

# Neuropsychological mechanisms of interval timing behavior

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## Summary

Interval timing in the seconds-to-minutes range is believed to underlie a variety of complex behaviors in humans and other animals. One of the more interesting problems in interval timing is trying to understand how the brain times events lasting for minutes with millisecond-based neural processes. Timing models proposing the use of coincidence-detection mechanisms (e.g., the detection of simultaneous activity across multiple neural inputs) appear to be the most compatible with known neural mechanisms. From an evolutionary perspective, coincidence detection of neuronal activity may be a fundamental mechanism of timing that is expressed across a wide variety of species. *BioEssays* 22:94–103, 2000. © 2000 John Wiley & Sons, Inc.

## Introduction

“Time flies when you’re having fun,” is an often quipped adage that demonstrates our sensitivity (or lack thereof) to the time course of events in our everyday lives. Indeed, research has shown that alterations in the perception of time in the seconds-to-minutes range, referred to as “interval timing,” are commonly induced by drugs, disease, signal modality, and context.<sup>(1–4)</sup> And yet, it is difficult to envision the extent to which timing plays a role in behavior. We believe that this difficulty results from the fact that all behaviors are expressed in time.

Nevertheless, we can identify a variety of human behaviors that may rely on a perception of time in the seconds-to-minutes range. Such behaviors range from perceiving a beat in a musical composition to returning to the stove just prior to the tea kettle whistling; from sensing when to begin a tennis swing based upon the estimation of the approaching ball’s speed to expecting a traffic light to change from red to green. Furthermore, interval timing is exhibited in a wide variety of vertebrates, including goldfish, starlings, rats, and humans.<sup>(5–7)</sup> For example, numerous experiments conducted in our laboratory show that rats and humans are

similarly sensitive to the passage of time. Data from typical timing experiments using the peak-interval procedure with humans and rats are presented in Figure 1A and C. (See legend to Fig. 1 for description of the peak-interval procedure).

As can be seen from Figure 1, interval-timing behavior has some characteristic properties that are consistent between these (and other) species, which is the first criterion for postulating the existence of a common interval timing mechanism. A number of timing models have been developed to explain these properties using psychological/information processing terminology, such as memory, attention, and similarity. Unfortunately, these terms do not yet have a neurobiological reality in terms of mechanisms at the level that neural systems operate. Furthermore, it is not clear that feasible neurobiological mechanisms could be proposed for some of these psychological models. The search for both the psychological and neurobiological mechanisms of interval timing is the goal of our research.

## *The scalar property*

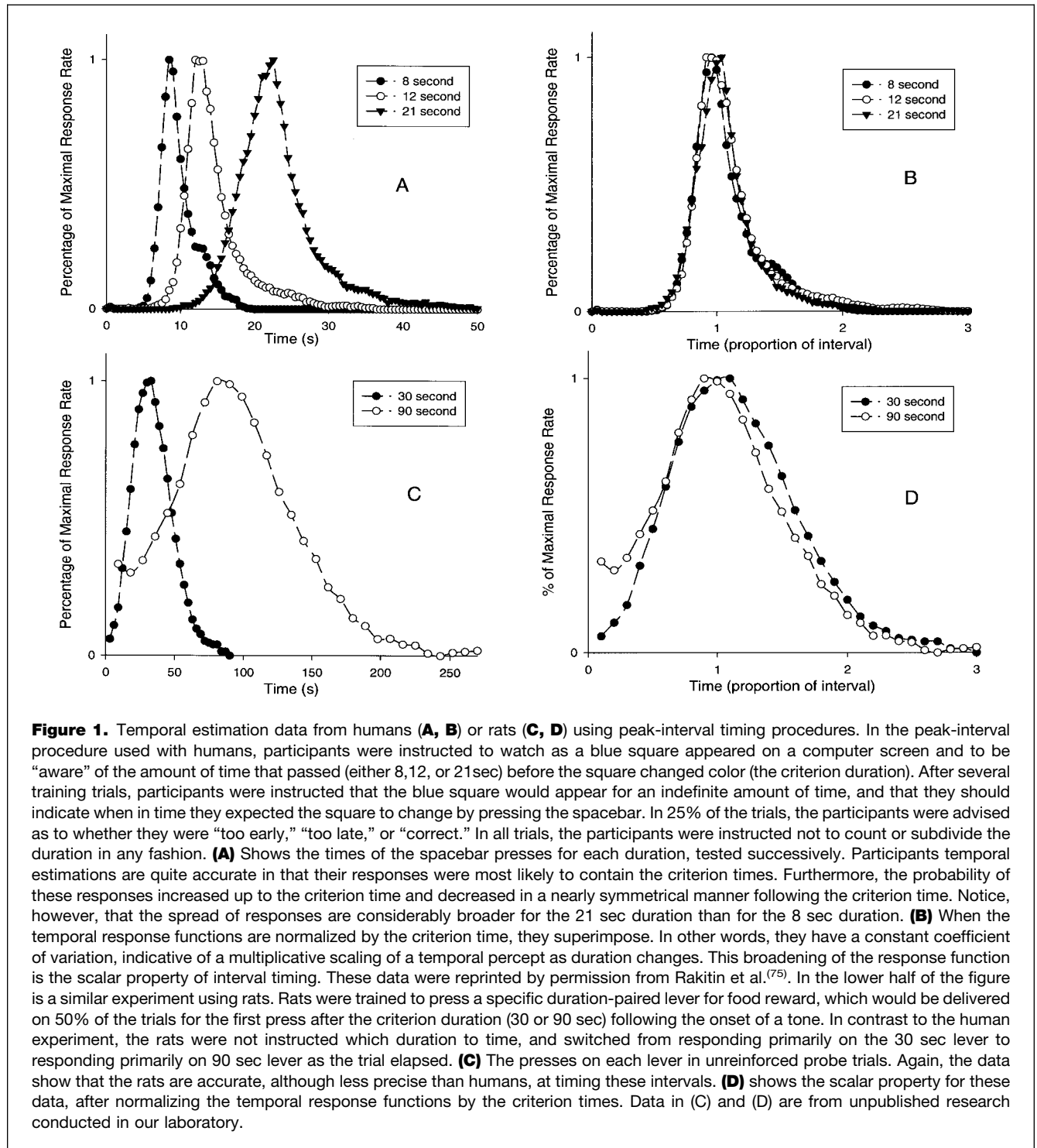
One of the fundamental properties of interval timing is that temporal variability in the behavioral output of an organism is proportional to the duration of the stimulus that the organism “times.” This characteristic behavioral manifestation, in which the standard deviation of the interval being timed grows proportionally to the mean of the interval, has been termed the scalar property.<sup>(8)</sup> The scalar property is graphically represented in Figure 1. This graph shows data from humans performing an experimental task designed to measure temporal discrimination. Both humans and rats are reasonably accurate in their timing as most respond around the criterion times displayed in Figure 1A and C. However, the spread of their responses ranges over a considerable amount of time. This variability in responding increases proportionally to the duration being timed (scalar property), such that they superimpose when the functions are normalized by their mean. This sort of superimposition of response functions for different time intervals is shown in Figure 1B and D.

The imprecision of interval timing differs markedly from the precision found in the circadian timing system. The circadian timing system, the 24 h clock guiding our sleep-wake cycle, temperature control, hormonal activity, etc., varies on a scale of only minutes in a 24 h cycle.<sup>(9)</sup> Fortunately, the cost of the

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imprecision found with the interval timer is offset by its flexibility. On a relative scale, organisms time equally well at 10 sec as they do at 90 sec. This relativity in interval timing is depicted in Figure 1B and D as the scalar property.

Furthermore, organisms are able to successfully start their interval “clocks” whenever an important stimulus is presented and even time multiple intervals concurrently.<sup>(10)</sup> This flexibility in when and what to time is another basic

property of interval-timing behavior. Again, this difference distinguishes interval timing from circadian timing, in that the latter takes days or weeks to readjust to a new starting point for the relevant time markers which, for example, is the cause of jet lag following a shift in dawn or dusk.

### *The utility of interval timing*

The interval timer has been proposed to form the basis of a variety of behaviors observed in the laboratory, such as associative learning, and in the field, such as rate estimation of prey capture.<sup>(11,12)</sup> Associative learning is loosely defined as the formation of an association between correlated events and is thought to underlie many behaviors of complex organisms. The most famous example of associative learning is based on evidence from Pavlov's dogs, when they made an association between the ringing of a bell (conditioned stimulus, CS) and the subsequent delivery of meat powder (unconditioned stimulus, US). This association was evidenced by the dogs salivating in response to the ringing of the bell (conditioned response, CR), a behavior previously elicited only by the delivery of the meat powder (unconditioned response, UR).<sup>(13)</sup>

Modern learning theorists agree that decreasing the amount of time between a CS (bell) and a US (meat) increases the rate of learning. However, explanations for why this phenomenon occurs differ. Many would argue that the memory strength of the CS decreases as absolute time increases, thereby lessening the strength of an association, and that a perception of time itself is unnecessary.<sup>(14)</sup> As an example, a tone followed 2 sec later by a food pellet will allow for more rapid conditioning than a tone followed 4 sec later by a food pellet. On the other hand, Gallistel and Gibbon<sup>(15)</sup> have recently proposed that the perception of time is fundamental for associative learning. Their hypothesis is that an organism forms an association between a CS and a US by computing a ratio comparison of the time between the CS and the US to the time between one US and a subsequent US. In other words, an organism's expectation that a lever press (A) will lead to reinforcement (B) is based upon whether the density of reinforcement is greater following a lever press (1/the interval from A to B) than it is globally (1/the interval from B to B). The logical prediction based on this hypothesis is that the rate of learning is controlled by the relative amounts of time between the CS and US and not the absolute amount of time between the CS and US. This prediction is indeed supported by experimental data.<sup>(15,16)</sup> In the example above, a 4 sec delay between a tone and food will actually condition just as fast as a 2 sec delay between tone and food, if the interval between trials (i.e., the food to tone interval) is also doubled.

A determination of the rate of reinforcement is another behavior that may be subserved by interval-timing mechanisms.<sup>(12,17,18)</sup> Such a determination could be made by di-

viding the amount of food an organism encounters by the duration in which the organism is in a particular context. This ability to determine rate and/or reinforcement density is fundamental to adaptive behavior, such as the determination of food patch density in foraging organisms. Another utilization of rate estimation is in interceptive timing, where judgments are made as to time of impact based upon rate of motion.<sup>(19)</sup> In many situations, such as deciding when and where to be to catch a ball, these interceptive judgments appear to be made with sub-second information. However, other similar judgments, such as whether one has enough time to cross the street without being hit by an oncoming car, might require longer, multiple second intervals to determine both speed and time to impact.

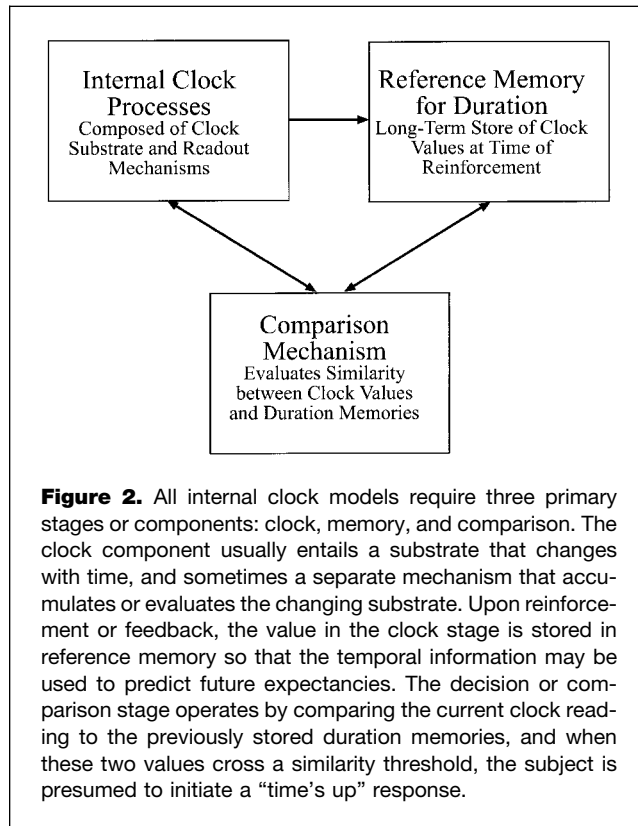
If interval timing does indeed form the basis for such a wide array of behaviors, understanding the underlying mechanisms of timing will help us understand and predict these complex behaviors. As previously mentioned, the development of interval-timing models has occurred almost entirely in the field of experimental psychology. As such, we will briefly describe the current state of affairs in this field before proposing some biological mechanisms for these models.

### **Psychological models of interval timing**

Although a variety of theories on the psychological components of interval timing have been proposed, it has been suggested that all internal-clock models must conform to a common basic structure,<sup>(20)</sup> in which there is a clock component, a memory component, and a decision/comparison component. In this basic "internal-clock" model shown in Figure 2, the clock component starts upon onset of a signal to be timed, and the output of the clock is compared via a decision mechanism to previously important duration codes held in reference memory.

The *clock component* is made up of a repeatable process that can be mapped onto time and is therefore expected to have an isomorphic mapping (e.g., each and every value of the clock is mapped to a specific duration of time).<sup>(21)</sup> However, recent evidence indicating nonlinearities in time perception<sup>(22,23)</sup> suggests that such an isomorphic mapping need not be the rule. It is important to note that the clock process is not necessarily periodic, only that it must proceed through a relatively repeatable pattern on each and every occasion it is started. The speed of this clock component is modifiable by drugs, disease, stimulus modality, as well as body temperature.<sup>(2,4,24,25)</sup> Attentional processes also can influence interval timing and might specifically impact the clock stage.<sup>(26)</sup>

The *memory component* is thought to be a long-term store of previously important, or reinforced, clock output values. The storage, maintenance, and retrieval of these temporal memories are also susceptible to a variety of influences, including



experimentally altered task constraints,<sup>(27–30)</sup> drug administration,<sup>(3,31,32)</sup> and pathologies such as Parkinson’s disease.<sup>(1,2)</sup>

The *decision or comparison component* is described as a mechanism that evaluates how well the current clock value matches previously stored temporal memories, and can be influenced by manipulations in task design.<sup>(33)</sup>

A variety of models of this basic “internal clock” exist, in which the formal descriptions of some or all of the components differ. These models can be divided into three classes, based primarily on their clock-stage mechanisms, termed pacemaker-accumulator models, process-decay models, and oscillator/coincidence-detection models. The six primary models of interval timing (two from each class), along with the difficulties in translating their psychological mechanisms into neurobiologically feasible physical mechanisms are summarized in Table 1.

The clock stage of the pacemaker-accumulator models is composed of a continuously running “pacemaker” and an “accumulator” that acts as a pulse counter. This accumulator, which is activated with the onset of the signal to be timed, “counts” the amount of pulses emanating from the pacemaker as the duration elapses. Conversely, the clock described by the process-decay models tracks the decay of neural activity following signal onset. Finally, the clock described by the oscillator/coincidence-detection models de-

fects a specific combination of periodic neural events, which are activated upon signal onset. Durations are encoded by associating this combinatorial activity code with a particular duration.

We will argue that the coincidence-detection models are the most feasible biologically, as these models’ mechanisms translate most easily into neural mechanisms. As such, one of these models, the beat-frequency model, will be explained in greater depth, and some neural underpinnings for this model will be proposed below. The differences in the various models culminate in whether the organism’s representation of time is expressed as a linear or logarithmic process.

### Neurobiological realization of an interval time keeper

In contrast to the extensive development of psychological models of interval timing, there has been relatively little investigation of the neurobiological mechanisms of time perception. Nevertheless, neuroanatomical and neuropharmacological mechanisms of interval timing are beginning to emerge (reviewed in Gibbon et al.<sup>(11)</sup> and Meck<sup>(34)</sup>). The basal ganglia, a set of subcortical brain nuclei traditionally considered important for motor functioning,<sup>(35–37)</sup> are now also thought to be involved in a variety of cognitive and motivational processes,<sup>(38–40)</sup> and appear to be critical for interval timing. Excitatory input from the cortex to the basal ganglia comes primarily into the striatum, the input nucleus of the basal ganglia. The striatum also receives modulatory dopaminergic input from the substantia nigra pars compacta (SNPC), a midbrain nucleus involved in reinforcement.

Lesions of the striatum or SNPC in the rat eliminate the ability to perform temporal discrimination tasks.<sup>(41,42)</sup> Unmedicated Parkinson’s patients, who have a deterioration of the dopaminergic neurons in the SNPC, also have difficulties in performing temporal discrimination tasks.<sup>(1,2)</sup> Furthermore, systemic modulation of the dopamine system using dopaminergic agonists or antagonists modulates the speed of the clock component.<sup>(3,34)</sup> More specifically, the degree to which the clock component is slowed down by dopaminergic antagonist administration correlates with the affinity of these drugs for the dopamine D<sub>2</sub> receptor.<sup>(43)</sup>

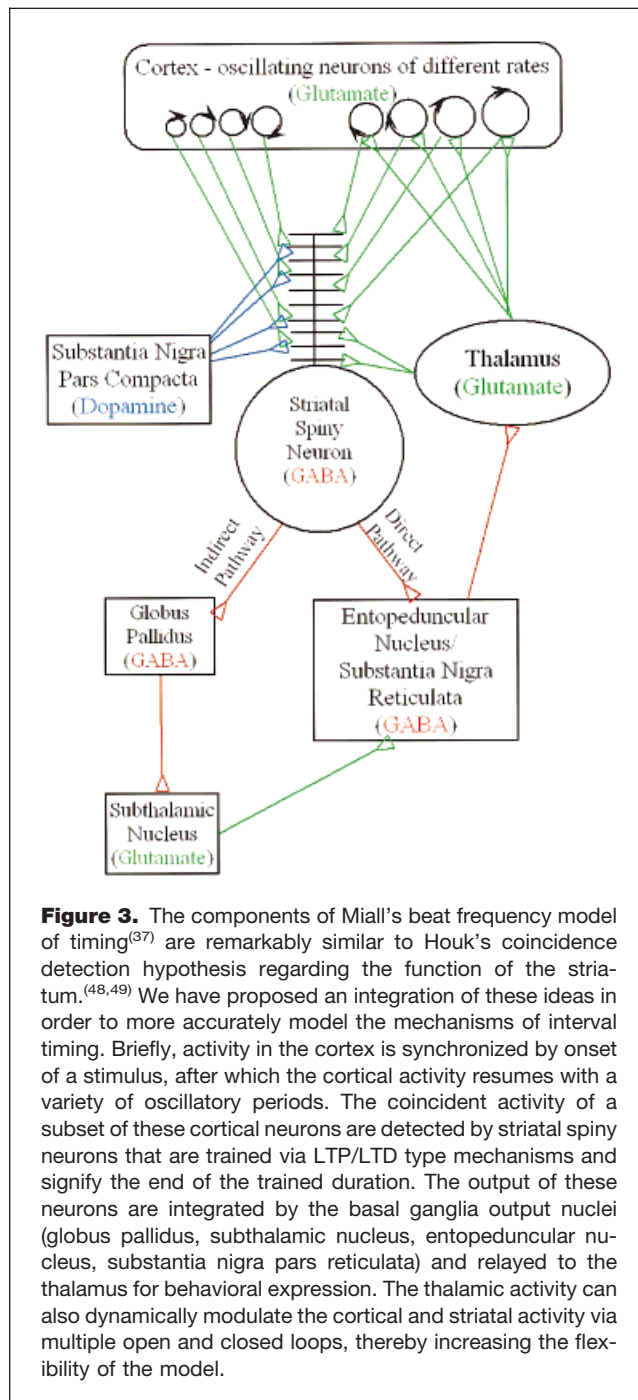
Finally, the cortex, which provides input to the striatum, and the thalamus, a brain relay nucleus that receives input from the basal ganglia and sends output back to the cortex, are also anatomical areas that influence timing behavior. fMRI and PET brain imaging techniques show that both of these areas are activated in humans during temporal discrimination tasks.<sup>(44,45)</sup> This anatomical arrangement produces a *cortico-striatal-thalamic-cortical* loop, an anatomical pathway that is proposed to underlie the necessary computations for the timing of behavior (Fig. 3).

**TABLE 1.** Models and Mechanisms of Interval Timing

Model name	Clock type	“Psychological” mechanism	Neurobiological feasibility/General criticisms
Scalar expectancy theory <sup>(8)</sup>	Pacemaker-accumulator	Continuously running pacemaker is gated into an accumulator upon signal onset. Value in accumulator is stored in reference memory. Comparison process utilizes a ratio comparison in order to achieve scalar variance.	Currently suffers from unbounded accumulation processes. Dopaminergic pacemaker hypothesis is unlikely due to limited range of modulation (~15%). May require chaining of pacemakers/accumulators.
Behavioral theory of timing <sup>(76)</sup>	Pacemaker-accumulator	Pacemaker is started upon signal onset that drives animals into different “behavioral” states with each pulse. Pacemaker speed is proportional to reinforcement density in order to achieve scalar property.	Reinforcement-density based pacemaker speed does not hold up to experimental findings. <sup>(77–79)</sup> Recent evidence has found successive states of striatal activity, which is likely gated by SNPC dopamine pulses, thus this could be a neural mechanism for this model.
Multiple Time Scales <sup>(80)</sup>	Process-decay	Decay of memory strength serves the role of the clock. Specific times are associated with specific amounts of decay. Scalar property is inherent in the “form” of the decay curve, as it approximates a negatively decelerating function.	Memory decay curves are computed over multiple trials in experimental situations, and it is unclear whether such activity could be found in the brain. Model has difficulty with gap and reset phenomenon.
Spectral timing model <sup>(81)</sup>	Process-decay	Differential activation rates of neurons lead to differential habituation rates. The combination of these rates leads to specific combinations of activity at different durations. Scalar property is achieved through habituation property specifying that rates of activation and habituation are roughly equivalent.	This type of neural activation/habituation phenomenon has not been found in regions of the brain thought to be involved in timing. Model has some difficulties with gap phenomenon.
Multiple oscillator model <sup>(82)</sup>	Oscillator/coincidence-detection	A large variety of oscillation periods are initiated at signal onset and time is coded by the combination of half-phase readouts across the ensemble of oscillators. Longer durations are primarily coded by longer oscillations, and scalar property is built into the oscillation periods.	Model requires oscillation periods of equal length to the duration being timed. The ability to find oscillations in the brain of sufficient duration (e.g., 90 secs) is questionable. However, recent evidence has found 60 sec oscillations in basal ganglia. <sup>(83)</sup>
Beat frequency model <sup>(57)</sup>	Oscillator/coincidence-detection	A variety of fast oscillation periods (~5–15 Hz) are initiated at signal onset and the time code consists of those neurons that fired spikes at the criterion time. This ensemble of coincidentally firing neurons produces maximal activity at the criterion duration, as well as a large degree of activity at the harmonics (1/2, 1/4, . . .) of the criterion duration, thereby inducing the scalar property	Model in its current form does not reproduce the data obtained in psychophysical timing experiments. However, the building blocks of the model are on the proper time scale of the brain (milliseconds), and therefore this model is easily adaptable to a neural implementation.

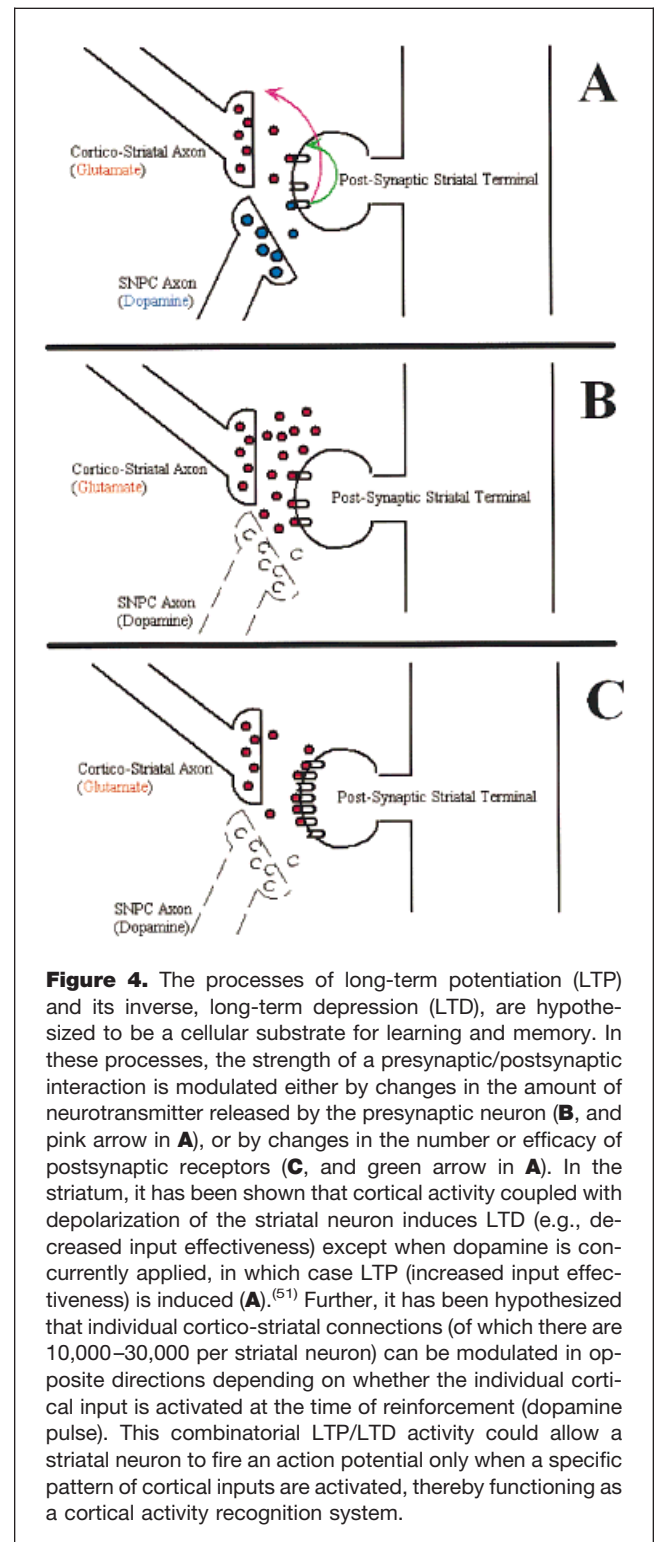
A variety of hypotheses based on anatomical and electrophysiological evidence suggest that the functional role of the striatum is to act as a “coincidence detector” for cortical and thalamic input.<sup>(46–48)</sup> Coincidence detection is a neural integration mechanism whereby single neurons are induced to fire as a result of receiving a large degree of simultaneous input (over a time range of ≈5–20 msec). This differs from the traditional “integrate and fire” processes of neurons, in which excitatory input is summated over a much longer time scale (≈20–100msec). Individual striatal spiny neurons have been estimated to have between 10,000–30,000 spines on their dendrites, which are areas specialized to receive input from other neurons. Each of these spines is believed to receive input from a different cortical or thalamic neuron.<sup>(49)</sup>

It has been proposed that the effectiveness of a particular input area is modulated by long-term potentiation (LTP) and long-term depression (LTD).<sup>(50)</sup> In these processes, the strength of a presynaptic/postsynaptic interaction is modulated either by changes in the amount of neurotransmitter released by the presynaptic neuron or by changes in the number or efficacy of postsynaptic receptors. In the striatum, cortical activity coupled with depolarization of the striatal neurons induces LTD (e.g., decreased input effectiveness) except when dopamine is applied concurrently, in which case LTP (increased input effectiveness) is induced. LTP and LTD are thought to serve as the cellular substrate for learning and memory by differentially weighting various inputs so that only specific patterns of input can induce the



integrating neuron to fire<sup>(51,52)</sup> (see Fig. 4 for a diagrammatic explanation of LTP/LTD).

A burst of dopaminergic activity from the SNPC is believed to induce LTP on cortico-striatal inputs.<sup>(53)</sup> Evidence from primates suggests that this dopaminergic input serves as a reinforcement signal for learning, by firing upon the delivery of reward.<sup>(54)</sup> In other words, particular spines will be



strengthened (and therefore be more effective at driving the neuron to fire) if a burst of dopamine occurs at the same time as wide scale cortical input. Thus, a striatal spiny neuron could be particularly effective as a coincidence detector by only firing an action potential when previously strengthened cortical inputs are active at the same time. This coincidence-detection hypothesis has been developed as a method for striatal neurons to detect specific contextual arrangements (including serial order) to which an organism should attend,<sup>(55)</sup> a phenomenon that has been shown to induce striatal activity in rodents.<sup>(56)</sup>

As described briefly above, the coincidence-detection models of timing propose that intervals are coded by evaluating the differential activity of various periodic neural events. From our perspective, because the striatum might function as a coincidence detector, it is the ideal substrate on which to map the beat-frequency model of interval timing (BF). BF proposes that the time at which a number of periodically firing neurons fire together (fire in coincidence) can be much greater than the periodicity of any of the neurons individually.<sup>(57)</sup> The ability to encode a duration that is longer than any of the individual neurons' periodicities is very similar to the determination of a least common multiple of a set of numbers (e.g., the LCM of 2, 3, 4, and 5 is 60, which is 12 times larger than the highest base number). By starting the periodic neurons together at signal onset and selectively weighting the neurons that fire together at a criterion duration, the maximal coincident activity of neural firing will occur at the same criterion duration on subsequent trials. Consequently, a criterion duration can be properly timed by waiting for this certain set of neurons to fire simultaneously.

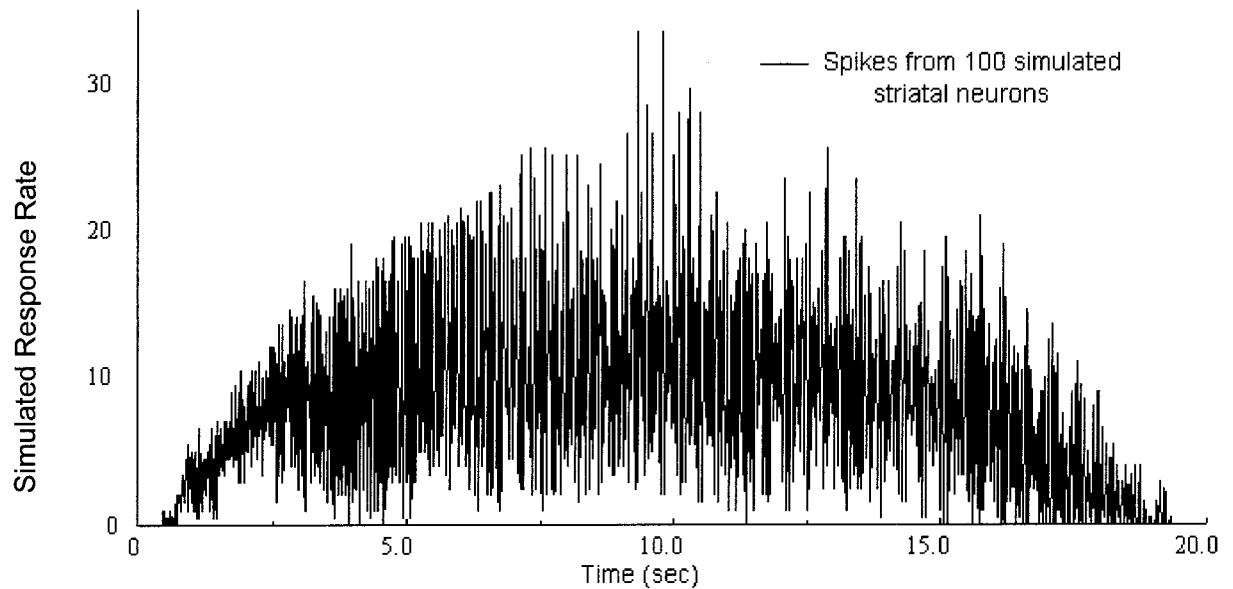
The scalar property in BF arises from the fact that high levels of coincident activity are found at the harmonics of the criterion duration. This means that the temporal pattern of activity is rescaled when different durations are timed (e.g., the maximal coincidence is always at the criterion duration, the secondary coincidence is always found at half the criterion duration, and the tertiary coincidence is always found at one-quarter of the criterion duration).

However, despite the ingenuity of this model, the pattern of coincident activation over time does not qualitatively match data from interval-timing experiments. Nonetheless, due to the similarity between BF's mechanism for coding time and the function hypothesized for the striatum (e.g., coincidence detection), we have modified the original model using the neurophysiological constraints of the cortico-striatal-thalamic loop to see if it can simulate data from psychophysical timing studies.<sup>(58)</sup> The revised model, the striatal beat frequency model (SBF), proposes that the striatal spiny neuron serves as the coincidence detector and the cortical input to the striatum serves as the oscillating input of the original BF.

One important neurophysiological constraint of the striatal spiny neuron is that it has a bistable membrane potential.<sup>(59,60)</sup> This bistability allows the neuron to be either in a constrictive down-state (a membrane state where the neuron does not fire an action potential,  $\approx -90$  mV), or in a potentiated up-state (a membrane state where a very minor change in input can induce an action potential,  $\approx -60$  mV). The switch from the down-state to the up-state is induced by a set amount of coincident input activity. Furthermore, once the neuron is switched into the up-state, the level of coincident activity necessary to keep it at that membrane potential is significantly reduced. This neurobiological constraint of the striatal spiny neuron is an important factor contributing to the temporal activity pattern produced in simulations of SBF.

Furthermore, as in all biological systems, variance is a necessity. Therefore, we have added variance to the oscillatory input speed for the ensemble of periodically firing cortical neurons between and/or within trials, variance to the precision of periodicity, and variance to the firing threshold. Simulations performed in our laboratory have shown that introducing these biological constraints produce results that qualitatively match psychophysical timing data. The results of the above manipulations in a 100-trial simulation of SBF is shown in Figure 5.<sup>(58)</sup>

One limitation of SBF is that certain properties dictated by the model have not yet been described in the brain while an organism times. Specifically, the model proposes that the oscillating cortical or thalamic cells can be induced to fire (their oscillations can be reset) at the onset of a stimulus to be timed. The model also requires that the coincidence detecting cells of the striatum are also able to be "reset" upon onset of the same stimulus. A potential solution to the former problem comes from work dealing with event related potentials (ERPs). ERPs are large changes in the electrical potential at fixed delays ( $\approx 300$  msec) following stimulus presentation during EEG recordings (electrical activity of the brain measured at the scalp). It is believed that these time-locked changes in the EEG signal are the result of simultaneous activity across many neurons, a phenomenon that would be congruent with a cortical "clock" reset. Furthermore, researchers investigating the use of coincidence-detection mechanisms and cell assemblies in neural coding<sup>(61)</sup> and feature binding<sup>(62)</sup> have reported the existence of oscillating cortical neurons. A solution to the latter problem (re-setting the striatal cells) comes from recent evidence showing that the burst of dopaminergic SNPC activity occurring during reinforcement (described above) is transferred to the earliest stimulus that predicts reinforcement.<sup>(63)</sup> Thus the SNPC dopamine pulse essentially serves as a preparatory signal. This dopaminergic pulse could function to "clear out" irrelevant or random activity by hyperpolarizing the striatal cell following the presentation of a timing signal. As such, it



**Figure 5.** This simulation of 100 striatal neurons shows a clear increase and decrease in activity as the time approaches the trained criterion duration of 10 sec. The peaks of activity at the harmonics of the duration (e.g., at 2.5, 5, 7.5, . . . sec) that occur in the beat frequency model have been eliminated in the current model by incorporating biological constraints, such as variance within and between trials in both oscillation speed and spike threshold, as well as by instituting a dynamic bistable membrane potential in the striatal neuron.

could be conceived to function as a perceptual “starting gun.” Indeed, recent evidence from our laboratory has shown that the delivery of food reward, which serves as a pre-potent stimulus for the induction of dopamine release from the SNPC, resets the interval timer.<sup>(64)</sup>

To reiterate, SBF proposes that striatal spiny neurons can detect the occurrence of coincident oscillating cortical input, and that the maximal coincident activity will occur at previously learned, biologically relevant, durations. By incorporating the neurophysiological constraints of the brain areas believed to be involved in timing, simulations of SBF have been able to qualitatively reproduce psychophysical data from interval-timing tasks. This model, therefore, marks an important step forward, by forging an integration between the known neurobiology and the known behavioral response functions.

#### **Coincidence detection as a mechanism for general timing**

Taken together, the anatomical and physiological evidence for a striatal coincidence detector coupled with evidence for cortico-striatal-thalamic loops in interval timing suggests that coincidence detection processes are a forerunner in the search for the mechanisms of interval-timing processes. The striatal spiny neurons, as well as the architecture of the basal

ganglia have been well conserved evolutionarily.<sup>(65)</sup> Similarly, the ability to behave according to the temporal constraints of the environment, as well as the scalar form of such behavior, is also found in a diverse array of vertebrates.<sup>(5–7)</sup> These similarities in both the psychophysical expression of interval timing and the functional architecture of the cortico-striatal-thalamic loop, suggest that there may be a common species-independent mechanism for such an internal clock.

The idea that coincidence-detection mechanisms may be used for interval timing leads us to suspect that similar mechanisms might be envisioned to be the basis for recognition and control across multiple time scales. Evidence for this hypothesis abounds. There are many examples of the use of neuronal delay lines (delays induced by differential axonal conduction speed or length) coupled with coincidence-detection mechanisms to determine extremely short temporal estimates such as auditory localization in barn owls,<sup>(66)</sup> echolocation in bats,<sup>(67)</sup> and electric organ transmission in a variety of fish species (duration estimates which are on the order of nanoseconds).<sup>(68)</sup> Slightly longer temporal-control processes functioning in the milliseconds range, such as precise motor control, have been associated with cerebellar functioning,<sup>(69,70)</sup> a brain nucleus which contains the only neurons with a greater number of inputs ( $\approx 200,000$ ) than those of the striatum.<sup>(71)</sup> Indeed, some researchers



have suggested that there is a breakdown of temporal estimation in the seconds-to-minutes range (interval timing) and the milliseconds range (millisecond timing), and have suggested the basal ganglia and cerebellum as the neural locales, respectively.<sup>(72)</sup> A recent hypothesis has even suggested that coincidence-detection mechanisms in the rhythmic pyloric network of the lobster (*Panulirus interruptus*) could conceivably function as temporal-pattern detectors.<sup>(73)</sup> Finally, coincidence-detection mechanisms may function to decrease the variability of circadian oscillation periods seen in highly variable individual cells<sup>(74)</sup> to the tight precision of an organism's behavior.

### Conclusion

The ability to perceive and behave according to the temporal constraints of the environment may be a fundamental requirement for successful behavior. Temporal perception can be a subserving factor in many complex behaviors, and a wide variety of organisms behave in a temporally controlled manner. Although a variety of psychological theories have been developed that accurately predict the scalar property, most are difficult to translate into biologically plausible mechanisms. Those theories that appear most biologically plausible propose that the detection of coincident neural activity encodes the duration of events. We have begun to adapt a coincidence-detection model of timing using the neurobiological constraints of an interval-timing circuit that successfully reproduces the known psychophysical timing data. Finally, we propose that coincidence-detection mechanisms are highly conducive to timing and time perception across a wide range of intervals and species.

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