

Pettersson, Andreas; Kaijser, Magnus; Richiardi, Lorenzo; Askling, Johan; Ekblom, Anders; and Akre, Olof

Women Smoking and Testicular Cancer: One Epidemic Causing Another?

International Journal of Cancer; (2004); 109(6): 941-944.

In many countries the incidence of testicular cancer has increased epidemically, but the etiology remains obscure. Maternal smoking during pregnancy has been suggested to be a cause, but a satisfactorily valid assessment of the hypothesis is still lacking. Analyses of data from cancer registries and published surveys in Europe suggest that maternal smoking during pregnancy increases male offspring's risk of testicular cancer, according to epidemiologists in Sweden and Italy. Tobacco smoke can decrease levels of pregnancy hormones, reduce placental blood flow and retard fetal growth. The carcinogens present in smoke can also cross the placenta. "If the testicular development is disturbed or delayed, this may result in neoplastic transformation as well as fertility problems and malformations of the urogenital tract," Pettersson's group concluded. The strong geographical and temporal correlations between female smoking and testicular cancer indicate that if smoking during pregnancy is a cause of testicular cancer in the offspring, part of the increasing trends could be explained, and more importantly: avoided, by primary prevention

Getahun, Darios, MD, MPH, Amre, Devendra, MD, PhD, Rhodes, George G., MD, MPH and Demissie, Kitaw, MD, PhD

Maternal and Obstetric Risk Factors for Sudden Infant Death Syndrome in the United States: Obstetrics & Gynecology; (2004); 103(4): 646-652.

As reported in Obstetrics and Gynecology for April, Dr. Darios Getahun, from the UMDNJ-Robert Wood Johnson Medical School in New Brunswick, New Jersey, and colleagues analyzed data from 12, 404 births that resulted in SIDS and 49, 616 control births to determine risk factors for SIDS. Maternal factors tied to a heightened risk included younger age, less education, nonwhite, smoking during pregnancy, and nonparticipation in prenatal care.

Band, Pierre R., MD; Le, Nhu D., PhD; Fang, Raymond, MSc; and Deschamps, Michele, PhD

Carcinogenic and Endocrine Disrupting Effects of Cigarette Smoke and Risk of Breast Cancer: Lancet; (2002) 360: 1044-49

We assessed the effect of smoking separately for premenopausal and postmenopausal women. Risk of breast cancer was significantly increased in women who had been pregnant and who started to smoke within 5 years of menarche, and in nulliparous women who smoked 20 cigarettes daily or more and had smoked for 20 cumulative pack years. Our results suggest that cigarette smoking exerts a dual action on the breast, with different effects in premenopausal and postmenopausal women. Our observations reinforce the importance of smoking prevention, especially in early adolescence.

Spira, Avrum; Beane, Jennifer; Shah, Vishal; Liu, Gang; Schembri, Frank; Yang, Xuemei; Palma, John; and Brody, Jerome S.

Effects of cigarette smoke on the human airway epithelial cell transcriptome

Proceedings of the National Academy of Sciences USA; (2004) 101(27): 10143-10148; published online before print as 10.1073/pnas.0401422101

Cigarette smoke is the major cause of lung cancer, the leading cause of cancer death, and of chronic obstructive pulmonary disease, the fourth leading cause of death in the United States. Using high-density gene expression arrays, we describe genes that are normally expressed in a subset of human airway epithelial cells obtained at bronchoscopy (the airway transcriptome), define how cigarette smoking alters the transcriptome, and detail the effects of variables, such as cumulative exposure, age, sex, and race, on cigarette smoke-induced changes in gene expression. We also determine which changes in gene expression are and are not reversible when smoking is discontinued. The persistent altered expression of a subset of genes in former smokers may explain the risk these individuals have for developing lung cancer long after they have discontinued smoking. The use of gene expression profiling to explore the normal biology of a specific subset of cells within a complex organ across a broad spectrum of healthy individuals and to define the reversible and irreversible genetic effects of cigarette smoke on human airway epithelial cells has not been previously reported. Cigarette smoking alters the gene expression of airway epithelial cells, and some changes do not return to normal after smoking cessation. Smokers who had stopped smoking at least 2 years before the study tended to cluster with nonsmokers, and more recent quitters tended to cluster with current smokers. Thirteen genes, among them potential tumor suppressor genes, did not return to normal levels even after 20 to 30 years of smoking cessation. These permanent changes might explain the persistent risk of lung cancer in former smokers.

Whincup, Peter H.; Gilg, Julie A; Emberson, Jonathan R; Jarvis, Martin J; Colin, Feyerabend; Bryant, Andrew; Walker, Mary; and Cook, Derek G.

Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement

British Medical Journal: (July 2004); 329: 200 - 205.

People who were nonsmokers but had relatively high levels of cotinine had a heart disease risk 50% higher than those people that were exposed to low levels. "The evidence is now compelling. The government should not delay any further in introducing legislation to protect nonsmokers from this unnecessary risk."

Murin, Susan MD, FCCP; and Incardi, John, PharmD, MS, the University of California-Davis Medical Center

Cigarette Smoking and the Risk of Pulmonary Metastasis From Breast Cancer
CHEST; (2001); 119(6):1635-1640

This case-controlled study suggests a relationship between active cigarette smoking and the development of pulmonary metastatic disease among women with breast cancer. This provides an intriguing and biologically plausible explanation for the higher rate of fatal breast cancer among smokers.

Vogt, Molly T., PhD; Department of Orthopaedic Surgery, University of Pittsburgh, PA
The Effect of Cigarette Smoking on the Development of Osteoporosis and Related Fractures
Medscape General Medicine; (1999); 1(3)

Smokers are not only more susceptible to fracture but are also more likely to have recurrent fractures. Healing of fractures is delayed in smokers, the maturation of the regenerating bone is reported to be abnormal, and nonunion or malunion is more frequent. Cigarette use is believed to have an adverse effect on most aspects of bone metabolism: decreased bone mass, increased rate of bone loss, and osteoporosis.

Faucher, Mary Ann, CNM, PhD
Factors that Influence Smoking in Adolescent Girls
Journal of Midwifery Women's Health 48(3): 199-205, 2003

The research findings relating less locus of control, less self-control, and a strong desire for social approval with body image dissatisfaction are particularly important risk factors for smoking. Individually and cumulatively they increase susceptibility to both peer pressure and peer influence, both validated as important risk factors for smoking initiation in both adolescent boys and girls.

Barclay, Laurie, MD
Nicotine Patch May be More Helpful Than Bupropion for Adolescent Smoking Cessation.
Journal of Consulting Clinical Psychology; (2004); 72:729-735

Using Bupropion plus nicotine patch does not appear to improve rates of smoking cessation in adolescents compared with using the nicotine patch alone.

Goodman, Elizabeth, MD, and Captiman, John, PhD
Depressive Symptoms and Cigarette Smoking Among Teens
Pediatrics; (October, 2000): 106(4): 748-755

“Cigarette use is a powerful determinant of developing high depressive symptoms,” says Elizabeth Goodman, MD, associate professor of pediatrics in the division of Adolescent Medicine at Children’s Hospital medical Center of Cincinnati and lead author of the study. “In fact, nondepressed teens who smoke face approximately a 4 times greater risk of developing depression than nonsmoking teens.” The effect of smoking on the development of depression may be attributable to the impact of nicotine or other smoking by-product on central noradrenergic receptor systems. Recent demonstrations of the efficacy of antidepressants in smoking cessation, independent of previous or current major depressive disorders, provide additional support for this view.

Vuckovic, Nancy Ph.D.; Polen, Michael R. M.A.; and Hollis, Jack F. Ph.D.
The problem is getting us to stop. What teens say about smoking cessation.
Preventive Medicine; (September 2003): 37(3) Pages 209-218

Participants preferred programs that respect the challenges that teens face in quitting, and acknowledge their choice in making the decision to quit. Teens wanted nonjudgmental and confidential support from cessation counselors, and preferred counselors who are ex-smokers, give useful quit tips, and can provide support for quit attempts. Private, computer-based programs and personalized telephone services were options for delivering cessation information and support.

Teen smokers can supply valuable information to improve youth cessation programs to fit teen lifestyles, respect the challenges teens face, and acknowledge their choice in making the decision to quit.

Joanna S. Fowler, Jean Logan, Gene-Jack Wang, Nora D. Volkow, Frank Telang, Wei Zhu, Dinko Franceschi, Naomi Pappas, Richard Ferrieri, Colleen Shea, Victor Garza, Youwen Xu, David Schlyer, S. John Gatley, Yu-Shin Ding, David Alexoff, Donald Warner, Noelwah Netusil, Pauline Carter, Millard Jayne, Payton King, and Paul Vaska
Low Monoamine Oxidase B in Peripheral Organs in Smokers
Proceedings of the National Academy of Sciences: (2003); 100: 11600-11605; published online before print as 10.1073/pnas.1833106100

One of the major mechanisms for terminating the actions of catecholamines and vasoactive dietary amines is oxidation by monoamine oxidase (MAO). Smokers have been shown to have reduced levels of brain MAO, leading to speculation that MAO inhibition by tobacco smoke may underlie some of the behavioral and epidemiological features of smoking. Because smoking exposes peripheral organs as well as the brain to MAO-inhibitory compounds, we questioned whether smokers would also have reduced MAO levels in peripheral organs. Here we compared MAO B in peripheral organs in nonsmokers and smokers by using positron emission tomography and serial scans. Binding specificity was assessed by using the deuterium isotope effect. We found that smokers have significantly reduced MAO B in peripheral organs, particularly in the heart,

lungs, and kidneys, when compared with nonsmokers. Reductions ranged from 33% to 46%. Because MAO B breaks down catecholamines and other physiologically active amines, including those released by nicotine, its inhibition may alter sympathetic tone as well as central neurotransmitter activity, which could contribute to the medical consequences of smoking. In addition, although most of the emphases on the carcinogenic properties of smoke have been placed on the lungs and the upper airways, this finding highlights the fact that multiple organs in the body are also exposed to pharmacologically significant quantities of chemical compounds in tobacco smoke.

Vander Straten, Melody, MD; Carrasco, Daniel, MD; Paterson, Martha S., MD; Mccrary, Monica L., MD; Meyer, Diane J., MD; Tying, Stephen K., MD, PhD
Tobacco Use and Skin Disease
Southern Medical Journal; (2001); 94(6): 621-634

The graphic picturing of health effects of smoking was rated by smokers to be an important motivational factor for smoking cessation. For many users of tobacco, however, the more immediate and visible effects of this drug such as wrinkling, yellow teeth, and bad breath may provide a more compelling stimulus for smoking cessation than the knowledge that tobacco kills. Dermatologic effects of cigarette smoking include facial wrinkling, facial gauntness, complexion color changes, decreased skin moisture, yellowed nails, harlequin nails, halitosis, nicotine stomatitis, and skin burns.

In a study of 1,109 professional baseball players, leukoplakia and erythroplakia were strongly associated with smokeless tobacco use. Of the current users, 46.3% had leukoplakia. Risk for leukoplakia was found to be associated with amount of tobacco used, recency of use, and type and brand of snuff used. Clinically, one cannot accurately distinguish dysplastic from benign lesions, and therefore biopsy is necessary to determine whether malignant transformation has occurred.

Copeland, Jan
Developments in the treatment of Cannabis Use Disorder
Current Opinion in Psychiatry: (May, 2004); 17(3): 161-168

The research literature on randomized controlled trials of interventions for cannabis use disorder is only a decade old. In the past year only one such trial has been accepted for publication. This trial of cognitive-behavioral interventions compared two sessions of motivational enhancement therapy with nine sessions of a multi-component therapy that added cognitive-behavioral therapy and case management. At 4 months from randomization the nine-session treatment reduced cannabis smoking and associated consequences, such as dependence symptoms, significantly more than the two-session treatment, which significantly reduced cannabis use relative to the delayed treatment controlled condition. At 15 months follow-up the superior outcome for the more intensive intervention had dissipated; however, a significant improvement from baseline was maintained. There were no sex or ethnic differences in treatment outcome. These findings are consistent with those of similar trials. There is a paucity of research into

pharmacological and psychological interventions for cannabis use disorder. There are no accepted pharmacotherapies available. Whereas relatively brief cognitive-behavioral therapy has the strongest evidence of success for adults with cannabis dependence, among adolescents involved in the juvenile justice system and those with severe, persistent mental illness, longer and more intensive therapies provided by interdisciplinary teams may be required.

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Ting-Kai Li, M.D., Statement by Director, National Institute on Alcohol Abuse and Alcoholism on **Substance Abuse and Mental Health**; (June 1, 2004)

Alcohol is the primary psychoactive substance of abuse by American children. About 78 percent of 12th graders, 67 percent of 10th graders, and 47 percent of 8th graders have used alcohol, according to the National Institute on Drug Abuse's *Monitoring the Future* survey. Youth who report having been drunk at least once include 62 percent of 12th graders, 44 percent of 10th graders, and 21 percent of 8th graders. Roughly half of those percentages say that they drank heavily (five or more drinks in a row) in the past 2 weeks.

National Institute on Alcohol Abuse and Alcoholism; Alcohol Alert No. 59; (April 2003)
Underage Drinking: A Major Public Health Challenge

People who begin drinking before age 15 are four times more likely to develop alcohol dependence at some time in their lives compared with those who have their first drink at age 20 or older.

National Institute on Alcohol Abuse and Alcoholism; Alcohol Alert No. 62; (July 2004)
Alcohol-An Important Women's Health Issue

While it is true that men are more likely to drink alcohol and more likely to drink greater amounts, women have a higher risk of developing problems from alcohol consumption. When a woman drinks, the alcohol in her bloodstream typically reaches a higher level than a man's even if both are drinking the same amount. This is because women's bodies generally have less water than men's bodies. Because alcohol mixes with body water, a given amount of alcohol is less diluted in a woman's body than in a man's. Women become more impaired by alcohol's effects and are more susceptible to alcohol-related organ damage.

During early adolescence, girls may be especially vulnerable to stress. Levels of perceived stress have been found to be the most powerful predictor of alcohol and other drug use, after peer substance abuse.

National Center for Chronic Disease Prevention and Health Promotion; (February, 2004)
Secondhand Smoke Fact Sheet

Definition: Secondhand smoke, also known as environmental tobacco smoke (ETS), is a mixture of the smoke given off by the burning end of tobacco products (sidestream smoke) and the smoke exhaled by smokers (mainstream smoke).^{1,2}

Secondhand smoke contains a complex mixture of more than 4,000 chemicals, more than 50 of which are cancer-causing agents (carcinogens).^{1,2}

People are exposed to secondhand smoke in the home, workplace, and in public venues such as bars, bowling alleys, and restaurants.³

Health Effects: Secondhand smoke is associated with an increased risk for lung cancer and coronary heart disease in nonsmoking adults.^{1,2,4} Secondhand smoke is a known human carcinogen (cancer-causing agent).^{2,4}

Because their lungs are not fully developed, young children are particularly susceptible to secondhand smoke. Exposure to secondhand smoke is associated with an increased risk for sudden infant death syndrome (SIDS), asthma, bronchitis, and pneumonia in young children.^{1,5}

Current Estimates: An estimated 3,000 lung cancer deaths and 35,000 coronary heart disease deaths occur annually among adult nonsmokers in the United States as a result of exposure to secondhand smoke.⁶

Each year, secondhand smoke is associated with an estimated 8,000–26,000 new asthma cases in children.⁴ Annually an estimated 150,000–300,000 new cases of bronchitis and pneumonia in children aged less than 18 months (7,500–15,000 of which will require hospitalization) are associated with secondhand smoke exposure in the United States.⁴

Approximately 60% of people in the United States have biological evidence of secondhand smoke exposure.⁷

Among children aged less than 18 years, an estimated 22% are exposed to secondhand smoke in their homes, with estimates ranging from 11.7% in Utah to 34.2% in Kentucky.⁸

U.S. Department of Health and Human Services; Center for Disease Control and Prevention

Targeting Tobacco Use: The Nation's Leading Cause of Death 2004

If current patterns of smoking persist, 6.4 million people currently younger than 18 will die prematurely from a tobacco-related disease. Paralleling this enormous health toll is the economic burden of tobacco use: more than \$75 billion per year in medical expenditures and another \$80 billion per year resulting from lost productivity.

Smokeless tobacco, cigars, and pipes also have deadly consequences, including lung, larynx, esophageal, and oral cancer. Low-tar cigarettes and novel tobacco products such as bidis and clove cigarettes are not safe alternatives.

Each year, primarily because of exposure to secondhand smoke, an estimated 3,000 nonsmoking Americans die of lung cancer, and more than 35,000 die of heart disease. An estimated 150,000–300,000 children younger than 18 months of age have respiratory tract infections because of exposure to secondhand smoke.

Morbidity and Mortality Weekly Report: (2004, March 5); 53(8): 174-175

Alcohol Use among Adolescents and Adults—New Hampshire, 1991-2003

Alcohol abuse is the third leading preventable cause of death in the United States. Binge and heavy drinking increase the risk for cirrhosis, cancer, heart disease, stroke, injury, and depression.

Alberg, Anthony J., PhD, MPH; and Samet, Jonathan M., MD, MS: From the Department of Epidemiology, Johns Hopkins University, Bloomberg School of Public Health
CHEST: (2003, January); 123:21S-49S

Epidemiology of Lung Cancer

In the United States, lung cancer remains the leading cause of cancer death in both men and women even though an extensive list of risk factors has been identified. Far and away the most important cause of lung cancer is exposure to tobacco smoke through active or passive smoking.

Lung cancer is a major health issue for women. Due to historical cigarette smoking patterns, the epidemic of lung cancer started later in women than in men, but, in contrast to the situation in men, lung cancer incidence rates in women are still increasing.

Alguacil, Juan; and Silverman, Debra T.: 2004 American Association for Cancer Research. *Cancer Epidemiology Biomarkers & Prevention*: (2004, January); vol. 13, 55-58

Smokeless and other Noncigarette Tobacco Use and Pancreatic Cancer

Cigarette smoking is an important and well-established cause of pancreatic cancer. These research results suggest that heavy use of smokeless tobacco, and to a lesser extent, cigar smoking may increase the risk of pancreatic cancer among nonsmokers of cigarettes. (2004, January); vol. 13, 55-58.

Centers for Disease Control and Prevention; National Center for Environmental Health **Second National Report on Human Exposure to Environmental Chemicals**: (2003, January); CAS No. 486-56-6; Table 60. Cotinine.

Cotinine is a metabolite of nicotine that tracks exposure to environmental tobacco smoke (ETS) among nonsmokers. Higher cotinine levels indicate more exposure to ETS, which has been identified as a known human carcinogen. The first *Report* found that among nonsmokers in the overall population, cotinine levels decreased substantially during the 1990s. The *Second Report* contains additional results for the years 1999 and 2000 and results for population groups defined by age, sex, and race/ethnicity. From 1991-1994 to 1999-2000, cotinine levels decreased 58% for children, 55% for adolescents, and 75% for adults. However, in 1999-2000, cotinine levels in children were more than twice those of adults. Non-Hispanic blacks had levels more than twice those of Mexican Americans and non-Hispanic whites. Although efforts to reduce ETS exposure during the 1990s were successful, ETS exposure remains a major public health concern.

Cotinine is currently regarded as the best biomarker in active smokers and in smokers exposed to ETS. Cotinine can be measured in serum, urine, saliva, and hair. Males continue to have higher levels than females. People younger than 20 years of age have higher levels than people aged 20 years and older.

National Cancer Institute; <http://plan.cancer.gov/tobacco.html#sec6>; (August 30, 2004)
Research on Tobacco and Tobacco-Related Cancers

For every woman who smoked in 1950, 600 women smoke today. Lung cancer has now replaced breast cancer as the number one cancer killer of women.

14% Hispanic, 22% White, 24% African American, 35% Native American & Alaska Native women

Of the millions of women who try to quit smoking each year, only a small percentage succeeds. African American, Hispanic, younger, less educated women have the lowest quit rates.

CA A Cancer Journal for Clinicians: News Briefs; (2004, May/June); 54(3); p 126
Less Lethal Smoking Still a Pipe Dream

Researchers from the Massachusetts Institute of Technology and the American Cancer Society found that low-tar and very low-tar were no less harmful than those with regular or medium-tar levels. The terms “light” and “extra light” are misleading because they imply less health risk but do not correspond to less hazardous cigarettes. Moreover, no product or strategy designed to reduce harm from smoking has been shown to work. Cessation is clearly the way to go because we don’t have conclusive evidence that anything else works. The tar and nicotine content listed on cigarette labels is based on measurements from a smoking machine. There is a very poor correlation between machine-measured yield and what people are actually taking in.

Rizzi, Maurizio, MD; Sergi, Margherita, MD; Andreoli, Arnaldo, MD; Pecis, Marica, MD; Bruschi, Claudio, MD; and Fanfulla, Francesco, MD
CHEST: (2004); 125(4); 1387-1393

Environmental Tobacco Smoke May Induce Early Lung Damage in Healthy Males

This study demonstrates that current exposure to ETS in healthy male adolescents is associated with lung function impairment independently of the effects of maternal smoking during pregnancy. A significant correlation was found between the level of exposure and functional impairment. There is evidence that neonatal lung mechanics are altered when the mother smokes during pregnancy. They studied two subgroups of passive smokers; those exposed only after birth and those exposed both during pregnancy and after birth.

The Toll of Tobacco in Washington State (2002) tobaccofreekids.org
Tobacco industry Influence in Washington

An estimated 237.8 million dollars is spent on marketing each year in Washington.

(60 Seconds X 60 minutes X 24 Hours X 365 days / \$237.8 million = over **\$450 a minute** spent each year on marketing)

Annual healthcare costs in WA that are directly caused by smoking are \$1.52 billion.

(60 seconds X 60 minutes X 24 Hours X 365 / 1.52 billion = **\$2891 a minute** spent each year on healthcare)

Published research studies have found that kids are three times more sensitive to tobacco advertising than adults and are more likely to be influenced to smoke by cigarette marketing than by peer pressure, and that one-third of underage experimentation is attributable to tobacco company advertising.