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A Mathematical Model of Performance on a Simple Reaction Time Test

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KRIEG, JR., E. F., D. W. CHRISLIP AND J. M. RUSSO. *A mathematical model of performance on a simple reaction time test.* NEUROTOXICOL TERATOL **18**(5) 587–593, 1996.—A nonlinear function with components for learning and fatigue was used to model individual performance on a simple reaction time test. The relationships between the parameters of the model and the mean and variance of the reaction times are discussed. The function is used to analyze data from a field study of agricultural workers exposed to organophosphate pesticides. Exposure had a significant effect on the relationships between education level and initial performance, age and fatigue rate, and age and performance variability. Parameter estimates from the model were able to distinguish between effects that the mean and standard deviation of the reaction times were unable to distinguish.

Simple reaction time Nonlinear model Organophosphate pesticides Intraclass regression model

A recent review of behavioral testing in the workplace reported that tests of reaction time have been employed in more than 80 workplace studies (4). These studies have revealed that reaction time performance is affected by many substances encountered in the workplace, including lead (19,22), mercury (5), manganese (34), pesticides (43), carbon disulfide (25,41), styrene (15,29), anesthetic gases (16), and solvent mixtures (9,26). The usefulness of the reaction time test as a tool for workplace testing has led to its being incorporated in a number of test batteries, including the Finnish Institute of Occupational Health (FIOH) Battery (17), the World Health Organization's Neurobehavioral Core Test Battery (WHO NCTB) (21), the London School of Hygiene Battery (10), the Williamson Battery (42), the Armed Forces Cooperative Performance assessment Battery (UTC-PAB) (13), and the Neurobehavioral Evaluation System (NES) (24).

A number of individual characteristics are also known to affect reaction time. These include age (1,38), sex (8,28), degree of physical fitness (37), mental retardation (6), fatigue (18), and sleepiness (12). Reaction times can also be affected by physical activity (38), alcohol intake (12,30), and cigarette smoking (30).

Reaction time data are typically analyzed by dropping out the reaction times of the initial trials and calculating the mean and SD of the remaining ones. Information is lost about the rate of acquisition of the reaction time response as well as any other trend over trials. The pattern of response over trials can vary substantially from subject to subject. Some subjects acquire the response skill more rapidly, some subjects show a gradual increase in reaction times as the number of trials increases, and the performance of some subjects is more variable than others.

The trends in reaction time over trials can be modeled mathematically. Parameters that reflect these trends can be estimated and used as variables in analyses done to determine the effects of toxic exposure, age, or other variables. A nonlinear model is described below; it is compared to the mean and variance of the reaction times, and it is applied to data from a field study of agricultural workers exposed to organophosphate pesticides.

DESCRIPTION OF THE MODEL

The model has two components, one to account for learning (the initial decrease in reaction time) and one to account for fatigue (the gradual increase in reaction time over trials). The reaction time at trial t can be written as a product of these two components: $R(t) = L(t) \times F(t)$.

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The learning function is

$$L(t) = L_\infty - (L_\infty - L_1)(1 - l)^{t-1}$$

for $t \geq 1$ and $0 \leq l \leq 1$. l is the learning rate, L_1 is the initial performance level, and L_∞ is the performance limit. If $l = 0$ there is no learning, and if $l = 1$ there is one-trial learning; maximum performance is reached by the second trial. $L(t)$ is undefined when $l = 1$ and $t = 1$.

The fatigue function is

$$F(t) = (1 + f)^{t-1}$$

for $t \geq 1$ and $f \geq 0$. f is the fatigue rate. If $f = 0$, there is no fatigue. If $f > 0$, there is no fatigue on the first trial, but as the trials progress fatigue increases without bound.

Combining the two functions one gets

$$R(t) = [L_\infty - (L_\infty - L_1)(1 - l)^{t-1}](1 + f)^{t-1}$$

$R(t)$ is undefined when $l = 1$ and $t = 1$.

RELATIONSHIPS TO THE MEAN AND VARIANCE

A statistical version of the model can be written as

$$R_t = R(t) + \varepsilon_t,$$

where ε_t is a variable representing a random disturbance at time t. If $E[\varepsilon_t] = 0$, then $E[R_t] = R(t)$. $\mathrm{Var}[R(t)] = 0$, so $\mathrm{Var}[R_t] = \mathrm{Var}[\varepsilon_t]$.

The sample mean of the reaction times over a set of T trials has expected value $E[\bar{R}] = (1/T)\,\Sigma\, R(t)$ and variance

$$\mathrm{Var}[\bar{R}] = \frac{1}{T^2}\sum_t \mathrm{Var}[\varepsilon_t] + \frac{2}{T^2}\sum_t \sum_{u>t} \mathrm{Cov}[\varepsilon_t, \varepsilon_u].$$

By examining the equation for $R(t)$ one can see that $\bar{R}$ increases as L_∞ increases, as L_1 increases relative to L_∞, and as f increases. $\bar{R}$ decreases as l increases.

The sample variance of the reaction times, using a divisor of $T - 1$, has expected value

$$E[S_R^2] = \frac{1}{T-1}\sum_t (R(t) - E[\bar{R}])^2 + \frac{1}{T}\sum_t \mathrm{Var}[\varepsilon_t] - \frac{2}{T(T-1)}\sum_t \sum_{u>t} \mathrm{Cov}[\varepsilon_t, \varepsilon_u].$$

If $l = 0$ and $f = 0$ then $R(t)$ is constant and will not contribute to S_R^2, otherwise the first term to the right of the equal sign will be greater than 0 and S_R^2 will contain variability due to the trend.

AN EXAMPLE

The data used here are from a study of agricultural workers exposed to organophosphate pesticides (39). Workers with medically documented episodes of acute poisoning by one or more organophosphate pesticides were examined 2–10 years after apparent recovery for signs of residual neurobehavioral deficits. The study subjects were selected from medical surveillance records maintained by the State of California Department of Food and Agriculture, and were restricted to cases in which males, age 16 years or older, had sought medical attention after a definite or probable episode of organophosphate pesticide overexposure. A total of 83 definite poisoning cases were identified in which symptoms compatible with organophosphate poisoning were accompanied by documented cholinesterase inhibition. An additional 46 cases of probable organophosphate poisoning lacked evidence of cholinesterase inhibition, but exhibited compatible symptoms accompanied by specified medical signs or by a history of direct overexposure to an organophosphate pesticide. Another 45 subjects were identified as having had one or more prior episodes of cholinesterase inhibition without symptoms of poisoning. Ninety adult male friends and neighbors of the exposed cases who were not currently exposed to pesticides constituted the nonexposed control group.

The subjects in the study were given an assessment battery that included a clinical neurological examination, evaluation of nerve conduction velocities, measurement of vibrotactile thresholds, and computerized tests of postural balance, mood, finger tapping speed, sustained visual attention, hand–eye coordination, symbol–digit matching, short-term pattern memory, short-term serial digit learning, and simple reaction time.

The simple reaction time test was from the NES. In this test, the subject is asked to press a button each time a large square appears on the computer screen. The number of trials can be set by the test giver and is typically set between 50 and 100 trials (in this study subjects were given 90 trials). The intertrial interval varies randomly between 2.5 and 7.5 s to prevent the subject from anticipating the next stimulus.

Steenland et al. (39) analyzed the definitely and probably poisoned groups, but not the group with inhibited acetylcholinesterase and no symptoms. This group was included in the present analysis. Twelve subjects from the definite group, three from the probable group, two from the inhibited group, and four controls did not have reaction time data. Additionally, data from four definite, four probable, and eight control subjects were not included in the analysis because their data was collected during pilot testing.

Fitting the Model

The reaction times (r_t) of individual subjects were fit using the Gauss–Newton algorithm [all calculations were done with the SAS System (SAS Institute, Inc., Cary, NC)]. The first reaction time (r_1) was used as a starting value for L_1, the arithmetic mean of reaction times 11 through 60 was used as an initial estimate of L_∞, and d was calculated as $d = L_\infty - L_1$. The initial value of the learning rate was calculated as $l = (r_2 - L_1)/(L_\infty - L_1)$, and the initial value for the fatigue rate was calculated as

$$f = \left[\prod_{t=61}^{90} \frac{r_t}{r_{t-1}}\right]^{1/30} - 1$$

Only values greater than 100 ms and less than 700 ms were included in the calculation of the initial values of L_∞ and f.

Conditional statements were used to adjust extreme initial values: if l was less than 0.2 or greater than 1, then it was set to 0.6; if d was greater than -50, then d and l were set to 0; and if f was less than or equal to 0 or greater than or equal to 0.01, then it was set to 0.001. Constraints were also placed on the parameter estimates: $L_\infty \geq 100$, $d \leq 0$, $0 \leq l \leq 1$, and $f \geq 0$.

A Ljung–Box (27) test was performed on the residuals of each of the fits. This test indicated significant autocorrelations for 21% of the subjects. The correlations for lags greater than zero ranged from -0.33 to 0.57. Their average was -0.01. Examining the autocorrelation functions and fitting autoregressive-moving average models to the residuals indicated that

some of the residuals varied cyclically, some appeared to be an autoregressive process (negative and positive), and some were a combination of both; about one-third of the 21% were in each category. We did not develop a model for the residuals because of the size and inconsistency of the correlations, and because correlated residuals have been shown not to bias parameter estimates (11).

The standard error of the estimate (SEE) was calculated for each regression and was used as a measure of performance variability. For purposes of comparison, the mean ($\overline{R}$) and SD (S_R) of the last 80 reaction times of each subject were also calculated. Examples of fitted regression lines are shown in Fig. 1.

Statistical Analysis of the Parameter Estimates

A linear model was used to test for the effect of exposure, the linear effects of age and education level (grade), as well as age × exposure and grade × exposure interactions on the parameter estimates. The model is called an intraclass regression model because a different regression is assumed for each class or group (36). The equation for the model can be written as

$$Y_{ij} = \mu + \alpha_i + \beta_{1i}Z_{1ij} + \beta_{2i}Z_{2ij} + \varepsilon_{ij}$$

Y_{ij} is the value of the dependent variable for the jth subject from the ith group, Z_{1ij} and Z_{2ij} are the values of the covariates age and grade, and ε_{ij} represents a random disturbance. $\mu + \alpha_i$ is the intercept for the ith group, and β_{1i} and β_{2i} are slope coefficients.

The design matrix for this model included a column of ones for the intercept and four columns to code for the groups. A subject was coded as 1 if he was in the group or 0 otherwise. Age and grade were included as continuous variables and columns for the interactions were calculated by multiplying the age and grade columns by the group columns.

If a main effect or an interaction was significant, contrasts were done to compare the groups. If an interaction was significant, the slope coefficient of each group was tested to see if it was significantly different from zero.

Results

Summary statistics for the variables used in the analysis are shown in Table 1, a summary table of the linear models is pre-

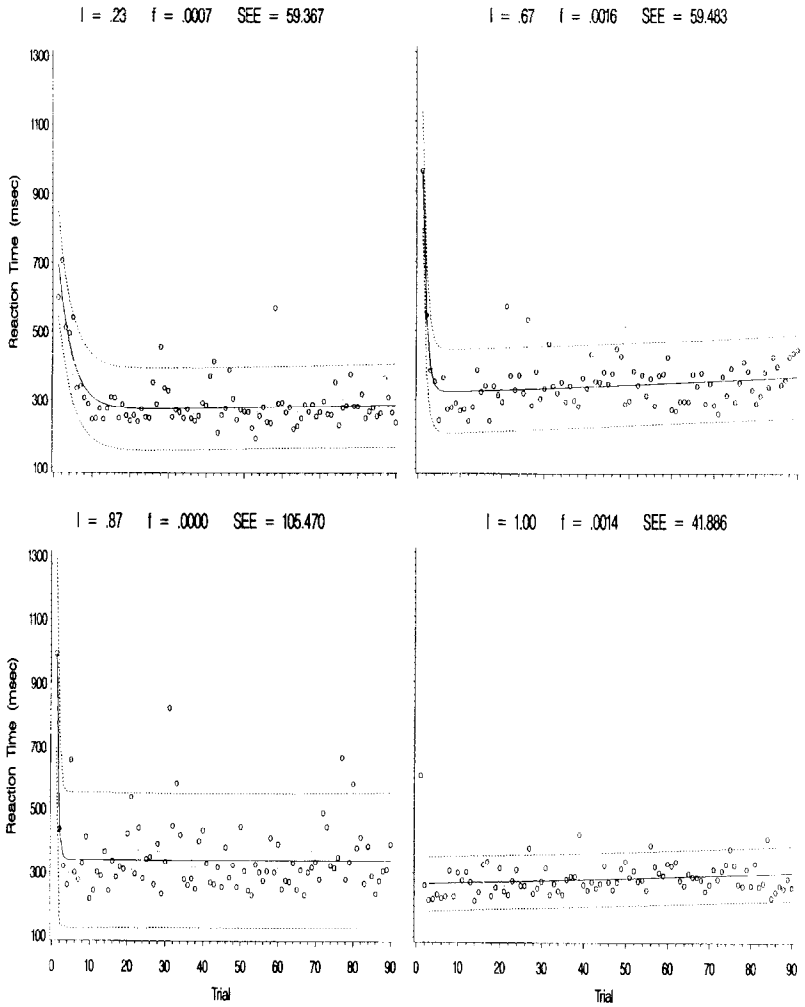

FIG. 1. Examples of data from individual subjects and their fitted regression lines (solid). 95% prediction intervals (dash) and confidence intervals (dot) are also included. l is the estimated learning rate, f is the estimated fatigue rate, and SEE is the standard error of the estimate.

TABLE 1
SUMMARY STATISTICS BY EXPOSURE GROUP

Variable	Group	n	Mean	SD	Min	Max
L_1	Control	78	713	274	178	1186
	Inhibited	43	792	251	269	1106
	Definite	67	758	258	239	1188
	Probable	39	700	258	312	1150
L_∞	Control	78	263	52	175	468
	Inhibited	43	264	56	100	422
	Definite	67	277	69	186	584
	Probable	39	241	42	100	322
l	Control	78	0.6405	0.3407	0.0000	1.0000
	Inhibited	43	0.6483	0.2967	0.0000	1.0000
	Definite	67	0.6593	0.2828	0.0000	1.0000
	Probable	39	0.6749	0.3095	0.0000	1.0000
f	Control	78	0.001448	0.001382	0	0.005020
	Inhibited	43	0.001292	0.001953	0	0.011110
	Definite	67	0.001591	0.001788	0	0.007621
	Probable	39	0.001782	0.002423	0	0.013015
SEE	Control	78	72.40	33.79	20.94	178.97
	Inhibited	43	70.37	34.43	24.69	197.61
	Definite	67	76.70	42.82	23.71	222.45
	Probable	39	67.89	28.62	18.16	130.99
$\overline{R}$	Control	78	283	54	202	458
	Inhibited	43	285	52	208	445
	Definite	67	301	82	210	706
	Probable	39	264	40	199	409
S_R	Control	78	70	34	19	176
	Inhibited	43	68	36	22	199
	Definite	67	76	44	24	236
	Probable	39	65	28	18	135
Age	Control	78	29.9	11.0	17	67
	Inhibited	43	38.4	12.0	19	67
	Definite	67	32.8	9.0	20	69
	Probable	39	35.2	10.0	21	67
Grade	Control	77	10.7	3.6	3	18
	Inhibited	43	10.2	3.9	2	16
	Definite	65	9.1	4.7	0	17
	Probable	39	12.7	2.5	6	17

All means were significantly different from zero, $p = 0.0001$.

sented in Table 2, and regression coefficients are shown in Table 3.

For L_1 there was a significant grade × exposure interaction. The slope of the control group was significantly greater than the inhibited ($p = 0.0372$) and definite ($p = 0.0046$) groups. The slope of the definite group was significantly less than zero ($p = 0.0019$) (see Fig. 2).

There were no statistically significant effects for L_∞ and l.

The age × exposure interaction was significant for f. The slope of the definite group was significantly greater than the control ($p = 0.0111$) group, and it was also significantly greater than zero ($p = 0.0107$) (see Fig. 2).

For SEE there were significant age × exposure and grade × exposure interactions. For age, the slope of the definite group was significantly greater than the control ($p = 0.0008$), inhibited ($p = 0.0161$), and probable ($p = 0.0007$) groups. The slope of the definite group was significantly greater than zero ($p = 0.0066$) (see Fig. 2). For grade, the slope of the probable group was significantly less than the control ($p = 0.0044$), inhibited ($p = 0.0080$), and definite ($p = 0.0103$) groups, and it was significantly less than zero ($p = 0.0013$).

The effect of grade was significant for $\overline{R}$, as the grade increased the mean reaction time decreased. The age × exposure interaction was also significant. The slope of the definite group was significantly greater than the control ($p = 0.0050$), inhibited ($p = 0.0085$), and possible ($p = 0.0220$) groups, and it was also significantly greater than zero ($p = 0.0009$).

For S_R the age × exposure interaction was significant. The slope of the definite group was significantly greater than the control ($p = 0.0004$), inhibited ($p = 0.0051$), and probable ($p = 0.0016$) groups, and it was significantly greater than zero ($p = 0.0012$). The grade × exposure interaction was also significant. The slope of the probable group was significantly less than the control ($p = 0.0076$), inhibited ($p = 0.0077$), and definite ($p = 0.0138$) groups, and it was significantly less than zero ($p = 0.0023$).

DISCUSSION

The components of the performance model come from a family of exponential equations of the form

$$G(t) = A + B(1 + r)^{h(t)}$$

which can be used to model other types of performance. If t is continuous the natural exponential form can be used:

$$G(t) = A + Be^{rh(t)}$$

TABLE 2
LINEAR MODEL SUMMARY TABLE

Dependent Variable	Source	df_N	df_D	F	p
L_1	Exposure	3	212	1.50	0.2154
	Age	1	212	0.00	0.9543
	Grade	1	212	6.73	0.0102
	Age × exposure	3	212	0.67	0.5727
	Grade × exposure	3	212	3.25	0.0227
L_∞	Exposure	3	212	0.59	0.6213
	Age	1	212	0.18	0.6701
	Grade	1	212	3.00	0.0846
	Age × exposure	3	212	1.52	0.2099
	Grade × exposure	3	212	2.32	0.0760
l	Exposure	3	212	0.49	0.6867
	Age	1	212	1.62	0.2048
	Grade	1	212	0.01	0.9140
	Age × exposure	3	212	0.84	0.4708
	Grade × exposure	3	212	0.31	0.8169
f	Exposure	3	212	2.71	0.0463
	Age	1	212	3.35	0.0685
	Grade	1	212	2.71	0.1009
	Age × exposure	3	212	2.66	0.0494
	Grade × exposure	3	212	1.25	0.2922
SEE	Exposure	3	212	5.06	0.0021
	Age	1	212	0.08	0.7717
	Grade	1	212	8.63	0.0037
	Age × exposure	3	212	5.11	0.0020
	Grade × exposure	3	212	2.93	0.0344
$\overline{R}$	Exposure	3	212	1.43	0.2351
	Age	1	212	2.11	0.1480
	Grade	1	212	7.64	0.0062
	Age × exposure	3	212	3.32	0.0206
	Grade × exposure	3	212	1.92	0.1273
S_R	Exposure	3	212	4.69	0.0034
	Age	1	212	0.00	0.9568
	Grade	1	212	7.21	0.0078
	Age × exposure	3	212	5.23	0.0017
	Grade × exposure	3	212	2.73	0.0451

Aldridge (3) has used a natural exponential equation as a model of mastery learning. In either case, r represents a rate and A and B are scaling parameters. A, B, r, and $h(t)$ can be positive, negative, or zero.

Hyperbolic (40) and power (31) functions have also been used to fit learning curves. By equating these functions to $L(t)$, one can show that the hyperbolic function is undefined when there is no learning, and that the power function is undefined when there is one-trial learning. $L(t)$ is defined in both of these circumstances. Bittner (7) used a quadratic polynomial to fit learning curves to estimate long-term individual and system performance. This polynomial provides an estimate of asymptotic performance unconfounded by learning, as does $L(t)$, but it does not provide a learning rate.

If the mean of a measurement is taken over several trials, a change in the mean could represent an overall shift that occurs at each trial or a change in the trend over trials. A change in the SD could represent a change in the variability around a trend or a change in the trend itself. Even if the initial trials are removed from a test, learning can still have an effect on the mean and SD if the learning rate is low or if the difference between initial performance and asymptotic performance is large.

In the example, it was possible to separate the information contained in the mean and SD. There was an effect of education level on mean reaction time that reflected an effect on the initial performance level, and there was a significant age × exposure interaction for mean reaction time that reflected an increase in the fatigue rate. The age × exposure interaction was also significant for the SD of the reaction times, reflecting an effect on the SEE, and the grade × exposure interaction was significant for the SD, reflecting an effect on the initial performance level and the SEE. One can be more certain that there was an actual effect on performance variability if the trends are taken into account.

It should be noted that the focus of the analysis in the example was on the effect of exposure on the linear relationships between age and education level and the dependent variables. If analysis of covariance had been used, the focus would have been on the group means adjusted for the covariates. Such an analysis would have been inappropriate for several of the dependent variables because the assumption of homogeneous slopes was violated.

In the example analysis the definite poisonings showed an increase in the fatigue rate and in performance variability as their age increased, and their initial reaction times were higher if their education level was lower. This group also showed an increase in the mean and SD of the reaction times as age increased. A previous study did not find an effect of organophosphate pesticides on reaction time (35). In their analysis of the data used in the example, Steenland et al. (39) also found no effect. The negative results may be explained by

TABLE 3
ESTIMATES OF THE REGRESSION COEFFICIENTS FOR THE FOUR EXPOSURE GROUPS

Coefficient	Group	Dependent Variable						
		L_1	L_∞	l	f	SEE	$\overline{R}$	S_R
Intercepts	Control	637	260	0.6140	0.002640	91	299	92
	Inhibited	951	255	0.9090	0.001733	78	292	78
	Definite	835	262	0.7730	−0.000819	37	244	31
	Probable	1180	347	0.6697	0.002752	202	389	186
Slopes								
Age	Control	−0.67	0.11	0.0010	−0.000016	0.58	−0.15	−0.62
	Inhibited	0.79	−0.19	−0.0070	0.000002	−0.12	−0.21	0.28
	Definite	3.83	1.55	−0.0010	0.000065	1.49	2.80	1.64
	Probable	−3.56	−0.83	−0.0037	0.000042	−1.06	0.12	−0.78
Grade	Control	9.16	−0.04	−0.0005	−0.000065	−0.12	1.05	−0.23
	Inhibited	−18.50	1.64	0.0009	−0.000036	−0.30	0.07	0.10
	Definite	−21.66	−4.15	−0.0080	0.000031	−1.07	−4.06	−0.99
	Probable	−27.87	−6.08	0.0107	−0.000192	−7.57	−9.53	−7.36

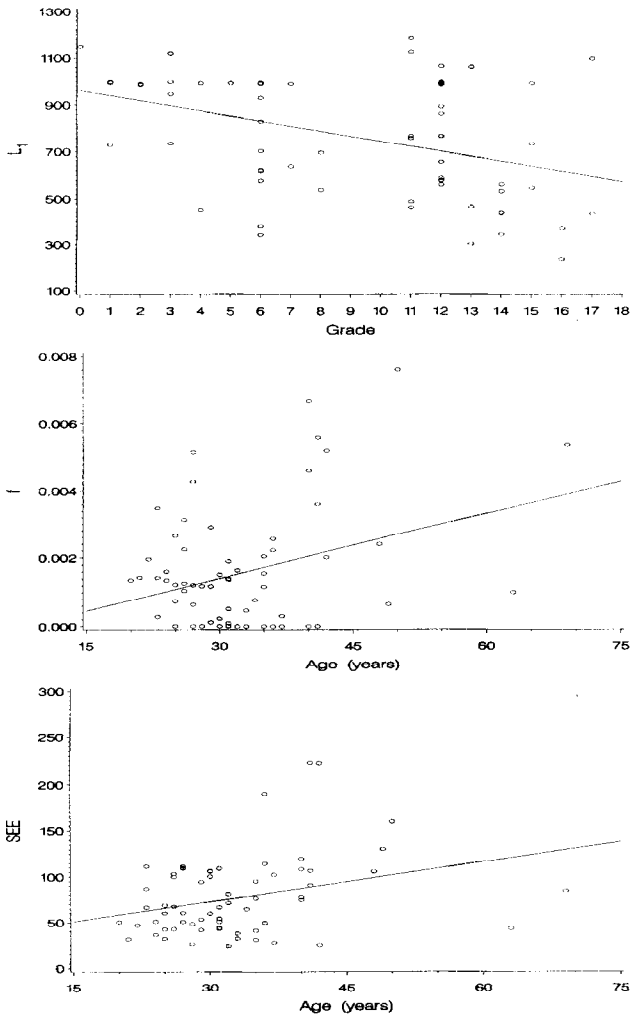

FIG. 2. The effect of education level (grade) on the initial performance level (L_1), and the effect of age on the fatigue rate (f) and performance variability [as measured by the standard error of the estimate (SEE)] in the definite exposure condition.

smaller sample sizes or by different methods of analyzing the data. These investigators looked for differences in group means, or group means adjusted for covariates.

At the present time the biological interpretation of the parameters in the model is not clear. The reaction time task used here involves the eyes, the peripheral and central nervous systems, and striate muscle. The effect of a variable on a model parameter could reflect an action on one or more of these areas. The two trends in the reaction time curves have been provisionally called learning and fatigue because these seem to be the likely processes underlying the trends. It is possible that other processes or a combination of processes may cause the trends, especially the one called fatigue.

Following the interpretation of temporary work decrement by Kohl et al. (23), fatigue in the present reaction time model may, in part, reflect the influence of reactive inhibition, a response-decrementing process viewed by some to reside strictly in the neuromotor apparatus, and by others to be a state of negative motivation capable of supporting inhibitory associative relationships (14,20). Because the usual method of detecting this type of decrement-producing variable is to measure its dissipation over time, it should be possible to design reaction time intervals to control the influence of reactive inhibition. The fatigue component of the present reaction time model would then be expected to differentiate between long and short intertrial intervals.

A second process capable of influencing fatigue in the reaction time response has been termed "inhibition of return" (33), an increased difficulty in directing attention to a previously attended location. Because there is considerable evidence indicating slower reaction times to previously attended targets (32), this potentially combined perceptual–motor process (2) may have influenced the present results independently of other types of inhibition.

Regardless of the details of interpreting the reaction time decrement, future studies may find it useful to apply the present mathematical model to uncover local decrementing trends in the reaction time response. Apart from addressing questions about the mechanisms controlling this response, the technique would permit more sensitive detection of untoward chemical effects on reaction time than has been possible previously.

REFERENCES

1. Abrahams, J. P.; Birren, J. E. Reaction time as a function of age and behavioral predisposition to coronary heart disease. J. Gerontol. 28:471–478; 1973.
2. Abrams, R. A.; Dobkin, R. S. Inhibition of return: Effects of attentional cuing on eye movement latencies. J. Exp. Psychol. Hum. Percept. Perform. 20(3):467–477; 1994.
3. Aldridge, B. G. A mathematical model for mastery learning. J. Res. Sci. Teach. 20:1–17; 1983.
4. Anger, W. K. Worksite behavioral research: Results, sensitive methods, test batteries and the transition from laboratory data to human health. Neurotoxicology 11:629–720; 1990.
5. Angotzi, G.; Camerino, D.; Carboncini, F.; Cassitto, M. G.; Ceccarelli, F.; Cioni, R.; Pradiso, C.; Sartorelli, E. Neurobehavioral follow-up study of mercury exposure. In: Gilioli, R.; Cassito, M. G., eds. Advances in the biosciences. Neurobehavioral methods in occupational health. New York: Pergamon Press; 1983:247–253.
6. Baumeister, A. A.; Kellas, G. Reaction time and mental retardation. In: Ellis, N. R., ed. International review of research in mental retardation. New York: Academic Press; 1968:163–193.
7. Bittner, A. C., Jr. Analysis of learning-curve asymptotes: Ergonomic applications. In: Karwowski, W.; Yates, J. W., eds. Advances in industrial and ergonomic safety III. London: Taylor & Francis; 1991:769–776.
8. Botwinick, J.; Thompson, L. W. Components of reaction time in relation to age and sex. J. Genet. Psychol. 108:175–183; 1966.
9. Cherry, N.; Hutchins, H.; Pace, T.; Waldron, H. A. Neurobehavioral effects of repeated occupational exposure to toluene and paint solvents. Br. J. Ind. Med. 42:291–300; 1985.
10. Cherry, N.; Venables, H.; Waldron, H. A. Description of tests in the London School of Hygiene Test Battery. Scand. J. Work Environ. Health 10(Suppl. 1):18–19; 1984.
11. Diggle, P. J. Time series—a biostatistical approach. Oxford: Oxford University Press; 1990.
12. Dinges, D. F.; Kribbs, N. B. Comparison of the effects of alcohol and sleepiness on simple reaction time performance: Enhanced habituation as a common process. Alcohol Drugs Driving 5(4)/6(1):329–339; 1989–1990.
13. Englund, C. E.; Reeves, D. L.; Shingledecker, C. A.; Thorne, D. R.; Wilson, K. P.; Hegge, F. W. Unified Tri-Service Cognitive Performance Assessment Battery (UTC-PAB) I. Design and specification of the battery (Report No. 87-10). San Diego, CA: Naval Health Research Center; 1987.
14. Eysenck, H. J. A three factor theory of reminiscence. Br. J. Psychol. 56:163–181; 1965.
15. Gamberale, F.; Lisper, H. O.; Anshelm-Olson, B. The effect of styrene vapour on the reaction time of workers in the plastic boat industry. In: Horvath, M., ed. Adverse effects of environmental chemicals and psychotropic drugs. New York: Elsevier Scientific Publishing Company; 1976:135–148.
16. Gamberale, F.; Svensson, G. The effect of anesthetic gases on the psychomotor and perceptual functions of anesthetic nurses. Work Environ. Health 11:108–113; 1974.
17. Hänninen, H.; Lindström, K. Neurobehavioral Test Battery of the Institute of Occupational Health. Helsinki: Helsinki Institute of Occupational Health; 1979.
18. Hanson, C.; Loftus, G. K. Effects of fatigue and laterality on fractionated reaction time. J. Mot. Behav. 10:177–184; 1978.
19. Hogstedt, C.; Hane, M.; Agrell, A.; Bodin, L. Neuropsychological test results and symptoms among workers with well-defined long-term exposure to lead. Br. J. Ind. Med. 40:99–105; 1983.
20. Hull, C. L. Principles of behavior. New York: Appleton-Century; 1943.
21. Johnson, B. L.; Baker, E. L.; El Batawi, M.; Gilioli, R.; Hänninen, H.; Seppäläinen, A. M.; Xintaras, C., eds. Prevention of neurotoxic illness in working populations. New York: John Wiley and Sons; 1987.
22. Johnson, B. L.; Burg, J. R.; Xintaras, C.; Handke, J. L. A neurobehavioral examination of workers from a primary nonferrous smelter. Neurotoxicology 1:561–581; 1980.
23. Kohl, R. M.; Roenker, D. L.; Turner, P. E. Toward a central-peripheral model of work decrement. Percept. Mot. Skills 58:619–624; 1984.
24. Letz, R. The Neurobehavioral Evaluation System: An international effort. In: Johnson, B. L., ed. Advances in neurobehavioral toxicology. Chelsea, MI: Lewis Publishers; 1990:189–201.
25. Liang, Y.-X.; Jin, X.-P.; Lu, P.-Q.; Gu, X.-Q.; Hirata, M.; Sugimoto, K.; Goto, S. A neuropsychological study among workers exposed to low level of carbon disulfide. In: Liang, Y.-X.; Jiang, X.-Z.; Wang, Y.-L., eds. Occupational health bulletin. Shanghai: Shanghai School of Public Health; 1985:142–144.
26. Lindström, K.; Wickstrom, G. Psychological function changes among maintenance house painters exposed to low levels of organic solvent mixtures. Acta Psychiatr. Scand. 67:81–89; 1983.
27. Ljung, G. M.; Box, G. E. P. On a measure of lack of fit in time series models. Biometrika 65:297–303; 1978.
28. Lock, L. K.; Berger, R. A. Influence of sex, presentation order, and trial blocks on young-adults simple and Type-C reaction times. Percept. Mot. Skills 76:1199–1210; 1993.
29. Mutti, A.; Arfini, G.; Ferrari, M.; Muzzucchi, A.; Posteraro, L.; Franchini, I. Chronic styrene exposure and brain dysfunction A longitudinal study. In: Neurobehavioral methods in occupational and environmental health (Environmental Health Document 3). Copenhagen: World Health Organization; 1985:136–140.
30. Myrsten, A.-L.; Andersson, K. Interaction between effects of alcohol intake and cigarette smoking (Reports from the Psychological Laboratories No. 402). Stockholm: University of Stockholm; 1973.
31. Newell, A.; Rosenbloom, P. S. Mechanisms of skill acquisition and the law of practice. In: Anderson, J. R., ed. Cognitive skills and their acquisition. Hillsdale, NJ: Lawrence Erlbaum Associates; 1981:1–55.
32. Posner, M. I.; Cohen, Y. Components of visual orienting. In: Bouma, H.; Bouwhis, D. G., eds. Attention and performance volume 10. Hillsdale, NJ: Erlbaum; 1984:531–556.
33. Posner, M. I.; Rafal, R. D.; Choate, L. S.; Vaughan, J. Inhibition of return: Neural basis and function. Cogn. Neuropsychol. 2:211–228; 1985.
34. Roels, H.; Lauwerys, R.; Buchet, J.-P.; Genet, P.; Sarhan, M. J.; Hanotiau, I.; deFays, M.; Bernard, A.; Stanescu, D. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system, and some biological indices. Am. J. Ind. Med. 11:307–327; 1987.
35. Rosenstock, L.; Keifer, M.; Daniell, W. E.; McConnell R.; Claypoole, K.; The Pesticide Health Effects Study Group. Chronic central nervous system effects of acute organophosphate pesticide intoxication. Lancet 338:223–227; 1991.
36. Searle, S. R. Linear models. New York: John Wiley and Sons; 1971.
37. Spieth, W. Cardiovascular health status, age, and psychological performance. J. Gerontol. 19:277–284; 1964.
38. Spirduso, W. W.; Clifford, P. Replication of age and physical activity effects on reaction and movement time. J. Gerontol. 33:26–30; 1978.
39. Steenland, K.; Jenkins, B.; Ames, R.; O'Mally, M.; Chrislip, D.; Russo, J. Chronic neurologic sequelae to organophosphate pesticide poisoning. Am. J. Public Health 84:731–736; 1994.
40. Thurstone, L. L. The learning curve equation. Psychol. Monogr. 26:1–51; 1919.
41. Tuttle, T. C.; Wood, G. D.; Grether, C. B. Behavioral and neurological evaluation of workers exposed to carbon disulfide (USDHEW [NIOSH] Publication No. 77-128). Cincinnati, OH: NIOSH Publication Office; 1976.
42. Williamson, A. M.; Teo, R. K. C.; Sanderson, J. Occupational mercury exposure and its consequences for behavior. Int. Arch. Occup. Environ. Health 50:273–286; 1982.
43. Xintaras, C.; Burg, J. R.; Tanaka, S.; Lee, S. T.; Johnson, B. L.; Cottrill, C. A.; Bender, J. Occupational exposure to leptophos and other chemicals (USDHEW [NIOSH] Publication No. 78-136). Cincinnati, OH: NIOSH Publication Office; 1978.