

magnetic field that generates the Zeeman splitting points in opposite directions in the two valleys.

Yuan *et al.* fabricated an ionic-liquid-gated field-effect transistor⁷ on a rather thick, bulk WSe₂ flake. In the bulk, the positions of the tungsten atoms and the dichalcogenide units (Se₂) alternate when moving from one crystalline layer of WSe₂ to the next. This means that the in-plane electric fields in adjacent layers are equal in magnitude, but in the opposite direction. The charge carriers experience the electric field averaged over different crystalline planes (which vanishes), and so no strong SOI is expected in the bulk. Nevertheless, when Yuan and co-workers applied a gate voltage to the device such that holes accumulated in the valence band at the surface of the WSe₂ flake, they observed a positive magnetoresistance with the characteristic shape of a well-established phenomenon known as weak antilocalization. This is an unambiguous signature that the accumulated holes experience a significant SOI.

The authors argue that the microscopic origin of the SOI in the FET channel, at the surface of a bulk crystal, is analogous to that present in an individual monolayer. The carriers in the channel are confined to the first few crystalline planes near the surface by electrostatic screening, and their wavefunction decays exponentially as it penetrates deeper inside the material. This prevents the averaging of the in-plane electric field that occurs in the bulk, so the unusual SOI associated with monolayers persists in the FET channel. The phenomenon comes with a bonus. Increasing the gate voltage shortens the screening length, resulting in a steeper exponential decay of the wavefunctions that further violates the compensation of

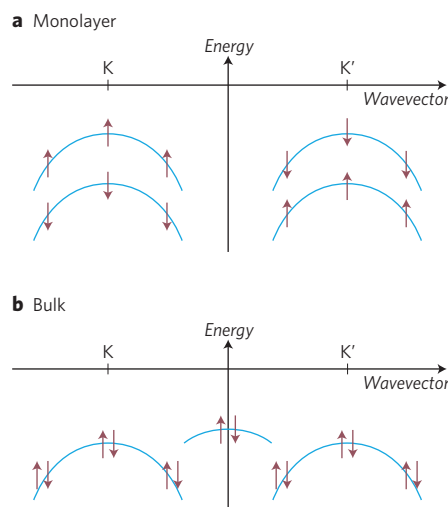


Figure 1 | Valence band structure of WSe₂.

a, The degenerate K and K' valleys in bulk WSe₂ split in single-layer samples owing to an unusual spin-orbit interaction that mimics the Zeeman effect of a magnetic field (that points in opposite directions in the two valleys). **b**, This unusual spin splitting is absent in the bulk. Yuan *et al.*¹ now discuss how, in the channel of an FET at the bulk surface, the spin-splitting characteristic of monolayers can reappear, with a magnitude that is tuned by the gate voltage.

the effects of the in-plane electric fields. Increasing the gate voltage, therefore, can tune the strength of SOI in an unprecedented way, from virtually zero to extremely large values.

More work will certainly follow. Yuan *et al.* have demonstrated the presence of SOI in their devices and produced a physically convincing theoretical explanation of its origin, however they do not provide direct experimental evidence of its anomalous nature. For that, a comparative study with

electrons accumulated in the conduction band — for which anomalous SOI is not expected — should be performed. The crystal quality of the WSe₂ was insufficient to reach the low-temperature metallic regime for electrons, but the experiment will be feasible in other semiconducting TMDs⁸. A more accurate analysis of the weak anti-localization magnetoresistance data should be performed, because it has become clear from graphene research that regimes not described by the conventional theory can occur in multi-valley systems^{9,10}. The possibility of controllably spin-polarizing the valleys of a semiconductor using a gate electrode represents a first step in the search for other unexpected physical phenomena, and possibly in the implementation of technologically relevant devices. This is why we should be ready for more surprises in the near future. □

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NEURONAL NETWORKS

Focus amidst the noise

High-resolution imaging of neuronal networks reveals that spontaneous bursts of collective activity are a consequence of an implosive concentration of noise.

John M. Beggs

At first glance, the brain can seem random. Neuron branches look tangled, the voltage across the membrane of a single neuron follows a random walk and groups of neurons that become simultaneously active seem to

be scattered with no particular pattern. One of the chief tasks in biophysics is to find regularities that could reveal order in this apparent disorder. In this respect, the work of Hodgkin and Huxley represents a major achievement from the past century:

they were able to show how the seemingly noisy fluctuations in an individual neuron's membrane voltage can trigger highly repeatable voltage spikes — known as action potentials — that are the fundamental neuronal signal of the brain¹. Now, over

60 years later, biophysics is faced with a similar problem: how does the apparently random, low-level spontaneous activity of a few-neuron system produce a structured network burst, where a large fraction of the neuronal population fires spikes nearly simultaneously? Javier Orlandi and colleagues have made an impressive step towards answering this question, as they now report in *Nature Physics*².

To understand their work, it is necessary to give some background about how neurons in networks interact. An individual neuron can be thought of as a threshold device: tiny currents injected through the synapses from other neurons add charge to a recipient neuron and can cause its membrane voltage to exceed a threshold for initiating a voltage spike (Fig. 1a–c). This spike travels down a cable-like fibre called an axon, in turn injecting tiny currents into many other neurons. The current injected by one neuron is rarely enough by itself to produce a spike in another neuron, and so activity in the network is driven by collective interactions among neurons. This interesting

arrangement allows a host of complex emergent phenomena to exist, including waves, oscillations, synchrony, avalanches and network bursts.

Perhaps the most fundamental of these is the network burst (Fig. 1d), and intensive work has been devoted to understanding its initiation, propagation and structure³. Previous research has investigated burst leader neurons⁴, network connectivity⁵ and the time course of burst onset⁶. Although each of these has a role to play, a unified picture of burst dynamics has been missing.

To address this, Orlandi *et al.* recorded activity in thousands of individual neurons over time². In contrast to more commonly used electrode-array recordings, their imaging approach enabled them to locate many more neurons, giving much-needed spatial details about how bursts developed. Interestingly, they found that although the activity preceding a burst seemed to be generated randomly, network bursts formed as waves that consistently flowed out of a few small regions they called nucleation sites. Each nucleation site was not merely a single

highly active neuron, but could consist of many neurons. Because this process converted apparently random activity into structured network bursts, they called the phenomenon ‘noise focusing’.

Orlandi and colleagues provide some insight into the mechanisms underlying these nucleation sites². When they cut away a peripheral part of the cultured network, the locations of the nucleation sites moved. This suggests that non-local interactions determine their placement. Detailed computer simulations also indicated that no single variable — whether firing rate of individual neurons or local clustering of connections — significantly correlated with the locations of the nucleation sites. Rather, a complex interplay of many dynamical and structural features is likely to be involved, suggesting that these sites are an emergent property of complex networks. An especially intriguing finding from the simulations is that each nucleation site establishes a basin of attraction, drawing in nearby cascades of spontaneous activity and amplifying them into bursts as they are sent out.

Although tantalizing, it is not yet clear how general this phenomenon is. The experiments used dishes of cultured networks of neurons, which may differ in their connectivity patterns from networks found in the intact brain. Thus, it will be important to see if noise focusing can indeed be found in awake animals, and if so, what functions it may serve. However, many non-neural networks are composed of units that have nonlinear activation functions similar to neural networks. Thus, the findings here in neurons might actually generalize to a wide variety of systems, like Twitter networks⁷, or collections of interacting economic agents. If so, then this could be the first example of a phenomenon in the brain serving as a model system for our understanding of the external world. Usually it is the other way around. □

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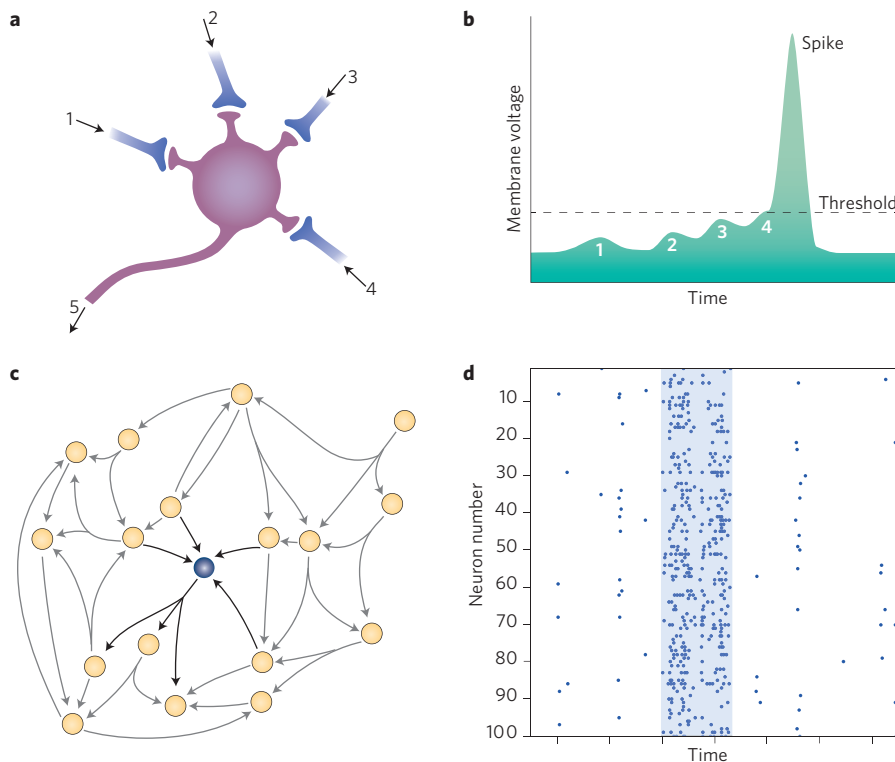


Figure 1 | Collective interactions among neurons. **a**, A single neuron can receive injected currents from other neurons (1–4) through synapses. Output travels along the axon (5). **b**, A single input (1) will not drive a neuron over threshold, but several inputs at the same time (2–4) will. Once activated, the neuron produces an action potential, also called a spike, leading to current injection at other neurons. **c**, The neuron is embedded in a network, where it receives and makes many contacts. Driving a neuron over threshold requires many simultaneous inputs. This means that network activity is typically driven by collective interactions. **d**, Network bursts are one example of such activity. Usually spontaneous activity is sparse, but occasional bursts (highlighted region) are characterized by a large fraction of the neurons spiking in a short interval.