LETTERS TO THE EDITOR


Population-based data on Parkinson’s disease reported by Mayeux et al. (1, 2) suggest continually increasing age-specific Parkinson’s disease incidence rates, even for very old persons (persons aged >84 years), and also identify incidence differences by race/ethnic affiliation. In a 30-year Parkinson’s disease incidence study in a cohort of 8,006 American men of Japanese or Okinawan ancestry (3, 4), we found annual incidence rates nearly identical to the total rates of Mayeux et al. in the age ranges of 65–74 years (55.6 per 100,000 person-years in our study vs. 54.2 in theirs) and 75–84 years (132.4 vs. 136.6 (1, 2)). In men aged >84 years, however, Mayeux et al. report an incidence rate higher by more than fourfold (77.8 vs. 353.8), attributable disproportionately to incident cases in black and Hispanic men.

Incidence determination in the very old is important to understanding the etiology of Parkinson’s disease. Continually rising incidence in later life would be most consistent with a maturational model of Parkinson’s disease, whereas an age-specific incidence peak, followed by declining incidence in older persons, would suggest an induction or incubation period (4). A few studies, including that of Mayeux et al., have not shown an incidence peak, but many other studies from the United States, Europe, Scandinavia, and the Pacific have detected a peak at around age 75 years. Parkinson’s disease incidence determination in the very old is subject to many potential errors. It is often difficult to define the population denominator in the presence of in- and out-migration that reflects, in part, changes in residence due to age-associated infirmity. More problematic is that the onset of Parkinson’s disease is insidious and easily obscured by other conditions, with the diagnosis sometimes being confused with clinically similar conditions (e.g., vascular parkinsonism). With current knowledge, Parkinson’s disease cannot be considered an etiopathologic entity and is probably subject to extreme competing pressures of over- and underdiagnosis exacerbated by advancing age. We believe that these cautions apply to our own data, to those of Mayeux et al., and to those of other investigators who have studied Parkinson’s disease occurrence.

A second observation from our own cohort data is that American men of Japanese/Okinawan ancestry experienced age-specific incidence rates similar to those of white or black men in Finland, Iceland, Rochester, Minnesota, and—with the exception of the oldest ages—Manhattan, New York (1, 2, 4). This may not strongly favor genetic differences in the risk of Parkinson’s disease. Here again, however, definitive answers are lacking because of concerns about population denominators and completeness/accuracy of Parkinson’s disease detection in all of the various studies. Mayeux et al. report a significantly higher Parkinson’s disease incidence in black men than in white or Hispanic men or women, or in black women, with incidence differences most pronounced in the oldest age groups. For instance, in the 65- to 74-year-old age range, the Parkinson’s disease incidence rates for men of Japanese/Okinawan ancestry are virtually identical to those of white and black men reported by Mayeux et al. (1, 2). For the ages of 75–84 years, the rates remain within a twofold range. However, the rates diverge from three- to 12-fold at ages >85 years. The argument for racial/ethnic differences in men thus rests strongly on case detection among the most elderly men. There may be several possible explanations for the apparent incidence differences. Differential diagnostic accuracy could be consistent with the findings by Mayeux et al. that black men had higher Parkinson’s disease incidence rates, but lower prevalence rates, than other men, to a degree not easily explained by differential mortality. In- or out-migration differences might also produce such an incidence disparity; for example, the 3-year residency requirement for enrollment, undoubtedly longer than the interval between symptom onset and diagnosis for many subjects, might have allowed non-black persons with “prediagnostic” Parkinson’s disease to preferentially “escape” from the catchment area before they could be diagnosed. Diagnostic delay in black persons could also explain the differences. Mayeux et al. report that the mean age of symptom onset in incident cases (75.2 years) was nearly 10 years older than for prevalent cases (65.7 years). Such incident cases could have been experiencing “catchup” diagnoses; some of the alternative explanations for the mean age differences (e.g., that extraordinarily high Parkinson’s disease mortality affects only older patients or that the epidemiology of Parkinson’s disease has changed dramatically in the past few years) seem less likely to us. These concerns underscore the difficulties in characterizing the basic descriptive epidemiology of Parkinson’s disease.

REFERENCES

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