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Incidence of dementia in a Munich community sample of the oldest old

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Abstract In a two-wave community study a representative sample of 402 very old people (older than 85 years) was restudied 1 year later. Four instruments in the clinical examination were used for case identification: (a) the Geriatric Mental State Interview (GMS-A); (b) the Structured Interview for the Diagnosis of Dementia (SIDAM); (c) the Global Deterioration Scale (GDS); and (d) the Mini Mental State Examination (MMSE). The clinical examination was performed by the interviewing physician who made a diagnosis according to DMS-III-R. The focus of the present study is on the (true) incidence of dementia in a representative community sample. The establishment of incidence rates is particularly important for dementia because the prevalence of dementia is affected by the length of survival, which is reduced in dementia and with increasing age. The annual incidence rates per 1000 person years on the basis of the SIDAM DSM-III-R were 116.6 for all cases at risk, 113.6 for those aged 85–89 years, 112.5 for those aged 90–94 years and 235.7 for those aged 95 years and older at first assessment (t_1). Incidence rates based on the other methods of assessment are reported. In order to obtain the most meaningful estimate of incidence rates a compound dementia diagnosis was defined. According to this the annual incidence rate per 1000 person years was 144.1 for all persons at risk. The incidence rate tended to be higher in the older-age cohorts: It was 126.2 for those aged 85–89 years, 193.1 for those aged 90–94 years and 295.5 for those aged 95 years and older. In comparison with the literature the incidence rates were high. The results are plausible when the very old age of the sample is taken into account.

Key words Incidence · Dementia · Psychiatric epidemiology · Gerontology

Introduction

Numerous studies on the prevalence of dementia have been published (Jorm et al. 1987; Hofman 1991; Fichter et al. 1995). According to these data the prevalence of dementia rates double with (old) age approximately every 5 years. Prevalence studies are of importance for estimating service needs. However, they have several limitations: They are not only affected by the onset of a disorder, but also by the duration of a disorder and by the survival of cases. Unbiased data on the true incidence can only be achieved on the basis of longitudinal studies in representative community samples. Incidence studies with unselected samples of elderly populations can also be of importance in causal research, and they can provide clues about the effects of risk exposure and its association with the onset of a disorder. As we have shown in a review on the subject (Schröppel et al. 1996) there is a limited but – in recent years – increasing number of incidence studies in the elderly based on representative community samples. The studies published differ considerably in methodology and results (see Discussion). Because population-based studies on the true incidence must be prospective in nature and afford considerable costs and efforts, some studies have attempted to estimate the incidence of dementia indirectly on the basis of hospital records (Larsson et al. 1963; Castrup 1987), psychiatric case registers (Adelstein et al. 1968; Helgason 1977) and retrospective assessment in cross-sectional surveys assessing the prevalence of dementia and its onset during the preceding years (Akesson 1969; Mölsä et al. 1982). Reliance on these sources of data can, however, lead to considerable underestimation of the incidence of dementia. At present, only a limited number of incidence studies on dementia in the community have been carried out with sophisticated methodology. Differences in diagnostic procedures, case identification and sampling are most likely responsible for

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considerable variations in the incidence rates obtained in these studies. For a reliable diagnosis of dementia and its subtypes the use of a structured diagnostic interview appears essential; reliance on a cognitive test only or on questionnaire data is insufficient for reliable diagnostic classification. Even when personal interviews are conducted results can differ considerably due to differences in interview methodology (Fichter et al. 1995).

The aims of the present study were to assess with up to data methodology the (true) incidence of dementia in a representative sample of very old persons living in an urban community (Munich, Germany). In order to achieve this, the sample was assessed longitudinally at two waves 1 year apart. Diagnostic classification and case identification was based on reliable structured interviews which were done by trained research physicians.

Subjects and methods

Details about the methods used in this study have been published in recent reports on the prevalence of dementia and depression (Fichter et al. 1995; Meller et al. 1993); sampling and methods of assessment are therefore reported only briefly herein.

The sample composition for the prevalence wave (t_1) and incidence wave (t_2) is shown in Fig. 1. A random sample of 402 persons aged 85 years and above was drawn from the community register. A total of 358 persons of the random sample (89.1%) were interviewed by psychiatrically trained physicians in the first wave. Of these, 276 (77.1%) were female and the majority of the subjects were widowed (62.9%). Of the 358 persons interviewed in the first wave, 263 were re-interviewed 1 year later (t_2). A total of 23 persons refused to participate in the second wave, 3 could not be located, 2 were located but it was impossible to conduct an interview with them and 67 had died. Possible effects of differential mortality are presented in results.

The following instruments were used to diagnose dementia, cognitive impairment and other mental disorders:

1. The Geriatric Mental State Interview (GSM-A) developed by Copeland and Dewey (1991), a semi-structured psychiatric interview designed for examining elderly subjects, and the History and Aetiology Schedule (HAS) developed by the same authors was used to interview the subject (GSM-A) or, when this was not possible, a key informant (HAS). Usually information from only one key was used. When discrepant answers were given from two or more different sources the issue was discussed with informants until the discrepancies dissolved. The AGECAT algorithm was used for analyses of the GSM-A.
2. The Structured Interview for the Diagnosis of Dementia of the Alzheimer Type, Multi-Infarct Dementia and Dementias of other Aetiology according to ICD-10 and DSM-III-R (SIDAM) developed by Zaudig et al. (1991) was used for diagnosing cognitive impairment and DSM-III-R dementia. Persons with a SIDAM-SISCO score of 34 points or less were defined as demented. The SIDAM includes the Hachinski Scale (Hachinski et al. 1975) and the Modified Ischemic Score (Rosen et al. 1979) and therefore also supplies information about the type of dementia.
3. For the assessment of cognitive impairment the Global Deterioration Scale (GDS) by Reisberg et al. (1982) was also used.
4. Clinical diagnosis of mental disorder according to DMS-III-R (American Psychiatric Association 1987) were made by the psychiatrically trained research physicians after completion of the total interview. The physicians coded the most important two current psychiatric diagnoses. Restricting the number of psychiatric diagnoses could artificially have decreased the rate of minor psychiatric illness. Our data showed that this was not the case because the clinical diagnoses led to higher, and not

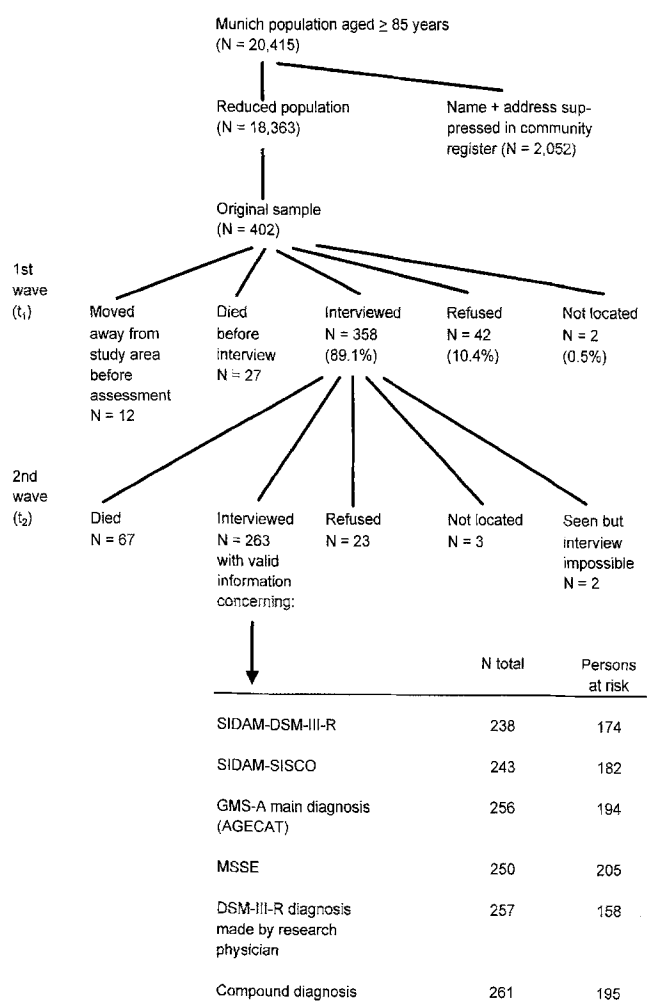


Fig. 1 Population, sample size and attrition in the first wave (t_1) and second wave (t_2)

lower, prevalence rates (Fichter et al. 1995). Subjects were also physically examined and somatic health problems were recorded. Interrater reliability of the GSM-A, SIDAM, GDS and clinical diagnoses and intercorrelations between them have been reported previously (Fichter et al. 1995).

5. A compound dementia diagnosis was calculated, which was based on all available data. The rationale for this compound dementia diagnosis was as follows: Some items could not be assessed due to problems in communication (deafness, blindness, dementia, mutism; see Fig. 1) or due to motor problems (tremor, etc.); therefore, data was not fully complete for each of the diagnostic procedures and instruments used [GMS-A, MMSE (Folstein et al. 1975), SIDAM/SISCO/DSM-III-R, clinical DMS-III-R diagnosis]. In order to reduce missing data effects and to compensate for selective dropout the following compound dementia diagnosis was defined and derived from the diagnostic procedures mentioned (see Note): Because we used three sources of information and because each of these sources (SIDAM, GMS-A, GDS, and research physician's clinical diagnosis) has its merits and limitations, we based our compound diagnosis on the following hierarchy: (a) The compound diagnosis was based on the SIDAM/SISCO (cutoff ≤ 34 points). (b) The SIDAM/SISCO was not used as the sole basis of classification when one of the following exceptions was given:

- (i) The GMS-A and the physician's diagnosis agreed that the SIDAM/SISCO score was falsely negative. Thirteen persons had been scored as cognitively impaired, but not de-

mented, on the basis of the SIDAM/SISCO. (ii) A falsely positive SIDAM/SISCO diagnosis was assumed if the research physicians did not diagnose a dementia according to DSM-III-R ($n = 9$). These persons were not counted as cases of dementia. The reason was that the SIDAM/SISCO exclusively measures the severity of cognitive impairment; it cannot rule out other illnesses such as delirium, tumour, depression, oligophrenia, etc. In order to avoid falsely positive SIDAM/SISCO dementia diagnoses a person had to score ≤ 34 points in the SIDAM/SISCO, and in addition, had to be classified as dementia according to DSM-III-R by the research physician. Prevalence rates based on the clinical DSM-III-R diagnosis were higher than those based on instruments such as the SIDAM or the GMS-A (Fichter et al. 1995). In other words, the diagnostic threshold for the research physicians to classify a person as demented was lower than that of the structured instruments. In seven of these nine cases in which the SIDAM/SISCO diagnosis of dementia was considered to be falsely positive, the GMS diagnosis coincided with the research physician's judgement. The remaining two persons had SIDAM/SISCO scores near the SIDAM/SISCO threshold (33 and 34 points). (iii) When the SIDAM-SISCO diagnosis was not obtainable (see Note); usually because the interviewee was too ill to be interviewed in detail, the GMS-A diagnosis was used for classification ($n = 27$). Nine cases had a GMS-A diagnosis of dementia as well as a research physician's DSM-III-R diagnosis of dementia. (iv) In one case the SIDAM/SISCO and GMS-A diagnosis was not obtainable and the physician's diagnosis was DSM-III-R dementia and it was counted as such. (v) There were five cases which could not be classified, neither on the basis of the interviews (SIDAM/SISCO, GMS-A) nor the physician's judgement. These five cases were not counted as dementia.

According to this compound diagnostic classification 103 persons of the originally interviewed sample ($n = 358$) were classified as having dementia (28.8%), 250 were classified as having no dementia (69.8%) and 5 could not be classified (1.4%). At the second assessment two persons were so ill (both in coma) that they could not be interviewed and a key informant was not obtainable.

Definition and analysis of incidence

For estimating the incidence rate of dementia we counted the number of new illnesses of dementia in the second cross section (t_2)

Table 1 Distribution of persons assessed at t_1 according to birth cohort and gender. Age at prevalence wave (t_1) and gender of the sample

Birth cohort	Age at t_1 (years)	Women		Men		Total	
		N	%	N	%	N	%
1901–1905	85–89	205	74.3	62	75.6	267	74.6
1896–1900	90–94	52	18.8	17	20.7	69	19.3
1891–1895	95–99	19	6.9	3	3.7	22	6.1
Total		276	100.0	82	100.0	358	100.0

Table 2 Age and Mini Mental State Examination (MMSE) scores at t_1 of persons studied at t_2

	N	Age (years) mean \pm SD	Valid No. for age ^a	MMSE mean \pm SD	Valid no. for MMSE
Completed	263	88.5 \pm 2.8	262	23.7 \pm 5.7	256
Died	67	89.6 \pm 3.2	67	19.9 \pm 7.4	59
Refused	23	87.9 \pm 2.2	23	23.3 \pm 4.6	17
Not located	3	88.3 \pm 2.5	3	18.3 \pm 5.5	3
Too ill to study	2	89.7 \pm 1.1	2	No valid MMSE	0

^aIn one case only year of birth but not date of birth known

among the non-demented persons of the first cross section (t_1). The population at risk consisted of persons not diagnosed as demented in the GMS-A, the SIDAM or compound dementia diagnosis at the first wave (t_1). This group at risk includes mentally healthy persons, depressed persons or persons suffering from mental illnesses other than dementia. Persons who had died during the 1-year interval ($n = 67$), who refused to participate at wave 2 or could not be located, and persons with whom it was impossible to conduct the interview were excluded from the analysis of incidence. The time at risk is defined as the time without dementia of the population at risk. For the incidence group it was necessary to estimate the time at risk, because the exact onset of dementia in the 1-year interval was not known. For the estimation we assumed that the beginning of dementia was possible at any time during the follow-up interval and the best estimation for the time without illness for ill people is half the distance between the first and the second interview. Our analyses of incidence were based on the concept of incidence density by Miettinen (1985), also described by Kleinbaum et al. (1982). The formula we use for calculation of the "incidence density" (ID) is as follows: $ID(t_1, t_2) = I/PT$ (I = number of new cases at t_2 ; PT = population time at risk). Because the course of dementia usually is progressive, the incidence density calculated in our study may approximate the rate of first incidence. Incidence was expressed as annual incidence rates per 1000 person years at risk.

Results

Age and gender distributions are given in Table 1. About three quarters of those interviewed at the first wave (t_1) were women. Due to higher mortality with increasing age, the number of persons is lower in the older-age cohorts. The mean ages and MMSE scores of those unavailable for follow-up are given in Table 2; those who died or could not be located had lower MMSE scores than completers. Those who refused to participate at wave 2 did not have reduced MMSE scores at t_1 .

Table 3 shows the average annual incidence rates per 1000 person years by age and gender for different methods of case identification: (a) SIDAM DSM-III-R, (b) SIDAM/SISCO, (c) GMS-A main diagnosis, (d) Mini Mental State Examination (MMSE) ≤ 19 and (e) the compound dementia diagnosis. The overall annual incidence rates per 1000 person years ranged between 71.8 (MMSE ≤ 19) and 144.1 (compound dementia diagnosis). The compound dementia diagnosis is probably the best estimate for the true incidence because it compensates for missing data on the basis of data available on other instruments. Therefore, most of the following data are based on the compound dementia diagnosis. (Data in Table 3 are presented for reasons of comparison with other studies). In all methods of case identification males tended to have a higher annual incidence rate of dementia than females. As shown in the compound dementia diagnosis, which compensates for missing data, inci-

Table 3 Average annual incidence of dementia (according to SIDAM DSM-III-R; see text for definitions)

	New cases of dementia (<i>n</i>)	No. of person years at risk	Annual incidence rates (per 1000 person years)	SE
SIDAM DSM-III-R				
Overall	19	162.9	116.6	25.2
By birth cohort (age at t_1 , years)				
1901–1905 (85–89)	15	132.0	113.6	27.6
1896–1900 (90–94)	3	26.7	112.5	61.2
1891–1895 (95–99)	1	4.2	235.7	206.1
By gender				
Men	6	38.9	154.4	58.0
Women	13	124.0	104.8	27.5
SIDAM SISCO				
Overall	14	173.9	80.5	20.6
By birth cohort (age at t_1 , years)				
1901–1905 (85–89)	11	136.3	80.7	23.3
1896–1900 (90–94)	1	30.8	32.5	32.0
1891–1895 (95–99)	2	6.8	292.9	174.2
By gender				
Men	5	40.0	124.9	52.2
Women	9	133.8	67.3	21.7
GMS-A main diagnosis				
Overall	22	180.7	121.7	24.3
By birth cohort (age at t_1 , years)				
1901–1905 (85–89)	18	145.8	123.4	27.2
1896–1900 (90–94)	4	28.2	142.0	65.8
1891–1895 (95–99)	0	6.7	0.0	0.0
By gender				
Men	9	38.0	236.7	68.9
Women	13	142.7	91.1	24.1
MMSE < 19				
Overall	14	194.9	71.8	18.5
By birth cohort (age at t_1 , years)				
1901–1905 (85–89)	13	149.4	87.0	23.1
1896–1900 (90–94)	0	36.3	0.0	0.0
1891–1895 (95–99)	1	9.2	108.3	102.3
By gender				
Men	5	44.0	113.7	47.9
Women	9	150.9	59.6	19.3
Compound dementia diagnosis				
Overall	26	180.5	144.1	26.1
By birth cohort (age at t_1 , years)				
1901–1905 (85–89)	18	142.6	126.2	27.8
1896–1900 (90–94)	6	31.1	193.1	70.8
1891–1895 (95–99)	2	6.8	295.5	175.4
By gender				
Men	8	42.7	187.4	59.7
Women	18	137.8	130.6	28.7
By birth cohort (age in years) and gender				
Men				
1901–1905 (85–89)	6	34.8	172.3	64.0
1891–1900 (90–94)	2	7.9	254.1	155.2
Women				
1901–1905 (85–89)	12	107.8	111.3	30.3
1891–1900 (90–94)	6	30.0	200.2	73.1

dence rates tended to be higher in (male as well as female) age cohorts with higher age. However, numbers are too low for statistical analyses breaking down the data by age and gender and conclusions must be drawn with caution.

Table 4 shows the average annual incidence rates per 1000 person years for other sociodemographic characteristics. A lower level of school education and low income was associated with higher incidence rates for dementia, whereas lower social classes (IV–V) had marginally higher incidence rates. In agreement with other incidence studies persons living in an institution had much higher dementia incidence rates than persons living in a private home. Those living in a single-person household had the lowest annual incidence rate for dementia.

Of all 26 new cases, 22 could be – clearly – classified on the basis of the Hachinski Score, and 23 according to

Rosen's Ischemic Score. The majority ($n = 19$) had dementia of Alzheimer type and three had multi-infarct- or mixed dementia according to the Hachinski Score and four according to the Rosen Score.

Table 5 shows the association between the average annual incidence rates of dementia per 1000 person years and indicators of cognitive impairment and depression at the first wave (t_1). Signs of cognitive impairment in the SIDAM/SISCO, the MMSE, the GDS, or being classified as GMS-A organic subcase were associated with clearly elevated annual incidence rates for dementia. Cognitive impairment can thus be seen as a risk factor for developing dementia. It is interesting to note, however, that GMS-A depression subcases and depression cases at t_1 are also associated with higher incidence rates for dementia.

Table 4 Average annual incidence of dementia (compound dementia diagnosis) by sociodemographic characteristics. DM Deutschmarks

	New cases of dementia (n)	No. of person years at risk	Annual incidence rates per 1000 person years	SE
Social class				
I–III	14	100.3	139.5	34.6
IV–V	12	80.1	149.8	39.9
Income ^a				
≤ 1.500 DM (lower)	10	61.7	162.2	46.9
> 1.500 DM (higher)	11	91.3	120.5	34.1
School education				
8 years ("Hauptschule")	21	116.5	180.2	35.6
Higher level of education	5	64.0	78.2	33.6
Living arrangement at t_1				
Private home (total)	14	145.3	96.3	24.5
Single-person household ^b	6	88.8	67.6	26.6
Larger household	8	55.4	144.5	47.2
Living in institution	12	35.1	341.4	80.0

^aMissing data for 31 persons at risk

^bMissing data for 1 person at risk

Table 5 Association between average annual incidence of dementia (compound dementia diagnosis) and indicators of cognitive impairments and depression at first assessment (t_1)

	New cases of dementia (n)	No. of person years at risk	Annual incidence rates per 1000 person years	SE
SIDAM/SISCO				
No cognitive impairment	0	73.1	0.0	0.0
Cognitive impairment ^a	23	101.2	227.2	41.6
MMSE				
No Cognitive impairment	1	61.8	16.2	16.0
Cognitive impairment	13	95.3	136.4	35.1
Probable dementia ^b	10	19.7	507.9	112.7
GDS				
Normal	1	78.5	12.7	12.7
Forgetfulness	15	79.5	188.7	43.9
Early confusional	8	18.9	423.5	113.7
Late confusional	2	3.6	557.9	262.3
GMS-A syndromes/ t_1				
No organic	10	137.6	72.7	22.1
Organic subcase ^c	16	42.9	373.2	73.9
No depression	9	89.9	100.1	31.6
Depression subcase	9	41.9	214.7	63.4
Depression case	8	48.6	164.5	53.2

^aIncludes 6 persons at risk with a SIDAM/SISCO diagnosis of dementia

^bIncludes 3 persons at risk with an MMSE diagnosis of dementia

^cIncludes 6 persons at risk with a GMS-A syndrome diagnosis of dementia

Concurrence of dementia with other mental disorders at the beginning: Of the 26 new cases with GMS-A main diagnosis of dementia at wave 2, 7 had a concomitant GMS-A subcase level depression, 4 depressive neuroses and 2 depressive psychoses, 14 were classified as GMS-A subcases of anxiety and 2 as cases of anxiety and 2 were classified as subcases of schizophrenia/paranoia. There were no concomitant cases or subcases of mania, obsessive-compulsive syndrome, hypochondriasis or phobia.

Discussion

Most of the few existing studies on the incidence of dementia have based their calculations on samples aged 60 or 65 years and older; because of age-related mortality very old people are underrepresented in epidemiological studies on the elderly. Thus, in the Swedish Lundby Study (Hagnell et al. 1981; Rorsman et al. 1986) covering the interval between 1947 and 1957, the incidence rates for males per 1000 person years at risk was 48.9 for those aged 80–89 years and 0 for those aged 90 years and above. This finding was most likely due to low sample size in the very old and did not reflect a true decline of the incidence of dementia in very old age. Because of the limited data on the incidence of dementia in the very old, we have chosen a representative sample of very old people (aged 85 years and above) listed in the register of citizens of an urban community. In the following discussion we restrict our comparison of data to prospective epidemiological studies on the incidence of dementia in unselected samples with at least two cross-sectional assessments.

Based on the various modes of assessment used in our study we found the following average annual incidence rates per 1000 person years at risk: (a) 71.8 based on the MMSE ≤ 19 ; (b) 80.5 based on the SIDAM/SISCO; (c) 116.4 based on the SIDAM/DSM-III-R diagnosis; (d) 121.7 based on the GMS-A main diagnosis and (e) 144.1 based on our compound dementia diagnosis. Thus, it does make a big difference what method of case identification is used. Incidence rates obtained by the SIDAM interview (DSM-III-R diagnoses) and the GMS-A interview led to quite similar incidence rates. In order to compensate for selective dropout of subjects in parts of the data, a problem frequently encountered in geriatric studies, we defined a compound diagnosis. Because it compensates missing data artefacts, the rates based on the compound dementia diagnosis (144.1 annual incidence per 1000 person years at risk) were higher than those based on a single instrument (SIDAM, GMS-A, MMSE). It was also higher than incidence rates published in other comparable studies, which we have recently reviewed (Schröppel et al. 1996). In other population-based prospective incidence studies the following average annual incidence rates per 1000 person years have been reported for very old people: (a) an incidence risk in Liverpool (UK) of 28.7 for males and females aged 90 years and above (Copeland et al. 1992); (b) an annual incidence risk of 43 for males and 46 for females aged 85–89 years in Miki Town, Japan (Fuku-

nishi et al. 1991; (c) an incidence rate of 29 per 1000 person years for men and 112 for women in Cambridge, UK (Paykel et al. 1994); (d) an incidence rate in Mannheim, Germany, for those aged 85–89 years of 19.8 for men and 43.1 for women (Bickel and Cooper 1994); (e) an incidence rate of 69 per 1000 person years at risk in the Dutch study (Gussekloo et al. 1995); (f) an incidence rate of 33.8 per 1000 person years per year for men and women aged 80–89 years in Beijing, China (Li et al. 1991); (g) an incidence rate of 39 for persons aged 80 years and above in the Gospel Oak Study, UK (Boothby et al. 1994); (h) an incidence rate of 48.9 for males and 85.2 for females aged 80–89 years in the Lundby study in Sweden, covering the years 1947–1957 (Hagnell et al. 1981); (i) an incidence rate of 23.6 for persons aged 85–89 years (males 35.0 and females 20.1; Bachmann et al. 1993); (j) an incidence of 73.8 for persons aged 90 years and above in Girande, France (age 85–89 years: males 37.2 and females 45.0; age 90+ years: males 71.4 and females 74.4; Letenneur et al. 1994). The population-based studies by Nilsson (1984), Yoshitake et al. (1995) and Boothby et al. (1994) do not report data specifically for persons aged 85 years and above. Other studies do not report data separately for very old persons, or the number of very old persons in the sample is far too low.

Possible reasons for differences in results are differences in methodology and sample composition:

1. The samples differ in age composition. In two of the studies mentioned a sizeable proportion of persons was – different from our study – below 85 years of age.
2. Methods of assessment: some other studies have used different instruments for case identification and different definitions from ours.
3. Using a compound dementia diagnosis as we have done results in larger incidence rates than using a single instrument.

If we compare our rates obtained on the basis of a single instrument (SIDAM/SISCO or the MMSE), our rates are not markedly higher for the particular age group than those reported in other studies. Some other studies on the incidence of dementia in representative community samples must be compared with some caution because they calculated risks rather than rates (Fichter and Rehm 1993; incidence risk of 80.0 for females in Upper Bavaria, Germany, aged 80–89 years). The studies of Jagger et al. (1989) and of Morgan et al. (1993) are based on a sample derived from the general practitioners' list in sections of Great Britain and not on a representative community sample. In some studies, such as that of Fukunishi et al. (1981), the whole sample, rather than the cases at risk, was used as the denominator. In the Icelandic study (Magnusson 1989; Helgason and Magnusson 1989) the annual incidence of severe dementia in 85-year-olds was 41.2 per 1000 per year for men and 46.1 for women. In this study, however, the method of case identification was indirect and partially retrospective using case-register data and physicians' information. Another possible explanation for differences in published true incidence rates in different studies is the fact

that these studies were not all carried out at the same time (period effects). Some were done decades ago when mortality rates were different from those of today. Also, sociocultural or nutritional effects may exist: whereas most studies were carried out in western Europe or North America, some were from Eastern Asia (Japan and China).

Almost all studies on the incidence of dementia in representative samples are in agreement that the incidence of dementia increases with age in the elderly (Hagnell et al. 1981; Nilsson 1984; Jagger et al. 1989; Bickel and Cooper 1994; Boothby et al. 1994; Fichter and Rehm 1993; Letenneur et al. 1994; Morgan et al. 1993 and Copeland et al. 1992). Paykel et al. (1994) concluded from their English incidence study in persons older than 75 years that incidence rates were increasing by the factor 2 over 5-year periods. Our present data confirm this notion and are in line with the hypothesis of an exponential increase in the incidence of dementia in the elderly with age.

The existing data on gender and the incidence of dementia is more controversial. In their review Schröppel et al. (1996) reported some evidence for age-dependent differences in the incidence of dementia for men and women. The majority of studies showed an increased incidence for men up to age 70 or 80 years. For very old persons studies tended to report the opposite: a higher incidence of depression for women (Gussekloo et al. 1995). The sample in our study was comparatively old. We did, however, not find any evidence for a higher incidence of dementia in females in any of the age groups considered. Men (85–89 years, 90+ years) tended to have higher incidence rates.

Bickel and Cooper (1994) and other have reported that residence in long-stay care was associated with high incidence rates for dementia. In a comparison of residents of nursing homes in a German and an English catchment area Weyerer et al. (1995) found that prevalence rates of both, dementia and depression, were particularly high among those residents who were impaired in their activities of daily living. We also found higher incidence rates for persons living in institutions (341.1 per 1000 person years) than for persons living in a private home (96.3 per 1000 person years). However, selective admission or a possible negative-influence institutionalization (lack of cognitive stimulation) cannot be excluded. In agreement with data of others (Paykel et al. 1994), low social class in our study was not associated with a significantly higher incidence of dementia. However, other indicators of a lower standard of living (lower level of school education, lower income group) showed some association with higher incidence of dementia in our study.

In our study the presence of minor cognitive deficits or cognitive impairment as measured with a scale (MMSE) at the initial examination (t_1) was strongly associated with higher incidence rates in the time following first assessment. The more (subdiagnostic) cognitive deficits there were in the MMSE or GDS at first assessment, the higher was the incidence rate in the following period. This finding is in agreement with data reported by Bickel and Cooper (1994) who reported that minor cognitive impairment was a very powerful predictor of dementia in their longitudinal study.

Persons who were cases or subcases of GMS-A depression at first assessment had a relatively high incidence of dementia. In addition, at wave two 11 of 26 new cases of dementia (GMS-A main diagnosis) had a concurrent GMS-A syndrome diagnosis of depression. The association between depression and dementia can be explained in several ways: Depression could (at least in some cases) be an early symptom of dementia. Alternatively, it could increase the risk to develop dementia or it could be a reaction to the realization of cognitive decay in early dementia. Although some studies support findings of depressive symptoms preceding dementia (Jorm et al. 1991, a case control study), others do not (Bickel and Cooper 1994). Weiner et al. (1994) concluded from their data that demented showed a low incidence of depression, but a diagnosis of major depression may herald the subsequent onset of dementia. The issue is too complex to be resolved herein and we therefore refer to recent publications on this issue (Helmchen and Linden 1993; Brodaty et al. 1993; Forsell 1995; Ballard et al. 1996; Devanand et al. 1996) and multicentred projects in progress which provide large data pools.

There are several limitations in our present study. Although we focussed on a very selected (high) age group, the sample is not large enough for a detailed breakdown of the data. Some of the data presented in the text and tables is exploratory and needs to be confirmed by future studies. Especially when data is broken down into several or many subgroups, type-II error (rejecting a hypothesis which is true) may occur.

Another shortcoming of our study is that presently we do not know how many of those who died may have been cases of (incidence of) dementia. If the incidence of dementia among those who died was higher than among survivors, our incidence estimates would be too low. According to our data, persons assessed at t_1 who died in the 1-year interval tended to have higher prevalence rates for dementia (compound dementia diagnosis) than those who survived (dementia without concurrent depression 27.7 vs 21.0%; dementia and depression 16.9 vs 4.2%). Bickel and Cooper (1994) reported an underestimation of the true incidence of dementia by 17% when persons were excluded who had died within a 7-year period. However, the follow-up period in our study was only 1 year, so that this effect of underestimating the incidence of dementia can be assumed to be relatively small.

Several studies on the incidence of dementia are presently conducted in Europe within the EURODEM concerted action on the epidemiology of dementia (Launer et al. 1992) and other continents and their results and the possibility of pooling the data will increase our knowledge on the incidence and aetiology of dementia of different types and may solve the problem of small sample sizes in particular age groups, which most incidence studies on dementia presently have in common.

Note: In 111 of 358 subjects assessed, the data set concerning calculations of the SIDAM-SISCO total score was incomplete. Data were missing mainly as a result of impaired vision or hearing or because of severe tremor. Missing responses were estimated on the

basis of valid responses, but only if not more than 20 responses were missing; also, only one missing subscore per person was estimated. To assess possible errors of estimation, missing data were simulated on the complete data set. The estimation resulted in a slight overestimation of cognitive functioning. The deviation of the estimated sample mean from the empirical mean varied between 0.12 and 1.76 points.

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