Conduction velocity of intracortical axons in monkey primary visual cortex grows with distance: implications for computation

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1   Abstract

A critical visual computation is to construct global scene properties from activities of early visual cortical neurons which have small receptive fields. Such a computation is enabled by contextual influences, through which a neuron's response to visual inputs is influenced by contextual inputs outside its classical receptive fields. Accordingly, neurons can signal global properties including visual saliencies and figure-ground relationships. Many believe that intracortical axons conduct signals too slowly to bring the contextual information from receptive fields of other neurons. A popular opinion is that much of the contextual influences arise from feedback from higher visual areas whose neurons have larger receptive fields. This paper re-examines pre-existing data to reveal these unexpected findings: the conduction speed of V1 intracortical axons increases approximately linearly with the conduction distance, and is sufficiently high for conveying the contextual influences. Recognizing the importance of intracortical contribution to critical visual computations should enable fresh progress in answering long-standing questions.

2   Introduction

Receptive fields of primate V1 neurons are small. In monkeys, their diameters are typically less than one degree in visual angle for parafoveal receptive fields, or less than two degrees for most V1 receptive fields (Hubel and Wiesel, 1968; Gattass et al., 1987). However, a V1 neuron's response to inputs within its receptive field can be influenced by contextual inputs which are up to several degrees away from this classical receptive field (Knierim and Van Essen, 1992; Sceniak et al., 1999; Angelucci and Bressloff, 2006). Such contextual influences enable V1 neurons to carry out important computations such as signal-

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ing visual saliency (Li, 2002), which is a global scene property that depends on contextual inputs outside the receptive field.

For example, a vertical bar is more salient in a contextual background of horizontal bars rather than vertical bars, because it evokes a higher V1 neural response in the horizontal context than in the vertical context. This higher response in the horizontal context is due to iso-orientation suppression, so that the contextual or surround suppression on the neural response is stronger when the orientation of the contextual bars is close to the orientation of the bar within the receptive field (Knierim and Van Essen, 1992). Indeed, when a monkey is searching for a uniquely oriented bar in a background of uniformly oriented bars, a faster onset of a saccade towards the target bar is typically preceded by a higher V1 neural response to this target bar (Yan et al., 2018).

Contextual influences are also observed in V2. For example, when a foreground figure surface occludes a background object surface, and when a V2 neuron's receptive field covers a small segment of the occluding border between the foreground and background surfaces, this neuron's response often depends on whether the foreground figure is at one or the other side of the border. In other words, this neuron's response level can depend on contextual input information that is entirely outside its receptive field, and it conveys the information regarding which surface owns the border (Zhou et al., 2000). Such V2 signals for border ownership serve figure-ground computation for object segmentation (von der Heydt and Zhang, 2018).

To enable the contextual influences, information about the contextual inputs outside the receptive field of a neuron must be transmitted to the neuron to impact its response. An important question is whether this transmission is through intracortical axons linking neurons with non-overlapping receptive fields or through top-down feedback axons from higher visual cortical areas. A neuron in a higher visual cortical area typically has a larger receptive field, which could cover visual locations inside and outside the receptive field of a neuron in a lower visual cortical area. Hence a feedback axon from this higher cortical neuron to the lower cortical neuron could convey the contextual information.

For a neuron in V1, when visual inputs inside and outside its receptive field appear simultaneously, the influence of the contextual inputs on its neural response starts at a latency of 10–20 milliseconds

(ms) after the start of the of the response to the inputs within the classical receptive field (Knierim and Van Essen, 1992). By comparing the latencies between influences from near versus far contextual inputs in monkeys, Bair et al. (2003) estimated that the contextual influences effectively propagate across the distances on the cortical surface at a speed of around one meter/second (m/s). For V2 neurons, the latencies of the contextual influences appear insensitive to the distance of the context (Zhou et al., 2000; von der Heydt and Zhang, 2018).

Meanwhile, previous works have concluded that the intracortical axons conduct at a speeds of 0.1–0.4 m/s, slower than the estimated speeds of around 3 m/s (or 2–6 m/s) by the inter-areal axons (Grinvald et al., 1994; Bringuier et al., 1999; Girard et al., 2001; Angelucci and Bressloff, 2006). Consequently, many believe that conduction velocities of intracortical axons are too slow to bring contextual information in time. A popular opinion is that the contextual influences, particularly influences from contexts further away from the receptive field, arise from feedback from higher visual areas (von der Heydt and Zhang, 2018; Angelucci and Bressloff, 2006).

However, many previous estimates of the conduction speeds by intracortical axons are underestimates. Specifically, this conduction speed should be computed from the propagation latency, i.e., the difference between the time of a spike at one location of an axon and the time of this spike at another location of this axon as the result of the spike propagation along this axon. Many (Grinvald et al., 1994; Bringuier et al., 1999) have estimated this latency from latencies of neural responses to contextual visual inputs at various distances from the receptive fields (Nowak and Bullier, 1997). However, these measured latencies include not only the time needed for signal propagation along the axons, but also the integration time, i.e., time needed (typically several milliseconds or longer) to integrate the inputs to charge up the membrane potentials of the post-synaptic neurons towards neural activation. This integration time applies not only to the post-synaptic neuron, but also be the neurons in the intervening neural circuit between the visual inputs and the recorded neural response. Hence, this approach overestimates the latency, leading to an underestimation of the conduction speed.

An accurate way to estimate the latency is by the latency of an antidromic spike relative to the time of an electrical stimulation at or near the axon of the same cell. The stimulation evokes a spike

on the part of the axon very near (within $\sim 15\mu m$) the tip of the stimulating electrode (Nowak and Bullier, 1998a,b; Histed et al., 2009; Bakkum et al., 2013), and this spike propagates along the axon and is recorded by the recording electrode somewhere downstream from the stimulating electrode. The technique of a collision test verifies that the recorded spike is the result of an antidromic propagation of the original, stimulation-evoked, spike towards the cell body without any intervening synapses (Nowak and Bullier, 1997). However, this requires that the stimulating and the recording electrodes are very near the axon of the same neuron. This is easier to achieve for intercortical axons between V1 and V2 by placing the two electrodes at retinotopically corresponding cortical locations. This is very difficult to achieve for intracortical axons extending horizontally within V1, without adequate guidance for placing the two electrodes near an axon of the same cell. Girard et al. (2001) was able to obtain the antidromic spike across many pairs of electrodes between V1 and V2, but could only obtain one such pair for intracortical axons.

For V1's horizontal axons, the best data so far for estimating the conduction speed are from the latencies by orthodromic propagation of action potentials recorded by Girard et al. (2001). Without a collision test, selecting recorded signals through the shape of the waveforms, and by considering only latencies that had a temporal jitter within the range 0.3–0.5 ms (Girard et al., 2001; Gold et al., 2006; Bakkum et al., 2013), the recorded spike was most likely post-synaptic to the axons of the stimulation-evoked spikes. The range of the temporal jitter would be shorter if the recorded spike and the directly evoked spike by the electrical stimulation were on the same axon without an intervening synapse, and the jitter would be longer when a polysynaptic route is involved (Bullier and Henry, 1980; Bullier et al., 1988; Nowak and Bullier, 1997; Bakkum et al., 2013). Meanwhile, the electrical stimulation, a current pulse of 0.7 mA lasting for 0.2 millisecond (ms) (Girard et al., 2001), was many times the threshold level needed for evoking axonal spikes (the threshold is around $10\mu A$ or less at similar or shorter pulse durations (Butovas and Schwarz, 2003; Histed et al., 2009)). It should directly evoke axonal spikes in many neurons, such that numerous excitatory postsynaptic potentials (EPSPs) can converge nearly synchronously at the post-synaptic neuron near the recording electrode, and the time needed between the arrival of afferent spikes and the triggering of a spike in the post-synaptic neuron could be only a fraction of a millisecond with a small jitter (< 0.5 ms) (Singer et al., 1975; Bullier and Henry, 1979; Nowak and Bullier, 1997; Xu-Friedman

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and Regehr, 2005). In other words, using orthodromic spikes from artificial, supra-threshold electric stimulations, rather than neural responses to more naturistic visual input stimuli, the over-estimation of the latency by Girard et al. (2001) could be limited to a fraction of a millisecond.

For each of the 156 pairs of stimulating and recording sites in V1 of three monkeys, Girard et al. (2001) measured the latency l of the orthodromic spike relative to the stimulation, the distance d between the two electrodes, and obtained an estimation of the conduction speed (or velocity) of the intracortical axons as $v = d/l$. They reported a median conduction speed of 0.33 m/s using these orthodromic spikes. This median speed value for V1's intracortical conductions is often quoted or used for analysis and arguments by previous works, for example, by Angelucci et al. (2002), Bair et al. (2003), Carrasco et al. (2003), Angelucci and Bressloff (2006), Craft et al. (2007), Jehee et al. (2007), Von der Heydt (2015), Muller et al. (2018), Nurminen et al. (2018), and Franken and Reynolds (2021). However, Girard et al. (2001) did not analyze the relationship between the latency l and the distance d, and did not explicitly report the distances d across their data sample. This paper re-analyzes these data kindly provided by Pascal Girard, and highlights this relationship between the latency l and the distance d to discover the following unexpected finding: the conduction speed v grows approximately linearly with the axonal conduction distance d. Accordingly, it further reveals that longer intracortical axons have their conduction speeds comparable to that in intercortical axons which transmit feedforward and feedback signals between different visual cortical areas. Consequently, it argues that these intracortical axons are sufficiently fast to be a main player in mediating influences from near and far context.

3 Methods

Girard et al. (2001) had 156 pairs of stimulating and recording sites in V1 of three monkeys. Electrical stimulation was by a 75-μm-tip tungsten microelectrodes assembled in a triple- or double-electrode assembly. The tips delimited an equilateral triangle of 1.2–1.5 mm side. They used cathodic current impulses, monophasic and unipolar, usually less than 1 mA (median, 0.7 mA) and with a duration of 0.2 ms. The recording electrode was a tungsten microelectrode with a tip about 10 μm and recorded single unit spikes. The latency l was defined as the time between the beginning of the electric stimulation artifact and the foot

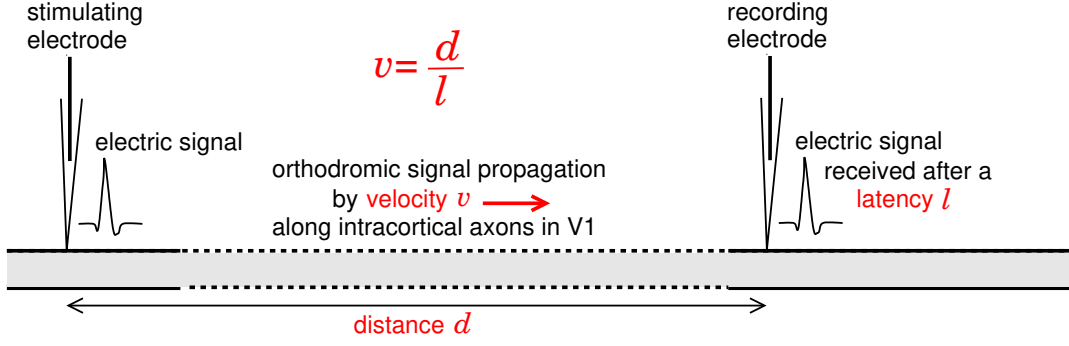**Measuring latency l and distance d to get velocity v of orthodromic signal propagation along axons in V1**

stimulating electrode

recording electrode

$$v = \frac{d}{l}$$

electric signal

orthodromic signal propagation by velocity v $\longrightarrow$ along intracortical axons in V1

electric signal received after a latency l

distance d

Figure 1: The method to investigate the intracortical orthodromic conduction in V1 used in Girard et al. (2001). The conduction latency l and distance d were measured to obtain a conduction speed $v = d/l$.

of the spike. The jitter in l was 0.3–0.5 ms. Using the waveform shape of the recorded spikes, they rejected spikes from passing-by axons so that the recorded spikes were most likely from a neuron postsynaptic to the axons in which original spikes were evoked by the electrical stimulation.

The conduction speed for the orthodromic propagation along the V1 axon for each pair of electrodes is calculated as $v = d/l$ (Fig. 1). Girard et al. (2001) found that the median and mean of the conduction speeds were 0.33 m/s and 0.6 m/s, respectively (Fig. 2B). More details of the methods of the experiments were described in Girard et al. (2001).

The matlab function "fit" was used for a linear fit between the latency l and the distance d, and also for a linear fit between the velocity v and the distance d. The slopes of the linear fits, as well as the 95% confidence intervals of the slopes, were obtained.

4   Results

4.1   The conduction speed in V1 axons increase with the propagation distance d

Fig. 2A shows a histogram of the 156 latencies l. This histogram is the same as the bar histogram for horizontal V1 axons in Figure 3 of Girard et al. (2001) from their orthodromic activation data. The median latency was $l = 4.0$ ms (Fig. 2A). Our analysis revealed that the 156 samples were dominantly those with short inter-electrode distances $d < 2$ millimeters (mm). Over-sampling of short d's is unsurprising, since, given a stimulating electrode, a recording electrode at a shorter distance away is more likely to pick up the
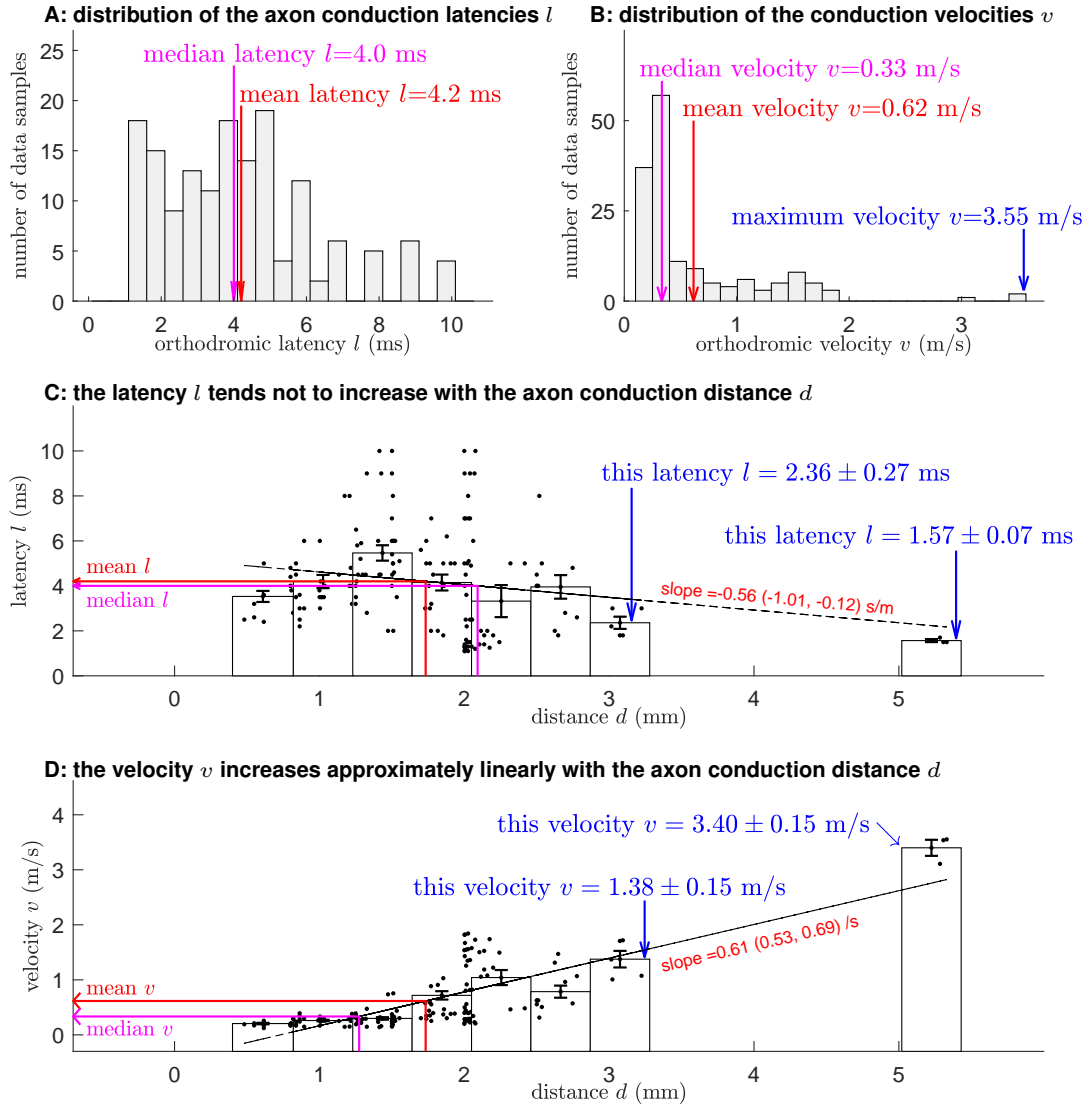
Figure 2: Intracortical orthodromic conduction speed increases with axon length in V1 from the re-analysis of data from Girard et al. (2001). A and B: distributions of the 156 recordings of orthodromic propagation latencies l and velocities v between the stimulating and recording electrodes in V1. C: l versus the distance d between the stimulating and recording electrodes. Each dot is one of the 156 recordings. Each bar is the mean l across recordings in a range of d specified by the borders of the bar. Error bars are the standard errors of the means. The dashed line (with a slope and, in brackets, its 95% confidence interval) is a linear fit of the data. D: like C, but for $v = d/l$. C and D share the same data bins according to d. Across all data samples, the median and mean velocities are dominated by short d samples.

signal propagating down the axons. However, if the conduction speed v depends on d, then the average v is biased by those of the short d samples. Re-examining the data indeed found that v increases about linearly with d, such that for $d > 5$ mm, the speed $v \geqslant 3$ m/s!

A linear fit of l versus distance d gives a negative slope -0.56 s/m (Fig. 2C). The 95% confidence

interval of this slope is $(-1.01, -0.12)$ s/m, entirely within the negative range (Fig. 2C). If anything, the average latency l decreased with distance d! Among the three longest distance $d > 5$ mm samples, the mean latency was $l = 1.57 \pm 0.07$ ms. The mean latency for $d \approx 3$ mm samples was $l = 2.36 \pm 0.27$ ms. A linear fit of conduction velocity v versus d (Fig. 2D) gives a positive slope 0.61/s, with a 95% confidence interval $(0.53, 0.69)$/s, consistent with the idea that v increases with d. Removal of the three $d > 5$ mm samples gives the confidence interval $(0.4, 0.6)$/s, which robustly remains in the positive range. Indeed, for the $d \approx 3$ mm samples, the velocity was $v = 1.38 \pm 0.15$ m/s, four times as large as the median $v = 0.33$ m/s. Among our three $d > 5$ mm samples, the average $v = 3.40 \pm 0.15$ m/s is similar to the speed of feedforward and feedback connections between V1 and V2 (Girard et al., 2001). Among all the 156 v's (Fig.2BD), the median and mean v were indeed dominated by the small $d < 2$ mm samples.

4.2 Considerations of possible factors contributing to the recorded data and the estimated speed

We note that our estimated velocity v may also be underestimates. If an axon has a trajectory with several changes of directions, rather than straight, between the stimulating and recording electrodes, our distance d would be shorter than the actual axon length, making our estimated v smaller than the actual conduction velocity. Hence, the intracortical V1 axons could conduct at a faster speed than what we report here, this would only make our conclusion stronger.

The recorded spike from a neuron is most likely the result of many input spikes propagated by many different axons, and these input spikes converge almost synchronously onto the postsynaptic neuron being recorded. The temporal jitter, 0.3-0.5 ms, in the latency of the recorded, post-synaptic, spike manifests the random (although nearly synchronous) temporal arrival times of the many input spikes (Xu-Friedman and Regehr, 2005).

We can ask whether it is likely that the recorded spikes involved a V1-V2-V1 route of signal propagation from the electrical stimulation sites via the feedforward and feedback axons between V1 and V2. For example, the electrical stimulation could evoke spikes in the feedforward axons towards V2, leading to postsynaptic V2 spikes which propagate orthodromically back to V1 before activating V1 neurons

whose spikes could then be recorded (since spikes from passing-by axons were rejected (Girard et al., 2001)). Another possibility could be that the electrically evoked spikes in V1 could propagate to V2 antidromically through feedback axons, invading the soma of V2 neurons to cause somatic spikes, after an antidromic-to-orthodromic delay (of about 0.18 ms, with a very small temporal jitter of 0.05 ms (Schmitz et al., 2001)), these spikes could then propagate orthodromically towards V1 via feedback fibers to activate post-synaptic V1 neurons. Both possibilities should lead to a larger temporal jitter in the latency of the recorded V1 spike, compared to the temporal jitter if intracortical propagation was involved instead. This is because the temporal jitter of the recorded neural spike could be sufficiently small only when the pre-synaptic inputs are sufficiently numerous and synchronous (Xu-Friedman and Regehr, 2005). Since V1 neurons receive about 10 times as many intracortical inputs as feedback inputs from V2 (Markov et al., 2011; Siu et al., 2021), if the recorded spike were caused by signal propagation in feedforward and/or feedback axons (rather than intracortical axons), the number of pre-synaptic inputs would be much smaller so that a much larger temporal jitter would arise in the recorded latency. Furthermore, when the V1-V2-V1 route involved an orthodromic rather than an antidromic route to V2, this route would be disynaptic from the stimulation to the recorded spike, making the temporal jitter even larger than that (0.3–0.5 ms, consistent with a monosynaptic pathway) recorded in the data. This is because each synapse gives additional temporal jitter through stochastic neurotransmitter releases. Hence this orthodromic V1-V2-V1 route is even less likely than the antidromic V1-V2-V1 route. In any case, considering that the temporal jitter of the latency is 0.3–0.5 ms in our data, it is quite unlikely that they involved feedforward and feedback axons between V1 and V2.

The V1-V2-V1 route would be even less likely for our data samples with distance $d > 3$ mm, since these data samples have short latencies $l \leqslant 3$ ms, or even $l \approx 1.5$ ms for distance $d \geqslant 5$ mm (See Fig. 2C). Using antidromic spikes verified by a collision test, Girard et al. (2001) showed that the latencies by the intercortical conduction on the feedforward and feedback axons (without involving any synapses) between V1 and V2 have a median of about 1.25 ms (see Figure 1 of (Girard et al., 2001) for a distribution of these latencies). Hence, the round-trip conduction latency, involving both the feedforward and feedback latencies, should have a median value around 2.5 ms. In addition, the probability p that the one-way latency

was less than 1 ms was $p \approx 0$ for the feedforward axons and $p \approx 0.04$ for the feedback axons, so that the round-trip latency has only a probability $p \approx 0.02$ to be $\leqslant 1.5$ ms. Furthermore, with $d > 3$ mm, neurons near the stimulating electrode were unlikely to have overlapping receptive fields with neurons near the recording electrode. Since the feedforward and feedback connections are more likely to link retinotopically corresponding neurons across different cortical areas, the number of pre-synaptic inputs from V2 to converge onto the recorded V1 neuron via the V1-V2-V1 route should be further reduced, making it even less likely that the temporal jitter of the recorded latency to be as small as 0.3–0.5 ms in our data. Therefore, we conclude that the data samples with $d \geqslant 3$ mm are much less likely than the data samples with $d \leqslant 2$ mm to involve the feedforward and feedback axons.

Among the data samples with $d \leqslant 2$ mm, many have latencies $l > 3$ ms, making it more difficult to rule out the possibility of a V1-V2-V1 route by the l value alone for these high l samples. This possibility, although small by the argument of the temporal jitter above, implies that our estimated conduction speed of the intracortical fibers for our low d samples may be quite inaccurate for unexpected reasons. In any case, our conclusion should still holds for our high d data samples, for which intracortical connections conduct at speeds that are much faster than previously thought.

5 Discussion

When two V1 neurons are $d > 2$ mm apart, they should be in different hypercolumns in V1 and most likely have non-overlapping receptive fields. In monkey V1, at eccentricity E, the area of the cortex devoted to one unit area of visual field size is described by this cortical magnification factor (van Essen et al., 1984)

$$M_a = 103(0.82^o + E)^{-2.28} \text{mm}^2/\text{degree}^2. \tag{1}$$

Accordingly, at eccentricities $E = 5^o$, 10°, 20°, and 40°, respectively, a distance of 2 mm (or 4 mm) in V1 spans roughly 1.5^o, 3°, 6.3^o, 13.5^o (or roughly 3°, 6°, 12.5^o, 27°) in visual angle, while the average size of the receptive fields is roughly 0.3^o, 0.6^o, 1.1^o, and 3.2^o in one dimension (according to Fig 7A of van Essen et al. (1984)). When the first neuron sends an intracortical axon to synapse on the second neuron, the activity of the second neuron can be influenced by the contextual visual inputs that are in the receptive

field of the first neuron (and outside its own receptive field). Our findings suggest that intracortical connections linking these two neurons have high conduction speeds. These speeds are sufficient for the fast propagation, at around $v = 1$ m/s, of contextual influences observed physiologically (Bair et al., 2003).

It is likely that these long intracortical V1 connections are myelinated (Waxman and Bennett, 1972). If one assumed a velocity v for $d > 2$ mm based on the median $v = 0.33$ m/s dominated by $d < 2$ mm samples, the latency for $d = 3$ mm, 5 mm, or 10 mm would be 9 ms, 15 ms, or 30 ms, respectively. However, all our data samples have a latency $l \leqslant 10$ ms, sufficiently short for the physiologically observed contextual influences, which emerge about 10–20 ms after the start of the neural responses to visual inputs (Knierim and Van Essen, 1992). It is therefore incorrect to assume that the intracortical connections are too slow for the contextual influences.

We found that conduction velocity of intracortical V1 axons grows approximately linearly with the conduction distance. Hence, when contextual influences are mediated by the intracortical axons, contextual inputs at different distances from a neuron can synchronize their influences on this neuron by a common latency. Since V1's intracortical axons have a finite range of several millimeters, contextual inputs that are too far can exert their influences via the intervening contexts. For example, let A, B, and C be three separate contour segments along a long and smooth object contour, such that the intracortical connections could directly link between segments A and B, and between segments B and C, but not between segments A and C because the distance between A and C is longer than the longest possible intracortical axon. Through mutually facilitative contextual influences, the responses to contour segments A and B can directly enhance each other, so can the responses to segments B and C (Kapadia et al., 1995; von der Heydt and Zhang, 2018). Through the intervening segment B, contour segments A and C can also enhance each other. Computational modeling of the neural circuits demonstrates that the mutual facilitation in such responses to contours emerges by a short latency as a collective behavior of the neural circuit (Zhaoping, 2005). This short latency is not overly sensitive to the spatial extent of the contour, and is part of a collective phenomenon in a neural circuit with recurrent interactions that reinforce positive feedbacks between interacting elements.

Fast intracortical axons do not preclude top-down feedback from also contributing to the contextual

computation (Bullier et al., 2001; Angelucci and Bressloff, 2006; Gilbert and Li, 2013; von der Heydt and Zhang, 2018). However, they compel us to investigate the respective roles, and relative importance, of intracortical and feedback contributions. Both computational models and careful analysis of experimental data have suggested potential roles by both intracortical and feedback connections (e.g., (Craft et al., 2007; Bushnell et al., 2011; Liang et al., 2017; Chavane et al., 2022; Davis et al., 2024)).

For example, some computational models (Li, 2002; Zhaoping, 2005) have demonstrated the feasibilities and potentials of the intracortical interactions for computing context-dependent signals for saliency and border-ownership in V1 and V2 neurons. Simultaneously recorded neurophysiological and behavioral data confirm that saliency signals for upcoming saccades emerge in V1 40 to 60 ms from the appearance of visual inputs, around the time of initial peak neural responses (Yan et al., 2018). These short latencies allow only the following as possible neural bases for saliency: (1) intracortical V1 mechanisms and (2) feedback only from visual areas that have response latencies shorter than 40 to 60 ms (Nowak and Bullier, 1997). Previous data suggest that contextual influences in V1 do not depend on feedback from V2 for some stimuli (Hupé et al., 2001), but, in responses to low-saliency inputs, are affected by feedback from MT (Bullier et al., 2001).

In addition, when neural responses are at longer latencies or when context arises from animal's task and internal state, feedback should contribute more significantly (Bullier et al., 2001; Angelucci and Bressloff, 2006; Gilbert and Li, 2013; Yan et al., 2018; von der Heydt and Zhang, 2018). For example, reversible inactivation of monkey MT showed that feedback connections serve to amplify and focus activity of neurons in lower-order areas for differentiating between figure and ground (Hupe et al 1998). Furthermore, while V2 neural responses can signal border ownership based on contextual inputs, memory-like persistence of this signal after the context is removed, and de novo transfer of this signal across saccades to responses of other neurons, suggest a strong role of feedback signals from higher visual areas such as V4 (O'Herron and von der Heydt, 2013; Franken and Reynolds, 2025). Meanwhile, it is preferable to refine the definition of "contextual influences" and categorize them into different kinds. Some contexts are in the visual input image, other contexts are from the animal's behavior and task, and the influence of a visual input context can be greatly modulated by attention and task (Freeman et al., 2003; Li et al.,

2004). Top-down feedback for visual recognition, using the computation of analysis-by-synthesis, are certainly present (see e.g., (Albers et al., 2013; Zhaoping, 2019, 2025; Xin et al., 2025)) and are likely to manifest in, e.g., figure-ground effects mediated by intracortical connections. Understanding the roles of feedforward, lateral, and feedback connections in various kinds of contextual influences is essential to understanding how vision works (Zhaoping, 2014).

In summary, this paper reports that the intracortical axons can be sufficiently fast, and their conduction speed increases with distances, allowing them to convey information from contextual inputs nearly synchronously from near and far. These findings should enable fresh progress in answering long-standing questions on how neural circuits combine feedforward, horizontal, and feedback connections to carry out critical computations on global properties about visual scenes in the three-dimensional world based on local image features in two-dimensional retinal images. Furthermore, they should help us dissect the roles of various circuit components for visual recognition and action modulated by attentional states and task demands.

6   Acknowledgement

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