GLOBAL CHANGE

Interglacial and future sea level

Peter U. Clark and Peter Huybers

A merger of data and modelling using a probabilistic approach indicates that sea level was much higher during the last interglacial than it is now, providing telling clues about future ice-sheet responses to warming.

Predicting sea-level rise in a warming world is one of science's great challenges. According to sea-rise projections for the twenty-first century, the 145 million people living within a metre of the present sea level risk losing their land and their homes. Many more would be affected by the resulting socio-economic disruption[1]. Our poor understanding of ice-sheet dynamics means that projecting sea-level rise beyond the twenty-first century is much less certain[2]. On page 863 of this issue, however, Kopp *et al.*[3] derive a new assessment of sea level during the last interglacial, around 125,000 years ago, that provides insight into this question. If their results are correct, the sea-level rise over the coming century will be followed by many more metres of rise over the ensuing centuries.

Increases in global sea level stem from both expansion of warming water (thermosteric change) and addition of new water from melting ice on land (eustatic change). Predictions of future thermosteric changes are relatively well constrained compared with those of the eustatic change associated with melting of the Greenland and Antarctic ice sheets[4]. There is thus a need to better determine both how much and how rapidly eustatic sea level will rise in response to a given forcing effect such as anthropogenic global warming.

Evidence that sea level during the last interglacial was 4–6 metres higher than at present has long been proposed as a possible analogue for the equilibrium sea-level response to future anthropogenic warming[5,6]. But the sea-level records may include a local response to geophysical adjustments from the preceding glaciation, and thus may not accurately record the global sea level[7]. Furthermore, the implications of 4 or 6 m of rise are quite different: if sea level increases by only 4 m, much of it can be reconciled as being due to thermosteric rise and partial loss of the Greenland ice sheet; anything more requires a contribution from Antarctica.

Kopp *et al.*[3] reach the startling conclusion that, during the last interglacial, global sea level was at least 6.6 m above present, and may have reached 9.4 m, much higher than previous estimates. The implication is that both the Greenland and Antarctic ice sheets were much smaller 125,000 years ago.

To derive this result, Kopp *et al.* compiled a database of proxy measurements of sea level that includes isotopic and coral records, as well as other records that are less well dated. Although this database is more comprehensive than those used in previous studies, constraining estimates for past global sea level from noisy and sparse data whose timing is uncertain is a formidable statistical problem. It is particularly difficult because one must also account for regionally varying geophysical effects, including local tectonic uplift or subsidence, and sea-level changes induced by gravitational, deformational and rotational effects associated with the redistribution of ice, ocean and mass of the solid Earth[8]. Using a physical model that includes these effects, Kopp *et al.* derived an estimate of the covariance between local and global sea level. They then merged the local–global covariance estimate with proxy estimates of sea level within a Bayesian framework to make temporally complete estimates of global sea level and assess their probability.

The redistribution of mass associated with individual ice-sheet melting causes distinct spatial patterns in sea level[9]. In conjunction with the proxy measurements, Kopp *et al.*[3] also used the modelled patterns to estimate that Greenland and Antarctica each contributed at least 2.5 m of sea-level rise. This estimate is consistent with independent constraints: the maximum Greenland contribution was probably 3.4 m (ref. 10), and the thermosteric plus mountain-glacier and ice-cap contribution was probably no more than 1 m. So, if sea level was at least 6.6 m higher, a minimum of 2.2 m must have come from Antarctica. The Antarctic contribution would probably have come from the inherently unstable West Antarctic Ice Sheet, which locks up the equivalent of at least 3.3 m of sea level[11], so that Kopp and colleagues' result implies that most, if not all, of this ice sheet melted about 125,000 years ago.

Perhaps of greatest socio-economic concern is the possible maximum rate of sea-level rise in a warmer world. According to Kopp *et al.*[3], sea-level rise during the last interglacial was in the range of 6–9 millimetres per year. By comparison, instrumental records indicate that the rate of global sea-level rise over the twentieth century was about 2 mm yr^{-1}. That may have accelerated between 1993 to 2003 to around 3 mm yr^{-1}, at least in part due to an acceleration in mass loss from the Greenland and Antarctic ice sheets[12].

Why was sea level so much higher 125,000 years ago? One possibility is that ice sheets have multiple potential steady states for a given climate[13]. However, the global temperature was apparently 1.5–2 °C warmer than the pre-anthropogenic global average of the past 10,000 years (Fig. 1), despite there being essentially no difference in atmospheric greenhouse-gas concentrations. Climate models have simulated a strong Northern Hemisphere summer warming in response to Earth's more eccentric orbit during the last interglacial, but almost no change in the Southern Hemisphere[14]. Southern warming may then have occurred through an oceanic teleconnection with the north[15], or through changes in the duration of the Southern Hemisphere summer[16], with accompanying feedbacks amplifying this warming.

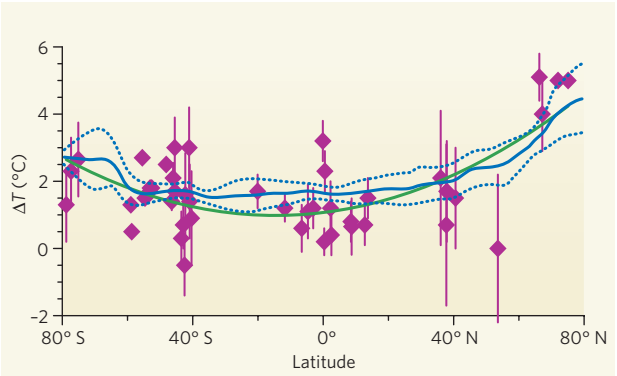

Figure 1 | Similarity of latitudinal warming (Δ*T*) during the last interglacial and a projection for the late twenty-first century. The green line summarizes proxy-data estimates of sea surface and air temperature during the last interglacial relative to the present interglacial before industrialization. Diamonds are largely sea surface temperatures, but include temperatures derived from polar ice cores and two high-latitude Northern Hemisphere pollen records. The temperatures reflect the interval between 120,000 and 130,000 years ago (mean and 1 standard deviation). The green line is a polynomial fit to these data. Surface air temperature estimates from less-well-dated pollen sites in Europe (not shown) similarly show warmer temperatures across most of Europe during the last interglacial[17]. The blue solid line is the zonal mean of the projected surface temperature changes (with 1 standard deviation shown by dotted blue lines) for the late twenty-first century relative to 1980–99; it is based on the SRES B1 greenhouse-gas-emission scenario obtained using the GFDL climate model. (Palaeoclimate data are available at www.ncdc.noaa.gov/paleo/pubs/clark2009.)

In any event, the latitudinal distribution of warming seems to be remarkably similar to the global temperature response to carbon dioxide under a commonly used scenario for greenhouse-gas emissions (compare the green and blue lines in Fig. 1). This suggests that the climate of the last interglacial might, by coincidence, provide a reasonable analogue for establishing ice-sheet sensitivity to global warming. Assuming that Kopp and colleagues' estimates are accurate, and that higher sea level resulted from higher temperatures, the disconcerting message is that the

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equilibrium response of sea level to 1.5–2 °C of global warming could be an increase of 7–9 metres. ■

Peter U. Clark is in the Department of Geosciences, Oregon State University, Corvallis, Oregon 97331, USA. Peter Huybers is in the Department of Earth and Planetary Sciences, Harvard University, Cambridge, Massachusetts 02138, USA.
e-mails: clarkp@onid.orst.edu; phuybers@fas.harvard.edu

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DNA REPAIR

A heavyweight joins the fray

Simon J. Boulton

Tagging of DNA-damage-associated proteins by ubiquitin is key to coordinating the DNA-damage response. The ubiquitin-related protein SUMO is revealed as a crucial regulator of ubiquitylation in DNA repair.

Ubiquitylation — the attachment of ubiquitin groups to cellular proteins — was initially characterized by its role in promoting protein destruction. However, we now know that the consequences of ubiquitylation are diverse, and that it affects many cellular systems. The ubiquitin modification comes in many flavours (addition of a single ubiquitin molecule, for example, or of polyubiquitin chains that differ in the position of the linkage between ubiquitin molecules), and the various types of ubiquitylation can alter the fate of target proteins in different ways. In addition, the cell has ubiquitin-related modifiers, such as the SUMO proteins, that also alter protein fate or function after conjugation[1]. One process that has been inextricably linked to ubiquitylation is the cellular response to DNA damage. Although studies[2,3] had suggested a link between the DNA-damage response and the SUMO pathway, proof that SUMOylation is important for DNA repair had remained elusive. In this issue, two groups, Morris *et al.*[4] (page 886) and Galanty *et al.*[5] (page 935), now provide good evidence that SUMO functions together with ubiquitin to coordinate DNA repair.

DNA double-strand breaks (DSBs) result in the recruitment and activation of the protein kinases ATM, ATR and DNA-PK, which phosphorylate target proteins, such as the variant histone H2AX. The phosphorylated proteins then promote the recruitment of other DNA-repair proteins to DSBs[6], including MDC1 (mediator of the DNA-damage checkpoint), 53BP1 and the E3 ubiquitin ligases RNF8, RNF168 and BRCA1 (ref. 6), which catalyse ubiquitylation events[7] at DSBs. (Conjugation of ubiquitin or related modifiers to target proteins requires an E1 activating enzyme, an E2 conjugating enzyme and an E3 ligase.)

To investigate the involvement of the SUMO pathway in the DNA-damage response, Morris *et al.*[4] and Galanty *et al.*[5] analysed the subcellular localization of SUMO-pathway components in mammalian cells. Both groups[4,5] report that the E1 SUMO-activating enzyme SAE1, the E2 SUMO-conjugating enzyme UBC9, and the three forms of vertebrate SUMO protein, SUMO1 and the closely related SUMO2 and SUMO3 (SUMO2/3), are recruited to DSBs.

The authors[4,5] used RNA interference and fluorescence microscopy to show that the SUMO E3 ligases PIAS1 and PIAS4 are responsible for SUMOylation events at DSBs. Depletion of PIAS1 impaired accumulation of SUMO2 and SUMO3 (but not SUMO1) at DSBs, whereas depletion of PIAS4 impaired recruitment of SUMO1 and SUMO2/3. Furthermore, recruitment of 53BP1 to DSBs depended on PIAS4, whereas recruitment of BRCA1 depended on both PIAS1 and PIAS4. Is SUMOylation necessary for DSB repair? The answer is, emphatically, yes — cells lacking PIAS1 or PIAS4 showed defects in DSB repair and were also highly sensitive to DSBs caused by ionizing radiation.

What are the targets of the SUMO pathway during the DNA-damage response? Prompted by a study showing interaction between UBC9 and BRCA1 in the nematode worm *Caenorhabditis elegans*[2], both groups[4,5] independently showed that BRCA1 is SUMOylated during the DNA-damage response in a PIAS1- and PIAS4-dependent manner (Fig. 1). Depletion of PIAS1 and PIAS4 impaired recruitment of BRCA1 to DSBs[4,5], significantly impaired ubiquitylation at DSBs, and reduced ubiquitylation of the histones H2A and H2AX; the latter process has been shown to require the ligase activities of RNF8, RNF168 and BRCA1 (ref. 7). Galanty *et al.*[5] also showed that 53BP1 is SUMOylated and that this affects its retention at DSBs.

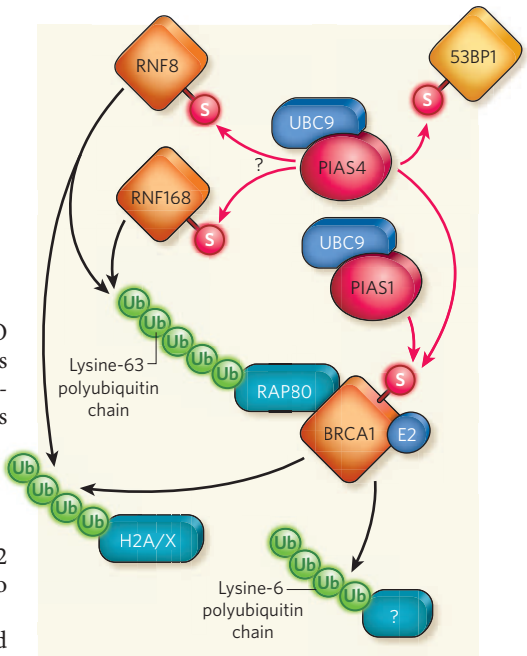

Figure 1 | Ubiquitylation and SUMOylation at DSBs. Double-strand DNA breaks (DSBs) result in the recruitment of DNA-repair proteins, including 53BP1 and the E3 ubiquitin ligases RNF8, RNF168 and BRCA1. Morris *et al.*[4] and Galanty *et al.*[5] observe that the SUMO-pathway components UBC9–PIAS4 and UBC9–PIAS1 also accumulate at DSBs, where they catalyse the SUMOylation of 53BP1 and BRCA1 (and possibly RNF8 and RNF168). SUMOylation stimulates BRCA1 E3 ubiquitin-ligase activity, leading to ubiquitylation of target proteins at DSBs, including the histone H2A and its variant H2AX. H2A and H2AX are also substrates for ubiquitylation by RNF8 and RNF168, as is RAP80, a ubiquitin-binding protein that also interacts with BRCA1. RNF8 and RNF168 catalyse the formation of lysine-63-linked ubiquitin chains, whereas BRCA1 and its E2 conjugating enzyme catalyse the formation of lysine-6-linked ubiquitin chains. S, SUMO; Ub, ubiquitin. Red arrows indicate SUMOylation; black arrows indicate ubiquitylation.

RNF8 and RNF168 catalyse the formation

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