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The course of depression in the elderly: a longitudinal community-based study in Australia

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ABSTRACT

Background. We report the outcome of depressive states after 3–4 years in a community sample of the elderly.

Methods. A sample of 1045 persons aged 70+ years in 1990–1 was re-interviewed after 3·6 years.

Results. Mortality (21·7%) and refusal or non-availability (10·4%) were higher in those who initially had had a diagnosis or symptoms of depression. Of those with an ICD-10 depressive episode in 1990–1, 13% retained that diagnosis. Of those who were not depressed initially only 2·5% had become cases. Depression was unrelated to age or apolipoprotein E genotype. The best predictors of the number of depressive symptoms at follow-up was the number at Wave 1, followed by deterioration in health and in activities of daily living, high neuroticism, poor current health, poor social support, low current activity levels and high service use. Depressive symptoms at Wave 1 did not predict subsequent cognitive decline or dementia.

Conclusions. Non-random sample attrition is unavoidable. ICD-10 criteria yield more cases than other systems, while continuous measures of symptoms confer analytical advantages. Risk factors for depressive states in the elderly have been further identified. The prognosis for these states is favourable. At the community level, depressive symptoms do not seem to predict cognitive decline, as they do in referred series.

INTRODUCTION

Research on depression in elderly persons is an important complement to the expanding interest in dementia and cognitive decline. In epidemiological studies, cross-sectional surveys have recently been supplemented by a number of prospective longitudinal enquiries that help to establish what factors are associated with the onset of depressive disorders, their persistence, relapse or remission. Such studies also provide an opportunity to examine changes over time in persons who have been symptomatic but not formal cases of depressive disorders. Such persons are numerically more common in the general population than cases defined by ICD-10 (World Health Organization, 1993) or DSM-III, III-R or IV (American Psychiatric Association, 1980, 1987, 1994). Indeed, it has been suggested that diagnostic criteria such as DSM-III, or its revisions, 'do not capture most depressed older adults in community populations' (Blazer *et al.* 1987). Symptomatic states of depression below the case threshold are important from a public health perspective (Rose, 1993), for care of the elderly in general practice (Bowers *et al.* 1990) and for aetiological research.

In his review of prognostic factors in depressive disorders, Jablensky (1987) specified as a research priority 'the refinement of the diagnostic and prognostic criteria for depressive

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states in the elderly' Outcome includes both an increased mortality (Murphy *et al.* 1988) and the possibility that depression may sometimes be a prodromal feature in Alzheimer-type dementia (Lazarus *et al.* 1987; Heeren, 1988; Meyers & Alexopoulos, 1988; Burns, 1990; Jorm *et al.* 1991*a*).

Based on clinical series of depressive disorders in the elderly, several studies have shown the prognostic value of accurate diagnosis (Roth, 1955), the high likelihood of relapse (Murphy, 1983), and the effectiveness of established treatment in improving prognosis (Baldwin & Jolley, 1986). On a clinical series of elderly patients with depressive disorders, Burvill *et al.* (1989) found little difference between early and late-onset cases on social, demographic and clinical measures, but concluded that neuroticism was an important underlying factor in both categories. They emphasized that findings on such clinical series 'do not necessarily apply to the numerically large and clinically wide range of depressive illness in the community' In a treated series, Brodaty *et al.* (1993) found that the prognosis improved with age, suggesting a more optimistic outlook than has commonly been held.

The longitudinal community studies by Phifer & Murrell (1986), Kaplan *et al.* (1987), Kennedy *et al.* (1990, 1991) Sargeant *et al.* (1990) and Callahan *et al.* (1994) all used the CES-D Scale (Radloff, 1977), rather than standardized clinical interviews, to measure depressive symptoms. These studies have consistently shown an association between failing physical health and the onset or persistence of depressive symptoms. In the community follow-up studies by Ben-Arie *et al.* (1990) in Cape Town, Copeland *et al.* (1992) in Liverpool and Kua *et al.* (1993) in Singapore, standardized clinical interviews were used to assess depression at two points in time, 3 to 5 years apart. These studies all pointed to a poor outcome, with a high probability of death or persistent depression.

Here we report a prospective longitudinal study with the following aims: to investigate the outcome of depressive states after 3–4 years; to identify factors associated with the onset or the persistence of depressive symptoms; and to test the hypothesis that depressive symptoms or disorder in the elderly are a risk factor for subsequent dementia or cognitive decline.

METHOD

The study

In 1990–1, a probability sample of 945 persons aged 70 years and over and living in the community was examined using the Canberra Interview for the Elderly (CIE) (Social Psychiatry Research Unit, 1992; Mackinnon *et al.* 1993). A further sample of 100 persons living in institutions (special hostels for the elderly or nursing homes) was also examined. Details of the methods and findings from the first Wave have been previously reported (Henderson *et al.* 1993, 1994). The CIE is a standardized assessment of depressive symptoms and cognitive performance, suitable for administration by non-clinician interviewers after brief training. It includes an informant interview, and allows persons to be identified as cases of depressive disorder or dementia by ICD-10 and DSM-III-R or IV In addition, a Depression Scale of 28 items from the CIE includes all the symptoms that are specified in the criteria for ICD-10 depressive episode and dysthymia and for DSM-III-R major depressive disorder and dysthymia. The response to each item is set at presence or absence of the item over the previous 2 weeks, and the score is the total number of items present in the previous 2 weeks. In the analysis of the Wave 1 data, this scale was found to have satisfactory psychometric properties (Henderson *et al.* 1993).

In the latter half of 1994, an attempt was made to re-interview those in the cohort who were still alive and accessible in the eastern states of Australia. The same instrument was used, but administered by a different interviewer. The following variables, measured at one or both interviews, were investigated for their association with depression. Most were selected on the basis of an association having been reported in the studies cited above.

(1) *Socio-demographic variables*

The following details were recorded: age, sex, marital status and level of education.

(2) *Psychological health variables*

The data collected were, reports of a past history of depression or nervous breakdown treated by a doctor (scored 0 to 1) and the neuroticism score of the short form of the Eysenck Per-

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sonality Questionnaire – Revised (Eysenck *et al.* 1985) (Wave 1 only). The past history of depression or nervous breakdown was also available from informant reports.

(3) *Measures of cognitive performance*

These measures were, the Mini-Mental State Examination (MMSE) (Folstein *et al.* 1975), the Symbol–Letter Modalities Test (SLMT) (Christensen *et al.* 1994) as a test of mental speed, an Episodic Memory Test (Jorm, 1992) and the National Adult Reading Test (NART) (Nelson, 1982) to estimate pre-morbid cognitive performance.

(4) *Physical health variables*

These were measured on both occasions: subjects were asked whether they had trouble with any of a list of 28 medical conditions such as heart attack, stroke or high blood pressure. The number of conditions was aggregated to form an index of 'medical conditions' Subjects also reported whether, during the previous month, they had suffered from any of 21 symptoms such as headaches, flu, pain in the chest or indigestion. The number of symptoms formed a second index, 'physical symptoms' They also made global self-ratings of current health, and how often they experienced pain. Their systolic and diastolic blood pressure was measured twice during the interview. Analyses were based on both continuous measures and the categories of systolic and diastolic hypertensives, normotensives and hypotensives (Gilmore *et al.* 1995). Disability was measured by a scale for activities of daily living (ADL) covering 8 items (Social Psychiatry Research Unit, 1992). A scale of sensory impairment covered hearing and visual difficulties, as reported by the subject and as observed by the interviewer (Christensen *et al.* 1996). Informant reports were available for ADL and sensory impairment.

(5) *Social support*

This was measured by two scales constructed on the basis of a factor analysis of responses to 13 questions. The first, 'Social support: close friends', reflects whether subjects had people to whom they felt close and from whom they could ask help and support. The second scale, 'Social support: social visit', reflects the amount of visiting to and from family and friends, neighbours and clubs. For both scales, a higher score represents a higher level of available social support.

(6) *Recent bereavement*

This was defined as 'death of anyone close within the previous 6 months'

(7) *Residence*

This was coded as either 'community' or 'institution'

(8) *Inactivity level*

These were measured by asking subjects how often 'these days' they read a newspaper, engaged in physical activity, engaged in hobbies, spent time sitting around without doing very much, spent time in planned activities, and had a daily nap. Each of the six activities was rated on a scale and the items summed. Higher scores indicate a greater level of inactivity.

(9) *The apolipoprotein E genotype*

This was determined at Wave 2 from buccal cells collected on cotton swabs (Henderson *et al.* 1995).

(10) *Service use*

This was reported for community dwelling subjects only, since residents in institutions have little control over their service use. Subjects were asked which of 18 different service providers (e.g. GP, specialist doctor, nurse) they had consulted in the previous 6 months, and the number of categories was taken as a scale of service use.

Statistical analysis

The main analytical strategy for studying the predictors of depression used multiple linear regression with Depression Score at Wave 2 as the dependent variable. Blocks of independent variables were entered hierarchically, with stepwise entry of individual variables within each block. Variables associated with change in the Depression Score between Waves were evaluated using a conditional regression approach (Plewis, 1985). Conditional multiple regression was also used to model changes in dementia scores as a function of depression.

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RESULTS

Response rates

Of the 1045 persons examined in 1990–1, either full or partial interviews were obtained in 1994 for 709 (67·9%). Their mean age was 80·1 (S.D. 4·9), with a range from 73 to 102 years. Of the remainder, 227 (21·7%) were known to have died in the interim, 22 (2·1%) could not be contacted and 87 (8·3%) refused to participate. Rates for refusal or failure to contact were independent of residence at Wave 1 (community or institution), age group and sex. Death rates were higher among the older subjects and among those living in nursing homes or hostels for the elderly.

Cases of depression

At Wave 2, there were 24 cases of ICD-10 depressive episode among the 672 subjects who provided sufficient data for diagnosis, a prevalence of 3·6% in this combined sample of community and institutional residents. Ten of these were cases of mild depressive episode. The mean age of the cases was 79·2 years (S.D. 4·8) and of the non-cases 80·0 years (S.D. 4·9). The age difference was non-significant ($t = 0{\cdot}78$, $P = 0{\cdot}44$). Both DSM-III-R and DSM-IV major depression were diagnosed in the same 7 subjects out of 615 with sufficient data for diagnosis. All of these were also diagnosed as ICD-10 depressive episode, while 17 were cases of ICD-10 depressive episode but not of DSM-IV major depression.

Only four persons out of the 24 diagnosed as depressed by ICD-10 at Wave 2 had also been cases at Wave 1. Table 1 shows the change in depression diagnosis between the first and second Waves for all subjects. Among the 31 persons who had been cases at Wave 1, 13 were no longer cases, three had insufficient data to make a diagnosis, eight had died and three either refused or could not be traced. Remission by Wave 2 was unrelated to whether the person had had contact with a GP at Wave 1. Considering only the 665 subjects who were assigned a diagnostic status at each Wave, there was no significant change in the point prevalence of ICD-10 depressive episode: 2·6% were depressed at Wave 1 and 3·6% were depressed at Wave 2 (McNemar's test, $P = 0{\cdot}47$). On the other hand, subjects diagnosed at Wave 1 were significantly more likely than others to have a diagnosis of depression at Wave 2 ($\chi^2 = 14{\cdot}46$; df = 1; $P = 0{\cdot}000$).

Of the 24 cases, 23 had been in contact with a general practitioner in the previous 6 months, compared with 86% of those not diagnosed with depressive disorder. This difference is not significant (Fisher's exact $P = 0{\cdot}10$). However, significantly more had been treated by a psychiatrist (12·5% compared with 1%, Fisher's exact $P = 0{\cdot}004$) or had seen other specialist doctors (70% *versus* 42%, $\chi^2 = 7{\cdot}8$, $P = 0{\cdot}005$).

Depressive symptoms

At Wave 2, there were 604 subjects with sufficient data to construct the Depression Scale. The mean score was 2·0 (S.D. 2·6) and its distribution was highly skewed. The mean score among those with a diagnosis of depression was 8·6 (S.D. 3·6) and for those who did not receive a diagnosis, 1·8 (S.D. 2·2). None of the subjects with insufficient data for diagnosis had sufficient data for the construction of the depression score. The item frequencies and their rank order,

Table 1. *ICD-10 depressive episode. changes over 3·6 years*

Wave 1 diagnosis (ICD-10)	Wave 2 diagnosis (ICD-10)				
	Not depressed at Wave 2	Depressed at Wave 2	Insufficient data for diagnosis	Died between Waves	No contact or refusal at Wave 2
Not depressed $N = 969$ (92·8%)	628 (64·8%)	20 (2·1%)	26 (2·7%)	198 (20·3%)	97 (10·1%)
Depressed $N = 31$ (3·0%)	13 (42·0%)	4 (12·9%)	3 (9·7%)	8 (25·8%)	3 (9·7%)
Insufficient data to make diagnosis $N = 45$ (4·4%)	7 (15·6%)	0 (0%)	8 (17·8%)	21 (46·7%)	9 (20·0%)
Total $N = 1045$ (100%)	648 (62·0%)	24 (2·3%)	37 (3·5%)	227 (21·7%)	109 (10·4%)

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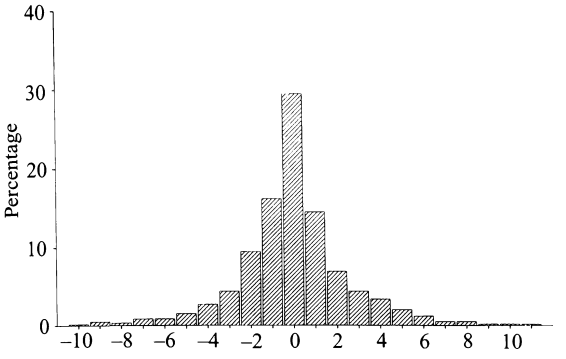

FIG. 1. Frequency distribution of change in Depression Scores over 3·6 years.

factor structure and reliability were all similar to those previously reported in Wave 1. Subjects with scores at both Waves had a mean score at Wave 1 of 2·0 (S.D. 2·4), the same as the mean score at Wave 2. The correlation between the score at Wave 1 and Wave 2 was $r = 0{\cdot}45$. The distribution of the individual change in Depression Scores is shown in Fig. 1. The Wave 1 Depression Scores of those who died between Waves (mean 3·2, S.D. 3·0) or survivors who were not re-interviewed (mean 2·7, S.D. 3·3) were, however, significantly higher than the score of those who were re-interviewed ($F_{(2,916)} = 12{\cdot}45$, $P = 0{\cdot}000$; planned contrasts: $P = 0{\cdot}000$, $P = 0{\cdot}012$).

Factors associated with depression at Wave 2

Table 2 shows the means and standard deviations or the sample proportions at each Wave for each of the variables considered. Data are shown only for those subjects for whom the Depression Score is available from both Waves of the survey.

Cross-sectional associations with depressive symptoms at Wave 2

The cross-sectional association of each variable with the Depression Score at Wave 2 was first investigated. Tests (*t* tests, ANOVA or correlation coefficients as appropriate) reached significance at a level below 0·005 for the following factors. Women had higher Depression Scores than men, and the widowed and divorced tended to have higher scores than married or single subjects. Higher levels of depression were associated with a past history of

Table 2. *Rates and means of variables at Wave 1 and Wave 2*

Variable	Wave 1*	Wave 2*
Sociodemographic variables		
Age (S.D.)	76·5 (4·9)	80·1 (4·9)
Sex (% male)	48·5%	48·5%
Marital status†		
Married	55·6%	48·7%
Widowed	36·5%	42·9%
Others	7·9%	8·4%
Level of education		
Primary	2·8%	2·8%
Secondary	69·5%	69·5%
Tertiary	30·5%	30·5%
Psychological health variables		
Depression Score (S.D.)	1·81 (2·16)	1·86 (2·51)
Past history of depression or nervous breakdown	18·8%	18·0%
Neuroticism (S.D.)	3·05 (2·78)	—
MMSE (S.D.)‡	27·9 (1·8)	27·3 (2·5)
SLMT (S.D.)‡	100·2 (14·5)	97·1 (16·4)
Episodic memory test (S.D.)‡	11·6 (2·4)	12·1 (3·2)
NART (S.D.)	114·1 (8·5)	114·4 (8·4)
Physical health variables		
ADL (S.D.)‡	1·8 (2·5)	2·6 (3·3)
Number of current symptoms (S.D.)	3·3 (2·7)	3·4 (2·7)
Number of medical conditions (S.D.)	2·9 (1·9)	2·9 (1·9)
Blood pressure		
Systolic (S.D.)	145·7 (19·8)	147·0 (22·4)
Diastolic (S.D.)	82·7 (11·9)	81·0 (13·6)
Pain level		
Hardly ever	65·1%	66·1%
Occasionally	15·6%	14·4%
Frequently	11·8%	11·2%
Constantly	7·5%	8·4%
Global health rating‡		
Excellent	19·8%	17·8%
Good	57·0%	55·5%
Fair	20·5%	23·0%
Poor	2·7%	3·8%
Sensory impairment (S.D.)‡	22·5 (7·9)	21·5 (4·5)
Social support, life events and inactivity		
Social support – close friends (S.D.)	914 (0·155)	917 (0·155)
Social support – social visits (S.D.)‡	645 (0·178)	627 (0·178)
Recent bereavement	27·5%	27·5%
Resident of an institution‡	4·1%	10·3%
Inactivity level (S.D.)‡	5·6 (3·7)	6·9 (4·4)
ApoE genotype		
At least one $\epsilon 4$ allele	—	24·2%
At least one $\epsilon 2$ allele	—	12·4%
Services (Community sample only)		
Total services used (S.D.)	2·6 (1·6)	2·5 (1·6)

* Includes only subjects assessed at both Waves ($N = 595$). Some missing data for some variables: N varies between 575 and 595.

† Significant difference between Waves (χ^2, $P = 0{\cdot}022$); $P > 0{\cdot}05$ for all other comparisons.

‡ Significant difference between Waves, $P < 0{\cdot}01$ (paired *t* tests or χ^2 tests).

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depression or nervous breakdown treated by a doctor, more current symptoms or illnesses, lower systolic blood pressure, higher levels of pain, poorer global health, more sensory impairment and more disablement. As at Wave 1, the Depression Score was weakly correlated with the MMSE, the SLMT and the Episodic Memory Test, but not with the NART. Higher Depression Scores were also associated with lower social support, lower levels of activity and living in a nursing home or hostel. But no significant association with depression, even at the 0·05 level, was found for age, education level, diastolic blood pressure, recent bereavement, or the apolipoprotein E genotype. In a multiple linear regression, 35% of the variation in the Wave 2 Depression Score was accounted for by the following variables measured at the same time: report of a past history of depression or nervous breakdown, current physical symptoms, medical conditions, systolic blood pressure, level of inactivity and social support from close friends. No other variable reached statistical significance at a level below 0·05 in the multiple regression.

Prediction of Depression Score at Wave 2

Multiple linear regression was used to determine which variables measured at Wave 1 predict the Depression Score 3·6 years later; and which variables measured at Waves 1 or 2 are associated with change in the Depression Score. The Depression Score at Wave 2 was the dependent variable and independent variables were selected stepwise within each of the successive blocks shown in Table 2. The first series of regressions considered only variables measured at Wave 1 as possible predictors. The next series considered both the Wave 1 and Wave 2 values for each variable. The stepwise procedure was adapted so that if a variable were chosen from either Wave 1 or Wave 2, possible effects of its change between waves were further examined. This was done by first including both the Wave 1 and Wave 2 values in the regression and then, in a separate regression, these were replaced by the change between waves. Although age was not significantly related to depression, it was included with sex in all regressions because these variables were used to define the original sampling strata. They were also correlated with other predictors of interest, and could possibly be significant once age-related variables had been controlled. Dummy variables were formed for the categorical variables of marital status and education. Global health and pain were treated as both categorical (dummy) and continuous measures. Continuous measures were found to be satisfactory and were retained in the final regressions. Polynomial regressions and scatter plots failed to identify any significant non-linear relationships between Depression Score and the predictors, except for ADL: at high levels of disablement, the Depression Score no longer increased. The linear and quadratic terms of ADL score were, therefore, entered into the regressions while all other variables were entered as linear terms.

Prediction of Wave 2 Depression Score from Wave 1 variables

The best Wave 1 predictor of depression at Wave 2 was the Depression Score, accounting for 20% of the variance. After controlling for Depression Score at Wave 1, the only significant predictive psychological and physical health variables measured at Wave 1 were neuroticism, the number of medical conditions and the number of physical symptoms. Together, these variables accounted for 28·4% of the variance in the Depression Score at Wave 2. A higher score in social support (close friends) at Wave 1 was also associated with a lower Depression Score (R^2 change = 0·5%, standardized beta = −0·08, $P = 0·03$). Neither age nor sex was significant. It is notable that none of the other Wave 1 variables, in particular those measuring health (ADL, pain, self-rating of overall health), or a past history of depression, further improved the prediction of the Depression Score 3·6 years later.

Prediction of Wave 2 Depression Score from Wave 1 and Wave 2 variables

The final model for predicting Depression Score at Wave 2, conditional on the Score at Wave 1, is shown in Table 3. When variables from Wave 2 were added to those from Wave 1, the total variance explained reached 44·2%. Depression Score at Wave 1 was again the single most important predictor of depression, explaining 20·5% of the variance. The predictors selected subsequently can then be regarded as variables associated with change in Depression Score.

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Table 3. *Predictors of Depression Score at Wave 2*

Predictor variable*	Beta†	*P*	R^2	R^2 change
Depression score, Wave 1	0·231	0·000	0·182	0·182
Sociodemographic variables				
Age	−0·024	0·528	0·187	0·005
Sex	0·034	0·370		
Psychological health variables				
Neuroticism, Wave 1	0·077	0·056	0·237	0·050
Past history of depression or nervous breakdown, Wave 2	0·136	0·000		
Physical health variables				
ADL, Wave 1	−0·103	0·033	0·411	0·174
ADL, Wave 2	0·283	0·012		
ADL squared, Wave 2	−0·150	0·076		
Number current symptoms, Wave 2	0·117	0·009		
Number medical conditions, Wave 2	0·226	0·000		
Blood pressure: systolic, Wave 2	−0·092	0·010		
Global health rating change between Waves	0·079	0·028		
Sensory impairment change between Waves	−0·064	0·073		
Social support/inactivity				
Social support – friends, Wave 2	−0·095	0·015	0·442	0·031
Social support – social visits, Wave 2	−0·087	0·032		
Activity level, Wave 2	0·095	0·025		
Services (community residents only)				
Total services used, Wave 2	0·135‡	0·001‡	0·438‡	0·015‡

* Only those variables are shown which were included in the final model.

† Standardized beta value, controlling for all other variables in the regression, except service use. Based on community and institutional residents.

‡ Regression limited to community sample only; coefficients for other variables vary only very slightly from those obtained with regression on the full sample.

Those of greatest importance over a period of 3·6 years were physical health variables: both the level of current physical symptoms and changes between Waves in disability, in self-rated general health and in sensory impairment. A deterioration in health between Waves was associated with a higher Depression Score, while improvement in ADL, in self-rated health or sensory impairment was associated with a lower Depression Score. Subjects with more current medical conditions or symptoms at Wave 2 had higher Depression Scores, although the magnitude or direction of any change in these variables over the period of 3·6 years was not significant. Higher levels of neuroticism (at Wave 1) were associated with higher levels of depression at Wave 2 and with an increase in depression since Wave 1.

Is depression a prodrome of cognitive decline?

No cases of ICD-10 depressive episode at Wave 1 were diagnosed as demented by ICD-10 at Wave 2. Change in cognitive scores was modelled in a series of conditional linear regressions where scores on the MMSE, SLMT, Episodic Memory and the NART at Wave 2 were dependent variables, and the respective cognitive scores at Wave 1 were independent variables. Age, sex and the Depression Score at Wave 2 were subsequently entered as independent variables, and finally Depression Score at Wave 1. Depression Score at Wave 1 did not add significantly to any of the regressions. That is, there is no evidence that higher levels of depressive symptoms at Wave 1 can be regarded as predictive of subsequent cognitive decline.

DISCUSSION

This is one of the few longitudinal studies of a community sample of the elderly who have been examined on two occasions with a standardized interview designed to assess cognitive decline, dementia and depression.

Although there was considerable attrition in the cohort due to mortality (21·7% in about 4 years), the loss of 8·3% from refusal and 2·1% from non-availability was more than satisfactory for a cohort study of elderly persons. The case rates and mortality are compared in Table 4 with findings in the three other cohort studies that used clinical interviews. Such comparisons, based on case rates, are necessarily very coarse. They would be greatly assisted if investigators reported scores for depressive symptoms. The overall sample attrition in the present study is very similar to two of the others with a comparable time interval. The outcome for ICD-10 depressive disorder in this study is more favourable than in the UK and Singapore surveys, although numbers are small and differences are not statistically significant. The increased mortality in depressed persons further confirms the consistent findings of Murphy *et al.* (1988), Jorm *et al.* (1991*b*) and Copeland *et al.*

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Table 4. *Outcome of depression in four community follow-up studies*

	South Africa (Ben-Arie *et al.* 1990)	United Kingdom (Copeland *et al.* 1992)	Singapore (Kua *et al.* 1993)	Australia (this study)
Number at Wave 1	150	1070	612	1045
Number re-examined at Wave 2	109 (72·7%)	701 (65·5%)	Follow-up only of those initially depressed	709 (67·9%)
Interval	3·5 years	3 years	5 years	3·6 years
Total mortality	34 (22·7%)	180 (16·8%)	Not stated	227 (21·7%)
Method of diagnosis	PSE/CATEGO Depression	GMS/AGECAT Depression	GMS/AGECAT Depression	CIE/ICD-10 Depressive episode
Depressed at Wave 1	23 (15·3%)	107 (10·0%)	35 (5·7%)	31 (3·0%)
Depressed at Wave 1 and re-examined at Wave 2	20 (87·0%)	82 (76·6%)	31 (88·6%)	20 (64·5%)
Mortality in all those depressed at Wave 1	2 (8·7%)	25 (23·4%)	5 (14·3%)	8 (25·8%)
Depressed at both Wave 1 and Wave 2	9 (45·0%)	33 (40·2)%	10 (30·3%)	4 (20%)
New cases at Wave 2	Not stated	44 (7·1%)	Not stated	20 (3·0%)

(1992). Among the survivors, those who refused or were unable to be re-interviewed at Wave 2 had higher depression scores at Wave 1. This lends support to the suggestion that prevalence and incidence rates are under-estimated in population surveys because of higher depression levels among those who refuse (Kessler *et al.* 1994).

Cases of depression

As in Wave 1, there were substantially more cases of depressive episode by ICD-10 than of major depression by DSM-III-R or IV The latter two DSM criteria showed no difference between each other. Individuals who met the DSM criteria also met criteria for the ICD-10 diagnosis, but 17 of the 24 were cases only of ICD-10 depressive episode. These data support our earlier observation that the ICD-10 criteria are set at a lower threshold than DSM-III-R (Henderson *et al.* 1993). Their relative performance suggests that ICD-10 has the advantage for research on community samples that it identifies both mild and severe states, producing about twice as many cases. Despite this, the low level of both depressive disorders and symptoms in the present study means that the associations reported are not necessarily applicable to elderly persons with more severe disorders who have reached psychiatric services.

Among those who were re-interviewed and provided sufficient data for diagnosis, 3·6% (24/672) were cases of ICD-10 depression. This cannot be taken as an estimate of point prevalence for this population because, unlike the Wave 1 data, the community and institutional samples cannot be meaningfully combined to represent the population at risk. For the development of intervention programmes, it is notable that all of the 24 ICD-10 cases had been seen by a GP in the previous 6 months. No information was obtained on the treatment received, but 3 (12·5%) had been referred to a psychiatrist.

The changes in ICD-10 caseness between Waves have to be cautiously interpreted because of two issues. First, is the possibility of selection effects in those who had died or were not examined at Wave 2. Second, is the likelihood that many who changed status between Waves 1 and 2 had depressive symptoms on the first occasion, but these failed to meet the criteria for being designated a case. That is, they may have had continuing or fluctuating symptoms which reached the threshold for being a case at only one of the two observation points. Only four (13%) of the ICD-10 cases at Wave 1 were again cases at Wave 2; and at least 13 (42%) were no longer cases. This suggests a favourable prognosis for depressive disorders in the general population of elderly persons. The 3·0% of the cohort (20 of 674) who had become new cases by Wave 2 can be considered only an approximate estimate of incidence in this cohort during the interval, because some individuals may have had an onset, but remitted by Wave 2, and reliable information on previous episodes is not available.

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Changes over 3·6 years in variables associated with depression

Between Waves 1 and 2, the changes shown in Table 2 are all in the expected direction for an elderly population. This applies to the increase in widowhood, disablement, sensory impairment and admission to an institution; and to some decline in cognitive performance, global health, social interaction and activity level. The unexpected improvement in performance on Episodic Memory may be attributable to a practice effect. Alongside these changes, it is notable that performance on the NART remained stable. This provides further evidence for the validity of that test as a measure of previous intelligence.

Depression Scale

It is apparent from the analyses reported here that a continuous measure of symptoms yields much more information than the traditional case/non-case dichotomy. This is consistent with the argument proposed by Rose (1992) in support of continuous measures of mental disorders. For the cohort as a whole, the mean scores were strikingly similar at both Waves. At the level of individuals, there was remarkable stability over time, with the majority showing little or no change, and marked symmetry between the numbers whose symptoms increased or decreased (Fig. 1). The higher level of Wave 1 depressive symptoms in those who subsequently died is in accord with the findings noted above.

Cross-sectional associations with depressive symptoms

The cross-sectional data at Wave 2 confirm the associations already established in Wave 1 between current depression and a past history of depression, impaired physical health, a lower level of activity and lower social support (Henderson *et al.* 1993). Although women had higher scores than men, these differences were lost once other variables were accounted for. Having depressive symptoms appears to increase the likelihood of consulting a GP, even after physical health variables had been controlled.

There are three further findings of note. First, because of the unresolved issue on whether rates of depression in the elderly are lower than in younger adults, it is important to note that increasing age was unrelated to depression caseness, the level of depressive symptoms, or change in depressive symptoms. Secondly, the apoE genotype plays no part in the aetiology of late-life depression. An increased risk for cognitive decline and dementia has been shown in the same sample for persons with an $\epsilon 4$ allele (Henderson *et al.* 1995). Thirdly, higher Depression Scores were found to be associated with lower current systolic blood pressure, after controlling for other variables. No relationship of depression with diastolic blood pressure was found. These findings do not replicate those found by Gilmore *et al.* (1995), but they are consistent with those of Pilgrim *et al.* (1992) and Barrett-Connor & Palinkas (1994). We are unable to offer any physiological explanation for the association.

Predictors of change in depressive symptoms between Waves

For a given level of Depression Score at Wave 1, variables associated with a higher score at Wave 2 include indicators of poor psychological health (high neuroticism and reports of a past history of depression), poor physical health (high ADL, more current symptoms and medical conditions, low systolic blood pressure), a worsening of physical health between waves (ADL, self-rating of global health, sensory impairment), low social support, low level of activity, and, for residents in the community, a high level of service use. A lower Wave 2 Depression Score, for a given Wave 1 score, is associated with better psychological and physical health, improvement between waves in health (ADL, self-rated health or sensory impairment), better Wave 2 social support, less Wave 2 inactivity and is accompanied by less service use. These findings confirm others (e.g. Bickel & Cooper, 1994; Kennedy *et al.* 1990, 1991). It has to be acknowledged, however, that reports of deterioration or lower levels in each of these variables could be influenced by current mood. It has been reported that depressed nursing home residents report more pain than the non-depressed, even after controlling for health status (Parmelee *et al.* 1991). Reports of a past history of depression or nervous breakdown given at Wave 2 were associated with higher levels of depression, but the tendency to recall such episodes, if they existed, could be a reflection of current state

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rather than a predictor of it (Jorm & Henderson, 1992). However, measures were also available from informants of a past history of depression, ADL and disablement. These correlated highly with the subjects' own ratings and showed similar associations with depression symptoms, lending support to our findings. Similarly, variables assessed at Wave 1 (depressive symptoms, health and neuroticism) are not contaminated by the subject's state at the time of the second assessment.

It is notable that change in health, as measured at the beginning and end of a 3·6 year period, had little effect on the number of depressive symptoms at the time of the second assessment, the more important correlates being current level of health, social support and activity levels. It is possible, however, that changes in these variables do precede the worsening of depression, but the time lag is considerably less than the 3·6 years we have used. Similarly, increased depression could precede changes in some of the variables, such as increased service use or lower levels of activity. Our data do not allow the investigation of such causal paths.

Lastly, in the present data there is no support for symptoms of depression predicting subsequent cognitive decline or for states of depression being prodromal for dementia, whether the latter be Alzheimer-type or vascular. That association may be observable only in clinical series, in contrast to community samples. Here, our findings are in accord with those of Bickel & Cooper (1994), Dufouil *et al.* (1996) and Sharma *et al.* (1996) which refer to community samples of the elderly.

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