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An Interacting Cognitive Subsystems Model of Relapse and the Course of Psychosis

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This paper outlines a theoretical analysis of current psychological conceptualizations of psychosis that draws on Teasdale and Barnard's (1993) Interacting Cognitive Subsystems (ICS) model of depression. The ICS model differs from Beck *et al.*'s (1979) model of emotional disorders in that it specifies two qualitatively different levels of meaning. These different levels of meaning play a different role in the production, maintenance and modification of emotion, and indeed, also recognize the distinction between 'intellectual' belief and 'emotional' belief. Intellectual beliefs are at the level of specific propositional meaning: they have a truth value which can be assessed and they are not directly influenced by sensory information such as voice tone, arousal and body state, or visual stimuli. Given this, propositional meaning does not have a direct link to the production of emotion. Rather the link to emotion is an indirect one, through the activation of more generic and holistic affect-related schematic models. These latter schematic models are hypothesized to have implicational meaning, and correspond to schematic models derived from emotive experiences. In consequence they do not have a specific truth-value and they reflect contributions from a wide variety of sources, including multiple patterns of specific meanings, and patterns of direct sensory input. This ICS perspective proposes that integration of these two levels of meaning can facilitate the psychological conceptualization of the initiation, acceleration, and maintenance of positive psychotic symptomatology during relapse. This theoretical analysis is illustrated by the use of a case study, which describes the process and application of ICS to facilitating recovery from acute psychosis. This paper also provides a detailed discussion of the implications of this analysis for future research and psychological therapy. In particular, ICS has relevance for tracing the evolution of schematic beliefs during the early episodes of psychosis, which are hypothesized to have a bearing on the future course of psychosis. This has implications for the further development and expansion of psychological treatments for relapse and recovery from psychosis. Copyright © 1999 John Wiley & Sons, Ltd.

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INTRODUCTION

For many years it was believed that psychotic symptoms were discontinuous from 'normal' psychological functioning. This position is becoming increasingly untenable in the light of research evidence that positive psychotic symptoms can be understood with reference to normal psychological processes. This paper will outline the evidence from current psychological conceptualizations that psychotic symptoms are closely related to a person's psychological functioning and that they are therefore amenable to psychological intervention (Yusupoff *et al*., 1996). Current psychological conceptualizations of hallucinations and delusions hold that the content of these symptoms contain material which is personally relevant to the individual. Indeed, this personal relevance is critical to the understanding of, and cognitive interventions with psychotic symptoms.

In this paper we will propose that an individual's attempts to assimilate and accommodate the changes associated with psychosis are central to the development and maintenance of symptomatology, associated disability and distress. It will be argued that the process of relapse encapsulates this interaction between an individual and their experience of psychosis. Indeed, given that relapse sees the emergence of positive symptomatology, there is a need to develop an integrative model of psychotic relapse that can accommodate existing psychological conceptualizations of hallucinations and delusions. These formulations have been predominantly linear conceptualizations of symptomatology. This paper will outline a conceptualization of psychotic relapse that draws upon Teasdale and Barnard's Interacting Subsystems Model (ICS; Teasdale and Barnard, 1993). It will be argued that this is an improvement on the linear models, which are integral to most other current cognitive theories of psychosis.

CURRENT PSYCHOLOGICAL CONCEPTUALIZATIONS OF PSYCHOSIS

Hallucinations

Attribution theory (Weiner, 1985) has been helpful in developing psychological models of hallucinations and delusions. Attributional models of hallucinations conceptualize an auditory hallucination as a mental event that is misattributed to an external source. This hypothesis has been supported by various studies which have shown that individuals with hallucinations demonstrate a bias toward assuming a voice had been presented when it had not (Bentall, 1996). Indeed, Slade and Bentall (1988) demonstrated that individuals with hallucinations are more inclined to judge a perceived event as real and make rapid and over-confident judgements about their perceptions. Bentall's (1990) cognitive model of hallucinations incorporates the concept of 'metacognition': the process whereby individuals are able to reflect upon their own experience and mental processes. Bentall argues that in normal reality discrimination, individuals determine the source (internal or external) of a perceived event by non-consciously applying a set of criteria, such as the properties of the event, contextual cues, and expectations. Factors that may influence application of these criteria include environmental stimulation and style of information processing. Therefore, individuals will hallucinate when they wrongly infer that internally generated cognitive events such as thoughts are externally generated stimuli. This misattribution may be influenced by environmental factors (e.g. overstimulation) leading to high arousal and shallow information processing of stimuli. Furthermore, the misattribution of an aversive cognitive event to an external 'non-self' source may be negatively reinforcing through arousal reduction. This process may be influenced by the individual's own schemata about self, world and future. Such schemata will influence individuals' expectations, interpretation and emotional response to both internal and external events (Padesky and Greenberger, 1995).

Morrison *et al*. (1995) have elaborated a cognitive model of auditory hallucinations which proposes that auditory hallucinations arise from an attributional bias. Morrison *et al*.'s model draws on existing cognitive conceptualizations of intrusive thoughts (Rachman, 1978). Intrusions are defined as repetitive thoughts, images or impulses that are unacceptable or unwanted and are usually accompanied by subjective discomfort. Salkovskis (1985) hypothesizes that such thoughts are also ego-dystonic; i.e. they are incompatible with the individuals's own belief system. Salkovskis and Kirk (1997) hypothesize that the occurrence and content of intrusive thoughts in obsessive compulsive disorder, are interpreted by individuals as meaning that they might be responsible for harm to themselves or others unless they take action to prevent it. It is the appraisal of responsibility, therefore, which is central to the development of distress. This results in attempts to suppress or neutralize the intrusive thought. Morrison *et al*. (1995) note that there is a

similarity in the content of intrusions and auditory hallucinations. Thus it is argued that the occurrence of an auditory hallucination is accounted for by the result of an attributional process whereby individuals misattribute ego-dystonic, unwanted, and uncontrollable thoughts to an external source thereby leading to reduced distress. Cognitive dissonance theory (Festinger, 1957; Beauvois and Joule, 1996) states that dissonance results when two cognitions contradict each other. Therefore when positive thoughts are inconsistent with the individual's own beliefs about self, dissonance will result. An attribution to an external source (resulting in an auditory hallucination) will reduce dissonance. This would account for the experience of pleasant auditory hallucinations in individuals with negative self-schemata. Morrison *et al*. also hypothesize that their theory may also be useful in explaining thought insertion, thought withdrawal, and thought broadcasting, which may be related to misattributions in response to uncontrollable and/or unwanted cognitive events.

Experimental support for this hypothesis is described by Morrison and Haddock (1997) who examined the cognitive processes underlying auditory hallucinations in an experiment which investigated delayed and immediate source monitoring for positive, negative and neutral verbal material. They found that individuals experiencing auditory hallucinations had an external attributional bias for their immediate thoughts, but not for their memories of those thoughts. This bias was not demonstrated in individuals with no auditory hallucinations but with other psychotic symptoms. This implies a bias in moment by moment source monitoring specific to auditory hallucinations. Morrison and Haddock also found that the emotional valence of verbal material was a significant factor, which influenced the bias in source monitoring. This is consistent with Bentall's (1990) and Morrison *et al*.'s (1995) theories which predict that emotional content of intrusions has a direct effect on their misattribution to an external source.

Chadwick and Birchwood (1994) have studied individual's beliefs about voices in relation to emotional and behavioural responses to voices. Using an ABC framework they formulated voices as activating events (A) and found that there was cognitive content-specificity in that voices believed (B) to be malevolent were associated with negative affect and were always resisted (C), whereas voices believed to be benevolent were associated with positive affect and were engaged with. Conceptualizations of auditory hallucinations by Bentall and Morrison have centred on explaining psychotic symptom occurrence in contrast to Chadwick and Birchwood's work which has focused upon the emotional and behavioural consequences of symptom occurrence.

The fact that these different models of auditory hallucinations have focused upon different aspects of the phenomenology of individuals' beliefs suggests that there may be a need for a multi-level conceptualization of symptoms which encompasses a number of psychological factors responsible for the initiation, acceleration and maintenance of auditory hallucinations. A key initiating factor may be the interaction between intrusive and unwanted cognitions, which conflict with core schemata producing dissonant or discrepant internal experiences. The process of misattribution leading to temporary reductions in such discrepancies plays a role in the acceleration of symptomatology through negative reinforcement. As individuals' idiosyncratic beliefs about symptoms crystallize and emerge, these beliefs play a role in the maintenance of symptomatology and the development of secondary morbidity and distress. For example, in the context of voices, which are ascribed power and malevolence, the experience of powerlessness and helplessness will follow.

Delusions

Attributional models have also been utilized to explain the development of delusions. Maher (1988) hypothesizes that delusions are formed by the same cognitive processes as normal theories and beliefs, and evolve as an explanation of puzzling anomalous experiences. Like Morrison *et al*.'s (1995) formulation of hallucinations, Maher's theory incorporates the concept of cognitive dissonance. When a discrepancy occurs between an expected sequence of events and an observed sequence of events, this leads to a sense of puzzlement and perplexity. The adoption of a delusional explanation which accounts for personally significant and/or anomalous experiences is accompanied by a reduction in tension and uncertainty (Maher, 1988). However this model can be criticized on the grounds that abnormal cognitive processes have been demonstrated in individuals with delusions (Garety, 1991). Garety found evidence of a judgmental bias in some deluded individuals where these individuals are excessively influenced by current information, and make less use of past learned regularities in making inferences. Garety also found an apparently contradictory bias in other individuals with paranoid

delusions, which was a tendency to rely excessively on prior expectations when processing new information. These contradictory biases are accommodated in her model of delusional formation (Garety and Hemsley, 1994). She hypothesizes that these two judgmental styles may reflect two stages of response to an information-processing abnormality. In this model the initial process of delusional formation arises from the excessive reliance on current perception. The resulting delusional belief will then generate strong expectations, which influence affective state and attention to 'belief'-congruent information.

Attributional processes associated with delusional beliefs have also been the focus of recent experimental investigation. Individuals with persecutory delusions seem to be more likely to attribute blame for negative events to external causes and positive events to internal causes (Kaney and Bentall, 1989, 1992; Fear *et al*., 1996). They also preferentially attend to (Bentall and Kaney, 1989) and recall (Bentall *et al*., 1995) threatening information. Kinderman (1994) found that individuals with persecutory delusions have a specific attentional bias for information of relevance to self-concept. It has been suggested that these experimental findings are consistent with the proposition that deluded individuals have negative self-schemata, which are experienced by the individual as a discrepancy between their actual and ideal selves. The model conceptualizes persecutory delusions as compensatory beliefs, which arise in response to this perceived discrepancy. This concept of delusional beliefs serving such a protective function has also been suggested for grandiose delusions.

Trower and Chadwick (1995) concur with the suggestions of Bentall and other investigators that persecutory delusions have a defensive function in that they are associated with low self-esteem which is outside the individual's awareness. They have expanded this theory to encompass an interpersonal focus. They suggest that sources of threat are a solely interpersonal negative evaluation and that the defence, too, has this interpersonal focus in that it is an interpersonal evaluation of the other person which characterizes the paranoid 'defence'. They also suggest that paranoia can be divided into two types—persecution paranoia (the type researched by Bentall and others) and punishment paranoia. These two types of paranoia are held to be characterized by differing attributional styles which relate to how information is processed with reference to self. Punishment paranoia is hypothesized to be characterized by an attributional style where the individual attributes negative events to self and positive events to external causes. With punishment paranoia the individual believes that they are bad and blameworthy and that others are justifiably punishing them. The defensive function of punishment paranoia is less clear than in persecutory paranoia.

Most of the work on cognitive theory and therapy has emphasized the importance of conceptualizing delusions with regard to core self-schemata. A number of psychological factors may play a role in the initiation, acceleration and maintenance of delusional beliefs. The initiation of delusions arises from the evolution of explanations for anomalous events, which are experienced as puzzling, confusing and discrepant with expectations. Central to the process of acceleration of these explanations is the appraisal of threat and the reinforcement contingencies inherent in the adoption of delusional explanations. Individuals' core beliefs form a central reference point for the appraisal of threat. Gilbert (1997) argues that attacks on, and/or losses of social attractiveness are associated with shame and humiliation. Gilbert's model proposes that both shame and humiliation are associated with sensitivity to criticism, a desire to protect oneself, increased arousal, and complex affects. However shame and humiliation differ on a number of dimensions including attributional style, view of self, attentional focus, emotion, and behaviour. We argue that the experience of shame or humiliation associated with attacks on or loss of social attractiveness is ontogenic in the development and acceleration of delusions. Humiliation, like persecutory paranoia, is characterized by an external attributional bias, where others are seen as bad and are blamed for negative events. In both humiliation and persecutory paranoia there is a strong sense of injustice and the individual is more likely to react to negative events with anger and hostility. On the other hand, both shame and punishment paranoia are characterized by an internal attributional style, where self is seen as bad and blameworthy of negative events. Shame and punishment paranoia are both associated with heightened self-consciousness, and no obvious sense of injustice. Individuals are more likely to react with depression, submissiveness and withdrawal. Both shame and humiliation are associated with differing evolutionary strategies aimed at the maintenance of private social attractiveness (humiliation) or public social attractiveness (shame). The maintenance of private and public social attractiveness may be implicit in the defensive functions of persecutory and punishment paranoia. This is not

currently addressed within current psychological conceptualizations of psychosis.

Relapse

Despite the advances in pharmacological management of schizophrenia, relapse remains a major factor in the development of illness chronicity and social disability. Indeed for the individual themselves, relapse is critical in the development of secondary depression. Birchwood *et al.* (1993) found that perception of control over illness was the most powerful predictor of depression in schizophrenia. Recognition of the social, emotional and psychological costs of relapse has led investigators to examine approaches to the detection and prevention of relapse.

Retrospective studies of individuals and their families (Herz and Melville, 1980; McCandless-Glincher *et al.*, 1986; Birchwood *et al.*, 1989) show that both groups are able to recognize reduced well-being. The most commonly reported early signs are sleeplessness, irritability, tension, depression, and social withdrawal. The consistency with which these early signs have been reported has led to the development of prospective investigations of early signs. In essence, these investigations have sought to identify the sensitivity and specificity of these early signs as an indicator of emerging relapse. Clearly if these early signs are sensitive and specific to relapse, the monitoring of such signs would enable early intervention leading to the prevention and/or amelioration of relapse. Malla and Norman (1995) in their systematic review of the early signs literature found very few studies that directly assessed the relationship between putative prodromal symptoms or early signs, and the exacerbation of relapse (Birchwood *et al.*, 1989; Jolley *et al.*, 1990; Marder *et al.*, 1991; Tarrier *et al.*, 1991; Gaebel *et al.*, 1993; Marder *et al.*, 1994; Jorgensen, 1998). Table 1 below provides a summary of the studies that have examined the sensitivity and/or specificity of putative prodromes.

The results of some of these studies have been disappointing. However, Birchwood (1995) points out that individual variations in the nature and timing of early signs will act to reduce their apparent amplitude in group studies. Group studies fail to capture the qualitative and quantitative differences between individuals in their early signs. Therefore it may be more appropriate to think of early signs as an individualized configuration of symptoms which Birchwood refers to as a 'relapse signature'.

Birchwood (1995) accounts for this variation in the nature and timing of early signs by integrating individual's own idiosyncratic response to emerging relapse. This cognitive explanation for the variation in early signs suggests that dysphoric symptoms such as anxiety, tension, withdrawal, depressed mood, suspiciousness, and sleeplessness arise from the way in which individuals explain and interpret internal and external events. Birchwood (1995) offers a compelling cognitive analysis of early relapse which draws upon Maher's (1988) model of delusional formation and Weiner's (1985, 1986) attribution theory. Birchwood proposes that the attributions made by individuals to account for and explain the emergence of disturbing symptoms can serve to either accelerate or retard the process of relapse. In this model, dysphoria is seen as a response to the fear of impending relapse (perhaps for those with previous experience of relapse) or a failure to explain symptoms and experiences (perhaps for those with less experience of relapse).

This model might therefore predict that those individuals with extensive prior experience of relapse and its associated negative repercussions would respond with high levels of fear and perhaps helplessness leading to depression and withdrawal. On the other hand, those with less experience may respond with puzzlement, confusion and perplexity. The model may also help explain the speed at which relapse proceeds by specifying the cognitive mechanisms and associated emotional consequences responsible for acceleration.

This model highlights the role of individual's attributions for and meaning ascribed to symptoms and internal experiences. One might hypothesize that where such attributions have an impact upon the acceleration of emotion, they will relate to personally relevant beliefs and assumptions held by

Table 1.

Author(s)	Relapses	Sensitivity %	Specificity %
Subotnik and Neuchterlein (1988)	17	59	NR
Birchwood *et al.* (1989)	8	63	82
Hirsch and Jolley (1989)	10	73	NR
Tarrier *et al.* (1991)	16	50	81
Gaebel *et al.* (1993)	162	8	90
		14	70
		10	93
Marder *et al.* (1994)	42	37	NR
		48	
Malla and Norman (1994)	24	50	90
Jorgensen (1998)	27	81	79

an individual. These beliefs might concern the individual themselves (I am worthless, I am a failure), the world (other people will criticize me, other people will harm me) or their illness (my illness is god's retribution on me, I will never control this). A model of relapse, which integrates such beliefs, may be more powerful in explaining the initiation and acceleration of relapse.

For example, the experience of visuo-perceptual changes or increased arousal associated with emerging psychosis may be interpreted by an individual as a sign of impending relapse. If this individual believes that they have no control over their illness, their response to these changes is likely to be increased vigilance, anxiety, worry, sleeplessness and perhaps withdrawal. Therefore this individual's beliefs about self and others and their meta-beliefs about illness and symptoms may serve to accelerate the speed at which relapse proceeds. The activation of individuals' beliefs about themselves, others, and/or their illness serves to generate a range of cognitive, emotional, behavioural and physiological changes which represent an individualized signature or configuration of symptoms that characterize the early relapse phase. Cognitive formulation and intervention may therefore provide an additional means of identifying, preventing and/or ameliorating relapse.

Within this analysis we propose that a number of psychological factors play a role in the initiation and acceleration of relapse. Initiation of relapse may be through the activation of core schemata about self, world, and future. Idiosyncratic beliefs about illness, which have evolved during, and following the first episode will have close relationships with these schemata. Activation of schemata and beliefs may be through external stressors (e.g. increasing tension and hostility at home) or internal cues (idiosyncratic symptoms which have psychological significance in that they resemble or foretell the emergence of illness). From the point of initiation, the activation of schematic models of self (including illness), world and future drive the emergence of a range of thoughts, attributions and inferences which accelerate the development of an acute episode.

INTERACTING COGNITIVE SUBSYSTEMS MODEL

Implicit in our analysis of the preceding literature of current psychological conceptualizations of psychosis is the construct of initiation, acceleration and maintenance. Within this construct we have proposed a number of psychological factors which could play a critical role in the evolution of psychosis. Teasdale and Barnard's (1993) Interacting Cognitive Subsystems model provides an existing theoretical framework, which may hold our emerging conceptualization.

The concept of persecutory and punishment paranoia poses similar issues to those associated with the understanding of auditory hallucinations which have a positive content as opposed to those which have a negative content. Critical to the attributional process are individuals' underlying beliefs about themselves, and the attribution of responsibility for negative and positive (internal or external) events. Single level approaches, which attempt to explain the role of personal meaning in the development of symptoms, are insufficient. Indeed, single level approaches have come under increased criticism within the literature (Power and Champion, 1986; Brewin, 1989; Greenberg and Pascual-Leone, 1997; Power and Dalgleish, 1997). Also, from a clinical viewpoint, Teasdale (1997a) argues that within cognitive therapy, the use of 'rational' argument is frequently ineffective in changing the emotional response, even when the individual is able to acknowledge the intellectual power of an argument. An analysis of attributional style in the development of psychotic symptoms provides an illustration of the limitations of single level approaches. Purely addressing attributions implicit in the development and maintenance of psychotic symptoms made by individuals with psychosis will have limited therapeutic value without recourse to underlying beliefs about self and others. As such, attributions can be thought of as providing a marker for personal meanings.

This is central to Teasdale and Barnard's (1993) Interacting Cognitive Subsystems (ICS) model of depression. They propose that negative thoughts and images may be useful markers for the state of 'parent' schematic models. In consequence, cognitive-behavioural or other therapeutic procedures, which address negative thoughts and images, will not necessarily lead to emotional change unless such thoughts and images relate to the parent schemata. Teasdale and Barnard (1993) and more recently Teasdale (1997a) provide an account of the relationship between meaning and emotion. The ICS model differs from Beck *et al.*'s (1979) model of emotional disorders in that it specifies two qualitatively different levels of meaning. These different levels of meaning play a different role in the production, maintenance and modification of emotion, and

indeed, also recognize the distinction between 'intellectual' belief and 'emotional' belief. Intellectual beliefs are at the level of specific propositional meaning: they have a truth value which can be assessed and they are not directly influenced by sensory information such as voice tone, arousal and body state, or visual stimuli. Given this Teasdale argues that propositional meaning does not have a direct link to the production of emotion. Rather the link to emotion is an indirect one, through the activation of more generic and holistic affect-related schematic models. These latter schematic models are hypothesized to have implicational meaning, and correspond to schematic models derived from emotive experiences. In consequence they do not have a specific truth-value and they reflect contributions from a wide variety of sources, including multiple patterns of specific meanings, and patterns of direct sensory input. In this analysis the sensory inputs from body state have an important role in the production of affect-related schematic models. The heart of the ICS model provides an eloquent explanation of the development and maintenance of depression.

Affect-related schematic models are derived from multiple sources including patterns of specific meanings arising from current environmental events and information processing, and patterns of internal and external sensory input. In depression, the sensory source will be internal, for example depressed posture, facial expression, retardation, anhedonia etc. The configuration of these multiple elements leads to the generation of idiosyncratic models of depression, where self is viewed as failure and the depressed state as aversive, uncontrollable and persistent. In this respect, depression might be triggered by any number of the elements in the configuration and is maintained by what Teasdale and Barnard (1993) refer to as the 'depressive interlock'. This interlock represents the interaction between depressive configurations and the individual's higher order view of depression. For example relapsing depression would be accounted for within an ICS model by initial (minor) depressive reactions associated environmental events which evoke schematic models of depression where self is viewed as powerless and depression as uncontrollable resulting in a self-perpetuating and accelerating depressive interlock.

AN ICS ANALYSIS OF RELAPSE AND COURSE OF PSYCHOSIS

We propose that ICS theory confers two distinct advantages in understanding psychosis. First, the use of its multilevel theory enables the integration and elaboration of existing psychological conceptualizations of psychotic symptoms. This will be demonstrated through introducing four classes of overlap between these models and ICS. Second, the ICS model contains a number of consequences for the development of further empirical work and the refinement of current therapeutic practice. Again, this will be demonstrated through examining the particular predictions implied by ICS, as opposed to other multilevel models, and through a case illustration.

To summarize, the ICS model, outlined by Barnard (1985) proposes that no single system controls the processing of information. Rather, it is the interaction of a number of subsystems, which process and encode information from multiple sensory sources (visual, acoustic and body-state). Information processing depends on information flowing from one subsystem to another, and that regular recurring patterns of information enables basic recognition, comprehension, processing of stimuli (see Barnard, 1985; Teasdale and Barnard, 1993, chapter 5). This is critical within the ICS theory. In ICS the processing configuration is motivated to reduce depression, by reducing discrepancies (dissonance) between current and highly desired states of affairs (for example, 'always seeking the approval of other people is the road to happiness'; Teasdale *et al.*, 1995). We propose that the initiation, acceleration, and maintenance of psychosis can be explained by individuals' attempts to make sense of, and reduce discrepancies arising from multiple and interacting sources of information. Indeed, this represents an area of overlap or convergence with current psychological conceptualizations of psychosis which have emphasized the role of cognitive dissonance and its reduction in the evolution of psychotic experiences such as auditory hallucinations and delusional beliefs.

Initiation of relapse

In a clinical setting the prediction of relapse is based upon the identification of a range of signs and symptoms which have been demonstrated in retrospective and prospective studies. These signs and symptoms are subject to considerable variance in their character and timing. This reduces their apparent amplitude in group studies, and increases the likelihood of false positive or false negative predictions of relapse in clinical practice. In ICS information processing relies on the recognition of recurring patterns of information which are derived

from multiple sources. These recurring patterns are embodied in the creation of propositional representations reflecting, on the one hand, intended and, on the other hand, current states of affairs. The creation of generic and holistic implicational models which reflect the discrepancy between intended and current states can then effectively monitor progress towards goals attainment. In this sense implicational meaning abstracts over sensory, body state, and semantic sources of information.

Within ICS the occurrence of a pattern or configuration of internal and/or external events which have a strong similarity with previous relapses will access implicational meaning more rapidly than if the configuration has a lower similarity. The activation of implicational meaning structures derived from previous experiences of psychosis will initiate the process of relapse. A primary source of information for a relapse configuration is internal information on body state and arousal (Tarrier and Turpin, 1992), and cognitive-perceptual change (Neuchterlein and Dawson, 1984; Frith, 1992). Indeed, feelings of lack of control over cognitive and perceptual processes during relapse have been reported by a number of authors (Bowers, 1968; Freedman and Chapman, 1973; Donlan and Blacker, 1975). There are two implications of this analysis.

First, definitions that are currently used to capture sensitivity of early signs could well benefit from definitions more closely allied to alternative multilevel views of how implicational meaning is constituted—rather than relying on a more closely delineated set of individual signs and symptoms alone. For example, for an individual a combination of signs and symptoms such as increased sleep disturbance, reduced attention and concentration and increased agitation will be experienced as a discrepancy from intended internal states and represented at an implicational level as a 'sense of losing control' or a 'sense of not feeling right'. This might then be accompanied by a sense of impending disaster or threat to oneself. As this sense is represented at an implicational level this will not have a specific truth-value, rather it emerges from a combination of sensory, body-state and semantic information. If the clinician pays sole attention to the occurrence of specific signs and symptoms, they risk failing to capture the more holistic and generic meaning experienced by the individual. Therefore, configurations of early signs which are more closely allied to the schematic meanings achieved during early relapse may increase sensitivity, and reduce the apparent variance in the nature and timing of experiences signalling future relapse.

Second, ICS proposes that information derived from different sources is managed by the evaluation of discrepancies between systems. In ICS, information processing is motivated to reduce these discrepancies. It is this process which we propose is responsible for the acceleration of relapse and the development of psychotic symptoms.

Acceleration of Relapse and the Development of Psychotic Symptoms

In order to examine how relapse is accelerated within an ICS perspective it is worth considering four classes of overlap between ICS and existing psychological conceptualizations of psychosis. First, psychological models of delusions and hallucinations imply that separate but related information-processing systems are involved in the development and maintenance of symptomatology. One system, such as attributional bias, manages specific moment by moment meanings. The second information-processing system relates to meta-cognition (Bentall, 1990), underlying schematic beliefs (Morrison *et al*., 1995), or self-concept (Kinderman, 1994; Trower and Chadwick, 1995). ICS specifies to subsystems that are responsible for the management of meaning; one manages specific moment by moment propositional meaning, the other higher order schematic or implicational meaning. It is the interaction between propositional meaning and implicational meaning subsystems that constitutes the 'central engine' of cognition.

Second, underlying schematic beliefs that are proposed within existing psychological conceptualizations are utilized to explain the emotional valence of an event to an individual. For example, in the case of auditory hallucinations. Bentall's (1990) and Morrison *et al*.'s (1995) theories both predict that the emotional content of intrusions has a direct effect on their misattribution to an external source. In ICS, depression is produced by the processing of depressogenic schematic models. Specific meanings, negative automatic thoughts, and images only contribute indirectly through their influence on higher order meanings. We propose that during relapse, acceleration of the central engine is through the processing of schematic models of self (e.g. 'I have no control', 'I'm a bad person'), world/others (e.g. 'people want to harm me', 'I'll be punished') and illness (e.g. 'my illness is a punishment', 'my illness is a weakness').

Third, beliefs about the power, purpose, malevolence/benevolence of symptoms are directly linked to the emotional and behavioural consequences of auditory hallucinations. In ICS, depression is maintained by the continuing regeneration of depressogenic schematic models where depression is viewed as aversive and uncontrollable. Therefore, beliefs about symptoms such as auditory hallucinations are critical to the maintenance of symptomatology through regeneration of implicational meaning structures. In Chadwick and Birchwood's (1994) study using cognitive therapy to modify beliefs about voices it was predicted that this approach would lead to a reduction in the levels of distress associated with voice-hearing but not a reduction in frequency. The reduction in frequency of auditory hallucinations following cognitive therapy, which was observed in this study, would be accounted for within an ICS perspective. If beliefs about voices are directly linked to the implicational subsystems responsible for the initiation and acceleration of symptoms, then modification of these beliefs will lead to a modification of underlying implicational meaning. ICS predicts that this would therefore lead to a reduction in the frequency of auditory hallucinations, as well as associated distress.

Fourth, current psychological models of psychosis, predict that the information processing implicit in the development of psychotic symptoms is motivated to reduce the dysphoria arising form dissonant or discrepant experiences, and to protect self in relation to self (e.g. persecutory paranoia) or self in relation to others (e.g. punishment paranoia). In ICS, the processing configuration is motivated to reduce depression by reducing discrepancies (dissonance) between current experience and intended experience. Critical to our analysis of the initiation, acceleration and maintenance of relapse is the role of the interlock between propositional and implicational meaning as the central engine of relapse. This engine is motivated to reduce discrepancies between intended and actual experience through the construction of implicational meaning structures. The activation of relevant implicational meaning serves to initiate the process of relapse, the interlock between propositional meaning and implicational meaning serves to accelerate relapse, and the regeneration of implicational models serves to maintain relapse. This is illustrated in Figure 1.

This model attempts to illustrate the process of initiation, acceleration and maintenance of relapse. Box 1 to 2 illustrates the relationship between early symptomatic changes in body-state and information processing and how these changes access implicational meaning structures. Within box 2, the interlock between implicational and propositional meaning which forms the central engine of relapse is illustrated, where the development of psychotic symptoms (box 3) serves to reduce some of the emotional and affective experiences produced by this interlock. The process of maintenance is hypothesized to be accounted for through the continuing regeneration of implicational meaning illustrated in the direct relationship between psychotic symptoms (box 3) and implicational meaning (box 2).

Persistent reactivation of implicational meaning will present as increased chronicity and resistance to treatment, whereas intermittent reactivation will present as a relapsing course with periods of remission. A range of factors may be responsible for this process of reactivation including a range of internal or external experiences, which have special and specific psychological significance for individuals. We believe that such a model may provide a useful means of identifying and preventing early treatment resistance by guiding clinicians in their conceptualization of the interaction between the individual and their beliefs, their experience of psychosis, and the subsequent development of secondary beliefs about self, illness, environment and future. We hypothesize that these elements have their basis in the early episodes of psychosis. Indeed, Jackson and Birchwood (1996) refer to the first few years of psychosis as the critical period. It is this period, which sees the emergence of secondary co-morbidity and treatment resistance. This period can be characterized by high levels of traumatization (McGorry *et al*., 1991) and negative consequences associated with reduced social attractiveness, status and rank.

PREDICTIONS AND APPLICATIONS

The key feature that distinguishes ICS from other multilevel models such as the Schematic Propositional Associative Analogue Representation Systems (SPAARS; Power and Dalgleish, 1997) approach, is the contribution of the interaction between propositional and implicational meaning in the process of change (be it relapse or recovery) and how self-related schematic models play a role in those processes. ICS offers a detailed view of what these representations are, how they relate to specific patterns of information flow, and how those

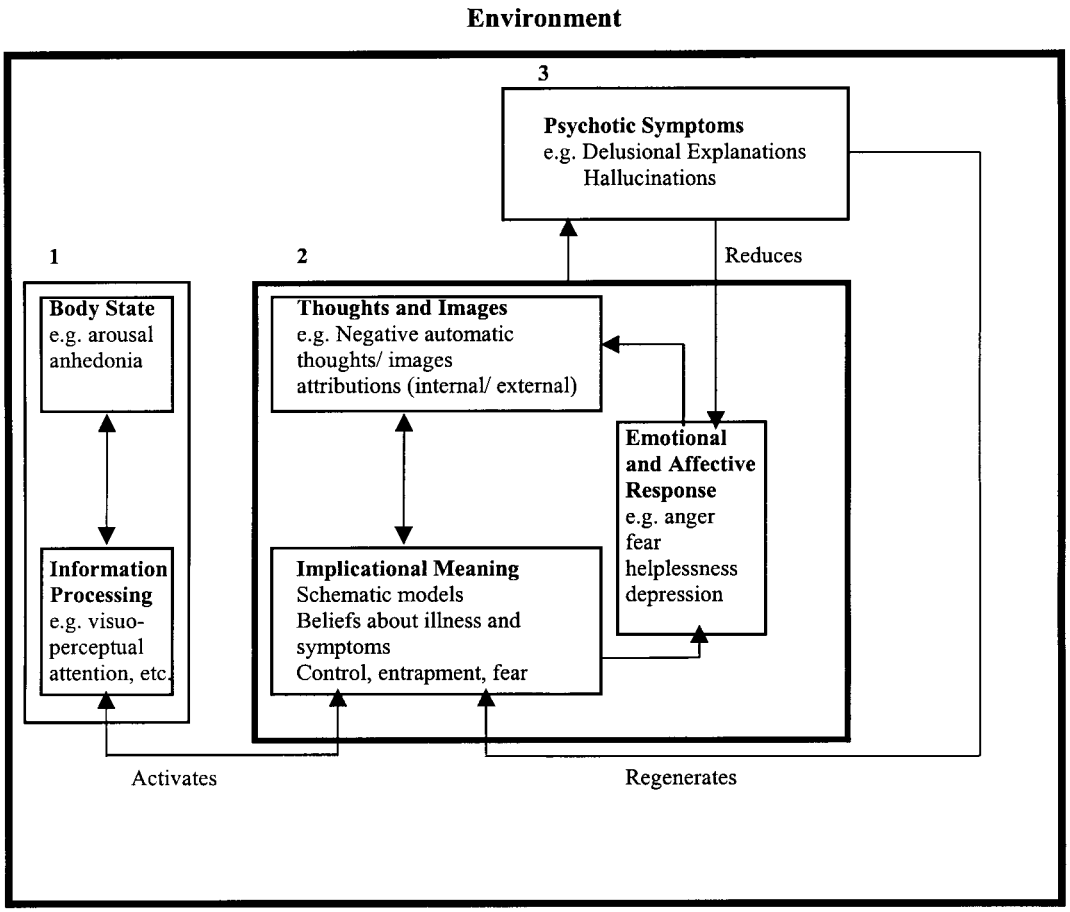

Figure 1.

exchanges between subsystems are managed by discrepancies among representations.

Specifically, SPAARS seeks to integrate the key components of ICS including implicational, propositional and other subsystems (visual, olfactory, gustatory, proprioceptive, tactile and auditory). These latter subsystems are considered within SPAARS as analogical representations. ICS provides a detailed analysis of how discrepancies between these systems are managed by the interaction of propositional and implicational meaning. This detailed view of how subsystems interact has important consequences for the formulation and prediction of the impact and involvement of psychological factors in the initiation, acceleration, maintenance and recovery from psychosis.

Early Intervention

Birchwood (1995) offers a cognitive conceptualization of relapse that specifies that attributions reflect the individual's idiosyncratic response to early relapse and that these attributions play a role in the acceleration of an acute psychotic episode. ICS provides a more detailed analysis by specifying how relapse is initiated and accelerated. We likened relapse to an engine that is initiated by discrepancies between subsystems, which activate implicational meaning. Acceleration of relapse then occurs through the interlock between propositional and implicational meaning. This interlock is geared towards the reduction of discrepancies between subsystems. For example, if the attribution 'I feel as if something bad will happen', is coupled with externally-derived sensory input (e.g. harsh tones or frowns from friends and family), externally-derived semantic contents (e.g. critical remarks) and bodily states (e.g. physical agitation and unease), the implicational meaning derived from these multiple sources of information might be 'Other people are trying to harm me'. Therefore during relapse, when a vulnerable individual is repeatedly exposed to

particular combinations of sensory inputs this serves to regenerate implicational and schematic models which will progressively come to dominate and control mental life.

This analysis predicts that the speed of relapse is governed by the extent to which implicational models are activated and regenerated. This process will be an idiosyncratic one which depends upon the particular schemata, assumptions and beliefs of individuals. In this sense the ICS analysis is consistent with Birchwood's (1995) view that early signs represent a continuous process as opposed to prodromal conceptualizations which see early signs as a discrete stage before acute psychosis.

In terms of its clinical application, ICS has a number of implications. First, early signs configurations which are more closely allied to concepts of implicational meaning may provide a means of reducing the apparent amplitude and variation in early signs, and increase the sensitivity and specificity of relapse prediction. Second, false positive increases in early signs imply incomplete activation of implicational meaning and therefore provide an opportunity for individuals and clinicians to rehearse coping scenarios for early relapse. Third, enabling individuals to increase their mindfulness of implicational meaning may facilitate delayed acceleration of relapse thus widening the window of early intervention. Fourth, ICS provides a detailed view of the initiation and acceleration of relapse, which may facilitate the development of cognitive therapy as an effective early intervention strategy.

Recovery from Psychosis

Within ICS the central engine of cognition is the interaction between propositional and implicational subsystems. This interaction is a motivated system which can be utilized to explain the maintenance of psychotic symptoms. In our analysis the development of psychotic symptoms such as delusions and hallucinations is a consequence of the central engine's attempts to reduce discrepant experiences between subsystems. In this sense delusions result from the interaction between externally-derived sensory inputs (e.g. voice tone, criticism, and visual images), propositional meanings (e.g. 'I believe ⟨X⟩ thinks this of me'), and internally-derived sensory inputs (e.g. body-state and information processing). Hallucinatory phenomena such as voices, thought insertion, withdrawal or broadcasting, result from the interaction between internally-generated events (e.g. thoughts and images), internally-derived sensory inputs and patterns of propositional meaning. Implicational meaning provides a schematically-derived and holistic level of meaning which abstracts over these subsystems. In depression, implicational meaning is motivated to reduce depression. We propose that in psychosis, implicational meaning offers a vehicle which serves to protect important evolutionary strategies associated with private and public social attractiveness. Private social attractiveness is maintained through the external attribution of discrepancies as in persecutory delusions or negative auditory hallucinations. Public social attractiveness is maintained through internal attribution of negative events (as in punishment delusions), or external attribution of positive internal events (as in pleasant auditory hallucinations). According to this analysis, psychotic symptoms could be thought of as providing a marker for the status of 'parent' schematic models that contain information about self- and other-related models (including illness). This has important implications for the role of psychological therapy in facilitating recovery from acute or chronic psychosis. The following case illustration aims to demonstrate the relevance of ICS in the course of treatment and recovery.

CASE ILLUSTRATION

Referral

Miss X is a 23-year-old female with a $2\frac{1}{2}$-year history of Schizoaffective Disorder (DSM-IV; American Psychiatric Association, 1994). During this period she has had two acute episodes of psychosis. Her first episode occurred during her final year studying chemistry at university. She experienced a probable 6-month duration of untreated illness before she was seen by her general practitioner and referred to a consultant psychiatrist. She was initially diagnosed as having manic depression. Her second episode occurred almost 6 months later and required a 6-week period of hospitalization. At discharge she was referred for assessment for cognitive therapy because of persisting depressed mood, delusions and ideas of reference. The decision to refer for cognitive therapy was based on the case management goals identified prior to discharge to facilitate recovery from acute psychosis.

Initial Presentation

Miss X's initial presentation was characterized by depressed mood. She reported symptoms of low

energy, poor motivation, excessive sleep, poor appetite, and anhedonia. She described spending much of her day in bed, and was frequently preoccupied with the supernatural. The main emotions reported were guilt, fear, shame and embarrassment. She reported feeling fearful in a range of social situations and avoided going outside, meeting friends, and playing golf and swimming. Miss X said that she had got 'involved' with the supernatural at university after she used a Ouija board. She described feeling ashamed and embarrassed of her behaviour and feared that some retribution would be carried out against her. During hospitalization, she had been admitted with punishment delusions. She believed that she was being talked about in the media and that the Queen, government and the church were operating a conspiracy against her. At initial presentation she continued to report fears that this retribution was continuing against her and indeed cited evidence from newspapers and television supporting her beliefs.

The First Episode

Miss X's first episode occurred during the final year at university. In the November, her mother and father noticed that she was becoming increasingly withdrawn. They attributed this to the long daily travel to university and so arranged for her to share a flat closer to the university. Miss X became increasingly isolated, and her work at university deteriorated. She recalls becoming increasingly worried about her exams as the year progressed and consulted a Ouija board in order to gain reassurance about her exams. Approximately 2 weeks later, she describes experiencing passivity phenomena, which she attributed to the act of an evil spirit, which she must have accessed by the Ouija board. At the time of experiencing passivity she was ripping up her university textbooks. In an effort to seek forgiveness from God, she visited her local church at night and carved crosses into the grass. Following a consultation with her general practitioner a referral was made to a consultant psychiatrist. Following successful pharmacological treatment, Miss X continued to have intrusive recollections of these events, particularly the memory of her body having been taken over. These memories were associated with increased fear and somatic manifestations of anxiety. She became increasingly worried about others finding out what had happened and became hypervigilant for any references to religion and the supernatural.

The Second Episode

Miss X became increasingly socially avoidant. She was encouraged to take part-time work in a local bar. During this period other employees bullied her. Miss X attributed this to her use of the Ouija board and making crosses in the ground. She became increasingly vigilant, and began selectively attending to any evidence of others knowing about her behaviour. She started noticing programmes on television and articles in newspapers, which referred to religion and the supernatural. She developed the belief that information about her was being passed to the media, and that the source of this information was the church and government. She believed that the purpose of publishing this information was to assist some retribution against her. She experienced a range of intrusive imagery representing this retribution. For example, she recalled an image of being boiled in a bath, after which her body was broken up and fed to crows.

She was seen by her consultant psychiatrist, who noted marked depressed mood characterized by delusional preoccupation, ideas of reference, and thought disorder. She was admitted informally to a local inpatient psychiatric unit for 6 weeks.

Background history

Miss X lives at home with her parents and younger sister. She described her family background as close and strongly Christian. She was encouraged to perform well at school and was given considerable approval on her academic performance. She recalls performing badly at school in English. This was a source of considerable tension within the family. Miss X recalled being told that she was doing well but not good enough. Her parent's response to difficulties at school was disappointment, which Miss X interpreted as failure to meet her parent's expectations. This gave rise to increased study and reduced contact with friends and peers. She recalls high levels of competitiveness with her sister, perceiving her sister as more talented than her. Some of these issues were observed during assessment of the family. There were strong pressures on Miss X to recover her social functioning. At times she perceived that she was being criticized by her parents for her lack of motivation and progress in recovery.

Formulation and Treatment

Figure 2 below provides a diagrammatical formulation of the initiation, acceleration and maintenance

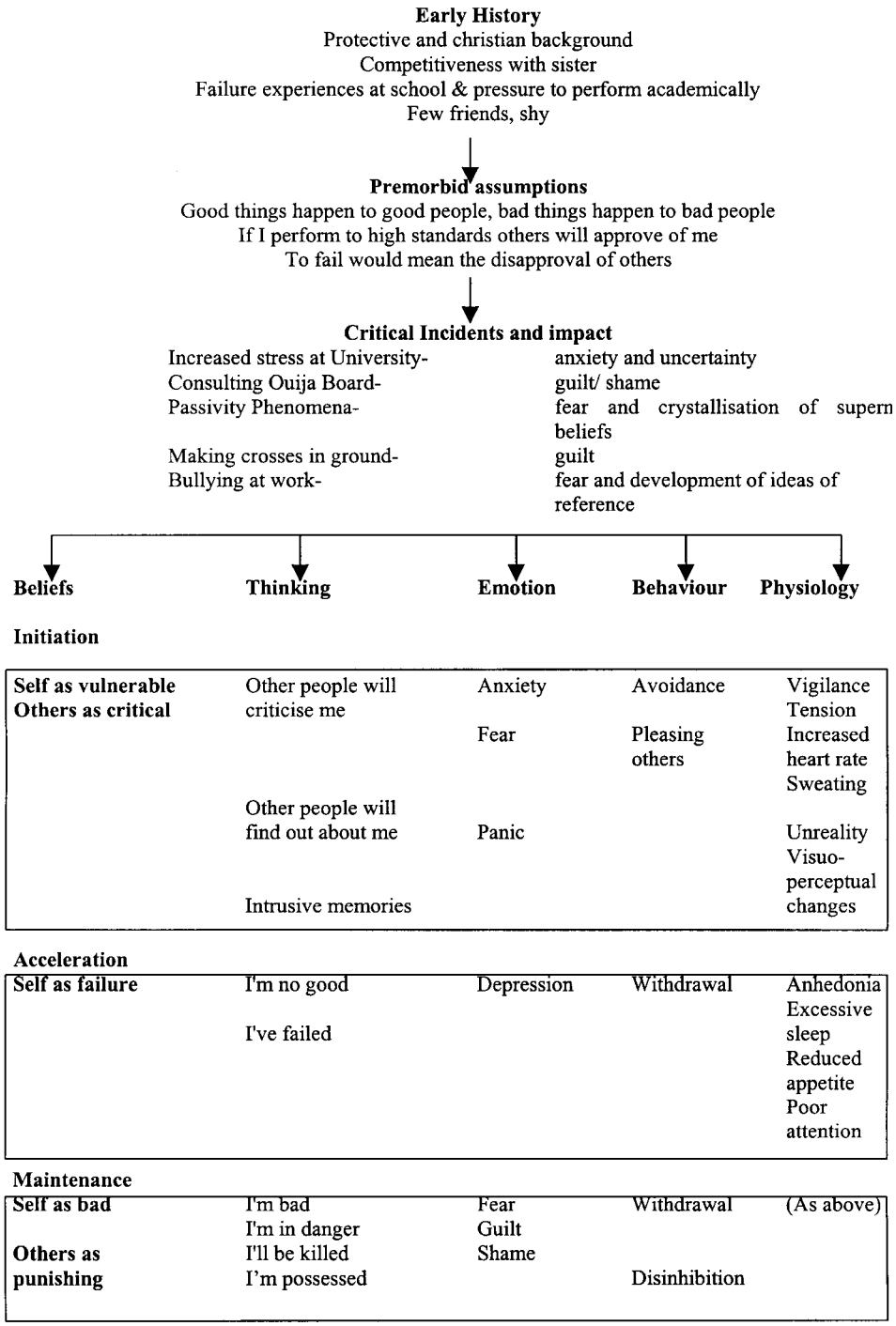

Figure 2.

of Miss X's psychosis. The patterns of implicational meaning which were hypothesized to abstract over these processes are detailed alongside the associated propositional and sensory-derived inputs (emotion, behaviour and physiology). For the purposes of facilitating recovery, intervention initially targeted maintenance processes before proceeding to accelerating and initiating factors.

Maintenance

ICS proposes that psychotic symptoms are maintained by the continuing regeneration of implicational meaning which abstracts over multiple patterns of internally- and externally-derived information. In addition, according to our model psychotic symptoms represent thoughts and beliefs which are markers for the state of these 'parent' schematic models. In the case of Miss X a number of information sources appeared to be relevant to the maintenance of her delusions and ideas of reference. Miss X reported a range of thoughts and images including intrusive memories of being bullied, using a Ouija board, and being possessed. These images were provoked by externally-derived sensory sources of information including religious or supernatural references contained in television programmes, newspapers, books and radio. Miss X's delusional beliefs reflected a pattern of propositional meanings represented by, 'I believe people are planning to harm me', 'I believe the church, media, and government are involved in these plans', and 'I believe that this is because I have offended society by committing crimes'. The implicational meaning of these beliefs (self as bad, others as punishing) was associated with experiencing fear, shame, guilt and depression. The combination of these multiple sources of internal and external information served to continually regenerate implicational meaning, and thus represents the interlock maintaining her symptomatology.

Initial treatment sought to reduce the conviction, preoccupation, and distress associated with Miss X's delusional beliefs. Strategies were selected which would directly or indirectly address implicational meaning structures. These strategies were the formulation of an alternative rationale, which accounted for the evidence, maintaining her beliefs, cognitive restructuring of intrusive memories and images, and modification of beliefs concerning television and other media.

Careful analysis of evidence cited by Miss X to account for her beliefs led to the identification of a series of critical incidents. These are illustrated in Figure 2 as consulting a Ouija board, passivity phenomena (ripping up textbooks), making crosses in the church grounds, and bullying at work. Her initial appraisal of these incidents was that she had sinned against God and the Church by using a Ouija board. In consequence she described experiencing shame and guilt. These emotions were transformed to fear after she ripped up her textbooks. She externally attributed this act to being possessed by an evil spirit, which she had accessed during her use of the Ouija board. Her subsequent carving of crosses in the church grounds compounded her increasing shame and fear associated with her actions. She later perceived the bullying at work as direct evidence of the intention of others to kill her as punishment for her sins. The therapist (A.G.) formulated these incidents as 'traumatic events', which challenged her pre-existing assumptions. These assumptions are illustrated in Figure 2, for example, 'Good things happen to good people, bad things happen to bad people'. The formulation proposed that Miss X's attempts to assimilate these events to her pre-existing beliefs lead her to assume that because these negative events occurred, she attributed blame to herself. Thus her pre-existing assumptions were preserved.

The formulation was also utilized to explain the occurrence of cognitive events including intrusive memories, personalization of media references, and the co-existing agitation, depressed mood, and avoidance/withdrawal. Subsequent cognitive restructuring of Miss X's intrusive memories enabled her to reattribute her explanations concerning her role in these events from evidence of her badness to evidence of her efforts to cope with the increasing fear and uncertainty generated by her expectations of the forthcoming university examinations. This enabled Miss X to consider alternative explanations for the occurrence of religious and supernatural references and articles in the media.

Acceleration

Criticism from family members was hypothesized to play an accelerating role in the development of psychosis. Miss X was exposed to a pattern of communication within the family, which implied that she was a failure. This implicational meaning was generic across her prior academic performance and indeed her rate of recovery and social functioning achievements. Perceived criticism from parents was addressed within family sessions. The development of expressed emotion was traced to her parents' attributions of her behaviour. They perceived her withdrawal as a sign that Miss X had not been motivated to 'get better'. Their evidence for this was her persistent rumination over material concerning the supernatural. Initially they were supportive of her recovery, but lack of progress in their eyes led to feelings of disappointment and anger. Miss X interpreted this as disapproval, which led to increased depression. Miss X believed that she was a failure to her family and that there was little that she could do. Family sessions focused

upon developing a shared information of symptoms and the family's response to Miss X's illness. The family and Miss X were taught strategies to manage these problems and difficulties.

Initiation

As Miss X progressed in therapy the formulation was used to develop a model of relapse which contained a configuration of symptoms, thoughts and behaviours. This configuration was given meaning by the formulation itself by specifying the beliefs responsible for the development of emotions such as shame, anxiety, depression and the paranoia itself. Early intervention strategies focused upon a number of factors. First the identification of early signs of relapse. Second the use of cognitive techniques to weigh evidence for and against the possibility of imminent relapse. This is particularly important in reducing the likelihood of false positive predictions of becoming unwell, and thereby reducing oversensitization to relapse. Third, the use of formulation to provide meaning to the appearance of early signs. Fourth, the delivery of cognitive and behavioural interventions aimed at decelerating relapse, reducing symptomatology and restoring functioning.

In terms of outcome at 24 months Miss X reports no paranoid or depressive symptomatology. Miss X has commenced mainstream full-time employment, and there have been no further relapses.

Discussion

The central element which distinguishes this approach from approaches using similar cognitive behavioural treatment strategies for psychotic symptoms is a more dynamic case conceptualization which serves to explain process variables outlined in the ICS model, as well as the content variables from other approaches (e.g. attributions) to formulating psychotic symptoms. The ICS approach to formulation enables the clinician to trace the development and evolution of implicational meaning structures through the encapsulation of salient pre-morbid cognitive structures and their action on the acceleration and maintenance of psychosis. In the case of Miss X, pre-morbid assumptions regarding acceptance by others and achievement, played a key role in the external misattribution of discrepant behaviours during the acute episode, e.g. ripping up textbooks was attributed to external spiritual forces. In addition, ICS shows how multiple patterns of sensory input have an impact on the maintenance and acceleration of implicational meaning. For example, during recovery the pattern of criticism expressed within the family regenerated the implicational meaning that Miss X was a failure. Therefore, therapeutic strategies, which targeted this pattern of expressed emotion within the family were hypothesized to be a more effective means of modifying implicational meaning, as opposed to use of cognitive techniques aimed at restructuring this belief within individual sessions.

CONCLUSIONS

The conceptualization presented in this paper aims to provide an integrated model of relapse and the course of psychosis which is both theoretically and clinically relevant. In order to achieve this aim, the conceptualization draws on Teasdale and Barnard's (1993) Interacting Cognitive Subsystems model of depression. The ICS approach enables a detailed view of how multiple sources of information interact to establish self-organizing, self-perpetuating, processing configurations that act to maintain persistent cognitive-affective states. The model predicts that implicational meaning is critically involved in the processes of initiation, acceleration and maintenance of relapse in psychosis.

The main predictions of the ICS model would be that it should be possible, for example, using single case methodology, to trace the evolution of implicational meaning regarding the illness process, and the gradual integration of self-schemata within the overall configuration. These testable predictions would seem to be the main ways in which theory could drive therapeutic endeavours. In clinical practice, patients could be enabled to understand the gradual evolutionary process that is developing. Indeed, this process would in itself influence implicational meaning, providing as it does a new meaning framework of the experience of prodromal signs and emerging psychosis.

Further research could classify illness trajectories into stages characterized by differences in the relative predominance of implicational meaning in initiation, acceleration or maintenance stages. Indeed, it should also be possible to test this model using cognitive psychology paradigms which would provide evidence on which meaning system is primary in the processing of information, and indeed, the relative transparency of implicational meaning. One might hypothesize, that this would be different according to illness history, and degree

of critical overlap in schematic models of self, others, illness and world.

In addition, this model provides a basis for the development of psychological early interventions for relapse. This is an area, which has remained underdeveloped. In this respect we are currently undertaking a randomized controlled trial of a cognitively orientated intervention for relapse. In this trial we are currently recruiting individuals who are considered to be relapse prone. Relapse proneness is defined by one or more of the following factors; those with a history of recent relapse, non-compliance with medication, socially isolated, and living in a high stress environment. This is a client group that has traditionally challenged services and we hope that such an approach as described in this paper will offer hope in the management and amelioration of potentially negative illness trajectories.

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