

NEWS & VIEWS

ENVIRONMENTAL SCIENCE

Forests and floods

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One strategy for reducing catastrophic floods invokes protecting or regenerating forests that lie upstream of a threatened region. Support for this approach comes from a global-scale analysis of flood risk.

Floods can be devastating, especially in developing countries, which are already struggling to improve the welfare of their people. Floods in such countries during the 1990s took almost 100,000 lives, displaced more than 300 million people and caused more than US\$1 trillion in damage to properties^{1,2}. To mitigate flooding, several nations are investing in projects to restrict the loss of forests or to reforest cleared lands that lie upstream of a potential flood region. But these strategies are controversial. Some argue that the link between forests and flooding is too weak to justify the often large outlays needed to retain or recover forests^{2,3}. In the journal *Global Change Biology*¹, however, Bradshaw *et al.* provide strong correlative evidence that native forests do reduce the frequency and severity of floods in developing nations.

Forests are thought to reduce flooding⁴ by acting as sponges — that is, they trap water during heavy rainfall, then release it slowly into streams, which lessens the severity of floods and maintains stream flows during dry periods. Forests also increase the permeability of the soil and emit water vapour into the atmosphere through evaporation and transpiration, further reducing the run-off of rainwater⁵. For these reasons, a nation such as Costa Rica, which places high value on natural-ecosystem services, and those such as China, India, Nepal and Bangladesh, which have been plagued by devastating floods (Fig. 1), have invested heavily in forest protection or reforestation.

Yet this approach is contentious. Most notably, a serious counterargument came from an influential report² published in 2005 by the Food and Agricultural Organization of the United Nations (FAO) and the Center for International Forestry Research (CIFOR) in Bogor, Indonesia. The analysis concluded that the evidence that forests reduce flooding is weak, especially for the largest and most devastating floods. The report suggested that retaining or regenerating large forest areas was an economically dubious strategy for developing nations, at least from a flood-reduction perspective.

Bradshaw *et al.*¹ step into the fray with a global-scale empirical analysis of the association between forests and flooding; earlier efforts had



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Figure 1 | Jiujiang, China, 12 August 1998. Local dyke defences in the Jiujiang region were overcome during a series of floods caused by relentless rain in the upper catchment of the Yangtze river.

invariably examined much smaller catchment areas or regional scales. The authors evaluated flood frequency, duration and damage (the number of people killed and displaced, and estimated economic losses) from 1990 to 2000 in 56 developing countries in Africa, Asia and Latin America. Using a battery of statistical models, they first controlled for confounding factors such as country size, rainfall, topography, degraded land and soil-moisture regime, then evaluated the residual associations between native-forest cover and flooding.

Their simplest and most realistic models account for more than 65% of the variability in flood frequency and duration, an impressive figure for such a vast, heterogeneous cross-section of Earth. Native-forest cover and the rate of forest loss account for 14% of this variation, on average. Associations between forest cover and the damage caused by floods were weaker but still evident. Statistical simulations

suggested that arbitrarily removing a tenth of the remaining native forest would increase the frequency of floods by 4–28%, and lengthen their duration by 4–8%. The authors emphasize that assessment of flood risk is complex and fraught with confounding factors. But they say that their study shows the need for large-scale forest protection and more reforestation to help reduce the frequency and severity of floods.

In my view, Bradshaw and colleagues' paper¹ will turn out to be a landmark study. But at least one point will provoke debate — their decision to exclude floods driven by extreme events, such as cyclones and typhoons, which they suggest can cause flooding “independently of landscape characteristics” such as forest cover. Their rationale is that massive storms can dump vast amounts of rainfall in just a few days. Even if upstream forests were intact, almost nothing could prevent downstream lowland areas from flooding.

The apparent inability of forests to buffer against extreme floods is one of the main arguments of the FAO–CIFOR team², who assert that expensive efforts to reforest steep catchments in densely populated, monsoonal countries such as Nepal, India and Bangladesh have been largely ineffective. Rather, they argue, investments would be better aimed at other measures, such as discouraging human settlement in flood plains, which can be devastated by flooding during monsoons. Bradshaw *et al.* side-step the issue of extreme flood events. But if nations are to be convinced that forests are valuable for reducing flooding, it will be necessary to show that they are of at least some benefit during the most catastrophic floods. In addition, the broad-brush approach of Bradshaw *et al.* cannot encompass all situations. Because local conditions vary greatly, forests will surely be better at reducing floods in some areas than in others.

Nonetheless, the study's ambitious geographical scope and statistical rigour make it persuasive. The findings add to other evidence that large expanses of native forest can have major benefits not only for reducing floods, but also for limiting wild fires, conserving biodiversity, and slowing regional and global climate change^{4–6}. Tropical forests, in particular, are crucial for combating global warming, because of their high capacity to store carbon and their ability to promote sunlight-reflecting clouds via large-scale evapotranspiration⁷. Such features are key reasons why preserving and restoring tropical forests could be a better strategy for mitigating the effects of carbon dioxide than dramatically expanding global biofuel production⁸.

Flood reduction is one area of environmental science in which the latest research is quickly invoked to justify land-management strategies, with all the pungent realities and controversies this implies. In a world facing potentially serious shifts in rainfall, water run-off⁹ and storm regimes¹⁰ from climate change, and where burgeoning populations are increasingly forced to live in flood-prone areas, the stakes could not be higher. ■

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IMMUNOLOGY

Changed destiny

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For practical and ethical reasons, researchers are on the lookout for ways to reprogramme one mature cell type into another. In one case, this might be as easy as switching off a single gene.

The norm is for most undifferentiated, progenitor cells to irreversibly mature into one particular type of cell. But some cells, even after commitment to a specific fate, can acquire properties of an entirely different cell through a process called cellular reprogramming.

An *in vitro* example of reprogramming is the conversion of skin cells into cells with characteristics of embryonic stem cells by adding a small set of nuclear factors to them^{1–3}. The molecular details of how reprogramming is carried out are not known. In this issue, Cobaleda *et al.*⁴ (page 473) shed light on this mystery by showing that, within the blood system, mature B cells can be converted to functional T cells, and reprogramming is achieved by B cells taking a step backwards to assume a more immature state.

As progenitor blood cells embark on their maturational path, the developmental options available to them become progressively more limited⁵. Lineage commitment is principally controlled by transcription factors that act as master regulators. Examples of these include PU.1 and C/EBP α , which control the commitment of myeloid cells; GATA-1 and FOG-1, which are involved in the development of erythrocytes and megakaryocytes; and Notch-1, GATA-3 and Pax5, which mediate T- and B-cell development.

Conventional wisdom holds that once a cell is committed to a given lineage through the concerted actions of transcription factors and epigenetic modifications to chromatin (complexes of DNA and histone proteins), its fate is sealed. In recent years, however, the unidirectional nature of differentiation has been questioned after reports that cells of the blood lineage are more 'plastic' than previously thought — that is, through various manipulations, they can be reprogrammed to a different lineage⁶. The fate of blood cells can be altered by introducing specific transcription factors not normally expressed in them. For example, introducing C/EBP α into B or T cells converts them into functional macrophages^{7,8}. GATA-1 can also reprogramme common B and T progenitor cells into megakaryocytic/erythroid cells⁹.

In principle, cellular reprogramming may proceed in one of three ways: by de-differentiation to a progenitor stage, followed by differentiation along a different route; by direct transdifferentiation from one lineage to another; or through a process involving both de-differentiation and transdifferentiation (Fig. 1).

In transdifferentiation, a new developmental

programme is established before the original one is terminated. And so cells pass through a stage in which they show characteristics of two lineages. For example, during the process of C/EBP α -induced reprogramming of B and T cells to macrophages, cells pass through an intermediate stage^{7,8}. In a subset of these intermediate cells, macrophage characteristics are dominant and switch off mechanisms that maintain B- and T-cell characteristics.

Expression of a master regulator is not the only way a cell's fate can be altered; loss of these regulators may also result in cellular plasticity. Whether this takes place through a trans-differentiation or a de-differentiation route is not known. For example, loss of Pax5 halts development of the B-cell lineage at the earliest (progenitor B cell) stage^{10,11}. In cell culture or after transplantation into irradiated hosts (whose normal blood-cell progenitors are destroyed), Pax5-deficient B-cell progenitors can mature into all blood lineages such as myeloid and T cells — except B cells^{10,11}. In mature B cells, inactivation of the Pax5 gene results in B-cell loss through an unknown mechanism¹².

Cobaleda *et al.*⁴ now show that, in mice, loss of Pax5 in mature B cells leads to a type of transplantable cancer known as malignant lymphoma in which blood cells show characteristics of progenitor cells. However, mature B cells are still present in the secondary lymphoid organs (such as lymph nodes and spleen) of these animals. When transplanted into mice that lack all lymphocytes (B cells, T cells and natural killer cells), B cells that lack Pax5 efficiently generate T cells by first reverting to pro-B cells. The authors confirm the 'durability' of the reprogrammed B cells by showing that, when purified pro-B cells from recipient mice are transplanted into secondary recipient mice, they can still support T-cell development. The newly acquired T cells in the recipient mice were found at all stages of development, with the overall frequency of each subpopulation showing its usual distribution. Importantly, the reprogrammed subpopulation of mature T cells was functional, responding to antigen stimulation.

The work of Cobaleda and colleagues clearly indicates that, in the context of Pax5-deficient B cells, cellular reprogramming takes place through de-differentiation. It also raises many questions. For example, is de-differentiation an essential step in cellular reprogramming? If so, must cells return to an uncommitted, 'ground' state, or is direct reprogramming

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