Duration Judgement Deficits and Schizophrenic Liability: What Drives Them and Can They Be Manipulated?

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Declarations

This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree.

Signed M.A. Hopkins

Date 07/11/2023

This thesis is the result of my own investigations, except where otherwise stated. Other sources are acknowledged by footnotes giving explicit references. A bibliography is appended.

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Acknowledgements

I was a precocious child, with a thirst for knowledge. One of the first questions I can remember asking my parents, at 7, was ‘how do you measure the present moment’? Since then, I have perpetually asked existential questions related to time, and existence; and have attempted to answer these questions by first reading Psychology, and secondly, by having an unhealthy addiction to mathematics and physics. After all, so the natural scientists would have us believe, mathematics is the language of the Universe though, I am unconvinced by that. Coupled with my knowledge of differential psychology, and the fact that time is relative, as opposed to absolute, I wanted to understand the biological reason for why some people experience time differently to others. As a result of this interest, I completed a Psychology degree to help me understand people. Little did I know that the more I learned about people, the less I understood about them! I consequently read Cognitive Neuroscience for my graduate studies at Swansea University to take a more biological twist on psychology. Come September 2019, I still had questions but, the questions became more complex and my knowledge, at the time, was not complex enough to help answer these questions. I remember, one night, I was smoking a cigar, starring at the stars, musing to myself that time perception is the fundamental property of existence. That prompted me to consider a Ph.D. investigating Time Perception and I emailed Prof. Phil Reed, who introduced me to Dr. Irene Reppa. I am eternally grateful to Swansea University for affording me the opportunity to study Time Perception. It is, by far, the best University in Wales. I initially intended to do my M.Sc. here, and join the British Army, but four years later, I am still here; and looking forward to further satisfying my precocious nature.

The idealists would have you believe that the modus operandi of psychology is to find a cure to the diseases of the mind however the realists posit that psychologists ‘treat’ the

behaviour that arises from the condition; as opposed to the condition itself, of which a cure is often seen as idealistic at best, misinformed at worst. But I ask, surely, psychology should aim to fully understand the trammels of mental illnesses and attempt to cure them to make people's lives better. Perhaps I am but an idealist in a world of realism but, I make absolutely no apology in hoping to contribute to a cure to the 'Dante's Inferno' of mental illnesses: schizophrenia. I hope that my work, which I present you with, contributes to the literature in understanding schizophrenia and eventually, building a consensus to finding a cure for this condition. My thesis has been born from existential curiosity but primarily, it seeks to help us delineate the timing deficits that contribute to schizophrenia. Once again, I make absolutely no apology for wanting to help people in society.

I am eternally grateful for the chances I have been afforded; and I must express my sincere gratitude to a variety of people, and organisations, who have facilitated my chances. My first thanks go to my supervisors; Irene and Phil. Irene always had faith in my work and kept me focused on key work related to time perception; as well as constantly keeping me upbeat, even in difficult times for all of us, given a global pandemic. Phil, for his insightful questions and comments on my work, as well as his expertise in statistical methodology and behavioural psychology has provided critical support, especially in his encyclopaedic knowledge of Psychology. With Irene and Phil, I would not have been able to complete my Ph.D. I am also eternally grateful to the other members of this department for helping me. These include John, Gabriella and Mark who were always willing to help; and often listened to me rant about politics, music, and phrenology.

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> "The important thing is not to stop questioning. Curiosity has its own reason for existence. One cannot help but be in awe when he contemplates the mysteries of eternity, of life, of the marvellous structure of reality. It is enough if one tries merely to comprehend a little of this mystery each day."
>
> *Albert Einstein*

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General Abstract

Duration judgements are a critical part of human life, and interest in this criticality is as ancient as the interest in the concept of time itself (e.g., Grondin, 2008). Judging the duration of an event or of passing time is also highly malleable, subject to both individual differences and environmental factors, e.g., heat or music. Retardation of duration judgements (e.g., judging the length of durations) are a common feature of schizophrenia (Elvevåg et al., 2003; Carroll et al., 2008; Carroll et al., 2009; Reed & Randell, 2014) however, there are patent difficulties in establishing the fundamental cause of timing deficits, in schizophrenia; which remains poorly understood. For example, it is well known that pharmaceutical interventions (e.g., haloperidol) can modify duration judgements (Rammsayer, 1990) in both normal adults and schizophrenic patients; however, external stimuli, such as an auditory Click Train (e.g., a series of clicks presented before stimuli), can also be used to modify duration judgments (albeit at a smaller effect-size than pharmaceutical interventions). Whether a Click Train has a similar effect as pharmacological interventions in schizophrenia has never been investigated and thus, there remains a paucity of research in this respect. To counter the potential effects of medication on duration judgements, schizotypy can be used as a useful construct for schizophrenia liability to investigate timing deficits in schizophrenia; as well as Click Train effectiveness in manipulating timing durations. Five experiments were conducted to examine what, fundamentally, contributes to timing deficits in schizotypy and to assess whether the Click Train can manipulate duration judgements in schizotypy. The first two experiments used the popular temporal bisection tasks in both visual and auditory modalities: as well as using a Click train The second set of experiments utilised the temporal generalisation tasks, using both visual and auditory modalities; as well as the Click Train. The final experiment used the classic estimation task,

in which subjects had to estimate how long a video lasted. The first set of experiments are indicative of High Schizotypy subjects showing better precision for auditory durations, and that the Click Train manipulated judgement durations in subjects, irrespective of schizotypy level. The second set of experiments suggested memory distortions are present in Schizotypy and could be driving the timing deficits reported. The final experimental also implicates better precision in identifying durations in High Schizotypy. The Click Train manipulated duration judgements, irrespective of schizotypy level. Overall, the current thesis provides evidence that (1) timing deficits in schizotypy (and potentially, schizophrenia) are the result of better precision in identifying durations (e.g., less variability) and (2)., that the Click Train can be used as an effective tool in manipulating duration judgements in schizotypy and potentially limiting some of the effects of timing deficits in schizophrenia.

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Chapter one: What is the Perception of Time?

"Time is what happens when nothing else happens"

Richard Feynman (Fenyman, Leigthon & sands, 2011)

1.1 What is Time perception?

Feynman, in his lectures on Physics, muses to himself that the Universe is an either/or dichotomy: there is nothing or there is time. Feynman's words imply time is a fundamental principal of existence though he contends that his words do not convey the answer to the question of what time is. Feynman captures the basic fundamental experience of time in the human being. Time, as a concept, is something we are aware exists, but scarcely do we consider its importance in guiding behaviour. Meck (1996) argued that, despite humans not typically aware of time, our perception of time is a fundamental force in human behaviour. Meck (1996) further expounds that to perceive durations (e.g., the passing of time), an internal clock is required to register an initial duration, and a criterion in memory against which this sensory input can be compared (Meck, 1996). For example, how would you know if the kettle overboiled without (a) having an ability to perceive the duration of the kettle's boil and (b) having a criterion to compare the kettle duration with? The answer is simple and unsatisfying: you would not know as you would not have the ability to perceive duration or compare this perceived duration with a criterion from long-term memory. This reasoning demonstrates how time is a multi-component structure; with many cognitive functions collating resources to perceive the duration (Meck, 1996). Moreover, the fact that humans are

intricately variable, and actively perceive time (Wearden, 2016), alludes to the notion that individual differences might contribute to variability that arises in judgement durations.

Individual differences that potentially contribute to duration judgement variability range from gender (Hancock, Vercruyssen & Rodenburg, 1992) to schizophrenia (Carroll, Boggs, O'Donnell, Shekhar & Hetrick, 2008) as well as a whole host of individual differences (Wearden, 2016) that fill the shelves of the Bodleian library. However, schizophrenia is a particularly interesting candidate for duration variability given the complex nature of the disorder. Whilst experimental psychology has demonstrated duration judgement is impaired in schizophrenia (Amdeo et al., 2022; Ciullo et al., 2016; Martina et al., 2014) questions remain whether duration judgements are clock specific or part of a general cognitive deficiency that is present in schizophrenia (Thones & Oberfeld, 2017)

Investigating how schizophrenia affects time perception presents its own difficulties (e.g., Carroll et al., 2008) such as antithetical findings (Elvevåg, McCormack, Brown, Weinberger & Goldberg, 2003; Reed & Randell, 2014), and confounding variables associated with schizophrenia, such as medication and the episodic nature of the condition (Reed & Randell, 2014). In addition to several findings suggesting schizophrenia influences duration judgements, research also indicates that attention, memory, arousal, and external factors, (e.g., Wearden, 2016; Wearden, Win & Philpott, 1999) arbitrate the perception of time, which further complicates an already complex construct in attempting to investigate how duration judgement variate in schizophrenia.

The aim of this thesis is to investigate how timing deficits in schizophrenia arise by focusing on schizotypy. Pointedly, I shall focus on what components of Gibbon's (1977) Scalar Expectancy Theory (SET) appear to drive timing deficits in schizotypy and schizophrenia, which can be explained parsimoniously. Furthermore, I shall investigate

whether an external manipulation can modify timing in schizotypy and schizophrenia, respectively.

1.2 Investigating schizophrenia and time perception: Why?

Schizophrenia is a debilitating mental illness, characterised by a range of cognitive, behavioural and emotional deficits (DSM-5, 2022), in which timing deficits, according to the *Cognitive Dysmetria* model of schizophrenia (Andreasen et al., 1999) represent a core deficit that provokes a cascade of effects (Amadeo et al., 2022; Elvevåg et al., 2003; Escelsior et al., 2019). These timing deficits are presumed to lead to secondary disturbances, such as cognitive and perceptual processes (Andreasen, Paradiso & O'Leary, 1998; Carroll et al., 2008). Since the perception of time is said to be fundamental to behaviour (Meck, 1996) deficits in duration judgements manifest as deficits in observable behaviour (Reed & Randell, 2014). Therefore, timing deficits associated with schizophrenia could have a marked effect on behaviour, which in turn, could affect medication regimens and general quality of life of patients suffering with schizophrenia. The first aim of this thesis is having a clearer understanding of which cognitive processes give rise to timing deficits in schizophrenia. The second aim is whether an external factor, such as a clicker train, can manipulate timing in schizophrenia.

The confounding variables, such as cognitive dysmetria, and timing deficits associated with schizophrenia are well known (e.g., Carroll et al., 2008; Reed & Randell, 2014). To overcome such confounding variables arising from schizophrenia, schizotypy provides a useful sub-clinical analogue (Reed & Randell, 2014) associated with schizophrenia. Consequently, by investigating timing deficits in schizophrenia, and identifying what components of Scalar Expectancy Theory interacts with schizophrenia,

coupled with investigating whether trains of clicks, of which there is evidence suggesting trains of clicks can manipulate duration judgements in people (Wearden, Win & Philpott, 1999) might help formulate an understanding of how timing deficits impacts schizophrenia, and how non-pharmaceutical interventions (e.g., trains of clicks) might help alleviate the secondary symptoms that arise, due to duration judgment deficits in schizophrenia. Prior to any investigation on how time perception deficits arise in schizophrenia, I first define what is meant by 'time perception'; as well as reviewing the internal clock models that model time perception; critical timing, and retrospective paradigms; and discuss some of the tasks used to measure time perception to have a clear conceptual understanding of what is meant by time perception, and how to model it.

1.3 History of Time research

1.3.1 Ancient History

Given the topic of this thesis, it is helpful that concepts, such as 'time perception' and 'internal clock' are clearly defined by exploring the history of time perception, and the development of internal clock models. Time perception is an ancient concept, which dates to the beginning of the human experience but, how did a human interest in the perception of time first arise? The concept of the perception of time appears to be an endeavour originating in the prehistoric world, with prehistoric humans. Roeckelein (2008) argues that the crafting of tools by Australopithecus (some two-million years ago) demonstrates how these ancient humans potentially anticipated the needs of some immediate present event, such as the need to build shelter because of an adverse immediate environment; and an imagined future, such as crafting shelter that could withstand the harsh environment of the ancient world. Roeckelein (2008) interpreted these anecdotes as implying that Australopithecus had a concept of time and could actively perceive time, as indexed by their crafting of tools

(Roeckelein, 2008); demonstrating how behaviour of Australopithecus was mediated by their perception of time, which implies, at least on a larger scale, they had the ability to perceive durations. This basic ability of time perception influencing behaviour in the past, present and future, has remained unchanged since Australopithecus' crafting of tools for a pressing present situation, and an imagined future event (Goudsmit & Claiborne, 1980) and quickly applied to other *homo* species. The emergence of the first humans, in China, some five-hundred thousand years ago, started using fire, which, as stated in Grondin (2008), shows they were perspicacious enough to keep supplies of fuel for fire; further demonstrating humans' concept of time perception from *Australopithecus* to *Homo sapiens*, and keeping with Meck's (1996) position that to be able to perceive time, you must first be able to judge a duration, and compare that duration to experience of a similar event in memory.

However, it was not just environmental needs in which duration judgement played a pivotal role in behaviour. Additional evidence of how time perception influenced human behaviour is to be found in the development of cultural tools, such as record keeping dating back to at least the 8th millennium BC (Schmandt-Besserat, 1982) in Tell Aswad, Syria (Contension, 1972). Writing and record keeping illustrates how time perception encapsulates the past, the present and future: the present, in writing an event down; the past, in writing about a past event; and the future, in writing about a potential future event. These examples serve to epitomise how the perception of time is essential to communicate how an event occurred, when an event occurred, and what future events could occur. Roeckelein (2008) argues that the perception of time is a necessity and without an ability to perceive – and record – time, society would not have been possible. Perhaps an advanced perception of time is the reason why organisms, such as *Homo sapiens* and other animals evolved, however, such a debate is beyond the scope of this thesis.

Time perception allowed the human to craft tools for an imagined future, or a pressing present situation; and allowed the recording of past events, which would not have been possible if there was an absence of time perception. However, not only is the recording and perceiving of time important, but the measuring of time is equally important, as humans must have the means to measure time (Wearden, 2016) to make sense of this abstract notion: *time.* Meck (1996) argued that to perceive time, we must have 'something' to compare a signal to. Therefore, the ability to measure durations dictates that there must be a previous duration to compare it to, held in memory and gained through previous experience. For example, if we presume that water takes 10 minutes to boil, we will have a rough idea that we must boil the water for 10 minutes for it to reach boiling point, due to a previously experienced duration that has been stored in memory. However, when did humans gain the ability to measure durations?

The earliest known attempt to measure and record durations is linked with the observations of cosmological and celestial events, such as the length of time it takes for the Earth to rotate around the Sun, or the position of certain stars in the night skies. This cosmological aspect of measuring time (Whitrow, 1972) was central to the belief of the Chaldeans, who inhabited Babylonia (Viglas, 2018) from around 626BC. The recording of time can be traced back to the Sumerian civilisation, who were the first to utilise cuneiform script (i.e., written language), which made the recording of their past achievements a possibility (Adam & Al-Ansari, 2020; Dalby, 1986). The Sumerians also developed the lunar calendar (Barton, 1913), around 4500 BC, which used the position of the star, Sirius, in the night sky to measure a year (Parker, 1974). The development of the calendar demonstrates the importance ancient civilisations placed on future events; where the calendar allowed such civilisations to perceive an imagined event, in the future on the pretext of past (e.g., the Sumerians recorded how long Sirius took to move its position around the sky and used this to

measure future durations). The calendar was further refined by the ancient Egyptians, who developed the notion of the lunar month, which was further subdivided into four 'weeks' based on each quarter of the Moon (Parker, 1974). This ancient calendar gave rise to the familiar 24-hour day and 365-day year (Goudsmit & Clairborne, 1980) which is still based on cosmological events (e.g., the rotation of the Earth around the Sun).

Therefore, the measuring of durations is based upon previous experiences (Meck, 1996) and led to the development of cultural tools to measure durations. For example, the Sumerians observed how long Sirius took to change position across sky, and established a 'remembered duration' then, subsequently measured durations could be compared to this remembered duration. The argument that Roeckelein (2008) presented (e.g., that human society would not have been able to formulate without the ability to measure durations) can, at least at the bear minimum, be seen to have some credence. As without the ability to perceive – and compare – durations, even something as basic as judging how long a day should be would not be possible (Grondin, 2008; Wearden, 2016).

The development of cultural tools, such as writing systems and calendars, demonstrates how ancient civilisations had both a concept and perception of time and developed a system to record present events (e.g., writing systems), reminisce on past events (e.g., diaries) and predict future events (the lunar calendar), such as rainfall (Grondin, 2008). Ancient civilisations' (such as the Sumerians and Egyptians), development of these tools indicates how time perception is essential for behaviour, and guides humans' actions (Meck, 1996). Ultimately, ancient human civilisations were preoccupied with timing, and devised a system to measure and perceive time (Goudsmit & Clairborne, 1980); however, whilst systems of measuring time have existed for millennia, how – and when – did humans cultivate an interest in investigating time and time perception? Furthermore, is time

perception and sense in the same way as vision and hearing are? A philosophical account of time perception will be required to answer that question.

1.3.2 Philosophical Accounts of Time Perception

Wearden (2016) argues that philosophers have had a fascination with the nature of time, including how humans perceive time, for thousands of years. The ancient Greek philosophers, informed by the dualistic approach of 'past and future', were the first to consider the notion of how humans perceive time and judge durations. The first philosophers to argue about the nature of time were Parmenides and Heraclitus (Hoy, 1994), which gave rise to two central aspects concerning time: the Parmenidean continuity aspect is concerned with how time extends from the past to the future, while the Heraclitan transience aspect is concerned with how things change in time (Roeckelein, 2008; Parmenides, 515 BC; cited in Nichols, 1891). As of 2023, these concepts of (a) how time extends from past to present and (b)., how things change in time, remain unreconciled though, this is more of a philosophical curiosity as opposed to a psychological problem.

Later, Plato considered time in his *Timaeus* and concluded that 'time doth not breathe on its fadeless bloom" which, in modern English, would mean that time is, paradoxically, constant (Plato, 385 BC; cited in Nichols, 1891) and changes (*constant* in that it exists as a single concept, and *changes* in that it changes from three stages – the past, present and future). Fraisse, (1963) eloquently summarised Plato's conclusion by opining that *'Man lives amidst change'.* There are many interpretations of Plato's conclusion; with Gunn (1930) interpreting Plato's *Timaeus* as Plato seeing time as '*mystic in character; remote and far beyond our mundane, everyday habitual experience*'. While not directly related to time perception *per se*, it illustrates how the fascination with time – and attempts to explain what it means to experience time; with even Greek mythology considering time. One such example of timing featuring in Greek mythology is where Cronus, who is equated with Saturn,

symbolises Father Time, and was conceived by Grecian scholas as a mythical being who both begat and devoured his own children. This mythological example demonstrates how Grecian philosophers ideated time as the constant becoming (e.g., where Chronos would begat his children) and transient (where Chronos would devour the children thereby ending time), according to Gunn, (1930); further illustrating the idea that time is constant and changes, in accordance with Plato's *Timaeus* as the dominant line of thought in ancient philosophy.

Of the Greek philosophers, Pythagoras first considered time perception (Roeckelein, 2008), followed by the likes of Aristotle and Plato. Aristotle was the first to ask, in a psychological sense, how the human perceives times (Nichols, 1891; Roeckelein, 2008; Whitrow, 1972). Therefore, Pythagoras considered the perception of time as arising from mind; whilst Aristotle asked how humans perceive time, which has similar parallels to the current interest in what it means to experience time. The ancient Grecians' interest in time would later inspire philosophers of the pre-scientific era to consider time perception. While Plato did not consider how humans perceive time (Nicolos, 1891), Aristotle did consider the perception of time (Benn, 1882) in which he said that *"Time is the number of motion"* which, psychologically, has been interpreted to mean that time *"is an immediate sense-perception of the number of motion´* (Aristotle, cited in Nicolos, 1891). In other words, according to Aristotle, time perception was a direct sense perception, in the same regard as vision and hearing are examples of sensation.

The consideration of time – and later time perception – by ancient philosophers would perpetuate to the medieval era – such as St. Augustine (Quinn, 1965), and beyond however, there was ambiguity as to what constituted time, which is an argument that, as of 2022, remains unsolved in the parlance of physics, philosophy, and psychology. However, the ancient philosophers seemingly consider time perception as a direct sense perception in the same vein as vision, hearing, olfactory or somatosensory senses. Whilst philosophy argues

that time is a sense like any other, it does not answer the question of what time is. Critically, for psychology to investigate the perception of time, a working definition of time perception is required.

"Without space and time nothing would be discriminated, or separate" declared Schulze and Maimon (1787; cited in Nichols, 1891). One could argue that there is no single definition of time but rather, that it is a multifaceted concept that includes a trichotomous state of the past, present, and future. Despite the difficulties in defining time, philosophers did not concede defeat in an attempt to explain the perception of time. It would take more than a millennium, from when the likes of Pythagoras, Plato, and Aristotle, questioned time, for a psychological analyse of time perception to take place. St. Augustine would partake in introspection and conclude that time is measured in the human's mind, which was a view shared by Guyau, Leibniz and Hartley (Roeckelein, 2008). St. Augustine would also ask – and fail to answer – how the mind could be an accurate chronometer for external events (Roeckelein, 2008) which is not too dissimilar to the questions asked by modern psychology (Wearden, 2016).

British philosophers, such as Hobbes, would reflect on the importance of time perception and theorise that there is not a single conception that is not associated with time (Nichols, 1881). The ontology of time has also been considered by philosophers, such as Locke and Guyau (1890, cited in Wearden, 2014) who would argue that time developed with experience, and ascertained that knowledge about time perception is best served by empiricism. Locke and Guyau envisaged that future experimentation of time perception would be based on generalisation regarding attention and time estimation (Nichols, 1891); which is the view taken by modern-day psychophysical studies. Locke classified time perception as partly reflection and sensation and summarised time perception with six propositions; the second of which is most valid to the *modus operandi* of the thesis:

By observing a distance in the parts of this succession, we get the idea of duration.

(Locke, cited in Nicolos, 1891).

Locke's propositions ascertain that time perception is not inbuilt but rather, something that is gained through experience (e.g., nurture). However, Locke's position contrasts sharply with that of the realists, Kant and Leibnitz; the latter of which argued the Universe comprises of an infinite number of individuals 'soul-units' or monads (Leibnitz, cited in Nicolas, 1891). The argument Leibnitz proposed was that thought, feeling, and perception were innate, and that time is thus innate due to the states that give rise to it (Wearden, 2016). However, this would imply time is absolute and objective and would further imply that humans perceive time on the basis of a biological organ. Consequently, the philosophers of the pre-scientific revolution would lay the foundations of the study of time perception in psychology.

There would be a dichotomous path for researchers to follow: those who adhered to Leibnitz, and pursued a biological explanation for time perception; and those who adhered to Locke, in that time perception is a sensation. Irrespective of the approach taken, the argument was settled that time perception was a disparate sense, similar to vision and hearing. The question, which led to the psychology of time perception, was weather time perception can be explained by a purely biological account, as posited by Leibnitz, or by a cognitive model, which is how sense and perception are generally modelled in modern-day cognitive psychology.

1.3.3 Psychology and Time Perception:

The study of time perception, in psychology, can be traced back to the foundations of experimental psychology and, to the work of Ernst Weber. In 1852, Weber published his treatise which asserted that the human's appreciation of distance and direction (i.e., length

and direction perception) was evidence of such appreciations being made based on a disparate sense (Weber, 1852; cited in Nicolos, 1891) which Weber termed the space-sense, which pertained to the German tradition of structuralism, emphasised by Leibnitz and Kant.

This led Johann Czermak, in 1862, to conclude that such a disparate sense, as posited by Weber, was necessary to perceive time. Czermak proposed the concept of an additional disparate sense, termed 'time-sense' (Nicolos, 1891). Czermak's modus operandi was to discover the physiological conditions of time perception (Debru, 2006), which diverged from the Lockian view of human timing (e.g., empiricism), and instead, adopted the Leibnitz school of thought that human timing is an inbuilt mechanism (e.g., structuralism. To answer the questions that Czermak proposed, the following had to be determined: (i): The shortest interval perceivable in each of the separate sense. (ii) How the same interval is interpreted by the different senses. (iii) How like rates of motion are interpreted by various regions of the skin, determined by Weber to be of different spatial sensibility. (iv) The least change in rate of motion perceivable for various dermal regions. (v) The relation between rates of motion and changes in the angle of convergence of the eyes. (vi) To investigate the formula: $V = r/t$ for points of the retina or skin having different spatial sensibility (Debru, 2006; Czermak, cited in Nicolos, 1891), which can aid psychological understanding of time perception Czermak's questions were symbiotic of his training as a physician however, unable to carry out such experimentation, this led to the first experimental evidence obtained for time perception by Vierordt and his student, Horing, in 1864. In a series of experiments, in which subjects were given 8 beats, by means of a Maelzel metronome followed by a weight which was moved. The subject, without having seen the pendulum, was asked to judge whether the second set of beats differed from the first set. Vierordt (1864) used his student, Horing, as a subject.

This marked the first experiment that directly addressed the points risen by Czermak (Wearden, 2016; Nicolas, 1891). One of the most striking of findings was that of *Vierordt's Law*, which is the finding that subjects overestimated shorter intervals (e.g., the intervals, in the .306 – 365s range); and underestimate longer intervals (e.g., those durations in the range of .454 – 1.428s), which remains a consistent finding in the literature (Wearden, 2016; Jazayeri & Shadlen, 2010). A further study by Vierordt (1868) was also conducted in which Vierordt sought to determine time perception in general by means of a temporal estimation study (Vierordt, 1868) which added – and replicated – the 1864 study and made other significant contributions to the timing literature (Wearden, 2016; Nicolos, 1891).

One of the founding fathers of psychology, Wilhelm Wundt, also conducted studies on time perception (Wearden, 2016) and asserted that Vierordt's studies were 'unwarrantably complicated' (Wundt, 1882; cited in Nicolos, 1891) and proposed that Vierordt's equipment was not sensitive enough to measure time perception, in the context of temporal estimation, due to the complication of muscular movement. Wundt's subsequent experiments failed to replicate Vierordt's findings regarding short and long intervals, which Vierordt (1882) argued was evidence of his results not being vitiated by Wundt. Over the next decade, there were many similar studies conducted in time perception (see Nicolos, 1891 for a review of these classic experiments). Arguably, these studies illustrate how time perception was a concern for psychologists; and demonstrates that study of time perception is rooted in experimental psychology, which, at its foundations expended much effort in studying sensation (e.g., vision, auditory and sense) using psychophysical experiments (Wearden, 2016) relating objective stimulus measures, such as the intensity to light, and measuring the sensations evoked by the intensity.

Time perception was considered a sensation much like vision or hearing (Wearden, 2016). Indeed, the ideation that time perception is a sensation (in keeping with the philosophy of Leibnitz), within psychology, can be traced back to William James (Myers, 1971; James, 1886). James argued that durations the human contends with on a day-to-day basis (i.e., second, hours, days etcetera) must be *symbolically conceived* (James, 1886) and constructed by mental addition to perceive durations, such as 4 hours, which is the sum of 4 individual hours and so on. Due to the purge of structuralism from psychology by Watson (Ornstein, 1969), the study of time perception progressed slowly from around 1900 until the 1930s, with the work of Hoagland (1933), and later Creelman (1962) and Treisman (1963) reigniting the interest in the perception of time. Often considered the 'founding fathers' (Wearden, 2016) of the mechanising of time perception; as prior to their work, few models existed for time perception; with one of the most significant stumbling blocks in studying time perception being based on the fact there is no physical 'time sensing' organ (Wearden, 2016) which led to time perception being purged from psychology, due to its materialistic nature (Ornstein, 1969). The formulation of the chemical – and later – cognitive models of timing, established a theoretical framework to study time perception, which persists to the present day (Wearden, 2016), sought to explain the mechanistic nature of time perception. The chemical clock model was based on the idea that time perception was driven by chemical interactions in the brain (Ornstein, 1969).

The chemical clock model was envisaged first by Pieron (1927, cited in Wearden, 2016) who used the method of diathermy (the passing of a high-voltage electrical current through the body) to induce bodily heating. Such experiments were also conducted by Francois (1927) who investigated whether the perception of time was contingent on temperature. Francois found that a heightened body temperature correlated to shorter intervals. The chemical clock hypothesis was furthered by Hoagland (1933), who

investigated temperature on time perception more thoroughly than Pieron or Francois did though, it was not without its critics, namely, Orenstein (1969), who argued the notion of the 'biological' or 'chemical' clock is illogical. The reasoning being that if there was an internal organ (or mechanism) which was responsible for the perception of time, would imply there is an objective time that is universally defined however, different cultures have different methods for measuring time (Ornstein, 1969) and the fact that Einstein (1907) demonstrated that time, as a construct, is relative to the frame of reference it is observed in, as opposed to absolute, rose further questions for the theoretical case for a biological clock.

To circumvent these theoretical issues associated with the biological clock, the internal clock models dawned in the 1960s on the basis of work by Creelman (1962) and later Treisman (1963). These models approached time perception as a sensation, as envisaged by James (1890), as opposed to a physical biological entity. These models (which seemingly have their origins in Fechner's (1860) ideas of human timing amounting to oscillations) assume that time perception is an information-processing cognitive model, in which the subject perceives a duration (information), then acts upon it (processing). The earliest cognitive models of human timing would bring the psychology of time perception to the most popular cognitive account of human timing: the mathematical model of *Scalar Expectancy Theory* (SET) which was devised by Gibbon (1977|). This model incorporates the Creelman and Treisman ideas of a pacemaker-accumulator mechanism. SET remains the most dominant model of human timing to the present day.

1.3.4. Summary

In summary, the history of time perception is rooted in the very first instances of evolution; from *Australopithecus'* crafting of tools for a pressing present, and an imagined future; to the Sumerians' developing of a writing system to record past events and the Chaldeans and ancient Egyptians basing timing on physical cosmological events. This ancient fascination of timing was given consideration by philosophers as varied as Plato, to more contemporary philosophers, such as Locke. The psychological treatment of human timing begun at the foundations of experimental psychology; by the work of Fechner, Weber, Wundt, Vierordt, and James. This placed time perception within the psychophysical tradition of psychology and treated the perception of time as a sense. As psychology moved away from materialism, which was led by Watson, this meant that the study of timing fell out of favour from 1900 to around 1930. However, this led psychologists to model time perception as a biological entity, in which researchers, such as Francois, Hoagland and Pierrot attempted to explain time perception in terms of biochemical reactions in the brain. Ultimately, these early studies illustrated the inconsistencies of biological models of time perception and led to the formulation of cognitive models of time perception, by Creelman (1962) and Treisman (1963); which viewed timing not as a biological process, but rather, as a sensation. This culminated in the establishment of the most dominant model of time perception: SET, which remains the most dominant model of human timing which researchers use.

1.4 Models of Human Timing

Whilst the chemical clock models are seldom considered in the present-day literature, they led to the development of the internal clock models, of which gave rise to SET, which is the dominant model of human timing (Wearden, 2016). Therefore, to understand how the cognitive models of time perception arose, it is important to review these biological models of time perception. While there are nuances which set the biological and internal clock

models apart, they both share a commonality in that they all have a ‘time base’ which Ornstein (1969) states is a repetitive, culminative pulse-dispensing mechanism. In terms of these ‘pulses’ they were termed as a ‘time quantum’ (Ornstein, 1969) or, in the modern literature, ‘ticks’ or ‘pulses’ (Wearden, 2016).

The idea of a pulse-dispensing mechanism has its origins in the publication of Gustav Fechner’s *Elements of Psychophysics* in 1860. In one study, Fechner noticed that two sensations, which follow each other at a rapid rate, converge (Fechner, Adler, Boring & Howes, 1966), such as viewing two light strobes separated by 500ms. This led Fechner to ask what length durations must be to be perceived separately (e.g., 500ms vs. 1000ms) and conducted experiments to investigate this research question. Fechner contended that time perception depends on ‘psychophysical oscillations’ which emits pulses at a given rate. The esteemed psychologist, Wilhelm Wundt, continued the ideas suggested by Fechner by proposing the mechanistic nature of time perception, positing that:

“Assume that … similar pendulum strokes follow each other at regular intervals in a consciousness otherwise void. When the first one is over, an image of it remains in the fancy until the second succeeds” (Wundt, 1887, cited in Nicolos, 1891, pp 483)

The consensus was, at least in psychophysical parlance, that time perception was modelled as some sort of time sensing internal clock, whether it be an oscillator (Fechner) or a pendulum (Wundt), though, whether this internal clock was biological or cognitive in nature was not discussed but what was hypothesised was that this internal clock emitted pulses. This idea that human timing is an oscillator or pendulum can be – and was – extended to the pulse-dispensing mechanism (e.g., pacemaker) that is found in all models of timing (Wearden, 2016).

The process by which a human might perceive timing was given serious consideration by psychologists; with one such process described by Theodor Lipps, who claimed that:

"*Sensations arise, occupy consciousness, fade into images and vanish; according as two of them, a and b, go through this process simultaneously, or as one precedes or follows the other, the phases of their fading will agree or differ; and the difference will be proportional to the time-difference between their serval moments of beginning*

(Lipps, cited in Nicolos, 1891, pp. 484)

This demonstrates how the idea of a pulse-dispensing mechanism arose in the perception of time, and such a mechanism dominated both biological and internal clock models, which persists to the present day. However, one subtle difference is that the biological models assume that 'objective time' exists that the human perceives, and the biological clock processes. The internal clock models make no such suggestion. Most of the models work on the premise of how accurately the human perceives durations, which is in keeping with the very first experiments in time perception. The result of these seminal suggestion of an internal clock was met with an abundance of models of human timing, including those by Hoagland (1933), Creelman (1962), Treisman (1963), Gibbon (1977) and Zakay & Block (1997). We shall discuss the biological clock models, followed by the internal clock models, including SET.

1.4.1 Biological Clock:

The ideation of time perception being contingent upon a biological clock residing in the human arises from the sensory nature of time perception (Ornstein, 1969), as established by the first investigators of time perception; as well as from the Leibnitz position that human timing is naturalistic (Leibnitz, cited in Nicolos, 1891). The logic is as follows: if time perception is a disparate sense (Czermak, cited in Debru, 2006), then time perception should

have a sense organ, much like the other senses do (e.g., the sensation of vision is made possible by the sense organ, the eye). The biological approach to time perception assumes that time is a real (i.e., independent of the human mind) objective concept, and the biological clock is the hypothesised internal organ that allows humans to perceive objective time.

Many philosophical arguments and experiments, in the field of time perception, were opined and carried out to test the biological clock hypothesis. In terms of how any sensation should be studied objectively, Titchner (1905) stressed the introspection approach to sensations (in keeping with Weber, 1860); while James (1890) attempted to relate human timing to the decay of 'brain traces' or, in modern parlance, memory (Ornstein, 1969). Conversely, attempting to escape the mentalist approach of time perception, in accordance with Nicolas' (1891) leitmotif and Watson's (1913) behaviourism, experimental psychologists attempted to determine whether the perception of time adhered to some sort of physical law, such as Weber's Law (Woodrow, 1951, cited in Stevens), which relates to the perceived change of a stimulus that is a ratio of the original stimulus, and finally, whether there was an organ that could account for time perception. The most obvious way to investigate time perception was to attempt to manipulate the alleged 'timing' organ.

Many experiments were carried out to test this hypothesis, but little was done to reconcile such experiments with theoretical work (Ornstein, 1969) apart from attempts by Lipps (cited in Nicolos, 1891) to explain how the human perceived time. The idea of a biological clock allowing humans to perceive time was first proposed by Lereboulett and Pierrot (1927) but extended by Francois, (1927). Francois' main findings were that increases in body temperature shortened durations produced though, lengthened subjects' duration estimates (Wearden, 2019). For example, the subject would produce a 1000ms duration as 500ms but perceive the 1000ms duration as 1500ms. This led Francois to believe there was a biological clock that underlined human time perception (Grondin, 2008), which dominated

the early theoretical development of a model for human timing. The logic posited by Francois and others was that chemical processes were largely responsible for these inconsistent findings in timing studies (Wearden, 2016); given rise to some controversial experiments within experimental psychology (Wearden, 2016; Grondin, 2008).

The concept of a biological basis for time perception has led researchers to conduct numerous experiments – sometimes controversial – that have focused on the manipulation of the so-called 'indifference interval'. The indifference interval is where the human is said to be most accurate at an interval and arises from the consistent interest in accuracy of human timing (Ornstein, 1969). This process reduces human timing to the apprehension of external processes and leads researchers to believe timing to be a sensory process amenable to biology. Earlier researchers believed there to be a 'real objective time' and that humans can perceive it through means of a chemical clock.

However, one of the first problems met with a so-called 'biological clock' is the indifference interval itself, as many researchers (e.g., Vierordt and Horing, 1864; Kollert, 1885; Treisman, 1963; Fraisse, 1963) have found different 'indifference intervals leading to the suspicion that such an interval might not exist at all, hence some argue, the inconsistent experiments demonstrated the indifference intervals do not exist. Many researchers appeared to believe that because this indifference interval was sometimes correct, this reflected 'true time' however, there is an issue in what constitutes 'true time' as to whether it is an objective property of nature, or a subjective concept (Einstein, 1907; Ornstein, 1969). Despite these theoretical difficulties, the investigation for a biological clock continued, with authors (e.g., Ornstein, 1969) considering the 'time quantum' units (or ticks emitted by this 'internal clock') being objective measures of an objective time. Early researchers attempted to correlate the ticks to breathing rate (Munsterberg e tal., 1894), brain cell metabolism (Hoagland, 1934), EEG alpha wave (Murphree, 1954), cerebral cortex functioning

(Braitenberg & Onesto, 1960), cellular metabolism (Bunning, 1960) and heart rate (Ochberg, Pollack & Meyer, 1964).

The research attempting to understand the biological origins of human timing is significant – and sometimes contradictory, however, there are biological reasons to believe humans possess a biological clock. The idea that living organisms possess a biological clock is found in the botany literature whereby Garner and Allard (1920) found evidence of photoperiodism (e.g., an organism that responses to day length) in plants, which led researchers to investigate whether circadian or diurnal cycles, or heart rate etcetera gives evidence to the existence of this so-called biological clock. In keeping with the tradition of experimental psychology, researchers have attempted to manipulate this biological clock, where some of the first studies have investigated whether temperature modifies this biological clock. Studies by Pieron (1927) and Francois (1927) attempted to modify the biological clock by passing a high-frequency electric current through the body to induce bodily heating (diathermy). The main finding of such studies was that it appeared that the perception of time (and counting) was dependent on a chemical reaction. The chemistry literature states that all chemical reactions proceed more quickly when heated therefore if body temperature is risen the human's chemical clock should emit pulses 'quicker'. This led to Hoagland (1935) to ask his wife, who had influenza at the time, to count (at a rate of one second) to sixty seconds, followed by the oral taking of Mrs. Hoagland's temperature. Much in keeping with Francois' findings, Hoagland found that counting appeared to be contingent on body temperature in that the higher one's body temperature, the quicker they count. However, as impressive as Hoagland's theory was, further findings on body temperature and the perception of time are mixed; with many studies being inconclusive (e.g., Kleber, Ronald, William, Lhamon & Goldstone, 1963; Bell & Provins, 1963; Fox, Bradbury, Hampton & Legg, 1967) however, some support for the hypothesis of body temperature and human

timing can be found from Hancock (1993) and Baddeley (1966). Indeed, Wearden & Penton-Voak (1995) reviewed the literature on time perception and body temperature and found that the rate of subjective time increased when body temperature increased. This would imply that perhaps there is a 'biological clock' mediated by temperature though, as Ornstein (1969) posits, if this were the case, then other nuances associated with time perception (e.g., duration judgements) would also have to be explained by a modification of body temperature.

It seems, at best, tenuous, to suggest that those who judge shorter durations as longer do so because they have a higher body temperature. Moreover, as research shows that individual differences contribute to differences in the perception of time (see chapter 2), one would assume those with schizophrenia, for example, do not have a higher body temperature than those who do not have schizophrenia. A further issue persisting is that neither Hoagland nor Francois were concerned with what psychological time is, let alone providing a mechanistic model for explaining it, but rather they were interested in whether human timing was driven by a chemical reaction (Wearden, 2016). However, the chemical hypothesis lost pace but not the biological hypothesis for timing.

Fischer (1967) also attempted to model human timing as a biological clock by giving subjects psilocybin (LSD) before and after timing tasks, which Fischer determined measured the perception of time, which was kept by a 'chronometer'. Fischer found that subjects' handwriting size, Weber fraction for taste, finger-tapping rate, and the experience of duration all increased; finally, there was a dramatic increase in optical nystagmus (Fischer, 1967). Studies by Rammsayer & Vogel (1992); Rammsayer (1993) and Meck (2004) imply internal timing is affected by the administration of drugs however, Ornstein (1969) points out shortcomings, in the Fischer studies, which are applicable to contemporary studies by Rammsayer and Meck, who attempt to find a biological clock. Firstly, all these studies have defined their own 'chronometers' without giving a criterion for what defines a 'chronometer'.

Whilst sound reasoning is apparent, thinking something is a chronometer does not make it so (Ornstein, 1969). For example, many have associated the basal ganglia with the perception of time, raising questions as to what a basal ganglia chronometer is., and why should there be a basal ganglia chronometer. Ornstein (1969) states: "why not hair growth as a metric for a chronometer?" Secondly, do biological accounts of time perception explain how humans experience time. While these accounts are useful in terms of periodic rhythms, they say nothing about how one experiences the perception of time. This is discussed further by Michon (1967) who showed that alternations in rhythm speed are related only to information-processing speed as opposed to the actual experience of time.

Finally, whilst the idea of timing as a biological sense might seem useful, such as when comparing one's experience of time to what is shown on the clock, as a scientific metaphor, it is of little value to the human experience of time perception, as it leads researchers to search for the illusive 'biological clock' (Ornstein, 1969). It would be more pertinent to suggest that biological interventions (e.g., narcotics) effect cognitive processes that drive time perception. However, a cognitive model of time perception has to exist to apply such reasoning.

Michon proposes, as does Ornstein, that it would be more parsimonious to consider a cognitive model of the perception of time, such as the internal models of time perception, as they make no reference to a biological basis of the perception of time. As Wearden (2016) states, these 'biological clocks' whilst stating little about the experience of the perception of time, did lead to clearly specified internal clock models, which were developed in the 1960s and 1970s, respectively, hence the necessity of reviewing the cognitive models of time perception.

The main points that arise from this discussion of the chemical clock is that it is inadequate for explaining the cognitive nuances of the perception of time (Ornstein, 1969). Furthermore, since it implies there is an objective 'time' , it calls into account the entire ideation of the biological clock. Researchers thus investigated cognitive models of time perception that do not entail the biological and philosophical ramifications of the biological clock, as espoused by Nicolos' leitmotif and Watson's behaviourism.

1.4.2 The cognitive Clock Models: Creelman and Treisman

The idea of a cognitive model of time perception is a contemporary one. Guyau (1891) attempted to relate time experience to human information-processing however, due to the rise of both objectivity and behaviourism, this idea was never fully developed (Nicolas, 1891; Ornstein, 1969) in the earliest days of time perception research. However, due to the inconsistencies of biological accounts, it was clear that a cognitive model was required. The seminal research, which gave rise to the birth of the Internal Clock Model of time perception, can be found in the thesis of Creelman (1962), who proposed the first model of an internal clock (Wearden, 2016) which did not rely on chemical or biological process.

Creelman was initially interested in how humans discriminate durations of auditory nature (Creelman, 1962) which was the purpose of his thesis, on behalf of the United States Navy (Creelman, 1962). Of significance is how he explained his resulting data, in terms of his proposing of a mathematical model termed the 'counting model. The model included a simple accumulator to store so-called 'pulses' of timing. Importantly, the counter mechanisms made no commitment to a physical model (Creelman, 1962). Creelman teases the reader in suggesting that the 'counter mechanism' could be neurological in nature, however, Creelman points out that the mathematical formulation of model does not commit to the existence of a "physical model" (Creelman, 1962) or the physical existence of this

counter model, therefore committing to ambiguity surrounding neurological substrates of this counter mechanism. Creelman's model proposes that pulses are emitted by this counter mechanism, which keeps a tally of total pulses. The collated pulses are subsequently compared to some standard that the subject has been trained on. Arguably, this model is a basic pacemaker-counter-decision model, and memory components (Wearden, 2016) which laid the foundations for the internal clock models.

Creelman's interpretation of his data would lay the foundations for the internal clock models and stand in direct contrast to the chemical clock models along with the biological models of Nicolos (1891) and Watson (1913) by proposing a cognitive model that makes no assumption of neurological foundations of time perception. However, it would be Treisman (1963) who would fully develop a cognitive model of time perception which Treisman (1963) termed the Information-Processing Model of Timing (IPMT).

Treisman (1963) developed a theoretical model for time perception, based on a pacemaker, counter store, and comparator (Wearden, 2016) which utilised the ideas of Creelman. As Wearden, (2013) states: *"(…) Creelman who proposed the basic clock, but omitted much of the 'clockwork' needed to instantiate the clock into a viable psychological model'* .Treisman (2013) also committed to the principle of parsimony in developing his model and noted that if such a model were to be developed, it would need to account for all the nuances associated with the numerous psychophysical procedures that have been employed in investigating the time experience (e.g., Hollingworth, 1913; Woodrow, 1951) in the simplest possible way (Treisman, 2013). In the seminal paper, *Temporal Discrimination and the Indifference Interval: Implications for a Model of the "Internal Clock",* Treisman (1963) extrapolated further on Creelman's thesis by running a total of seven experiments which culminated in the development of the information-processing model of time

perception. These experiments used the production and estimation timing tasks, which is what Treisman's IPM model sought to model. The model is shown in figure 1.1.

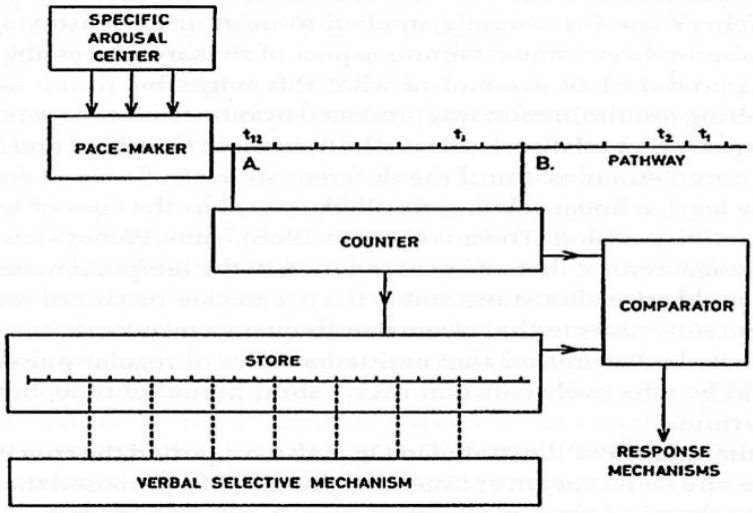

Figure 1.1: The Information-Processing model postulated by Treisman (1963). The model consists of an arousal-driven pacemaker, a pathway, feeding into a counter, which feeds into both a store and comparator. The result of this model is that the subject will decide (e.g., a response mechanism) on the basis of durations compared from the comparator and the store (From Treisman, 1963)

IPMT begins with a pacemaker, which is driven by a specific arousal centre (Wearden, 2016; Treisman, 2013; Treisman, 1963). Treisman's logic for the existence of this pacemaker of time arises from two sources: the first from the existence of neural cyclic pacemakers (e.g., Gu, Jia and Chen, 2013) found in nature, such as the circadian (Roenneberg et al., 2007); and the second source comes from private correspondence between Treisman and Doctor Gabriel Horn. Horn (Horn, cited in Treisman, 2013) informed Treisman that he had observed a 'pacemaker cell' in a cat, which produced a single spike at intervals of approximately 10 seconds however, when the spike failed to occur, the resulting pause lasted a multiple of 10 seconds. Horn had observed one such cell regularly activating at a mean interval of 10.1 s, however, on one occasion, it produced an interval of 20.3s (Treisman,

2013). This finding, however, was never followed up but Treisman contended that it was difficult to determine what its significance was other than in relation to timing. Treisman also used the reasoning that Weber's Law is a commonality shared by most sensory systems (Stevens, 2022). Therefore, if timing is a sense (Nicolos, 1890; James, 1891; Wearden, 2016) and all senses share an adherence to Weber's Law, then a pacemaker-driven clock should also adhere to this principle. As a result of the reasoning of both the circadian clocks, Creelman (1962) and the adherence to Weber's Law, Treisman posited a pacemaker which emitted a series of regular pulses at a constant rate however, they might vary from time to time. By devising the arousal-driven pacemaker, IPTM required a mechanism for extracting the temporal information arising from the operation of the arousal-driven pacemaker. The pacemaker emits pulses, which transverse an output path (See figure 1.1); where a counter associated with the path monitors the number of pulses passing, what Treisman (2013; 1963), calls a 'prescribed point' during each presentation of a given interval. Treisman presumed that the pacemaker functions continuously; however, if an interval is presented, the pacemaker is delimited by an initial stimulus, s_1 and a final stimulus, s_2, then these stimuli force the Counter to begin operation at s_1 and cease operation at s_2. The Count, N, recorded between these two events (e.g., s_1 and s_2) provides a metric of the time elapsed (Treisman, 1963; 2013). As can be seen in Figure 1.1, there are other components, including the store and comparator. The purpose of the store, which can be thought of as a memory storage, is to store temporally important information (Wearden, 2016). Treisman (2013); whilst the purpose of the Comparator is to compare information from the store and counter. Treisman notes that one of the strengths of the model is its adaptability with respect to modelling data extracted from different timing tasks.

1.4.3 Theoretical Issues

Treisman (2013) determined there were problems with the model, which are covered in Treisman (1963, 2013). Treisman noted that when subjects make a response, it takes time to make that response, therefore, when S_3 initiates the comparing of a duration to that held in storage. Treisman reasoned that if the comparator waits till it has received a number of ticks that matches $N_{crit} = N_s$, there will be a delay of approximately 100ms while the subject executes a response (Treisman, 2013; 1963), thus, the internal clock must have a way of correcting for this additional response by 'learning' the mean duration of durations and adjusting the model to correct for this.

To account for this error, the counter (Figure 1.1) is shown as having two reading-out points, A and B. Treisman reasoned that if the motor delay the system wants to compensate for is approximately 100ms, A and B are chosen so that the time for a pulse to travel from A to B is 100ms. At *T*, the counter does not start at 1 but rather, at the moment presented between A and B, in Treisman's example, 100ms. Thus, when $T \geq N_{crit}$ the time elapsed will be 100ms shorter allowing the system to correct for the mean response delay. Thus, the first of the problems Treisman identified was rectified by building a correction parameter into the pacemaker.

The second problem that Treisman identified is illustrated in Figure 1.1 and associated with the finding that the occurrence of lengthening during the course of the condition (i.e., subjects think the experiment lasted longer than it did). Treisman initially attributed this this to the gradual slowing of the pacemaker (Treisman, 2013; 1963; Wearden, 2016) as the subjects' arousal levels decreased due to boredom, which are symbiotic of psychophysical experiments (Stevens, 1951). When the pacemaker runs more slowly, it will take longer to count N ticks. This is an effect found in some subjects with numerous psychopathologies (e.g., Carroll et al., 2008; Reed & Randell, 2014). In spite of the argument

making its way in some of the literature (i.e., Wearden, 2008), Treisman points out the tenuous nature of such an argument. The argument being is that if the onset of a estimation task, the pacemaker is running at 100Hz, and the subject is presented with a standard interval, K = 1s. The counter will record a measure of U = 100 ticks during the standard interval, where this is stored in the store. When the subject is asked to reproduce this, this value will be retrieved by the comparator, which serves as N_r = 100 during the subsequent estimation of said interval. Ignoring the motor delay, above, and assuming the pacemaker rate is effectively constant over short intervals. To reproduce K, the subject will compare N with the preceding U. As soon as N_r is greater than, or equal to 100, the estimation ends, and the two counts are approximately 100 ticks each.

Treisman asks readers to subsequently suppose the pacemaker is 'dawdling' along at 80HZs when the same standard above is presented but ticks = 80; which will be stored in the counter and when the duration to be judged is presented, the comparator will compare the incrementing count with the criterion (i.e., 80 ticks) and both counts of 80 ticks each will mate one second to one second. However, this is not what happens. Treisman equates the criterion to the value on the same trial should negate the effects in the variation of pacemaker rate thus, a more sophisticated mechanism is responsible for setting the critical value. Treisman suggested that if the same standard is presented repeatedly over a series of trials, the comparator might be exploiting statistical stability of the accumulate ticks in the memory store. Thus, Treisman (2013) states, if pacemaker rate falls from 100 to 80 Hz during a given sessions, ticks for T = 1s stored during that fall may range from 100 to 80 ticks, which gives a mean critical value of 90, which means the subject would terminate T at 90 ticks. This would imply the pacemaker is slowing down, and manifests as a lengthening effect.

These considerations helped formulate the nature of the store in Figure 1.1 On that basis, the store needs to retain quantitative information and thus, Treisman represented it as

being built around an axis or scale on which such quantities can be recorded. Treisman assumed that if a given standard, $|T_s| = \tau$ is repeatedly presented to the subject, the ticks, N_s obtained from the presentations are distributed over a corresponding 'τ-region' of the store axis, which means that a sample of the discriminable dispersion for $|T_s| = \tau$ is available in memory. On subsequent trials, a function of that distribution, such as the mean, can be retrieved from the store to give a T_{crit} for that given trial. The effect would be that the value of T_{crit} will lag behind changes in pacemaker tick rate, whereas T_r is directly determined by the rate of the pacemaker at the time, which provides a basis for lengthening.

Treisman (2013) makes the case that the scale provides a basis for modelling the method of production (MP) task; with the production of T_p on each trial will proceed as T_r but in the absence of recent pacemaker ticks, there will need to be a mechanism for defining the criterion, which Treisman (2013) claims brings a role for language into the mode. As experience of named durations will have create schemas between given points on the scale, which represent particular durations and corresponding verbal labels, termed a 'language store' in the store. When the experiment asks the subject to produce an interval, such as 1s, the language store processes either a verbal or written label '1s' and derives from it an instruction from the store to the comparator to provide T_{crit}. As this criterion is fixed over time, it is largely unaffected by pacemaker rate, which will cause the MP to show lengthening the pacemaker ticks slow, and to do so for MR tasks, which is what Treisman (1963) found.

The problems that Treisman (2013) identified in his model were accounted for by adding parameters to the model though, as Wearden, (2016) comments, such parameters would be difficult to justify, psychologically. Despite the complexities and potential theoretical issues of Treisman's information-processing model, one ideation that persisted in subsequent – and current – research was that the pacemaker of the internal clock is sensitive to arousal levels; with the basic premise that the pacemaker emits more ticks when arousal

was high and less ticks when arousal was low (Wearden, 2016; Treisman, 2013; Treisman, 1963). However, the Treisman model, despite its shortcomings, gave way to the *Scalar Expectancy Model* (SET) that remains dominant in the literature.

1.4.4 Scalar Expectancy Theory (SET)

These models would pave the way for arguably, the most parsimonious of all timing models: Scalar Expectancy Theory (SET). SET was posited by Gibbon, Church & Meck (1984) in *Scalar Timing in Memory* and was developed from earlier work by Gibbon (1977) SET has its origins in animal timing and behaviourism (Wearden, 2016) however, its development as a theoretical account for psychological time was originally developed to account for data for the classic psychophysical task, temporal generalisation (Wearden, 2016; Gibbon, Church & Meck, 1984).

The basic tenets of SET are similar to those of Treisman's (1963) model, in which there is a pacemaker and a comparator, of which the latter is termed the accumulator in SET. SET deviates from Treisman's model in that it introduces a memory component (Wearden, 2016) comprised of a working-memory model, a reference memory model (Gibbon, 1977) and a switch, the latter being assumed to be served by an attentional mechanism. The model was initially developed to account for animal timing (Gibbon, Church & Meck, 1984; Gibbon, Church, Fairhurst & Kacelink, 1988) but later, was adopted to human timing (Wearden & McShane, 1988; Church & Gibbon, 1990; Allan & Gibbon, 1991; Wearden, 1991; Wearden & Lejeune, 2008;). As Wearden (2016) notes, SET derives its name from the so-called 'scalar properties' of time; where 'scalar' is used in mathematical sense (e.g., a dimensionless unit without a direction).

One way to represent this idea is to suppose the subject is measuring different timing intervals, t, 2t, 3t, etcetera. SET would presume each of these intervals is relatively similar,

but expressed on different scales (Wearden, 2016). To give an example, 200ms and 300ms are relatively the same, but are on different scales. Scalar timing, however, requires that time representations fulfil two criteria: the first is mean accuracy, which is the requirement for a subject's estimate of 'real time', t_s, is equal to t most of the time. In the context of a estimation task, when the subject is tasked with reproducing 400ms, the average time the subject produces should track onto 400ms, with deviations either side of the mean (e.g., Standard Deviations) expected. A theoretical flaw with this argument is where Ornstein (1969) discusses how it is irrational to contemplate 'real time' though, to address this theoretical difficultly, 'real time' in the context of the subject is what the duration represents to them.

The second requirement of SET is the so-called scalar property of variance (Gibbon, 1977; Wearden, 2016). In essence, to show conformity to the scalar property of variance is that the standard deviation of response measures should be a linear function of their mean (Wearden & McShane, 1988; Wearden, 2016). This can also be represented by the coefficient of variation, where smaller values mean that response measures cluster closer to the mean, and thus, represent greater sensitivity to timing, which is similar to the Weber Fraction (Wearden, 2016). Once again, a smaller Weber Fraction implies greater timing sensitivity and thus, subject responses grouping around the mean. In terms of what this means, psychologically, it means that subjects should show the same sensitivity irrespective of intervals used in that the Weber Fraction for differentiating between 200ms and 400ms should be the same for 1200ms and 1400ms. One of the central attractions towards SET is the apparent ease at which its subcomponents can be isolated and thus manipulated (Wearden, 2016).

1.4.4.1 The SET pacemaker

The pacemaker in SET is similar to that postulated by Treisman (1963), from the earlier work of Creelman (1962) however, Gibbon, Church & Meck (1984) assumed there are two potential sources of variation in pacemaker operation. In both sources of variation (discussed below) the pacemaker is presumed to emit pulses at *n* mean rate, where *n* is presumed to be determined either by some sort of arousal centre (Treisman, 1963; Gibbon, 1977; Church & Gibbon, 1990; Block & Zakay, 1990; Wearden, 2016). Mathematically, the pacemaker is defined by Gibbon et al. (1984) as $\lambda = \frac{1}{E(x)}$ where *E(x)* is a function of pacemaker pulses and is illustrated in figure 1.2. The pacemaker emits pulses with an interpulse intervals of τ and a mean rate of $\Lambda = \lambda$.

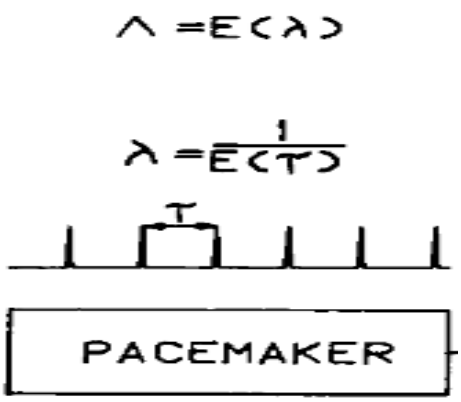

Figure 1.2: The Pacemaker (Gibbon et al., 1984) emits pulses with interpulse intervals, τ with a mean rate of $\Lambda = \lambda$. The pulses are evenly spaced to represent no variance in the model.

The pacemaker has been the subject of discussion of much investigation in cognitive and experimental psychology (Treisman, 1963; Gibbon et al., 1984; Block & Zakay, 1990; Gibbon, 1990; Meck, 1994) with research focusing on what mediates the pulses that the pacemaker emits (Wearden, 2016). Behavioural evidence for the existence of a cognitive process which resembles the pacemaker has been sought by manipulating the pulse rate**,** τ.

Evidence suggests, at a behavioural level at least, the pacemaker rate can be manipulated with a clicker, (Treisman, 1990; Penton-Voak, Edwards, Percival & Wearden, 1996; Wearden, Williams & Jones, 2014), flicker and vibratactile timing (Jones & Ogden, 2015). In most of these cases, the pacemaker is said to emit a greater number of ticks in the presence of a clicker, which has been interpreted as 'speeding up' the length of durations (Wearden, 2016). This manifest as subjects underestimating durations, as there are a greater number of pulses within an interval, when the clicker is present. The inverse of this would be the pacemaker 'slowing down' when arousal levels are reduced (Droit-Valet, Fayolle & Gill, 2011). To give an example, a 4s interval might feel like an 8s interval due to there being more pulses within the interval. With respect to why a clicker train should speed-up an alleged pacemaker, many authors (e.g., Wearden, Win and Philpott, 1999; Wearden, 2016) suggest the presence of a clicker arouses the pacemaker, which excites it (Wearden, 2016).

The evidence for the pacemaker component is evidenced by psychopharmacological approaches (Meck, 1996). In one such study, Rammsayer (1999) demonstrated that administering narcotics manipulated how subjects perceived intervals. This gives evidence to the dopaminergic hypothesis for the pacemaker, which suggests that the dopaminergic pathways of the basal ganglia play a prominent role in time perception and the pacemaker (Penney & Vaitilingam, 2008 cited in Grondin, 2008; Coull et al., 2004; Ivry & Spencer, 2004; Malapani & Fairhurst, 2002; Gibbon et al., 1997). Therefore, the pacemaker operation can be approached dichotomously in that (1), arousal levels manipulate pacemaker speed and (2), dopaminergic levels in the basal ganglia can also manipulate its speed (Grondin, 2008, 2010); which is in keeping with the information-processing nature of SET (Grondin, 2010). However, since the pacemaker is thought to be driven by arousal levels (Treisman, 1963), the

dopamine hypothesis should be thought of as affecting the *cognitive* processes (e.g., arousal) of the pacemaker, as opposed to time perception itself.

1.4.4.2 The Switch

The component that follows the pacemaker is what Gibbon et al., (1984) termed the switch. The switch is an addition to Treisman's (1963) Information-Processing model. The switch gates these pulses into an accumulator but is assumed to do so with some latency to close (t_1), once the timing signal goes on (i.e., when the subject is presented with a duration) and open (t_2), after the signal goes off (i.e., when the duration the subject is asked to judge, terminates) These latencies define the effective pacemaker rate, $\lambda = (T - T_0)$, where T corresponds to the length of the duration (e.g., the amount of pacemaker pulses indicative of duration), and T_0 is equal to $t_1 - t_2$ (e.g., the difference between switch closing and opening) that the pulses are accumulated at (Gibbon, 1990). When both t_1 and $t_2 = 0$, the switch is open, and the duration can pass through without variance however, when $t_1 - t_2$ is greater than 0, variance can affect the duration which, depending on the value of the difference, T_0 can increase the duration.

The switch closes when there Is a temporally significant event occurring (Gibbon, 1977; Lustig & Meck, 2002) and allows the pulses emitted from the pacemaker to flow linearly to the accumulator. When these temporally significant events cease, the switch opens, which stops the accumulation of pulses (Lustig & Meck, 2002). The operation of the switch is typically thought of as being contingent upon attentional resources (Zakay & Block, 1997; Lejeune, 1998; Zakay & Block, 1999; Lustig & Meck, 2002). However, to explore the origin of the attentional mechanism of the switch, one can consult the paper entitled *Attention and Psychophysical Time* by Kristofferson (1967) which proposes a similar component to SET's switch (Gibbon, Church & Meck, 1984). Kristofferson proposed the Theory of Central Intermittency which controls temporal integration of timing data by controlling the clock

(Kristofferson, 1967). In terms of the 'clock', it is assumed this is analogous to the pacemaker (Gibbon, Church and Meck, 1984). This assumption is valid, as Kristofferson (1967) posits that the clock generates a series of points in time. Apart from the assumption that the 'clock' emits pulses at 50 msec, Kristofferson's clock is a good analogue to the Treisman (1963) and Gibbon et al (1984) pacemaker. Kristofferson's hypothesis postulated that attention is the result of a "gating mechanism" which controls the flow of pulses from the pacemaker into a central data processor (the analogue to Gibbon et al.'s accumulator). The gating mechanism is said to operate on an 'all-or-nothing' bases. In terms of what this means, psychologically (and in the context of SET) when a subject is paying attention to time, the gate is fully opened and allows the pulses to flow to the accumulator without variability. However, when the subject is not paying attention to time, the pulses arrive at the switch, but, because the subject is not paying attention to the duration, they must switch attention to it, which introduces latency to the 'opening' of the switch. This causes a flicker in the switch mechanism, which leads to some pulses being lost.

In terms of variability in the switch, Gibbon, Meck & Church (1984) propose that variation in the switch could be introduced by varying latency to open and close the switch when gating pulses into the accumulator (Gibbon et al., 1984); which is the analogous argument that Kristofferson (1967) presented. The switch could malfunction due to a deficit in attentional resources and cause variability in timing (Gibbon & Meck, 1984). This could cause an increased latency to close the switch at the start of an interval randomly causing a flicker in the switch and consequently, durations would be underestimated (Thomas &

Weaver, 1975; Macar, Grodin & Casini, 1994; Lustig & Meck, 2002). A graphical representation of the switch is shown in figure 1.3.

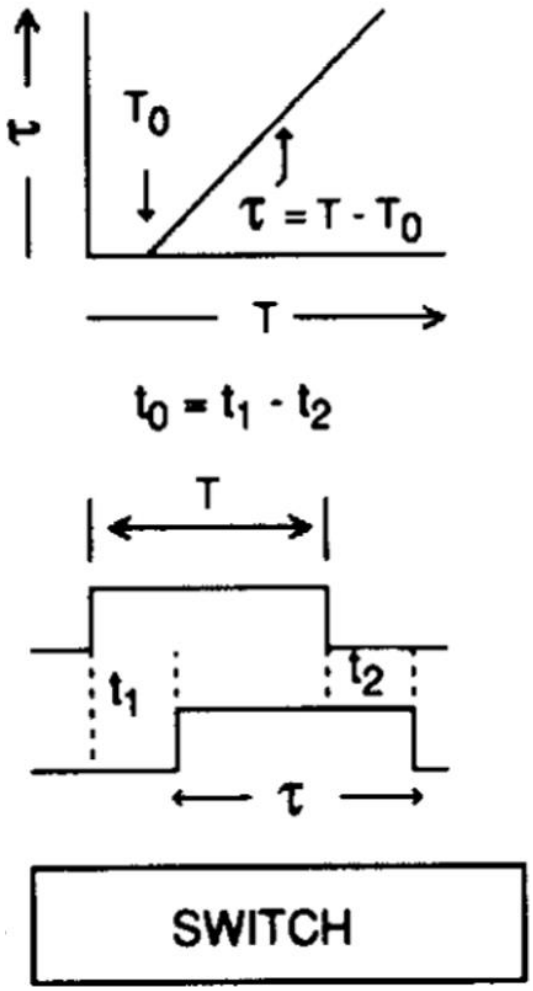

Figure 1.3: The Switch component, as visualised (and from) by Gibbon, Church and Meck (1984) shows how the effective duration of pulses, τ, is defined by the latencies of close (t_1) and open (t_2). T_0 represents the minimal signal duration, where pulses cannot be gated from the pacemaker into the accumulator. If the switch is mediated by attention resources, then the latencies for close and open can operator as if they were flickers and cause time to be perceived more slowly than it is.

1.4.4.3 The Accumulator

In the context of SET, the accumulator records the number of pulses that are gated to it from the switch; and its value can be represented, mathematically, as $N = \lambda\tau$ (where λ =

pulses emitted from the pacemaker and τ = effective duration of the pulses that pass through the switch). The accumulator is said to be in the striatum (Lustig & Meck, 2002) and much like the pacemaker, it could be argued the accumulator is mediated by the basal ganglia, though some authors suggest the supplementary motor area mediates the cognitive processes that serve the accumulator (Macar et al., 2002), while others have suggested the middle frontal gyrus (Smith et al., 2003) mediates the accumulator.

Mathematically, the accumulated pulses are dependent on two variables, λ and τ, and as a result, when either of these variables are manipulated, N can be affected. Therefore, whilst the accumulator itself is probably not subjected to internal or external variability (Gibbon, Church & Meck, 1984), the variables that determine N can be a source of variation (Wearden, 2016). The accumulator acts as a 'junction' for pulses which are either diverted to long-term memory (LTM) or short-term memory (STM). The pulses deemed as either having been reinforced (Gibbon, Church & Meck, 1984) or important (Wearden, 2016), such as in a bisection task where subjects are told to remember durations in the training stage are transferred to the reference memory (Wearden, 2016). Pulses subsequently produced for experimental trials are transferred into working memory. Once the accumulator has distributed the pulses either to short- or long-term memory, the pacemaker resets to 0

Gallistel, 1990) and the process of the pacemaker-switch starts again. The graphical representation of the accumulator is shown in figure 1.4.

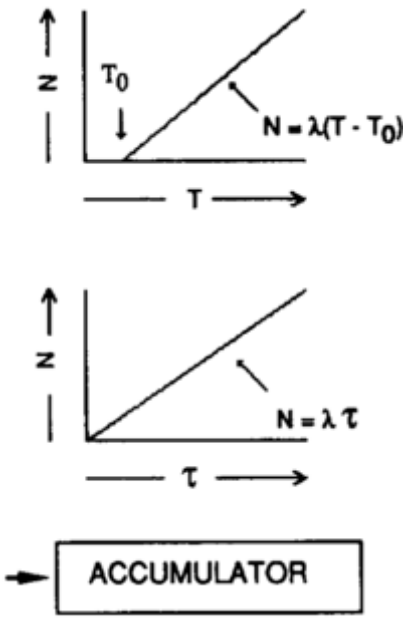

Figure 1.4: The mathematical and graphical representation of the accumulator from Gibbon et al. (1984). Here, N is the product of the pacemaker pulses (λ) and the effective duration of the switch (τ). As can be seen, the graph represents a straight-line (Gibbon, Church and Meck, 1984)

1.4.4.4 Working Memory

In SET, the memory component proposed is, according to Gibbon et al. (1984), 'realistic and simplified' and is similar to the system proposed by Treisman (1963). Working memory (WM) is considered important for any model of time perception (Teki, Gu and Meck, 2017; Ivry & Spencer, 2004). When the accumulator has collected pulses, which reflect an experimental trial (e.g., in terms of a generalisation or bisection task, those durations that subjects have not been trained on) the accumulated pulses are transferred to the WM. Mathematically, STM can be represented as $M_T = \lambda\tau$, which directly reflects the

accumulated pulses (Gibbon, Church & Meck, 1984). Since WM is assumed to reflect the contents of the accumulator, any variability in the product of the pacemaker and switch, will also be reflected in the WM component however Gibbon et al. (1984) would postulate that since WM involves a proportional transformation (e.g., as $M_T = \lambda\tau$) variability might be introduced from the WM component (WM itself can be variable in subjects – see Lett, Voineskos, Kennedy, Levine & Daskalais, 2014, for a review in the context of schizophrenia).

There is evidence for time perception being contingent on WM by attempting to isolate the WM component from the rest of the SET mechanism (Fortin, 1999). Fortin (1999) showed that tasks that require a higher degree of WM (e.g., temporal bisection task, as opposed to a temporal generalisation task) can interfere with time perception tasks. Further WM investigations have shown that when the contents of WM do not match the stimuli presented, time perception can be distorted by WM (Pan & Luo, 2011).

A graphical representation of WM is shown in Figure 1.5.

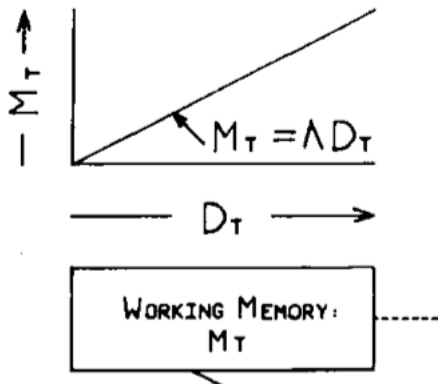

Figure 1.5: The graphical representation of WM in SET, from Gibbon, Church & Meck (1984). Mathematically, WM is represented as $\boldsymbol{M_T = \lambda\tau}$, which illustrates how WM receives the accumulated pulses from the accumulator, and therefore, reflects the value transferred from the accumulator.

1.4.4.5 Reference Memory (RM)

Reference Memory (RM) is responsible for storing reference points, or anchored durations (Gibbon & Meck, 1984); as well as acting as a reference point for subjects to refer to (Meck, 1998). To give an example, in the context of the temporal generalisation task, the subject would be asked to learn a standard duration (say 400ms). Because this duration is 'of interest' it is passed to the RM store; along with any variability that might arise from the pacemaker, switch, or WM components, respectively. RM serves two purposes in SET; the first is to store important durations, the second is to produce the scalar property (Jones & Wearden, 2003). Conceptually, it is presumed that pulses stored in WM reflect the number of ticks from the pacemaker, when they pass to reference memory, they are multiplied by the memory storage constant, K (Jones & Wearden, 2003). To give an example from a bisection task, the durations that the subjects are asked to remember during the training stage (e.g., 200ms or 800ms) would be stored in RM due to the fact they are of importance. We shall call these durations M_T and is initially housed in WM. Mathematically, the duration of importance held in RM can be represented as $M_T^* = K^* M_T$ where M_T is the value that is held within WM, and K^* is a multiplicative constant, that relates the reference interval to the experienced interval (Church, 1984; Gallistel, 1990). Pulses are transferred to the reference memory at some modifiable baud rate. This baud rate, K, influences the quantitative aspects of the represented signal duration (Allman, Teki, Griffiths & Meck, 2013). When K > 1, the subject would expect the end of a timing event to occur later than the signal duration. When K < 1, the subject would expect the timing event to occur earlier than the signal duration (Allman, Teki, Griffiths & Meck, 2013).

With respect to variability in the duration held in RM, it would include those variabilities from the pacemaker, switch and WM (Gibbon, Church and Meck, 1984); as well

as its own variability (Wearden, 2016). Overall, the RM component of SET is also susceptible to variability. A graphical representation of the RM component is shown in Figure 1.6.

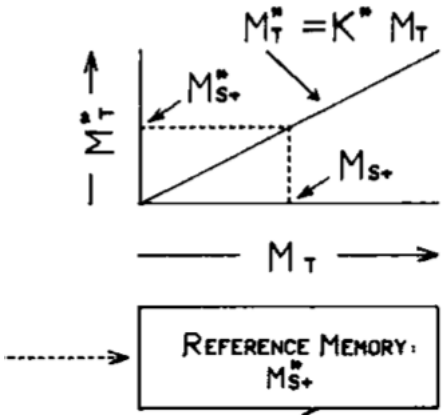

Figure 1.6. The graphical representation of the reference memory component from Gibson & Meck (1984). Clearly, as can be seen, reference memory depends on the durations in STM memory multiplied by the constant, K.

Investigations focusing on the RM component of SET has received much less attention than the pacemaker and WM components, respectively (Wearden, 2016). Evidence, of the pivotal role RM plays in time perception is shown when RM is flooded with remembered durations. This leads to subjects' performance on timing tasks decreasing (Jones & Wearden, 2003, 2004; Delgado & Droit-Volet, 2007; Ogden & Jones, 2009). Further evidence also shows that interference (Flippopoulos, Hallworth, Lee & Wearden, 2013) to memory can affect duration recall, while other studies show that the duration in RM can be completely erased (Ogden, Wearden & Jones, 2008) by subsequently acquired durations.

Curiously, the K constant in RM can be modified due to cholinergic modulation, (Ch) which has been shown to be important for both working and reference memories (Haam & Yakel, 2017), and is also said to modulate both the positive and negative aspects of

schizophrenia (Tandon, 1999). As Ch decreases in the subject, this can cause a rightwards shift in remembered durations and thus make them remembered as longer than they are by the subject (Meck & Benson, 2002). Conversely, an increase in Ch levels leads to the subject remembering the duration as shorter than it actually is (Meck & Benson, 2002). This psychopharmacological evidence of RM is indicative of RM playing an important role in time perception. Once the durations have passed through the memory components, a decision is made.

1.4.4.6 The Comparator and Decision Process

Once a duration is held in the RF, subsequent durations will flow from the pacemaker into WM. The durations which are held in RM and WM are then compared. For example, in the context of the temporal generalisation task, the subject has learned the standard duration (e.g., 400ms), which is stored in RM. Subsequently, a trial duration (e.g., 300ms) passes through the accumulator and is stored in the WM. These durations are then compared in the comparator (Gibbon, Church and Meck, 1984).

Mathematically, the comparator can be expressed as $\frac{M_T}{M_T^*}$, where M_T is equal to the duration held in WM and M_T^* is equal to the duration held in RM. When the timing from the reference and working memories are close enough, according to a specific rule in the comparator, a response is made (Gallistel, 1990; Meck, 2002). To give an example, when a trial duration is 300ms, compared to the standard of 400, this gives a value of 75%, in that the trial duration is 75% of the standard duration, therefore, there is a 75% chance the subject will say 'YES' to the 300ms duration. Research on the comparator is scarce in the literature for obvious reasons (e.g., it is difficult to isolate) however, research into decision-processes,

in the context of time perception, is an active area of research, but takes a more behaviourist – as opposed to cognitivist – approach to time perception (Ivry & Schlerf, 2008).

The comparator gives rise to a decision process, whereby the subject makes a response on the basis of some criterion factor, which Gallistel (1990) termed b. This factor, b, determines how close the ratio, in the comparator, must be to 1.0 for the subject to make a response in with respect to a trial duration. Mathematically, this can be represented by:

$$b < \frac{M_T}{M_T^*} < \frac{1}{b}$$

The ratio is determined by the duration of interest held in the RM (M_T^*) and the duration that is held in WM (M_T). The criterion factor, b, determines how close to 1 the ratio must be for a response to by made. The closer it is to 1, the more likely a 'Yes' response, in the context of the temporal estimation task, will ensue. (Gallistel, 1990).

1.4.4.7 Challenges to SET

While SET remains one of the most popular models in the psychology of time perception (Wearden, 2016) it is not without its critics. Those critics often state that due to SET's high degree of flexibility, this makes SET unfalsifiable (Wearden, 2016). Staddon & Higa (1999) argued that SET is overcomplicated and has empirical shortcomings that can only be corrected by elaborate modifications. They suggested that a model that focused only on memory. One of the empirical shortcomings of SET is how does the RM system select the correct value for comparison to the duration in WM (Bruner, Fairhurst, Stolovitsky & Gibbon, 1997); which is termed the credit-assignment problem by Staddon & Higa (1999). Staddon & Higa (1999) also argue the pacemaker concept is troubling, due to its fundamental

'at odds' approach to 'real time', though, as we have discussed, the concept of 'real time' is itself troubling. Whilst the theoretical and methodological challenges remain an issue with STE, focusing on a component that has been well researched (e.g., reference memory) might give rise to a more parsimonious explanation for behaviour.

In response to the methodological and theoretical issues identified in SET, including the apparent unfalsifiable nature of the model (Wearden, 2016), and not having a dedicated component to attention (Zakay and Block, 1994), but see Staddon & Higa (1999)) for a rebuttal, alternative models were devised. To address these theoretical issues with SET, Zakay & Block (1994) conceived the Attentional-Gate Model of Time perception. The attentional-gate model (AGM) combined Treisman's (1963), Gibbon's (1984) and Thomas & Cantor (1975) models. An illustration is shown in figure 1.7

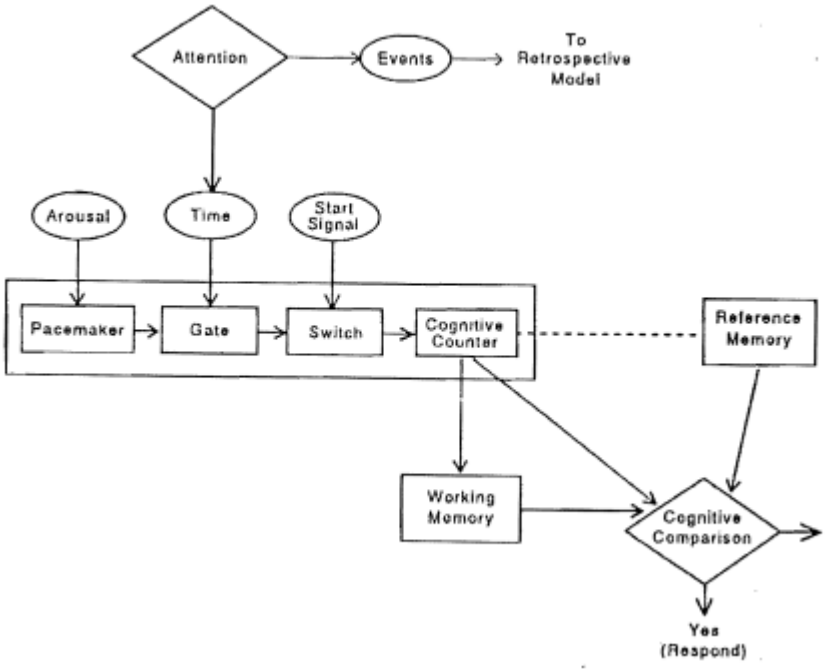

Figure 1.7: The Attentional Gate (AG) model of prospective duration from Block and Zakay, 1996.

The AGM model shares similarities with both Treisman's (1963) information-process model and Gibbon's (1977) SET model. The most obvious similarity is that both have an arousal-driven pacemaker, both have a gate, a memory component and a response component. This is unsurprising, as AGM is based on both SET and Thomas' model (Block and Zakay, 1996). The deviation, however, is that the pacemaker in the AGM model is driven by both general (e.g., circadian) and specific (e.g., stimulus-induced) arousal. In this context, the pacemaker in the AGM model would emit pulses on the basis of circadian rhythms.

This leads to the question whether subjects' internal clock pulses' faster' during peak arousal times of the day/night which introduces further variability. This model also assumes that when the organism attends to a time event (as opposed, Zakay contends, to external stimulus events) this opens a gate, which allows the pulses from the pacemaker to flow to the switch. At the onset of a given duration, the switch can open or close accordingly, however, when a start signal indicates a timing duration, the switch fully opens allowing the pulses to flow to the 'cognitive counter'. The 'cognitive counter' in this context is the analogue to the accumulator in SET. Block and Zakay (1996) justify calling it the cognitive counter, as it is controlled by cognitive processes, such as attentional, and perceptual content.

The rest of the model's components are analogous to SET. In the AG model, once the pulses have accumulated at the cognitive counter, they can be transferred to working memory though Block & Zakay add the proviso that his process occurs only when attention is deployed; which contrasts with SET, which assumes the process is a constant and automatic (Gibbon, 1977; Block & Zakay, 1996). The reference component acts in a similar vein to the reference memory component of SET; whereby previously encountered (average) pulses are stored and consequently compared to those stored in working memory, when the organism is asked to (Block & Zakay, 1996).

With respect to the attentional-gate, the author of this thesis would contend that SET can accommodate attention, as Kristofferson (1967) already envisaged a switch-like mechanism as served by attention, which served as the inspiration for the switch mechanism. Furthermore, the addition of components above SET complicates an already complex picture and ultimately, the question must be asked what the Attentional-Gate Model tells us over and above SET? In my opinion, the model is a derivative of SET that focuses more on attention. In Table 1.1, a comparison between the different models is shown to further strengthen my case for using SET within this thesis.

Table 1.1: Comparison of the different models used in time perception research.

Model	Similarities	Differences	Conceptual Overlaps
IPMT	Includes a pacemaker, counter, comparator and reference memory store, which is similar to SET	No switch or working memory components in IPMT, which are present in SET/AG IPMT claims that the 'store' leads to a verbal mechanisms and comparator to response mechanism	Pacemaker, accumulator, decision process (comparator) and reference memory (store) are similar concepts to those components in AG and SET
SET	Much like IPMT and AG, contains a pacemaker, an	Unlike IMPT, contains a specific working memory	Pacemaker, accumulator, decision process and

	accumulator and a decision process	component and a switch	reference memory are similar concepts to those components in AG and IMPT
AG	Contains a pacemaker, a cognitive counter, reference memory and comparator components	Unlike either SET or IMPT, contains a 'gate' which is mediated by attention	Pacemaker, accumulator, decision process and reference memory are all similar concepts to those components in AG and SET

1.6 Model Summary

In terms of the different cognitive timing models discussed, including IMPT, AG and SET, I have identified several conceptual overlaps, including the pacemaker, accumulator, decision process and reference memory. There are also subtle differences, including the fact that both SET and AG have a short-term memory component. In terms of the AG model, it was seen as an improvement to SET (Block & Zakay, 1992) given its inclusion of a separate gate however, Lejune (1999) states that the attentional switch (as in SET) is a more appropriate notion than an attentional gate. Furthermore, SET is considered the most popular model of human/animal timing (Reed & Randell, 2014); and has been used by several theorists within the timing literature, especially for psychopathological conditions, such as schizophrenia and ADHD (Carroll et al., 2009). On this basis, I will be using the SET model

throughout this thesis to model the data that I will collect in Experiments 1,2, 3, 4 and 5, respectively.

1.7 Chapter Summary

Chapter 1 has discussed how human timing was initially modelled, biologically and later, cognitively, with the dawn of the internal clock model of timing. The primarily focus has been an in-depth discussion on the biological models, followed by a discussion on Creelman's (1962) and Treisman's (1963) ideation of a pacemaker-accumulator clock. This laid the foundations for the SET model, proposed by Gibbon (1977) and fully developed by Gibbon, Meck & Church (1984). This model remains the most relevant and accurate for modelling human timing. It has its critics (e.g., Staddon & Higa, 1999) and, as a response, other models have surfaced, such as the AGM model (Block & Zakay, 1994) however, few have satisfactorily challenged SET's dominance and it remains, at the time of writing, the most developed model of human timing (Wearden, 2016). The model can also be used with many timing tasks. However, what other factors mediate time perception? Researchers have to be think carefully about durations used (e.g., suprasecond or subsecond), paradigm (e.g., retrospective or prospective), and the task itself (for the purpose of this thesis, temporal generalisation and temporal bisection) which shall be the focus of Chapter 2. Table 1.1. summarises the common points, similarities and differences, and conceptual overlaps.

Chapter Two: Paradigms and Measures of Time Perception?

2.1 Introduction:

In the previous chapter, we discussed the history of time perception, along with how to model it. In chapter 2, we discuss how time perception can be manipulated by a clicker condition. We also discuss what factors, paradigm (e.g., retrospective, or prospective) influence the tasks that are used to measure time perception, critical timing (e.g., suprasecond or subsecond durations), as well as how different modalities (e.g., visual and auditory) are a potential factor in time perception. Finally, we discuss those tasks that this thesis has used., which are the temporal bisection and generalisation tasks, along with the specific dependent variables of these tasks that we have used in the thesis. However, we shall first discuss external manipulations to time perception, namely the clicker.

2.2 External Manipulations to time perception

It is well known that external manipulations can influence human timing, such as temperature (Wearden & Penton-Voak, 1995), and a clicker train (Wearden, Win & Philpott, 1999). Of most interest, due to the fact that it is a manipulation that works (Wearden, Win & Philpott, 1999), is the clicker train, which was first investigated by Treisman, Faulkner, Naish & Brogan (1990). The premise was that if the pacemaker is an arousal-driven component, then, a stimulus which causes arousal should lead to a faster pacemaker (Treisman, 1963). Treisman et al.'s motivation was to provide evidence that human timing is mediated by an arousal-driven pacemaker (Treisman et al., 1990) and had chosen a clicker train on the basis of the classic research by Hirsh, Bilger and Deatherage (1956). Hirsh et al. were originally

interested in whether the perception of time was largely dependent on auditory stimulation; and hypothesised that perceived time varies with the level of auditory stimulation (Hirsh et al., 1956). Utilising a estimation task (e.g., 1, 2, 4, 8, 16s), Hirsh et al. (1956) showed that subjects overestimated the durations 1 – 4s in the presence of a noise, and underestimated durations 8 and 16s, respectively. Treisman et al. (1990) interpreted this as an auditory clicker being able to manipulate pacemaker speed. In the first of several experiments, Treisman et al. conducted a estimation task on subjects in which they had to estimate.

Whilst Treisman et al.'s paper is complex, its key finding was that a clicker train appears to manipulate pacemaker rate (Treisman et al., 1990). Along with Hirsh, Treisman's study appeared to show that an auditory clicker train could manipulate pacemaker rate and thus provided experimental evidence for the pacemaker however, the pacemaker Treisman et al. (1990) discusses is an oscillator pacemaker, as opposed to the SET pacemaker. A more contemporary article by Penton-Voak, Edwards, Percival & Wearden (1996) utilised a clicker train and explained their results in terms of SET. In their first experiment (which is relevant to this thesis) they applied a clicker train to the temporal generalisation task. The temporal gradient shifted to the left (e.g., lower peak) in the presence of the clicker; indicative of lengthened durations (Penton-Voak et al., 1996). In conclusion to their study, Penton-Voak et al. argued that the clicker train lengthened durations by arousing the pacemaker which accords with Treisman (1963) and Gibbon's (1977) pacemakers utilised in their models (Wearden, 2016). It appears that the clicker can manipulate duration perception relatively safely and have a similar effect of modifying time perception as pharmacological agents (Rammsayer, 1991; Penton-Voak et al., 1996), though, as Penton-Voak et al. note, the effect size is larger for pharmacological agents than the clicker (Penton-Voak et al., 1996).

A later study, utilising the temporal bisection task (Wearden, Win & Philpott, 1999) provided further evidence for the clicker speeding-up the pacemaker. Once again, Wearden et

al. (1999) explained this speeding-up effect in terms of the pacemaker being 'aroused' by the clicker train; or alternatively, that the clicker train is 'assimilated' in the overall length of the duration. In either case, the evidence suggests that the click train is effective in mediating the pacemaker; and provides further evidence for a cognitive model of time perception.

Whilst most researchers assume the clicker arouses the pacemaker, it is entirely possible that other components of SET are affected by the clicker. For example, perhaps the duration is modified by the clicker because the subject misremembers it. However, what effect does subsecond and suprasecond durations have on time perception? And furthermore, how do we measure time perception? These issues shall be considered.

2.3 Retrospective and Prospective Paradigms of Time Perception

Prior to any conversation regarding time perception tasks, one must establish a general paradigm surrounding prospective or retrospective timing (Grondin, 2010). These paradigms are the prospective and retrospective approaches, respectively. In the prospective paradigm, the subject is informed they are partaking in a timing task (Eisler, Eisler & Montgomery, 2004). Conversely, in the retrospective task, the subject is not told they are partaking in a timing task (Block & Zakay, 1997; Eisler, Eisler & Montgomery, 2004 Grondin, 2010) until after the task has ended. In both cases, the subject is asked to judge the length of a duration (Wearden, 2016).

James (1890) concluded that numerous variables influence the retrospective and prospective paradigms (Block & Zakay, 1997). Terms, such as retrospective, some authors contest, are confusing (Wearden, 2014) as all time judgements are 'retrospective' in the sense that a subject is asked to recall the duration that was presented to them, in the past. To abate this confusion, researchers, such as Block (1990) refer to prospective timing as *Experienced Time* (Block & Zakay, 1997; Block, 1990) due to the subject, in the prospective paradigm,

intentionally encoding temporal information as a critical part of the timing experience (Block & Zakay, 1990). Conversely, the retrospective paradigm has been termed *Remembered Durations* (Block & Zakay, 1997; Block, 1990) due to the subject, in this paradigm, retrieving temporal information from memory later (Block, 1990). However, the author of this thesis (Hopkins) has concluded that the terms, prospective and retrospective timing, are appropriate.

In the majority of studies in time perception, the prospective paradigm is used; with very few studies adopting the remembered durations paradigm (Block & Zakay, 1997). Importantly, some authors contend that the prospective and retrospective paradigms use different cognitive mechanisms (Wearden, 2016; Grondin, 2010). Prospective durations are claimed to be mediated by attentional resources (Wearden, 2008); whilst remembered durations are claimed to be mediated by long-term memory (Block, 1990); which makes intuitive sense. Evidence supporting this claim has its origins in an article by Hicks, Miller & Kinsbourne (1976) who found that prospective estimates of time decreased monotonically with the amount of information processed duration an interval (e.g., a greater amount of information decrease the estimate of durations) which Hicks et al. interpreted as indicative of prospective timing being contingent upon attention. Block (1992) gave further evidence of both paradigms being served by different mechanism.

Prospective durations are claimed to increase when subjects allocate greater attentional resources to processing temporal information e.g., lengthening of durations. Evidence, as well as common-sense intuition demonstrates that when the human pays more attention to time, it appears to lengthen (Wearden, 2016). Conversely, retrospective durations are said to be minimally impacted by attentional resources (Block & Zakay, 1998). Despite there being evidence for different cognitive functions driving these different paradigms,

Brown (1985) contends that both paradigms are remarkably similar however, the choice of paradigm can have profound methodological and theoretical ramifications.

There are advantages and disadvantages to both paradigms. The retorspective paradigm is considered the least popular of the paradigms (Ordgen, Wearden, Gallagher & Montgomery, 2011, Block & Zakay, 1997; Brown & Stubbs, 1988), despite the retrospective paradigm possessing a greater degree of ecological validity than the prospective paradigm (Block, 1990) by mimicking real-life environments with respect to time perception. Since the person in the real-world environment would not actively be monitoring the time, and never asked to determine time (Block, 1990), some others contend the retrospective paradigm possesses greater ecological validity (Brown, 1985). Given that a retrospective and prospective timing task has been used in this thesis, it is pertinent to discuss the prospective and retrospective paradigms.

Some authors further claim the retrospective paradigm might reveal the effects of variables effecting timing that would otherwise be missed by the prospective paradigm (Brown & Stubbs, 1988). Despite the apparent strengths of the retrospective paradigm, the methodological and theoretical flaws within the retrospective paradigm should be a cause of concern. Methodologically, as soon as the subject is asked to make an estimation of time, the retrospective paradigm becomes a prospective task and last for a single trial only (Wearden, 2016). Theoretically, it is unknown whether the subject's estimation of a duration is guesswork thereby limiting its accuracy (Brown & Stubbs, 1988). Cognitive biases could also render the retrospective paradigm vulnerable due to its dependency on long-term memory, such as representativeness, availability, and anchoring (Zakay, 1990). Statistically, the retrospective paradigm would normally utilise a between-subjects design, which is less powerful than a within-subjects design (Cremers, Wager & Yarkoni, 2017) and thus, makes group comparison difficult without a very large sample size. Conversely, the prospective

paradigm can be used with a within-subjects design. Finally, in terms of how this effects task selection, one cannot readily run TB or TG tasks in the retrospective paradigm meaning the tasks that can be used as limited (Wearden, 2016) and restricted to TR tasks, in which the subject has to be deceived.

In summary, whilst the retrospective paradigm is ecologically valid, we cannot ascertain whether the subject has simply 'guessed' the duration. Nonetheless, it remains an important tool for assessing reference memory in subjects. Conversely, the prospective paradigm lacks ecological validity but is straightforward to implement. Both paradigms have their advantages and disadvantages. The choice of paradigm and critical timing (e.g., subsecond or suprasecond durations) dictate which timing task can be used.

2.4 Critical Timing

Researchers often concern themselves with subsecond (< 1s) and suprasecond (> 1s) durations when discussing time perception, as durations experienced (be they subsecond or suprasecond durations) are important both at an individual level, and a societal level (Buonomano, 2007). There are distinct and important differences associated with the subsecond and suprasecond duration, including processes that are associated with them, and the underlying mechanisms of them; both, however, are considered independent.

The idea of separate timing mechanisms is not a new one; with Mūsterberg (1889, cited in Rammsayer, 1999) first hypothesising that humans possess a sensory mechanism for processing durations under one-third of a second, and other mechanisms for longer durations. The subsecond duration is thought to be associated with a whole host of critical cognitive processes (Buonomano, 2007) and motor control (Lewis & Miall, 2003) and is typically thought to be an automatic process (Mitrani, Shekerdjiski, Gourevitch & Yanev 1977) and is generally favoured by researchers seeking to delineate the complex concept that is time

perception (Grondin, 2010). The suprasecond durations are thought to be mediated by higher cognitive processes, such as memory, attention and general cognition (Mitrani et al., 1977). Both the subsecond and suprasecond durations shall be reviewed in this part of the thesis.

2.4.1 Subsecond Durations

Subsecond durations are thought to be more sophisticated and complex than suprasecond durations (Grondin, 2010; Mauk & Buonomano, 2004), and are of critical importance for speech generation and recognition as well as motion detection and coordination visual and haptic processing (Buonomano & Karmarkar, 2002). Morse code is an example of the sophistication and complexities that underlie subsecond durations. For example, subject must first discriminate between short or long tones accurately and then discriminate between the elements between those tones. Finally, the subject would have to perceive the sequence of tones and determine which sequence relates to a message (Mauk & Buonomano, 2004) all within milliseconds which demonstrates how speech recognition, movement, and coordination are reliant upon subsecond timing.

Speech recognition relies on the sequencing of syllables and speech segments to formulate the recognition of a word (Mauk & Buonomano, 2004; Lisker & Abramson, 1964). The complexities of speech recognition also mean that subjects are required to constantly discriminate between vowels (Lehiste et al., 1976). Deficits in subsecond timing can thus have a detrimental impact on speech recognition (Mauk & Buonomano, 2004). Motor control also utilises short durations because most movement control involves the coordination of agonist muscles, which initiate a given movement, and antagonist muscles, which are equivalent to a brake (Mauk & Buonomano, 2004). The subject would have to coordinate when to begin a muscle movement and when to move it, such as driving a vehicle, which requires accurate fine motor control (Wearden, 2016). Furthermore, shorter durations are

associated with cognitive sequencing (Grondin, 2010), which is vital in determining the nature of an event (Capa, Duval, Blaison & Giesch, 2014), and illustrates how deficits in subsecond durations could have a perilous consequence, such as in conditions like schizophrenia (Reed & Randell, 2014).

These examples demonstrate how subsecond durations are imperative for everyday behaviour, such as speech recognition and perception, as well as fine muscle movement. However, how are subsecond durations explained? The subsecond durations can be theoretically modelled by SET (e.g., Rammsayer, 1997; Wearden, 2016); specifically, the pacemaker component. Since many authors suggest that the pacemaker rate is positively (e.g., the greater the level of D2 receptors, the faster the pacemaker pulse rate) related to dopamine D2 receptor activity (Church, 1984; Meck, 1996; Rammsayer, 1999) suggesting that deficits in this area would give rise to aberrations in subsecond durations; which is one of the working hypotheses of why schizophrenia (Reed & Randell, 2014) manifests as timing deficits in subsecond durations.

2.4.2 Suprasecond durations

Suprasecond duration perception (SDP) are also critical for decision making and conscious time estimation, (Hayashi, Kantele, Walsh, Carlson & Kanai, 2014), learned behaviours (Brunner, Kacelnik & Gibbon, 1992) and foraging (Pyke, Pullman & Charnov, 1977) suggesting that SDP is critical for higher cognitive processes, such as memory, attention, and other higher cognitive processes (Grondin, 2010; Hellstrom & Rammsayer, 2004; Block & Zakay, 1994, 1997). A typical example in how the human would utilise suprasecond durations is when asked to estimate how long a traffic light takes to change from red to green (e.g., estimate the duration of the red light). In such a scenario, the subject would first pay attention to when a green light would display red. They would continually attend to

the duration of the red light by employing a counting strategy. Then, when the light would change from red to amber, the duration length of the red light would be held in working memory. Since the subject had been asked to give an estimate of the length of the light remaining red, this duration would be transferred from working memory to reference memory. Then, when asked to estimate how long the light was red, they could recall this information from the reference memory; which demonstrates how suprasecond durations utilise information from higher cognitive processes (Block, 1992; Wearden, 2016).

2.4.3 Evidence for independence of subsecond and suprasecond durations

Many researchers assume there is an element of independence between subsecond and suprasecond durations (Grondin, 2010). In one of the earliest studies to provide evidence for difference between subsecond and suprasecond durations, Mitrani, Shekerdjiiski, Gourevitch & Yanev (1977) conducted a study in which subjects were given LSD_{25} and mescaline, respectively. The subjects were asked to complete a timing task before the administration of the narcotics, and then after. There were no changes in subsecond duration perception (Mitrani et al., 1977). However, in a landmark study, Rammsayer (1999), investigated the effects of the dopamine antagonist, haloperidol, and the benzodiazepine, midazolam on durations of both subsecond and suprasecond nature; reasoning that suprasecond durations rely on memory, and since amnesic effects of midazolam are known, if there was a difference related to memory, subjects in the midazolam group should exhibit differences. This is the finding Rammsayer (1999) reported only in the suprasecond durations. Conversely, if the dopaminergic levels in the basal ganglia mediate subsecond duration perception, one would expect haloperidol to effect subsecond durations, but not midazolam. Once again, this is what Rammsayer (1999) reported, which he explained in terms of subsecond durations being processed by dopamine levels in the basal ganglia, and suprasecond durations being mediated by memory (Rammsayer, 1999). However, as Penney & Vaitilingam (2008) suggest, the

comparison of critical timing appears to be quantitative as opposed to qualitative; meaning that, fundamentally, the subsecond and suprasecond durations are likely served by the same cognitive processes in the brain, but manifest differently.

In summary, both subsecond and suprasecond durations are considered in time perception research. Some authors contend that different mechanisms are responsible for each of the durations though, these are often considered quantitative as opposed to qualitative (Grondin, 2008). However, despite the differences of critical timing considered quantitative, it does affect what timing tasks can be used. For example, one could not utilise a temporal estimation task for subsecond durations; as it is likely the subject could not perceive durations shorter than 400ms. Therefore, it is critical that we review some of the methods used in time perception research however, the review is not exhaustive.

2.5. Temporal Bisection

The Temporal Bisection task is one of the most well used tasks in the timing literature (Wearden, Win & Philpott, 1999); and has seen wide use in psychopathologies, including schizophrenia (Carroll et al., 2009; Elvevåg et al., 2003) and Schizotypy (e.g., Reed & Randell, 2014; Lee et al., 2006). Given its wide use in time perception, as well as its inclusion in measuring schizotypy, it would be pertinent to include the the temporal bisection task in this thesis. However, before a methodological justification is provided, a wider discussion on the temporal bisection task is necessary. Any discussion on the temporal bisection task must involve a wider discussion on psychophysics (Fechner, 1860; Gundlach, 1993). The temporal bisection task is often thought of as being developed by Church and Deluty (1977) in animals, and Wearden (1991) in humans however, Wearden (2016) fails to mention (justifiably) that the temporal bisection task has its roots, as an experimental method, in psychophysics, and namely, in a two-response classification experiment (Macmillan &

Creelman, 1991) in which the subject is asked whether, in the context of time perception, a duration is ‘short’ or ‘long’. Mathematically, the temporal bisection task can be represented by a function:

$$R = f(S)$$

In the above formula, R is a response given by the subject, that is equal to the function *f(S),* which is a duration presented to the subject. To give an example in the context of the temporal bisection task, R = YES/NO, which depends on S = (200 – 800ms). In other words, the subject’s response is a function of the durations presented in the temporal bisection task.

The temporal bisection task belongs to a family of methods termed discrimination methods. In these discrimination methods, the subject is involved in determining whether a duration is short or long, as is the case for the bisection task. The bisection task is often associated with SET (Wearden, Win & Philpott, 1999), hence its inclusion in the thesis. The task is a prospective timing task (Penney & Cheng, 2018). The task, due to its prospective nature, allows multiple trials to be presented to the subject thereby allowing an accurate and robust estimate of timing behaviour (Wearden, 2016; Penney & Cheng, 2018).

The task has seen extensive use since its introduction, in the context of timing (e.g., Church & DeLuty, 1977; Wearden, Philpot & Win, 1998; Reed & Randell, 2014) and is often used for studying individual differences (Carroll et al., 2008; Carroll et al., 2009), as well as addressing fundamental questions concerning the cognitive, perceptual and neural mechanics that underlie time perception (Church & Deluty, 1977; Wearden, 2016; Penney & Cheng, 2018). The bisection task, used in the context of duration discrimination, has its roots in the non-human animals, dating back to at least the 1940s (e.g., Cowles & Finan, 1941; Heron, 1949; Stubbs, 1968; Bovet, 1969) however, it was Church & Deluty (1977) who introduced

the bisection task in its modern form. The task was later extended to human subjects (Allan & Gibbon, 1991; Wearden, 1991; Wearden, 2016) and has proved popular, with over 100 studies utilising the bisection task (Penney & Cheng, 2018). It has many procedures when implementing it.

2.5.1 Procedure – temporal bisection

As Wearden et al., (1999) notes, there are many variants of the temporal bisection task, that utilise both auditory (e.g., durations represented by tones) and visual (e.g., durations represented by visual stimuli) modalities, however, the procedures for most of the bisection tasks, both visual and auditory, follow a similar procedure. The procedure of the task is as follows: the subject partakes in a training phase. Here, they are asked to learn so-called 'anchors' that accord with SHORT or LONG durations that repeat around 3 to 5 times (Wearden et al., 1999). The SHORT or LONG anchors are arbitrarily chosen however, they usually accord with the shortest and longest durations among stimuli range used in the temporal bisection task (Reed & Randell, 2014). In a typical training phase, the subject is presented with the duration (e.g., SHORT), followed by whether it was short or long (e.g., SHORT). For explanatory purposes, we shall presume the anchor durations are 200 and 800ms, respectively. Once the subject has learned the anchors, the experimental stage begins, in which comparison stimuli between SHORT and LONG are presented, along with SHORT and LONG anchors themselves. The comparison stimuli are usually 5 intervals between 200 and 800ms (e.g., 300, 400, 500, 600, 700ms). The spacing is usually linear but not exclusively (Wearden & Ferrara, 1995; Wearden, 2016), with logarithmic spacing sometimes applied (Wearden, 2016). Once the subject is presented with a comparison stimulus, they are asked to decide if it is SHORT or LONG by means of a keyboard button.

Typically, each stimulus is presented 20 times for a total of 240 trials (Reed and Randell, 2014) however, 10 presentations are sufficient for a total of 120 trials.

The bisection task allows researchers to implement a manipulation, typically in the form of a click train (Wearden, Philpot & Win, 1999). Here, a click train of an arbitrarily-defined duration, is inserted just before the presentation of each trial. Such experiments are typically conducted as a within-subject design; whereby the subjects first complete the 'baseline' condition, followed by the 'clicker' condition (Wearden, Philpot &Win, 1999). The bisection task, in this thesis, utilised the clicker manipulation, and presented a click train before the presentation of a duration.

2.5.2 Data Presentation – temporal bisection

In terms of data presentation, the probability with which the subject classifies a duration as LONG is often plotted across durations. The probability of long concerns the ordinate-axis, while the duration concerns the abscissa-axis (Church & Deluty, 1977; Wearden, 1991; Wearden, Philpot & Win, 1999; Carroll et al., 2008; Penney & Cheng, 2018). A subject who has successfully learned the anchor durations will usually manifest as near 0% of LONG for SHORT and near 100% of LONG for LONG. The resulting data takes the form of one of many functions, though the data that arises can be modelled by (among others) a Gudermannian function (Altun, 2018), given by:

$$f(x) = 2\arctan\left(\tanh\left(\frac{x}{2}\right)\right)$$

From this function, there are a number of parameters of interest to timing researchers, which can be derived from the function, which include the Point of Subjective Equality

(PSE), the Difference Limen (DL) and the Weber Fraction (WF). Each of these parameters of interest will be discussed, along with their psychological meaning. These parameters are especially pertinent to this thesis, given that they are involved in measuring the temporal perception of durations (e.g., overestimation or underestimation) and sensitivity to durations.

2.5.3 Percentage of Long Responses

Most researchers, who have used the temporal bisection task, present the data as the sigmoidal function. In simple terms, the durations used in the experiment are presented on the abscissa and the percentage of which the subject thought the duration corresponded to the long anchor is presented on the ordinate. Equally, we could present the short anchor on the ordinate but, most researchers have opted to use long durations (Wearden, 2016). In terms of what qualitative information can be derived from this function, especially when investigating group differences (e.g., schizotypy) or manipulations (e.g., a clicker); a leftward shift (relative to a baseline) is indicative of the subject responding long to durations, while a shift to the right suggests the subject tends to respond short to the presented durations. Such a function usually takes the form as that shown in Figure 2.1

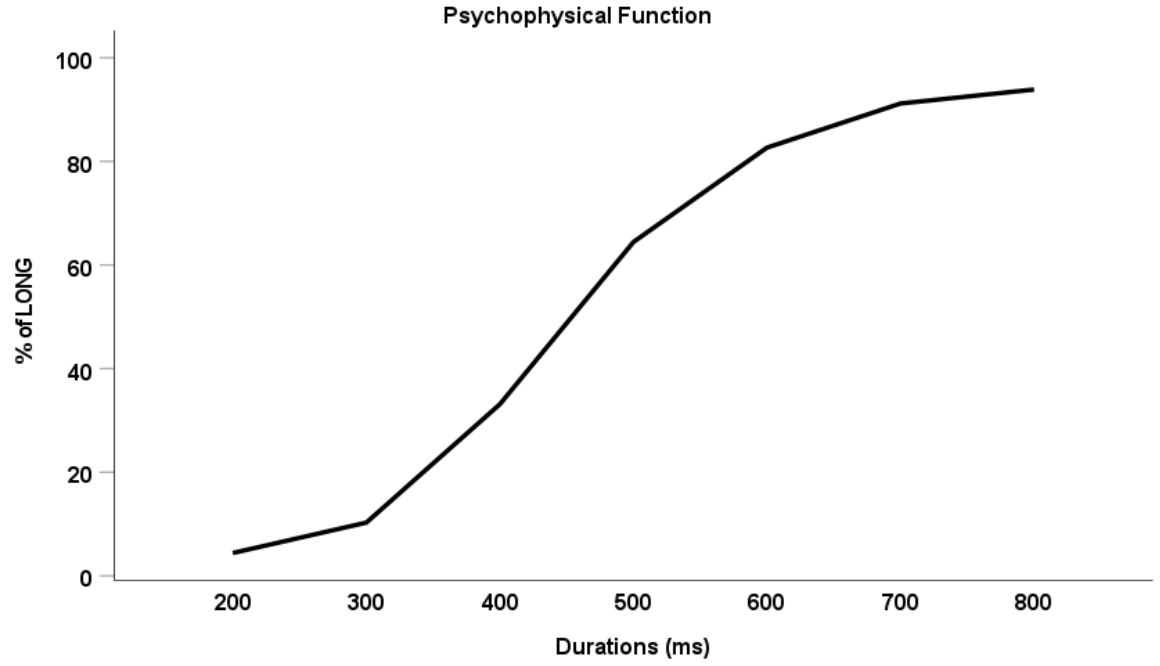

Figure 2.1: The typical psychophysical function, in the context of a temporal bisection task using a 200 – 800ms duration range. As can be seen, the percentage of LONG is plotted on the ordinate and the durations, presented to subjects are presented on the abscissa.

What is apparent is that if subjects have successfully learned the anchor durations, they manifest as a near 0% for short anchors and a near 100% for long anchors on the graph. Clearly, in figure 1.8, this is the case. The curve takes the form of the psychophysical function. Numerous curves can be plotted on the plane, which is useful for examining manipulations (e.g., baseline vs. clicker) or individual differences (schizophrenia vs. non-schizophrenia). An example, in the context of a baseline vs. clicker manipulation, is shown in figure 1.9.

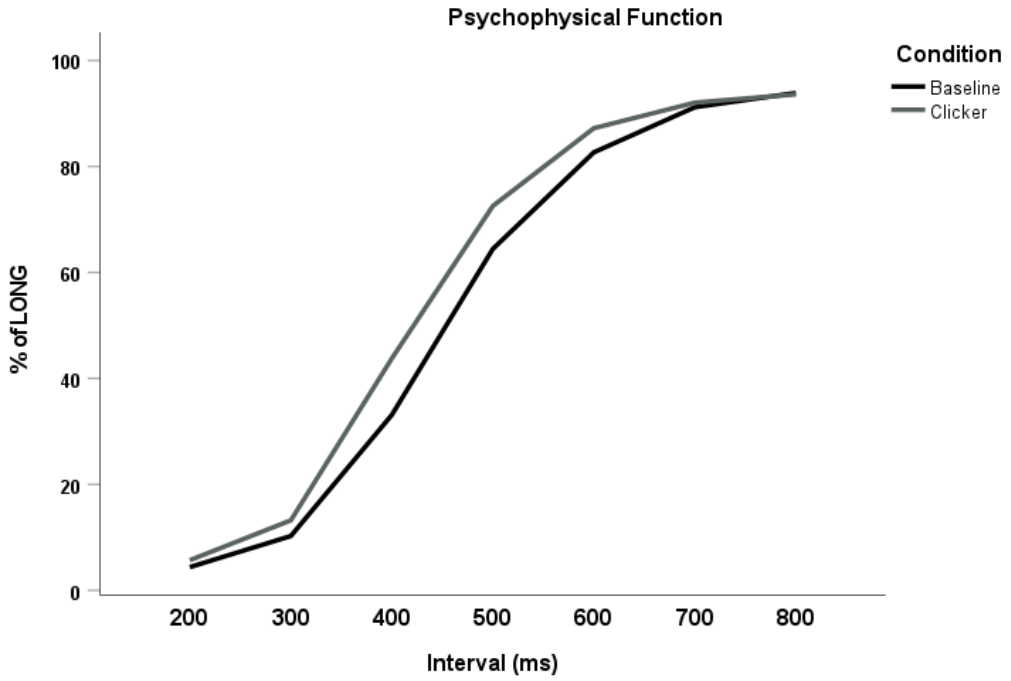

Figure 2.2: As can be seen, the subjects were subjected to both baseline and clicker conditions; represented as curves in the plane. This is useful, as it allows researchers to infer timing behaviour, in manipulations of choice (e.g., clicker) and also allows a robust statistical analysis.

The presentation of the data in figure 2.2 is useful, as it allows researchers to statistically analyse the significance of the relative shift of the function, compared to baseline (Wearden, Philpot & Win, 1999). Such an analyse is typically conducted by a repeated-measures analysis of variance. This presentation of the data is popular however, atheoretical – or indeed, theoretical – interpreting of inconsistent effects is difficult (Penney & Cheng, 2018); therefore, the PSE, DL or WF is typically analysed however, these values are derived from the psychophysical function. Analysis of the psychophysical function is the main measure this thesis has used in experiments concerning the temporal bisection task. Given the psychophysical function is often what is analysed in the context of Schizophrenia and Schizotypy, it is important for the thesis to include the percentage of long responses.

2.5.5. Point of Subjective Equality

The magnitude of the sensation that is evoked by the stimulus is considered critical in psychophysics. This magnitude of sensation (e.g., the PSE) is the duration value at which the subject is likely to equally classify as both short and long (Church & Deluty, 1977; Wearden, Philpot & Win, 1999; Reed & Randell, 2014). Mathematically, one would expect this to be the mean of the short and long anchors (e.g., 200 + 800 / 2 = 500ms) however, this is not often the case when researching humans, especially those with conditions (e.g., Reed & Randell, 2014).

In terms of calculating the PSE, there are numerous statistical and mathematical methods to derive the PSE (Pearson, 1908); with the most common method being introduced by Church & Deluty (1977) from the psychophysical function, as shown in figures 1.8 and 1.9 above. In their method, a straight line is fitted to the three most central durations, and from this equation, the half-way point is taken to indicate the PSE.

The location of the PSE has been deemed theoretically important (Allan, 2002; Wearden, 2016; Penney & Cheng, 2018) as, for example, if the PSE occurs at the geometric mean of the intervals, or the arithmetic mean of the durations has been interpreted as indicating whether the subject has a temporal scale that is linear or logarithmic and also, according to Penney & Cheng (2018) whether the response decision rule of SET, operates on the basis of ratios or differences between the elapsing time on a trial and memory representation of previously reinforced or learned durations (Penney & Cheng, 2018).

However, in human subjects, the evidence does not indicate conclusively whether timing is linear or logarithmic (Penney & Cheng, 2018). Once again, whether timing is linear or logarithmically is a quantitative issue, as opposed to a qualitative explanation. Ultimately, irrespective of the location of the PSE, it can be taken to be a single measure that represents

the range of durations used in a given study. The usefulness of such a single metric is obvious; in that researchers can reduce complex data into a single measure, such as in schizophrenia (Carroll et al., 2008).

2.5.6 Difference Limen

The difference limen (DL) is taken to reflect temporal sensitivity (Penney & Cheng, 2018; Wearden, 2016) in the subject and is loosely defined as the minimum physical difference between two stimuli which the subject can notice (Bausenhart, Luca & Ulrich, 2015). Given the psychophysical origin of the temporal bisection task, it is unsurprising that the DL is referred to in the psychophysical literature. The definition of the difference limen can be explained in terms of the bisection task: to give an example, the subject should not be able to differentiate between 200ms and 300ms (most of the time, they will respond 'short' to such durations) however, when durations, such as > 450ms (assuming that is the subject's PSE) are to be judged, the subject will respond 'long' as the duration has crossed the PSE or, in the original German literature, the Reiz Limen (Woodworth & Schlosberg, 1938) or the absolute threshold however, at precisely what moment will the subject confidently say a duration is 'long'? Given there is a discussion on whether Schizophrenics are more or less precise at recognising durations (Elvevag et al., 2003), the DL is an important measure to include within this thesis.

In the bisection task, if durations are presented that are longer than the PSE, the subject will respond 'long' at a higher percentage. Mathematically, the DL is the value that can be added to the PSE for subjects to define it as 'longer' e.g., if a subject's PSE = 500ms and their DL = 70ms, then any duration equal to 570ms will be deemed confidently long (Ferchner, 1860; Woodworth & Schlosberg, 1938; Stevens, 1988; Macmillan & Creelman, 1991). However, in the timing literature, the DL is often seen to represent temporal sensitivity (Wearden, 2016; Penney & Cheng, 2018), or the gradient of the psychophysical

function, where a lower DL indicates greater temporal sensitivity than a higher DL (Penney, Gibbon & Meck, 2000; Wearden, 2018). This accords with the psychophysics literature, as a lower DL would accord to the subject being able to respond 'longer' closer to the PSE (e.g., if PSE = 500ms and DL = 20ms, then the subject would respond 'long' at durations greater than, or equal to 520ms, therefore, they have greater temporal sensitivity), which implies greater temporal sensitivity. Numerically, it is calculated by the following formula:

$$\mathrm{DL} = \frac{1}{2}(\mathrm{Q}_3 - \mathrm{Q}_1)$$

Where Q_3 = 75% of long, and Q_1 = 25% of short, relative to the subject's individual psychophysical function.

2.5.7 Analysis of Temporal Bisection

There are two approaches in analysing the bisection data (Penney & Cheng, 2018): atheoretical or theoretical. In the atheoretical approach, the experimenter usually compares the probability of long responses between conditions to assess whether there are differences between conditions (e.g., baseline and click train). To give an example: if one had a bisection task with a clicker and baseline condition, then one would compare the probability of LONG responses for each with a Repeated-Measures ANOVA, as figure 2.3 shows.

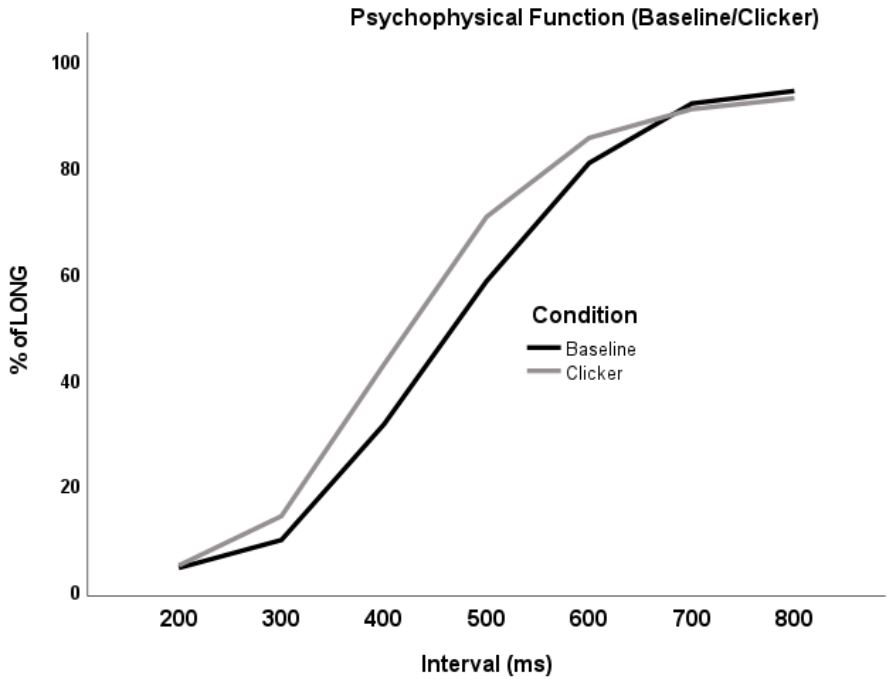

Figure 2.3: As can be seen, the baseline and clicker conditions plotted. The typical effects of near 0% and near 100% for SHORT and LONG, respectively, manifest on it.

Usually, the percentage of Long response are analysed using an Analysis of Variance (ANOVA) or a regression analysis however, despite the ease of this atheoretical approach, there are several shortcomings (Penney & Cheng, 2018). One of those shortcomings is, that if there is a main effect of condition (e.g., click train), this might conceal individual probe differences. Furthermore, simple-effects analysis (i.e., paired-samples t-test) would have to be conducted for a Condition x Duration interaction, which might lead to contradictory reports (e.g., a difference might arise at 400ms for schizophrenia in baseline, but at 300ms in clicker, for example) Therefore, most researchers usually use the percentage of long as a visual aid to readers, as opposed to a theoretical analysis; with the PSE, WF and DL being subjected to analyse (Wearden, Philpot & Win, 1999; Meck, 2006; Wearden, 2016; Penney & Cheng, 2018) the temporal bisection task. However, I interpret this as a simplification, and

believe the % of long responses captures the differential behaviour of subjects that the PSE ignores.

As already discussed, the PSE is the hypothetical duration in which subjects would perceive of being both long and short (Church & DeLuty, 1977; Reed & Randell, 2014). The duration of the PSE can reveal putative information about a subject's timing, overall. For example, a PSE with a lower value than a PSE with a higher value would imply the subject would overestimate durations (Wearden, Philpot & Win, 1999). Conversely, a higher-value PSE would imply the subject underestimates durations (Reed &Randell, 2014). The DL, as also reflects the steepness of the psychophysical function (Penney & Cheng, 2018); and provides a metric of temporal precision. A subject who has a higher DL will be less sensitive to temporal durations than a subject with a low DL (Penney & Cheng, 2018). In the context of classical psychophysics theory (Woodsworth & Schlosberg, 1938), the DL is the duration at which subjects will classify a duration as either long or short, relative to the PSE (e.g., if PSE = 450ms and DL = 50ms, then PSE ± DL = SHORT/LONG). Finally, the WF provides a metric of temporal acuity corrected for the magnitude of the duration (Penney & Cheng, 2018). However, this definition is ambiguous therefore, the classical psychophysical definition of the WF is more appropriate; which conceptualises the WF as a value of 'sensory noise' (Woodworth &Schlosberg, 1938; Ekman, 1959).

In the context of the bisection task the WF could be summed to each of the intervals (e.g., 300ms + WF) which implies that a higher WF would imply greater 'sensory noise' to the duration than a lower WF (Penney & Cheng, 2018). The PSE, LD and WF have the further advantage that they can be analysed with a further variety of statistical tests than the percentage of long responses (Carroll et al., 2008; Reed & Randell, 2014), such as discriminant analysis, multiple regression, correlation, and multiple analysis of variance (MANOVA) though, the issue remains that the PSE loses some of the nuances associated

with differential effects. The PSE, DL and WF thus capture the essence of timing and manifests it as a single data point (Reed and Randell, 2014); which is something that is difficult for the percentage of LONG responses, alone, to do. However, given the theoretical issues with the PSE, DL and WF (Penney & Cheng, 2018), some researchers might decide to focus on the percent of LONG responses, instead.

2.5.8 Theoretical Analysis and issues

One of the strengths of the bisection task is the fact it can be analysed atheoretical; which also implies it can be analysed from many theoretical viewpoints. As discussed, temporal bisection is usually associated with SET (Church & DeLuty, 1977; Gibbon, Church & Meck, 1984; Wearden Philpot & Win, 1999; Reed & Randell, 2014; Penney & Cheng, 2018); however, since the temporal bisection task has its roots in classical psychophysics (Woodsworth & Schlosberg, 1938) it is not obliged to any model. To put this into context, temporal bisection data can be modelled with SET, and indeed, any pacemaker-accumulator model (e.g., SET, ATT and information-processing). To give an example, the PSE can be thought of as representing the pacemaker speed (e.g., the PSE represents whether the subject has overestimates or underestimates durations, based on its value relative to a baseline). Conversely, the LD and WF can be interpreted as sensitivity to timing (Penney & Cheng, 2018); which in the context of SET, could represent the 'switch' component. The Temporal Bisection task can also be used in the context of Block & Zakay's (1997) attentional gate model. Hence, we have selected the Temporal Bisection task for its flexibility in applying it to a wide range of atheoretical and theoretical models of timing.

There are, however, theoretical, and methodological issues with the temporal bisection task, in spite of its relative popularity (Reed & Randell, 2014; Wearden, 2016). One

of those issues concerns the space between durations can impact the location of the PSE (Penney &Cheng, 2018). For example, Church & Deluty (1977) did not find evidence for an effect of stimulus spacing on the PSE. Conversely, Raslear (1983) did find evidence that rats had a significantly higher PSE for linearly spaced stimuli, as opposed to logarithmically spaced durations. In the human analogue, Wearden (1991) would report the PSE at the arithmetic mean for conditions using 200 vs. 800ms, and again for 100 vs. 900ms however, they subsequently found (e.g., Wearden & Ferara, 1991) a leftward shift of PSE in logarithmically spaced intervals, as opposed to linearly spaced intervals. Wearden & Ferrara (1995) would propose this anomaly arises due to subjects 'somehow' calculating the midpoint of the stimulus range. To give an example for the linearly-spaced 200 – 800ms range:

$$\mathrm{m} = \frac{(200 + 300 + 400 + 500 + 600 + 700 + 800\mathrm{ms})}{7}$$

Would give a mean of 500ms. Conversely for the logarithmically spaced intervals:

$$\mathrm{m} = \frac{(200 + 252 + 317 + 400 + 504 + 646 + 800\mathrm{ms})}{7}$$

Gives a midpoint range of 444ms. Attempts have been made to develop a model to account for these (e.g., Wearden, 1991; Penney & Cheng, 2018) apparent results; and none of them can account for the effect of stimulus spacing on L/S ratio. Thus, there is ambiguity on what range to use, with the most common being 200ms and 800ms, however, irrespective of which spacing durations are used, they can affect the location of the PSE and thus, other measures of temporal precision (LD) or acuity (WF).

There are other issues in respect to timing precision, involving the DL and WF (Penney & Cheng, 2018). It was found, bizarrely, that the more difficult the L/S ratios are, the greater temporal sensitivity is (Church & Deluty, 1977; Penney et al., 2008) however, when the ratios are extremely difficult, this impacts sensitivity (Penney et al., 2008); which implies that care must be taken with the bisection task so as to not introduce unwanted variation of the PSE and DL/WF. With respect to the psychophysical nature of the PSE, this too leads to issues surrounding the PSE. Woodsworth & Schlosberg (1938) state that the PSE might be controlled by the subject's response biases rather than their sensory system, which is a recognised problem in psychophysics though, as Swets et al.(1961) comments, it might be that such a bias should be viewed psychologically, as opposed to psychophysically.

While the Temporal Bisection task remains a relatively simply and popular task for investigating the perception of time (Wearden, Philpot & Win, 1999; Carrol et al., 2008; Reed & Randell, 2014; Penney & Cheng, 2018) its theoretical and methodological issues demand an additional timing task is introduced to determine whether any individual differences that impact timing are truly impacting timing, or whether the theoretical and methodological issues due to the temporal bisection task are the reason. Finally, SET, too, has its problems, therefore, a theoretical-free task is desirable, as well as a task which focuses on memory, which makes the temporal generalisation task an ideal candidate.

2.5.9 Methodological Justification

Given that the temporal bisection task is associated with SET (Wearden et al., 1999) and has been widely used for both research in Schizophrenia (Carroll et a., 2009; Elvevåg et al., 2003) and Schizotypy (Lee et al., 2006; Reed & Randell, 2014) has led me to believe that the temporal bisection task must be included in this thesis. Given that I am interested in how Schizotypy subjects perceive durations, and how precise they are at identifying durations.

This leads me to the conclusion that the percentage of long responses, the PSE and the DL are justified, given that these three measures are typically used throughout the timing literature, with respect to Schizophrenia and Schizotypy.

2.6 Temporal Generalisation

As stated in 2.5, the temporal bisection task is one of the most well used tasks in measuring time perception in people however, there is a lack of literature on the temporal generalisation task, especially in the context of Schizotypy. The temporal generalisation task is a prospective timing task, much like temporal bisection (Klapproth, 2017) however, its methodology is based on the equality of judgements (Bausenhart, Di Luca & Ulrich, 2018, cited in Vtakis et al., 2018); in which the subject would be presented with a single duration (e.g., 400ms) and asked to judge whether subsequently presented durations match the single duration (Yes) or do not match the single duration (No). The task was created by Kalish (1958), who noted that the generalisation gradient was dependent upon the characteristics of the underlying stimuli. The temporal generalisation task was first used in the context of time perception by Church & Gibbon (1982) in animals; and a human analogue would be developed by Wearden (1992). It the context of time perception, it is a discrimination method (Grondin, 2010; Wearden, 2016) and a prospective timing task, in that subjects are usually aware that it is a timing task. It is a popular task in the timing literature (Wearden, 1992; Elvevåg et al., 2002; Carroll et al., 2008) but has not been used, until this thesis, to investigate timing differences in Schizotypy leading me to include it in this thesis.

2.6.1 Procedure – Temporal Generalisation

In the temporal generalisation task, the human is typically trained on a ‘standard duration’ (e.g., a tone or visual stimulus which lasts around 400ms long (Wearden, 1992)). They are typically trained on the standard duration by means of a training phrase (Wearden,

1992). The training stage that I have utilised in the temporal generalisation studies involve the subject being presented with the standard duration and asked to identify the standard duration among other durations. They are given feedback as to whether they have correctly identified the standard duration. Once the training stage has come to an end, the subject is presented with comparison stimuli (e.g., 100 – 700ms linearly spaced in 100ms increments). Their task is to determine whether the comparison matches the standard duration (YES) or does not match the standard duration (NO). In the temporal generalisation task, there is always a correct answer (Wearden, 2016) thus, the subject can be given feedback after each comparison duration though, due to time constraints of experiments – and knowing that longer experiments can affect time perception due to boredom (Wearden, 2016) – this is rarely done (Klapproth, 2017) in the literature. Usually, each comparison duration is presented a total of 5 – 10 times summing to a total of 35 – 70 trials (Wearden, 1992). Much like the temporal bisection task, one can introduce a manipulation in the form of a clicker, (Wearden, Philpott & Win, 1999).

2.6.2. Data Presentation – Temporal Generalisation

In terms of how the data is presented a temporal generalisation gradient is typically presented, which is the proportion of YES responses plotted against the comparison durations (Wearden, 2016, Wearden, Philpot & Win, 1999). Usually, there is a 'peak' at the standard, with both the lowest and highest durations normally recording a near 0% of YES responses (Wearden, 1992) and giving the temporal generalisation curve its characteristic shape. The data can also be plotted by taking the relatively frequency of the YES responses, which can be calculated by dividing the proportion of YES responses by the number of presentations of that comparison stimuli. The measures that can be subjected to statistical analysis are the mean proportion of YES responses at comparison durations (Ogden, Wearden & Montgomery, 2014; Droit-Volet & Clement, 2001; Wearden & Towse, 1994; Wearden,

1992), the location of the peak (Wearden, 2016) and the weighted-mean of the gradient for each subject (Klapproth & Wearden, 2011); as well as the steepness of the curve can be subjected to statistical analysis.

2.6.3 Proportion of YES responses

The data takes form of that in figure 2.4 from Wearden, (1992).

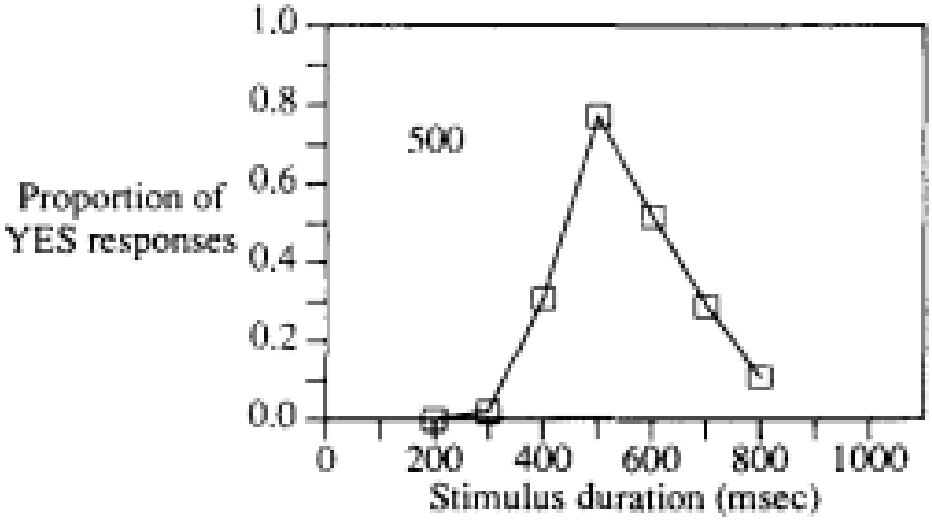

Figure 2.4: The frequency of YES responses to each of the comparison stimuli. In this example from Wearden (1992), the standard duration was 500ms. As can be seen, subjects peak at the standard.

As can be seen, the proportion of YES responses are represented on the ordinate-axis, and the comparison stimuli is represented on the abscissa. The data are then plotted within this coordinate system. In most species, they 'peak' at the duration (Wearden, 1992), with there being symmetrical drops either side of the standard (Wearden, 2016). To analyse these data, a 7 (comparison durations) way Analyse of Variance is often conducted (Elvevåg, 2002) to determine if there was a difference between each duration. In respect to a manipulation (i.e., a clicker), the data can be presented as two temporal generalisation gradients as shown

in figure 2.5 from Voak, Edwards, Percival & Wearden (1996)

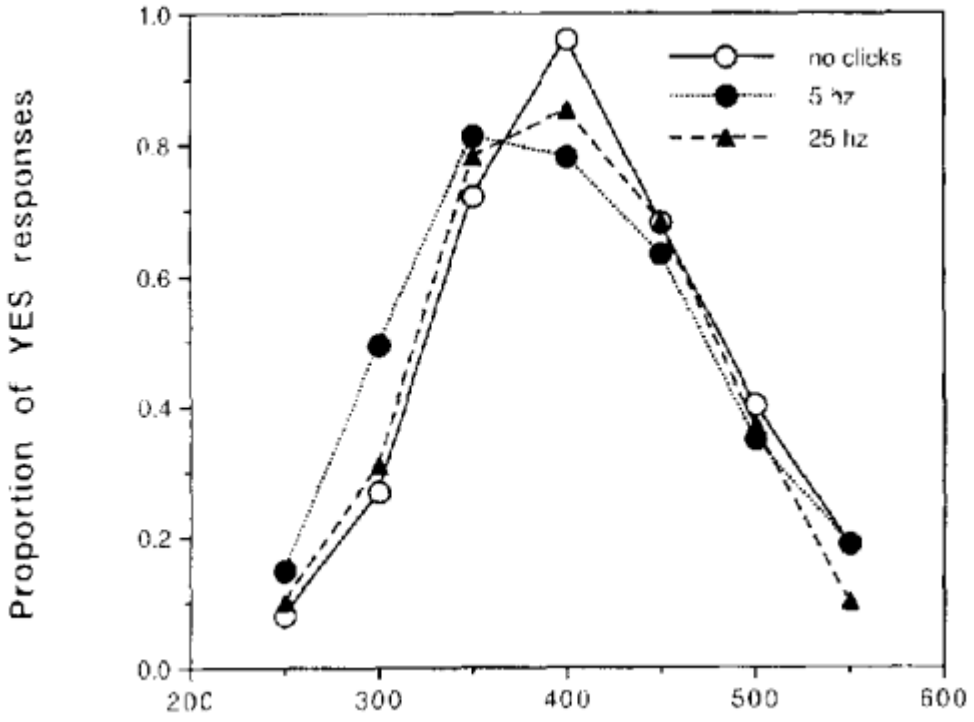

Figure 2.5: As can be seen, there is a baseline (no clicks) and two manipulations (5 and 25hz). Each of the temporal generalisation gradients are plotted from Penton-Voak, Edwards, Percival and Wearden, 1996.

As can be seen, the clicker usually shifts the temporal generalisation gradient to the left, as well as the peak (Penton-Voak, Edwards, Percival & Wearden, 1996). Much like the percent of long responses in the temporal bisection task, the proportion of yes responses reveals information across the whole spectrum of durations. This is one of the most popular methods of analysis within the temporal generalisation task.

2.6.4 Position of peak

It is expected that the subject will most likely peak at the standard (Wearden, 2016), which is the result that is usually found in temporal generalisation tasks (Wearden, 2016,

1992). The peak represents the comparison duration that subjects thought matched the standard duration. It is calculated by the highest proportion of YES responses to a given comparison stimuli. To determine whether the subject 'overpearked' or 'underpeaked', it is simply the case of finding the difference between where subjects peaked and the standard duration by the following equation:

$$\text{Peak} = \text{P}_\text{d} - \text{S}$$

In which P_d = position of peak for subject, S = standard duration (e.g., if a subject's peak position was 300ms and the standard duration was 400, the subject underpeaked by 100ms). By taking the difference, and converting it into a percentage, we have obtained a single value that captures the temporal generalisation task. This allows us to subject this point to additional analysis, such as Multivariate Analysis of Variance (MANOVA) or Discriminant Analysis (DA). The peak difference might be the analogue to the PSE in the temporal bisection tasks. This difference can be used to capture the overall shape of the temporal gradient.

2.6.5 Analysis of Temporal Generalisation

Much like temporal bisection, the temporal generalisation task can be analysed atheoretically or theoretically. In terms of the atheoretical approach, experimenters usually focus on the percentage of YES responses (Wearden, 2016). These percentage of yes responses are subjected to an ANOVA and any differences between a baseline and manipulation condition are claimed to illustrate differences in timing (Wearden, 1992). At each individual duration, one could conduct a paired-samples t-test though, it would be difficult to understand what differences, at an individual duration, mean without a theoretical model, however. Why should a subject show differences between 400ms and 500ms

durations? With respect to both the weighted-mean and the steepness parameters, these can be subjected to other analysis, such as a discriminant analysis, correlational studies, or multivariate analysis and thus, theoretical analysis is possible.

2.6.6 Theoretical Analysis

There are many models which can be applied to temporal generalisation (Wearden, 1994), including MMC, ATT and others (Wearden, 2016). The prominent timing model that can be applied to the temporal generalisation task is SET (Wearden, 2016). In this case, the subject learns the duration, which is assigned n-number of pulses, and which we shall term D_p. This value is transferred to 'reference memory' according to SET, as it is considered a 'duration of interest. Once the subject has completed the training, they are then exposed to the experimental condition and are presented with one of the several comparison durations, C_d. Once again, a number of pulses accumulate for this duration; which is subsequently 'sent' to the working memory component from the accumulator. C_d is compared with D_p, which is held in reference memory, at the decision process, therefore if:

$$C_d = D_p$$

The subject will respond YES and the process repeats (Wearden, 1992) however, if :

$$C_d \neq D_p$$

The subject will respond 'NO' and the process repeats once again (Wearden, 1992; Wearden, 2016).

This gives rise to the temporal gradient which, in an experiment that has employed a clicker manipulation, will lead to a leftward shift relative to a baseline condition (Penton-Voak,

Edwards, Percival &Wearden, 1996). This is implied as meaning the pacemaker has accumulated more pulses due to the clicker manipulation (Wearden, 1994). This could be the analogue to when the temporal bisection psychophysical function shifts to the left hand, on the basis of a quicker pacemaker. In terms of what it means in the context of the temporal generalisation task, the subject would have a greater propensity of saying yes to comparator durations under the standard durations, as the click gets 'integrated' into the comparator durations and therefore the subject would be more likely to say yesto them. Furthermore, the weighted-mean (Klapproth & Wearden, 2011) can be used as a measure, where lower values imply a faster pacemaker.

Regarding the 'peak' duration location, in the context of SET (and indeed, all models which utilise the pacemaker-accumulator models, such as ATT and the information-processing models), the peak would shift left-ward in the presence of a clicker, as, much like the PSE shifting in the context of the temporal bisection task, as a greater number of ticks have accumulated and thus, subjects would claim a comparison duration under the peak would be considered the peak (Wearden, 1994).

The steepness gradient with respect to temporal bisection is the analogue to the difference limen in the temporal bisection task, discussed earlier. The steepness statistic represents subjects' temporal precision, whereby a lower value accords to greater temporal precision. To give an example, presuming the steepness gradient is the analogue of the DL, a lower DL, added to the standard duration (e.g., 400ms + 20ms = 420ms) would mean that a subject would respond 'YES' to comparison durations that are closer to the standard duration. Conversely, if one's steepness gradient was a higher value, which would be added to the standard duration (e.g., 400ms + 100ms = 500ms), the subject would be unlikely to identify the standard duration accurately and consequently, they would be inaccurate. In the context of SET, this would imply that a higher gradient value would accord with the pacemaker

emitting a greater number of pulses (Treisman, 1963) however, where would these 'extra' pulses emanate from? I would argue that the pacemaker emits a given number of pulses, which are regulated by the switch. When the switch detects there are 'too many' pulses, it adjusts its latency to 'trim' the extra pulses, which are lost. Thus, in subjects who have a low gradient, the switch is successfully 'trimming' the number of pulses according to the steepness mean however, those subjects whose steepness gradient has a higher value, the switch has a deficit in that it is not adjusting its latency accordingly. ATT provides a better context for this (Zakay & Block, 1994); as the model pertains to an 'attentional' gate. Thus, in the context of ATT, one could argue that when the subject is paying attention to the comparison duration, the attentional gate adjusts its latency accordingly. Only in those subjects with attentional deficits would the latency deviate from the mean.

2.6.7 Theoretical/Methodological issues – Temporal Generalisation

Much like any task in the domain of psychology, all suffer with theoretical and methodological challenges, and the generalisation task is no exception. As well as the methodological issues that persist with bisection, they are to be found in the generalisation task (Klapproth, 2018) however, there are theoretical issues that are unique to the generalisation task, such as changes to the standard duration. Also, it appears that SET cannot readily explain where the 'extra' pulses arise from when gradient steepness is taken into account. However, as opposed to the temporal bisection task, the temporal generalisation task allows us to rule out any variance arising due to the nature of the bisection task however, as Wearden (2016) notes, no model current exists to model the temporal generalisation task exactly.

The temporal generalisation task is a classic in the timing literature (Grondin, 2010; Wearden, 2016) and has been used for many years, in the context of time perception. Some researchers argue (e.g., Block & Zakay, 1994) that the temporal generalisation task is suited

to studying memory in time perception. Much like temporal bisection, the temporal generalisation task has theoretical and methodological issues however, when the tasks are used in together, they provide an excellent metric for researching all components of time perception.

2.6.8 Methodological justification:

The methodological justification for using the temporal generalisation in this thesis is that it has not been conducted to investigate time perception in Schizotypy. Also, given that the temporal generalisation task is deemed as having a greater sensitivity as opposed to the temporal bisection task (Penney & Cheng, 2018), this allows me to either replicate the findings in the temporal bisection task, or reject them.

2.7 Temporal Estimation:

The temporal estimation task is a simple task to implement, and was one of the first timing tasks to be used to measure time perception (Grondin, 2010). This task differs from both the bisection and generalisation tasks in that it can be either a prospective or a retrospective task (Wearden, 2016). It is a very simple task to both initiate and to subject to statistical analysis. However, it is often considered 'problematic' in that there are few models of estimation and none of them seem satisfactory (Wearden, 2016) however, that does not mean that temporal estimation cannot be modelled theoretically (Ulbrich, Churan & Wittman, 2007). However, in the absence of a model, the constraints of that model do not apply, allowing for an atheoretical approach, nor its usefulness. Given the fact that I want to investigate which components of SET are retarded in Schizotypy, which implies that both the prospective (temporal bisection and generalisation) and retrospective (temporal estimation)

tasks have to be used. Only the temporal estimation task can be used within the retrospective paradigm.

One of the key findings from this task was that subjects tend to estimate shorter intervals as longer, and longer intervals as short (Lejeune &Wearden, 2009; Vierordt, 1868). Furthermore, with respect to the discussion on the retrospective paradigms, the temporal estimation task can utilise either paradigm (Grondin, 2010; Wearden, 2016) which allows researchers to tap into the retrospective nature of the perception of time and potentially, memory resources that contribute to the variance of timing. In terms of which cognitive functions the temporal estimation task depends on, in either paradigm (i.e., prospective of retrospective), the subject would require both attention and reference memory (Lejuene & Wearden, 2009) however, the retrospective task focuses more on memory (Block & Zakay, 1997). For example, since it is presumed that subjects will not be paying to the temporal information of a duration (e.g., how long the duration lasts) in the retrospective task, then it is presumed they draw information from the context of the duration (Klapproth, 2007) meaning that subjects should be less accurate in estimating durations in the retrospective paradigm than the prospective paradigm.

2.7.1 Procedure – Temporal Estimation:

There are many variations on how the temporal estimation task is conducted however, simply put, the subject is presented with a duration (which can be within the auditory, visual or somatic modalities) and they are asked to estimate it (either via written, typed or verbal estimations). The presentation of the duration varies according to whether the task is prospective or retrospective in nature. In the prospective paradigm, the subject would be told they are within a timing task whilst in the retrospective task, the subject is not aware they are in a timing task (Grondin, 2010). Only when they are asked to estimate the duration are they aware it is a timing task (Wearden, 2016). The subject can be asked to estimate the duration

in numerous ways (Wearden, 2016); which can include tapping the duration, in which the subject hears the duration, then estimates it by tapping for the length of the duration on a specialised pad (Vierordt, 1868; Jazayeri & Shalden, 2010), or they can reproduce it verbally (Wearden, 2016).

2.7.2 Data Presentation – Temporal Estimation

In terms of how the data is presented, it is common for the mean estimated interval, the ratio between estimated and standard interval, and the coefficient of variation, which is the standard deviation divided by the mean estimated interval (Ulbrich, Churan, Fink & Wittmann, 2007). The mean estimated interval can be presented in graph form, as shown in figure 2.6.

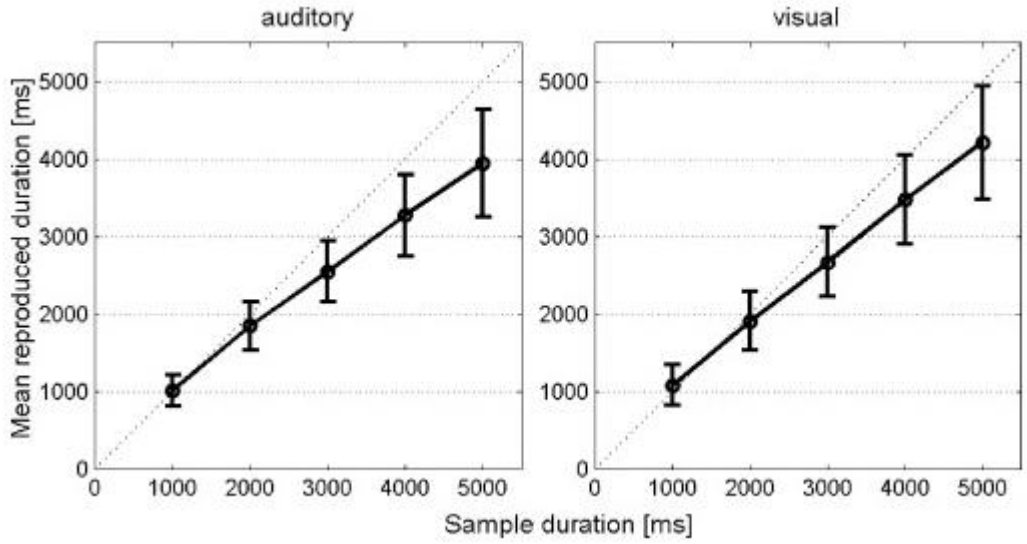

Figure 2.6: In this graph, from Ulbrich, Chuan, Fink & Wittman (2007) we can see that the mean estimated duration is plotted on the ordinate and the standard duration on the abscissa. The horizontal lines indicate accurate estimations.

However, a standard bar graph can be used to represent the data, including the mean estimated interval, the ratio score, and the coefficient of variation (Wearden, 2016). These

data are usually analysed using a one-way ANOVA, with the individual difference of interest used as a between-subject factor; illustrating how simple the temporal estimation task is to model (Wearden, 2016).

2.7.3 The Mean Estimated Interval

The mean estimated interval is the value the subject believes the standard interval was. In retrospective tasks, it is but a single value, whilst in prospective tasks, it is a range of values, depending on how many times the interval was presented to the subject. It is usually presented in graph form, with the estimated time (be it retrospective of prospective) on the ordinate and the sample duration plotted on the abscissa. It can be subjected to an ANOVA. Due to the nature of the retrospective task, it is drawn only from memory (Wearden, 2016) therefore, any variance in the task is mostly contributed to by memory (Block, 1994). In the context of the retrospective task, it would make sense to calculate a mean estimated interval of specific between-subject factors (e.g., low and high schizotypy) which can be used to compare different groups in the data.

2.7.4 Coefficient of Variation

The coefficient of variation is calculated by dividing the standard deviation by the mean. In the context of the retrospective task, the mean and standard deviations of each group can be used in the retrospective context. The coefficient of variation is often used as a measure of variability (Wearden, 2016). The higher the value given by the coefficient of variation, the higher the dispersion (Searls, 1964). A value of 30 or greater is usually indicative of a problem in the data det, or researcher incompetence. The equation used to calculate the coefficient of variation is given by:

$$cv = \frac{X}{\sigma}$$

Where X = mean and õ = standard deviation.

2.7.5 Standard Deviation and Ratio

The standard deviation can also be used as a measure of dispersion; where subjects whose standard deviations are greater than 2 can be considered as having a greater dispersion than those with a lower value. In temporal estimation studies, it is common to use all three measures of analysis to analyse timing (Wearden, 2016). The ratio of durations is also analysed, in which the subject's estimated duration is divided by the actual estimation (Boltz, 2005). A value of 1 indicates that the subject has correctly identified the duration, whilst a value < 1 indicates underestimation and a value > 1 indicates overestimation (Boltz, 2005). The ratio can often be used as a measure of accuracy, and a metric for underestimation and overestimation.

2.7.6 Analysis of Temporal Estimation, theoretical analysis, and issues:

There are relatively few models of temporal estimation though as Wearden (2016) notes, none of them offer a satisfactory paradigm. The models that do exist (such as for Vierordt's law) do not hold (Lejeune & Wearden, 2009); including the more complex dual clepsydra model (Wackerman and Ehm, 2006). However, this does not mean that models, such as SET, cannot be applied to temporal estimation. To apply SET to temporal estimation in a retrospective context, it could be argued that the pacemaker is 'ticking' away in the background. Thus, when the subject perceives the presentation of the stimulus (e.g., a 1500ms kitten video) the pacemaker accumulates the pulses for this stimuli and stores them in working memory. When the subject is then asked to recall how long the duration lasted for,

they can recall it from reference memory however, as Wearden, (2016) notes, no theoretical model accounts for temporal estimation hence why it might be wise to apply an atheoretical approach to temporal estimation. As implied throughout the discussion of temporal estimation, it is patently difficult to model theoretically (Wearden & McShane, 1988; Wearden, 1991; Wearden, 2016). Without a theoretical framework, it can be difficult to interpret the results that ensue. However, the estimation task is relatively simple to implement and allows an atheoretical analysis (Wearden, 2016). The methodological issues that arises from the temporal estimation task is that the task is limited to suprasecond durations (Wearden, 1991) due to subjects not being able to reproduce subsecond durations adequately (Wearden, 2016). As a consequence of this restriction, it is argued that temporal estimation is limited to analysing higher cognitive functions, such as memory, cognition and attention, as opposed to nuanced timing functions (Grondin, 2010) such as speech and motor processing. Thus, the estimation task is limited to suprasecond durations only (Wearden, 2016). The temporal estimation task is unique in that it cannot be adequately modelled (Wearden, 2016) though of course, SET and others can be applied to it. The inclusion of temporal estimation in the thesis is to explore the higher cognitive functions, such as reference memory.

2.7.8 Methodological Justification:

The temporal estimation task has not been used before in either Schizophrenic or Schizotypy populations. Given the fact that I want to investigate the full suite of components of SET, to determine which component is likely retarded in Schizotypy, I have elected to use the temporal estimation task to investigate the reference memory component of SET. Since

the most appropriate way to measure reference memory's contribution to SET is via the retrospective paradigm, I chose to include the temporal estimation task.

2.8 Overall Summary

The choice of paradigm (e.g., prospective or retrospective) and critical timing (subsecond and suprasecond durations) determine which task is best suited to the researcher's aim, especially in the case of this thesis's key aims. Critical timing, such as subsecond and suprasecond durations have been discussed to demonstrate to the reader why we have chosen the subsecond durations only in our thesis. The most popular tasks, including temporal bisection and temporal generalisation can be used theoretically and atheoretically. Since we are using *SET* in this thesis, tasks that can be specifically modelled using this theoretical model have been used. Furthermore, each of the tasks measures a different element of timing. For example, the temporal bisection task can be used to measure attention, and working memory (Wearden, 2016); whilst the temporal generalisation task can be used to measure memory and sensitivity. In conclusion, this chapter has explored the key factors that are directly relatable to the thesis.

To conclude, I have included the temporal bisection, temporal generalisation and temporal estimation tasks within this thesis. I have chosen the temporal bisection task, given its widespread use in Schizophrenia and Schizotypy. I have also employed the temporal generalisation task, given that it has a greater sensitivity to timing than the temporal bisection task, allowing me to potentially replicate the findigns that emerge in Chapters 1, and 2 respectively. Finally, given that I want to investigat e the full range of components of SET, I have included a temporal estimation task, that allows me to investigate the retrospective paradigm.

Chapter three: Individual Differences: Schizophrenia and Schizotypy

The idea of how psychological processes are contingent upon individual differences has an illustrious history (Tyler, 1947). Time perception is no exception, with the earliest of investigations of what impact individual differences have on time perception focusing on humans' age (Oakden & Sturt, 1922) however, investigations as early as 1909 have been reported, such as those by Woodrow (1909), and Dunlap (1911). Therefore, it is no exaggeration to state that the differential psychology of time perception has a rich, and varied history (Wearden, 2016).

Differential psychology in time perception has since been expanded into many areas, such as gender, psychopathology, neurodegenerative diseases and neurological aberrations (Weber, 1933; Gilliland, Hofeld & Eckstrand, 1946; Whyman & Moos, 1967; Rammsayer, 1990; Meaux & Chelonis, 2003; Iwamoto & Hosiyama, 2012; Matthews & Meck, 2014; Reed & Randell, 2014) which has culminated in a good understanding in how individual differences might affect time perception. However, one area where time perception remains contentious and mysterious (often contradictory) is schizophrenia (Reed & Randell, 2014) and, as a result, a paucity in how this condition effects the perception of time remains (Reed & Randell, 2014).

Due to the side effects associated with schizophrenia-prescribed neuroleptics, such as the phenothiazines (chlorpromazine) and the butyrophenones (haloperidol), to treat schizophrenia, this further complicates the link between time perception and schizotypy (Reed & Randell, 2014). For example, as Chapter 1 has discussed, the basal ganglia is implicated in the pacemaker-accumulator operator of SET (Grondin, 2010). Since the

positive symptoms of schizophrenia are associated with excessive dopamine release from mesolimbic neurones (Meyer & Quenzer, 2018) and that the mesolimbic pathway (which is a dopaminergic pathway in the brain – Meyer & Quenzer, 2018) connects with the basal ganglia, the administering of dopamine antagonists would lower levels of dopamine in the basal ganglia, which could potentially mediate time perception. For example, Rammsayer (1989) found that the administering of haloperidol decreased performance on timing tasks manifesting as a 'slowing down' in the clock (Rammsayer, 1990), which manifests as an underestimating of durations (Wearden et al., 1999). This could account for some of the contradictory findings in time perception and schizophrenia, such as in some tasks, subjects overestimating durations, and in other tasks, underestimating durations (e.g., Elvevåg et al., 2003; Carroll et al., 2008, 2009) possibly due to the fact that schizophrenics who take part in timing studies could have recently taken dopaminergic medication. To circumvent these confounding variables, schizotypy provides a useful concept to study time perception in schizophrenia (Reed & Randell, 2014).

Prior to my review of the literature concerning time perception and schizotypy, it is necessary to develop the rationale of utilising schizotypy as a metric for schizophrenia. To do this, I shall first discuss the core of this thesis' research, which is Schizotypy, including how schizotypy was developed as a latent form of schizophrenia first by Rado (1953); followed by a discussion on how Schizotypy relates to Schizophrenia; in which Meehl (1962) identified Schizotypy as a sub-clinical personality organisation that leaves the subject at a high degree of liability for developing schizophrenia (Reed & Randell, 2014). Finally, this chapter will conclude with the research aims and methodology of the thesis.

3.1 Schizotypy

Schizotypy, in its original conceptualisation, can be defined as a latent personality organisation reflective of a latent liability for schizophrenia that manifests as Schizotypic psychological and behavioural manifestations, such as thought disorder (Meehl, 1962, 1990; Lenzenweger, 2006) and arises due to an interaction between genetically determined hypokrisia (e.g., Schizotaxia) and environmental factors. Figure 3.1 pictorialises how schizotypy and schizophrenia are related.

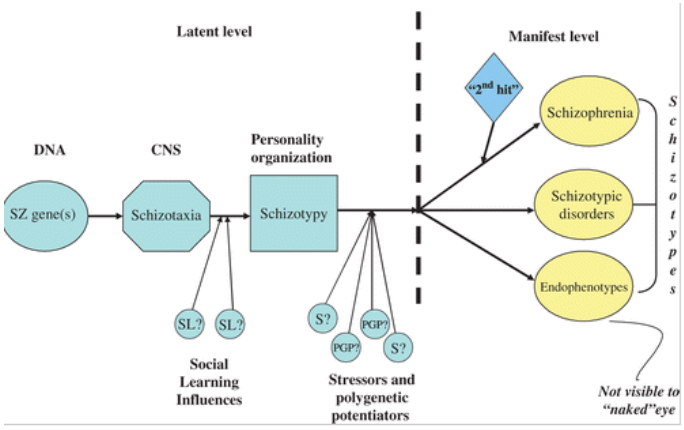

Figure 3.1: A summary of the Lenzenweger (2006) and Meehl (1990) concepts of schizotypy, from Lenzenweger (2006). As can be observed, subjects have a genetically-determined gene termed the 'schizogene'. This gives rise to what Meehl termed hypocrisy, and manifests as a Central Nervous System aberration, termed Schizotaxia. In turn, due to social learning influences, this can manifest as the personality organisation, schizotypy. Polygenetic potentiators and stressors may or may not lead to the subject developing schizophrenia

Schizotypy, as a concept, has allowed researchers to investigate schizophrenia however, as Meehl (1962) has stated, schizotypy cannot be spoken of as entirely genetic but rather, as an interaction between what Meehl (1990) termed Schizotaxia and social learning; as well as polygenic potentiators. Nonetheless, due to schizotypy manifesting in the general

population and being defined as a personality organisation for liability of schizophrenia (Lenzenweger, 2006a) it has provided a useful sub-clinical population to research schizophrenia. With this in mind, can schizotypy be used as a model for schizophrenia? Mheel's (1962) assertion that schizotypy can not be seen as a "*watered down"* version of schizophrenia suggest not, however, according to Lenzenweger (2006), since both share a common gene, and CNS aberration, it is not impossible to suggest that both share similar cognitive deficits, such as timing deficits.

The history of a subclinical form of schizophrenia can be attributed to both Bleuler and Kraepelin, who acknowledged the existence of sub-clinical schizophrenia-like symptoms in relatives of those with schizophrenia; a well as those who had subtle schizophrenic-like disorders (Lenzenweger, 2006). Therefore, the concept of sub-clinical schizophrenia is as old as the concept of schizophrenia itself (Lenzenweger, 2006) however, the term schizotypy was first introduced by Rado (1953) however, it was Mheel's (1962) classic, *Schizotaxia, Schizotypy, Schizophrenia* which properly defined the term schizotypy and provided a sound theoretical basis for the model of schizotypy, first mentioned by Rado (1952). Meehl's conceptualisation of Schizotypy had a profound impact on the way schizophrenia research has unfolded since the early 1960s (Lenzenweger, 2006). Addressing the genetic arguments of schizophrenia, Meehl proposed that an 'integrative neural defect' is the only direct phenotypic consequence produced by a genetic mutation. It was assumed by Meehl that this neural defect was an "aberration in some parameter of a single cell function" (Meehl, 1962) which could manifest in the functioning of other Central Nervous System (CNS) stems, depending upon, Meehl contended, "*the organisation of the mutual feedback controls and upon the stochastic parameters of the reinforcement regime*" (Meehl, 1962). Consequently, Meehl contended that the only direct phenotypic manifestation of schizophrenia (and later, schizotypy) produced by hereditary genetics is the integrative neural defect (Lenzenweger,

2006), which Meehl christened *Schizotaxia (*Meehl, 1962). Meehl explicitly stated that Schizotaxia is "*all that can properly be spoken of as inherited*" (Meehl, 1962).

Schizotaxia was characterised by Meehl (1990) as a ubiquitous functional aberration that is present throughout the entire CNS at the neuronal level (e.g., functional parametric aberration of the synaptic control system). Indeed, Meehl (1990) contended that cognitive disorganisation (Mason, 2005) is caused by the disorganisation at the synapse (Meehl, 1990). Ultimately, this parametric functional control aberration (e.g., aberrations at the neuronal level, specifically, the synapse) was called *hypokrisia* by Meehl (1990), which is characterised by an insufficiency of separation, differentiation or discrimination in neuronal transmission (Meehl, 1962, 1990; Lenzenweger, 2006a, 2006b). In other words, Meehl contends that Schizotaxia is a genetically determined integrative defect, that predisposes to schizophrenia and is a sine qua non to schizophrenia (Meehl, 1962; Lenzenweger, 2006). Meehl suggested that most schizotaxics become schizotypal (Meehl, 1990) and that up to 10% of the general population have Schizotaxia (Lenzenweger & Korfine, 1992; Lenzenweger, 2006). Importantly, as Lenzenweger (2006) points out, Schizotaxia is not an observable polygenetic trait or observable personality pattern. However, Schizotypy was thought to develop by (a)., the subject having a Schizotaxic brain, (b)., the subject having environmentally mediated social learning experiences (Meehl, 1962; 1990).

Mheel's account, as well as others' (Claridge, 1997) would suggest that schizotypy is a subclinical form of schizophrenia however, as Lenzenweger (2006) points out, schizotypy is a personality organisation reflective of latent liability, which Meehl (1990) commented was not a directly observable personality organisation that can manifest itself phenotypically (Lenzenweger, 2006; Meehl, 1990). Indeed, not all schizotypes develop schizophrenia, but, all schizotypes will display evidence of their schizophrenic liability in aberrant

psychobiological or psychological functioning (Lenzenweger, 2006). Furthermore, there is some confusion in the literature as to what exactly 'schizotypy' refers to; as well as whether it is entirely genetic in origin according to Mheel's (1990) original definition, how it manifests, and whether all schizotypes develop schizophrenia.

Some authors contend that there is a highly genetic determinant to schizotypy (e.g., Morton et al., 2016) however, according to Mheel's original definition of schizotypy, only hypokrisia (Meehl, 1990) can be spoken of as being inherited genetically and leads to Schizotaxia. It is the interaction between Schizotaxia and one of many factors, including social learning history (i.e., environmental factors), or other genetic factors that Meehl (1962, 1990) termed polygenic potentiators (i.e., social introversion, anxiety proneness, aggressiveness and anhedonia) that give rise to schizotypy (Meehl, 1962, 1990; Lenzenweger, 2006a, 2006b) which ultimately, is a personality organisation set of traits. However, that is not to say that genetics do not play a part in schizotypy, but rather, that they interact with the factor identified.

In terms of how schizotypy manifests, Meehl (1962) contended that it can manifest as deviance on psychometric or neurocognitive measures and experiments (e.g., Mason et al., 2005; Reed & Randell, 2014), or as clinical schizophrenia (Lenzenweger, 2006). Therefore, an assumption that some make that schizotypy manifests strictly as a reserved-assessed clinical feature is incorrect (Lenzenweger, 2006); as the likes of Reed & Randell, (2014), have shown. A final point that Lenzenweger (2006) makes is that many assume that those who are schizotypal often develop schizophrenia. Meehl (1972) made it clear that not all schizotypals develop schizophrenia (Lenzenweger, 2006); and he further elucidated that schizotypy should not be seen as a lesser form of schizophrenia (Meehl, 1962; Lenzenweger, 2006). That is not to say that the polygenic potentiators and environmental factors that

interact with the established schizotypic personality organisation will not give rise to schizophrenia; as Meehl (1962, 1990) contended in some cases, these interactions can – and do – facilitate the development of schizophrenia (Lenzenweger, 2006b).

Schizotypy, as a concept, has allowed researchers to investigate schizophrenia however, as Meehl (1962) has stated, schizotypy cannot be spoken of as entirely genetic but rather, as an interaction between what Meehl (1990) termed Schizotaxia and social learning; as well as polygenic potentiators. Nonetheless, due to schizotypy manifesting in the general population and being defined as a personality organisation for liability of schizophrenia (Lenzenweger, 2006a) it has provided a useful sub-clinical population to research schizophrenia. With this in mind, can schizotypy be used as a model for schizophrenia? Mheel's (1962) assertion that schizotypy can not be seen as a "*watered down"* version of schizophrenia suggest not, however, according to Lenzenweger (2006), since both share a common gene, and CNS aberration, it is not impossible to suggest that both share similar cognitive deficits, such as timing deficits.

3.1.1 Theoretical Models of Schizotypy

Whilst I have defined schizotypy, I have not shown how it can be used as a model to investigate cognitive and perceptual deficits in schizophrenia. Since I am invested in the Mheelian approach to schizotypy, and the Kraepelin approach to schizophrenia (discussed later), this thesis shall follow the logical reasoning of those approaches and make the assumptions that (I)., only Schizotaxia can be discussed in the context of a genetic influence on schizotypy; (II)., that schizotypy is not an analogue of schizophrenia, but rather, a personality organisation which increases the chances that the schizotypy subject will develop schizophrenia; (III)., that schizotypy manifests either on deviant behaviour on laboratory measures or psychometric measures (e.g., schizotypy questionnaires); and (IV)., that

schizotypy subjects' performance on timing tasks implies schizophrenic patients would perform similarly; sharing the cognitive deficit that arises on aberrations in such tasks. These foundational assumptions will guide the thesis's justification for using schizotypy as a model for schizophrenia however, it would be prudent to review the evidence that suggests schizotypy and schizophrenia share a similar aetiology. There are three fundamental theoretical positions of schizotypy, which include the *Quasi-Dimensional*¸ the *Totally-Dimensional* and the *Fully-Dimensional*. This thesis shall discuss the *fully-dimensional* position, as that is the position that the thesis has taken, due to it being the most widely accepted theoretical position of schizotypy (Claridge, 1997). At its starting point, the *fully dimensional* model of schizotypy, which Claridge (1997) endorsed, assumes normal variation in personality, of the schizotypal spectrum. Claridge (1997) contends that schizotypy denotes a range of enduring personality traits, that manifest in cognitive style (e.g., cognitive disorganisation) and perceptual experiences. These, according to Claridge (1997) arise from a combination of polygenetic and environmental factors, which bear striking similarity to Mheel's (1962) ideation of how schizotypy decomposes into schizophrenia and, as Green & Boyle (2004) observe, draws parallels between psychiatric illness and systemic diseases of the body. Indeed, Claridge (1985) contends that mental diseases arise from a breakdown in the otherwise normal functioning of a biological system, rather than affliction that has been imposed on the body (Green & Boyle, 2004). Claridge (1997) also contends that there are a number of factors that could give rise to psychopathology, including genetic, environmental, and psychosocial (Green & Boyle, 2004). In summary, Claridge (1997) argued that genetically influenced variations in CNS organisation should be construed as "dispositions to varying forms of mental disorder; and the emergence of such disorder is, in essence, a transformation of these biological dispositions into signs of illness" (Claridge, 1985). It is only at the extremes, Claridge adds, that the disease entities of psychiatry are differentiable.

Since the *fully dimensional* model presumes schizotypy is a naturally occurring result in biological variability, the model can account for the high prevalence of anomalous experiences (Verdoux et al., 1998; Nelson et al., 2013).

At first glance, it might be argued that the *totally dimensional* and *fully dimensional* theoretical models of schizotypy are not too dissimilar however, a key difference between the *totally dimensional* and *fully dimensional* models is that Claridge's (1997) *fully dimensional* model distinguishes between the boundaries of health and illness (Green & Boyle, 2004); whereby signs of discontinuity of function, either via a psychometric sign, or experimental measures, are used to denote disorder. Claridge (1997), in keeping with Meehl (1962) contends that many variables might lead to the decomposition of schizotypy to schizophrenia. Conversely, the *totally dimensional* model assumes psychopathology is on the extreme end of a personality spectrum, and that disease is not the cause of the condition. Therefore, it can be argued that the *fully dimensional* model of schizotypy encapsulates both the *quasi-dimensional* view (e.g., schizotypy decomposes into schizophrenia due to a disintegration of functioning that is deemed abnormal) and the *totally dimensional* view (e.g., that the continuity of schizotypal behaviours and experiences should be perceived as inherently normal in personality variation). In other words, schizotypal behaviours are considered normal within the sphere of personality variation (Eysenck, 1960, 1973, 1977) which represents a liability to develop a disorder on the schizophrenic spectrum (Meehl, 1962; Lenzenweger, 2006); and that the decompensation to schizophrenia must involve a disintegration of functioning that leads to the abnormal domain (Green & Boyle, 2004).

3.1.2 Schizotypy as a model for Schizophrenia

Considering the discussion on the theoretical models of schizotypy, this thesis will accept the *fully dimensional* theoretical model of schizotypy for two reasons: (1)., it assumes

that schizotype subjects have a greater liability in developing schizophrenia (consistent with the *quasi-dimensional* model) and (2)., schizotypy manifestations (e.g., deviance on laboratory measures or perceptual experiences) are regarded as normal personality variation (Green & Boyle, 2004). By assuming this position, this allows a justification, to be built, for using schizotypy as a model for schizophrenia by accepting the tenet of the *quasi-dimensional* model that schizotypy increases liability to decompensating into schizophrenia, due to the 'schizogene' (Meehl, 1962, 1990; Lenzenweger, 2006a); as well as the tenet of the *totally dimensional* model, which assumes that the manifestation of schizotypy is a normal expression of personality variability, that is found within the general population (Claridge, 1985; Green & Boyle, 2004). Furthermore, Nelson et al., (2013) argued that the *fully dimensional* model of schizotypy accords with a large body of evidence in genetics, neurobiological, neurobiological, sociological, and environment factors. Finally, Nelson et al. (2013) comment on how the *fully dimensional* model accounts for a large cohort of society reporting anomalous experiences; something of which Meehl (1990) stated was only in 10% of the general population. However, what justification is there for accepting that schizotypy provides an adequate model for schizophrenia?

As discussed, schizotypy (in the Mheelian tradition) is a trait that is amenable to psychometrics and reflects schizophrenia liability (Grant, Green & Mason, 2018; Nenadic et al., 2022) and lends itself as a candidate risk phenotype for schizophrenia (Nenadic et al., 2022). Evidence suggests that non-clinical subjects, with high schizotypy, exhibit deficits in attention, working memory, and general executive functions (Nenadic et al., 2022; Matheson & Langdon, 2008). For example, in a study by Barrantes-Vidal, Chu, Myin-Germeys & Kwapil (2013), who employed the experience-sampling methodology (ESM), which assesses positive and negative schizotypy symptomology in daily life; it was found that positive schizotypy was associated with paranoid symptoms and psychotic-like symptoms, which is

similar in clinically diagnosed schizophrenics. Indeed, the Barrantes-Vidal et al. (2013) study suggests that psychometric schizotypy predicts psychotic-like symptoms in daily life, which are found in schizophrenic subjects. The study showed how schizotypy and schizophrenia share a multidimensional (e.g., positive and negative symptomology) element with schizophrenia. Further work by Cochrane, Petch & Pickering (2012) studied whether cognitive task performance in schizotypal and schizophrenic subjects corresponded when utilising a verbal fluency task and negative priming task giving to both schizotypals (study 1) and schizophrenics (study 2). They found that high levels of schizotypy predicted reduced verbal fluency, as found in schizophrenic subjects (Liddle & Morris, 1991; Liddle, Friston, Frith, Hirsch, Jones & Frackowaik, 1992), as well as in negative priming for schizotypals and schizophrenics; further demonstrating the link between schizotypy and schizophrenia; in that they share similar cognitive deficits. Matheson & Langdon (2008) investigated the impact that schizotypal traits had on executive working memory, by utilising the Letter-Number Sequencing Task (LNT) and found that high schizotypals (irrespective of dimension) reported low LNS scores, which accords with an abundance of research, demonstrating that LNT scores are lower in clinical schizophrenic groups than in non-schizophrenics (Twamley, Palmer, Jeste, Taylor & Heaton, 2006; Manglam, Ram, Praharah & Sarkhel, 2010) further illustrating the link between schizotypy and schizophrenia and suggesting a common aetiology between both. In a wide-ranging meta-analysis of cognitive deficits and schizotypy, Steffens, Meyhofer, Fassbender, Ettinger & Kambeitz (2018) focused on cognitive control, of which they focused on an influential model of cognitive control by Miyake, Friedman, Emerson, Witzki & Howerter (1999), suggested that 3 overlapping yet separable dimensions are involved in cognitive control, including *shifting* (i.e., switching attention between tasks), *updating* (i.e., flexible updating and monitoring of information in working memory) and *inhibition* (ability to withhold a undesirable response that is dominant). In their meta-analysis,

Steffens et al. (2018) concluded that both negative and positive schizotypy were associated with poorer performance on switching attention (i.e., *shifting*) though, they add the proviso that the effect sizes for deficits in *shifting* for schizotypy was $g = 0.32$., whilst for schizophrenics, the effect sizes ranges from $g = 0.86$ to $g = .099$., suggesting that whilst the effect of cognitive decline is weaker in schizotypy than schizophrenia, there is still an effect there. Finally, there is evidence, in the form of a longitudinal study from Barrantes-Vidal et al. (2018) that the multidimensional mode of schizotypy provides a valid framework for studying both the aetiology mechanisms and trajectories of the positive symptomology o schizophrenia. Barrantes-Vidal et al.'s main finding was that that positive schizotypy predicated psychotic-like symptoms. However, the similarities between schizotypy and schizophrenia are not just behaviourally related. There are some biological similarities between the two conditions.

Evidence suggests that both schizophrenics, and those individuals at clinical high risk of developing psychosis have reported an increase in presynaptic dopamine capacity at the striatum (e.g., Howes et al., 2012) which, as discussed in chapter 1, accords with the dopaminergic hypothesis of the pacemaker (Wearden, 2016). Much like schizophrenic patients, it was found that high scoring schizotypal subjects exhibit striatal hypoconnectivity (Waltmann et al., 2019). The evidence suggests that schizotypy and schizophrenia share similar cognitive and perceptual deficits, as well as similar structural variations. In conclusion -- and in keeping with Meehl (1962) -- schizotypy can be used as a phenotypic marker of psychosis liability (Nenadic et al., 2020) for schizophrenia (Lenzenweger, 2006a). This thesis is going to operationalise the concept 'schizotypy' as a personality organisation that is a phenotypic marker of schizophrenic liability. Finally, whilst Meehl (1990) and Lenzenweger (2006a) both argued that schizotypy should never be seen as a 'watered down' schizophrenia; many researchers agree that schizophrenia is the most extreme manifestation of schizotypy

(Barrantes-Vidal et al., 2013). The cognitive deficits associated with schizotypy are numerous, however, similarly to schizophrenia, time perception deficits in schizotypy are also reported. This justifies the thesis using schizotypy as subclinical manifestation of the positive symptomology of schizophrenia (Reed & Randell, 2014).

3.1.3 Timing deficits in schizotypy

There are very few studies that have investigated timing deficits in schizotypy. Lee, Dixon, Spence & Woodruff (2006) employed a temporal bisection task, with two conditions: a 400/800ms condition, and a 1000/2000ms condition. They hypothesised that subjects who scored highly on schizotypy would show and underestimation in the bisection task. They further hypothesised that subjects with cognitive disorganisation would have a leftward bisection point; whilst those with negative schizotypy would have a rightward bisection point meaning they overestimate durations, in line with Elvevåg et al. (2003) and positive schizophrenia, and negative schizotypals would underestimate durations. They found that High schizotypal subjects had a rightward bisection point (e.g., underestimated durations) and a wider DL, similar to Elvevåg et al. (2003).

They explained their results as an attentional error however, it should be noted that Lee et al. (2003) appeared to use a questionnaire that was better suited to schizotypal personality disorder, as opposed to schizotypy per se. As Lenzenweger (2006a) stated, schizotypy and schizotypal personality disorder are not the same thing. Therefore, the results of Lee et al. (2003) could reflect timing deficits in schizotypal personality disorder, as opposed to schizotypy. Nonetheless, their findings (e.g., rightward shift in bisection point and flatter DL) with the findings of Elvevåg et al. (2003); suggesting that timing deficits in schizotypy map onto similar timing deficits in schizophrenia. However, their finding of a rightward shift in psychophysical function is confined only to the 1000/2000ms duration, as

opposed to the 400/800ms duration. Lee et al. (2006) claim this is due to a generalised cognitive deficit in schizophrenia however, Grondin (2010) argues that the suprasecond duration does not measure time perception, per se, but higher cognitive functions. Therefore, it would be expected, given schizotypal/schizophrenics deficits in cognitive functionality (Reed & Randell, 2014), that there would be deficits reported for suprasecond durations. Subsecond durations are said to better reflect time perception. Furthermore, there was a lack of robust theoretical application in Lee et al. (2006), such as SET, which would be required to determine whether timing deficits are the result of memory, or attentional deficits.

Reed & Randell's (2014) research was a vast improvement on Lee et a., (2006), as they utilised a more robust questionnaire termed the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) and adopted SET as a theoretical framework. Again, they used the temporal bisection task, (200/800ms durations). They found that those who scored highly on the unusual experiences O-LIFE category underestimated durations, as measured by the PSE. Reed and Randell (2014) make the point that why those, who scored highly on the unusual experiences scale should underestimate durations, in the context of SET, requires further exploration, which is the purpose of this thesis however, prior to investigating schizotypy in the context of SET, the measures of schizotypy must be discussed.

3.1.4 Measures for Schizotypy: Oxford-Liverpool Inventory of Feeling and Experiences

In terms of measuring Schizotypy, there are a plethora of questionnaires in use (e.g., for reviews, see Chapman et al., 1995, cited in Raine, Lencz & Mednick, 2007), however, the Oxford-Liverpool Inventory of Feeling and Experiences (O-LIFE) is one of the most popular (Reed & Randell, 2014; Mason & Claridge, 2006) and is in alignment with the *fully dimensional* model of schizotypy, which is the theoretical framework this thesis is working with. Mason, Claridge & Jackson (1995) suggested that alternative scales, such as the

Perceptual Aberration Scale (PAS; Chapman, Edell & Chapman, 1980) and the Launay Slade Hallucination Scale (Mason et al., 1995) were highly skewed, in nature and attempted to identify schizotypy as a unique 'taxon' distinct from low scorers which, Lenzenweger & Moldin (1990) estimated to be approximately 90% of the population. Conversely, Eysenck's P Scale had been criticised for its lack of validity to psychotic illnesses (Claridge, 1983; cited in Mason, 1995) and, as Mason et al. (1995) add, several analytical studies have shown how the P Scale fails to load onto the most prominent and unique psychotic factors. The O-LIFE scale was derived, in part, due to schizotypy reducing to three components which corresponded well to the three-factor model of schizophrenia (Vollema & Hoijtink, 2000), which are positive schizotypy, negative schizotypy and cognitive disorganisation (Mason & Claridge, 2005).

The O-LIFE scale consists of four scales: Unusual Experiences (UE), Cognitive Disorganisation (CD), Introvertive Anhedonia (IA) and Impulsive Nonconformity (IN). In terms of what each of these scales mean, UE accords to perceptual, hallucinatory, and magical thinking items, and is deemed as consistent with the positive symptomology of schizotypy (Mason et al., 1995; Reed & Randell, 2014). CD accords with difficulties in attention, concentration, and decision-making together with a sense of purposelessness, moodiness, and social anxiety. IA describes a lack of deriving pleasure from social sources and other activities and indicates a dislike of emotional and physical intimacy; whilst subjects scoring high on this scale are likely to favour independence and solitude. Finally, IN accords to the disinhibited and impulse-ridden characteristics of psychosis, and contains reference to violent, self-abusive and reckless behaviours (Mason et al., 1995). To derive and formulate these scales, Mason et al. (1995) computed a covariance matrix by taking the scores from 1095 subjects and applied the standard psychometric approach using factor analysis (see Kline, 1993 for details). Mason et al. (1995) tested internal reliability and validity of the scale

with a fresh sample. Mason et al. reported that all scales had alpha coefficients of over .70. Such internal consistency has been replicated (e.g., Rawlings & Freeman, 1997). Mason et al.'s O-LIFE questionnaire further discovered that males scored more highly on IA and IN; whilst females scored more highly on the positive traits (e.g., UE and CD) which reflects findings in the schizophrenic literature (Claridge & Hewitt, 1987). O-LIFE's construction also took into account the *totally dimensional* model of schizotypy. Therefore, O-LIFE can be used as a questionnaire to measure Schizotypy, as it incorporates the *totally dimensional* model of schizotypy, and maps well to schizophrenia (Reed & Randell, 2014). However, it might justificably be asked how Schizotypy is related to Schizophrenia which is what the next section shall discuss.

3.2 Understanding of schizophrenia

Schizophrenia is defined as a severe long-term mental health condition that manifests as a range of concomitant psychological symptoms (NHS, 2022). These symptoms include visual, auditory, tactile, and olfactory hallucinations (Mueser, Bellack & Brady, 1990), delusions, (Bovet & Parnas, 1993), cognitive disorganisation (Sousa, Sellwood, Griffiths and Bentall, 2019) and anhedonia (Strauss & Gold, 2012). Typically, hallucinations, cognitive disorganisation, and aberrant behaviour are classified as positive schizophrenia (Andreasen & Olsen, 1982); whilst anhedonia is classified as negative schizophrenia (Andreasen & Olsen, 1982). Schizophrenia has often been described as the 'cruellest disease of the Western world' and as a disease "worthy of Dante's Inferno" (Aronson, 1980; cited in Torrey, 1980); which illustrates the negative societal attitudes directed towards this condition. The monetary cost of schizophrenia to the United Kingdom is incalculable however, both the psychological and monetary costs of this disease warrant exploring it to determine its underlying mechanisms remain important.

The existence of schizophrenia, before the nineteenth century, has courted significant controversy (Youssef & Youssef, 1996). The controversy concerns some authors contending that schizophrenia was relatively rare prior to the nineteenth century (Aronson, 1980); whilst others contend the schizophrenias are as old as mankind itself (Adityanjee et al., 1999). Cooper & Sartorius (1977) argue that schizophrenia was prevalent before the nineteenth century however, the advent of the industrial revolution led to better healthcare and larger communities, resulting in schizophrenics no longer being absorbed into the community (Hare, 1988) and, as a result, the condition became more visible. However despite not having an anthropological account of the ontology of schizophrenia, Kraepelin and Bleuler further developed the meaning of schizophrenia.

3.2.1 Emil Kraepelin and the semblance of order

Emil Kraepelin's work underpinned the fundamental understanding of psychosis, namely, schizophrenia (Adityanjee et al., 1999) and introduced a semblance of order to insanity that, Kraepelin hoped, would lead to both the aetiology and effective therapy for, what he termed, dementia praecox (Odegard, 1966). Uniquely, Kraepelin attempted to emphasise both the aetiology and outcome of schizophrenia (Odegard, 1967); as well as opining on the biological and social tenets that could give rise to schizophrenia (Williams & Wilkins, 1921). Kraepelin also proposed the episodic nature that characterises schizophrenia (Adityanjee et al., 1999); as well as differentiating schizophrenia into three classic subtypes, including hebephrenic, catatonic (in line of his contemporary, Khalbaum, (Kendler & Engstrom, 2017) and paranoid (Kraepelin, 1904; Kendler, 1986; Adityanjee et al., 1999). Kraepelin elucidated a strictly psychiatric approach to schizophrenia, as opposed to a psychodynamic approach to the illness (Odegard, 1966; Hoenig, 1983) which some argue does not appreciate the effects on psychological processes of the condition. It is also noted, by Gruhle (1932) that Kraepelin had introduced this disease of dementia praecox without

providing an unequivocal symptomology of it (Gruhle, 1932; cited in Hoeing, 1983). Obviously, dementia praecox, as defined by Kraepelin needed further work as a concept, which Eugen Bleuler would contribute.

3.2.2 Eugen Bleuler and the organising of schizophrenia

Eugen Bleuler was a Swiss psychiatrist, whose famed book *Dementia Praecox or the Group of Schizophrenias* was published in 1911 (Adityanjee et al., 1999) as a direct critique to Kraepelin's *Dementia Praecox*. Bleuler's main point of contention was that the term, *dementia praecox* referred to the disease, as opposed to the diseased (Bleuler, 1911, 1923; Adityanjee et al., 1999). The second point was, according to Bleuler, dementia praecox patients could be influenced, behaviourally though, similar to Kraepelin, Bleuler did not see cure as an option (Odegard, 1966) but rather, treatment. Bleuler's book was considered seminal in that Bleuler considered the psychological aspects of schizophrenia (Hoeing, 1983), in conjunction with work done with Swiss psychiatrist and founder of analytical psychology, Carl Jung (Bleuler & Jung, 1908; cited in Adityanjee et al., 1999). These psychological aspects of schizophrenia included disturbed thoughts and associated splitting (Adityanjee et al., 1999). Bleuler considered the schizophrenias to be split (*schism*) within the mind (*phrenos*) and subsequently, Bleuler would propose the term 'schizophrenia' to refer to this mental illness that manifests, according to Bleuler (1911), as primary features in (i)., *loosening of associations* (ii)., *affective flattening*, (iii)., *autism*, and (iv)., *ambivalence* (Shean, 1978; Adityanjee et al., 1999) and secondary (or accessory) features, which were psychological reactions to the patient's refusal to adapt to the primary features (Adityanjee et al., 1999) and were defined as hallucinations, delusions, changes in writing and speech, catatonia, and somatisation (Bleuler, 1911). Bleuler, whilst introducing the term 'schizophrenia' and proposing a distinct theory of schizophrenia, in which schizophrenic

patients presented with primary features (e.g., fundamental) that manifested as accessory features (Hoenig, 1983), did not create a new nosology of schizophrenia (Aditayanjee et al., 1999). Bleuler was satisfied with Kraepelin's schizophrenic subtypes, but hypothesised two additional subtypes, including simplex and latent (Diem, 1903; Bleuler, 1911) schizophrenias. Bleuler's major contribution to dementia praecox was naming it with an adjective (schizophrenia) and recognising the heterogeneity of the schizophrenias (e.g., paranoid, simple, latent etcetera) however, his contributions were not without controversy, with one of Bleuler's own student – Wyrsch – expressing Switzerland's wider usage of the concept of 'schizophrenia' to many diseases that were not (Wyrsch, 1920; cited in Aditayanjee et al., 1999); which was also a criticism Kraepelin himself aimed at Bleuler. Therefore, later research attempted to streamline these definitions whilst maintaining the Kraepelinian concept of schizophrenia.

3.2.3 Kurt Schneider and current trends in schizophrenia

The Kraepelinian concepts of schizophrenia have persisted – and were expanded upon – by Kurt Schneider (1939, cited in Adityanjee et al., 1997) however, unlike both the Bleuler and Kraepelin approach, which saw schizophrenia as a psychiatric condition, (Bleuler was appreciative of its psychological tenets) methodological reflection shows that the nosology of psychosis are psychological entities (Hoeing, 1983); in which the schizophrenic should be apperceived as a person with a psychological existence. Schneider (1925, cited in Hoenig, 1983) did more than his predecessors to apply this psychological approach to schizophrenia. Furthermore, Schneider described the longitudinal and cross-sectional dimensions of schizophrenia (Adityanjee et al., 1997) and gave it a narrower definition than Bleuler (Adityanjee et al., 1997). Schneider's inclusion in this thesis is an important one, as Schneider's contributions have had a dramatic influence on the nosology of schizophrenia, which persists to the current day (Adityanjee et al., 1997) however, Bleuler's descriptions of

latent and acute schizophrenia have been removed from the diagnostic criteria of schizophrenia (Adityanjee et al., 1997). Schneider also suggested that schizophrenic symptomology could be divided into first-rank (e.g., certain types of auditory hallucinations, delusions of passivity, cognitive disorganisation and delusional perceptions) or second-rank symptoms (Hoenig, 1980) which laid the foundations for the [often distinct and concomitant] positive and negative symptomology of the present day (Andreasen et al., 1980; Hoenig, 1980) dichotomising of schizophrenic symptomology.

Whilst the idea of distinct schizophrenias has existed since the days of Kraepelin and Bleuler, schizophrenia is typically referred to as phenomenologically heterogeneous however, the symptoms of schizophrenia often manifest as numerous psychological deficits (Andreasen et al., 1994) and create a complex array of symptomology. In an attempt to simplify this complex symptomology, the categories of 'positive' and 'negative' schizophrenia has been adopted and was influenced by Schneider (1938; cited in Hoenig, 1980). It was, however, Crow's (1980) dichotomous symptomology which had a further influence on the 'positive and negative' symptomology. Crow defined Type I schizophrenia as including hallucinations, delusions, and cognitive disorganisation; whilst the Type II symptoms were defined as affective flattening, poverty of speech and loss of drive (Crow, 1980). Critically, Crow hypothesised that patients who exhibited positive symptomology of schizophrenia were associated with good premorbid functioning, typical cognition, and responsiveness to treatment (Adityanjee et al., 1997). Further work also demonstrated the coexistence of positive and negative schizophrenia in the same patient (Adityanjee et al., 1997) giving further strength to the 'positive and negative' symptomology of schizophrenia, which is the current nosological emphasis on schizophrenia. The history regarding schizophrenia, including its origins, is incomplete and requires more work, however, such work is not the pursuit of this thesis.

In the DSM-5 (DSM, 2022), it is implied the positive symptomology of psychotic disorders (including schizophrenia) are delusions, which include persecutory delusions (Bentall, Corcoran, Howards, Blackwood & Kinderman, 2001), grandiose delusions (Knowles, McCarthy-Jones & Rowse, 2011), erotomanic delusions (Greyson & Akhtar, 1977), nihilistic delusions (Radovic, 2017), and somatic delusions (McGilchirst & Cutting, 1995), hallucinations (typically auditory in the schizophrenic but occasionally visual and somatic – Baber, Reniers & Upthegrove (2021)), disorganised thinking, abnormal motor behaviour and catatonia (e.g., childish action, negativism, mutism etcetera). Conversely, the DSM-5 defines the negative symptoms of schizophrenia to include diminished emotional expression (prosody, and lack of hand gestures), avolition (decrease in motivation and self-initiated purposeful activities), anhedonia and asociality (DSM-5, 2022). This positive and negative symptomology of schizophrenia, as defined by Crow (1980) is useful to accept; and will be how this thesis views schizophrenia. The cognitive deficits associated with schizophrenia are vast, and range from attentional (Laurent et al., 1999), memory (Lett et al., 2014), perceptual (Butler, Silversein & Dakin, 2008) and timing (Elvevåg et al., 2003), of these, time perception is argued to be one of the most, in terms of research, neglected of all perceptual deficits to occur in schizophrenia (Bonnot et al., 2011). However, it must be stipulated that the aim of this thesis is to explore schizotypy, as opposed to schizophrenia.

3.2.4 Time Perception and Schizophrenia

As Clausen (1950) noted in his classic manuscript *An Evaluation of Experimental Methods of Time Judgement*, timing deficits are common in patients with psychotic disorders, which demonstrates that timing deficits in schizophrenia have been known for at least 70 years (Elvevåg et al., 2003). In one of the earliest studies of time perception in schizophrenia Lhamon and Goldstone (1956, cited in Weistein, Goldstone & Boardman, 1958), it was found that schizophrenic patients overestimate short durations (e.g., durations of 1s) to a greater

extent than non-schizophrenic subjects. This finding was later confirmed by Weinstein, Goldstone & Boardman (1958), who offered no explanation for why schizophrenics should overestimate durations but rather, suggested it is consistent with passivity and autism accompanying schizophrenia.

A more robust experimental paradigm, similar to current experimental paradigms in human timing (Wearden, 2016) was tested by Webster, Goldstone and Webb (1962). In the study, Webster et al. used an electronic timer, which activated auditory durations with a range of .01 – 9.99s; an auditory oscillator provided a tone of 725MHz; and subjects were asked to make a single judgement of a randomised series of durations (.15, .45, .75, 1.05, 1.35, 1.65, and 1.95) along nine categories (1, very much less than a clock second; 2 much less; 3, less; 4 slightly less; 5 equal; 6 slightly more; 7 more; 8 much more; 9 very much more than a clock second). Much like the popular bisection task, subjects were presented with each of the durations 10 times and the resulting function was similar to the bisection curve, as shown in figure 3.1.

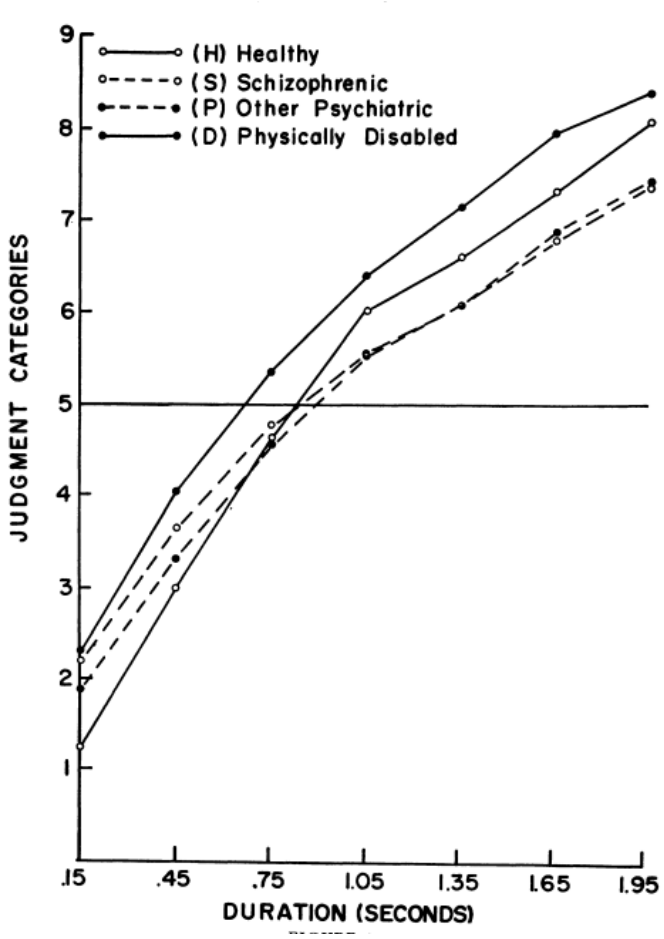

Figure 3.2: The psychophysical function, as reported by – and attributed to – Webster et al. (1962). Note the similarities between this graph and the psychophysical graph produced by the bisection task.

Figure 3.2 shows that schizophrenic subjects underestimated durations; meaning that Webster et al. (1962) did not replicate the previous findings of Weinstein, Goldstone and Boardman (1958), who reported that schizophrenics overestimate short durations. Webster et al. (1962) explained their findings in terms of schizophrenics using less categories than non-schizophrenic subjects, and thus, timing perception might be contextually influenced. Furthermore, in a similar study by Wright, Goldstone, and Broadman (1962), who used both geometric and altimetric intervals, it was also found that schizophrenic subjects' ideation of an interval was equivalent to their concept of that interval, as opposed to non-schizophrenic

subjects. This was interpreted as a general disturbance of time judgement that was not contingent on experimental controls (Wright et al., 1962).

This demonstrates that, even in the earliest of research, time perception studies studying schizophrenia were mixed, with some stating that schizophrenics overestimated durations, and others stating that time perception deficits were tied-up with contextual and experimental paradigms. Baker et al. (unpublished, cited in Orme, 1966) stated, the appraisal and interpretation of both the methodology and data resulting from time perception studies are not straight-froward, and often give unanticipated difficulties; which are apparent in the modern era (Wearden, 2016). The early literature suggests (e.g., Orme, 1966) this was caused by lack of standardisation or even whether a given timing task measures the perception of time (Loehlin, 1959), as well as theoretical reasons, such as critical timing (See chapter 1). Interestingly, Orme (1966) suggested that the psychotic conditions do not manifest as timing deficits as stand-alone conditions, but rather, only when they involve a non-psychotic factor; which could account for why the schizophrenia literature was – and remains (Carroll et al., 2008; Reed & Randell, 2014) contradictory in terms of time perception however, many of these early investigations into time perception and schizophrenia failed to utilise models of time perception.

One of the first investigations that attempted to explain schizophrenic timing was by Calrson and Feinberg (1968), who described how a potential internal clock times an external clock by means of an empirical example. Their interpretation of the data, which resulted from estimation, production, and estimation timing tasks were that schizophrenic subjects overestimate durations, which accords with the previous literature. Interestingly, we can see the establishment of the idea that schizophrenics potentially possess a ‘faster pacemaker’, which is a conclusion that the likes of Carroll et al. (2008) have reached though, it is

important to say that any part of the SET process might be retarded, hence the importance of Carlson & Feinberg's (1968) work. Much of the research in the 1970s concluded that schizophrenics err in the direction of overestimation, which means that they perceive more time has passed than has elapsed in reality (Wahl & Sieg, 1980); or that time appears to be passing more slowly than is expected though, as Wahl & Sieg (1980) contend, because of methodological and terminological confusion, there is a lack of consistency among patterns, with discrepant findings (e.g., Densen, 1977). To address this, Wahl & Sieg defined 'overestimation' as having judged more time had elapsed than had objectively elapsed; and underestimation as having judged less than the actual time had elapsed. Wahl & Sieg found that schizophrenic subjects made considerably more errors in judgements of time passage. They also report, at least for longer intervals, that schizophrenics' accuracy and perception of durations is not significantly different (Wahl & Sieg, 1980), which accords with current thinking that schizophrenic time perception deficits manifest in subsecond durations more strongly than suprasecond durations (Reed & Randell, 2014). Wahl & Sieg make the argument that subsecond and suprasecond durations might be served by different cognitive processes, something which Grondin (2010) discusses, at length, in his review of critical timing. However, does the choice of task mediate duration judgement, when schizophrenia is a between-subjects factor?

Tysk (1983) evaluated the three different methods (e.g., estimation, production and metronome) of measuring short-time durations by using a range of methods. In the study, they had 15 schizophrenic patients, who were asked to estimate the duration of 1s. The methods were adjusting a metronome to their estimate of one beat per second, verbal estimation, and operative estimation (e.g., estimation). Whilst reference to the 'internal clock' was mentioned, Tysk (1983) stated that schizophrenic subjects' overestimation is possibly due to high arousal (Venables & Wing, 1962; cited in Tysk, 1983). The 1990s saw evidence

for a dopamine-dependent internal clock, with research by Thomas Rammsayer (1990). Rammsayer also referred to the differences between the suprasecond and subsecond durations; in which the suprasecond durations (e.g., > 1s) are mainly served by cognitive functions but subsection durations (< 1s) are perceptual, in nature (Michon, 1985; cited in Rammsayer, 1990). Rammsayer (1990) also hinted at neuroleptics accounting for changes in time perception, such as haloperidol, which has long been known to influence time perception (Meck, 1984).

Later research into timing and schizophrenia has also shown that schizophrenics typically overestimate durations however, much of the current research focuses on neuropsychological correlates and biological accounts using more robust methods, such as the temporal bisection and generalisation tasks (see Chapter 2, as well as discussions surrounding temporal accuracy. For example, Elvevåg et al. (2003) conducted both a temporal bisection and temporal generalisation task which are argued to rely on timing processes only. Elvevåg et al. (2003) showed that schizophrenic subjects are more variable in their estimation of durations, and accounted for this variability by memory deficits though, they note, that due to the basal ganglia being associated with the pacemaker (Grondin, 2010); and dopamine levels in the basal ganglia potentially mediating schizophrenia symptomology (Reed & Randell, 2014), medication could impact any results. Carroll et al., (2008) also conducted research on schizophrenics and concluded that auditory time perception appears to be more variable in schizophrenics, again, suggesting memory. Once again, Carroll et al. (2008) emphasises a psychobiological point of view for this deficit. There are many factors involved in timing deficits associated with schizophrenia however, the dopamine hypothesis is one such factor that has received attention.

3.2.5 Schizophrenia and Schizotypy

There is evidence that schizophrenics overestimate durations (e.g., time lasts longer) as opposed to control subjects; and are typically more variable however, since it has been hypothesised that the pacemaker component is potentially served by dopaminergic network of the basal ganglia, which especially effects subsecond durations (Rammsayer, 1993; Meck, 1996; Reed & Randell, 2014), this could give rise to a potential cofounding variable. It has been shown, quite robustly, by Buhusi and Meck, (2002) that the dopamine agonist, methamphetamine, increases the pacemaker speed (manifesting as an overestimating of durations), while the dopaminergic antagonist, haloperidol, decreases the pacemaker speed (manifesting as an underestimation of durations). Therefore, it could be argued that DA agonists lead to an overestimating of durations, while DA antagonists lead to an underestimating of durations. This was further shown by Rammsayer (1989, 1993) in which haloperidol decreased pacemaker speed. Given the fact that schizophrenic patients are often given dopaminergic-altering neuroleptics, and the pacemaker appears to be served by dopaminergic levels, in the basal ganglia, this could account for some of the contradictory findings in the literature. Perhaps the 'robust' finding that schizophrenics overestimate durations are due to haloperidol though, most of the studies surveyed have not investigated this line of thought. Therefore, it would be prudent to find a sub-clinical manifestation of schizophrenia to further research timing in schizophrenia. Schizotypy provides one such solution (Reed & Randell, 2014; Lenzenweger, 2006b) due to the fact that it is free from the contaminatory effects of neuroleptics, cognitive deficits and institutionalisation associated with schizophrenia (Reed & Randell, 2014; Lee et al., 2006; Lenzenweger, 2006b), however, schizotypy must be defined correctly due to some researchers' patent incompetency in

properly operationalising this term. Furthermore, to better understand the cognitive deficits that schizophrenia drives, reliance on investigation that focuses on developed schizophrenia can never give answers on how schizophrenia emerges, and develops (Lenzenweger, 2006b) rather, such studies only contribute to the already large corpus of literature that demonstrates cognitive deficits in schizophrenia, as opposed to how those deficits arise at a fundamental level. The dopaminergic hypothesis is one of many different theories on how schizophrenia arose, with both genetic and sociobiological accounts of the disease. To date, none offer a satisfactory answer to the cause of schizophrenia.

3.3 Research Aims and Methodology

This thesis has two research questions: the first is *why* should schizophrenia have timing deficits at all, and secondly, *why* are the findings of time perception studies mixed, when it comes to schizophrenia? The second of these questions can be answered intuitively: some studies have not measured the effects of medication that schizophrenics are typically prescribed, which introduces a potential confound of medication effects in data resulting from time perception studies. Given this, and the limited research existing on the effects of time perception and Schizotypy, it would be prudent to conduct further research in time perception and Schizotypy in an attempt to address the issue of *why* timing deficits should exist in schizophrenia. Indeed, contemporary views suggest that schizophrenia is a result of over-activity of the neurotransmitter dopamine (Davis et al., 1991; Howes & Kapur, 2009; Meltzer & Stahl, 1976), as discussed at the beginning of this chapter. As a result, pharmacological treatment for patients with schizophrenia is directed at suppressing dopamine release and/or uptake in the form of antipsychotics (e.g., Risperdal, Haloperidol etcetera). While such medication can effectively suppress symptoms of schizophrenia (Johnstone & Crow, 1978), it is not without side-effects (Leucht et al., 1999). Such side-effects present significant barriers

to treatment compliance (Theida et al., 2003). Given these potential negative impacts for patients and their treatment, a research goal is an enhanced understanding of the psychological functions possibly disrupted both by dopamine over-activity and its suppression, which have implications for a range of everyday functioning in patients with schizophrenia, including compliance with treatment regimes. This thesis will provide an enhanced understanding of the psychological functions that moderate time perception, with later work investigating dopamine over-activity.

These considerations have implications for patients with schizophrenia (Body et al., 2009), and may contribute to treatment non-compliance. For example, the schizophrenic subject, who has a slower-running internal clock (e.g., a leftward shift in psychophysical/temporal function) could result in the subject taking their medication before an optimum time. Conversely, the schizophrenic subject who has a faster-running clock (e.g., a rightward shift in psychophysical function) could result in the subject taking medication after the optimum time. Further implications include speech deficits and motor deficits (Grondin, 2010). Thus, alterations in the subject's internal clock, either through onset of psychotic episodes, or through medication, could have serious implications for their behaviour, and ability to comply with treatment regimes. The impacts of procedures to speed the internal clock on those with a tendency to have sporadically slow running clocks, such as those undergoing an acute schizophrenic episode, are unclear. Thus, this area requires further investigation to understand the problems experienced by patients with schizophrenia and represents a clear gap in current knowledge however, this thesis is concerned with how subjects with Schizotypy experience time perception as opposed to Schizophrenia.

However, research into this area requires precise experimental control, which can be problematic when using schizophrenic patients, as discussed in this chapter, especially in an

acute phase of schizophrenia when timing deficits would greatest. Moreover, such research can be confounded by medication, hospitalisation, and symptom severity, specificity, and co-morbidity (as well as posing ethical concerns). A solution is to use a measure of schizotypy in a non-clinical population (e.g., Randell et al., 2009). Schizotypy, as has been discussed, refers to characteristics related to the positive symptomology of schizophrenia, but which are not severe enough to warrant clinical treatment, and this has served as a model for schizophrenia (McCreery & Claridge, 1996; Reed & Randell, 2014), as discussed and justified in this chapter. Factor analyses have shown schizophrenia and schizotypy to be closely related, and the same experimental effects have been found in schizophrenic patients and those scoring high in schizotypy (Dagnall & Parker, 2009; Tsakanikos & Reed, 2005). Moreover, the use of schizotypy as a model for schizophrenia can highlight the influence of specific symptoms areas on task performance (Shrira & Tsakanikos, 2009), and may be useful in identifying the needs of patients and enhancing their future outcomes. By using Schizotypy, it might be possible to identify which cognitive functions give rise to timing deficits in schizophrenia.

3.3.1: Objectives

The current thesis examined the influence of schizotypy on well-established timing procedures to develop a novel model for exploring timing performance in schizophrenia. Differences in timing will be examined between high and low scorers in positive schizotypy, both in general, and in relation to characteristics associated with the specific symptom areas of schizophrenia. Moreover, the well-established behavioural method of auditory Clic Trains is used in the current Experiments to adjust the speed of the internal clock within high and low schizotypy scorers, in order to examine the potential effects of clock speed adjustment in individuals differing in schizotypy levels. This knowledge can help develop human models

that would be extremely useful in advancing understanding in this area concerning schizophrenia and time perception. For example, if the Click Train is shown to modify time perception in Schizotypy subjects, could this be adapted to modify the clock speed in schizophrenia? Finally, since the clicker appears to mediate time perception in similar vain to psychopharmacological agents (Penton-Voak et al., 1996), (though, not with such a strong effect size) this gives further impetus to utilising the Click Train as a method to modify clock speed in schizophrenics.

Subjects completed the computer-based experimental procedures outlined below, and were measured on a range of well-validated psychometric tests. The psychometric factors associated with schizotypy were investigated using the O-LIFE (Mason et al., 1995; reliability = 0.80; which measures unusual experiences, impulsive nonconformity, cognitive disorganisation, and introverted anhedonia). The negative aspect of schizotypy (e.g., introvertive anhedonia) will be excluded from analysis to ensure there are no confounds on task performance on the timing tasks used.. To do this, I shall first conduct a Temporal Bisection task, followed by a Temporal Generalisation task and finally, the Temporal Estimation task.

3.3.2 Temporal Bisection Task

The temporal bisection task investigated percentage of long responses (the extent to which subjects overestimate or underestimate durations) and the Point of Subjective Equality (PSE) measures the subjective point where subjects confuse short durations with long. The Limen Difference (LD) measures variability and precision in durations. The temporal generalisation task examines deviations in time judgements from a mid-range standard, respectively. It is expected that any timing differences will be related only to the positive

subscales of the O-LIFE questionnaire (e.g., UE, CD, and IN) as dopamine over-activity is implicated in brain regions associated both with timing and the emergence of the positive symptoms of schizophrenia measured by positive schizotypy. Furthermore, the inclusion of the Click Train is to further test the pacemaker component in Schizotypy; in that if pacemaker is retarded in Schizotypy, then I would expect the Click Train not to lead to overestimation of durations in Schizotypy subjects.. During training, subjects were presented with a stimulus, randomly presented for one of two lengths of time (200ms and 800ms), with each being presented along with the word "Short" (200ms) or "Long" (200ms) five times each. During the test phase, subjects were presented with the same stimuli for random lengths of time ranging from 200ms to 800ms, at 100ms intervals, and will be required to respond to each stimulus by pressing one of two keys, representing "Short" or "Long", according to how close they feel the presentation time of each stimulus was to the presentation time of the training stimulus. Each duration will be presented ten times each, giving a total of 70 trials. It is predicted that high schizotypy scorers will judge longer periods of time as "short", more often than low schizotypy scorers, thus, producing a larger bisection-point difference above the arithmetic mean. The rationale for this prediction is based on the research of Lee et al. (2006) and Reed and Randell (2014), who both found that subjects who are schizotypy are likely to overestimate durations. There were two experiments: one utilised the visual modality; whilst the other utilised the auditory modality. In both experiments, there was a Click Train condition. To analyse the data, the psychophysical function was constructed for percentage of LONG responses for both baseline and Click Train conditions for both High and Low Schizotypy subjects. Whilst the expectation was that the pacemaker *should* be affected, I did not discount the possibility of other components of SET, such as the attentional switch, or the memory components, moderating timing deficits in Schizotypy, given, as discussed, the wide variety of

symptomology associated with positive schizophrenia. To analyse these data, a repeated-measures ANOVA was first conducted, and interactions were decomposed with simple-effects analysis. Once I had established which components are likely to be retarded in Schizotypy, I attempted to replicate these results in a more sensitive timing task, the temporal generalisation task.

3.3.2 Temporal Generalisation Task:

During training, subjects were presented with a stimulus for a standard length of time (400s), five times, where subjects will have to learn the duration During the experimental stage, subjects were presented with the same stimulus for lengths of time ranging from 100ms to 700ms, in 100ms increments, randomly presented ten times each, and were required to respond to each stimulus by pressing one of two keys, representing "YES" or "NO", according to how close they felt the presentation time of each stimulus is to the presentation time of the training stimulus. It was predicted that High Schizotypy subjects would judge longer periods of time as the "same" as the standard more often than Low Schizotypy subjects, meaning High Schizotypy will be less accurate at identifying the standard. Since no studies have investigated Schizotypy within the temporal generalisation task, this prediction was based on the finding from Elvevåg et al., (2003). Once again, there will be a visual and auditory vision of this experiment, with a Click Train preceding durations in each condition. I also predicted that the Click Train would result in overestimation of durations in Low but not High Schizotypy, given the pacemaker component is implicated in deficits in timing in Schizophrenia. To analyse the data, the temporal gradient was constructed from the percentage of YES responses subjects gave to durations, for Click Train and baseline conditions. The temporal gradients were calculated for both High and Low Schizotypy. Repeated-measure ANOVAs and simple-effects analysis were used to analyse the data. My

justification for using the temporal generalisation task was that it has a greater sensitivity than the temporal bisection task (Elvevåg et al., 2003). Once again, the temporal generalisation task can reveal information about the pacemaker (Wearden, 2016), along with other components of SET, such as reference memory and working memory (Grondin, 2010).

3.3.3 Temporal Estimation task:

To fully analyse the components of SET that might contribute to duration distortion in Schizotypy, a further task was required. That task was the temporal estimation task. In this study, subjects were presented with a kitten video lasting either 15s, 30s, or 45s in either a retrospective (e.g., subjects unaware they are in a timing task) or prospective paradigm (e.g., subjects away that they are in a timing task). Once subjects had watched the video, they were asked to estimate how long the video lasted. This allowed me to further explore the memory components of SET, as, as discussed, the retrospective task appears to isolate the reference memory component of SET. I predicted numerous findings to arise from this study: the first is that paradigm used will be irrelevant (Boltz, 2005), and that Schizotypy subjects should overestimate durations in accordance with the findings by Carroll et al., 2008. Once again, since a temporal estimation task has not be conducted on Schizotypy subjects before, thus predictions were informed by studies on schizophrenic subjects, in other paradigms.

Objectives

It was expected that the results found in temporal bisection could be replicated in the temporal generalisation task and further decomposed in the temporal estimation task. Together, these five Experiments – which had not been conducted, in this manner before, on Schizotypy subjects – should help investigate how timing deficits manifest in Schizotypy. By identifying the potential cognitive process(es) that is (are) retarded in Schizotypy (via the components of SET), this could provide future impetus for researchers to focus on a specific

component of SET, when exploring timing deficits in Schizophrenia. Furthermore, by investigating the effect of the Click Train (also novel) I hoped to demonstrate a potential behavioural intervention that could alleviate timing deficits that arise, due to Schizophrenia, and are said to be a fundamental deficit of Schizophrenia, as discussed in Chapter 3 (Carroll et al., 2008).

To summarise, my predictions for the temporal bisection task is that subjects who score highly on Schizotypy will judge longer periods of time (e.g., 800ms durations) as "short", more often than Low Schizotypy subjects; manifesting as an overestimation of durations. To test this prediction, I first analysed the percentage of long responses, via a within-subjects Analysis of Variance, followed by analysing the PSE to further explore the results obtained by Reed and Randell (2014) and Lee et al., (2006). To examine subjects' precision of durations, I also measured the LD via a repeated-measures ANOVA. Any interactions were decomposed via a Simple-Effects analysis. I also predicted that the Click Train should result in Low Schizotypy overestimating durations (Wearden, et al., 1999) but not High Schizotypy. In terms of the temporal generalisation task, I predicted that High Schizotypy scorers will judge longer periods of time as the "same" as the standard more often than Low Schizotypy scorers meaning High Schizotypy will be less accurate at identifying the standard than Low Schizotypy subjects. To investigate accuracy within Schizotypy, I used a within-subjects ANOVA to determine whether subjects identified longer durations as the standard more often, and a further ANOVA on the accuracy of schizotypy subjects, as indicated by the temporal gradient, with a similar prediction for the Click Train as the temporal bisection tasks. Finally, for the temporal estimation task, I predicted that the paradigm (e.g., prospective or retrospective) will be irrelevant to judgement durations, irrespective of Schizotypy score; and that High Schizotypy subjects should overestimate durations, in accordance with the findings in the schizophrenic literature (e.g., Carroll et al.,

2008). I also predicted that short durations will be overestimated and long durations will be underestimated. In all cases, when interactions arise, they were decomposed using simple-effect analyses.

Chapter four: Effect of a concurrent stimulus on timing depends on level of schizotypy: Evidence from a visual and an auditory temporal bisection task

4.1 Introduction

In this chapter, I present two temporal bisection tasks that have explored timing deficits in Schizotypy, as outlined in Chapter 3. The first experiment is the visual temporal bisection task, in which subjects were trained on two durations (200ms and 800ms), and then asked to judge whether subsequent durations were short or long, in accordance with training. In addition, I used a Click Train to determine whether the arousal-driven pacemaker is retarded in Schizotypy subjects. Experiment 2 was much the same however, as opposed to visual stimuli (i.e., a 4in x 4in black square), I used auditory stimuli. In both cases, I used the O-LIFE questionnaire to measure Schizotypy, as outlined in chapter 3. Given the fact that the temporal bisection task is typically used to model the pacemaker component of SET (Wearden, 2016; see Chapter 3), these two experiments have focused on the pacemaker component of SET though, as I discovered, other components of SET could account for the findings. In terms of what I expected to find, I predicted that High Schizotypy subjects would overestimate durations in both auditory and visual modalities, due to Schizophrenics appearing to have a 'faster' pacemaker in both paradigms (e.g., Carroll et al., 2008). However, these first two experiments were very much exploratory in nature, as many other components of SET have been implicated in driving deficits in schizotypy/schizophrenia time perception, as discussed in Chapter 2 and 3, respectively. I also predicted that the Click Train would lead to overestimation of durations in Low Schizotypy but not High Schizotypy on the basis of Reed and Randell's (2014) paper, in which they hypothesised the pacemaker is

responsible for retarded time perception. I also explored the precision of Schizotypy durations by analysing the LD.

4.2 Experiment 1: Visual Temporal Bisection Task

Experiment 1 used the classic temporal bisection task, in which a Click Train was included to assess the duration judgments of visual stimuli and the performance of the pacemaker component in SET based on previous the work of Penton-Voak et al (1996; Exp.3) and Penton-Voak (2010). Based on the work of Elvevåg et al. (2003) with schizophrenic patients, I predicted that High Schizotypy subjects would overall overestimate durations compared to Low Schizotypals, driven by the pacemaker or other components of SET. I also predicted that the Click Train would result in Low Schizotypy subjects overestimating durations but not High, to further assess the pacemaker component of SET in the context of Schizotypy, given pacemaker deficits have been implicated in Schizotypy (Reed & Randell, 2014). Because depression affects time perception (e.g., Gil & Droit-Volet, 2009), in the current study I excluded the introvertive anhedonia subscale of schizotypy, to ensure that we have isolated only the *positive* symptoms of schizotypy (e.g., Tsakanikos & Claridge, 2005), which map onto the positive symptomology of schizophrenia (e.g., Lenzenweger, 2006).

4.3 Method

4.3.1 Subjects

130 subjects (60 females, 70 males) were recruited via Prolific experiment participation platform. The mean age was 28.0 (SD $\pm$ 9.2; range = 18 – 55) years. The study was approved by the Psychology Ethics Committee. All were naïve to the purpose of the study and were paid £4.50p for their participation. G-Power analysis for a mixed-model analysis of variance (ANOVA) with two groups and two conditions, using a rejection

criterion of $p < .05$, 90% power, and for a medium effect size, suggested a sample size of 108.

4.3.2 Stimuli & Measures

The stimuli used for presenting the durations (in both training and test phrases) was a black rectangle (3.4in x 2.1in). The clicker used was a 144Mhz tone, that lasted for 10ms, with 190ms of silence, over 5000ms, which was perceived as a 5s clicker train.

Oxford Liverpool Inventory of Feelings and Experiences – Brief Version: OLIFE(B) (Mason, Linney, & Claridge, 2005) is a 43-item self-report scale for measuring schizotypy traits. The scale comprises four subscales: Unusual Experiences, Cognitive Disorganisation, Impulsive Nonconformity, and Introvertive Anhedonia. The scale is based upon empirically observed structures of schizotypal traits, has good validity in the general population (e.g., Green et al., 2008; Mason et al., 2005), and has been used extensively in examining the effect of schizotypy on behaviours and cognitions (e.g., Reed & Randell, 2014; Tsakanikos & Reed, 2005). The first three subscales map onto the positive symptoms of schizophrenia, and their sum is used as an index of that trait. The internal reliability (Cronbach α) for the current sample was 0.69. The mean value of the sum of the positive symptom scales for both experiment 1 and 2 was 13.59. To maintain consistency of analysis across the series of studies, those with a score below 14 were classed as lower schizotypal scorers, and those with a score of 14 or greater were classed as higher schizotypal scorers. This was computed by summing the positive sub-scales of the O-LIFE questionnaire and then conducting a mean split of the summation in accordance with Reed and Randell (2014). In this study, 70 subjects were in the low schizotypy group (mean = 8.1 $\pm$ 3.72; range 2 – 13) and 55 subjects in the high schizotypy group (mean = 18.67 $\pm$ 3.74; range 14 – 28).

4.3.3 Design

Experiment 1 was based on a mixed design manipulating Duration with 7 levels: 200, 300, 400, 500, 600, 700 or 800ms, and Clicker Presence with two levels: baseline (no clicker) and clicker presence (clicker). This design produced a total of 14 within-subject conditions, with 10 trials per condition, yielding a total of 140 trials per subject. The between-subjects variable was Positive Schizotypy, calculated based on the scores of the positive symptomatology scales of the OLIFE (B) questionnaire, with two levels: low vs. high. The dependent variables were: the percentage of long responses to each duration (e.g., subjects' percentage of 'Long' for x duration). Also, the PSE was analysed, and its value was the dependent variable; with the independent variable being condition (baseline or clicker). Finally, the DL was analysed in which its value was the dependent variable; and the condition, once again, was the independent variable.

4.3.4 Procedure

Subjects completed the study online using the Gorilla experimental platform (Gorilla.sc). To ensure there were no issues with accurate timing of millisecond stimuli, subjects could complete the experiment only on a laptop or desktop computer. The experiment consisted of two cycles of training and test phases. For the first training phase, each trial started with a blank white screen presented for 1 second. The next screen showed the words Long or Short at the top of the screen, alone for 1 second, followed by a 3.4in x 2.1in black rectangle presented at screen centre, for either 200ms or 800ms. Those were the anchor stimuli. Each duration was presented 5 times, in a random order, each presentation followed by a 1s inter-trial interval.

During the test phase, the same rectangle as before appeared for one of the 7 possible durations – 200, 300, 400, 500, 600, 700, or 800ms – in a random order. Each of these

durations were presented 10 times giving rise to a total of 70 trials. In addition, the words 'Short' and 'Long' was presented at the bottom of the screen, preceding the letters 'z' and 'm', which was reversed for the counterbalanced condition (e.g., Z = LONG, M = SHORT). The task was to indicate whether the rectangle stayed on screen for a 'long' (e.g., 800ms) or a 'short'(e.g., 200ms) time, in accordance with training. Half of the subjects pressed Z for 'long' and M for 'short, while the mapping was reversed for the other half (e.g., Z for 'short' and M for 'long').

The training-test cycle was repeated but this time, a 5s, 144Mhz auditory Click Train was presented prior to each stimulus presentation. This Click Train was created in the Audacity software program and included a 10ms 144Mhz and 190ms of silence, creating a 'click' sound over five seconds (Wearden et al., 1999). I decided not to counterbalance the baseline and clicker condition, but instead to always show the baseline (no Click Train) condition first. That was done to avoid the clicker potentially arousing the subjects' (hypothetical) pacemaker before a baseline condition. Furthermore, restricted randomisation can increase the risk of technical error (e.g., Hewitt & Torgeson, 2006) within a study which, when using an on-line programme, such as Gorilla, is especially pertinent.

4.3.5 Data analysis

The proportion of 'long' responses made with respect to the anchor durations and intermediate durations was calculated for each subject. Trials with response times greater than 2 SDs from the mean response time were removed from both the baseline and Click Train conditions. In the baseline condition, this amounted to 3.24% of the data being removed, and in the Click Train condition, this amounted to 1.40% of the data being removed. This resulted in five subjects being removed from the data (4 males, 1 female), with a mean age of 34.4 (SD $\pm$ 7.3, range 25 – 44). The remaining data was plotted, and yielded a sigmoidal function, where the graph takes the appearance of percentage of 'long' responses

on the ordinate-axis, and stimulus durations on the abscissa-axis. The graph should indicate a near-absence of ‘long’ responses for 200ms, and almost-complete ‘long’ response for 800ms (e.g., Wearden, 2016), indicating subjects had learned the anchors. From the psychophysical function, I analysed the percentage of long responses, and used this graph to calculate the oint of subjective equality, and the difference lime via a custom-coded Python program.

4.4 Results and Discussion

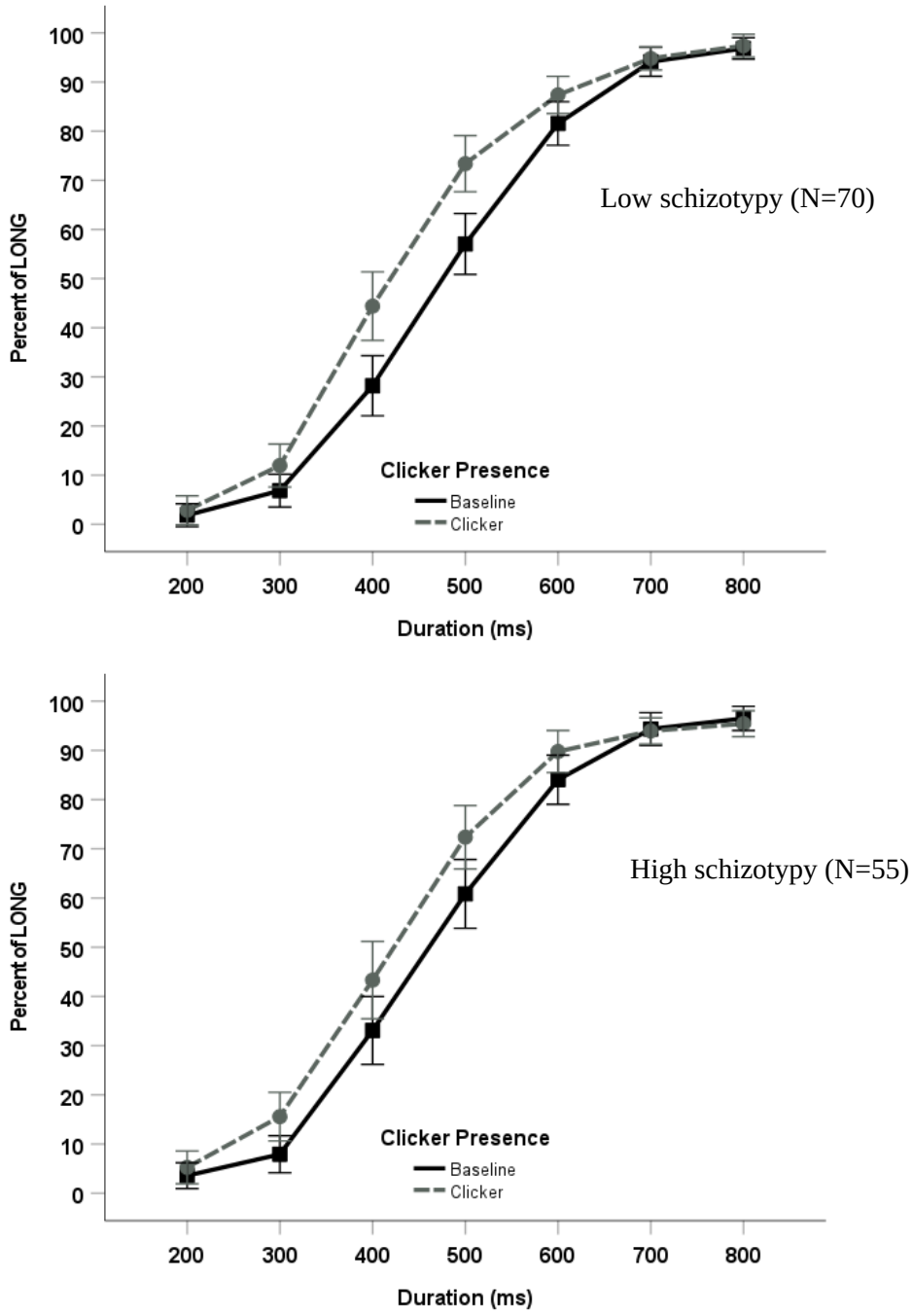

Figure 4.1: Percent of 'long' responses per condition for Low Schizotypy (top panel) and High Schizotypy (bottom panel) subjects in Experiment 1 (visual stimuli).

The mean percent of 'long' responses per condition, for Low (top panel) and High (bottom panel) Schizotypy appears in Figure 4.1. As expected, (e.g., Wearden et al., 1999), the Click Train shifted Low Schizotypy subjects' psychophysical functions to the left (Figure

4.1, top panel), indicating that the Click Train led to an overestimation of durations, in accordance with a visual inspection of Figure 4.1, however, visual inspection of Figure 4.1 (bottom panel) also shows that the Click Train led to High Schizotypy overestimating durations. A 2 (Clicker presence[Baseline, Click Train]) x 7 (Duration [200, 300,400, 500, 600, 700, 800ms]) X 2 (Schizotypy[Low, High) mixed Analysis of Variance (ANOVA), with Schizotypy as the between-subjects factor was carried out on the percentage of 'long' responses. There was a significant main effect of Clicker Presence $F(1,123)=29.182$, $p<.001$, $\eta_p^2=.192$, [.0000, .9094], with a higher proportion of 'long' responses in the Click Train condition compared to baseline, and a significant main-effect of Duration, $F(6, 738) = 1176.458$, $p <.00$, $\eta_p^2 = .905$, [.0000, .9855], with increasing proportion of long responses at durations within the middle of the 200ms and 800ms anchors.. The two main effects were further qualified in a significant Clicker Presence X Duration interaction, $F(6, 738) = 12.257$, $p < .001$, $\eta_p^2 =.091$, [.0000, .4143]. Schizotypy did not yield a significant main effect, $F(1,123)=.572$, $p=.451$, $\eta_p^2=.005$, [.0000, .1644], and was not involved in any significant interactions [Clicker Presence X Schizotypy, $F(1,123)=.478$, $p=.490$, $\eta_p^2=.004$, [.0000, .1238]; Duration X Schizotypy, $F(1,123)=.383$, $p=.890$, $\eta_p^2=.003$, [.0000, .1164; Clicker Presence X Duration X Schizotypy, $F(6,738)=.793$, $p=.575$, $\eta_p^2=.006$, [.0000, .0438].

To decompose the Clicker Presence and Duration interaction and determine which durations the Click Train effectively shifted to the left, a simple-effects analysis was conducted, and is displayed in Table 4.1.

Table 4.1

Simple-Effects analysis examining the Click Presence and Duration interaction

Duration (ms)	Baseline		Click Train		*F(1, 123)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	2.52	9.84	3.92	12.50	1.520	.220	.012	.0000	.0755
300	7.33	14.05	13.56	18.51	13.498	<.000*	.099	.0214	.2051
400	30.36	25.85	43.93	29.32	20.814	<.000*	.145	.0457	.2539

500	58.73	26.15	72.95	24.03	29.417	<.001*	.193	.0820	.3096
600	82.66	18.69	88.44	15.99	9.374	.002*	.073	.0099	.1730
700	94.24	12.43	94.44	9.93	.012	.914	.000	.0000	.0041
800	96.70	9.18	96.54	9.88	.082	.775	.001	.0000.,	.0274

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

Table 4.1 confirms that subjects, independent of Schizotypy level, overestimated durations between 300ms – 600ms, as shown in Figure 4.2 and suggests that the Click Train was effective in shifting the psychophysical function, to the left, between 300ms and 600ms.

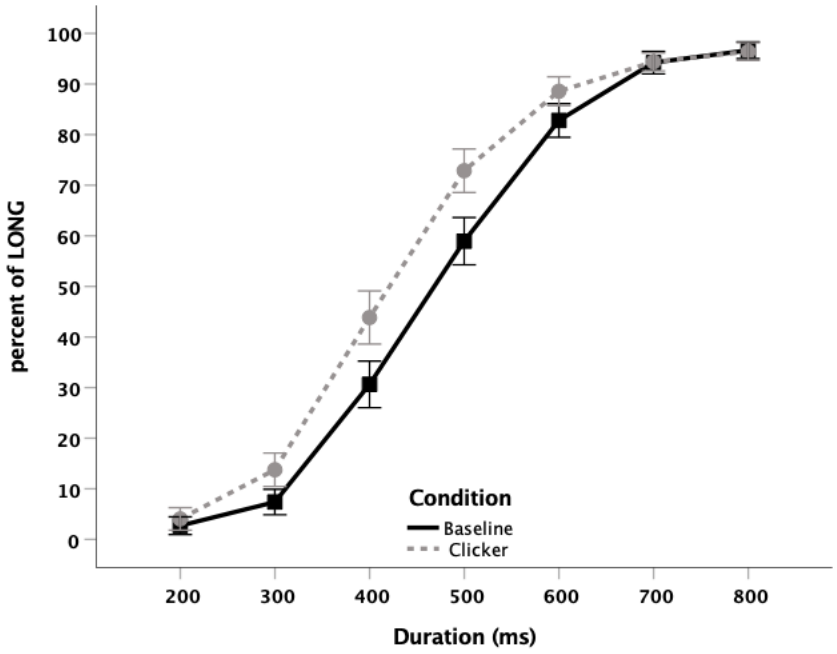

Figure 4.2: The psychophysical function showing the comparison between baseline and clicker conditions. note the significant durations between 300 and 400ms.

The finding that the psychophysical function shifted leftward and showed no interaction with Schizotypy is further quantified by the analysis of the PSE in which a 2[Clicker Presence (Baseline, Click Train)-way ANOVA, with Schizotypy as the between-subjects factor was carried out. There was a significant main effect of Clicker Presence, $F(1, 123) = 16.651$, $p < .001$, $\eta_p^2 = .119$, [.0324, . 2292]. There was no significant interaction between Click Train and Schizotypy, $F(1, 123) = .603$, $p = .439$, $\eta_p^2 = .005$, [.0000., 0564].

Finally, there was no main effect of Schizotypy, $F(1, 123) = .382$, $p = .538$, $\eta_p^2 = .003$, [.0000., .0500]. Figure 4.3 and Table 4.2 confirms that the Click Train led to subjects overestimating durations, irrespective of Schizotypy level, meaning that my second hypothesis, that the Click Train would not be effective in Schizotypy rejected.

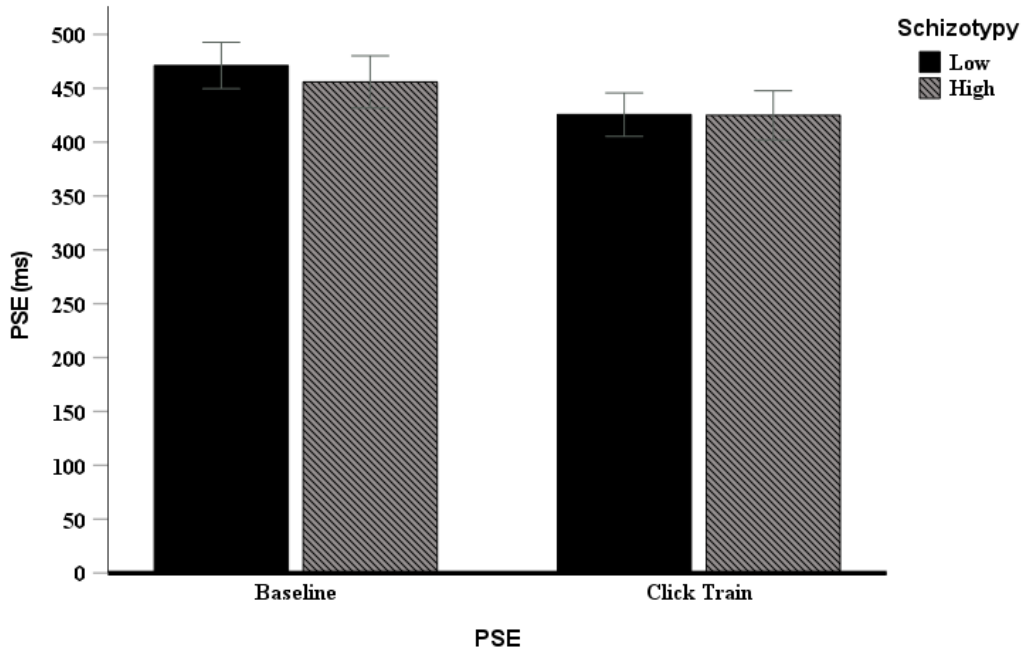

Figure 4.3: The PSE showing that the click train was effective in leading subjects to overestimate durations, irrespective of schizotypy level.

Furthermore, to explore subjects' sensitivity to durations, a 2[Clicker Presence (Baseline, Click Train)] X 2 (Schizotypy[Low, High] was conducted. There was no main effect of condition, $F(1, 123) = .649$, $p = .422$, $\eta_p^2 = .005$, [.0000, .0576]. However, there was a significant interaction between Clicker presence and Schizotypy, $F(1, 123) = 8.141$, $p = .005$, $\eta_p^2 = .062$, [.0058, .1578]. Finally, similar to the PSE analysis, there was no significant main effect of Schizotypy, $F(1, 123) = .969$, $p = .327$, $\eta_p^2 = .008$, [0000., 0649]. The descriptive statistics for the Difference Limen are displayed in Table 4.3. The significant interaction between condition and Schizotypy warrants further investigation. A simple-effects analysis showed that the mean difference ($M = 7.78$; SD = 67.65) between Low and High Schizotypy,

in baseline was not significant $F(1, 123) = 1.654$, $p = .201$, η_p^2=.013, [0000., 0779]. However, the mean difference (*M = - 18.97; SD = 94.81)* between Low and High Schizotypy was significant, *F(1, 123) = 5.003, p = .027,* η_p^2=.039, [0000., 1234]. This interaction is shown in Figure 4.4; and implies that High Schizotypy are less sensitive to the Click Train than their Low Schizotypy compatriots, as indexed by a higher percentage of DL. Furthermore, whilst there is no significant interaction between Schizotypy and LD, Schizotypy subjects do appear to have greater precision in durations than Low Schizotypy at baseline, though, this result is not statistically significant .

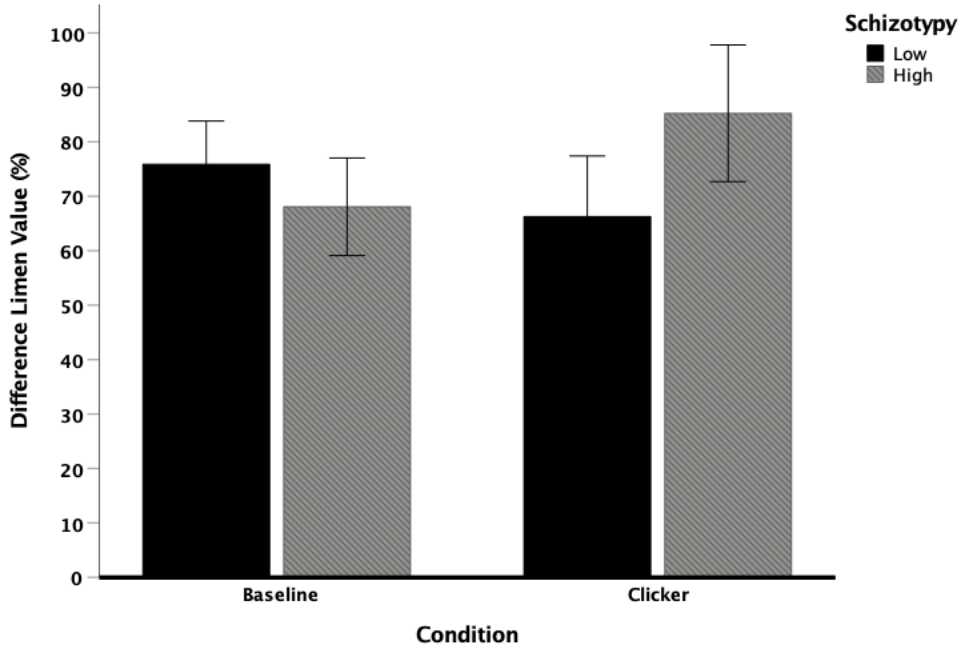

Figure 4.4: The Difference Limen values for High and Low Schizotypy in Baseline and Click Train conditions.

Table 4.2

Descriptive statistics of the PSE for baseline and clicker (High/Low Schizotypy)

Schizotypy	Baseline		Clicker	
	M	*SD*	*M*	*SD*
High	471.2	87.4	425.6	90.9
Low	456.0	95.2	425.0	81.7

Table 4.3

Descriptive statistics of the DL for baseline and clicker (High/Low Schizotypy)

Schizotypy	Baseline		Clicker	
	M	*SD*	*M*	*SD*
High	69.0	31.6	85.3	53.8
Low	75.9	35.1	74.6	47.8

In summary, Experiment 1 showed that the duration of visual stimuli were overestimated when preceded by a Click Train, irrespective of Schizotypy level, meaning that I have to reject my second hypothesis that the Click Train would not shift High Schizotypy subjects' psychophysical function to the left relative to baseline. The simple-effects analysis showed this left-ward shift was present between 300ms – 600ms; whilst the PSE showed an overall shift, irrespective of Schizotypy level (main effect) There were no differences in the pattern of 'long' responses between the two Schizotypy groups meaning that I reject my first hypothesis that High Schizotypy would overestimate durations. The findings replicate previous findings in the visual domain (e.g., Penton-Voak et al., 1996). In terms of how this finding is typically interpreted, the consensus (e.g., Wearden, 2016) is that the pacemaker is 'aroused' by the presence of the Click Train (e.g., Treisman, 1990; Wearden, 2016). This 'arousal' increases the rate of the pacemaker, meaning that it will emit a greater number of ticks, resulting in an overestimation of durations, which is precisely the pattern of results shown in Figure 4.1. In terms of Schizotypy, the results showed that the Click Train worked for both Low and High Schizotypy subjects, implying the Schizotypy pacemaker is not retarded. However, interestingly, High Schizotypy subjects demonstrated less sensitivity to the Click Train as evidenced by the Difference Limen, despite this, the Click Train condition was still effective in shifting their psychophysical function to the left, as shown in and Figure 4.1 (top panel) for the psychophysical function and Figure 4.3 for the PSE.

Unlike earlier evidence (e.g., Reed & Randell, 2014; Lee te al., 2006), High Schizotypal subjects did not show a tendency to overestimate durations of visual stimuli. The literature is scant with regard to visual bisection and Schizotypy however, Reed and Randell's (2014) study was the closest analogue to the current study. They showed that subjects scoring high in the unusual experiences Schizotypy subscale overestimated durations. We did not observe any such differences in the current study. Note, however, Reed and Randell (2014) found this difference in terms of the PSE, as opposed to the percent long' responses however, it might be argued that the PSE and percentage of long responses measure different timing processes, according to SET (See Chapter 2). Nonetheless, I analysed the PSE which only showed a main effect of Click Train, similar to the percent of long response analysis. A further study, by Carroll et al. (2008) showed Schizophrenics tended to underestimate durations, which contradicts my findings. However, it is unknown whether this pattern of results might have resulted from the effects of medication and/or other comorbidities that could have affected timing. Furthermore, modality could have affected the Experiment, hence the requirement for an auditory bisection task.

4.5 Experiment 2: Auditory Temporal Bisection Task

Experiment 2 was a replication of Experiment 1 with auditory, instead of visual, stimuli. On the basis of Experiment 1, it was expected that the Click Train would shift subjects' psychophysical function to the left, manifesting as an overestimation of durations (e.g., Penton-Voak et al., 1996; Wearden, Win & Philpott, 1999), though, not as strong as the visual domain (Penton-Voak et al., 1996. Previous evidence from Schizophrenic patients has shown that they tend to overestimate durations (e.g., Elvevåg et al., 2003), compared to matched healthy controls. Others, however, have found no difference between High and Low Schizotypy in judging the durations of auditory domain (e.g., Lee et al., 2006) therefore, my

second hypothesis, similar to the visual paradigm, is that High Schizotypy subjects will overestimate durations relative to Low Schizotypy subjects. Finally, I expected a significant interaction between Schizotypy Level and DL, based on Experiment 1. I also analysed the PSE and DL within this experiment for continuity across experiments.

4.6 Method

4.6.1 Subjects

A new sample of 174 naïve subjects (102 females, 42 males) were recruited via the prolific experiment participation website. Subjects received £4.50 for their time. The mean age was 25.7 (SD + 8.6; range = 18 – 58) years. The study was approved by the School of Psychology Ethics Committee, Swansea University. G-Power analysis for a mixed-model analysis of variance (ANOVA) with two groups and two conditions, using a rejection criterion of $p < .05$, 90% power, and for a medium effect size, suggested a sample size of 108, which is no surprise, considering this was the same power analysis for Experiment 1.

4.6.2 Stimuli & Materials

The stimulus used for presenting the durations (in both training and test phrases) was a single 144Mhz tone. The click-train for the clicker conditions was a 144Mhz tone, that lasted for 10ms, with 190ms of silence, over 5000ms, which was perceived as a 5s click train. Schizotypy scores were determined using the O-LIFE questionnaire (see Experiment 1 for description).

4.6.3 Design and Procedure

The design and procedure of Experiment 2 were the same as Experiment 1, apart from the fact that the stimuli were auditory, as opposed to visual. The same data cleaning method

as in Experiment 1 was used and resulted in two male subjects being excluded. They had a mean age of *34.5 (SD ± 16.5*) and a positive schizotypy score of 8, and 5, respectively. In Experiment 2 (after cleaning) there were 76 subjects in the lower schizotypy group (mean = 8.51 ± 3.25; range 1 – 13), and 96 in the higher schizotypy group (mean = 19.22 ± 3.51; range 14 – 30). Once again, the dependent variables were: the percentage of long responses, the value of the PSE and value of the DL.

4.7 Results and Discussion

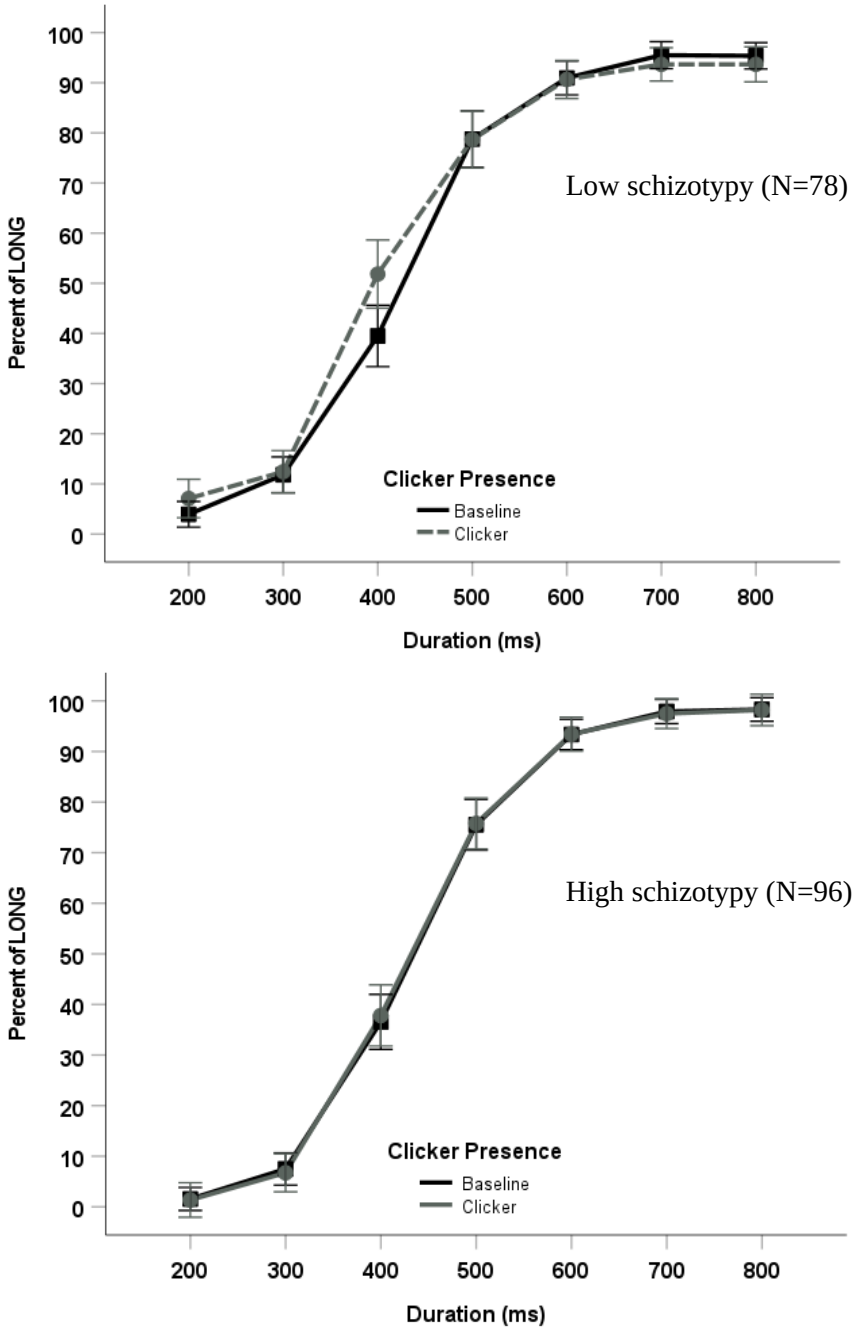

Figure 4.5: Percent of 'long' responses for Low Schizotypy (top panel) and High Schizotypy (bottom panel) subjects in Experiment 2 (auditory stimuli).

Mean percent of 'long' responses per condition appears in Figure 4.5. A 2 (Clicker Presence (Baseline, Click Train) x 7 (Duration[200, 300, 400, 500, 600, 700, 800ms]) X 2

(Schizotypy[Low, High) was carried out on percentage of 'long' responses. There was a significant main effect of *Duration*, $F(6,1020) = 1385.58$, $p<.0001$, $\eta_p^2=.89$, [.0000, .9831]. Although there was no significant main effect of Clicker Train $F(1,170)=1.33$, $p=.25$, $\eta_p^2=.008$, [.0000, .2390], there was a significant Click Presence X Duration interaction, $F(6, 1020)=2.92$, $p=.008$, $\eta_p^2=.017$, [.0000, .1089]. To determine what duration the Click Train was effective for, a simple-effects analysis was conducted. This simple-effect analysis is shown in Figure 4.6 and Table 4.4 in which it can be seen that the Click Train was only effective for shifting the 400ms duration to the left resulting in an overestimation of the 400ms duration.

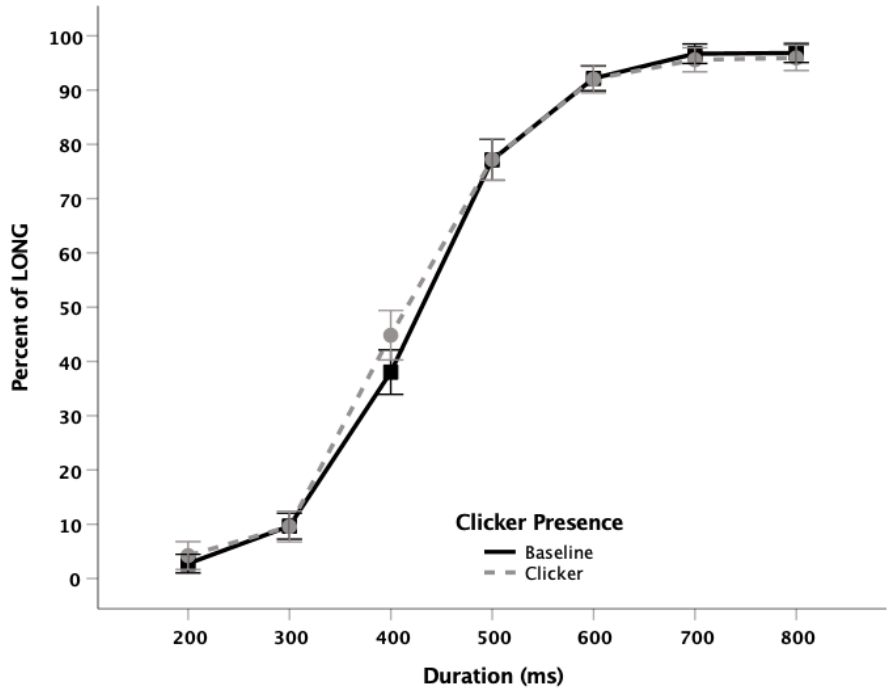

Figure 4.6: The Clicker Presence x Duration interaction. The duration where the Click Train had a significant effect was the 400ms duration.

Table 4.4

Simple-Effects analysis examining the Click Presence and Duration interaction

Duration (ms)	Baseline		Click Train		*F(1, 170)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	2.52	11.32	3.90	17.12	1.229	.269	.007	.0000	.0520
300	9.41	15.90	9.26	18.80	.002	.972	.000	.0000	.0001
400	37.87	26.94	44.03	30.74	8.432	.004*	.047	.0048	.1218
500	76.97	24.80	77.07	25.03	.001	.972	.000	.0000	.0001
600	92.30	14.94	92.21	16.71	.007	.933	.000	.0000	.0017
700	96.85	11.93	95.81	14.75	1.045	.308	.006	.0000	.0492
800	97.02	11.65	96.21	15.53	.561	.455	.003	.0000	.0408

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

Although there was no significant main effect of Schizotypy, $F(1,170) = 1.52$, $p = .219$, $\eta_p^2 = .009$, [.0000, .2752], the *Schizotypy X Duration* interaction was significant, $F(6,170) = 4.68$, $p < .001$, $\eta_p^2 = .027$, [.0000, .1638].There were no other significant interactions [Clicker Presence X Schizotypy, $F(1,170) = 1.28$, $p = .26$, $\eta_p^2 = .007$, [.0000, .2423]; Clicker Presence X Duration X Schizotypy, $F(6,1020) = 1.934$, $p = .072$, $\eta_p^2 = .011$, [.0000, .0749]].

To decompose the Schizotypy X Duration interaction, a simple effects analysis was conducted, and the result of this simple-effects analysis is shown in Table 4.5. The Schizotypy X Duration interaction is illustrated in Figure 4.7 in which it can be seen that High Schizotypy underestimates (e.g., rightward shift of psychophysical function) durations from 200ms – 400ms; and the 800ms duration is overestimated and is shown in Table 4.5.

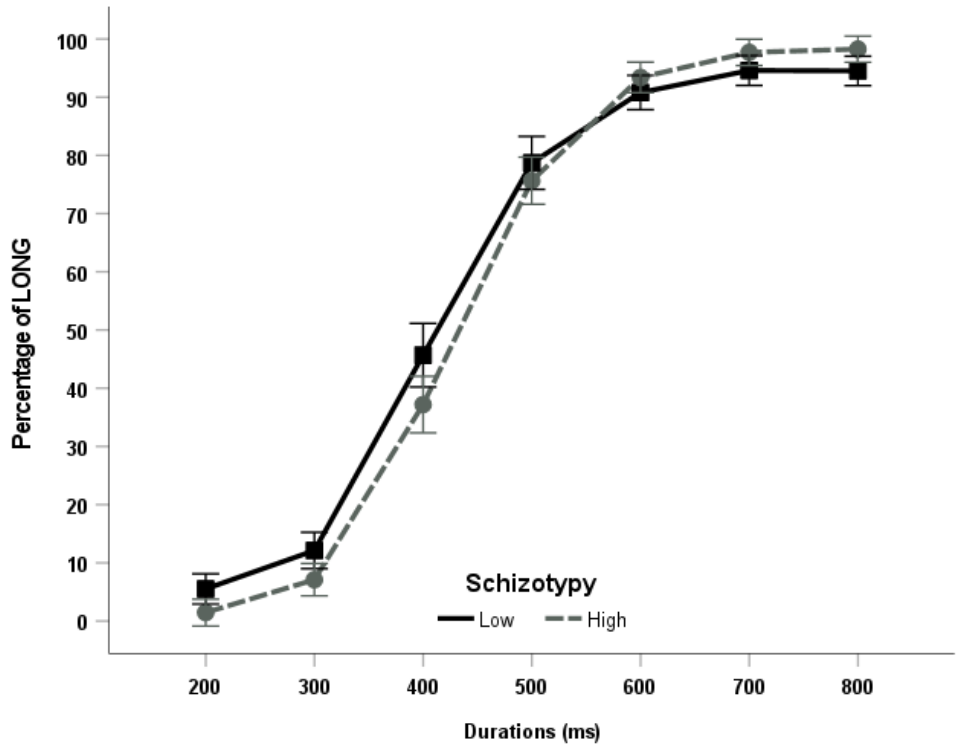

Figure 4.7: Illustration of the significant Schizotypy X Duration interaction in Experiment 2 (auditory stimuli).

Table 4.5

Simple-Effects analysis examining the Schizotypy and Duration interaction

Duration (ms)	High Schizotypy		Low Schizotypy		*F(1, 170)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	1.46	15.34	5.54	17.25	5.33	.022*	.030	.0000	.0696
300	7.12	18.64	12.13	20.71	5.629	.019*	.032	.0006	.0989
400	37.20	32.29	45.68	36.29	5.247	.023*	.030	.0002	.0955
500	75.67	26.87	78.73	30.23	.989	.321	.006	.0000	.0483
600	93.40	17.44	90.81	19.61	1.676	.197	.010	.0000	.0581
700	97.71	15.10	94.60	14.75	3.225	.074	.019	.0000	.0760
800	98.27	14.95	94.53	16.80	4.749	.031*	.027	.0000	.0910

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

Since the Duration x Schizotypy interaction effect size is small, it would be pertinent to analyse the PSE to determine if the same interaction is present overall in a single metric. A 2(Clicker Presence[baseline, clicker])-way ANOVA, with Schizotypy as a between subject factor was conducted. There was a just-significant main effect of Clicker Presence, *F*(1, 170)

= 4.501, p = .035, η_p^2 = .026, [.0000., .0887] and a strong main-effect of Schizotypy, F(1,170) = 9.146, p = .003, η_p^2 = .051, [.0062., .1272]; but no significant interaction between the Clicker Presence and Schizotypy, F(1, 170) = 2.854, p = .093, η_p^2 = .017, [.0000., .0721.]. The descriptive statistics for the PSE for both conditions are displayed in Table 4.7. To fully explore the main effect of Schizotypy, an overall PSE was calculated and subjected to an independent-samples t-test. The mean difference (*M = 27.99; SD = 121.40)* between High Schizotypy (*M = 436.77; SD = 55.46)* and Low Schizotypy *(M = 408.78; SD 65.90)* was significant, *t(170) = -3.024, p = .001, [- 46.27., - 9.72]* implying that overall, High Schizotypy underestimated durations overall, in accordance with the percentage of Long responses analysis in Table 4.6 and Figure 4.7, respectively.

To test subjects' precision to durations, I conducted a 2 (Clicker Presence[baseline, Click Train]-way ANOVA on the Limen Difference. There was no main effects of condition, F(1, 170) = 2.269, p = .134, η_p^2 = .013, [.0000., .0654] or Schizotypy, F(1, 170) = 2.747, p = .099, η_p^2 = .016, [.0000., 0709]; and finally, no interaction between condition and Schizotypy, F(1, 170) = 3.089, p = .081, η_p^2 = .018, [.0000., .0746]. The descriptive statistics for the Limen Difference are displayed in Table 4.7.

Table 4.6

Descriptive statistics of the PSE for baseline and clicker (High/Low Schizotypy)

Schizotypy	Baseline		Clicker	
	M	*SD*	*M*	*SD*
High	438.18	66.0	435.4	64.7
Low	421.2	75.5	396.4	90.1

Table 4.7

Descriptive statistics of the DL for baseline and clicker (High/Low Schizotypy)

Schizotypy	Baseline		Clicker	
	M	*SD*	*M*	*SD*
High	58.6	22.6	59.3	29.0
Low	58.8	22.3	50.3	20.5

In summary, several findings emerged from Experiment 2. First, the effect of Click Train on duration judgments of auditory stimuli had a much smaller effect than for visual stimuli in Experiment 1, as indicated by only shifting the 400ms duration to the left (Table 4.4 and Figure 4.6, respectively). This is consistent with previous evidence for a smaller effect of Click Trains on increasing perceived durations of auditory stimuli (e.g., Penton-Voak et al., 1996; Wearden et al., 1999), meaning that I can accept my first hypothesis that the Click Train would work, but not be as effective for the auditory duration as the visual duration. Secondly, compared to Low Schizotypy subjects, High Schizotypals showed a steeper gradient in the psychophysical function for auditory stimuli, with a tendency to underestimate durations below the arithmetic mean (e.g., 200ms – 400ms) and overestimate the 800ms duration as shown in Table 4.5 and Figure 4.7, meaning that I can reject my second hypothesis that High Schizotypy subjects would overestimate durations, though I note that they did overestimate the 800ms duration. This pattern of result appears to demonstrate better precision in identifying durations as opposed to Low Schizotypy, as a steeper gradient is taken as indicating greater temporal precision (Wearden et al., 1996). Whilst direct evidence for better precision would be reflected in the DL, of which the Schizotypy X Condition interaction would be significant. Figure 4.7, nonetheless, implies High Schizotypy had a steeper gradient, despite the lack of interaction. Finally, the pattern of results seen in Experiment 1, with respect to High Schizotypy being less sensitive to the Click Train is not replicated in Experiment 2, with respect to the analysis of the Limen Difference.

Further evidence to support my finding was Carroll et al.'s (2008) investigation into schizophrenia and time perception, using the auditory bisection task. They found that compared to healthy controls, Schizophrenia patients were more likely to underestimate durations in the auditory domain, which is similar to my result for High Schizotypals for the

200 – 400ms durations, though, there needs to be a degree of caution in interpreting our results given the small effect sizes for some main effects and interaction. Both Carroll et al., and the current Experiment 2 suggest that schizophrenia-spectrum disorders appear to manifest as timing deficits in terms of underestimating some durations. Carroll et al's (2009) data also implies this 'switch over' effect in the data, however, the direction is different, in which I have found Schizotypy subjects have steeper gradients, implying greater temporal precision, as opposed to Carroll et al.'s (2009) findings, which were explained in terms of the memory component. To further analyse these results, I conducted analysis between Experiments 1 and 2, respectively.

4.8 Between-Experiment Analysis:

To assess how subjects differed between Experiments, a 7 (Duration [200, 300, 400, 500, 600, 700, 800ms]) X 2 (Clicker Presence[baseline, Click Train]) X 2 (Experiment [Experiment 1, Experiment]) X 2 (Schizotypy [High, Low) mixed ANOVA with repeated measures on Duration and Click Train, showed a significant main-effect of Clicker Presence, $F(1,293)=27.175$, $p<.001$, $\eta_p^2=.085$, [.0000, .7984]. There was also a main effect of Duration, $F(6,1758) =2452.201$, $p<.001$, $\eta_p^2 = .893$, [.0000, .9835]. There was also a significant main effect of Experiment, $F(1, 293)=9.873$, $p=.002$, $\eta_p^2 = .033$, [.0000, .5900] with higher percentage of 'long' responses in Experiment 2 (auditory) compared to Experiment 1 (visual). The main effect of Schizotypy was not significant, $F(1,293)=.031$, $p=.860$, $\eta_p^2 =.000$, [.0000, .0045].

There was a significant Clicker Presence X Experiment interaction, $F(1,293)=14.730$, $p<.001$, $\eta_p^2=.048$, [.0000, .6822] a significant Duration X Experiment interaction, $F(6, 1758)=7.42$, $p<.001$, $\eta_p^2 =.025$, [.0000, .1531]; and Clicker Presence X Duration interaction, $F(6,1758)=10.90$, $p<.001$, $\eta_p^2 =.04$, [.0000, .2097]. Those two-way interactions were further

qualified in a significant Experiment X Schizotypy X Duration interaction, $F(6,1758)=3.175$, $p=.004$, $\eta_p^2 = .011$, [.0000, .0718] and a Clicker Presence X Duration X Experiment interaction, $F(6,1758)=4.218$, $p<.001$, $\eta_p^2 = .014$, [.0000, .0932]. The Clicker Presence X Duration X Experiment was expected, given the results that the Click Train was more effective in shifting the visual psychophysical function to the left (Figure 4.2 – Experiment 1) than the auditory psychophysical function (Figure 4.6 – Experiment 2). The remaining interactions were not significant[1].

The first of these two-way interactions, Clicker Presence and Experiment was expected, given the results of Experiments 1 and 2 respectively. The simple-effects analysis shows, as expected that the mean difference (*M = -5.79; SD = 142.82)* between Baseline (*M = 53.35; SD = 9.87*) and Click Train *(M = 59.14; SD = 10.76)* was significant for Experiment 2 visual), *F(1, 293) = 35.35, p < .001, η_p^2 = .108,* [.0496., .1768] however, the mean difference *(M = -.830; SD 121.70)* between Baseline (*M = 59. 05; SD = 9.90)* and Click Train (*M = 59.93; SD = 10.79)* was not significant, *F1, 293)* = 1.124, *p* = .290, η_p^2 = *.004, [0000., 0300]*. This interaction is shown in Figure 4.8 and shows that Click Train was more effective in Experiment 1 (auditory) than Experiment 2 (visual).

[1] [Clicker Presence X Schizotypy, $F(1, 293) = 1.57$, $p = .211.$, $\eta_p^2 = .005$, [.0000, .1866]; Duration X Schizotypy, $F(6, 1758) = 1.52$, $p = .166$, $\eta_p^2 = .005$, [.0000, .0358]; and Schizotypy X Experiment, $F(1, 293) = 1.89$, $p = .170$ $\eta_p^2 = .006$, [.0000, .2158]; Clicker Presence, Schizotypy and Experiment, $F(1, 293) = .009$, $p = .924$, η_p^2 .000, [.0000, .0013]; Clicker Presence, Duration and Schizotypy, $F(6, 1758) = 1.834$, $p = .127$, $\eta_p^2 = .006$, [.0000, .0428]; Clicker Presence, Duration, Schizotypy and Experiment, $F(6, 1758) = .745$, $p = .614$, $\eta_p^2 = .003$, [.0000, .0178].

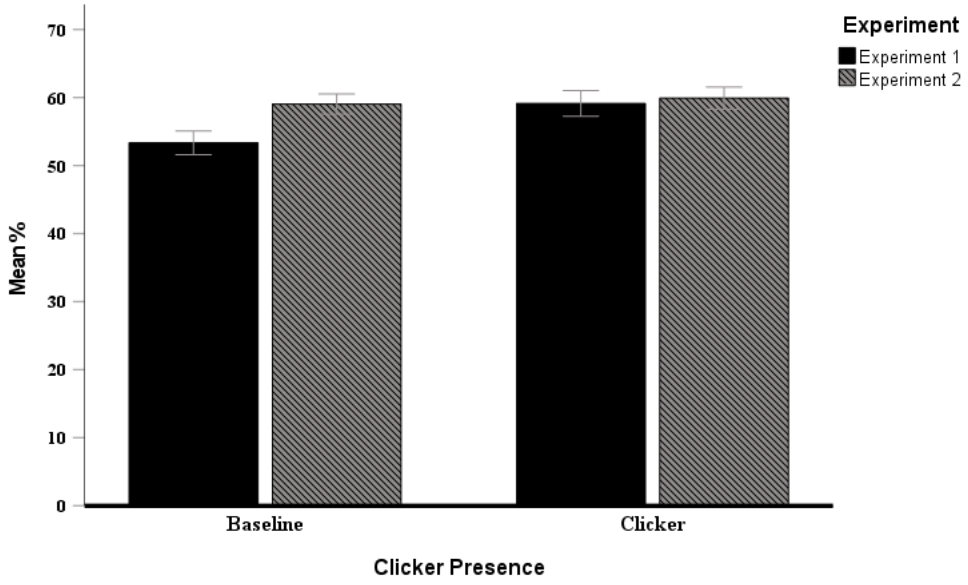

Figure 4.8: The Clicker Presence and Experiment interaction, showing how the Click Train was more effective in Experiment 1 (auditory) than Experiment 2 (visual).

The second of these interactions was Duration and Experiment, in which a simple-effects analysis was used to decompose the results, which is shown in Table 4.8. This interaction is shown in Figure 4.9, in which it can clearly be seen that overall, subjects in Experiment 2 (auditory) overestimate durations, specifically at the 500ms and 600ms durations, implying a steeper gradient and greater precision in Experiment 2 (auditory).

Table 4.8

Simple-Effects analysis examining the Duration and Experiment interaction

Duration (ms)	Experiment 1 (visual)		Experiment 2 (auditory)		*F(1, 293)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	3.39	10.76	3.50	10.82	.006	.937	.000	.0000	.0009
300	10.59	13.68	9.62	13.68	.360	.549	.001	.0000	.0215
400	37.26	27.75	41.44	20.16	2.266	.133	.008	.0000	.0392
500	65.92	20.54	77.20	20.51	21.849	<.001*	.069	.0237	.1309
600	85.70	13.57	92.11	13.56	16.137	<.001*	.052	.0137	.1088
700	94.32	10.31	96.15	10.31	2.288	.131	.008	.0000	.0393
800	96.55	10.08	96.40	10.09	.017	.897	.000	.0000	.0025

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

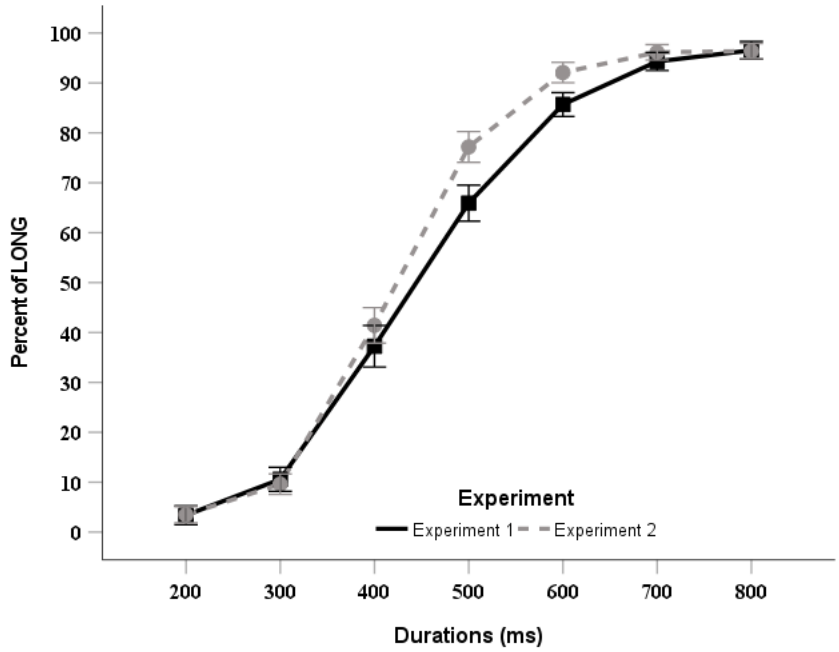

Figure 4.9: The Duration and Experiment interaction, clearly showing that subjects overestimated durations in Experiment 2 (auditory) relative to Experiment 1(visual); specifically between 500ms and 600ms.

Of particular interest was the significant three-way interaction of Experiment X Schizotypy X Duration interaction, illustrated in Figure 4.10. To explore the interaction, two separate mixed 2 (Experiment: visual vs. auditory) X 7 (Duration: 200ms – 800ms) ANOVAs were carried out on percent of 'long' responses of low and high schizotypy subjects. For low schizotypals, there were significant main effects of Duration, $F(6,864)=966.564$, $p<.000$, $\eta_p^2=.870$, [.0000, .9795]; Experiment, $F(1,144)=8.853$, $p=.003$, $\eta_p^2=.058$, [.0000, .7228]; and a significant Experiment X Duration interaction, $F(6,864)=4.755$, $p<.001$, $\eta_p^2=.032$, [.0000, .1901]. To further investigate which durations Low Schizotypy subjects differed on between experiments, a simple-effects analysis was conducted and the results of this simple-effects analysis are reported in Table 4.9 and shown in Figure 4.10 (upper panel) respectively.

Table 4.9

Simple-Effects analysis examining the Experiment X Schizotypy X Duration for Low Schizotypy

Duration (ms)	Experiment 1 (visual)		Experiment 2 (auditory)		*F(1, 144)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	2.36	8.71	5.53	16.74	2.006	.159	.014	.0000	.0726
300	9.42	13.1	12.13	17.75	1.051	.307	.007	.0000	.0576
400	36.30	23.06	45.68	24.44	5.528	.020*	.037	.0005	.1136
500	65.23	21.69	78.73	20.32	15.080	<.001*	.095	.0233	.1919
600	84.48	14.49	90.81	16.94	5.835	.017*	.039	.0000	.1167
700	94.49	7.60	94.60	16.26	.003	.958	.000	.0000	.0009
800	97.14	24.59	94.53	16.19	1.641	.202	.011	.0000	.0672

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

The simple-effects analysis shows that Low Schizotypy subjects overestimated auditory durations between 400ms – 600ms (Experiment 2) relative to visual durations (Experiment 1) as shown in the upper panel of Figure 4.10, and is indicative of the classic effect that auditory stimuli are perceived longer than visual stimuli (e.g., Wearden et al., 1990 Carrol et al., 2008).

For High Schizotypals there was a significant main effect of Duration, $F(6,894)=1640.767$, $p<.001$, $\eta_p^2=.917$, [.0000, .9874]; but not Experiment, $F(1, 149)=1.846$, $p=.176$, $\eta_p^2=.012$, [.0000, .3446]. There was, however, a significant Experiment X Duration interaction, $F(6, 894)=6.164$, $p<.001$, $\eta_p^2=.040$, [.0000, .2268]. Similar to the Low Schizotypy, a simple-effects analysis was conducted for High Schizotypy which are reported in Table 4.10 and shown in Figure 4.10 (bottom panel). This simple-effects analysis confirms that High Schizotypy subjects underestimated durations of 200ms and 300ms and overestimated auditory durations of 500ms – 700ms implying steeper gradients.

Table 4.10

Simple-Effects analysis examining the Experiment X Schizotypy X Duration for High Schizotypy

Duration (ms)	Experiment 1 (visual)		Experiment 2 (auditory)		*F(1, 149)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
200	4.43	10.38	1.46	3.70	5.432	.012*	.01	.0019	.1188
300	11.76	12.67	7.12	9.0	6.510	.012*	.042	.0020	.1196
400	38.21	21.08	37.20	23.86	.068	.794	.000	.0000	.0190
500	66.62	19.51	75.66	19.91	7.322	.008*	.047	.0034	.1270
600	86.91	13.51	93.40	8.79	12.748	<.001*	.079	.0159	.1702
700	94.16	9.69	97.71	4.35	9.577	.002*	.024	.0079	.1460
800	95.97	10.62	98.27	4.04	3.607	.059	.024	.0000	.0901

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

For continuity with Experiments 1 and 2, I also analysed the PSE by running a 2(Clicker Presence [Baseline, Click Train) x 2(Experiment [Experiment 1, Experiment 2]) x 2(Schizotypy [Low, High]) mixed ANOVA. There were main-effects of Click Presence, *F(1, 293) = 22.192, p < .001*, , η_p^2=.070, [.0243., .1322] and Experiments, *F*(1, 293) = 7.916. *p* = .005, , η_p^2=.026, [.0024., .0720]. There were also two significant two-way interactions in terms of Clicker Presence and Experiment, *F(1, 293) = 4.895; p = .028,* η_p^2=.016, [.0000., .0559] and Experiment and Schizotypy, *F*(1, 293) = 5.433, *p* = .020, η_p^2=.018, [.0002., .0589]. However, there was no main effect of Schizotypy, *F*(1, 293) = 1.695, *p = .194*, η_p^2=.006, [.0000., .0348] and no two-way interaction between Clicker Presence and Schizotypy, *F(1, 293)* = 2.735, *p* = .099, η_p^2=.009, [.0000., .0325] and finally, no three-way interaction between Clicker Presence, Experiment and Schizotypy, *F(1, 293) = .113, p = .737*, η_p^2=.000, [.0000., .0162]. The two-way interaction between Clicker Presence and Experiment is to be expected on the basis of the results of the PSE analysis in Experiment 1 (visual modality) and Experiment 2 (auditory modality) and has been explored in both, in which the PSE demonstrated a significant main effect in Experiment 1 but not Experiment 2. These results are discussed in Experiments 1 and 2 respectively. However, to explore the two-way interaction between Experiment and Schizotypy, a simple-effects analysis was

conducted. The mean difference in Experiment 2 (*M = - 27.99; SD = 111.76)* between Low Schizotypy (*M* = 408.78; *SD = 90.24)* and High Schizotypy *(M = 436.78; SD = 81.64)* was significant, *F(1, 293) = 7.843, p = .005,* η_p^2=.026, [.0023., .0716]. However, the mean difference in Experiment 1 (*M = - 7.930; SD = 131.16)* between High Schizotypy *(M = 440.49; SD = 107.87.02)* and Low Schizotypy, *(M = 448.42; SD = 94.02)* was not significant, *F*(1, 293) = .457, *p* = .500, η_p^2=.002, [.0000., .0229] which is to be expected, given the results of Experiment 2, in which High Schizotypy subjects underestimated durations from 200ms – 400ms (Figure 4.7), and a higher PSE relative to Low Schizotypy (Table 4.6).

The simple-effects analysis of the percentage of long responses, and the PSE implies that both Low and High Schizotypy have greater precision in auditory durations than visual durations, which should be reflected in the overall DL across Experiments, in which a lower value implies greater precision. To assess this a 1(Overall DL) x 2(Experiment[1,2] x 2(Schizotypy[Low, High]) mixed-methods ANOVA was conducted. The descriptive statistics for this ANOVA are shown in Table 4.6. There was a main effect of Experiment, *F(1, 293) = 35.342, p = <.001,* η_p^2=.108, [.0495., .1767] however, there was no main effect of Schizotypy, *F(1, 293) = 3.022, p = .083,* η_p^2=.010, [.0000., .0444] and no significant interaction between Experiment and Schizotypy, *F(1, 293) = .040, p = .841,* η_p^2=.000, [.0000., .0058]. As can be seen from Table 4.11, the difference between Experiments 1 and 2 is significant; implying that both Low and High Schizotypy subjects were more precise in identifying durations in Experiment 2 (auditory) as indicated by lower overall LD values.

Table 4.11: The descriptive statistics of the Difference Limen for High and Low Schizotypy for Experiments 1, and 2, and overall.

Experiment	Schizotypy	*M*	*SD*
Experiment 1	Low	71.08	30.52
	High	76.67	32.79
	Total	73.54	31.53
Experiment 2	Low	54.52	15.41
	High	58.95	18.85
	Total	57.99	17.51
Total	Low	62.46	25.20
	High	65.41	26.18
	Total	63.96	25.70

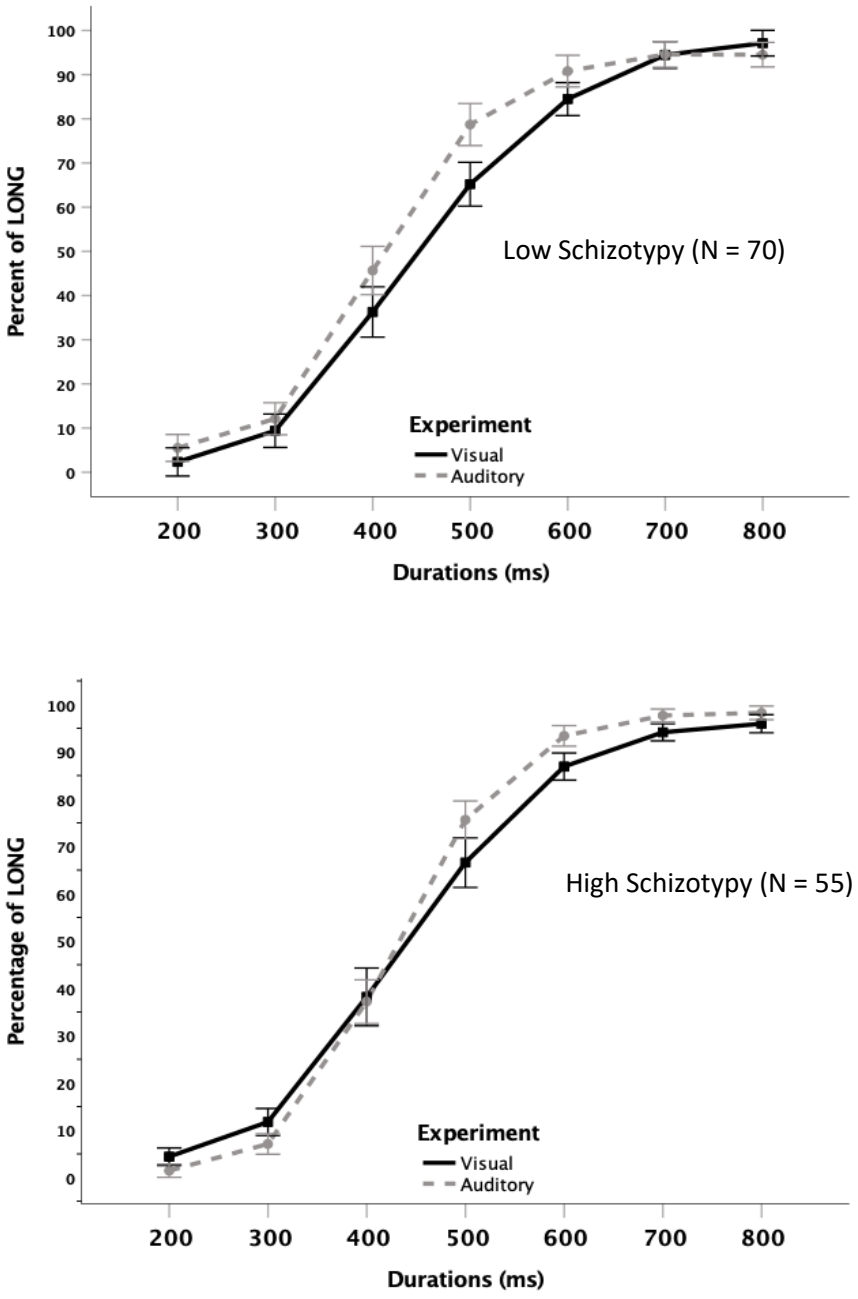

Figure 4.10: percent of 'long' responses per modality for Low Schizotypy (top panel) and High Schizotypy (bottom panel).

Overall, the results from the between-experiment analysis demonstrate that the Click Train was more effective in Experiment 1 (visual) than Experiment 2 (auditory). Furthermore, it was also shown that auditory durations were overestimated relative to visual durations (Figure 4.9). It was also shown that Low Schizotypy overestimated auditory

durations and finally, that High Schizotypy underestimated durations (200 – 300ms) and overestimated durations (500 – 700ms) implying a steeper gradient and thus, greater precision (Figure 4.10, bottom panel).

4.9 General Discussion

The aim of Chapter 4 (Temporal Bisection) was threefold. First, as Schizotypy being used as a model for examining timing functioning in Schizophrenic individuals by investigating timing functions in Schizotypy. The current data adds to existing evidence, to help understand the similarities and differences between Schizotypy subjects and Schizophrenic individuals. Specifically, the current study aimed to address a knowledge gap with regards to the time perception of Schizotypy subjects. Secondly, I examined the effect of a Click Train prior to the presentation of a stimulus on duration judgments by Low and High schizotypy subjects to test the hypothesis of the retarded pacemaker in Schizotypy subjects. I also examined the differences between visual and auditory domains in terms of how time perception may differ between the two. The Experiments were partially exploratory in nature but also informed by previous research.

The key findings across the two experiments were as follows: *First*, for visual stimulus durations, the Click Train prior to the to-be-judged stimulus increased the percentage of long responses for both Low and High schizotypy subjects (Figure 4.2) while the Click Train had less of an effect on the duration judgments of auditory stimuli, manifesting as an underestimation of the 400ms duration only (Figure 4.6). *Secondly*, the Click Train was similarly effective in leading to overestimation of durations regardless of Schizotypy level, demonstrating that High Schizotypy did not have a retarded pacemaker. *Thirdly*, High Schizotypy subjects showed a pattern in Experiment 2 of having better precision of durations (Figure 4.7), as indicated by a steeper gradient, compared to Low

Schizotypy. These findings were interpreted from the fact that High Schizotypy subjects underestimated 200ms – 400ms durations and overestimated the 800ms duration (Figure 4.7). These results were further analysed by a between-experiments analysis, in which I found that the Click Train was more effective in the visual domain, than the auditory domain (Figure 4.8) and that auditory durations were overestimated relative to visual durations (Figure 4.9). I also found that High Schizotypy underestimated auditory durations (200 – 300ms) and overestimated auditory durations (500ms – 700ms) relative to visual durations, demonstrating the finding that High Schizotypy show greater precision in the auditory modality than the visual modality (Figure 4.10, bottom panel), as indicated by a steeper gradient for the auditory domain, These results suggest that there are differences in perception of durations among Low and High Schizotypy subjects.

In Experiment 1, the Click Train led to overestimation of durations, irrespective of Schizotypy level. Penton-Voak, Edwards, Percival & Wearden (1996) showed that the Click Train successfully shifted the psychophysical curve to the left, which is in accordance with Experiment 1 (see Figure 4.2) and Penton-Voak (2010). I have shown that the Click Train is effective for speeding-up *visual durations* in the sub-second paradigm. This is in keeping with Penton-Voak et al (1996) and Wearden et al. (1999) who both showed the Click Train effect was small, in shifting the psychophysical function to the left. The Click Train is thought to influence timing by speeding up the pacemaker (e.g., Treisman, 1990; Penton-Voak et al., 1996; Wearden et al., 1999); Wearden, 2016). The idea is that the presentation of repetitive stimuli arouses the pacemaker, which in turn, causes the pacemaker to emit a greater number of pulses, leading the subject to overestimate durations, which is what I showed in Figure 4.2 Furthermore, in the auditory domain, I did not find a main effect of Click Train but an interaction between Click Train and duration, manifesting as an overestimation of the 400ms duration, demonstrating that the Click Train is less effective in

the auditory modality than visual modality (Figure 4.6) One explanation of this finding is that since auditory durations are already overestimated, compared to visual durations (e.g., Wearden, et al., 1999), which is evidence I report in Section 4.8 – and illustrated in Figure 4.9 the Click Train would be less effective in the auditory domain, as shown in Figure 4.8. and in accordance with Penton-Voak et al. (1996).

In the absence of a Click Train, previous effects of modality and Schizophrenia and Schizotypy were partially confirmed. That is, the auditory modality (Experiment 2) moved the psychophysical function to the left suggesting an underestimation of the 200ms to 400ms durations and an overestimation of the 800ms duration, relative to Low Schizotypy (Figure 4.7). Most interestingly, levels of Schizotypy interacted with stimulus modality and stimulus duration: for auditory but not visual stimuli, High Schizotypy subjects showed a 'switchover' whereby they *underestimated* durations from 200ms to 300ms duration, while they appeared to *overestimate* durations from 500ms to 800ms durations (see Figure 4.10, bottom panel). This 'switchover' suggests that High Schizotypy subjects had a steeper gradient, which is indicative of a more precise representation of duration, which contradicts the findings of Papagerigiou et al. (2013). However, it should be noted that Papagerigiou et al. (2013) focused on Schizophrenia as opposed to Schizotypy. If High Schizotypy subjects demonstrate greater temporal precision, this implies that the psychophysical curve will be steeper than in Low Schizotypy subjects, which is the case in terms of the High Schizotypals in which the auditory short anchor curve (up to 400ms) is steeper (see Figure 4.7). A similar (though opposite) pattern of results is observed in Schizophrenia patients by Elvevåg et al. (2003), in which they found that Schizophrenic subjects overestimate durations from 200 – 300ms but underestimate durations from 400ms – 700ms, which is in the opposite direction to my results. Elvevåg et al. (2003) explained this in terms of a memory deficits. In terms of what it means to say that High Schizotypy have greater precision of temporal representation,

precision refers to the degree of variability of judgements (Grondin, 2010). In the auditory experiment, Schizotypy subjects showed greater precision in (a) the auditory modality, relative to Low Schizotypy and (b), greater precision in the auditory modality than in the visual domain, meaning there was less variability around durations. This is contrary to some of the literature, in that it is often found that Schizophrenia results in less temporal precision (Thoenes & Oberfeld, 2017). However, given the fact that the pacemaker is not retarded in High Schizotypy in neither visual (Figure 4.1., bottom panel) or auditory (Figure 4.5, bottom panel) as shown by the effectiveness of the pacemaker, irrespective of Schizotypy level, this would imply that the pacemaker is emitting pulses are at a less variable rate than Low Schizotypy. This could be explained by the Schizotypy subjects being hyperaroused, which is similar to Schizophrenic subjects (Nakamura et al., 2003) however, it could be argued reasonably that a hyperaroused pacemaker would emit pulses at an increased rate, leading to overestimation of durations, which is the evidence I find in Experiment 2 (Figure 4.7) for the 800ms duration only. Therefore, I explain my finding that High Schizotypy subjects are more precise as their pacemaker is hyperaroused, leading to overestimation of the 800ms duration. However, this only partially satisfies the findings, given that the 200ms – 400ms durations are underestimated.

Applying SET to my findings presents a theoretical explanation for my findings however, applying SET to the data presents mixed findings. The Click Train led to an overall overestimation of durations in the visual modality (Experiment 1) for the 200 – 400ms but less so in the auditory modality (Experiment 2) suggesting the involvement of the pacemaker. On the other hand, Schizotypy differentially influenced judgments of duration for auditory stimuli. For visual stimuli (Experiment 1) Schizotypy did not influence time perception, while for auditory stimuli (Experiment 2) High Schizotypy subjects tended to underestimate the 200 – 400ms durations compared to Low Schizotypals and overestimated the 800ms

duration. This would imply that the pacemaker is emitting pulses at a quicker – but less variable – rate than Low Schizotypy subjects (at least for the 800ms durations) and consequently, durations are underestimated. However, there are difficulties in accepting the pacemaker hypothesis for the single 800ms duration, given the results of the 200ms – 400ms durations. If the pacemaker in High Schizotypy subjects deviates from Low Schizotypy subjects then we should see the same set of results in both modalities (e.g., auditory and visual) and in both Low and High Schizotypy subjects. Whilst my results suggest the pacemaker component explains High Schizotypy overestimating the 800ms duration, due to hyperarousal, it does not explain why High Schizotypy subjects' durations do not deviate from Low Schizotypy in Experiment 1; nor why they underestimated the 200ms to 400ms duration the auditory domain. Whilst it is suggested the pacemaker is mediated by the basal ganglia (e.g., Grondin, 2010), there is no evidence of there being a 'visual' and 'auditory' pacemaker though, the possibility cannot be ruled out in terms of Schizotypy and the discussion of auditory and visual pacemakers is beyond the scope of this thesis. There is, however, evidence that in the auditory modality, High Schizotypy subjects have greater precision than in the visual modality, implying that other components of SET are involved.

A further component of SET to consider would be the switch component, which is said to be mediated directly by attentional resources in human and non-human animal timing (e.g., Gibbon, 1977; Block & Zakay, 1997). In the context of SET, when this switch is fully opened, it allows pulses to pass from the pacemaker to the accumulator without variability; indicative of subjects paying full attention to the stimulus attributes, including its duration (e.g., Treisman et al., 1990). Evidence suggests that attentional deficits are present in Schizotypy (e.g., Lenzenweger, Comblatt & Putnick, 1991; Gooding, Matts & Rollmann, 2006). Therefore, the Schizotypal pacemaker may emit pulses at a veridical rate (e.g., the pacemaker emits pulses exactly in accordance to the duration that the subject is presented

with in that that if a 300ms duration is presented, the pacemaker would emit pulses in accordance with 300ms), and as they pass to the Schizotypy switch, the switch 'flickers' which is caused by variability in attention (e.g., Meck, Church & Olton, 1984; Reed & Randell, 2014), symbiotic of attentional deficits in Schizotypy and Schizophrenia (e.g., Chen & Faraone, 2000). As a result, this would result in pulses, from the pacemaker, being 'lost' as they cross the switch, because it is flickering. This would result in fewer accumulated pulses, from the pacemaker, which would manifest as a rightward shift in the temporal gradient and thus, an underestimation of durations, which is what I find for the 200ms – 400ms durations in Experiment 2 (visual), along with the 200ms – 300ms durations in the auditory modality relative to the visual modality. However, a more viable switch would imply that Schizotypy subjects are less precise in durations, which is not the evidence I find in Experiment 2.

In summary, for Chapter 4 (Experiments 1 and 2), I explain the findings that the Click Train was effective in the visual modality as indicating that the pacemaker was aroused by the Click train, leading to an overestimating of durations, irrespective of Schizotypy level. This finding indicates that the Schizotypy pacemaker is not retarder. In terms of my finding in Experiment 2, in which the Click Train only led to overestimation of the 400ms duration, I explain this finding in the effect of the Click Train not being as strong as in the visual domain, in that it had a less arousing effect on the pacemaker. In terms of my finding that High Schizotypy underestimate (200ms – 400ms) and overestimate (800ms) durations, I explain the 800ms duration in High Schizotypy subjects having a hyperaroused pacemaker, which manifests as a steeper gradient, resulting in better precision, though, this hyperaroused pacemaker is revealed only in the presence of the 800ms duration. Given the potential that other components of SET (e.g., working memory, reference memory, and the switch) are implicated in my findings from the temporal bisection studies, a more robust timing method

is required to fully explore these findings. To do this, I shall use the temporal generalisation task in Chapter 5, which allowed me to further elucidate on the other components of SET.

Chapter five: Temporal Generalisation Gradient Dependent on Schizotypy Level:

5.1 Introduction

In Chapter 4, I interpreted the findings as High Schizotypy having greater precision in temporal processing, driven by a hyperaroused pacemaker. However, as discussed in Chapter 3, the temporal generalisation task is generally more sensitive than the temporal bisection task and allows other components of SET to be readily explored, such as reference memory, and working memory (Grondin, 2010), along with the pacemaker (Wearden, Win & Philpott, 1999). To do this, I have used, in this chapter, temporal generalisation tasks, as discussed in Chapter 3. In Experiment 3, the visual temporal generalisation task was used, in which my subjects were first trained on a 400ms standard duration, and then asked whether subsequent durations matched, or did not match the duration. Much like the experiments in Chapter 4, I have utilised both an auditory and visual temporal generalisation task to fully explore whether the finding in Chapter 4 that High Schizotypals demonstrated greater precision in the auditory domain, as opposed to the visual domain is driven by a hyperaroused pacemaker. In summary, I expected to find similar pattern of results in the temporal generalisation tasks as was found in the Temporal Bisection tasks in Chapter 3 along with evidence from Elvevåg et al., (2003). These are: (i), that the Click Train will be effective in shifting the psychophysical function to the left, irrespective of Schizotypy group, showing, once again, that the pacemaker component is not retarded in Schizotypy. (ii) That the Click Train effect will be weaker in the auditory modality than the visual modality, in accordance with my findings in

Experiment 2 (iii), that High Schizotypy will demonstrate greater precision in the auditory modality as opposed to the visual, again, in accordance with my findings in Experiment 2.

5.2 Experiment 3: Auditory Temporal Generalisation Task

Experiment 3 examined how the auditory modality of the temporal generalisation task could be impacted by Schizotypy levels, and the presence of a Click Train presented prior to the to-be-judged stimulus duration. In terms of the components of SET affected, if Schizotypy subjects have greater precision in identifying durations, then a steeper gradient should be observed indicating that the pacemaker is less viable in High Schizotypy. This can be investigated by examining how asymmetrical responses are. The steeper the gradient, the more likely 'same' response occur with stimuli longer than the standard (Klapproth & Wearden, 2011).The Click Train is also expected to influence performance independently, leading an overall leftward shift of the gradient compared to the baseline, as reported in Penton-Voak et al. (1996). Once again, on the basis of my hypotheses in Chapter 3, I predict that the Click Train will not be effective in shifting the Schizotypy temporal gradient to the left (e.g., causing an overestimation of durations) if the pacemaker component is faulty.

The following hypotheses were tested: (1), High Schizotypy would exhibit a leftward shift in temporal gradients (as a result of overestimation durations), relative to Low Schizotypals (Elvevåg et al., 2003; Penney et al., 2005); (2) based on Elvevåg et al. (2003) I was expecting higher asymmetry, indicating a steeper gradient and thus, greater temporal precision for High Schizotypy subjects; and (3) the Click Train would shift the temporal gradient to the left, reflecting a faster pacemaker and manifesting as an *overestimation* of durations (Penton-Voak et al., 1996).

5.3 Method

5.3.1 Subjects

141 subjects (92 female; 48 male) were recruited via the School of Psychology's subject pool, and the Prolific website. The mean age was 29.01(*SD*=12.10; range=18–69) years. There were 79 subjects in the lower schizotypy group (*M*=8.18, *SD*=4.04), and 62 in the higher schizotypy group (*M*=19.18 *SD*=3.41). All were naïve to the purpose of the study and were awarded 4 subject pool credits for their time or £4.50. G-Power analysis for a mixed-model analysis of variance (ANOVA) with two groups and two conditions, using a rejection criterion of $p<.05$, 90% power, and for a small effect size, suggests a sample size of 84.

5.3.2 Stimuli & Measures

The stimuli used for presenting sample and comparison durations were 144MHz tones that lasted, according to duration length (e.g., for a 400ms duration the tone would last for 400ms). In both the training and test sessions. For the click condition, a 144MHz tone, that lasted for 10ms, with 190ms of silence, over 5000ms was used. This gave the impression of a click train that lasted for 5s.

To measure psychometric self-reported schizotypy similar to Experiments 1 and 2, I used O-LIFE, which, once again, Unusual Experiences, Cognitive Disorganisation and Impulsive Nonconformity subscales map onto the positive symptoms of schizophrenia, and their sum is used as an index of that trait. The internal reliability (Cronbach α) for the current sample was 0.60. The mean value of the sum of the positive symptom scales for the whole study was 13.49. To maintain consistency of analysis across the series of studies, those with a score below 14 were classed as lower schizotypal scorers, and those with a score of 14 or greater were classed as higher schizotypal scorers. This is similar to how Reed & Randell,

(2014) categorised subjects into low or high schizotypy using a median split however, the median split can result in significant information loss (Knüppel & Hermsen, 2010) as well as producing Type I errors (Icobucci, Posaav, Kardes, Schnieder & Popovich, 2014) therefore, I categorised subjects on the basis of their mean positive schizotypy score. This allows a robust analysis of timing deficits for those with low schizotypy and those with high schizotypy (Reed & Randell, 2014).

5.3.3 Design

Experiment 3 was based on a mixed design manipulating stimulus Duration with 7 levels: 100, 200, 300, 400, 500, 600 or 700ms, and Clicker Presence with two levels: baseline (no clicker) and clicker. This design produced 14 within-subject conditions. The between-subjects variable was schizotypy level, calculated based on the scores of the OLIFE (B) questionnaire. We were specifically interested in the positive schizotypy scales. Therefore, the between-subjects variable was Positive Schizotypy with two levels: low vs. high. There were 76 Low Schizotypy (*M*=8.16, *SD*=4.10), and 59 High Schizotypy subjects (*M*=19.22, *SD*=3.44). The dependent variable was percentage of 'Yes' responses for each duration. The peak was calculated for each subject by taking the duration with the highest percent of 'Yes' responses; of which the percentage was the dependent variable. To measure the level of asymmetry, I took the mean proportion of yes responses for the durations under the standard (e.g., 100ms – 300ms) and the mean proportion of Yes responses for durations over the standard (e.g., 500ms – 700ms), in which the mean proportions were the dependent variables.

5.3.4 Procedure

Trial presentation and collection of responses was administered via the Gorilla experimental platform. In the training stage, subjects were asked to pay attention to the tone and press the spacebar to begin. On pressing of the spacebar, subjects were presented with a

400ms auditory duration, identified as the standard duration. Once they had heard the tone, they were informed that the preceding tone was the standard duration. Subjects were then instructed to press the spacebar to hear one of the seven comparison tones. Once the comparison tone had played, subjects were asked whether the sound matched the tone, and asked to press Z for 'Yes' or M for 'No'. Subjects were then informed, by a green cross, whether they had correctly identified the standard or a red cross if they did not. The cycle would then repeat for a total of 7 times. The keyboard responses were randomised to match the testing session (e.g., if the training stage had Z (No) and M (Yes), the corresponding testing session would have the same keyboard responses). Once subjects had completed the training stage. They moved onto the experimental stage.

In the experimental stage, the comparison stimuli were presented in random order, for each of the 7 possible durations. Each duration appeared 10 times yielding 140 test trials per subject. The subject was presented with a blank white screen for 1000ms. Thereafter, the subject was presented with a screen, asking 'does the tone match the standard duration?' for 1000ms. The sound was presented on the next screen, with subjects asked 'does the tone match the standard duration?'. Once the duration had stopped, subjects were presented with the option of two keyboard buttons: Z (no) or M (yes). Once subjects had completed the baseline condition, they completed the training phase, once again, followed by the clicker condition. They were first presented with a 5000ms click. Once the click had ceased, subjects advanced to a screen lasting 1000ms asking them 'Does the tone match the standard duration?'. This screen was succeeded by the duration followed by the presentation of two keyboard buttons: Z(no) and M (yes). After subjects completed all 280 trials, they completed a demographics and schizotypy questionnaire, which was followed by a debrief. Keyboard responses were randomly counterbalanced (e.g., Z (yes) M (no) in line with Reed & Randell (2014). All durations were randomised in both baseline and clicker conditions.

5.4 Results and Discussion

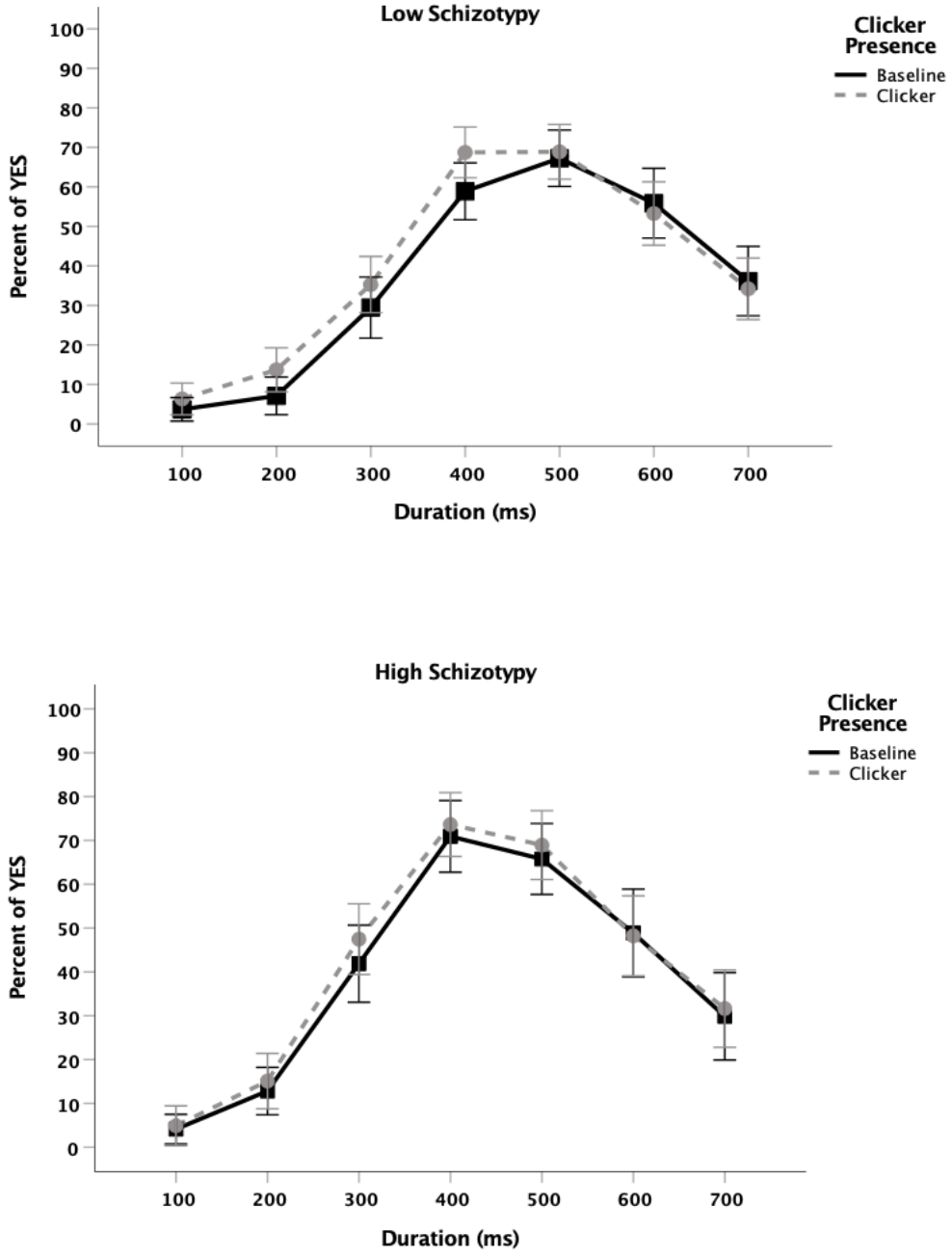

Figure 5.1: Mean percent of 'Yes' responses per condition for Low Schizotypy (top panel) and High Schizotypy (bottom panel) in Experiment 3 (auditory temporal generalisation).

The proportion of 'Yes' responses made with respect to the standard and the comparison durations was calculated for each subject for the experimental phase. The data was plotted to yield the temporal generalisation gradient, where the graph takes the appearance of percentage of 'Yes' responses on the ordinate-axis and presented stimulus durations on the abscissa-axis. Subjects whose peaks corresponded to 100ms or 700ms were removed, as they were unlikely to be paying attention to the task.. This led to the removal of 6 subject, 3 from the High and 3 from the Low Schizotypy groups.

The percentage of 'Yes' responses are shown in Figure 5.1 for Low Schizotypy (top panel) and High Schizotypy (bottom panel) groups, respectively. It is (visually) apparent that the presence of the Click Train shifted the temporal gradient, of Low Schizotypy, to the left. This was tested statistically. A 7(Duration) [100, 200, 300, 400, 500, 600, 700ms]) X 2(Clicker Presence (baseline, Click Train) X 2(Schizotypy[Low, High) with repeated measures on the first two revealed significant main effects of Clicker Presence, $F(1,133)=9.30$, $p=.003$, $\eta_p^2=.065$., [.0000, .7477], with a greater percentage of 'Yes' responses when the comparison tones were preceded by a Click Train, and Duration, $F(6,798)=129.26$, $p<.001$, $\eta_p^2=.493$ [.0000..8735], but not Schizotypy, $F(1,133)=.812$, p=.369. There was a significant Duration X Schizotypy interaction, $F(6,798)=2.32$, $p=.032$, $\eta_p^2=.017$, [.0000, .1102]. There were no other significant interactions: Clicker Presence X Schizotypy, $F(1,133)=.263$, $p=.609$, $\eta_p^2=.002$, [.0000, .0425].; Clicker Presence X Duration, $F(6,798)=1.204$. $p=.30$, $\eta_p^2=.009$, [.0000, .0182]. Clicker Presence X Duration X Schizotypy, $F(6,798= .502$, $p=.807$, $\eta_p^2=.003$, [.0000, .0074].

The Duration x Schizotypy interaction warrants further investigation via a simple-effects analysis. These results are shown in Table 5.1 and illustrated in Figure 5.2., in which High Schizotypy overestimate shorter durations (e.g., the 400ms and 500ms durations).

Table 5.1

Simple-Effects analysis examining the Duration x Schizotypy interaction

Duration (ms)	Low Schizotypy		High Schizotypy		*F(1, 133)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
100	5.04	13.36	4.53	13.37	.048	.827	.000	.0000	.0151
200	10.43	18.37	13.80	18.37	1.242	.267	.009	.0090	.0655
300	32.38	27.56	44.68	27.56	6.622	.011*	.047	.0024	.1335
400	63.82	24.00	72.28	24.00	4.122	.044*	.030	.0000	.1060
500	68.07	26.67	67.36	26.67	.024	.878	.000	.0000	.0076
600	54.58	33.50	48.57	33.51	1.069	.303	.008	.0088	.0623
700	35.20	33.22	30.77	33.23	.592	.442	.004	.0000	.0522

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

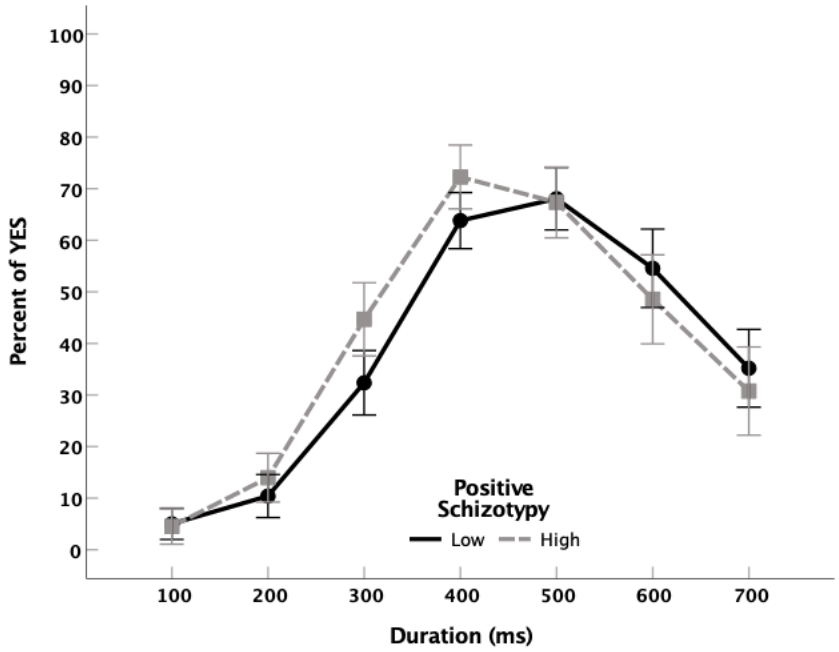

Figure 5.2: Illustration of the Duration x Schizotypy interaction in Experiment 3.

I also examined whether Schizotypy showed greater asymmetry in their temporal gradient, indicating greater precision, by taking the difference between Click Train and baseline durations for the three shortest durations (e.g., 100 – 300) and three longest durations (e.g., 500 – 700). A 2(Asymmetry [short vs. long]) X 2 (Schizotypy [Low vs. High]) repeated-measures ANOVA was conducted. A main effect of asymmetry was reported

by the ANOVA $F(1, 133) = 29.80$, $p = <.001$, $\eta_p^2 = .18$ [0000., .9047], suggesting that both groups exhibited asymmetry, giving significantly more YES responses to longer durations ($M = 30.0$; $SD = 43.8$) than to shorter durations ($M = 4.1$; $SD = 20.8$). There was, however, no significant interaction between asymmetry and Schizotypy, $F(1, 133) = .21$, $p = .65$, $\eta_p^2 = .00$, [0000., .0624] suggesting that Schizotypy level did not moderate precision, as shown in Figure 5.3. Finally, there was no main effect of Schizotypy, $F(1, 133) = 1.8$, $p = .19$, $\eta_p^2 = .13$ [0000., .3609].

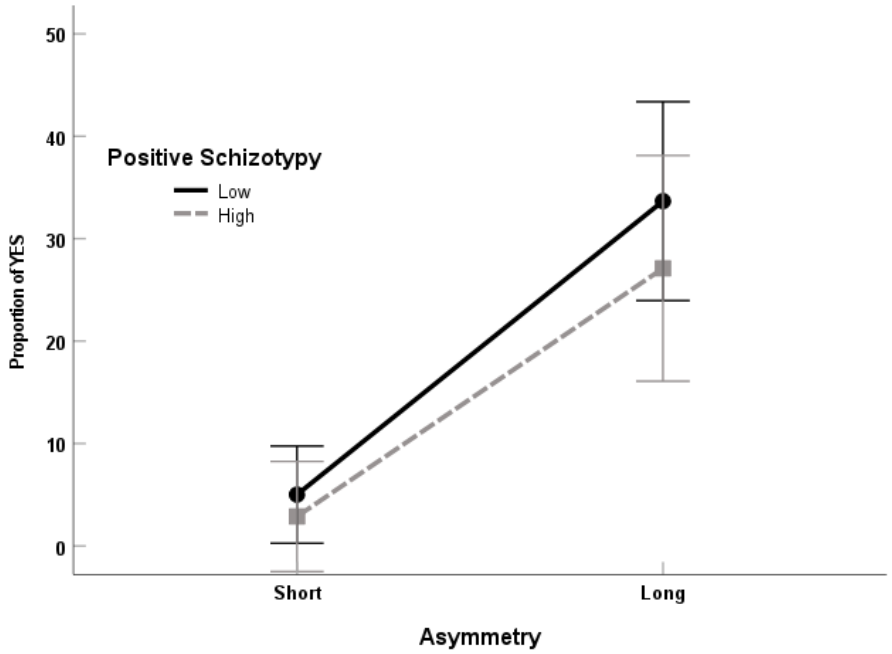

Figure 5.3 Significant asymmetry for overall short and long durations clearly showing that a higher percentage of Yes responses were made for long durations as opposed to short durations for Experiment 3.

In summary, Experiment 3 showed that for auditory stimuli, High Schizotypy shifted the temporal gradient to the left, suggesting an overall *overestimation* of durations of high compared to low schizotypy subjects, as expected from hypothesis (1) meaning that I can accept my first hypothesis that High Schizotypy would overestimate durations. however, I

failed to find evidence for hypothesis (2), since there was no difference in the shape of the temporal gradient as a result ofSschizotypy – both Sigh and Low schizotypy subjects showed the expected asymmetry consistent with precision in identifying the standard duration. Hypothesis (3) was also confirmed in that the Click Train shifted the temporal gradient to the left, as expected based on previous evidence (e.g., Penton-Voak et al., 1996). My results seem to contradict those obtained by Elvevåg et al., who in contrast to the current study found that schizophrenic patients underestimated temporal durations (while here, I find evidence for overall overestimation of durations), and evidence of a *lengthening of the standard in memory was stronger for patients* than for controls; while, here I showed evidence of lengthening of the standard in memory for both groups. Furthermore – and similar to Elvevåg et al., (2003) High Schizotypals were more accurate at identifying the standard peak than Low Schizotypals, as shown by High Schizotypy peaking closer to the standard than Low Schizotypal subjects, which is shown in Figure 5.2.

What might explain this difference between Experiment 3 and the Elvevåg et al (2003) study? One reason may be that schizophrenic patients have a different pattern of temporal processing profile than high Schizotypals (Reed & Randell, 2014). Although there may be similarities in some tasks (e.g., temporal bisection), in temporal generalisation the two groups show different performance. However, this would assume that there are distinctly different mechanisms underlying the two tasks, a notion that has not been explored by the extant literature, though, the temporal bisection and generalisation tasks share a similar psychophysical origin (e.g., Wordsworth & Schlosberg 1971) the temporal generalisation task is considered the more sensitive of the timing tasks (Wearden et al., 1997; Elvevåg et al., 2003). However, a more parsimonious explanation may have to do with the differences in the procedure and stimuli in the current study and the Elvevåg et al study. Specifically, Elvevåg et al. presented the standard duration alongside a visual stimulus (a picture of an owl). When

an event is committed to memory subjects will use all available cues at their disposal to store and subsequently retrieve the event from memory (e.g., Eich, 1980). Visual stimuli are known to be better remembered compared to auditory stimuli (e.g., Cohen, Horowitz & Wolfe, 2009). Therefore, in the Elvevåg et al study, it is likely that controls used the visual cue of the image of the owl to store and retrieve the duration more accurately, leading to higher accuracy for controls than patients at the standard duration during test, and a more symmetrical gradient for controls than the patients, indicating less distortion of the standard in memory. However, why High Schizotypal subjects should peak at the standard better than Low Schizotypals cannot be readily explained. Javitt et al. (1997) suggested precision is impaired in schizophrenia, which is not the evidence from Chapter 5 but, normal retention of auditory information is normal in schizophrenics therefore, in terms of my findings, I suggest High Schizotypals had retained the standard duration but, their precision for durations other than the standard duration was retarded. This accords with Figure 4.2 whereby Schizotypals give a higher proportion of Yes responses to durations under the standard implying they were less precise at differentiating between the standard and other durations. This implies the decision-component of *SET* or *Working Memory* components are retarded. Furthermore, Schizotypy is a measure of schizophrenic liability as opposed to a 'lesser form' of schizophrenia (Lenzenweger, 2006). These results are partially in accordance with High Schizotypy in the auditory bisection task, in that High Schizotypy also showed an overestimation of durations implying their pacemaker is emitting pulses at a faster rate. However, given the fact that in Chapter 4, I had a visual and auditory bisection task, I also report on a visual temporal generalisation task.

5.5 Experiment 4: Visual Temporal Generalisation Task

Experiment 4 examined temporal generalisation performance in High and Low Schizotypals using visual stimuli for the standard duration. I proposed that visual stimuli have

a benefit over auditory stimuli in memory as such stimuli tend to be better remembered than auditory stimuli (Cohen et al., 2009; Bigelow & Poremba, 2014; Standing, 1973). Based on this I would predict that Low Schizotypy subjects would show a more symmetrical gradient in Experiment 4 compared to subjects in Experiment 3 (where auditory stimuli was used), because there would be less distortion of the standard duration in LTM. This can be tested by examining the differences between the average short durations (e.g., those below the standard) and the average long durations (e.g., those above the standard duration), as I did in Experiment 3

Recently, Xie, Cappiello, Park, Deldin, Chan, & Zhang (2018) showed that schizotypal subjects have a *less precise working memory representation of visual stimuli* than controls, consequently, I make the following predictions: (1), that High Schizotypy will, much like Experiment 3 and Elvevåg et al., (2003) underestimate durations under the standard duration. (2), that the Click Train will lead to an overestimation of durations, irrespective of Schizotypy level, and (3) that High Schizotypal subjects will show greater asymmetry compared to Low Schizotypals in Experiment 4, indicating a larger distortion of memory of the standard.

5.6 Method

5.6.1 Subjects

A new sample of 144 naïve subjects (102 females, and 42 males) were recruited as described in Experiment 1. Subjects were between the ages of 18 and 75 (M=31.31, SD=12.29). They were awarded 4 subject pool credits or £4.50. Once again, a G-Power analysis was conducted for a mixed-model ANOVA, with two groups and two conditions, using a rejection criterion of $p < .05$, and at 90% power, for a small effect size, a sample size of 84 was suggested.

5.6.2 Stimuli & Measures

The stimulus for the training phase was a grey oval measuring 40x50 mm. For the test phase, an array of randomised rectangles (86 x 54 mm) and circles (80 mm) in either red, blue, or green were presented to subjects . The clicker used was a 144Mhz tone, that lasted for 10ms, with 190ms of silence, over 5000ms. This gave the impression of a click train that lasted for 5s. As in Experiment 1, the OLIFE (B) was used to measure self-reported schizotypy.

5.6.3 Design & Procedure

Experiment 4 was based the same design as Experiment 3. Based on the OLIFE(B) scores, there were 71 Low Schizotypy (*M*=8.86, *SD*=3.83), and 73 High Schizotypy subjects (*M = 18.92; SD = 3.32*). All other design aspects were the same as in experiment 3. Subjects completed the study online using the Gorilla experimental platform. During training, trials began with a grey oval at screen centre for 400ms –the *standard duration*. Subjects pressed the spacebar to see a comparison stimulus (a grey oval) and decide whether it matched the standard in terms of duration. Subjects pressed Z for 'No', or M for 'Yes' (counterbalanced across subjects). Feedback was given in the form of a green cross for correct, and red for incorrect responses. There were 7 training trials – one for each of the durations. In the test phase, comparison stimuli were different shapes in colour, and were presented randomly, 10 times for each of the 7 possible durations. The task was identical to the training phase – determine whether the comparison stimulus matched the standard duration, but this time no feedback was given. This training-test cycle repeated a second time, with an auditory click train presented prior to each comparison stimulus. Much like Experiment 3, the dependent variables were the percentage of Yes responses to each duration; the percentage of 'Yes'

responses for the peak and finally, the mean proportion was the dependent variable for asymmetry.

5.7 Results and Discussion

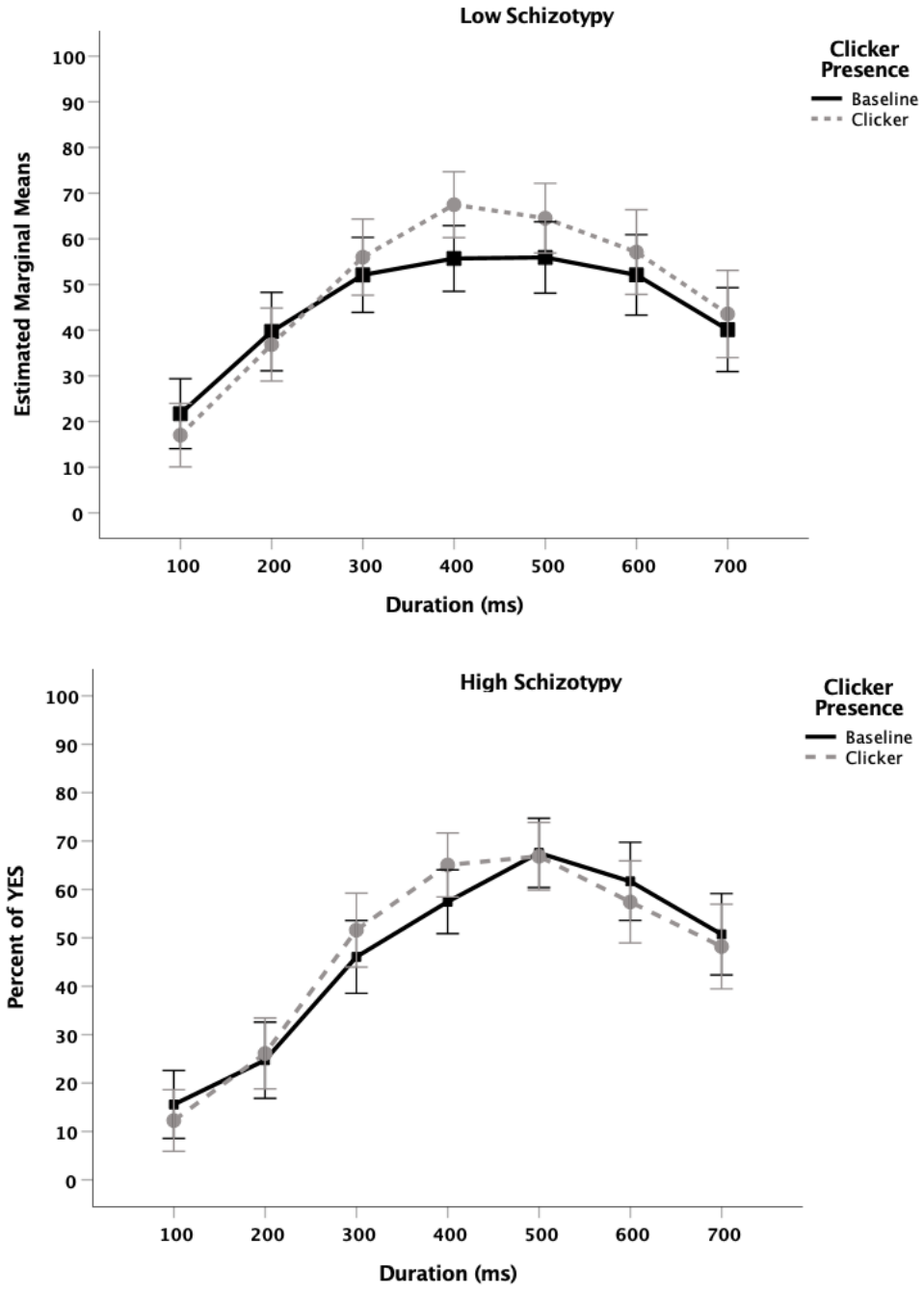

Figure 5.4 Mean percent of 'yes' responses per condition for Low Schizotypy (top panel) and High Schizotypy (bottom panel) in Experiment 4.

Thirty subjects' data (20%) were removed from the study, whose peaks corresponded to 100ms or 700ms using the same reasoning as Experiment 3. The percentage of 'Yes' responses per condition is shown in Figure 5.4 for Low (top panel) and High (bottom panel) Schizotypy subjects. A 2(Clicker Presence: baseline vs. clicker) X 7(Duration: 100, 200, 300, 400, 500, 600, 700) X 2(Schizotypy: low vs. high) mixed ANOVA with repeated-measures on the first two variables, did not reveal significant main effects of Clicker Presence, $F(1,112)=3.55$, $p=.062$, $\eta_p^2 =.031$, [.0000., .1153] or Schizotypy, $F(1,112)=.080$, $p=.78$, $\eta_p^2 =.001$, [.0000., .0293]. The main effect of Duration was significant, with more 'Yes' responses at the 400ms standard duration, $F(6,672)=58.74$, $p<.001$, $\eta_p^2 =.34$.[.0000., .7881], as was the Schizotypy X Duration interaction, $F(6,672)=2.93$, $p=.008$, $\eta_p^2 =.25$, *[.0000, .1564*]. There were no other significant interactions: Clicker Presence X Schizotypy, $F(1,122)=1.94$, $p=.17$, $\eta_p^2 =.017$, *[.0000., .0833;* Clicker Presence X Duration, $F(6,672)=2.00$, $p=.063$, $\eta_p^2 =.018$*;* Task X Duration X Schizotypy, $F(6,627)=.767$, $p=.60$, $\eta_p^2 =.007$, [.0000., .0152].

The Schizotypy X Duration interaction warrants further investigation via a simple-effects analysis; which is shown in Table 5.2 and illustrated in Figure 5.5

Table 5.2

Simple-Effects analysis examining the Duration x Schizotypy interaction

Duration (ms)	Low Schizotypy		High Schizotypy		*F(1, 112)*	*p*	Eta	LCI	UCI
	M	*SD*	*M*	*SD*					
100	19.38	22.41	13.93	22.41	1.674	.198	.015	.0000	.0852
200	38.29	25.02	25.43	25.02	7.471	.007*	.063	.0045	.1635
300	54.01	25.51	48.84	25.51	1.182	.279	.010	.0000	.0754
400	61.60	25.53	61.27	21.53	.007	.935	.000	.0000	.0026
500	60.24	22.82	67.13	22.75	2.641	.107	.023	.0000	.1017
600	54.62	27.03	59.56	27.02	.946	.333	.008	.0000	.0702
700	41.85	28.93	49.49	28.93	1.970	.163	.017	.0000	.0906

Note. Asterisks (*) denote significant differences following the simple-effects analysis.

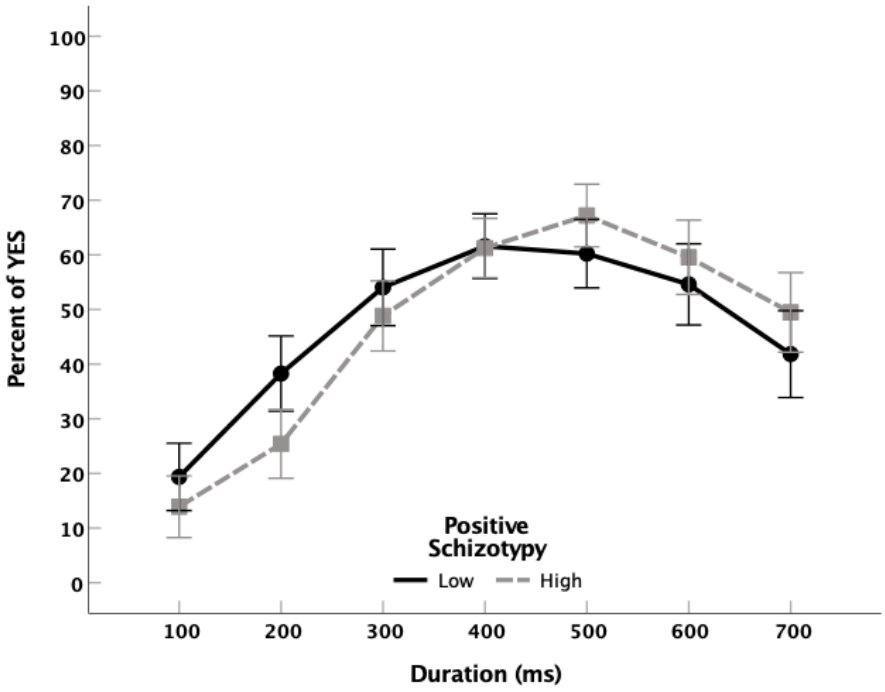

Figure 5.5 Illustration of the Duration X Schizotypy interaction in Experiment 4

Figure 5.5 suggests that the source of the significant interaction was that High Schizotypals showed a rightward shift of the gradient compared to low schizotypals, and their 'Yes' responses peaked later than the standard, however, the simple-effects analysis confirmed this result only for the 200ms duration.We further examined whether both groups showed an asymmetry in their temporal gradient by taking the difference between Click Train and baseline durations for the three shortest durations (e.g., 100 – 300) and three longest durations (e.g., 500 – 700). A 2(Asymmetry [short vs. long]) X 2 (Schizotypy [Low vs. High]) repeated-measures ANOVA was conducted. A main effect of asymmetry was reported by the ANOVA $F(1, 112) = 17.25$, $p = <.001$, $\eta_p^2 = .13$ [0000., .8668], suggesting that both groups exhibited asymmetry, giving significantly more YES responses to longer durations ($M = 23.4$*; SD* = 43.4*)* than to shorter durations ($M = .09$; *SD = 26.3)*. There was, however, no significant interaction between asymmetry and Schizotypy, $F(1, 112) = .36$, $p = .55$, $\eta_p^2 = .00$, [0000., .1193] suggesting that Schizotypy level did not moderate asymmetry as shown in

Figure 5.6. Finally, there was no main effect of Schizotypy, $F(1, 112) = 2.2$, $p = .14$, $\eta_p^2 = .19$ [0000., .4547],

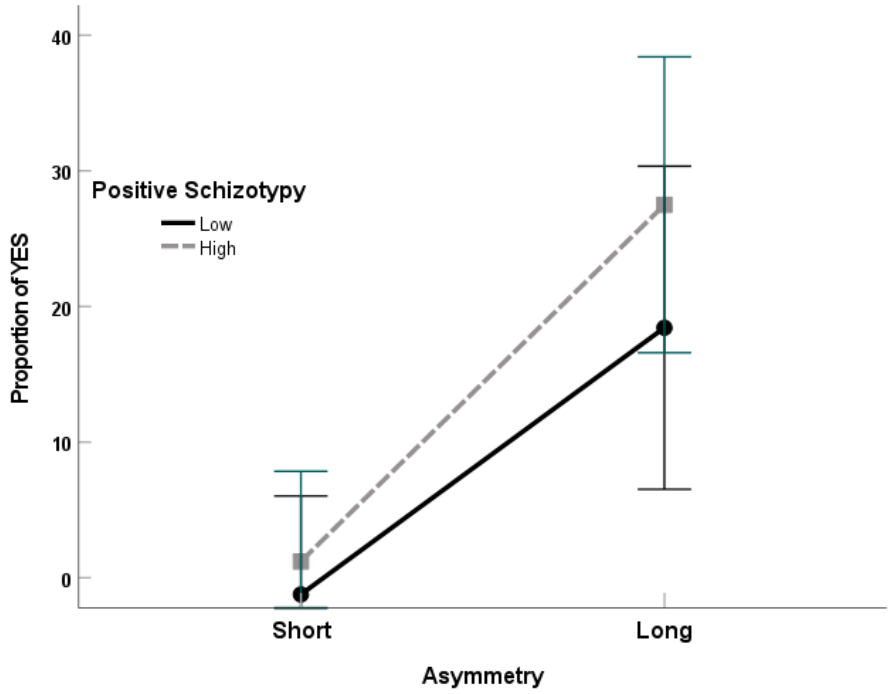

Figure 5.6 Significant asymmetry for overall short and long durations clearly showing that a higher percentage of Yes responses were made for long durations as opposed to short durations for Experiment 4.

In summary, in Experiment 4, for visual stimulus durations, hypothesis (1) was not confirmed, in that while High Schizotypals showed retarded timing in accordance with Figure 5.2 and Experiment 3, it was that they overestimated duration the 200ms duration, under the standard, meaning a reverse in the findings of Experiment 3. This finding was potentially driven by the fact that High Schizotypals identified the standard as *longer* than it was (e.g., schizotypals peaked further to the right relative to low schizotypals). My second hypothesis (2) was also not confirmed in that the Click Train did not shift the psychophysical function to the left, implying the Click Train was ineffective in leading subjects to underestimate durations. Finally, my final hypothesis (3) that High Schizotypals would show greater

asymmetry than Low Schizotypals was not confirmed demonstrating they did not have greater precision.

5.8 General Discussion

The aim of Experiments 3 and 4 was threefold. First, as Schizotypy being used as a model for examining cognitive functioning in schizophrenic individuals, the current data adds to existing evidence, to help disseminate the similarities and differences between Schizotypals and Schizophrenic individuals and more importantly, the similar aetiology they share. Specifically, the current study aimed to address a knowledge gap with regards to the time perception of schizotypals. Second, I examined the differences between visual and auditory domains in terms of how time perception may differ between the two. Finally, I investigated the effectiveness of the clicker condition in schizotypy. I now address each of these points, linearly.

The key findings across two experiments were as follows: Firstly, for the auditory modality (Experiment 3), I showed that High Schizotypals perceived durations longer than they are objectively (e.g., overestimation), as index by a leftward shift of the temporal gradient (Figure 5.2), which is compatible with the notion of a faster pacemaker within SET, relative to Low Schizotypals. Secondly, I did not show any differences in the *asymmetry* in the temporal gradient for Schizotypy, with all subjects having a higher percentage of 'Yes' responses for stimuli above (e.g., 400ms) and lower percentage of 'Yes' responses for stimuli below the standard (e.g., 400ms) compared to below the standard (Figure 5.3) meaning that Schizotypy subjects were no more precise in temporal processing compared to Low Schizotypy subjects. Finally, for Experiment 3, I showed that the Click Train led to an overestimation of durations, irrespective of Schizotypy group.

For the visual modality (Experiment 4) , I showed that High Schizotypy perceived the 200ms duration shorter than it is objectively (e.g., underestimation), as index by a leftward shift of the temporal gradient (Figure 5.5), which is a reverse of Experiment 3. This finding is potentially driven by High Schizotypy identifying the standard (i.e., 400ms) as longer than it was, compared to Low Schizotypy. I also did not find evidence for the Click Train effectiveness in shifting the temporal gradient to the left, leading to overestimation of durations, again, in contrast to Experiment 3. Finally, asymmetry was not moderated by Schizotypy level, as shown in Figure 5.6.

In the auditory domain, I explain the difference of High Schizotypy identifying the standard more accurately than Low Schizotypy (see Figure 5.2) as auditory information being normally perceived in Schizophrenics, but precision retarded (Javitt et al., 1997), which is not the finding of Chapter 4.. However, how does this finding fit to SET? During the training stage, it appears High Schizotypy encoded the standard duration in reference memory better than Low Schizotypy. Therefore, when they recognised the standard was presented, in the test stage, High Schizotypals recognised it more accurately. In terms of Javitt et al., (1999) they suggested that precision was retarded in schizophrenia. This would imply, and according to *SET,* that once the pulses accumulate from the pacemaker, they are transferred to the *working memory* component and then to the comparator. Silver et al. (2003) suggests that working memory deficits (e.g., lower capacity is a key deficit in schizophrenia; especially verbal working memory (Conklin, Clayton Curtis, Ktsanis & Lacono, 2000)) is present in schizophrenia; which were discussed in Chapters 2 and 3, respectively. This might imply that when a comparator duration reached the working memory component of *SET,* of which is retarded in Schizotypy, they are perceived as longer than they are (Pan & Luo, 2011). This accords with Figure 5.2, in that comparison durations were perceived as being closer to the standard than they really are.

However, I cannot rule out the possibility of other components of *SET* such as pacemaker, switch, and the decision process decision giving rise to this finding. Since the pacemaker and switch are presumed to be independent of modality, we would expect to see a similar set of deficits in the visual domain. This is the case however, the results are reversed (e.g., schizotypals are *less* likely to identify the standard duration than their low schizotypal compatriots) which appears to rule out the prospect of the pacemaker-switch-accumulator models interfering with the visual domain. However, since the standard duration appears to be misremembered, in this case, I suggest that that the reference memory component of *SET* is responsible for the deficit in durations. This accords with some evidence, with Smucny, Zarubin, Ragland and Carter (2020) suggesting visual memory is degenerative in schizophrenia' implying that they would misremember the standard duration. Therefore, in terms of *SET* to explain Experiment 4, one could speculate that when the High Schizotypals were trained on the standard duration, it was remembered as *longer* than it is. There is evidence suggesting that subjects with Schizotypal traits show less precise visual representations in memory (e.g., Xie et al., 2018), which could account for the contradictory set of results I reported for Schizotypy between Chapters 4 and 5, respectively

There was a main effect of Clicker Presence in the auditory modality (Experiment 3) but not the visual modality (Experiment 4), which might be compatible with a memory distortion of the standard. Specifically, in Experiment 3 (auditory durations) the Click Train presented before the comparison tone led to higher 'Yes' responses for comparison stimuli below the standard, but not for stimuli above the standard. This can be explained, if one accepts that the standard is lengthened in LTM – the durations below the standard are made perceptually longer and now match the lengthened LTM representation. The opposite pattern of influence of the click-trains on performance I noticed in Experiment 4 (visual durations) where, for low schizotypals, there is no asymmetry in the temporal gradient, suggesting no

significant lengthening of the standard in LTM. Therefore, this time the clicker leads to higher number of 'Yes' responses for durations at and above the standard – e.g., a shape lasting for 500ms preceded by a series of clicks to lengthen its perceptual representation is now (as opposed to its auditory equivalent in Experiment 3) judged as longer than the standard. This trend, although shy of significance, is compatible with the notion that duration judgments were determined by differential distortions in the memory of the standard.

In terms of the pacemaker component of SET, the results present a more mixed picture, much like the findings in Chapter 4 For the visual modality (Experiment 4) High Schizotypals showed a rightward shift of the gradient which is compatible with a slower pacemaker leading to the underestimation of time duration. In contrast, for auditory stimuli (experiment 3), there is a leftward shift compared to low schizotypals, compatible with a faster pacemaker leading to overestimation of time. It is not clear why High Schizotypals showed evidence of a slower pacemaker for the visual modality but a faster paeemaker for the auditory modality.

A further component of SET to consider would be the switch component, which is said to be mediated directly by attentional resources (Gibbon, 1977; Block and Zakay, 1997). In the context of SET, when this switch is fully opened, it allows pulses to pass from the pacemaker to the accumulator without variability; indicative of subjects paying full attention to the stimulus attributes, including its duration (e.g., Treisman et al., 1990). Evidence suggests that attentional deficits are present in Schizotypy (e.g., Lenzenweger, Comblatt & Putnick, 1991; Gooding, Matts & Rollmann, 2006). Therefore, the Schizotypal pacemaker may emit pulses at a veridical rate (e.g., the pacemaker times presented durations exactly in that if a 300ms duration is presented, the pacemaker would time this as 300ms), and as they pass to the Schizotypal switch, the switch 'flickers' which is caused by variability in attention (e.g., Meck, Church & Olton, 1984; Reed & Randell, 2014), symbiotic of attentional deficits

in Schizotypy and schizophrenia (e.g., Chen & Faraone, 2000). As a result, this would result in pulses, from the pacemaker, being 'lost' as they cross the switch, because it is flickering. This would result in fewer accumulated pulses, from the pacemaker, which would manifest as a rightward shift in the temporal gradient and thus, an underestimation of durations, which is what we found in Experiment 4 for visual stimuli (see Figure 5.4). However, in Experiment 3, the aberrant switch hypothesis can be ruled out, as the switch should somehow have to *add ticks* to High Schizotypals' accumulated ticks which makes little theoretical sense.

At present, the overall pattern of results is more parsimoniously explained as a difference between high and low schizotypals in the distortion of working memory in the auditory domain, and reference memory in the visual domain. Of course, it is possible that both pacemaker and switch have deficits (Reed & Randell, 2014) and the sum of these deficits may contribute to an underestimation of timing (e.g., Wearden, 2016), as reported in the auditory modality (Experiment 3) and an overestimation (Experiment 4) in the visual modality. Finally, the current study shows that timing deficits in Schizotypy are similar to schizophrenia however, one of the unfortunate consequences of this similar finding between schizophrenia and Schizotypy, are the contradictory findings. This has important implications for future research on schizophrenia as our results contribute to the literature illustrating a common aetiology between schizophrenia and Shizotypy. As a result of memory appearing to be the key driving force in timing deficits in Schizotypy, revealed by Chapter 5, the verbal estimation task can be used, in which both the prospective and retrospective paradigms can be used. In the final experimental chapter, I used a verbal estimation task to further explore timing deficits in schizotypy

Chapter Six: Verbal Estimation and Schizotypy:

6.1 Introduction

Chapters 4 implies that Schizotypy have better precision in timing, while in Chapter 5, I have argued the sum of working memory *and* reference memory (which are modality dependent) contribute to deficits duration judgements, somewhat contradictory results. To fully test the hypothesis that either memory deficits or better precision are the key ingredient to Schizotypy, the retrospective temporal estimation task can be used. In this task, I have used suprasecond durations as opposed to subsecond durations (see Chapter 3) due to the fact that the estimation of suprasecond durations relies heavily on higher cognitive functions, such as memory (Grondin, 2010; Hellstrom & Rammsayer, 2004; Block & Zakay, 1994, 1997) however, as I discussed in Chapter 3, Penney & Vaitilingam (2008) suggest, the comparison of critical timing appears to be quantitative as opposed to qualitative implying that the subsecond and suprasecond durations are likely served by the same cognitive processes in the brain, but manifest differently. On the basis that the subsecond and suprasecond durations are possibly served by the same cognitive process (e.g., the memory component), I would expect duration recall being driven by the memory component of SET however, how that manifested surprised me.

By studying both the prospective and retrospective paradigm, along with suprasecond durations, I reasoned that I could fully explore the memory components of SET, which are implemented in 5, respectively. Using a similar set-up to Klapproth (2007), I employed a between-subjects design, in which subjects were asked to estimate how long a visual duration lasted in either a prospective or retrospective paradigm. In both paradigms, subjects watched

a kitten video lasting for either 15s, 30s, or 45s and had to type how long they thought this video lasted for. Given the fact that memory deficits are present in Schizotypal subjects (Ettinger et al., 2015; Sahakyan & Kwapil, 2016) as well the conclusions I reached in 5, respectively, I would expect Schizotypy subjects to misremember durations in the retrospective paradigm, given there is some evidence of memory deficits in Chapter 5 and the retrospective task specifically targets memory. Few studies have examined time perception in Schizotypy (e.g., Fenner et al., 2020; Lee et al., 2006; Reed & Randell, 2014; Chapters 4, and 5), but none, to date, have explored verbal estimation in both the retrospective and prospective paradigm, respectively. As Schizotypy has been suggested as a useful model for understanding schizophrenia, it would be productive to assess these aspects of performance in those with Low and High schizotypy scorers. Therefore, I make the following three hypotheses based on my findings from Chapters 4 and 5 and from Klapproth (2007). The first hypothesis, (i), is that there will be a significant difference between the three conditions, irrespective of both Schizotypy level and paradigm, in that subjects will underestimate the 45s duration and overestimate the 15s duration in accordance with Vierordt's Law. My second hypothesis (ii) is that High Schizotypy subjects will underestimate conditions in the retrospective task, relative to Low Schizotypy subjects, in accordance with my findings in Chapter 5, which implied a memory deficit. The final hypothesis (iii), is that High Schizotypy Subjects will show greater precision for durations, given my findings in Chapter 4, where I showed Schizotypy subjects were more precise in recognising durations.

6.2 Method

6.2.1 Subjects

A total of 325 subjects (180 Females; 145 Males) were recruited via School of Psychology's subject pool and the Prolific platform. The mean age of subjects was 34.7 (*SD = 15.6*; range 18 – 79 years). There was a total of 158 subjects in the Low Schizotypy group (*M* = 6.92; *SD = 3.24)* and 167 subjects in the High Schizotypy group (*M* = 19.22; *SD* = 3.81). Those in the retrospective paradigm were naïve to the purpose of the study; whilst those in the prospective paradigm were informed that they were partaking in a timing task. Subjects were paid a total of £1.10 for their participation. A G-Power analysis, for a mixed-factor ANOVA revealed that a sample size of 172 subjects would achieve a small effect size however, given the fact that I had 6 conditions, I collected double the number of subjects.

6.2.2. Stimuli and measures

The experiment was designed in the Gorilla.sc programme. Subjects completed the experiment on their own personal computers, which controlled all experimental events, and recorded their data. Responses were made on the subject's own computer keyboard. In terms of the target stimuli, a video depicting kittens was used, in which the same video was used to create the 15, 30, and 45 second duration., depending on the randomised experimental condition. The video was preceded by a black cross, on a white screen, that was displayed for 500ms. The interstimulus interval between the cross and the video was 1500ms.

Similar to Experiments 1,2, 3, and 4, I used the O-LIFE short questionnaire, in which I calculated the overall positive Schizotypy score by taking the sum of Unusual Experiences, Cognitive Disorganisation and Impulsive Nonconformity. In this study, 158 subjects were in the low schizotypy group (mean = 6.92 $\pm$ 3.24; range 0 – 12) and 167 subjects in the high schizotypy group (mean = 19.22 $\pm$ 3.81; range 13 – 30).

6.2.3. Design

The experiment was a between-subjects design. The predictor variables were (a) the paradigm (prospective and retrospective) and (b) the duration of the kitten video (15 sec., 30 sec., and 45 sec.). The factors were randomised between subjects for a total of six conditions. Subjects were randomly allocated to one of the six conditions. The dependent variable was the estimates that subjects made to each of the three durations. For intersubject variability, the mean value was the dependent variable, and the ratio value between actual and estimated durations was the dependent variable.

6.2.4. Procedure

Subjects in the retrospective conditions were told that they were partaking in a visual perception task. Conversely, those in the prospective conditions were told that they were partaking in a time perception task. In all cases, subjects were asked to pay close attention to the video. In all cases, the subjects were presented with a set of instructions. Once they had read the instructions, they were instructed to press the spacebar to continue. Once they had pressed the spacebar, a black cross on a white screen was shown for 500ms. After the cross, a white screen was displayed for 1500ms. After the white screen, a estimation task followed. Here, the subjects were asked to estimate, in seconds, how long the video lasted. Once they had entered a value, they were instructed to press the spacebar. Subjects then completed a basic demographics questionnaire, and the O-LIFE questionnaire. All subjects received a single trial; with the experiment lasting, on average, seven minutes. Subjects in the retrospective paradigm were informed of the purpose of the study on the debrief.

6.4. Results

Similar to Klapproth (2007), the mean and standard deviation of the estimationswas calculated for each experimental condition, as well as the ratio of each condition (e.g.,

subjects' responses were divided by the actual response), where > 1 equates with an overestimation of durations, and < 1 equates with an underestimation of durations (Wallace & Happé 2008). The ratio allows us to deduce whether subjects overestimated or underestimated durations, along with precision of identifying durations. Unless otherwise stated, three-factorial ANOVAs were performed on the estimates data and ratio data. Subjects who gave estimates less than 2s were removed from the data set.

To analyse my data, a Three-Factorial ANOVA was conducted first on subject estimation times. There was a significant main effect of Condition (15s, 30s, 45s), *F(2, 301)* = 59.359, <.001, , η_p^2 = .283, [.1990., .3570], however, there was no main effect of paradigm (retrospective vs. prospective), *F(1, 301)* = 2.005, *p* =.158, , η_p^2 = .007, [.00000., .0363] or positive schizotypy (High vs. Low), *F(1, 301)* = 3.088, *p* =.080, , η_p^2 = .010, [.0000., .0437] however, I note this main effect was just shy of significance. There were no significant interactions between paradigm and condition, *F(2, 303)* = .645, *p* =.525, , η_p^2 = .004, [.0000., .0251], condition and Schizotypy, *F(2, 301)* = .192, *p* =.826, , η_p^2 = .001, [.0000., .0139], paradigm and Schizotypy, *F(1, 301)* = .059, *p* =.808, , η_p^2 = .000, [.0000., .0083],and finally, there was no significant three-way interaction between paradigm, condition and positive schizotypy, *F(2, 301)* = .106, *p* =.899, η_p^2 = .001, [.0000., .0096], confirming, as expected (hypothesis (i)), that there was a difference between the conditions (e.g., 15s, 30s and 45s) irrespective of paradigm or Schizotypy level, which is shown in Figure 6.1. however, the main effect of Schizotypy was just shy of significance, which led me to conduct an independent-samples t-test to explore this. The mean difference (*M = 4.83*) between Low Schizotypy (*M = 35.61; SD = 26.19*) and High Schizotypy *(M = 30.78; SD = 18.61*) was significant, *t(311) = 1.888, p = .030, [- .20457, 9.85893]* implying that Schizotypy subjects tended to underestimat durations, a trend that is pertinent for precision however. Given that there was no significant interaction between paradigm and Schizotypy, I am rejecting my

second hypothesis that Schizotypy subjects would underestimate durations in the retrospective paradigm.

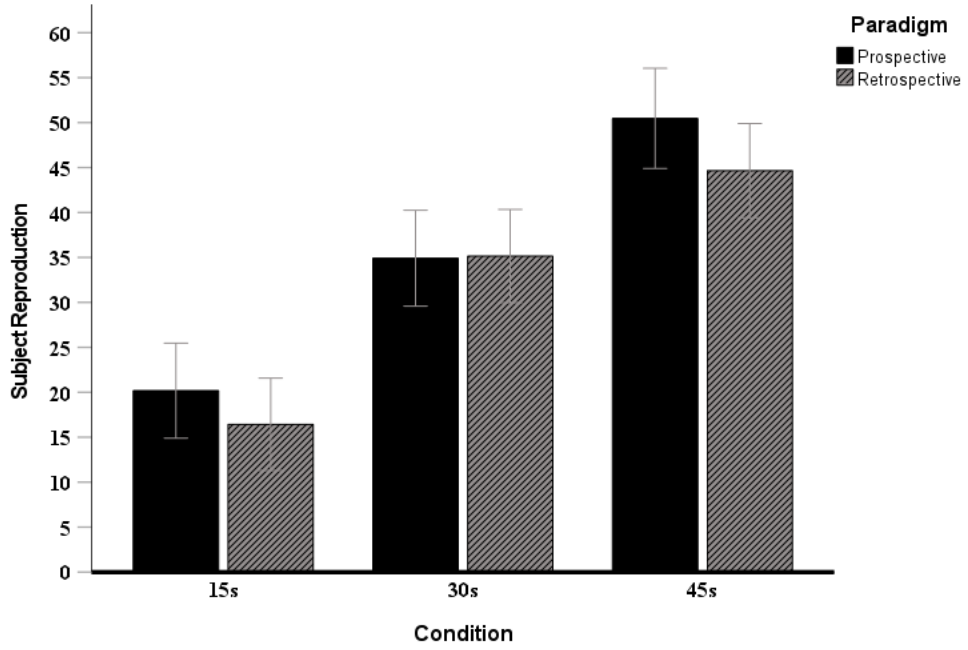

Figure 6.1: The main effect of condition, showing the main effect of each condition (15s, 30s, 45s), irrespective of paradigm (e.g., prospective or retrospective).

Whilst there appears to be a trend in Schizotypy subjects underestimating durations, the interaction is only approaching significance. A more robust analysis would focus on the ratio of durations to assess whether subjects underestimated or overestimated durations and to determine the precision of High Schizotypy on durations, I calculated the ratio of duration (Wallace & Happé 2008) by dividing subjects' estimations by the actual estimation (e.g., if the subject had estimated 10s for the 15s duration, this would result in 0.67). A value greater than 1 equates with an overestimation of durations; while a value less than 1 quates to an underestimation of durations. A three-factorial ANOVA, with ratio as a dependent variable, and condition (15s, 30s, 45s), paradigm (retrospective vs. prospective) and schizotypy (High vs. Low) was conducted. The means and standard deviations for all subjects, with respect to

ratios, are shown in Table 6.1, for Low Schizotypy in Table 6.2 and for High Schizotypy in Table 6.3. The ANOVA yielded a significant main effect of Schizotypy $F(1, 301) = 4.458$, $p = .036$, , $\eta_p^2 = .015$, [.0000., .0520] however, there was no main effect for condition, $F(2, 301) = 1.484$, $p = .228$, $\eta_p^2 = .010$ or paradigm, $F(1, 301) = 2.472$, $p = .117$, $\eta_p^2 = .008$, [.00., .0396], and no significant two-way interactions for paradigm and condition, $F(2, 301) = .910$, $p = .404$, , $\eta_p^2 = .006$, [.0000., .0299]; Schizotypy and paradigm, $F(1, 301) = .198$, $p = .656$, , $\eta_p^2 = .001$, [.0000., .0181], or Schizotypy and condition, $F(2, 301) = .683$, $p = .506$, , $\eta_p^2 = .005$, [.0000., .0260]. Finally, there was no three-way interaction between Schizotypy, paradigm and condition, $F(2, 301) = .145$, $p = .865$, , $\eta_p^2 = .001$, [.0000., .0118]. These results seem to suggest that High Schizotypy subjects are, overall, more precise (see Figure 6.2) at identifying the durations across all conditions (15s, 30s, 45s) and paradigms (prospective and retrospective), as indexed by their ratios being closer to 1 than Low Schizotypy subjects.

To explore the main effect of Schizotypy, a one-way ANOVA was conducted on the ratio with Schizotypy (Low, High) as the between-subjects factor. The mean difference $(M = .17)$ between High Schizotypy ($M = 1.06$; $SD = .53$) and Low Schizotypy ($M = 1.23$; $SD = .83$) was significant, as indicated by the main effect of Schizotypy, $F(1, 311) = 4.590$, $p = .033$, $\eta_p^2 = .015$, [.0000., .0512] implying that High Schizotypy subjects' ratio was more precise (e.g., closer to 1) than Low Schizotypy subjects. This finding can be clearly seen in Figure 6.2, in which Low Schizotypy are further away from 1.00 (1.23) as opposed to High Schizotypy (1.06). this indicates that Low Schizotypy overestimate durations overall, and that High Schizotypy underestimate durations overall, but are more precise in recognising durations, which is in accordance with the finding of Chapter 4, in which again, I showed that High Schizotypy underestimated the auditory durations, and were more precise in temporal processing.

Table 6.1: Means and Standard Deviations of Ratios

Stimulus Duration	Ratio	
	M	*SD*
Prospective Paradigm		
15 sec.	1.34	.86
30 sec.	1.16	.42
45 sec.	1.12	.37
Total M	1.21	.60
Retrospective Paradigm		
15 sec.	1.07	.63
30 sec.	1.18	1.07
45 sec.	.99	.49
Total M	1.14	.70

Table 6.2: Means and Standard Deviations of Ratios for Low Schizotypy

Stimulus Duration	Ratio	
	M	*SD*
Prospective Paradigm		
15 sec.	1.44	.97
30 sec.	1.23	.45
45 sec.	1.16	.42
Total M	1.28	.68
Retrospective Paradigm		
15 sec.	1.27	.66
30 sec.	1.28	1.38
45 sec.	1.01	.57
Total M	1.18	.95

Table 6.3: Means and Standard Deviations of Ratios for High Schizotypy

Stimulus Duration	Ratio	
	M	*SD*
Prospective Paradigm		
15 sec.	1.25	.74
30 sec.	1.09	.38
45 sec.	1.09	.33
Total M	1.15	.52
Retrospective Paradigm		
15 sec.	.92	.56
30 sec.	1.06	.58
45 sec.	.98	.40
Total M	1.06	.53

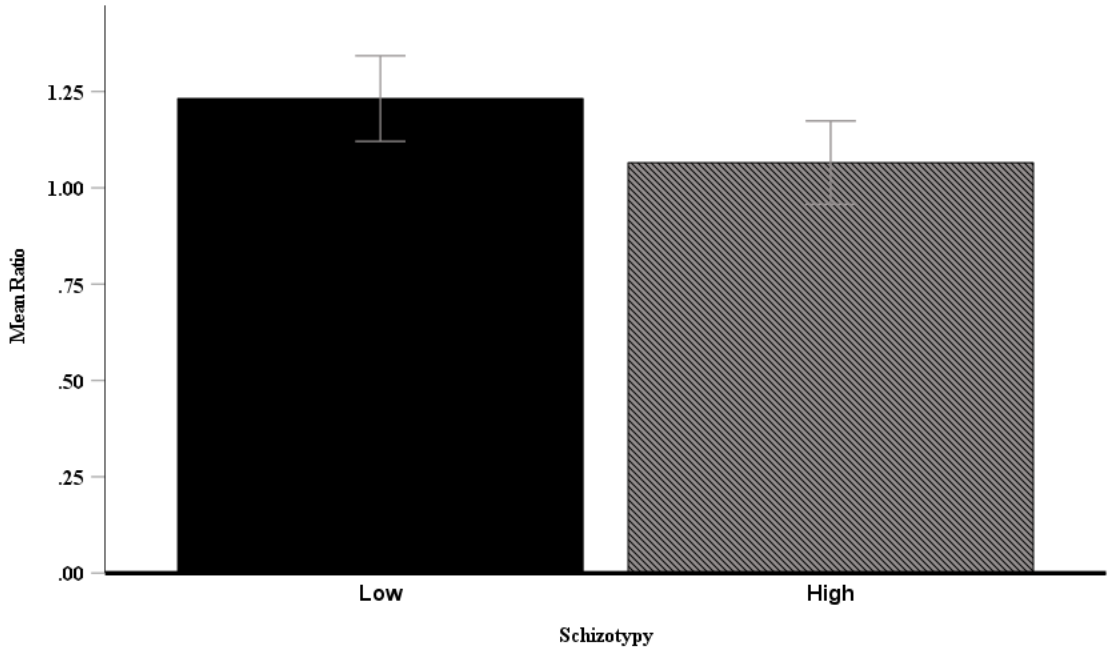

Figure 6.2: Mean ratios of Low and High Schizotypy showing that High Schizotypy had a greater degree of precision in reproducing values across conditions and paradigms.

6.5 Discussion

The objective of Experiment 6 was twofold: first, I wanted to investigate whether Schizotypy subjects are more precise at identifying durations, as indicated by Chapter 4 respectively, and secondary, whether the prospective and retrospective paradigms differ across a range of suprasecond conditions. The findings were as follows: the stimulus (15s, 30s, 45s) were significantly different from each other, across paradigms, confirming our first hypothesis (i). Secondly, there was no significant interaction between Paradigm and Schizotypy meaning that I have rejected my second hypothesis that Schizotypy would underestimated the retrospective paradigm, implying a memory deficit. However, I can accept my third hypothesis (iii), in that High Schizotypal subjects appeared to be more precise at identifying durations than Low Schizotypals.

Firstly, the finding that subjects perceived each duration differently is to be expected based on others' work in this area (e.g., Klapproth, 2007). Applying SET to this data would suggest that when the subject was presented with one of the three conditions (e.g., 15s, 30s or 45s) in the form of the kitten video, subjects' accumulators would accumulate pacemaker pulses and the accumulator would accumulate an appropriate number of pulses that accords to each of the durations (Wearden, 2016). Therefore, I accept my first hypothesis that conditions would differ from each other. Such a finding would also accord with the AGM of time perception which, apart from the attention switch, is similar to SET (Block, 1997) and of course, at a simpler level, this result is entirely consistent with the between-subject design of the study and should be expected.

Curiously, I report a lack of significant difference between paradigms reported both for estimations and intersubject variability suggesting that the divide between the prospective and retrospective paradigms are not clear cut. For example, if the prospective paradigm draws on attentional resources, and the retrospective paradigm draws on memory resources, then I would expect to see a higher level of intersubject variability in the retrospective condition as well as subjects overestimating durations in the retrospective paradigm (Block & Zakay, 1997). In terms of AGM, which how these retrospective and prospective paradigms are typically modelled (despite the critique of the model – see Lejeune 1998), one would expect the attentional gate to be 'fully opened' in the prospective paradigm, thereby allowing pulses through to the accumulator and beyond. However, in the retrospective task, since subjects are not paying attention to time, it is assumed to reflect lower levels of accuracy, as the subject is attending to the event's nontemporal (e.g., the kittens) information (Boltz, 2005).

Consequently, the attentional gate would not be fully 'open' in the retrospective paradigm, and pulses from the pacemaker could not accumulate fully (e.g., less pulses would accumulate). If this were the case, I would expect to see (a), underestimation of durations (b),

a greater intersubject variability however, for this to be the case, I would expect estimation in the retrospective condition to be *underestimated* relative to the prospective paradigm. This is not the case for either estimations or ratios (Table 6.1 and 6.2) implying that the prospective and retrospective paradigm did not alter subject duration, which was a similar finding to Boltz (2005) and Grondin and Laflamme (2015), irrespective of paradigm used. They explained their findings in terms of the relationship between temporal and nontemporal structures (e.g., prospective vs. retrospective) structures being encoded in a unitary fashion thereby allowing duration judgements to be learned, even when attention is directed to the nontemporal aspect of the task, which aligns with my findings and contrary to those of Klapproth (2007). Therefore, my data would indicate that the retrospective and prospective paradigms are not served by different mechanisms (Klapproth, 2007), which is a similar conclusion by Grondin and Laflamme (2015) and Boltz (2005) who, similar to this study 6, showed a naturalistic event on a video however, more detailed research is required between the propspective and retrospective paradigms. Applying *SET* to my data, it would be presumed that both retrospective and prospective durations accumulate the same number of pulses, which would be stored in working memory for the prospective paradigm (because subjects would partake in chronometric counting – Wearden, 2016) and reference memory for the retrospective durations (because subjects are drawing on pacemaker pulses that must be drawn *from* memory.

Why my results should deviate from Klapproth (2007) is interesting, given that my experiment was, apart from a kitten video, identical. Firstly, the Klapproth study had a total of 72 subjects whilst I had 325. From a statistical power point of view, it could be argued Klapproth's study was underpowered, which could potentially lead to a misleading positive result (Blake & Gangestad, 2020). Of course, I am not saying that my results are the correct ones and Klapproth's incorrect, but I cannot discount the possibility that their study was

underpowered. However, a more parsimonious explanation would simply be that there are no differences between the prospective and retrospective paradigms, as shown by Boltz (2005), and is the explanation I am arguing for.

My final finding is one of most interest, in that schizotypal subjects have a greater degree of precision in reproducing the durations in all conditions, as opposed to their low schizotypy compatriots (see Figure 1 and Tables 5 and 6) which is contrary to what was founded by Ueda, Maruo, and Sumiyoshi, (2018), who found that schizophrenia was related to inaccuracies in duration judgement. This finding demonstrate that Schizotypy did moderate estimates. In terms of rations, a ratio of 1 is indicative of a perfect estimation of durations (Wallace & Happé 2008), where values < 1 accord with underestimate and values > 1 accord with overestimation of durations. As can be seen from Tables 6.2 – 6.6 , all subjects overestimated durations, however, schizotypal subjects were closer to 1 than their non-schizotypal compatriots. In line with SET, this would imply that emitted pulses, from the High Schizotypy pacemaker, were closer to the actual duration than Low Schizotypy implying they are more precise (less variable) in identifying duurations, which is a similar finding in Experiment 2 (temporal bisection – auditory durations). Alternatively, High Schizotypy memory is more precise for durations (e.g., they remember durations better) than in Low Schizotypy..

Given the robust finding that schizotypal subjects exhibit memory deficits (Ettinger et al., 2015) I can rule out the reference memory component of SET contributing to greater precision of remembered durations, as the opposite effect should have been observed (e.g., subjects should have had a lower degree of accuracy). Instead, the focus should shift to the 'switch' component of SET (Gibbon, 1977). When the switch is fully open, pulses can accumulate veridically from the pacemaker to the accumulator. A variable switch (e.g., that 'flickers') leads to pulses being lost and subjects underestimating durations (Wearden, 2016).

Switch variability can often be explained in terms of attentional resources but since schizotypal subjects exhibit attentional deficits (Cornblatt & Keiip, 1994), I would expect less precision, as schizotypy subjects should be paying *less* attention to the durations. Therefore, a more parsimonious explanation might focus on the pacemaker component, of SET (Gibbon, 1977), which is often thought to be driven by an arousal mechanism (Treisman, 1963; Wearden, 2016). For example, the more aroused the pacemaker is the more pulses it should accumulate (Reed & Randell, 2014). This might explain the greater degree of precision in my findings, in that the Low Schizotypy group exhibited the typical variability in timing, that is to be expected when applying SET (Wearden, 2016) however, due to increased arousal in the High Schizotypy pacemaker, the pacemaker was emitting a greater number of pulses than the Low Schizotypy group. There is evidence that accords with this line of reasoning in that paranoid schizophrenics have increase emotional arousal to stimuli (Haralanova, Haralanov, Beraldi, Moller & Hennig-Fast, 2011), which is consistent with the positive symptoms of schizotypy (Reed & Randell, 2014). Consequently, schizotypals were more precise in reproducing durations due to a heightened level of arousal. This heightened level of arousal would then lead to the pacemaker emitting a *greater* number of pulses.

Despite the findings that High Schizotypy are more precise at reproducing durations, due to heightened pacemaker arousal, there are issues with this denouement. For example, I cannot rule out, for certain, that other components of SET were responsible for a greater degree of precision in high Schizotypy, or that each component of the system contributed to a greater degree of precision. I also note that the stimuli used (e.g., a kitten video) could have contributed to the finding and modified both attentional and memory processes associated with time perception. Therefore, any future study should utilise neutral stimuli to further delineate the finding of why schizotypal subjects should perceive durations more accurately than non-schizotypal subjects. Finally, there is the issue of using suprasecond – as opposed to

subsecond – durations. This leads to the question whether schizotypal subjects are *more* accurate at estimating suprasecond durations, as opposed to subsecond durations.

In conclusion, study 5 has replicated some well-known findings in the literature: that subjects perceive different durations differently. Surprisingly, I have found that the retrospective and prospective paradigms do not appear to be driven by different mechanism, contrary to Klapproth (2007) though, this conclusion needs substantiating with further experimentation. Finally, I have found that High Schizotypy subjects are more precise at reproducing durations than Low Schizotypy subjects, which I have explained in terms of an aroused pacemaker in High Schizotypy, similar to in Chapter 4.

Chapter Seven: General Discussion

"Time is relative; its only worth depends upon what we do as it is passing".

Albert Einstein

In terms of the objective of this thesis, I set out to explore what drives timing deficits in Schizotypy by applying SET to a range of timing tasks.

Chapter summaries:

Experiment 1 & 2 Precision and Schizotypy:

The first experiment was exploratory in identifying which components of SET were responsible for timing deficits in positive schizotypy. To explore the roles of individual components in SET, two experiments – a visual and auditory temporal bisection task – were conducted in the subsecond duration, with a baseline and clicker condition, respectively Behaviourally, three key findings emerged from this study: that the clicker shifted the psychophysical function to the left in both Low Schizotypy and High Schizotypy in both visual (Experiment 1) and auditory (Experiment 2) modalities; secondly all subjects overestimated visual durations compared to auditory durations. Finally, High Schizotypy subjects showed a pattern consistent with showing greater precision for auditory durations The results are interpreted as suggesting that High Schizotypal subjects have a greater degree of precision however, I note that the significant 3-way interaction of Experiment x Schizotypy x Duration, reported in Chapter 4 has a very small effect size and must be interpreted with caution.

Experiments 3 & 4: Temporal Generalisation Gradient Dependent on Schizotypy Level

The second experiment sought to replicate the findings of Experiments 1 and 2 (Chapter 4) but, using the temporal generalisation task. This was a world-first in that a temporal generalisation task has not been conducted on positive Schizotypy. Once again, a baseline and Click Train condition were used for both a visual and auditory temporal generalisation task. hree key findings emerge: the first was that in the auditory domain, High Schizotypy subjects overestimated durations compared with Low Schizotypy subjects, consistent with lengthening distortion of the standard duration in memory. The second finding was that that the Click Train influenced durations in Experiment 3 (auditory durations), by lengthening durations but not visual durations. The final finding is that there were no differences in asymmetry effects between Low and High Schizotypy subjects. I interpret these results as High Schizotypy subjects utilising visual cures to prevent information loss in memory for visual durations. Therefore, the result suggest High Schizotypy subjects have memory deficits, resulting in a similar aetiology between schizotypy and schizophrenia, in the context of time perception.

Experiment 5: Temporal Estimation and Schizotypy

To further test the findings of Experiments 1, 2, 3 and 4, I conducted an estimation task. This time, a between-subjects design to investigate the paradigm (prospective vs. retrospective) among three conditions (15s, 30s, 45s). Two key findings emerged: the first was that subjects perceived stimulus durations differently, which of course, would be expected, given the between-subject nature of the experiment, and the finding of Klapproth (2007). Finally, I found that High Schizotypy subjects appear to be *more* precise in estimating

durations, implying that Schizotypy levels do moderate estimation. Overall, this experiment did not reveal a memory deficit in schizotypy which is in direct contradiction with Chapters 4 and 5, respectively. However, the finding that High Schizotypy are more precise for reproducing durations is similar to the findings across Experiments 1, and 2 respectively.

7.1 General interpretation of the results

A key aim of this thesis was to establish which component of SET contributed to timing deficits in Schizotypy, with a focus on the components of SET due to the literature on schizophrenia and schizotypy. A secondary aim was to establish whether a Click Train could be used to manipulate the perception of time in High Schizotypy subjects, at least in the temporal bisection and generalisation tasks. Experiments 1, 2, and 5, are interpreted as resulting in greater precision of durations. Experiments 3 and 4 are indicative of deficits in the short-term memory component of SET, in High Schizotypy. Experiments 1 and 2 demonstrate this precision within the auditory modality of the temporal bisection task, in which High Schizotypy subjects' temporal gradient was steeper in the auditory domain, indicative of greater precision. This is an interesting – and unexpected – finding that High Schizotypy should have greater precision, as shown in Experiments 1, 2 and clarified in Experiment 5, respectively. Finally, consistent with the literature, I showed that the introduction of a Click Train leads to subjects overestimating durations, irrespective of modality or schizotypy level. Overall, I would suggest that the evidence presented in this thesis supports the finding that High Schizotypy is associated with a greater precision of durations, especially in the context of temporal bisection and estimation tasks. Furthermore, given that the Click Train can be used to modify time perception in Schizotypy, I would suggest that further research is conducted on the effect the Click Train has on Schizophrenic patients. In terms of the findings of Experiments 3, and 4, these indicate that High Schizotypy subjects show deficits in the short-term memory component of SET, which is not what the

evidence from Experiments 1,2 and 5; demonstrating how limitations (e.g., domain, task etcetera) might have given rise to the results I report.

7.1.1. Theoretical interpretations – Schizotypy

There are many behavioural observations from our experiments that can be interpreted in the context of SET. The first such interpretation is that of the pacemaker-switch-accumulator (herein known as the 'clock'). In Experiment 2 (auditory temporal bisection), High Schizotypy subjects have higher precision, implying that there is less variability in temporal processing. There could be numerous reasons for this, including High Schizotypy having a less variable pacemaker than Low Schizotypy subjects, which is indicative of greater precision (Wearden et al., 1999) however, this finding contradicts findings within Schizophrenia, that explain the pacemaker as emitting pulses with greater variability (Thoenes & Oberfeld, 2017). Using the reasoning from Triesman (1963) that the pacemaker is an arousal-driven component, it could be argued that High Schizotypy subjects have higher levels of arousal than Low Schizotypy subjects. There are few studies which have directly measured arousal and schizotypy, with one study showing that Schizotypy was associated with an increased heartrate within a social context (Premkumar et al., 2021) however, the schizophrenia literature appears to suggest that schizophrenics are hyper-aroused (Nakamura et al., 2003); which is indicative of the schizotypal pacemaker emitting a greater number of pulses (Treisman, 1963) and potentially less variable pulses If this were the case, with respect to Experiment 2, it would explain the finding that High Schizotypy subjects are more precise in temporal processing, given that they are hyper-aroused. There is evidence for more precise temporal processing in Chapter 4 for baseline conditions shows that High Schizotypy have (albeit not statistically significant) lower Difference Limen values than Low Schizotypal subjects though, this value is lower in the visual duration, indicating they are more precise (less variable) in auditory durations than visual durations. This explanation

would also explain the findings in Experiment 6, in which High Schizotypy had greater precision in the Temporal Estimation task. Once again, when High Schizotypy Subjects were presented with the kitten video to remember, their pacemakers were less variable in temporally processing the duration of the videos, leading to greater precision, as indicated by the ratio analysis.

The second component in the clock mechanism is the so-called 'switch'. When a subject is paying full attention to durations, the switch is 'closed' allowing pacemaker ticks to travel along to the accumulator without external variability (e.g., variability arising from the pacemaker only) however, if there were variability in the switch, it would manifest as a 'flicker' (Wearden, 2016) meaning that pacemaker pulses would get 'lost' as they travel towards the accumulator thereby meaning subjects would underestimate durations. There is good evidence to suggest this is the case in schizophrenia and schizotypy (Gooding, Matts & Rollman, 2006); with attentional deficits present in schizotypy, especially for shifting attention (Steffens, Meyhofer, Fasbender, Ettinger & Kambeitz, 2018) in positive schizotypy. However, for the switch interpretation to be correct (and presuming there is a single timing system for both auditory and visual modalities), Schizotypy Subjects would have to underestimate durations in both modalities (visual and auditory) and all experiments, which is not what we report in experiments one, two, three and four, or five. Instead, I report them underestimating the 200ms – 400ms durations in Experiment 2 only.

For example, in the auditory temporal generalisation task, I showed that schizotypal subjects overestimate durations but underestimate durations in the visual modality. This would imply that the switch somehow 'adds' pulses to the durations in the auditory modality, which makes little sense unless one contends with the idea that there are visual and auditory timing mechanisms (Wearden, 2016). Since the implication of the memory component in timing deficits seems to arise from experiments 3– 4; whilst the pacemaker is implicated in

Experiments 1 – 2, I conducted a temporal estimation task. The results from this task were not what I predicted, on the basis of the previous experiments. For example, I predicted that schizotypal subjects' estimation would be underestimated in the retrospective paradigm however, such a condition rested on the finding the prospective and retrospective adhered to previous findings. However, a surprising result emerged in that (i) the retrospective and prospective paradigms could not be considered different and (ii) that schizotypal subjects were more accurate across estimations. I have explained these results by arguing that schizotypals have a higher level of arousal which, according to the SET hypothesis, drives the pacemaker; which appears to be the case in Experiment 5. Though, of course, there are caveats attached to this finding in that the other components of SET could account for this finding.

Therefore, an explanation which focuses on the long-term memory component (Reference memory) might be a more appropriate and parsimonious consideration, as ultimately, both the bisection and generalisation tasks are memory tasks in nature, in that the subject is asked to remember durations (e.g., anchors in the temporal bisection task; the standard in temporal generalisation). For example, the subject is asked to remember a duration, and compare this remembered duration to comparison durations. The only difference being that in the bisection task, there are two anchors to remember, whilst in the generalisation task, there is a single anchor to remember. Starting with the temporal generalisation task, I showed that schizotypal subjects overestimate auditory durations. I interpret this as indicative of schizotypal subjects having misremembered the 400ms standard duration, which would result in the standard (400ms) being remembered as shorter (350ms) and so forth. This would result in the temporal gradient shifting to the left (e.g., greater proportion of YES responses) which is precisely what I have found in my study. It is difficult to compare this finding with other studies; as only Elvevåg et al. (2003) provides an analogue

however, their study utilised a visual cue with auditory durations meaning that subjects could utilise visual memory cues to aid their recall of the duration. To address this issue, Experiment 3 utilised auditory durations; and demonstrated that High Schizotypal subjects exhibited a rightward shift in temporal gradient; indicative of an underestimating of durations. To explain this result, it is known that visual memory tends to be richer and more accurate than memory for auditory events (Gloede, Paulauskas & Gregg, 2017); therefore, in terms of the visual generalisation task, Low Schizotypals utilised a richer memory representation of the standard duration which contributed less to distortion of the standard in LTM. For High Schizotypy, it is known that subjects scoring high with schizotypal traits are less precise in visual representations of memory (Xie et al., 2018). Therefore, since the high schizotypals are less precise in their visual memory representations, they will misremember the standard meaning that durations will be remembered as longer (e.g., the standard of 400ms would be remembered as 450ms). In summary, the findings of the temporal generalisation task suggest a working memory deficit is present in High Schizotypy subjects, mediated by the modality (e.g., auditory and visual durations) as well as schizotypy level.

In terms of the temporal bisection task data, SET can be used for a theoretical approach. The critical finding is that High Schizotypy subjects showed a pattern higher precision of temporal processing; with them underestimating auditory durations at shorter durations (200ms and 300ms) and then, switching to overestimating durations from 500 – 800ms, compared to visual stimuli, resulting in a steeper curve. Overall, High Schizotypy subjects underestimated durations in the auditory domain. In terms of explaining this in SET, one would be tempted to suggest the pacemaker is emitting pulses at a higher rate in High Schizotypy subjects, and at a less variable rate; of which there is evidence for in Experiments 1, 2 and 5. The same reasoning applies to the switch; in that if the switch was retarded in schizotypal subjects, one would expect the deficit to be recorded in both auditory and visual

durations, ruling out the switch component. The working memory (WM) component of SET is also of interest, given the results of the temporal generalisation task. High Schizotypy subjects, according to Experiments 3 and 4 , remembered the short anchor as shorter than it was, and the long anchor as longer than it was, which would explain why the 'switchover' occurs. This accords with findings by Javitt et al. (1999) who alludes to the fact that there are distortions in auditory memory, which, when Schizotypy is added to the mix, exasperates those deficits. Therefore, in terms of why Schizotypy subjects should show deviant timing in auditory durations, compared to visual, suggests that a memory deficit is the culprit. Of course, it is possible that the clock mechanism is retarded in schizotypy, but the overall pattern of results is more parsimoniously explained that High Schizotypy subjects are more precise in temporal processing in Experiments 1, and 2 (Temporal Bisection – see Chapter 4) and Experiment 5 (Temporal Estimation – see Chapter 5). However, the results for the Temporal Generalisation task (Experiments 3, and 4) seem to be explained by a deficit in Working Memory in terms of the standard duration.

The data from both the temporal generalisation and temporal bisection tasks appear to suggest that duration judgements are moderated by schizotypal level and explained in terms of reference memory deficits. However, are these results generalisable to schizophrenia? Lenzenweger (2006) would suggest that schizotypy *is not* a watered-down version of schizophrenia but rather, that schizotypy is measure of schizophrenic liability in subjects who score high on this scale. For example, one who has a High Schizotypy score is not guaranteed to develop schizophrenia; but has a higher chance of developing schizophrenia than one who has a low schizotypy score (Claridge, 1997). Some authors also interpret the findings from temporal bisection and temporal generalisation tasks as demonstrating reference memory deficits in schizophrenia (e.g., Elvevåg et al. (2003) and Carroll et al. (2008); who both reported temporal auditory deficits in schizophrenia). However, the findings in the Temporal

Bisection task and Temporal Estimation task suggest High Schizotypy subjects have a higher precision in temporal processing; whilst the memory deficit makes sense in terms of the Temporal Generalisation task. Since memory deficits are a core aspect of cognitive dysfunctionality in schizophrenic patients (Guo, Ragland & Carter, 2019), it would appear that timing deficits in schizophrenia are driven by memory deficits, as indicated by the data from High Schizotypy subhects in Experiments 3, and 4.

However, despite my arguing that the memory component of SET is potentially responsible for timing deficits in Schizotypy, such a finding does not accord with the findings in Experiment 1, 3 and 5 unless the argument was that the memory 'deficit' in schizotypy manifests as *better* memory in suprasecond durations Whilst there are no shortages of articles arguing that schizophrenic patients have *better* memory, the differences are usually discussed in the between-subject factor of medication *between* schizophrenic groups (Dong et al., 2020). I am not aware of any studies which suggest that Schizophrenic patients have better memory over and above non-schizophrenic subjects, which makes the argument of reference memory troublesome if continuity is to be maintained across timing studies. Therefore, whilst the memory deficit is driving timing deficits the temporal generalisation tasks, it does not appear to be driving the deficits in temporal bisection and temporal estimation tasks and, given the diversity of SET, many other components could be driving timing deficits. For example, both working memory and decision-making are affected by Schizotypy (Park & McTigue, 1997; Wout & Sanfey, 2011) which are two components of SET (see Chapter 2). It is also conceivable that one or all of these components of SET might give rise to variability in timing however, as argued, it appears to be more parsimonious to suggest the reference memory component is, at least, partially responsible for timing deficits in schizotypy with the experiments also suggesting an overaroused pacemaker might be culpable. Overall, the results seem to depend on the tasks used and whether subsecond or suprasecond durations are

used. Evidence, however, suggests (Grondin, 2010) that the suprasecond and subsecond durations draw on different mechanisms but, given the fact that reference memory seems to distort subsecond durations, I expected the same component to distort suprasecond durations in Chapter 6, which is not the case. Overall, it appears that High Schizotypy subjects exhibit greater precision in timing that Low Schizotypy (Experiments 1, 2 and 5), and memory deficits (Experiments 3 and 4).

7.1.2. Theoretical interpretations – Click Train

My findings of the effect of the Click Train on durations is mixed. For Experiments 1 (visual durations), I showed that the clicker overestimated durations whilst, in 2 (auditory durations), I showed that the clicker had no main effect. Conversely, in Exerpiemnts 3 (auditory durations), I showed the Click Train led to an overestimation of durations; whilst in 4 (visual stimuli), I showed the Click Train had no main effect. One of the most common theoretical interpretations of the effect of the Click Train on durations is that it has an arousing effect on the pacemaker (Wearden, Win & Philpott, 1999). In terms of Experiment 3 (temporal generalisation – auditory durations), the Click Train was significantly effective in leading subjects to overestimate durations however, in Experiment 4 (temporal generalisation – visual durations), the Click Train was not effective is leading subjects to overestimate durations. Our results partially replicate the finding by Penton-Voak, Edwards, Percival & Wearden (1996). Their interpretation was that the Click Train changes the subjective length in a manner that is broadly indicative of increasing the pulse-rate of the pacemaker (Penton-Voak et al., 1996). The consensus is that the pacemaker is an arousal-driven mechanism (Treisman, 1963, 1990), as discussed in Chapter 2, and our results are indicative of that consensus, at least in Experiment 3 Furthermore, since it is reported that the Click Train appears to work in both modalities, (e.g., auditory and visual modalities), this provides further impetus that there is a master pacemaker (Penton-Voak, 2006) that drives timing

though, I must stress that I report only a trend in the auditory domain of the temporal generalisation task, which is in contradiction to Poole, Lees & Jones (2020) however, they did not utilise either a temporal bisection or temporal generalisation task. Despite the reasoning, there remains a contradiction in that the Click Train was effective in leading subjects to underestimate durations, in the auditory domain in the temporal generalisation task (Experiment 3), but ineffective visual domain (Experiment 4).

Experiments 3 and 4 had a single standard duration (400ms) in which the subject is constantly comparing to comparison durations. It could be, at least when the duration matches the Click train (in which the Click Train matches the modality of the durations) the Click Train gets 'integrated' into the auditory durations (Wearden, Win & Philpott, 1999) due to the Click Train being of the same modality and frequency as the duration (as was the case in Experiment 3). However, a more straightforward answer is that the Click Train only weakly 'speeds-up' the clock (Penton-Voak et al. 1996), as opposed to pharmacological interventions (Wearden, 2016). This would accord with my findings in that the Click Train was significant in the Auditory-Auditory domain (Experiment 3) but not significant in the Visual-Auditory doman (Experiment 4), as the pacemaker is aroused only slightly by the Click Train. This is broadly similar to the argument presented by Penton-Voak et al. (1996); who contended that whilst the Click Train can manipulate the pacemaker pulse emittance, it does so only weakly. However, I cannot rule out the possibility of other individual differences mediating Click Train effectiveness in the auditory-auditory domain (Experiment 3), that we have not measured (e.g., depression, anxiety etcetera) and furthermore, the temporal generalisation task is not easily amenable to theoretical models, such as SET (Wearden, 2016). The implication of this is that SET might not be able to explain the findings in Experiments 3 and 4 consistently.

In terms of the temporal bisection experiments (Experiments 1 and 2) there are, once again, mixed results, in which Experiment 1 (visual durations) led to an overestimation of durations yet, there was no main effect of Click Train in Experiment 2 (auditory durations), though, I note there was a singnifcant interaction between Click Train and durations at the 400ms duration, in which subjects underestimated the 400ms duration in the Click Train condition (see Figure 4.5). Once again, I interpret these findings as the Click Train arousing the master pacemaker and lengthening durations (Wearden, Win & Philpott, 1999). However, in terms of the auditory bisection task (Experiment 2), I explain the interaction for duration and Click Train with a smaller effect size for auditory stimuli than visual stimuli (Penton-Voak et al., 1996). There are numerous reasons for this however, one potential reason is that because the tone of the Click Train and the tone of the duration was the same frequency, the arousal mechanism of the pacemaker did not interpret the Click train as 'novel' as the comparison stimuli. This would fit with my finding, in that the Click Train yielded a main effect of duration because it was novel, compared to the duration (visual) however, in the auditory modality, its novelty was not strong enough to drive a main effect but was strong enough to interfere with individual durations (e.g., the 400ms duration – Figure 4.5). Evidence which accords with my interpretation is to be found in Wada et al. (2003), who reported the influence of audition on judgement of visual frequency. Therefore, in terms of the stronger effect size of the Click Train in the visual modality, it could be argued that the presence of an auditory Click Train influenced the judgement of visual stimuli stronger than in the auditory-auditory durations, which is what I find in the visual modality of Experiment1 and the auditory modality of Experiment 2. Therefore, Click Train effectiveness appears to be dependent on the task used; with temporal generalisation yielding antithetical results, compared to temporal bisection. Of course, the nature of my experiments being online also raises the question of whether the same affect could be found in a traditional face-to-face

experiment. Finally, where the Click Train was effective, it was effective, irrespective of Schizotypy group (e.g., it worked or did not work equally well, irrespective of Schizotypy group). This implies that the Click Train could be used to manipulate the perception of time in schizotypy subjects, and possibly schizophrenia. This method should be investigated in Schizophrenia for a definitive answer.

7.3 Limitations, methodological implications, and improvements

With respect to the individual experimental chapters, there are limitations associated with each. The first involves the online nature of the experiments, the second involves the tasks themselves, the third the theoretical model we have applied (e.g., SET), the fourth is auditory versus visual durations and the final is schizotypy itself. Each of these limitations shall be discussed in turn.

Many of these differences have good explanations. For example, the differences between online and offline experiments are often negligible but, the issues associated with psychophysical experiments warrants discussion as the limitation does have implications for this thesis. The temporal bisection and temporal generalisation tasks can each be used to measure different cognitive components. Both for time perception, but temporal generalisation directly measures memory (Wearden, 2016) though, once again, they are two distinct tasks, each with their limitations. Then there is the issue of the temporal estimation task, which is notoriously difficult to model, theoretically (Wearden, 2016), with no models satisfactorily modelling the data. Furthermore, the finding that auditory durations are overestimated, compared to visual durations (Wearden, 2016) also gives rise to limitations of the studies, as does the concept of schizotypy. Therefore, it is important not to overstate our results and not to generalise without considering these limitations and considering further experiments on schizophrenia, as opposed to schizotypy. Finally, since there are many

methods in deriving variables of interest from both temporal bisection (Reed & Randell, 2014) and temporal generalisation tasks (Wearden, 2016). In my opinion, a robust mathematical approach would be more appropriate.

7.3.1. Online experiments

The first limitation is associated with the fact that, due to the pandemic, I had to conduct my studies on-line, (Groilla.sc). One key weakness to this approach is that I did not have complete control over the study and thus, subjects could have (a) been distracted by external stimuli (e.g., a cat meowing) and (b) not been paying full attention to the task. Furthermore, a pertinent question that arises is what noise online experiments contribute to the data (Chetverikov & Upravitlve, 2016); which is a specific problem with studies that measure response times or display subsecond durations due to monitor refresh rate. Whilst Chetverikov & Upravitlve (2016) did not show any detrimental effects for online experiments; Al-Salom & Miller (2019) showed that computerised platforms are more prone to invalid responding than in-person experiments however, this is not the experience of my studies. Of course, there are arguments that suggest the ecological validity of an online experiment is increased; as well as the cost-effectiveness and quicker data collection (Kuroki, 2021). Therefore, it is not impossible that the online nature of my studies contributed to some of the variance associated with our findings, which might explain the contradictory findings found in the Temporal Generalisation task (Experiments 3 and 4). Therefore, it would be interesting to add ‘experiment type’ as a between-subject factor in any future analysis for schizotypy/schizophrenia to compare findings between offline and online studies and to determine what amount of variance is contributed by online experiments.

7.3.2. Temporal Bisection, Temporal Generalisation and Estimation

As discussed, at length, in Chapter 3, there are differences between temporal bisection and temporal generalisation tasks. One theoretical difference is that temporal generalisation is difficult to model with SET (Wearden, 2016) and consequently, SET might not be an appropriate model to apply to Temporal Generalisation data. In my findings, Experiments 1, and 2 (temporal bisection) showed High Schizotypal subjects were more precise in temporal processing, as shown by an underestimation of (200ms – 400ms) durations in the auditory modality, explained by a less variable pacemaker. However, for the temporal generalisation task it appears to be a working memory deficit that drives the results, in which High Schizotypy underestimated durations in the visual modality of the temporal generalisation task and underestimated durations in the auditory modality of the temporal generalisation task. At first glance, these findings would suggest that schizotypal subjects show memory lengthening in both tasks however, if this were the case, High Schizotypy subjects would underestimate all durations, which is not the case of the simple-effect analysis in Experiments 1 and 2.. Previous experiments (e.g., Elvevåg et al., 2003; Carroll et al., 2008) have shown similar findings in both modalities; with results that are similar to my (e.g., underestimating durations). One of the key differences is that the temporal generalisation task trains subjects on a specific standard (e.g., 400ms); whilst the temporal bisection task trains subjects on two standards (e.g., one short and one long). In the temporal generalisation task, there is a right answer (e.g., the standard appears in the experiment); whilst there is no right answer in the temporal bisection task (a duration is either short or long).

To minimise the contribution of variance due to duration range, I used the common 200ms – 800ms for the temporal bisection task (Wearden, Win & Philpott, 1999; Elvevåg et al., 2003; Reed & Randell, 2014); and 100ms – 700ms for the temporal generalisation task (Ogden, Wearden & Montgomery, 2014), which are well used in the literature for

schizophrenic subjects. For duration probes, I used the arithmetic spacing (Penney & Cheng, 2018) to help minimise any of the variation associated with duration range and spacing probes (Penney & Cheng, 2018). A further consideration is the length of the experiment (Wearden, 2016; Penney & Cheng, 2018). Experiments which are too long (especially in the timing research) can impact attention due to boredom (Penton-Voak, Edwards, Percival & Wearden, 1996; Wearden, 2016). Therefore, to control for this, I ensured the experiments were 10 trials per duration however, an experiment that is too short comes with its own weaknesses (Wearden, 2016). The shortness of the experiment could have contributed to some of the variance in our experiments in that subjects did not have 'time' to habituate to the study. To address this issue, it would be prudent to (a)., have numerous duration ranges, and probes; (b)., as well as experimental length as between-subject factors. This would allow control for boredom and loss of interest in the studies.

In terms of the estimation study, I used a short kitten video, given the online nature of the task in both a prospective and retrospective paradigm This was chosen to (i) sustain subjects' attention and (ii) the assumption that kittens are fairly neutral. Given the discussion in Chapter 2 and 3,I had to utilise the suprasecond duration which, as Grondin (2010) implies, taps into higher cognitive functions though, as I discuss in chapter 2, some authors believe differences between the subsecond and suprasecond durations are quantitative in nature but manifest differently. The study, whilst implicating memory in driving time perception, demonstrated that schizotypal subjects were *more* accurate in reproducing durations than non-schizotypal subjects. Surely, if I am arguing there is a deficit, then such a deficit should manifest as schizotypal subjects being *less* accurate in reproducing durations. Given the fact that it is difficult to theoretically model the temporal estimation task (Wearden, 2016), and the fact the study itself was online, the sum of these issues could have led to schizotypal subjects showing an improvement in reproducing durations. In future studies, it might be

interesting to include ‘duration lengths’ as a between-subject factor to determine whether individual differences impact duration lengths.

7.3.3. Auditory and Visual Durations

One of the strongest findings in the timing literature is that auditory durations are overestimated compared to visual durations (Wearden, 2016). Theoretically, this modality effect in time perception has been explained by the onset of visual durations being delayed compared to auditory durations (Grondin, 1993; cited in Zelanti & Droit-Volet, 2012). In terms of SET, this would mean the switch closes later for visual stimuli thereby meaning visual durations are judged as shorter than for auditory durations (Zelanti & Droit-Volet, 2012). At first sight, this might explain why schizotypal subjects experience a ‘switchover’ in experiment two however, it would make no sense to suggest the switch closes earlier for visual durations at the switchover. Nonetheless, we do repot the classic result of subjects overestimating auditory durations (see experiments one and two, respectively) implying that some of the variance in the data could be explained by this effect, especially in the ‘switchover’.

7.3.4. SET

One of the methodological issues that often arise is whether SET is falsifiable (Wearden, 2016). This has led to a flurry of models being proposed, including Block & Zakay’s (1997) Attentional-Gate Model and other such models. Indeed, the data in our experiment cannot be explained parsimoniously by the clock model of SET; which is why I have opted for the memory component however, by opting for the reference memory component, I have neglected other components, such as the working memory and decision-making components of SET. There are further issues with SET, which researchers, such as

Block & Zakay (1997) have sought to address with their Attentional-Gate Model however, this model itself has issues.

7.3.5. Schizotypy

As discussed in Chapter two, one of the central issues with schizotypy is whether it can be used as an analogue for schizophrenia. The likes of Lenzenweger (2006) would contend that one cannot; however, I take the view that schizotypy is a metric for schizophrenia liability (Claridge, 1997) which assumes that whilst schizotypy is not a watered-down version of schizophrenia (Rado, 1952); it is likely driven by the same cognitive deficits that drive deficits reported in schizophrenia (Reed & Randell, 2014). One particular issue might be my use of the O-LIFE(B) scale (Mason et al., 2005). Whilst the scale measures schizotypy very well, and the three positive subscales (e.g., UE, CD and IN) map well onto positive schizophrenic symptomology, there are alternative questionnaires that can be used (see Claridge & Mason, 2014, for a review).

7.3.6. Future Research

As discussed, my studies were conducted on-line due to the pandemic. Therefore, whilst our findings are in line with 'off-line' studies, there is still a question of the on-line experiments not being completely controlled. A solution might be to conduct both on-line and off-line studies so that we have a between-subjects factor, in terms of where the experiment took place. This would be interesting for a number of reasons: the first is that I could compare findings between both on- and off-line studies, to determine if there is a difference in 'attended' time perception (e.g., when the subject knows they are being watched) and 'unattended' time perception. Secondly, any unaccounted-for variance due to the on-line nature of the experiments could be controlled for as a between-subjects factor.

Importantly, future studies which investigate time perception differences in terms of whether the experiment was on-line or off-line could expand the field significantly. On-line experiments are simple to implement and cost effective; as well as time effective. This would ultimately save money and resources and would, hopefully, contribute significantly to the timing literature.

7.3.7. Schizotypy Scales and Schizophrenia

Whilst O-LIFE is one of the more robust measures of schizotypy (Reed & Randell, 2014; Mason et al., 2005), it would be pertinent to investigate whether different scales' scores give rise to similar timing deficits. Once again, the scale that was used could be used as a between-subject factor to determine whether high scores on a specific scale give rise to a strong effect of timing deficits than other scales. Finally, to determine whether schizotypy and schizophrenia share the same timing deficits, it might be prudent to compare schizophrenic and schizotypy subjects' timing performance in a mixed-design experiment. For example, one could have three independent groups (baseline, schizotypy and schizophrenia) that are each subjected to a timing task (e.g., temporal bisection etcetera). This would allow a greater comparison of schizotypy and schizophrenia timing deficits. As far as we know, this has yet to be conducted, and would, in itself, be subjected to numerous confounding variables however, such a study could paint a fuller picture of the similar aetiology of timing deficits in schizophrenia and schizotypy (Reed & Randell, 2014).

Such a design could also provide further context on the significance of the clicker train manipulating duration judgements in schizophrenia. Whilst I have good evidence that the clicker appears to modify duration judgments, irrespective of schizotypy level, it would be interesting to determine whether the same affect arises in schizophrenia. The current thesis provides a theoretical justification in utilising the clicker condition in schizophrenic patients. Any resulting data demonstrating the effectiveness of clicker manipulation on judgement

duration could be used to alleviate some of the effects of retarded time perception in schizophrenia (Elvevåg et al., 2003; Carroll et al., 2008; Carroll et al., 2009; Reed & Randell, 2014). We have provided evidence that schizophrenia liability is amenable to the effects of a clicker; and it is hoped such an effect is found in schizophrenia, but further research is required.

7.3.8. Clicker Frequencies

Many studies have utilised a variety of clicker frequencies (e.g., Treisman et al., 1990; Wearden, Win & Philpott, 1999) with varying success. We used a 144Mhz Clicker train however, would schizotypal/schizophrenic patients react differently to frequencies? Such a question remains unanswered however, any future study could investigate the effect of different clicker frequencies on duration judgements to find an optimum frequency where the subject responds. Furthermore, in hindsight, I should have randomised the baseline/clicker conditions however, we did not want to introduce a cofounding variable in the form of arousing the schizotypal subjects before the clicker condition. Any future study should randomise these trials, in accordance to Wearden, Win & Philpot (1999) since this thesis provides evidence that the clicker train is effective, irrespective of clicker condition.

7.3.9. Genetic Selection of Schizotypal Subjects:

As reviewed in Chapter 2, Rado (1952) and Mheel (1963) both argued that there is a genetic component to schizotypy and schizophrenia (e.g., the so-called ‘schizogene’ which gives rise to Schizotaxia – see chapter 2). A fairly recent review by Tarbox & Pogue-Geile (2011) suggested elevation of schizotypal symptomology among relatives of schizophrenic patient. Conversely, offspring of high schizotypical subjects have a higher risk of developing schizophrenia than offspring of low schizotypical subjects (Gruen, Asnis & Kane, 1983; Battaglia et al., 1995; Kendler & Walsh, 1995). Linney et al. (2003) suggested that up to 50%

of schizotypy variance is explained by genetics (Barrantes-Vidal, Grant & Kwapil, 2015). Furthermore, a wide variety of genes and polymorphisms that have been identified as relevant to schizophrenia are associated with schizotypy, including the single nucleotide polymorphism, rs4680 (COMT val^{158}met) and other dopaminergic polymorphisms (see Grant et al., 2013 for a review). This is a significant gene, as it ties into the dopaminergic clock hypothesis of the pacemaker (Wearden, 2016) and would suggest that schizotypy is a useful construct in researching the aetiology of schizophrenia (Barrantes-Vidal et al., 2015).

Whilst the genetic basis of schizotypy is not a new ideation (Mheel, 1962; see Chapter 2) in the context of time perception, such a genetic selection could complement schizotypy questionnaires. For example, the UK BioBank could be used to find subjects with the COMT expression and they could be contacted to determine whether they would be willing to partake in timing experiments. By utilising genetic markers of schizotypy, it complements the use of questionnaires, and could provide further evidence on what drives timing deficits in schizotypy and schizophrenia (Reed & Randell, 2014)

7.3.10. Focusing on other components of SET

As stated, I have suggested it is more parsimonious to focus on the reference memory component of SET, which should explain deviations in both suprasecond and subsecond durations however, I have neglected the working memory and decision process components of SET due to time constraints – and the online nature – of the study. One way future researchers could measure the additional components of SET is to have a working memory task, such as the N-back task (Yaple & Arsalidou, 2018), to determine whether schizotypal subjects have deficits in working memory. A decision-making task (Deverett, Koay, Oostland & Wang, 2018) could also be used to determine whether schizotypal subjects have deficits in other components of SET. Of course, Carroll et al. (2008) utilised the SKE-memory model of

SET to measure memory deficits however, this method relies on computer-derived data, as opposed to behaviourally-observed data which, in my opinion, does not explain the nuances of human behaviour but rather, the nuances of computer behaviour! I propose to analyse the components of SET more robustly, researchers should hark back to the mathematical origins to model the components of SET.

7.5 Conclusion

This thesis explored why schizophrenics should have timing deficits at all, and why should time perceptions present mixed findings. To research this, I asked whether a click train could modify time perception in schizotypy and what component of SET is responsible for timing deficits in schizophrenia and schizotypy. To do this, I investigated time perception in schizotypy. An auxiliary aim of this thesis was to establish whether a clicker train could be used to modify time durations in schizotypal subjects. Despite the limitations that have arisen in this thesis, there are two critical findings that emerge from this thesis. The results of Chapters 4 and 5 appear to show that timing deficits in schizotypy are driven by the reference memory component of SET and therefore, we have proposed that memory deficits are the cause of timing deficits in schizotypy and possibly, schizophrenia. However, Chapter 6 implies that the memory 'deficit' leads to better accuracy of durations, at least in suprasecond durations. We have also shown that the Clicker train effectiveness (or lack of) is not mediated by schizotypy level; suggesting that a clicker train could be used to modify time perception in schizophrenia. Furthermore, we have also suggested how researchers might solidify these results by considering genetic bases of schizotypy, and the potential of using both schizotypy and schizophrenic patients in future studies. Such research has the potential of allowing us to understand what drives timing deficits in schizophrenia; and how we might be able to alleviate such deficits by use of a clicker train. In summary, my thesis suggests that

the (i), memory appears to drive timing deficits in schizotypy, at the subsecond range, but improves accuracy in the suprasecond range. I have also shown (ii) that a clicker train is effective in modifying time perception in schizotypy subjects, and thus, opening a door on a potential treatment option for timing deficits in schizophrenia.

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Appendices

Appendix 1: Experiment 1 Training (Baseline)

The purpose of the study is to see how good people are at deciding whether something lasts for a short or a long time.

For the next half a minute, there will be 10 training trials. You will see squares for either a *short* or for a *long* amount of time.

In each trial, we will tell you which square is considered *long* and which *short*.

Press the spacebar to begin.

Appendix 2: Experiment 1 Test (Baseline)

Next, you will see squares like the ones you have just seen, of varying lengths. Your task is to decide whether the square lasts for a 'Short' or for a 'Long' amount of time.

Press **(Z)** to indicate a **SHORT** time and **(M)** to indicate a **LONG** time.

Press the spacebar to begin.

Appendix 3 Experiment 1 Test (Click Train Training)

Almost finished! This is the final training-testing session of the experiment.

As before, you will be reminded of what we mean by 'long' and 'short' tones for about 1/2 a minute.

Press the spacebar to continue.

Appendix 4 Experiment 1 Test (Click Train):

This is the final part of the experiment. As before, we will ask you to determine whether the square is displayed for a 'Long' or for a 'Short' amount of time. However, this time, a short burst of clicks will play right before each square.

Press **(Z)** to indicate a **LONG** time and **(M)** to indicate a **SHORT** time.

Please press the spacebar to begin.

Appendix 5: Experiment 2 Training (Baseline)

Thank you very much for taking part in our study.

The purpose of the study is to see how good people are at deciding whether something lasts for a short or a long time.

For the next half a minute, there will be some training trials. You will hear tones that correspond to different lengths. The tone will play either for a *short* amount of time or for a *long* amount of time.

In each trial, we will tell you whether the tones accord to short or long.

Please pay attention to those training trials, because you will be asked to remember them later.

Press the spacebar to begin.

Appendix 6: Experiment 2 Test (Baseline)

Excellent - that's the training over.

Next, it is your turn to decide whether the tone plays for a 'Short' or for a 'Long' amount of time, based on your training.

The tones will be of a varied length.

Press **(Z)** to indicate a **SHORT** time and **(M)** to indicate a **LONG** time.

Try to be as accurate as possible.

Press the spacebar to begin.

Appendix 7: Experiment 2 Test (Click Train Training)

Almost finished! This is the final training-testing session of the experiment.

As before, you will be reminded of what we mean by 'long' and 'short' tones for about 1/2 a minute.

Press the spacebar to continue.

Appendix 8: Experiment 2 Test (Click Train)

Excellent. The training is over.

As before, we will ask you once more to determine whether the tone plays for a 'Short' or for a 'Long' amount of time.

However, this time, a short burst of clicks will play right before each tone.

Press **(Z)** to indicate a **SHORT** time and **(M)** to indicate a **LONG** time.

Please press the spacebar to begin.

Appendix 9: Experiment 3 Training (Baseline)

Thank you for taking part in our study. We want to investigate how good people are at comparing a tone to other tones. But before you do that, we first have to train you on a practice trial.

First, you will hear the tone, and then, you will hear another tone. Your task is to determine whether the second tone matches the first tone. In all cases, you will be given feedback telling you whether the tones matched (green cross) or did not match (red cross).

Please press the spacebar to continue.

Appendix 10: Experiment 3 Test (Baseline)

Excellent - that's the training over.

Your task is similar to the training; where you decide whether the
matches the comparison tone this time, though, you will not be g
feedback and the first tone will not be played before the compari
tones.

The tones will be of a varied length.

Press **(Z)** to indicate **(NO)** and **(M)** to indicate **(YES)**.

Try to be as accurate as possible.

Please press the spacebar to begin and good luck!

Appendix 11: Experiment 3 Test (Click Train Training)

This is the final part of the experiment! But, once again, you have to do training.

First, you will hear the tone, and then, you will hear another tone. Your task is to determine whether the second tone matches the first tone. In all cases, you will be given feedback telling you whether the tones matched (green cross) or did not match (red cross).

Please press the spacebar to continue.

Appendix 12 Experiment 3 Test (Click Train)

Excellent -- that's the training over and like before, you will be asked t decide whether the following tones match the standard duration or d not match it, based on your training. The only difference is that the tones will be preceded by a click. This is the final part of the experiment.

The tones will be of a varied length.

Press **(Z)** to indicate **(NO)** and **(M)** to indicate **(YES)**.

Try to be as accurate as possible.

Please press the spacebar to begin and good luck!

Appendix 13: Experiment 4 Training (Baseline)

Thank you for taking part in our study. We want to investigate how good people are at comparing a shape to other shape durations. But before you do that, we first have to train you on a practice trial.

First, you will see an oval, and then, you will see another oval. Your task is to determine whether the second oval matches the duration of the first oval. In all cases, you will be given feedback telling you whether the ovals matched (green cross) or did not match (red cross).

Please press the spacebar to continue.

Appendix 14: Experiment 4 (Baseline)

Excellent - that's the training over.

Your task is similar to the training; where you decide whether the square matches the comparison square this time, though, you will not be given feedback.

The squares will be of a varied length.

Press **(Z)** to indicate **NO** and **(M)** to indicate **YES**.

Try to be as accurate as possible.

Please press the spacebar to begin and good luck!

Appendix 15 Experiment 4 Test (Click Train Training)

This is the final part of the experiment! But, once again, you have to do training.

First, you will see the oval that is of a set duration, and then, you will see another oval of a different duration. Your task is to determine whether the second oval duration matches the first oval duration. In all cases, you will be given feedback telling you whether the ovals matched (green cross) or did not match (red cross).

Please press the spacebar to continue.

Appendix 16 Experiment 4 Test (Click Train)

Excellent -- that's the training over and like before, you will be asked to decide whether the following tones match the standard duration or do not match it, based on your training. The only difference is that the tones will be preceded by a click. This is the final part of the experiment.

The tones will be of a varied length.

Press **(Z)** to indicate **(NO)** and **(M)** to indicate **(YES)**.

Try to be as accurate as possible.

Please press the spacebar to begin and good luck!

Appendix 17: Experiment 5 (Retrospective Paradigm)

Thank you for consenting to the experiment. Please watch the video and you will be directed on an additional task after the video.

Please press the spacebar to continue.

Appendix 18: Experiment 5 (Prospective Paradigm)

Thank you for consenting to the experiment. Your task is to watch a video, and then tell us how long you think it lasted.

Please press the spacebar to continue.