## Time dilates after spontaneous blinking

Devin Blair Terhune,[1,2,*] Jake G. Sullivan,[1] & Jaana M. Simola[3]

Accumulating evidence from pharmacology, neuroimaging, and genetics indicates that striatal dopamine influences time perception [1-5]. Despite these converging results, it is unknown whether endogenous variations in dopamine underlie transient fluctuations in our perception of time. Here, we leveraged the finding that striatal dopamine release is associated with an increase in spontaneous eye blink rate [6-8] to examine the relationship between intra-individual fluctuations in dopamine and interval timing. In two studies, participants overestimated visual subsecond and suprasecond and auditory subsecond intervals if they had blinked on the previous trial. These results are consistent with the hypothesis that transient fluctuations in striatal dopamine contribute to intra-individual variability in time perception.

Dopamine has been repeatedly linked to individual differences in time perception in the milliseconds to seconds range (interval timing) [2, 4, 5]. Dopamine agonists and antagonists produce relative overestimation and underestimation of temporal intervals, as reflected in leftward and rightward shifts of psychometric functions fitted to psychophysical data [1, 3, 9]. Convergent evidence from functional neuroimaging suggests that temporary dopamine depletion through a pharmacological manipulation reduces interval timing accuracy through attenuation in timing-specific activation in striatum [2]. Further research has implicated genetic polymorphisms associated with alterations in striatal and prefrontal dopamine with inter-individual differences in interval timing and brain morphometry in regions widely associated with timing [4].

The cumulative evidence for a role of dopamine in interval timing, however, does not offer any information regarding whether endogenous fluctuations in dopamine contribute to *intra*-individual differences in timing, namely why our perception of time varies from one moment to the next. Although intra-individual variability in interval timing has been almost wholly neglected, it undoubtedly influences performance variability in a variety of contexts requiring precise timing of the environment and it is closely intertwined with transient fluctuations in conscious states [10]. Relating dopamine to interval timing at the intra-individual level will more firmly clarify how dopamine modulates time perception. That is, if striatal dopamine phasically modulates perceived duration, then transient fluctuations in dopamine should shape intra-individual fluctuations in interval timing [3].

Spontaneous eye blinking provides an opportunity to test this hypothesis. Eye blink rate has long been associated with dopaminergic activity and is widely used as a biomarker of striatal dopamine receptor availability [6-8]. As is the case with time perception, spontaneous blinking is altered in clinical conditions characterized by aberrant dopamine levels, including Parkinson's disease and schizophrenia,

and it is responsive to pharmacological manipulations targeting dopamine [8, 9]. Further evidence specifically links spontaneous blinking to $D_2$ receptor availability in the nigrostriatal dopamine pathway [6, 8], which projects from substantia nigra to the caudate and putamen (dorsal striatum). This complements data pointing to a specific role of $D_2$ receptors in this pathway in the modulation of the speed of a putative internal clock [1]. Here we tested the prediction that participants would exhibit a leftward shift of psychophysical functions fitted to timing data, reflecting a relative overestimation of intervals, if they had blinked on the previous trial.

Participants completed visual subsecond (300-700ms) and suprasecond (1400-2600ms) temporal bisection tasks (Study 1) or an auditory subsecond (300-700ms) temporal bisection task (Study 2) whilst having their spontaneous blinks recorded by an eye tracker (for methodological details, see **Supplemental Information**). In each task, trials were coded as to whether participants blinked or not in the inter-stimulus interval preceding the judgment prompt in the *previous* trial (**Figure S1A**). We fitted post-no-blink and post-blink trials with psychometric functions and computed each participant's bisection point (BP). The BP is the temporal interval that is perceived to be equidistant to the shortest and longest comparison intervals in the task and provides a measure of the perceived duration of comparison intervals (**Figure S1B**).

As predicted, participants exhibited a leftward shift of psychometric functions on post-blink trials relative to post-no-blink trials in all three tasks (**Figure 1A-C**). This was reflected in a smaller BP (reflecting relative overestimation) in post-blink than post-no-blink trials in the visual subsecond task, $t(20)=2.44$, $p_{perm}=.008$, representing a difference of approximately one-half of a standard deviation, Cohen's $d=0.53$ [bootstrap 95% CIs: 0.29, 0.88]. This effect was also observed in the visual suprasecond task, $t(27)=2.50$, $p_{perm}=.019$, $d=0.47$ [0.10, 1.00], and in the auditory subsecond task, $t(26)=2.17$, $p_{perm}=.017$, $d=0.42$ [0.18, 0.68]. The latter effectively rules out the possibility that the observed post-blink temporal dilation is driven by blink-induced changes in visual attention or visual processing (see **Supplemental Information**). The tendency to overestimate comparison intervals in post-blink trials was present at each temporal interval in all three tasks and the leftward shift of psychometric functions, reflecting lower BPs, is readily apparent in the bootstrap resampling distributions of BPs (**Figure 1D-F**). Further analyses revealed that these effects remained when controlling for a number of potential confounding variables; in addition, participants did not differ in temporal precision between post-blink and post-no-blink trials in any of the tasks (see **Supplemental Information**).

We observed that spontaneous eye blinking, demonstrated to be a reliable biomarker of striatal dopamine receptor availability [6-8], was associated with a tendency to overestimate both visual subsecond and suprasecond and auditory subsecond intervals. These results converge with a wealth of research showing that dopamine, particularly $D_2$ receptors in the nigrostriatal pathway, contributes to

inter-individual differences in both subsecond and suprasecond interval timing [1-5]. The present work expands upon these studies by suggesting that endogenous fluctuations in striatal dopamine [6, 8] phasically modulate perceived duration, resulting in transient intra-individual variations in time perception. Increased dopamine availability may produce overestimation of temporal intervals through an acceleration of a neural oscillator [1]. According to a dominant model of interval timing [1, 3], this may occur through the modulation of the dopaminergic pulse that synchronizes the oscillations of cortical neurons at the onset of a to-be-timed stimulus. A transient increase in dopamine availability may speed up or magnify this pulse, resulting in earlier onset of the timing mechanism and thereby relative overestimation. Fluctuations in dopamine availability may underlie variance in the characteristics of this pulse and thereby introduce variability in perceived duration as computed by medium spiny neurons in striatum, which are hypothesized to be responsible for matching the duration of a comparison interval to intervals held in working memory [1, 3]. Alternatively, it is plausible that the suggested association between striatal dopamine and interval timing is mediated by a change in temporal attention (see **Supplemental Discussion**).

## Supplemental Information

Supplemental Information includes experimental procedures, further results and discussion, and two figures and can be found with this article online at *bx

## Acknowledgments

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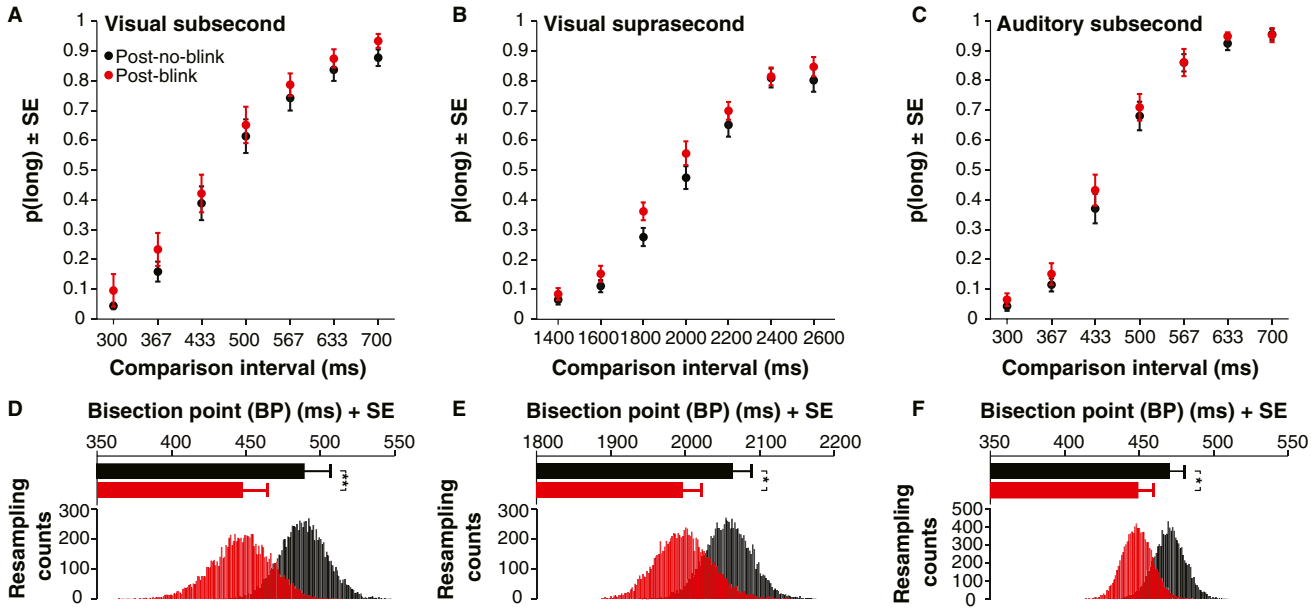


**Figure 1. Interval timing as a function of spontaneous blinking.**
(A–C) Proportion of long responses [p(long)] in trials in which the participant did not blink (post-no-blink, black) and did blink (post-blink, red) on the previous trial in (A) the visual subsecond temporal bisection task, (B) the visual suprasecond task, and (C) the auditory subsecond task. (D–F) Bisection points (BPs) (lower values reflect relative overestimation of comparison intervals) and bootstrap resampling counts (10,000 resamples) of mean BPs as a function of trial type in (D) the visual subsecond task, (E) the visual suprasecond task, and (F) the auditory subsecond task. $^{*}p < 0.05$, $^{**}p < 0.01$.

## Supplemental Information

Document S1. Experimental Procedures, further results and discussion, and two figures.

# Supplemental Information: Time dilates after spontaneous blinking

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## Author Contributions

Conceptualization, D.B.T.; Methodology, D.B.T., J.G.S., and J.M.S.; Software, D.B.T., J.G.S., and J.M.S.; Formal Analysis, D.B.T., Investigation, D.B.T. and J.G.S.; Resources, D.B.T. and J.M.S.; Writing – Original Draft, D.B.T.; Writing – Review & Editing, D.B.T., J.G.S., & J.M.S.; Funding Acquisition, D.B.T.

## Supplemental Methods

### Participants

In Study 1, 31 right-handed individuals, $M_{Age}$=23.9, *SE*=0.9, 45% female, with normal or corrected-to-normal vision consented to participate in accordance with local ethical approval. All participants had completed secondary school and had an average of 3.7±0.5 years of higher education. In Study 2, 42 right-handed individuals, $M_{Age}$=23.3, *SE*=0.7, 90% female, with normal or corrected-to-normal vision consented to participate in accordance with local ethical approval. All participants had completed secondary school and had an average of 2.8±0.4 years of higher education.

### Tasks

In Study 1 participants completed two visual temporal bisection tasks [S1] in subsecond and suprasecond interval ranges (**Figure S1A)**. They were first trained to discriminate two standard anchor intervals (a centrally-located light green circle) (subsecond: 300ms vs. 700; suprasecond: 1400ms vs. 2600) and subsequently judged whether comparison intervals (subsecond: 300ms, 367, 433, 500, 567, 633, and 700; suprasecond: 1400ms, 1600, 1800, 2000, 2200, 2400, and 2600) were closer in duration to the short or the long standard interval. Each trial consisted of a blank screen for a jittered ISI (1250-1450ms), followed by

a comparison interval (a centrally-located light green circle). The circle and background (purple-grey) were matched for luminance using a ColorCAL MkII Colorimeter (Cambridge Research Systems Ltd; Rochester, UK). After a post-stimulus jittered ISI (subsecond: 800-1200ms; suprasecond: 900-2100ms), participants were presented with a response screen (S L) and judged whether the preceding comparison interval was closer in duration to the short or the long standard interval by pressing one of two keys on a standard keyboard using their index and middle finger. Stimuli were presented at a distance of 76cm, subtending a visual angle of 1.73° × 1.73° with Experiment Builder® (v. 1.6.121; SR Research, Ontario, Canada).

In Study 2 participants completed an auditory subsecond temporal bisection tasks that was nearly identical in structure to the visual subsecond task in Study 1. Participants were first trained to discriminate two standard auditory anchor intervals (300ms vs. 700) and subsequently judged whether comparison intervals (300ms, 367, 433, 500, 567, 633, and 700) were closer in duration to the short or the long standard interval. Auditory stimuli were white noise bursts (0.5 amplitude, 44100 Hz digitization) presented via headphones; volume was individually adjusted for each participant and presented at a comfortable volume [S2]. Each trial consisted of a jittered ISI (blank gray screen; 500ms, 625, 750), followed by a comparison interval (with a blank screen). After a post-stimulus ISI (1000ms), participants were presented with a response screen (S L) and judged whether the preceding comparison interval was closer in duration to the short or the long standard interval by pressing one of two keys on a standard keyboard using their index and middle finger. Participants were seated at a distance of 70cm from the monitor and stimuli were presented with MATLAB® (2012a, MathWorks, Natick, MA).

## Eye tracking

In both Study 1 and Study 2, eye blinks and movements were recorded using an Eye Link 1000 Desktop Mount eye tracker (SR Research, Ontario, Canada). Data were monocularly sampled with the right eye at a rate of 500Hz. Participants' heads were kept in a stable position throughout the task using a chin and

forehead rest. A blink was defined as a period in which a pupil was not detected for three or more consecutive samples.

## Procedure

Participants were first seated with their head in a chin rest and the eye tracker was calibrated using a 9-point calibration routine prior to each task. The calibration was accepted if the average error was less than 0.5°. In Study 1, participants completed the two visual tasks in counterbalanced order in a dimly lit, sound-attenuated room; they completed one 20-trial training block (10 short and 10 long anchor intervals) followed by four 70-trial experimental blocks of randomized comparison intervals (40 trials of each interval). In Study 2, participants completed the auditory task wearing headphones in a room that was not sound-attenuated, but which had minimal environmental noise; they completed one 20-trial training block (10 short and 10 long anchor intervals) followed by six 49-trial experimental blocks of randomized comparison intervals (49 trials of each interval). Finger-response mappings in all three tasks (e.g., index finger=short, middle finger=long) were counterbalanced across participants. Participants were encouraged to blink normally and were naïve to the predictions. One participant was unable to complete the visual subsecond task, resulting in available data for 30 and 31 participants in the visual subsecond and suprasecond tasks in Study 1, respectively.

## Analyses

All analyses were performed in MATLAB® (2014a, MathWorks, Natick, MA). Each trial was coded as to whether a participant blinked or not in the ISI preceding the judgment screen on the *previous* trial (**Figure S1A**); data were subsequently grouped into post-no-blink and post-blink trials, as done in a previous study of cognitive control [S3]. We did *not* code trials according to whether a participant blinked or not during the pre-stimulus ISI on the *current* trials because blinks during this interval may elicit saccades, which are known to distort time perception [S4, S5], and such effects may carryover to the

stimulus period (**see Supplemental Discussion**). The present coding approach circumvents these potential confounds. We did not exclude trials in which participants blinked during pre-stimulus ISI or during the stimulus on the current trial so as to minimize the exclusion of trials and strengthen the robustness of the psychophysical results. We justify this assumption by controlling for spontaneous blink frequency during different trial phases in a series of ANCOVAs (**see Supplemental Results**).

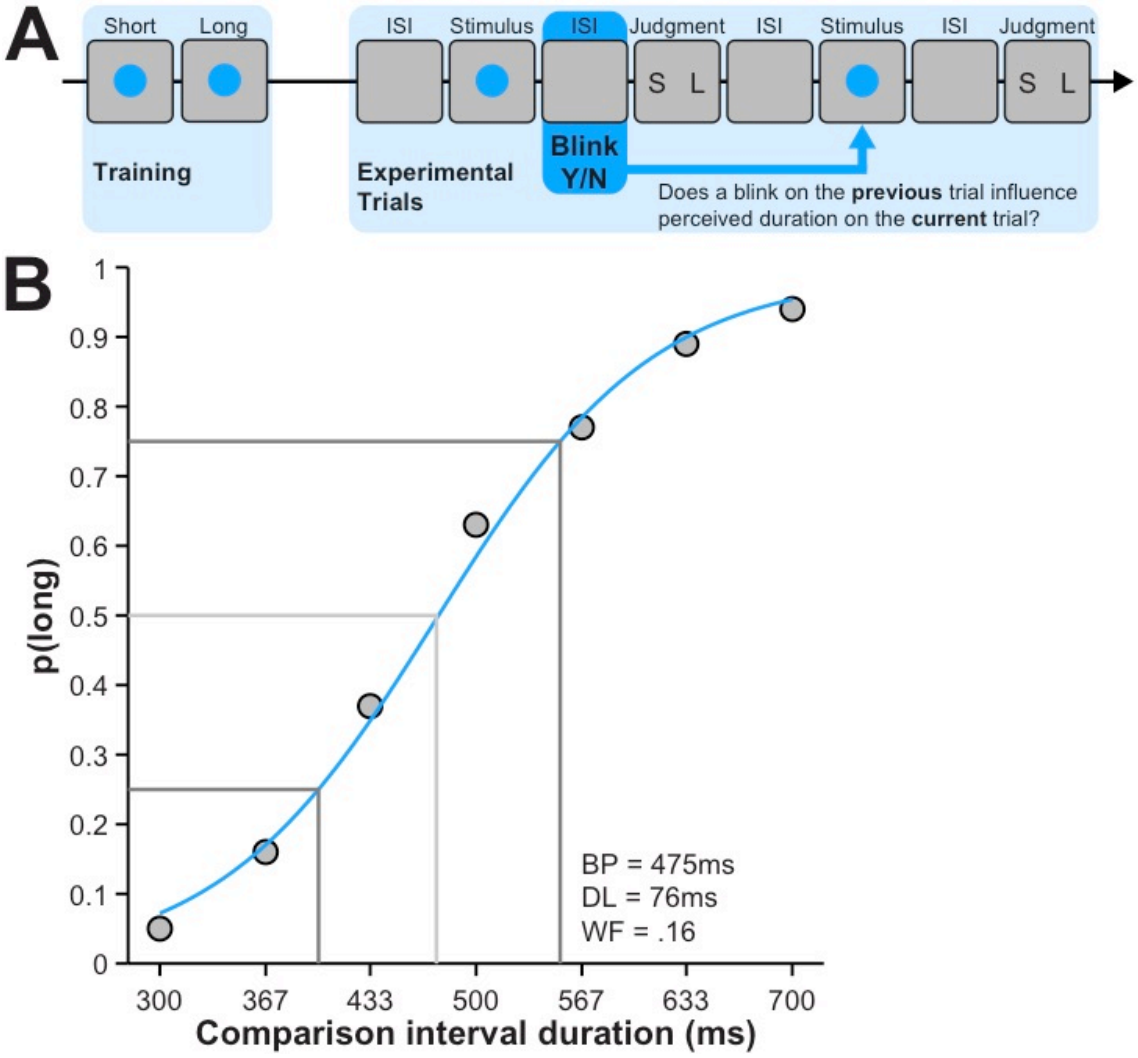


**Figure S1. Task structure and psychophysical data fitting.**
(A) Schematic diagram of the visual temporal bisection tasks and the coding of post-no-blink and post-blink trials; (B) Example fit (blue line) of simulated data (gray circles) in the visual subsecond temporal bisection task; the bisection point (BP) is identified by the intersection of the logistic function fitted to the data and the p(long)=0.5 threshold (light gray line); the *difference limen* (DL) is identified by halving the difference between the comparison intervals corresponding to p(long)=0.75 and p(long)=0.25 (dark gray lines); the *Weber fraction* (WF) is identified by dividing the DL by the BP (smaller values reflect superior temporal precision).

Data for each trial type were independently modeled using the Palamedes toolbox [S6] for MATLAB. The proportion of long responses (p(long)) at each comparison interval were fitted with logistic functions defined by four parameters: threshold $\alpha$, slope $\beta$, guess rate $\gamma$, and lapse rate $\lambda$. We fixed $\gamma$ at 0 because of the 2-alternative forced-choice response format and $\lambda$ at 0.1 to allow for occasional lapses. $\alpha$ and $\beta$ were set as free parameters and estimated using maximum likelihood estimation. Three psychometric parameters were computed **(Figure S1B)**: the duration corresponding to the 50% threshold on the psychometric function was used as the *bisection point* (BP), which corresponds to the estimated comparison interval that is perceived as equidistant to the standard anchor intervals (lower values reflect relative overestimation of comparison stimulus intervals). Temporal precision was computed with two separate measures: the *difference limen* (DL) and the *Weber fraction* (WF). The former is computed by: $(t_{p(\text{long})=0.75} - t_{p(\text{long})=0.25})/2$, where $t$ is the comparison interval duration at the respective location on the fitted psychometric function, whereas the latter is the DL divided by the BP (in both cases, *lower* values reflect *superior* precision or less variability).

To increase reliability in the analyses of psychometric parameters as a function of trial type, we excluded participants who blinked on fewer than 10% of trials, resulting in 21 and 28 participants in the visual subsecond and suprasecond tasks in Study 1, respectively. In Study 2, 4 participants' data were excluded because of poor model fit (*pdev*s<.05; [5]) and/or outlying values ($M$±3 $SD$s); of the remaining 38 participants, 27 blinked on 10% or more trials. After these exclusions, participants blinked on slightly fewer than half of the previous trials (during the post-stimulus ISI) in the visual subsecond task, 40% [CIs: 30, 52], and in the visual suprasecond task, 45% [CIs: 37, 53] in Study 1, and approximately one third of previous trials in the auditory subsecond task, 33% [27, 42] in Study 2. Aside from the latter task, which may have elicited a lower spontaneous blink rate because of reduced demand on visual attention, these proportions are roughly equivalent to the spontaneous blink rate (42%) in a previous study examining trial-by-trial effects of spontaneous blinking on cognitive control [S3]. Blink frequency during other phases of the trial were relatively consistent across tasks with a range of 40-64% during the pre-stimulus ISI (Study1: visual subsecond: 50% [39, 62]; visual suprasecond: 64% [57, 71]; Study 2:

auditory subsecond: 40% [31, 51]), 6-24% during the comparison interval (Study 1: visual subsecond: 6% [2, 25], visual suprasecond: 24% [17, 33]; Study 2: auditory subsecond: 10% [7, 16]), and 8-20% during the judgment prompt (Study 1: visual subsecond: 8% [6, 11]; visual suprasecond: 12% [10, 16]; Study 2: auditory subsecond: 20% [16, 24]).

Data in the visual subsecond and suprasecond tasks in Study 1 were analyzed separately because of unequal participant numbers in each task. Data from all three tasks were analyzed using paired-samples *t*-tests (post-no-blink vs. post-blink trials), with two-tailed *p*-values computed using permutation analysis (10,000 samples) [S7]. Cohen's *d*s were computed for all analyses to provide a measure of effect size; bootstrap resampling was used to estimate 95% confidence intervals for effect sizes and other measures [S10,000 samples; bias-corrected and accelerated method; 8] and to compute bootstrap resampling distributions for mean BPs (**Figures 1D-F**).

## Supplemental Results

*Temporal precision*

**Figure S2** presents the two measures of temporal precision (DL and WF) as a function of blink trial type (*lower* values reflect *superior* temporal precision). Participants did not display differential DL values between post-blink and post-no-blink trials in the visual subsecond task, $t(20)=0.22$, $p_{perm}=.93$, $d=0.05$ [CIs: -0.65, 0.36], visual suprasecond task, $t(27)=1.23$, $p_{perm}=.24$, $d=0.23$ [CIs: -0.14, 0.59], or in the auditory subsecond task, $t(26)=1.05$, $p_{perm}=.34$, $d=0.20$ [CIs: -0.22, 0.41]. Similarly, WF values did not differ across blink trials in the visual subsecond task, $t(20)=0.92$, $p_{perm}=.60$, $d=0.20$ [CIs: -0.40, 0.43], visual suprasecond task, $t(27)=1.60$, $p_{perm}=.12$, $d=0.30$ [CIs: -0.06, 0.67], or in the auditory subsecond task, $t(26)=1.09$, $p_{perm}=.20$, $d=0.21$ [CIs: -0.11, 0.45]. Effect sizes ranged from 5% to 30% of a standard deviation although the direction of numerical differences was uniform across tasks (poorer temporal precision during post-blink trials; see **Figure S2**). Cumulatively, these results suggest that, unlike perceived duration, temporal precision does *not* differ after spontaneous blinking.

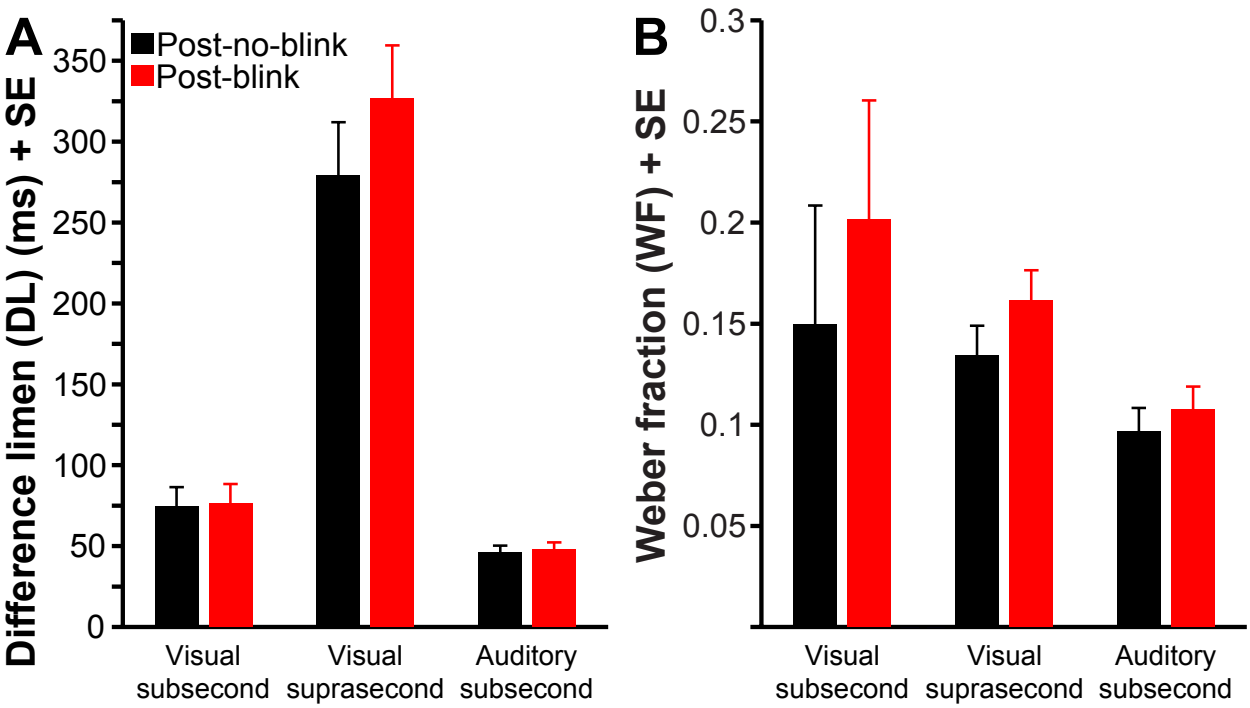


**Figure S2. Temporal precision as a function of spontaneous blinking.**
Psychophysical measures of temporal precision [(A) difference limens (DLs) and (B) Weber fractions (WFs) (lower values reflect greater temporal precision)] as a function of trial type in the visual subsecond, visual suprasecond, and auditory subsecond temporal bisection tasks. Neither measure varied across trial types in any of the three tasks.

*Spontaneous blink rate cutoff*

The primary analyses were performed on participants who blinked on 10% or more trials to ensure a sufficient number of post-blink and post-no-blink trials for the fitting of psychometric functions (see **Supplemental Methods**). Although the full data that includes all participants is less reliable, we repeated the analyses on BPs to ensure that the observed effects were not unique to participants who blinked frequently. When the analyses were repeated on the entire data set in the visual subsecond task ($n$=30), the results remained significant for perceived duration, BP: $t(29)=2.28$, $p=.018$, $d=0.42$ [0.14, 0.70], corroborating the tendency to overestimate comparison intervals after blinking. The results remained stable when a more liberal inclusion criterion of 5% or more blink trials was used ($n$=26), $t(25)=2.64$, $p=.005$, $d=0.52$ [0.30, 0.84]. Similarly, when all participants were included in the analysis of the visual suprasecond task (except a single outlier, $M\pm3$ $SD$s; $n$=30) (this was equivalent to a 5% cut-off inclusion), the results were replicated for BP, $t(29)=2.11$, $p=.044$, $d=0.38$ [0.02, 0.90]. The results in the auditory

subsecond bisection task did not change when a more liberal blink rate cutoff criterion was used (5%) ($n$=31) (this was equivalent to all participants with acceptable psychometric fits). In particular, BPs were lower in post-blink than post-no-blink trials, $t(30)=2.06$, $p=.029$, $d=0.37$ [0.13, 0.61].

*Sequential effects and the influence of spontaneous blinking during other phases of the trial*

Interval timing is influenced by the preceding stimulus duration [S2] and it is possible that the comparison stimulus interval on the previous trial represents a confounding, or mediating, influence on the association between spontaneous blinking and perceived duration. Similarly, it is plausible that blinks during other phases of the previous or current trial may have impacted the results (see **Fig. S1A**). To address these possible confounds, we partialled out the variance in BPs attributable to differences between post-no-blink and post-blink trials in terms of (1) the mean comparison interval on the previous trial, (2) the blink frequency during the judgment prompt on the previous trial, (3) the blink frequency during the pre-stimulus ISI on the current trial, and (4) the blink frequency during the comparison interval on the current trial. In all cases, we computed the difference in these measures between post-no-blink and post-blink trials and included these difference measures as covariates in a series of one-way repeated-measures analyses of covariance (ANCOVAs) examining BP differences between blink trials. The main effect of blink trial (post-no-blink vs. post-blink) on BPs, reflecting relative underestimation in post-blink than post-no-blink trials, remained significant or suggestive in all but one analysis in one task.

The first set of analyses controlled for blink trial differences in the mean comparison interval on the previous trial as a covariate. In the visual subsecond temporal bisection task (Study 1), there was no main effect of Blink trial on BPs, $F(1,19)=0.96$, $p=.34$, $\eta_p^2=.05$, with no effect of the covariate on BPs, $F(1,19)=2.72$, $p=.12$, $\eta_p^2=.13$ (and no interaction, $F(1,19)=1.64$, $p=.22$, $\eta_p^2=.08$). In contrast, in the visual suprasecond task (Study 1), the main effect of Blink trial on BPs remained suggestive, $F(1,26)=3.16$, $p=.087$, $\eta_p^2=.11$, with no effect of the covariate, $F(1,26)<0.01$, $p=.97$, $\eta_p^2<.01$ (and no interaction, $F(1,26)=0.47$, $p=.50$, $\eta_p^2=.02$). Similarly, in the auditory subsecond task (Study 2), the main effect was significant, $F(1,25)=5.38$, $p=.029$, $\eta_p^2=.18$, with no effect of the covariate, $F(1,25)<0.01$, $p=.96$, $\eta_p^2<.01$

(and no interaction, $F(1,25)=0.82$, $p=.37$, $\eta_p^2=.03$). These results show that the observed differences in perceived duration of comparison intervals between post-no-blink and post-blink trials are not an artifact of blink-related differences in the comparison interval on the previous trial.

The next set of analyses controlled for blink trial differences in blink frequency during the judgment prompt of the previous trial. The main effect of Blink trial on BPs was significant in the visual subsecond task (Study 1), $F(1,19)=5.31$, $p=.033$, $\eta_p^2=.22$ (with no effect of the covariate, $F(1,19)=0.40$, $p=.54$, $\eta_p^2=.02$, and no interaction, $F(1,19)=0.18$, $p=.68$, $\eta_p^2=.01$), and in the visual suprasecond task (Study 1), $F(1,26)=7.05$, $p=.013$, $\eta_p^2=.21$ (with no effect of the covariate, $F(1,26)=2.40$, $p=.13$, $\eta_p^2=.08$, and no interaction, $F(1,26)=0.99$, $p=.33$, $\eta_p^2=.04$), and suggestive in the auditory subsecond task (Study 2), $F(1,25)=3.24$, $p=.084$, $\eta_p^2=.12$ (with no effect of the covariate, $F(1,25)=2.40$, $p=.13$, $\eta_p^2=.09$, and no interaction, $F(1,25)=0.97$, $p=.34$, $\eta_p^2=.04$). These results suggest that the observed effect of lower BPs in post-blink than post-no-blink trials is independent of possible differences between spontaneous blinking during the judgment prompt of the previous trial between post-blink and post-no-blink trials.

The next set of analyses controlled for blink trial differences in blink frequency during the pre-stimulus ISI on the current trial. The main effect of Blink trial on BPs was significant in the visual subsecond task (Study 1), $F(1,19)=6.33$, $p=.021$, $\eta_p^2=.25$ (with no effect of the covariate, $F(1,19)=0.97$, $p=.34$, $\eta_p^2=.05$, and no interaction, $F(1,19)=0.67$, $p=.43$, $\eta_p^2=.03$), and the visual suprasecond task (Study 1), $F(1,26)=4.68$, $p=.040$, $\eta_p^2=.15$ (with no effect of the covariate, $F(1,26)=2.98$, $p=.10$, $\eta_p^2=.10$, and no interaction, $F(1,26)=1.08$, $p=.31$, $\eta_p^2=.04$), and suggestive in the auditory subsecond task (Study 2), $F(1,25)=3.60$, $p=.069$, $\eta_p^2=.13$ (with no effect of the covariate, $F(1,25)=0.09$, $p=.76$, $\eta_p^2<.01$, and no interaction, $F(1,25)=0.27$, $p=.61$, $\eta_p^2=.01$). These results, again, suggest that the observed effect of lower BPs in post-blink than post-no-blink trials is independent of possible differences in spontaneous blinking during the pre-stimulus ISI on the current trial between post-blink and post-no-blink trials.

The final analyses controlled for blink trial differences in blink frequency during the comparison interval on the current trial. The main effect of Blink trial on BPs was significant in the visual subsecond task (Study 1), $F(1,19)=6.49$, $p=.020$, $\eta_p^2=.26$ (with no effect of the covariate, $F(1,19)=0.01$, $p=.91$,

$\eta_p^2$<.01, and no interaction, $F(1,19)=0.69$, $p$=.42, $\eta_p^2$=.04). The effect was also suggestive in the visual suprasecond task (Study 1), $F(1,26)=3.58$, $p$=.070, $\eta_p^2$=.12, although there was also an effect of the covariate on BPs, $F(1,26)=5.88$, $p$=.023, $\eta_p^2$=.19, reflecting a positive correlation between blink trial differences and mean BPs (across blink trials), $r(28)=.43$, $p$=.023. This suggests that participants who blinked more often during the comparison interval on the current trial in post-blink than post-no-blink trials tended to exhibit *larger* BPs across blink trial types (there was no interaction, $F(1,26)=0.07$, $p$=.80, $\eta_p^2$<.01). Finally, the main effect of Blink trial on BPs was significant in the auditory subsecond task (Study 2), $F(1,25)=4.65$, $p$=.041, $\eta_p^2$=.16 (with no effect of the covariate, $F(1,25)=0.17$, $p$=.69, $\eta_p^2$=.01, and no interaction, $F(1,25)=0.19$, $p$=.67, $\eta_p^2$<.01). These results suggest that the observed effect of lower BPs in post-blink than post-no-blink trials is independent of spontaneous blinking during the comparison interval on the current trial. The significant main effect of the covariate in the suprasecond visual task points to an independent association between spontaneous blinking during the current stimulus and relative *underestimation* of comparison stimuli. It is plausible that this effect is driven by visual suppression perhaps in a manner similar to the impact of saccades on time perception [S4, S5]. However, this effect was not observed in the other two tasks and thus should be treated with caution.

The ANCOVAs presented above strongly suggest that blink-related BP differences are not driven by a range of possible confounding factors, but they do not conclusively demonstrate this as the analyses partialled out the influence of covariate difference scores from mean BPs (*across* post-no-blink and post-blink trials) rather than from BP *differences* (*between* post-no-blink and post-blink trials). For this reason, we performed a second set of analyses in which we corrected BP difference scores (BPdiff) by difference scores of the four covariates (CVdiff) above, as described elsewhere [S9]. This method partials out the variance in BPdiff scores attributable to CVdiff by adjusting the BPdiff scores with the product of the slope of the regression of CVdiff on BPdiff and mean-centered CVdiff scores, as follows:

$$\mathrm{rBPdiff}_i = \mathrm{BPdiff}_i + b \times (\mathrm{CVdiff}_i - \Sigma\mathrm{CVdiff}/N)$$

where $\mathrm{rBPdiff}_i$ = regressed BP difference for participant $i$; $\mathrm{BPdiff}_i$ = original BP difference for participant $i$; $b$ = slope of the regression of CVdiff on BPdiff across all participants; $\mathrm{CVdiff}_i$ = original CV difference

for participant *i*; ΣCVdiff/*N* = mean CVdiff of all participants. We computed adjusted BPdiff scores for each of the four covariates described above for each of the three tasks and analyzed BPdiff scores using one-sample *t*-tests. BPdiff scores were reliably significantly or suggestively different from 0 in all three tasks when adjusted for blink trial differences in the mean comparison interval of the previous trial (Study 1: visual subsecond: *M*=40, *SE*=20, *t*(20)=2.05, *p*=.054; visual suprasecond: *M*=60, *SE*=26, *t*(27)=2.30, *p*=.029; Study 2: auditory subsecond: *M*=22, *SE*=10, *t*(26)=2.19, *p*=.038), blink frequency during the judgment prompt of the previous trial (Study 1: visual subsecond: *M*=40, *SE*=17, *t*(20)=2.37, *p*=.028; visual suprasecond: *M*=60, *SE*=24, *t*(27)=2.45, *p*=.021; Study 2: auditory subsecond: *M*=22, *SE*=11, *t*(26)=1.98, *p*=.058), blink frequency during the prestimulus ISI on the current trial (Study 1: visual subsecond: *M*=40, *SE*=16, *t*(20)=2.47, *p*=.023; visual suprasecond: *M*=60, *SE*=26, *t*(27)=2.27, *p*=.032; Study 2: auditory subsecond: *M*=22, *SE*=10, *t*(26)=2.08, *p*=.048), and blink frequency during the comparison interval on the current trial (Study 1: visual subsecond: *M*=40, *SE*=16, *t*(20)=2.46, *p*=.023; visual suprasecond: *M*=60, *SE*=25, *t*(27)=2.39, *p*=.024; Study 2: auditory subsecond: *M*=22, *SE*=10, *t*(26)=2.15, *p*=.041). These results are consistent with the Blink type × Covariate interactions in the ANCOVAs above, which reflect the correlations between BPdiff and CVdiffs and which were uniformly non-significant and small in magnitude ($\eta_p^2$ range: <.01-.08; *M*=.028±.01). Together these results indicate that blink-related changes in perceived duration are not reliably related to blink-related changes in the foregoing covariates and that post-blink temporal dilation is independent of blink-related differences in these covariates.

## Supplemental Discussion

Here we observed that participants exhibited leftward shifts of psychometric functions fitted to psychophysical timing data in temporal bisection tasks, reflecting relative overestimation of comparison intervals. This effect was observed for visual subsecond and suprasecond intervals (Study 1) and auditory subsecond intervals (Study 2). The effect sizes ranged from approximately one third to one half of a

standard deviation shift in perceived duration. The magnitude of the effect appeared to be smaller in the auditory task, which may be due in part to superior temporal precision for auditory than visual interval timing [S10, S11]. In contrast, participants did not appear to differ in temporal precision after blinking, thus suggesting that the effects of spontaneous blinking on interval timing are specific to perceived duration.

Cumulatively these results suggest that transient fluctuations in striatal dopamine receptor availability, as indexed by spontaneous blinking [S12], are related to perceived duration of temporal intervals across interval ranges and sensory modalities. Spontaneous blink rate was previously found to positively correlate with $D_2$, but not $D_1$, receptor availability in ventral striatum, caudate nucleus, and putamen [S12]. This suggests that intra-individual variability in interval timing is specifically associated with $D_2$ receptor availability, although a role for $D_1$ cannot be entirely ruled out at this stage. Given the aforementioned imaging results, we are unable to specify which regions of striatum are specifically involved in post-blink temporal dilation, although putamen is the striatal region that is arguably most consistently activated during interval timing when controlling for other cognitive factors [S13]. Furthermore, despite the consistency of the present results, we maintain that it is unlikely that fluctuations in striatal dopamine are the sole factor contributing to intra-individual variability in interval timing. Rather, it is likely that it is one of multiple factors, including variability in sensory processing and working memory and coordination between striatum and prefrontal and cerebellar regions, that contribute to intra-individual variability in interval timing.

One outstanding question is whether the observed relationship between spontaneous blinking and interval timing is influenced by the frequency of spontaneous blinking. Specifically, if the spontaneous blink rate linearly relates to striatal dopamine release [S12], the magnitude of post-blink temporal dilation may scale with the spontaneous blink rate during individual post-stimulus ISIs on the previous trial. This analysis presents a number of challenges and was not pursued in the present study because the spontaneous blink rate in humans is relatively low (approximately 18/min [S14]) and thus there are relatively few trials in which participants will blink multiple times in a short time window (inter-blink

interval is typically around 4s [S14]). One way of addressing this question would be to expand the duration of post-stimulus ISIs and to include a larger number of trials. We would expect that the magnitude of the post-blink temporal dilation effect would increase as spontaneous blink rate increases.

Another issue that warrants further attention is the temporal window of the association between spontaneous blinking and perceived duration. The window in which we coded for blinks (the post-stimulus inter-stimulus interval (ISI) of the preceding trial) was *M*=1000ms in the visual subsecond (Study 1) and auditory subsecond (Study 2) tasks and *M*=1500ms in the visual suprasecond task (Study 1). This may account for the marginally higher spontaneous blink rate in the latter but it does not appear to have affected the magnitude of the observed difference in interval timing. In contrast, the pre-stimulus ISIs on the current trial were *M*=1350 in both tasks of Study 1 and *M*=625 in the auditory subsecond task of Study 2. Insofar as the effect sizes for the blink-associated temporal dilation were comparable across the three tasks, there is no clear evidence that this variability influenced the results. Cumulatively, these design parameters suggest that the impact of blinking on interval timing is present with a lower temporal window of approximately 1000ms and an upper window of at least 3000ms (considering response times for judgment prompts), however which temporal window is optimal to observe this effect is unclear as are the lower and upper bounds of this window. Neuroimaging research has not yet been able to determine the temporal constraints on the association between spontaneous blinking and striatal dopamine receptor availability [S12, S15, S16]. Nevertheless, future research may gain insights into this by systematically varying ISIs to determine bounds and characteristics of this temporal window, with possible implications for the mechanistic role of dopamine fluctuations in intra-individual variability in timing.

Previous research has shown that dopamine agonists impair timing performance or temporal precision [S17] and overexpression of $D_2$ receptors in striatum is associated with a reduction in temporal precision in suprasecond timing [S18] so it is somewhat surprising that spontaneous blinking was unrelated to temporal precision in the present study. The pattern of results – a numerical decline in temporal precision (larger DL and WF values) in post-blink trials in all three tasks – is in the same direction as previous studies, but non-significant in all cases. Aside from DL values in the visual subsecond task (Study 1),

effect sizes (Cohen's *d*) for DL and WF values reliably varied from 0.20 to 0.30. Assuming 80% power, a two-tailed test, and a-level of .05, an *a priori* power analysis indicates that future studies will need at least 90 (and possibly as many as 200) participants to detect such effects if our effect size estimates are reliable. It is plausible that the subtle shifts in striatal dopamine release, as indirectly indexed by spontaneous blinking, are sufficient to produce changes in perceived duration but insufficient to produce marked changes in temporal precision akin to what is observed with pharmacological agents. Nevertheless, future research on spontaneous blinking and interval timing should consider this question further.

These results are limited by their reliance on spontaneous blinking, an indirect, proxy measure of striatal dopamine. Although spontaneous blinking is moderately to strongly related to striatal $D_2$-like receptor availability [S12], it is contaminated by other factors unrelated to dopamine, such as dryness of the eyes [S14]. Although this introduces noise into this measurement, the replicability of the effect across a range of intervals and modalities and the relative consistency of effect sizes across tasks strongly suggests that this represents a meaningful relationship. Similarly, it should be noted that the results are correlational and imply, rather than establish, a causal link between striatal dopamine and intra-individual variability in interval timing. Despite these limitations, the present method represents the most robust approach for testing the hypothesis that striatal dopamine relates to intra-individual variability in interval timing. That is, at present, there is no way to measure, or otherwise modulate, striatal dopamine levels in humans with sufficient temporal resolution as to interrogate this relationship at the level of moment-to-moment intra-individual variability. These results nicely complement the extant literature linking dopamine and interval timing and advance our understanding of the mechanisms underlying intra-individual variability in interval timing.

An alternative explanation of the current results is that blink-induced changes in visual processing impacted perceived duration. Insofar as the association between spontaneous blinking and perceived duration of visual subsecond and suprasecond intervals found in Study 1 was replicated with auditory subsecond intervals in Study 2, this explanation seems highly unlikely. Indeed, we maintain that blink-

induced changes in visual processing are unlikely to have had any impact on the perceived duration of visual intervals in post-blink trials. Blinking reduces processing of visual stimuli in two ways, by physically closing the eyelid and by generating cortical suppression both before and after the time of actual lid closure [S19]. Eye blinks trigger cortical deactivation of areas responsible for processing of external visual stimuli [S20, S21] and are further associated with reduced activity in parietal and prefrontal cortices, suggesting more general inhibition of awareness [S21, S22]. Due to this *blink suppression* effect, the perceptual effect of blinks is small compared to the changes that it actually produces on the retina [S19]. Consistent with the duration of the blink suppression (200-250ms) [S19], in one study, visual perception of rapid shape changes decreased when a blink happened within a time window from 75ms before until 150ms after the stimulus onset [S23]. The duration of blink suppression is thus significantly shorter than the latency between blinks and the interval timing effects in Study 1 and thus is unlikely to play a significant role in post-blink changes in visual interval timing.

A final open question is whether the observed relationship between spontaneous blinking and intra-individual variability in interval timing is mediated by attention. We have proposed that fluctuations in striatal dopamine modulate perceived duration through a speeding up or magnification of a dopaminergic pulse that synchronizes the oscillations of cortical neurons at the onset of the comparison stimulus or through a change in the speed of an internal clock-like neural oscillator [S24, S25]. In contrast, an attentional account would instead posit that a transient increase in dopamine receptor availability, as reflected in a blink, temporarily enhances attention to the comparison interval duration, which in turn leads to overestimation. Spontaneous blinking is associated with superior cognitive control [S3] and attention toward time reliably augments perceived duration [S26] and so this interpretation is plausible. Insofar as we replicated the principal result using auditory intervals in Study 2, we can rule out that the effect is driven by changes in dopamine-mediated visual attention. However, at present, we are unable to dissociate an account in which dopamine directly modulates neural oscillatory activity used to estimate the passage of time from one in which dopamine enhances temporal attention. However, irrespective of whichever account is ultimately correct, our results still suggest an association between transient

fluctuations in striatal dopamine and intra-individual variability in interval timing and thereby help to further clarify the mechanisms underlying moment-to-moment variability in our perception of time.

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