From: "Stan Shatenstein" <shatensteins@sympatico.ca>
To: Undisclosed-Recipient:;
Date: 17.9.2012 13:00:17
Subject: STAN Bulletin: 33rd Edition: 17-September-2012

Smoking & Tobacco Abstracts & News
STAN Bulletin
33rd Edition
17-September-2012

Editor's Note: In the Open Access journal, BMC Cancer, Philip Morris Products S.A. has funded long-time industry consultant PN Lee and colleagues to conduct a 'Systematic review with meta-analysis of the epidemiological evidence in the 1900s relating smoking to lung cancer'. The authors conclude: "The association of lung cancer with smoking is strong, evident for all lung cancer types, dose-related and insensitive to covariate-adjustment." Nice to see tobacco money well spent for a change.

Stan Shatenstein

Noteworthy:
"More than 250 experiments have been published during the last 40 years in order to test various aspects of the mere exposure effect, according to which repeated unreinforced exposure to a stimulus is sufficient to enhance one's attitude toward the stimulus. In the context of tobacco marketing it might be a useful tool for explaining advertising effects that are not well covered by traditional theoretical models like the theory of reasoned action, i.e. 'Why do people engage in behaviors such as smoking they know are harmful and potentially life threatening?'. Rather than assuming people weigh the pros and cons in a reflective way and then make reasoned choices, research on implicit cognitions and on the mere exposure effect may lead to the assumption that the positive effects of advertising for products like cigarettes can also occur without conscious cognitions. 'Implicit preferences' have also been shown in studies that address cue reactions, suggesting that smokers have an attentional bias towards smoking-related cues like advertising images." [Morgenstern M, Isensee B, Hanewinkel R. Seeing and Liking Cigarette Advertisements: Is There a 'Mere Exposure' Effect?, *Eur Addict Res*]

In the News:

- Canada: NCACT: Industry-funded Coalition claims illegal cigarettes fund criminals
- Canada: MNI: How genetics shape our addictions; PR [Neuroimage: Tang]
- China: Long way to go before country becomes smoke-free
- China: Yunnan: Tobacco production proposed as earthquake relief; Lobby opposed; Scheme goes up in smoke
- EU: PITOC: Sixteen countries jointly warn that additives increase health hazards of cigarettes; Factsheets
- Germany: Smokers may have more sleep problems [Addict Biol: Cohrs]
- India: Gutka: Smokeless tobacco spells doom: Cancers of the gums, throat & oral cavity
- Norway: Court rejects lawsuit over store display of tobacco products; Philip Morris loses [Ruling]
- Philippines: Cigarette Tax Assessment Sought by Tobacco Firms; Half would quit on increase: Survey
- Spain: Passive smoking also affects neurodevelopment in babies; PR [Early Hum Dev: Hernández-Martínez]
- Thailand: Tobacco growers oppose tough plain pack marketing law
- Ukraine: Law banning cigarette advertising comes into force
- UK: NI: MLAs vote to ban tobacco advertising & promotion to prevent youth uptake
- US: Gallup Poll: The War On Smoking Is Working: Nearly eight in 10 want to quit
- US: Forbes: Opinion: Are Federal Taxes Driving Smokers to Stop Lighting Up?

In this Edition:

- Addiction - St. Helen: US: Racial differences in tobacco dependence & nicotine & carcinogen exposure
- Addict Biol - Durazzo: Smoking & age effects on brain volumes in early abstinence in alcohol dependence
- Am J Commun Psych - Lorenzo-Blanco: US: Latino/a Depression & Smoking: Culture, Gender & Ethnicity
- Am J Gastroenterol - Rosenfeld: IBD: Truth about cigarette smoking & inflammatory bowel disease risk
- BMC Cancer - Lee: Epidemiological evidence in the 1900s relating smoking to lung cancer

- Cell - Govindan: Non-Small Cell Lung Cancer Genomic Landscape in Smokers & Never-Smokers
- Eur Addict Res - Morgenstern: Germany: Seeing & Liking Cigarette Ads: Is There a 'Mere Exposure' Effect?
- Glob Pub Health - Kenny: Proctor: Golden Holocaust: Origins of the cigarette catastrophe & the case for abolition
- Ind J Dent Res - Sharma: India: AgNOR expression in tobacco chewers' exfoliated oral mucosal cells & lesion
- Int J Cancer - Zhou: US: New England: Smokeless tobacco & head & neck cancer risk
- IJPH - Schunck: Germany: No causal effect of unemployment on smoking? Panel study
- J Dent Hyg - Sutton: US: Environmental tobacco smoke & periodontitis in non-smokers
- J Health Psych - Avallone: US: Asthma, Smoking Behavior & Cessation Motives among Adult Daily Smokers
- JIMR - Streja: US: CA: LA: SHS Exposure & Low Income Minority Children with Asthma: Minimal Intervention
- JMIR - Graham: US: Online advertising to reach & recruit Latino smokers to an internet cessation program
- JPHP - Drope: FCTC: Tobacco control & trade policy: Proactive strategies for integrating policy norms
- Medicus J - Daube: Australia: WA: Time to stub out any obstacles that lie in the way of a smoke-free society
- Lancet - Abascal: Uruguay: FCTC: Tobacco control campaign: population-based trend analysis
- Neurol Sci - Ojeda-López: Mexico: Parkinson's: Caffeine drinking, smoking & dopaminergic replacement therapy
- PLoS One - Hosseinpoor: WHS: Socioeconomic inequality in smoking in low- & middle-income countries
- Psychopharmacol - Wachter: Nicotine, antisaccade eye-gaze & emotional stimuli in nonsmokers
- Toxicol In Vitro - Fearon: BAT: In vitro models for assessing potential CVD smoking risk

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Abstracts:

Racial differences in the relationship between tobacco dependence and nicotine and carcinogen exposure

Addiction
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: **13 SEP 2012**

Gideon St.Helen, Delia Dempsey, Margaret Wilson, Peyton Jacob III and Neal L. Benowitz

Abstract

Aims
To investigate the relationships between tobacco dependence, biomarkers of nicotine and carcinogen exposure, and biomarkers of nicotine and carcinogen exposure per cigarette in Black and White smokers.

Design and participants

204 healthy Black (n=69) and White (n=135) smokers were enrolled in two clinical studies.

Measurement

Nicotine equivalents (nicotine and its metabolites), 4-(methylnitrosamino)-1-(3)pyridyl-1-butanol (NNAL), and polycyclic aromatic hydrocarbon (PAH) metabolites were measured in urine. The Fagerström Test for Nicotine Dependence (FTND) and time to first cigarette (TFC) measured tobacco dependence.

Findings

Average TFC and FTND for Blacks and Whites were not significantly different. Urine NNAL and nicotine equivalents increased with increasing FTND in Whites but did not increase in Blacks (race x FTND interaction, both $p<0.031$). The interaction term was not significant for PAHs. An inverse relationship was seen between FTND and nicotine equivalents, NNAL, and PAH metabolites per cigarette in Blacks but remained flat in Whites (race x FTND interaction, all $p\leq0.039$). Regardless of dependence (low dependence, TFC>15 minutes; high dependence, TFC≤15 minutes), FTND and TFC were not significantly correlated with urine nicotine equivalents and carcinogen exposure in Blacks. We found moderate correlations between FTND and TFC and nicotine equivalents and carcinogen exposure among Whites of low dependence and non-significant correlations among Whites of high dependence.

Conclusion

In the US, tobacco dependence measures were linearly related to nicotine intake and carcinogen exposure in White but not in Black smokers. The relationship between dependence measures and tobacco biomarkers in Black smokers regardless of level of dependence resembled highly dependent White smokers.

http://onlinelibrary.wiley.com/doi/10.1111/j.1360-0443.2012.04077.x/abstract

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Interactive effects of chronic cigarette smoking and age on brain volumes in controls and alcohol-dependent individuals in early abstinence

Addict Biol. 2012 Sep 3. doi: 10.1111/j.1369-1600.2012.00492.x. [Epub ahead of print]

Durazzo TC, Mon A, Pennington D, Abé C, Gazdzinski S, Meyerhoff DJ.

Abstract
Chronic alcohol-use disorders (AUDs) have been shown to interact with normal age-related volume loss to exacerbate brain atrophy with increasing age. However, chronic cigarette smoking, a highly co-morbid condition in AUD and its influence on age-related brain atrophy have not been evaluated. We performed 1.5 T quantitative magnetic resonance imaging in non-smoking controls [non-smoking light drinking controls (nsCONs); n = 54], smoking light drinking controls (sCONs, n = 34), and one-week abstinent, treatment-seeking alcohol-dependent (ALC) non-smokers (nsALCs, n = 35) and smokers (sALCs, n = 43), to evaluate the independent and interactive effects of alcohol dependence and chronic smoking on regional cortical and subcortical brain volumes, emphasizing the brain reward/executive oversight system (BREOS). The nsCONs and sALCs showed greater age-related volume losses than the nsALCs in the dorsal prefrontal cortex (DPFC), total cortical BREOS, superior parietal lobule and putamen. The nsALCs and sALCs demonstrated smaller volumes than the nsCONs in most cortical region of interests (ROIs). The sCONs had smaller volumes than the nsCONs in the DPFC, insula, inferior parietal lobule, temporal pole/parahippocampal region and all global cortical measures. The nsALCs and sALCs had smaller volumes than the sCONs in the DPFC, superior temporal gyrus, inferior and superior parietal lobules, precuneus and all global cortical measures. Volume differences between the nsALCs and sALCs were observed only in the putamen. Alcohol consumption measures were not related to volumes in any ROI for ALC; smoking severity measures were related to corpus callosum volume in the sCONs and sALCs. The findings indicate that consideration of smoking status is necessary for a better understanding of the factors contributing to regional brain atrophy in AUD.

http://onlinelibrary.wiley.com/doi/10.1111/j.1369-1600.2012.00492.x/abstract

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Latino/a Depression and Smoking: An Analysis Through the Lenses of Culture, Gender, and Ethnicity

Am J Community Psychol. 2012 Sep 7. [Epub ahead of print]

Lorenzo-Blanco EI, Cortina LM.

Abstract

Rates of major depressive disorder (MDD) and cigarette smoking increase with Latino/a acculturation, but this varies by gender and ethnic subgroup. We investigated how lived experiences (i.e., discrimination, family conflict, family cohesion, familismo) clustered together in the everyday lives of Latina/os. We further examined associations of cluster profile and Latino/a subgroup with MDD and smoking, and tested whether gender moderated these associations. Data came from the National Latino Asian American Study, which included 2,554 Latino/as (48 % female; mean age = 38.02 years). K-means cluster analysis revealed six profiles of experience, which varied by gender and socio-cultural characteristics. Proportionately more women than men were in groups with problematic family lives. Acculturated Latino/as were disproportionately represented in profiles reporting frequent discrimination, family conflict, and a lack of shared family values and cohesion. Profiles characterized by high discrimination and family problems also predicted elevated risk for MDD and smoking. Findings suggest that Latino/a acculturation comes jointly with increased discrimination, increased family conflict, and reduced family cohesion and shared family values, exacerbating risk for MDD and smoking. This research on pathways to depression and smoking can inform the development of targeted assessment, prevention, and intervention strategies, tailored to the needs of Latino/as.

http://www.springerlink.com/content/p686635l72h65t5h/
http://www.springerlink.com/content/p686635l72h65t5h/fulltext.pdf

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

Editorial

The truth about cigarette smoking and the risk of inflammatory bowel disease

Am J Gastroenterol. 2012 Sep;107(9):1407-8. doi: 10.1038/ajg.2012.190.

Rosenfeld G, Bressler B.

Abstract
The etiology of inflammatory bowel disease (IBD) is generally believed to be multifactorial in nature involving both genetic and environmental factors. Cigarette smoking has been shown through previous retrospective observational studies to be an environmental factor with both positive and negative influences in IBD. Smoking increases the risk of developing Crohn's disease (CD) but not the risk of ulcerative colitis (UC). Meanwhile smoking cessation increases the risk of a UC flare while CD patients are more likely to show a decrease in disease severity. Unfortunately, these observational studies cannot control for bias the way a randomized controlled trial can, however, they still reveal meaningful truths about smoking and IBD. The study by Higuchi et al. (1) adds to our understanding of the impact of smoking on IBD in several ways. They showed that increasing exposure to smoking is associated with an increased risk of developing CD. They also showed that the risk of UC is highest in the first 2-5 years after smoking cessation but remains elevated for >20 years. This research also raises several new issues regarding the association between smoking and IBD, which hopefully will be answered through future well-designed observational studies.

http://www.nature.com/ajg/journal/v107/n9/full/ajg2012190a.html

Referenced *Am J Gastroenterol* study:

A Prospective Study of Cigarette Smoking and the Risk of Inflammatory Bowel Disease in Women
http://www.nature.com/ajg/journal/vaop/ncurrent/abs/ajg2012196a.html

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Systematic review with meta-analysis of the epidemiological evidence in the 1900s relating smoking to lung cancer

BMC Cancer. 2012 Sep 3;12(1):385. [Epub ahead of print]

Lee PN, Forey BA, Coombs KJ.

Abstract

BACKGROUND:

Smoking is a known lung cancer cause, but no detailed quantitative systematic review exists. We summarize evidence for various indices.

METHODS:

Papers published before 2000 describing epidemiological studies involving 100+ lung cancer cases were obtained from Medline and other sources. Studies were classified as principal, or subsidiary where cases overlapped with principal studies. Data were extracted on design, exposures, histological types and confounder adjustment. RRs/ORs and 95% CIs were extracted for ever, current and ex smoking of cigarettes, pipes and cigars and indices of cigarette type and dose--response. Meta-analyses and meta-regressions investigated how relationships varied by study and RR characteristics, mainly for outcomes exactly or closely equivalent to all lung cancer, squamous cell carcinoma ("squamous") and adenocarcinoma ("adeno").

RESULTS:

287 studies (20 subsidiary) were identified. Although RR estimates were markedly heterogeneous, the meta-analyses

demonstrated a relationship of smoking with lung cancer risk, clearly seen for ever smoking (random-effects RR 5.50, CI 5.07-5.96) current smoking (8.43, 7.63-9.31), ex smoking (4.30, 3.93-4.71) and pipe/cigar only smoking (2.92, 2.38-3.57). It was stronger for squamous (current smoking RR 16.91, 13.14-21.76) than adeno (4.21, 3.32-5.34), and evident in both sexes (RRs somewhat higher in males), all continents (RRs highest for North America and lowest for Asia, particularly China), and both study types (RRs higher for prospective studies). Relationships were somewhat stronger in later starting and larger studies. RR estimates were similar in cigarette only and mixed smokers, and similar in smokers of pipes/cigars only, pipes only and cigars only. Exceptionally no increase in adeno risk was seen for pipe/cigar only smokers (0.93, 0.62-1.40). RRs were unrelated to mentholation, and higher for non-filter and handrolled cigarettes. RRs increased with amount smoked, duration, earlier starting age, tar level and fraction smoked and decreased with time quit. Relationships were strongest for small and squamous cell, intermediate for large cell and weakest for adenocarcinoma. Covariate-adjustment little affected RR estimates.

CONCLUSIONS:

The association of lung cancer with smoking is strong, evident for all lung cancer types, dose-related and insensitive to covariate-adjustment. This emphasises the causal nature of the relationship. Our results quantify the relationships more precisely than previously.

Competing interests

PNL, founder of P.N.Lee Statistics and Computing Ltd., is an independent consultant in statistics and an advisor in the fields of epidemiology and toxicology to a number of tobacco, pharmaceutical and chemical companies. This includes Philip Morris Products S.A., the sponsor of this study. BAF and KJC are employees of P.N.Lee Statistics and Computing Ltd.

Acknowledgements

This research was funded by Philip Morris Products S.A. However the opinions and conclusions of the authors are their own, and do not necessarily reflect the position of Philip Morris Products S.A...

http://www.biomedcentral.com/1471-2407/12/385/abstract
http://www.biomedcentral.com/content/pdf/1471-2407-12-385.pdf

Note: Tobacco industry-funded research. Open Access. Full text PDF freely available from link immediately above.

Genomic Landscape of Non-Small Cell Lung Cancer in Smokers and Never-Smokers

Cell, Volume 150, Issue 6, 1121-1134, 14 September 2012

Ramaswamy Govindan, Li Ding, Malachi Griffith, Janakiraman Subramanian, Nathan D. Dees, Krishna L. Kanchi, Christopher A. Maher, Robert Fulton, Lucinda Fulton, John Wallis, Ken Chen, Jason Walker, Sandra McDonald, Ron Bose, David Ornitz, Donghai Xiong, Ming You, David J. Dooling, Mark Watson, Elaine R. Mardis, Richard K. Wilson

Summary

We report the results of whole-genome and transcriptome sequencing of tumor and adjacent normal tissue samples from 17 patients with non-small cell lung carcinoma (NSCLC). We identified 3,726 point mutations and more than 90 indels in the coding sequence, with an average mutation frequency more than 10-fold higher in smokers than in never-smokers. Novel alterations in genes involved in chromatin modification and DNA repair pathways were identified, along with *DACH1*, *CFTR*, *RELN*, *ABCB5*, and *HGF*. Deep digital sequencing revealed diverse clonality patterns in both never-smokers and smokers. All validated *EFGR* and *KRAS* mutations were present in the founder clones, suggesting possible roles in cancer initiation. Analysis revealed 14 fusions, including ROS1 and ALK, as well as novel metabolic enzymes. Cell-cycle and JAK-STAT pathways are significantly altered in lung cancer, along with perturbations in 54 genes that are potentially targetable with currently available drugs.

http://www.cell.com/retrieve/pii/S0092867412010227

Related PR:

In Lung Cancer, Smokers Have 10 Times More Genetic Damage Than Never-Smokers
http://www.sciencedaily.com/releases/2012/09/120913122836.htm

Seeing and Liking Cigarette Advertisements: Is There a 'Mere Exposure' Effect?

Eur Addict Res. 2012 Aug 28;19(1):42-46. [Epub ahead of print]

Morgenstern M, Isensee B, Hanewinkel R.

Abstract
Aims: We aimed to explain the association between exposure to a cigarette advertisement and favorable attitudes towards the advertisement. Methods: We used data from an observational cross-sectional study with a sample of 3,415 German schoolchildren aged 10-17 years. Cigarette advertising exposure was assessed with an image of a Marlboro ad, asking for contact frequency (number of times seen the ad) and brand name. Liking of the ad was measured with two items (alpha = 0.78). Results: We found a positive linear association between exposure to the Marlboro ad and liking it. This association remained significant (standardized $\beta = 0.09$; $p < 0.001$) even after statistical control for smoking status, smoking of friends and parents, attitudes towards smoking in general, cigarette advertising receptivity (having a favorite cigarette ad), exposure to other advertisings, age, sex, socioeconomic status, rebelliousness and sensation seeking, self-reported school performance, and study region. Conclusions: The association between exposure to an advertisement and liking it was robust and could not be fully explained without referring to either unmeasured confounding or implicit advertising effects (e.g. mere exposure). Implicit effects have implications for prevention strategies as it may be very difficult to counteract unconscious advertising effects.

http://content.karger.com/produktedb/produkte.asp?DOI=10.1159/000339836

Book Review

Golden holocaust: Origins of the cigarette catastrophe and the case for abolition

Glob Public Health. 2012 Sep 6. [Epub ahead of print]

Kenny KE.

During the twentieth century, about a hundred million people were killed by tobacco. This pales, however, in comparison with the one billion deaths we can anticipate during the twenty-first century if current trends continue. Tobacco now claims six million lives each year, more than from AIDS, malaria and traffic accidents combined. Cigarettes, it turns out, are a greater cause of global death than bullets. Such telling statistics animate Robert Proctor's new book. Proctor's treatment of the cigarette catastrophe makes no pretense to scholarly detachment or academic neutrality. That is not to say it is somehow *less* scholarly – far from it: Proctor's extensive use of previously secret tobacco industry documents makes his case convincing, even compelling. His narrative couples careful, thoroughly documented, analysis of the history of cigarette design, cigarette marketing and cigarette science with a moral outrage that is almost palpable. This outrage inflects every one of the book's seven hundred plus pages – from the design of ‘coffin-nails’ to the deceptive tactics of the industry ‘smoke folk’ – and motivates Proctor's call for the abolition of the tobacco industry. Much of the terrain that Proctor covers, if not his ultimate conclusion, will be familiar to scholars of tobacco control and public health, especially readers of Stanton Glantz, Richard Kluger and Allan Brandt. But the extent to which Proctor engages the Legacy Tobacco Documents, the comprehensive treatment given to the history and future of the tobacco epidemic and the grip this book keeps on its reader make *Golden Holocaust* very worthwhile...

Ultimately, while Proctor's message is clearly impassioned, it is also thoroughly considered at every turn. In a note defending his use of the term ‘holocaust’ in the title, for example, Proctor explains that although there are clear differences between the murder of six million Jews under the Nazi regime and the suffering of smokers due to tobacco, in both instances we see a calamity of epic proportions with too many idle bystanders failing to intervene. ‘In polite society’, he goes on: ‘we tend to trade in euphemisms, but when the truth itself is outrageous, weak words can falsify the realities of needless, outrageous sufferings’ (p. 11). Proctor's words are anything but weak, his outrage at the needless suffering caused by the tobacco industry, clear. In drawing attention to the evils perpetrated by the tobacco industry over the last half-century Proctor has made his intervention. His history of the origins of the cigarette catastrophe and the case he makes for its abolition are convincing, and will surely be welcomed by the global health community.

http://www.tandfonline.com/doi/abs/10.1080/17441692.2012.717961

Quantification of AgNOR expression in exfoliated oral mucosal cells of tobacco chewers with and without lesion

Indian J Dent Res. 2012 Mar;23(2):251-6.

Sharma A, Saxena S.

Abstract
Background: Nucleolar organizer regions (NOR) are associated with proliferative activity and represent a diagnostic and prognostic marker. Materials and Methods: Smears were taken from smokers, tobacco chewers, oral squamous cell carcinoma patients, and normal subjects and evaluated by 2 silver-staining nucleolar organizer region (AgNOR) counting methods: (1) mean number of AgNORs per nucleus (mAgNOR); and (2) percentage of nuclei with >3 and >5 AgNORs (pAgNOR). Results: A statistically significant difference was observed between normal subjects, smokers, tobacco chewers, and oral cancer patients and between tobacco chewers with and without lesion. No significant difference was observed between tobacco chewers and smokers except in the percentage of >5 criteria. Conclusions: AgNOR enumeration using noninvasive methods, such as the cytobrush appears to be useful technique in distinguishing between normal mucosa, mucosa with and without lesions exposed to carcinogens, such as tobacco and frank oral carcinoma.

http://www.ijdr.in/article.asp?issn=0970-9290;year=2012;volume=23;issue=2;spage=251;epage=256;aulast=Sharma

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Smokeless tobacco and risk of head and neck cancer: Evidence from a case-control study in New England

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: **14 SEP 2012**

Jiachen Zhou, Dominique S. Michaud, Scott M. Langevin, Michael D. McClean, Melissa Eliot and Karl T. Kelsey

Abstract
Current studies suggesting that smokeless tobacco use increases the risk of head and neck cancer are hampered by small numbers. Consequently, there remains uncertainty in the magnitude and significance of this risk. We examined the relationship between smokeless tobacco use and head and neck squamous cell carcinoma (HNSCC); in a population-based case-control study with 1,046 cases and 1,239 frequency-matched controls. Logistic regression models were used to estimate odds ratios (OR); and 95% confidence intervals (95% CI);, adjusting for age, gender, race, education level, cigarette smoking, and alcohol consumption. A non-significant elevated association between having ever used smokeless tobacco and HNSCC risk (OR = 1.20, 95% CI: 0.67, 2.16); was observed. Individuals who reported 10 or more years of smokeless tobacco use had a significantly elevated risk of HNSCC (OR = 4.06, 95% CI: 1.31, 12.64);, compared to never users. In an analysis restricted to never cigarette smokers, a statistically significant association was observed between ever use of smokeless tobacco and the risk of HNSCC (OR = 4.21, 95% CI: 1.01, 17.57);. These findings suggest that long term use of smokeless tobacco increases the risk of HNSCC.

http://onlinelibrary.wiley.com/doi/10.1002/ijc.27839/abstract

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No causal effect of unemployment on smoking? A German panel study

Int J Public Health. 2012 Sep 4. [Epub ahead of print]

Schunck R, Rogge BG.

Abstract

OBJECTIVES:

This study analyses the effects of different unemployment durations on smoking behaviour in Germany by investigating smoking take-up, relapse, quitting and smoking intensity.

METHODS:

Longitudinal data from the German Socio-Economic Panel from the years 1998, 2001, 2002, 2004, 2006, and 2008 were used to examine the effect of unemployment (52,940 observations from 17,028 respondents, aged 17-65 years). Unemployment duration was measured at 1-6, 7-12, 13-24, and 24+ months. Effects were estimated using zero-inflated negative binomial regressions and fixed effects logistic panel regressions.

RESULTS:

The zero-inflated negative binomial regression models suggest that the likelihood of smoking increases with unemployment, while smoking intensity is not affected. However, fixed effects logistic regression models demonstrate that unemployment is neither a significant predictor for taking up smoking, relapsing, nor quitting.

CONCLUSIONS:

The results indicate that in Germany, there is no direct causal effect of unemployment on smoking behaviour. The observed relationship between smoking and unemployment appears to be driven by stable, unobserved differences between employed and unemployed respondents.

http://www.springerlink.com/content/b202803p5vw33750/

Environmental tobacco smoke and periodontitis in U.S. Non-smokers

J Dent Hyg. 2012;86(3):185-94. Epub 2012 Aug 27.

Sutton JD, Ranney LM, Wilder RS, Sanders AE.

Abstract

PURPOSE:

The association of second hand smoke or environmental tobacco smoke (ETS) and periodontitis in non-smokers has not been confirmed using a biomarker of ETS exposure. To estimate periodontitis prevalence in non-smokers with detectable serum cotinine, and to investigate racial/ethnic and socioeconomic variation in ETS exposure in a representative sample of the U. S. adult population. Determining periodontitis risk indicators occurring with ETS appears to be a salient purpose as this study is the first of its kind to provide a link (a salivary biomarker) between second hand smoke and risk for periodontitis.

METHODS:

Data were collected from the 1999 to 2004 National Health and Nutrition Examination Survey (NHANES). Subjects were 3,137 adults who had smoked fewer than 100 cigarettes and had not used other forms of tobacco. ETS exposure was classified as negligible (cotinine concentrations below sex and race/ethnicity cut-points for smokers), moderate (cotinine 0.5-<1.5 µg/mL) or high (cotinine≥1.5 ng/mL). Periodontitis was classified according to the Centers for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) case defini tion for moderate-severe disease. Survey estimation procedures were used to estimate prevalence and odds ratios (OR) were from multivariable logistic regression models.

RESULTS:

ETS exposure was observed in 40.5% of subjects and 2.6% had periodontitis. ETS exposure was inversely associated with educational attainment and family income and was higher in non-Hispanic blacks than whites. After adjusting for age, sex and year of survey, adults with high ETS exposure (cotinine≥1.5 ng/mL) had more than twice the odds of periodontitis as people with negligible exposure (OR=2.3, 95% confidence interval=1.3, 4.1).

CONCLUSION:

High ETS exposure was a risk indicator for periodontitis in lifetime non-smokers.

http://adha.publisher.ingentaconnect.com/content/adha/jdh/2012/00000086/00000003/art00005

Asthma and its Relation to Smoking Behavior and Cessation Motives among Adult Daily Smokers

J Health Psychol. 2012 Sep 4. [Epub ahead of print]

Avallone KM, McLeish AC, Zvolensky M, Kraemer KM, Luberto CM, Jeffries ER.

Abstract

Despite the negative effects of smoking on lung functioning and overall health, smoking is more prevalent among individuals with asthma compared to those without asthma. The purpose of this study was to examine the predictive ability of asthma diagnosis in terms of smoking behavior and reasons for quitting. Participants were 251 regular daily smokers: 125 smokers with self-reported, physician-diagnosed asthma and 126 smokers without asthma. Asthma diagnosis significantly predicted age of regular smoking onset, number of quit attempts, and reasons for quitting related to self-control suggesting that smokers with asthma may have more difficulty quitting and unique reasons for quitting.

http://hpq.sagepub.com/content/early/2012/09/04/1359105312456322.abstract

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Can a Minimal Intervention Reduce Secondhand Smoke Exposure Among Children with Asthma from Low Income Minority Families? Results of a Randomized Trial

J Immigr Minor Health. 2012 Sep 4. [Epub ahead of print]

Streja L, Crespi CM, Bastani R, Wong GC, Jones CA, Bernert JT, Tashkin D, Hammond SK, Berman BA.

Abstract

We report on the results of a low-intensity behavioral intervention to reduce second hand smoke (SHS) exposure of children with asthma from low income minority households in Los Angeles, California. In this study, 242 child/adult dyads were randomized to a behavioral intervention (video, workbook, minimal counseling) or control condition (brochure). Main outcome measures included child's urine cotinine and parental reports of child's hours of SHS exposure and number of household cigarettes smoked. Implementation of household bans was also considered. No differences in outcomes were detected between intervention and control groups at follow-up. Limitations included high attrition and low rates of collection of objective measures (few children with urine cotinine samples). There continues to be a need for effective culturally and linguistically appropriate strategies that support reduction of household SHS exposure among children with asthma in low income, minority households.

http://www.springerlink.com/content/11022t785442xm55/

Online advertising to reach and recruit Latino smokers to an internet cessation program: impact and costs

J Med Internet Res. 2012 Aug 27;14(4):e116.

Graham AL, Fang Y, Moreno JL, Streiff SL, Villegas J, Muñoz RF, Tercyak KP, Mandelblatt JS, Vallone DM.

Abstract

BACKGROUND:

Tobacco cessation among Latinos is a public health priority in the United States, particularly given the relatively high growth of this population segment. Although a substantial percentage of American Latinos use the Internet, they have not engaged in Web-based cessation programs as readily as other racial/ethnic subgroups. A lack of culturally specific advertising efforts may partly explain this disparity.

OBJECTIVE:

Phase I of this study focused on the development of four Spanish-language online banner advertisements to promote a

free Spanish-language smoking cessation website (es.BecomeAnEX.org). Phase II examined the relative effectiveness of the four banner ads in reaching and recruiting Latino smokers to the cessation website.

METHODS:

In Phase I, 200 Spanish-speaking Latino smokers completed an online survey to indicate their preference for Spanish-language banner ads that incorporated either the cultural value of family (familismo) or fatalism (fatalismo). Ads included variations on message framing (gain vs loss) and depth of cultural targeting (surface vs deep). In Phase II, a Latin square design evaluated the effectiveness of the four preferred ads from Phase I. Ads were systematically rotated across four popular Latino websites (MySpace Latino, MSN Latino, MiGente, and Yahoo! en Español) over four months from August to November 2009. Tracking software recorded ad clicks and registrants on the cessation website. Negative binomial regression and general linear modeling examined the main and interacting effects of message framing and depth of cultural targeting for four outcomes: number of clicks, click-through rate, number of registrants, and cost per registrant.

RESULTS:

In Phase I, smokers preferred the four ads featuring familismo. In Phase II, 24,829,007 impressions were placed, yielding 24,822 clicks, an overall click-through rate of 0.10%, and 500 registrants (2.77% conversion rate). Advertising costs totaled US $104,669.49, resulting in an overall cost per click of US $4.22 and cost per registrant of US $209.34. Website placement predicted all four outcomes (all P values < .01). Yahoo! en Español yielded the highest click-through rate (0.167%) and number of registrants (n = 267). The message framing and cultural targeting interaction was not significant. Contrary to hypotheses, loss-framed ads yielded a higher click-through rate than gain-framed ads (point estimate = 1.08, 95% CI 1.03 1.14, P = 0.004), and surface-targeted ads outperformed deep-targeted ads for clicks (point estimate = 1.20, 95% CI 1.13 1.28, P < .001), click-through rate (point estimate = 1.22, 95% CI 1.16 1.29, P < .001), and number of registrants (point estimate = 2.73, 95% CI 2.14 3.48, P < .001).

CONCLUSIONS:

Online advertising can be an effective and cost-efficient strategy to reach and engage Spanish-speaking Latino smokers in an evidence-based Internet cessation program. Cultural targeting and smoking-relevant images may be important factors for banner ad design. Online advertising holds potential for Web-based cessation program implementation and research.

http://www.jmir.org/2012/4/e116/

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

Tobacco control and trade policy: Proactive strategies for integrating policy norms

J Public Health Policy. 2012 Sep 6. doi: 10.1057/jphp.2012.36. [Epub ahead of print]

Drope J, Lencucha R.

Abstract

Palpable tension continues at the intersection of tobacco control and trade policy. Through consideration of four major tobacco control-related trade disputes, we suggest how to empower public health proponents in the face of entrenched economic policymaking norms. We argue that a more effective pro-tobacco control message should: (a) seek to be broadly consistent with core principles of the world trading system, (b) boldly assert countries' international commitments to the Framework Convention on Tobacco Control, (c) marshal deep scientific evidence, and (d) come from a broad range of actors, including from low- and middle-income countries as well as from other trade policy community members.

http://www.palgrave-journals.com/jphp/journal/vaop/ncurrent/full/jphp201236a.html

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Tobacco control campaign in Uruguay: a population-based trend analysis

The Lancet
Available online **13 September 2012**

Winston Abascal, Elba Esteves, Beatriz Goja, Franco González Mora, Ana Lorenzo, Amanda Sica, Patricia Triunfo, Jeffrey E Harris

Summary

Background In 2005, Uruguay initiated a series of comprehensive anti-smoking measures. We aimed to assess the effect of Uruguay's anti-tobacco campaign.

Methods

We did a population-based trend analysis, using neighbouring Argentina, which has not instituted such extensive anti-tobacco measures, as a control. We assessed three key endpoints in both countries: per-person consumption of cigarettes, as measured by tax records; the prevalence of tobacco use in adolescents, as measured by school-based surveys; and the prevalence of tobacco use in adults, as measured by nationwide household-based surveys.

Findings

During 2005–11, per-person consumption of cigarettes in Uruguay decreased by 4·3% per year (95% CI 2·4 to 6·2), whereas per-person consumption in Argentina increased by 0·6% per year (–1·2 to 2·5; p=0·002 for difference in trends). During 2003–09, the 30-day prevalence of tobacco use in Uruguayan students aged 13 years, 15 years, and 17 years decreased by an estimated 8·0% per year (4·5 to 11·6), compared with a decrease of 2·5% annually (0·5 to 4·5) in Argentinian students during 2001–09 (p=0·02 for difference in trends). From 2005 to 2011, the prevalence of current tobacco use in Uruguay decreased annually by an estimated 3·3% (2·4 to 4·1), compared with an annual decrease in Argentina of 1·7% (0·8 to 2·6; p=0·02 for difference in trends).

Interpretation

Uruguay's comprehensive tobacco-control campaign has been associated with a substantial, unprecedented decrease in tobacco use. Decreases in tobacco use in other low-income and middle-income countries of the magnitude seen in Uruguay would have a substantial effect on the future global burden of tobacco-related diseases.

http://www.sciencedirect.com/science/article/pii/S0140673612608265

Related *Lancet* Comment:

Tobacco control: learning from Uruguay
http://www.sciencedirect.com/science/article/pii/S014067361261143X

BUTT SERIOUSLY
It is time for WA to stub out any obstacles that lie in the way of a smoke-free society says Professor Mike Daube

Medicus Journal August 2012

The High Court decision on tobacco plain packaging is a massive win for public health, a massive defeat for the tobacco industry, and – as international reactions have shown – a massive boost for tobacco control around the world. We should celebrate – but we cannot a!ord to be complacent. There is much work yet to be done. Smoking is declining in Western Australia as in other states. Health Department figures show that only 11.1 per cent of adult Western Australians over 16 smoke daily and 4.8 per cent of 12–17 year-olds are weekly smokers. Anyone under 21 has grown up without exposure to direct tobacco advertising. The introduction of plain packaging in December will further reduce the attraction of smoking – which is why it has been so ferociously opposed by Big Tobacco.

But international experience in tobacco control shows that complacency can halt the decline...

WA has a long tradition of leadership in tobacco control, led by medical and health groups and governments of all parties. It is time to reclaim that leadership both in the aspiration to a smoke-free society and in the action that will take us there.

http://www.amawa.com.au/newsmedia/medicusjournal.aspx
http://www.acosh.org/resources/Butt-Seriously AUGUST-MEDICUS.pdf

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

Caffeine drinking, cigarette smoking, and dopaminergic replacement therapy dose in Parkinson's disease

Neurol Sci. 2012 Sep 7. [Epub ahead of print]

Ojeda-López C, Cervantes-Arriaga A, Rodríguez-Violante M, Corona T.

Abstract

The objective of this study is to assess the effect of smoking and caffeine intake in the dosage of dopaminergic replacement therapy. Patients were recruited from the movement disorders clinic of the National Institute of Neurology and Neurosurgery in Mexico City. An interviewer-administered structured questionnaire was given to all subjects regarding their smoking and caffeine drinking habits. Dopaminergic replacement therapy information was collected and levodopa, dopamine agonists, and levodopa equivalent daily doses were calculated. 146 Parkinson's disease patients (50 % female) were included. All patients were on antiparkinsonian treatment, with a mean levodopa equivalent daily dose (LEDD) of 550.2 ± 408. Patients were stratified according to smoking and caffeine drinking status. 104 (71.2 %) of the patients were "never smokers", 33 (22.6 %) were "former smokers" and 9 (6.2 %) were "current smokers". 40 (27.4 %) patients reported no history of caffeine intake, 36 (24.7 %) were former consumers and 70 (47.9 %) were current caffeine drinkers. No association between LEDD and smoking or caffeine intake was found. A weak positive correlation ($r = 0.22$, $p < 0.04$) was found between the daily dose of pramipexole and the daily intake of caffeine. LEDD, levodopa daily dose and dopamine agonist daily dose were not related to smoking or caffeine intake status. We found a weak correlation between caffeine daily intake and pramipexole dose. Further prospective exploration is needed to address the interaction of concomitant A2A antagonism induced by caffeine intake and dopaminergic replacement therapy.

http://www.springerlink.com/content/h36852x02r805g62/

Socioeconomic inequality in smoking in low-income and middle-income countries: results from the World Health Survey

PLoS One. 2012;7(8):e42843. Epub 2012 Aug 29.

Hosseinpoor AR, Parker LA, Tursan d'Espaignet E, Chatterji S.

Abstract

OBJECTIVES:

To assess the magnitude and pattern of socioeconomic inequality in current smoking in low and middle income countries.

METHODS:

We used data from the World Health Survey [WHS] in 48 low-income and middle-income countries to estimate the crude prevalence of current smoking according to household wealth quintile. A Poisson regression model with a robust variance was used to generate the Relative Index of Inequality [RII] according to wealth within each of the countries studied.

RESULTS:

In males, smoking was disproportionately prevalent in the poor in the majority of countries. In numerous countries the poorest men were over 2.5 times more likely to smoke than the richest men. Socioeconomic inequality in women was more varied showing patterns of both pro-rich and pro-poor inequality. In 20 countries pro-rich relative socioeconomic inequality was statistically significant: the poorest women had a higher prevalence of smoking compared to the richest women. Conversely, in 9 countries women in the richest population groups had a statistically significant greater risk of smoking compared to the poorest groups.

CONCLUSION:

Both the pattern and magnitude of relative inequality may vary greatly between countries. Prevention measures should address the specific pattern of smoking inequality observed within a population.

http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0042843

Also:

Cigarette Smoking Decreases Global MicroRNA Expression in Human Alveolar Macrophages
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0044066
Nicotine Promotes Proliferation of Human Nasopharyngeal Carcinoma Cells by Regulating α7AChR, ERK, HIF-1α and VEGF/PEDF Signaling
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0043898
CYP1A1 Ile462Val Polymorphism Contributes to Lung Cancer Susceptibility among Lung Squamous Carcinoma and Smokers: A Meta-Analysis
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0043397

Note: Open Access. Full text PDFs freely available from links immediately above.

Nicotine differentially modulates antisaccade eye-gaze away from emotional stimuli in nonsmokers stratified by pre-task baseline performance

Psychopharmacology (Berl). 2012 Sep 7. [Epub ahead of print]

Wachter NJ, Gilbert DG.

Abstract

RATIONALE AND OBJECTIVE:

Studies indicate that nicotine enhances some aspects of attention and executive functioning and attenuates the attentional salience of emotionally negative distractors. The purpose of this study was to assess whether nicotine can enhance executive control over prepotent responses in emotional contexts in nonsmokers and whether such enhancement is greater in individuals with low baseline performance (BP).

METHODS:

The antisaccade task (AST) measures the inhibition of the tendency to glance in the direction of the onset of a visual stimulus and thus is an index of control over prepotent responses. Ten male and 14 female nonsmokers wore nicotine and placebo patches on counterbalanced days that included emotional picture primes and targets.

RESULTS:

There were significant beneficial effects of nicotine on antisaccade reaction time (RT). These beneficial effects occurred in individuals with poor and average BP, but not in high baseline performers. In slow baseline RT individuals, nicotine reduced RTs associated with negative targets in the left visual field (VF) and reduced RTs associated with positive and neutral targets in the right VF. In contrast, in the average baseline group, nicotine reduced RTs for positive targets in both VFs and neutral targets in the left VF.

CONCLUSIONS:

The results suggest that nicotine may produce its effects by enhancing executive functions and that the differential effects as a function of VF, target emotion, and group may also reflect lateralized differences in the effects of nicotine on brain reactivity to emotional stimuli.

http://www.springerlink.com/content/78h088w608063w86/

In vitro models for assessing the potential cardiovascular disease risk associated with cigarette smoking

Toxicol In Vitro. 2012 Aug 23. [Epub ahead of print]

Fearon IM, Gaça MD, Nordskog BK.

British American Tobacco, Group R & D, Regents Park Road, Southampton, UK.

Abstract

Atherosclerotic cardiovascular disease is a prevalent human disorder and a significant cause of human morbidity and mortality. A number of risk factors may predispose an individual to developing atherosclerosis, and of these factors, cigarette smoking is strongly associated with the development of cardiovascular disease. Current thinking suggests that exposure to toxicants found in cigarette smoke may be responsible for this elevated disease likelihood, and this gives rise to the idea that reductions in the levels of some smoke toxicants may reduce the harm associated with cigarette smoking. To assess the disease risk of individuals who smoke cigarettes with altered toxicant levels, a weight-of-evidence approach is required examining both exposure and disease-related endpoints. A key element of such an assessment framework are data derived from the use of in vitro models of cardiovascular disease, which when considered alongside other forms of data (e.g. from clinical studies) may support evidence of potential reduced risk. Importantly, such models may also be used to provide mechanistic insight into the effects of smoking and of smoke toxicant exposure in cardiovascular disease development. In this review the use of in vitro models of cardiovascular disease and one of the contributory factors, oxidative stress, is discussed in the context of assessing the risk potential of both conventional and modified cigarettes. Practical issues concerning the use of these models for cardiovascular disease understanding and risk assessment are highlighted and areas of development necessary to enhance the power and predictive capacity of in vitro disease models in risk assessment are discussed.

Summary

Cigarette smoking poses a substantial risk to cardiovascular health, a risk which could potentially be reduced by the production of modified risk tobacco products with altered toxicant yields. Any reduction in risk needs to be substantiated using a framework of pre-clinical and clinical studies designed to characterise the modified risk and a pivotal component of this framework is the use of in vitro models. This was further emphasised with the recent report by the Institute of Medicine (2012) examining the scientific standards for studies on modified risk tobacco products. While our knowledge of how these in vitro models operate is strong, furthern development is necessary in areas such as cellular metabolic capacity, cell-to-cell interactions, co-culture models, flow-based vs. static models and exposure systems. The use of in silico modelling as a predictive tool is also a potential area for future exploration. Finally, despite the large number of in vitro models described in this review and elsewhere, none are fully validated or currently required in regulatory testing. This highlights the need to validate and standardise methods for in vitro disease models, not only of cardiovascular disease but also of other smoking-related diseases.

Conflict of interest statement

Ian M. Fearon and Marianna D. Gaça are employees of British American Tobacco Group Research and Development. Brian K. Nordskog is an employee of R.J. Reynolds Tobacco. IMF and MDG hold stock in their employer's Company.

http://www.sciencedirect.com/science/article/pii/S0887233312002251

Note: Tobacco industry research.

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