OCEAN SCIENCE

North Atlantic circulation slows down

Evidence suggests that the circulation system of the North Atlantic Ocean is in a weakened state that is unprecedented in the past 1,600 years, but questions remain as to when exactly the decline commenced. SEE ARTICLE P.191 & LETTER P.227

SUMMER K. PRAETORIUS

The warm, salty waters of the Gulf Stream make a northeasterly meander across the Atlantic Ocean, eventually forming the North Atlantic Current, which then funnels into the Nordic Seas. In the chill of winter, these waters cool and descend with the heavy load of their salinity. This deep convection is a key part of the Atlantic meridional overturning circulation (AMOC; Fig. 1), which can be thought of as an ocean conveyor belt that releases heat to the atmosphere above the North Atlantic Ocean before travelling through the abyssal ocean to resurface in other areas of the world[1].

Given the importance of the AMOC to heat exchange between the ocean and the atmosphere, the varying strength of this system is thought to have major impacts on the global climate, and has been implicated widely in some of the most remarkable and abrupt climate changes of the past[2]. Direct measurements of the modern AMOC flow rates show a decline in its strength in the past decade[3]. Reconstructions of the natural variability and long-term trends of the AMOC are needed, however, to put these recent changes in context. In this issue, Caesar *et al.*[4] (page 191) and Thornalley *et al.*[5] (page 227) report on past AMOC variability using different approaches. Both conclude that the modern AMOC is in an unusually subdued state, but they diverge in the details of how and when the AMOC's decline commenced.

Caesar and colleagues inferred changes in the strength of the AMOC in the past century from patterns of anomalies in sea surface temperature (SST) that arise in the North Atlantic when the AMOC weakens. The weakening leads to a warming in the Gulf Stream and a cooling in the subpolar gyre — the cyclonic system of wind-driven ocean currents that lies to the south of Iceland (Fig. 1). Although the link between the relatively cool SSTs of the North Atlantic's subpolar gyre and a slowdown of the AMOC have been studied previously[6–8], the main advance of Caesar and colleagues' work is their comprehensive comparison of global SST data sets with state-of-the-art, high-resolution climate models.

The authors' data analysis shows that this bipolar pattern of cooling and warming emerged in the mid-twentieth century. When they performed climate simulations under a 1% yearly increase in carbon dioxide, the model produced a pattern of SST anomalies in the North Atlantic similar to that seen in the observational data, and demonstrated that this pattern was associated with a decline in AMOC strength. The authors then calibrated the model's results with their SST data to estimate that the AMOC has declined by about 15% in the past half-century. They infer that the slowdown in the AMOC was probably a response to warming caused by anthropogenic greenhouse-gas emissions. A possible mechanism could be enhanced melting of the Greenland Ice Sheet[7], which adds fresh water to the surface ocean and reduces the density of the water that drives deep convection.

Thornalley *et al.* provide a longer-term perspective on changes in AMOC strength during the past 1,600 years using a proxy measurement — the 'sortable-silt' grain size[9] — of deep-sea sediment cores that reflects the speeds of the bottom waters that flow along the path of the North Atlantic Deep Water, the deep-water return flow of the AMOC (Fig. 1). They combined this approach with a method similar to that used by Caesar and colleagues: they used past, near-surface temperature anomalies recorded in the marine sediments to provide additional constraints on the AMOC.

The researchers found that the strength of the AMOC was relatively stable from about AD 400 to 1850, but then weakened around the start of the industrial era. This transition coincides with the end of the Little Ice Age — a multicentennial cold spell that affected many regions of the globe[10]. Thornalley and colleagues infer that the weakening of the AMOC at that time was probably a result of the input of fresh water from the melting of Little Ice Age glaciers and sea ice. They estimate that the AMOC declined in strength by about 15% during the industrial era, relative to its flow in the preceding 1,500 years. This is remarkably similar to Caesar and co-workers' estimate, despite the different time periods on which they base their estimates.

However, the roughly 100-year difference in the proposed timing of the start of the AMOC decline in these two studies has big implications for the inferred trigger of the slowdown. Caesar *et al.* clearly put the onus on anthropogenic forcing, whereas Thornalley *et al.* suggest that an earlier decline in response to natural climate variability was perhaps sustained or enhanced through further ice melting associated with anthropogenic global warming. Nevertheless, the main culprit in both scenarios is surface-water freshening.

The two studies are classic examples of 'top-down' and 'bottom-up' approaches, and so it is unsurprising that there is some misalignment

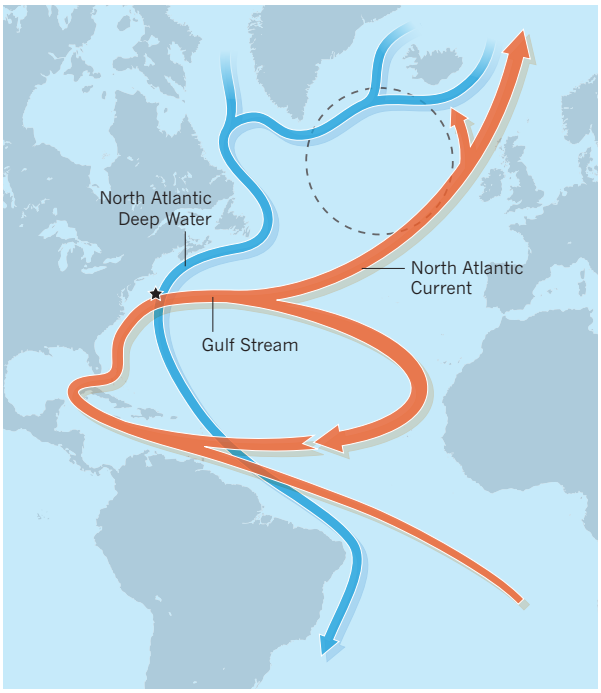

Figure 1 | The Atlantic meridional overturning circulation (AMOC) and the subpolar gyre. The AMOC is an ocean circulation system that consists of warm surface currents (orange) and cold deep-water return flows (blue), as shown in this simplified representation. The surface currents include the Gulf Stream, which feeds a branch of the AMOC known as the North Atlantic Current. The deep-water return flows start from three branches that merge into the North Atlantic Deep Water. Thornalley *et al.*[5] used measurements of silt in sediment cores to reconstruct the flow speed of the AMOC in the past 1,600 years; the black star indicates the approximate location at which the sediment cores were collected. Caesar *et al.*[4] analysed temperature anomalies in the North Atlantic subpolar gyre (dashed line) to infer changes in AMOC flow in the past century. Both studies conclude that the AMOC has weakened by about 15% during the periods considered, but they differ on when the flow started to decline.

between them. Caesar *et al.* take the top-down approach: their inferences of changes in the AMOC strength are made from reconstructions of regional and global SSTs that are derived from direct measurements of temperature. It is possible that regions other than the North Atlantic in which there has been decadal-scale variability in SSTs could influence the mean global SST from which the AMOC strength is calculated — although the authors do attempt to quell such doubts by showing that the subpolar-gyre SST anomaly is robust relative to the global mean SST for a subset of time periods (see Extended Data Fig. 2 in ref. 4).

Thornalley and colleagues' strategy is more of a bottom-up approach: they use a proxy for deep-water current strength to measure AMOC strength more directly than do Caesar and co-workers. The weaknesses of this approach are that it accounts for only the local bottom currents at the sites from which the cores are taken, which might not capture the entire AMOC system, and that it could be susceptible to local nonlinear effects such as abrupt shifts in the position of the current. However, Thornalley *et al.* show that there is a striking correlation between their grain-size proxy and the measured density of the Labrador Sea Water (a major component of the North Atlantic Deep Water), as well as with the heat content of the subpolar gyre; these correlations shore up the bridge that links their localized proxy measurements to broader-scale changes in the AMOC.

For now, the timing of the AMOC decline remains a source of intrigue. Future studies that provide a more-detailed spatial network of multiple proxy reconstructions will help to clarify some of the remaining ambiguity about which elements of the AMOC were changing and when. It is — at least scientifically — reassuring to see that the present two studies converge on the conclusion that the modern AMOC is in a relatively weak state. However, in the context of future climate-change scenarios and a possible collapse in the AMOC[11] in response to the continued melting of the Greenland Ice Sheet[12], it is perhaps less reassuring, because a weakened AMOC might lead to considerable changes in climate and precipitation patterns throughout the Northern Hemisphere[13]. ■

"The two estimates of the decline are remarkably similar, despite the different time periods on which they are based."

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TISSUE REGENERATION

The telomerase enzyme and liver renewal

Cell-tracing analysis reveals that a disperse group of cells in the mouse liver express the enzyme telomerase, which preserves chromosome ends. These cells contribute to liver maintenance and regeneration. SEE LETTER P.244

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The enzyme telomerase maintains the length of specialized repetitive structures called telomeres, which are found at the ends of chromosomes. When they become damaged or shortened, telomeres can stop cells from dividing[1]. Most cells in adult humans have very low or undetectable levels of telomerase and relatively short telomeres, and therefore have a limited ability to replicate[2]. However, elevated telomerase levels are seen in various animal and human stem cells that must retain their replicative capacity for self-renewal[3]. Telomerase defects are associated with tissue scarring (fibrosis) in the livers of both mice and humans[4,5], but which cells in the liver express telomerase, and whether they act as stem cells, has been unclear. On page 244, Lin *et al.*[6] characterize this cell population in mice.

First, the authors identified telomerase-expressing cells in the mouse liver and tracked descendent cells. The group genetically engineered mice to contain a modified version of the gene *Tert*, which encodes a subunit of telomerase. When the mice are treated with a drug, this alteration causes cells expressing *Tert* to be indelibly labelled by a fluorescent protein. Once the genetically modified cells are triggered in this way, they and all their descendants produce the fluorescent protein, even if the cells no longer express *Tert* itself.

Lin *et al.* found that 3–5% of hepatocytes, the most prevalent type of cell in the liver, fluoresce in response to drug treatment. The authors confirmed, by quantitation of messenger RNA levels, that these cells express *Tert*. Next, they examined the livers of adult mice one year after drug treatment. The initially labelled cells (dubbed $Tert^{High}$) had given rise to clusters of descendants dispersed throughout the liver's lobes, making up about 30% of the liver's total mass (Fig. 1). Adult hepatocytes die and are replaced infrequently, so the increase in labelled cells over long periods indicates that the $Tert^{High}$ hepatocytes contribute to the gradual renewal of the liver under normal conditions.

A key question is whether the $Tert^{High}$ hepatocytes are a stable, self-renewing population. Alternatively, *Tert* could be expressed in certain cells for a period of time, then shut off in those hepatocytes and expressed in others. In support of the former case, when Lin *et al.* triggered fluorescent-protein labelling three times over a ten-week period, they found that the numbers of labelled hepatocytes were comparable to those for a single trigger. Next, they showed that 75% of labelled hepatocytes expressed high levels of *Tert* mRNA when they were examined a month after a single drug treatment, whereas only 18% did so after a year, indicating that, as the population gradually expands, $Tert^{High}$ cells not only self-renew but also give rise to progeny that do not express *Tert* ($Tert^{Low}$). Finally, the researchers demonstrated that $Tert^{High}$ hepatocytes proliferate more than $Tert^{Low}$ cells, whereas $Tert^{Low}$ cells exhibit higher expression of genes relating to metabolism and biosynthesis than do $Tert^{High}$ cells.

Taking these data together, the authors suggest that $Tert^{High}$ hepatocytes behave like stem cells. But before concluding that the $Tert^{High}$ cells are bona fide stem cells for the liver, it will be necessary to determine whether the $Tert^{High}$ population becomes exhausted or remains at similar levels in older mice (because hepatocytes are still renewed in ageing mice), and whether $Tert^{Low}$ cells convert to $Tert^{High}$ over longer periods than those used here (which would indicate that this population is not acting as stem cells). It will also be interesting