MAO activities between index twins and their nonschizophrenic co-twins indicates that the low MAO activities are not solely secondary to being schizophrenic but may be genetically determined. We have also found a high correlation (r = .94) in platelet MAO activity between nine pairs of normal monozygotic twins, as have Nies and Robinson (11). In addition, the mean value for the normal twin pairs was no different from other normals who were not twins. Because the present twins are discordant for schizophrenia, it appears that low platelet MAO is not a marker for the disorder, but rather may be a genetic marker for the vulnerability to schizophrenia. A smaller reduction in platelet MAO activity is sometimes observed for patients with bipolar depression but not for those with unipolar depression; this suggests that there may be a common factor affecting MAO activity in bipolar depression and schizophrenia (9). The evidence indicating a reduction in MAO activity in some schizophrenic patients and their relatives is so far confined to blood platelets, and there are no data to indicate whether MAO activity may also be reduced in brain or other tissue. Whatever platelet MAO activity may reflect regarding brain function, it can be at most only one part of a complex system leading to schizophrenia. Prior studies have shown that although genetic factors are important, they can account for less than half of the disposition to become schizophrenic (1). Thus, there is no essential contradiction between our results indicating that MAO activity is related to severity of impairment and is also correlated between co-twins discordant for schizophrenia.

It cannot be concluded that low MAO activity in platelets is a primary genetic alteration in schizophrenia. While such an alteration could be involved in the pathogenesis of this disorder, the alteration could be secondary to other genetic factors that might regulate MAO activity in platelets or might be incidentally related but genetically linked to the actual pathogenetic mechanism of schizophrenia. It is also conceivable that a factor occurring in utero or in early life could yield this difference in MAO activity. It should be possible to determine whether relatives of schizophrenic individuals also have reduced MAO activity and, if so, how this alteration is transmitted. Also, it would be useful to know whether the difference in platelet MAO is repre-

sentative of a generalized deficiency in MAO or one of its isoenzymes, and, specifically, whether there is a deficiency in brain MAO. Since a generalized deficiency in MAO activity would be consistent with many current theories relating to abnormalities in indoleamine and catecholamine metabolites (2, 4, 9, 12) in schizophrenia, it will be of interest to determine which of the many substrates for MAO is most affected by this abnormality.

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References and Notes

1. D. Rosenthal, P. Wender, S. Kety, F. Schul-singer, J. Welner, L. Østergaard, in The Transmission of Schizophrenia, D. Rosenthal and S. Kety. Eds. (Pergamon, New York, 1968), pp. 377–391; S. Kety, D. Rosenthal, P. Wender, F. Schulsinger, in *ibid.*, pp. 345– 362; L. 3 363–376; . Heston and D. Denney, in *ibid.*, pp. 545 6; W. Pollin, M. Allen, A. Hoffer, J. R. Stabenau, Z. Hrubec, Amer. J. Psychiat. 126, 597 (1969).

- 2. R. Wyatt, B. Termini, J. Davis, Schizophrenia Bull. 4, 8 (1971); ibid., p. 45.
- 3. D. Murphy and R. Wyatt, Nature 238, 225 (1972)
- 4. W. Pollin, Arch. Gen. Psychiat. 27, 29 (1972). The pairs reported on here were selected as the most clearly discordant subgroup of an original series of 17 discordant pairs. selection employed a somewhat nat original series of narrower definition of schizophrenia, and was done before the laboratory data were available. One pair was not included because of a laboratory error.
- and J. Stabenau, J. Psychiat. Res. 6 (Suppl. 1), 317 (1968). 5. -
- 6. A. Hoffer and W. Pollin, Arch. Gen. Psychiat. 23, 469 (1970). 7. R. Wyatt, J. Saavadra, J. Axelrod, Amer. J.
- Psychiat., in press. Axelrod, Biochem.
- R. Wurtman and J. A Pharmacol. 12, 1417 (1963). 9. D. Murphy and R. Weiss, Amer. J. Psychiat.
- 128, 35 (1972). S. Siegel, Nonparametric Statistics for the Behavioral Sciences (McGraw-Hill, New York, 10,
- 1956), 11. A. Nies and D. Robinson, personal communication.
- 12. S. Snyder, Arch. Gen. Psychiat. 27, 169 (1972); A. J. Mandell, D. Segal, R. Kuczenski, S. Knapp, Psychol. Today 6, 68 (1972).
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Multilane Vehicular Traffic and Adaptive Human Behavior

Abstract. The parameters in a statistical theory of multiple-lane traffic have been determined from two independent sets of data. The numerical values of the parameters calculated by four different methods of estimation are in essential agreement with one another. The data suggest the important role of adaptive human behavior in determining the characteristics of congested flow.

Traffic flow on a multiple-lane highway has been studied in recent years by using various approaches (1, 2). The manner in which human behavior is involved in this problem makes it one of the most interesting in the theory of traffic flow, and in addition, similar features are likely to appear in other complicated problems of sociology and economics. In this connection it has been stated in a monograph (1, p. 87) on the theory of multilane traffic: "We have here an example of a game in which the rules are not fixed once for all, but vary according to circumstances. The modification of the rules implies a modification of behavior of the entire collectivity of participants." The main intent of the theory of traffic we have presented (1) is to permit us to gain some insight into the mechanisms through which the change of concentration on a highway leads to modification of the traffic pattern. We have discussed the theory (3) with respect to data obtained from two different sources (4, 5). As it is of great importance to validate our theoretical approach with respect to experimental data (6), we examine here a comparison of theory and data from a somewhat different viewpoint. We have estimated from the data the numerical values of the parameters included in our description of the multiple-lane traffic. The values of the parameters calculated in various independent ways from the lower moments of the speed distribution function are in satisfactory agreement with one another; moreover, the variations of the parameters with concentration are along lines predicted by the theory.

Oualitative conclusions are also drawn from the cumulative distribution of the observed speeds. The dominating effect of the "adaptive behavior" of the drivers exhibited in the speed distributions at high concentrations is observed. It appears that one of the basic problems regarding congested flow is to describe quantitatively the adaptive behavior brought about by the interactions between drivers. This pattern of behavior illustrates the essential nonlinearity of human behavior involving the continuous interplay between "program" and "realization."

Table 1. Estimation of the parameters. For the SDC data (4), a speed distribution observed at a concentration of 32.7 vehicles per mile was used as the desired speed distribution, f_0 ; for the PNYA data (5), a speed distribution at 62.0 vehicles per mile was used. Values denoted by asterisks are imaginary.

Distri- bution	Concen- tration (vehicles per mile)	β-1	γ				λ			
			Method 1	Method 2	Method 3	Method 4	Method 1	Method 2	Method 3	Method 4
• • • • • • • • • • • • • • • • • • •					SDC					
<i>f</i> 1	49.5	53.80	57.45	59.61	87.00	57.51	3.65	5.81	33.20	3.71
f_2	88.4	4.75	11.79	12.51	12.71	*	7.04	7.76	7.96	*
					PNYA					
f_1'	74.3	18.98	26.76	21.63	21.82	20.96	7.78	2.65	2.84	1.98
f_{2}'	93.0	13.17	20.13	20.26	20.21	*	6.96	7.09	7.04	*

Let us summarize briefly our general approach [see (1)]. Based on the speed distribution, $f_0(v)$, in very dilute traffic, the evolution of the speed distribution, f(v,x,t), to a higher concentration, c(x,t), is studied. The speed distribution, $f_0(v)$, at $c \rightarrow 0$ is assumed to represent the desired speeds of the drivers on the road. Three different processes are included in the evolution equation for the function f: the relaxation process, the interaction process, and the adjustment process. These processes are expressed in that order in a kinetic equation of the form:

$$df/dt = -(f - f_0)/T + (1 - P)c(\bar{v} - v)f + \lambda(1 - P)c[\delta(v - \bar{v}) - f]$$
(1)

The mean speed determined from f(v) is denoted by \overline{v} . The relaxation time, T; the probability of passing, P; and the parameter λ are all considered to be functions of concentration; and δ is a Dirac delta function. The expressions in Eq. 1 are only thought of as plausible mathematical representations of rather complex phenomena involving human behavior.

The steady-state solution of Eq. 1 satisfies the relation

$$f_{0} = [1 + \lambda\beta + \beta(\nu - \bar{\nu})]f - \lambda\beta\delta(\nu - \bar{\nu})$$
(2)

where

$$\beta = (1 - P)cT \tag{3}$$

For most calculations involving the steady-state solution, the various parameters can be further consolidated into a single parameter,

$$\gamma = \lambda + \beta^{-1} \tag{4}$$

In some previous calculations, we have assumed

$$\beta = \tau c_{\rm p} \eta^3 / (1 - \eta)$$

$$\gamma = \lambda + (1 - \eta) / (\tau c_{\rm p} \eta^3) \qquad (5)$$

where $\eta = c/c_{\rm p}$ is the normalized concentration with respect to the limiting or jam concentration, $c_{\rm p}$, and τ is a 2 MARCH 1973 characteristic constant. The parameter β increases with concentration, and the parameter γ must decrease with concentration in order that the average speed decrease with concentration. It follows that the parameter λ may either first increase with concentration at low concentrations and then decrease with concentrations, or have a slope with respect to η always less than $(\tau c_p)^{-1}$.

We have shown (3) that from Eq. 2 a set of relations can be determined between the kth central moment, $m^{(k)}$, of the prevailing speed distribution fat arbitrary concentration and the kth central moment, $m_0^{(k)}$, of the desired speed distribution f_0 . For example, we obtain

$$m^{(3)}/m^{(2)} = m_0^{(2)}\Delta^{-1} + \Delta - \gamma \qquad (6)$$

$$n^{(4)}/m^{(2)} = [m_0^{(3)} + (3\Delta - \gamma)m_0^{(2)}]\Delta^{-1} + \Delta^2 - \Delta\gamma + \gamma^2 \qquad (7)$$

where $\Delta = \bar{\nu}^0 - \bar{\nu}$ is the difference between the mean desired and the mean prevailing speeds. The quantity Δ is related to β as follows:

$$\Delta = \beta m^{(2)} \tag{8}$$

Finally, it has been shown (7) that the mean speed $\bar{\nu}$, and thus Δ , is uniquely determined by the parameter γ and the desired speed distribution f_0 .

Let us now turn to the comparison between data and theory. One of the available sets of data was obtained from an observational study carried out at the Port of New York Authority (PNYA) on the George Washington Bridge (5). The other set of data was collected on a freeway by the Department of Transportation and was used in a study by the System Development Corporation (SDC) (4). These data sets are described and discussed in (3). In both cases we do not know if the conditions under which the data were collected were such that the concentration of cars was the only independent variable. All conclusions based on these data have, therefore, to be considered as preliminary and should be tested

against more accurate data whenever they become available.

An essential feature of the theory is the role played by the parameter γ defined in Eq. 4. We have calculated γ from the data by four methods.

1) From \bar{v} and f_0 by using the method described in (3).

2) From $m^{(3)}/m^{(2)}$ by using Eq. 6, which also involves $m_0^{(2)}$ and Δ .

3) From $m^{(3)}/m^{(2)}$ by using again Eq. 6 and $m_0^{(2)}$, but considering Δ itself as a function of γ .

4) From $m^{(3)}/m^{(2)}$ by using Eq. 7 together with $m_0^{(3)}$, $m_0^{(2)}$, and Δ .

The results of these calculations are given in Table 1. The agreement is reasonable although it may well be fortuitous, and, as mentioned earlier, new data are highly desirable.

It may be worthwhile to mention that the errors in the experimental data lead to a rapidly increasing incertitude when we go to higher moments. The values of γ as calculated from the fourth moments with method 4 comes from the solution of a second order equation. As indicated in Table 1, method 4 does not yield real roots for the higher concentrations. However, small changes in $m^{(4)}/m^{(2)}$ well inside the probable experimental error lead to real values for γ of the same order of magnitude as the values obtained from the other methods. The value of γ determined from the data decreases with concentration as does β^{-1} . We also expect (see Eq. 5) that the variation of β with concentration would follow a power law with a characteristic exponent μ of order 2 to 3. It is interesting that the experimental data indicated a higher concentration dependence ($\mu = 4.2$) for the SDC data than for the PNYA data ($\mu = 1.6$). There may be an intuitive interpretation of these results: on a bridge, the "hindrance" between drivers develops more slowly with concentration as drivers may make less of an effort to maintain their desired speed distribution over the limited span of the

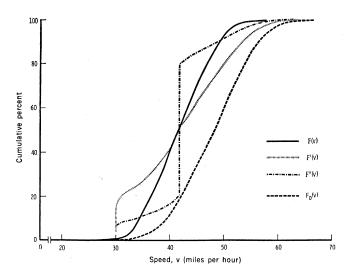


Fig. 1. The two theoretically detercumulative mined speed distributions. F'(v)and F''(v), for the concentration of 88.4 vehicles per mile are shown in conjunction with the observed speed distribution. F(v), from the SDC data (4) at the same concentration. Also shown is the cumulative desired speed distribution, $F_0(v)$, employed in the theoretical calculations.

bridge than over the more extended length of a freeway.

Let us now discuss some of the overall features of the speed distribution function. As we mentioned before (3)the comparison between theory and experiment has to be made on the basis of some integral form, since Eq. 2 involves a Dirac delta function, which is only meaningful in conjunction with integration. We shall therefore consider here the cumulative distribution function.

A specific example corresponding to the SDC data at the concentration of 88.4 vehicles per mile is considered, as the effects we wish to discuss are less marked in the more dilute traffic. In Fig. 1 we have plotted the observed cumulative speed distribution, F(v), as well as two calculated distributions F'(v) and F''(v), for the same concentration. The cumulative distribution of the desired speeds, $F_0(v)$, employed for the calculations is also shown. The distribution $F_0(v)$ was determined from observations at a lower concentration. The parameter γ was estimated by method 1 (see Table 1). In the case of F'(v), λ is assumed to be zero. For F''(v) we use $\lambda = 7.04$, which was estimated from Δ and the second moment (see Table 1). As can be seen in Fig. 1, if we neglect the adjustment term by setting $\lambda = 0$ as in the case of F'(v), the distribution would start too rapidly as compared to F(v) and then approach unity at a higher speed than does F(v). In other words, by ignoring the adjustment term the theory predicts too many cars at the very high and the very low speeds. If we take $\lambda = 7.04$ we overestimate the cooperative character of the drivers. The distribution F''(v) thus shows too many cars in the neighborhood of the mean

speed. The fact that the observed distribution lies between the two predictions indicates the essential role of the adjustment process, which is in agreement with our general approach with regard to the importance of drivers adapting to changes in traffic conditions.

Because of the importance of driver adaptation, it is worthwhile to model the adjustment terms more precisely. The delta function type of adjustment in Eq. 1 is only an approximation. As various groups of drivers adjust primarily to their own local conditions and local average speeds, the distribution must have a finite spread. In accordance with this result, we see in Fig. 1 that the large deviations between F(v) and F''(v) around $v = \bar{v}$ take place in an interval of the order of $[(v - \bar{v})^2]^{\frac{1}{2}}$. The quantitative formulation of this idea requires replacing the delta function in Eq. 1 with a function having a spread of the correct order of magnitude.

In order to avoid misinterpretation, we emphasize that a theory which includes effects as complex as human adaptive behavior cannot be deduced from a priori arguments. The goal of the theory is first to describe general characteristics, and then, if possible, to compare the relative importance of the various factors and processes in order to establish a rational approach to problems such as highway design and traffic control.

It is also hoped that the relative success of the one-dimensional multilane traffic problem may stimulate the development of similar mathematical models in other disciplines where adaptive behavior is also present. In all such cases the adaptive strategy is intimately related to a self-imposed program of objectives whose realization must in turn depend on the strategy.

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References and Notes

1. I. Prigogine and R. Herman, Kinetic Theory of Vehicular Traffic (American Elsevier, New York, 1971).

2. For example, see D. C. Gazis, Amer. Sci. 60, 414 (1972)

3. R. Herman, T. Lam, I. Prigogine, Transp. Sci. 6, 440 (1972).

A. V. Gafarian, P. K. Munjal, J. Pahl, Transp. Res. 5, 211 (1971).
 J. L. Kraus, "Distribution of vehicular speeds," Port of New York Authority, internal report, unpublished (1966).
 E. A. Haight, Science 172 512 (1971).

6. F. A. Haight, Science 173, 513 (1971). 7. R. Herman and T. Lam, Transp. Sci. 5, 314 (1971).

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Cannabis and Alcohol: Effects on Simulated Car Driving

Abstract. The effects of cannabis and alcohol on simulated car driving were studied. Cannabis resin containing 4 percent Δ^1 -tetrahydrocannabinol was administered orally in three doses equivalent to 8, 12, and 16 milligrams of that component. Alcohol was given orally in one standard dose of 70 grams. Both cannabis and alcohol increased the time required to brake and start, whereas alcohol increased while cannabis decreased the number of gear changes. An effect of dosage on response was observed with cannabis.

Cannabis metabolites have been detected in urine after oral administration of cannabis to volunteers (1). The research reported here is part of a program designed to combine metabolic and psychological studies of the acute effects of cannabis on man. We have used a different type of car simulator than was used in an earlier study (2), and oral administration of cannabis preparations with known, adequate tetrahydrocannabinol (THC) contents. The research design included placebos, a double-blind procedure, tests for re-