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Properties of the Internal Clock: First- and Second-Order Principles of Subjective Time

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Abstract

Humans share with other animals an ability to measure the passage of physical time and subjectively experience a sense of time passing. Subjective time has hallmark qualities, akin to other senses, which can be accounted for by formal, psychological, and neurobiological models of the internal clock. These include first-order principles, such as changes in clock speed and how temporal memories are stored, and second-order principles, including timescale invariance, multisensory integration, rhythmical structure, and attentional time-sharing. Within these principles there are both typical individual differences—influences of emotionality, thought speed, and psychoactive drugs—and atypical differences in individuals affected with certain clinical disorders (e.g., autism, Parkinson's disease, and schizophrenia). This review summarizes recent behavioral and neurobiological findings and provides a theoretical framework for considering how changes in the properties of the internal clock impact time perception and other psychological domains.

Contents

INTRODUCTION 21.2
FIRST-ORDER PRINCIPLES OF SUBJECTIVE TIME 21.6
Clock Speed/Thought Speed 21.6
Memory Translation Constant 21.8
SECOND-ORDER PRINCIPLES OF SUBJECTIVE TIME 21.9
Scalar Property 21.9
Modality Effects/Memory Mixing 21.13
Beat-Based Versus Duration-Based Timing 21.14
Attentional Time-Sharing 21.15
SUMMARY AND DESCRIPTION OF A UNIFIED MODEL OF TEMPORAL INTEGRATION 21.16
FUTURE DIRECTIONS 21.18

INTRODUCTION

Time shapes many aspects of our daily lives (Allan 1979, Buhusi & Meck 2005): The reader may have set aside a certain amount of objective (clock) time to read this article before moving onto other activities, which may themselves need to performed at or for specific times. We rely heavily on external timekeepers and temporal organizers (clocks, calendars) to keep track of the temporal properties of events (i.e., when and for how long an event will occur), but we are also quite adept at keeping track of time on our own given the appropriate circumstances (Nobre & Coull 2010). The challenge, therefore, is to identify the clock inside our heads, something referred to as the "internal clock" (Church 1984). As in other animals, our ability to time external events in the seconds to minutes range (interval timing) allows us to subjectively experience the passage of physical time and allows us to integrate action sequences, thoughts, and behavior and to detect emerging trends and anticipate future outcomes (Bechara et al. 1996, Kotz et al. 2009, Nussbaum et al. 2006). This temporal yardstick (internal clock) can also be both typically and atypically distorted by a variety of sensory, psychological, and physiological factors (e.g., arousal, modality, and pharmacological treatments; see Allman & Meck 2012, Paule et al. 1999, Vatakis & Allman 2013). Perhaps parsimoniously related to the internal clock is our ability to temporally structure our use of language and other cognitive processes such that past events serve as agents for current events (e.g., episodic memory) and for planning and sequencing our intended behaviors toward events in the future (e.g., executive function and prospective memory), which although perhaps uniquely human may not necessarily be so (Allman et al. 2013, Diedrichsen et al. 2003, Nyberg et al. 2010, Schirmer 2004, Siegel et al. 2012, Suddendorf et al. 2009, Ullman 2004, Ullman & Pierpont 2005).

Most researchers agree that there is no single neurological locus that serves as the core (master) clock in the brain, although the primordial circadian clock in the suprachiasmic nucleus can regularly and persistently modulate a variety of species' sleep-wake to day-night cycles (even in conditions devoid of light-dark cues). However, to account for interval timing ability, which has been unequivocally demonstrated, some sort of general-purpose, cognitively controlled internal clock is required (Agostino et al. 2011a; Allman & Meck 2012; Guilhardi & Church 2009; Hinton & Meck 1997a,b; Lewis & Meck 2012; Lewis & Miall 2003; Matell & Meck 2000; Meck 1984;

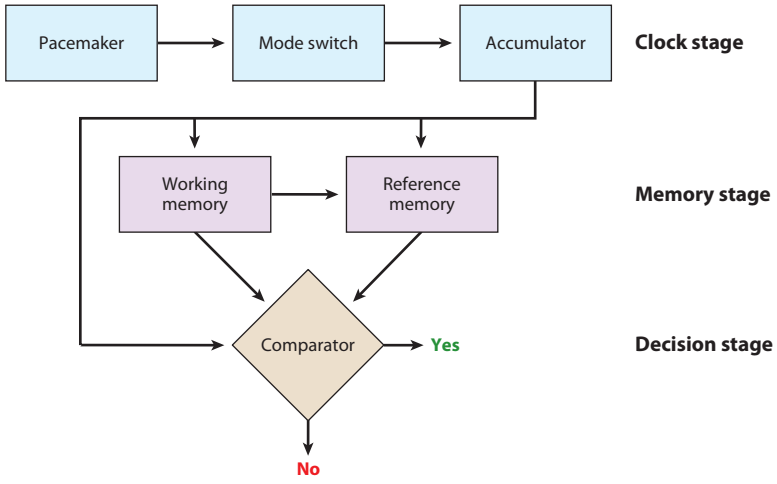

Figure 1

The information-processing model of interval timing as specified by scalar timing theory and other internal clock theories of psychological time. Adapted from Church (1984), Gibbon et al. (1984), Meck (1984), and Treisman (1963, 1984).

Treisman 1963, 1984, 2013). The primary goal of this review is to specify the forms this internal clock may take, i.e., psychological, biological, and formal.

As originally proposed, the internal clock (Church 1984, Gibbon et al. 1984, Treisman 1963) is composed of a three-process model (see **Figure 1**). At the onset of a to-be-timed event (stimulus), a pacemaker emits pulses that are gated into an accumulator by attention. The current pulse tally (in working memory) is compared with a previously stored value (in reference memory) for that particular event duration. When the two values match closely enough, a decision rule operates to produce an estimate of time, which can be influenced by feedback. Much like a stopwatch, this system can be started (run mode), paused (stop mode), and reset (repeat mode) to time specific events or multiple events occurring at the same time (Buhusi & Meck 2009b, Meck & Church 1983).

As revealed by psychophysical methods used to quantify sensory responses to physical stimuli (see Gescheider 1997), timing and time perception have a variety of features (indices derived from psychophysical functions) that must be accounted for by all plausible models of the internal clock, regardless of the differences in their proposed mechanisms (Buhusi et al. 2002, 2005; Matell & Meck 2000; Penney et al. 2008; Wencil et al. 2010). For not only is the intensity of the internal perception linearly related to the magnitude of external stimulation (subjective time increases with physical time), but also increases in the magnitude of a physical stimulus produce proportional increases in the variance of the perception (it is more difficult to time precisely for longer durations)—referred to as scalar variance or Weber's law (see Allan 1998). Thus interval timing ability shares many characteristic hallmarks with sensory perception (like vision and hearing; see Fraisse 1984). Considerable progress has been made in specifying the basic operating principles (Wearden 2005) and identifying the behavioral mechanisms and neural substrates involved in interval timing. Indeed models of interval timing have been called "the most successful [models] in the whole of psychology" (Wearden 2001, 2003).

Although neural-network states may be utilized to time subsecond durations without the need for a dedicated clock (Karmarkar & Buonomano 2007, Laje & Buonomano 2013), the timing of suprasecond durations on the order of seconds to minutes appears to involve a number of

first- and second-order principles that can be used to classify (within- and between-) individual differences in interval timing ability (for details, see Buhusi & Meck 2005, Gibbon et al. 1984, Meck 2003). These principles define how the internal clock works and how it can be manipulated by sensory, psychological, and physiological factors. First-order principles are those that can be applied to individual event durations and typically involve the accuracy and precision with which the criterion duration is timed. These include differences in (*a*) how fast the subjective clock is ticking—clock speed (α), the rate of pulse accumulation; see **Figure 1**); and (*b*) how the breadth of durations is subjectively stored and compared with ongoing (present) durations—associated memory translation parameter (k^*, comparing the current pulse count to the stored pulse count). Second-order principles compare multiple durations with each other in terms of (*c*) timescale invariance, that is, explaining why it is easier to detect one additional increment in stimulus value (i.e., brightness, duration) between high-intensity events compared with those of low intensity—the scalar property. The influence of nontemporal factors on interval timing also needs to be considered, such as (*d*) why auditory events of a given physical duration are judged subjectively as being longer than visual events—modality effects and memory mixing; (*e*) how our interval timing ability appears intimately related to our sensitivity to organizing rhythms—beat-based versus duration-based timing; and (*f*) how our subjective sense of time can be influenced by how much we are thinking about the temporal quality of events (e.g., the notion that a watched pot never boils)—attentional time-sharing. Of course, perhaps the most compelling challenge, and evidence, for the existence of the internal clock is in its neurobiological instantiation as described in the sections below (Allman & Meck 2012, Coull et al. 2011, Merchant et al. 2013, Salvioni et al. 2013, Teki et al. 2012).

William James (1890) first asked, "To what cerebral process is the sense of time due?" Experimental findings heavily implicate the cerebral cortex (Buonomano & Laje 2010; Durstewitz & Deco 2008; Harrington et al. 1998, 2004, 2010, 2011b; Ivry & Spencer 2004; Ivry & Schlerf 2008; Karmarkar & Buonomano 2007; Koch et al. 2009; Kotz & Schwartze 2010, 2011; Livesey et al. 2007; Matell et al. 2003, 2011; Meck et al. 2008; Van Rijn et al. 2013; Wiener et al. 2010a,b), along with the basal ganglia and cerebellum (motor and cognitive sequencing centers). The oscillatory properties of cortical neurons appear to produce the internal clock ticks and, hence, provide a distinct pattern of activity (ticks) to represent a given duration (cf. accumulator in **Figure 1**), which appear to be detected by striatal medium spiny neurons tuned to trigger an action potential to a target duration by detecting coincident oscillatory patterns (cf. memory processes; see Brody et al. 2003; Kaufman et al. 2005; Lustig et al. 2005; Treisman et al. 1990, 1994). There also appears to be a general form of ordinal (e.g., how much?) analog representation of magnitude (time, space, and number) within the parietal cortex (Bueti & Walsh 2009; Cordes et al. 2007; Meck & Church 1983; Walsh 2003a,b). These various contributions of the cerebral cortex to the properties of the internal clock are outlined in **Table 1**.

The overall strategy of this review is to discuss first- and second-order principles of the internal clock with an emphasis on their neurobiological bases (Allman & Meck 2012, Claassen et al. 2013, Jones et al. 2011, Meck 2005, Merchant et al. 2013, Tregellas et al. 2006, Teki et al. 2012, van Rijn et al. 2011). We examine animal models; typical and atypical individual differences in human timing behavior, such as those affected by certain clinical disorders (e.g., autism, Parkinson's disease, and schizophrenia); drug and lesion effects; and candidate neural substrates for the internal clock (i.e., corticostriatal and cortico-cerebellar circuits). The goal is to provide the basis for a unified model of interval timing based on our current understanding of its underlying psychological and neural mechanisms (Allman & Meck 2012, Claassen et al. 2013, Cope et al. 2013, Jones & Jahanshahi 2013, Jones et al. 2011, Meck 2005, Merchant et al. 2013, Tregellas et al. 2006, Teki et al. 2012).

Table 1 Summary of cortical contributions to interval timing

Cortical area(s)	Technique(s)	Species	Findings	Reference(s)
Neocortex	Decortication by pial stripping	Rat	Rightward shift of timing functions consistent with a change in k^*	Jaldow et al. (1989, 1990)
Lateral AFC/primary motor cortex	Single-unit recording	Rat	Units demonstrate peaks of activity centered around the appropriate signal durations	Matell et al. (2003)
Medial AFC	Single-unit recording	Rat	Units demonstrate a heterogeneous population code of ramps, peaks, and troughs as a function of signal duration	Matell et al. (2011)
Lateral AFC	Aspiration lesion	Rat	Rightward shift of timing functions consistent with a change in k^*	Meck et al. (1987)
Lateral AFC	Aspiration lesion	Rat	Reduced clock speed effect of DA agonists and antagonists	Meck (2005)
Lateral AFC	Aspiration lesion	Rat	Impairments in STP	Olton et al. (1988)
Lateral AFC/primary motor cortex	Single-unit recording	Rat	Units respond to compound stimuli in an STP task	Pang et al. (2001)
PFC, presupplementary, and SMA	Single-unit recording	Monkey	Units code specific target durations by temporal filtering	Mita et al. (2009), Oshio et al. (2008)
PFC, Area 9	Single-unit recording	Monkey	Units respond in perceptual recognition and during the internal generation of a target duration	Yumoto et al. (2011)
Dorsolateral PFC, posterior inferior parietal cortex, basal ganglia, and posterior cingulate cortex	PET	Monkey	Local application of bicuculline resulted in selective impairments in timekeeping within the identified neural networks	Onoe et al. (2001)
Hippocampal structures	Dorsal hippocampal, fimbria-fornix, or medial septal area lesions	Rat/mouse	Leftward shift and sharpening of timing functions consistent with a change in k^*	Balci et al. (2009), Meck (1988), Meck et al. (1984, 1987, 2013), Olton et al. (1988), Yin & Meck (2013), Yin et al. (2010)
Medial temporal lobe	Left or right temporal lobe resection	Human	Left temporal lobe resection leads to a leftward shift of timing functions consistent with a change in k^*. Right resection leads to increased variability	Melgire et al. (2005), Vidalaki et al. (1999)
Left inferior parietal cortex	fMRI	Human	Left-hemispheric bias for implicit timing	Coull & Nobre (2008), Wiener et al. (2010a)
Right dorsolateral PFC	fMRI and TMS in normal controls as well as in patients with right PFC lesions	Human	Right-hemispheric bias for explicit timing with underestimation of duration following TMS and timing deficits in PFC patients	Coull & Nobre (2008), Harrington et al. (1998), Koch et al. (2002), Meck & Malapani (2004), Wiener et al. (2010b)
Left posterior parietal, striatal, and bilateral inferior frontal regions	fMRI	Human	Attending to time	Coull & Nobre (2008), Coull et al. (2003, 2008, 2011), Wencil et al. (2010)

(*Continued*)

Table 1 (*Continued*)

Cortical area(s)	Technique(s)	Species	Findings	Reference(s)
Left inferior frontal, superior temporal, and SMA regions	fMRI	Human	Accumulator component of timing	Wencil et al. (2010)
Inferior frontal cortices bilaterally	fMRI	Human	Comparator component of timing	Coull et al. (2008), Teki et al. (2011), Wencil et al. (2010)
Frontostriatal circuits: includes presupplementary motor area and right frontal operculum	fMRI	Human	Three areas of a timing network that survive a task-difficulty manipulation have been identified: inferior gyrus, anterior insula, left supramarginal gyrus, and putamen	Coull et al. (2003, 2008 , 2011), Harrington et al. (2010), Hinton & Meck (2004), Livesey et al. (2007), Meck et al. (2008), Rao et al. 2001
Right parietal cortex	fMRI in normal controls and brain-damaged patients with spatial neglect	Human	Cortical involvement of sensorimotor transformations with regard to space, time, and other magnitudes	Bueti & Walsh (2009) Danckert et al. (2007)
SMA, left premotor cortex, and left insula	fMRI	Human	Beat-based perceivers showed greater activation in comparison with duration-based perceivers	Grahn & McAuley (2009); see also Teki et al. (2011)
Left posterior superior, middle temporal gyri, and right premotor cortex	fMRI	Human	Duration-based perceivers showed greater activation in comparison with beat-based perceivers	Grahn & McAuley (2009); see also Teki et al. (2011)
Right superior temporal gyrus	TMS	Human	Impaired auditory temporal processing	Bueti et al. (2008a,b)
Auditory cortex	TMS	Human	Impaired auditory and visual temporal processing	Kanai et al. (2011)
Visual cortex	TMS	Human	Impaired visual temporal processing only	Kanai et al. (2011)

Abbreviations: AFC, agranular frontal cortex; DA, dopamine; fMRI, functional magnetic resonance imaging; k^*, memory translation constant; PET, positron emission tomography; PFC, prefrontal cortex; SMA, supplementary motor area; STP, simultaneous temporal processing; TMS, transcranial magnetic stimulation.

FIRST-ORDER PRINCIPLES OF SUBJECTIVE TIME

Clock Speed/Thought Speed

The rate at which subjective time grows as a function of elapsing physical time is thought to be influenced by clock speed, which is reflected by the number of clock ticks or oscillations per unit time (Church 1984, Maricq et al. 1981, Meck 1983, Treisman 2013). This concept of clock speed and the subjective experience of time can be shown to be logically related to formulations of thought speed, which is independent of thought content but dependent on the level of arousal, mood, and the frequency of environmental events (Pronin 2013, Pronin et al. 2008, Pronin & Jacobs 2008, Pronin & Wegner 2006). Thus, the first-order principle of clock speed can accommodate the second-order principle of the influence of nontemporal features (i.e., modality, arousal, affect) on interval timing. Changes in clock speed (e.g., via arousal, emotion)

lead to proportional changes in the representation of event durations, through the rate at which the criterion number of pacemaker pulses (clock ticks) is accumulated. Various pharmacological and psychiatric conditions appear to modify clock speed, and changes in mean clock speed can be modeled either as a within- or between-trials phenomenon, although the convention is to rely on between-trial variation (Gibbon & Church 1984, 1990, 1992; Matell & Meck 2004; Meck 1983). Changes in clock speed are typically measured relative to a baseline condition, and absolute measures (of pulse accumulation) are difficult to obtain (Williamson et al. 2008).

How might we attempt to measure the absolute speed of an internal clock? One strategy would be to attempt to synchronize the time base with a repetitive signal (e.g., visual flicker or auditory click trains) presented at a known frequency (Treisman et al. 1990; Wearden et al. 1999, 2009). This process is referred to as entrainment, and assumptions must be made about the underlying frequencies of these putative oscillatory processes (see Lustig et al. 2005, Matell & Meck 2004, Treisman et al. 1994). Once participants have been entrained to this external metronome, they could then be released into a free-run condition and allowed to return to their normal (clock speed) state. Differences in the horizontal placement of their psychophysical timing functions under these two conditions (entrainment versus free run) would allow investigators to determine their natural oscillation frequency or clock speed (Treisman 2013).

Conventionally, when compared relative to a nondrug baseline (assigned a nominal value of 1.0 in the model), administration of indirect dopamine agonists—including drugs of abuse such as cocaine, methamphetamine, and nicotine—increases clock speed as indexed by a leftward shift in the position of timing functions (i.e., 20% faster; a value of 1.2 in the model; Cheng et al. 2006, 2007; Maricq et al. 1981; Matell et al. 2004, 2006; Meck 1983, 2007). In contrast, dopamine receptor antagonists such as haloperidol and raclopride produce proportional rightward shifts in functions that are interpreted as reflecting a decrease in clock speed (and assigned multiplicative constants <1.0 in the model; Buhusi & Meck 2002; MacDonald & Meck 2004, 2005, 2006; Maricq & Church 1983; Meck 1983, 1986, 1996). In the internal-clock model, increases and decreases in clock speed produce a multiplicative change in the stored clock reading associated with a particular event duration.

Such pharmacological challenges (e.g., methamphetamine and haloperidol) have been coupled with excitotoxic lesions of cholinergic cell bodies in the medial septal area and the nucleus basalis magnocellularis, as well as with radiofrequency lesions of the fimbria-fornix and aspiration lesions of the frontal cortex, to investigate control of the internal clock (Meck 1996, 2006a; Meck et al. 1986, 1987). Lesions of the nucleus basalis magnocellularis (which contains cholinergic cell bodies that project to the frontal cortex), as well as lesions of the frontal cortex itself, selectively reduce the pharmacological modification of clock speed. This ability suggests that clock speed is likely mediated by dopamine receptors located on corticostriatal neurons in the nigrostriatal pathway (recall that cortical oscillations likely serve as the time base of the internal clock coincidence detected by medium spiny neurons in the striatum). Such combined lesion/genomic/pharmacological studies provide a crucial foundation for understanding how the oscillatory properties of corticostriatal circuits subserve clock ticks and the memory processes used in duration discrimination (Agostino et al. 2013; Balci et al. 2012; Buhusi & Oprisan 2013; Meck 1996, 2001; Meck et al. 2012; Oprisan & Buhusi 2011). The selectivity of this cortical control of clock speed is supported by recent findings showing that dopamine in the nucleus accumbens (mesolimbic pathway) plays a crucial role in motivation/incentive salience but not in the regulation of clock speed (Kurti & Matell 2011).

The fact that time can subjectively pass by faster or slower under various conditions (i.e., low/high arousal and/or drug effects) may also account for the pace of behavioral and social interactions involving impulsivity (Wittmann et al. 2011, Wittmann & Paulus 2008). Various clinical populations, considered to have impairments in internal clock–related abilities, have the potential

to provide absolute measures of clock speed, including individuals with abnormal and normal aging, attention-deficit/hyperactivity disorder (ADHD), autism, schizophrenia, Parkinson's disease, and drug abuse (Allman 2011; Allman & Meck 2012; Cheng et al. 2011; Kotz et al. 2009; Lustig & Meck 2001, 2005; Grahn & Brett 2009; Malapani et al. 1998; Meck 2006a,b,c; Meck & Benson 2002; Penney et al. 2005; Smith et al. 2007; Wittmann et al. 2007).

Memory Translation Constant

Researchers assume that the accumulation (current tally) of pacemaker pulses grows in a linear fashion as physical duration increases but may not necessarily be stored (remembered) veridically. The difference between the current duration (clock reading) and the encoded duration stored in reference memory is reflected by a memory translation constant (see Church 1984; Gibbon et al. 1984; MacDonald et al. 2007; Meck 2002a,b). That is, the number of pacemaker pulses is transferred from the accumulator to reference memory at some modifiable baud rate that ultimately influences the quantitative aspects of the represented signal duration (e.g., Meck 1983, 1996).

In a modified version of the original timing model, known as scalar timing theory (e.g., Gibbon et al. 1984; Gibbon & Church 1984, 1990, 1992), memory translation has formally been referred to as the K* parameter, which is a multiplicative constant (Church 2003, Meck 1983). An individual would consistently expect the end of an event to occur later than the programmed time (i.e., if its memory storage constant was greater than 1.0) or earlier (i.e., if its memory storage constant was less than 1.0). The remembered duration of an event is based on the amount of time required to transfer the clock reading (e.g., oscillation vector or number of pulses in the accumulator) into reference memory. Transfer time would be directly proportional to the size of the oscillation vector or the number of pulses in the accumulator and the speed of transfer (i.e., baud rate). Pulse accumulation essentially functions as an up counter and memory transfer functions as a down counter, with the additional assumption that neural networks can function as look-up or conversion tables (Dali & Zemin 1993, Kimura & Hayakawa 2008).

If the speed of this memory storage process were to deviate from normal values, then it would be possible for proportionally shorter or longer values to be represented in memory. Unlike changes in clock speed, changes in memory storage speed would not self-correct, even in the case where individuals were surprised by the mismatch between their clock readings and a value sampled from reference memory. This lack of flexibility is because any updating of memory in this case would lead to the continued distortion of stored values for as long as the modification in memory storage speed remained in effect, i.e., as long as k^* was greater than or less than 1.0 (e.g., Meck 1983, 1996, 2002a).

Derived k^* parameters have been obtained from various typical and atypical populations. For example, principled computer modeling (Wearden 2001) of time-perception (temporal generalization) data in children reveals that the k^* parameter (and hence, memory distortion) quantifiably improves across childhood (Droit-Volet et al. 2001, Lustig & Meck 2011), and this typical trend in k^* is not observed in a broader age range of individuals with autism (Allman 2011, Allman et al. 2011). Damage to hippocampal and cortical circuits produces distortions in temporal memory that are proportional to the physical durations of the events being timed; hippocampal-related damage produces underproductions and cortical-related damage produces overproductions of target durations (Coull et al. 2011; Harrington et al. 1998; Jaldow et al. 1989, 1990; Koch et al. 2003; Meck 1988; Meck et al. 1984, 1987, 2013; Olton et al. 1988; Vidalaki et al. 1999; Yin & Meck 2013; Yin & Troger 2011; Yin et al. 2010). Moreover, the relationship between the magnitude of the error in the content of temporal memory and activity in the frontal cortex and hippocampus has been examined in mature (10- to 16-month-old) and aged (24- to 30-month-old) rats (Meck 2002a,b, 2006c). Regression analyses indicated that neural activity in the frontal cortex of both mature and

aged rats, and in the hippocampus only for aged rats, is proportional to the absolute error in the content of temporal memory. The observed changes in neural activity were dependent on the predictability of the programmed time of feedback and age-related changes in memory encoding and retrieval.

SECOND-ORDER PRINCIPLES OF SUBJECTIVE TIME

Scalar Property

The scalar property of interval timing is a form of Weber's law that is shared with the other senses (Gescheider 1997, Gibbon 1977). It is characterized by proportionality between the standard deviation of a response distribution and the target duration being timed, reflected in the linearity of the relationship between timing variability and duration magnitude (Church 1984, 1989; Church et al. 1994; Gibbon 1977, 1991; Gibbon et al. 1984; Malapani & Fairhurst 2002; Piras & Coull 2011). This notion suggests that the coefficient of variation (CV; ratio between the standard deviation and the mean of the sample) should be constant across a range of durations; multiple timescales may also be identified that reflect timing mechanisms with different levels of precision, such as subsecond and suprasecond timing (Cordes & Meck 2013, Gibbon et al. 1997; but see Lewis & Miall 2009 for an alternative view of violations of the scalar property). Moreover, a strong form of Weber's law applied to interval timing can be evaluated by plotting the entire response function obtained from the timing task, e.g., peak-interval or temporal bisection procedures, on a relative timescale, i.e., normalized by the obtained peak time or the point of subjective equality as illustrated in **Figures 2**, **3**, **4**, and **5**. The superimposition of the entire psychometric function for

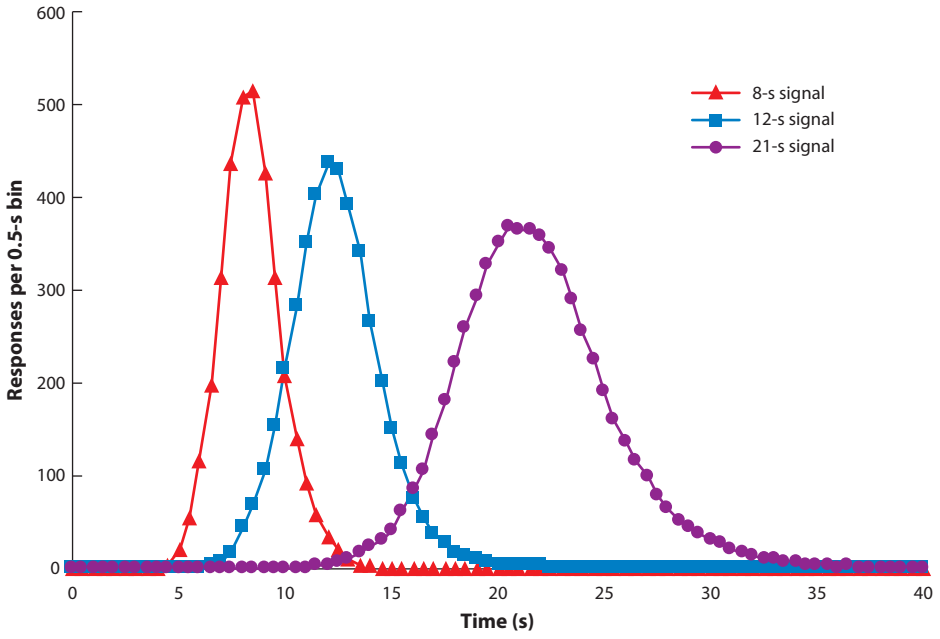

Figure 2

Human peak-interval timing functions for 8-, 12-, and 21-s target durations. Mean keyboard responses per 0.5 s plotted as a function of duration (seconds) for a visual (*blue square*) signal. Data are replotted from Rakitin et al. (1998) with permission.

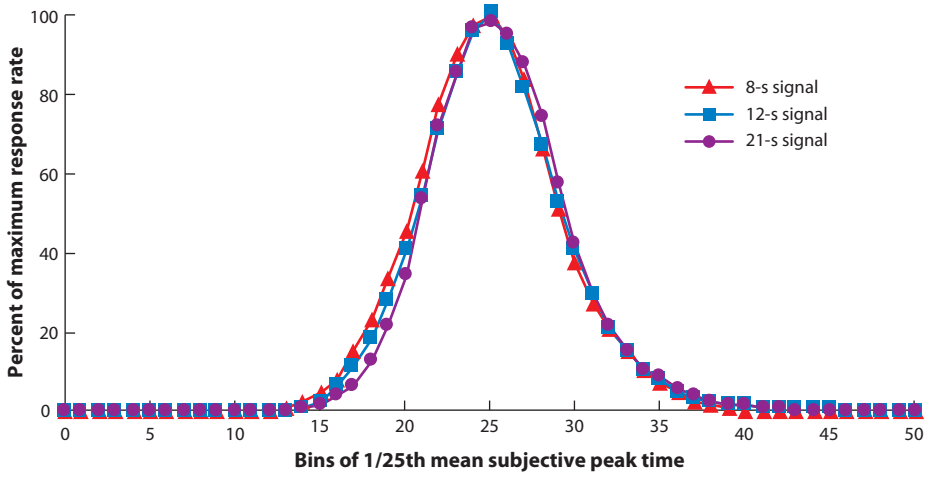

Figure 3

Scalar property. Human peak-interval timing functions for 8-, 12-, and 21-s target durations. Mean percentage of maximum response rate plotted from the data presented in **Figure 1** as a function of relative time in bin widths of 1/25th of the obtained peak times. Data are replotted from Rakitin et al. (1998) with permission.

a relatively wide range of durations provides strong evidence for timescale invariance and for the scalar property of interval timing (Cheng & Meck 2007, Church & Deluty 1977, Church et al. 1991). In general, excellent superimposition has been observed in human temporal perception using the bisection procedure with durations ranging from 50 to 8,000 ms (Melgire et al. 2005), from 750 to 2,100 ms (Allan & Gibbon 1991), and from 2,000 to 12,000 ms (Penney et al. 2000).

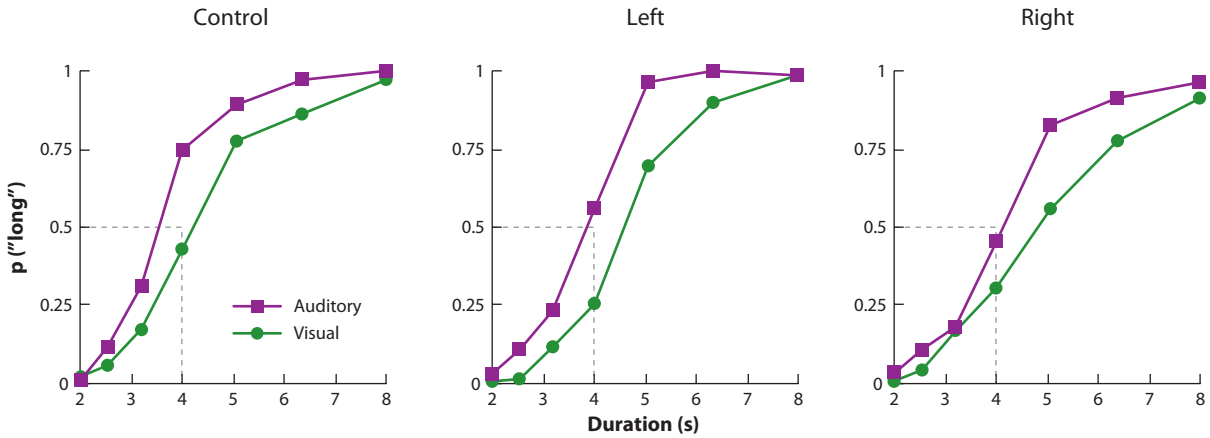

Figure 4

Modality difference/memory mixing. Group probability of mean long response, p ("long"), functions averaged across participants for the auditory and visual signals presented during the same session in the 2-s versus 8-s temporal bisection procedure. Data are from control participants and patients with either left or right temporal lobe resection. The broken lines indicate the signal duration typically associated with the point of subjective equality for auditory or visual signal presented alone, which is at the geometric mean (GM) of the short- and long-anchor durations (GM of 2 s and 8 s = 4 s). Data for auditory and visual signal durations are replotted from Melgire et al. (2005) with permission.

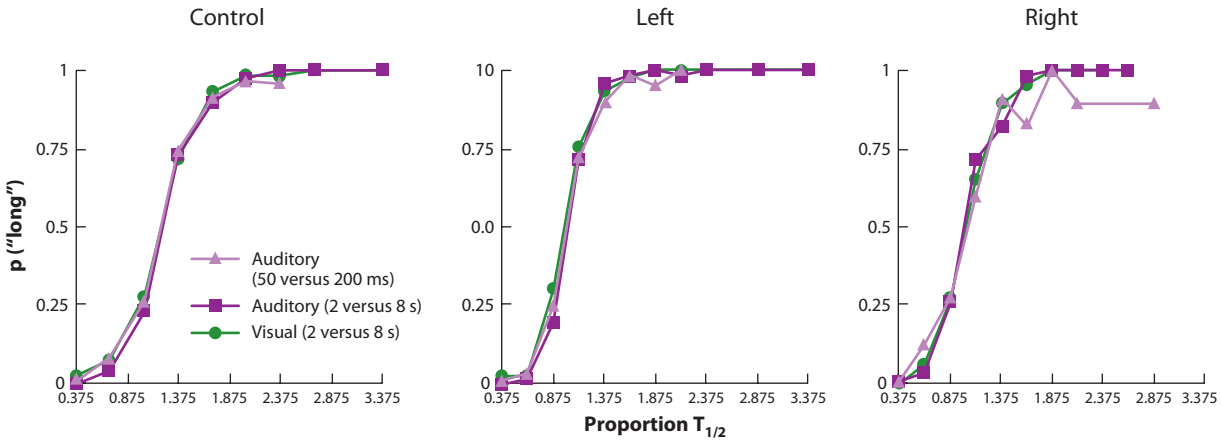

Figure 5

Scalar property. Superimposition plot of temporal bisection functions in control participants and patients with either left or right temporal lobe resection. These rescaled response functions from the data in **Figure 3** were created in three steps. First, for each participant and condition, the probe durations were divided by the corresponding point of subjective equality in order to normalize the data. Second, the resulting proportion $T_{1/2}$ values were rank-ordered across participants by condition and grouped in bins of 0.25 unit width. Third, the probability long response, p ("long"), values corresponding to the $T_{1/2}$ values in a given relative time bin, were averaged for that bin. Note that for these plots the values in the end time bins are less reliable than those in the central bins because fewer p ("long") values are likely to contribute to the end bin averages because not all participants will show the same range of proportion $T_{1/2}$ scores. Data for auditory and visual signal durations are replotted from Melgire et al. (2005) with permission. See Allan & Gibbon (1991) for additional details on this method for evaluating superimposition in the temporal bisection procedure.

Superimposition has been equally consistent for human temporal production and reproduction using the peak-interval procedure with durations ranging from 8 to 21 s (Rakitin et al. 1998) and from 8 to 24 s (Hinton & Rao 2004). Moreover, identification of brain regions showing the scalar property in the hemodynamic response associated with the timing of 11- and 17-s durations in an fMRI experiment is a powerful technique for studying corticostriatal circuits and isolating the perceptron component of the neural representation of time in the putamen and other areas of the dorsal striatum (Coull et al. 2008, 2011; Forstmann et al. 2010; Hinton & Meck 2004; Jin et al. 2009; MacDonald et al. 2012; Matell & Meck 2004; Meck & Malapani 2004). The scalar property (i.e., Weber's law) as applied to the mismatch negativity in event-related potentials (ERPs) associated with temporal deviants has also been used as a marker to study the development and preattentive aspects of interval timing in preverbal infants and in healthy adults (Brannon et al. 2004, 2008).

Violations of the scalar property frequently result from procedural issues (Buhusi et al. 2009, Wearden & Lejeune 2008) or data analysis considerations, i.e., whether the CV should be calculated for peak-interval data based on the distribution of peak times from single-trial analysis or from the mode and spread of the mean response function (e.g., Church et al. 1994, Gibbon & Church 1990, Rakitin et al. 1998). When individual differences are observed in the scalar property, it is important to investigate whether some participants are employing a chronometric counting strategy to improve their accuracy and precision for longer durations (Hinton & Rao 2004). Altered variance patterns have also been observed following damage to the underlying timing mechanisms in the basal ganglia in both Huntington's and Parkinson's diseases (e.g., Aparicio et al. 2005, Artieda et al. 1992, Hinton et al. 2007, Malapani et al. 1998). Consequently, individual differences in the scalar property can be diagnosed and classified according to their variance properties. If participants are counting rather than timing, and count at similar rates for all durations, their count totals will be

proportional to the durations that they are instructed to time. In this case, the standard deviations of their counting distributions will be proportional to the square root of the duration rather than to the target duration itself as required for scalar timing. Explicit counting improves the precision of timing performance as reported by Rakitin et al. (1998) and modeled by Killeen (1991) and Killeen & Weiss (1987). Chronometric counting of this sort involves the participant subdividing a given duration into a series of smaller intervals that are counted. Nevertheless, the variability of these subintervals typically displays the scalar property over the range of 0.5–1.3 s (Hinton & Rao 2004).

Damage to and/or pharmacological blockade of corticostriatal circuits impairs the regulation of clock speed as a function of the target duration (MacDonald & Meck 2004, 2005, 2006; Meck 1986, 1996, 2006a,b). This limitation is important because timescale invariance is thought to result from the clock reading increasing monotonically within trials at a rate that depends on the duration of the interval (see Almeida & Ledberg 2010, Killeen & Fetterman 1988). As a consequence, diminished control over this systematic increase in clock speed would result in a violation of the scalar property such that longer intervals will be timed more precisely than shorter intervals owing to the loss of the scalar source of variability in clock speed (assuming that the default is Poisson variability; see Gibbon 1992). Weakened regulation of clock speed in Parkinson's disease can also lead to the coupling or migration of multiple target durations such that the shorter duration is overproduced and the longer duration is underproduced (Jones et al. 2008, Koch et al. 2008, Malapani et al. 1998, Shea-Brown et al. 2006).

Furthermore, the scalar property is also demonstrated in the quality of parent-infant interactions (including gaze and vocalizations); specifically, their coordination alternates between scalar and absolute timing patterns in delicate arrangements (see Jaffe et al. 2001). As noted above, the scalar property is also evidenced in the ERPs of infant brains on a passive duration deviation task employing several different ratios of deviants (1:4; 2:3) to regular 375- to 1,500-ms interstimulus intervals, as the amplitude of the deviant-triggered ERP varied according to the stimulus ratio used (Brannon et al. 2008). These improvements in timing sensitivity correspond to critical periods for the development of other cognitive and behavioral abilities. The steepness of children's temporal generalization functions generally increases across childhood (between 3 and 8 years of age; Droit-Volet et al. 2001, Droit-Volet & Wearden 2001). Characteristic differences in the Weber ratio and quality of superimposition have been observed in the temporal bisection performance of children with a developmental disability or autism (Allman et al. 2011) and in adults at high genetic risk for schizophrenia or major affective disorder (Penney et al. 2005).

Observed deviations in the scalar property of interval timing (e.g., the standard deviation of temporal estimates is proportional to the mean of the interval being timed) are often idiosyncratic, and no principled explanation has been offered for when or at what durations they should occur (except for a hypothesized boundary or transition from one type of timing mechanism to another around 1–3 s; see Gibbon et al. 1997, Grondin 2010). Moreover, most quantitative theories of timing incorporate the scalar property because without it they would fail to account for the vast majority of published data. A number of timing theories implicitly assume that the amount of training or experience with specific durations will reduce the variability in their detection/production, hence providing a basis for a lower coefficient of variation for those particular durations compared with durations that are less well trained (Matell & Meck 2004). These factors, of course, are likely to vary as a function of the task demands and other details specific to the individual participants. At present, it does not seem useful to make a laundry list of exceptions to the scalar property when it is clearly a dominant factor in both the perception and production of durations in the millisecond-to-minutes range. Also, scalar timing theory (Gibbon et al. 1984) is based on the assumption of a Poisson (nonscalar) pacemaker/accumulator that is dominated by scalar sources of variance from memory and/or decision mechanisms. Implicit in this quantitative model is the

idea that if the scalar sources of variability could be reduced by practice and/or other factors, then the nonscalar timing mechanism would be revealed. Hence, temporal discrimination is the result of a combination of different stages of information processing (e.g., clock, memory, and decision), each with a unique form of variance, the balance of which produces the scalar property of interval-timing behavior. Evaluation of the scalar property also depends on which response measures are used [e.g., measure of central tendency and variance of a distribution of temporal estimates versus the form of the entire distribution as determined by the degree of superimposition of different response functions (see Allan & Gibbon 1991, figures 10, 11)].

Modality Effects/Memory Mixing

The original internal clock model (Church 1984) has been used to investigate the phenomenon that sounds of a given duration are judged longer than lights of the same duration. This modality difference in the subjective experience of time highlights the roles of attention and memory mixing in duration judgments (see Allan 1998, Penney et al. 1998). It is typically easier (improved performance) to discriminate differences in time with auditory rather than visual events; transfer from visual to auditory events is greater than from auditory to visual events; and although the attribute of duration may be used independently of stimulus modality, modality-specific information is also encoded and available for use in temporal accumulator models of interval timing (Bartolo & Merchant 2009, Bueti & Macaluso 2011, Gibbon et al. 1984, Gilaie-Dotan et al. 2011, Hass et al. 2012, Meck & Church 1982).

If both modalities are presented within the same experimental session, and with the same criterion duration (in temporal bisection), individuals are more likely to give "long" judgments to auditory rather than to visual intermediate durations (Cheng et al. 2008; Meck 1991; Penney et al. 1998, 2000; Wearden et al. 1998). This difference in perceived duration may occur because auditory stimuli capture and hold attention relatively automatically, whereas attending to visual stimuli requires controlled attention (Meck 1984); for instance, older adults show an exaggerated modality effect compared with young adults, consistent with age-related reductions in attentional control (Cheng et al. 2008, 2011; Lustig & Meck 2001). Within the context of the model (Church 1984), pacemaker pulses (clocks ticks) for auditory stimuli are more efficiently accumulated, allowing larger pulse accumulations and less uncertainty in the stimulus duration (see Cicchini et al. 2012). If relatively small visual and relatively large auditory pulse tallies are intermixed and stored together as a reference memory for that criterion duration, then ongoing pulse tallies (in working memory) for auditory events have an increased probability of being judged long, relative to the reference memory randomly drawn from the sample (vice versa for visual events).

Large individual differences in memory mixing as explained by Bayesian models of optimization have been observed in time-perception experiments (temporal bisection, generalization, and reproduction) using children and young adults (e.g., Acerbi et al. 2012; Burr et al. 2013; Cicchini et al. 2012; Droit-Volet et al. 2007; Jazayeri & Shadlen 2010; Ogden et al. 2010; Penney et al. 2000; Shi et al. 2010, 2013a,b). The amount of memory mixing can be influenced by both positive and negative feedback (e.g., Gu & Meck 2011), by neurodegenerative conditions such as Parkinson's disease (Gu et al. 2013, Smith et al. 2007), by psychiatric illnesses such as schizophrenia (e.g., Carroll et al. 2008, Penney et al. 2005), and by temporal lobe resection as a treatment for epilepsy (Melgire et al. 2005). Differences in the temporal bisection functions for auditory and visual signal durations for control participants and patients following left or right temporal lobe resection are illustrated in **Figures 4** and **5**.

Various cortical areas are involved in the timing of both auditory and visual stimuli (Bueti et al. 2008a, N'Diaye et al. 2004). Moreover, intrasensory timing can be distinguished from intersensory

timing, in part by decreased striatal and increased superior parietal activation (Harrington et al. 2011a). Combined electroencephalography and magnetoencephalography studies have revealed contributions from both auditory and visual cortices in terms of sustained sensory responses, whereas prefrontal and parietal regions appear to integrate modalities in terms of representing event durations (N'Diaye et al. 2004).

With respect to the neurological basis for the phenomenon that sounds are judged longer than lights, there appears to be an interesting asymmetry in how events in different modalities are timed: The right posterior parietal cortex is involved in the timing of both auditory and visual stimuli, whereas visual areas MT/V5 are involved only in the timing of visual events (Bueti et al. 2008b). Disruption of the auditory cortex using transcranial magnetic stimulation (TMS) impaired timing not only for auditory events, but also for visual events (Kanai et al. 2011). In contrast, TMS administered over the primary visual cortex impaired timing performance only for visual events. These results suggest a superiority of the auditory cortex in temporal precision; likewise, they corroborate the proposal that the temporal dimensions of both auditory and visual events are rapidly encoded in the auditory cortex, thereby contributing to the supramodal role for auditory modalities in timing and time perception (see Bueti 2011, Bueti & Macaluso 2010). In contrast, visual events are encoded in the visual cortex in a more selective and controlled (less automatic) fashion (Kanai et al. 2011; see also Meck 1984, Penney et al. 1996). This asymmetry is consistent with findings from cross-modal transfer of duration studies in which discrimination performance is typically better for auditory than for visual events and transfer from visual to auditory events is greater than from auditory to visual events, and although the attribute of duration may be used independently of stimulus modality, modality-specific information is also encoded and available for use in temporal accumulator models of interval timing (Bueti & Macaluso 2011, Gibbon et al. 1984, Gilaie-Dotan et al. 2011, Hass et al. 2012, Meck & Church 1982).

Beat-Based Versus Duration-Based Timing

An intriguing principle of the internal clock, as it relates to its scope, is highlighted by the large and striking individual differences in sensitivity to an implied beat (much like being able to dance to a tune in a coordinated fashion). The question is whether beat perception and interval timing are mediated by different neural circuits: In essence, are they component processes of a broader internal clock, or are they two separate timing systems? Beat perception has been investigated by presenting participants with ambiguous tone sequences; a periodic 600-ms beat is implied but not explicitly emphasized, and participants are required to judge whether the sequences are speeding up or slowing down at the end (Grahn & McAuley 2009, McAuley & Henry 2010). Individuals who did not detect the implied beat perceived the sequences ending with intervals between 300 and 600 ms to be slowing down, whereas individuals who did detect the implied beat perceived the same sequences to be speeding up. Grahn & McAuley (2009) developed an index of the nature of the decision process and its application to the ambiguous sequence paradigm (termed a temporal contrast metric), which determines whether an individual tends toward or against beat-based or duration-based timing and is based on the general theoretical framework of previous dynamic systems models developed by McAuley and colleagues (e.g., McAuley & Jones 2003, McAuley & Miller 2007). The required speeding up or slowing down judgments are assumed to involve the simultaneous consideration of two temporal referents corresponding to different interstimulus intervals or beats. Neuroimaging during this task revealed higher levels of activity in the supplementary motor area, the left premotor cortex, the left insula, and the left inferior frontal gyrus for the strong-beat group as compared with the weak-beat group. In contrast, the weak-beat group showed higher levels of activity in the right premotor cortex and the left posterior

superior and middle temporal gyri. No regions of activation were associated with years of musical experience, and researchers found no evidence that duration-based timing was less accurate or precise than beat-based timing.

Various findings support the proposal that the cerebellum may subserve absolute timing of subsecond intervals (Diedrichsen et al. 2003; Grube et al. 2010a,b; Teki et al. 2011), whereas corticostriatal circuits are involved in the relative timing of longer intervals as well as rhythmic sequences with a regular beat (Coull et al. 2011; Grahn & Brett 2009; Grahn & McAuley 2009; Grahn & Rowe 2012; Meck 2005; Schwartze et al. 2011, 2012a,b; Teki et al. 2011). Other conceptions have stressed the importance of task difficulty; the cerebellum plays a critical role in basic timing and corticostriatal circuits are recruited in a load-dependent manner (Livesey et al. 2007, Tregellas et al. 2006). How stimulus factors, attention and task factors, and development and training factors mediate the strength of beat-based timing is currently under investigation. The assumption is that beat-based timing is engaged only within a limited range of durations, corresponding to an entrainment region that is influenced by the precision of oscillator synchronization and the resetting of coincidence-detection mechanisms at the beginning of a sequence (Coull et al. 2011, Matell & Meck 2004, McAuley et al. 2006).

Understanding the degree to which individuals engage in a beat-based versus duration-based timing mode is critically important for a better characterization of temporal-processing deficits implicated with various developmental disorders (e.g., autism spectrum disorder and ADHD) as well as neurological (e.g., Huntington's and Parkinson's diseases) and psychiatric disorders (e.g., ADHD, schizophrenia) as outlined by Allman & Meck (2012), Conners et al. (1996), Levin et al. (1996), Meck (2005), and Noreika et al. (2013). An underlying propensity to engage in beat-based timing may somehow be related to, or manifested in, the timing of other behavioral tendencies, such as the rhythmical nature of many certain repetitive behaviors performed by those with certain sensory processing disorders (e.g., autism, schizophrenia). Beat-based timing shares a similar neurological basis with stereotypic behavior (of the type also expressed by nonhuman animals), specifically the basal ganglia (and striatum) and action on the D2 receptor. The length and complexity of behavioral habits have been posited to correspond to the nature of fixed routines in autism and may function as forms of a compensatory behavioral clock (Allman & DeLeon 2009, Boucher 2001). It remains to be seen if clinical populations with severe repetitive behaviors are more or less susceptible to beat-based timing; this notion is currently being investigated in autism (Allman et al. 2012, Falter et al. 2012).

Attentional Time-Sharing

As noted above, in the internal clock model the accumulated number of pacemaker pulses depends on attentional resources allocated to interval timing (Meck 1984). When an individual attends to a second task, or when intruder/distractor events are presented, estimated durations are shorter, presumably owing to resources being diverted from timing (Champagne & Fortin 2008, Fortin et al. 2010, Grondin 2010, Macar et al. 1994, Macar & Vidal 2009). Investigators have recently extended this time-sharing hypothesis by proposing that resource reallocation is proportional to the perceived contrast, both in temporal and nontemporal features, between intruders and the timed events. Experimental findings support this extension by showing that the effect of an intruder/distractor is dependent on the relative duration of the intruder to the intertrial interval (Buhusi & Meck 2006, 2009a,b).

Individual differences in the probability of attending to separate events (e.g., multiple to-be-timed signals presented simultaneously, dual-task components, distractors, and contextual stimuli) can be monitored during an experimental session to evaluate factors governing fatigue and vigilance

(e.g., Buhusi & Meck 2006; Meck 1987; Meck & Williams 1997a,b); an overview of the importance of simultaneous temporal processing to the general architecture of the internal clock is provided by Church (1984) and Meck & Church (1982, 1984). Differences in the ability to reproduce previously experienced auditory and visual target durations either in isolation (focused attention) or concurrently (divided attention) have been reported in young adults who typically produce trained criterion durations equally well in either condition and also in older adults who often exhibit an age-related increase in timing variability in divided attention conditions and simultaneous temporal processing (e.g., Bherer et al. 2007, Lustig & Meck 2001, McAuley et al. 2010, Pang et al. 2001). Age-related impairments are often associated with a decrease in working memory span, and the relationship between working memory and timing performance is usually largest for visual targets in divided attention conditions. Moreover, time-of-day effects are frequently observed such that younger adults show better timing performance in the evening, and older adults show better performance in the morning; performance at the optimal time of day for each age group was relatively similar, but the performance for aged adults at the nonoptimal time of day was the most impaired (Lustig & Meck 2001). How these time-of-day effects emerge as a function of normal aging is uncertain, but the circadian modulation of interval timing as well as the associated genetic and molecular bases of individual differences in timing are currently topics of considerable interest (Agostino et al. 2011a,b; Balci et al. 2009).

This second-order principle is also evidenced by behavioral and neurobiological data (e.g., Bherer et al. 2007; Buhusi & Meck 2000, 2006, 2009a,b; Fortin et al. 2009; Gooch et al. 2009; McAuley et al. 2010). Brain circuits engaged by timekeeping comprise not only those primarily involved in pulse accumulation, but also those involved in the maintenance of attentional and memory resources for timing as well as in the monitoring and reallocation of those resources among tasks (Buhusi & Meck 2009a, Van Rijn et al. 2011). This view is consistent with dynamic attending theory, which proposes that attention can be modeled as a self-sustained oscillation capable of entrainment to the relevant temporal sequence of events (Large & Jones 1999).

Neuroimaging studies in humans suggest that timekeeping tasks engage brain circuits typically involved in attention and working memory (Coull et al. 2008, 2011; Coull & Nobre 2008; Livesey et al. 2007; Lustig et al. 2005; Meck & N'Diaye 2005). Studies with unilateral neglect patients have shown a dramatic underestimation of time for durations up to 60 s (Danckert et al. 2007, Merrifield et al. 2010). These findings have been extended to normal participants who indicate an increased sensitivity to the duration of events presented in left visual space as a result of the right parietal cortex being more dominant in the allocation of attentional resources required for temporal integration (Allman & Danckert 2005). Many behavioral, pharmacological, lesion, and electrophysiological studies in animals support this time-sharing hypothesis as well (Meck et al. 2008).

SUMMARY AND DESCRIPTION OF A UNIFIED MODEL OF TEMPORAL INTEGRATION

Although our sense of time is necessarily subjective and varies according to context (Agostino et al. 2008, Droit-Volet et al. 2013, Droit-Volet & Meck 2007, Wittmann 2013), fundamental properties of interval timing ability have been identified, some of which are common to the perception of other senses. These first- and second-order principles of the internal clock provide diagnostic criteria with which to assess typical and atypical individual differences in timing and time perception (see Gilaie-Dotan et al. 2011, Lake & Meck 2013). Examining the nature of both individual and population-level differences in interval timing is an exciting prospect to better understand our awareness of subjective time and how time plays such a central role in our physical and mental lives (see Tulving 2002).

Our goal has been to provide the basis for a unified model of the internal clock. For instance, Teki et al. (2012) have recently proposed a timing system within core regions of the motor network such as the cerebellum, the inferior olive, the basal ganglia, the presupplementary and supplementary motor areas, and the premotor cortex, as well as higher-level areas such as the prefrontal cortex (as illustrated in **Figure 6**). In this manner, Teki et al. (2012) have built on previous proposals

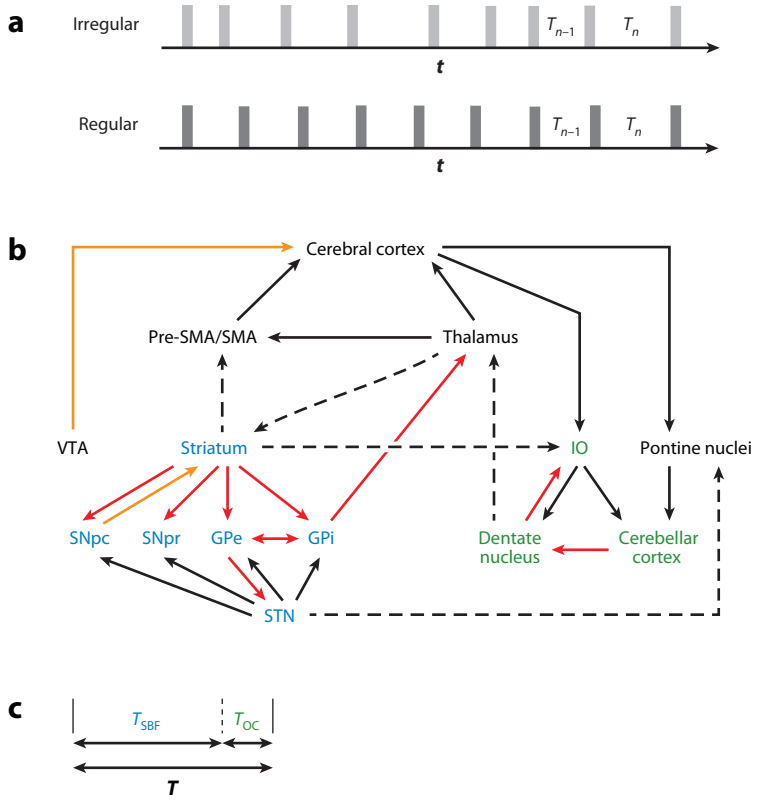

Figure 6

(*a*) Absolute and relative timing task. Irregular: A sequence of clicks with an average of 15% jitter was used to study absolute, duration-based timing. Participants were required to compare the duration of the final interval, T_n, to the penultimate interval, T_{n-1}, where T_n incorporates a difference (ΔT) of 30% of the interonset interval (range: 440–560 ms) from that of the preceding interval such that $T_n = T_{n-1} \pm \Delta T$ 30%. Regular: A sequence of clicks with no jitter is used to study relative, beat-based timing. Participants were required to compare the duration of the final interval, T_n, to the penultimate interval, T_{n-1}, where T_n incorporates a difference (ΔT) of 15% of the interonset interval from that of the preceding interval such that $T_n = T_{n-1} \pm \Delta T$ 15% (see Teki et al. 2011 for further details). (*b*) A unified model of time perception. The striatal network (*blue*) and the olivocerebellar network (*green*) are connected to each other via multiple loops, and with the thalamus, pre-SMA/SMA, and the cerebral cortex. Dopaminergic pathways are shown in orange, inhibitory projections in red, excitatory and known anatomical connections in solid and dashed black lines, respectively. Abbreviations: GPe, globus pallidus external; GPi, globus pallidus internal; IO, inferior olive; SMA, supplementary motor area; SNpc, substantia nigra pars compacta; SNpr, substantia nigra pars reticulate; STN, subthalamic nucleus; VTA, ventral tegmental area. (*c*) Timing mechanism underlying the unified model. To estimate an interval of duration *T*, both the striato-thalamo-cortical (SBF) networks and olivocerebellar (OC) networks act in parallel to produce timing signals T_{SBF} and T_{OC}, respectively, such that the combined output of the system approximates the length of the criterion time interval, *T*. Adapted from Meck (2005) and Teki et al. (2011).

by Allman & Meck (2012) and Meck (2005) to show how corticostriatal and cortico-cerebellar systems can subserve different aspects of a perceptual and motor timing system. Previous work by this research group established that olivocerebellar circuits support absolute, duration-based timing and that striato-thalamo-cortical circuits support relative, beat-based timing (Teki et al. 2011). Moreover, recent neuroimaging studies indicate that the timing functions of these circuits are codependent (Jahanshahi et al. 2010). Hence, we have proposed an integrative view of time perception based on coordinated activity in the core striatal and olivocerebellar networks, which are interconnected with each other and with the cerebral cortex through multiple synaptic pathways. Timing in this unified model may involve serial beat-based striatal activation followed by absolute olivocerebellar timing mechanisms (Allman & Meck 2012, Cope et al. 2013, Teki et al. 2012; see also Merchant et al. 2013).

FUTURE DIRECTIONS

The properties and nature of the internal clock can be investigated in many ways, and the strengths of the psychophysical approach coupled with well-specified psychological models and neurobiological mechanisms provide a fertile ground for timing research. In the context first posed by William James, future studies will need to address the degree to which the prefrontal cortex can self-generate multisecond time intervals in the absence of an external to-be-timed event (Lustig et al. 2005, Matell et al. 2011, Mita et al. 2009, Yumoto et al. 2011). Evidence of this type of temporal representation would demonstrate the successful disengagement of cortical circuits from the striatal mechanisms initially required for beat detection and entrainment to external stimuli (see Grahn & Brett 2009, Grahn & McAuley 2009, Grahn & Rowe 2012, Gu et al. 2011). Moreover, the involvement of the prefrontal and parietal cortices may need to be reconsidered in terms of understanding how the basal ganglia can train multiple cortical areas to recognize and/or produce specific sequences of durations in the manner of actor-critic models (e.g., Hampshire et al. 2011, Joel et al. 2002, O'Reilly & Frank 2006). The quest for the internal clock (translating objective time into subjective time) and its neurological basis, which is modifiable by nontemporal stimuli and pharmacological states and which reveals typical and atypical differences in various populations (individuals with neurological, psychiatric, and psychological disorders), is perhaps the elusive key to understanding consciousness in the mind and brain (Smythies et al. 2012).

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LITERATURE CITED

Acerbi L, Wolpert DM, Vijayakumar S. 2012. Internal representations of temporal statistics and feedback calibrate motor-sensory interval timing. *PLoS Comput. Biol.* 8(11):e1002771

Agostino PV, Cheng RK, Williams CL, West AE, Meck WH. 2013. Acquisition of response thresholds for timed performance is regulated by a calcium-responsive transcription factor, CaRF. *Genes Brain Behav.* 12:633–44

Agostino PV, Golombek DA, Meck WH. 2011. Unwinding the molecular basis of interval and circadian timing. *Front. Integr. Neurosci.* 5:64

Agostino PV, Nascimento MD, Bussi IL, Eguia MC, Golombek DA. 2011. Circadian modulation of interval timing in mice. *Brain Res.* 1370:154–63

Agostino PV, Peryer G, Meck WH. 2008. How music fills our emotions and helps us keep time. *Behav. Brain Sci.* 31:575–76

Allan LG. 1979. Perception of time. *Percept. Psychophys.* 26:340–54

Allan LG. 1998. The influence of the scalar timing model on human timing research. *Behav. Process.* 44:101–17

Allan LG, Gibbon J. 1991. Human bisection at the geometric mean. *Learn. Motiv.* 22:39–58

Allman A, Danckert J. 2005. Time flies when you're having fun: temporal estimation and the experience of boredom. *Brain Cogn.* 59:236–45

Allman MJ. 2011. Deficits in temporal processing associated with autistic disorder. *Front. Integr. Neurosci.* 5:2

Allman MJ, DeLeon IG. 2009. No time like the present: time perception in autism. In *Causes and Risks for Autism*, ed. AC Giordano, VA Lombardi, pp. 65–76. New York: Nova Sci.

Allman MJ, DeLeon IG, Wearden JH. 2011. A psychophysical assessment of timing in individuals with autism. *Am. J. Intellect. Dev. Disabil.* 116:165–78

Allman MJ, Meck WH. 2012. Pathophysiological distortions in time perception and timed performance. *Brain* 135:656–77

Allman MJ, Pelphrey KA, Meck WH. 2012. Developmental neuroscience of time and number: implications for autism and other neurodevelopmental disabilities. *Front. Integr. Neurosci.* 6:7

Allman MJ, Yin B, Meck WH. 2013. Time in the psychopathological mind. In *Subjective Time: An Anthology of the Philosophy, Psychology, and Neuroscience of Temporality*, ed. D Lloyd, V Arstila. Cambridge, MA: MIT Press. In press

Almeida R, Ledberg A. 2010. A biologically plausible model of time-scale invariant interval timing. *J. Comp. Neurosci.* 28:155–75

Aparicio P, Diedrichsen J, Ivry RB. 2005. Effects of focal basal ganglia lesions on timing and force control. *Brain Cogn.* 58:62–74

Artieda J, Pastor MA, Lacruz F, Obeso JA. 1992. Temporal discrimination is abnormal in Parkinson's disease. *Brain* 115:199–210

Balci F, Meck WH, Moore H, Brunner D. 2009. Timing deficits in aging and neuropathology. In *Animal Models of Human Cognitive Aging*, ed. JL Bizon, A Woods, pp. 161–201. Totowa, NJ: Humana

Balci F, Wiener M, Çavdaroğlu B. 2012. Epistasis effects of dopamine genes on interval timing and reward magnitude in humans. *Neuropsychologia* 51:293–308

Bartolo R, Merchant H. 2009. Learning and generalization of time production in humans: rules of transfer across modalities and interval durations. *Exp. Brain Res.* 197:91–100

Bechara A, Tranel D, Damasio H, Damasio AR. 1996. Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cereb. Cortex* 6:215–25

Bherer L, Desjardins S, Fortin C. 2007. Age-related differences in timing with breaks. *Psychol. Aging* 22:398–403

Boucher J. 2001. "Lost in a sea of time": time parsing and autism. In *Time and Memory*, ed. C Hoerl, T McCormack, pp. 111–35 Oxford, UK: Oxford Univ. Press

Brannon EM, Libertus ME, Meck WH, Woldorff MG. 2008. Electrophysiological measures of time processing in infant and adult brains: Weber's law holds. *J. Cogn. Neurosci.* 20:193–203

Brannon EM, Wolfe L, Meck WH, Woldorff M. 2004. Timing in the baby brain. *Cogn. Brain Res.* 21:227–33

Brody CD, Hernández A, Zainos A, Romo R. 2003. Timing and neural encoding of somatosensory parametric working memory in macaque prefrontal cortex. *Cereb. Cortex* 13:1196–207

Bueti D. 2011. The sensory representation of time. *Front. Integr. Neurosci.* 5:34

Bueti D, Bahrami B, Walsh V. 2008a. Sensory and association cortex in time perception. *J. Cogn. Neurosci.* 20:1054–62

Bueti D, Macaluso E. 2010. Auditory temporal expectation modulates activity in visual cortex. *NeuroImage* 51:1168–83

Bueti D, Macaluso E. 2011. Physiological correlates of subjective time: evidence for the temporal accumulator hypothesis. *NeuroImage* 57:1251–63

Bueti D, van Dongen EV, Walsh V. 2008b. The role of superior temporal cortex in auditory timing. *PLoS ONE* 3(6):e 2481

Bueti D, Walsh V. 2009. The parietal cortex and the representation of time, space, number and other magnitudes. *Philos. Trans. R. Soc. B* 364:1831–40

Buhusi CV, Aziz D, Winslow D, Carter RE, Swearingen JE, Buhusi MC. 2009. Interval timing accuracy and scalar timing in C57BL/6 mice. *Behav. Neurosci.* 123:1102–13

Buhusi CV, Meck WH. 2000. Timing for the absence of a stimulus: the gap paradigm reversed. *J. Exp. Psychol. Anim. Behav. Process.* 26:305–22

Buhusi CV, Meck WH. 2002. Differential effects of methamphetamine and haloperidol on the control of an internal clock. *Behav. Neurosci.* 116:291–97

Buhusi CV, Meck WH. 2005. What makes us tick? Functional and neural mechanisms of interval timing. *Nat. Rev. Neurosci.* 6:755–65

Buhusi CV, Meck WH. 2006. Interval timing with gaps and distracters: evaluation of the ambiguity, switch, and time-sharing hypotheses. *J. Exp. Psychol. Anim. Behav. Process.* 32:329–38

Buhusi CV, Meck WH. 2009a. Relative time sharing: new findings and an extension of the resource allocation model of temporal processing. *Philos. Trans. R. Soc. B* 364:1875–85

Buhusi CV, Meck WH. 2009b. Relativity theory and time perception: single or multiple clocks? *PLoS ONE* 4(7):e6268

Buhusi CV, Oprisan SA. 2013. Time-scale invariance as an emergent property in a perceptron with realistic, noisy neurons. *Behav. Process.* 95:60–70

Buhusi CV, Perera D, Meck WH. 2005. Memory for timing visual and auditory signals in albino and pigmented rats. *J. Exp. Psychol. Anim. Behav. Process.* 31:18–30

Buhusi CV, Sasaki A, Meck WH. 2002. Temporal integration as a function of signal/gap intensity in rats (*Rattus norvegicus*) and pigeons (*Columba livia*). *J. Comp. Psychol.* 116:381–90

Buonomano DV, Laje R. 2010. Population clocks: motor timing with neural dynamics. *Trends Cogn. Sci.* 14:520–27

Burr D, Rocca ED, Morrone MC. 2013. Contextual effects in interval-duration judgements in vision, audition and touch. *Exp. Brain Res.* In press. doi:10.1007/s00221-013-3632-z

Carroll CA, Boggs J, O'Donnell BF, Shekhar A, Hetrick WP. 2008. Temporal processing dysfunction in schizophrenia. *Brain Cogn.* 67:150–61

Champagne J, Fortin C. 2008. Attention sharing during timing: modulation by processing demands of an expected stimulus. *Percept. Psychophys.* 70:630–39

Cheng RK, Ali YM, Meck WH. 2007. Ketamine "unlocks" the reduced clock-speed effect of cocaine following extended training: evidence for dopamine-glutamate interactions in timing and time perception. *Neurobiol. Learn. Mem.* 88:149–59

Cheng RK, Dyke AG, McConnell MW, Meck WH. 2011. Categorical scaling of duration as a function of temporal context in aged rats. *Brain Res.* 1381:175–86

Cheng RK, MacDonald CJ, Meck WH. 2006. Differential effects of cocaine and ketamine on time estimation: implications for neurobiological models of interval timing. *Pharmacol. Biochem. Behav.* 85:114–22

Cheng RK, Meck WH. 2007. Prenatal choline supplementation increases sensitivity to time by reducing non-scalar sources of variance in adult temporal processing. *Brain Res.* 1186:242–54

Cheng RK, Scott AC, Penney TB, Williams CL, Meck WH. 2008. Prenatal choline availability differentially modulates timing of auditory and visual stimuli in aged rats. *Brain Res.* 1237:167–75

Church RM. 1984. Properties of an internal clock. *Ann. N.Y. Acad. Sci.* 423:566–82

Church RM. 1989. Theories of timing behavior. In *Contemporary Learning Theories: Instrumental Conditioning Theory and the Impact of Biological Constraints on Learning*, ed. SB Klein, RR Mowrer, pp. 4 1–71. Hillsdale, NJ: Erlbaum

Church RM. 2003. A concise introduction to scalar expectancy theory. See Meck 2003, pp. 3–22

Church RM, Deluty MZ. 1977. Bisection of temporal intervals. *J. Exp. Psychol. Anim. Behav. Process.* 3:216–28

Church RM, Meck WH, Gibbon J. 1994. Application of scalar timing theory to individual trials. *J. Exp. Psychol. Anim. Behav. Process.* 20:135–55

Church RM, Miller KD, Meck WH, Gibbon J. 1991. Symmetrical and asymmetrical sources of variance in temporal generalization. *Anim. Learn. Behav.* 19:207–14

Cicchini GM, Arrighi R, Cecchetti L, Giusti M, Burr DC. 2012. Optimal encoding of interval timing in expert percussionists. *J. Neurosci.* 32:1056–60

Claassen DO, Jones CR, Yu M, Dirnberger G, Malone T, et al. 2013. Deciphering the impact of cerebellar and basal ganglia dysfunction in accuracy and variability of motor timing. *Neuropsychologia* 51:267–74

Conners CK, Levin ED, Sparrow E, Hinton SC, Erhardt D, et al. 1996. Nicotine and attention in adult attention deficit hyperactivity disorder (ADHD). *Psychopharmacol. Bull.* 32:67–73

Cope TE, Grube M, Singh B, Burh DJ, Griffiths TD. 2013. The basal ganglia in perceptual timing: timing performance in multiple system atrophy and Huntington's disease. *Neuropsychologia*. In press

Cordes S, Meck WH. 2013. Ordinal judgment in the rat: an understanding of longer and shorter for suprasecond, but not subsecond, durations. *J. Exp. Psychol. Gen.* In press

Cordes S, Williams CL, Meck WH. 2007. Common representations of abstract quantities. *Curr. Direct. Psychol. Sci.* 16:156–61

Coull JT, Cheng RK, Meck WH. 2011. Neuroanatomical and neurochemical substrates of timing. *Neuropsychopharmacology* 36:3–25

Coull JT, Nazarian B, Vidal F. 2008. Timing, storage, and comparison of stimulus duration engage discrete anatomical components of a perceptual timing network. *J. Cogn. Neurosci.* 20:2185–97

Coull J, Nobre A. 2008. Dissociating explicit timing from temporal expectation with fMRI. *Curr. Opin. Neurobiol.* 18:137–44

Coull JT, Vidal F, Nazarian B, Macar F. 2003. Functional anatomy of the attentional modulation of time estimation. *Science* 303:1506–8

Dali Y, Zemin L. 1993. A multilayer feedforward neural network with adaptive lookup table weight. *Circuit. Sys. IEEE* 4:2411–14

Danckert J, Ferber S, Pun C, Broderick C, Striemer C, et al. 2007. Neglected time: impaired temporal perception of multisecond intervals in unilateral neglect. *J. Cogn. Neurosci.* 19:1706–20

Diedrichsen J, Ivry RB, Pressing J. 2003. Cerebellar and basal ganglia contributions to interval timing. See Meck 2003, pp. 457–83

Droit-Volet S, Clément A, Wearden JH. 2001. Temporal generalization in 3- to 8-year-old children. *J. Exp. Child Psychol.* 80:271–88

Droit-Volet S, Fayolle S, Lamotte M, Gil S. 2013. Time, emotion and the embodiment of timing. *Timing Time Percept.* In press

Droit-Volet S, Meck WH. 2007. How emotions colour our perception of time. *Trends Cogn. Sci.* 11:504–13

Droit-Volet S, Meck WH, Penney TB. 2007. Sensory modality and time perception in children and adults. *Behav. Process.* 74:244–50

Droit-Volet S, Wearden JH. 2001. Temporal bisection in children. *J. Exp. Child Psychol.* 80:142–59

Durstewitz D, Deco G. 2008. Computational significance of transient dynamics in cortical networks. *Eur. J. Neurosci.* 27:217–27

Falter CM, Noreika V, Wearden JH, Bailey AJ. 2012. More consistent, yet less sensitive: interval timing in autism spectrum disorders. *Q. J. Exp. Psychol.* 65:2093–107

Forstmann BU, Anwander A, Schäfer A, Neumann J, Brown S, et al. 2010. Cortico-striatal connections predict control over speed and accuracy in perceptual decision making. *Proc. Natl. Acad. Sci. USA* 107:15916–20

Fortin C, Fairhurst S, Malapani C, Morin C, Towey J, Meck WH. 2009. Expectancy in humans in multisecond peak-interval timing with gaps. *Atten. Percept. Psychophys.* 71:789–802

Fortin C, Schweickert R, Gaudreault R, Viau-Quesnel C. 2010. Timing is affected by demands in memory search but not by task switching. *J. Exp. Psychol. Hum. Percept. Perform.* 36:580–95

Fraisse P. 1984. Perception and estimation of time. *Annu. Rev. Psychol.* 35:1–36

Gescheider GA. 1997. *Psychophysics: The Fundamentals*. Mahwah, NJ: Erlbaum

Gibbon J. 1977. Scalar expectancy and Weber's law in animal timing. *Psychol. Rev.* 84:279–325

Gibbon J. 1991. Origins of scalar timing theory. *Learn. Motiv.* 22:3–38

Gibbon J. 1992. Ubiquity of scalar timing with a Poisson clock. *J. Math. Psychol.* 35:283–93

Gibbon J, Church RM. 1984. Sources of variance in an information processing theory of timing. In *Animal Cognition*, ed. HL Roitblat, TG Bever, HS Terrace, pp. 465–88. Hillsdale, NJ: Erlbaum

Gibbon J, Church RM. 1990. Representation of time. *Cognition* 37:23–54

Gibbon J, Church RM. 1992. Comparison of variance and covariance patterns in parallel and serial theories of timing. *J. Exp. Anal. Behav.* 57:393–406

Gibbon J, Church RM, Meck WH. 1984. Scalar timing in memory. *Ann. N.Y. Acad. Sci.* 423:52–77

Gibbon J, Malapani C, Dale CL, Gallistel CR. 1997. Toward a neurobiology of temporal cognition: advances and challenges. *Curr. Opin. Neurobiol.* 7:170–84

Gilaie-Dotan S, Kanai R, Rees G. 2011. Anatomy of human sensory cortices reflects inter- individual variability in time estimation. *Front. Integr. Neurosci.* 5:76

Girardi G, Antonucci G, Nico D. 2013. Cueing spatial attention through timing and probability. *Cortex* 49:211–21

Gooch CM, Stern Y, Rakitin BC. 2009. Evidence for age-related changes to temporal attention and memory from the choice time production task. *Aging Neuropsychol. Cogn.* 16:285–310

Grahn JA, Brett M. 2009. Impairment of beat-based rhythm discrimination in Parkinson's disease. *Cortex* 45:54–61

Grahn JA, McAuley JD. 2009. Neural bases of individual differences in beat perception. *NeuroImage* 47:1894–903

Grahn JA, Rowe JB. 2012. Finding and feeling the musical beat: striatal dissociations between detection and prediction of regularity. *Cereb. Cortex* 23:91–21

Grondin S. 2010. Timing and time perception: a review of recent behavioral and neuroscience findings and theoretical directions. *Atten. Percept. Psychophys.* 72:561–82

Grube M, Cooper FE, Chinnery PF, Griffiths TD. 2010a. Dissociation of duration-based and beat-based auditory timing in cerebellar degeneration. *Proc. Natl. Acad. Sci. USA* 107:11597–601

Grube M, Lee K-H, Griffiths TD, Barker AT, Woodruff PW. 2010b. Transcranial magnetic theta-burst stimulation of the human cerebellum distinguishes absolute, duration-based from relative, beat-based perception of subsecond time intervals. *Front. Psychol.* 1:171

Gu B-M, Jurkowski AJ, Lake JI, Malapani C, Meck WH. 2013. Bayesian models of interval timing and distortions in temporal memory as a function of Parkinson's disease and dopamine-related error processing. See Vatakis & Allman 2013. In press

Gu B-M, Meck WH. 2011. New perspectives on Vierordt's law: memory-mixing in ordinal temporal comparison tasks. In *Multidisciplinary Aspects of Time and Time Perception*, ed. A Vatakis, A Esposito, M Giagkou, F Cummins, G Papadelis, pp. 67–78. Lect. Notes Comp. Sci. Ser. Vol. 6789. Berlin: Springer

Gu BM, Cheng RK, Yin B, Meck WH. 2011. Quinpirole-induced sensitization to noisy/sparse periodic input: temporal synchronization as a model of obsessive-compulsive disorder. *Neuroscience* 179:143–50

Guilhardi P, Church RM. 2009. The generality of empirical and theoretical explanations of behavior. *Behav. Process.* 81:205–15

Hampshire A, Thompson R, Duncan J, Owen AM. 2011. Lateral prefrontal cortex subregions make dissociable contributions during fluid reasoning. *Cereb. Cortex* 21:1–10

Harrington DL, Boyd LA, Mayer AR, Sheltraw DM, Lee RR, et al. 2004. Neural representation of interval encoding and decision making. *Cogn. Brain Res.* 21:193–205

Harrington DL, Castillo GN, Fong CH, Reed JD. 2011a. Neural underpinnings of distortions in the experience of time across senses. *Front. Integr. Neurosci.* 5:32

Harrington DL, Castillo GN, Greenberg PA, Song DD, Lessig S, et al. 2011b. Neurobehavioral mechanisms of temporal processing deficits in Parkinson's disease. *PLoS ONE* 6(2):e17461

Harrington DL, Haaland K, Knight R. 1998. Cortical networks underlying mechanism of time perception. *J. Neurosci.* 18: 1085–95

Harrington DL, Zimbelman JL, Hinton SC, Rao SM. 2010. Neural modulation of temporal encoding, maintenance, and decision processes. *Cereb. Cortex* 20:1274–85

Hass J, Blaschke S, Herrmann JM. 2012. Cross-modal distortion of time perception: demerging the effects of observed and performed motion. *PLoS ONE* 7(6):e38092

Hinton SC, Meck WH. 1997a. How time flies: functional and neural mechanisms of interval timing. In *Time and Behaviour: Psychological and Neurobiological Analyses*, ed. CM Bradshaw, E Szabadi, pp. 409–57. New York: Elsevier

Hinton SC, Meck WH. 1997b. The "internal clocks" of circadian and interval timing. *Endeavour* 21:82–87

Hinton SC, Meck WH. 2004. Frontal-striatal circuitry activated by human peak-interval timing in the supra-seconds range. *Cogn. Brain Res.* 21:171–82
Hinton SC, Paulsen JS, Hoffmann RG, Reynolds NC, Zimbelman JL, Rao SM. 2007. Motor timing variability increases in preclinical Huntington's disease patients as estimated onset of motor symptoms approaches. *J. Int. Neuropsychol. Soc.* 13:1–5
Hinton SC, Rao SM. 2004. "One-thousand *one* . . . one-thousand *two* . . .": Chronometric counting violates the scalar property in interval timing. *Psychon. Bull. Rev.* 11:24–30
Hoerl C, McCormack T, eds. 2001. *Time and Memory: Issues in Philosophy and Psychology*. Oxford, UK: Clarendon
Ivry RB, Schlerf JE. 2008. Dedicated and intrinsic models of time perception. *Trends Cogn. Sci.* 12:273–80
Ivry RB, Spencer RMC. 2004. The neural representation of time. *Curr. Opin. Neurobiol.* 14:225–32
Jaffe J, Beebe B, Feldstein S, Crown C, Jasnow M. 2001. *Rhythms of Dialogue in Infancy: Monographs for the Society for Research and Child Development*. Serial No. 265, Vol. 66. Boston, MA: Blackwell
Jahanshahi M, Jones CR, Zijlmans J, Katsenschlager R, Lee L, et al. 2010. Dopaminergic modulation of striato-frontal connectivity during motor timing in Parkinson's disease. *Brain* 133:727–45
Jaldow EJ, Oakley DA, Davey GC. 1989. Performance of decorticated rats on fixed interval and fixed time schedules. *Eur. J. Neurosci.* 5:461–70
Jaldow EJ, Oakley DA, Davey GC. 1990. Performance on two fixed-interval schedules in the absence of neocortex in rats. *Behav. Neurosci.* 104:763–77
James W. 1890. The perception of time. In *The Principles of Psychology*, Vol. 1. New York: Holt
Jazayeri M, Shadlen MN. 2010. Temporal context calibrates interval timing. *Nat. Neurosci.* 13:1020–26
Jin, DZ, Fujii N, Graybiel AM. 2009. Neural representation of time in cortico-basal ganglia circuits. *Proc. Natl. Acad. Sci. USA* 106:19156–61
Joel D, Niv Y, Ruppin E. 2002. Actor-critic models of the basal ganglia: new anatomical and computational perspectives. *Neural Netw.* 15:535–47
Jones CR, Claassen DO, Yu M, Spies JR, Malone T, et al. 2011. Modeling accuracy and variability of motor timing in treated and untreated Parkinson's disease and healthy controls. *Front. Integr. Neurosci.* 5:81
Jones CRG, Jahanshahi M. 2013. Contributions of the basal ganglia to temporal processing: evidence from Parkinson's disease. *Timing Time Percept*. In press
Jones CRG, Malone TJL, Dirnberger G, Edwards M, Jahanshahi M. 2008. Basal ganglia, dopamine and temporal processing: performance on three timing tasks on and off medication in Parkinson's disease. *Brain Cogn.* 68:30–41
Kanai R, Lloyd H, Bueti D, Walsh V. 2011. Modality-independent role of the primary auditory cortex in time estimation. *Exp. Brain Res.* 209:465–71
Karmarkar UR, Buonomano DV. 2007. Timing in the absence of clocks: encoding time in neural network states. *Neuron* 53:427–38
Kaufman J, Csibra G, Johnson MH. 2005. Oscillatory activity in the infant brain reflects object maintenance. *Proc. Natl. Acad. Sci. USA* 102:15271–74
Killeen PR. 1991. Counting the minutes. In *Time, Action, and Cognition: Towards Bridging the Gap*, ed. F Macar, V Pouthas, WJ Friedman, pp. 203–14. Dordrecht: Kluwer
Killeen PR, Fetterman JG. 1988. A behavioral theory of timing. *Psychol. Rev.* 95:274–95
Killeen PR, Weiss NA. 1987. Optimal timing and the Weber function. *Psychol. Rev.* 94:455–68
Kimura D, Hayakawa Y. 2008. Reinforcement learning of recurrent neural network for temporal coding. *Neurocomputing* 71:3379–86
Koch G, Costa A, Brusa L, Peppe A, Gatto I, et al. 2008. Impaired reproduction of second but not millisecond time interval in Parkinson's disease. *Neuropsychologica* 46:1305–13
Koch G, Oliveri M, Caltagirone C. 2009. Neural networks engaged in milliseconds and seconds time processing: evidence from transcranial magnetic stimulation and patients with cortical or subcortical dysfunction. *Philos. Trans. R. Soc. B* 364:1907–18
Koch G, Oliveri M, Carlesimo GA, Caltagirone C. 2002. Selective deficit of time perception in a patient with right prefrontal cortex lesion. *Neurology* 59:1658–59
Koch G, Oliveri M, Torriero S, Caltagirone C. 2003. Underestimation of time perception after repetitive transcranial magnetic stimulation. *Neurology* 60:1844–46

Kotz SA, Schwartze M. 2010. Cortical speech processing unplugged: a timely subcortico-cortical framework. *Trends Cogn. Sci.* 14:392–99

Kotz SA, Schwartze M, Schmidt-Kassow M. 2009. Non-motor basal ganglia functions: a review and proposal for a model of sensory predictability in auditory language perception. *Cortex* 45:982–90

Kotz SAE, Schwartze M. 2011. Differential input of the supplementary motor area to a dedicated temporal processing network: functional and clinical implications. *Front. Integr. Neurosci.* 5:86

Kurti AN, Matell MS. 2011. Nucleus accumbens dopamine modulates response rate but not response timing in an interval timing task. *Behav. Neurosci.* 125:215–25

Laje R, Buonomano DV. 2013. Robust timing and motor patterns by taming chaos in recurrent neural networks. *Nat. Neurosci.* 16:925–33

Lake JI, Meck WH. 2013. Differential effects of amphetamine and haloperidol on temporal reproduction: dopaminergic regulation of attention and clock speed. *Neuropsychologia* 51:284–92

Large EW, Jones MR. 1999. The dynamics of attending: how we track time-varying events. *Psychol. Rev.* 106:119–59

Levin ED, Conners CK, Sparrow E, Hinton SC, Erhardt D, et al. 1996. Nicotine effects on adults with attention-deficit/hyperactivity disorder. *Psychopharmacology* 123:55–63

Lewis PA, Meck WH. 2012. Time and the sleeping brain. *Psychologist* 25:594–97

Lewis PA, Miall RC. 2003. Distinct systems for automatic and cognitively controlled time measurement: evidence from neuroimaging. *Curr. Opin. Neurobiol.* 13:250–55

Lewis PA, Miall RC. 2009. The precision of temporal judgement: milliseconds, many minutes, and beyond. *Philos. Trans. R. Soc. B* 364:1897–905

Livesey AC, Wall MB, Smith AT. 2007. Time perception: manipulation of task difficulty dissociates clock functions from other cognitive demands. *Neuropsychologia* 45:321–31

Lustig C, Matell MS, Meck WH. 2005. Not "just" a coincidence: frontal-striatal synchronization in working memory and interval timing. *Memory* 13:441–48

Lustig C, Meck WH. 2001. Paying attention to time as one gets older. *Psychol. Sci.* 12:478–84

Lustig C, Meck WH. 2005. Chronic treatment with haloperidol induces working memory deficits in feedback effects of interval timing. *Brain Cogn.* 58:9–16

Lustig C, Meck WH. 2011. Modality differences in timing and temporal memory throughout the lifespan. *Brain Cogn.* 77:298–303

Macar F, Grondin S, Casini L. 1994. Controlled attention sharing influences time estimation. *Mem. Cogn.* 22:673–86

Macar F, Vidal F. 2009. Timing processes: an outline of behavioural and neural indices not systematically considered in timing models. *Can. J. Exp. Psychol.* 63:227–39

MacDonald CJ, Cheng R-K, Meck WH. 2012. Acquisition of "start" and "stop" response thresholds in peak-interval timing is differentially sensitive to protein synthesis inhibition in the dorsal and ventral striatum. *Front. Integr. Neurosci.* 6:10

MacDonald CJ, Cheng R-K, Williams CL, Meck WH. 2007. Combined organizational and activational effects of short and long photoperiods on spatial and temporal memory in rats. *Behav. Process.* 74:226–33

MacDonald CJ, Meck WH. 2004. Systems-level integration of interval timing and reaction time. *Neurosci. Biobehav. Rev.* 28:747–69

MacDonald CJ, Meck WH. 2005. Differential effects of clozapine and haloperidol on interval timing in the supraseconds range. *Psychopharmacology* 182:232–44

MacDonald CJ, Meck WH. 2006. Interaction of raclopride and preparatory-interval effects on simple reaction-time performance. *Behav. Brain Res.* 175:62–74

Malapani C, Fairhurst S. 2002. Scalar timing in animals and humans. *Learn. Motiv.* 33:156–76

Malapani C, Rakitin B, Meck WH, Deweer B, Dubois B, Gibbon J. 1998. Coupled temporal memories in Parkinson's disease: a dopamine-related dysfunction. *J. Cogn. Neurosci.* 10:316–31

Maricq AV, Church RM. 1983. The differential effects of haloperidol and methamphetamine on time estimation in the rat. *Psychopharmacology* 79:10–15

Maricq AV, Roberts S, Church RM. 1981. Methamphetamine and time estimation. *J. Exp. Psychol. Anim. Behav. Process.* 7:18–30

Matell MS, Bateson M, Meck WH. 2006. Single-trials analyses demonstrate that increases in clock speed contribute to the methamphetamine-induced horizontal shifts in peak-interval timing functions. *Psychopharmacology* 188:201–12

Matell MS, King GR, Meck WH. 2004. Differential adjustment of interval timing by the chronic administration of intermittent or continuous cocaine. *Behav. Neurosci.* 118:150–56

Matell MS, Meck WH. 2000. Neuropsychological mechanisms of interval timing behaviour. *BioEssays* 22:94–103

Matell MS, Meck WH. 2004. Cortico-striatal circuits and interval timing: coincidence-detection of oscillatory processes. *Cogn. Brain Res.* 21:139–70

Matell MS, Meck WH, Nicolelis MAL. 2003. Interval timing and the encoding of signal duration by ensembles of cortical and striatal neurons. *Behav. Neurosci.* 117:760–73

Matell MS, Shea-Brown E, Gooch C, Wilson AG, Rinzel J. 2011. A heterogeneous population code for elapsed time in rat medial agranular cortex. *Behav. Neurosci.* 125:54–73

McAuley JD, Henry MJ. 2010. Modality effects in rhythm processing: Auditory encoding of visual rhythms is neither obligatory nor automatic. *Atten. Percept. Psychophys.* 72:1377–89

McAuley JD, Jones MR. 2003. Modeling effects of rhythmic context on perceived duration: a comparison of interval and entrainment approaches to short-interval timing. *J. Exp. Psychol. Hum. Percept. Perform.* 29:1102–25

McAuley JD, Jones MR, Holub S, Johnston HM, Miller NS. 2006. The time of our lives: lifespan development of timing and event tracking. *J. Exp. Psychol. Gen.* 135:348–67

McAuley JD, Miller NS. 2007. Picking up the pace: effects of global temporal context on sensitivity to the tempo of auditory sequences. *Percept. Psychophys.* 69:709–18

McAuley JD, Wang M, Miller JP, Pang KCH. 2010. Dividing time: concurrent timing of auditory and visual events. *Exp. Aging Res.* 36:306–24

Meck WH. 1983. Selective adjustment of the speed of internal clock and memory processes. *J. Exp. Psychol. Anim. Behav. Process.* 9:171–201

Meck WH. 1984. Attentional bias between modalities: effect on the internal clock, memory, and decision stages used in animal time discrimination. *Ann. N.Y. Acad. Sci.* 423:528–41

Meck WH. 1986. Affinity for the dopamine D_2 receptor predicts neuroleptic potency in decreasing the speed of an internal clock. *Pharmacol. Biochem. Behav.* 25:1185–89

Meck WH. 1987. Vasopressin metabolite neuropeptide facilitates simultaneous temporal processing. *Behav. Brain Res.* 23:147–57

Meck WH. 1988. Hippocampal function is required for feedback control of an internal clock's criterion. *Behav. Neurosci.* 102:54–60

Meck WH. 1991. Modality-specific circadian rhythmicities influence mechanisms of attention and memory for interval timing. *Learn. Motiv.* 22:153–79

Meck WH. 1996. Neuropharmacology of timing and time perception. *Cogn. Brain Res.* 3:227–42

Meck WH. 2001. Interval timing and genomics: What makes mutant mice tick? *Int. J. Comp. Psychol.* 14:211–31

Meck WH. 2002a. Choline uptake in the frontal cortex is proportional to the absolute error of a temporal memory translation constant in mature and aged rats. *Learn. Motiv.* 33:88–104

Meck WH. 2002b. Distortions in the content of temporal memory: neurobiological correlates. In *Animal Cognition and Sequential Behavior: Behavioral, Biological, and Computational Perspectives*, ed. SB Fountain, MD Bunsey, JH Danks, MK McBeath, pp. 175–200. Boston, MA: Kluwer Acad.

Meck WH. 2003. *Functional and Neural Mechanisms of Interval Timing*. Boca Raton, FL: CRC Press

Meck WH. 2005. Neuropsychology of timing and time perception. *Brain Cogn.* 58:1–8

Meck WH. 2006a. Frontal cortex lesions eliminate the clock speed effect of dopaminergic drugs on interval timing. *Brain Res.* 1108:157–67

Meck WH. 2006b. Neuroanatomical localization of an internal clock: a functional link between mesolimbic, nigrostriatal, and mesocortical dopaminergic systems. *Brain Res.* 1109:93–107

Meck WH. 2006c. Temporal memory in mature and aged rats is sensitive to choline acetyltransferase inhibition. *Brain Res.* 1108:168–75

Meck WH. 2007. Acute ethanol potentiates the clock-speed enhancing effects of nicotine on timing and temporal memory. *Alcohol. Clin. Exp. Res.* 31:2106–13

Meck WH, Benson AM. 2002. Dissecting the brain's internal clock: how frontal-striatal circuitry keeps time and shifts attention. *Brain Cogn.* 48:195–211

Meck WH, Cheng RK, MacDonald CJ, Gainetdinov RR, Caron MG, Çevik MÖ. 2012. Gene-dose dependent effects of methamphetamine on interval timing in dopamine-transporter knockout mice. *Neuropharmacology* 62:1221–29

Meck WH, Church RM. 1982. Abstraction of temporal attributes. *J. Exp. Psychol. Anim. Behav. Process.* 8:226–43

Meck WH, Church RM. 1983. A mode control model of counting and timing processes. *J. Exp. Psychol. Anim. Behav. Process.* 9:320–34

Meck WH, Church RM. 1984. Simultaneous temporal processing. *J. Exp. Psychol. Anim. Behav. Process* 10:1–29

Meck WH, Church RM, Matell MS. 2013. Hippocampus, time, and memory—a retrospective analysis. *Behav. Neurosci.* In press

Meck WH, Church RM, Olton DS. 1984. Hippocampus, time, and memory. *Behav. Neurosci.* 98:3–22

Meck WH, Church RM, Wenk GL. 1986. Arginine vasopressin inoculates against age-related increases in sodium-dependent high affinity choline uptake and discrepancies in the content of temporal memory. *Eur. J. Pharmacol.* 130:327–31

Meck WH, Church RM, Wenk GL, Olton DS. 1987. Nucleus basalis magnocellularis and medial septal area lesions differentially impair temporal memory. *J. Neurosci.* 7:3505–11

Meck WH, Malapani C. 2004. Neuroimaging of interval timing. *Cogn. Brain Res.* 21:133–37

Meck WH, N'Diaye K. 2005. Un modèle neurobiologique de la perception et de l'estimation du temps. *Psychol. Fr.* 50:47–63

Meck WH, Penney TB, Pouthas V. 2008. Cortico-striatal representation of time in animals and humans. *Curr. Opin. Neurobiol.* 18:145–52

Meck WH, Williams CL. 1997a. Characterization of the facilitative effects of perinatal choline supplementation on timing and temporal memory. *Neuroreport* 8:2831–35

Meck WH, Williams CL. 1997b. Simultaneous temporal processing is sensitive to prenatal choline availability in mature and aged rats. *Neuroreport* 8:3045–51

Melgire M, Ragot R, Samson S, Penney TB, Meck WH, Pouthas V. 2005. Auditory/visual duration bisection in patients with left or right medial-temporal lobe resection. *Brain Cogn.* 58:119–24

Merchant H, Harrington DL, Meck WH. 2013. Neural basis of the perception and estimation of time. *Annu. Rev. Neurosci.* 36:313–36

Merrifield C, Hurwitz M, Danckert J. 2010. Multimodal temporal perception deficits in a patient with left spatial neglect. *J. Cogn. Neurosci.* 1:244–53

Mita A, Mushiake H, Shima K, Matsuzaka Y, Tanji J. 2009. Interval timing coding by neurons in the presupplementary and supplementary motor areas. *Nat. Neurosci.* 12:502–7

N'Diaye K, Ragot R, Garnero L, Pouthas V. 2004. What is common to brain activity evoked by the perception of visual and auditory filled durations? A study with MEG and EEG co-recordings. *Cogn. Brain Res.* 21:250–68

Nobre K, Coull J. 2010. *Attention and Time*. Oxford, UK: Oxford Univ. Press

Noreika V, Falter CM, Rubia K. 2013. Timing deficits in attention-deficit/hyperactivity disorder (ADHD): evidence from neurocognitive and neuroimaging studies. *Neuropsychologia* 51:235–66

Nussbaum S, Liberman N, Trope Y. 2006. Predicting the near and distant future. *J. Exp. Psychol. Gen.* 135:152–61

Nyberg L, Kim AS, Habib R, Levine B, Tulving E. 2010. Consciousness of subjective time in the brain. *Proc. Natl. Acad. Sci. USA* 107:22356–59

Ogden RS, Wearden JH, Jones LA. 2010. Are memories for duration modality specific? *Q. J. Exp. Psychol.* 63:65–80

Olton DS, Wenk GL, Church RM, Meck WH. 1988. Attention and the frontal cortex as examined by simultaneous temporal processing. *Neuropsychologia* 26:307–18

Onoe H, Komori M, Onoe K, Takechi H, Tsukada H, Watanabe Y. 2001. Cortical networks recruited for time perception: a monkey positron emission tomography (PET) study. *NeuroImage* 13:37–45

Oprisan SA, Buhusi CV. 2011. Modeling pharmacological clock and memory patterns of interval timing in a striatal beat-frequency model with realistic, noisy neurons. *Front. Integr. Neurosci.* 5:52

O'Reilly RC, Frank MJ. 2006. Making working memory work: a computational model of learning in the prefrontal cortex and basal ganglia. *Neural Comp.* 18:282–28

Oshio K, Chiba A, Inase M. 2008. Temporal filtering by prefrontal neurons in duration discrimination. *Eur. J. Neurosci.* 28:2333–43

Pang KCH, Yoder RM, Olton DS. 2001. Neurons in the lateral agranular frontal cortex have divided attention correlates in a simultaneous temporal processing task. *Neuroscience* 103:615–28

Paule MG, Meck WH, McMillan DE, McClure GYH, Bateson M, et al. 1999. The use of timing behaviors in animals and humans to detect drug and/or toxicant effects. *Neurotoxicol. Teratol.* 21:491–502

Penney TB, Allan LG, Meck WH, Gibbon J. 1998. Memory mixing in duration bisection. In *Timing of Behavior: Neural, Psychological and Computational Perspectives*, ed. DA Rosenbaum, CE Collyer, pp. 165–93. Cambridge, MA: MIT Press

Penney TB, Gibbon J, Meck WH. 2000. Differential effects of auditory and visual signals on clock speed and temporal memory. *J. Exp. Psychol. Hum. Percept. Perform.* 26:1770–87

Penney TB, Gibbon J, Meck WH. 2008. Categorical scaling of duration bisection in pigeons (*Columba livia*), mice (*Mus musculus*), and humans (*homo sapiens*). *Psychol. Sci.* 19:1103–9

Penney TB, Holder MD, Meck WH. 1996. Clonidine-induced antagonism of norepinephrine modulates the attentional processes involved in peak-interval timing. *Exp. Clin. Psychopharmacol.* 4:82–92

Penney TB, Meck WH, Roberts SA, Gibbon J, Erlenmeyer-Kimling L. 2005. Interval-timing deficits in individuals at high risk for schizophrenia. *Brain Cogn.* 58:109–18

Piras F, Coull JT. 2011. Implicit, predictive timing draws upon the same scalar representation of time as explicit timing. *PLoS ONE* 6(3):e18203

Pronin E. 2013. When the mind races: effects of thought speed on feeling and action. *Curr. Dir. Psychol. Sci.* 22:282–88

Pronin E, Jacobs E. 2008. Thought speed, mood, and the experience of mental motion. *Perspect. Psychol. Sci.* 3:461–85

Pronin E, Jacobs E, Wegner DM. 2008. Psychological effects of thought acceleration. *Emotion* 8:597–612

Pronin E, Wegner DM. 2006. Manic thinking: independent effects of thought speed and thought content on mood. *Psychol. Sci.* 17:807–13

Rakitin BC, Gibbon J, Penney TB, Malapani C, Hinton SC, Meck WH. 1998. Scalar expectancy theory and peak-interval timing in humans. *J. Exp. Psychol. Anim. Behav. Process.* 24:15–33

Rao SM, Mayer AR, Harrington DL. 2001. The evolution of brain activation during temporal processing. *Nat. Neurosci.* 4:317–23

Salvioni P, Murray MM, Kalmbach L, Bueti D. 2013. How the visual brain encodes and keeps track of time. *J. Neurosci.* 33:12423–29

Schirmer A. 2004. Timing speech: a review of lesion and neuroimaging findings. *Cogn. Brain Res.* 21:269–87

Schwartze M, Keller PE, Patel AD, Kotz SA. 2011. The impact of basal ganglia lesions on sensorimotor synchronization, spontaneous motor tempo, and the detection of tempo changes. *Behav. Brain Res.* 216:685–91

Schwartze M, Rothermich K, Kotz SA. 2012a. Functional dissociation of pre-SMA and SMA-proper in temporal processing. *NeuroImage* 60:290–98

Schwartze M, Tavano A, Schroger E, Kotz SA. 2012b. Temporal aspects of prediction in audition: cortical and subcortical neural mechanisms. *Int. J. Psychophysiol.* 83:200–7

Shea-Brown R, Rinzel J, Rakitin BC, Malapani C. 2006. A firing rate model of Parkinsonian deficits in interval timing. *Exp. Brain Res.* 1070:189–201

Shi Z, Chen L, Müller HJ. 2010. Auditory temporal modulation of the visual Ternus effect: the influence of time interval. *Exp. Brain Res.* 203:723–35

Shi Z, Church RM, Meck WH. 2013a. Bayesian optimization of time perception. *Trends Cogn. Sci.* In press

Shi Z, Ganzenmüller S, Müller HJ. 2013b. Reducing bias in auditory duration reproduction by integrating the reproduced signal. *PLoS ONE* 8:e62065

Siegel JJ, Kalmbach B, Chitwood RA, Mauk MD. 2012. Persistent activity in a cortical-to-subcortical circuit: bridging the temporal gap in trace eyelid conditioning. *J. Neurophysiol.* 107:50–64

Smith JG, Harper DN, Gittings D, Abernethy D. 2007. The effects of Parkinson's disease on time estimation as a function of stimulus duration range and modality. *Brain Cogn.* 64:130–43

Smythies J, Edelstein L, Ramachandran V. 2012. Hypotheses relating to the function of the claustrum. *Front. Integr. Neurosci.* 6:53

Suddendorf T, Addis DR, Corballis MC. 2009. Mental time travel and the shaping of the human mind. *Philos. Trans. R. Soc. B* 364:1317–24

Teki S, Grube M, Griffiths TD. 2012. A unified model of time perception accounts for duration- based and beat-based timing mechanisms. *Front. Integr. Neurosci.* 5:90

Teki S, Grube M, Kumar S, Griffiths TD. 2011. Distinct neural substrates of duration-based and beat-based auditory timing. *J. Neurosci.* 31:3805–12

Tregellas JR, Davalos DB, Rojas DC. 2006. Effect of task difficulty on the functional anatomy of temporal processing. *NeuroImage* 32:307–15

Treisman M. 1963. Temporal discrimination and the indifference interval: implications for a model of the "internal clock." *Psychol. Monogr.* 77(13):1–31

Treisman M. 1984. Temporal rhythms and cerebral rhythms. *Ann. N.Y. Acad. Sci.* 423:542–65

Treisman M. 2013. The information-processing model of timing (Treisman, 1963): its sources and further history. *Timing Time Percept.* In press

Treisman M, Cook N, Naish PL, MacCrone JK. 1994. The internal clock: electroencephalographic evidence for oscillatory processes underlying time perception. *Q. J. Exp. Psychol. A* 47:241–89

Treisman M, Faulkner A, Naish PLN, Brogan D. 1990. The internal clock: evidence for a temporal oscillator underlying time perception with some estimates of its characteristic frequency. *Perception* 19:705–43

Tulving E. 2002. Chronesthesia: awareness of subjective time. In *Principles of Frontal Lobe Function*, ed. DT Stuss, RC Knight, pp. 311–25. New York: Oxford Univ. Press

Ullman MT. 2004. Contributions of memory circuits to language: the declarative/procedural model. *Cogn.* 92:231–70

Ullman MT, Pierpont EI. 2005. Specific language impairment is not specific to language: the procedural deficit hypothesis. *Cortex* 41:399–433

van Rijn H, Gu B-M, Meck WH. 2013. Dedicated clock/timing-circuit theories of interval timing. In *Neurobiology of Interval Timing*, ed. H Merchant, V de Lafuente. New York: Springer-Verlag. In press

van Rijn H, Kononowicz TW, Meck WH, Ng KK, Penney TB. 2011. Contingent negative variation and its relation to time estimation: a theoretical evaluation. *Front. Integr. Neurosci.* 5:91

Vatakis A, Allman MJ. 2013. *Time Distortions in Mind: Temporal Processing in Clinical Populations*. Leiden, The Neth.: Brill. In press

Vidalaki VN, Ho M-Y, Bradshaw CM, Szabadi E. 1999. Interval timing performance in temporal lobe epilepsy: differences between patients with left and right hemisphere foci. *Neuropsychologia* 37:1061–70

Walsh V. 2003a. A theory of magnitude: common cortical metrics of time, space and quantity. *Trends Cogn. Sci.* 7:483–88

Walsh V. 2003b. Time: the back-door of perception. *Trends Cogn. Sci.* 7:335–38

Wearden JH. 2001. Internal clocks and the representation of time. See Hoerl & McCormack 2001, pp. 37–58

Wearden JH. 2003. Applying the scalar timing model to human time psychology: progress and challenges. In *Time and Mind II: Information Processing Perspectives*, ed. E Helfrich, pp. 21–39. Germany: Hogrefe & Huber

Wearden JH. 2005. Origins and development of internal clock theories of psychological time. *Psychol. Fr.* 50:7–25

Wearden JH, Edwards H, Fakhri M, Percival A. 1998. Why "sounds are judged longer than light:" application of a model of the internal clock in humans. *Q. J. Exp. Psychol. Comp. Physiol. Psychol.* 51B:97–120

Wearden JH, Lejeune H. 2008. Scalar properties in human timing: conformity and violations. *Q. J. Exp. Psychol.* 61:569–87

Wearden JH, Philpott K, Win T. 1999. Speeding up and (. . .relatively. . .) slowing down an internal clock in humans. *Behav. Process.* 46:63–73

Wearden JH, Smith-Spark JH, Cousins R, Edelstyn NM, Cody FW, O'Boyle DJ. 2009. Effect of click trains on duration estimates by people with Parkinson's disease. *Q. J. Exp. Psychol.* 62:33–40

Wencil EB, Coslett HB, Aguirre GK, Chatterjee A. 2010. Carving the clock at its component joints: neural bases for interval timing. *J. Neurophysiol.* 104:160–68

Wiener M, Turkeltaub PE, Coslett HB. 2010a. Implicit timing activates the left inferior parietal cortex. *Neuropsychologia* 48:3967–71

Wiener M, Turkeltaub PE, Coslett HB. 2010b. The image of time: a voxel-wise meta-analysis. *NeuroImage* 49:1–15

Williamson LL, Cheng RK, Etchegaray M, Meck WH. 2008. "Speed" warps time: methamphetamine's interactive roles in drug abuse, habit formation, and the biological clocks of circadian and interval timing. *Curr. Drug Abuse Rev.* 1:203–12

Wittmann M. 2013. The inner sense of time: how the brain creates a representation of duration. *Nat. Rev. Neurosci.* 14:217–23

Wittmann M, Leland DS, Churan J, Paulus MP. 2007. Impaired time perception and motor timing in stimulant-dependent subjects. *Drug Alcohol Depend.* 90:183–92

Wittmann M, Paulus MP. 2008. Decision making, impulsivity and time perception. *Trends Cogn. Sci.* 12:7–12

Wittmann M, Simmons AN, Flagan T, Lane SD, Wackermann J, Paulus MP. 2011. Neural substrates of time perception and impulsivity. *Brain Res.* 1406:43–58

Yin B, Meck WH. 2013. Comparison of interval timing behaviour in mice following dorsal or ventral hippocampal lesions with mice having δ opioid receptor gene deletion. *Philos. Trans. R. Soc. B*. In press

Yin B, Troger AB. 2011. Exploring the 4th dimension: hippocampus, time, and memory revisited. *Front. Integr. Neurosci.* 5:36

Yin B, Yin HH, Meck WH. 2010. Dorsal hippocampal lesions lead to double leftward shifts of peak time in a bi-peak procedure in mice. *Soc. Neurosci. Abstr.* 98.20

Yumoto N, Lu X, Henry TR, Miyachi S, Nambu A, et al. 2011. A neural correlate of the processing of multi-second time intervals in primate prefrontal cortex. *PLoS ONE* 6(4):e19168

RELATED RESOURCES

Broadway JM, Engle RW. 2011. Individual differences in working memory capacity and temporal discrimination. *PLoS ONE* 6(10):e25422

Cui X. 2011. Hyperbolic discounting emerges from the scalar property of interval timing. *Front. Integr. Neurosci.* 5:24

Dyjas O, Bausenhart KM, Ulrich R. 2012. Trial-by-trial updating of an internal reference in discrimination tasks: evidence from effects of stimulus order and trial sequence. *Atten. Percept. Psychophys.* 74:1819–41

Filippopoulos PC, Hallworth P, Lee S, Wearden JH. 2013. Interference between auditory and visual duration judgements suggests a common code for time. *Psychol. Res.* In press

Gaudreault R, Fortin C. 2013. To count or not to count: the effect of instructions on expecting a break in timing. *Atten. Percept. Psychophys.* 75:588–602

Matthews WJ. 2013. How does sequence structure affect the judgment of time? Exploring a weighted sum of segments model. *Cogn. Psychol.* 66:259–82