# Regulation of Atmospheric $O_2$: Feedback in the Microbial Feedbag

Lee R. Kump and Fred T. Mackenzie

What controls the oxygen content of the atmosphere? This fundamental question has been asked innumerable times in the last several decades without a fully satisfactory answer. From conceptual models (*1*), increasingly complex numerical models (*2*) have been created. The results reported by Van Cappellen and Ingall on p. 493 of this issue (*3*) build upon these earlier models and add a new wrinkle that focuses the hunt for the controlling process on the microbial world of marine sediments.

Given the few-million-year residence time of $O_2$ in the atmosphere, slight imbalances in the biogeochemical cycle of oxygen could have led, over geological time intervals, to large fluctuations in concentration. Catastrophic phenomena sensitive to extremes in oxygen levels, such as global wildfire or the extinction of large animals, would have occurred if $O_2$ concentrations had exceeded 30% (*4*) or had fallen to less than 10% or so. However, there is no geological evidence of prolonged global wildfires, although charred material is concentrated at the Cretaceous-Tertiary boundary, a major extinction horizon. Also, the sedimentary record of more typical charcoal accumulation is remarkably continuous; flames cannot be sustained below an oxygen concentration of around 12%; thus, atmospheric oxygen concentration has not dipped below this level in the last 400 million years (*5*). It appears that there are feedback mechanisms in the natural cycle of $O_2$ operating on land and in the ocean that prevent such large fluctuations from occurring. Van Cappellen and Ingall argue that the important feedback mechanism resides in the ocean and involves the dependence of nutrient recycling rates on the oxygen content of the deep sea.

Several nutrient and toxic elements affect the biological productivity (and thus rate of oxygen production) of the ocean, but nitrogen (N) and phosphorus (P) are usually considered most important. The growth of marine algae involves the incorporation of N and P into living tissue. Ingestion by animals leads to the packaging of this material into relatively dense fecal pellets. The settling of fecal pellets and other large organic aggregates through the water column transfers both carbon (C) and nutrients to the deep sea. Both N and P are preferentially regenerated by microbial degradation processes in the water column and are transported by upwelling waters moving to the surface, where they become available for biological uptake once again.

**Burning issue.** Can marine-based oxygen regulation prevent global conflagration? Too much oxygen, and the Earth's forests would have been consumed by wildfire; too little oxygen and fires are unsustainable. Nutrient recycling and retention by marine sediments may act as part of a feedback mechanism to control atmospheric oxygen concentration. [Photo: E. R. Degginger/Photo Researchers Inc.]

A small fraction of organic-bound P and N escapes regeneration, however, and is buried in seafloor sediment. Although N can be replenished by the atmosphere, P must ultimately come from the continents by way of rivers. Thus P is typically considered to be the element that limits the amount of organic matter that can be buried in marine sediments. Because the burial of organic matter represents net oxygen production (that is, an excess of oxygen production over consumption during respiration and decomposition), the P supply also limits oxygen production rates in the marine realm.

Following their earlier discovery that the efficiency of P removal with organic matter falls as the ocean becomes anoxic (*6*), Van Cappellen and Ingall have taken the next logical step, to explore the ramifications of this link between the P and C cycles for the regulation of atmospheric oxygen. They sidestep the difficult question of whether the overall rate of organic matter decomposition depends on the oxygen content of deep waters (*7*), instead focusing on the effect that preferential P recycling under anoxic conditions has on rates of organic burial and oxygen production. If the atmospheric oxygen concentration falls, less oxygen is stirred into the deep sea. The demand on $O_2$ generated by animals and microbes feeding on the rain of organic debris through the water column depletes the deep sea of all oxygen. Under anoxic conditions, P retention by sediments diminishes, and a large flux of P from the sediments to the ocean results. The C/P ratio of the buried organic matter increases, and thus, a higher rate of organic matter burial ($O_2$ production) can be supported by a given riverine supply of P. The overall response of the system is to restore the $O_2$ level, a negative feedback.

The authors construct a simple model of the linked C, P, and iron (Fe) cycles. They show that their proposed feedback is capable of damping large changes in $O_2$ concentration that would otherwise occur in response to such forcings as fluctuating rates of ocean mixing or increased rates of oxidative weathering during episodes of mountain building. They conclude that anoxia is unlikely to have persisted for periods greater than $10^8$ years in the geological past because of the efficacy of this feedback mechanism. However, its effectiveness depends in part on specific features of their model that require further investigation [see reference 16 in (*3*)].

The arguments are appealing, but important questions remain. Would this mechanism prevent global conflagration of the world's forests? There is no obvious sensitivity of their model to such a disaster. The seeming necessity of such feedback prompted one of us several years ago (*8*) to propose a land-based feedback, one in which the effect that wildfires have on the global P cycle is explicitly considered. Ironically, it was the inability of Ingall and Van Cappellen (*9*) to find data in support of this hypothesis that in a sense led to the current one, but the link to terrestrial ecosystems was lost in the process. Also, if the newly proposed mechanism prevents long periods of oceanic anoxia, how was oxygen regulation different more than 2 billion years ago, when the at-

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mosphere and deep sea seem to have been permanently anoxic? Philosophically, should we expect to find a single process that overwhelms all others in regulating atmospheric oxygen levels, or is it more likely that multiple feedback loops are working in concert to prevent large oxygen fluctuations?

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# A Docking Receptor for HDL Cholesterol Esters


Daniel Steinberg


Cholesterol and cholesterol esters, very hydrophobic molecules, are carried through the hydrophilic environment of the bloodstream in lipoproteins. Perhaps the most familiar, low density lipoprotein (LDL), delivers cholesterol and its metabolites to cells by binding to specific receptors on the cell surface. In this process of "holoparticle uptake," the entire LDL particle is bound, endocytosed, and ultimately delivered to lysosomes where degradation of both protein and lipid occurs (*1*). Although uptake of high density lipoprotein (HDL) into most tissues can probably occur by a similar mechanism, HDL also uses a more selective means of delivering its cargo: In certain cells, HDL attaches ("docks"), delivers some of its cholesterol esters (and perhaps other lipids), and then dissociates from the cell surface and continues to circulate in the blood, now as a partially lipid-depleted particle [(*2*); see figure]. A receptor for HDL that mediates this "selective cholesterol ester uptake" has been identified by Acton *et al.* (*3*) and is reported in this issue to be SR-BI, a previously reported cell-surface molecule (*4*).

Selective cholesterol ester uptake occurs both in vivo and in vitro (*5*–*9*). By labeling HDL with "trapped ligands" (molecules that cannot escape from the cells after endocytotic uptake and delivery to the lysosome), the selective uptake of cholesterol ester from HDL has been shown in the rat to occur primarily in liver, adrenal gland, and ovary (*5*–*7*). The rate of uptake of cholesterol ester in these tissues is two to seven times more rapid than the uptake of apoprotein A-I, the major HDL protein. In other tissues, the uptake rates of HDL and apoprotein A-I are equal (with the exception of the kidney, which in the rat filters lipid-unassociated apoprotein A-I into the glomerular fluid). Selective cholesterol uptake in vitro does not appear to depend strongly on the nature of the apoproteins in HDL (*8*). Furthermore, other nonpolar lipids in the lipid core of HDL can also be selectively transferred. Selective uptake does not require endocytosis (*9*), but the precise mechanism of transfer across the cell membrane into the cytoplasm is not known. Acton *et al.* (*3*) now show that murine SR-BI, originally cloned on the basis of its ability to bind modified lipoproteins (such as acetyl LDL and oxidized LDL) (*4*), also binds HDL and mediates selective cholesterol ester uptake in transfected Chinese hamster ovary cells. They show further that SR-BI in mouse is expressed almost exclusively in liver, adrenal gland, and ovary, precisely those tissues in which selective uptake of HDL cholesterol esters has been demonstrated in vivo. These findings strongly support the identification of SR-BI as an HDL receptor.

High density lipoprotein selectively delivers cholesterol esters to steroidogenic tissues, and SR-BI is almost certainly involved in this process. High density lipoprotein also serves another crucial purpose: It picks up excess free cholesterol from peripheral tissues, which do not have the capacity for HDL degradation or excretion. The free

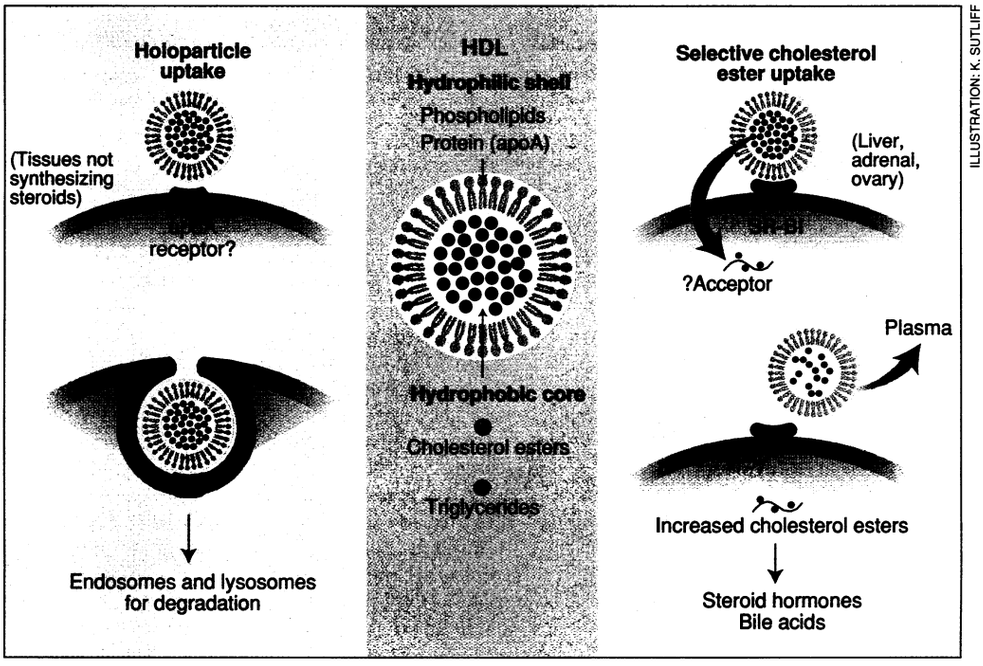


ILLUSTRATION: K. SUTLIFF

**Two ways to get cholesterol into a cell.**


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