Psychological Medicine, 1997, **27**, 989–994. Printed in the United Kingdom
© 1997 Cambridge University Press

EDITORIAL

Reviving the diagnosis of neurasthenia[1]

'Whether or not it is worthwhile to distinguish between "neurasthenia" and "dysthymic disorders" must depend either on the demonstration that such syndromes have different social covariates, or pursue a different course, or have particular responses to treatment. Until such studies are forthcoming, the distinction seems especially insubstantial.' (Goldberg & Bridges, 1991)

Epidemiological studies now indicate that fatigue is one of the most common symptoms of ill-health in the community, primary care and other medical settings, and that syndromal diagnoses based on fatigue (including chronic fatigue and neurasthenia) are prevalent and major sources of health care utilization. Such syndromes are characterized by a combination of persistent and disabling fatigue and neuropsychological and neuromuscular symptoms (Lloyd *et al*. 1990; Angst & Koch, 1991; Sharpe *et al*. 1991; Fukuda *et al*. 1994). Essentially, the differences between these syndromes reflect variations in duration criteria rather than symptom constructs. Specifically, the Centers for Disease Control (CDC) defines 'prolonged fatigue' as a syndrome of at least 1 month's duration, and chronic fatigue (including idiopathic and chronic fatigue syndrome – CFS) as a fatigue syndrome of at least 6 months duration (Fukuda *et al*. 1994). The ICD-10 classification system (World Health Organization, 1992) now includes a formal diagnosis of neurasthenia (F48.0) based on mental and physical fatigue of at least 3 months duration. Despite the current international and epidemiological interest in these disorders, DSM-IV has simply included them within the 'Undifferentiated Somatoform Disorders – 300.81' category (American Psychiatric Association, 1994).

Historically, the diagnosis of neurasthenia has had a chequered history, particularly in English-speaking countries (Wessely, 1990, 1994; Shorter, 1992). Sporadic attempts to revive the diagnosis have encountered cogent objections, based largely on the clear overlap with conventional anxiety and depressive disorders (Wessely & Powell, 1989; Goldberg & Bridges, 1991). The issue has often been further complicated by cross-cultural considerations, with neurasthenia in non-English speaking communities being equated with anxiety and/or depression in English-speaking countries (Kleinman, 1982). The recognition of formal fatigue syndromes by the CDC and ICD-10, however, has encouraged clinicians to investigate the degree of overlap between such disorders and the more commonly recognized psychiatric disorders.

COMMUNITY STUDIES

In a longitudinal study of 591 young Swiss adults, the prevalence of neurasthenia and other psychiatric syndromes was assessed repeatedly over a 10-year-period (Angst & Koch, 1991). Two classes of neurasthenia were identified: 'recurrent brief neurasthenia' (episodes of short duration, occurring at least monthly for 1 year); and 'extended neurasthenia' (episodes lasting at least 2 weeks). The 1 year prevalence ranged between 3·4% and 10·7%, with women being affected three times more commonly. Of the cases of neurasthenia, 79% were associated with a concurrent or successive diagnosis of depression or anxiety disorder (odds ratio 4·65). Further analyses, however, revealed that neurasthenia was a more stable diagnostic entity over time than anxiety or depression (Merikangas & Angst, 1994).

[1] Address for correspondence: Professor Ian Hickie, Academic Department of Psychiatry, St George Hospital & Community Health Service, 7 Chapel Street, Kogarah 2217, NSW, Australia

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press

In Great Britain, the prevalence of common psychological syndromes was recently examined by the Office of Population Censuses and Surveys (OPCS) (Mason & Wilkinson, 1996). This study used the Clinical Interview Schedule – Revised (CIS-R) to elicit 14 neurotic symptoms including fatigue and somatic symptoms, with ICD-10 being used for diagnoses. The most common neurotic symptom was fatigue (27%) and the most prevalent neurotic disorder was mixed anxiety and depressive disorder (7·7%).

In the USA, the National Co-morbidity Study (NCS) was designed specifically to examine patterns of psychological co-morbidity and associated health care utilization (Kessler *et al.* 1994). It used a modified version of the Composite International Diagnostic Instrument (CIDI) and DSM-III-R for diagnoses. Prior experience with the Diagnostic Instrument Schedule (DIS) during the course of the Epidemiologic Catchment Area (ECA) Survey (Regier *et al.* 1988) had, however, led American epidemiologists to conclude erroneously that somatic distress syndromes were rare (average 0·1%) and, hence, were not sought in the NCS. This unfortunate decision reflected a belief that the DIS/CIDI systems had included a useful methodology for the identification of somatic distress.

Although the ECA studies demonstrated that severe somatization (Briquet's hysteria) was extremely rare, re-analysis of that data suggested that less severe syndromes were indeed prevalent. Escobar *et al.* (1989) demonstrated that an abridged somatization construct resulted in lifetime prevalence estimates of 9% to 20%. Two-thirds of these patients also met criteria for another psychiatric disorder, and as a group they demonstrated high rates of health care utilization and disability. Swartz *et al.* (1986), using grade of membership analysis with the ECA data, showed that although a severe 'somatization' class was evident, so were a range of other somatic syndromes including gastrointestinal, cardiovascular and musculoskeletal disorders.

PRIMARY CARE STUDIES

In an Australian study of 1593 patients, 25% of subjects had a fatigue syndrome of at least 2 weeks duration (Hickie *et al.* 1996). Two-thirds of these patients were also classified as cases of psychological disorder by the General Health Questionnaire (GHQ), and GHQ and fatigue severity scores were moderately correlated ($r = 0{\cdot}51$). Fatigue syndromes were particularly associated with female gender, lower socio-economic status and fewer years of formal education. In the United Kingdom, the prevalence of fatigue and psychological morbidity was assessed via a postal survey of 15283 patients (Pawlikowska *et al.* 1994), with 18·3% reporting substantial fatigue of greater than 6 months duration. Women were more likely to report fatigue than men and the most commonly cited reasons for fatigue were psychosocial. Again, GHQ and fatigue severity scores were moderately correlated ($r = 0{\cdot}62$).

Kirmayer & Robbins (1991) used confirmatory factor analysis with somatic symptom data (collected using the DIS) from 698 Canadians attending family-medicine clinics to identify five latent constructs. These were labelled chronic fatigue, fibromyalgia, irritable bowel, somatic depression and somatic anxiety. They concluded that 'the pattern of symptom reporting … was better characterized by several distinct functional syndromes than by a single somatization disorder'. Latent class analyses of symptom data from clinic-based patients with chronic fatigue were similarly inconsistent with a single somatization dimension (Hickie *et al.* 1995*a*).

In designing the WHO multi-centre study of mental illness in general health care, Sartorius *et al.* (1993) recognized the importance of such non-specific somatic syndromes and so adapted the CIDI (CIDI-PHC) for use in primary care settings (unfortunately, this adaptation has not been included in the most recent CIDI (version 2.0) for general psychiatric epidemiology). The WHO study recorded a prevalence for neurasthenia of 5·4% (range 1·1–10.5%), with two-thirds of patients having co-morbid anxiety or depressive disorders (Üstün & Sartorius, 1995).

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press

CLASSIFICATION OF COMMON FORMS OF PSYCHIATRIC MORBIDITY

Given that epidemiological studies now demonstrate that 'neurasthenia' is common in most cultures, the question still arises as to whether it is useful as yet another diagnostic category. A single dimensional view of psychological and somatic 'caseness' may be most useful (Goldberg *et al.* 1987; Katon & Russo, 1992; Wessely *et al.* 1996). Within this view somatic and psychological symptoms simply increase as a function of severity, so that the most severe 'cases' will demonstrate an admixture of anxiety, depressive and somatic symptoms. This pattern is consistent with the data from most large community and primary care studies and the results of the NCS, where 14% of the US population accounted for over half of the lifetime diagnoses (Kessler *et al.* 1994). Within this single-dimensional model, the specific syndrome identified at any particular point is less relevant than the underlying vulnerability which operates across the lifespan. This model supports treatment options that aim to reduce long-term vulnerability (e.g. high trait anxiety) in addition to any short term reduction of specific symptoms.

A single dimensional model, however, may not adequately represent the variety of clinical syndromes, or important specific symptoms (e.g. phobic avoidance), identified by mental health professionals. Consequently, a two-axes model (anxiety–depression) has generally been preferred (Goldberg *et al.* 1987; Goldberg, 1996). Within the two-axes model, it has been argued that 'neurasthenia' simply represents the somatic features of anxiety and depression (Goldberg & Bridges, 1991). This model implies that all relevant patients will be captured by existing screening instruments for anxiety and depression. It is clear, however, that the GHQ does not capture all such cases (Farmer *et al.* 1996; Hickie *et al.* 1996) and the WHO primary care study confirms this issue across a range of language groups. Clinic-based studies of CFS similarly demonstrate that at least 25 to 50% of these patients will fail to meet criteria for lifetime psychiatric disorders (Kroenke *et al.* 1988; Wessely & Powell, 1989; Hickie *et al.* 1990).

The approach taken by WHO in the multicentre study, however, has been challenged. Goldberg (1996) asserts that the inclusion of a large number of somatic symptoms created a 'radically different symptom content', which was 'virtually certain to emerge as a separate dimension'. Indeed, such symptoms are likely to emerge as a separate dimension if: (*i*) they are common; and, (*ii*) they do occur somewhat independently of anxiety and depression. In the WHO study, somatic symptoms were modestly correlated with anxiety (0·53) and depression (0·48), though these correlations were somewhat smaller than that found between anxiety and depression alone (0·68) (Goldberg, 1996). Importantly, a third dimension of neurasthenic symptoms can also be detected with the GHQ alone in primary care attenders (i.e. without including additional somatic items – Goldberg *et al.* 1987). Further, the two dimension model presupposes that Goldberg's preferred anxiety–depression symptom content is the most appropriate universe of symptoms for general practice and community studies. For the advocates of neurasthenia (Angst & Koch, 1991; Sartorius *et al.* 1993), the studies described demonstrate the prevalence of these symptoms, their salience in general practice settings, and the tendency for a somatic dimension to be independent of the traditional axes of anxiety and depression. Indeed, the WHO study could be interpreted as demonstrating the redundancy of separate anxiety and depression axes, while supporting the utility of a separate somatic symptom dimension.

International efforts to educate primary care and other medical practitioners in the identification and treatment of common psychiatric disorders continue to advocate largely the two-dimensional model. Typically, such efforts (e.g. PRIME- MD, Spitzer *et al.* 1994) place great value on screening questions which capture the psychological constructs (e.g. depressed mood, feelings of hopelessness and feelings of panic) underpinning major depression and anxiety. Although they include reference to somatic symptoms, such systems encourage the practitioner to view patients as having discrete anxiety or depressive disorders with secondary somatic distress. While 'classical' anxiety and depressive disorders do present in general practice settings, more commonly the clinician will be assessing patients with an admixture of somatic and psychological symptoms, patients who are just below or close to the threshold for psychiatric 'caseness' and patients who do not fit readily into

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press

the dichotomous anxiety and depression categories (Kessler *et al.* 1994; Goldberg & Lecrubier, 1995; Mason & Wilkinson, 1996). This may create a major credibility problem for psychiatric education in primary care settings. If our diagnostic systems do not readily match the symptom profiles presented, practitioners will continue to underdiagnose psychological disorders (Kessler *et al.* 1994; Üstün *et al.* 1995; Mason & Wilkinson, 1996). Further, data drawn largely from clinic-based populations of 'pure' anxiety and depressive disorders will create unrealistic predictions as to the efficacy of conventional antidepressant and anti-anxiety treatments in general practice attenders.

VALIDITY OF NEURASTHENIA

Critics of the neurasthenia entity emphasize the degree of overlap with other non-psychotic disorders. This criticism, however, is equally applicable to the more commonly accepted disorders (e.g. major depression, generalized anxiety and panic disorder) that show large degrees of overlap cross-sectionally, longitudinally and within family history data. This obvious difficulty has led some authors to conclude that broader concepts such as the 'general neurotic syndrome' (Andrews, 1996) or 'depressive neurosis' (Merikangas *et al.* 1994) underpin a more valid nosology. Interestingly, neurasthenia in adolescence or early adulthood has been proposed as a risk factor to later bipolar disorder. In the Swiss study, 23·3% of cases of neurasthenia that overlapped with anxiety or depression reported hypomanic symptoms, while such symptoms were not reported at all by patients with 'pure' neurasthenia (Angst & Koch, 1991).

With regard to specific neurobiological markers, chronic fatigue and neurasthenia do not appear to be associated with either the characteristic sleep abnormalities (i.e. shortened REM latency) (Moldofsky, 1993) or hypothalamic–pituitary–adrenal (HPA) axis changes encountered in at least some depressive subtypes (Demitrack *et al.* 1991). With regard to HPA axis function, 'fatigue' patients actually appear to have an Addisonian-like picture of hypocortisolism, a situation closer to post-traumatic stress disorder (Demitrack *et al.* 1991). Patients with CFS have a wide range of minor and non-specific immunological changes (Lloyd *et al.* 1994), though of greater severity than that encountered in patients with depressive disorders (Lloyd *et al.* 1992). Importantly, patients with major depression are themselves quite heterogeneous with regard to these neuroendocrinological, immunological and sleep markers, suggesting the lack of a unifying neurobiology for depressed mood (Hickie *et al.* 1990, 1993; Maes *et al.* 1990, 1994; Kupfer & Reynolds, 1992; Hickie, 1996).

TREATMENT OPTIONS FOR NEURASTHENIA

For the practising clinician, diagnostic categories are sterile unless they have treatment implications. Despite the enthusiasm for diagnosing affective disorders in patients with CFS (Kroenke *et al.* 1988; Manu *et al.* 1989; Wessely & Powell, 1989), to date selective serotonin reuptake inhibitors (SSRIs) have not been useful in treating CFS (Hickie & Wilson, 1994; Vercoulen *et al.* 1996), or the closely related syndrome of fibromyalgia (Wolfe *et al.* 1994), while reversible inhibitors of monoamine oxidase (RIMAs) may be of some therapeutic value (Hickie & Wilson, 1994; Wilson *et al.* 1994*a*). Consistent with the notion of atypical depression, low dose phenelzine has been of some benefit (Natelson *et al.* 1996).

By contrast, psychological and behavioural interventions designed to reduce somatic distress and improve physical functioning are clearly relevant (Wilson *et al.* 1994*b*; Hickie *et al.* 1995*b*; McCully *et al.* 1996). A series of cognitive–behavioural therapies (CBT) for patients with chronic fatigue syndrome have now been trialed (Butler *et al.* 1991; Lloyd *et al.* 1993, Hickie *et al.* 1995*c*; Sharpe *et al.* 1996; Deale *et al.* 1997). These programmes draw heavily on the chronic pain rather than affective disorder literature, although all emphasize concurrent pharmacotherapy for overt depressive disorders. Long-term improvements in disability and fatigue were noted in patients receiving CBT as compared with relaxation training (Deale *et al.* 1997), or standard medical care

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press

(Sharpe *et al*. 1996). Differences in the CBT trial results appear to reflect largely differences in the style of control treatments utilized, the duration and intensity of treatments provided, and the balance between cognitive and behavioural strategies employed. Behavioural therapies may be necessary to reduce disability initially, while cognitive approaches may contribute to sustained and long-term improvement. The complimentary fibromyalgia literature also demonstrates the efficacy of adjunctive agents such as non-steroidal anti-inflammatory agents and low dose tricyclic antidepressants (Goldenberg *et al*. 1986). The available studies now suggest the importance of long-term continuous and appropriate medical care, with the judicious use of pharmacological, cognitive–behavioural, sleep hygiene and physical rehabilitation strategies (Hickie *et al*. 1995*b*). Ongoing research in this field will result in clarification of these issues.

CONCLUSION

Recent epidemiological studies clearly establish the prevalence of neurasthenia. Current statistical, longitudinal, neurobiological and treatment response data suggest that 'neurasthenia/chronic fatigue' may be at least as robust a diagnostic category as the common anxiety and depressive disorders and requiring specifically designed pharmacological and non-pharmacological treatment interventions.

I. HICKIE, D. HADZI-PAVLOVIC AND C. RICCI

REFERENCES

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th edn). American Psychiatric Association: Washington, DC.

Andrews, G. (1996). Comorbidity and the general neurotic syndrome. *British Journal of Psychiatry* **168** (suppl. 30), 76–84.

Angst, J. & Koch, R. (1991) Neurasthenia in young adults. In *Problems of Psychiatry in General Practice* (ed. M. Gastpar and P. Kielholz), pp. 37–48. Hogrefe & Huber Publishers: Lewiston, NY.

Butler, S., Chalder, T., Ron, M. & Wessely, S. (1991). Cognitive behaviour therapy in chronic fatigue syndrome. *Journal of Neurology, Neurosurgery and Psychiatry* **54**, 153–158.

Deale , A., Chalder, T., Marks, I. & Wessely, S. (1997). Cognitive behaviour therapy for chronic fatigue syndrome: a randomised controlled trial. *American Journal of Psychiatry* (in the press).

Demitrack, M. A., Dale, J. K., Straus, S. E., Laue, L., Listwak, S. J., Kruesi, M. J. P., Chrousos, G. P. & Gold, P. W. (1991). Evidence for impaired activation of the hypothalamic–pituitary–adrenal axis in patients with chronic fatigue syndrome. *Journal of Clinical Endocrinology and Metabolism* **73**, 1224–1234.

Escobar, J. I., Rubio-Stipec, M., Canino, G. & Karno, M. (1989). Somatic Symptom Index (SSI): a new and abridged somatization construct: prevalence and epidemiological correlates in two large community samples. *Journal of Nervous and Mental Disease* **177**, 140–146.

Farmer, A., Chubb, H., Jones, I., Hillier, J., Smith, A. & Borysiewicz, L. (1996). Screening for psychiatric morbidity in subjects presenting with chronic fatigue syndrome. *British Journal of Psychiatry* **168**, 354–358.

Fukuda, K., Straus, S. E., Hickie, I., Sharpe, M. C., Dobbins, J. G., Komaroff, A. (1994). The chronic fatigue syndrome: a comprehensive approach to its definition and study. *Annals of Internal Medicine* **121**, 953–959.

Goldberg, D. (1996). A dimensional model for common mental disorders. *British Journal of Psychiatry* **168** (suppl. 30.), 44–49.

Goldberg, D. & Bridges, K. (1991). Minor psychiatric disorders and neurasthenia in general practice. In *Problems of Psychiatry in General Practice* (ed. M. Gastpar and P. Kielholz), pp. 79–88. Hogrefe & Huber Publishers: Lewiston, NY.

Goldberg, D. P. & Lecrubier, Y. (1995). Form and frequency of mental disorders across centres. In *Mental Illness in General Health Care: An International Study* (ed. T. B. Üstün and N. Sartorius), pp. 323–334. John Wiley & Sons: Chichester.

Goldberg, D. P., Bridges, K., Duncan-Jones, P. & Grayson, D. (1987). Dimensions of neuroses seen in primary care settings. *Psychological Medicine* **17**, 461–470.

Goldenberg, D. L., Felson, D. T. & Dinerman, H. (1986). A randomized controlled trial of amitriptyline and naproxen in the treatment of patients with fibromyalgia. *Arthritis and Rheumatism* **29**, 1371–1377.

Hickie, I. (1996). Issues in classification. III. Utilising behavioural constructs in melancholia research. In *Melancholia: A Disorder of Movement and Mood* (ed. G. Parker and D. Hadzi-Pavlovic), pp. 38–56. Cambridge University Press: New York.

Hickie, I. & Wilson, A. (1994). A catecholamine model of fatigue. *British Journal of Psychiatry* **165**, 275–276.

Hickie, I., Lloyd, A., Wakefield, D. & Parker, G. (1990). The psychiatric status of patients with chronic fatigue syndrome. *British Journal of Psychiatry* **156**, 534–540.

Hickie, I., Hickie, C., Lloyd, A, Silove, D. & Wakefield, D. (1993). Impaired in vivo immune responses in patients with melancholia. *British Journal of Psychiatry* **162**, 651–657.

Hickie, I., Lloyd, A., Hadzi-Pavlovic, D., Parker, G., Bird, K. & Wakefield, D. (1995*a*). Can the chronic fatigue syndrome be defined by distinct clinical features? *Psychological Medicine* **25**, 925–935.

Hickie, I., Lloyd, A. R. & Wakefield, D. (1995*b*). Chronic fatigue syndrome: current perspectives on evaluation and management. *Medical Journal of Australia* **163**, 314–318.

Hickie, I., Wilson, A., Hickie, C. & Lloyd, A. (1995*c*). Cognitive behaviour therapy for chronic fatigue syndrome – reply. *American Journal of Medicine* **98**, 419–422.

Hickie, I. B., Hooker, A. W., Hadzi-Pavlovic, D., Bennet, B. K., Wilson, A. J. & Lloyd, A. R. (1996). Fatigue in selected primary care settings: sociodemographic and psychiatric correlates. *Medical Journal of Australia* **164**, 585–588.

Katon, W. & Russo, J. (1992). Chronic fatigue syndrome criteria: a critique of the requirement for multiple physical complaints. *Archives of Internal Medicine* **152**, 1604–1609.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H. & Kendler, K. S. (1994). Lifetime

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press

and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: results form the National Comorbidity Survey. *Archives of General Psychiatry* **51**, 8–19.

Kirmayer, L. J. & Robbins, J. M. (1991). Functional somatic syndromes. In *Current Concepts of Somatization: Research and Clinical Perspectives* (ed. L. J. Kirmayer and J. M. Robbins), pp. 79–106. American Psychiatric Press, Inc.: Washington, DC.

Kleinman, A. (1982). Neurasthenia and depression. A study of somatization and culture in China. *Culture, Medicine and Psychiatry* **6**, 117–190.

Kroenke, K., Wood, D. R., Mangelsdorff, D., Meier, N. J. & Powell, J. B. (1988). Chronic fatigue in primary care. *Journal of the American Medical Association* **260**, 929–934.

Kupfer, D. J. & Reynolds, C. (1992). Sleep and affective disorders. In *Handbook of Affective Disorders* (2nd edn) (ed. E. S. Paykel), pp. 311–323. Churchill Livingstone: Edinburgh.

Lloyd, A. R., Hickie, I., Boughton, C. R., Spencer, O. & Wakefield, D. (1990). Prevalence of chronic fatigue syndrome in an Australian population. *Medical Journal of Australia* **153**, 522–528.

Lloyd, A. R., Hickie, I., Hickie, C. & Wakefield, D. (1992). Cell-mediated immunity in patients with chronic fatigue syndrome, healthy control subjects and patients with major depression. *Clinical and Experimental Immunology* **87**, 76–79.

Lloyd, A. R., Hickie, I., Brockman, A., Hickie, C., Wilson, A., Dwyer, J. & Wakefield, D. (1993). Immunologic and psychologic therapy for patients with chronic fatigue syndrome: a double-blind, placebo-controlled trial. *American Journal of Medicine* **94**, 197–203.

Lloyd, A. R., Hickie, I. & Wakefield, D. (1994). Studies of the pathophysiology of chronic fatigue syndrome in Australia. In *Chronic Fatigue Syndrome* (ed. S. Straus), pp. 353–384. Marcel Dekker: New York.

McCully, K. K., Sisto, S. A. & Natelson, B. H. (1996). Use of exercise for treatment of chronic fatigue syndrome. *Sports Medicine* **21**, 35–48.

Maes, M., Maes, L., Schotte, C., Vandewoude, M., Martin, M., D'Hondt, P., Blockx, P., Sharpe, S. & Cosyns, P. (1990). Clinical subtypes of unipolar depression: Part III. Quantitative differences in various biological markers between the cluster-analytically generated nonvital and vital depression classes. *Psychiatry Research* **34**, 59–75.

Maes, M., Calabrese, J. & Meltzer, H. Y. (1994). The relevance of the in- versus outpatient status for studies on HPA-axis in depression: spontaneous hypercortisolism is a feature of major depressed inpatients and not of major depression *per se*. *Progress in Neuropsychopharmacology and Biological Psychiatry* **18**, 503–517.

Manu, P., Matthews, D. A., Lane, T. J., Tennen, H., Hesselbroch, V., Mendola, R. & Affleck, G. (1989). Depression among patients with a primary complaint of chronic fatigue. *Journal of Affective Disorders* **17**, 165–172.

Mason, P. & Wilkinson G. (1996). The prevalence of psychiatric morbidity: OPCS survey of psychiatric morbidity in Great Britain. *British Journal of Psychiatry* **168**, 1–3.

Merikangas, K. & Angst, J. (1994). Neurasthenia in a longitudinal cohort study of young adults. *Psychological Medicine* **24**, 1013–1024.

Merikangas, K. R., Risch, N. J. & Weissman, M. M. (1994). Co-morbidity and co-transmission of alcoholism, anxiety and depression. *Psychological Medicine* **24**, 69–80.

Moldofsky, H. (1993). Fibromyalgia, sleep disorder and chronic fatigue syndrome. In *Chronic Fatigue Syndrome* (ed. G. Bock and J. Whelan), pp. 262–271. (Ciba Foundation Symposium 1973). John Wiley & Sons: New York.

Natelson, B. H., Cheu, J., Pareja, J., Ellis, S. P., Policastro, T. & Findley, T. W. (1996). Randomized, double blind, controlled placebo-phase in trial of low dose phenelzine in the chronic fatigue syndrome. *Psychopharmacology* **124**, 226–230.

Pawlikowska, T., Chalder, T., Hirsch, S. R., Wallace, P., Wright, D. J. M. & Wessely, S. C. (1994). Population based study of fatigue and psychological distress. *British Medical Journal* **308**, 763–766.

Regier, D. A., Boyd, J. H., Burke, J. D., Rae, D. S., Myers, J. K., Kramer, M., Robins, L. N., George, L. K., Karno, M. & Locke, B. Z. (1988). One-month prevalence of mental disorders in the United States. *Archives of General Psychiatry* **45**, 977–986.

Sartorius, N., Üstün, T. B., Costa e Silva, J. A., Goldberg, D., Lecrubier, Y., Ormel, J., Von Korff, M. & Wittchen, H. U. (1993). An international study of psychological problems in primary care. *Archives of General Psychiatry* **50**, 1069–1077.

Sharpe, M. C., Archard, L. C., Banatvala, J. E., Borysiewicz, L. K., Clare, A. W., David, A., Edwards, A. H. T., Hawton, K. E. H., Lambert, H. P., Lane, R. J. M., McDonald, E. M., Mowbray, J. F., Pearson, D. J., Peto, T. E. A., Preedy, V. R., Smith, A. P., Smith, D. G., Taylor, D. J., Tyrrell, D. A. J., Wessely, S., White, P. D., Behan. P. O., Rose, F. C., Peters, T. J., Wallace, P. G., Warrell, D. A. & Wright, D. J. M. (1991). A report – chronic fatigue syndrome: guidelines for research. *Journal of the Royal Society of Medicine* **84**, 118–121.

Sharpe, M., Hawton, K., Simkin, S., Surawy, C., Hackmann, A., Klimes, I., Peto, T., Warrell, D. & Seagroatt, V. (1996). Cognitive behaviour therapy for the chronic fatigue syndrome: a randomized controlled trial. *British Medical Journal* **312**, 22–26.

Shorter, E. (1992). *From Paralysis to Fatigue: A History of Psychosomatic Illness in the Modern Era*. Free Press: New York.

Spitzer, R. L., Williams, J. B. W., Kroenke, K., Linzer, M., deGruy, F. V., Hahn, S. R., Brody, D. and Johnson, J. G. (1994). Utility of a new procedure for diagnosing mental disorders in primary care – the PRIME-MD 1000 study. *Journal of the American Medical Association* **272**, 1749–1756.

Swartz, M., Blazer, D., Woodbury, M., George, L. & Landerman, R. (1986). Somatization disorder in a US Southern community: use of a new procedure for analysis of medical classification. *Psychological Medicine* **16**, 595–609.

Üstün, T. B. & Sartorius, N. (eds.) (1995). *Mental Illness in General Health Care: An International Study*. John Wiley & Sons: Chichester.

Üstün, T. B., Sartorius, N., Costa e Silva, J. A., Goldberg, D. P., Lecrubier, Y., Ormel, J., von Korff, M. & Wittchen, H.-U. (1995). Conclusions. In *Mental Illness in General Health Care: An International Study* (ed. T. B. Üstün and N. Sartorius), pp. 371–375. John Wiley & Sons: Chichester.

Vercoulen, J. H. M. M., Swanink, C. M. A., Zitman, F. G., Vreden, S. G. S., Hoofs, M. P. E., Fennis, J. F. M., Galama, J. M. D., van der Meer, J. W. M. & Bleijenberg, G. (1996). Randomised, double-blind, placebo-controlled study of fluoxetine in chronic fatigue syndrome. *Lancet* **347**, 858–861.

Wessely, S. (1990). Old wine in new bottles: neurasthenia and 'ME'. *Psychological Medicine* **20**, 35–53.

Wessely, S. (1994). Neurasthenia and chronic fatigue: theory and practice in Britain and America. *Transcultural Psychiatric Research Review* **31**, 173–209.

Wessely, S. & Powell, R. (1989). Fatigue syndromes: a comparison of chronic postviral fatigue with neuromuscular and affective disorders. *Journal of Neurology, Neurosurgery and Psychiatry* **52**, 940–948.

Wessely, S., Chalder, T., Girsch, S., Wallace, P. & Wright, D. (1996). Psychological symptoms, somatic symptoms, and psychiatric disorder in chronic fatigue and chronic fatigue syndrome: a prospective study in the primary care setting. *American Journal of Psychiatry* **153**, 1050–1059.

Wilson, A., Hickie, I., Wright, M., Bennett, B. & Wakefield, D. (1994*a*). Moclobemide in chronic fatigue syndrome: a double blind, placebo-controlled trial. *Neuropsychopharmacology* **10** (No. 3S), 245.

Wilson, A., Hickie, I., Lloyd, A. & Wakefield, D. (1994*b*). The treatment of chronic fatigue syndrome: science and speculation. *American Journal of Medicine* **96**, 544–550.

Wolfe, F., Cathey, M. A. & Hawley, D. J. (1994). A double-blind placebo controlled trial of fluoxetine in fibromyalgia. *Scandinavian Journal of Rheumatology* **23**, 255–259

World Health Organization. (1992). *The Tenth Revision of the International Classification of Diseases and Related Health Problems* (ICD-10). WHO: Geneva.

https://doi.org/10.1017/S0033291797004893 Published online by Cambridge University Press