Techniques in Cognitive Behavioural Therapy: Using Normalising in Schizophrenia

Normalising is a main factor predicting a good clinical outcome when it is linked to other formulation techniques such as tracing the antecedents of breakdown, decatastrophising schizophrenia and education about illness.

TEKST
Robert Dudley
Caroline Bryant
Katherine Hammond
Ronald Siddle
David Kingdon
Douglas Turkington

PUBLISERT 4. mai 2007

ABSTRACT:
The history and use of normalising psychotic symptoms is described. The relative importance of normalising as a technique in cognitive behavioural therapy for schizophrenia has not been investigated. A logistic regression is undertaken on those patients with a good clinical outcome (50% improvement) in overall symptoms to identify the roles of the various components of cognitive behavioural therapy. Normalising as part of the broader process of formulation was the only significant predictor of good outcome. When normalising is linked to other formulation techniques such as tracing the antecedents of breakdown, decatastrophising schizophrenia and education about illness, it is seen to be the main factor predicting a good clinical outcome. Normalising techniques should be widely used and taught to front line clinicians who are treating patients with schizophrenia.

Keywords: normalisation, psychosis, schizophrenia, cognitive behavioral therapy

EMNER Normalisation Psychosis Schizophrenia Cognitive behavioral therapy

Acknowledgements

We acknowledge the authors of the original Wellcome trial (Tom Sensky, Jan Scott, Jonathon Scott, Madeline O'Carroll and Thomas Barnes).

The Role of Normalising in Cognitive Therapy

Normalisation is a central process within cognitive behavioural therapy (CBT) and not just CBT for psychosis (CBTp). This is because CBT is based on the cognitive model which emphasises that the appraisal of an internal or external event determines emotion and behaviour. Central to the model is the notion that if we understand the cognition or appraisal, the emotion and behaviour will make sense to us. In fact, if we believed the same thought there is a good chance we would feel and act in the same way. The goal of treatment in CBT is to help the person appraise the world, other people and the self as they really are and to not overestimate threat (i.e. palpitations are normal and a reaction to arousal not usually a sign of a heart attack), or over value the event if it has happened (i.e. just because your lost a job it does not mean you are a failure).

Normalising is used in CBT in a number of different ways. Firstly, by drawing on the cognitive model we are making the experience of distress normal and understandable. The therapist will say to a person with compulsive washing something like "So you believed you had poison on your hands and that you would be responsible for killing your children, well no wonder you felt anxious, and wanted to wash your hands". This style is evident from the first session and is a powerful form of normalisation. Furthermore, the therapist helps the clients see that they are not alone in experiencing certain feelings or thoughts, and this can enhance feelings of self-esteem, facilitate improved coping and reduce stigmatisation. Normalising can help reduce secondary emotional reactions such as being anxious about anxiety or depressed about being depressed. Secondary behaviours which perpetuate the primary symptom are also reduced, e.g. safety behaviours and social withdrawal. This process can be carried out through the therapist providing the client with reading material, e.g. fight/flight response material to normalise physical sensations in anxiety and examples of people also suffering depressive symptoms after losing their job.

Personal disclosure is also potent in the process of normalising. For instance, the therapist might describe how they had a phobia of public speaking and how they overcame this or perhaps reveal one of their own experiences of intrusive thoughts when working with a person with obsessions (Salkovskis, 1999). Normalisation can also be seen as an active element within group CBT as people can relate to one another within the group and see that other people experience similar problems (Finucane & Mercer, 2006).

Normalising experience is also at the heart of current appraisal models of anxiety disorders (Salkovskis, 1996). In these problems central to treatment is creating a change in catastrophic or unhelpful appraisals of normal phenomena such as bodily sensations (panic disorder, health anxiety and social phobia), intrusive thoughts (obsessive compulsive disorder, generalised anxiety disorder), and intrusive memories (PTSD). A key treatment common to all these approaches is normalisation of these physiological or cognitive phenomena.

It is important to remember that this process is not purely restricted to CBT. For example, the medical model is, perhaps surprisingly, not exclusive of a normalising approach. A good example is asthma. It is useful for the person with asthma to know that anyone will wheeze with a severe chest infection. This reduces catastrophic thoughts about the meaning of the bronchospasm, e.g. "I am dying and this is untreatable". Another example is epilepsy, again the patient is reassured to hear that seizures are extremely common and that anyone can have one. This can lead to reduced shame and improved compliance with anticonvulsants.

Definition of Normalisation

When we describe normalisation the intention is not to say that the experiences are a sign of health or wellbeing. Rather normalisation is a process that emphasises that the experiences a person finds upsetting exist within the range of normal functioning and can be experienced in the absence of distress, or disability. The cognitive model of psychotic symptoms like that of obsessive compulsive disorder (OCD) (Salkovskis, 1999) would argue that the development of delusional beliefs or hallucinations has its origins in normal experiences. The difference between non distressing and distressing experiences lies not in the occurrence, content, or even the uncontrollability of these experiences but rather in the inter-pretation by the person of the meaning of the experience.

Normalising Psychotic Symptoms

This view of what normalisation is immediately brings us up against the issue of whether we can normalise psychosis. Most of us would accept that we know what it feels like to be low or anxious. Our own experiences help us develop empathy for those with depression or anxiety problems. However, normalisation is not just about developing empathy. The message given is that these experiences are not in themselves problematic, and that other people can have them without being distressed. However, psychotic experiences (and in particular those labelled as schizophrenia) have been catastrophised rather than normalised, not just by the patient, but also by society and the media. Psychotic experiences were seen to be discontinuous with normal experience and be a sign of a qualitative change in a person, presumably owing to a biological disease process (Read, 2004).

This view of psychosis has consequences. The mere act of labelling an individual with psychosis as mentally ill is linked with an increased perception of their unpredictability and dangerousness (Angermeyer & Matschinger, 2005; Phelan, Cruz-Rojas, & Reiff, 2002; Pote & Orrell, 2002). Clinicians have been known to catastrophise the diagnosis of schizophrenia and so find it hard to communicate the diagnosis to the person directly, thus leaving them to find out by other means. The patient may then learn of their diagnosis from a carer or deduce it through recognition of the symptoms from a television programme, the Internet, or reading the information accompanying any prescribed medication. This process then leaves the client to draw unhelpful conclusions about why they weren't told directly, again often leading to

catastrophisation of the problem such as beliefs that they are “mad”, there “is no hope” for their recovery or they will be “locked up” (Bentall, 2003). There has also been a general opinion that discussing psychotic symptoms with a client can lead to an exacerbation of the problem, but a lack of discussion can again lead to the client catastrophising their perceived “untreatability” which in turn leads to greater distress (Kingdon & Turkington, 1994).

However, it has become increasingly evident through research that psychotic experiences exist in the general population on a continuum of severity rather than as categorically different phenomenon (Strauss, 1969, Van Os, Hanssen, Bijl, & Vollebergh, 2001). Surveys of the general population have been carried out using questionnaires or interviews to measure psychotic symptoms, and findings show that a range of symptoms from paranoia to hallucinations are relatively common in apparently healthy community samples. For example, Poulton et al. (2000) reported a prevalence figure of 17.5%, and Van Os et al. (2001) reported 25% of people had experienced psychotic symptoms. Wiles et al. (2006) reported a prevalence of psychotic symptoms of 4.4% in the general population. Freeman et al. (2005) reported that over a third of their sample of 1200 undergraduates had experienced paranoid thoughts about the intentions of others within the last week. Their survey revealed thoughts that friends, acquaintances, or strangers might be hostile or deliberately watching them. Hence, suspiciousness and paranoid ideation appears to be an everyday occurrence for many people. In fact, 52% endorsed the idea that “I need to be on my guard against others” as occurring on a weekly basis. To a lesser extent people believed that there may be someone plotting against them or there being an active conspiracy against them (8% in the last week). The prevalence of symptoms in the general public could provide a key element to the normalising process as it indicates the presence of psychotic symptoms and experiences are far greater than the level of identified mental illness.

Johns et al. (2004) investigated self-reported psychotic symptoms from the general public. The annual prevalence of psychotic symptoms in the general population was 5.5%. They found that the psychotic symptoms were more likely to occur in people with factors such as substance misuse, neurotic symptoms, adverse life events and victimisation in their lives, and each of the different factors has supporting literature that can be used as part of the normalisation process. One of the socio-demographic influences highlighted by Johns et al (2004) was urbanisation, and previous research has highlighted that psychotic symptoms in the general community increase in prevalence as the level of urbanisation increases (Krabbendon & Van Os, 2005; Van Os et al., 2001). Patients who live in highly urbanised areas may be able to identify with these findings, and it may be helpful for them to discover that the general public are affected in a similar way. Other studies show that the levels of victimisation and stressful life events specifically increase levels of paranoid ideation (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Janssen et al., 2005). Wiles et al. (2006) support the above in relation to quoting more adverse life events, lack of social support, neurotic symptoms and perhaps surprisingly rurality as being linked to increased

likelihood of experiencing psychotic symptoms. However, they suggest that urban residence at birth or upbringing could account for the discrepancy.

Significant life events often precede the onset of psychosis, similar to the onset of Depression or Post-Traumatic Stress Disorder (Zubin & Spring, 1977). For instance, hallucinations are common in those who have suffered prolonged or brutal sexual abuse (Ensink, 1992; Read, Mosher, & Bentall, 2004); although many patients will not disclose such things even when given the opportunity to do so, when they do it can be useful to highlight the possible connection to help give an understanding of symptom development. Grassain (1983) has also identified that prisoners who were kept for prolonged periods in solitary confinement were prone to develop psychotic symptoms. Excessive bed rest or other sensory deprivations have also been found to induce hallucinations (Slade, 1973). Research has also identified that sleep deprivation can be a trigger for psychotic symptoms, leading to illusions, hallucinations and paranoid ideation (Oswald, 1974). Such literature can be presented to patients so that they can identify with the triggers and feel less alienated by their experiences.

Hence, we have seen that normalisation is a common component of CBT when working with non-psychotic disorders. Also that the assumption that psychotic experiences are categorically different from other experiences does not hold up when considering the circumstances in which these develop nor the fact that they can be experienced without a person experiencing distress. Therefore, we turn our attention to the process of normalisation within cognitive behavioural therapy for psychosis (CBTp).

Normalising Process in CBT for Psychosis

The first stage of CBTp is engaging the client and forming a therapeutic alliance that will allow a collaborative approach. This first step is crucial, and generally this is encouraged through empathy, warmth, genuineness and unconditional acceptance displayed by the therapist, who would also display a knowledge of typical modalities of psychotic expression (hallucinations, delusional perceptions, systematisation of delusions etc). Therapists can often be put off by the large delusional systems, but through the engaging phase you can work towards a formulation of symptom emergence to allow therapy to begin. The therapist can also provide reading material, case examples and personal disclosures about how one has used that particular technique to overcome problems (e.g. anxiety).

The process of normalisation can be used as a therapeutic tool towards forming this alliance through work on non-threatening exploratory areas prior to tackling the patient's own symptomatology and reducing possible experiences of shame. Plus it can help pave the way for collaborative formulation, thus helping the client become an active agent in his or her own treatment. The therapist should also convey accuracy and consistency in their approach to the client, being careful not to invalidate any experiences through verbal or non-verbal cues, for example directly confronting a belief. Care should be taken that normalisation is not used in the extreme, which may be perceived to minimize the problem. If normalisation is used insensitively the client

may perceive that his or her problem is something that other people just cope with (e.g. everyone hears voices), or therapeutic work could miss out important issues such as the patient believing that he or she is bad (e.g. "If this isn't my illness making me think such things then I must be bad"). Therapists must remain aware of how far they are going in saying that psychotic symptoms are normal, and it is also important to identify possible influences from their own personal beliefs (Turkington & Kingdon, 1996).

After engaging the client it is useful to provide an explanation of the puzzling and distressing symptoms as well as deal with the catastrophic cognitions concerning insanity. The client can be led towards an understanding that there is probably a discernable reason or reasons why the symptoms have occurred and the possibility that anyone stressed in certain ways would develop psychotic symptoms. If there is a family predisposition to respond in this way this can also be fully explored to help the patient to feel less different and isolated. At this point literature detailing the prevalence of psychotic symptoms in the general population (Johns et al., 2004; Krabbendon & Van Os, 2005; Van Os et al., 2001) or more specific literature about particular life experiences, such as sexual abuse or solitary confinement, could be discussed with the client.

The Vulnerability Stress Hypothesis (Neuchterline & Dawson, 1984; Zubin & Spring, 1977) could also be introduced as this model simply states that vulnerabilities and stresses combine to produce the symptoms characteristic of psychosis. A close examination of the anecedents of psychotic breakdown may be necessary, and it is often useful to itemise the types of stressors that can typically produce psychotic symptoms (Brabban & Turkington, 2002). The crucial period leading up to a breakdown should be worked through with inductive questioning, imagery and role-play. Key cognitions can be detected from this period pointing to underlying schemas concerning achievement, approval and control that may be addressed in later sessions.

The normalising approach can help patients realise that everyone has upsetting automatic thoughts, intrusive thoughts or even obsessions during times of stress and worry. Generally the experiences of these thoughts can be similar to voice hearing experiences (e.g. they can be quite violent, sexual or religious), and it can be helpful for the patient to discover that others get anxious about their thoughts too, but most people choose not to act on them (Morrison, 1998). This process of normalisation can pave the way for imagery, role-play or schema level work to be undertaken to help deal with beliefs about voices and hence manage to command hallucinations differently (Birchwood et al., 2000; Trower et al., 2004).

Is Normalisation an Important Part of Treatment?

We know that CBTp is an effective treatment for people with persistent symptoms of schizophrenia (NICE, 2002; Sensky et al.. 2000; Turkington & Dudley, 2004). Whilst its value is proven, there is less evidence as to what are the mechanisms of change. CBT, generally, consists of a number of core components (Beck, 1995) that include a good therapeutic relationship, a style of collaborative empiricism, the use of cognitive and

behavioural change techniques, and the use of a disorder specific model as the basis of the formulation. There has been limited investigation of the active and successful components of treatment in CBT generally (see Dimidjian et al., 2006; Jacobson et al., 1996; Shaw et al., 1999). There is evidence that positive therapy alliance can potentiate the effectiveness of empirically supported therapies (Horvath, 1994; Raue & Goldfried, 1994), and also evidence that the use of effective therapy approaches leads to a more positive therapy alliance (DeRubeis, Brotman, & Gibbons, 2005). However, there is virtually no such research undertaken with regard to psychotic illness (Garety et al, 1997). Broadly speaking, there is only emergent empirical evidence that any or all of these components are necessary.

Despite the lack of empirical support these components are also incorporated into the CBTp treatment manuals (Chadwick, Birchwood, & Trower, 1996; Fowler, Garety, & Kuipers, 1995; Morrison, Renton, Dunn, Williams, & Bentall, 2004). Commonly, in these specialised approaches there is an increased emphasis on engagement and rapport building, techniques such as normalising unusual psychotic symptoms, as well as decatastrophising distressing appraisals of what it means to have a psychotic illness such as Schizophrenia (Kingdon & Turkington, 1994). Hence, it is apparent that CBTp relies on the core components of CBT as well as components more specific to the condition. However, within CBTp the process and role of formulation is considered to be especially important (Morrison et al., 2004).

Formulation is the process of integrating the person's specific information with the cognitive model and serves to help understand the onset and maintenance of the current difficulties as well as directing the therapist to key points of intervention (Tarrier & Calam, 2002). Given this function, formulation is considered to be the lynchpin of CBT (Butler, 1998; Persons, 1989). Despite the central prominence given to the role of formulation there is actually an absence of evidence for the value of a formulation in producing a successful outcome (Beiling & Kuyken, 2003; Kuyken, 2006). Hence, even one of the most important components of CBT has strikingly little evidence for its value. Formulation in work with people with psychotic illness helps provide a shared understanding, and this is particularly relevant when working with symptoms that can initially appear "incomprehensible" (Dudley & Kuyken, 2006). Formulation in CBTp is heavily reliant on normalisation as together the therapist and client are trying to generate and test a new, less threatening alternative explanation for the person's experiences, and information about the symptoms is vital in this process.

To date, the only study of formulation in CBTp was undertaken by Chadwick, Williams and McKenzie (2003). These authors reported that formulation had no impact on the perceived therapeutic relationship, psychotic symptoms, or levels of anxiety and depression. Hence, despite the practice there is an absence of evidence for the value of formulation.

This present study investigated which components of CBTp were used most in working with those people with schizophrenia that did respond to CBTp, in comparison to those that did not respond in a randomized controlled treatment trial (Sensky et al., 2000).

Whilst it is possible to predict differences in the use of a number of different components of treatment, particular emphasis is given to the role of formulation in working with people with psychotic illness. Hence, we hypothesise that formulation including the process of normalisation will be more commonly used with people who benefited from treatment than with people who do not benefit.

Method

Participants

The treatment data utilised in this study is derived from a randomised controlled trial of CBT for treatment-resistant schizophrenia (see Sensky et al., 2000, for full details of the trial). Participants met DSM-IV diagnostic criteria (APA, 1994) and were randomised to either CBTp or a Befriending (BF) control condition lasting up to approximately 20 sessions over a period up to nine months. Follow-up at nine months post treatment indicated that 21 out of the 45 in the CBT and 9 of the 43 BF group demonstrated a good clinical outcome, defined as a 50% reduction in total symptomatology as measured on the Comprehensive Psychopathological Rating Scale (Asberg, Montgomery, Perris, Schalling, & Sedvall, 1978). The 21 demonstrating a good clinical outcome following CBT were classified as responders, whereas the remaining 24 were classed as non-responders.

The treatment trial was conducted at two different sites. However, the data reported here was only collected from one site in which there were 30 people in the CBT group, with 10 classed as non-responders and 20 as responders.

Pre treatment there were no differences between the responders (R) and non-responders (NR) in duration of illness (R mean years of illness = 10.1, *sd* = 6.9, and NR mean = 13.5, *sd* = 8.7, $t(26) = 1.1$, $p = .3$) or main symptom measures, including overall symptomatology on CPRS total score (R mean = 34.3, *sd* 14.6, and NR mean = 45.1, *sd* 14.8, $t(26) = 1.8$, $p = 0.9$), negative symptoms (Scale for Affective and Negative Symptoms, SANS total score, Andreasen, 1989, R mean score = 34.0, *sd* 20.0, NR mean score = 47.1, *sd* 26.5, $t(26) = 1.09$, $p = .28$), Depression (as measured by the Montgomery and Asperg Depression Rating Scale MADRS total score, Montgomery and Asberg, 1978, R mean score = 9.95, *sd* = 4.9 NR mean score = 11.0 sd 5.4, $t(26) = .5$, $p = 0.62$), nor on schizophrenia specific symptoms (i.e. hallucinations, delusions, as measured on the CPRS R mean score = 10.15, *sd* 5.65, NR mean score = 12.13, *sd* = 5.3, $t(26) = 0.84$, $p = 0.4$).

Whilst there were no demographic or symptom differences between responders and non-responders pre treatment there was a trend towards differences in the non-responders having higher symptom scores generally, and in the number of attended appointments with non-responders attending less appointments (mean 11.88, *sd* 11.7 sessions attended vs 20.6, *sd* = 5.3, $t(26) = 2.04$, $p = 0.08$). Whilst the non-responders did attend less sessions the non-responders were still showing substantial engagement. Despite less time in treatment they were no less satisfied with the overall treatment (mean of responders 71.7, *sd* 11.4, and non-responders, 61.4, *sd* 5.2 $t(13) = 1.5$, $p > 0.16$).

Content of Treatment Scale

Following each session of therapy the research therapist completed forms indicating components of treatment that were used in that session. The list consisted of 49 items including components such as Rapport Building, Normalisation, Decatastrophisation, Formulation and Cognitive and Behavioural change techniques. The list was developed by the research group undertaking the trial. It was based on consultation with relevant literature (Kingdon & Turkington, 1994) and clinical experience. The therapist indicated whether a component had been used or not in each session. The recording was only whether a technique had been used, not to what extent it had been well received, or been successful.

The items, which were ordered into eight subscales combining items within themes such as Relationship, Formulation, Cognitive techniques, and Psychosis Specific treatment components such as analysis of hallucinations, and peripheral questioning of delusional beliefs (Kingdon & Turkington, 1994). The items and subscales are illustrated in appendix 1. Formulation incorporated items that were used to provide an alternative and less distressing explanation for a person's experience. (These items have been grouped retrospectively. Two experienced cognitive therapists independently endorsed the items as relevant to each category, with a high rate of agreement, kappa of 0.89.)

Results

The data for session content was available from one of the two sites representing 20 of 21 responders and 10 of the 23 non-responders. In three instances of the non-responders data on session content was not recorded consistently. Hence, data is available for seven non-responders.

Items Analysis

Non-parametric analyses on all of the items indicated that Personal disclosure (mean number of times used for the non-responders (NR) = 2 , and for the responders (R) = 7 , U = 22.5, p = 0.006), Normalisation (Mean times used NR = 1.8, R = 4.3, U = 32, p = 0.04), Education about the illness (Mean times used NR= 3.6, R = 10.05,U = 18, p = .003), Focus on schemas (Mean times used NR= 1.5, R = 4.4, U = 31, p = 0.03), Relapse Prevention (Mean times used NR = 0.4, R = 2, U = 26.5, p = 0.013) were all used to a greater extent with the responders more than the non-responders. Controlling for multiple comparisons by accepting an alpha value of 0.01 differences are evident between the groups only in the use of "Education about Schizophrenia", and "Personal Disclosure". There was no difference in the frequency of use of the individual item labelled Normalisation or Formulation.

The items in the scale demonstrated good internal consistency (Cronbach's alpha = 0.87) and were summed to produce a total score of techniques used. There was a difference between the groups in the mean total score on the number of components of therapy used with non-responders having a mean total score of 40.8 (sd = 42.3), which

was significantly different from the responders (mean = 88.7, *sd* 33.5, $t(25) = 3.1$, $p = 0.005$).

Whilst individual components are of interest, the reality is that these items do not exist in isolation from each other. Rather a number of different components may serve similar aims and hence may be used interchangeably to achieve that aim. Hence, items were combined into related subscales with Normalisation being incorporated with Education about illness, and Stress vulnerability work within the broader subscale of formulation and mean scores of the responders and non-responders are shown in table 1.

TABLE 1. TOTAL AND SUBSCALE SCORES OF RESPONDERS AND NON-RESPONDERS

Subscale	Responders mean (*sd*)	Non-responders Mean (*sd*)
Relationship	17.9 (9.6)	7.3 (8.3)
Information gathering and giving	5.4 (3.7)	4.2 (4.9)
Formulation	22.2 (8.8)	10.1 (9.2)
Cognitive change techniques	6.2 (4.9)	2.1 (3.4)
Behavioural change techniques	6.8 (4.9)	3.7 (4.1)
General change techniques	10.5 (6.9)	3.5 (3.8)
Cognitive schema level techniques	11.2 (7.5)	4.7 (5.8)
Psychosis specific techniques	8.4 (7.0)	5 (6.4)
Total score	88.8 (33.5)	40.8 (42.2)

Analysis of the subscales indicated significant differences between responders and non-responders on Relationship ($t(25) = 2.6$, $p = 0.016$), Formulation ($t(25) = 3.07$, $p < 0.005$) , General Change techniques ($t(25) = 2.5$, $p < 0.02$) and Schema Change techniques ($t(25) = 2.5$, $p < 0.05$). There were no differences on the other subscales.

The above analyses allow comparison of the responders and non-responders in terms of what are the differences in the frequency of the use of different components and different categories of components of CBTp. However, the groups differed in the total number of sessions and hence, the dose of treatment. Hence, a backwards stepwise logistic regression with outcome as the criterion variable and all of the subscales being entered along with number of sessions was undertaken. As there is no previous research on which to base hypotheses for testing it was considered appropriate to use

backward stepwise regression to explore which elements were associated with good outcome (Field, 2005).

A convention for number of cases needed to run a regression is N > 50 + 8M (where N is number of cases and M is number of predictor variables; Tabachnick and Fidell, 2001). The dependent variable is Response (denoted by the binary code). Independent (predictor) variables were the eight subscales as indicated in the appendix. The first time the analysis was run, no significant outliers were identified by SPSS. The results of the final regression are shown in Table 2

TABLE 2. RESULTS OF BACKWARD STEPWISE LOGISTIC REGRESSION

						Exp B 95% CI		
Predictor	**B**	**S.E.**	**Wald**	**df**	**Wald Sig.**	**Exp B**	**Upper**	**Lower**
Formulation	.15	0.06	5.66	1	.017	1.16	1.03	1.31

Note. * signifies variable is a significant predictor of quality score ($p < 0.05$)
B = Beta
SE = standard error of beta
Wald = Wald statistic
df = degrees of freedom of Wald statistic
Wald sig. = significance of Wald statistic
Exp B = indicator of change in odds (odds-ratio)

The model overall correctly classified 74% of the data, which shows that the model is able to predict well which group a case will belong to.

The best test of "goodness of fit", the Hosmer and Lemeshow test (8.68 (6), $p > 0.19$) shows the model to have good fit. The R squared figures are 0.26 and 0.38, which means that between 26% and 38% of the variability is explained by this formulation variable.

Overall, this data means that, when considering Hosmer and Lemeshow's formula (Field, 2005), 32% of the variance in production of a response to CBT that the model accounted for by formulation is a good fit with the data. This means that we can cautiously proceed in discussing the outcome of the regression. Formulation is a significant predictor of a response to treatment. However, if number of sessions is added to the regression equation Formulation is no longer significant (B = 0.11, S.E. 0.87, Wald 1.5 (df1) P = .22).

Discussion

Individual techniques that differentiated responders and non-responders included the use of Education about Schizophrenia as well as the use of Personal Disclosure. Both in essence would appear to be very normalising processes. Overall, responders received

more sessions and hence a bigger dose of treatment, and this is reflected in the differences in the total scores. There appeared to be a particular emphasis on the use of formulation components of treatment. Formulation was the only significant predictor in a backward stepwise regression. However, this did not remain when number of sessions was controlled for. What is evident then is that when people respond to CBTp they receive a greater number of sessions than non-responders. Within this greater dose there appear to be more schema focussed and general change techniques used as well as a greater emphasis on the relationship.

Besides the limitations of the statistical procedures owing to the sample size a number of other limitations need to be considered. Firstly, we have only used the data from one of the two treatment sites. This naturally reduces the potential generalisability of the findings. Secondly, only the presence of techniques was recorded and not other potentially relevant information such as the amount of time spent during the session on that technique, nor the quality of the implementation of the component. In the RCT, independent ratings of sessions indicated that the sessions were reliably identifiable as CBT. However, we have no data as to the quality of the individual components. Hence we only have an indication of the quantity rather than the quality. Neither do we know the perceived value of these components from the therapists or, perhaps of more relevance. from the perspective of the patient. The impact is unknown. Clearly the scale is not validated and is based on clinical and theoretical assumptions. However, it does show excellent internal consistency, and demonstrated good agreement of judges on the subscale items.

Whilst we have controlled for dose statistically, it is clear that the tasks of later sessions are likely to be different from earlier sessions as there is a greater emphasis on schema level interventions that will target vulnerability factors such as rules or assumptions as well as relapse prevention work. Hence, the effect of increased sessions is likely to be sessions of a different content, rather than just more of the same. Ideally, we would have compared frequency of use of components up to session 10 in both groups. However, the data was only available in summary form, and therefore could not be used.

Clearly, we cannot address questions of cause or consequence by this methodology. We cannot know that the people responded well because of the use of these techniques or that because they responded well the therapist could then employ these techniques. However, the results of this work are useful. Despite the limitations outlined above it is clear that people who respond to treatment are engaged in a process that is helping them develop alternative, less distressing explanations for their experiences. This provides some preliminary evidence for the value of normalisation in the context of formulation (Kingdon & Turkington, 1991; Kuyken, 2006).

Hence, we have some preliminary evidence for the value of Formulation in CBTp and within that for the vital role of Normalisation. However, it brings us back to how far we can normalise psychotic experiences. We have all experienced memory slips perhaps forgetting someone's name or the name of an object. However, few of us would say we

could use this experience to normalise the experience of amnesia or dementia. The same issue applies to our normalisation of psychotic experiences. How far can we accept these experiences are normal? We may have experienced feeling suspicious of other people, or heard a voice calling our name whilst waking from sleep, but does this really map onto the experience of believing that your parents are dead, and replaced by alien impostors, or a voice shouting that you are an evil whore for hours on end? There may be a point at which the frequency, loudness, and vividness of a voice or its content make the experience different to that experienced by people who are not distressed by these experiences. At present we do not know if it is a difference of degree or of quality. This is a challenge to us as clinicians and researchers, and there is difference within the CBTp models (Garety et al., 2001; Morrison, 1998) as to how far these experiences can be normalised. However, it is also the case that what makes an experience abnormal is to some extent culturally defined. The apparent improved outcome of people with psychotic experiences in non-Western societies (WHO, 1992) may in part be attributable to differences in appraisal of these experiences. Moreover, as experiences are culturally defined as normal it is important to remember that these definitions can and do change over time as the often quoted example of the removal of homosexuality from DSM illustrates.

Conclusion

Normalising has been increasingly incorporated into CBT treament manuals for anxiety, depression and OCD. It now appears that normalising is one of the most important components of successful CBT in psychosis. It has been incorporated in recent treatment manuals (Kingdon & Turkington, 2005), and it can be effectively taught to psychiatrists in training (Garrett et al., 2006). The challenge now is to disseminate this training more widely and to make formulation-based CBT available for those who need more intensive treatment due to chronicity or comorbidity.

Robert Dudley, Ph.D.

Doctorate of Clinical PsychologyNewcastle University

Ridley Building, Newcastle, NE1 7RU

Email: r.e.j.dudley@ncl.ac.uk

Tel: 0191 222 7925

ROBERT DUDLEY

Robert Dudley, Ph.D., is a Consultant Clinical Psychologist for the Early Intervention in Psychosis service for Sunderland, Gateshead and South Tyneside in the North East of England, and a Research Tutor for the Doctorate of Clinical Psychology course of Newcastle University. In these roles the purpose is to combine research and clinical practice and work to develop better understanding of the psychological processes in operation in experiences such as voice hearing, or paranoia, and by this help increase the value and impact of treatments such as Cognitive therapy.

Key publications

Dudley, R.E.J., & Kuyken, W. (2006). Formulation in cognitive therapy. In L. Johnstone and R. Dallos (Eds). *Formulation in psychology and psychotherapy.* Brunner Routledge.

Garety, P.A. , Freeman, D, Jolley,S., Dunn, G., Bebbington, P., Fowler, D., Kuipers, E., & Dudley, R. (2005). Reasoning, emotions and delusional conviction in psychosis. *Journal of Abnormal Psychology, 114,* 373–384.

Turkington, D., & Dudley, R. (2004). Cognitive behavioural therapy in the treatment of schizophrenia. *Expert Review of Neurotherapeutics 4(5),* 861–868.

RONALD SIDDLE

Ronald Siddle Ph.D is a consultant psychologist and the clinical lead for the Early intervention in psychosis service (EIS) in Cumbria , UK. The aim of the EIS is to improve outcomes in psychosis. The service aims to deliver an individualized, formulation driven course of cognitive-behavioural therapy to all users of the service by the care coordinators. Research has been particularly focused on religious delusions and the delivery of cognitive-behaviour therapy to service users with psychosis.

Key publications

Barrowclough, C., Haddock, G., Lobban, F., Jones, S., Siddle, R., Roberts, C., & Gregg, L. (2006). Group cognitive-behavioural therapy for schizophrenia. British Journal of Psychiatry, 189, 527–532.

Lewis, S., Tarrier, N., Haddock, G., Bentall, R., Kinderman, P., Kingdon, D., Siddle, R., Drake, R., Everitt, J., Leadley, K., Benn, A., Grazebrook, K, Haley, C Akhtar, S., Davies, L., Palmer, S., Faragher, B., & Dunn, G. (2002). Randomised controlled trial of cognitive-behavioural therapy in early schizophrenia: acute-phase outcomes. British Journal of Psychiatry, 181, (suppl 43), 91–97

Sensky, T., Turkington, D., Kingdon, D., Scott, J., Siddle, R., O'Carroll, M., Scott, J. L., & Barnes, T. R. E. (2000). A randomised controlled trial of Cognitive- Behavioural therapy for persistent symptoms in schizophrenia resistant to medication. Archives of General Psychiatry, 57, 165–172.

DOUGLAS TURKINGTON

DOUGLAS TURKINGTON

Douglas Turkington is a major research figure within the history of the development of cognitive behavioural therapy (CBT) for schizophrenia. Professor Turkington has project managed a number of high impact randomized controlled trials in CBT of schizophrenia as well as continuing with a busy clinical caseload as a psychiatrist with the Liaison Psychiatry service of Northumberland, Tyne and Wear NHS Trust. His work has strongly influenced the NICE guidelines which recommended the routine use of CBT for the treatment of schizophrenia. Professor Turkington has also lectured widely throughout Europe and North America on the CBT techniques to be used with particular psychotic presentations. He has written more than 100 articles on the subject of CBT in schizophrenia, and with Professor David Kingdon co-authored three books on CBT for schizophrenia. Currently he is working on a CBT self help book for psychotic patients and carers.

Key publications

Turkington, D., Kingdon, D., & Turner, T. (2002). Effectiveness of a brief cognitive-behavioural therapy intervention in the treatment of schizophrenia. British Journal Psychiatry, 180, 523–527.
Turkington, D., Kingdon, D., Rathod, S. et al. (2006). Outcomes of an effectiveness trial of cognitive-behavioural intervention by mental health nurses in schizophrenia. British Journal of Psychiatry, 189, 36–40.
Turkington, D., Kingdon, D., & Weiden, P. J. (2006). Cognitive behavior therapy for schizophrenia. American Journal of Psychiatry, 163, 365–373.

Appendix 1

APPENDIX 1. COMPONENTS OF TREATMENT ITEMS AS ORGANISED INTO SUBSCALES

1. Relationship items: Components considered to be important in engagement, rapport building and managing potential ruptures in the therapeutic alliance.

1. Rapport building
2. Agree to differ
3. Humour
4. Personal disclosure

2. Information components: Components used to elicit information from the person or to do with the provision of information relevant to the person's needs but not directly tied to change techniques. For instance, provision of information about the use of medication and side effects.

1. Clarify language used
2. Direct questions
3. Measures

5. Cognitive focussed automatic thought level change techniques: Here primarily the techniques rely on verbal challenge of the automatic thought level of cognition

1. Rational responding
2. Tackling emotional investment
3. Evaluate the evidence
4. Negotiate and deal with cognitions
5. Pie chart for responsibility
6. Thoughts are not the same as actions
7. Change attributions

APPENDIX 1. COMPONENTS OF TREATMENT ITEMS AS ORGANISED INTO SUBSCALES

4. Medication discussed
5. Inductive questioning
6. Problem list

3. Formulation components: Items used to help provide an alternative explanation for the person's experiences, or methods used to help develop such a theory based explanation. Examples include:

1. Tracing antecedents of breakdown,
2. Normalisation
3. Decatstrophisation of Schizophrenia,
4. Education about Illness,
5. Stress Vulnerability models
6. Formulation
7. Generating alternative hypotheses

4. Behavioural focussed change techniques: These are techniques of treatment the function of which is to promote behavioural change, and are distinct from cognitive techniques in their emphasis on active behavioural change. The items are

1. Social skills practice
2. Role play
3. Activity scheduling
4. Exposure
5. Overcoming withdrawal
6. Behavioural change techniques
7. Behavioural experiments
8. Relaxation techniques

6. Cognitive focussed schema level change techniques

1. Downward arrow
2. Inference chaining
3. Focus on schemas
4. Focus on themes
5. Positive data logging
6. Continuum methods
7. Change beliefs

7. Psychosis specific change techniques

1. Critical collaborative analysis of hallucination
2. Peripheral questions of delusional beliefs

8. General change techniques: Includes general skills in cognitive behavioural therapy such as agendas, problem lists, and other such techniques

1. Paradoxical techniques
2. Relapse prevention
3. General coping strategies
4. Advantages and disadvantages of suicide
5. Anxiety and depression management
6. Reassurrance

Teksten sto på trykk første gang i Tidsskrift for Norsk psykologforening, Vol 44, nummer 5, 2007, side 562-572

TEKST
Robert Dudley
Caroline Bryant
Katherine Hammond
Ronald Siddle

David Kingdon
Douglas Turkington

Vis referanser

References

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders IV (4th ed.) (DSM-IV). Washington, DC: Author.

Andreasen, N. (1989). Scale for the assessment of negative symptoms (SANS). British Journal of Psychiatry, 155 (Suppl. 7), 53–58.

Angermeyer, M. C., & Matschinger, H. (2005). Labeling – stereotype – discrimination: An investigation of the stigma process. Social Psychiatry and Psychiatric Epidemiology, 40, 391–395.

Asberg, M., Montgomery, S. A., Perris, C., Schalling, D., & Sedvall, G. (1978). A comprehensive psychopathological rating scale. Acta Psychiatrica Scandinavica Supplement, 271, 5–27.

Beck, J. S. (1995). Cognitive therapy: Basics and beyond. London: Guilford Press.

Bentall, R. (2003). Madness explained: Psychosis and human nature. Allen Lane: The Penguin Press.

Bieling, P. J., & Kuyken, W. (2003). Is cognitive case formulation science or science fiction? Clinical Psychology: Science & Practice, 10, 52–69.

Birchwood, M., Meaden, A., Trower P., Gilbert, P., & Plaistow, J. (2000). The power and omnipotence of voices: Subordination and entrapment by voices and significant others. Psychological Medicine, 30, 337–344.

Brabban, A., & Turkington, D. (2002). The search for meaning: Detecting congruence between life events, underlying schema and psychotic symptoms. In A. P. Morrison (Ed.), A casebook of cognitive therapy for psychosis (59–76). New York: Brunner Routledge.

Butler, G. (1998). Clinical formulation. In A. S. Bellack and M. Hessen (Eds.), Comprehensive clinical psychology. New York: Pergamon.

Chadwick, P., Birchwood, M., & Trower, P. (1996). Cognitive therapy for delusions, voices and paranoia. Chichester: Wiley & Sons.

Chadwick, P., Williams, C., & Mackenzie, J. (2003). Impact of case formulation in CBT for psychosis. Behaviour Research & Therapy, 40, 671–680.

DeRubeis, R. J., Brotman, M. A., & Gibbons, C. J. (2005). A conceptual & methodological analysis of the nonspecifics argument. Clinical Psychology – Science & Practice, 12, 174–183.

Dimidjian, S., S. D. Hollon, et al. (2006). Randomized trial of behavioural activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. Journal of Consulting and Clinical Psychology, 74, 658–670.

Dudley, R., & Kuyken, W. (2006). Formulation in cognitive therapy: A heuristic framework. In L. Johnstone & R. Dallos (Eds.), Formulation in psychology and psychotherapy. Brunner-Routledge.

Ensink, B. J. (1992) Confusing realities: A study on child sexual abuse and psychiatric symptoms. Amsterdam: Free University Press.

Field, A. (2005). Discovering statistics using spss (and sex drugs and rock n' roll) (2nd ed.). London: SAGE Publications.

Finucane, A., & Mercer, S. W. (2006). An exploratory mixed methods study of the acceptability and effectiveness of mindfulness based cognitive therapy for patients with active depression and anxiety in primary care. BMC Psychiatry, 6(14).

Fowler, D., Garety, P. A., & Kuipers, L. (1995). Cognitive behaviour therapy for psychosis: Theory and practice. Chichester: Wiley.

Freeman, D., Garety, P. A, Bebbington, P. E., Smith, B., Rollinson, R., Fowler, D., Kuipers, E., Ray, K., & Dunn, G. (2005). Psychological investigation of the structure of paranoia in a non-clinical population. British Journal of Psychiatry, 186, 427–435.

Garety, P. A., Fowler, D., Kuipers, E., Freeman, D., Dunn, G., Bebbington, P. E., Hadley, C., & Jones, S. (1997). The London-East Anglia Randomised Controlled Trial of Cognitive Behaviour Therapy for Psychosis II: Predictors of outcome. British Journal Of Psychiatry, 171, 420–426.

Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. Psychological Medicine, 31, 189–195.

Garrett, M., Stone, D., & Turkington, D. (2006) Normalizing psychotic symptoms. Psychology and Psychotherapy: Theory, Research and Practice, 79, 595–610.

Grassian, G. (1983). Psychopathology of solitary confinement. American Journal of Psychiatry, 140, 1450–1454.

Horvath, A. O. (1994). Research on the alliance. In A. O. Horvath & L. S. Greenberg (Eds.), The working alliance: Theory, research and practice (pp. 259–287). New York: Wiley.

Jacobson, N. S., K. S. Dobson, Truax, P. A., Addis, M. E., Koerner, K, Gollan, J. K. et al. (1996). A component analysis of cognitive-behavioral treatment for depression. Journal of Consulting and Clinical Psychology, 64, 295–304.

Janssen, I., Hanssen, M., Bak, M., Bijl, R. V., DeGraffe, R., Vollebergh, W., McKenzie, K., & Van Os, J. (2005). Discrimination and delusion idetion. British Journal of Psychiatry, 182, 71–76.

Johns, L., Singleton., N., Murray, R. M., Farrell, M., Brugha, T., Bebbington, P., Jenkins, R., & Meltzer, H. (2004). Prevelance and correlates of self reported psychotic symptoms in the British population. The British Journal of Psychiatry, 185, 298–305.

Kingdon, D. G., & Turkington, D. (1994). Cognitive-behavioral therapy of schizophrenia. New York: Guilford Press.

Kingdon, D. G., & Turkington, D. (2005) Cognitive therapy of schizophrenia. London: Guilford Press.

Kingdon, D. G., & Turkington, D. (1991). The use of cognitive behaviour therapy with a normalising rationale in schizophrenia: A preliminary report. Journal of Nervous & Mental Disease, 179, 207–211.

Krabbendon, L., & Van Os, J. (2005). Schizophrenia and urbanicity: A major environmental influence – Conditional on genetic risk. Schizophrenia Bulletin. 31,795–799.

Kuyken, W. (2006). Research and evidence base in case formulation. In N. Tarrier (Ed.), Case formulation in cognitive behaviour therapy: The treatment of challenging and complex clinical cases. London: Brunner Routledge.

Montgomery, S. A., & Asberg, M. (1978). A new depression scale designed to be sensitive to change. British Journal of Psychiatry, 134, 382–389.

Morrison, A. P. (1998). A cognitive analysis of the maintenance of auditory hallucinations: are voices to schizophrenia what bodily sensations are to panic? Behavioural & Cognitive Psychotherapy, 26, 289–302.

Morrison, A. P., Renton, J. C., Dunn, H., Williams, S., & Bentall, R. P. (2004). Cognitive therapy for psychosis: A formulation based approach. New York: Brunner Routledge.

Neuchterline, K. H., & Dawson, M. D. (1984). A heuristic vulnerability stress model of schizophrenic episodes. Schizophrenia Bulletin, 10, 300–312.

National Institute for Clinical Excellence (2002). Clinical guideline 1. Schizophrenia: core interventions in the treatment and management of schizoprenia in primary and secondary care. London: NICE

Oswald, I. (1974). Sleep (3rd ed.). Harmondsworth: Penguin.

Persons, J. B. (1989). Cognitive therapy in practice: A case formulation approach. New York: Norton.

Phelan, J. C., Cruz-Rojas, R., & Reiff, M. (2002). Genes and stigma: the connection between perceived genetic etiology and attitudes and beliefs about mental illness. Psychiatric Rehabilitation Skills, 6(2), 159–185.

Pote, H. L., & Orrell, M. W. (2002). Perceptions of schizophrenia in multi-cultural Britain. Ethnicity & Health, 7(1), 7–20.

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

Raue, P. J., & Goldfried, M. R. (1994). The therapeutic alliance in cognitive-behavioral therapy. In A. O. Horvath & L. S. Greenberg (Eds.), The working alliance: Theory, research and practice (pp. 131–152). New York: Wiley.

Read, J. (2004). A history of madness. In J. Read, L. R. Mosher & R.P. Bentall (Eds.), Models of madness. Hove: Routledge.

Read, J., Mosher, L. R., & Bentall, R. P. (Eds.). (2004). Models of madness. Hove: Routledge.

Salkovskis, P. M. (1996). The cognitive approach to anxiety: Threat beliefs, safety-seeking behaviour, and the special case of health anxiety and obsessions. In P. M. Salkovskis (Ed.), Frontiers of cognitive therapy (pp. 48–74). New York: The Guilford Press.

Salkovskis P. (1999). Understanding and treating obsessive compulsive disorder. Behaviour Research and Therapy, 37, 29–52.

Sensky, T., Turkington, D., Kingdon, D., Scott, J. L., Scott, J., Siddle, R. et al. (2000). A randomised controlled trial of cognitive-behavioural therapy for persistent symptoms in schizophrenia resistant to medication. Archives of General Psychiatry, 57, 165–172.

Shaw, B. F., Elkin, I., Yamaguchi, J. Olmsted, M., Vallis, T. M., Dobson, K. S., Lowrey, A., Sotsky, S., Watkins, J. T., & Imber, S. D. (1999). Therapist competence ratings in relation to clinical outcome in cognitive therapy for depression. Journal of Consulting and Clinical Psychology, 67, 837–846.

Slade, P. D. (1973). The psychological investigation and treatment of auditory hallucinations: A second case report. British Journal of Medical Psychology, 46, 293–296.

Strauss, J. S. (1969). Hallucinations and delusions as points on continua function. Archives of General Psychiatry, 21, 581–586.

Tabachnick, B. G., & Fidell, L. S. (2001). Using multivariate statistics (4th ed.). New York: Harper Collins.

Tarrier, N., & Calam, R. (2002). New developments in cognitive behavioural case formulation. Epidemiological, systemic and social context: An integrated approach. Behavioural and Cognitive Psychotherapy, 30, 311–328.

Trower P, Birchwood M, Meaden A. et al. (2004). Cognitive therapy for command hallucinations: Randomized controlled trial. British Journal of Psychiatry, 184, 312–320.

Turkington, D., & Kingdon, D. G. (1996). Using a normalising rationale in the treatment of Schizophrenic Patients. In G. Haddock & P. D. Slade (Eds.), Cognitive behavioural interventions with psychotic disorders. New York: Routledge.

Turkington, D., & Dudley, R. (2004). Cognitive-behavioural in the treatment of schizophrenia. Expert Review of Neurotherapeutics, 4 (5) , 861–868.

Van Os, J., Hanssen, M., Bijl, R. V., & Vollebergh, W. (2001). Prevalence of psychotic disorder and community level of psychotic symptoms: An urban rural comparison. Archive of General Psychiatry, 58, 663–668.

World Health Organisation. (1992). International classification of diseases (10th ed.). Geneva: WHO.

Wiles, N. J., Zammit, S., Bebbington, P., Singleton, N., Meltzer, H., & Lewis, G. (2006). Self-reported psychotic symptoms in the general population: results from the longitudinal study of the British National Psychiatric Morbidity Survey. British Journal of Psychiatry, 188, 519–526.

Zubin, J., & Spring, B. (1977). Vulnerability a new view of schizophrenia. Journal of Abnormal Psychology, 86, 103-126.