PART I

THEORETICAL OVERVIEW

CHAPTER 1

CURRENT PERSPECTIVES ON RELAPSE, RELAPSE DETECTION AND PREVENTION

INTRODUCTION

In this chapter, we will briefly review the evidence for psychological therapies in the prevention of relapse. Following this brief review, we describe a psychological approach to conceptualising the early signs of relapse in psychosis. We will argue that relapse needs to be understood from (1) the context of the person, their beliefs and appraisals of relapse and psychosis; (2) the individual's interpersonal context; and (3) the manner in which service systems respond to the challenge of relapse prevention for individuals who are prone to recurrent psychotic experiences. Our general definition of relapse is as a medical term that is used to describe the recurrence of an episode of illness or the exacerbation of illness symptoms, which had been partially remitted. Importantly, however, in relation to psychosis, relapse refers predominantly to the return or exacerbation of positive psychotic experiences that are likely to be associated with high levels of emotional distress and impaired social, vocational and interpersonal functioning. The combination of psychotic experiences, emotional distress and impaired functioning may lead to the person being voluntarily or involuntarily admitted to hospital. It is important to recognise that there is the breakdown of educational, vocation and social resources that accompanies relapse. Furthermore, relapse brings with it greater threats to the family structure and functioning.

Relapse has been associated with the evolution of greater residual and pervasive psychotic experiences (Wiersma et al., 1998), greater social disability (Hogarty et al., 1991), increased risk of depression and suicidal thinking (Iqbal et al., 2000) and heightened, more intense levels of family distress (Barrowclough et al., 2001). In psychological terms, relapse is a potentially devastating and critical life event with profound consequences for the emotional and psychological well-being of the person and their family or loved ones. In addition, the effects of an individual's relapse on care staff and health professionals connected with their well-being should not be under-

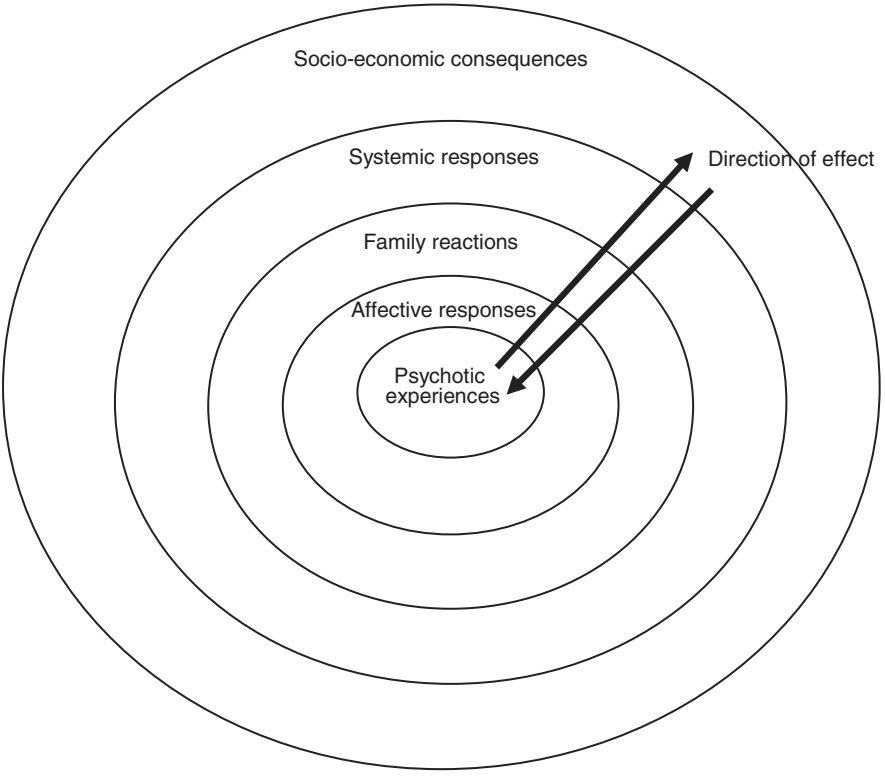

Figure 1.1 Personal, interpersonal and systemic context of relapse

estimated. Many staff may experience feelings of disappointment and self-criticism, or, in contrast, feelings of frustration and anger. Therefore, any attempt to develop a comprehensive formulation of relapse needs to fully integrate factors at the individual level (for example, personal meanings and appraisals, affective reactions and behavioural responses), the interpersonal level (for example, family attributions and their associated emotional and behavioural reactions), and the systemic level (that is, the responses of services, health professionals and care staff). The bidirectional relationship between the personal, interpersonal and systemic factors is graphically illustrated in Figure 1.1.

PSYCHOLOGICAL THERAPIES AND RELAPSE PREVENTION

Before describing our psychological approach to conceptualising the early signs of relapse, we will briefly review the current evidence for psychological treatments being effective in the prevention of relapse among individ-

uals diagnosed with schizophrenia. In particular, we focus on family therapy and cognitive behavioural therapy (CBT). We will argue that while there is robust evidence for family interventions being an effective approach to supporting families and individuals in staying well after psychosis, there is a need to develop individually based therapies that can support and enhance family-based work, or indeed stand alone as a psychological approach to the prevention of relapse.

Family Interventions

Pitschel-Walz and colleagues (2001) described a meta-analysis of 25 studies, which evaluated the effectiveness of family interventions for relapse among individuals diagnosed with schizophrenia. Previous meta-analyses (Mari & Streiner, 1994, 1996; Pharoah et al., 1999), containing up to 13 studies, had suggested that family interventions for schizophrenia were moderately effective in decreasing the frequency of relapse and re-hospitalisation. Pitschel-Walz and colleagues were able to include more studies unpublished at the time of these earlier reviews. In total, they identified 39 studies; four were excluded because of lack of control group, nine studies were not randomised studies, and in one study group comparisons were missing. Twelve studies compared family intervention with usual care (Goldstein et al., 1978; Leff et al., 1985; Spiegel & Wissler, 1987; Spencer et al., 1988; Tarrier et al., 1989; Kelly & Scott, 1990; Hogarty et al., 1991; Posner et al., 1992; Vaughan et al., 1992; Randolph et al., 1994; Xiong et al., 1994; Zhang et al., 1994). Pitschel-Walz and colleagues found that family interventions were better than routine care at 6, 9, 12, 18, and 24 months after therapy. Longer-term therapies (those lasting between 9 to 24 months) were more effective than shorter-term interventions (those lasting less than 3 months). Furthermore, in five studies that combined family intervention with individual intervention (Cranach, 1981; Kelly & Scott, 1990; Hogarty et al., 1991; Buchkremer et al., 1997; Pitschel-Walz et al., 1998) this combination of therapies performed better than routine care. However, adding individual therapy to family intervention did not result in significant improvements in relapse rates compared to family intervention alone. A further six studies compared family intervention with individual therapy (Ro-Trock et al., 1977; Falloon et al., 1982, 1985; Kelly & Scott, 1990; Hogarty et al., 1991; Telles et al., 1995; Hogarty et al., 1997a, 1997b). There were no significant differences in efficacy between the two interventions during the first year after study entry. However, two years after participants entered the studies, the family intervention performed better than individual therapy in the prevention of relapse. In addition, five studies (Clarkin et al., 1998; Colom et al., 2003; Miklowitz et al., 2000, 2003; Rae et al., 2003) have also shown that family-focused interventions in bipolar disorders are also effective in preventing relapse.

Cognitive Behavioural Therapy

The basic design, participants, outcomes and follow-up periods are summarised in Table 1.1. A total of 35 papers describing 23 randomised controlled trials involving the comparison of CBT with at least one other treatment condition, involving a total of 2,206 participants with a diagnosis of schizophrenia or similar was identified.

The studies identified for this review were heterogeneous in terms of the participant populations and outcome measures used. One study (Barrowclough et al., 2001; Haddock et al., 2003) focused on participants with co-morbid substance use problems. Four studies (Drury et al., 1996; Haddock et al., 1999; Lewis et al., 2002; Startup et al., 2004) delivered cognitive therapy during the acute/recovery phase. Eight studies (Lecompte, 1996; Garety et al., 1998; Pinto et al., 1999; Tarrier et al., 1999; Sensky et al., 2000; Turkington & Kingdon, 2000; Durham et al., 2003; Trower et al., 2004) focused on those with drug-resistant symptoms. Two studies (Daniels, 1998; Bechdolf et al., 2004) studied group cognitive therapy. Two studies (McGorry et al., 2002; Morrison et al., 2004) delivered CBT to individuals who were at ultra-high risk of developing psychosis. Two studies (Kemp & David, 1996; Turkington et al., 2002) focused on the development of insight as the primary outcome. Only four studies (Buchkremer et al., 1997; Hogarty et al., 1997; Bach & Hayes, 2002; Gumley et al., 2003) had relapse or readmission as their primary outcome.

Although a wide variety of outcome measures was used, there were three main primary outcome measures employed including the Brief Psychiatric Rating Scale (BPRS: Overall & Gorham, 1962), the Comprehensive Psychiatric Rating Scale (CPRS: Montgomery et al., 1978), and the Positive and Negative Syndrome Scale (PANSS: Kay et al., 1987). The BPRS was employed in Buchkremer et al. (1997), Daniels (1998), Garety et al. (1998), Haddock et al. (1999), McGorry et al. (2002), Pinto et al. (1999), Startup et al. (2004) and Tarrier et al. (1999). The CPRS was used in Sensky et al. (2000), Turkington and Kingdon (2000) and Turkington et al. (2002). The PANSS was used in Barrowclough et al. (2001), Bechdolf et al. (2004), Durham et al. (2003), Gumley et al. (2003), Lewis et al. (2002) and Morrison et al. (2004). Other studies used the Psychiatric Assessment Scale (PAS: Drury et al., 1996), the David Insight Scale (Kemp & David, 1996; Turkington et al., 2002), the Voice Compliance Scale and the Beliefs About Voices Questionnaire (Trower et al., 2004) as primary outcome measures. Over this time period, CBT studies seem to have been concerned with symptomatic improvement and therefore have been orientated to psychiatric ratings of outcome. CBT studies have been less concerned with emotional recovery, quality of life, social functioning and staying well. In this respect the Trower et al. (2004) study marks a shift towards more CBT-specific outcomes.

Table 1.1 Randomised controlled clinical trials of CBT for schizophrenia or similar[1]

Study	*Country*	*Design*	*Diagnostic criteria*	*Treatment conditions*	*Main outcomes*	*End of study results*	*Length of follow-up*	*Follow-up outcomes*	*Follow-up results*	*Comments*
Bach & Hayes, 2002	United States	RCT		ACT (1: n =) TAU (2: n =)	Relapse	1 > 2	NA	NA	NA	This was the first trial of Acceptance and Commitment Therapy (ACT) with individuals with psychosis
Barrowclough et al., 2001	United Kingdom	RCT	ICD-10 DSM-IV	CBT + MI + FI (1: n = 18) TAU (2: n = 18)	GAF[9] PANSS Relapse	1 > 2 1 = 2 1 > 2	18 months	Patients: GAF PANSS Carers: BDI[10] GHQ[11]	 1 > 2 1 = 2 1 = 2 1 = 2	Main outcomes reported for 9 and 12 months following randomisation MI = Motivational Interviewing FI = Family Intervention

Table 1.1 Continued

Study	*Country*	*Design*	*Diagnostic criteria*	*Treatment conditions*	*Main outcomes*	*End of study results*	*Length of follow-up*	*Follow-up outcomes*	*Follow-up results*	*Comments*
Bechdolf et al., 2004	Germany	RCT	ICD-10	GCBT (1: n = 40) GPE (2: n = 48)	PANSS[5] Compliance	1 > 2 1 = 2	6 months	Relapse PANSS[5] Compliance	1 > 2 1 > 2 1 = 2	Therapies were delivered with a 6-week treatment envelope with follow-up reported at 6-months post-treatment GCBT = Group CBT GPE = Group Psychoeducation
Buchkremer et al., 1997	Germany	RCT	DSM-III-R	PMT + L (1: n = 32) PMT + L + KC (2: n = 35) PMT + CP (3: n = 34) PMT + CP + C (4: n = 33) L (5: n = 57)	Readmission BPRS[2] SANS[3] GAS[4]	1,2,3,4 = 5 4 > 5 1,2,3,4 = 5 1,2,3,4, = 5 1,2,3,4 > 5	5 years	Readmission BPRS SANS GAS	1,2,3,4 = 5 4 > 5 1,2,3,4 = 5 1,2,3,4 = 5 1,2,3,4 = 5	Main outcomes for this study are reported at 2-years post-randomisation PMT = Psychoeducation L = Leisure group KC = Key-person Counselling CP = Cognitive Psychotherapy

Daniels, 1998	United States	RCT	DSM-IV	IBT (1: n = 10) WL (2: n = 10)	GAF CGI[20] QLS[21] BPRS	Group differences not reported.				CBT participants significantly improved on GAF, but not CGI, QLS, BPRS Group treatment was conducted over 16, twice-weekly sessions. Outcomes are at post-treatment IBT = Integrative Behaviour Therapy WL = Waiting List
Drury et al., 1996, 2000	United Kingdom	RCT	WHO	CBT (1: n = 20) ATY (2: n = 20)	12 weeks: PAS[17] Positive PAS Disorganisation PAS Negative 9 months: PAS Positive PAS Disorganisation PAS Negative	 1 > 2 1 = 2 1 = 2 1 > 2 1 = 2 1 = 2	5 years	Readmission PAS	1 = 2 1 = 2	ATY = Activity/ recreation

Table 1.1 Continued

Study	*Country*	*Design*	*Diagnostic criteria*	*Treatment conditions*	*Main outcomes*	*End of study results*	*Length of follow-up*	*Follow-up outcomes*	*Follow-up results*	*Comments*
Durham et al., 2003	United Kingdom	RCT	DSM-IV ICD-10	CBT (1: n = 22) SP (2: n = 23) TAU (3: n = 21)	PANSS PSYRATS[6] GAS	1 = 2 = 3 1 = 2 = 3 1 = 2 = 3	12 months	PANSS PSYRATS GAS	1 > 2,3 1,2 > 3 1 = 2 = 3	End of treatment results were taken at 9 months with 3-month follow-up. SP = Supportive Psychotherapy
Garety et al., 1998	United Kingdom	RCT	DSM-III-R	CBT (1: n = 28) TAU (2: n = 32)	BPRS	1 > 2	18 months	BPRS Delusional[7] distress Hallucinations[7] frequency	1 > 2 1 > 2 1 > 2	
Gumley et al., 2003	United Kingdom	RCT	DSM-IV	CBT (1: n = 72) TAU (2: n = 72)	Relapse Readmission PANSS SFS[8]	1 > 2 1 = 2 1 > 2 1 > 2				PANSS positive, negative and global psychopathology scales analysed separately
Haddock et al., 1999	United Kingdom	RCT	DSM-IV	CBT (1: n = 10) TAU (2: n = 11)	Days to discharge BPRS PSYRATS	1 = 2 1 = 2 1 = 2	24 months	Readmission	1 = 2	

Hogarty et al., 1997	United States	RCT	RDC	PT (1: n = 74) FT (2: n = 50) ST (3: n = 53)	Relapse (psychotic and affective)	Trial 1 1 > 2,3 Trial 2 1 < 3				Outcomes for patients living with (Trial 1: n = 97) and outwith family (Trial 2: n = 54) were analysed separately. Results are presented at 3 years following randomisation: PT = Personal Therapy FT = Family Therapy ST = Supportive Therapy
Kemp, 1998	United Kingdom	RCT	DSM-III-R	ComT (1: n = 39) TAU (2: n = 35)	Compliance Attitudes to treatment[12] Insight[13] GAF	1 > 2 1 > 2 1 > 2 1 > 2	18 months	Compliance Attitudes to treatment Insight GAF	1 = 2 1 > 2 1 > 2 1 > 2	ComT = Compliance Therapy
Lecompte, 1996	United States	RCT		CBT (1: n = 32) Con (2: n = 32)						Con = Non-directive Conversation

Table 1.1 Continued

Study	*Country*	*Design*	*Diagnostic criteria*	*Treatment conditions*	*Main outcomes*	*End of study results*	*Length of follow-up*	*Follow-up outcomes*	*Follow-up results*	*Comments*
Lewis et al., 2002	United Kingdom	RCT	DSM-IV	CBT (1: n = 101) SC (2: n = 106) RC (3: n = 102)	PANSS PSYRATS	1 > 2,3 1 = 2 = 3	18 months	PANSS PSYRATS Readmission	1,2 > 3 1,2 > 3 1 = 2 = 3	CBT was associated with faster improvement compared to SC and RC SC = Supportive Counselling RC = Routine Care
McGorry et al., 2002	Australia	RCT	N/A	CBT + Risp (1: n = 31) CM (2: n = 28)	Transition BPRS SANS	1 > 2 1 = 2 1 = 2	12 months	Transition BPRS SANS GAF	1 = 2 1 = 2 1 = 2 1 = 2	End of treatment outcomes reported at 6 months. Trial conducted with people at ultra-high risk of developing schizophrenia. CBT + Risp = CBT in combination with low-dose risperidone. CM = Case Management

Morrison et al., 2004	United Kingdom	RCT	N/A	CBT (1: n = 37) Monitoring (2: n = 23)	Transition PANSS	1 > 2 1 > 2				Outcomes reported at 12-months post randomisation Trial conducted with people at ultra-high risk of developing schizophrenia
Pinto et al., 1999	Italy	RCT	DSM-IV	CBT (1: n = 20) SP (1: n = 21)	BPRS SAPS[14] SANS	1 > 2 1 > 2 1 = 2				All participants in receipt of Clozapine. Outcomes reported at post-treatment, 6 months following randomisation
Sensky et al., 2000	United Kingdom	RCT	ICD-10	CBT (1: n = 46) BF (2: n = 44)	CPRS[15] MADRS[16] SANS	1 = 2 1 = 2 1 = 2	18 months	CPRS[15] MADRS[16] SANS	1 > 2 1 > 2 1 > 2	Post-treatment outcomes taken at 9 months BF = Befriending
Startup et al., 2004	United Kingdom	RCT	DSM-IV	CBT (1: n = 47) TAU (2: n = 43)	SAPS SANS BPRS SFS	1 > 2 1 = 2 1 > 2 1 > 2	12 months	SAPS SANS BPRS SFS	1 > 2 1 > 2 1 > 2 1 > 2	Post-treatment outcomes taken at 6 months

Table 1.1 Continued

Study	*Country*	*Design*	*Diagnostic criteria*	*Treatment conditions*	*Main outcomes*	*End of study results*	*Length of follow-up*	*Follow-up outcomes*	*Follow-up results*	*Comments*
Tarrier et al., 1999	United Kingdom	RCT	DSM-III-R	CBT (1: n = 33) SC (2: n = 26) RC (3: n = 28)	BPRS Readmission	1 > 2,3 1,2 < 3	12 months 24 months	BPRS Readmission BPRS Readmission	1 > 3 1 = 2 = 3 1,2 > 3 1 = 2 = 3	Follow-ups took place 12 and 24 months post-treatment. Treatment duration was 3 months.
Trower et al., 2004	United Kingdom	RCT	ICD-10	CBT (1: n = 18) TAU (2: N = 20)	Compliance[18] Malevolence[19] Omniscience[19] PSYRATS Control	1 > 2 1 = 2 1 > 2 1 > 2	12 months	Compliance Malevolence Omniscience Control	1 > 2 1 = 2 1 > 2 1 > 2	
Turkington & Kingdon, 2000	United Kingdom	RCT	ICD-10 DSM-IV	CBT (1: n = 13) BF (2: n = 6)	CPRS MADRS	1 > 2 1 = 2				Outcomes reported at 6 months
Turkington et al., 2002	United Kingdom	RCT	ICD-10	CBT (1: n = 257) TAU (2: n = 165)	CPRS Insight[13] MADRAS	1 > 2 1 > 2 1 > 2				Outcomes presented at 20 weeks

Notes:

[1] Based on the search strategy conducted by Cochrane Schizophrenia group (2004) supplemented by further search strategy combining SCHIZOPHRENIA with COGNITIV* and/or BEHAVIO* and/or THERAP* in CINAHL, EMBASE, PsychINFO, MEDLINE databases.

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CBT was compared with Treatment as Usual (TAU) in 11 trials (Garety et al., 1998; Kemp et al., 1998; Haddock et al., 1999; Tarrier et al., 1999; Barrowclough et al., 2001; Lewis et al., 2002; Durham et al., 2003; Gumley et al., 2003; Morrison et al., 2004; Startup et al., 2004; Trower et al., 2004). A total of 12 trials incorporated a comparison psychological intervention into the study design (Drury et al., 1996; Lecompte, 1996; Buchkremer et al., 1997; Hogarty et al., 1997; Pinto et al., 1999; Tarrier et al., 1999; Sensky et al., 2000; Turkington et al., 2000; Lewis et al., 2002; McGorry et al., 2002; Durham et al., 2003; Bechdolf et al., 2004). One trial (Daniels, 1998) compared CBT to a waiting list control.

In terms of overall psychiatric symptomatology, CBT shows encouraging evidence of being more effective in comparison to TAU (Garety et al., 1998; Tarrier et al., 1999; Barrowclough et al., 2001; Turkington et al., 2002; Gumley et al., 2003; Bechdolf et al., 2004; Morrison et al., 2004; Startup et al., 2004; Trower et al., 2004) or another psychological intervention (Drury et al., 1996; Pinto et al., 1999; Tarrier et al., 1999; Turkington & Kingdon, 2000) post-treatment. However, the results comparing CBT to other psychological interventions are less clear. CBT was also associated with a reduction in relapse in comparison to treatment as usual in one study (Gumley et al., 2003), in comparison to group psychoeducation (Bechdolf et al., 2004) and in comparison to the family therapy (Hogarty et al., 1997). On the other hand, Tarrier et al. (1999) did not find a reduction in relapse, and indeed for patients who are living alone, Hogarty et al. (1997) found an increase in relapse rate in comparison to Supportive Therapy. Two studies (Drury et al., 1996; Lewis et al., 2002) found that CBT was associated with a faster time to remission, or a more complete remission (Drury et al., 1996). However, Lewis et al. (2002) found that the advantage for CBT (compared to TAU) at four weeks was lost at six weeks; in other words, patients who receive CBT during their acute phase achieve remission two weeks before patients treated in routine care or with supportive therapy. Kemp and David (1998) and Turkington et al. (2002) found that CBT improved attitudes to drug treatment and increased acceptance of illness. McGorry et al. (2002) and Morrison et al. (2004) found that CBT was associated with a lower rate of transition to psychotic disorder, including schizophrenia.

Of the 22 trials, 11 have reported follow-ups beyond 12-months post-randomisation (Drury et al., 1996; Buchkremer et al., 1997; Hogarty et al., 1997; Garety et al., 1998; Kemp & David, 1998; Haddock et al., 1999; Tarrier et al., 1999; Sensky et al., 2000; Barrowclough et al., 2001; Lewis et al., 2002; Startup et al., 2004). These follow-ups were conducted at 18 months (Garety et al., 1998; Kemp et al., 1998; Sensky et al., 2000; Barrowclough et al., 2001; Lewis et al., 2002; Startup et al., 2004), 24 months (Haddock et al., 1999; Tarrier et al., 1999), three years (Hogarty et al., 1997) and five years (Drury et al., 1996; Buchkremer et al., 1997). Therefore, in terms of quantity of studies

completed, the best evidence for the maintenance of treatment gains post-treatment comes from the six studies that have conducted 18-month post-randomisation assessments. In the Barrowclough et al. (2001), Garety et al. (1998), Kemp & David (1998) and Startup et al. (2004) studies, immediate treatment gains were largely maintained at follow-up. All these studies compared CBT to TAU. The Sensky et al. (2000) study did not find a difference between CBT and befriending (BF) at post-treatment. However, at 18 months those who received CBT had continued to improve, whereas those who received BF had lost much of their gains. The Lewis et al. (2002) study (reported as Tarrier et al., 2004) did not find a specific effect for CBT at 18 months, rather, receipt of psychological intervention appeared to improve outcome although this positive finding did not translate to relapse or readmission outcomes. To date, there is no evidence for a specific effect for CBT at two years (Tarrier et al., 1999). The Hogarty et al. (1997) study did find an effect for Personal Therapy for those patients who lived with families at three years; however, treatment had been continuous over that period. Finally, there was little evidence for the efficacy of CBT at five-year follow-up (Buchkremer et al., 1997; Drury et al., 2000). However, the Drury et al. (2000) study found that those who relapsed more than once during the intervening period had a very poor outcome, raising the importance of relapse prevention for this group. There was a little evidence from the Buchkremer et al., (1997) study that receipt of CBT in combination with psychoeducation plus counselling protected participants against relapse compared to leisure activity control over the intervening five years.

In their Cochrane review, Jones et al., (2004) concluded:

> the use of cognitive behavioural therapy has been associated with some reduction in symptoms, especially the positive symptoms of schizophrenia. However, there is considerable variability in the findings of the various studies and, at present, it is not possible to assert any substantial benefit for cognitive behavioural therapy over standard care or supportive therapies.

We would argue that some of this variability in findings is due in part to a number of factors including the variety of measures employed by the research teams and the different populations (in terms of symptoms and diagnoses) investigated in trials. It does not seem reasonable to compare CBT for stable yet drug-refractory positive symptoms with CBT delivered during the acute phase. In addition, it has recently been argued by Birchwood (2003) that the outcomes for CBT should not be the same as the outcomes for antipsychotic medication. In an example of this, Trower et al. (2004) have reported positive outcomes for their trial of cognitive therapy for command hallucinations. There are also limited data pertaining to the maintenance of therapy gains following treatment; further research is needed in this regard.

In addition, there is insufficient evidence at this juncture to support the use of CBT as a relapse prevention strategy. This may in part be due to the fact that many trials recruit participants who have chronic drug-resistant psychotic symptoms, but who are otherwise stable. In addition, few studies have had relapse as their primary outcome. However, examination of the treatment manuals adopted by trial investigators reveals that relapse prevention strategies are included within these protocols (Kingdon & Turkington, 1994; Fowler et al., 1995). These strategies focus on helping individuals recognise and respond to early signs of relapse by seeking help but do not specify particular cognitions or behaviours associated with the development of relapse acceleration, nor do these manuals specify psychological strategies to address cognition or behaviour during relapse. It would not seem unreasonable to suggest that the current lack of results with regard to relapse could, perhaps, be due in part to the inadequacy of existing CBT treatment protocols for relapse in psychosis. Indeed, those studies that specifically target the prevention of relapse (Bach & Hayes, 2002; Gumley et al., 2003) as a primary outcome show that cognitive behavioural intervention can be effective in the maintenance of recovery and staying well after psychosis. Therefore, there is a need for a specific manualised psychological intervention aimed at facilitating emotional recovery and relapse prevention. This book aims to achieve this goal.

AFFECT, MEANING AND RELAPSE

A key aspect of relapse is the experience of high levels of emotional distress and affective dysregulation in the period before, during and following the acute phase of psychosis. This has long been recognised by researchers and clinicians alike. For example, Docherty and colleagues (1978) proposed that prior to the development of a full-blown relapse there were identifiable and sequential phases, which they saw as an unfolding of a series of psychological states. These phases were conceptualised as being characterised by feelings of over-extension, restricted consciousness, behavioural and affective disinhibition, psychotic disorganisation and resolution. During the first phase of over-extension, the person experiences a sense of being overwhelmed by stressful demands or internal/external conflicts and is accompanied by feelings of fear, threat, anxiety and nervousness. This phase is followed by the appearance of a variety of intrusive mental phenomena, which limit the person's ability to concentrate and think. The person experiences feelings of helplessness, hopelessness, dissatisfaction and loneliness. During the disinhibition phase, the ability of the individual to modulate or regulate their internal impulses becomes impaired. The signs and symptoms of this phase are thought to be rage, panic and hypomania.

This precedes increasing perceptual and cognitive disorganisation, loss of self-identity and fragmentation of control during the active phase of psychosis. Docherty and colleagues' formulation emphasises a sequential view of the nature of relapse, where relapse is characterised by the progression of increasing non-psychotic symptoms, through increased emotional distress, affective dysregulation, psychological fragmentation, and feelings of loss of control, culminating in the evolution of psychosis.

The importance of the role of affect in psychotic relapse has been consistently demonstrated in a number of retrospective and prospective studies examining the prediction of relapse itself. Retrospective studies of individuals and their families (Herz & Melville, 1980; McCandless-Glimcher et al., 1986; Birchwood et al., 1989) show that the most commonly reported early signs of relapse are fearfulness, anxiety, poor sleep, irritability, tension, depression and social withdrawal. In their seminal study, Herz and Melville (1980) made the first attempt to systematically identify and characterise the early signs of relapse. A total of 145 people who had been diagnosed with schizophrenia and 80 of their relatives in the study were asked for their responses to the following question: 'Could you tell if there were any changes in your thoughts, feelings, and behaviours that might have led you to believe that you were becoming sick and might have to go to the hospital?' Approximately 70 per cent of participants reported noticing changes. Families were marginally more likely than the patients themselves to identify changes and, in about 66 per cent of cases, both families and individuals were in agreement. For most individuals and their families the time interval before relapse was more than one week. Between 50 and 60 per cent of individuals and families sought professional help; however, less than 4 per cent had been advised by a health professional to do so. In addition, Creer and Wing (1974) also reported in a survey of 80 relatives that virtually none had been given advice about the nature of early signs of relapse. The experiences described by relatives were ranked in terms of their frequency of being reported. The most commonly reported experiences were fearfulness/anxiety, tension and nervousness, sleeplessness, trouble concentrating, and loss of appetite and pleasure. These data are consistent with Docherty and colleagues' (1978) proposal that the earliest stages of relapse appear to be characterised by increased anxiety, fearfulness and tension. Herz and Melville's findings have been confirmed by three further retrospective studies (Thurm & Haefner, 1987; Kumar et al., 1989; Hamera et al., 1992).

The consistency with which early signs have been reported has led to the development of prospective investigations of early signs. In essence, these studies have sought to identify the sensitivity and specificity of these early signs as an indicator of emerging relapse. Clearly if these early signs are sensitive and specific to relapse, the monitoring of such signs, and associated emotional distress, would help facilitate earlier interventions – potentially

leading to the prevention and/or amelioration of relapse. In the investigation of the predictive power of early signs monitoring, *sensitivity* refers to the ability of the monitoring system to correctly identify a forthcoming relapse. It is essentially the proportion of individuals who experience early signs prior to a relapse. *Specificity* refers to the power of these early signs to correctly identify those individuals or times when a relapse will not occur (see Table 1.2).

Table 1.3 provides a summary of prospective studies of early signs and relapse. Three of these studies (Jolley et al., 1990; Gaebel et al., 1993; Marder et al., 1994) were conducted in the context of a concurrent intervention trial which makes the relationship between early signs and relapse more difficult to ascertain due to the impact of interventions on this relationship. Four studies investigated observer-rated early signs alone (Subotnik & Neuchterlein, 1988; Tarrier et al., 1991; Gaebel et al., 1993; Marder et al., 1994), three studies investigated both observer- and self-rated early signs (Birchwood et al., 1989; Malla & Norman, 1994; Jorgensen, 1998) and one study investigated self-rated early signs alone (Hirsch & Jolley, 1989). Subotnik and Neuchterlein (1988) reported a prospective study of early signs in relation to relapse among 50 individuals. Participants were monitored fortnightly and relapse was defined by a rating of severe or extremely severe on the BPRS Unusual Thought Content, Conceptual Disorganization, and/or Hallucinations items. Greater suspiciousness and thought disturbance symptoms correctly identified 10 out of the 17 relapses. This gave a sensitivity to relapse of 59 per cent. Birchwood and colleagues (1989) recruited 17 individual participants who were monitored fortnightly using the self-rated or observer-rated Early Signs Scale over a nine-month period. Relapse was defined as any hospital admission or a clinician's judgement of imminent relapse or probable admission. Some 82 per cent of those who experienced a relapse had an increase in early signs prior to relapse; 62 per cent of those who did show an increase in early signs went on to have a relapse, meaning that 38 per cent had an increase in early signs but did not go on to relapse. Tarrier et al. (1991) monitored 56 participants on a monthly basis. Relapse was defined as a reappearance of positive psychotic symptoms or the worsening of persistent or residual positive symptoms, which lasted for at least one week. Depressed mood alone was associated with a sensitivity of 50 per

Table 1.2 Sensitivity and specificity

	High	*Low*
Sensitivity	Low false positives	High false positives
Specificity	Low false negatives	High false negatives

Table 1.3 Studies of the sensitivity and specificity of early signs to relapse in schizophrenia

Study	*Assessment of early signs*	*Number of relapses*	*Sensitivity (%)*	*Specificity (%)*
Subotnik & Neuchterlein (1988)	Observer-rated	17	59	NR
Birchwood et al. (1989)	Self-rated Observer-rated	8	62	82
Hirsch & Jolley (1989)	Self-rated	10	73	NR
Tarrier et al. (1991)	Observer-rated	16	50	81
Gaebel et al. (1993)	Observer-rated	162	8 14 10	90 70 93
Marder et al. (1994)	Observer-rated	42	37 48	NR
Malla & Norman (1994)	Self-rated Observer-rated	24	50	90
Jorgensen (1998)	Self-rated Observer-rated	27	78 30	45 58

cent and specificity value of 81 per cent. When depression was combined with hallucinations, the sensitivity value increased to 62.5 per cent and the specificity value was 87.5 per cent. Malla and Norman (1994) monitored 55 participants on a monthly basis over a period of at least 12 months (range: 12–29 months). In this study many increases in psychotic experiences were not preceded by increases in emotional distress, unless accompanied by increases in psychotic symptoms. Jorgensen (1998) monitored 60 individuals, 30 of whom had residual positive psychotic symptoms ('symptomatic'), and 30 who were fully remitted ('asymptomatic'). Participants were interviewed every fortnight over six months or to relapse. A priori, different cut-off points were selected to identify early signs. These cut-off points were defined as changes in early signs scores $\geqslant 5$, $\geqslant 10$ or $\geqslant 15$. Sensitivity and specificity values in Table 1.3 are given for $\geqslant 5$. In total, 45 per cent participants relapsed, 27 per cent of whom were readmitted to hospital. For symptomatic participants sensitivity of early signs to relapse was 88 per cent and specificity 64 per cent, and for asymptomatic participants the sensitivity value was 73 per cent and the specificity value was 89 per cent. Across the eight studies reporting sensitivity and specificity for early signs to relapse, the findings on sensitivity values range from 8 to 88 per cent, and for speci-

ficity values from 45 to 93 per cent. Strict comparison across these studies is problematic given the nature of differences in methodology and design. However, a number of conclusions are possible on the basis of these data.

Observer versus Self-rated Detection

While it is not easy to group together studies examining observer-related and studies examining self-rated early signs due to important methodological differences, it is noteworthy that the median sensitivity of observer-rated early signs (Subotnik & Neuchterlein, 1988; Tarrier et al., 1991; Gaebel et al., 1993; Marder et al., 1994) was 37 per cent, while for those studies incorporating self-rated early signs monitoring (Birchwood et al., 1989; Malla & Norman, 1994; Jorgensen, 1998; Hirsch & Jolley, 1989) the median sensitivity results were 68 per cent. As the reader will recall, sensitivity refers to the ability of early signs to correctly identify a forthcoming relapse. Therefore, in this case self-rated early signs seem much more powerful in predicting relapse, suggesting that individuals' unique knowledge of their own experiences gives them a better ability to predict relapse than the health professionals who provide support and treatment for them. This also means that it is likely that individuals are detecting their own idiosyncratic signs of relapse at an early stage. Depending on their experiences of previous episodes of relapse/psychosis, this is likely to generate a high degree of emotional distress.

Emotional Distress as a Psychological Reaction

It is also apparent that when studies include positive psychotic experiences or incipient psychosis in their definitions of early signs (Subotnik & Nuechterlein, 1988; Birchwood et al., 1989; Tarrier et al., 1991; Jorgensen, 1998), this increases the sensitivity of early signs detection to relapse. The inclusion of low-level positive psychotic symptoms, such as ideas of reference or thought control, suggests that the development of emotional distress signals the person's emotional reaction to the re-emergence of psychotic experiences. The consistency of the findings on specificity of early signs to relapse, which is reported across these studies (64 to 93 per cent), is supportive of this proposal. That is, when there is a relapse, there is almost always an increase in emotional distress beforehand.

Underlying Experiences

It is likely that individuals may well be responding to quite subtle changes in their cognition, perception and attention that are psychologically significant or reminiscent of psychotic experiences. Early studies (e.g. McGhie & Chapman, 1961; Chapman & McGhie, 1963; Freedman & Chapman, 1973;

Docherty et al., 1978; Henrichs et al., 1985) found in clinical interviews that idiosyncratic changes in the perception of cognition, emotion and interpersonal experience appeared to be associated with psychosis, and that these experiences are different to those whose psychosis has remitted or those suffering from depression (Cutting, 1985). Chapman and McGhie (1963) suggested that individuals with psychosis become aware of unusual experiences, and that their reactions to these experiences may play an important role in the development and maintenance of psychosis. They recommended that a psychotherapeutic understanding of the individual's perceptual and experiential difficulties would aid improved communication. In addition, they suggested that psychotherapy should aim: (1) to discover individuals' subjective experiences and cognitive difficulties; and (2) to reduce unhelpful or ineffective reactions to these experiences. Bowers (1968) argued that self-experienced changes in perception and awareness were critical to the transformation of normal experience into psychosis. In an experiential account drawn from interviews with 15 people with psychosis, Bowers described changes in heightened awareness of internal and external stimuli. Associated with these perceptual changes, he described individuals as having an increasing sense of urgency, reduced need for sleep, exaggerated affect, and a heightened sense of self. Alongside this heightened experience, internal and external events and stimuli normally outside awareness became meaningful and self-relevant. Individuals described becoming engaged, fascinated, perplexed or indeed scared by their own experience. This state of heightened awareness of self gave way to what Bowers referred to as 'a dissolution of self' or 'loss of mental self-representation'. This loss of meaning combined with a heightened awareness of internal and external stimuli, gave way to the development and evolution of delusional beliefs constructed to make sense of 'heightened and altered sensory influx and self experience, widened categories of relevance and a push for closure or meaning' (1968, p. 352).

A COGNITIVE BEHAVIOURAL MODEL OF EARLY SIGNS AND RELAPSE

There have been a number of psychological conceptualisations of relapse (Thurm & Haefner, 1987; Birchwood, 1995; Gumley et al., 1999). All these models have emphasised how individuals interpret subtle signs (e.g. cognitive perceptual changes) and/or isolated symptoms (e.g. interpersonal sensitivity) as evidence of a forthcoming relapse of their psychosis. In this context individuals' interpretations of their experiences will be informed by their specific autobiographical memories (of psychosis). For some individuals who do not accept the construct of psychosis or illness, these signs may signal elevated interpersonal danger (e.g. 'if my doctor sees that I'm suspicious, he'll put me in hospital again'). These memories and appraisals drive

the development of heightened emotional distress and trigger affective dysregulation. Coping strategies adopted by individuals may enable them to reduce their levels of emotional distress or support affective stabilisation. For example, being able to talk with a trusted friend or family member, being able to self-soothe, having a kindly, accepting and compassionate attitude to oneself, being able to decatastrophise relapse, or being able to access appropriate support and assistance available may all positively impact on coping. Three studies (Brier & Strauss, 1983; McCandless-Glimcher et al., 1986; Hultman et al., 1997) show that patients monitor and regulate their symptoms in order to prevent relapse. Among individuals with bipolar disorder, Lam (1997, 2001) reported on the use of spontaneous cognitive and behavioural coping strategies during prodromal stages, and also the impact that these had on functioning. They reported that the use of behavioural coping strategies had an effect on reducing the likelihood of manic relapse. On the other hand, having few interpersonal resources, living in a highly stressful environment or being socially isolated may well limit the availability and flexibility of coping strategies or the opportunities for help-seeking. The use of coping strategies such as substance use or medication discontinuation (to reduce side effects) may provide short-term relief but enhance relapse risk in the medium and long term. Social avoidance and withdrawal may enhance interpersonal sensitivity, rumination and emotional distress leading to feelings of helplessness, hopelessness and suicidal thinking. Hultman et al. (1997) found that individuals with a withdrawal-orientated coping style were more likely to relapse than individuals who had a socially orientated coping style. In addition, problematic thought control strategies or avoidance strategies may prevent disconfirmation of excessively negative beliefs about relapse, thus (1) maintaining an elevated sense of threat of relapse; and (2) increasing the likelihood of greater relapse acceleration at the manifestations of early signs. Safety behaviours are a kind of coping strategy specifically targeted at attempting to avoid a feared outcome. Not only do these behaviours attempt to avert a feared outcome, they also prevent the individual from disconfirming unhelpful beliefs, and thus play a role in the maintenance of anxiety and psychological distress. This clinical account of relapse is summarised in Figure 1.2.

Freeman et al. (2001) examined safety behaviours associated with persecutory delusions among individuals with psychosis. In this study all participants reported the use of safety behaviours to reduce the perceived threat arising from their persecutory beliefs, and the authors hypothesised that these safety behaviours were involved in the maintenance of delusions by preventing disconfirmation of threat beliefs. In a modification of the Freeman et al. (2001) interview, Gumley and colleagues (in preparation) found that when participants with psychosis (n=24) were asked if they had done anything to prevent or minimise relapse or readmission to hospital in the month

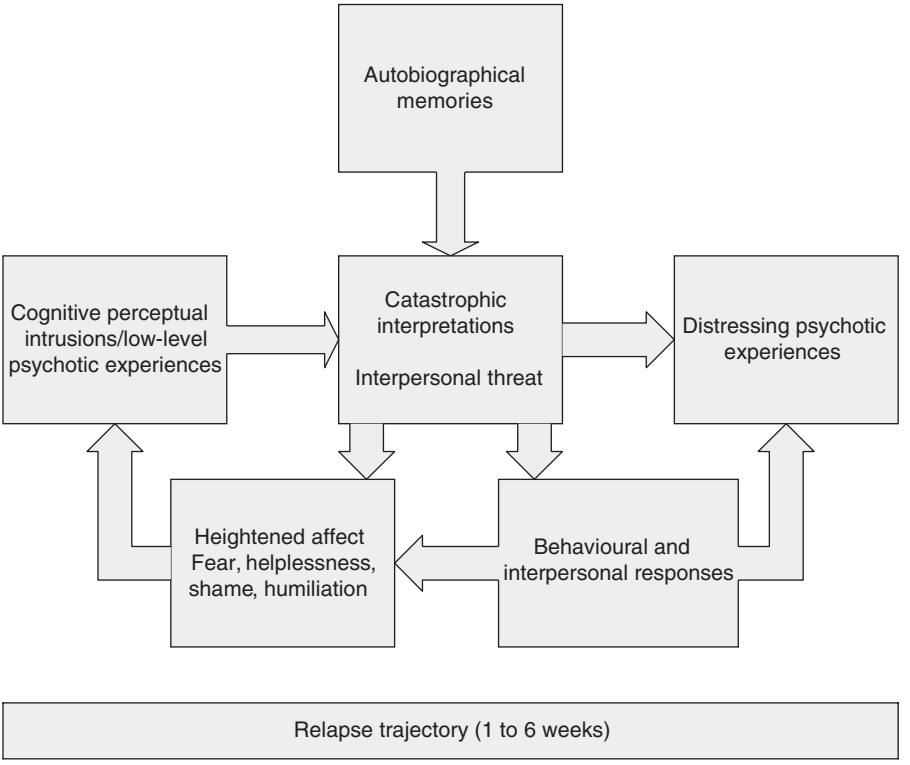

Figure 1.2 A cognitive behavioural model of early signs and relapse

prior to interview, 20 (83.3 per cent) reported using some strategy to prevent recurrence. These strategies included avoidance of people or places (n=14, 58.3 per cent), help seeking from friends, family or mental health staff (n = 10, 41.7 per cent) or avoidance of thinking about relapse or memories of relapse (n=12, 50 per cent). These studies emphasise a particular pathway of avoidant coping strategies which may well be influenced by further emotional and interpersonal processes.

Rachel

Rachel lives alone in a block of flats. She has had two periods of being in hospital after experiencing frightening voices telling her that she was evil, that she should kill herself, and that other people were plotting to kill her and her family. On both occasions Rachel was admitted to her local psychiatric hospital under section. Both admissions

were prolonged, lasting for three and five months respectively. Prior to her first episode she worked in a hair salon and was in the second year of her apprenticeship to be a hairdresser. In the twelve months before her first episode her mother and father split up. She had made strong efforts to keep up contact with her mum and dad but frequent arguments between her parents made it difficult for Rachel not to feel that her loyalty was being stretched. Rachel became increasingly depressed and paranoid before her first episode of psychosis. She felt responsible for her parents' marriage break-up, she felt that she was a bad person and that she was being punished for being an inadequate daughter. During her hospital admission, Rachel made two suicide attempts and was under constant observation for a considerable period of time.

Prior to her second admission less than a year after her discharge, Rachel began to feel low, increasingly sensitive to criticism and self-conscious. She was afraid that she was becoming depressed again ('I'm getting unwell again'), that the stress of returning to work was too much ('I can't cope') and that other people would see that she was not coping ('I'm a failure and a disappointment'). Rachel gave a vivid visual account of her memory of how her voices would talk to her, telling her to kill herself for the sake of her family. This memory was contextualised by her psychiatric admission. She recalled experiencing this while lying in her hospital bed, simultaneously being observed by a nurse sitting just outside her room. Her accompanying thoughts were that ('I am defective, obsolete'). Rachel began to experience increased feelings of demoralisation, shame and helplessness. Her self-critical thoughts became amplified and when she began to hear a voice telling her that she was useless, Rachel experienced high levels of panic. She felt that she couldn't tell anyone, that she would have let down her parents, her doctor, her colleagues at work and her community nurse. In addition, the intensity of distress was overwhelming, undermining her ability to reflect upon her own thoughts and memories and find a way of communicating these to others. In addition, it is noteworthy that it was Rachel's perception of others' opinions of her that triggered negative self-appraisals. She withdrew further, fearful that she would be punished if people found out that she was unwell again.

Rachel's relapse formulation was collaboratively developed with her during her recovery following her second hospital admission. As you will see, this formulation emphasises the dynamic nature of relapse and aims to give form and structure to her experience rather than relying on a simple checklist of signs and symptoms. Previously we have argued:

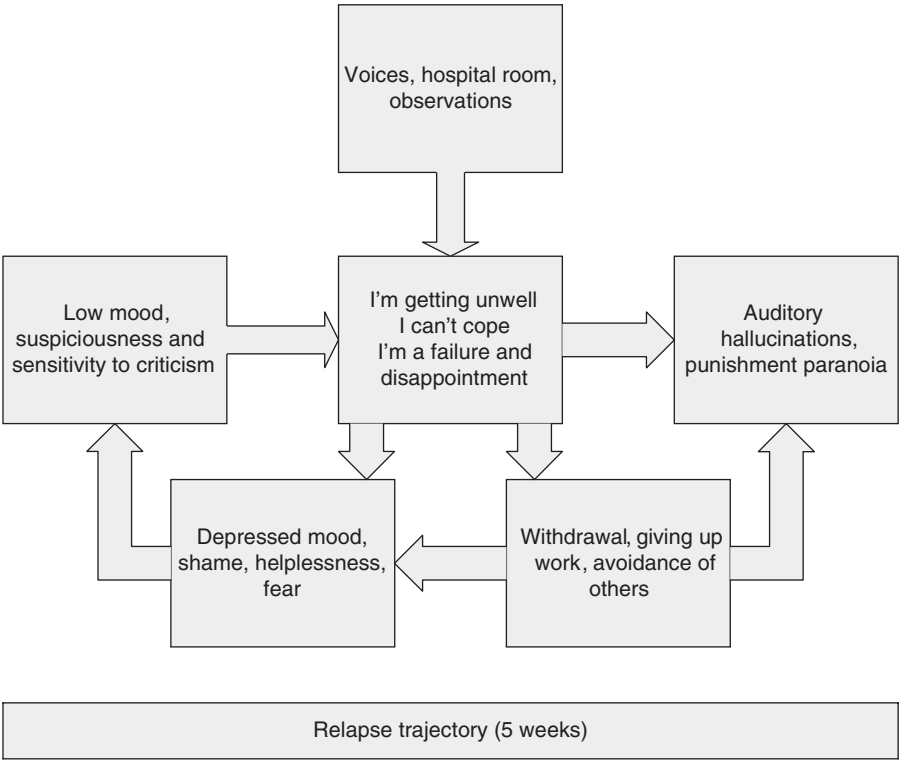

Figure 1.3 Rachel: A cognitive behavioural formulation of early signs and relapse

> definitions that are used to capture the sensitivity of early signs could well benefit from definitions more closely allied to those negative beliefs about self and illness, which are hypothesised to dictate relapse speed and acceleration – rather than relying on a more closely delineated set of individual signs and symptoms alone.
>
> (Gumley et al., 1999)

Figure 1.3 shows Rachel's formulation of her relapse.

ANTIPSYCHOTIC MEDICATION AND RELAPSE

The routine care received by individuals with psychosis emphasises the prescription of and adherence to antipsychotic medication. For many individuals, adherence to antipsychotic medication is difficult. In a systematic review of the literature, Lacro and colleagues (2002) found an average rate of non-adherence to antipsychotic medication between 41.2 and 49.5 per cent. In the 39 articles included in the review, the factors most consistently associated

with non-adherence included poor insight, negative beliefs about medication, previous non-adherence, substance use, shorter duration of psychosis, poor discharge planning after inpatient treatment, and poorer therapeutic alliance. It seems interesting to us that negative attitudes to medication, lack of insight and previous non-adherence are related to non-adherence. These factors seem to suggest a lack of acceptance of psychiatric constructs of illness which are then used by investigators to define insight or describe attitudes to medication. This seems to lead to circularity in conceptualising non-adherence. We find that our clients reject medication because of negative side effects and lack of successful experiences of help for their problems.

Robinson and colleagues (1999) conducted a long-term study of individuals following being diagnosed with a first episode of schizophrenia or schizoaffective disorder. A total of 104 participants were followed up for a period of five years. At the end of the five-year follow-up, the cumulative rate of relapse for the 104 participants was 81.9 per cent. Of those participants, 63 recovered after the first relapse. The cumulative rate for second relapse was 78.0 per cent. Of this group, 20 made a recovery after a second relapse. Among this group, 86.2 per cent relapsed for a third time. In their analysis, the risk for a first and second relapse was almost five times greater when not taking medication than when taking medication. Thirteen stable participants who were not taking antipsychotic medication dropped out of the study. When these participants were included in the analysis, the risk of relapse was three times greater for those not taking medication. Robinson and colleagues considered that the relationship between discontinuation of medication and relapse may have been an artefact of discontinuation as a result of the relapse process itself (for example, loss of insight). This was examined in subsequent analyses by looking at the length of time between stopping medication and relapse. The effect for this relationship remained significant using 14-, 28- and 56-day time differences, thus indicating that medication discontinuation was unlikely to be a manifestation of relapse itself. Those who discontinued their medication also showed poorer pre-morbid adjustment in terms of social isolation and poor adaptation to school. In conclusion, this study shows that sudden discontinuation of antipsychotic medication is a risk factor for early relapse.

Another important study of antipsychotic medication is the Northwick Park First Episode Study (Johnstone et al., 1986) that followed up 120 individuals with a diagnosis of first episode of schizophrenia. For the first month following discharge from hospital, all participants remained on their antipsychotic medication. Participants who completed this one-month phase without readmission were then randomly assigned to receive either placebo or active medication. Relapse was defined as (1) readmission to psychiatric care for any reason; (2) readmission considered necessary but not possible; and (3) the prescription of antipsychotic medication considered essential by the person's psychiatrist due to features of imminent relapse (i.e. early signs).

Table 1.4 Northwick Park Study of first episodes of schizophrenia. Relapse outcome at 6, 12, 18, and 24 months

Group	*Entered N*	*Relapsed N (%)*	*Relapse-free (months) (N, %)*			
			6	*12*	*18*	*24*
DUP < 1 year						
Active	31	10 (32)	25 (80)	25 (80)	22 (71)	18 (59)
Placebo	51	26 (51)	39 (66)	23 (45)	21 (42)	21 (42)
Total	82	36 (44)	64 (72)	48 (59)	43 (54)	39 (48)
DUP > 1 year						
Active	22	15 (75)	17 (76)	7 (33)	6 (26)	4 (18)
Placebo	13	13 (100)	3 (23)	1 (8)	–	–
Total	25	28 (80)	20 (55)	8 (23)	6 (14)	4 (10)
Whole group						
Active	54	25 (46)	43 (79)	33 (62)	29 (54)	23 (42)
Placebo	66	41 (62)	38 (57)	24 (37)	22 (33)	20 (30)
Total	120	66 (55)	81 (67)	57 (48)	51 (42)	43 (35)

Johnstone and her colleagues found that of those on maintenance antipsychotic medication, 46 per cent relapsed, and of those on placebo, 62 per cent relapsed by 24 months. These results are illustrated in Table 1.4. It is of interest that Table 1.4 shows that for those with a duration of untreated psychosis (DUP) greater than one year the relapse rate for those on maintenance medication is 75 per cent and those on placebo is 100 per cent. In contrast, those with a DUP less than one year, the relapse rates are 32 per cent and 52 per cent respectively. This shows how adjustment to early psychosis (as measured by DUP) mediates the impact of antipsychotic medication on relapse rate. Indeed, for those with a shorter duration of untreated psychosis, 50 per cent remain relapse-free on placebo.

Low-dosage or Discontinued Antipsychotic Medication

Although research shows that adhering to antipsychotic medication is an effective way of preventing relapse (for example, Crow et al., 1986; Robinson et al., 1999), antipsychotic medication exerts a social cost, and a number of serious complications can occur after prolonged use. Indeed, the occurrence of extra pyramidal Parkinsonian side effects is a major problem, which compromises both the long-term use and acceptability of antipsychotic medication. Even new so-called atypical medications exert major metabolic side effects including clinical obesity cardiovascular difficulties and heightened risk of Type II diabetes. Robinson et al. (2002) found that the major pre-

dictor of discontinuation in their 1999 study (described earlier) was the occurrence of Parkinsonian side effects. Low-dose strategies have been developed, with the aim of establishing the minimum dosage needed to prevent relapse. Such an approach to treatment is based on the proposal that antipsychotic dosages can be prescribed at dosages less than those required in the acute phase (Marder, 1999), and that the dosage can then be increased if necessary to prevent relapse. Barbui and colleagues (1996) conducted a meta-analysis of randomised controlled studies, which had compared low-dosage antipsychotic medication with conventional dosage (Kane et al., 1983; Marder et al., 1984 1987; Johnson et al., 1987; Hogarty et al., 1988; Inderbitzin et al., 1994). A total of 415 individuals (214 in low dosage, and 201 in the conventional dosage group) participated in these studies. In all the studies selected, relapse was defined as either an increase in two or more points on the BPRS (Overall & Gorham, 1962) or a worsening of psychotic experiences requiring hospital admission. Low dosage was defined in three of the trials as 20 per cent of the conventional dosage (Marder et al., 1984, 1987; Hogarty et al., 1988); 10 per cent in one trial (Kane et al., 1983) and 50 per cent in two trials (Johnson et al., 1987; Inderbitzin et al., 1994). All studies employed a 12-month follow-up, except two, which followed participants to 24 months (Marder et al., 1987; Hogarty et al., 1988).

Table 1.5 illustrates the results of this review. In Table 1.5, relative risk refers to the risk of relapse in the low-dosage group compared to the risk of relapse in the conventional group. A relative risk ratio of 2.00 means that the risk of an event occurring in an experimental group is two times greater than the risk of that event occurring in a control group. Relative risk reduction (RRR) is the complement of relative risk, expressed as a percentage. The results showed that there was a significant increase in relapse rate associated with low-dosage antipsychotic medication over 12 months. However, over 24 months, relapse rates did not significantly differ. There are two implications of these findings. First, over 12 months at least, it was not possible to make substantial reductions in antipsychotic dosage without increasing the risk of relapse. There is a fine balance between maximising the protection against risk of relapse, and minimising the social costs exerted by antipsychotics. Second, it is notable that no significant differences were found between low and conventional dosages for relapse rate over 24 months. Therefore, those who do not relapse within the first year following a reduction in their medication dosage tend not to relapse in the second year.

Gilbert and colleagues (1995) conducted an extensive systematic review of studies investigating relapse rate following the withdrawal of antipsychotic medication. In this review 66 studies including a total of 4,365 participants conducted between 1958 and 1993 were reported. Twenty-nine of these studies involved paired comparisons with groups who continued to receive their antipsychotic medication. Overall, the risk of relapse among those who

Table 1.5 Low-dosage studies of antipsychotic medication and relapse. Intention to treat analysis at 12 and 24 months

Study	*Relapse*		*Relative risk (95% CI** RR)*	*RRR* (%) (95% CI RRR)*	*p*
	Conventional	*Low dosage*			
Kane et al. (1983)	48	77	1.60 (1.20–2.13)		<0.001
Marder et al. (1984)	36	29	0.79 (0.35–1.76)		NS
Johnson et al. (1987)	10	34	3.56 (1.09–11.07)		<0.05
Hogarty et al. (1988)	24	35	1.45 (0.69–3.05)		NS
Inderbitzin et al. (1994)	35	35	0.99 (0.44–2.25)		NS
12 months overall				–47 (–15 to –88)	<0.005
Hogarty et al. (1988)	42	43	1.02 (0.59–1.75)		NS
Marder et al. (1987)	45	49	1.08 (0.64–1.80)		NS
24 months overall				–5 (28 to –52)	NS

Notes: *RRR: relative risk reduction. **CI: Confidence Interval.
Source: Barbui et al. (1996). Low-dose neuroleptic therapy and relapse in schizophrenia: meta-analysis of randomized controlled trials, *European Psychiatry*, **11**(6), 306–313.

discontinued their antipsychotic medication was greater (53 per cent versus 16 per cent) over an average follow-up period of almost 10 months. Risk of relapse was greatest in the first three months after discontinuing medication (50 per cent versus 4 per cent) demonstrating that relapses occur early following discontinuation and that those who remain free of relapse after three months tend to remain relapse-free for the longer term. Of the studies included in the review, 33 involved abrupt discontinuation of medication (less than 14 days and usually one day). These studies found that the risk of relapse was three times greater following abrupt withdrawal compared to gradual withdrawal, and those receiving a higher antipsychotic dosage were at even higher risk of relapse.

The above findings support the proposition that antipsychotic medication reduction strategies are feasible, advantageous and effective for many individuals, but do not necessarily require complete drug withdrawal (Carpenter & Tamminga, 1995). Low-dosage strategies are associated with an increased

risk of relapse over 12 months, but at 24 months, 50 per cent of those on lower dosage remain well. Similarly, in drug discontinuation studies the risk of relapse is heightened considerably, particularly in the first three months after medication is withdrawn. However, over the longer term, almost 50 per cent remain relapse-free without medication. Indeed, the Johnson study suggests that those who remain well at 12 months continue to remain well at 24 months. Those with a longer duration of untreated psychosis (Crow et al., 1986), those who have an unplanned (Robinson et al., 1999, 2002) or abrupt discontinuation (Gilbert et al., 1995) are at very high risk of relapse and relapse itself is likely to occur within three months of discontinuation. It is important to consider the very high rate of relapse (81.9 per cent over five years) in the Robinson study, as this study strongly indicates that adherence to antipsychotic medication is a necessity to prevent relapse. However, in this study, discontinuation from medication was defined as an *unplanned* period of non-adherence from medication lasting at least one week or longer. These discontinuations were therefore likely to be abrupt and not in consultation with those involved in the individuals' care.

More recent evidence from Gaebel and colleagues (2002) showed that 58 per cent of individuals who have a *planned* discontinuation of their medication following a first episode of psychosis and who are offered systematic regular early signs monitoring (with reinstatement of medication if a possible relapse is indicated) remain relapse-free, compared to 62 per cent of individuals with a first episode of psychosis who continue on maintenance antipsychotic medication. Furthermore, Gitlin and colleagues (2001) followed up 53 individuals who had a planned discontinuation of their medication for an average of 18 months. During that period 78 per cent and 96 per cent experienced an exacerbation or relapse within one year and two years respectively. That notwithstanding, when hospitalisation was used as the principal relapse criterion, only six out of the 50 experienced an exacerbation or relapse that required readmission. This study shows the importance of continued support, follow-up and early signs monitoring (with prompt intervention if indicated) for those individuals who have the opportunity to discontinue or reduce their overall antipsychotic medication. It is striking that few, if any, studies describe provision of concurrent and robust psychological therapies for individuals who are participating in lower dosage or medication discontinuation studies. Furthermore, many of the aforementioned studies were conducted in an era that predates the (relatively recent) development of cognitive behavioural therapies specifically designed for individuals with psychosis.

IMPLICATIONS FOR STAYING WELL AFTER PSYCHOSIS

We have argued that the early signs of relapse are best conceptualised within a psychological framework, where affective experiences including fear, help-

lessness, shame, embarrassment and humiliation arise from the person's appraisal of subtle cognitive perceptual or low-level psychotic experiences. Individuals' appraisals have their origins in the specific and often distressing autobiographical memories of previous episodes of psychosis and their personal, interpersonal, social and vocational consequences. Individuals' emotional and behavioural responses to the emergence of early signs of relapse have the potential to accelerate, decelerate or prevent the onset of a full-blown return of psychotic experiences and re-hospitalisation. In this sense, early signs of relapse can be alternatively conceptualised as 'at-risk mental states'. Such at-risk mental states are more common in the context of non-adherence or sudden abrupt and unplanned discontinuations of antipsychotic medication. This has led to interventions and treatments, which emphasise adherence or compliance with antipsychotic medication (e.g. Kemp & David, 1998). While these interventions are potentially powerful in reducing risk of subsequent readmission to hospital, at least when delivered to inpatients, many individuals do not accept traditional constructs of illness to explain their experiences. In addition, they are distressed by taking antipsychotic medication or have negative beliefs about and/or expectations of medication. Measures to enforce compliance with medication are likely to engender a greater sense of alienation in patients and an increased likelihood of non-engagement with services. Thus, these individuals will correspondingly become alienated or excluded from other potentially valuable interventions, such as family therapy, which have proven value in the prevention of relapse.

We therefore feel that there is an urgent need to develop an individually tailored psychological approach to staying well after psychosis. Such an approach may support individuals in reducing the overall burden of their antipsychotic medication. However, an alternative treatment strategy such as this also has to engage individuals who are at ultra-high risk of relapse. Viewed as a group, these individuals are often difficult to engage in services (Tait et al., 2003) and are likely to be non-adherent to medication (Robinson et al., 2002). Our treatment approach outlined in Parts II and III is complementary to the many excellent CBT approaches in psychosis, for example, Max Birchwood and colleagues, Tony Morrison and colleagues, and David Fowler and colleagues. The aim of this book is to describe a cognitive interpersonal approach to engage individuals who are *at risk* of relapse, to support the emotional recovery and adjustment after psychosis, to reduce psychological vulnerability to relapse, and to provide prompt and immediate support to individuals with at-risk mental states for relapse.