

HVSSSC

Hudson Valley Student Support Services Center

175 Route 32 North | New Paltz, NY 12561 | Phone: 845-255-4874 | Fax: 845-255-3836

FACT SHEET

June 2009

Mary Grenz Jalloh, M.S., M.P.H., CHES, B.C.S.C.R.
Executive Director

Tammy Rhein, L.M.S.W., C.A.S.A.C., CPP
Program Coordinator

TOBACCO ADDICTION FOUND TO BE NEARLY IMMEDIATE

August 29, 2002

WORCESTER, Mass.—A startling new study published today in the international journal *Tobacco Control* shows that kids typically get hooked on nicotine with alarming speed and at levels of tobacco use that are so low that few researchers had even considered addiction possible. Because adults who are hooked on nicotine generally smoke at least ten cigarettes every day, scientists have always assumed that a person could not become hooked until he or she smoked at least that much. It usually takes a few years for young smokers to progress to smoking ten cigarettes per day so it was also assumed that nicotine dependence was very slow to develop. The study, however, indicated that just the opposite is true: kids get hooked more quickly while smoking much less. The results of the study, published in the journal's September issue, surprised even the researchers themselves. "I expected that some kids would get hooked quickly, but I thought that the average kid would have to smoke for a few years to get hooked," admitted Joseph R. DiFranza, MD, professor of family medicine & community health at the University of Massachusetts Medical School (UMMS) and lead author of the study. "I thought that kids who got hooked quickly would be the exception to the rule. As it turned out, the kids who did *not* get hooked quickly were the exception." The study was sponsored by the National Cancer Institute and conducted by a team of researchers at UMMS, the University of London and Harvard University.

The study showed that for the teenage girls who got hooked, it took only an average of three weeks from when they started to smoke occasionally. Among the boys who got hooked, half were hooked within six months of the start of occasional smoking. "Some of these kids were hooked within a few days of starting to smoke," reported Dr. DiFranza. "We are unable to explain why girls get hooked faster but we have begun a new study sponsored by the National Institute on Drug Abuse to explore the differences between the sexes."

Perhaps even more surprising than the speed with which symptoms of addiction appeared was the very small amount of tobacco required. At the time when they first got hooked, the young smokers were typically smoking only one day per week, averaging only two cigarettes per week. This contradicts the assumption that addiction

does not begin until youths are smoking at least ten cigarettes per day. In two thirds of the cases, addiction had appeared prior to daily smoking. This means that youths who have never smoked as much as one cigarette per day are finding it very difficult to quit when they try. DiFranza commented: "It's startling to find that kids who are smoking two cigarettes per week need help to overcome a dependency on nicotine but the data shows that youths who showed signs of being hooked at these very low levels of consumption were 44 times more likely to be still smoking at the end of the study." Previous research has shown that it takes the average teenage smoker 18 years to break the habit for good.

After the addiction begins, a growing tolerance to the effects of nicotine requires the addicted smoker to gradually smoke more and more. The common wisdom had been that tolerance to nicotine must precede the addiction. The study results suggest that the opposite is true, that the addiction comes first.

In order to determine how long it takes for kids to get hooked, the researchers followed 679 seventh grade students over a period of 30 months. The research subjects were interviewed in great detail eight times over the course of the study. Among 332 youths who had ever used tobacco, 40% reported symptoms of addiction. "This study has overturned a lot of conventional wisdom," DiFranza said. Addiction to tobacco does not require daily smoking, heavy smoking or prolonged smoking, and tolerance to nicotine doesn't precede addiction, it follows it. Researchers are now scrambling to determine the effects of small intermittent doses of nicotine on the brain. Already, one such study has concluded that a single exposure to nicotine has long lasting effects on the brain. The authors coin a new term, "juvenile onset nicotine dependence" to emphasize that children are different from adults when it come to the effects of nicotine. The authors site previous research showing that people who begin to smoke as adolescents are more likely to get hooked, to have greater difficulty quitting, to smoke for a greater number of years and to smoke more heavily as adults. The brain does not stop growing at birth; brain development continues into adolescence. The fact that the adolescent brain is still developing may make adolescents more vulnerable to addiction than adults. Animal research has shown that at doses seen with smoking, nicotine causes brain damage in adolescent

animals that is not seen in adult animals. The impact of nicotine on the brain is also stronger and longer lasting in adolescents.

The study's authors also note that it has been known for several decades that a physical dependence to narcotics can develop after a single dose of morphine. "A convergence of data from human and animal studies leads me to suspect that addiction to nicotine begins, in many cases, with the first cigarette," commented DiFranza. The University of Massachusetts Medical School is one of the fastest growing academic health centers in the country, attracting more than \$131 million in research funding annually. Ranked fifth in the annual *US News & World Report* ranking of primary care medical schools, UMMS is a leader in health sciences education, research, clinical care and public service.

Contact:

Joseph R. DiFranza, MD, 508-856-5658

NY TIMES article: By DULCIE LEIMBACH

Published: June 20, 2005

One factor in this debate is that scientists understand more about how the adolescent brain works. Two years ago, a study led by Dr. Chambers, then an assistant professor of psychiatry at Yale, found that because of continuous neurological developments in adolescence and young adulthood, increased preferences for risky behavior and novelty seeking emerge, predisposing teenagers to experiment with drugs and ending up with addictive behaviors.

The neural circuits that release chemicals that link new, adultlike experiences with the motivation to repeat them develop more rapidly during the teenage years than do the mechanisms that control these urges and impulses (which depend on exercising reason and judgment at the conscious level, basically knowing right from wrong). As a result, teenagers are not only more likely to feel enticed by drugs than older people are, but the effects on their brain can also be long lasting.

Teenage smoking, which continues to decline but slower than in the past, offers an illustration. A report published in 2000 said that just a few cigarettes could lead to addiction. The study, which tracked the smoking habits of 700 12- and 13-year-olds in Massachusetts for a year, revealed that addiction could begin within days of inhaling a first cigarette.

Children that young "have an extremely hard time quitting compared to 18-year-olds," said Dr. Joseph R. DiFranza, who led the study and is a professor of family medicine at the University of Massachusetts Medical School in Worcester.

D. Neurodevelopmental effects

When compared to children of nonsmokers, children of smokers perform more poorly in school. They also have lower scores in cognitive functioning tests - in particular, language and auditory processing - and have more behavioural problems, including conduct disorders,

hyperactivity, and decreased attention spans. Cognitive and behavioural deficits in children have lifelong consequences and result in increased costs for education and social services.

Seventeen studies have addressed the effects of ETS exposure on child development and behaviour. Most have controlled for sociodemographic characteristics and some have demonstrated dose-response relationships, with greater deficits among children with higher exposures. In some studies, children's postnatal ETS exposure and ETS exposure of nonsmoking mothers during pregnancy have been independently associated with subtle changes, albeit statistically significant, in child development and behaviour. Adverse effects resulting from children's postnatal exposure to tobacco smoke are biologically plausible in light of evidence of altered brain development in animal models. Taken as whole, however, these studies are difficult to interpret, in part due to the possible influence of uncontrolled confounding factors. Thus, the effects of prenatal and postnatal ETS exposure on cognition and behaviour remain unclear.

From www.ASH.org

*Tobacco smoke and nicotine are among the best studied agents for their effects on the developing brain. Children born to women who smoke during pregnancy are at risk for IQ deficits, learning disorders, and attention deficits. Children born to women who are passively exposed to cigarette smoke are also at risk for impaired speech, language skills, and intelligence. Children exposed to tobacco smoke after birth also are at risk for various behavioral problems.

*The Collaborative on Health and the Environment (CHE) is a nonpartisan partnership of individuals and organizations concerned with the role of the environment in human and ecosystem health.

CHE seeks to raise the level of scientific and public dialogue about the role of environmental contaminants and other environmental factors in many of the common diseases, disorders and conditions of our time.

Established in 2002, participation is open to health professionals, researchers, health-affected and patient groups, advocacy organizations and indeed anyone concerned about protecting the health of current and future generations from environmental harm.

***Nicotine is a neurotoxin in the adolescent brain: critical periods, patterns of exposure, regional selectivity, and dose thresholds for macromolecular alterations.**

Abreu-Villaca Y, Seidler FJ, Tate CA, Slotkin TA.

Department of Pharmacology and Cancer Biology, Duke University Medical Center, 27710, Durham, NC, USA. In the fetus, nicotine is a neuroteratogen that elicits cell damage and loss and subsequent abnormalities of synaptic function. We explored whether these effects extend into adolescence, the period when most people begin smoking. Beginning on postnatal day 30, rats were

given a 1 week regimen of nicotine infusions or twice-daily injections, at doses (0.6, 2 and 6 mg/kg/day) set to achieve plasma levels found in occasional to regular smokers. We assessed indices of cell packing density and cell number (DNA concentration and content), cell size (total protein/DNA ratio) and neuritic projections (membrane/total protein) in the midbrain, hippocampus and cerebral cortex, three regions known to be vulnerable to developmental effects of nicotine. With either route of administration, nicotine evoked shortfalls in DNA concentration and content, compensatory elevations of total protein/DNA, and reductions in the membrane/total protein ratio. Nearly all of the effects were apparent even at the lowest dose of nicotine and remained fully evident 1 month posttreatment. Although both males and females showed significant alterations, in general the effects were larger in females. Our results indicate that in adolescence, even a brief period of continuous or intermittent nicotine exposure, elicits lasting alterations in biomarkers associated with cellular and neuritic damage. As the effects are detected at exposures that produce plasma concentrations one-tenth of those in regular smokers, the exquisite sensitivity of the adolescent brain to nicotine neurotoxicity may contribute to lasting neurobehavioral damage even in occasional smokers.

PMID: 12850578 [PubMed - indexed for MEDLINE]
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12850578

*Nicotine as a Teratogen

According to American Heritage a teratogen is a substance that causes the malformation of an embryo or fetus.¹ The below studies examine nicotine as a teratogen in rats. It should be noted that for obvious reasons no study has yet examined nicotine exposure alone (without accompanying cigarette smoke) as a teratogen in the human embryo or fetus.

Smoking may lead to [teen depression](#)

CHICAGO ([Reuters Health](#)) October 03, 2000- Contrary to the notion that depressed teenagers were more likely to take up smoking, a study found that young people who became smokers were more likely to become depressed, researchers said on Monday.

Cigarette smoking was the "strongest predictor" of developing depressive symptoms among a group of 8,704 teenagers who were not depressed a year earlier, said study author Elizabeth Goodman of Children's Hospital Medical Center and the University of Cincinnati College of Medicine.

The adolescents who were not depressed at the start of the study--and may or may not have been experimenting with cigarettes--were four times more likely to have depressive symptoms if they were moderate or heavy smokers a year later.

The impact of nicotine or other cigarette additives on certain brain receptors could be to blame for the onset of depressive symptoms, Goodman said.

There has been some success in using anti-depressants to help smokers stop, suggesting a close link between the effects of cigarettes and the brain's chemistry that dictates mood.

Smoking prevalence among American teenagers remained high despite drops in adult smoking rates, Goodman reports in the journal *Pediatrics*, and researchers have been trying to figure out why. Previous research has produced conflicting results, and the common belief was that smoking was a sought-after if temporary relief from depression.

"Typically, increased likelihood of smoking initiation and progression have been viewed as consequences of depression," Goodman wrote. But "high depressive symptoms were not predictive of smoking progression in this study."

Smoking may be a cause of depression in teen-agers

CHICAGO (Oct 03, 00; AP) - A new study suggests smoking may be a cause of depression in teen-agers, contradicting the current thinking that says depressed people may smoke to feel better.

The study found that teens who smoked were about four times more likely to develop highly depressed symptoms during a year's time.

The researchers speculated that nicotine or other smoking byproducts may have a depressive effect on the central nervous system.

The study adds to a growing body of conflicting research on links between tobacco and the mind.

"The thing that bolsters the idea is that there is evidence that anti-depressant drugs are helpful in treating nicotine addiction," said Dr. Elizabeth Goodman, an adolescent-medicine specialist at Children's Hospital Medical Center of Cincinnati who led the study.

The study appears in the October issue of *Pediatrics*, the monthly journal of the American Academy of Pediatrics. Other researchers have linked teen smoking with suicide, and smoking with depression in adults, but they disagree over whether tobacco use is a cause or merely a result of a depressed state.

Most people think that those who tend to be depressed "self-medicate by smoking. This is probably not the case," said Naomi Breslau, director of research at Henry Ford Health Systems in Detroit.

Breslau's own research also has suggested tobacco may somehow contribute to depression. She said that while the new findings do not prove smoking is a cause, they strongly support that theory.

"They find absolutely no evidence that depressive symptoms per se increase the risk for smoking," she said. "They do find very clear evidence in the other direction." She added: "It's just one more adverse effect of smoking on health."

The study relied not on doctors' diagnoses but on teen-agers' reports of having symptoms suggestive of depression.

The study analyzed data from teens questioned in 1995 and 1996 in a national study on adolescent health. It included 8,704 teens who were not initially depressed and 6,947 teens who were not initially smokers.

Evidence suggesting depression was a cause rather than a result of smoking evaporated when the researchers took into account other factors that may have prompted the teens to start smoking, such as friends' use of tobacco and poor grades.

Current smokers included those who smoked as little as one cigarette in the previous month and those who smoked a pack a day or more. The researchers did not examine whether teens who smoked the most were the most likely to develop depression, but some of their other findings suggest that may have been the case. After a year's time, 4.8 percent of the nonsmokers had developed depressed symptoms compared with 12 percent of those who initially smoked at least a pack a day.

Linda Pederson, an epidemiologist at the Centers for Disease Control and Prevention's office on smoking and health, said the study was well-done, larger and more nationally representative than previous research that reached similar conclusions. see also www.aap.org

Gene may keep smokers hooked on nicotine-

Smokers who cannot quit may have a gene that makes them enjoy the nicotine in tobacco too much. This could explain why some people find it easier to stop smoking than others. Some individuals get enhanced pleasure when they smoke. They are going to enjoy it more and it's going to be harder for them to stop. The gene exists in about 10 percent of the general population and about 30 percent of smokers. This gene controls the way nicotine stimulates the production of dopamine, a chemical that passes messages between nerve cells, in the brain. Studies have found that nicotine, alcohol, and illicit drugs induce pleasure by causing dopamine releases. The same gene that contributes to nicotine addiction has been implicated in drug addiction, alcoholism and obesity. The Houston researchers found the genetic link to smoking in a study of 156 lung cancer victims and 126 control subjects. This finding could eventually lead to new, more effective drugs for quitting smoking. (32)

32) Spitz, Margaret chief of epidemiology at M.D. Anderson Cancer Center. Houston 98-3-1

Youth Tobacco and Marijuana Use Relative to Prenatal Cigarette and Marijuana Exposure

As part of their long-term follow-up of prenatal marijuana and tobacco exposure, researchers at Carleton University have examined whether maternal cigarette smoking and marijuana use during pregnancy were associated with an increased risk of initiation and daily/regular use of tobacco and marijuana among one hundred fifty-two 16- to 21-year-old adolescent offspring. The participants were from a low risk, predominately middle-class sample participating in an ongoing, longitudinal study. Findings indicated that offspring whose mothers reported smoking cigarettes during their pregnancy were more than twice as likely to

have initiated cigarette smoking during adolescence than offspring of mothers who reported no smoking while pregnant. Offspring of mothers who reported using marijuana during pregnancy were at increased risk for both subsequent initiation of cigarette smoking (OR=2.58) and marijuana use (OR=2.76), as well as daily cigarette smoking (OR=2.36), as compared to offspring of whose mothers did not report using marijuana while pregnant. There was also evidence indicating that dose-response relationships existed between prenatal exposure to marijuana and offspring use of cigarettes and marijuana. These associations were found to be more pronounced for males than females, and remained after consideration of potential confounding variables. The authors note that these results suggest that maternal cigarette smoking and marijuana use during pregnancy are risk factors for later smoking and marijuana use among adolescent offspring, and add to the weight of evidence supporting the importance of programs aimed at drug use prevention and cessation among women during pregnancy. Porath, A.J. and Fried, P.A. Effects of Prenatal Cigarette and Marijuana Exposure on Drug Use Among Offspring. *Neurotoxicology and Teratology*, 27(2), pp. 267-277, 2005.

Other articles:

An, L.C., Bernhardt, T.S., Bluhm, J., Bland, P., Center, B., Ahluwalia, J.S., et al. (2004). Treatment of tobacco use as a chronic medical condition: Primary care physicians self-reported practice patterns. *Preventive Medicine*, 38, 574-585.

Berlin, I., Vorspan, F., Singleton, E.G., Warot, D., Notides, C., & Heishman, S.J. (2005). Reliability and validity of the French version of the Tobacco Craving Questionnaire. *European Addiction Research*, 11, 62-68.

Berlin, I., Vorspan, F., Warot, D., Manéglier, B., & Spreux-Varoquaux, O. (2005). Effect of glucose on tobacco craving. Is it mediated by tryptophan and serotonin? *Psychopharmacology*, 178, 27-34.

Breslau, N., Schultz, L. R., Johnson, E. O., Peterson, E. L., & Davis, G. C. (2005). Smoking and the Risk of Suicidal Behavior. *Archives of General Psychiatry*, 62, 328-334.

Bricker, J.B., Leroux, B.G., Andersen M.R., Rajan, K.B., & Peterson, A.V. (in press). Parents' smoking cessation and children's smoking: Mediation by antismoking actions. *Nicotine and Tobacco Research*.

Bricker, J.B., Rajan, K.B., Andersen M.R., & Peterson, A.V. (2005). Does parents' quitting smoking encourage young adults to quit smoking?: A prospective study. *Addiction*, 100, 379-386.

Carpenter, M. J., & Hughes, J. R. (2005). Defining quit attempts: What difference does a day make? *Addiction*, 100, 257-258.

Chiamulera, C. (2005). Cue reactivity in nicotine and tobacco dependence: A "dual-action" model of nicotine as a primary reinforcement and as an enhancer of the effects of smoking-associated stimuli. *Brain Res. Rev.*, 48, 74-97.

Coleman, T., Pound, E., Adams, C., Bauld, L., Ferguson, J., & Cheater, F. (2005). Implementing a national treatment service for dependent smokers: Initial challenges and solutions. *Addiction*, 100 (Suppl2), 12-18.

Conklin, C. A., & Perkins, K. A. (2005). Subjective and reinforcing effects of smoking during negative mood induction. *Journal of Abnormal Psychology*, 114, 153-164.

Dar, R. & Frenk, H. (2004). Do smokers self-administer pure nicotine? A review of the evidence. *Psycho-pharmacology*, 173, 18-26.

Dar, R., Stronguin, F., & Etter, J.-F. (2005). Assigned versus perceived

placebo effects in nicotine replacement therapy for smoking reduction in Swiss smokers. *Journal of Consulting and Clinical Psychology*, 73, 350-353.

Ebbert, J. O., Klinkhammer, M. D., Stevens, S. R., Rowland, L. C., Offord, K. P., Ames, S. C., & Dale, L. C. (2005). A survey of characteristics of smokeless tobacco users in a treatment program. *American Journal of Health Behavior*, 29, 25-35.

Fagerström, K. (2005) The nicotine market: An attempt to estimate the nicotine intake from various sources and the total nicotine consumption in some countries. *Nicotine and Tobacco Research*, 7.

Fagerström, K. O. (2005). Can reduced smoking be a way for smokers not interested in quitting to actually quit. *Respiration*, 72.

Fagerström K., & Lewin F. (2005). Treatment of Tobacco Dependence. In *Lung Cancer: Principles and Practice*. Pass et al (Eds). Philadelphia, PA: Lippincott Williams & Wilkins.

Fagerström K., & Rennard S. (2005). Treatment of tobacco dependence. In *Pulmonary Rehabilitation*. Donner et al (Eds). London: Hodder Arnold.

Friend, K. B., Malloy, P. F., & Sindelar, H. A. (2005). The effects of chronic nicotine and alcohol use on neurocognitive function. *Addictive Behaviors*, 30, 193-202.

Glantz, S. A., & Mandel, L. L. (2005). Since school-based tobacco prevention programs do not work: What should we do? *Journal of Adolescent Health*, 36, 157-159.

Gomez, C., Berlin, I., Marquis, P., & Delcroix, M. (2005). Expired air carbon monoxide concentration in mothers and their spouses above 5 ppm is associated with decreased fetal growth. *Preventive Medicine*, 40, 10-15.

Grabus, S. D., Martin, B. R., Batman, A. M., Tyndale, R. F., Sellers, E., & Damaj, M. I. (2005). Nicotine physical dependence and tolerance in the mouse following chronic oral administration. *Psychopharmacology*, 178, 183-192.

Gray, N.J., Benowitz, N.L., Connolly, G.N., Dresler, C., Fagerström, K., Jarvis, M.J., et al. (2005). Toward a long term comprehensive nicotine policy. *Tobacco Control*, 14,(3).

Henningfield, J. E., & McLellan, A. T. (2005). Medications work for severely addicted smokers: Implications for addiction therapists and primary care physicians. *Journal of Substance Abuse Treatment*, 28, 1-2.

Hitsman, B., Spring, B., Wolf, W., Pingitore, R., Crayton, J. W., & Hedeker, D. (2005). Effects of acute tryptophan depletion on negative symptoms and smoking topography in nicotine-dependent schizophrenics and nonpsychiatric controls. *Neuropsychopharmacology*, 30, 640-648.

Jacobsen, L. K., Krystal, J. H., Mencl, W. E., Westerveld, M., Frost, S. J., & Pugh, K. R. (2005). Effects of smoking and smoking abstinence on cognition in adolescent tobacco smokers. *Biological Psychiatry*, 57, 56-66.

McNeill, A., Raw, M., Whybrow, J., & Bailey, P. (2005). A national strategy for smoking cessation treatment in England. *Addiction*, 100 (Suppl2), 1-11.

Moolchan, E. T., Zimmerman, D., Sehnert, S. S., Zimmerman, D., Huestis, M. A., & Epstein, D. H. (2005). Recent marijuana blunt smoking impacts carbon monoxide as a measure of adolescent tobacco abstinence. *Substance Use & Misuse*, 40, 231-240.

Munafò, M. R., Roberts, K., Johnstone, E. C., Walton, R. T., & Yudkin, P. L. (2005). Association of serotonin transporter gene polymorphism with nicotine dependence: No evidence for an interaction with trait neuroticism. *Personality & Individual Differences*, 38, 843-850.

Nollen, N.L., Adewale, S., Okuyemi, K.S., Parakoyi, B., & Ahluwalia, J.S. (2004). Smoking cessation practices of Nigerian physicians. *Journal of the National Medical Association*, 96, 838-842.

Okuyemi, K., Ahluwalia, J.S., Banks, R., Harris, K.J., Mosier, M., & Powell, J. (2004). Differences in smoking and quitting experiences by

levels of smoking among African Americans. *Ethn Dis*, 14, 127-133.

Okuyemi, K.S., Ebersole-Robinson, M., Nazir, N., Ahluwalia, J.S. (2004). African American menthol and non-menthol smokers: Differences in smoking and cessation experiences. *Journal of the National Medical Association*, 96, 1208-1211.

Patten, C.A., Lopez, K, Thomas, J.L., Offord, K.P., Decker, P.A., Pingree, S., et al. (2004). Reported willingness among adolescent nonsmokers to help parents, peers, and others to stop smoking. *Preventive Medicine*, 39, 1099-1106.

Philbin, S.D., Vann, R.E., Varvel, S.A., Covington III, H.E., Rosecrans, J.A., James, J.R., et al. (2005). Differential behavioral responses to nicotine in Lewis and Fischer-344 rats. *Pharmacology Biochemistry and Behavior*, 80, 87-92.

Pyle, S. A., Haddock, C. K., Hymowitz, N., Schwab, J., & Meshberg, S. (2005). Family rules about exposure to environmental tobacco smoke. *Families, Systems, & Health*, 23, 3-16.

Raw, M., McNeill, A., & Coleman, T. (2005). Lessons from the English smoking treatment services. *Addiction*, 100 (Suppl2), 84-91.

Richter, K., Ahluwalia, H.K., Resnicow, K., Nazir, N., Mosier, M.C., & Ahluwalia J.S. (2004). Cigarette smoking among marijuana users in the United States. *Substance Abuse*, 25, 55-62.

Sarna, L., Bialous, S. A., Wewers, M. E., Froelicher, E. S., & Danao, L. (2005). Nurses, smoking, and the workplace. *Research in Nursing & Health*, 28, 79-90.

Sayette, M. A., Loewenstein, G., Kirchner, T. R., & Travis, T. (2005). Effects of smoking urge on temporal cognition. *Psychology of Addictive Behaviors*, 19, 88-93.

Sherman, S.E., Yano, E.M., Lanto, A.B., Simon, B.F., & Rubenstein, L.V. (in press). Smokers' interest in quitting and services received: Using practice information to plan quality improvement and policy for smoking cessation. *Am J Med Qual*.

Sherman, S.E., Fu, S.S., Joseph, A.M., Lanto, A.B., & Yano E.M. (in press). Gender differences in smoking cessation services received among veterans. *Women's Health Issues*.

Sherman, S.E., Joseph, A.M., Yano, E.M., Simon, B.F., Arikian, N., Parkerton, P., et al. (in press). Characteristics of VA facility smoking cessation programs and practices. *Mil Medicine*.

Shiffman, S., Di Marino, M. E., & Pillitteri, J. L. (2005). The effectiveness of nicotine patch and nicotine lozenge in very heavy smokers. *Journal of Substance Abuse Treatment*, 28, 49-55.

Slotkin, T. A., Seidler, F. J., Qiao, D., Aldridge, J. E., Tate, C. A., Cousins, M. M., et al. (2005). Effects of prenatal nicotine exposure on primate brain development and attempted amelioration with supplemental choline or Vitamin C: Neurotransmitter receptors, cell signaling and cell development biomarkers in fetal brain regions of Rhesus monkeys. *Neuropsychopharmacology*, 30, 129-144.

Strasser, A. A., Kaufmann, V., Jepson, C., Perkins, K. A., Pickworth, W. B., Wileyto, E. P., et al. (2005). Effects of different nicotine replacement therapies on postcessation psychological responses. *Addictive Behaviors*, 30, 9-17.

Williams, G. C. (2005). It's time practitioners help families clear the air of secondhand smoke: A commentary on Pyle et al. (2005). *Families, Systems, & Health*, 23, 25-29.