CO₂ limits may initially worsen warming

Policies designed to control fossil-fuel emissions might temporarily hasten the greenhouse warming before ultimately limiting the global temperature rise, according to calculations by climate researcher Tom Wigley. Yet that possibility should not deter efforts to control greenhouse-gas emissions, he says.

Wigley, of the University of East Anglia in Norwich, England, says the real message of his findings is that success will not come easily. "It might take decades for even a strong policy to produce some noticeable response," he says.

Wigley's calculations spotlight a highly uncertain arena in climate-change scenarios: the influence of sulfur dioxide (SN: 8/25/90, p.118). Like carbon dioxide, sulfur dioxide is produced by the combustion of fossil fuels. But while carbon dioxide gas traps heat, sulfur dioxide turns into tiny sulfate droplets that reflect sunlight back toward space. These sulfate "aerosols" also cool the Earth's surface indirectly by making clouds more reflective.

Scientists don't know the strength of such cooling effects, especially the effect on clouds. But if sulfate aerosols have an important influence, policies that limit fossil-fuel use would exert two opposing forces on the climate by reducing emissions of both the warming gas and the cooling gas.

To investigate the outcome of that tugof-war, Wigley calculated how various pollution controls would affect the carbon dioxide "forcing" and the sulfate aerosol "forcing." His study, detailed in the Feb. 7 NATURE, represents the first attempt to quantify the impact of both direct and indirect aerosol effects.

Because carbon forcing appears to dominate aerosol forcing, a policy that cuts emissions would eventually limit a temperature rise. But Wigley found that the aerosol effect would delay the climate's response to any emissions control strategy and would reduce the overall effectiveness of such policies.

Since the cooling power of sulfate aerosols remains unknown, Wigley tested a range of cases. In a scenario where aerosols exerted considerable effect, fossilfuel limitations enhanced greenhouse warming for more than three decades before beginning to slow the temperature rise. That's because carbon dioxide stays in the atmosphere for more than 100 years, while aerosols fall out within days. Thus, controls would rapidly reduce the aerosol cooling, and only later begin to curb the carbon dioxide warming, he found.

Although the sulfate aerosol effect might appear to represent an ameliorating force, "it cannot be considered to be a good thing," Wigley maintains.

Because industrial centers in the

Northern Hemisphere produce the most sulfur pollution, the aerosol effect could throw the world's climate off balance by cooling the north more than the south. Although this might limit an increase in average global temperatures, the hemispheric imbalance could significantly alter weather patterns around the world, possibly producing a situation "as severe as what we might be heading for with the plain greenhouse effect," says Wigley.

Atmospheric scientist Robert J. Charlson agrees. "It would be a fundamental mistake to think that the aerosols in any

way balance the greenhouse forcing," he says.

Charlson, of the University of Washington in Seattle, views aerosols' influence on clouds as a priority for future climate research. Investigators must study not only pollution-generated aerosols but also natural ones, he says.

Wigley adds, however, that unanswered questions about aerosols should not hold up negotiations on an international climate treaty, which formally began in Chantilly, Va., last week. Rather, he says, "the possible effects of fossil-fuel-derived sulfate aerosols should be seen as further reason for implementing controls on fossil-fuel use." — R. Monastersky

Genetic markers improve colorectal screen

A rare genetic defect causes tiny polyps—sometimes hundreds of them—to grow in the large intestine. Without surgery to remove the polyp-ridden section of the colon, cancer eventually results. Now researchers have developed a genetic test to predict a person's risk of developing the precancerous condition, known as familial adenomatous polyposis (FAP).

People who have a parent with FAP run a 50-50 chance of inheriting the gene and developing the disorder, which often emerges during adolescence. To detect FAP in time to stop its progression to cancer, physicians typically begin checking for polyps when youngsters in families carrying the gene reach their teens, repeating the exams annually and watching for additional symptoms such as bony growths on the head.

Used in conjunction with standard diagnostic methods, the new genetic screen for members of FAP-prone families should reduce the need for frequent colorectal exams among those who don't inherit the gene while increasing the chance of identifying those who do, says study leader Malcolm G. Dunlop of the Medical Research Council Human Genetics Unit in Edinburgh, Scotland.

Scientists have yet to find the gene for FAP, but they have found a number of DNA probes, or "markers," positioned on the chromosome very close to it. Dunlop and his colleagues, who describe their work in the Feb. 9 LANCET, used six previously identified markers to screen blood samples from 41 members (including children, teens and adults) of seven families with a history of FAP.

The screen revealed a high risk of FAP in four adults who had stopped having annual colon exams in their 30s or late 20s because their results had consistently come up negative. Upon subsequent examination, one showed cancerous growths and the other three showed polyps that probably would have progressed to cancer if not detected, Dunlop says. All four underwent colorectal surgery.

For 18 of the study's 41 participants, the blood test indicated a very low probability of carrying the FAP gene. "That's obviously pretty important, because that means they don't have to be [examined] so often," Dunlop says.

The researchers suggest that people from affected families who show a low risk on the genetic test and also test negative for clinical symptoms at age 15 need undergo only one additional exam at about age 30. Teenagers showing a moderate genetic risk should receive colorectal exams every two to three years, they recommend, while high-risk test results call for yearly exams.

Dunlop cautions, however, that the six markers yielded inconclusive results for nearly a third of the study group. Until researchers improve the technique's accuracy by finding markers for additional DNA sequences linked to the FAP gene, physicians using the test must combine it with clinical exams, he says.

Scientists could develop a better screen for FAP by identifying the DNA sequence of the culprit gene. But even then, the linked markers used in Dunlop's study would "undoubtedly prove useful," says Bert Vogelstein of the Johns Hopkins School of Medicine in Baltimore, who directs a search for the gene. Because the FAP gene's apparently large size allows for many defect sites, researchers could never be sure that their DNA probes uncovered every possible glitch along its length — but by incorporating markers near the gene, they could boost the odds of detection, Dunlop says.

Although FAP is uncommon — underlying fewer than 1 percent of all colon cancers — colorectal cancer itself ranks as the second leading cancer in the United States. Understanding the genetics of FAP may help clarify the causes of colon cancer in people who have not inherited the defect, Dunlop suggests. "The exciting thing," he says, "is that this gene might be involved in ordinary [colorectal] cancer," perhaps by mutating at some later point in life. — W. Gibbons

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