Lifestyle Factors and Stroke Risk: Exercise, Alcohol, Diet, Obesity, Smoking, Drug Use, and Stress

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Various lifestyle factors have been associated with increasing the risk of stroke. These include lack of exercise, alcohol, diet, obesity, smoking, drug use, and stress. Guidelines endorsed by the Centers for Disease Control and Prevention and the National Institutes of Health recommend that Americans should exercise for at least 30 minutes of moderately intense physical activity on most, and preferably all, days of the week. Recent epidemiologic studies have shown a U-shaped curve for alcohol consumption and coronary heart disease mortality, with low-to-moderate alcohol consumption associated with lower overall mortality. High daily dietary intake of fat is associated with obesity and may act as an independent risk factor or may affect other stroke risk factors such as hypertension, diabetes, hyperlipidemia, and cardiac disease. Homocysteine is another important dietary component associated with stroke risk, while other dietary stroke risk factors are thought to be mediated through the daily intake of several vitamins and antioxidants. Smoking, especially current smoking, is a crucial and extremely modifiable independent determinant of stroke. Despite the obstacles to the modification of lifestyle factors, health professionals should be encouraged to continue to identify such factors and help improve our ability to prevent stroke.

Introduction

Various lifestyle factors have been associated with increasing the risk of stroke. These include lack of exercise, alcohol, diet, obesity, smoking, drug use, and stress. Strategies for effective modification of these factors include risk factor identification, goal attainment for risk factor control, compliance strategies, and continued follow-up. Although each of these of these lifestyle factors is unique and an important risk factor on its own, the combination of these lifestyle risks represents a heavy burden of increased stroke risk [1].

The recent consensus statement dealing with prevention of a first stroke established evidence-based recommendations for decreasing stroke risk that act as template for reduction of risk factors [2]. Lifestyle modifications to reduce stroke risk may present a great challenge, in that social, behavioral, and cultural factors increase the complexity of the risk reduction strategy. It must remain the priority of health professionals to define and promote a lifestyle conducive to changing diet, increasing activity, evaluating alcohol and drug use, and promoting cessation of cigarette smoking.

Physical Activity

Currently available data support the benefits of physical activity. Guidelines endorsed by the Centers for Disease Control and Prevention and the National Institutes of Health recommend that Americans should exercise for at least 30 minutes of moderately intense physical activity on most, and preferably all, days of the week [3,4]. Regular physical activity has well-established benefits for reducing the risk of premature death and cardiovascular disease. Moderate and heavy levels of physical activity have been associated with reduced coronary heart disease [CHD] incidence in individuals when compared with inactive persons. Additionally, there was no evidence that heavy physical activity conferred any greater benefit than moderate levels [5,6]. In recent years, evidence has been accumulating which supports the protective effect of moderate physical activity on stroke incidence in men and women. For stroke, the benefits are apparent even for light-to-moderate activities, such as walking, and the data support additional benefits to be gained from increasing the level and duration of one's recreational activity [5–14]. Other studies that have shown the protective effect of physical activity for men include the Framingham Study, Honolulu Heart Program, and the Oslo Study [7–9]. In the Framingham Study, physical activity in subjects with a mean age of 65 years was associated with a reduced stroke incidence. In men, the relative risk was 0.41 after accounting for the

effects of potential confounders, although there was no evidence of a protective effect of physical activity on risk of stroke in women [8]. Both the Nurses' Health Study and Copenhagen City Heart Study have demonstrated an inverse association between the level of physical activity and the incidence of stroke among women [10,11]. The protective effects of leisure-time physical activity have also been found for blacks and Hispanics in the National Health and Nutrition Examination Survey I follow -up study and in the Northern Manhattan Stroke Study [12,13]. Dose-response relationships have sometimes been difficult to demonstrate with from no effect to deleterious effects of vigorous physical activity compared with lower levels of physical activity [8,14,15]. In the Northern Manhattan Stroke Study, heavy forms of physical activity provided additional benefits compared with light-to-moderate activities, and additional protection was observed with increased duration of exercise. However, the prevalence of such activities in the elderly was quite low [13]. Finally, the large prospective cohort of the Atherosclerosis Risk in Communities study (ARIC) also recently reported a small but protective effect of different types of physical activity on ischemic stroke risk [16].

The protective effect of physical activity may be partly mediated through its role in controlling various known risk factors for stroke, such as hypertension [17], cardiovascular disease [18], diabetes [19], and body weight. Other biological mechanisms are also associated with physical activity, including reductions in plasma fibrinogen and platelet activity, and elevations in plasma tissue plasminogen activator activity and high-density lipoprotein (HDL) concentrations [20–23].

Physical activity is a modifiable behavior that requires greater emphasis on stroke prevention campaigns. Indeed an odds ratio (OR 2.7; CI 2.1–3.4) and attributable risk of 18% to 38% for physical inactivity, as reported in a recent population-based case-control study further illustrates the broad public health problem this risk factor has become in the community [24].

Alcohol Use

Alcohol is consumed by millions of Americans annually and poses a major health threat. An estimated 107,800 alcohol-related deaths occur each year [25]. These include, but are not restricted to, deaths from traffic crashes, various cancers, accidental deaths, suicides, and homicides. The economic cost of alcohol abuse and dependence is estimated to be $100 billion. Although the health risks of alcohol consumption are well known, moderate drinking may reduce cardiovascular disease risk and total mortality.

The effect of alcohol as a risk factor for ischemic stroke is controversial and probably is quite dependent on dose. For hemorrhagic stroke, prospective cohort studies have shown that alcohol consumption has a direct dose-dependent effect [26–28]. For infarction, chronic heavy drinking and acute intoxication have been associated with an increased risk among young adults [29]. In older adults, studies have shown an increased risk among men who drink heavily, no effects among men and women after controlling for other confounding risk factors [30–32], and a protective effect for moderate alcohol consumption [33–36].

Recent epidemiologic studies have shown a U-shaped curve for alcohol consumption and coronary heart disease mortality with low-to-moderate alcohol consumption associated with lower overall mortality [37]. In an overview analysis of stroke studies, a J-shaped association curve was suggested for the relation of moderate customary alcohol consumption to ischemic stroke [36,38]. As in CHD, alcohol could be protective for ischemic stroke if consumed in moderation. The latter relationship was most consistent for white populations; however, little if any association existed for Japanese and possibly black populations. Although the weight of the epidemiologic evidence suggests that customary heavy alcohol consumption is a risk factor for brain hemorrhage, careful scrutiny of the data from cohort studies shows inconsistencies between studies regarding the proposed J-shaped association curve for alcohol consumption and ischemic stroke [36]. Finally, a large prospective cohort of male physicians has recently confirmed the protective effects of light-to-moderate alcohol on ischemic stroke. This study found an overall relative risk of 0.79 (95% CI 0.66–0.94) for all strokes and an RR of 0.77 (95% CI 0.63–0.94) for ischemic stroke in a study of more than 22,000 men during 12 years of follow-up [39].

The dose-dependent relationship between alcohol and stroke is consistent with the observed deleterious and beneficial effects of alcohol. The deleterious effects of alcohol for stroke may operate through various mechanisms, including increasing hypertension, hypercoagulable states, cardiac arrhythmias, and reducing cerebral blood flow. However, there is also evidence that light-to-moderate drinking can reduce the risk of coronary artery disease, increase HDL-cholesterol, and increase endogenous tissue plasminogen activator, improve endothelial function, stabilize plaque, and have antithrombotic properties [40].

Numerous studies have also attempted to clarify whether the protective effect of alcohol was actually limited to wine versus beer and other "hard" alcohol. It has been suggested that polyphenolic compounds found in red wine, white wine, grape juice, and beer act as antioxidants, possibly reducing the atherogenicity connected with LDL [41]. Supporting this hypothesis is a large prospective cohort study from Spain that demonstrates the protective effect of wine consumption on stroke mortality [42]. However, other case-control and prospective studies have not found a difference between the effects of wine, beer, and liquor [38].

It is difficult to consider recommending alcohol to those who are nondrinkers. However, for those who are currently drinking, the elimination of heavy drinking and reduction of alcohol intake to moderate levels (*ie,* no more

than two drinks per day) could undoubtedly reduce their incidence of stroke. Based on alcohol-related longevity and cardiac risk data, we should adopt public health policy for stroke prevention in alcohol users similar to that for cardiac disease. In the United States, this would conform to a recommendation of no more than two standard drinks of beer, wine, or liquor in those who wish to drink and do not have a contraindication to alcohol use.

Diet

Diet may be an important modifiable stroke risk factor. High daily dietary intake of fat is associated with obesity and may act as an independent risk factor or may affect other stroke risk factors such as hypertension diabetes, hyperlipidemia, and cardiac disease. Although data suggest that diet may play an important role as a stroke risk factor, few studies have been able to clarify this relationship because of the complex issues associated with dietary intake and nutritional status. Early large ecological studies have suggested that excess fat intake associated with migration to Western nations may lead to increased risk of both CHD and stroke [43]. Prospective studies such as the Nurses Health Study have demonstrated a significant association between dietary fat intake, especially from trans-unsaturated fats, and overall CHD, although total fat intake was not significantly associated with CHD [44]. Results from the Framingham Study have suggested conflicting findings with an inverse association between dietary fat and ischemic stroke [45].

Dietary sodium is another important factor that may be associated with increased stroke risk. Specifically, increased sodium intake is involved with an increased risk of hypertension. Indeed, reduction in salt consumption will significantly lower blood pressure in many individuals and could lead to a decrease in stroke mortality [46].

Homocysteine is another important dietary component associated with stroke risk. This amino acid is involved in methionine metabolism and is associated with dietary intake of folate and vitamin B_{12}. The Framingham Study found that deficiencies in folate, B_{12} levels, and pyridoxine accounted for the majority of elevated homocysteine levels in the study cohort [47]. Case-control studies have demonstrated an association between moderately elevated homocysteine and vascular diseases, including stroke [48,49]. Evidence from the Framingham Study demonstrates that elevated homocysteine levels are independently associated with stroke in the elderly with adjusted RR ranging from 1.3 to 1.9 over the top three quartiles of homocysteine as compared with the first quartile [50]. Finally, genetic and environmental factors may interact in the complex relationship between homocysteine and stroke.

Other dietary stroke risk factors are thought to be mediated through the daily intake of several vitamins and antioxidants. The protective effects of fruits and vegetables originate from antioxidant mechanisms [51,52], or through elevation of potassium levels [53]. Dietary antioxidants, including vitamin C, vitamin E, and beta carotene belong to a group of antioxidants called flavonoids. These scavengers of free radicals are thought to be associated with stroke risk reduction through the free-radical oxidation of LDL, a process that inhibits the formation of atherosclerotic plaques [54,55]. While strong epidemiologic studies demonstrating a protective effect are scarce, the large Western Electric cohort did find a moderate decrease in stroke risk associated with a higher intake of both beta-carotene and vitamin C [56]. Other dietary factors associated with a reduced risk of stroke include potassium [53], milk and calcium [57], green tea [58], and fish oils [59,60]. Large studies, such as the Vitamin in Stroke Protection (VISP) trial are currently investigating the protective effects of vitamin B_6 vitaminB_{12} and folate for recurrent stroke. Additionally, recent evidence from case-control studies has suggested that increased dietary and supplemental intake of vitamin E or vitamin B_6 may decrease stroke risk [61,62].

Studies regarding the association of stroke and diet are difficult to interpret because of the complexity of collecting accurate dietary histories. It is clear that dietary factors are directly correlated with aggravating some of the traditional stroke risk factors such as hypertension, cardiac disease, and hypercholesterolemia. The dietary guidelines for a stroke prevention recommended by the National Stroke Association are shown in Table 1 and include limiting dietary fat, limiting the total cholesterol, increasing intake of fruits and vegetables, maintaining energy balance through diet and exercise, and maintaining an adequate intake of dietary calcium [2].

Obesity

An estimated 97 million adults in the United States have been characterized as overweight or obese. The need to prevent or modify this lifestyle factor poses a major public health challenge. Obesity is a predictor of overall mortality and is associated with an overall increase in mortality. Further, obesity has been associated with independent risk factors for stroke, including higher levels of blood pressure, blood glucose, and atherogenic serum lipids. In the Framingham Study, obesity, defined as a Metropolitan Relative Weight greater than 30% above average, was a significant independent contributor to brain infarction incidence in men aged 35 to 64 and in women 65 to 94 years [63]. In the Honolulu Heart Study, obesity was identified as an independent factor related to stroke incidence [64]. The Nurses Health study prospectively examined the association between obesity and stroke risk. Using obesity groupings for body mass index (BMI) of 27 kg/mg^2 as compared with individuals with BMI of less than 21 kg/m^2 the investigators documented significant relative risks for stroke ranging between 1.75 and 2.37 among the cohort, even after controlling for age, smoking, hormone use, and menopausal status (Table 2) [65].

Table 1. Nutritional status assessment dietary guidelines

Guidelines	Nutritional Advice	Practical Recommendations
Reduce intake of dietary fat	Fat should be less than 30% of total energy	Limit saturated fats
Reduce intake of dietary cholesterol	Cholesterol should be less than 10% of total energy	Check labels for cholesterol content
Increase intake of fruits, vegetables, and high-fiber foods	5 Servings per day: fruits, vegetables	All fruits and vegetables
	6 servings per day: high fiber	Foods high in fiber include whole wheat breads and cereals
Maintain energy balance	Proper diet include protein sources (*eg*, fish) Exercise, 8400–12595 kJ/wk	Increase moderate physical activity to 30 min/d
Maintain adequate intake of dietary calcium	1200–1500 mg, 12–24 yrs 1000–1500 mg, 25–50 yrs 1000–1500 mg, postmenopausal 1200–1500 mg, pregnant/nursing	Increase calcium through dairy products, and supplements after advice from doctor
Reduce intake of dietary sodium	Salt intake to 2400–3000 mg unless specified otherwise by physician	Avoid prepared and high salt foods
Increase intake of beta carotene and antioxidants	Antioxidant photochemicals	Consume fruits and vegetables especially carrots, cantaloupes, tomatoes, grapefruit, kale, green turnips, and ligants

Table 2. Relative risk of stroke for body mass index defined levels of obesity*

Body Mass Index	Relative Risk	95% CI
27–28.9	1.75	1.17–2.59
29–31.9	1.90	1.28–2.82
> 32	2.37	1.6–3.5

*Body mass index less than 27 used as reference.

The pattern and distribution of obesity may also influence stroke risk. It has been demonstrated that central obesity manifested by abdominal deposition of fat and abnormal hip/waist ratios have been related to the occurrence of atherosclerotic disease [66]. In a prospective study of US male health professionals, the age-adjusted relative risk associated with extreme quintiles of hip/waist ratio was 2.33 (1.23–4.37) [67]. These findings suggest that abdominal obesity may be a stronger predictor of increased stroke risk in men than body mass index.

Tobacco Use

The use of tobacco, most frequently cigarette smoking, is a widespread public health problem. It is estimated that tobacco is responsible for the deaths of 434,000 US smokers and 53,000 nonsmokers annually [68]. Recent reports suggest that there has been no decrease in the prevalence of cigarette smoking over the last 20 years. Despite these dire health statistics, almost 50 million Americans continue to smoke, and the majority of adolescents experiment with cigarettes. Worldwide, tobacco is estimated to cause about 3 million deaths each year [69].

Smoking, especially current smoking, is a crucial and extremely modifiable, independent determinant of stroke [70]. In case-control studies, the effect of cigarette smoking remained significant after adjustment for other factors, and a dose-response relationship was apparent. In cohort studies, cigarette smoking was an independent predictor of ischemic stroke. A meta-analysis showed that the relative risk (RR) of stroke was 1.9 [71]. Furthermore, a dose-response effect and an interaction with age was noted, with the average risk being relatively greater among younger persons. Women smokers were at slightly higher risk than men who smoke [72]. The association between cigarette smoking and ischemic stroke has been consistent among major cohort studies such as the study of smoking cessation and decreased risk of stroke in women and the Framingham Study [72,73]. Additionally, smoking is highly associated with development of carotid atherosclerosis. For different stroke types, the stroke risk attributed to cigarette smoking was greatest for subarachnoid hemorrhage, intermediate for cerebral infarction, and lowest for cerebral hemorrhage.

Cessation of smoking leads to reduction in stroke risk. Several studies showed that substantial reduction in risk occurs within 2 to 5 years [72,73]. However, some studies reported a more gradual decline in risk over one or two decades [70]. Targeted community interventions for smoking cessation have resulted in modest gains among light-to-moderate smokers, but led to little change in heavy smokers, when recent secular trends for smoking are considered [74]. Counseling, health incentives, and the various nicotine substitutes such as the patch or gum have shown modest success over time. At the individual level, physicians have been criticized for not taking an active role in patient

education for smoking cessation. Public health education programs, economic measures, and individual counseling should be continued and expanded to discourage initial smoking behavior and encourage smoking cessation.

Illicit Drug Use

Drug abuse is a major social problem that primarily affects millions of adolescents and young adults in the United States [75]. Although cocaine has been the street drug most commonly associated with stroke [76], other drugs reported to be associated with stroke include heroin, amphetamines, lysergic acid diethylamide (LSD) phencyclidine hydrochloride (PCP), "Ts and Blues," marijuana, sympathomimetic decongestants, cold remedies, and diet aids such as phenylpropanolamine, ephedrine, and pseudoephedrine [76]. Some of these drugs are associated with cerebral hemorrhagic and ischemic stroke mechanisms. The majority of information about stroke and drug abuse comes from case reports or case series. Furthermore, the relationship between drug abuse and stroke is often confounded by use of more than one drug in individual cases. There is a paucity of well-designed epidemiologic studies concerning drug abuse and stroke, which may result from the fact that use of these drugs is a criminal offense. Although many researchers believe that a significant relationship exists, rigorous epidemiologic evidence is lacking.

Acute Triggers

Evidence is emerging that acute triggers may play a role in the onset of acute coronary syndromes, particularly myocardial infarction, and possibly stroke. A variety of acute stressors, often involving more than one simultaneously-occurring event, have been shown to increase the relative risk of myocardial infarction [77]. Although the authors note that the population-attributable risk from a given episode of physical activity, anger, or sexual activity is low, they make the case that this research has important implications in understanding the pathogenesis of acute cardiovascular events. Despite growing data in the filed of cardiology and widespread acceptance by the public of the significance of triggering events, that relationship with stroke has not been systematically addressed.

Conclusions

There is compelling evidence that the lifestyle factors presented here are significant factors for stroke risk. What is equally important is that amount of physical activity, alcohol consumption, diet, obesity, smoking, drug use, and stress are all modifiable. Clinicians, along with other public health professionals, must understand that alterations of such lifestyle factors require continued support and encouragement. Strategies such as goal attainment may be effective in changing certain behaviors, such as decreasing dietary intake and increasing physical activity. Other behaviors, including smoking, may require broader social and political interventions. Despite the obstacles to modification of lifestyle factors, health professionals should be encouraged to continue to identify such factors and help improve our ability to prevent stroke.

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