Accuracy of diagnosing depression in primary care: the impact of chronic somatic and psychiatric co-morbidity

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ABSTRACT

Background. Depression is highly co-morbid with both psychiatric and chronic somatic disease. These types of co-morbidity have been shown to exert opposite effects on underdiagnosis of depression by general practitioners (GPs). However, past research has not addressed their combined effect on underdiagnosis of depression.

Method. Co-morbidity data on 191 depressed primary-care patients selected by a two-stage sampling procedure were analysed. Diagnoses of major depression and/or dysthymia in the last 12 months were assessed using a standardized psychiatric interview (CIDI) and compared with depression diagnoses registered by GPs in patient contacts during the same period. Presence of psychiatric and chronic somatic co-morbidity was determined using the CIDI and contact registration, respectively.

Results. Regression analysis showed a significant interaction effect between psychiatric and chronic somatic co-morbidity on GPs’ diagnosis of depression, while taking into account the effects of sociodemographic variables, depression severity and number of GP contacts. Subsequent stratified analysis revealed that in patients without chronic somatic co-morbidity, a lower educational level, a less severe depression, and fewer GP contacts all significantly increased the likelihood of not being diagnosed as depressed. In contrast, in patients with chronic somatic co-morbidity, only having no psychiatric co-morbidity significantly decreased the likelihood of receiving a depression diagnosis.

Conclusions. Our results indicate that the effects of psychiatric co-morbidity and other factors on underdiagnosis of depression by GPs differ between depressed patients with and without chronic somatic co-morbidity. Efforts to improve depression diagnosis by GPs seem to require different strategies for depressed patients with and without chronic somatic co-morbidity.

INTRODUCTION

Major depressive disorder (MDD) is a common disorder associated with significant disability, poorer quality of life, increased morbidity and mortality, and increased use of health services (Cassano & Fava, 2002; Papakostas et al. 2004). MDD does not occur frequently in pure form, but is often co-morbid with other psychiatric disorders, in particular anxiety disorders (Kessler et al. 1996; Ravelli et al. 1998), and a wide range of long-term medical conditions, including endocrine, neurological, cardiac, digestive, and respiratory disorders, cancer, diabetes
mellitus, arthritis, hypertension, and acquired immunodeficiency syndrome (Moldin et al. 1993; Wittchen et al. 1999; Gagnon & Patten, 2002).

Most depressive disorders are managed exclusively by general practitioners (GPs) (Ormel & Tiemens, 1997). However, despite being one of the most prevalent disorders in primary care (Cassano & Fava, 2002), depression is poorly recognized and diagnosed by GPs, with most studies reporting a rate of underdiagnosis falling in the 60–70% range (Bensing & Verhaak, 1994; Docherty, 1997). Although the clinical significance of this underdiagnosis has been disputed (Ormel & Tiemens, 1997), several lines of research underline that it is worthwhile trying to improve the diagnosis of depression in primary care. First, a substantial proportion of undetected depressed patients have persistently poor outcomes over the course of one year (Goldberg et al. 1998; Rost et al. 1998). Secondly, there is evidence for the efficacy of both pharmacological and psychotherapeutic interventions for the treatment of MDD in primary care (Mulrow et al. 2000; Schulberg et al. 2002). The third and most important piece of evidence is provided by recent systematic reviews of primary-care trials, which have concluded that, compared with usual care, quality improvement efforts aimed at improving detection of depression can improve patients’ outcomes provided that those recognized receive adequate treatment and follow-up (Pignone et al. 2002; Bijl et al. 2004). To improve the detection rate of depression by GPs, it is important to have detailed knowledge about the barriers to the diagnosis of depression. In this respect the impact of the frequent presence of co-morbidity on underdiagnosis of depression should be more thoroughly examined.

Previous studies have shown that patients with MDD have a higher risk of being not diagnosed as depressed by GPs if they have additional somatic illness(es) (Freeling et al. 1985; Coulehan et al. 1990; Tylee et al. 1993, 1995; Sartorius et al. 1996). In contrast, the co-occurrence of MDD and anxiety has been found to facilitate recognition of depression (Coyne et al. 1995) or psychiatric caseness (Ormel et al. 1990; Sartorius et al. 1996; Pini et al. 1997, 1999). However, these studies examined the effects of psychiatric and somatic co-morbidity on underdiagnosis of depression separately, and did not address their combined effect at all. Because a substantial proportion of depressed primary-care patients are expected to have both somatic as well as psychiatric co-morbidity (Wittchen et al. 1999; Maier & Falkai, 1999) it is important to examine their combined effect, in particular in the context of their suggested opposite effects on underdiagnosis of depression. The current study examines, therefore, whether there is an interaction effect between psychiatric and chronic somatic co-morbidity on GPs’ diagnosis of depression, while accounting for the effects of factors that are frequently reported to be associated with depression diagnosis (i.e. socio-demographic factors, severity of depression, and number of contacts with GP). If so, then the effects of psychiatric co-morbidity and the other factors will be studied in subgroups of depressed patients with and without chronic somatic co-morbidity. The results could lead to a more precise identification of barriers to the diagnosis of depression and thereby contribute to improved quality of GP care and outcomes of MDD in primary care.

METHOD

Study setting

Data collection took place within the framework of the second Dutch National Survey of General Practice (2001; DNSGP-2) (Westert et al. 2005), a nationwide study of morbidity and interventions in general practice in The Netherlands. The DNSGP-2 was carried out in 104 practices comprising 195 GPs, who served approximately 390,000 persons. The participating GPs were representative of all GPs in The Netherlands regarding age, gender, region of residence and urbanization. Dutch GPs are gatekeepers for secondary health care and nearly all non-institutionalized persons are listed to a GP. Three datasets were used, that is data from (a) a health interview survey, (b) a standardized psychiatric interview, and (c) a contact registration.

Participants

A random sample of the practice population \(n=12699\) participated in an extensive health interview survey (response rate = 64.5%), spread over a whole year to avoid seasonal patterns. In total 1379 patients aged 18 and
older had an indication of psychopathology as measured by two screening instruments included in the health interview (see below). These patients were approached for follow-up psychiatric assessment using the Composite International Diagnostic Interview (CIDI) (WHO, 1997), and 58.8% \((n = 811)\) were actually assessed. CIDI data on 235 patients were not examined in this study because: (1) the data were either incomplete \((n = 11)\) or obtained by using erroneously an earlier version of the CIDI; (2) patients had no contact with their GP within the time frame covered by the CIDI \((n = 214)\). The remaining 576 patients did not differ significantly from the eligible patients who were not examined \((n = 803)\) regarding age, educational level, and scores on the 12-item General Health Questionnaire (GHQ-12), and CAGE questionnaire, though a higher proportion was female \((p < 0.01)\). Of the 576 patients those who fulfilled the DSM-IV criteria for MDD and/or dysthymia according to CIDI \((n = 191)\) were included in the final study population.

Measures

The Dutch version of the GHQ-12 was administered as a screener for non-psychotic psychiatric morbidity (Koeter & Ormel, 1991). Scores of \(\geq 4\) (first half of 2001) or \(\geq 3\) (second half of 2001) were used as thresholds for follow-up psychiatric assessment. The cut-off score was lowered to enlarge the group of eligible patients. A three/four threshold has a high sensitivity (84.6%) and specificity (89.3%) in a primary-care setting (Goldberg et al. 1997). Additionally, the Dutch version of the CAGE questionnaire was used to identify patients with alcohol problems (Ewing, 1984). The maximum score of four positive answers was used as a threshold for further psychiatric assessment. Although this criterion is highly specific for detecting alcohol abuse/dependence in primary-care patients, it has a low sensitivity (23%; Aertgeerts et al. 2004). However, since this study examined patients with MDD who were already detected by the highly sensitive GHQ-12, falsely low prevalence rates of alcohol-related disorders due to this low sensitivity were avoided.

The CIDI is a fully structured interview that allows administration by trained lay interviewers. The Dutch version of the computerized CIDI-auto 2.1 was used (Ter Smitten et al. 1998). The fully specified structure does not allow judgement of the interviewer to intervene. Standardized probe questions establish that psychiatric symptoms are clinically significant and not due to medication, drugs or alcohol, or to a physical illness or injury. After completion of the interview, computerized algorithms provide diagnoses according to the DSM-IV. The presence of the following psychiatric disorders in the past 12 months was determined: phobic and other anxiety disorders, depressive disorders and dysthymic disorder, manic and bipolar affective disorder, and disorders resulting from the use of alcohol. During the interview, respondents are asked about the first and last occurrence of psychiatric symptoms, on the basis of which the period during which a psychiatric disorder was present was estimated for each patient.

During one year, all GPs electronically recorded each diagnosis made during their contacts with a patient, coded according to the International Classification of Primary Care (ICPC) (Lamberts & Wood, 1987). Contacts belonging to the same health problem were clustered into disease episodes.

Definitions

DSM-IV diagnoses obtained from the CIDI were considered as the reference standard. Patients were regarded as depressed in case of a diagnosis of MDD and/or dysthymia (hereafter referred to as ‘depressed patients’). For all patients the diagnosis was considered positive when all diagnostic criteria (inclusion as well as exclusion criteria) were fulfilled, with the exception of two patients with dysthymic disorder for whom only inclusion criteria were met.

A GP diagnosis of depression could be coded under depression (ICPC code P76), which is based on the criteria of the International Classification of Health Problems in Primary Care (WONCA, 1983). These criteria are largely consistent with those of DSM-IV for MDD. In addition, depressive symptoms could also be coded under depressive feelings (ICPC code P03). Therefore, patients having an episode P76
and/or P03 were considered to be diagnosed as depressed by their GP, while patients without such episodes were not.

The presence of psychiatric co-morbidity was determined on the basis of the CIDI data and was defined as having at least one CIDI diagnosis other than MDD/dysthymia. Psychiatric co-morbidity had to be present during at least one GP–patient contact. The following three categories of psychiatric co-morbidity were formed: alcohol abuse/dependence, bipolar disorder, and anxiety disorder, including the five subcategories panic disorder, agoraphobia (without panic), social phobia, generalized anxiety disorder (GAD), and simple phobia.

The presence of chronic somatic co-morbidity was determined on the basis of ICPC-coded diagnoses of chronic conditions recorded by GPs and defined as having at least one episode of chronic disease. It was ascertained that chronic somatic co-morbidity was present during the period of MDD and/or dysthymia. The following eight categories of chronic somatic co-morbidity were created, based on the body systems involved: neurological conditions (migraine or regular serious headache, dizziness, Parkinson’s disease, multiple sclerosis, epilepsy); musculoskeletal conditions (chronic rheumatism, rheumatic complaints of hips and knees, serious or persistent neck/shoulder, back, and hands/elbow/wrist disorder); circulatory conditions (hypertension, vascular disorder, myocardial infarction, other serious heart disorders, stroke); respiratory conditions (asthma/chronic bronchitis or chronic nonspecific lung disease); skin conditions (chronic eczema, psoriasis); endocrine, metabolic, and nutritional conditions (diabetes mellitus, hyperthyroidism, hypothyroidism); digestive conditions (serious disorders of the intestine longer than three months, e.g. Crohn’s disease); and a rest category (incontinence, cancer, HIV infection, glaucoma).

Other explanatory variables included sociodemographic and clinical variables which frequently have been found to be associated with GPs’ diagnosis of depression (Docherty, 1997; Tylee, 1999). Sociodemographic data (age, gender, highest educational level attained) were derived from the health interview survey. Educational level was categorized into three classes: low (none, elementary school), middle (high school), and high (college or university). Severity of depression was derived from the CIDI and was operationalized as the number of depressive symptoms with scores ranging from 5 to 9 (DSM-IV criterion A for MDD). Patients who had a diagnosis of dysthymia alone (n = 7) were given a score of four on this measure. Annual number of GP–patient contacts was categorized by quartiles, which resulted in the categories 1–3, 4–6, 7–10, and ≥11 contacts.

Statistical analyses

Rates of GPs’ depression diagnosis in subgroups of depressed patients based on co-morbidity characteristics were calculated to explore the relationship between co-morbidity status and depression diagnosis. In a second explorative analysis, the bivariate relationships between co-morbidity, sociodemographic (age, gender, educational level) and clinical (depression severity and GP contact rate) variables and depression diagnosis were examined using simple logistic regression analyses. Next, multivariate logistic regression analysis was conducted in three steps. The first model entered psychiatric co-morbidity and chronic somatic co-morbidity. In addition to the co-morbidity variables, model 2 entered the sociodemographic and clinical variables. The final multivariate model included model 1 and 2 variables, plus the interaction term between psychiatric and chronic somatic co-morbidity. In case of a significant interaction effect, separate multivariate analyses were carried out in patient subgroups stratified by presence or absence of chronic somatic co-morbidity.

Additional regression analyses were performed to explore the effects of the number of co-morbidities (categorized into three categories: 0, 1, and ≥2) and the specific co-morbid disease categories. Also, a number of supplementary analyses were performed to test the robustness of the multivariate logistic regression results. First, several studies have reported an association between depression severity and psychiatric co-morbidity, in particular anxiety co-morbidity (e.g. Roy-Byrne et al. 2000). To examine a possible collinearity effect between these variables multivariate regression analyses were repeated without the variable depression severity. Secondly, to examine whether the possible inclusion of unexplained symptoms...
affected the results, analyses were repeated after excluding patients with symptom diagnosis only and no somatic disease diagnosis. Thirdly, to test whether lowering the GHQ-12 cut-off score influenced the results, analyses were repeated including a dummy variable GHQ threshold. Finally, all analyses were repeated in multilevel models to examine whether the results were affected by variations among the general practices.

Analyses were conducted using SPSS version 11.0 for Windows, except for the multilevel analyses, which were performed using the MLwiN software version 1.1. Significance was accepted at the 5% level.

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**RESULTS**

**Study population characteristics and depression diagnosis rates**

Table 1 illustrates the characteristics of the 191 depressed patients. According to the CIDI, 157 patients had MDD alone, seven had dysthymia alone, and 27 had both MDD and dysthymia. Psychiatric co-morbidity was present in just over half of the depressed patients, about the same prevalence rate as chronic somatic co-morbidity. Anxiety disorder was by far the most common co-morbid psychiatric disorder, with GAD being the most frequent specific anxiety disorder; the most prevalent co-morbid chronic somatic disease category was musculoskeletal, followed by the circulatory and neurological categories. About a quarter of the patients had both psychiatric and chronic somatic disease in addition to their depression, while two-fifths had no co-morbidity.

As shown in Table 2, 55 of the 191 depressed patients were diagnosed as depressed by GPs (ICPC code P76: n = 41; ICPC code P03: n = 11; both codes: n = 3), while 136 depressed patients were not diagnosed, resulting in an overall rate of underdiagnosis of 71.2%.
Interestingly, the depression diagnosis rates in patient subgroups based on co-morbidity characteristics suggest a possible interaction effect between psychiatric and chronic somatic co-morbidity on depression diagnosis. That is, the difference in depression diagnosis rate between depressed patients with psychiatric co-morbidity and those without psychiatric co-morbidity was small in the subgroup of patients without chronic somatic co-morbidity, as compared with the substantial difference observed in the subgroup of patients with chronic somatic co-morbidity.

**Logistic regression results**

As shown in Table 3, bivariate analyses revealed that having no psychiatric co-morbidity, having fewer contacts with a GP, and being less severely depressed all significantly increased the risk of underdiagnosis of depression. The multivariate results are also presented in Table 3. Shown are the main effects of psychiatric and chronic somatic co-morbidity on depression diagnosis when considered jointly in the same model (model 1) and when all other explanatory variables were entered (model 2). As a last step, the interaction between the two co-morbidity types was entered. Importantly, this final multivariate model showed a significant interaction effect between psychiatric and chronic somatic co-morbidity on depression diagnosis. Subsequent stratified analysis (see Table 4) revealed that psychiatric co-morbidity had no significant effect on depression diagnosis in patients without chronic somatic co-morbidity. Having a lower educational level, having a lower annual number of GP contacts and having a less severe level of depression all significantly increased the risk of underdiagnosis of depression in this patient subgroup. In contrast, in patients with chronic somatic co-morbidity, psychiatric co-morbidity was significantly associated with depression diagnosis: depressed patients with chronic somatic co-morbidity but no psychiatric co-morbidity were more likely to receive no depression diagnosis than those with both psychiatric and chronic somatic co-morbidity. None of the other variables was significantly associated with depression diagnosis in this patient subgroup.

**Additional multivariate analyses: number and category of co-morbidities**

The number of psychiatric co-morbidities exerted no significant effect in patients without chronic somatic co-morbidity, whereas among patients with chronic somatic co-morbidity a lower number of psychiatric co-morbidities...
decreased the likelihood of receiving a depression diagnosis (OR 2.40, 95% CI 1.26–4.59, p < 0.01). Again, significant effects of education, number of GP contacts, and depression severity were confined to patients without chronic somatic co-morbidity. Regarding specific co-morbidity categories, patients with a co-morbid musculoskeletal condition but no co-morbid GAD were more likely to receive no depression diagnosis than patients with both a co-morbid musculoskeletal condition and a co-morbid GAD (OR 6.17, 95% CI 1.39–27.41, p < 0.05).

In contrast, in patients without a co-morbid musculoskeletal condition no effect of co-morbid GAD was found. Again, only in these latter patients significant effects were present for education, contact rate and depression severity. The effects of other specific categories were not analysed, because patient numbers were too small for meaningful analysis.

**DISCUSSION**

About half of our sample of patients who had depression as assessed according to the CIDI had either co-morbid psychiatric or co-morbid chronic somatic disease. Nearly a quarter of the depressed patients exhibited both types of co-morbidity, and this high rate underlines the importance of studying the impact of having both chronic somatic as well as psychiatric co-morbidity on underdiagnosis of depression by GPs. Previous studies examined the effects of psychiatric and somatic co-morbidity only separately. Our study elaborated on the past research and showed that there is an interaction effect between psychiatric and chronic somatic co-morbidity on depression diagnosis. Only in depressed patients with a co-morbid chronic somatic condition did having no psychiatric co-morbidity increase the risk of being not diagnosed as depressed. Furthermore, none of the other factors under study were found to exert significant effects in this subgroup of patients, while in those without chronic somatic co-morbidity, a lower educational level, a lower annual number of GP contacts and a less severe level of depression all increased the likelihood of being not diagnosed as depressed.

It should be noted that the effect of psychiatric co-morbidity in patients with chronic somatic co-morbidity was no longer significant after excluding patients with only symptom decreased the likelihood of receiving a depression diagnosis (OR 2.40, 95% CI 1.26–4.59, p < 0.01). Again, significant effects of education, number of GP contacts, and depression severity were confined to patients without chronic somatic co-morbidity. Regarding specific co-morbidity categories, patients with a co-morbid musculoskeletal condition but no co-morbid GAD were more likely to receive no depression diagnosis than patients with both a co-morbid musculoskeletal condition and a co-morbid GAD (OR 6.17, 95% CI 1.39–27.41, p < 0.05).

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It should be noted that the effect of psychiatric co-morbidity in patients with chronic somatic co-morbidity was no longer significant after excluding patients with only symptom
diagnoses. Further research is needed to determine whether this was caused by reduced statistical power or whether it indicates that the significant psychiatric co-morbidity effect in our total group of patients with chronic somatic co-morbidity is a coincidental finding. A possible explanation for a facilitating effect of psychiatric co-morbidity on depression diagnosis is that GPs may be better able to detect a mental problem in chronically medically ill patients when non-depressive psychiatric symptoms, that is, symptoms more specific to anxiety and/or alcohol-related disorders, are also present. This higher detection rate might result in an increased likelihood of diagnosing depression, because GPs are probably better acquainted with depression than other psychiatric disorders encountered in primary care and will, therefore, interpret any mental distress as indications of depression (Sartorius et al. 1996). Another possible explanation is that a specific co-morbidity pattern accounted for the facilitating effect on diagnosing depression in patients with concomitant chronic somatic disease. Explorative analysis suggested that having a co-morbid GAD facilitated depression diagnosis in patients with a co-morbid musculoskeletal condition. However, the effects of other specific co-morbidity patterns remained unclear, because too few cases precluded meaningful analysis.

The higher annual GP contact rate among depressed patients with chronic somatic co-morbidity compared with those with no concomitant chronic somatic condition could explain the differential effect of contact rate in the two subgroups. Because almost all depressed, chronically somatically ill patients already had contact with their GP on a regular basis (i.e. ≥4 contacts: 92%; ≥7 contacts: 64%), it is probable that a higher frequency of contact will not have much influence on diagnosing depression. In contrast, a substantial number of depressed patients without somatic co-morbidity consulted their GP only once or a few times during the last year (i.e. ≤3 contacts: 32%; 1–2 contacts: 22%). Accordingly, GPs have little opportunity to recognize depressive symptoms in these patients, and the likelihood of depression diagnosis will be increased in the non-chronically ill patients with a higher contact rate.

One could question the clinical relevance of the finding that less severe depression decreased the likelihood of receiving a depression diagnosis in patients with no chronic somatic co-morbidity. Diagnosis may not be needed in all patients with relatively mild forms of depression, because quite a large number of these patients seem to recover spontaneously without being detected as depressed (Simon & VonKorff, 1995; Simon et al. 1999). On the other hand, it has been indicated that a substantial number of undetected depressed primary-care patients have persistently poor outcomes over the course of one year (Goldberg et al. 1998; Rost et al. 1998). Of concern is that a more severe level of depression did not facilitate diagnosis among patients with chronic somatic co-morbidity, particularly in the context that most of these patients had regular contact with their GPs. It is of special importance to diagnose major depression in chronically medically ill patients, because its presence has been demonstrated to lead to amplification of chronic medical illness symptoms, additive functional impairment, and poorer self-care and adherence (Whooley & Simon, 2000; Katon & Ciechanowski, 2002). Also, depression co-morbid with chronic somatic disease may have poorer course and outcome than depression without co-morbidity (Maier & Falkai, 1999; Wittechen et al. 1999). For these reasons, it is widely advocated that major depression must be appropriately and aggressively treated in patients with chronic somatic disease (Sutor et al. 1998; Whooley & Simon, 2000; Cassano & Fava, 2002). Indeed, appropriate treatment has been found to improve both the course and outcome of depression and the co-morbid somatic disease as well as patient quality of life (Maier & Falkai, 1999; Koike et al. 2002; Stockton et al. 2004).

It should be kept in mind that the discussed results were obtained by using a multivariate model that included both psychiatric co-morbidity and depression severity. In the vast majority of cases psychiatric co-morbidity implied co-morbid anxiety disorder(s), with GAD being the most prevalent specific co-morbid anxiety disorder. As anxiety disorders in general, and GAD particularly, share several common symptoms with MDD, one might question whether anxiety co-morbidity and
depression severity are distinct. Indeed, some researchers view them not as distinct entities, but rather conceptualize anxiety-depression co-morbidity as an indicator of severity of psychopathology (Preisig et al. 2001; Angst et al. 2002; Schoevers et al. 2003). However, we consider anxiety co-morbidity and depression severity as related but distinct constructs. This position was based on reviews of research on the most common anxiety-depression co-morbidity, that is, MDD–GAD co-morbidity, which argue against the view that GAD should be conceptualized as a severity marker for MDD rather than as an independent disorder (Kessler, 2000; Kessler et al. 2001). Several lines of evidence point to this conclusion, including reports that the symptoms of GAD form an empiric cluster distinct from the symptoms of major depression. Viewing psychiatric co-morbidity and depression severity as distinct entities is supported by the finding that the effects of psychiatric co-morbidity on depression diagnosis remained essentially the same after dropping depression severity from the multivariate model. If psychiatric co-morbidity and depression severity were indistinguishable entities, one would expect the model excluding depression severity to show a significant effect of psychiatric co-morbidity on depression diagnosis among depressed patients without chronic somatic co-morbidity, which was not the case. Anyhow, further longitudinal research is needed to disentangle the concepts of psychiatric co-morbidity and depression severity and their relationship with depression diagnosis.

Some limitations of our study must be noted. First, the generalizability of the findings might be restricted by the relatively high attrition rate. The significant difference between participant and non-participants regarding gender seemed to be of no major influence given that gender consistently did not exert a significant effect in our analyses. However, it cannot be ruled out that other differences between participants and non-participants could have affected the findings. Secondly, not all sections of the CIDI were administered, which may have confounded assessment of psychiatric co-morbidity. For instance, the assessment of somatoform disorders was lacking, which are known to be common in primary care and co-morbid with depression (Maier & Falkai, 1999). Finally, only small numbers of patients had certain specific diseases, which precluded meaningful analysis of the effects of specific (combinations of) psychiatric and chronic somatic co-morbidity categories.

In conclusion, the results of our study indicate that the factors that are associated with under-diagnosis of depression by GPs are different for depressed patients without chronic somatic co-morbidity in comparison with depressed patients with chronic somatic co-morbidity. This implies that efforts to improve GPs’ diagnosis of depression require different approaches for depressed patients with and without co-morbid somatic illness. Our results suggest that GPs need to be more alert to symptoms of depression in the less well-educated, non-chronically somatically ill patients. The awareness of (the importance to diagnose and treat) depression in chronically somatically ill patients should be raised among GPs. Educating GPs in overcoming the diagnostic challenge of differentiating depressive symptomatology from co-morbid chronic somatic disease and/or to refer chronically somatically ill patients to mental health specialists if they suspect a depression could increase the quality of care for these patients.

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DECLARATION OF INTEREST

None.

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