EXPLORING RELATIONSHIPS BETWEEN TEMPORAL PROCESSING DEFICITS AND PSYCHOSIS USING THE PEAK INTERVAL TASK

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ABSTRACT

Persons with schizophrenia commonly report distortions in their subjective experience of time. Mirroring these subjective experiences are findings that persons with schizophrenia are both less accurate, and precise, in detecting time in the range of seconds to minutes (interval timing). However, the mechanisms which give rise to these deficits in interval timing remain unknown as previous studies have relied on paradigms which do not allow us to easily dissect the influence of timing processes from memory, and decision making, on task performance. In addition, these studies have typically depended on samples who are taking antipsychotic medications making it difficult to determine whether deficits in interval timing are related to psychosis specifically or the consequences of antipsychotic medication. To address these concerns, I developed an online peak interval task. The peak interval task is a gold standard paradigm in which participants are instructed to learn, and reproduce, an unknown duration of time; analysis of trial-by-trial reproductions of this duration allows for the relative influence of internal clock, memory, and decision-making processes on temporal processing to be teased apart. In a series of studies, I tested the validity of this newly developed task and then tested the extent to which temporal processing deficits could predict psychosis-risk status in a non-clinical sample. In *Experiment 1*, 524 undergraduate students completed an online peak interval task in which they were asked to learn, and reproduce, two durations of time (6s and 20s) over multiple trials. Performance on this task was compared to that of 14 rats completing an analogous task. Data from humans broadly aligned with the general principles of interval timing, attesting to the validity of the paradigm. While the general pattern of performance was similar in rats and humans, there were some quantitative differences: the human sample was more accurate and precise than the rodents. This improved performance was related to a greater influence of memory and decision-making processes on performance. In *Experiment* 2, I recruited 61 individuals who were classified as at-risk for psychosis, had no formal psychotic disorder, and were not taking antipsychotic medications. The peak interval performance of these individuals was compared against 90 randomly selected controls. Timing accuracy and precision in reproducing the 6s duration predicted risk-group membership. Additionally, timing accuracy in reproducing the 6s duration explained significant variance in the presence of positive, and negative, schizotypal traits across the sample. These findings suggest that disruptions in temporal processing may be a risk marker for schizophrenia which may help illuminate illness mechanisms.

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INTRODUCTION

Schizophrenia is a psychological disorder which affects approximately 1% of the global population. Persons with schizophrenia frequently experience frightening hallucinations, delusions, and chronically lowered motivation; symptoms which contribute to significant decreases in their quality of life (Eack & Newhill, 2007) and life expectancy (Wildgust et al., 2010). Despite the significant health costs posed by schizophrenia, treatment options remain limited with up to 30% of people (Ackenheil & Weber, 2004) not responding to the gold-standard treatment – antipsychotic medication. Development of new treatments for schizophrenia is slow (Weston-Green, 2022) and hindered by a poor understanding of the mechanisms which give rise to symptoms of psychosis (Jablensky, 2010). Difficulty in developing new treatments for schizophrenia may be driven, in part, by the fact that physiological accounts of schizophrenia rarely address the subjective experiences reported by persons with psychosis. Attempting to elucidate the neurobiological mechanisms which underly the subjective experiences of persons with psychosis may inform the development of new treatments.

Persons with schizophrenia often report distortions in their subjective experience of time such that time “falls apart and no longer processes” (Fuchs, 2013; Stanghellini et al., 2016). An accurate sense of time is necessary for navigating the environment. For instance, to safely cross the road, I must gauge when to start crossing and when to adjust my pace. This requires that I also track the duration of time that has passed, and integrate it with the rhythm of the light signals and oncoming traffic. Given the importance of timing for navigating one’s environment, it is possible that disrupted temporal processing could not only impact a person’s subjective experience of time, but also influence a range of cognitive processes. For example, disruptions in temporal processing could influence an agent’s ability to learn relationships between actions and outcomes. Disruptions in these processes could, in turn, account for the widespread changes in thoughts and behavior which characterize schizophrenia as disrupted action-outcome learning could lead an agent to attribute the consequences of an action (e.g. a light turning on) to an external force (e.g. a ghost). Overtime, this misattribution would instill a reduced sense of self-agency which could give rise to the experience of delusions (‘I am being controlled’) or to internal experiences being misinterpreted as hallucinations. Alternatively, disruptions in temporal processing could inhibit an agent from associating action with reward, thereby reducing the likelihood that those actions would be repeated. Over time, this would lead to an instance where a person might enjoy pleasurable stimuli but make no effort to obtain them (Strauss et al., 2014) which is characteristic of negative symptoms of schizophrenia. In support of the idea that disruptions in interval timing could contribute to symptoms of schizophrenia, recent meta-analyses have concluded that persons with schizophrenia are significantly less accurate (Ciullo et al., 2016) and precise (Thoenes & Oberfeld, 2017) in their perception of time in the range of seconds to minutes (interval timing) with worse interval timing ability predicting more severe positive symptoms of schizophrenia (Ueda et al., 2018). Combined, these data suggest that disruptions in temporal processing could contribute to symptoms of schizophrenia.

However, the clinical relevance of interval timing deficits in psychosis remains understudied, and limitations exist within the current literature. While multiple studies have reported differences in the interval timing abilities of persons with schizophrenia compared to controls (Bolbecker et al., 2014; Carroll et al., 2009; Lhamon & Goldstone, 1973; Papageorgiou et al., 2013), the exact pattern of distortion is inconsistent. Some findings suggest that persons with schizophrenia are less accurate than controls (Ciullo et al., 2016), while others suggest persons with schizophrenia are less precise (Thoenes & Oberfeld, 2017). These findings raise questions about the origins of timing deficits in persons with schizophrenia as the causes of inaccurate, and imprecise, timing are different according to contemporary models of interval timing.

One such model, scalar expectancy theory (SET; Gibbon, 1977), posits that agents track time through the use of an internal pacemaker that emits pulses at a variable, but mostly steady, pace and an accumulator which counts the pulses generated by a pacemaker. These systems are supported by an attention-controlled switch which, upon closing, allows pulses to flow from the pacemaker into the accumulator and a memory store which encodes the output of the accumulator. The contents of the accumulator comprise an agent’s representation of time. In this way, temporal processing depends upon three stages: a clock stage during which the attention-mediated switch closes and pulses accumulate into the accumulator, a memory stage during which the contents of the accumulator are encoded into memory and previous accumulator values are retrieved from long-term memory, and a decision-making stage during which current accumulator values are compared with a retrieved value from memory to decide if the values are ‘close enough’ to some reference value. When the values are deemed to be equivalent, the attention modulated switch opens, and the contents of the accumulator are reset. Extensive literature supports the predictions made by SET (see Malapani & Fairhurst (2002) for review).

According to SET, reduced accuracy is likely due to the speed of the internal pacemaker or difficulty in maintaining the attentional switch, whereas differences in precision could be related to either a) a more variably ticking pacemaker, b) issues with the attentional switch leading to an inconsistent loss of pulses in the accumulator or c) disruptions in memory processes (e.g. difficulty in retrieving or encoding values in memory). As such, current findings suggest that persons with schizophrenia may show deficits in either their internal timing system or memory processes. Differentiating between these effects is complicated, however, by a reliance on patient samples that are using antipsychotic medications (e.g. Roy et al., 2012; Tracy et al., 1998)). Antipsychotic medications exert their effects through modulating dopaminergic receptor activity (Amato et al., 2018); this is important because interval timing depends on a dopaminergically-modulated fronto-striatal circuit (Buhusi & Meck, 2005). For example, increasing dopamine D2 receptor activity can lead agents to significantly underestimate durations of time (Mikhael & Gershman, 2019), which can lead to a decrease in timing accuracy without influencing precision. Thus, whether the interval timing deficits observed in persons with schizophrenia reflect an issue with the internal timing system, memory store, or the influence of antipsychotic medication is unknown.

Although interval timing has primarily been examined in people diagnosed with a psychotic disorder, mounting evidence suggests that schizophrenia represents the most severe end of a psychosis spectrum (van Os, 2016; van Os et al., 2009). On the other end of this spectrum are persons who experience sub-threshold psychotic-like experiences. These individuals are people with no formal psychotic diagnosis who are at increased risk for developing schizophrenia (Kelleher & Cannon, 2011). While these psychotic-like experiences comprise sub-clinical delusions and hallucinations, these experiences are also associated with deficits in motivation which are akin to those observed in clinical populations (Schlosser et al., 2014). To date, few studies have investigated interval timing in people with high levels of psychotic-like experiences. Those that have suggest persons with psychotic-like experiences show deficits in timing accuracy (Osborne et al., 2021) and precision (Penney et al., 2005). Interestingly, reduced timing accuracy in individuals with high levels of psychotic-like experiences explained up to 11% of variance in the worsening of negative symptoms over time (Osborne et al., 2021). That interval timing deficits are observed in individuals with psychotic-like experiences and that these deficits predict the worsening of motivational impairments over time supports the notion that disruptions in the subjective experience of time may precede the formal onset of a psychotic disorder.

However, both of these aforementioned studies relied on the same experimental paradigm to measure interval timing ability – the serial bisection task. On this task, subjects are required to learn two benchmark durations (a short and long duration), then judge whether a series of novel stimuli are closer in length to the short or long benchmark. Responses on this task can be analyzed to produce a ‘bisection point’ which is the duration that is equally likely to be classified as ‘short’ or ‘long’ (Allan & Gibbon, 1991). The difference between the veridical location of the bisection points and the mean of the ‘short’ and ‘long’ benchmarks reflect the temporal accuracy of a subject (Wearden et al., 1997). Extensive research backs the use of the bisection point as a sensitive measure of temporal processing (Penney & Cheng, 2018). However, changes in the bisection point could reflect several different mechanisms: differences in the pacemaker rate (clock stage), dysfunction in representing the benchmark stimuli in memory (memory stage) or fluctuations in attention (e.g. difficulty closing the attentional switch; Levy et al., 2015). Unfortunately, responses on the bisection task do not easily permit researchers to differentiate between these hypotheses.

The influence of clock, memory, and decision-making stages on timing can potentially be teased apart using a different interval timing paradigm – the peak interval task (Balcı et al., 2013; Catania, 1970; Roberts, 1981). The peak interval task is a gold standard paradigm in animal research in which agents must learn to respond immediately after a fixed time has passed in order to achieve a reward. Responses on each trial of the peak interval task are usually characterized by a low-high-low pattern of responding such that agents begin with a low response rate which rapidly increases before reaching a maximum (the peak time). After this point, responses decline at a rapid pace. In some trials, a reward is never given; responses on these no-reward trials provide information about an agent’s timing ability. Averaging responses across these no-reward trials and fitting a function to this data permits measures of timing accuracy (peak time; the time at which the maximal responses occur), precision (the variability of an agent’s timed responses) and motivation (peak rate; the maximum number of responses per second) can be derived (see Figure 1; Freestone & Balcı, 2018).

In addition to averaging responses across trials, performance on the peak interval task can be examined on a trial-level basis to disentangle the relative influence of clock, memory, and decision-making stages on performance. To do so, the point at which the response rate initially increases (start time) and decreases (stop time) is calculated for each trial. These metrices are then correlated with each other. The magnitude of correlations between start-stop time, start time-middle (arithmetic mean of start and stop time), and middle-spread (difference between start and stop time) has been shown to reflect the influence of different processes on timing. For example, positive correlations between start-stop, start-spread, and middle-spread reflect a strong influence of an agent’s internal clock mechanism (Church et al., 1994) because an agent who responds earlier should also see an earlier peak in responding and earlier cessation of responding. In this way, responding that depends on clock mechanisms will result in proportional relationships between these metrics: a delay in start-time should produce similar delays in middle- and stop-times. On the other hand, negative correlations between start-stop and start-spread values indicate the decision to start responding is being influenced by a purposeful decision to delay (or begin) responding while stop-times remain the same; in this way, negative correlations indicate the influence of decision-making processes. A later start time, in the context of preserved stop time, also leads to a tighter response function hence leading to negative relationships between start-spread values. Thus, by examining changes in the strength and sign of these correlations, the peak interval task allows us to dissect which cognitive processes contribute to differences in temporal processing performance.

Extensive animal research has elucidated the biological mechanisms which support performance on the peak interval task (see Balcı (2014) for review) making it ideal for exploring potential causes of abnormal temporal processing. However, despite substantial research backing its utility in understanding interval timing behaviors, the peak interval task has not often been used in humans (Fortin et al., 2009; Lake & Meck, 2013; Lustig & Meck, 2005; Malapani et al., 1998; Rakitin et al., 2006) and never, to my knowledge, in individuals with, or at risk for, psychosis. The relative scarcity of studies using the peak interval task in humans may, in part, be driven by the lack of data demonstrating equivalence between humans and animals, which limits researchers' ability to use animal literature to guide their predictions about the behavioral, and neurobiological, correlates of peak interval performance in clinical populations.

Developing and validating an online peak interval task for use in humans would offer several benefits. First, adapting the peak interval task for online human participation would allow researchers to leverage the extensive animal literature exploring mechanisms of interval timing to infer the causes of abnormal time perception in psychosis, thereby potentially informing the development of new treatments. Second, developing an online peak interval task would allow researchers the opportunity to rapidly recruit large numbers of participants while reducing recruitment costs (Gagné & Franzen, 2023). For example, individuals with high levels of psychotic-like experiences are relatively uncommon (e.g. 7%; Linscott & van Os, 2013) but can be identified using self-report questionnaires. Thus, the development of an online paradigm would allow for easy recruitment of larger numbers of individuals with psychotic-like experiences as well other clinical samples. Indeed, abnormal temporal processing has been observed across many clinical samples including Parkinson's, depression, and ADHD (See Allman & Meck (2012) for review).

In this paper, I report on the results of a series of experiments. In *Experiment 1*, I developed an online peak interval task for use in human subjects. A large sample of undergraduate students were recruited from Michigan State University and completed a newly developed peak interval task that was administered online. The performance of my human sample was compared against that of rodents completing an analogous task to test whether time perception processes differed between species. I predicted that peak interval performance – as measured by timing accuracy and precision – would be similar across species. In *Experiment 2*, I used this task to examine whether the interval timing abilities of individuals at elevated risk for psychosis are similar to a sample of controls.

EXPERIMENT ONE

Methods

Participants

Humans. 524 undergraduate students were recruited from Michigan State University's Psychology SONA Pool. Mental health and medication history were established via self-report questionnaires. Participants were excluded if they reported a personal history of mental illness and/or psychotropic medication use. After the exclusion criteria were applied, there was a final sample of 327 participants (mean age=19.40 years old, SD = 1.50; 69.4% female, 29.4% male; 67.9% white, 14% Asian, 9.2% Black or African American, 8.5% other). All study design procedures were reviewed, and approved, by Michigan State University's institutional review board.

Rats. Data from the rodent sample has been reported elsewhere (see Raycraft, 2023). Briefly, 14 Sprague-Dawley rats were housed in groups of 2-3 in standard, plexiglass cages with metal tops. All rats were maintained on a standard 12-hr light-dark cycle with free access to food pellets and water. All experimental procedures for these rats were conducted in compliance with Michigan State University's Institutional Animal Care and Use Committee.

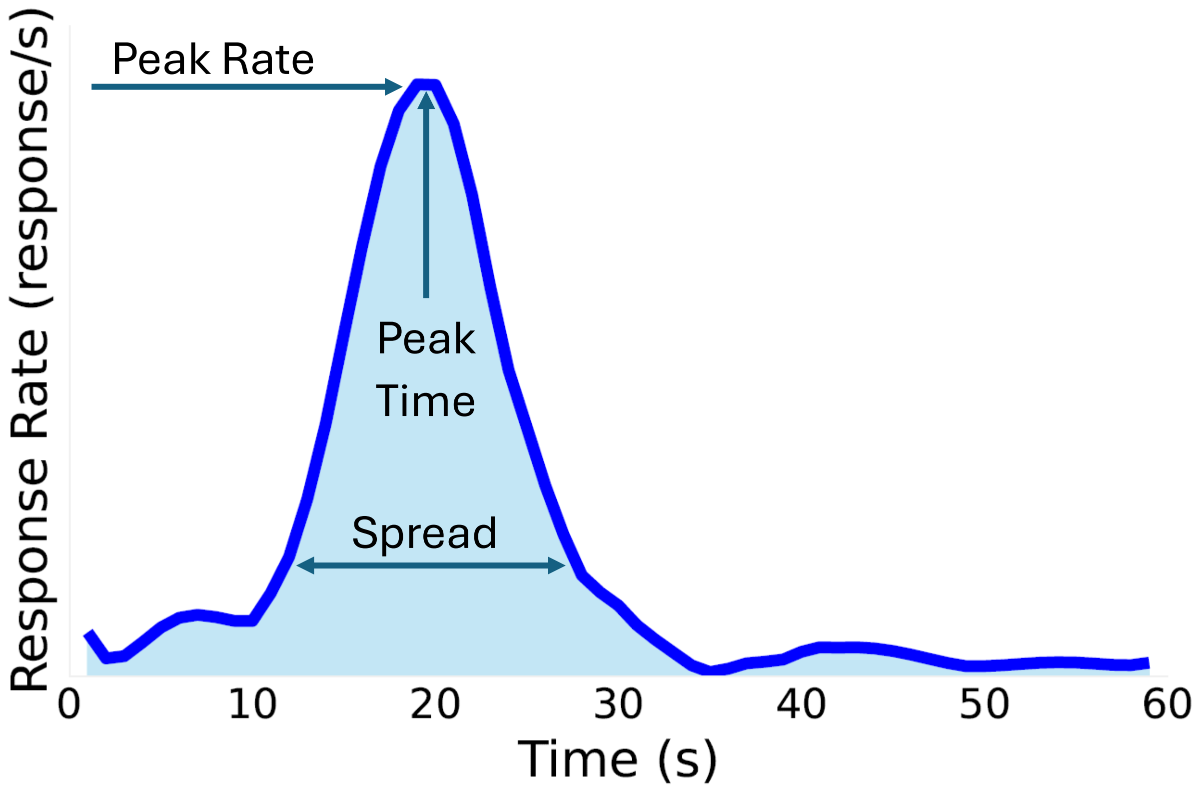

Figure 1. Representative data from a single participant completing the peak interval task for the 20-second condition. Participant responses were binned into 1-second bins to produce a response curve from which peak time (the time corresponding to maximal responding), rate (maximum response rate) and spread (width of the curve) were calculated. Peak time represents the participant's internal representation of the target duration, spread represents the variability associated with this internal representation and peak rate represents a participant's motivational state for the task.

Human Peak Interval Task

Design and Procedure. I coded a peak interval task using PsychoPy (version 2.4, Peirce et al., 2019, 2022). The experiment was hosted online through a third-party recruitment platform, Pavlovia (pavlovia.org). Human participants completed the online peak interval task (Figure 2) which required them to learn a fixed interval reward schedule. This task consisted of practice, training, and test trials. Blocks of practice, training, and test trials were completed for a singular target duration at a time (either 6s or 20s). Participants completed the task for both target durations. The order of target durations was counterbalanced between participants to account for potential learning effects. The task began with 10 training trials, the purpose of which was for participants to learn the target duration. On these trials, participants were instructed to fixate on a central stimulus (white cross). The stimulus would turn red after the target duration (either 6 or 20s) had passed, and participants were told to press the spacebar as soon as the stimulus changed color. Following these training trials, participants completed a single block of 20 practice trials. These trials were like training trials except the cross never changed color. Instead, participants were asked to indicate when they thought the cross should have changed color by creating a window of spacebar presses around the target time; these instructions were adapted from previous studies using the peak interval task in humans (Fortin et al., 2009; Lustig & Meck, 2005; Rakitin et al., 1998, 2006). Participants were instructed to press the enter key as soon as they were certain the target duration had passed. The trial terminated once the enter key was pressed or once a maximum duration of three times the target time had expired. At the end of each trial, participants were awarded points according to how close they were to the target time when they terminated the trial: termination within 8.5% of the target duration was awarded 3 points, termination within 17% of the target duration was awarded 2 points and termination within 25% of the target duration was awarded 1 point. After a single block of practice trials, participants completed a block of 55 test trials. Test trials were identical to practice trials. Upon completion of the test trials, participants repeated the procedure for the second target duration.

To discourage the use of counting, which would aid in determining elapsed time, participants were instructed to read aloud a series of randomly generated letters that were presented below the fixation cross at a rapid, but variable, rate. Similar strategies have been used in other human adaptations of the peak interval task (Malapani et al., 1998; Rakitin et al., 1998, 2006). Any trial which was terminated too early (sooner than half the target duration) or had no responses was repeated.

Following completion of the peak interval task, participants completed a battery of self-report measures, including basic demographics and mental health history. We also asked participants to indicate whether they had completed the distractor task, and whether they had used counting strategies to solve the task. Completion of the surveys took 35 minutes on average. Full details of the questionnaires are reported in *Experiment 2.*

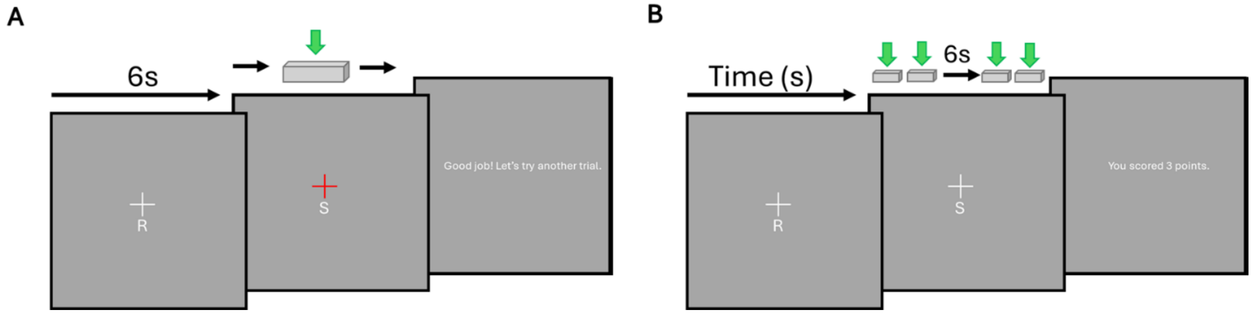

Figure 2. The peak interval task. This task consisted of blocks of training (panel A), practice and test trials (panel B). On training trials (panel A), participants were required to press the space bar as soon as the white cross became red. As soon as the spacebar was pressed, the trial ended, and the next trial began. This was repeated across 10 successive trials to help participants learn the target duration (6s or 20s). Following the training trials, participants completed 20 practice and 55 test trials. These trials were identical (Panel B). On these trials, the cross never changed color and pressing the spacebar would not terminate the trial. Instead, participants were instructed to create a window of key presses around the time they believed the cross *should* change color. Practice and test trials would last for 3 times the target duration. However, participants were able to end these trials early by pressing the enter key when they were sure the target duration had passed.

Rodent's Peak Interval Task

Design and procedure. Full details of the peak interval task completed by the non-human sample are reported elsewhere (see Raycraft (2023) for full details). Briefly: Sprague Dawley rats were trained to complete a peak interval task using a total of 16 training sessions. Each training session was composed of 25 peak trials randomly intermixed with 25 fixed interval trials. Training sessions were completed in four blocks, each comprising four sessions. Once the task had been acquired, these rodents completed a peak interval task with a target duration of 20s. Testing sessions were identical to training sessions. The start of each trial was indicated by the illumination of the house light and presentation of a response lever. On a fixed interval trial, the first lever press that occurred at, or after, 20s was reinforced by the delivery of liquid sucrose; simultaneously, the house light was extinguished and response lever was retracted. Peak trials were identical to fixed trials except that no lever press was reinforced. Instead, trials automatically terminated after at least 3 times the target duration (60s) had elapsed. For the purpose of the analysis, I did not include any responses that occurred 3 times after the target duration had elapsed.

Analysis

Participant-Level Analysis. Performance on the peak interval task can be evaluated at the level of both participants and individual trials (see Supplementary Table 1). Participant-level performance was evaluated by collapsing each participant's responses across all trials and binning each response into 1s bins to produce a response curve for each participant, in accordance with prior studies (Balci & Freestone, 2018; Figure 1). From this curve, several timing-related metrices were obtained: peak time, spread, and peak rate (Supplementary Table 1; see Balcı & Freestone, 2020; Freestone & Balcı, 2018). Peak time was defined as the time of maximal responding and represents an agent's internal representation of the target duration. Spread was defined by the width of the response curve and was measured by the difference between the time at which the response rate first exceeded a participant's average response rate, and the time at which a participant's response rate dropped below their average response rate. Spread represented the variability associated with an agent's perception of the target time. Finally, peak rate was defined as the maximal response rate (average number of responses per second) of each participant. Peak rate is believed to represent an agent's motivational drive such that higher values indicate more task motivation.

Trial-Level Analysis. Trial-level analyses inform about the relative contributions of decision-making, memory, and timing on peak interval performance. Metrices for trial-level analyses were derived using the procedures described by Church et al. (1994). This is accomplished through examining relationships between the start (the time at which response rate first exceeds average response rate), middle (the arithmetic mean of the start and stop values), and stop times (the time at which the response rate first drops below average response rate) across trials, as well as the spread of responses within individual trials (see Figure 3). Peak Interval performance that depends on an internal timing mechanism conforms to several rules (Balcı, 2014; Gibbon et al., 1984). Responding on individual trials is typically characterized by a break-run-break pattern in which responding rapidly accelerates as the target time approaches and decelerates as it passes (Balcı, 2014). This pattern of responding is believed to reflect an increase in reward expectancy which rapidly diminishes as time passes. As such, the decision to accelerate responding is usually proportional to the decision to decelerate responding as both actions are being coordinated by an agent's internal representation of time. In this case, start and stop times should be positively correlated as a delay in the acceleration of responding should lead to a delay in the deceleration of responding. Proportional delays in start and stop-times will also produce a delay in their arithmetic mean. Thus, performance that is influenced by the internal timing mechanism should produce positive correlations between start and middle times. Finally, the variability surrounding an estimate of time should increase as the duration becomes longer; this is because the pacemaker produces somewhat variable ticks – the longer a duration, the more variability is contributed to the internal representation of time, which would manifest as a positive relationship between middle times and spread. Thus, performance which is influenced by clock mechanisms, should produce positive correlations in start-stop, start-spread, and middle-spread values.

On the other hand, inverse relationships between start and stop times can indicate the influence of decision-making factors. For example, a negative start-stop correlation could indicate an agent is increasing their responding much later than would be expected given their stop-time. This would indicate the agent is purposefully delaying the start of their responding which suggests their responses are being based on the expectation of when the target time is more likely to be close, rather than being driven by their internal passage of time. In turn, this pattern of performance is likely to lead to a narrower response curve as the start-time is delayed without a proportional delay in stop time; this would be represented by a negative correlation between start and middle times.

In this way, trial level analyses involve two steps: 1) examining the magnitude of correlations between different performance metrics, and 2) comparing how these correlations differ across species. The pattern of significant correlations can inform about the relative influence of the internal timing mechanism, memory, and decisions to accelerate/delay responding on the peak interval task. For example, as listed above, positive correlations between start-stop, start-spread, and middle-spread would indicate performance that is primarily driven by an agent’s internal timing mechanism (Balcı, 2014; Church et al., 1994) whereas negative start-stop and start-spread correlations indicate an agent’s decisions to response were influenced by factors other than their internal timing mechanism. A combination of both positive, and negative, correlations should indicate a mixture of both timing mechanisms, and decision-making processes, on peak interval performance.

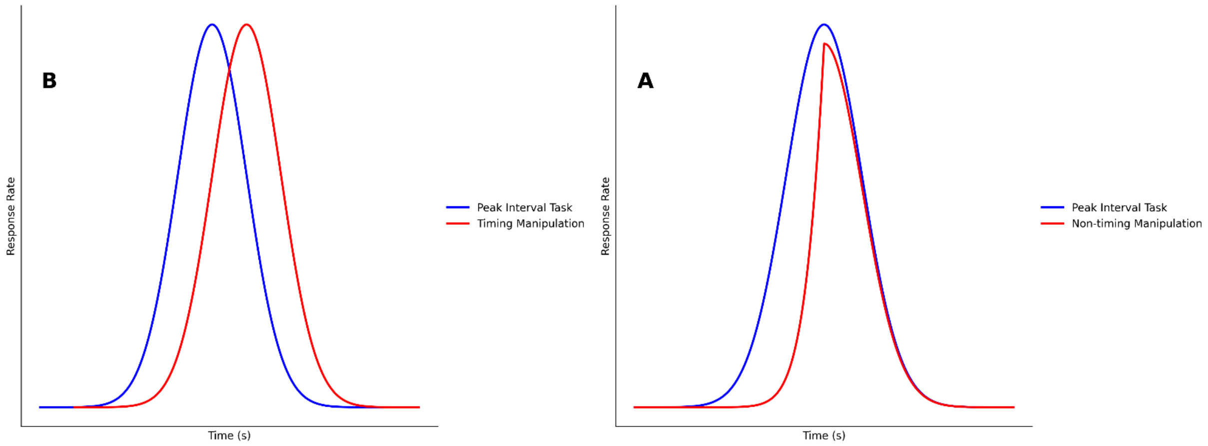

Figure 3. Simulated trial-level data on the peak interval task. The influence of clock/memory can be differentiated from the influence of decision-making factors by comparing correlations between start and stop times, start-spread times, and middle-spread. Performance which is influenced by a clock mechanism should demonstrate a gradual increase in responding as the target time approaches followed by a decrease in responding as the target time passes. Across trials, this should produce positive relationships between the time at which responding begins, and ends, as these both serve similar benchmarks to a passage of time. In other words, a change in start time should produce similar changes in stop time as the relationship between these variables is modulated by a pacemaker which ticks at a steady pace. This would also result in positive relationships between the start time and spread as the longer an agent waits before responding, the larger their uncertainty around the target time (hence leading to a larger spread). Likewise, a positive relationship between middle and spread indicates that uncertainty increases as the target duration grows larger: this would be indicative of a timing mechanism which ticks with a variable, but mostly, steady pace. B) Performance that is influenced by non-timing factors (such as motivation for reward) will lead to negative correlations between start and stop times, and start times and spread. Negative correlations between start and stop times indicate that an agent may be delaying the start of their responding while their stop-time remains unaffected. In this case, the agent appears to be adjusting their behavior based on the expectation of when a reward is likely to appear; thus indicating a deliberate decision making process. In a similar manner, an agent who delays their start-time without also adjusting their stop-time will produce a narrower spread of responses, thus resulting in a negative correlation between start-time and spread values.

<u>Hypotheses.</u> To evaluate the efficacy of my paradigm, I first compared whether the timing accuracy, precision, and peak rate of the human subjects differed significantly between the 6 and 20s conditions. I calculated timing accuracy as the signed difference between a

participant's peak time, and the target time, for each condition. To compare the variability of timing performance across conditions, I transformed spread scores to be on an equivalent scale by dividing a participant's spread by the mean peak time of all participants (akin to the coefficient of variation, Lejeune & Wearden, 2006). Based on previous literature, I predicted that participant-level timing accuracy, and precision, of human subjects should be equivalent across both conditions (Wearden & Lejeune, 2008). I made no predictions about peak rate as this metric has seldom been studied in humans.

Given previous reports suggesting interval timing ability is similar across species (Lejeune & Wearden, 2006), I compared the timing accuracy, precision, and peak rate of human participants to that of a sample of rats completing a 20s peak interval task. I hypothesized that peak interval metrices would not differ as a function of species. Finally, I examined the trial-by-trial performance of participants to examine whether there were differences between species in a) relative contributions of start and stop time on timing accuracy and precision (e.g. whether earlier start times differentially impacted the accuracy and precision of humans and rodents) and b) correlations between start-stop, start-spread and middle-spread. I expected start-stop time, start-spread time, and middle-spread relationships to be similar across species.

<u>Exclusion criteria</u>. Any trial in which the start time exceeded 2 times the target duration was deemed to reflect a poor understanding of the task and excluded from further analysis. Likewise, any trial in which the stop time was less than half the target duration was excluded. Using these criteria, I excluded a total of 1027 trials, leaving a total dataset of 17671 trials nested within 327 participants.

<u>Statistical Analyses.</u> T-tests and ANOVA were used to explore the extent to which peak interval performance was similar across conditions (human analysis). In the human-only analysis, condition was modelled as a within-subjects factor. To compare peak interval performance across species, I conducted an equivalence test (Lakens et al., 2018). Unlike traditional statistical tests in which the alternative hypothesis assumes there is a difference between groups, an equivalence test runs on the assumption that a lack of differences is present such that the null hypothesis probes whether group differences are big enough to matter. Equivalence tests are conducted in two steps: first, a margin of equivalence, which represents the smallest meaningful difference between group means (that is, the smallest difference which would lead one to conclude the two means are not equivalence), is calculated. Second, the 90% confidence intervals for group means are estimated and compared to the margin of equivalence. In order for two means to be considered equivalent, the 90% confidence interval of difference must fit entirely within the margin of equivalence. For this study, the margin

of equivalence was set to be equal to the mean +- 1.96*standard error of the mean (SEM) of timing accuracy, precision, and peak rate for the entire human sample respectively. As I only had data for the 20s target duration in rats, this analysis only included human data for the 20s target duration.

Multilevel models using restricted maximum likelihood were constructed to investigate the relative contributions of start, and stop, functions on timing accuracy and precision across species. I included as fixed effects: start time or stop time respectively, species, and their interaction. To account for individual differences in performance, the random effects included: variances for the intercept. Finally, to statistically compare the start-stop, start-spread, and middle-spread correlations between species, I converted *r* values to *z* scores using Fisher's *r to z* transformation.

Results

Condition effects on timing metrices.

I first tested whether the timing accuracy, precision, and peak rate of human subjects differed between the 6s and 20s conditions. Contrary to my predictions, I observed condition effects on timing accuracy (t(324)=2.19, p=0.014), timing precision t(322)=3.00, p=0.001), and peak rate (t(326)=16.54, p<0.001). Participants tended to overestimate the target duration for the 6s condition (m=1.12, SD=2.02) relative to the 20s condition (m=0.29, SD=6.64). Participants were also significantly more variable in their timing estimates for the 20s condition (m=0.95, SD=0.33) relative to the 6s condition (m=0.88, SD=0.31). Finally, I found that participants responded more vigorously on the 6s condition (m=63.49, SD=56.10) relative to the 20s condition (m=38.03, SD=42.70). These results were unexpected given previous literature indicating that timing functions remain similar across different durations of time (Wearden & Lejeune, 2008).

Influence of counting

Given the significant condition effect, I conducted a post-hoc analysis to test the validity of the distractor task to prevent counting as a way to estimate elapsed time. A series of models were constructed to test whether timing metrices differed between individuals who did, and did not, report counting during the task. There was a significant effect of counting for both timing accuracy (F(1,323)=4.47, p=0.035) and precision (F(1,321)=7.829, p=0.005). Individuals who endorsed counting were significantly *less* accurate in predicting the 6s (m=1.20, SD=2.01) and 20s (m=1.81, SD=7.08) durations compared to non-counters (m=1.10, SD=2.01; m=-0.11, SD=6.47 respectively). Despite its negative impact on timing accuracy, participants who endorsed counting were significantly more precise in their timing estimates for both the 6 (Counters: m=0.85, SD = 0.32; Non-Counters: m=0.89, SD=0.31) and 20-second (Counters :m=0.83, SD=0.34; Non-Counters: m=0.98; SD=0.32) conditions. Finally, there was no difference in the peak rate (F(1,325)=0.046, p=0.830) of participants who endorsed counting (m=49.74) compared to those who did not (m=51.03). Combined, these findings suggest that chronometric counting strategies resulted in participants producing more consistent timing estimates; however, these estimates were less accurate as counters tended to overestimate the target duration.

Condition and Counting effects in top performers

Next, I tested whether the observed effects of condition, and counting, could be due to a poor understanding of the task. To do this, I restricted the analyses to only those participants whose peak estimates were within 10% of the target duration for both conditions (n = 39). I found that timing accuracy (t(38)=1.749, p=0.044), and peak rate (t(38)=3.019, p=0.002), differed significantly between conditions. Participants tended to overestimate the peak time on the 6s condition (m=0.05) relative to the 20s condition (m=-0.29). Likewise, peak rate tended to be higher for the 6s condition (m=74.44) relative to the 20s condition (m=49.79). Timing precision did not significantly differ (t(38)=-0.517, p=0.304) between the 6s (m=0.785) and 20s condition (m=0.8156). Thus, while the top-performers demonstrated equivalent precision across conditions, in line with predictions, they significantly overestimated the peak time on the 6s condition; this overestimation may have been due, in part, to participants responding more vigorously on the 6s condition.

Effect of species on timing metrices

Next, I tested whether timing accuracy and precision differed across species. As I only had data for the 20s condition in the animal sample, and timing estimates significantly differed between conditions, only human data from the 20s duration were used in this analysis. Additionally, given the significant influence of counting on results, I included only human participants who indicated that they did not count during the experiment. Finally, to minimize the influence of poor task understanding, I restricted the analysis to include only participants who had acquired the timing task (Buhusi et al., 2022). I defined acquisition of the task as a peak time that was within 10% of the target duration (i.e. 18-22s); the final sample composed 6 rats and 143 human participants.

First, I tested whether timing accuracy, precision, and peak rate were equivalent between humans and rodents (see Figure 4 for a smoothed timing curve of participants). The margin of equivalence was set to be equal to the mean ± 1.96*SEM of timing accuracy (m=0.293, SEM=0.4164), precision (m=0.945, SEM = 0.020), and peak rate (m=35.92, SEM=2.30) of the full human sample on the 20s condition. The 90% confidence interval of the difference between group means was -2.16 to -0.548 for timing accuracy which exceeded the equivalence margins of -0.567 to 1.054 suggesting timing accuracy was not equivalent across species. Rodents tended to underestimate (m=-1.33) the target duration relative to humans (m=0.02). Likewise, for timing precision, the 90% confidence interval of difference was 0.671-1.159 which exceeded the equivalence margin of 0.9056 to 0.9846; rodents were significantly less precise (m=1.71) compared to humans (m=0.80). Finally, the confidence interval of difference for peak rate was -24.321-41.430 which exceeded the equivalence margin of 31.408-40.437 indicating that equivalence was not met; rodents responded more vigorously (m=54.83) than humans (m=46.33).

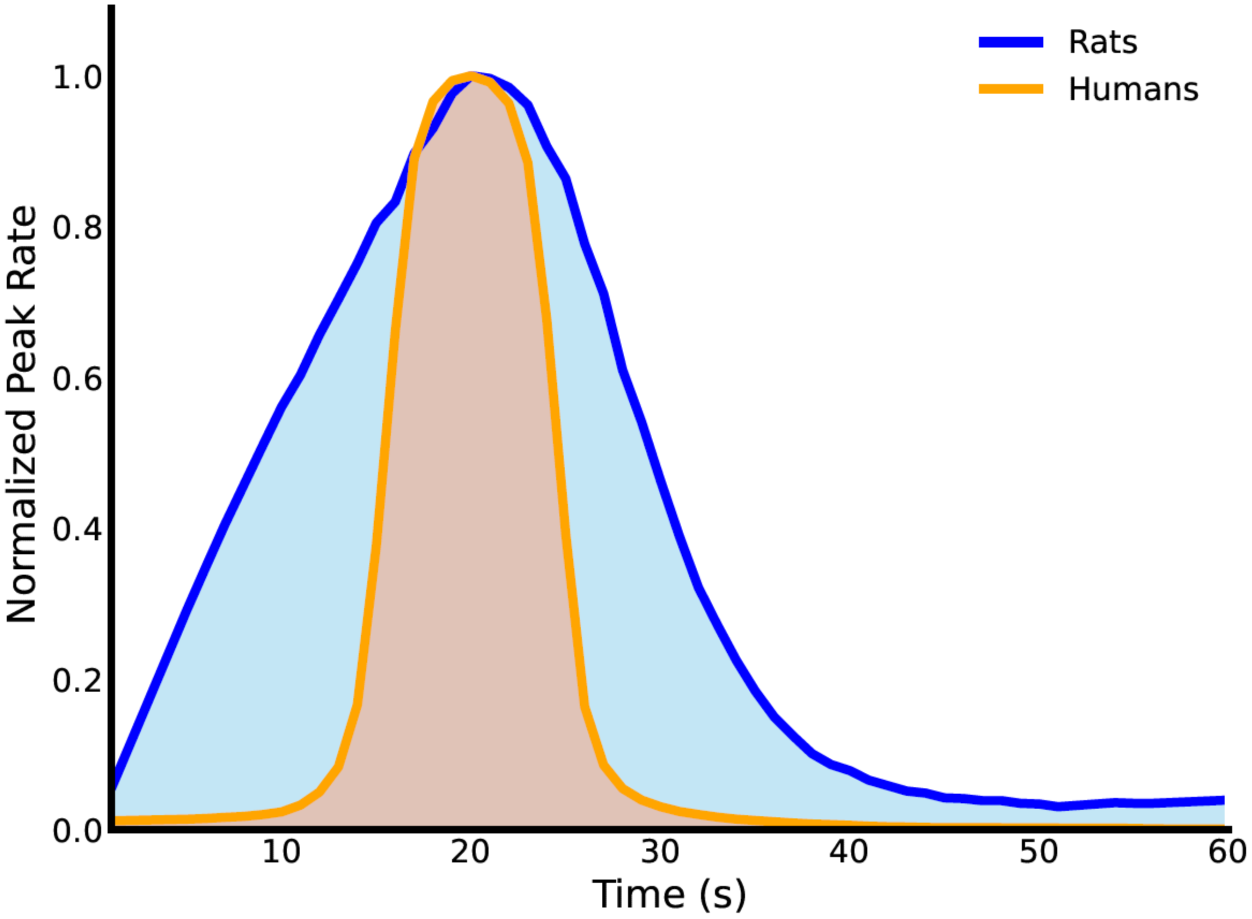

Figure 4. Smoothed timing curve of human and animal subjects. Rodents were less accurate and precise than human subjects.

To explore the cause of reduced accuracy and precision in rats as compared to humans, I constructed a series of multilevel models. First, I tested whether there was a significant effect of species on timing accuracy and precision on a trial-by-trial basis in two separate models. Trial-by-trial timing accuracy did not differ significantly between species (F(1,468)=2.477, p=0.116) whereas precision did (F(1,275)=189.654, p<0.001) such that humans were significantly more precise than rodents (m=0.224 vs m=1.031). Next, I examined whether changes in start or stop time may be driving this significantly reduced precision (Figure 5; Tables 1 and 2). I found significant main effects of species (F(1,519)=113.585, p<0.001) and start time (F(1,197)=102.763, p<0.001) but no significant species-by-start time interaction (F(1,197)=0.061, p=0.805) suggesting the influence of start time on timing precision was similar across species. The main effect of species revealed that humans (m=0.192) were significantly more precise than rodents (m=0.646) on a trial-by-trial basis. The main effect of start time revealed that each one second increase in start time predicted a 0.037 decrease in imprecision.

In the model testing the influence of stop time on timing precision, I found significant main effects of species (F(1,370)=287.537, $p<0.001$) and stop time (F(1,188)=86.918) but no species-by-stop time interaction (F(1,188)=0.350, $p=0.555$). The main effect of species revealed that humans (m=0.188) were significantly more precise than rodents (m=0.769) on a trial-by-trial basis. The main effect of stop time indicated for each one second increase in stop time, precision was reduced by 0.043 seconds. Combined, these results suggest that the significant difference in trial-by-trial precision cannot be explained solely by differences in internal timing mechanisms.

Table 1. Regression coefficients for a multilevel modeling predicting timing precision using start time

	b	*Std. Error*	F(*df*)	σ^2	*Std. Error*	*Wald z*
Fixed Effects						
Intercept	.646	.042				
Species			113.585 (1,519) ***			
Start Time	-.037	.007	102.763 (1,197) ***			
Species x Start Time			0.061 (1, 197)			
Random Effects						
Intercept				.013	.001	10.067 ***
Start Time				.001	.000	8.557***

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

Table 2. Regression coefficients for a multilevel modeling predicting timing precision using stop time

	b	*Std. Error*	F(*df*)	σ^2	*Std. Error*	*Wald z*
Fixed Effects						
Intercept	.769	.034				
Species			287.537 (1,370) ***			
Stop Time	.043	.008	85.918 (1,188) ***			
Species x Stop Time			.350 (1, 188)			
Random Effects						
Intercept				.012	.001	10.372 ***
Stop Time				.001	.000	8.662***

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

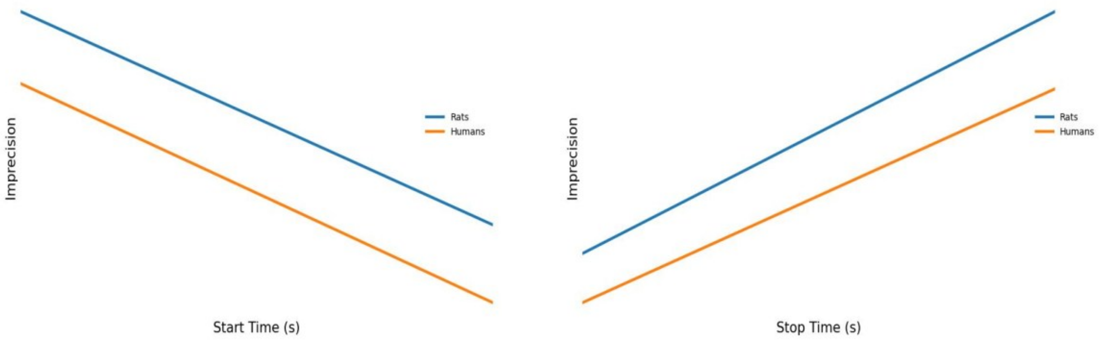

Figure 5. Earlier start times and later stop times both contributed to the significantly higher imprecision in rats.

Start-Stop Correlations

Finally, I probed the extent to which timing processes, and decisions to respond, were influencing the performance of the humans and rats separately. Start-stop, start-spread, and middle-spread correlations for the human and rat samples are shown in table 3. There were significant differences in the start-stop correlations of humans and rodents (Z=-3.19, p <0.001): start-stop values were negatively correlated in humans whereas start-stop values were uncorrelated in rodents. There were also significant differences in the start-spread correlations of humans and rodents: start times exerted a stronger effect on spread values in humans compared to rodents such that later start-times resulted in smaller spread values. Finally, middle-spread correlations differed significantly across species (Z=-2.49, p=0.006): middle-spread values were more strongly correlated in humans than rodents. That the start and stop times of the rodents were uncorrelated suggests the performance of the rodents was driven primarily by anticipation of reward, rather than an internal timing mechanism, whereas the human's performance was influenced by strategic responding that was, on some level, informed by their internal representation of time.

Table 3. Task metric correlations across species

	Start-Stop	Start-Spread	Middle-spread
Human (n = 4151)	-0.168****+	-0.788 ****+	0.684 ****+
Rat (n=115)	0.135	-0.592 ****+	0.148

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

+Significance at any statistical level in this table represents whether a correlation was significant within, not between, each group.

Peak Interval Performance in the Full Sample

Findings using the full sample can be found in Supplementary Results. Results were generally similar for both the top performers and full sample. Briefly, in both samples, human participants were more accurate and precise than rodents, whereas rodents responded more vigorously than human participants. Analysis of start and stop times in the full sample suggested the reduced precision of rodents was due to a combination of both earlier start times, and later stop times, than human participants; this finding was not replicated in the top performers. Finally, trial-level analysis of the full sample suggests human and rodent performance on the peak interval task was driven primarily by internal timing mechanisms; this stands in contrast to the top performers who demonstrate evidence of decision-variability influencing performance.

Discussion

In this study, undergraduate students with no history of mental health diagnoses or psychotropic medications completed an online peak interval task designed to measure interval timing ability. Participants were asked to learn and estimate two durations of time across multiple trials. Performance on this task was then compared to archival data of rats completing an analogous peak interval task. Performance generally conformed with the principles of interval timing (Staddon, 2005); however, some discrepancies were present. Trial-level analyses suggested these discrepancies were due to the influence of non-timing related factors. These factors will be discussed in greater detail below. Our results add to a small literature indicating similar timing processes across species (Rakitin et al., 1998) and suggest an online peak interval task could be a viable method for measuring interval timing abilities.

According to SET, there are several fundamental principles that peak interval performance should conform to were it driven by internal timing mechanisms (Wearden, 2003). First, estimates of time should, approximately, be equal to the duration timed: this is the principle of *mean accuracy* (Wearden, 2003). As timing accuracy was defined as peak time minus the target duration, this would manifest as timing accuracy remaining similar across conditions because estimates that are approximately equal to the target duration should lead to timing accuracy, in both conditions, being close to 0. The second core principle indicates that the variability of a time estimate should scale, linearly, with the duration to be timed (Gibbon, 1977). This is known as *scalar variance* (Gibbon et al., 1984). As the measurement of variability (timing precision) is a ratio of variance to peak time, scalar variance would be achieved if no significant differences in timing precision were observed. SET has also been applied to trial-level analyses with authors reporting that the onset (start time) and offset (stop time) of responding should be proportional to the duration-to-be-timed. Thus, performance which relies on an internal clock mechanism would manifest as positive correlations between start and stop times (demonstrating they are proportionally related), positive correlations between start and middle times (as the start-time should increase as the time of peak responding grows later), and positive middle-spread correlations as the variance of a time estimate should grow larger as the target duration increased (Church et al., 1994; Rakitin et al., 1998).

I observed a significant difference in timing accuracy and precision between conditions, suggesting the principles of mean accuracy and scalar variance were violated. To ensure these violations were not due to a poor understanding of task instructions, I restricted the analysis to include only subjects whose peak time was within 10% of the target duration. Consistent with the principle of scalar variance, the timing precision of these top performers did not significantly differ between the 6s and 20s durations. However, they tended to overestimate the 6s duration relative to the 20s condition. While this observation indicates that my paradigm violates the scalar principle of mean accuracy, systematic patterns of underestimating longer durations and overestimating shorter durations are not uncommon in humans (see Balcı et al. (2023)). Indeed, these patterns of under- and over-estimation during interval timing have been argued to be the results of experimental design choices (Glasauer & Shi, 2021) or might reflect the 6s and 20s representations migrating towards each other in memory (van Rijn, 2016).

Exploratory analyses (see Supplementary Results) suggest the latter option is unlikely as there were no significant interactions between counterbalancing order and target duration: participants who completed the 6s condition first were no more likely to underestimate the 20s duration than those who completed the 6s condition last. An alternative explanation is that fatigue could have influenced responding between conditions as the 20s condition was much longer than the 6s condition. This may explain why participants who completed the 20s duration first produced larger overestimates of time in the 6s condition, as fatigue would result in a greater loss of pulses in the accumulator due to difficulty in maintaining the attentional switch. In this way, the significant differences I observed across conditions may reflect the influence of fatigue and could be mitigated in future studies by implementing a longer rest period between conditions.

I also found significant differences in the performance of human and rodent subjects such that human participants were more accurate, and precise, in their peak interval performance. These results are aligned with other studies which reported that humans had more accurate, and precise, interval timing performance than rhesus monkeys (Zarco et al., 2009). Trial-level analyses indicated the differences between species were driven by differences in the relative contributions of clock/memory, and decision-making, variance on performance. Specifically, while the performance of the rodents was primarily driven by decision-making factors (as indicated by the lack of significant positive correlations between start-stop, start-spread, and middle-spread correlations), human performance was influenced by both clock and decision-making processes. The significant positive correlation between middle and spread suggests that the trial-by-trial variance of timing estimates scaled proportionally with a participant’s perception of the peak time. In other words, as the peak-time grew larger, so did the uncertainty around that estimate. Significant middle-spread associations have been observed in other studies comparing the peak interval performance of humans against other animals (Rakitin et al., 1998) and suggests human performance was more greatly impacted by variance in memory for the target duration.

The larger negative start-stop and start-spread correlations observed in the human participants suggest their performance was also significantly more impacted by decision-making variables than the rodent sample. Negatively correlated start-stop values are believed to reflect differences in decision-making thresholds (e.g. motivation) as a bias for later-start times is leading to earlier stop times, thereby affecting the variables in different directions (Gür et al., 2020). This difference in motivation may have stemmed from differences in reward offered by the paradigms: the rodent subjects received a food reward on fixed trials, which may have encouraged earlier responding (Galtress et al., 2012), whereas the human subjects received no such reward. Without an external motivator, the human participants may have favored response styles which prioritize minimal responding (e.g. late start times and early stop times). Alternatively, the instruction to create a window of responses directly around the target duration may also have discouraged earlier responding from the human participants, thereby artificially reducing noise in their timing estimates.

The increased influence of decision-making variance in humans could reflect differences in the level of feedback provided by the human and rodent peak interval paradigms. While the rodent sample only received feedback on fixed trials (i.e., when a reward was presented; ~50% of overall trials), human subjects received points relative to their timing performance on every trial. In this way, the human subjects were receiving feedback about their performance on each trial which could lead to increased timing accuracy (Saito et al., 2015; Sohn & Lee, 2013) and better precision (Montare, 1988). Notably, while the effects of feedback should manifest as proportional changes in start/stop time, participants in this study also received warnings when terminating a trial too early. In this way, the feedback may have exerted a greater influence on stop-times (as participants had more opportunity to encode the stop time into memory than rodents), thus leading to a negative relationship between start and stop times. In support of this idea, when I expanded the analyses to include all participants, rather than just the top performances who may have learned how to do the task most efficiently, the correlation between start-stop times became positive (see supplemental results), suggesting a stronger effect of memory with minimal threshold variance, which is consistent with previous research (Rakitin et al., 1998). Thus, the feedback participants received may have exerted a greater influence on stop-times, leading to a negative relationship between start and stop times.

While the significant differences observed in human and animal participants ran counter to my hypotheses, they are not entirely surprising. The pattern of differences between the human and rodent participants were similar to those observed in other studies which directly compared human and animal performance on tasks of interval timing (Zarco et al., 2009). Thus, while the species effect ran counter to my hypotheses, my data conforms to patterns observed in similar studies suggesting my online paradigm is a viable method for measuring interval timing behaviors. Furthermore, the ability to dissociate between the influence of task, and timing-based, factors (Fortin et al., 2009; Gibbon & Church, 1990) on interval timing performance and to rapidly recruit large numbers of participants online highlights several advantages of the online peak interval task.

Several limitations must be addressed. First, a large proportion of participants endorsed using chronometric counting strategies to assist with the task. Given that counting influenced both timing accuracy and precision, this raises concerns about the effectiveness of the distractor task. It is possible the significant effects I observed reflect the influence of counting, rather than interval timing ability. Second, about 40% of participants discontinued the experiment early or did not complete both timing conditions suggesting it is difficult for participants to tolerate the entire task in its current form. While dropout rates are typically high for online studies (Yetano & Royo, 2017), this rate is substantially higher than the 20% dropout rate observed in other studies (Peer et al., 2022). The higher drop-out rate observed in this study could reflect differences in compensation, as the rates reported by Peer et al. (2022) reflect studies which paid for participation; participants in the current study were compensated with course credit. Regardless of the reason, the high dropout rates suggest this task may be poorly tolerated by many individuals, thus my results may also reflect an influence of persistence on task performance. In turn, this suggests that this paradigm may be inappropriate for individuals with reduced attention and motivation (e.g. clinical samples). Future studies may wish to account for this by reducing the number of trials, testing only one time duration, or consider providing monetary incentives to promote task completion.

In summary, my findings suggest an online peak interval task is a viable paradigm for measuring human interval timing ability. While the results differed from those of rodents, trial-by-trial analyses suggested this was, in part, due to differences in experimental design. Notably, my results were similar to those of other studies testing the peak interval task in human participants.

EXPERIMENT TWO

Introduction

Having established the validity and feasibility of an online peak interval task in *Experiment 1*, I next sought to replicate and extend previous literature exploring the temporal processing abilities of individuals with psychosis. Specifically, I used the peak interval task to probe the relationship between interval timing abilities and psychosis-spectrum symptoms in a sample of undergraduate students with no formally diagnosed psychotic disorder. Given the relatively low prevalence rate of psychotic-like symptoms in the general population (Linscott & van Os, 2013), I enriched the sample for psychotic-like experiences to ensure adequate variability. I compared performance on the peak interval task between individuals reporting distressing psychotic-like experiences and those who did not to test whether disruptions in temporal processing might indicate a proneness to psychosis without the confounds of medication and social factors related to having a severe mental illness which pervade patient studies. I predicted that individuals with distressing psychotic-like experiences would be both less accurate, and precise, than controls.

Methods

Participants

Participants in this study were a subsample of the full sample recruited in *Experiment 1*. This sample was enriched for psychosis such that at least 40% of participants would be individuals who were identified as at-risk for psychosis (see below for criteria). The remaining 60% of participants were randomly selected from the remaining sample using SPSS Version 28's Random Sample Command (IBM Corp., 2021). This resulted in a total sample of 151 participants: 61 individuals who were classified as at-risk for psychosis and 90 controls.

Assessments

Measures of psychotic-like traits and experiences. Psychotic-like experiences were measured using the Prodromal Questionnaire-Brief (PQ-B; Loewy et al., 2011). The PQ-B measures psychotic-like experiences across 21-items which are rated for presence (yes/no) and distress (scored on a 5-point Likert scale). Participants who endorsed at least one psychotic-like experience as being maximally distressing were classified as at-risk (Mittal et al., 2011, 2012). The decision to classify participants as at-risk based on the endorsement of a maximally distressing psychotic-like experiences, rather than the total number of psychotic-like experiences, was based on accruing literature which suggests that the distress associated with psychotic-like experiences is more predictive of later transition to a formal psychotic disorder (Yung et al., 2006).

The Schizotypal Personality Questionnaire-Brief Revised Updated (SPQ-BRU; Davidson et al., 2016) and select subscales from the Wisconsin Schizotypy Scales (WSS; Chapman & Chapman, 1995) were used to measure psychotic-like traits. The SPQ-BRU is a 32-item self-report questionnaire which screens non-clinical populations for symptoms of schizotypal personality disorder; a collection of personality traits that are associated with increased vulnerability for schizophrenia (Lenzenweger, 2018). Scores on the SPQ-BRU can be broken into two sub-scales: cognitive-perceptual (e.g., odd perceptions and beliefs) and interpersonal (e.g., flattened affect; social paranoia). The cognitive-perceptual subscale was used as a measure of positive schizotypal traits. The WSS-short form was administered to provide a measure of anhedonic-like negative schizotypal traits. The WSS-short form consists of 60 items spread across four scales. As I was only interested in anhedonic-like schizotypal traits, I administered only two of these scales: the Revised Social Anhedonia and Revised Physical Anhedonia. Each scale consisted of 15 items. Items on the WSS-short form are scored as on a binary scale such that a 1 indicates the presence of a symptom. The scores of these two subscales were summed to create a general measure of negative schizotypy.

Measures of sleepiness. As fatigue can influence interval timing behavior (Goudini et al., 2024), I used the Stanford Sleepiness Scale (SSS) to measure a participant's level of sleepiness at the time of the experiment. The SSS is a single-item questionnaire which measures a person's current sleepiness on a scale of 1 (feeling active) to 7 (sleep onset soon). The SSS has been validated for use in adult populations aged 18 or over (Shahid et al., 2011). Sleepiness was measured after participants had completed both conditions of the experimental task.

Measures of reward sensitivity. Given that reward processes have been shown to be disrupted in people on the psychosis spectrum (Akouri-Shan et al., 2021; Simpson et al., 2012) and neurobiological data demonstrating the circuitries that modulate reward processes and interval timing have substantial overlap (e.g. dopaminergic activity in the basal ganglia; Ludvig et al., 2011; Niv, 2009), I included two measures of reward sensitivity to the test extent to which individual differences in reward sensitivity are related to timing: The Temporal Experience of Pleasure (TEPS; Chan et al., 2012), and the Sensitivity to Punishment and Reward (SPSR-Q; Torrubia et al., 2001). The TEPS is an 18-item questionnaire which asks participants to rank statements regarding anticipatory and consummatory pleasure on a 6-point likert scale. Example questions on the TEPS include: a) when I fear about a new movie starring my favorite actor, I can't wait to see it and b) I love it when people play with my hair. The Sensitivity to Punishment and Reward Questionnaire (SPRQ) is a 48-item questionnaire which measures an individual's sensitivity to reward (24 items) and punishment (24 items) on a binary scale. Example questions on the SPRQ include: a) Does the prospect of obtaining money motivate you strongly to do some things? And b) Do you like taking some drugs because of the pleasure you get from them?

Measures of substance use. I measured daily caffeine consumption and cigarette use of each participant as research suggests caffeine (Keen et al., 2024; Stine et al., 2002) and nicotine (Hinton & Meck, 1996) can exert an influence on interval timing performance. Daily caffeine consumption was measured using the following item: "how much, if anything, do you drink of the following drinks on a typical day?" with options for the following beverages: "coffee, tea, soda/pop that contains caffeine (e.g. Coca-cola) and energy drinks that contain caffeine." Response options ranged from 1 (none) to 7 (six glasses/cups or more); responses on this question were weighted in accordance with previous research (James et al., 2015; Kristjansson et al., 2015) to reflect differences in caffeine content of the various drinks. Nicotine intake was measured using a single item in which participants indicated whether they smoke and, if so, how many cigarettes they smoked per day.

Measures of mood and anxiety symptoms. Current literature suggests potential relationships between depression, anxiety, and interval timing, such that depressive symptoms can lead to an overestimation of time (Mioni et al., 2016) in a manner consistent with slower clock speed whereas symptoms of anxiety distort estimations of time (Mioni et al., 2016) in a manner consistent with the loss of pulses in the accumulator due to difficulty maintaining the attentional switch. Given these data, mood and anxiety symptoms were measured using the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001) and the Generalized Anxiety Disorder 7-item (GAD-7; Spitzer et al., 2006). The PHQ-9 has been used in a variety of settings and measures common symptoms of depression using a 4-point Likert scale. The GAD-7 is a 7-item, self-report questionnaire which assesses common symptoms of anxiety in individuals.

Experimental paradigm and procedure

Every participant completed an online peak interval task with target durations of 6s and 20s. This procedure was identical to that described in *Experiment 1*. Following completion of the peak interval task, participants completed the battery of self-report measures described above. Completion of the surveys took 35 minutes on average.

Data Analysis

Analysis Overview and Hypothesis. Timing parameters were derived using identical procedures to *Experiment 1* (see Figure 1; Supplementary Table 1). Briefly: response curves were generated separately for each participant's performance on the 6s and 20s conditions. From these curves, I derived measures of peak time (the time of maximal responding), spread (width of the curve), and peak rate (maximal response rate/second). Given previous research suggesting that persons at-risk for psychosis have significantly less accurate (Ciullo et al., 2016) and precise (Thoenes & Oberfeld, 2017) timing than controls, I predicted that differences in peak interval timing metrices would explain significant variance in the classification of at-risk status.

Second, I predicted that peak interval metrices would not only be related to psychosis-risk status but may significantly account for variation in positive and negative schizotypal traits across the sample.

Statistical Tests. Independent sample t-tests and chi-square tests were used to test whether at-risk and control groups differed on smoking status, sleepiness, caffeine intake, counting, and severity of mood and anxiety symptoms. Next, I calculated two variables: timing accuracy (the signed difference between a participant's peak time and the target time) and timing precision (spread divided by mean peak time). I tested whether these metrices would differ significantly across duration conditions. Logistic regression was used to test the extent to which timing accuracy, precision, peak rate and start-stop correlations explain variance in psychosis risk. As there were low correlations between the timing metrices, I deemed the risk of multicollinearity to be low. Thus, for each timing metric, I included estimates for each condition (e.g. 6s timing accuracy and 20s timing accuracy) in the same model. I included, as covariates, any of the potential confounding factors which differed significantly between the at-risk and control groups (e.g. sleepiness; mood symptoms).

Finally, I conducted a series of multiple regressions to examine whether timing accuracy, precision, and/or peak rate could account for variance in positive and negative schizotypal traits across the sample while controlling for any participant characteristic that significantly differed across groups (e.g., sleepiness). In cases where I observed a significant influence of peak interval metrices on schizotypal traits, follow-up mediation models were conducted to test whether the influence of timing was mediated by reward sensitivity, anxiety, and/or depression. Mediation analyses were conducted using the Hayes (2017) PROCESS macro.

All analyses were conducted using SPSS version 28. Data was preprocessed using Python version 3.12.

Results

Participant characteristics.

At-risk and control groups were matched on age, gender identity, race, smoking status, proportion of participants using psychotropic medications, caffeine use, and proportion of participants who endorsed counting (table 1). Persons at-risk for psychosis were significantly more tired and reported significantly higher levels of depression and anxiety than controls. Contrary to expectations, the at-risk group were *more* sensitive to reward.

Performance metrices.

First, I tested whether timing accuracy and precision differed significantly between the 6s and 20s conditions. Given that the proportion of participants who endorsed counting did not differ between groups (Table 4), I did not control for this factor. On the 6s condition, participants tended to significantly overestimate the target duration (t(178)=4.09, p<0.001; m=1.09, SD=1.70) relative to the 20s condition (m=-0.83; SD=5.53). Participants were also significantly less precise on the 20s condition (t(299)=2.20, p=0.014; m=0.94, SD=0.34) relative to the 6s condition (m=0.86, SD=0.31).

Table 4. Demographic and clinical information

	Psychosis-Risk (n=61)	**Control (n=90)**	**Statistics**	**P**
Age	19.28 (1.23)	19.61 (1.44)	t (149) = 1.48	0.071
Gender Identity (F/M/Other)	51/8/2	64/23/3	χ^2 = 3.625	0.305
Race (White/African American/Asian/Other)	47/4/5/5	73/5/7/6	χ^2 = 8.184	0.416
Handedness (left/right/ambidextrous)	53/7/1	82/7/1	χ^2 = 0.685	0.710
Grade (Freshman/Junior/Senior/Sophomore)	23/10/8/20	23/25/12/30	χ^2 = 3.799	0.284
Psychotropic Medication (Yes/No)	19/42	24/65	χ^2 = 0.309	0.578
Smoking status (yes/no)	2/88	5/56	χ^2 = 2.936	0.087
Caffeine Use (mg)	3.84 (4.24)	3.98 (6.80)	t (149) = 0.145	0.443
Counting (yes/no)	16/45	22/66	X^2 = 0.719	0.698

Table 4. (cont'd)

Sleepiness	3.56 (1.52)	2.93 (1.46)	t (149) = 2.534	0.006**
Depression	13.31 (6.37)	5.10 (4.87)	t (105) = 8.523	< 0.001***
Anxiety	12.66 (5.30)	5.10 (5.00)	t (149) = 8.889	<0.001***
TEPS Total	82.93 (15.15)	78.54 (17.63)	T (134) = 1.60	0.056
SPRQ_Reward	12.17 (4.65)	9.75 (5.02)	T (139) = 2.92	0.002**

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

Peak Interval metrices and their relation to psychosis risk.

I performed a logistic regression to assess whether a person's timing accuracy could explain significant variance in who would be classified as at-risk for psychosis. Given low correlations between the timing accuracy of the 6s and 20s condition, both were included in a single model. First, I constructed a basic model which contained only timing accuracy and sleepiness as a predictor. This model was significant ($X^2(3)=12.12$, $p=0.007$, pseudo $R^2=0.11$). I found significant main effects of timing accuracy on the 6s condition (Wald(1)=4.81, $p=0.028$) and sleepiness (Wald(1)=6.87, $p=0.009$). For every one-second increase in timing inaccuracy, the odds of being classified as at-risk for psychosis increased by 1.26 times. Likewise, for every one unit increase in sleepiness, the odds of being classified as at-risk for psychosis increased by 1.36. Next, I constructed a follow-up model to assess whether timing accuracy could explain psychosis risk above, and beyond, the influence of mood and anxiety symptoms. This model was also significant ($X^2(5)=73.39$, $p<0.001$, pseudo $R^2=0.52$) and correctly classified 78% of cases. The model revealed significant main effects of timing accuracy in the 6s condition (Wald(1)=4.35, $p=0.037$), depression (Wald(1)=7.30, $p=0.007$), and anxiety (Wald(1)=6.70, $p=0.010$) in explaining psychosis risk status (Table 5). According to this model, each one second increase in timing inaccuracy in the 6s condition increased the odds of being classified as at-risk by 1.32 times. Similarly, each one unit increase in depressive or anxious symptoms increased the odds of being classified as at-risk by 1.15 times.

Table 5. Logistic Regression testing ability of Timing Accuracy to predict Likelihood of Being Classified as High Risk for Psychosis

	B	**SE**	**Wald**	**df**	***p***	**Odds Ratio**	**95 % CI for Odds ratio**	
							Lower	**Upper**
Timing Accuracy (6s)	0.281	0.135	4.349	1	0.037*	1.325	1.017	1.726
Timing Accuracy (20s)	0.002	0.037	0.003	1	0.953	1.002	0.932	1.077
Sleepiness	0.210	0.158	1.752	1	0.186	1.233	0.904	1.682
Depression	0.141	0.052	7.297	1	0.007**	1.151	1.039	1.275
Anxiety	0.144	0.056	6.693	1	0.010**	1.155	1.036	1.289
Constant	-0.588	0.277	6.708	1	0.010**	0.555		

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Next, I tested the extent to which timing precision could explain which participants may be classified as at-risk for psychosis. The base model which included only timing precision for the 6s and 20s conditions, and sleepiness was significant ($X^2(3)=12.65$, $p=0.005$, $R^2=0.11$). Precision in the 6s condition (Wald(1)=4.733, p=0.030) and sleepiness (Wald(1)=5.96, p=0.015) were significantly associated with at-risk status. For each one-unit increase in timing imprecision, the odds of being classified as at-risk for psychosis increased by 3.56 times. Likewise, for each one unit increase in sleepiness, the odds of being classified as at-risk increased by 1.33 times. I constructed a follow-up model to test whether the predictive effect of timing precision remained after controlling for depressive and anxious symptoms (Table 6). This model was significant ($X^2(5)=77.20$, $p<0.001$, $R^2=0.54$) and successfully classified 77% of participants. According to this model, for every one unit increase in imprecision in the 6s condition, the relative odds of being classified as at-risk increased by 8.81 times (Wald(1)=7.51, p=0.006). Likewise, every one unit increase in depressive and anxious symptomatology increased the odds of being classified as at-risk by 1.15 and 1.17 times respectively.

Table 6. Logistic Regression testing ability of Timing Precision to predict Likelihood of Being Classified as High Risk for Psychosis

	B	**SE**	**Wald**	**df**	***p***	**Odds Ratio**	**95 % CI for Odds ration**	
							Lower	**Upper**
Timing Imprecision (6s)	2.176	0.794	7.512	1	0.006**	8.81	1.86	41.2
Timing Imprecision (20s)	0.046	0.676	0.005	1	0.946	1.05	0.28	3.94
Sleepiness	0.173	0.160	1.169	1	0.280	1.19	0.87	1.63
Depression	0.139	0.054	6.660	1	0.010**	1.15	1.03	1.28
Anxiety	0.155	0.057	7.240	1	0.007**	1.17	1.04	1.31
Constant	-0.561	0.228	6.033	1	0.014*	0.57		

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Next, I tested the extent to which peak rate was related to the odds of a participant being classified at at-risk for psychosis and found no significant relationship between peak rate and at-risk status (Table 7).

Table 7. Logistic Regression testing ability of Peak Rate to predict Likelihood of Being Classified as High Risk for Psychosis

	B	**SE**	**Wald**	**df**	***p***	**Odds Ratio**	**95 % CI for Odds ration**	
							Lower	**Upper**
Peak Rate (6s)	-0.005	0.005	0.984	1	0.321	0.995	0.986	1.005
Peak Rate (20s)	0.010	0.006	2.354	1	0.125	1.010	0.997	1.022
Sleepiness	0.178	0.155	1.315	1	0.251	1.195	0.882	1.619
Depression	0.133	0.053	6.353	1	0.012*	1.142	1.030	1.266
Anxiety	0.156	0.055	7.980	1	0.005**	1.169	1.049	1.303
Constant	-0.529	0.222	5.711	1	0.017*	0.589		

Table 7. (cont'd)

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

Finally, I tested whether there was a relationship between start-stop correlations and psychosis risk. I found no significant relationship between start-stop correlations, in either condition, and psychosis risk status (Table 8).

Table 8. Logistic Regression testing ability of Peak Rate to predict Likelihood of Being Classified as High Risk for Psychosis

	B	**SE**	**Wald**	**df**	***p***	**Odds Ratio**	**95 % CI for Odds ration**	
							Lower	**Upper**
Start-Stop Correlation 6s	0.39	1.92	0.04	1	0.84	1.48	0.03	63.99
Start-Stop Correlation 20s	-1.61	1.48	1.18	1	0.27	0.20	0.01	3.64
Sleepiness	0.19	0.15	1.50	1	0.22	1.21	0.89	1.63
Depression	0.13	0.05	6.32	1	0.01*	1.14	1.03	1.26
Anxiety	0.15	0.05	8.02	1	0.01**	1.17	1.05	1.30
Constant	0.48	1.84	0.07	1	0.79	1.61		

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

Peak Interval Timing and Schizotypal Traits.

Having established a significant relationship between peak interval performance and psychosis risk status, I constructed a series of models to test whether disrupted temporal processes may relate to specific types of psychotic-like traits.

Timing Accuracy. First, I tested whether timing accuracy was related to the presence of schizotypal traits across the sample, whilst controlling for sleepiness. Given my interest in peak interval performance, here I only interpret model results which included a significant effect of timing accuracy. The model probing the relationship between timing accuracy and positive schizotypal traits was significant ($F(5,144)=14.397$, $R^2=0.33$, $p<0.001$) and explained 33% of variance in schizotypy scores. In this model, I observed a significant effect of timing accuracy (Table 9) in the 6s, but not the 20s, condition such that, for every one second increase in timing inaccuracy, positive schizotypal scores increased by 1.25. Next, I tested whether the significant relationship between timing inaccuracy and positive schizotypal scores was mediated by a person's reward sensitivity, anxiety and depression symptoms. There was no evidence of mediation exerted by any of these variables (Table 10).

Table 9. Regression coefficients for a model probing the relationship between timing accuracy and positive schizotypal traits

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	22.128	2.186	17.807	26.449	<0.001***
6s Timing Accuracy	1.249	0.507	0.248	2.250	0.015**
20s Timing Accuracy	0.125	0.154	-0.178	0.431	0.415
Sleepiness	0.741	0.741	-0.387	1.879	0.200
Depression	0.248	0.248	-0.167	0.663	0.240
Anxiety	0.778	0.778	0.335	1.222	<0.001***

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Table 10. Regression testing the mediating effects of reward sensitivity, anxiety, and depression, on the relationship between timing accuracy and SPQ-Cognitive Scores

	Total Effect (p)	**Direct Effect (p)**	**Indirect Effect**	**Confidence Interval**		***t***	**Conclusion**
				Lower Bound	**Upper Bound**		
6s Timing Accuracy -> Depression -> SPQ-Cognitive	1.08 (0.07)	0.743 (0.15)	0.01	-0.13	0.17	0.09	No direct or indirect effect of timing accuracy
6s Timing Accuracy -> Anxiety -> SPQ-Cognitive	1.08 (0.07)	0.743 (0.15)	0.30	-0.24	0.89	1.06	No direct or indirect effect of timing accuracy
6s Timing Accuracy -> TEPS Total -> SPQ-Cognitive	1.08 (0.07)	0.743 (0.15)	-0.03	-0.24	0.12	0.41	No direct or indirect effect of timing accuracy
6s Timing Accuracy -> SPRQ-Reward> SPQ-Cognitive	1.08 (0.07)	0.743 (0.15)	0.06	-0.07	0.28	0.72	No direct or indirect effect of timing accuracy
20s Timing Accuracy -> Depression -> SPQ-Cognitive	0.71 (0.68)	0.10 (0.50)	-0.01	-0.06	0.04	0.29	No direct or indirect effect of timing accuracy

Table 10. (cont'd)

20s Timing Accuracy -> Anxiety -> SPQ-Cognitive	0.71 (0.68)	0.10 (0.50)	-0.04	-0.16	0.09	0.72	No direct or indirect effect of timing accuracy
20s Timing Accuracy -> TEPS Total -> SPQ-Cognitive	0.71 (0.68)	0.10 (0.50)	0.00	-0.03	0.04	0.12	No direct or indirect effect of timing accuracy
20s Timing Accuracy -> SPRQ-Reward-> SPQ-Cognitive	0.71 (0.68)	0.10 (0.50)	0.02	-0.02	0.09	0.63	No direct or indirect effect of timing accuracy

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

The model exploring the extent to which timing accuracy was associated with negative schizophrenia-like traits was significant ($F(5,144)=7.086$, $p<0.001$, $R^2=0.198$) and revealed a significant effect of timing accuracy in the 6s, but not 20s, condition on negative schizotypal traits (Table 11). This model revealed that for every one unit increase in timing inaccuracy, WSS scores increased by 0.44. This effect remained after controlling for sleepiness. The relationship between timing accuracy and negative schizotypal traits was not mediated by reward sensitivity, depression or anxiety symptoms (Table 12).

Table 11. Regression coefficients for a model testing the relationship between negative schizotypal traits and Timing Accuracy

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	14.721	0.944	12.855	16.588	*<0.001****
6s Timing Accuracy	0.440	0.219	0.007	0.872	*0.046**
20s Timing Accuracy	0.125	0.067	-0.007	0.256	*0.064*
Sleepiness	-0.479	0.249	-0.971	0.012	*0.056*
Depression	0.103	0.091	-0.076	0.283	*0.257*
Anxiety	0.202	0.097	0.010	0.393	*0.039**

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Table 12. Regression testing the mediating effects of reward sensitivity, anxiety, and depression, on the relationship between timing accuracy and negative schizotypal traits

	Total Effect (p)	**Direct Effect (p)**	**Indirect Effect**	**Confidence Interval**		***t***	**Conclusion**
				Lower Bound	**Upper Bound**		
6s Timing Accuracy -> Depression -> WSS-Total	0.38 (0.10)	0.25 (0.21)	0.01	-0.06	0.11	0.14	No direct or indirect effect
6s Timing Accuracy -> Anxiety -> WSS-Total	0.38 (0.10)	0.25 (0.21)	0.04	-0.06	0.18	0.75	No direct or indirect effect

Table 12. (cont'd)

6s Timing Accuracy -> TEPS Total-> WSS-Total	0.38 (0.10)	0.25 (0.21)	0.07	-0.06	0.24	0.93	No direct or indirect effect
6s Timing Accuracy -> SPRQ-Reward-> WSS-Total	0.38 (0.10)	0.25 (0.21)	0.01	-0.05	0.10	0.41	No direct or indirect effect
20s Timing Accuracy -> Depression -> WSS-Total	0.12 (0.09)	0.13 (0.04)*	-0.01	-0.04	0.02	0.54	Direct effect of Timing Accuracy
20s Timing Accuracy -> Anxiety -> WSS-Total	0.12 (0.09)	0.13 (0.04)*	-0.01	-0.04	0.01	0.51	Direct effect of Timing Accuracy
20s Timing Accuracy -> TEPS Total-> WSS-Total	0.12 (0.09)	0.13 (0.04)*	-0.00	-0.04	0.04	0.22	Direct effect of Timing Accuracy
20s Timing Accuracy -> SPSR-Q-> WSS-Total	0.12 (0.09)	0.13 (0.04)*	0.00	-0.01	0.03	0.33	Direct effect of Timing Accuracy

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

*** indicates significant at $p<0.001$

Timing Precision. In the model probing the relationship between timing precision and positive schizotypy, I found no significant effect of timing precision in either condition (Table 13).

Likewise, I found no significant relationship between timing precision and negative schizotypal traits (Table 14). Given the lack of significant effects, I did not explore any potential mediating effects of depression, and anxiety, on the relationship between timing precision and schizotypal traits.

Table 13. Regression coefficients for a model probing the relationship between Timing Precision and positive schizotypal traits

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	20.950	3.840	13.361	28.540	*<0.001****
6s Timing Precision	4.850	2.831	-0.746	10.445	*0.089*
20s Timing Precision	-1.728	2.561	-6.790	3.335	*0.501*
Sleepiness	0.643	0.587	-0.517	1.803	*0.275*
Depression	0.202	0.212	-0.217	0.620	*0.343*
Anxiety	0.853	0.225	0.408	1.298	*<0.001****

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Table 14. Regression coefficients for a model testing the relationship between Timing Precision and negative schizotypal traits

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	16.289	1.675	12.978	19.600	*<0.001****
6s Timing Precision	0.106	1.235	-2.335	2.546	*0.932*
20s Timing Precision	-1.384	1.117	-3.593	0.824	*0.217*
Sleepiness	-0.485	0.256	-0.991	0.021	*0.060*

Table 14. (cont'd)

Depression	0.077	0.092	-0.106	0.260	*0.405*
Anxiety	0.234	0.098	0.039	0.428	*0.019***

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

<u>Peak Rate.</u> In the model probing the relationship between the vigor of participant's responses (peak rate) and positive schizotypal traits, I found no significant effect of peak rate in either the 6s or 20s condition (Table 15). Likewise, there were no significant relationships between timing precision and negative schizotypal traits (WSS scores; Table 16). Given the lack of significant effect of peak rate, I did not explore any potential mediating effects of depression, and anxiety, on the relationship between peak rate and positive schizotypal traits.

Table 15. Regression coefficients for a model probing the relationship between peak rate and positive schizotypal traits

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	23.060	2.363	18.366	27.706	*<0.001****
6s Peak Rate	-0.020	0.018	-0.055	0.014	*0.247*
20s Peak Rate	0.045	0.023	-0.001	0.091	*0.055*
Sleepiness	0.588	0.588	-0.574	1.749	*0.319*
Depression	0.173	0.217	-0.256	0.601	*0.428*
Anxiety	0.889	0.227	0.440	1.338	*<0.001****

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Table 16. Regression coefficients for a model testing the relationship between Timing Precision and negative schizotypal traits

	Estimate	Standard Error	95% Confidence Interval		*P*
			Lower Bound	Upper Bound	
Intercept	15.103	1.038	13.052	17.154	*<0.001****
6s Peak Rate	-0.003	0.008	-0.019	0.012	*0.658*
20s Peak Rate	0.005	0.010	-0.015	0.025	*0.633*
Sleepiness	-0.482	0.258	-0.992	0.028	*0.028***
Depression	0.077	0.095	-0.112	0.265	*0.423*
Anxiety	0.232	0.100	0.035	0.429	*0.021***

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Start-Stop Correlations. In the model probing the relationship between the time at which a person's responding accelerated and decelerated, (start-stop correlation) and positive schizotypal traits, I found no significant effect of start-stop correlations in either the 6s or 20s condition (Table 17). Likewise, there were no significant relationships between start-stop correlations and negative schizotypal traits (table 18). Given the lack of significant effects, I did not explore any potential mediating effects on the relationship between start-stop correlations and schizotypal traits.

Table 17. Regression coefficients for a model probing the relationship between Start-Stop correlations and positive schizotypal traits

	Estimate	Standard Error	95% Confidence Interval		*P*
			Lower Bound	Upper Bound	
Intercept	20.94	7.64	5.83	36.05	*0.007***
6s Start-Stop Correlation	7.39	7.47	-7.37	22.16	*0.324*

Table 17. (cont'd)

20s Start-Stop Correlation	-4.61	5.31	-15.11	5.88	*0.386*
Sleepiness	0.76	0.59	-0.42	1.93	*0.205*
Depression	0.17	0.21	-0.26	0.59	*0.434*
Anxiety	0.87	0.23	0.42	1.32	*<0.001****

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

Table 18. Regression coefficients for a model testing the relationship between Start-Stop Correlations and negative schizotypal traits

	Estimate	**Standard Error**	**95% Confidence Interval**		***P***
			Lower Bound	**Upper Bound**	
Intercept	12.25	3.32	5.70	18.81	*<0.001****
6s Start-Stop Correlation	3.74	3.24	-2.67	10.15	*0.25*
20s Start-Stop Correlation	-0.44	2.31	-5.00	4.12	*0.85*
Sleepiness	-0.44	0.26	-0.95	0.07	*0.09*
Depression	0.07	0.09	-0.11	0.26	*0.43*
Anxiety	0.23	0.10	0.03	0.42	*0.02**

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

<u>Trial-by-trial analysis</u>.

Finally, to examine the relative contributions of clock/memory and decision-making thresholds on timing variance in individuals reporting distressing psychotic-like experiences, I calculated start-stop, start-spread, and middle-spread correlations across trials, and compared them by group and condition. On the 6s condition, individuals at-risk for psychosis had significantly smaller, negative, start-spread correlations (z=5.21, p<0.001 for 6s) and larger, positive, middle-spread correlations than controls (z=5.74, p<0.001; table 17). On the 20s condition, individuals at high-risk for psychosis had significantly smaller, negative, start-spread (z=2.29, p=0.022) and middle-spread (z=2.76, p=0.005) correlations relative to controls. These results suggest there was a greater influence of decision variance on peak interval performance in controls relative to the psychotic-like experiences, particularly in the 6s condition.

Table 19. Task metric correlations across conditions

	Start-Stop	Start-Spread	Middle-spread
6s			
psychotic-like experiences (n = 3230)	0.754***+	-0.106***+	0.160***+
Controls (n=4844)	0.750***+	-0.221***+	0.031**+
20s			
PLE (n=3230)	0.732***+	-0.319***+	-0.074***+
Controls (n=4844)	0.745***+	-0.365***+	-0.136**+

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001

+Significance at any statistical level in this table represents whether a correlation was significant within, not between, each group.

Discussion

In this study, a sample of undergraduate students who were classified as at-risk for psychosis based on self-reported psychotic-like experiences and a sample of undergraduate controls without significant psychotic-like experiences performed a peak interval task designed to isolate, and measure, the factors which influence interval timing. My findings were partly in line with an altered experience of time being associated with the experience of distressing, psychotic-like experiences. These findings replicate previous reports of altered timing mechanisms in persons with schizophrenia (Ciullo et al., 2016; Thoenes & Oberfeld, 2017) and individuals at high risk for psychosis (Osborne et al., 2021; Penney et al., 2005). Thus, my results add to a small but growing literature indicating that impairment in temporal processing could be a marker of psychosis.

Consistent with the notion of impaired temporal processes in the psychosis spectrum, I found that timing accuracy and precision were significant predictors of who would be classified as at-risk for psychosis, but only for the 6s condition. Specifically, a tendency to overestimate the 6s duration and greater imprecision when reproducing 6s durations increased the odds of a participant being classified as at-risk for psychosis. Timing inaccuracy on the 6s condition also explained significant variance in the presence of positive and negative schizophrenia-like traits across my sample. These effects remained significant after controlling for mood and anxiety symptoms suggesting the temporal processing deficits observed in individuals at-risk for psychosis do not appear to be driven by general psychological distress.

That peak interval performance on the 20s condition did not significantly explain variance in schizotypal traits or psychosis risk, was unexpected. However, it is important to consider that no other study has tested the interval timing ability of persons at-risk for psychosis with a duration of longer than 10 seconds (Osborne et al., 2021; Penney et al., 2005) and few have tested longer durations in persons with psychosis (Thoenes & Oberfeld, 2017). While speculative, it is possible that timing longer durations may recruit other cognitive processes. For example, participants could have recruited working memory processes to help retain, and track, the passage of time, rather than depending on their automatic, internal, timing systems to solve the task. In this way, the equivalent performance of the at-risk and control groups could indicate a greater ability to use compensatory strategies in the 20s condition. In support of this idea, I observed significant negative correlations between start and middle times, and start times and spread, in individuals at-risk for psychosis on the 20s condition only. These negative correlations indicate that variance around the target duration was not increasing in line with the scalar property, and that start-times were not proportionately related to peak time, which is consistent with an influence of decision-making processes on peak interval performance (see Figure 3). Thus, these data support the notion that the at-risk group appeared to be strategically responding on the 20s condition.

Alternatively, the presence of a clock/memory effect on the 6s, but not 20s, condition in individuals at-risk for psychosis could indicate that psychosis-risk status is related to a specific timing deficit which differentially impacts the perception of shorter durations. One potential explanation for such a finding would be a flickering attentional switch (Penney et al., 2005). A flickering switch is believed to be driven by a deficit in attentional control which can lead to a pattern of impaired performance on timing tasks which diminishes over time. This is because reduced attentional control would lead to an inconsistent loss of pulses in the accumulator, which are more likely to disproportionately affect shorter durations. Such an idea has research support as individuals with lesions in the cerebellum, which is believed to control the attentional switch during timing, typically show inaccurate timing for durations up to 12s in length (Gooch et al., 2010) with more pronounced deficits for shorter durations of time (Gooch et al., 2010). A growing literature indicates the cerebellum in the development of psychosis (Moberget & Ivry, 2019) as disrupted cerebellar connectivity has been observed in individuals with distressing psychotic-like experiences (Karcher et al., 2022) and individuals with schizophrenia (Peters et al., 2016). Thus, these findings suggest that disrupted cerebellar activity could disrupt temporal processing in individuals at-risk for psychosis via its impact on the attention mediated switch.

While a flickering attentional switch could account for the reduced timing precision in the psychosis-risk group, it is unclear how a flickering switch could lead to a consistent over-estimation of time. Indeed, overestimation of time is typically associated with a decreased clock speed (Drew et al., 2003), which leads to fewer pulses being accumulated in the same physical unit of time. Notably, other studies have reported evidence of slowed clock speed in individuals with psychotic-like experiences (Osborne et al., 2021). Internal clock speed is believed to be controlled by striatal dopamine as research has demonstrated that administration of dopamine antagonists leads to a consistent overestimate of time on the peak interval task (Buhusi & Meck, 2005; Drew et al., 2003). As such, the overestimation of time in the psychosis-risk group could be a consequence of abnormal striatal dopamine activity. Several avenues of research support this idea. First, current literature suggests dopamine dysfunction is present in individuals at risk for psychosis (see van Hooijdonk et al. (2022) for review). Second, the peak interval performance of the psychosis-risk group was similar to rodents who overexpressed striatal dopamine D2 receptors at a similar level to those seen in persons with schizophrenia (Ward et al., 2012). These rodents were observed to significantly overestimate the target duration, and had worse timing precision, than controls when completing the peak interval task (Ward et al., 2012). Timing accuracy, but not precision, was restored after striatal activity was normalized in these mice. Combined, these data suggest that the overestimation of time observed in the psychosis-risk group may be a consequence of striatal dopamine dysfunction.

Together, these findings suggest that timing accuracy and precision may be supported by different neural circuitries. Neurobiological data indicates an overestimation of time, such as that observed in the psychosis-risk group, is likely the result of striatal dopamine dysfunction (Ward et al., 2012) whereas increased variability in temporal processing is more indicative of cerebellar dysconnectivity (Gooch et al., 2010). Given accruing research indicating the presence of cerebellar dysconnectivity (Karcher et al., 2022) and dopamine dysfunction (van Hooijdonk et al., 2022) in individuals at risk for psychosis, future studies may benefit from exploring how these systems may interact to give rise to distressing psychotic-like experiences.

Building on this idea, I found that timing accuracy, but not precision, explained significant variance in the presence of psychotic-like traits (i.e. schizotypy) across my sample. The discrepancy between timing accuracy and precision in predicting psychotic-like traits is curious and suggests timing accuracy and timing precision may be influenced by different neurobiological systems. Indeed, considering differences in psychotic-like traits (i.e. schizotypy) and psychosis risk (i.e. distressing psychotic-like experiences) supports this idea: schizotypal traits are typically stable over time and do not necessarily engender distress (Tabak & Mamani, 2013) whereas psychosis-risk was characterized by the experience of extremely distressing psychotic-like experiences. Individuals with greater trait schizotypy tend to find psychotic-like experiences to be less impairing and frightening (Kline et al., 2012). That timing precision predicted psychosis risk, but not psychotic-like traits, thus suggests that inaccurate temporal processing for shorter durations may contribute, generally, to the unusual experiences which characterize psychosis, whereas imprecision in temporal processing determines how distressing these experiences may be. But why might reduced timing accuracy only lead to distressing experiences in the context of temporal imprecision?

One suggestion is that temporal imprecision might reduce the predictability of events in the world. While a consistent overestimation of time may lead to these events being perceived as longer than they are, an agent would be able to adapt to the difference in clock speed over time. For example, while a delayed clock speed may lead an agent to assume they have more time to cross the road than they do in reality, they may be able to adjust their expectations based on repeated experience of crossing the road. An agent with considerable variability, on the other hand, would have less opportunity to adjust their expectations as time may move very rapidly in one instance, and very slowly in another. Over time, this unpredictability is likely to lead the agent to feel out of sync with their environment, hence promoting feelings of persistent uncertainty. Feeling chronically uncertain may, in turn, lead to the agent developing a perceived lack of control or agency over their environment. A lack of agency has been related to the experience of psychological distress broadly (Keeton et al., 2008) and psychotic symptoms specifically (Krugwasser et al., 2022). However, the relationship between temporal processing and psychotic symptoms remains relatively unstudied, thus this idea is speculative and remains an avenue for future research.

The results of this experiment support the notion that temporal processing may be disrupted in individuals at-risk for psychosis (Osborne et al., 2021; Penney et al., 2005) and may contribute to the distressing hallucinations and delusions which characterize schizophrenia. I found that both timing accuracy and precision were impaired in individuals at-risk for psychosis. Results suggest that these impairments may be influenced by different neurological systems; while timing inaccuracy may reflect striatal dopamine dysfunction, timing imprecision is more likely to be driven by cerebellar dysfunction. Notably, my results suggest that, while timing accuracy relates to the experience of psychotic-like experiences, impairments in precision appear to lead to these experiences being distressing. Given that distress related to psychotic-like experiences can predict transition to a formal psychotic disorder (Sullivan et al., 2020), these results indicate that impaired timing precision could be a potential risk marker for schizophrenia.

Several limitations of this experiment must be noted. While my results suggest temporal processes may be impaired in individuals at-risk for psychosis, the total variance explained by these temporal disruptions is relatively small. Further, while I observed no significant difference in the proportion of individuals taking psychotropics between the control and psychosis-risk samples, it is possible that the dosages or type of psychotropic medication may drive differences between controls and the at-risk sample. Finally, my results reflect the interval timing abilities of undergraduate students at a large-midwestern university. Given that college attendance is associated with higher levels of family resources (Tompsett & Knoester, 2023) and social capital (Sandefur et al., 2006), my results may not be generalizable to broader clinical samples. Thus, future studies may benefit from using my task in a more diverse sample of participants.

FUTURE DIRECTIONS

Future studies can build on these findings in several ways. First, while peak rate is a commonly reported measure in rodents (Balcı & Freestone, 2020), it remains an underused and understudied metric of the peak interval task in humans. Animal literature suggests an agent's peak rate may reflect their motivational state at the time of testing. Future research exploring whether peak rate reflects similar states in human participants would allow researchers to better capitalize on the wealth of animal research which has used the peak interval task.

Second, most research exploring temporal processing in persons with psychosis has focused on durations of less than 10 seconds (see Ciullo et al. (2016); Thoenes & Oberfeld (2017) for review) which raises questions as to whether the significant differences between the 6s and 20s condition represent a genuine difference in temporal processing, the influence of cognitive factors, or an artefact of my paradigm. As such, future studies may benefit from building on these findings by exploring the temporal processing abilities of individuals at-risk for psychosis at longer durations.

Finally, while this study suggests that temporal processing may be present in individuals at-risk for psychosis and, thus, may represent a risk-factor for psychosis, this data is correlational. Whether disrupted temporal processing might predict transition to a formal psychotic-disorder remains unknown. Indeed, the transition rate for individuals with distressing psychotic-like experiences to formal psychosis is relatively low (e.g. 25%, De Pablo et al. (2021). Future studies could build on these findings by using longitudinal designs to examine whether impairments in timing accuracy, or precision, can predict the transition from at-risk status to a formal psychotic disorder.

CONCLUSION

Through a series of experiments, I developed and validated an online paradigm for measuring interval timing ability--the peak interval task—and used it to probe interval timing in people at-risk for psychosis. In *Experiment 1*, I demonstrated the feasibility and validity of an online peak interval task. In *Experiment 2*, I found that peak interval performance could predict psychosis risk status such that a tendency to overestimate the target duration, in combination with reduced timing precision, predicted at-risk group membership. These findings contribute to a larger literature indicating that temporal processing may be disrupted in individuals at-risk for, and who experience, psychosis. Drawing on neurobiological evidence, my findings suggest that striatal dopamine dysfunction, in combination with a reduced ability of the cerebellum to modulate and direct attention, may place individuals at-risk for distressing psychotic experiences through enhancing the uncertainty that a person faces when navigating events in the world. Future studies are encouraged to build on these findings by exploring whether peak interval performance can predict who transitions from psychosis-risk to a formal psychotic disorder.

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APPENDIX A: SUPPLEMENTARY RESULTS

Effect of species on timing metrices

I tested whether timing accuracy and precision differed across species. As I only collected data for the 20s condition in the animal sample, and timing estimates significantly differed between conditions, only human data from the 20s duration was analyzed. Additionally, given the significant influence of counting on my results, I included only human participants who did not count during the experiment. The human sample were significantly more accurate (t(270)=2.134, p=0.017; m=-.15, SD=6.34 vs m=-3.79, SD=2.86) and precise (t(270)=7.483, p<0.001, m=1.00, SD=0.33) than the rodents (m=1.67, SD=0.27). Finally, I found a significant effect of species on peak rate (t(339)=1.74, p=0.041) such that the peak rate of rats was greater (m=58.00) than that of humans (m=38.03).

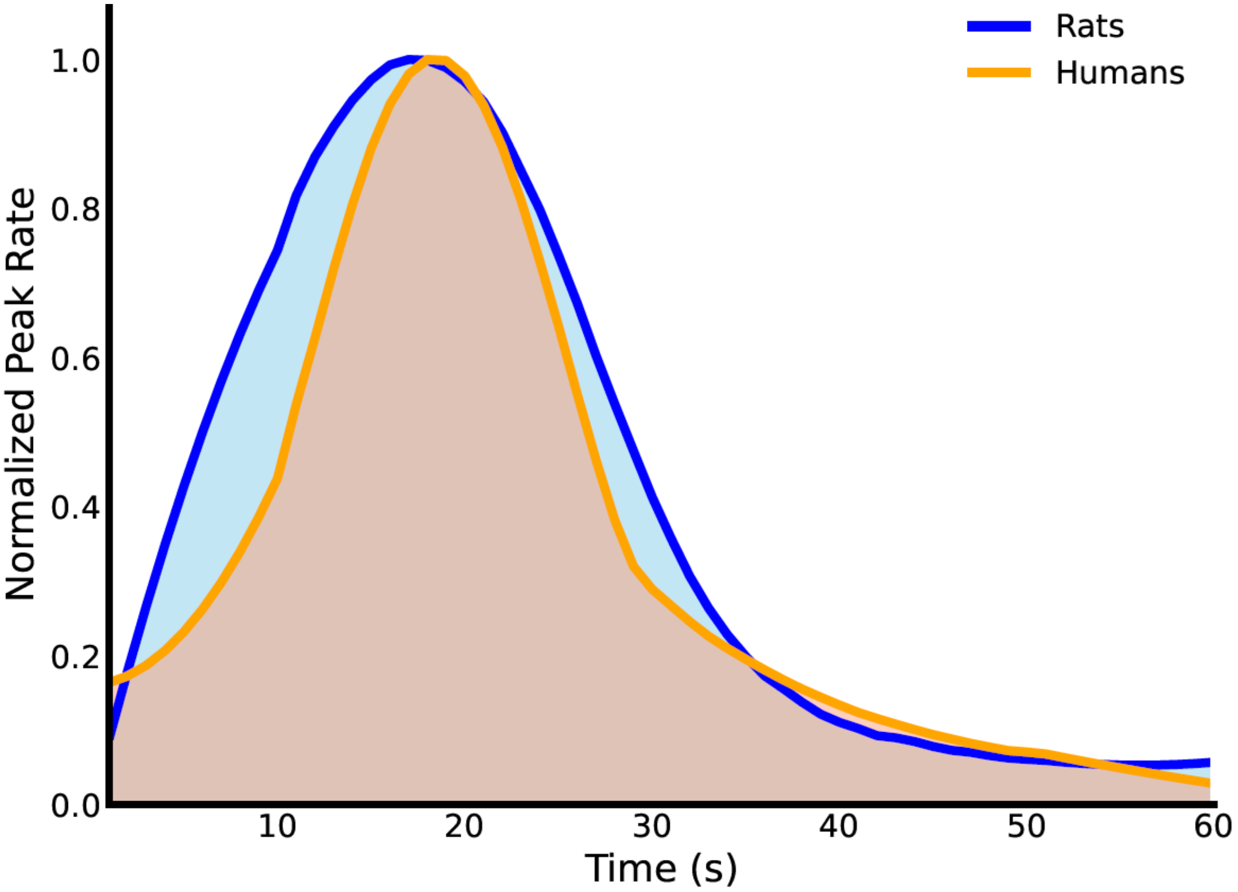

Figure S.1. Timing curve of human and animal subject. Rodents were significantly less accurate and precise than the human subjects.

To explore the cause of this reduced accuracy and precision in the animal sample, I constructed a series of multilevel models. First, I tested whether there was a significant effect of species on timing accuracy and precision on a trial-by-trial basis in two separate models. Trial-by-trial accuracy did not differ significantly between species (F(1,275)=3.488, p=0.063) whereas precision did (F(1,275)=189.654, p<0.001) such that rodents were significantly less precise than humans. Next, I examined whether changes in start or stop time may be driving this significantly reduced precision (Figure 4). The influence of start time on precision was significantly greater in rats compared to humans: each 1s increase in start time was associated with a 0.015 increase in precision in rats compared to just a 0.007 increase in humans. Likewise, the influence of stop time also differed significantly between humans and rats: a 1s increase in stop time reduced precision by 0.02s in rats compared to a 0.01 reduction in humans. Combined, these data suggest the significant trial-by-trial imprecision observed in the rats was a result of both earlier start times and later stop times.

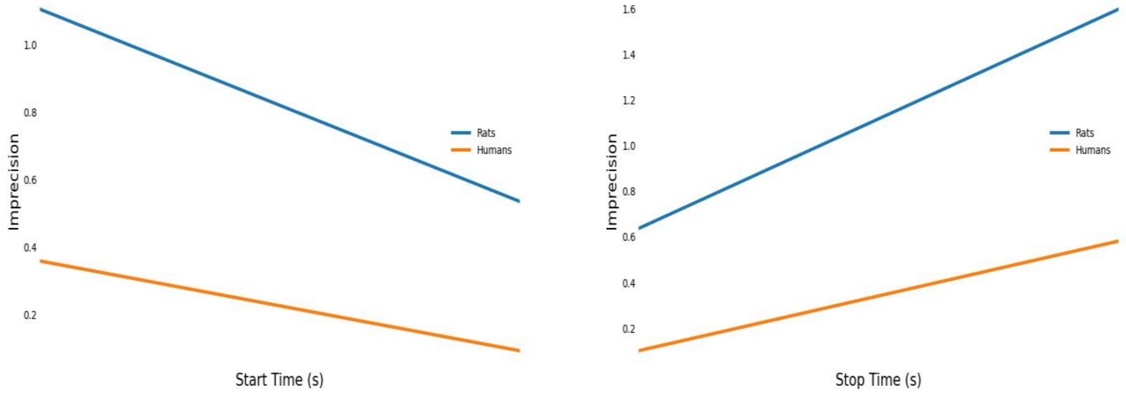

Figure S.2. Earlier start times and later stop times were contributing to the significantly higher imprecision in rats.

Start-Stop Correlations

Finally, I probed the extent to which correlations between single-trial responses may influence the behavioral responses of the humans and rats separately. Start-stop, start-spread, and middle-spread correlations for the human and rat samples are shown in table 2. There were significant differences in the start-stop (Z=4.95, p <0.001) and start-spread (Z=3.36, p < 0.001) correlations of humans and rats: humans had significantly larger positive start-stop and smaller negative start-spread correlations than the rodents. There were no such differences between middle-spread correlations (z = 1.46, p = 0.07). Reduced start-stop correlations in the context of increased negative start-spread correlations suggest that the timing performance of rats may have been influenced more strongly by decision-making thresholds (e.g. motivation) than the subject human subjects.

Counterbalance Influence

Timing Accuracy. There was a significant main effect of condition (Wilks Lambda = 0.99, F(1,325)=5.09, p=0.025) and counterbalance order (F(1,325)=4.499, p=0.035), but no significant condition-by-counterbalance order interaction (Wilks Lambda = 0.99, F(1,325)=3.211, p=0.07) on timing accuracy. Participants tended to overestimate the 6s duration relative to the 20s duration. Exploring the main effect of counterbalance order revealed that participants who completed the 20s condition first tended to overestimate target durations (m=1.18, SE=0.28) more than those who completed the 6s condition first (m=0.34, SE=0.37).

Timing Precision. There was a significant main effect of condition (Wilks Lambda = 0.97, F(1,321)=9.42, p=0.002) and counterbalance order (F(1,321)=5.74, p=0.02), and a condition-by-counterbalance order interaction (Wilks Lambda = 0.90, F(1,321)=37.72, p<0.001) on timing precision. Participants were less precise in estimating the 20s duration relative to the 6s duration. Exploring the main effect of counterbalance order revealed that participant who completed the 20s condition first (m=0.95) were less precise compared to those who completed the 6s duration (m=0.88) first. Exploring the condition-by-counterbalance order revealed an influence of counterbalance order for the 20s duration only. For the 20s duration, participants who completed the 20s duration condition first were significantly less precise (m=1.04, SE=0.03) than those who completed the 6s duration first (m=0.85, SE=0.02).

Peak Rate. There was a significant main effect of condition (Wilks Lambda = 0.77, F(1,325)=99.30, p<0.001) but no main effect of counterbalance order (F(1,321)=5.74, p=0.02) or significant interaction between counterbalance order and condition (Wilks Lambda = 1.00, F(1,325)=0.83, p=0.36) on peak rate. Participants responded more vigorously on the 6s relative to 20s conditions.

Counterbalance Influence in Top Performers

Timing Accuracy. There was a significant main effect of condition (Wilks Lambda = 0.89, F(1,37)=4.464, p=0.041) but no significant effect of counterbalance order (F(1,37)=0.819, p=0.37), and no significant condition-by-counterbalance order interaction (Wilks Lambda = 0.96, F(1,7)=1.474, p=0.232) on timing accuracy. Participants tended to overestimate the 6s duration relative to the 20s duration.

Timing Precision. There was no significant main effect of condition (Wilks Lambda = 1.00, F(1,37)=0.02, p=0.89), counterbalance order (F(1,37)=2.633,p=0.11), nor a significant condition-by-counterbalance order interaction (Wilks Lambda = 0.95, F(1,37)=1.976, p=0.17) on timing precision.

Peak Rate. There was a significant main effect of condition (Wilks Lambda = 0.82, F(1,37)=8.338, p=0.006) but no main effect of counterbalance order (F(1,321)=5.74, p=0.02) or significant interaction between counterbalance order and condition (Wilks Lambda = 0.99, F(1,37)=0.208, p=0.65) on peak rate. Top performers responded more vigorously on the 6s relative to 20s conditions.

APPENDIX B: SUPPLEMENTARY TABLE 1

Table S.1. Parameters that can be derived from the peak interval task

Metric	Participant/Trial level	How its derived	Interpretation
Peak time	Participant level	The time at which a participant's maximal responding occurs across all trials	Measures a participant's internal representation of the target duration
Precision	Participant level	The spread (standard deviation) of a participant's timing curve across all trials	Measures the noise of a participant's internal representation of the target duration
Peak rate	Participant level	The maximal rate of responding combined across all trials	Measure's a participant's motivation
Timing Accuracy	Participant level	The signed deviation between the peak time of each participant and the target duration	Measures the accuracy of a participant's representation of the target duration
Coefficient of variation (CoV)	Participant level	Precision divided by peak time	Measures the variability of a participant's timing estimates on a standard scale. CoVs of different target durations can be directly compared against each other.

Table S.1. (cont'd)

Start Time	Trial level	The time at which a participant's responding begins to rapidly accelerate on a single trial.	Represents an agent's recognition that the target duration is approaching. Earlier start times can represent impulsive responding or a change in clock speed.
Stop Time	Trial level	The time at which a participant's response rate begins to rapidly decline on a single trial.	Represents an agent's motivation to continue responding or recognition that the target duration has passed. Earlier stop times typically represent lower motivation for responding.
Start-Stop Correlations	Trial Level	Correlation between a participant's start and stop time on a single trial	Positive correlations between start and stop times indicate an influence of clock or memory effects. Negative correlations indicate the decisions to start, or stop, responding are related to non-timing factors
Start-Spread Correlation	Trial Level	Correlation between a participant's start time and their peak responding on a single trial	Positive correlations between start times and middle values indicate an influence of clock or memory as the decision to begin responding is proportionally influenced by the time at which responding peaks. Negative correlations indicate an agent may be purposefully delaying responding due to non-timing reasons.

Table S.1. (cont'd)

Middle-Spread Correlation	Trial Level	Correlation between the peak responding and difference between start- and stop times on a single trial	Positive correlations between middle and spread values indicate an influence of clock or memory as the variability of an estimate is scaling with the size of the estimate. Negative correlations indicate an agent's variability is not scaling with the duration-to-be-timed which indicates the influence of other factors (such as reduced motivation)

APPENDIX C: SUPPLEMENTARY TABLE 2

Table S.2. Task metric correlations across species

	Start-Stop	Start-Spread	Middle-spread
Human (n = 13122)	.721 ***	-.277 ***	.119 ***
Rat (n=684)	.614 ***	-.394 ***	.062

* indicates significance at p<0.05

** indicates significance at p<0.01

*** indicates significant at p<0.001