Population studies play important roles in assessing disparities and temporal changes in the burden of stroke, guiding interventional assessment in clinical trials, and describing risk factors that for ethical or design reasons cannot be addressed by clinical trials.

Population Description, Disparities, and Temporal Patterns

Concerns regarding the impact of population shifts were heightened this year with the reports describing several disturbing trends that were independent of the demographic population shift. Data from the National Hospital Discharge Study suggested that stroke hospitalization rates for the population aged 45 to 54 increased between 1980 and 2000 from 164.8/100 000 to 172.9/100 000, private insurance coverage decreased from 73% to 50%, and the proportion of stroke patients discharged home decreased from 72% to 62%. While national data on incident stroke are lacking, the few available data suggests that at there are at most very modest declines (perhaps there are increases). Finally, between 1990 to 1991 and 2000 to 2001, the inflation-adjusted costs for stroke increased 54% for infarctions, 57% for intracerebral hemorrhage, and 76% for subarachnoid hemorrhage. Collectively, when coupled with the demographic shifts in age, these trends portend an explosion in the public health impact of stroke.

Racial disparities in stroke have not been substantially reduced. In 1983 stroke was the fourth largest contributor to the male black-to-white detriment in life expectancy, contributing 7.0% of the 6.42-year disparity. By 2003 the contribution of stroke had fallen marginally to 6.2% of the 6.33-year disparity. For women in 1983 stroke contributed 10.8% of the 5.07-year disparity, and was second largest contributor. In 2003 stroke contributed 8.2% of the 4.54-year disparity and had fallen to the third largest contributor.

Reports suggest persistent geographic disparities in stroke. One of few reports examining geographic disparities in stroke incidence showed that compared to the northeastern United States, the hazard among “southern” male physicians was 22% higher (95% CI: 2% to 47%) for all stroke, and 30% higher (95% CI: 6% to 58%) for ischemic stroke. There is also an apparent racial difference in the geographic disparities in stroke mortality, with 6% to 21% larger black-to-white mortality ratios for southern states than for nonsouthern states. Finally, the prevalence of stroke was higher in the southeastern United States, with the age-adjusted estimated prevalence of stroke in the stroke belt states at or above the national average.

Geographic variations in stroke incidence rates were also reported within the European community, ranging from 210/100 000 in Dijon, France, to 600/100 000 in Novosibirsk, Russia. Important reports also expanded the description of stroke epidemiology countries including China, Brazil, sub-Saharan Africa, and Chile.

Associations With Risk Factors

Although general prospective risk functions for cerebral infarction have proven valuable, relatively low event hemorrhage rates have been a barrier to developing hemorrhage risk functions. The combination of the ARIC and CHS cohorts collectively provided over 260 000 person-years of follow-up on 21 680 participants resulting in 135 intracerebral hemorrhages—and overcome this barrier. Results suggested age, black race, and hypertension positively associated with ICH risk, whereas LDL and triglycerides were negatively associated. A recently reported risk function based on over 47 000 stroke events among over 1.2 million Koreans is noteworthy for broadening risk functions to nonwhite populations, and showed a slightly different collection factors associated with stroke risk (notably obesity, cholesterol and alcohol use).

Finally, a general risk function was reported from the 14 432 diabetic patients from the DIA study, showing predictive factors among diabetics including age, and hemoglobin A1c and smoking among men, and microvascular complications among women.

In addition to reports of general risk functions, substantial advances were made in the understanding specific risk factors for stroke, including:

1. The association between air pollution and heart disease is being mirrored by mounting evidence of an association with stroke. For example, during warm weather, stroke mortality in Helsinki was positively associated with levels of fine particles (<2.5 µm in diameter) and carbon monoxide, associations that did not persist during colder weather. A 10 µg/m³ increase in fine
particulate matter (again <2.5 μm) was also associated with a hazard of 1.28 (1.02 to 1.61) for death from stroke in the participants of the Women’s Health Initiative study.18

2. Evidence of an association of stroke risk and the metabolic syndrome was strengthened by reports including those in Asian and Australian populations.19,20

3. The understanding of dietary risk factors was advanced by reports including 2 that failed to support the hypothesis of decreased stroke risk with higher fish intake.21,22 Likewise, there was no association of calcium or vitamin D supplementation on stroke risk among women randomized in the Women’s Health Initiative.23 There was also little support for an association between sodium and potassium intake and the risk of stroke in the Rotterdam Study.24

4. The evidence of a link between indices of socioeconomic status (SES) and stroke risk was strengthened by several reports including evidence from the Brain Attack Surveillance in Corpus Christi (BASIC) study showing over a 20% difference in the risk of stroke between neighborhoods defined by SES measures.25 The Atherosclerosis Risk in Communities study provided insights to the potential pathways of the association of stroke risk and SES, showing a hazard ratio of 1.65 (95% CI: 1.22 to 2.22) for increased stroke risk associated with the absence of insurance coverage, which was in turn associated with higher risk ratios of never having a routine physical examination, being more likely to have undiagnosed hypertension and inadequate control of hypertension.26

5. The Women’s Health Initiative documented that pre-hypertension (120≤SBP≤139 or 80≤DBP≤89) is related to increased stroke risk.27

6. The link between psychosocial factors and stroke risk was strengthened by reports from the Framingham study showing depression symptoms among those <65 years of age were related to stroke risk,28 and evidence from Sweden that a lack of social support at work was associated with increased risk of stroke in women (but not men).29

7. The need for immediate treatment of TIAs was supported by the results of the EXPRESS study, where immediate treatment for TIAs reduced the 90-day stroke event rate from 10.3% to 2.1%.30 This finding adds importance to awareness that TIA and stroke event rate from 10.3% to 2.1%.30 This finding adds importance to awareness that TIA and stroke

8. The link between measures of subclinical atherosclerosis and stroke risk was strengthened by reports from the Northern Manhattan Study showing greater stroke risk with prevalent irregular carotid plaques (an approximate 5-times increased risk of stroke).36

Numerous other articles related associations with individual risk factors that must be excluded from this summary for page limitations.

Contributions advancing the understanding of stroke in young persons include associations of stroke risk with migraine headache (particularly among young women who smoke or use oral contraceptives)37 and Type I diabetes.38

Results from genetic studies have been mixed, perhaps suggesting challenges in understanding the genetics of stroke. The first report on a genome-wide association of ischemic stroke suggested that there is no single locus of main effect.39 In addition, a report suggested that heritability of ischemic stroke may be greater for women than men,40 and ischemic stroke in a probands poorly associated with ischemic stroke in siblings (suggesting genetic risk factors may not be subtype specific).41 On the positive side, a meta-analysis of major candidate genes suggest consistency between non-European and European stroke patients,42 and additional evidence for specific candidates have been advanced including Gly460Trp43 adaponecin (ADIPOQ),44 and arachidonate 5-lipoxygenase activating protein (ALOX5AP).45

Disclosures

None.

References


