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Neuropsychological deficit and psychodynamic defence models of schizophrenia
Towards an integrated psychotherapeutic model

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Introduction

In recent psychiatric literature on schizophrenia the contribution of psychoanalytic ideas tends to be discounted on the basis of two claims: the first is that psychoanalytic treatments of schizophrenia have been shown to be ineffective and the second repeats Karl Popper's original critique that psychodynamic explanations are a priori unscientific because they are unfalsifiable (Bentall, 2006; McGorry, 2004). As Glen Gabbard notes, such statements follow the recent fashion in psychiatry which assumes that the success of the neurosciences renders psychodynamic contributions both redundant and outdated (Gabbard, 1994; Gabbard et al., 2002). While it is disappointing to read statements which ignore important theoretical debates, such summary dismissal also indicates a missed opportunity to consider the experience of many therapists and patients who have found some benefit in a psychoanalytic approach (Cullberg and Johannessen, 2004; Gottdiener and Haslam, 2003).

Psychoanalytic theory and practice is not an inert museum piece. At its best, psychoanalytic theory responds to developments in other sciences, changes in clinical presentations and the shifting social demands inherent in providing public mental health services. Psychoanalytic theories of affect and motivation, for example, are being integrated with developmental and social psychology, drawing on direct observation of young children and experimental studies (Westen, 1998). So too, some psychoanalytic theorists are proposing models of psychopathology which consider the roles of genetic factors in their interplay with developmental and psychosocial factors (Fonagy, 2001b; Plomin and Rutter, 1998; Rutter, 2000, 2005). The broader implications of such findings are that mental health sciences in general have renounced the dream of achieving scientific purity by being based exclusively on genetics and the neurosciences and have begun to develop complex and multi-causal models which synthesize the biological and psychosocial causal factors of psychopathology. Such models therefore require the synthesis of research findings that investigate phenomena at different levels of analysis.
and using divergent methods. A growing number of psychoanalytically ori-
ented researchers realize that these more integrated and sophisticated models
offer opportunities for the confirmation, modification and falsification of
psychodynamic propositions in a way that has not previously been possible
(Emde and Fonagy, 1997; Fonagy, 2001b; Gabbard et al., 2002).

Nevertheless, discoveries in biological psychiatry are not easily translated
into clinical applications – be they pharmacological or psychotherapeutic
(Fenton, 2000). The clinical application of these findings requires a focus on
the individual patient, the uniqueness of the therapeutic relationship and the
level of psychological meaning which any illness state produces, whereas the
epistemological framework of biological sciences seeks generalized aeti-
ological mechanism of psychopathology. Integration of these disciplines can
begin only from an understanding that different epistemologies and thus
methodologies apply to these different levels of analysis – biological, cognitive-
psychological and behavioural. A complete research programme for the men-
tal health sciences integrates these idiographic and nomothetic approaches.
As Eric Kandel has noted, the traditional psychoanalytic methodology,
based on the study of case histories, has exhausted its usefulness. In his view,
psychoanalysis now needs to adopt a new intellectual framework integrating
the latest findings in cognitive and neurosciences (Kandel, 1999). The future
of the mental health sciences consists of developing the pharmacological and
psychotherapeutic applications of gene, brain, cognitive, affective and social
studies in their complementary role of treating what we now know to be
complex mental illnesses (Kandel, 1998).

This is particularly true when considering how psychoanalysis might
renew its understanding of schizophrenia while still retaining the key clinical
insights it has accumulated. This chapter will examine one aspect of the
neuropsychology of schizophrenia, namely deficits in theory of mind. First,
the work of Chris Frith is used to introduce the idea that aspects of schizo-
phrenia can be understood neuropsychologically as a deficit in theory of
mind (Frith, 1992). This can be contrasted with the psychodynamic theories
which are based on psychotic symptom formation resulting from specific
defences against conflicting motivations. The chapter will put the argument
that what psychodynamic theory typically describes as psychotic symptoms
in schizophrenic patients, such as delusion and hallucination, may be a defen-
sive distortion in the cognitive productions which are attempts to explain
or respond to deficits in theory of mind processing. While the neuro-
psychological deficit model is aimed at a level of analysis consistent with the
neurosciences, the defence model is an abstraction of the ensuing cognitive
and affective processes and lends itself more readily to psychotherapeutic
application. Therefore, the integration of both deficit and defence models
may provide a better basis upon which to conceptualize the psychotherapy
of schizophrenia by delimiting specific therapeutic strategies for deficits
and others for defences. The chapter will conclude by discussing what
aspects of the psychotherapy of schizophrenia might benefit from such a framework.

**Neuropsychological deficits of ‘theory of mind’ in schizophrenia**

Psychoanalysis, and indeed many of the current psychosocial interventions involved as a component of treatment for schizophrenia, would benefit from the integration of a theory of how neuropsychological deficits underpin the key features of psychotic symptoms. First, some definitional clarity is required with respect to the use of diagnostic terms. Psychoanalysts often loosely refer to psychosis and ‘psychotics’, and more specifically to psychotic defence mechanisms in cases that would not meet the criteria for a set of symptoms consistent with such a psychiatric diagnosis. In the present discussion the term ‘schizophrenia’ will be used to refer to a specific clinical condition which is a subset of the psychotic disorders whereas the term ‘psychosis’ will be used to describe symptoms that consist of impairment in reality testing. Psychotic symptoms form one characteristic of schizophrenia but are also associated with other disorders and can be artificially induced without implying the full syndrome of schizophrenia. Indeed, as a diagnostic entity, ‘schizophrenia’ may lack construct validity. Both clinical and genetic investigations suggest that the term may cover several different disorders with different aetiologies and mechanisms (Bentall et al., 1988a, 1988b; Elkin, 2004). Nancy Andreasen analysed data from her own symptom scales which suggested that the symptoms of schizophrenia tend to fall into three clusters: psychotic (delusion and hallucinations), negative (flattened affect, alogia, avolition, anhedonia) and disorganized (thought disorder, incongruent affect and bizarre behaviour) (Andreasen et al., 1995a; Andreasen et al., 1995b). Notwithstanding this important critique, this chapter will use the terms ‘schizophrenia’ and ‘psychotic symptom’ according to the more or less traditional psychiatric definition.

A growing body of research considers schizophrenia to be a disorder of social cognition – a term which can be defined as the capacity to mentally represent self and other as a guide to social interaction. Social cognition includes processes such as ‘theory of mind’ abilities, social perception, and attributional style. Impairment in social cognition may be an effect of information overload during the acute phase of schizophrenia (Drury et al., 1998) or may reflect a broader impairment in social functioning. Addington and colleagues found that deficits in social functioning are present throughout the course of schizophrenia and may persist despite antipsychotic treatment (Addington and Addington, 2000). Davidson and colleagues suggested that deficits in social functioning are present even before the onset of schizophrenia (Davidson et al., 1999). Some studies have suggested that this neurocognitive deficit in social functioning has a genetic origin. For example, one
study found the same social deficits in children of a biological parent who has schizophrenia suggesting that social deficits may be related to what these researchers call a broader schizophrenic genotype (Hans et al., 2000). Although in some cases social functioning tends to remain stable in severity of impairment, or may even worsen in subsequent phases of the illness, there is considerable evidence that this is not necessarily the case and the social functioning of some patients does improve over time (Harding et al., 1987).

Chris Frith (1992) proposed a more specific hypothesis that schizophrenia is essentially a dysfunction of meta-cognition or mentalization, that is, the ability to represent or think about one's own thoughts. Drawing on the original observations of Bleuler, Frith made a comparison of aspects of schizophrenia such as social withdrawal and dysfunction to similar social deficits in autism – both can be conceptualized as deficits in the development of a theory of mind. The term 'theory of mind' (ToM) was originally used in the context of research on primate cognition and introduced by Premack and Woodruff (1978). It refers to what has turned out to be a uniquely human ability to understand human actions as based on the underlying mental states of their agent (Povinelli and Vonk, 2003). The definition of the term also includes the attribution of mental states, such as beliefs or desires, to explain one's own and other people's current, past and predicted behaviour.

ToM provides a framework within which to understand how the self is linked to other mental representations. For example, Frith noted that normal cognitive functioning permits awareness of perceptions ('I know, 'it is raining') to be represented as beliefs attributed to others (John believes, 'it is raining') (Frith, 1994). Through the attribution of analogous mental states from the self to another, one might then infer the reason for certain behaviours (John is carrying an umbrella today because he believes it will rain). Beliefs about others' states of mind are formed in conjunction with a stable understanding of the self as having ownership of received perceptions, self-generated cognitions, and affects.

Impairment in ToM provides a useful theoretical model through which to understand the impairment of the ability to represent beliefs and intentions, particularly in the psychotic symptoms of schizophrenia. According to Frith (1992, 1994) anomalies can occur at three different levels of the perception of self and other. These consist of, first, disorders of willed action in which apathy or bizarre behaviours might result from the self's inability to perceive its own intentions or will, second, disorders of self-monitoring whereby there is a reduced awareness that thoughts and actions are generated by the self, and third, deficits in other monitoring which impairs the capacity to read others' thoughts, intentions, desires and behaviours. Bentall applied the argument to hallucinations saying that the ability to perceive if an event is real or imagined relies upon interpreting and integrating a number of sources of information in their relation to the agent who receives and processes the perceptions, that is, the self. Therefore, hallucinations may well result from a
failure to locate the perceiver as the recipient of the perception, i.e., a failure in meta-cognitive ability to locate the self in the representations it receives and processes (Bentall et al., 1991).

Such a theory can be applied more broadly to features of other common psychotic symptoms. The loss of the self as ‘owner’ of thoughts is apparent in thought insertion or thought broadcasting. The impaired capacity for the correct perception of another is apparent in delusions of alien control and persecution. So too delusions of persecution, jealousy or erotomania can be understood as a failure to monitor or accurately understand another’s intention or desire towards the self. Melancholic delusion appears to imply a misattribution of the value of the self by the self. This very brief overview at least shows that psychotic symptoms can be understood as a deficit in the capacity to relate thoughts, actions, or perceptions to subjective intentions, particularly in terms of understanding the self as the agent of mental events.

Of particular interest is research into the association of certain symptoms of schizophrenia with the ToM deficit. For example, Langdon and colleagues compared the pragmatic language skills and formal thought disorder of patients with schizophrenia to unaffected controls and found that poor appreciation of irony and poor mind-reading were associated with high ratings of thought disorder (Langdon et al., 2002). A further study found that people diagnosed with disorganized schizophrenia performed the most poorly on ToM tasks (Greig et al., 2004). This study suggested that ToM performance was also correlated with higher levels of thought disorder, cognitive disorganization and verbal memory. ToM impairment has also been studied in the relatives of patients with schizophrenia by comparing performance on a traditional false belief task and also a hinting task for a group of patients with schizophrenia, their first degree relatives and unaffected controls. Significant associations were found between schizophrenia risk, and failure on the hinting task which was designed to test a subject’s ability to infer a speaker’s real intentions when ‘dropping a hint’ in indirect speech. In this study first degree relatives scored intermediate values between patients and controls in the hinting task but the association between schizophrenia risk and failure on the false-belief tasks did not reach significance (Janssen et al., 2003).

Although, as previously discussed, this area of research was originally inspired by seeing a parallel between the social deficits in schizophrenia and those manifested in autism, two important distinctions have been noted which are likely to have important clinical implications. A study by Langdon et al. (2006) compared patients with schizophrenia to unaffected controls in their ability to discern the correct expression of affect corresponding to an emotionally laden cartoon strip. In addition to replicating findings suggesting impaired ToM abilities when measured by a subject’s capacity to attribute false beliefs, they also found general difficulties in patients with schizophrenia attributing the correct emotions to the depicted social exchanges. They therefore proposed that schizophrenia is a generalized deficit in empathic
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perspective taking which is responsible for the inability to understand not only others’ beliefs and perceptions but also emotions. In contrast, autism is a more specific deficit in social understanding, particularly beliefs and intentions, but the processing of the relation between simple events and basic emotions is not impaired (Langdon et al., 2006). The other major difference between autism and schizophrenia is developmental. As Frith (1992) originally pointed out, deficits in ToM in schizophrenia consist of a breakdown in more or less intact ToM, whereas in autism ToM is assumed to be impaired from birth. Therefore, it would appear that an understanding of the development of ToM is also relevant to understanding both its breakdown in schizophrenia and how one might integrate this developmental understanding into a mode of psychotherapy in order to repair ToM impairments, which will be discussed in the final section of the chapter.

There is already a substantial body of evidence to support the view that ToM is impaired in people with schizophrenia. The bulk of evidence is based on neuropsychological studies, but there have also been important neuroimaging studies. Frith and Frith (1999) have provided a discussion of the general neuroanatomy of ToM abilities. A subsequent neuroimaging study compared the performance of schizophrenic patients to controls when performing a socio-emotional task requiring mental state attribution and found lower activity in the left inferior frontal gyrus, a region of the brain believed to be involved in ToM function (Russell et al., 2000). While deficits in ToM capacities are clearly associated with schizophrenia, it is only one of several deficits which contribute to the susceptibility and course of schizophrenia. As several recent reviews have emphasized, greater precision in understanding the specific role of ToM deficits amongst other deficits in social cognition in schizophrenia awaits further research. In a review of some 30 studies on ToM in schizophrenia, for example, it was unclear how researchers ought to best measure ToM in schizophrenia and thus studies did not use standard measures (Harrington et al., 2005). Before drawing conclusions regarding the implications of this research for clinical practice, more data is required regarding the fluctuation of ToM over the course of schizophrenia, and how it affects patients’ use of language, and social behaviour (Brune, 2005).

**Psychoanalytic theories of the defensive function of psychotic symptoms**

Although this research is still in its early stages, there is broad agreement that ToM deficits provide a useful conceptualization of an underlying social deficit in schizophrenia. While broadly speaking the research supports Frith’s hypothesis, the theory does not provide a complete account for all aspects of psychotic symptom formation, most particularly delusional symptoms. Max Coltheart and Robyn Langdon extended the deficit theory and proposed that a single neurocognitive deficit is insufficient to explain the generation of some
psychotic symptoms such as multi-themed delusional beliefs (Langdon and Coltheart, 2000). They argued that an additional deficit in the capacity to evaluate belief formation is also required, what psychoanalysts refer to as ‘reality testing’. Both deficits need to operate in tandem in order to generate more complex forms of delusional belief. The first factor accounts for the perceptual aberrations which generate the content of a delusion while the second factor accounts for why a delusion is first adopted and then maintained despite the lack of evidence to support it (McKay et al., 2005).

Capgras delusion offers a good test of the deficit theory. In this delusion a person forms the belief that their spouse or lover is an impostor who looks identical to the real partner. According to a deficit theory such as Langdon and Coltheart’s model, such a delusion would result from a deficit in the capacity to link an affective response to facial recognition, i.e., the other person looks like my spouse but doesn’t feel like him or her. In addition, those who adopt and maintain such a belief lack an ability to reject unsubstantiated beliefs. However, McKay argued that Capgras delusion could also be understood as a belief formation that is influenced by an attempt to satisfy unacknowledged motivations (McKay et al., 2005). In this case the motivation would be based on ambivalent feelings towards the partner. McKay’s argument suggested that in addition to the neurocognitive deficits producing perceptual aberrations, defensive operations also distort information. Defensive operations process information in an unconscious manner in order to satisfy conflicting motivations. Impaired reality testing, the second factor, would enable beliefs formed on the basis of such erroneous information and the distortion derived from defensive modification of unpalatable motives to go unchallenged. Therefore, at least in some cases, the first factor in a two-factor model may be less the result of a neurocognitive deficit than a psychological defence. As McKay et al. (2005) noted, it may also be the case that the first factor arises out of a multitude of sources, both deficits and defences, because each interacts with the other.

The argument hinges on building a coherent theory of how internal motivations contribute to mechanisms that distort information concerning one’s self and one’s place in the world. As McKay et al. (2005) noted, psychoanalytic theories of motivation are only a subset of a broader set of theories of motivation. However, McKay does not elaborate more recent developments within psychoanalysis which recognize the theory of motivation as one of the key areas in need of theoretical renewal. Under the growing influence of attachment systems theory, many analysts have moved beyond Freud’s articulation of drive theory and any requirement to posit underlying infantile, libidinal strivings in all human motivations. Motivation is not explicable by the pleasure inherent in the reduction of drive tensions but better attributable to the need to maintain a sense of security and self-worth, the need to seek proximity to attachment figures and the need for adjustment to social and developmental demands (Bowlby, 1988; Fonagy, 2001a). Therefore,
motivations can be seen in a relational context, as meeting social demands and satisfying current needs, while drawing on what Bowlby (1988) called internal working models of social interaction based on past experiences in close relationships.

One of the hallmarks of psychoanalytic theory is its theory of defences which operate not only in psychopathology but are a component of everyday life. The notion of defence is integral to the psychoanalytic concept of the self. Very briefly, the self defends against incompatible ideas or affects in defence of its own integrity. This is threatened when certain ideas encroach which are incompatible or dangerous to a person's sense of himself or herself. Incompatible ideas may be motivated by desires which escape conscious awareness. While all defence involves a level of self-deception, not all defences require a distortion of reality.

Supreme Court Judge Daniel Paul Schreber's autobiographical account of his multi-thematic, paranoid and hypochondriac delusions provides the *locus classicus* of psychoanalytic theories of psychosis (Freud, 1958 [1911]). His psychotic break occurred after he had the idea that it would be pleasurable to be a woman submitting to intercourse. One of the key points that Freud emphasized in his discussion of the Schreber case is that the manifestations of psychotic symptoms are attempts to restore a gap which had opened in the relation of the self to the world. This gap has opened because an incompatible idea has been expelled in a radical form of defence. The precipitant to a psychotic break is therefore a vulnerability within the self which is later triggered by certain social demands and this initiates the process of psychotic symptom formation. Freud recognized very early in his work that the defensive operations which produce psychotic symptoms are of a quite different order of magnitude than those which repress ideas internally (i.e., into the unconscious) and therefore produce psychoneurotic symptoms such as conversion symptoms which have an unconscious meaning. Freud considered that psychotic defence was a radical form of defence based essentially on the mechanism of projection which attempts to modify the external world by projecting incompatible ideas into the external world, i.e., onto others. The defensive function of projection is to defend against self-generated ideas by denying that those ideas even belong to one's self. Projection therefore makes the world the bearer of all that is defended against, which may result in either withdrawal from a menacing world or greeting it with aggression. Therefore, implicit in Freud's account of psychotic symptom formation is a significant dysfunction and fragmentation of the self for the purpose of defence.

Leaving aside the argument over the accuracy of such a view, there are several key points to retain from Freud's initial account of the process of psychotic symptom formation. The impairment in social functioning means that therapeutic relations with patients with psychotic symptoms are of a different order to other patients. The therapeutic relation preferred by Freud assumes a relatively normal capacity for social cognition and intact reality
testing whereas psychotic symptoms arise precisely in the context of an impairment in such cognitions. In short, we arrive at a reason why Freud considered that psychoanalysis has no clinical application when the patient does not manifest a transference neurosis. One can readily agree with those critics who suggest that Freud has little to directly offer for the psychological treatment of schizophrenia, indeed Freud says this himself. However, it is clear that the development of ideas of psychotic defence and symptom formation can be used to create a substantially different theory and clinical technique to be applied to psychotic symptoms. This is a position that has permeated most schools of psychoanalysis for many decades and yet is never considered in negative appraisals of the outcomes of so-called ‘psycho-dynamic therapy’. The second point is that Freud’s notion of defensive projection, like Frith’s theory, considers the production of psychotic symptoms to result from a breakdown in the boundaries which contain thoughts, emotions and motivations as belonging to oneself. In this respect, although it may be stretching the point, one could also say that Freud’s defence model is also based on a difficulty in mentalization. The difference is that in Freud’s case impairment in mentalization is motivated and serves the function of defending the self while in Frith’s model the problem is a neurocognitive abnormality not requiring explanation at a psychological level. Finally, it is apparent that Freud’s view of defence implies that not only can defence motivate distorted perceptual information which may lead to psychotic symptoms such as delusion, but also psychotic defence impairs the capacity for reality testing which thereby maintains the delusional belief. These different theories of the relation of defence and deficit models of psychotic symptom formation are summarized in Table 4.1.

**Therapeutic implications of the integration of defence and deficit models**

To conclude, it is possible to briefly outline some of the therapeutic implications of the integration of deficit and defence models of schizophrenia

| Table 4.1 Defence and deficit theories of the formation of delusional symptoms |
|---|---|
| **Theory** | **Domain** |
| Frith | Deficit |
| Langdon and Coltheart | Deficit |
| McKay | Defence |
| Freud | Defence |

**First factor**
- Social cognition (i.e., ToM)

**Second factor**
- Belief evaluation/Reality

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<th>Theory</th>
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<tr>
<td>Frith</td>
<td>Deficit</td>
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<td>Langdon and Coltheart</td>
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<td>McKay</td>
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Towards an integrated psychotherapeutic model proposed by McKay et al. (2005) using a psychodynamic framework. There are four broad principles which can be elaborated:

1. The therapeutic relationship can be used as a means of understanding and processing mental states and thereby enhancing ToM capacities in a stable and secure setting.
2. The reflection on developmental and life history in the therapy should be restricted to addressing the motivation factors underlying defensive functions only when clearly indicated. However, developmental and life histories cannot be used as a means of explaining neuropsychological deficits in ToM capacities.
3. Practical social skills training can be focused on deficits in social-cognition abilities and integrated with techniques to help the patient understand the meaning of their illness and its optimal management.
4. Therapeutic intervention needs to actively address self-representations and the role of the self in meta-cognition in addition to reparative and palliative interventions following the acute phase of illness. This technique may also have a role in preventative interventions with populations at high risk of developing schizophrenia.

Using the therapeutic relation in the treatment of schizophrenia

Psychotherapeutic outcome research suggests that the therapeutic relationship may be the major contributor to positive therapeutic outcomes. Although this is a matter of considerable debate within the outcome literature on psychological treatments of schizophrenia, controlled trials have often struggled to show a significant effect of experimental therapies compared with supportive therapy or effective standard case management (Jackson et al., 2005; Turkington and McKenna, 2003). Although this is usually interpreted as a negative finding, it suggests that the role of social contact, the therapeutic relationship and forms of implicit social learning from professional contact may be the more active ingredients of therapy. Addington and Gleeson (2005) suggested that CBT, for example, ought to target directly the patient’s social needs and that the therapy ought to emphasize the ‘interpersonal context’ and ‘social consequences of relationships, including the therapeutic relationship’. A mentalization approach provides a more explicit focus on these aspects as well as a theory of why they might contribute to therapeutic efficacy.

Freud’s often noted pessimism that psychoanalysis was an effective treatment of ‘cases of paranoid and dementia praecox’ was in part derived from the inadequate regression-fixation model of developmental psychopathology which suggested that the most severe forms of mental illness were a result of the earliest of libidinal fixations in development (Freud, 1958 [1911]). Freud’s
model of analytic technique was based on the role of free association which aims to relax the function of reality testing in order to reduce the ego's censorship of unconscious thinking. The regressive consequences were quickly discovered and a therapeutic technique was developed which made use of the experiential impact of regression within the analytic setting. However, one aspect of technique in the therapy of psychosis emphasized by analysts as diverse in their theories as Donald Winnicott, André Green and Jacques Lacan is that free association is to be used only when there is sufficient stability of ego function to allow regression to be beneficial, i.e., within the treatment of neuroses only (Green, 1998; Lacan, 2006 [1959]; Winnicott, 1975 [1954]). Since free association is designed to promote regression to earlier developmental stages, it is not a technique to apply in cases of schizophrenia (and so too the more severe personality disorders) where its use is more likely to result in what Balint (1992) termed malignant regression. Instead, in contemporary practice a conversational dialogue is encouraged with the therapist working actively to maintain mental closeness and prevent fragmentation, derailment and incoherence in speech. So too, in the treatment of schizophrenia a high degree of modulation of affect is called for in order to make sessions a safe and productive experience. Psychotic content is never directly challenged or contradicted. Instead the therapist works with the patient to try to understand from where such thoughts or perceptions might arise. Therefore, the treatment is highly collaborative, aims at promoting reflection on mental processes, and emphasizes the alliance between therapist and patient in seeking these common goals. Mentalization, that is the capacity to think about thinking, cannot be achieved in an adversarial atmosphere.

**Understanding developmental and family dynamics in the therapy of defences**

Psychoanalytic theory typically considers delusions to be productions which serve a restorative or adaptive function for the patient, even if they might contribute to what appears to be a social dysfunction. Therefore, the treatment approaches tend to favour constructive elaboration and examination of these productions, rather than a crude attempt to eradicate such symptoms on the grounds of an appeal to their evident unreality (evident, that is, to the therapist). The specific content of such productions, and in particular delusions and hallucinations, is considered to arise in the context of a patient's unique life history, a view which is not incompatible with the idea that the symptoms are formed because of underlying neurocognitive deficits in social cognition.

A psychodynamic treatment of schizophrenia based on mentalizing principles would include attempts to understand the content of psychotic symptoms within a framework of the patient’s current social world, developmental
history and family dynamics. Links between the content of delusions and hallucinations and a patient's personal history and developmental context may provide a narrative framework but ought not to be conceptualized as the sole cause of psychotic symptoms. Following McKay's model, psychotherapies directed towards some delusional symptoms, for instance, are more likely to be effective if they target the underlying motivation for the aberrant belief formation, rather than attempting to convince a patient that their belief is a delusion through recourse to some objective refutation (McKay et al., 2005).

To some extent this would require the development of a formulation that entails aspects of developmental history and family dynamics which, following the psychodynamic tradition, informs the therapist's interventions but would not be communicated directly to the patient. This might be done effectively only on the basis of careful history taking, extended reflection of the patient's material and careful hypothesis testing within the context of a strong treatment alliance.

However, a common error in the application of psychodynamic ideas to the treatment of schizophrenia is to assume that developmental aspects offer an explanatory framework that provides a causal explanation to the patient for the neuropsychological deficits from which they suffer. Evidence from ToM studies would suggest that this approach is misguided in many if not most cases. One can draw from this research a clear demarcation between the deficit and defence model that is valuable clinically. The operationalization of these concepts clinically draws on the distinction between causes and meaning, i.e., deficits in ToM in schizophrenia are likely to have a strong biological basis and can be described using a physical causal model, whereas defences produce effects of meaning; they are a lived experience of the self and its relations with others. The specific form of psychotic symptoms, the content of delusions and hallucinations, may well have links to a patient's life history, development and family dynamics. Working through these aspects of a patient's symptoms can have beneficial results because it provides a meaningful framework and may be restorative of a personal narrative which includes, but is not overwhelmed by, the experience of psychotic symptoms. In this case, working through would consist of trying to help a patient gain some understanding of how their unique life events, family interactions, developmental pathways and family or developmental life stage interacts and influences the onset, content or response to psychotic symptoms. As we shall discuss, personal meaning, a palatable explanatory framework for their mental state experiences, and narrative continuity are critical to the patient's restoration of a functional sense of self.

In essence, the demarcation between deficits and defences reduces the likelihood of damaging modes of practice that result from the failure of integration in the mental health sciences. First, the likelihood of the iatrogenic effect of destroying a patient's sense of agency by declaring they suffer from a mental disease state that has nothing to do with them. Second, the equally
damaging use of speculative and largely unfounded reconstructions of failures in early development or in the ‘care-giving environment’ which the therapist suggests to the patient are the cause of their schizophrenia.

**Skills training to address social cognition deficits**

Given the clear social deficiencies present in schizophrenia and the possibility that, at least in some cases there will be a progressive deterioration over the course of the illness, the direct targeting of ToM abilities would appear to be well within the domain of therapeutic treatments. It also follows that treatments such as the psychodynamic approach, which emphasizes the therapeutic relationship, would predominately address the misinterpretations of self and other implied in psychotic symptom formation, within the therapeutic relationship.

However, a more didactic and explicit focus on enhancing a patient’s social cognition capacity needs to be considered. On the one hand, given the clear developmental and relational context in which ToM emerges, a discrete and simple ToM training model may be of little effect and even be somewhat alienating. Brune (2005) in his review of the ToM literature asserted that ‘future studies may also address whether patients could benefit from cognitive training in this domain’ (Brune, 2005, p. 21). There is somehow an assumption that ToM is acquired by some kind of social-learning process and therefore one could somehow ‘train’ it into patients when it is found to be defective. This is a problematic idea given that we know that ToM is not simply a cognitive skill and even less a learnt behaviour. Acquiring ToM is more likely to be achieved within the context of a specific social relation where aspects of the social interaction can be used for didactic purposes. On the other hand, it follows that if deficits in ToM have a neurocognitive basis, then therapies need to actively and explicitly address these deficits. This will involve a more directed and skill focused approach than is traditionally used in psychodynamic approaches. Whether such a training approach can be integrated with a psychodynamic or cognitive approach or would be better delivered as a separate adjunct therapy in selected cases can be determined only empirically.

**Self-representations in the therapy of schizophrenia**

Both cognitive and psychodynamic therapies for psychosis have specifically targeted the impact upon the self. For instance Jackson et al. (1996) used notions derived from constructivism which suggests that people construct their personal representation of the world, which creates representational models that serve as a framework for the attribution of meaning to personal events. Jackson et al.’s (1996) CBT model is focused on the protection of the self and the resumption of lifespan development following an initial
psychotic episode. The means by which the developmental process is integrated into the therapy is limited in this cognitive-behavioural approach because the focus is more on the impact on the self of schizophrenia, rather than targeting a vulnerability in the self-schema. Indeed, researchers of differing orientations have considered the self to be a key target of therapeutic intervention (Frith, 2005; Morrison et al., 2004). However, the deficit/defence model provides a more robust view of the role of the self in both the vulnerability to psychotic symptoms and the role of the self in the formation of these symptoms.

For example, by way of comparison with autism, ToM research suggests that patients with schizophrenia suffer from an inability to understand themselves by accurately assimilating the perspective another has of them and this deficit crosses not only cognitive abilities but the processing of emotional states. One can infer that impairment in the critical feedback mechanism of self development would be an accumulating impoverishment in the representational schema of the self and other. Although clearly exacerbated by the onset and severity of psychotic symptomatology, some degree of ToM deficits typically predate onset as well as persisting beyond the resolution of a specific psychotic episode. This suggests that the psychotherapeutic targeting of ToM, broken down into self, other and self-other representations, could well be a key strategy in both preventative interventions for groups at high risk of their first episode of psychosis as well as having an important role in the prevention of relapse following initial psychotic episodes.

The use of a mentalizing framework aims to recreate within a therapeutic setting a situation where the boundaries of self can be recognized, intentionality of thought correctly attributed and the mental processes of others can be interpreted. This provides an experience of the patient’s self as real via the therapist. Based on this theory, one can suppose that therapeutic gains are made at the point where this recognition can be clearly perceived by the patient. Unlike prior psychodynamic models, each of the goals which follow from this theory can be defined and measured within a programme of research. One would predict that a patient’s recovery from illness and decline in psychotic symptoms would improve alongside improved capacities for mentalization.

Given the preliminary nature of research into ToM deficits in patients with schizophrenia, carefully targeted psychotherapeutic techniques must be framed in a tentative manner. The conceptual integration of deficit and defence models described in this chapter may well provide an enhanced framework within which to consider new therapeutic models and to reconfigure psychodynamic therapies in a manner guided by this research. In theory, it appears that orienting interventions around different strategies for addressing deficits in social cognition as distinct from defensive processes appears to be a reasonable approach. Many of the clinical applications discussed here can be integrated with the current variety of psychosocial
interventions used as interventions for schizophrenic patients since all need to be guided by a coherent theory of the therapeutic aspects of their intervention. Furthermore, it is unlikely that any single psychological therapy will meet all of the needs of the diversity of patient suffering from schizophrenia. Whether enhanced efficacy for the psychosocial treatment of schizophrenia can be derived from the integration of deficit and defence models will need to await the further development, implementation and testing of such therapies in a clinical setting.

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References

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