

5. R. Woodworth, *Psychol. Bull.* **3**, 68 (1906); *Experimental Psychology* (Holt, New York, 1938), p. 592; D. MacKay, *Nature* **225**, 90 (1970); *ibid.*, p. 872. See also R. Wurtz, *J. Neurophysiol.* **32**, 987 (1969).
6. L. Mitrani, St. Mateeff, N. Yakimoff, *Vision Res.* **11**, 1157 (1971).
7. L. Matin, *J. Opt. Soc. Amer.* **54**, 1008 (1964); — and D. Pearce in *Biomedical Sciences Instrumentation*, W. Murry and P. Salisbury, Eds. (Plenum, New York, 1964), pp. 79–95.
8. All stimuli were presented at optical infinity with a three-channel Maxwellian system that demagnified the craters of three Sylvania R1131C glow modulator tubes (to approximately 0.5 mm) and focused their images in the center of the pupil of the observer when he looked at the stimulus in the middle of the fixation array. Because of the small size of these images, no light was lost at the edges of the pupil as the observer made the 4° saccades required in the experiments. The right eye was covered with a patch.
9. The decrease in smear length with increase in duration of the illumination of the slit was found in each of the three observers we have studied. However, only subject E.M. was studied intensively. The phenomenal description of the inhibition is based primarily on the reports of this subject. There were some individual differences among observers. Subject A.K. sometimes noted pronounced apparent movement. Such movement has been reported previously in the metacontrast literature [E. Fehrer, *J. Exp. Psychol.* **71**, 612 (1966); D. Kahneman, *Percept. Psychophys.* **2**, 577 (1967)]. Neither A.K. nor L.M. reported that the pattern was fragmented as E.M. sometimes did. Whereas this may reflect a true difference among subjects, the following observations suggest that it is due to practice: At various times, both A.K. and L.M. reported a complete inhibition of the smear but also a "feeling" that something else had happened or that a movement had occurred. Such reports were also common in early pilot work with E.M. With further practice in observing smears, however, this subject noted that a faint short smear was visible some distance away from the slit (that is, the pattern was fragmented) on trials that elicited these sensations. It should be emphasized that for E.M. complete smear suppression would have been found with the low and intermediate luminances as well as the high luminance if she had not detected this faint peripheral smear (see Table 1 in conjunction with Fig. 1).
10. Maximum distances of 1.5° to 3.5° have been reported by M. Alpern, *J. Opt. Soc. Amer.* **43**, 648 (1953).
11. Although subject E.M. was instructed to saccade from the left to the right fixation target (4°) (see Fig. 1), the average length of the saccades, as could be observed on the permanent record of the eye movements, was somewhat less than 4°. Therefore, with the trigger point at the 1° position, the maximum physical length of the smear on the retina was somewhat less than 3°.
12. Two types of temporal functions have been reported. Functions with a maximum when mask and inhibited stimuli are presented simultaneously are called type A and those with a maximum when the mask follows are called U-shaped or type B [after P. Kolers, *Vision Res.* **2**, 277 (1962)]. See review by Kahneman (4) for further details.
13. For the constant luminance flashes employed in the experiment, the energy received at any given retinal point is inversely proportional to the velocity of the eye at the moment that point was stimulated; that is, while the eye is moving, $E = LW/V$, where E is energy, L is the luminance of the slit, V is the velocity of the eye, and W is the width of the slit. Energy is minimal in the middle of the saccade (where the ocular velocity is greatest), but the smear in that region is most resistant to suppression. Although the spatial and temporal gradients of inhibition considered in the text offer plausible explanations for this, it is clear that further experiments are desirable to determine the effects of different parts of the saccadic pattern in the suppression and to ascertain what happens when the spatiotemporal saccadic pattern is presented to the fixating eye. Moreover, to determine whether the temporal sequencing of the luminous energy is essential to the masking effect we reported, it will be of interest to present an equivalent spatial distribution of energy simultaneously in a brief (for example, 1 msec) flash.
14. For a more complete analysis of the localization question and a discussion of the role of a saccade-contingent extraretinal signal in maintaining stability of visual direction when voluntary saccades occur, see L. Matin, in *Handbook of Sensory Physiology*, D. Jameson and L. Hurvich, Eds. (Springer-Verlag, Heidelberg, 1972), vol. 7, part 4, pp. 344–355; L. Matin and E. Matin, in *Cerebral Control of Eye Movements and Motion Perception*, J. Dichgans and E. Bizzi, Eds. (Karger, Basel, in press).
15. G. von Békésy, *Experiments in Hearing* (McGraw-Hill, New York, 1960).
16. Supported by PHS grant RO1-EY00375 from the National Eye Institute, NIH, and by NSF research grant GB-5947. We thank R. Bowen, K. Kowal and J. Pola, who assisted in pilot work, and A. Kornheiser, who was a subject.

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Tax the Integrated Pollution Exposure

Abstract. *The president has proposed a tax based on the amount of sulfur dioxide emitted from smokestacks and power stations. An alternative method of taxation is suggested which would spread the tax burden among polluters in proportion to their impact on public health. This would be based on the product of the concentration of the pollutant and the population at risk.*

President Nixon has proposed that polluters pay a tax of \$0.15 per pound (0.373 kg) on sulfur dioxide (SO₂) emitted from smokestacks and power stations. In this report an alternative method of taxation is suggested in which the tax would be proportional to the product of the pollutant concentration and the population at risk, not to the quantity of pollutant, as in Nixon's proposal. The purpose of Nixon's tax, as stated by the president, is to encourage, by financial incentives, the use of either fuels with low sulfur content or devices that remove SO₂ from smoke-

stacks. There is a price differential on the East Coast of \$1.50 per barrel between oils of low and high sulfur content. This leads to a cost difference of about \$0.10 per pound and would give an overall incentive of \$0.05 per pound to burn low-sulfur fuel.

Nixon's proposal is a good forward step, but by itself it is not enough. It must not, for example, be used as a replacement for adequate air quality standards. Moreover, Nixon proposes to exclude from his tax, emission in areas where the air quality standards are met, in order to give credit in some

fashion to industries which already, in the public interest, have been located away from population centers. But the proposed method is very crude, and the sharp limit will lead to inequities. The tax proposed here should be more equitable.

It is hard to be sure of the effect on man of low concentrations of SO₂ or any other pollutant in the air; but three studies in Norway (1), and Japan (2), and the United States (3) suggest that the death rate from acute respiratory disease increases linearly with SO₂ concentration. This linear curve has been obtained at concentrations as low as the primary national air quality standard (4), which is a concentration of SO₂ in the air of 0.03 part per million (100 µg/m³). The Norwegian results are shown in Fig. 1. The line appears to be straight, even at the lowest concentration. Pathologists dislike extrapolations of such linear curves, but prudent public policy demands that we assume that no threshold of safety exists if none has been found, and that even a small concentration can affect a few people. This idea has already been accepted for radiation, even though no somatic effects have been measured for doses as low as the dose limit recommended by the International Committee on Radiological Protection and the National Council for Radiation Protection (5).

With a linear curve for death rate versus concentration, we find that a single number expresses the hazard to a population: the sum of the concentrations to which the individual members of the population are exposed. More precisely, if $N(c)dc$ is the number of people exposed to a concentration between c and $c + dc$, we define the integrated concentration:

$$\int cN(c)dc$$

which we call the integrated exposure to pollutants in units of men times concentration.

If everyone (200 million persons) in the United States is exposed to the primary national air quality standard for SO₂, 0.03 ppm, the integrated exposure would be 6 million man-concentrations (in parts per million). If half the population has no exposure and the other half is exposed to 0.06 ppm the integrated exposure would still be 6 million man-concentrations and the death rate would be the same, according to our assumptions. If we take them literally, the data for Norway (1) and Japan (2) suggest that there is a 3 percent increase in the death rate at an

SO₂ concentration of 0.03 ppm. In the United States there are 200 million people and the death rate is approximately 2 million per year; this leads to 10,000 deaths per million man-concentrations.

A source of uncertainty in evaluating the effect of SO₂ is the belief that pure SO₂, by itself, does not cause a hazard to health. In the presence of particulates, sulfuric acid is formed, which is hazardous. Ferris (6) has proposed that the air quality standards should be products of SO₂ and particulate concentrations. Since the pollution tax is based inherently on emissions, I do not see how to take account of such refinements, but suggest that we instead assume a high concentration of particulates—which is not unusual.

How hard should we try to stop these deaths? That depends on the value of human life—or rather, the last 10 years or so of human life, since it is the sick people who die a little earlier. Many people would assume a figure of \$30,000 here, which is a typical value of a life insurance policy. But the exposure is involuntary; Farmer (7) and Starr (8) have pointed out that when the exposure is involuntary people are, and should be, willing to accept only 1/100 of the risk. I therefore assign a value of \$3 million, so that an integrated exposure of 6 million man-concentrations costs 6 million × 10,000 per million × \$3 million = \$180 billion or \$30,000 per man-concentration.

In matters of radiation exposure, this type of calculation has been suggested before. The product of radiation dose and population leads to the unit of a man-rem. From a calculation similar to the above, I obtain \$500 per man-rem. Lederberg (9) finds the same, and Sagan (10) finds \$30 per man-rem. Clearly, the numbers are not precise. On the basis of my value, for example, the "cost" of medical x-rays averages \$45 per person per year, which is about what the patient pays the hospital to administer the x-rays. A nuclear power station would be taxed between \$100 and \$100,000 a year on the same basis.

We are now in a position to specify a procedure for calculating a pollution tax. For any proposed emission, the winds around the site must be measured over a year, or else a conservative figure taken. Then, for a measured or calculated rate of emission of SO₂, a concentration averaged over a year can be calculated at any point. This calculation has already been done for radiation around nuclear power plants. Unless the polluter can justify using another

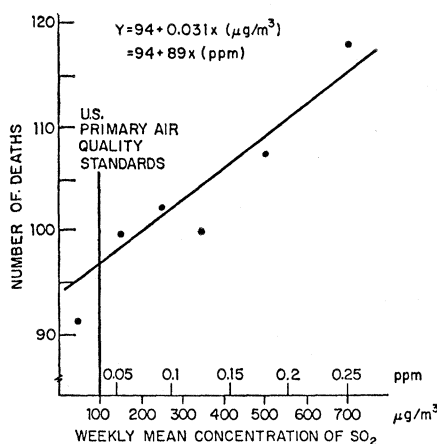


Fig. 1. Total number of deaths in 156 winter weeks in Oslo, from 1958-1959 to 1964-1965, as a function of weekly mean SO₂ concentrations (1).

figure, the winds should be taken as specified in the Atomic Energy Commission's safety guides (4, 11). The concentration is then multiplied by the population to arrive at the integrated exposure in man-concentrations, and hence the tax.

By this means we can take into account the pollution caused by domestic oil heaters; these do not have high chimneys, and they cause local high concentrations of SO₂ over densely populated areas. Of course, we do not expect a million householders to measure the wind speed. But a "standard" conservative calculation can be performed for "typical" domestic homes with "stacks" 15 m high, situated in neighborhoods with "typical" populations. We should evaluate this separately for the categories town, suburbia, and country. The large polluters—industry and power stations—are able to perform a complete calculation, and the tax should encourage them to disperse the SO₂ by using tall stacks, or to locate in relatively unpopulated areas.

Without performing the calculations in detail it is difficult to tell exactly how such a tax would be distributed among polluters. We can, however, calculate the total tax from concentration measurements. I estimate that in

1972 the integrated exposure in the Boston metropolitan area will be 10,000 man-concentrations. The total pollution tax would then be about \$300 million. Of this, perhaps one-fourth (about \$25 per person) will be and should be paid by many individuals as they pay their bills for heating oil. This is big enough to be a significant, and proper, incentive for people to consider carefully their burning of oil. Perhaps this tax is too large overall; it could be changed by any desired factor, by changing the value assigned to a human life.

The same principle could be applied to other pollutants; in the case of NO_x we are concerned with a threshold for smog formation. In this case the sum over concentrations can be cut off at the lower level, but the averaging is not so good. Again the tax should be related to people because smog where there are no people is unimportant. For example, there are mountain valleys filled with smog from natural NO_x, but no one is worried about this natural smog.

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References

1. W. Lindberg, *Den Alminnelige Luftforurensning i Norge* ("The General Air Pollution in Norway") [Utgitt av Røykskaderadet (Norwegian Smoke Damage Council), Oslo, 1968].
2. Y. Nishiwaki, Y. Tsunetoshi, P. Shimizu, M. Ueda, N. Nakayama, H. Takahashi, A. Ichinosawa, S. Kajihara, A. Ohshino, M. Ogino, K. Sakaki, in *Environmental Effects of Nuclear Power Stations* (International Atomic Energy Agency, 1971), p. 247.
3. L. B. Lave and E. P. Seskin, *Science* **169**, 723 (1970).
4. *Fed. Regist.* **36** (No. 67), part 2 (1971).
5. *NCRP Report 39* (National Council for Radiation Protection, Washington, D.C., 1971).
6. B. G. Ferris, "Effects of Selected Air Pollutants on Human Health," report to the World Health Organization (1971).
7. F. R. Farmer, *Int. At. Energy Ag. Rep. SM-89/34* (1967).
8. C. Starr, in *Environmental Effects of Nuclear Power Stations* (International Atomic Energy, Vienna, 1971), p. 895.
9. J. Lederberg, *Bull. At. Sci.* **27** (No. 8) (October 1970).
10. L. Sagan, *Science*, **177**, 487 (1972).
11. L. Rogers and C. C. Gamertsfelder, in *Environmental Effects of Nuclear Power Stations* (International Atomic Energy Agency, Vienna, 1971), p. 127. See also appendix A in (4).

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Survival of *Rana pipiens* in Deionized Water

Kalckar's statement that frogs die within a few hours if placed in distilled water (1) expresses a view that is apparently generally accepted (2). His statement is based on the premise that frogs lose sodium chloride rapidly through permeable skin and inefficient

salt reabsorptive mechanisms (via the kidney) to aqueous environments. Only the presence of an inwardly directed active transport mechanism for Na⁺ allows a fasting frog to effect a net gain of sodium chloride if some salt is present in its bathing medium. In apparent