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Alzheimer's Disease, Other Dementias, Depression and Pseudodementia: Prevalence, Incidence and Three-Year Outcome in Liverpool

J. R. M. COPELAND, I. A. DAVIDSON, M. E. DEWEY, C. GILMORE, B. A. LARKIN, C. McWILLIAM, P. A. SAUNDERS', A. SCOTT, V. SHARMA and C. SULLIVAN

A group of 1070 community-living persons aged 65 and over was assessed using the GMS–AGECAT package and other interviews at years 0 and 3. Year 3 interviewers were 'blind' to the findings at year 0, and the prevalence of organic disorders and depression was very similar in both years. According to the results at year 3, minimum and maximum prevalence figures for dementia at year 0 were 2.4% and 3.8% for moderate to severe and 0.4% and 2.4% for mild or early cases, with a best estimate of 3.5% and 0.8%, or 4.3% overall, divided into: senile, Alzheimer's type 3.3%; vascular 0.7%; and alcohol-related 0.3%. The overall incidence of dementia, clinically confirmed by six-year follow-up, was 9.2/1000 per year (Alzheimer type 6.3, vascular 1.9, alcohol related 1.0). Three years later, 72.0% of those with depressive psychosis and 62.3% of those with depressive neurosis were either dead or had some kind of psychiatric illness. Nearly 60% of milder depressive cases (7.2% of the total sample) had either died or developed a chronic mental illness. The outcome of depressive pseudodementias is equivocal so far. Findings at year 3 provide validation of AGECAT computer diagnosis against outcome; organic and depression diagnoses are seen to have important implications for prognosis.

The prevention and treatment of psychiatric illness would be facilitated by its early recognition, and by a knowledge of the prevalence and incidence of the condition. Accurate methods for determining the latter are important steps in the search for risk factors; such information is best obtained from community samples.

In 1987, we reported the prevalence of mental disorders among elderly persons living in Liverpool (Copeland *et al*, 1987*a*). Recently, we have estimated mortality for diagnostic groups (Davidson *et al*, 1988) and examined the risk factors for dementia (Dewey *et al*, 1988) and the alcohol and benzodiazepine consumption by subjects in the sample (Sullivan *et al*, 1988; Saunders *et al*, 1989).

Here we report the follow-up of this sample three years later, reviewing the outcome of illness, updating the estimate of the prevalence of dementia and deriving a figure for overall incidence after age 65. We draw on the six-year follow-up for the clinical confirmation of the incidence cases of dementia identified at year 3.

Method

We have argued, as have others (Wing *et al*, 1974), for the importance of standardised, semi-structured methods for assessment and data collection, and for the need to computerise psychiatric diagnosis in order to provide a thoroughly consistent technique for comparing results between different geographical areas and within the same area at different points in time. During a period of economic retrenchment it is also important to provide a reliable method which can be used by lay personnel.

The Geriatric Mental State (GMS)–AGECAT package was based on the GMS (Copeland *et al*, 1976; Gurland *et al*, 1976), to which the History and Aetiology Schedule (HAS) informant interview (Copeland & Dewey, 1991) and the Social Status Schedule (SSS) were added. AGECAT uses data collected with the GMS. The US–UK Diagnostic Project developed the GMS to meet the need for a standardised method for eliciting symptoms from the older population, parallel to the Present State Examination (PSE) and Present Status Schedule (PSS) for younger adults (Copeland *et al*, 1976; Gurland *et al*, 1976). The GMS was formed by combining a selection of items from the PSS and PSE, simplifying some of the items and ratings, and adding new items to deal with organic symptoms and cognitive behaviour. Subsequently a short version of the GMS developed for community studies and called GMS–A was produced. This schedule was used for the study reported here.

The GMS covers the following sections: orientation, worry, general anxiety, depression, memory, hypochondriasis, tension, somatic dysfunction, phobias, autonomic symptoms, thinking difficulties, slowing, elation, hypomania, grandiosity, loneliness, persecution, guilt, irritability, obsessions, interest, concentration, perceptual distortion, affective response to delusions or hallucinations, medication, drug abuse, alcohol intake, error behaviour, insight, behavioural ratings, affect, movement, lack of social restraint, social speech, communication difficulties, and confidence in data.

The AGECAT computerised diagnostic package (Copeland *et al*, 1986; Dewey & Copeland, 1986) is used to produce diagnoses from GMS data. The package has three stages which are logically distinct and, by a historical accident, correspond to three separate programs.

The preliminary stage of AGECAT takes the responses on the schedule items, and aggregates them together into what we have called symptom components. It does this in one of three ways: sometimes the symptom component consists of a single rating, sometimes the maximum rating is taken from a group of ratings, and finally the sum of a group may be taken. Whatever route is taken, the symptom component is then scaled to have value 0, 1 or 2.

Stage I of the AGECAT system has eight parallel sections. It takes the symptom components and summates them into groups, and then, using a logical decision tree, arrives at the level of confidence. The eight clusters are: organic brain syndrome, schizophrenia and resented paranoia, mania, depression divided into undifferentiated, depressive neurosis, and depressive psychosis, obsessional neurosis, hypochondriasis, phobia, and anxiety.

Stage II uses the levels of confidence output from Stage I; by a hierarchical comparison of these it arrives at a final diagnosis. This is the diagnostic part of AGECAT proper. In the first stage, the level on organic is compared with the level on schizophrenia and resented paranoia, then the output of that is compared with the level on mania. The result of that is compared with the level on depression, using depressive psychosis levels only. At this point if the current result is at level 3 or above the algorithm terminates with the current level as the diagnosis. If not it proceeds in the same manner through obsessional, hypochondriasis, phobia, depression (neurotic and undifferentiated), and anxiety.

In our early studies we found that levels of confidence of three and above correspond to what psychiatrists would usually recognise as a case. Subsequent replication studies (Copeland *et al*, 1988) have confirmed this. This leaves two levels of subcases for each cluster, levels one and two, whose outcome is of interest to follow-up studies. Since each subject is allocated a level of confidence on each of eight clusters, it is possible to examine the outcome of co-morbid states. Of particular interest is the outcome of co-morbid depression and dementia, the so-called 'pseudodementias'.

GMS–AGECAT has been shown to agree well with psychiatrists' diagnoses, with kappa values for organic disorders of 0.86–0.88 and depressive disorders of 0.80–0.86. Comparisons have been made between AGECAT diagnoses and those derived according to DSM–III rules (American Psychiatric Association, 1980) showing good agreement for dementia, and for depression as a whole against major affective disorder and dysthymic mood (Copeland *et al*, 1990).

This paper also reports for the first time results from HAS–AGECAT. Using data from the HAS, this program refines and subdivides the diagnoses produced by GMS–AGECAT, which are based exclusively on mental state information. In this paper HAS–AGECAT is only used in the discussion of organic illness, and the way in which it subdivides dementia is outlined in the appropriate part of the results section.

The sample

In year 0, a random sample, derived from general practitioners' (GP) lists, of 1070 subjects aged 65 and over living at home, in residential accommodation, nursing homes, and hospital wards for less than two years, in the city of Liverpool, was examined by trained interviewers (four psychologists and a senior nurse with geriatric nursing experience) using the community version of the GMS–AGECAT package and a battery of psychological tests (Copeland *et al*, 1987*a*).

A subsample of subjects with organic disorder thought to represent early dementia and a random subsample of well subjects was reinterviewed by psychiatrists approximately one year after the initial interview using the GMS and an early form of the HAS, referred to as the 'year 1 follow-up'.

In the first stage of the year 3 follow-up study all the GPs involved in year 0 were recontacted in order to confirm their consent to continuing the study and to check whether the subjects were still living at the year 0 address.

In the second stage, the subjects received a letter informing them that an interviewer from the study would be calling. The interviewer then visited in person to explain the purpose of the follow-up survey and to arrange a date and time for the interview. Subjects were free to refuse at any stage.

Where subjects were no longer at the year 0 address every effort was made to trace their new address. The help of the Office of Population Censuses and Surveys was enlisted. When the subject was thought to be still resident at the year 0 address, but had failed to answer the door, appointment cards were left, and up to four visits to establish contact were attempted. If this failed, a follow-up letter was sent seeking the subject's cooperation. Only if all these efforts failed was a covert refusal assumed.

Subjects were reinterviewed using the GMSA, HAS, and SSS and the Mini Mental State Examination (MMSE; Folstein *et al*, 1975) by psychiatrists in training but of several years' experience, who were unaware of the findings at year 0. It was not strictly necessary to use psychiatrists as interviewers (although we recommend the HAS be given by a psychiatrist if possible). Psychiatrists were available to do the interviewing as part of research training and were able to provide an intuitive diagnosis on the cases.

Results

Since we reported the prevalence of mental disorders among the elderly in Liverpool, a revised version of GMS–AGECAT has been produced. The data sets from some of the large number of studies now employing the system have been used to reconcile disagreements and correct minor anomalies in the original program. The improved version, 'GMS–AGECAT 1.1', is used in this paper and so the year 0 results differ slightly from those presented earlier (Copeland *et al*, 1987*a*).

In this paper, we were concerned with GMS–AGECAT 'diagnostic' syndrome cases and subcases only, i.e. where AGECAT has selected the organic, depressive or neurotic disorder as the principal diagnosis. We ignored syndrome levels which were co-morbid and secondary to other diagnoses unless specifically stated otherwise. The term 'well' refers to those subjects with no case or subcase levels for any syndrome cluster.

Analysis used χ^2 and McNemar's test with their exact equivalents where appropriate. Odds ratios are presented where appropriate, and they and proportions are followed by 95% confidence intervals (CIs) in parentheses.

Demography and follow-up

In all, 701 subjects were located and reinterviewed. Earlier papers have given the number of reinterviewed as 702, but we now believe that one of these was the twin of the original year 0 interviewee. We have therefore deleted this individual. During the three years, 20.2% of the men and 14.9% of the women had died (a total of 180 individuals), giving an annual mortality rate of 6.7% and 5.0% respectively (see Davidson *et al* (1988) for a more detailed analysis of the relationship between mortality and diagnosis).

Twenty subjects (1.9%) were known to be alive but were not reinterviewed because they had moved to other parts of the country, and five (0.5%) were untraceable. There were 145 (13.6%) who refused interview, and for a further 19 (1.8%) the GP refused access to his patients. It was possible to obtain data with permission for some of the subjects not interviewed from a variety of sources including informants and GPs, or from death certificates. We indicate when these data are used.

Table 1 compares the age distribution of the cohort reinterviewed at year 3 with that for the full sample at year 0 and for those who refused reinterview, were untraced, or not reinterviewed for other reasons. Although the overall proportion of men and women remained the same at year 3 as at year 0, as expected there were now smaller proportions of men in the upper age group, 85 and above.

Table 2 compares prevalence figures for the original random sample with those for the survivors three years later. Because there are some small differences due to the use of GMS–AGECAT 1.1 we set out the new figures for year 0 with the old ones reported by Copeland *et al* (1987*a*) in parentheses, if they no longer agreed. In spite of the fact that the psychiatrists were unaware of the previous findings and the sample has been subject to selective mortality (16.8% having died) the levels are not dissimilar. The mean (95% CI) prevalence for case-level depressive illness as a whole at year 0 was 11.5% (9.6% to 13.6%) and at year 3, 11.0% (8.7% to 13.6%). That for organic disorders at year 0 was 5.0% (3.8% to 6.5%) and at year 3, 4.9% (3.3% to 6.7%).

Outcome of the organic disorders

Of the organic disorders (100, cases and subcases), 18.0% refused to be followed-up, one case was not interviewed and for three the GP refused access. For most of these subjects, additional information is available and some had already been followed-up at year 1.

AGECAT provides six levels of diagnostic confidence for organic disorders, O0–O5, O3 and above representing case levels, and O1 and O2 subcase levels.

At year 3 the bulk of the year 0 organic cases were either still organic cases (26.2%), and hence confirmed as cases of dementia, or dead (57.1%). Of the remaining year 0 organic cases, one had developed depression and the organic symptoms had resolved. Five no longer had any organic level. These latter were almost certainly acute or subacute confusional states at year 0.

We can now examine what happens to those organic subcases (O1 and O2) which have probably been referred to as 'mild dementia' by other studies, or ignored. Taking first level O2 alone, only two subjects refused to be interviewed. Of the remaining 16, seven (43.8%) had died, a rate greater than that for the sample as a whole, and only one had become a syndrome organic case at level O3 (diagnosed as dementia by the psychiatrists). One subject had developed depression and six were now well and must have had some minor confusional state at initial interview. One remained an organic diagnostic subcase. Nevertheless, half AGECAT O2s either died or became demented within three years. Of the subjects with level O1 at year 0, eight refused reinterview. Of the remaining 20, 6 had died and one had developed dementia, while 12 were no longer organic subcases. Based on the year 3 interview data the odds ratio for becoming a case for subcases compared with non-cases was 2.90 (95% CI, 0.46 to 12.45).

Reviewing all the available evidence on deaths and refusals, four organic subcases are known to have developed dementia before year 3.

For those interviewed in year 3 the outcome for cases was worse than for subcases (exact $P = 0.001$) and they were more likely to be confirmed cases (odds ratio = 16.50 (2.84 to 116.58)).

Discussion

The AGECAT diagnoses of cases O3 or above are seen to carry a grave prognosis. The death rate is significantly

Table 1

Age and sex distribution of sample at year 3 ($n = 701$) (men $n = 282$, women $n = 419$) and year 0 ($n = 1070$)

Age: years	Men year 3: *n* (%)	Women year 3: *n* (%)	Total year 3: *n* (%)	Total year 0: %	Refusals/untraced etc: %
65–69	45 (47)	50 (53)	95 (13)	32	26
70–74	117 (45)	144 (55)	261 (37)	29	30
75–79	69 (39)	106 (61)	176 (25)	21	27
80–84	37 (33)	74 (67)	111 (16)	11	12
85–89	11 (24)	35 (76)	46 (7)	5	3
90+	3 (23)	10 (77)	13 (2)	2	2
Total	282 (40)	419 (60)	701 (100)	100	100

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Table 2
Prevalence of diagnostic cases and subcases at year 3 compared with year 0 using AGECAT 1.1 (original AGECAT values in parentheses where they differ from new values)[1]

Levels of confidence of diagnosis	Year 0 ($n=1070$)		Year 3 ($n=701$)	
	n	%	*n*	%
Organic				
O1-O2	46	4.3 (4.8)	15	2.1
O3-O5	54	5.0 (5.2)	34	4.9
Schizophrenic/paranoid				
S1-S2	2	0.2	3	0.4
S3-S5	1	0.1	2	0.3
Depression				
D1-D2	114	10.7	72	10.3
DN3-DN4 (neuroses)	91	8.5 (8.3)	53	7.6
DP3-DP5 (psychoses)	32	3.0	24	3.4
Anxiety				
AN1-AN2	191	17.9 (17.5)	133	19.0
AN3-AN5	12	1.1	6	0.9
Phobia				
PH1-PH2	52	4.9	20	2.9
PH3-PH4	8	0.7	0	0.0
Obsessional				
OB1-OB2	19	1.8	11	1.6
OB3-OB4	1	0.1	1	0.1
Hypochondriases				
HC1-HC2	2	0.2	0	0.0
HC3-HC4	5	0.5	3	0.4
Other psychotic	2	0.2	1	0.1

1. Levels 1-2 = AGECAT subcases, levels 3-5 = AGECAT cases.

greater than that for the sample as a whole even when controlled for age, and it can be shown that the risk of death increases monotonically with increasing AGECAT levels of organic diagnostic confidence (Davidson *et al*, 1988).

Do these subcases represent early cases in the making, chronic states, temporary mood and cognitive changes, or life-long disability? The data reported here suggest that a proportion of them will go on to become cases of dementia, but this is not significantly higher than the figure for those with no organic level. However, the death rate was high, so some may have developed dementia and died; although the overall prognosis is grave it is not one of inevitable progression.

Outcome of depressive disorders

Findings

As can be seen from Table 3, which shows the outcome of diagnostic cases and subcases of depression, the bulk of the depressive psychosis cases (72.0%) and of the depressive neurosis cases (62.3%) were either dead or suffering from a psychiatric illness of one kind or another three years later (including organic cases of dementia). Only two of the psychotic depressives had recovered to the point where they had no depressive level, a further five were now only subcases of depression and one had become a subcase of neurosis. The corresponding figures for neurotic depressives are higher. For all depression cases as a whole, five (4.7%) had developed an organic illness and six (5.6%) a neurotic one. There was no significant difference between the outcomes for depressive psychosis and neurosis ($\chi^2=5.84$, d.f. = 5). The odds ratio for remaining a case comparing depressive psychosis with depressive neurosis was 1.46 (0.51 to 4.30).

If we ignore the subcases (D1 and D2) which form 10.7% of the total year 0 sample, and take only the depressive neurosis cases which still make up 7.2% of the total sample, we find that 41 (59.4%) had died, were still ill or were suffering from some form of mental illness three years later. It would appear that a substantial proportion of these 'milder' community depression cases do not have a good outcome.

Comparing the outcome of subcases with that of cases we find a significant difference ($\chi^2=21.19$, d.f. = 10) attributable to the higher proportion of subcases recovering. The odds ratio for being a case at year 3 is 2.56 (1.24 to 5.41) comparing year 0 cases with subcases.

Discussion

The outcome for depressive cases is poor – their death rate is significantly higher than the rate for the well subjects. There is a non-significant trend for increased level of confidence on the syndrome cluster to be associated with increased risk of mortality, when age is taken into account. There is also a trend in male depressives towards a higher risk of dying over and above age effects (Davidson *et al*, 1988). Among those who survive, over a third are still found to be suffering from a case-level depressive illness at year 3.

By year 3, the AGECAT clinical picture of psychotic depression has changed to one of neurotic depression for a minority of cases or *vice versa*. Some of these subjects could have recovered from the illness at year 0 but subsequently relapsed, developing a different type of depression. There is no evidence of asymmetric change in one direction being more common than change in the other (McNemar's test: exact $P=0.87$).

By contrast with the organic cluster, subcase levels of depression do predict later development of depressive illness.

Both Murphy (1983) and Baldwin & Jolly (1986) found increased rates of death among older persons with depression referred to psychogeriatric services. The increased rate of death in the depressive subjects in this community-based study was also significant, with a clear adverse trend for men.

Of particular interest is the gloomy outcome for depression in the community. Cases of depressive psychosis and depressive neurosis formed 11.5% of the original sample. They are therefore not a tiny minority. The larger proportion is the depressive neuroses, 8.5%. The sceptical view that these cases in the community are probably trivial and transitory is not borne out. We record nearly 11% of depressed subcases in addition to the cases, who can be shown to have substantial numbers of symptoms. However, that more than one-fifth of the cases have recovered also gives the lie to the view that they are chronic conditions intractably associated with ageing or physical illness. The association with physical illness will be examined in a subsequent paper. In the meantime these ill people receive

Table 3
Outcome of diagnostic cases and sub-cases of depressive disorder

AGECAT diagnoses at year 0	AGECAT diagnoses at year 3						
	Dead	DP3–DP5	DN3–DN4	D1–D2	Organic cases	Other cases	Other subcases or well
Cases							
DP3–DP5 (n = 25)	6 (24.0%)	4 (16.0%)	5 (20.0%)	5 (20.0%)	2 (8.0%)	1 (4.0%)	2 (8.0%)
		(60.0%)					
DN3–DN4 (n = 82)	19 (23.2%)	8 (9.8%)	16 (19.5%)	9 (11.0%)	3 (3.7%)	5 (6.1%)	22 (26.8%)
		(52.4%)					
All cases	(23.4%)	(11.2%)	(19.6%)	(13.1%)	(4.7%)	(5.6%)	(22.4%)
		(54.2%)					
Subcases							
D1–D2 (n = 95)	16 (16.8%)	4 (4.2%)	13 (13.7%)	13 (13.7%)	2 (2.1%)	2 (2.1%)	45 (47.4%)
Rest of sample (n = 679)	139 (20.5%)	8 (1.2%)	19 (2.8%)				

Minimum incidence cases = 44 over 3 years.
Year 0. Depressive disorder n = 237; rest of sample n = 833; total sample n = 1070.
Year 3. Depressive disorder, interviewed or dead n = 202; refused interview n = 35; rest of sample interviewed or dead n = 679; refused interview n = 154.

very little treatment. Only 4% were receiving antidepressants at year 0, and we do not know whether these were given in adequate doses. Of the same subjects, 80% were receiving medication for general medical conditions, so the majority were attending GPs' surgeries. It is possible that antidepressants are not the answer to depressive neurosis, but little alternative treatment appears to be on offer. Medication is indicated for most of the subjects with depressive psychosis (3.0%), many of whom were as ill as those found in our hospital studies. Their prognosis is particularly grave. This is an important area for further research (Copeland, 1987).

Outcome of 'pseudodementias'

There were only eight subjects who were organic diagnostic cases with co-morbid and syndrome case-level depression at year 0. Two refused follow-up, three were still organic at year 3, one became a simple case of depression and two died. Of the 14 who were diagnostic cases of organic disorder and had subcase depressive level, four refused follow-up, six remained organic (one at level O2), one became a pure depression, two became well and one died.

Only six (0.6%) of the total sample of 1070 were true pseudodementias, i.e. diagnostic cases of depression with case levels on the organic cluster. Of these, four were followed up, two developed dementia and two remained depressed (subcase). If we look at all subjects who were diagnostic cases of depression and either co-morbid case or subcase levels of organic disorder (30, 2.8%), 21 were reinterviewed; four were known to have died, a rate no greater than for depressive cases as a whole, and five refused reinterview. Of the 21 interviewed, six were still cases and four were subcases of depression; depression had resolved in a further two but they were left with subcase level organic state; five had become demented; one had developed a paranoid illness, one a hypochondriacal illness, and one an anxiety state. Only one had become 'well' without any syndrome level.

Discussion

What will happen to these pseudodementias in the future is uncertain; five out of the 21 interviewed became demented over three years and six still had an organic subcase level (two no longer accounted for by the presence of depression). It remains to be seen whether others develop dementia in subsequent years. In the meantime we can say that AGECAT seems to be making a sensible division between the two principal groups in terms of outcome. Three years is insufficient follow-up, but the fact that a number retained their organic subcase status after the depression had resolved is ominous.

Prevalence and incidence of dementia

Because the HAS was not available at year 0 it was not possible to distinguish dementias from other organic states at that time. However, on the basis of the follow-up it is now possible to re-examine the prevalence of the organic disorders at year 0 and determine a prevalence for dementia. We have seen how the prevalence of organic disorders at year 0 using GMS–AGECAT 1.1 on the mental state data alone is 5.0% (3.8% to 6.5%) of the total sample. Follow-up data are available from the selective follow-up at year 1 and the full sample follow-up at year 3. Here we use both these data sets to re-examine the status of year 0 organic disorders in order to arrive at a more accurate estimate of

Table 4
Prevalence of all dementias at year 0

Prevalence	Moderate to severe		Mild		All types
	n	%	*n*	%	%
Minimum	26	2.4	4	0.4	2.8
Maximum	41	3.8	26	2.4	6.2
Best estimate	37	3.5	8	0.8	4.2

the prevalence of dementia. As a rule we use the year 3 data as the principal source of information, but rely on year 1 data for those not interviewed at year 3.

The interviewing psychiatrists agreed with all AGECAT year 3 cases of dementia (O3 and above). AGECAT identified three of the psychiatrists' dementia cases as O1, AN2 and HC3. Reviewing the available evidence, including written summaries made at the time of interviews, we have designated these as not demented and await the outcome of the year 6 study. One case of dementia at year 3 is unusual because this subject at year 0 had levels O3 D1. By year 1 the subject had developed O1 DP3 and by year 3, O3 D2. The psychiatrist had diagnosed dementia. Pending year 6 data this subject has been included as a case of dementia, possibly vascular.

Moderate to severe dementia

There were 54 organic cases at year 0. Of those reinterviewed at year 3 one was shown to be a case of mental handicap, one was a case of depression only and five had recovered (probably acute confusional states at year 0). We were left with $54-7=47$ (4.4%) possible dementias at this stage.

Of the 24 organic subjects who died before year 3, three are now known to have recovered before death and two to have developed diagnostic depression (nine are known to have died demented) reducing the number of possible dementias to $(47-5)=42$. For the 12 refusals/missing, one is known to have recovered (six known to have developed dementia), thus bringing the figure to 41 or 3.8% of the full sample. This figure represents the maximum prevalence (all potential cases) for moderate to severe dementia (Table 4).

If we take only those subjects confirmed by follow-up to have had dementia (11 interviewed, nine dead and six refused on whom there is good informant or GP confirmation of dementia), we calculate a minimum figure of 26 cases or 2.4% for moderate to severe dementia.

If we assume that for both the ten deaths and five refusals for whom no information is available, corresponding proportions are non-demented and demented (deaths 6.4 and refusals 4.3, 11.0 cases in all), we have 12 confirmed or 16 confirmed or estimated non-dementias, and 26 confirmed or 37 confirmed or estimated dementias. The 37 would represent a best estimate of moderate to severe dementia confirmed or estimated of 3.5% of the total sample of 1070.

Mild dementia

In all, considering the available evidence (including year 1 and year 3 follow-up), there are four organic subcases who are known to have developed dementia. If we assume that the organic subcases whose outcome is not known all developed dementia, we have a maximum figure for mild dementia of 22 cases or 2.4% of the total sample. Taking only those subjects with organic subcase levels at year 0 who are known to have developed dementia (four) we derive a figure of 0.4% as the minimum prevalence of mild dementia. Calculating as above for cases, on the knowledge that one in five of the deaths and refusals of known outcome developed dementia and assuming that those of unknown outcome behaved similarly, a further four cases could be added to make eight, or a best estimate (confirmed or estimated cases) of the prevalence of mild dementia of 0.8% at year 0.

Total prevalence of dementia of all grades of severity

We have therefore, a *minimum* figure for the total prevalence of dementia based only on cases clinically confirmed by follow-up, of 2.8% (26 moderate to severe cases, 2.4%; four mild, 0.4%), a *maximum* figure based on all organic cases who could have developed dementia but whose outcome was unknown of 6.2% (41 moderate to severe possible cases, 3.8%; 22 mild, 2.4%) and a *best estimate* of clinically confirmed cases and estimated cases based on the assumption that subjects who died or refused of unknown outcome progressed to morbidity in the same proportions as those of known outcome, of 4.3% (37 moderate to severe cases, 3.5%; eight mild, 0.8%).

This method of calculating 'confirmed and estimated cases' gives a biased estimate of prevalence as it can only take into account one type of misclassification. It was not possible to include subjects who may have had dementia at year 0 but were misdiagnosed (false negatives) perhaps because their illness had a fluctuating course and who may later have died or refused reinterview and so were missed by the method. All the figures given in this section are therefore lower bounds on the true figures.

Senile (Alzheimer's type) and vascular dementias

Because the HAS was not available at year 0 it is not possible to provide prevalence figures for the different types of dementia present at this time. However, because the prevalence figures at year 3 are so close to those at year 0, it may be of interest to quote these instead, derived from HAS data (Table 5). They are based on a standardised

Table 5
Prevalence and incidence of senile dementia, Alzheimer type (SDAT), multi-infarct or vascular (MID) and alcohol-related (ARD) at year 3, and incidence by age of all dementias

	Prevalence		Incidence	
	n	%	*n*	per 1000
Dementia all types			19	9.2
SDAT	23	3.3	13	6.3
MID	5	0.7	4	1.9
ARD	2	0.3	2	1.0
All dementias				
age 65–74			4	3.8
age 75–84			10	11.8
age 85+			5	28.7

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version of the Hachinski score (Hachinski *et al*, 1975) in which questions and answers have been defined to reduce ambiguities. Most studies to date have not used standardised methods of eliciting and recording data for this scale. A vascular dementia score (VDS) has also been developed for the HAS and both are available with this interview. Taking the 11 cases still dementing at follow-up and the 19 incidence cases (four of the original 23 organic incidence cases were found not to be dementias at year 6 follow-up, see below) (30, 4.3% of the year 3 sample) and using a cut-off of four on the Hachinski score, we obtain 23 cases (3.3%) of senile dementia of the Alzheimer type, five cases (0.7%) of multi-infarct or vascular dementia and two cases (0.3%) of alcohol-related dementia (showing evidence of substantial heavy drinking in the past).

Incidence of the dementias

It is now possible to assess the incidence of the dementias in Liverpool from the results of the year 0 and year 3 follow-up studies. We are also able to draw on the results of the year 6 study for following up the incidence cases and confirming diagnoses. As far as we are aware this is the first study with this advantage. At year 3, 23 cases of organic disorder were found which had not been recognised at case level at year 0. Four of these are now known not to have been dementias (two had recovered by year 6 and were acute confusional states at year 3, another was a case of stroke with aphasia but no clear intellectual impairment and one was a case of Parkinson's disease whose cognitive impairment had resolved by year 6). Removing these four cases leaves 19 incidence cases (at year 6, three were confirmed dementias, 11 were dead and five refused follow-up). Removing the year 0 dementias who survived to year 3 (11) from the total interviewed in that year (701 – 11) to give a population at risk of 690, an incidence figure can be calculated as 19/690 over three years or 9.2/1000 per year (0.92%) (95% CI 0.52% to 1.42%).

The HAS–AGECAT defines 13 of the 19 incidence cases as senile dementia of the Alzheimer type (6.3/1000), four as multi-infarct or vascular dementia (1.9/1000) and two as alcohol-related dementia (1.0/1000). If the alcohol-related diagnoses are ignored and redistributed (to make the figures more comparable with other studies which have ignored this category) the figures for senile dementia become 6.8/1000 and for vascular dementia 2.4/1000.

The incidence for all dementias by age at onset, between the ages of 65 and 74 per thousand of the population at risk is 3.8, for 75–84, 11.8, and for 85 and over, 28.7 (Table 5), approximately trebling every ten years.

Incidence of depression

Of the cases of depression found at year 3, 27 had no previous level of depression at year 0, and 17 depressive cases at year 3 had been only subcases at year 0. The calculation of incidence for depression cannot be very satisfactory in this study because an unknown number of cases will have developed and recovered within the three years. However, a minimum estimate can be given, by taking the 17 subcases who progressed to case level and those 27 not identified on any level at year 0. Thus there were 44 cases from a population at risk of 619 or 23.7/1000 incidence cases per year. The incidence is higher among subcases, odds ratio = 8.44 (3.87 to 18.31).

Discussion

The use of standardised methods

There have been many prevalence studies of dementia and other mental illnesses in community-based elderly persons but few have been done using standardised clinical interviews developed specially for this age group. The US/UK Cross-national project in London and New York (Gurland *et al*, 1983; Copeland *et al*, 1987*b*), the Hobart study (Kay *et al*, 1985) and the Liverpool study (Copeland *et al*, 1987*a*) have each used the Geriatric Mental State in one of its forms and were able to apply AGECAT computer diagnoses. This is the first study to apply these methods to a longitudinal design in elderly persons.

The Epidemiologic Catchment Area surveys used the Mini Mental State Examination together with a standardised interview developed for younger age groups, the Diagnostic Interview Schedule (Robins *et al*, 1981). The Nottingham study (Morgan *et al*, 1987) used the MMSE, and the Melton Mowbray study (Clarke *et al*, 1986) used the CAPE interview for case finding. The Cambridge study (Brayne & Galloway, 1987) used CAMDEX.

One of the purposes of introducing standardised methods for both collecting clinical data and processing diagnosis is to increase the reliability and validity of the diagnosis, and to provide a yardstick for facilitating accurate comparisons between studies in different geographical areas, to compare like with like. However, diagnosis is only one stage in clinical assessment. It has proved its worth by predicting appropriate management and treatment, and in promoting the search for causal factors. It is nevertheless only a convenient metaphor for clinicians and research workers, and as such need not be confined to a simple statement or label. The realisation that it can be informative to qualify that statement has led compilers of international classifications to adopt multiaxial approaches. Thus the computer AGECAT system used here is designed to provide more than a brief diagnostic statement: it can attempt more complicated descriptions than a human examiner can apply consistently, levels of certainty of diagnosis for main and co-morbid states; computed symptom profile scores, for example, can widen the scope of comparisons and generate new questions. When these are linked to levels of social handicap and social network typologies they begin

to provide important information for the planners of services.

Such consistency is also important when comparing the clinical picture of illness at different time periods. It was therefore gratifying to discover that, in spite of the fact that those who remained in the sample after three years could no longer be regarded as a random sample from the population, the prevalence of illness at year 3 was so similar to that found at year 0. It was unlikely that a shift of three years in the age of the population would make much difference to the level of depression. The finding of similar levels, suggesting that new cases balance the number of recoveries and deaths, as expected, confirms this. It was not expected that the level of dementia would vary very much, and it does not. It must be remembered that the interviewers in year 3 did their interviews completely unaware of the findings at year 0.

Although we have shown, in a number of studies totalling over a thousand subjects, that GMS–AGECAT diagnoses agree well with those made by psychiatrists on the same subjects, and with those made using the criteria of DSM–III (Copeland *et al*, 1990), the validity of these diagnoses in terms of outcome needed to be substantiated. Davidson *et al* (1988) and the study reported here go some way towards providing such validation. These studies provide evidence that AGECAT diagnoses for the principal illnesses of dementia and depression do have important prognostic implications, and seem to make a reasonable job of differentiating between the two conditions.

We report here HAS–AGECAT's distinction between Alzheimer's disease and multi-infarct dementia. As yet we have no special way of making this distinction clinically, apart from a standardised version of the Hachinski score (Hachinski *et al*, 1975) and similar measures. We are now testing algorithms for ICD–10 (World Health Organization, 1986) and DSM–III–R (American Psychiatric Association, 1987) diagnoses.

We shall also report later the association between social measures and the levels of confidence of diagnosis, which for depression and dementia seem to equate well with levels of severity in terms of variety and quantity of symptoms. These will give meaning to the AGECAT diagnoses and levels, in terms of service requirement.

Dementia and organic disorders

In the report of the prevalence study (Copeland *et al*, 1987*a*) attention is drawn to the close agreement between the findings for organic disorders and those for dementia reported by Kay *et al* (1964). We have a number of prevalence figures for moderate to severe dementia which can be compared with other studies using the GMS–AGECAT. The prevalence figure for possible dementias, refined by follow-up interviews of cases, of 4.4% is surprisingly similar to the London figure of 4.3% where cases were also followed up (Copeland *et al*, 1987*b*) and after the outcome of some refusals and deaths was known, of 3.8%, again similar to that in a rural sample in Nantwich (Copeland *et al*, 1991) in which it was possible to follow-up refusals and deaths. These figures are not dissimilar to those given by Kay *et al* (1985) using GMS–AGECAT in Hobart of 4.4% for those aged 70–79. The initial figure of 5.0% for organic disorders, although lower than the figure of 7.4% found by Lobo *et al* (1990) in Zaragoza, is not significantly different from it, allowing for differences in the age and sex composition of the samples. Thus, there is some correspondence between prevalence figures when similar methods are used.

Other studies from Britain reporting community samples only, but using different methods, show results which are also not dissimilar; Parsons (1965) in Wales, 4.4%; Broe *et al* (1976) in Scotland, 3.8%; and Morgan *et al* (1987) 4.3% in Nottingham. All the figures quoted in this section lie within our 95% confidence interval for prevalence quoted above.

It now seems increasingly clear that the usual figure for the prevalence of moderate to severe dementia is likely to lie between 3.5% and 4.5%, and that the figure for mild or early dementia is probably very much less, in this study, confirmed by outcome, 0.4% (best estimate 0.8%), making a total prevalence for all types of dementia of 4.8%, or 4.3% as a best estimate, not the 10% sometimes quoted.

Of course, the prevalence of an illness may vary according to a number of factors, even though the incidence may be the same. It is the latter measure, therefore, which is of the most scientific interest. Henderson (1986) has drawn attention to the paucity of incidence studies. He also points to the disagreements which may arise because of the failure to standardise methods. He describes the data from the Lundby study (Hagnell *et al*, 1981, 1983) as the most robust. It is certainly an important study, extending over more than 25 years. Inevitably, it was started before standardised methods were developed. However, the findings of 0.7% and 0.5% for men and women respectively aged between 70 and 79, and 1.9% and 2.5% for those aged over 80 which Henderson calculates from the data, would span our own figure of 0.92% for both sexes aged 65 and over. Another study quoted by Henderson (Mortimer *et al*, 1981) cites an incidence figure very close to our

own, of approximately 1.0% for persons aged 65 and over. Bergmann *et al* (1971) following up the Newcastle study some four years later found a figure for senile dementia of 0.8% and arteriosclerotic dementia of 0.7%.

The figure for the general incidence of all types of dementia in persons aged 65 and over, which begins to emerge, is about 1.0%. Larger studies are required to provide age-specific rates.

The prevalence of confusional states seems low in community studies. This has been our previous experience and that of Kay *et al* (1985). Our figure for this study is 0.5%.

We will be reporting the year 4 follow-up of depressive cases, the year 5 follow-up of both depressive and organic cases, and the full follow-up of the total remaining sample at year 6, in subsequent papers. Meanwhile we will be examining in greater detail the depressive and organic clusters. We shall also be reporting the results of our studies with European collaborators and those participating in the Mental Health Section of the World Health Organization's multisite studies using the same measures. The Medical Research Council's Alpha study of 6000 elderly subjects in Liverpool, now underway, and the MRC/Department of Health multisite studies in the UK using GMS–HAS–AGECAT will provide age-related incidence rates.

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*J. R. M. Copeland, MA, MD, FRCP, FRCPsych, DPM, *Professor and Head, University of Liverpool Department of Psychiatry and the Institute of Human Ageing*; I. A. Davidson, MRCPsych, *Lecturer in Psychiatry, now Consultant Psychiatrist, Royal Liverpool Hospital*; M. E. Dewey, BA, CPsychol, *Lecturer in Psychological Statistics*; C. Gilmore, MRCPsych, *Research Registrar*; B. A Larkin, MRCPsych, *Research Registrar, now Senior Registrar, Manchester Higher Training Scheme*; C. McWilliam, MRCPsych, *Lecturer in Psychiatry, now Senior Lecturer, Psychiatry of Old Age, University of Keele*; P. A. Saunders, MRCPsych, *Senior Research Fellow*; A. Scott, MA, *Experimental Officer, University of Liverpool, Department of Psychiatry, Liverpool*; V. Sharma, MRCPsych, *Consultant Psychiatrist, Walton Hospital, Liverpool*; C. Sullivan, MRCPsych, *Registrar, Liverpool General Psychiatric Rotation Scheme, now Consultant Psychiatrist, Wirral Health Authority*

*Correspondence: *PO Box 147, Liverpool L69 3BX*