

genes of haemostatic and inflammatory system, homocysteine metabolism, and the renin-angiotensin aldosterone system, suggest significant effect for several SNIPs. Genome-wide linkage studies on Iceland population showed the correlation between several haplotypes in PDE4D and ALOX5AP genes and a risk of stroke. Their significance in other populations is unclear. Recently, a new technology allows looking at thousands of variants across the human genome. One small genome-wide association study in stroke was performed so far, however, SNPs affecting stroke risk were not found. Identifying genetic factors in stroke is important because it may allow identify new stroke mechanisms, which can allow formulating novel treatment strategies.

JANIKA KÓRV

Epidemiology of stroke

Department of Neurology and Neurosurgery, University of Tartu, Tartu, Estonia

Stroke incidence and case-fatality, their time trends and geographical variations have already been in the centre of interest for several years. Despite some progress in primary prevention the incidence of stroke remains high. Population-based stroke registries are valuable sources in providing information about stroke epidemiology in different geographical regions. However, serious restrictions for processing personal sensitive data exist at least in Estonia and limit the use of national databases for research purposes. The incidence rates of stroke vary between study centres. The variations are probably related to environmental and life-style factors, socioeconomic differences and perhaps genetics. The results from time trend studies of stroke have been conflicting. Mostly, decline in stroke mortality is reported and some centres have shown a trend of increasing stroke incidence. Increasing incidence has been linked to the implement of computerised tomography and unfavourable changes in risk factor profiles in certain communities. Stroke time trend studies have shown that improvements in primary prevention have a significantly higher impact on the incidence of stroke compared to case-fatality (CFR). A decline in CFR has been detected in several populations over time. CFR is mostly dependent on the acute care of stroke and stroke severity. The severity of stroke might be related to the extent and quality of primary prevention. It has been shown that pre-stroke use of antiplatelets agents and antihypertensive treatment for patients with hypertension results in less severe incident stroke. The goal is to prevent stroke from happening, and therefore it is necessary to intensify the primary prevention of stroke.

DANUTA RYGLEWICZ

Post-stroke epilepsy

First Department of Neurology, Institute of Psychiatry and Neurology, Warsaw, Poland

Stroke is the most common cause of the epilepsy among those age 60 and over. The overall occurrence of post-stroke epilepsy is estimated at 3-8%. In the Oxfordshire Community Stroke Project recurrent seizures in the first year of follow-up were diagnosed in 5.7% and within 5 years in 11.5%. According to others about 15% of patients experienced unprovoked seizures within 5 years following stroke. The risk of developing epilepsy following stroke remain significantly elevated for at least 20 years following stroke. In Poland on the basis of Polish National Stroke Registry that included 3238 patients, within one year follow-up period, post stroke epilepsy was diagnosed in 3.9% of patients, more frequently among those with intracerebral hemorrhage (4.1%). In the group of patients with ischemic stroke recurrent epileptic fits more frequently have been observed among patients with cardioembolic stroke – 7.5% (95% CI 4.1-11.7) than among patients with lacunar stroke – 3.9% (95% CI 1.9-6.8). In the Oxfordshire Community Stroke Project the difference was even higher, only 1% of patients with lacunar strokes developed poststroke epilepsy vs 11% of patients with total anterior circulation infarct.

Post-stroke epilepsy is related to different clinical factors (Copenhagen Stroke Study): 1. younger age (OR – 1.7/10 years, 95% CI 1.3-2.1), 2. increasing stroke severity at the onset of stroke (OR – 1.3/10 point decrease Scandinavian Stroke Scale, 95% CI 1.0-1.6), 3. lesion size (OR 1.2/10 mm, 95% CI 1.0-1.3), 4. intracerebral hemorrhage (OR – 3.3, 95% CI 1.3-8.6), 5. early seizures (OR 4.5, 95% CI 1.3-16.0).