Ontologies Classes Object Properties Data Properties Annotation Properties Individuals Datatypes Clouds

Class: Second-hand_Smoke_LC

Annotations (3)

- rdfs:comment ""Environmental factors play a determining role in human cancer 34. Many cancer-causing agents (carcinogens) are present in the air we breathe, in the food we eat, and in the water we drink 34,45. Humans' constant and to some extent unavoidable exposure to environmental carcinogens makes investigation of cancer etiology extremely complicated. The complexity of human cancer etiology is particularly challenging for those types of cancer with long latency, which are associated with exposure to ubiquitous environmental carcinogens 34. The small and somehow disputable risk of lung cancer development in relation to SHS exposure exemplifies the intricacy of establishing human cancer etiology when omnipresent carcinogens are concerned. Because of temporal variabilities in source, composition and concentration of SHS, conventional exposure assessment using indices of SHS, as measured in the ambient air or in the body fluids of exposed individuals, at certain times has failed to estimate long-term SHS exposure 10. Consequently, although the causal link between SHS exposure and lung cancer development is well-established 1–3, the estimated risk for developing lung cancer consequent to SHS exposure remains somewhat debatable. Establishing the mechanism(s) of action of SHS of relevance for carcinogenesis can help identify unique biological markers that can be used for assessing lung cancer risk in relation to SHS exposure. Because SHS contains basically the same carcinogens as mainstream smoke, albeit at different concentrations 33, it is conceivable that SHS may induce genotoxic and epigenetic effects similar to those already established for mainstream smoke. The well-characterized genotoxic and epigenetic effects of mainstream smoke include the formation of persistent DNA adducts at crucial cancer-related genes, and transcriptional silencing of cancer relevant genes, respectively (see, Fig. 2) 15,16,34. These genotoxic and epigenetic effects have already been observed in different in vitro and/or in vivo systems, including animal models and humans, e.g., cell culture, tissues, biopsies, etc., and have been shown to be related to tumor development. Future investigations should employ comprehensive footprinting of SHS-induced DNA adducts in relation to mutagenicity together with thorough analysis of DNA methylation status in relation to gene expression in cancer-related genes. Not only can these investigations improve our knowledge of the underlying mechanism(s) through which SHS may contribute to lung carcinogenesis, but they may also help identify specific biomarkers that can be used for early detection and prognosis of lung cancer as well as for assessment of its treatment strategies.""(xsd:string)
- rdfs:comment ""Several constituents of SHS are known carcinogens in experimental animals and/or humans, including aromatic amines, e.g., 4-aminobiphenyl, 2-naphthylamine, PAH, e.g., benzo[a]pyrene, tobacco specific nitrosamines, e.g., 4-(methylnitrosamine)-1-(3-pyridyl)-1-butanone (NNK), benzene, cadmium, nickel, etc. 9,22,33. The lists of the International Agency for Research on Cancer for known and suspected tobacco smoke carcinogens are presented in Web-Table 1, and Web-Tables 2, ,3,3, respectively 33. A genotoxic mode of action for some of these carcinogens has been elucidated, which relies upon their ability to either directly or after biotransformation generate electrophilic species capable of forming covalently bound DNA lesions, i.e., DNA adducts 9,13,22. Formation of DNA adducts is an event of potential significance in initiating carcinogenesis inasmuch as irreparable (persistent) DNA adducts may be misinstructional during DNA replication, thus, giving rise to mutations 34. Targeted mutations in key genes encoding proteins for, e.g., cell-cycle and growth control might lead to tumorigenesis (Fig. 2) 34,35. Furthermore, an epigenetic mode of action for some SHS-derived carcinogens can be proposed that involves DNA and histone modifications, which result in gene silencing without affecting the coding sequence of the turned-off gene (Fig. 2) 36,37. Epigenetic silencing of cancer-related genes has been shown to occur frequently and in the early stages of carcinogenesis 38,39. It is plausible that SHS-induced genotoxic and epigenetic effects, once identified mechanistically, can be utilized as integrated biomarkers of exposure and early effects for pulmonary carcinogenesis (see, Fig. 2)."(xsd:string)
- rdfs:comment ""Tobacco combustion results in the formation of mainstream smoke and sidestream smoke18. Mainstream smoke is generated during puff drawing from the burning cone and hot zone of a tobacco product, which travels through the tobacco column outward mouthpiece 19. Sidestream smoke is emitted from the smoldering coal of a tobacco product into the air between puffs 20. Both mainstream smoke and sidestream smoke are comprised of (I) vapor phase containing volatile agents, e.g., benzene, vinyl chloride, acrolein, etc. and (II) particulate phase (tar) containing semi-volatile and non-volatile agents, such as alkaloids, e.g., nicotine and its derivatives, aromatic amines, polycyclic aromatic hydrocarbons (PAH), etc. 21,22. For the most part, the chemical compositions of sidestream smoke and mainstream smoke are qualitatively similar 13. However, because sidestream smoke is produced at lower burning temperature, the quantities of its chemical constituents in both vapor and particulate phase differ from those of mainstream smoke, e.g., sidestream smoke is richer than mainstream smoke in certain carcinogens, e.g., aromatic amines 10,19,22,23. SHS is a mixture of mainstream smoke and sidestream smoke 10,23. Whereas sidestream smoke comprises ~85% of total SHS, mainstream smoke constitutes <15% of the overall SHS, i.e., the smoke first inhaled by an active smoker and then exhaled, while being briefly retained in the lung

and scrubbed of some of its constituents, most notably, nicotine, carbon monoxide, and much of the particulate matter 24. Minor contributors to SHS include small amounts of smoke that escape during puff drawing from the burning cone of tobacco product and some vapor phase agents that diffuse through the wrapping materials e.g., cigarette paper, into the air 10. Once released into the environment, SHS may further aggregate with pollutants already present in the air and change character 10,23. Thus, the physicochemistry of SHS might considerably be different from that of mainstream smoke 10,19,22,23. Nonetheless, most toxic or carcinogenic agents present in mainstream smoke can also be found in SHS, of course, in different concentrations due to aging and dilution with ambient air 10,19,23. Smokers, who actively inhale massive doses of mainstream smoke-carcinogens, have higher intake of carcinogens relative to SHS-exposed individuals. However, the observation that sidestream smoke-condensate is more potent than mainstream smoke-condensate in inducing mouse skin tumors has given rise to the idea that SHS imposed on non-smokers might be even more carcinogenic than mainstream smoke inhaled by active smokers 25,26.”"(xsd:string)

Superclasses (1)

- Smoke_LC

Disjoints (2)

Second-hand_Smoke_LC, Smoking_LC

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