a whopping 1.6 °C in only 700 years. This is about 1.7 times faster than the ocean is warming now because of global climate change. The reasons for this large warming should be investigated.

The authors also show that the ocean warmed faster than the atmosphere during the Younger Dryas, and then stopped warming before the atmosphere did. By contrast, there was a remarkable synchronicity between Antarctic air temperature, mean ocean temperature and atmospheric CO_2 levels at all other times in the new record. Researchers must now find an explanation for the unusual asynchronicity during the Younger Dryas.

Bereiter and colleagues' work provides an unambiguous record of the average temperature of the entire ocean, from the surface to the greatest depths. However, it does not directly quantify surface temperatures — either global average surface temperature or sea surface temperatures, both of which are useful for understanding and quantifying glacial–interglacial temperature differences and processes. The authors do provide a rough estimate of average surface temperatures from their data, by using a cohort of models to estimate the ratio between sea surface temperature and mean ocean temperature. But this constrains surface temperatures only weakly, highlighting the need for more work in this area.

The authors present several fascinating hypotheses that stem from their data. For example, the observed synchronicity of mean ocean temperature with atmospheric CO_2 levels and Antarctic air temperatures leads Bereiter *et al.* to conclude that the Southern Hemisphere drove Earth out of the glacial period. Furthermore, the large warming during the Younger Dryas suggests that changes in ocean dynamics beyond simple changes to the Atlantic Meridional Overturning Circulation (a climatically crucial component of ocean circulation) could be the cause of this climatic event. Climate modellers must now test these and other hypotheses by adding processes and feedbacks to their climate models, to see how the resulting ocean-temperature changes compare with those in the authors' noble-gas-derived record. Much work will be needed to exploit the full potential of this beautiful record. ■

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CLINICAL NEUROSCIENCE

A bloody brake on myelin repair

In multiple sclerosis, the blood-coagulation factor fibrinogen can enter the brain. It emerges that fibrinogen inhibits the maturation of cells called oligodendrocytes that repair nerve-fibre insulation and maintain neuronal communication.

KLAUS-ARMIN NAVE & HANNELORE EHRENREICH

Multiple sclerosis (MS) is a debilitating neurological disease in which the body's immune system destroys the myelin sheath that provides electrical insulation for nerve fibres. Myelin repair subsequently fails owing to a lack of new myelin-producing cells called oligodendrocytes, and this contributes to an irreversible loss of neuronal projections called axons. Why oligodendrocyte precursor cells (OPCs) located at sites of MS-related tissue damage fail to differentiate into oligodendrocytes has been poorly understood. Writing in *Neuron*, Petersen *et al.*[1] report that a blood-coagulation factor called fibrinogen (which enters the brain when the blood–brain barrier is damaged in MS[2]) puts a brake on OPC differentiation. This insight offers hope for future treatment strategies.

Myelin is made by oligodendrocyte processes that spiral around axonal segments, and it forms a multilayered membrane sheath that speeds up electrical conduction. Oligodendrocyte processes also support axon metabolism. Myelin growth is a fast process in which oligodendrocyte mass multiplies in just a few days[3]. In mammals, myelination begins around birth and OPCs are maintained throughout life; myelination in the cortex of the adult brain is thought to contribute to learning and higher brain functions[4]. Orchestrating timely OPC generation, oligodendrocyte differentiation and energy-demanding myelin synthesis under changing metabolic conditions and in phases of physiological low-oxygen levels[5] is a major challenge. Unsurprisingly, OPCs must integrate a plethora of external stimuli to determine when to differentiate.

Similarly, myelin repair following acute brain injury depends on optimal timing of OPC proliferation and differentiation. Unless cellular

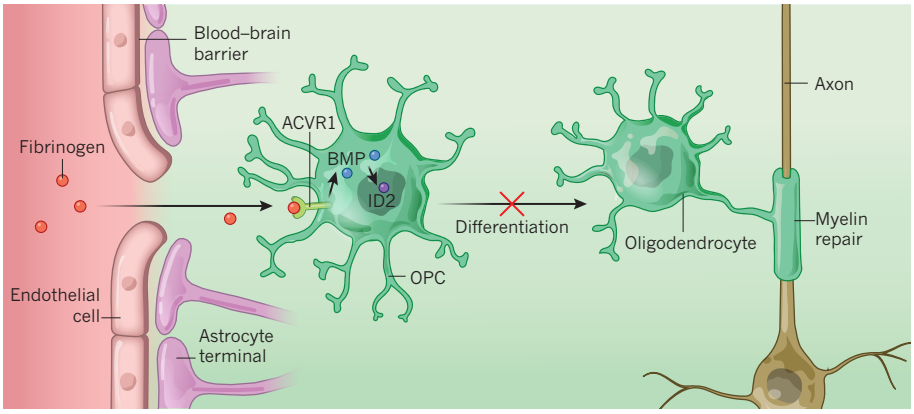

Figure 1 | A coagulation factor and multiple sclerosis (MS). In MS, neuronal projections called axons are stripped of their insulating myelin sheath. Subsequent myelin repair often fails, but the reason for this has been unclear. The blood-coagulation factor fibrinogen crosses the blood–brain barrier (composed of endothelial cells lined with the termini of cells called astrocytes) in MS, and Petersen *et al.*[1] provide evidence that fibrinogen acts to inhibit myelin repair. They show that it binds to the receptor protein ACVR1 on the surface of oligodendrocyte precursor cells (OPCs), triggering an intracellular signalling cascade in which bone morphogenetic protein (BMP) activates the transcription factor ID2. BMP signalling prevents OPCs from differentiating into mature oligodendrocyte cells, which would produce myelin and so drive myelin repair.