Time-scale invariance as an emergent property in a perceptron with realistic, noisy neurons

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ABSTRACT

In most species, interval timing is time-scale invariant: errors in time estimation scale up linearly with the estimated duration. In mammals, time-scale invariance is ubiquitous over behavioral, lesion, and pharmacological manipulations. For example, dopaminergic drugs induce an immediate, whereas cholinergic drugs induce a gradual, *scalar* change in timing. Behavioral theories posit that time-scale invariance derives from particular computations, rules, or coding schemes. In contrast, we discuss a simple neural circuit, the perceptron, whose output neurons fire in a clockwise fashion based on the pattern of coincidental activation of its input neurons. We show numerically that time-scale invariance emerges spontaneously in a perceptron with realistic neurons, in the presence of noise. Under the assumption that dopaminergic drugs modulate the firing of input neurons, and that cholinergic drugs modulate the memory representation of the criterion time, we show that a perceptron with realistic neurons reproduces the pharmacological clock and memory patterns, and their time-scale invariance, in the presence of noise. These results suggest that rather than being a signature of higher order cognitive processes or specific computations related to timing, time-scale invariance may spontaneously emerge in a massively connected brain from the intrinsic noise of neurons and circuits, thus providing the simplest explanation for the ubiquity of scale invariance of interval timing.

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1. Time-scale invariance is ubiquitous in many species

Interval timing is the ability of perceiving and using the passage of time in the seconds-to-minutes range. Interval timing is essential for psychological processes critical for survival and adaptation, such as decision making and rate estimation, and its impairment leads to severe cognitive and motor dysfunctions (Buhusi and Meck, 2005; Gallistel, 1990). In most species interval timing is not only *accurate* but also *time-scale invariant*, or simply *scalar*, in that *the errors in time estimation scale-up with the estimated duration* (Gibbon, 1977; Gibbon et al., 1984; Meck et al., 2008). For example, the left panel of Fig. 1A shows that when timing different durations (2 s, 4 s, 6 s, and 8 s), human adults distribute their responses quasi-Gaussian around the target durations, indicating that they acquired an accurate representation of the target intervals (Wearden et al., 1997). Most important for this paper, the width of these distributions

scale-up with the estimated duration, such that when the timing functions are displayed in time units relative to the timed criterion, as shown in the right panel of Fig. 1A, the timing functions *superimpose*. In other words, interval timing is increasingly less precise as the interval being timed lengthens, i.e., it is *time-scale invariant*.

The time-scale invariance of interval timing is ubiquitous in many species from invertebrates such as bees (Boisvert and Sherry, 2006), to many vertebrates, such as fish (Talton et al., 1999), birds (Cheng and Westwood, 1993), and mammals such as rats (Dews, 1962), mice (Buhusi et al., 2009) and humans (Rakitin et al., 1998). For example, in humans, time-scale invariance can be observed throughout development, as shown in Fig. 1B: in similarity to human adults (Fig. 1A), the responses of children trained to time multiple durations follow a quasi-Gaussian distribution around the target durations (here, 4 s and 8 s), and their response functions superimpose when displayed in time units relative to the target interval (Droit-Volet et al., 2001). Moreover, the linear relationship between estimation error and estimated duration does not depend on the modality of the timed stimulus (Fig. 1C, Zarco et al., 2009), on whether human participants time implicitly or explicitly (Fig. 1D, Piras and Coull, 2011), on sensorimotor processes involved, or on the number of intervals timed (Fig. 1E, Merchant et al., 2008).

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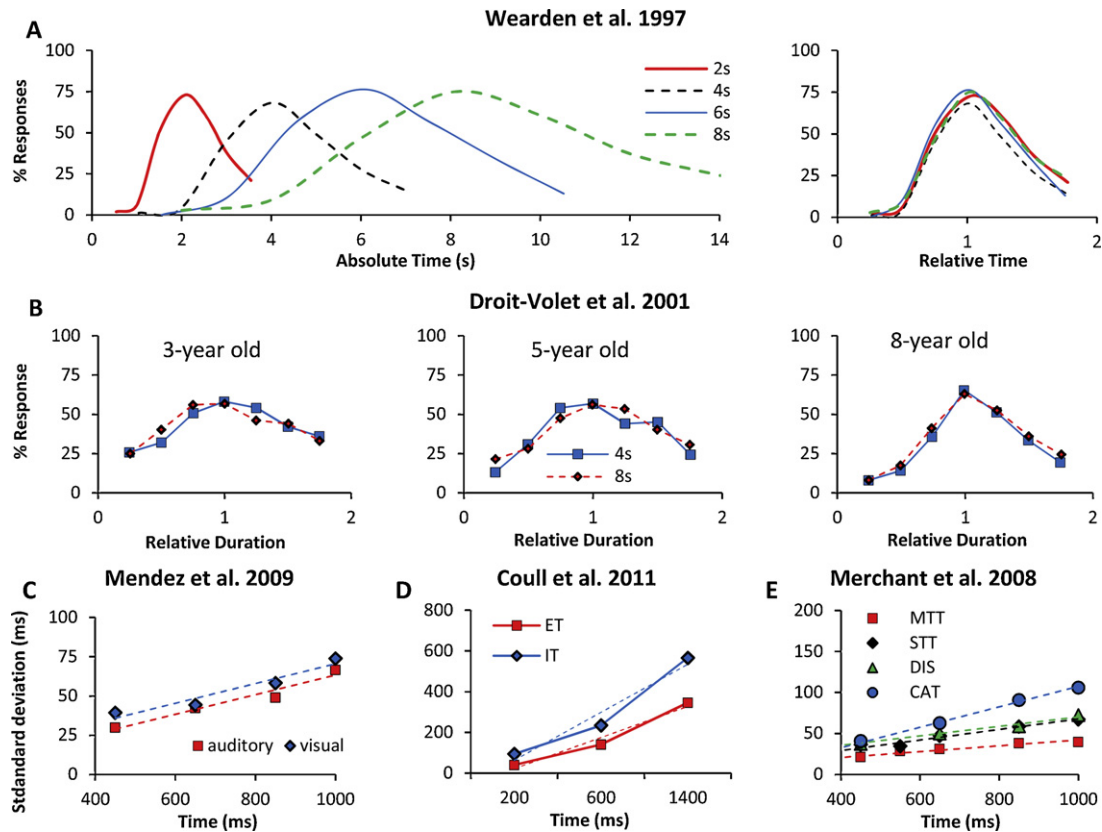
Fig. 1. The ubiquity of scale-invariant timing in humans. (A) In human adults, interval timing functions peak at the target duration, and their width increases with duration (left), such that they superimpose in relative time units, indicating scale-invariant timing (right) (adapted from Wearden et al., 1997). (B) Scale-invariant timing is ubiquitous throughout human development, in 3-year old (left), 5-year old (center), and 8-year old children (right) (adapted from Droit-Volet et al., 2001). (C) Scale-invariance of timing does not depend on the modality of the timed stimulus (adapted from Zarco et al., 2009). (D) Scale-invariant timing is ubiquitous in both explicit (generalization) and implicit (estimation) timing tasks (adapted from Piras and Coull, 2011). (E) Scale-invariant timing is ubiquitous in timing tasks with different motor requirements, and number of to-be-timed signals: production of single (STT) or multiple time intervals (MTT), temporal categorization (CAT) and discrimination (DIS, adapted from Merchant et al., 2008).

2. Time-scale invariance is resilient to behavioral, sensorimotor, and pharmacological manipulations

In similarity to human adults (Fig. 2A and B, Rakitin et al., 1998), rodents trained in a peak-interval procedure produce quasi-Gaussian distributions of responses around the criterion durations, whose width scale up with the estimated intervals (Fig. 2C, Matell et al., 2004). In similarity to humans and rodents, estimation errors increase linearly with estimated duration in Rhesus monkeys (Fig. 2D, Zarco et al., 2009). Interestingly, time-scale invariance is particular to timing in the seconds-to-minutes range, but not to circadian timing, which is far more accurate than interval timing, and whose estimation errors increase very little with the mean of the interval (Gibbon, 1977; Hinton and Meck, 1997).

The ubiquity of scalar timing extends over behavioral, lesion (Meck et al., 1987), and pharmacological manipulations of interval timing (Buhusi and Meck, 2010). Considerable progress has been made in recent years toward elucidating the neural bases of time perception in the seconds-to-minutes range (Buhusi and Meck, 2005; Mauk and Buonomano, 2004; Meck et al., 2008). Recent studies consistently point toward the cortico-striatal circuits as being critical for interval timing both in animals (Matell and Meck, 2000; Matell et al., 2003; Meck, 2006) and humans (Coull et al., 2011, 2004; Stevens et al., 2007). These studies also consistently indicate that the dopaminergic system is crucial for interval timing, and for its time-scale invariance. For example, severe deficiencies in reproducing temporal intervals were found in various neuropsychiatric disorders, such as Parkinson's (Harrington and Haaland, 1991), characterized by loss of dopaminergic cells. With few notable exceptions (Malapani et al., 2002, 1998), time-scale invariance is resilient to behavioral, lesion and pharmacological manipulations.

For example, acute administration of dopaminergic (DA) agonists, such as cocaine, result in a characteristic immediate leftward shift of response functions (Fig. 2C), consistent with the speeding up of an internal clock (Matell et al., 2004). At the same dose, cocaine speeds up timing of a 90 s interval three times more than when timing a 30 s interval (Fig. 2C), suggesting that the effect of the drug is proportional – scalar – to the timed interval. On the other hand, pharmacological manipulations (Meck, 1983, 1996; Meck and Church, 1987a,b) and lesions (Meck et al., 1987) aimed at the cholinergic (ACh) systems produce gradual (rather than immediate) effects on the memory storage. Importantly, time-scale invariance of both the *clock pattern* of DA drugs and the *memory pattern* of ACh drugs is not restricted to the drug state, but that the linear relationship between estimation error and estimated duration extends over the estimated durations and drugs states (vehicle versus drug) (Fig. 2C) (Matell et al., 2004).

How does time-scale invariance, including the scalar effect of DA and ACh drugs, emerge from unreliable, noisy neurons firing in the milliseconds range? Here we discuss a biologically plausible neural circuit, the *perceptron*, in which large numbers of inputs activate a relatively small number of output neurons. Due to its simple yet general architecture, the perceptron has long been considered a suitable model for information storage and organization in the brain (Rosenblatt, 1958). Here we discuss the biological support for the perceptron architecture in interval timing, and we show computer simulations suggesting that in a perceptron, time scale