Critically evaluate the contribution of cognitive and psychoanalytical models to our understanding of mental health difficulties in terms of emergence, maintenance and intervention.

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Introduction

In this essay I will assess the contributions of psychodynamic and cognitive theories, models and interventions to the assessment, understanding and treatment of depressive spectrum disorders. Ireland has a high prevalence of depression, currently 12.3% (95% CI 5.7-26.3) of urban Irish and and 7.9% (95% CI 3.7-16) of rural Irish suffer from major depressive spectrum disorders (Ayuso-Mateos et al, 2001). A surprising disparity, given that suicide rates in rural Ireland have increased at a much more dramatic rate in recent years (Kelleher, Chamber & Corcoran, 1999).

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (APA, 1994), together with the International Statistical Classification of Diseases and Related Health Problems (ICD-10) (WHO, 1992) demarcate the Western bio-psycho-social approach to the diagnosis and differentiation of depressive disorders. In defining depression, the DSM-IV-TR (closely mirroring the ICD-10), distinguishes several Axis I (Clinical) Disorders on a multi-axial assessment designed to capture the relevant environmental, psychological, social, and functional factors involved in mental illness. These include Dysthymic Disorder (DD) – typified by an ongoing low mood, unusual patterns of eating, tiredness, sleep disturbance and lack of hope, over at least a two year period, free from episodes of major depression (MD) (although it may later combine with a major depressive episode, leading to ‘Double Depression’); and Major Depressive Disorder (MDD) or unipolar depression – a single episode or recurrent disorder, from mild to severe, typified by disruption of sleeping patterns, decreased mood, despair and increased or omnipresent anxiety (APA, 1994).

The diagnosis of depressive spectrum disorders is essentially syndromal (based on external symptoms), and although psychosocial and environmental functioning are recorded – both are negatively defined (focusing on problems, rather than the combination of risk and resilience factors), limited to the previous year, and diagnostically irrelevant – collected in order to provide additional treatment and outcome estimation information, rather than illness classification or causation.
Morris (2006), provides an alternative ‘Life Event and Contextualised Social Model of Depression,’ - rooted in the ‘three systems approach’ underlying cognitive behavioural therapy (Rachman and Hodgson, 1974, cited Hawton et al, 1989) - emphasising social, cognitive, and physical (somatic, appetitive and focus) domains as both causative and amenable to direct intervention (Morris, 2006).

Emergence of Depression

To the psychodynamic perspective, depression represents both MD, and the maladaptive anhedonic Depressive Personality (DP); the specific disorder being distinguished by its underlying psychodynamic structure, rather than a checklist of behaviours or mood states (Vaslamatzis, 2002). DP, a more pervasive and long lasting ‘pattern of inner experience’ although not formerly recognised in the DSM-IV-TR, is listed as of interest for further study in its appendices (APA, 1994).

Freudian psychoanalysts view an (actual or affective) loss of a primary caregiver as responsible for the reaction formation of self directed anger, that becomes repressed in the depressive personality (Abraham, 1948, cited in Huprich, 2001).

Psychodynamic Object Relations Theorists by contrast, construe distinct modes of depression as arising from varying developmental outcomes of the establishment of symbolic relationships to the introjected ‘good object’, during the paranoid-schizoid stage of infancy. In MD, immature defences (such as denial) are resorted to in order to restore the destroyed ‘love object’ (an internalised representation of the primary care giver), while dysthymia is caused by an innate unconcern on the part of the primary carer, and DP results from the infants perception of injuries to the care giver suffered under the poisonous attack of the infants bodily substances (Vaslamatzis, 2002) (Gomez, 1997).

This rooting of depression in the primary (usually maternal) bond seems initially reminiscent of the discredited schizophrenogenic mother hypothesis, which posited the conflicting demands of a simultaneously over indulgent and neglectful mother as the root of the personality dissolution of schizophrenia (Hartwell, 1996). However, research does
support a link between depression in mothers and corresponding depressive disorders in children and adolescents.

Investigations of depression from a developmental psychopathology perspective - emphasising an adaptive, integrative, causal view of disorder arising from developmental disruption or delay - have found increased levels of depression in the children of depressives, concurrence of onset of child and parent depression, and relationships between the behaviour of depressed mothers (and even non-depressed mothers, merely adopting similar aversive or unresponsive behaviours) and age appropriate responsiveness, behavioural control, and socialisation in their offspring (Chichetti & Toth, 1998).

Similarly, behavioural studies in very young (6 week old) infants, have confirmed a vulnerability to disruptions of parental engagement. Murray (1980), found that specific patterns of infant withdrawal resulted depending on the nature of experimental mother child interaction disruption. Interruption by a third party conversing with the mother resulted in attentive observation on the part of the infant. Facial unresponsiveness on the part of the mother led to increased infant signalling, distress and ultimately withdrawal. Disruptions of the pattern of maternal responses caused confusion and avoidance. Such behaviours support the retreat from the object in response to inadequate affective care proposed by object relations theorists (Murray, 1980, cited in Murray 1991).

Huprich (2001), lent support to the typology of depressive disorders as dictated by the underlying structure of psychodynamic object relations. Depressive personality, dysthymic and dependent adolescent individuals – as judged by a variety of psychometric tests rather than clinical diagnosis, and not currently suffering from dysphoric mood - differed from controls in their ability to comprehend the relationships of others, and exhibited decreased positivity and satisfaction with their own relationships (Huprich, 2001).

A wide variety of cognitive aetiologies have also been proposed for depression. Cognitive systems theory proposes a ‘dyadic’ structure in the developing infant, in which

Beck’s cognitive theory of depression (Beck, 1972 cited in Moilanen, 1995) situates the etiology of depression in negative and distorted thinking, maladaptive schema leading to the development of dysfunctional attitudes across a variety of domains. Here too, evidence exists of the role of maternal depression; a variety of studies have established a link between maternal depression and negative cognitive styles in children (Kuyken, Dalgleish & Holden, 2007).

A meta-analysis of studies of chilren’s attributional style (causal expanations) found that children and adolescents self reporting depressive symptomology, interpreted negative events as caused by unchanging internal deficiencies, and positive events as resulting from brief specific external factors; supporting another cognitive model - Seligman’s cognitive Learned Helplessness Theory (Gladstone et al, 1997), which views conditioned hoplessness combined with a negative explanatory style, as responsible for reductions in percieved agency underlying depression (Abramson et al, 1978).
A more recent lonitutidinal study indicates that while internal attributional style (consistently negative over a broad range of domains) combined with stress to contribute to depressive symptoms in adolescence, in childhood it did not appear to be causal in depression (Cole et al, 2008).

**Maintenance of Depression**

The defence mechanisms initially identified by Sigmund Freud and elaborated into a hierarchy by Anna Freud & Vaillant, have been one of the least controversial and most widely utilised facets of psychodynamic theory (Hentschel et al, 2004). Defence mechanisms act to protect the ego from neurotic anxiety, but in doing so may maintain
the maladaptive behaviours and negative perspective typical of depression. Cramer & College (2002), found support for this hypothesis, relating the use of the defence mechanism of denial by young adults to anxiousness and immature behaviour and (in men) the use of projection to a distrustful, hypervigilent cognitive style. They suggest that the use of such developmentally inappropriate defences symptomises poor ego development, and leads to a distorted construal of social interactions and the environment; the defences adopted interacting with socially conditioned gender roles to produce socially approved or dysfunctional behaviours (Cramer & College, 2002).

Bandura’s Socio-Cognitive Theory suggests that self-efficacy beliefs - client perception of their ability to perform in a specific domain – regulate affective states (Muris, 2001), and influence depression. A large cohort (n=596) study of Dutch adolescents, confirmed that low efficacy levels contributed to variance in anxious and depressive symptoms, even when controlling for neuroticism (Muris, 2001).

Central to Cognitive Therapy’s model of the maintenance of depression (Beck 1976, cited in Hawton et al, 1989), is the idea that thinking influences mood – specifically that assumptions acquired during the course of development establish criteria for success, which can result in mood reducing negative automatic thoughts when they are not met. In this dysphoric state individuals become vulnerable to distorted self perpetuating cognitions, negatively framing their self concept, social interactions and environmental functioning, and resulting in depression. Thus vulnerabilities established though negative childhood schema, rooting self esteem in social or occupational success, can be reactivated by challenging life events (Carr, 2004).

**Therapeutic Interventions**

Psychodynamic approaches to the treatment of depression can be found in orthodox Freudianism; in the theories of Melanie Klein and the wider Object Relations and Attachment models her work inspired (Gomez, 1997); and in the brief psychodynamic
therapy of Strupp and Blinder, amongst a myriad of others. Disparate though such
theories and therapies are – in practice their efficacy has repeatedly been evaluated as
broadly similar to one another and to cognitive therapy (Oei & Free, 1995), and roughly
equal to medication alone when the effect of therapist alliance is removed (Robinson et
al, 1995). Such parity of outcomes can be explained in part by the fact that the
mechanisms of positive intervention may lie more in the shared features of all ‘talking
cures’ – the client therapist relationship, the esteem generated from the attention and trust
placed in the client; in the monitoring of clients overt behaviour; and in the contemporary
‘eculmenical’ approach to the delivery of therapeutic interventions.

Goldsamt et al (1992), in an examination of the application of psychotherapy to a single
client by key theorists in three schools (Beck’s Cognitive Therapy, Miechenbaum’s
Cognitive Behaviour Modification, and Strupp’s Short Term Psychodynamic Therapy);
found that when client interactions were isolated from the specific terminology of a given
model, the therapeutic focus of the three approaches did not cohere as clearly with their
avowed content as might have been expected. While Strupp’s Psychodynamic session
focused on the clients past life more than either of the cognitive approaches, both of
which emphasised cognitive distortions and focused on future behaviours; Strupp and
Meichenbaum were more similar in their attendance to the clients own responsibility for
their interpersonal difficulties (Goldsamt et al, 1992). The single session, single case
methodology of this study limits the generalisability and representativeness of its
conclusions.

Psychodynamic therapy adds to the empathy and tolerance present in most forms of
psychotherapy, an awareness of the transference of inappropriate emotions and
attachments during therapy. To this construal, the resolution of both the clients
perspective of the therapist as an initially supportive but later threatening object, and the
therapists counter-transference (internalisation) or rejection of this role, together with the
dilution of underlying oedipal and pre-oedipal (object relations) conflicts, are key in the
successful resolution of depressive personality (Vaslamatzis, 2002). This is a profoundly
interactive perspective, emphasising the capacity of the therapist to internalise the
projected identities and needs of the client, the necessity of identifying the unconscious motivation behind their own reactions.

Ellis’s Rational Emotional Therapy, a subdiscipline of cognitive behavioural therapy, is based upon the fundamental interconnectedness of cognition, behaviour and emotion. Changes in maladaptive causal attributions, self demands and commitments are seen as modulating the destructive emotional and behavioural consequences of ‘activating events’, (real or imagined) challenges which might otherwise result in psychological disorder (Ellis, 2005). Ellis’s concept of hierarchical goals motivating human behaviour mirrors Maslow’s pyramids of needs; his core of ‘musts, ought to’s, have to’s and got to’s, resembling Roger’s conditional positive regard, and the Freudian superego.

MacInnes, 2004, examined the theoretical assumptions underlying Rational Emotive Therapy from the perspective of evidence based medicine – the movement to root therapeutic interventions in efficacy research (Sackett, 1996). MacInnes carried out a metastudy of research examining the relationship between beliefs, emotions and consequences, and found a deficit of evidence for both Ellis’s proposed connection between irrational beliefs and dysfunctional emotions, and the primacy of individual’s self demand beliefs in establishing dysfunctional inferences (MacInnes, 2004).

The duration and expense of psychodynamic therapy are frequently cited as evidence for the greater utility of cognitive therapy, even if outcomes of both are broadly similar. Shapiro et al (1994) contrasted the efficacy of cognitive behavioural therapy (CBT - cognitive therapy emphasising behavioural strategies) and psychodynamic therapy (Hobson’s Conversational Model) for the treatment of depression, in a large group (n=117) of clients. While Shapiro et al (1994), did not investigate recurrence (beyond a single three month follow up); when controlling for researcher theoretical orientation and the initial severity of client depression, both therapies were equally effective and equally rapid in improving a variety of measures of mood, self esteem and social adjustment over brief (8 and 16 week) course of intervention. CBT did however demonstrate greater efficacy at improving scores on the Beck Depression Inventory (BDI), while the longer
16 week course of therapy was found to be significantly more effective for severe depression (Shapiro et al, 1994). Shapiro et al (1994) suggest that the common theoretical background of CBT and the BDI, may to some extent bias treatment efficacy comparisons on this measure.

A consensus has formed in recent years that depressive disorder represents a chronic illness (Glass, 1999). Researchers have been critical of the levels of remission, which may be as high as 80% over the life course (Judd, 1997 cited in Teasdale et al, 2000), identifying that residual symptoms predict recurrence in MD (Fava et al, 2007). A ten year follow up of the recovery of patients at a variety of US academic treatment centres demonstrated that residual depressive symptoms were strongly predictive of repeated episodes of unipolar depression (Judd et al, 1998). A higher relapse rate seems to apply to ‘double depression’, as well as a lower level of recovery - when defined as the resolution of both underlying depression and MDD (Keller et al, 1982).

Psychiatrists faced with this dilemma are resorting to ever more extreme and potentially damaging interventions like magnetic seizure therapy, deep brain stimulation and vagus nerve stimulation (Rau et al, 2007).

A meta-analysis of cognitive and behaviour therapies revealed an improvement in recurrence rates (when the effect of therapist alliance was removed) over the use of psychopharmacological interventions (Gloaguen et al, 1998). More recently Vittengl & Clark (2007) confirmed a reduced rate of relapse into unipolar depression with cognitive behavioural therapy (especially with the addition of continuation phase therapy) versus treatment with medication alone.
Conclusion

While the psychodynamic and cognitivist traditions represent diametrically opposed developmental and functional models of cognition and dysfunction - positing either the cathartic release of unconscious energies or the restructuring of conscious and preconscious cognitive distortions as the routes to the amelioration of psychological distress; these contrasting perspectives share a faith in the significance of phenomenological interventions. They emphasise the internal meaning of disorders and symptoms – rejecting tabula rasa behaviourist, or biomedically deterministic representations of psychological functioning and illness. Although cognitive therapies emphasise a change in current frames, schemas and scripts rather than an examination of the origins of neurotic conflict, they share with psychodynamic approaches a developmental perspective, rooting the maladaptive counterproductive thoughts and behaviours of the depressive disorders in the psycho-social environment of infancy, childhood and adolescence.

The significance of the maternal bond to the emergence of depression, proposed by psychodynamic theorists, has been supported. This does not however, necessarily lend credibility to the underlying exchanges of anxiety and antagonism, or the paranoid schizoid and depressive stages of development hypothesised by Klein. As with many psychodynamic theories, such hypotheses may be intrinsically untestable.

Both the psychodynamic and cognitivist perspectives represent broad heterogeneous schools, with a diversity of theoretical approaches, with both common and contradictory implications for therapeutic intervention. At the same time both approaches represent a viable alternative to pharmaceutical and other psychiatric interventions for the treatment of depression, free from the danger of iatrogenic disorders; and possessing a theoretical explanation for their efficacy, that neuropsychiatric approaches have thus far failed to produce. Research is needed to contrast the efficacy of brief and traditional forms of psychoanalytic therapy, cognitive therapies and both time limited and ongoing psychopharmacological interventions, in preventing the recurrence of MD, and resolving
the underlying disease process hypothesised as responsible for depressive spectrum disorders.
References


