

No repetition of sequences at all is necessary to produce the rabbit effect. With favorable ISI, hopping may be induced with a few pulses on one contactor and (without break in rhythm) only a single one on a spatially removed contactor. With such paucity of stimulation the effect is not a lively one, but it is unmistakably there.

As to theoretical bases, an early hypothesis was that a traveling mechanical shock wave was being sent through the skin and underlying tissues with each brief pulse of the contactors, and that the anomalous localizations produced must represent reflections, impingements of surface waves on stationary contactors, and perhaps standing waves of some complexity. Such speculation was brought to naught when the "traveling wave" idea was tested by eliminating the wave but preserving comparable stimulation. This was done by substituting for the mechanical tap an electrocutaneous one.

The "rabbit" could not be electrocuted. Three pairs of electrodes, each pair separated from the others by its own isolation transformer, were arrayed on the forearm in a fashion analogous to that with the mechanical contactors. Trains of five 2-msec pulses separated by ISI's between 300 and 30 msec were delivered sequentially to the electrodes. Painless although sharp "taps" were felt at all loci, and, more important, the rabbit effect was present as with mechanical taps. Not all the variables investigated with the mechan-

ical pulses have yet been studied with the electrocutaneous ones, but there appears to be no essential difference in the effects of the two modes of stimulation. The electrocutaneous rabbit effect is more vigorous because of its qualitative advantage—the taps are sharper—but timing is much the same in the two instances and the optimal effect is in the same parametric range.

A comparison can be made with synthetic movement ("phi" phenomenon), reported commonly for visual and also for haptic, especially vibrotactile, stimulus arrays (1). Any simple equating of the present phenomenon with apparent movement on the skin is discouraged by qualitative considerations, however. Vibrotactile movement is perceived as a vibrating, continuous "gouging" of the skin between loci of stimulation. The rabbit effect gives a quite discontinuous and altogether superficially localized impression, as unlike apparent vibrotactile movement as light contact is from deep pressure. Moreover, vibrotactile movement never yields a discrete tap between stimulus loci and this, of course, is the very essence of the rabbit effect.

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Reference and Note

1. C. E. Sherrick and R. Rogers, *Percept. Psychophys.* **1**, 175 (1966).
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Metacontrast and Saccadic Suppression

Abstract. *A vertical slit of light illuminated during horizontal saccadic eye movements appeared as a horizontally extended smear when stimulation was terminated before the saccade ended. However, on trials for which duration of illumination of the slit was extended into the period after the saccade, the smear appeared shorter and dimmer, and a clear image of the slit was seen. With further increases in duration, no smears were seen at the highest luminance of the slit employed, although smears were more than 2 log units above threshold when flashes were brief. This saccadic suppression is discussed in terms of metacontrast, with the accumulated luminance in the period after the saccade primarily responsible for masking the effects of the stimulation received during the movement of the eye.*

When an individual changes his direction of gaze by means of a saccadic eye movement, an image of the environment sweeps across his retina at very great speeds (1). However, the blurring of perception that might be expected from this rapid movement does not occur under ordinary viewing

conditions. The perception of the saccadic smear is suppressed (2).

Suggestions about a major cause of this suppression grew out of experiments in which we presented flashes of fixed luminance during saccades and varied the duration of the flash from trial to trial. In preliminary work, these

flashes were presented on an otherwise dark field as the eye crossed the midpoint of a 2° horizontal saccade. The resulting spread of light on the retina was perceived as a luminous pattern, the apparent length of which was a function of the duration of the flash. For stimuli that were extinguished before the end of the saccade, this saccade-generated pattern appeared simply as a horizontally extended smear, which increased in length with increasing durations of the flash. When the flash extended sufficiently far into the period after the saccade, a sharp image of the flashed stimulus was seen at one end of the smear. These results are in simple correspondence with the spatial distribution of light on the retina. However, on trials with flashes of longer duration (extending further into the period after the saccade) the smeared portion of the perceived pattern appeared shorter and dimmer. When duration of the flash was increased even further, no smear was seen at all, and the stimulus appeared as it did when presented to the fixating eye. These results with longer flashes would not be predicted from the classical theory that attributes saccadic suppression to a central inhibition; neither would they be expected of suppression that is due to a shearing of the retina produced by the movement of the eye (3). Instead, they suggest that temporally backward and spatially lateral inhibition (metacontrast) occurs when the duration of the flash is long enough to permit sufficient temporal integration of illumination in the period after the saccade to develop a "mask" (4). This inhibition prevents perception of stimulation that the flash produced during the movement of the eye. If this explanation of the saccadic suppression found in our study is correct, the suppression is determined essentially by the spatiotemporal pattern of illumination produced on the retina by the saccade and is only incidentally contingent on the occurrence of the eye movement itself. It could, therefore, easily account for reports that "saccadic suppression" can be found when the eye is held still and the environment is moved rapidly with a mirror (5). It is also consistent with an experiment in which no saccadic suppression was found when a stimulus was presented on a dark field and in which suppression increased as the complexity of a background against which the stimulus was presented increased (6).

To study the above phenomenon

quantitatively, we measured the perceived length of the pattern of light (the saccadic pattern) that was generated at the retina when a vertical slit of light was illuminated in the course of a horizontal saccadic eye movement. The luminance of the slit and the duration of its illumination were varied by the experimenter to produce different saccadic patterns. While the subjects viewed these patterns monocularly with the left eye, their horizontal eye movements were monitored by means of a contact lens technique (7). The field of view consisted of a horizontal array of five small, square stimuli (the fixation array) separated by 1° intervals on an otherwise dark field (8) (see Fig. 1, inset a). Each of these squares, which were 1.8 by 1.8 m (6 by 6 feet), had a luminance of -1.53 log millilambert. Fixation at the beginning of each 7-second trial was on the leftmost target of the fixation array (the

left fixation target). At the click of a relay, the subject made a 4° saccade (Fig. 1, inset b) to the rightmost target of the array (the right fixation target). As his eye swept over the stimulus located 1° to the right of the left fixation target (the trigger), a vertical slit of light was flashed 0.75° below the right fixation target. The dimensions of this slit were 0.6 m (horizontal) by 9 m (vertical).

The saccadic pattern appeared either as a smear with a perceived length and brightness that depended on the luminance and duration of the slit, as a smear with a slit at one end, or simply as a slit with no smear at all. To measure the perceived length of this pattern psychophysically, a line 2 msec in duration was flashed 350 msec after the onset of the slit (that is, after the saccade, while the eye was fixating on the right fixation target). This 0.53 log mlam comparison line, the length of which

was set between trials by the experimenter, was located 0.75° above the fixation array. On each trial, the subject reported whether the comparison line was longer or shorter than the perceived pattern. He then moved his eye back to the left fixation target to await the click that signaled the beginning of the next trial. Lengths of the line for a given luminance and duration of the slit were presented according to the psychophysical method of constant stimuli. The resulting data were used to determine the length of the comparison line that appeared longer (or shorter) than the perceived pattern on 50 percent of the trials. Such a length (the perceived length of the saccadic pattern) was determined for each of 12 values of duration studied at each of three luminance levels.

The results for subject E.M. show the perceived length of the saccadic pattern as a function of the duration of the slit (Fig. 1). For each of the three values of luminance employed, the perceived length was maximum at some intermediate flash duration, and was shorter with further increases in duration. However, whereas contrast between the parts of the saccadic pattern was, of course, identical for all values of luminance, masking was most effective with the highest luminance (2.23 log mlam). Although smears at this luminance were more than 2 log units above threshold for brief flashes, no smears were seen at all when the duration of the flash was greater than or equal to 100 msec. The stimulus then appeared as it did when presented to the fixating eye.

Although the apparent brightness of the saccadic pattern was not studied quantitatively, the subjects noted that the brightness of the smeared portion of the pattern was a decreasing function of the duration of the flash for durations greater than approximately 25 msec. Moreover, on some trials the pattern appeared fragmented; that is, it looked like a bright slit, surrounded by darkness, with a faint smear further in the periphery. On such trials, the subject reported on the full length of the pattern (including the faint peripheral smear) relative to the length of the comparison line but also reported that the pattern was fragmented (Table 1). No fragmentation was ever seen at durations on the rising portion of the curves, and smears were hardly ever seen in the immediate neighborhood of the slit at long durations (9).

If the masking in our experiments

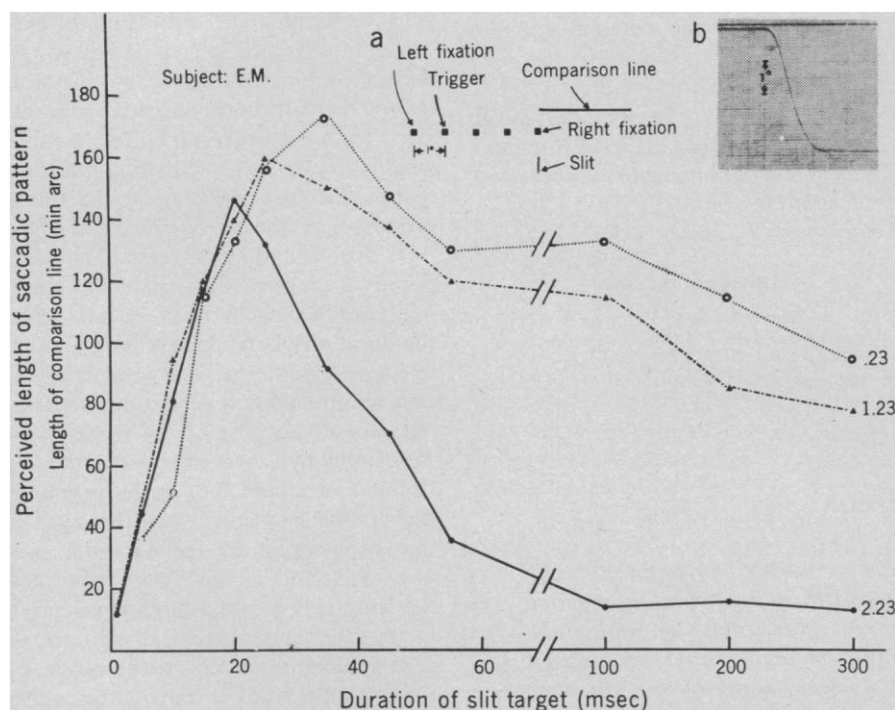


Fig. 1. Perceived length of the saccadic pattern for subject E.M. is shown as a function of the duration of the luminous slit, which was presented when the eye had traversed a distance of 1° in the course of a 4° saccade. The three values of luminance used are shown in log millilamberts at the right ends of the curves. No data point is shown at 1 msec on the 0.23 log mlam curve because the smear was below threshold. For the data shown, the subject made one saccade every 7 seconds. Brief control experiments revealed even greater inhibitory effects when saccades occurred only once per minute (for example, complete smear suppression at 100 msec with the luminance of 1.23 log mlam). Inset a shows the stimuli as they appeared to the subject when all fields were illuminated, the eye was still, and the comparison line was at full length. Light and dark in the drawing are reversed relative to the actual view, and the figure is only roughly to scale. Whereas the five squares used to calibrate the eye movement apparatus were present throughout the experiment, only the two end targets were used to control fixation on experimental trials. Inset b (photographed from a Visicorder recording) shows the time course of the eye position during a 4° saccade by the subject E.M. Downward in the figure represents a horizontal eye movement to the right. The vertical lines are 10-msec time markers.

were due entirely to stimulation after the saccade, it would imply a spread of inhibition over 3° (the distance from the trigger to the end of the saccade). This is approximately the outer limit of the range over which metacontrast has been found in experiments with more conventional stimuli (10). However, we have no reason to believe that 3° represents a limit with the saccade-generated stimulus we used. Although we have not studied longer saccades systematically, we have seen smear suppression with sufficiently long flashes triggered at the 1° position in an 8° saccade (the longest eye movement we examined). The occurrence of inhibition over such long distances suggests that some masking may occur from stimulation presented while the eye is still moving. In line with this suggestion is the finding that the maximum perceived length of the smear corresponded approximately to the actual length of the spread of light on the retina for the lowest luminance of the slit only (11).

With the intermediate luminance and even more emphatically with the higher luminance, the perceived maximum was significantly shorter than the actual spread. This would be expected if successive portions of the smear were partially inhibiting the earlier portions. Whereas this inhibition from stimulation to the moving eye is minimal by itself, it may well play a critical role by initiating the suppression, which is then completed with further stimulation in the period after the saccade.

The tail of the smear (the part farthest from the perceived slit) was usually suppressed with durations of the flash at which the rest of the smear was still visible. This would be expected if the later portions of the smear were contributing to the masking of the earlier portions. However, the early suppression of the tail may also be expected of masking that was due to stimulation after the saccade, if the temporal masking function involved was the kind that is called U-shaped or type B (12). Such functions show inhibitory effects that first increase rapidly with time between the inhibited stimulus and the mask, come to a maximum at 50 to 120 msec, and then decrease again. Because the tail area of the smear in our experiments was produced by stimulation that arrived at the retina some 25 msec earlier than the part of the smear closest to the perceived slit, it would be suppressed more easily than the latter area. There is also

Table 1. The fragmentation data for subject E.M. For each luminance and duration of the slit, the table shows the proportion of trials on which the saccadic smear was completely inhibited in the neighborhood of the perceived slit but was still visible more peripherally (that is, the saccadic pattern was separated into two parts). Starred entries are for values of luminance and duration where inhibition was always complete (only a narrow slit visible, without any smear).

Slit duration (msec)	Proportion of trials fragmented at slit luminance (log mlam)		
	0.23	1.23	2.23
1-25	0	0	0
35	.48	0	0
45	.77	0	.02
55	.73	0	.14
100	.92	.33	*
200	.98	.92	*
300	.96	1.0	*

evidence of a spatial gradient of inhibition. This manifests itself at somewhat longer durations by the fragmentation of the perceived pattern (the part of the smear nearest the perceived slit was completely inhibited while parts further away were still visible). Contrary to what might be expected from simple considerations about the time-integrated luminance that the slit produces at any given point on the retina, the middle of the smear was most resistant to suppression (13).

Although metacontrast appears to be the appropriate interpretation for the effects we have described, it should be noted that our findings in no way rule out the occurrence of some saccadic suppression through mechanisms other than visual masking. They do suggest, however, that future experiments about such mechanisms should be designed to preclude the possibility that masking could be the cause of any observed effects. It should also be emphasized that the large suppression found in our study occurred under severely reduced stimulus conditions which only permitted a limited amount of backward lateral inhibition. When saccades occur in more complex environments, we could expect richer possibilities for lateral masking (both forward from stimulation prior to the saccade and backward from stimulation after the saccade). In addition, masking in which the mask and the inhibited stimulus fall on the same retinal area could then come into play. The combined effect of stimulation from these various sources seems sufficient to account for the fact that perceptual blurring does not result from the rapid image displacement caused

by saccadic eye movements under ordinary conditions of illumination.

Detailed measurements of the visual direction of the saccadic pattern were not made in our experiment. However, it should be noted that the briefly presented slit was perceived below and to the right of the right fixation target, although the physical location of the slit was immediately below this target. As the duration of the flash was increased, the smear length increased toward the left (in the direction opposed to the eye movement). Masking was manifested by a decrease in the length of the smear from its right edge. Finally, when a definite image of the slit was formed with the stimuli of longer duration, it appeared to lie below the right fixation target, in accordance with its actual position in physical space. Clearly, therefore, smear suppression can be an aid in visual spatial localization (14). Similar suppression and sharpening of localization have been demonstrated in the somesthetic system with cutaneous stimulation by traveling waves on the arm, and it is extremely likely that analogous mechanisms are involved in pitch perception and auditory localization (15).

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References and Notes

1. Regardless of length, all saccades have similar temporal characteristics. Velocity increases monotonically (first with increasing acceleration and then with decreasing acceleration), reaches a peak near the middle of the saccade, and then decreases monotonically for the rest of the eye movement. However, average velocity, peak velocity, and duration of a saccade increase with its length. See R. Dodge and T. Cline, *Psychol. Rev.* 8, 145 (1901); D. Robinson, *J. Physiol. London* 174, 245 (1965); A. Yarbus, *Eye Movements and Vision* (Plenum, New York, 1967); and also Fig. 1, inset b.
2. For references on saccadic suppression see F. Volkman, A. Schick, L. Riggs, *J. Opt. Soc. Amer.* 58, 502 (1968).
3. For a study of the central inhibition hypothesis that includes a summary of the literature, see (2). The suggestion that a shearing of the retina leads to suppression was proposed by W. Richards [*J. Opt. Soc. Amer.* 59, 617 (1969)].
4. For reviews on metacontrast, a phenomenon which has been studied extensively under conditions of steady fixation, see M. Alpern, *Amer. J. Optom.* 29, 631 (1952); D. Raab, *Psychol. Bull.* 60, 118 (1963); D. Kahneman, *ibid.* 70, 404 (1968). Lateral inhibition in visual perception has been discussed by T. Cornsweet [*Visual Perception* (Academic Press, New York, 1970)] and F. Ratliff [*Mach Bands* (Holden-Day, San Francisco, 1965)]. Our use of the words backward and lateral refers to stimulus events at the retina. Nothing is implied about the neural level or levels where the inhibition we are describing may be effected. Similarly, by our juxtaposition of the metacontrast interpretation with the classical central inhibition hypothesis we do not imply that metacontrast is peripheral.

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. Psychophysics*, **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. Kolars, *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