Pergamon

PII S0272-7358(01)00104-0

IS IT TIME TO DEVELOP A NEW COGNITIVE THERAPY FOR PSYCHOSIS — COGNITIVE REMEDIATION THERAPY (CRT)?

Til Wykes

Kings College London, UK

Mark van der Gaag

Leiden University/Parnassia Research Centre, Netherlands

ABSTRACT. *The cognitive difficulties associated with the disorder of schizophrenia seem to be predictive of future dependence on psychiatric services and predict social functioning. Cognitive remediation therapy (CRT) was designed to rehabilitate these cognitive functions, and the interest in it has grown dramatically over the past ten years. However, the programmes are rarely based on clear theoretical principles and sometimes rely heavily on practice rather than guided learning. The evidence for their efficacy is variable and seems to be dependent on the use of specific components of training. The current review tries to put this evidence into context and sets out a programme of research, which is essential in this area if future progress is to be made. © 2001 Elsevier Science Ltd.*

KEY WORDS. Cognitive therapy, Psychosis, Treatment, Rehabilitation

INTRODUCTION

COGNITIVE REMEDIATION THERAPY (CRT) is a term we use in this review to describe a number of different methods of teaching ''thinking'' skills. It is similar to other thinking skills therapies currently on offer to people with psychosis, e.g. Cognitive Behaviour Therapy (CBT), but is also distinct in that it uses teaching material that has no personal relevance to the individual and is neutral in its affective content. CRT is also specific in that it focuses on those cognitive abilities that are said to be affected in a proportion of people with schizophrenia, e.g.

Correspondence should be addressed to Til Wykes, Professor of Clinical Psychology and Psychiatric Rehabilitation, Department of Psychology, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK. Phone: (20) 7848-0596. Fax: (20) 7848-5006. E-mail: t.wykes@iop.kcl.ac.uk

memory and attention, whereas CBT concentrates on reducing psychotic symptoms, particularly delusions.

Cognitive remediation therapy for psychosis can be traced back thirty years to the original studies of Meichenbaum and Cameron (1973) who worked with patients who were disabled by negative symptoms and a lack of behavioural control. Their technique was to encourage behaviour to be under the control of the therapist who spoke the instructions out loud. The patient then took control of the instructions and spoke them aloud and later said the same instructions in their own head. The result was that patients became more active and showed more initiative. Subsequently, monetary reward and verbal encouragement were shown to be effective in improving reaction time (Steffy & Galbraith, 1980), graduated distraction training improved attention (Adams et al., 1981) and improved memory was achieved when patients with schizophrenia were provided with an encoding strategy (Koh et al., 1976). This is a brief history of CRT up to the renewed and more thorough investigations in the 1990s of techniques that could improve cognition for people with schizophrenia.

Although the history of CRT can be traced back thirty years, it has only begun to be tested in clinical trials fairly recently. In our view this was due to the assumptions about the ways in which cognitive deficits are implicated in schizophrenia, which led to therapeutic pessimism. We review various putative relationships between cognition and schizophrenia with particular reference to the relationship between deficits and outcomes in order to ascertain if there is any likelihood of secondary gain following CRT. We present some models of cognitive deficits which, in our opinion, support the types of thinking skills of therapies that have been used to rehabilitate cognition, then we go on to describe the actual therapies with an evaluation of their successes and limitations. We have to be guided by the different strengths of the evidence. CRT developed with evidence from case studies, case series, controlled trials, and randomised control trials. But in the current corpus there is a dearth of data from randomised controlled trials, and even when it is available some trials fall short of the criteria for methodological rigour (see, for instance, The Standards of Reporting Trials Group [1994] for a list of such criteria). The final outcome measures of these trials also differ, making it hard to draw relevant comparisons between them. Unlike other reviews, which have generally concentrated on the identification of effect sizes from disparate studies (e.g. Hayes and McGrath, 2000; Suslow et al., 2001), we will try to extract similarities between studies of CRT so the general principles for success can be used in its further development.

A pictorial guide to the increased interest in the rehabilitation of cognitive difficulties, as measured by publication rate, is given in Fig. 1. These publications are not about one consistent type of therapy; rather, the increase in publications camouflages a number of disparate procedures that have been adopted under the general heading of interventions for cognition, and some have been more successful than others in achieving change. The theoretical perspectives also differ widely with some authors, generally in the USA, referring to neurocognition as if the intervention was specifically aimed at training brain systems, whereas others, usually in Europe, refer to thinking styles and information processing strategies. These two points of view could lead to different developments in CRT. We will argue for those continuing to carry out research into CRT to be more specific in their overall goals so that future meta-analyses can adequately reflect on specific therapeutic content. This heterogeneity disguised under one therapeutic label is likely to hamper further developments.

We will conclude that more information is necessary, both regarding which cognitive processes should be the targets for therapy and the clinical efficacy and
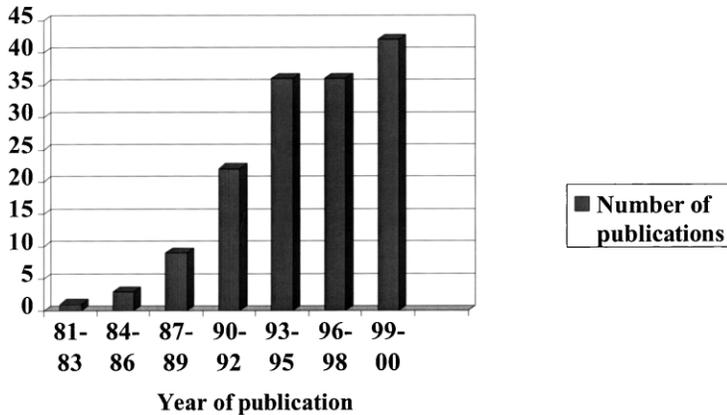
FIGURE 1. Number of Publications over Time on Cognitive Rehabilitation for Schizophrenia.

effectiveness of today's most successful techniques. Two well-controlled studies of CRT have shown links to improvements in cognition and modest changes in social functioning, but because of the dearth of sound RCT evidence we cannot make unequivocal claims for the inclusion of CRT as a technique in clinical practice. We can conclude, however, that it is a process that shows promise. We will also conclude that CRT should not be a stand-alone therapy in clinical practice, but rather it should be included in comprehensive programmes for rehabilitation and recovery so that any improvements following CRT can be exploited in further training or recovery therapy.

BACKGROUND

Central to Kraepelin's (1913) conception of schizophrenia or dementia praecox was the notion that they were cognitive deficits, especially those of attention. He also considered dementia praecox as a neurodegenerative disease, and though this conception has persisted for many decades, research findings in recent years favour a neurodevelopmental theory of schizophrenia over a degenerative one. This means that there is not an ongoing destructive process in schizophrenia; rather, the neurodevelopmental theory proposes that the architecture of the brain is slightly derailed by genetic and/or environmental causes during pregnancy or by birth complications. This ''damage'' results in a progressive developmental delay until the first episode in adulthood, in which the organisation of information processing and subjective experience collapses. From this moment on, no significant further deterioration takes place (Hoff et al., 1999; Rund, 1998). This model predicts subtle cognitive deficits before the onset of psychosis, and severe and quite stable cognitive deficits after the first episode. During psychotic episodes, some additional cognitive functions will be disturbed, but these will recover after the remission of symptoms.

Which Cognitive Functions Show Deficits in Schizophrenia?

Most patients with schizophrenia show variable performance across a number of different tasks. They are not usually uniformly poor. Some tasks show mild inefficiency, with performance below the mean but within normal ranges. Some tasks show

moderate deficits (defined here as 1 to 2 standard deviations below the mean), and severe deficits (if there is a performance decrement of 2 to 3 standard deviations). The most severely disabled patients sometimes perform 5 standard deviations below average, and, as a group, 85% of the patients with schizophrenia perform below normal on one or more cognitive domains (Harvey & Serper, 1999). In a normal population only 5% have such low standards of functioning, so cognitive deficits can be considered to be a core feature of schizophrenia.

Mild impairments are found in perceptual skills, delayed recognition memory, confrontation naming, and verbal and full-scale IQ (Goldberg et al., 1989; Gruzelier et al., 1999; Harvey et al., 1995; Nathaniel-James et al., 1996; Purcell et al., 1998) whereas patients with Alzheimer's disease are have severely impaired recognition memory and naming skills (Saxton et al., 2000). Moderate impairments of 1 to 2 standard deviations below the mean are found on tasks that assess distractibility, delayed recall, memory span, and working memory (Aleman et al., 1999; Goldberg et al., 1989; Harvey et al., 1995; Spindler et al., 1997; Verdoux & Liraud, 2000) and severe impairments (2–3 S.D.s below the mean) have been found in serial learning, executive functioning [as measured by card sorting], vigilance, motor speed, and verbal fluency (Morice & Delahunty, 1996; Velligan et al., 1996; Bilder et al., 2000; Kelly et al., 2000; Riley et al., 2000). The profile of cognitive deficits found in patients with a diagnosis of schizophrenia is therefore different to that found in patients with Alzheimer's dementia. But there seem to be only quantitative differences between those with a diagnosis of schizophrenia and those with either bipolar disorder or delusional disorder, with patients with schizophrenia being the most disabled patients (Hawkins et al., 1997; Rossi et al., 2000; Goldberg, 1999).

The Nature and Aetiology of Cognitive Deficits in Schizophrenia

We propose to make a distinction in cognitive deficits to further clarify the aetiology and nature of cognitive deficits (see Table 1). These are:

(i) *Trait deficits* that have been present long before the outbreak of schizophrenia and which do not (or only mildly) become aggravated during the course of schizophrenia, even when episodes of illness occur. For instance, symptoms improve with classic and modern anti-psychotic medication, but the performance of some tasks, e.g. performance on the CPT, is not affected (Liu et al., 2000). In addition, other evidence for this category of deficits comes from studies of developmental delay in reaching milestones and mild cognitive disorders that can be measured before the first episode. Subtle cognitive deficits result in mild academic performance decrements as compared to their siblings and normal controls, and some developmental retardation. Recruits to the army in Sweden who later develop schizophrenia had

TABLE 1. A Distinction of the Nature of Cognitive Deficits Associated to Aetiology and Evolution Symptoms of Schizophrenia

	Premorbid	During episode	In remission
Trait deficit	Mild	Mild	Mild
Acquired deficit	Absent or Mild	Severe	Moderate to Severe
State deficit	Absent or Mild	Severe	Absent or Mild

lower IQ scores than their age- and sex-matched peers (David et al., 1997). Cognitive deficits and delays in childhood in a New Zealand birth cohort have also been shown to distinguish those children who develop schizophrenia or schizophreniform disorder from those who later develop other psychiatric diagnoses, and similar results have been shown in school children in Finland (Poulton et al., 2000; Cannon et al., 1999). Deficits in attention and memory can also be detected in children at genetic risk for schizophrenia, and these deficits can predict the development of schizophrenia (Erlenmeyer-Kimling et al., 2000). These data confirm the importance of poor cognitive ability as a risk factor for schizophrenia and other psychoses. The association could be directly causal, with cognitive impairment leading to false beliefs and perceptions, or it could be indirect, with any factors causing lower IQ, such as abnormal brain development, increasing the risk for schizophrenia.

(ii) *Transient state-dependent deficits* that are strongly associated with symptoms. Selective attention, as measured by the Stroop task, is an example of a task that is performed poorly by some psychotic patients (Baxter & Liddle, 1998) but performance improves in remission (Nopoulos et al., 1994) making it a transient-stage-dependent deficit.

(iii) *Acquired deficits* that are mildly present before the first episode but aggravate into a severe cognitive deficit in the months before and during the first episode and remain stable after psychosis. For example, only very mild memory deficits are found in the pre-morbid phase, and they interfere only minimally with everyday life and school performance. In the months before, during, and after the first episode, these memory deficits become severe and hinder all kinds of learning and adaptation to new environmental challenges (Saykin et al., 1994). In addition, social and vocational functioning decline during this prodromal period (Hafner et al., 1992). Cognitive processing seems to become remarkably stable after the first episode (Censits et al., 1997; Hoff et al., 1999). The stability of these deficits has been demonstrated in numerous studies. For instance, there are no differences in cross-sectional studies in the cognitive functioning of young patients with a short duration of illness, old patients with a short duration of illness, and old patients with a long duration of illness (Heaton et al., 1994; Jeste et al., 1995); no differences between adolescent and chronic patients (Goldberg & Weinberger, 1988); no differences between first episode patients and chronic patients (Albus et al., 1996; Hoff et al., 1992); and no differences between age groups (Goldstein et al., 1991; Hyde et al., 1994). The evidence is therefore in favour of stability in most cognitive deficits.

We have tried to describe as briefly as possible the ways in which cognitive deficits have been linked in the life course of the disorder of schizophrenia. This sort of categorisation is independent of the cognitive targets chosen for CRT and has no implications for whether or which particular deficits are likely to be open to amelioration. However, the opposite may be true; CRT may have implications for such a categorisation. If it is discovered that some trait deficits can be restored, then it is clear that developmental delays are not biologically inflexible and may be affected by the interaction with environmental support (e.g. experience or teaching).

The Relationship of Deficits to Symptoms

The most accepted assessment of the relationship between cognition and the most spectacular symptoms is that there is none, and yet it seems clear that thinking skills, which underlie performance on cognitive tests, must also be important in determining

the interpretation of abnormal events into a delusional belief. However, the majority of published data show that cognitive deficits share only a small common variances with symptoms, and the most spectacular symptoms like delusions and hallucinations have an almost absent relation to cognition as measured by neuropsychological tests (Cuesta & Peralta, 1995; Frith, 1992; Nuechterlein et al, 1986; O'Leary et al., 2000).

This lack of relationship may be merely a reflection of the lack of subtlety of the current measures of both symptoms and cognition. Symptoms are often measured as the total score from a disparate group of delusions, hallucinations, passivity phenomena etc., and the scoring often seems to confuse both frequency and severity (interference with everyday life). More refined psychological measures such as PSYRATS (Haddock et al., 1999) may allow these more subtle relationships to be determined. The correlations measured are also usually with the current presence of symptoms, whereas the relationship may be more complicated. Cognitive difficulties may play a causal role in the propensity for symptoms, and will therefore not distinguish within the group of patients with diagnoses of schizophrenia, as a large number by definition will have had symptoms of passivity, auditory hallucinations, etc. In addition, the effect of a cognitive difficulty may only be clear if the cognitive system is stressed in some way so that subtle difficulties, which can be compensated for under normal circumstances, break down.

Delusions and hallucinations. A comprehensive description of the possible relationships between cognition and some positive symptoms has been given by Frith (1992). He suggests that a number of different thinking skills are related to individual symptoms. For instance, the ability to self-monitor seems to be related to the experience of hallucinations. In one study, patients with hallucinations, unlike normal control participants, were asked to distinguish between their own voice saying a word and someone else's voice when this voice was presented at the same time to the patient. The patients' problem was in distinguishing their own voices, which they mistakenly said were alien voices when the voices were distorted. In other words, the patients who experienced hallucinations were biased in their interpretations when the task became a little more difficult (Johns & McGuire, 1999).

Frith suggests that some delusions, such as delusions of control by alien forces and thought insertion and passivity phenomena are a result of the person's inability to recognise his/her own actions, and particularly the person's difficulties in monitoring his/her own intentions to act. This difficulty prevents the person from distinguishing actions and events that are caused by external agents and those that are the result of his/her own goals and plans. This can also lead to passivity phenomena as the person attributes his or her own movements to external causes.

Negative symptoms. Current data do link negative symptoms to cognitive deficits, although the common variance is seldom more than 15% (Addington & Addington, 1993; Addington et al., 1991; Breier, et al., 1991; Liddle, 1987; O'Leary et al., 2000; Wong et al., 1997). When negative symptoms wane, cognitive deficits ameliorate (Censits et al., 1997; Gold et al., 1999).

Disorganisation. The person with disorganisation is unable to continue with his or her intentions as he or she is totally distracted by each novel stimulus. This particular characteristic is, as might be expected, linked to a number of cognitive difficulties, particularly planning and the selection of responses to inhibit impulsive first reac-

tions (Cuesta & Peralta, 1995; Mahurin et al., 1998; Norman et al., 1999; Nuechterlein et al., 1986; Passerieux et al., 1997; Racenstein et al., 1999; Rowe & Shean, 1997; Van der Does et al., 1996).

Depression.   Depression is associated with encoding problems in memory (Brebion et al., 2000; Holthausen et al., 1999, Gorman, 1997). In schizophrenia both depression and memory deficits are quite frequent, and it is unclear what the causal relations to social outcome are.

Predictive Value of Cognition

In a meta-analysis, Hegarty et al. (1994) showed that a century with varying medical interventions has not dramatically improved the social outcome of patients with schizophrenia. Anti-psychotic medication raised the percentage of patients with moderate to good social outcome from 25% at the beginning of the twentieth century to about 35% in the mid-nineties. Positive symptoms such as delusions and hallucinations do not have high predictive value for outcome in most meta-analyses, while verbal memory, vigilance, and executive functioning have been shown to strongly predict social outcome (Green, 1996; Green et al., 2000). For example, in a series of studies of the closure of a large mental hospital in the UK, Wykes and colleagues found that patients who had a deficit in their ability to select correct responses and inhibit alternative responses were more likely to remain in services that provided high levels of psychiatric care (Wykes et al., 1990; Wykes, 1994). In their study neither symptoms nor initial social behaviour added much to the predictive value of cognition. The predictive value also does not seem to be confounded by initial functioning levels. So, some cognitive deficits are the best predictor of social outcome, but few other studies correct for symptoms or initial social functioning on outcome, and it has been suggested by Van Os & Verdoux (2001) that studies should correct specifically for disorganisation, negative symptoms, and depression when assessing the significance of cognition as a predictor.

   Table 2 shows the predictive value of cognitive measures for social outcome in 37 studies (Green et al., 2000). Strong predictors are replicated findings in four or more studies, while the weak predictors are replicated in 2–3 studies. Secondary verbal memory was reliably related to every outcome domain. Card sorting was related to community outcome, and vigilance was associated with skill performance. A meta-analysis showed highly significant p-values ($p < 0.000001$) with medium to large effect size for immediate verbal memory, medium effect size for secondary verbal memory, and small to medium effect sizes for card sorting and vigilance.

TABLE 2. Cognitive Predictors of Functional Outcome

Outcome domain	Strong predictor	Weak predictor
Community/daily activities	Secondary verbal memory Card Sorting	
Social problem solving	Secondary verbal memory Vigilance	Card Sorting
Psychosocial skill acquisition	Secondary verbal memory Memory span	Card Sorting Vigilance

We hope we have set out some cogent reasons why cognitive difficulties or thinking skills may be a potential source of difficulty for people with schizophrenia. They seem to be linked to social outcome as well as to symptoms, and therefore their rehabilitation may have implications for both types of functioning. There are three possible ways in which these difficulties might be treated in order to affect outcome:

1. providing direct cognitive treatments aimed at improving cognitive skills, which may then relieve a number of problems (e.g. reducing the effect of cognition as a rate limiting factor in other rehabilitation programmes);
2. changing rehabilitation programmes so the treatments are designed to enhance individual strengths rather than weaknesses (e.g. errorless learning approaches);
3. changing the environment so it has less effect on cognition, a model similar to that adopted for the people with dementia.

Clearly all three approaches are probably warranted, but this review is mainly concerned with the first: direct intervention for thinking skills. There is no theoretical evidence to suggest that changes in cognitive processing will have a direct effect on outcome, but there is no evidence to the contrary either. Only studies where cognition is the target in a relatively stable population will verify the direct nature of the association. So this approach is not only valuable in its putative practical effects (improved cognitive functioning) but it is also necessary if we are to test some theoretical relationships between cognition and functioning. Below is a description of the models on which the various attempts to improve cognition have been based.

WHICH MODELS HAVE BEEN USED IN CRT PROGRAMMES?

The Learning Model

Silverstein et al. (1998) used shaping procedures to teach sustained attention to patients who could not focus their attention for more than two minutes in a social skills training programme. By setting small incremental goals he shaped patients to sustain their attention to the training situation minute by minute. He succeeded in doing so by reinforcing attention behaviours such as eye contact and verbal and non-verbal responding.

Another example of shaping behaviour through environmental control is Cognitive Adaptational Training (CAT; Velligan et al., 1996, 2000). This compensatory approach is based on using environmental cues and prompting to shape behaviours, particularly those for apathetic behaviour. Signs and equipment for everyday activities are placed directly in front of the patient (e.g. signs for the steps involved in brushing teeth) to cue and sequence behaviour. The effects of possible disinhibited behaviour are reduced by organising the environment to prevent inappropriate use of equipment. For example, outfits with one item each of all the appropriate clothes for a day are placed in individual boxes so the patient does not put on many different layers of clothing. The results of a randomised controlled trial comparing CAT, a treatment control, and a follow-up group indicate that patients who received CAT improved their level of symptoms and global functioning over a nine-month period when therapy was provided. Although measures were assessed blind to the treatment group and there was random assignment, the authors used a rather primitive analysis method for their

end point evaluation. They used the last observation carried forward, which is known to be problematic, and repeated measures ANOVA (see Everitt, 1998 for a discussion). There is also a lack of adequate data description in the paper, which makes it impossible to evaluate whether this biased the results, although it is clear that these problems could also have affected the control groups (relapse 67% and 33%) more than the CAT group (relapse 13%). Despite these inadequacies, these data do suggest that changing the environment can affect overall functioning. However, data on the step from stimulus-control to self-control that is usually expected for this type of clinical service are absent. This is not part of the CAT programme. It is therefore not clear whether this type of intervention will allow for the maintenance of functioning improvements when it is removed or whether the effectiveness of therapy is dependent on continual external support.

The Retraining Model

Most retraining experiments have been carried out in academic laboratories with the focus on a single deficient cognitive function, which is retrained using instruction and repeated exercise. The targets have been attention, mnemonic function, and performance on neuropsychological tasks such as reaction time, card sorting, and verbal memory. In general the retrained function improves, sometimes to normal levels. For example, computer training using 18 sessions resulted in improved performance on the CPT as compared to a control group (Medalia et al., 1998). More complex cognitive functions have also been suggested for retraining with computer assistance (e.g. Olbrich & Mussgay, 1990). Although the functions can be retrained, the recovered function does not (or only weakly does) generalise to other cognitive domains, and it is questionable whether improved function has any consequences for the functioning of patients in everyday life.

Neither the learning model nor the retraining model take into account either the psychology of delusions or hallucinations, or which cognitive deficits are characteristic in schizophrenia. One of the first models that did take this into account was the model by Brenner et al. (1980) called *Integrierties Therapie Programm (IPT)*.

Hierarchical Model

The IPT programme is based on an assumed hierarchical model of cognitive and social processes that is unlikely to be accepted now in such a simplified form. At the lowest level, neurotransmitter irregularities are said to be responsible for information processing disorders. These information processing disturbances lead to disordered psychological processing and hence the appearance of symptoms and social and community functioning deficits. At an even higher level it is suggested that the symptomatic and functioning difficulties lead to rejection by society and a life of alienation and loneliness. Therapy was thought to be a hierarchical process as well. If medication compensates for the neurotransmitter disturbances, new therapeutic avenues can be approached by first building up cognitive processing, then social perception, social skills, and problem solving later. The model presupposes that symptoms cannot be ameliorated unless cognitive and thinking disorders have been ameliorated.

The assumption of the hierarchy not only as a model of the relationship between physiological and psychological processes but also as a model for therapy would not now be accepted by the majority of psychologists. More sophisticated models with

empirical support suggest that there are psychological effects on basic physiological mechanisms. For example, changes in brain mechanisms measured by MRI scans can result from cognitive treatments (Wykes et al., 2001; Baxter et al., 1992).

Neurodevelopmental Model

Fundamental to the neurodevelopmental model is a vulnerability for psychosis that is partly genetic and partly environmental, where the vulnerability changes depending on the developmental stage of the individual, and the highest likelihood is during late adolescence. The environmental causes are usually defined as those in the uterus and can be multiple physical stressors such as viruses (McGrath et al., 1994), food deprivation (Susser et al., 1996; Susser & Lin, 1992), atomic bomb radiation (Imamura et al., 1999), or the death of the father during pregnancy (Huttunen & Niskanen, 1978). The vulnerability, e.g. subtle attention deficits, in ''at risk'' children results in a developmental delay that increases with age. Hogarty and Flesher (1999) use this model and consider this delay in development as the target for intervention. Patients have missed opportunities to learn, but learning still has to take place. Although this seems a sound argument, it is not obvious how the specific cognitive remediation programme relates to the cognitive developmental model or how the specific tasks and interventions were chosen.

Energetical Model

The energetical model, proposed by Sanders (1983), differentiates computational mechanisms from energetical mechanism. The patient can complete the computational level stimulus perception, response selection, response preparation, and response. However, deficits occur in the energetical mechanisms. These mechanisms are: arousal on the perceptual side, activation on the motor side, and effort as a general supervising process. Arousal modifies selective attention. Overarousal results in a tunnel view with little exploration of meaning, and underarousal results in distraction by irrelevant stimuli. The model suggests that overactivation results in negative symptoms and mutism and underactivation result in disinhibition of behaviour. Effort balances arousal and activation according to task requirements and is essential in the planning and supervision of complex behaviour.

The remediation programme by Van der Gaag (1992) was partly based on this energetical model and partly on compensatory strategies. Emotion perception was shaped by gradually increasing perceptual working memory load and gradually integrating visual, auditory, and proprioceptive modes in emotion perception. Emotional response-provoking stimuli were also gradually added to prevent disruption of the information processing by emotions. Deficient emotion recognition in facial expression is a pre-attentive process, so patients were taught to compensate for their lack of skill by learning the discrete features of emotion displays (e.g. anxiety is the only emotion in which the white of the eye above the iris is displayed).

Executive Functioning

Shallice (1988) developed a model of executive functioning that included a supervisory attentional system. This system defines the types of processing of information that must be available to complete a task efficiently and effectively. The model was

devised following empirical data from people with damage to their frontal lobes. People with diagnoses of schizophrenia have some similar difficulties to these patients: they lack self-initiated activity, show perseverative responding, and make inappropriate responses to stimuli. The supervisory attention in Shallice's model is responsible for modifying the strengths of competing action systems through its interaction with the environment so that inappropriate responses can be inhibited and novel actions can be triggered. This model was the basis for the work of Delahunty et al. (1993) and Wykes et al. (1999a, 1999b). They used a programme that targeted key features of the cognitive deficit profile of people with schizophrenia (defined in terms of Shallice's model) and designed a programme that taught patients information processing strategies or thinking skills which could be used to solve many different everyday tasks. This may be thought of as compensating for deficits, but may also be seen as the development of contention schedules that are used automatically in solving problems. In effect, the programme increased task performance not by increasing the capacity of the process but by engaging the participant in a number of processing strategies that reduced information overload. For instance, planning was improved by encouraging the participant to break up the task into smaller sub-goals. Information about sub-goals was then rehearsed or held in mind using other mnemonic techniques. Sub-goals were monitored for the correct output before going onto the next part of the task. This model has the benefit of being based on those underlying cognitive deficits that characterise the performance of people with schizophrenia.

In summary, a number of different theoretical models have driven efforts to devise cognitive remediation programmes. The learning model seems to be a misnomer as it is not clear that anything is actually learned. Rather it seems that behaviour comes more under environmental control, and there is little evidence that when these environmental triggers are removed that there will be any continuation of behaviours. The model may be useful for a group of patients who are severely disabled and who lack any basic engagement with treatment programmes. However, as stand-alone systems they provide very low potential for a recovery process.

Three models (retraining, hierarchical, and energetical) assume that the system of processing information is functional but that with some re-training of specific systems or adjunctive systems they can be reinstated. Although it is not made explicit, these models seem to suggest the most optimistic outcome, as intact functions could produce full recovery. The remaining two models (neurodevelopmental and executive functioning) suggest a delay in the maturation of an information processing mechanism that may not necessarily be able to be reinstated. These models are therefore more pessimistic about the process of change and the likelihood of reinstating functioning.

Although different models have been drawn on in the development of CRT it is not clear that models themselves lead to more or less successful programmes. However, they do define the sorts of learning mechanisms which may be incorporated into a CRT programme.

HOW SUCCESSFUL ARE CURRENT REMEDIATION PROGRAMMES?

There are two main strands of research that contribute to the development of CRT: laboratory and clinical studies. The two strands are independent in that they are often carried out in parallel with very little crossover of knowledge between the two. They

provide separate but complementary evidence for the development of successful CRT and will be reviewed separately.

Laboratory Studies

The studies reviewed under this heading concentrate on the rehabilitation of a single task or component of a task and they are never integrated into general rehabilitation services. However, these studies are useful because they have helped to identify those components of the learning process that seem to be useful to people with schizophrenia and—perhaps even more importantly—those components that are not helpful and that lead patients to experience yet more failure.

Many of the studies in the laboratory have concentrated on a single task, the Wisconsin Card Sort Test (WCST). This followed a paper by Goldberg et al. (1987) that suggested that the deficit in performance on the WCST was a stable deficit that was not possible to remediate because the deficit was one of frontal lobe functioning abnormalities. However, both these propositions have now been disputed as some procedures seem to be more likely to improve performance than others, and improvements in behaviour may also be changes to strategies rather than changes in brain functioning (Wykes, 1998).

Although it is difficult to untangle the specific components of the teaching method in these studies there are some general methods, which have been summarised in Table 3. The table also shows the number of studies that have included each of these methods and what proportion have shown successful learning. Some studies have been counted under several headings as they have included several different techniques. Details of the eighteen studies, which have been reviewed for this summary table

TABLE 3. Different Teaching Strategies in the Laboratory and their Success Rates

Type of techniques	Detail of technique	Success rate
1. Card by card instruction	Total learning support plus trial and error correction	1/3
2. Didactic teaching	Told of rules then prompting, feedback and error correction and later reminders of rules. More practice and more reminders than card by card instruction	6/7
3. Monetary reward	Paid for correct performance and have didactic training in addition	1/5
4. Simplify task	Reducing task complexity often in conjunction with scaffolding instruction.	1/2
5. Verbalise action criteria	Overt verbalisation of the task instructions, preventing loss of goal and encouraging planning	2/2
6. Errorless learning	Based on backward chaining techniques where cues for successful performance are faded	2/2
7. Scaffolding	Task tailored to participants' current knowledge with errors reduced considerably. Participant has high potential for correct performance	3/3
8. Practice on similar tests	Similar tasks requiring similar cognitive skills are practised over long periods of time	1/4

are given elsewhere (Wykes, 2000). Table 3 ignores methodological difficulties and possible causal misinterpretations for individual studies. There are not enough studies in any one type of therapy, and the measurement of improvement differs so much between them that it is impossible to carry out a meta-analysis. The main goal here is to try to identify learning procedure themes that may lead to success or otherwise.

As can be seen in Table 3 there are a number of different teaching techniques that do not seem to be very helpful. Neither paying people to learn the task has little effect nor does practice on similar tasks. The most helpful techniques seem to involve some learning effort on the part of the participant and some reduction in the learning of errors (errorless learning, scaffolding, speaking the action criteria aloud).

Verbalisation techniques may be helpful to this group not only because they are a control over behavioural disinhibition, but also because they can provide a support for people who have problems with verbal memory by providing spontaneously generated rehearsal. Errorless learning — and scaffolding in particular — may be suited to teaching adults whose disabilities have led them to many experiences of failure. These techniques support the learner to produce successful behavioural outputs, which the person can monitor. It is likely that these experiences of success can also affect self-esteem which is known to affect problem solving ability and particularly the time a participant is willing to spend on a task. These techniques have also been shown to be helpful in normal learning environments. Unfortunately, the historical background in education and learning has generally been ignored by many rehabilitation researchers and has led to wasted effort of both patients and researchers.

We want to highlight the issue of practice here as it seems to have been investigated in laboratory studies, though it is also cited as a component of interventions in clinical studies. Practice is not a well-defined learning process. It can mean making processes that are under conscious control become more automatic and therefore take less cognitive effort. Alternatively, practice could lead to the identification and use of different information processing strategies that are more efficient for a particular task. Both options have been suggested by the authors of CRT studies. If learning efficient information processing strategies is the key, then practice is a particularly hit-or-miss approach. It is not only dependent on individual characteristics but also on chance variables occurring in the task from day to day. This may be why the effects of some types of CRT are so variable. For instance, in a study of improving the WCST, Stratta et al. (1994) found that about that one third of their participants did not gain any improvement, one third made gains but lost them at follow-up, and one third made gains that were still obvious after treatment had ceased. Unfortunately the sample was small, although Stratta and colleagues suggest that the poor performers suffered from a lack of planning so that they effectively performed at random.

There is little evidence from laboratory studies that these techniques have durable effects as there were few examples of any follow-up, and it was rarely more than a week. But there were some examples that did demonstrate durability and show an effect of CRT on task specific behaviour (e.g. Stratta et al., 1994) but again this was not true for everyone in their study (see above). There was also no indication of which characteristics were essential to these durable gains.

Clinical Studies

In contrast to the laboratory studies of instruction, clinical studies teach a wide range of tasks or component cognitive skills. The key idea is that changes in cognitive

performance will lead not only to improved cognitive ability but also changes in social functioning. Training elements from the laboratory studies are often involved in these more comprehensive programmes, although there is also an emphasis on the practice and rehearsal of specific cognitive abilities. All the programmes consist of a number of different types of training tools, including paper and pencil activities, computerised activities, and group activities. The main programmes are described below with an evaluation of the type of instructional activity and the efficacy as measured by a selective review of the outcome data.

Integrated Psychological Therapy. Integrated Psychological Therapy (IPT; Brenner et al., 1994) was one of the first clinical programmes to be well developed. IPT involves a number of different sub-programmes, only one of which focuses on cognitive abilities. Activities are run in a group format where training is didactic. Controlled studies of this approach have produced variable results. Most do show improvements in cognitive functioning, although there is little support for the subsequent effects on social skills (Brenner et al., 1994). However, the majority of these studies, although controlled, have a number of methodological difficulties. Participants were not randomised (Heim et al., 1989), ratings were not carried out blind to treatment status, and sample sizes were small (Roder et al., 1987). The most well-controlled study, by Funke and colleagues, did not show cognitive improvements following IPT (Funke et al., 1989). In fact, more recent evaluations have suggested that it is not possible to conclude that cognitive improvements are specific to the cognitive sub-programme rather than the remaining psycho-social sub-programmes (Hodel & Brenner, 1994).

In a recent tightly controlled study, Spaulding and colleagues in Nebraska have addressed the problem of the specificity of cognitive interventions and have tried to tease out the specific effects of the sub-programme. Patients in their study received either the cognitive sub-programme of IPT plus social skills training or social skills training alone. Both of these therapies were provided within an inpatient intensive rehabilitation setting. Their results do suggest a specific effect of the cognitive sub-programme on improvements in social skills, although there are few unequivocal cognitive improvements (Spaulding et al., 1998, 1999). The main links appear to be between changes in ''top down'' modulation (e.g. rapid processing of stimulus features) and social competence. In other words, the differential change was in executive level processing rather than attentional or pre-attentional processing. More recent analyses have suggested a direct effect of improvements in social competence with improvements on card sorting, and that improvements in verbal memory were associated with improved psycho-social skill acquisition (Spaulding et al., 1999). Spaulding points out that one problem for IPT is group training. Whereas this may be appropriate for some skills, he reports that some patients found the overall level of the group to be too low and were bored, whereas others found the level too high and found it difficult to keep up with other participants. This lack of individual tailoring is problematic with IPT and is certain to affect the overall outcome. Because of the nature of clinical samples it is highly unlikely that homogenous groups can be found because of the variable cognitive profile in people with schizophrenia.

Van der Gaag (1992) also tested a programme derived from the work of Brenner and colleagues, but his programme had a stronger dependence on experimental research of the deficits of people with schizophrenia and individual training. He found improvements in the perception of emotion in faces, which was targeted in the training. There was some generalization to executive functioning (mazes, word fluency, and

WAIS picture arrangement), but attention and memory functioning were unaffected. The findings should be interpreted with an awareness of some of the more salient methodological limitations. The sample size was modest, which provided enough power to detect medium to large effect sizes, but may have been slightly underpowered to detect small group differences in performance. The findings lend some modest support for generalization to executive functioning, but the possibility that generalization effects are due to Type I error cannot be ruled out. A second concern is the relatively short duration of training. Training took place in a total of approximately eight hours of instruction (two 20-minute sessions each week over three months) plus another eight hours of homework assignments. It is possible that stronger generalization effects would have emerged had training been intensified and some bridging mechanisms inserted into the training exercises to facilitate generalization. A third concern is the lack of data on symptoms. By not including symptom measures in the study, claims about the relative independence of training effects apart from improvements in symptoms cannot be made.

Cognitive Enhancement Therapy. Cognitive Enhancement Therapy (CET; Hogarty & Flesher, 2000a, 2000b) assumes the neurodevelopmental model of schizophrenia deficits and uses the notion that anomalies in development produce delays in social learning. The programme is aimed at higher functioning patients, i.e. those who are ''stable outpatients.'' These patients are assessed and divided into three groups; (a) impoverished, (b) disorganised, and (c) rigid, on the basis of their cognition. For example, someone who is described as rigid would show relatively few hallucinations but a persistence of mild delusional thinking. They are also characterised by having difficulties in developing alternative responses to social problems as the requirements of context change and they are disabled by having fixed and restricted cognitive schemata and a reduced tolerance for ambiguity.

CET's goals are to facilitate the attainment of social cognitive milestones by providing meaningful and self-directed experiences rather than responses based on role modelling in contrived situations. The programme uses computerised training of memory and attention based on software devised for the rehabilitation of people with closed head injuries. However, in each programme session one patient is paired with another whose cognitive problems are different than his or her own. The pair collaborate on the software exercises and maintain records of performance. After three months patients enter larger groups of 6 to 8 people who socialise and work together on the programmes. In total, the patients take part in 6 months of nonsocial cognition training before they begin the social cognition training. This consists of group exercises that focus on ''gistful'' interpretations of information such as summing up an article in a newspaper to another person. The patient collaborator or the therapist will try to encourage the speaker to be as clear as possible in their communications. Non-participant members of the group remain silent during an exercise but they are expected to take notes and give feedback. Each of the group sessions for social cognition is completed with 15 minutes of psychoeducation.

Data on this programme are sparse, as are methodological details. The first 44 patients receiving CET are reputed to show that there are significant increases in nonsocial cognition as well as on social cognition and disability. For the control group there are few changes in any of the measures. No information is provided about the costs of the therapy.

A similar programme has been carried out by Bell et al. (2001) together with a vocational rehabilitation programme. Participants in their study were randomised to two conditions, CRT plus work rehabilitation and work rehabilitation alone, although the assessments were not carried out blind to group membership. These authors found a significant benefit on cognitive performance following the CRT condition in both cognitive flexibility measures, working memory, and ''thought disorder'' (as measured by a series of tests including logical memory). No data are yet available on the work rehabilitation outcomes between the two groups.

This form of therapy is similar to some of the modules produced by Brenner et al. (1994) for improving social cognition but also includes computerised testing, which is discussed below.

Computerized Training

The rehabilitation of cognitive functioning using computers has a relatively long history beginning with the development of software programmes for people with closed head injuries. It is clear that these programmes improve performance on the tasks that are practised, but there is much less information on their ability to generalise the skills to other domains or other settings. For instance, Dorn (1996) used verbal problem solving software and showed improvements in this skill but not in nonverbal problem solving.

Most of the deficits targeted are attention skills rather than problem solving ability. The results are extremely mixed. In a recent review by Suslow et al. (2001) that concentrated on attention training, the authors found that 19 out of 35 outcome variables showed no performance improvement. A training effect was observed for only 10 variables (e.g. Olbrich & Mussgay, 1990; Hermanutz & Gestrich, 1991) but for most of these outcomes there exist negative results. Benedict and colleagues (Benedict & Harris, 1989; Benedict et al., 1994) carried out training using a limited number of tasks with around 150 hours of training. The first study suggested differences even when compared to an attention placebo and a no treatment control, but in the larger study that compared only treatment versus treatment as usual, improvements were found only on the training tasks. In contrast, Medalia et al. (1998) showed improvements in scores on the Continuous Performance Test (a measure of attention); surprisingly, the symptoms of the treated group also declined.

The results of this type of CRT are currently very mixed. The two well-controlled studies have produced opposite results. The training programmes—although similar—were not identical, and the size of the sample in the Benedict et al. study may not have been large enough to detect modest effect sizes. In addition, two different forms of the CPT which may be differentially sensitive to specific attention training were used as outcome measures. But a further possibility is the differences in the level of functioning at outset. In Benedict's study, not only were the treatment and control groups not matched for pre-treatment scores, but there was considerable variability in terms of the amount of change within the training group. A median split revealed that the ''improved group'' changed their scores by 205 seconds whereas the poorer responding group only changed an average of 31 seconds. The rather unsophisticated correlation analysis of change scores did not indicate that success on the training had an impact on the outcome measures, but the median split does suggest that there may be individual differences that contribute to training outcomes and that could account for the differences in outcome for similar training packages.

One obvious criticism of studies falling into this category is that it is not clear what instructional method is the basis of any of these programmes, apart from the restoration of function by practice. Different outcomes are therefore likely if the only similarity between the programmes is that they are provided on a computer. What they do have in common is the intrinsic motivation provided by the computer presentation as well as clear and fast feedback on past behaviour. They also give the learner the experience of control over the learning situation. Most educational software was designed with these and other principles in mind but currently this sort of software is rarely used in computerised training studies. Medalia et al. (2000a) also point out that students like computers because they have the opportunity to take part in a socially valued activity and this too will improve self-esteem, which may aid learning. The main problem with this type of training is the lack of theoretically driven software that improves the learning environment and can be individually tailored so that errorful learning, which seems to be a problem for people with schizophrenia, is reduced. What is clear from these results is that continued practice on one task has no direct impact on other tasks that use the same cognitive operations in the same domain. The chances of this sort of training (unless it is aimed at strategic problem solving) having any generalisation to alternative tasks are therefore very low.

Individual Executive Functioning Training

A further type of programme was derived from a theoretical analysis of the deficits of people with schizophrenia in a similar way to the training tasks devised by Van der Gaag (Van der Gaag, 1992; Van der Gaag et al., 2001). The programme consists of three separate modules: cognitive flexibility, memory, and planning, although some of the tasks are similar across modules (Delahunty & Morice, 1993). The programme was implemented on an individual basis with the main instructional technique being scaffolding but other techniques were also included, such as practice on tasks, instruction on mnemonic techniques, and encouragement of self-monitoring of errors. In addition, an errorless approach was adopted as much as possible. The tasks mostly use paper and pencils and are very easy at the outset but are graded so that they introduce complexity very slowly and progress is set at the learner's own pace. Reinforcement is given throughout in the form of praise or the instructor drawing the learner's attention to task improvements and individual efficacy. The tasks in the cognitive flexibility module provide patients with practice in engagement, disengagement, and re-engagement activities for a particular cognitive set. For instance, they are given a page with a set of numbers on it and are asked to cross off the odd or even numbers. This requires them to maintain a set but also to shift set when requested. The Working Memory module requires the person to maintain two sets of information simultaneously and to carry out transformations on a held information set. There is an emphasis here on categorising and chunking information as well as some self-instructional training in the use of mnemonic strategies. The Planning module consists of tasks in which the participant has to plan a sequence of moves to acquire a goal. The emphasis in this module is to organise information and to create and use sub-goals.

Several tests of this programme have shown positive results. Using only the cognitive flexibility module, Delahunty et al. (1993) found that there were improvements in WCST performance post-treatment that were maintained at six-month follow-up. There were also improvements in social functioning and symptom measures. A further trial of the whole programme from the same laboratory also showed modest improve-

ments on a range of test scores, with many people achieving normal performance following treatment (Delahunty & Morice, personal communication). A randomised control trial of the programme was recently completed at an independent laboratory in which the cognitive remediation programme was tested against a condition where patients received an attention control therapy with high face validity. The patients entering the trial were highly symptomatic but also had social and cognitive disability. The results were very encouraging. There were differential improvements in test scores in favour of cognitive remediation and for tests within the domains of memory and cognitive flexibility there were also generalised improvements, with memory improvements being maintained to follow-up. When the improvements reached a threshold (over 50% of tests improved) there were concomitant improvements in social functioning (Wykes et al., 1999a). There was also some evidence of improvements in some symptoms (auditory hallucinations) and in self-esteem. This last variable may be a key one to the issue of generalisation of treatment to functioning.

The studies cited here do look promising, but there is currently only one RCT. This particular trial, however, has highlighted a difficult issue for CRT — what are the appropriate outcomes. If the model of the relationship between social functioning and cognition is correct then CRT will not have a direct effect on social functioning. Rather, change in social functioning can only be expected when there is change in cognition. There is as yet no specification on how much change in cognition or a change to which cognitive process is required to get a measurable change in social functioning. Because of the variability in outcome of CRT trials and the rather blunt instruments of measuring social functioning change, it is surprising that any effects can be assessed.

Below we address other issues that have shaped current CRT, specifically which sorts of targets are being addressed and the expectations of durability of training.

WHAT SHOULD WE TARGET?

Predictive Variables?

Clearly the most appropriate target for intervention is one that is likely to have an impact on the quality of life of an individual. Improving performance on the Wisconsin Card Sort Test may be an aim for academics, but it is certainly not one shared by mental health service users. The initial section of this review suggested a number of different possible relationships between cognitive functions and outcome variables. For example, there are those that are linked to symptoms and/or social functioning and/or use of services cross-sectionally and there are a few studies with limited assessments of cognitive markers that suggest predictive relationships. It is of course not clear that the mechanism underlying these relationships is causal. But in the current state of knowledge, the predictive cognitive measures would seem to be the more appropriate targets.

Recovery-Associated Variables?

An alternative method for the identification of targets is not to look for the ways in which cognitive difficulties predict failures in rehabilitation but to carry out a cognitive analysis of the sorts of processing abilities that are necessary for recovery. This type of analysis may of course lead to the same targets but could lead to a

hierarchy of targets that might be more or less essential in recovery. This approach has also been suggested by Bellack and colleagues (1999).

Individual Variation?

Individuals vary in their cognitive profile and it may be that CRT should be tailored to this variability. Because laboratory studies focused on specific task performance, entry criteria to these studies was generally narrowly defined as difficulty on the target outcome measure. These studies therefore provide little information about how other cognitive abilities impact on the target function. Currently most tested clinical programmes are relatively prescriptive with little variation taken into account. This is particularly problematic for programmes run in groups where there might be wide variation in performance and so there is little scope for accommodation. The CRT programme devised by Delahunty et al. (2001) now describes some adaptations for individual cognitive difficulties in order to be able to clinically engage the patient. However, the individual tailoring approach has most successfully been adopted not by CRT programmes but in a programme that was devised to provide environmental support, Cognitive Adaptation Training (Velligan et al., 2000). In this programme the type of environmental support depends on the specific assessment profile of the individual. These assessments cover both the level of executive impairment and levels of apathy or disinhibition as well as impairments in activities of daily living. In addition, there is an environmental assessment in the individual's own home.

As well as variation across a number of different measures, participants may also vary in the level of the deficit and this may interact with training effects. The result found by Stratta et al. (1994) — that patients who can achieve at least 3 or 4 categories on the WCST achieve the best outcome in their study — suggests that there may be little benefit for those who have the most difficulties. Although a further analysis of data (Bell, personal communication) collected in the programme described by Bell et al. (2001) found that after dividing the group (N = 96) on the basis of initial working memory impairment, there was an effect of treatment condition but no interaction with impairment. Graphs of the data show that there is an equivalent positive effect of cognitive training regardless of severity of initial impairment. Thus, their training was effective for all patients. The training methods adopted in these conflicting studies appear to be similar, but the outcome variables are different, suggesting an interaction between training and individual variation.

Green et al. (2000) suggest that each individual has an associated learning potential that may mediate the ability to acquire new behaviours. This learning potential is a dynamic measure and estimates not memory, but whether the subject can learn new material. Three groups of patients can be distinguished: high performers that are not affected and do not need training, learners that start with a low performance and improve with training, and non-learners that have low performance standards and that do not benefit from training at all (Wiedl, 1999; Wiedl & Wienobst, 1999). It would therefore follow that individuals with low levels of performance would be excluded from CRT. We suggest that it is too early to come to such a pessimistic conclusion and that more information is needed about the interaction between cognitive style and therapy. For instance, some people may find it easier to learn with verbal supports whereas others are helped much more by visual imagery. These distinctions exist in the normal pedagogic literature and should be

referred to much more. It is true that the learning potential in social skills treatments in people who have memory problems is reduced, but it is not non-existent and may be overcome by changing the learning environment.

Capacity or Strategy?

After specifying the cognitive process there are two possible choices of mechanisms for improving performance. One aim could be to increase the capacity of basic mechanisms, such as verbal memory for digits or improvements in the speed of processing. This is possible as attested by the number of do-it-yourself manuals on most railway stations. However, psychologists such as Thorndike and Woodworth reported in 1901 (Thorndike & Woodworth, 1901) that learning to expand the capacity of verbal memory by practice does not necessarily generalise to other capacities or to improvements in the recall of other material. However, it might be an important factor in vocational rehabilitation, where fast performance on a task component is specifically required. Component-specific rehabilitation may then be designed using the principles of errorless learning, scaffolding etc., which were shown to be useful in other CRT studies.

In our view CRT probably should not usually be designed to target basic cognitive functions because of the uncertainty in whether the skills learnt can be generalised. The focus would therefore be on the higher level organisation of perception, apprehension, and problem solving which may be more easily generalised. In particular, cognitive skills that seem to be directly related to social outcome (see above) should be targeted.

TRAINING OR COMPENSATION?

Several authors have argued about whether CRT is an issue of retraining or compensation (e.g. Bellack, Gold, & Buchanan, 1999). This is even more confused with the use of terms such as ''neurocognitive remediation'' which seem to suggest that the effect of CRT is directly on brain systems rather than on the information processing strategies or cognitive capacities. The evidence for increased efficiency of higher order cognitive functions seems to come from strategy training and not from studies adopting endless rehearsal. It is a moot point whether this is a reinstatement of normal processes (i.e. training or retraining) or a compensation via different systems. It is likely that this is a false dichotomy and that different cognitive systems need different approaches for improvement to occur. As it is not yet clear (and cannot be without some attempt at intervention) whether some cognitive deficits are immutable, we suggest that the most optimistic approach, strategy training for most cognitive operations, should be the assumed mechanism until we are proved wrong. However, a few cognitive processes by definition do not seem open to cognitive strategy training (e.g. fast pre-attentive processes). In these cases compensation may be appropriate. An example is the emotion perception training that uses an analytic strategy instead of relying on fast Gestalt perception (van der Gaag, 1992).

Compensation can also take place within the CRT programme itself. For example people with schizophrenia have difficulty distinguishing in memory between correct performance and mistakes even after feedback. Errorless learning approaches compensate for this difficulty in order to improve learning on a specific task (Kern et al.,

1996; O'Carroll et al., 1999, Kern et al., 2000). The learning process is restricted to reinforcement learning in an extremely shaped skills training. In this way the learning-by-error process that is problematic can be omitted.

In conclusion, it is too early to distinguish between those cognitive difficulties that are reliant on the development of compensation skills and those that are open to some retraining. At the end of this review we will list some of the specific empirical support that needs to be generated to differentiate between them.

WHAT OUTCOMES SHOULD WE MEASURE?

Cognitive Outcomes

CRT success has generally focused on statistically significant changes in performance on a single neuropsychological task. This may seem relatively uncontroversial as the task is presumed to encompass cognitive processes that are being trained in CRT, but this similarity is only assumed because of face validity. There are few studies of normal participants receiving CRT that have concentrated on whether training skills are generalised to task performance, which would provide some empirical support for these assumptions. It is also possible, as suggested by Suslow et al. (2001), that these neuropsychological outcomes are not sensitive enough to pick up changes in specific cognitive systems following CRT. Outcome on a single task is also problematic as it does not even show whether generalisation to other tasks that require the same putative cognitive operation can take place. One simple solution is to add further tasks. This has been adopted by several authors (Spaulding et al., 1998, Wykes et al., 1999a), but it increases the number of comparisons made, and unless the analyses are merely exploratory this should be taken into account when assessing the significance of the results.

A more sophisticated approach would be to measure outcome using cognitive factors that could be derived from a wide range of tasks. These might more fairly represent the underlying cognitive difficulties and could act as the main cognitive outcomes. This method has been adopted in more recent studies by Bell et al. (2001) and Reeder et al. (2000). Z scores could be used within a study so a single task improvement can be compared across studies with some idea of the relative comparability of each of the single outcome measures. Alternatively, and the most optimistic outcome, normal functioning as defined by standard test manuals could be used as a threshold outcome. Bell and colleagues adopted this method and found that the number of patients with normal working memory performance at post-treatment rose significantly with CRT from 45% to 77% compared to a drop from 56% to 45% for those in the control condition.

Functioning Outcomes

The main outcome measure for any thinking skills therapy should be the thinking skills themselves. We have suggested, however, that these targets are chosen on the basis that they would be related to other factors that are valued by health services and individuals, e.g. social functioning and symptoms. These factors are also likely to improve given successful CRT, so their measurement is also important if only as secondary outcome measures. However, both factors are usually measured as global

scores, which may not be appropriate. Clearly if self-monitoring is the thinking skills target as it is related to the experience of hallucinations, then a specific hallucinations measure needs to be generated rather than using the total BPRS score. There is some evidence (Medalia et al., 1998, 2000b; Wykes et al., 1999a) that CRT may have consequences for levels of symptoms. It is not so easy to generate these sorts of components for social functioning, but Spaulding and colleagues (1999) have attempted such a distinction and have tried to tease out how changes in cognitive functioning have an effect on these different components. Their analyses were again exploratory, but these component analyses could actually be programmed into the power calculations of future studies. The evidence is not overwhelming that change in cognition has implications for future functioning, and this evidence would be required to verify its causal relationship but there are a number of other issues that may interfere with the current assessments of this link.

Cognitive processes and functioning measures may be linked in a number of different ways. For example the links could be indirect in that cognitive improvements may be necessary but may interact with other conditions such as the opportunity for further rehabilitation. The model to be tested in the analyses of post-treatment data is therefore not the direct effect of CRT on functioning but the effect of successful CRT on functioning. This was shown in the Wykes et al. (1999a) studies in which there were no direct effects of CRT on social functioning, but cognitive improvements (which were mainly due to CRT) produced reductions in the number of social functioning problems. In fact, when there are direct effects of therapy on functioning as there were on BPRS scores in Medalia et al. (1998) this result actually begs more questions than it answers. If not all participants receiving therapy improved then it is not clear that the functioning improvement was due to any of the purported experimental treatment effects (i.e. improvements in attention). In fact, it could be due to other factors such as increases in self-esteem through interaction with a computer, which Medalia discusses as an adjunct to the main treatment. These path models can be tested by covariance modelling, but the numbers of participants needed is much larger than the usual corpus of data.

Assessments of durability of therapy outcomes are severely limited by the length of follow-up. For instance improved cognitive function might not only improve current symptom status but may also affect later symptom occurrence (Nuechterlein & Subotnik, 1998). This, however, is a long-term outcome that is likely to be noticeable over years rather than the few months of the current studies. This is particularly problematic if what CRT is changing is vulnerability indicators, as the effects of these changes are unlikely to be noticed unless the information processing system is stressed in some way. Substantial and measurable increases in these irregular stressors in the environment make it unlikely that the effects would be obvious for a number of years.

Mediating Factors

One study (Wykes et al., 1999a,b) found that there was a statistically significant increase in self-esteem at post-therapy assessment in the CRT group compared to the control therapy condition. It is possible that the changes in self-esteem, which may be an integral part of the errorless learning types of CRT, have the effect of improving cognitive performance through increasing time on task and motivational mechanisms rather than improving strategic processing. At follow-up assessment in the same

study, however, there were no self-esteem differences between the groups (i.e. the CRT group had returned to baseline) but the differential improvement in memory performance remained (Wykes, 2000). This result does not invalidate the argument of an effect of self-esteem; rather it suggests that CRT must also have an independent effect that is durable.

In summary, if we make our models of the relationship between CRT and outcome explicit then we can make them testable. Information about what is a clinically significant change (i.e. what cognitive change is likely to have an impact on functioning) is absent, although several ongoing studies (e.g. Bell et al. 2001) may provide some of these essential data.

CONCLUSION AND SPECULATIONS

What is Cognitive Remediation?

As clearly seen from this review it is a number of quite disparate things varying along dimensions such as group versus individual, type of target, and length of treatment (one day to 40 daily sessions). If CRT is to be rigorously tested and compared across laboratories, then it needs to begin to be a little more homogenous. If it is not, difficulties will arise as very dissimilar studies are evaluated in the same meta-analysis. For instance a recent meta-analysis (Hayes & McGrath, 2000) found only three studies fitted their stringent criteria. One of these was a laboratory study carried out within one day (Tompkins et al., 1995). One was a computerised CRT programme that was carried out over six weeks (Medalia et al., 1998) and the third was a clinical CRT programme carried out over approximately 12 weeks (Wykes et al., 1999a). Even if there were some significant results from the meta-analysis (which there weren't) it would be extremely hard to interpret them.

Quality of Studies

Although not specifically addressed in detail in this review, it has been commented here and elsewhere that the methodological rigour of many of the CRT studies was poor. Hayes and McGrath (2000) suggest that this is improving following the Wykes et al. (1999) study, and in our view this has continued with others not available to those reviewers e.g. Bell et al. (2001). The method of randomisation is now made clear, although blind assessment (apart from Spaulding et al., 1998) is rare. Only a small number of participants have been included in studies of CRT to date, and as the effects on outcome measures are likely to be modest, these small sample sizes may not allow such effects to be measured. The main aim of early studies was to show that cognitive functioning was not immutable but could be improved in a variety of ways. The studies did increase therapeutic optimism, but modern studies must try to establish clear cognitive and clinical benefits for CRT. This requires good quality RCTs with a design that enables a distinction to be drawn between the specific effects of CRT and treatment-as-usual as well as the additive effects of this type of therapy on other rehabilitation services, which has become a recent preference (e.g. Bell et al., 2001). However, there is still the need for studies that will test both the theoretical models underlying CRT and will increase our understanding of the relationship between therapeutic effects and functioning measures.

Principles for Successful CRT

The principles, which were established from a review of studies, seem to suggest that an individual therapy based on three principles (errorless learning, targeting strategic processing, and scaffolded therapy) is likely to be successful. Satisfaction by most participants seems to be high using these techniques, although this evidence is anecdotal. Specific techniques such as verbalising the instructions are just one of a number that may be adopted during therapy to reduce information overload and increase attention to task-relevant strategies. There may be more techniques that are helpful but have not appeared from the studies reported here. It is not clear whether these principles can be embodied in a computer programme or whether they need to be supplied by a therapist, but in our opinion it will be difficult to provide an errorless, individual tailored therapy without a therapist being in close contact at least in the early stages of therapy. The more successful therapies (Medalia et al., 1998; Spaulding et al., 1998; Wykes et al., 1999a,b; Bell et al., 2001) are not short. Participants receive at least 15 hours of therapy or more on a variety of tasks. It may be that longer treatments as well as massed practice are essential.

Maintenance of Treatment Effects

There are so little data available on the durability of learning that it is not possible to make any claims or otherwise about durability. A few studies have suggested some durability of some effects (e.g. memory in Wykes et al., 1999b). However, schizophrenia is a chronic and relapsing condition and it may be that any improvements in thinking skills may disappear without any further maintenance. This maintenance could take the form of further rehabilitation, which allows generalisation of the treatment effects to take place. This has not been specifically built into the current programmes, and it is likely that clinically significant durable effects (if they exist) will be elusive until this is possible.

Is It Half Empty or Half Full?

Even setting this question may, for some people, be stepping outside current evidence. Recent reviews have concluded that the case for CRT is not proven (Wykes, 2000; Suslow et al., 2001; Hayes & McGrath, 2000). The initial CRT studies were designed to determine whether it was possible for cognitive performance to be enhanced. There has been a development of CRT investigations from studies attempting to improve performance on a specific task through training on that task to independent outcome measures of the same cognitive process, and now an increase in the variety of outcome measures to include symptoms and functioning. Unfortunately the achievements of CRT have been evaluated by comparing individual studies on the basis of a similarity of outcome measures with little regard for vital individual differences in method of CRT. We hope that our topology (see Table 4) will allow future reviewers to make these less-than-subtle distinctions.

   The recent studies are growing larger with better designs and more consistent use of specified programmes. Our concern is that because of the lack of clarity about CRT it will lose its credibility with research funding organisations before good evaluative evidence can be produced. The necessary standards for future RCTs are targeting more on attention, memory, and executive functioning with compensatory and other

TABLE 4. Descriptive Components of CRT Programmes

Type of intervention	Shaping behaviour through reinforcement
	Changing the environment (e.g. CAT)
	Practice alone
	Didactic teaching
	Scaffolding
Target of intervention	Specific task performance (e.g. vocational rehab skill)
	Specific cognitive strategy (e.g. use of planning)
Type of outcome	Neuropsychological test performance
	Cognitive factor (derived from test performance)
	Social functioning (or components of functioning)
	Symptom level (or specific symptoms)
Medium of training	Group versus individual treatment
	Computer versus paper and pencil

strategies; training extensively; selecting learners; and correcting for (persistent) negative, disorganisation, and depressive symptoms.

REFERENCES

Adams, H. E., Malatesta, V., Brantley, P. J., & Turkat, I. D. (1981). Modification of cognitive processes: a case study of schizophrenia. *Journal of Consulting Clinical Psychology*, *49*, 460–464.

Addington, J., & Addington, D. (1993). Premorbid functioning, cognitive functioning, symptoms and outcome in schizophrenia. *Journal of Psychiatry Neurosci*, *18* (1), 18–23.

Addington, J., Addington, D., & Maticka-Tyndale, E. (1991). Cognitive functioning and positive and negative symptoms in schizophrenia. *Schizophrenia Research*, *5* (2), 123–134.

Albus, M., Hubmann, W., Ehrenberg, C., Forcht, U., Mohr, F., Sobizack, N., Wahlheim, C., & Hecht, S. (1996). Neuropsychological impairment in first-episode and chronic schizophrenic patients. *European Archive of Psychiatry and Clinical Neuroscience*, *246*, 249–255.

Aleman, A., Hijman, R., de Haan, E. H., & Kahn, R. S. (1999). Memory impairment in schizophrenia: a meta-analysis. *American Journal of Psychiatry*, *156* (9), 1358–1366.

Baxter, L. R., Jr., Schwartz, J. M., Bergman, K. S., Szuba, M. P., Guze, B. H., Mazziotta, J. C., Alazraki, A., Selin, C. E., Ferng, H. K., Munford, P., et al. (1992). Caudate glucose metabolic rate changes with both drug and behaviour thearapy for obsessive-compulsive disorder. *Archives of General Psychiatry*, *49*, 681–689.

Baxter, R. D., & Liddle, P. F. (1998). Neuropsychological deficits associated with schizophrenic syndromes. *Schizophrenia Research*, *30*, 239–249.

Bell, M., Bryson, G., Tamasine, G., Corcoran, C., & Wexler, B. (2001). Neurocognitive enhancement therapy with work therapy: effects on neuropsychological test performance. *Archives of General Psychiatry* (in press).

Bellack, A. S., Gold, J. M., & Buchanan, R. W. (1999). Cognitive rehabilitation for schizophrenia: problems, prospects, and strategies. *Schizophrenia Bulletin*, *25*, 257–274.

Benedict, R., & Harris, A. (1989). Remediation of attention deficits in chronic schizophrenia patients: a preliminary study. *British Journal of Clinical Psychology*, *28*, 187–188.

Benedict, R., Harris, A., Markow, T., McCormick, J., Nuechterlein, K., & Asarnow, R. (1994). Effects of attention training on information processing in schizophrenia. *Schizophrenia Bulletin*, *20*, 537–546.

Bilder, R. M., Goldman, R. S., Robinson, D., Reiter, G., Bell, L., Bates, J. A., Pappadopulos, E., Willson, D. F., Alvir, J. M., Woerner, M. G., Geisler, S., Kane, J. M., & Lieberman, J. A. (2000). Neuropsychology of first-episode schizophrenia: initial characterization and clinical correlates. *American Journal of Psychiatry*, *157* (4), 549–559.

Brebion, G., Amador, X., Smith, M., Malaspina, D., Sharif, Z., & Gorman, J. M. (2000). Depression, psychomotor retardation, negative symptoms, and memory in schizophrenia. *Neuropsychiatry Neuropsychology Behavior Neurology*, *13* (3), 177–183.

Brenner, H. D., Stramke, W. G., Mewes, J., Liese, F., & Seeger, G. (1980). Erfarungen mit einem spezi-fischen therapieprogramm zum training kognitiver und kommunikativer fähigkeiten in der rehabilita-tion chronisch schizophrener patienten (A treatment program, based on training of cognitive and communicative functions, in the rehabilitation of chronic schizophrenic patients). *Nervenarzt*, *51*, 106–112.

Brenner, H. D., Roder, V., Hodel, B., Kienzle, N., Reed, D., & Liberman, R. P. (1994). *Integrated psycho-logical therapy for schizophrenic patients (IPT)*. Goettingen, Germany: Hogrefe & Huber Publishers.

Cannon, M., Jones, P., Huttunen, M., Tanskanen, A., Huttunen, T., Rabe-Hesketh, S., & Murray, R. (1999). School performance in Finnish children and later development of schizophrenia. *Archives of General Psychiatry*, *56*, 457–463.

Censits, D. M., Ragland, J. D., Gur, R. C., & Gur, R. E. (1997). Neuropsychological evidence support-ing a neurodevelopmental model of schizophrenia: a longitudinal study. *Schizophrenia Research*, *24*, 289–298.

Cuesta, M. J., & Peralta, V. (1995). Cognitive disorders in the positive, negative, and disorganization syndromes of schizophrenia. *Psychiatry Research*, *58* (3), 227–235.

David, A. S., Malmberg, A., Brandt, L., Allebeck, P., & Lewis, G. (1997). IQ and risk for schizophrenia: a population-based cohort study. *Psychological Medicine*, *27* (6), 1311–1323.

Delahunty, A., Morice, R., & Frost, B. (1993). Specific cognitive flexibility rehabilitation in schizophrenia. *Psychological Medicine*, *23*, 221–227.

Delahunty, A., & Morice, R. (1993). *A training programme for the remediation of cognitive deficits in schizo-phrenia*. Albury, NSW: Department of Health.

Delahunty, A., Reeder, C., Wykes, T., Morice, R., & Newton, E. (2001). *Cognitive remediation therapy manual* (2nd ed.). London: Kings College Institute of Psychiatry.

Erlenmeyer-Kimling, L., Rock, D., Roberts, S. A., Janal, M., Kestenbaum, C., Cornblatt, B., Adamo, U. H., & Gottesman, I. I. (2000). Attention, memory, and motor skills as childhood predictors of schizo-phrenia-related psychoses: the New York High-Risk Project. *American Journal of Psychiatry*, *157* (9), 1416–1422.

Everitt, B. (1998). Analysis of longitudinal data. Beyond MANOVA. *British Journal of Psychiatry*, *172*, 7–10.

Frith, C. (1992). *The cognitive neuropsychology of schizophrenia*. Hove, East Sussex: Lawrence Erlbaum and Associates.

Funke, B., Reinecker, H., & Commichau, A. (1989). The limits of cognitive training methods in long-term schizophrenic patients. *Nervenarzt*, *60* (12), 750–756.

Gold, S., Arndt, S., Nopoulos, P., O'Leary, D. S., & Andreasen, N. C. (1999). Longitudinal study of cognitive function in first-episode and recent-onset schizophrenia. *Am J Psychiatry*, *156* (9), 1342–1348.

Goldberg, T. E. (1999). Some fairly obvious distinctions between schizophrenia and bipolar disorder. *Schizophrenia Research,* *39* (2), 127–132; discussion 161-162.

Goldberg, T. E., & Weinberger, D. R. (1988). Probing prefrontal function in schizophrenia with neuro-psychological paradigms. *Schizophrenia Bulletin*, *14*, 179–183.

Goldberg, T. E., Weinberger, D. R., Berman, K. F., Pliskin, N. H., & Podd, M. H. (1987). Further evidence for dementia of the prefrontal type in schizophrenia? A controlled study of teaching the Wisconsin Card Sorting Test. *Archives of General Psychiatry*, *44*, 1008–1014.

Goldberg, T. E., Weinberger, D. R., Pliskin, N. H., Berman, K. F., & Podd, M. H. (1989). Recall memory deficit in schizophrenia. A possible manifestation of prefrontal dysfunction. *Schizophrenia Research*, *2* (3), 251–257.

Goldstein, G., Zubin, J., & Pogue-Geile, M. F. (1991). Hospitalization and the cognitive deficits of schizo-phrenia. The influences of age and education. *Journal of Nervous and Mental Diseases*, *179*, 202–206.

Gorman, J. M. (1997). Clinical correlates of memory in schizophrenia: differential links between depres-sion, positive and negative symptoms, and two types of memory impairment. *American Journal of Psy-chiatry*, *154* (11), 1538–1543.

Green, M. F. (1996). What are the functional consequences of neurocognitive deficits in schizophrenia? [see comments]. *American Journal of Psychiatry*, *153*, 321–330.

Green, M. F., Kern, R. S., Braff, D. L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the ''right stuff''? *Schizophrenia Bulletin*, *26*, 119–136.

Gruzelier, J., Wilson, L., & Richardson, A. (1999). Cognitive asymmetry patterns in schizophrenia: retest reliability and modification with recovery. *International Journal of Psychophysiology*, *34* (3), 323–331.

Haddock, G., McCarron, J., Tarrier, N., & Faragher, E. B. (1999). Scales to measure dimensions of hallu-cinations and delusions: the psychotic symptom rating scales (PSYRATS). *Psychological Medicine*, *29* (4), 879–889.

Hafner, H., Riecher-Rossler, A., Maurer, K., Fatkenheuer, B., & Loffler, W. (1992). First onset and early symptomatology of schizophrenia. A chapter of epidemiological and neurobiological research into age and sex differences. *European Archive of Psychiatry and Clinical Neuroscience*, *242*, 109–118.

Harvey, P. D., Lombardi, J., Kincaid, M. M., Parrella, M., White, L., Powchik, P., & Davidson, M. (1995). Cognitive functioning in chronically hospitalized schizophrenic patients: age-related changes and age disorientation as a predictor of impairment. *Schizophrenia Research*, *17* (1), 15–24.

Harvey, P., & Serper, M. (1999). The nature and management of cognitive dysfunction in patients with schizophrenia. *Directions in Psychiatry*, *19*, 21–35.

Hawkins, K. A., Hoffman, R. E., Quinlan, D. M., Rakfeldt, J., Docherty, N. M., & Sledge, W. H. (1997). Cognition, negative symptoms, and diagnosis: a comparison of schizophrenic, bipolar, and control samples. *Journal of Neuropsychiatry and Clinical Neuroscience*, *9* (1), 81–89.

Hayes, R. I., & McGrath, J. (2000). Cognitive rehabilitation for people with schizophrenia and related conditions. *Cochrane Review*, (November).

Heaton, R., Paulsen, J. S., McAdams, L. A., Kuck, J., Zisook, S., Braff, D., Harris, J., & Jeste, D. V. (1994). Neuropsychological deficits in schizophrenics. Relationship to age, chronicity, and dementia. *Archives of General Psychiatry*, *51*, 469–476.

Hegarty, J. D., Baldessarini, R. J., Tohen, M., Waternaux, C., & Oepen, G. (1994). One hundred years of schizophrenia: a meta-analysis of the outcome literature [see comments]. *American Journal of Psychiatry*, *151*, 1409–1416.

Heim, M., Wolf, S., Gothe, U., & Kretschmar, J. (1989). Kognitives training bei schizophrenen erkrankungen (Cognitive training in schizophrenic diseases). *Psychiatr Neurol Med Psychol (Leipzig)*, *41* (6), 367–375.

Hermanutz, M., & Gestrich, J. (1991). Computer-assisted attention training in schizophrenics. A comparative study. *European Archives of Psychiatry and Clinical Neuroscience*, *240*, 282–287.

Hoff, A. L., Riordan, H., O'Donnell, D. W., Morris, L., & DeLisi, L. E. (1992). Neuropsychological functioning of first-episode schizophreniform patients. *American Journal of Psychiatry*, *149*, 898–903.

Hoff, A. L., Sakuma, M., Wieneke, M., Horon, R., Kushner, M., & DeLisi, L. E. (1999). Longitudinal neuropsychological follow-up study of patients with first-episode schizophrenia. *American Journal of Psychiatry*, *156* (9), 1336–1341.

Hodel, B., & Brenner, H. D. (1994). Cognitive therapy with schizophrenic patients: conceptual basis, present state, future directions. *Acta Psychiatrica Scandinavica*, *90* (Suppl.), 108–115.

Hogarty, G. E., & Flesher, S. (1999). Developmental theory for a cognitive enhancement therapy of schizophrenia. *Schizophrenia Bulletin*, *25*, 677–692.

Holthausen, E. A., Wiersma, D., Knegtering, R. H., & Van den Bosch, R. J. (1999). Psychopathology and cognition in schizophrenia spectrum disorders: the role of depressive symptoms. *Schizophrenia Research*, *39*, 65–71.

Hyde, T. M., Nawroz, S., Goldberg, T. E., Bigelow, L. B., Strong, D., Ostrem, J. L., Weinberger, D. R., & Kleinman, J. E. (1994). Is there cognitive decline in schizophrenia? A cross-sectional study. *British Journal of Psychiatry*, *164*, 494–500.

Imamura, Y., Nakane, Y., Ohta, Y., & Kondo, H. (1999). Lifetime prevalence of schizophrenia among individuals prenatally exposed to atomic bomb radiation in Nagasaki City. *Acta Psychiatrica Scandinavica*, *100,* 344–349.

Jeste, D. V., Harris, M. J., Krull, A., Kuck, J., McAdams, L. A., & Heaton, R. (1995). Clinical and neuropsychological characteristics of patients with late-onset schizophrenia. *American Journal of Psychiatry*, *152*, 722–730.

Johns, L. C., & McGuire, P. K. (1999). Verbal self-monitoring and auditory hallucinations in schizophrenia. *Lancet*, *353*, 469–470.

Kelly, C., Sharkey, V., Morrison, G., Allardyce, J., & McCreadie, R. G. (2000). Nithsdale Schizophrenia Surveys. 20. Cognitive function in a catchment-area-based population of patients with schizophrenia. *British Journal of Psychiatry*, *177*, 348–353.

Kern, R. S., Wallace, C. J., Hellman, S. G., Womack, L. M., & Green, M. F. (1996). A training procedure for remediating WCST deficits in chronic psychotic patients: an adaptation of errorless learning principles [published erratum appears in *Journal of Psychiatric Research*, *31* (3), 1]. *Journal of Psychiatric Research*, *30*, 283–294.

Kern, R. S., Mitchell, S. S., Doran, D., Herrera, A. L., & Liberman, R. P. (2000).''Old wine in new bottles'': applications of errorless learning for vocational rehabilitation. In A. Medalia's (Chair), *Cognitive rehabilitation for people with schizophrenia.* Symposium conducted at the 34th annual meeting of the Association for the Advancement of Behavioral Therapy, New Orleans, LA, USA.

Koh, S. D., Kayton, L., & Peterson, R. A. (1976). Affective encoding and consequent remembering in schizophrenic young adults. *Journal of Abnormal Psychology*, *85*, 156–166.

Kraepelin, E. (1913). Achte Auflage (Ed.), *Psychiatrie: ein lehrbuch für studierende und ärtze*. Leipzig: Verlag von J.A. Barth.

Liddle, P. F. (1987). Schizophrenic syndromes, cognitive performance and neurological dysfunction. *Psychological Medicine*, *17* (1), 49–57.

Liu, S. K., Chen, W. J., Chang, C. J., & Lin, H. N. (2000). Effects of atypical neuroleptics on sustained attention deficits in schizophrenia: a trial of risperidone versus haloperidol. *Neuropsychopharmacology*, *22*, 311–319.

Mahurin, R. K., Velligan, D. I., & Miller, A. L. (1998). Executive-frontal lobe cognitive dysfunction in schizophrenia: a symptom subtype analysis. *Psychiatry Research*, *79* (2), 139–149.

McGrath, J. J., Pemberton, M. R., Welham, J. L., & Murray, R. M. (1994). Schizophrenia and the influenza epidemics of 1954, 1957 and 1959: a southern hemisphere study. *Schizophrenia Research*, *14*, 1–8.

Medalia, A., Dorn, H., & Watras-Gans, S. (2000a). Treating problem-solving deficits on an acute care psychiatric inpatient unit. *Psychiatry Research*, *97*, 79–88.

Medalia, A., Revheim, N., & Casey, M. (2000b). Remediation of memory disorders in schizophrenia. *Psychological Medicine*, *30*, 1451–1459.

Medalia, A., Aluma, M., Tryon, W., & Merriam, A. E. (1998). Effectiveness of attention training in schizophrenia. *Schizophrenia Bulletin*, *24*, 147–152.

Meichenbaum, D., & Cameron, R. (1973). Training schizophrenics to talk to themselves: a means of developing attentional controls. *Behavior Therapy*, *4*, 515–534.

Morice, R., & Delahunty, A. (1996). Frontal/executive impairments in schizophrenia. *Schizophrenia Bulletin*, *22* (1), 125–137.

Nathaniel-James, D. A., Brown, R., & Ron, M. A. (1996). Memory impairment in schizophrenia: its relationship to executive function. *Schizophrenia Research*, *21*, 85–96.

Nopoulos, P., Flashman, L., Flaum, M., Arndt, S., & Andreasen, N. (1994). Stability of cognitive functioning early in the course of schizophrenia. *Schizophrenia Research*, *14*, 29–37.

Norman, R. M., Malla, A. K., Cortese, L., Cheng, S., Diaz, K., McIntosh, E., McLean, T. S., Rickwood, A., & Voruganti, L. P. (1999). Symptoms and cognition as predictors of community functioning: a prospective analysis. *American Journal of Psychiatry*, *156* (3), 400–405.

Nuechterlein, K. H., Edell, W. S., Norris, M., & Dawson, M. E. (1986). Attentional vulnerability indicators, thought disorder, and negative symptoms. *Schizophrenia Bulletin*, *12* (3), 408–426.

O'Carroll, R. E., Russell, H. H., Lawrie, S. M., & Johnstone, E. C. (1999). Errorless learning and the cognitive rehabilitation of memory-impaired schizophrenic patients. *Psychological Medicine*, *29*, 105–112.

Olbrich, R., & Mussgay, L. (1990). Reduction of schizophrenic deficits by cognitive training: an evaluative study. *European Archives of Psychiatry and Clinical Neuroscience*, *239* (6), 366–369.

O'Leary, D. S., Flaum, M., Kesler, M. L., Flashman, L. A., Arndt, S., & Andreasen, N. C. (2000). Cognitive correlates of the negative, disorganized, and psychotic symptom dimensions of schizophrenia. *Journal of Neuropsychiatry and Clinical Neuroscience*, *12* (1), 4–15.

Passerieux, C., Segui, J., Besche, C., Chevalier, J. F., Widlocher, D., & Hardy-Bayle, M. C. (1997). Heterogeneity in cognitive functioning of schizophrenic patients evaluated by a lexical decision task. *Psychological Medicine*, *27* (6), 1295–1302.

Poulton, R., Caspi, A., Moffitt, T., Cannon, M., Murray, R., & Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder: a 15-year longitudinal study. *Archives of General Psychiatry*, *57*, 1053–1058.

Purcell, D. W., Lewine, R. R., Caudle, J., & Price, L. R. (1998). Sex differences in verbal IQ-performance IQ discrepancies among patients with schizophrenia and normal volunteers. *Journal of Abnormal Psychology*, *107* (1), 161–165.

Racenstein, J. M., Penn, D., Harrow, M., & Schleser, R. (1999). Thought disorder and psychosocial functioning in schizophrenia: the concurrent and predictive relationships. *Journal of Nervous and Mental Diseases*, *187* (5), 281–289.

Reeder, C., Wykes, T., & Newton, E. (2000). The use of executive factors in schizophrenia to monitor change following cognitive remediation. *Schizophrenia Research*, *41*, 221.

Riley, E. M., McGovern, D., Mockler, D., Doku, V. C., O'Ceallaigh, S., Fannon, D. G., Tennakoon, L., Santamaria, M., Soni, W., Morris, R. G., & Sharma, T. (2000). Neuropsychological functioning in first-episode psychosis—evidence of specific deficits. *Schizophrenia Research*, *43* (1), 47–55.

Roder, V., Studer, K., & Brenner, H. (1987). Erfahrungen mit einem integrierten psychologischen ther-

apieprogramm zum training kommunikativer fähigkeiten in der rehabilitation schwer chronisch schizophrener patienten (Experiences with an integrated psychological therapy program for training communication and cognitive abilities in the rehabilitation of severely ill chronic schizophrenic patients). *Schweizer Archiv fur Neurologie und Psychiatrie*, 138 (1), 31–44.

Rossi, A., Arduini, L., Daneluzzo, E., Bustini, M., Prosperini, P., & Stratta, P. (2000). Cognitive function in euthymic bipolar patients, stabilized schizophrenic patients, and healthy controls. *Journal of Psychiatric Research*, 34 (4/5), 333–339.

Rowe, E. W., & Shean, G. (1997). Card-sort performance and syndromes of schizophrenia. *Genetic, Social, and General Psychology Monographs*, 123 (2), 197–209.

Rund, B. R. (1998). A review of longitudinal studies of cognitive functions in schizophrenia patients. *Schizophrenia Bulletin*, 24 (3), 425–435.

Sanders, A. (1983). Towards a model of stress and human performance. *Acta Psychologica*, 53, 61–97.

Saxton, J., Munro, C. A., Butters, M. A., Schramke, C., & McNeil, M. A. (2000). Alcohol, dementia, and Alzheimer's disease: comparison of neuropsychological profiles. *Journal of Geriatric Psychiatry and Neurology*, 13 (3), 141–149.

Saykin, A. J., Shtasel, D. L., Gur, R. E., Kester, D. B., Mozley, L. H., Stafiniak, P., & Gur, R. C. (1994). Neuropsychological deficits in neuroleptic naive patients with first-episode schizophrenia. *Archives of General Psychiatry*, 51, 124–131.

Shallice, T. (1988). *From neuropsychology to mental structure*. Cambridge: Cambridge University Press.

Silverstein, S. M., Pierce, D. L., Saytes, M., Hems, L., Schenkel, L., & Streaker, N. (1998). Behavioral treatment of attentional dysfunction in chronic, treatment-refractory schizophrenia. *Psychiatric Quarterly*, 69 (2), 95–105.

Spaulding, W., Reed, D., Storzbach, D., Sullivan, M., Weiler, M., & Richardson, C. (1998). The effects of remediational approach to cognitive therapy for schizophrenia. In T. Wykes, N. Tarrier, & S. Lewis (Eds.), *Outcome and innovation in psychological treatment of schizophrenia*. Chichester, England: Wiley.

Spaulding, W. D., Fleming, S. K., Reed, D., Sullivan, M., Storzbach, D., & Lam, M. (1999). Cognitive functioning in schizophrenia: implications for psychiatric rehabilitation. *Schizophrenia Bulletin*, 25, 275–289.

Spindler, K. A., Sullivan, E. V., Menon, V., Lim, K. O., & Pfefferbaum, A. (1997). Deficits in multiple systems of working memory in schizophrenia. *Schizophrenia Research*, 27, 1–10.

Standards of Reporting Trials Group. (1994). A proposal for structured reporting of randomized control trials. *Journal of the American Medical Association*, 272, 1926–1931.

Steffy, R. A., & Galbraith, K. J. (1980). Relation between latency and redundancy-associated deficit in schizophrenic reaction time performance. *Journal of Abnormal Psychology*, 89, 419–427.

Stratta, P., Mancini, F., Mattei, P., Casacchia, M., & Rossi, A. (1994). Information processing strategy to remediate Wisconsin Card Sorting Test Performance in schizophrenia: a pilot study. *American Journal of Psychiatry*, 151, 915–918.

Suslow, T., Schonuer, K., & Arolt, V. (2001). Attention training in the cognitive rehabilitation of schizophrenia patients: a review of efficacy studies. *Acta Psychiatrica Scandinavica*, 103, 15–23.

Susser, E. S., & Lin, S. P. (1992). Schizophrenia after prenatal exposure to the Dutch Hunger Winter of 1944–1945 [see comments]. *Archives of General Psychiatry*, 49, 983–988.

Susser, E., Neugebauer, R., Hoek, H. W., Brown, A. S., Lin, S., Labovitz, D., & Gorman, J. M. (1996). Schizophrenia after prenatal famine. Further evidence [see comments]. *Archives of General Psychiatry*, 53, 25–31.

Thorndike, E. L., & Woodworth, R. R. (1901). The influence of improvement in one mental function upon the efficiency of other functions. *Psychological Review*, 8, 247–261.

Tompkins, L., Goldman, R., & Axelrod, B. (1995). Modifiability of neuropsychological dysfunction in schizophrenia. *Biological Psychiatry*, 38, 105–111.

Van der Does, A. J., Dingemans, P. M., Linszen, D. H., Nugter, M. A., & Scholte, W. F. (1996). Symptoms, cognitive and social functioning in recent-onset schizophrenia: a longitudinal study. *Schizophrenia Research*, 19 (1), 61–71.

Van der Gaag, M. (1992). *The results of cognitive training in schizophrenic patients*. Delft: Eburon.

Van der Gaag, M., Kern, R., van den Bosch, R. J., & Liberman, R. P. (2001). A controlled trial of cognitive remediation in schizophrenia. *Schizophrenia Bulletin*, (under review).

Van Os, J., & Verdoux, H. (2001). Diagnosis and classification: categories or dimensions, diseases of distributions? In M. Cannon, P. Jones, M. Murray, E. Susser, & J. Van Os (Eds.), *The epidemiology of schizophrenia*. Cambridge: Cambridge University Press (in press).

Velligan, D. I., Mahurin, R. K., True, J. E., Lefton, R. S., & Flores, C. V. (1996). Preliminary evaluation of

cognitive adaptation training to compensate for cognitive deficits in schizophrenia. *Psychiatric Services*, *47*, 415–417.

Velligan, D. I., Bow-Thomas, C. C., Huntzinger, C. D., Ritch, J., Ledbetter, N., Prihoda, T. J., & Miller, A. L. (2000). A randomized controlled trial of the use of compensatory strategies in schizophrenic outpatients: cognitive adaptation training. *American Journal of Psychiatry*, *157*, 1317–1323.

Verdoux, H., & Liraud, F. (2000). Neuropsychological function in subjects with psychotic and affective disorders. Relationship to diagnostic category and duration of illness. *European Psychiatry*, *15* (4), 236–243.

Wiedl, K.H. (1999). Rehab rounds: cognitive modifiability as a measure of readiness for rehabilitation. *Psychiatric Services, 50* (11), 1411–1413, 1419.

Wiedl, K. H., & Wienobst, J. (1999). Interindividual differences in cognitive remediation research with schizophrenic patients-indicators of rehabilitation potential? *International Journal of Rehabilitation Research*, *22* (1), 55–59.

Wong, A. H., Voruganti, L. N., Heslegrave, R. J., & Awad, A. G. (1997). Neurocognitive deficits and neurological signs in schizophrenia. *Schizophrenia Research*, *23* (2), 139–146.

Wykes, T. (1994). Predicting symptomatic and behavioural outcomes of community care. *British Journal of Psychiatry*, *165*, 486–492.

Wykes, T. (1998). What are we changing with neurocognitive rehabilitation? Illustrations from two single cases of changes in neuropsychological performance and brain systems as measured by SPECT. *Schizophrenia Research*, *34*, 77–86.

Wykes, T. (2000). Cognitive rehabilitation and remediation. In T. Sharma, & P. Harvey (Eds.), *Schizophrenia in cognition in schizophrenia: impairments, importance and treatment strategies*. Oxford: Oxford University Press.

Wykes, T., Sturt, E., & Katz, R. (1990). The prediction of rehabilitative success after three years. The use of social, symptom and cognitive variables. *British Journal of Psychiatry*, *157*, 865–870.

Wykes, T., Reeder, C., Corner, J., Williams, C., & Everitt, B. (1999a). The effects of neurocognitive remediation on executive processing in patients with schizophrenia. *Schizophrenia Bulletin*, *25*, 291–307.

Wykes, T., Reeder, C., Williams, C., & Corner, J. (1999b). Cognitive remediation—the durability of effects. *Schizophrenia Research*, *36*, 335.