trials there will be another stimulus presented in the opposite side of the visual field. You are required to make an eye movement to the target circle.

The experimenter will tell you what task you will be experiencing throughout the experiment. Measurements will be taken using an eye tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time
* without having to give a reason for withdrawing
* without any adverse result of any kind? YES / NO

**Signed** ..... **Date.....**

**Appendix F – Experiments 9 & 10 Consent Form**

***Participant Consent Form***

I am conducting an experiment looking at human visual reactions when presented with a visual stimulus. Today you will be required to complete two tasks.

**Task One**

You will be presented with a visual stimulus in either the left or right side of the screen and you are required to make an eye movement towards it. There are three phases within this task:-

1. **Baseline Phase**

You will be required to make eye movements towards these visual stimuli which will change colour after an accurate eye movement towards them.

2. **Reward Phase**

You will see the same as you have in the baseline condition. However, on some of these trials you will receive a reward for your eye movement.

3. **No Reward Phase**

Again, you will be required to make eye movements towards visual stimuli which will change colour after an accurate eye movement towards them. However, no reward is available in this phase.

Once the target has changed colour the trial is over and you can press the response button to continue the experiment.

**Task Two**

In the second task you will be presented with two possible target locations (squares): one on the left and one on the right side of the screen. A smaller target square will appear in one of these squares and you will be required to make a speedy and accurate eye

movement to the location of this target. On some trials you will see a flash at one of these target locations. On some trials there will be no flash.

Measurements will be taken using an eye tracker. If you agree to participate you can change your mind at any time. If you would like your results to be omitted from the study after participating, then this is also possible.

Thank you for participating!

(NAME IN BLOCK LETTERS) .....

Have you read the Participant Information Sheet overleaf? YES / NO

Have you had an opportunity to ask questions and to discuss the study? YES / NO

Are you colour blind? YES / NO

Have you received satisfactory answers to all of your questions? YES / NO

Have you received enough information about the study and the Intended uses of, and access arrangements to, any data which you supply? YES / NO

Were you given enough time to consider whether you want to participate? YES/NO

Do you consent to participate in the study? YES/NO

Do you understand that you are free to withdraw from the study:

* at any time and
* without having to give a reason for withdrawing and
* without any adverse result of any kind? YES / NO

**Signed……. Date…….**

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