temperature ranges. Clearly, there are problems with such a procedure. Also, care must be taken in using evidence from past warm epochs, where various causal aspects of the warming have been somewhat different, to predict the effects of greenhouse warming on the ocean ecosystem. The dynamic ocean processes that determine the temperature distributions could be fundamentally altered.

In the absence of counteracting effects (*21*), intensified upwelling would tend to enhance primary organic production in these systems. But whether this increased primary production would be channeled to trophic components that society particularly values is unclear. There has been little clear demonstration that increased primary production actually promotes reproductive success and population growth of commercial fishes (*19, 22*). For example, increased production might be channeled to the mesopelagic fish communities that are diffused over wide areas and thereby largely lost from the neritic ecosystem. In addition, increased organic production might cause large areas of these systems to become anoxic at depth (*23*) and thereby promote sedimentation of unoxidized organic matter on the sea floor. In any case, if primary production increases, the rate at which carbon is sequestered beneath the ocean thermocline should likewise increase, and thus the rate of buildup of CO_2 in the atmosphere should be reduced.

If greenhouse warming leads to less global temperature contrast between tropical and polar regions, ocean basin–scale atmospheric and ocean circulations might slow down (*24*). However, as this example indicates, there is a competing tendency toward intensification where oceanic-continental temperature contrasts are involved. Many of the consequences of global climate change to marine ecosystems and also to marine-influenced terrestrial systems could depend on the relative importance, in each local situation, of these competing effects.

REFERENCES AND NOTES

1. R. H. Parrish, A. Bakun, D. M. Husby, C. S. Nelson, in (*2*), pp. 731–777.
2. G. D. Sharp and J. Csirke, Eds., *Proceedings of the Expert Consultation to Examine Changes in Abundance and Species Composition of Neritic Fish Resources* (*Fish. Rep. 291*, Food and Agriculture Organization of the United Nations, Rome, 1983).
3. J. H. Ryther, *Science* **166**, 72 (1969); D. Cushing, *Fish. Tech. Pap. 84* (Food and Agriculture Organization of the United Nations, Rome, 1969).
4. G. T. Trewartha, *An Introduction to Climate* (McGraw-Hill, New York, 1968).
5. A. Bakun, *NOAA Tech. Rep. NMFS SSRF671* (1973).
6. V. Ramanathan, *Science* **240**, 293 (1988).
7. J. E. Mason and A. Bakun, *NOAA Tech. Memo. NMFS-SWFC67* (1986).
8. M. M. Holl and B. R. Mendenhall, *Fleet Numeric. Weather Cent. Tech. Note 72-2* (FNOC, Monterey, CA, 1972).
9. D. A. Cole and D. R. McLain, *NOAA Tech. Memo. NMFS-SWFC125* (1989).
10. W. S. Wooster, in preparation; R. R. Dickson, P. M. Kelly, J. M. Colebrook, W. S. Wooster, D. H. Cushing, *J. Plankton Res.* **10**, 151 (1986).
11. H. Belvèze and K. Erzini, in (*2*).
12. W. S. Wooster, A. Bakun, D. R. McLain, *J. Mar. Res.* **34**, 131 (1976).
13. A. Bakun, in *The Peruvian Anchoveta and Its Upwelling Ecosystem: Three Decades of Change*, D. Pauly and I. Tsukayama, Eds. (International Center for Living Aquatic Resources Management, Manila, Philippines, 1987), pp. 46–74; A. Bakun and R. Mendelssohn, in (*14*).
14. D. Pauly, P. Muck, J. Mendo, I. Tsukayama, Eds., *The Peruvian Upwelling Ecosystem: Dynamics and Interactions* (International Center for Living Aquatic Resources Management, Manila, Philippines, in press).
15. M. Torn, in preparation.
16. W. Wooster, D. Fluharty, Eds., *El Niño North* (University of Washington Sea Grant Program, Seattle, 1985).
17. J. Picaut, *Calif. Coop. Oceanic Fish. Inves. Rep.* **26**, 41 (1985).
18. L. Legendre and S. Demers, *Can. J. Fish. Aquat. Sci.* **41**, 2 (1984).
19. A. Bakun, in *L'Homme et les Ecosystems Halieutiques*, J. P. Troadec, Ed. (Institut Français de Recherche pour l'Exploitation de la Mer, Paris, 1990), pp. 155–187.
20. P. Cury and C. Roy, *Can. J. Fish. Aquat. Sci.* **46**, 670 (1989).
21. F. P. Chavez, R. T. Barber, M. P. Sanderson, in (*14*).
22. R. Lasker, in *Toward a Theory of Biological-Physical Interaction in the World Ocean*, B. J. Rothschild, Ed. (Kluwer, Dordrecht, 1989), pp. 173–182.
23. G. A. Jackson *et al.*, *Eos* **70**, 146 (1989); M. S. Quimby-Hunt, P. Wilde, W. N. E. Berry, paper presented at the First Workshop on Global Climate Change and Its Effects on California, University of California, Davis, 10 to 12 July, 1989.
24. On the other hand, the trade winds in the tropical Pacific likewise seem to have increased during this same period [K. D. B. Whysall, N. S. Cooper, G. R. Bigg, *Nature* **327**, 216 (1987)]. Whether seasonally increased alongshore winds along the continental boundaries (equatorward along the eastern ocean boundary, poleward along the western boundary) resulting from enhanced onshore-offshore temperature contrasts could contribute to an increased trade wind circulation is a matter for further study.
25. Indicated significance levels are taken directly from standard tables of the Student's *t* distribution, without adjustment for residual autocorrelation.

11 August 1989; accepted 13 November 1989

Ozone Control and Methanol Fuel Use

A. G. RUSSELL,* D. ST. PIERRE, J. B. MILFORD

Methanol fuel use in motor vehicles and stationary combustion has the potential to improve air quality. A modeling study of methanol fuel use in Los Angeles, California, shows that the low chemical reactivity of methanol vapor slows ozone formation and would lead to lower ozone concentrations. Predicted peak ozone levels decreased up to 16 percent, and exposure to levels above the federal standard dropped by up to 22 percent, when pure (M100) methanol fuel use was simulated for the year 2000. Similar results were obtained for 2010. Use of a gasoline-methanol blend (M85) resulted in smaller reductions. Predicted formaldehyde levels and exposure were not increased severely, and in some cases declined, in the simulations of methanol use.

OVER HALF OF THE PEOPLE IN THE United States live in areas that experience ozone levels above the limits set by the National Ambient Air Quality Standards (NAAQS). Compliance with the standard is proving to be a difficult task. One measure that has been proffered as a solution is the use of alternative, "clean" fuels in motor vehicles. Federal, state, and local agencies are promoting, and have suggested mandating, use of clean fuels in order to reduce ozone and other components of urban smog (*1*). In particular, the use of alternative fuels is integral in the recent plan adopted for the Los Angeles, California, area, and the President's recent proposals would extend the use to other areas. Of the fuels considered, methanol appears to be one of the most feasible for widespread use and improving air quality (*2, 3*). Several studies have shown that methanol, because of its low atmospheric chemical reactivity, could be effective in reducing the formation of photochemical smog and ozone (*3, 4*). The extent of improvement, and whether significant improvement would be realized at all during multiday smog episodes, has been brought into question by recent experiments (*5*). Because methanol-fueled vehicles (MFVs) emit more formaldehyde than their gasoline-fueled counterparts, however, concern has also arisen over the possibility of increased ambient levels of formaldehyde. To evaluate better these concerns and to establish a scientific basis for future strategies, we have used a three-dimensional, Eulerian, photochemical air quality model to investigate how the use of methanol fuel would affect air quality. In this report, we use the model results to address (i) the impact of methanol on atmospheric chemi-

A. G. Russell, Department of Mechanical Engineering, Carnegie Mellon University, Pittsburgh, PA 15213.
D. St. Pierre, Lowry Air Force Base, Denver, CO 80230.
J. B. Milford, Department of Civil Engineering, University of Connecticut, Storrs, CT 06269.

*To whom correspondence should be addressed.

cal formation of pollutants; (ii) whether methanol buildup during multiday episodes would negate the benefits of its lower photochemical reactivity; (iii) whether emissions from MFVs would severely increase ambient formaldehyde or negate ozone reductions; and (iv) the comparative benefits of the use of nearly pure methanol fuel (M100) versus M85 fuel (nominally, a blend of 85% methanol and 15% gasoline, by volume). Several technological and safety considerations still need to be overcome before M100 can be used. M100 has cold-start problems and the flame is nearly invisible in daylight. In practice, M100 may need to contain additives, which could degrade its reactivity benefits. In contrast, M85 is not as severely limited from safety and performance standpoints.

Model calculations were conducted simulating the extensive utilization of methanol fuel over 3 days in two future years, 2000 and 2010, in the South Coast Air Basin of California (SoCAB) (*6*). These years were chosen to allow for significant penetration of methanol into the vehicle fleet and because of the availability of detailed source emission projections. The SoCAB, which surrounds Los Angeles, was chosen because it (i) is more severely impacted by photochemical smog than any other area in the United States (ozone concentrations in excess of three times the national standard of 0.12 ppm for a 1-hour average); (ii) is likely to be the first area targeted for use of MFVs (*7*); and (iii) is ideal for the study of multiday pollution episodes. Although other areas are also impacted by the buildup of ozone over multiple days (*8*), the larger geographical domains and lower resolution of available input data compared to these in the SoCAB inhibit detailed modeling of those regions.

The modeling domain was a 250-km by 150-km region surrounding Los Angeles, resolved horizontally into 5-km by 5-km computational cells and vertically into five layers ranging from 39 to 430 m in height. Emissions and meteorological variables are similarly resolved. Meteorological conditions were set to those during 30 August to 1 September 1982 because those conditions were conducive to the formation of ozone and detailed supplemental data are available for model evaluation (*9*). The maximum ozone concentration recorded on 1 September 1982 was 0.35 ppm, a value approaching the highest levels recorded in recent years.

Table 1. Forecast emissions of NO_x and reactive organic gases in 2000 and 2010 in 10^3 kg per day. Values in parentheses are percent of total.

Source	NO_x	Reactive organic gas
	2000	
Motor vehicles	394 (53%)	261 (23%)
Stationary	350 (47%)	874 (77%)
Total	744	1135
	2010	
Motor vehicles	430 (50%)	389 (26%)
Stationary and off-road vehicles	436 (50%)	1107 (74%)
Total	866	1496

In the simulations, forecast emission inputs for 2000 and 2010 (*10*) were used (Table 1) for the base (nonmethanol) case. These inventory estimates account for controls expected to be in place to lower emissions from conventionally fueled vehicles (*11*). With these forecasts as a baseline, the emissions were perturbed to account for motor vehicle conversion to methanol. For example, emissions from passenger cars were set equal to the same standards as gasoline-fueled cars (*11*). In addition, HCHO emissions were set to the California standard of 9.3 mg km^{-1} (15 mg per mile). Recent tests indicate that these levels are achievable (*11*). Impacts on emissions from related sources, such as retail fuel distribution, were included, as was vehicle deterioration and the continued presence of old conventionally fueled vehicles (CFVs). In one simulation, we examined the use of methanol in stationary sources, such as power plants and stationary internal-combustion engines.

The simulations show that utilizing methanol fuel, either as M100 or as M85, would decrease ozone levels throughout the SoCAB. Three metrics are used to describe the degree of improvement in air quality. First, the maximum predicted concentration in the region is used to show how effective controls are at reducing the peak toward the national standard. However, the peak concentrations for the meteorological period simulated occur in the less populated eastern part of the SoCAB. In an effort to show the effect on potential human exposure, population projections (for the years 2000 and 2010) were combined with spatial and temporal variations in ozone concentrations. This population "exposure" is defined as

$$E_{C_0} = \int_A \int_T P(\bar{x})\, C(\bar{x}, t)\, H(C - C_0) dt\, d\bar{x}$$

where $P(\bar{x})$ is the population as a function of location, $\bar{x}$; $C(\bar{x}, t)$ is the predicted ozone concentration as a function of time, t, and location; and $H(C - C_0)$ is the Heaviside function, which is 1 if the predicted concentration is above a threshold concentration, C_0, and 0 otherwise. In essence, this is the population-weighted ozone exposure above a prescribed ozone concentration. We used two threshold concentrations (1-hour average): 0.12 ppm (the national standard) and 0.20 ppm (California first-stage health alert).

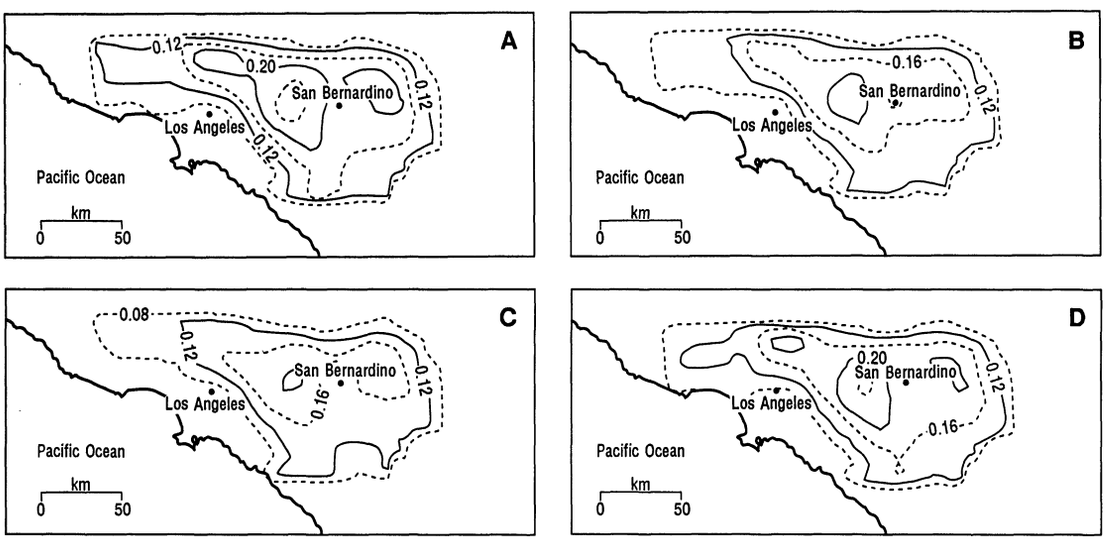

Fig. 1. Predicted ozone concentrations (in parts per million) at 1300 hours on the third day of the simulation. Base inventory corresponds to year 2000 emissions. Fields shown correspond to (**A**) Base case; (**B**) M100-fueled vehicle fleet; (**C**) no mobile source emissions; and (**D**) M85-fueled fleet.

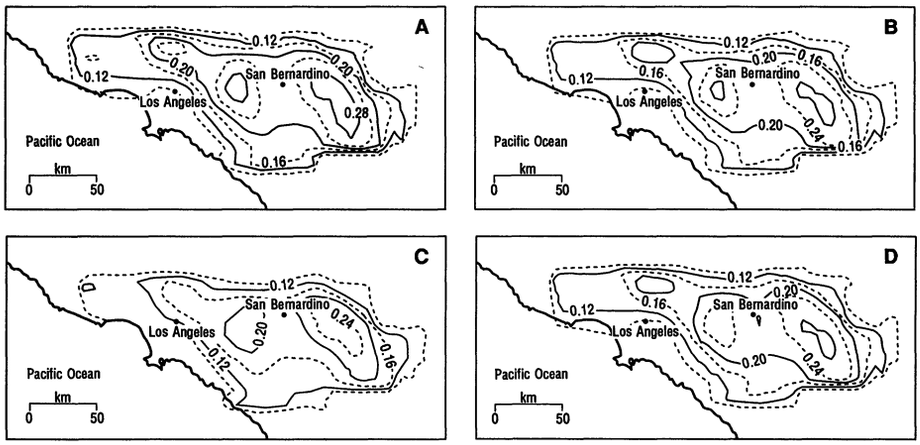

Fig. 2. Predicted ozone concentrations (in parts per million) at 1300 hours on the third day of the simulation. Base inventory corresponds to year 2010 emissions. Fields shown correspond to (**A**) Base case; (**B**) M85-fueled fleet; (**C**) no on-road mobile source emissions; and (**D**) M100-fueled vehicle fleet.

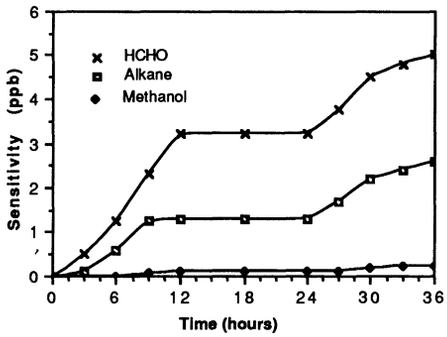

Fig. 3. Semi-normalized local sensitivity P_i ($\partial[O_3]/\partial P_i$) of ozone formation to source emissions of formaldehyde, alkanes, and methanol, over a 2-day simulation. Methanol is seen to be relatively unreactive whereas formaldehyde (HCHO) is quite reactive and an efficient promoter of ozone formation. For comparison, the sensitivity to alkane, the most abundant class of reactive organic gases, is also shown.

Peak ozone concentrations and population exposure indices for 2000 and 2010 were determined for four simulations in each future year (Table 2 and Figs. 1 and 2): (i) the "base case" corresponding to a conventionally fueled fleet; (ii) "M100" corresponding to 100% penetration of M100 fueled vehicles, starting with new vehicles in the year 1990; (iii) "M85" representing 100% penetration of M85 fueled vehicles from 1990 on; and (iv) "no mobile" in which emissions from on-road motor vehicles are eliminated. In (ii) and (iii), pre-1990 CFVs contribute to the mobile source emissions and thus decrease the apparent effectiveness of conversion.

Dramatic improvements were not predicted relative to the base case, even when mobile sources were removed (Table 2 and Figs. 1 and 2). This result is attributable to the decrease in on-road motor vehicle emissions that is expected to occur as a result of increasingly stringent regulations whether or not methanol is used. A somewhat smaller fraction of the forecast reactive organic gas (ROG) emissions in future years is due to mobile sources. In 2000, 23% of the ROG is from on-road sources, and in 2010, 26% is attributable to those sources. Moreover, because of the continued presence of older CFVs and out-of-state vehicles, only a fraction of the emissions are displaced by methanol use. As an example of the maximum impact that can be expected from controlling motor vehicles, totally removing on-road emissions decreases peak ozone by only 21% in 2000 and 13% in 2010. Part of that reduction is attributable to lower NO_x emissions (*12*).

Relative to reductions from elimination of on-road motor vehicle emissions, the simulation of M100 fuel use shows peak ozone and exposure decreases of, respectively, 69% and 88% in 2000, and 33% and 91% in 2010. Peak ozone reduction is less sensitive to methanol use than is exposure, because the peak occurs in the eastern part of the basin where ROG controls have less effect than in the more densely populated, central part of the Basin. This trend is particularly evident for 2010. Conversion to M85 resulted in less improvement than M100 because of the more volatile and reactive gasoline it contains. M85 gives about half the improvement expected with M100 fuel use (*13*).

That MFVs now emit more formaldehyde than comparable gasoline-fueled vehicles raises two issues: whether increased HCHO emissions would (i) substantially lessen the ozone-related benefits of methanol use, or (ii) lead to greatly increased exposure to HCHO. If either of these were the case, further controls to reduce formaldehyde emissions might be warranted. These issues were addressed with a "high HCHO" calculation simulating the use of M100 vehicles with an HCHO emission rate of 34 mg km^{-1} (55 mg per mile, such emissions might occur if controls are not as effective as planned), and the earlier M100 calculation with the nominal HCHO emission rates of 9.3 to 14 mg km^{-1} (15 to 23 mg per mile). The results show that, even with the higher HCHO emissions, ambient HCHO exposure (calculated as in Eq. 1 with C_0 = 0.0 ppb) increases no more than 15%. In some of the standard methanol cases, HCHO exposure declines. The reason for the decline, despite increased direct emissions, is that 80% or more of the ambient HCHO is produced by atmospheric photo-oxidation of other organics (*14*). The low reactivity of methanol reduces atmospheric formation of HCHO and leads to the apparent decrease. The predicted maximum HCHO concentrations, about 30 ppb in most calculations, are about one third of the level that would affect most sensitive individuals and are similar to

Table 2. Predicted peak ozone, ozone exposure, and formaldehyde exposure for 1 September for the expected population distributions in 2000 and 2010. Simulation descriptions and definition of exposure are given in the text. For ozone exposure and peak, the values in parentheses are the reductions relative to removing mobile source emissions of NO_x, ROG, and CO. The value in parentheses following formaldehyde exposure is relative to the base calculation.

	Peak $[O_3]$ (ppm)	O_3 and HCHO exposure (person-ppm-hours)		
		$[O_3]>0.12$ ppm	$[O_3]>0.20$ ppm	[HCHO]>0.0 ppm
		2000		
Base case	0.26 (0)	7314 (0)	2586 (0)	2779 (1)
M85	0.24 (0.30)	6167 (0.60)	1260 (0.52)	2551 (0.92)
M100	0.22 (0.69)	5673 (0.5)	364 (0.88)	2326 (0.84)
No mobile	0.21 (1.0)	5384 (1.0)	58 (1.0)	1578 (0.57)
High HCHO	0.24 (0.35)	6070 (0.64)	1108 (0.58)	3203 (1.15)
		2010		
Base case	0.32 (0)	9840 (0)	5159 (0)	3071 (1.0)
M85	0.31 (0.31)	8700 (0.56)	4234 (0.29)	3328 (1.08)
M100	0.31(0.33)	7976 (0.91)	3823 (0.42)	3478 (1.13)
No mobile	0.28 (1.0)	7804 (1.0)	1950 (1.0)	2082 (0.68)

recently observed levels (*15*). The 1-hour average methanol concentrations peaked at 0.7 ppm in the calculations, well below the level of toxicity; this result suggests that ambient methanol buildup and resulting exposure are of minor concern (*16*).

Use of methanol in stationary combustion sources can reduce NO_x emissions. Applications include over-firing in utility boilers and use as the primary fuel in stationary internal combustion engines. A separate simulation was conducted to determine the possible impact of stationary-source use. The effect of the potential 7% reduction in NO_x emissions was a 2% reduction in peak O_3.

Conversion to methanol would not be expected to exacerbate ozone concentrations in downwind regions. Predicted methanol concentrations 1 day downwind of the urban area were less than 0.1 ppm. At this level, methanol would add little to the reactivity of the ambient organic gases. Farther transport would significantly decrease methanol levels because of vertical and horizontal dispersion. With transport, NO_x is lost more quickly from the system than ROG, and thus ozone formation is primarily dependent on the availability of NO_x and less so on ROG. In that methanol can reduce NO_x emissions from diesel-type engines and stationary sources, methanol use could lead to a decrease in downwind ozone concentrations. In those urban areas where NO_x-limited conditions prevail, the reactivity of the organics is of lesser importance and the benefits from methanol fuel substitution would be enhanced by lowered NO_x emissions from diesel-type engines and use in stationary sources.

In order to account for experiments that have shown diminished benefits of methanol substitution over multiday periods (*5*), in contrast to our modeling results, a series of calculations were performed simulating the smog chamber experiments. These simulations show that the declining effectiveness of methanol substitution over the course of the experiments may have resulted from relatively rapid depletion of NO_x and the presence of chamber wall artifacts. Sensitivity analysis, with the direct decoupled method (*17*), shows that methanol contributes little to ozone formation under typical urban conditions (Fig. 3). The 36-hour simulation was for typical urban conditions, except that M100 vehicles were assumed to contribute 40% of the ROG emissions. Sensitivity to HCHO is strong.

Use of methanol-based fuels would improve urban air quality by decreasing the reactivity of the organic gases emitted from motor vehicles. Detailed modeling indicates that the methanol fraction of the emissions contributes little to ozone formation, even over 3-day episodes. Controlling formaldehyde and gasoline-derived emissions is critical for deriving the full benefits of conversion to methanol. Use of methanol to reduce NO_x emissions in both heavy-duty vehicle applications and stationary sources is also predicted to reduce peak ozone. Thus, methanol can be used as a combined control on reactive organic gas and NO_x emissions.

Reduced atmospheric formation of HCHO from oxidation of reactive organic gases offsets the increased direct emissions, and this process leads to minor increases or possible reductions in ambient HCHO exposure. Predicted peak levels are similar to current concentrations and are significantly less than historic peaks. Likewise, on the basis of these simulations, multiday buildup of methanol should not pose a problem in terms of ambient exposure.

REFERENCES AND NOTES

1. *Air Quality Management Plan* (South Coast Air Quality Management District, El Monte, CA, 1988); Office of Technology Assessment, U.S. Congress, *Fed. Regist.* **54**, 14425, 11 April 1989; *ibid.* **40**, part 86, 29 August 1986; G. Bush, Proposed Amendments to the Clean Air Act, 22 July 1989.
2. R. O'Toole *et al.*, *California Methanol Assessment Study* (Jet Propulsion Laboratory, Pasadena, CA, 1983).
3. T. Chang *et al.*, *Atmos. Environ.* **8**, 1629 (1989).
4. G. Whitten *et al.*, *Impact of Methanol on Smog* (Systems Applications Incorporated, San Rafael, CA, 1983); J. Norbeck and R. Nichols, *Air Pollution Control Assoc. Rep. 85-383* (1987).
5. W. Carter *et al.*, "Effects of Methanol Fuel Substitution on Multiday Air Pollution Episodes" (report to California Air Resources Board, Sacramento, CA, 1986).
6. A complete description of the study is in A. Russell, *et al.*, "Quantitative Estimate of the Air Quality Impacts of Methanol Fuel Use" (final report to the California Air Resources Board, Sacramento, CA, 1989). The model used is a fully three-dimensional, photochemical transport model [G. McRae and J. Seinfeld, *Atmos. Environ.* **17**, 501 (1983)]. An extended and updated version of the chemical mechanism was used, which includes the methanol-OH reaction at a rate of 23 ppm^{-1} s^{-1}. In all, 36 simulations were conducted to document model sensitivity to input parameters. Although changing boundary and initial conditions had only a small effect, model results are quite sensitive to changes in HCHO and NO_x emissions. The ten cases discussed in the text best show the likely effects of extensive methanol fuel substitution.
7. *Air Quality Management Plan* (South Coast Air Quality Management District, El Monte, CA, 1988).
8. W. Chameides *et al.*, *Science* **241**, 1473 (1988); S. Schwartz, *ibid.* **243**, 753 (1989).
9. A. Russell and G. Cass, *Atmos. Environ.* **18**, 1815 (1984).
10. P. Allen, personal communication; L. Mahoney, personal communication.
11. Regulatory action is expected to continue to reduce automobile and stationary source emissions. Because of vehicle deterioration, compounded with less stringent regulations in earlier years, older vehicles are responsible for a large part of the vehicle emissions. Although 1990 is an infeasible target for the beginning of MFV introduction it was chosen to allow for a significant conversion of the vehicle fleet to methanol by the years 2000 and 2010 for which available emission estimates have been developed. Reactive organic exhaust emissions from light-duty automobiles were 0.36 g km^{-1} (0.59 g per mile) for 1990 to 1992, 0.21 g km^{-1} (0.35 g per mile) for 1993 to 1994, and 0.15 g km^{-1} (0.25 g per mile) for 1995 on. The base case is for the conventional organic speciation. For M100 simulations, the organics were speciated as methanol and HCHO emissions were set at 9.3 to 14.3 mg km^{-1} (15 to 23 mg of HCHO per mile); M85 vehicle emissions were 50% methanol and 50% gasoline-derived products, by mass, and HCHO emissions were set as for the M100 case. In both of the methanol simulations, the mass emission rate of methanol was calculated on an equivalent C basis; therefore, the molé exhaust emission rate of carbon is equal for methanol and nonmethanol simulations. Emissions of NO_x from MFVs were set equal to the equivalent size and age CFVs, except for heavy-duty compression ignition vehicles where the methanol-fueled engine is expected to emit half as much as diesel-fueled engines. In the 2010 simulations, only light-duty vehicle emissions were modified. Recent tests have shown that current MFVs emit comparable amounts of ROG, CO, and NO_x as we assumed in our study, and as similar gasoline-fueled vehicles [P. A. Gabele, *Characterization of Emissions from a Variable Gasoline/Methanol Fuel Car* (U.S. Environmental Protection Agency, Research Triangle Park, NC, 1989); "Alcohol-Fueled Fleet Test Program Eighth Interim Report" (California Air Resources Board, El Monte, CA, 1988); R. L. Williams *et al.*, *Formaldehyde, Methanol, and Hydrocarbon Emissions from Methanol-Fueled Cars* (GMR-6728, General Motors Research Laboratories, Warren, MI, 1989)]. Evaporative and CO emissions may be lower from an MFV. Meeting the in-use standard of 9.3 mg km^{-1} for formaldehyde appears to be a greater technological challenge. Tests by the California Air Resources Board indicated that HCHO emissions from newer vehicles are about 6 mg km^{-1} (10 mg per mile). Recent tests of a heated catalyst have demonstrated that HCHO emission reductions are >50% or 3 mg km^{-1}. Although these tests were for relatively new catalysts, they indicated that a standard of 9.3 mg km^{-1} is feasible, as has been suggested by automotive engineers involved in MFV development [E. Marshal, *Science* **246**, 200 (1989)].
12. Discussions of the benefits of NO_x control for reducing ozone can be found in W. Chameides *et al.*, *Science* **241**, 1473 (1988); J. Pitts *et al.*, *Environ. Sci. Technol.* **17**, 54 (1983); J. Milford *et al.*, *ibid.* **23**, 1290 (1989); and E. Meyer, *Review of Control Strategies for Ozone and Their Effects on Environmental Issues* (U.S. Environmental Protection Agency, Research Triangle, NC, 1986). Methanol use has been promoted primarily as a control on ROG reactivity, although use in some applications will also reduce NO_x. In our study, reducing NO_x lowered peak ozone levels.
13. The gasoline components in M85 evaporate preferentially over methanol. Also, about half the exhaust emissions are assumed to be similar in composition to that from gasoline-fueled vehicles. The higher reactivity of the gasoline-type emissions decreases the benefits of M85 as compared to M100. Recent experiments indicate that emissions from M85-fueled vehicles may be lower in gasoline-type emissions than we assumed and that M100-fueled vehicles might have more emission (3%) than modeled ["Report on Potential Emissions and Air Quality Effects of Alternative Fuels" (California Air Resources Board, El Monte, CA, 1989); R. Snow *et al.*, *JAPCA* **39**, 48 (1989)]. In this case, the benefits of M85 will be greater and those of M100 will be marginally smaller.
14. D. Grosjean, *Environ. Sci. Technol.* **16**, 254 (1982); M. Rogozen and R. Ziskind, *Formaldehyde: A Survey of Airborne Concentrations and Sources* (*Pub. 84162*, Science Applications International, San Diego, CA, 1984).
15. D. Lawson *et al.*, *Aerosol Sci. Technol.*, in press; L. Salaz and H. Singh, *Atmos. Environ.* **20**, 1301 (1986); D. Grosjean and K. Fung, *JAPCA* **34**, 537–543 (1984).
16. L. J. Marnett, in *Air Pollution, The Automobile and Public Health* (National Academy of Science, Washington, DC, 1988), pp. 579–603; *Automotive Methanol Vapors and Human Health* (Health Effects Institute, Cambridge, MA, 1987). "Hot spots," such as enclosed garages, may experience locally high levels

of methanol and formaldehyde beyond those expected in the ambient atmosphere. See, for example, M. Gold and C. Moulis, *Air Pollution Control Association Rep.* 84-41.4 (1988).
17. A. M. Dunker, *J. Chem. Phys.* **81**, 2385 (1984). The Direct Decoupled Method is a local sensitivity-analysis procedure and is used to test the impact that small variations in input parameters (for example, emissions or initial conditions) have on output concentrations. These results can be compared to the sensitivity analyses by W. P. L. Carter and R. Atkinson [*Environ. Sci. Technol.* **23**, 864 (1989)]. They described a similar ranking of the reactivity of the compounds. Absolute sensitivity depended on the ROG/NO_x ratio. At ROG/NO_x = 4 (similar to Los Angeles), the reactivity of HCHO to methanol was between 20 and 70 to 1, about the same as in our study.
18. This work was supported by the California Air Resources Board and the South Coast Air Quality Management District.

14 August 1989; accepted 3 November 1989

Repression of c-*fos* Transcription and an Altered Genetic Program in Senescent Human Fibroblasts

TARA SESHADRI AND JUDITH CAMPISI*

Normal cells in culture invariably undergo senescence, whereby they cease proliferation after a finite number of doublings. Irreversible changes in gene expression occurred in senescent human fetal lung fibroblasts: a non–cell cycle–regulated mRNA was partially repressed; an unusual polyadenylated histone mRNA was expressed; although serum induced c-H-*ras*, c-*myc*, and ornithine decarboxylase mRNA normally, ornithine decarboxylase activity was deficient; and serum did not induce mRNA for a replication-dependent histone and for the c-*fos* proto-oncogene. The loss of c-*fos* inducibility was the result of a specific, transcriptional block. The results suggest that senescent fibroblasts were unable to proliferate because of, at least in part, selective repression of c-*fos*; moreover, the multiple changes in gene expression support the view that cellular senescence is a process of terminal differentiation.

A FUNDAMENTAL FEATURE OF NORmal cells is their limited ability to proliferate in culture. Cells capable of proliferation in vivo often go through an initial mitotic period in culture, but invariably there is a gradual decline in cell division; in cells from humans and certain other species, the decline is virtually irreversible and complete (*1*). This progression has been termed the finite life-span phenotype or cellular senescence.

Cellular senescence has been studied most extensively in cultures of human fibroblasts. Generally, human fibroblasts senesce after 20 to 60 population doublings (PDs). Because the PD at which senescence occurs depends inversely on the age of the tissue donor (*1, 2*), it has been suggested that, at the cellular level, senescence in culture reflects aging in vivo. Another view of senescence suggests that it constitutes a mechanism for curtailing tumorigenic transformation and may attenuate the establishment of metastases. Human cell cultures never spontaneously give rise to immortal variants (cells having an unlimited life-span in culture), whereas cells from several rodent and other species give rise to immortal cells at low but readily measurable frequencies. Relative to rodent fibroblasts, human fibroblasts are exceedingly resistant to transformation by chemical carcinogens, radiation, and oncogenic viruses (*3, 4*).

Senescence is not programmed cell death. Senescent fibroblasts remain viable for long periods (many months), during which they synthesize RNA and protein (*5, 7*). Early passage fibroblasts enter a reversible, proliferatively quiescent state (G_0) when deprived of growth factors; they resume proliferation when appropriate growth factors are resupplied (*8*). However, senescent cells cannot be stimulated to enter the S phase of the cell cycle by any combination of growth factors or physiological mitogens (*9*). This failure to synthesize DNA in response to mitogens is not due to a breakdown in growth factor signal transduction. Senescent fibroblasts possess apparently normal receptors for major mitogenic growth factors (*9, 10*), and serum growth factors fully induce the mRNAs for several growth factor–inducible genes (*6*). Several lines of evidence suggest that senescent human fibroblasts express one or more dominant inhibitors of proliferation (*7*) and that the finite life-span phenotype is dominant (*11*).

The link between senescence, aging, and cancer is not clear. Many studies have focused on the inability to proliferate and have viewed the senescent fibroblast as a special or extreme example of a quiescent cell. Within this context, one or more growth-related proto-oncogenes might be under dominant repression in senescent cells. However, quiescent and senescent fibroblasts showed similar basal and serum-inducible expression of the c-*myc* and c-H-*ras* proto-oncogenes (*6*), both of which stimulate proliferation and are required for the ability to leave G_0 (*12*). Because serum generally induces these genes in the early and midportions of the interval preceding DNA synthesis (the G_0/G_1 interval), it has been suggested that senescent fibroblasts arrest growth in late G_1 (*6, 9*). On the other hand, based on morphology and the pattern of proteins synthesized throughout the proliferative life-span, it has also been suggested that senescent fibroblasts have undergone terminal differentiation (*11*). The growth arrest therefore may be one manifestation of a more complex phenotypic change.

Here, we describe several differences in gene expression between quiescent and senescent fibroblasts. These include gene repression and novel gene induction that occur only in senescent cells. Most striking, c-*fos*, which in quiescent fibroblasts is induced early in the G_0/G_1 interval (*14*) and is essential for proliferation (*15*), is repressed at the level of transcription in senescent cells. Our results suggest that the growth arrest shown by senescent fibroblasts is distinct from growth-arrest states described for early passage cells, and c-*fos* repression may at least partially explain why senescent cells fail to proliferate.

To gain insight into the nature of cellular senescence, we examined the pattern of gene expression in human fibroblasts at the extremes of their proliferative life-span. Human fetal lung fibroblasts [strain WI-38 (*1*)] were used at early passage, when the cells had undergone <30 PDs and >70% (typically 75 to 85%) were capable of DNA synthesis, and at late passage (senescence) >48 PDs, when <10% (typically 4 to 8%) were capable of DNA synthesis (*16*). The cells were studied under three conditions in order to identify differences between the quiescent and senescent states. Subconfluent cells in 10% serum were considered exponentially growing; the proliferating fraction was >70% at early passage and <10% at late passage. Confluent cells were given 0.2% serum for 72 hours to generate quiescent cells; the proliferating fraction was <5% for both early- and late-passage cells. Finally, serum-deprived cells were stimulated with fresh 15% serum; >40% of the early-passage cells resumed DNA synthesis, whereas, in senescent cultures, DNA synthesis remained below 10%.

RNA was isolated from the cells and

Department of Biochemistry, Boston University Medical School, Boston, MA 02118.

*To whom correspondence should be addressed.