Special Issue: Vascular Disease Risk Factors

Smoking: A risk factor for vascular disease

Phyllis Gordon, MSN, APRN, ACNS-BC, and Patty Flanagan, MSN, RN-BC

Smoking in the United States includes at least 16% of the adults, 24% of high school students, nearly 8% of middle school students and is more prevalent in men than women; however, a decline in smoking has been documented in recent years. Cardiovascular disease continues to be a leading cause of death. Smoking is identified as a significant risk factor for cardiovascular disease, carotid disease, and peripheral artery disease with peripheral artery disease documented in 5%–10% of all Americans. Smoking is also a significant risk factor in the development of abdominal aortic aneurysm in 7% of men aged 65–75 years with a smoking history. Toxic chemicals found in tobacco smoke are reported at 7,357 chemical compounds including the addictive chemical of nicotine. A substantial number of large studies and well-known trials have identified an increase in proinflammatory cells and cellular processes in the smoker diagnosed with atherosclerosis and in the mechanism attributed to abdominal aortic aneurysm development. The cost of smoking to health care is significant, and smoking cessation can demonstrate benefits to health improvement and the cost of health care. (J Vasc Nurs 2016;34:79-86)

EPIDEMIOLOGY OF SMOKING

The National Health Interview Survey defines current smoking among adults as having smoked at least 100 cigarettes during one's lifetime and smoking every day or some days. In 2014, the Centers for Disease Control and Prevention (CDC) estimated 16.8% (40.0 million) of US adults were current cigarette smokers. Of these, 76.8% (30.7 million) smoked daily and 23.2% (9.3 million) smoked less than daily.[1]

For youth, aged 18 years or younger, the current smoker is defined by the National Survey on Drug Use and Health as having smoked part or all of a cigarette in the past 30 days. In 2014, the CDC reported that 24.6% of high school students and 7.7% of middle school students were current tobacco users; cigarette smoking being 9.2% of high schoolers and 2.5% of middle schoolers.[2]

The prevalence of smoking has declined between 2005 and 2014 from 20.9% to 16.8%, with a full 1% drop occurring between 2013 and 2014.[1] Although smoking has declined overall, the CDC in 2014 reported an increase in the initiation of smoking in youth and young adults since 2002.[3]

In 2014,[3] smoking rates were higher among men (18.8%) compared with women (14.8%). Among the racial and ethnic groups, the American Indian and/or Alaska Natives have the highest prevalence of smoking (29.2%), followed by Whites (18.2%), Blacks (17.5%), Hispanics (11.2%), and Asians (9.5%). Rates were higher in persons aged 25–44 years (20%) and lowest among those ≥ 65 (8.5%). Persons living below the poverty level had higher rates (26.3%) than those living at or above the poverty level (15.2%). Persons aged >25 years with a general education certificate have a higher prevalence of smoking (43%) compared with those with a graduate degree (5.4%). Regionally, persons in the Midwest have the highest rates (20.7), and the West has the lowest at 13.1%.[1] See Figure 1 smoking activity per state.

From the Division of Vascular Surgery, School of Nursing, University of Texas Health Science Center, San Antonio, Texas; Vascular Surgery, Albany Medical Center, Albany, New York.

Corresponding author: Phyllis Gordon, MSN, APRN, ACNS-BC, Division of Vascular Surgery, 8911 Whitney Circle, San Antonio, TX 78254 (E-mail: gordonp@uthscsa.edu).

1062-0303/$36.00

http://dx.doi.org/10.1016/j.jvn.2016.04.001

INCIDENCE OF VASCULAR DISEASE IN SMOKERS

Cardiovascular diseases (CVD) accounted for more deaths in the United States than any other major cause of death, in every year since 1900, except 1918.[4] Vascular diseases such as peripheral artery disease (PAD), carotid artery disease, and abdominal artery aneurysms are all affected by smoking; as smoking enhances the atherosclerotic process increasing the risk for CVD.

Five to 10 percent (7–12 million) of Americans are affected by PAD.[5] Men and women are equally affected, and people of Hispanic origin may have higher rates than non-Hispanic whites.[6] PAD affects 12%–20% of those aged > 60 years. The single most important cause of PAD is smoking with a strong correlation between number of cigarettes smoked and risk for PAD.

Stroke is the major cause of disability in the United States. Smoking not only affects blood coagulability which can obstruct blood flow, but also, has shown an association between smoking and carotid intima-media thickness, atherosclerotic disease, and carotid intima-media thickness progression.[7] Eighty percent of the approximately 700,000 strokes in the United States each year are caused by an occlusion.[8] The risk for stroke among current smokers was found to be highest among men aged 60–64 years (Relative Risk (RR) = 3.9; 95% Confidence Interval (CI), 3.2–4.8) and among women aged 65–69 years (RR = 3.8;

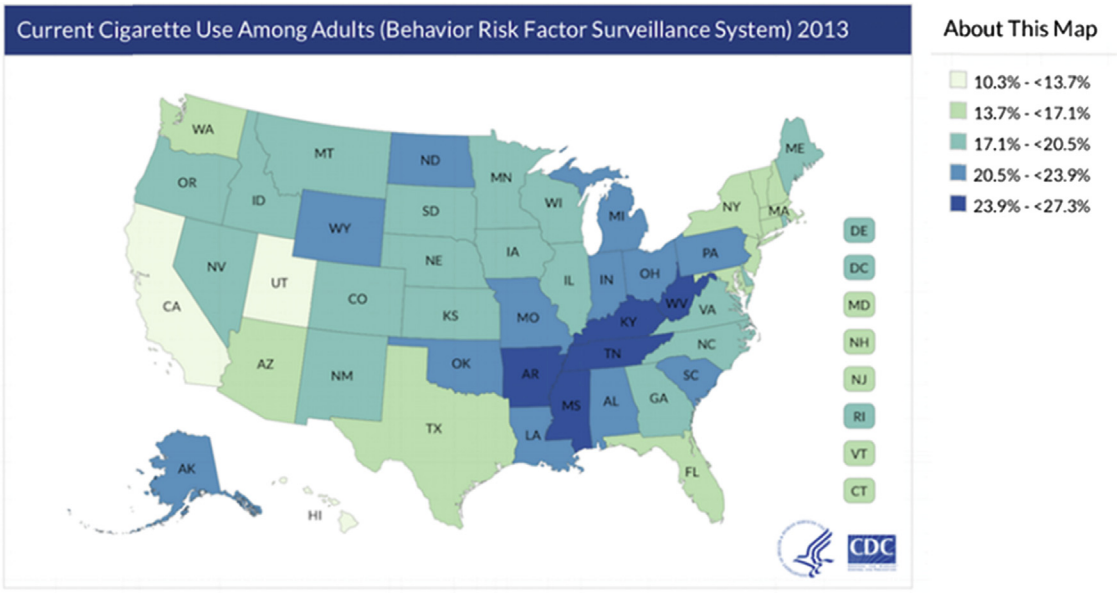

Figure 1. *State tobacco activities tracking and evaluation system. Interactive maps: cigarette use—adult current smokers— http://www.cdc.gov/statesystem/cigaretteuseadult.html.*

95% CI, 2.3–6.3).[3] In addition to the effects on smokers, second-hand smoke increases an individual's risk for stroke by 20% −30%. Second-hand smoke exposure causes more than 8,000 deaths from stroke annually.[9]

Abdominal aortic aneurysms (AAAs) are most prevalent in men who have a history of smoking; occurring in approximately 6%–7% in men aged 65–75 years.[10] A screening study in Sweden found that the prevalence of AAA in women aged 70 years was low (0.8%) for women who previously smoked but increased to 2.0% for current smokers.[10] The CDC in 2014[3] reported that autopsies revealed that smoking in adolescents and young adults cause early abdominal aortic atherosclerosis. The National Vital Statistic Reports in 2011[11] identified aortic aneurysms as the primary cause of 10,597 deaths and a contributing cause in more than 17,215 deaths in the United States in 2009.[11] People who have a history of smoking are 3–5 times more likely to develop an AAA.[12]

WHAT IS IN A CIGARETTE?

Nicotine is the primary addictive property in cigarette smoke. Nicotine reaches the brain within 10–15 seconds after smoke is inhaled.[13] A portion of the nicotine molecule is similar to acetylcholine, an important brain neurotransmitter.[13] In the central nervous system, acetylcholine has a range of effects including arousal and reward. As a result, the euphoria induced via tobacco use serves as a reinforcer for its use.

Rodgman and Perfetti's (as cited in the Surgeon Generals report, 2014)[3] work on identifying 7,357 chemical compounds, in a cigarette. Identified the gas phase of cigarette smoke to include nitrogen (N_2), oxygen (O_2), carbon dioxide (CO_2), CO, acetaldehyde, methane, hydrogen cyanide (HCN), nitric acid, acetone, acrolein, ammonia, methanol, hydrogen sulfide (H_2S), hydrocarbons, gas phase nitrosamines, and carbonyl compounds. In addition, the constituents in the particulate phase to include carboxylic acids, phenols, water, humectants, nicotine, terpenoids, paraffin waxes, tobacco-specific nitrosamines, polyaromatic hydrocarbon (PAHs), and catechols.

The Tobacco Products Scientific Advisory Committee[14] reviewed chemical compounds in cigarette smoke and determined the following effects related to vascular disease. Inhalation exposure to arsenic causes vasospasticity. Carbon monoxide and oxidizing gasses can cause ischemic effects. Aldehydes are associated with lipid peroxidation, a contributing factor in vascular disease. Lead at low exposures has shown to elevate systemic blood pressure which can result in endothelial dysfunction leading to vascular disease. As well as PAHs having been found to accelerate the development of atherosis in animal studies and are associated with the development of atherosclerotic disease in humans. PAH's found in cigarette smoke include Benz[a] anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, and Dibenz[a,h]acridine.

Smokers can potentially have varying levels of exposure to smoking-related chemicals, based on differences in smoker's style. The depth of draw, intervals between puffs, the length of cigarette smoked, depth of inhalation into the lungs, and the number of puffs influences the individual's exposure to some smoke chemicals.[15]

EFFECTS OF SMOKING ON THE VASCULAR SYSTEM

Smoking, as previously noted, is well established as a significant risk factor in the development of vascular disease. The effects of smoking on CVD were first suspected in the 1940s,[16] and the pathogenic effects on the arterial wall began to be discussed in the 1960s.[17] In 1967, researchers in the Framingham Heart Study[17] identified smoking as a risk factor for CVD. In a correspondence to the British Medical Journal in 1967, Szanto[18] submitted a discussion suggesting that dietary sugar and smoking had similar effects on the arterial wall. In addition, in 1998, trial researchers from the Atherosclerosis Risk in Communities