COGNITIVE NEUROSCIENCE

Efficiently adding up our sensory evidence

How do we effectively process the information arriving to our senses to make adaptive decisions and behave appropriately, and which brain areas are responsible? A new study combines multimodal noninvasive neuroimaging in humans to reveal the anatomical locus of efficient sensory evidence accumulation.

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We constantly encode and process sensory information from the environment to make decisions and respond appropriately. This must all happen very efficiently: if you are driving on a dark, foggy road in the rain, effective sampling and processing of visual information directly facilitates potentially life-altering decisions, such as whether to stop, swerve or accelerate at any given moment. So, perhaps unsurprisingly, decades of research have been dedicated to revealing how brains accomplish this fundamental feat from both neural and computational standpoints. One resulting influential framework is that of evidence accumulation[1], which posits that sequential 'samples' of sensory evidence are taken from the environment and 'added up' over time until they reach a threshold, triggering the organism to act. But how does this evidence accumulation occur, and which brain areas do the work? Much of what we know so far comes from studies involving invasive neural recordings in non-human animals[2]. But such invasive studies are all but impossible in humans, where we are most often restricted to neuroimaging measures that have good temporal resolution or good spatial resolution, but not both; this means questions about the neural basis of evidence accumulation in humans remain largely unanswered. Importantly, however, answering these questions could not only provide basic science insights, but also shed light on cases where evidence accumulation—and, hence, efficient perceptual decision-making—becomes impaired due to disease or injury, as can occur in Parkinson's disease, schizophrenia and attention deficit hyperactivity disorder (ADHD) among others. A new study in this issue of *Nature Human Behaviour* by Brosnan and colleagues[3] tackles this gap in our understanding.

One candidate set of brain regions for evidence accumulation, based on previous animal studies, is the dorsal frontoparietal network (dFPN)[4]. To investigate how the dFPN may underlie efficient information accumulation in humans, the researchers in this study cleverly combined a behavioral task with scalp electroencephalography (EEG) recordings and structural and functional magnetic resonance imaging (MRI). They investigated the relationship between a specific EEG measure—the centroparietal positivity (CPP), a specific marker of evidence accumulation distinct from both early stimulus processing and generation of motor responses[5]—and various aspects of the dFPN, including white matter tract volume and functional connectivity.

Human observers viewed randomly moving dots presented in patches on a computer screen. On each trial, one of the patches briefly 'pulsed' (i.e., 50% of the dots moved coherently downward), and observers pressed a button as fast as possible to indicate that they saw the coherent motion. Simultaneously, the researchers measured various EEG components, including how quickly the CPP waveform grew in magnitude before the participant responded on each trial. Results first confirmed that this CPP build-up rate, or slope, was a significant trait-level predictor of individuals' reaction times over other EEG measures: the steeper the slope, the faster the person's responses.

Importantly, the average CPP slope for each person also predicted individual differences in MRI-based measures of the dFPN, directly addressing the study's goal of revealing the neural networks critical for perceptual evidence accumulation efficiency in humans. First, the researchers computed the white matter tract volume in three branches of the superior longitudinal fasciculus (SLF; a network of white matter tracts connecting frontal and parietal regions): dorsal, middle and ventral. The dorsal branch projects to dFPN, while the middle projects to both dorsal and ventral FPN (vFPN), and the ventral projects to vFPN only. Evaluating all three is important, because doing so speaks to the specificity of any findings to the dFPN per se, and not to the FPN in general or to other regions of the brain. Critically, statistical analysis revealed that volume of the dorsal SLF tract only (excluding the middle or ventral tracts) significantly predicted participants' CPP build-up rates, suggesting that more efficient transmission along this relay may directly facilitate efficient perceptual information accumulation.

Second, the researchers computed resting-state functional connectivity (functional MRI blood-oxygen-level dependent (BOLD) signal covariation) from nodes in the dFPN and vFPN to all other areas of the brain. CPP slope significantly predicted functional connectivity between dFPN and the left dorsal premotor cortex, as well as between a higher visual region associated with the dFPN and nodes in visual cortical networks, but not any connectivity with the vFPN. Finally, another EEG measure—one that also predicted participants' reaction times but is associated with motor movement preparation instead of evidence accumulation (LHB; the latency of stimulus-locked left-hemisphere beta power near motor cortex)—did not correlate with any of the structural or functional MRI measures the researchers tested, showing that the findings are specific to evidence accumulation markers. Together, these findings support the interpretation that information transmission specifically between the dFPN and the visuomotor system directly underlies efficient evidence accumulation, facilitating speeded behavioral responses in perceptual decision-making.

Brosnan and colleagues' findings build on previous literature because the CPP is related to the P300 EEG signal (especially the P3b), which: (1) has been shown to partly rely on dFPN structures in humans[6,7]; (2) may predict clinically relevant differences in several neurological and psychiatric populations; and (3) can be altered by pharmacological[8] and behavioral[9] interventions. But the P300 is not specific to evidence accumulation only, having been implicated in many other perceptual and cognitive processes too. Thus, the present study provides important new insight about the neural source of the CPP signal as a

specific marker of evidence accumulation efficiency, zeroing in on the white matter projections to the dFPN and on functional information transmission between dFPN and visuomotor brain regions.

This study is of course limited in its causal implications, meaning it cannot reveal whether dorsal SLF integrity causes efficient evidence accumulation or whether efficient evidence accumulation in the brain in general induces plasticity to, from and within the dFPN, increasing structural and functional connectivity. It is also possible that other brain areas or functional properties not tested by the researchers could also correlate with evidence accumulation efficiency, and future studies should focus on expanding on these results. Nevertheless, the present study uses a novel, theory-motivated, multimodal imaging approach in humans to provide important new understanding of how one neural locus may be responsible for facilitating perceptual evidence accumulation efficiency, and results suggest that structural or functional connectivity between the dFPN and visuomotor regions could be an exciting potential target for future interventions to improve clinically relevant outcomes in various patient populations. ❐

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References

1. Ratcliff, R., Smith, P. L., Brown, S. D. & McKoon, G. *Trends Cogn. Sci.* **20**, 260–281 (2016).
2. Hanks, T. D. & Summerfield, C. *Neuron* **93**, 15–31 (2017).
3. Brosnan, M.B. et al. *Nat. Hum. Behav*. https://doi.org/10.1038/s41562-020-0863-4 (2020).
4. Huk, A. C. & Shadlen, M. N. *J. Neurosci.* **25**, 10420–10436 (2005).
5. O'Connell, R. G., Dockree, P. M. & Kelly, S. P. *Nat. Neurosci.* **15**, 1729–1735 (2012).
6. Soltani, M. & Knight, R. T. *Crit. Rev. Neurobiol.* **14**, 199–224 (2000).
7. Linden, D. E. J. *Neuroscientist* **11**, 563–576 (2005).
8. Loughnane, G. M. et al. *J. Cogn. Neurosci.* **31**, 1044–1053 (2019).
9. Ziegler, D. A. et al. *Nat. Hum. Behav.* **3**, 746–757 (2019).

Competing interests

The author declares no competing interests.