Blooms Like It Hot

Article *in* Science · May 2008

DOI: 10.1126/science.1155398 · Source: PubMed

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RNA pieces in the spliceosome, has a domain V counterpart, containing a 2-nucleotide bulge located 5 base pairs away from an AGC triad (*10*). Formation of an analogous metal-binding platform in this region of U6 (*11*) may explain the apparent ability of spliceosomal RNAs to retain catalytic activity in the complete absence of the many protein components that usually accompany splicing (*12*). A domain V-like element could have played a major role during the RNA world era of evolution, serving as the catalytic center for RNA cleavage, transesterification, and polymerization reactions.

The new structure provides a powerful starting point for future investigations of group II introns and the spliceosome. The lack of electron density for domain VI, which is important for the first step of splicing in many group II introns, and the absence of exons from the structure preclude us from seeing how these elements dock onto the surface created by domains I to V. Thus, the structural details of substrate recognition and catalysis remain undefined. The nature of the conformational change known to separate the two steps of splicing (*13*) also remains unclear. Finally, it will be important for our understanding of group II intron self-splicing to capture the structures of the other intermediates along the splicing pathway and to pursue experiments that link features of these structures with functionally defined interactions.

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10.1126/science.1156721

CLIMATE

Blooms Like It Hot

A link exists between global warming and the worldwide proliferation of harmful cyanobacterial blooms.

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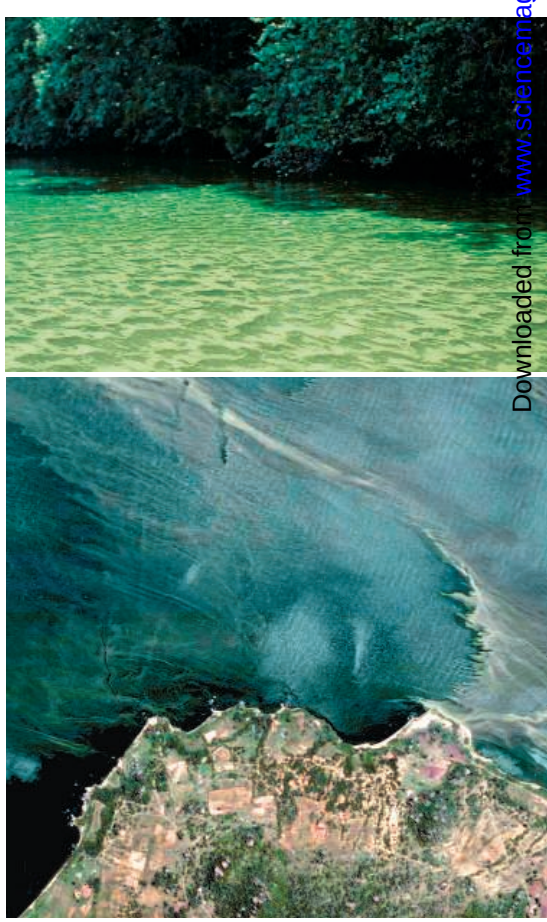
Nutrient overenrichment of waters by urban, agricultural, and industrial development has promoted the growth of cyanobacteria as harmful algal blooms (see the figure) (*1*, *2*). These blooms increase the turbidity of aquatic ecosystems, smothering aquatic plants and thereby suppressing important invertebrate and fish habitats. Die-off of blooms may deplete oxygen, killing fish. Some cyanobacteria produce toxins, which can cause serious and occasionally fatal human liver, digestive, neurological, and skin diseases (*1*–*4*). Cyanobacterial blooms thus threaten many aquatic ecosystems, including Lake Victoria in Africa, Lake Erie in North America, Lake Taihu in China, and the Baltic Sea in Europe (*3*–*6*). Climate change is a potent catalyst for the further expansion of these blooms.

Rising temperatures favor cyanobacteria in several ways. Cyanobacteria generally grow better at higher temperatures (often above 25°C) than do other phytoplankton species such as diatoms and green algae (*7, 8*). This gives cyanobacteria a competitive advantage at elevated temperatures (*8*, *9*). Warming of surface waters also strengthens the vertical stratification of lakes, reducing vertical mixing. Furthermore, global warming causes lakes to stratify earlier in spring and destratify later in autumn, which lengthens optimal growth periods. Many cyanobacteria exploit these stratified conditions by forming intracellular gas vesicles, which make the cells buoyant. Buoyant cyanobacteria float upward when mixing is weak and accumulate in dense surface blooms (*1*, *2*, *7*) (see the figure). These surface blooms shade underlying nonbuoyant phytoplankton, thus suppressing their opponents through competition for light (*8*).

Cyanobacterial blooms may even locally increase water temperatures through the intense absorption of light. The temperatures of surface blooms in the Baltic Sea and in Lake IJsselmeer, Netherlands, can be at least 1.5°C above those of ambient waters (*10*, *11*). This positive feedback provides additional competitive dominance of buoyant cyanobacteria over nonbuoyant phytoplankton.

Global warming also affects patterns of precipitation and drought. These changes in the hydrological cycle could further enhance cyanobacterial dominance. For example, more intense precipitation will increase surface and groundwater nutrient discharge into water bodies. In the short term, freshwater discharge may prevent blooms by flushing. However, as the discharge subsides and water residence time increases as a result of drought, nutrient loads will be captured, eventually promoting blooms. This scenario takes place when elevated winter-spring rainfall and flushing events are followed by protracted periods of summer drought. This sequence of



Undesired blooms. Examples of large water bodies covered by cyanobacterial blooms include the Neuse River Estuary, North Carolina, USA (**top**) and Lake Victoria, Africa (**bottom**).

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events has triggered massive algal blooms in aquatic ecosystems serving critical drinking water, fishery, and recreational needs. Attempts to control fluctuations in the discharge of rivers and lakes by means of dams and sluices may increase residence time, further aggravating cyanobacteria-related ecological and human health problems.

In addition, summer droughts, rising sea levels, increased withdrawal of freshwater for agricultural use, and application of road salt as a deicing agent have led to rising lake salinities in many regions. Several common cyanobacteria are more salt-tolerant than freshwater phytoplankton species (*12*, *13*). This high salt tolerance is reflected by increasing reports of toxic cyanobacterial blooms in brackish waters (*2*, *6*).

Some cyanobacteria have substantially expanded their geographical ranges. For example, *Cylindrospermopsis raciborskii*—the species responsible for "Palm Island mystery disease," an outbreak of a severe hepatitis-like illness on Palm Island, Australia (*4*)—was originally described as a tropical/subtropical genus. The species appeared in southern Europe in the 1930s and colonized higher latitudes in the late 20th century. It is now widespread in lakes in northern Germany (*14*). Similarly, the species was noted in Florida almost 35 years ago and is now commonly found in reservoirs and lakes experiencing eutrophication in the U.S. southeast and midwest (*2*). It is adapted to the low-light conditions that typify eutrophic waters, prefers water temperatures above 20°C, and survives adverse conditions through the use of specialized resting cells (*14*). These bloom characteristics suggest a link to eutrophication and global warming.

More detailed studies of the population dynamics in cyanobacterial blooms are needed. For example, competition between toxic and nontoxic strains affects the toxicity of cyanobacterial blooms (*15*). Furthermore, viruses may attack cyanobacteria and mediate bloom development and succession (*16*). It is unclear how these processes are affected by global warming. What is clear, however, is that high nutrient loading, rising temperatures, enhanced stratification, increased residence time, and salination all favor cyanobacterial dominance in many aquatic ecosystems. Water managers will have to accommodate the effects of climatic change in their strategies to combat the expansion of cyanobacterial blooms.

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10.1126/science.1155398

DEVELOPMENT

Deconstructing Pluripotency

The requirements for reprogramming different somatic cell types to a pluripotent state may not be equivalent.

Anne G. Bang and Melissa K. Carpenter

In 2006, Yamanaka and colleagues (*1*) discovered that mouse fibroblasts could be reprogrammed to a pluripotent, embryonic stem (ES) cell–like state by the simple introduction of four transcription factors, Oct4, Sox2, Klf4, and c-Myc. This finding has since been reproduced (*2*–*6*) and extended to human fibroblasts using the same cocktail of genes (*7*, *8*) or one composed of Oct4, Sox2, Nanog, and Lin28 (*9*). These so-called "induced pluripotent stem cells" (iPS cells) appear similar to ES cells in that they can give rise to all the cells of the body and display fundamental genetic and morphologic ES cell characteristics (see the figure). The concept of an iPS cell brings together decades of work in the fields of ES cell biology and nuclear reprogramming that predicted it might be possible to impose pluripotency upon a somatic cell (*10*). iPS cells not only have the potential to produce patient-specific stem cells, but they also provide a platform to study the biology of pluripotency and cell reprogramming. In *Science* Express, Aoi *et al.* (*11*) broaden the application of iPS cell methodology to murine epithelial cell types, highlighting differences when compared with reprogramming of fibroblasts. And on page 97 of this issue, Viswanathan *et al.* (*12*) address the role of one of the reprogramming factors, Lin28, in regulating microRNAs (miRNAs) in ES cells. The findings of Viswanathan *et al.*, and recent work by Benetti *et al.* (*13*) and Sinkkonen *et al.* (*14*), advance our knowledge of the little-understood roles of miRNAs in ES cells. Collectively, these studies take us closer to understanding how ES cells maintain an undifferentiated, self-renewing, and pluripotent state, and to defining how pluripotency can be imposed on other cell types.

To date, fibroblasts and mesenchymal stem cells have been used to generate iPS cells (*1*–*9*). A next step is to determine whether other cell types are susceptible to reprogramming. Toward this end, Aoi *et al.* produced iPS cells from two epithelial cell populations, adult mouse hepatocytes and gastric epithelial cells, by expressing Oct4, Sox2, Klf4, and c-Myc. Like iPS cells generated from fibroblasts (iPS-fibroblast), those from primary hepatocytes (iPS-Hep) and gastric epithelial cells (iPS-Stm) were pluripotent and gave rise to adult and germline chimeras. However, iPS-Hep and iPS-Stm differ from iPS-fibroblast cells in several important respects, indicating that the dynamics of reprogramming may not be equivalent in these cell types. For instance, although c-Myc was used, iPS-Hep and iPS-Stm cell–derived chimeric mice did not display the c-Myc–dependent tumorigenicity observed in iPS-fibroblast–derived chimeric mice. In addition, iPS-Hep and iPS-Stm cells could be generated using less stringent selection conditions. Thus, epithelial cell types may be more prone to reprogramming than fibroblasts.

How do these differences inform us about the mechanism of reprogramming? Given that ES cells are an epithelial population, characterized by cell adhesion (mediated by the membrane protein E-cadherin), one possibility is that epithelialization is an event required

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