A glance at the recent history of the treatment of psychoses allows us to identify various stages and draw certain conclusions. Prior to the 1950s, attention to these disorders consisted fundamentally in simply locking the affected people away. Over the course the 1950s and in the 1960s, however, with the advent of the first-generation antipsychotics (chlorpromazine, fluphenazine and haloperidol), substances included on the WHO list of essential medication (World Health Organization, 2009), there emerged the hope of being able to control the positive symptoms – that is, the most pathological characteristics such as hallucinations and delusions – and to treat seriously affected patients in the community, reducing the frequency and severity of psychotic episodes and improving levels of functioning and quality of life. Moreover, on the assumption that negative symptoms such as apathy, lack of motivation, affective impoverishment and functional impairment might be the result of prolonged confinement of such patients in psychiatric institutions, there was optimism that once the positive symptoms had been controlled,
and on allowing patients to live at home with their families and in more natural environments in the community, such symptoms would not emerge.

In the 1970s, however, the outlook was distinctly less optimistic, as it was found that the side effects of antipsychotic medication meant that many patients failed to adhere to their therapeutic programme; there was low tolerance of extrapyramidal symptoms (especially akathisia and tardive dyskinesia), and relapse rates reached as much as 30-50% in the year to two years following the start of treatment. Moreover, in a small proportion of medicated patients there was no improvement of acute symptoms.

The introduction of atypical (or second-generation) antipsychotics appeared to improve therapeutic adherence, on achieving a significant reduction of motor side effects in both the short and long term (despite the emergence of some adverse effects, especially metabolic effects). In particular, clozapine showed no unwanted extrapyramidal effects and was used with some success in patients refractory to other treatments, but its use was limited by other undesirable consequences, such as agranulocytosis. In general, large-scale comparisons of the results of first- and second-generation antipsychotics fail to suggest substantive progress with regard to effective control of symptoms or improvements in the cognitive and social functioning of the patients treated (Jones et al., 2006; Keefe et al., 2007; Lieberman et al., 2005; Swartz et al., 2007). In any case, it has also been demonstrated that the failure to treat psychosis with drugs, especially in the case of schizophrenia, leads to more symptomatic relapses, impaired social functioning and quality of life and a marked increase in mortality, by comparison with patients receiving pharmacological treatment (Tandon, Nasrallah, & Keshavan, 2009).

The work of Brown, Birley and Wing (1972) and of Vaughn and Leff in the 1970s and 80s (Leff, 1987; Vaughn & Leff, 1976) opened up a new line of research and extended the therapeutic intervention framework, on proposing the existence of stress factors, potentially present in patients' homes, which could be related to high symptomatic relapse rates. Such work gave rise to the fruitful construct of expressed emotion, which attracted considerable interest from clinicians and researchers. Expressed emotion refers to certain behaviours of family members living with a patient, characterized by high levels of criticism, hostility and emotional over-involvement usually directed towards him or her, and which have been shown to correlate strongly with exacerbation of psychotic symptoms and with a need for rehospitalization.

In this same period, and integrating these findings, there emerged the vulnerability-stress model, proposed by Zubin and Spring (1977), as an explanation of schizophrenia. This model, superseding radically biological perspectives, suggests that schizophrenia is the result of endogenous factors of predisposition or vulnerability, genetic or acquired during neurodevelopment, and that the manifestation of symptoms of the illness is triggered by the presence of environmental stressors. Thus, psychosis could be the result of certain predispositional constitutional characteristics (innate or acquired in critical periods of early development), but which develop into this disorder only when the person is subjected to problematic or stressful life circumstances.

By and large, all subsequent research on the aetiology of schizophrenia, as well as the development of therapeutic intervention programmes, have been influenced both by this model and by family-based research on expressed emotion. Particular research attention has been given to the combination of pharmacological therapy and family intervention over the last few decades.

The vulnerability-stress model, then, conceives psychotic symptoms as resulting from the combined action of environmental stressors and personal vulnerability. Markers of vulnerability would include deficient information processing, revealed through diverse laboratory tasks; biochemical factors as yet insufficiently well identified; and deficits in social skills and the processing of affective signals. Added to these, subsequently, would be poor adherence to pharmacological treatment, once it had been prescribed. These factors also increase the emergence of environmental stressors or amplify their effects; and among the environmental stressors would be drug abuse and diverse problematic life events and critical, hostile or excessively anxious family environments.

PSYCHOLOGICAL TREATMENTS

Traditionally, negative symptoms of psychosis have been addressed with psychological therapies, which are based on the training of skills (mainly cognitive and social), or by means of reinforcement techniques in institutional contexts. There is a widely held belief that positive symptoms can only be contained or attenuated through pharmacological treatment (classical or new-generation antipsychotics), though other drugs have also been used as adjunctive treatment in psychoses. Thus, there are reports of some success in the treatment of
schizophrenia with other substances, including anticonvulsants, antidepressants, lithium (normally employed in bipolar disorders as a mood stabilizer), and benzodiazepines (which are used to treat anxiety disorders). In general, however, and with the exception of antipsychotics, evidence of the efficacy of these is weak (Buchanan et al., 2010).

Although antipsychotic medication has continued to be the principal treatment of choice for psychotic disorders, it has been shown how pharmacotherapy, as stand-alone treatment, produces only quite limited improvements in negative symptoms, in cognitive functions, in social functioning and in patients’ quality of life. Many patients even continue to show persistent positive symptoms and to relapse, especially when therapeutic adherence is relaxed or abandoned.

Thus, pharmacotherapy does not provide all the answers to psychotic disorders, if we consider treatment failures, residual symptoms, chronicity and recurrence of symptoms. For example, the degree of improvement in positive symptoms in schizophrenia, treated with antipsychotics, is typically less than 20% (Khan, Khan, Leventhal, & Brown, 2001). Even today it is still found (with 5-year follow-ups) that 50% of patients with schizophrenia present poor outcome, despite taking antipsychotic medication; and that 75% take their medication erratically, due, among other factors, to unpleasant side effects. Also, the relapse rate in bipolar disorder, treated with mood stabilizers, is between 30% and 40% over a period of 1-4 years (Geddes, Burgess, Hawton, Jamison, & Goodwin, 2004; Ginsberg, 2006), while the remission rate for major depression, treated with antidepressants, is 13% to 37%, depending on the number of drugs (Rush et al., 2006).

Therefore, authors have stressed the need to include psychosocial therapies complementary to pharmacotherapy, with a view to alleviating symptoms, improving therapeutic adherence, social functioning and quality of life, and helping patients to live independently (Kern, Glynn, Horan, & Marder, 2009; Patterson & Leeuwenkamp, 2008; Vallina Fernández, Lemos Giráldez, & Fernández Iglesias, 2010). The psychological treatments, applied at an individual level, which enjoy the strongest experimental support are psychoeducation, cognitive-behaviour therapy (CBT), training in social skills, cognitive remediation and assertive community treatment. The adaptation of psychological therapies to psychoses has gained momentum in the last 20 years, and the efficacy of this type of intervention has permitted its incorporation in the principal international guides to clinical practice for the treatment of schizophrenia (American Psychiatric Association, 2004; Canadian Psychiatric Association, 2005; Dixon et al., 2010; Grupo de trabajo de la Guía de Práctica Clínica sobre la Esquizofrenia y el Trastorno Psicótico Incipiente, 2009; National Institute for Health and Clinical Excellence, 2008; Royal Australian and New Zealand College of Psychiatrists, 2005).

As a basis for the use of psychological therapies in psychoses, it is important to take into account the continuous nature of psychopathology. The continuous model of psychopathology emphasizes the phenotypical similarities between normality, neuroses and psychoses, breaking with the belief that “neurosis=psychological therapy” and “psychosis=pharmacological treatment” (Turkington, Kingdon, & Chadwick, 2003). However, for some professionals it is true that to talk of psychological therapies in schizophrenia still equates to opening up Pandora’s box; that is, straying into highly dangerous territory, given the view that the symptoms are inaccessible to reason, that they might even worsen if questions are asked about them, and that these patients are too disturbed to benefit from a psychological intervention. Although in some circumstances this may be true, it has been argued that these problems can be overcome by adapting the cognitive-behavioural approaches that have been used in anxiety disorders.

The acceptance of the principle that there is no clear dividing line between psychosis and other clinical categories permits a focus on the similarities between disorders and in particular on the notion that the symptoms of one are presented in others, and that each disorder involves dysfunctions in thought, emotion and behaviour and chemical imbalances within the brain.

The positive symptoms of schizophrenia are an exaggeration of the normal functions, so that they differ from normal functioning in the extent of the conviction with which they are sustained and the degree of concern they generate, among other aspects; but they are phenomena that are sometimes also present in normal people, or at least, in people without a diagnosis of psychosis.

Garety and cols. (2001) refer to two routes in the development of psychotic symptoms: through cognitive and affective changes (the most common), and through affective alterations only.

Cognitive and perceptual changes, prodromal to psychosis, tend to be strange experiences that heighten activation levels and trigger the search for a causal explanation; it is here that biased processes of conscious
appraisal play a fundamental role. Garety and Freeman (1999) have highlighted the existence of bias in the cognitive style of obtaining information, characterized by jumping to conclusions, bias in external attributions and deficits in the understanding of social situations and the intentions of others. Such biased processes exacerbate negative emotional states (e.g., anxiety, depression, anger). These authors believe such processes to be more likely where there is a personal history of early adversity and deprivation (e.g., social marginalization, losses in childhood, severe childhood trauma), giving rise to permanent vulnerability, characterized by negative beliefs about oneself and the world, and which would facilitate external attributions and low self-esteem.

However, psychotic-like experiences do not translate into openly psychotic symptoms when the person is capable of rejecting the externality hypothesis (that is, when he/she does not make an external attribution of those experiences) and making a decision of protective self-correction (e.g., “I think God is talking to me, but my head is probably playing tricks on me”). The externality factor is thus a crucial one, so that psychosis is considered to be present when the individual appraises certain experiences as caused by external and personally significant factors – which is what permits their formal identification as hallucinations or delusions.

The second route to psychosis involves a situation whereby the stressful event, according to Garety and Freeman, produces only affective disorders, which in turn activate biased appraisal processes and maladaptive beliefs about oneself or others, which give rise to an external attribution (e.g., delusions) of the life event or of the altered affect. In such cases, delusions occur independently of hallucinations or other psychotic symptoms.

TREATMENT OF THE POSITIVE SYMPTOMS OF PSYCHOSIS

In line with what we saw above, CBT formulates the clinical goal of acting directly on psychological processes, being effective when it succeeds in the following: the person reappraises externally attributed experiences as internally caused; the person changes negative beliefs about him/herself; and the person manages to redress the biased reasoning processes. CBT is considered complementary to dopaminergic blocking, which reduces the salience of environmental stimuli (Howes & Kapur, 2009).

CBT was first applied to psychoses in the 1980s by Tarrier and cols. (Tarrier et al., 1993), with the aim of helping patients to confront their symptoms. At the same time, Chadwick and Lowe (1990) showed that it was possible to subject delusional beliefs to “reality testing.” Up to now, cognitive models on psychotic experiences have been developed which suggest that, as already mentioned, the distress and incapacity they produce are generated not directly by the experiences themselves but by the way the person interprets them. That is, the distress and the behaviours related to the psychotic symptoms are not always caused by the presence of psychotic experiences per se, but are rather the result of the person’s appraisal of them (e.g., their potential threat). On the basis of this perspective, various manuals have been published that describe the application of these models in detail (Bentall, 2005; Birchwood & Tarrier, 1992; Chadwick, Birchwood, & Trower, 1996, 1997; Fowler, Garety, & Kuipers, 1995; Freeman, Bentall, & Garety, 2008; French & Morrison, 2004; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Haddock & Slade, 1996; Kingdon & Turkington, 1994; Morrison, 2001; Nelson, 2005; Perona Garcélán, Cuevas Yust, Vallina Fernández, & Lemos Giráldez, 2003; Perris & McGorry, 2004; Wright, Turkington, Kingdon, & Ramirez Basco, 2009).

A basic CBT model explores relationships among: 1) environmental events (e.g., memories of events, delusional perceptions, hallucinations), 2) cognitions (the specific meaning of such events or perceptions), 3) emotional responses, and 4) behaviours, as a guide for treatment. The core of CBT is normalization, understanding and the verification of beliefs or perceptions.

CBT in psychoses has developed in the direction of exploring the content and meaning of the symptoms in greater depth.

Intervention procedures with CBT in psychoses should include the following steps:

1. Optimizing the therapeutic relationship (achieving commitment from the patient and carrying out the initial assessment). In psychoses, the development of a collaborative relationship takes more time than in the case of other mental disorders; thus, it is advisable to proceed step by step, using non-threatening, supportive interviews, and showing genuine interest in the patient’s problems. Potential obstacles to collaboration should be explored, and there should be an assessment of the dimensions of delusions, hallucinations and negative symptoms (degree of
Treating comorbid substance abuse or misuse

1. Assessment of barriers to adherence (forgetting, negative attitudes or lack of support from family, side effects, financial limitations, etc.).

2. Normalizing and destigmatizing. The goals are to examine the cognitive errors and distortions that exacerbate feelings of shame and guilt; explore automatic thoughts about the diagnosis; make healthy attributions with regard to the disorder; reduce self-criticism to a minimum; adopt a problem-solving attitude; accept the vulnerability-stress model; and get the patient to see the therapist as an ally in fighting the symptoms.

3. Modifying core beliefs. Direct confrontation of delusions is counter-productive. The CBT approach consists in gently suggesting to the person that they might question their belief, inviting them to see how thoughts can be linked to triggering events), and a therapeutic plan drawn up.

4. Implementing behavioural strategies. Use of gradual exposure techniques, learning of breathing-relaxation, drawing-up of a list of early symptoms of exacerbation-relapse, sleeping habits, control of impulsivity, list of activities (especially in cases of severe depression).

5. Modifying automatic thoughts. By means of a record of the patient’s thoughts, the therapist should examine the data on which they are based, and make reattributions. Another method consists in focusing on the most common cognitive distortions: ignoring data, personalization, magnification and minimization, over-generalization, and thoughts in terms of all or nothing. In sum, an explanation should be formulated of the development and maintenance of the symptoms, in each case (attempting to discover how thoughts can be linked to triggering events), and a therapeutic plan drawn up.

6. Addressing problems with concentration or thought disorders. Setting of goals or an agenda; identification of factors that increase anxiety or disorganize thought.

7. Improving treatment adherence. Assessment of barriers to adherence (forgetting, negative attitudes or lack of support from family, side effects, financial limitations, etc.).

8. Treating comorbid substance abuse or misuse.


In particular, treatments for the delusional symptoms of chronic schizophrenia are based on the following principles: 1) the assumption that these patients are prone to the phenomenon of psychological reactance, whereby when they are directly confronted about a belief they maintain it more firmly, or it can even become more extreme, so that the approach to take is to explicitly avoid any attempt to coerce the patient; 2) patients should not be explicitly asked to abandon their own beliefs and adopt those of the therapist; 3) patients should be made to see that they are only being asked to consider the facts and arguments put forward, and to think about possible alternative opinions; 4) the territory in which the Socratic dialogue takes place is not that of the belief itself, but that of the data on which it is based; and 5) the therapist should encourage patients themselves to try and argue against their own beliefs.

Thus, starting by directly confronting the beliefs of some patients only succeeds in strengthening their delusions, instead of weakening them; therefore, the objective should be to focus on the phenomenology of the belief, rather than on trying to change it. The therapist should try to get the patient to become involved in seeking answers to some questions, together with the therapist: does the patient take into account information inconsistent with his or her beliefs? If any change occurs in the belief, what explains such changes?

As Chadwick and Lowe (1994) point out, rather than telling patients that they are wrong, the therapist must try to make them see the delusional idea as only one possible interpretation of the facts or events, getting them to consider and appraise alternative points of view. To this end, the therapy can include four elements: 1) the therapist must devote a considerable proportion of the time to Socratic dialogue and to making clear to patients the strength of the influence of their beliefs on their behaviour and interpretation of events; 2) discussion should focus on questioning the internal consistency or plausibility of the system of beliefs, highlighting all the irrational characteristics and inconsistencies; 3) in line with the view expressed by Maher (1988), on the formation of the delusion as a response to or attempt to give meaning to a specific experience or important life events, alternative perspectives should be offered; and 4) the patient’s delusion and the therapist’s alternative explanation should be appraised in the light of the information available.
In essence, with CBT it is attempted to demonstrate that beliefs are not always maintained tenaciously, and that, when the person undertakes an analysis together with the therapist, cognitions can be subjected to empirical testing, in the same way as occurs with neuroses.

Thus, empirical testing alone produces mild and inconsistent changes, whilst initial verbal confrontation, followed by empirical testing, substantially improves the effects of the therapy.

As regards hallucinations, there is evidence that they are sometimes present in normal persons (even with greater frequency than it is commonly believed) (De Loore et al., 2008; Hanssen, Bak, Bijl, Vollebergh, & Van Os, 2005; Hanssen, Bijl, Vollebergh, & van Os, 2003; Johns & van Os, 2001; Nelson & Yung, 2009; Verdoux & van Os, 2002); however, what defines psychosis is not only the existence of such phenomena, their implausible nature or a personal conviction of their reality. What can truly mark the transition to psychosis is the absence of cultural context for these experiences and the degree of preoccupation or distress they generate. In this sense, it does not make sense for psychological intervention to eliminate these experiences, but rather to reinterpret their significance and their origin (in particular, their internal rather than external origin, as generally judged by a person with psychosis). To this end, operant procedures have been used to treat hallucinations, such as stop-thinking, distractor or verbal suppression procedures like listening to music or mental counting, self-observation, aversion therapy, wearing headphones, and so on. As far as the way such interventions work, three groups can be identified: techniques that promote distraction from the voices; techniques that promote focusing on the voices; and techniques aimed at reducing anxiety.

Distraction techniques (e.g., wearing a walkman or similar, reading, singing), while useful for some patients, have only transitory effects, on failing to address the core problem of the cognitive disorder, which is the mistaken attribution of self-generated events to an external source.

Focusing techniques, in which the essential requirement is that hallucinators identify the voices as coming from themselves, should have more lasting effects. To achieve such effects, it is attempted to reduce the frequency of the voices and the associated distress through the gradual reattribution of the voices to oneself. The phases of the intervention include: 1) making the patient pay attention to the form and physical characteristics of the voices, such as their number, intensity or volume, tone, accent, apparent sex and location in space, and discussing these in the therapy session; 2) once the patient feels comfortable attending to the physical characteristics of the voices, he or she is asked to pay attention to their content (e.g., noting it down or discussing it in the therapy session) – content which obviously reflects his or her preoccupations and anxieties; likewise, the patient is asked to listen to and report the voices heard between sessions, as a homework assignment; 3) subsequently, the patient is asked to pay attention to his or her beliefs about the voices. To this end, patients are encouraged to note the events that precede the voices, the voices themselves, and the thoughts and feelings that follow them, both during the sessions and as homework. This leads to the formulation of a meaning and function of the voices that is shared by therapist and patient, and which serves as a basis for intervention in later sessions. This formulation normally implies an acceptance that the voices are self-generated in a context that involves acknowledging oneself as affected by a mental disorder.

**COGNITIVE-BEHAVIOUR THERAPY FOR DRUG-RESISTANT PSYCHOTIC SYMPTOMS**

In the UK, three research groups developed different methods of CBT for chronic psychotic patients (Perona Garcelán, Cuevas Yust, Vallina Fernández, & Lemos Giráldez, 2003; Vallina Fernández, Lemos Giráldez, & Fernández Iglesias, 2010): (a) Coping strategy enhancement (Tarrier et al., 1993), in Manchester, which reflects the influence of Meichenbaum, and self-instructions training, for strengthening coping skills; (b) Comprehensive cognitive-behaviour therapy (Garety, Kuipers, Fowler, Chamberlain, & Dunn, 1994), by the London group; and (c) Cognitive-behaviour therapy with a normalizing rationale (Kingdon & Turkington, 1994), also in Manchester, and with an emphasis on the notion of the continuity of psychopathology, and the adaptation of Beck’s therapy to psychosis.

Coping strategy enhancement includes training and information on cognitive-behavioural techniques, to improve the control of symptoms: analysis of antecedents, of the cognitive-behavioural response and of the consequences of situations; Comprehensive cognitive-behaviour therapy, first practiced in the early 1990s, included coping strategy enhancement, modification of beliefs, normalization and psychoeducation. It is a therapy strongly influenced by CBT for depression, considering that up to 40% of
patients present affective symptoms, in addition to the positive symptoms; both types of symptom are the object of treatment. Finally, in cognitive-behaviour therapy with a normalizing rationale there is an emphasis on the essential importance of the formulation and management of negative beliefs about the illness. The therapeutic process involves using reasoning based on normalization and de-stigmatization, for explaining the appearance of the symptoms, and this is achieved by highlighting the importance of stress in normal people and, under very serious circumstances, also in schizophrenia.

By emphasizing the similarities between normal errors in reasoning and those of schizophrenia, patients are encouraged to see the link between their stressful life events and the origin of the psychosis. This should be combined with psychoeducation and the model of vulnerability, within a perspective of the continuity of psychopathology.

It is interesting, finally, to consider the reflections of Birchwood and Trower (2006), who stress that the future of CBT for psychosis lies in gaining an understanding of the (cognitive) interface between emotion and psychosis, and in developing interventions either solely to resolve the emotional/behavioural dysfunction or to prevent or mitigate psychoses and their positive symptoms.

In fact, the emotions are clearly involved in the ontogenesis of psychosis. In studies on the factors that intervene in the transition to psychosis, in high-risk populations, or on symptomatic relapses, it is found that depression, anxiety and, in particular, social anxiety are among the most powerful predictors (Owens, Miller, Lawrie, & Johnstone, 2005).

In sum, CBT accompanies neuroleptic drugs with a distinct and complementary objective; it is not merely a substitute for them.

**PSYCHOEDUCATION**

Psychoeducational interventions, whose goal is to provide information to the patient and his or her immediate family about the disorder and its treatment, at the same time as providing them with some strategies for coping with the disorder, has also shown its usefulness. Various meta-analyses have demonstrated the efficacy of these interventions in the reduction of expressed emotion, of relapses and of re-hospitalization rates (Girón et al., 2010; Pilling et al., 2002; Pitschel-Walz, Leucht, Bäuml, Kissling, & Engel, 2001). The efficacy of family-based interventions in schizophrenia (which include psychoeducation or psychosocial intervention) has been confirmed in various studies, in comparison with standard treatment, based on medication (Dixon & Lehman, 1995; Smith & Birchwood, 1990; Tarrier, 1996; Tarrier et al., 1988). In contrast, family interventions using a dynamic or counselling approach have not been seen to produce significant improvement, compared to standard treatment (Birchwood & Spencer, 2003; McFarlane, 1994; Vaughn et al., 1992).

A range of factors influence the way a family responds to an illness, including: a) the social support system available; b) the family’s previous experience with the illness and knowledge about it; c) coping patterns and resources; d) quality of the available health services and access to them; e) economic status; f) type of illness onset (sudden vs. gradual, public vs. private, etc.); g) nature of the symptoms; h) the demands made by carers; i) the patient’s agreement or refusal to participate in the therapeutic programme; and j) illness course and prognosis. Nevertheless, the type and intensity of the psychoeducational programme should be adapted to the phase of the illness and the capacity of both the family and the patient to interpret what is happening.

**PREVENTIVE INTERVENTIONS WITH INDIVIDUALS AT HIGH RISK OF PSYCHOSIS**

The prospective studies carried out in recent decades have helped to improve our knowledge about the period just before the onset of psychosis, at the same time as facilitating the generation of clinical markers for risk of transition to psychosis, which have been able to be replicated internationally, and which have made possible the first experimental studies on the primary prevention of psychosis. The main objective of a series of studies was to analyze the predictive power of the three clinical profiles or high-risk categories, designed by the Melbourne group (McGorry, 1998; Yung et al., 1998). Another type of study has been concerned with determining whether the application of different therapeutic formats in individuals with high clinical risk of developing psychosis succeeds in delaying or preventing the emergence of the disorder. Up to now, experimental studies have been conducted with various methodologies (randomized, double-blind, prospective), and which have compared the efficacy of diverse intervention modalities: pharmacological treatments (neuroleptics versus placebo, neuroleptics versus antidepressants); combined treatments (neuroleptics and cognitive
therapy versus antidepressants and support therapy; psychosocial treatments and neuroleptics versus psychosocial treatments and antidepressants); psychological treatments (cognitive therapy versus monthly supervision; integrated early intervention treatments versus standard treatments) (Broome et al., 2005; Cornblatt, Lencz, Smith, & Ather, 2004; Lemos-Girádez et al., 2009; McGlashan et al., 2007; McGlashan et al., 2003; McGorry et al., 2002; Morrison et al., 2007; Morrisson et al., 2004; Nordentoft et al., 2006; Phillips et al., 2007). In all of them it was observed: a) that the treatments, be they pharmacological or psychological, are superior to placebo or simple supervision, as regards success in delaying the transition to psychosis; b) that combined interventions (psychological and pharmacological) are those which obtain the best results; and c) that services oriented to early detection and intervention obtain better results than traditional services. It was observed, moreover, that these results are particularly important during the first year of active intervention, and that if the treatment is interrupted in the follow-ups at three years, the inter-group differences disappear.

NEW PROPOSALS FOR INTERVENTION IN PSYCHOSIS

Recovery from psychosis has begun to be understood in recent years as somewhat more than the mere reduction of symptoms, emphasis being placed on patients' social functioning and on their ability to undertake gratifying activities, achieve goals and live a life with meaning, which is worthwhile. With this in mind, the importance has been underlined of instilling hope in patients and of encouraging realistic optimism and a positive self-concept, especially in those for whom it is not easy to find a stable job – in other words, integrating concepts derived from positive psychology, focused more on mental well-being than on mental disorder, and thus contributing to reducing the social stigma.

On the basis that people with psychosis generally develop states of desperation, becoming progressively intense, it is important to consider in the treatment the establishment of a feeling of self-control and of the capacity to organize one’s own life.

The development of positive beliefs about oneself and about others is a form of clinical intervention of a cognitive nature, in contrast to the traditional CBT approach, aimed at reducing the positive psychotic symptoms, and hence more focused on the negative and self-defeating cognitions.

With the aim of achieving this optimum recovery from the first psychotic episodes, the last ten years or so have seen the development of a series of new psychological interventions: Cognitively oriented psychotherapy for first-episode psychosis (COPE) (Henry, Edwards, Jackson, Hulbert, & McGorry, 2002); Cognitive Interpersonal Therapy for Recovery and Relapse Prevention (Gumley & Schwannauer, 2006); Cognitive Recovery Intervention (CRI) (Jackson et al., 2009); Social Recovery Cognitive Behaviour Therapy (SRCBT) (Fowler et al., 2009); The Graduated Recovery Intervention Program for first episode psychosis (GRIP) (Waldheter et al., 2008); and Cognitive-Behavioural Relapse Prevention Therapy (RPT) (Gleeson et al., 2009).

These new intervention modalities are aimed at achieving symptomatic recovery from a first episode of psychosis, with an emotional recovery that includes the development of personal skills of acceptance, comprehension, self-reflection and introspective awareness; that helps the patient overcome the stigmatizing and traumatic aspects which tend to be present in 35-50% of cases, related mainly to the symptoms experienced during the psychotic episode (referential delusions, persecutory delusions, auditory hallucinations, etc.), and to painful experiences associated with the type of intervention and care provided during the crisis (involuntary hospitalization, restraint, isolation, etc.) (Bendall, Jackson, Hulbert, & McGorry 2008); and that puts the person in a position to recover his or her various personal and social roles in the shortest time possible and with the highest expectations of being able to continue his or her social and professional development. Table 1 shows the initial, encouraging results obtained with these different types of intervention in psychological and functional recovery from a first psychotic episode.

SPECIFIC TREATMENTS TAILORED TO EACH PHASE

Currently, and in contrast to what is customary, characterized by the use of common therapeutic procedures in all phases of the disorder, it is recommended that interventions in psychoses, and particularly in schizophrenia, be tailored to each phase of the course of the disorder, according to the following principles (Tandon, Nasrallah, & Keshavan, 2010): 1) bearing in mind that the disorder begins in adolescence or early adulthood, and that various aetiological factors
interact or have an additive effect in the process of the disorder’s development, prevention and intervention should be tailored to the stage of the disorder that the person has reached (and the identification of risk factors and clinical markers is fundamental to the adjustment of the treatment); 2) therapeutic interventions will be more effective the earlier they are applied in the critical periods of clinical course (Birchwood, 2000; Birchwood, Todd, & Jackson, 1998); and 3) therapeutic interventions should take into account the important role of neuroplasticity in the pathogenesis of schizophrenia, and the need to restore the altered plasticity through neurobiological and psychological therapies (Fisher, Holland, Merzenich, & Vivogradov, 2009).

In the initial phases of psychosis, the aim of the therapy should be the early identification of the disorder and the rapid application of an effective treatment, with a view to halting its progression and limiting the impairment of social functioning. In the later phases of the disorder, on the other hand, the therapeutic goals should be the reduction of the symptoms and the possible reversion of brain pathology. And in all cases, the principal objective must be the maximum possible restoration of the affected functions (McGorry, 2000).

**CONCLUSIONS**

As yet there is no effective preventive treatment for psychoses aimed at the general population (*universal* primary prevention). Research on risk profiles for psychosis, and particularly schizotypal personality traits, is a fruitful and promising field (Fonseca, Muñiz, Lemos, Paino, & Villazón, 2010; Fonseca Pedrero et al., 2007; Raine, 1991, 2006), but it is still not possible to predict with confidence which individuals will make the transition to psychosis (*selective* primary prevention). In particular, there is a limited capacity to identify levels of psychotic-like experiences and to predict which persons with attenuated positive symptoms will develop a full-blown psychosis, since such experiences do not constitute a unitary phenomenon: there are various types, probably with different courses and underlying causes. Moreover, progress is still required in the identification of the phenotypical factors that predict the onset of a psychosis, as well as psychotic-like experiences.

In the last ten years, however, there have been substantial advances in approaches to psychoses, and especially in the area of early intervention, based on the identification of certain prodromal clinical

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<td>COPE: Better adaptation to the illness, quality of life, negative symptoms and insight at end of treatment. At 1-year follow-up Better adaptation to the illness and at 4 years, no differences with respect to TAU.</td>
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<td>Jackson &amp; McGorry, 2009</td>
<td>66</td>
<td>CBT(CRI)+ TAU vs. TAU</td>
<td>6 months</td>
<td>CRI: More general improvement and lower levels of post-psychotic trauma, no differences in depression and self-esteem.</td>
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<tr>
<td>EIS, Birmingham, UK</td>
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<td>Fowler et al., 2009</td>
<td>77</td>
<td>SRCBT+TAU vs. TAU</td>
<td>9 months</td>
<td>SRCBT: Significant improvements in structured activities, economic activities and the PANSS. Trend toward improvement in desperation, in functioning of instrumental roles and in needs on the Camberwell Assessment of Needs (CAN) for the non-affective psychoses group.</td>
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<td>EIS, Norfolk, CAMERO, UK</td>
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<td>Waldheter et al., 2008</td>
<td>10</td>
<td>GRIP+ TAU vs. Piloto pre-post</td>
<td>9 months</td>
<td>Improvement in positive and negative symptoms, social functioning and achievement of personal goals. Treatment retention rate of 67%.</td>
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<td>OASIS Clinic and STEP programme, London, UK</td>
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<td>Gleeson et al., 2009</td>
<td>81</td>
<td>RPT+TAU vs. TAU</td>
<td>7 months</td>
<td>RPT: Significantly lower relapse rate, longer period before relapsing.</td>
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<td>EPPIC, Melbourne, Australia</td>
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characteristics (indicated primary prevention) (McGorry et al., 2009), but problems persist with the precise definition of risk, giving rise to the possibility of unnecessary intervention (false positives). The recent proposal to include a new category, Psychosis Risk Syndrome, in the next edition of the DSM-V (American Psychiatric Association, 2010) has stirred up some heated debate over the risks and benefits that the consideration of such a syndrome might bring (Carpenter, 2009; Johannessen & McGorry, 2010; Kaymaz & van Os, 2010; Kingdon, Hansen, & Turkington, 2010; McGorry, in press; Morrison, Byrne, & Bentall, 2010; Ross, 2010; Yung, Nelson, Thompson, & Wood, in press-a, in press-b).

Psychosis Risk Syndrome indicates different courses leading to specific syndromes. Therefore, in the near future, it will be necessary to improve its predictive value, based for now solely on signs and symptoms, with biomarkers, neuropsychological markers and endophenotypes; it will be necessary to develop safe and effective interventions (psychological and neurological protection strategies) and to set up specific services for the identification and treatment of “high-risk states”. With this in mind, broad-spectrum interventions (stress management, CBT, case management) should be applied, so as to allay concerns over the potential treatment of false positives.

Where the psychotic symptomatology is already present, however, the aim of intervention should be to reduce as far as possible the time the patient remains without treatment, so as to avoid aggravating the symptoms and to limit the patient’s functional impairment.

The goal of treatment should be to go beyond the positive psychotic symptoms and also address other clinical dimensions, such as cognitive deficits and negative symptoms. The final purpose must be to improve the patient’s quality of life and restore the affected functions. Current research on effective therapies recommends combining pharmacotherapy with the psychological treatments that have produced the best results. Nevertheless, while current clinical practice tends to use the same therapy in all cases, the treatments of the future should be tailored to the specific phase of the disorder that the patient is in at the time.

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