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Smoking & Tobacco Abstracts & News

**STAN Bulletin
41st Edition
15-October-2012**

Editor's note: The *Waikato Times* [op-ed](#) by Joshua Drummond does a fine job eviscerating the BAT New Zealand [Agree/Disagree](#) campaign. And the *Sydney Morning Herald* [quote](#) below from Anne Jones, chief executive, ASH Australia, on the obscene billboard campaign by unclear-on-the-concept Christian churches' billboard campaign, pretty much says all that need be said.

Stan Shatenstein

Noteworthy:

"I think it's better to be alive and deal with the religious issues rather than be dead and not be able to deal with anything." [Anne Jones, chief executive, ASH Australia, Blessed are the smokers for they are welcome in heaven, [Sydney Morning Herald](#)]

"A variety of factors are limiting the acceptability of products currently available – minimum safety information, price, accessibility, lack of knowledge in health and other professionals, an ethical stance against the use of nicotine by some advocates and no clear strategic direction from government or regulators. Modelling studies based on the use of snus in Australia (where there is currently a similar ban to the UK) have shown that switching to snus will result in individual and population benefits if enough inveterate smokers do this (Gartner et al, 2007). Combined with cessation and initiation approaches, harm reduction, under appropriate regulatory oversight, has the capacity to have a significant and immediate impact on reducing the death and disease currently caused by tobacco use." [McNeill A, Munafò MR. Reducing harm from tobacco use, [J Psychopharm](#)]

In the News:

- Australia: [Christian billboards employ mock graphic cigarette pack health warnings](#)
- Australia: [Cigarette prices to rise on navy ships. 1901 Excise Act concession to end](#)
- EU: [E-cigarettes could be banned according to leaked Directives draft: UNITAB warning](#)
- France: [ECTRIMS: Excess Mortality in Multiple Sclerosis Largely From Smoking \[Programme\]](#)
- Japan/Zambia: [JTI signs contract with 7000 farmers to supply raw tobacco](#)
- NZ: [Waikato Times: Opinion: Fighting for the right to brand poisonous products \[BAT: Agree/Disagree\]](#)
- NZ: [U. Otago: Response to BAT criticism of new plain packaging research \[ANZJPH: Hoek\]](#)
- NZ: [Mental Health Foundation supports plain packaging of tobacco products](#)
- US: MO: [Smoking tax opponents wary of Prop B, argue funds could be redirected](#)
- US: NY: [Albany Times-Union: Editorial: RYO: The right way to tax loose tobacco](#)
- US: VA: [PM/Altria: City Council members say no 'giff' given in cigarette sting](#)

In this Edition:

- Am J Orthopsych - Balsam: US: WA: Understanding sexual orientation health disparities in smoking
- AJPH - Lee: US: IOM: Up in Smoke: Sexual & Gender Minority Health: Vanishing Evidence of Tobacco Disparities
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- ANZJPH - Gartner: Australia: Analysis of national data shows mixed evidence of hardening among smokers
- ANZJPH - Marsh: NZ: Access to cigarettes by young smokers: little change from 2000 to 2008
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- Braz Oral Res - Lima: Brazil: Oral leukoplakia manifests differently in smokers & non-smokers
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- Fertil Steril - Pier: US: Cigarette smoking effect on human oviductal ciliation & ciliogenesis
- Front Pharmacol - Durazzo: Smoking, BDNF polymorphisms, COMT, neurocognition & alcohol abstinence
- Health Psych - Baldwin: US: Self-Generated Health Arguments for Exercise & Smoking Cessation
- Hum Reprod - Ernst: Denmark: Maternal smoking during pregnancy & daughters' reproductive health
- Int J Cancer - Psaltopoulou: Smoking & multiple myeloma risk: Meta-analysis of 40 observational studies
- Invest Ophthalmol Vis Sci - Ye: Smoking & Age-Related Cataract Risk: Meta-Analysis
- J Bone Joint Surg Br - Chassanidis: Greece: Smoking & mRNA expression of periosteum morphogenetic proteins
- JEADV - Emre: Turkey: Oxidative stress, smoking & clinical severity of psoriasis
- J Hazard Mater - Wang: Taiwan: Arsenic, smoking, VEGF risk genotypes, UUTUC & bladder cancer
- J Psychopharm - McNeill: UK: Perspectives: Reducing harm from tobacco use
- JSAD - Kushner: US: NESARC: Nicotine Dependence Risk Elevation per Pack Smoked & Anxiety Disorder
- Lancet - Dowell: US: New York City: Viewpoint: Prevention of non-communicable diseases
- Mutat Res - Cavallo: Filter & non-filter cigarette cyto-genotoxic effects on bronchial & pulmonary cells
- N&TR - Cui: Alpha Oscillations in Response to Affective & Cigarette-Related Stimuli in Smokers
- Schizophr Res - Moran: US: Insular & anterior cingulate circuits in smokers with schizophrenia
- Urology - Taha: Egypt: Smoking effect on fertile men's sperm vitality, DNA integrity, seminal oxidative stress & zinc

Abstracts:

Understanding sexual orientation health disparities in smoking: a population-based analysis

[Am J Orthopsychiatry](#). 2012 Oct;82(4):482-93. doi: 10.1111/j.1939-0025.2012.01186.x.

[Balsam KF](#), [Beadnell B](#), [Riggs KR](#).

Abstract

Lesbian, gay, and bisexual populations are at elevated risk for tobacco use compared to their heterosexual peers. However, there is little research examining reasons for this disparity. Drawing on prior literature regarding psychosocial variables associated with both sexual orientation and smoking, the authors tested a path model of risk and protective factors to help explain sexual orientation differences in smoking using data from the Washington State Behavioral Risk Factor Surveillance System from 2003 to 2007. The authors estimated separate models for men and women, comparing lesbians or gays and bisexuals to heterosexuals. Results indicated that the explanatory variables accounted for most of the variance in this relationship, with both risk-enhancing and risk-reducing pathways. Mental health, life dissatisfaction, alcohol use, exposure to tobacco marketing, and single relationship status were risk enhancers for most LGB participants. Health-care access and income level were risk enhancers for bisexual participants only. Neither emotional support nor attitudes and knowledge about tobacco use helped explain the relationship between sexual orientation and smoking. These findings have significant implications for tobacco prevention and control efforts in this high-risk population.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1939-0025.2012.01186.x/abstract>

Up in Smoke: Vanishing Evidence of Tobacco Disparities in the Institute of Medicine's Report on Sexual and Gender Minority Health

American Journal of Public Health: November 2012, Vol. 102, No. 11, pp. 2041-2043.

Joseph G. L. Lee, John R. Blosnich, and Cathy L. Melvin

Abstract

The Institute of Medicine (IOM) released a groundbreaking report on lesbian, gay, bisexual, and transgender (LGBT) health in 2011, finding limited evidence of tobacco disparities. We examined IOM search terms and used 2 systematic reviews to identify 71 articles on LGBT tobacco use. The IOM omitted standard tobacco-related search terms. The report also omitted references to studies on LGBT tobacco use (n = 56), some with rigorous designs. The IOM report may underestimate LGBT tobacco use compared with general population use.

<http://ajph.aphapublications.org/doi/abs/10.2105/AJPH.2012.300746>

Attitudes Toward Nonsmoking Policies and Tobacco Tax Increases: A Cross-sectional Study Among Vietnamese Adults

[Asia Pac J Public Health](#). 2012 Oct 2. [Epub ahead of print]

[Dao TM](#), [Hoang VM](#), [Le TH](#), [Kim BG](#), [Le TT](#), [Pham TQ](#), [Hsia J](#).

Abstract

Following the 2009 update of the 2005 Framework Convention on Tobacco Control, Vietnam issued a new policy to ban smoking at workplaces and public places. This cross-sectional survey explored public attitudes toward this new regulation and provides evidence to inform future laws. Using stratified cluster sampling, 10 383 Vietnamese people older than 15 years were drawn from 11 142 selected households. Policies mandating "no smoking at workplaces" were supported by 88.7% of Vietnamese adults, whereas "no smoking in public places" and "increasing the tobacco tax" received less support. Educational level, knowledge of health effects, access to information on quitting and smoking health risks, smoking status, ethnicity, and region had significant associations with positive attitudes toward all 3 tobacco control policies. Adults belonging to the non-Kinh ethnic group, those who do not live in the Red river delta, people with lower educational levels, and current smokers should be targeted in tobacco control communication programs.

<http://aph.sagepub.com/content/early/2012/09/24/1010539512460568.abstract>

Analysis of national data shows mixed evidence of hardening among Australian smokers

[Aust N Z J Public Health](#). 2012 Oct;36(5):408-14. doi: 10.1111/j.1753-6405.2012.00908.x

[Gartner C](#), [Scollo M](#), [Marquart L](#), [Mathews R](#), [Hall W](#).

Abstract

Objective : According to the 'hardening hypothesis', the proportion of smokers that are 'low-probability quitters' will increase as societal disapproval of smoking increases. This paper examines whether there has been increased hardening in Australian smokers over the past decade as reflected in an increased prevalence of psychological distress and social disadvantage among current smokers. Methods: The relationship between psychological distress, living in a disadvantaged area and level of education was determined using logistic regression at two time points 7 to 10 years apart in three cross-sectional household survey series: National Drug Strategy Household Survey (NDSHS), National Health Survey (NHS) and National Survey of Mental Health and Well-being (NSMHW). Results: The relationships between smoking and living in the most disadvantaged areas and having completed less than 12 years of schooling strengthened between 2001 and 2010 in the NDSHS, but there were no significant changes between survey years in the NHS and NSMHW. There was no significant change in the relationship between smoking and psychological distress between survey years in any of the survey series. Conclusion: Social disadvantage may be increasing among current smokers, but the results were inconsistent between survey series, presenting weak evidence that the population of Australian smokers hardened as smoking prevalence declined by approximately 4% over the last decade. Implications: A greater focus on intensive individual-level tobacco cessation interventions does not appear warranted at this time.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1753-6405.2012.00908.x/abstract>

ANZJPH Editorial & related Research Commentary:

The plain facts about tobacco's future

<http://onlinelibrary.wiley.com/doi/10.1111/j.1753-6405.2012.00905.x/abstract>

Strong public support for plain packaging of tobacco products

<http://onlinelibrary.wiley.com/doi/10.1111/j.1753-6405.2012.00907.x/abstract>

Access to cigarettes by young New Zealand smokers: little change from 2000 to 2008

[Aust N Z J Public Health](#). 2012 Oct;36(5):415-20. doi: 10.1111/j.1753-6405.2012.00909.x

[Marsh L](#), [Gray A](#), [McGee R](#), [Newcombe R](#), [Patterson R](#).

Abstract

Objective: To examine trends in young New Zealanders' access to cigarettes from 2002 to 2008. Methods: Self-reports of

young smokers' commercial sources of cigarettes, requests for proof of age and being refused a sale, and the amount spent on cigarettes, were analysed from the 2002 and 2004 Youth Lifestyles Surveys and 2006 and 2008 Year 10 In-depth Surveys of 14 to 15 year olds in New Zealand (NZ). Results: Most young smokers obtain cigarettes through non-commercial sources; however, one-third bought their cigarettes from shops in 2008, with no evidence of a change in proportions purchasing from major retail outlets since 2000. Few young smokers were asked to show proof of age when purchasing cigarettes, with no evidence over time of more young smokers being asked for proof of age, but reports of being refused the sale of cigarettes decreased from 2000. Participants reporting being asked for proof of age had nearly six times the odds of being refused sale. The amount spent on cigarettes did not change over the study period.

Conclusion: Many young smokers continue to purchase cigarettes from commercial outlets, with no evidence of change in purchasing from major outlets such as dairies, service stations, supermarkets or liquor stores since 2000. The fact that few young smokers reported being asked to show proof of age suggests the need for stronger measures to ensure that retailers comply with New Zealand legislation regarding under-age sales of tobacco. Implications: With an infringement notice scheme being implemented and tobacco price rises, there may be an increase in accessing cigarettes through social sources. It remains important to monitor commercial tobacco sales to young people to determine the efficacy of policy change.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1753-6405.2012.00909.x/abstract>

Also:

A refreshing poison: one-quarter of young New Zealand smokers choose menthol

<http://onlinelibrary.wiley.com/doi/10.1111/j.1753-6405.2012.00926.x/abstract>

Exposure to second-hand smoke and direct healthcare costs in children -- results from two German birth cohorts, GINIplus and LISAplus

[BMC Health Serv Res](#), 2012 Oct 2;12(1):344. [Epub ahead of print]

[Batscheider A](#), [Zakrzewska S](#), [Heinrich J](#), [Wenig CM](#), [Menn P](#), [Bauer CP](#), [Hoffmann U](#), [Koletzko S](#), [Lehmann I](#), [Herbarth O](#), [von Berg A](#), [Berdel D](#), [Krämer U](#), [Schaaf B](#), [Wichmann HE](#), [Leidl R](#).

Abstract

BACKGROUND: Although the negative health consequences of the exposure to second hand tobacco smoke during childhood are already known, evidence on the economic consequences is still rare. The aim of this study was to estimate excess healthcare costs of exposure to tobacco smoke in German children.

METHODS:

The study is based on data from two birth cohort studies of 3,518 children aged 9-11 years with information on healthcare utilisation and tobacco smoke exposure: the GINIplus study (German Infant Study On The Influence Of Nutrition Intervention Plus Environmental And Genetic Influences On Allergy Development) and the LISAplus study (Influence of Life-Style Factors On The Development Of The Immune System And Allergies In East And West Germany Plus The Influence Of Traffic Emissions And Genetics). Direct medical costs were estimated using a bottom-up approach (base year 2007). We investigated the impact of tobacco smoke exposure in different environments on the main components of direct healthcare costs using descriptive analysis and a multivariate two-step regression analysis.

RESULTS:

Descriptive analysis showed that average annual medical costs (physician visits, physical therapy and hospital treatment) were considerably higher for children exposed to second-hand tobacco smoke at home (indoors or on patio/balcony) compared with those who were not exposed. Regression analysis confirmed these descriptive trends: the odds of positive costs and the amount of total costs are significantly elevated for children exposed to tobacco smoke at home after adjusting for confounding variables. Combining the two steps of the regression model shows smoking attributable total costs per child exposed at home of [euro sign]87 [10--165] (patio/balcony) and [euro sign]144 [6--305] (indoors) compared to those with no exposure. Children not exposed at home but in other places showed only a small, but not significant, difference in total costs compared to those with no exposure.

CONCLUSIONS:

This study shows adverse economic consequences of second-hand smoke in children depending on proximity of exposure. Tobacco smoke exposure seems to affect healthcare utilisation in children who are not only exposed to smoke

indoors but also if parents reported exclusively smoking on patio or balcony. Preventing children from exposure to second-hand tobacco smoke might thus be desirable not only from a health but also from an economic perspective.

<http://www.biomedcentral.com/1472-6963/12/344/abstract>

<http://www.biomedcentral.com/content/pdf/1472-6963-12-344.pdf>

Note: Open Access. Full text PDF freely available from link immediately above.

Oral leukoplakia manifests differently in smokers and non-smokers

[Braz Oral Res.](#) 2012 Sep 27. pii: S1806-83242012005000024. [Epub ahead of print]

[Lima JS](#), [Pinto Jr DD](#), [Sousa SO](#), [Corrêa L](#).

Abstract

Oral leukoplakias (OL) are potentially malignant lesions that are typically white in color. Smoking is considered a risk factor for developing OL, and dysplastic lesions are more prone to malignant transformation. The aim of this study was to describe the clinical features observed in dysplastic and non-dysplastic OL in both smokers and non-smokers. A total of 315 cases of OL were retrieved and separated into either dysplastic or non-dysplastic lesions, and these cases were further categorized as originating in either smokers or non-smokers. Frequencies of the type of OL lesion, with respect to whether the patients smoked, were established. The results demonstrated that 131 cases of OL were dysplastic (74 smokers and 57 non-smokers), and 184 were non-dysplastic (96 smokers and 88 non-smokers). For OL cases in smokers for which information about alcohol consumption was also available (84 cases), the results revealed no significant difference in the amount of dysplastic and non-dysplastic lesions. Dysplastic lesions were more frequent in male smokers and in non-smoking females. The median age of smokers with cases of OL was significantly lower than in non-smokers; the lowest median ages were observed for female smokers with dysplastic OL. The most frequent anatomical sites of dysplastic lesions were the floor of the mouth in smokers and the tongue in non-smokers. Dysplastic lesions in smokers were significantly smaller than non-dysplastic lesions in non-smokers. Being a male smoker, being female, being younger, and having smaller lesions were associated with dysplastic features in OL. These clinical data may be important for predicting OL malignant transformation.

http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1806-83242012005000024&lng=en&nrm=iso&tlng=en

<http://www.scielo.br/pdf/bor/2012nahead/pint974-aop.pdf>

Note: Open Access. Full text PDF freely available from link immediately above.

A rare case of gamma knife-induced smoking cessation in a patient with a vestibular schwannoma

[Br J Neurosurg.](#) 2012 Sep 28. [Epub ahead of print]

[Ramakrishna R](#), [Rostomily R](#), [Rockhill J](#).

Abstract

Objective. We report a rare case of a patient with a vestibular schwannoma who underwent gamma knife irradiation and subsequently lost unilateral taste sensation. As a result, the patient ceased smoking.

<http://informahealthcare.com/doi/abs/10.3109/02688697.2012.725873>

Simultaneous measurement of urinary total nicotine and cotinine as biomarkers of active and passive smoking among Japanese individuals

[Environ Health Prev Med.](#) 2012 Sep 26. [Epub ahead of print]

[Matsumoto A](#), [Matsumoto A](#), [Ichiba M](#), [Payton NM](#), [Oishi H](#), [Hara M](#).

Abstract

OBJECTIVES:

Measuring urinary cotinine is a popular and established method of biologically monitoring exposure to tobacco smoke. However, the lower detection limit of cotinine often impedes the evaluation of passive (second-hand) smoking and this, together with unconverted nicotine, does not reflect actual levels of exposure. Furthermore, a portion of the Japanese population might have decreased ability to metabolize nicotine. The present study was therefore carried out to validate the simultaneous analysis of total concentrations of free nicotine and cotinine and their glucuronides to determine actual levels of voluntary and involuntary exposure to cigarette smoke.

METHODS:

Urine samples from 118 Japanese smokers and 117 non-smokers were analyzed using gas chromatography-mass spectrometry. Voluntary and involuntary smoking status was self-reported and workplace smoking restrictions were objectively evaluated.

RESULTS:

The integrated sum of all concentrations showed 2.2- and 2.4-fold higher total levels (free and glucuronide) of nicotine and cotinine relative to the free levels. Median (quartiles) of total nicotine and cotinine were 1635 (2222) and 3948 (3512) ng/mL in smokers, and 3.5 (5.3) and 2.8 (4.2) ng/mL in non-smokers. Concentrations of urinary nicotine were higher than those of cotinine in 21 % of smokers and in 54 % of non-smokers. Nicotine and cotinine levels were significantly associated with a smoking habit, as well as being significantly associated with the workplace and home environments of non-smokers.

CONCLUSIONS:

The present method can monitor voluntary and involuntary exposure to tobacco smoke. Measuring total urinary nicotine levels might be useful for analyzing exposure to cigarette smoke among non-smokers.

<http://www.springerlink.com/content/cv8l4282354770g4/>

Effect of cigarette smoking on human oviductal ciliation and ciliogenesis

Fertil Steril. 2012 Sep 22. pii: S0015-0282(12)02129-2. doi: 10.1016/j.fertnstert.2012.08.041. [Epub ahead of print]

[Pier B](#), [Kazanjian A](#), [Gillette L](#), [Strenge K](#), [Burney RO](#).

Abstract**OBJECTIVE:**

To investigate the effect of cigarette smoke exposure on ciliation and ciliogenesis in human oviductal epithelium.

DESIGN:

Molecular analysis using human tubal segments.

SETTING:

Academic medical center.

PATIENT(S):

Twenty women undergoing elective tubal sterilization procedure.

INTERVENTION(S):

Expression of ciliated cell-specific markers was compared in tubal segments from smokers and nonsmokers using quantitative immunohistochemistry and Western blot analysis. The expression of transcription factors in the motile ciliogenesis program was compared using quantitative polymerase chain reaction and quantitative immunohistochemistry.

MAIN OUTCOME MEASURE(S):

Oviductal ciliation and expression of transcription factors involved in ciliogenesis.

RESULT(S):

No significant differences were detected in density of ciliation between groups. Neither number of years of smoking nor pack-year history correlated with density of ciliation. Expression of ciliogenic transcription factors FOXJ1, RFX2, and RFX3 was consistent between groups.

CONCLUSION(S):

Few studies have evaluated the relationship between smoking and ciliated epithelium in human oviducts. Cigarette smoking does not seem to result in quantitative differences in the density of ciliation nor expression of ciliogenesis factors. Our findings suggest that pathophysiologic mechanisms other than ciliation account for the increased risk of ectopic pregnancy in women who smoke.

<http://www.fertstert.org/article/S0015-0282%2812%2902129-2/abstract>
<http://www.sciencedirect.com/science/article/pii/S0015028212021292>

Associations of cigarette smoking and polymorphisms in brain-derived neurotrophic factor and catechol-O-methyltransferase with neurocognition in alcohol dependent individuals during early abstinence

Front. Pharmacol., 11 October 2012 | doi: 10.3389/fphar.2012.00178

Timothy C. Durazzo, Kent E. Hutchison, Susanna L. Fryer, Anderson Mon and Dieter J. Meyerhoff

Abstract

Chronic cigarette smoking and polymorphisms in brain-derived neurotrophic factor (BDNF) and catechol-O-methyltransferase (COMT) are associated with neurocognition in normal controls and those with various neuropsychiatric conditions. The influence of BDNF and COMT on neurocognition in alcohol dependence is unclear. The primary goal of this report was to investigate the associations of single nucleotide polymorphisms (SNPs) in BDNF Val66Met (rs6265) and COMT Val158Met (rs4680) with neurocognition in a treatment-seeking alcohol dependent cohort and determine if neurocognitive differences between non-smokers and smokers previously observed in this cohort persist when controlled for these functional SNPs. Genotyping was conducted on 70 primarily male treatment-seeking alcohol dependent participants (ALC) who completed a comprehensive neuropsychological battery after 33 ± 9 days of monitored abstinence. After controlling for COMT and BDNF genotypes, smoking ALC performed significantly worse than non-smoking ALC on the domains of auditory-verbal and visuospatial learning and memory, cognitive efficiency, general intelligence, processing speed, and global neurocognition. In smoking ALC, greater number of years of smoking over lifetime was related to poorer performance on multiple domains after controlling for genotypes and alcohol consumption. In addition, COMT Met homozygotes were superior to Val homozygotes on measures of executive skills and showed trends for higher general intelligence and visuospatial skills, while COMT Val/Met heterozygotes showed significantly better general intelligence than Val homozygotes. COMT Val homozygotes performed better than heterozygotes on auditory-verbal memory. BDNF genotype was not related to any neurocognitive domain. The findings are consistent with studies in normal controls and neuropsychiatric cohorts that reported COMT Met carriers demonstrated better performance on measures of executive skills and general intelligence. Results also indicated that the poorer performance of smoking compared to non-smoking ALC across multiple neurocognitive domains was not mediated by COMT or BDNF genotype. Overall, the findings lend support to the expanding clinical movement to make smoking cessation programs available to smokers at the inception of treatment for alcohol/substance use disorders.

<http://www.frontiersin.org/neuropharmacology/10.3389/fphar.2012.00178/abstract>

Note: Open Access. Full text PDF freely available from link immediately above.

Related PR:

Veterans are at higher risk of alcohol abuse relapse due to smoking
http://www.eurekalert.org/pub_releases/2012-10/f-vaa101212.php

Examining Causal Components and a Mediating Process Underlying Self-Generated Health Arguments for Exercise and Smoking Cessation

[Health Psychol.](#) 2012 Oct 1. [Epub ahead of print]

[Baldwin AS](#), [Rothman AJ](#), [Vander Weg MW](#), [Christensen AJ](#).

Abstract

Objective: Self-persuasion-generating one's own arguments for engaging in a specific behavior-can be an effective strategy to promote health behavior change, yet the causal processes that explain why it is effective are not well-specified. We sought to elucidate specific causal components and a mediating process of self-persuasion in two health behavior domains: physical activity and smoking. Methods: In two experiments, participants were randomized to write or read arguments about regular exercise (Study 1: N = 76; college students) or smoking cessation (Study 2: N = 107; daily smokers). In Study 2, we also manipulated the argument content (matched vs. mismatched participants' own concerns about smoking) to isolate its effect from the effect of argument source (self vs. other). Study outcomes included participants' reports of argument ratings, attitudes, behavioral intentions (Studies 1 & 2), and cessation attempts at 1 month (Study 2). Results: In Study 1, self-generated arguments about exercise were evaluated more positively than other arguments ($p = .01$, $d = .63$), and this biased processing mediated the self-generated argument effect on attitudes toward exercise ($\beta = .08$, 95% CI = .01, .18). In Study 2, the findings suggested that biased processing occurs because self-generated argument content matches people's own health concerns and not because of the argument source (self vs. other). In addition, self-generated arguments indirectly led to greater behavior change intentions (Studies 1 & 2) and a greater likelihood of a smoking cessation attempt (Study 2). Conclusions: The findings elucidate a causal component and a mediating process that explain why self-persuasion and related behavior change interventions, such as motivational interviewing, are effective. Findings also suggest that self-generated arguments may be an efficient way to deliver message interventions aimed at changing health behaviors.

<http://psycnet.apa.org/psycinfo/2012-26679-001/>

Maternal smoking during pregnancy and reproductive health of daughters: a follow-up study spanning two decades

[Hum Reprod.](#) 2012 Oct 2. [Epub ahead of print]

[Ernst A](#), [Kristensen SL](#), [Toft G](#), [Thulstrup AM](#), [Håkonsen LB](#), [Olsen SF](#), [Ramlau-Hansen CH](#).

Abstract

STUDY QUESTION:

Does in utero exposure to constituents of cigarette smoke have a programming effect on daughters' age of menarche and markers of long-term reproductive health?

SUMMARY ANSWER:

In utero exposure to constituents of cigarette smoke was associated with earlier age of menarche and to a lesser extent changes in the testosterone profile of the young women.

WHAT IS KNOWN ALREADY:

Studies observe potential effects of in utero exposure to constituents of cigarette smoke on the intrauterine formation of female gonads, but the consequences on long-term reproductive health in daughters remain unclear.

STUDY DESIGN, SIZE AND DURATION:

A prospective cohort study was designed using data from 965 pregnant women enrolled prior to a routine 30th-week antenatal examination at a midwifery practice in Denmark from 1988 to 1989 and a follow-up of their 19-21-year-old daughters in 2008.

PARTICIPANTS/MATERIALS, SETTING AND METHODS:

The pregnant women provided information on lifestyle factors during pregnancy, including the exact number of cigarettes smoked per day during the first and the second trimesters. A total of 438 eligible daughters were asked to complete a web-based questionnaire on reproductive health and subsequently invited to participate in a clinical examination during 2008. Of the 367 daughters (84%) who answered the questionnaire, 267 (61%) agreed to further examination. Information on menstrual pattern was provided at examination, blood samples were drawn to be analyzed for serum levels of reproductive hormones [FSH, LH, estradiol (E(2)), sex hormone-binding globulin, anti-Müllerian hormone, dehydroepiandrosterone-sulphate (DHEAS), free testosterone and free E(2)] and number of follicles (2-9 mm) were examined by transvaginal ultrasound. The daughters were divided into three exposure groups according to the level of maternal smoking during first trimester [non-exposed (reference), low-exposed (mother smoking >0-9 cigarettes/day) and high-exposed (mother smoking \geq 10 cigarettes/day)]. Data were analyzed by multiple regression analyses in which we adjusted for potential confounders. Both crude and adjusted test for trend were carried out using maternal smoking during the first trimester as a continuous variable.

MAIN RESULTS AND THE ROLE OF CHANCE:

We observed an inverse association between in utero exposure to constituents of cigarette smoke and age of menarche ($P = 0.001$). Daughters exposed to >0-9 cigarettes/day debuted with -2.7 [95% confidence interval (CI) -5.2 to -0.1] percentage earlier age of menarche, whereas daughters exposed to \geq 10 cigarettes/day had -4.1 (95% CI: -6.6 to -1.5) percentage earlier age of menarche corresponding to 6.5 (95% CI: -10.7 to -2.2) months. There was a non-significant tendency towards lower levels of testosterone and DHEAS with increasing in utero exposure to constituents of cigarette smoke but no associations with follicle number, cycle length or serum levels of the other reproductive hormones were observed.

LIMITATIONS AND REASONS FOR CAUTION:

We collected information on age of menarche retrospectively but the recall time was relatively short (2-10 years) and the reported values were within the normal range of Caucasians. Analyses of reproductive hormones are presented only for the group of daughters who were non-users of hormonal contraceptives because users were excluded, leaving only a low number of daughters available for the analyses ($n = 75$), as reflected in the wide CIs. The analyses of hormones were further adjusted for menstrual phase at time of clinical examination (follicular, ovulation and luteal phase) because blood samples were not collected on a specific day of the menstrual cycle.

WIDER IMPLICATIONS OF THE FINDINGS:

This study supports the limited evidence of an inverse association between maternal smoking during pregnancy and age of menarche and further addresses to what extent reproductive capacity and hormones may be programmed by maternal smoking during pregnancy. A trend toward earlier maturation of females is suggested to have implications on long-term reproductive function.

<http://humrep.oxfordjournals.org/content/early/2012/10/02/humrep.des337.abstract>

Tobacco smoking and risk of multiple myeloma: A meta-analysis of 40 observational studies

International Journal of Cancer

[Accepted Article. These manuscripts have been accepted, but have not been edited or formatted. They will be published at a future date.](#)

Accepted manuscript online: **10 OCT 2012**

Theodora Psaltopoulou, Theodoros N. Sergentanis, Nick Kanellias, Prodromos Kanavidis, Evangelos Terpos and Meletios A. Dimopoulos

Abstract

This meta-analysis aims to quantitatively synthesize all available data on the association between tobacco smoking and multiple myeloma (MM) risk. Eligible studies were identified and pooled effect estimates (odds ratios and relative risks) were calculated regarding ever, current and former smoking. Separate analyses were performed on case-control and cohort studies, as well as on males and females. Meta-regression analysis with percentage of males, mean age, years of smoking, pack-years, cigarettes per day, years since quit and age at onset was performed. 40 articles were deemed eligible; of them 27 used a case-control design (4,625 cases and 21,591 controls) and 13 used a cohort design (2,228 incident cases among a total cohort size equal to 1,852,763 subjects). Ever smoking was not associated with MM risk (pooled effect estimate= 0.92, 95%CI: 0.85-1.00); similar results were obtained for current (pooled effect estimate= 0.87, 95%CI: 0.74-1.03) and former smoking (pooled effect estimate= 1.04, 95%CI: 0.96-1.13). Regarding ever smoking, the null association was reproducible upon cohort studies (pooled effect estimate= 1.01, 95%CI: 0.89-1.15), whereas the inverse

association in case-control studies (pooled effect estimate= 0.87, 95%CI: 0.78-0.96) was particularly due to the bias-prone hospital-based ones. Meta-regression analysis did not yield statistically significant results. In conclusion, MM does not seem to be associated with tobacco smoking. There is a need to further explore how molecular mechanisms are involved in the resistance of multiple myeloma progenitor cells towards smoking.

<http://onlinelibrary.wiley.com/doi/10.1002/ijc.27898/abstract>

Smoking and Risk of Age-Related Cataract: A Meta-Analysis

Invest. Ophthalmol. Vis. Sci. June 25, 2012 vol. 53 no. 7 3885-3895

Published online before print May 17, 2012, doi: 10.1167/iovs.12-9820

[Juan Ye](#), [Jinjing He](#), [Changjun Wang](#), [Han Wu](#), [Xin Shi](#), [Huina Zhang](#), [Jiajun Xie](#) and [Sang Yeul Lee](#)

Abstract

Purpose. We conducted a meta-analysis to evaluate the relationship between smoking and age-related cataract (ARC).

Methods. Eligible studies were identified via computer searches and reviewing the reference lists of the key articles. The summary relative risk ratio (RR) or odds ratio (OR) and 95% confidence interval (CI) were calculated. Study-specific risk estimates were pooled using a random-effects model. Meta-regression to assess heterogeneity by several covariates and subgroup analysis on ARC types were performed.

Results. A total of 13 prospective cohort and eight case-control studies met our inclusion criteria. Ever smoking was statistically significantly associated with increased risk of ARC among cohort studies (OR 1.41, 95% CI 1.23–1.62) and case-control studies (OR 1.57, 95% CI 1.20–2.07). In subgroup analysis, ever smoking exhibited a positive relationship with nuclear cataract (NC; OR 1.66, 95% CI 1.46–1.89) and a marginally significant relationship with posterior subcapsular cataract (OR 1.43, 95% CI 0.99–2.07) in cohort studies. Similar results were found in case-control studies (NC OR 1.86, 95% CI 1.47–2.36; posterior subcapsular cataract OR 1.60, 95% CI 0.97–2.65). Current smokers were at higher risk of ARC than past smokers. No association between smoking and cortical cataract was observed.

Conclusions. The overall current literature suggests that smoking was associated with increased risk of ARC, especially NC. Further efforts should be made to confirm these findings and clarify the underlying biological mechanisms.

<http://www.iovs.org/content/53/7/3885>

Related PR:

Smoking May Lead to Cataracts in Aging Population

<http://www.sciencedaily.com/releases/2012/10/121013174121.htm>

Smoking may lead to cataracts in aging population

http://www.arvo.org/About_ARVO/Press_Room/Smoking_may_lead_to_cataracts_in_aging_population/

Cigarette smoking may increase risk of age-related cataract

<http://www.news-medical.net/news/20121013/Cigarette-smoking-may-increase-risk-of-age-related-cataract.aspx>

Smoking affects mRNA expression of bone morphogenetic proteins in human periosteum

J Bone Joint Surg Br. 2012 Oct;94(10):1427-32.

[Chassanidis CG](#), [Malizos KN](#), [Varitimidis S](#), [Samara S](#), [Koromila T](#), [Kollia P](#), [Dailiana Z](#).

Abstract

Periosteum is important for bone homeostasis through the release of bone morphogenetic proteins (BMPs) and their effect on osteoprogenitor cells. Smoking has an adverse effect on fracture healing and bone regeneration. The aim of this study was to evaluate the effect of smoking on the expression of the BMPs of human periosteum. Real-time polymerase chain reaction was performed for BMP-2, -4, -6, -7 gene expression in periosteal samples obtained from 45 fractured bones (19 smokers, 26 non-smokers) and 60 non-fractured bones (21 smokers, 39 non-smokers). A hierarchical model of BMP gene expression (BMP-2 > BMP-6 > BMP-4 > BMP-7) was demonstrated in all samples. When smokers and non-smokers were compared, a remarkable reduction in the gene expression of BMP-2, -4 and -6 was noticed in smokers. The comparison of fracture and non-fracture groups demonstrated a higher gene expression of BMP-2, -4 and -7 in the non-fracture samples. Within the subgroups (fracture and non-fracture), BMP gene expression in smokers was either

lower but without statistical significance in the majority of BMPs, or similar to that in non-smokers with regard to BMP-4 in fracture and BMP-7 in non-fracture samples. In smokers, BMP gene expression of human periosteum was reduced, demonstrating the effect of smoking at the molecular level by reduction of mRNA transcription of periosteal BMPs. Among the BMPs studied, BMP-2 gene expression was significantly higher, highlighting its role in bone homeostasis.

<http://www.bjj.boneandjoint.org.uk/content/94-B/10/1427.long>

The relationship between oxidative stress, smoking and the clinical severity of psoriasis

[J Eur Acad Dermatol Venereol.](#) 2012 Sep 25. doi: 10.1111/j.1468-3083.2012.04700.x. [Epub ahead of print]

[Emre S](#), [Metin A](#), [Demirseren DD](#), [Kilic S](#), [Isikoglu S](#), [Erel O](#).

Abstract

Background Recent studies suggested that increased oxidant products and decreased antioxidant system functions may be involved in the pathogenesis of psoriasis. In this study, we investigated total oxidative status, Paraoxonase (PON)1/arylesterase enzyme activities and severity of the disease in smoker and non-smoker psoriatic patients. **Methods** Fifty-four patients with plaque type psoriasis (28 smokers and 26 non-smokers) and 62 healthy volunteers (16 smokers and 46 non-smokers) were enrolled in the study. Serum total oxidant status (TOS), total antioxidant capacity (TAC) and arylesterase levels were measured, and oxidative stress index (OSI) was calculated in all participants. **Results** Psoriasis Area and Severity Index scores were significantly higher in smoker patients than in non-smoker patients ($P = 0.014$). Both smoker and non-smoker patients had significantly increased TOS levels and OSI values and decreased TAC levels than healthy subjects (all P values = 0.000). The TAC and TOS levels, OSI values and arylesterase activities were similar between smoker and non-smoker patients. The levels of triglyceride (TG), total cholesterol (TC), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) were not significantly different between smoker and non-smoker psoriasis patients. When compared with non-smoking controls, only smoking psoriasis patients had significantly higher TG ($P = 0.005$), lower HDL ($P = 0.022$) and lower arylesterase levels ($P = 0.015$). There were no significant correlations with Psoriasis Area and Severity Index (PASI) scores and TAC, TOS, OSI, TG, TC, HDL and LDL levels in all psoriasis patients. **Conclusions** Oxidative stress is increased in psoriasis patients regardless of their smoking status. The decreased arylesterase activity in smoker psoriasis patients suggested that smoking may be a considerable risk factor that increases the severity of psoriasis by increasing oxidative stress in these patients.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1468-3083.2012.04700.x/abstract>

Comparing the joint effect of arsenic exposure, cigarette smoking and risk genotypes of vascular endothelial growth factor on upper urinary tract urothelial carcinoma and bladder cancer

[J Hazard Mater.](#) 2012 Sep 7. pii: S0304-3894(12)00871-0. doi: 10.1016/j.jhazmat.2012.08.056. [Epub ahead of print]

[Wang YH](#), [Yeh SD](#), [Wu MM](#), [Liu CT](#), [Shen CH](#), [Shen KH](#), [Pu YS](#), [Hsu LI](#), [Chiou HY](#), [Chen CJ](#).

Abstract

Arsenic exposure and cigarette smoking are environmental risk factors for urothelial carcinoma (UC). Vascular endothelial growth factor (VEGF) is the key regulator of angiogenesis in various malignancies. This study investigates the joint effect of arsenic exposure, cigarette smoking, and VEGF polymorphisms on UC risk. This was a hospital-based case-control study consisting of 730 histopathologically confirmed UC cases, including 470 bladder cancers, 260 upper urinary tract UCs (UUTUCs), and 850 age-matched controls, recruited from September 1998 to December 2009. UC risk was estimated by odds ratios and 95% confidence intervals using unconditional logistic regression. Ever smokers with high arsenic exposure had significantly increased risks of 5.7 and 6.4 for bladder cancer and UUTUC, respectively. Moreover, ever smokers with high arsenic exposure carrying 1 or 2 risk genotypes of the VEGF gene had a significantly increased risk of 6.6 for bladder cancer and a strikingly higher risk of 9.9 for UUTUC. Additionally, UUTUC cases with high arsenic exposure carrying 1 or 2 risk genotypes of the VEGF gene had a non-significant increased risk of advanced tumor stage. Our findings suggest that arsenic exposure, cigarette smoking, and risk genotypes of VEGF contribute to a higher risk of UUTUC than of bladder cancer.

<http://www.sciencedirect.com/science/article/pii/S0304389412008710>

Reducing harm from tobacco use

[J Psychopharmacol](#). 2012 Oct 3. [Epub ahead of print]

[McNeill A](#), [Munafò MR](#).

Abstract

If current trends in smoking prevalence continue, even with the implementation of enhanced tobacco control measures, millions of smokers will continue to fall ill and die as a direct result of their smoking. Many of these will be from the most deprived groups in society - smoking continues to be one of the strongest drivers of health inequalities. The personal costs of this morbidity and mortality, as well as costs to business and the economy, are unequalled and will therefore remain high for several decades to come. However, there is an addition to the tobacco control armoury that could have a marked impact on public health, but it requires radical action to be taken. This would be to embrace harm reduction, but this approach is as controversial in the case of tobacco as it is in the case of illicit drugs from where it derives. However, harm reduction remains the Cinderella of the three major strategies for reducing smoking-related harm, the others being prevention and cessation. Here we make the case that harm reduction has an important role to play in reducing the health burden of tobacco use.

<http://jop.sagepub.com/content/early/2012/09/26/0269881112458731.abstract>

Greater Elevation in Risk for Nicotine Dependence per Pack of Cigarettes Smoked Among Those With an Anxiety Disorder

J. Stud. Alcohol Drugs, 73, 920–924, 2012

Matt G. Kushner, Kyle R. Menary, Eric W. Maurer, Paul Thuras

Abstract

Objective: Recent work shows that the time from the initial use of nicotine, cannabis, and alcohol to the onset of dependence on these substances is shorter ("telescoped") in anxiety-disordered individuals. Previously, we hypothesized that telescoping may result from a shared neurobiology underlying both anxiety disorders and dependence. This hypothesis implies that telescoping occurs because individuals with an anxiety disorder transition to dependence with less overall drug exposure ("dependence susceptibility"). To investigate this further, we examined an estimate of the amount smoked (rather than the time transpired) from smoking initiation milestones to the onset of nicotine dependence in those with and without an anxiety disorder. **Method:** We used the subset of respondents in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) Wave 1 who reported having smoked at least 100 cigarettes (N = 18,013). All data were based on face-to-face interviews. **Results:** Individuals with any anxiety disorder transitioned to nicotine dependence after smoking fewer total cigarettes than did individuals with no anxiety disorder. Furthermore, those with more than one anxiety disorder transitioned to nicotine dependence after smoking fewer cigarettes than did those with one anxiety disorder only. Several potentially confounding covariates were controlled for in these analyses.

Conclusions: Dependence susceptibility is a novel concept with the potential to inform theoretical accounts of and prevention strategies for substance dependence among those with an anxiety disorder. In addition to nicotine, our theory and past data suggest that dependence susceptibility for other addictive substances (e.g., alcohol) also would be found among those with an anxiety disorder.

<http://www.jsad.com/jsad/article/Greater Elevation in Risk for Nicotine Dependence per Pack of Cigarettes Sm/4751.html>

Also:

Barriers to Effective Tobacco-Dependence Treatment for the Very Poor

<http://www.jsad.com/jsad/article/Barriers to Effective TobaccoDependence Treatment for the Very Poor/4746.html>

Viewpoint

Prevention of non-communicable diseases in New York City

[Deborah Dowell](#), [Thomas A Farley](#)

Non-communicable diseases cause more than three of five deaths worldwide. *The Lancet* has published reviews on the burden of non-communicable diseases¹ and on so-called best buys for prevention [2], [3] and [4] based on health effects, cost-effectiveness, and political and financial feasibility.⁵ Although several communities including Somerville (MA, USA) and North Karelia (Finland) have undertaken interventions to prevent non-communicable diseases, [6] and [7] much of the work assessing such interventions has been theoretical. Local and national prevention programmes need more examples of actual best-buy interventions and lessons learned from implementation.

Over the past decade, New York City has pursued an agenda intended to solve the problems with the largest effects on its residents' health.⁸ This agenda has had a particular focus on factors underlying non-communicable diseases, including tobacco use, unhealthy diet, and physical inactivity. A report published by the New York City Department of Health and Mental Hygiene, *Preventing Non-Communicable Diseases and Injuries*,⁹ summarises these initiatives. In this Viewpoint we discuss a few examples that show the city's overall approach.

In 2002, New York City launched a tobacco control campaign to protect New Yorkers from secondary smoke and to make smoking less convenient and easier to quit. Increases in city and state excise taxes made cigarettes, at more than US\$11 a pack, more expensive than anywhere else in the USA. The Smoke-Free Air Act of 2002 made all New York City workplaces (including restaurants and bars) smoke-free beginning in 2003, and was amended to make public parks and beaches smoke-free in 2011. Mass media campaigns graphically depict the health consequences of smoking...

Several lessons have emerged from New York's experience. First, approaches that change the physical and social environment (eg, smoke-free workplaces, healthier default food options, infrastructure that facilitates physical activity) mean that people are likely to make healthier choices. Second, prevention of non-communicable diseases is not necessarily costly. Many health-promotion policies (eg, bans on smoking and trans-fat use) cost government very little in public funds, and some initiatives (eg, cigarette taxes) generate revenue for government that can be used to pay for prevention programmes. Finally, effective interventions require support from the highest levels of government. Local government support was crucial in implementation of the policies that we describe. Political commitment is fundamental to success in the fight against non-communicable diseases. As New York City Mayor Michael Bloomberg has noted, "while government action is not sufficient alone, it is nevertheless absolutely essential. There are powers only governments can exercise, policies only governments can mandate and enforce, and results only governments can achieve. To halt the worldwide epidemic of non-communicable diseases, governments at all levels must make healthy solutions the default social option. That is, ultimately, government's highest duty."²⁵

<http://www.sciencedirect.com/science/article/pii/S0140673612607338>

[http://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(12\)60733-8/fulltext](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(12)60733-8/fulltext)

Referenced report:

Preventing non-communicable diseases and injuries: innovative solutions from New York City
New York City Department of Health and Mental Hygiene, New York (2011)

<http://www.nyc.gov/html/doh/html/ncd/nyc-solutions.shtml>

<http://www.nyc.gov/html/doh/downloads/pdf/ip/un-rpt.pdf>

Note: Open Access. Full text PDF freely available from link immediately above.

Cyto-genotoxic effects of smoke from commercial filter and non-filter cigarettes on human bronchial and pulmonary cells

Mutat Res. 2012 Sep 23. pii: S1383-5718(12)00284-7. doi: 10.1016/j.mrgentox.2012.06.013. [Epub ahead of print]

[Cavallo D](#), [Ursini CL](#), [Freseigna AM](#), [Maiello R](#), [Ciervo A](#), [Ferrante R](#), [Buresti G](#), [Iavicoli S](#).

Abstract

Cigarette smoke is a complex mixture of chemicals, some of which are known as carcinogens. The cyto-genotoxic effects of cigarette-smoke extract (CSE) from commercial cigarettes without (A and B) and with filter (C and D) were evaluated at different CSE concentrations on A549 and BEAS-2B cells. The particle content of the cigarette smoke and the metal composition of the CSE were also analyzed. The cells were exposed to 1-10% of the CSE from one cigarette per experiment. Cytotoxicity was evaluated by use of the MTT assay after 24h, and the lactate dehydrogenase (LDH) assay after 30min and 24h. The Fpg-modified comet assay was used to evaluate direct-oxidative DNA damage on cells exposed for 30min. As expected, unfiltered cigarette smoke (particularly from the B cigarette) contained a higher number

of particles than filtered smoke. With smoke extract from the B cigarette we found a decrease in cell viability only in BEAS-2B cells. The results of the LDH test showed membrane damage for B-cigarette smoke extract, particularly in BEAS-2B cells. Extracts from unfiltered cigarette smoke induced significant direct DNA damage, to a larger extent in A549 cells. Filtered cigarette-smoke extract induced a significant direct DNA damage at 5-10%. A significant induction of oxidative DNA damage was found at the highest CSE concentration in both cell types (by smoke extracts from B and C cigarettes in A549 cells, and from A and D cigarettes in BEAS-2B cells). Smoke extracts from filter cigarettes induced less direct DNA damage than those from unfiltered cigarettes in A549 cells, probably due to a protective effect of filter. In BEAS-2B cells the smoke extract from the B-cigarette showed the highest genotoxic effect, with a concentration-dependent trend. These findings show a higher cyto-genotoxicity for smoke extracts from the B-cigarette and oxidative effects for those from the A and D cigarettes, particularly in BEAS-2B cells. Moreover, there was a higher responsiveness of A549 cells to genotoxic insult of CSE, and a cigarette-dependent genotoxicity in BEAS-2B cells. Our experimental model demonstrated to be suitable to sensitively detect early genotoxic response of different lung-cell types to non-cytotoxic concentrations of complex inhalable mixtures.

<http://www.sciencedirect.com/science/article/pii/S1383571812002847>

Alpha Oscillations in Response to Affective and Cigarette-Related Stimuli in Smokers

Nicotine Tob Res 2012 published 11 October 2012

Yong Cui, Francesco Versace, Jeffrey M. Engelmann, Jennifer A. Minnix, Jason D. Robinson, Cho Y. Lam, Maher Karam-Hage, Victoria L. Brown, David W. Wetter, John A. Dani, Thomas R. Kosten, and Paul M. Cinciripini

Abstract

Introduction: The presence of cigarette-related cues has been associated with smoking relapse. These cues are believed to activate brain mechanisms underlying emotion, attention, and memory. Electroencephalography (EEG) alpha desynchronization (i.e., reduction in alpha power) has been suggested to index the engagement of these mechanisms. Analyzing EEG alpha desynchronization in response to affective and smoking cues might improve our understanding of how smokers process these cues, and the potential impact of this processing on relapse.

Methods: Before the start of a medication-assisted cessation attempt, we recorded EEG from 179 smokers during the presentation of neutral, pleasant, unpleasant, and cigarette-related pictures. Wavelet analysis was used to extract EEG alpha oscillations (8–12 Hz) in response to these pictures. Alpha oscillations were analyzed as a function of picture valence and arousal dimensions.

Results: Emotional and cigarette-related stimuli induced a higher level of alpha desynchronization (i.e., less power in the alpha frequency band) than neutral stimuli. In addition, the level of alpha desynchronization induced by cigarette-related stimuli was similar to that induced by highly arousing stimuli (i.e., erotica and mutilations).

Conclusions: These results suggest that, for smokers, cigarette-related cues are motivationally significant stimuli that may engage emotional, attentional, and memory-related neural mechanisms at a level comparable to that seen in response to highly arousing stimuli. This finding suggests that activation of emotional, attentional, and memory-related brain mechanisms may be an important contributor to cue-induced smoking relapse.

<http://ntr.oxfordjournals.org/content/early/2012/10/11/ntr.nts209.abstract>

Insular and anterior cingulate circuits in smokers with schizophrenia

Schizophr Res. 2012 Sep 26. pii: S0920-9964(12)00516-6. doi: 10.1016/j.schres.2012.08.033. [Epub ahead of print]

[Moran LV](#), [Sampath H](#), [Stein EA](#), [Hong LE](#).

Abstract

Schizophrenia (SZ) is associated with high rates of smoking. We previously found that resting state functional connectivity (rsFC) between the dorsal anterior cingulate (dACC) and striatum is independently associated with nicotine addiction and psychiatric illness. Since the insula is implicated in nicotine dependence, we hypothesized that SZ smokers will have greater dysfunction in smoking-related insular and dACC circuits than normal control smokers (NC) independent of smoking severity, consistent with an inherent disease-related weakening of smoking-related circuits. Nicotine challenge was used to demonstrate that decreased rsFC in identified circuits reflects addiction trait and is not affected by pharmacological state. Twenty-four NC smokers and 20 smokers with SZ matched on nicotine addiction

severity participated in a resting state fMRI study and were scanned during two separate sessions while receiving a placebo or nicotine patch, in a randomized, cross-over design. Using individualized, anatomically defined anterior and posterior insula and dACC as regions of interest (ROI), whole brain rsFC was performed using each ROI as a seed. Significant negative correlations between smoking severity and rsFC between insula, dACC and striatum were found for both groups. Furthermore, smokers with SZ demonstrated additive reductions in circuit strength between the dACC and insula compared to NC smokers independent of smoking severity. Nicotine challenge did not significantly alter rsFC in insula-dACC-striatal circuits. Reduced rsFC strength between the insula, dACC and striatum is associated with nicotine addiction severity in both non-psychiatrically ill and in SZ smokers. Decreased insula-dACC rsFC may index overlapping circuitry associated with smoking and SZ.

<http://www.schres-journal.com/article/S0920-9964%2812%2900516-6/abstract>

<http://www.sciencedirect.com/science/article/pii/S0920996412005166>

Effect of smoking on sperm vitality, DNA integrity, seminal oxidative stress, zinc in fertile men

Urology. 2012 Oct;80(4):822-5. doi: 10.1016/j.urology.2012.07.002.

Taha EA, Ez-Aldin AM, Sayed SK, Ghandour NM, Mostafa T.

Abstract

OBJECTIVE:

To assess the effect of smoking on sperm vitality, sperm DNA integrity, semen reactive oxygen species, and zinc levels in fertile men.

METHODS:

One-hundred sixty men were investigated. They were divided into 2 equal groups: healthy fertile nonsmokers and healthy fertile smokers. They were subjected to history taking, clinical examination, and semen analysis. In their semen, sperm hypo-osmotic swelling test, sperm DNA fragmentation test, seminal reactive oxygen species, and zinc were assessed.

RESULTS:

Compared with fertile nonsmokers, fertile smokers were significantly associated with lower hypo-osmotic swelling test and seminal zinc levels and significantly associated with higher sperm DNA fragmentation percent and seminal reactive oxygen species levels.

CONCLUSION:

Smoking (cigarettes/day and duration) has detrimental effects on sperm motility, viability, DNA fragmentation, seminal zinc levels, and semen reactive oxygen species levels, even in fertile men, and it is directly correlated with cigarette quantity and smoking duration.

<http://www.goldjournal.net/article/S0090-4295%2812%2900768-6/abstract>

<http://www.sciencedirect.com/science/article/pii/S0090429512007686>

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