Cognition and Intelligence

Identifying the Mechanisms of the Mind

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From Description to Explanation in Cognitive Aging

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Two different approaches have been employed in contemporary research to investigate the effects of aging on cognitive and intellectual abilities. One approach can be termed *process analysis* because it relies on task analyses or formal models to attempt to identify the specific processes in a cognitive task that are responsible for the observed age differences. This approach has used a variety of analytical methods such as subtraction, additive factors, and process dissociation to attempt to partition the variance in the target variable into theoretically distinct processes. Because the primary interest is in decomposing the variance in a single variable into different processes, process analysis research has typically involved comparing performance in one or more conditions in a single task, frequently in relatively small samples of young and old adults.

The second approach taken to investigate aging and cognition can be termed *covariance analysis* because it attempts to specify which combinations of variables covary together with respect to their age-related influences. A primary goal of this type of research is to partition the variance in the target variable into a portion not related to age, a portion related to age and shared with other variables, and a portion uniquely related to age. Covariance analysis research necessarily requires data from multiple variables and tasks and usually involves moderately large samples of adults across a wide age range.

One way to conceptualize the difference between the two perspectives is portrayed in Figure 1. The left side represents the process analysis perspective in which the variance in the target variable is partitioned into hypothesized processes, and the right side represents the covariance perspective in which the variance in the variable is partitioned according to age-related individual differences. Because the two approaches focus on different ways of partitioning variance in the same variable, they can be considered to address different questions, and thus it is not surprising that

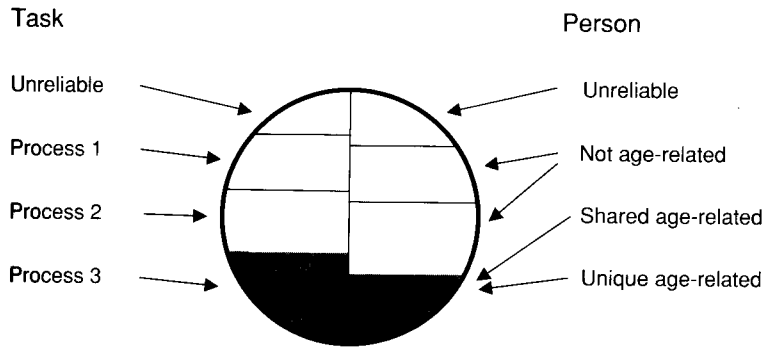

FIGURE 1. Schematic illustration of different approaches to partitioning the variance in the variable of interest.

there is often little communication between researchers working within each perspective.

Much of my research has been motivated by the belief that when trying to understand age-related individual differences in cognitive functioning, it is important to combine the two approaches and to interpret age-related effects on variables representing processes hypothesized to be responsible for performance in a particular task in the context of age-related effects occurring on other variables. Another major assumption guiding my research is that before attempting to explain the phenomenon of cognitive aging, it is essential to have an accurate description of the phenomenon in terms of the number and nature of statistically distinct age-related influences that are operating because that will determine the scope of the explanations that will eventually be needed.

The point that an accurate description has implications for the explanation can be illustrated with examples from the sensory domain. Assume that age-related effects were found on measures of visual acuity (i.e., the smallest visual angle that can be resolved), visual accommodation (the closest distance at which one can see with clear focus), and color discrimination (distinguishing between colors such as blue and purple). The age-related effects on these variables could all be independent of one another in the sense that they are caused by separate and distinct mechanisms. However, these particular variables were selected because they could each be manifestations of the accumulation of dead cells in the lens of the eye, which leads to blurred vision (affecting visual acuity), reduced flexibility in altering the shape of the lens (affecting accommodation), and yellowing that selectively absorbs short wavelength light in the blue region of the spectrum (affecting color discrimination). In this example, therefore, a single age-related change in the structure of the lens may be able to account for age-related effects on what might, at least initially, appear to be quite different variables.

Of course, independent age-related influences could also occur. Consider the phenomena of presbyopia – the reduced accommodation ability of the lens that leads to a decreased ability to focus on near objects – and presbycusis – the reduced sensitivity to, and discrimination among, high-frequency tones. These are both age-related problems (as indicated by the common root presby, which refers to elders), but it is likely that they originate from different, and potentially independent, causes. That is, as just noted, presbyopia is largely a consequence of the accumulation of dead cells in the lens, whereas presbycusis is a disorder associated with degeneration of the bones of the middle ear and/or death of hair cells on the basilar membrane. The probability or rate that an individual develops presbyopia as he or she ages may therefore be unrelated to the probability or rate that he or she will develop presbycusis. If this is the case, such that knowledge of the effects of aging on one of these conditions is not informative about the effects of aging on the other condition, then they can be inferred to be independent with respect to their age-related influences. Because information of this type is critical for determining exactly what needs to be explained, and for specifying the most meaningful level of analysis in characterizing the phenomenon of cognitive aging, a major focus of my research has been to investigate the extent to which the age-related influences on different cognitive variables are independent of one another.

DESCRIPTION OF COGNITIVE AGING

Alternative analytical models that can be used to investigate age-related influences in cognitive functioning are schematically illustrated in Figure 2. The model in panel A represents the simple univariate approach because the focus is on a single variable. The remaining models in Figure 2 are multivariate in that age-related effects on the target variable are examined in the context of effects on other variables. However, it is important to note that process analyses of the target variable can still be conducted within the multivariate perspective, but because multiple variables are examined it is also possible to partition the variance in the target variable into portions shared with other variables and portions uniquely related to age. In each case, direct age–variable relations (represented in the figure by dotted lines) can be evaluated in the context of relations of age to other variables or constructs. Furthermore, only if the direct age relation is equal in magnitude to the observed (or total) age relation could one infer that age-related influences on the target variable are independent of age-related influences on other variables or constructs.

The model in panel B represents the hypothesis that the age-related effects on variable *x* are at least partially mediated through age-related effects on variable *a*. A relatively large number of studies have examined

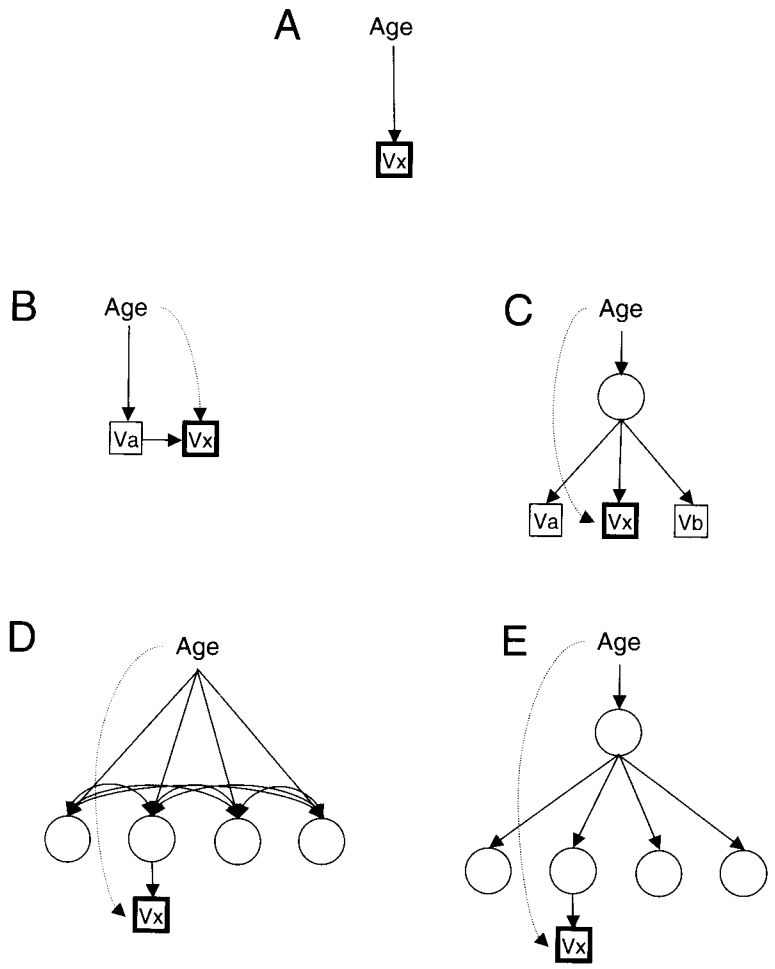

FIGURE 2. Alternative analytical models portraying relationships between age and cognitive variables. See text for details.

variants of this type of mediation model with measures of processing speed or working memory as the hypothesized mediator of age-related effects on other cognitive variables. In nearly every study the age-related variance in the target variable was considerably reduced when the variance in the hypothesized mediator variables was statistically controlled (e.g., Lindenberger, Mayr, & Kliegl, 1993; Park et al., 1996; Salthouse, 1996b, c, 1998; Salthouse et al., 1996b; Verhaeghen & Salthouse, 1997). Although this pattern of results has been interpreted as supporting the hypothesis that the controlled variable partially mediates the age-related effects on other cognitive variables, it is important to recognize that this is not the only

possible interpretation. That is, causal direction is ambiguous in single-occasion correlational studies, and the effects on both variables could be attributable to an unknown third variable. However, the important point in the current context is that because results from mediational models indicate that the age-related influences on different cognitive variables vary according to the other variables included in the analysis, we can conclude that the age-related influences are not completely independent of one another.

The model portrayed in panel C in Figure 2 represents a shared influence model that differs from the mediational model in that it does not assume that any single variable or construct has special status, or causal priority, in terms of the age-related influences on different types of cognitive variables. Shared influence models are therefore agnostic about the existence or identity of mediators; instead they simply examine age-related effects on individual variables after controlling the age-related effects on an estimate of what is common to all variables. These types of models are sometimes referred to as common cause models because many variables are assumed to be influenced by a common cause, even though the specific nature of that common cause is unknown.

Several different types of analytical procedures have been used within shared influence models to partition the age-related effects on a target variable into a portion shared with other variables, and a portion unique to that variable. For example, the first principal component in a principal components analysis can be used to estimate the variance common to all variables, and then that estimate can be controlled in a hierarchical multiple regression analysis before examining age-related effects on the target variable. Alternatively, a structural equation model can be specified in which all variables are assumed to be influenced by a common factor, which in turn is influenced by age (e.g., Kliegl & Mayr, 1992; McArdle & Prescott, 1992; Salthouse, 1994). A variety of methods can also be used within shared influence methods to examine unique age-related influences. For example, the predicted age–variable correlation based on a single shared influence can be compared to the observed age correlation, with the difference between the two correlations inferred to be attributable to unique age-related influences. Alternatively, the path coefficient for a direct relation from age to the variable can be examined when age-related influences also operate through the shared or common factor.

Many different combinations of variables have been examined with shared influence analyses in different data sets from my laboratory (e.g., Salthouse, 1994, 1996a, b, 2001b; Salthouse & Czaja, 2000; Salthouse, Hambrick, & McGuthry, 1998; Salthouse et al., 1996b; Salthouse, McGuthry & Hambrick, 1999; Salthouse et al., 1997; Verhaeghen & Salthouse, 1997) and in data sets from other laboratories (e.g., Anstey & Smith, 1999; Christensen et al., 2001; Hultsch et al., 1998; Lindenberger & Baltes, 1994; Lindenberger et al., 1993; McArdle & Prescott, 1992; Park et al., 2002). A consistent finding in each of these studies has been that the unique age-related influences on cognitive variables are few in number and small in magnitude. Quantitative estimates of the magnitude of shared and unique age-related influences on a given variable have varied according to the method of estimating the common and unique variances and according to the particular combination of variables included in the analysis. However, it has almost always been the case that a relatively small proportion of the total age-related variance was unique to the target variable and independent of the age-related effects on other variables.

An interesting implication of shared influence models is that a systematic relation should exist between the degree to which a variable shares variance with other variables and the magnitude of the correlation of age with the variable. That is, if a large proportion of the age-related effects on a set of variables operates through the common factor that represents variance shared among the variables, then one would expect a strong relationship between the variable's relation to the common factor and the magnitude of the variable's relation to age. These predicted functions can be termed AR functions because they link the age (A) effects on the variables with the relatedness (R) of the variables to each other.

Examination of the correspondence between two sets of relations has been used in several areas of psychology as a means of investigating the pattern by which different variables are related to one another. For example, Hart and Spearman (1914) compared normal individuals and mental patients on a number of different variables and reported that an index of the "intellectual saturation" of a variable correlated .47 with the amount of impairment in the variable exhibited in the sample of mental patients. In the area of attention and performance, Duncan et al. (1992) examined a set of variables representing different aspects of driving performance and found a correlation of .67 between the variable's correlation with the score on an intelligence test and the degree to which performance on the variable was impaired when the research participant was simultaneously performing another task. In the field of behavioral genetics, Plomin et al. (1994) reported a correlation of .77 between loadings of a set of cognitive variables on the first (unrotated) principal component and estimates of the heritability of the variables. The most extensive use of this technique has been by Jensen (e.g., 1998), who termed it the *method of correlated vectors*. He has primarily used it as a method of determining the degree to which a factor affects intellectual *g* rather than specific abilities, and in different data sets he has reported positive correlations between a variable's *g* loadings and its correlation with factors such as head size, heritability, and reaction time.

The degree to which a variable is related to other variables can be assessed with a variety of indices, such as the median correlation of the

variable with all other variables, the multiple correlation between that variable and other variables, the loading of the variable on the first principal factor in a factor analysis, or the loading of the variable on the first principal component in a principal components analysis. However, the estimates from each of these procedures tend to be highly correlated with one another (see Salthouse, 2001a, b), and hence only results with the method of assessing relatedness based on the first principal component loading will be described here. Positive AR functions have been reported in a number of different studies (e.g., Salthouse, 2001a, b), including a recent analysis of data from over 5,000 adults under the age of fifty (Schroeder & Salthouse, 2004). Re-analyses of thirty different data sets from my laboratory revealed that the phenomenon of positive AR functions appears to be quite robust because across these data sets the median rank-order correlation between the relation of a variable to age and the variable's relation to other variables was .80 (Salthouse, 2001b). Moreover, most of the exceptions with low correlations were interpretable in terms of restriction of range in either the relatedness dimension or the age correlation dimension. Additional analyses revealed that similar values of relatedness were obtained when estimates of the reliability of the variables, and of the relations of age to the variables, were statistically controlled before determining the degree to which the variables were related to one another (Salthouse, 2001a).

Figure 3 contains a summary AR function created by plotting the means (and standard errors) of the AR coordinates for variables that were assessed

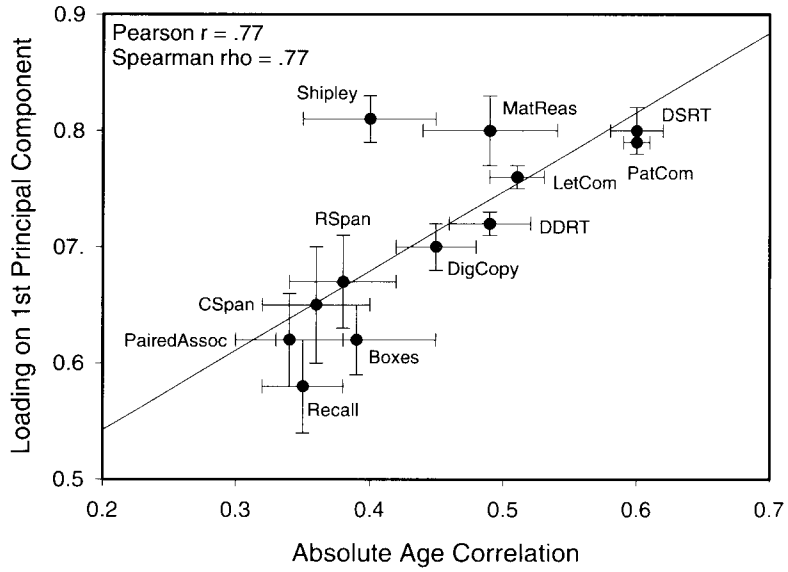

FIGURE 3. Means and standard errors of age correlations and loadings on the first principal component of variables included in at least five different studies.

in at least five studies from my laboratory. Notice that a positive AR function is evident even with these meta-analytic results. Although not portrayed here, a similar pattern was evident when the variables were represented in terms of percentiles of the A and R values from each data set (i.e., rho = .63) instead of absolute values of the loadings and correlations. The systematic relations apparent in AR analyses are intriguing because they raise the possibility that the ordering of variables along the AR function might be informative about the dimension underlying shared age-related influences. Although convincing interpretations of how variables at various positions along the AR function differ from one another are not yet available (but see Salthouse, 1994, 2001a, b, for speculations), the outcomes of shared influence analyses are clear in suggesting that, as was the case with mediational models, large proportions of the age-related influences on different cognitive variables are not independent of one another.

The model portrayed in panel D in Figure 2 is a correlated factors model in which the observed variables are structured into first-order latent factors, and then those factors are allowed to correlate with one another. This type of model does not allow a direct investigation of age-related influences that are shared across factors because the covariation among factors is represented by correlations instead of by a higher-order factor that could be examined with respect to its age relations. Nevertheless, the correlations among the factors are informative about the potential independence of age-related influences on the factors (and on the constituent variables) because the larger the correlations among the factors, the less likely the age-related influences are independent. That is, age-related effects can operate directly on the factors, or they can be indirect and operate through other factors with which the target factor is correlated. To the extent that the cognitive factors are moderately correlated with one another, therefore, it is unlikely that age-related effects upon them would be completely independent.

Evidence relevant to the interrelations of cognitive factors is contained in Table 1, which summarizes results from ten recent studies in my laboratory in which estimates of relations among age and cognitive factors could be obtained. Participants in each of the studies ranged from about 18 to 80 years of age, and most had completed at least some college education and reported themselves to be in good to excellent health. In each case, we analyzed the data with a model similar to that portrayed in panel D of Figure 2, except that direct relations of age to individual variables were ignored. All of the analyses provided reasonably good fits to the data (i.e., the median CFI was .99, and the median RMSEA was .07). Despite different samples of participants and combinations of variables, the results in Table 1 are quite consistent in indicating moderately strong correlations among the factors.

A crude indication of the extent of dependence among the age-related influences on the factors can be obtained by comparing the simple age

TABLE 1. *Correlations among Factors and Age in 10 Recent Studies*

Study	N	Age-g_f	Age-Mem	Age-Spd	Age-Voc	g_f-Mem	g_f-Spd	g_f-Voc	Mem-Spd	Mem-Voc	Spd-Voc
A	261	−.46	−.47	−.58	.36	.61	.56	.66	.51	.65	.50
B	204	−.50	−.61	−.79	.17	.61	.48	.71	.54	.66	.50
C	206	−.71	−.60	−.64	.00	.41	.47	.56	.25	.42	.36
D	220	−.76		−.66	.02		.66	.70			.52
E	229	−.78		−.67	.14		.61	.45			.56
F	207	−.46		−.45	.41		.42	.54			.38
G	380	−.46	−.40	−.64	.35	.53	.66	.54	.59	.51	.44
H	124		−.41	−.73	.40				.34	.33	.29
I	259	−.57	−.62	−.72	.22	.47	.64	.67	.40	.42	.46
J	178		−.64	−.77	.22				.75	.51	.46
Wt. Avg.		−.58	−.53	−.66	.26	.53	.57	.60	.50	.51	.46

A – Salthouse, Atkinson, & Berish (2002), g_f = Ravens, Letter Sets, Spatial Relations, Paper Folding, Form Boards; Mem = Recall across 4 trials, Logical Memory, Paired Associates; Speed = Digit Symbol, Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary, WAIS III Vocabulary, Woodcock–Johnson Picture Vocabulary

B – Salthouse & Ferrer-Caja (2003), g_f = Analysis-Synthesis, Spatial Relations, Ravens, Paper Folding, Block Design; Mem = Recall across 4 trials, Recall on new list, Logical Memory, Paired Associates; Speed = Digit Symbol, Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary, WAIS III Vocabulary, Woodcock–Johnson Picture Vocabulary

C – Salthouse (2001b), g_f = Ravens, Analysis-Synthesis, Paper Folding, Letter Sets; Mem = Recall on trials 1, 2, and 3, Paired Associates; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary, Knowledge 1, Knowledge 2

D – Salthouse (2001a, Study 1), g_f = Ravens, Figure Classification, Locations; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

E – Salthouse (2001a, Study 2), g_f = Analytical Reasoning, Figure Classification, Locations; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

F – Salthouse, Toth, et al. (2000), g_f = Ravens, Spatial Relations; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

G – Salthouse et al. (1998), g_f = Ravens, Cube Assembly; Mem = Recall on trials 1, 2, 3, 4, and 5; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

H – Salthouse, Toth, et al. (1997), Mem = CVLT Recall Trials 1 through 3, CVLT Recall on New List; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

I – Salthouse et al. (1996a), g_f = Shipley Abstraction, Block Design, Object Assembly; Mem = Recall across 5 trials, Recall on new list, Paired Associates; Speed = Digit Symbol, Letter Comparison, Pattern Comparison; Voc = Shipley Vocabulary

J – Salthouse (1996b), Mem = Recall at 0.5, 1.0, and 2.0 sec rates; Speed = Letter Comparison, Pattern Comparison; Voc = Synonym Vocabulary, Antonym Vocabulary

correlations with the partial age correlations in which the variance in one of the other factors is statistically controlled before examining the relation of age to the other factor. To illustrate, the weighted average correlation between age and the fluid intelligence (g_f) factor was −.58, but this was reduced to −.42 after partialling the variance in the episodic memory factor, and it was reduced to −.33 after partialling the variance in the perceptual speed factor. Because the partial correlations are considerably smaller than the simple correlations, and the corresponding proportions of variance even smaller, it can be inferred that the age-related influences on the factors are not independent of one another.

The model in panel E represents a hierarchical structure in which correlations among the first-order factors are interpreted in terms of one or more higher-order factors. That is, rather than representing the covariation among factors as correlations, which leave the relations unexplained in the context of the model, a hierarchical model attributes the covariation to the operation of higher-order factors. When variables with both positive (e.g., vocabulary and knowledge) and negative (e.g., reasoning, spatial visualization, episodic memory, and speed) age relations are included in the analysis, at least two distinct age-related influences are likely to be required. However, it is not yet clear whether it is more meaningful to think of the two influences as operating in opposite directions on factors at the same level in the hierarchy (as in models based on the distinction between fluid and crystallized abilities), or as a negative age-related influence operating at the highest level and a positive age-related influence, perhaps representing the benefits of experience, operating at an intermediate level in the hierarchy (as in models postulating a unitary g factor). Hierarchical models have also been applied in analyses restricted to variables with negative age-related influences to determine the number of independent age-related effects that may be operating on the factors and variables. Results from several data sets suggest that there are at least three statistically distinct age-related influences, one affecting the highest level of the hierarchy that represents what all factors and variables have in common, one affecting an episodic memory factor, and one affecting a perceptual speed factor (e.g., Salthouse, 2001b; Salthouse & Czaja, 2000; Salthouse & Ferrer-Caja, 2003). More research of this type with a broader range of variables is needed before definitive conclusions can be reached about the number and nature of distinct age-related influences. However, the important point in the current context is that because age-related effects have been found to operate at the highest level in the hierarchical structure, age-related influences on variables at lower levels can be inferred to be at least partially shared with one another.

To summarize, results from several different types of multivariate analyses conducted on data from my laboratory and from other laboratories have been consistent in suggesting that the age-related influences on a wide

range of cognitive variables are not independent of the age-related influences on other variables. To the extent that this is an accurate description of the phenomenon of cognitive aging, a primary theoretical challenge is to explain age-related influences that are shared across different types of variables. It should be emphasized that acceptance of this goal does not deny the existence, or the importance, of unique age-related effects on individual variables or constructs. Rather, the point is that understanding shared age-related effects should be considered a high priority because such effects have a broader impact, and are frequently larger in magnitude, than age-related effects that are specific to particular variables or factors.

EXPLANATION OF COGNITIVE AGING

What types of explanations might be plausible to account for shared age-related influences on different types of cognitive variables? By definition, the mechanisms are unlikely to involve processes or strategies that are restricted to a small set of cognitive tasks. Instead, the relevant mechanisms must be broad enough to affect a wide variety of cognitive variables, ranging from those assessing perceptual speed to those assessing episodic memory and inductive reasoning.

Although there is little consensus at the current time with respect to how shared age-related effects are to be explained, a number of researchers have attempted to interpret individual differences on a variety of different cognitive variables in terms of constructs at the same level of analysis as the to-be-explained phenomena. For example, among the constructs that have been proposed to be critical with respect to individual differences in a variety of other cognitive variables are goal neglect (e.g., Duncan et al., 1996), context maintenance (e.g., Braver et al., 2001), controlled attention (e.g., Engle, Kane, & Tuholski, 1999), working memory (e.g., Kyllonen & Christal, 1990), processing speed (e.g., Salthouse, 1996c), and various types of inhibition (e.g., Hasher, Zacks, & May, 1999). Hypotheses of this type have often been accompanied by plausible theoretical arguments and intriguing experimental findings, but most share two important weaknesses.

First, the relevant construct has often been assessed with a single variable, with little or no evidence provided to indicate that the variable exclusively, and exhaustively, reflects that construct.[1] The correspondence between the variable and the theoretical construct has typically been justified by subjective judgments of face validity, and by arguments based on plausibility. However, in the absence of relevant empirical evidence,

[1] Some researchers have attempted to assess the same construct with different variables, but because the variables were usually examined in separate studies the relations among the variables could not be examined to determine whether they represented the same dimension of individual differences.

questions can be raised with respect to whether the variables actually represent the intended construct, and whether that construct is distinct from other constructs.

A second weakness of most of the existing theoretical explanations is that the empirical data offered in support of the hypothesis are frequently in the form of correlations between measures of the presumed critical construct and other cognitive variables. However, because one of the best-established results in all of psychology is that most cognitive variables are positively correlated with one another, a discovery of significant correlations involving the relevant variables is merely necessary, and is not sufficient, to establish that the hypothesized construct is responsible for effects on other variables. What is also needed is evidence that the critical construct is the primary cause of the individual differences in other variables and constructs. Unfortunately, it is difficult to determine causal priority when all of the data are of the same type – namely, observations of overt behavior at a single point in time.

The first weakness can be addressed with more sophisticated research designs that allow investigations of convergent and discriminant aspects of construct validity. That is, one way to investigate the validity of a theoretical construct is to determine whether the variables hypothesized to assess that construct have moderate to strong correlations with one another (i.e., exhibit convergent validity), but have weak to nonexistent correlations with variables representing other constructs (i.e., exhibit discriminant validity). Research of this type can be time-consuming and expensive because moderately large samples of participants with multiple measures of several constructs are required to allow patterns of correlations to be examined. Nevertheless, it is one of the few methods currently available for evaluating what variables actually represent and for determining the extent to which they assess something different from what is assessed by established constructs. An example of this type of research is a recent study (Salthouse et al., 2003) that we designed to investigate the construct validity of the neuropsychological concept of executive functioning, and of aspects of executive control corresponding to inhibition, updating, and time sharing. The major finding in the study was that nearly all of the individual-difference variance common to measures of the neuropsychological construct of executive functioning, and to measures of updating and time-sharing aspects of executive control, overlapped with the individual-difference variance in a fluid intelligence (g_f) construct. Such results are potentially important because they suggest that researchers who assume that they are investigating one theoretical construct may also, or instead, be investigating manifestations of another construct.

Determination of causal priority will likely require several different types of research. Two approaches that may prove informative involve the investigation of plausible neurobiological substrates and the investigation of lead–lag relationships with longitudinal comparisons.

Although brain–behavior relations have been the focus of considerable research, a fundamental assumption of the perspective outlined here is that it is important that the neurobiological substrate be at a level of analysis that is appropriate to account for broad age-related effects on cognitive performance. One possible candidate for the neural substrate of shared age-related influences is impairment in the effectiveness of a single neural structure responsible for coordinating or controlling multiple cognitive operations. The dorsal lateral prefrontal cortex has been mentioned as a possible site of the "CEO" of the cognitive system, but this or any other speculation must be accompanied by evidence that the structure is actually involved in many different cognitive tasks and that its efficiency or effectiveness is impaired with increased age. A second possible candidate to account for shared age-related effects is an alteration in the effectiveness of communication among different neural regions. That is, the critical age-related influences may not be on a discrete structure, but rather on the efficiency or effectiveness in communicating among different regions.

Both the critical structure and the communication deficiency hypotheses would benefit from somewhat different approaches to functional neuroimaging than the currently dominant discrete localization approach. For example, examination of patterns of activation that are common to several different cognitive tasks, which Price and Friston (1997) termed *conjunction analysis*, would likely be informative about which neuroanatomical structures are involved in multiple cognitive tasks. Furthermore, examination of co-activation patterns across different regions within a single cognitive task, in what has been termed *functional connectivity analysis* (e.g., Grady et al., 2002; Esposito et al., 1999; Nyberg & McIntosh, 2001), would likely be informative about the efficiency of cortical communication.

Ultimately, of course, research will have to determine why particular structures or circuits are affected by increased age. Among the possible candidates at a somewhat lower level of analysis are age-related differences in the density of receptors for the dopamine neurotransmitter (e.g., Backman et al., 2000; Kaasinen et al., 2000; Rinne et al., 1993; Volkow et al., 1998), and in the integrity of axonal myelination (e.g., Abe et al., 2002; Nusbaum et al., 2001; O'Sullivan et al., 2001). The particular level of reductionism that will eventually be most meaningful in interpreting age-related differences in cognitive functioning is not yet obvious. However, a key assumption motivating the search for neurobiological substrates is that mechanisms responsible for age-related changes in neural substrates are likely to be more primitive or fundamental than those responsible for changes at higher conceptual levels, such that investigation of neural substrates may help identify the sequence in which changes in relevant constructs occur.

A second type of research that might help establish causal priority among constructs is evidence from longitudinal comparisons that changes in different measures of the critical construct (including neurobiological measures if they are available) occur together and precede changes in the cognitive variables they are hypothesized to affect. Evidence of this type will probably be difficult to obtain because a broad variety of variables representing the critical construct and other cognitive constructs is needed, and the retest interval between successive measurements must be long enough to capture the phenomenon of interest, namely age-related changes occurring over a span of decades. Furthermore, the analytical methods should allow evaluation of whether age-related changes in the measures of the critical construct occur together and before changes in the cognitive variables they are presumed to mediate. Even with the appropriate data and suitable analytical methods, however, determination of the sequential order among relevant constructs will be challenging because little is currently known about the timing of the changes (e.g., do they begin at age 70, at age 20, or somewhere in between?), about the interval between changes in the critical construct and changes in the cognitive variables affected by that construct (e.g., is the lag on the order of days, years, or decades?), or about individual differences in these parameters (e.g., how much do people vary in the age at which the first changes occur, and in the interval between the initial and subsequent changes?).

STRENGTHS AND WEAKNESSES

The primary strength of the description-to-explanation approach that I have been pursuing is that the multivariate perspective provides a broader and more comprehensive assessment of the phenomenon of cognitive aging than that based on univariate research. Moreover, because of the greater amount of information about exactly what needs to be explained, explanations based on that data are more likely to be at the most meaningful level of analysis. However, a nontrivial weakness is that multivariate research is more difficult and expensive to conduct than the more typical type of research focusing on a single variable in relatively small samples of young and old adults. Not only does multivariate research require large samples with a continuous range of ages, but also more time is needed from each participant to allow the assessment of multiple variables, and the analytical procedures are frequently more complex. Partly because of this difficulty, the most progress made thus far has been in the description of the cognitive aging phenomenon rather than in its explanation.

FUTURE DIRECTIONS

One direction for future research is to revise and extend the methods of characterizing the nature, and estimating the relative magnitude, of shared and unique age-related influences on cognitive variables. A primary goal of these efforts should be a more refined assessment and characterization

of each type of influence. Efforts in this direction are also valuable because all analytical methods require assumptions that may not be valid, and thus it is always desirable to converge on conclusions with analyses based on different combinations of assumptions and procedures.

A second direction for future research is to investigate relations between age-related influences identified from behavioral studies and possible neural substrates of those influences. As noted above, a key assumption of the current perspective is that progress in understanding brain–behavior relations involving aging is likely to be faster when the correspondence is examined at the appropriate level of analysis. In the cognitive domain this will not only be single variables, but also combinations of variables that share age-related variance, and in the neurobiological domain the neural substrates should be able to account for age-related effects that are shared across different types of cognitive variables.

A third direction for future research is to examine candidates for causal influences by investigating potential moderators of the relations between age and measures of cognitive functioning. Many factors have been found to be related to the level of cognitive performance; but to be plausible as a determinant of the age differences in cognition the factor should significantly interact with age, such that the relations between age and measures of cognitive functioning are moderated by the level of that factor.

Because the strongest evidence for causality is based on experimental manipulation, a final goal for future research is to examine interventions that might alter the course of age-related change in both cognitive constructs and their neurobiological substrates. The specific nature of the interventions are not yet obvious, but definitive conclusions about the causes of age-related differences in cognitive functioning will probably not be possible until interventions are available to eliminate those differences.

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