



New York State
Office of Alcoholism & Substance Abuse Services
Addiction Services for Prevention, Treatment, Recovery

ADDICTION FYI YEARBOOK 2009/2010 - Part 2

This yearbook is a compilation of all the Addiction Medicine Briefs and FYI's sent out during the later part of the year 2009 and the early part of 2010.

There will be at least one question asked per FYI and free CASAC credit can be earned by answering 75% or more of the questions correctly.

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New Smoking/Nicotine Products



In England, pubs are using a new type of electronic cigarette to circumvent the country's smoking ban which went into effect on July 1, 2008. The Ban prohibits smokers from lighting up in enclosed public places. However, the ban does not extend to electronic cigarettes which the pubs are using as a loophole in the law in order to get their clients to return to their establishments.

"E-cigarettes" or electronic cigarettes are made of long stainless steel tubes with chambers to hold replaceable cartridges filled with various concentrations and flavors of liquid nicotine. They are powered by a rechargeable battery, and because the vapor produced is odorless and doesn't contain tar or carbon monoxide. The manufacturers say that their products don't put people at risk for second-hand smoke inhalation. The U.S. Food and Drug Administration (FDA) is investigating manufacturers' health claims about electronic cigarettes and deciding whether to ban the nicotine-delivery devices as the agency did with nicotine lollypops and drinks.

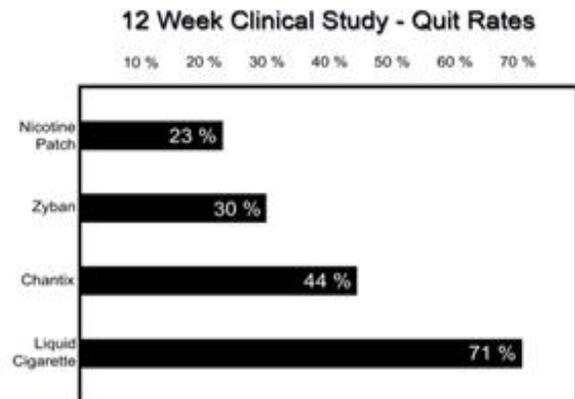
In another smoking development, a "liquid cigarette" has been developed and approved by the FDA for clinical study. The 12-week clinical study of the new smoke-cessation device called Smoke-Break had its data released in September 2008. The results of the study show 71 percent of the study participants smoke-free after 12 weeks.



Smoke-Break is a "liquid nicotine cigarette" that resembles an unlit cigarette in size and shape. The clear tube contains a cherry-flavored gel along with 1.5 milligrams of nicotine, about as much as is delivered by smoking one cigarette. Users consume the liquid by lifting the tube to their mouths, and sipping through a mouthpiece, much like they would draw on a cigarette.

"There were no serious adverse events during the study," said Dr. Carl E. Olson, Chairman of the Radiation Oncology Department at Columbia St. Mary's in Milwaukee," and only a few minor events, such as sore throat or heartburn. The real surprise for me was the rate of smoke cessation. It is unprecedented."

The study began in the spring of 2008 with 52 smokers in a 12-week clinical study. The results of the study: 71.1 percent of the subjects were still smoke-free at the end of 12 weeks, as verified by CO monitor testing.



The last new product is the Camel Dissolvables which include Camel Sticks, Camel Orbs, and Camel Strips. The products purportedly melt in the mouth within three to 15 minutes. RJR said the Strips melt fastest, the toothpick-like Sticks dissolve in about 10 minutes, and the pellet-size Orbs last the longest.



The nicotine delivery of the products is said to be high: whereas a cigarette smoker typically takes in about 1 milligram of nicotine, the Camel Dissolvables are said to deliver about 0.6 to 3.1 mg of nicotine each.

Remember, the smoke of a burning cigarette or cigar contains the toxins and carcinogens.

Impact of Drugs on Liver Fibrosis

Steatosis: fatty degeneration of the liver

Fibrosis: formation of fibrous (fibers of connective tissue) as a reactive process. This is seen in cirrhosis and if greater than 50% of the liver is scarred, normal liver functions are impaired.

Chronic viral hepatitis includes mainly chronic hepatitis B and chronic hepatitis C infection. They are the major cause of chronic liver disease and account for the increased morbidity and mortality seen in hepatitis. The spectrum of liver injury caused by hepatitis B and C is broad. The cause of fibrosis and its progression is not completely understood. It is known that older age, male gender and a high viral load are factors in chronic B fibrosis. Older age, older age at the time of acute infection, male gender and alcohol intake all are factors in chronic C fibrosis progression. Insulin resistance and liver steatosis have recently been recognized as important factors in fibrosis progression in chronic hepatitis C. The role of insulin resistance and steatosis is not as clear in chronic hepatitis B.

In a paper by Emmanuel Tsochatzis et al in the *Scandinavian Journal of Gastroenterology* (2009, pp 1- 8), the researchers sought to evaluate the association of smoking and progression of steatosis and fibrosis in chronic hepatitis B and C. They studied 271 patients with chronic hepatitis B or C who had undergone liver biopsies. Their conclusions were that heavy smoking is associated with severe fibrosis in chronic hepatitis C but not chronic hepatitis B. Heavy smoking was also significantly associated with steatosis in chronic hepatitis C and this may be the link between smoking and ultimate progression to fibrosis.

In a study by C. Hézode et al in the journal *Hepatology* in 2005 (42(1):63-71), the researchers evaluated daily cannabis smoking as a risk for progression of fibrosis in chronic hepatitis C. Research has shown that cannabinoids present in *Cannabis sativa* (marijuana) exert biological effects in the brain via cannabinoid receptors CB1 and CB2. It has been demonstrated that CB1 and CB2 receptors regulate progression of experimental liver fibrosis. Studying 270 untreated patients with chronic hepatitis C who were undergoing liver biopsy, the relationship between cannabis use and fibrosis progression rate or fibrosis stage was assessed. The patients were categorized as: non-cannabis users (52.2%), occasional users (14.8%) and daily users (33.0%).

The conclusion of the authors was that daily cannabis smoking is significantly associated with fibrosis progression during chronic hepatitis C infections. Patients with ongoing CHC should be advised to refrain from regular cannabis use.

Impact of Diet on Liver Fibrosis

Host factors and virus characteristics impact the progression of chronic hepatitis and the response to therapy in patients with HCV-related chronic hepatitis. There has been an increase of interest in complementary food and chemopreventers (foods and food components that have antimutagenic or anticarcinogenic effects) to treat various liver diseases including cirrhosis. It is known that diet influences body mass index (BMI), iron content in the liver, insulin, enzyme activities and metabolic pathways in liver cells. Foods have been reported to exert protective or toxic effects on the liver in animal models and humans.

C. Loguercio et al in the *Am J Gastroenterology* (2008; 103(12):3159-3166) undertook a prospective study to evaluate the effect of diet on the severity of liver damage and on the response to antiviral therapy in patients with HCV-related chronic hepatitis. A total of 1,084 patients with HCV-related chronic hepatitis were enrolled in the study. Excluded from the study were patients with chronic HBV and HIV infection, decompensated cirrhotics, and patients with decompensated diabetes, kidney diseases, pulmonary diseases, collagen diseases and tumors.

The study, carried out on a large number of HCV patients and controls, demonstrated that dietary intake affects liver cells and indirectly, response to treatment. **A high intake of calories, carbohydrates, and lipids was associated with more severe fibrosis.** This finding illustrates that diet is an important factor in the management of patients affected by HCV.

One surprising finding was that both groups consumed a similar amount of alcohol. Moreover, the number of heavy drinkers (> 40 g/day) was similar in the two groups. Alcohol affects HCV by enhancing oxidative stress and HCV replication. Alcohol intake also increases insulin resistance in obese individuals by interfering with the movement of lipids between adipose (fat) tissue and the liver. Alcohol also accelerates the progression to fibrosis in HCV-infected patients.

The paper noted that obesity and diabetes have been found to be risk factors for hepatocellular carcinoma. Taken together, the data suggest a synergistic mechanism whereby alcohol, diet and HCV alter the metabolism of lipids and carbohydrates, which in turn, results in liver damage. Viral characteristics, including RNA, viremia (virus in the bloodstream) and genotype are important in determining the outcome of treatment with interferon (IFN) and ribavirin in HCV-positive patients. Weight reduction has been shown to reduce fatty infiltration of liver cells and abnormal liver enzymes and improve fibrosis in patients with chronic hepatitis C.

The finding that alcohol intake was similar in patients and controls might indicate that physicians need to consider this an important aspect of treatment and advise their patients to abstain from drinking. It is noted by the authors of the study that since there are frequent contacts with medical professionals during treatment, the HCV patient should undergo regular counseling about this issue in order to improve their awareness of alcohol-related problems.

This study suggests that educational programs aimed at correcting the overall lifestyle of liver disease patients should be part of the therapeutic strategy of HCV patients. The simple recommendation of a low calorie, low fat diet may not be effective alone.

Treatment of Stimulant Dependence – Part I

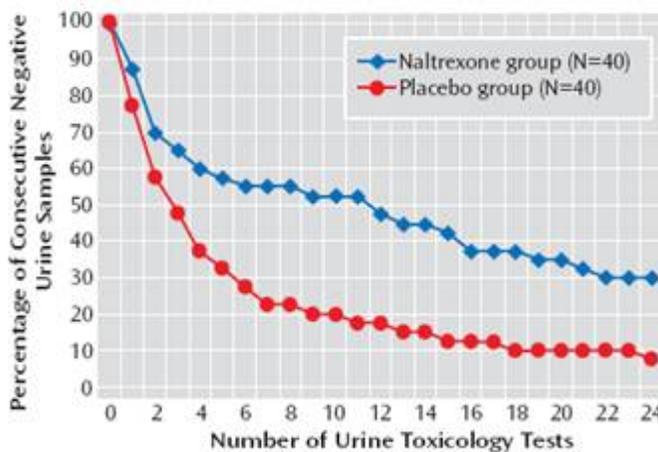
Naltrexone for the Treatment of Amphetamine Dependence

In the September 2, 2008, American Journal of Psychiatry, Jayaram et al. published an article on the use of naltrexone in patients who are amphetamine dependent. There are several important points that are worth considering: there is no approved treatment for amphetamine dependence, though almost all addiction medications have been tried; naltrexone is an opiate blocker which is used for the treatment of alcohol dependence (Revia); the total number of amphetamine users worldwide is estimated at 34 million, which exceeds the estimates for cocaine and heroin users combined.

While amphetamines work primarily via stimulation of the reward centers in the brain through the dopamine system, research has found that there is a functional interaction of the dopamine and opioid system in stimulant users and that naltrexone appears to blunt the effect of the stimulant and reduce craving for the drug.

In this study, 80 patients were enrolled and 55 completed the study. There was no statistical difference in completers in the treatment or placebo group. Medication compliance was measured by looking at urinary metabolites of naltrexone and there was a positive correlation between adherence to medication and the

FIGURE 4. Survival Curves Representing Continuous Rates of Abstinence for the Naltrexone and Placebo Groups During the 12-Week Trial (Intention-to-Treat Analysis)



number of amphetamine negative urine samples.

The naltrexone group showed a reduction in craving during the 12 week study as compared to the placebo group. Treatment with naltrexone did not produce any serious adverse effects nor did it lead to other drug use. The placebo group did attend relapse prevention groups and this may have accounted for some of the success seen in this group.

Limitations of the study include only looking at treatment outcomes for three months, mental health diagnoses were excluded, and a small sample size.

Methadone for the Treatment of Cocaine Dependence

A comprehensive study of an urban methadone clinic by Borg et al. (*J Addict Dis*, 1999) conducted over an 18 month period on 133 clinic patients showed that with effective methadone maintenance using adequate dosages, the majority of patients remain in treatment and reduce cocaine abuse as well as illicit opioid use. The question remains as to whether this was the methadone or just good treatment?

In new research presented in the journal, *European Neuropsychopharmacology* (2008), the effects of methadone on cocaine-dependent rats was evaluated and it was found that the rats did not experience

cocaine highs after getting methadone. The researchers also found that methadone appeared to have a "resetting" effect on portions of the rats brains responsible for addictive behavior in that among the rats given cocaine and then methadone, the regions of the brain looked similar to how they appeared in the rats that were never exposed to cocaine.

The National Institute for Drug Abuse is also researching the effects of buprenorphine on cocaine-dependency.

The search continues for an effective medication for stimulant dependence. It should be remembered that behavioral treatment is important as well, even if using addiction medications.

The second part of this FYI series will discuss some of the current medications being researched for the treatment of stimulant dependence.

Treatment of Stimulant Dependence – Part II (Medications)

Demand for amphetamine dependence treatment has increased 8-fold from 1992 to 2005 (SAMHSA 2006).

Drop out rates in cocaine dependence treatment programs frequently exceed 50% (Alterman et al, 1996) and many patients do not benefit from psychotherapy alone. Cocaine withdrawal symptoms (dysphoria, fatigue, sleep disturbance, appetite change, irritability) contribute to difficulties in quitting.

- **Modafinil**

- Currently approved for the treatment of narcolepsy
- A mild stimulant so it may reduce cocaine withdrawal symptoms
- Enhances glutamate neurotransmission (glutamate depletion is seen in chronic cocaine use)
- Blocks euphoric effect of cocaine (Dackis et al, 2003; Malcolm et al, 2006)
- Improved abstinence rates in several trials using 200 mg and 400 mg a day (Dackis, 2007)
- Promotes wakefulness and may cause insomnia
- Avoid use in patients with bipolar disorder, psychosis and patients with left ventricular hypertrophy or mitral valve prolapse

- **Propranolol**

- Beta blocker used to treat angina and hypertension
- Shows promise for treatment of severe cocaine withdrawal symptoms
- Reduces the impact of the neurotransmitter adrenaline, thus reduces anxiety and agitation
- Several trials show that it promotes abstinence from cocaine (Kampman et al, 2001,2006)
- Main side effect is sedation, though must avoid use in patients with history of cocaine induced cardiac ischemia (tissue that is not receiving enough oxygen and is at risk for cell death)

Pharmacologic strategies for relapse prevention aim to reduce cocaine craving and many are GABAergic compounds (compounds that promote GABA release or conservation; GABA inhibits the effect of dopamine)

- **Gamma-vinyl GABA**

- May be considered for abstinence initiation and relapse prevention
- Antiepileptic (anti seizure) medication that elevates brain GABA concentrations
- In several trials it has shown efficacy in the treatment of stimulant dependence (Brodie et al 2003, 2005)
- Used in other countries, though not in US due to association with visual field defects (a loss of part of the usual field of vision)

- **Topiramate**

- Anticonvulsant (also used to treat depression and bi-polar disorder)

- Increases brain GABA, facilitates GABA transmission, and weakens Glutamate's effect
- Can cause sedation and memory problems, especially if the dose is titrated rapidly
- Cannot be used if there is a history of kidney stones

- **Tiagabine**
 - Approved for treatment of seizures
 - Raises GABA levels
 - Well tolerated in small sample research protocol

- **Disulfiram**
 - Disulfiram (Antabuse) blocks enzymatic degradation of cocaine and dopamine leading to extremely high cocaine and dopamine levels when cocaine is ingested
 - Cocaine high is not increased, but instead there is increased anxiety which is unpleasant (published trials: Carroll et al 1998, 2004; George et al 2000; Petrakis et al 2000)

- **TA-CD Vaccine**
 - Stimulates the production of cocaine-specific antibodies that bind to cocaine and prevent them from entering the brain, thus blunting the effect
 - Several trials are ongoing

Currently, there are no medications approved by the FDA for the treatment of stimulant dependence.

Alcohol Consumption: Risks and Benefits

Alcohol use has a complicated role in health, though it is clear that excessive use can cause considerable morbidity and mortality. It is difficult to be certain about the risks and benefits of alcohol consumption. The following data pertain to only the recommended limits of drinking – one drink per day for women and two drinks per day for men (Mukamal et al, *Current Atherosclerosis Reports*, 2008). **It must be noted that this review does not pertain to alcohol binge use, chronic alcohol misuse or alcohol dependence.**

- Atherosclerotic Cardiovascular Disorders
 - Coronary heart disease
 - 20 percent to 40percent lower risk than abstainers
 - Most positive effects seen in men
 - Ischemic stroke
 - Positive effects are seen with lower alcohol use and not as significant as seen in coronary heart disease
 - Peripheral vascular disease
 - Effect requires more research

Table 1. Qualitative effects of light/moderate and heavier alcohol intake on cardiovascular risk factors

Cardiovascular risk factor	Light/moderate alcohol intake	Heavier alcohol intake (> 2 drinks per day)
HDL-C	↑↑	↑↑↑
Triglycerides	↑	↑↑
LDL-C	↔	↔
Blood pressure	↔	↑↑
Platelet function/ coagulability	↓	↓↓
Inflammation	↓	↑
Atrial fibrillation	↔	↑↑

HDL-C—high-density lipoprotein cholesterol; LDL-C—low-density lipoprotein cholesterol.

- Cancer
 - **Overall the evidence does not show that there is any level of consumption below which there is not an increased risk of cancer**
 - The risk of mouth, pharynx, and larynx cancer is approximately 25 percent higher per drink per day
 - Esophageal cancer is similar to above but complicated by increased risk of smoking
 - Risk of colon cancer increases as drink consumption increases over 2 drinks per day

- Breast cancer risk shows a 10 percent increase for each 2/3 glass of alcohol per day due to effect on sex steroid hormones
- Miscellaneous disorders
 - Lower risk of gallstones with moderate alcohol consumption
 - Lower risk of diabetes with moderate alcohol consumption
 - Lower risk of congestive heart failure with moderate alcohol consumption
 - Possible lower risk of dementia with moderate alcohol consumption
 - Higher risk of hemorrhagic stroke with moderate alcohol consumption
 - Detrimental effects of alcohol consumption have been seen on sex steroid hormones, folate metabolism and viral hepatitis

Tobacco in Cigarettes Grow Bacteria – A Potential Health Risk

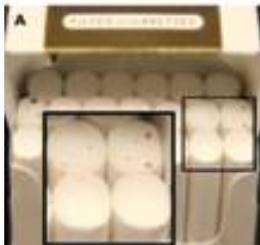
An article in *Tobacco Control* (2008, Vol. 17), highlights another potential health risk associated with tobacco in a cigarette or in the smokeless form. Several studies were highlighted:

- One study showed the presence of bacteria, bacterial toxins, fungal spores and fungal toxins in cigarette tobacco.
- A second study showed the presence of 23 different species of bacteria in cigarette tobacco.
- An additional study revealed that in 14 brands of cigarettes, the fungal spores were identified and that *Aspergillus fumigatus* was the most prevalent one.
 - Invasive aspergillus is a significant cause of morbidity and mortality in immunocompromised patients.

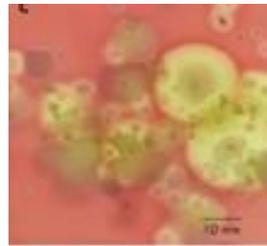
Several questions were raised by the *Tobacco Control* study. Would the bacterial growth occur on tobacco dust and microparticulates that could reach the lung? The answer was that 90 percent of tobacco particulates grew bacteria and when filters were looked at, the tobacco flakes in the filters all grew bacteria (11 different brands).

A question that will remain unanswered at present is what happens in the long term smoker, who does not clear deposited tobacco smoke particulates (tar) from the lung due to: impaired mucociliary clearance, reduced host defenses, chronic pulmonary inflammation and recurring respiratory infections?

It is known that chronic inflammation is associated with cancer. It is also known that tobacco associated inflammation is associated with lung cancer and other non-cancerous pulmonary diseases. Chronic inflammation can be caused by bacterial derived toxins. So is the smoker setting up the chain of events that will ultimately lead to cancer by inhaling particulate matter that has bacteria and fungi on it?



Tobacco flakes on filters of a new pack of cigarettes



Numerous bacterial colonies after 24 hours on a blood agar plate

Contaminated Cocaine - Update

It has been reported by the Departments of Health in New Mexico, Canada and Delaware that there have been several cases of agranulocytosis (*uh-gran-yuh-loh-sahy-toh-sis*) following the use of cocaine. This condition leaves patients unable to fight off infections.

The unusual condition can be fatal because it compromises the immune system. It appears that the condition could be related to using cocaine that is contaminated with levamisole. The U.S. Department of Justice reported that between January and April 2008, levamisole found in tested cocaine had increased from 9 percent to 19 percent.

Levamisole is a cancer (antineoplastic) medication used in cancer treatment. It is also widely used in veterinary medicine in the de-worming of many animals. Serious side effects have been reported with the use of levamisole including: allergic reactions (difficulty breathing; closing of the throat; swelling of the lips, tongue, or face; or hives); decreased bone marrow function or blood problems (fever or chills; or signs of infection); nervous system problems (confusion or loss of consciousness, extreme fatigue, memory loss, muscle weakness, numbness or tingling; seizure, speech disturbances); and others.

People who use cocaine and experience one or more of the following should see a doctor and inform them of their cocaine use:

- Persistent or recurrent fever and chills
- Worsening swollen glands
- Painful sores in the mouth or around the anus
- Frequent, persistent or worsening skin infections
- Pneumonia
- Worsening or persistent sore throat
- Thrush - a white coating of the mouth, tongue, or throat
- Other unusual infection

ADULTERATED COCAINE UPDATE – 09/ 22 /09

According to the Drug Enforcement Administration and State testing laboratories, the percentage of cocaine specimens containing levamisole has increased steadily since 2002, with levamisole now found in over 70 percent of the illicit cocaine analyzed in July, 2009. In addition, a recent analysis in Seattle, Washington, found that almost 80 percent of the individuals who test positive for cocaine also test positive for levamisole.

According to the SAMHSA Alert, substance abuse treatment providers, clinicians, outreach workers, and individuals who abuse cocaine need to be aware of the following:

“A dangerous substance, levamisole, is showing up with increasing frequency in illicit cocaine powder and crack cocaine. Levamisole can severely reduce the number of white blood cells, a problem called agranulocytosis. THIS IS A VERY SERIOUS ILLNESS THAT NEEDS TO BE TREATED AT A HOSPITAL. If you use cocaine, watch out for:

- high fever, chills, or weakness
- swollen glands
- painful sores (mouth, anal)
- any infection that won't go away or gets worse very fast, including sore throat or mouth sores, skin infections, abscesses, thrush (white coating of the mouth, tongue, or throat) and/or pneumonia (fever, cough, shortness of breath).

Individuals are encouraged to report suspected and confirmed cases of agranulocytosis that are associated with cocaine abuse to their respective state health departments. Cases can also be reported to local Poison Control Centers (1-800-222-1222); these centers may also provide assistance in clinical management and additional reporting.

RESOURCES

- Update: Neutropenia and adulterated cocaine use - Alberta Health Services 1/27/09 (Update of original alert, Agranulocytosis Related to Cocaine Adulterated with Levamisole dated 11/21/08)
- Department of Health Investigates Unusual Condition that Harms Immune System Potential Cocaine Contamination Could Be Cause - New Mexico Department of Health Press Release 1/16/09
- Users get tests for bad cocaine - Straight.com Vancouver's Online Source 1/1/09
- Public Health Advisory - Tainted Cocaine - British Columbia Press Release 12/11/08
- Agranulocytosis in Cocaine Users - Toronto Public Health Surveillance Alert 12/10/08
- Toxic cocaine on streets, health officials warn - Calgary Herald 11/29/08
- Delaware Health Advisory LEVAMISOLE - Delaware Division of Public Health Press Release 9/2/05
- SAMHSA website: <http://www.samhsa.gov/newsroom/advisories/090921vet5101.aspx>

Soma

Soma is the trade name for the prescription drug carisoprodol.

Carisoprodol is a centrally acting skeletal muscle relaxant that is a colorless, crystalline powder with a bitter taste.

Soma is sold under the name Sanoma or Carisoma in other countries. Carisoprodol is available by itself or mixed with aspirin or codeine and caffeine (Soma compound with codeine). Carisoprodol was developed in the late 1950's by Wallace Laboratories.

Carisoprodol abuse has escalated in the last decade in the United States. According to 2007 National Survey on Drug Use and Health (NSDUH) data, non-medical use by U.S. population aged 12 and older of Soma (1.1%) was similar to or greater than other commonly abused schedule IV controlled drugs such as Klonopin (1.5%), and Librium (0.3%). Street Names include: Ds, Dance, Las Vegas Cocktail (soma and vicodin) and Soma Coma (soma and codeine).

Carisoprodol is metabolized into hydroxycarisoprodol, hydroxymeprobamate and meprobamate.

Meprobamate is a schedule IV drug that has sedative properties and significant potential for abuse, dependence, overdose and withdrawal. Carisoprodol is considered a prodrug as it is an active drug only when metabolized. Due to the significant abuse potential, some countries have taken Carisoprodol off of the market (Sweden in 2007). According to the DEA, meprobamate was introduced as an anti-anxiety agent in 1955 and is prescribed primarily to treat anxiety, tension, and associated muscle spasms. More than 50 tons are distributed annually in the United States under its generic name and brand names such as Miltown® and Equanil®.

Carisoprodol use causes analgesia, anxiolysis (decreased anxiety), muscle relaxation, sedation and somnolence. It has a rapid onset of action usually within 30 minutes and the effects can last two to six hours. It is metabolized in the liver and excreted by the kidneys.

Carisoprodol is taken orally in pill or tablet form. It is frequently taken in combination with other drugs and alcohol to enhance its effects. Soma and Vicodin is a common combination as is Soma, Xanax and Vicodin. In 2006, the FDA required labeling changes to the package insert to stress the risk of abuse and dependence.

Overdose can lead to chills, palpitations, vomiting, sedation, difficulty breathing, shock, coma and death.

Withdrawal symptoms include abdominal pain and cramps, depression, headache, insomnia and nausea. Soma should never be abruptly stopped but should be slowly tapered.

On November 23, 2009, the Department of Justice, DEA has given notice of a proposed rule change making Carisoprodol a Schedule IV drug.

Tobacco and Cannabis - Is There a Relationship to Depression and Suicide?

Cannabis is the most commonly used illicit psychoactive substance among our patients and approximately 90 percent of our patients smoke cigarettes. Is mental health negatively impacted by these substances?

Julie A. Pasco et al (British Journal of Psychiatry 2008) sought to investigate cigarette smoking as a risk factor for major depressive disorder by looking at a sample (165 women with major depressive disorder and 806 controls). Smoking was associated with increased odds for major depressive disorder (age-adjusted odds ratio (OR) =1.46, 95% CI 1.03 - 2.07). Compared with non-smokers, odds for major depressive disorder more than doubled for heavy smokers (>20 cigarettes/day). Their conclusions were that evidence from cross-sectional and longitudinal data suggests that smoking increases the risk of major depressive disorder in women. What was not explained was that depression could be associated with smoking and not be a causal event.

However, in another study it was noted that many studies have found an association of current smokers and suicide. In the overview of this subject by Hughes in *Drug and Alcohol Dependence* (2008), the term suicide refers to thoughts, behaviors, attempts or completed suicide.

There are several theories why smoking could be associated with suicide:

1. Smoking as a non-causal marker whereby smoking and the risk of suicide is caused by a third factor.

There are many risk factors for suicide that are seen also as risk factors for smoking:

- Younger age
- Non-white
- Lower income
- Less education
- Unmarried
- Unemployed
- Less religious
- Anxiety
- Depression
- Psychosis
- Substance use
- Low self esteem
- Risk taking
- Serious physical illness
- Impulsivity
- Aggression
- Anti-social personality disorder
- Fatalism

2. Smoking is a psychological or physical toxin i.e. smoking worsens mood. Smoking is known to decrease serotonin, thus worsening mood. Reductions in serotonin have been repeatedly linked to increased hostility, aggression, and importantly, to increased suicide. Chronic nicotine exposure reliably reduces serotonin and its metabolites in animals. Smoking can cause physical illness that can lead to depression and suicide as well.

3. There is an association with suicide and smoking cessation and smoking cessation medications. If one stops smoking this can lead to withdrawal that can increase depression (not seen in studies however). Varenicline (a prescription medication used to treat smoking addiction) has been linked weakly to suicide.

Cannabis

Cannabis is the most commonly used illegal psychoactive substance and many adolescents and young adults have used this drug. Pedersen in *Acta Psychiatr Scand* (2008) sought to look at the relationship of cannabis use in early teenage years to the late twenties (sample of over 2000 Norwegians) to depression and suicidal behaviors. The sample revealed that 36% had used cannabis at some point in their life (slightly lower than expected as compared to a US sample due to the fact that Norway has a very restrictive cannabis policy). He found no association with depression later on in life but did see that all levels of cannabis use were linked to later suicidal ideation. The odds ratio being 2.9 for later suicide attempts in the group who used cannabis 11 + times during the past 12 months with slightly lower odds ratios in the less regular users.

Why is there an association? The simplest explanation according to the author is that “doses of the active ingredient in cannabis, THC, affect serotonin and other neurotransmitters in a way that produces depressive or other mental health symptoms. However, no animal model supports this”. Another explanation is that there is an association between cannabis use and educational failure and other social problems. Also, there is an increase in suicide in drug users as compared to the general population. The cannabis user may have greater exposure to the drug using culture. Perhaps, the suicidal behaviors arise from these factors.

Finally, why an increase in suicidal behavior but not depression? Here again one could surmise that the use of cannabis leads to an increase in impulsivity and loss of control, factors impacting on suicidal behavior but not the development of depression.

Clearly, more studies are needed to determine which of these theories proves to be true.

Hepatitis and Liver Cancer

Notes from the January 2010 Report from the Institute of Medicine: A National Strategy for Prevention and Control of Hepatitis B and C

- Up to 5.3 million people - 2 percent of the U.S. population - are living with chronic hepatitis B or hepatitis C.
- These diseases are more common than HIV/AIDS in the U.S. Yet, because of the asymptomatic nature of chronic hepatitis B and hepatitis C, most people who have them are unaware until they have symptoms of liver cancer or liver disease many years later.
- Each year about 15,000 people die from liver cancer or liver disease related to hepatitis B and hepatitis C.
- In the next 10 years, about 150,000 people in the United States will die from liver cancer and end-stage liver disease associated with chronic hepatitis B and hepatitis C
- Hepatitis B and hepatitis C can be either acute or chronic:
 - The acute form is a short-term illness that occurs within the first six months after a person is exposed to hepatitis B virus (HBV) or hepatitis C virus (HCV) which cause hepatitis B and hepatitis C, respectively.
 - Although the number of people with acute hepatitis B is declining in the U.S., mostly because of the availability of hepatitis B vaccines, about 43,000 people still develop acute hepatitis B each year.
 - People at risk for hepatitis B include infants born to women with the disease and those who have sexual contact or share injection drug equipment with a person with the disease
 - African American adults have the highest rate of acute HBV infection in the United States and the highest rates of acute HBV infection occur in the southern region.
 - People from Asia and the Pacific Islands comprise the largest foreign-born population that is at risk for chronic HBV infection.
 - The diseases can become chronic, although this does not always happen and, particularly in the case of hepatitis B, the likelihood of this becoming a chronic disease depends on a person's age at the time of infection.
 - Persons likely to have chronic HCV infection include those who received a blood transfusion before 1992 and past or current injection-drug users (IDUs).
 - There is no vaccine for hepatitis C.

Viral Hepatitis Services (Recommendations)

- Due to the lack of health services related to viral hepatitis prevention at the federal, state, and local levels, a coordinated approach is necessary to reduce the numbers of new HBV and HCV infections and the illnesses and deaths associated with chronic viral hepatitis. Comprehensive viral hepatitis services should have five core components:
 - Outreach and awareness
 - Prevention of new infections
 - Identification of infected people
 - Social and peer support
 - Medical management of chronically infected people

- Federal and state governments should expand services to reduce the harm caused by chronic hepatitis B and hepatitis C. The services should include testing to detect infection, counseling to reduce alcohol use and secondary transmission, hepatitis B vaccination, and referral for or provision of medical management.
- Federal, state, and local agencies should expand programs to reduce the risk of hepatitis C virus infection through injection-drug use by providing comprehensive hepatitis C virus prevention programs. At a minimum, the programs should include access to sterile needle syringes and drug-preparation equipment because the shared use of these materials has been shown to lead to transmission of hepatitis C virus.
 - Health care for both IDUs and NIDUs is sporadic and typically received in hospital emergency rooms, corrections facilities, and STD clinics. Given that population's poor access to health care and services, it is important to have prevention and care services in settings that IDUs and NIDUs are likely to frequent or to develop programs that will draw them into care.
- The Centers for Disease Control and Prevention should provide additional resources and guidance to perinatal hepatitis B prevention program coordinators to expand and enhance the capacity to identify chronically infected pregnant women and provide case-management services, including referral for appropriate medical management.
- The Centers for Disease Control and Prevention and the Department of Justice should create an initiative to foster partnerships between health departments and corrections systems to ensure the availability of comprehensive viral hepatitis services for incarcerated people.
 - Correctional institutions should offer hepatitis B vaccination to all incarcerated persons and accelerated schedules for vaccine administration should be considered for jail inmates.
- The Health Resources and Services Administration and the Centers for Disease Control and Prevention should provide resources and guidance to integrate comprehensive viral hepatitis services into settings that serve high-risk populations such as STD clinics, sites for HIV services and care, homeless shelters, and mobile health units.

Conclusion

The current approach to the prevention and control of chronic hepatitis B and hepatitis C is not working. These diseases are not widely recognized as serious public health problems in the U.S. As a result, inadequate resources are being allocated to viral hepatitis prevention, control, and surveillance programs. Unless action is taken to prevent chronic hepatitis B and hepatitis C, thousands more Americans will die each year from liver cancer or liver disease related to these preventable diseases.

Barriers To The Use Of Addiction Medications

In an article by Alexander Walley et al. in the *Journal of General Internal Medicine* (2008), 235 physicians in Massachusetts were surveyed and it was found that non-prescribers of buprenorphine said they would be more likely to prescribe if barriers were eliminated.

The barriers noted in the paper include:

- insufficient nursing support;
- insufficient office support;
- lack of institutional support;
- office staff stigma;
- low demand;
- insufficient physician knowledge;
- insufficient staff knowledge;
- payment issues;
- pharmacy issues;
- lack of immediate telephone access to an Addiction specialist;
- inability to refer to a chemical dependence treatment program;
- fear of taking on increased medicolegal risks;
- overly complicated patients; and
- issues of medication diversion.

NYS OASAS held Addiction Medicine Forums several years ago and the information was presented at the Annual ASAM meeting which was held in Miami. Looking at all addiction medications, the benefits of incorporating them into practice included:

- retention in treatment environment;
- fostering a sense of teamwork between clinicians and medical staff;
- reducing withdrawal effects;
- fostering the concept of “addiction” as a medical condition;
- including more tools in clinicians tool box;
- promoting individualized treatment planning;
- reducing craving to enhance ego functioning;
- increasing staff’s general knowledge;
- improving interdisciplinary approaches; and
- including more physicians who may enter the field if the chemical dependency field is open to the use of addiction medications.

The barriers to incorporating addiction medicines were:

- clinician concerns of losing control of case;
- lack of evidence for or against addiction medicines in the treatment setting;

- lack of training for counselors about the neuroscience of addiction;
- patients' failure to comply with taking medications;
- counselors may believe that "Drug Free" is the only way;
- treating cravings with medicines is new'
- lack of funding;
- doctor's hours limited in some OASAS levels of care;
- lack of assessment resources;
- need for increase in nursing time;
- cost of medications; and
- the side effects and organizational responsibility.

The forums and the Walley paper continue to provide valuable information in how we approach the use of addiction medications as a system. The key areas for counselors and medical staff are:

- the importance of communication between disciplines;
- the importance of each respecting the others' role;
- counselor concerns that patients who were prescribed addiction medications would become the cases that would be "controlled" by the physicians;
- the reduction of stigma; and
- increasing the knowledge base.

THC Patch

Marijuana is the most commonly used illicit drug in the United States. According to the 2008 National Survey on Drug Use and Health, more than 15 million Americans (nearly 5% of the U.S. population) age 12 or older used marijuana at least once in the month prior to being surveyed. More than half of the individuals addicted to marijuana experience significant withdrawal symptoms if they try to quit. The withdrawal symptoms contribute to the relapse and continuation of marijuana use. Withdrawal symptoms are similar to those experienced by people who quit cigarettes, cocaine and other drugs. Nearly two-thirds of marijuana users experience pronounced withdrawal symptoms that can include anxiety, irritability, depression, mood swings, and sleep problems.

Marijuana contains more than 400 chemicals, including many of the harmful substances found in tobacco smoke. Smoking one marijuana cigarette deposits about four times more tar into the lungs than a filtered tobacco cigarette and Harvard University researchers report the risk of a heart attack is five times higher than usual in the hour after smoking marijuana.

AllTranz, Inc., a specialty pharmaceutical company focused on developing drugs to treat a variety of neurologic and inflammatory disorders, today announced that it has been awarded a \$4 million research grant from the National Institute on Drug Abuse (NIDA), a component of the National Institutes of Health (NIH), to advance the company's transdermal tetrahydrocannabinol (THC) patch for the treatment of marijuana dependence and withdrawal.

AllTranz's patented system presents significant advancements in transdermal drug delivery. The novel drug delivery technology allows for the non-invasive, non-oral, controlled delivery of THC. The non-plant based THC patch is designed to decrease the chance of side effects including drowsiness, dizziness, and the "high" feeling, as compared to ingesting a capsule, using a spray inhaler, or smoking marijuana. The patch will reduce dosing frequency and is expected to improve marijuana withdrawal and addiction symptoms, similar to nicotine patch treatment for tobacco smoking cessation.

The patch is initially being developed for marijuana withdrawal and dependence. Additionally, because of THC's wide-ranging therapeutic and clinical benefits, AllTranz is proceeding with development of the patch for other indications, including cancer chemotherapy nausea and vomiting, AIDS patient appetite stimulation, and multiple sclerosis (MS) pain. Separate Phase II clinical trials would be pursued for each indication.

Bupropion – Zyban

Research presented at the 2010 American Society of Addiction Medicine Annual Medical - Scientific conference revealed that a significant percentage of substance use counselors - about 40 percent - have little knowledge of the tobacco cessation medication bupropion. Tanja C. Rothrauff, PhD, of the Institute for Behavioral Research at the University of Georgia in Athens and colleagues used data from the National Treatment Center Study (NTCS). Results indicated that 38 percent of counselors tested in 2002-2004 were unaware of the effectiveness of bupropion and 44 percent of counselors in 2007-2008 were unfamiliar with the medication's usefulness for tobacco cessation.

Bupropion was originally marketed as an antidepressant (Wellbutrin®). Bupropion was found to aid in tobacco cessation therapy and it was approved for use by the FDA as Zyban® in 1997. It is a non-nicotine treatment modality and is sold with a recovery plan (Zyban Advantage Plan®).

The mechanism of action in the smoker is not known, though it may work through the dopaminergic and adrenergic systems [1]. Negative mood states certainly are a component of nicotine withdrawal [2] and the usefulness of this antidepressant may lie at least partially in this domain. **Bupropion has been successful as a cessation modality with 27 percent abstinence at 6 months compared to 16 percent in the placebo group as reported by Hurt et al [3].**

The dosing recommendations are to start with 150 mg a day for three days, then increase to 150mg twice a day. A quit date for tobacco products should be set for approximately one to two weeks after bupropion treatment starts and the duration of treatment can be up to 12 weeks. Bupropion appeared to not relieve all withdrawal symptoms, though the symptomatology was less with bupropion than with placebo. Weight gain was lessened by the use of the nicotine patch in combination with bupropion as compared to the patch and a placebo pill [4]. Bupropion is well tolerated. Adverse effects include dry mouth and occasional insomnia. It should be noted that bupropion is contraindicated in patients with a seizure history, anorexia, bulimia, current use of other antidepressant agents which contain bupropion, and use in patients on MAO inhibitors within the previous 14 days.

In a recent article in the British Journal of Psychiatry where a systematic review of randomized controlled trials compared bupropion with placebo in adult smokers with schizophrenia, it was found that bupropion increased the rates of smoking abstinence in smokers with schizophrenia, without jeopardizing their mental state [5].

Bupropion should be considered when treating tobacco dependence in both the substance using patient and mental health patient.

- [1] GlaxoSmithKline. Physicians' Desk Reference. 56th ed. Monvale, NJ: Medical Economics Co; 2002: 1710-1715
- [2] Jorenby, DE. New developments in approaches to smoking cessation. *Current Opinion in Pulmonary Medicine* 1998; 4:103-106
- [3] Hurt RD, Sachs DPL, Glover ED, et al. A comparison of sustained - release bupropion and placebo for smoking cessation. *N Engl J Med.*1997; 337:1195-1202
- [4] Holm KJ, Spencer CM. Bupropion: A review of its use in the management of smoking cessation. *Drugs* 2000; 59 (4): 1007-1024
- [5] Daniel Tai-yin, T et al Efficacy and safety of bupropion for smoking cessation and reduction in schizophrenia: systematic review and meta-analysis, *The British Journal of Psychiatry* 2010: 196: 346-353

Black Tar Heroin



The DEA describes heroin as a white or brownish powder or as a black sticky substance known as "black tar heroin". Heroin is manufactured from morphine, which is one of the two most commonly known drugs that can be extracted from the poppy pod fluid (the other is codeine). Heroin is synthesized by adding two acetyl groups onto the morphine (diacetyl morphine).

Black Tar Heroin is a variety of heroin produced primarily in Mexico, though South America (Colombia), Southeast Asia (principally Burma), and Southwest Asia (principally Afghanistan) can also be a source. It is one of the most prevalent forms of heroin in the western United States, while occasionally

found in western Canada and Europe.

The color and consistency of black tar heroin result from the crude processing methods used to illicitly manufacture heroin in Mexico. Black tar heroin may be sticky like roofing tar or hard like coal, and its color may vary from dark brown to black.

Black tar heroin is often sold in chunks weighing about an ounce. Its purity is generally less than South American heroin and it is most frequently smoked, or dissolved, diluted, and injected.

"Black tar heroin" is the typical street term for the drug - but it has many other street (colloquial) names, such as:

- Pigment
- Black
- *Ache: the Spanish Pronunciation of the letter H. "H" is silent in Spanish so "ache" is pronounced "atchay"*
- *Negro: the Spanish word for black*
- *Piedra: the Spanish word for stone*
- *Chiva: a Spanish word for a young goat*
- Nut Job
- Capital B
- Cheesums
- Black Clown

The assumption that Tar has less adulterants and dilutents is a misconception. The most common adulterant is lactose which is added to Tar via dissolution of both substances in a liquid medium, reheating and filtering, and then recrystallizing. This process is very simple and can be accomplished in any kitchen with no level of expertise needed.

Users who intravenously inject black tar heroin are at higher risk of venous sclerosis (a condition where the veins narrow and harden, making injection there nearly impossible) than users of powder heroin.

Researchers at UC-San Francisco have found that the rapidity with which black tar heroin destroys veins (forcing users to inject subcutaneously), along with its gummier consistency (requiring that needles be thoroughly rinsed between use), may put users at a lower risk of HIV infection (*Substance Use & Misuse* (12.31.03; 38(14)2049-2063).

Users of black tar heroin are at increased risk of life-threatening bacterial infections, in particular necrotizing soft tissue infection (commonly know as flesh eating disease). The practice of "skin-popping" or subcutaneous injection predisposes to necrotizing cellulitis (occurring below the skin) or necrotizing fasciitis

(in the deep layers of the skin), while deep intramuscular injection predisposes to necrotizing myositis (in skeletal muscles).

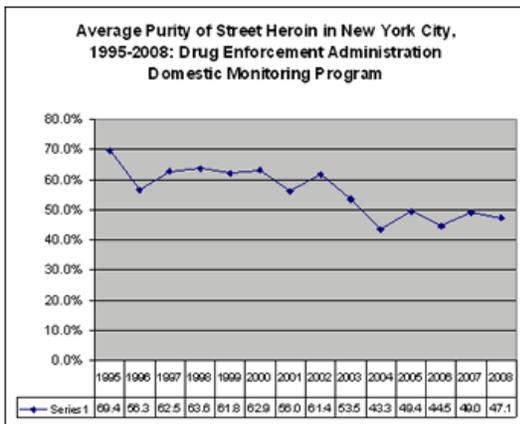
Heroin Trends in NYS

Lately there have been several press articles about an extremely pure and or potent heroin named in the press "Ultra-Heroin". Does this heroin exist in NYS? Some articles have labeled black tar heroin (Mexican) this way. According to the DEA Domestic Monitor Program, almost all the heroin samples analyzed in New York City this decade are of South American origin. Mexican heroin is very rare in New York. (For further information please read the Black Tar Heroin FYI.)

The retail price of heroin, according to National Drug Intelligence Center (NDIC) reports from 2003 to the first half of 2009, was fairly stable between 2003 and 2008 in the three cities in New York State that they analyze (New York City, Buffalo, and Albany).

- For **New York City** the price for a bag of heroin ranged from \$10-14 in 2003 to \$5-15 or \$5-12 in 2006, 2007, and 2008.
- In the first half of 2009, however, the price in New York City increased slightly to \$10-20 per bag.
- For **Buffalo** the price for a bag of heroin ranged from \$20 in 2003 to \$10-20 from 2006 to 2008.
- In the first half of 2009, the price in Buffalo increased to \$15-40 per bag.
- For **Albany** the price for a bag of heroin ranged from \$20-40 in 2003 to either \$10-40 or \$20-40 in 2006 to 2008.
- In the first half of 2009, the price in Albany increased to \$40-80 per bag.

New York State data do not support a decrease in price for heroin, but rather points to relative stability followed by a recent increase in price.



The chart displayed here, based on the Drug Enforcement Administration's Drug Monitoring Program (DMP) shows the trend in average purity street heroin in New York City between 1995 and 2008.

The DMP data would seem to suggest that while there were minor increases in heroin purity between 1996 and 1997, 2001 and 2002, 2004 and 2005, and 2006 and 2007, **over the long term, heroin purity in New York City has, in fact, declined considerably since the mid-1990s.** Unfortunately, these data are not available for other parts of the State.

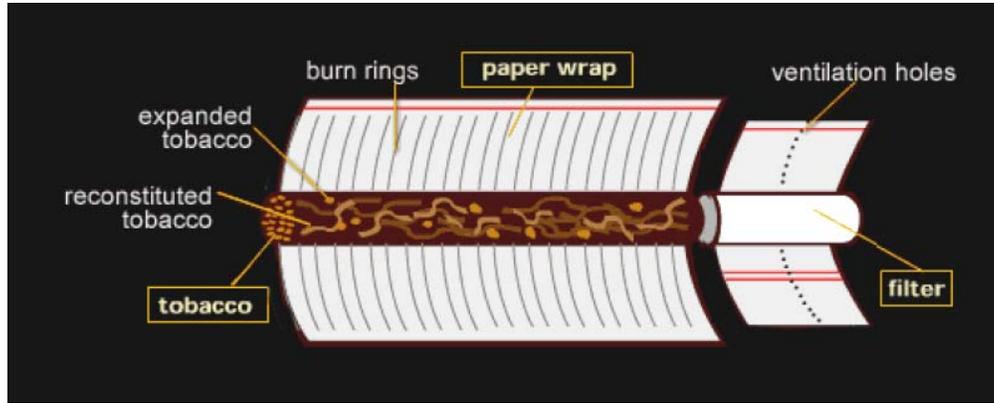
Additional Information: Report from Raymond J. Toledo, Ph.D., Manager, OASAS Street Studies Unit (SSU):

On March 5th 2010, the unit was notified by the DEA about reports of the presence of black tar heroin in the Washington Heights section of Manhattan . In response, the SSU field staff were directed to check with their regular street contacts and other known heroin users as to the availability of "black tar" heroin in New York City. To this end, multiple inquiries were made in Washington Heights, Harlem and the lower eastside of Manhattan, as well as various locations in the South Bronx, and Brooklyn .

To date, no respondent reported any indication that black tar heroin is locally available. In addition, no one mentioned any "street talk" or gossip about this form of heroin. Furthermore, some heroin users interviewed did not even recognize the term.

Anatomy of a Cigarette

A cigarette is designed as a drug delivery device that delivers nicotine to the brain in seconds. Tobacco smoke contains more than 4,000 chemicals and carbon monoxide. Some of the chemicals are carcinogens. These chemicals come from a variety of sources: produced by pyrolysis (the burning of a cigarette); from the soil and the tobacco paper and others that are added in the manufacturing process.



Tobacco in the cigarette

- One third shredded tobacco
- One third all other parts of the tobacco plant
 - Known as "reconstituted tobacco" or "homogenized sheet tobacco," it is made from a pulp of mashed tobacco stems and other parts of the tobacco leaf that would otherwise go to waste. Manufacturers spray and impregnate this reconstituted product with nicotine and as many as 600 chemical additives. The 'recon' is sliced to resemble shredded leaf tobacco.
- One third expanded or puff tobacco (Dry Ice Expanded Tobacco – DIET)
 - Tobacco is impregnated with Freon and/or ammonia, and then freeze dried, causing the dried tobacco to expand so that less can be used per cigarette. Ammonization increases nicotine volatility and increases efficiency of extraction of nicotine during smoking.

Paper Wrap

- The paper displays a pattern of concentric circle striations called "burn rings." The burn rings correspond to two different thicknesses in the paper, which serve to precisely control the speed at which the cigarette burns, slowing it automatically when the smoker is not inhaling in order to prolong the cigarette's consumption and speeding it up as the smoker takes a drag so as to maximize smoke intake.
- Like the tobacco, the cigarette paper contains a host of chemicals, among them titanium oxide, which accelerates and maintains burning so the cigarette does not go out and the smoke is delivered evenly with each puff. These chemicals have contributed too many cigarette-caused fires.

Filter

- The filter cigarette was a specialty item until 1954, when manufacturers introduced it broadly as a means to offset concerns about smoking and lung disease. Two main types are:
 - The more common dense, synthetic fiber filters
 - The "charcoal filter," which contains bits of charcoal embedded within the fiber filters - claimed to reduce certain toxins in the smoke. No evidence exists that these cigarettes are significantly less dangerous for the user.

- Most filter cigarettes have ventilation holes punched around the circumference of the filter tip. (Regular cigarettes might feature one ring of ventilation holes, while light and ultra-light cigarettes of the same brand might have two or more rings.)

These tiny holes can allow enough fresh air into the smoke that such cigarettes can test quite low in tar and nicotine levels when smoked by machines, which do not cover the holes. However, smokers' fingers or lips often cover some of these holes as they puff, giving them much higher doses of tar and nicotine than advertised. Also, studies have shown that people who smoke light cigarettes satisfy their nicotine cravings by inhaling the smoke more deeply, smoking more cigarettes and taking more puffs on each cigarette.

New Law takes effect

A US law banning the selling of so-called "light" or "mild" cigarettes took effect on June 22, 2010, but some anti-tobacco groups say the makers are sidestepping the rules by using color-coded packaging. The measure signed into law a year ago by President Barack Obama regulates tobacco for the first time and prohibits, packaging using the terms "light," "mild," or "low" - which could lead smokers into believing they are not as harmful. In a move that simply skirts the new rules, tobacco companies plan to use packaging to make those same distinctions: light colors for light cigarettes etc. The Food and Drug Administration has begun a federal review of the color-coding approach, a step that could conceivably lead to further actions against products designated as light.

K2

K2 is the brand name of an incense blend, made of herbs, spices and synthetic cannabinoids (notably JWH-018 and JWH-073), which mimic the brain's reaction to cannabis. Reportedly produced in China and Korea, it is sold as incense but has been used in similar ways as marijuana. K2 is widely available for purchase online and in smoke shops.



Legal alternatives to marijuana are being used in increasing numbers across the United States. Various formulations are being sold under names such as: K2 Incense, Spice, Blonde, Summit, Standard, and Citron. Some of these formulations include *Salvia divinorum*. *Salvia divinorum* or “*Salvia*”, also called Diviner’s sage is a perennial herb in the mint family. The herb is native to Mexico but grows in the United States as well. In Mexico, the Mazatec Indians use the herb in healing ceremonies.

Salvia contains Salvinorin A which is a hallucinogen. Salvinorin A can be absorbed from the plant by smoking, chewing, tea infusions or inhaling vapors of the burning leaves. When absorbed the effects that can be seen include hallucinations and synesthesia (where one misinterprets sensory input; smell a color for example). Long-term effects are unknown, but may be similar to LSD where one can see flashbacks and depression.

Users report highs that last between 30 minutes and two hours, and at times describe out-of-body experiences. The most common symptoms that have been reported include:

- tachycardia (increased heart rate)
- agitation/irritability
- nausea and vomiting
- confusion
- drowsiness
- headache
- hypertension
- electrolyte abnormalities
- seizures
- syncope (loss of consciousness).

Cause for concern:

- Overdose reactions, especially in adolescents.
- The mixture will not show up on a urine drug screen as THC.
- JWH-018 and CP 47,497 are synthetic substances which have not been licensed anywhere worldwide for medical applications and little is known about their effect on humans, as not even pre-clinical studies have been conducted to determine potential toxicity.

E-Cigarettes

Description and History

- "E-cigarettes" or electronic cigarettes are made of long stainless steel tubes with chambers to hold replaceable cartridges filled with various concentrations and flavors of liquid nicotine. They are powered by a rechargeable battery, and because the vapor produced is odorless and doesn't contain tar or carbon monoxide, the manufacturers say their products don't put people at risk for second-hand smoke inhalation.
- At the time of the enactment of the Tobacco-Free Regulation E-cigarettes were not an important factor to consider.

Evidence Reviewed on the Issue

- The FDA has not evaluated any e-cigarettes for safety or effectiveness.
- When the FDA conducted limited laboratory studies of certain samples, FDA found significant quality issues that indicate that quality control processes used to manufacture these products are substandard or non-existent. FDA found that cartridges labeled as containing no nicotine contained nicotine and that three different electronic cigarette cartridges with the same label emitted a markedly different amount of nicotine with each puff.
- Experts have also raised concerns that the marketing of products such as e-cigarettes can increase nicotine addiction among young people and may lead kids to try other tobacco products.
- The high cost of the apparatus and refills.
- The unknown effect of the nicotine carrier on the lung in its vaporized state.
- The effect of the E-cigarette on patients attempting to quit cigarette use by introducing cues and triggers into the treatment environment.
- Free NRT is available through the NYS DOH/NYS OASAS program.
- Limited research on the effects/success of this form of treatment

Provider Concerns on the Issue

- The need for a consistent policy across the OASAS provider system.

Policy Statement

- It is the NYS OASAS policy as of this date that E-cigarettes should be prohibited in a similar fashion as tobacco containing products in the NYS OASAS provider system.

Chantix (Varenicline) and the Patient with Tobacco and Alcohol Dependence

Varenicline is approved for the use of tobacco dependence. Over the last several years there have been a number of articles on the use of varenicline in the alcohol dependent patient.

It has been recognized that alcohol dependence and tobacco dependence are frequently co-morbid conditions. It has been reported in rodent studies that varenicline, a partial nicotinic agonist with high affinity for the $\alpha 4\beta 2$ nAChR receptor reduced alcohol intake. Ericson and his group (J Pharmacol Exp Ther. 2009; 329(1):225-30) showed that varenicline exhibits properties with respect to its interaction with ethanol and nicotine in the brain reward system that may be beneficial for treating patients with alcohol dependence with (and possibly also without) concomitant nicotine dependence.

Mckee et al (Biol Psychiatry. 2009; 66(2):185-90) looked at varenicline in heavy drinking smokers. They developed a double-blind, placebo-controlled trial using varenicline at a 2mg per day dose vs. placebo in 20 subjects who were daily smokers but non-alcohol dependent heavy drinkers. Following 7 days of medication pretreatment, participants were first administered a priming dose of alcohol (.3 g/kg) and subjective, and physiologic responses were assessed. A 2-hour alcohol self-administration period followed during which participants could choose to consume up to 8 additional drinks (each .15 g/kg).

The results showed that varenicline significantly reduced the number of drinks consumed compared to placebo and increased the likelihood of abstaining from any drinking during the self-administration period. Following the priming drink, varenicline attenuated alcohol craving and reduced subjective reinforcing alcohol effects (high, like, rush, feel good, intoxicated). Adverse events associated with varenicline were minimal and, when combined with alcohol, produced no significant effects on physiologic reactivity, mood, or nausea.

It should be noted that there can be adverse effects to varenicline in addition to the much reported increase in depression/suicidality. Pirmoradi et al reported (Am J Health Syst Pharm. 2008; 65(17):1624-6) that there was the development of severe anxiety, nausea, vertigo, blurred vision, and dizziness after initiation of varenicline in a patient with a history of alcohol dependence (stopping alcohol use 2 years earlier) and major depression.

More research is needed, though treating two conditions with one medication may be the optimal way to proceed as far as limiting side effects, medication interactions and increasing patient compliance.