surface lakes could provide the tipping point, which, once reached, would trigger the break-up of an ice shelf. If this is true, many ice shelves could be exposed to an ever-increasing risk of break-up, given their already extensive lake coverage. However, such a threshold might not be reached if surface water can instead be efficiently exported from the ice shelf to the ocean through large river networks.

Various physical factors will determine which of the above processes dominates for individual ice shelves. For example, relatively flat topography and extensive snow coverage will encourage surface-water ponding, which is likely to increase instability (Fig. 1a). Conversely, steeper slopes and bare ice surfaces will encourage water flow and stream development, potentially offsetting some of this increased instability (Fig. 1b). The latter scenario is not currently accounted for by ice-sheet models, which assume that all meltwater is stored on top of ice shelves, making them increasingly unstable. For example, the results of these models suggest that some of Antarctica's major ice shelves — such as the Amery, Filchner–Ronne, Larsen C and Ross — will disintegrate after melt rates exceed 1.5 metres per year, in the next century[3]. However, Bell and colleagues' analysis of the surface topography of these four ice shelves puts this prediction into question.

These two studies suggest that the surface hydrology on Antarctica's ice shelves will play a crucial part in deciding their individual fates, and those of the outlet glaciers that feed them. However, the authors do not explicitly address the likely additional role of increased melting on the undersides of ice shelves caused by ocean warming[4]. Given that the Antarctic Ice Sheet contains enough ice to raise global sea levels by 60 m (ref. 13), identifying and quantifying the role of all surface and subsurface processes on the potential stability of ice shelves is becoming increasingly important. ■

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STRUCTURAL BIOLOGY

A receptor that might block itself

The structure of the angiotensin II type 2 receptor reveals a potential mode of self-blocking action. This might explain its lack of signalling, and opens up avenues of investigation into its function and role in disease. SEE ARTICLE P.327

CHRISTOPHER G. TATE

Throughout the human body, the coordination of cell activity by hormones and neurotransmitter molecules is mediated by a multitude of cell-membrane proteins known as G-protein-coupled receptors (GPCRs). These form the largest membrane-receptor family in humans, and are implicated in a range of disorders and diseases, including high blood pressure, migraine and cancer. It is essential for scientists to understand both the pharmacology and the structures of these receptors to develop new therapeutics. On page 327, Zhang *et al.*[1] describe the crystal structure of a particularly enigmatic GPCR, the angiotensin II type 2 receptor (AT_2R), which is a potential target for the treatment of cardiovascular disease[2]. Unexpectedly, its structure deviates from the conventional GPCR structure, a finding that might explain its unusual signalling behaviour.

The structures of GPCRs are highly evolutionarily conserved, despite having widely divergent amino-acid sequences[3]. Each GPCR has two main structural states: inactive,

50 Years Ago

Some elegantly simple results of Defendi, Ephrussi *et al.* … have provided strong support for the general hypothesis that cancer induced by virus results from the addition of genetic information.

Uncontrolled growth is the characteristic property that distinguishes all cancer cells from normal cells. Cancer cells are unable to regulate cell division and, since this characteristic is inherited, it is highly likely that it results from a genetic change. Before the discovery that DNA and RNA viruses can both transform normal cells into cancer cells, the hypothesis most frequently considered was that cancer is caused by the accumulation of somatic mutations leading to the loss of the function of some essential regulatory gene. With the discovery of cancer inducing or oncogenic viruses, an alternative hypothesis could be considered—that cancer results from the acquisition of genetic information. … it is equally possible that oncogenic viruses either cause a deletion of part of the cell genome or induce recessive mutations. The crucial question then is whether the genome of an oncogenic virus persists in transformed cells, perhaps incorporated into the chromosome as a prophage is in a bacterium.
From *Nature* 22 April 1967

100 Years Ago

To gain daylight by adjustment of the clock is a brilliant practical idea, but the present method of realising it by moving the hands of the clock is grossly unscientific, and should, I think, be changed for the alternative one.

Let the circular disc of the clock-dial be put in place by screws in curved slots. … when changing time, we should rotate the dial backwards and forwards respectively, leaving the hands untouched.
From *Nature* 19 April 1917