

# Depression and cardiovascular disease: ongoing need for diagnosis and appropriate management to improve quality of life

David L. Hare

Department of Medicine, University of Melbourne, Heidelberg, Australia

---

Article Piwoński et al., see p. 18

---

The relationship between depression and cardiovascular disease (CVD) is well established. Whilst both have a high prevalence in most societies, the higher prevalence of depression in persons with CVD suggests a relationship beyond coincidence. It seems that depression is both a cause and consequence of CVD [1]. In balance, it is probable that depression is more commonly a consequence of clinical CVD.

However, there are some data suggesting that those who are depressed are more likely to subsequently develop CVD. These data rely on long-term follow-up analyses as well as case-control studies. The former require population studies that have assessed baseline depression, followed many years later by the accurate diagnosis of incident CVD [2]. The latter studies require a matched control group of patients who are presenting without CVD, so that tight control of covariates is possible [3].

This study of Piwoński et al. [4] sheds some additional light on the relationship between depressive symptoms and CVD in a Polish population. Excellent subject selection protocol of the NATPOL2011 study suggests that the data are likely to be representative of the overall Polish population. There are also well defined *a priori* definitions for hypertension, diabetes, obesity and smoking, although no lipid data are provided as covariates for this publication. The diagnosis of CVD relies on self-reported medical history.

The use of the term “depressive symptoms” is appropriate, given the use of a Beck Depression Inventory (BDI) score  $\geq 10$  for the dichotomous cut-off diagnosis. A score of 10 to 18 generally indicates only “mild depression,” and even scores of 19 to 29 still only indicate “moderate depression.” In addition, the BDI scores can also be inflated, independently of depression, by physical symptoms such as fatigue, although it must be recognised that fatigue is actually a common symptom of true depression. Therefore, the prevalence of depression in this population will appear higher than those reported in

other studies of both general populations and in those with existing CVD [1]. For example, the 12-month prevalence of major depression in the United States is 6.6%, similar to many other general populations [5].

Logistic regression analysis shows that both age and educational level are independent sociodemographic characteristics associated with depressive symptoms. The very low odds ratio for depression in divorced and widowed men is surprising, given that isolated men generally have high levels of loneliness and depression, possibly because non-occupational sociological connections are more often determined by the female partner. It is possible that there was a higher degree of symptom denial while responding to the BDI self-report depression questionnaire. In this regard, it would be interesting to know the rates of inappropriate alcohol use in the different sub-populations.

The small number of subjects reporting previous myocardial infarction (61 subjects) will unfortunately be not sufficient enough to exclude any significant relationship with depressive symptoms. Similarly, there would have been far too few subjects with heart failure to describe any meaningful relationship with depressive symptoms, even though heart failure patients have an extremely high prevalence of depression [6].

The detection of depression (either just its symptoms or as a clinical entity) remains extremely important in CVD patients, whether one just asks a couple of relevant questions or uses a validated brief screening tool [7]. Whilst there are good data for improving depression in cardiac patients, there remain no randomised controlled trial data for any improvement in CVD endpoints by treating depression in CVD patients [8]. However, depression is arguably the single most important driver of quality of life and therefore needs to be prevented and appropriately managed if it is subsequently detected in CVD patients.

**Conflict of interest:** none declared

---

**Address for correspondence:**

Prof. David L Hare, Department of Medicine, University of Melbourne, Austin Health, Heidelberg, Vic 3084, Australia, e-mail: david.hare@unimelb.edu.au

Kardiologia Polska Copyright © Polish Cardiac Society 2019

Note: The opinions expressed by the authors are not necessarily those of the journal editors, Polish Cardiac Society or publisher.

### References

1. Hare DL, Toukhsati SR, Johansson P, et al. Depression and cardiovascular disease: a clinical review. *Eur Heart J*. 2014; 35(21): 1365–1372, doi: [10.1093/eurheartj/eh462](https://doi.org/10.1093/eurheartj/eh462), indexed in Pubmed: [24282187](https://pubmed.ncbi.nlm.nih.gov/24282187/).
2. Nicholson A, Kuper H, Hemingway H. Depression as an aetiological and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146 538 participants in 54 observational studies. *Eur Heart J*. 2006; 27(23): 2763–2774, doi: [10.1093/eurheartj/ehl338](https://doi.org/10.1093/eurheartj/ehl338), indexed in Pubmed: [17082208](https://pubmed.ncbi.nlm.nih.gov/17082208/).
3. Yusuf S, Hawken S, Ôunpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004; 364(9438): 937–952, doi: [10.1016/s0140-6736\(04\)17018-9](https://doi.org/10.1016/s0140-6736(04)17018-9).
4. Piwoński J, Piwońska A, Jędrusik P, et al. Depressive symptoms and cardiovascular diseases in the adult Polish population. Results of the NATPOL2011 study. *Kardiol Pol*. 2019; 77(1): 18–23, doi: [10.5603/KP.a2018.0213](https://doi.org/10.5603/KP.a2018.0213), indexed in Pubmed: [30406941](https://pubmed.ncbi.nlm.nih.gov/30406941/).
5. Kessler RC, Berglund P, Demler O, et al. National Comorbidity Survey Replication. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). *JAMA*. 2003; 289(23): 3095–3105, doi: [10.1001/jama.289.23.3095](https://doi.org/10.1001/jama.289.23.3095), indexed in Pubmed: [12813115](https://pubmed.ncbi.nlm.nih.gov/12813115/).
6. Rutledge T, Reis VA, Linke SE, et al. Depression in heart failure: a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol*. 2006; 48(8): 1527–1537, doi: [10.1016/j.jacc.2006.06.055](https://doi.org/10.1016/j.jacc.2006.06.055), indexed in Pubmed: [17045884](https://pubmed.ncbi.nlm.nih.gov/17045884/).
7. Hare DL, Selvadurai LP, Wang JQ, et al. Psychometric assessment of the Cardiac Depression Scale Short Form in cardiac outpatients. *Eur J Cardiovasc Nurs*. 2017; 16(3): 249–255, doi: [10.1177/1474515116652759](https://doi.org/10.1177/1474515116652759), indexed in Pubmed: [27231395](https://pubmed.ncbi.nlm.nih.gov/27231395/).
8. Blumenthal J, Sherwood A, Babyak M, et al. Exercise and pharmacological treatment of depressive symptoms in patients with coronary heart disease. *J Am Coll Cardiol*. 2012; 60(12): 1053–1063, doi: [10.1016/j.jacc.2012.04.040](https://doi.org/10.1016/j.jacc.2012.04.040).