PERSPECTIVES: CLIMATE CHANGE

Soot Takes Center Stage

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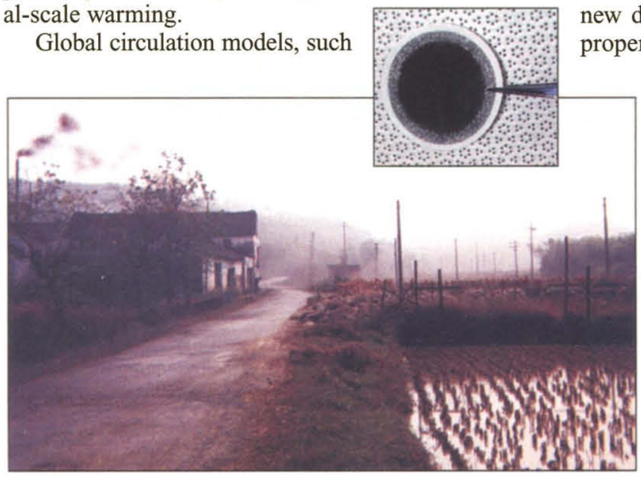
Earth's atmosphere is warming (*1*). Scientists believe the primary culprits to be pollutants that perturb Earth's radiative balance—the greenhouse gases. If so, then the best way to put the brakes on global warming is to curb pollutant emissions; but which pollutants?

Carbon dioxide (CO_2) has long been recognized as a greenhouse gas (*2*). Today, the cast of climate-changing pollutants has expanded to include other greenhouse gases such as methane, nitrous oxide, halocarbons, and ozone (*3*), as well as atmospheric aerosols (suspended particulate matter) commonly associated with air pollution and haze (*4*). Most aerosols cool the atmosphere by increasing Earth's reflectivity, but aerosols containing black carbon (BC)—that is, soot—warm it by absorbing sunlight (*5*). The contribution of BC to global warming may be substantial, perhaps second only to that of CO_2 (*6*, *7*).

On page 2250 of this issue, Menon *et al*. (*8*) point out that BC may also seriously perturb regional climate. Their model simulations suggest that BC emissions from China and India may be responsible for the increase in droughts in northeast China and flooding in southeast China in the summer observed during the last 20 years (*9*). Such calculations, coupled with evidence linking soot to adverse human health effects (*10*), strengthen proposals to target BC emissions to slow global warming in the near term (*6*, *7*).

BC warms the atmosphere, but it is not a greenhouse gas. First, it is a solid. Second, greenhouse gases warm by absorbing infrared or terrestrial radiation, whereas BC warms by absorbing sunlight. BC therefore has a slightly different climatic effect. Finally, unlike most greenhouse gases, BC is short-lived; its concentration varies by orders of magnitude from urban-industrial areas to remote locations. Its effect on climate should therefore also vary spatially.

In Menon *et al*.'s calculations, the spatially varying atmospheric heating caused by BC alters the Asian summer monsoonal circulation and causes the aforementioned change in precipitation patterns over China. The authors also predict an Asian BC-induced cooling over China, large warming over northern Africa, and cooling over the southern United States, all superimposed upon a more general global-scale warming.



A typical hazy day near Lin An, China. Black carbon emanates from a small brick factory. **(Inset)** Aerosol particles collected nearby on a 47-mm Teflon filter (~8 m^3 of air sampled). The blackness of the filter indicates the presence of black carbon.

Global circulation models, such as that of Menon *et al*., are not designed to simulate regional-scale climate variations (*1*, *11*). As the authors note, further studies are needed to confirm their detailed regional-scale predictions. But BC's entrance into the climate-change debate raises some intriguing scientific questions. If BC is a major warmer, are climate simulations that reproduce the temperature trend of the past century or so without including BC meaningful (*1*)? Given that BC's effects will likely be large in urban-industrial source areas, does the practice of eliminating urban-influenced data when calculating global temperature trends (to avoid the urban heat-island effect) mask the BC effect?

There are conundrums for policy-makers also. BC is produced through incomplete combustion of biomass, coal, and diesel fuel. Unlike CO_2 emissions, BC emissions are largest in developing countries (see table and figure) (on a per capita basis, BC emissions from China and the United States are roughly equivalent). Do the roles of the developed and developing world in global warming need to be reevaluated? Do global agreements and treaties need to be revised?

How could BC be included in international protocols on climate change? Greenhouse gases are evaluated by use of the global warming potential (GWP). But deriving a GWP for BC poses new difficulties. The absorptive properties of BC can change by a factor of 2 or more depending on its source and atmospheric processing. Thus, 1 Tg of BC from the United States will not necessarily have the same warming potential as 1 Tg of BC from, say, China.

An added complication arises from the uncertainty inherent in BC measurements. Two methods are generally used. The thermal method determines the organic carbon (OC) and elemental carbon (EC) content by heating and oxidizing aerosols collected on quartz filters and measuring the volatized carbon as a function of heating temperature (*12*). However, EC concentrations determined by independent labs with slightly different thermal analysis techniques can differ by a factor of 2 or more (*12*, *13*). Moreover, it is often assumed that EC is equivalent to BC, but this is not necessarily correct.

Optical techniques measure the aerosol absorption coefficient (*14*), which is often used to estimate BC assuming a mass absorption efficiency. BC mass concentrations estimated with this approach are, however, highly uncertain owing to uncertainties in the techniques and in the assumed value of the mass absorption efficiency, which varies with time and location.

There have been calls to develop a more

CO_2 AND BC ANNUAL EMISSIONS

	USA	Mexico	Germany	UK	Japan	China	India	Global
CO_2 (Tg C)	5,576	397	983	596	1,285	3,749	991	26,939
BC (Tg C)	0.32	0.08	0.05	0.04	0.09	1.2	0.56	7.00

1 Tg = 10^{12} g CO_2 emissions from http://arch.rivm.nl/env/int/coredata/edgar/. BC emissions from T. Bond and D. Streets (personal communication).

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rational, standardized methodology for measuring and characterizing EC and BC (*15*). In the meantime, models simulating the climate effect of BC must use emission inventories derived from a myriad of measurements by an unspecified combination of two poorly characterized, nonequivalent techniques. This makes for a hazy understanding of BC's climatic effect. Until these problems are addressed, the issue of BC's role in global warming and what to do about it will be difficult to resolve.

References and Notes

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PERSPECTIVES: MOLECULAR BIOLOGY

An RNA-Guided Pathway for the Epigenome

Thomas Jenuwein

Like twins separated by early evolution, DNA and RNA are the principal molecules for storing and transducing genetic information. Messenger RNAs (mRNAs) are transcribed from genes that constitute as little as 10% of the genome. Most of the remaining noncoding DNA is occupied by a diverse family of repeats including arrays of satellite sequences and transposons. Since the pioneering work of Muller (*1*) and McClintock (*2*), these apparently "nonspecific" sequences have been thought of as "epigenetic elements" that control the stability of gene expression programs and organize heterochromatic domains at centromeres and telomeres (*3*). Although this concept has been instrumental in guiding epigenetics research (*4*), it has also posed a major paradox: How can stretches of DNA that appear so heterogeneous in sequence organize similar higher order chromatin structures (*5*)?

A recent flurry of papers published in *Science* and *Cell* have started to unravel this riddle by demonstrating the presence of short RNA transcripts that are complementary to repeats in centromeres and elsewhere in the genome (*6–9*). These short transcripts resemble small interfering RNAs (siRNAs) that bind to complementary RNA sequences (*6*), targeting them for destruction. In the fission yeast *Schizosaccharomyces pombe*, mutants of the RNA interference (RNAi) machinery impair the definition of heterochromatin (*7*, *8*). Analogous mutants in the ciliate protozoan *Tetrahymena thermophila* affect the most dramatic reshaping of the genome, the directed elimination of DNA sequences (*9*). These exciting findings suggest that short heterochromatic RNAs (shRNAs) (*10*) are among the primary signals required for converting nonspecific sequence information into distinct chromatin states, thereby regulating the plasticity of the "epigenome." (The epigenome can be defined as a collection of biochemical modifications to chromatin that indexes genetic information.)

Heterochromatin in a wide variety of organisms is characterized by high density methylation of H3-K9 (that is, methylation of histone H3 protein at amino acid lysine 9). This modification results in binding of the chromo domain protein HP1 (heterochromatin protein 1) to chromatin (*11*). In turn, H3-K9 methylation directs DNA methylation in the fungus *Neurospora crassa* (*12*) and in the weed *Arabidopsis thaliana* (*13*). In plants, DNA methylation of integrated transgenes is thought to depend on complementary RNA signals (*14*, *15*). These links prompted Volpe and colleagues (*7*) to investigate whether *S. pombe* mutants with defects in the RNAi machinery had altered H3-K9 methylation. They disrupted the genes encoding the *S. pombe* homologs of proteins involved in RNAi: dicer (*dcr*), RNA-dependent RNA polymerase (*RdRp*), and argonaute (*ago*). Deletion of these genes resulted in the transcriptional derepression of transgenes integrated at pericentric repeats in the outer *cen* region of centromeres. It also resulted in loss of H3-K9 methylation and the association of the Swi6 protein (the *S. pombe* HP1 homolog) with this chromatin domain. Importantly, the *dcr*, *RdRp*, and *ago* mutants displayed aberrant accumulation of complementary RNA transcripts (1.4 and 2.4 kb) from both strands of the outer *cen* region (*7*). These results suggest that double-stranded RNA (dsRNA) originating from the pericentric repeats triggers the nuclear production of shRNAs, which in turn initiates H3-K9 methylation and heterochromatin formation. Indeed, ~22-nucleotide long RNAs with 5′-phosphate and 3′-hydroxyl groups, complementary to both strands of the outer *cen* repeats present at all three *S. pombe* centromeres, were detected in wild-type cells (*6*).

If DNA repeats represent nuclear foci for RNAi-like processing of dsRNA and the subsequent initiation of heterochromatin formation, then this model predicts that almost any repetitive DNA transferred to an ectopic (nonphysiologic) site should induce a heterochromatic state. In an elegant series of experiments reported on page 2232 of this issue, Hall and colleagues (*8*) inserted a *cen*-related DNA fragment (a 3.6-kb *cenH* repeat) normally present at the *S. pombe* silent mating–type (*mat*) domain in a euchromatic position (*ura4* locus). This repeat was sufficient to initiate variegated silencing of a linked reporter gene and also induced H3-K9 methylation and recruitment of Swi6.

CenH-dependent heterochromatinization at this ectopic site was again abrogated in *dcr*, *RdRp*, and *ago* mutants. Additionally, Hall *et al.* provide evidence that the RNAi machinery is required for the initiation but not the maintenance of the heterochromatic state. This is suggested by the persistence of H3-K9 methylation at endogenous *cenH-mat* sites in mutant strains with abrogated RNAi. Furthermore, segregant analysis of offspring from diploid strains, which are heterozygous for the H3-K9 histone methyltransferase (HMTase) *clr4* but null for RNAi mutants, revealed that these cells fail to establish H3-K9 methylation at *cenH-mat*. Complementary data were obtained after treatment of mutant cells with a histone deacetylase inhibitor, which results in hyperacetylation of chromatin and transcriptional derepression of silent *cenH-mat* epialleles; rapid

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