Timing Patterns in the Extended Basal Ganglia System

Michael Schwartze and Sonja A. Kotz

Abstract

The human brain is a constructive organ. It generates predictions to modulate its functioning and continuously adapts to a dynamic environment. Increasingly, the temporal dimension of motor and non-motor behaviour is recognised as a key component of this predictive bias. Nevertheless, the intricate interplay of the neural mechanisms that encode, decode and evaluate temporal information to give rise to a sense of time and control over sensorimotor timing remains largely elusive. Among several brain systems, the basal ganglia have been consistently linked to interval- and beat-based timing operations. Considering the tight embedding of the basal ganglia into multiple complex neurofunctional networks, it is clear that they have to interact with other proximate and distal brain systems. While the primary target of basal ganglia output is the thalamus, many regions connect to the striatum of the basal ganglia, their main input relay. This establishes widespread connectivity, forming the basis for first- and second-order interactions with other systems implicated in timing such as the cerebellum and supplementary motor areas. However, next to this structural interconnectivity, additional functions need to be considered to better understand their contribution to temporally predictive adaptation. To this end, we develop the concept of *interval-based patterning*, conceived as a temporally explicit hierarchical sequencing operation that underlies motor and non-motor behaviour as a common interpretation of basal ganglia function.

Keywords

Temporal processing · Interval timing · Patterning · Chunking · Temporal prediction

All state changes in an individual and in the environment generate events that are defined by their type (‘what’) and timing (‘when’). To successfully interact with an ever-changing environment, individuals continuously adapt to these what and when dimensions of events (Schwartze & Kotz, 2013). In this fundamental action, humans not only react to but also predict events (Friston, 2009; Friston et al., 2006). This essentially affords optimised allocation of neural and cogni-

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tive resources and timely action, e.g. in the case of an athlete commencing a sprint with the last sound of a starting signal, or a musician falling in with an orchestra on a specific note. The underlying principle is simple and thus readily transferable between different contexts: knowing when something happens affords better cognitive and behavioural adaptation to what happens.

The efficiency of this temporally predictive adaptation partially depends on the capacity to exploit 'when', i.e. timing information, to tune into the dynamic environment. Models of 'predictive coding' (Friston, 2009; Friston et al., 2006) suggest that this capacity involves a fundamental division of labour, in which slower neural dynamics inform and interact with faster neural dynamics to guide adaptation (Friston, 2012; Schwartze et al., 2012a). However, unlike 'what' information, which can be manifold (e.g. touch, movement, colour, words, musical tones), 'when' information is strictly one-dimensional and thus solely defined by the interplay of change (event) and persistence (interval; Fig. 1). Consequently, other timing-related characteristics such as regularity, periodicity, but also grouping, are ultimately temporal ordering principles that specify the configuration of change and persistence. However, although these components of timing are well-defined and quantifiable in physics, it has long been recognised that they are much less so for perception and neurocognitive functions, and thus require a distinct taxonomy of temporal experience to capture the mechanisms underlying temporally predictive adaptation in humans (Jones, 1976; Pöppel, 1978).

Neurocognitive processes add further subjective components to temporally predictive adaptation. A well-established example is the phenomenon of 'subjective accentuation', i.e. the emergence of a perceptual dissociation of 'strong' and 'weak' events when listening to a train of physically identical stimuli such as metronome clicks that commonly leads to the 'tick-tock' illusion (Brochard et al., 2003; Abecasis et al., 2005; Criscuolo et al., 2023). Physical and neurocognitive timing are therefore not necessarily equivalent in how temporal ordering principles are realised. However, both, either independent or in combination, can lead to perceived temporal regularity, which, in turn, seems a prerequisite for efficient temporally predictive adaptation. Perceived temporal regularity essentially corresponds to the recognition of a 'pattern *of* time', which also may or may not constitute a 'pattern *in* time' (Handel, 1974). Although the ability to produce, perceive and synchronise movement

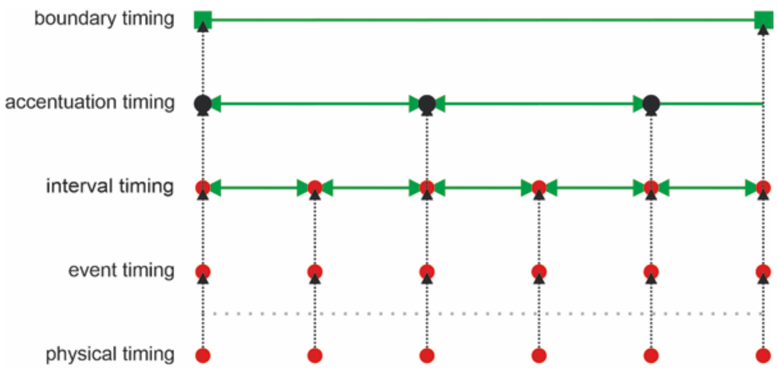

Fig. 1 Patterns of time. The interplay of change and persistence (e.g. the clicks of a metronome) generates successive events that constitute an environmental timing pattern (physical timing: red circles). Neural encoding generates representations of the temporal locus of events (event timing) and inter-event relations (interval timing: green lines). Additional mechanisms and functions can lead to subjective accentuation of some events (accentuation timing: black circles) and to distinct markings of the beginning and the end of a pattern (boundary timing: green squares)

with patterns of time is increasingly recognised as a fundamental of neurocognitive function (Merchant et al., 2015; Penhune & Zatorre, 2019), the dissociation of physical and neurocognitive timing warrants further differentiation of their basic constituents.

Perceived temporal regularity and grouping imply at least two intervals (and correspondingly three events). As indicated, additional mechanisms and functional components can lead to subjective accentuation over the course of a longer sequence but also mark boundaries, i.e. the beginning and the end of a pattern such as that established by an action sequence (Fujii & Graybiel, 2003; Graybiel, 2008). The resultant accentuation timing and boundary timing (Fig. 1) are essentially chunking phenomena that may relate to specific cognitive constructs and constraints, e.g. the dynamic allocation of attention and working memory capacities (Jones, 1976; Large & Jones, 1999; Schwartze et al., 2020). However, these examples illustrate why next to a taxonomy of temporal experience (Pöppel, 1978), additional mechanistic and functional components have to be considered to obtain a better understanding of temporally predictive adaptation as a form of interaction of the organism with a pattern structure (Jones, 1976).

Eventually, these additional subjective components are anchored in the brain and different, partly interdependent, neural mechanisms have been associated with the precise encoding of when an event occurs and the encoding of the respective inter-event relations or intervals that form the basic constituents of any pattern of time (Ivry & Schlerf, 2008; Spencer & Ivry, 2013; Buhusi & Meck, 2005; Bares et al., 2019).

Next to multiple other functions, chunking and interval timing specifically engage the subcortical basal ganglia system, which, in turn, interfaces with other cortical and subcortical systems. Together, these systems form a large-scale network that seems to support interleaved and differential aspects of adaptive timing (Buhusi & Meck, 2005; Ivry & Schlerf, 2008; Merchant et al., 2013; Petter et al., 2016). This interactive network architecture may not only explain phenomena such as subjective accentuation in timing but also how timing factors into other well-established basal ganglia functions, e.g. in relating an event to a subsequent reward (Schultz, 2004) or in triggering sequential behaviour (Graybiel, 2008).

The basal ganglia system and associated thalamocortical circuits link to prefrontal and supplementary motor cortices and the cerebellum. However, much less is known about if and how this core timing network interfaces with further systems and whether such interaction guides temporally predictive adaptation. One example of a direct functional interaction between several systems is the initiation and termination of the basal ganglia interval timing mechanism through dopaminergic bursts emitted by the ventral tegmental area (VTA; Buhusi & Meck, 2005; Petter et al., 2016). However, when considering such interactions between systems, critical questions arise such as how sensory input triggers these bursts in the first place, and how they respond to repeating events that delineate the consecutive intervals of a pattern. To answer these questions, it seems necessary to take a more holistic perspective on timing that considers further second-order interactions and information flow from the earliest stages of sensory processing.

Previous work developed such a holistic perspective into an integrative timing framework (Schwartze & Kotz, 2013). A central tenet of this framework is the adaptive cerebellar filtering of sensory input into an event-based stimulus representation and its rapid transmission to the thalamus as a means to translate physical timing into an intermittent neural ‘clock’ signal (Fig. 2).

Following the cerebellar encoding of event timing, the clock signal is transmitted to cortical targets that include the supplementary motor area (SMA), where successive events trigger continuous oscillatory activity at different frequencies. The thalamus relays and amplifies the signal by employing a bursting firing mode (Sherman, 2001). As originally suggested by the striatal beat frequency model of interval timing, a snapshot of the oscillatory activity at the end of an interval is integrated by the basal ganglia into a distinct code of an inter-event interval (‘timestamp’; Matell et al., 2003; Matell & Meck, 2004; Buhusi & Meck, 2005) and relayed to the cortex. However, one may speculate that the intermittent

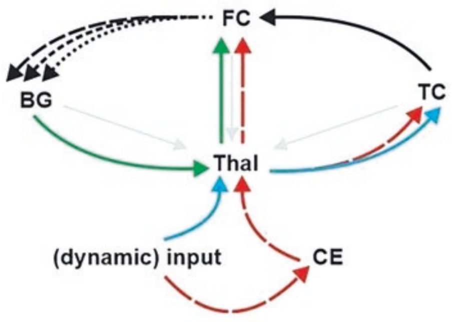

Fig. 2 A timing network. Dynamic input (here exemplified by a sound signal) is differentially encoded along ascending connections that include a direct bottom-up route via the cerebellum (CE). The CE performs temporally precise sparse coding of events (e.g. onsets, offsets, abrupt changes in energy) of the input into an intermittent (dashed red) 'clock signal' transmitted via the thalamus (THAL) to frontal cortices (FC). Thalamic signals trigger cortical oscillatory activity. A snapshot of the concerted cortical oscillatory activity (dashed black) is integrated by the basal ganglia (BG) into a relational (interval) representation of the time between events. Successive events end the current and open the next interval. Interval representations are transmitted to frontal cortices (green) for timing judgments and pattern recognition. Parallel to the intermittent clock signal, the ascending auditory pathway transmits a detailed continuous input representation. This signal reaches sensory cortices (here temporal cortex, TC) to interface with memory representations (blue) that are then conveyed to frontal areas to bridge input elements (e.g. successive words forming utterances, tones forming melodies). The thalamus also receives modulatory input from other structures, potentially allowing top-down modulation of functioning and reactivity (grey) to predictively adapt the organism to the environment

cerebellar clock signal and entirely subcortical pathways are not only instrumental in triggering thalamic bursting but also the dopaminergic bursting that opens and ends the integration of cortical oscillatory activity by the basal ganglia (Petter et al., 2016).

Among the cortical targets of the cerebellar clock signal the medial frontal SMA insofar plays a special role as it tightly connects to the basal ganglia as well as to prefrontal, premotor and primary motor cortices. Already in his original definition of the SMA, Wilder Penfield (Penfield, 1950) provided evidence that electrostimulation of this area just anterior of the sensorimotor cortex produces varied motor responses that include vocalisations. Most of the responses could be categorised as either slow and sustained (i.e. persistent) or rhythmic (i.e. patterned). SMA activity in monkeys has been shown to indicate total elapsed time as well as rhythm intervals (Cadena-Valencia et al., 2018). The SMA is furthermore one of the only brain systems that is consistently recruited in perceptual and motor timing tasks (Wiener et al., 2010). However, this general SMA engagement across different contexts and pattern levels also illustrates that in addition to a holistic perspective, further structural and functional differentiation of the systems that make up the timing network is critical to better understand temporally predictive adaptation.

The general importance of the SMA for timing is supported by its anatomical differentiation into the more anterior pre-SMA and the more posterior SMA-proper (Picard & Strick, 2001). Connectivity between the SMA and the striatum of the basal ganglia maintains this anterior-posterior organisation, while distinct cortical connections link the pre-SMA to prefrontal cortices and the SMA-proper to premotor and primary motor cortices (Picard & Strick, 2001; Lehéricy et al., 2004; Akkal et al., 2007; Kotz et al., 2013). This organisation is likely part and parcel of functional separations, with single and longer interval timing hosted by the pre-SMA and multiple interval timing by the SMA-proper (Schwartze et al., 2012b; Cona et al., 2021). Alternatively, it may reflect a shifting functional gradient that indicates the initial recruitment of single-interval pre-SMA timing in a cyclic manner, and an associated activation of the SMA-proper with an evolving pattern of time. In both cases, it seems likely that information flow through this anterior-posterior system would be modulated by factors such as interval duration or tasks demands (Coull et al., 2004; Macar et al., 2006), reflecting the distinct prefrontal and premotor connectivity patterns of pre-SMA and SMA-proper in interaction with cognitive processes such as attentional resource allocation, working memory, or movement control.

These interactive dynamics and functional gradients furthermore allow considering whether the same or at least similar concepts and mechanisms that have been identified in one field of research can be applied to another field of inquiry to derive refined and testable hypotheses. This rationale can be exemplified by SMA to basal ganglia connectivity and by linking basal ganglia functions that are expressed on a timescale that spans days or even years to the milliseconds-to-seconds range that is typically relevant for sensorimotor timing research. Accordingly, the previously documented role of the SMA in establishing a temporal link between action and effect (Moore et al., 2010) may differentially engage interval timing in a context-dependent manner that reflects interval duration or single interval as opposed to multiple interval patterns and corresponding recruitment of anterior-posterior SMA to basal ganglia connections. Similarly, the formation and expression of 'habits', both associated with the basal ganglia system (Graybiel, 2008; Smith & Graybiel, 2016), may engage interval timing in a cyclic manner (Fig. 3). According to Graybiel (2008), habits are largely defined by five central characteristics: they are learned, occur repeatedly, are performed almost automatically, manifest as a triggered action or thought sequence and can be completed without constant oversight. Structural and functional differentiation of the SMA and connections to the basal ganglia as identified for timing may factor into these characteristics, forming an explicitly temporal component of the general motor and cognitive patterning and chunking mechanisms that have been ascribed to the basal ganglia system (Graybiel, 1997, 1998).

Next to refined conceptions in several domains, this integrative perspective may also help to better explain a range of pathological phenomena. For example, insufficient differentiation along the structural and functional gradients of the timing network may diffuse production and perception of patterns of time, while compensatory strategies may target such insufficient differentiation. This may be the case in stuttering, a disorder that affects the temporal pattern of speech sequences. However, delayed auditory feedback can typically improve speech fluency dramatically in people who stutter. This effect may indicate a lower degree of automaticity or that speakers are prevented from hearing their own errors, reducing or prohibiting erroneous basal ganglia activity (Alm, 2004; Guenther & Hickok, 2016). Alternatively, the higher degree of temporal differentiation introduced by the

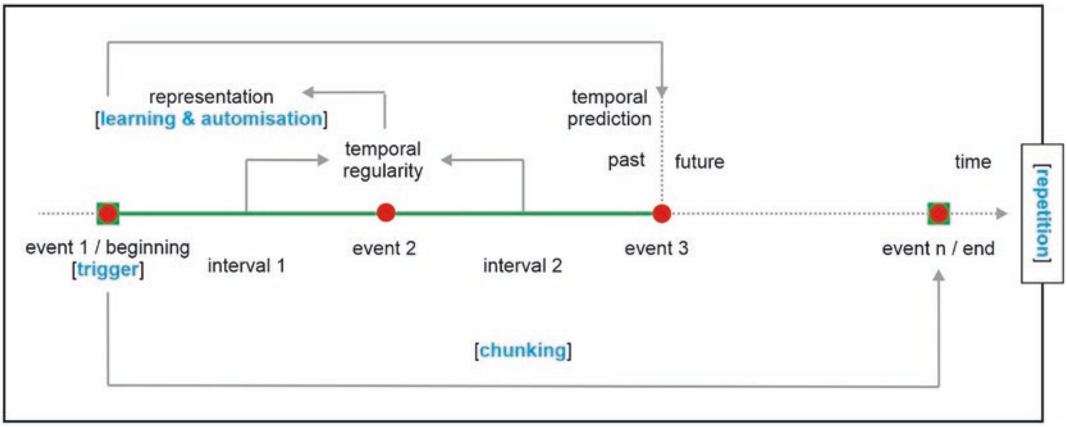

Fig. 3 Interval timing and habitual behaviour. The interval timing capacity of the basal ganglia system is initiated via a dopaminergic burst with the first event (event1/beginning) of sequential behaviour. Subsequent events close and open successive intervals until the final event ends the sequence (event n/end). The temporal regularity extracted from a pattern (through representation and learning) drives temporally adaptive behaviour in prediction of future events, e.g. by allocating attention to times at which events are predicted to occur. In turn, this facilitates automisation of sequential behaviour, e.g. by reducing overall attentional demands for rigid and habitual forms of behaviour. Subjective accentuation through 'boundary timing' facilitates the chunking of the sequence and specifies an interval that corresponds to the duration of the entire sequence

delay may improve a speaker's sense of agency and the differentiation of perceptual and sensorimotor timing as linked to the interplay of the SMA and basal ganglia. This could potentially explain why not only the iterative build-up of a pattern of time during speech production is affected but also that people who stutter can show subtle sensorimotor and sensory as well as nonverbal timing dysfunctions (Etchell et al., 2014; Falk et al., 2015; Schwartze et al., 2020).

Dysfunctional timing is also a hallmark of Parkinson's disease. Although Parkinson's disease is not a unitary pathology and comprises various forms and subtypes, the two most commonly associated structural and functional features are the well-documented cell loss in the substantia nigra pars compacta of the basal ganglia system and cardinal motor symptoms (Weingarten et al., 2015). However, Parkinson's disease is a progressive disease that affects multiple brain systems outside the basal ganglia, and also leads to non-motor symptoms that can precede motor symptoms in early non-medicated patients (Pont-Sunyer et al., 2015). Dysfunctional timing may manifest in disturbances of gait fluency, problems to produce and to maintain a steady movement pace, or an impaired ability to synchronise with pacing stimuli (Allman & Meck, 2012; Dalla Bella et al., 2015). However, it is currently largely unclear at which point in the progression of the neurodegenerative process timing is affected, suggesting that more subtle timing dysfunctions manifest before or after other typical symptoms. Especially if one considers the expression of a pattern of time as the outcome of general operations that define not only the order of successive motor but also of non-motor events and cognitive behaviour such as complex problem solving (Graybiel, 2008), it would be meaningful to explore the timing aspect of existing diagnostic tools more systematically. Dysfunctional timing may already be present and indicative of pre-diagnostic disease mechanisms due to the engagement of the basal ganglia, associated pathways and secondary regions in the selection and sequencing of most forms of motor and non-motor behaviour (Graybiel, 2008).

The notion of aberrant timing as an early marker of PD is further supported by neuroimaging studies that show dynamic patterns of SMA and cerebellar hyper- and hypo-activity that may indicate compensatory mechanisms during early disease stages (Kotz & Schwartze, 2011; Schwartze & Kotz, 2016). Complementing such findings, post-mortem studies of PD patients indicate a selective loss of pyramidal neurons in the SMA and suggest that this loss may precede basal ganglia pathology (McDonald & Halliday, 2002).

Although certainly selective, these examples clearly illustrate that the human capacity for temporally predictive adaptation to the environment is a fundamental but complex phenomenon. A better understanding of this capacity requires combining a taxonomy of temporal experience with a holistic neurofunctional perspective that explains how the human organism encodes and decodes patterns of time. One important open question concerns the dimensionality of facilitatory compared to pathological manifestations of temporal adaptation. Although temporally predictive adaptation necessarily involves an aspect of temporal regularity, overreliance on regularity might mark inflexible, habitual behaviour.

The main argument put forward here is that decomposition of basal ganglia function and their interaction with other brain systems in timing can be used to derive refined explanations and testable assumptions across different contexts. This approach has not least a number of methodological implications, as the parameters that define temporal experience and subjective components of temporally predictive adaptation impose several constraints for the design of respective empirical investigations, e.g. in terms of interval durations, stimulus rates, grouping, regularity, or jitter that specify the interplay of change and persistence.

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