# When did the horse cross the road?

*Sequential effects in the go/no-go variable foreperiod paradigm*

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## Abstract

Timing is important to everyday functioning; performing the right action at the wrong time can be just as ineffective as performing the wrong action entirely. 'Inhibition' has been proposed to allow us to perform the correct response at the right time, and to stop that response completely if necessary. This thesis focuses on one theory linking inhibition and temporal expectancy, the 'trace-conditioning model' proposed by Los and van den Heuvel (2001). The trace-conditioning model posits that imperative time points have associated traces which are reinforced/extinguished by inhibition based on events at that time point. Traces are proposed to explain the asymmetrical sequential foreperiod effect; a prior trial with a long foreperiod slows current short imperative reaction times, while a short imperative in the prior trial does not affect current trial long imperative reaction times. To further elaborate on how traces are controlled, I have performed three behavioural and one transcranial magnetic stimulation (TMS) experiment utilising varieties of the go/no-go variable foreperiod experiment as described by Los (2013). Each behavioural experiment manipulated the foreknowledge of an upcoming event, while the TMS experiment repeated Los' study with the addition of a single TMS pulse 100ms after the short imperative time. By manipulating the foreknowledge of an upcoming event, I examined how trace-conditioning may interact with explicit information provided by cueing. TMS was applied to determine if corticospinal excitability was affected by prior trial dynamics.

The behavioural experiments suggest that, while prior trial response effects were attenuated given response foreknowledge, temporal trace effects were not attenuated unless both response and temporal foreknowledge were provided. TMS results indicate prior trial timing, mediated by the current trial type, affected motor cortex excitability. I argue that these findings indicate a separation between response and temporal traces.

**Declaration**

I declare that this work, either in part or in whole, has not been submitted elsewhere as either part of a degree or for publication, in any other university or institution. The sources of information for this work were wholly from my own research, except where referenced. The writing of this thesis was independent and is free from plagiarism. This research was approved by the Macquarie University Ethics Committee (protocol numbers: 5201200035, 5201400585)

Jordan Wehrman

**Acknowledgements**

Many people made this thesis possible. My supervisor, Dr. Paul Sowman, deserves the first mention. I initially came to study under him with only a vague idea of what I wanted to do. He fostered my interests and directed me towards the study of inhibition. Initially, I was not super enthused, it seemed like there had been so much research already done on the topic; I did not know how I could contribute to the field. However, he put up with my constant stream of (mostly bad) ideas until I came across the paper by Dr. Sander Los, entitled 'The role of response inhibition in temporal preparation: Evidence from a go/no-go task.' Though not directly related to his own research interests, Dr. Sowman allowed me to pursue my interest in Dr. Los' paper and helped me make sense of a topic not entirely familiar to me. As one further acknowledgement, Dr. Sowman has been an amazing editor of my rambling writing, calling me out on my sometimes far-fetched ideas, and equally far-fetched grammar.

Further acknowledge is owed to Dr. Sowman's other students. Andrew Etchell was amazing at listening to my ideas on this topic, and giving me insight from his own studies. Further, he constantly provided advice on how to approach findings, and deal with the weird world of academics. He was also always good for a laugh, which is one of the most important factors in finishing a thesis. Leidy Castro-Meneses, having just finished her thesis, fought through brain-numbness to assist me with transcranial magnetic stimulation. Without her, I would still be floundering, looking for someone to help me ensure that I was not stimulating people unconscious. Finally, Margaret Ryan was an excellent resource in helping me organise any administrative issue I encountered. If it weren't for Margaret I would not have been able to get ethics approval for my studies, let alone participants.

Broadly, I would like to thank Macquarie University, and specifically the members of the cognitive sciences department. This department provided a strong support network and fostered my learning. I could always find someone to have a conversation with, ask for help (often) or get a much-needed participant.

Lastly I would like to thank my lovely fiancé, Clara Chung. She put up with me sitting up to all hours paying more attention to my thesis than her. She gave me time to complete this project and supported me with delicious treats as needed. Without her patience and generosity, I would not have started a thesis, let alone finished one. She is a constant source of both strength and inspiration.

## 1. Introduction

Timing is everything. Once, while I was walking and reading, I almost wandered face first into an oncoming bus. Being able to stop my movement before my foot landed saved my life. Not being the smartest person, my first thought was not 'maybe I should stop reading while I walk,' but was 'wow, stopping movement is really important.' What exactly is timing? Simply put, it is doing the right thing at the right time. It is waiting until the right moment to perform an action, and stopping that action when it is no longer appropriate. Thus, 'timing' and 'stopping' may be intrinsically linked. Inhibition, the restraining, hindering or arresting of an action, impulse, or thought (Aron, Robbins, & Poldrack, 2004; McPhee & Read, 2010) has been proposed as the cognitive function underlying appropriately timed action initiation and cessation. Inhibition allows us to wait until we get off a crowded train before we start to dance, and stop boogying when people start laughing.[1]

This thesis is concerned with the role of inhibition in response timing forwarded by Los and van den Heuvel (2001). Termed the 'trace-conditioning model,' this theory is proposed to explain sequential asymmetries in the variable foreperiod (VF) paradigm. In its simplest form, a VF experiment is a simple reaction time (SRT) task that requires a response at one of two (or more) imperative time points. A common finding of these experiments is an asymmetry of sequential effects; reaction times (RTs) depend on whether the imperative in the prior trial was earlier or later than in the current trial. While short foreperiod RTs may be slower if preceded by a long foreperiod, long foreperiod RTs are not affected by prior shorter foreperiods. The trace-conditioning model explains this by assigning a 'trace' to each time point, a subjective link between when a trial is initiated and when an imperative (i.e. the signal to respond) is expected to occur. As a trial continues, and possible imperative times are passed, their associated traces are weakened. Inhibition is proposed to control trace reduction; if a time point is passively not responded to as it is skipped, or actively not responded to (a no-go trial) as per Los (2013), the trace is 'extinguished', resulting in slower RTs in future trials at that time point. If a time point is not passed by the time an imperative occurs, there is no effect on the associated trace, explaining why two consecutive long trials, and a long preceded by a short trial have

[1] It may be painfully obvious I have experienced this many times.

approximately equal RTs.[2] This thesis further explores the temporal and response dynamics of trace conditioning.

I will begin by discussing the VF paradigm and the role trace-conditioning is proposed to play in this. I will then discuss response inhibition, the stopping of a prepared or planned action (Nigg, 2000).[3] This will facilitate an understanding of why inhibition is important, how it is studied, and how it may affect traces in the trace-conditioning model.

## 1.1 Variable Foreperiods and Sequential Effects

A VF experiment is a SRT experiment in which the response imperative may occur at one of two (or more) possible imperative times. This type of experiment has a long history in the cognitive sciences (e.g. Bertelson, 1967; Drazin, 1961; Hick and Welford, 1956; Woodrow, 1914). One common finding in these types of experiments is that, the longer the foreperiod is, the quicker the response to an imperative is (long foreperiods elicit faster responses than short foreperiods). This is an intuitive result based on the perceived probability of the imperative occurring; given 100 possible foreperiods, at time 99 there is only one possible imperative time left therefore the probability of an imperative occurring at the next time point is 100%. In other words, as time passes, the probability of the imperative appearing increases (e.g. Hick and Welford, 1956; Li, Krystal, and Mathalon, 2005; Nobre, Correa, and Coull, 2007; Stuss et al., 2005; Woodrow, 1914). This increase in conditional probability is termed the 'hazard function.' However, a further predictor of RTs is the length of the prior trial foreperiod in relation to the current trial foreperiod. This is termed the asymmetrical sequential foreperiod effect. If a long foreperiod precedes a short foreperiod, then the response to an imperative at the short time point is slower than if the prior trial imperative occurred at the short foreperiod. However, the reverse is not true. A short foreperiod in the prior trial has little if any effect on the RT to a long foreperiod imperative; RTs are equally fast whether the preceding trial contained a long or short foreperiod. This asymmetry has been demonstrated repeatedly (e.g.

[2] This is not exactly true, a long-long combination tends to be slightly faster, but this is discussed below. Also, the sequential effect may be attenuated when using a non-aging distribution (Capizzi, Correa, Wojtowicz, & Rafal, 2015; Näätänen, 1971).

[3] Though it is debatable whether motor inhibition actually represents a distinct category from motor reprogramming (Boecker, Gauggel, & Drueke, 2013) or response selection (Mostofsky & Simmonds, 2008); some evidence has shown dissociable improvements in inhibition but not selection following medication, possibly indicating different underlying processes (Scheres et al., 2003).

Drazin, 1961; Karlin, 1959; Steinborn, Rolke, Bratzke, and Ulrich, 2008; Vallesi and Shallice, 2007; Woodrow, 1914; Zahn, Rosenthal, and Shakow, 1963; for review, see Los, 2010).

The 'trace-condition model' by Los and van den Heuvel (2001) proposes an explanation for this asymmetry. As time passes and critical moments (points of possible imperative) are skipped, their associated traces are weakened, resulting in slower RTs to imperatives at those times in subsequent trials. Trace weakening is caused by the inhibition of a prepotent response (a response tendency) at the time of a possible imperative. For example, if, in trial n-1 ($T_{n-1}$) the response is required at the long imperative time, the short imperative time will have been passed and inhibition applied at that point, weakening the trace, decreasing its subjective likelihood in trial n ($T_n$). RTs will therefore be slower if the imperative occurs at that time. However, the reverse is not true; if $T_{n-1}$ is short and $T_n$ is long, RTs will not be affected as the long imperative time was not passed in $T_{n-1}$ before an imperative was given and therefore was not suppressed. This implies that, once an imperative has occurred, remaining traces (i.e. traces with longer foreperiods) are no longer affected by their critical moments being passed. In terms of classical conditioning, a 'trace,' the time from the conditioned stimulus (the initiation signal for the trial) to the unconditioned stimulus (the response imperative), can be 'conditioned,' either reinforced or extinguished, by pairing or unpairing with the delay time before the response imperative. Ring a bell a thousand times when a dog sees food, and the dog will salivate to the sound of the bell, but the more times the bell is unpaired with food, the less effect the sound will have.

An alternative to trace-conditioning is the dual-process model. Vallesi and Shallice (2007) found that the asymmetrical sequential foreperiod effect and the hazard function develop at different times throughout childhood. This, they argued, was counter to trace-conditioning (which they suggested implied a single developmental trajectory, i.e. inhibitory control). They proposed that a dual-process underlies sequential foreperiod effects. The first process reflects 'refractory costs' (see Welford (1952)); the longer the $T_{n-1}$ foreperiod, the slower the RTs in $T_n$. The second process attenuates these adverse effects; the longer the $T_n$ foreperiod, the faster an imperative is reacted to (i.e. the hazard function). Recent imaging studies (Vallesi, McIntosh, Shallice, & Stuss, 2009) and SRT versus CRT task comparisons (Vallesi, Lozano, & Correa, 2013) are purported to support the dual-process model.

Before comparing these models, and further discussing the studies in this thesis, it is important that we grasp what inhibition is, and how it is studied. Without this understanding,

it may be difficult to see how inhibition could mediate the dynamic interaction between $T_n$ and $T_{n-1}$ timing, and the speed with which we respond to stimuli.

## 1.2 Why Inhibition?

### *1.2.1 The lizard and the foot*

Though 'not doing' may sound simple, inhibition is implicated in several complex cognitive processes (Clark, 1996; Dillon & Pizzagalli, 2007), for example adaptive control (Folstein & Van Petten, 2008; Goldman-Rakic, 1996), context-mandated default behaviour suppression (Aron, 2007), and goal-oriented action (Zanto & Gazzaley, 2009). Inhibitory control measures may also generalise to real life situations (see Wessel and Aron, 2014), for example in sports (Gray, 2009; Gutierrez-Davila, Rojas, Caletti, Antonio, & Navarro, 2013; Nakamoto & Shiro, 2007), impact prediction (Marinovic, Reid, Plooy, Riek, & Tresillian, 2011; Marinovic, Reid, Plooy, Riek, & Treslian, 2010), and driver safety (e.g. Cascio et al., 2015). It is intrinsic to our successful functioning; several disorders have been linked to inhibitory abnormalities. These include **Parkinson's disease** (Berardelli, Rona, Inghilleri, & Manfredi, 1996; Kehagia et al., 2014), **Tourette syndrome** (Stern, Blair, & Peterson, 2008; Stinear, Coxon, & Byblow, 2009; Thibault, O'Connor, Stip, & Lavoie, 2009), **obsessive compulsive disorder** (Chamberlain & Sahakian, 2007; Penades et al., 2007; van Velzen, Vriend, de Wit, & van den Heuvel, 2014), **cocaine addiction/use** (Fillmore & Rush, 2002; Prisciandaro et al., 2014; Simon, Mendez, & Setlow, 2007),[4] **eating disorders** (Lock, Garret, Beenhakker, & Reiss, 2011), **hoarding** (Tolin, Witt, & Stevens, 2014), **attention deficit hyperactivity disorder** (Booth et al., 2005; Vaidya et al., 1998), **gaming addiction** (Ding et al., 2014) **and schizophrenia** (Enticott, Ogloff, & Bradshaw, 2008; Hughes, Fulham, Johnston, & Michie, 2012).[5] In short; inhibition is central to everyday life.

My friend told me an excellent story exemplifying what is often cited to occur in the brain during response inhibition. He was walking in his kitchen at night when he glimpsed a movement underfoot. Immediately he stopped his footfall, placing it slightly to the side of a giant lizard (goanna). He had already initiated movement however, given a signal indicating his step was no longer appropriate, a race occurred within his brain between the inhibition and

[4] This may be related to status of drug use (see Bell, Foxe, Ross, and Garavan, 2014; Morie et al., 2014)
[5] For review see Chambers, Garavan, and Bellgrove (2009) and Nigg (2000). More recently see Wright, Lipszyc, Dupuis, and Thayapararajah (2014) for a meta-analysis specifically regarding go-no/go tasks (below).

completion of this action. Thankfully for him (and the lizard), inhibition won. This 'race model' was formalized by Logan and Cowan (1984) as two independent processes,[6] stop and go, which race for completion. The go process has a head start but the stop process is faster. Which process finishes first depends upon the lag time at which the inhibitory 'lizard' appears. This race is often explored using the stop-signal task (SST) (Logan, Cowan, & Davis, 1984) in which an individual is directed to begin an action when a signal appears, and if a subsequent signal occurs, to stop this already-initiated action before behavioural actualization. The same effect may be demonstrated using a stop signal before the response imperative; given a predictable 'go' time, people require roughly 200ms to successfully inhibit action, indicating the existence of a prepotent response (tendency to respond) prior to performance (see Marinovic et al., 2011; Marinovic et al., 2010).

A further paradigm used to investigate response inhibition, and which is employed in this thesis, is the go/no-go (GNG) task (Donders, 1969).[7] Classically, this task requires a response if one signal appears and none if another does. Several reviews of inhibition tasks are available (e.g. Aron, 2011; Chambers et al., 2009; Simmonds, Pekar, and Mostofsky, 2008; Swick, Ashley, and Turken, 2011). The GNG is intrinsically different to the SST. In the SST, responses are initiated and inhibited as required, while the GNG relies on inhibition of a prepotent response if the imperative does not match that of the required 'go' signal (Swick et al., 2011). Further, while the GNG utilizes direct mapping of stimulus to response, such that one stimuli[8] indicates action and another indicates inaction, in the classic SST, event timing indicates meaning (Verbruggen & Logan, 2008b). Thus, the SST strategy for effective responding can be thought of as 'go unless' while for the GNG, a subject could alternatively adopt a 'do not go unless' strategy.

Though the distinction between 'go unless' and 'do not go unless' may seem trivial, it has major ramifications for any explanation involving inhibition and the GNG task (i.e. Los (2013) and the experiments in this thesis). In the GNG task, if the subject is simply waiting for a signal to occur, then no response inhibition is required. Without a prepotent response, any sequential effects in a GNG task would not be related to inhibition but would rather be some

[6] These may interact in later stages of processing (Boucher, Palmeri, Logan, & Schall, 2007).

[7] Though several more exist, for example flanker tasks (e.g. Kawai, Kubo-Kawai, Kubo, Terazawa, and Masataka (2012)), anti-saccade tasks (e.g. Hallett (1978), Munoz and Everling (2004)) masked priming tasks (e.g. Schlaghecken, Bowman, and Eimer (2006)), Stroop tasks (e.g. Dimoska-Di Marco, McDonald, Kelly, Tate, and Johnstone (2011)) and a combination of these tasks (e.g. Boy, Husain, and Sumner (2010)).

[8] Signals may vary, for example categorical (Smith, Jamadar, Provost, & Michie, 2013), letters (Smith et al., 2013), or matching versus mismatching stimuli (Kropotov, Ponomarev, Hollup, & Mueller, 2011).

function of other factors, for example the timing of the imperative or whether or not an action was performed. In the following section, we will examine one argument against the contention that there is response prepotency in the GNG task, and utilise this to facilitate discussion of how response inhibition does not just reactively stop a response, but may also allow the holding back of a response until appropriate.

### *1.2.2 – Here's the problem with prepotency…*

Given foreknowledge of an action and when it will occur (e.g. one second after a fixation cross), if a startling event occurs at the imperative,[9] RTs shorten beyond what is considered possible otherwise. This has been termed the 'start-react' effect, and is often cited as proof of a prepotent response (for review, see Carlsen, Maslovat, and Franks, 2012). If movement is only decided upon and initiated at the onset of a response imperative, then a startling event would not induce faster RTs, as there is no movement for the start-react to initiate. Even if the imperative is subsequently removed, or the timing of the imperative is uncertain, the startling event can still elicit release of the action (Crossman, Carlsen, Chua, & Franks, 2006; MacKinnon et al., 2013; Valls-Solé, Rothwell, Goulart, Cossu, & Munoz, 1999; Valls-Solé et al., 1995). If responses are prepotent in the GNG, a similar early release should be present if combined with a start-react. However Carlsen et al. (2008) and Washington and Blumenthal (2015) found that a startling acoustic event in the GNG did not reduce RTs, though it did increase false-alarms.

These findings were argued to indicate a lack of GNG response prepotency.[10] Carlsen, Maslovat, et al. (2012), in their review of the start-react motor preprograming assessment, argued that Carlsen et al's (2008) findings indicated a strategic balancing of RT and correctness, however this does not necessitate an absence of prepotency. One may balance the ability to respond quickly and accurately while still having a response at the ready; being ready

[9] Or before the imperative, given a predictable imperative time (MacKinnon, Allen, Shiratori, & Rogers, 2013).

[10] The increase of false-alarms was argued to not be relevant as errors were similar to the number of false alarms when no-go signals were rare. However, lower no-go probability induce higher false alarm rates (e.g. Low and Miller (1999)), so why this argues against preprograming is unclear. Further, at least in Carlsen et al. (2008), each participant only underwent one probability distribution of go to no-go events (20-80, 50-50, and 80-20), of which there were 10 startle responses out of 80 trials, half of which were go and half of which were no-go. This represents a separate startle distribution of go to no-go trials in two of the conditions, which may be disassociated from the underlying distribution of the normal trials (See Steinborn, Rolke, Bratzke, and Ulrich (2009) for an example of how separate cuing may interfere with intertrial information continuity) making conclusions regarding false alarms rates difficult.

to respond does not mean you are constantly on the verge of action.[11] Further, CRT tasks involving pointing (which allow trajectory tracking) demonstrate that incorrect initial movements may be initiated by a startling acoustic stimulus, more so than in non-startling trials, indicating a prepotent response in CRT tasks (Blinch, Franks, Carpenter, & Chua, 2015) (of which a GNG may be a special case if 'not going' is considered a response trajectory, see Gomez, Ratcliff, and Perea, 2007).

Proactive inhibition could be the mechanism that restrains a prepotent action in the presence of a startling stimulus. Aron (2011) discussed a reactive-proactive divide in inhibition; the former actively stops us once an action has been initiated, while the latter allows us to withhold a response until it is appropriate.[12] For example, you reactively inhibit movement if about to step on a lizard. However, if you wish to step on a lizard you might withhold your step until the lizard moves into the optimal position. Proactive inhibition allows the maintenance of readiness while preventing premature release of the prepotent response (Correa, Trivino, Perez-Duenas, Acosta, & Lupianez, 2010; Los, 1996; Narayanan, Horst, & Laubach, 2006). If a start-react event is a jump in excitability (or momentary release from inhibition (Carlsen, Almeida, et al., 2012; Carlsen, Maslovat, et al., 2012)) then in the SRT, where proactive inhibition holds us just below threshold, a movement may be initiated. Conversely, in the GNG task, the action is too far below threshold for movement to be initiated by the startle. The reduced response readiness in the GNG is likely due to the combination of temporal and response uncertainty, reducing net excitability and increasing RTs.[13] This is supported by MacKinnon et al. (2013) who found the start-react occurs more often closer to a known imperative time point, in line with progressive inhibition release over time, as shown by TMS studies (below). Perhaps the start-react response release occurs when a response is already 'close.'

Evidence for inhibition's role in temporal response control has been shown in several studies. Transcranial magnetic stimulation (TMS) which allows for the examination of corticospinal excitability (CSE) while performing cognitive tasks (Hallett, 2000, 2007; Reis et

[11] Interestingly, it may also be the case that a 'stop' is preprogrammed; Carlsen, Almeida, and Franks (2012) found that a startle response may not only improve RTs but also SSRTs. Alternatively it could be the case that again the salience of the sound improves the person's ability to react.

[12] Proactive inhibition may be applied on a trial by trial basis (Chikazoe et al., 2009), in a set (Verbruggen & Logan, 2009), or at a higher, strategic level (Bogacz, Wagenmakers, Forstmann, & Nieuwenhuis, 2010),

[13] We do not entirely cancel the response however. Instead, a 'brake' appears to be applied, allowing action cessation if required (i.e. the active braking hypothesis (Jahfari, Stinear, Claffey, Verbruggen, & Aron, 2010), see also Aron, Robbins, and Poldrack (2014)).

al., 2008), is one source of such evidence.[14] Utilising this method of investigation, suppression of cortico-motor excitability (as measured by motor evoked potentials; MEP) has been shown during response preparation (e.g. Duque et al., 2010; Kinoshita, Yahagi, and Kasai, 2007; Van Elswijk, Kleine, Overeem, and Stegeman, 2007). This is proposed to result from inhibition rather than decreased excitation (Sinclair & Hammond, 2008). Furthermore, motor excitability tends to increase as an expected imperative approaches (Bolton, Vesia, Lakhani, Staines, & McIlroy, 2014). While a warning signal may induce motor cortex activation, the prepotent response is withheld via inhibition (Hasbroucq, Kaneko, Akamatsu, & Possamaï, 1997; Sinclair & Hammond, 2009). CSE may also be mediated by the probability of response, important in the GNG paradigm (Jahfari et al., 2010; Van Elswijk et al., 2007). Generally, at the time of intended motor movement if a no-go signal is presented, motor excitability is decreased, and short interval cortico-spinal inhibition (sICI, a measure of GABAergic inhibition) is increased (Coxon, Stinear, & Byblow, 2006; Hoshiyama et al., 1997; Leocani, Cohen, Wassermann, Ikoma, & Hallett, 2000; Sohn, Wiltz, & Hallett, 2002; van den Wildenberg et al., 2010).

### 1.3 Inhibition and the Variable Foreperiod

Given that CSE increases as an expected imperative approaches, and that response suppression may be required in order to withhold response until cued, it seems reasonable that inhibition may play some role in mediating our expectations of upcoming events. This mediation device is formulated as a ‘trace’ in the trace-conditioning model of Los and van den Heuvel (2001). Motor effects on information/response processes (Hommel, 2004, 2009; Prinz, 1990; Shin, Proctor, & Capaldi, 2010; Steinhauser & Hübner, 2006) further supports the link between motor (in)action and trace-conditioning (see Los (2013) for discussion). Furthermore, in terms of conditioning, reinforcement and extinction has been repeatedly linked to inhibition (e.g. Jordan, Todd, Bucci, and Leaton, 2015; Kirkwood, 2015; Magal and Mintz, 2014; Quirk, Garcia, and González-Lima, 2006; VanElzakker, Dahlgren, Davis, Stacey, and Shin, 2014). Let us now return to the discussion of inhibition in the VF paradigm.

[14] Other sources of evidence also exist, for example spinal reflexes (e.g. Duque, Lew, Mazzocchio, Olivier, and Ivry (2010) and Hasbroucq et al. (1999)). TMS is focused on here as this relates to the current research.

If inhibition is the cause of trace weakening, then inhibiting a response (i.e. actively withholding a response to a no-go signal) should also slow $T_n$ RTs in consecutive long-long or short-short trials.[15] Los (2013) showed this to be the case. By combining GNG and VF tasks, he demonstrating not only the effects of skipping imperatives, but also 'actively' inhibiting responses. A no-go in $T_{n-1}$ increased $T_n$ RTs when the imperatives in $T_{n-1}$ and $T_n$ were at the same times. However a no-go at the short imperative time had no effect on a subsequent long imperative time. Similarly, there was no effect beyond that associated with passing the short imperative if $T_{n-1}$ presented a no-go after a long foreperiod and the $T_n$ imperative was at the short time. Therefore response inhibition appears temporally linked to a single time point's trace. Importantly, RT increases in Los (2013) were not a function of simply not responding; when a relax (i.e. definite no-go) trial was presented, the lowered state of expectation (relaxed rather than ready) had reduced impact on the passed (short) foreperiod. This may indicate that the effects of $T_{n-1}$ are mediated by the state of readiness; the closer the prepotent response is to threshold, the greater the effect on the subsequent trial.[16] Once a response has been performed, it thus makes sense that critical moments not yet passed are unaffected. Figure 1.1 below exemplifies the effects on short-imperative RTs in trial two given different variants of the initial trial, as per Los (2013).

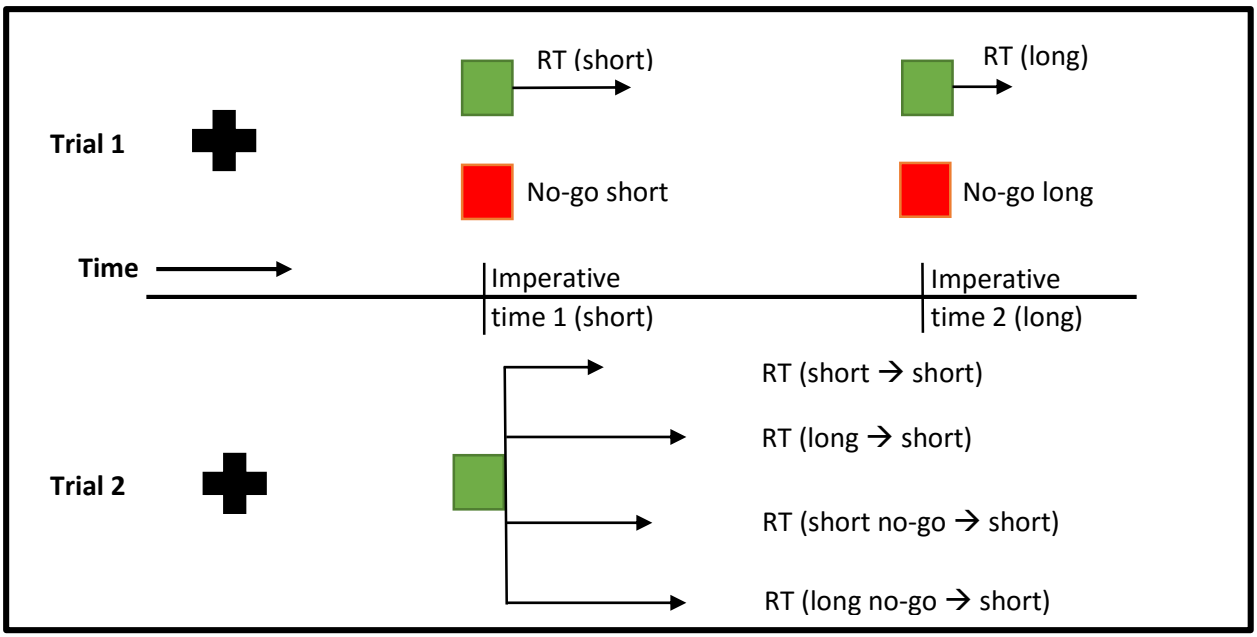


*Figure 1.1: Graphical representation of RTs in trial two given a short go imperative with various possible trial one events. The RT for a long foreperiod in the prior trial is quicker than all four possibilities in trial two. RTs improve slightly in trial one given the short-short trial combination, while a long go or long no-go equally slow RTs. Given a short no-go in trial one, the RTs for trial two are slowed, but not as much as if the time period is passed (i.e. a long prior trial).*

[15] It would be interesting to apply this model to task switching; perhaps switching tasks may equally inhibited the trace associated with a specific foreperiod.

[16] Manipulation of 'go' probability may further elucidate the dynamics of this interaction.

As discussed in Los (2013), the dual-process model has trouble accounting for the effects of a no-go $T_{n-1}$ on $T_n$ RTs. Under the dual-process model, a go imperative at the short critical moment in $T_{n-1}$ should result in slower RTs in $T_n$ compared to a NG at the short critical moment in $T_{n-1}$ due to the effects of the response on motor readiness (i.e. refractory costs). Alternatively, if we conceptualise response inhibition as another response trajectory (as per Gomez et al., 2007), or as effortful (Hester & Garavan, 2005), then a NG at the long critical moment in $T_{n-1}$ should result in slower RTs if given an imperative at the short critical moment in $T_n$. However, this also did not appear to be the case; response inhibition effects on $T_n$ RTs appear temporally-specific.

Capizzi et al. (2015) sought to explain why RTs were shorter in ($T_{n-1}$ to $T_n$) long-long versus short-short trial combinations; a result not predicted by the trace conditioning model.[17] To do this, they employed a non-aging foreperiod distribution (in which the conditional probability of response does not increase with time), including catch trials in which no imperative was given. When a catch trial was presented in $T_{n-1}$, long imperative RTs increased in the following trial. They proposed that this was due to catch trials impairing re-preparation, or re-orientation at the ready signal. Effectively, catch trials altered the conditional probability of an imperative occurring at the long time point.[18] In a recent reformulation of the trace-condition model, Los, Kruijne, and Meeter (2014) proposed a multiple trace theory in which, for each critical moment, memory traces of prior events (including those prior to $T_{n-1}$) at that time point are used to construct the individual's preparatory state, and these memories decay towards an asymptote over time. These traces are constructed not only by response history, but also by prior levels of both excitation and proactive inhibition. Further, trials in which a time point is not passed do not affect the current readiness construct for later $T_n$ foreperiods. In other words, a short foreperiod, no matter how far back, does not affect the long trace, and so, does not affect the readiness to respond at the long foreperiod in $T_n$. This, they found, better accounted for foreperiod effects and the faster long-long trial combination RTs compared to short-short trial RTs,[19] however this theory is untested empirically.

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[17] Though it is possible that absolute temporal preparation (i.e. long foreperiods allow faster responding than short) plays a role here, above that of repeating an event.

[18] Hence why this is called non-aging; as time passes the conditional probability of an imperative being given does not increase, i.e. does not age (see Gottsdanker, Perkins, and Aftab (1986), Granjon, Requin, Durup, and Reynard (1973), Näätänen (1971) and Rowell and Siegrist (1998))

[19] This may imply that RTs in short-long trial combinations should be closer to a 'true' long RT, while a no-go at the long $T_{n-1}$ should be a reduction of reaction time, and long-long may present a facilitation effect. For the short trials, it may be that it is impossible to access 'true' RTs.

## 1.4 Current Study Approach

In this study I propose that proactive inhibition levels are dynamically set based on implicit and explicit information from $T_{n-1}$ and $T_n$, such that RTs are minimized while the expected ability to stop is optimized. Two experiments were used to investigate this inhibition setting.

In the first study, I performed three varieties of the variable foreperiod GNG task by Los (2013). In the first instance a definite-go cue was added to the Los (2013) structure to investigate how a definite-go may affect following trials, and be affected by $T_{n-1}$. This concept has been previously published by Berchicci, Lucci, Spinelli, and Di Russo (2015),[20] however they did not examine sequential trial effects, and also provided a countdown which allowed precise knowledge of the imperative onset, as opposed to relying on a (possibly imprecise) internal clock. I expect response certainty at a given critical moment to alter RTs such that a long definite-go will be responded to fastest, while a short definite-go and long uncertain-go will have roughly equal RTs (both have equal probability of response, the former driven by temporal uncertainty, the latter driven by response uncertainty), and a short uncertain-go will have the slowest RTs. Within this general structure, prior definite-go trials may have a different effect on subsequent trial RTs compared to uncertain-go trials. This is because (as demonstrated by relax trials in Los, 2013) effects on the trace may relate to the level of readiness to respond; a definite-go trial is basically a SRT task and therefore the prepotent response is close to threshold. Perhaps $T_{n-1}$ response release will not have as strong an effect on the trace as when response is initially further from threshold.[21] When $T_n$ is a definite-go, I expect the effect of $T_{n-1}$ to be attenuated due to foreknowledge of the upcoming response; $T_n$ information may override the implicit information gained from $T_{n-1}$ (see Adam and Koch, 2009). Uncertain-go RTs should follow the pattern described by Los (2013).

In experiment 1.2 a blue fixation cross provided temporal response certainty, without response type certainty. In experiment 1.1, if there was a separable effect of response and timing when given response-specific information, it may be possible to similarly override temporal information by providing temporal certainty. Experiment 1.2 may demonstrate such an effect. In the third variation (experiment 1.3) a green fixation provided both temporal and

[20] This was also published after initiating these experiments.

[21] In a definite go condition, going is 'meh,' while in a maybe go condition, given a go, well, that's just down right exciting! Alternatively, it may be that passing an imperative moment has more effect due to the prepotent response being closer to threshold and therefore requiring greater inhibition to suppress.

response certainty. In this case, I expect no sequential RT effects as foreknowledge is perfect and so overrides $T_{n-1}$ evidence.[22]

Generally, I argue for optimization of both RTs and stopping ability, which may be reflective of inhibition and excitation balancing (figure 1.2 below). Further, as information accrues (e.g. critical moments pass, or definite foreknowledge is given), levels of excitation and inhibition adjust. Foreknowledge of the upcoming imperative (whether temporal, response, or both) may also affect the trace strength and thus expectations regarding the upcoming imperative timing/type.

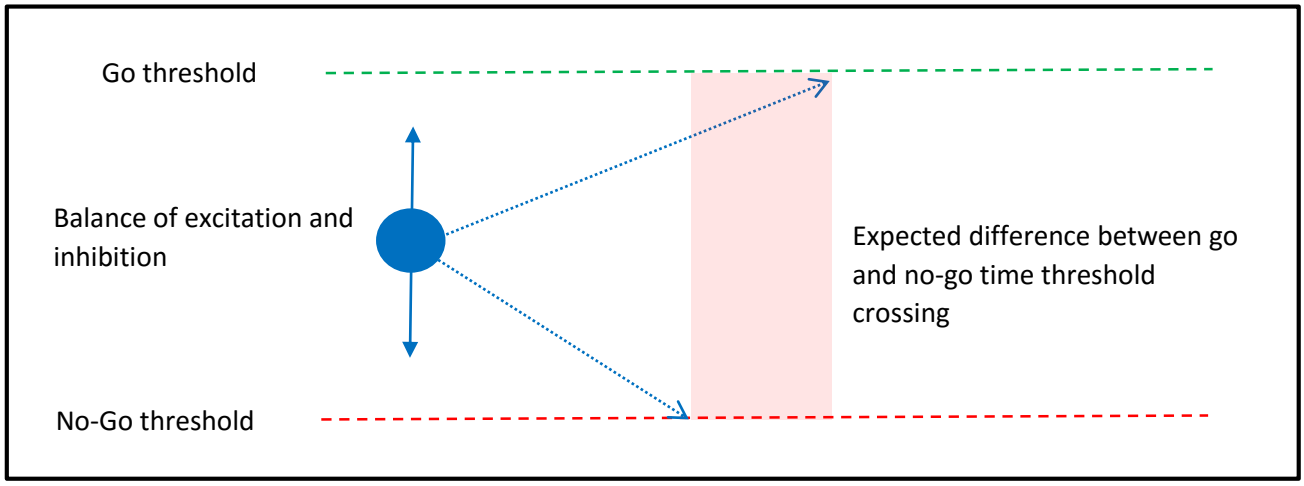


*Figure 1.2: Graphical representation of balance performed on the initial setting, based on prior and current information and beliefs. This level is set dynamically such that the time to reach the go threshold is minimized, while maintaining as little as possible difference between the go and no-go threshold crossing points (the shaded area). This optimization of the difference between the go and no-go threshold crossing times interacts with these beliefs, such that optimization is not merely a function of expected crossing times (otherwise we would have a stable optimization level at any given time). Further, note, the level of excitement is dynamically balanced across time, not just set and left at the beginning of a trial.*

In the second experiment, Los' (2013) paradigm was repeated with the addition of TMS in order to examine sequential trial effects on CSE. Single-pulse TMS was applied 100ms after the short imperative time (whether or not an imperative was present). The short imperative time was chosen as it allowed investigation of the effect go or no-go imperatives, as well as no imperative (when the short imperative time is passed) have on CSE. In the current study I chose to apply TMS 100ms after the imperative time, anticipating that this would reveal $T_n$ effects without totally overriding $T_{n-1}$ effects. Thus, the relationship between $T_n$ and $T_{n-1}$ may be examined in terms of CSE. 100ms post-imperative has been found to demonstrate signal-specific CSE changes; Hoshiyama et al. (1997) found that a no-go signal reduced MEPs as

[22] The results from an additional experiment are presented in appendix one. This variation presented a definite go cue as per the first behavioural experiment, but in the uncertain condition (the white fixation) a no-go only occurred at one time point.

early as 100-250ms after presentation. However CSE is more commonly found to be significantly reduced only after 180ms has passed following the stop signal (e.g. van den Wildenberg et al., 2009).

In this experiment, I will firstly demonstrate how including $T_{n-1}$ effects in MEP analysis may better stratify CSE effects in sequential tasks. However some variability is still expected, for example due to external events affecting an intrinsically noisy temporal expectation mechanism (see Schuur, 2012).[23] Further, I hypothesize that CSE levels will vary based on $T_{n-1}$ and $T_n$ events. $T_n$ events may alter CSE levels as per Hoshiyama et al. (1997) (above). At the short imperative time in $T_n$, if either a no-go imperative or no imperative is given, CSE levels may be less than when given a go signal. Though relative MEP suppression may represent either inhibition increases or excitation decreases (see Leocani et al., 2000), if representing increased inhibition, as hypothesised by Sinclair and Hammond (2009), MEP decreases may provide an electrophysiological marker of trace reduction. Further, if $T_{n-1}$ events are considered to convey implicit information regarding the upcoming imperative, then $T_{n-1}$ imperative type and/or timing may also affect CSE (see Wardak, Ramanoël, Guipponi, Boulinguez, and Ben Hamed, 2012).

**Research Questions**

**1)** *How do traces, reinforced or extinguished in the prior trial, interact with response type and/or temporal foreknowledge?*

**2)** *Are traces reinforced or extinguished differently based on response readiness?*

**3)** *Are traces evident in current trial corticospinal excitability?*

[23] Though CSE has shown to be fairly stable within individuals (Hoonhorst, Kollen, van den Berg, Emmelot, & Kwakkel, 2014).

## 2. General Experimental Setup

### 2.1 Ethical Declaration

All experiments were conducted in accordance with the Declaration of Helsinki, and approved by the human ethics committee of Macquarie University. TMS participants were given a TMS safety screener to ensure no contraindications to stimulation.

### 2.2 Equipment

All experiments were programmed and controlled by Neurobehavioral System's Presentation software (version 16.3). Experimental stimuli were presented on a Samsung SyncMaster SA950 (27 inch) monitor controlled by a Dell Optiplex 9010 PC (8GB RAM, 3.2Ghz Intel i5-3470 CPU) running 64-bit Windows 7. Button responses for the behavioural-only experiments were collected using an in-house three-button box connected via a Measurement Computing PCI-DIO24 I/O port. A Cedrus RB-840 UST button box was used to collect TMS behavioural responses. All experiments took place in dimly lit rooms with participants seated comfortably in a chair 0.8m away from the screen with the button box positioned 0.4m away. For the TMS experiment the button box was mounted sideways such that responses were initiated by abduction of the index finger, thereby activating the first dorsal interosseous muscle (FDI), from which MEPs were measured.

Surface EMG (sEMG) was recorded (1000×gain, bandpass filtered from 0.3-1000Hz) from a bipolar electrode (Medi-Trace 100, Kendall/Tyco Healthcare, USA) montage. One electrode was placed over the muscle belly of the dominant hand's FDI muscle and the other electrode was placed over the proximal metacarpal of the index finger. A wrist strap was used for grounding. EMG was amplified using an ADInstruments dual bio-amp and digitized at 4kHz via an ADInstruments Powerlab 8/30 controlled by a computer (Dell Optiplex 9010, 8GB RAM, 2.9GHz Intel i5-3470s CPU, 64-bit Windows 7) running LabChart 7 (ADInstruments).

A monophasic transcranial magnetic stimulator (Magstim model 200, Magstim, Whitland, UK), with focal figure-of-eight stimulating coil (90-mm outer diameter), was used to elicit MEPs from the dominant hand's FDI muscle. The stimulating coil was held tangentially to the skull with the coil oriented 45° to the parasagittal plane and the handle pointing laterally and posteriorly. The centre of the coil junction was placed over the primary

motor cortex (M1) hand area of the dominant hemisphere and the "motor hot spot" was determined as the site where TMS consistently elicited the largest MEPs.

Resting motor threshold (MT) was determined by finding the lowest stimulation intensity of the motor hotspot for the dominant FDI needed in order to obtain an MEP with a peak-to-peak amplitude of 50µV in 3 out of 5 consecutive stimulations. The TMS test intensity was then set at 120% of resting MT.

## 2.3 General Experimental Procedure

Prior to each experiment, participants were introduced to the task using a picture (similar to figure 2.1). They were told to perform the tasks as quickly and accurately as possible and to look at the fixation cross between trials. It was explained that they were expected to make some mistakes, but that they should try not to respond before the 'go' imperative appeared.

All experiments required participants to push a button with the index finger of their dominant hand upon the appearance of a green square, and to withhold response if a red square appeared. [24] In the behavioural experiments, a fixation cross ('+' set at 150 point font size) was presented at the middle of the screen for 500ms, followed by a 150x150 pixel white perimeter box (20 pixel wide perimeter, RGB=[255;255;255]). This box was filled after either 200ms or 800ms with either green or red (RGB=[0;255;0], [255;0;0] respectively). After 200ms the imperative disappeared, and after 1200ms (800ms or 200ms after the short/long imperatives respectively) the white box disappeared. A 200ms blank preceded the beginning of the next trial. In all experiments, the fixation cross was either green (if indicative of a definite go, with or without temporal information), blue (if indicative of definite timing without response information) or white (RGB=[0;255;0], [0;0;255], [255;255;255] respectively). No performance feedback was given.[25] Fixed imperative timings were used to maximize predictability. For the TMS task, the fixation was 600ms and the blank period 300ms long. Otherwise stimulus timings were identical to the behavioural experiments. This was to ensure

[24] These signals can be of various modalities, including somatosensory (e.g. Nakata et al., 2004; Nakata et al., 2006) and auditory (e.g. Leocani et al., 2000; Barry, De Blasio, Rushby, and Clarke, 2010) and use various response methods including vocal (e.g. Dembowski and Watson, 1991; Watson and Alfonso, 1987), and non-motor (e.g. counting, Smith et al., 2013). See Nakata, Sakamoto, Honda, and Kakigi (2014) for review. Visual signalling and manual responses are done here to facilitate comparison with Los (2013).

[25] Though I did hear the occasional curse when someone missed a no-go signal

adequate TMS recharging times. Overall, each experiment ran for just under an hour (including setup/breaks), with each block running between five and seven minutes, and a variable, participant determined, break between each block. The general task appears thus:

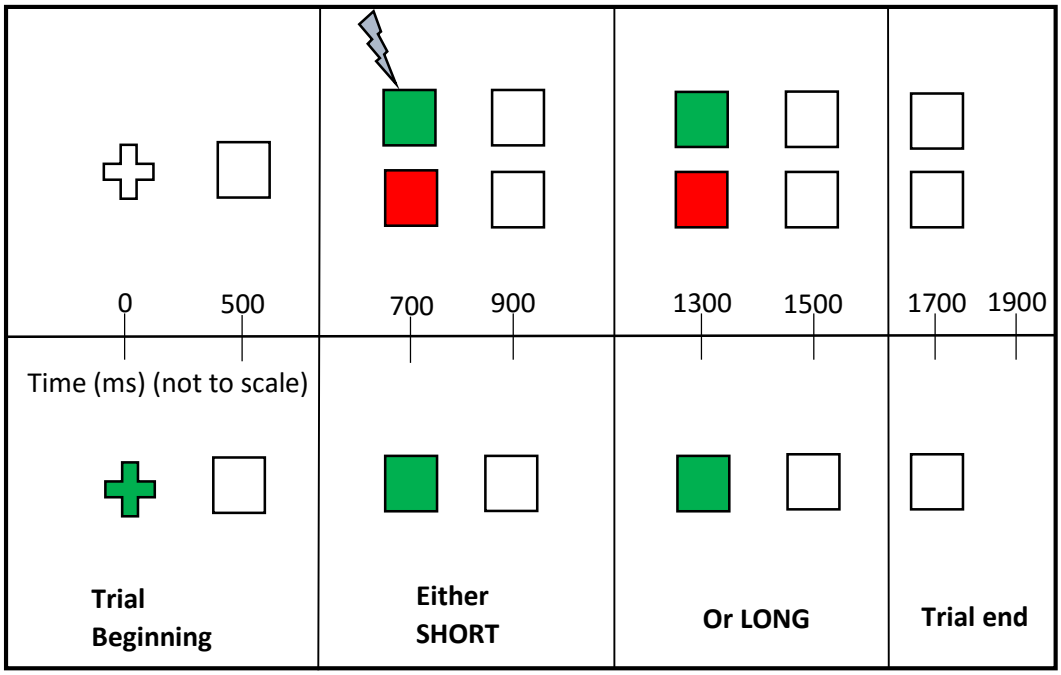


*Figure 2.1: Graphical representation of experiments. In each experiment, an imperative could occur at one of two possible time points. In experiments 1.1-1.3, either fixation cross could be presented. If the fixation was white, a go or no-go signal could be given at either the short or long time. If a marked fixation was given (in the above diagram, the green fixation from experiment 1.1) then, in experiment 1.1, response was certain to occur but without foreknowledge of when the imperative would be given. In experiment 1.2 a blue fixation gave temporal but not response type certainty, and in experiment 1.3 both temporal and response certainty was provided by a green fixation. In experiment two, only the top section was used. The lightning bolt represents TMS pulse timings (100ms post imperative onset).*

All experiments were programmed such that there was a set number of paired events, for example short-no-go and short-definite-go. These paired events were presented in a randomized order and were all equiprobable. However, due to the randomization of the order of event-pairing presentations, the exact number of duplet repetitions was not identical between subjects.[26] No rules were imposed on which trials could follow one another. Block order, where relevant (i.e. experiment 1.2-1.4), was varied randomly. In each block/experiment, only two different fixations occurred, either white or coloured. Trial/block information for each experiment is presented in table 2.1.

[26] For example, given two paired events, say SM-SM, and SD-SD, these two events as well as an SM-SD pair created between these trials, was analysed. All randomization was handled automatically by the Presentation software.

| **Experiment** | *Trials per block* | *Number of blocks* | *Duplets of each type per block (Minimum)* | *Total duplets of each type (Minimum)* | *Block time in minutes* |
|---|---|---|---|---|---|
| **TMS** | 160 | 5 | 5 | 25 | 5:36 |
| **GNG + Def Go (no time)** | 216 | 8 | 3 | 24 | 6:50 |
| **GNG + Def Go and time** | 200 | 8 | 4 | 32 (16 each block) | 6:20 |
| **GNG + Def time (no response information)** | 216 | 8 | 3 | 24 (12 each block) | 6:50 |
| **GNG + One NG timing** | 200 | 8 | 4 | 32 (16 each block) | 6:20 |

*Table 2.1: Table of information regarding each experiment's structure, including total time per block. In the bottom three rows, the numbers in the brackets represent the minimum number of each type of trial for each block. For example in the long timing blocks of the GNG + Def time experiment, in which a blue fixation represented definite knowledge of a long go timing, but with no information regarding the response, there were 12 minimum of each type of pair. These minimums are far off the actual number, often reaching roughly double this amount The yellow row indicates the experiment included in appendix one.*

For the TMS experiment, TMS pulses were delivered on every other trial. Pulses were delivered 100ms after the short-imperative onset timing, whether an imperative was present or not.

## 2.4 Participants

Participants for all experiments were recruited from first and second year psychology at Macquarie University, from the cognitive sciences department, or from public advertisement. All participants were reimbursed with either course credit or paid between $20 and $40, depending on the experiment. Participants provided written consent and were allowed to withdraw their participation at any time. Participants were not allowed to do more than one experiment. Table 2.2 below gives information regarding participants for each experiment. Throughout the experiments, only two datasets were discarded due to non-completion, one participant failed to comprehend the task, and the other's experiment computer malfunctioned. The remaining dataset discards were due to preemptive responding on the part of the participant. A minimum of ten correct responses per duplet was set as the criterion for inclusion of a dataset. The one participant discarded in the TMS experiment was due to TMS malfunction.

| **Experiment** | *Total Participants* | *Number discarded* | *Age range (mean)* | *Handedness (Right)* | *Gender (Female)* |
|---|---|---|---|---|---|
| **TMS** | 10 | 1 | 23-34 (29.4) | 8/9 | 7/9 |
| **GNG + Def Go (no time)** | 18 | 2 | 18-29 (21.1) | 14/16 | 11/16 |
| **GNG + Def Go and time** | 20 | 6 | 18-32 (22.7) | 14/14 | 9/14 |
| **GNG + Def time (no response information)** | 16 | 2 | 18-29 (21.6) | 12/14 | 11/14 |
| **GNG + One NG timing** | 23 | 11 | 18-37 (21.9) | 11/12 | 8/12 |

*Table 2.2: Table of participant information. Note, the last three columns only include information regarding the actual participants used. For example, in the last row, the total number of participants was 23, however I discarded 10 of these due to numerous problems (mostly early responding, before an imperative was presented). The rest of the information is based on the remaining 13 participants. The yellow row indicates the experiment included in appendix one.*

## 2.5 Analysis

Trial analysis was performed using 64-bit Matlab version 8.3 (Mathworks, 2014). The first block in each experiment was excluded from the analysis (half a block for TMS), and the first three trials of each block were also excluded to allow reorienting to the experimental timing (which occurs rapidly, see Repp, 2005). Further, if a mistake was made in a trial, data from that trial and the following trial were removed; a mistake may alter the following trial and is not controlled for (see Los, 2013). A mistake was considered to be any incorrect response (either responding to a no-go signal or not responding to a go signal), or a response which occurred outside the 100-800ms post-stimulus window. Early responses were considered to be predictive, and therefore representative of a different underlying construct (i.e. knowledge of timing structures) than that which was to be examined here. The current focus was on trials in which an individual was assumed to have responded to a signal rather than pre-empted it. The same argument applies to late responses, though these rarely occurred.

To detect outliers, the median absolute deviation (MAD) method was applied (Hampel, 1974; also see Leys, Ley, Klein, Bernard, and Licata, 2013; for alternatives see Rousseeuw and Croux, 1993).[27] However, as RT data is skewed, a simple application of this method would not cut off possible early outliers therefore a double-mad function was performed.[28] This split the

[27] RT data may be dealt with in a number of ways however (see Ulrich and Miller (1994), Ratcliff (1993), and Lachaud and Renaud (2011) for various considerations).
[28] I discovered this while perusing a statistics message board, as we all do right?

data at the median and applied MAD to both halves, giving two separate MAD scores. A conservative MAD cut-off was applied (3xMAD, roughly corresponding to three standard deviations).[29]

For the TMS data, an offline Butterworth bandpass filter at 10-500hz was applied to the electromyogram (EMG). The data were then rectified, and averaged across each trial combination type. Two epochs were extracted for analysis. The first was from -50 to 0ms relative to the imperative. This epoch acted as a baseline EMG measure for the MEP data. The second epoch was obtained by finding the MEP peak, and taking 25ms on either side of this.[30] The area under the EMG was then calculated for each epoch, and the second (MEP) epoch was divided by the first (pre-imperative epoch) to obtain a normalised MEP ratio. See Konrad (2005) for discussion.

Analysis for statistical significance was performed using R (R-Core-Team, 2015). To facilitate comparison with prior experiments, data was aggregated within factors and participants, then the ezANOVA function from the 'ez' package (Lawrence, 2013) was used to perform a repeated measures anova on means of RTs/MEPs.[31] Due to each experiment having varying levels of mutually exclusive factors (e.g. in experiment 1.2, a temporally certain short imperative never occurred in the block where a blue fixation indicated a definite long imperative time), factor releveling was required for each experiment. This is presented as necessary. A block factor was additionally included when appropriate. The TMS experiment included both go and no-go short trial types as factors, as MEPs were examined rather than RTs (no-go RTs are, obviously, unattainable). However, because measurements were taken at the short imperative, and there was no way to predict the long imperative type at this point, the long imperative type was not included in analysis, instead being combined into a single short-imperative type (i.e. no imperative). Mauchly's sphericity-corrected p-values are reported as required (see Field et al., 2012). Significant interactions were explored graphically and with

[29] For example, in experiment 1.1 this changed the group RT distribution; skewness changed from 9.34 with no MAD to 0.53 with MAD and kurtosis changed from 1.9 with no MAD to 2.94 with MAD.
[30] This only varied by 1/4000 of a second.
[31] See Barr, Levy, Scheepers, and Tily (2013) regarding loss of ability to model random intercepts, and why using generalized linear models may be a better approach to modeling, though is less comparable to prior research.

paired t-tests using Bonferroni-adjusted p-values. Table 2.3 is a glossary of abbreviations used within this thesis.

| Abbreviation | Meaning |
|---|---|
| **SM** | A short go trial following a maybe-go fixation |
| **LM** | A long go trial following a maybe-go fixation |
| **SNG** | A short no-go trial following a maybe-go fixation |
| **LNG** | A long no-go trial following a maybe-go fixation |
| **SD** | A short go following a definite go (green) fixation |
| **LD** | A long go following a definite go (green) fixation |
| **STG** | A short go trial following a temporally certain (blue) fixation |
| **LTG** | A long go trial following a temporally certain (blue) fixation |
| **STNG** | A short no-go trial following a temporally certain (blue) fixation |
| **LTNG** | A long no-go trial following a temporally certain (blue) fixation |
| **SG** | A short go, irrespective of indicator |
| **LG** | A long go, irrespective of indicator |
| **NG** | No-go |
| **SM_LD** | An SM followed by an LD; the code after the underscore indicates trial n |
| **LM_X** | A long maybe trial followed by another trial |
| **LP** | Long pass (i.e. the short imperative had passed). |
| **$\text{Timing}_{n-1}$** | Timing of the prior trial. |

*Table 2.3: Abbreviations used to signify various types of trials.*

## 3. Experiment 1.1

In this experiment a white fixation indicated that a go or NG signal could occur at either the short or long imperative time (200ms or 800ms post-fixation respectively). A green fixation indicated that a NG would not appear. All trials (SM, LM, SNG, LNG, SD, LD) were equiprobable. This experiment investigated the effect of response certainty without temporal certainty on RTs. Specifically, given response certainty in $T_n$, do response type and response timing of $T_{n-1}$ affect RTs in $T_n$, or is $T_{n-1}$ information overridden by the information provided by the green fixation? Secondly, are traces, and hence $T_n$ RTs, differently affected by certain versus uncertain $T_{n-1}$ trials; are trace changes sensitive to response readiness?

### 3.1 Visualization of Results

Figure 3.1 demonstrates mean RTs across $T_{n-1}$-$T_n$ trial combinations.

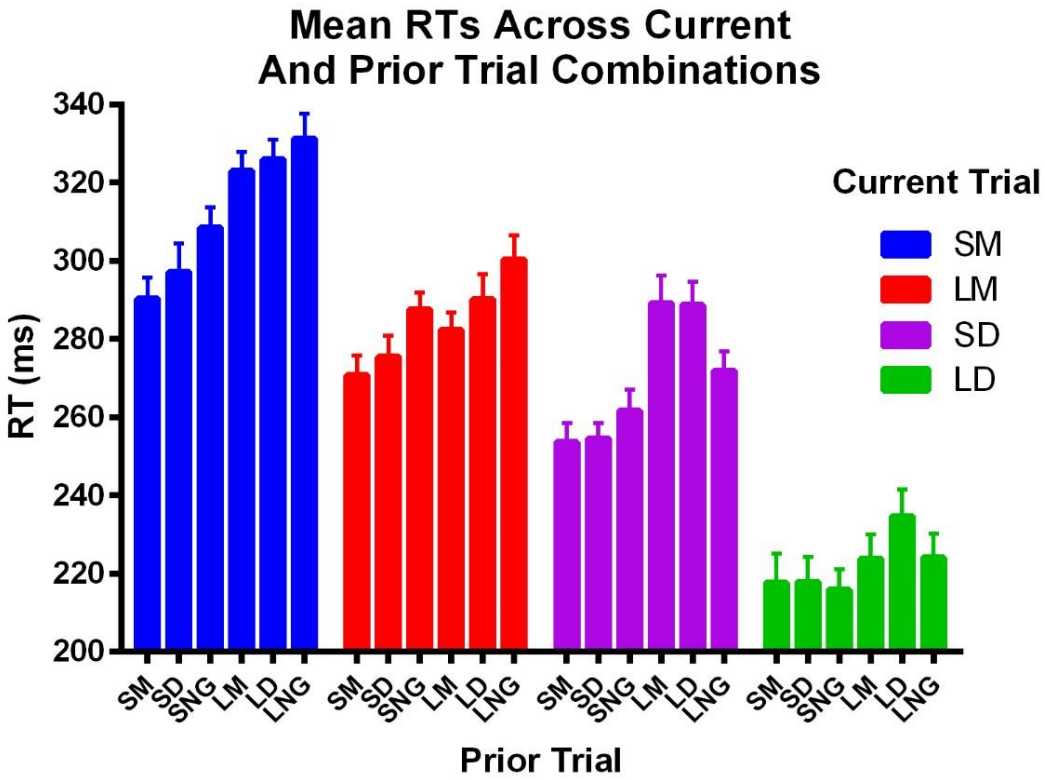


*Figure 3.1: Mean reaction times in experiment one. Grouping is by current trial type, while each bar represents a different prior trial type. Generally, reaction times get faster as more information about the response becomes available (i.e. definite trials are faster than maybe trials). Further, RTs generally repeats the findings by Los (2013) in which NG trials cause longer RTs in $T_n$, and a long $T_{n-1}$ before a short $T_n$ increases RTs. Of particular interest is the short definite condition (purple block); both long and short NG $T_{n-1}$ are associated with shorter RTs in the SD $T_n$ compared to the long $T_{n-1}$.*

Of particular interest, LD RTs (figure 3.1, far right) appear unaffected by $T_{n-1}$ trial types compared to the other three $T_n$, though a LD $T_{n-1}$ may cause comparatively slower LD RTs. Further, SD RTs (figure 3.1, middle right), do not appear differently affected by SNG $T_{n-1}$ compared to a SD/SM $T_{n-1}$. LNG $T_{n-1}$ effects on SD RTs also warrant discussion; at the LNG imperative time, the short imperative time has passed, and therefore, according to Los (2013), a LNG $T_{n-1}$ should have the same effect on $T_n$ RTs as does a LG $T_{n-1}$ by virtue of its timing.[32]

### 3.2 Results

Mean RTs ranged from 210-344ms per individual (mean=272.4ms, SD=52.9ms). The number of errors per participant ranged from 18-238 (13.8% of trials). Response errors were rare (<2% of trials); most errors were early responses near the LD $T_n$. Occasionally, participants released responses near the SD imperative time despite no imperative being given. This constituted ≈10% of errors. Significant interactions not superseded are presented in table 3.1 below.[33]

**Table 3.1: Significant interactions in experiment**

| Interaction | DF (n/d) | F-value | Probability | $\eta_p^2$ |
|---|---|---|---|---|
| $Timing_n$ x $Certainty_n$ | 1, 15 | 23.45 | <0.05 | 0.61 |
| $Certainty_n$ x $Type_{n-1}$ | 2, 30 | 8.01 | <0.01 | 0.24 |
| $Timing_{n-1}$ x $Timing_n$ | 1, 15 | 9.67 | <0.01 | 0.39 |
| $Timing_{n-1}$ x $Type_{n-1}$ | 2, 30 | 9.70 | <0.01 | 0.28 |

*Table 3.1: Statistical significance of those interactions not superseded by other interactions in experiment 1.1. The first column specifies the interaction. The second specifies the degrees of freedom. F-values are then presented, followed by their corresponding p-value. The highlighted, row is the corrected value due to sphericity violations (Greenhouse-Geisser estimates for sphericity corrections of DF were used, see Field et al. (2012)). The final column is the effect size (partial eta-squared).*

[32] This effect seemed surprising, however was repeated in the experiment in the appendix. Further, preliminary results from a vocal version of this experiment also demonstrate this effect (not reported here).

[33] Though I have used $\eta_p^2$ (for calculations see Lakens, 2013) as a measure of effect size, this may not be the ideal measure (see Bakeman, 2005). However, if using the more appropriate generalised $\eta^2$, effect sizes were under one percent. This does not mean that the significant effects were irrelevant, but rather that individual variation superseded $T_n$ and $T_{n-1}$ dynamics. Further, $\eta_p^2$ is the measure often employed in psychology papers, therefore facilitates comparison with other research. This argument holds for the remainder of the thesis.

*Interaction between current trial timing and certainty* – The first interaction was between the certainty and timing of $T_n$, demonstrated in figure 3.2 below.

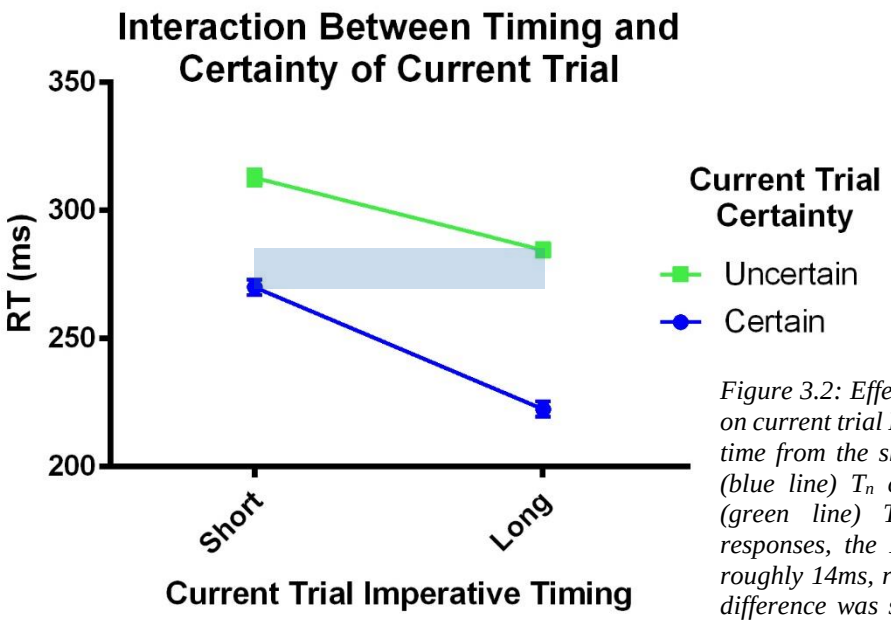


*Figure 3.2: Effect of current trial timing and certainty on current trial RTs. RTs appear to decrease more over time from the short to long imperative in the certain (blue line) $T_n$ condition compared to the uncertain (green line) $T_n$ condition. Despite equiprobable responses, the LM and SD points are separated by roughly 14ms, represented by the blue rectangle. This difference was significant. All individual points were significantly different from all others (maximum $p<0.001$)*

In the response-certain condition (figure 3.2, blue line), RTs decreased from the short to long imperative time by ≈48ms, while the decrease in the response-uncertain condition was ≈28ms. Response certainty in $T_n$ resulted in shorter RTs than when responses were uncertain. A response-certain $T_n$ was responded to faster than a temporally-certain $T_n$, despite the same probability of response (i.e. LM/SD). The difference between these is marked by the blue box in figure 3.2, representing a significant RT difference (≈14ms, $p<0.001$).[34]

[34] Each point in figure 3.2 was significantly different than all other points (all $p<0.001$).

*Interaction between certainty of the current trial and the prior trial type* – The second significant interaction was between the certainty of $T_n$ and type of $T_{n-1}$, demonstrated in figure 3.3.

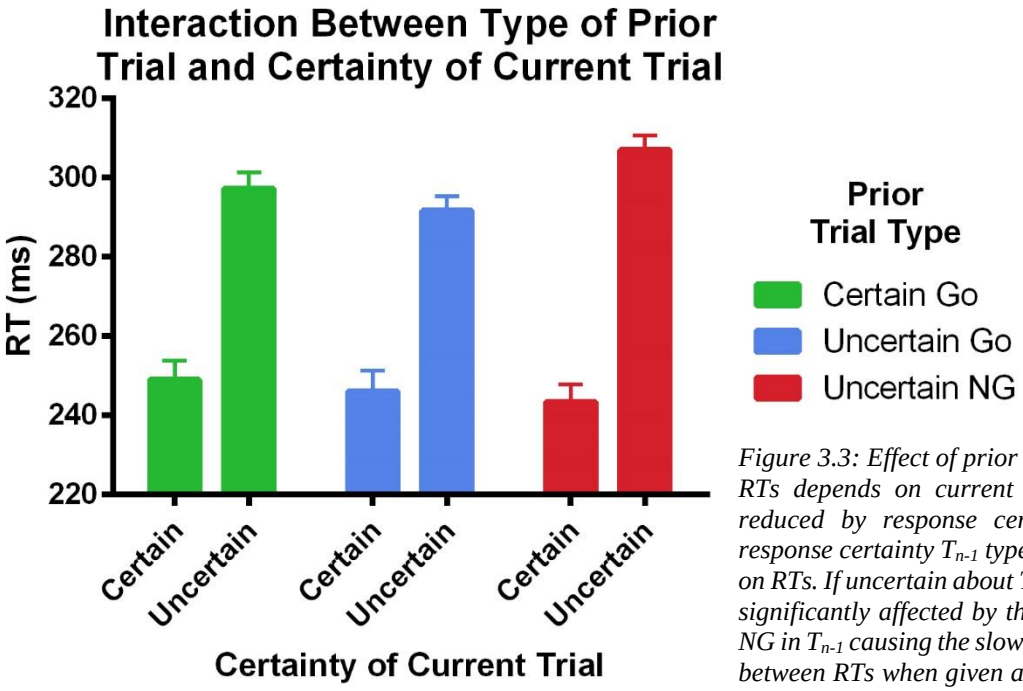


*Figure 3.3: Effect of prior trial type on current trial RTs depends on current trial certainty. RTs are reduced by response certainty in $T_n$. Given $T_n$ response certainty $T_{n-1}$ type had no significant effect on RTs. If uncertain about $T_n$ response, type, RTs are significantly affected by the prior trial type, with a NG in $T_{n-1}$ causing the slowest $T_n$ RTs. The difference between RTs when given a certain versus uncertain $T_n$ is also greatest when $T_{n-1}$ was a NG (i.e. the difference between the red bars).*

The difference between certain and uncertain $T_n$ RTs was not different when a response was made in $T_{n-1}$ (≈49ms between the green (left) bars in figure 3.3, ≈45ms in the middle group), while the difference between certain and uncertain $T_n$ RTs is increased when a response was withheld in $T_{n-1}$ (figure 3.3, right, difference ≈63ms). The difference between certain-uncertain $T_n$ groups was significant when comparing the NG and uncertain-go $T_{n-1}$ trials ($p<0.005$) and approached significance between the NG and certain-go $T_{n-1}$ trials ($p=0.082$). The difference between the certain- and uncertain-go groups was not significant ($p=0.863$). The type of $T_{n-1}$ made no significant difference to $T_n$ RTs if a $T_n$ was a definite-go (all $p>0.16$ minimum), while, when $T_n$ was uncertain, all $T_{n-1}$ types made a significant difference to $T_n$ RTs (all $p<0.01$, except the difference between uncertain- and certain-go in $T_{n-1}$ where $p<0.05$).

*Interaction between current and prior trial timing* – Figure 3.4 illustrates the interaction between $T_{n-1}$ and $T_n$ imperative timing. This interaction demonstrates the classical VF asymmetry (e.g. Los (2013)). Each point was significantly different from the others ($p<0.0001$)).

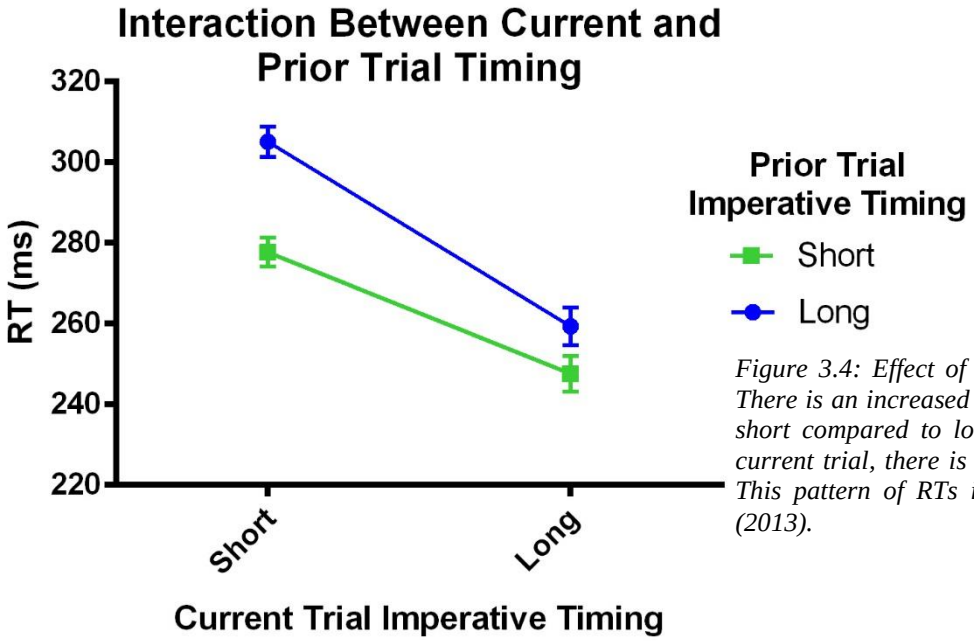


*Figure 3.4: Effect of current and prior trial timing. There is an increased effect of a long prior trial on a short compared to long current trial. Given a long current trial, there is less effect of prior trial timing. This pattern of RTs is in line with findings of Los (2013).*

To better compare these results to those of Los (2013) all definite-go trials (up to $T_{n-1}$) were removed (i.e. only analysing SM, SNG, LM, LNG). Reanalysis revealed that the significant difference between long $T_n$ RTs given a short/long $T_{n-1}$ timing, remained ($p<0.001$). Given a long $T_{n-1}$ (figure 3.4, blue line), RTs decreased from the short to the long $T_n$ by ≈46ms, while if given a short $T_{n-1}$ (figure 3.4, blue line), RTs decreased from the short to the long $T_n$ by ≈30ms. Further, for the short $T_n$, RTs were ≈27ms slower following a long $T_{n-1}$ than a short $T_{n-1}$. The results of this interaction indicate that, firstly, decreases in RTs from a short $T_{n-1}$ to long $T_{n-1}$ were less for the long $T_n$, secondly that a short $T_n$ was responded to slower than a long $T_n$, irrespective of $T_{n-1}$ timing ($p<0.0001$). And finally, that a long $T_n$ was responded to faster when preceded by a short rather than long $T_{n-1}$ ($p<0.0001$).

*Interaction between the timing and type of the prior trial* – The final significant interaction was between $T_{n-1}$ type and timing (figure 3.5, below). When preceded by a long $T_{n-1}$, no $T_n$ RTs were different irrespective of $T_{n-1}$ type (all $p>0.14$ minimum). When given a short $T_{n-1}$, only the RTs for uncertain-go and NG were significantly different from each other ($p<0.001$, all others $p>0.25$). If there was a response (i.e. certain- or uncertain-go) in $T_{n-1}$, the difference in $T_n$ RTs was approximately equivalent irrespective of $T_{n-1}$ timing (i.e. the difference between the bars in the left two groups of figure 3.5) (certain≈23ms, uncertain≈21ms) while the difference when a NG occurred in $T_{n-1}$ was ≈13ms. Thus there was a smaller effect of $T_{n-1}$ response type on $T_n$ RTs when the response in $T_{n-1}$ occurred at the short time compared to the long time.

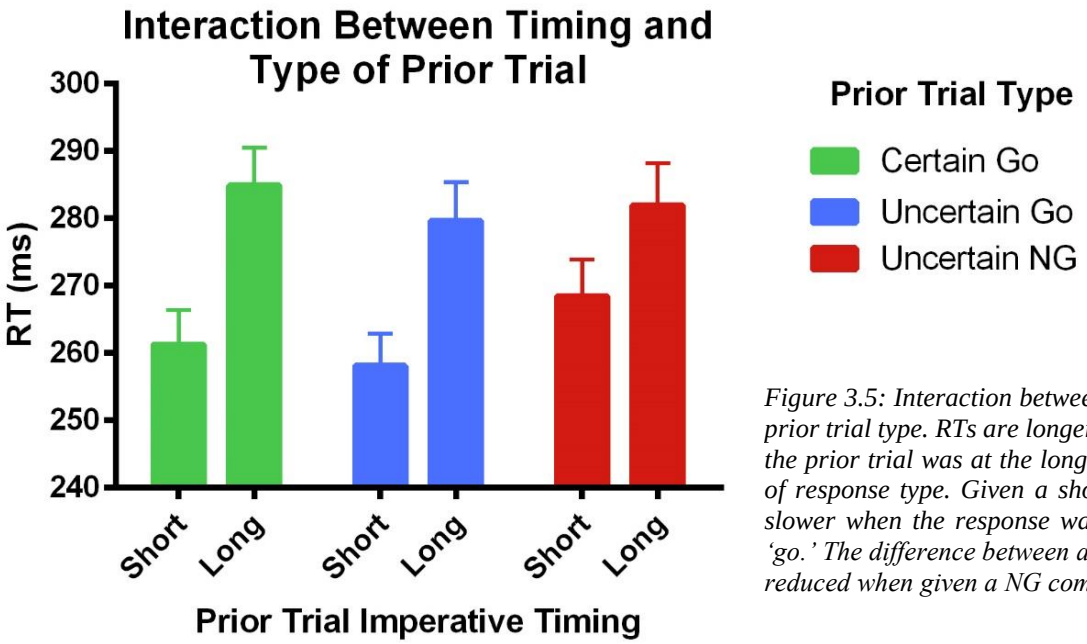


*Figure 3.5: Interaction between prior trial timing and prior trial type. RTs are longer when the imperative in the prior trial was at the long time point, irrespective of response type. Given a short $T_{n-1}$, RTs in $T_n$ were slower when the response was a NG compared to a 'go.' The difference between a short and long $T_{n-1}$ was reduced when given a NG compared to a 'go' $T_{n-1}$.*

This result is congruent with the reduced effect of a NG $T_{n-1}$ when the required response in $T_n$ was certain; a SNG $T_{n-1}$ was significantly slower than a SM $T_{n-1}$ in the SM $T_n$ ($p<0.01$), but not in the SD, LD or LM $T_n$ (all $p>0.9$). The LNG $T_{n-1}$ was not significantly different from any trial in the SD $T_n$ ($p\approx1$), except a SM ($p<0.05$). A LNG was ≈17ms faster than a LM $T_{n-1}$ in the SD $T_n$ condition. The lack of effect of a SNG $T_{n-1}$ on RTs is notable in comparison to SM RTs which were significantly slower when preceded by a SNG trial ($p<0.01$). SD RTs were significantly faster given a short-go rather than long-go $T_{n-1}$ (all $p<0.05$, except between SD and LM $T_{n-1}$, $p=0.062$).

The table on the following page represents significant differences between individual duplets.

**Table 3.2: Significant differences between individual duplets in experiment 1.1**

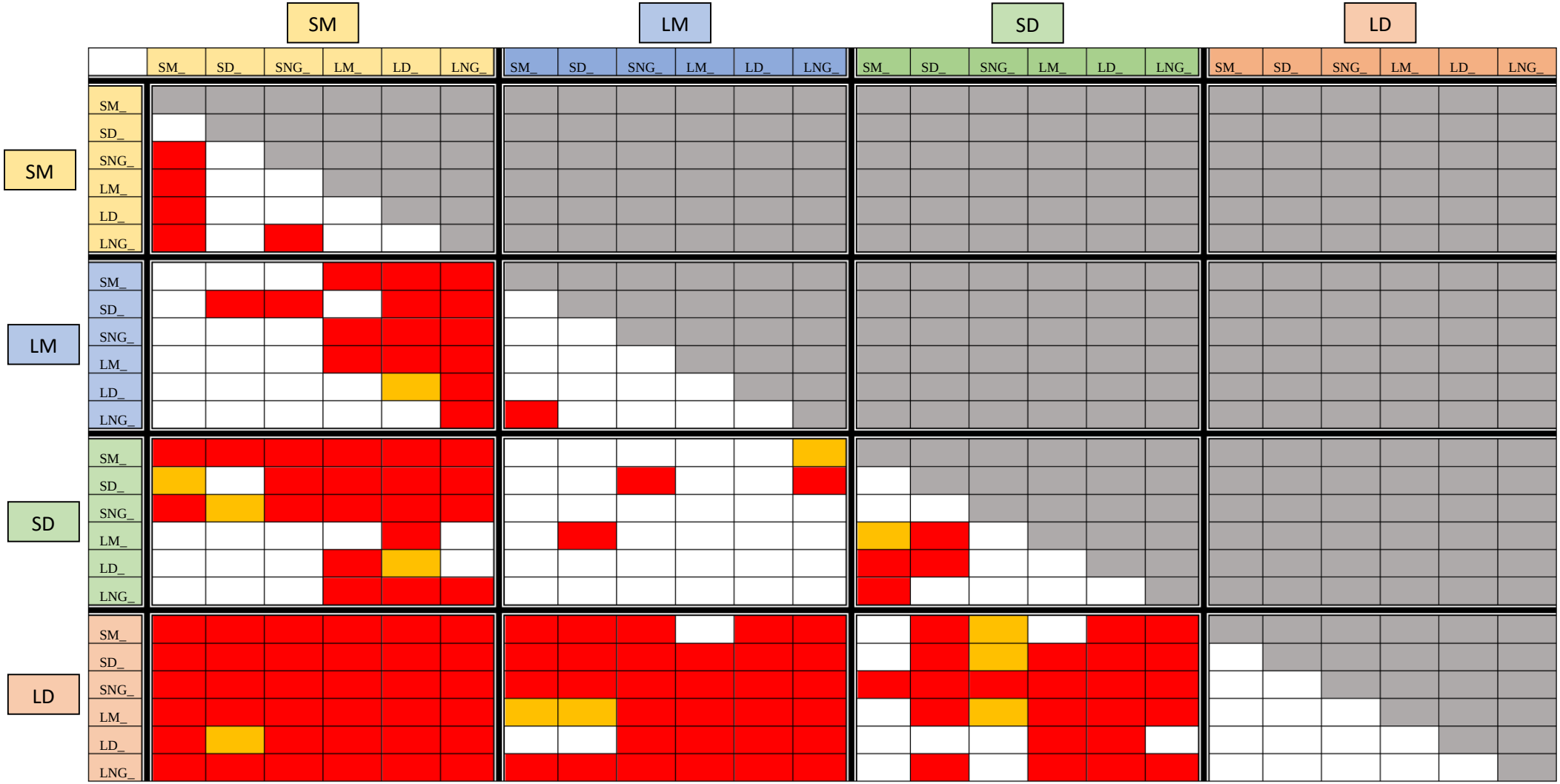


*Table 3.2: Table showing the significant differences between pairs of duplets in experiment 1.1. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

## 3.3 Discussion

### *3.3.1 Does current trial response certainty affect prior information utilisation?*

Given $T_n$ response certainty, RTs decrease.[35] Further, while $T_{n-1}$ types make a difference to RTs when the upcoming response type is uncertain, these effects are diminished when the upcoming response type becomes certain, as evidenced by the interaction between $T_n$ certainty and $T_{n-1}$ type. In the LD $T_n$ condition it makes sense that $T_{n-1}$ dynamics have little or no effect on RTs. In line with Criaud, Wardak, Hamed, Ballanger, and Boulinguez (2012), passing the short imperative time in the response-certain trials may act like a signal for proactive inhibition release; the timing and response of the upcoming imperative are known and therefore the prepared response can be relatively disinhibited. In terms of proactive response 'braking', perhaps the response-oriented brake is released by response certainty (Aron, 2011; Jahfari et al., 2010; Van Elswijk et al., 2007). This finding is not necessarily in conflict with the trace-conditioning model. It may be that the trace is irrelevant to the current response likelihood therefore $T_{n-1}$ effects are neutralised (as per Adam and Koch, 2009).

Before the response-certain short imperative time, response information is still known; a green imperative gives perfect foreknowledge of the event type. However when the imperative will occur is unknown. You can release your brakes, but you do not know when the light will turn green. If proactive inhibition is disengaged, as in the experiment by Criaud et al. (2012), at the beginning of the trial, the odds of responding at the short imperative time without an imperative being given may be relatively high (this happened on occasion) due to the temporally-linked possibility of response.[36] Generally however, this did not appear to be the strategy taken; a long compared to short $T_{n-1}$ slowed SD RTs. The trace-conditioning model theorises that RTs will be affected by the short imperative trace, an effect determined by inhibition applied to that imperative time's trace, during $T_{n-1}$. This predicts that a SNG $T_{n-1}$ should slow $T_n$ RTs regardless of $T_n$ knowledge; the trace is affected in $T_{n-1}$ before knowledge of $T_n$. However, RTs were not significantly different between SNG_SD[37] and SM/SD_SD duplets, while LM/LD_SD and SM/SD_SD RTs were significantly different. This indicates that the response of $T_{n-1}$, selectively, had a reduced effect on current response expectations,

[35] This is a common, and logical finding; for example see Soto, Valls-Sole, and Kumru (2010).
[36] Think of a temporal version of the start-react (see Carlsen, Maslovat, et al., 2012)).
[37] As a reminder, this indicates a SNG in $T_{n-1}$ and a SD in $T_n$.

while $T_{n-1}$ timing effects remained. This may indicate separable temporal and response $T_{n-1}$ effects on $T_n$ RTs.[38]

There are other possible explanations for this, for example separate definite-go traces or psychological refraction which will be discussed in chapter seven.

### *3.3.2 Does prior trial response certainty differentially affect current trial RTs?*

Given any $T_{n-1}$ certain-uncertain pair (i.e. a SM-SD, or LM-LD), there was no significant difference in $T_n$ RTs. This seems to indicate that there is little, if any, difference in $T_n$ RT effects driven by response readiness in $T_{n-1}$. Supporting this, the interaction between $T_{n-1}$ type and timing showed no significant difference between LD-LM or SD-SM $T_{n-1}$ effects on $T_n$ RTs. If the effects of passing the short imperative moment were related to response readiness, then a LD and LM $T_{n-1}$ should have caused different SM or SD RTs. Thus response certainty appears irrelevant to trace extinction. Proactive (temporal) inhibition may uniformly inhibit an imperative trace when an imperative time is passed, despite the current level of preparedness to respond.

However, this is not the full story. If $T_n$ response is uncertain, certainty in $T_{n-1}$ results in significantly longer $T_n$ RTs (≈6ms) than uncertainty in $T_{n-1}$, as shown by the interaction between $T_n$ certainty and $T_{n-1}$ type.[39] Perhaps switching from the definite task to the uncertain task has some cost on uncertain-go RTs (for further discussion, see Ruthruff, Remington, and Johnston, 2001).[40] Further, perhaps the cost of switching (and gains from repetition) are ameliorated by the foreknowledge of the upcoming response, explaining the lack of differential $T_{n-1}$ effects on response-certain $T_n$ trials.

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[38] Though LNG_SD and SD_SD RTs were not significantly different, LNG_SD and SM_SD RTs were and therefore is not commented on, requiring further research.

[39] This difference may well have showed up in the definite condition as well, however definite conditions tended to have higher variability, at least in part due to less numbers of trials making up the means as will be discussed shortly.

[40] This may be relatively low due to the higher probability of the uncertain trial type.

## 4. Experiment 1.2

In this experiment, a white fixation indicated a go or NG could occur at either the short or long imperative time. A blue fixation indicated a go or NG could occur at one imperative time (defined at the beginning of each block). SM, LM, SNG, LNG, and either STG/STNG or LTG/LTNG were equiprobable. The prior experiment demonstrated a reduction of $T_{n-1}$ response effects on $T_n$ when given knowledge of the upcoming response. This experiment provides temporal certainty without response certainty, investigating if a similar pattern of reduced $T_{n-1}$ effects on $T_n$ RTs exists in the temporal domain. Specifically, given temporal certainty, do $T_{n-1}$ temporal dynamics affect $T_n$ RTs? Further, does temporal certainty in $T_{n-1}$ have a distinguishable effect on $T_n$ RTs?

### 4.1 Visualization of Results

Figures 4.1 and 4.2 below represent mean RTs for the long (i.e. a blue fixation indicated a long $T_n$ without response certainty) and short blocks respectively.

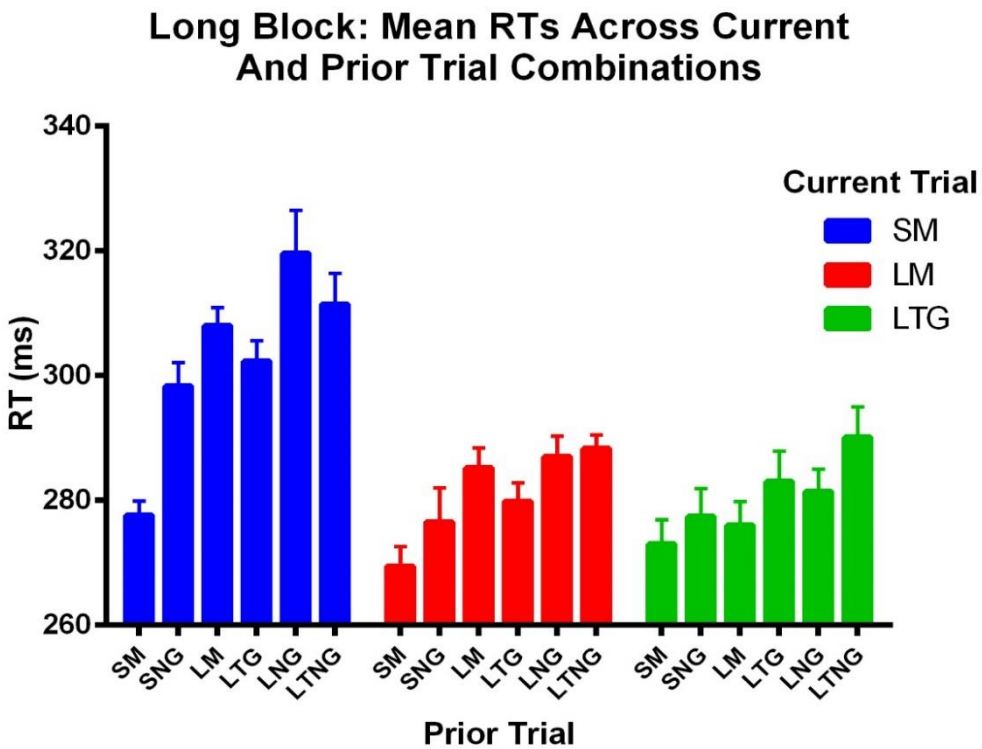


*Figure 4.1: Mean RTs in the long block (blue fixation indicates imperative will occur at the long time point). Each column represents a different prior trial, while each group is a different current trial. In the SM $T_n$ a SNG appears to slow RTs, and a long $T_{n-1}$ further slows $T_n$ RTs (as per Los (2013)). LM RTs are slower given a long $T_{n-1}$ compared to a short $T_{n-1}$ despite response. LTG RTs appear relatively unreactive to $T_{n-1}$ trial type, except possibly when comparing the effects of a LTNG, which may slow $T_n$ RTs.*

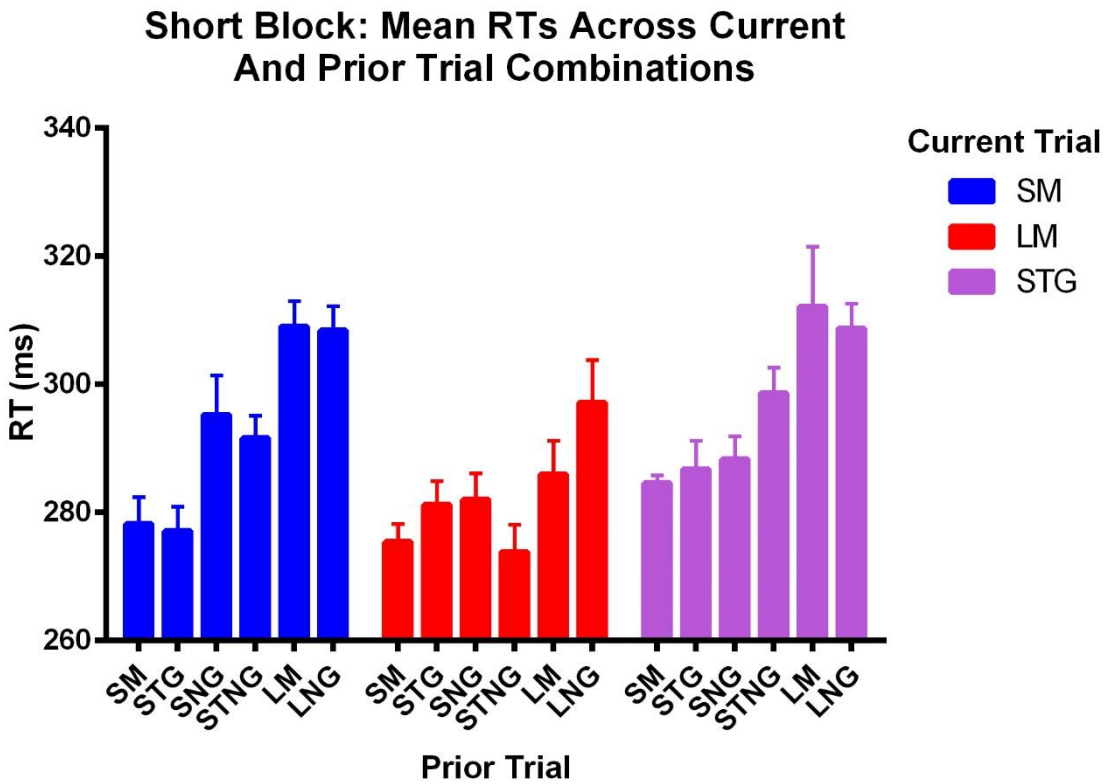


*Figure 4.2: Mean RTs in the short block (blue fixation indicates imperative will occur at short time point). In the SM $T_n$ a SNG appears to slow RTs, and a long $T_{n-1}$ further slows $T_n$ RTs (as per Los (2013)). The LM group also appears similar to Los (2013), with a LNG slowing $T_n$ RTs compared to other $T_{n-1}$ types. STG RTs appear slower given a STNG in $T_{n-1}$ compared to a SM/STG/SNG $T_{n-1}$ and further slowed following a LM/LNG $T_{n-1}$.*

As in experiment 1.1, the marked fixation trials are of interest. LTG RTs (figure 4.1, right) appeared similar to LM RTs; $T_n$ RTs appeared relatively unreactive to $T_{n-1}$ effects. STG RTs (figure 4.2, right) also appeared similar to SM RTs. However, while a long $T_{n-1}$ still slowed SD RTs compared to a SM $T_{n-1}$, the difference in $T_n$ RTs when given short versus long $T_{n-1}$ did not appear to be as large as when $T_n$ was a SM.

## 4.2 Results

Long block (blue fixation=long imperative) mean RTs per participant ranged from 247-327ms (mean=288, SD=29.6ms) and from 250-340ms in the short blocks (mean=291ms, SD=33ms). The number of errors per participant varied from 2 (0.2% of trials) to 102 (4.9% of

trials) (mean=31, SD=27.9).[41] For the participant with the most errors, all but two were caused by responding inappropriately to a NG signal.

Before investigating significant interactions within each block, block effects were examined; do the different meanings of the blue fixation in the two blocks have a generalised effect on RTs? To examine this all $T_n$ and $T_{n-1}$ temporally certain trials were removed. This meant that, at least as far as $T_{n-1}$, the two blocks were equivalent. Comparing the two blocks indicated no differential effect on SM and LM RTs (paired t-test, t(111)=0.548, p=0.59). Long block RTs were ≈1ms longer than short blocks on average.[42]

To analyse individual blocks required splitting data into three factors to avoid mutual exclusivity.[43] The factors used in the analysis were; $T_{n-1}$ response (go, NG), $T_n$ type (SM, LM, LTG or STG) and $T_{n-1}$ type (SM, LM, LT or ST). Tables 4.1 and 4.2 present significant effects and interactions not superseded in the long and short blocks respectively.

**Table 4.1: Significant interactions in long blocks**

| Interaction | DF (n,d) | F-value | Probability | $\eta_p^2$ |
|---|---|---|---|---|
| $Response_{n-1}$ | 1, 13 | 7.39 | <0.05 | 0.36 |
| $Type_n$ x $Type_{n-1}$ | 4, 52 | 4.40 | <0.005 | 0.06 |

*Table 4.1: Statistical significance of those interactions not superseded by other interactions in the long blocks of experiment 1.2. No other interactions were significant nor approached significance.*

**Table 4.2: Significant interactions in short blocks**

| Interaction | DF (n,d) | F-value | Probability | $\eta_p^2$ |
|---|---|---|---|---|
| $Type_{n-1}$ | 2, 26 | 10.56 | <0.01 | 0.22 |
| $Type_n$ | 2, 26 | 21.41 | <0.001 | 0.31 |
| $Type_{n-1}$ x $Type_n$ x $Response_{n-1}$ | 4, 44 | 3.3 | = 0.057 | 0.07 |

*Table 4.2: Statistical significance of those interactions not superseded by other interactions in the short blocks of experiment 1.2. The top and bottom highlighted rows are corrected values due to sphericity violations (Greenhouse-Geisser estimates for sphericity corrections of DF were used) (see Field et al. (2012)). The three way interaction was initially significant (type of prior/current trial and prior trial response) however was no longer significant when corrected for sphericity violation. This interaction still approaches significance. Further, the $response_{n-1}$ main effect approached significance (p = 0.057) however would have been superseded by the three way interaction.*

[41] As this experiment was the only one with 50% probability of going in both situations at both time points, the smaller number of errors compared to the other experiments makes sense (the high error rate of the other experiments tended to be driven by early response release).

[42] This was also true for the individual blocks when compared in a similar fashion (LM: p=0.34, SM: p≈1).

[43] A long trial was never presented given a blue fixation in the short block for example.

*4.2.1 Long blocks*

*Main effect of prior trial response* – The first significant effect in table 4.1 indicates a main effect of $T_{n-1}$ response on $T_n$ RTs; the withholding of a response in $T_{n-1}$ (NG) resulted in significantly longer (≈8.5ms) $T_n$ RTs compared to when a response was made (figure 4.3 below). For both LTG and LM $T_n$ trials there was no significant difference in RTs on comparison of go or NG $T_{n-1}$ contingency ($p \approx 1$ and $p=0.86$ respectively). However SM RTs were significantly longer when $T_{n-1}$ was a NG ($p<0.01$). Examining LM and LTG $T_n$ trials, RTs were unreactive to $T_{n-1}$ NG presentation compared to any other $T_{n-1}$ (minimum $p>0.4$). SM RTs were slower when preceded by any NG $T_{n-1}$ compared to a SM $T_{n-1}$ (SNG; $p<0.05$, LNG; $p<0.05$, and LTNG; $p<0.001$, SNG≈21ms slower, LNG≈49ms slower, and LTNG≈34ms slower).

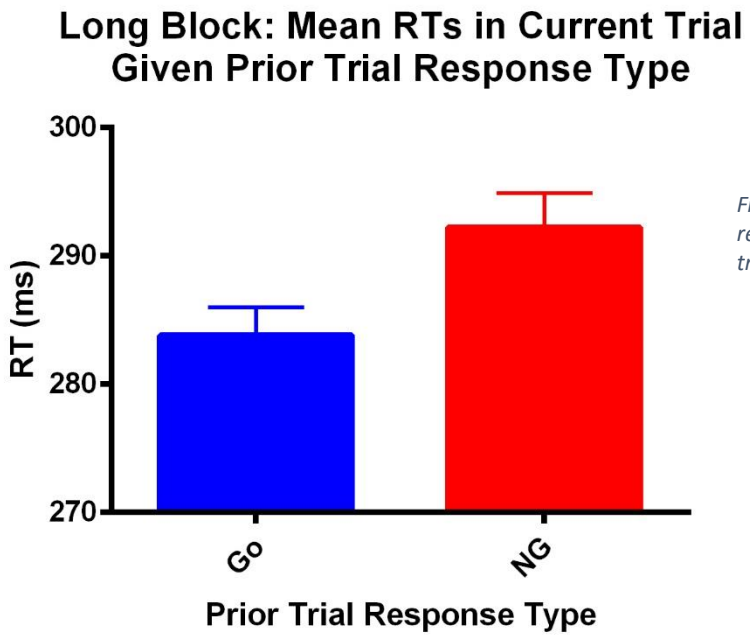


*Figure 4.3: Mean RTs in $T_n$ given a go versus NG response in $T_{n-1}$. When releasing a response in the prior trial, $T_n$ RTs were faster by ≈8.5ms.*

*Interaction between prior and current trial type* – The second significant effect in table 4.1 indicates that RTs were affected by the interaction between $T_{n-1}$ and $T_n$ type (i.e. a LM, SM or LT at both time points). This is presented in figure 4.4 below.

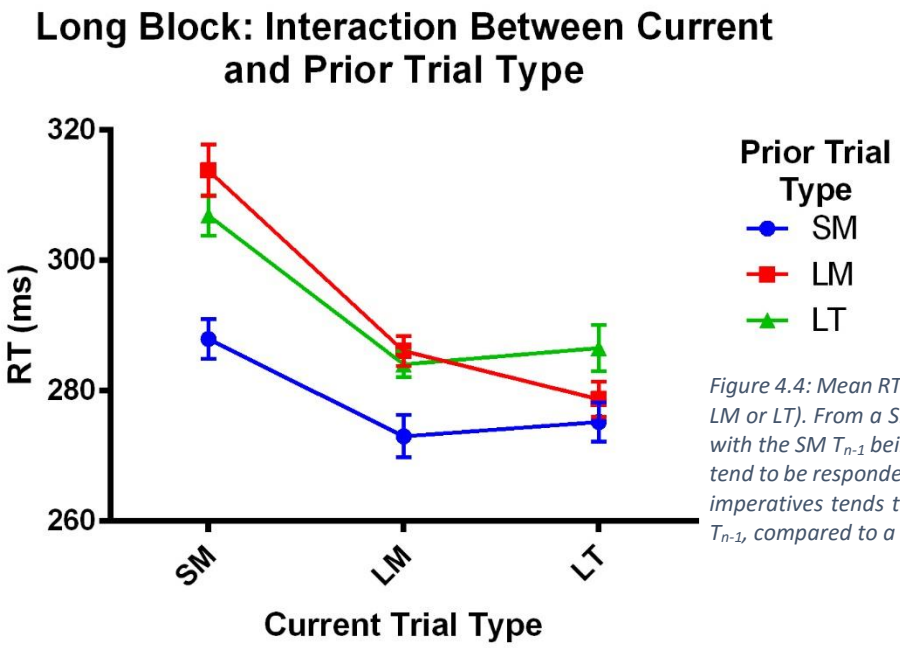


*Figure 4.4: Mean RTs in $T_n$ given prior and current trial type (SM, LM or LT). From a SM to a LM $T_n$ RTs generally drop in parallel, with the SM $T_{n-1}$ being responded to quickest in $T_n$. A SM and LT tend to be responded to equally quickly in the LT $T_n$ however LM imperatives tends to be responded to faster when given a LM $T_{n-1}$, compared to a LT $T_{n-1}$.*

SM and LM RTs were not significantly different when preceded by either LT or LM $T_{n-1}$ (figure 4.4, green and red lines respectively) (both p≈1), while the SM $T_{n-1}$ (figure 4.4, blue line) resulted in significantly faster RTs than both LT and LM $T_{n-1}$ in SM/LM $T_n$ (all $p<0.001$), except when comparing the effects of LM and SM $T_{n-1}$ on LM RTs (which approached significance, $p=0.085$). LT RTs were equally fast irrespective of what trial type preceded it (however the difference in LT RTs between a SM and LT $T_{n-1}$ approached significance, $p=0.098$). SM RTs were ≈7ms apart given a LT and LM $T_{n-1}$, while SM $T_{n-1}$ resulted in ≈19ms faster SM RTs compared to a LT $T_{n-1}$. LM RTs were again similar given a LM or LT $T_{n-1}$ (difference≈2ms), while a SM $T_{n-1}$ produced faster LM RTs (≈12ms compared to LT $T_{n-1}$). LT RTs were more similar given a SM or LM $T_{n-1}$ (difference≈3ms), while LT RTs were ≈11ms slower given a LT compared to LM $T_{n-1}$. When averaging $T_n$ RTs across all $T_{n-1}$, LM/LD RTs were not significant different (p≈1),[44] while both these groups' RTs were significantly different from SM RTs (both $p<0.0001$).

[44] This was also true when comparing each $T_{n-1}$ $T_n$ pair; all p≈1, indicating no difference in average RTs between $T_{n-1}$ in terms of LM and LTG $T_n$ RTs.

*4.2.2 Short blocks*

*Main effect of prior trial type* – Figure 4.5 below demonstrates the main effect of $T_{n-1}$ type on $T_n$ RTs. A LM $T_{n-1}$ was responded to ≈ 20ms slower in $T_n$ than a ST $T_{n-1}$. The difference in $T_n$ RTs between LM $T_{n-1}$ and SM/ST $T_{n-1}$ were both significant (all $p<0.001$ maximum). There was no significant difference in $T_n$ RTs given a SM or ST $T_{n-1}$ ($p=0.53$, RT difference≈1ms).

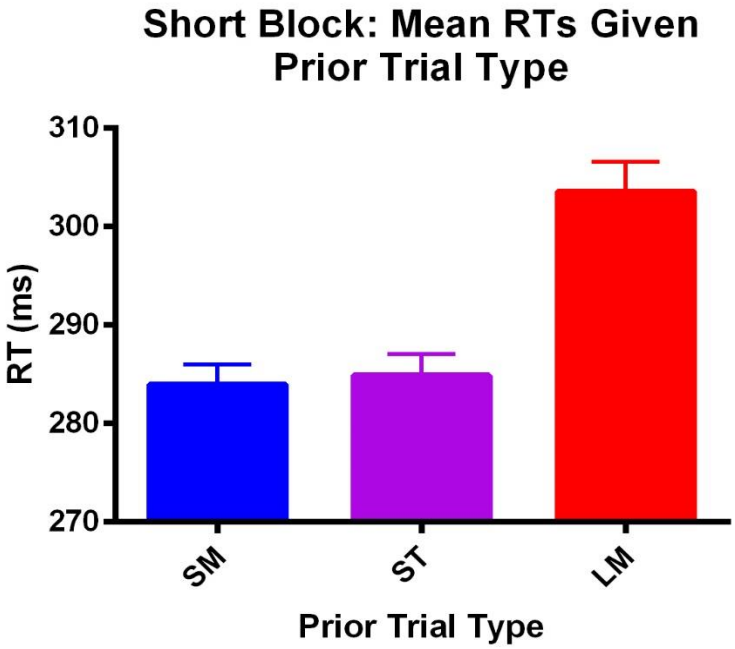


*Figure 4.5: Mean RTs changes in $T_n$ depending on prior trial type within the short block. While a ST and SM $T_{n-1}$ affect $T_n$ RTs similarly, LM $T_{n-1}$ resulted in significantly slower $T_n$ RTs by ≈20ms.*

For SM $T_n$, all short 'go's in $T_{n-1}$ resulted in faster RTs than all long trial types in $T_{n-1}$ (all $p<0.05$ maximum). SNG/STNG $T_{n-1}$ did not produce significantly different SM RTs compared to any long/short $T_{n-1}$ (minimum $p=0.15$). ST RTs were only significantly different when comparing LNG versus SM $T_{n-1}$ effects ($p<0.05$). LM RTs were not affected by $T_{n-1}$ timing (or type for that matter) (all $p\approx1$).

*Main effect of current trial type* – Figure 4.6 below demonstrates the main effect of $T_n$ type on $T_n$ RTs. RTs were not significantly different when given either a temporally certain or uncertain short $T_n$ ($p\approx1$),[45] however both short $T_n$ RTs were significantly slower than LM RTs (all $p<0.0001$). LM RTs were, on average, ≈11ms faster than SM RTs, while ST RTs were a further ≈3ms slower. Comparing the effect of any single $T_{n-1}$ trial (for example SM $T_{n-1}$) between SM

[45]No individual combination of $T_{n-1}$ $T_n$ RTs were significantly different when comparing between SM and ST $T_n$ RTs.

and ST RTs did not reveal any significant differences (all p≈1). Differences between SM-LM and SNG-LNG $T_{n-1}$ did not have significantly different effects on $T_n$ RTs when comparing between SM and STG RTs (p≈1) (e.g. the SM RT difference when given a SM compared to LM $T_{n-1}$ was not significantly different than when comparing the same $T_{n-1}$ effects on STG RTs).

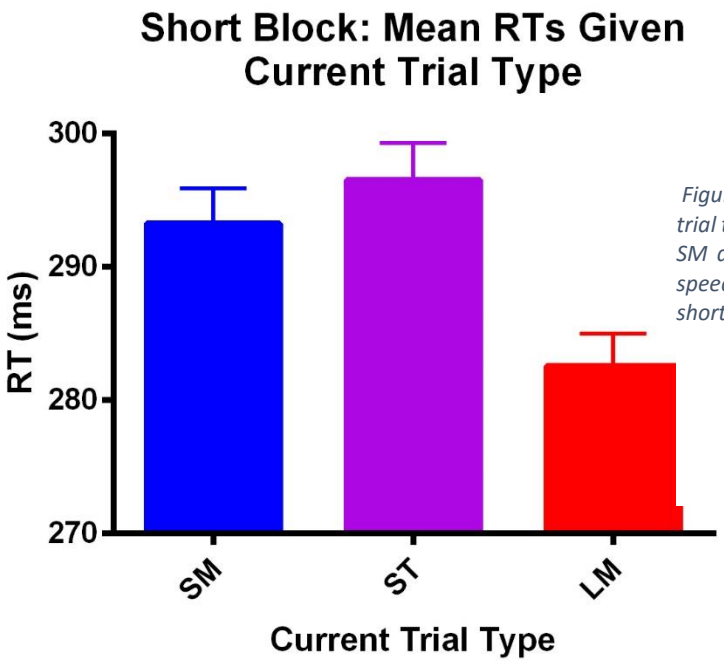


*Figure 4.6: Mean RTs changes in depending on current trial type, within the short block. As in figure 4.5 above, a SM and ST $T_n$ was responded to at roughly equivalent speeds. A LM $T_n$ was responded to quicker than either short $T_n$ trial type.*

The tables on the following pages represents significant differences between individual duplets in the long and short blocks respectively.

**Table 4.3: Significant differences between individual duplets in the long blocks of experiment 1.2**

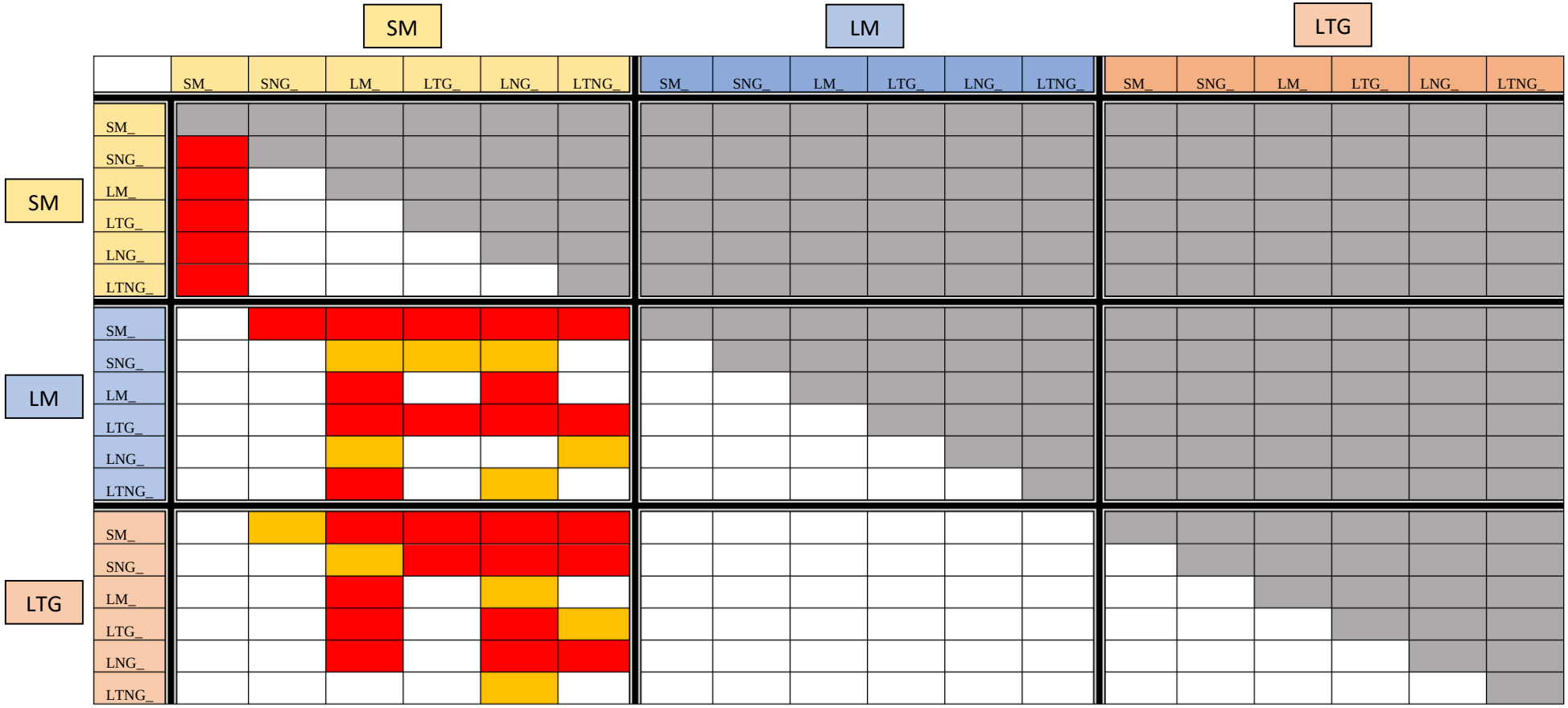


*Table 4.3: Table showing the significant differences between pairs of duplets in the long blocks of experiment 1.2. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

**Table 4.3: Significant differences between individual duplets in the short blocks of experiment 1.2**

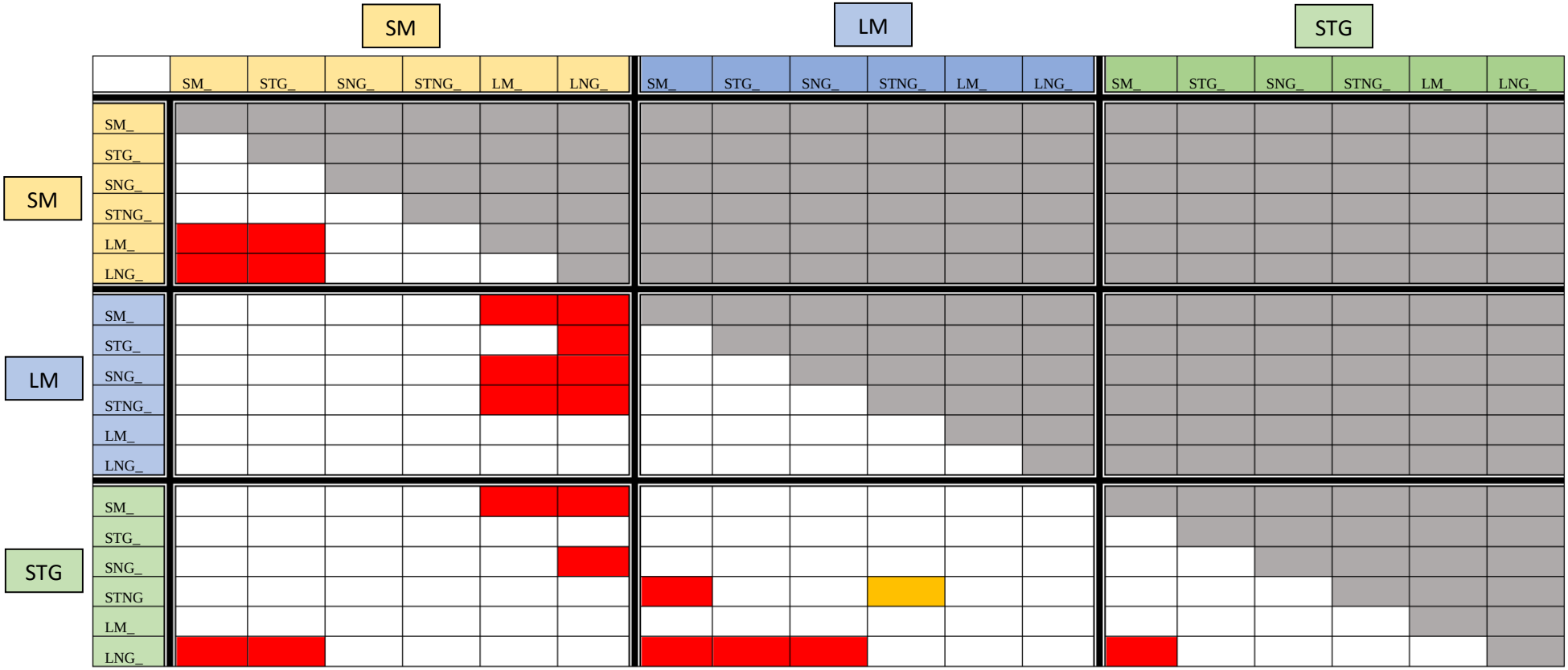


*Table 4.4: Table showing the significant differences between pairs of duplets in the short blocks of experiment 1.2. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

## 4.3 Discussion

### *4.3.1 Does prior trial timing affect RTs when temporally certain of an upcoming response?*

Temporal certainty of an upcoming response does not appear to facilitate faster RTs in general, let alone dissipate the effects of $T_{n-1}$ temporal effects. This is in contrast to findings by Berchicci et al. (2015) who found timing cues facilitated faster RTs in a GNG task.[46] This is demonstrated by STG RTs being no different from SM RTs, as seen in the short block main effect of $T_n$ type. Only a LNG $T_{n-1}$ caused significantly slower ST RTs compared to SM/ST $T_{n-1}$. Further supporting this, neither any individual $T_{n-1}$ effect on $T_n$ RTs, nor difference between short and long $T_{n-1}$ effects on $T_n$ RTs, was significantly different across SM and ST $T_n$. In figure 4.2, it appears that perhaps SM/ST $T_{n-1}$ resulted in slower ST RTs compared to SM RTs, while long $T_{n-1}$ resulted in the same ST/SM RTs. Thus, one could think that ST RTs were generally slowed, and that long $T_{n-1}$ effects being the same across SM and ST indicated that ST RTs did benefit from temporal certainty. Otherwise, we would expect Long_ST RTs to be even longer than Long_SM RTs. However LM_ST minus SM_ST RTs compared to LM_SM minus SM_SM RTs were not significantly different. This lack of significance does not support temporal certainty providing a RT advantage when there is uncertainty about the upcoming imperative type.

Generally, these results may fit with the active braking hypothesis of Jahfari et al. (2010); given the possibility of not responding, perhaps active braking slows RTs more than any RT improvements from temporal certainty. However this does not explain why a short $T_{n-1}$ resulted in quicker ST RTs compared to a long $T_{n-1}$. If a short $T_{n-1}$ *can* result in faster ST RTs than a long $T_{n-1}$, then the RT effect difference must be due to something about the timing of $T_{n-1}$ rather than general braking. Thus, to better demonstrate temporal foreknowledge RT advantages, response probability and temporal certainty RT differences may require balancing. In future experiments it may be interesting to alter the probabilities of the two time frames occurring via fixation colour, and also alter the probability of a NG being given in these trials. Finding the balancing point between temporal and response effects may allow conclusions to be drawn regarding the relative weighting and interaction of these two sources of information.

[46] This may indicate that there was some cost of switching between temporally certain and uncertain trial types, discussed in chapter seven. Alternatively, it could be because the Berchicci experiment gave a countdown timer to response, while the experiment here relied on an internal clock; perhaps there were different 'levels' of temporal 'certainty.'

*4.3.2 Does prior trial temporal certainty cause any change in current trial RTs?*

RTs in $T_n$ were not differentially affected by $T_{n-1}$ temporal certainty. In neither block did temporally certain and uncertain duplets (i.e. LTG/LM, LTNG/LNG, STG/ST, STNG/SNG) have significantly different effects on $T_n$ RTs from one another. Further, within the long block, at no point were temporally certain and uncertain long trials significantly different, irrespective of $T_n$ type. This was shown by the interaction between $T_n$ and $T_{n-1}$ type; at no $T_n$ did LT and LM $T_{n-1}$ trials have significantly different effects on RTs. In the short block, short RTs were not significantly different despite their temporal certainty. In the long block it appears that a LTG/LTNG $T_{n-1}$ resulted in faster SM RTs compared to LM/LNG trials respectively, however this difference was unlikely to be significant.[47] Overall, this data does not support the hypothesis that $T_{n-1}$ temporal certainty differentially affects $T_n$ RTs.

This finding is interesting in comparison to the relax-NG trials in Los (2013) where a relax trial was proposed to not have as large an effect on $T_n$ RTs due to trace disengagement. In this experiment, when given a temporally certain long trial, it seems reasonable to suppose that the short imperative trace would be disengaged; when given foreknowledge of an upcoming long imperative, there is no 'short imperative time,' we are just waiting for the long imperative. Thus, a LTG/LTNG $T_{n-1}$ should induce significantly faster SM RTs compared to a LM/LNG $T_{n-1}$. However, as mentioned above, this was not the case. Though a SM_SM may have faster RTs than a LT_SM due to stimulus repetition (for example Adam and Koch, 2009; Schulz et al., 2007; Soetens, 1998; Vervaeck and Boer, 1980), or trace reinforcement (as discussed in Los, 2013; Capizzi et al., 2015),[48] LM/LTG and LNG/LTNG should still have caused different $T_n$ RTs. This could indicate that, in the case where there is temporal but not response certainty, traces are not disengaged.[49] However, in figure 4.1 above, there does appear to be a trend for LT $T_{n-1}$ to result in faster SM RTs than a LM/LNG $T_{n-1}$, possibly warranting further investigation.

[47] Though may be tested in future.

[48] Alternatively, it may be that the temporal or response aspects are selectively reinforced/extinguished. This will be discussed further in chapter seven.

[49] This could mean that perhaps the cost of trace disengagement and reengagement is too high when uncertain of an upcoming response; response uncertainty does not allow the 'switching off' of a trace. Alternatively, perhaps traces are either 'reinforced,' 'extinguished' or 'not affected,' without there being levels of 'reinforcement' for example. However further research is required before commenting on this.

## 5. Experiment 1.3

In this experiment, a white fixation indicated a go or NG could occur at either the short or long imperative time, while a green fixation indicated a go imperative would definitely appear at only the short or long time, depending on the block. SM, LM, SNG, LNG, and either SD or LD were equiprobable. This experiment tests the hypothesis that SD and LD RTs are unreactive to $T_{n-1}$ trial type.

### 5.1 Visualization of Results

Figures 5.1 and 5.2 show the mean RTs across the long blocks (i.e. a green fixation indicated a LD) and short blocks respectively.

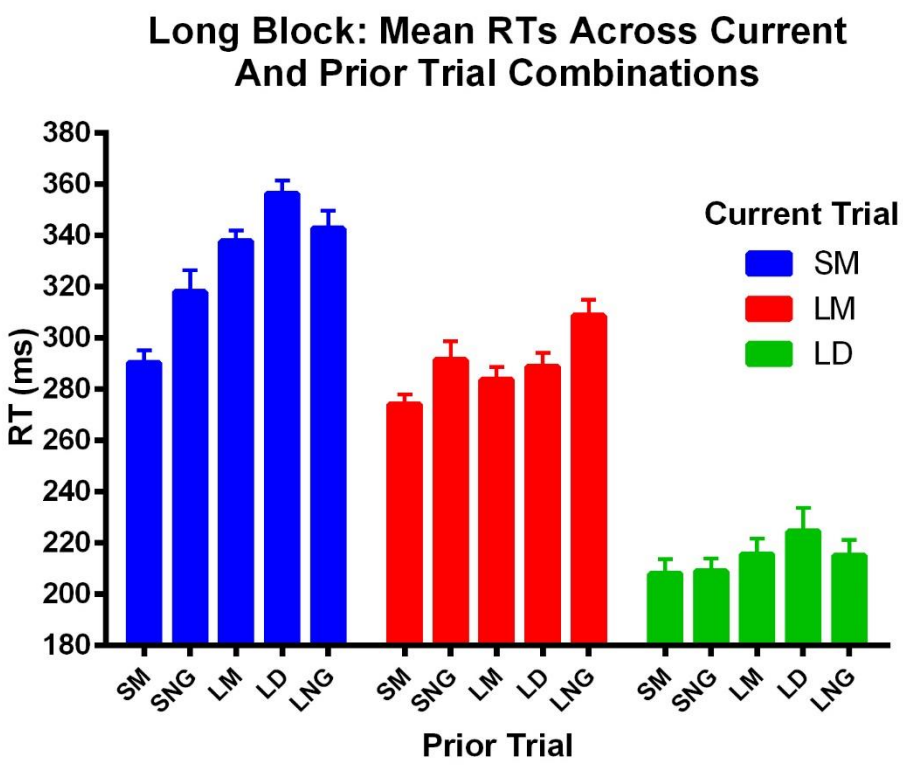


*Figure 5.1: Graphical representation of RTs in the long block, where a green fixation indicated a long definite go would occur. Each column represents the prior trial, while each group represents the current trial. In this block, the SM and LM $T_n$ RT patterns are the same as presented in Los (2013); a SNG resulted in slower SM RTs than a SM, while long trials resulted in the slowest SM RTs. LM RTs are slowest when preceded by a LNG $T_{n-1}$, though again a SNG $T_{n-1}$ appears to slow LM RTs compared to a SM $T_{n-1}$. LD RTs appear relatively stable irrespective of $T_{n-1}$ events, though LD RTs appear slower when preceded by a LD $T_{n-1}$ than when preceded by a short$T_{n-1}$.*

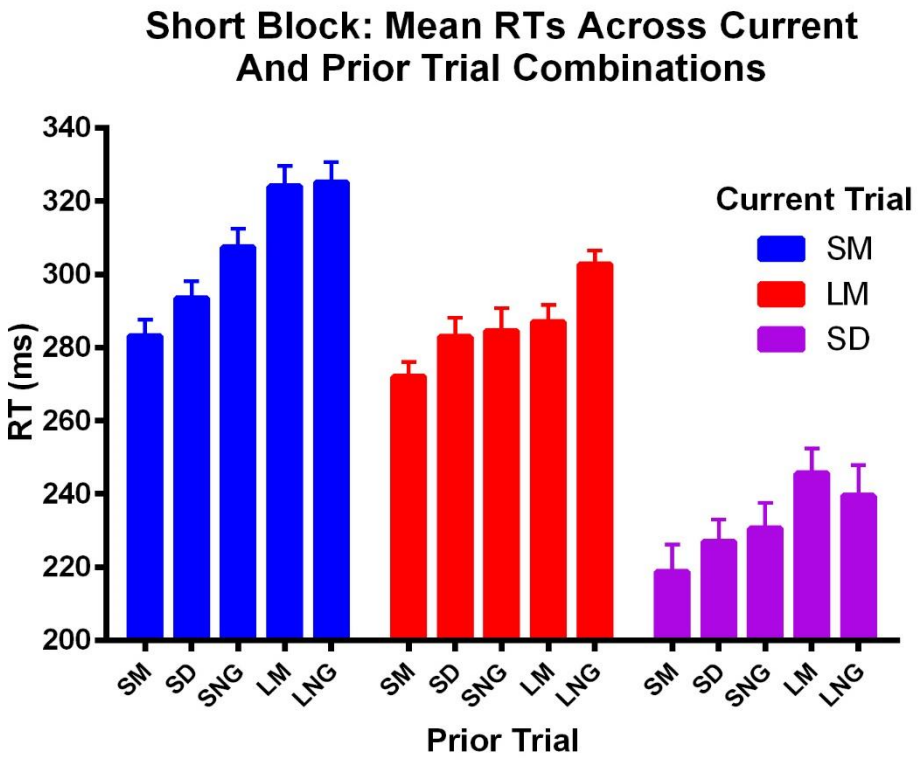


*Figure 5.2: Graphical representation of RTs in short block, where a green fixation indicates a short definite go. The SM and LM $T_n$ appear to follow the same pattern as in figure 5.1; specifically SM RTs are fastest given a SG $T_{n-1}$, then a SNG $T_{n-1}$ then a LG $T_{n-1}$. LM RTs are slower when given a LNG $T_{n-1}$ compared to other $T_{n-1}$. SD RTs appear slower when given a long $T_{n-1}$ compared to a short $T_{n-1}$, despite precise information regarding imperative timing.*

In the long block, LD RTs (figure 5.1, right) appear relatively stable irrespective of $T_{n-1}$ events, though a LD $T_{n-1}$ may slow RTs compared to short $T_{n-1}$ trials. In the short block (figure 5.2), a SD $T_n$ also appears to attenuate the effect of $T_{n-1}$ trial type; SD RTs appear less slowed by a LM $T_{n-1}$ than SM RTs.

## 5.2 Results

Mean RTs per participant varied from 241-338ms in the long block (mean=278ms, SD=62ms), and 239-326ms in the short block (mean=274ms, SD=48ms). The number of mistakes varied from 25 (1.6% of all trials) to 207 (12.9% of all trials). Of these, few were incorrect responses to a NG stimulus. For example, the person with the greatest number of mistakes responded incorrectly to four imperatives (0.3% of all trials). Most mistakes were early responses. Compared to experiment 1.1, there were less responses at the SD imperative time when no imperative was presented, though this still occurred occasionally (for the participant with the most mistakes this occurred 12 times, 0.8% of all trials). There was also

the occasional response at the beginning of a trial when a green fixation appeared (for the participant with the most mistakes this occurred 8 times, 0.5% of all trials).

Before analysing individual blocks, differences between the blocks were investigated. To do this all green fixation RTs (to $T_{n-1}$) were removed, meaning the two blocks contained only the same trial types (i.e. SM, SNG, LM, LNG). A paired t-test indicated a significant difference between the blocks ($t(119)=4.07$, $p<0.0001$). LM RTs were not different between the blocks ($p>0.3$), but SM RTs were ($p>0.0001$, long block RTs were ≈12ms slower than short block RTs).

To analyse within each block, mutually exclusivity[50] was removed by collapsing into two variables; $T_{n-1}$ trials were split into SM, SNG, LM and LNG plus one of either LD or SD, and $T_n$ trials were split into SM, LM and either LD or SD. For both the long and short block the interaction between $T_n$ and $T_{n-1}$ trials was significant, $F(8,112)=10.96$, $p<0.0001$, $\eta_p^2=0.05$; $F(8,112)=3.28$, sphericity-corrected $p<0.05$, $\eta_p^2=0.03$, respectively.[51]

To better understand these interactions, the effects of each $T_{n-1}$ trial on each $T_n$ trial RT was compared within each block (i.e. how the five possible $T_{n-1}$ types affected each $T_n$ RT). Within the long block, SM RTs (figure 5.1, left) were not significantly different when preceded by any long $T_{n-1}$ compared to other long $T_{n-1}$ (all $p>0.9$). SM RTs were significantly slower for any long $T_{n-1}$ compared to a SG $T_{n-1}$ (all $p<0.001$). A SNG $T_{n-1}$ resulted in significantly faster $T_n$ RTs compared to LD and LNG $T_{n-1}$ ($p<0.05$ and $p<0.01$ respectively), though not significantly different compared to SM or LM $T_{n-1}$ ($p\approx1$). However, on average, SM_SM RTs were ≈28ms faster than SNG_SM RTs. LM RTs, (figure 5.1, middle) appeared unaffected by $T_{n-1}$ except when comparing the effects of a SM and LNG $T_{n-1}$ ($p<0.05$). Despite this, when given a LNG $T_{n-1}$, RTs were ≈19ms slower in a LM $T_n$, compared to the next-slowest LM RT (i.e. a SNG $T_{n-1}$). Finally, LD RTs (figure 5.1, right) did not significantly change depending on $T_{n-1}$ (all $p\approx1$).

Within the short block, SM RTs (figure 5.2, left) were not significantly different when comparing between long $T_{n-1}$ ($p\approx1$). A SM/SD $T_{n-1}$ caused significantly faster SM RTs compared to all long $T_{n-1}$ trials (all $p<0.005$, except when comparing the effects of a SD $T_{n-1}$ to LNG $T_{n-1}$, which was $p<0.05$). SD and SM $T_{n-1}$ did not result in significantly different SM RTs ($p\approx1$). A SNG $T_{n-1}$ resulted in significantly different SM RTs compared to LNG ($p<0.01$) and

[50] A short $T_n$ never co-occurred with a definite-go in the long block for example.
[51] Graphing these interactions is redundant; it looks like figures 5.1 and 5.2.

SM $T_{n-1}$ ($p<0.05$) however a SNG $T_{n-1}$ was not significantly different from SD and LM $T_{n-1}$ (all $p\approx1$). LM RTs (figure 5.2, middle) were not significantly different when preceded by either long $T_{n-1}$, or by a SNG $T_{n-1}$ compared to any other $T_{n-1}$ type (all $p\approx1$, except comparing LNG to SNG $T_{n-1}$, $p=0.18$). LM RTs were significantly shorter when preceded by a SD/SM $T_{n-1}$ compared to a LNG $T_{n-1}$ (both $p<0.01$). Within SD trials (figure 4.2, right), LM/LNG $T_{n-1}$ did not cause significantly different RTs in $T_n$ ($p\approx1$). A LM $T_{n-1}$ resulted in significantly slower SD RTs compared to SD and SM $T_{n-1}$ (both $p<0.01$). LNG $T_{n-1}$ effects on SD RTs were not significantly different from SD and SM $T_{n-1}$ ($p\approx0.3$, $p\approx1$ respectively). A SNG $T_{n-1}$ did not cause significantly different SD RTs compared to any other $T_{n-1}$ ($p\approx1$). Comparing the difference between LM_SM and SM_SM RTs, and LM_SD and SM_SD RTs, SD RTs were significantly less affected by LM $T_{n-1}$ compared to SM RTs ($p<0.01$, ≈14ms less difference between SM and LM $T_{n-1}$ effects).

The tables on the following pages represents significant differences between individual duplets in the long and short blocks respectively.

**Table 5.1: Significant differences between individual duplets in the long blocks of experiment 1.3**

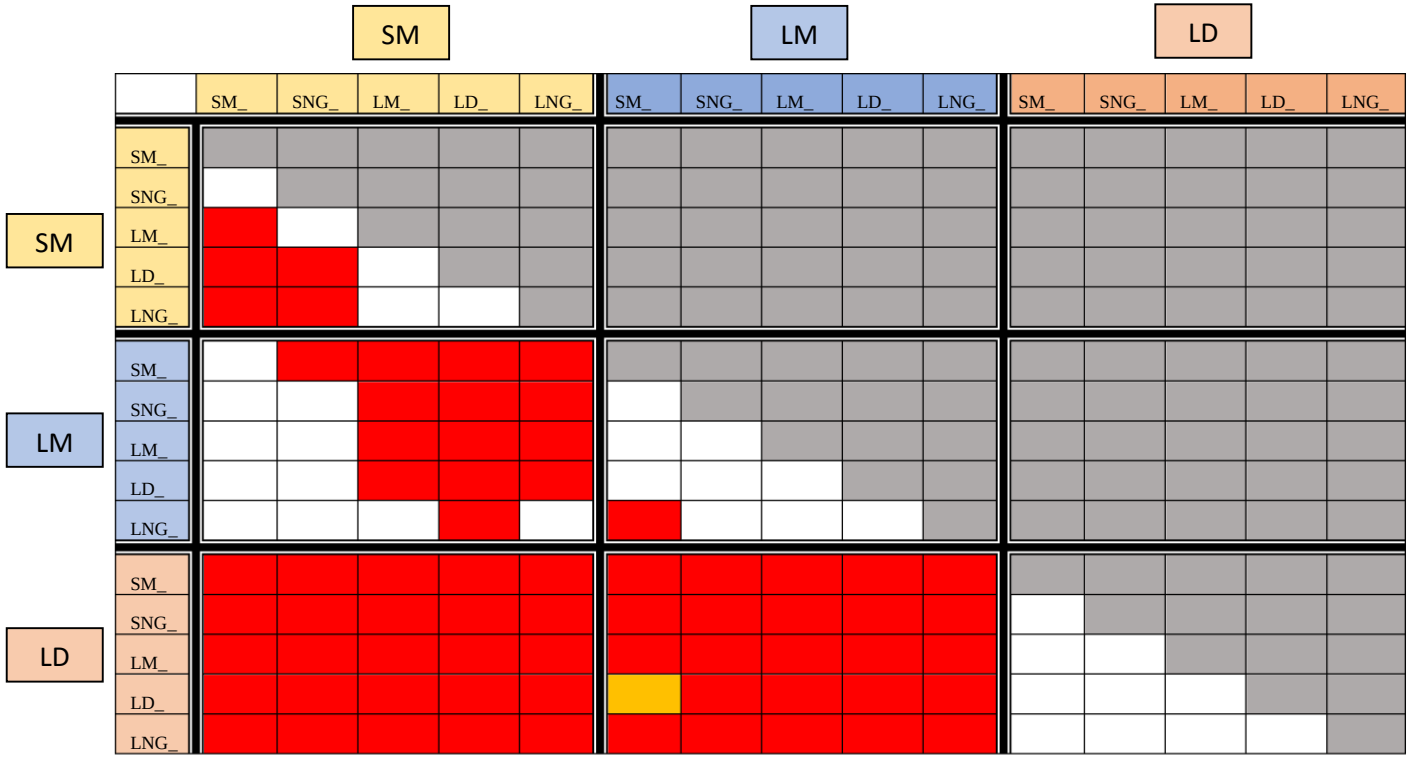


*Table 5.1: Table showing the significant differences between pairs of duplets in the long blocks of experiment 1.3. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

**Table 5.2: Significant differences between individual duplets in the short blocks of experiment 1.3**

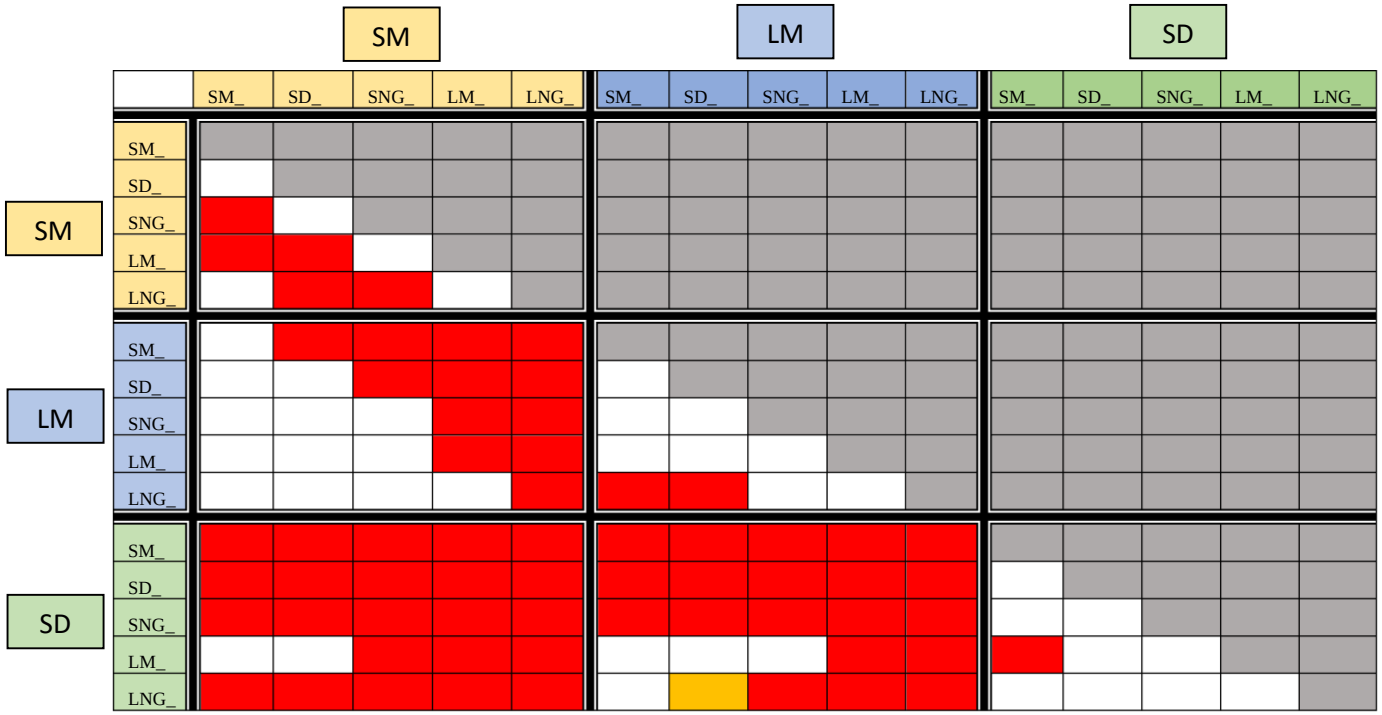


*Table 5.2: Table showing the significant differences between pairs of duplets in the short blocks of experiment 1.3. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

## 5.3 Discussion

### *5.3.1 Does perfect foreknowledge in the current trial reduce the effect of prior trial dynamics?*

Perfect foreknowledge was given in the LD and SD trials of the long and short blocks respectively, and therefore these cases are the focus of this discussion. Within LD/SD $T_n$ trials there was a general reduction of $T_{n-1}$ effects on $T_n$ RTs, an effect that is congruent with the hypothesis that current, explicit knowledge supersedes prior, implicit information (as per Adam and Koch, 2009). In experiment 1.1, LD RTs were similarly unaffected by $T_{n-1}$. In this experiment, there was also a reduction in $T_{n-1}$ effects on SD $T_n$ trials, demonstrated by a reduced effect of a LM $T_{n-1}$ compared to other $T_{n-1}$ on SD RTs. Importantly, this reduction of $T_{n-1}$ effect from SM to SD $T_n$ was not present when response certainty *only* was provided (i.e. experiment 1.1, $p>0.5$, difference≈2.5ms). Despite this reduction, in the current experiment SD RTs were still significantly slower when $T_{n-1}$ was a LM compared to when it was a SM/SD. This may indicate some effect of a psychological refractory period, or a switching cost.[52] However, due to the reduced effects of $T_{n-1}$ temporal dynamics in this experiment compared to experiment 1.1, the findings here indicate that psychological refraction and switching cannot fully account for the RT asymmetries between SM and SD trials in experiment 1.1. Alternatively, perhaps this indicates that part of $T_{n-1}$ temporal effects cannot be overridden despite $T_n$ foreknowledge.

[52] This is equally true of the prior two experiments, and will be discussed in chapter seven.

## 6. Experiment 2

In this experiment, a go or NG could occur at either the short or long time point. SM, LM, SNG, and LNG trials were equiprobable. This experiment examined electrophysiological ramifications of $T_{n-1}$ and $T_n$ dynamics. Analysis was performed on both the normalised MEP ratio (nMEP-R) data (i.e. MEP epoch area divided by pre-imperative epoch area) and on the EMG levels in the pre-imperative epoch.

### 6.1 Visualization of Results

Figure 6.1 demonstrates mean nMEP-Rs over $T_{n-1}$-$T_n$ combinations.

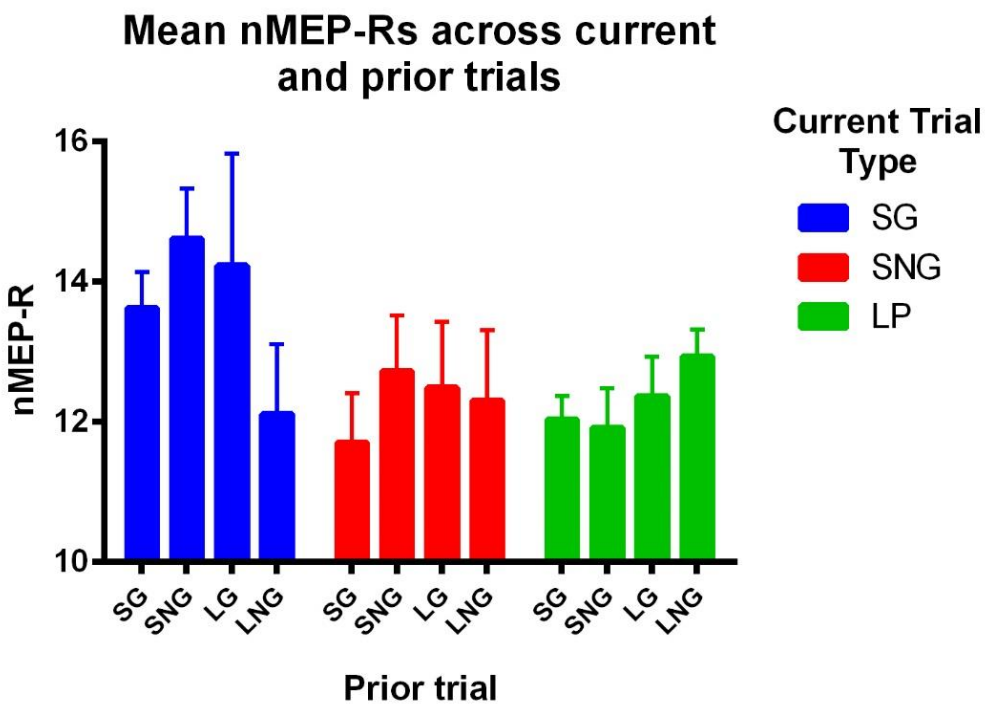


*Figure 6.1: Mean peak-to-peak nMEP-R values given different trial combinations. Each bar indicates a prior-current trial combination. SG $T_n$ nMEP-Rs appear larger than in either SNG or LP $T_n$. Further, given a SG $T_{n-1}$, both SG and SNG $T_n$ nMEP-Rs are not as large as when given a SNG or LG $T_{n-1}$. A LNG $T_{n-1}$ appears to induce smaller nMEP-Rs in a SG $T_n$, while in a SNG and LP $T_n$, LNG $T_{n-1}$ does not appear to reduce nMEP-Rs.*

Firstly, nMEP-R standard errors appeared fairly large, more so on the short than long trials. This may be due to a fairly small number of trials[53] and inconsistent levels of pre-imperative muscle activation. Overall, a SG in $T_n$ appears to elicit the largest nMEP-Rs, except when it is preceded by a LNG in $T_{n-1}$. While a SNG in $T_{n-1}$ induced the largest $T_n$ nMEP-Rs in

[53] Hence why LP nMEP-Rs are less variable; they contained roughly double the number of trials, including LNG and LG $T_n$ data.

both the SG and SNG $T_n$, a LNG in $T_{n-1}$ resulted in the largest LP nMEP-Rs. For short $T_n$ trials, both a SNG and LG $T_{n-1}$ induced larger nMEP-Rs than a SG. These $T_{n-1}$ trial types are predicted to induce slower RTs in a short $T_n$ according to trace conditioning. Generally, SG nMEP-Rs appear more reactive to $T_{n-1}$ dynamics than SNG and LP nMEP-Rs, which are relatively unaffected.

## 6.2 Results

Mean RTs for TMS trials ranged from 328.6-410.1ms (mean=368.3ms, SD=80.2ms). For non-TMS trials, RTs ranged from 314.9-410.7ms (mean=374.9ms, SD=70.3ms).[54] RTs were significantly faster in TMS than non-TMS trials, paired t-test, $t(71)=2.10$, $p<0.05$. SM RT patterns were similar to those described by Los (2013); a SNG and long $T_{n-1}$ slowed SM RTs (though a SNG/LNG did not noticeably change RTs compared to a SG in TMS trials). However, LM RTs appeared longer if preceded by a LM $T_{n-1}$ compared to a short $T_{n-1}$, and furthermore, a LNG $T_{n-1}$ did not lengthen LG RTs. 4.1% of trials were removed for errors (SD=12).[55] MEP thresholds ranged from 42-67% of stimulator output.

In the pre-imperative epoch, $T_{n-1}$ response type had a significant effect, $F(1,8)=10.64$, $p<0.05$, $\eta_p^2=0.57$ on EMG activity. This was such that, when $T_{n-1}$ was a NG, the 50ms prestimulus epoch had an area of 6.18mVms$^{-1}$ (SD=1.08 mVms$^{-1}$), while following a go in $T_{n-1}$ it had an area of 7.48 mVms$^{-1}$ (SD=1.29 mVms$^{-1}$). Therefore, EMG activity was ≈1.30 mVms$^{-1}$ larger when given a go compared to NG $T_{n-1}$. Variation of pre-movement contraction levels may have masked/generated MEP interactions if not normalised for. Timing of $T_{n-1}$, $F(1,8)=3.97$, $p=0.08$, $\eta_p^2=0.33$, and the interaction between timing and response type of $T_{n-1}$, $F(1,8)=3.56$, $p=0.096$, $\eta_p^2=0.31$, both approached significance. No effect involving $T_n$ type approached significance.[56]

[54] A slow responding subject without TMS was also slow to respond with TMS.
[55] an error in $T_{n-1}$ also removed $T_n$, therefore actual mistakes were half this.
[56] This makes sense; the $T_n$ imperative was yet to be determined.

In the MEP epoch, there was a main effect of $T_n$ type, $F(2,16)=4.12$, $p<0.05$, $\eta_p^2=0.23$. This main effect demonstrated that nMEP-Rs were larger given a SG compared to either a SNG or LP $T_n$. Overall, SG nMEP-Rs were larger than SNG and LP by ≈1.3 (ratio of MEP to prestimulus EMG) for both ($p=0.016$ and $p=0.042$ respectively). nMEP-Rs were not significantly different between SNG and LP trials (difference<0.01, p≈1). This main effect was superseded by the interaction between $T_n$ type and $T_{n-1}$ timing ($F(2,16)=5.32$, $p<0.05$, $\eta_p^2=0.29$). The interaction between $T_n$ type and $T_{n-1}$ timing is shown in figure 4.2 below.

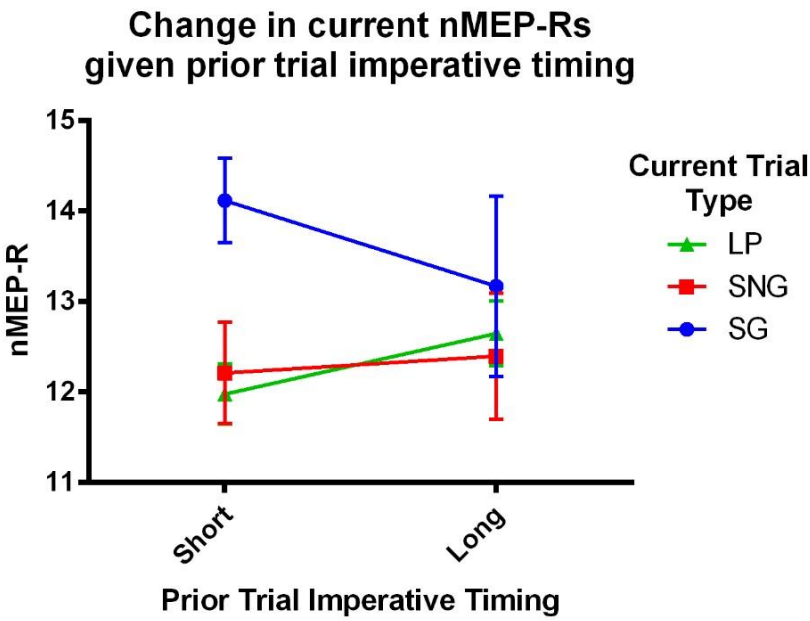


Figure 6.2: Interaction between prior trial timing and current trial type. Each line represents a separate current trial type. SG $T_n$ nMEP-Rs appear larger than both SNG and LP $T_n$ nMEP-Rs. While SNG $T_n$ nMEP-Rs appear similar given either $T_{n-1}$ timing, SG $T_n$ nMEP-Rs decreased given a longer $T_{n-1}$ and LP $T_n$ nMEP-Rs increased over the same time.

When the imperative was at the long time point in $T_{n-1}$, nMEP-Rs were not significantly affected by $T_n$ type (all p≈1). When the imperative was at the short time point in $T_{n-1}$, nMEP-Rs were significantly larger given a SG rather than a LP in $T_n$ (difference=2.2, $p=0.011$), while the difference between a SG and SNG $T_n$ was not (difference=1.9, $p=0.283$). Furthermore, though changes in nMEP-Rs were not significant from a short to long $T_{n-1}$ (e.g. SG nMEP-R changes between a short and long $T_{n-1}$ were not significant), when given a long $T_{n-1}$, SG $T_n$ nMEP-Rs decreased compared to a short $T_{n-1}$ by an average of ≈1.0,[57] a SNG $T_n$ increased by ≈0.2 and a LP increased by ≈0.7. When examining this difference (subtracting the long $T_{n-1}$ nMEP-Rs from the short $T_{n-1}$ nMEP-Rs) SG and LP changes from short to long $T_{n-1}$ were significantly different ($p=0.036$).

[57] This is likely driven by the large drop when given a LNG $T_{n-1}$, as seen in figure 3.1.

## 6.3 Discussion

### *6.3.1 Main effect of current trial type*

Though this main effect was superseded by the interaction discussed below, it is worth examining on its own. This is because most prior studies of CSE (e.g. Coxon et al., 2006, Jahfari et al., 2010; Leocani et al., 2000; Van Elswijk et al., 2007; van den Wildenberg et al., 2010) have focused on $T_n$ dynamics. Therefore this finding facilitates comparison with prior literature.

nMEP-Rs were significantly larger given a go imperative at the short time point (100ms prior to TMS pulse) than when given no signal (LP), or a NG imperative. This is in line with prior research that found imperative type (go versus NG) may differentially affect CSE even within 100ms of presentation (Hoshiyama et al., 1997), however is earlier than found in other studies (e.g. Burle et al., 2002; Chen, Yaseen, Cohen, and Hallett, 1998; Leocani et al., 2000; McMillan, Nougier, and Byblow, 2004; Pascual-Leone et al., 1992). For example Leocani et al. (2000) found MEPs increased only within an epoch 120ms prior to movement. The quickest RT in this paradigm was ≈125ms post-TMS timing, and the average was ≈268ms after the TMS pulse, indicating that nMEP-Rs increased earlier that 120ms prior to movement in this experiment. However, Hoshiyama used temporal certainty, while Leocani used a VF.[58] Though the current experiment also utilised a VF, here there were only two possible imperative times, and therefore this experiment is probably more comparable to the Hoshiyama study; it is likely that nMEP-Rs decrease earlier than in Leocani et al. (2000) due to temporal certainty. Thus it may be that, given direct mapping of signal to response (i.e. simple cueing (see Mostofsky and Simmonds, 2008; Simmonds et al., 2008; Verbruggen and Logan, 2008a) and reduced temporal uncertainty (Davranche et al., 2007; Duque et al., 2010; Kennefick, Maslovat, & Carlsen, 2014; Tandonnet et al., 2012; Van Elswijk et al., 2007), there is earlier signal analysis resulting in signal-specific nMEP-R effects in this experiment. If this result is considered under active braking (Jahfari et al., 2010), it may be more likely that the active response is released from inhibition, while the non-active (i.e. inhibited) response is withheld. However, given that inhibition is often cited as being applied to M1 at the time when a response is normally initiated (Coxon et al., 2006; Sohn et al., 2002; van den Wildenberg et al., 2010), this requires further

[58] In this case the VF was varied evenly across a two second period. Further Leocani used auditory signalling while Hoshiyama used visual signalling, however auditory and visual RTs are similar in GNG tasks (Falkenstein, Hoormann, & Hohnsbein, 1999; Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995). Auditory RTs may be even faster than visual RTs in SRT tasks however (Shelton & Kumar, 2010).

investigation, specifically utilising paired-pulse TMS, or silent period analysis (see for example van den Wildenberg et al., 2010).

It is interesting to note that both SNG and LP had similar nMEP-Rs. These two situations are also proposed to drive inhibition-induced trace extinction (Los, 2013; Los et al., 2014; Los & van den Heuvel, 2001) by virtue of the widespread effects of motor actions (see Los (2013) for discussion). Specifically, given that motor actions can affect information processing (Hommel, 2004, 2009; Prinz, 1990; Shin et al., 2010; Steinhauser & Hübner, 2006),[59] an effect on 'traces' is not a far-fetched idea. The reduction of nMEP-Rs in 'inhibited' situations in this experiment further supports the inhibition-driven trace reduction proposition. This does not necessitate that CSE reduction drives trace extinction (or CSE increases drive trace reinforcement), however it may indicate a CSE correlate of the function driving both CSE and trace reduction. A logical source for this driver is inhibition; proposed to underlie both CSE and trace suppression (Los & van den Heuvel, 2001; Sinclair & Hammond, 2009).

### *6.3.2 Interaction between timing of prior trial and current trial type*

The interaction between $T_{n-1}$ timing and $T_n$ type demonstrated that, when a SG occurred in $T_{n-1}$, SG nMEP-Rs were significantly larger than LP nMEP-Rs. Though SG and SNG nMEP-Rs were not significantly different, as discussed above, nMEP-Rs on average were larger for a SG than a SNG. When $T_{n-1}$ was long, nMEP-Rs were not significantly different between a LP, SG, and SNG $T_n$. Comparing nMEP-R changes from a short $T_{n-1}$ to a long $T_{n-1}$, SG nMEP-Rs decreased, SNG nMEP-Rs increased marginally, and LP nMEP-Rs increased. No $T_n$ nMEP-R change over $T_{n-1}$ timing was significant within the $T_n$ types, however the difference in this change was significant between the SG and LP nMEP-Rs.

However, though not significantly different from any other $T_{n-1}$ trial type, the main difference driving the long $T_{n-1}$ reduction of SG nMEP-R appears to be the LNG; A LG $T_{n-1}$ appears to have roughly the same nMEP-R as a SNG $T_{n-1}$. In figure 6.1 this can be seen in the left-most group; nMEP-Rs following SNG and LG $T_{n-1}$ appear to be increased to a similar extent given a SG or SNG $T_n$. However, when $T_{n-1}$ was a LNG, in the SG $T_n$ there was a nMEP-R reduction of ≈2.1 from the LG $T_{n-1}$, while in the SNG $T_n$ this reduction was only ≈0.2.

[59] Though, as discussed by Los (2013), the trace-conditioning model does not propose how traces affect $T_n$ RTs, it may be perceptual- or motor-based.

It seems likely that response inhibition at the long $T_{n-1}$ resulted in residual effects on short $T_n$ nMEP-Rs. This drop may be attenuated in the SNG nMEP-Rs due to response suppression in $T_n$. The difference in LNG $T_{n-1}$ effects on $T_n$ nMEP-Rs may thus account for $T_{n-1}$ timing interacting with $T_n$ type. As will be discussed in chapter seven, this may be due to the psychological refractory period, indicating that, perhaps, given longer inter-trial breaks, $T_{n-1}$ effects would be absent. However, if normalising in a more appropriate way (below), it still seems likely that $T_{n-1}$ response type (at least) would have a significant effect on MEP. Furthermore, LNG $T_{n-1}$-driven SG $T_n$ nMEP-R changes do not account for why a SG, SNG and LP have roughly the same nMEP-Rs following a LNG in $T_{n-1}$; does a LNG $T_{n-1}$ have an effect irrespective of $T_n$ timing? Finally, this does not explain why LP nMEP-Rs were larger in the short compared to the long $T_{n-1}$. As temporal expectation may modulate MEPs (Davranche et al., 2007; Duque et al., 2010; Kennefick et al., 2014; Tandonnet et al., 2012; Van Elswijk et al., 2007), perhaps equivalent nMEP-Rs following a LNG $T_{n-1}$ indicate an uniform, overriding, effect on $T_n$ imperative timing expectations.

### *6.3.3 Is there a reason to include prior trial effects on future TMS experiments?*

There appears ample reason to further investigate sequential MEP effects. Firstly, this experiment revealed a significant interaction between $T_{n-1}$ timing and $T_n$ trial type. This indicates that if there is more than one trial time in an experiment then, even when normalising MEPs against a baseline earlier in the trial (which assumedly would decrease the $T_{n-1}$ effects),[60] a sequential effect remains. This may not be an issue for all experiments, for example those which utilise one predictable time point (e.g. Hoshiyama et al., 1997). However given that temporal and response dynamics can alter CSE (e.g. Jahfari et al., 2010; Kennefick et al., 2014; Tandonnet, Garry, and Summers, 2010; Van Elswijk et al., 2007) and that responses can be altered by both future expectations (e.g. Thomas, Gonsalvez, and Johnstone, 2009) and prior events (e.g. Los, 2013; Vallesi, Shallice, and Walsh, 2007), then controlling for these inter-trial effects may be appropriate.

Though most studies employ MEP normalisation, the sequential effect finding of this study may indicate an issue with normalisation technique. For example, a common technique

[60] Given that baseline levels of CSE affect nMEP-R amplitudes, as demonstrated explicitly through transcranial direct current stimulation studies (Mordillo-Mateos et al., 2012; Nitsche & Paulus, 2000), the effects of $T_{n-1}$ response dynamics, shown to affect baseline EMGs, also require consideration in future studies utilising TMS in GNG tasks.

is to normalise data against some control condition (e.g. Hoshiyama et al., 1997; Jahfari et al., 2010; Kinoshita et al., 2007; Leocani et al., 2000; Marinovic et al., 2011). However, if $T_{n-1}$ affects $T_n$ MEPs, then normalising data with a baseline without considering $T_{n-1}$ effects may aggregate MEPs around a mean value (based on random $T_{n-1}$), therefore missing a possibly important predictor of MEP variability. In future renditions of this experiment, I wish to set up such a baseline recording with which to normalise sequential MEPs to, however include $T_{n-1}$ dynamics as factors in the normalised MEP analysis. This may allow better characterisation of sequential effects. As can be seen in the analysis of pre-imperative EMG data in this experiment, $T_{n-1}$ response had a significant effect on EMG levels, which may have been present in MEPs if not normalised out.[61] Another normalisation procedure is the conversion of MEPS to Z-scores based on mean and SD over all trials (e.g. Davranche et al., 2007;, Tandonnet et al., 2012; van den Wildenberg et al., 2010; van Elswijk, Schot, Stegeman, and Overeem, 2008). Again however, this does not control for $T_{n-1}$ variability.

Furthering the argument for extending the current experiment, the number of participants here was low. Running more participants may have revealed further interactions. To demonstrate this, I ran the above anova without collapsing $T_n$ into type (i.e. including SG, SNG, LG and LNG). This revealed that the interaction between $T_n$ timing and response, and $T_{n-1}$ response approached significance. I mention this not because I believe that long $T_n$ responses may retrospectively alter MEPs; MEPs *are* interesting, but not 'time-travel' interesting. Instead this interaction likely relates to having more cells representing $T_{n-1}$ (i.e. four data points per participant rather than three). Regardless of current results, these are good reasons to expand the number of participants/trials in future investigations of $T_{n-1}$ effects on $T_n$ MEPs.

The current findings seem to indicate that sequential MEP analysis may be a worthwhile endeavour. In future, various TMS protocols at various intra-trial time points may reveal, for example, when $T_{n-1}$ timing information is incorporated into $T_n$ (is it present at the beginning of the trial or only when a possible response approaches?), how $T_{n-1}$ timing interacts with $T_n$ timing (is information integrated at the long imperative time as well, or is it specific to an uncertain (i.e. short) time point?) and if sICI varies with $T_{n-1}$ timing, is greater sICI required to stop a response when a response was released in $T_{n-1}$? All these questions are interesting in

[61] In fact, initially I had run my analysis on pure MEP data, which demonstrated an effect of $T_{n-1}$ response on MEP amplitude. This reflected that the data was non-normalized, however indicates if using a different normalization method, these effects may have had consequences for nMEP-Rs.

terms of how $T_{n-1}$ timing interacts with $T_n$ timing. For example, when given a SNG, are levels of sICI dependant on the expectation of a short $T_n$; does increased temporal expectancy induced by $T_{n-1}$ timing increase the inhibitory load on $T_n$ response inhibition?

## 7. General Discussion

In this chapter I will discuss the findings of the prior experiments, relating these to the trace-conditioning model and inhibitory control. I will then propose how the current research can be improved, and some future directions for research on this topic.

### 7.1 When Should the Horse Cross the Road?

The current research into sequential foreperiod effects provides an explanation for how $T_{n-1}$ information affects $T_n$ RTs given varying levels of temporal and/or response foreknowledge. As per Los (2013), when the timing and response type of an upcoming imperative is uncertain, RTs are slowed if the foreperiod in the current trial is shorter than that of the previous trial. Furthermore, a NG in $T_{n-1}$ at the same time as the imperative in an uncertain $T_n$ similarly slows RTs. An important addition that the current study makes is the finding that foreknowledge regarding the type of an upcoming imperative (whether it will be a go or NG) reduces the effects of a NG in $T_{n-1}$ on $T_n$ RTs but does not affect the temporal trace effects of $T_{n-1}$.

Moreover, temporal foreknowledge (knowing whether the response imperative will be at the long or short time) does not affect the temporal trace effects of $T_{n-1}$ on $T_n$; $T_{n-1}$ foreperiod length still affects $T_n$ RTs when the timing of the upcoming imperative is explicitly signalled. Significantly though, when both response type and temporal foreknowledge are provided together, the effects of both $T_{n-1}$ response timing and $T_{n-1}$ response type are reduced.

In terms of CSE, a $T_{n-1}$ NG response resulted in smaller $T_n$ pre-imperative EMG levels. SG nMEP-Rs at the short imperative time were also relatively larger when following a short $T_{n-1}$ compared to a long $T_{n-1}$, while LP nMEP-Rs were smaller. The findings of these experiments may indicate a hierarchy of $T_{n-1}$ effects on RTs; response type information is easier to 'override' than temporal information, which requires both temporal and response type certainty to overcome.[62] What does this tell us about how we ready a response given temporal and response (un)certainty?

[62] To put this another way, foreknowledge of response type allows overriding of temporal information when given temporal certainty, while temporal certainty without response type certainty does not.

*7.1.1 Now and then* – According to the trace-conditioning model, traces are extinguished when an imperative moment is either passed without an imperative being given, or a response is actively inhibited due to a NG signal (Los, 2013; Los & van den Heuvel, 2001).[63] If both temporal (proactive) and response (reactive) inhibition result in the same effect on a trace, and the origins of these are indistinguishable once they have occurred, then it should not be possible to selectively attenuate trace effects associated with a response when given foreknowledge of an upcoming imperative type. However, when comparing effects on RTs from $T_{n-1}$ on an SD $T_n$ in experiment 1.1 (response but not temporal certainty), a long $T_{n-1}$ resulted in significantly slower SD RTs than did a short $T_{n-1}$, while a SNG $T_{n-1}$ (nor a LNG compared to SD $T_{n-1}$) did not significantly slow SD RTs compared to a short $T_{n-1}$. Response type information appeared selectively attenuated. As shown in experiment 1.3, it is possible to reduce the effects of both temporal and response information; it is not an intrinsic property of the long imperative response being in close proximity to the short imperative that increases RTs.[64]

The simplest implication of this finding is that a 'trace' may not be a unified entity, influenced equally by both temporal and response dynamics. Even if the passing of an imperative moment and active response inhibition of a NG imperative both have similar effects on subsequent trial RTs, if the cause of inhibition is lost/unknown, then it should be impossible to 'stream' only the relevant information in $T_n$. One could propose that, like light passing through coloured glass, only certain 'wavelengths' of information are allowed into the $T_n$ construction of response expectancy. However, in order for wavelengths to be selectively absorbed, there must be more than one wavelength of light. Similarly, selective information use from $T_{n-1}$ requires a separation of response and temporal effects. Further, the attenuation of $T_{n-1}$ temporal effects appears to require temporal *and* response certainty, as found in experiment 1.2 and 1.3, supporting some type of differential effect of the two sources of $T_{n-1}$ information. Due to the response foreknowledge requirement for temporal attenuation, there may also be some interaction between temporal- and response-based sources of information. Alternatively, perhaps temporal effects are 'hard-wired' into $T_n$ while response-based effects are 'soft-wired,'

[63] Though it may be possible that the application of response inhibition to prior time points occurs during 'trial evaluation' (see electroencephalographic studies, e.g. Smith, Johnstone, and Barry, 2007; Kropotov et al., 2011).
[64] It is possible that the attenuation of both response and temporal aspects of the prior trial in experiment 2.3 result from the perfect foreknowledge trial resulting in disengagement from the prior traces, however this does not explain why a LD_SM and a LM_SM have the same RTs. If the trace was totally disengaged then the prior LD should not affect SM RTs as greatly as other long trials. This contention is supported by Los (2013) who found that a relaxed NG (i.e. perfect foreknowledge of a NG) did not result in as much $T_n$ RT effect, due to the trial being disengaged.

allowing easier adjustment/overriding. Or, there may be two separate 'traces,' which are entirely separate devices. Though there appears to be evidence for separation between sources of trace extinction in the current research, the precise nature of this division is unknown.

This separation between response- and temporally-driven effects has some support in the literature. Timing and response have been shown to have separable effects on event-related potentials. Nakata et al. (2005) hypothesised, based on their findings of later ERP peak latencies with increased foreperiods and increased ERP peak amplitudes with decreased response probability, that foreperiods affect stimulus evaluation, while response probability affects prepotency. This argument will be furthered below. Secondly, while RTs and inhibition appear to be a function of both explicit and implicit response probability (Durston, Thomas, Worden, Yang, & Casey, 2002; Jahfari et al., 2010; Nakata et al., 2005; Pfefferbaum & Ford, 1988; Thomas et al., 2009), sICI decreases with increasing temporal certainty (Sinclair & Hammond, 2008) irrespective of the probability of response (Sinclair & Hammond, 2009). Furthermore Thomas et al. (2009) demonstrated RT adjustments based on upcoming NG probability; if such implicit foreknowledge affects RTs, it seems reasonable that explicit $T_n$ foreknowledge may affect $T_n$ RTs (and may override implicit $T_{n-1}$ effects as per Adam and Koch, 2009).

*7.1.2 Traces galore* – Perhaps the two different trial initiation signals in the current behavioural tasks generated two separate traces, in a similar way to which shifting initiator modalities may have in Steinborn et al. (2009). However, in all three experiments, a $T_{n-1}$ initiated by unmarked or marked cues did not have separable effects on either definite or non-definite $T_n$ RTs. For example, in experiment 1.1, SM_SM and SD_SM RTs were not significantly different, indicating that $T_{n-1}$ certainty did not differentially affect SM RTs. LM_SD and LD_SD RTs were also not significantly different; response-certain RTs were affected by $T_{n-1}$ timing irrespective of what type of cue initiated $T_{n-1}$. This was equally true in experiment 1.2 and 1.3; it appears that different response probability cues, whether temporal, response type, or both, did not induce different associated traces. One interesting future direction of this idea is to find ways in which different traces (or trace-like explanations of attenuated sequential effects) may

be used. In Steinborn et al. (2009), the trial initiation modality may have served this role; perhaps response modality may induce a similar result.[65]

*7.1.3 Refraction and switching* – The psychological refractory period might explain how a long $T_{n-1}$ reduced SD/STG RTs compared to a short/NG $T_{n-1}$ in experiments 1.1/1.2. A prior response may slow a current response due to its temporal proximity with a subsequent imperative (Welford, 1952).[66] This proposition is not unreasonable, given the widespread effects of motoric actions[67] on signal/response processing (e.g. Hommel, 2009; Shin et al., 2010). The time from long imperative response until the next imperative was always >700ms in this experiment. Muroi, Naito, and Matsumura (1997), utilising a GNG task, found RTs were significantly faster when given an 800ms break after initial go presentation compared to a 400ms break. The beginning of the next trial in this experiment began 1100ms after the initial presentation of the long imperative (i.e. the time from the initial presentation of the stimuli at the long imperative to the fixation cross disappearing in the next trial), far longer than the times used in Muroi's study. Furthermore, in chapter five, I demonstrated a marked decrease in RTs when response and temporal certainty were provided. This difference between a SM_SD and LM_SD compared to a SM_SM and LM_SM was not present in experiment 1.1. Together, these findings indicate that the psychological refractory period (at least by itself) is unlikely to explain the results in this study.[68]

A further possibility is a switching cost when changing from an expected white fixation, to an unexpected marked fixation (see Ruthruff et al., 2001; for review of how inhibition interacts with switching costs see Koch, Gade, Schuch, and Philipp, 2010). Though the 'tasks' are not different, perhaps changing foreknowledge, or processing an 'unexpected' (i.e. non-sequential) fixation had a similar 'switching' effect. This has previously been shown,

[65] Which I am currently testing by intermixing ordered and unordered vocal and manual responses.
[66] See Sigman and Dehaene (2006) for integration of this theory with task switching, having direct implications on inhibitory control.
[67] Though if we assume 'not going' is a type of action, as implied by the (Gomez et al., 2007)7 model then this would not explain why there would be psychological refraction from 'doing button pushes' and not from 'doing not button pushes.'
[68] It may be possible that the psychological refractory period reduces our ability to utilise information regarding the upcoming imperative (i.e. we were slower to process a green fixation indicating a definite trial). This cannot be disproven here; perhaps in the long trial the extra time to imperative allowed analysis of trial type in time for use in the response. However response inhibition is likely not only effortful (Hester & Garavan, 2005), but may be a 'response' of its own (e.g. Gomez et al., 2007) and as such should slow subsequent information processing at least as much as responding does.

unexpected stimuli may alter various aspects of perception (e.g. Grossberg, 1987; Kok, Brouwer, van Gerven, and de Lange, 2013; Kok, Failing, and de Lange, 2014; Ulrich, Nitschke, and Rammsayer, 2006). As found previously, a NG $T_{n-1}$ results in reduced task switching costs (see Koch et al., 2010). This may explain why a LNG/SNG $T_{n-1}$ resulted in relatively faster SD/STG RTs in experiment 1.1-1.3. However, this does not explain why a SM, STG and SD $T_{n-1}$ do not cause significantly different RTs in a SM, STG or SD $T_n$, or why a SM $T_{n-1}$ resulted in faster SD/STG RTs than a LM $T_{n-1}$. Further, 'switching-induced' response attenuation does not argue against selective attenuation; temporal effects remain in SD RTs in experiment 1.1 and 1.2. That response-related $T_{n-1}$ effects are selectively attenuated by switching may support the separation of response- and temporal-driven trace reduction.

To rule out these possible confounds, foreperiods should be extended in future renditions of this experiment, eliminating the possibility of psychological refraction or other motoric effects. Further, adding visual feedback[69] may cut down on early responses, reducing variability across trials and allowing more accurate results to be gathered. Alternatively, if switching costs were relevant, perhaps altering the trial initiation cue occurrence rate may allow analysis of how high this cost is (i.e. making both trial types (definite/non-definite) equiprobable, rather than the individual trials).

*7.1.4 Challenged to a dual* – Given a split between response and temporal inhibition, we might conceptualise two separate 'traces' which work to co-construct $T_n$ response readiness. This 'dual trace' proposition is the main difference between the model proposed here and the trace-conditioning model of Los and van den Heuvel (2001) and Los (2013). Considering that proactive inhibition withholds a response until the appropriate time, while reactive inhibition withholds a response when cued, perhaps these different 'forms' of inhibition underlie the separate trace substrates. One trace may be affected by the passing of an imperative moment, and the other by the active inhibition of a response (or the active reaching of the non-response threshold, as per Gomez et al., 2007). This separation does not necessitate neural substrate differences; as Aron (2011) points out, proactively inhibiting response in anticipation of stopping, and reactively stopping to a NG signal, use similar neural mechanisms. It may be that

[69] Or adding on the incorrect responses onto the end of the experiment. Though given that the current hour tested the limits of human endurance, this may not be advisable.

'inhibition' acts as a brake to allow the withholding of response until appropriate, and the stopping of response when required, via the same mechanism.[70]

Another possibility explaining the selective attenuation of $T_{n-1}$ response type effects is pure probability; perhaps traces did not matter in experiment 1.1. However, two situations existed in experiment 1.1 where response probability was 50%; a LM was equally likely to have a go or NG imperative, while the SD was equally likely to have a go imperative or be passed without incident. However SD RTs were significantly faster than LM RTs (average≈13ms). This seems to indicate that, on average, response uncertainty induces slower RTs than temporal uncertainty. Both types of uncertainty affect RTs; probability of response contributes to response error rates (e.g. Low and Miller (1999)), levels of CSE (e.g. Jahfari et al. (2010); Van Elswijk et al. (2007)) and RTs in the GNG (e.g. Thomas et al. (2009)), and VF paradigms (Li et al., 2005; Los, Hoorn, Grin, & Van der Burg, 2013). These findings may indicate that response uncertainty results in active braking, allowing adequate time for response inhibition if required (Jahfari et al., 2010), while temporal uncertainty does not require the same degree of inhibitory control. However, in experiment 1.2, when given temporal certainty, response uncertainty still required active braking due to response uncertainty, resulting in attenuation of RT decreases that may otherwise result from temporal foreknowledge.

If indeed there are two separate traces driving RTs, one which is response specific and one which is timing specific, then we can further venture other aspects of such a model. Firstly, it seems that once a possible imperative moment is passed in $T_{n-1}$, earlier imperative times are responded to slower in $T_n$. In terms of the dual-process model (Vallesi & Shallice, 2007), a later $T_{n-1}$ response may lead to slower RTs in $T_n$ by virtue of its proximity to the upcoming imperative, an effect which is attenuated the longer the foreperiod is in $T_n$.[71] Whichever model we use, the present point is supported; the position of the imperative in $T_{n-1}$ affects the response speed in $T_n$ by virtue of their relative positions through time, rather than being a result of 'absolute' temporal effects. It could be argued that the dual-process model proposes absolute timing; the later the $T_{n-1}$ imperative occurs, the higher the cost for $T_n$ RTs. However this is balanced by the hazard function, resulting in faster RTs as time passes in $T_n$, and therefore it is

[70] One possibility regarding neural dynamics is that perhaps response braking in a prior trial (i.e. via passing a time point, or inhibiting a response) primes further braking, hence why sequential effects occur. However this requires a great deal of research, and is not discussed further here.

[71] Though as just discussed, psychological refraction does not appear to be the driving factor of RT changes.

the relative position of $T_{n-1}$ and $T_n$ timing, rather than the absolute timing of $T_n$/$T_{n-1}$ that makes a difference.

The relative nature of the imperative timing is supported by the work of Thomaschke and colleagues. Specifically testing if the absolute timing of a response or the relative order of imperatives cause RT differences, Thomaschke, Kunchulia, and Dreisbach (2015) associated timings with responses in a CRT task through a learning phase. In the test phase, these time periods were shortened or lengthened. Whether shortened or lengthened, people still transferred their learning of imperative association to the test phase, demonstrating that, rather than employing absolute time representations (i.e. one imperative was associated with a specific time), timing was relative (i.e. one imperative was associated with being earlier than the other).[72] Furthermore, Thomaschke, Wagener, Kiesel, and Hoffmann (2011a) utilised a continuous foreperiod design, with two responses having two peak foreperiod distributions (i.e. one response was associated with 500ms and one 1,100ms, or in another experiment 300ms and 500ms), demonstrating RT advantages for adjacent foreperiods to each response (not just the exact foreperiod which the signal was most often associated with). In the experiment utilising closer foreperiods, the later foreperiod also appeared to override RT advantages from short-adjacent foreperiods. This finding is in line with discussions regarding an imprecise internal clock (see Schuur, 2012) or imprecise interval timing based on memory (see Lewis and Miall, 2006; Staddon, 2005).[73]

Perhaps the response trace is also temporally specific; a LNG inhibits the timing trace of the short imperative, and the response imperative of the long trace. If this is the case, then RT effects make sense within a dual-trace model. A SG_SG combination would result in reinforcement of the short imperative timing and the short imperative 'go' response in $T_{n-1}$, giving the fastest SG RTs. A SNG_SG would similarly reinforce the timing of the short imperative, but a NG would extinguish the response trace. Thus RTs would be slower in $T_n$, due to response-related RT slowing, though possibly faster than a Long_SG combination (which appears to be a trend in current/prior research). A LNG_SG and LG_SG would both slow RTs by way of the temporal trace being extinguished, while the short imperative response trace has a 'null' associated with it due to the response not being relevant to the short

[72] It would be interesting to test something similar with regards to $T_{n-1}$; perhaps the short imperative timing seems 'earlier' when preceded by a long $T_{n-1}$ compared to a short $T_{n-1}$, hence why LM_STG RTs are longer than LM_SM RTs.

[73] See Eagleman (2008), Ivry and Schlerf (2008) and Meck (2005) for some discussion of the fascinating topic of temporal perception.

imperative. In experiment 1.1, a SD overrides the response trace from $T_{n-1}$ resulting in SM_SD, SD_SD and SNG_SD having similar RTs. However the temporal trace still exists; a long_SD still has significantly slower RTs than a short_SD. In experiment 1.2, a STG did not override the temporal trace due to this being 'blocked' by response uncertainty. However, switching the task (as mentioned briefly above) may still attenuate the effects of the response trace. Thus a long_STG still has slower RTs than a short_STG, while a SNG_STG is still responded to roughly as quickly as a SM_STG. In experiment 1.3, a SD allows the overriding of both response and temporal traces, resulting in faster RTs, irrespective of the $T_{n-1}$ trial type, though some separate RT effect may remain from a long $T_{n-1}$ (discussed above). This is generally supported by Adam and Koch (2009); $T_n$ certainty may override $T_{n-1}$ repetition priming (or, in this case perhaps $T_{n-1}$ response type and timing 'priming').

Before continuing, I wish to briefly discuss the nature of the proposed response trace. It may make sense to view response as being generated by crossing a response threshold and response inhibition resulting from failure to cross this threshold. However, the model by Gomez et al. (2007) makes explicit how the trace is affected by response; crossing the 'go' or 'NG' threshold results in reinforcement or extinction of the trace respectively (see figure 7.1). In line with this model, I propose that, rather than trace 'extinction' and 'reinforcement,' the response trace is also a dual-threshold mechanism. A NG response moves the trace more towards the NG boundary, while a go response moves the trace more towards the go boundary. When a response trace is not involved in the current trial, perhaps there is no movement between these boundaries. Or, perhaps, as discussed in Los et al. (2014), there is some drift back to an asymptote. Along these lines, it would be interesting to only sporadically associate an imperative with a short time frame, and provide even less NG events for this imperative. For example, perhaps 30% of trials are at the short imperative time, and then only 10% of these are a NG, allowing for analysis of asymptotic drift.[74] This may only be a conceptual difference to having a single threshold, however it may aid visualisation of how the trace is affected.

[74] Though this would require some clever designs to prevent experiments lasting hours.

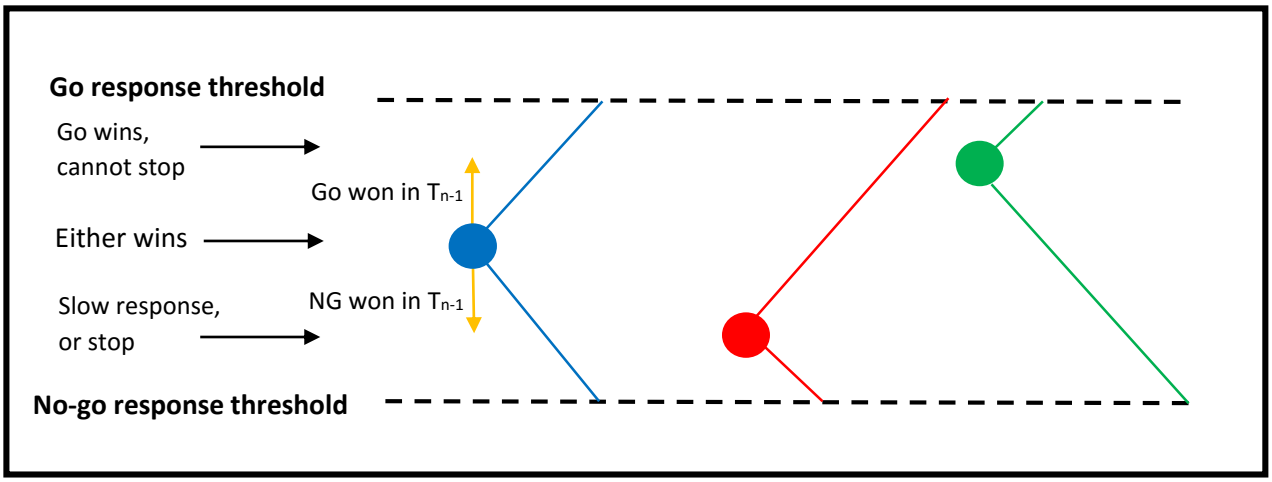


*Figure 7.1: One possible representation of different response scenarios. In each, the appropriate response may be either a go or no-go, but depending on the initial position RTs may be slower (red dot), or response inhibition may be impossible (green dot). Note, in the case of the slow response (the red circle), it is important for the two responses to be processed, rather than reaching the no-go threshold and not responding, it takes longer for the 'go' response to reach threshold. However there may be some interaction between these processes. The initial position is dynamically adjusted based on our expectations of what response will follow. This may be (at least partially) based on the response in $T_{n-1}$, represented by the yellow arrows above and below the blue circle.*

An alternative proposition to temporally-linked responses traces is that there is only one, universal, response trace; response may be a unitary concept such that inhibiting a response in $T_{n-1}$ affects *the* response trace, rather than *a* response trace. To test this, experiment two could be repeated with TMS applied at both the short and long imperative time (in separate trials). If there were a unitary trace, a SNG and LNG $T_{n-1}$ should equally affect both the short and long $T_n$ imperative nMEP-Rs. If there is only one response trace (i.e. response in $T_{n-1}$ was either released or not, regardless of time), then the relationship between the response and temporal traces requires further explanation. Temporally-specific versus universal response traces[75] cannot be differentiated in the current research, however a recent review by Kenemans (2015) discusses the reactive inhibitory mechanism as both independent and generic compared to proactive inhibition, which may support this contention. Further, in experiment two, pre-imperative EMG levels varied based on $T_{n-1}$ response, which may indicate a different 'initial level of response readiness' irrespective of when the NG occurred. Let us examine how this could work. This may facilitate a discussion of how two traces may differentially affect RTs without necessitating trace unity.

One possibility is that the temporal trace and response trace act together to give one unified 'starting point' from which a response is launched when an imperative is given. This could explain how response dynamics are overridden when given response certainty (i.e.

[75] An interesting extension of this may be temporally 'broad' response traces. While a temporal trace affects a specific time point, perhaps response traces affect a larger range of traces, without affecting the entire distribution of timings.

experiment 1.1), the trace effects from $T_{n-1}$ response type are overridden from a 'we did not go last time so to the best of our knowledge we will not go this time' to a 'we know we will go this time.' Response foreknowledge decreases the RT component associated with $T_{n-1}$ response type, and facilitates faster RTs. Further, it may be that response uncertainty in $T_n$ is a 'stronger' source of RT slowing than temporal certainty, and therefore temporal traces cannot be overridden unless response type is already known.[76] However this fails to account for why a LNG $T_{n-1}$ does not induce slower RTs than a LM $T_{n-1}$ in a SM $T_n$. If the two effects were cumulative, then having a response trace universally inhibited, and a temporal trace which was also extinguished should lead to longer RTs than when only one trace type was extinct.

Another possibility is that the two traces affect sequential processes; temporal traces affect the processing of a stimulus, and once processing is done, the response is released. The starting point for the response is then dictated by the effects of the response trace. However, again this model is 'additive;' a LNG_SM should have longer RTs than a LM_SM. Therefore this also cannot be the case.

*7.1.5 Model me this* – Before starting this thesis, I was fairly fit. In fact, I used to enjoy doing team obstacle courses. These competitions had two elements; a team portion and an individual portion. I could be the fastest individual (I wasn't) but what counted was when my team finished. As per Los and van den Heuvel (2001) perhaps the trace sets up the current readiness to respond. However, instead of their being one, unified, readiness to respond, there are two distinct races going on. This postulation has some support in the literature; Duque et al. (2010) suggested that there is one mechanism for withholding a response until appropriate (i.e. proactive inhibition) and one for determining what response to make (i.e. response selection, proactively calibrated). If a go-NG decision is the difference between two choices of response (implied by Gomez et al., 2007),[77] then perhaps $T_{n-1}$ response calibrates the response selection 'channel' and temporal inhibition calibrates the expected time of imperative 'channel,' affecting how quickly we process the imperative. This idea also has support in the literature; there is evidence of temporal readiness affecting perceptual processes (for example,

[76] Think of putting a flour sifter inside a colander; it does not matter how big the colander holes are because the flour sifter is finer grained and therefore already sifts out the chunks which would be caught by the colander.
[77] This is also supported by Labruna et al. (2013) who found that action selection is constrained by the similarities of potential responses (in this case movement versus non-movement, in other words 'different'), balanced by prior experiences and the competitive history between these responses.

see Bausenhart, Rolke, and Ulrich, 2008; Grondin and Rammsayer, 2003; Los and Schut, 2008; Nobre et al., 2007, Rolke and Hofmann, 2007) and more broadly, expectations affecting visual processing (for example Kok, Brouwer, van Gerven, and de Lange, 2013). Though I speak of selective calibration, it may be that both temporal and response traces play a weighted role in response selection and imperative processing. For example, Adam and Koch (2009) found that cueing and repetition have separate effects on a single processing stage and that response cueing can offset/obscure the effects of $T_{n-1}$ response repetition. Perhaps, rather than repetition, $T_{n-1}$ temporal and response 'traces' affect this single processing stage. This may explain why, in experiment 1.1 (and the appendix experiment) a LNG appears to have a reduced effect on SD RTs, despite the short trace being impaired, and furthermore why temporal information alone does not significantly attenuate $T_{n-1}$ temporal dynamics in experiment 1.2. It may be that, given uncertainty of response, the gains in perceptual processing from temporal certainty are cancelled out.

The above proposition has some support. Thomaschke, Kiesel, and Hoffmann (2011) paired either response or signal characteristics (i.e. what the imperative looked like, but not what it indicated) with specific foreperiods, demonstrating that temporal expectancy was only evident when paired with responses rather than imperative characteristics (see also Thomaschke, Wagener, et al., 2011a; Thomaschke, Wagener, Kiesel, and Hoffmann, 2011b). Furthermore, Thomaschke and Dreisbach (2013) paired response-effector (what hand performed the response), response (which could be done by either effector) or visual signal with timing, demonstrating that temporally-predictable effector foreknowledge alone (i.e. not visual signal or response) was required for RT shortening driven by temporal predictability. This, they argued, demonstrated that temporal expectation affected effector-specific motor processing rather than visual processing. If we consider 'not doing' as a separate response effector, then these findings may be in line with response information being required for temporal predictability to have an effect. However it should be noted that Thomaschke's experiments linked timing with response types, while in experiment 1.2 timing was predictable but response type was not.

*7.1.6 Wait for me!* – Let me summarise the above model before continuing. I propose two traces; a temporal- and response-based trace. The effects of these may be selectively attenuated/overridden when given 'better' information, however temporal information cannot be overridden without first having response type information. These traces then affect imperative processing, and response initiation. However to explain LNG_SM and LM_SM RT similarities a 'wait for me' component is necessary such that signal analysis slows response release, as per figure 7.2 below.

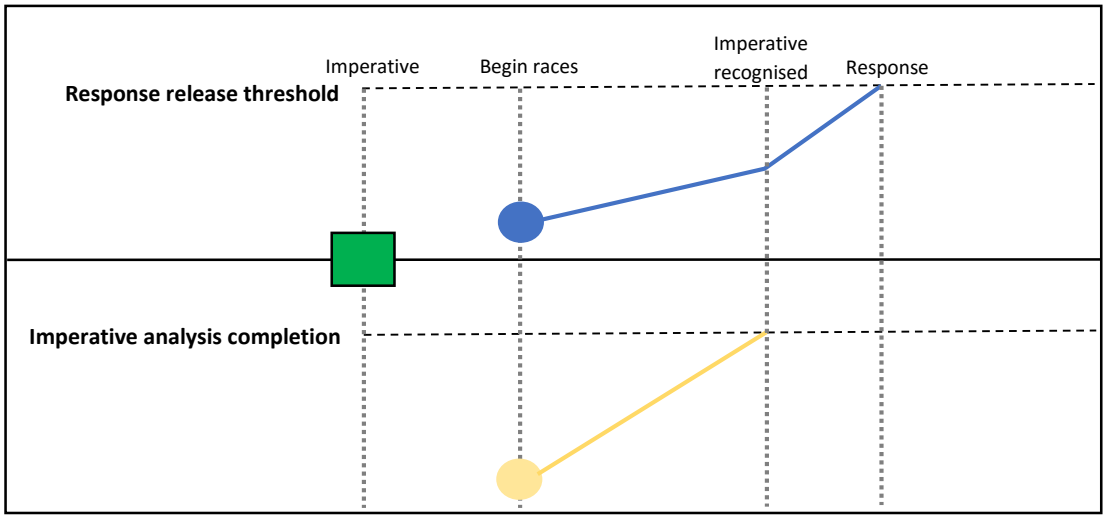


*Figure 7.2: Graphical representation of the dual race. Here, once an imperative is presented, there is a slight lag as this is recognised, then the dual race begins. Throughout this race, the yellow circle, representing imperative analysis, slows the response release race. Once this is done, then the response release race is 'let go' and the rate towards response is increased. Though this is a linear type of model, in reality it may be that the closer the imperative analysis gets to completion, the more quickly the response release trajectory increases, hence the blue line may look more like a curve rather than a straight line.*

In figure 7.2, both dots represent a starting point for two separate races. The finish line for the first race is completion of signal analysis, while the finish line for the second is the threshold for response release. The imperative analysis race slows the response release race (possibly via active braking, as in Jahfari et al. (2010)). Once processing is finished, or enough information is gathered, the response race brake is released, resulting in faster completion of the remaining distance to threshold. In order for the response to be inhibited, the imperative analysis race must gather enough information to be able to inhibit the response (as in Brittain et al., 2012; Bissett and Logan, 2014), or give the NG trajectory enough of a boost to win (as in Gomez et al., 2007).

Importantly, these two processes may be effected by stimulus/rule manipulations not performed in this experiment. For example, more complex imperatives extend RTs (e.g. Mostofsky et al., 2003; for review see Simmonds et al., 2008); perhaps more complex tasks require more evidence before response trajectory release. Further, perhaps the level of evidence

required to release response may also be reduced by increased RT pressure; Benikos, Johnstone, and Roodenrys (2013) found that increasing time pressures decreased RTs and increased response errors. The analysis 'strategy' changed from, 'let's see what the signal is' to 'just go!'

This model accounts for the data in the current experiments. In experiment 1.1, a SM_SD and a SNG_SD are both responded to equally quickly; the expectation of the imperative occurring at that time point is the same, while the response characteristics are already known, and therefore the response race is close to finishing. LM_SD and LNG_SD are reacted to slower due to the analysis race occurring earlier than expected (i.e. at the short rather than long $T_n$), slowing the response release. A LNG_SD may be responded to slightly quicker due to the response trace being overridden, which perhaps provides a stronger signal to start the response race closer to threshold compared to when a go was given in $T_{n-1}$.[78] All LD signals are equally fast due to perfect foreknowledge once the short imperative time has passed, and therefore both races are on the verge of completion. This is also the case for the definite response and timing trials in experiment 1.3. A LM $T_{n-1}$ may induce slightly slower RTs in the SD condition in experiment 1.3 due to the effects of the psychological refractory period, or due to imperfect temporal expectations (as briefly mentioned above, see also Schuur, 2012; Thomaschke et al., 2015).[79] In experiment 1.2, STG RTs may still be affected by foreperiod length due to the temporal trace not being able to be overridden unless first given response foreknowledge, hence a LM_STG was still responded to ≈25ms slower than a SM_STG. This dual race theory also predicts the classic sequential foreperiod effect; a SM_SM is responded to faster than a SNG_SM due to the response race being further from completion, while a LM_SM is responded to slower due to the analysis race being further from completion. A LNG_SM is no longer than a LM_SM because, even though the response race is further from completion, by the time the analysis of the imperative is done, the response is close enough to release that the initial starting point difference has no RT ramifications.

The above model also predicts response errors. Both overtly responding to a NG signal and releasing a response early may be due to the response race being too close to completion such that a response is released before the NG signal is able to be identified and reach the non-

[78] In other words, overriding a trace may override prior response dynamics when given a NG but maybe not when given a go, as the response in $T_{n-1}$, and the expected response in $T_n$, are the same.
[79] Alternatively, this may simply be an artefact of the number of trials that are responded to early in the SD and LD condition meaning that the SD and LD trials which were analysed were the slower subset of response.

response threshold to stop the mistake from being made. Further, while 'active' proactive inhibition may be required at an imperative moment to withhold response unless an actual imperative occurs, this requirement may be stronger for a response certain condition (i.e. at the short imperative moment when in a LD trial) given that the response race is so close to completion. Occasionally, this may result in critical timepoint-induced response release, like a temporal version of a start react (see Carlsen, Maslovat, et al., 2012, for how the start react may increase response probability/speed, see chapter one for discussion). Alternatively, perhaps response urgency may be too high, inducing easily released responses (Kiani & Shadlen, 2009; Thura & Cisek, 2014).

There is some evidence of the analysis and response processes interacting. Brittain et al. (2012) found that, when given an incongruent cue regarding an upcoming response, neural oscillations (recorded with EEG) in the canonical beta band resynchronized before a response was released, while this resynchronization occurred after the response when participants were unable to withhold an incorrect response.[80] This was proposed to indicate a 'pausing' of the motor system while conflict was resolved. In the experiments performed here, rather than expectations being overtly subverted, implicit expectations are set up by the trace, and perhaps, while the conflict between temporal expectations are resolved, the response trajectory is also slowed. This is in line with the active braking hypothesis of Jahfari et al. (2010); a response may be slowed to allow time for the NG to reach threshold. A response is slowed without being cancelled in order to facilitate correct responses. This may indicate that inhibition plays the intermediary role between the response and imperative analysis races; slowing the response race in relation to the imperative race.

*7.1.7 But I am so excited by the LAST trial…* – $T_{n-1}$ response prepotency did not appear to make a significant difference to $T_n$ RTs; there was no significant evidence supporting the contention that prepotency strength affected how strongly a trace was modulate. Having said this, I only implicitly measured prepotency, assuming that having a definite upcoming response increased response prepotency. This contention is supported by faster RTs in $T_n$ when given either response certainty, or response and temporal certainty. Further, responses were more often released early under response certain conditions, implying higher response prepotency. This contention is supported by other RT studies, for example where RTs decreased and

[80] This was in a Stroop experiment.

response errors increased given faster RT requirements (Benikos et al., 2013), and by TMS studies that indicate increased excitation given higher response probability (e.g. Van Elswijk et al., 2007). This finding may indicate that 'inhibition' is channelled in its effects on the temporal trace; temporal inhibition either occurs or does not occur, rather than being a graded effect. Response inhibition difficulty has not been specifically manipulated here however. This may have an interesting avenue of future research. Perhaps, different response probabilities may be differential effects on $T_n$ RTs.

*7.1.8 Dual traces or dual processes?* – As discussed in Los (2013) and in chapter one, the dual-process theory has difficulty accounting for NG $T_{n-1}$ effects on $T_n$ RTs. Though the effect of LNG $T_{n-1}$ on LM RTs was not repeated in the current research, the reason for this is likely due to the reduced probability of these trials occurring compared to other trial types, and, as discussed in appendix one, this resulted in an attenuated LNG $T_{n-1}$ effect on long $T_n$ RTs. However SNG_SM and SM_SM RTs should also not be significantly different according to the dual-process theory. The current research did repeat Los' (2013) finding in this regard, providing further evidence against a dual processing model (see Capizzi et al., 2015, for further comparisons between the two models). Even if timing and response separately affect RTs, the dual-process account of Vellesi cannot explain the $T_{n-1}$-$T_n$ RT patterns easily. If RTs are affected by a NG along a separate stream, then a SNG_SM would have a longer RT than a SM_SM, due to perhaps effortful response inhibition (Hester & Garavan, 2005). Further, the time from one short imperative to a subsequent short imperative is the same as in long-long trial combination, therefore a LNG_LM should have longer RTs than a LM_LM trial combination. Again, this appears to be the case. However, given that the time from a long to short trial is reduced, and that response inhibition causes RT increases along a separate stream, then a LNG_SM should have longer RTs than a LM_SM. This was not the case. The dual-process theory does not account for SD RT changes either; neither the hazard function nor psychological refraction can adequately account for SD RTs without some supplementary process.[81]

[81] For example reduction of refractory costs or increased hazard function RT improvements over time.

## 7.2 So Much for the Short Imperative

The short imperative time has passed. The question now is, 'how will we respond in the future?' The current research suggests a division between $T_{n-1}$ timing and response type effects. This final section examines issues with the current research and proposes improvements.

*7.2.1 Don't give me problems, give me solutions!* – In the behavioural experiments there is room for further analysis. Though I have briefly mentioned error patterns, these deserve proper analysis, including when response errors occur (i.e. if there are particular $T_{n-1}$ to $T_n$ patterns in which response errors are more likely), and by how long these responses were early. Further, within each experiment, some RT patterns had greater variability than others. This may have been due to individual differences and the number of trials discarded due to early responses. I did not use response feedback as I wished to analyse mistakes and separate those who are good at inhibiting early responses from those who were not. However in order to reduce this variability, perhaps in future renditions of these experiments feedback should be provided after early or incorrect responses, reinforcing 'correct' behaviour. Performing similar experiments utilising a non-aging imperative distribution may also be interesting.

Conclusions regarding the effect of temporal certainty require caution. In the short blocks of experiment 1.2, where a STG indicated a temporally certain imperative without foreknowledge of the required response, there was no significant interaction between $T_n$ and $T_{n-1}$ type nor timing, except when comparing the $T_n$ RTs caused by a LNG $T_{n-1}$ versus a SM $T_{n-1}$. If $T_{n-1}$ timing had a significantly reduced effect on $T_n$ RTs we would expect a reduction of $T_{n-1}$ effects when comparing SM to STG RTs. This was not the case. Further, SM and STG RTs were the same whether proceeded by a LM or LNG in $T_{n-1}$. This appears to provide some support for the contention that temporal information remains when given a cue for temporal certainty. The lack of a statistical difference between LM and SM $T_{n-1}$ effects is likely due to the large variability in LM effects.[82] One possibility, considering the variation in numbers of errors between individuals, is there may have been two separate populations within the sample. Correa et al. (2010), using a similar VF GNG task, demonstrated that while a low impulsivity group tended to demonstrate sequential effects on both RTs and response inhibition, a high

[82] Given that experiment 1.2 had the highest probability of a NG occurring, and early responses were fairly uncommon, it seems unlikely that this variability was due to decreased trials contributing to LM_STG mean RTs.

impulsivity group demonstrated only sequential benefits on RTs but not response inhibition. This may also indicate some separation between response and temporal inhibition.

Despite the comparative weaknesses of experiment 1.2, there appears ample evidence to indicate that temporal and response information have separable effects on $T_n$ RTs. However, here I have only tested implicit temporal inhibition, it may be that explicit response inhibition has a stronger effect on a respective trace. To test this we could set up an experiment in which different fixation crosses indicate different probabilities of going (e.g. 25%, 50% and 75%) and see if these had variable effects on $T_n$ RTs. Given prior research, the required 'effort' or proactive inhibitory level, required to inhibit a response may be different given different (subjective/objective) probabilities of going (for example Benikos et al., 2013; Vink, Kaldewaij, Zandbelt, Pas, and du Plessis, 2015) therefore perhaps a timing trace is equally extinguished despite the certainty of $T_{n-1}$.

How exactly inhibition relates to trace extinction may also be of interest. A GNG task requires direct mapping of a stimulus to a response (Verbruggen & Logan, 2008a), it could be that accessing the mapping related to the non-response stimulus is the actual cause of trace extinction. Therefore it may be pertinent to look at inhibition as a process and not just a single action; future studies may seek to examine what aspect of inhibition causes trace extinction, and why this is directly related to a particular time rather than a more general process.

In experiment two, when examining TMS-only RTs, though LG $T_{n-1}$ resulted in slower short $T_n$ RTs, a SNG and LNG $T_{n-1}$ did not have the same slowing effect. However, without TMS the pattern of $T_{n-1}$ effects were as described in Los (2013); a SNG $T_{n-1}$ increased SM $T_n$ RTs, as did long $T_{n-1}$. Further, in both TMS and non-TMS trials, a long $T_{n-1}$ resulted in longer LG RTs compared to short $T_{n-1}$. These variations from the expected RT sequence may have been due to TMS trials being associated with separate traces compared to non-TMS trials (as per Steinborn et al., 2009), or (either the sound or effects on M1) disrupting some aspect of the trace effect. The remaining effects on TMS trials may be those that survived from a trial prior to $T_{n-1}$. Alternatively, as TMS may either increase or decrease RTs (Pascual-Leone et al., 1992; Ziemann, Tergau, Netz, & Hömberg, 1997), perhaps TMS-driven RT changes acted to mask the effects of a $T_{n-1}$ NG while retaining the RT difference caused by $T_{n-1}$ timing. The variation of TMS-induced effects on RTs in itself is an interesting area of further investigation. This may cause some hesitation in drawing conclusions from the TMS results here, especially if linking RTs with CSE levels. To better deal with this in future, I wish to randomly vary TMS

presentation such that the participant cannot predict when TMS will occur. Though this may not overcome the problem of associating TMS with different trials, due to the random order it may reduce any 'prepared' or 'expectation' based TMS-related sequential effect attenuation. Alternatively, though the current TMS study was timed to allow for TMS capacitor recharging, perhaps in future TMS pulses could be delivered on every trial, preventing differential trace association.[83]

Rather than linking RT to CSE levels, it may be interesting to examine how $T_n$ MEPs vary as a function of $T_{n-1}$ MEPs. This may provide a more subtle measure of $T_{n-1}$ effects on $T_n$ expectations. Combining TMS with the experimental manipulations of the purely behavioural experiments in this thesis may provide further insight into how $T_{n-1}$ response dynamics influence current levels of readiness. These sequences of MEP alterations may be linked to RTs, furthering our knowledge of how RTs are affected by $T_n$ and $T_{n-1}$ CSE levels. Further time points should also be tested using both single- and paired-pulse TMS protocols, allowing excitation/inhibition tracking and imperative-moment-specific versus non-imperative-moment comparisons. Within the current data there is also room for further analysis, for example of those trials proceeded by or containing a response error. However, to gain further insight into this, the experiment should be re-performed with manipulations making response inhibition more difficult. Finally, Vallesi et al. (2007) utilised repetitive TMS to cause a virtual lesion to the right dorsolateral prefrontal cortex (rDLPFC), causing attenuation of the sequential foreperiod effect. Right frontal areas are also often associated with response inhibition (Aron, 2011; Aron et al., 2004; Chambers et al., 2009; Kroeger et al., 2010; Levy & Wagner, 2011; Mostofsky et al., 2003; Nakata et al., 2014; Picazio et al., 2014; Rubia et al., 2001). It may be interesting to depress activity in the rDLPFC, and other frontal areas, for example the pre-supplementary motor area, and look at differential patterns of sequential effects in both response type and timing. For example, in the experiment by Los (2013), a virtual lesion to the rDLPFC would likely diminish the sequential foreperiod effect, as discussed by Vallesi et al. (2007), however would the sequential effects of response inhibition (or the attempt thereof) remain?

In the current thesis, while I have explored MEP-based sequential analysis, it may be equally interesting to pursue sequential analysis of neuroimaging techniques. This type of 'predictive coding' is currently gaining momentum (e.g. de-Wit, Machilsen, and Putzeys,

[83] This would also gain more data per cell, and allow more trials to be performed with TMS, reducing nMEP-R variability and resulting a stronger ability to detect significant effects.

2010). Predictive coding-type analysis utilising neuroimaging techniques (e.g. Rauss, Schwartz, and Pourtois, 2011; Vuust, Ostergaard, Pallesen, Bailey, and Roepstorff, 2009) and neuronal modelling (e.g. Wacongne, Changeux, and Dehaene, 2012),[84] may be of interest in furthering our understanding of the link between inhibition and the VF. For example, a predictive coding type of neuroimaging approach could answer how $T_{n-1}$ dynamics predict $T_n$ brain states, and how these in turn predict our reactions to imperatives?

*7.2.2 The end of time* – Timing is everything. And now is the time to finish this thesis. Utilising four different experimental manipulations, I have proposed two aspects of trace control. Firstly, I have presented evidence which I believe supports a separation between temporal- and response-based trace effects on $T_n$ RTs. I went on to describe two possibilities of how these may fit together depending on how response inhibition is actualised in the response trace. Secondly, I provided evidence which indicates further experiments utilising sequential analysis of CSE. Though the conclusions from this chapter may be weaker compared to other findings, it seems that sequential effects may better stratify how CSE is affected. Finally, in the last section, and sporadically throughout this thesis, I have presented several ideas of how to address the problems with the current research, and further the related findings.

So finally, how did my prior experiences crossing busy roads while reading predict that I would not be hit by a bus? From the prior and current research, two factors are apparent. Firstly, that I am constantly almost walking into things has made me fairly cautious when I walk and read. The number of close calls I have had also meant that the short foreperiod had a relatively strong representation; I was ready for a close call. But the main factor that saved my life? Dumb luck.

[84] More generally, see Adams, Friston, and Bastos (2015) and Brodski, Paasch, Helbling, and Wibral (2015).

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**Appendix 1: Experiment 1.4**

In this experiment a white fixation indicated a go could occur at either the short or long imperative time, but a NG could only occur at one imperative time, as specified at the beginning of each block. A green fixation indicated a no-go would not appear, but did not give temporal certainty (as per experiment 1.1). On any trial, SM, LM, SD, LD and either SNG or LNG were equiprobable therefore 80% of trials required a response. This experiment had the highest response probability of all experiments performed. Broadly; the question of this experiment is 'how does temporal- and response-driven uncertainty from the prior and current trial alter RTs in $T_n$?'

This experiment manipulates response probability asymmetrically across time. In the long blocks (LNG only) response probability was 50% in two trial types; once the short imperative time had passed in the uncertain condition, a long imperative would definitely occur but may be either associated with a go or NG (equiprobable). At the beginning of the response-certain trials, a short imperative would definitely be a 'go' imperative, however the time could be passed. At the beginning of an uncertain trial, a go at the SM time point had a 33% chance of occurring, due to the possibility of it being passed, and the two alternative possibilities at the long imperative time. However, if an imperative did occur, it was guaranteed to be a 'go' imperative. In the short blocks (SNG only), a SM had a 33% chance of requiring a response. However, this time, the probability of response was driven by both the uncertainty of response (a NG and go imperative were equiprobable) and a chance of the short imperative not occurring (also 33% chance of occurring). Once the short imperative had passed, response and timing became certain at the LM imperative time. As above, a SD had a 50% chance of occurring given a green fixation. In both blocks, once the short imperative had passed in the definite-go trials, timing and response became certain at the LD imperative. This meant that, in the long block, there was an increase in response probability from the SM to LM imperative from 33% to 50%, driven by a loss of response certainty but a gain in temporal certainty, while in the short block there was an increase in response probability from the SM to LM imperative from 33% to 100% driven by an increase in both response and temporal certainty. The results of the three behavioural experiments indicate a possible separation between response and temporal (proactive) inhibition. This experiment may further these results via requiring different levels of response 'caution' at different times throughout the trials.[85]

[85] Further, this experiment allowed the examination of reengagement of proactive inhibition after a short imperative time has passed if the no-go could only occur at the long time point, or turning off of inhibition after

### A1.1 Visualization of Results

Figures A.1 and A.2 represent the mean RTs in the long (i.e. a NG can only occur at the long time) and short blocks respectively.

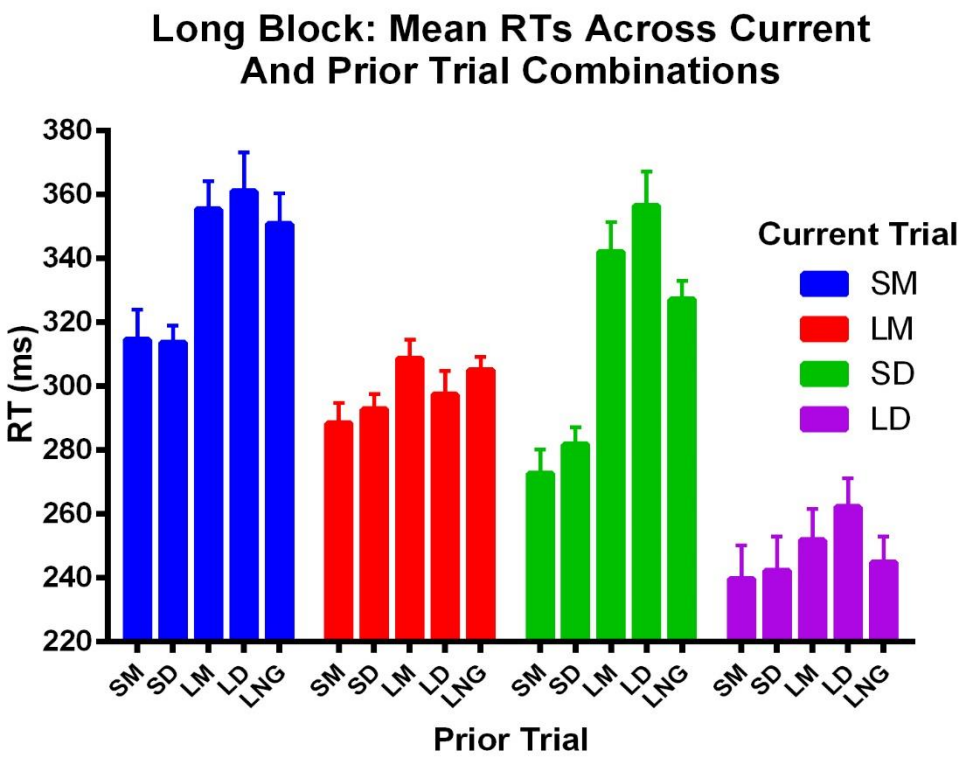


*Figure A.1: Mean RTs in the long block, where a NG could only occur at the long time frame. SM RTs follow the pattern described by Los (2013); $T_n$ RTs were slower given a long $T_{n-1}$ compared to a short $T_{n-1}$. LM and LD $T_n$ RTs appear relatively unrelated to $T_{n-1}$ type. SD and LD $T_n$ RTs appear similar to the pattern seen in experiment 2.1, where LD RTs are relatively unaffected by $T_{n-1}$, while SD RTs are slower when proceeded by a long compared to a short $T_{n-1}$. Again, a LNG $T_{n-1}$ does not appear to slow RTs in a SD $T_n$ compared to a LM/LD $T_{n-1}$.*

In the long block, in which a NG could only occur at the long time frame, SM RTs (figure A.1, far left) appear to follow the RT patterns reported in Los (2013); a long $T_{n-1}$ appears to slow $T_n$ RTs compared to a SG $T_{n-1}$ irrespective of long $T_{n-1}$ response type. LM RTs (figure A.1, middle left) appear relatively non-reactive to $T_{n-1}$, with no apparent effect of timing or response. SD RTs (figure A.1, middle right) follow the pattern discussed in chapter three, in which $T_{n-1}$ response type appears to have less effect on RT in $T_n$, while $T_{n-1}$ timing still appears to affect RTs in $T_n$. A LNG in $T_{n-1}$ appears to be responded to quicker in a SD $T_n$ compared to

the short imperative time if this was the only no-go time. Further, this allowed examination of how prior and current trial certainty affects inhibition alteration; if in $T_n$ there was a reduction of inhibition due to the passing of the only no-go time point, then perhaps this trial's RTs would be equivalent to the long definite time condition. This experiment was on a slight tangent to the others and therefore placed in the appendix.

a long go $T_{n-1}$. LD RTs (figure A.1, far right) also appear to follow the trend of experiment 1.1 and 1.3, in which RTs in $T_n$ appear largely unaffected by $T_{n-1}$ dynamics, except possibly when

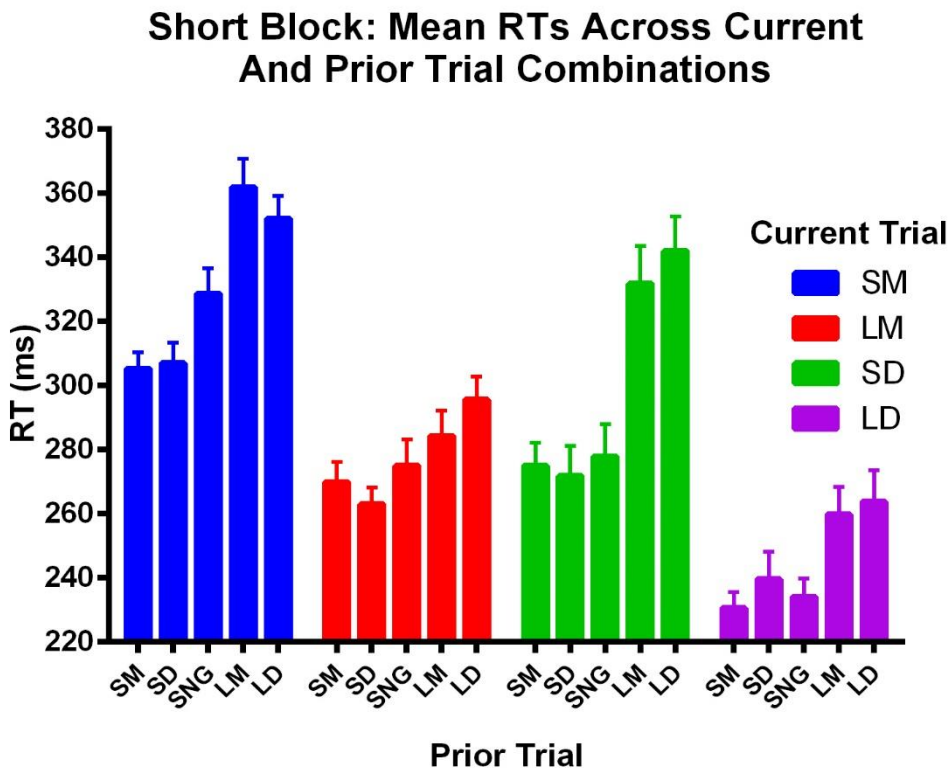


*Figure A.2: Graphical representation of RTs in the short block, where a NG can only occur at the short imperative time in the uncertain condition. SM RTs again appears similar to Los (2013); a SNG slows $T_n$ RTs in the following trial compared to a SG $T_{n-1}$, while the long trials further slow RTs in $T_n$. LM RTs appear relatively irrespective of $T_{n-1}$, though a LD $T_{n-1}$ appears to cause slightly slower LM RTs than the short $T_{n-1}$ trials. SD RTs appear consistent with experiment 2.1, with longer RTs in $T_n$ when proceeded by a long $T_{n-1}$ trial. Within the short $T_{n-1}$ trials there appears no significant difference between trial types. LD RTs appears fairly consistent with experiment 1.2, however RTs appear slower when proceeded by a long $T_{n-1}$ compared to a short $T_{n-1}$.*

given a LD in $T_{n-1}$ which may cause slower LD RTs.

In the short block, in which a NG could only occur at the short time point, SM RTs (figure A.2, far left) follow the pattern reported in Los (2013); SG $T_{n-1}$ result in fastest $T_n$ RTs, followed by SNG $T_{n-1}$ and finally long $T_{n-1}$ appear to cause the slowest $T_n$ RTs. LM RTs (figure A.2 middle left) appear to be relatively unaffected by $T_{n-1}$ trials, however long $T_{n-1}$ do appear to cause slower $T_n$ RTs, especially when given a LD $T_{n-1}$. Both SD and LD $T_n$ RTs (figure A.2, right two groups) appear slower when preceded by long rather than short trials. This difference is greater in SD $T_n$ than in LD $T_n$.

**A1.2 Results**

Mean RTs varied from 260 to 350ms in the long block (mean=301ms, SD = 55ms) and from 241 to 346ms in the short block (mean= 288ms, SD =56ms). Generally, in both the long and short blocks, each participant presented similar RT patterns in the LM, SM and LD $T_n$ conditions. Within the SD $T_n$ condition, some participants' RTs were less reactive to prior trials than for others. For those with less variation, the difference appeared to arise from long $T_{n-1}$ trials being responded to relatively quickly in $T_n$, almost at the same speed as when preceded by a short $T_{n-1}$. As mentioned previously, this may indicate variation in individuals' $T_n$ information use (i.e. how they utilised the information from a green fixation). This is not analysed further here. The number of errors ranged from 32 to 200 (2% to 12.6% of all trials respectively). There were few response errors,[86] of the 200 mistakes from the person with the most errors, 16 were from incorrect responses (1.0% of all trials). Most errors were from responding prior to an imperative being given. The person with the most errors also released a response 43 times (2.7% of all trials) at the short imperative time when given a definite go fixation but no imperative (i.e. released response at a short imperative time though no imperative was presented).

When comparing the two blocks (after removal of NG trials, to equalise the trials contained in each) using a paired t-test, there appeared to be a significant difference between the blocks ($t(191)=3.57$, $p<0.001$), with RTs in the short blocks being, on average, ≈8ms faster than in the long blocks. When comparing the four $T_n$ trial types (i.e. a short-block SM with a long-block SM) LD, SD and SM $T_n$ trials were not different ($p\approx1$) while long-block LM $T_n$ trials were responded to significantly slower than in the short block by ≈19ms ($p<0.01$).

[86] Interestingly, the individual with the least total errors actually made a high percent of response mistakes (26/32). This may indicate that the participant who was able to withhold from responding early, had worse reactive inhibition, in line with the dichotomy discussed in Aron (2011). Though this pattern deserves further attention, it is beyond the current scope of discussion.

Due to mutual exclusivity (e.g. in the short block a long trial never co-occurs with a NG), $T_{n-1}$ information was collapsed such that there were five $T_{n-1}$ levels (SM, LM, SD, LD and either SNG or LNG). This resulted in three factors; type of $T_{n-1}$, timing of $T_n$ and certainty of $T_n$. Tables A.1 and A.2 summarize results for the long and short blocks respectively, presenting those interactions which were not superseded.

**Table A.1: Significant interactions in long blocks**

| Interaction | DF (n,d) | F-value | Probability | $\eta_p^2$ |
|---|---|---|---|---|
| $Timing_n$ x $Certainty_n$ | 1, 11 | 36.81 | <0.0001 | 0.78 |
| $Timing_n$ x $Type_{n-1}$ | 4, 44 | 12.47 | <0.0001 | 0.26 |
| $Certainty_n$ x $Type_{n-1}$ | 4, 48 | 4.68 | < 0.005 | 0.10 |

*Table A.1: Statistical significance of those interactions not superseded by other interactions in the long blocks of experiment 1.4. The first column specifies the interaction. The second specifies the degrees of freedom. F-values are then presented, followed by their corresponding p-values and finally eta-squared.*

**Table A.2: Significant interactions in short blocks**

| Interaction | DF (n,d) | F-value | Probability | $\eta_p^2$ |
|---|---|---|---|---|
| $Timing_n$ x $Type_{n-1}$ | 4, 44 | 6.09 | <0.01 | 0.13 |
| $Certainty_n$ x $Type_{n-1}$ | 4, 44 | 3.26 | <0.05 | 0.07 |
| $Timing_n$ x $Certainty_n$ x $Type_{n-1}$ | 4, 44 | 2.68 | = 0.0795 | 0.06 |

*Table A.2: Statistical significance of those interactions not superseded by other interactions in the short blocks of experiment 2.4. The top and bottom highlighted rows are corrected values due to sphericity violations (Greenhouse-Geisser estimates for sphericity corrections of DF were used) (see Field, Miles, and Field (2012)). The three way interaction was significant initially (Timing/certainty of current trial, and type of prior trial) however it was no longer significant when corrected for sphericity violation. This interaction still approaches significance.*

*A1.2.1 Long blocks*

To begin with, long blocks (figure A.1, table A.1) in which a NG could only occur at the long time point, are analysed.

*Interaction between timing and certainty of current trial* – The first interaction reported in table A.1 indicates that there is an interaction between the timing and certainty of $T_n$. This is shown in figure A.3 below.

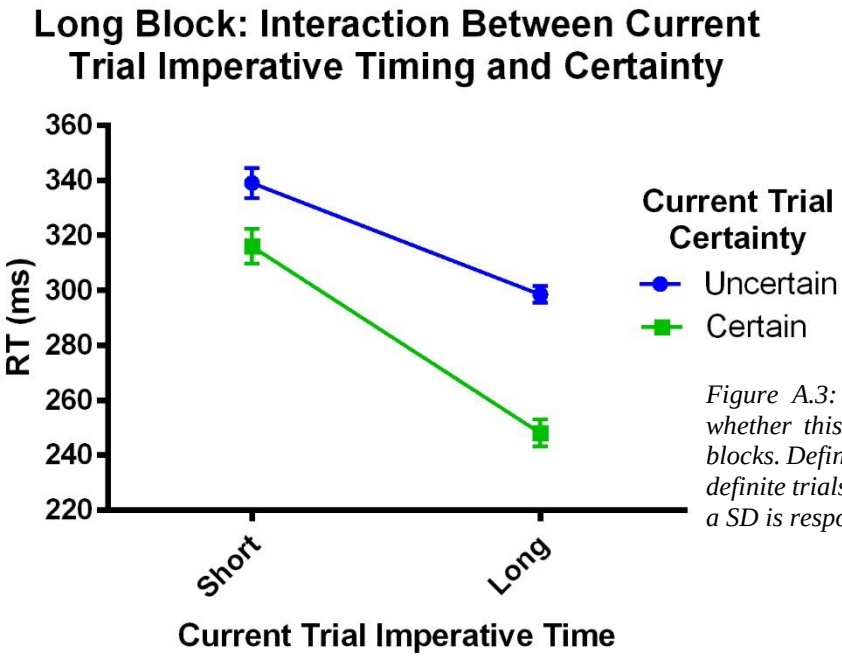


*Figure A.3: Effect of the imperative time of $T_n$, and whether this trial is certain or uncertain, in the long blocks. Definite trials are responded to quicker than non-definite trials at each corresponding imperative time (i.e. a SD is responded to faster than a SM).*

Each of the above points were significantly different from one-another (all $p<0.0001$, except for the difference between SD and LM points, $p<0.05$). A SM $T_n$ was responded to ≈23ms slower than a SD and ≈41ms slower than a LM $T_n$, and a SD $T_n$ was responded to ≈18ms slower than a LM $T_n$. Finally, RTs were ≈68ms faster when moving from a SD to LD $T_n$ (figure 7.3, green line), while the difference in the uncertain condition was ≈41ms (figure 7.3, blue line), indicating RT decreases ≈27ms more from the short to long imperative time in the certain compared to the uncertain condition.

*Interaction between timing of current trial and prior trial type* – Figure A.4 below demonstrates RTs given the interaction between the timing of the $T_n$ imperative and the type of $T_{n-1}$.

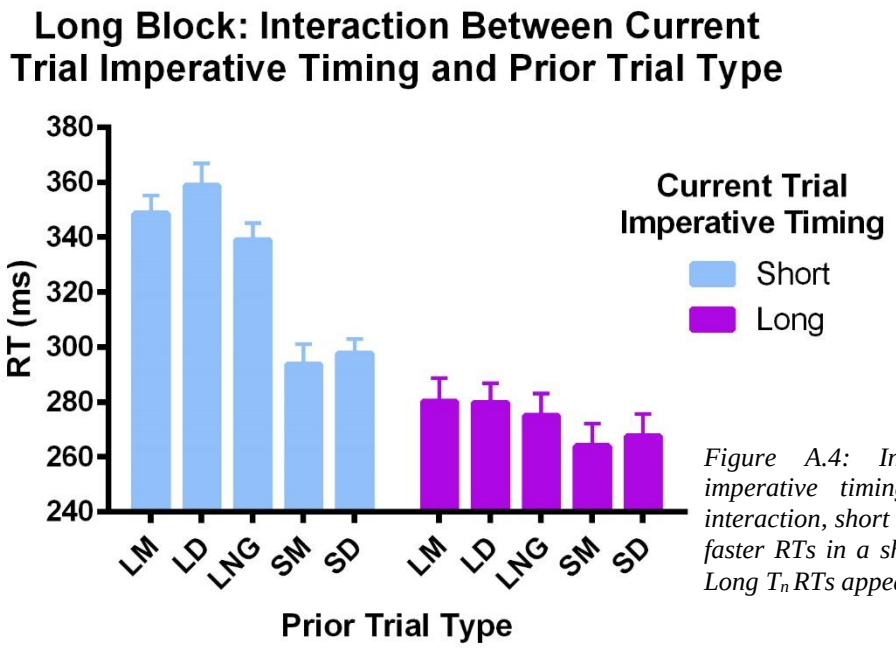


*Figure A.4: Interaction between current trial imperative timing and prior trial type. In this interaction, short trials in $T_{n-1}$ appear to associate with faster RTs in a short $T_n$ compared to long $T_{n-1}$ trials. Long $T_n$ RTs appear to not be as affected by $T_{n-1}$ type.*

In the short $T_n$ trials (left group) RTs were significantly faster given a short rather than long $T_{n-1}$ (all $p<0.0001$, minimum difference (between SD and LNG $T_{n-1}$) was ≈41ms). Short and long $T_{n-1}$ did not differentially affect $T_n$ RTs compared to other short and long $T_{n-1}$ respectively (all $p\approx1$). In the long $T_n$ trials, there were no significant interactions between $T_{n-1}$ types (all $p>0.4$ minimum, except SM versus LM where $p=0.098$, the difference in $T_n$ RTs between these two was 16ms). This indicates that RTs in response to a short $T_n$ imperative are affected by $T_{n-1}$ timing, but not imperative type, while long $T_n$ RTs are largely unaffected by $T_{n-1}$ type and timing. Between the two $T_n$ timings, there was no significant effect on $T_n$ RTs between a long-long and short-short trial combination, except when given a LNG $T_{n-1}$ in the long condition (the middle of the long $T_n$ group) and the SD $T_{n-1}$ in the short condition (the column second from the right in the short $T_n$ group) ($p<0.05$).

*Interaction between certainty of current trial and prior trial type* – Figure A.5 below illustrates the interaction between the certainty of $T_n$ and the trial type of $T_{n-1}$.

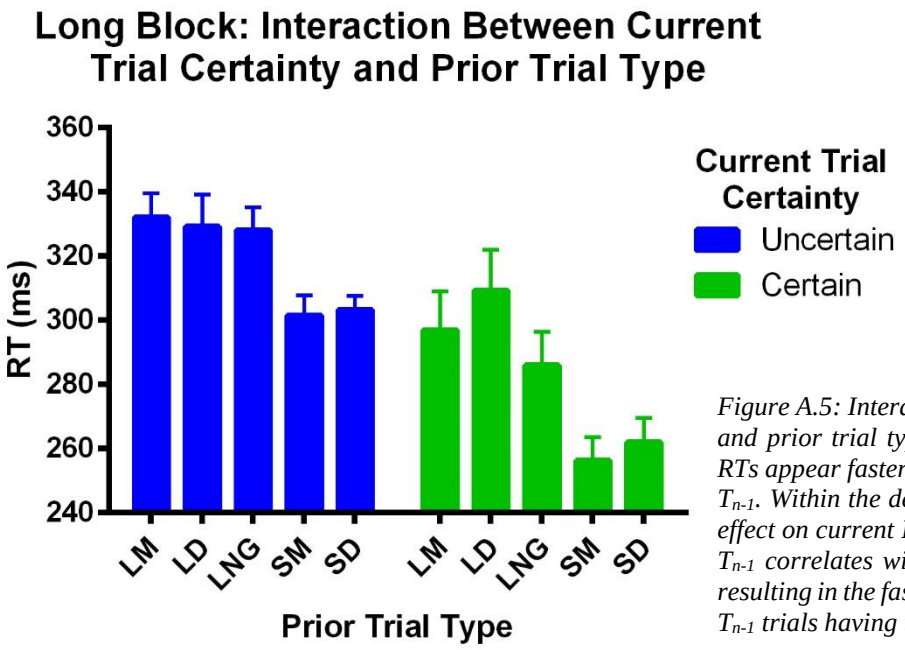


*Figure A.5: Interaction between current trial certainty and prior trial type. In this interaction, uncertain $T_n$ RTs appear faster when given a short rather than long $T_{n-1}$. Within the definite $T_n$ trials (right) the prior trial effect on current RTs appears graded such that a long $T_{n-1}$ correlates with the slower $T_n$ RTs, and the LNG resulting in the fastest of the slow $T_n$ RTs. The two short $T_{n-1}$ trials having the quickest RTs in $T_n$.*

In the uncertain $T_n$ trials (left group), short and long $T_{n-1}$ type made no significant difference to $T_n$ RTs compared to other short/long $T_{n-1}$ trials respectively (all $p \approx 1$). LM and LNG $T_{n-1}$ resulted in significantly slower $T_n$ RTs than did all short $T_{n-1}$ (all $p<0.01$). There were no significant difference between short and LD $T_{n-1}$ effects on $T_n$ RTs (SD-LD $T_{n-1}$ $p>0.26$, SM-LD $T_{n-1}$ $p<0.11$). Long and short $T_{n-1}$ resulted in a minimum $T_n$ RT difference of ≈24ms given an uncertain $T_n$ (between a SD and LNG $T_{n-1}$). When given a definite $T_n$ (figure A.5, right group), within the long and short $T_{n-1}$ trials there were no significant differences in $T_n$ RTs compared to other long/short $T_{n-1}$ trial effects on $T_n$ RTs respectively (all $p \approx 1$, except the difference between a LNG and LD $T_{n-1}$, $p=0.18$). All $T_{n-1}$ long trials were responded to significantly slower in $T_n$ compared to all short $T_{n-1}$ (maximum $p<0.05$), except when comparing LNG $T_{n-1}$ to SD and SM $T_{n-1}$ (both comparisons approached significance; $p=0.085$ and 0.056 respectively). The minimum difference in mean $T_n$ RTs between the two $T_{n-1}$ times within the definite $T_n$ group was ≈24ms (between SD and LNG). Between the certain and uncertain $T_n$ groups, there was no significant difference in mean $T_n$ RTs between an uncertain $T_n$ short $T_{n-1}$ and certain $T_n$ long $T_{n-1}$ ($p \approx 1$), however a definite $T_n$ short $T_{n-1}$ was significantly different from all uncertain $T_{n-1}$ trials (maximum $p<0.005$).

Briefly, examining the effect of $T_{n-1}$ response on $T_n$ RTs, in both the LD and LM $T_n$ trials, there were no significant differences in $T_n$ RTs when given a LNG $T_{n-1}$ compared to any

other trial (p≈1). SD RTs were significantly slower when preceded by a long compared to short $T_{n-1}$ (all maximum $p<0.05$, except comparing LNG_SD to SD_SD, where $p=0.068$). SM RTs were significantly slower (or approached significance) when preceded by either a LM or LNG $T_{n-1}$ compared to a short $T_{n-1}$ (all maximum $p<0.05$, except SD_SM compared to LM_SM ($p=0.090$), and SD_SM compared to LNG_SM ($p=0.078$)).

*A1.2.2 Short blocks*

For the short block (figure A.2, table A.2) where a NG could only occur at the short imperative time, there was no significant interaction between $T_n$ timing and $T_n$ certainty ($F(1,11)= 0.010$, $p= 0.922$). This is in contrast to the long block. As mentioned above, a SM $T_n$ had a 33% chance of requiring a response, while a SD had a 50% chance at the beginning of the trial. Once the short imperative time point had passed, LM and LD were both certain. The lack of interaction between $T_n$ timing and certainty demonstrates that RT decreases from a SM to LM (i.e. from a 33% chance of responding, driven by response and temporal uncertainty, to response certainty) and from a SD to LD (i.e. 50%, driven by purely temporal uncertainty, to response certainty) were not significantly different (SM to SD RT difference ≈31ms, LM to LD RT difference ≈32ms). A LM (a certain response) was ≈22ms faster than a SD however (50% certain response) $T_n$. This difference was significant ($p<0.01$) (see figure 7.3 to visualise points).[87]

[8787] SM, SD, LM, and LD all had significantly different RTs (all $p<0.0001$).

*Interaction between timing of current trial and type of prior trial*– Figure A.6 below represents the interaction between $T_n$ timing and $T_{n-1}$ trial type.

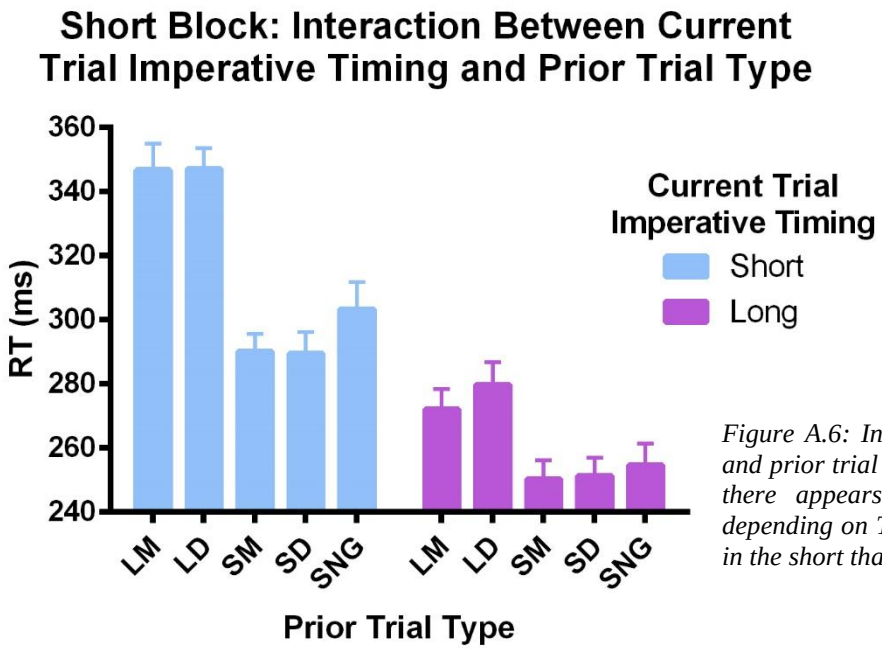


*Figure A.6: Interaction between current trial timing and prior trial type. In both $T_n$ groups (left and right) there appears a noticable difference in $T_n$ RTs depending on $T_{n-1}$ timing. This difference was greater in the short than the long $T_n$ trials.*

Within the short $T_n$ trials (figure A.6, left), short and long $T_{n-1}$ did not cause significantly different $T_n$ RTs from other short and long $T_{n-1}$ respectively (minimum $p>0.3$). All short $T_{n-1}$ trials were responded to significantly faster than all long $T_{n-1}$ trials in $T_n$ (maximum $p<0.01$). The minimum difference between $T_n$ RTs within $T_{n-1}$ timings was ≈34ms (between the SNG and LM $T_{n-1}$ trials). In the long $T_n$ condition (figure A.6, right), within the short $T_{n-1}$ and long $T_{n-1}$ timings there were no significant differences in $T_n$ RTs (all $p\approx1$), while between the $T_{n-1}$ timings, all $T_n$ RTs were significantly different (maximum $p<0.05$), except when comparing the effect of a SNG and LM $T_{n-1}$ ( not significantly different from each other; $p=0.15$). The minimum difference in $T_n$ RTs between a short and long $T_{n-1}$ was ≈17ms (between SNG and LM $T_{n-1}$). Between the $T_n$ timing groups (i.e. when comparing short and long $T_n$), there was no significant difference between a long $T_n$ long $T_{n-1}$ combination and a short $T_n$ short $T_{n-1}$ combination (all $p>0.1$ minimum). This interaction appears to be driven by different effects on $T_n$ RTs between a long and short $T_{n-1}$ within the $T_n$ groups.

*Interaction between certainty of current trial and type of prior trial* – Figure A.7 below demonstrated the final significant interaction found in this experiment, between $T_n$ certainty and $T_{n-1}$ trial type.

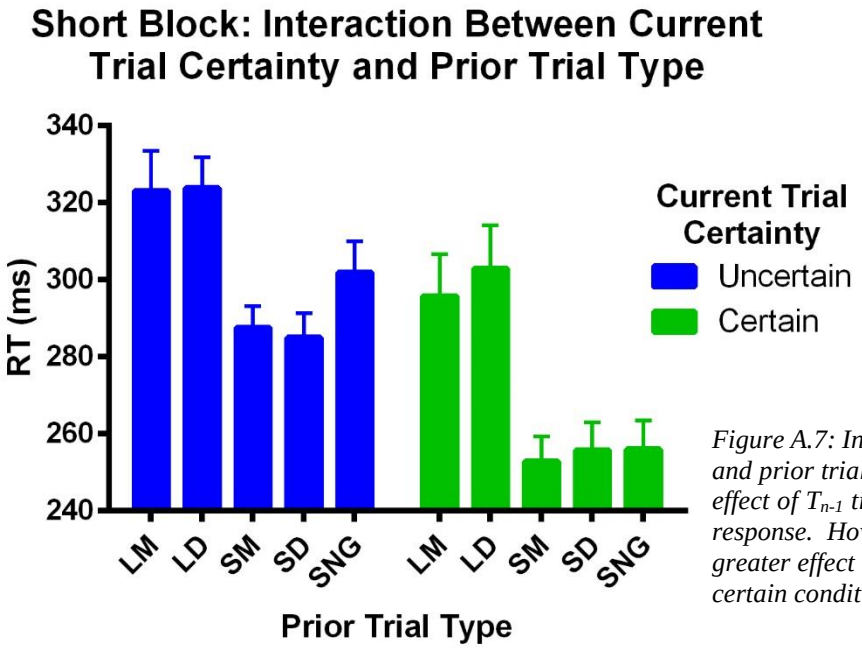


*Figure A.7: Interaction between current trial certainty and prior trial type. This interaction demonstrates the effect of $T_{n-1}$ timing on $T_n$ RTs, above the effect of $T_{n-1}$ response. However a SNG $T_{n-1}$ appears to have a greater effect in the uncertain condition (left) than the certain condition.*

Within the uncertain $T_n$ trials (figure A.7, left), there was no significant difference in $T_n$ RTs between the 'go' short (SM-SD) or long (LM-LD) $T_{n-1}$ trials (both $p \approx 1$). A SNG $T_{n-1}$ resulted in significantly different $T_n$ RTs compared to both a SD and LD $T_{n-1}$ (both $p<0.05$), but not compared to SM or LM $T_{n-1}$ (both $p>0.15$). Between the short and long 'go' $T_{n-1}$, $T_n$ RTs were a minimum of ≈37ms different (between a SM and LM), while between a short go and short NG $T_{n-1}$ $T_n$ RTs differed by a minimum of ≈14ms, and between a long go and short NG $T_{n-1}$ there was a minimum difference of ≈21ms. Within the definite $T_n$ group (figure A.7, right) there was no significant difference between $T_n$ RTs when comparing long with long or short with short $T_{n-1}$ (all $p \approx 1$), however between the $T_{n-1}$ timings, $T_n$ RTs were all significantly slower given a long $T_{n-1}$ compared to a short $T_{n-1}$ (all $p<0.01$ minimum, except between SNG and LM $T_{n-1}$, $p<0.05$). The minimum difference between short and long $T_{n-1}$ groups in terms of $T_n$ RTs was ≈40ms (between a SNG and LM). There were no significant interactions between the slowest definite $T_n$ trials (i.e. the long $T_{n-1}$) and the fastest uncertain $T_n$ trials (i.e the short 'go' $T_{n-1}$) (all $p \approx 1$).

Within each $T_n$ group in figure A.2, no RT was significantly different when preceded by a SNG $T_{n-1}$ compared to any other $T_{n-1}$ (minimum $p>0.4$) though a SNG $T_{n-1}$ was responded to ≈23ms slower than a SM $T_{n-1}$ when given a SM $T_n$, but only ≈3ms slower given a SD $T_n$.

Differences between LM and SM $T_{n-1}$ effects on SM and SD $T_n$ RTs were both ≈57ms. Finally, in the LD $T_n$ condition, though it appears from figure 7.1 that a LM and LD $T_{n-1}$ may slow $T_n$ RTs compared to a short $T_{n-1}$, this difference was not significant (minimum $p>0.4$).

The tables on the following pages represents significant differences between individual duplets in the long and short blocks respectively.

**Table A.3: Significant differences between individual duplets in the long blocks of experiment 1.4**

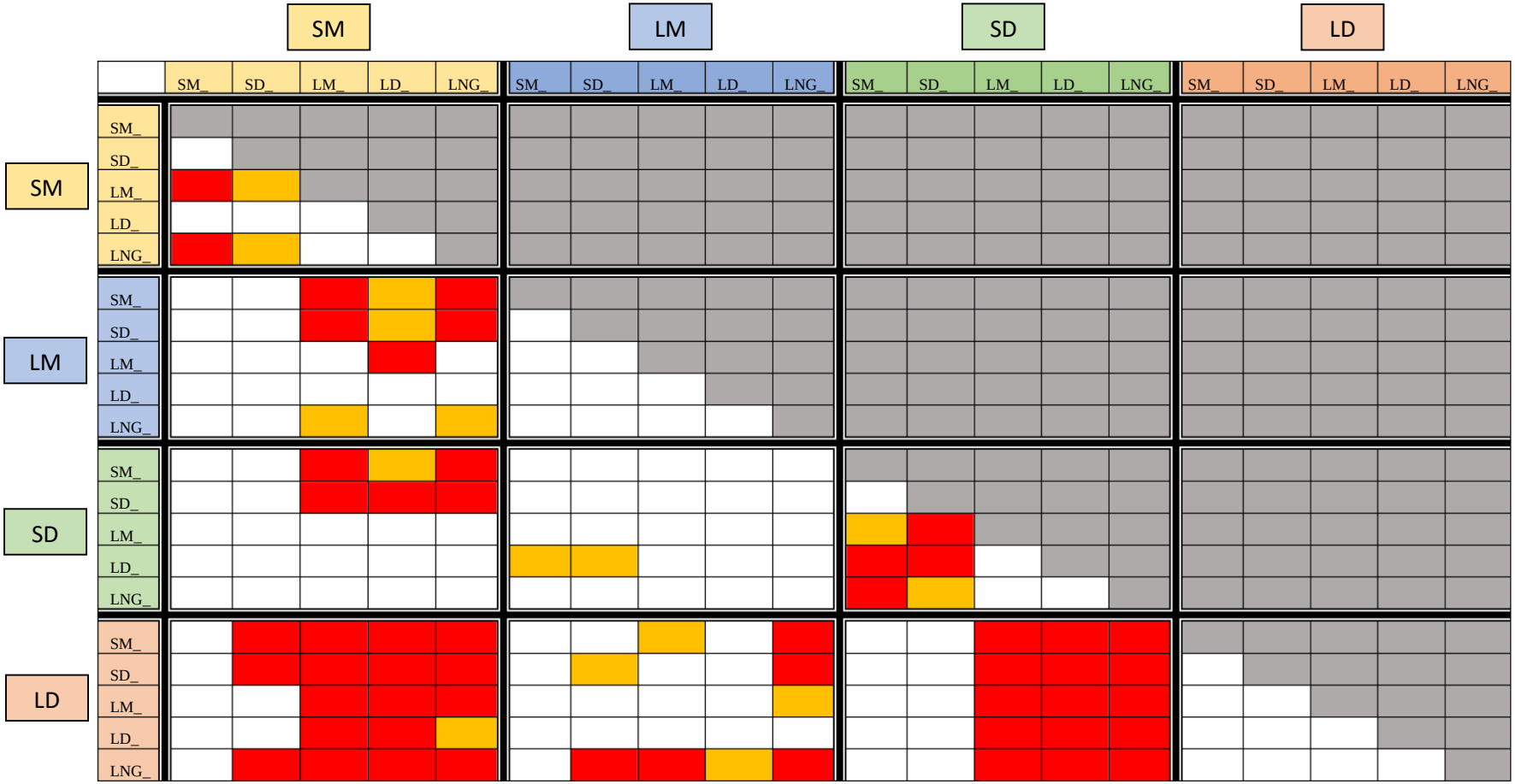


*Table A.3: Table showing the significant differences between pairs of duplets in the long blocks of experiment 1.4. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

**Table A.4: Significant differences between individual duplets in the short blocks of experiment 1.4**

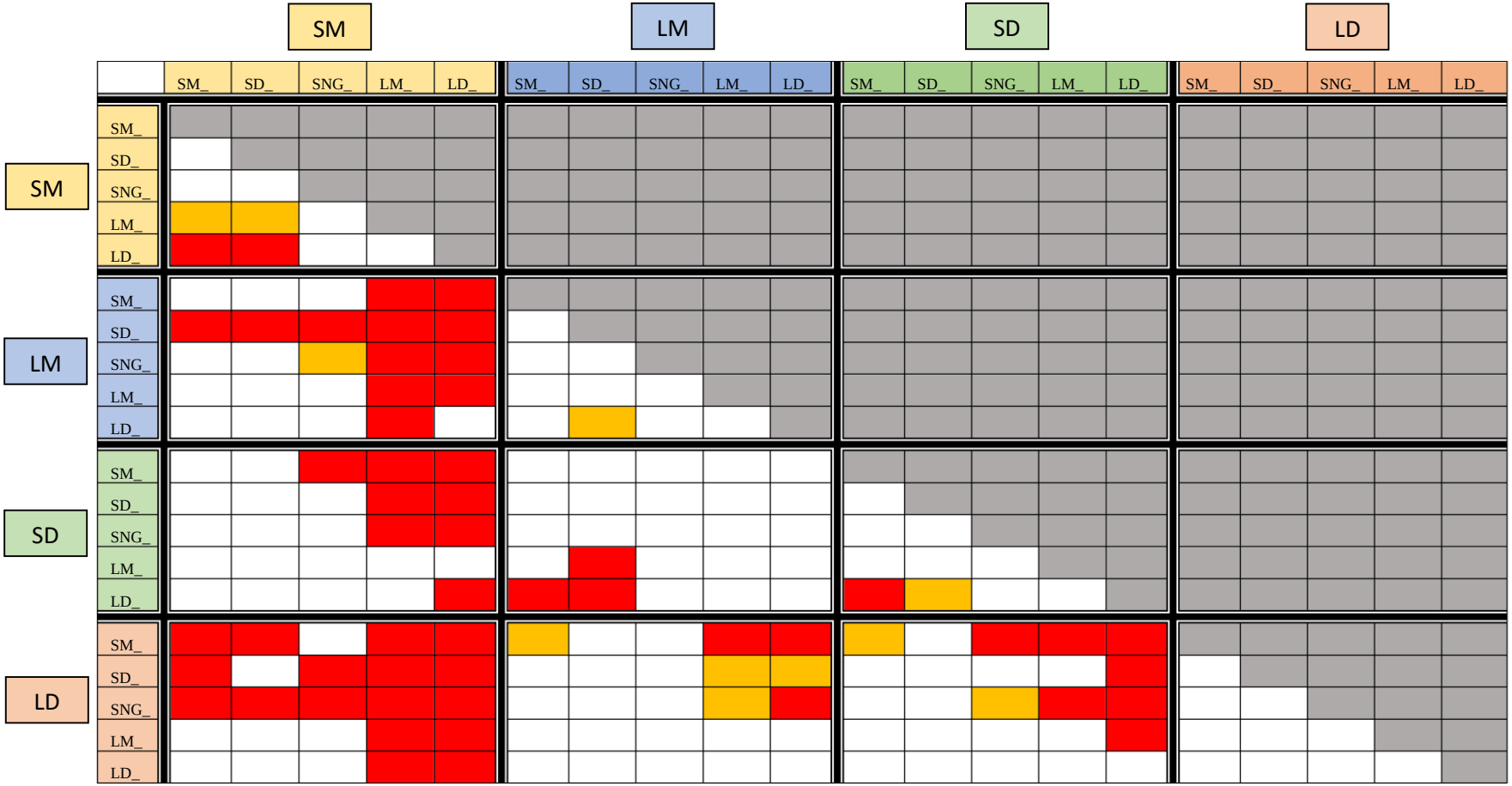


*Table A.4: Table showing the significant differences between pairs of duplets in the short blocks of experiment 1.4. Each column/row represents an individual duplet. Each group represents a current trial while each column/row within that group represents a prior trial. So, for example, the top left bog represents SM $T_n$ compared to SM $T_n$, with each $T_{n-1}$ as an individual cell within that box. Red cells represent a significant difference between the duplets (at $p<0.05$), while yellow cells indicate approaching significance ($p<0.1$).*

## A1.3 Discussion

This experiment examined the effects of foreknowledge of when a response may be required to be withheld, on $T_n$ RTs. Manipulating the time at which a NG could occur also had the effect of altering the progressive gain in knowledge regarding the probability of response in the uncertain condition. This was reflected by an increase in response probability from 33% to 50% in the long block, and from 33% to 100% in the short blocks. Further, the 33% chance of response at the short imperative time was generated differently between the two blocks; in the short block this was due to both response and timing uncertainty, while in the long block probability was generated purely by timing uncertainty. In both blocks, a SD had a 50% chance of response, due to temporal uncertainty, while LD $T_n$ were 100% temporally and response certain. This set up an interesting situation in which to examine how proactive inhibition may be fine-tuned in regards to response probability driven by imperative and/or temporal uncertainty.

### *A1.3.1 How does temporal- and response-driven uncertainty alter RTs in $T_n$?*

In the long block, there was an interaction between the timing and certainty $T_n$, indicating that RT improvements were greater as time passed in the certain condition compared to the uncertain condition. This makes logical sense; in the long block, the probability of response changed when passing the short imperative time from 33% to 50% given a white fixation, while when given a green fixation this increase was from 50% to 100%. However, if $T_n$ response probability alone determined RTs, it would be expected that LM and SD RTs should be responded to equally quickly, and that the RT difference between a SM and SD, and SM and LM $T_n$ trial should be the same. This was not the case; SD RTs were slower than LM RTs, and further a SM $T_n$ was responded to ≈23ms slower than a SD and ≈41ms slower than a LM $T_n$; a difference of ≈18ms. Note that the maxim that longer foreperiods reduce RTs (e.g. Los, 2010; Zahn and Rosenthal, 1966), cannot entirely explain this difference; a SD was responded to faster than a LM. Therefore this may imply some interaction between the different sources of uncertainty; response uncertainty slows RTs more than temporal uncertainty. Again, this supports the contentions of Jahfari et al. (2010) and Criaud et al. (2012); response uncertainty actively brakes reactions to stimuli (also see Thomas et al., 2009), while response certainty allows disengagement of proactive inhibition (at least to the level of controlling response, but without releasing to the point of not allowing temporal control). This is

interesting; one could think that this is because of the effects of $T_{n-1}$. Indeed, in experiment 1.1 there appeared to be a reduced effect of a NG $T_{n-1}$ on response-certain $T_n$ RTs. However, given that the participant had explicit knowledge of response (they are told at the beginning of the block) and that a NG was never associated with a short imperative time in the long blocks (information is accurate; people are very good at detecting probability of events and acting accordingly (Durston et al., 2002; Low & Miller, 1999; Thomas et al., 2009)) this seems unlikely; a participant should utilise prior information at the SM imperative time in a similar way to when given a SD. This has some support; a LM and LD $T_{n-1}$ are responded to in roughly the same amount of time despite whether $T_n$ is a SD or SM (see figure 7.1). However, a LNG, SM and SD $T_{n-1}$ are responded to faster in a certain $T_n$ versus an uncertain $T_n$.

This sounds complicated, but put simply it seems to indicate that the possibility of not going at the long imperative time affects RTs in the short $T_n$. Recall that Criaud et al. (2012) demonstrated that proactive inhibition may be turned off when given response certainty, however this requires time to be achieved; if an imperative was presented before inhibition had been reduced, the resultant gains in response speed from perfect foreknowledge deteriorated. Perhaps, in this situation, though given knowledge that a SNG will not occur, it is more costly to turn proactive inhibition off and then back on, than to just leave it on. Therefore readiness at the short imperative time is suboptimal, artificially inflated by a drive to optimize RTs for the (relatively more likely) long imperative. Think of it like a lightbulb.[88] Turning a light off, leaving the room for one minute, then turning it back on actually costs more than just leaving it on; the cost of restarting is higher than the gains from the short time off. Given that the timing of the long imperative is known, one could think that we could turn off proactive inhibition and then restart it straight away after the short imperative; we know when we will have to possibly withhold a response. Therefore, it may be that the time from short to long imperative is not enough to do this, and temporal certainty does not aid in this restarting of proactive inhibition. To further test this, perhaps extending the times from the short to long imperative, until we find a point where proactive inhibition may be turned off at the short imperative, may be informative.

Another possible way of viewing this is RT optimisation. To optimise SM RTs in $T_n$ it may be wise to disregard $T_{n-1}$ response information, as response is certain, as per experiment 1.1. However, if this information is discarded it cannot then be used in the long $T_n$ and therefore

[88] Don't quote me on this, it may one of those facts you hear as a child but are totally made up, but it illustrates the point.

may cause suboptimal performance at the more likely imperative time. If I eat 3/4s of a cake (which happens often), feel like a fatty, and throw the last 1/4 away, I cannot then come back five minutes later after looking to make sure I do not LOOK any different and finish the cake. It is (sadly) gone. That is why I never throw away cake. Likewise, rather than discarding information and then trying to scoop it out of the rubbish, perhaps here all $T_{n-1}$ response information is kept throughout the trial. This is in line with increased RT differences between a LNG $T_{n-1}$ in the SM versus SD $T_n$ (while LM and LD differences were ≈13 and ≈4ms faster respectively, the LNG was ≈23ms faster).

There is likely some interaction between probability and improved RTs caused by time passing. In the long block, the asymmetry of RT changes between a certain and uncertain condition may relate to improvements in foreknowledge (comparatively less RT decrement in the SM to LM $T_n$ may be attributed to improvements from 33% to 50% certainty). However, in the short blocks, the RT decreases from 33% to 100% (SM to LM) were similar to the RT decreases from 50% to 100% probabilities (SD to LD) (31ms versus 32ms respectively). The lack of difference in the short block may indicate that, though there is some RT improvement due to time passing, any change in probability will speed up responses (whether from 33% or 50% to 100%). It may be that the reduction in RTs from a response type and temporally uncertain 33% go imperative probability to 100% just so happens to decrease RTs the same amount as the change from temporal-only 50% go imperative probability to 100%. Therefore, perhaps RT differences may partially represent the improvements from absolute time being passed (i.e. we are more ready at the long time point despite the probability of response) (see, for example, the discussion by Los, 2010), balanced by decreased response uncertainty and therefore the maintenance of a higher state of response readiness (e.g. Jahfari et al., 2010).

Though, as discussed in prior chapters, a psychological refractory period does not seem likely here, there may be some effect of task switching in this case (see Ruthruff et al., 2001). In both blocks, a definite fixation occurs relatively less often (40% of trials). Therefore, perhaps this decreased probability (and less definite-definite combinations) results in slightly slower SD RTs compared to LM RTs. This possibility cannot be excluded here. To better test this possibility, in a future rendition of this type of experiment, perhaps the probability of either fixation should be balanced, with the rules associated with each maintained.

The interaction between the timing of $T_n$ and the $T_{n-1}$ trial type, found in both blocks, sheds further light on the above discussion. In both blocks, there appeared to be an absolute

effect of the timing of $T_{n-1}$, regardless of $T_{n-1}$ response, with a long $T_{n-1}$ resulting in slower RTs in $T_n$. Though it may be thought that this effect is due to the inclusion of both certain and uncertain $T_n$, in which a prior NG appears to have a reduced effect, this does not appear to be the case; a NG $T_{n-1}$ did not appear to have an effect separate to the timing of that trial, either in the short or long block. This does not appear congruent with prior research in which a prior trial NG slows current RTs when at the same imperative time (e.g. prior studies in this thesis, and Los (2013)). Further, prominently in the short block, response in a long $T_n$ is faster when preceded by a short trial in $T_{n-1}$ than a long trial in $T_{n-1}$. This also seems at odds with prior research where $T_{n-1}$ has little effect on a long $T_n$ unless passed over or given a NG response at the long imperative time.

The underlying cause of this requires further research. Trials prior to $T_{n-1}$ do not appear to adequately explain effects in the LM $T_n$; a LNG $T_{n-1}$ is the only effective alteration of $T_n$ according to prior findings. Therefore, longer RTs in a LM $T_n$ given a long $T_{n-1}$ but not a LNG $T_{n-1}$ appears counter-intuitive. However, it is possible that this pattern is due to proactive inhibition disengagement. In the short blocks, proactive inhibition may be turned off after the short time frame has passed, however, as per Criaud et al. (2012), this may take time to occur. Perhaps, in the long $T_n$ we are still caught in the middle of turning off proactive inhibition, inflating RTs and, as proactive inhibition is being turned off, there is some inhibitory effect on the long trace, slowing RTs when given a long imperative in $T_n$. Why then does a LNG, in the long block, not have an effect on long $T_n$ RTs? Perhaps, counter to the prior discussion, we are trying to increase our level of proactive inhibition; after having our foot off the brake for the short $T_n$ we are trying to find the pedal, and actively brake our response (as discussed by Jahfari et al., 2010). This ‘restarting’ proactive inhibition slows our RTs to a ‘go’ long imperative in $T_{n-1}$ and the effortful process of time-pressured inhibition re-initiation has an effect on the trace of the long imperative time. To examine this proposition, and compare it to that discussed above (where inhibition is not disengaged at the short imperative time due to the possibility of it being required at the long imperative time) neuroimaging techniques may be useful. There are several neural correlates of active braking and inhibition (see Jahfari et al., 2010; Levy and Wagner, 2011; Smittenaar, Guitart-Masip, Lutti, and Dolan, 2013, for review, see Aron, 2011); analysing the temporal activation/deactivation of these as possible future imperatives are changed is an interesting future direction for this research.

## Appendix 2: Evidence for the Multiple Trace Theory

Though not the focus of the thesis, within the RT experiments, there is some support for the multiple trace theory as proposed by Los et al. (2014). This also explains why, in the current research, LM_LM and LNG_LM RTs were not found to be different, a finding that is difficult to account for without considering the effects of trials prior to $T_{n-1}$. The current research provides some support for the effects of trials prior to $T_{n-1}$ however. Short of controlling for trials further back than $T_{n-1}$, the method of contrasting different blocks with different rule sets is likely to supply the strongest support of the multiple trace theory. The multiple trace theory states that current-trial expectations are a weighted sum of the prior events affecting the relevant trace, such that events further back than $T_{n-1}$ may alter trace strength, however the further back the trace-affecting event, the less weighting this has. When a trace is not involved in the current trial, there is no effect on that trace, other than time-related decay back to some asymptote. For example, a short trial does not have an effect on the traces associated with the long imperative. In the current trial, the memory trace of each prior imperative moment affects RTs at that imperative moment.

In experiments 1.2 and 1.3 I commented on the RT differences between the long and short blocks. In experiment 1.2 there was no significant RT difference between blocks, while in experiment 1.3 SM RTs were significantly slower in the long block.[89] Let us examine experiment 1.3 block differences before returning to experiment 1.2. Firstly, RT differences between the two blocks were specific to the SM trials, LM RTs were not significantly different. Therefore, this was not some epiphenomenon in which a long block was intrinsically different than a short block, it has a specific effect on SM trials, increasing RTs in the long block-SM trials by ≈12ms compared to the short block-SM trials. One possible explanation for this is that perhaps trials prior to $T_{n-1}$ affect $T_n$ RTs. If a long $T_{n-1}$ affects a short $T_n$ and not the other way around (as per the trace-conditioning theory, and as reported in, for example, Los, 2013; Steinborn et al., 2008), then, in both blocks a LNG is the only trial type that will result in comparatively slower long imperative RTs. For SM RTs however, 80% of long block trials will result in comparatively slower RTs (SNG, LM, LD, LNG) while in the short block, only 60% have this effect (SNG, LM, LNG). Thus, if trials further back than $T_{n-1}$ affect $T_n$ RTs, then, even when controlling for $T_{n-1}$, SM $T_n$ RTs are likely to be slower in the long block where there

[89] This is not simply explained by there being more long trials in the long block; those trials not included in both blocks were excluded in this comparison.

are more 'slowing' trials, compared to the short block. This is also congruent with short and long blocks having roughly similar LM $T_n$ trials; short and long $T_{n-1}$ do not have separable effects on LM $T_n$ (again according to the findings by Los, 2013), and therefore the relative frequencies of short and long trials will make no difference.

In experiment 1.2 there was no significant difference in average RTs between the short and long blocks. If we consider the multiple trace model, then this may imply that trials before $T_{n-1}$ equally affect SM and LM RTs irrespective of the block. One's initial thought may be that, given that the long block has more trials in which the short imperative time is bypassed (four compared to two out of six), if there is an effect of trials prior to $T_{n-1}$ then the short block should have significantly faster RTs than the long block, and therefore this may act as evidence against the multiple trace theory. However in the long block there were five trial types that slow SM $T_n$ RTs compared to a SM $T_{n-1}$ within the trace-condition theory; a LM, LNG, LTG, LTNG and SNG. In the short blocks, there were four such trials; a LM, LNG, SNG and STNG. Given that trials prior to $T_{n-1}$ are theorised to have a reduced effect on $T_n$ RTs the further back they are, a difference of one fifth of trial types may not result in significantly different SM $T_n$ RTs between the two blocks.[90] This seems especially probable considering that (though not significant), LT trials appeared to be responded to slightly quicker than non-blue long trials when given a SM $T_n$, possibly due to the foreknowledge of the SM imperative time being passed and therefore partially disengaged. This requires further research, or an increased sample size to show significance, however if this difference is significant, perhaps blue trials may have a smaller weighting in temporally affecting traces, and as such a long trial has two reduced trace effectors (i.e. LTG/LTNG).

LM RTs in experiment 1.2 require further explanation; LM $T_n$ RTs should only be significantly slower than other trial combinations when proceeded by a LNG $T_{n-1}$ according to the trace condition model. However in the long block, there were double the trials which resulted in response inhibition at the long imperative time; the long block had both a LNG and LTNG, while the short block only had the LNG. Despite this, in the long block RTs did not appear to be any slower in $T_n$ when proceeded by ether a LNG or any other $T_{n-1}$ trial. This was also true in the short block, likely due to increased variability of LM RTs when given a LNG $T_{n-1}$. These findings may implicate a problem in the trace conditioning theory itself. However,

[90] One interesting possibility may be that timing or response dynamics of trials have a stronger 'lasting' effect, and stay in the memory trace better than the other. For example perhaps $T_{n-2}$ temporal dynamics are still fairly strong effectors of $T_n$ RTs, while $T_{n-2}$ response type has basically no effect.

if utilising trials further back than $T_{n-1}$ an explanation may be proposed. In the long blocks, four trials should not increase LM RTs (SM, SNG, LM, LTM). This may result in an attenuation of the LNG effect; the probability of a LNG $T_{n-1}$ being proceeded by a trial which does not affect LM RTs is relatively higher than a LNG, and so may attenuate the effects of a LNG $T_{n-1}$ on LM RTs. In the short block, there are even more trial types that do not affect LM RTs (SM, STG, SNG, STNG, LM), therefore again LNG_LM RTs may be more variable. In the short block specifically, time since the last long $T_n$ is likely relatively higher and therefore, combining asymptotic decay with no overt effect on LM RTs results in more RT variability and therefore no significant difference in RTs.

Though presented in the appendix, the final experiment also found a difference between blocks. In the long block, a SM, LM, LNG, SD, and LD were possible, while in the short block a SM, SNG, LM, SD, and LD were possible (see appendix one for further discussion). Briefly, in this experiment LM RTs were significantly different across blocks, with the short block-LM RTs being ≈19ms faster than the long block-LM RTs. The other $T_n$ trials (SM, SD, LD) were not significantly different across the blocks. In the long block, as mentioned above, short $T_n$ RTs should be slower than in the short block due to a higher probability of $T_{n-2}$ and further back being a long trial. However, in this case, it may be that the added probability of a long trial is counterbalanced by the absence of a SNG in the long block, and vis-versa in the short block. In the long block there are three trial types that slow SM RTs (LM, LD, and LNG) while in the short block there are again three trial types that slow SM RTs (SNG, LM and LD). The inquisitive reader may then ask, why are SD RTs not affected? I am glad you asked; recall in chapter four where I have put forward the finding that a LNG in $T_{n-1}$ was responded to quicker in the SD $T_n$ compared to other long $T_{n-1}$ trials (at least, the LNG $T_{n-1}$ did not result in significantly slower RTs compared to a SM $T_{n-1}$, while LM/LD $T_{n-1}$ did). Perhaps this reduction of the effect of passing an imperative time in $T_{n-1}$ when given a LNG is responsible for no difference in SD $T_n$ RTs; in the SD $T_n$ the only trials less recent than $T_{n-1}$ that make a large enough difference to still have an effect on $T_n$ RTs are LM and LD trials. The difference between the blocks in LM RTs also makes sense; in the short block there are no trials which should affect LM RTs; the LM imperative time is never passed, nor is there an inhibitory signal associated with it. In the long block, a LNG is possible, therefore resulting in slower long-block LM RTs compared to short-block LM RTs.

One further detail of the multiple trace theory requires discussion here. It is assumed that a memory trace is established for each repetition of a trial. In Steinborn et al. (2009) it was

found that the sequential foreperiod effect was attenuated when the trial initiation signal was varied across modalities. This may indicate that varying the trial initiation signal instantiates two separate traces; a green trial trace may be stored separately from a white trial trace for example. However in these experiments this does not appear to be the case; SD and SM RTs in experiment 1.1 were equally affected by either LD or LM $T_{n-1}$. Therefore it may be that the trials are considered 'the same' in terms of the associated traces. However, as mentioned above, while a temporally uncertain, response certain trial (i.e. green fixation in experiment 1.1) may affect the short trace when passing the short imperative moment, it may be that the short trace is not as affected when the trial passing the short imperative moment is initiate by a temporally certain, response uncertain trial (e.g. blue fixation). This reduction of effect cannot be entire (or indeed may not even be present), however if there, may indicate a partial disengagement of the short trace time point in the temporally certain long trial. This seems to be supported by Los (2013); even when given foreknowledge of an upcoming NG imperative, there is still some effect on the trace of the short imperative. If the trace is affected by inhibition, and inhibition may take time to disengage, as found by Criaud et al. (2012), then perhaps this reduced, but still present, effect is due to non-immediate disengagement of the short trace, or disengagement of the associated inhibition taking time to occur.

This does not provide direct evidence of the multiple trace theory, and is relatively speculative, based on probabilities, however does seem to agree with the tenants of the theory. To further analyse this contention requires investigations involving $T_{n-2}$ and further back effects on $T_n$ RTs. Another option for furthering this discussion is the creation of a model which utilises $T_{n-1}$ probabilities, weighted by their effect on $T_n$ RTs to attempt to replicate these findings. Modelling may then suggest appropriate ways to further test the multiple trace theory, for example how best to alter probabilities across blocks to ensure comparative experimental evidence. However, the concept of the multiple trace theory itself is based on fairly robust cognitive principles. For example multiple trace theories have been applicable in explaining visual search and recognition (Chun & Jiang, 1998; Huang, Holcombe, & Pashler, 2004) lexical memory tasks (Ans, Carbonnel, & Valdois, 1998; Hintzman & Block, 1971; Logan, 1990)[91] and various memory type storages (Moscovitch et al., 2005).

[91] An interesting related proposition is that memory strength may act as a 'clock' (Staddon, 2005). Further, there are some challenges to multiple trace models, for example see Graham (1999) regarding semantic memory, however this is beyond the current discussion.

# Appendix 3: Ethics Approval



MACQUARIE University

Samantha Baggott <samantha.baggott@mq.edu.au>

## Amendment request for 5201200035 and 5201200036

**Ethics Secretariat** <ethics.secretariat@mq.edu.au> Thu, Apr 9, 2015 at 2:26 PM
To: Samantha Baggott <samantha.baggott@mq.edu.au>
Cc: Anne Castles <anne.castles@mq.edu.au>, Shiree Heath <shiree.heath@mq.edu.au>

Dear Sam,

RE: "Recognising, naming, classifying and understanding visually and/or auditorily presented stimuli" (Ref: 5201200036-Student & 5201200036-Staff)

Thank you for your recent correspondence requesting an amendment to the above studies. The following personnel changes have been approved:

1) Addition of Mr Jordan Wehrman (MRes Year 2 student) and Ms Bianca Slater (intern) to project Ref:5201200035 (Student).
2) Addition of Ms Bianca Slater (intern) to project Ref:5201200036 (Staff).

Please do not hesitate to contact us if you have any queries regarding this approval.

Kind regards,
Eliza Harrison
[Quoted text hidden]

MACQUARIE UNIVERSITY

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8 July 2014

Dr Paul Sowman
Department of Cognitive Science
Faculty of Human Sciences
MACQUARIE UNIVERSITY NSW 2109

Dear Dr Sowman

RE: *Stimulating and Recording the Brain in Studies of Cognitive Control*

Thank you for submitting the above application for ethical and scientific review. Your application was considered by the Macquarie University Human Research Ethics Committee (HREC (Medical Sciences)) at its meeting on 29 May 2014 at which further information was requested to be reviewed by the Ethics Secretariat.

The requested information was received with correspondence on 25 June and 7 July 2014.

I am pleased to advise that ethical and scientific approval has been granted for this project to be conducted at:

- Macquarie University

This research meets the requirements set out in the *National Statement on Ethical Conduct in Human Research* (2007 – Updated March 2014) (the *National Statement*).

**Details of this approval are as follows:**

**Reference No:** 5201400585

**Approval Date:** 8 July 2014

The following documentation has been reviewed and approved by the HREC (Medical Sciences):

| Documents reviewed | Version no. | Date |
|---|---|---|
| Macquarie University Ethics Application Form | 2.3 | July 2013 |
| Short Protocol | | |
| Correspondence from Dr Paul Sowman responding to the issues raised by the HREC (Medical Sciences) | | Received 25/6/2014 & 7/07/2014 |
| Flyer entitled *Stimulating and recording the brain in studies of cognitive control* | 1 | |
| Newspaper Advertisement | 1 | |
| Study advertisement in SONA-system | 1 | |

| | | |
|---|---|---|
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (tCS and Stuttering) | 3 | July 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (tCS and Non-Stutterers) | 3 | July 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (TMS: Control Subjects) | 2 | June 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (TMS and Stuttering) | 2 | June 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (Synaesthesia Research) | 2 | June 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (EEG: Control Subjects) | 2 | June 2014 |
| MQ Participant Information and Consent Form (PICF) entitled *Stimulating and recording the brain in studies of cognitive control* (EEG and Stuttering) | 2 | June 2014 |
| Questionnaire for New Stuttering Participants – Adults | 1 | Click here to enter a date. |
| TMS Screening Form | 1 | 20/12/2013 |

This letter constitutes ethical and scientific approval only.

**Standard Conditions of Approval:**

1. Continuing compliance with the requirements of the *National Statement*, which is available at the following website:

http://www.nhmrc.gov.au/book/national-statement-ethical-conduct-human-research

2. This approval is valid for five (5) years, subject to the submission of annual reports. Please submit your reports on the anniversary of the approval for this protocol.

3. All adverse events, including events which might affect the continued ethical and scientific acceptability of the project, must be reported to the HREC within 72 hours.

4. Proposed changes to the protocol must be submitted to the Committee for approval before implementation.

It is the responsibility of the Chief investigator to retain a copy of all documentation related to this project and to forward a copy of this approval letter to all personnel listed on the project.

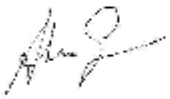
Should you have any queries regarding your project, please contact the Ethics Secretariat on 9850 4194 or by email ethics.secretariat@mq.edu.au

The HREC (Medical Sciences) Terms of Reference and Standard Operating Procedures are available from the Research Office website at:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics

The HREC (Medical Sciences) wishes you every success in your research.

Yours sincerely



**Professor Tony Eyers**
Chair, Macquarie University Human Research Ethics Committee (Medical Sciences)

This HREC is constituted and operates in accordance with the National Health and Medical Research Council's (NHMRC) *National Statement on Ethical Conduct in Human Research* (2007) and the *CPMP/ICH Note for Guidance on Good Clinical Practice*.