PALEOCLIMATE

Superlakes, Megafloods, and Abrupt Climate Change

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As concern about the magnitude and rate of future climate change looms, it becomes increasingly important to understand the mechanisms underlying past abrupt climate change events. A cold event that occurred 8200 years ago, although much less extreme than some events during the Ice Ages, is probably most amenable to detailed examination because it is the most recent such event.

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According to the ice-core record from Greenland, the abrupt cooling 8200 years ago was the largest climate excursion of the past 10,000 years (*1, 2*): The mean temperature dropped by about 5°C for about 200 years (see the figure, A), snow accumulation decreased sharply, precipitation of chemical impurities increased, and forest fires became more frequent. The event, which affected much of the Northern Hemisphere (*3–5*), appears to have been triggered by the sudden release of fresh water from a huge, glacier-dammed lake that had formed during the deglaciation of North America (*6*).

Changes in the volume and extent of the ice sheets that once covered much of North America directly influenced the freshwater balance of the North Atlantic and are implicated in many abrupt climate events of the past 100,000 years (*7, 8*). During the last Ice Age, when a kilometers-thick ice sheet covered most of Canada and parts of the northern United States, armadas of icebergs were episodically launched into the North Atlantic. The melting of this freshwater ice and the associated freshening of ocean surface waters are believed to have changed the strength of the oceanic thermohaline circulation (*9*), thereby causing abrupt climate changes.

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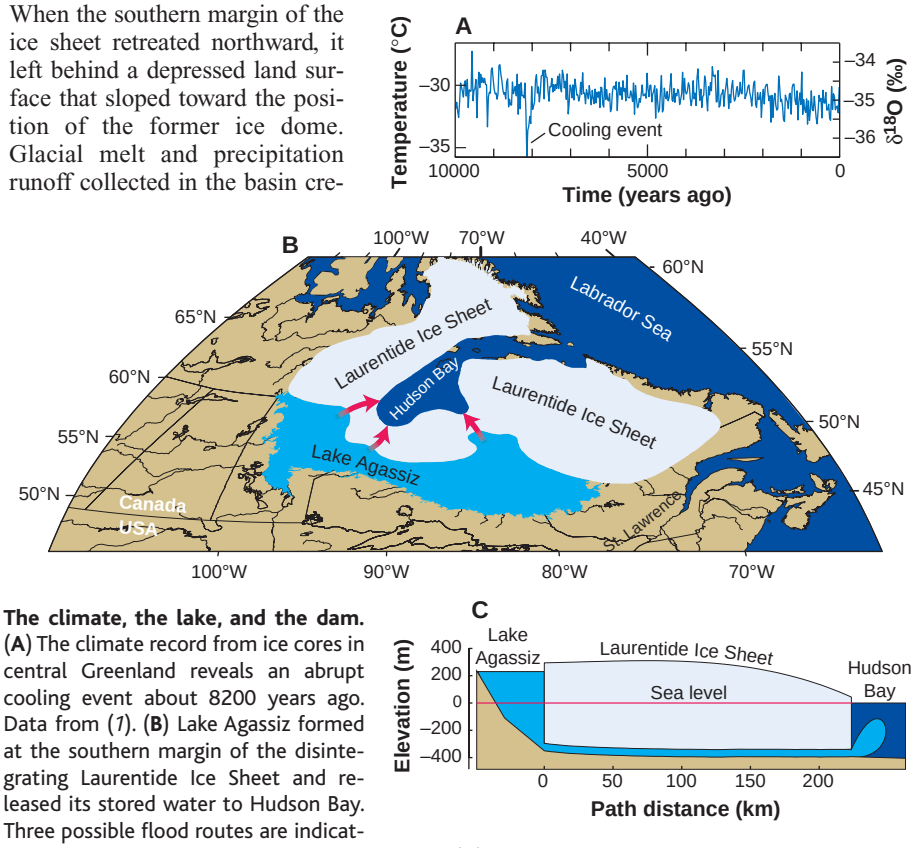
The deglaciation of North America produced large volumes of glacial meltwater, which also appears to have influenced the circulation of the North Atlantic. The Younger Dryas cooling event, which began about 12,700 years ago, is thought to have been triggered by an outburst of waters from a large ice-dammed lake and sustained by the redirection of meltwater from the Mississippi to the St. Lawrence Valley (*7, 10*). To explain the 8200-year cold event, a search for large sources of fresh water is thus a good starting point.

Around 8500 years ago, the Laurentide Ice Sheet, which at its maximum formed a 3-km-thick dome over Hudson Bay, was disintegrating rapidly. A marine calving bay extended into Hudson Bay from the Labrador Sea (see the figure, B). When the southern margin of the ice sheet retreated northward, it left behind a depressed land surface that sloped toward the position of the former ice dome. Glacial melt and precipitation runoff collected in the basin created by the sloping land surface and the ice barrier to the north. The estimated rate of inflow to this basin was ~0.1 sverdrup (1 sverdrup = 10^6 m^3 s^{-1}). Overflow waters from the resulting lake flowed through the St. Lawrence Valley into the North Atlantic.

Shortly before the Laurentide Ice Sheet finally disintegrated, the glacial lake—Lake Agassiz—had become a superlake (see the figure, B). Its maximum volume has been estimated as 163,000 km^3 (*11*)—at least double that of the largest contemporary lake, the Caspian Sea. The maximum elevation of the lake surface was fixed by a spillway about 230 m above sea level.

The ultimate release of Lake Agassiz waters to Hudson Bay was unavoidable. On the basis of radiocarbon dating, the outburst occurred 8450 years ago (*6*). A marine geophysical survey (*12*) provides evidence for high rates of water discharge in Hudson Bay associated with one or more outburst floods from the lake. The 8200-year cold event was thus most likely triggered by a flood of fresh water from superlake Agassiz that flowed northward through Hudson Bay into the North Atlantic.



The climate, the lake, and the dam. (**A**) The climate record from ice cores in central Greenland reveals an abrupt cooling event about 8200 years ago. Data from (*1*). (**B**) Lake Agassiz formed at the southern margin of the disintegrating Laurentide Ice Sheet and released its stored water to Hudson Bay. Three possible flood routes are indicated by red arrows. Many more routes are possible. (**C**) At the time of the flood, the ice dam was probably several hundred kilometers wide. Subglacial drainage from the lake to Hudson Bay would have started when the pressure of lake water approached that for flotation of the dam.

Modern analogs and the known physics of outburst flooding (*13*) indicate that tunneling below the ice is the most probable flood release mechanism (see the figure, C). Because ice floats on water, thinning ice dams are unstable. Initiation of a flood routed beneath the ice therefore preempts the possibility of a flood routed across the ice. Once a subglacial path is established, an ice-walled conduit will tend to grow by melting its walls. If the hydrostatic pressure of the ice enclosing the conduit exceeds the water pressure in the conduit, a creep closure process will also be active. Competition between melting and closure determines the progress of the flood.

Modern analogs to subglacial outburst floods from Lake Agassiz can be found in Iceland and elsewhere. However, in terms of the released water volume, the flood from superlake Agassiz is by far the largest known glacial outburst of the past 100,000 years. A physical model of subglacial outburst flooding (*13*) suggests that the maximum discharge of the flood was 5 to 10 sverdrups and that it lasted less than a year. There is geological evidence that this first flood was followed by a smaller one from a lower water level of ~125 m (*10*).

There are two possible explanations for these findings: (i) The reservoir was drained by two successive drops in lake level, and (ii) the first flood drained the reservoir to sea level, and the second flood occurred after the ice dam had been resealed and the reservoir partially refilled. Either way, once the dam had been permanently breached, the ~0.1 sverdrup discharge that formerly overflowed to the St. Lawrence Valley was routed northward to Hudson Bay.

Marine sediments provide clear evidence for the 8200-year cold event (*3*) but are equivocal about changes in ocean circulation that may have accompanied it. The response of the North Atlantic circulation to injection of fresh water into the Labrador Sea has been explored with a coupled ocean–atmosphere–sea ice model (*14*) in which a fixed volume of fresh water was released to the Labrador Sea at a steady rate over intervals of 10 to 50 years. All simulations show a weakening of the thermohaline circulation in the Nordic Seas in response to freshwater input. For some simulations, the recovery time is greater than 200 years. However, the released water volume in the model exceeds a recent estimate (*11*) of maximum lake volume by a factor of ~3, and the rate of release differs from the sharp pulse of less than 1 year duration suggested by flood modeling (*13*).

Much remains unknown about the 8200-year cold event. Further studies of how ice sheet margins, oceans, and vegetation zones were affected will help us to understand the cascade of responses that followed the initial outburst. Geological and geophysical studies in the Hudson Bay region and Labrador Sea could determine where the water release occurred and whether it took place as one or multiple events.

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VIROLOGY

Weapons of Mutational Destruction

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The remarkable array of defenses that cells use to fight viral infections is rapidly becoming more visible. An oft-proposed strategy for fighting viral infections is to design drugs that induce a high rate of mutation, potentially causing the viruses to succumb to "error catastrophe." A recent cluster of papers (*1*–*5*) describes a new function for a cellular protein called APOBEC3G that may act in just this way to block the replication of retroviruses such as human immunodeficiency virus (HIV).

Researchers have long recognized that the viral protein Vif is essential for replication of HIV in "nonpermissive" primary human $CD4^+$ T cells, as well as in some transformed T cell lines (*6*–*8*). But it remained unclear how Vif enables HIV to replicate in nonpermissive cells. Vif appeared to exert its effect in either the production or transmission of new virus particles (*9*–*11*). Although virus particles produced by nonpermissive cells in the absence of Vif appear to be physically indistinguishable from those produced in its presence, their ability to infect any host cell type is greatly diminished (*12*–*14*). Initial examination of the replication block indicated that HIV produced in nonpermissive cells without Vif could not efficiently complete reverse transcription of the viral RNA genome (*10*, *11*, *15*). Elegant experiments have shown that the nonpermissiveness of cells is a dominant effect and that Vif's ability to counteract it is species specific (*16*–*18*). These results and others implied the presence of a dominant factor in nonpermissive cells that reduces virus infectivity and that is counteracted by Vif.

In 2002, Sheehy and colleagues (*19*) identified this inhibitory protein as CEM15, later called APOBEC3G. This protein belongs to a family of nucleic acid editing enzymes related to APOBEC1, a cytidine deaminase that edits the apolipoprotein B messenger RNA (mRNA). Expression of APOBEC3G, normally restricted to nonpermissive cell types, converts permissive cells into nonpermissive cells. The APOBEC enzyme family deaminates specific cytidine (C) residues in either DNA or mRNA, converting them to uridine (U) residues (*19*, *20*). In addition to APOBEC1, the family includes the activation-induced cytidine deaminase (AID), a protein involved in the generation of antibody diversity. Expression of either APOBEC1 or APOBEC3G in *Escherichia coli* greatly increases the rate of C→T mutations in the DNA (*20*). This observation suggested that deamination of cytidines in either the HIV RNA genome or its DNA copy might mediate the antiviral effect of APOBEC3G. Such a mechanism would require that APOBEC3G targets retroviral particles and that Vif regulates this process. Both hypotheses have been borne out by a series of recent papers that appeared rapidly after the identification of APOBEC3G as the cellular target of Vif (*1*–*5*).

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