Thirty-Year Projections for Deaths From Ischemic Stroke in the United States

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Background and Purpose—The age adjustment of stroke mortality rates may obscure the impact of population changes on the total burden of disease. Deaths from ischemic stroke may rise unless future declines in stroke death rates offset the projected growth in high-risk populations.

Methods—Using data on ischemic stroke mortality from the National Center of Health Statistics for 1979 to 1998, we fit a logistic model to predict changes in stroke death rates as a function of time for each of 42 sex-race-age groups. Using population projections from the US Census Bureau, we then calculated the expected number of deaths in the United States from ischemic stroke over the next 30 years on the basis of age, sex, and race.

Results—Models generally fit historical data well (median $R^2=0.81$; interquartile range, 0.43 to 0.97) and consistently predicted small declines in future death rates. The total predicted number of stroke deaths increased by 98% from 139 000 in 2002 to 275 000 in 2032, whereas the total US population was projected to increase by only 27% in the same period. The largest percentage increases in stroke deaths were predicted to occur in blacks (134%) and nonwhite, nonblack races (221%).

Conclusions—If recent trends in ischemic stroke mortality continue, increases in US stroke deaths will outpace overall population growth, with a doubling in deaths over the next 30 years. (Stroke. 2003;34:2109-2113.)

Key Words: epidemiology ■ mortality ■ racial differences ■ stroke, ischemic
We calculated future population-specific mortality rates for each of 42 demographic groups by fitting the following 4-parameter nonlinear function to historical mortality rates using least-squares regression:

\[
SMR(\tau; \alpha, \gamma, \beta, \beta_0) = \alpha + \gamma \left( \frac{1}{1 + e^{\beta_0 + \beta \tau}} \right)
\]

In this equation, the future stroke mortality rate (SMR) is modeled as an S-shaped curvilinear function of calendar year (\(\tau\)) and 4 parameters that represent a theoretical asymptote (\(\alpha\)) to which stroke mortality rates are approaching, the difference (\(\gamma\)) between the asymptote and the higher historical rate, and a measure of the width (\(\beta\)) and steepness (\(\beta_0\)) of the declining slope of the curve. This model acknowledges that there must be a rate below which further reduction in the stroke mortality rate is impossible. If the logistic model did not converge using the historical mortality rates, the raw data were smoothed with a kernel-like estimator. Model fit was based on minimization of the residual sum of squares and was calculated as a correlation coefficient. To calculate total stroke mortality and crude mortality rates in future years, we used population projections from the US Census Bureau. Age-adjusted stroke mortality rates were calculated by the direct method with the year 2000 population standard. Crude mortality rates were calculated as the total number of deaths divided by the total number of persons in the population. All calculations were performed with the Stata statistical package (version 7.0, Stata Corp).

Results

Logistic models generally fit the historical data closely (median \(R^2=0.81\); interquartile range, 0.43 to 0.97). In the 10 age-race-sex groups that accounted for >90% of the ischemic stroke mortality between the years 1978 and 1998, the median \(R^2\) of the logistic models was 0.98. In 2 small population strata (female, other race, and age 1 to 20 and 45 to 54 years) accounting for <0.05% of total stroke mortality between the years 1978 and 1998, the logistic models did not converge using the crude mortality data; therefore, a smoothing function was applied before the calculations.

Models generally predicted small future declines in age-adjusted stroke mortality rates (the Table). Between 2002 and 2032, the overall age-adjusted mortality rate is projected to decline \(\sim6\)% from 47.6 to 44.7 deaths per 100 000 person-years. During this period, age-adjusted mortality rates for men are projected to decrease by 5.1% and for women by 7.3%.

Nonwhite, nonblack races are predicted to experience greater declines in age-adjusted mortality rates (11.7%) than blacks (6.4%) or whites (1.5%) over the next 30 years. In contrast, crude mortality rates are predicted to increase substantially for all subgroups over the next 30 years, with the greatest increases occurring in men (68.6%) and blacks (67.4%).

Between 2002 and 2032, the total US population is projected to grow by 27% from 280 million to 356 million. During this time, we project total annual stroke deaths to increase by 98% from 139 000 to 275 000 (Figure 1). The greatest percentage increases are projected to occur in minority populations, in which the total number of deaths in blacks is projected to increase by 134% and total number of deaths in nonwhite, nonblack groups is projected to grow by 221% (the Table). As a percentage of total stroke mortality, the greatest increases in deaths are predicted to occur in men and blacks. A projected 12.6% of deaths from stroke will occur in blacks in 2032 compared with 10.6% in 2002 (Figure 2). The proportion of total stroke deaths occurring in men is projected to increase from 38% in 2002 to 41.4% in 2032 (Figure 3). Although the percentage of total mortality occurring in individuals \(\geq65\) years of age in 2032 is projected to increase slightly from 93% to 94%, our models predict that the mean age at stroke death will remain virtually unchanged at 82 years.

![Figure 1. Historical and projected total stroke deaths per year in the United States, 1979 to 2032. Projected values are the product of future age-race-sex-specific mortality rates and US Census Bureau projections.](https://stroke.ahajournals.org/issue/September/2003/Stroke2003-09-20/Changes-in-Ischemic-Stroke-Mortality-2002-to-2032 Dx1.png)
Mortality from ischemic stroke has declined in most countries over the last several decades. In the United States, the rate of decline of stroke mortality leveled off in the 1990s. The pattern of decline followed by the suggestion of a plateau has applied across most US demographic groups despite large initial differences in stroke mortality rates. Furthermore, levels that stroke mortality rates appear to be approaching leave significant disparities across populations defined by race, sex, and geography. Prior studies have suggested that different demographic groups are approaching their theoretic plateaus at different rates, with some closer to and some further from a predicted steady state. Although similar trends have been observed in other countries such as Japan, there is significant heterogeneity in international stroke mortality trends, and the pattern seen in the United States is not readily generalizable to other nations. The effect of variable declines in US stroke mortality rates across multiple demographic groups will be determined by both the magnitude of future declines and the absolute growth in the underlying populations. In this analysis, we have shown that, in the United States, expected declines in stroke mortality rates will not counterbalance projected increases in the growth of high-risk populations. Total stroke mortality in the United States is predicted to rise 3 times as fast as the general population over the next 30 years and result in a doubling of annual stroke deaths by 2032.

Discussion

Previous investigators have used several different methods to make projections of stroke mortality. Some have estimated stroke mortality from state-event transition models that integrate information about incidence and case fatality. Such models have the potential to create more refined estimates of risk in the population but require multiple assumptions about incidence and mortality rates that are difficult to estimate in the US population. Another group used an age-period-cohort model to forecast future stroke mortality in Sweden. This type of model incorporates additional variables to account for factors present around the time of death (period effect) and factors present in early life (cohort effect) in their study population. Although such models are useful in identifying factors that account for changes in historical mortality data, their utility in making future projections is constrained by the multiple arbitrary assumptions that must be made to predict future cohort, age, and period effects. Our model is simpler than previous ones; therefore, the effects of its underlying assumptions are more transparent. The principal assumptions of our model are that stroke mortality rates will not start to rise again and that historic age-, race-, and sex-specific trends will continue. If stroke mortality rates begin to rise again, the projected increases in the burden of stroke mortality will be magnified. Improved implementation of established prevention measures and the development of novel therapies could result in lower-than-expected rates in stroke mortality and make our results obsolete.

Our results are dependent on death certificate data for the analysis of historical trends in stroke mortality. Studies that have examined the validity of the death certificate diagnosis of stroke have generally found a high specificity but only a moderate sensitivity for the diagnosis. For example, in an analysis of 180 deaths of participants in the Minnesota Heart Study in 1980, a death certificate diagnosis of stroke had a 97% positive predictive value but only a 58% sensitivity for ischemic stroke. Although such findings may not be generalizable to national death certificate databases, a lack of sensitivity in the death certificate diagnosis of stroke would be expected to result in an underestimation of the actual recent and projected rates of US stroke mortality.

Population projections from the US Census Bureau rely on a number of assumptions about fertility, mortality, and net migration. Census Bureau projections of future mortality rates assume continued increases in life expectancy and a gradual narrowing of the disparities in death rates between sex and racial groups. They do not specifically account for factors related to individual diseases; therefore, new therapies that selectively affect mortality from competing causes of mortality such as coronary artery disease could change the population at risk for stroke in ways not accounted for in our model. These projections indicate that the doubling of the elderly population (≥65 years of age) over the next 30 years will be the predominant sociodemographic change affecting stroke mortality. The proportions of men and women in the population are expected to remain nearly constant over the next 30 years. The effect of changes in the racial and ethnic compositions of society is more difficult to assess. The highest relative growth is expected in nonwhite, nonblack populations in whom historical mortality data are sparse. Although both the crude and age-adjusted mortality rates of these populations are reported to be lower than those for both whites and blacks,
significant heterogeneity in stroke mortality rates is likely within this segment of the population, which includes Native Americans and a number of diverse populations of Asian origin. In addition, the NCHS mortality data do not contain information about ethnic origin. People of Hispanic ethnicity made up 5% of the white population in 1970 and are projected to account for \( \approx 25\% \) of the white population in 2032.\(^{20}\) Our conclusions are limited by our inability to model adequately the effects of such growth in minority ethnic and racial groups. Significant changes in the geographic distribution of Americans would also be likely to affect mortality rates in ways not accounted for in our models.

Assessing the likelihood that historic trends will continue is complicated by persisting uncertainty about the causes of past changes in stroke mortality rates. Although declines in stroke mortality may be attributable to societal reductions in stroke risk factors, declines in stroke mortality rates have correlated poorly with measured reductions in stroke risk factors in comparative studies.\(^{21}\) Furthermore, many studies have indicated that stroke incidence rates remained stable during the years that stroke mortality rates declined.\(^{22–28}\) If recent trends continue, however, increases in total stroke mortality will dwarf population growth in the near future. The social and financial impact of the projected rise in stroke deaths is likely to be substantial. Previous studies have shown that hospitalizations that result in death from ischemic stroke are associated with higher costs and lengths of stay than hospitalizations leading to successful discharge.\(^{29–31}\) Furthermore, the finding that the average age at stroke death is likely to remain stable suggests that the increased number of deaths is likely to result in a proportional increase in the number of quality-adjusted life-years lost because of ischemic stroke. Concerted efforts to reduce stroke mortality rates are needed to prevent this scenario from becoming a reality.

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References

The Next 30 Years of Stroke for Patients, Providers, Planners, and Politicians

Quibble with the models or puzzle over the formula, but heed the message of Figure 1. Stroke is more common in the elderly, and the proportion of elderly in the United States is rapidly growing. When questions involve comparisons, absolute numbers are shunned in favor of crude and especially adjusted rates. But here the question is what numbers we—as providers and planners of health care—should anticipate in the coming years. Figure 1 and its rising absolute numbers should alarm us. Are we prepared for this 98% projected increase over the next 30 years, from 139 000 patients dying from stroke in 2002 to 275 000 in 2033?

Considering mortality rather than morbidity underestimates the number, and many elderly are less frightened by death than by the prospect of long-term disability from stroke. Using Medicare hospital claims, the CDC estimated that during 2000 a total of 445 452 hospitalizations among Medicare enrollees were attributed to stroke. Only 8.7% of these 445 452 patients died during the hospitalization. Data on death following hospitalization were not reported. Additionally, focus on fatal and nonfatal stroke ignores the even more ubiquitous vascular injury to the brain—such as with small infarcts, small bleeds, and white matter changes—that can erode function in the elderly without ever leading to signs and symptoms recognized as stroke.

The solution rests in primary prevention in people who have not had a stroke and secondary prevention in patients who have. Paradoxically, developing effective treatments for stroke may only worsen the problem by enriching the population with survivors whose risk of recurrent fatal or nonfatal stroke is higher than that of the rest of the population. Competing risks complicate prevention as a solution. Successful prevention may simply shift the types of strokes that are disabling and killing people. For instance, antithrombotic therapy may reduce the occurrence of ischemic stroke but increase that of hemorrhagic stroke. Currently, the leading causes of death are heart disease and cancer. Whether stroke overtakes these two in the next 30 years is a matter of how successfully these diseases can be prevented. Fewer people dying of heart disease and cancer would mean more people at risk of stroke. Consequently, the authors’ call for “concerted efforts to reduce stroke mortality rates” is a competition with other investigators trying to reduce the mortality rate of other common diseases of the elderly.

The types of investigations needed to identify modifiable risk factors for stroke and to implement programs to control these risk factors define the fields of epidemiology and health services research. Why have the investigators of this report focused on death, jury-rigging an administrative database to their purpose? Why not use a population-based national database with patient-specific information on the occurrence and outcome of all strokes? Such a study would have yielded more useful information than the current report. The line of researchers waiting to use such a database would be long indeed, if only such a database existed and were readily available. Instead, inherently compromised studies will likely remain the standard to address important questions of stroke prevention.

Funding is part of the problem. Stroke researchers compete for dollars distributed to advance knowledge in many fields. Even among the stroke researchers, those studying prevention compete with those performing basic research and concentrating on treatment of acute stroke. In addition, recent attempts to protect the privacy of individuals provide additional disincentives to perform epidemiology and health services research. As opposed to current trends in the United States, the federal government should be looking for ways to encourage investigators to perform such research and to facilitate, not impede, its conduct. Perhaps a better balance can be found between sharing patient-specific information for the public good and protecting the rights of the individual, especially when the studies are observational and low risk.

So the solution for these projected numbers will involve the concerted efforts of not only stroke researchers and health care planners but also patient-advocacy groups and politicians. More money alone will not be enough. The solution will also require a fundamental revision of how research is performed with a reduction of barriers and disincentives. To the extent that preventative strategies fail and these projected numbers are correct, we need to develop the capacity to care for the anticipated flood of patients with their stroke-associated morbidity and mortality.

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