Fattori di rischio del carcinoma orale

Elisabetta MERIGO

AMBULATORIO DI PATOLOGIA E CHIRURGIA ORALE LASER

UNIVERSITA' DI PARMA

Fattore di rischio

Agente in grado di determinare un aumento del rischio di ammalare di cancro in soggetti esposti a tale agente rispetto a quelli non esposti

National Cancer Institute

at the National Institutes of Health

Questions About Cancer?
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NCI Director Harold E. Varmus, M.D.

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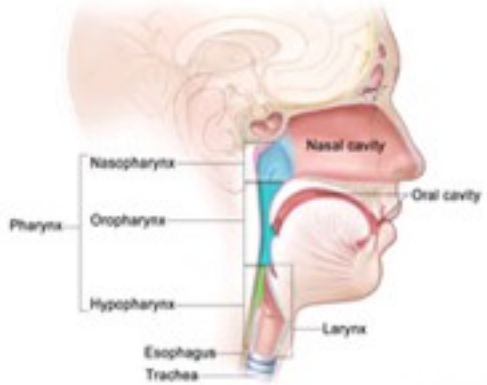
Priorities for the nation's investment in cancer research

Provocative Questions Project
Explore provocative research questions with the research community

NCI Budget Overview
Explore how NCI allocates appropriated funds



Head and Neck Cancers: Are You at Risk?

Head and neck cancer includes cancers of the mouth, nose, throat, sinuses, and salivary glands. At least 75 percent of these cancers are caused by tobacco (including smokeless tobacco) and alcohol use. HPV infection is another risk factor. If found early, these cancers are often more treatable.

- NCI Factsheet: Head and Neck Cancers
- Treatment information for head and neck cancers
- Snapshot of NCI's investment in head and neck cancer research

Head and Neck Cancers: Are You at Risk?

SEER 2012 Update: New Data on Cancer Trends

Reducing Cancer Health Disparities

Types of Cancer

Common Cancer Types

Bladder Cancer
Breast Cancer
Colon and Rectal Cancer
Endometrial Cancer
Kidney (Renal Cell) Cancer
Leukemia
Lung Cancer
Melanoma
Non-Hodgkin Lymphoma
Pancreatic Cancer
Prostate Cancer
Thyroid Cancer

All Cancer Types

A to Z List of Cancers
Cancers by Body Location/System
Childhood Cancers
Adolescents and Young Adults
Women's Cancers

Cancer Research News

Stanford study shows anxiety increases cancer severity in mice

Mayo Clinic identifies gene critical to development and spread of lung cancer

St. Jude study finds protein prevents DNA damage in the developing brain and might serve as a tumor suppressor

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NCI Cancer Bulletin

NATIONAL CANCER INSTITUTE
FactSheet

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Cancer Advances In Focus Index
En español

Head and Neck Cancers

Key Points

- Most head and neck cancers begin in the squamous cells that line the moist surfaces inside the head and neck.
- Tobacco use, alcohol use, and human papillomavirus infection are important risk factors for head and neck cancers.
- Typical symptoms of head and neck cancers include a lump or sore (for example, in the mouth) that does not heal, a sore throat that does not go away, difficulty swallowing, and a change or hoarseness in the voice.
- Rehabilitation and regular follow-up care are important parts of treatment for patients with head and neck cancers.

1. What are cancers of the head and neck?

Cancers that are known collectively as head and neck cancers usually begin in the squamous cells that line the moist, mucosal surfaces inside the head and neck (for example, inside the mouth, the nose, and the throat). These squamous cell cancers are often referred to as squamous cell carcinomas of the head and neck. Head and neck cancers can also begin in the salivary glands, but salivary gland cancers are relatively uncommon. Salivary glands contain many different types of cells that can become cancerous, so there are many different types of salivary gland cancer.

Cancers of the head and neck are further categorized by the area of the head or neck in which they begin. These areas are labeled in the figure below and listed in the table included in the answer to Question 3.

NATIONAL CANCER INSTITUTE
FactSheet

Reviewed: 04/17/2012

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Fattori di rischio per il cancro orale

- Fumo
- Masticare tabacco
- Alcool
- Abuso di collutori alcoolici
- HPV 16
- Dieta
- Esposizione al sole
- Noce di areca con/senza tabacco
- Gutka
- Pan masala
- Mate

Oral Oncology 45 (2009) 340–350

Contents lists available at ScienceDirect

Oral Oncology

journal homepage: www.elsevier.com/locate/oraloncology

Review

Lifestyle risk factors for oral cancer

Stefano Petti *

Department of Public Health Sciences "G. Sanarelli", "Sapienza" University, P.le Aldo Moro 5, 00185 Rome, Italy

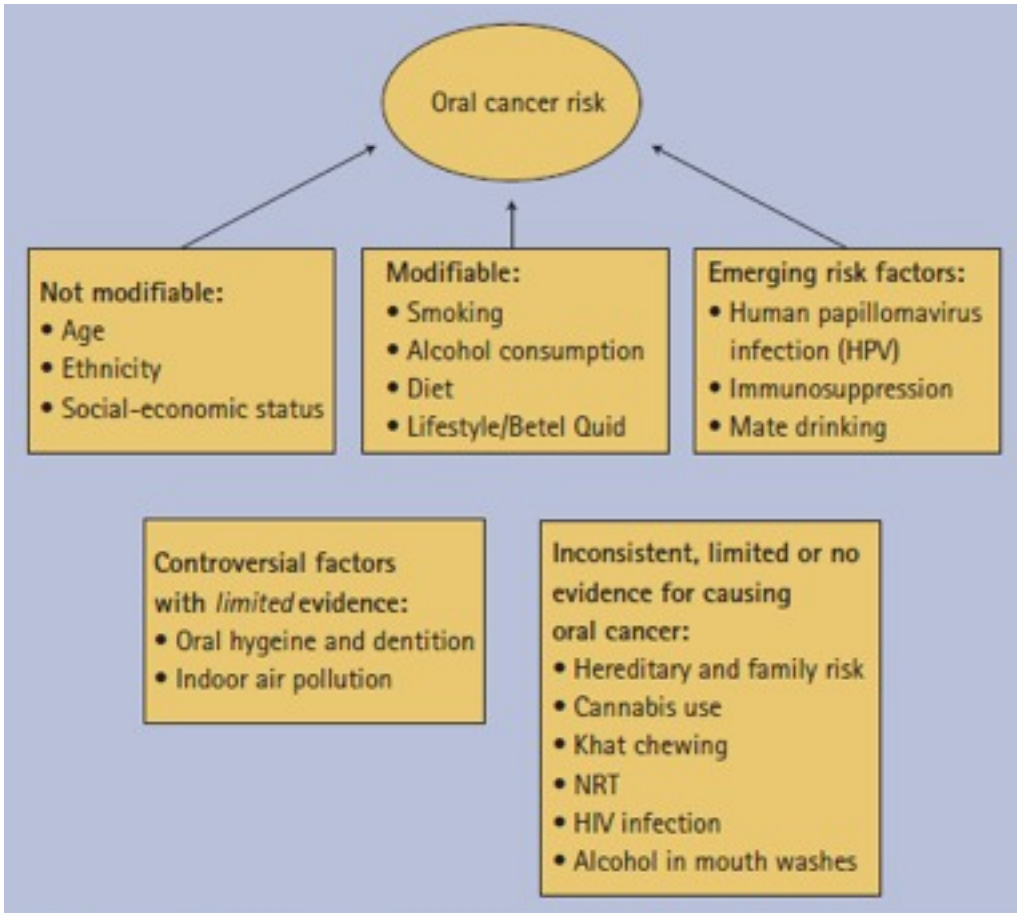
Oral cancer, lifestyle and public health

Most oral cancer cases and deaths are due to both individual predisposition, linked to specific genetic characteristics, and exposure to carcinogens, caused by lifestyle behaviours. Specifically, 20–30% overall cases are attributable to tobacco/bidi smoking,[11,54,111] 50% (men) and almost 90% (women) cases to frequent betel quid without tobacco chewing in areas where chewing prevalence is particularly high,[54] 7–19% cases to heavy alcohol drinking,[11,79] 10–15% cases to micronutrient deficiency,[76,78] and also 3% cases to human papillomavirus infection, generally (but not exclusively) associated to sexual behaviour.[112] In addition, exposure to two or more of these factors has a synergistic effect in increasing oral cancer risk.[18,19,95]

Causes of oral cancer – an appraisal of controversies

S. Warnakulasuriya[1]

BRITISH DENTAL JOURNAL VOLUME 207 NO. 10 NOV 28 2009



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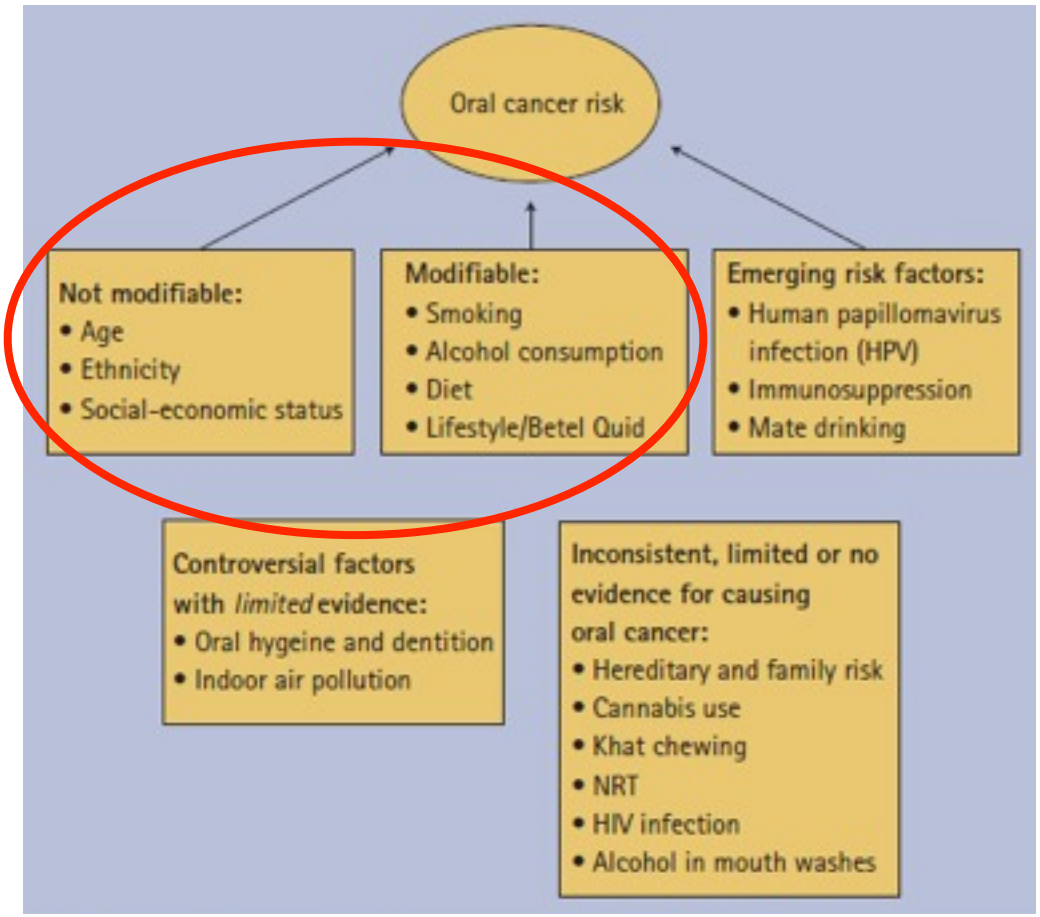
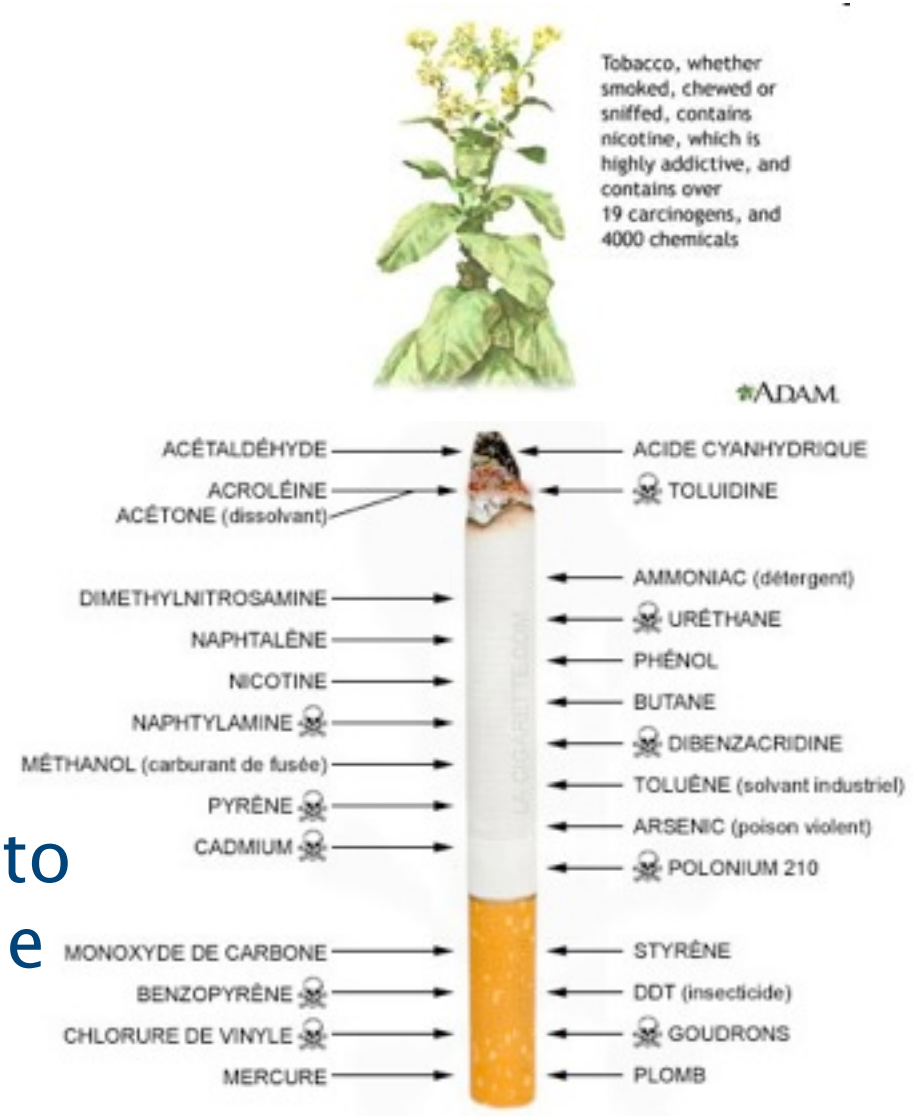


Fattori di rischio per il cancro orale – Fumo

- Il tabacco risulta carcinogeno in tutte le sue forme.
- Il tabacco genera carcinogeni quali nitrosamine e radicali liberi.
- Nei fumatori le alterazioni genetiche risultano a carico di tutto il tratto aero-digestivo e possono persistere per molti anni anche dopo l'interruzione del fumo.



American Journal of Epidemiology

Vol. 167, No. 4
DOI: 10.1093/aje/kwm318
Advance Access publication December 4, 2007

Original Contribution

Tobacco Smoking, Smoking Cessation, and Cumulative Risk of Upper Aerodigestive Tract Cancers

Cristina Bosetti[1], Silvano Gallus[1], Richard Peto[2], Eva Negri[1], Renato Talamini[3], Alessandra Tavani[1], Silvia Franceschi[4], and Carlo La Vecchia[1,5]

[1] Istituto di Ricerche Farmacologiche "Mario Negri," Milan, Italy.
[2] Clinical Trial Service Unit and Epidemiological Studies Unit, University of Oxford, Oxford, United Kingdom.
[3] Unità di Epidemiologia e Biostatistica, Centro di Riferimento Oncologico di Aviano, Aviano, Italy.
[4] International Agency for Research on Cancer, Lyon, France.
[5] Istituto di Statistica Medica e Biometria "G. A. Maccacaro," Università degli Studi di Milano, Milan, Italy.

	All upper aerodigestive tract sites				Oral cavity and pharynx			
	No. of cases	No. of controls	OR*,†	95% CI*	No. of cases	No. of controls	OR†	95% CI
Never smokers	87	943	0.12	0.09, 0.15	24	701	0.07	0.05, 0.11
Current smokers‡	1,514	1,476	1.00		712	1,176	1.00	
Ex-smokers								
Time since cessation (years)								
<10	338	478	0.70	0.56, 0.88	144	383	0.51	0.40, 0.65
10–19	155	489	0.58	0.47, 0.73	56	330	0.20	0.14, 0.28
≥20	95	391	0.27	0.22, 0.34	23	232	0.12	0.08, 0.20
Age at cessation (years)								
<35	40	248	0.19	0.13, 0.27	13	162	0.14	0.08, 0.26
35–44	105	391	0.26	0.21, 0.34	45	279	0.20	0.14, 0.29
45–54	215	426	0.44	0.36, 0.53	90	301	0.36	0.27, 0.48
55–64	228	293	0.69	0.56, 0.86	75	203	0.48	0.34, 0.66

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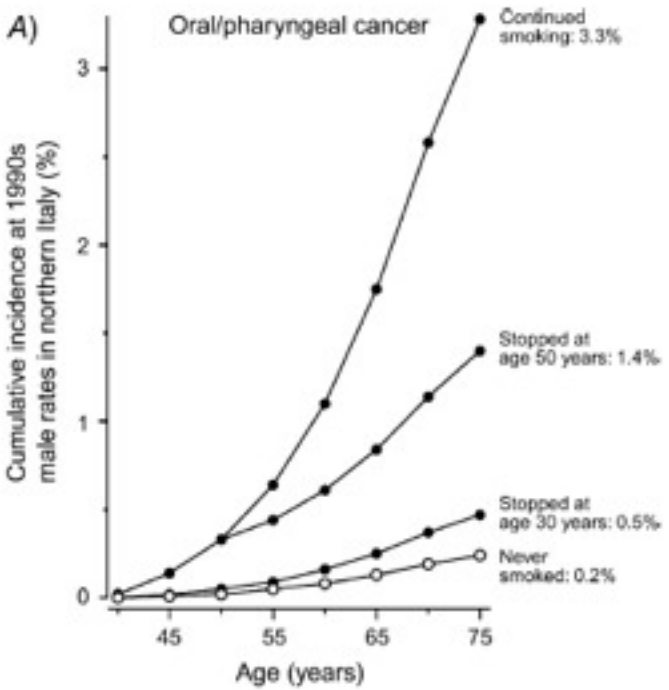
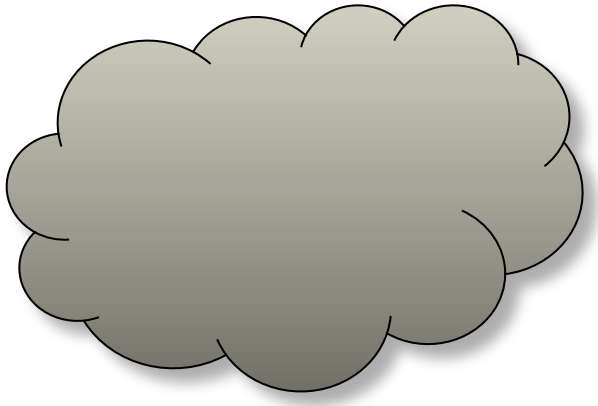
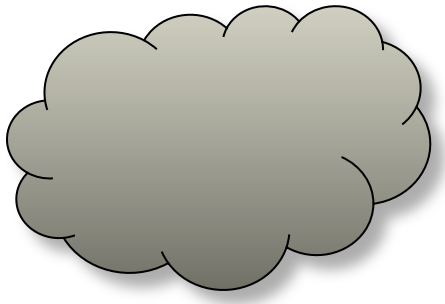
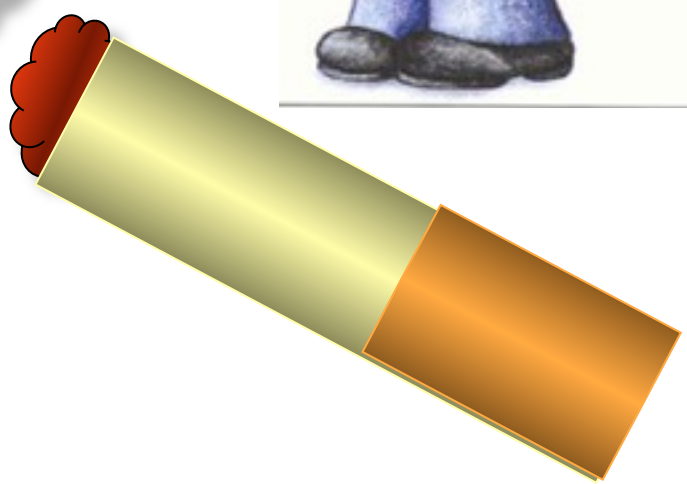


- Consumo di sigarette: per i forti fumatori (>15 sigarette/die) il rischio di sviluppare neoplasia risulta maggiore di 9 volte rispetto ai non-fumatori
- Durata dell'esposizione
- Alcool/tabacco: la cessazione diminuisce il rischio ma occorrono 10 anni per esser considerati come non fumatori/bevitori

Fumo passivo







Donne
NON FUMATRICI
spose
di MARITI FUMATORI:

PERCENTUALE
DOPPIA DI
CANCRO AL POLMONE
rispetto a quelle di
MARITI NON FUMATORI

Hirayama (Japan 1981)

BRIEF COMMUNICATION

Cancer of the Oral Cavity and Pharynx in Nonsmokers Who Drink Alcohol and in Nondrinkers Who Smoke Tobacco

Renato Talamini, Carlo La Vecchia, Fabio Levi, Ettore Conti, Adriano Favero, Silvia Franceschi

In developed countries, cancer of the oral cavity and pharynx is rare in individuals who do not smoke and do not drink alcohol. Few studies, however, have included large enough groups of case and control subjects to provide useful information on the effect of alcohol consumption in the absence of tobacco smoking and the effect of tobacco smoking in the absence of alcohol consumption *(1)*.

Table 2. Odds ratio (OR) of cancer of the oral cavity and pharynx and 95% confidence interval (CI) for alcohol-drinking nonsmokers and tobacco-smoking nondrinkers (1992–1997)

	No. of subjects			
	Case	Control	OR*	95% CI
Alcohol consumption				
Never drinkers	16	139	1†	
Current drinkers				
<21 drinks/wk	23	334	0.8	(0.4–1.6)
21–34 drinks/wk	4	112	0.8	(0.2–2.7)
35–55 drinks/wk	7	47	5.0	(1.5–16.1)
≥56 drinks/wk	3	18	5.3	(1.1–24.8)
χ_1^2 trend				6.2; $P = .01$
Ex-drinkers	7	42	2.0	(0.7–5.4)
Smoking status‡				
Never smokers	16	139	1†	
Current smokers‡				
<25 cigarettes/d	6	44	1.5	(0.5–4.6)
≥25 cigarettes/d	3	5	7.2	(1.1–46.6)
χ_1^2 trend				3.2; $P = .07$
Ex-smokers	7	33	2.2	(0.8–6.2)

*Estimates from logistic regression equations, including terms for study center, age, sex, and education.
†Reference category.
‡One control subject who smoked a pipe only and two control subjects who smoked an unknown number of cigarettes are not included.

Journal of the National Cancer Institute, Vol. 90, No. 24, December 16, 1998

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Tumori. 2010 Jan-Feb;96(1):1-10.

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Department of Environmental Medicine and Public Health, University of Padua, Italy. cristina.canova@unipd.it

Abstract

AIMS AND BACKGROUND: Each year in Italy there are approximately 14,000 new cases and 7,000 deaths from cancer of the upper aerodigestive tract, which includes malignant tumors originating from the oral cavity, pharynx, larynx and esophagus. Established etiological factors include tobacco consumption and heavy alcohol drinking. The study of single nucleotide polymorphisms in upper aerodigestive tract cancer etiology may help to identify high-risk subgroups and to better understand the pathways leading to the development of these cancers.

METHODS: Italian results on about 500 cases and 500 controls from a large case-control study (ARCAGE) conducted in 10 European countries are presented with the major objectives of updating results on the effects of alcohol and tobacco consumptions in northern Italy, investigating the role of genetic variation with regard to the metabolism of alcohol and carcinogens from tobacco smoke, and evaluating possible interactions of these single nucleotide polymorphisms with these carcinogens.

RESULTS: The present study confirmed the importance of tobacco smoking and alcohol drinking as the main risk factors for upper aerodigestive tract cancers, indicating that about 68% of cancers among populations in northern Italy can be attributed to the combination of these risk factors. Significant associations between metabolizing phase I genes (CYP1A1 and CYP2A6), phase II genes (GSTA2) and upper aerodigestive tract cancers were found. A polymorphism of ADH1C has been associated with an increased risk of upper aerodigestive tract cancers, suggesting that the less rapid alcohol metabolizers are more susceptible to upper aerodigestive tract cancer risk.

CONCLUSIONS: Our results suggest that the ADH1C allele modifies the carcinogenic dose response for alcohol in the upper aerodigestive tract, giving rise to a gene-environment interaction. The role of genes as possible modifiers of life-style risks seems the most reliable.

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Consumo alcolico a rischio (ultimi 30 giorni) Pool di Asl	
Consumatori a maggior rischio*	17%
- Consumo abituale elevato**	4%
- Consumo fuori pasto	8%
- Consumo *binge****	9%

* consumo abituale elevato e/o fuori pasto e/o *binge*.
** più di 2 unità alcoliche in media al giorno per gli uomini e più di 1 per le donne
*** consumo di 5 o più unità alcoliche (uomini) e 4 o più unità alcoliche (donne) in una unica occasione, almeno una volta negli ultimi 30 giorni

Azione diretta

- Etanolo
- Contaminanti: nitrosammine, micotossine, uretani, asbesto,derivati arsenicali)

Azione diretta co-carcinogenetica

- vasodilatazione determinante maggior contatto e penetrazione di di sostanze carcinogenetiche

Azione indiretta in epatopatia alcoolica

Oral Oncology (2004) 40 904–909

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ORAL ONCOLOGY

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Wine, beer and spirits and risk of oral and pharyngeal cancer: a case–control study from Italy and Switzerland

Andrea Altieri[a,*], Cristina Bosetti[a], Silvano Gallus[a], Silvia Franceschi[b], Luigino Dal Maso[c], Renato Talamini[c], Fabio Levi[d], Eva Negri[a], Teresa Rodriguez[e,f], Carlo La Vecchia[a,g]

Table 1 Distribution of 749 cases of oral and pharyngeal cancer and 1,772 controls, and corresponding odds ratio[a] (OR) and 95% confidence intervals (CI), according to daily consumption of different types of alcoholic beverages (Italy and Switzerland, 1992–1997)

Type (drinks/day)	Cases[b]	Controls[b]	OR	95% CI
Total alcohol				
Non-drinkers	32	223		
1–2	93	796	1	
3–4	95	365	2.1	1.5–2.9
5–7	132	208	5.0	3.5–7.1
8–11	199	118	12.2	8.4–17.6
⩾ 12	196	60	21.1	14.0–31.8
χ^2 trend (*p*-value)			272.07 (<0.0001)	
Wine[c]				
Non-wine drinkers	43	265		
1–2	110	825	1	
3–4	127	393	2.2	1.6–3.0
5–7	157	151	7.1	5.0–10.1
8–11	177	93	11.8	8.1–17.2
⩾ 12	134	43	16.1	10.2–25.3
χ^2 trend (*p*-value)			221.83 (<0.0001)	
Beer[c]				
Non-beer drinkers	284	949	1	
1–2	380	781	1.2	1.0–1.5
⩾ 3	84	41	2.3	1.4–3.7
χ^2 trend (*p*-value)			9.86 (0.02)	
Spirits[c]				
Non-spirit drinkers	297	969	1	
1–2	386	775	1.0	0.8–1.2
⩾ 3	66	27	1.9	1.1–3.3
χ^2 trend (*p*-value)			1.14 (0.29)	

Oral Oncology (2004) 40 904–909

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ORAL ONCOLOGY

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Wine, beer and spirits and risk of oral and pharyngeal cancer: a case–control study from Italy and Switzerland

Andrea Altieri[a,*], Cristina Bosetti[a], Silvano Gallus[a], Silvia Franceschi[b], Luigino Dal Maso[c], Renato Talamini[c], Fabio Levi[d], Eva Negri[a], Teresa Rodriguez[e,f], Carlo La Vecchia[a,g]

Table 1 Distribution of 749 cases of oral and pharyngeal cancer and 1,772 controls, and corresponding odds ratio[a] (OR) and 95% confidence intervals (CI), according to daily consumption of different types of alcoholic beverages (Italy and Switzerland, 1992–1997)

Type (drinks/day)	Cases[b]	Controls[b]	OR	95% CI
Total alcohol				
Non-drinkers	32	223		
1–2	93	796	1	
3–4	95	365	2.1	1.5–2.9
5–7	132	208	5.0	3.5–7.1
8–11	199	118	12.2	8.4–17.6
⩾ 12	196	60	21.1	14.0–31.8
χ^2 trend (*p*-value)			272.07 (<0.0001)	
Wine[c]				
Non-wine drinkers	43	265		
1–2	110	825	1	
3–4	127	393	2.2	1.6–3.0
5–7	157	151	7.1	5.0–10.1
8–11	177	93	11.8	8.1–17.2
⩾ 12	134	43	16.1	10.2–25.3
χ^2 trend (*p*-value)			221.83 (<0.0001)	
Beer[c]				
Non-beer drinkers	284	949	1	
1–2	380	781	1.2	1.0–1.5
⩾ 3	84	41	2.3	1.4–3.7
χ^2 trend (*p*-value)			9.86 (0.02)	
Spirits[c]				
Non-spirit drinkers	297	969	1	
1–2	386	775	1.0	0.8–1.2
⩾ 3	66	27	1.9	1.1–3.3
χ^2 trend (*p*-value)			1.14 (0.29)	

Oral Oncology 46 (2010) 720–726

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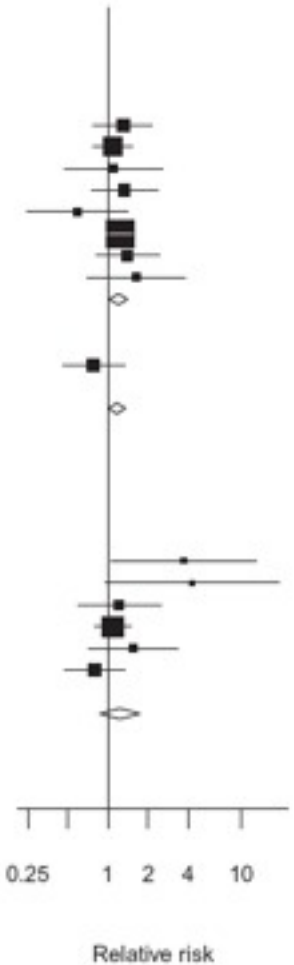
Review

A meta-analysis of alcohol drinking and oral and pharyngeal cancers. Part 2: Results by subsites

Federica Turati [a,b], Werner Garavello [a,c], Irene Tramacere [a], Vincenzo Bagnardi [d,e], Matteo Rota [d,f], Lorenza Scotti [d], Farhad Islami [g,h], Giovanni Corrao [d], Paolo Boffetta [i,j], Carlo La Vecchia [a,b,*], Eva Negri [a]

Study	Sex	Cancer Cases <=1 drink/non- or occasional	RR	95% CI
Oral cavity				
Case-control studies				
Bross and Coombs, 1976	W	53/74	1.30	0.78- 2.16
Graham et al, 1977*	M	102/91	1.09	0.77- 1.54
Elwood et al, 1984§	Both	25/39	1.10	0.47- 2.56
Zheng et al, 1990	M	42/83	1.33	0.75- 2.37
Choi and Kahyo, 1991	M	9/16	0.59	0.25- 1.40
Znaor et al, 2003	M	213/780	1.23	0.98- 1.54
Applebaum et al, 2007	Both	41/34	1.40	0.81- 2.42
Subapriya et al, 2007§	Both	33/299	1.62	0.69- 3.80
All case-control studies			1.20	1.03- 1.40
Cohort studies				
Boffetta and Garfinkel, 1990	M	16/55	0.78	0.46- 1.33
All studies			1.17	1.01- 1.35
p for heterogeneity=0.620				
Pharynx				
Case-control studies				
Elwood et al, 1984§	Both	18/9	3.70	1.05-13.02
Choi and Kahyo, 1991	W	4/14	4.30	0.97-19.11
Choi and Kahyo, 1991	M	20/16	1.22	0.60- 2.49
Znaor et al, 2003	M	70/297	1.09	0.80- 1.49
Vlajinac et al, 2006	Both	39/19	1.55	0.72- 3.34
Applebaum et al, 2007	Both	36/38	0.80	0.48- 1.34
All studies			1.23	0.87- 1.73
p for heterogeneity=0.152				



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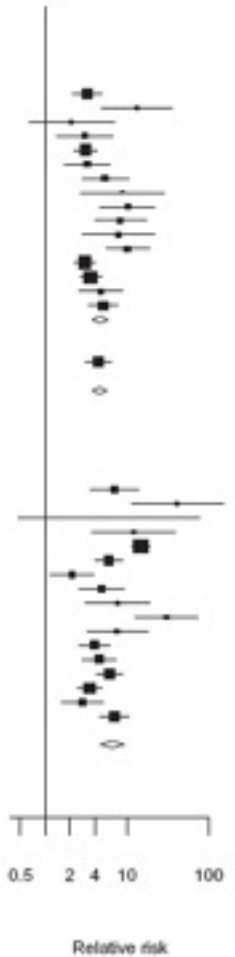
Study	Sex	Cancer Cases >=4 drinks/non- or occasional	RR	95% CI
Oral cavity				
Case–control studies				
Wynder and Bross, 1957*	M	245/126	3.30	2.22– 4.89
Vincent and Marchetta, 1963*	Both	23/7	13.14	4.91– 35.16
Martinez, 1969#	M	7/14	2.10	0.83– 5.29
Elwood et al, 1984§	Both	50/39	3.01	1.37– 6.60
Brugere et al, 1986#	M	–	3.15	2.24– 4.31
Franceschi et al, 1990#	M	128/15	3.28	1.73– 6.21
Choi and Kaliyo, 1991	M	43/16	5.40	2.81– 10.37
Oreggia et al, 1991	M	39/4	8.80	2.75– 28.16
Boffetta et al, 1992#	M	170/7	10.11	4.72– 21.66
Bundgaard et al, 1994	Both	44/91	6.40	4.00– 17.62
Andre et al, 1995#	M	58/4	7.86	2.60– 22.06
Franceschi et al,1999#	M	195/13	10.11	5.49– 18.60
Herrero et al,2003	Both	274/388	3.05	2.31– 4.04
Peters et al, 2006	Both	–	3.58	2.67– 4.80
Applebaum et al, 2007	Both	68/34	4.80	3.39– 6.88
De Stefani et al, 2007	M	254/34	5.09	3.42– 7.57
All case–control studies			4.70	3.76– 5.88
Cohort studies				
Boffetta and Garfinkel, 1990	M	44/55	4.41	3.07– 6.33
All studies			4.64	3.78– 5.70
p for heterogeneity=0.001				
Pharynx				
Case–control studies				
Wynder and Bross, 1957*	M	54/13	7.05	3.54– 14.04
Vincent and Marchetta, 1963*	Both	31/3	41.33	11.41–149.73
Martinez, 1969§	M	26/2	6.03	0.47– 77.68
Elwood et al, 1984§	Both	41/9	12.15	3.74– 39.46
Brugere et al, 1987#	M	–	14.99	11.68– 19.24
Tuyns et al, 1988#	M	–	6.03	4.05– 8.98
Franceschi et al, 1990#	M	107/13	2.12	1.15– 3.92
Choi and Kaliyo, 1991	M	53/16	4.94	2.83– 9.28
Boffetta et al, 1992#	M	89/5	7.75	3.13– 19.19
Maier et al, 1994#	M	77/11	30.50	12.72– 73.15
Andre et al, 1995#	M	77/6	7.60	3.24– 17.83
Franceschi et al,1999#	M	240/29	4.04	2.61– 6.26
Herrero et al., 2003	Both	114/38	4.53	2.84– 7.22
Menvielle et al, 2004	M	153/6	6.14	4.26– 8.84
Peters et al, 2006	Both	–	3.49	2.48– 4.91
Applebaum et al, 2007	Both	75/38	2.90	1.61– 5.23
De Stefani et al, 2007	M	355/33	7.06	4.77– 10.45
All studies			6.62	4.72– 9.29
p for heterogeneity<0.001				



BMC Public Health

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Research article

Open Access

The synergistic effect of cigarette taxes on the consumption of cigarettes, alcohol and betel nuts

Jie-Min Lee*

Background: Consumption of cigarettes and alcoholic beverages creates serious health consequences for individuals and overwhelming financial burdens for governments around the world. In Asia, a third stimulant – betel nuts – increases this burden exponentially. For example, individuals who simultaneously smoke, chew betel nuts and drink alcohol are approximately 123 times more likely to develop oral, pharyngeal and laryngeal cancer than are those who do not.

To discourage consumption of cigarettes, the government of Taiwan has imposed three taxes over the last two decades. It now wishes to lower consumption of betel nuts. To assist in this effort, our study poses two questions: 1) Will the imposition of an NT$10 Health Tax on cigarettes effectively reduce cigarette consumption? and 2) Will this cigarette tax also reduce consumption of alcoholic beverages and betel nuts? To answer these questions, we analyze the effect of the NT$10 tax on overall cigarette consumption as well as the cross price elasticities of cigarettes, betel nuts, and alcoholic beverages.

Methods: To establish the Central Bureau of Statistics demand function, we used cigarette, betel nut, and alcoholic beverage price and sales volume data for the years 1972–2002. To estimate the overall demand price elasticity of cigarettes, betel nuts, and alcoholic beverages, we used a seemingly unrelated regression analysis.

Results: We find that the NT$10 health tax on cigarettes will reduce cigarette consumption by a significant 27.22%. We also find that cigarettes, betel nuts, and alcoholic beverages have similar inherent price elasticities of -0.6571, -0.5871, and -0.6261 respectively. Because of this complementary relationship, the NT$10 health tax on cigarettes will reduce betel nut consumption by 20.07% and alcohol consumption by 7.5%.

Conclusion: The assessment of a health tax on cigarettes as a smoking control policy tool yields a win-win outcome for both government and consumers because it not only reduces cigarette consumption, but it also reduces betel nut and alcoholic beverage consumption due to a synergistic relationship. Revenues generated by the tax can be used to fund city and county smoking control programs as well as to meet the health insurance system's current financial shortfall.

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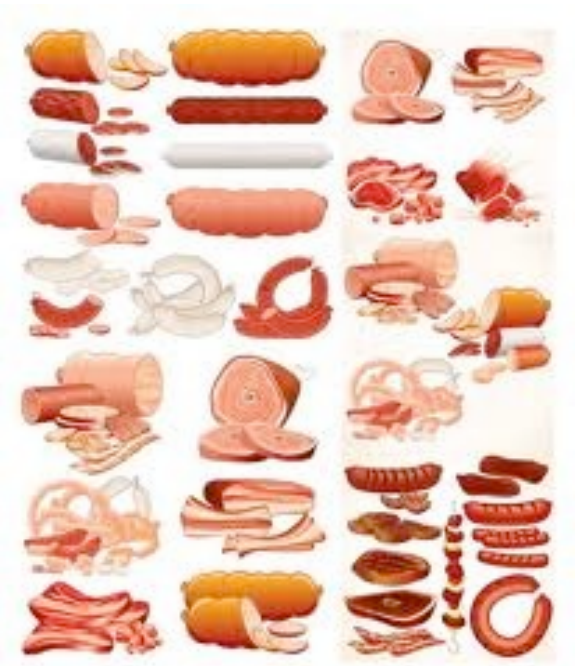
- Tabacco da masticare: noci di Betel (Areca nut) o Gutka, carcinogene sia con che senza tabacco.
- Tipico di minoranze Sud-est asiatico.

Dieta ed alimentazione

- Correlazione tra bassa introduzione di verdure e frutta fresca ed aumentato rischio di cancro orale.
- Riduzione del rischio del 50% per adeguato consumo di verdure e frutta fresca.
- Alto rischio associato a dieta ricca di carne .

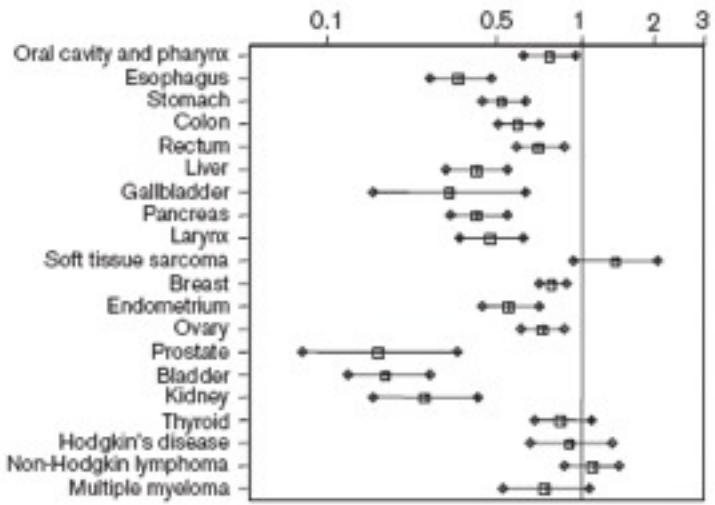


Mediterranean diet and cancer risk

S Gallus[1,2], C Bosetti[1] and C La Vecchia[1,3]

Various aspects of the Mediterranean diet are considered favourable with regard to cancer risk. These aspects were analysed using data from a series of case–control studies conducted in northern Italy between 1983 and 2001 on over 12 000 cases of 20 cancer sites and 10 000 controls. For most epithelial cancers, the risk decreased with increasing vegetable and fruit consumption, with odds ratios (OR) between 0.3 and 0.7 for the highest versus the lowest tertile. Subjects reporting frequent red meat intake showed ORs above unity for several common neoplasms. Conversely, fish (and consequently, *n*-3 fatty acids) tended to be another favourable dietary indicator. Wholegrain food intake was related to reduced risk of several types of cancer, particularly of the upper digestive tract, probably on account of its high fibre content. Fibres were in fact found to be protective with regard to colorectal and other selected cancers. In contrast to wholegrain, refined grain intake, and consequently glycaemic load, was associated with an increased risk of different types of cancer, including those of the upper digestive tract, colorectum, breast and endometrium. These results thus suggest that a low-risk diet for cancer entails increasing vegetables and fruit, reducing meat, but also refined carbohydrate consumption. Furthermore, olive oil and other unsaturated fats, which may be a unique common characteristic of the Mediterranean diet, should be preferred to animal and saturated fats. A score summarizing the major characteristics of the Mediterranean diet was inversely and consistently related to the risk of selected cancer sites. Regular consumption of pizza, one of the most typical Italian foods, showed a reduced risk of digestive tract cancers. Pizza could however simply be an indicator of a typical Italian diet. *European Journal of Cancer Prevention* 13:447–452 © 2004 Lippincott Williams & Wilkins.

European Journal of Cancer Prevention 2004, 13:447–452



Odds ratios and 95% confidence intervals of selected cancers according to vegetable consumption, Italy, 1983–1997.

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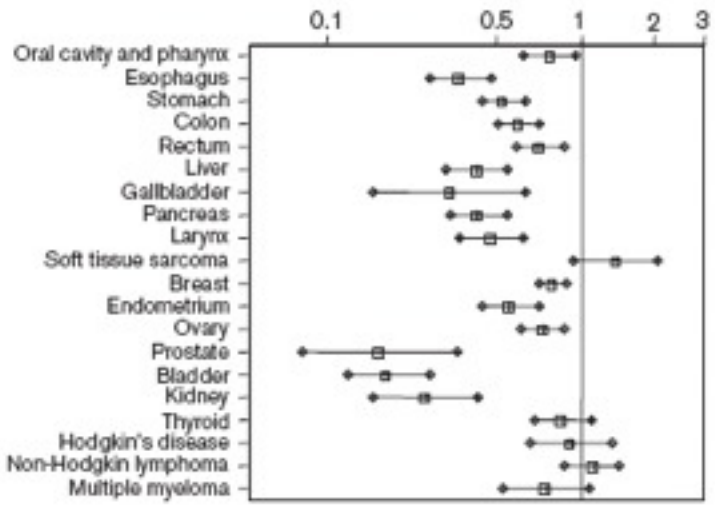


Odds ratios and 95% confidence intervals of selected cancers according to vegetable consumption, Italy, 1983–1997.

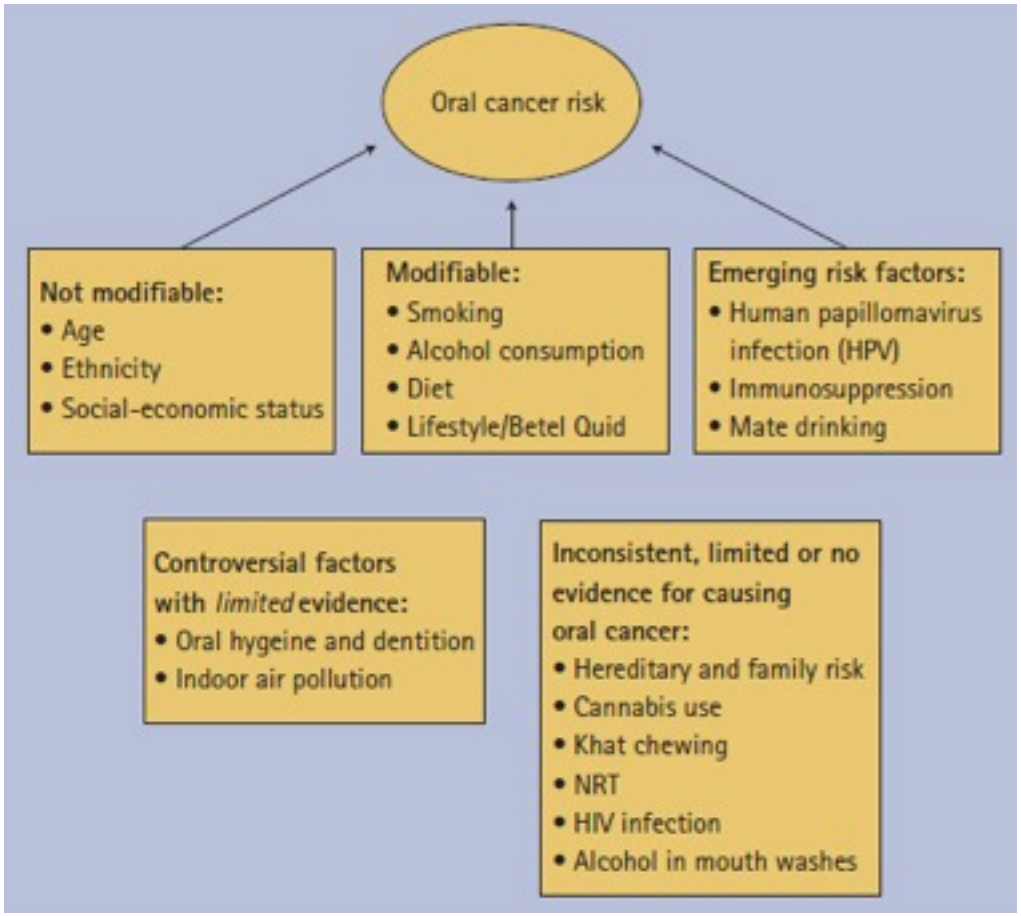
Stato socio-economico

- Maggiore prevalenza di abitudine al fumo ed all'alcool e dieta povera per basso livello socio-economico.
- Studi recenti attestano una maggiore incidenza per il cancro orale nei ceti medio-bassi indipendentemente dalle abitudini viziate.

Causes of oral cancer – an appraisal of controversies

S. Warnakulasuriya[1]

BRITISH DENTAL JOURNAL VOLUME 207 NO. 10 NOV 28 2009



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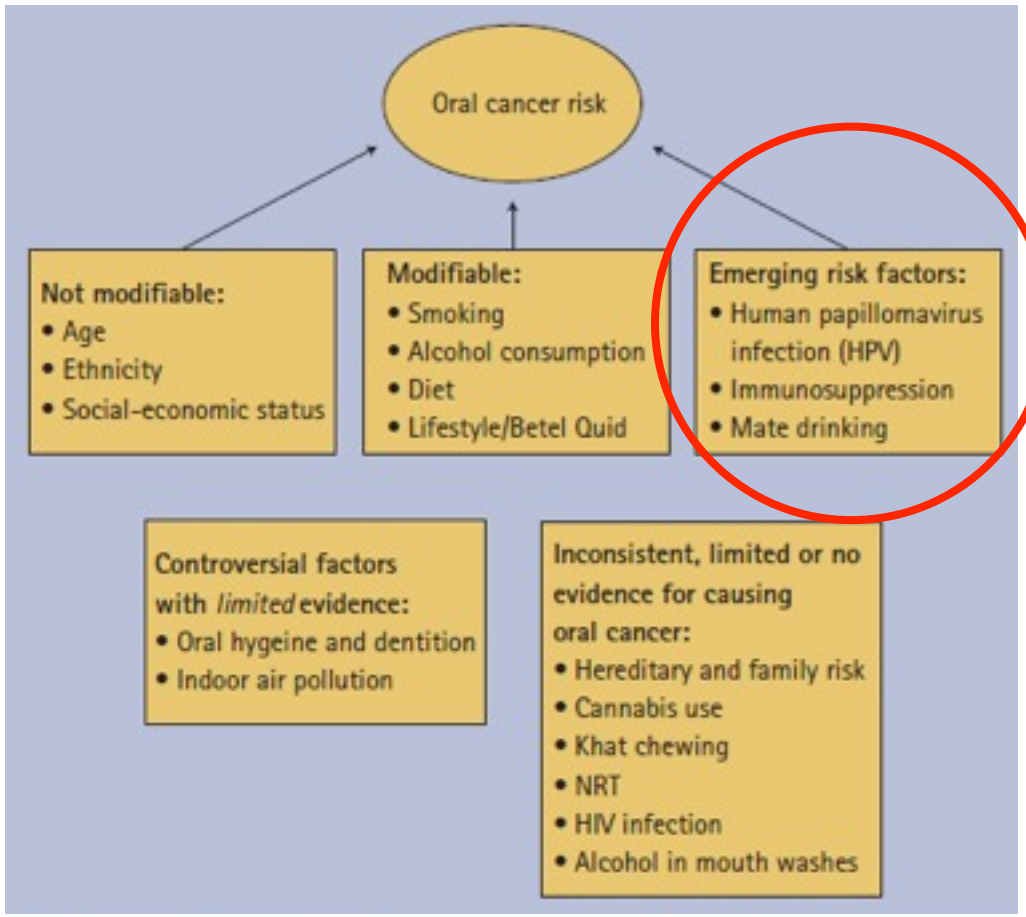
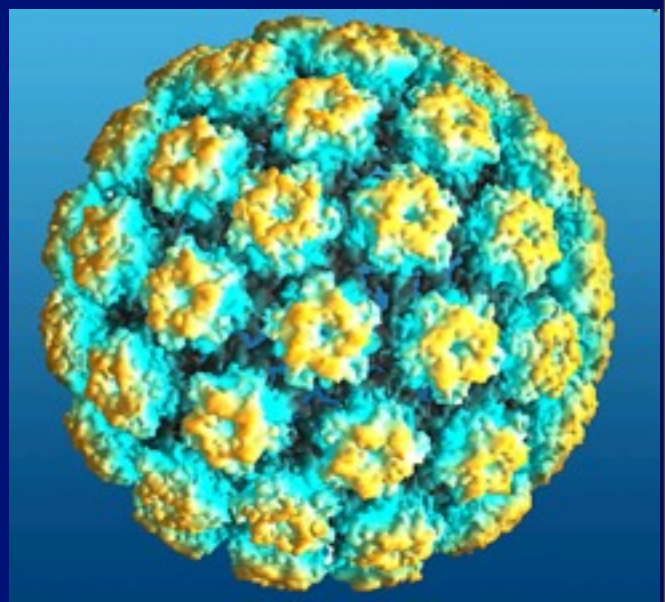


Human Papilloma Virus (HPV)

- Aumento incidenza carcinoma orofaringeo squamocellulare in maschi con età < 50 anni senza fattori di rischio.
- Stabilita associazione con infezione da HPV 16 (60% in USA)
- Associazione tra HPV 6-16 e **carcinoma orale** meno chiara.
- Presenza di HPV nelle cellule tumorali o presenza markers di HPV nel siero non dimostra il coinvolgimento di HPV nella patogenesi
- Non ancora stabilito la metodica ottimale per la rilevazione HPV orale nella clinica
- Vaccini (?)



Immunosoppressione

- Riportato aumento del rischio per ca orali (labbro) in trapiantati renali (farmaci immunosoppressori).
- Prolungato utilizzo di immunosoppressori in patologie infiammatorie croniche in report per aumentato rischio di ca orale.

Mate

- Infusione preparata con le foglie di erba Mate, una pianta originaria del Sud America, tradizionalmente bevuta calda.
- Studi epidemiologici realizzati in Sud America.
- L'alta temperatura a cui viene consumato può risultare come cofattore nell'irritazione cronica.

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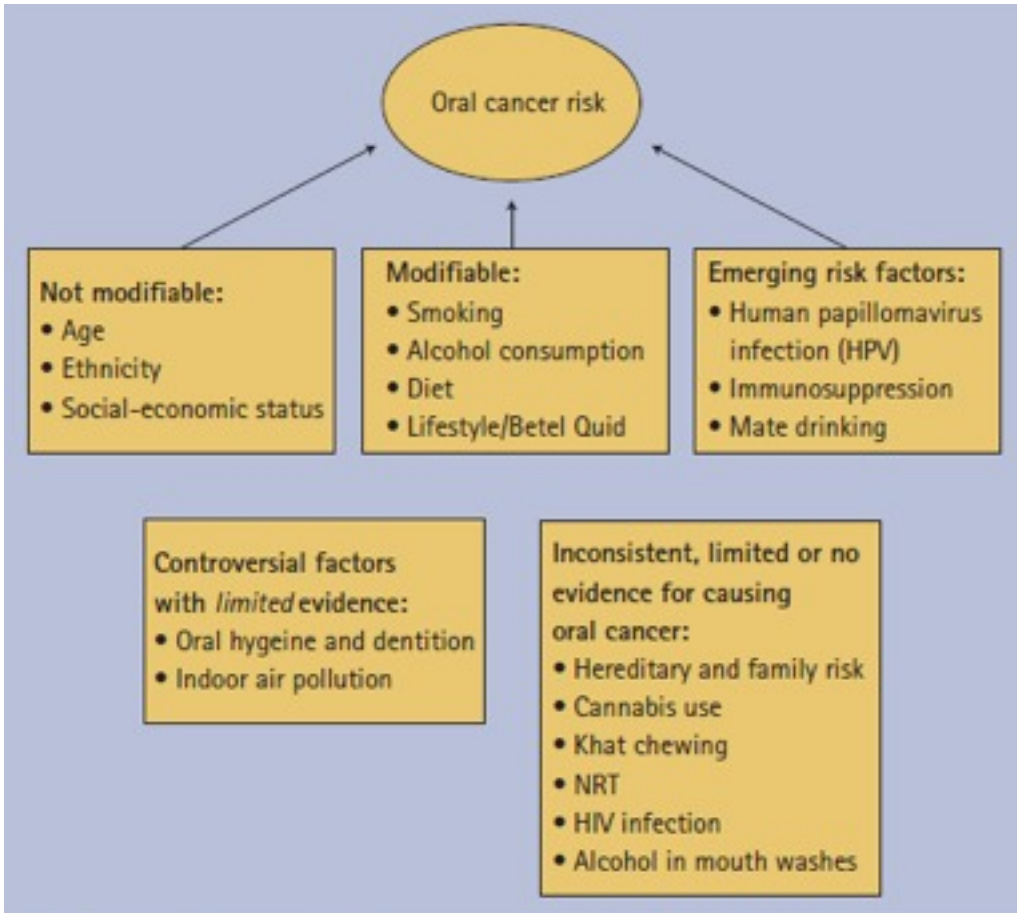

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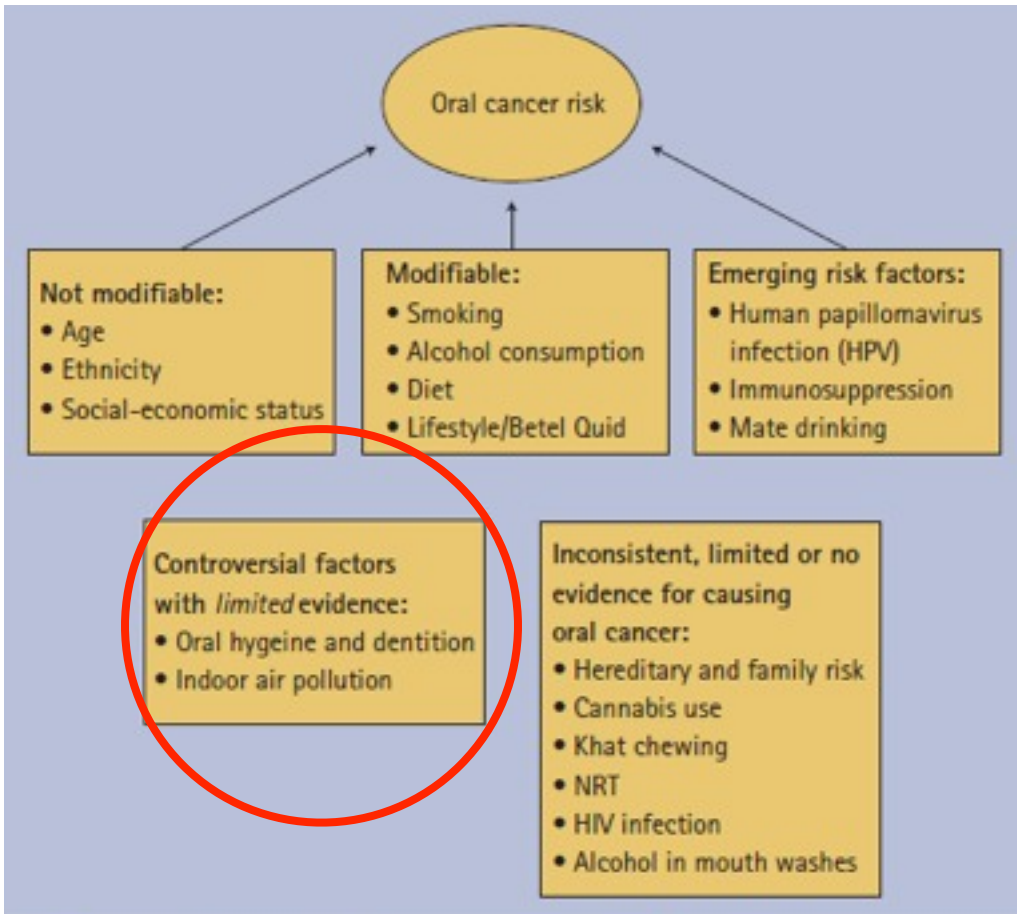

Igiene orale e status dentale

- Scarsa igiene orale o condizioni dentarie scadenti (restauri traumatizzanti, protesi inadeguate)
- Irritazione cronica facilitante l'esposizione a fattori carcinogeni.
- Microbi orali favorenti il metabolismo dell'etanolo in acetaldeide: alterazione flora batterica e micotica, aumento dei markers sistemici di infiammazione circolatoria.

Inquinamento ambienti interni

- Esposizione ai combustibili fossili da stufe a legna utilizzate per riscaldare o cucinare.

Causes of oral cancer – an appraisal of controversies

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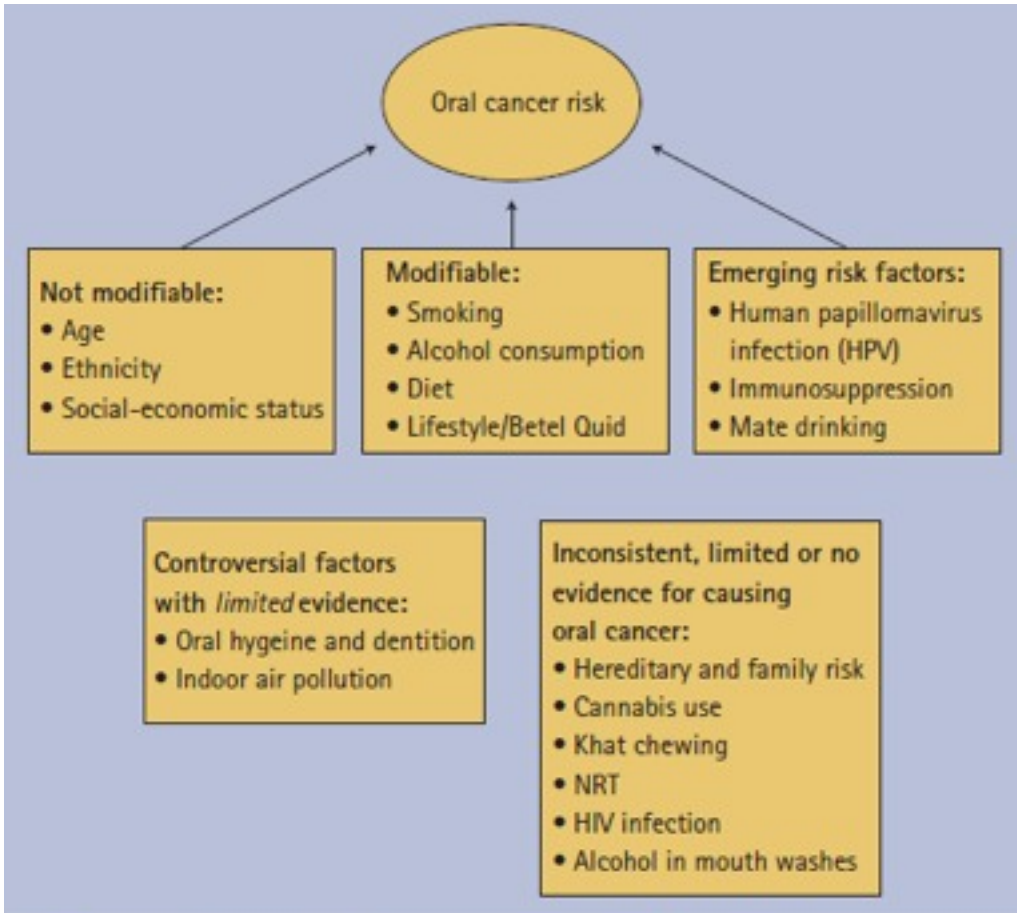

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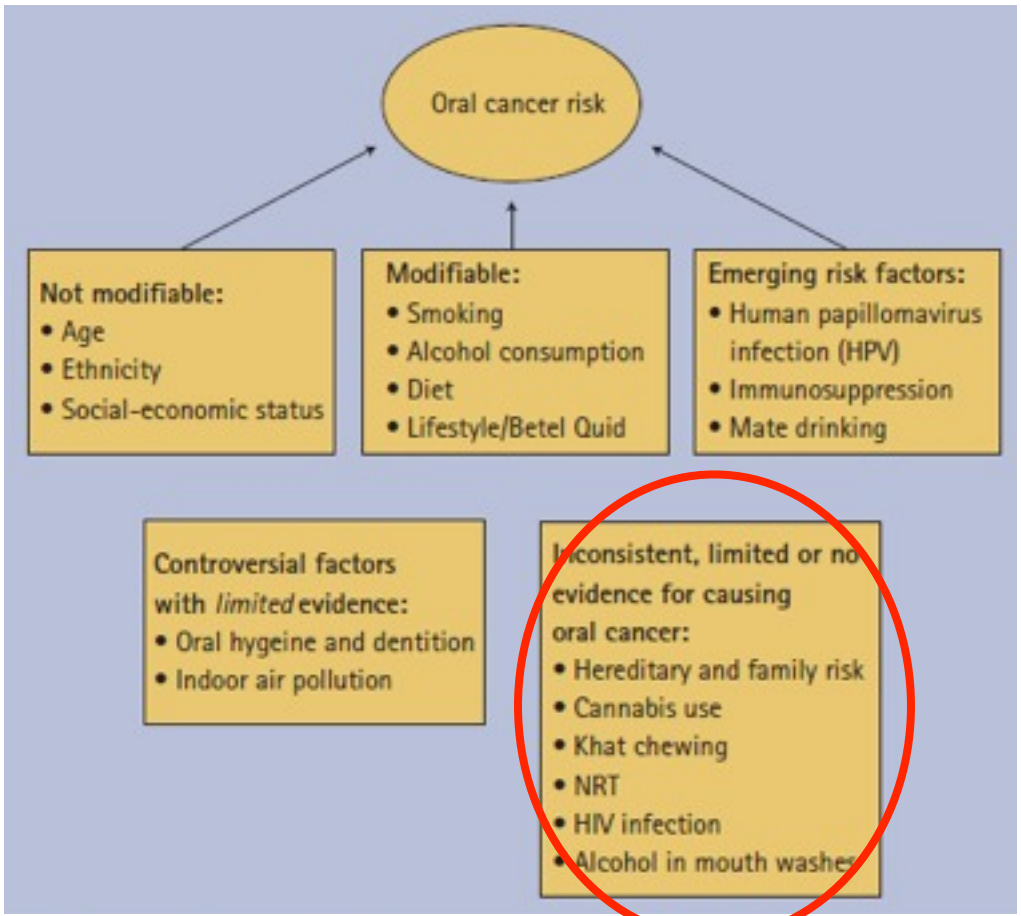
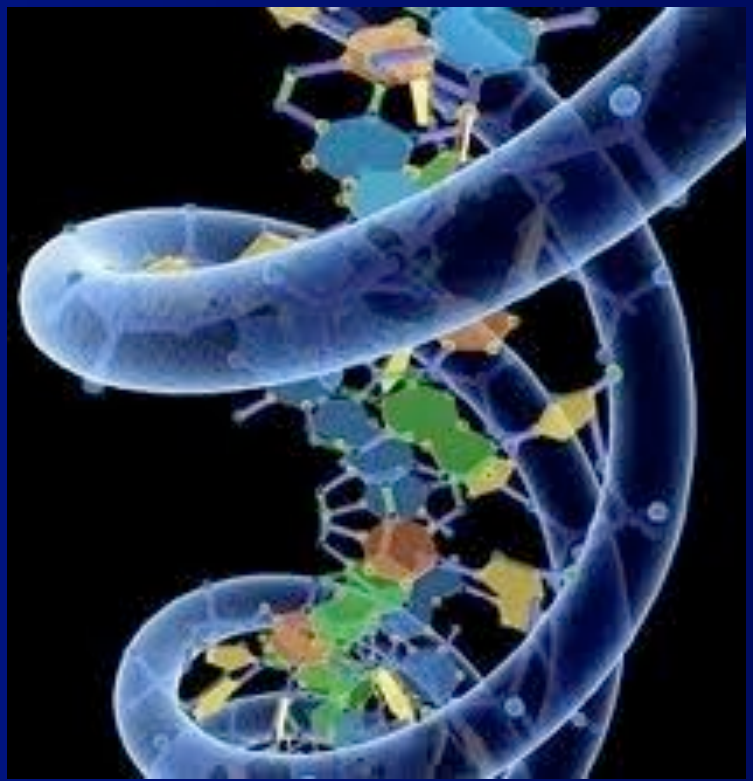


Ereditarietà e familiarità

- Non evidenza che suggerisca che il cancro orale sia più comune in famiglie ad elevato rischio per altri cancri.



Marijuana

- Droga maggiormente utilizzata in moltissime nazioni.
- Non evidenza che definisca la cannabis come agente causale per il cancro orale.
- Possibile ruolo del tabacco utilizzato in associazione.


Anmol
Pan
Masala
CANDIES
60
Pieces

Collutori alcoolici

- Etanolo presente come solvente in molti collutori in percentuali dal 6 al 29%.
- Riportata un'aumentata evidenza tra relazione diretta tra presenza di alcol nei collutori e sviluppo carcinoma orale.

Oral Oncology (2009) 45, 198–200

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REVIEW

Mouthwash and oral cancer risk: An update

Carlo La Vecchia *

Istituto di Ricerche Farmacologiche ''Mario Negri'' Milano e Istituto di Statistica Medica e Biometria ''G.A. Maccacaro'', Università degli Studi di Milano, Via Giuseppe La Masa 19, 20156 Milano, Italy

In conclusion, therefore, epidemiological findings on mouthwash and oral cancer were not consistent across various studies, populations and strata of major risk factors considered, including smokers and nonsmokers.[15] More specifically, the pattern of risk is not different with reference to alcohol-containing mouthwashes, and other types or mixed use of mouthwashes. This, again, weighs against any relevant association between alcohol-containing mouthwashes and oral cancer risk. This absence of association is also consistent with our knowledge of the dose-risk relationship between alcohol consumption and risk of upper digestive tract cancers, which show no excess risk for low doses of ethanol.[16]

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ORAL ONCOLOGY

REVIEW

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THE SURGEON 9 (2011) 278–283

available at www.sciencedirect.com

The Surgeon, Journal of the Royal Colleges of Surgeons of Edinburgh and Ireland

www.thesurgeon.net

A review of the relationship between alcohol and oral cancer

*J. Reidy**, *E. McHugh*, *L.F.A. Stassen*

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Conclusion

The relationship between alcohol consumption and oral cancer is clearly complex. Oral cancer is a disease with multiple interacting aetiologies, resulting in difficulties in determining the precise role of each agent independently. Despite this, the evidence supporting the role of alcohol in the aetiology of oral cancer is convincing, with a significant proportion of oral cancer deaths attributable to heavy alcohol consumption.[10] This illustrates the need for increased public awareness campaigns to reinforce the detrimental effects that chronic heavy alcohol consumption can have on both general and oral health. Evidence regarding the carcinogenic effect of alcohol-containing mouthrinses is conflicting, and a link between alcohol-containing mouthrinses and oral cancer has not yet been firmly established. However, considering what is known about the local effects of ethanol on the oral mucosa, it may be judicious to limit their use, particularly in high-risk patients such as smokers.

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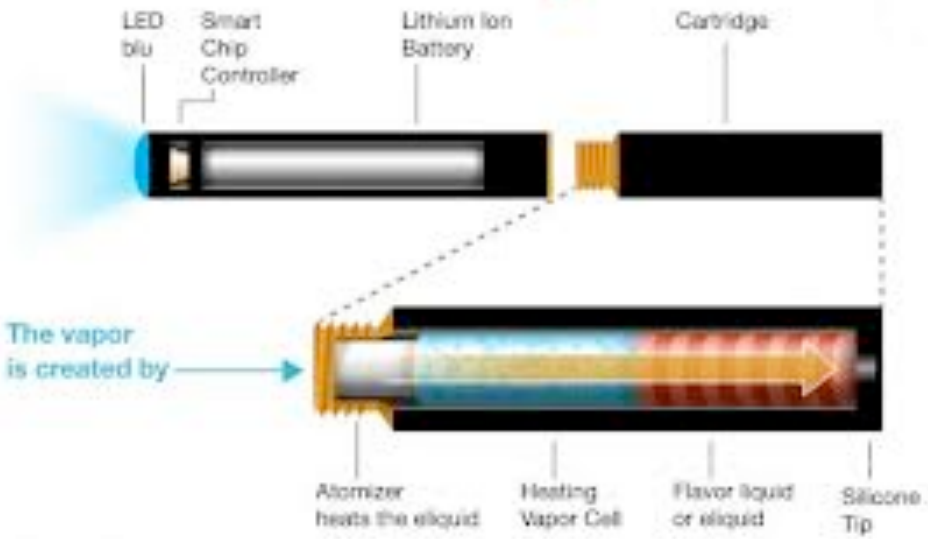
Inside of Electronic Cigarette



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Le Iene ti dicono cosa c'è nel vapore della sigaretta elettronica

di Redazione - 06/11/2013 - La bionda tecnologica su Italiauno

694 Consiglia

31 Tweet

La **sigaretta elettronica** fa bene o male alla salute? A fornire una risposta al frequente interrogativo ci hanno provato **Le Iene** con un servizio firmato da **Matteo Viviani** mandato in onda nella puntata di ieri.

LA DEFINIZIONE – «Tecnicamente – ha spiegato la trasmissione di Italiauno parlando della bionda tecnologica – è un vaporizzatore personale. Grossomodo funziona così: premendo un pulsante, la batteria comincia a fornire energia ad un atomizzatore, il quale, scaldando il liquido contenuto in un piccolo serbatoio, lo vaporizza».

IL BOOM – «Appena arrivato sul mercato, quattro anni fa, questo aggeggino ha avuto un successo enorme. Nel 2012 già un milione di italiani svaporava la sigaretta elettronica. Migliaia di negozi sono stati aperti in tutta Italia generando un fatturato, solo nel 2012, di 350 milioni di euro. Un boom impressionante che secondo molti sondaggi ha portato moltissime persone a smettere di fumare e molte altre a dminuire drasticamente il consumo delle bionde, guadagnandone in salute».

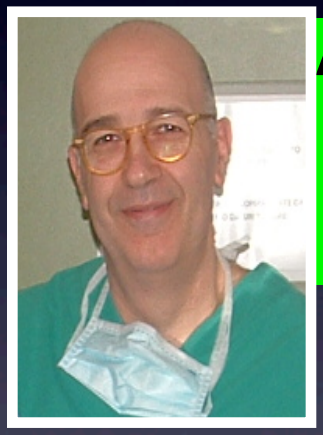
LA COMUNITÀ SCIENTIFICA DIVISA – «La comunità scientifica si è divisa in due gruppi, nettamente distinti, tra chi, come Veronesi sostiene che 'la sigaretta elettronica è assolutamente innocua, soprattutto senza nicotina' e chi, come il dottor Garattini dell'Istituto Mario Negri di Milano, dice fermamente no». E il **web** non ha certamente aiutato i consumatori a farsi un'idea chiara. In Rete sono infatti spuntate associazioni di commercianti, lobby di settore e pagine favorevoli e contrarie alla sigaretta tecnologica. A pagare le spere del caos sono soprattutto i negozianti che hanno investito i loro risparmi per aprire i nuovi negozi, molti dei quali, intervistati dalle Iene, si dicono prossimi alla chiusura.

Elisabetta Merigo

Maddalena Manfredi

Paolo Vescovi

Marco Meleti



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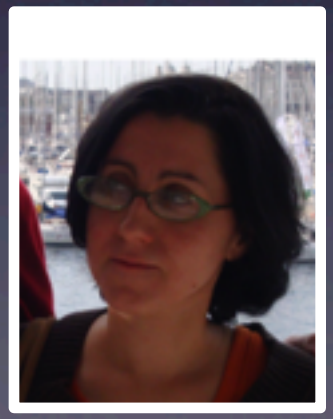
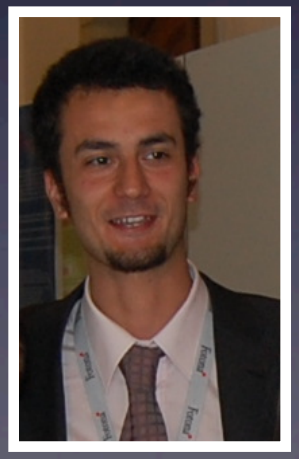
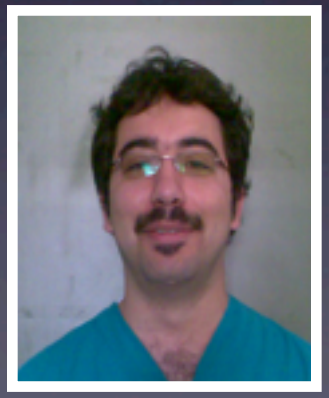


Tamara Simonazzi



Giovanni Mergoni



Amin Sarraj