

The Effects of Behavioral Context on Motor Performance and Neural Activity

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University of Pittsburgh, 2019

Context greatly influences our behavior. How you react to a ball flying toward you is completely different if you are playing baseball or dodgeball. What neural mechanisms endow our behavior with context sensitivity? It is well known that contextual signals influence neural activity in many different brain areas, but we do not know how the brain can distinguish context information from the signals needed for successful motor behavior. In this thesis, I show two examples of context-dependent processing in monkeys: the first is behavioral, the second is neurophysiological.

First, I present an example of behavioral context that is known to drastically change performance: “choking under pressure”. Everybody – from competitors in a spelling bee to NFL kickers – knows how “nerves” can affect performance. Are animals also susceptible to this behavioral quirk? I trained two monkeys to perform a difficult reaching task while cueing the amount of reward given for successful task completion. As potential rewards increased, the animals’ performance improved. But, when the reward was exceptionally high, their performance paradoxically declined. Simply put, the monkeys choked under pressure. I observed this effect across multiple sessions and in two separate tasks. This work establishes an animal model of choking under pressure, allowing us to further explore the neural mechanisms of this phenomenon.

In a second study, I examined whether task instructions affected neural activity in the dorsal premotor cortex (PMd). Two monkeys performed a delayed reaching task with no

constraints on their gaze behavior. I recorded neural activity and the animals' eye position, and I found almost no relationship between eye position and neural activity. This is in stark contrast to previous studies that reported eye position signals in PMd. Those previous studies, however, instructed the monkeys to fixate their gaze while reaching. Our results demonstrate that task requirements influence the tuning properties of neurons involved in motor control, and it shows that context affects neural processing, even in areas that control movements.

Taken together, these studies show that context influences behavioral performance in monkeys, as it does in humans, and they begin to reveal the neural underpinnings of context-dependent sensory-motor processing.

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Preface

In one of the first conversations I had with my advisor, Dr. Aaron Batista, we talked about how athletes' brains seem to process information so much better than non-athlete brains. This is something that has always interested me, and being able to discuss such exciting topics with so much enthusiasm made me want to join Aaron's lab. While we may not have gained a full understanding of why athlete brains function so much better, I have had the opportunity to develop and work on two projects that answered small parts of that big question. Thank you, Aaron, for all that you have done to help me answer the questions that interest and excite me. You gave me the freedom to dive into projects that may not be the main focus of the lab's scientific goals, and you supported me throughout my journey. Your enthusiasm for your work, and science in general, pushed me to answer my own questions with that same level of passion and excitement. Your mentorship and guidance helped me develop into a scientist, communicator, and educator, and I cannot thank you enough for this.

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1.0 General Introduction

When we interact with our surroundings, we generally do so with a goal in mind – pick up our mug to take a drink, reach for our phone to answer a call, shake a friend’s hand to say hello. Choosing the actions taken to achieve these goals depends not only on our intended outcome but also on the situation in which it is encountered. The context in which we perform greatly influences both our behavior and the neural activity that drives our behavior. Our brains need to assess the context of our actions and then transform that information into motor actions. The work described here will explore how the context of a task influences behavior and neural activity in nonhuman primates.

In the following sections, I provide an overview of how task context has been studied in the brain (Section 1.1). I then focus on how the brain converts sensory information into motor actions (Section 1.2) and how a specific type of sensory information, rewards, can modulate neural activity (Section 1.3). Finally, I discuss an extreme case in which task context and reward size cause a disruption of normal performance: “choking under pressure” (Section 1.4).

1.1 Task Context and Neural Activity

Let us begin with an example of how task context can influence behavior. Assume you are walking down the street. Suddenly, out of nowhere, a baseball comes flying towards your head. In this situation, most people would cover their head and duck. Now, assume you are at a baseball game and walking through the stands. Again, a baseball comes hurtling towards you.

Most people in this situation will not duck for cover but rather try to catch the ball coming towards them. So, why does the same sensory stimulus cause two completely different behavioral responses? The answer lies in the context of the situation. In the baseball game scenario, we can expect to have a baseball coming towards us; in the other scenario, not so much. Based on this general example, we will now explore what contextual factors affect our behavior and how neural activity changes to drive these distinct behaviors.

Context can refer to both the external environment and our internal states. External context includes factors such as physical environment, the presence of food and water, the presence of others, or the rules of a task (Palmer & Kristan, 2011). On the other hand, internal context includes factors such as hunger, thirst, and emotional state (Palmer & Kristan, 2011). Any of these contextual factors can affect how we make a decision or perform an action, and oftentimes they will combine together to influence behavior. In this section, I will mainly focus on external context.

Studies with songbirds show how social context can influence their singing behavior. Male zebra finches sing either “directed” (oriented toward a female bird) or “undirected” (not oriented toward a female bird) songs that are repetitive, stereotyped sequences of syllables (Zann, 1996). Directed songs are more stereotyped than undirected songs. When the songs are undirected, neural activity in Area X of the striatum is higher and more variable than when the songs are directed (Hessler & Doupe, 1999; Jarvis, Scharff, Grossman, Ramos, & Nottebohm, 1998). This activity in Area X is modulated by dopaminergic neurons from the midbrain (J. W. Lewis, Ryan, Arnold, & Butcher, 1981; Reiner et al., 2004). During directed singing, dopamine levels in Area X increase relative to baseline, whereas undirected singing did not result in a significant increase in dopamine levels from baseline (Sasaki, Sotnikova, Gainetdinov, & Jarvis,

2006; Stepanek & Doupe, 2010). These studies show how the social context, such as the presence of others, can alter behavior and neural activity.

Studies in nonhuman primates have examined how neural activity changes based on task context. A study by Cohen and Newsome (M. R. Cohen & Newsome, 2008) looked at the noise correlation between pairs of middle temporal area (MT) neurons while monkeys viewed identical visual stimuli under two different contexts. The behavioral context changed based on the axis of motion the monkeys had to discriminate. One context promoted cooperative interactions between the neurons while the other promoted competitive interactions, and these interactions were based on neurons' preferred direction of motion. They found that neurons with relatively similar preferred directions had an increased correlation during the cooperative interactions and lower correlation during the competitive interactions. On the other hand, neurons with very different preferred directions had an increased correlation during the competitive interactions and lower correlation during the cooperative interactions. These results suggest that the changes in the correlations among neurons are due to the animal's understanding of the context in which it is behaving, and ultimately arise from task-dependent changes in the inputs to MT. Additionally, these results show that task context affects neural activity early within the sensorimotor processing stream. Thus, it only stands to reason we will also see contextual effects further along, in brain areas closer to the behavioral output or more associated with cognitive processing.

One of the main brain areas associated with representing task context and task rules is the prefrontal cortex (J. D. Cohen & Servanschreiber, 1992; Grafman, 1994; Miller, 1999; Passingham, 1993; Steven P Wise, Murray, & Gerfen, 1996). Asaad et al. (Asaad, Rainer, & Miller, 2000) performed a study where they recorded neural activity from the prefrontal cortex of

monkeys while they performed three separate tasks. The three tasks were a spatial task (memory-guided delayed saccade), an object task (delayed match-to-sample), and an associative task (associate cue with a specific response). The object and associative tasks used the same stimuli to cue different behaviors. The spatial and associative tasks used different stimuli to cue the same behavioral responses. They found that baseline firing rates, firing rate profiles, and neural activity related to particular stimuli and behavioral responses changed based on which task the animals performed. The behavioral context in which the animals performed had a substantial effect on neural activity.

A study by Hoshi et al. (Hoshi, Shima, & Tanji, 1998) examined how behavioral factors affected movement-related activity in dorsolateral prefrontal cortex and primary motor cortex while monkeys performed two different delayed motor tasks. The two tasks used in the study were a shape-matching task and a location-matching task. In the shape-matching task, the animals had to reach to a target matching the shape of the given cue. The location-matching task required the animals to reach to a target in the same location as the given cue. They found that a majority of neurons in prefrontal cortex fell into two classes: “target-shape selective” and “type of task selective”. The target-shape selective neurons showed changes in firing rate for a specific target shape. The task-selective neurons changed their activity based on which task the animals performed. Some neurons showed a combination of the two classes. These results demonstrate that prefrontal cortex encodes a broad range of information related to a reaching goal, and task context plays a large role in affecting this neural activity. Additionally, the authors performed the same analyses on neurons from primary motor cortex, and the movement-related information present in prefrontal cortex does not seem to carry over to primary motor cortex. At some point between prefrontal cortex and primary motor cortex, the task-relevant information gets

transformed into signals encoding motor output. This leaves us with the questions of how this transformation occurs and where the transformation takes place between prefrontal cortex and primary motor cortex.

1.2 Sensorimotor Transformations and Reference Frames

How does our brain enable us to make a reach? Arm movements are rather complicated, yet we generally make smooth, accurate reaches to objects. Does the cerebral cortex monitor and control our reaches on a moment-by-moment basis? Or does it only specify the desired endpoint of the hand, and allow downstream circuitry to handle the details of the movement? When planning and executing a reach, how does the brain transform the information from the sensory inputs (such as the visual location of the object) into a representation of motor output that can implement the reach? These questions have been studied extensively, and this section will discuss the main results from those studies.

When someone asks us where we live, we usually give them the address of our house. Our address is usually defined in relation to Earth. But, what stops us from using the sun or moon as our reference point? Each reference frame is valid, but one makes more sense than the others. That is, it is an encoding of a location that is stable, can be acted upon, and represents information at an appropriate granularity. A reference frame is a coordinate system centered on a rigid body used to describe the relative position or orientation of another object. The brain uses multiple reference frames to represent sensory information and to guide our behavior (Arbib, 1991; Colby & Duhamel, 1996; Gross & Graziano, 1995). These reference frames can be classified as egocentric or allocentric. Egocentric reference frames represent locations relative to

the observer. Examples of egocentric reference frames are eye-centered or hand-centered coordinate systems. Allocentric reference frames represent locations in coordinate systems outside of the observer. For example, room-centered or object-centered coordinates are allocentric reference frames. Although evidence indicates that the brain can use allocentric reference frames (Olson & Gettner, 1995), this section will discuss how the brain uses egocentric reference frames to guide our reaches, especially those that allow us to acquire an object.

What reference frames does our brain use to guide our reaches? Studies that ask this question are usually well-posed because the candidate coordinate frames are easily identified and can often be separated in the experiments (Kalaska & Crammond, 1992). However, interpreting the results can be rather difficult because most studies report a mixture of reference frames affecting individual neurons. We often gain the most insight when these studies report the relative proportions of cells in a brain area that use a certain coordinate frame. This suggests that many areas of the brain work together to perform reference frame transformations and that each area can contribute to multiple stages of this process.

Two of the most prevalent reference frames used in the brain are eye-centered and arm-centered coordinate systems. The posterior parietal cortex (PPC) is an area that many consider to be a visual-motor interface for action goals (Mountcastle, Lynch, Georgopoulos, Sakata, & Acuna, 1975). The PPC receives major inputs from the occipital lobe, and it projects to motor areas in the frontal lobe, such as premotor cortex and the frontal eye fields (Cavada & Goldmanrakis, 1989a, 1989b; Tanne, Boussaoud, BoyerZeller, & Rouiller, 1995). This puts PPC at the center of transforming visual information into spatial information for motor outputs, and human lesion studies have highlighted this importance (Bisiach, Perani, Vallar, & Berti, 1986; Faglioni & Basso, 1985; Rondot, Recondo, & Ribadeaudumas, 1977). Playing such an important

role in visual-motor processing suggests that it might use both gaze-centered and arm-centered reference frames.

The intraparietal sulcus splits PPC into the superior parietal lobule (SPL) and the inferior parietal lobule (IPL). The SPL has areas that show responses to gaze location and arm movements, specifically area 5, which projects directly to premotor cortex (Jones & Powell, 1970; Pandya & Kuypers, 1969; Petrides & Pandya, 1984). A specific region of area 5, the medial intraparietal cortex (MIP), shows involvement in the control of visually-guided arm movements (Caminiti, Ferraina, & Johnson, 1996; Cui & Andersen, 2007; Kalaska & Crammond, 1995; Pesaran, Nelson, & Andersen, 2006; Snyder, Batista, & Andersen, 2000). Furthermore, it represents target locations relative to the direction of the eyes (Batista, Buneo, Snyder, & Andersen, 1999; Buneo, Jarvis, Batista, & Andersen, 2002; Pandya & Seltzer, 1982), suggesting it uses eye-centered reference frames.

The SPL contains areas that project directly to premotor cortex (Jones & Powell, 1970; Pandya & Kuypers, 1969; Petrides & Pandya, 1984). The dorsal premotor cortex (PMd) plans and executes visually-guided reaches (He, Dum, & Strick, 1993; Weinrich & Wise, 1982). When planning a reach, PMd activity is robust and reflects the direction and amplitude of movement (Caminiti, Johnson, Galli, Ferraina, & Burnod, 1991; Crammond & Kalaska, 1994, 1996; Fu, Flament, Coltz, & Ebner, 1995; Fu, Suarez, & Ebner, 1993; Riehle & Requin, 1989). It contains strong outputs to primary motor cortex (M1) and to the spinal cord (He et al., 1993). These areas require arm-centered coordinate systems. But, PMd also receives inputs from area MIP, which uses eye-centered coordinates (Batista et al., 1999). This convergence of visual input from PPC and motor output to M1 makes PMd an excellent area to study sensorimotor transformations.

Knowing the inputs and outputs of PMd, it begs the question: how much does visual information influence its activity? Many studies show PMd activity is mainly influenced by hand position and movement direction using an arm-centered reference frame (Caminiti et al., 1991; Crammond & Kalaska, 1996). However, later studies have shown modulation of PMd activity due to the visual location of targets (Batista et al., 2007; Boussaoud, Jouffrais, & Bremmer, 1998; Cisek & Kalaska, 2002; Pesaran et al., 2006). These studies have shown discrepant results, though, with respect to the importance of eye-centered reference frames on PMd activity.

One of the first studies to show eye position modulation in PMd was done by Boussaoud and colleagues (Boussaoud et al., 1998). In this study, monkeys were trained to perform a reaching task while holding gaze fixation. As in previous studies, they found that PMd activity modulated with reach direction, but they also found that the activity varied with angle of gaze in the orbit. This implies that an eye-centered representation of reach goals might be at play. Based on this result, it would appear that the transformation from visual information to motor action is not complete by the time it reaches PMd.

Cisek and Kalaska (Cisek & Kalaska, 2002) also investigated the strength of eye-centered signals of target location in PMd. Their animals performed a delayed reaching task, but they did not require the animals to fixate their eye position. Using similar analysis techniques to Boussaoud's study, they found only a modest effect of gaze position on PMd activity. The majority of neurons were influenced by hand position, just as the classical PMd studies had found (Caminiti et al., 1991; Weinrich & Wise, 1982). Thus, Cisek and Kalaska concluded that modulation related to eye position was present in PMd, but it was rather weak compared to the modulation related to hand position.

These two studies together left some ambiguity about the tuning properties in PMd. One possibility is that PMd tuning is complex and not amenable to description by any single reference frame. Other cortical areas have been shown to have tuning in multiple reference frames (Buneo et al., 2002; Duhamel, Bremmer, BenHamed, & Graf, 1997; Stricanne, Andersen, & Mazzoni, 1996). Pesaran et al. (Pesaran et al., 2006) and Batista et al. (Batista et al., 2007) studied PMd using task that specifically dissociated reference frames. Another key feature of these two studies is that animals were instructed to direct their gaze at specific locations in the workspace. Both studies found neurons that were modulated by hand position and eye position. Pesaran found that a majority of neurons used a combination of at least two reference frames, and these combinations were inseparable. They concluded that PMd used a combination of hand, eye, and target positions to encode reach plans. Batista found that the predominant reference frame in PMd was hand-centered, but a small percentage of neurons were more eye-centered than hand-centered. They reported that there were idiosyncratic mixtures of reference frames in individual neurons, and that no area-wide assignation of an eye + hand reference frame was possible for PMd. The studies led by Pesaran and Batista are compatible in that it appears PMd does indeed partially encode targets in eye-centered coordinates.

Taken together, these four studies on the reference frames used in PMd leave us wondering exactly how much gaze modulation is present in PMd. A unifying interpretation of these seemingly disparate results is that task requirements may play a role in whether or not eye-centered reference frames are present. Specifically, in the one study that did not require animals to fixate (Cisek & Kalaska, 2002), eye-centered reference frames were only modestly present. In the three studies for which monkeys were required to fixate, eye-centered modulations of neural activity were more prominently evident. Could it be that the behavioral context in which the

animals perform the reaching task determines the neural activity present in PMd? My work will attempt to reconcile these differences.

1.3 Reward Signals in the Cerebral Cortex

Rewards, and the avoidance of aversive stimuli, are perhaps the greatest drivers of our behavior. Like all animals, much of what we do revolves around harvesting rewards, whether it is money at our job, good grades in school, or ice cream after exercising. Apart from bad habits, our actions can be defined as goal-directed. Thus, if we want to understand how context influences behavior, it is crucial to understand how rewards are represented in the brain and how these representations affect our behavior.

A reward is some stimulus, such as food, that creates positive emotions and pleasurable feelings (Schultz, 2010). Rewards often elicit approach behaviors, and they aid in the learning of such behaviors through positive reinforcement. When an object or something in our environment signals a reward, it becomes a goal that influences our behavior. Those objects gain a motivational value, which then shapes our behavior and decision making. All of this is done through a reward circuit in the brain (Schultz, 1998).

Seminal work by Olds and Milner (Olds & Milner, 1954) showed that electrical stimulation of certain brain areas in rats evoked pleasure and positive reinforcement. This finding led to the idea that there is a reward circuit in the brain that leads to these outcomes. Recent work has supported this idea, and we now know that the dopamine neurons of the nucleus accumbens (NAcc), ventral tegmental area (VTA), pars compacta of the substantia nigra (SNc), and ventral striatum (VS) play a central role in the reward circuit (O. Hikosaka, Bromberg-Martin, Hong, &

Matsumoto, 2008; Kelley & Berridge, 2002; Rolls, 2000; Schultz, 2000; Schultz, Tremblay, & Hollerman, 2000; Stefani & Moghaddam, 2006; R. A. Wise, 2002). These dopamine neurons exhibit phasic activity after receiving rewards and after the presentation of stimuli that predict reward (Ljungberg, Apicella, & Schultz, 1992; Romo & Schultz, 1990; Schultz, 1986; Schultz & Romo, 1990). These same neurons also show activity after the presentation of novel stimuli that have attentional and rewarding properties (Horvitz, 2000; Strecker & Jacobs, 1985); however, few show a phasic response after the presentation of aversive stimuli (Guarraci & Kapp, 1999; Matsumoto & Hikosaka, 2009; Mirenowicz & Schultz, 1996). Thus, it appears that the dopamine neurons of these different brain areas play a role in reward signaling.

Close examination shows that the phasic responses of dopamine neurons may not encode the reward itself, but rather a reward prediction error (RPE). The RPE hypothesis states that dopamine neuron activity increases for rewards that were not expected and decreases when an anticipated reward is omitted (Schultz, 1998, 2007a, 2007b). In other words, dopamine neuron activity changes when there is a discrepancy between the reward that was predicted for a given behavior and the reward that was received. This activity scales with the magnitude of the reward. The RPE response has been shown to occur during learning (Hollerman & Schultz, 1998). During learning, the reward prediction error signal can help modify synapses and neural circuitry until predictions match behavioral outcomes. This process has been proposed as one of the major facilitators of optimizing action selection to increase the successful procurement of rewards (Schultz, Dayan, & Montague, 1997). The RPE hypothesis has gained strong traction in the field, and numerous studies support it, even human imaging studies (Chowdhury et al., 2013; D'Ardenne, McClure, Nystrom, & Cohen, 2008).

While the dopamine neurons initially process the reward and calculate a reward prediction error, other neurons and brain areas show an expectation of reward. These brain areas exhibit sustained activity changes after the presentation of a well-learned stimulus that predicts reward. Studies have shown that striatum (Apicella, Scarnati, Ljungberg, & Schultz, 1992; O. Hikosaka, Sakamoto, & Usui, 1989; Schultz, Apicella, Scarnati, & Ljungberg, 1992; Shidara, Aigner, & Richmond, 1998), orbitofrontal cortex (K. Hikosaka & Watanabe, 2000; Schoenbaum, Chiba, & Gallagher, 1998; Tremblay & Schultz, 1999, 2000), and amygdala (Schoenbaum et al., 1998) all contain neurons with reward-expectation signals. In striatum and orbitofrontal cortex, these neurons discriminate between rewarded and non-rewarded trials (Hollerman, Tremblay, & Schultz, 1998; Tremblay & Schultz, 2000). Additionally, delaying reward delivery prolongs the duration of the sustained activity while delivering the reward early reduces it (Apicella et al., 1992; Schultz et al., 1992; Tremblay & Schultz, 2000).

These reward-related changes in neural activity have led to researchers studying their effects on behavior (Dickinson & Balleine, 1994). Oftentimes, higher rewards result in more motivated behaviors, such as shorter reaction times and faster movements (Hassani, Cromwell, & Schultz, 2001; Kobayashi, Lauwereyns, Koizumi, Sakagami, & Hikosaka, 2002; Leon & Shadlen, 1999). These behavioral changes have led to the idea that neural responses to larger rewards will change in such a way as to produce the seen behaviors. This idea would require the integration of reward expectation information with movement planning information leading to acquisition of the reward. Studies have shown that striatum, supplementary motor area, dorsolateral premotor cortex, parietal cortex, and dorsolateral prefrontal cortex may play a role in planning and executing movements to rewards (Kurata & Wise, 1988; Leon & Shadlen, 1999; Okano & Tanji, 1987; Romo & Schultz, 1987; Schultz & Romo, 1988; Sugrue, Corrado, &

Newsome, 2004; Watanabe, 1996). These areas exhibit enhanced activity before movement initiation towards expected rewards, and in some neurons, the activity will continue until the reward is obtained.

Despite decades of research into the influences of rewards on neural activity, a large debate exists as to whether cortical reward signals represent the value of the reward or the change in motivation during motor planning and performance. To parse this question, a study by Roesch and Olson (Roesch & Olson, 2003) looked at single neuron activity in multiple cortical areas in macaque monkeys performing a saccade task for small and large rewards. They found that the large reward elicited more motivated behavior as evidenced by lower error rates, faster reaction times, and fewer fixation breaks. For each brain area studied, large reward trials caused increased firing rates and stronger directional tuning signals. These reward-related signals became stronger in the more posterior areas along the anterior-posterior axis of cortex. They found weak reward signals in prefrontal cortex, moderate reward signals in frontal eye field, and strong reward signals in premotor cortex. These results show that reward-related activity is widespread in frontal cortex. Furthermore, the reward-related signals correlate with both reward value and the animals' motivation due to the reward value. Thus, based on this study, neuronal activity that represents reward value cannot easily be distinguished from neuronal activity representing motivation or motor readiness. To best understand what the reward-related signals in cortex represent, nonstandard task designs need to be used in order to decouple the value of the reward from the intensity of motor preparation.

In an effort to dissociate between neural activity related to reward value and that related to motivation, Roesch and Olson (Roesch & Olson, 2004, 2007) ran new experiments that had monkeys perform a task in which motivation was manipulated independently of reward value. To

do this, they used both potential rewards and punishments that would be received on each trial. In this task, neurons sensitive to motivation will show increased firing rates for both large rewards and large punishments. On the other hand, neurons sensitive to reward value will only increase firing for large rewards or change their firing rate for large punishments in an opposite manner to the way they changed for the large reward. In this study, they recorded from single neurons in two cortical areas: orbitofrontal cortex and premotor cortex. They found that neurons in orbitofrontal cortex responded specifically to reward value. Conversely, the neurons in premotor cortex responded to the motivation of the animal. A separate study in rats performing a similar task that dissociated reward value and motivation showed that separate populations of neurons in ventral striatum encode value and motivation (Bissonette et al., 2013). Thus, it appears that separate populations of neurons encode different aspects of a reward, sometimes in separate brain areas and sometimes within the same area.

All of this work has provided great insight into how rewards affect our neural activity and behavior. However, they still leave us with several unanswered questions. For example, how do value and motivation signals interact? Where does the transformation from reward value to motivation occur in the brain? The findings of these studies highlight that motor preparation, arousal, and attention can be affected by motivational modulation, yet how does the brain integrate reward and motivation information with motor preparatory activity? Are the interactions between reward signals and motor activity always a boon, or can there be “too much of a good thing,” in that reward signals interfere with motor control signals? This dissertation will help to explain some of these questions in Chapters 3 and 4 by contributing to our understanding of how rewards impact the control of voluntary behavior.

1.4 Choking Under Pressure

Many people associate increasing rewards and incentives with increased performance. This is why some jobs provide extra monetary incentives to motivate employees to perform well (Lazear, 2000). While performance generally does increase with increasing rewards, in certain situations, when potential reward for successfully completing a task becomes extremely high, performance paradoxically declines. This phenomenon, known as “choking under pressure”, is best described as a decrease in performance in high-stakes situations that demand good or increased performance (R. F. Baumeister, 1984). Choking under pressure is an extreme example of how context affects performance. The task and its requirements for success remain the same, but the environment in which the task is performed changes, whether it is due to increased reward or some other motivating factor.

Choking under pressure has been documented consistently in the laboratory, and psychological explanations for it are well developed and thoroughly researched. However, the neural mechanisms of this paradoxical performance decrement are not well understood. This section will explore the current understanding of choking under pressure in humans. It will discuss the psychological theories of why humans choke under pressure, the behavioral changes associated with the phenomenon, and our current understanding of the neural underpinnings of choking under pressure.

1.4.1 Definitions used in choking under pressure research

Before diving into the literature, it is important to understand a couple of the key terms that occur throughout this research. *Pressure* is defined as the presence of situational factors that

increase the importance of optimal performance (R. F. Baumeister, 1984). Examples of pressure include rewards or punishments based on performance, the presence of an audience, and a competitive environment. *Choking under pressure* is defined as sub-optimal performance when pressure is high (R. F. Baumeister, 1984). This definition has many subtle nuances. First, sub-optimal performance refers to a decrease in performance as compared to a performer's current level of performance. Studies of choking under pressure often record a baseline performance level from each subject in a low-pressure situation. Then, they can compare each subject's performance in a high-pressure situation to the baseline performance level from the low-pressure situation. Second, we often associate choking under pressure with a single failed action, such as a missed field goal in a game of football. A given performance can only be described as choking under pressure if we know that the performer could have done better (Roy F Baumeister & Showers, 1986). In most studies, there will be multiple trials attempted in both low-pressure and high-pressure situations, and the average performance in each condition will describe whether the subjects choked under pressure or not. Finally, choking under pressure demands that some form of increased pressure causes the decreased performance. An athlete in an extended period of poor performance, known as a slump, does not exhibit choking under pressure unless this decline in performance is caused by an extended period of high pressure (Grove, 2017).

In addition to these definitions, it is important to understand how choking under pressure is studied. We are familiar with choking under pressure in real-world situations, such as missing a game-winning field goal in football or a tournament-winning putt in golf. Research in the laboratory, however, provides a tightly controlled environment with which to study this phenomenon. Researchers can easily manipulate the amount and type of pressure induced, and they can also measure the subjects' perceptions of pressure (Beilock & Gray, 2012). This allows

for an easy and direct correlation between perceived pressure and performance, creating clear results that can be extrapolated to real-world situations.

1.4.2 Psychological theories of choking under pressure

The current literature has several different psychological theories about why humans choke under pressure. The most prominent ones are attentional theories (explicit monitoring; distraction) and drive theories (over-arousal).

1.4.2.1 Attentional theories of choking under pressure

Attentional theories predict that high-pressure situations cause a performer to shift their attention away from task-relevant cues to task-irrelevant cues. These theories suggest that as pressure increases, a performer will either shift their attention to irrelevant internal cues (explicit monitoring) or irrelevant external cues (distraction) rather than maintaining optimal attention to the task.

Distraction theories propose that pressure creates a distracting environment that causes the performer to shift their attention away from task execution and instead attend to task-irrelevant cues such as worry about the task (Ashcraft & Kirk, 2001; Beilock, 2008; Beilock, Kulp, Holt, & Carr, 2004; Carver & Scheier, 1981; Eysenck, 1979; Sarason, 1972; Wine, 1971). These task-irrelevant thoughts and worries compete for working memory resources that would ideally be used to successfully perform the task. Much support for the distraction theory has come from research involving the effects of test anxiety on test-taking performance (Ashcraft & Kirk, 2001; Eysenck, 1979; Sarason, 1972; Wine, 1971). Wine (Wine, 1971) and Sarason (Sarason, 1972) both show that people with high anxiety focus on task-irrelevant thoughts, and

ultimately cannot use their full cognitive processing that would allow them to perform well on the test. Additionally, Beilock and colleagues (Beilock, Kulp, et al., 2004) looked at the effects of pressure on cognitive task performance. In this study, they had subjects perform easy and hard math problems in both low and high-pressure situations. They found that the subjects did perform worse in the high-pressure situation, but only on the difficult problems that required more working memory to correctly solve. Thus, these studies show that pressure can consume working memory resources and lead to choking under pressure, especially on cognitive tasks that are highly reliant on the working memory system.

A second branch of attentional theories is explicit monitoring or self-focus theories. These theories propose that increased pressure causes the performer to become self-conscious and focus their attention on the specific processes required to complete the task (R. F. Baumeister, 1984; Beilock & Carr, 2001; G. A. Kimble & Perlmutter, 1970; Langer & Imber, 1979; B. P. Lewis & Linder, 1997; Masters, 1992). Well-learned or proceduralized tasks that normally function outside of conscious control are most susceptible to explicit monitoring of each step. Unlike the distraction theories, where decreased attention causes poor performance, explicit monitoring theories posit increased attention to task execution, which leads to choking under pressure. Intuitively, one can imagine thinking about how you walk while you are walking. It is likely to make you trip. One example of explicit monitoring is Masters' reinvestment theory (Masters, 1992). This theory proposes that increased pressure causes a well-learned task to become "dechunked", or broken down into a sequence of smaller units that are performed in series. When this happens, errors can occur between each unit, leading to poor performance. Additionally, since each unit is processed separately, performance becomes slower.

More recent studies have tried to understand these results without inducing pressure but instead manipulating the attentional requirements of the tasks that subjects perform (Beilock, Carr, MacMahon, & Starkes, 2002; Gray, 2004). Beilock and colleagues (Beilock et al., 2002) had experienced soccer players dribble a ball through a series of pylons while performing a secondary task. One secondary task involved monitoring an auditory tone, which mimicked distraction theories. The other secondary task required the players to monitor which side of their foot most recently touched the ball, which represented explicit monitoring theories. The auditory task did not affect the players' ability to perform the task, but the monitoring task led to slower and decreased performance. Gray (Gray, 2004) found similar results in a study of baseball batting. In this study, expert baseball players performed a hitting task while attending to an external auditory tone. This secondary task had little effect on their hitting performance. However, when the players were required to focus on the position of their bat during the swing, their performance declined. Taken together, these studies show that increased attention on task performance, without any added pressure, causes decreased performance. These findings fall in line with explicit monitoring theories of choking.

Based on the studies discussed here, both distraction theories and explicit monitoring theories can account for choking under pressure in certain situations. Distraction theories better explain cognitive tasks such as math problem solving and test-taking. Explicit monitoring theories better explain complex sensorimotor tasks such as golf putting, soccer dribbling, and baseball bat swinging.

1.4.2.2 Drive theories of choking under pressure

Drive theories, also known as over-arousal theories, link an individual's performance to their levels of arousal or "drive". These theories assume that an increased motivation to perform

well leads to increased arousal. The main theory associated with over-arousal is the Yerkes-Dodson effect (Yerkes & Dodson, 1908). This effect shows that as arousal increases, so does performance, but only to a certain point. Many researchers refer to this as the inverted-U theory, and it shows that performance is optimal at intermediate levels of arousal. If arousal is too low or too high, a performer will not succeed at the task. Easterbrook (Easterbrook, 1959) proposed a variant of the inverted-U, called the cue utilization hypothesis. This hypothesis argues that levels of arousal affect the range of cues used in a task. If arousal is too low, attention may be too broad to successfully complete a task. On the other hand, if arousal is too high, attention may be too narrow and will result in decreased performance. In essence, this hypothesis links arousal and performance by shifting an individual's attention. Another variation of the inverted-U hypothesis is the cusp catastrophe model (Hardy, 1996). This theory differs from the inverted-U in that it predicts a sudden, catastrophic decrease in performance once arousal levels move beyond optimal levels. Inverted-U theories, on the other hand, predict gradual decreases in performance beyond optimal arousal levels. Furthermore, the catastrophe model predicts that once this catastrophic drop in performance occurs, it is difficult to recover back to previous levels of performance. While this model fits well with the expected effects of choking under pressure, more work is needed to better understand how this theory and its predictions relate to real-world scenarios.

A second class of drive theories involves social facilitation and dominant-response theories. Social facilitation shows a relationship between arousal and performance, and it often assumes that the increased arousal levels are due to the presence of an audience (Butler & Baumeister, 1998; Zajonc, 1965), but a recent study showed that monetary rewards also increase arousal levels (Ariely, Gneezy, Loewenstein, & Mazar, 2009). It also assumes that heightened

levels of arousal will lead a performer to exhibit their dominant response more often. For novices, the dominant response is likely poor performance, whereas for experts the dominant response is likely good performance. In other words, if the task is simple or well-learned, the presence of an audience will lead to high performance; if the task is complex or not well-learned, an audience will cause poor performance. An extension of this is the fear of negative evaluation that many athletes experience (Mesagno, Harvey, & Janelle, 2012). Athletes who have a high fear of negative evaluation show a significant decrease in performance in high pressure situations, often created by the presence of an audience. While social facilitation makes sense intuitively, it does not match well with real-world examples. If the presence of an audience elicits a performer's dominant response, then, technically, professional athletes should never choke under pressure. Kimble and Rezabek (C. E. Kimble & Rezabek, 1992) attempted to address this concern by having subjects play the game *Tetris* in front of an audience. If social facilitation holds, then highly skilled players should perform well in front of the audience. They found, however, that the most skilled players performed worse in front of an audience as compared to a situation with no audience. Thus, the dominant-response theories do not always hold when the pressure to perform well is highest.

While drive theories have helped account for some types of performance failures, they have often been criticized for being too vague. They link arousal and performance, but they do not explain the mechanisms by which arousal affects performance. For this reason, the attentional theories of choking under pressure are often preferred to the drive theories.

1.4.3 Behavioral changes due to choking under pressure

The cognitive processes of choking under pressure have received much attention from the field and are fairly well understood, but the behavioral changes that lead to decreased performance are not as well understood. When individuals choke under pressure, this phenomenon is manifested in some type of behavioral change. It is important to understand both the cognitive processes and the biomechanical changes associated with choking under pressure in order to gain a full understanding of the phenomenon.

The main theory that describes these behavioral changes is known as “freezing degrees of freedom” (Bernstein, 1967). The human body allows for numerous possible solutions to successful performance of complex motor tasks because many different combinations of joint rotations could lead to successful completion of motor tasks. This is an advantage for motor behavior, but it creates a complex calculation that the brain must perform. Bernstein’s theory (Bernstein, 1967) suggested that novice performers coordinate their movements during a task by “freezing” some of these degrees of freedom in order to reduce task complexity. They freeze the degrees of freedom by either keeping some joints rigid or coupling the movements of multiple joints. As novices gain more skill, they begin to “unfreeze” the joints to allow for better, more fluid movements. This theory was given empirical support in a study by Vereijken and colleagues (Vereijken, Emmerik, Whiting, & Newell, 1992). They had five inexperienced subjects learn to make a slalom movement on a ski apparatus. While the subjects performed the task, the researchers measured multiple movement variables and joint angles. They found that in the beginning of the learning phase, the subjects rigidly fixed the joint angles of the hip, knee, and ankle and tightly coupled these joint movements to reduce the complexity of the task. However, as learning progressed over the course of seven days, the subjects showed more

variability in their joint angles and the joints became more independent during the task, showing that more experience led to the “unfreezing” of the degrees of freedom in the joints required for this task.

Bernstein’s theory also suggested that high-pressure situations may cause expert performers to revert back to the novice strategy of freezing degrees of freedom. Several studies have provided evidence for this idea (Collins, Jones, Fairweather, Doolan, & Priestley, 2001; Higuchi, Imanaka, & Hatayama, 2002; Pijpers, Oudejans, & Bakker, 2005; Pijpers, Oudejans, Holsheimer, & Bakker, 2003). Collins and colleagues (Collins et al., 2001) studied the movements of weight lifters during both training and competitive scenarios. When a lifter failed to complete a lift during a competition that was successfully completed in practice, the researchers found a higher correlation between the neck and hip joints. This finding is consistent with freezing degrees of freedom, and it occurred during the high-pressure situation of competition. Pijpers and colleagues (Pijpers et al., 2003) investigated the movements of rock climbers. They induced anxiety on the climbers by having them climb at different heights on a climbing wall. Subjects climbing higher on the wall exhibited more rigid and less fluid movements compared to when they were climbing at a lower level. These sub-optimal movements led to slower climbing times under high pressure.

Other studies have used electromyography (EMG) to study behavioral changes caused by increased pressure. Duffy (Duffy, 1932) found that increased pressure can lead to increased muscle tension. Weinberg and Hunt (Weinberg & Hunt, 1976) tested this idea in participants performing a throwing task. They recorded EMG data from the biceps and triceps of the subjects during the task, and they found that high-anxious subjects contracted both muscles for longer periods than low-anxious subjects when performing in a high-pressure situation. Another study

by van Loon and colleagues (van Loon, Masters, Ring, & McIntyre, 2001) also found increased limb stiffness under conditions of high stress. The researchers measured EMG activity from the biceps and triceps of the subjects while they performed a load perturbation task. Pressure was induced by performing difficult arithmetic during the load perturbation task. The researchers found that conditions of high arousal caused increased limb stiffness and higher, more continuous force exerted against the load. Taken together, the findings from these studies show that high pressure situations result in decreased neuromuscular efficiency. They also suggest that increased muscle tension under high levels of pressure may influence performance in a negative manner.

The studies focusing on behavioral changes due to high pressure provide differing results about the movement changes exhibited in these situations. Some studies show that high pressure leads to decreased variability in movement (Collins et al., 2001; Pijpers et al., 2003) while others show an increase in movement variability (Gray, 2004; Pijpers et al., 2005). Of course, movement variability does not have a straightforward relationship to task goals. Future research will need to identify the most detrimental behavioral changes associated with high pressure, and determine the root causes of those changes.

1.4.4 Neural mechanisms of choking under pressure

These psychological-level explanations for choking under pressure are not mutually exclusive. Some of the theories actually complement each other. While these behavioral studies have yielded insight and have some explanatory value, they have not shed any light onto the neural basis of this phenomenon. Each psychological theory can be extended to form predictions about what brain areas may be involved in choking under pressure. Despite these predictions

about the underlying neural activity, few studies have directly tested these predictions. In order to fully understand the phenomenon of choking under pressure, researchers need to understand the neural mechanisms by which choking under pressure occurs. It is possible to imagine that if we had a neural understanding of choking under pressure, we might be able to offer techniques to ameliorate it.

Dean Mobbs and colleagues (Mobbs et al., 2009) provided the first study of neural activity and choking under pressure. They had human subjects perform a task that induced high pressure situations with high rewards. The subjects underwent functional magnetic resonance imaging (fMRI) while performing the task. Their results showed that activity increased for both high and low rewards in the reward systems of the brain, such as the dorsolateral striatum and medial orbitofrontal cortex. This result shows that basic reward systems are engaged by the task, but it does not show specifically why choking under pressure occurs. To find this, they searched for brain areas that showed significantly different activity during high-reward trials than low-reward trials. They found that activity in the ventral midbrain was significantly higher on the high-reward trials. Furthermore, they found that task performance on high-reward trials decreased as ventral midbrain activity increased. On the other hand, they also found that increased activity in the medial prefrontal cortex (mPFC) led to better task performance on high-reward trials. Their results suggest that increased activity in midbrain structures hinders performance whereas increased activity in cortical structures improves performance. Based on their findings, they interpreted that over-motivation led to this increased midbrain activity, which fits well with the over-arousal theories of choking under pressure (Easterbrook, 1959; Hardy, 1996; Landers, 1980; Short & Sorrentino, 1986).

Vikram Chib and colleagues (Chib, De Martino, Shimojo, & O'Doherty, 2012) also looked at the neural mechanisms underlying choking under pressure. They expanded upon the Mobbs study by using six different monetary rewards ranging from \$0 to \$100. The subjects in the study performed a novel motor task for the different rewards while undergoing fMRI scanning. It is important to note that the subjects performed the task at an easy and a hard difficulty level, and the subjects only choked under pressure during the hard difficulty level. The researchers found that ventral striatal activity appeared to play a role in task performance. The ventral striatum was active during both the time of incentive presentation and the execution of the motor task. When the incentive was first presented, striatal activity increased with increasing reward. However, they found that once the subjects began execution of the task, striatal activity deactivated with respect to the magnitude of the monetary reward. Previous work has shown that ventral striatum increases activity for potential gains and decreases activity for potential losses (Tom, Fox, Trepel, & Poldrack, 2007). Taken together, these results suggest that the subjects are exhibiting loss aversion. Loss aversion is the tendency to prefer avoiding losses to acquiring equivalent gains (Kahneman & Tversky, 1979). In this study, when the monetary reward was initially presented, the subjects encoded it as a potential gain. When actually performing the task, however, they reframed the potential gain in terms of a loss. The larger potential gains led to larger potential losses, and this was ultimately manifested as performance decrements at the highest reward levels. To push this finding further, they looked at each subject's level of loss aversion and compared that to their striatal activity and task performance at the \$100 reward level. They found that more loss averse subjects had higher levels of deactivation of ventral striatum during task performance on the \$100 reward trials. Furthermore, higher striatal

deactivation led to worse performance. Their results point to loss aversion as the cause of performance decrements when the incentives are high.

As a follow-up to their study in 2012, Chib and colleagues performed another set of experiments where they framed the reward as a potential gain or a potential loss (Chib, Shimojo, & O'Doherty, 2014). The subjects performed the same task as in the previous study while undergoing fMRI scanning. Additionally, the subjects' level of loss aversion was measured outside the fMRI scanner using a prospect theory task. In this study, the researchers found that the framing of the reward had a profound effect on an individual's performance. Prospective gains elicited choking responses in subjects with high loss aversion whereas prospective losses elicited choking responses in subjects with low loss aversion. Their neural results showed that ventral striatum played a role in performance for both prospective gains and losses. They found that ventral striatum increased its activity in response to increasing gains and losses. This type of response reflects increased motivation or arousal (Cooper & Knutson, 2008; Knutson, Adams, Fong, & Hommer, 2001; Knutson, Fong, Adams, Varner, & Hommer, 2001). Additionally, just as in their previous study, they found an initial increase in activity when the cue was first presented followed by a decrease in activity at the time of task execution. This was true of both prospective gains and prospective losses. These results suggest that ventral striatum may influence behavioral performance regardless of how the task is framed; higher deactivation of ventral striatum during task execution leads to worse behavioral performance. Finally, they found that ventral striatum and premotor cortex decrease their functional connectivity during trials in which the subjects choke under pressure. This result sheds light onto how ventral striatum influences motor cortical activity when an individual chokes under pressure.

Finally, Lee and Grafton (Lee & Grafton, 2015) study the neural mechanisms of choking under pressure using a task similar to the classic arcade game *Snake* while subjects underwent fMRI. The subjects performed this task for rewards of \$5, \$10, or \$40. The subjects exhibited choking under pressure on the trials where \$40 was stake. The neural results showed evidence of dorsolateral prefrontal cortex involvement in choking under pressure. They investigated this finding by running a functional connectivity analysis with motor cortex. This analysis revealed that dorsolateral prefrontal cortex showed higher levels of functional connectivity with motor cortex during the pre-movement period on \$40 trials compared to \$10 trials. They also compared \$5 trials to \$10 trials in order to see if there was increased connectivity due to increased reward. They did not find this to be the case. Thus, their results suggest that even before movement onset on highly rewarding trials, prefrontal cortex influences motor cortical activity. To investigate this finding further, they analyzed how this increased connectivity influenced behavior on the \$40 trials. They found that the increased connectivity between prefrontal cortex and motor cortex did not directly predict task accuracy, but the subjects who showed the highest connectivity showed the least amount of choking under pressure. Based on this result, it seems that increased functional connectivity between prefrontal cortex and motor cortex helps prevent choking under pressure. This finding supports the distraction theory of choking under pressure because only individuals who did not increase their prefrontal activity showed choking under pressure.

Our current understanding of the neural mechanisms of choking under pressure is still in its early stages. The current studies have shown that many different brain regions are involved in choking under pressure. However, these studies have provided mixed results as to how neural activity relates to the psychological theories. Some studies support over-arousal theories (Chib et al., 2014; Mobbs et al., 2009), one supports loss aversion (Chib et al., 2012), and another

supports distraction theories (Lee & Grafton, 2015). It could be that many different theories hold true and no single brain area causes choking under pressure. Further work will be needed to fully understand the neural mechanisms of choking under pressure and dissociate between or expand upon the current psychological theories. Studying the brain during high pressure situations opens new avenues for understanding the mechanisms underlying choking under pressure.

1.5 Research objectives and outline

The unifying perspective of this dissertation is to assess how task context influences both neural activity and behavioral performance. In Chapter 3, I show that high rewards lead to choking under pressure in nonhuman primates, which is the first demonstration of choking under pressure in a nonhuman species. In Chapter 4, I discuss how different potential reward sizes influence motor cortical activity in a nonhuman primate that exhibits choking under pressure. In Chapter 5, I investigate how the requirements of a task influence neurons in the dorsal premotor cortex of nonhuman primates. Finally, in Chapter 6, I propose future directions that this research on task context and choking under pressure can take. To begin, Chapter 2 describes experimental methods common to Chapters 3-5.

2.0 General Methods

In this chapter, I will describe the general methods used in the experiments of Chapters 3-5. In section 2.1, I describe the collection of behavioral data recorded from the nonhuman primates during the experiments. Section 2.2 details the neural recording methods used in the experiments of Chapters 4 and 5. Chapters 3-5 provide more specific methodological details. All animal handling protocols and experiments were approved by the University of Pittsburgh Institutional Animal Care and Use Committee (IACUC), in accordance with the US Department of Agriculture (USDA), the International Association for the Assessment and Accreditation of Laboratory Animal Care, and the National Institutes of Health (NIH).

2.1 Behavioral Recordings

Four adult male Rhesus macaques (*Macaca mulatta*) were used in the experiments of this dissertation: Monkey F, Monkey I, Monkey L, and Monkey N. The animals sat in a custom-built primate chair during the experiments. During the experiments, the animals worked in a virtual reality environment. They sat facing a mirror, onto which visual stimuli were projected from a 144 Hz computer monitor. While working, the animals' head was restrained using three titanium head posts and a custom "halo" (Davis, Torab, House, & Greger, 2009). Additionally, one arm was comfortably restrained while the animals performed the task with their working arm. For all tasks, the animals used their working arm to control a cursor in the virtual reality environment. We used Velcro patches and a cloth glove to attach an LED marker to the animals' hand. The

LED marker was tracked at 120 Hz by multiple cameras from a motion tracking system (PhaseSpace Inc., San Leandro, CA). With this system, hand position corresponded directly to cursor position on the screen. Eye position was tracked using an Eyelink 1000 system (SR Research Ltd., Ontario, Canada). This system used infrared light to track the eyes at 1000 Hz throughout the experiments. We calibrated the eye tracker at the beginning of each session to ensure proper tracking. All tasks were run on custom software developed using LabVIEW, and all analyses were performed using MATLAB software.

2.2 Electrophysiology Recordings

After the animals were fully trained on the behavioral tasks, they were implanted with a 96-channel microelectrode array (Blackrock Microsystems, Salt Lake City, UT) in the dorsal aspect of the premotor cortex (PMd). Each array was implanted in the hemisphere contralateral to the working arm. Array placement was guided by cortical landmarks as shown in pre-operative MRI scans. Tucker Davis hardware and software were used to record signals from the arrays (Tucker Davis Technologies, Alacua, FL).

Neural data are used in Chapters 4 and 5. Monkeys I and L were studied in Chapter 5. Their arrays were implanted in the left hemisphere of PMd. Monkey F was studied in Chapters 3 and 4. His array was also implanted in the left hemisphere of PMd. Although Monkey F's array was implanted in what anatomically appeared to be PMd, the neural activity recorded from the array was more consistent with primary motor cortex (M1). Because of this, we focused more on movement-related neural activity than delay period activity. Monkey N did not receive an array implant and was instead used for behavioral analyses in Chapter 3, along with Monkey F.

3.0 Monkeys Show a Paradoxical Decrement in Performance in High-Stakes Scenarios

In this chapter, I describe my work studying choking under pressure in monkeys. Prior to this work, choking had only been demonstrated in humans. This study establishes an animal model of choking under pressure, which allows for a new method of studying the neural basis of the phenomenon. I have completed a manuscript of this paper, which was formatted for submission to the *Proceedings of the National Academy of Sciences*. The principal investigators (Chase and Batista) are refining it for submission.

3.1 Introduction

“Choking under pressure” is defined as performing worse than expected given your abilities (R. F. Baumeister, 1984). It occurs in high-pressure situations where there is a strong incentive to perform well. Choking under pressure is well-known to athletes and their fans (Connolly, 2019; Fryer, Tenenbaum, & Chow, 2018; Gucciardi, Longbottom, Jackson, & Dimmock, 2010) and has been demonstrated under a variety of controlled laboratory conditions, including challenging motor tasks (Beilock & Carr, 2001; Beilock et al., 2002; Jackson, Ashford, & Norsworthy, 2006), test taking (Beilock & Carr, 2005; Beilock, Kulp, et al., 2004), and performing in front of an audience (Mesagno et al., 2012; Wallace, Baumeister, & Vohs, 2005). Choking occurs in these studies when the task is difficult and the incentives for good performance are extremely high. The choking phenomenon is best represented by an “inverted-U” shape to the relationship between performance and incentive (Yerkes & Dodson, 1908). That

is, performance increases with increasing reward, but only up to a point; for unusually high stakes, performance paradoxically declines.

Psychological explanations for choking under pressure range from high-level cognitive and social theories to low-level arousal theories (Roy F Baumeister & Showers, 1986; Beilock & Gray, 2012; Mesagno & Beckmann, 2017; Yu, 2015). If choking under pressure results from high-level cognitive influences (Beilock, Bertenthal, McCoy, & Carr, 2004; B. P. Lewis & Linder, 1997; Masters, 1992) then perhaps humans and other cognitively sophisticated animals are uniquely prone to choking under pressure (Huguet, Barbet, Belletier, Monteil, & Fagot, 2014). On the other hand, it could be that choking under pressure arises from increased levels of arousal (Ariely et al., 2009; Easterbrook, 1959; Hardy, 1996; Zajonc, 1965), something that many species of animals experience. These possibilities are not mutually exclusive. Knowing whether animals also choke under pressure would help constrain the psychological theories.

While human behavioral studies can help strengthen the psychological theories of choking under pressure, they do not provide any insight into the neural mechanisms of why we decrease performance under high levels of pressure. Recently, though, investigators have begun to examine the neural basis of choking under pressure using functional magnetic resonance imaging (fMRI) while subjects perform challenging motor tasks for various rewards. These studies have revealed several brain regions that may play a role in choking under pressure. Some studies have shown that changes in activity in reward processing systems, such as the ventral midbrain (Mobbs et al., 2009) and ventral striatum (Chib et al., 2012), may play a role in choking under pressure. Another study (Lee & Grafton, 2015) found that diminished activity in prefrontal cortex control regions may lead to decreased performance in high-pressure situations. Together, these studies suggest that the neural processes that lead to choking under pressure involve the

interaction of circuits in disparate parts of the brain and the coordination of the activity of large populations of neurons. An animal model that exhibits choking under pressure would allow us to further explore the neural underpinnings of this paradoxical phenomenon.

Here we report that Rhesus monkeys choke under pressure. We demonstrate that this behavior is reliable (that is, it occurs over many sessions) and robust (it occurs in different behavioral paradigms). Behaviorally, the animals fail under high stakes conditions because their movements are hypometric – they move too slowly. Taken together, these results mean that monkeys can provide an animal model with which to study the neural basis of choking under pressure.

3.2 Methods and Data Analysis

3.2.1 Monkeys and equipment

Two adult male Rhesus monkeys (*Macaca mulatta*, Monkeys F and N) were used in this study. All animal procedures were approved by the University of Pittsburgh Institutional Animal Care and Use Committee, in accordance with the guidelines of the US Department of Agriculture, the International Association for the Assessment and Accreditation of Laboratory Animal Care, and the National Institutes of Health.

During experiments, the monkey sat in a primate chair with his head braced facing a mirror ~8 cm in front of his eyes to view a computer monitor displaying task events. The LCD monitor had a 144 Hz refresh rate. A photodiode was used to measure the timing of the visual display, which was used in analyses to compensate for the lag of the monitor. The monkey

performed the task by making hand movements (right arm for Monkey N, left arm for Monkey F) in the open space in front of him. The reaching hand and arm were unrestrained. The hand was not visible to the monkey during experiments, as it moved in the space behind and below the mirror. Hand position was tracked via an LED marker attached to the monkey's index finger (120 Hz sampling rate, nominal resolution <1 mm; PhaseSpace, San Leandro, CA). The monkey's hand movements corresponded to a cursor position displayed to the animal on the LCD monitor. The virtual reality environment was calibrated such that 1 cm of lateral hand displacement corresponded to 1 cm of cursor movement.

3.2.2 Speed-Accuracy Task

In our speed-accuracy task, the monkey had to reach as fast as possible to a small reach target. Each trial began with the appearance of a green circle (17 mm diameter) in the center of the screen. To initiate a trial, the animal moved a circular red cursor (6 mm diameter) onto the green circle by moving the cursor fully within the central target. The entire cursor needed to be fully enclosed within the target to continue with the trial. After holding the cursor within the central target for 250 ms, a reach target (11 mm diameter) appeared 8.5 cm from the central target. The reach target remained visible while the animal held the cursor in the central target for a variable delay period. The delay period lengths could be 200, 300, 400, or 500 ms, and they were drawn randomly from a uniform distribution. At the end of the delay period, the central target disappeared, signaling the animal to reach to the cued reach target. The animal had 625 ms to reach the target. Once he acquired the reach target, the animal had to hold the cursor fully within the target for 400 ms. Upon successful completion of the target hold, the animal received a water reward.

We adjusted the parameters of the task such that the animals performed at an overall success rate of around 70%. We chose this success rate based on human literature showing that choking under pressure only occurs on difficult tasks (Chib et al., 2012). If we made the task too difficult, however, the monkeys would give up and stop working. To make the task more difficult, we fine-tuned the reach time and the size of the reach target. These parameters required the animals to reach quickly and accurately to the targets. For all data shown here, a 625 ms reach time was used. Monkey N had an 11 mm diameter reach target while Monkey F had a 10.5 mm diameter reach target.

The task had three possible failure modes. First, if the cursor left the central target prior to the go cue, the trial failed. We term these “false-start failures.” Second, the trial failed if the cursor did not reach the cued target within the allotted reach time. We term these “reach time-out failures.” Finally, the trial failed if the cursor left the reach target prior to finishing the 400 ms hold. We term these “target-hold failures.” When a trial failed, the cursor froze at the point of failure for 300 ms to provide the animal with visual feedback about his failure mode. To encourage the animals to perform all trials equally well (even those with the smallest rewards), when the animals failed a trial they had to perform an extra unrewarded reach before a new rewarded trial would be initiated. These unrewarded trials did not end until the animal acquired the reach target.

The task had two potential reach targets: left or right. One randomly selected target appeared on each trial. The reach target conveyed the potential reward size for successfully completing the trial. For Monkey N, a shape inscribed within the reach target conveyed the reward size; for Monkey F, the color of the reach target conveyed the reward size. The different reward cues were not isoluminant. There were 4 potential reward sizes in this task: small (0.1

mL), medium (0.2 mL), large (0.3 mL), and jackpot (2.0 mL). We displayed the jackpot reward on 5% of trials, and the small, medium, and large rewards were evenly divided on the remaining 95% of trials. We designed the reward percentages in this way as a balance between maximizing jackpot reward size and the number of trials that would be completed within a session before the animals became too satiated. We verified the animals' knowledge of the reward cues through a two-target choice task. The reach time and reach target size were the same in this choice task as in the speed-accuracy task. Each of the two reach targets cued a reward size. The animals could reach to either target and receive the reward cued at that target. Monkey N chose the higher reward on 90% of choice trials and Monkey F chose the higher reward on 100% of choice trials.

3.2.3 Precision Task

In addition to the speed-accuracy task, Monkey F performed a precision task. The animal was presented with a curved path to follow to reach the target. The path started at the center target and ended at the reach target. The paths had a single inflection in either the clockwise or counterclockwise direction. The center of the cursor had to stay within the path in order to successfully complete the trial. This task emphasized precise movements rather than speed. The allotted reach time was 2000 ms, the delay period lasted between 300 and 800 ms (drawn from a uniform distribution at 100 ms intervals), and the reach target was held for 400 ms. The reward sizes and cues were the same for Monkey F in both tasks.

For the precision task, we varied the “punishments” that happened after a failed trial. We had four levels of punishment associated with the task, and they were also cued at the reach target by the shape inscribed within the target. The punishments corresponded to the number of unrewarded reaches the animal had to make prior to initiating a new trial. We did not see any

change in behavior associated with the different punishments; therefore, we have combined the data analyzed here across all different conditions.

3.2.4 Success Rate Analysis

We analyzed the success rate as a function of reward size. We did this for each individual session and for all sessions combined. For each individual session, we measured the success rate at each reward size. We computed the overall success rates for each reward by combining all trials from all sessions and measuring the success rate at each reward. To compute a confidence interval for the success rates, we grouped all trials from each reward. For each reward size, we randomly selected, with replacement, a new set of trials corresponding to the total number of trials for that reward. Then, for those randomly selected trials, we calculated the success rate. We did this 10,000 times for each reward and found the 95% band. To test for significant differences in performance at each reward size, we used a binomial proportion test.

We also calculated success rates for each reward at specific epochs of the trials. These “epoch success rates” were used to ascertain why the monkeys failed the task and how reward size affected task failure. When calculating these success rates, we only used trials that made it to each epoch. We calculated a “delay period success rate” as the percentage of trials that successfully completed the delay period; a “reach success rate” as the percentage of trials successfully completing the reach; and a “target hold success rate” as the percentage of trials successfully completing the target hold (that is, successfully completing the trial).

3.2.5 Kinematic Analysis

We analyzed the animals' kinematics to determine how behavior changed for each reward. We calculated reaction times, reach times, and peak speeds for each trial where the animal made it to the reach epoch of the task. To compute these quantities, we used the hand position trace for each trial. We computed the speed by differentiating the horizontal and vertical components of the position (calculating the velocities), and then taking the square root of the sum of the squared horizontal and vertical velocities. We smoothed the speed trace using a 30th order FIR filter. The "peak speed" is the largest value of the speed trace. The "movement onset time" is the time when the speed first exceeded 20% of the peak speed. The "reaction time" is the time from the go cue to the movement onset time. The "reach time" is the time from movement onset until the speed drops below 20% of the peak speed. We computed the average and standard error of the mean of each kinematic value for each reward for each individual session and for all sessions combined. Once these quantities were computed, we compared them between different reward sizes using Welch's t-test due to the unequal sample sizes between rewards. In addition to the kinematic values above, we computed hand positions at different times during the reaches. We calculated these positions for each trial, and then calculated the average and standard error of the mean for each reward for all sessions combined. We found the hand positions at 25 ms intervals between the time of the go cue and 625 ms after the go cue. We again compared the average hand positions at each time point across reward sizes using Welch's t-test. All analyses on our data were performed using custom software written in Matlab.

3.3 Results

We trained two monkeys to perform a challenging reaching task that required them to make fast, accurate reaches (hereafter referred to as the “speed-accuracy task”) (Figure 3-1A). In this task, animals were instructed to reach from a central target to one of two possible reach targets in the workspace. We titrated the difficulty level of the task by reducing the reach target diameter and the time allowed to complete the reach (Figure 3-11B). The task parameters were set such that the animals had an average success rate of roughly 70% throughout a session. We informed the animals of the stakes prior to them making a reach by presenting cues about which of the four different reward sizes would be received upon successful completion of the task (Figure 3-1C). After a brief delay period, the animals were given a go cue and permitted to reach to the target. If they acquired the target in time, and held the cursor within it for 400 ms, they were provided with a liquid reward of the volume indicated by the cue.

To establish that the animals understood the cues, we used a two-target choice task (Figure 3-2A). This task presented the animals with two reach targets, each showing a different reward size, and the animals were free to reach to either target upon receiving the go cue. If the animals understand the different reward sizes used in the task, they will consistently choose the higher of the two rewards presented in the two-target choice task. Our animals demonstrated a strong understanding of the reward cues, with Monkey N choosing the higher reward on 90% of choice trials and Monkey F choosing the higher reward on 100% of the trials (Figure 3-2B). After establishing an understanding of the reward cues, we collected 6 sessions of behavioral data from the speed-accuracy task with Monkey N and 8 sessions with Monkey F. We selected this number of sessions to have over 250 jackpot-reward trials for each animal.

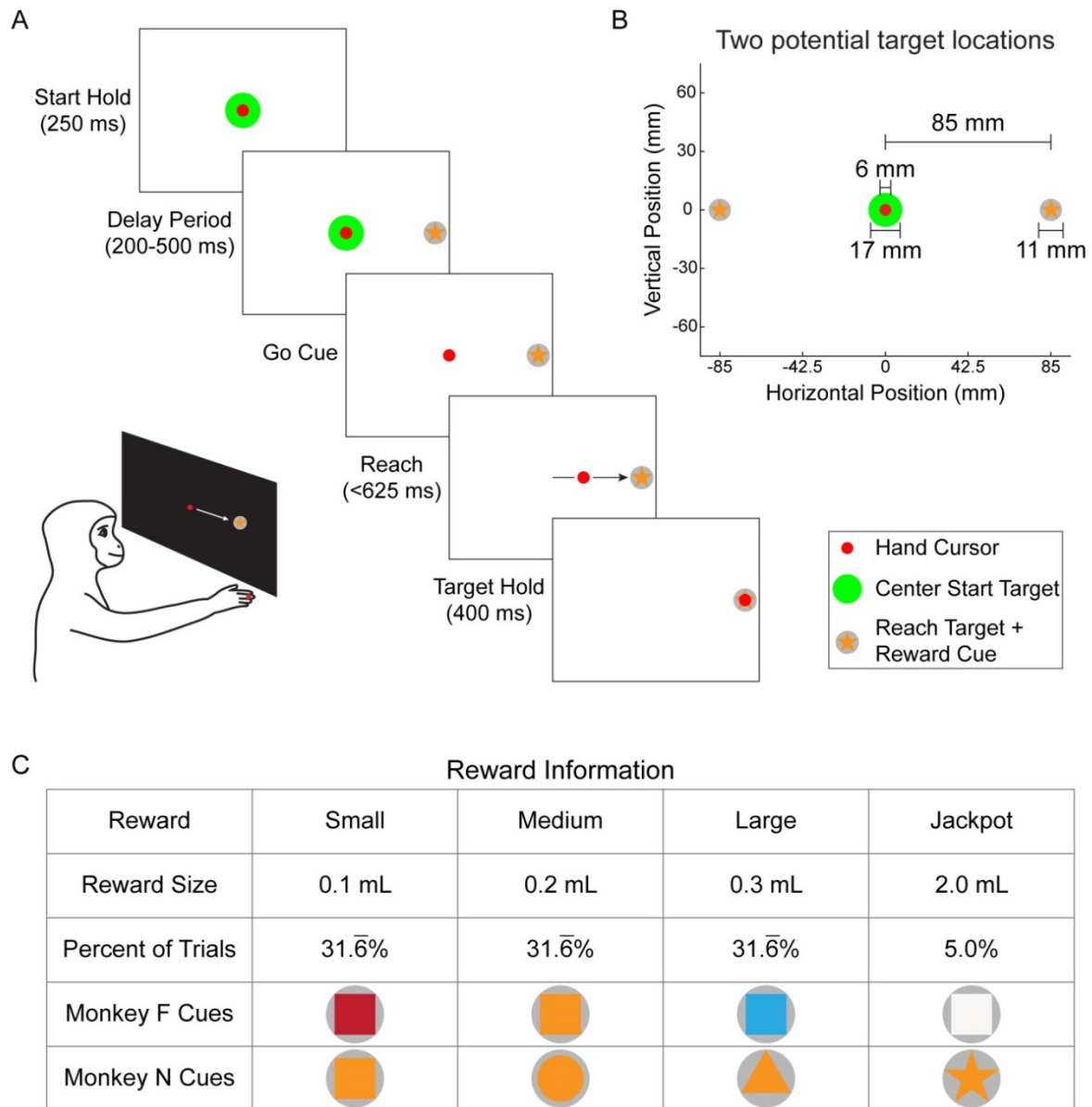


Figure 3-1 A speed-accuracy task for studying choking under pressure in monkeys. (A) Task progression. Monkeys were required to first place their hand cursor (red circle) within the start target (green circle). Once the start target was acquired, the reach target appeared (gray/orange circle) and showed the animal the potential reward available for the trial. After a short, variable delay period, the start target disappeared, cueing the animals to reach to the peripheral target. Animals were required to reach the target within 625 ms of the go cue and hold the cursor within the target for 400 ms in order to receive the cued reward. Targets not drawn to scale in this panel. **(B) Task layout.** This task was designed to emphasize fast reaches and accurate movements. Each animal had target sizes adjusted to keep the overall task success rate near 70%. Two

potential reach targets were used: left and right. For both animals, reach targets were spaced 85 mm from the center target. Monkey N (illustrated) had reach targets of 11 mm diameter, and Monkey F had reach targets of diameter 10.5 mm. Cursor and start target were the same size for both animals. (C) Reward cue information for each monkey. Each animal was presented with one of four potential reward sizes for each trial. The jackpot reward was given on 5% of trials in order to keep it highly motivating. The other rewards were evenly distributed over the remaining 95% of trials. The reward cues were given at the location of the reach target. For Monkey F, rewards were cued by color, and for Monkey N, rewards were cued by shape.

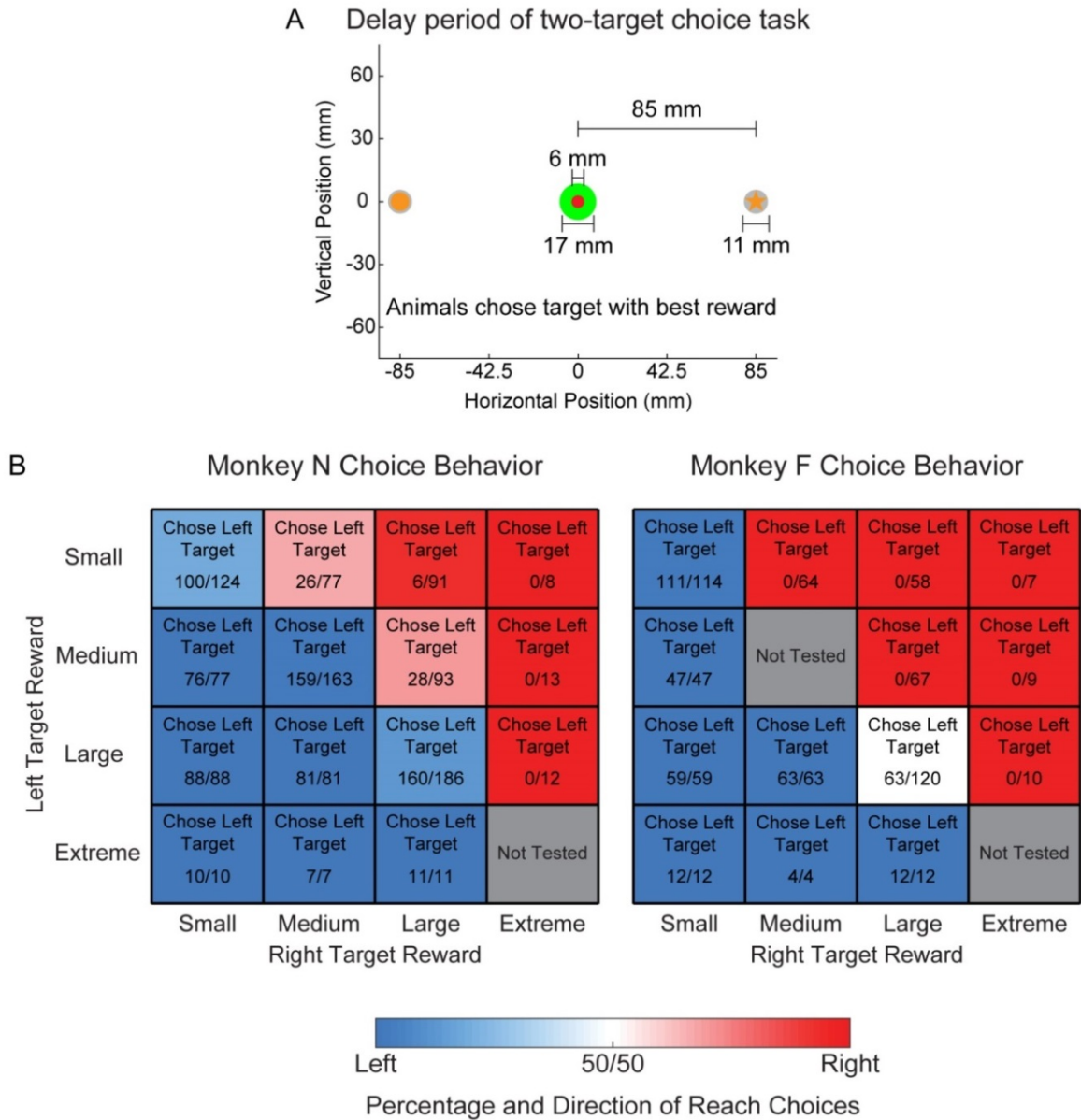


Figure 3-2 Both animals understood the cues for each reward size. (A) Choice task layout. Each trial presented the animal with two potential reach targets. The goal was to have the animals reach to the target with the higher reward. The target sizes and reach distances were the same as in the speed-accuracy task. (B) Choice behavior in each animal. Monkey N (left) chose the higher reward on 90% of trials. The trials with matching rewards (main diagonal of grid) revealed a leftward bias to his choices, which may account for his incorrect choices when the higher reward was at the rightward target. Monkey F (right) chose the higher reward on 100% of trials. This animal also had a bias to the left target, but it was not as strong as Monkey N and did not affect his choice behavior.

Both monkeys choked under pressure. We defined choking under pressure as a decrease in performance from the large reward to the jackpot reward. We analyzed the animals' performance by looking at success rates for each reward in several different ways. First, we looked at success rates for each reward for all sessions combined (Figure 3-3). Combining sessions gave us higher trial counts in each reward category, allowing for greater statistical power. The combined data had 280 jackpot-reward trials for Monkey N and 263 jackpot-reward trials for Monkey F. The other three reward sizes had an average of 1891 and 1660 total trials for Monkey N and F, respectively. In both animals, performance increased with each reward from small to large. For the jackpot reward, however, performance decreased. Monkey N decreased performance from the large to jackpot reward by 25.4% (from 71.1% correct to 45.7%; $p < 10^{-17}$, binomial proportion test) and Monkey F had a 9.8% decrease (from 72.2% to 62.4%; $p = 0.0011$).

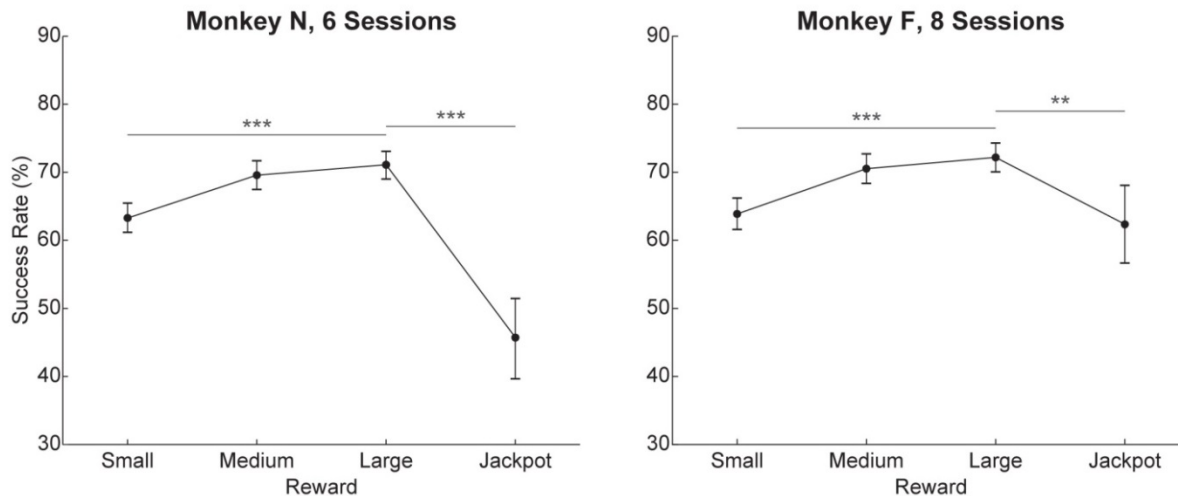


Figure 3-3 Monkeys choke under pressure. Both animals (left, Monkey N; right, Monkey F) increased performance with increasing reward from the small to large reward. Then, from the large to jackpot reward, performance decreased. The points at each reward represent the average success rate on trials of that reward. The error bars represent the 95% confidence interval of the average success rate computed using a bootstrapping technique (see methods). Significant differences between success rates at each reward were computed using a binomial proportion test. Significance levels are indicated with asterisks as follows: **: $p < 0.01$, ***: $p < 0.001$. For visual clarity, we only show differences in performance between large and jackpot rewards as well as small and large rewards. See Table 1 for all comparisons.

Next, we examined the reliability of choking under pressure (Figure 3-4). We analyzed sessions individually to see if the animals continue to show the effect with repeated exposure to the jackpot reward. Monkey N exhibited a decrease in performance from the large to jackpot-reward trials in all 6 sessions for which data were collected. On average, Monkey N performed $25.8 \pm 7.1\%$ (mean $\pm$ SE) worse on the jackpot-reward trials than the large-reward trials. Monkey F performed worse at the jackpot reward than the large reward for 7 out of 8 sessions. For the 7 sessions that showed a decrement, his performance on the jackpot-reward trials was $13.9 \pm 8.2\%$

lower than the large-reward trials. See Table 1 for a full breakdown of trial counts, success rates, and significant differences between rewards on each individual session.

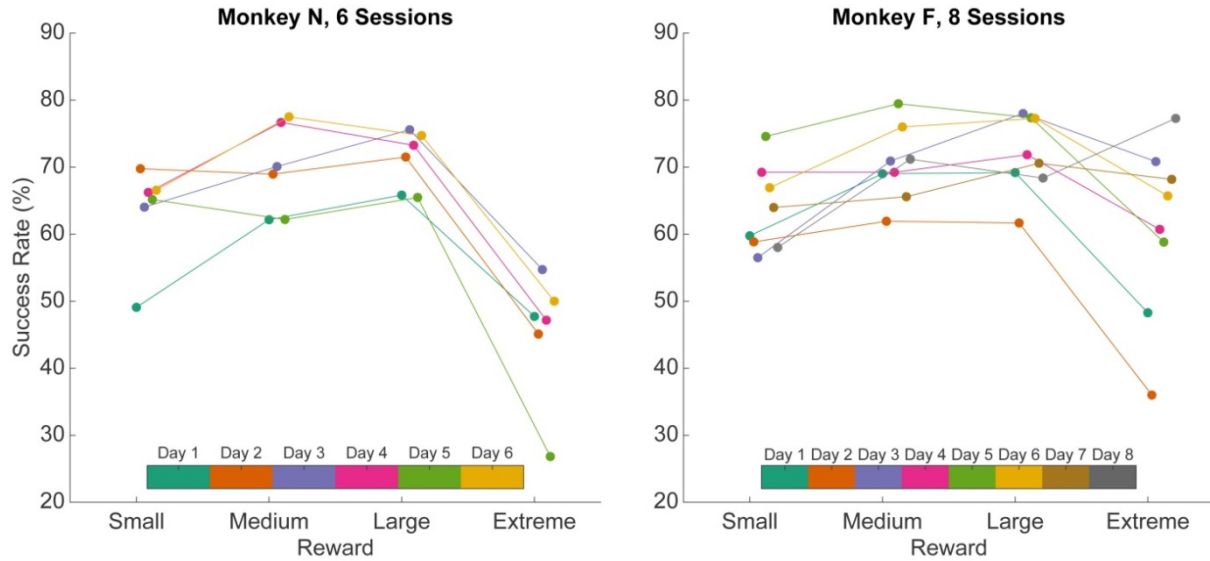


Figure 3-4 Monkeys reliably show choking under pressure for multiple sessions. Both animals (left, Monkey N; right, Monkey F) showed a decrease in success rate between the large and jackpot rewards for multiple sessions. Monkey N exhibited choking under pressure in all 6 sessions. Monkey F choked under pressure in 7 of 8 sessions. Data are jittered on the horizontal axis for visual clarity.

In the human literature, choking has been reported across a range of behavioral paradigms (R. F. Baumeister, 1984; Beilock & Carr, 2001; Beilock et al., 2002; DeCaro, Thomas, Albert, & Beilock, 2011). To examine whether choking under pressure is also a robust phenomenon in monkeys, we had one animal, Monkey F, perform a precision task (Figure 3-5A). The animal was instructed to follow a path from the center of the screen to the reach target. The movements did not have to be fast, but they had to keep the center of the cursor within the specified boundary (Figure 3-5B). The reward cues were the same as for the speed-accuracy task (reach target color), but punishment cues were also given in this task as target shape (see

Methods, Section 3.2.3). We collected 29 sessions of precision task data from this monkey, which provided a total of 611 jackpot reward trials. We combined all of the sessions together and found that the animal also choked under pressure in this task (Figure 3-5C). Performance decreased between the large-reward and jackpot-reward trials by 8.1% ($p < 10^{-5}$, binomial proportion test). This phenomenon persisted throughout all 29 sessions. We found a 10.8% ($p = 0.0048$, binomial proportion test) decrease in performance at the jackpot reward when only looking at the final 8 sessions of data collected for the precision task, showing that the animal did not improve performance with increased exposure to the jackpot-reward trials.

Table 1 Success rates and trial counts for each reward and each session in each animal. The cumulative trial counts for the precision task are also included. The trial counts are broken down into successful trials and total trials for each reward. The table shows whether or not each day's reward success rate is significantly different from the other rewards for that session using a binomial proportion test. All six sessions in Monkey N showed a significant decrease in success rate from the large to jackpot reward. On average, Monkey N showed a 25.8% decrease in success rate from the large reward to jackpot reward. Seven out eight speed-accuracy task sessions in Monkey F had a decrease in success rate from the large to jackpot reward, but only 3 of those sessions showed a significant decrease. For the sessions that showed a decrease in success rate from large to jackpot reward, there was an average decrease of 13.9%. For the "Stats" column: * = $p < 0.05$, ** = $p < 0.01$, * = $p < 0.001$.**

		Trial Count Data							
		Small Reward		Medium Reward		Large Reward		Jackpot Reward	
		Successful/ Total Trials	Stats	Successful/ Total Trials	Stats	Successful/ Total Trials	Stats	Successful/ Total Trials	Stats
Monkey N, Speed- Accuracy Task	Day 1	49.1% 163/332	- ** *** ns	62.2% 184/296	** - ns ns	65.8% 210/319	*** ns - *	47.7% 21/44	ns ns * -
	Day 2	69.8% 210/301	- ns ns ***	69.0% 211/306	ns - ns ***	71.5% 236/330	ns ns - ***	45.1% 23/51	*** *** *** -
	Day 3	64.0% 203/317	- ns ** ns	70.1% 225/321	ns - ns *	75.6% 229/303	** ns - **	54.7% 29/53	ns ** ** -
	Day 4	66.2% 204/308	- ** ns **	76.7% 253/330	** - ns ***	73.3% 222/303	ns ns - ***	47.2% 25/53	** *** *** -
	Day 5	65.2% 204/313	- ns ns ***	62.2% 204/328	ns - ns ***	65.5% 203/310	ns ns - ***	26.8% 11/41	*** *** *** -
	Day 6	66.6% 207/311	- ** * *	77.5% 224/289	** - ns ***	74.7% 266/356	* ns - **	50.0% 19/38	* *** ** -
Monkey F, Speed- Accuracy Task	Day 1	59.8% 101/169	- ns ns ns	69.0% 127/184	ns - ns *	69.2% 128/185	ns ns - *	48.3% 14/29	ns * * -
	Day 2	58.9% 103/175	- ns ns *	61.9% 122/197	ns - ns *	61.7% 103/167	ns ns - *	36.0% 9/25	* * * -
	Day 3	56.5% 100/177	- ** *** ns	70.9% 117/165	** - ns ns	78.0% 149/191	*** ns - ns	70.8% 17/24	ns ns ns -

Table 1 (continued)

Monkey F, Speed- Accuracy Task	Day 4	69.2% 126/182	- ns ns	69.2% 126/182	ns - ns	71.9% 120/167	ns ns -	60.7% 17/28	ns ns -
	Day 5	74.6% 179/240	- ns ns	79.5% 178/224	ns - **	77.4% 188/243	ns ns *	58.8% 20/34	ns ** *
	Day 6	66.9% 166/248	- * *	76.0% 171/225	* - ns	77.3% 187/242	* ns -	65.7% 23/35	ns ns -
	Day 7	64.0% 151/236	- ns ns	65.6% 162/247	ns - ns	70.6% 156/221	ns ns -	68.2% 30/44	ns ns -
	Day 8	58.0% 141/243	- ** *	71.2% 168/236	** - ns	68.4% 160/234	* ns -	77.3% 34/44	* ns -
Monkey F, Precision Task	All Day s	63.1% 2485/3939	- *** ***	67.0% 2650/3957	*** - ***	71.4% 2656/3720	*** *** -	63.3% 387/611	ns ns ***

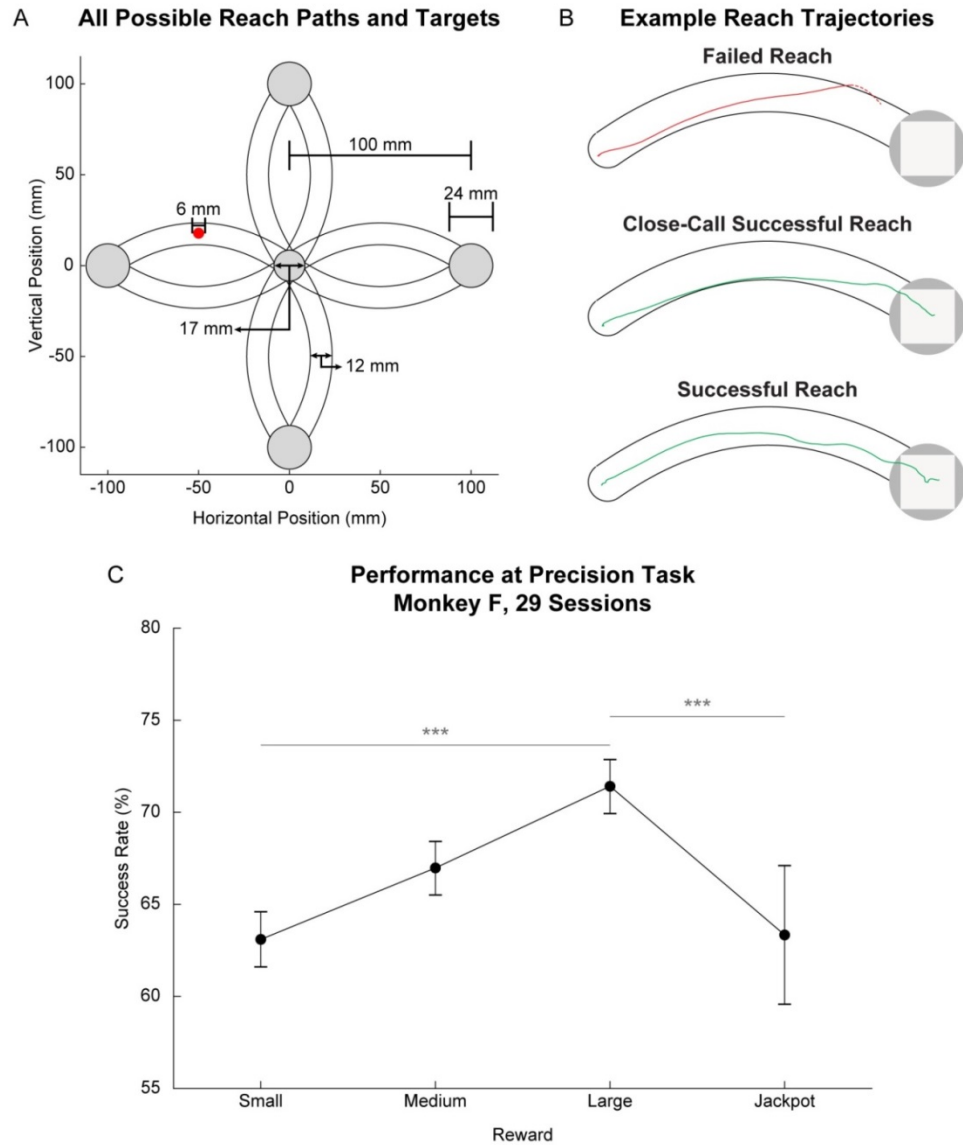


Figure 3-5 A precision task also induces choking under pressure. One animal (Monkey F) performed this task. **(A)** Task layout. The precision task required the animal to follow a pre-defined path and thus it required precise movements. This task had four potential reach targets. Each reach target had two possible paths. One of the eight target-path possibilities was presented on each trial. The reach targets were 24 mm in diameter and 100 mm from the center. The reach paths were 12 mm in width. The hand cursor was 6 mm in diameter. The reward sizes, reward cues, and percentage of trials that each reward was shown were the same as in the speed-accuracy task (as detailed in Figure 3-1A). **(B)** Three example reach trajectories. If the center of the cursor exceeded the path boundary, the trial failed immediately. **(C)** Choking under pressure also occurred in the precision task. Data for all 29 sessions is shown.

Taken together, these results show that monkeys choke under pressure and that this effect is reliable and robust. Next, we sought to determine what the animals did differently on jackpot-reward trials that led them to fail. For this analysis, we examined the data from the speed-accuracy task because behavior was more similar across trials and better suited for kinematic analysis. We investigated how various aspects of kinematics depended on reward in each monkey (Figure 3-6). Reward had subtle and different effects on behavior in each animal. Monkey N reacted more slowly when a jackpot reward was presented whereas Monkey F reacted more quickly on jackpot-reward trials. Both animals showed significant decreases in peak reach speed between large-reward trials and jackpot-reward trials, but Monkey N had a more drastic decrease in speed. Monkey F's peak speeds on jackpot-reward trials went down to similar levels seen on small- and medium-reward trials. Monkey N had significantly more time elapse between the go cue and peak reach speed on jackpot trials. Monkey F, on the other hand, reached peak speed significantly sooner after the go cue on jackpot trials. Finally, both animals reached their peak speed on jackpot trials significantly closer to the start target than on trials for the other reward sizes.

Many of the kinematic analyses showed different results for each animal, but one behavioral change that held true for both monkeys was that they both made hypometric movements (Figure 3-7). The animals reached more slowly and wound up short of the reach target by the end of the reach period. Of the failed jackpot trials, most of them (66.4% for Monkey N; 77.8% for Monkey F) failed because 625 ms elapsed before the monkey had reached the target. Each animal reached more slowly on the jackpot-reward trials in a unique manner. Monkey N reacted more slowly to the go cue and moved more slowly through the entire reach

for jackpot-reward trials. Monkey F had faster reaction times on jackpot-reward trials, but then started to slow down his reach too soon.

In addition to failing during the reach, the animals could also fail the task by moving out of the center target prior to the go cue or not holding within the reach target for the full time requirement. We found that each of these epochs could show decreased performance at the jackpot reward (Figure 3-8). Monkey N showed choking under pressure during all epochs of the speed-accuracy task. Monkey F, on the other hand, only showed choking under pressure during the reach epoch of the speed-accuracy task. Taken together, we see multiple modes of failure due to subtle changes in behavior, but the most evident behavioral adjustment that both animals made in response to a potential jackpot reward was slow, hypometric movements.

Finally, we tested whether it was just the rarity of the jackpot reward that caused the animals to choke under pressure or whether the magnitude of the reward mattered. In early experiments (296 total jackpot trials), we used a smaller jackpot reward (0.8 mL rather than 2.0 mL) with the same rate of jackpot trials (5%). We found that the same rarity of jackpot-reward trials but with a lower reward magnitude led to a smaller choking effect (Figure 3-9). The animal still performed worse at the jackpot reward than the large reward, but performance only decreased by 2% ($p = 0.3949$, binomial proportion test), which is not a significant decrease in performance. Based on this result, it appears that reward rarity on its own would not be enough to induce choking under pressure; reward magnitude also plays a role.

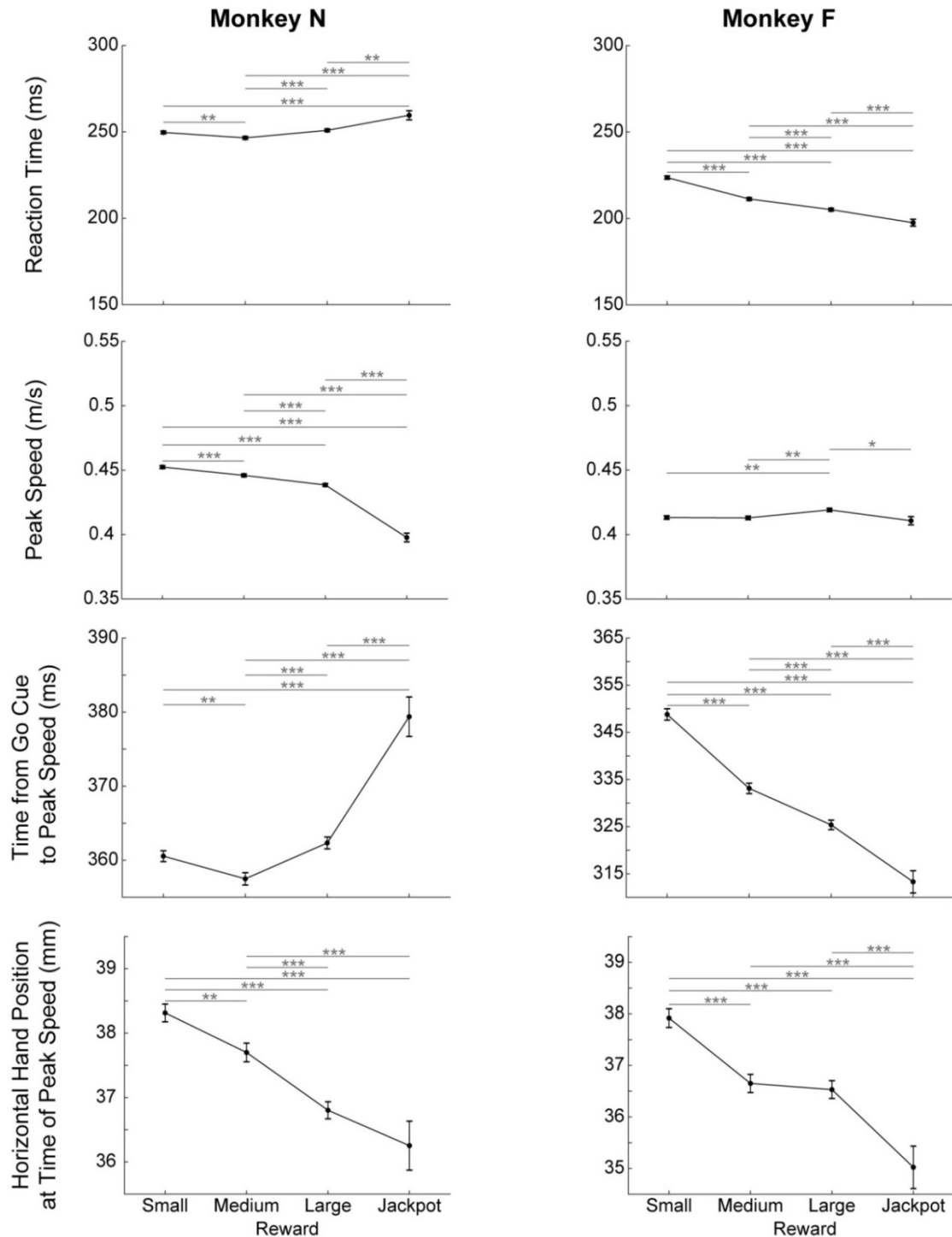


Figure 3-6 Reward had subtle and idiosyncratic effects on behavior. We analyzed many different kinematic features (6 in total) of the behavioral data from each animal (Monkey N, left; Monkey F, right). We found that reward changed behavior in unique ways for each animal. Here we show reaction times (top row), peak speeds (second row), time to peak speed (third row), and horizontal hand position at the time of peak speed

(bottom row) for each reward. The common behavioral change we found in both animals was hypometric reaches for jackpot-reward trials. Both animals failed the jackpot trials most often by not reaching the target within the allotted time constraint. The hand positions at the time of peak speed show that both animals reached their peak speed on jackpot-reward trials at a closer distance to the center of the workspace than the other rewards. This means that the rest of the reach was spent slowing down, which ultimately led to the reach falling short of the target.

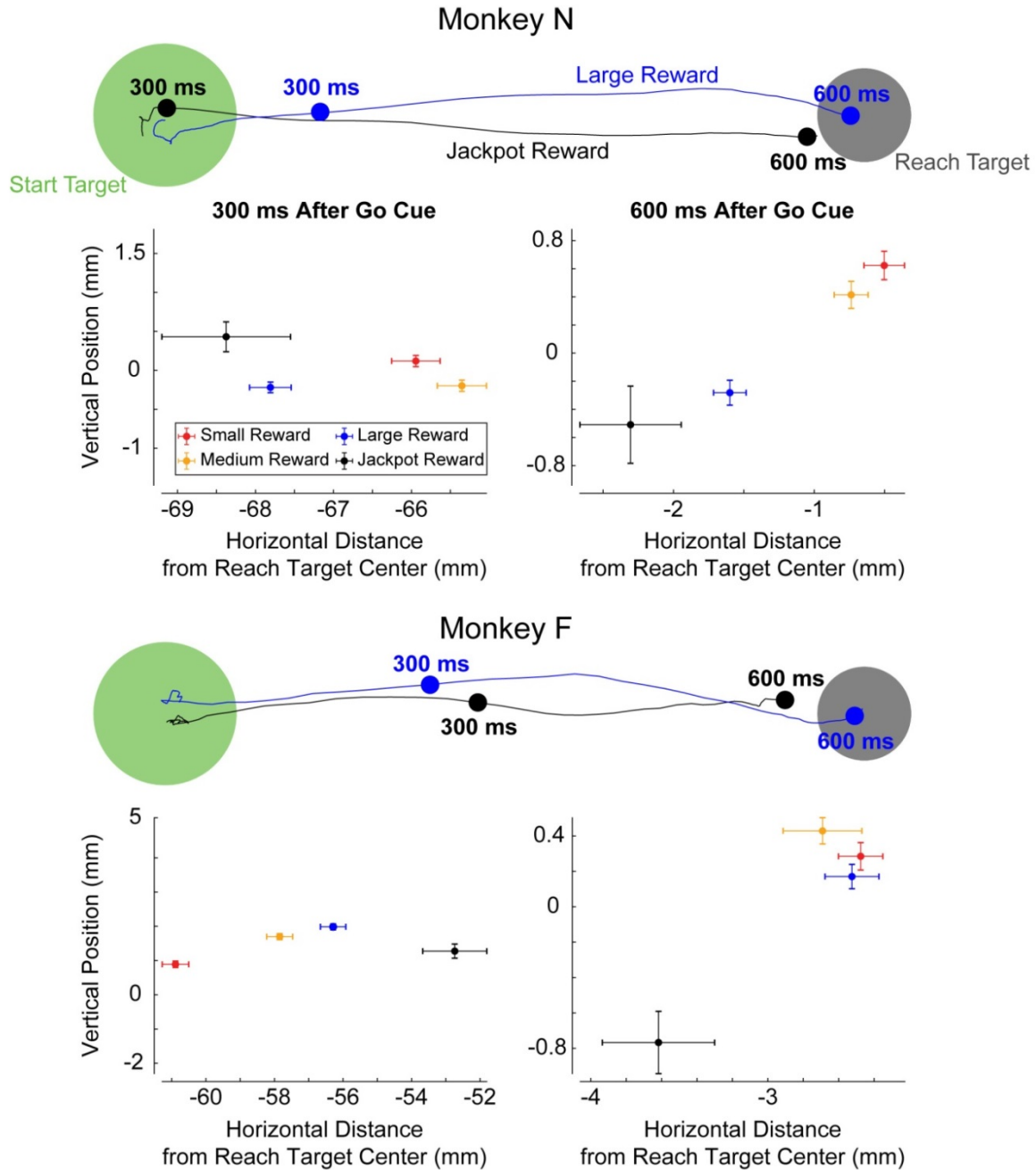


Figure 3-7 Jackpot reward led to hypometric reaching. The first row shows two example hand trajectories for Monkey N (blue, successful large reward; black, failed jackpot reward). The second row shows the average (error bars, standard error of the mean) hand position for all trials grouped by potential reward size at two key points in time: 300 ms (soon after the movement begins) and 600 ms (just before the allotted time to acquire the target expires). Monkey N (top) started slowly for jackpot reward trials then continued to move slowly throughout the entire reach. Monkey F (bottom) started his jackpot reward reaches fast, but then

slowed down too early. In the plots for Monkey N, the jackpot reward hand position lags the other reward sizes, but it does not significantly differ from the large reward at either 300 ms or 600 ms (300: $p = 0.6223$; 600: $p = 0.0812$, Welch's t-test). For Monkey F, the jackpot reward hand position significantly leads the large reward at 300 ms ($p = 0.0008$, Welch's t-test) and significantly lags the large reward at 600 ms ($p = 0.0022$, Welch's t-test). Both animals' jackpot reward hand positions differed significantly from small reward and medium reward hand positions at 300 ms and 600 ms.

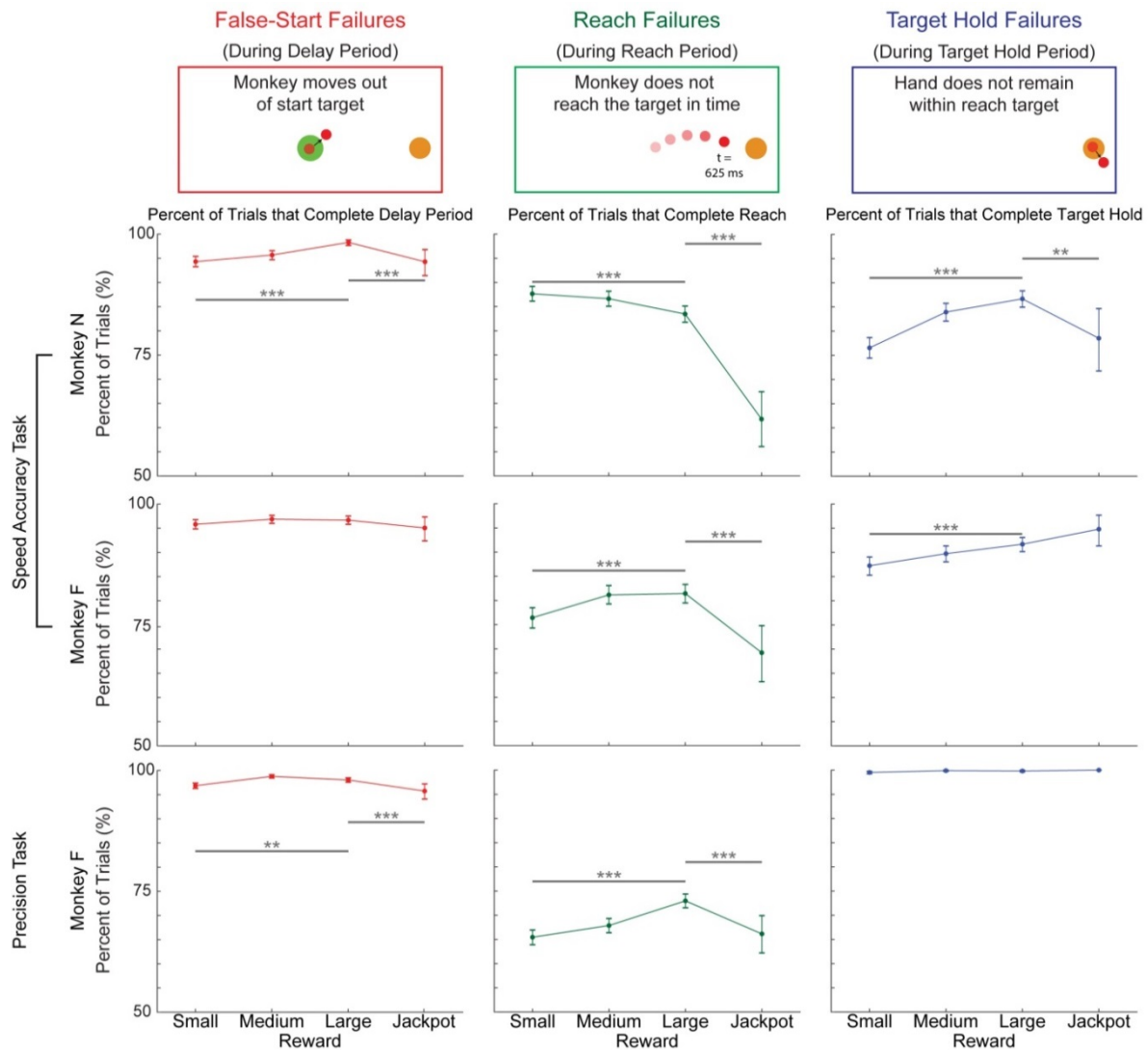


Figure 3-8 Choking under pressure can occur at multiple stages of the task. Choking under pressure can occur at multiple stages of the task. The top row shows the three different ways the animals could fail at the task: they could move out of the center target before the go cue, not acquire the reach target in time or move out of the reach path, or not hold within the reach target long enough. In Monkey N (second row), we found choking under pressure in all three phases of the task. He failed more often on the jackpot-reward trials during the delay period, the reach period, and the target hold period. During the speed-accuracy task, Monkey F only exhibited choking under pressure during the reach period. On the other hand, he exhibited choking behavior during the delay period and the reach period for the precision task. These results show that high incentives lead to multiple failure modes, and each individual changes their behavior in a different manner.

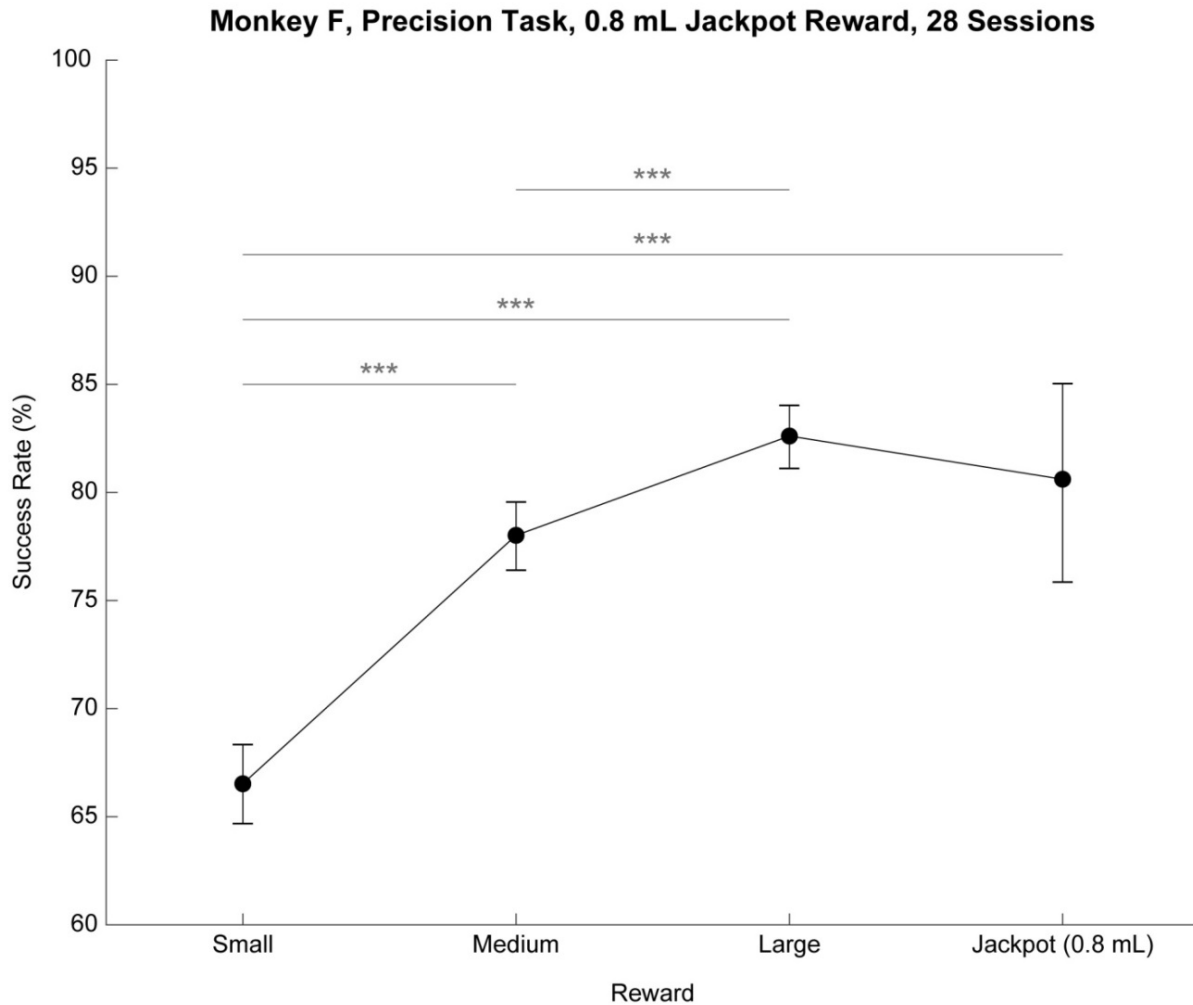


Figure 3-9 Reward rarity is not enough to induce choking under pressure. Monkey F performed 28 sessions of the precision task with a jackpot reward of 0.8 mL before switching to a jackpot reward of 2.0 mL. This smaller jackpot reward resulted in a decrease in performance from the large reward to the jackpot reward of 2%, which was not significant ($p = 0.3949$, binomial proportion test). This result suggests that rarity of reward alone is not sufficient to induce choking under pressure; reward magnitude also plays a role.

3.4 Discussion

It is known from common experience and well-established in the scientific literature that humans choke under pressure in a wide variety of situations (R. F. Baumeister, 1984; Beilock & Carr, 2001; Masters, 1992; Wine, 1971). Here we show that monkeys also choke under pressure. They did so when offered the possibility to receive a very high (“jackpot”) reward for successfully completing a challenging motor task. They exhibited this behavior consistently across days and, at least for the one animal we tested, across tasks that exerted different movement demands. To our knowledge, this is the first direct demonstration of choking under pressure in a nonhuman animal.

We sought to understand how the animals altered their behavior when they anticipated a jackpot reward. The effects of the jackpot reward on motor kinematics were subtle and nuanced. One monkey slowed his reaction times and reaches, while the other sped up his reaction times and then slowed down his reaches too early. One common behavioral change between the animals was hypometria: failure to reach the target in time was the most common failure mode for jackpot-reward trials in both animals. While human studies have not directly pointed out hypometric movements as a cause of choking under pressure, some have shown that high pressure situations lead to slow movements in human subjects (Beilock et al., 2002; Gray, 2004; Masters, 1992). The slow, hypometric movements seen in our animals match well with these human results. This may be a sign of reduced vigor in their reaches. It is interesting to speculate that reduced vigor and hypometria may be a general phenomenon that leads to choking under pressure across motor and cognitive tasks.

Several human studies have looked at how to prevent choking under pressure in high stakes situations (Balk, Adriaanse, de Ridder, & Evers, 2013; Hazell, Cotterill, & Hill, 2014;

Hill, Hanton, Matthews, & Fleming, 2011). These studies focus on emotion regulation (Balk et al., 2013) or other cognitive strategies (Hazell et al., 2014; Hill et al., 2011), but we wanted to know if increased training can be used to avoid choking under pressure. The data shown in this study come at the end of extensive training to confirm that the animals understand the task and the reward cues. By the time we started collecting the data shown here, the animals had experienced the jackpot reward for several months. Monkey N exhibited choking under pressure in all sessions. Monkey F choked under pressure in 7 out of 8 sessions collected with the speed-accuracy task. Additionally, we collected 29 sessions of the precision task with Monkey F. Even with nearly a month of consistent exposure to the jackpot reward, he still showed significant choking under pressure on the last 8 sessions of that data set. This leads to the conclusion that choking under pressure cannot be easily trained out in our animal model.

Numerous psychological explanations have been proposed for choking under pressure in humans, including explicit monitoring (R. F. Baumeister, 1984; Beilock, Bertenthal, et al., 2004; Beilock & Carr, 2001; G. A. Kimble & Perlmutter, 1970; Langer & Imber, 1979; B. P. Lewis & Linder, 1997; Masters, 1992), distraction (Ashcraft & Kirk, 2001; Beilock, Kulp, et al., 2004; Carver & Scheier, 1981; Wine, 1971), loss-aversion (Chib et al., 2012; Chib et al., 2014), over-arousal (Ariely et al., 2009; Easterbrook, 1959; Hardy, 1996), and social monitoring (Roy F. Baumeister & Steinhilber, 1984; Butler & Baumeister, 1998; C. E. Kimble & Rezabek, 1992). Although our behavioral tasks were not designed to distinguish between these hypotheses, our data are consistent with some theories and not others. In particular, our finding of slow, hypometric movements on jackpot-reward trials is consistent with the view that choking under pressure is caused in part by explicit monitoring of performance when stakes are high. Human studies have shown that expert performers who closely attend to their actions end up making

slower movements (Beilock et al., 2002; Gray, 2004). While over-arousal theories do not make predictions about how arousal affects kinematics, we can posit that movements will be more variable when arousal levels are too high. We can see hints of this effect by the monkeys moving out of the center target before receiving the go cue more often for jackpot-reward trials. Again, this effect was subtle and it affected one monkey more than the other. For our tasks, social monitoring does not explain the behaviors we saw because the monkeys were alone in the room. However, there are reports that social monitoring is important and can lead to changes in behavior in nonhuman primates (Huguet et al., 2014).

Why do we choke under pressure? Evolutionarily speaking, there is no benefit to it. And yet, something about the way the system evolved leaves this particular configuration sub-optimal in both humans and monkeys. One possibility is that there is some kind of tradeoff or limitation to the processing of rewards: something about how the system works optimizes it for performance in common cases, and this mechanism, for some reason, makes it perform worse for unlikely, extremely large rewards. But, it is precisely these situations where the circuit breaks down that give us the ability to learn the most. These cases can be particularly revealing, as they have been for visual neuroscience using optical illusions (Eagleman, 2001). We can use this animal model of choking under pressure to gain insight into the neural underpinnings of the phenomenon and the constraints of the reward processing system.

Perhaps the greatest value in our demonstration that monkeys choke under pressure is that it provides us an opportunity to examine the neurophysiological basis of choking under pressure. Functional MRI evidence suggests that this interesting phenomenon is likely to emerge from interactions between cortical and subcortical areas (Chib et al., 2012; Chib et al., 2014; Lee & Grafton, 2015; Mobbs et al., 2009). There is ample neurophysiological evidence of the impact

of anticipated reward magnitude on neural signal processing in premotor (Roesch & Olson, 2003, 2004) and parietal (Platt & Glimcher, 1999; Sugrue et al., 2004) cortices, as well as other areas (Cromwell & Schultz, 2003; Hassani et al., 2001; K. Hikosaka & Watanabe, 2000; Leathers & Olson, 2017). All of these studies raise the same interesting question: how is it that the enhanced neural activation caused by large anticipated rewards does not impact motor performance? Our findings hint that perhaps beyond a certain reward level, reward-related modulation of neural activity does indeed impact motor behavior. By using an animal model of choking under pressure, we may be able to answer this question and others like it.

This study provides a powerful new model for studying the neural basis of choking under pressure. This will have wide-ranging impacts on our understanding of how the brain processes high-pressure situations and rewards.

4.0 The Neural Basis of Choking Under Pressure

In this chapter, I describe the results of neural analyses performed on data collected from Monkey F while performing the precision task described in Chapter 3. These analyses are in the preliminary stages, and they will constitute the next phases of our studies on choking under pressure. The goal of this work was to gain an understanding of how neural activity changes due to varying reward sizes and how this might lead to choking under pressure. In this chapter, I will discuss the results seen thus far, and then discuss the next steps we will take with the analysis of the data. The analyses shown here were performed by Adam Smoulder, a graduate student in Dr. Chase's lab. I contributed to this work by collecting the data and helping guide the analyses in our weekly meetings.

4.1 Introduction

In Chapter 3, we showed that monkeys choke under pressure when rewards become extremely high. This effect occurred repeatedly across sessions and robustly on two different reaching tasks. Previously, choking under pressure had only been shown in humans (R. F. Baumeister, 1984; Beilock & Carr, 2001; Chib et al., 2012). Human studies of choking under pressure have largely focused on the psychological aspects of the phenomenon (Roy F. Baumeister & Steinhilber, 1984; Beilock & Carr, 2001; Beilock & Gray, 2012; B. P. Lewis & Linder, 1997; Masters, 1992; Wine, 1971). While these studies have provided great insight into the attentional processes that contribute to choking under pressure, they have not examined the

neural basis of this phenomenon. Recent work, however, has begun to probe the neural mechanisms of choking under pressure using functional magnetic resonance imaging (fMRI) (Chib et al., 2012; Chib et al., 2014; Lee & Grafton, 2015; Mobbs et al., 2009). This work has shed light onto which areas of the brain change their activity while performing complex motor tasks in high pressure situations. The goal of our work is to expand upon this knowledge and study how activity changes at the single neuron and population level in motor cortical areas when performing under high pressure.

Presumably, primary motor cortex (M1) outputs that activate muscles are driving the behavioral changes seen when humans and monkeys choke under pressure, but it is not known how these changes occur. Premotor cortex, specifically the dorsal aspect of premotor cortex (PMd), plans goal-directed reaching movements and sends that reach plan to M1 to be executed. Thus, it could be that PMd plays a strong role in affecting the behavior seen during choking under pressure. By studying how PMd controls this reward-mediated performance, we can gain a deeper understanding of how reward impacts motor cortical activity and how we control neural variability.

Previous work in nonhuman primates has shown that neurons in M1 and PMd modulate their firing rate due to differing reward probability (Ramkumar, Dekleva, Cooler, Miller, & Kording, 2016), varying reward value (Roesch & Olson, 2004), and receiving versus not receiving a reward (Marsh, Tarigoppula, Chen, & Francis, 2015; Ramakrishnan et al., 2017; Zhao, Hessburg, Kumar, & Francis, 2018). However, these studies do not relate neural activity to behavioral performance. Additionally, they tend to use easy tasks with high success rates, which precludes any analysis of how these areas control performance under varying reward conditions. Tasks that only show increasing success rates with increasing reward hinder analyses that

attempt to relate neural activity to success. This is where choking under pressure comes into the picture. Using a task that induces choking under pressure will cause performance to increase at first and then decrease when the stakes become extremely high. The performance follows an inverted-U, making it possible to dissociate reward level and performance while studying both in a motor context.

The goal of this chapter is to study how PMd coordinates its activity and how reward drives performance. By using a task that induces choking under pressure, we can delineate reward from performance, and address how PMd controls reward-mediated performance. We hypothesize that rewards will cause changes in the signal-to-noise ratio (SNR) of neural activity, and we will be able to see this change at the single neuron and population level. The first step to showing this effect is verifying that single neurons in PMd are sensitive to reward (Roesch & Olson, 2003). Then, we can look for this effect at the population level by combining our neural data across days and examining the stable factor space of the neural activity. Furthermore, we hypothesize that extremely high rewards will cause population reach planning activity to shift away from its ideal space and become less accurate due to changes in SNR. This shift in activity will lead to poor behavioral performance at the highest rewards.

4.2 Methods and Data Analysis

4.2.1 Subjects and task

We collected data from one animal (Monkey F) while he performed the precision task described in Chapter 3 (Figure 3-5, Chapter 3). Briefly, the task began with the appearance of a

“start target” at the center of the workspace. The animal had to move the cursor to this target and hold for 250 ms. Then, one of four reach targets appeared with a path connecting the start target to the reach target. Once the reach target and path appeared, the animal continued holding the cursor within the start target for a variable delay period of 500-1000 ms. The reach target and path stayed visible throughout the delay period. At the end of the delay period, the start target disappeared, signaling the animal to make his reach. The animal could perform a reach to the reach target by moving the cursor through the path to the target. Once the cursor was within the reach target, the animal had to hold the cursor within the target for 400 ms to successfully complete the trial. The animal failed a trial if the cursor left the start target before the go cue, if the cursor left the path during the reach, or if the cursor left the reach target before completing the target hold.

Each trial presented the animal with a different potential reward and punishment. If the animal successfully completed the trial, he received the reward; if he failed the trial, he received the punishment. We used four different reward sizes and four different punishment sizes. The rewards were the same as those described in Chapter 3 (Figure 3-1, Chapter 3), and the punishments were variable length timeouts after failing a trial. This information was presented at the reach target on each trial. The color of the reach target indicated the reward size, and the shape of the target indicated the punishment size. We presented the highest reward (jackpot reward) on 5% of trials, and the other three reward sizes were evenly presented on the remaining trials. We presented the punishments at the same rates. We found no evidence of the punishments affecting task performance or kinematics, so we combined trials across punishment sizes and only looked at the effects of reward and reach path direction.

4.2.2 Neural recordings

We collected neural data from PMd using a 96-channel electrode array implanted contralateral to the working arm. We collected action potentials from each electrode channel. The activity on each channel was hand-sorted during the experimental sessions, and we have a mixture of single-unit and multi-unit activity for our analyses. We collected 29 sessions of data, but due to recording artefacts during one session, we only analyzed 28 sessions.

4.2.3 Single unit data analysis

We analyzed data for each of the instructed paths and for each reward. We had 8 different paths and 4 different reward sizes. In order to create a detailed tuning function, we needed roughly 40 trials per reward and path (Roesch & Olson, 2004). Because we only presented the jackpot reward on 5% of trials, each reach path only had about 2 jackpot reward trials per session. Thus, we needed to combine data across days to get the desired number of jackpot reward trials per path. To do this, we identified units present across sessions using a semi-supervised Gaussian mixture model clustering algorithm (Fraser & Schwartz, 2012). Approximately 115-130 units were identified for each session. In all sessions combined we had 1361 units. For the single unit analyses, we only used units that were present for at least 15 sessions, which gave us a total of 46 units. This ensured we had at least 25 trials for each reach path with the jackpot reward, a sufficient number for preliminary analyses.

We quantified mean neural activity using trial-average peri-stimulus time histograms (PSTHs). We created PSTHs for each reward size, and aligned to either target cue onset or movement onset. We also created population PSTHs that combined all of the single units. These

population PSTHs were made based on each neuron’s preferred direction in the task epoch of interest.

4.2.4 Population data analysis

We also analyzed the neural data at the population level. Again, we needed to combine data across days. We first used the unit identification method described above in the single unit analyses. Then, we binned spike counts in non-overlapping 200 ms windows and applied a recently developed unsupervised neural stitching algorithm, known as the stitching algorithm, to find one factor analysis (FA) model that described neural population activity across all sessions (Bishop, 2015). A FA model describes the neural population firing rates in a low-dimensional space that removes mean unit firing rates and then explicitly accounts for individual neuron noise (private variance) versus fluctuations across units (shared variance). FA models can be fit with missing data, such as neurons dropping out of sessions. In our dataset, we had a large number of neurons drop out after each session, which required us to use the stitching algorithm rather than a standard random initialization of the model. The stitching algorithm first fits individual FA models to each session of data. Then, it uses units present in sequential sessions to align their models. It iteratively continues this process until all sessions have been aligned. This provides an initialization for fitting FA with missing values that achieves a consistent optimum. We used cross-validation techniques to identify the ideal number of factors to use, which for these data was 20.

After creating the FA model for all sessions, we used a diagonalization procedure to obtain orthogonal factors of equal scaling (orthonormalized factors). These orthonormalized factors (henceforth, known as factors) reflect the underlying trends of neural population

covariations and will be used for all population level analyses (Athalye, Ganguly, Costa, & Carmena, 2017; Pandarinath et al., 2018). We will again compute PSTHs for the individual factors in a similar manner to that described for the single unit analyses.

We analyzed the reach path information present in the low-dimensional space using a Gaussian Naïve Bayes decoder. This method predicted path direction for trials from each epoch's neural activity. This acts as a proxy for the reach path information represented by the neural population. In order to accurately compare across rewards with different amounts of data, we trained individual decoders on each reward type and subsampled data to ensure equal amounts of training data. We used cross-validation techniques to test the accuracy of the decoders.

4.3 Results

4.3.1 Single unit analysis

For each of the 46 units used in the single unit analyses, we created PSTHs to evaluate changes in the temporal course of firing rates due to reward size. To analyze large-scale changes in single unit firing rates, we made population PSTHs by averaging each unit's preferred direction PSTH for both the delay epoch and reach epoch (Figure 4-1). In the delay epoch (Figure 4-1, left), firing rates increased monotonically with increasing reward size starting around 100 ms after the target cue and remained separated throughout the majority of the delay period ($p < 0.001$, Kruskal-Wallis test). The separation in firing rates becomes more salient during the reach period ($p < 1 \times 10^{-4}$) (Figure 4-1, right).

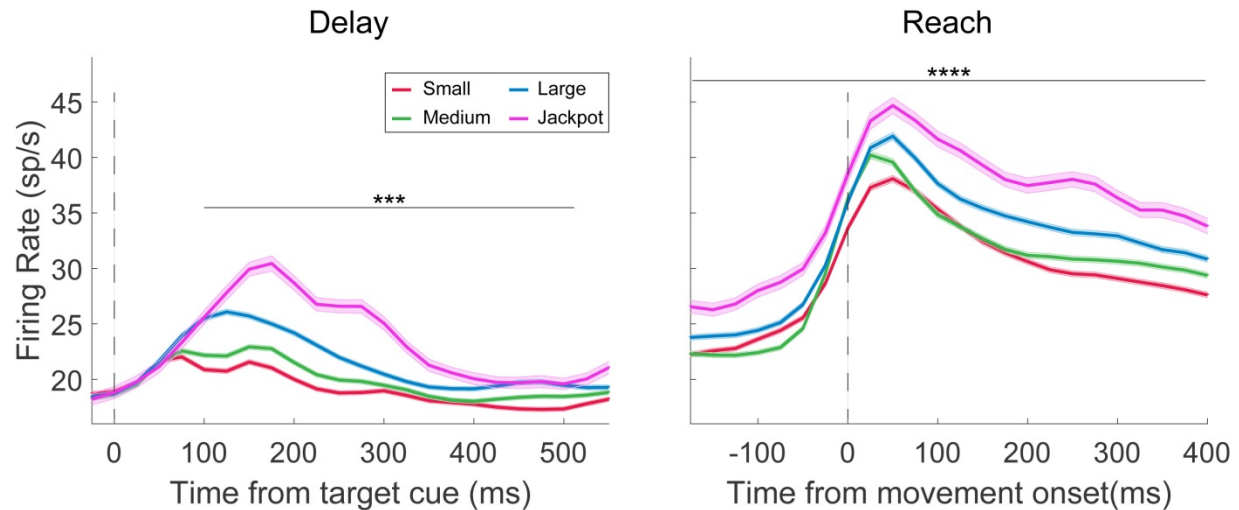


Figure 4-1 Population PSTHs for delay and reach epochs. The PSTHs for the different reward sizes increase firing rates monotonically with increasing reward during both the delay period and reach period. The black vertical dashed lines indicate target onset (left) and movement onset (right). Color traces indicate rewards: red = small, green = medium, blue = large, magenta = jackpot. Shading around PSTHs is standard error of the mean. Stars show significant reward mean-rank difference at each 25ms bin (Kruskal-Wallis test).

These results imply that at the individual neuron level, overall activity increases with reward size regardless of performance. We do not see any type of inverted-U signal in the single unit analyses, which we may expect based on performance. However, marginalizing over single units treats each unit as equally contributing to the task, which is not necessarily the case.

4.3.2 Population analysis

We used the neural stitching algorithm to find a single low-dimensional space to represent data across all sessions. We found the cross-validated log-likelihood asymptoted at 20 factors and fit the model with this. We orthonormalized these factors for equal comparison and ordered them by their amount of shared variance explained. For our analyses, we will focus

primarily on the top 10 factors since these compose > 90% of the model's shared variance (we will only visualize the top 2 factors for clarity). It is important to note that we ordered the factors based on shared variance across the whole trial; individual epochs may exhibit greater variance in different factors (Elsayed, Lara, Kaufman, Churchland, & Cunningham, 2016; Kaufman, Churchland, Ryu, & Shenoy, 2014).

Figure 4-2 shows the reach path-marginalized PSTHs of the top 2 orthonormalized factors during both the delay epoch and reach epoch. For factor 1, the reward traces were different in most of the epoch for both the delay and reach, often with monotonic separation. This result matches well with the population PSTH seen in the single unit analyses, and factor 1 accounts for the most shared variance explained in the data.

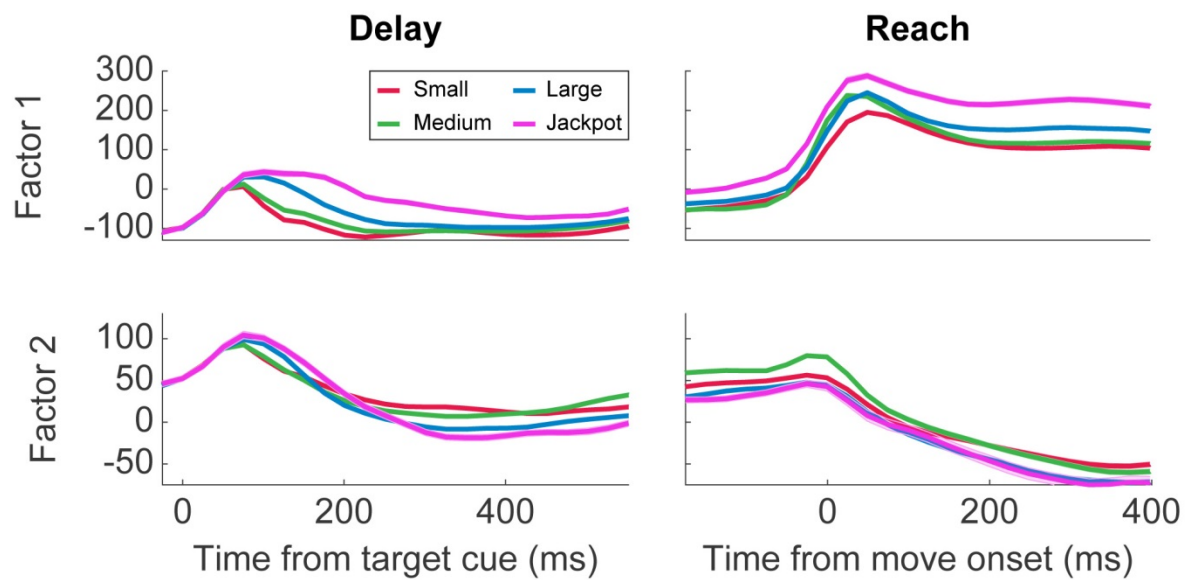


Figure 4-2 Factor PSTHs for each reward size in delay and reach. Factor 1 shows monotonic separation between reward sizes on both the delay (left) and reach (right) periods. Factor 2 does not have high separation between reward sizes in either epoch. Shading around PSTHs is standard error of the mean. Stars show significant reward mean-rank difference at each 25ms bin (Kruskal-Wallis test).

We also determined how much information about the reach path was present in the population across factors during the delay epoch and reach epoch. To do this, we trained Gaussian Naïve Bayes decoders individually for each reward size to decode reach path direction (Figure 4-3). These decoders quantified how easily identifiable each reach path was in the population activity. We found that reach path decode accuracy followed an inverted-U during the delay period, but not in the reach period. The overall accuracy across reward sizes was higher in the reach period than the delay period.

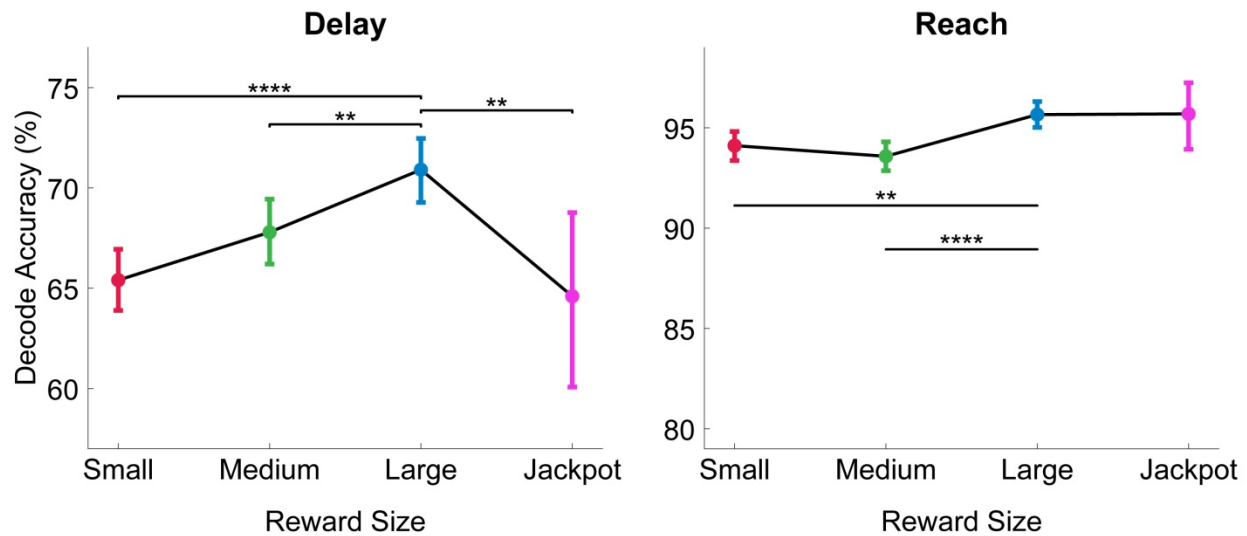


Figure 4-3 Naive Bayes decoder accuracy for population activity during delay and reach. Decoders were fit for each reward size individually for delay (left) and reach (right) epochs. Error bars are 95% confidence intervals. Stars represent significant difference in test accuracy (two-tailed t-test).

4.4 Discussion

This work studied how PMd neural activity changed at the single unit and population level when driven to improve performance by increased reward. This work is still in a

preliminary stage. Based on the analyses performed so far, we found single unit activity primarily showed monotonic increases with reward. At the population level, we found that information of the reach path during the delay epoch correlated to the behavioral effect of choking under pressure. To our knowledge, this is the first evidence of choking under pressure found in motor cortical spiking activity, and it provides an example of intracortical PMd neural activity tracking reward-mediated performance as opposed to reward size alone.

Our results match well with previous single unit studies focusing on the representation of reward signals throughout the brain. Several areas of the brain modulate their activity based on an upcoming reward (Amador, Schlag-Rey, & Schlag, 2000; Cromwell & Schultz, 2003; Hassani et al., 2001; K. Hikosaka & Watanabe, 2000; Roesch & Olson, 2003, 2004). In frontal cortical areas, upcoming rewards have been shown to affect the average delay period firing rates of single neurons as well as the strength of directional signals for the impending movement, and larger rewards often lead to increased firing rates (Roesch & Olson, 2003). While our results agree with previous work, they do not tell us anything about the behavioral significance of the modulation. There are numerous potential reasons for an increase in firing rate for larger rewards, but the current literature has not confirmed or ruled-out any of these possibilities. Studying a population of neurons may provide answers to the behavioral significance of reward modulation.

Our population decode analysis hints at a neural mechanism of reward-mediated changes in performance. In the delay epoch, information about the reach path follows the inverted-U of choking under pressure. Previous literature on motor cortex has described neural dynamics of reaching akin to a dynamical system, where the initial state is set during the delay period and the reach is executed via lawful neural trajectory dynamics (Churchland et al., 2012; Kaufman et al.,

2014). Thus, choking under pressure may be a result of a poor initial state being set before the reach by premotor cortex. The planned reach is executed properly, but the initial plan for the reach is incorrect.

Further experiments will be needed in order to fully understand how PMd controls its activity under different reward conditions. We can extend our current population results by studying neural trajectories to test the idea that the reach is planned incorrectly before execution. Using this technique, we can look for trajectories that start in a different location of factor space, and this may signal improper reach plans. Additionally, we can record from both PMd and M1 during this task to better understand the motor outputs to the muscles. This may elucidate more precisely why behavior changes when rewards become high.

Ideally, we want to find a neural signature that allows us to predict when an animal, or human, is going to choke under pressure. Our decode analysis is a first step in this direction, but a real-time measure of susceptibility to choking under pressure would help to prevent failures in the moments we want to succeed the most. Human studies using functional magnetic resonance imaging (fMRI) suggest that multiple brain areas play a role in choking under pressure (Chib et al., 2012; Chib et al., 2014; Lee & Grafton, 2015; Mobbs et al., 2009). Our future animal work will investigate multiple brain regions in order to better understand the neural mechanisms of choking under pressure.

4.5 Work in Progress

Our hypotheses for how neural activity changes during high pressure situations relate to the signal-to-noise ratio (SNR) of the variability in firing rates. The results shown above set the

stage for us to look at how SNR changes for different reward conditions. We are currently in the process of running new analyses to study the SNR at the single neuron and population levels. We plotted the tuning curves of each single neuron and each factor as a function of reach path direction. The tuning curves were created for the delay period (200-400 ms after cue onset) and for the reach period (0-200 ms after movement onset). The tuning curves provided a method of assessing mean activity changes with reward size and reach path direction as seen by baseline shifts of the curves. More importantly, the tuning functions allow for the separation of neural firing rate variance into signal and noise variance (Figure 4-4). Signal variance is the variance between mean firing rates for each reach path, and noise variance is the average variance of firing rates conditioned on a given reach path. This allowed us to define a signal-to-noise ratio that reflected a unit's tuning strength:

$$SNR = \frac{Var[E_{path}[FR]]}{E[Var_{path}[FR]]} \quad (4.1)$$

where FR is the firing rate of a unit. We examined how signal variance, noise variance, and SNR changed with reward size in both delay and reach epochs.

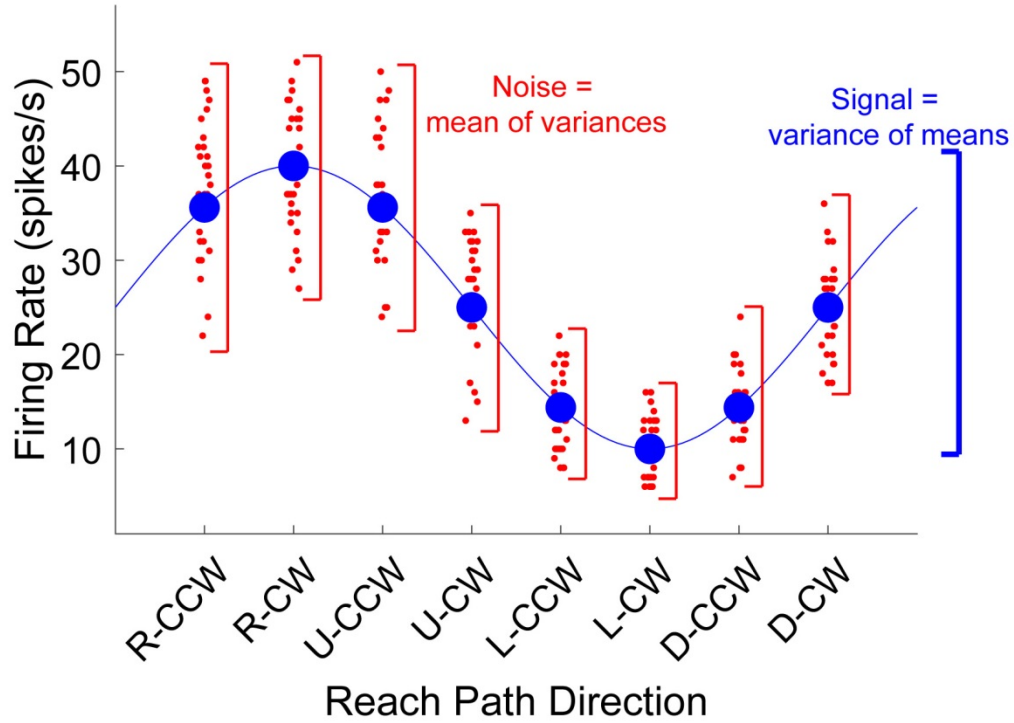


Figure 4-4 Definition of signal and noise variance. Noise variance is defined as the average scatter around each condition (reach path) mean, while signal variance is defined as the spread of the condition means. R: Right, U: Up, L: Left, D: Down, CW: Clockwise, CCW: Counterclockwise.

We used signal variance, noise variance, and SNR to quantify differences in tuning strength for the individual factors. We plotted these for the 5 most task-relevant (highest SNR) factors in each epoch (Figure 4-5). In the delay epoch, we see that signal variance across factors seems to follow either some form of inverted-U or monotonic increase, while noise variance is relatively flat across reward sizes. The SNR across factors is driven by changes in signal variance, and in some cases follows an inverted-U. In the reach epoch, two factors appear to dominate SNR (factors 2 and 3). Both factors show monotonic increases in signal variance across rewards. The noise variance for these two factors is highest at the medium reward, and there is some resemblance of an inverted-U shape centered on the medium reward. This is enough to yield a decrease in SNR at the medium reward between small and large reward sizes.

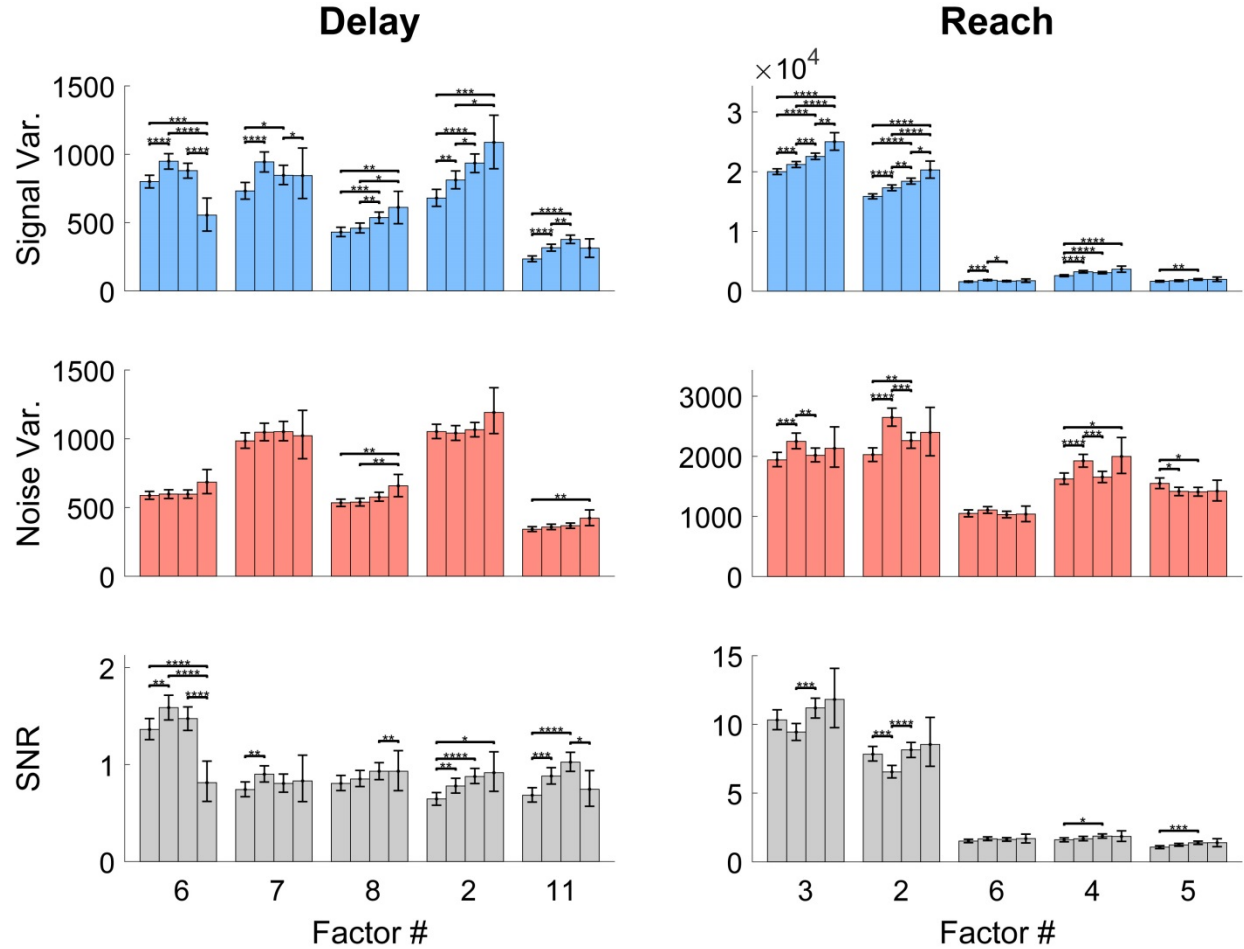


Figure 4-5 Signal variance, noise variance, and SNR for the most task-relevant factors. Changes in signal variance drive SNR more than noise variance for both the delay (left) and reach (right) epochs. Error bars are 95% confidence intervals. Factor number is labeled on the x-axis. Reward size is represented by the 4 bars present for each factor, with small reward being the farthest left and jackpot reward being farthest right. Stars represent significant difference in variance/SNR (two-tailed t-test).

From these analyses, it appears that reward size affects signal variance more than noise variance. When we initially started analyzing the neural data, we speculated that extremely high rewards might cause the neural activity to become noisy, which might result in poor behavioral

performance. After performing this SNR analysis, it appears that signal variance is the driving force behind changes in neural activity.

We will continue to analyze the data we currently have from Monkey F to determine how these changes relate to behavioral performance, reward size, and choking under pressure. Furthermore, we are in the process of collecting data from another animal performing a choking under pressure task. This animal has an array implanted in M1, and we will begin analyzing that data in the near future.

5.0 Task Demands Influence Tuning Properties of Neurons in Premotor Cortex

In this chapter, I describe my research analyzing the effects of eye position on neural activity in dorsal premotor cortex (PMd) of monkeys. Previous literature has provided conflicting results as to whether PMd uses eye-centered reference frames. This work adds to the current literature and suggests that it is task requirements that determine whether PMd represents eye position information.

Dr. Berook Alemayehu and Grant Jones contributed to this research. This work formed the bulk of Dr. Alemayehu's PhD dissertation. I collaborated with him for three years on this project. I helped collect all of the physiological data from Monkey I. Furthermore, I contributed to the neural data analysis, designed and ran the neural simulations, and analyzed the eye behavior data. The figures used in this chapter were created jointly by Dr. Alemayehu and me, and they were used in Dr. Alemayehu's dissertation (Alemayehu, 2016). This work is currently unpublished, but Grant Jones and I are working together to bring this study to publication quality.

5.1 Introduction

When making visually-guided reaches, our brains coordinate eye and hand movements in an elegant manner that allows for smooth, accurate reaches. This coordination begins with the encoding of reach goals based on eye position (Batista et al., 1999) and ends with motor commands sent to the muscles (Dum & Strick, 2002; Oby, Ethier, & Miller, 2012). Despite much

research into how eye and arm movements are planned and executed by the brain, far less is known about how these two systems work together to transform visual information into motor actions. One key piece of information missing from the literature is where in the brain this transformation takes place. Previous reports (Tanne et al., 1995) have shown that dorsal premotor cortex (PMd) plays a key role in the transition from visual representations of reach goals to motor representations. This area receives strong inputs from the posterior parietal cortex, which encodes reach goals relative to gaze (Batista et al., 1999). It also directly projects to the spinal cord, and should therefore encode reach goals relative to the hand. Despite more than two decades of research on this topic (Boussaoud et al., 1998), the extent to which PMd neurons are sensitive to the direction of gaze remains unclear.

Numerous studies show that PMd neurons are tuned to the location of the reach target relative to the hand (Batista et al., 2007; Boussaoud et al., 1998; Caminiti et al., 1991; Cisek & Kalaska, 2002; Pesaran et al., 2006; S. P. Wise, Boussaoud, Johnson, & Caminiti, 1997). When it comes to PMd neurons being tuned to the location of the reach target relative to eye position, however, previous studies begin to disagree. One study reported a weak sensitivity of PMd neurons to the location of the reach target relative to the location of the eyes (Cisek & Kalaska, 2002). However, other studies demonstrated that PMd neurons are more robustly sensitive to the location of the reach target relative to the eyes (Batista et al., 2007; Boussaoud et al., 1998; Pesaran et al., 2006). While there are important interpretational differences among these studies, the main takeaway is that three studies show a strong sensitivity to eye position in PMd neurons while one does not.

Each of the studies had their own methodological differences, but the most prominent is whether or not the animals were required to fixate their eye position. The three studies showing

sensitivity to gaze position trained the animals to fixate their eye position during the period of reach planning. On the other hand, the study that found weak sensitivity did not require fixation, but instead had unconstrained eye position. In this study, we sought to resolve this issue by designing our experiments using the methods of Batista et al. (Batista et al., 2007) with the exception that our animals did not have to fixate their eye position during the task. This will allow us to investigate if task requirements and previous training influence the information present in PMd neurons.

In this study, we had two animals perform a memory-guided delayed reaching task with unconstrained eye position. We recorded from PMd using a 96-channel electrode array. Consistent with previous studies of PMd, we found that 90% of neurons were tuned to the reach target location relative to the hand during the delay period. We also found that 86% of neurons showed tuning to the reach target's location relative to eye position. However, we ran further analyses and simulations that show this eye tuning is in fact much lower and an artifact of the natural non-uniformity of the animals' eye position behavior in the task. Our results suggest that tuning of the reach target relative to eye position in PMd neurons is virtually nonexistent in animals not trained to fixate their eye position.

5.2 Methods

5.2.1 Definitions

Throughout this chapter, I will use several terms related to reference frames that are important to define before proceeding. The two reference frames studied in this work are target

location relative to the hand (target-hand, TH) and target location relative to eye position (target-eye, TE) (Figure 5-1). A neuron has TH tuning if its firing rate is dependent on target location relative to hand position. Neurons showing TH tuning can be described as having a hand-centered reference frame. If the firing rate of a neuron depends on target location relative to eye position, it has TE tuning. A neuron with TE tuning will show changes in firing rate in response to changes of the target location relative to the position of the eyes. Target-eye tuning can be described at an eye-centered reference frame. A neuron can be described as TH, TE, or a combination of both; these reference frames are not mutually exclusive.

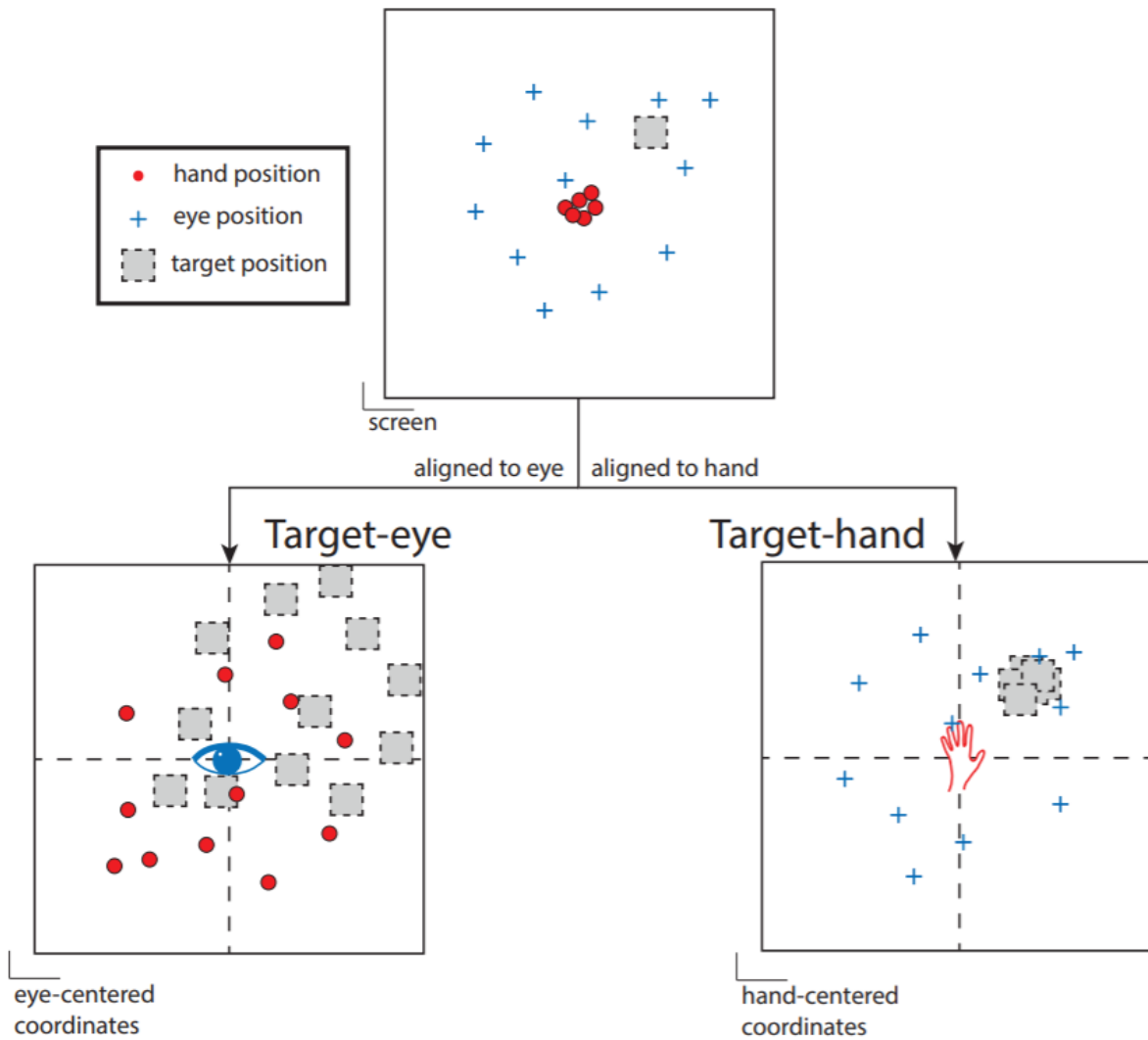


Figure 5-1 Visual representation of target-hand and target-eye reference frames. Eye positions and hand positions were recorded during each trial of the task (top). Target positions were defined relative to the eyes (bottom left, TE reference frame) or the hand (bottom right, TH reference frame). (This figure originally appeared in (Alemayehu, 2016))

5.2.2 Subjects and task

Two monkeys, Monkey I and Monkey L, performed a memory-guided delayed center-out reaching task in a virtual reality environment. The monkeys were seated in a custom-built

primate chair, and they were head-fixed using a modified halo system (Davis et al., 2009). The non-reaching arm was also lightly restrained using Velcro straps. An LED marker was placed on the animals' reaching hand, and hand positions were presented to the animals as a red circular cursor in the virtual reality environment. The hand cursor matched the movement of the animal's hand. During the experiments, the animals could not see their hand.

For the task, a trial begins with the appearance of a green square target (14x14 mm) in the center of the workspace ("start target") (Figure 5-2). The animal has to move his hand cursor to acquire the start target. Then, after holding the cursor at the start target for 500 ms, a yellow square target (14x14 mm; "reach target") appeared in the periphery of the workspace. The reach target could appear in one of eight positions equidistant from the center of the workspace, and the reach target was chosen randomly from a uniform distribution on each trial. The reach target remained visible to the animal for 200 ms. Monkey I performed a version of the task where light gray silhouettes of all eight targets remained visible throughout the trial. After the reach target disappeared, the animals held the cursor at the start target for a variable delay period (Monkey I: 1000-3000 ms; Monkey L: 500-2000 ms). The disappearance of the start target ("go cue") signaled the animals to reach to the remembered location of the reach target. They had 1000 ms to move the cursor to the reach target location. When the hand cursor reached within 20 mm of the reach target center, the target reappeared and the animals had to hold the cursor at the reach target for 350 ms. If the animals successfully held the cursor at the reach target, they received a water reward.

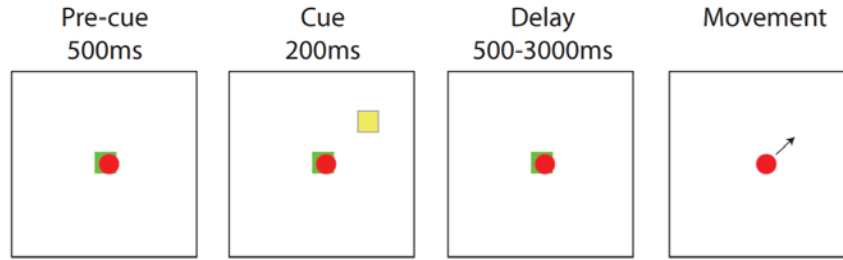


Figure 5-2 Memory-guided delayed center-out task. The animals performed this task by controlling a cursor (red circle) in a virtual reality environment. They initiated a trial by moving the cursor to a central target (green square). Then, one of eight peripheral targets appeared for a brief period (yellow square). The animals held for a variable delay period before reaching to the remembered peripheral target location when given the go cue. (This figure originally appeared in (Alemayehu, 2016)).

5.2.3 Data analysis

Our analyses for this study focused on neural activity that occurred during natural eye fixations within the delay period of successful trials. An eye fixation was defined as a period of at least 150 ms where the speed of the eye did not exceed a threshold of 50 deg/sec. The firing rates during a fixation were calculated over a window that started 50 ms after the end of a saccade until the start of the next saccade. To avoid capturing any visual transients from the presentation of the reach target, the first 150 ms of the delay period were removed from analysis.

We used planar regressions to study the relationship between firing rates and target position in each reference frame. The firing rate was regressed against the horizontal and vertical position the target in both TH and TE coordinates. In a TH regression, target position was calculated by subtracting the average hand position during a fixation from the location of the cued reach target. In a TE regression, target position was calculated by subtracting the average eye position during a fixation from the location of the cued target. For each neuron, we ran a TH

regression and a TE regression. We quantified the goodness of fit for each planar regression using the coefficient of determination (R^2). We used an F-test ($p < 0.05$) to calculate the significance of the regressions.

We used a bootstrap method to estimate the 95% confidence interval of the R^2 values. For each neuron, we randomly sampled N firing rates, with replacement, from the N firing rates calculated during a session based on the number of fixations. We did not shuffle target locations. With the new randomly sampled firing rates, we calculated a new R^2 value. We did this 10000 times, and we defined the confidence interval as the range within which 95% of the R^2 values fell during the 10000 runs.

After running the planar regressions, we were left with residual firing rate variance that was unaccounted for by the reference frame used in the regression. We tested whether this residual variance was accounted for by the other reference frame using partial regression analysis. For this analysis, we firing rate was regressed against target position in one reference frame. From this regression, we calculated firing rate residuals from the best-fit plane. Then, we firing rate residuals were regressed against target position from the second reference frame. This provided us with a partial R^2 value that quantified how well the second reference frame accounted for the unexplained firing rate variance. We calculated 95% confidence intervals for the partial regressions in the same way as the planar regressions.

We investigated the effect of unconstrained eye position on our reference frame analyses by simulating neurons with known tuning properties. We used a linear firing rate model to construct the simulated neurons:

$$FR_{i,j} = b_{0i} + b_{xi} * x_j + b_{yi} * y_j + \varepsilon_{i,j} \quad (5.1)$$

where b_{0i} , b_{xi} , and b_{yi} are the coefficients taken from the planar regression analysis of neuron i ; x_j and y_j represent the horizontal and vertical components of the target location during fixation j ; and ε_{ij} represents noise drawn from a zero-mean normal distribution whose variance was equal to the standard deviation of the firing rate residuals from the planar regression. The target location was defined in either TH or TE coordinates, which allowed us to explicitly build neurons with tuning to a specific reference frame. We built both “TH-only” and “TE-only” neurons. The TH-only neurons only had tuning to the target relative to the hand and the TE-only neurons only had tuning to the target relative to the eye position. Once we built the simulated neurons, we ran them through the same analyses as the real neurons.

5.3 Results

In this study, we recorded from a total of 447 PMd neurons from two monkeys while they performed a memory-guided delayed center-out reaching task. From Monkey L we recorded from 171 neurons, and from Monkey I we recorded 276 neurons. We did not track neurons across sessions. Instead, we treated neurons recorded in each session as distinct from the previous day. Additionally, we collected eye position data throughout each session. The animals were free to move their eyes during a trial. All data analyzed were from successful trials.

5.3.1 Eye position behavior

We only analyzed fixations that occurred within the delay period. Each animal exhibited idiosyncratic eye position behavior (Figure 5-3). Monkey I tended to fixate his eyes opposite of

the cued reach target. For example, if the upward target was cued, he would look toward the bottom portion of the workspace. This behavior occurred in all sessions and was consistent for all reach targets. We performed a bootstrap analysis on his eye position data by randomly assigning fixation locations to reach targets. Using these randomized fixations, we calculated the average eye position for each of the reach targets. We found that the average eye positions at each reach target significantly differed between the randomized and experimentally observed eye positions ($p < 0.05$, Student's t-test). Monkey L tended to look at the start target or above the workspace during the delay period. He would move his eye position between the upper portions of the workspace and the start target, presumably waiting to receive the go cue to make a reach. The animals performed the task in a dark room, and the virtual reality environment had a black background, so we are unsure of what exactly the animal was looking at above the workspace. In this case, Monkey L may have found a visual feature in the periphery that he chose to look at while waiting for the go cue. Cisek and Kalaska (Cisek & Kalaska, 2002) also showed a similar result in their eye position behavior.

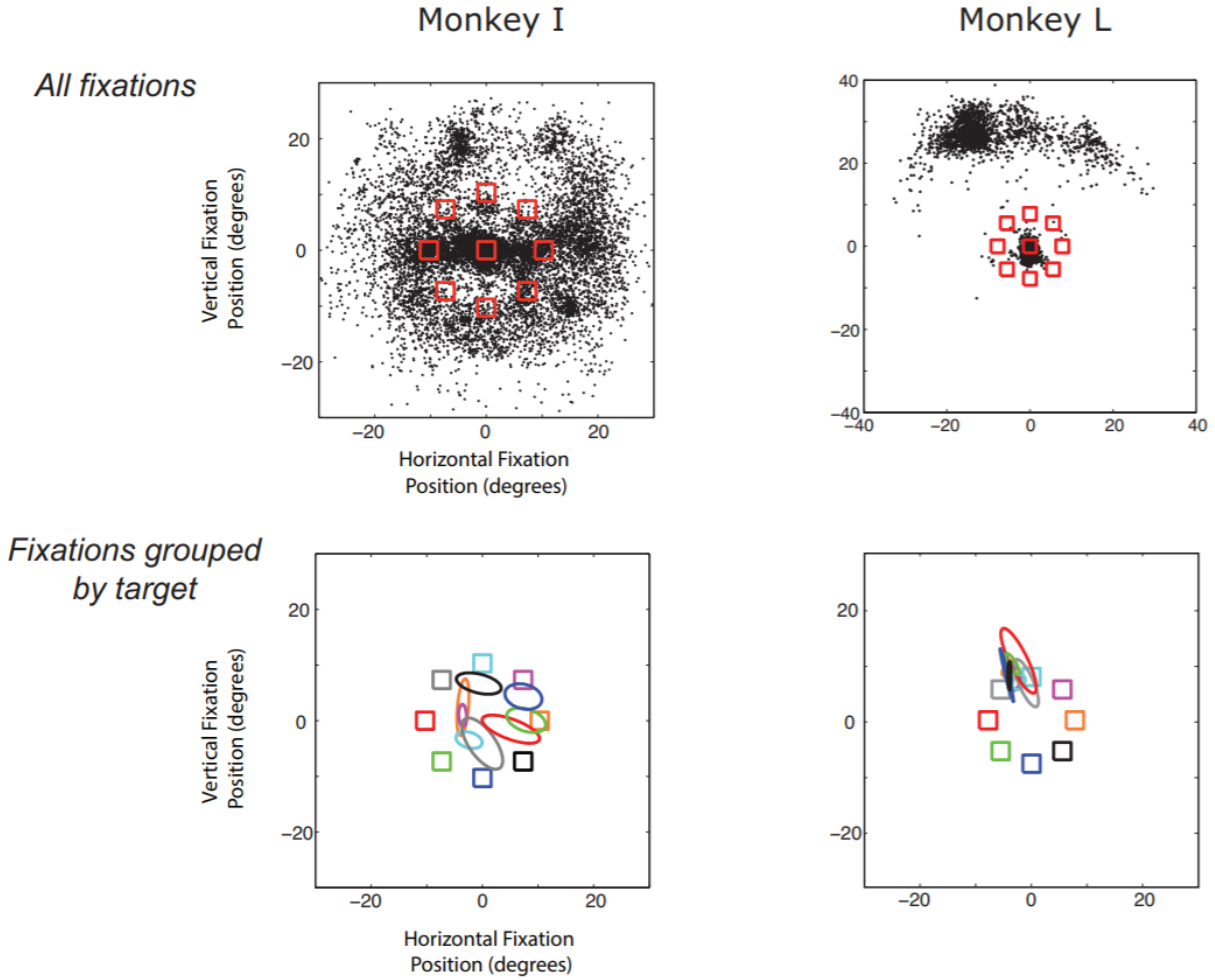


Figure 5-3 Eye position behavior of both animals. *Top:* All fixation locations during the delay period for Monkey I (left) and Monkey L (right). Each black dot represents one fixation and the red squares represent the target locations. *Bottom:* Covariance ellipses of average fixation positions during each session. Each covariance ellipse is color coded to match one of the reach targets. Both animals had consistent, idiosyncratic eye position behaviors. (This figure originally appeared in (Alemayehu, 2016)).

5.3.2 Reference frame tuning

A majority of the neurons we recorded from are represented by Figure 5-4 (top). Prior to the reach target cue, this neuron fired at its baseline level. After the cue, however, it showed a

burst of activity and increased firing throughout the delay period. It showed highest activation for targets in its preferred direction, in this case reach targets to the right of the workspace.

The firing rates of the neurons were fit to a planar regression for each reference frame. We represented these regressions as response heatmaps, which show the direction and strength of tuning in the different reference frames (Figure 5-4, bottom). In the majority of neurons (83%), we saw tuning in both hand-centered coordinates and eye-centered coordinates. The TH tuning would commonly be stronger than the TE tuning. Figure 5-5 shows the R^2 values for TH tuning and TE tuning across the population of neurons. Points that fell below the diagonal represented stronger TH tuning, while points that fell above the diagonal represented stronger TE tuning. A little more than half of the neurons (52%) were confidently TH tuned, whereas only 4% were confidently TE tuned. Thus, our neurons appear to encode reach goals in a hand-centered reference frame, but we cannot rule out an eye-centered reference frame.

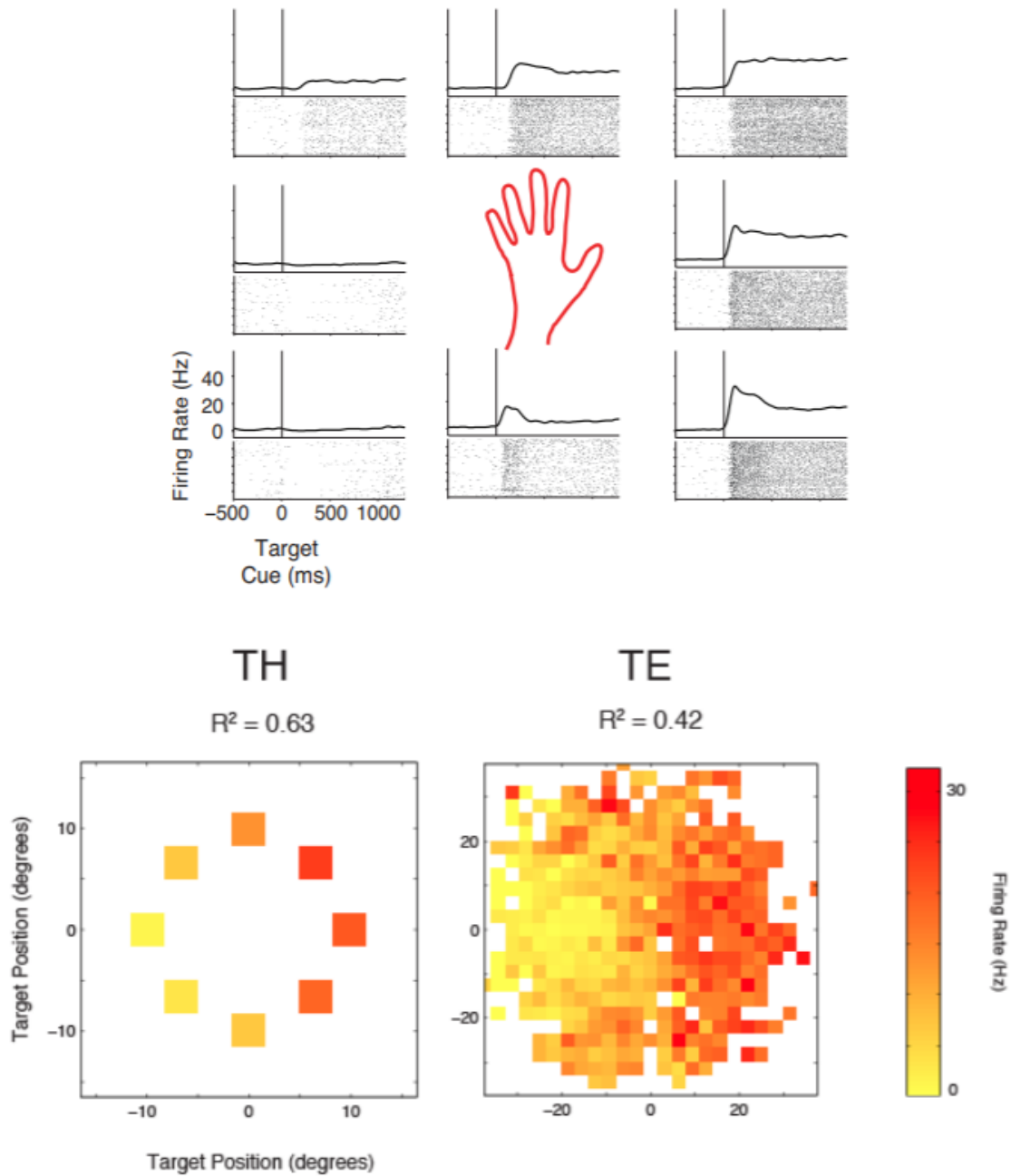


Figure 5-4 PSTH and tuning of representative PMd neuron. *Top*: Peri-stimulus time histograms (PSTHs) for each of the eight reach targets aligned on the time of target cue. Spikes that occurred 500 ms before and 1300 ms after the target cue were averaged across all trials to compute the PSTH at each target. *Bottom*: Firing rates were binned and averaged for each reference frame. A planar regression was run for both TH (left) and TE (right) reference frames, and R^2 values were computed for each regression. (This figure originally appeared in (Alemayehu, 2016)).

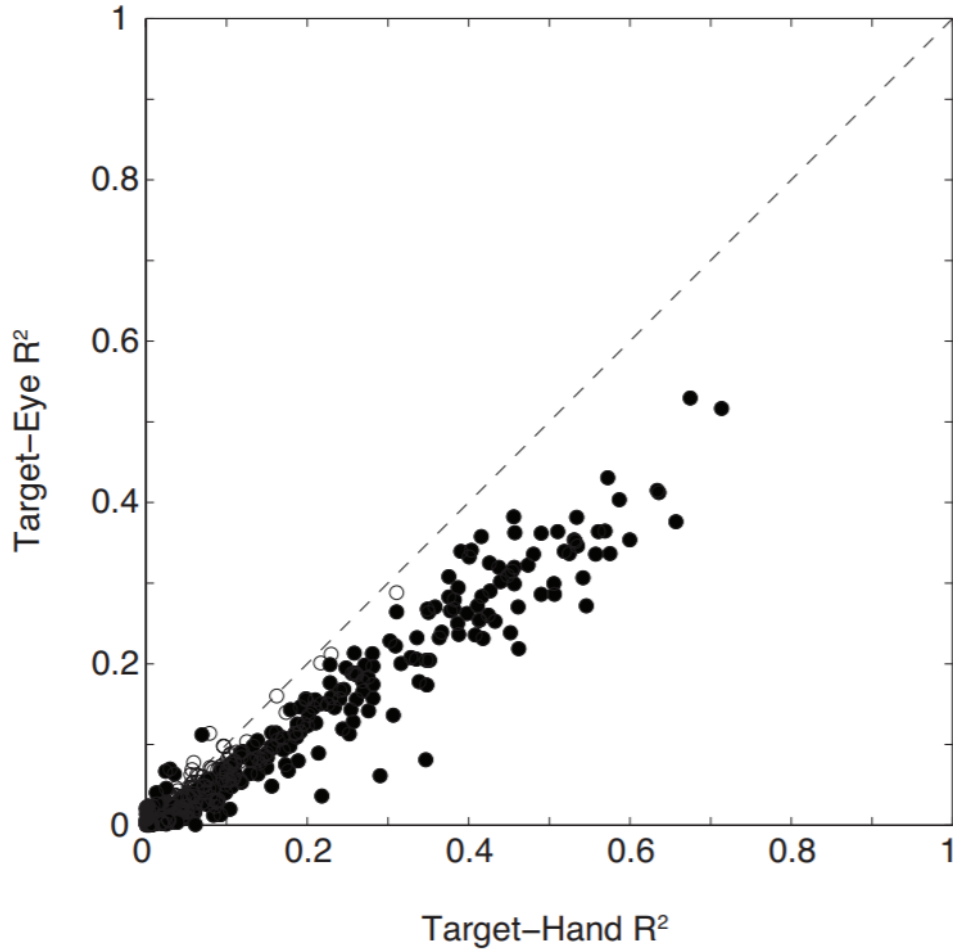


Figure 5-5 Population plot comparing TH and TE regression R^2 . Each circle represents one neuron from the population of neurons recorded from both animals. Filled circles are neurons with confidence intervals that do not cross the unity diagonal. Neurons better explained by TH tuning lie below the diagonal. Neurons better explained by TE tuning lie above the diagonal. The majority of neurons in the population exhibit stronger TH tuning. (This figure originally appeared in (Alemayehu, 2016)).

5.3.3 Target-eye tuning is nearly non-existent

The animals in our study did not uniformly explore the workspace with their eye positions. Because of this non-uniformity, we may be seeing higher TE tuning than is actually present. To make it clear why this is, consider an example scenario where a PMd neuron is

purely tuned to target location in a hand-centered reference frame. Also assume that the monkey chooses to hold his eye position directly at his hand position during the delay period. In this situation, not only would TE tuning be present, but it would look identical to the TH tuning. While this is an extreme example, any non-uniformity in eye behavior can lead to artefactual TE tuning in our neurons.

To account for the potential over-estimate of TE tuning in our PMd neurons, we ran a partial regression analysis. This will allow us to quantify how much influence each reference frame has on neural activity after accounting for the influence of the other reference frame. In our population, we found that after accounting for the variance in neural activity explained by a TH reference frame, TE tuning is low (Figure 5-6). We also performed the reverse of this: regress against TE and then regress the residuals against TH. From the partial regression analysis, we found that 36% of neurons were still significantly tuned in the TE reference frame and 76% were still significantly tuned in the TH reference frame. Nearly half of the neurons (42%) that were significantly tuned in the TE reference frame lost that tuning after TH tuning was accounted for.

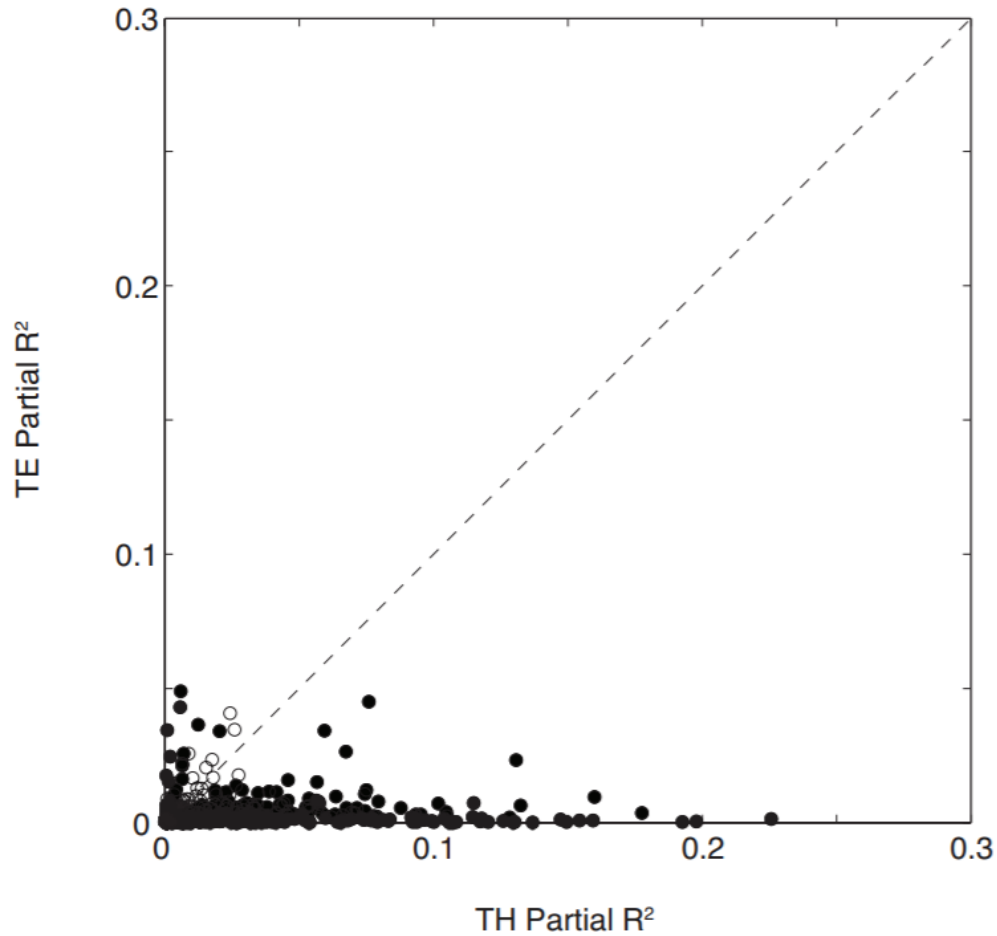


Figure 5-6 Population partial R^2 analysis. We performed a partial R^2 analysis on each neuron in the population. Firing rates were first regressed against target-hand position, and the residuals were then regressed against target-eye position (TE Partial R^2). Furthermore, we regressed firing rates against target-eye position and then regressed the residuals against target-hand position (TH Partial R^2). Each circle represents one neuron, and the filled circles represent neurons whose confidence interval does not cross the unity diagonal. Neurons that lie below the unity diagonal explain the remaining firing rate variance from a TE reference frame with a TH reference frame. Neurons that lie above the diagonal explain the remaining firing rate variance from a TH reference frame with a TE reference frame. (This figure originally appeared in (Alemayehu, 2016)).

The partial regression analysis showed that eye position did not account for much of the residual variance in our data. So what then is causing such high TE tuning in our neurons? Could the eye position behavior of our animals play a role in this apparent TE tuning? To test this, we created simulated neurons that were purely tuned in either a TH reference frame or a TE reference frame. We then ran the simulated neurons through the planar regression analyses using the actual eye position behavior from the monkeys. Figure 5-7 shows an example neuron from our population and its corresponding TH and TE simulation results. When we simulated a purely TH neuron, we see strong TH tuning, as expected. Surprisingly, we also see strong TE tuning. This is not the case in the simulated TE neuron. We see strong TE tuning in this neuron, but we do not get much TH tuning.

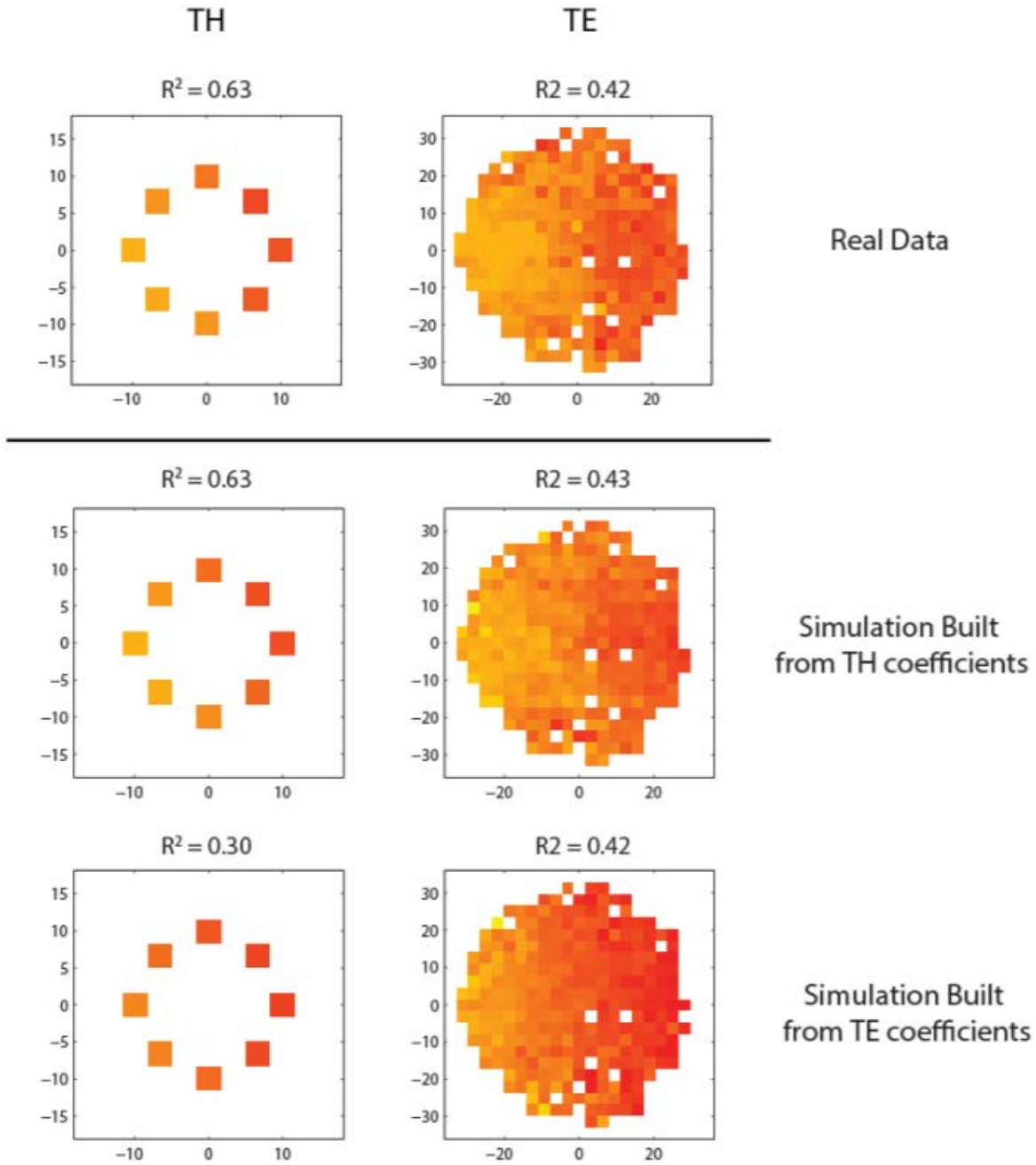


Figure 5-7 Tuning in a simulated neuron. *Top:* Tuning in TH (left) and TE (right) reference frames using real neural data from one neuron. *Middle:* TH-simulated neuron. This neuron was built to have only TH tuning. *Bottom:* TE-simulated neuron. This neuron was built to have only TE tuning. (This figure originally appeared in (Alemayehu, 2016)).

Why, then, do we see TE tuning in a purely TH neuron? Across all model TH neurons, we saw TE tuning that was as strong, or nearly as strong, as the real neuron from our population (Figure 5-8). This suggests that the TE tuning we observed in our real data is simply a result of the eye behavior exhibited by the monkeys combined with the TH tuning of the neurons. To clarify this result, we ran another simulation using a purely TH neuron and simulated eye positions that uniformly sampled the workspace. In this case, TE tuning essentially disappeared (Figure 5-9).

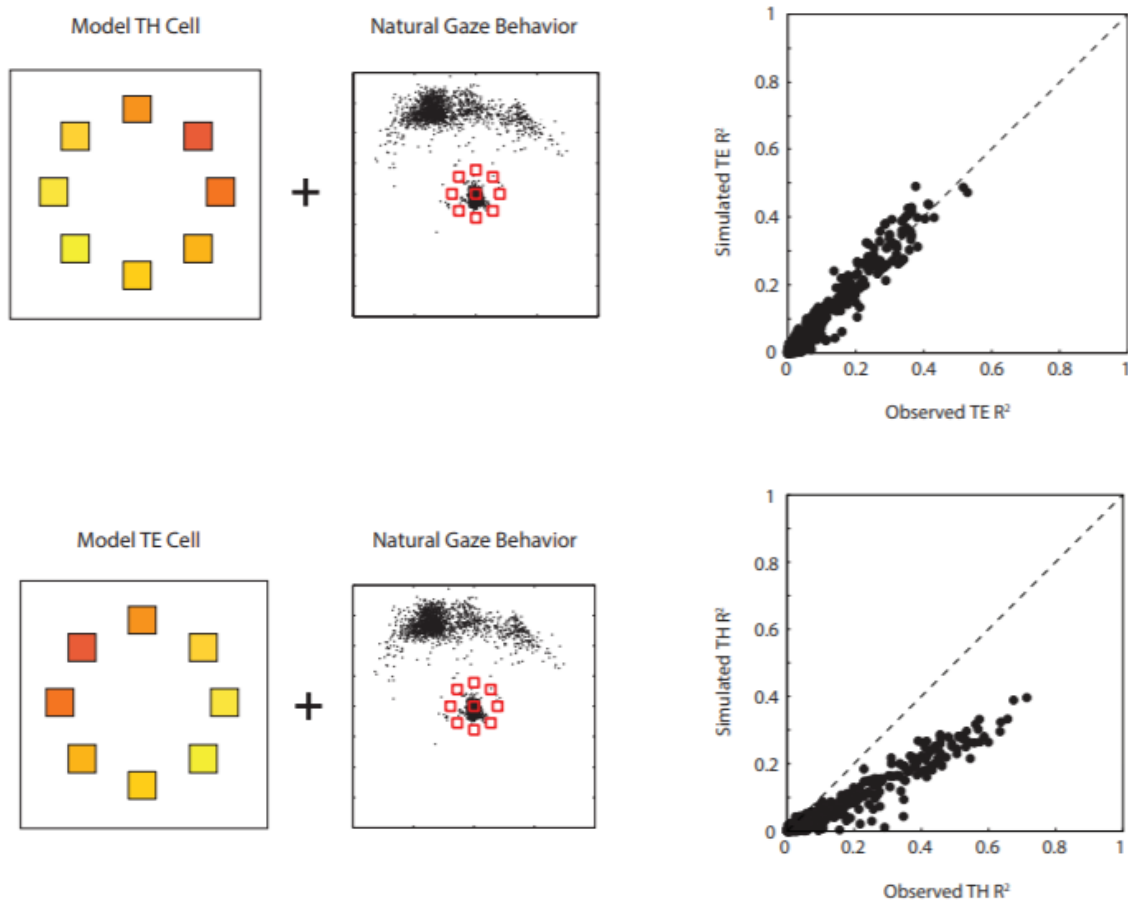


Figure 5-8 Comparison of tuning strength between real and simulated neurons. *Top:* Simulated TH neurons showed similar TE tuning to our actual neural data (top right). *Bottom:* Simulated TE neurons did not show the same level of TH tuning as the real neural data. (This figure originally appeared in (Alemayehu, 2016)).

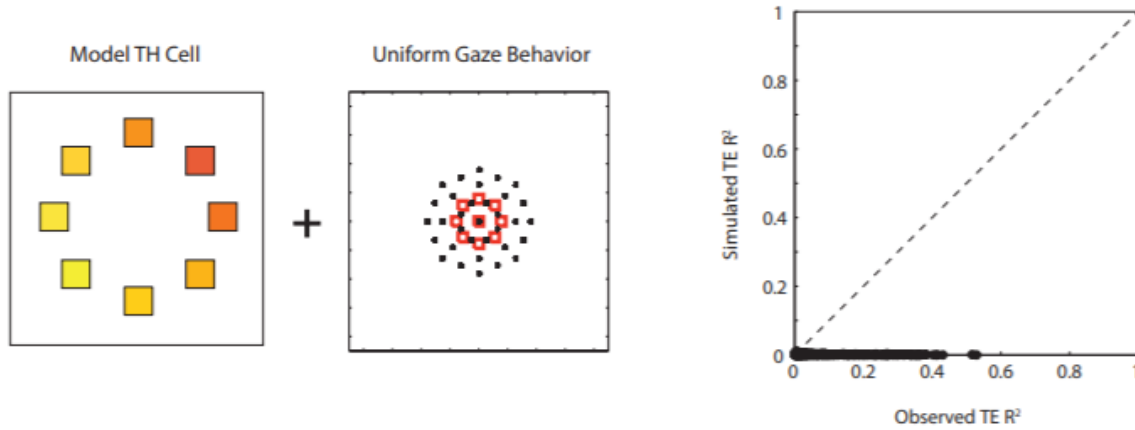


Figure 5-9 Uniform gaze eliminates spurious TE tuning. Simulated TH neurons lose their TE tuning when regressed against simulated uniform gaze positions. (This figure originally appeared in (Alemayehu, 2016)).

5.4 Discussion

This work studied the role of PMd in sensorimotor transformations. Previous work has provided discrepant results as to whether the transformation of visual information to motor information is complete before or after PMd. Here, we addressed this question by investigating the extent to which PMd neurons are tuned to target position relative to eye position. Our initial planar regressions showed tuning in both hand-centered and eye-centered coordinates for the majority (86%) of our neurons. However, only a very small percentage of our neurons (4%) had a significantly stronger tuning in a TE reference frame than a TH reference frame. Furthermore, our analyses revealed that the TE tuning seen in the planar regressions was likely due to biases in the monkeys' natural eye behavior. Based on our analyses, we show that eye-centered tuning is virtually nonexistent in PMd when animals are not trained to fixate their eye position.

In our experiments, we did not constrain the eye positions of our animals. They could look freely around the workspace during the task, and they often made several fixations per trial. A confounding factor, though, is that the animals never uniformly sampled the workspace. They each had a unique and consistent eye behavior throughout each session. This idiosyncratic behavior caused our regression analysis to overestimate the amount of TE tuning observed in our neurons. To account for this, we built simulated neurons modeled off of the spiking properties of our actual neurons. These simulated neurons were purely tuned to either a TH or TE reference frame. Neurons built with only TH tuning showed strong TE tuning when regressed against the monkeys' natural eye behavior. However, simulated TE neurons did not show strong TH tuning. Through this analysis, we determined that the TE tuning observed in the planar regressions was a result of the animals' non-uniform gaze behavior combined with the natural TH tuning present in PMd neurons.

Our results suggest that, in natural behavior, eye position information does not influence reach signals in PMd. Many researchers have examined at what stage in the brain eye position information is eliminated from reach command signals. Some studies indicate the transformation is complete prior to PMd (Cisek & Kalaska, 2002; Graziano & Gross, 1998) while others indicate that it must be completed after PMd (Batista et al., 2007; Boussaoud et al., 1998; Pesaran et al., 2006). These studies differ in their results about eye position information, and they also have a distinct difference in the methods used to study reference frames in PMd. The studies that found eye position effects in PMd trained their animals to fixate their eye position throughout the delay period. On the other hand, the studies that saw weak eye position effects did not train the animals to control their eye position. Our study did not train animals to fixate, and we did not see tuning to eye-centered reference frames. Thus, it may be that PMd neurons

flexibly use reference frames based on the task demands. When eye position is controlled, it becomes pertinent to successful completion of the task, and therefore reward. This indicates that there may actually be many cortical mechanisms of sensorimotor transformations that are created through training and experience and selected according to context.

Studies in humans (Sober & Sabes, 2005) and theoretical work (Deneve, Duhamel, & Pouget, 2007) support this idea of flexible sensorimotor transformations in the brain. Sober and Sabes (Sober & Sabes, 2005) showed that the reference frame in which subjects encode reach goals is flexible and is influenced by task conditions, especially the reliability of the sensory information. As this was a purely behavioral study, it could not establish whether individual neurons change their spatial properties or whether a downstream mechanism simply selects between two different coding schemes utilized by different brain areas. Given these results and those from primate studies, though, it appears that training animals to dissociate their natural arm and eye movement behaviors may alter the normal control circuitry for reaching.

Training animals to reliably perform specialized tasks is one of the most common techniques used to study the brain. As scientists, we often feel that this technique is uncovering the innate function of a brain area, but we need to consider how the training itself affects neural activity. Qi and Constantinidis (Qi & Constantinidis, 2013) discuss this idea of training related changes in prefrontal cortex. After training a cognitive task, prefrontal cortex exhibits higher firing rates with less variability. Additionally, training causes more information to be encoded in populations of prefrontal neurons (Meyers, Qi, & Constantinidis, 2012). Our findings in PMd agree with these results and show that PMd can encode a variety of signals relevant to the task at hand.

6.0 Conclusions and Future Directions

6.1 Summary

The theme of this dissertation is how context influences our behavior, and the neural activity that underpins behavior. Here we report results from two different studies that examined two different types of context: task incentives and task demands. First, we showed that monkeys choke under pressure when performing a task for extremely high potential rewards. Additionally, preliminary analyses show changes in the information contained within the neural population when the animal is working for the highest potential reward. We believe that this is the first intracortical evidence of neural signal changes with choking under pressure. Second, we showed that reference frame selection in premotor cortex may be flexible and context dependent. It appears, by synthesizing our study with other previous work, that PMd can use various reference frames when forming a reach plan. The demands of the task play an important role in which reference frames are used.

6.2 Future Directions

6.2.1 Choking under pressure

In Chapter 3, we introduced an animal model of choking under pressure. This is the first time choking under pressure has been shown in a non-human. By showing the phenomenon in

monkeys, we can now study its neural mechanisms intracortically, which has not been possible prior to this work.

Chapter 4 provides a preliminary analysis of intracortical neural data collected from an animal while performing a choking under pressure task. All of the analyzed data come from primary motor cortex (M1). While M1 executes the behavior associated with choking under pressure, it may not be the best brain area to study the neural mechanisms of choking under pressure. A major part of the brain's reward system resides sub-cortically. Human studies of choking under pressure that use fMRI (Chib et al., 2012; Chib et al., 2014; Lee & Grafton, 2015; Mobbs et al., 2009) have seen changes in activity in the ventral striatum and other midbrain structures due to high rewards. Studying these areas at a more granular level will provide a clearer understanding of how these areas change when the stakes get high and potentially show the mechanisms underlying choking under pressure.

Reward signals in the brain are strongly driven by dopamine (Schultz, 2000). Dopamine neurons encode a reward prediction error that reflects the difference in value between a received reward and a predicted reward at each moment in time (Bayer & Glimcher, 2005; Schultz, 1998; Schultz et al., 1997). The dopamine system plays a role in several diseases, such as Parkinson's disease (Kish, Shannak, & Hornykiewicz, 1988; Lotharius & Brundin, 2002) and schizophrenia (Crow, 1981; Laruelle, Kegeles, & Abi-Dargham, 2003). Patients with schizophrenia and depression have been shown to have abnormal reward prediction error signals (Gradin et al., 2011; Murray et al., 2008), which may be a sign of improper valuation of rewards. Could a similar phenomenon be happening in choking under pressure? Maybe in high stakes situations, we value the reward differently, which causes us to have a different behavior than would be expected. With our animal model of choking under pressure, we can record directly from

dopaminergic neurons to study their responses during a choking under pressure task. Another key question is whether the activity of dopaminergic neurons anticipates whether the monkey is about to choke under pressure. Our decode analysis from Chapter 4 shows that it may be possible to predict choking under pressure from cortical signals. If dopaminergic neurons provide an earlier sign that an individual is about to choke under pressure, we may be able to prevent this from happening with some type of intervention. Studying these questions will help to confirm human fMRI studies that posit a role for the basal ganglia in choking under pressure and allow us to push the field of choking under pressure forward.

6.2.2 Reference frame studies

The work in Chapter 5 indicates that perhaps reference frames can be flexibly used in PMd based on task demands. To test this, we can run an experiment with changing task demands and then look at which reference frames show the strongest tuning. The ideal experiment would have animals perform the same task outlined in Chapter 5, and then learn to fixate their eyes and perform the task with fixation. In the first task, eye position would be recorded but not pertinent to the success of the task. In the second task, eye position would be necessary in order to successfully complete the task. If the requirement to fixate the eyes affects reference frame tuning in PMd, this experiment will highlight the differences between the non-fixation and fixation tasks.

Can PMd change its sensitivity to eye position after being trained to fixate? Once the animals are trained to fixate, we can interleave both trial types to study how flexible PMd is when it comes to reference frame tuning and eye position sensitivity. We may see that once the animals know how to fixate, the eye position sensitivity remains present throughout all tasks. Or,

we may see that eye position sensitivity decreases or disappears when it is not required by the task. We can study this by titrating how much time is between the animals seeing the fixation and non-fixation tasks. We can interleave the task types so that only one or two trials separate the tasks, or we can separate them into blocks or sessions. This will allow us to explore the timing and flexibility of tuning changes in PMd. If we can show that neural computations as seemingly hard-wired as how we reach out to visible objects are malleable simply by changing task instructions, we may be able to use that knowledge to provide better rehabilitation techniques for patients recovering from stroke or other brain injuries.

A key feature of behavioral neurophysiology research is animal training. Many studies overtrain their animals on a specific task prior to collecting any data. Our work points to this training being a key factor in some of the effects seen in neural tuning properties. We can study the effects of training from start to finish by training a naïve animal to perform the non-fixation task. Then, we can train the same animal, with the same electrode array implanted, to perform the fixation task. Once training is complete, we can have the animal perform the non-fixation task, and then look for differences and similarities in the pre- and post-training data. Any differences seen in the neural data will likely be due to the training of the fixation task. Before drawing that conclusion, though, we will need to account for changes in the quality of signals from the electrode array and any behavioral changes in eye behavior or reaching behavior.

Our current reference frame study focused on single neuron analyses. However, we collected our neural data from a 96-channel electrode array, which provided us with a population of neurons. We can study these data at the population level to investigate any changes in activity that may not be evident at the single neuron level. One way to do this is to apply dimensionality reduction techniques to the data in order to visual features of the population. These techniques

conceptualize the activity of many neurons as a point (called a state) in a high-dimensional state space, where each dimension is defined by the firing rate of one neuron in the population. The neural population may enter different states under different task conditions. A key feature of dimensionality reduction is that the states are organized into clusters due to dependencies between the neurons, and are not homogeneously distributed throughout firing rate space. These techniques seek to reveal the lower-dimensional subspace within which neural activity resides. We can look for distinct clusters in our data representing the fixation and non-fixation tasks. It could be that training the animals to fixate causes PMd to fire in a different part of the firing rate space, and we would be able to visualize this with dimensionality reduction techniques applied to our array recording data.

A key application of these reference frame studies is in the calibration of brain-computer interface (BCI) systems. If neural tuning depends on where the BCI user is looking some of the time, but not at other times, then this might present a challenge to how BCI systems currently function. By understanding context-dependent tuning in motor areas, we can form a more accurate picture of the neural control of movements, and use this to improve future clinical technologies.

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