

Is smoking a risk factor for decreased semen quality? A cross-sectional analysis.

Ramlau-Hansen CH; Thulstrup AM; Aggerholm AS; Jensen MS; Toft G; Bonde JP. *Human Reproduction* 22(1): 188-196, 2007. (58 refs.)

BACKGROUND: Previous studies suggest a deleterious effect of cigarette smoking on semen quality, but their results have not been consistent. We studied the association between current smoking and semen characteristics and hormonal levels in a large group of healthy men. **METHODS:** From 1987 to 2004, seven separate occupational or environmental semen quality studies were co-ordinated by our department. A total of 2562 men participated, each providing semen and blood sample and answering a questionnaire about lifestyle and factors related to health. Appropriate semen and smoking data were available for 2542 men. **RESULTS:** Adjusting for study, age and other covariates, we observed an inverse dose-response relation between smoking and semen volume, total sperm count and percentage motile sperm. Heavy smokers had a 19% lower sperm concentration than non-smokers. We found a positive dose-response relationship between smoking and testosterone, LH and the LH/free testosterone ratios. **CONCLUSION:** Current smoking in adult life moderately impairs the semen quality. It is well known that semen quality is associated to fecundity. Therefore, it would be sensible to advise men to abstain from smoking to avoid decreased fecundity. Copyright 2007, Oxford University Press.

A brief exposure to moderate passive smoke increases metabolism and thyroid hormone secretion.

Metsios GS; Flouris AD; Jamurtas AZ; Carrillo AE; Kouretas D; Germenis AE et al. *Journal of Clinical Endocrinology and Metabolism* 92(1): 208-211, 2007. (20 refs.)

Context: Active smoking influences normal metabolic status and thyroid function. **Objective:** The objective was to assess experimentally the effects of 1 h of moderate passive smoking in a controlled simulated bar/restaurant environment on the metabolism and thyroid hormone levels in healthy nonsmokers. **Participants:** Eighteen (nine females, nine males) healthy individuals (mean +/- SD: age, 25.3 +/- 3.1 yr;

height, 174.0 +/- 10.1 cm; weight, 65.2 +/- 13.7 kg) participated in the study. **Design:** In repeated-measures randomized blocks, participants visited the laboratory on 2 consecutive days. In the experimental condition, they were exposed to 1 h of moderate passive smoking at a carbon monoxide concentration of 23 +/- 1 ppm in an environmental chamber, whereas in the control condition participants remained in the same chamber for 1 h breathing normal atmospheric air. **Main Outcome Measures:** In both conditions, cotinine serum and urine levels, resting energy expenditure (REE), as well as concentration of T-3, free T-4, and TSH were assessed before participants entered the chamber and immediately after their exit. Heart rate and blood pressure were tested in 10-min intervals during all REE assessments. **Results:** The mean +/- SD difference of serum and urine cotinine levels (- 0.27 +/- 3.94 vs. 14.01 +/- 6.54 and 0.05 +/- 2.07 vs. 7.23 +/- 3.75, respectively), REE (6.73 +/- 98.06 vs. 80.58 +/- 120.91) as well as T3 and free T-4 (0.05 +/- 0.11 vs. 0.13 +/- 0.12 and 0.02 +/- 0.15 vs. 0.22 +/- 0.20) were increased in the experimental compared with the control condition at baseline and follow-up (P < 0.05). No statistically significant variation was observed in the mean difference of the remaining parameters (P > 0.05). Serum and urine cotinine values were linearly associated with REE (P < 0.05). **Conclusion:** One hour of passive smoking at bar/restaurant levels is accompanied by significant increases in metabolism and thyroid hormone levels. Copyright 2007, Endocrine Society.

Carboxyhemoglobin and thiocyanate as biomarkers of exposure to carbon monoxide and hydrogen cyanide in tobacco smoke. (review).

Scherer G. *Experimental and Toxicologic Pathology* 58(2/3): 101-124, 2006. (204 refs.)

The determination of biomarkers in human body fluids is a useful tool, which allows the quantitative assessment of the exposure to chemicals or complex mixtures of chemicals and of early biological effects as a result of the exposure. Biomarkers require validation before their successful application in human studies. This review describes some general purposes of human biomonitoring and biomarkers including the requirements for validation. Risk assessment and harm reduction of smoking and tobacco products,

respectively, is a very suitable field for the application of biomarkers. A brief historical review shows that the application of biomarkers of exposure and effect in human smoking goes back more than 150 years. Two 'classical' biomarkers of exposure to tobacco, namely carboxyhemoglobin (COHb and its equivalent carbon monoxide in exhalate, COex) and thiocyanate (SCN) in body fluids are discussed in terms of sources of exposure, metabolism, disposition kinetics and influencing host factors. Data on COHb/COex and SCN in nonsmokers and smokers as well as the power to discriminate between smokers and nonsmokers are presented. Both biomarkers are significantly correlated with the daily cigarette consumption. Smoking machine-derived yields of the precursors carbon monoxide and hydrogen cyanide were not correlated with COHb/COex and SCN, respectively. It is concluded that, while COHb/COex is a useful biomarker for assessing the smoke inhalation, preferably in controlled studies, the application of SCN in body fluids as a biomarker for smoking is limited, mainly due to the abundance of other sources for SCN. Copyright 2006, Elsevier Science.

Early postnatal exposure to cigarette smoke impairs the antigen-specific T-cell responses in the spleen.

Singh SP; Razani-Boroujerdi S; Pena-Philippides JC; Langley RJ; Mishra NC; Sopori ML. *Toxicology Letters* 167(3): 231-237, 2006. (40 refs.)

Annually, approximately two million babies are exposed to cigarette smoke in utero and postnatally through cigarette smoking of their mothers. Exposure to mainstream cigarette smoke is known to impair both innate and adaptive immunities, and it has been hypothesized that the effects of in utero exposure to cigarette smoke on children's health might primarily stem from the adverse effects of cigarette smoke on the immune system. To simulate the environment that babies from smoking mothers encounter, we examined the effects of prenatal mainstream and postnatal sidestream cigarette smoke on spleen cell responses. Results show that postnatal exposure of newborn Balb/c mouse pups to sidestream cigarette smoke through the first 6 weeks of life strongly suppresses the antibody response of spleen cells to the T-cell-dependent antigen, sheep red blood cells. The reduction in the antibody response seen within 6 weeks of postnatal smoke exposure is much quicker than the published data on the time (25 weeks) required to establish reproducible immunosuppression in adult rats and mice. Moreover, the immunosuppression is not associated with significant changes in T-cell numbers or subset distribution. While the postnatal

exposure to cigarette smoke did not affect the mitogenic response of T and B cells, the exposure inhibited the T cell receptor-mediated rise in the intracellular calcium concentration. These results suggest that the early postnatal period is highly sensitive to the immunosuppressive effects of environmental tobacco smoke, and the effects are causally associated with impaired antigen-mediated signaling in T cells. Copyright 2006, Elsevier Science.

Nicotine: The link between cigarette smoking and the progression of renal injury?

Jaimes EA; Tian RX; Raji L. *American Journal of Physiology: Heart and Circulatory Physiology* 292(1): h76-h82, 2007. (52 refs.)

Cigarette smoke (CS) is the most important source of preventable morbidity and mortality in the United States. Recent clinical studies have suggested that, in addition to being a major cardiovascular risk factor, CS promotes the progression of kidney disease. The mechanisms by which CS promotes the progression of chronic kidney disease have not been elucidated. Here we demonstrate for the first time that human mesangial cells (MCs) are endowed with the nicotinic ACh receptors (nAChRs) alpha 4, alpha 5, alpha 7, beta 2, beta 3, and beta 4. Studies performed in other cell types have shown that these nAChRs are ionotropic receptors that function as agonist-regulated Ca²⁺ channels. Nicotine induced MC proliferation in a dose-dependent manner. At 10⁻⁷ M, a concentration found in the plasma of active smokers, nicotine induced MC proliferation [control, 1,328 +/- 50 vs. nicotine, 2,761 +/- 90 counts/minute (cpm); P < 0.05] and increased the synthesis of fibronectin (50%), a critical matrix component involved in the progression of chronic kidney disease. We and others have shown that, in response to PKC activation, MC synthesize reactive oxygen species (ROS) via NADPH oxidase. In the current studies we demonstrate that PKC inhibition as well as diphenylethylideneiodonium and apocynin, two inhibitors of NADPH oxidase, prevented the effects of nicotine on MC proliferation and fibronectin production, hence establishing ROS as second messengers of the actions of nicotine. Furthermore, nicotine increased the production of ROS as assessed by 2', 7'-dichlorofluorescein diacetate fluorescence [control, 184.4 +/- 26 vs. nicotine, 281.5 +/- 26 arbitrary fluorescence units (AFU); n = 5 experiments, P < 0.05]. These studies unveil previously unrecognized mechanisms that indict nicotine, a component of CS, as an agent that may accelerate and promote the progression of kidney disease. Copyright 2007, American Physiological Society.

Natural history of nicotine withdrawal.

Shiffman S; Patten C; Gwaltney C; Paty J; Gnys M; Kassel J et al. *Addiction* 101(12): 1822-1832, 2006.

(56 refs.)

Aim: To examine the natural history of nicotine withdrawal and individual differences associated with withdrawal duration and severity. **Design and setting:** Prospective study of withdrawal symptoms among smokers who quit for at least 24 hours. Participants used Ecological Momentary Assessment to monitor symptoms in their natural environment using an Electronic Diary (ED). Participants A total of 214 cigarette smokers (59% female, 92% Caucasian). **Intervention** All participants received a clinic-based, behavioral, group cessation intervention. Severity and duration of withdrawal was not addressed explicitly in treatment. **Measurements** Participants were 'beeped' by the ED approximately five times/day to complete affect assessments (negative affect, arousal, attention disturbance, restlessness), and daily assessments of sleep disturbance (at waking) and of cognitive performance (each evening) for a week prior to quitting and for up to 21 days after quitting. **Withdrawal** was considered resolved when withdrawal scores returned to baseline levels for at least 2 consecutive days. **Findings:** All symptoms returned to baseline levels within 10 days of quitting. All variables except arousal and sleep disturbance showed change over time. No robust predictors of individual differences in withdrawal responses emerged. **Conclusions** The time-course of withdrawal may be shorter than previously reported. The natural history of nicotine withdrawal may have implications for theories of withdrawal and smoking relapse and for smoking cessation treatment. Copyright 2006, Society for the Study of Addiction to Alcohol and Other Drugs.

Orofacial cleft risk is increased with maternal smoking and specific detoxification-gene variants.

Shi M; Christensen K; Weinberg C; Romitti P; Bathum L; Lozada A et al. *American Journal of Human Genetics* 80(1): 76-90, 2007. (45 refs.)

Maternal smoking is a recognized risk factor for orofacial clefts. Maternal or fetal pharmacogenetic variants are plausible modulators of this risk. In this work, we studied 5,427 DNA samples, including 1,244 from subjects in Denmark and Iowa with facial clefting and 4,183 from parents, siblings, or unrelated population controls. We examined 25 single-nucleotide polymorphisms in 16 genes in pathways for detoxification of components of cigarette smoke, to look for evidence of gene-environment interactions. For genes identified as related to oral clefting, we studied gene-expression profiles in fetal development

in the relevant tissues and time intervals. Maternal smoking was a significant risk factor for clefting and showed dosage effects, in both the Danish and Iowan data. Suggestive effects of variants in the fetal NAT2 and CYP1A1 genes were observed in both the Iowan and the Danish participants. In an expanded case set, NAT2 continued to show significant overtransmission of an allele to the fetus, with a final P value of .00003. There was an interaction between maternal smoking and fetal inheritance of a GSTT1-null deletion, seen in both the Danish (P = .002) and Iowan (P = .002) studies, with a Fisher's combined P value of <.001, which remained significant after correction .03 for multiple comparisons. Gene-expression analysis demonstrated expression of GSTT1 in human embryonic craniofacial tissues during the relevant developmental interval. This study benefited from two large samples, involving independent populations, that provided substantial power and a framework for future studies that could identify a susceptible population for preventive health care. Copyright 2007, University of Chicago Press.

Exposure to marijuana during pregnancy alters neurobehavior in the early neonatal period.

Barros MCD; Guinsburg R; Peres CD; Mitsuhiro S; Chalem E; Laranjeira RR. *Journal of Pediatrics* 149(6): 781-787, 2006. (37 refs.)

Objective: To assess the neurobehavior of full-term neonates of adolescent mothers exposed to marijuana during pregnancy. **Study design:** This prospective cross-sectional study included full-term infants within 24 to 72 hours of life born to adolescent mothers at a single center in Brazil. Data on sociodemographic and obstetrical and neonatal characteristics were collected. The mothers underwent the Composite International Diagnostic Interview, and the infants were assessed with the Neonatal Intensive Care Unit Network Neurobehavioral Scale (NNS). Maternal hair and neonatal meconium were analyzed. Neonates exposed in utero to tobacco, alcohol, cocaine, and/or any other drugs except marijuana were excluded. **Results:** Of 3685 infants born in the study hospital, 928 (25%) were born to adolescent mothers. Of these, 561 infants met the inclusion criteria and were studied. Marijuana exposure was detected in 26 infants (4.6%). Infants exposed (E) or not exposed (NE) to marijuana differed in the following NNS variables: arousal (E, 4.05 +/- 1.19 vs NE, 3.68 +/- 0.70), regulation (E, 5.75 +/- 0.62 vs NE, 6.04 +/- 0.72), and excitability (E, 3.27 +/- 1.40 vs NE, 2.40 +/- 1.57). After controlling for confounding variables, the effect of marijuana exposure on these scores remained significant. **Conclusions:** Marijuana exposure during pregnancy

alters the neurobehavioral performance of term newborn infants of adolescent mothers. Copyright 2006, Elsevier Science.

Alcohol dosing and total mortality in men and women: An updated meta-analysis of 34 prospective studies. (review).

Di Castelnuovo A; Costanzo S; Bagnardi V; Donati MB; Iacoviello L; de Gaetano G. *Archives of Internal Medicine* 166(22): 2437-2445, 2006. (58 refs.)

Background: Moderate consumption of alcohol is inversely related with coronary disease, but its association with mortality is controversial. We performed a meta-analysis of prospective studies on alcohol dosing and total mortality. Methods: We searched PubMed for articles available until December 2005, supplemented by references from the selected articles. Thirty-four studies on men and women, for a total of 1,015 835 subjects and 94,533 deaths, were selected. Data were pooled with a weighed regression analysis of fractional polynomials. Results: AJ-shaped relationship between alcohol and total mortality was confirmed in adjusted studies, in both men and women. Consumption of alcohol, up to 4 drinks per day in men and 2 drinks per day in women, was inversely associated with total mortality, maximum protection being 18% in women (99% confidence interval, 13%-22%) and 17% in men (99% confidence interval, 15%-19%). Higher doses of alcohol were associated with increased mortality. The inverse association in women disappeared at doses lower than in men. When adjusted and unadjusted data were compared, the maximum protection was only reduced from 19% to 16%. The degree of association in men was lower in the United States than in Europe. Conclusions: Low levels of alcohol intake (1-2 drinks per day for women and 2-4 drinks per day for men) are inversely associated with total mortality in both men and women. Our findings, while confirming the hazards of excess drinking, indicate potential windows of alcohol intake that may confer a net beneficial effect of moderate drinking, at least in terms of survival. Copyright 2006, American Medical Association.

Coronary atherosclerosis and alcohol consumption: Angiographic and mortality data.

Femia R; Natali A; L'Abbate A; Ferrannini E. *Arteriosclerosis, Thrombosis, and Vascular Biology* 26(7): 1607-1612, 2006. (34 refs.)

Objective - Moderate alcohol consumption is associated with reduced cardiovascular disease (CVD) risk. Whether this protection is based on a lesser degree of coronary atherosclerosis has not been established. Methods and Results - We studied 1676

men and 465 women consecutively undergoing coronary angiography. A score (ATS) was calculated by summing the percent lumen narrowing of all main vessels; alcohol consumption was quantitated by questionnaire. In univariate analysis, ATS was significantly ($P \leq 0.001$) associated with male sex, age, familial CVD, smoking, diabetes, hypertension, and serum cholesterol levels; alcohol consumption was associated with less frequent diabetes ($P < 0.001$) and lower ATS ($P=0.02$). By multivariate analysis, alcohol intake was associated with lower ATS ($P < 0.01$) independently of the other risk factors; the estimated effect size was comparable to that associated with a 1-mmol decrement in serum cholesterol. Over a median follow-up of 93 months, 37 women and 194 men died from a cardiac cause. By Cox analysis, positive predictors for cardiac mortality were male sex (hazard ratio [HR], 1.7; 95% confidence interval [CI], 1.1 to 2.6), age (HR, 2.1; 95% CI, 1.8 to 2.5 per decade) and diabetes (HR, 1.7; 95% CI, 1.2 to 2.4), whereas alcohol consumption was the only negative predictor (HR, 0.84; 95% CI, 0.71 to 1.00). Conclusions - In a selected high-risk population, moderate alcohol consumption was independently associated with less coronary atherosclerosis and lower risk for cardiac mortality. Copyright 2006, Lippincott, Williams & Wilkins.

Comparative cardiac pathology among deaths due to cocaine toxicity, opioid toxicity and non-drug-related causes.

Darke S; Kaye S; Duflo J. *Addiction* 101(12): 1771-1777, 2006. (36 refs.)

Aim: To compare cardiac and cerebrovascular pathology among deaths due to cocaine toxicity, deaths due to opioid toxicity and deaths from hanging that were toxicologically negative for cocaine/opioids. Design: Case-control. Findings: The cocaine group had significantly higher proportions of left ventricular hypertrophy and ischaemic heart disease than either comparison group. Coronary artery atherosclerosis was also detected in significantly higher proportions of cocaine cases than in either comparison group. The cocaine group was more likely than either comparison group to have atherosclerosis of the left anterior descending coronary artery, right coronary artery and circumflex artery. Only in the left anterior descending coronary artery did the cocaine group exhibit higher levels of moderate-severe atherosclerosis. Cocaine cases also had significantly higher levels of cerebrovascular atherosclerosis than either comparison group. Conclusions: Levels of cardiac and cerebrovascular pathology were higher among cocaine users than either comparison group. The high levels of cardiac and cerebrovascular pathology seen among

cocaine cases do not appear to be artefacts of a drug-dependent life-style, but relate specifically to cocaine. Copyright 2006, Society for the Study of Addiction to Alcohol and Other Drugs.

Characteristics of fathers who have children with fetal alcohol syndrome or incomplete fetal alcohol syndrome.

Kvigne VL; Leondardson GR; Welty TK. *South Dakota Medicine* 59(8): 337-340, 2006

OBJECTIVES: Determine alcohol use, referrals to treatment, receiving treatment, and medical problems related to alcohol among fathers who have children with FAS or incomplete FAS. METHODS: Fathers who had American Indian children with FAS (Study 1) or incomplete FAS (Study 2) were compared with fathers whose children did not have FAS. RESULTS: About half of case and control fathers had alcohol use and alcohol-related medical problems documented in their medical records. Case fathers were more likely to receive alcohol treatment and have injuries related to alcohol abuse. CONCLUSION: Significantly more fathers of children with FAS were referred for alcohol treatment, received alcohol treatment, experienced injuries, and had delirium tremens than control fathers. Fathers of children with incomplete FAS were significantly more likely to drink alcohol, to have received alcohol treatment, and to have alcohol-related medical problems and injuries than control fathers. Copyright 2006, South Dakota State Medical Association.

Early intervention for children prenatally exposed to cocaine.

Bono KE; Sheinberg N; Scott KG; Claussen AH. *Infants and Young Children* 20(1): 26-37, 2007. (59 refs.)

This article brings together information from our experience of providing research-based intervention to more than 600 children who were prenatally exposed to cocaine and from the research literature on the effects of prenatal cocaine exposure. Based on our experience and this literature, it is now clear that there are no large negative independent effects of cocaine exposure and that there is no "crack baby" syndrome. However, many of these children who have been exposed to cocaine are at risk for poor developmental outcomes, particularly in the area of language development, primarily because of the environmental factors that are associated with parental substance abuse. As our intervention research shows, early intervention can be effective in ameliorating some of this risk. Suggestions are made regarding programmatic steps that should be taken next to further improve program effectiveness and enhance

the knowledge base. Copyright 2007, Lippincott, Williams & Wilkins.

Infant stress reactivity and prenatal alcohol exposure.

Haley DW; Handmaker NS; Lowe J. *Alcoholism: Clinical and Experimental Research* 30(12): 2055-2064, 2006. (81 refs.)

Background: Animal studies have shown that prenatal alcohol exposure (PAE) is linked to alterations in the stress response systems. To date, little is known about the impact of PAE on stress systems in human infants. The current study examined PAE effects on the stress response, as evidenced by the activation of the limbic-hypothalamic-pituitary-adrenal (L-HPA) axis and autonomic system and changes in negative affect during a social-emotional challenge in human infants. We also examined whether the effects of PAE on infant responsiveness differed in boys and girls. Methods: Measures of cortisol, heart rate, and negative affect were obtained during a modified version of Tronick's still-face procedure, a standardized developmental paradigm used to study emotion and stress regulation. Our sample included fifty-five 5- to 7-month-old infants whose mothers were enrolled in an alcohol intervention study. Measures of maternal alcohol consumption during pregnancy and after delivery were obtained using Timeline Followback interviewing methods. Relationships between prenatal alcohol consumption and infant outcomes were examined. In addition, mothers were divided into high and low-frequency drinkers, based on the mean percent of prenatal drinking days (PDD) to facilitate between-group comparisons of infant stress measures. Results: Mothers enrolled in our study reported significant reductions in alcohol consumption after learning of their pregnancies. Nevertheless, PDD from conception to pregnancy recognition was related to increases in cortisol reactivity, elevated heart rate, and negative affect in their infants. The effects of PAE on infant responsiveness were significant after controlling for the effects of maternal depression and annual income. In addition, the effects of PAE on cortisol reactivity differed for boys and girls. Conclusions: Greater PAE was related to greater activation of stress response systems. Our findings suggest that PAE affects the development of infant stress systems and that these effects differ in boys and girls. This work supports the possibility that PAE is related to alterations in infant stress systems, which could underlie problems in cognitive and social-emotional functioning that are common among persons exposed prenatally to alcohol. Copyright 2006, Research Society on Alcoholism.

Is caffeine addictive? A review of the literature.

Satel S. *American Journal of Drug and Alcohol Abuse* 32(4): 493- 502, 2006. (33 refs.)

The common-sense use of the term addiction is that regular consumption is irresistible and that it creates problems. Caffeine use does not fit this profile. Its intake does no harm to the individual or to society and its users are not compelled to consume it. Though cessation of regular use may result in symptoms such as headache and lethargy, these are easily and reliably reversed by ingestion of caffeine. Some have argued that continued caffeine use is an attempt to suppress low grade withdrawal symptoms such as sleepiness and lethargy. In some moderate users, this is possible; however, in experimental contexts, the phenomenon is too inconsistent to constitute a reliably valid syndrome. Copyright 2006, Marcel Dekker, Inc.

Oral health of the methamphetamine abuser.

Donaldson M; Goodchild JH. *American Journal of Health-System Pharmacy* 63(21): 2078-2082, 2006. (39 refs.)

Purpose. The pharmacology of methamphetamine is reviewed, and the effects of methamphetamine use on oral health are described. Summary. Methamphetamine is a highly addictive amphetamine analogue, initially synthesized in 1919. Illicit methamphetamine use leads to devastating effects on health, particularly the dentition. Illegal production of methamphetamine has skyrocketed in recent years, as have the number of users. The chief complaint of methamphetamine users is xerostomia. Without the protective effects of saliva, caries development in these patients is rampant. The typical pattern of decay involves the facial and cervical areas of both the maxillary and mandibular teeth, with eventual progression to frank coronal involvement. The acidic substances used to manufacture this drug have also been implicated as a cause of tooth decay and wear in users, as has bruxism as a result of drug-induced

hyperactivity. When possible, these patients should be referred to a dentist to improve their oral health status and minimize the potential for adverse cardiovascular sequelae. Other preventive measures for methamphetamine users include stimulating saliva flow and increasing fluoride supplementation. Pharmacists should also counsel users to avoid carbohydrate-rich soft drinks in favor of water. Oral moisturizers may also be effective. Conclusion. Methamphetamine use causes xerostomia secondary to sympathetic central nervous system activation, rampant caries caused by high-sugar intake in the absence of protective saliva, and bruxism as a result of hyperactivity. Practitioners should know how to recognize the signs of and manage the oral health of patients with a history of methamphetamine use. Copyright 2006, American Society of Health-System Pharmacists.

Risk factors for delirium tremens: A retrospective chart review. (review).

Wright T; Myrick H; Henderson S; Peters H; Malcolm R. *American Journal on Addictions* 15(3): 213-219, 2006. (36 refs.)

review was performed within an inpatient VA hospital setting in an attempt to identify risk factors for delirium tremens (DTs). Cases of delirium tremens were compared to cases where patients' alcohol withdrawal during hospitalization did not progress to DTs. Significant differences were found in regard to prior histories of DTs and laboratory values at admission. The amount and duration of benzodiazepine use during hospitalization, antipsychotic use during hospitalization, and length of hospitalization were also statistically different between the groups. While not reaching statistical significance, there were differences in reason for admission and relapse rate upon follow-up between the groups. Copyright 2006, American Academy of Psychiatrists in Alcoholism and Addictions.