flood extents and population exposure[3,4]. They found that the number of people exposed to floods is likely to continue to increase more quickly than the overall population in 59 countries, mostly in Asia and Africa.

Previous studies in this area relied on global flood models that use rainfall statistics and elevation models to map potential riverine and coastal flood zones[5]. Tellman *et al.* instead mapped an unprecedented number of validated events, including various flood types — such as those caused by dam breaks, local rainfall events and snowmelt — that had not been considered in the earlier analyses. As a result, the authors' estimate of the increase in the percentage of people exposed to floods globally is ten times higher than previous estimates.

As with all global assessments, the new work has its limitations. The flood events considered are still just a subset of all the floods that occurred during the study period. This is because the satellite observations capture only floods above a certain spatial extent and that were followed by a period of cloud-free weather, thereby allowing reliable optical detection. Furthermore, the spatial resolution of the satellite data and the use of global population models do not allow a detailed analysis of flood impact in urban areas. Given that the world is rapidly urbanizing and that urban disaster risk is an increasing concern, future studies should develop improved approaches for estimating global flood risk in cities.

The trends revealed in Tellman and colleagues' study might seem daunting, but there is also good news to be drawn from the statistics: the capacity of communities to manage and respond to floods has increased over time. Investments in flood protection, drainage infrastructure and early-warning systems, together with improved building standards, schemes for supporting flood-affected people and strengthened government policies enforcing risk-informed land planning, can both prevent floods and buffer the impacts when they occur[6]. The number of fatalities and extent of flood damage, relative to the number of people and economic assets exposed to floods, has declined globally over the past few decades[7].

As the global population grows and cities expand, natural ecosystems that once provided flood protection will also be under threat. Mangroves, coral reefs, dune systems and urban parks can damp flood waves, reduce peak flows and significantly reduce flooding and other climate-related hazards[8]. Investments in solutions that restore or construct ecosystems often provide a cost-effective way of reducing flood damage while improving biodiversity and providing other benefits[9]. Satellite technology can track changes in protective ecosystems[10], similarly to its use in monitoring flooding and population changes. However, even the best combination of infrastructure and nature-based approaches might be insufficient to deal with rising sea levels — the only option for some communities will be to manage their retreat out of flood-prone areas[11].

Understanding the links between climate change, socio-economic development and flooding is a big scientific challenge, but is essential for developing robust decision-support models that will enable policymakers to calculate and communicate the best mix of measures for future challenges. Tellman and colleagues' improved global estimates of risk are a crucial step in that direction.

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Immunology

Private protection at the brain's border

Britta Engelhardt

At the outer border of the brain and spinal cord, immune cells have been observed that originate from the bone marrow of the adjacent skull and vertebrae. They reach this site through special bone channels, without passing through the blood.

Barriers around the brain and spinal cord of the central nervous system (CNS) protect neuronal cells from the changeable milieu of the bloodstream by controlling movement of molecules and cells between the blood and the CNS. These barriers also ensure that the CNS can be kept under surveillance by certain immune cells, but restrict the access of blood-derived immune cells and molecules to specific compartments at the border of the CNS[1]. Writing in *Science*, Cugurra *et al.*[2] and Brioschi *et al.*[3] report that the dura mater, a tissue layer around the outermost barrier of the CNS, sources a private immune protection from nearby bone marrow.

Encasing the brain and the spinal cord are three meningeal membranes[1,4] (Fig. 1). The outermost membrane, the dura mater, lacks a blood–brain barrier, and so the entry of blood-derived components, including immune cells, into this layer is unrestricted[1,4]. The arachnoid mater is attached to the inner surface of the dura mater. Between the arachnoid mater and the innermost meningeal layer, the pia mater, is the subarachnoid space, which contains cerebrospinal fluid (CSF) and resident immune cells that enter during embryonic development[5]. The arachnoid mater acts as a blood–CSF barrier between the dura mater and the subarachnoid space. The pia mater lies directly on top of the glia limitans, a thin layer of extracellular-matrix material and cell-protrusion endings at the surface of the CNS tissue[4]. The anatomy of the meningeal layers has been likened to the defences around a medieval castle, with two walls (the arachnoid barrier and the glia limitans) bordering a guard-patrolled moat (the subarachnoid space and its immune cells)[6].

The two new studies focused on different subsets of immune cells, namely, myeloid cells of the innate branch of the immune system (which recognizes stereotypical changes characteristic of infection)[2] and B cells of the adaptive immune system (which responds to and remembers specific foreign invaders)[3]. The authors attached the circulatory system of one mouse, in which these subsets of immune cells were fluorescently tagged, to that of a second, untreated, mouse, and made the surprising finding that fewer tagged cells than untagged cells were observed in the dura mater of the second mouse. This finding suggests that a considerable proportion of immune cells in the dura mater do not arrive from the bloodstream, but instead originate from the bone marrow in the skull and the vertebrae of the spine. This shortcut is made possible by the cells crawling along the outside

of blood vessels inside small, bony channels, identified previously[7], between the bone marrow and the dura mater (Fig. 1). Thus, the dura mater sources a private immune protection right outside the outer CNS barrier (the arachnoid mater) from adjacent bone marrow through a previously unrecognized route.

Analysis of gene expression and other characteristics of the individual dura mater immune cells supported the idea that these bone-marrow-derived immune cells are programmed to ensure CNS health, whereas those arriving from the blood tend to be pro-inflammatory and thus more ready to fight potential infections. Moreover, with ageing, increasing numbers of blood-derived immune cells were observed in the dura mater, suggesting a shift in CNS-border immune protection.

Unexpectedly, Cugurra *et al.*[2] found a large number of a type of bone-marrow-derived immune cell called granulocytes in the dura mater. Granulocytes are not typically resident in tissue: they are short-lived, circulate in the blood and usually infiltrate tissue only during acute inflammation. However, the authors found these cells in the apparently uninflamed dura mater, and, indeed, granulocytes have been observed in healthy meninges previously[6].

Therefore, the bone-marrow-derived granulocytes might be a special subset of granulocytes with functions different from those of the cells from the blood. This finding is highly relevant to stroke caused by insufficient oxygen reaching the brain, because the presence of granulocytes in the meninges is a major hallmark of such an event[8,9], and yet blocking granulocyte infiltration from the blood had no effect on the outcome of stroke in clinical trials[10]. Previous work[7] showed that the oxygen-deprived brain can recruit granulocytes directly from the skull bone marrow to the brain tissue by way of the same bony channels described in the present studies.

The signals that recruit cells from the bone marrow to the dura mater remain to be identified. Cugurra *et al.* and Brioschi *et al.* propose a role for the chemokine molecule CXCL12 and its cell-surface receptor CXCR4. However, CXCL12 usually promotes immune-cell retention in the bone marrow[11], and blocking CXCR4 would release these bone-marrow cells into the blood[11]. Thus, these roles of CXCL12 and CXCR4 are difficult to reconcile with a role in direct immune-cell recruitment to the dura mater. One possibility is that blocking CXCR4 on bone-marrow granulocytes might enable these cells to sense gradients of the inflammatory chemokines CCL2 and CCL8, which Cugurra and colleagues found to be highly expressed in the dura mater.

Another indication that the dura mater might provide the CNS with a special form of immune surveillance was that it contains

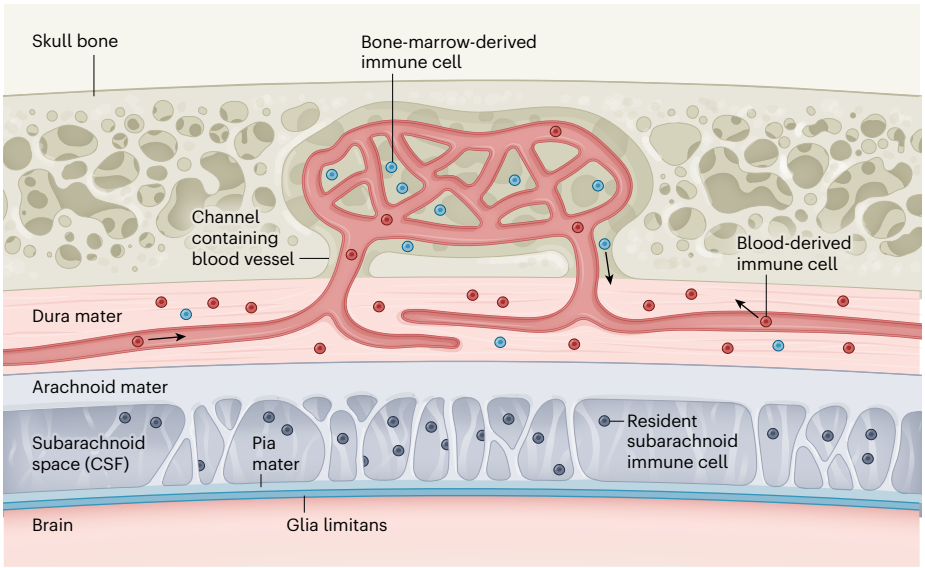

Figure 1 | Immune cells from the bone marrow outside the border of the central nervous system (CNS). Cugurra *et al.*[2] and Brioschi *et al.*[3] studied immune cells in the dura mater, the outermost of three meningeal membranes that surround the brain and spinal cord. In addition to immune cells derived from the bloodstream, they observed immune cells in the dura mater that originated directly from the neighbouring bone marrow in the skull or vertebrae. Immune cells from the bone marrow enter the dura mater by moving along the outside of blood vessels in channels in the bone. The other two meningeal membranes — the arachnoid mater and pia mater — surround the subarachnoid space, which contains cerebrospinal fluid (CSF) and its own immune cells that enter during embryonic development. The arachnoid mater establishes a cellular barrier between the dura mater and the CNS. Under the pia mater, the glia limitans layer, which is made of extracellular matrix material and cellular processes, establishes a further barrier.

a substantial number of immature B cells[3] expressing CXCR4, similar to those in the bone marrow. This suggests that the bone marrow outsources part of its role in B-cell maturation to the dura mater. Indeed, cells of the dura mater were found to produce CXCL12, thus providing a bone-marrow-like environment for these immature B cells.

The signals that drive movement of premature B cells from the bone marrow to the dura mater remain to be identified. The authors speculate that the brain and spinal cord might program immune cells coming from adjacent bone-marrow niches to provide CNS-tailored immune protection. A key remaining question is how the CNS communicates with the immune cells in the dura mater or the bone marrow in the skull and vertebrae, because they are on different sides of the arachnoid barrier, which establishes a barrier between the CNS and the changing blood milieu.

Notably, in this context, the dura mater sends many blood vessels into the skull bone[9]. And, intriguingly, both studies found high levels of myeloid cells and B cells in the dura mater that encases the dural sinus (the large central vein that drains blood, and probably CSF, from the brain), particularly at sites where blood vessels from the CNS, and the subarachnoid space, join the dural sinus. Previous work from some of the authors of the current studies suggested that the dura mater along the dural sinus constitutes a special neuroimmune interface[12]. It is thus tempting to speculate that the signals driving the recruitment of immune cells from the bone marrow to the dura mater, and potentially into the CNS itself, do not cross the arachnoid barrier, but instead move along the walls of blood vessels that form a bridge between brain barriers such as the arachnoid barrier and the glia limitans.

Future research should determine whether skull and vertebra bone marrow are different from bone marrow elsewhere. Furthermore, how do the functions of bone-marrow-sourced immune cells in the dura mater compare with those of the immune cells that keep the subarachnoid space under surveillance? The signals attracting immune cells to the dura mater from the bone marrow, as opposed to from the blood, must also be explored. And how do the bone-marrow-sourced immune cells in the dura mater interact with those from the blood?

The channels connecting the skull and vertebra bone marrow with the dura mater also exist in humans[7], suggesting that observations of immune-cell migration and function in mice might translate to humans. Moreover, because the dura mater lacks a blood–brain barrier, immune cells there could be more easily therapeutically targeted than immune cells in the subarachnoid space or in CNS tissue itself. Understanding the precise role of these dura mater immune cells, the signals that control them and how they contribute to CNS immunity could therefore open up entirely new avenues for the design of treatments for disorders involving CNS inflammation.

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Fluid dynamics

Bouncing droplets mimic spin systems

Nicolas Vandewalle

Experiments show that a collection of bouncing fluid droplets can behave like a microscopic system of spins — the intrinsic angular momenta of particles. This discovery could lead to a better understanding of the physics of spin systems. **See p.58**

In 2005, researchers found that bouncing fluid droplets on the surface of a vibrating liquid bath can self-propel[1]. Remarkably, the dynamical and statistical features of this macroscopic system resemble those of microscopic quantum systems. Building on this work, Sáenz *et al.*[2] report on page 58 that arrays of bouncing droplets can mimic systems of spins (the intrinsic angular momenta of particles). The authors' discovery could increase knowledge of these spin systems, which have uses in spin-based electronics and computing.

In their quest for a better understanding of the emergence of order in typically disordered systems, physicists have developed many models in fields ranging from animal behaviour to materials science. A few of these models have become archetypes that are taught today in advanced physics courses. Let us consider two of them.

The first model concerns the dynamical synchronization of oscillators, which is described in every textbook on nonlinear physics[3] — the study of systems in which cause and effect are not directly proportional to each other. Such synchronization is often illustrated by considering the flashing of fireflies. In the model, when one firefly sees others flashing nearby, it speeds up or slows down its own flashing to be in sync with its neighbours. This behaviour explains why, in some areas of south Asia, the synchronicity of fireflies that land on trees at dusk builds up during the night, as shown in an acclaimed 1990 BBC nature documentary series, *The Trials of Life*. In the model, the collective flashing of fireflies results from their subtle interactions mediated by light.

The second model, from statistical physics, is known as the spin model[4]. It was introduced to study ferromagnetism — the familiar type of magnetism found in iron magnets. In the model, spins are arranged on a lattice that is in thermal equilibrium with a reservoir of heat called a thermal bath. A spin can point either up or down. As with the fireflies, complex physical behaviour emerges when each spin is influenced by its neighbours.

The competition between thermal agitation and spin alignment leads to a transition between ordered phases (for strong spin–spin interactions at low temperature) and disordered phases (for weak spin–spin interactions at high temperature). In the ordered phases, the overall symmetry of the spin lattice is broken because the pattern of spins would look different if flipped upside down, whereas in the disordered phases, such symmetry is retained. The properties of this system are therefore governed by the interactions between spins. The ordered phases can correspond to ferromagnetism, in which spins point in the same direction, or antiferromagnetism, in which neighbouring spins point in opposite directions.

Following on from pioneering work[1,5], Sáenz and colleagues studied fluid droplets bouncing on the surface of a vertically vibrating liquid bath (Fig. 1a). For particular values of the vibration amplitude and frequency, close to those associated with a surface instability called the Faraday instability, each bounce of the droplets generates a surface wave that causes the droplets to self-propel. Furthermore, these surface waves eventually reach other bouncing droplets, inducing non-trivial droplet–droplet interactions and triggering complex droplet trajectories. Collections of such droplets form aggregates of interacting bouncing entities. Two droplets can bounce in sync or out of sync with each other[5]. And in some cases, more than two bouncing droplets can share a single surface wave that exhibits a phenomenon known as coherence[6].

Sáenz and co-workers considered submerged wells that locally change the depth of the liquid bath. Because these depth variations influence the propagation of the surface waves, the bouncing droplets are piloted along specific paths. In particular, circular submerged wells cause the droplets to follow clockwise or anticlockwise circular trajectories. By analogy with magnetic spin, the spin of such a droplet can be defined as the

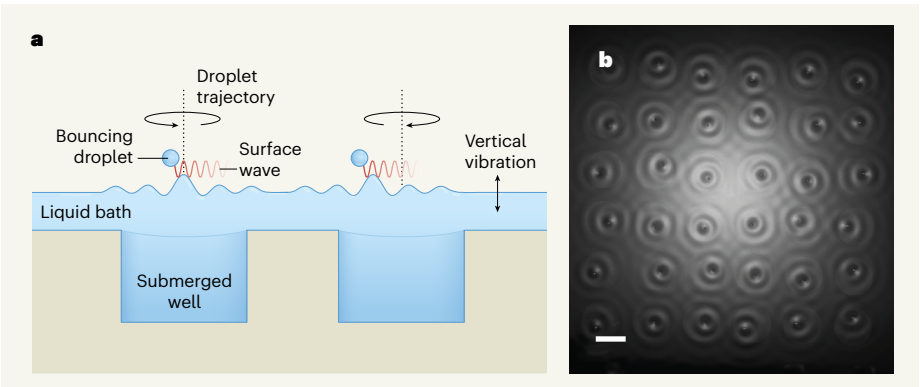

Figure 1 | A system of bouncing droplets. a, Sáenz *et al.*[2] studied the behaviour of fluid droplets bouncing on the surface of a vertically vibrating liquid bath. The depth of the bath varied owing to the presence of submerged wells. Under certain conditions, the droplets generated gradually decaying surface waves that caused the droplets to follow clockwise or anticlockwise circular trajectories and interact with each other in complex ways. **b**, The authors found that arrays of these bouncing droplets (pictured) share many features with systems of spins — the intrinsic angular momenta of particles. Scale bar, 1 centimetre. (Adapted from Fig. 1b and Fig. 4a of ref. 2.)