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Review Article

Dysthymia in Clinical Practice

THE WPA DYSTHYMIA WORKING GROUP[1]

Background. Dysthymia has been reconceptualised in recent years from a personality disorder to a chronic affective disorder. It is incorporated into both the DSM and ICD diagnostic systems.
Method. The members of the WPA Dysthymia Working Group combined the results of their manual literature searches with a search using Medline.
Results. Available data are summarised under the headings of classification, epidemiology, validity, comorbidity, course and outcome, pharmacotherapy and psychotherapy. The co-existence of major depressive disorder, constituting 'double depression' is of particular importance.
Conclusions. Improved knowledge of this disorder has led to a more positive approach to treatment, in which antidepressants can usefully be complemented by psychosocial measures. A high proportion of cases remain unrecognised in most populations, leading to prolonged morbidity and distress, much of which is now treatable.

In its Greek origins, the term 'dysthymia' means 'ill-humoured' and can be traced back to the description by Hippocrates of the melancholic temperament. The first clinical description was by Kahlbaum in 1863; he regarded it as a chronic form of melancholia, in contrast to 'cyclothymia' which was a disorder characterised by fluctuating mood. Following the same conceptual tradition, Kraepelin (1921), referred to the 'depressive temperament', and believed that this was the substrate from which manic–depressive illness developed; it often led to a protracted depressive phase, preceding a more acute illness, but was essentially part of the same morbid process. The difficulty of separating an individual's habitual mood from low-grade depression led to repeated discussion as to whether 'depressive personality', in the Schneiderian sense, is different from dysthymia as a sub-affective disorder (Hirschfeld *et al*, 1989).

DSM–II (American Psychiatric Association, 1968) included 'Neurotic depression', in which the emphasis was placed on personality aspects, rather than on symptoms; chronic states of depression were classified under personality disorders and neuroses, but persistent affective pathology was insufficiently recognised. ICD–9 (World Health Organization, 1978) listed 'Depressive neurosis', which also subsumed shorter, non-chronic episodes; as in DSM–II, this was a diagnostic concept heavily influenced by psychoanalysis. In 1978, Akiskal *et al* reported a prospective follow-up of neurotic depressives; 40% were diagnosed as having major depressive disorder (MDD) while many of the remainder were considered to be dysthymic. The latter pursued a low-grade, intermittent, or chronic course. In DSM–III (American Psychiatric Association, 1980), all chronic depression lasting more than two years was defined as 'Dysthymic disorder'; this newly created category subsumed many depressive illnesses that had been previously considered to be characterologically based. For this diagnosis, at least a two-year history of continuous or numerous periods of depressive symptoms, characteristic of major depression but not meeting the full severity and/or duration criteria for MDD, was required. Thus, there was a shift from the original view of dysthymia as a neurotic personality disorder to the current concept of it as an affective or mood disorder, characterised by its chronicity and low-grade or subsyndromal nature.

This reconceptualisation has led to a gradual change in the approach of psychiatrists to its treatment (Burton & Akiskal, 1990). DSM–III–R (American Psychiatric Association, 1987) brought together mild affective conditions with severe ones which they otherwise resemble phenomenologically. The key characteristics of dysthymia were:

(a) not a residuum of major depressive disorder
(b) a chronic course of more than two years with persistent or intermittent symptoms
(c) low-grade symptomatology
(d) insidious onset

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(e) concurrent pathology of character
(f) compatibility with stable social functioning in the community.

On the other hand, no spontaneous upward swings towards hypomania that would qualify for a 'cyclothymic' diagnosis should have been present. In DSM–IV, although the core set of symptom criteria for dysthymia remains largely unchanged, the appendix contains a new set which concentrates on the more cognitive features and the various patterns of course. ICD–10 (World Health Organization, 1992) defines dysthymia as a chronic depression of mood which does not currently fulfil the criteria for recurrent depressive disorder (of mild or moderate severity) in terms of either severity or duration of individual episodes. ICD–10 dysthymia includes depressive neurosis, depressive personality disorder, neurotic depression (of more than two years' duration), and persistent anxiety–depression.

Classification

Dysthymic disorder can be classified as a sub-syndromal yet more chronic form of MDD, without psychotic features; the primary distinction is that dysthymia is chronic, but symptomatically less severe (Smith & Weissman, 1992). In dysthymia, the depression tends to be more subjective than objective; it is not sharply demarcated from the patient's usual self, there is no severe agitation or retardation, and marked disturbances in appetite or libido are unusual (Akiskal, 1993). Similarly, the irritability, impulsivity, and instability of mood or bipolar disorder are not seen. However, large areas of overlap inevitably exist between the definitions of dysthymia, MDD, and personality disorders. While Klein (1990) maintained that there is still a need for the category of non-dysthymic depressive personality disorder, Akiskal (1981*a*, 1983) states that many patients with borderline personality disorders also suffer from lifelong cyclothymia and dysthymia, with transient shifts into major affective episodes, for example melancholic or mixed states.

Epidemiology

Data from the NIMH Epidemiologic Catchment Area (ECA) study indicated a lifetime prevalence rate in those aged over 18 of 3.1% for dysthymia and 4.4% for major depression (Eaton & Kessler, 1985). Diagnosis was based on the NIMH Diagnostic Interview Schedule (DIS), a structured interview for use in epidemiological studies (Robins *et al*, 1981). Dysthymia is defined by the DIS as depressed mood lasting two years or more, with symptoms from at least three symptom groups defined by DSM–III, and no psychotic features. Rates were highest in the 18–64 age group, declining from age 65, and significantly higher in poorer people aged between 18 and 44. More than 75% of people with dysthymia had other disorders, particularly major depression, anxiety disorders, and substance abuse (Weissman *et al*, 1988). During a one-year period, it was estimated that in the USA, 3 500 000 persons with dysthymia were seen in an ambulatory mental health or addiction setting, in addition to 430 000 who had at least one in-patient admission (Narrow *et al*, 1993).

In a study that compared early- and late-onset dysthymics on demographic, clinical, and familial variables as well as short-term outcome, it was found that the relatives of early-onset probands had significantly higher rates of major affective disorder than those of the late-onset subjects (Klein *et al*, 1988*a*). Early-onset dysthymics sought treatment more frequently and had significantly higher rates of lifetime major depressive episodes and anxiety disorders, as well as higher rates of major affective disorders in their first-degree relatives. Throughout the study and at the six-month follow-up, these subjects also suffered from significantly higher levels of depression.

Most studies have been of dysthymics whose illness began insidiously in childhood or adolescence (Akiskal *et al*, 1980; Keller *et al*, 1983; Kovacs *et al*, 1984; Kashani *et al*, 1985; Klein *et al*, 1988*b*; Hammen *et al*, 1990; Harrington *et al*, 1990; Lewinsohn *et al*, 1991; Garrison *et al*, 1992), and it is not known whether or not these patients bear any relationship to late-onset cases (McCullough, 1988). However, low-grade depressive symptoms seem relatively common in the elderly (Kivela & Pahkala, 1989), and the low incidence of first-onset major depression in that age group (Cross National Collaborative Group, 1992), compared with young and middle-aged adults, might be partly accounted for by patients with dysthymic-like clinical presentations who had been missed earlier. Studies of low-grade affective symptoms in this age group, though, have been hampered by the degree of comorbidity with other disorders.

Clinical and scientific validity

Clinical features

Despite the formal recognition of dysthymia as a subtype of affective or mood disorder in both DSM–IV and ICD–10, two questions have been raised about its validity: can dysthymia be better

conceptualised as a personality (or neurotic character) disorder, rather than an affective disorder (Peron-Magnan, 1992); and can it be distinguished from major depressive illness (Kocsis & Frances, 1987)? This challenge has therefore come from opposite positions. The need for the dysthymic construct (Burton & Akiskal, 1990) arose because of the existence of a large spectrum of patients with fluctuating, intermittent, or chronic depression. The basic pattern in these patients is one of low-grade symptoms which have begun insidiously, in most instances before the age of 25. Dysthymia without superimposed major depression accounts for about a third of all subjects meeting this diagnosis over a lifetime, their disability stemming primarily from the long-standing morbidity. They often consult general practitioners with complaints involving ill-defined malaise, lethargy, and/or fatigue.

The symptoms consistently used to define major depression and dysthymia respectively differ in severity and duration, rather than in kind, leading some researchers to question whether they are actually separate disorders. Distinctions between chronic major depression and dysthymia are based on the previous course: in the former, this has been of a well-defined mood disorder, without prior low-grade symptoms. The most typical pattern of dysthymia, on the other hand, is an insidious onset of symptoms at an early age, followed by a fluctuating or progressive course, which will often meet current diagnostic criteria for MDD. This may represent a more severe form of typical recurrent unipolar affective disorder than the episodic type, but many patients with this pattern seem to be responsive to treatment with antidepressants. Whenever dysthymia and major depression co-exist, regardless of which came first, there is believed to be a unitary phenomenon which can only be differentiated on a continuum of severity and chronicity (Keller & Russell, in press). However, dysthymic patients often seek psychiatric help when their depression is accentuated to a level at which it meets the criteria for MDD, giving rise to the 'double depressive' picture (Keller *et al*, 1983).

Although attempts to categorise depressive illness according to its phenomenology have been mixed, the distinction between primary, secondary, and double depression has been demonstrated to have predictive value in terms of course of illness, time to recovery, and rate of relapse (Keller *et al*, 1983; Keller & Lavori, 1984; Black *et al*, 1987; Klein *et al*, 1988*b*). Low-grade manifestations of dysthymia are no less affective than the full-blown clinical features of depressive illness, but long-standing sub-affective symptoms can lead to such interpersonal difficulties as dependence, low frustration tolerance, and rigidity (Akiskal, 1983). Appropriate treatment of both the dysthymic substrate and superimposed MDD should contribute to a lessening of interpersonal conflicts (Akiskal *et al*, 1980), since low-grade depression, beginning in late childhood or adolescence, could result in later neurotic disorders (Versiani & Nardi, in press), including social-phobic and avoidant patterns. However, overlap between dysthymia and generalised anxiety disorder (GAD) seems to be relatively minimal (Sanderson *et al*, 1992; Shores *et al*, 1992); the gloomy, withdrawn, guilt-ridden, lethargic, and anhedonic dysthymic, with low self-confidence, is quite distinct from the worrying GAD type with fluctuating autonomic hyperactivity, excessive vigilance, tension, and gastrointestinal distress.

External validating strategies

Clinically, dysthymia appears to show reasonable delimitation from related conditions; it can be understood within a pathogenetic framework whereby subdepressive manifestations chronologically precede more florid episodes. Furthermore, studies in adult dysthymics have shown high rates of familial mood disorders (Rosenthal *et al*, 1981; Kovacs *et al*, 1984; Hammen *et al*, 1990; Harrington *et al*, 1990), strengthening the postulated link between dysthymic and major affective disorders. Further confirmation has come from the increased risk of dysthymia in the offspring of both unipolar (Klein *et al*, 1988*c*) and bipolar (Akiskal *et al*, 1985) adults. Furthermore, studies in dysthymic children (Kovacs *et al*, 1984; Hammen *et al*, 1990; Harrington *et al*, 1990) have consistently showed high rates of superimposed major depression of a recurrent nature, as well as of familial mood disorders. Finally, dysthymia has been found to be the precursor, over a 12-year period, of MDD in a prospectively examined cohort of dysthymic children; as they passed through puberty, some of these children developed hypomanic and mixed-manic episodes (Kovacs & Gatsonis, 1989).

The link to primary mood disorders is further strengthened by psychopharmacological studies demonstrating the efficacy of various antidepressants in dysthymia. The possible connection of a subgroup of dysthymic patients to bipolar disorder is indicated by reports of transient hypomanic-like responses to antidepressants (Rosenthal *et al*, 1981) or to sleep (Rihmer, 1990), or even occurring spontaneously (Klein *et al*, 1988*b*). Without treatment, the naturalistic outcome of dysthymia – especially in its 'double depressive' form – is relatively poor (Akiskal &

Table 1
Evidence of considering dysthymia as a sub-affective disorder[1]

Familial affective loading
Phase advance of REM sleep
Diurnality of inertia, gloominess and anhedonia
TRH–TSH challenge test abnormalities
Prospective course complicated by recurrent major depressive episodes
Positive response to sleep deprivation
Positive response to selected thymoleptics
Treatment-emergent hypomania

1. Updated from Akiskal (1993).

Weise, 1992): an average of two-thirds of these patients continue to exhibit depressive symptoms over a decade.

The main biological similarity between dysthymia and major depression comes from sleep electroencephalographic studies. Both residual depressive (Akiskal, 1982) and dysthymic (Akiskal *et al*, 1980) patients have generally been shown to have short REM latency and related circadian abnormalities, similar to those of primary major depressives. Indeed, some patients show marked diurnality (Akiskal *et al*, 1980), with gloominess, anhedonia, and lassitude much worse in the morning. Such trait markers as the TRH–TSH test and REM latency findings, have generally been positive (Howland & Thase, 1991), supporting the view that dysthymia represents a trait depressive condition – a sub-affective disorder or one of the sub-symptomatic predisposing conditions for affective illness (Akiskal & Akiskal, 1992), rather than a 'character' or 'neurotic' disorder. The frequent association of dysthymic and major depressive disorders can be seen as indicating the fluid boundaries in a spectrum concept of affective illness (Table 1).

Course and outcome

Course

Symptoms normally associated with dysthymia such as anger, irritability, and anxiety are also criteria for a variety of other medical or psychiatric diagnoses, but one of the distinct features of dysthymic disorder is chronicity. Keller *et al* (1982) reported a median duration of five years before the first superimposition of an episode of major depression. The Collaborative Depression Study (CDS) (Keller & Shapiro, 1982) showed that 26% of cases also had a previous underlying chronic depression of at least two years' duration. In fact, the great majority of clinical cases of dysthymia go on to develop a major depression, thus constituting 'double depression' (DD). Of the DD patients, 96% had previously had chronic minor depression lasting three years or more, and follow-up showed that those with DD had a significantly higher rate of relapse than those with MDD only. Perry (1985) found that 87% of borderline patients who had dysthymic disorder also had a major depression, and that of those with antisocial personality who had a dysthymic disorder, 93% also had a major depression.

Markowitz *et al* (1992) investigated DSM–III dysthymic disorder in 90 psychiatric out-patients, finding that chronic depression followed one of four temporal patterns. Primary dysthymia, denoted by insidious onset at an early age becoming complicated by a subsequent psychiatric disorder, particularly MDD, was found in 71% of the subjects, while 21% suffered from secondary dysthymia following other psychiatric disorders. Chronic major depression, present in 9% of the subjects, was characterised by a more acute onset at an older age and greater symptomatic severity. There was also a pattern wherein anxiety disorders were the precursor of a later onset of dysthymia: 22% of the dysthymics meeting criteria for MDD in this study also met criteria for panic disorder. However, only 43% of the dysthymic subjects reporting early onset and chronicity had been clinically diagnosed as having dysthymia.

Wells *et al* (1992) compared the course of depression among 626 subjects suffering from a combination of serious physical disorders and major depression, dysthymic disorder, and either DD or depressive symptoms with no current depression. The baseline severity of the depressive symptoms was greatest in patients with DD, but after two years, patients with dysthymia had the worst outcome, even in the absence of MDD.

Children and adolescents

Until the late 1970s, depressive disorders before adult life, including dysthymia, were usually overlooked because they were thought to be changes which occur during adolescence. However, several studies of early-onset dysthymia (Kovacs *et al*, 1984) suggest that in children, dysthymia has an earlier age of onset than MDD. The age-range for the onset of dysthymia in school-age depressed children was 6 to 13 years, and recovery was found to be slow, with a median duration of 3.5 years; over 70% of these children experienced at least one episode of major depression over a 7-year follow-up period. It was concluded that although dysthymia and MDD are related, they represent separate disorders in school-age children,

and that a younger age of onset predicts a more prolonged episode of dysthymia. Akiskal *et al* (1981*b*) found that in 137 out-patients with chronic depression, 37% were patients with 'intermittent subsyndromal depression' which had an insidious onset in childhood or adolescence.

Recovery and relapse

There is a significantly higher recovery rate at two years from MDD for patients with a 'double depression' (97%), compared to patients with major depression alone (79%). However, this does not indicate that on recovery, people with DD are healthier than those with major depression alone. Rather, it is easier to return to a state of chronic or intermittent minor depression than to a 'usual' self characterised by no depression at all (Keller & Shapiro, 1982). A two-year follow-up study by Keller & Lavori (1984) found that although 97% of the patients had recovered from their MDD, only 39% had recovered from the underlying acute and chronic phases of dysthymia. Thus, subjects with dysthymia are more likely than those with MDD and no dysthymia to continue to have depressive symptoms after two years of follow-up.

Approximately 25% of the subjects who entered the CDS Study with an episode of major depression were found to have a dysthymic disorder which preceded the onset of major depression by at least two years (Keller, 1985). The presence of DD greatly influences recovery rates, time to relapse following recovery, and predictors of these outcomes in patients who have MDD. Over a two-year follow-up period, the 32 with double depression had significantly faster cycles of recovery and relapse during that time than the 101 without DD.

Comorbidity

Akiskal *et al* (1981*b*) reported that 90% of 137 out-patients originally diagnosed with dysthymic disorder also suffered from major affective episodes. Data from the ECA project (Weissman *et al*, 1988) showed that 75% of the patients with dysthymia had other disorders, particularly MDD, anxiety disorders, and substance abuse; the comorbidity of dysthymia was highest with major depression, followed by panic disorder. Keller & Sessa (1993) estimated that 75% of the people who have MDD, and almost all of those who suffer from dysthymia, also have a substantial number of anxiety symptoms. Kocsis & Frances (1987) found that 41% of out-patients with chronic depression were diagnosed as having not only dysthymic disorder and major depression, but also personality disorder – 13% dependent, and 11% atypical, mixed.

Considerable comorbidity of dysthymia with substance abuse has been identified, though it is still uncertain whether or not dysthymia precedes – and predisposes – to these disorders (Weissman *et al*, 1988; Markowitz *et al*, 1992). Matthew *et al* (1993) explored the differences in the prevalence of psychiatric disorders in persons who did or did not have a history of familial alcoholism: adult children of alcoholics experienced significantly higher lifetime rates of dysthymia, compared to age- and sex-matched controls. A community survey of psychiatric morbidity in Shatin, Hong Kong showed that alcohol misuse and dysthymic disorder were among the most common diagnoses (Chen *et al*, 1993). King *et al*'s (1993*a*) studies of depressed adolescent in-patients reported moderate to heavy alcohol use: all those with dysthymia were classified in the moderate/heavy drinking group, and 41% had preceding dysthymia, compared to only 4% of other depressed in-patients. Moderate-to-heavy alcohol consumption was also associated with greater severity of current symptoms; alcohol misuse and depression have been associated with an increased risk of suicidal behaviour in both adolescents and adults (Asnis *et al*, 1993; King *et al*, 1993*b*; Pfeffer *et al*, 1988).

Pharmacotherapy

Clinical and methodological considerations

The relatively recent identification of dysthymia as a discrete entity has been the main reason for the relatively late application of pharmacotherapeutic approaches; before 1980, chronic depressive disorders, including dysthymia, had not been considered responsive, so that there was little interest in controlled studies. Most were either single-drug, open trials or had used a mixture of drugs, and many were analysed retrospectively (Conte & Karasu, 1992). However, even these may be given some credibility, because of the poor response to non-specific dynamic psychotherapy and low placebo response (10–20%) found in these patients (Frances *et al*, 1993).

Prior to the recognition of dysthymia as primarily an affective disorder, benzodiazepines were commonly prescribed to control the associated anxiety; however much this complaint may be reduced, depressive symptoms do not show sustained improvement, so that anxiolytics are often over-prescribed (Finlayson, 1989). Earlier studies with imipramine (Kocsis *et al*, 1985), desipramine, nortriptyline and lithium

(Akiskal *et al*, 1980) suggested that improvement could occur with these in a proportion of patients complaining of dysthymia for five or more years. Interpersonal and social functioning often improved in tandem with symptomatic control of the dysthymia. Overall, the ratio of response to antidepressants v. placebo strongly supports the efficacy of antidepressants in dysthymia: tricyclics (TCA), selective serotonin reuptake inhibitors (SSRIs), and reversible inhibitors of monoamine oxidase-A (RIMAs) were 2–3.75 times more effective than placebo (Kocsis, 1993). As a result, antidepressants have been proposed as the mainstay of the management of dysthymia (Akiskal *et al*, 1980; Kocsis *et al*, 1985, 1988; Finlayson, 1989).

Controlled studies

Most placebo-controlled studies with antidepressants have lasted 6–12 weeks. In a six-week study in a general practice setting, Paykel *et al* (1982) found amitriptyline superior to placebo in mild depressives, but only in those satisfying RDC criteria for probable or definite major depression or in whom the initial 17-item Hamilton Scale score was 13 or more. Kocsis *et al* (1985, 1988) compared imipramine with placebo in 54 patients, many of whom presented with symptoms of double depression, and were of early-onset dysthymia; imipramine was clearly superior. Stewart *et al* (1988), in a similar six-week study, compared phenelzine, imipramine, and placebo, finding that the two active drugs were superior. Thus, TCAs appear to be of therapeutic benefit in dysthymia, particularly in patients with symptom levels approaching those of major depression; responders also tended to show improvement in social and vocational functioning.

So far as monoamine oxidase inhibitors (MAOIs) are concerned, phenelzine was the first antidepressant to be studied in depression akin to dysthymia, following reports of its therapeutic value in atypical depression. In depressed out-patients with a chronic 'characterological' pattern, phenelzine was superior to placebo but inferior to amitryptiline (Paykel *et al*, 1982), while Versiani & Nardi (in press) found tranylcypromine to be effective over at least two years. It seems likely that MAOIs are equivalent or possibly even superior to tricyclic agents for dysthymia, but this remains to be verified in controlled studies. Stabl *et al* (1989) reviewed studies comparing moclobemide, a tricyclic antidepressant, and placebo in various types of depression, concluding that RIMAs might be of benefit in dysthymia. Subsequently, a large multi-centre study (Versiani, 1992) demonstrated that moclobemide was significantly superior to placebo and of equivalent efficacy to imipramine; this applied equally to patients who had chronic dysthymia, with or without major depression.

The SSRI, fluoxetine was compared with placebo by Hellerstein *et al* (1993) in 35 patients with dysthymic disorder unaccompanied by major depression; after eight weeks, patients on the active medication showed significantly greater improvement. Studies by Arriaga *et al* (1986) and Reyntjens *et al* (1986) suggested that ritanserin, a 5-HT2 antagonist, may be superior to placebo in dysthymic disorder, while Bersani *et al* (1991) confirmed this at a dose of 10 mg daily. Bakish *et al* (in press) demonstrated the superiority of both ritanserin and imipramine over placebo. It thus seems likely that treatment through serotonin mechanisms may be an effective approach to dysthymia. Ravindran *et al* (1993) found that sub-affective dysthymics were more likely to respond to SSRIs, and that female gender, late onset, and a positive dexamethasone suppression test were also indicators of response.

In the case of 'double depression', recent studies (Kocsis *et al*, 1985; Kocsis & Frances, 1987) have shown that effective control of the major depression usually results in improvement of the dysthymic syndrome. Continuation of treatment therefore becomes an important consideration, particularly because patients with untreated dysthymia are at high risk for occurrence of MDD (Keller *et al*, 1992). When dysthymia is associated with either panic disorder (Bech *et al*, 1991) or eating disorders (Cummins, 1992), treatment of the primary condition by antidepressants may vicariously treat the dysthymic disorder.

There have been few attempts to identify dysthymic patients who may be more likely than others to respond to pharmacotherapy, although Akiskal *et al* (1980) suggested that the sub-affective type with positive sleep EEG and family history for depression is more responsive than those showing a *mélange* of abnormal personality or spectrum disorder. These and other characteristics of sub-affective dysthymia and character spectrum disorder are listed in Table 1.

Implications for practitioners

While antidepressants are currently the mainstay of treatment in dysthymic disorder, they should not be administered in isolation, but as part of a treatment strategy which includes interpersonal, marital, family, and work adjustment. Dysthymic patients may be more sensitive to the side-effects of tricyclics than those with more severe depression, but an appropriate benefit/discomfort ratio can only be

determined individually. The newer antidepressants, viz. SSRIs and RIMAs, so far appear to be well tolerated.

Psychotherapy

Adaptation and coping

There is good evidence of considerable social and interpersonal disability in dysthymia (De Lisio *et al*, 1986); whatever the initial circumstance, there will be major adverse effects on work, activities, and relationships as time goes on. Moreover, depression may itself provoke increased hostility, with important secondary effects (Weissman *et al*, 1971; Paykel & Weissman, 1973). Markowitz (1993) has pointed out the value of psychotherapy in the acquisition of social skills in the dysthymic, after the remission of symptoms through drug treatment. In such a chronic illness as dysthymia, provision of a repertoire of coping skills to enable better self-management of symptoms and problems is important, even in the absence of complete remission, and psychological approaches to treatment may provide these.

Interpersonal, marital and family therapies

Psychotherapy is a commonly used treatment for dysthymia in the USA, but due to few direct controlled trials, inferences have to be made from studies in other groups of depressives, and from some uncontrolled work in defined dysthymia. Non-specific psychotherapy (brief or longer term) needs to be distinguished from more focused approaches such as interpersonal psychotherapy (IPT) – tailored specifically to depression – and from marital therapy. Cognitive therapy (CBT) is a focused approach, with techniques directed specifically towards depressive cognition. The only controlled trial of psychotherapy specifically in dysthymia (Waring *et al*, 1988) compared doxepin, placebo, marital therapy, and minimal contact in 26 married women; both active treatments appeared to reduce symptoms, but marital therapy had a superior effect on marital intimacy.

Studies of the efficacy of briefer psychotherapies in mild to moderate depression in out-patients or general practice patients show that the effects are stronger than those of medication on social adjustment; symptoms are relieved to some extent, but effects in preventing relapse or recurrence are weakest. Several studies also tested the efficacy of medication, and of antidepressant–psychotherapy combinations: in all but one, medication produced stronger benefit on symptoms or relapse/recurrence, but most showed evidence of benefit from the combination and no studies showed adverse interactions. Most of these studies failed to examine effects in dysthymia separately, although patients with double depression are likely to have been included. Markowitz (1993) has adapted a form of IPT for dysthymia (Mason *et al*, in press).

Cognitive therapy

Cognitive–behavioural therapy (CBT) is the main behaviourally-orientated treatment approach which has been applied to depression, but there have been others, including Lewinsohn's reinforcement approach, aimed at increasing positive interactions and decreasing negative ones. Many studies on depression in general, which recruited relatively mild depressives, provide evidence of short-term efficacy of CBT on depressive symptoms, but one limitation is the small number comparing CBT with placebo or no treatment. Although cognitive therapy appears of comparable efficacy to antidepressants, this is probably limited to the more mildly depressed patients – who correspond more closely to those with dysthymia, at least in a symptomatological sense. Studies including combined treatment groups receiving both CBT and an antidepressant did not show greater benefit from the combined treatment than the better of the single treatments. However, given the absence of explicit trials in dysthymia, the generally poorer outcome in this disorder, and the uncertainty as to exactly which patients benefit from CBT, it would still be prudent to recommend combined treatment.

Some additional controlled trials, not in depressives, are also relevant to dysthymia. Linehan *et al* (1991) in a controlled trial in borderline patients, found that a modular cognitive–behavioural approach ('dialectical behaviour therapy'), compared with treatment as usual in the community, significantly reduced suicide attempts and in-patient days, although not feelings of depression. Suicidal attempts are an important problem in dysthymia: in a small sample of patients at high risk of repeat suicide attempts, Salkovskis *et al* (1990) found that a cognitive–behavioural problem-solving approach reduced both these and depressed feelings.

Some small or uncontrolled studies have indicated good results from CBT in dysthymic or similar samples. McCullough (1991) reported on ten patients treated with a treatment tailored to dysthymia – the 'cognitive–behavioural analysis' system, which combined cognitive elements with an interactional and interpersonal approach. Nine of the ten were in remission at two-year follow-up. Stravynski *et al* (1991) reported improvement with orthodox CBT, as did Mercier *et al* (1992) in six out of 15 dysthymics

or double depressives with atypical depression. De Jong *et al* (1986) reported a trend to better results in in-patients with combined major depression and dysthymia treated with a combined activity scheduling, social competence training, and a cognitive approach, compared with cognitive therapy alone or waiting list controls. Anecdotal accounts from cognitive therapists suggest better results in late-onset than early-onset dysthymia.

Recommendations

In the absence of firm research evidence, only tentative recommendations are possible. Marked social and interpersonal disability, high relapse rates of major depression, and a need for the acquisition of enduring coping skills to assist in self-management of symptoms and problems form potential indicators for psychosocial treatment. The evidence for benefit is best for IPT and marital therapy, including related social work; there is some evidence for family therapy, but no adequate evidence at present for group therapy. Evidence of efficacy of CBT in mild acute depression is good, and its ultimate role may turn out to be considerable. It can be used to facilitate control of symptoms and prevention of relapse, when drug therapy is only producing either a partial response or none at all. For dysthymia, psychosocial therapies should be used in combination with antidepressants, rather than alone.

Conclusions

A shift has occurred from the original view of dysthymia as a neurotic personality disorder to the current one of a chronic (sub)-affective disorder that often precedes major episodes of affective or anxiety illness; it may persist both during such major episodes (constituting double depression) and after their remission. The view that long-term cases are untreatable 'neurotic' patients with marked abnormalities of personality is now much less widely held.

If the association between dysthymic disorder and major depression can be more clearly defined, accurate diagnosis will more often lead to appropriate treatment. Although pharmacotherapy is the mainstay of treatment in dysthymic disorder, it should not be administered in isolation, but as part of a global treatment strategy.

The low-grade chronicity of this illness probably contributes most to the problem of under-treatment and misdiagnosis.

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