Cigarette Smoke And Oxidative Stress

This book is unique due to its collective approach to the key aspects of the interdependency between three entities: oxidative stress, tobacco smoke and carcinogenesis, operating in a cause-effect sequence, in a concise and to the point manner. This book will prove to be a helpful companion to the internist, the oncologist and the research scientist in molecular biology as well as a work of reference for the general practitioner and physician interested in cancer research.

Tobacco smoking is a major public health issue worldwide. Smoking causes more than a quarter of all cancer deaths, with nearly 80% of deaths from lung cancer, 80% of deaths from bronchitis and emphysema and almost one fifth of deaths from cardiovascular disease. The role of tobacco smoke in the development of cancer, cardiovascular and respiratory diseases is widely discussed in the chapters of this book. It is responsible for many preventable diseases, contributes to a large number of premature deaths and accounts for enormous economic costs. The chapters in this book review a variety of topics related to the sociodemographic characteristics of people consuming tobacco, tobacco product promotion and merchandising consequences of smoking on health, the studied mechanism of damage and the different interventions promoted for tobacco control. The mechanisms by which cigarette smoke affects health are diverse. Thousands of chemical components -- mainly toxins and carcinogens -- are part of tobacco smoke. These components could act through specific or nonspecific mechanisms in the development of cancer, cardiovascular and respiratory disease. Common pathways include DNA damage, gene mutations, vasomotor dysfunction and oxidative stress, among others. The effects on health of first-hand and second-hand smoke exposure have been widely studied, and there is growing evidence regarding consequences of third-hand smoke exposure. The constituents, dynamic transformation and distribution of third-hand smoke are a fruitful area of study, as much as the quantification of its exposure. In this book, many useful indicators of exposure to environmental tobacco smoke, ranging from surrogate indicators to direct measurements of the components that reflect dose are analysed. Advances in this field can provide useful information on the extent and effects of smoking, implementing and assessing tobacco control policies. Furthermore, the World Health Organization developed a framework for an international treaty that provides evidence-based recommendations for health promotion and tobacco control. After more than ten years of its implementation, the effectiveness of different strategies adopted worldwide is analysed and reflections on the new challenges of its implementation are presented. In this book, smoking is reviewed pertaining to the effects and implications for health, as well as the current challenges on implementation and evaluation of tobacco control interventions.

This report considers the biological and behavioral mechanisms that may underlie the pathogenicity of tobacco smoke. Many Surgeon General's reports have considered research findings on mechanisms in assessing the biological plausibility of associations observed in epidemiologic studies. Mechanisms of disease are important because they may provide plausibility, which is one of the guideline criteria for assessing evidence on causation. This report specifically reviews the evidence on the potential mechanisms by which smoking causes diseases and considers whether a mechanism is likely to be operative in the production of human disease by tobacco smoke. This evidence is relevant to understanding how smoking causes disease, to identifying those who may be particularly susceptible, and to assessing the potential risks of tobacco products.

Effects of Chinese Green Tea on Cigarette Smoke-induced Oxidative Stress, Inflammation and Proteases/anti-proteases in Rat Lung in Vivo

Volume 1

Secondhand Smoke Exposure and Cardiovascular Effects

Smoking Prevention and Cessation

Relationship Between Oxidative Stress, Cigarette Smoking, and Periodontal Disease

Linking Individual Chemicals to Human Diseases

This is the second volume of the comprehensive, two-volume work on oxidative stress in lung diseases. Adopting a multidisciplinary approach, it demonstrates the cellular and molecular mechanisms associated with ROS (reactive oxygen species)-induced initiation and progression of a variety of lung diseases, such as COPD, emphysema, asthma, cystic fibrosis, occupational pulmonary diseases and pulmonary hypertension and discusses points for therapeutic intervention. The book also covers translational research and the latest research on prevention and therapeutics. Each chapter includes in-depth insights into the mechanisms associated with lung diseases and into identifying targets for drug development. Bridging the gap between fundamental and translational research, and examining applications in the biomedical and pharmaceutical industry, it is a thought-provoking read for basic and applied scientists engaged in biomedical research.

Tobacco smoking is the single most preventable risk factor related to the development of cardiovascular disease. It was demonstrated that tobacco smoke contains a thousand compounds potentially harmful to human health. As tobacco use declined over time, electronic cigarettes were introduced as an alternative. E-cigarettes are a modern and technological surrogate of traditional cigarettes and use heat to convert a nicotine solution or a flavored nicotine-free solution into vapor. Even though all the ingredients contained in the liquid of E-cigarettes are approved as food additives, the harmlessness of these electronic devices is still not fully proven in humans. The general mechanisms by which smoking results in cardiovascular events include the development of atherosclerotic changes with a hypercoagulable state and an increased risk of thrombosis. Endothelial dysfunction has been recognized as a hallmark of preclinical systemic atherosclerosis and as a useful marker to stratify the risk of cardiovascular disease. Based on these considerations, in this chapter, we (1) discussed the role of endothelial dysfunction and its contributing factors, such as oxidative stress and inflammation, in the development of cardiovascular diseases and (2) reported the studies which investigated the effect of tobacco and electronic smoking on the biomarkers of endothelial dysfunction, oxidative stress, and inflammation.

The health effects of tobacco smoke on smokers are well defined. However, the effects on non-smokers are not so clear. Which of the many diseases, cancers, and pathologies that are certainly associated with smoking are also induced by tobacco smoke in non-smokers? What are the effects on non-smokers of smoking bans in the workplace and changes in advertising? How can we effectively curtail the effects of environmental tobacco smoke (ETS)? Environmental Tobacco Smoke brings together in one source the key observations on the nature and effects of exposure to environmental tobacco smoke. The book focuses on the pathological effects of ETS on pregnant women, newborns, youths, adults, and the elderly. In addition, it investigates ETS' contribution to the development of asthma, tobacco allergy, heart disease, and cancer. The book also examines the role of ETS in bringing about other maladies such as DNA damage, gene activation, and immunosuppression. The materials also explore the problems associated with establishing incontrovertible links between ETS and health problems in non-smokers. Environmental Tobacco Smoke also probes the role of the political and legal systems in modifying behaviors, exposure risks, and health consequences of ETS. The book also summarizes the role of antioxidant supplements in lowering ETS damage and the usefulness of animal models in refining the precision of studies. Clearly, environmental tobacco smoke poses significant health risks. It is also abundantly clear that these risks can be eliminated. It is even more obvious that, in order to establish effective prevention mechanisms, we need to define the extent of health damage attributable to ETS. Environmental Tobacco Smoke provides a plethora of information that educates us on the effects of environmental tobacco smoke on the non-smoking public and thereby equips us to eradicate the problems created by ETS.

Effects of Cigarette Smoke on Antibiotic Resistance and Oxidative Stress Response of Pseudomonas Aeruginosa

A Report of the Surgeon General

Mechanisms and Treatment

Effects of Human Mesenchymal Stem Cells on Cigarette Smoke-Induced Lung Damage

Comparison of Severity of DNA-damage in Gastric Tissue, Levels of Nitric Oxide and Oxidative Stress in Gastric Juice of

The Involvement of Serotoninergic System in Cigarette Smoke-Induced Oxidative Stress and Inflammation

Smoking was and remains one of the most important public healthcare issues. It is estimated that every year six million people die as a result of tobacco consumption. Several diseases are caused or worsened by smoking: different cancer types, heart disease, stroke, lung diseases and others. In this book we describe the different toxic effects of smoke on the human body in active and in passive smokers. It is also well known that many people who smoke wish to quit, but they rarely succeed. Smoking prevention and cessation are of utmost importance, thus we also describe different strategies and aspects of these issues. We hope that this book will help readers to understand better the effects of smoking and learn about new ideas on how to effectively help other people to stop smoking.

Cardiorespiratory function is prominently affected by oxidative stress. Cigarette smoking is the archetype of oxidative and nitrative stress and free radical formation. New adverse effects of smoking keep on propping up in research. The chapters provide the comprehensive view of new developments in this area regarding cardiovascular and lung function and muscle catabolism. Alterations in inflammatory cytokines and proteins as well as degradation of muscle proteins due to smoking, by far unrecognized, caused by oxidative stress also are presented. Much less is known about the effect of cognitive stress on vagally-mediated cardiorespiratory function and surprisingly, on vagal immune pathway. The experimental studies also show that clinically important meconium aspiration syndrome contains an oxidative trait which is amenable to antioxidative treatment. This volume creates a source of information on the damaging role of oxidative stress in cardiorespiratory function that has by far not been available.

This first volume of the comprehensive, two-volume work on oxidative stress in lung disease introduces the molecular mechanisms, and the role of oxidants in the progression of different lung diseases. The lungs of humans and animals are under constant threat from oxidants from either endogenous (e.g. in situ metabolic reactions) or exogenous sources (e.g. air pollutants). Further, oxidative stress causes the oxidation of proteins, DNA and lipids, which in turn generates secondary metabolic products. The book consists of sections, each focusing on different aspects of oxidant-mediated lung diseases. As such it is a unique reference resource for postgraduate students, biomedical researchers and also for the clinicians who are interested in studying and understanding oxidant-mediated lung diseases. The second volume will incorporate other aspects of oxidant-mediated lung diseases, including prevention and therapeutics.

Cigarette Smoking

Oxidation: The Cornerstone of Carcinogenesis

Oxidative Stress, Inflammation, and Health

Health Effects and Challenges for Tobacco Control

Cytochrome P450 Enzymes and Oxidative Stress in Tobacco/nicotine Mediated HIV Pathogenesis

Oxidative Stress and Cardiorespiratory Function

This work tested the hypothesis that oxidation of methionine 388 in thrombomodulin is higher in cigarette smokers, and thus a likely contributor towards the hypercoagulable state in smokers. Thrombomodulin, a protein cofactor found on endothelial cell surfaces, regulates the activity of thrombin. Thrombin bound to thrombomodulin no longer converts fibrinogen to fibrin, but instead activates Protein C which, in turn, stops the coagulation cascade by inactivation of clotting factors. The oxidation of methionine 388 of thrombomodulin has been shown in vitro to dramatically decrease the anticoagulant cofactor activity of thrombomodulin. The blood of cigarette smokers is more prone to clot than that of non-smokers, a major factor in their premature deaths from cardiovascular disease. Cigarette smoke consists of many oxidizing species that impose oxidative stress on the body. These species include organic radicals and hydrogen peroxide, which can oxidize methionine. The fact that smokers are in a hypercoagulable state has been established, however the molecular origins of such a state have not been elucidated. Techniques were developed to isolate thrombomodulin cleared from the blood stream in urine, proteolytically digest it, and identify using mass spectrometry the peptide containing methionine 388 in both its oxidized and reduced forms. In many cases the oxidized version of the peptide was below the limits of detection in non-smokers and the reduced version was not detected in smokers. The intensity of these peaks in the mass spectra do not allow calculation of absolute percentages of oxidation because of differences in proton affinity of the two forms, but there is a very statistically significant difference (P=0.002 by Mann-Whitney Rank Sum test) in the apparent median reduced to oxidized ratios of >2.043 for non-smokers and of

Millions of Americans use e-cigarettes. Despite their popularity, little is known about their health effects. Some suggest that e-cigarettes likely confer lower risk compared to combustible tobacco cigarettes, because they do not expose users to toxicants produced through combustion. Proponents of e-cigarette use also tout the potential benefits of e-cigarettes as devices that could help combustible tobacco cigarette smokers to quit and thereby reduce tobacco-related health risks. Others are concerned about the exposure to potentially toxic substances contained in e-cigarette emissions, especially in individuals who have never used tobacco products such as youth and young adults. Given their relatively recent introduction, there has been little time for a scientific body of evidence to develop on the health effects of e-cigarettes. Public Health Consequences of E-Cigarettes reviews and critically assesses the state of the emerging evidence about e-cigarettes and health. This report makes recommendations for the improvement of this research and highlights gaps that are a priority for future research.

Data suggest that exposure to secondhand smoke can result in heart disease in nonsmoking adults. Recently, progress has been made in reducing involuntary exposure to secondhand smoke through legislation banning smoking in workplaces, restaurants, and other public places. The effect of legislation to ban smoking and its effects on the cardiovascular health of nonsmoking adults, however, remains a question. Secondhand Smoke Exposure and Cardiovascular Effects reviews available scientific literature to assess the relationship between secondhand smoke exposure and acute coronary events. The authors, experts in secondhand smoke exposure and toxicology, clinical cardiology, epidemiology, and statistics, find that there is about a 25 to 30 percent increase in the risk of coronary heart disease from exposure to secondhand smoke. Their findings agree with the 2006 Surgeon General's Report conclusion that there are increased risks of coronary heart disease morbidity and mortality among men and women exposed to secondhand smoke. However, the authors note that the evidence for determining the magnitude of the relationship between chronic secondhand smoke exposure and coronary heart disease is not very strong. Public health professionals will rely upon Secondhand Smoke Exposure and Cardiovascular Effects for its survey of critical epidemiological studies on the effects of smoking bans and evidence of links between secondhand smoke exposure and cardiovascular events, as well as its findings and recommendations.

The Impact of Cigarette Smoke on Cell Survival and Inflammation

L-Menthol Alleviates Cigarette Smoke Extract Induced Lung Injury in Rats by Inhibiting Oxidative Stress and Inflammation Via Nuclear Factor Kappa B, P38 MAPK and Nrf2 Signalling Pathways

Neuroscience of Nicotine

The Health Consequences of Involuntary Exposure to Tobacco Smoke

How Tobacco Smoke Causes Disease

The Biology and Behavioral Basis for Smoking-attributable Disease : a Report of the Surgeon General

The mechanism by which cigarette smoke causes or contributes to inflammatory diseases like chronic obstructive pulmonary disease, cardiovascular disease and cancer remains unclear. Recent developments in our knowledge of cellular signaling suggest that cigarette smoke may cause oxidative stress in cellular systems. The assessment, consequences and possible modulation of these effects are discussed in this book which will interest oncologists and researchers in Biochemistry.

This dissertation, "Effects of Chinese green tea on cigarette smoke-induced oxidative stress, inflammation and proteases/anti-proteases in rat lung in vivo" by Ka-ho, John, Chan, ???, was obtained from The University of Hong Kong (Pokfulam, Hong Kong) and is being sold pursuant to Creative Commons: Attribution 3.0 Hong Kong License. The content of this dissertation has not been altered in any way. We have altered the formatting in order to facilitate the ease of printing and reading of the dissertation. All rights not granted by the above license are retained by the author. DOI: 10.5353/th_b4519933 Subjects: Green tea - Therapeutic use Oxidative stress Inflammation Proteolytic enzymes Rats as laboratory animals

Smoking causes and contributes to a large number of human diseases, yet due to the large number of potentially hazardous compounds in cigarette smoke -- almost 5,000 chemicals have been identified, establishing the link between smoking and disease has often proved difficult. This unbiased and scientifically accurate overview of current knowledge begins with an overview of the chemical constituents in cigarette smoke, their fate in the human body, and their documented toxic effects on various cells and tissues. Recent results detailing the many ways components of cigarette smoke adversely affect human health are also presented, highlighting the role of smoking in cardiovascular, respiratory, infectious and other diseases. A final chapter discusses current strategies for the treatment and prevention of smoking-induced illness. Despite the obvious importance of the topic, this is the first comprehensive reference on tobacco smoke toxicity, making for essential reading for all toxicologists and healthcare professionals dealing with smoking-related diseases.

Public Health Consequences of E-Cigarettes

Omega-3 Polyunsaturated Fatty Acids Protect Against Cigarette Smoke-Induced Oxidative Stress and Vascular Dysfunction

Oxidation and Tobacco Smoke Carcinogenesis. A Relationship Between Cause and Effect

Relevant to Chronic Obstructive Pulmonary Disease

The Involvement of Serotoninergic System in Cigarette Smoke-induced Oxidative Stress and Inflammation

Making Sense of the Evidence

In the US, the prevalence of cigarette smoking in the HIV-infected population is 50-70% compared to 15-20% in the general population, which further increases the risk of smoking-related health problems in this group. For example, HIV-infected smokers show decreased immune responses, poorer responses to antiretroviral therapy (ART), and greater risk of virological rebound, compared to HIV-infected non-smokers. Several in vivo and in vitro studies have shown that smoking/nicotine is associated with decreased immune responses, increased inflammation, increased oxidative stress, and increased numbers of opportunistic infections. Furthermore, in vitro studies have shown that smoking/nicotine enhances HIV replication in alveolar macrophages, microglia, and T cells. However, the mechanism by which smoking or nicotine increases HIV replication is largely unknown. As an exception, a report suggests that iron and oxidative stress are possible mechanisms of enhanced production of HIV by alveolar macrophages in cigarette smokers. The role of CYP enzymes has not been studied in context with smoking/nicotine and HIV pathogenesis. However, there are several elegant studies that show the involvement of CYP2A6, CYP2A13, CYP1A1, and CYP1B1 in smoking/nicotine-mediated toxicity leading to various types of cancers and hepatic toxicity. The majority of tobacco constituents, including nicotine, are metabolized and/or activated by CYP enzymes to generate reactive oxygen species (ROS) and/or reactive metabolites, NNK. Based on the fact that CYP pathways play a critical role in smoking mediated cell/organ toxicity, there is a critical need to explore the involvement of CYP pathways in smoking/nicotine-mediated HIV pathogenesis. The present study was based on the central hypothesis that tobacco/nicotine, or its metabolites, enhance HIV replication in monocytes/macrophages through CYP pathway. The key findings of our studies presented in this thesis indicate that (a) CYP2A6-mediated metabolism of nicotine increased the generation of ROS in HIV cell model, SVGA astrocytes, (b) Cigarette smoke condensate caused significant induction of CYP1A1, CYP2A6, ROS production, and cytotoxicity in U937 monocytes as well as enhanced HIV replication in HIV-infected primary macrophages, (c) Mild-to-moderate smoking increased viral load in HIV-infected individuals. Furthermore, our study suggested that smoking and HIV independently increase oxidative stress in the plasma as well as in monocytes. (d) There was a decrease in the level of nicotine and subsequent increase in the level of nicotine metabolites, suggesting an increase in nicotine metabolism in HIV-infected smokers compared with uninfected smokers. In conclusion, our in vitro and ex vivo results are consistent with the hypothesis that CYP and CYP-mediated oxidative stress by tobacco/nicotine are associated with increased viral load by smoking/smoking constituents. This study has clinical implications in terms of targeting CYP and oxidative stress pathways to find potentially novel therapeutic interventions, as well as drug dose adjustment to treat HIV-infected smokers effectively.

In the United States, cigarette smoking holds a significant portion of preventable causes of death and disease. Studies have shown that cigarette smoke can modify the human defense system as well as leading the cigarette smoker to have higher risk bacterial infection. However, the field of host-pathogen interactions after exposure to cigarette smoke is still poorly understood. In this study, we focus on the effects of cigarette smoke on the virulent properties of Pseudomonas aeruginosa (PSA), an important airway pathogen that is extremely detrimental to cystic fibrosis and immunocompromised patients. We uncover that PSA exposed to cigarette smoke extract (CSE) demonstrated increase antibiotic resistance to levofloxacin and gentamicin. In our current paper under review, we have demonstrated that

PSA exposed to CSE (CSE-PSA) showed increase resistance to oxidative burst by neutrophil and hydrogen peroxide treatment. To examine the underlying mechanism of these increases in virulence, RT-qPCR was used on control- and CSE-PSA to observe changes in gene expression. Genes that encode parts and regulation of PSA efflux pumps (mexA, mexX, mexZ) showed increased gene expression in CSE-PSA over control-PSA. Of three oxidative stress response genes (gpx, oxyR, tpx), only tpx, a gene encoding a thiol peroxidase homolog, demonstrated statistical significance. However, all three genes showed a trend of overall increase, suggesting that cigarette smoke is inducing changes in PSA gene expression. We conclude that cigarette smoke increases virulence in PSA virulence by increasing gene expression involved in antibiotic resistance and oxidative stress.

The FHIT gene is located on the most fragile site in the human genome. FHIT gene deletions are among the earliest and most frequent events in carcinogenesis, particularly in carcinogen-exposed tissue. Previous work in mouse and cell culture models established FHIT to be an authentic tumor suppressor. Re-expression of FHIT in cell culture causes cell death via initiation of apoptosis, but the precise mechanism underlying this process is unclear. It is well established that cellular transition from normal to transformed occurs in multiple steps and requires the accumulation of several genetic changes. Relying on the compelling phenotype of tumor development in FHIT knockout mice, this project aimed to elucidate a mechanism through which FHIT-deficient cells are primed to survive multiple genetic and environmental stresses, and promote progression of cancer. My work indicates that FHIT expression is required for the normal cellular response to oxidative stress, and presents evidence that in the absence of FHIT, an oxidative stress response pathway is superinduced. When FHIT is depleted from cells exposed to cigarette smoke, the expression of a subset of oxidative stress response genes is enhanced. Enhanced activation of these genes can occur as an adapative response to stress induced by reactive oxygen species production, and is frequently detected in cancer. Investigation into the mechanism underlying the enhanced gene expression determined that FHIT loss is associated with decreased levels of the transcriptional repressor Bach1. In this manner, we propose that loss of Fhit supports an antioxidant program that is pivotal in establishing and maintaining carcinogenic transformation.

Environmental Tobacco Smoke

The Role of Oxidative Stress and VEGF/KDR Signalling and Its Implications for COPD.

The Role of Oxidative Stress and VEGF/KDR Signalling and Its Implications in COPD

Effects on Mitochondrial Function, the Lipidome and Glucocorticoid Responsiveness in Airway Epithelium

Effects of Chinese Green Tea on Cigarette Smoke-Induced Oxidative Stress, Inflammation and Proteases/Anti-Proteases in Rat Lung in Vivo

Volume 2

Neuroscience of Nicotine: Mechanisms and Treatment presents the fundamental information necessary for a thorough understanding of the neurobiological underpinnings of nicotine addiction and its effects on the brain. Offering thorough coverage of all aspects of nicotine research, treatment, policy and prevention, and containing contributions from internationally recognized experts, the book provides students, early-career researchers, and investigators at all levels with a fundamental introduction to all aspects of nicotine misuse. With an estimated one billion individuals worldwide classified as tobacco users—and tobacco use often being synonymous with nicotine addiction—nicotine is one of the world's most common addictive substances, and a frequent comorbidity of misuse of other common addictive substances. Nicotine alters a variety of neurological processes, from molecular biology, to cognition, and quitting is exceedingly difficult because of the number of withdrawal symptoms that accompany the process. Integrates cutting-edge research on the pharmacological, cellular and molecular aspects of nicotine use, along with its effects on neurobiological function Discusses nicotine use as a component of dual-use and poly addictions and outlines numerous screening and treatment strategies for misuse Covers both the physical and psychological effects of nicotine use and withdrawal to provide a fully-formed view of nicotine dependency and its effects

This dissertation, "Effects of Human Mesenchymal Stem Cells on Cigarette Smoke-induced Lung Damage" by Xiang, Li, , was obtained from The University of Hong Kong (Pokfulam, Hong Kong) and is being sold pursuant to Creative Commons: Attribution 3.0 Hong Kong License. The content of this dissertation has not been altered in any way. We have altered the formatting in order to facilitate the ease of printing and reading of the dissertation. All rights not granted by the above license are retained by the author. Abstract: Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease characterized by persistent airway obstruction that is only partially reversible. It is the fourth leading cause of death and is predicted to be the third by 2030. The progression of the disease involves chronic inflammation, oxidative stress, excess protease activity, increased lung cell apoptosis and accelerated lung aging, but the exact pathogenesis is still unclear. The major cause of COPD is cigarette smoking(CS). Although COPD is associated with increasing social and economical burden, there have been few advances in pharmacological therapy of COPD. Mesenchymal stem cells (MSCs) are fibroblast-like multipotent stem cells which can be isolated from a broad range of sources including bone marrow (BM) and adipose tissue. Administration of BM-derivedMSCs (BM-MSC) or adipose tissue-derived MSCs was reported to attenuate CS-induced emphysema in murine models. Induced pluripotent stem cell-derived MSC (IPSC-MSC) are MSCs differentiated from induced pluripotent stem cells(IPSCs), which are pluripotent cells generated by somatic cell reprogramming in vitro. IPSC-MSCs have several advantages over BM-MSC, including more abundant sources and high capacity of doubling without loss of differentiation potency. A general exploration and comparison on the effects of human IPSC-MSC and BM-MSC treatments were carried out in a 56-day CS-exposed rat model. Compared to BM-MSC, IPSC-MSC showed a higher capacity to reside in lung tissue. The two treatments shared similar efficacy to attenuate CS-induced lung cell apoptosis, to restore CS-induced reduction of lungIL-10and to alleviate CS-induced elevation of systemic TGF-β1. In addition, IPSC-MSC was found to cause reduction in CS-induced elevation of systemic oxidative stress and reversal of CS-induced reduction of lung adiponectin. Furthermore, in order to understand the possible paracrine mechanism involved, human airway epithelial cells were treated with IPSC-MSC or BM-MSC-conditioned medium in a cell culture system in the presence of cigarette smoke medium (CSM). Potentiation rather than attenuation of CSM-induced release of pro-inflammatory cytokine IL-8, MCP-1 and IL-6 was observed with IPSC-MSC or BM-MSC conditioned medium. It is currently unknown whether cultured IPSC-MSCs or BM-MSCs will release pro-inflammatory mediators into the conditioned medium or not. In order to study CS-induced oxidative stress and inflammation in a short time frame, anacute (5-day) CS-exposed rat model was established in juvenile and adult groups. An age-dependent alteration of CS-induced oxidative and inflammatory responses was demonstrated in this model. In summary, our in vivo rat model provides a platform for elucidating the effects of stem cell treatment in CS-induced oxidative stress and inflammation, leading to lung damage. Our findings suggest that treatment of IPSC-MSC or BM-MSC might be able to slow down CS-induced disease progression, possibly through anti-oxidant, anti-inflammatory and anti-apoptotic properties. However, caution should be taken as our in vitro data revealed that conditioned medium from MSCs may provoke pro-inflammatory responses. Further studies on the regulation of the activity of MSCs in vivo will be needed before developing IPSC-MSC into cell therapies for COPD to halt the progression over time. DOI: 10.5353/th_b4961820

Specifically focusing on the redox regulation of cell signaling responsible for oxidative stress and inflammatory tissue damage, this reference provides a comprehensive overview of cutting-edge research on the intracellular events mediating or preventing oxidative stress and pro-inflammatory processes induced by endogenous and xenobiotic factors-analyzing the implications of oxidative stress and inflammatory damage in the pathogenesis of human disorders such as cancer, neurodegenerative disease, and diabetes.

Effects of Commonly Used Air Filters on Secondhand Tobacco Smoke and the Induction of Oxidative Stress and Inflammation in Mice

Cigarette Smoke as a Risk Factor for Delayed Fracture Healing

Cigarette Smoke Toxicity

Role of Oxidative Stress Impairs Primary Cilia on Osteogenic Differentiation

Oxidative Stress in Lung Diseases

Cigarette Smoking & DNA-damage