## Title
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## Authors
Loewy, Rachel
Hardy, Kate

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# Cognitive Behavioral Therapy for Adolescents at Clinical High Risk for Psychosis

Kate V. Hardy* and Rachel Loewy

*University of California, San Francisco, USA*

**Abstract:** Cognitive Behavioral Therapy (CBT) is an established adjunctive treatment for schizophrenia with a growing evidence base. More recently, CBT has been applied to individuals identified as being at risk for developing psychosis in an attempt to delay or prevent a transition to psychosis, to reduce symptoms and improve functioning. CBT has also been employed effectively with adolescents in the treatment of depression, post traumatic stress disorder (PTSD), anxiety disorders and eating disorders. This paper reviews the evidence for the use of CBT with the clinical high risk for psychosis population and addresses adaptations to the approach for use specifically with adolescents.

**Keywords**: Adolescents, clinical high risk, cognitive behavioral therapy, psychosis, psychosocial interventions.

Cognitive behavioral therapy (CBT) has been utilized in the treatment of schizophrenia as an effective adjunct to pharmacotherapy for over a decade. More recently, this approach has been adapted as an intervention for those identified as being at a high risk of developing psychosis. In this paper we will review the rationale for using CBT with this population, the evidence base for this approach and provide a case example of using CBT with an adolescent who is experiencing attenuated psychotic symptoms.

The basic premise behind early intervention for psychosis is to reduce the duration of untreated psychosis (DUP) and to provide treatments as early as possible in the course of the psychotic disorder. Shorter DUP has been associated with better long-term outcomes for individuals with schizophrenia, above and beyond a variety of confounding factors that might lead to earlier treatment, such as symptom severity, social support and comorbid conditions (Norman, Lewis, & Marshall, 2005). Providing interventions early, during the critical window after the first symptoms appear, takes advantage of the period during which individuals are thought to be the most receptive to pharmacological and psychosocial interventions, thereby achieving optimal outcomes. (Birchwood, Todd, & Jackson, 1998).

Wyatt (1995) suggested that exposure to untreated psychosis is responsible for long term morbidity while Thompson and colleagues (2001) mapped adolescent brain changes following early onset schizophrenia and reported a dynamic progression of brain change involving increasing amounts of cortex over time with later grey matter loss. Although others have suggested that exposure to untreated positive symptoms does not have a lasting neurotoxic effect (Ho *et al*., 2003; Perkins, Gu, Boteva, & Lieberman, 2005) it would appear that given the potential for disruption on the developing adolescent brain at a time critical to cognitive development, any interventions that can reduce exposure to fully psychotic symptoms may be of long term benefit (Yung & McGorry, 2007). There is early evidence suggesting that CBT for psychosis results in functional brain changes with Kumari and colleagues (2011) demonstrating that CBT for psychosis delivered over an average of sixteen sessions resulted in decreased activation in the inferior frontal, insula, thalamus, putamen and occipital areas during fMRI when exposed to fearful and angry expressions, as compared to baseline. Furthermore, the decreased activation was significantly correlated with reduced symptoms. This early data suggests that, as is the case with CBT for other psychiatric disorders such as depression and anxiety, CBT for psychosis can actually affect the neural substrate underlying psychosis, presenting a potential alternative to medication-only paradigms.

As discussed in detail elsewhere in this issue (Adelsheim, 2012; McFarlane *et al*., 2012; Pearson, Stuart, & Loewy, 2012) there has recently been a movement towards identifying individuals prior to the onset of psychosis, with an eye towards delaying or even preventing full onset of schizophrenia. Assessments and clinical criteria now exist that identify individuals in the stage prior to the onset of psychosis, although these assessments are not 100% accurate in predicting who will go on to develop a psychotic disorder. As such, the term *clinical high risk* (CHR) is typically used to avoid the connotation of inevitable conversion to psychosis that is associated with the term *prodromal* (French & Morrison, 2004). The aim of identifying individuals in this CHR period is to provide interventions that will delay or prevent the transition to psychosis, while targeting the symptoms for which the individual is seeking help. These include non-specific symptoms associated with the onset of psychosis, such as depression and social anxiety, as well as attenuated psychotic symptoms, which include unrealistic ideas or perceptual disturbances that are not yet fully believed to be real. A further potential benefit of identifying and treating people during this stage is that the individual is ideally placed to receive appropriate services should they

*Address correspondence to this author at the Department of Psychiatry, 401 Parnassus Avenue, San Francisco, CA, 94143, USA; Tel: 415 502 4557; Fax: 415-476-7719; E-mail: kate.hardy@ucsf.edu

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develop full psychosis, thus ensuring a shorter DUP (Yung *et al*., 2011) and a less traumatic, and hopefully less disruptive, transition to psychosis.

Conversion rates to full psychosis in CHR samples vary among studies and have recently been reported to range between 16% over two years (Yung, *et al*., 2008) and 35% over two and a half years (Cannon *et al*., 2008). This high level of false positives (people identified as being at risk of developing psychosis but who do not go on to develop a formal psychotic disorder) has led to debate in the field regarding the most appropriate treatments for the CHR population. Bentall and Morrison (2002) argue that anti-psychotic medications for this population do "more harm than good" and strongly advocate for the use of psychosocial interventions, such as CBT, which have less severe side effects, are less stigmatizing, provide a normalizing rationale and are targeted to the individual's presenting symptom profile. Yung and McGorry (2007) address the issue of false positives by proposing a clinical staging model reminiscent of interventions in general medicine, which were designed to identify and treat cancer at various stages of malignancy. They advocate for psychosocial interventions such as CBT to be used as indicated prevention for the CHR population with the use of anti-psychotic medications only indicated upon transition to full psychosis.

## CBT FOR THE CHR POPULATION: RATIONALE

CBT for schizophrenia has a growing evidence base and has been established as a recommended treatment for schizophrenia; for example, it is included in the schizophrenia treatment guidelines published by the National Institute of Clinical Excellence (NICE) in the United Kingdom (2009). In a meta-analytic review of 34 randomized controlled trials, Wykes and colleagues (2008) concluded that CBT for psychosis is associated with improvements in positive symptoms, negative symptoms, and overall functioning. More recently Sarin and colleagues (2011) conducted a meta-analytic review of 22 blinded randomized controlled trials to determine the effectiveness of CBT for psychosis at the end of treatment and follow up. They concluded that there was a trend in favor of CBT at the end of treatment which became statistically significant at follow up, with CBT for psychosis showing a small effect size on positive symptoms, negative symptoms and general symptoms. The authors concluded that CBT for psychosis has a delayed impact that can be seen several months after therapy is terminated and suggested that the small effect size seen earlier was due to the positive effects of supportive therapy, which is often used as a comparison group. Thus, non-specific supportive intervention leads to clinical improvement in the short term, but the improvement is not maintained over time compared with CBT, which continues to benefit the individual after therapy.

Given the evidence for the effectiveness of CBT in fully psychotic disorders, investigators have naturally extended this intervention to working with clients experiencing the attenuated and/or intermittent psychotic symptoms associated with the CHR syndrome. This extension to the CHR group is particularly appropriate given the focus of CBT on reducing distress associated with psychotic symptoms (Birchwood, Iqbal, Jackson, & Hardy, 2004). Distress about psychotic symptoms may be a factor in increased psychotic experiences; for example distress about hallucinatory experiences in the general population has been shown to predict later development of delusions (Hanssen, Bijl, Volleburgh, & van Os, 2003). Therefore, CBT may serve to reduce symptoms in CHR syndromes, improve functioning, and may even delay or reduce transition to psychosis by reducing distress related to attenuated psychotic symptoms, although this possibility has not yet been formally tested.

Depression and anxiety are often associated with the prodromal period (Møller & Husby, 2000) and CBT is an established treatment for these disorders in adolescents (James, Soler, & Weatherall, 2005; Lewisohn & Clarke, 1999) and adults (Butler, Chapman, Forman, & Beck, 2006). Therefore, by using a CBT framework for the CHR population, it is possible to draw upon established CBT treatment models to target non-specific symptoms. CBT for the CHR population is based on a stress-vulnerability model allowing individuals to understand their difficulties, including mood, anxiety and psychotic-like symptoms, in the context of biological vulnerability combined with environmental stress. Thus interventions aim to teach strategies to identify and deal with environmental stressors with the intention of decreasing the stress experienced by the individual.

A key principle of CBT is to provide psychoeducation within a normalizing framework. Sufficient evidence is now available to suggest that psychotic-like experiences exist on a continuum with normal human experience. Up to 18.1 % of adults in the general population report psychotic experiences outside of formal psychotic disorders (Hanssen *et al*., 2003). Psychotic-like experiences in adolescents are especially common, with 8.4% of adolescents reporting hallucinations (Scott, Martin, Bor, Sawyer, Clark, & McGrath, 2009). Given this evidence, normalization can help people to conceptualize their experience as within the realm of usual experience, thus decreasing the self-stigma and fear typically associated with these symptoms.

## CBT FOR THE CHR POPULATION: RANDOMIZED TRIALS

To date, there are six published studies of four randomized controlled trials of CBT for those at clinical high risk for developing psychosis. Two of these examine the effect of CBT alone (Morrison *et al*., 2004, Addington, Epstein, Liu, French, Boydell, & Zipursky, 2011) while the studies originating from the PACE (Personal Assessment and Crises Evaluation) Clinic in Melbourne, Australia examine CBT in combination with an anti-psychotic medication (McGorry *et al*., 2002). One of the PACE studies also includes a placebo medication arm for comparison (Yung *et al*., 2011). Table 1 details a comparison of these studies' design and outcomes. Of particular relevance to this review, the mean age in the CHR CBT studies was around 20 years old, in the period of early young adulthood. Study entry criteria spanned adolescence and young adult years, requiring participants to be aged between 14 and 30 (McGorry *et al*., 2002, Yung *et al*., 2011, Addington *et al*., 2011) or 16 and 35 (Morrison *et al*., 2004).

**Table 1. CHR CBT Intervention Studies**

| Author and year | Inclusion Criteria | Age (years) | Intervention | Conversion to psychosis | Symptom Outcomes |
|---|---|---|---|---|---|
| McGorry *et al*., (2002)<br>Phillips *et al*., (2007) | Age 14-30, Ultra High Risk (UHR) identified by 1 or more of:<br>Family history of psychosis plus a decrease in functioning<br>Attenuated psychotic symptoms > 1 week<br>Brief fully psychotic symptoms <1 week | Mean = 20<br>Range = 14 - 28 | Active Treatment = Cognitive Therapy plus low dose atypical antipsychotic (risperidone) (n= 31) for six months<br>Control group = Care management<br>(n= 28)<br>Active Treatment:<br>Cognitive Therapy plus low dose atypical antipsychotic (risperidone) for six months n= 24 at follow-up<br>Control Group:<br>Care management n=17 at follow-up | End of treatment: Transition to psychosis<br>10% in active treatment group (n=3),<br>36% control group (n=10);<br>(Fisher exact test p=.03).<br>After 6 months follow-up: Transition to psychosis<br>19% in active treatment group (n= 6),<br>36% control group (n=10);<br>(p=.24). NS<br>After 36 months follow up: Transition to psychosis<br>16% in active treatment group (n=4)<br>11.8% in control group (n=2)<br>NS (p=0.19) | No significant difference between groups on:<br>BPRS<br>HRSA<br>HRSD<br>MRS<br>QLS<br>SANS<br>GAF<br>No significant difference between groups on:<br>BPRS<br>HRSA<br>HRSD<br>QLS<br>SANS<br>GAF<br>Compare converted vs. non-converted<br>Active treatment group less negative symptoms (F=12.73; p=0.004) |
| Morrison *et al*., (2004)<br>Morrison *et al*., (2007) | Age 16 – 35, UHR identified by 1 or more of:<br>Family history of psychosis plus a decrease in functioning<br>Attenuated psychotic symptoms > 1 week<br>Brief fully psychotic symptoms <1 week | Mean = 20.6 (therapy)<br>21.5 (control)<br>Range = 16 - 36 | Active treatment CT over 6 months+ monitoring over 12 months (n=35)<br>Control group<br>Monitoring only over 12 months (n=23)<br>Active treatment n=17 at follow-up<br>Control group<br>n= 10 at follow-up | End of treatment: Transition to psychosis<br>6% in active treatment (n=2),<br>22% in control group (n=5)<br>96% reduction in odds of making transition to psychosis when exposed to CT (Odds ratio = 0.04, p = 0.028)<br>After 36 months follow up: Transition to psychosis<br>20% in active treatment (n=2),<br>22% in control group (n=5)<br>NS (p=0.79) | No significant difference between groups on:<br>GHQ<br>GAF<br>CT group showed significantly fewer positive symptoms compared with monitoring group (F=4.09; p=0.049) over 12 months monitoring period<br>87% reduction in prescription of anti-psychotics in CT group (p=0.24) |
| Yung *et al*., (2011) | Age 14-30, Ultra High Risk (UHR) identified by 1 or more of:<br>Family history of psychosis plus a decrease in functioning<br>Attenuated psychotic symptoms > 1 week<br>Brief fully psychotic symptoms <1 week | NR | Active Treatment = CT plus risperidone (n=36), CT plus placebo (n=35) provided up to 12 months<br>Control Groups = Supportive therapy (ST) plus placebo (n = 22)<br>Monitoring only (n=37) up to 12 months | 6 Month Follow up: Transition to psychosis<br>4.7% in CT+ risperidone (n=2),<br>9.1% in CT + placebo (n=4),<br>7.1% in ST + Placebo (n=2)<br>5.1% in monitoring (n=4)<br>NS (p=0.93) | No significant difference between groups on:<br>BPRS<br>HRSA<br>HRSD<br>QLS<br>SANS<br>GAF |
| Addington *et al*., (2011) | Age 14 – 30, Meet Criteria of Prodromal State (COPS) on the Structured Interview of Prodromal Symptoms (SIPS) | Mean = 20.8 (CT) & 21.1 (ST)<br>Range - NR | Active Treatment = CT (n = 27) provided for up to six months<br>Control Group = Supportive therapy (n=24) up to six months | Up to 12 months follow up: Transition to psychosis<br>0% in CT,<br>12.5% in ST (n=3)<br>NS (p=0.059) | Decline in positive symptoms for both groups by 6 months<br>CT improved faster over five months follow up (t= 3.95 to 5.2; p<.05 to p<0.0001) |

BPRS = Brief Psychiatric Rating Scale, HRSA = Hamilton Rating Scale for Anxiety, HRSD = Hamilton Rating Scale for Depression, QLS = Quality of Life Scale, SANS = Scale for the Assessment of Negative Symptoms, GAF = Global Assessment of Functioning, GHQ = General Health Questionnaire, CT = Cognitive Therapy, ST = Supportive Therapy, NR = not reported.

Early studies reported optimistic results suggesting that CBT was effective in preventing the onset of psychosis with statistically significant low transition rates at the end of treatment in treatment versus control groups. In one study, 10% in the CBT plus risperidone group compared with 36% in the control group converted to psychosis (McGorry *et al*., 2002); in another the conversion rates were 6% in the CBT group versus 22% in the control group (Morrison *et al*., 2004). However, the difference between groups on transition rates was not maintained in either study at 36-month follow up, with 16% in the CBT plus risperidone group and 11.8% in the control group (Phillips *et al*., 2007), and 20% in the CBT group compared to 22% in the control group for the second study (Morrison *et al*., 2007). Unfortunately, the final sample sizes for these studies were small (N = 58 in both studies) and the findings of post-treatment differences in transition rate were not replicated in later studies (Yung *et al*., 2011, Addington *et al*., 2011).

Low sample sizes and a global trend towards lower conversion rates (Yung *et al*., 2007) may be partly responsible for some of the null findings. Yung and colleagues (2007) posit that this reduction in conversion to psychosis may be due to earlier detection of the CHR syndrome and subsequent earlier intervention. They also suggest that decreasing conversion rates may be a result of identifying more false positive individuals who were never at risk of developing full psychosis thus diluting the sample. As such, a full scale multi-site randomized control trial is required to fully test the efficacy of CBT for CHR syndromes. Non-significant results at follow-up may suggest a need to provide booster sessions following the intensive period of intervention.

Although conversion to psychosis is often measured as the primary outcome in CHR intervention trials, it is important to consider outcomes related to symptom severity across domains as well as functioning. Morrison and colleagues (2004) reported that the group that received CBT experienced a reduction in positive symptoms compared to the control group over a 12 month period (6 months active treatment for CBT group plus 6 months monitoring versus 12 month monitoring for controls). Addington and colleagues (2011) described an earlier and faster reduction in positive symptoms for the CBT group compared to the control group between months 1 and 5 of treatment; however, by the end of six months there was no statistically significant difference in positive symptoms between the groups. As described previously, interventions that reduce the duration of exposure the individual has to fully psychotic positive symptoms may be beneficial. Across all studies there was no difference between groups on measures of functioning post-treatment, nor at 36-month follow-up, possibly indicating that the interventions used were targeted specifically to attenuated psychotic symptoms, but may not be sufficient to affect functioning.

Due to the early stage of research into this population, there is little known about the effectiveness of these interventions specifically for adolescents and further research is required to assess the acceptability of this approach with a younger population. Morrison and colleagues (2004) reported that the high rate of consent (95%) and low attrition rate (14%) in an adult population suggest that the intervention was acceptable to their population. The authors also argue that the number of sessions received, which was lower than the maximum available, reflected the collaboratively derived contract for a number of sessions based upon a problem list rather than difficulties with engagement. However, further specific assessment of client satisfaction of this approach is required with a particular focus on the components of CBT and their acceptability to clients of different ages; for example, do collaborative decisions to end the treatment reflect a measurable change in symptoms, functioning or distress levels? Do school age clients appreciate the homework setting element of CBT or do they regard it as burdensome, as they might school-distributed homework?

## MODELS OF CBT FOR CHR

To date, there are two individual CBT models for the at-risk population that have been described in the published literature. Although both models have their genesis in CBT for schizophrenia and emphasize an individual approach, there is variation in the theoretical foundation and application of change strategies. The model developed at the PACE Clinic in Melbourne, Australia, and described by Phillips and Francey (2004) has its roots in a stress-vulnerability model of psychosis and promotes stress management through identification of stressors and development of stress management techniques. This model draws upon established treatments for first episode and chronic schizophrenia (Garety. Kuipers, Fowler, Freeman, & Bebbington, 2001) to address cognitive biases and appraisals and encourages clients to develop an understanding of their symptoms and ways to manage them, covering four treatment modules: 1) stress management, 2) depression/negative symptoms, 3) positive symptoms, and 4) other co-morbidities.

The model utilized at the Early Detection and Intervention Evaluation (EDIE) clinic, in Manchester, United Kingdom, is based on the cognitive model of psychosis (Morrison, 2001) and is described in a treatment manual (French & Morrison, 2004). This model focuses on the interpretation of and response to psychotic experiences and emphasizes strategies to alter the interpretation of the event in order to decrease the distress associated with this interpretation. An individually tailored case formulation is collaboratively developed with the client within the first few sessions with the aim of increasing understanding of the experiences and identifying specific treatment targets. Although not a modular approach, this model covers several target areas including normalization of experiences, development of a problem list, treatment of co-morbid disorders, altering metacognitive beliefs and the development of a relapse prevention plan.

In addition to these models, Valmaggia and colleagues (2008) describe an intervention for an individual at risk of developing psychosis. Through a series of case studies they explore the different treatment targets across the different stages of psychosis. They assert the need to attune the generic CBT approach to the specific stages in the development of psychosis and do this with a focus on co-

morbidity when working with the client at risk for psychosis, using an established model by Clark and Wells (1995) to address symptoms of anxiety. This approach demonstrates the utility of adapting and applying existing models of CBT for other disorders to target identified symptoms with which the CHR client presents.

## CBT WITH A CHR ADOLESCENT: A CASE EXAMPLE

The following case example describes an adolescent at clinical high risk for psychosis who participated in a CBT treatment based on the intervention described by French and Morrison (2004). The case is an amalgam of clients and identifying information has been altered, in order to protect privacy.

Matthew is a 15 year old African-American male who, until recently, had performed well at school. He enjoyed sports and had a good circle of friends whom he saw a couple of times a week outside of school. A year ago, his oldest brother, Michael, had been hospitalized following an acute psychotic episode while away at an Ivy League college. Although Matthew's parents report they had a sense something was "wrong with Michael" his hospitalization and subsequent diagnosis of schizophrenia came as a shock to Matthew. Michael returned to live in the family home nine months ago. In the last six months Matthew has become withdrawn, he has stopped socializing with his friends and his teacher contacted the family because she was concerned about his drop in grades. His parents report that he used to be an outgoing boy, but that he has become withdrawn and sullen, resisting all efforts by his parents to engage him in activities. He spends most of his time when he is at home in his room, on the computer and listening to music at a loud volume. His mother is concerned and states that she would have just dismissed this behavior as Matthew being "a typical teenager" if it weren't for Michael's recent diagnosis.

Matthew's recent deterioration in functioning (social withdrawal, poor academic performance) and first degree relative with a diagnosis of schizophrenia indicated a risk of developing psychosis. Careful assessment using the Structured Interview for Prodromal Symptoms (McGlashan, Miller, Woods, Rosen, Hoffman, & Davidson, 2001) indicated that he is also experiencing attenuated positive symptoms in the form of perceptual disturbances. Matthew grudgingly disclosed that he has been hearing "muffled sounds like voices" for approximately three months three to four times a week. He said that he couldn't discern specific words but thought they were getting louder. Although he was pretty sure they were not real he wondered if they were trying to communicate something to him. He said he becomes upset talking about these "noises" and explained that he overheard his brother talking to his parents about the voices he hears and has since become convinced he is "going crazy like his brother."

Matthew was a reluctant participant in therapy but agreed to meet for one session to "shut his Mom up." The therapist explored what Matthew understood about therapy and, not surprisingly, Matthew expected it to be "boring" and "like being lectured." Socialization to the model is a key feature of CBT and involves a thorough explanation of CBT to the client, thus asserting a collaborative relationship from the beginning of therapy and de-mystifying the process. The basic premise of socialization is an explanation of how thoughts influence emotions and behaviors. How this is undertaken is dependent upon the client. Use of real life examples that are meaningful to the client will result in the most insight into the model.

*Therapist*: Imagine you walk into a store and a girl you like from school is in the store. She looks over to you and smiles. Your first thought is "yeah, I'm looking good today, I'm glad I'm wearing my new jacket." How might you feel?

*Matthew*: Pretty good.

*Therapist*: Yeah, and what might you do?

*Matthew:* Smile back.

*Therapist*: Now imagine the same situation but this time you think "what is she looking at? Did she notice the zit on my chin? What if she talks to me? I'm going to sound stupid." How might you feel?

*Matthew*: Pretty bad probably.

*Therapist*: Bad? How?

*Matthew*: Nervous maybe.

*Therapist*: Ok, and what do you think you might do? Would you smile at her?

*Matthew*: No! I'd probably leave the store real quick.

The therapist then drew parallels between this imaginary scenario and the premise of CBT that interpretation of a situation can alter how a person feels and how they act.

Another important aspect of socialization to the model is the explanation of the structure of CBT. French and Morrison (2004) assert that "when working with a pre-psychotic client group, the usual structure of CT should be adhered to" (p32). However, this structure, which includes agenda setting to ensure a mutually determined focus for the session, should be fully explained to the client. An adolescent, or any client, who finds this format too restrictive should be encouraged to add an agenda item of "free time." This can be negotiated to occur at the end of the session or part way through. Lewisohn and Clarke (1999) have suggested that CBT for *adolescents* needs to be adapted to be less structured and incorporate more flexibility in order to counteract the expectation of the adolescent of an authoritarian therapist. This might include altering the length and frequency of the sessions, utilizing creative methods to aid engagement such as board games, computer time, walking outside together, or scheduling breaks within the session. With this in mind, Matthew and the therapist mutually developed an agenda for the session which included a ten minute break half way through in which time Matthew showed the therapist his favorite online computer game using the computer in the office.

Following socialization to the model, the therapist elicited a problem list from Matthew which included, in order of priority for Matthew: 1) his brother being back at

home, 2) hearing noises, 3) not seeing his friends anymore and 4) not doing well at school. The therapist validated the number of stressors in Matthew's life currently. When asked, Matthew agreed to take a look at how these stressors were affecting him. CBT for the CHR population derives in part from a stress-vulnerability model (Zubin & Spring, 1977). A common way of describing this to clients is to explain that an individual vulnerable to experiencing a certain disorder is more likely to do so if they are placed under stress, whereas someone who is less vulnerable may require higher levels of stress to experience the same symptoms. This can be displayed graphically or presented in a narrative.

However, a more engaging view of this model is the "stress bucket." The individual's vulnerability can be conceptualized as the size of the bucket and the amount of stress the person is reporting as the water filling the bucket. This was utilized with Matthew in the following manner:

The therapist brought out an empty container and a slightly larger container filled with water on a tray.

*Therapist*: Remember that list of problems we just came up with? Well, I want us to think about how those problems fit into your stress bucket. Your stress bucket is the amount of stress you can take in your life before it becomes a problem. Everyone has one and the different things going on can determine how much your bucket can take.

*Therapist* (indicating to Matthew to take the two containers): The empty container is your stress bucket and let's imagine your problems are the water.

*Matthew*: Uh huh.

*Therapist*: Now remind me, what was number one on your problem list?

*Matthew:* (mumbles) My brother being back home.

*Therapist*: Ok, how much stress does that cause in your life? How much does it fill your bucket?

*Matthew*: A lot! About half. (Matthew starts pouring water into the empty container.)

*Therapist*: Wow, that bucket is looking pretty full already, what about hearing the noise?

*Matthew*: Yeah, that adds to the stress (pours more water into the container), and not seeing my friends adds more (pours more water into the container until it is nearly full).

*Therapist:* Looks like the bucket is full and you haven't added the last problem you identified yet. Do you want to add the water?

*Matthew*: But it will overflow.

*Therapist*: That's OK, the tray will catch the water. What do you make of the fact that the bucket wasn't big enough to hold all the water?

*Matthew*: I guess there's too much stress in my life right now.

*Therapist*: Looks like it. How about we really focus on what you already do to decrease that stress in your life currently, and what you can do in the future?

This approach allowed exploration of the strategies Matthew had already implemented to decrease his stress along with an evaluation of the effectiveness of the strategies. For example, Matthew identified that in order to cope with having his brother at home he spent a lot of time in his room to avoid having to talk to him. Matthew was worried that talking to his brother would be overwhelming because he felt uncomfortable around his brother. Closer examination showed that while retreating to his room was effective in avoiding his brother, Matthew spent his time in the room ruminating about having his brother home. Thus he was able to see that while staying in his room decreased his anxiety in the short term, it led to increased anxiety overall. Matthew was willing to try an "experiment" relating to this problem to explore his anxiety around talking to his brother. This was set up initially through a role play with the therapist playing Matthew's brother and helping Matthew think of topics in which he would like to engage his brother. Matthew rated the anxiety he expected to feel before, during and after talking to his brother and agreed to try this twice over the next week. When Matthew returned he was able to report that he had spoken to his brother and his level of anxiety was as he expected before and during the conversation but was much lower than he had anticipated following the conversation. As a consequence, he had spent more time in the living room where his brother spent most of his time watching the television.

Following this successful initial intervention it was agreed to explore possible explanations for his current concerns relating to the noises he heard. Fig. (**1**) shows this formulation, developed collaboratively between Matthew and the therapist, which was based upon Morrison (2001). The formulation was used to explore his interpretation of intrusions, in this case the auditory perceptual disturbances he experiences. Through the formulation Matthew was able to identify that he interpreted the noises he heard as being a sign of impending mental illness based upon his brother's recent diagnosis ("I'm going crazy"). Matthew also identified that he had tried all his life to be like his brother but that his older brother had always been better than him at school and in sports no matter how hard he tried. From these experiences had arisen beliefs about himself and the world including the beliefs "I am not good enough" and "things are out of my control," as well as the more recently formed belief, "I am different," as a consequence of the perceptual disturbances he experienced. In order to engage Matthew directly in the process of developing the formulation and to ensure that this portion of the therapy did not reflect his previous fears that therapy would be "lecturing," he was encouraged to draw the formulation himself, using his own language and altering it until it accurately described his experience. This formulation allowed Matthew to see that things could change at different levels (interpretation of the intrusion, beliefs about self and others and cognitive and behavioral consequences) with an initial focus on how he interprets the noises. Once the formulation was developed the therapist and Matthew started to identify possible interventions.

*Therapist*: Matthew, from what you've drawn there it looks like your interpretation "I am going crazy" is causing

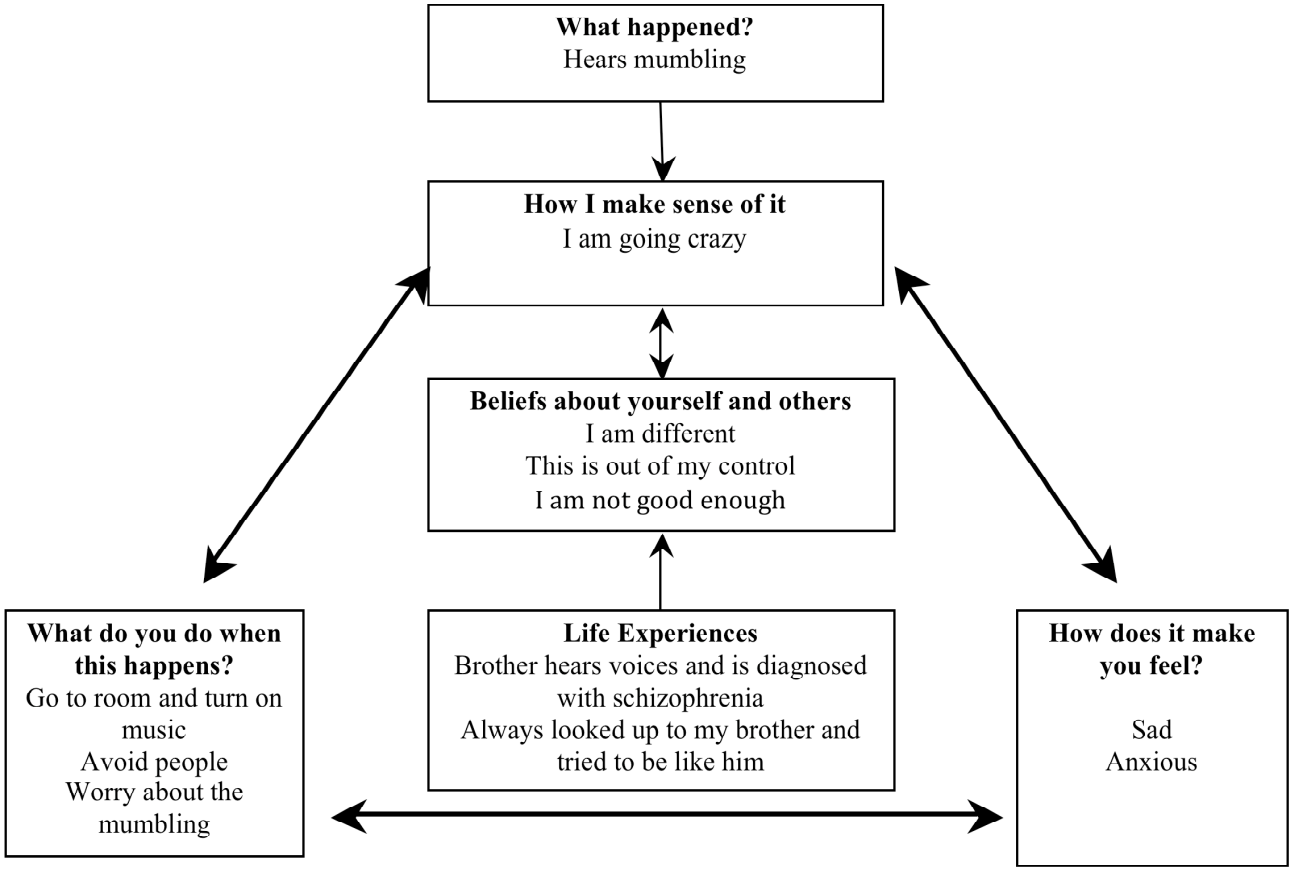


**Fig. (1).** Formulation for Matthew Based on Morrison (2001).

you a lot of worry and really getting in the way of things. What if you had a different interpretation of the situation?

*Matthew*: Like what? I hear noises and that means you're crazy.

*Therapist*: Well, for some people hearing voices is a sign of mental health problems, but I wonder if there are other people out there who hear things but don't have mental health problems? Given this is something that's causing you a lot of concern, maybe it's something we should look into together so that we can be sure that you have all the information necessary to make that interpretation?

The therapist went on to provide psychoeducation regarding the prevalence of hearing voices and auditory hallucinations in a non-psychiatric population. Matthew was given homework to monitor when he heard the noises and he found that it typically occurred when he was struggling with school work and when he was up late on his computer. Taking this new information into account, Matthew was encouraged to generate an alternative interpretation of the noises and to complete a formulation using this new interpretation (see Fig. **2**). Future sessions focused on continuing to explore alternative interpretations as well as evidence for and against his beliefs about himself. In addition, a family session was arranged, with Matthew's consent, to provide psychoeducation regarding the CHR period and to look into support for the family, including Matthew's older brother.

## FUTURE DIRECTIONS

It is still early in the development and evaluation of interventions for the CHR population. Although promising results have emerged from trials, there needs to be further large scale research to address the small sample sizes and decreasing conversion rates reported globally (Heinssen, Cuthbert, Breiling, Colpe, & Dolan-Sewell, 2003; Yung *et al*., 2007), as well as to fully test the durability of treatment effects and identify who is most likely to benefit from CBT for CHR. Although the two CBT for CHR models presented, and the case study, all originate from the same theoretical background of CBT, there is distinct variation between the models. As such, it is difficult to ascertain the active ingredients of each model and further investigation is required to explore the essential components of CBT needed to effect change.

As discussed, it is necessary to explore the utility of this intervention for a younger adolescent population. Extrapolating from research employing CBT for other mental health problems suggests that this is an acceptable treatment approach for adolescents but this must be explored further. Drawing upon existing models of CBT for adolescents and expertise in adapting treatment approaches to specific developmental stages may allow increased

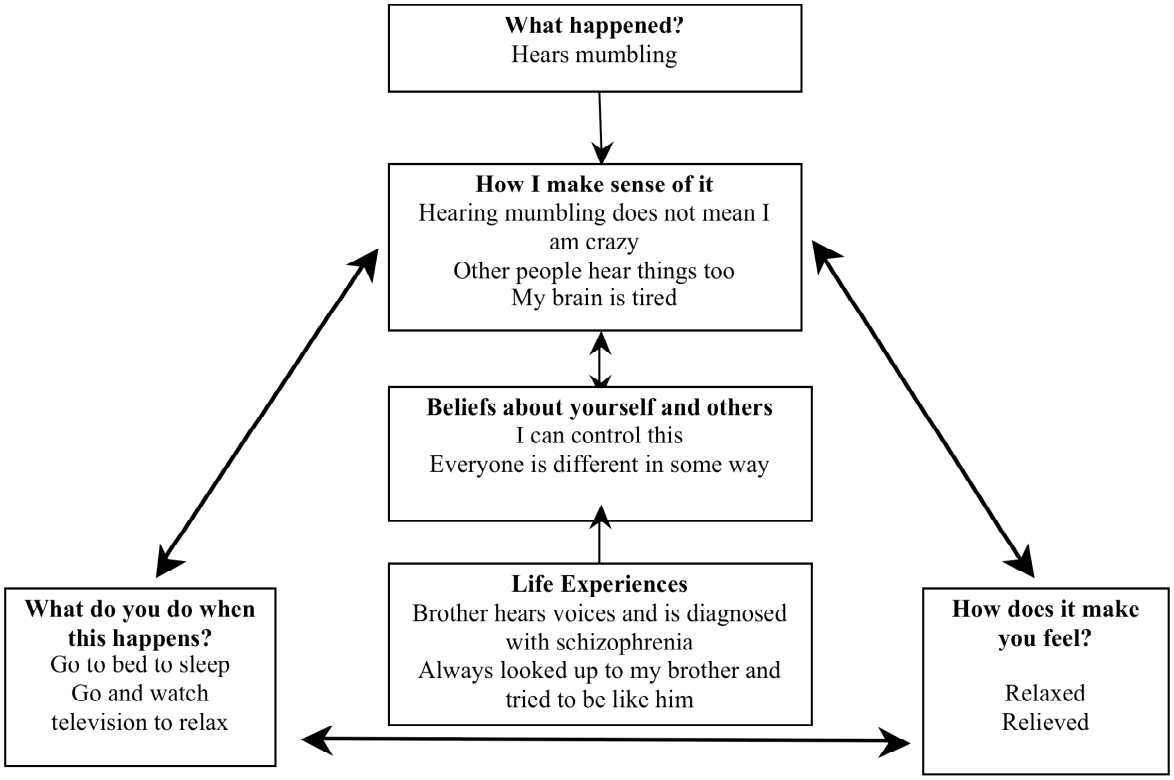


**Fig. (2).** Formulation for Matthew using alternative interpretation.

generalizability of this approach, thus improving accessibility and appropriateness of this treatment for the adolescent population.

However, accessibility can only be improved if this treatment is readily available in community settings. Addington and colleagues (2011) suggested that the non significant difference between the two groups (supportive therapy versus cognitive therapy) was due to the lack of experience of their trial therapists who did not have a doctoral level of specialist CBT experience. They recommended that interventions targeted at the CHR population should be undertaken by specially trained and experienced therapists. However, this is potentially problematic in community-based clinical programs that are predominantly staffed by Master's level clinicians. Training clinicians already familiar and experienced in working with adolescents within a CBT framework may help to overcome this issue, but education and training is necessary to help non-specialist clinicians to overcome possible misperceptions and prejudice about providing psychotherapeutic interventions for psychotic spectrum disorders. Valmaggia and colleagues' (2008) case study, which emphasizes treatment of co-morbidities in the CHR population, elucidates that basic CBT skills in treating anxiety and depression in adolescence can be applied to this population with the addition of specialist knowledge regarding the CHR period. Future research should specifically assess whether Master's level clinicians can implement CBT to fidelity, in order to ensure accessibility of this intervention to the general public (Hardy *et al*., 2010).

## CONCLUSION

This article has reviewed the evidence for the use of CBT with the CHR population. There is data to suggest that providing psychosocial interventions prior to the onset of psychosis is indicated on the grounds of preventing or delaying a transition to psychosis, although the evidence for efficacy of this approach is currently mixed and needs additional research. Psychosocial treatments are generally preferable with this population given the high proportion of false positives and low side effect profile associated with this type of intervention compared with anti-psychotic medications. These considerations become especially pertinent when treating adolescents, who are particularly sensitive to anti-psychotic medication side effects (Ratzoni *et al*., 2002; Correll & Carlson, 2006). CBT already has established efficacy with adolescents in the treatment of depression and anxiety (James *et al*., 2005), and we have tried to illustrate the similarity of CBT for CHR to CBT for other disorders in adolescence. We hope that this paper encourages practitioners seeing CHR adolescents to proactively address these symptoms through CBT.

## AUTHOR INFORMATION

Kate Hardy, Clin.Psych.D., Clinical Psychologist and Training Director, Prevention and Recovery from Early Psychosis (PREP).

Rachel Loewy, Ph.D., Assistant Adjunct Professor of Psychiatry and Co-Director, Prodrome Assessment Research and Treatment (PART) Program.

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