484. Translational models of airway disease

4683

Bitter taste receptor agonists as a novel class of bronchodilators in guinea-pig airways

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Rationale: Deshpande et al. (Nat Med 2010) reported that several bitter taste receptor (TAS2R) agonists evoked relaxation of mice and human airways. We examined the effects of three prototype agonists in segments of guinea pig trachea (GPT).
Methods: GPT was pre-contracted with 0.1 μM carbachol in both absence and presence of 3 μM indomethacin or prostaglandin antagonists. The segments were either exposed to denatonium, chloroquine or saccharin, or kept untreated. Expression of TAS2Rs in guinea pig tracheal epithelium and smooth muscle was measured with real-time PCR.
Results: Denatonium and chloroquine induced concentration-dependent relaxations whereas saccharin had no effect. In consistency with these findings, there was expression of TAS2R4 and TAS2R10 for denatonium, and TAS2R3 and TAS2R10 for chloroquine, but not of TAS2Rs for saccharin in guinea pig airways. Denatonium was 6.1-fold more potent than chloroquine (pEC_{50} 4.7±0.1 and 3.8±0.1, respectively). Indomethacin had no effects on the potency of denatonium and chloroquine. However, the magnitude of the denatonium-induced relaxation (57.5±5.2%; n=8) was enhanced by indomethacin (97.7±2.3%; n=8) and the prostaglandin E_2 receptor (EP_1) antagonist ONO-8310 (99.3±0.7; n=5). Chloroquine induced almost complete relaxation (98.2±1.1%; n=6) that was unaffected by indomethacin (99.9±0.04%; n=7).
Conclusion: Denatonium and chloroquine induced relaxation of GPT and their respective TAS2Rs were expressed. There was an interaction between denatonium and PGE_2 acting on EP_1 receptors. The findings support the concept that airway TAS2Rs represent a novel target for anti-asthmatic therapy.

4684

Prostacyclin modifies VEGF synthesis in fibroblasts from healthy and COPD patients

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Background: Involvement of vascular remodelling in the lung is a characteristic sign in chronic obstructive lung disease (COPD). The vascular mediator prostacyclin may regulate fibroblast activity. The objective was to study the effect of prostacyclin on synthesis of vascular endothelial growth factor (VEGF) and interactions with transforming growth factor (TGF) B_1 in distal lung fibroblasts from patients with COPD and control subjects.
Method: Primary human lung fibroblast cultures were established from peripheral airway biopsy samples from healthy individuals (controls, n=5) and after lung resection from patients with COPD (GOLD IV) (n=7). The lung fibroblasts were cultured in 0.4% medium and stimulated with the cyclooxygenase inhibitor indomethacin 3μM in combination with the prostacyclin analogue iloprost 1μM and TGF-B 10ng/ml for 24h. VEGF production was measured in the cell culture supernatant by ELISA.

Results: Iloprost enhanced VEGF synthesis in both fibroblasts from control subjects (774±171 vs 438±55 pg/ml, $p<0.05$) and patients with COPD (350±34 vs 512±81 pg/ml, $p<0.05$). TGF-B increased the production of VEGF 5-fold in fibroblasts from control subjects ($p<0.05$) and 6.1-fold from patients with COPD ($p<0.01$). However, iloprost showed no effect on VEGF synthesis after TGF-B stimulation, whereas indomethacin reduced VEGF production in fibroblasts from patients with COPD ($p<0.05$) but not in control subjects.
Conclusions: Iloprost enhanced VEGF synthesis in fibroblasts from both healthy and patients with COPD. Though, iloprost had no effect on VEGF after TGF-B_1 stimulation. This data implicate that also other prostanoids are involved in the regulation of VEGF in fibroblasts from COPD patients.

4685
Capsazepine inhibits NF-κB subunits in dsRNA-exposed human bronchial epithelial cells from asthmatic and COPD donors
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Background: Novel drugs are needed to treat viral-induced exacerbations of asthma and COPD. We have shown that capsazepine (CPZ) inhibits viral-induced cytokine production (including thymic stromal lymphopoietin, TSLP) in human asthmatic bronchial epithelial cells (HBEC) more effectively than steroids.
Objective: Investigate NF-κB mechanisms potentially involved in actions of CPZ in HBECs from patients with asthma and COPD.
Methods: Primary HBEC were obtained by fibre optic bronchoscopy from individuals with asthma (n=6) and COPD (n=3), then grown in 12-well plates and stimulated with viral surrogate dsRNA (10 μg/ml) to induce pro-inflammatory cytokine (TSLP, TNF-α, IL-8, IFN-β) mRNA expression (RT-qPCR) and production (ELISA). CPZ (3-30 μM) was added 1 h prior to dsRNA. NF-κB-signaling was studied (western blot) using specific antibodies for NF-κB subunits p65, p105 and IκB-α. De novo gene synthesis of IκB-α was studied by RT-qPCR.
Results: dsRNA induced marked expression and release of TSLP, TNF-α, IL-8, and IFN-β ($p<0.001$). These effects were dose-dependently reduced ($p<0.05$-$p<0.001$) by CPZ that also tended to inhibit TSLP more than IFN-β ($p=0.09$). dsRNA induced degradation and de novo gene synthesis of IκB-α and increased the release of p65 and p105 ($p<0.01$). CPZ prevented the degradation of IκB-α and inhibited p65 and p105.
Conclusion: CPZ effectively reduced dsRNA-induced cytokine overproduction in HBECs from asthmatic and COPD donors. CPZ inhibited dsRNA-induced IκB-α degradation preventing dissociation and translocation of p65 and p105 to the nuclear NF-κB promoter. CPZ may stabilize IκB-α thus inhibiting NF-κB-dependent cytokine production.

4686
Bradykinin-induced contractions in guinea pig trachea after incubation and culture
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Background: Inflammatory conditions can alter responsiveness to the endogenous mediator bradykinin (BK) in mouse and human airway smooth muscle. Guinea-pig and human airways generally display similar smooth muscle responsiveness. The aim of this study was to develop an *in vitro*-model in guinea pig trachea (GPT) to study the regulation of BK responses by culture procedures.
Methods: The contractile response to BK was determined in organ tissue baths. Either fresh, organ bath incubated (6-12 hours) or in DMEM/ F12 media cultured (4 days) GPT segments were used for these measurements. The contractile responses to BK were determined in the presence of 3μM indomethacin and 10μM captopril. Contractions were presented as a percentage of the maximal contraction of the GPT.
Results: In fresh GPT, BK induced only weak contractions (E_{max}: 8±2%). This response was not changed after 6 hours incubation, however after 12 hours incubation a strong enhancement of the BK induced contraction was observed (E_{max}: 53±2%, pEC_{50} 7.4±0.2). This up-regulated response was partly regulated by prostanoids, since both E_{max} (28±2%) and pEC_{50} (6.3±0.1) were decreased in segments incubated for 12 hours with indomethacin. In contrast to the 12-hour incubation in the tissue bath, the contractile response to BK after 4 days organ culture was only slightly enhanced (E_{max}: 21±3%).
Conclusion: This study shows that contractions in GPT to BK are inducible over time. The different effects of incubations in the tissue bath and the organ culture may be used to study different aspects of regulation of BK responsiveness in airway inflammation.

4687
Protective effect of a protein epitope mimetic (PEM) CCR10 antagonist, POL7085, in an allergic model of asthma
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Potential involvement of the CCR10/CCL28 axis was recently reported in a murine model of allergic asthma [1]. Blockade of the CCR10 receptor might therefore represent a novel alternative to the current treatment of asthma. We have evaluated the effect of the PEM CCR10 antagonist, POL7085, in an allergic model of asthma in mice. Nine week-old male Balb/c mice were sensitized to ovalbumin (OVA) administered intraperitoneally in the presence of alum (D0 and D7), and challenged to OVA administered intranasally (D18 to D21). POL7085 was administered once daily 1 hour before each OVA challenge at 9 and 18mmol/kg intra-nasally (I.N.) *vs* dexamethasone I.N. (DEX; 2.3mmol/kg) *vs* vehicle.
In vehicle treated animals, OVA induced airway hyperresponsiveness (AHR) as measured by whole body plethysmography, and hypereosinophilia in the bronchoalveolar lavage (BAL) fluid. POL7085 dose-dependently and significantly decreased AHR by 34±16% and eosinophil numbers in BAL by 66±6%. In addition, the higher dose of POL7085 also inhibited IL-5 secretion in BAL (42±13%), IgE and IgG1 synthesis in serum (47±31% and 61±15%, respectively), and lung collagen synthesis (43±11%), although not significantly. POL7085 as compared to DEX also modified body (6.5±1.7% *vs* 4.5±1.5% for DEX) and spleen weight (24±3% *vs* 44±3% for DEX).
In conclusion, the PEM CCR10 antagonist, POL7085, significantly and dose-dependently decreased asthma symptoms after once daily local administration, in particular AHR and eosinophilia. Blocking the CCR10 chemokine receptor therefore appears as a promising novel approach for treating asthma.
Reference:
[1] English et al, Immunol Lett; 2006;103:92-100.

4688
Characterisation of TNF-alpha lectin-like domain derived peptides associated with improved alveolar fluid clearance in pulmonary oedema
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The beneficial effect of the lectin-like domain of TNF-alpha, including the TIP peptide which mimics this domain, on activation of oedema resorption, improved alveolar clearance and protection of lung function after transplantation, is well documented from several independent *in vitro and in vivo* studies using animal models. The effect is mediated by activation of sodium uptake through the amiloride-sensitive epithelial sodium ion channel (ENaC), which plays a major role in alveolar fluid clearance in normal and diseased lungs. Several peptides mimicking the lectin-like domain of human TNF-alpha, and differing from the human TIP peptide by mutation or substitution of amino acid residues with non-natural derivatives, were synthesised and tested for their ability to enhance sodium current through ENaC in A549 cells with the patch clamp technique.
For all ENaC-activating TIP peptides a maximum effect was observed at 120 nM. Compared to TNF-alpha (EC_{50} 8.2 nM) and the original human TIP peptide (EC_{50} 54.3 nM), several of the newly-designed peptides were more effective at enhancing amiloride-sensitive current than the latter: the most active peptide had an EC_{50} of 19.9 nM. Our data suggest that TIP peptides with charge distribution and interatomic distances most closely resembling the 3D structure of the native lectin-like domain of TNF-alpha, are those with greater ability to enhance activation of sodium current through ENaC. No standard therapy exists for pulmonary oedema, thus these TIP peptides represent promising therapeutic agents for activating sodium uptake from the alveolar fluid through ENaC and improving clinical outcome in this condition.

4689
Consequences of chronic pulmonary TLR9 activation in the lung and beyond
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Background: Toll like receptor 9 (TLR9) agonist CpG-ODN is being explored as an anti-allergic drug for asthma. However TLR9 could play a role in COPD. Cigarette smoke induced IL-8 production is partly TLR9 mediated.
Aim: To investigate the (extra)-pulmonary effects of CpG-ODN. We hypothesized that pulmonary TLR9 activation induces neutrophil influx which could lead to adverse effects.
Methods: A single dose of 0.01, 0.05 or 0.25nMol/gBW GpG-ODN was targeted to balb/c mice lungs by aspiration (acute). Next 0.01nMol/gBW GpG-ODN was administered repeatedly for 5 days (subchronic) or 5 weeks (chronic). 24 hours after last exposures, measurements were done: lung function; hypertension and

heart hypertrophy; lung weight; blood and bronchoalveolar lavage (BAL) analysis; morphology of unlavaged lungs.
Results: Total BAL cells were increased in all CpG-ODN mice ($p<0.05$). PMNs were increased in blood (30%) and BAL (39%) acute which persistent upon prolonged exposure duration. Blood lymphocytes were subchronically 19% decreased which was reflected in an abundant pulmonary (sub)- chronic lymphocyte influx. Chronic exposure leads to 21% decreased peak expiratory flow (ml/sec) and 71% increased airway resistance (cm H2O/ml/sec). Right ventricle heart hypertrophy was observed upon chronic exposure; ratio right ventricle weight/total heart weight CpG-ODN=0.23±0.007; control=0.19±0.008. Wet lung weight was 15.2% increased subchronically and 70.8% chronically.
Conclusion: An interplay between neutrophils and lymphocytes could possibly play a role in TLR9 induced adverse pulmonary and cardiovascular effects. Caution is needed when CpG-ODNs are chronically administered for therapeutical purposes.
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4690
Efficacy of the TRPV1 antagonist SB-705498 in an MRI guinea pig model of rhinitis
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Rationale: Antagonism of nasal TRPV1 receptors is a potential target for inhibition of symptoms associated with rhinitis. Here, we explore the pharmacology of SB-705498 on the contralateral nasal secretory response to ipsilateral nasal capsaicin challenge in the guinea pig.
Methods: Experiments were conducted on intranasally sensitised guinea pigs. Absolute nasal fluid volumes were measured using MRI techniques that provide images of the total nasal cavity of the guinea pig. Animals were pre-treated with SB-705498 or vehicle (-1, -6 and -24h) and MRI scanned to produce first a baseline measurement and then a further measurement at 10 minutes post capsaicin challenge (0.3mM; 50ul).
Results: 1 hour pre-treatment with SB-705498 resulted in approximately 50% inhibition of the secretory response at 10mg/kg (oral; $p<0.05$), 1mg/kg (intranasal suspension; $p<0.005$) and 0.1mg/kg (intranasal particle reduced suspension; $p<0.0005$). At 12 hour pre-treatment, approximately 60% inhibition ($p < 0.0001$) was observed with SB-705498 at 1mg/kg (intranasal particle reduced suspension); at 24h this had reduced to ~50% inhibition ($p<0.01$).
Conclusion: These studies demonstrate that SB-705498 inhibits capsaicin-induced intranasal parasympathetic responses in guinea pig. Topical application of SB-705498 was associated with a approximately 10-fold reduction in total dose compared with oral administration. In addition, particle reduction of SB-705498 was associated with a further improvement in intranasal potency.

485. Smoking-related disorders

4691
Late-breaking abstract: Effectiveness of easy smoking cessation clinic in tertiary health care settings: Observational study of cohorts
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Introduction: Tobacco treatment programs should be offered in clinical settings for all smokers who need to quit smoking. We have co-operated with every units and departments in hospital and changed our service patterns of smoking cessation clinic into easy way. Cessation assistance was provided on working time every day except holiday. It requires approximately 20-30 minutes to complete and involves asking patients about smoking behavior and acting to help them quit. Telephone helpline was used to follow up and encourage smokers trying to continue quit smoking.
Methods: This is an observational study of cohorts of participants in smoking cessation clinic, Buddhachinnaraj hospital during June 1 to November 30, 2009. The main outcome measurements were self report abstinence rate at 6 and 12 months, and cost per quit.
Results: Over a period of 6 months, a cohort of 315 smokers were enrolled in this study.The self report abstinence rate at 6 and 12 months was 33.7% (106/315) and 27.9% (88/315).The mean cost per quit was 3,145 baht (70 Euro).Lost follow up rate by telephone helpline at 6 and 12 months was 18.4% and 27.6%.
Conclusions: Easy smoking cessation clinic is the intervention that are simple, cheap, and effective.Strategies for incorporating effective smoking cessation clinic into routine clinical care needs to become a key part of routine intervention for managing smoking cessation.

4692
LSC 2011 Abstract: Evaluation of morpho-functional changes in airways of young cigarette smokers
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We assumed that even in asymptomatic young smokers, with relatively short smoking duration and normal lung function, induced sputum could be found some changes indicative for early inflammatory process.
Aim: The aim of this study was to evaluate morpho-functional changes in airways of young cigarette smokers.
Method: We enrolled 23±3 years old 12 non-allergic smokers (1.59±0.67 pack-years) and 7 healthy non-smoking volunteers. Lung function measurements, sputum induction (IS) and sputum cell analysis were performed.
Results: Demographic data for both study groups did not differ significantly. Non-smokers and smokers had normal lung function indices. In smokers induced sputum contained statistically significantly ($p=0.026$) increased relative count of eosinophils 0.923 (0.355-1,753)% compared with non-smokers 0.069 (0.046 - 0.550)%. We also found significant reduction of absolute ($r=0.482$; $p=0.037$) and relative ($r=0.682$; $p=0.004$) count of bronchial epithelial cells in induced sputum that correlated to number of smoked pack-years. A trend towards statistical significance showed the correlation between smoked pack-years and the relative number of macrophages in induced sputum ($r =0.402$; $p=0.0872$). A trend towards statistical significance was also found in correlation between smoked pack-years and diminished FEV1% of predicted ($r= - 0.463$; $p= 0.046$).
Conclusion: In this study we showed that even smokers with short duration of the smoking habit have already initial signs of inflammation with eosinophil involvement.

4693
Cannabis use in patients with a primary spontaneous pneumothorax
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Introduction: It's Dutch policy to tolerate cannabis use. In literature active cannabis use is 10% amongst Dutch youth. The association between cannabis use and primary spontaneous pneumothorax (PSP) is unknown.
Aims: To determine the frequency of cannabis use in addition to tobacco smoking in patients with a PSP and to investigate the presence of underlying abnormalities on High Resolution CT (HRCT).
Methods: In a descriptive retrospective study patients were included who presented in a large Dutch teaching hospital with a PSP between august 2008 and august 2010. Because of an increased risk on secondary pneumothorax in older patients, only patients under 50 years were included. Age, gender, BMI, tobacco (T) and cannabis (C) use and (when available) HRCT data were recorded.
Results: In 2 years 53 patients presented with a PSP (42 male, 11 female, mean age 28 years, mean BMI 21). 74% (8% ex) smoked tobacco, 49% (8% ex) used cannabis (cannabis use unknown in 6%). The findings on HRCT are presented in Table 1.

Table 1. HRCT findings

	N (%) abnormal HRCT – N (%) blebs / N(%) bullae	N (%) normal HRCT	N (%) unknown
Total	30 (57) – 11 (37) / 19 (63)	10 (19)	13 (24)
C+T+ (no C without T)	15 (58) – 2 (13) / 13 (87)	3 (12)	8 (30)
C–T+	7 (70) – 3 (43) / 4 (57)	2 (20)	1 (10)
C–T–	6 (43) – 6 (100) / 0 (0)	5 (36)	3 (21)
C?T+	2 (67) – 0 (0) / 2 (100)	0 (0)	1 (33)

+ = use of, – = no use of, ? = unknown.

Conclusions: In patients under 50 years with PSP the use of cannabis was much higher than in the general population. However, all cannabis users also smoked tobacco. Only 12% of the cannabis users had a normal HRCT (30% unknown). On HRCT, bullae were present in 87% of cannabis users, in contrast to 57% in only tobacco smokers and none in nonsmokers.

4694
Cigarette smoke induces β2-integrin-dependent neutrophil migration across human umbilical vein endothelium
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Background: Cigarette smoking induces peripheral inflammatory responses in all smokers and is the major risk factor for neutrophilic lung diseases such as chronic obstructive pulmonary disease. The aim of this study was to investigate the effect of cigarette smoke on neutrophil chemotaxis and on β2-integrin activation and function in neutrophilic transmigration through endothelium.

Methods and results: Utilizing freshly isolated human neutrophils, the effect of cigarette smoke on chemotaxis and β2-integrin activation was studied. We demonstrate that cigarette smoke extract (CSE) dose dependently induced chemotaxis of neutrophils in vitro. Moreover, CSE promoted neutrophil adherence to fibrinogen. Using functional blocking antibodies against CD11b and CD18, it was shown that Mac-1 (CD11b/CD18) was responsible for the cigarette smoke-induced firm adhesion of neutrophils to fibrinogen. Furthermore, neutrophils transmigrated through endothelium by cigarette smoke due to the activation of β2-integrins, since pre-incubation of neutrophils with functional blocking antibodies against CD11b and CD18 attenuated this transmigration.
Conclusion: This is the first study to describe that cigarette smoke extract is a direct chemo-attractant for neutrophils and an activator of β2-integrins on the cell surface. Blocking this activation of β2-integrins might be an important target in cigarette smoke induced neutrophilic diseases.

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Waterpipe smoking and dependence are associated with chronic obstructive pulmonary disease: A case control study

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Waterpipe (WP) smoking is gaining in popularity in the Lebanese population. Although WP smokers are potentially exposed to the same noxious substances found in cigarettes, popular belief considers WP smoking harmless. Our objective was to evaluate the association between WP smoking, dependence and COPD.
We conducted a case-control study in two tertiary care hospitals. Cases were included if diagnosed as COPD by a chest physician and confirmed by a post-bronchodilator spirometry (FEV1/FVC<0.7); controls were included if free of any respiratory disease or symptom. After an oral informed consent, a standardized questionnaire was administered and spirometry results were collected by trained technicians.
211 COPD cases and 554 healthy controls were enrolled. In previous smokers, any type of smoking was associated with COPD: OR=28.3 (p<0.001) for cigarette smoking, OR=12.2 (p<0.001) for waterpipe smoking, and OR=41.9 (p<0.001) for mixed smoking. Lower odds ratios were found in current smokers: OR =19.6 (p<0.001) for cigarette smoking, OR=1.8 (p=0.299) for waterpipe smoking and OR=9.5 (p<0.001) for mixed smoking. However, assessing WP dependence by the validated LWDS-11 scale in current WP smokers, found an OR=15.0 (p=0.001) for the association between WP dependence and COPD. These results were confirmed by stratified and multivariate analysis, after adjustment for cigarette smoking and other potential confounding variables.
This is the first study that looked at the relation between COPD and WP smoking, and that showed a high risk of COPD in ex-smokers of WP. In current smokers of WP, dependent individuals have an increasing risk of COPD, as much as cigarette smokers.

4696

The mother's smoking during pregnancy influences on endothelial dysfunction in newborns

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Smoking during pregnancy is a risk for dangerous consequences. Smoking causes increased chances for miscarriage, growth restriction, preterm birth, and health problems in the future.
The aim of the investigation was to reveal effect of mother's tobacco smoking during pregnancy on endothelial dysfunction in newborn.
127 children born from mothers - smokers were examined. The control group was consisted of 32 healthy babies born from never smoking parents. A level of thiocyanate (a marker of passive smoking) was determined in a blood serum by spectrophotometry method. Levels of S-nitrosothiols and endotheline-1 were determined in blood serum by spectrophotometry and enzyme immunoassay methods respectively for the estimation of the endothelium dysfunction.
The levels of thiocyanate were 8,64±0,52 mg/l (high level – I group) and 3,75±0,21mg/l (low level – II group) in serum of babies born from mothers – smokers. The level of thiocyanate was 1,03±0,07mg/l in control group. The levels of S-nitrosothiols were 2,37±0,16 fmol/ml in I group (P1<0,001; P2<0,001) 1,29±0,11fmol/ml in II group (P2<0,001); 0,45±0,02fmol/ml in control. The levels of S-nitrosothiols were 0,18±0,01mmol/ml (P1<0,001; P2<0,001); 0,35±0,02 mmol/ml (P2<0,02), 0,53±0,02 mmol/ml in newborns of I, II and control groups respectively. P.S.: P1- vs. II group; P2- vs. control.
The tobacco derivatives came through placenta from mother-smoker to fetus. The endothelial dysfunctions are characterised by the decreasing of vasodilatator S-nitrosothiols and increasing of vasoconstrictor endotheline-1 level in newborn depended from level of thiocyanate.

4697

Smoking ages your lungs – Results from the COLD cohort

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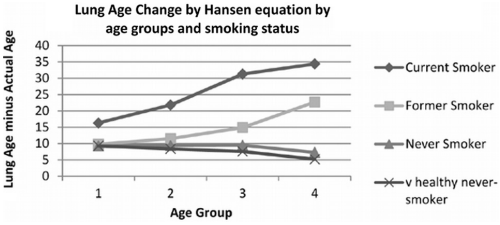
Background: Calculated Lung Age could be used as motivation to quit smoking. It is unknown whether smoking cessation results in improvement of Lung Age.
Hypothesis: We hypothesize that smoking cessation can reduce lung age.
Aim: To determine the lung age for current smokers, ex smokers and life long non-smokers using different lung age equations.
Methods: We calculated the change in Lung age by 4 different Lung Age equations: Morris [1985], Hansen [2010] Newbury [2010] and the COLD equation [2011]. We utilized the respiratory questionnaire data and pre and post bronchodilator spirometric data from 3,042 people aged 40 years and older in the Canadian Obstructive Lung Disease (COLD) study. Four groups of subjects were evaluated: current smokers, ex smokers, lifelong nonsmokers and a subgroup of lifelong nonsmokers who never had respiratory diseases or symptoms.
Results: The figure shows Lung age change by smoking status and by 4 age groups: 40-49 years; 50-59 years; 60-69 years; 70 years and older. The results from this cross sectional analysis from all 4 equations were consistent, and showed that active smoking is associated with faster aging of the lungs than life long non-smokers and "quitters"; lifelong non-smokers maintain normal lung age as they grow older; and active smoking is associated with greater lung aging than in "quitters", for the same tobacco load.



Conclusions: Smoking is associated with accelerated lung aging.

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The impact of smoke-free legislation on smoking-related emergency admissions in Istanbul

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Introduction: Reductions in exposure to tobacco smoke was shown to attenuate the risk of exacerbations of chronic respiratory and cardiac conditions both in adults and children. The aim of this study was to compare the changes in emergency department (ED) admissions for smoking-related diseases before and after the implementataion of smoking ban in Istanbul.
Methods: Admissions to ten major hospitals in Istanbul in the first five months of 2009 and 2010 were evaluated and compared, using International Classification of Diseases, (ICD-10) diagnostic codes.
Results: In 2009, there were 115030 ED admissions for the associated diagnostic codes, whereas this decreased to 87212 in 2010. There was a 16% decrease in acute nasopharyngitis, 32.9% decrease in pneumonia, 18.8% decrease in acute broncitis, 59,2% decrease in allergic rhinitis, 61.3% decrease in lower respiratory tract diseases, 21.4% decrease in chronic obstructive lung disease, 20,5% decrease in asthma, 33.6% decrease in ischemic heart disease and acute myocardial infarction. All differences were found statistically significant. Cost saving of emergency drugs and services on site were 437,104 euros for 10 hospitals in a 5 month period, which is projected to be 3,147,148 euros for Istanbul annually, without calculating the prevented hospital treatment cost.
Conclusion: ER admission rates for diseases associated with active and passive smoking were reduced by 24.2% as a result of smoking ban in Istanbul. Positive effects of clear indoor air ordinances are observed in a very short period, and therefore respiratory health professionals should be advocates for this policies against all odds.