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Group and individual based cognitive behavioural therapy for severe psychiatric disorders

The challenge of complex and comorbid disorders

Thesis for the degree philosophiae doctor

Trondheim, June 2008

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Contents

Summary

Epidemiological studies suggest that psychiatric comorbidity is widespread and common, and most psychiatric patients could be expected to show comorbid patterns of psychopathology. Since comorbid disorders are regarded as harder to treat and also plays a crucial role for the outcome of psychological treatment, there is of importance to investigate how cognitive behaviour therapy, may be adapted to treat complex and comorbid psychiatric conditions. The aim of this thesis has been to explore the effectiveness of group and individual based cognitive behaviour therapy in treating complex and comorbid psychiatric disorders.

Results suggest that cognitive behaviour therapy seems to make a clinical impact that improves symptoms and functioning of patients in the presence of multiple comorbid conditions, and different patterns of psychiatric comorbidity with high level of severity could be treated effectively using cognitive behaviour therapy both in individual therapy and in a group setting.

Acknowledgement

This study has been carried out at the Department of Psychology, Norwegian University of Science and Technology (NTNU), and has been supported by a grant from NTNU.

I would like to thank and acknowledge the following persons:

- The patients which have contributed to my research.

- My supervisor Hans Nordahl, who has introduced me to the field of cognitive behavioural therapy, and whose support and enthusiasm has provided me with inspiration and motivation during the process of writing this thesis.

- My supervisor Rolf. W. Gråwe, who has introduced me to the field of research related to psychotic disorders, and the significance of psychological research in this area.

- My colleagues at the PhD forum at the Department of Psychology, NTNU for inspiration and feedback during the hard times writing the papers.

- Finally and most of all, I want to especially thank Eli and my children Markus and Ida for being supportive and patient during this period, and for always reminding me about the important things in life.

List of papers included in the thesis.

The thesis is composed of 4 articles, referred by their Roman numerals.

I. Hagen, R., Nordahl, H. M., Kristiansen, L., & Morken, G. (2005). A randomised trial of cognitive group therapy vs. waiting list for patients with co-morbid psychiatric disorders: Effect of cognitive group therapy after treatment and six and 12 months follow up. *Behavioural and Cognitive Psychotherapy, 33,* 33-44.

II. Hagen, R., Nordahl, H. M., & Gråwe, R. W. (2005). Cognitive-behavioural group treatment of depression in patients with psychotic disorders. *Clinical Psychology & Psychotherapy, 12,* 465-474

III. Gråwe, R. W., Hagen, R., Espeland, B., & Mueser, K. T. (2007). The Better Life Program: Effects of group skills training for persons with severe mental illness and substance use disorders. *Journal of Mental Health, 16,* 625-634

IV. Hagen & Nordahl (2007) Behavioural experiments in the treatment of paranoid schizophrenia: A single case study. Accepted for publication in *Cognitive and Behavioural Practice.*

1. 0. Comorbidity.

For psychiatric disorders, comorbidity emerged as an important issue in classification as editions of the DSM/ICD10-manuals over time generated ever narrower and numerous diagnoses. With the introduction of DSM-IV (APA, 1994) and ICD-10 (WHO, 1992), it is reported an increase in comorbidity among psychiatric conditions. A challenge related to this categorical classification is the concept of heterogeneity, meaning that many diagnoses share the same symptoms. Heterogeneity and comorbidity are therefore closely linked with each other, and without this categorical classification schema there would not be a reason to discuss the concept of comorbidity (Maser, Cassano & Michelini, 1997).

The DSM /ICD tendency towards comorbidity could be viewed as allowing for the description of the various facets and manifestations of psychiatric illness (First, 2002). Although less comorbidity may be desirable to reduce diagnostic complexity, in the absence of knowledge about underlying pathophysiology, the trend in successive editions of the diagnostic manuals has been to reduce diagnostic hierarchies and increase comorbidity (First, 2005). It is important to understand that comorbidity in psychiatry does not imply the presence of multiple diseases or dysfunctions, but rather reflects our current inability to apply Occam's razor (i.e., a single diagnosis to account for all symptoms).

There are different ways to look at the idea of comorbid disorders. For instance comorbidity may be defined by the similarity across classes of disorders or by the period of time in which the disorders occur (Maser & Cloninger, 1990). In line with this definition, the co-occurrence of substance abuse and other mental disorders can be termed a "heterotypic" comorbidity (meaning comorbidity between different classes of mental disorders), which may be

contrasted with a "homotypic" comorbidity (meaning a comorbidity between different members of a general class of mental disorders). An example of a heterotypic comorbidity could be the co-occurrence of a depressive disorder in a patient with a diagnosis of schizophrenia, while a homotypic comorbidity could for example be the co-occurrence of social phobia and generalized anxiety disorder in a patient with comorbid anxiety disorders (Hall, Lynskey & Teeson, 2001). A second definition and distinction could be made between "concurrent" and "successive" comorbidity. Concurrent comorbidity is that in which two or more disorders are present at the same time, (e.g. schizophrenia and drug dependence), while successive comorbidity can be defined as comorbidity in which disorders may occur at different times in a person's life, in ways that may or may not be causally related to each other (Hall et al, 2001). Successive comorbidity is also known in the literature as lifetime comorbidity (Maser & Cloninger, 1990).

A number of studies have established that clinicians frequently underrecognize comorbidity when using unstructured clinical interviews, mainly because the semi-structured interview approach to identifying comorbidity has somewhat limited utility for the practicing clinicians, because clinicians do not generally have time available to conduct these structured interviews (Zimmermann & Mattia, 1999; Basco et al, 2000; Shear et al, 2000; Zimmermann & Chelminski 2003; Rush et al., 2005). Clinicians who seek only the diagnostic criteria for a specific disorder will probably miss a more global perspective of the entire pathology. Instead of a broad field, the clinician only sees a part of the whole picture. Published treatment guidelines and manuals reinforce this way of thinking, as they consider treatments for only one single diagnosis. One problem with such a classification system and thinking is that it can provide the clinician with a false sense of patient homogeneity (meaning all patients with a principal diagnosis of X are alike). By definition, categorical models of classification assume

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the existence of homogenous groups with little overlap. Patients within a particular diagnostic group can be quite heterogeneous, largely influenced by their comorbid symptoms.

The DSM- IV (APA, 1994) and the ICD-10 (WHO, 1992) does not suggest specific treatments for each disorder category and subcategory. However, modern treatment researchers have attempted to design treatments tailored to specific DSM/ICD-10 diagnostic categories. In this way comorbidity presents a challenge not only to the official nomenclature, it also presents a similar challenge to treatments designed and targeted for the same diagnostic categories (Maser, Cassano & Michelini, 1997). This way of looking at psychopathology influence treatment studies that try to examine the efficacy of psychotherapeutic treatments, because participants usually have been carefully screened to identify a homogenous group with a single psychiatric disorder. Almost all treatment outcome studies today investigate the efficacy of a treatment within a group of patients with one specific diagnosis, and either exclude or ignore comorbidity (Sanderson & McGinn, 1997). Such a design of doing treatment-studies tends to overestimate treatment effects, because patients with comorbid conditions often have been excluded. Randomized controlled trials are also complicated by comorbidity, because comorbidity can either act as a confounder and threaten the internal validity, or as an effect modifier threatening the internal and external validity of these studies (Groot, Beckerman, Lankhorst & Bouter, 2003).

1.2 The prevalence of comorbid disorders

Based on several epidemiological studies on comorbidity, diagnostic overlap seems to be the rule rather than the exception (First, 2005). Prevalence studies of diagnoses in both clinical samples and general population samples show that comorbidity among psychiatric disorders is highly common (Kessler, 1997). The Epidemiologic Catchment Area-study (ECA) was the first to document that comorbidity is widespread in the general population (Robins & Regier,

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1991). Over 54 percent of respondents in this study, who had a lifetime history of at least one DSM-III psychiatric disorder, were found to have a second diagnosis as well. Similar results as reported in the ECA-study have also been found in the National Comorbidity Survey (NCS). In the NCS-study, 56 percent of the respondents with a lifetime history of at least one DSM-III- R disorder also had one or more additional psychiatric disorders (Kessler et al., 1994). Results from The National Comorbidity Survey-Replication (NSC-R), using DSM- IV diagnoses, are consistent with the ECA and NCS studies (Kessler, Chiu, Demler, Walters, 2005). Data from a Norwegian psychiatric epidemiological study are also similar to the studies mentioned above. The results from this Norwegian study shows that 13 percent of the sample had two diagnoses, and 14 percent had three or more diagnoses (Kringlen, Torgersen & Cramer, 2001). An interpretation of these epidemiological data could be that since neither shared symptoms nor shared risk factors explained the strong co-occurrence between disorders, the most likely explanation is that having one disorder puts the affected person at risk of developing other disorders (Robins & Regier, 1991; Kessler et al., 1994).

These results mentioned above concern lifetime comorbidity, but maybe of greater clinical interest is the joint occurrence of multiple disorders in the same person over a recent interval of time. Results show that 45 percent of those who reported one psychiatric disorders as assessed in the NCS over a 12-month period, carried multiple diagnoses (Kessler et al., 1994). Clinical samples show that high concurrent and / or lifetime comorbidity rates have been demonstrated for virtually all Axis I DSM disorders, and the comorbidity rates for the Axis II personality disorders are even higher (Clark, Watson & Reynolds, 1995). Research data also indicate that comorbidity is not random, because most disorders show systematic comorbidity patterns with specific diagnoses or diagnostic classes (Clark et al, 1995). For instance various anxiety disorders have found to be strongly comorbid with other anxiety disorders,

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depression, substance abuse and personality disorder (Zimmermann & Mattia, 1999; Wilk et al, 2006)

The results from these different psychiatric epidemiological studies show that psychiatric comorbidity is highly prevalent. Results also show that the majority of people who suffer from any psychiatric disorder also have a history of at least one other. A comparison of pure versus comorbid cases of each disorder assessed in the NCS shows consistently, that comorbidity is associated with higher severity of symptoms, and that comorbidity also is associated with disorder chronicity (Kessler, Foster, Saunders & Stang, 1995). The NCS results also suggest that comorbid disorders are, in general, more severe than pure disorders.

The consequences of psychiatric comorbidity are not confined to increased treatment complexity and more severe illness course. Comorbid disorders are associated with greater social consequences than pure disorders, and findings suggest that the major societal burden of psychiatric disorder falls on people with comorbidity, with nearly 90 percent of NCS respondents having a severe 12-month psychiatric impairment reporting a lifetime history of three or more comorbid psychiatric disorders (Kessler, 1994: Kessler et al, 1994). The NCS data further show that respondents with a lifetime history of psychiatric comorbidity are significantly more likely than those with a history of only one disorder to experience impairments in a wide range of adult roles, and persons with comorbid disorders are more likely to be divorced, socially isolated and unemployed (Kessler et al, 1994). McDermud et al. (2001) have also found that comorbidity burden showed the strongest relation to impairment over and above the presence of any particular class of disorders.

What causes such a high rate of comorbidity among psychiatric disorders is not clear. Common causes of either a biological or environmental sort, effects of one disorder could affect the onset of other disorders, and the problem related to a categorical classification system could probably all work as good hypotheses. However, as Kessler (1997) wrote in relation to possible connections; the main impression one got when looking at the epidemiological results related to comorbidity is that most disorders are related to most other disorders in ways, that are very difficult to sort out.

Based upon these already mentioned findings it could be suggested that persons suffering from comorbidity are in need of a more complex and broad treatment, than persons suffering from just one psychiatric disorder (Wilk et al., 2006). In addition, in many contexts assessing the severity of the individual's dysfunction may be of greater importance than specifying the precise nature of the disorder (Clark et al, 1995).

1.3 Summary

The consistent result from comorbidity studies point to a pragmatically important clinical reality, namely that a patient presenting him or her for treatment with a single disorder would be the exception rather than the rule. Further, despite the high prevalence of comorbidity found in community samples, it is possible that comorbidity is undetected in routine clinical practice because of the typical process of establishing diagnoses through clinical interviews.

Even if we have problems finding explanations of the connection between different psychiatric disorders, there are a number of reasons why comorbidity should matter:
1. Since comorbidity seems to be the rule rather than the exception with psychiatric disorders, and if we do not take comorbidity into account, we could mistake characteristics of the disorder under study for those that are due to an ignored comorbid condition.

2. Patients with comorbid psychiatric disorders are often more impaired, suffer greater social disability and generate larger social costs. This is probably in part because comorbid disorders are not diagnosed and treated, and in part because persons with more than one disorder are difficult to treat.

3. To understand why different psychiatric disorders co-occur may also provide clinicians with important opportunities for prevention.

4. Comorbidity has important implications for treatment.

Owing to the fact that the implications of comorbidity in psychological treatment are of crucial importance to the outcome of therapy, and because it affects how psychotherapy best could be delivered to handle this complexity, this link will be further looked in to.

1.4 Implications for psychological treatment.

Clinicians and researchers are increasingly recognizing the importance of comorbidity in the application of therapeutic technique and its impact on efficacy (Clarkin & Levy, 2004). Comorbidity is important because it affects a host of variables related to treatment and treatment planning. Evidence suggests that comorbidity have a huge impact on psychological treatment, usually resulting in more challenging therapies with poorer outcomes (Roth & Fonagy, 2005). Comorbid conditions are also likely to lead to poorer end-state functioning, because at the end of treatment patients may continue to exhibit symptomatology related to the untreated comorbid condition (Roth & Fonagy, 2005).

Different studies have tried to look for specific client variables that influence the course and outcome of psychological treatment (Beutler, Clarkin & Bongar, 2000; Beutler & Harwood, 2000). One of these specific client variables relates to the complexity of the client's problems (which may be defined as comorbidity). The second variable relates to the duration of the

difficulties (i.e. the chronicity, frequency and extent of recurrence), which also relates to the concept of comorbid disorders. If the patient's problems are complex and they have been suffering from their psychiatric problems over a long time-period, results suggest that the effect of treatment was less good and the patients were also more vulnerable for relapses (Beutler et al., 2000).

Such findings create a major problem in the recommendation regarding appropriate treatment for patients suffering of psychiatric comorbid disorders. Considering the emerging data on the extent and significance on comorbidity, patients who are suffering from multiple diagnoses are in need of a comprehensive treatment, and this treatment requires interventions that address the patient's full range of symptoms (Sanderson & McGinn, 1997). This is also related to clinical practice where the patient's response would be maximized if the treatment was broadened to address each patient's entire symptom presentation, instead of focusing and treating just one disorder at a time (Wolfe, 1994). This approach would also to some degree solve the problem of heterogeneity and treatment implications. Since many of the diagnoses share the same symptom representation, maybe a symptom-oriented treatment would be effective (Clark et al, 1995).

A therapeutic approach that emphasizes comorbidity and also provide a comprehensive and integrated treatment for the patient's comorbid symptoms, would be a symptom-oriented model as an opposed to a diagnosis-based treatment. This shift from a disorder-model to a symptom-oriented model has already been applied in the treatment and understanding of psychotic disorders, and has successfully accumulated research and treatment approaches where the concept of comorbidity also has been included (Gould, Mueser, Bolton, Mays & Goff, 2001; Rector & Beck, 2001; Gaudiano, 2005, Rathod & Turkington, 2005; Turkington,

Kingdon & Weiden, 2006 Bentall, 2007). A symptom focused model allows clinicians to

tailor their treatment to address the specific symptom configuration of each patient,

maximising treatment efficiency (Sanderson & McGinn, 1997). Also, a treatment model that

focuses on symptoms may provide more optimal care and prevent relapse to a larger extent

than treatments that focuses on diagnoses (McGinn, Young & Sanderson, 1995).

2.0 Cognitive behavioural therapy.

2.1 The cognitive behavioural treatment model.

Cognitive- behavioural therapy (CBT) is a psychological approach that has a symptom -

focused perspective, and could be used with patient suffering from multiple diagnoses.

In addition to having a focus on the patient's individual comorbid symptoms, CBT is also

formulation driven, which could be beneficial both in the understanding and treatment of the

patient's comorbid symptoms (Tarrier, 2006). As a contrast to a diagnosis-based model,

which is based on a categorical way of thinking, the CBT approach facilitates the

customization of treatment on a more individual basis, and could therefore be more successful

in treating complex and comorbid problems.

The generic model of CBT is applied to most psychological disorders. According to Beck

(1976), CBT should be:

1) Based upon a cognitive model that describes the onset and maintenance of the disorder

being treated;

2) Formulation driven;

3) A structured process;

4) Based on shared problems and goals;

5) Educational, allowing the client to understand the process of therapy;

6) Utilising guided discovery as the engine for change;

7) Involve homework tasks; and finally

8) Be time-limited.

Based on the notion that cognitions plays a role in the maintenance of most psychiatric disorders, cognitive behavioural interventions seek to reduce distress and enhance adaptive coping by changing maladaptive beliefs and providing new skills. The various approaches differ somewhat in the extent whether they emphasize cognitive mechanisms at the sacrifice of more behavioural ones (Hollon & Beck, 2004). The goal of the cognitive-behavioural interventions is to change maladaptive beliefs, using a wide range of techniques. These techniques include elements of self-monitoring, identifying and challenging negative thoughts and assumptions that maintain problematic behaviour and experiences, de-catastrophization, scheduling activities, and behavioural experiments that in turn aid further self-monitoring and challenge dysfunctional beliefs (Wright, Basco & Thase, 2005)

2.2 The efficacy of cognitive-behavioural therapy.

CBT appears to be effective for a broad range of clinical and medical disorders, and moreover there are numerous indications that the cognitive and cognitive-behavioural interventions may produce more lasting changes than other interventions. A review of 16 meta-analyses shows large controlled effect sizes found for CBT for unipolar depression, generalized anxiety disorders, panic disorders with or without agoraphobia, social phobia, posttraumatic stress disorder, and childhood depressive and anxiety disorders (Butler, Chapman, Forman & Beck, 2006). Effect sizes for CBT for marital distress, anger, childhood somatic disorders, and chronic pain were in the moderate range. CBT was somewhat superior to antidepressants in the treatment of adult depression, and was equally effective as behaviour therapy in the treatment of adult depression and obsessive compulsive disorder. Large uncontrolled effect sizes were found for bulimia nervosa. Disorders such as substance abuse, bipolar disorders, psychotic symptoms, personality disorders, and anorexia nervosa are among the disorders receiving recent empirical attention regarding the effectiveness of CBT (Butler et al., 2006).

Comorbidity has been found to impact treatment response to cognitive-behaviour therapy negatively. Overall, patients with comorbid conditions responded less robustly to CBT, though the degree of impact differed between studies (Brown, Antony & Barlow, 1995; Erwin, Heimberg, Juster & Mindlin 2002; Newman, Mosfitt, Caspi & Silva, 1998). Equally striking in these studies of comorbidity and CBT, were the fact that despite the presence of multiple conditions, CBT nonetheless made a significant clinical impact that improved symptoms and functioning (Barlow, Allen & Choate, 2004).

2.3 Modalities of cognitive- behavioural therapy.

Although cognitive therapy was originally developed as an individual therapy, the approach has also been applied to groups. Cognitive-behaviour group therapy (CBGT) may be defined as therapy that uses the dynamics of the group format, in addition to the common cognitive behavioural therapy techniques to change distorted, maladaptive, and dysfunctional beliefs, interpretations, behaviours, and attitudes (Bieling, McCabe & Antony, 2006). The format of CBGT could be psycho-educative in nature, being open-ended or manualized. The frequency and duration of session in CBT-groups can vary considerably, depending on the choice of format and other patient-dependent variables. The number of participants in CBT-groups differs, but a number of 6-8 persons are usually regarded as maximum, with the exception of psycho-educational groups where the number of participants could be larger (Morrison, 2001).

Group therapy has been associated with many advantages, such as potential time and cost savings per patient, the possibility of treating a greater number of people, and reducing waiting lists. The effects of group cohesion, imitative behaviour, interpersonal learning, and opportunity for group members to serve as group therapist and offer mutual support, are claimed positive by-products of group treatment (Morrison, 2001; Yalom, 1995). Group

settings are also said to provide group members with the opportunity to recognise and share common experiences with other group members (Yalom, 1995). However, there are also many disadvantages associated with group therapy. These include the risk of one patient monopolizing therapy, confrontation between group members, and the development of subgroups. Group settings may also impact the group members to become more reluctant to discuss disturbing cognitions, and as a result of this fail to undertake and challenge these distressing cognitions (Morrison, 2001; Yalom, 1995). Advantages of cognitive behavioural groups compared to individual CBT, includes the opportunity to demonstrate the relationship between thoughts and feelings through the negative thoughts of the group members, and recognize the cognitive distortions of others, which could help the reattribution of their own cognitions (Bieling et al., 2006).

Early studies of CBGT focused mainly on depression, but over time group therapy has been extended to other areas in line with the evolvement of cognitive behavioural therapy models for others disorders, such as anxiety disorders, eating disorders, schizophrenia, bipolar disorders, substance disorders, etc. (Bieling et al., 2006). A conclusion based on Morrison's review (2001) and Petrocelli's meta-analysis (2002) is that for the most part there is little difference in results between CBGT and individual CBT treatment studies. However, depending on the client group, for example patients who are more severely impaired, more depressed, or have an OCD or social phobia disorder, the advantages of an individual approach sometimes outweighs the advantages of a group condition (Morrison, 2001).

3.0 Objectives and outline of the thesis.

As seen from the introduction comorbidity plays a crucial role in the outcome of psychological treatment. Comorbidity is of great importance regarding the wellbeing of people suffering from different concurrent psychiatric disorders, and it is important to explore how these problems could be treated from a CBT-point of view. The main objective of this thesis is to empirically explore the effectiveness of group and individual based cognitive behavioural therapy in treating complex and comorbid psychiatric disorders. The objectives for each paper were as follows:

Paper I. A randomized trial of cognitive group therapy.

In the literature, few studies have tested the effect of cognitive group therapy on a heterogeneous sample of patients with psychiatric disorders. In particular, patients with anxiety, depression, and cluster C personality disorders are comorbid conditions that the clinicians frequently encounter which are sometimes are difficult to treat. The aim of this study was to assess the effectiveness of cognitive therapy in a group format (CBGT) with a sample of heterogeneous psychiatric disorders.

Paper II. Cognitive behavioural group therapy of depression in patients with psychosis.

Comorbidity between depression and psychosis is common, but the literature is rather sparse related to psychological treatments of depression in patients with concurrent psychotic disorders. The aim of the study was explore the effects of CBGT for depression with schizophrenic and schizoaffective disorders.

Paper III. Group skills training for persons with severe mental illness and substance use disorders.

There is a growing consensus regarding the critical ingredients of effective group interventions for persons with co-occurring mental disorders and substance use disorders. Based on these clinical consensus and guidelines, "The Better Life Program" was developed and manualized to help individuals who suffer from this pattern of psychiatric comorbidity. The primary goal of this study was to evaluate the effect of the "The Better Life Program" on substance use and mental health outcomes.

Paper IV. Behavioural experiments in paranoid schizophrenia.

The empirical support for using CBT to treat psychotic symptoms has been widely established. This paper reflects the symptomatic complexity of a psychotic patient suffering from depression, substance abuse, and psychotic symptoms. The aim of the paper is to show how behavioural experiments could be used to change delusional thinking in a patient with schizophrenia, and also to discuss ideas for future research in CBT for psychotic symptoms.

Conclusion and shared objectives in the papers.

All the papers in this thesis involve the empirical investigation on the effect of cognitive behavioural therapy in both individual therapy and group therapy settings, to see if this approach could be an effective aid to both treat and understand comorbid disorders. The fundamental components of CBT, which have been emphasized in this thesis, related to the treatment of comorbid conditions are (Barlow et al, 2004);

(1) trying to change cognitive appraisals of events,

(2) reduce emotional and behavioural avoidance and,

(3) facilitate action that counters negative emotional states.

Instead of treating concurrent disorders in a sequential manner, a more integrated treatment

approach is used. This perspective in treating comorbid disorders draws on a tradition that

focuses on common cognitive behavioural techniques (Wright, Basco & Thase, 2005), and a

formulation-based approach to CBT (Persons, 1989; Tarrier, 2006).

4.0 Method.

4.1 The samples.

The papers in this thesis are based on four samples. Below is a closer description of these patient samples.

4.1.1 Sample 1.

49 participants were referred from psychologists and psychiatrists working at the psychiatric in-patient clinics or the psychiatric outpatient clinics at the St. Olav Psychiatric University Hospital in Trondheim. Patients with psychosis, substance abuse, suicidal behaviour and cluster A or cluster B personality disorder were excluded. Three patients were excluded because they fulfilled these criteria, leaving 46 remaining participants. All of the participants were assessed with the Structured Clinical Interview for the DSM-IV on axis I and axis II. Thirty two subjects completed eight weeks of therapy. There were 22 % males, and 78 % females in this sample, with an age range from 20 to 60 (M = 38.12, SD = 10.21). The sample that completed therapy fulfilled the criteria of 73 diagnoses based on the SCID- interview, with sixty four diagnoses on Axis I, and nine diagnoses on Axis II. The most common diagnoses were anxiety disorders, followed by depressive disorders. Sixty four percent used psychopharmacological medication in at least six months before they started therapy. See table 1 for further description of the sample.

Table 1: The patients' age, sex, and diagnoses in sample 1.

	Waiting list controls (n=17)	CBGT (n=15)	Total (N=32)
Age (Mean, SD)	38.82 (11.57)	37.33(8.76)	38.12 (10.21)
Sex (n)			
Females	14	11	25
Males	3	4	7
Diagnoses (n)			
Anxiety disorders	28	17	45
Depressive disorders	9	7	16
Eating disorders	1	2	3
Cluster C personality disorders	6	3	9
Total Axis I	38	26	64
Total Axis II	6	3	9
Total number of diagnoses	44	29	73

Note: Each patient could receive more than one diagnosis.

4.1.2 Sample 2.

19 participants were included from psychiatric in-patient clinics or psychiatric outpatient clinics at the St. Olav Psychiatric University Hospital in Trondheim. Two participants dropped out of therapy. A sample of 17 completed the eight weeks of therapy. Eighty percent of the sample were males, and twenty percent females, with an age range from 20 to 47 (M =32.6, SD = 7.99). Diagnostic assessments were made by an experienced psychiatrist and an experienced psychologist. Eighty percent of the sample had a diagnosis of schizophrenia, and the remaining 20 percent had a diagnosis of schizoaffective disorder. All of the participants received antipsychotic medication during the study and at follow-up. There was no change in

prescribed dosage or type of medication during the trial or in the follow up period. See table 2

for a further description of the sample.

Table 2: The patients' age, sex, and diagnoses in sample 2.

	CBT-group (n=17)
Age (Mean, SD)	32.6 (7.99)
Sex (%)	
Females	20
Males	80
Diagnoses ICD-10 (%)	
Schizophrenia	80
Schizo-affective disorder	20

4.1.3 Sample 3.

Eighty-two patients from the nine treatment centres in Norway participated in the study.

Sixty-three patients (77%) completed the treatment. There was an age range from 17 to 54

(M= 29.90, SD = 7,88). The samples consisted of sixty eight percent males and thirty-two

percent females. The most common disorders including a comorbid substance problem were

psychotic disorders (schizophrenia and schizoaffective disorders), and thereafter personality

disorders. Diagnoses were set by experienced clinicians at the different centres participating

in this study. See table 3 for a further description of the sample.

Table 3. The patients' age, sex, and diagnoses in sample 3

	The Better Life Group (n=63)
Age (Mean, SD)	27.90 (7.88)
Sex (n, %)	
Females	20 (32%)
Males	43 (68%)
Diagnoses (n, %)	
[*] Psychotic disorder	26 (41.3%)
Non-psychotic disorder	12 (19%)
Personality disorder	19 (30.2%)
Unspecified mental disorder	6 (9.5%)

[*] Included: Schizophrenia and schizoaffective disorder

4.1.4 Sample 4

A male with the diagnosis of paranoid schizophrenia (N=1), at the age of twenty-five, with a primary diagnosis of paranoid schizophrenia, which at the time of assessment also fulfilled a diagnosis of a substance abuse and a moderate depressive episode.

4.2 Instruments

Different instruments were used in this thesis including self-reports, interviews, and clinicians' ratings. These measures were:

4.2.1 Self reports: Symptoms.

SCL-90-R. Symptom Checklist List -90-Revised (Derogatis, 1983) is a 90 item self report, designed to screen for a broad range of psychological problems and distress during a

one-week period prior to administration. Each of the 90 items is rated on a 5-point scale of distress ranging from none to extreme. In addition to a total score (General Symptom Index), the SCl-90- R yields scores on nine subscales. The SCL-90-R has evidenced good psychometric properties (Bech, Allerup, Maier, Albus, Lavori & Ayuso, 1992; Derogatis & Cleary, 1977; Lipman, Covi & Shapiro, 1979*).*

BDI. The Beck Depression Inventory (Beck, Rush, Shaw, & Emery, 1979) is a 21 item self-report inventory, which has been shown extensively to be a reliable and valid measure of syndrome depression severity in both clinical and non-clinical populations (Beck, Steer, & Garbin, 1988)

BAI. The Beck Anxiety Inventory (Beck, Epstein, Brown & Steer, 1988) is a 21 item self-report instrument that measures anxiety severity for the past week, including the day of completion. The BAI is established as a reliable and valid measure, and it is recommended as a companion instrument to the BDI, particularly for individuals with comorbid depression and anxiety (Beck & Steer, 1993a).

BHS. The Beck Hopelessness Scale (Beck, Weissman, Lester, & Trexler, 1974) is a 20-item self report questionnaire of pessimism and hopelessness. Each item is rated as true or false and scores can vary from 0 to 20. Internal consistency of items is satisfactory (Cronbach's alpha =.88) (Steed, 2001).

SMAST. The Short Michigan Alcoholism Screening Test (Selzer, Vonokur, van Roijen, 1975) is a 13-item list of common signs and symptoms of alcoholism. The subject indicates whether he/she has experienced each symptom of problem drinking during his or her

lifetime. Item scores are equally weighted and added together to form a total score. Higher

scores are suggestive of greater alcohol-related problems. The SMAST has been reasonably

well validated as a measure of alcoholism (Selzer et al., 1975; Harburg et al., 1988). The

SMAST has yielded adequate reliability estimates with a mean coefficient alpha of 0.93 for

combined samples of alcoholics and non-alcoholics (Selzer et al., 1975)

DAST. Drug Abuse Screening Test- 20 (Skinner 1982) comprises 20 items assessing

drug use over the past 12 months, which it yields a quantitative index of problems associated

with drug use. Good validity and reliability values have been reported for the DAST (Cocco

& Carrey, 1998; Skinner, 1982; Skinner & Goldberg 1986)

4.2.2 Self reports: Cognitive style and personality traits.

RSE. The Rosenberg Self Esteem Scale (Rosenberg, 1965) consists of 10 items

assessing global self esteem, with a range in score from 10 to 40. Previous studies have

reported acceptable to high reliabilities for the RSE ranging from .72 to .88 (Gray-Little,

Williams, & Hancock, 1997).

SAS. The Sociotropy-Autonomy Scale (Beck, Epstein, Harrison & Emery, 1983) is a

60 item self-report inventory, which measures two stable, independent dimensions of

cognitive traits called sociotropy and autonomy. Thirty items assess sociotropy and 30 items

assess autonomy. Sociotropy refers to dependent traits, characterized by an intense need for

love, approval and being esteemed by others. Autonomy is defined as an excessive personal

demand for accomplishment and freedom from control by others. Test-retest reliability has

been reported to be overall high for both scales (Robins & Block, 1988)

IIP-64. Inventory of Interpersonal Problems 64- Circumplex (Alden, Wiggins &

Pincus, 1990) is a self-report inventory designed to measure interpersonal problems. The IIP–

64-C has eight scales that form a circumplex of interpersonal problems around the octants of

dominance and nurturance. This measure shows strong convergence between self-, therapist-,

and peer-rating profiles. It discriminates between subgroups of depressed and of socially

anxious clients, and has strong test–retest reliability (total r = .98; average subscale r = .81),

and acceptable to good alpha coefficients (.72–. 85) (Horowitz, Rosenberg, Baer, Ureno, &

Villasenor, 1988; Kachin, Newman, & Pincus, 2001)

YSQ. The Young Schema Questionnaire (Young, 1994) is a 205-item questionnaire,

developed to measure 16 schemas. The items are answered on a six point Likert Scale,

ranging from "almost never true for me" to "almost always true for me". In addition to a total

score, you can also calculate scores on the 16 schemas. The YSQ has demonstrated good

levels of psychometric properties and clinical utility (Schmidt, Joiner, Young & Telch, 1995;

Lee, Taylor & Dunn, 1999; Hoffart et al, 2005)

YSQ-SF. The Young Schema Questionnaire-Short Form (Young, 1994) is a 75-item

questionnaire, developed to measure 15 schemas. The items are answered on a six point

Likert Scale, ranging from "almost never true on me" to "almost always true on me". In

addition to a total score, it is possible to calculate scores on the 15 schemas. The YSQ has

reported alpha coefficients for the YSQ- SF subscales ranging from .71 to .93. (Glaser,

Campbell, Calhoun, Bates, & Petrocelli, 2002)

MCMI-III. The MCMI-III (Millon, 1994) includes personality disorder scales and scales for more prevalent Axis I disorders reflecting Millon's multiaxial model (Millon & Davis, 1997). It consists of 24 clinical scales, as well as three modifier scales. Since its introduction in 1977, the MCMI has become one of the most widely used and researched clinical assessment instruments.

4.2.3 Clinical interviews.

SCID I. The Structured Clinical Interview for the DSM-IV on axis I (First, Spitzer, Gibbon & Williams, 1995) is a structured interview developed to assess DSM-IV diagnoses most commonly seen by clinicians, and includes the diagnostic criteria for these disorders with corresponding interview questions. The SCID-I is divided into six self-contained modules that can be administered in sequence: Mood episodes; psychotic symptoms; psychotic disorders; mood disorders; substance use disorders; and anxiety, adjustment, and other disorders.

SCID II. The Structured Clinical Interview for DSM-IV Axis II Personality Disorders (First, Spitzer, Gibbon, Williams & Benjamin 1995) is a structured interview developed to assess the 10 DSM-IV Axis II personality disorders as well as depressive personality disorder, passive-aggressive personality disorder, and personality disorder not otherwise specified.

CDSS. The Calgary Depression Scale for Schizophrenia (Addington, Addington, & Schizzel, 1990) is a structured interview developed to assess symptoms of major depressive disorders in patients with schizophrenia. The time frame of interest is typically the two weeks before the interview. Each item includes interview questions and descriptive anchors for scoring. The CDSS consists of nine items: Depressed mood, hopelessness, self-deprecation,

guilty ideas of reference, pathological guilt, morning depression, early awakening, suicide, and observed depression. Items are scored 0 = absent, 1 = mild, 2 = moderate, and 3 = severe. Total scores range from 0 to 27. A total score of 5 identifies individuals at high risk for a major depressive disorder. The items on the CDSS are all typical depressive symptoms and do not appear to overlap with the negative symptoms of schizophrenia (Addington, Addington, & Maticka-Tyndale, 1992). Reliability of the CDSS has been reported in several studies (Addington et al., 1992; Lancon, Auquier, Reine, Toumi, & Addington, 1999; Mueller et al., 1999; Kontaxakis et al., 2000). The internal consistency appears to be good for both inpatients and outpatients, with Cronbach's alphas ranging from 0.7 to 0.9 (Addington et al., 1990).

GAF. Psychosocial functioning was assessed by the Global Assessment of Functioning Scale (American Psychiatric Association, 1994). The GAF is a single measure ranging from 0-100, with higher scores represents better functioning. The instrument has shown to be reliable and valid in a sample of severely mentally ill (Jones, Thornicroft, Dunn, & Coffey, 1995).

4.2.4 Clinical ratings.

AUS and DUS. The Alcohol Use Scale (Mueser et al, 1995) and the Drug Use Scale (Mueser et al, 1995) are 5-point scales based on the DSM- IV criteria for alcohol and drug use disorders, rated for the worst period over the past 6 months (1 = no use, 2 = use with impairment, 3 = abuse, 4 = dependence, and 5 = dependence with institutionalization).

SATS-R. The Substance Abuse Treatment Scale- Revised version, (Mueser, Noordsy, Drake & Fox, 2003) is a measure of the patient's motivation to change, and progress in changing his or her substance use behaviour. The score is selected from one of the following 8

stages of treatment: Pre-engagement (1), engagement (2), early persuasion (3), late persuasion (4), early active treatment (5), late active treatment (6), relapse prevention (7), and in remission or recovery (8). The SATS-R is based on the patient's pattern of substance use over the last 6-month period, with an emphasis on the patient's most recent substance abuse and involvement in treatment.

DDFS. The Dual Disorder Fidelity Scale (DDFS) (Mueser, Fox, Bond, Salyers, Yamamoto & Williams, 2003) is a 20-item scale assessing the adequacy of specific evidence-based practices for treatment of individuals with dual disorders. The scale is organized around seven principles: integration of assessments and treatment of both disorders, comprehensiveness of services, assertive outreach services, applying a harm-reduction model, long-term treatment, stage-wise and motivation-based treatment, and using multiple treatment modalities (e.g., individual- and group psychosocial strategies, pharmacological strategies). Ratings is based upon a review of 10 randomly selected treatment records of patients with dual disorders and an interview of a representative sample of clinicians at each centre. Each of the 20 items in the DDFS is rated on a 5-point scale (1=not implemented to 5=fully implemented). Each item-score has descriptive anchors based on clinical expert sources and research. The total score, which is the sum of all 20 items, reflects the degree of implementation of evidence based treatment, namely: Score 24-49; not implemented 50-78; moderately implemented 63-100; fully implemented.

4.3 Ethical considerations

In all of the studies, patients gave their informed consent to participate in the trials, and the studies in this thesis have been approved by the Research Ethics Committee, Midt-Norge.

5.0 Overview of the studies and the main results.

5.1 Paper I. A randomized trial of cognitive group therapy vs. waiting list for patients with comorbid psychiatric disorders: Effect of cognitive group therapy after treatment and six and twelve months follow up.

In paper I we wanted to investigate if CBGT would be superior to a waiting list in the treatment of comorbid psychiatric disorders. The study included 46 patients from the psychiatric inpatient's clinic and the psychiatric outpatient's clinic at the St. Olav University Psychiatric Hospital in Trondheim. The patients were assessed with the Structured Clinical Interview on Axis I and Axis II (SCID I + II). After the diagnostic interview they were randomly assigned, either to a cognitive behavioural group therapy (CBGT) program or a waiting list (WL). The aims of the study were to assess the effectiveness of cognitive behaviour therapy in a group format in a sample of comorbid, non- psychotic psychiatric disorders, and also to assess if the effectiveness was upheld at six and twelve months follow up. The participants who were in to the CBGT- condition received 8 weeks of treatment, with two sessions each week, in all a total of sixteen sessions of ninety minutes duration. The participants who were randomly assigned in to the WL- condition waited eight weeks before they entered treatment and went through the same program as the CBGT- condition. Thirty-two participants completed the cognitive-behaviour group therapy.

The results show that an 8-week program of CBGT performed better than the WL with respect to symptom relief at post-treatment, especially for anxiety and depression. On the other inventories measuring interpersonal stress, sosiotropy/dependency, and maladaptive schemas, no significant differences were found between these two conditions at post-treatment. Depression and general psychiatric distress showed a significant reduction from

pre-treatment to post-treatment for all the subjects that completed CBGT. Follow up results showed a strong significant reduction on the general symptom level at both six and twelve months follow up. Interpersonal distress, maladaptive schemas, and sosiotropy/dependency showed no significant differences from pre to post-treatment for the patients who completed the program, but interpersonal problems, maladaptive schemas, and sosiotropy/dependency were significantly lower after the six months follow up, compared to pre-treatment. The sosiotropy/dependency score also showed a significant reduction at twelve months follow up. In sum, the study showed that an eight weeks program of CBGT, seems to have a favourable effect both on short-term and long-term results. The study also indicates that cognitive-behaviour therapy can be conducted in a group format aimed at treating patients suffering from comorbid states of anxiety and depression.

5.2 Paper II. Cognitive behavioural group treatment of depression in patients with psychotic disorders.

In paper II we wanted to investigate if CBGT could be effective in treating patients with comorbid depressive and psychotic disorders. The study included 19 patients who were included from psychiatric in-patient clinics or psychiatric out-patient clinics at the St. Olav University Psychiatric Hospital in Trondheim. The inclusion criteria for participation in the study were the following: First, having a diagnosis of schizophrenia or schizoaffective disorders, second a minimum duration of one year in having these diagnoses, and thirdly a comorbid depression in addition to the diagnosis of schizophrenia or schizoaffective disorders. Diagnostic assessments related to the schizophrenia and schizoaffective disorders were made by an experienced psychiatrist and an experienced psychologist. The primary target of the study was to assess if CBGT would reduce the depressive symptoms, and secondly to assess whether CBGT could reduce hopelessness and increase self-esteem and

psychosocial functioning, and thirdly to examine any changes of personality traits and maladaptive schemas connected with the comorbidity in depression and psychosis. In all 17 patients completed the eight weeks of CBGT. The group members received eight weeks of treatment, with two sessions each week, in all 16 sessions of 90 minutes duration.

The results showed that an eight week program of CBGT had a significant effect on treating depression in schizophrenic disorders, both at post-treatment and at six months follow up, with no incidents of relapse. Also, the psychosocial functioning measured by GAF showed a significant increase from pre-treatment to the six months follow up for the participants, but there were no significant changes in hopelessness or self-esteem. Three of the scales on the MCMI-III regarding clinical personality patterns showed significant changes at post treatment, with a significant decrease in the scales of Compulsive and Negativistic, and a significant increase on the Narcissistic scale. At the six months follow up there were no significant changes on the MCMI-III scales. Results regarding personality-traits and maladaptive schemas connected with the co-morbidity in depression and psychosis, revealed a high prevalence of avoidance, dependency, depressive thinking, and anxiety among the patients in the sample. The results from the YSQ also supported this trend. To summarize the findings, the study showed that CBGT had been effective in treating depressive symptoms and improving psychosocial functioning, but had been less effective in reducing hopelessness, increasing self-esteem, changing maladaptive schemas, or decreasing psychotic symptoms within the group participants.

5.3 Paper III. The Better Life Program: Effects of group skills training for persons with severe mental illness and substance disorders.

In paper III the primary goals were to evaluate the feasibility of the Better Life program, to examine its effects on substance use and mental health outcomes, and to explore individual predictors of outcome. Patients with dual disorders at nine centres in Norway were recruited to participate in a study related to The Better Life program. The inclusion criteria for participation in the Better Life group program were: Minimum 18 years of age, having a severe mental disorder with accompanying functional impairment, and alcohol or drug misuse (i. e., substance use resulting in problems related to self-care, relationships, legal or health problems or exacerbation of their mental illness). Eighty-two patients participated in the study. The mean number of scheduled sessions was thirty-seven, and the mean participation rate was thirty-two sessions.

The results showed that the Better Life program was associated with a low drop-out rate and that participants significantly improved in drug and alcohol use severity as well as their stage of substance abuse treatment. Clinicians' ratings of alcohol and drug abuse/dependence on the AUS and DUS indicated that about one half of the patients with alcohol or drug use disorders were in stable remission at the end of the program. Furthermore, patients' self-ratings of changes in substance use on the DAST and SMAST corroborated the clinicians' ratings. In addition, program participants demonstrated significant improvements in global psychosocial functioning from pre-treatment to post-treatment. The positive results associated with the participation in the Better Life program are in line with several other group interventions for patients with dual disorders.

5.4 Paper IV. Behavioural experiments in the treatment of paranoid schizophrenia: A single case study.

In paper IV we wanted to investigate if behavioural experiments could be used as a powerful tool to change the delusional thinking in a patient with a diagnosis of paranoid schizophrenia. In addition to highlighting the use of behavioural experiments as means to change the patient's delusional thinking, the paper also tries to demonstrate how techniques of cognitive behavioural therapy can be adapted in treating patients with psychotic symptoms. Finally, some suggestions for research to explore the use of behavioural experiments in cognitive therapy of psychosis are suggested.

The results demonstrated that CBT, and in particular behavioural experiments, can be used with good results in treating a patient suffering from comorbid symptoms of substance abuse, depression, and psychotic symptoms. Although behaviour experiments are considered as a really important part of CBT for psychosis, there has been little attention related to research on how to best utilize it in CBT for psychotic symptoms. The paper therefore suggest that future research to a larger extent should examine behavioural interventions in contrast to more pure cognitive interventions, to find out what makes CBT in psychosis effective.

6.0 Summary of the thesis

6.1 Main results from the studies

Paper I shows that CBT has a favourable effect on both short- and long- term results, and also indicates that CBT in a group format could be used to treat patients suffering from comorbid states of anxiety and depression. Paper II shows that an eight-week program of cognitive behaviour group therapy had a significant effect on treating depression in schizophrenic disorders, both at post treatment and at six months follow up, with no incidents of relapse. Although there was a decrease in depression, the CBGT had no effect on the psychotic symptoms. This study indicates that CBGT could be effective in treating comorbid depression in schizophrenic disorders. Paper III shows that a group intervention focusing on motivational enhancement interventions and skills training had an effect on both substance abuse and mental health outcomes. This study indicates that a CBGT focusing on motivational interviewing and skills training is a promising psychological intervention with people suffering from substance abuse and psychiatric disorders. Paper IV demonstrates that CBT could be effective in treating a patient suffering with comorbid disorders of depression, substance abuse and delusional disorders. This study indicates that especially behavioural experiments could be an effective element in CBT for psychosis.

Overall, the studies indicate that both individual and group CBT may bed adapted effectively to treat complex comorbid disorders, and have acceptable effects. Other main results could be summarized as follows:

- Results from the studies indicate that CBGT fall in line with other studies showing that cognitive group therapy is effective in treating psychiatric problems.

- Results from the studies illustrate that CBT could be useful in treating patients with psychosis.

- CBT seems to make a significant clinical impact that improves symptoms and functioning in the presence of multiple comorbid conditions.

- All the studies appear to support the idea that different patterns of psychiatric comorbidity could be treated effectively using a more general trans-diagnostic CBT-approach.

6.2 Limitations of the studies

There are some potential limitations of the studies. These limitations will briefly be mentioned in the following:

- Two of the studies (Paper II & III) did not include a no-treatment control group, which could influence the internal validity of these results. Without a control group it is difficult to determine whether the observed change is related to the treatment effect of CBT, or other treatments the patients were receiving, spontaneous recovery, or the natural course of the patients' disorder. The use of randomized controlled trials is needed to address these questions in a proper way.

- Two of the studies (Paper II & Paper III) had a lack of a blind assessment which could act as a threat to the external validity of the results. It is essential to keep the rater "blind" and independent, and if this condition is not maintained, scoring can be affected by observer or rater expectations. It is possible that the clinicians' judgement of improvement was influenced by their expectation and beliefs regarding the effect of CBT.

- In two of the studies (Paper II & III) there was a lack of a diagnostic structured interview, which could be a threat to the internal validity of the results. It is important to ensure that the patients who participate in a study fulfil the criteria of a diagnosis which serve as an inclusion to a treatment study. The best way to ensure these demands is the use of a structured diagnostic interview.

- Finally, there could be a threat to validity related to the use of self-reports in the studies. One phenomenon often observed in clinical trials is called the *hello-goodbye effect,* where patients tend to exaggerate their symptoms at the beginning of therapy and to minimize the symptoms at the end, probably to please the therapist or obeying demand characteristics related to the study. Nordahl (1999) also reported that self-report questionnaires seem to be influenced by a patient's actual mood, which could affect the psychometric properties in some of the self-reports used in the studies.

Despite these limitations related to control groups, the lack of blind assessment, structured diagnostic assessment, and the use of self-reports, the results of the studies in this thesis have strong ecological validity. Patients treated in the studies are typical and representative for patients treated in clinical practice, where the occurrence of comorbidity and heterogeneity of disorders is a common problem. Many modern treatment outcome studies typically exclude or ignore comorbidity. Thus, the studies included in this thesis that include comorbid patients, will maybe reflect the "real-world" conditions for clinicians in a better way than randomised controlled trials for single disorders.

6.3 Implications of the findings

The key to managing comorbid conditions seem to be to recognize this complicated problem by trying to assess the patient's mental health problems as broadly as possible. Further, it is important to use the clinical information to determine a treatment plan that best identify the

multiple disorders and maximize potential changes in all of them. The final point in treating comorbid problems would be to choose a type of treatment that could be tailored and individualized to the patient's comorbid conditions. Since CBT place an emphasis on an individualized case formulation of a patient's complex problems, CBT seem to best adapted to treat these comorbid psychiatric conditions. However, to use CBT in treating complex comorbid disorders, some basic principles are fundamental, and these have been emphasized in the studies:

I.   The first component is to learn to the patients to identify and restructure their negative appraisals, which leads to a more objective appraisal of distressing situations.

II. The second component focuses on the exposure to distressing situations, and shows that approaching instead of avoidance could lead to change in cognitions and distress.

III. The third component is that successful treatment involves changing usual behaviours that undermine coping and functioning. The implementation of new coping skills and behavioural experiments could be useful in facilitating such change

The epidemiological data on comorbidity seem to challenge the meaningfulness of the classic psychotherapeutic research paradigm, where researchers try to identify a group of individuals who share one common diagnosis and then examine them on variables hypothised to be relevant to the aetiology, course, or treatment of the disorder. Research has shown us rather thoroughly that pure, uncomplicated cases are highly uncommon, and maybe to study such patient populations does not represent a good pathway of therapy research. Even more important is the fact that even if non-comorbid cases can be identified, they are likely to be atypical and less representative of the overall population of individuals with the disorder. To produce ecologically valid psychotherapeutic outcome studies, there is maybe a need of a

shift from emphasizing controlled psychotherapy outcome studies on single disorders, to include more naturalistic psychotherapy outcome studies, which may heighten ecological validity, and reduce the gap between psychotherapy research and clinical practice (Werbart, 1989, Birchwood 2006).

The research on the effect of CBT for comorbid disorders is still in its infancy, and the tailoring of CBT to different types of comorbid disorders will be a major area of interest in the coming years. The evolution of metacognitive therapy which emphasize shared cognitive processes in psychology across disorders, is an expansion of CBT in such a direction (Wells, 2000; Wells. 2007)

6.4 Main points of the thesis.

- ❖ Comorbidity is the rule than the exception. Epidemiologic studies suggest that psychiatric comorbidity is widespread and common, and most patients could be expected to show comorbid patterns of psychopathology.
- ❖ Comorbidity should be assessed by structured diagnostical interviews.
- ❖ In most studies of the effect of psychotherapy, patients with significant levels of comorbidity are excluded. Results from these studies do not give indications for treatment of patients with comorbid disorders.
- ❖ There are rather few results on the effect of CBT on complex and comorbid psychiatric disorders.
- ❖ A trans-diagnostic case formulation based CBT-approach to complex and comorbid psychiatric disorders seems to be useful, both in group and single-case formats.

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8.0 Papers I-V

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Running head: Behavioural Experiments in Paranoid Schizophrenia

Title: Behavioural Experiments in Cognitive Behavioural Therapy for Paranoid

Schizophrenia: A Single Case Study

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Abstract

Since the first description of cognitive therapy of paranoid delusions, the empirical support for cognitive behavioural therapy in treating psychotic symptoms has been widely established. The aim of the present case study is to show how the behavioural experiment can be used as a powerful tool to change delusional thinking in a patient with paranoid schizophrenia. In addition to highlighting the use of behavioural experiments to change the patient's delusion, we will illustrate various cognitive techniques that can be adapted to treat patients with psychotic symptoms.

KEYWORDS: Cognitive behavioural therapy, psychosis, delusions, auditory hallucinations, behavioural experiments.

Introduction

Since the first description of cognitive therapy of paranoid delusions, (Beck, 1952), the empirical support for using cognitive behavioural therapy (CBT) to treat psychotic symptoms has been widely established (Gould, Mueser, Bolton, Mays & Goff, 2001; Rector & Beck, 2001; Dickerson, 2004; Gaudiano, 2005; Rathod & Turkington, 2005; Turkington, Kingdon & Weiden, 2006). Previously psychosis was considered a biological condition insusceptible to psychological interventions, however, more recent research has shown that positive symptoms are on a continuum with normality and therefore may be susceptible to the same cognitive behavioural therapy techniques used to treat anxiety and depression (Kuipers et al., 2006; Bentall 2007). The cognitive model of psychosis conceptualises a combination of factors that shape and maintain positive symptoms such as delusions and auditory hallucinations (Garety, Kuipers, Fowler, Freeman & Bebbington, 2001), where reasoning and attributional biases may play a particular role in symptom formation and the maintenance of these symptoms (Freeman & Garety, 2004; Bentall 2003; Frith 1992).

The primary goals when using CBT to treat psychosis are to teach the patient to identify and monitor their thoughts and assumptions in specific situations, and to evaluate and correct these thoughts and assumptions against objective external evidence and actual circumstances. Challenging delusional thinking may stimulate the patient to come up with more functional alternatives (different phases of CBT in psychosis see figure 1).

Figure 1 approximately here

Delusional beliefs and auditory hallucinations are based upon thoughts and assumptions that the patient believes to be true and real, and they are held with great intensity and preoccupation. These beliefs cause distortions in the processing of new information, which also maintains the patient's delusional beliefs and auditory hallucinations. Evidence to challenge delusional cognitions may be gathered in a variety of ways (Chadwick, Birchwood, & Trower, 1996; Fowler, Garety & Kuipers 1995; Nelson 2005; Morrison et al., 2002, 2004, Kingdon & Turkington, 2005). Behavioural experiments and verbal strategies such as the use of automatic thought records and guided discovery are strategies that can be used to gather this evidence. The three main purposes of using behavioural experiments to treat psychotic symptoms are to formulate a testable and specific assumption, to test the reliability of this assumption, and to construct new and more functional assumptions and beliefs (Rouf et al., 2005). The designs commonly used in behavioural experiments are formal hypothesis testing, (if so …what will happen ...), or investigative or discovery-oriented methods (What will happen if…?). Behavioural experiments can therefore be used to test the reality in the patient's distorted interpretations. Reality testing includes setting up situations for the specific purpose of producing evidence that is directly relevant to the delusional belief, or to find evidence for an alternative belief (Nelson, 2005). Provided that these reality tests are properly set up, the evidence against a delusion or auditory hallucination can be very persuasive. In using reality tests, it is important to build alternative explanations that can counterbalance the patient's delusional beliefs, and thereafter use evidence to support these alternative explanations. When these possible alternatives and supporting evidence are well prepared, the patient's delusional interpretations can be tested (Close & Schuller, 2004)

The aim of the present case study is to show how the behavioural experiment can be used as a powerful tool to change delusional thinking in a patient with paranoid schizophrenia. In addition to highlighting the use of reality testing as a means to change the

patient's delusional interpretations, we will also try to show how techniques of cognitive-behavioural therapy can be adapted to treat patients with psychotic symptoms.

Case presentation and symptoms

In order to protect patient anonymity, a number of essential details have been altered in this case study. The patient gave his consent in an early phase of the therapy, that his case story could be used as a presentation. Tony is 25 years old. He is a single, Caucasian male, who was hospitalised with a diagnosis of paranoid schizophrenia. The patient has a younger brother, and his parents divorced when he was a child. The patient and his brother were taken into care due to gross neglect by their parents and put in to a foster home when he was seven years old. During his childhood years he had regular contact with his biological mother, but did not meet his biological father again until the age of seventeen. Shortly after the death of his father, Tony started using drugs (cannabis, amphetamine and ecstasy) and started to drink heavily. Despite Tony's apparent heavy substance abuse he completed college with satisfactory results. After graduation from college, he enrolled in some courses at the university, but did not attend any of the lectures and failed all of his exams. During these years he developed a severe substance abuse. The last year before hospitalisation Tony lived on unemployment benefits, but also dealt drugs to support his drug use. He was never arrested for dealing drugs.

At the age of twenty-two he became psychotic. At the time of admission to the psychiatric hospital he was experiencing psychotic symptoms in the form of auditory hallucinations and delusions. His voices told him that he should be killed when the right opportunity presented itself, and also that he was kept under surveillance by the police. His appraisal of the voices was that they were policemen haunting him because of his history of selling drugs. He was certain that these policemen had put a contract out to kill him. His

delusional ideas were of a paranoid form. Tony mentioned several persons by name who worked in the anti-narcotic squad that he thought were involved in this conspiracy to kill him. Tony believed that these policemen were using various methods of surveillance to keep him under their control. Among his delusional ideas was that he was being used for an experiment. He believed receivers had been implanted in his head, and that the policemen were using radio beams to over-stimulate his brain, which caused him pain. He was certain that his head would explode if this "torture" continued. He had strong delusional convictions about the damage to his brain that was caused by the radio beams. Tony consistently insisted that more physical tests ought to be taken because of his conviction that something was physically very wrong with him. There was a link between the auditory hallucinations Tony experienced and his delusional ideas of radio beams. He was convinced that the voices he heard were transferred by the policemen's radio transmitter to the implants in his head, which was his proof that the voices were inflicted on him by an outside agency.

After Tony had been at the acute ward for three weeks he was transferred to a ward for young people suffering of psychotic disorders. Though he had not been using narcotics since hospitalisation he still experienced auditory hallucinations, had delusional ideas, and responded poorly to the antipsychotic drugs (olanzapine) he was administered. The therapist undertook a structured diagnostic interview (SCID I and II). Based on the SCID-interview and information from the patient, Tony was diagnosed with a paranoid schizophrenia. The patient also met SCID I criteria for diagnoses of substance abuse and a current moderate depressive episode. The comorbid symptoms of substance abuse and depression are rather common in patients suffering from psychotic disorders (Michail & Birchwood, 2007; Kavanagh & Mueser, 2007), but differentiating schizophrenia from schizoaffective disorders and mood disorders with psychotic features can sometimes be particularly difficult (Frances & Ross, 2005). Tony's psychotic symptoms in the form of bizarre delusions had been present

for at least six months before he was admitted to the psychiatric hospital, and his mood symptoms were not a significant part of Tony's symptom presentation. The depressive symptoms that the patient experienced could probably best be understood as a psychological reaction to his delusional ideas and auditory hallucinations. Based on this information the occurrence of a mood disorder with psychotic features and a schizoaffective disorder could be ruled out. The psychotic symptoms Tony experienced persisted even though he had stopped drinking alcohol and smoking cannabis the last month before the assessment took place. This indicates that the psychotic symptoms were probably not a direct physiological result from the substances he had been using. In addition, the symptoms he experienced could not be explained merely by intoxication or withdrawal.

Case formulation

The assessment of Tony suggested that the delusions and auditory hallucinations he experienced were mixed with his symptoms of depression and substance abuse. Morrison's model of development and maintenance of voices was used to make a formulation of Tony's auditory hallucinations (Morrison, 2001). In his model Morrison suggests that different internal or external triggers result in the patient hearing voices, and that the voices may be interpreted as threatening for the voice hearer. The catastrophic interpretations of the voices result in increased negative mood and physiological arousal, and elicit safety-seeking behaviours and hypervigilance because the patient feels threatened by the voices. This behaviour can increase the future frequency of auditory hallucinations, and also prevent the disconfirmation of the threat presented by the voices (therefore maintaining distress and anxiety).

In Tony's case, the substance abuse and anxiety (thoughts of threat) seemed to be triggers for hearing voices. The voices said that he would be harmed and that a contract had

been put on his head. Tony believed that these voices belonged to a policeman who he once knew, and who he thought had the power to make this happen. The voices triggered feelings of depression and anxiety, and he tried to help himself by using substances and by staying home to avoid potential attacks. This form of safety behaviour maintained the auditory hallucinations, and probably increased the frequency of the auditory hallucinations. The pattern of Tony's coping behaviour resulted in a vicious circle: When the intensity of negative emotions (depression and anxiety) increased, Tony craved large amounts of alcohol, amphetamine, and cannabis, which in turn increased his auditory hallucinations. This pattern also illustrates what is known as the stress-vulnerability hypothesis of schizophrenia, where stress combined with a psychological vulnerability produce the symptoms characteristic of psychosis (Zubin & Spring, 1977; Neuctherline & Dawson, 1984).

Freeman, Garety, Kuipers, Fowler & Bebbington (2002) cognitive model of persecutory delusions also guided Tony's case formulation. Freeman et al. (2002) hypothesise in their model that anxiety and depression play a direct role in symptom development and the maintenance of persecutory delusions. They suggest that emergence of psychotic symptoms depends upon an interaction between vulnerability and stress (such as drug use). People who experience symptoms, such as voices, will search for a reason for their anomalous experiences by drawing upon pre-existing beliefs about the self, others, and the world. The delusions will be maintained because disconformatory evidence could be discarded (e.g. by the use of safety behaviours) and confirmatory evidence for the delusions is obtained (e.g. confirmation bias, attention bias, and the tendency to jump to conclusions).

The powerful, threatening nature of his voices and the somatic torment he experienced were Tony's evidence for his delusional ideas. He experienced his life situation as hopeless, because he believed that no one could help him against his external enemies, which made him feel depressed. He started to withdraw from others because he was unable to sort out who was

not a threat to him. The social isolation lead to a viscous circle of depression and substance abuse which seemed to increase his delusional beliefs and auditory hallucinations (see figure 2 for a case formulation).

Figure 2- approximately here

Based on the current case formulation it was probable that many of the symptoms Tony experienced were based on his appraisal of his voices (persecutory delusions, depression, and substance abuse), and that the main focus of treatment had to be directed toward working with these catastrophic misinterpretations. In order to work with Tony's auditory hallucinations, it would be important to directly test the content and reality of the voices (Chadwick & Birchwood 1994; Birchwood & Chadwick 1997; Byrne, Birchwood, Trower & Meaden, 2006). In treating Tony's delusions of persecution the goals were to evaluate his evidence for being under surveillance by the police through verbally challenging these ideas and then testing these beliefs by using behavioural experiments. A final goal for the therapy was to construct alternative explanations of his experiences that did not cause emotional distress.

Treatment

The patient was treated by one of the authors, a clinical psychologist (HMN), in an inpatient ward at the university psychiatric hospital. The therapist is a well-trained CBT-therapist with over fifteen years of experience in conducting cognitive behavioural therapy. The patient received a combination of therapy, two sessions a week of CBT over a period of 6 months and medication (Olanzapine, 15 mg). In the first stage of the cognitive behavioural therapy the emphasis was to engage the client and form a therapeutic alliance that would allow for a collaborative approach (Kingdon & Turkington, 2005). The process of normalisation can be used as a therapeutic tool towards forming this alliance, and can also pave the way for a collaborative formulation helping the client to be an active part in his treatment (Dudley et al, 2007). Based on the cognitive model, the experience of distress due to psychotic symptoms becomes understandable. Studies show that this process is one of the most important components of successful cognitive behavioural therapy for psychosis (Dudley et al, 2007). In this process the therapist discussed in an empathic and genuine manner Tony's psychotic expression, and together they worked on finding an explanation of how these symptoms had been shaped and maintained, based on the cognitive models of auditory hallucinations and persecutory delusions. In this phase, by discussing the patient's symptoms from a normalising stance, a strong therapeutic alliance was formed between Tony and the therapist.

Based on the cognitive case formulation the treatment was to have three focal points. The first focal point was to treat the patient's substance abuse due to the negative impact it would have on the patient's psychiatric symptoms, the second was to challenge the patient's appraisal of the voices, and the third was to find disconfirmatory evidence for Tony's persecutory delusions.

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In the treatment of the patient's substance abuse, techniques from motivational interviewing were implemented. Motivational interviewing is a set of therapeutic strategies designed to help the patient understand the impact of substance abuse on their lives in their own terms (Miller & Rollnick, 2002). Studies using extended cognitive behavioural therapy, which addresses both substance use disorders and serious mental illness tend to have a good outcome on both of the disorders (Barrowclough et al, 2001; Haddock et al; 2003), though gains of an integrated treatment have shown decay over time (Barrowclough et al., 2001). Interventions in this phase of Tony's therapy were focused on showing the discrepancy between Tony's personal goals and his current substance abuse, in an effort to motivate him to reduce his substance abuse behaviour. In a non-confrontational manner and using a collaborative approach, Tony's personal goals were identified and explored. His main goal in life was to either get a job or to continue his disrupted education. The therapist explained to the patient how the viscous circle of the substance abuse behaviour maintained the patient's psychiatric symptoms, and gradually Tony started to see that there was a contradiction between his life goals and his substance use. He understood that if he continued his pattern of drinking and taking drugs to solve his problems, his symptoms would get worse.

The therapist explained to him the cognitive model of relapse in substance abuse. To counter further relapses, the patient's automatic thoughts and permissive beliefs in these high-risk situations for using substances were examined (Beck, Wright, Newman & Liese, 1993). To avoid relapses Tony received psycho-education about urges and cravings and learned how to make use of cognitive-behavioural techniques to fight urges and cravings (Graham et al, 2004). After this phase of motivational interventions, his substance abuse decreased. Although there was a considerable reduction in his consumption of alcohol and drugs, Tony experienced two minor relapses in his substance abuse later in therapy. These occurred when the patient was on leave from his psychiatric ward. On these leaves he chose to smoke

cannabis with some old friends, which increased his psychotic symptoms, though they were not as dramatic as his initial symptoms. To prevent new relapses antecedents and triggers for his substance behaviour in these relapse situations were examined, and coping strategies discussed.

After working through his problems related to his substance abuse, the therapy focus shifted to trying to modify Tony's safety behaviours. These safety behaviours maintained his appraisal of his auditory hallucinations, and prevented disconfirmatory evidence of the persecutory delusions. Due to his paranoid ideation, he felt that both the other patients and the nurses who were working at the ward were a threat to him. Treatment was directed towards making Tony feel safer in his surroundings and to overcome his social isolation, which had become safety behaviour for him (and reinforced his depressive and anxious symptoms). Because Tony spent most of his time in bed, he felt useless and inactive. To fight these negative cycles of inactivity and social isolation, the therapist and Tony scheduled activities. Activity scheduling is a standard cognitive behavioural therapy technique proven to be effective in treating depressive symptoms (Person, Davidson, Tompkins, 2000), and can also be effective in treating psychotic symptoms (Kingdon & Turkington, 2005). Tony and the therapist planned activities he could do during the week: He took part in activities with other people in the ward (for instance making food), and participated in a work-out group in a gym near the hospital together with some of the other patients. He was also motivated by the therapist to leave the ward on occasion to go to the shops and to the nearby town to meet other people. Slowly these interventions made him feel less depressed, although the delusions and the auditory hallucinations he experienced still made him anxious.

Reality testing: using a match-mismatch technique

In addition to his depressive and anxious symptoms, his auditory hallucinations, and his paranoid delusions, the patient also experienced somatic delusions. Tony was especially concerned that his pituitary gland was destroyed because of the radio beams he been exposed to from the police. His assigned homework related to these belief, was to evaluate his own beliefs about his pituitary gland against anatomic information about the pituitary gland. He searched for information in medical textbooks and on the Internet about the pituitary gland, and how it functioned. The therapist asked Socratic questions about comparing the symptoms of a damaged pituary gland with his own symptoms. Although this new knowledge weakened Tony's delusion of being physically harmed, he still continued to believe that he was being exposed to radiation because he still heard voices and continued to suffer from his bodily pain.

Behavioural Experiment: Increase-decrease manoeuvre

In order to protect himself against the radiation, he had put several bottles of water under his bed together with some aluminium foil. These were seen as safety behaviours, because Tony believed the water and the aluminium foil could deflect the radio beams. This delusion was tested out in a behavioural experiment by using an increase-decrease technique. An increase-decrease procedure is based on a simple behavioural experiment, where the first part is based on what will happen if you increase the use of safety behaviours (less beams - weaker voices) and the second is based on what will happen if the safety behaviours are removed (more beams - stronger voices). The behavioural experiment was done over the course of two nights. The first night the patient was instructed to put more bottles of water under his bed to see if this caused him to hear fewer voices. On the second night this procedure was reversed; some of the bottles were removed to see if this resulted in hearing more voices. The patient found out there was no connection between the amount of bottles of

water he had put under his bed and the intensity of his auditory hallucinations. Tony then stopped these safety behaviours because the evidence from the reality test suggested that it did not have any effect on the intensity of his auditory hallucinations.

Behavioural Experiment: Challenging the voices

Tony's auditory hallucinations, however, still caused him a lot of distress. Challenging his appraisals of the voices was the next step. First, the therapist used Socratic questioning to find out more about the content of Tony's voices, so the foundation for a behavioural experiment to challenge the omnipotence and truthfulness of these voices could be laid. Secondly as a behavioural experiment, Tony started to register how often the voices were correct in their predictions about him (e.g.: Why haven't I been harmed or killed yet?", "Do the voices lie to me?", "How often have the voices been correct in their threats to me")? Based on these registrations, Tony began to get doubts regarding how truthful the voices were. He started to see the voices as phoney, which stated things that hadn't come true. Confronting the omnipotence Tony had attributed to his voices using this technique made him feel more empowered, and he started thinking that he had more control than he originally had thought he could have. In line with Morrison's model of auditory hallucinations, Tony experienced that the voices became less distressing. The voice's frequency and intensity was reduced, because he now perceived them as lies trying to disturb him. Tony managed by detaching more and more from the voices to handle the presence of the voices, and discovered that the voices disappeared for a short period over some days, which he found very strange.

Behavioural Experiment: Emotional reasoning

After challenging the patient's interpretation of the power and control of the voices, the cognitive behavioural therapy shifted towards reality testing some of Tony's delusional beliefs (see table 1 for behavioural experiments used in CBT with Tony).

Table 1 approximately here

In a collaborative manner Tony and the therapist set up behavioural experiments to evaluate the validity of his beliefs of being persecuted and under surveillance. Together with the therapist, Tony formulated his beliefs about someone observing him and his level of convictions related to these beliefs. To examine these beliefs further the therapist and Tony checked the parking lot where Tony insisted that he had seen suspicious persons sitting in a car. Tony and the therapist did this behavioural experiment every time Tony saw cars that he found suspicious. When reviewing the behavioural experiments, the role of anxiety in maintaining delusions was discussed, and Tony was able to understand how high levels of anxiety could serve as strong confirmatory evidence even in the absence of external evidence (i.e.. emotional reasoning).

Behavioural Experiments: Radio waves

Further behavioural experiments were set up, and because Tony's delusions were closely connected to his appraisal of the voices, a new behavioural experiment was carried out. Tony was still certain that the voices came from outside his head, and were inflicted upon him by radio beams, though he had begun to understand that they didn't tell him the truth. To help him understand that the voices were not caused by radio beams, the therapist and Tony agreed to test the hypothesis "the voices are externally inflicted". Tony and the therapist agreed that if the voices were caused by radio beams, then he would not hear them if he was

in a room that blocked out all radio beams and mobile phone signals. If Tony still heard the voices in such a room, it would be evidence suggesting that the voices did not come from outside by radio beams, but rather took place inside his head. In trying to test the hypothesis, the therapist and Tony used a cellular phone and a radio and went in search of a room that could not receive signals. After visiting several places in the city, the therapist located a bomb shelter from World War II where they made sure that no signals from the mobile phone or radio-programs could be received. It is noteworthy that Tony seemed to be comforted by being in a shelter where the resistance had hid during the war. After staying in this facility for one hour Tony discovered that he still heard the voices. His response, for several minutes, was confusion and silence. After this behavioural experiment he reported feeling less threatened by the police. The delusions of being persecuted were still there, but the levels of anxiety and depression were clearly diminishing. The results of the behavioural experiments seemed to motivate Tony to continue critically investigating his thoughts and the nature of the voices.

Behavioural Experiment: Setting up a confrontation

Next, the therapist approached Tony's belief that a particular policeman led the persecution of him. This policeman had worked in the drug-squad. Tony believed that because he had sold drugs this policeman hunted him. Tony's delusion was based on an actual person, so the policeman (now living in another city) was contacted to see if he would meet with Tony. The therapist explained the background for this rather peculiar request to the policeman, and he agreed to meet them to answer specific questions Tony had prepared for him. The meeting was set up as a behavioural experiment, and Tony was given two tasks: To prepare concrete questions for the meeting, and to hypothesize how the policeman would look, behave, speak and answer his questions. The questions and hypotheses with the expected outcome were formulated as a questionnaire prior to the meeting. A tape recorder

was used during the meeting to ensure accuracy in later discussions between the therapist and Tony. As anticipated from the therapist's phone call with the police officer, the officer proved to be competent and understanding, and had no problems handling Tony's questions and accusations. The meeting resulted in Tony becoming more certain that this policeman could not be a part of the plot against him. He seemed to be relieved by this.

Tony's response to this behavioural experiment was monitored by a questionnaire developed for the experiment by both the therapist and the patient. The patient's symptoms of stress, voice-hearing, and delusions were recorded once a month, once a week and one hour prior to the behavioural experiment. Further more the same registrations took place one hour after and one week after the behavioural experiment. The results can be seen in table 2, and show a rather unexpected shift in some of the delusional content. He experienced a shift from thinking that the voices were telling him the truth and was threatening, to that they are mendacious and the threats empty. However he still reported in this questionnaire that he believes that the voices are beyond his control and that the voices come from outside his head. Tony still felt that the voices had the same intensity; he was still convinced that the voices were inflicted on him from outside, and that they were beyond his control. However, he felt safer and doubted that the police were actually after him. Clinical observations suggested that Tony was less anxious and depressed than before the therapy started. The questionnaire made especially for this behavioural experiment, only measured change in cognitions for a time period of one week after the confrontation with the policeman had taken place. There are therefore no measures related to changes in Tony's appraisal of localization and control of the voices after this time period. We do know from clinical practice and research that cognitive restructuring needs time to take place, and Tony's history after the termination of therapy, shows that his cognitions about the control of the voices and localization have been altered as a result of the cognitive behavioural therapy.

Table 2 approximately here

In retrospect, we see that the meeting between the policeman and the patient was very important and useful for the patient. Tony was pleased to take an active part testing his assumptions and fears. The cognitive behavioural therapy used with this patient emphasized to actively test reality in a simple and straightforward way. This made Tony receptive for a re-evaluation of his strongly held delusional beliefs and reappraisal of the voices. In therapy, Tony's delusional thinking was challenged, which stimulated him to explore alternative beliefs. The anxiety, depression, and distress caused by his delusions and his appraisal of his auditory hallucinations were reduced through these cognitive behavioural techniques.

Discussion

The case study has illustrated how behavioural experiments can be used to treat delusions and auditory hallucinations in a patient diagnosed with paranoid schizophrenia. The models of Morrison (2001) and Freeman et al. (2002) provide a good foundation for formulation to guide the treatment. The principles of cognitive behavioural therapy using Socratic questioning and designing specific targeted behavioural experiments resulted in disconfirmatory evidence for Tony's appraisal of the voices and his delusions were weakened. These cognitive changes considerably reduced Tony's distress and symptoms. The behavioural experiments Tony and the therapist set up used this rationale by presenting new, corrective information to be processed. Threat-related cognition that maintained the auditory hallucinations and persecutory delusions were changed. The good outcome in this case study could be related to different factors. Firstly, the patient and the therapist had an especially

good therapeutic alliance which is of crucial importance in doing CBT for psychosis. Secondly Tony's high level of suffering could also be seen as an important motivating factor in therapy. Finally, the patient's high degree of psychological mindedness made him very suitable for the rationale of cognitive behavioural therapy. Even if his conviction in his delusional beliefs faded as a result of the therapy, he still experienced some level of paranoid readiness to other people after termination of therapy. These negative core beliefs could probably be seen as a result of an insecure attachment to other people (Gumley, 2007). Some months after the termination of therapy, the patient was discharged from the psychiatric ward. Together with his case manager Tony and the therapist planned how Tony could build a new network, which did not reinforce substance use, and how he could stay well. He has now gone back to university to study, and has not yet relapsed.

Limitations and alternative explanations

The case study has some limitations. First, is the problem of generalisation from single cases? Caution is needed to generalise from small sample trials, and in particular from one single case. Second, the use of anti-psychotic drugs may influence the course and the intensity of the psychotic symptoms although his medication was kept at a stable dosage throughout the whole treatment. Thirdly, it may be difficult to assign all the changes Tony experienced during therapy to the behavioural techniques and psychosocial interventions alone. Most probably the treatment gains could be assigned to both medication and therapy, but the gains from the treatment was so swift and closely related to the interventions that we find the highly likely that the behavioural experiments influence on the changes was substantial. However this needs to be investigated in a larger trial. Finally, we cannot establish the long-term effect of the present approach to the treatment of paranoid schizophrenia based on the current case study. This must be considered in future studies.

Implications

Though the empirical support for CBT to treat psychosis has been established, to date there has not been a well-controlled trial that has attempted to dismantle the components of CBT for psychosis, or to identify the specific mechanisms responsible for treatment effectiveness (Gaudiano, 2005). Even if no dismantling studies have been attempted, results suggest that if interventions in cognitive behavioural therapy of psychosis are coded as being more cognitive or behavioural in nature, behavioural interventions seem to be more effective (Tarrier & Wykes, 2004). There have been some attempts to develop theories to explain particular elements of change in CBT for psychosis (Beck & Rector 2002; Beck & Rector, 2003; Beck 2004; Rector, Beck & Stolar, 2005), but we still lack a detailed theoretical and psychological understanding of the psychopathological processes in psychosis that could specify these changes observed in therapy (Tarrier, 2005). Although behavioural experiments are considered a valuable part of cognitive therapy for psychosis, there has been little attention to research on how to best utilise it in cognitive behavioural therapy of psychotic symptoms. An interesting unanswered question is whether the process of change is best described through change in thought content or if it is more a function of metacognitive awareness (Gaudiano, 2005)? Evidence from different studies has failed to show the specific efficacy of cognitive interventions compared to behavioural interventions (Jacobson et al., 1996; McLean et al, 2001; Borkovec, Newman, Picus & Lytle, 2003). The evidence suggests that the use of behavioural techniques is a powerful therapeutic strategy, and that behavioural experiments promote great cognitive, affective, and behavioural change (Rouf, Fennell, Westbrook, Cooper & Bennett- Levy, 2005). This should also be explored more carefully in psychotic disorders. Future research in CBT for psychosis should therefore examine empirically supported principles of change; studies using dismantling designs could prove useful for this.

It ought to be noted, though, that there are some negative expectations that such a research programme could be productive for these purposes (Tarrier & Wykes, 2004; Tarrier 2005). A dismantling study, comparing behavioural interventions against more cognitive interventions, could indicate whether it is the behavioural interventions or the cognitive interventions that make CBT of psychosis effective. With this kind of research as the foundation we might be able to create more specific models of the mechanisms involved in the aetiology and maintenance of psychotic symptoms and tailor CBT to the different types of patients who suffer from psychosis.

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Tables

Table 1. Behavioural experiments used in the cognitive behavioural therapy with Tony.

Table 2. Changes in symptoms, appraisal of voices, conviction in delusions before and after the behavioural experiment (BE) of meeting the policeman

Table 1. Behavioural experiments used in the cognitive behavioural therapy with Tony.

Target cognitions	Alternative cognitions	Experiment	Results	Conclusion
<u>Paranoid ideation:</u> Police wants to attack me	There is no one who is trying to hurt me.	Go out to shops and the nearby gym to see what happens	No one tried to hurt me.	Even if I feel threatened by other people, this does not mean that they are dangerous. Some people are also really nice to me. Going shopping and working out at the gym also has a positive effect on my depression
<u>External forces:</u> The voices I hear comes from an external force	The voices I hear are coming from inside of me. These voices can be my thoughts.	Find a room where no radio beams can enter to see if I still can hear the voices.	I can still hear the voices, which means that the voices are probably coming from inside of me.	These voices I hear could be my thoughts. Maybe these thoughts make me anxious and I hear them as externally generated? If these thoughts make me anxious, I should find out more about them, and try to find out if what they say is true
<u>Somatic concerns:</u> There is something wrong with my pituary gland	There is nothing wrong with my pituary gland. My symptoms do not fit with gland disease	Read about how the pituary gland is functioning. Search the Internet for information.	Even if I feel something is wrong, it does not mean that this is a fact.	If there is nothing wrong with my pituary gland, there must be an alternative explanation? The bottles of water and the aluminium foil I put under my bed to protect me from the radio beams may be unnecessary. Maybe I could put them away and see what happens?
<u>Ideas of persecution:</u> The police are watching me, and they have put a price on my head	The police are not watching me. They are really not interested in me. I am just afraid.	Meet the policeman who I think is responsible for the persecution of me, and ask him questions about this.	The policeman I thought was trying to kill me, was a rather nice person. His answers were reassuring.	I don't think the police are trying to kill me, and there is not a conspiracy to kill me. What is the explanation of the voices I hear, and the symptoms I feel in my body? Could there be an alternative explanation to this? Maybe I could explore this?

Table 2 Changes in symptoms, appraisal of voices, conviction in delusions before and after the behavioural experiment (BE) of meeting the policeman [1]

	How anxious do you feel right now? [a]	How depressed do you feel right now? [b]	Voices coming from outside your body? [c]	Voices beyond your control? [c]	Voices telling you the truth? [c]	Do the policemen want to kill you? [c]	Do the police conspire against you? [c]	Do the police use radiation to hurt you? [c]	Do you think the policemen know where you live? [c]
One month before BE	8	8	8	8	7	8	8	8	8
One week before BE	2	0	8	8	3	5	8	8	8
One hour before BE	1	0	8	8	4	6	8	8	8
One hour after BE	0	0	8	8	2	0	0	4	0
One week after the BE	0	0	8	8	0	0	0	0	0

Note [1]: Scores in symptoms, appraisal of voices, and conviction in delusions before and after the behavioural experiment (BE)

[a] 0= not anxious at all, 8= very anxious,

[b]: 0 = not depressed, 8= very depressed;

[c]: 0 = not certain at all, 8 = very certain it's true

Figure captions

Figure 1. The different phases in cognitive behavioural therapy of psychosis.

Figure 2. A cognitive formulation of Tony's auditory hallucinations.

Figure 1. The different phases in cognitive behavioural therapy of psychosis.

Engage the patient in therapy, and create a therapeutic alliance.

↓

Normalization of psychotic symptoms.

↓

Develop and share a case formulation with the patient

↓

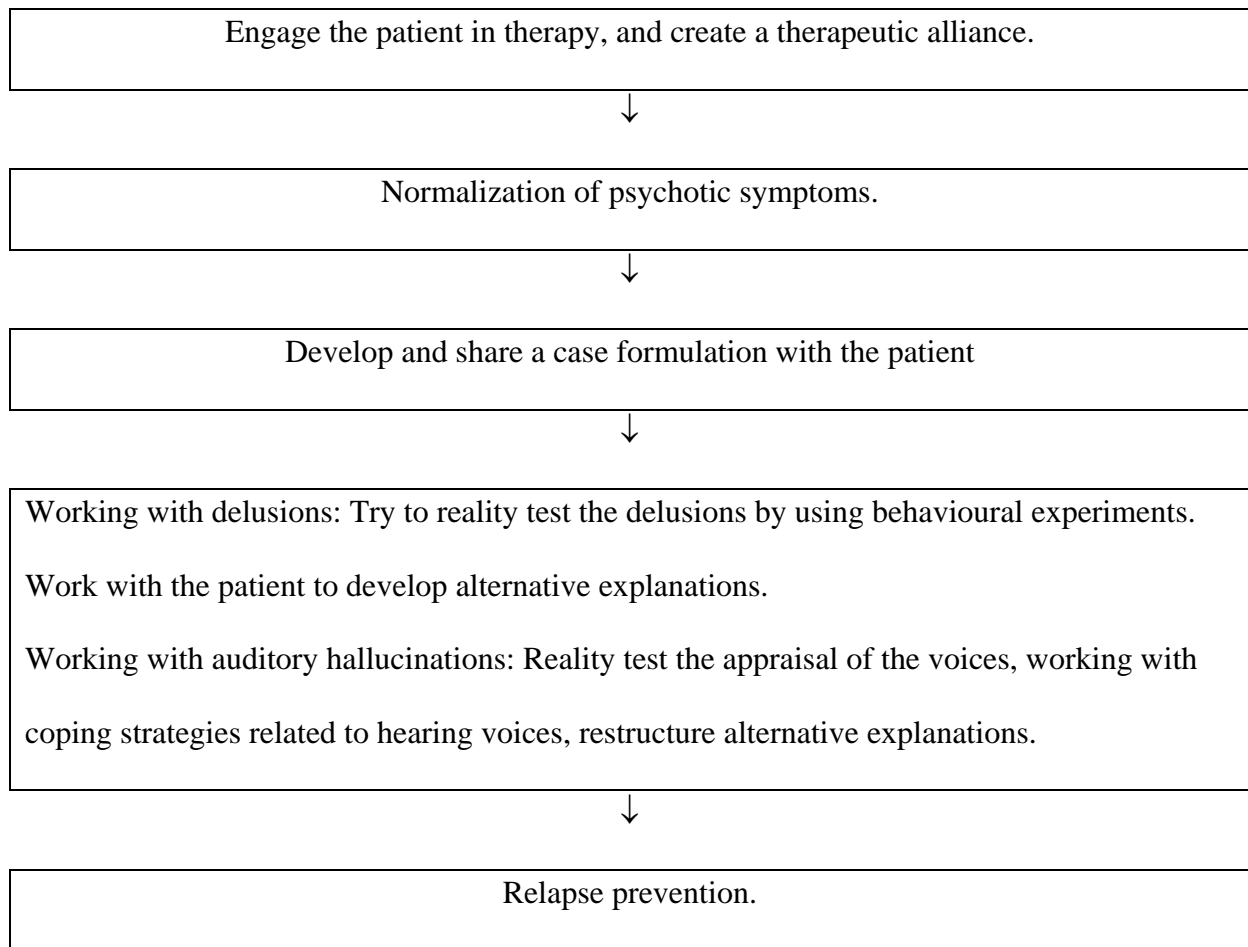| Working with delusions: Try to reality test the delusions by using behavioural experiments. Work with the patient to develop alternative explanations. Working with auditory hallucinations: Reality test the appraisal of the voices, working with coping strategies related to hearing voices, restructure alternative explanations. |
|---|

↓

Relapse prevention.

Figure 2. A cognitive formulation of Tony's auditory hallucinations.

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                    ┌─────────────────────────────┐
                    │         Triggers:           │
                    │      Smoking cannabis        │
                    │  Thoughts of being persecuted│
                    └──────────────┬──────────────┘
                                   │
                                   ▼
┌──────────────────────┐  ┌─────────────────┐  ┌──────────────────────┐
│   Mood and arousal:   │  │ Hearing a voice:│  │ Avoidance and safety  │
│    Anxiety and low     │─▶│ "We are watching│◀─│      behaviour:       │
│  mood, hyper vigilance │  │      you"       │  │ Stays at home, using  │
└──────────────────────┘  └────────┬────────┘  │     substances        │
         ▲                          │            └──────────────────────┘
          \                         ▼                     ▲
           \        ┌─────────────────────────────┐      /
            _____│          Appraisal:          │_____/
                    │ The police are going to hurt me│
                    └─────────────────────────────┘
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