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## Dysthymic Disorder

### Forlorn and Overlooked?

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## Abstract

This ongoing column is dedicated to the challenging clinical interface between psychiatry and primary care—two fields that are inexorably linked.

Dysthymic disorder is a smoldering mood disturbance characterized by a long duration (at least two years in adults) as well as transient periods of normal mood. The disorder is fairly common in the US general population (3–6%) as well as in primary care (7%) and mental health settings (up to one-third of psychiatric outpatients). While the etiology of dysthymia remains unknown, there appears to be a genetic susceptibility, which may manifest in the presence of various psychosocial stressors. While the *Diagnostic and Statistical Manual of Mental Disorders* diagnostic criteria are fairly clear, the disorder can be easily under-recognized for a variety of reasons. Treatment may include pharmacotherapy and psychotherapy, although the overall treatment course is oftentimes characterized by protracted symptoms and relapses.

**Keywords:** dysthymia, dysthymic disorder, depression, mood disorder

## Introduction

In this edition of The Interface, we review the mood disorder, dysthymia. Dysthymia, or dysthymic disorder (DD), is a longstanding mood disorder that is characterized by fluctuating dysphoria that may be punctuated by brief periods of normal mood. Far less symptomatically dramatic than its cousin major depression, DD is fairly common in the community and in primary care and mental health

settings. While no consistent biological findings are evident, DD appears to have a genetic predisposition. In both psychiatric and primary care settings, DD can be difficult to detect. Treatment may include both pharmacotherapy and psychotherapy, although responses to either may be modest and/or short-lived. The course of DD may be lengthy and a number of prognostic factors are associated with poor outcome.

## Definition of the Disorder

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According to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)*,<sup>1</sup> DD is an Axis I mood disturbance distinguished by seemingly low-grade depressive symptoms as well as symptom persistence (i.e., at least two years in duration). Individuals with this disorder experience a depressed mood for most of the day, for more days than not, as well as at least two of the following diagnostic symptoms: (1) poor appetite or overeating; (2) insomnia or hypersomnia; (3) low energy or fatigue; (4) low self esteem; (5) poor concentration or difficulty making decisions; and (6) feelings of hopelessness. Afflicted individuals may experience fleeting periods of normal mood, but these cannot exceed two months. In addition, during the first two years following onset, there can be no discernable episodes of major depression.

## Prevalence of Dysthymia

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According to the 1988 Epidemiological Catchment Area Study, the prevalence of DD in the US general population is 3.1 percent<sup>2</sup> whereas data from the 1994 National Comorbidity Survey indicate a prevalence rate of 6.4 percent.<sup>3</sup> In a 2004 analysis of the literature, investigators determined a lifetime prevalence of DD in US communities of 3.6 percent.<sup>4</sup> These rates of DD appear comparable to those found in the Netherlands (4.6%).<sup>5</sup> To summarize, the lifetime prevalence rate of DD in US communities appears to be between 3 and 6 percent.

In US primary care settings, the prevalence of DD is somewhat higher than in community samples. For example, Howland reported rates from 1.3 to 31.9 percent, with a pooled prevalence rate of seven percent.<sup>6</sup> Spitzer and colleagues found that most US primary care settings harbored rates of DD between 5 and 15 percent.<sup>7</sup>

In comparison with US primary care settings, rates in international primary care settings seem to vary more. For example, Lecrubier and Weiller<sup>8</sup> reported that the point prevalence rate of DD in primary care settings of 14 countries was 2.1 percent. Baldwin<sup>9</sup> summarized the available international data and reported lifetime prevalence rates in foreign primary care settings between 3.7 and 20.6 percent. In a recent study among primary care patients in Spain, Aragonés et al<sup>10</sup> found a current prevalence rate of 4.8 percent.

As one might expect, the rates for DD are higher in psychiatric settings as well as among women compared with men.

## Etiology of Dysthymia

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No consistent or pervasive biological abnormality has been confirmed among patients with DD. This may relate to clinical and/or etiological heterogeneity, which is associated with this disorder. Sporadic abnormalities include polysomnographic sleep irregularities,<sup>1</sup> elevations in interleukin-1,<sup>11</sup>

serotonergic dysfunction,<sup>12</sup> and lower platelet monoamine oxidase activity in female patients.<sup>13</sup>

In dysthymic probands, family studies indicate higher rates of DD<sup>14</sup> as well as major depression and personality disorders.<sup>1</sup> This implies some degree of genetic susceptibility. In addition, a number of psychosocial factors may contribute to the disorder, such as stress in childhood and adulthood, and unfavorable social circumstances (e.g., isolation, lack of support).<sup>15</sup>

## Clinical Assessment

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**The DSM approach.** The *DSM-IV-TR* diagnostic criteria for DD were presented in the introduction of this article. The *DSM* also notes one clinically relevant specifier—a distinction between early versus late-onset DD, which is defined as symptom onset before or after age 21, respectively. In comparison with late-onset DD, early-onset dysthymia is characterized by higher relapse rates, more psychiatric hospitalizations, and a greater likelihood of comorbid major depression and personality disorders.<sup>16</sup>

**Comorbidity issues.** DD is characteristically associated with high frequencies of psychiatric comorbidity.<sup>17</sup> Indeed, “pure” dysthymia is so uncommon that the National Institutes of Mental Health Collaborative Study on the Psychobiology of Depression had to change recruitment strategies to obtain sufficient participants for study.<sup>18</sup> Common psychiatric comorbidities include major depression (up to 75%), anxiety disorders (up to 50%), personality disorders (20–40% or more among those with early-onset DD), somatoform disorders (2.8%–45.2%), and substance abuse (up to 50%).<sup>19,20</sup>

**Difficulties with the clinical detection of DD.** Given the preceding assessment guidelines for DD, there may be notable difficulties in detecting and diagnosing this disorder.

*Soft mood symptoms.* The fluctuating and/or seemingly modest nature of the symptoms<sup>21</sup> may lead to under-recognition by the patient as well as the clinician. Explicitly, the waxing and waning, smoldering course of these symptoms can be readily masked by patients in social situations, making it less likely for family and clinicians to detect the existence of underlying depression. In addition, compared with other types of psychiatric disorders, the symptoms of DD are relatively covert (e.g., concentration difficulties and low self esteem versus hallucinations in schizophrenia, compulsive behaviors in obsessive-compulsive disorder, or purging in bulimia nervosa). Likewise, because DD symptoms have varying amplitudes in different patients, individuals with mild symptoms may be easily overlooked.

*Distracting psychiatric comorbidity.* DD rarely exists in a pure form. Therefore, in the majority of cases, there will be comorbid psychiatric disorders that are competing for diagnostic attention. As we noted previously, these include various mood and anxiety disorders as well as personality, somatoform and substance use disorders—all of which are likely to present with more dramatic symptoms (e.g., major depression).

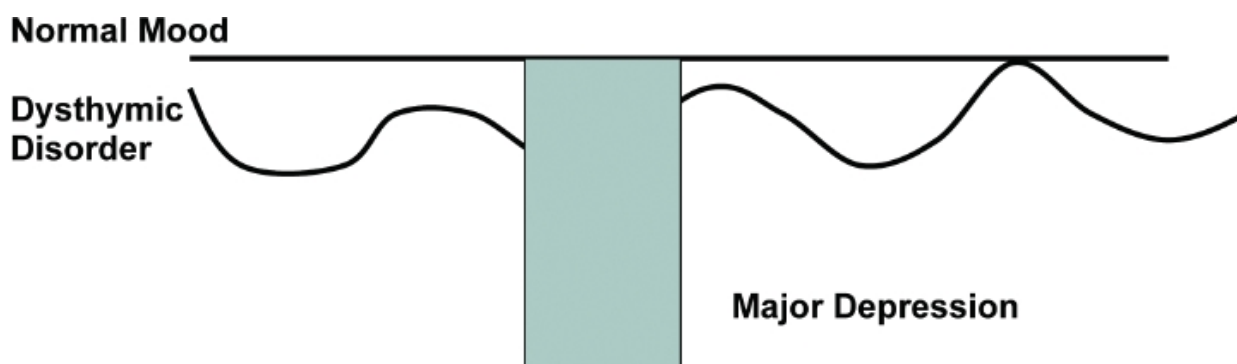
*Distracting somatic comorbidity.* In primary care settings, patients with DD oftentimes present with somatic comorbidity, particularly those with lower education and milder symptoms.<sup>22</sup> Somatic symptoms may over-shadow the underlying mood disorder.

*Lack of patient recognition in early-onset DD.* When the symptoms of DD have been longstanding (i.e., date back to childhood and/or adolescence), affected individuals may conclude that their malady are actually personality characteristics. In other words, they may not identify the mood disturbance as

separate from self.

*Misdiagnosis of symptoms.* Most primary care clinicians are fairly well-trained in the detection and diagnosis of major depression. This is likely because the symptoms of major depression are more dramatic and oftentimes anchored around identifiable alterations in neurovegetative rhythms. Therefore, when patients broach the subject of “depression,” many clinicians promptly cue to their mental templates for major depression, unintentionally overlooking the diagnosis of DD. This phenomenon may also occur in mental health settings.

**Discriminating depressive disorders: a simple approach.** All patients with depression need to be screened for DD. This can be easily accomplished by presenting the patient with a figure that compares and contrasts DD and major depression ([Figure 1](#)). In explaining the differences to patients, we emphasize that DD is characterized by an insidious onset, waxing/waning symptoms of at least two years duration, and possibly brief periods of normal mood. In contrast, major depression is characterized by a fairly well-defined onset, sustained symptoms, and discrete episodes. We have found that simultaneously discussing these syndromes and illustrating them with patients enables rapid determination of the offending syndrome.



[Figure 1](#)

Example of a figure that can be presented to patients to help illustrate the difference between dysthymia disorder and major depressive disorder

## Treatment

**Pharmacotherapy.** According to Dunner, “all treatments for depressive mood disorders are effective for dysthymia.”<sup>23</sup> Indeed, with regard to pharmacotherapy, most if not all studies confirm a degree of efficacy in the treatment of dysthymia, including those with newer antidepressants such as duloxetine.<sup>24</sup> However, despite statistically meaningful improvements in symptoms in short-term studies, overall responses tend to be modest.<sup>24–26</sup> This finding has resulted in the recommendation of sufficient drug-evaluation trials (i.e., 3 months) as well as possibly higher doses of antidepressants and the use of augmentation strategies.

While the duration of pharmacotherapy in the treatment of DD has not been established, from a clinic

perspective, ongoing or lifelong treatment seems likely in many cases. In addition, many individuals experience relapses and/or a loss of medication efficacy over time such that ongoing treatment is characterized by changes in antidepressants and/or adjustments in augmentation strategies.

**Psychotherapy.** In addition to pharmacotherapy, psychotherapy may be helpful. However, Dunner<sup>23</sup> cautions that “treatment with psychotherapy is difficult.” A number of psychotherapies have been advocated including cognitive behavioral analysis system of psychotherapy (CBASP),<sup>27</sup> interpersonal psychotherapy (IPT),<sup>28,29</sup> cognitive behavioral therapy,<sup>30</sup> manualized group therapy,<sup>31</sup> and problem-solving therapy.<sup>32</sup> These do not exclude the potential value of supportive or psychodynamic psychotherapies.

**Pharmacotherapy vs. Psychotherapy.** In comparing pharmacotherapy with psychotherapy, studies are mixed in their conclusions. However, the recent empirical trend appears to be in support of pharmacotherapy over psychotherapy.<sup>29,33</sup> This is not to exclude the possibility of individuals with dysthymia who respond well to psychotherapy or that better outcomes may be achieved with both types of interventions in some patients.

## Course and Prognosis

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Long-term outcome studies in the treatment of DD are few in number, and study comparisons are difficult because of varying methodologies. However, one theme continually emerges—a substantial proportion of sufferers do not experience a sustained recovery. For example, Klein et al<sup>34</sup> described the 10-year outcome of a cohort of dysthymic patients and findings included protracted symptoms and high relapse rates. Comorbid major depression, which nearly all DD patients experience, may increase the risk for poor outcome.<sup>35</sup> DD patients may also experience low quality of life, disability, and poor social support and marital adjustment.<sup>36</sup> In addition, these individuals seem to be more stress-responsive.<sup>37</sup>

A number of prognostic variables have been described in DD. Less favorable outcomes are associated with early-onset of symptoms;<sup>38</sup> history of sexual abuse, poor relationships with both parents, family histories of drug abuse, and Cluster A personality disorders;<sup>39</sup> comorbid anxiety disorders, Cluster C personality features, chronic stress, and eating disorders;<sup>40</sup> greater mood symptoms and Axis II disturbance;<sup>41</sup> older age, less education, concurrent anxiety disorder, a positive family history of depression, a poorer maternal relationship in childhood, and sexual abuse;<sup>42</sup> and poor medication adherence, lower self efficacy in managing depression, and histories of childhood trauma.<sup>43</sup> To summarize, the outcome for DD appears to be greatly diminished by comorbid psychopathology, history of trauma, poor early family relationships, and stress.

## Conclusions

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DD is a common and debilitating mood disorder that can easily escape clinical detection. When assessing depression, either in psychiatric or primary care settings, we encourage all clinicians to consider DD in the differential diagnosis. The differentiation between DD and major depression can be easily accomplished with patients by providing them simultaneously with a figure contrasting the two disorders and a verbal explanation. This disorder is treatable, although the results may be modest and short-lived, and the course troublesome. Clearly, DD is a forlorn mood disorder than is prone to being

overlooked in all clinical settings.

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## References

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1. American Psychiatric Association. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Press, Inc.; 2000. [[Google Scholar](#)]
2. Weissman MM, Leaf PJ, Bruce ML, Florio L. The epidemiology of dysthymia in five communities rates, risks, comorbidity, and treatment. *Am J Psychiatry*. 1988;145:815–819. [[PubMed](#)] [[Google Scholar](#)]
3. Kessler RC, McGonagle KA, Zhao S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States from the National Comorbidity Survey. *Arch Gen Psychiatry*. 1994;51:8–19. [[PubMed](#)] [[Google Scholar](#)]
4. Waraich P, Goldner EM, Somers JM, Hsu L. Prevalence and incidence studies of mood disorders: a systematic review of the literature. *Can J Psychiatry*. 2004;49:124–138. [[PubMed](#)] [[Google Scholar](#)]
5. Beekman AT, Deeg DJ, Smit JH, et al. Dysthymia in later life: a study in the community. *J Affect Disord*. 2004;81:191–199. [[PubMed](#)] [[Google Scholar](#)]
6. Howland RH. General health, health care utilization, and medical comorbidity in dysthymia. *Int J Psychiatry Med*. 1993;23:211–238. [[PubMed](#)] [[Google Scholar](#)]
7. Spitzer RL, Williams JB, Kroenke K, et al. Utility of a new procedure for diagnosing mental disorders in primary care. (The PRIME-MD 1000 study). *JAMA*. 1994;272:1749–1756. [[PubMed](#)] [[Google Scholar](#)]
8. Lecrubier Y, Weiller E. Characteristics, recognition and treatment of dysthymics in primary care. *Eur Psychiatry*. 1998;13:198–202. [[PubMed](#)] [[Google Scholar](#)]
9. Baldwin DS. Dysthymia: options in pharmacotherapy. In: Palmer KJ, editor. *Managing Depressive Disorders*. Philadelphia: Lippincott: Williams, & Wilkins; 2000. pp. 17–28. [[Google Scholar](#)]
10. Aragonés E, Pinol JL, Labad A, et al. Prevalence and determinants of depressive disorders in primary care practice in Spain. *Int J Psychiatry Med*. 2004;34:21–35. [[PubMed](#)] [[Google Scholar](#)]
11. Anisman H, Ravindran AV, Griffiths J, Merali Z. Interleukin-1 beta production in dysthymia before



and after pharmacotherapy. *Biol Psychiatry*. 1999;46:1649–1655. [[PubMed](#)] [[Google Scholar](#)]

12. Ravidran AV, Chudzik J, Bialik RJ, et al. Platelet serotonin measures in primary dysthymia. *Am J Psychiatry*. 1994;151:1369–1371. [[PubMed](#)] [[Google Scholar](#)]

13. Tripodianiakis J, Markianos M, Sarantidis D, et al. Platelet MAO activity in patients with dysthym disorder. *Psychiatry Res*. 1998;78:173–178. [[PubMed](#)] [[Google Scholar](#)]

14. Lizardi H, Klein DN. Parental psychopathology and reports of the childhood home environment in adults with early-onset dysthymic disorder. *J Nerv Ment Dis*. 2000;188:63–70. [[PubMed](#)] [[Google Scholar](#)]

15. Anonymous. Dysthymia: psychotherapists and patients confront the high cost of “low-grade” depression. *Harv Ment Health Lett*. 2005;21:1–3. [[PubMed](#)] [[Google Scholar](#)]

16. Arnow BA, Constantino MJ. Effectiveness of psychotherapy and combination treatment for chronic depression. *J Clin Psychol*. 2003;59:893–905. [[PubMed](#)] [[Google Scholar](#)]

17. Autonell J, Vila F, Pinto-Meza A, et al. One year prevalence of mental disorders comorbidity and associated sociodemographic risk factors in the general population of Spain. (Results of the ESEMeD Spain study). *Actas Esp Psiquiatr*. 2007;35:4–11. [[PubMed](#)] [[Google Scholar](#)]

18. Klein DN, Riso LP, Anderson RL. DSM-III-R dysthymia: antecedents and underlying assumption. *Prog Exp Pers Psychopathol Res*. 1993;16:222–253. [[PubMed](#)] [[Google Scholar](#)]

19. Sansone RA, Correll T. Dysthymia disorder: the persistent depression. *Hosp Physician Board Rev Man*. 2005;9:1–12. [[Google Scholar](#)]

20. Klein DN, Schwartz JE, Rose S, Leader JB. Five-year course and outcome of dysthymic disorder: prospective, naturalistic follow-up study. *Am J Psychiatry*. 2000;157:931–939. [[PubMed](#)] [[Google Scholar](#)]

21. Kessing LV. Epidemiology of subtypes of depression. *Acta Psychiatr Scand Suppl*. 2007;433:85–89. [[PubMed](#)] [[Google Scholar](#)]

22. Nuyen J, Volkers AC, Verhaak PE, et al. Accuracy of diagnosing depression in primary care: the impact of chronic somatic and psychiatric co-morbidity. *Psychol Med*. 2005;35:1185–1195. [[PubMed](#)] [[Google Scholar](#)]

23. Dunner DL. Dysthymia and double depression. *Int Rev Psychiatry*. 2005;17:3–8. [[PubMed](#)] [[Google Scholar](#)]

24. Koran LM, Aboujaoude EN, Gamel NN. Duloxetine treatment of dysthymia and double depression: an open-label trial. *J Clin Psychiatry*. 2007;68:761–765. [[PubMed](#)] [[Google Scholar](#)]

25. Howland RH. Pharmacotherapy of dysthymia: a review. *J Clin Psychopharmacol*. 1991;11:83–92. [[PubMed](#)] [[Google Scholar](#)]

26. Fawcett J. Antidepressants: partial response in chronic depression. *Br J Psychiatry Suppl*. 1994;26:37–41. [[PubMed](#)] [[Google Scholar](#)]

27. McCollough JP. Treatment for Chronic Depression: Cognitive Behavioral Analysis System of Psychotherapy. New York: Guildford; 2000. [[Google Scholar](#)]
28. Markowitz JC. Interpersonal Psychotherapy for Dysthymic Disorder. Washington, DC: American Psychiatric Press, Inc; 1998. [[Google Scholar](#)]
29. Markowitz JC. Interpersonal psychotherapy for chronic depression. J Clin Psychol. 2003;59:847–858. [[PubMed](#)] [[Google Scholar](#)]
30. McCullough JP. Psychotherapy for dysthymia. A naturalistic study of ten patients. J Nerv Ment Dis. 1991;179:734–740. [[PubMed](#)] [[Google Scholar](#)]
31. Hellerstein DJ, Little SA, Samstag LW, et al. Adding group psychotherapy to medication treatment in dysthymia: a randomized prospective pilot study. J Psychother Pract Res. 2001;10:93–103. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
32. Williams JW, Jr., Barrett J, Oxman T, et al. Treatment of dysthymia and minor depression in primary care: a randomized controlled trial in older adults. JAMA. 2000;284:1519–1526. [[PubMed](#)] [[Google Scholar](#)]
33. Markowitz JC, Kocsis JH, Bleiberg KL, et al. A comparative trial of psychotherapy and pharmacotherapy for “pure” dysthymic patients. J Affect Disord. 2005;89:167–175. [[PubMed](#)] [[Google Scholar](#)]
34. Klein DN, Shankman SA, Rose S. Ten-year prospective follow-up study of the naturalistic course of dysthymic disorder and double depression. Am J Psychiatry. 2006;163:872–880. [[PubMed](#)] [[Google Scholar](#)]
35. Hybels CF, Blazer DG, Steffens DC. Predictors of partial remission in older patients treated for major depression: the role of comorbid dysthymia. Am J Geriatr Psychiatry. 2005;13:713–721. [[PubMed](#)] [[Google Scholar](#)]
36. Subodh BN, Avasthi A, Chakrabarti S. Psychosocial impact of dysthymia: a study among married patients. J Affect Disord. 2008;109:199–204. [[PubMed](#)] [[Google Scholar](#)]
37. Rashed S, Kamel S, Hassan M, Mahfouz A. Psychometric study of dysthymic patients and their firstdegree relatives. J Egypt Public Health Assoc. 2001;76:89–105. [[PubMed](#)] [[Google Scholar](#)]
38. Flory V, Vance ALA, Birlson P, Luk ESL. Early-onset dysthymic disorder in children and adolescents: clinical implications and future directions. Child Adolesc Ment Health. 2002;7:79–84. [[Google Scholar](#)]
39. Durbin CE, Klein DN, Schwartz JE. Predicting the 2 1/2-year outcome of dysthymic disorder: the role of childhood adversity and family history of psychopathology. J Consult Clin Psychol. 2000;68:57–63. [[PubMed](#)] [[Google Scholar](#)]
40. Hayden EP, Klein DN. Outcome of dysthymic disorder at 5-year followup: the effect of familial psychopathology, early adversity, personality, comorbidity, and chronic stress. Am J Psychiatry. 2001;158:1864–1870. [[PubMed](#)] [[Google Scholar](#)]



41. Tyrer P, Seivewright H, Johnson T. The Nottingham Study of Neurotic Disorder: predictors of 12-year outcome of dysthymic, panic and generalized anxiety disorder. *Psychol Med*. 2004;34:1385–139. [[PubMed](#)] [[Google Scholar](#)]
42. Klein DN, Shankman SA, Rose S. Dysthymic disorder and double depression: prediction of 10-year course trajectories and outcomes. *J Psychiatr Res*. 2008;42:408–415. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
43. Gopinath S, Katon WJ, Russo JE, Ludman EJ. Clinical factors associated with relapse in primary care patients with chronic or recurrent depression. *J Affect Disord*. 2007;101:57–63. [[PubMed](#)] [[Google Scholar](#)]

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