Record 16 of 81 in CC Search(R) 5 Sci. Ed. Week 30 (2001/07/24-2001/07/30)

AN: 0001695900-0009See Contents-Page
PT: Journal
TI: Cigarette smoking and aneuploidy in human sperm
AU: Shi-QH; Ko-E; Barclay-L; Hoang-T; Rademaker-A; Martin-R
SO: MOLECULAR-REPRODUCTION-AND-DEVELOPMENT. AUG 2001; 59 (4) : 417-421
PY: 2001
IS: 1040-452X
AB: Cigarette smoke contains chemicals which are capable of inducing aneuploidy in experimental systems. These chemicals have been shown to reach the male reproductive system, increasing oxidative DNA damage in human sperm and lowering semen quality. We have examined the association between smoking and aneuploid sperm by studying 31 Chinese men with similar demographic characteristics and lifestyle factors except for cigarette smoking. None of the men drank alcohol. These men were divided into three groups: nonsmokers (10 men), light smokers (< 20 cigarettes/day, 11 men), and heavy smokers (greater than or equal to 20 cigarettes/day, 10 men). There were no significant differences in semen parameters or in age across groups. Two multi-color fluorescence in situ hybridizations (FISH) were performed: two-color FISH for chromosomes 13 and 21, and three-color FISH for the sex chromosomes using chromosome 1 as an internal autosomal control for diploidy and lack of hybridization. The mean hybridization efficiency was 99.78%. The frequency of disomy 13 was significantly higher in light and heavy smokers than in nonsmokers, while no significant differences in the frequency of disomy 21, X or Y were observed across groups. Significant inter-donor heterogeneity in every category of disomic sperm examined was found in both light and heavy smokers, while in nonsmokers only XY disomy showed significant inter-donor differences. Thus, we conclude that cigarette smoking may increase the risk of aneuploidy only for certain chromosomes and that men may have different susceptibilities to aneuploidy in germ cells induced by cigarette smoking.
UD: 200131

Record 45 of 81 in CC Search(R) 5 Sci. Ed. Week 30 (2001/07/24-2001/07/30)

AN: 0001696169-0008See Contents-Page
PT: Journal
TI: Air pollution and blood markers of cardiovascular risk
AU: Schwartz-J
SO: ENVIRONMENTAL-HEALTH-PERSPECTIVES. JUN 2001; 109 Suppl. 3 : 405-409
PY: 2001
IS: 0091-6765
AB: Recent studies have linked air pollution to tens of thousands of premature cardiovascular deaths per year. The mechanisms of such associations remain unclear. In this study we examine the association between blood markers of cardiovascular risk and air pollution in a national sample of the U.S. population. Air pollution concentrations were merged to subjects in the Third National Health and Nutrition Examination Survey (NHANES III) in the United States, and the association with fibrinogen levels and counts of platelets and white blood cells were examined. The subjects in NHANES III are a representative sample of the U.S. population. Regressions controlled for age, race, sex, body mass index, current smoking, and number of cigarettes per day. The complex survey design was dealt with using mixed models with a random sampling site effect. In single-pollutant models,

PM10 (particulate matter with a mass median aerodynamic diameter less than 10 mum) was associated with all three outcomes ($p < 0.05$). Sulfur dioxide (SO_2) was significantly associated only with white cell counts, nitrogen dioxide (NO_2) with platelet counts and fibrinogen, and ozone with none of the outcomes. In two-pollutant models, PM10 remained a significant predictor of white cell counts controlling for SO_2 but not vice versa. PM10 was marginally significant in a model for platelet counts with NO_2, and the sign of the NO_2 coefficient was reversed. These results were stable with control for indoor exposures (wood stoves, environmental tobacco smoke, gas stoves, fireplaces), dietary risk factors (saturated fat, alcohol, caffeine intake, n-3 fatty acids), and serum cholesterol. The magnitude of the effects are modest [e.g., 13 <mu>g/dL fibrinogen for an interquartile range (IQR) change in PM10, 95% confidence interval (CI) 4.6-22.1 mg/dL]. However, the odds ratio of being in the top 10% of fibrinogen for the same IQR change was 1.77 (95% CI 1.26-2.49). These effects provide considerable biologic plausibility to the mortality studies. PM10, but not gaseous air pollutants, is associated with blood markers of cardiovascular risk, and this may explain epidemiologic associations with early deaths.
UD: 200131

Record 67 of 81 in CC Search(R) 5 Sci. Ed. Week 30 (2001/07/24-2001/07/30)

AN: 0001696376-0016See Contents-Page
PT: Journal
TI: Human SLPI inactivation after cigarette smoke exposure in a new in vivo model of pulmonary oxidative stress
AU: Cavarra-E; Lucattelli-M; Gambelli-F; Bartalesi-B; Fineschi-S; Szarka-A; Giannerini-F; Martorana-PA; Lungarella-G
SO: AMERICAN-JOURNAL-OF-PHYSIOLOGY-LUNG-CELLULAR-AND-MOLECULAR-PHYSIOLOGY. AUG 2001; 281 (2) : L412-L417
PY: 2001
IS: 1040-0605
AB: The role of oxidative stress in inactivating antiproteases is the object of debate. To address this question, we developed an in vivo model of pulmonary oxidative stress induced by cigarette smoke (CS) in mice. The major mouse trypsin inhibitor contrapsin is not sensitive to oxidation, and the mouse secretory leukoprotease inhibitor (SLPI) does not inhibit trypsin. Instead, human recombinant (hr) SLPI inhibits trypsin and is sensitive to oxidation. Thus we determined the effect of CS in vivo on hrSLPI antiproteolytic function in the airways of mice. CS caused a significant decrease in total antioxidant capacity in bronchoalveolar lavage fluid (BALF) and significant changes in oxidized glutathione, ascorbic acid, protein thiols, and 8-epi-PGF2(alpha). Intratracheal hrSLPI significantly increased BALF antitryptic activity. CS induced a 50% drop in the inhibitory activity of hrSLPI. Pretreatment with N-acetylcysteine prevented the CS-induced loss of hrSLPI activity, the decrease in antioxidant defenses, and the elevation of 8-epi-PGF-2 alpha. Thus an inactivation of hrSLPI was demonstrated in this model. This is a novel model for studying in vivo the effects of CS oxidative stress on human protease inhibitors with antitrypsin activity.
UD: 200131

Record 68 of 81 in CC Search(R) 5 Sci. Ed. Week 30 (2001/07/24-2001/07/30)

AN: 0001696376-0027See Contents-Page
PT: Journal
TI: Cytotoxic effects of cigarette smoke extract on an alveolar type II cell -derived cell line
AU: Hoshino-Y; Mio-T; Nagai-S; Miki-H; Ito-I; Izumi-T
SO: AMERICAN-JOURNAL-OF-PHYSIOLOGY-LUNG-CELLULAR-AND-MOLECULAR-PHYSIOLOGY. AUG 2001; 281 (2) : L509-L516
PY: 2001
IS: 1040-0605
AB: Injury of the alveolar epithelium by cigarette smoke is presumed to be an important process in the pathogenesis of smoking-related pulmonary diseases. We investigated the cytotoxic effects of cigarette smoke extract (CSE) on an alveolar type II cell-derived cell line (A549). CSE caused apoptosis at concentrations of 5% or less and necrosis at 10% or more. When CSE was exposed to air before application to A549 cells, the cytotoxic effects were attenuated. CSE caused cell death without direct contact with the cells. Acrolein and hydrogen peroxide, two major volatile factors in cigarette smoke, caused cell death in a similar manner. Aldehyde dehydrogenase, a scavenger of aldehydes, and N-acetylcysteine, a scavenger of oxidants and aldehydes, completely inhibited CSE-induced apoptosis. CSE and acrolein increased intracellular oxidant activity. In conclusion, apoptosis of alveolar epithelial cells may be one of the mechanisms of lung injury induced by cigarette smoking. This cytotoxic effect might be due to an interaction between aldehydes and oxidants present in CSE or formed in CSE-exposed cells.
UD: 200131