EARTH SCIENCE

Erosion by cooling

The thermal history of thousands of rock samples convincingly confirms the idea that climate cooling accelerates the rate of erosion at Earth's surface — and implicates glaciers in particular. SEE LETTER P.423

DAVID LUNDBEK EGHOLM

On page 423 of this issue, Herman *et al.*[1] report an analysis of mountain-range erosion rates which shows that global cooling in the past 6 million years has accelerated the destruction of mountains. This result reignites a long-lived debate about links between climate, topography and plate tectonics.

The high topography of Earth's mountain ranges is made when plate tectonics force the continental plates to slowly collide. However, erosion by rivers, glaciers and landslides constantly counteracts this mountain-building process by breaking down bedrock and moving the resulting sediment to lower elevations, where it accumulates in sedimentary basins or oceans. The structure of mountain ranges therefore reflects a complex balance between constructive and destructive forces.

Although we can measure current changes in topography using the Global Positioning System, finding out what happened in the past and obtaining data that span the extremely long timescales of these processes are major challenges. An intriguing question relates to how climate influences the erosion processes, and thereby the elevation and morphology of mountain ranges[2,3].

About 6 million years ago, Earth's global climate started a strong cooling trend that led to glaciations in high mountain ranges and at high latitudes[4]. Extensive ice masses then developed at the beginning of the Quaternary period (the most recent 2.6 million years). It has long been recognized that the volume of sediment that has accumulated in the oceans within the past few million years far exceeds that measured for any other period of a similar length[5], which points to increased erosion rates during the Quaternary[6]. But some have questioned whether this observation is biased by the difficulty of measuring the correct volume of older sediments and, therefore, whether surface processes truly led to faster erosion when the global climate cooled and started to fluctuate[7]. Herman *et al.* address this question by analysing new information: the thermal history of rocks.

Using a technique known as thermochronology, the thermal history of a rock sample can be reconstructed from the relative concentrations of certain noble gases within it or from the distribution of damage trails produced by radioactive decay[8]. Specifically, thermochronology allows the dating of the time when a rock cooled to a 'closure' temperature — the temperature below which gaseous isotopes no longer diffuse out of the rock and/or when damage trails stop annealing. This, in turn, provides an estimate of how fast erosion brought the rock closer to the surface, because temperature decreases with distance from the centre of the Earth. Closure temperatures vary from 70 to 250 °C, depending on the specific thermochronometer used.

By combining several thermochronometers that had different closure temperatures, Herman and colleagues implemented a clever approach to determine past changes in erosion rate. They compiled a global set of nearly 18,000 thermochronology data points and then used an algorithm to reconstruct patterns of erosion rate for several time intervals. The results reveal that the erosion of Earth's mountain ranges did indeed accelerate globally as the climate cooled, confirming the information provided by sediment volumes.

The progressive increase in erosion rate is most pronounced at intermediate latitudes (30° to 50°) within the past 2 million years. Herman and co-workers therefore propose that glaciers are the main driver of the accelerated erosion because, at these latitudes, many high-elevation landscapes were glaciated for the first time. Their conclusion is supported by the fact that glaciers are known to have left their distinctive imprint on the morphology of landscapes within a fairly short period. For example, the substantial landscape modifications that formed the spectacular glacial fjord systems of Norway, Greenland, western North America, Chile and New Zealand (Fig. 1) must have occurred within a few million years. Glaciers are efficient agents of erosion because they can abrade and quarry bedrock as they slide down through steep topography.

The hypothesis that climate change was the main driver of recent increased mountain-range erosion has provoked intense debates[9,10]. Sudden pulses of erosion have conventionally been attributed to changes in tectonic activity rather than climate. Many geologists have therefore interpreted the increased erosion of the recent past as a product of tectonic uplift — even in places where no direct evidence of tectonic plate movements exists.

Unfortunately, Herman and colleagues'

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Figure 1 | Carved by glaciers. Herman *et al.*[1] demonstrate that erosion processes, including those that led to the formation of glacial fjords (here at Bradshaw Sound Fjordland National Park, New Zealand), accelerated globally over the past 6 million years.

analysis cannot resolve what happened in these controversial regions. The reason is that erosion rates in these areas were generally low before they started to increase less than 10 million years ago, but the authors' thermochronological method requires total erosion to be high enough to uncover rocks from depths that are associated with closure temperatures. This amounts to kilometre-scale erosion, even for the thermochronometers that have the lowest closure temperatures. Such levels of erosion are generally reached only in areas where tectonic uplift has maintained high erosion rates for a long period, which is why the researchers' analysis is limited to areas where substantial tectonic activity happens today or occurred at about the time of global cooling.

Even with this limitation, Herman *et al.* convincingly demonstrate the global scale of the recent erosion phenomenon. Their results suggest that climate drives erosion, because, unlike tectonic activity, climate can change synchronously on a global scale. ■

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CANCER

Discrepancies in drug sensitivity

Large panels of human cancer cell lines have been profiled at the DNA, RNA and pharmacological levels to accelerate the search for cancer therapies. But two of those large data sets show only partial concordance. SEE ANALYSIS P.389

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Despite obvious limitations in their ability to model clinical disease, cultured cell lines remain central to research on cancer. But a study by Haibe-Kains *et al.*[1] on page 389 of this issue reveals apparent inconsistencies between two large studies of the sensitivity of hundreds of cell lines to dozens of drugs. The findings sound a note of caution about the interpretation of data from such projects, but do not undermine their value.

The first cell-line panel used for large-scale screening of compounds for anticancer activity was the NCI-60, a diverse set of 60 human lines that has been used to screen more than 100,000 compounds since 1988 (ref. 2). Because the same lines have been profiled at the DNA, RNA, protein and chromosomal levels, molecular aberrations in the cells can be correlated with their sensitivity to drugs[3]. But 60 is a relatively small number. So it was exciting when, in March 2012, the Cancer Cell Line Encyclopedia (CCLE)[4] and Cancer Genome Project (CGP)[5] were published, presenting gene-expression profiles and drug-sensitivity assays for 1,036 cell lines and 24 drugs, and 727 cell lines and 138 drugs, respectively. The publications also contained information on gene-copy number and genome sequence for some of the cell lines, and protein-level data have since been added to the mix. Those extensive databases are being used in numerous laboratories to guide research on the molecular mechanisms of cancer, to generate hypotheses for the development of new therapies, and in conjunction with clinical studies[6].

Haibe-Kains *et al.* analysed the relationships between gene expression and drug sensitivity for 471 cell lines, 15 drugs and 12,187 genes that were in both the CCLE and CGP data sets. They found a rather low correlation between the two. Both original studies were carefully done and carefully documented; there is no implication that the apparent discrepancy involved error on the part of either team. So what is the source of the difference? In principle, it could be due to differences in the gene-expression profiles, pharmacological assays, computational methods or any combination thereof.

The authors found that the gene-expression profiles, which were obtained from microarray studies, showed quite good concordance between the two projects, whereas the pharmacological assays did not (Fig. 1). But that should come as no surprise. The pharmacological assay used by the CGP (the CellTiter 96 AQueous One Solution Cell Proliferation Assay from Promega) measures metabolic activity in terms of a reductase-enzyme product after a 72-hour incubation of cells with a drug; that used by the CCLE (the CellTiter-Glo assay from Promega) measures metabolic activity by assessing levels of the energy-transfer molecule ATP, after 72–84 hours of incubation. Both assays provide indices of the drug's activity against the cells, but they would not be

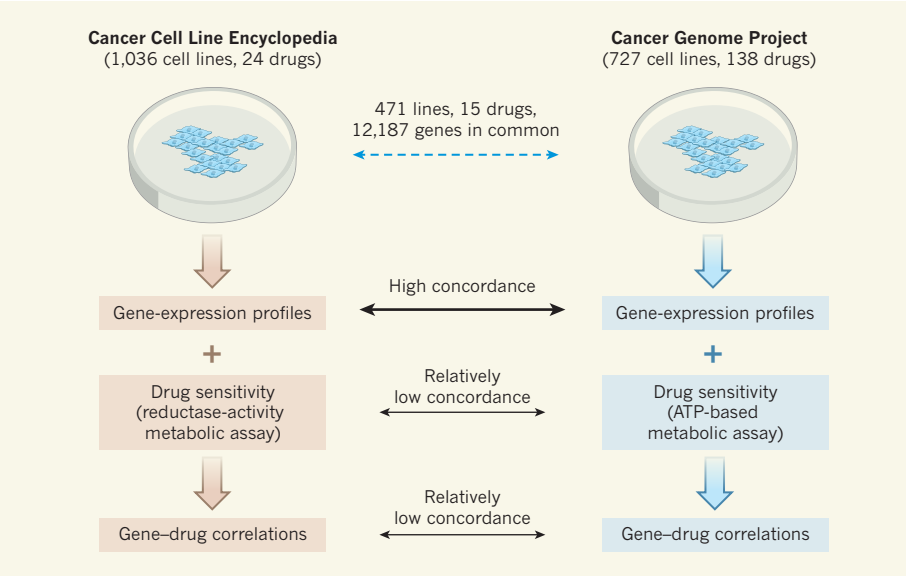

Figure 1 | Study comparison. The Cancer Cell Line Encyclopedia[4] and Cancer Genome Project[5] data provide rich resources for the cancer research community. Haibe-Kains *et al.*[1] analysed the concordance of the two data sets from several perspectives and identified a high concordance between the gene-expression data sets, but a relatively low correspondence between drug-sensitivity assays. That apparent difference propagates into the gene–drug correlations found by the two studies.