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The Logical and Psychometric Prerequisites for Cognitive Therapy of Schizophrenia

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This discussion follows from a key premise that there is potential clinical value in identifying and directly treating the cognitive abnormalities which for over a century have been seen as the hallmarks of schizophrenia. This is both an old and a relatively new idea. It is old, in that the psychodynamic approaches to psychotherapy, especially ego psychology, have long sought to modify cognition in schizophrenia, if unsystematically and indirectly. It is new, in that only in the last decade have both the contemporary technologies of the information processing laboratory and of cognitive-behaviour modification been applied to the problem of treatment. The failures of psychodynamic treatment approaches with schizophrenic patients have perhaps discouraged widespread experimentation with cognitive models in clinical assessment and treatment. This is unfortunate, as contemporary clinical procedures and laboratory technologies are hardly comparable to psychodynamic methods. In the past decade findings have accumulated, in the form of case reports, single-subject experiments and treatment trials, which support the potential usefulness of assessing and directly treating schizophrenic patients' cognitive abnormalities (reviewed by Spaulding *et al*, 1986).

If we are to see cognitive abnormalities as deficits to be identified and treated we must know more about them. We must determine their role in the developmental aetiology and the phenomenology of schizophrenia. We must learn more about the causal relationships between cognitive deficits, neurophysiological abnormalities and social behaviour, and we must understand how and why the abnormalities appear and disappear over the longitudinal course of the schizophrenic disorder. These are the logical prerequisites for a complete model of cognitive therapy.

Only after the logical prerequisites are satisfied can we fully address questions of clinical assessment and diagnosis. We must determine the number and nature of cognitive measures which are necessary to differentiate between vulnerabilities and impairments in the premorbid, prodromal, acute and residual phases. We must be able to identify and quantify deficits which are amenable to treatment and monitor them longitudinally as treatment proceeds. These are the psychometric prerequisites of a cognitive therapy model.

Perhaps the most basic research question to be posed pursuant to logical and psychometric prerequisites concerns the functional autonomy of cognitive abnormalities. Can we identify deficits which are relatively independent of other problems in the patients' biopsychosocial organismic system, or are all cognitive abnormalities continuously mediated by a more primary dysfunction?

Older cognitive models hypothesised that all or most deficits and clinical symptoms are secondary to a single functionally autonomous key deficit within the cognitive domain, such as Shakow's (1963) 'segmental set' or Arieti's (1955) 'paleo-logic'. This has not remained a sound hypothesis, for several reasons. One reason is that no single cognitive deficit has been found which is exclusively associated with the symptoms of schizophrenia. Another reason is that experimental studies of schizophrenia suggest that at least some cognitive deficits are the result of neurophysiological dysfunction. A third reason is that the older theories make unwarranted assumptions about diagnostic validity and the aetiological homogeneity of schizophrenia. Failure to appreciate these realities has lead to what Cromwell (1984) has called the 'Holy Grail Error' of schizophrenia research – a fruitless search for the one true deficit.

Researchers have also committed the Holy Grail Error in the biological and social domains. A partial antidote for this is our contemporary mainstream consensus about diathesis-stress models of aetiology. We now see schizophrenia as a final common pathway whose tributaries come from the biological, psychological and socio-environmental realms. Despite this more sophisticated view, however, the question of functional autonomy remains. We do not know whether particular cognitive deficits play an independent role in the expression of schizophrenia, or whether they are simply secondary to pervasive disruptions across many levels of organismic functioning.

If a cognitive abnormality is functionally autonomous and contributory to deficits in social functioning, then remediation of that abnormality

may be expected to produce improvements in social functioning. If it is only a marker of something else, remediation is irrelevant.

We are thus confronted at the beginning with two alternative presumptions for a cognitive model. The first is that all the cognitive deficits associated with schizophrenia stem from a single causal process, probably external to the cognitive realm. If some of these deficits appear more severe than others, it is because some are less affected by the external factor than others, or because some of our measurement techniques are more sensitive than others. Recently, the cognitive construct of 'processing capacity' has been proposed as a mechanism by which this effect could occur. The external factor, originating as dysregulation of CNS activation levels, produces a generalised reduction in processing capacity, and some aspects of cognitive performance suffer more from this reduced capacity than others. Nuechterlein & Dawson (1984) have shown that a capacity model fits nicely in retrospective interpretation of experimental findings.

The second alternative is that there are many independent cognitive deficits, associated with many aetiological pathways whose sources are either within or external to the cognitive realm. The cognitive and behavioural heterogeneity of schizophrenia seems to be the best support for this view. No cognitive deficit is unique to schizophrenia, and no deficit is invariably found in all schizophrenic subjects. Most of the research which has expressly examined relationships between deficits in schizophrenia has found low intercorrelations. The concept of compensatory mechanisms within the cognitive realm, which has been suggested by numerous theorists over the years, is yet another reason to believe in functional autonomy.

One reason that cognitive research has not resolved this dilemma is that with few exceptions only one or two cognitive measurements are made per study, and these measurements are then interpreted in terms of differences in average levels of performance between groups (schizophrenic *v.* non-schizophrenic). This reveals little about relationships between different cognitive measurements or about relationships between the processes which the measurements supposedly represent. After all, the issue of functional autonomy is essentially an issue of the interrelationships between cognitive processes, both within and between diagnostic groups. We may even expect that in addition to differences between performance levels there are differences between the patterns of interrelationships, and therefore, between the structure of cognition, across groups.

For several years now our research group has been developing a collection of measures which were selected for the purpose of studying their applicability to clinical assessment and treatment. We have integrated the collection into a test battery we call COGLAB, for 'cognitive laboratory'. Accumulation of data on this battery has allowed us to begin looking at the measures' interrelationships.

The battery is administered by a microcomputer. It takes about 40 minutes for a normal college student to finish, and 40–60 minutes for a chronic schizophrenic patient. All the individual tasks have an honourable history in the experimental psychopathology of schizophrenia (see Cromwell & Spaulding, 1978), and so we know that they are all associated with abilities which are impaired by that disorder.

The battery generates ten measures from six different tasks. Apprehension is measured by accuracy of report of briefly-presented digit pairs. The target stimuli are followed by no mask or a backward mask with stimulus onset asynchrony of 33 ms or 66 ms. Performance on this task is thought to represent efficiency of pre-attentional visual feature processing. Reaction time is measured with a traditional 80-trial protocol, with preparatory intervals (the time between a preparatory and a 'go' signal) varying from 1 to 8 s. The response timed is lifting the index finger from a depressed button. Conceptual processing is the total error score on a version of the Wisconsin Card Sorting Test, which tests concept formation, trial-and-error learning and conceptual flexibility. Vigilance is measured by a version of the well-known Continuous Performance Task. The subject watches digits presented briefly, one per second, and presses a button when a specified target appears. In the COGLAB version, which was originally constructed by Robert Asarnow, capacity demand is increased first by adding eight distraction digits to the stimuli, and then by changing the specified target. Distraction effects are measured in the reaction time task as the increase in response latency produced by a surprise stimulus (a border of red Xs around the preparatory signal) presented at the beginning of the preparatory interval. A redundancy effect is also measured in the reaction time protocol, as the effect of series of four 7-s preparatory intervals embedded in the irregular series. Anticipatory errors are premature finger-lifts in the reaction time task. Vigilance false alarms are errors of commission (button presses when the target is not present) in the continuous performance task. Field articulation is measured in COGLAB with the well-known Müller-Lyer illusion, as units of error in the direction of the illusion. Size estimation is measured in a task designed specifically for COGLAB. The subject views geometric figures, and after

a 5-s latency judges two different-sized figures for their size proximity to the standard stimulus.

A standardisation group of 140 college students and a group of 125 chronic schizophrenic patients were tested with COGLAB. The test scores were submitted to a series of factor analyses. The purpose of the analyses was to identify patterns of interrelationships among the ten measured cognitive tasks. Such patterns hypothetically represent interrelations between actual cognitive processes or operating characteristics. Thus by examining the functional measures' statistical interrelationships, we may draw some inferences about relationships between the actual cognitive processes and operating characteristics which underlie those functions. Also, we can determine whether the patterns of relationships are the same or different across groups.

The two key aspects of factor analysis solutions are the number and membership of the factors. By membership, we mean which measures come together to make up the factor. We employed a combination of exploratory and confirmatory analytic procedures to address each of these aspects.* Confirmatory analyses have a number of advantages over exploratory procedures. Most importantly, one may directly test specific hypotheses about which variables combine to form which factors, rather than hoping that the factors will 'appear' during exploratory analyses and rotations (for reviews and discussions of confirmatory methods see Gorsuch, 1983; Bernstein & Garbin, 1985).

Figure 1 shows a comparison of the factor structures of the two groups as derived by the confirmatory procedures. The five normal factors can be interpreted as follows. Factor 1 is named 'task accommodation'. The measures which compose it have in common the demand to establish a cognitive 'readiness' set with which to process task-relevant information in a pre-attentional time frame. Factor 2 is a regulatory or 'trade-off' factor, in that good performance on one task reflects poor performance on another. It is named 'readiness modulation', a trade-off of distractibility for a more liberal response bias. Factor 3 is named 'selective processing'. All the tasks require selective processing of a stimulus array in conceptual, attentional and spatial modes. Factor 4 is another regulatory factor. It is named 'apprehension accommodation', in that better stimulus apprehension is traded for a more liberal response bias. Factor 5 is named 'scanning rate', because both tasks involve pre-attentional scanning of a complex stimulus field. Size estimation is traditionally associated with pre-attentional scanning, more rapid scanning being associated with overestimation. In the COGLAB vigilance task, conditions 2 and 3 require rapid pre-attentional scanning of the target/distractor array.

Notice that in the patients the regulatory or 'trade-off' relationships disappear, leaving a single factor wherein all the members vary in the same direction. The schizophrenics' factor is almost but not completely pervasive, including eight of the ten COGLAB measures.

The diversity of the tasks associated with the patients' single factor suggests that a single causal process is affecting different aspects of cognitive functioning. This could be the 'general deficit' or 'low capacity' factor frequently hypothesised in experimental cognitive studies of schizophrenia. However, at least one of the five 'normal' factors could also be interpreted in terms of processing capacity. If this is a low capacity factor, then it is different from capacity variations found within normals. It seems more parsimonious to say that schizophrenics' Factor 1 represents a relatively pervasive effect which probably originates outside the cognitive realm. At the same time, there are clearly complex differences between normals' and patients' cognitive performance which are not accounted by that single factor.

Here is another way to demonstrate the limited pervasiveness of the patients' single factor. If we were to compute the patients' Factor 1 scores for both groups, based on both normal subjects and patients' distribution,† about 38% of the patients fall within the normal subjects range of variance, or 'within normal limits' (Fig. 2). The shapes of these two distributions are very similar to those produced by a discriminant function, although the COGLAB measures involved are different. The discriminant function can be thought of as a factor expressly derived to separate or discriminate the two populations. The best cross-validated discriminant function correctly classifies 81% of all the subjects with respect to normal *v.* patient. The discriminant function has about half the group overlap of the patients' general factor. Nevertheless, it includes only four of the COGLAB measures. This means there are important differences within the patients that do not distinguish them as a group from normal subjects.

*Though we use the more general term 'factor' in our discussion, principle components are presented hereafter because we are interested in representing the pattern of interrelationships among particular tasks, as well as in 'capturing' the constructs which underlie the tasks. Parallel analyses using common factor technique produced equivalent statistical and substantive conclusions.

†This is referred to as an 'ordination analysis' (e.g. Pimentel, 1979). It may profitably be performed using factor scores even though the factor structure is not optimal for both populations being compared, as in this case.

NORMAL SUBJECTS

factor 1
+ apprehension
+ reaction time
+ vigilance
+ redundancy effect

factor 2
+ apprehension
− distraction effect
+ anticipatory errors

factor 3
conceptual processing
+ vigilance
+ mueller-lyer effect
− redundancy effect

factor 4
+ apprehension
+ vigilance false alarms

factor 5
+ vigilance
− mueller-lyer effect

SCHIZOPHRENIC PATIENTS

factor 1
+ apprehension
+ reaction time
+ conceptual processing
+ vigilance
+ distraction effects
+ redundancy effects
+ anticipatory effects
+ vigilance false alarms

FIG. 1 Comparison of factor structures of COGLAB in normal and schizophrenic subjects.

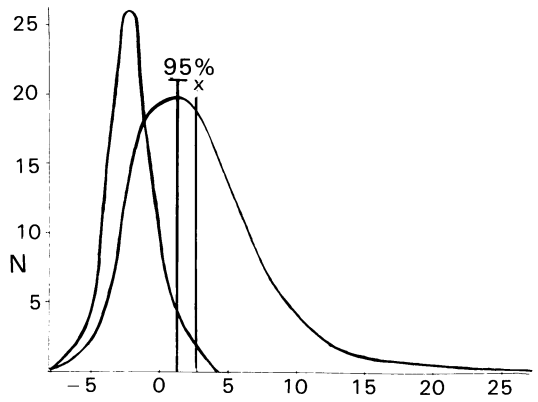

FIG. 2 Distributions of the patients' FACTOR 1 for patients (right-hand curve) and normals (left-hand curve), showing the normals' 95th percentile and the patients' mean.

We may tentatively conclude that there is indeed a relatively pervasive effect on cognition in schizophrenia, but it is not completely pervasive. There are important differences between groups not accounted for by a single deleterious effect. We have not specifically identified the functionally autonomous deficits, but the data suggest that they must exist.

The implication for clinical assessment and treatment is that the performance of any patient is influenced by two types of effect. The first is relatively general, affecting many but not all cognitive abilities. One aetiological hypothesis about this effect is that it represents a phasic process, possibly the same one that we have come to associate with the prodromal, acute and residual phases of schizophrenia. If it is phasic, it is probably linked to the neurochemical and psychophysiological processes which produce decompensation. If this is true, therapeutic normalisation of the general deficit would probably require intervention at the neurochemical or psychophysiological level, and this would logically precede treatment of cognitive deficits which are not so 'phase-linked'.

A second hypothesis about the general effect is that it represents a vulnerability-linked deficit. This is rather more speculative than the first hypothesis, in that there is as yet no reason to believe that vulnerable non-schizophrenic subjects are so pervasively deficient. In fact, data recently collected in our laboratory suggest that schizotypal subjects are as good or better than normals on some tasks.

A third hypothesis is that the general factor represents an accumulation of the effects of episodic psychosis, chronic neuroleptic use, illicit drug use and non-specific illnesses. This would be consistent with

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the well-known findings of neurological 'soft signs', diffuse neuropsychological deficits and cortical atrophy in chronic schizophrenic patients.

Longitudinal analyses are necessary to resolve the alternative interpretations of the general factor. We are currently undertaking such analyses in our laboratory. Great caution is indicated here, however. To take a lesson from the present results, we must not assume that the structure of cognition is the same in different groups or across time within a group. For example, if the general factor does represent a phasic or chronicity-linked effect, we may expect the factor structure of non-psychotic or less chronic patients to be more similar to that of normals. One cannot test this hypothesis merely by observing changes in the general factor score over time.

The second type of cognitive deficit in schizophrenia is specific to particular operations or abilities. Collectively, such deficits account for some part of the 70% of variance in schizophrenic performance not accounted for by the general factor. As with the general factor, we may hypothesise that specific deficits are linked to vulnerability, phase or chronicity. Additionally, we may ask whether specific deficits are associated with specific subtypes of schizophrenia or related disorders. Most importantly, specific cognitive deficits which are not phase-linked (and therefore not expected to respond to neurochemical or psychophysiological normalisation) are hypothetically the ones which will require highly specialised cognitive treatment procedures.

Having identified the general factor in COGLAB, we may now identify more specific deficits by 'subtracting' the general effect from patients' performance profiles. That is, deficits which remain after the general effect has been removed can be presumed to be functionally autonomous. We can begin to design cognitive treatments to address the general and specific deficits, and using batteries like COGLAB we can test the treatments in groups selected for homogeneity with respect to the deficit being treated. We can begin to determine which deficits are phase-linked and which are not, and we can then strategically sequence treatments accordingly. Thus with further multivariate analyses of COGLAB and other cognitive measures, together with longitudinal analyses of treatment effects, the logical and psychometrics prerequisites of cognitive therapy for schizophrenia can gradually be fulfilled.

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