shortcomings mean that researchers have had limited confidence in using these images to assess whether the historical records of tropical cyclones contain evidence that such storms have intensified.

Wang *et al.* took a different approach, by making use of the relationship between ocean-current velocities and the forces that winds exert on the ocean's surface. Using a vast network of floating devices called drifters, which move with the ocean currents, they pulled together a record of 84,110 wind-speed measurements taken during tropical cyclones that occurred between 1991 and 2020 (Fig. 1).

Ocean currents near the surface exhibit extremely high velocities in a tropical cyclone, in response to the rapid onset of strong winds at the surface[8]. However, the instruments used by Wang *et al.* are equipped with a funnel-shaped device, known as a drogue, that allows the instruments to drift smoothly through the ocean's mixed layer — a thin layer of warm water adjacent to the surface. The stability of these surface drifters enables them to record highly accurate data[9], even when violent storms are raging. The authors showed that estimates of the surface winds obtained with these instruments were consistent with wind measurements recorded by nearby buoys.

A clear message emerges from this data set: the ocean currents beneath tropical cyclones have grown stronger over the past three decades, and there are upward trends in all ocean basins in which these storms occur. The coverage of these devices in the western North Pacific Ocean is sufficient for the authors to conclude that the increase occurred beneath storms of all intensities. However, the network of drifters intersects strong storms infrequently in other ocean basins, so Wang *et al.* restricted their analysis to weak tropical cyclones.

The authors' results are supported by further data showing that surface cooling in the wake of tropical storms has increased in the past 30 years. Strong currents near the surface create a shear across the base of the mixed layer, which, in turn, induces mixing to bring deeper, colder waters up to the surface. This process leaves the ocean surface colder after the passage of a tropical cyclone than it was before. Wang *et al.* note that there were no substantial changes to other factors that could modify the extent of cooling, such as the depth of the mixed layer or the speed of currents during storms. They therefore conclude that the trend towards increased cooling beneath tropical cyclones confirms their results from ocean-current velocities: storms are stronger now than they were 30 years ago.

Wang and colleagues caution that their results are robust only for weak storms, because the sample of data collected during stronger storms is, at present, too small to allow their analysis to be extended to the full range of intensities. I suspect, though, that their data constitute evidence that all tropical cyclones have grown in intensity. After all, even the strongest storms spend a considerable time in a relatively weak form, both as they are developing and — for those that remain at sea — during their decline. The authors are confident that their data show an increase in the wind speeds of storms with a maximum intensity that means they are classed as tropical storms and weak hurricanes. But the data actually pertain to any storm that passes over an ocean drifter while its wind speed is in this low-intensity range, so some of the data do actually correspond to stronger storms.

Previous work has shown that the strongest typhoons and hurricanes tend to have occurred more often in recent years, compared with the 1970s[10]. When viewed in conjunction with this finding, Wang and colleagues' analysis should boost our confidence in predictions that the intensity of storms will also increase as the planet warms. It certainly leaves little doubt that tropical storms around the globe have intensified in the past 30 years.

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Spinal-cord injury

Cells that aid recovery from paralysis identified

Kee Wui Huang & Eiman Azim

Improved treatments for spinal-cord injury require both technological development and insights into the biology of recovery. High-resolution molecular maps of the nervous system are beginning to provide the latter. **See p.540**

Spinal-cord injury can lead to a debilitating loss of movement and sensation. Although full recovery of mobility remains an elusive goal, electrical stimulation of the spinal cord during rehabilitation has enabled substantial improvements in movement, even in individuals who were completely paralysed[1]. On page 540, Kathe *et al.*[2] begin to uncover the neural mechanisms that underlie this improvement. The authors present a detailed molecular map of the injured spinal cord in mice during recovery from paralysis, and use this map to identify a cell type that has a crucial role in rehabilitation. This type of biological understanding is an important step towards achieving full restoration of motor function.

Epidural electrical stimulation (EES) was originally developed more than 50 years ago as a method of pain relief[3,4]. EES involves implanting flexible paddles that contain multiple electrodes under the muscle and bone, just above the spinal-cord dura mater (the outermost membrane that encases the nervous system). The electrodes deliver electric currents that can activate nearby neurons in the spinal cord, as well as neuronal pathways that enter and exit the spinal cord. In the context of spinal-cord injury, this approach has been used to stimulate surviving neurons below the injury, improving motor function[5].

However, progress in treating paralysis using EES initially faced several challenges. For example, the designs of early EES electrodes were not optimal for activating components of the spinal cord that are crucial for motor recovery. Furthermore, EES devices could not flexibly produce the diverse patterns of stimulation that are needed to support a wide range of movements in a manner tailored to the individual[6].

In the past decade, much progress has been made in improving both the technology and our understanding of the biological mechanisms that underpin successful rehabilitation. A key step has been the development of improved implants and stimulation programs[1,7], motivated in part by the discovery that previous designs disrupted sensory

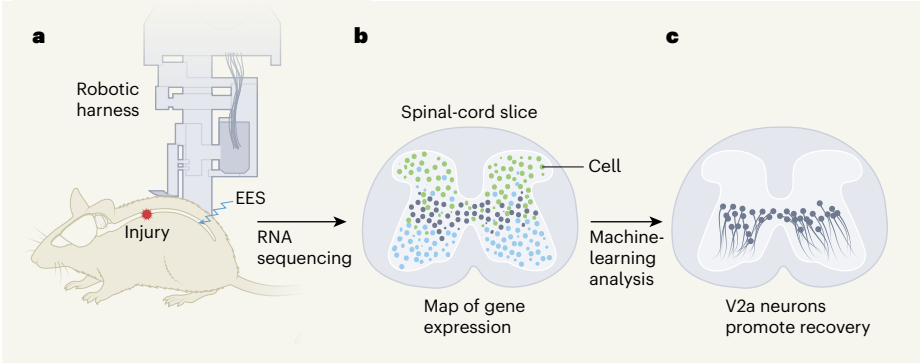

Figure 1 | A mouse model to identify neurons that restore the ability to walk. a, Kathe *et al.*[2] placed mice that had been given a spinal-cord injury in a robotic harness to support the animals' movements during recovery. The authors developed a customized epidural electrical stimulation (EES) approach to activate sensory neuronal pathways during rehabilitation. **b**, They took slices from the animals' spinal cords at various stages of rehabilitation, and used RNA sequencing to generate maps of gene expression across the spinal cord over time. **c**, Using a machine-learning approach to analyse these maps, they found a subpopulation of V2a neurons in the lumbar spinal cord, called $SC^{Vsx2::Hoxa10}$ neurons, that consistently responded to EES. Activating these neurons promoted the restoration of motor function, and improved the ability of the mice to walk.

signals that promote recovery[8,9]. Specifically, newer designs can more-selectively target the 'dorsal root' regions of the spinal cord, which contain neuronal pathways conveying sensory information that can help to enable walking.

The first part of the current study involved a clinical trial in which nine individuals who had severe or complete paralysis were given EES, with three of the participants (all with complete paralysis) receiving treatment with a newly designed electrode. All individuals immediately regained some ability to walk with robotic support during stimulation, and most showed a considerable increase in their ability to bear weight and a sustained improvement in walking after five months of EES treatment and rehabilitation.

What has remained unclear is how EES treatment leads to a reorganization of neural circuits, so that neurons that are spared injury can aid in recovery[6,10]. Understanding how EES reshapes spinal circuits could help researchers to develop targeted techniques to restore walking, and potentially enable the recovery of more-complex movements.

The spinal cord comprises many diverse and highly interconnected cell types. To explore how distinct cell types might be involved in EES-mediated recovery, Kathe *et al.* developed a mouse model that recapitulates many key features of EES neurorehabilitation in humans. They then sequenced RNA from single cells and spinal-cord slices to generate high-resolution maps of gene expression across several stages of rehabilitation (Fig. 1). This strategy allowed them to capture detailed changes in gene expression that occur during EES-mediated recovery.

The group had previously developed a machine-learning approach to analyse gene-expression data that enabled identification of the cell types that respond to a biological stimulus[11]. Kathe and colleagues made use of this tool to pinpoint a specific type of excitatory neuron in the lumbar spinal cord that shows consistent and robust responses to both acute and chronic application of EES. The authors call these cells $SC^{Vsx2::Hoxa10}$ neurons. They are a lumbar subset of a type of cell in the vertebrate brainstem and spinal cord known as V2a neurons, which contribute to various aspects of locomotion and limb movement[12]. The current findings suggest that this cell type is also activated by EES and might have a key role in recovery.

To determine whether these V2a neurons promote recovery of walking, the authors performed a set of experiments to examine the effects of silencing or activating the cells in mice. They found that silencing these neurons impaired the EES-mediated recovery of walking after spinal-cord injury, whereas activating the neurons — even in the absence of EES treatment — produced improvements in walking. These results support a model in which EES triggers V2a neurons to drive a reorganization of spinal circuits and promote the restoration of motor function. More generally, the findings are consistent with the idea that certain types of spinal neuron that have lost their inputs from the brain after injury can be 'reawakened' or repurposed to restore movement if they are given the appropriate combination of stimulation and rehabilitation[6].

"The findings are consistent with the idea that certain types of spinal neuron that have lost their inputs from the brain after injury can be 'reawakened'."

The identification of a recovery-organizing cell type is a big step forward in our understanding of the mechanisms that underlie EES-mediated rehabilitation. But a full picture of how the spinal cord is reorganized has yet to be obtained. Kathe and colleagues' data show that, in addition to V2a neurons, numerous other cell types respond to EES, including many inhibitory neurons. Indeed, a surprising finding of the study is that EES is associated with an overall decrease in neural activity in the spinal cord, suggesting that neural inhibition is part of recovery. How the activation of V2a neurons by EES induces changes to inhibitory neurons, and how EES alters neural dynamics in the spinal cord to promote motor output, remain unknown.

What lies upstream of these neurons is another central question. Spinal-cord injury often spares some of the neuronal pathways that descend from the brain to the spinal cord, causing their connections to become reorganized. If EES treatment alters the signals that the V2a neurons receive from the brain, then identifying and modulating these descending pathways might provide another avenue for treatment.

Despite the challenges that remain, detailed molecular maps and sequencing approaches — such as those described by Kathe *et al.* — will serve as a useful resource to guide future investigation of the circuits that underlie the recovery of movement. The development of approaches for enhancing neurorehabilitation, together with a rapidly growing set of tools for accessing specific cell types in the nervous system[13], make the prospects for targeted, circuit-based treatments for spinal-cord injury seem ever-more promising.

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