Stone & KWS '81

Structural Modeling of the Relations Among Intelligence and Illness Variables

The present study examined the relationship between diagnosis-derived illness variables and intellectual functioning in a 3-occasion, 14-year longitudinal panel. A system of linear structural equations was used to replicate an "autoregressive model" of cross-occasion intelligence (g) and to test hypothesized structural relationships among illness variables and g. Three of the several concatenated disease variables proved to be significant predictors of intellectual functioning--circulatory disorders, neoplasmas, and musculoskeletal disorders. It was particularly interesting that the illness variables predicted variance in intelligence at Times 2 and 3 of the longitudinal sequence indicating that they were related to time-related change in adult cognitive performance.

No topic in the field of adult development has received more attention than that of cognitive abilities and the developmental progression of cognitive performance over the life span. While general or average trends have been reported and replicated (see Botwinick, 1977; Schaie, 1979), a number of investigators have noted the wide individual differences in life-span cognitive development (Botwinick, 1978; Baltes & Labouvie, 1973). Unfortunately, little research in this area has progressed beyond the descriptive level; there have been few attempts to explain developmental differences intelligence in terms of other variables which might exert a causal influence on overall level of functioning or performance change.

One explanation for decline in cognitive functioning among the aged rests on the increased pathology in later years. It is estimated that 75 percent of persons 65 and over have one or more chronic illnesses (Siegler, Nowlin & Blumenthal, 1980) and this percentage increases dramatically in the upper age brackets (Gruenberg, 1979). Cardiovascular diseases are the primary causes of death in older people. Thus, a major area of interest for geropsychologists concerns the relationship between cardiovascular processes and psychological functioning (see Simonson & Enzer, 1941; Jalavisto (1964/65); Wilkie & Eisdorfer, 1971; Spieth, 1964; Schultz, Dineen, Elias, Pentz & Wood, 1979; Hertzog, Schaie & Gribbin, 1978; Hertzog, Notel 1).

In a few studies intellectual functioning has been related to omnibus measures of health. Klonoff & Kennedy (1966) compared WAIS performance in hospitalized and community-dwelling residents in

their 80's and 90's. The latter group, which evidenced fewer complaints and considerably better health, scored significantly higher on the WAIS than did the hospital group, across all eleven subtests.

In a somewhat similar study, Correll, Rokorz, and Blanchard (1966) found that the presence of both cardiovascular and cerebrovascular signs was associated with lower intellectual performance than either of these conditions alone. Overall health status made a major contribution to the R^2 only among a group of hospitalized patients, thereby supporting Birren's (1965) hypothesis that physiological factors play a minimal role in determining psychological functioning in normal ranges, but may show a greater relationship in disease states.

Taken together, the various studies relating health variables to cognitive functioning suggest a functional relationship between cardiovascular symptoms or health status and cognitive performance. However, with only a few notable exceptions (i.e. Wilkie & Eisdorfer, 1971; Hertzog, Schaie & Gribbin, 1978; Hertzog, Note 1) the existing research has utilized cross-sectional methodologies, rather than examining change over time.

In the present study, the influence of illness variables on cognitive functioning is examined by applying structural modeling techniques to longitudinal health and cognitive data spanning 14 years. It is predicted that illness variables will be negatively associated with both initial status cognitive performance and to change over time in cognitive performance. It is further expected that this relationship will be stronger among older relative to

younger subjects since greater cognitive change is seen in later life and because this is the period of the life span where the prevalence of pathology sharply rises.

Subjects. Subjects were first sampled in 1963 from the membership rolls of a health maintenance organization in the northwestern part of the United States. There were two subsequent 7-year follow-ups, so that there is 3-occasion longitudinal data available for 253 of the original 995 subjects tested in 1963.

Observed variables. Cognitive functioning at each measurement occasion was assessed based on the five subtests of the Primary Mental Abilities Test (PMA) (Thurstone & Thurstone, 1949). These include Verbal Meaning (V), Space (S), Reasoning (R), Number (N), and Word Fluency (W).

Illness variables were derived from the diagnoses recorded on each subject's medical history. (Since the subjects in this study received virtually all their medical care from the health maintenance organization which constitutes our population, detailed medical histories were available for all subjects.) Diagnoses made by the physician at the time of each clinic visit or hospital stay were translated by medical librarians using the International Classification of Diseases (ICDA). While this medical data contains a wealth of information, reducing it to variables suitable for parametric data analyses presents a number of problems. For example, frequency data for specific disease codes yield severely skewed J-shaped distributions. Furthermore, there is no way of assessing physicians' reliabilities in assigning diagnoses. To

address these issues, diagnosis codes from related categories were collapsed into 16 categories, which follow the major division of the ICDA. Eleven of these were sufficiently represented in the sample under study to warrant investigation. The original coding scheme listed diagnosed incidents of illness by year. In order to reduce the data further and keep it compatible with the PMA measurement occasions, year-by-year incidents were combined into three 7-year time frames, corresponding to the assessment dates for the PMA.

Structural modeling. The basis of structural modeling is a series of simultaneous regression equations involving latent variables. Latent variables are hypothetical constructs which cannot be directly observed or measured, but which can be inferred from other directly observable variables by means of a factor analysis. LISREL IV (Joreskog & Sorbom, 1978) was used to derive the latent variables and to test hypothesized structural models.

RESULTS AND DISCUSSION

Longitudinal measurement model for the PMA. A LISREL IV replication of the PMA single-factor model derived by Hertzog (Note 1) was first tested. This is a 3-occasion "autoregressive model" (Joreskog & Sorbom, 1978) in which all five PMA subtests load on a single factor at each measurement occasion. Test-specific correlated residuals were permitted in order to allow for test-specific reliable variance not associated with the factor of general intelligence (g).

Measurement models using the illness variables. Exploratory factor analyses were conducted using the 15 concatenated disease categories discussed above. The attempt to find underlying factors

among the sets of medical categories was not successful. Therefore, separate structural models were tested using the various illness variables as predictors of cross-occasion intellectual performance. Based on a median split, the sample was divided into a younger and an older half, and the medical models were tested separately in the two age groups. The autoregressive model for the PMA was used on the y-side of the structural equations. A diagram of the initial model tested for the various medical independent variables is shown in Figure 1.

Of the eight categories tested, three yielded at least one significant relationship between a medical independent variable and g: circulatory disorders, neoplasms, and musculoskeletal disorders.

Significant concurrent relationships were found for all three of these variables at Time 2. That is, neoplasms, circulatory disorders, and musculoskeletal diseases collapsed over Time Frame 2 (1957-1963) were all significant predictors of g at Time 2. In addition, a time-lagged relationship between neoplasms and g at Time 3 was significant, and a nonrecursive relationship between circulatory disorders and third occasion g was found.

The actual R^2 values for the three concurrent structural relations involving medical predictors were rather low, ranging from 1.6 to 4 percent of the variance in Time 2 g. However, these seemingly low R^2's must be evaluated in light of the fact that much of the variance in intellectual performance at Time 2 is already accounted for by initial level intelligence. The variance due to the illness variables represents change over time (from 1963 to 1970) in individuals' relative ranking about the mean of g.

We can only speculate concerning the precise causal mechanism whereby musculoskeletal disorders or neoplasms might affect intellectual performance. Chronic rheumatoid problems could limit intellectual performance peripherally, interms of psychmotor speed. Contrary to prediction, the relationship between neoplasms and g was positive. This has led us to speculate that persons who survive the ordeal of cancer treatment and remain active in such activities as participating in a psychological study represent a highly select subsample. Of course, because of the novelty of this approach to studying the relationship between pathology and mental tests performance , such conclusions must be viewed cautiously until replications are carried out.

REFERENCE NOTES

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