DOCUMENT RESUME

ED 191 036 CS 205 749

AUTHOR Watt, James H., Jr.
TITLE Evaluating Causal Models.
PUB DATE Aug 80
NOTE 18p.: Paper presented at the Annual Meeting of the Association for Education in Journalism (63rd, Boston, MA, August 9-13, 1980).

EDRS PRICE MF01/PC01 Plus Postage.
DESCRIPTORS *Communication (Thought Transfer); Communication Research; Evaluation Methods; Influences; *Mass Media; *Media Research; *Models; Relationship; *Research Methodology
IDENTIFIERS *Causal Models

ABSTRACT

Pointing out that linear causal models can organize the interrelationships of a large number of variables, this paper contends that such models are particularly useful to mass communication research, which must by necessity deal with complex systems of variables. The paper first outlines briefly the philosophical requirements for establishing a causal relationship, including the need for a spatial contiguity between cause and effect, the need for temporal priority between cause and effect, and the need for some necessary connection between the cause and effect. It notes that the last stage is often omitted by communication researchers. The paper then describes the stages in causal analysis and relates each to the philosophical requirements. Next, it analyzes three very different studies involving causal analysis. In conclusion, it illustrates the power of the often omitted third stage of causal analysis in detecting errors in causal ordering and linkages. (FL)

****
* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *
****

U.S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE
NATIONAL INSTITUTE OF EDUCATION

THIS DOCUMENT HAS BEEN REPRODUCED EXACTLY AS RECEIVED FROM THE PERSON OR ORGANIZATION ORIGINATING IT. POINTS OF VIEW OR OPINIONS STATED DO NOT NECESSARILY REPRESENT OFFICIAL NATIONAL INSTITUTE OF EDUCATION POSITION OR POLICY

ED191036

EVALUATING CAUSAL MODELS

James H. Watt, Jr.
Department of Communication Sciences
University of Connecticut
Box U-85
Storrs, CT 06268

(203) 486-2628

"PERMISSION TO REPRODUCE THIS MATERIAL HAS BEEN GRANTED BY

James H. Watt, Jr.

TO THE EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)."

Paper presented to the Communication Theory and Methodology Division of the Association for Education in Journalism. Boston, Mass. August 1980.

I would like to acknowledge the aid of Rebecca [illegible] and Richard Allen, both of whom willingly provided access to the [illegible] examples used in this paper would have been much more limited had they not exhibited a highly cooperative attitude in exposing their work to potential criticism.

CS205749

EVALUATING CAUSAL MODELS

The use of linear causal models in mass communication research has been increasing over the past decade. Because these models can organize the interrelationships of a large number of variables, they are particularly useful to communication research which must by necessity deal with complex systems of variables.

Causal analysis is based on the existence of a specific set of conditions between two or more variables. The philosophic requirements for causality provide the assumptions on which causal analysis is based. These requirements imply three different stages in the causal analysis process. But researchers typically carry out only the first two stages, leaving themselves open to possible errors in conclusions drawn from the resulting causal model.

This paper will first briefly outline the philosophical requirements for establishing a causal relationship. The steps in causal analysis will next be described, and each stage will be related to the requirements. Finally, three very different examples of causal analysis will be analyzed. The power of the omitted third stage of causal analysis in detecting errors in causal ordering and linkages will be shown.

REQUIREMENTS FOR CAUSALITY

First, there must be <u>spatial contiguity</u> between the cause and the effect, that is, the cause and effect must exist together in physical space (Mandelbaum, et. al., 1957). This requirement rules out magical "action at a distance".

Second, there must be a <u>temporal priority</u> between cause and effect, with the cause preceeding the effect in time and not vice versa. (Hume, in Mandelbaum, et al., 1957; Mill, in Nagel, 1950). If one changes the level of a cause variable, there is a corresponding change in the effect variable later in time; if the effect variable is man[illegible]ulated, there is no corresponding change in the cause variable.

Third, the cause and effect variables must <u>covary</u>. Changes in the value of the cause variable must produce change[illegible] [illegible]e effect variable which correspond in magnitude. (Mill, in Nagel, [illegible] [illegible]arson, 1911)

Finally, there must be some <u>necessary</u> connec[illegible] [illegible]en the cause and effect variable. Even if all the other condit[illegible] [illegible] mechanism by which the cause produced the effect must [illegible] [illegible] [illegible]ondition rules out accidental covariance or spur[illegible] [illegible] both variables being [illegible] a third variabl[illegible]

This requ[illegible] subject of a [illegible] (in Mandelbau[illegible] [illegible]nd Pearson [illegible] the idea of [illegible]ection in cau[illegible] [illegible] that the mech[illegible] [illegible] was mer[illegible] [illegible]he [illegible] obs[illegible] cause and eff[illegible] [illegible]on, in a[illegible]gume[illegible] [illegible] of Hume's, s[illegible] [illegible]s were [illegible]senti[illegible] [illegible] effect event[illegible] [illegible] to be si[illegible]ilar. [illegible] concluded th[illegible] [illegible]ary co[illegible]necti[illegible] favor of obs[illegible] [illegible]nection (cova[illegible] identical eve[illegible] [illegible] the correl[illegible] this search [illegible]ad [illegible]929) argued [illegible] provided the [illegible]ing the necessary [illegible] is clear th[illegible] [illegible]searcher must have [illegible] [illegible]ishing

between covariance which occurs indirectly as a result of a causal chain involving other variables (and which may have a definite temporal sequence) and covariance which is direct and temporally ordered.

CAUSAL ANALYSIS

Causal analysis is neither a statistical procedure, nor a research design. It is an analysis strategy which is relatively independent of the statistical procedures used and the experimental or observational design employed. Unlike a statistical procedure which is concerned only with establishing covariance between variables, causal analysis is based on the assumption that all conditions for causal relationships are met.

Let us first outline the steps in causal analysis, and relate them to the necessary conditions for establishing causal relationships outlined above.

Causal analysis has three clear-cut stages:

1) Specification of the variables and relationships among them, and the creation of a causal diagram which graphically represents these relationships. There are three fundamental relationships which can exist between variables. They are: a) Causal, which implies the variables meet the conditions of covariance, temporal sequence, and necessary connection; b) Unanalyzed, which implies that the variables meet the condition of covariance, but not the conditions of temporal sequence or necessary connection (the relationship between them might be the result of their common covariance with an unspecified outside variable); and c) Null, implying that none of the conditions hold.

Causal relationships at this stage are simply well-stated hypotheses, of the form "X causes Y, through the mechanism of ...(rationale)...". These can be graphically represeted by drawing an arrow from the cause variable to the effect variable. The arrow points in the direction of the temporal flow (from cause to effect).

Unanalyzed relationships are just sloppy hypotheses, of the form "X and Y will covary". These are represented by a double-headed curved arrow. The two arrowheads symbolize the lack of temporal sequence and the curved line represents the possible spurious nature of the relationship.

Null relationships are null hypotheses in the form of "There is no relationship between X and Y". They are graphically represented by the lack of any line between the two variables.

In this stage of the causal analysis, the conditions of spatial contiguity, temporal priority and necessary connection are met.

Spatial contiguity is met in the researcher's definition of the problem. Cause variables and effect variables are defined so that they are located in the same physical space, and can thus interact with each other by some physical mechanism.

Temporal priority is met in one of three ways:

a) It may be imposed by experimental design. Causal analysis is an excellent way to analyze a complex experimental investigation. The time sequence of variables is under the control of the experimenter and thus can be unambiguously asserted in the causal diagram.

b) It may be obvious. For example, a person's educational level obviously preceeds in time his/her current linguistic abilities. Variables which are not under the control of the researcher, but have obvious temporal ordering, are also easily stated in the causal diagram.

c) It may be tested for plausibility. Here is the place in which causal analysis excells. It is often not possible to manipulate variables to control their temporal ordering, or to clearly determine which variable preceeds the other in time. But it is possible to set up two alternative causal models in which the temporal order of two time-ambiguous variables is reversed. If one of the models is plausible, and the other is not, that is evidence supporting the temporal ordering of the plausible model. If both models are plausible, there is nothing to do except admit the ambiguity.

Note that this way of determining temporal priority requires some method of evaluating the model which results from the first two stages of causal analysis. In current practice, this evaluation is rarely carried out.

Causal analysis also requires that a theoretical linkage or rationale be stated for each causal relationship, as part of the creation of the causal diagram. This fulfills the condition of necessary connection, although it of course does not assure that the rationale is correct.

2) The next step in causal analysis is to estimate the structural coefficients between variables for each of the hypothesized relationships. These coefficients are estimates of the change to be expected in variable Y as a result of variable X.

The coefficients can be estimated a number of ways, but if the variables are at an interval level of measurement, there are some standard, although not universally agreed upon, ways of obtaining these values (Duncan, 1970).

The causal coefficients are often estimated by standardized partial regression coefficients (beta weights). A regression is carried out for each effect variable. In the regression, the effect variable is the dependent variable, and the independent (predictor) variables are all variables which have direct causal links to the dependent variable in the causal diagram. Each beta weight estimates the change to be expected in Y (the dependent variable) as a result of one standard unit change in the X (predictor) variable associated with the beta weight.

Unanalyzed relationships are best estimated with partial correlation. What one must do is hold constant all variables which might intervene between the two variables of interest, or which might serve to coordinate the values of the two (see Heise, 1975, for a cogent set of rules for determining these coordinating paths).

The estimates of covariance coefficients are often incorrect in published causal models. The prevailing convention is to specify as zero value the zero-order correlation between the variables. This is correct only if there are no common causes by which the variables can be related. Typically, exogenous variables are considered to covary with each other in an unanalyzed manner. For example, suppose there are three exogenous variables AGE, SEX, and SES, all of which covary. Specifying the AGE--SEX coefficient as a zero-order correlation is clearly incorrect as this observed correlation includes the covariance path AGE--SES--SEX. What should be specified is the AGE--SEX covariance which is independent of the SES variable. In statistical terms, a partial correlation rather than a zero-order correlation should be used.

Incorrect covariance coefficients will cause errors in the later stages of causal analysis. Fortunately since researchers often do not carry this last stage of the process, faulty specification of the covariance coefficients does not lead to substantial error in interpretation.

This stage of analysis fulfills the requirement of establishing covariance between cause and effect variables. Covariance is estimated by regression beta weights used to compute the causal coefficients and the partial correlation coefficients used to compute the unanalyzed covariance relationships. The condition of covariance is met by significant values of these statistics.

The structure of the causal model created in the first stage will exert a strong effect on the size and significance of these structural weights. This implies that some test for the plausibility of the assumptions of covariance (and non-covariance) made in stage one should be carried out. It rarely is.

3) The last step in causal analysis is to evaluate the adequacy of the causal model. This step involves identifying all the possible paths by which each pair of variables might covary, computing the amount of covariance between the variables which each of these paths contributes, and adding up the results to estimate the total relationship between the two variables. Blalock (1964) presents one procedure for this evaluation based on predicting which partial correlations should vanish when intervening variables are held constant. The procedure suggested here is more general, and seeks to estimate the actual non-zero magnitude of all covariance relationships among variables in the model.

Variables may covary when: a) one causes the other directly; or b) when one causes changes in the other indirectly via another [illegible] or variables; or c) when both variables have a common cause [illegible]. The magnitude of the direct effect of one variable on another [illegible] the structural coefficient. To find the indirect effect of any [illegible] any oth[illegible] must multiply the causal coefficients for all i[illegible] steps in [illegible]

[illegible] covariance when variables share a common ca[illegible] is somewhat hard[illegible], but a simple set of two rules for determining co[illegible]ting paths can [illegible]ated:

a [illegible]ne may travel in a reverse causal direction in [illegible]ding a path, ther[illegible]h to a forward causal direction. But one may [illegible] then travel [illegible] reverse causal direction. This rule is simply a [illegible]chanical way [illegible] a common cause for the two variables under con[illegible]ration. The [illegible] which causal direction is reversed from backwa[illegible] to forwa[illegible] [illegible]mmon cause. To allow a second reversal would permit an effect [illegible]e modify a cause variable, which would violat[illegible] the temporal [illegible]cy condition.

b) Unanalyzed covariance paths, since they have no temporal direction [illegible]fied, may be traversed in either direction.

Two variables may be related by a number of paths. The basic [illegible]tion of linearity in causal models allows one to compute the total [illegible] of one variable on another by just adding the path values for all [illegible] different paths by which they are coordinated. When one has done this, the result is an estimate of the zero-order correlation, based on the assumptions of the casual diagram, and the estimates of the stuctural coefficients.

If the structure of the relationships among variables is accurately represented by the causal diagram, this value should correspond to the observed zero-order correlation. Values which are substantially different from the observed values indicate that the structure of the causal model is not supported by the observed data.

If one makes the assumption that the correlations computed from the causal diagram are really population values (that is, that the causal model is correct), tests for differences between observed and population correlations can be made by simple Fisher r-to-Z tests. In these tests, a non-significant result indicates that the computed and observed values are sufficiently close to have come from the same population and thus the model structure relating the two variables is not incorrect.

It is important to realize that the model structure has not been proven by these tests. There may be a large number of models which will accurately reproduce the zero-order correlations. However, if significant differences between the computed and observed values are found, the model which produced them can be rejected. Thus the procedure tests the plausibility, rather than the adequacy of the causal model. Implausible models can be rejected, but plausible ones must be further evaluated.

One way to test the overall adequacy of a model is to determine the average accuracy of the reproduced correlations, as compared to the observed correlations. A "goodness-of-fit" figure can be obtained by squaring the correlations to get estimates of the covariances, finding the absolute value of the difference between the computed and observed covariance, and averaging this value over all pairs of variables. Thus a model which receives a .85 goodness-of-fit rating has an average of 15% error in predicting the covariance of any pair of variables. A model which receives a .95 rating would clearly be a better representation of the data, even if both models were plausible.

EXAMPLES OF CAUSAL MODELS WITH EVALUATION

The first two stages of analysis are usually carried out by communication researchers, but the model evaluation stage rarely is. This stage actually tests the implications of the assumptions about the structure of the model made by the researcher. Any model structure may be specified in the first stage, and structural coefficients can be obtained for it in the second stage. But with the evaluation stage, the resulting model may be found to violate one or more of the conditions of causality. Obviously, conclusions drawn from a model in violation of the requirements of causality are suspect.

Three examples of causal models based on real data will be used to illustrate the additional information provided by the evaluation stage.

The first example is from a study of nonverbal communication impairment in brain-damaged patients (Duffy, Watt and Duffy, 1978, 1979). This may seem a bit removed from mass communication research, but the analysis situations are actually very similar: experimental manipulation is pragmatically and ethically impossible, so all causal statements must be made from observational data; there are a number of variables involved; and the causal ordering of some of the variables is a subject of controversy. The other two examples are from mass communication research in two different areas: political communication and subcultural media use.

Nonverbal Communicative Impairment Models.

The first three figures contain three models causally relating the following variables: PICA (Porch Index of Communicative Abilities), an index of generalized symbolic ability; RPM (Ravens Progressive Matrices), a measure of general intelligence; MAT (Manual Apraxia Test), a motor skills test; PRT (Pantomime Recognition Test), a nonverbal recognition abilities index; and PET (Pantomime Expression Test), a nonverbal expression index.

Figure 1 represents one theoretical perspective on the origin of nonverbal communication deficits. Simply put, it states that deficiencies in general symbolic abilities, intelligence, and manual skills all occur simultaneously as a result of a brain lesion due to injury or stroke, and thus there is no causal ordering among them. They covary because they are a result of a common cause. These variables in turn cause deficits in nonverbal abilities.

As the table associated with this model shows, this is a plausible causal statement. Using the previously outlined rules to determine unique causal paths, then adding the effects of all paths between pairs of variables, produces estimates of the observed correlations which do not differ significantly from the actual correlations. In traditional causal analysis, this information would not be available. Nor would the goodness-of-fit value. Conclusions drawn would be based upon the magnitude and possibly the significance levels of the structural coefficients obtained from the second stage of analysis, and the assumed causal structure would be tacitly accepted.

But because we have this additional evaluative information, it is possible to contrast the first model with a theoretically competing one. Figure 2 shows this model. It states that the predominant cause variable is general intelligence. Deficits in intelligence caused by brain lesion cause reductions in symbolic ability and motor skills, which subsequently cause deficiencies in nonverbal skills.

The table associated with this model shows that this is not a plausible causal model, as the covariances predicted by the model differ significantly from the observed correlations. Clearly, Model 1 is a better representation of the observed facts.

Figure 3 represents another theoretical perspective. It merely reverses the causal order of the intelligence and symbolic abilities variables. The rationale here is that symbolic abilities are central to any conceptualization of intelligence, and changes in symbolic ability thus cause changes in measured intelligence. As the table shows, this is a plausible statement. The goodness-of-fit value also indicates that it is a better statement of the observed data than is Model 1.

Comparing the results of the second and third models leads to the conclusion that deficits in symbolic abilities probably cause deficits in general intelligence, and not vice versa. We are in the rather astounding position of being able to offer evidence for causal ordering from observational data taken at a single time point. It should be emphasized that this is possible only if the evaluation stage of analysis is carried out. The structural coefficients of the second model could be used to draw conclusions, but these conclusions would be in error, as they would be based on a causal structure which was not capable of representing the observed data.

Adolescents and Political Mass Communication

This causal analysis was carried out by Quarles (1979). Figure 4 is the causal diagram for this model of the antecedents of accurate perceptions of candidates' positions on issues. Education (EDUC), Political interest of respondents (POLI) and interest in the campaign by respondents (CAMI) are assumed to be exogenous and to covary. The values reported in the diagram are (incorrect) zero-order coefficients. They were replaced by partial correlations for the analysis.

Increases in any of these exogenous variables are assumed to cause increases in newspaper use (NEWU), which in turn causes increases in political system knowledge, such as knowledge of the length of terms of senators, the name of representatives, etc. (SYSK). Campaign interest and education are also assumed to cause increases in political system knowledge. All variables are presumed to cause increases in the accuracy with which respondents could identify campaign issues (ACCU).

As the table associated with Figure 4 shows, this is a very plausible model. None of the predicted zero-order correlations differs significantly from the observed correlations, and the fit of this model to the data is excellent, with an average covariance error of less than 1%.

The evaluation of this model has added some credence to the assumptions of temporal priority which were made by the researcher. There are a number of other time orderings which could be justified, however. For example, a plausible argument could be made for reversing the time ordering of newspaper use and political system knowledge. A stronger case for the causal conclusions which were drawn from this data could be made if the alternatives were investigated and found to be poorer at predicting the observed data.

Use of Television By Blacks

Allen and Bielby (1979) provide the next example. This causal model, outlined in Figure 5, is an example of a very complex situation. Four exogenous variables are defined, education (EDUC), socioeconomic status (SES), AGE and SEX, all of which are assumed to covary. The covariance coefficients were not reported in the original research, but were reconstructed as partial correlations from the zero-order correlation matrix and entered in the model evaluation stage.

Education is assumed to have a direct causal effect on all other variables, which were black identity (BLID), the sense of personal control over life (PERC), anomie, or alientation from white society (ANOM), total television-viewing (TOTV), black oriented public affairs television viewing (BLPA), and the perceived bad points of black situation comedies (BADP). Black identity is a function of age and education, and in turn produces effects in total television viewing and perceived bad points. Personal control is a function of education and SES and produces changes in total television viewing levels. Alienation is a function of education and sex, and causes differences in black public affairs viewing. All variables with the exception of SES, personal control, and anomie produce effects in the the perception of bad points of black situation comedies.

The causal ordering is justified by the authors, fulfilling the necessary connection condition for causality. But there remain a number of rather arbitrary causal relationships. For example, it could be argued that black public affairs viewing should preceed black identity, as in a learning process. As the model is stated, there is no relationship at all between black identity and black public affairs viewing.

It is particularly necessary to carry out the evaluation stage in a complex model with non-obvious structures like this one. As the results in Figure 5 show, the authors of this model did an admirable job of creating a structure which was consistent with the reality of the observed data. However, there is one striking difference between the correlations observed and those predicted by the model. The observed relationship between personal control and anomie was -.33, but the model predicted a correlation

of only -.02! A look at the structure of the model indicates why: anomie and personal control are assumed to be unrelated, so any correlation between the variables should be spurious, i.e., the result of covariance with common cause variables.

The spurious paths could not provide covariance of the magnitude observed, so it appears that there might be a real relationship between the two variables which was overlooked by the authors. It is certainly plausible to think that a sense of personal control and a feeling of anomie should be directly related in a negative fashion.

It would be interesting to recompute the structual coefficients with the addition of this causal path, and then to reevaluate the model. To be sure, this is a somewhat atheoretical way of approaching causal modeling. But it is also unsatisfying to be in the situation of drawing conclusions from a model with a known flaw.

CONCLUSIONS

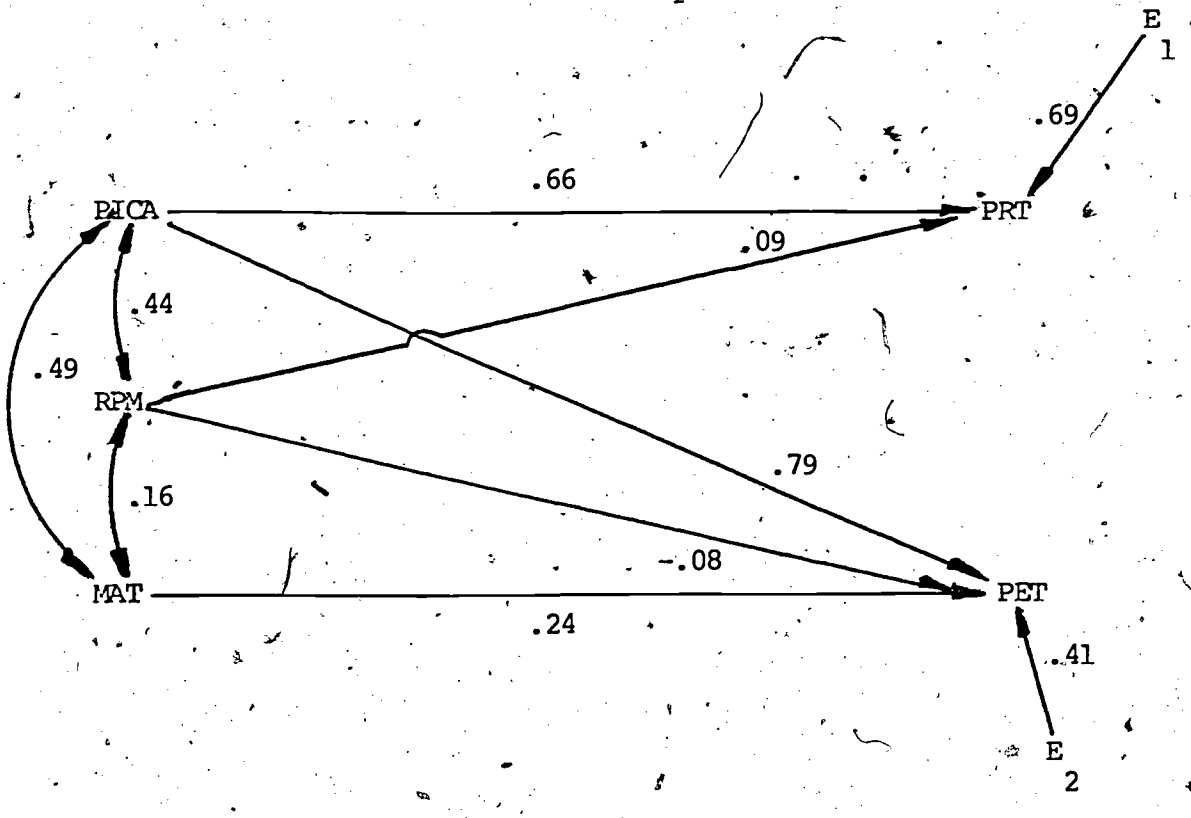
These examples have illustrated the additional power which evaluation of models brings to causal analysis. It enables the researcher to reject implausible model structures, to find evidence of temporal ordering of variables, to detect missing relationships in models, and possibly to permit self-congratulation for creating a good theoretical model.

It seems clear that the philosophical requirements for establishing casual relationships imply that this evaluation stage should be carried out. The conclusions drawn from a causal model are only as good as the structure of the model. It is necessary that researchers test the plausibility of the assumptions made in creating the structure, lest they draw conclusions and implications from an invalid set of postulates.

Except in the simplest models, this evaluation must be carried out with the aid of a computer. The number of indirect paths by which variables can be related increases exponentially with the number of causal and unanalyzed links included in the model. As an example, the Allen and Bielby model produced about 8000 coordinating paths between variables. Reproduced correlations and significance tests for the models in this paper were produced by a computer program which is available from the author upon request.

FIGURE 1

Nonverbal Communication Impairment — Model 1



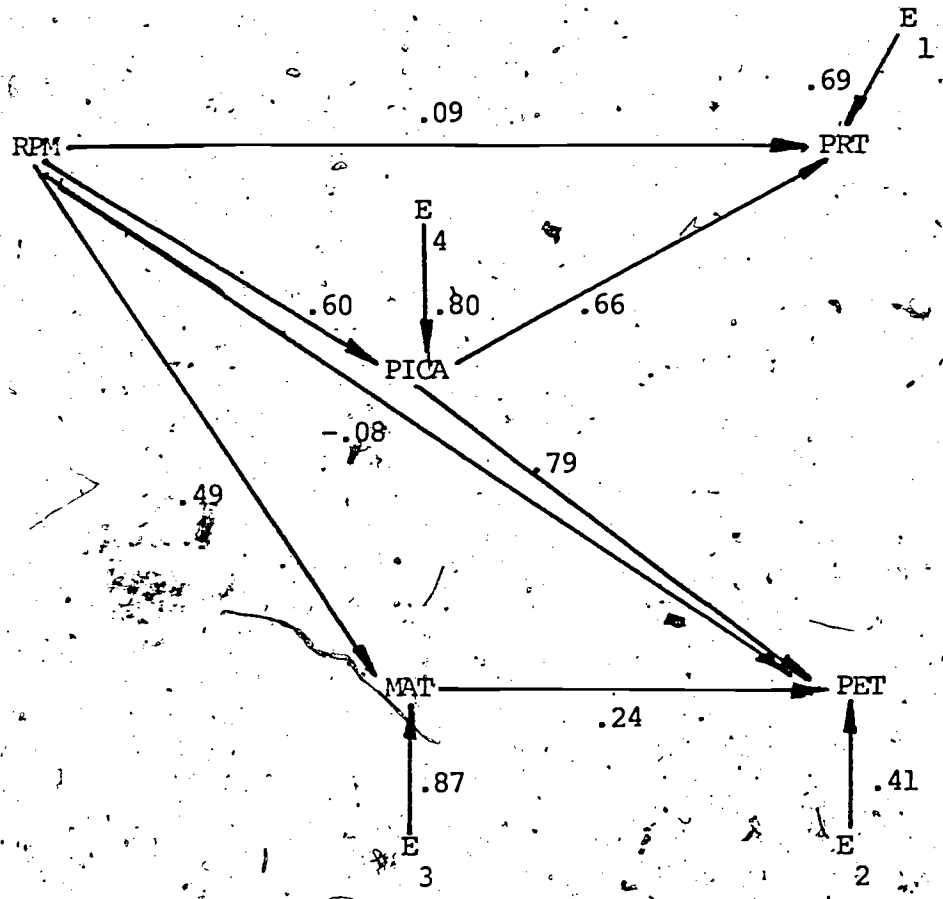
ORIGINAL AND REGENERATED CORRELATION COEFFICIENTS

	PRT	RPM	PICA	PET	MAT
PRT	1.000	0.489	0.716	0.676	0.346
REGEN	1.000	0.432	0.707	0.621	0.404
SIG	1.000	0.648	0.905	0.545	0.671
RPM	0.489	1.000	0.599	0.511	0.490
REGEN	0.432	1.000	0.518	0.420	0.376
SIG	0.648	1.000	0.458	0.460	0.372
PICA	0.716	0.599	1.000	0.892	0.638
REGEN	0.707	0.518	1.000	0.883	0.560
SIG	0.905	0.458	1.000	0.789	0.443
PET	0.676	0.511	0.892	1.000	0.703
REGEN	0.621	0.420	0.883	1.000	0.653
SIG	0.545	0.460	0.789	1.000	0.556
MAT	0.346	0.490	0.638	0.703	1.000
REGEN	0.404	0.376	0.560	0.653	1.000
SIG	0.671	0.372	0.443	0.556	1.000

GOODNESS OF FIT = 0.9368

FIGURE 2

Nonverbal Communication Impairment -- Model 2



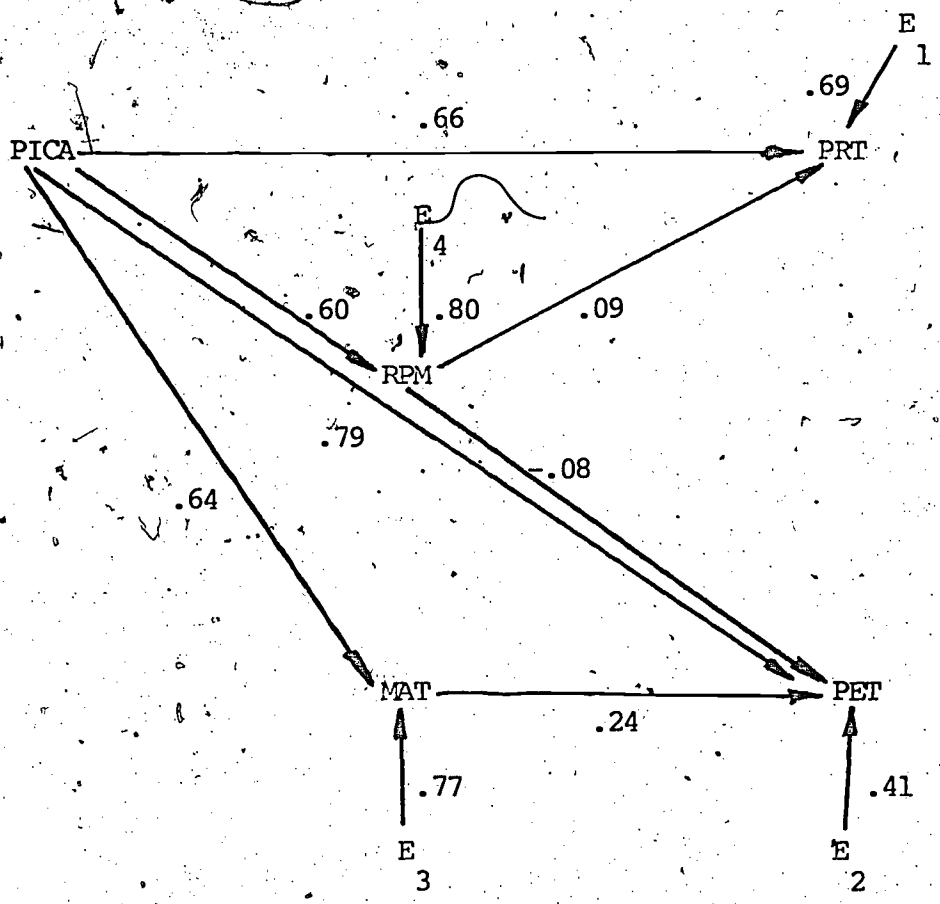
ORIGINAL AND REGENERATED CORRELATION COEFFICIENTS

	PRT	RPM	PICA	PET	MAT
PRT	1.000	0.489	0.716	0.676	0.346
REGEN	1.000	0.486	0.714	0.582	0.238
SIG	1.000	0.982	0.982	0.325	0.455
RPM	0.489	1.000	0.599	0.511	0.490
REGEN	0.486	1.000	0.600	0.512	0.490
SIG	0.982	1.000	1.000	1.000	1.000
PICA	0.716	0.599	1.000	0.892	0.638
REGEN	0.714	0.600	1.000	0.813	0.294
SIG	0.982	1.000	1.000	0.061	0.004
PET	0.676	0.511	0.892	1.000	0.703
REGEN	0.582	0.512	0.813	1.000	0.433
SIG	0.325	1.00	0.061	1.000	0.010
MAT	0.346	0.490	0.638	0.703	1.000
REGEN	0.238	0.490	0.294	0.433	1.000
SIG	0.455	1.000	0.004	0.010	1.000

GOODNESS OF FIT = 0.9049

FIGURE 3

Nonverbal Communication Impairment -- Model 3



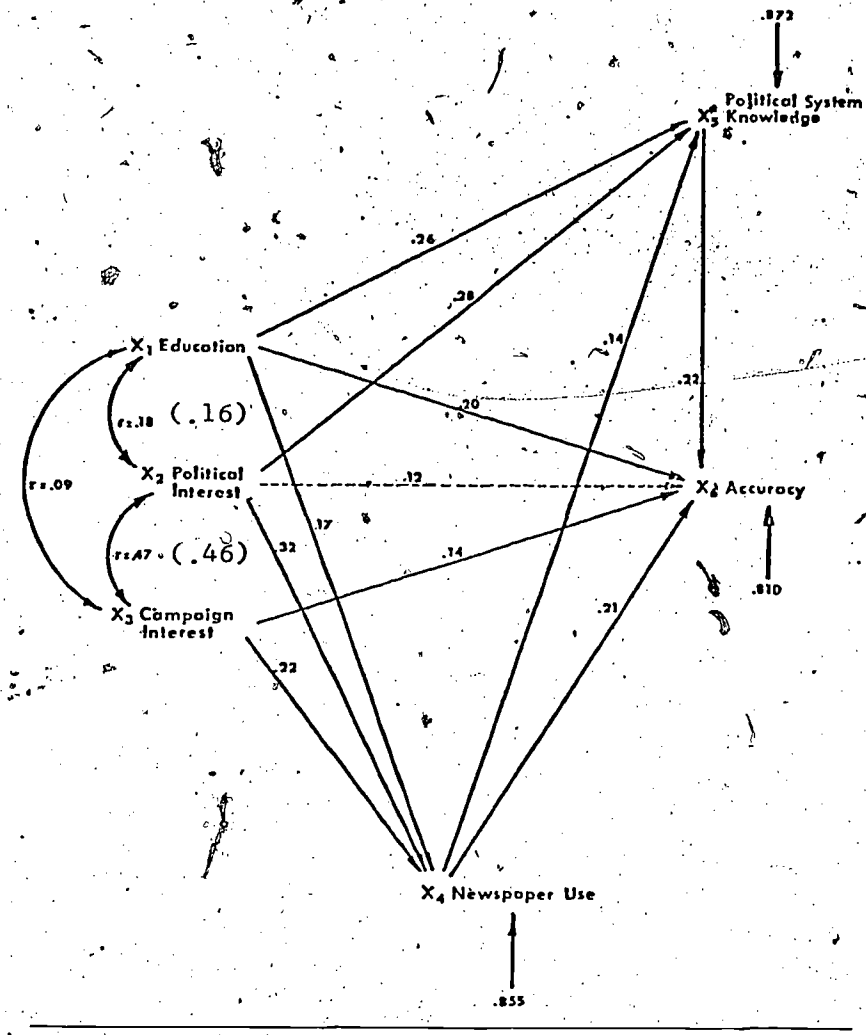
ORIGINAL AND REGENERATED CORRELATION COEFFICIENTS

	PRT	RPM	PICA	PET	MAT
PRT	1.000	0.489	0.716	0.676	0.346
REGEN	1.000	0.486	0.714	0.635	0.457
SIG	1.000	0.982	0.982	0.648	0.402
RPM	0.489	1.000	0.599	0.511	0.490
REGEN	0.486	1.000	0.600	0.486	0.384
SIG	0.982	1.000	1.000	0.834	0.407
PICA	0.716	0.599	1.000	0.892	0.638
REGEN	0.714	0.600	1.000	0.896	0.640
SIG	0.982	1.000	1.000	0.910	0.986
PET	0.676	0.511	0.892	1.000	0.703
REGEN	0.635	0.486	0.896	1.000	0.715
SIG	0.648	0.834	0.910	1.000	0.880
MAT	0.346	0.490	0.638	0.703	1.000
REGEN	0.457	0.384	0.640	0.715	1.000
SIG	0.402	0.407	0.986	0.880	1.000

GOODNESS OF FIT = 0.9704

FIGURE 4

Adolescents and Newspaper Use



Path Analysis for Young Madison Sample Using Newspaper Use as Media Indicator

NOTE: Figures in parentheses are partial correlations which replaced the zero-order correlations in tests of the model.

FIGURE 4 (Continued)

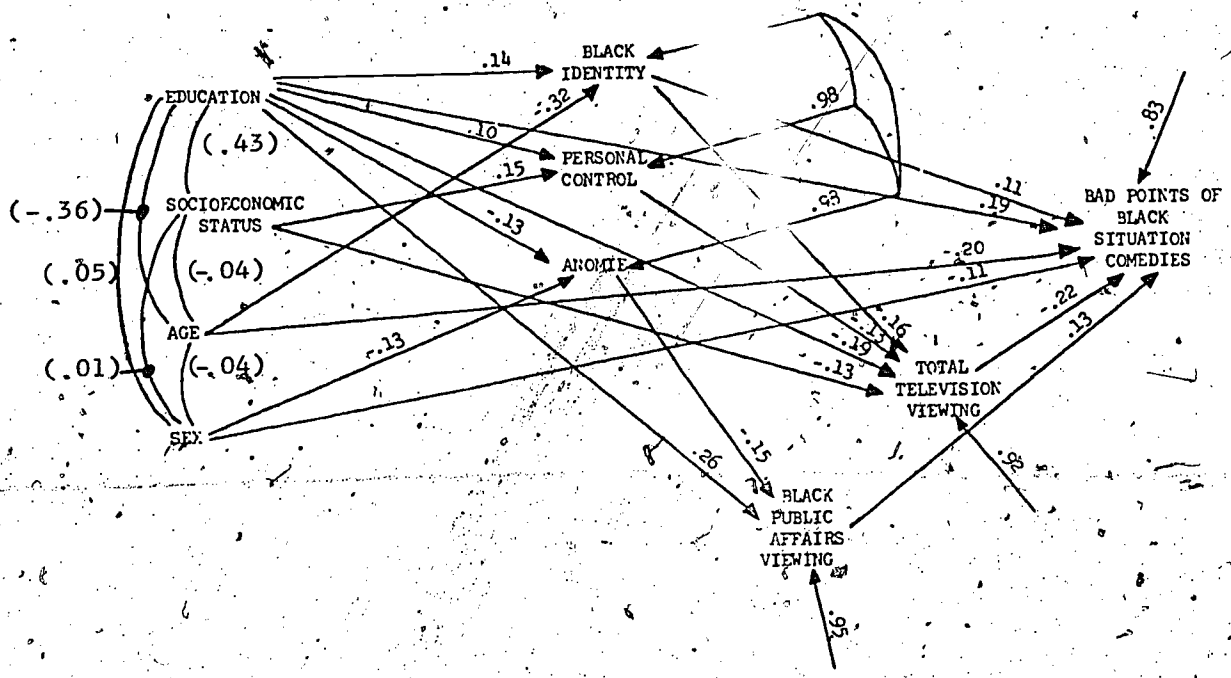
ORIGINAL AND REGENERATED CORRELATION COEFFICIENTS

	ACCU	EDUC	POLI	CAMI	SYSK	NEWU
ACCU	1.000	0.360	0.400	0.320	0.420	0.440
REGEN	1.000	0.355	0.396	0.337	0.431	0.437
SIG	1.000	0.929	0.943	0.787	0.843	0.961
EDUC	0.360	1.000	0.180	0.090	0.340	0.250
REGEN	0.355	1.000	0.160	0.079	0.338	0.239
SIG	0.929	1.000	0.763	0.870	0.978	0.859
POLI	0.400	0.180	1.000	0.470	0.390	0.450
REGEN	0.396	0.160	1.000	0.464	0.384	0.449
SIG	0.943	0.763	1.000	0.911	0.924	1.000
CAMI	0.320	0.090	0.470	1.000	0.110	0.380
REGEN	0.337	0.079	0.464	1.000	0.204	0.382
SIG	0.787	0.870	0.911	1.000	0.160	0.975
SYSK	0.420	0.340	0.390	0.110	1.000	0.330
REGEN	0.431	0.338	0.384	0.204	1.000	0.328
SIG	0.843	0.978	0.924	0.160	1.000	0.972
NEWU	0.440	0.250	0.450	0.380	0.330	1.000
REGEN	0.437	0.239	0.449	0.382	0.328	1.000
SIG	0.961	0.859	1.000	0.975	0.972	1.000

GOODNESS OF FIT = 0.9941

FIGURE 5

Use of Television by Blacks



Path Diagram of Determinants of Perceived Bad Points of Black Situation Comedies among Black Adults

NOTE: Figures in parentheses are partial correlations which replaced the zero-order correlations in tests of the model.

FIGURE 5 (c ed)

ORIGINAL AND REGENERATED CORRELATION COEFFICIENTS

	EDUC	SES	AGE	SEX	BLID	PERC	ANOM	BLPA	TOTV	BADP
EDUC	1.000	0.470	-0.393	0.089	0.270	0.169	-0.140	0.286	-0.319	0.395
REGEN	1.000	0.441	-0.354	0.074	0.253	0.166	-0.140	0.281	-0.310	0.385
SIG	1.000	0.554	0.465	0.811	0.772	0.963	1.000	0.929	0.864	0.851
SES	0.470	1.000	-0.217	0.053	0.164	0.197	-0.105	0.178	-0.274	0.244
REGEN	0.441	1.000	-0.184	0.042	0.121	0.194	-0.083	0.124	-0.258	0.202
SIG	0.554	1.000	0.572	0.860	0.471	0.963	0.490	0.370	0.784	0.474
AGE	-0.393	-0.217	1.00	075	-0.381	-0.057	0.017		0.202	-0.371
REGEN	-0.354	-0.184	1.00	064	-0.370	-0.063	0.054		0.159	-0.349
SIG	0.465	0.572	1.00	858	0.829	0.922	0.542		0.465	0.679
SEX	0.089	0.053	-0.075	1.000	0.129	0.057	-0.141	0.035	-0.020	-0.050
REGEN	0.074	0.042	-0.064	1.000	0.031	0.014	-0.140	0.040	-0.026	-0.069
SIG	0.811	0.860	0.858	1.000	0.108	0.481	0.986	0.932	0.918	0.761
BLID	0.270	0.164	-0.38	0.129	1.000	-0.031	-0.060	0.151	-0.228	0.296
REGEN	0.253	0.121	-0.37	0.031	1.000	0.043	-0.03	0.071	-0.229	0.288
SIG	0.772	0.471	0.829	0.108	1.000	0.226	0.70	0.190	0.982	0.893
PERC	0.169	0.197	-0.057	0.057	-0.031	1.000	-0.32	0.106	-0.184	0.130
REGEN	0.166	0.194	-0.063	0.014	0.043	1.000	-0.02	0.047	-0.194	0.096
SIG	0.963	0.963	0.922	0.481	0.226	1.000	0.000	0.332	0.869	0.577
ANOM	-0.140	-0.105	0.017	-0.141	-0.060	-0.326	1.000	-0.184	0.132	-0.085
REGEN	-0.140	-0.063	0.054	-0.140	-0.037	-0.023	1.000	-0.186	0.044	-0.060
SIG	1.000	0.490	0.542	0.986	0.707	0.000	1.000	0.969	0.147	0.682
BLPA	0.286	0.178	-0.118	0.035	0.151	0.106	-0.184	1.000	-0.184	0.264
REGEN	0.281	0.124	-0.100	0.040	0.071	0.047	-0.186	1.000	-0.087	0.226
SIG	0.929	0.370	0.771	0.932	0.190	0.332	0.969	1.000	0.108	0.511
TOTV	-0.319	-0.274	0.202	-0.020	-0.228	-0.184	0.132	-0.184	1.000	-0.366
REGEN	-0.310	-0.258	0.159	-0.026	-0.229	-0.194	0.044	-0.087	1.000	-0.344
SIG	0.864	0.784	0.465	0.918	0.982	0.869	0.147	0.108	1.000	0.684
BADP	0.395	0.244	-0.371	-0.050	0.296	0.130	-0.085	0.264	-0.366	1.000
REGEN	0.385	0.202	-0.349	-0.069	0.288	0.096	-0.060	0.226	-0.344	1.000
SIG	0.851	0.474	0.679	0.761	0.893	0.577	0.6	0.511	0.684	1.000

GOODNESS OF FIT = 0.9897

REFERENCES

Allen, R.L. and W.T. Bielby (1979) "Blacks' attitudes and behaviors toward television." Communication Research 6:437-462.

Blalock, Hubert M. (1964) Causal Inferences in Nonexperimental Research. Chapel Hill, NC: University of North Carolina Press.

Duffy, Joseph R., J. Watt and R.J. Duffy. (1978) "Deficits of nonverbal communication in aphasic patients: a test of some current causal theories." Presented at the Eighth Annual Clinical Aphasiology Conference, Keystone Colorado.

Duffy, Joseph R., J. Watt and R.J. Duffy (1979) "Pantomime Impairment in aphasia: path analysis of proposed causes." Presented to the American Speech-Language-Hearing Association conference, Atlanta.

Duncan, O.D. (1970) "Partials, partitions, and paths," in E.F. Borgatta and G.W. Bohrnstedt (eds) Sociological Methodology 1970. San Francisco: Jossey-Bass.

Heise, D.R. (1975) Causal Analysis. New York: Wiley.

Quarles, R. (1979) "Mass media use and voting behavior: the accuracy of political perceptions among first-time and experienced voters." Communication Research 6:407-436.

Hume, D. (1957) "An inquiry concerning human understanding.," in M. Mandelbaum, F. Gramlich, and A. Anderson (eds) Philosophic Problems. New York: Macmillan.

Mandelbaum, M., F. Gramlich, and A. Anderson (1957) "Problems in scientific method," in M. Mandelbaum, F. Gramlich and A. Anderson (eds) Philosophic Problems. New York: Macmillan.

Mill, J.S. (1950) "Systems of logic," in E. Nagel (ed) Philosophy of scientific method. New York: Hafner.

Pearson, K. (1911) The Grammar of Science. London: Adam and Charles Black.

Whitehead, A.N. (1929) Process and Reality. New York: Macmillan.