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# The Epidemiology of Dementia in North America

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**Summary.** In this brief review, the epidemiology of dementia in North America is examined. The areas covered are prevalence and incidence studies, longitudinal studies, risk factors, distribution of types of dementia, cross-cultural factors and current research programmes.

**Key words:** Epidemiology – Alzheimer's disease – Dementia – History of medicine, twentieth century

## Prevalence and Incidence of Dementia in North America

### *Hospital Studies*

Early epidemiological studies of dementia in North America were based on first admissions to mental hospitals. Elkind [8] examined the rates of first admission with "diseases of the senium" from 1880 to 1925 in private and state mental institutions in the state of Massachusetts. An increase in overall admission rates was described up to 1912, with a levelling off after this date. The changes in mental hospital admission rates for "senile psychoses" were compared for the states of Massachusetts (1920–1933) and New York (1917–1934) by Elkind and Taylor [9]. The trends over time indicated no overall change in Massachusetts, as a slight increase in admissions for "cerebral arteriosclerosis" was balanced by a decrease in the rates for "senile dementia". In New York there was a slight overall increase, due to a more marked increase in "cerebral arteriosclerosis". The observed increase over time was not attributed to a change in the incidence of any specific mental disorder, but rather to changes in classification, better detection and diagnosis, improved institutional care, more effective administrative control and changing public opinion regarding hospitalization. Observed differences in admission rates between the states were attributed to the greater urbanization of New York, and the added difficulty of caring for the elderly at home in cities.

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Further studies of admission rates in New York state over the time period 1910–1936 were described by Malzberg [31] and Landis and Page [30]. These identified marked urban and rural differences in age-standardized rates of admission for all mental disorders, including the senile psychoses, and increasing rates for the diagnostic category of "cerebral arteriosclerosis". Landis and Page considered both findings artefactual, reiterating the susceptibility of admission rates of policy, bed availability, and changes in diagnostic practice. With regard to the differences in rates for "cerebral arteriosclerosis" and "senile dementia", they pointed out that the differential diagnosis of these disorders is often difficult, and that the conditions may frequently co-exist.

At mid-century, Goldhammer and Marshall [18] reported on an examination of the previous 100 years of first admissions to mental hospitals in Massachusetts. From calculations of age-specific admission rates for the major psychoses in the years 1840, 1885 and 1941, they concluded that "... there has been no increase in the overall frequency of psychotic illness during the past 100 years". Rates of first admission for individuals over the age of 60, however, were reported to have increased from 128 per 100000 for the period 1870–1895 to 232 per 100000 for the period 1939–1941. Seventy-five percent of the first admissions after age 60, and 90% of first admissions after age 70, were attributed to "disease of the senium". Goldhammer and Marshall considered the possibility of a "true increase" in the incidence of dementia, but presented evidence for an increasing tendency to hospitalize elderly individuals with moderate cognitive deterioration.

### *Community Studies*

Deficiencies in hospital admission rates as indices of the prevalence and incidence of psychiatric disorders, particularly of disorders with late life onset, were not lost on early investigators. The trend, therefore, has been increasingly toward community survey methods of measuring the prevalence of dementia. Occasionally, community-based studies have attempted to incorporate an evaluation of the sensitivity and specificity of screening instruments [13, 28]. The methodological difficulties inherent in a multi-stage screening and diagnostic protocol neces-

sary for the differential diagnosis of dementia syndromes, however, are formidable [22].

Using criteria which would have defined only severe cases, an interview survey of individuals over age 65 conducted in New York state in 1960 derived a point prevalence of 6.8% for dementia [33]. This survey reconfirmed the inadequacy of institutional data, as the ratio of hospitalized to non-institutional cases was low and declined with age, and there was no correlation between severity and placement. Gruenberg [21] also reported a low correlation between severity of dementia and hospitalization, and noted that the inverse relationship between age and placement suggested most late onset cases (SDAT) would die in the community.

Until recently, however, most estimates of the prevalence of dementia in North America were derived from data from other countries. On the bases of studies conducted in Sweden, Denmark, England and Scotland, and knowledge of the demographic structure of the U.S. population, the prevalence of dementia was estimated to be approximately 50 per 1000 (i.e., 5%) among those aged over 65 [39]. The 1981 publication of an edited volume entitled *The Epidemiology of Dementia* [39] was the hallmark of a growing appreciation of the need for North American data. A few of the subsequent studies are described below.

The multi-site Epidemiologic Catchment Area studies (ECA), sponsored by the National Institute of Health, employed the Mini-Mental State Examination (MMSE) as a measure of cognitive impairment [12, 13]. Across three sites (i.e., Baltimore, New Haven, St. Louis), the prevalence of cognitive impairment ranged narrowly from 4.0% to 5.1% [40]. These prevalence rates varied by race, gender, and education. However, whether these were risk factors is not known [29, 37].

A Canadian study, conducted in Alberta between 1983 and 1986, was similar to the ECA Studies, but included institutions [2]. The prevalence in the community was similar to the U.S. studies but was 68.8% in the institutions. No "severe" cognitive impairment (i.e., MMSE score less than 18 correct) was found in the community. Only 10% of the elderly population were living in institutions, yet less than half of the cognitively impaired remained in the community.

Two other recent U.S. community studies reported higher prevalence rates of cognitive impairment and Alzheimer's disease (DAT) than previously reported. The first, conducted in a Southern California retirement community by Pfeffer and his colleagues [43], reported an age- and sex-adjusted "probable" DAT prevalence of 15.3% in those over 65 and 35.8% in those over 80. A follow-up study was conducted to validate the diagnostic approach used in this study. The second, conducted in East Boston [10], obtained prevalence rates by neurological examination of a stratified sample of those screened with a brief memory test. Estimated prevalence of DAT was 3.0% for those 65–74, 18.7% for those 75–84, and 47.2% of those aged 85 years and over.

In both the California and East Boston study, "mild" or "early" DAT cases were included if they met diagnostic criteria, but the dementias were described separately. The rates for "secondary" dementias, including MID, were lower than previously observed in either institutional or community studies. The East Boston finding was attributed to racial differences, specifically the small proportion of blacks. This is plausible, as cerebrovascular disease is lower in whites than in blacks and orientals [7, 15].

### *Longitudinal Studies*

The few longitudinal studies of dementia in North America have provided data on incidence and risk factors. The Baltimore Longitudinal Study, initiated in 1958 by the Gerontology Research Centre of the National Institute on Aging, was designed to examine the process of aging within a volunteer panel [50]. To examine incident dementia, Sluss et al. [49] selected from this cohort a sample of 519 men born before 1919, with a record of two or more examinations between 1958 and 1978, who were not cognitively impaired at the first examination. They were predominantly white, well-educated and generally healthy, with a median age of 58 years at first examination. From a diagnostic algorithm, 27 cases of probable DAT were identified. Using life-table analyses, age-specific annual incidence rates were estimated. Although the numbers were small, incidence increased from under 0.5% between 60 and 64, to 3.2% between 80 and 90 years. The probability of escaping probable DAT among survivors fell from 0.97 at 70 to 0.71 at 85 years [46]. The feasibility of risk factor analysis was also tested. Although the direct test of creatinine clearance was negative, it enabled the authors to describe the practical issues involved [49].

A New York longitudinal study of volunteers was created to study the incidence of dementia [27]. After 5 years, 56 cases were found in a cohort of 434 volunteers, aged 75–85 at entry, ambulatory and functional on first examination. Of these 56 cases, 32 (57%) met diagnostic criteria for DAT, 15 (27%) met criteria for vascular or "mixed" dementia (MID/MIX), 8 (14%) had other disorders, and 1 was undiagnosed at the time of report. With an accumulated follow-up time of 1585 person years, the observed incidence of dementia was 3.53 per 100 person-years at risk and for DAT alone, approximately 2.0 per 100 person-years. This was intermediate to the rates reported for the oldest age groups in the Baltimore longitudinal study [30, 46]. The only risk factors for DAT in the New York sample were age over 80, female gender and low score on mental state examination at entry; and for MID and "mixed" dementia were diabetes, left ventricular hypertrophy and history of stroke.

## Risk Factors and Dementia

Within North America, as elsewhere, the only undisputed risk factor for DAT is age. The aging of the North American population clearly affects the observed and projected prevalence of dementia syndromes. This is especially so since the age group over 85 is the fastest growing segment of the North American population [3, 34]. While several case-control investigations of DAT have been conducted

with North American samples, these have provided few leads regarding other possible risk factors. Elevated odds ratios have been observed for history of head injury [14, 20, 24], and family history of dementing illness [24, 48]; however, these findings have not been consistent across studies. Familial DAT, perhaps associated with chromosomal disorders (e.g., Down's syndrome), or other central nervous system disorders (e.g., Parkinson's disease), is now considered by some investigators to represent a variant form of DAT [5, 25].

Although exogenous agents may play some role in the genesis of DAT, North American epidemiological studies have provided limited evidence regarding putative agents. In one case-control study an elevated odds ratio was observed for exposures to certain aluminium-containing products [19]; however, the findings were not congruous for all of the products studied (e.g., the odds ratio was elevated for aluminium-containing antiperspirants and *all* antacids, but not antacids restricted to brands containing aluminium). With some revisions in the method of data collection and control subject selection, this study is currently being replicated. The possible risks of population exposure to aluminium were examined by an alternate method in a recent Canadian study [44]. This exploited a "natural" experiment in which Northern Ontario gold miners were exposed to a respirable aluminium dust for daily 10- to 20-min intervals over a period of 35 years. In contrasts of men with differing exposure histories, there was no evidence of an association between exposure and either mortality or diagnosed central nervous system disorder; however, there was evidence of a negative association between exposure and performance on cognitive state examinations. The implication of these findings requires further investigation.

## Distribution of Dementia by Types in North America

DAT and the cerebrovascular disease (MID) type are considered the most common in North America, with DAT being the most prevalent. A difficulty noted by Landis and Page [30] and by Katzman et al. [27] is that DAT, MID and other disease processes may also be coincidental. This complicates both the calculation of estimated rates and the investigation of risk factors.

In regard to other cognitive deficits, the secondary dementias associated with neurosyphilis, nutritional deficiencies and metabolic disorders appear to be uncommon in the general North American population. However, the dementias associated with other disorders of the central nervous system have become a concern. These include brain damage due to ethanol, neurotoxins in the environment, closed head injuries, other central nervous system disorders like idiopathic Parkinson's disease, and acquired immunodeficiency syndrome (AIDS).

The latter has been of growing concern, given the projected magnitude of this manifestation. In 1989, the Centre for Disease Control in Atlanta estimated that one million people in the United States were already infected with the human immunodeficiency virus [4]. Although rates of sero-conversion and the range of clinical expression of the virus remain speculative, neurological complications of patients with AIDS have so far been common. Berger [1] estimated that 40% of AIDS patients display a "sub-cortical" dementia complex during the course of their illness. In some cases, deterioration of mental function, with ensuing dementia, has been described as the only manifestation of the AIDS virus [41].

## Cross-Cultural Differences in Rates and Risk Factors

There has been increasing interest in the variation in prevalence rates for dementia and this has been recently summarized [26]. The racial and cultural heterogeneity of the North American population provides an opportunity to examine this variability. Early this century, Landis and Page [30] looked at differences in mental hospital admission rates for mental illness in urban versus rural communities, between immigrant and American born, and between white, black and Indian groups within the American born. Although there were marked differences between these sub-groups of the population, these were considered a reflection of socio-economic, demographic and cultural differences, rather than indicative of racial or ethnic susceptibility or resistance to mental disorder. Higher rates of admission for blacks, and lower rates of admission of orientals and Indians, relative to the white non-immigrant population, were attributed to socio-cultural differences affecting case detection and hospitalization. While subsequent investigations have, in general, supported this view, there is variability in the distribution of dementia. Among black Americans there appear to be higher rates for cognitive impairment, as detected by population screening measures [47]. If dementia is then diagnosed, cerebrovascular disease is a major contributor. In contrast, among white Americans Alzheimer's disease is the most common diagnosis [10, 43]. Among American Indian and the Inuit peoples, O'Nell [42] noted that, prior to 1960, research had been largely anthropological, and the absence of mental illness was frequently remarked upon [42]. The observed prevalence of the dementias has been remarkably low [23, 45]. Using symptoms to identify cases, only 30 of about 1700 Manitoba Indians over the age of 65, or under 2%, had a probable dementia [23]. The reasons for this low rate of dementia have not been determined. However, it is not due to different demography amongst the Indians [Hendrie et al., unpublished work (1990)].

## Current Programmes of Research in North America

First, it should be mentioned that an attempt has been made to clarify the definition of dementia, and, more specifically, DAT. A "Work Group on the Diagnosis of Alzheimer's Disease" was established by the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the Alzheimer's Disease and Related Disorders Association (ADRDA) in 1983 [32]. The resulting preliminary clinical criteria for the diagnosis of "probable", "possible" and "definite" dementia of Alzheimer's type have since been widely adopted in clinical and research practice on this continent.

Meanwhile, dementia research is growing apace in North America and, in particular, three major research initiatives are underway. These are the multi-centre Consortium to Establish Registries for Alzheimer's Disease (CERAD) in the United States, the Project Investigations de la Maladie d'Alzheimer: Genetique et Epidemiologie (IMAGE) in the Saguenay-lac-Saint-Jean region of Quebec, and the Canadian Study of Health and Aging (CSHA) to be conducted in five regions across Canada.

CERAD was established in 1986, under the sponsorship of the National Institute on Aging. By the end of 1988, 21 sites were members of this consortium and contributing specified data on diagnosed cases of DAT and control subjects for collective analyses [35, 36]. In addition to providing these data, in accordance with a standard protocol, member sites are engaged in a variety of individual projects, ranging from on-going analyses of the large patient data-base accumulated by the Mayo Clinic in Minnesota to community prevalence, clinical and case-control studies.

IMAGE has also taken a registry approach, but within a single geographic region [17]. The Saguenay-lac-Saint-Jean region of Quebec presented a unique opportunity to examine cases of Alzheimer's disease within the context of a population for which genealogical records had been maintained for more than 145 years. Described as a "geographical laboratory", the Saguenay-lac-Saint-Jean region is located north of Quebec City, covers an area of approximately 22500 $km^2$ and has a population of just under 300000 [16]. IMAGE was initiated in 1985 as a multi-disciplinary collaborative project, with the planned research components to include clinical and neuropathological investigations of Alzheimer's disease cases, genealogical and molecular genetics studies of families with case members, and sociogeographic distribution and histories of exposures to putative risk factors of identified cases and control subjects. In keeping with the "multi-matrix" nature of this project, funding of IMAGE has been obtained from a variety of sources.

A Canadian cross-national study of dementia (CSHA) has recently been developed in which the planned objectives are to estimate the prevalence of dementia in five regions of Canada, examine for evidence of risk factors, describe current patterns of care for individuals with dementia and to establish a data-base for future longitudinal studies. This initiative was funded by the National Health Research and Development Programme, and is scheduled to begin data collection in 18 centres across Canada in early 1991.

It can be seen from this brief account of the North American contribution to the epidemiology of dementia that the area is starting to recruit significant attention and research funding.

## References


1. Berger JR (1988) The neurological complications of HIV infection. Acta Neurol Scand 77:40–67
2. Bland RC, Newman SC, Orn H (1988) Prevalence of psychiatric disorders in the elderly in Edmonton. Acta Psychiatr Scand 77 [Suppl 338]:57–63
3. Brody JA (1985) Prospects for an ageing population. Nature 315:463–466
4. Centers for Disease Control (1990) Estimates of HIV prevalence and projected AIDS cases. MMWR 39:110–119
5. Chandra V, Schoenberg BS (1989) Inheritance of Alzheimer's disease: epidemiologic evidence. Neuroepidemiology 8:165–174
6. Chandra V, Kokmen E, Schoenberg BS (1989) Head trauma with loss of consciousness as a risk factor for Alzheimer's disease. Neurology 39:1576–1578
7. Cummings JL (1987) Multi-infact dementia: diagnosis and management. Psychosomatics 28:117–126
8. Elkind H (1927) The epidemiology of mental disease: a preliminary discussion. AM J Psychiatr 6:623–640
9. Elkind H, Taylor M (1936) The alleged increase in the incidence of the major psychoses. Am J Psychiatr 92:817–825
10. Evans DA, Funkenstein HH, Albert MS, Scherr PA, Cook NR, Chown MJ, Hebert LE, Hennekens CH, Taylor JO (1989) Prevalence of Alzheimer's disease in a community population of older persons. JAMA 262:2551–2556
11. Farrer LA, Myers RH, Cupples LS, St George-Hyslop PH, Bird TD, Rossor MN, Mullen MHJ, Polinsky R, Nee L, Heston L, Van Broeckhoven C, Martin J-J, Crapper-McLachlan D, Growdon JH (1990) Transmission and age-at-onset patterns in familial Alzheimer's disease: evidence for heterogeneity. Neurology 40:395–403
12. Folstein MF, Folstein SE, McHugh PR (1975) "Mini-Mental State". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 12:189–193
13. Folstein M, Anthony JC, Parhad L, Duffy B, Gruenberg EM (1985) The meaning of cognitive impairment in the elderly. J Am Geriatr Soc 33:228–235
14. French LR, Schuman LM, Mortimer JA, Hutton JR, Boatman RA, Christians B (1985) A case-control study of dementia of the Alzheimer type. Am J Epidemiol 121(3):414–421
15. Friday G, Lai SM, Alter M, Sobel E, LaRue L, Gil-Peralta M, McCoy RL, Levitt LP, Isack T (1989) Stroke in the Lehigh Valley: racial/ethnic differences. Neurology 39:1165–1168
16. Gauvreau D, Bouchard R, Gauthier S, Mathieu J, Boily C, Cholette A, Robitaille Y, Bouchard P, Bouchard N, Doyon L-P, Gaudreault M, Ouellet A, Dumont M, Kishka P, Fournier C, Nalbantoglu J, Lacoste-Royal G, Gautrin D, Froda S, de Braekeleer M, Bouchard G, Mortimer J (1988) The IMAGE project: a geographical laboratory for the integration of multidisciplinary data: In: Sinet PM, Lamour Y, Christen Y (eds) Genetics and Alzheimer disease. Springer, Berlin Heidelberg New York, pp 40–50
17. Grauvreau D, DeBraekeleer M, Bouchard R, Gauthier S, Mathieu J, Boily C, Cholette A, Robitaille Y, Bouchard P, Bouchard N, Doyon L-P, Gaudreault M, Ouellet A, Dumont M, Kishka P, Fournier C, Nalbantoglu J, Lacoste-Royal G, Gautrin D, Froda S, Bouchard G (1989) The IMAGE project: a multimatrix model for the study of Alzheimer disease. In: Blass JP, Miner G, Miner LA, Richter RN, Valentine JL (eds) Familial Alzheimer's disease: molecular genetics, clinical aspects and societal prospects. Dekker, New York, pp 331–365
18. Goldhammer H, Marshall A (1949) Psychosis and civilization. The Free Press, Glencoe, Illinois
19. Graves AB, White E, Koepsell TD, Reifler BV, Van Belle G, Larson EB (1990) The association between alumium containing products and Alzheimer's disease. J Clin Epidemiol 43:35–44
20. Graves AB, White E, Koepsell TD, Reifler BV, Van Belle G, Larson EB, Raskind M (1990) The association between head trauma and Alzheimer's disease. Am J Epidemiol 131:491–501
21. Gruenberg EM (1978) Epidemiology of senile dementia. In: Schoenberg BS (ed) Neurological epidemiology: principles and clinical applications. Raven Press, New York, pp 437–455
22. Henderson AS, Jorm A (1987) Is case-ascertainment of Alzheimer's disease in field surveys practicable? Psychol Med 17:549–555

23. Hendrie HC, Hall KS, Austrom MC (1989) Some thoughts on the cross cultural prevalence of dementia. In: Carstensen LL, Neale JM (eds) Mechanisms of psychological influence on physical health with special attention to the elderly. Plenum Press, New York, pp 161–184
24. Heyman A, Wilkinson WE, Stafford JA, Helms MF, Sigmon AH, Weinberg T (1984) Alzheimer's disease: a study of epidemiological aspects. Ann Neurol 15:335–341
25. Hofman A, Schulte W, Tanja TA, van Duijn CM, Haaxma R, Lameris AJ, Otten VM, Saan RJ (1989) History of dementia and Parkinson's disease in 1st-degree relatives of patients with Alzheimer's disease. Neurology 39:1589–1592
26. Jorm AF, Korten AE, Henderson AS (1987) The prevalence of dementia: a quantitative integration of the literature. Acta Psychiatr Scand 76:465–479
27. Katzman R, Aronson M, Fuld P, Kawas C, Brown T, Mogenstern H, Frishman W, Gidez L, Eder H, Ooi WL (1989) Development of dementing illnesses in an 80-year-old volunteer cohort. Ann Neurol 25:317–324
28. Klein LE, Roca RP, McArthur J, Vogelsang G, Klein GB, Kirby SM, Folstein M (1985) Diagnosing dementia: Univariate and multivariate analyses of the Mental Status Examination. J Am Geriatr Soc 33:483–488
29. Kramer M, German PS, Anthony JC, Von Korff M, Skinner EA (1985) Patterns of mental disorders among the elderly residents of eastern Baltimore. J Am Geriatr Soc 33:236–245
30. Landis C, Page PD (1938) Modern society and mental disease. Farrar and Rinehart, New York
31. Malzberg B (1935) Prevalence of mental diseases among the urband and rural populations of New York state. Psychiatr Q 9:55–87
32. McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM (1984) Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force of Alzheimer's disease. Neurology 34:939–944
33. Mental Health Research Unit, New York State Department of Mental Hygiene (1961) A Mental Health Survey of Older People. State Hospitals Press, Ulica, New York
34. Monroe RT (1953) The effect of aging of population on general health problems. N Engl J Med 249:277–285
35. Morris JC, Heyman A, Fillenbaum G, Clark C (1989) Consortium to Establish a Registry for Alzheimer's disease (CERAD). Neurology 39:1159–1165
36. Morris JC, Reyman A, Fillenbaum G, Clark C (1990) Longitudinal assessment of probable Alzheimer's disease: 2-year follow-up data from the Consortium to Establish a Registry for Alzheimer's disease: 2-year follow-up data from the Consortium to Establish a Registry for Alzheimer's disease (CERAD). Neurology 40 [Suppl 1]:450
37. Mortimer JA (1988) Do psychosocial risk factors contribute to Alzheimer's disease? In: Henderson AS, Henderson JH (eds) Etiology of dementia of Alzheimer's type. John Wiley & Sons, New York, pp 39–52
38. Mortimer JA, Schuman LM (eds) (1981) The epidemiology of dementia. Oxford University Press, New York
39. Mortimer JA, Schuman LM, French LR (1981) Epidemiology of dementing illness. In: Mortimer JA, Schuman LM (eds) The epidemiology of dementia. Oxford University Press, New York, pp 3–23
40. Myers JK, Weissman MM, Tischler GL, et al (1985) Six month prevalence of psychiatric disorders in three communities. Arch Gen Psychiatr 41:959–967
41. Navia BA, Price RW (1987) The acquired immunodeficiency syndrome dementia complex as the presenting or sole manifestation of human immunodeficiency virus infection. Arch Neurol 44:65–69
42. O'Nell TD (1989) Psychiatric investigations among American Indians and Alaska Natives: a critical review. Cult Med Psychiatry 13:51–89
43. Pfeffer RI, Afifi AA, Chance JM (1987) Prevalence of Alzheimer's disease in a retirement community. Am J Epidemiol 125:420–435
44. Rifat SL, Eastwood MR, McLachian DRC, Corey PN (1990) Effect of exposure of miners to aluminum powder. Lancet 336: 1162–1165
45. Roy C, Choudhuri A, Irvine D (1970) The prevalence of mental disordes among Saskatchewan Indians. J Cross-cult Psychol 1:383–392
46. Sayetta RB (1986) Rates of senile dementia-Alzheimer's type in the Baltimore longitudinal study. J Chronic Dis 39:271–286
47. Schoenberg BS, Anderson DW, Haerer AF (1985) Severe dementia: prevalence and clinical features in a biracial US population. Arch Neurol 42:740–743
48. Shalat SL, Seltzer B, Pidcock C, Baker EL (1987) Risk factors for Alzheimer's disease: a case-control study. Neurology 37: 1630–1633
49. Sluss TK, Gruenberg EM, Kramer M (1981) The use of longitudinal studies in the investigation of risk factors for senile dementia-Alzheimer type. In: Mortimer JA, Schuman LM (eds) The epidemiology of dementia. Oxford University Press, New York, pp 132–154
50. Stone JL, Norris AH (1966) Activities and attitudes of participants in the Baltimore Longitudinal Study. J Gerontol 21: 575–580