

# Association of depression with subsequent mortality, cardiovascular morbidity and incident dementia in people aged 80 and over and suffering from hypertension. Data from the Hypertension in the Very Elderly Trial (HYVET)

RUTH PETERS<sup>1</sup>, ELISABETE PINTO<sup>1</sup>, NIGEL BECKETT<sup>1</sup>, CAMERON SWIFT<sup>2</sup>, JOHN POTTER<sup>3</sup>, TERRY MCCORMACK<sup>4</sup>, MARIA NUNES<sup>5</sup>, JOHN GRIMLEY-EVANS<sup>6</sup>, ASTRID FLETCHER<sup>7</sup>, CHRISTOPHER BULPITT<sup>1</sup>

<sup>1</sup>Care of the Elderly, Faculty of Medicine, Imperial College London, Du Cane Road, London W12 0NN, UK

<sup>2</sup>Clinical Age Research Unit, Department of Clinical Gerontology, King's College Hospital, Bessemer Road, London SE5 9PJ, UK

<sup>3</sup>Ageing & Stroke Medicine Section, School of Medicine, Health Policy and Practice, University of East Anglia, Norfolk NR4 7TJ, UK

<sup>4</sup>Whitby Group Practice, Spring Vale Medical Centre, Whitby YO21 1SD, UK

<sup>5</sup>Centro Universitário São Camilo, Rua Raul Pompéia, 144, São Paulo 05025-010, Brazil

<sup>6</sup>Nuffield Department of Clinical Medicine Division, John Radcliffe Hospital, Oxford OX3 9DU, UK

<sup>7</sup>Department of Epidemiology & Population Health, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK

Address correspondence to: Ruth Peters. Tel: +44 (0)20 75948974; Fax: +44 (0)20 74022150. E-mail: r.peters@imperial.ac.uk

## Abstract

**Background:** depression is common in elderly people and may be associated with increased cardiovascular risk and incident dementia.

**Method:** participants in the Hypertension in the Very Elderly Trial (HYVET) completed a depression screening instrument, the Geriatric Depression Score (GDS), at baseline and annually. We examined the association of GDS score with incident stroke, mortality and dementia using Cox proportional hazards models (hazard ratios, HR and 95% confidence intervals, CI) adjusted for treatment group and other potential confounders.

**Results:** 2,656 HYVET participants completed the GDS. The mean follow-up was 2.1 years. A GDS score  $\geq 6$  was associated with increased risks of all-cause (HR 1.8, 95% CI 1.4–2.3) and cardiovascular mortality (HR 2.10, 95% CI 1.5–3.0), all stroke (HR 1.8, 95% CI 1.2–2.8) and all cardiovascular events (HR 1.6, 95% CI 1.2–2.1). Risk of incident dementia also tended to be increased (HR 1.28, 95% CI 0.95–1.73). Each additional GDS point at baseline also gave rise to a significantly increased risk of fatal and non-fatal cardiovascular events, all-cause mortality and dementia.

**Conclusion:** there was a strong association between baseline depression scores and later fatal and non-fatal cardiovascular endpoints over a mean follow-up of 2 years in a hypertensive very elderly group. The mechanism of this association warrants further study.

**Keywords:** cardiovascular diseases, aged, depression, hypertension, mortality

## Introduction

Depression or depressed mood is common among older people with an average prevalence of clinically relevant syndromes estimated at 13.5% at ages  $\geq 55$  years [1, 2] and higher in women than in men [2]. Many studies have found an association between depression, poor quality of

life and increased risk of cardiovascular disease [3–5]. Depression was associated with an increased risk of coronary disease in a meta-analysis [6] and with increased risk of all-cause mortality and stroke or transient ischaemic attack in the Framingham Heart Study [7, 8]. Plausible causal mechanisms have been put forward in the literature [9–13].

Depression may also be associated with increased risk of later dementia [14].

Depression is common after stroke, heart attack and in early dementia [13–15], and is strongly associated with disability [16, 17], raising the possibility that the association could be confounded or subject to reverse causation.

The Hypertension in the Very Elderly Trial (HYVET) [18] enrolled hypertensive people aged  $\geq 80$  years. As part of a quality-of-life sub-study, depression in HYVET was assessed at baseline and then annually using the Geriatric Depression Scale (GDS) [19], and provided an opportunity to study the association of depression with stroke, cardiovascular morbidity, mortality and dementia in a high-risk group of people.

## Method

### Participants

The HYVET was a randomised double-blind, placebo-controlled trial and employed an antihypertensive treatment regimen of indapamide sustained release 1.5 mg with the optional addition of perindopril 2–4 mg. Ethical and regulatory approvals were obtained prior to data collection.

All participants were hypertensive defined by a sitting systolic blood pressure of  $\geq 160$  mmHg and a standing pressure of  $\geq 140$  mmHg, with a baseline diastolic pressure of  $\leq 110$  mmHg. Trial participants were aged  $\geq 80$  years, had no clinical diagnosis of dementia at baseline and did not require daily nursing care. The trial had a 2-month placebo run-in phase with collection of baseline data on participant characteristics prior to randomisation. Participants gave informed consent and were recruited from hospital and general practice settings.

Depression scores were collected using the 15-item GDS administered as part of a Quality of Life (QoL) questionnaire at baseline and annually thereafter. The QoL questionnaire was completed by the respondents willing to participate in this sub-study to the main trial. Participants were asked to complete the questionnaire themselves prior to having their blood pressures measured and other clinical assessments. The QoL questionnaire was provided in large text and in the local language after being translated, checked and back-translated where validated language versions were not previously available. Sixty-nine percent of people entering HYVET completed the GDS in the baseline QoL questionnaire. Those participants completing the QoL questionnaire were from centres in Eastern (45.9%) and Western (1.8%) Europe, China (49.0%), North Africa (2.6%) and Australasia (0.7%). Respondents were instructed to respond 'yes' or 'no' to 15 questions relating to how they had been feeling in the preceding week.

### Outcomes

All-cause mortality, incident stroke, incident and worsening heart failure, and incident myocardial infarction (MI) were

reported by the trial investigators and validated by an independent endpoint committee blinded to trial treatment. Mortality was further categorised into cardiovascular and non-cardiovascular mortality. The endpoint committee required copies of the case report forms declaring the event and supporting documentation such as death certificates, hospitalisation reports, CT scan reports and information on the results of investigations. Where these were provided in a language other than English, translations were also obtained. The committee included members that were representative of the high recruiting countries in HYVET and who could read Chinese and Cyrillic scripts.

Dementia was suspected in those participants who had a mini-mental state examination (MMSE) score that fell to  $< 24$  or by  $> 3$  points annually, and in these cases further diagnostic information was requested in the form of the Diagnostic Statistical Manual criteria, a CT scan (a copy of the film) and completed Modified Hachinski Score. A contemporary MMSE was also requested. If the participant did not consent to a CT, a full Hachinski score was required. The CT scan was evaluated by two independent neuroradiologists, and an expert committee made a diagnosis of dementia using the diagnostic information in addition to other information that had been gathered in the course of

Table 1. Baseline characteristics

	Geriatric Depression Score $< 6$ N=1,769	Geriatric Depression Score $\geq 6$ N=887	P
Age—years (SD)	83.3 (3.0)	83.7 (3.2)	0.003
Percent female (%)	56.6 (1,001)	68.7 (609)	$< 0.001$
Previous stroke (%)	8.3 (146)	8.9 (79)	0.605
Previous cardiovascular disease <sup>a</sup> (%)	11.2 (199)	15.4 (137)	0.002
Education <sup>b</sup> (%)			
None	36.9 (653)	25.0 (222)	$< 0.001$
Some	63.1 (1,116)	75.0 (665)	
Mean sitting systolic blood pressure (mmHg) at entry (SD)	172.8 (8.9)	174.0 (8.7)	0.001
Mean standing systolic blood pressure (mmHg) at entry (SD)	168.0 (11.9)	168.2 (11.8)	0.591
Smoker (%)	7.1 (126)	5.4 (48)	0.097
Consumes alcohol (%)	15.7 (278)	15.9 (141)	0.910
Mean number of co-morbidities (SD)	1.6 (1.4)	2.0 (1.6)	$< 0.001$
Living alone (%)	18.5 (328)	22.9 (203)	0.009
Mean GDS (baseline) (SD)	2.2 (1.6)	9.3 (2.8)	$< 0.001$
Median GDS (baseline) (interquartile range)	2 (2–4)	9 (9–12)	$< 0.001$
Mean MMSE (baseline) (SD)	24.7 (4.5)	24.4 (4.7)	0.02
Median MMSE (baseline) (interquartile range)	26 (22–28)	25 (22–28)	0.008

<sup>a</sup>Includes participants who reported having suffered from a stroke, myocardial infarction or heart failure prior to entry into the trial.

<sup>b</sup>Participants reporting having received no formal education compared to those who reported some level of formal education.

**Table 2.** Depression as a risk factor for major events, GDS  $\geq 6$ 

Geriatric Depression Score $\geq 6$ N=2,656	Unadjusted		Adjusted for treatment		Fully adjusted <sup>a</sup>	
	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
All-cause mortality	1.79 (1.42–2.25)	<0.001	1.79 (1.43–2.25)	<0.001	1.78 (1.40–2.27)	<0.001
All stroke	1.88 (1.26–2.80)	0.002	1.88 (1.26–2.81)	0.002	1.82 (1.19–2.78)	0.006
Cardiovascular mortality	2.21 (1.60–3.06)	<0.001	2.22 (1.60–3.07)	<0.001	2.10 (1.50–2.96)	<0.001
Cardiovascular events <sup>b</sup>	1.72 (1.33–2.23)	<0.001	1.72 (1.33–2.22)	<0.001	1.59 (1.21–2.09)	0.001
Dementia (n=2,320)	1.30 (0.97–1.72)	0.75	1.29 (0.97–1.72)	0.758	1.28 (0.95–1.73)	0.110

<sup>a</sup>Adjusted for age, gender, treatment allocation, country area, educational level, living alone, number of co-morbidities, previous cardiovascular disease, previous treatment and previously diagnosed hypertension.

<sup>b</sup>Includes cardiovascular mortality, non-fatal stroke, non-fatal myocardial infarction and non-fatal heart failure.

**Table 3.** Depression as a risk factor for major events, continuous variable

Geriatric Depression Score—continuous variable N=2,656	Unadjusted		Adjusted for treatment		Fully adjusted <sup>a</sup>	
	HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
All-cause mortality	1.07 (1.04–1.10)	<0.001	1.07 (1.04–1.10)	<0.001	1.07 (1.04–1.10)	<0.001
All stroke	1.09 (1.04–1.14)	0.001	1.09 (1.04–1.14)	0.001	1.09 (1.04–1.14)	0.001
Cardiovascular mortality	1.09 (1.05–1.13)	<0.001	1.09 (1.05–1.13)	<0.001	1.09 (1.05–1.13)	<0.001
Cardiovascular events <sup>b</sup>	1.08 (1.05–1.11)	<0.001	1.08 (1.04–1.11)	<0.001	1.08 (1.05–1.11)	<0.001
Dementia (n=2,320)	1.04 (1.01–1.07)	0.044	1.04 (1.00–1.08)	0.046	1.04 (1.00–1.07)	0.046

<sup>a</sup>Adjusted for age, gender, treatment allocation, country area, educational level, living alone, number of co-morbidities, previous cardiovascular disease, previous treatment and previously diagnosed hypertension.

<sup>b</sup>Includes cardiovascular mortality, non-fatal stroke, non-fatal myocardial infarction and non-fatal heart failure.

the trial, e.g. serial MMSE, clock drawing tests, GDS, concomitant medication and disease including history of stroke.

### Statistical analysis

In common with other studies [20–22], we categorised the GDS score as a binary variable of a score  $\geq 6$  (to indicate depression) compared to  $<6$ . In secondary analyses, we also explored the GDS as a continuous variable. We used Cox proportional hazards models to investigate the association between the baseline GDS score with all-cause and cardiovascular mortality, fatal and non-fatal stroke events, fatal and non-fatal cardiovascular events (including cardiovascular mortality, all stroke, all heart failure and all MI) and incident dementia.

Analyses were carried out for the association of GDS score with outcome (i) unadjusted, (ii) adjusted for treatment group and (iii) adjusted for multiple factors including age, sex, treatment allocation, country area, educational level, living alone, number of co-morbidities, previous cardiovascular disease, previous treatment and previously diagnosed hypertension. Proportional hazards assumptions were tested. This paper presents baseline GDS data and its relationship to subsequent trial endpoints. Effect of trial treatment on GDS scores will be published separately.

### Results

A total of 2,656 completed questionnaires were received with GDS information and for 2,320 of these longitudinal

MMSE data were also available (and therefore the possibility of assessment for incident dementia).

The participants who completed the QoL questionnaires were of a similar age to those who did not (mean age of those who completed the QoL was 83.5 years compared with 83.7 years) and sex, with 60.6% female compared with 60.2%. Mean follow-up was 2.06 years with only 17 participants being lost to follow-up during the HYVET.

A third of participants (n=887) had GDS scores of  $\geq 6$  indicating depressed mood, although investigators reported clinical depression in only 15 (0.6%). When this was examined by prior cardiovascular disease (stroke, myocardial infarction or heart failure; previous CVD), 32.3% of those without previous CVD reported GDS scores  $\geq 6$  compared to 40.8% in those with previous CVD.

The baseline characteristics of patients with a GDS of  $\geq 6$  compared with those with GDS  $<6$  are shown in Table 1. A score of  $\geq 6$  was associated with being older, a female, a baseline history of CVD, some level of education, living alone, a slightly higher sitting systolic blood pressure, an increased number of co-morbidities and a slightly lower baseline MMSE.

Of those who completed QoL questionnaires, 294 (11%) participants died during follow-up; 146 (5.5%) of these were classified as cardiovascular deaths. There were 233 (8.8%) cardiovascular events and 173 (7.5%) cases of incident dementia.

A baseline GDS score of  $\geq 6$  was associated with a significantly increased risk of all-cause mortality [hazard ratio (HR) 1.8, 95% confidence intervals (CI) 1.4–2.3], all stroke

(HR 1.9, 95% CI 1.3–2.8), cardiovascular mortality (HR 2.2, 95% CI 1.6–3.1) and cardiovascular events (HR 1.7, 95% CI 1.3–2.2) (Table 2). These hazard ratios were not substantially changed after adjustment for treatment or in the fully adjusted model. When sensitivity and specificity values were calculated for a GDS of  $\geq 6$  compared to a GDS of  $< 6$ , specificity was high,  $> 90\%$  for all-cause mortality, all stroke, cardiovascular mortality, cardiovascular events and dementia. Corresponding sensitivity values were low at 16% for all-cause mortality, 5% for stroke, 9% for cardiovascular mortality, 12% for cardiovascular events and 10% for dementia.

Similar results were observed in the analysis for GDS as a continuous variable (Table 3). In these analyses, there was an additional association with increased risk of dementia both in unadjusted analyses and when adjusted for multiple confounders (Table 3). Further adjustment adding baseline sitting systolic blood pressure, smoking and consumption of alcohol at baseline to the multivariate analysis made little material difference to the effect sizes.

Further examination of these relationships by subgroups of gender, ethnicity (Chinese versus non-Chinese) and presence of previous cardiovascular disease resulted in point estimates over unity for all outcomes and the majority of subgroups retaining significance. Caution must be applied in this area as although the association of the GDS and subsequent events was planned a priori, the evaluation of subgroups was not. The subgroups are by definition smaller than the main data set and, in the case of ethnicity, are also affected by length of follow-up with the Chinese patients entering the trial later and therefore contributing less patient years.

## Discussion

We found that a GDS score of  $\geq 6$  was associated with an increased risk of all-cause and cardiovascular mortality and cardiovascular morbidity. The relationship between depression, mortality and cardiovascular mortality and morbidity is likely to be complex, with potential for confounding and reverse causation as well as a possible causal relationship between depressed mood and mortality or cardiovascular outcomes.

Mood was worse in those with a prior cardiovascular event [11–13, 15]. The association between mood and outcomes was seen over a relatively short follow-up period of 2 years. These suggest the possibility of reverse causation: outcome cardiovascular events may have reflected the greater occurrence, on average, of recurrent events in those with cardiovascular disease at recruitment.

There is a possibility of confounding by unmeasured disability. But adjusting for prior events, or other potential confounders, did not alter the risk estimates (although the possibility of residual confounding remains).

A further possibility is that depressed mood causes cardiovascular disease and mortality. This could be by several mechanisms such as less physical exercise, worse diet, the

effects of antidepressant drugs or other postulated pathways including hypertension, hyperlipidaemia, endothelial injury, progressive atherosclerosis and thrombus formation [3–5]. This raises the possibility that treatment of depression could reduce the risk of vascular disease. Our findings require replication and exclusion of some alternative possibilities (such as following up a population known to be free of vascular disease or disability at baseline, or carefully controlling for the confounding effect of disability) before testing in an intervention trial.

A third of participants in the quality-of-life sub-study of the HYVET had GDS scores of  $\geq 6$ , indicating depressed mood. This is much higher than found in population studies [20–22], and a possible limitation of the study is the use of the GDS, especially across different countries and cultures. However, the GDS is a well-established and a good screening instrument for depression, and was designed for use among older people [23–31]. Multiple cut-off points have been used with varying levels of sensitivity and specificity, the most frequently used, as in our study, being 5/6 with higher scores indicating more severe depression [23]. With cut offs  $< 6/6+$ , the 15-item GDS has a specificity for depression of 93.7% and sensitivity of 50%, respectively.

Respondents who completed the GDS were representative of the HYVET population and were very elderly, with the majority being female. They were hypertensive but relatively healthy, with a mean number of co-morbidities of 1.7, and almost 20% were living alone. The mean MMSE of participants was 24.6 (SD 4.5), a score compatible with their age and varied levels of education [23].

A strength of the study was the administration of the questionnaire with the answers provided by the patients before commencing the trial clinical examination. This may have provided a more accurate representation of the respondents' views than would be reflected in the low levels of depression reported by the investigators.

The HYVET is the first to study a large population of very elderly individuals with hypertension and to have included the assessment of depression using the GDS.

However, there are several limitations. We report here the association between baseline GDS scores and later events; however, the study was not designed to investigate this and was stopped early at the time of the second interim analysis due to a positive finding in favour of active treatment, resulting in a relatively short mean follow-up of 2.1 years. Moreover, although participants were unable to enter the study if they required nursing care, we did not collect rigorous information about activities of daily living, disability levels or maintenance of social networks, socio-economic status or activity level. Participants were all hypertensive, and although a proportion of them were reported to have previous cardiovascular disease, there may also have been unreported or subclinical disease that we were not able to take into account. There is, therefore, the potential for uncontrolled confounding from unmeasured factors, including those above, or of reverse causality

whereby unreported or unrecognised ischaemic events lead to depression rather than the other way around.

### Key points

- Depressed mood is common in older people with hypertension.
- Higher depression scores were associated with an increased risk of a subsequent cardiovascular event, mortality and possibly dementia.
- These associations occurred over a short period of time, mean follow-up 2.1 years.
- The association may or may not be causal.

### Acknowledgements

All persons mentioned in the acknowledgements have given written consent.

### Conflict of interest

Imperial College received funding from the British Heart Foundation and Servier International to run the trial and support salary and consultancy costs for staff including Christopher Bulpitt, Nigel Beckett, Ruth Peters and Elisabete Pinto. Honoraria for speaking at symposia have also been received by Christopher Bulpitt, Nigel Beckett and Ruth Peters. No other authors report conflicts of interest.

### Funding

The HYVET trial is registered with ClinicalTrials.gov number NCT00122811. HYVET was supported by grants from the British Heart Foundation and the Institut de Recherches Internationales Servier. Imperial College was the sponsor and co-ordinator of the trial. The analysis, interpretation of the data, generation of the manuscript and decision to submit for publication were carried out independently of the funding bodies and the primary author had full access to all of the data and final responsibility for the decision to submit for publication. All ethical and regulatory approvals were received from participating countries and centres.

### Sponsor's role

HYVET was funded by grants from the British Heart Foundation and the Institut de Recherches Internationales Servier. Imperial College was the sponsor and co-ordinator of the trial. The funding bodies had no influence on design, methods, recruitment, data collection, analysis or preparation of the paper.

The HYVET is registered with ClinicalTrials.gov number NCT00122811 <http://clinicaltrials.gov/>

The committee members and investigators for HYVET were as follows: **Co-ordinating Centre:** C.J. Bulpitt (lead investigator), A.E. Fletcher (co-investigator), N.S. Beckett (trial co-ordinator), R. Peters (deputy trial co-ordinator), HYVET co-ordinating team at Imperial College London (1999–2008);

**HYVET Committees: Steering Committee:** T. McCormack, J. Potter, B.G. Extremera, P. Sever, F. Forette, D. Dumitrascu, C. Swift, J. Tuomilehto, J. Coope (retired in 2001), C. Nachev (deceased); **Data Monitoring Committee:** J. Staessen, L. Thijs, R. Clarke, K. Narkiewicz; **End Points Committee:** C. Davidson (retired in 2003), J. Duggan, G. Leonetti, N. Gainsborough, M.C. De Vernejoul, J. Wang, V. Stoyanovsky; **Dementia Validation Committee:** J. Tuomilehto, R. Clarke, A. Waldman, I. Walton, C. Ritchie; **Ethics Committee:** R. Fagard, J. Grimley Evans, B. Williams;

### Investigators: (\*national co-ordinators)

**Australia**—R. Warne\* and I. Puddey\*, M. Woodward, R. Penhall, C. Inderjeeth, S. Roger, R. Scholes, C. Johnson; **Belgium**—H. Celis\*, G. Adriaens, W. Onsea, K. Cornelli, D. Vantroyen, P. Cleen, P. de Voogt; **Bulgaria**—C. Nachev\* (deceased) (national co-ordinator from 1998 to 2005), V. Stoyanovsky\* (national co-ordinator after 2005), P. Solakov, R. Prokopova, E. Mantova, D. Smilkova, S. Mantov, K. Yankulova, R. Kermova, D. Popov, V. Sirakova, V. Gergova, D. Kamenova, F. Grigorov, T. Vassileva, R. Alahverdian, M. Tzekova, A. Postadjian, M. Geneva, V. Mincheva, T. Petrusheva, A. Toncheva, I. Gruev, V. Tsanova; **China**—L. Liu\*, H. Ge, S. Wang, J. Wang, W. Zhang, S. Jin, L. Ge, Y.F. Lu, S. Ma, L. Shen, J. Guo, Z. Lv (deceased), R. Huang, X. Li, B. Guo, G.E. Yuan, T. Zhang, L. Zhang, J. Feng, Z. He, J. Wang, L. Deng, L. Liu, Q. Yuan, F. Zhang, H. Li, D. Wang, K. Yang, M. Sun, H. Liu, X. Yan, F. Ren, J. Tang, M. Zhao, X. Luo, H. Zhou, H. Sang, Jie Wang, L. Yan, Zhixing Wang, J. Zhang, Chengzhi Wang; **Finland**—R. Antikainen\*, T. Strandberg, T. Konttila, A. Hynninen, M. Jääskivi, J. Airas, T. Jääskeläinen, J. Tuomilehto, H. Litmanen, T. Karhi, H. Yliharsila; **France**—F. Forette\*, J. Doucet, J. Belmin, A. Benetos, G. Berrut, T. Boge, M. Bonnefoy, A. Carre, N. Charasz, J. Covillard, T. Dantoine, M. Escande, Y. Frances, R. Joire, C. Jeandel, S. Legrain, A. Lion, M. Maillet-Vioud, J.P. Escaillas, S. Meaume, P. Pfitzenmeyer, F. Puisieux, Quercy, O. Rodat, J. Soubeyrand, B. de Wazieres, H. Hindennach, L. Lugassy, J. Rossi, M. Martel, J.-M. Paladel, C. Ravier, A. Visconti, J.P. Gallet, D. Zygouritsas, D. Charles, F. Flamand, G. Grandmottet, M. Grandmottetgermann, C. Gevrey, P.L. Mesnier, G. Robert, C. Besset-Prat, A. Brousse, P. Lafont, J. Morelli, P. Vernede, A. Volkmann, X. Bodin, B. Destrube, R. Eoche, A. Boye, F. Seropian, P. Gernigon, D. Meker, J. Thomere, Y. Thual, F. Volny, E. Grassart, M. Herent, D.

Lejay, J.-P. Lopez, B. Mannesier, G. Pruvost, J.-C. Urbina; **Ireland**—J. Duggan\*; **New Zealand**—C. Anderson\*, S. Lillis, J. Gommans; **Poland**—T. Grodzicki\*, Z. Chodorowski, Z. Gaciong; **Romania**—D. Dumitrascu\*, M. Comsa, V. Sandru, G. Prada, M. Dunca-Moisin, D. Jianu, D. Jinga-Lazar, V. Enachescu, C. Zaharia; **Russia**—Y. Nikitin\*, A. Kirichenko, L. Olbinskaya, A. Martynov, V. Zadionchenko, V. Moiseev, G. Storozhakov, S. Nedogoda, R.S. Karpov, O. Barbarash, G. Efremushkin, V. Kostenko, M. Boyarkin, S. Churina, T. Tyurina, M. Ballyuzek, L. Ermoshkina, A. Timofeev, S. Yakusheva, N. Shilkina, V. Barbarich, L. Latunceva, S. Burakova, T. Ripp, S. Pekarsky, V. Mordovin; **Tunisia**—A. Belhani\*, E. Boughzela, S. Soraya, B. Youssef-Zouari, A.B. Khalfallah, M.H. Houman, A.K. Abida; **UK**—C. Rajkumar\*, M. Wilkins, N.D. Pandita-Gunawardena, J. Potter, E. Ekpo, M. Price, N. de Kare-Silver, A. Starczewski, S. Chandran, N. Nasar, M. Datta-Chaudhuri, T. McCormack, N. Majmudar, A. Gordon, L. Brawn, T. Solanki, F. Dockery, R. Schiff.

We wish to acknowledge the work of Professor C. Nachev (Steering Committee member, National Co-ordinator of Bulgaria and HYVET investigator from 1998 until his death in 2005).

## References

1. Beekman A, Copeland J, Prince M. Review of community prevalence of depression in later life. *Br J Psychiatry* 1999; 174: 307–11.
2. McDougall F, Kvaal K, Matthews F *et al.* Prevalence of depression in older people in England and Wales: the MRC CFA study. *Psychol Med* 2007; 37: 1787–95.
3. Barger S, Muldoon M. Hypertension labelling was associated with poorer self-rated health in the Third US National Health and Nutrition Examination Survey. *J of Hum Hypertens* 2006; 20: 117–23.
4. Mena-Martin F, Martin-Escudero J, Simal-Blanco F *et al.* Health-related quality of life of subjects with known and unknown hypertension: results from the population-based Horteiga study. *J Hypertens* 2003; 21: 1283–9.
5. Jorge R, Robinson R, Arndt S *et al.* Mortality and poststroke depression: a placebo-controlled trial of antidepressants. *Am J of Psychiatr* 2003; 160: 1823–9.
6. Wulsin L. Do depressive symptoms increase the risk for the onset of coronary disease? A systematic quantitative review. *Psychosom Med* 2003; 65: 201–10.
7. Salaycik K, Kelly-Hayes M, Besier A *et al.* Depressive symptoms and risk of stroke: the Framingham Study. *Stroke* 2007; 38: 16–21.
8. Wulsin L, Evans J, Vasan R *et al.* Depressive symptoms, coronary heart disease and overall mortality in the Framingham Heart Study. *Psychosom Med* 2005; 67: 697–702.
9. Laghrissi-Thode F, Wagner W, Pollock B *et al.* Elevated platelet factor 4 and  $\beta$ -thromboglobulin plasma levels in depressed patients with ischemic heart disease. *Biol Psychiatry* 1997; 42: 290–5.
10. Carney R, Saunders R, Freedland K *et al.* Association of depression with reduced heart rate variability in coronary heart disease. *Am J Cardiol* 1995; 76: 562–4.
11. Taragano F, Bagnatti P, Allegri R. A double blind randomised clinical trial to assess the augmentation with nimodipine of antidepressant therapy in the treatment of “vascular depression”. *Int Psychogeriatr* 2005; 17: 487–98.
12. Krishnan K. Depression as a contributing factor in cerebrovascular disease. *Am Heart J* 2000; 140: S70–6.
13. Brodaty H, Withall A, Altendorf A *et al.* Rates of depression at 3 and 15 months poststroke and their relationship with cognitive decline: the Sydney Stroke Study. *Am J Geriatr Psychiatry* 2007; 15: 477–86.
14. Jorm A. History of depression as a risk factor for dementia: an updated review. *Aust N Z J Psychiatry* 2001; 35: 776–81.
15. van Melle JP, de JP, Spijkerman TA, Tijssen JG *et al.* Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis. *Psychosom Med* 2004; 66: 814–22.
16. Prince M, Harwood R, Blizard R, Thomas A, Mann A. Impairment, disability and handicap as risk factors for depression in old age. The Gospel Oak Project V. *Psychol Med* 1997; 27: 311–21.
17. Prince M, Harwood R, Thomas A, Mann A. A prospective population-based cohort study of the effects of disablement and social milieu on the onset and maintenance of late life depression. The Gospel Oak Project VII. *Psychol Med* 1998; 28: 337–50.
18. Yesavage JA, Brink TL, Rose TL, Lum O *et al.* Development and validation of a geriatric depression screening scale: a preliminary report. *J Psychiatr Res* 1983; 17: 37–49.
19. Beckett N, Peters R, Fletcher A *et al.* for the HYVET Study Group\* Treatment of hypertension in patients 80 years of age or older. *N Engl J Med* 2008; 358: 1887–98.
20. Arthur A, Jagger C, Lindsay J *et al.* Using an annual over-75 health check to screen for depression: validation of the short Geriatric Depression Scale (GDS15) within general practice. *Int J Geriatr Psychiatry* 1999; 14: 431–9.
21. De Craen A, Heeren T, Gussekloo J. Accuracy of the 15 item Geriatric Depression Scale (GDS-15) in a community sample of the oldest old. *Int J Geriatr Psychiatry* 2003; 18: 63–6.
22. Osborn D, Fletcher A, Smeeth L *et al.* Factors associated with depression in a representative sample of 14 217 people aged 75 or over in the United Kingdom: results from the MRC trial of assessment and management of older people in the community. *Int J Geriatr Psychiatry* 2003; 18: 623–30.
23. Spreen O, Strauss E. A Compendium of Neuropsychological Tests: Administration, Norms and Commentary, 2nd edition Oxford: Oxford University Press, 1998.
24. Purandare N, Burns A, Craig S *et al.* Depressive symptoms in patients with Alzheimer’s disease. *Int J Geriatr Psychiatry* 2001; 16: 960–4.
25. Isella V, Letizia M, Appollonio I. Screening and quantification of depression in mild-to-moderate dementia through the GDS short forms. *Clin Gerontol* 2001; 24: 115–25.
26. Almeida O, Almeida S. Short versions of the Geriatric Depression Scale: a study of their validity for the diagnosis of a major depressive episode according to ICD-10 and DSM-IV. *Int J Geriatr Psychiatry* 1999; 14: 858–65.
27. Aikman G, Oehlert M. Geriatric Depression Scale: long form versus short form. *Clin Gerontol* 1994; 22: 63–70.

28. Chattat R, Ellena L, Cucinotta D *et al.* A study on the validity of different short versions of the Geriatric Depression Scale. *Arch Gerontol Geriatr* 2001; Suppl: 81–6.
29. Montoro I, Izal M. The Geriatric Depression Scale: a review of its development and utility. *Int Psychogeriatr* 1996; 8: 103–12.
30. Vinkers D, Gussekloo J, Stek M *et al.* The 15-item Geriatric Depression Scale (GDS-15) detects changes in depressive

symptoms after a major negative life event. *The Leiden 85-plus study*. *Int J Clin Psychiatry* 2004; 19: 80–4.

31. Cannon B, Thaler T, Roos S. Oral versus written administration of the Geriatric Depression Scale. *Aging Ment Health* 2002; 6: 418–22.

Received 4 August 2009; accepted in revised form  
11 April 2010

*Age and Ageing* 2010; **39**: 445–451  
doi: 10.1093/ageing/afq038  
Published electronically 7 May 2010

© The Author 2010. Published by Oxford University Press on behalf of the British Geriatrics Society.  
All rights reserved. For Permissions, please email: journals.permissions@oxfordjournals.org

# Agreement between self-reported and measured height, weight and body mass index in old age—a longitudinal study with 20 years of follow-up

ANNA K. DAHL<sup>1</sup>, LINDA B. HASSING<sup>2</sup>, ELEONOR I. FRANSSON<sup>1</sup>, NANCY L. PEDERSEN<sup>3</sup>

<sup>1</sup>Institute of Gerontology, School of Health Sciences, Box 1026, Jönköping University, 551 11 Jönköping, Sweden

<sup>2</sup>Department of Psychology, University of Gothenburg, Göteborg, Sweden

<sup>3</sup>Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden

Address correspondence to: A. Dahl. Tel: +46 36 10 13 24; Fax: +46 36 10 11 80. Email: anna.dahl@hhj.hj.se

## Abstract

**Background:** self-reported body mass index (BMI) based on self-reported height and weight is a widely used measure of adiposity in epidemiological research. Knowledge about the accuracy of these measures in late life is scarce.

**Objective:** the study aimed to evaluate the accuracy and changes in accuracy of self-reported height, weight and BMI calculated from self-reported height and weight in late life.

**Design:** a longitudinal population-based study with five times of follow-up was conducted.

**Participants:** seven hundred seventy-four community-living men and women, aged 40–88 at baseline (mean age 63.9), included in The Swedish Adoption/Twin Study of Aging.

**Methods:** participants self-reported their height and weight in a questionnaire, and height and weight were measured by experienced research nurses at an in-person testing five times during a 20-year period. BMI was calculated as weight (kilogramme)/height (metre)<sup>2</sup>.

**Results:** latent growth curve modelling showed an increase in the mean difference between self-reported and measured values over time for height (0.038 cm/year) and BMI (0.016 kg/m<sup>2</sup>/year), but not for weight.

**Conclusions:** there is a very small increase in the mean difference between self-reported and measured BMI with ageing, which probably would not affect the results when self-reported BMI is used as a continuous variable in longitudinal studies.

**Keywords:** *body mass index, height, weight, reliability, elderly*

## Introduction

Older people are at a high risk of functional impairment and morbidity. Body mass index (BMI), calculated as kg/m<sup>2</sup>, might give an estimation of a person's health status. In the prediction and treatment of various diseases, the trajectory of BMI over time might be of greater clinical value than a sin-

gle assessment. In epidemiological studies, BMI values are often based on self-reported weight and height (hence forth called self-reported BMI). The accuracy of self-reported BMI in old age has been evaluated by only a few studies, and to our knowledge, the accuracy of self-reported height, weight and BMI has not been previously studied in a longitudinal trial including older people.

© British Geriatric Society 2010. Published by Oxford University Press. All rights reserved. Copyright of Age & Ageing is the property of British Geriatric Society and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.