

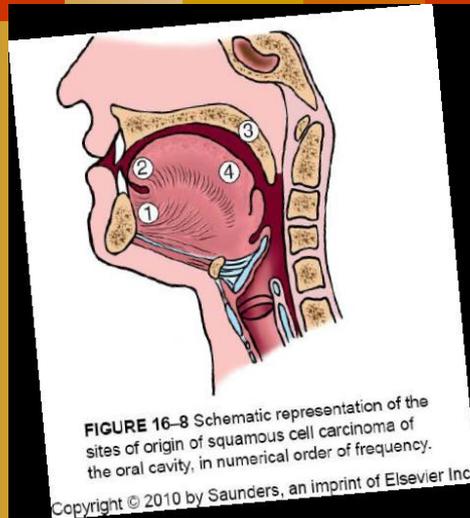
Università degli Studi di Ferrara
Corso di Laurea in Odontoiatria
Corso di Laurea in Igiene Dentale
Anno Accademico 2018-2019

Corso di Anatomia Patologica

Dr. Stefano Ferretti

Dipartimento di Morfologia, Chirurgia e Medicina sperimentale

Università di Ferrara



Cavo orale VI: lesioni reattive e neoplasie

Epitelio:

Ortocheratosi

- Cheratinizzazione superficiale normale

Cheratosi

- cheratinizzazione di epitelio normalmente non cheratinizzato

Paracheratosi

- Cheratinizzazione con cellule superficiali nucleate

Ipercheratosi

- Aumento di spessore dello strato di cheratina

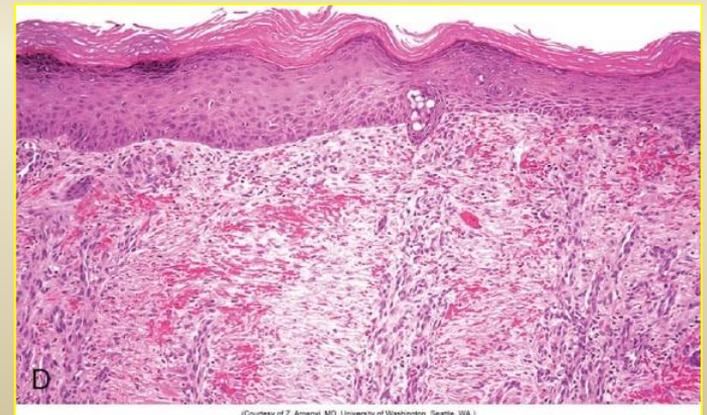
Acantosi

- Aumento di spessore da incremento strato spinoso

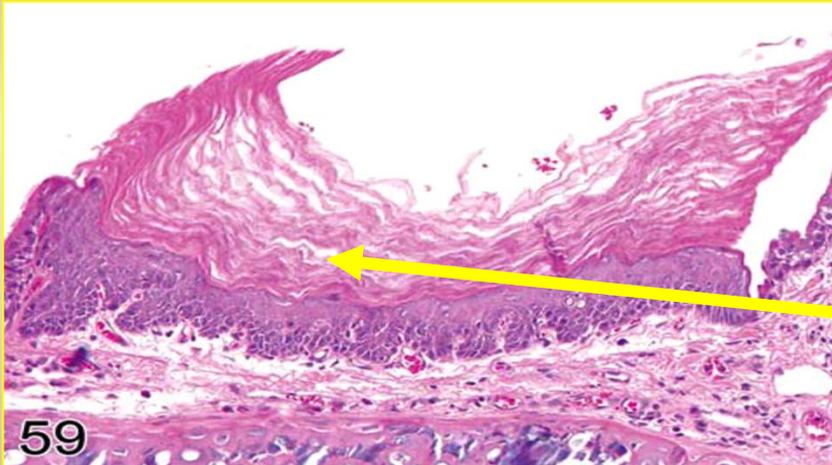
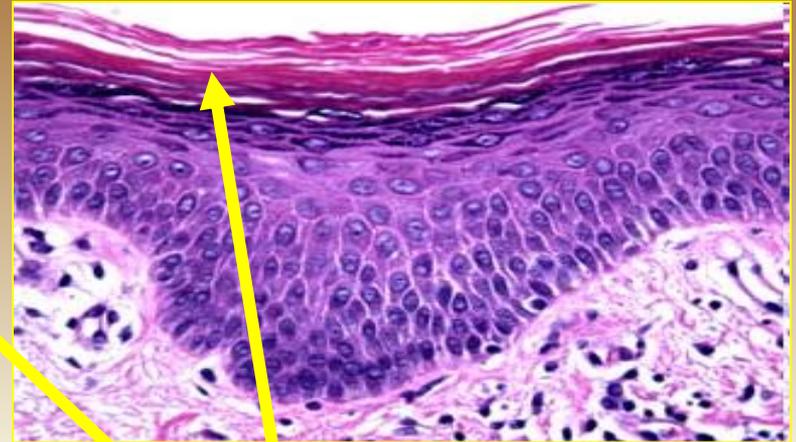
Atrofia

- riduzione dell'epitelio con scomparsa delle creste

Risposta al danno lesioni reattive



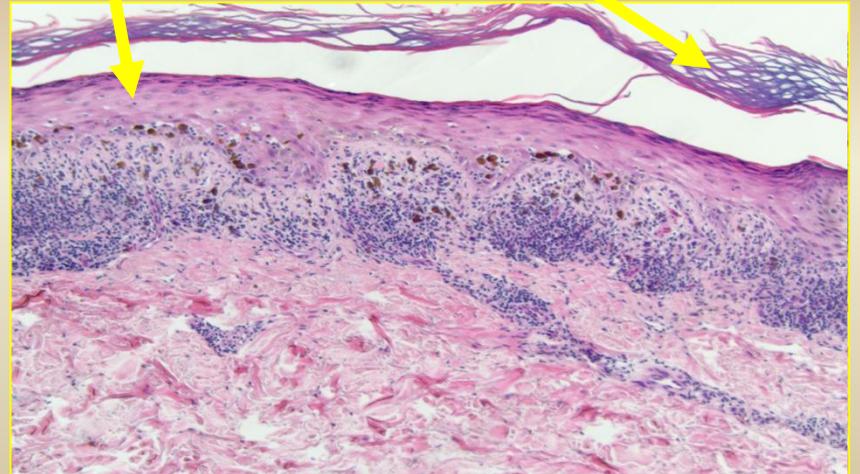
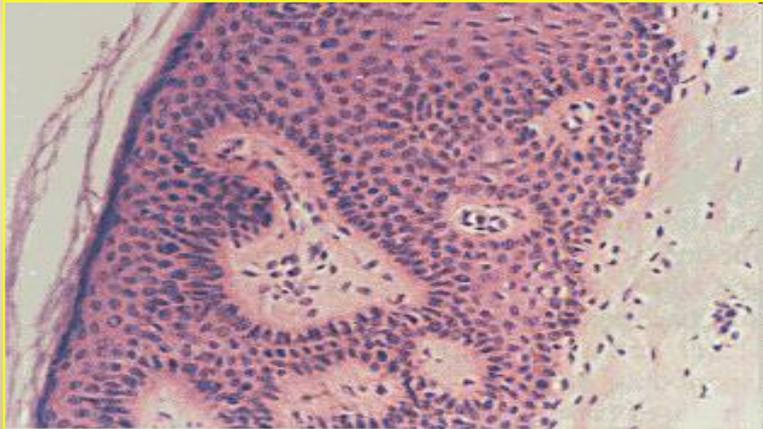
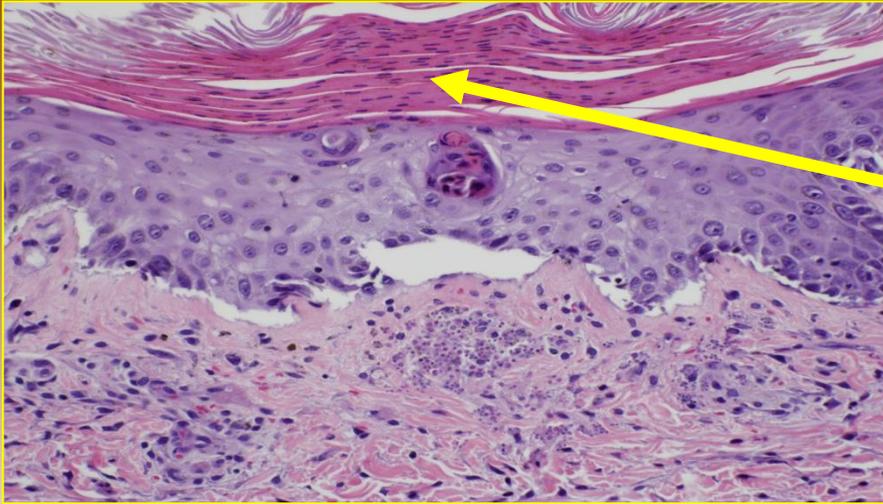
**Risposta al danno
lesioni reattive**



- Ortocheratosi
- Paracheratosi
- Ipercheratosi

**Risposta al danno
lesioni reattive**

- Iperparacheratosi
- Cheratosi
- Acantosi
- Atrofia



Tessuto fibroso:

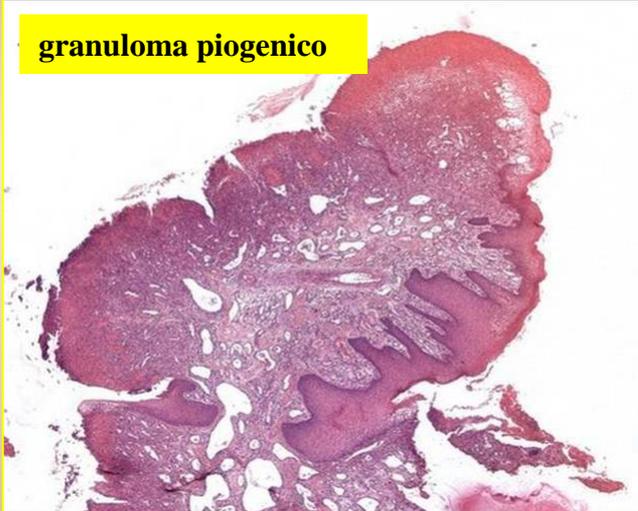
- Epulide fibrosa
- Granuloma piogenico
- Polipo fibroepiteliale
- Iperplasia irritativa
- Iperplasia papillare

**Risposta al danno
lesioni reattive**

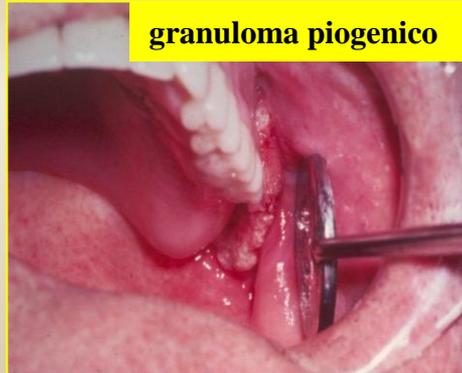


epulide fibrosa

granuloma piogenico



granuloma piogenico

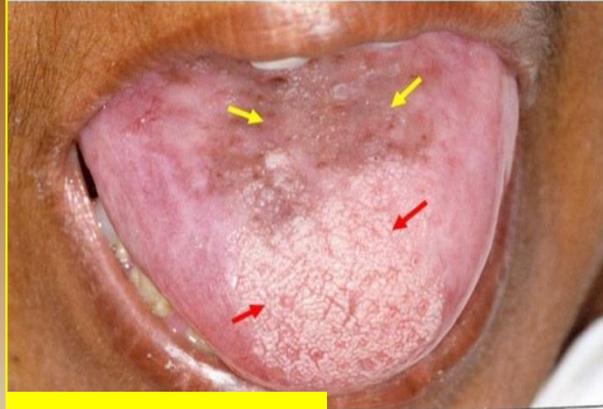


granuloma piogenico



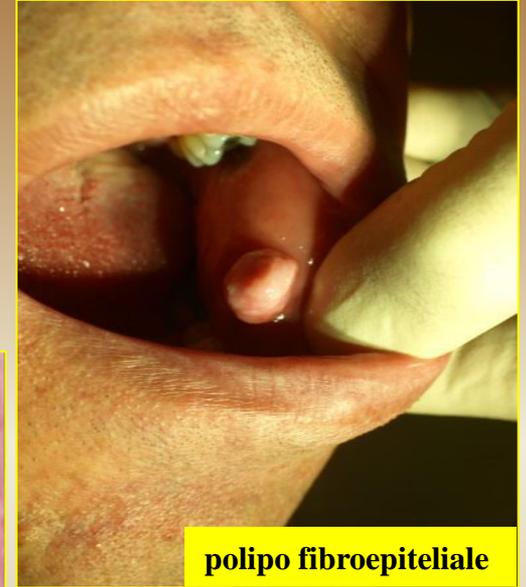
Tessuto fibroso:

- Epulide fibrosa
- Granuloma piogenico
- Polipo fibroepiteliale
- Iperplasia irritativa
- Iperplasia papillare



fibrosi sottomucosa

Risposta al danno lesioni reattive



polipo fibroepiteliale



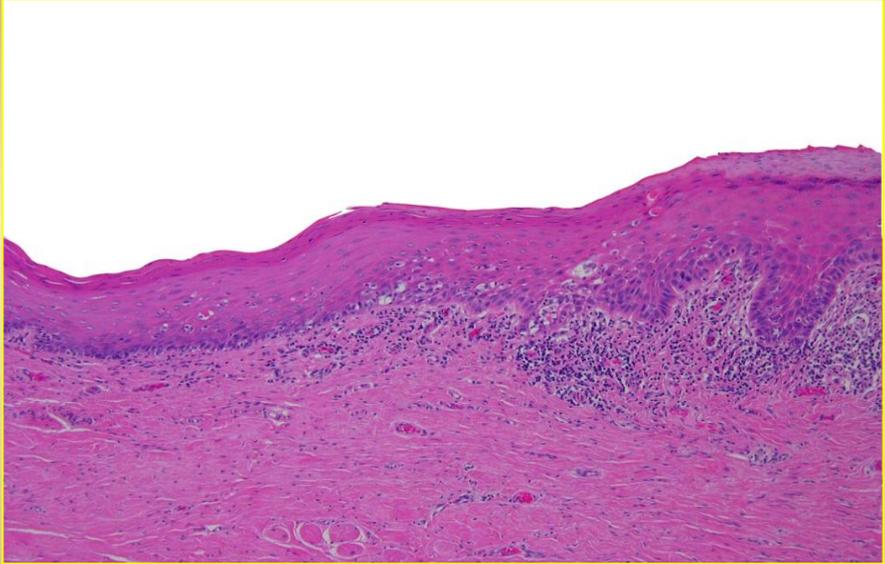
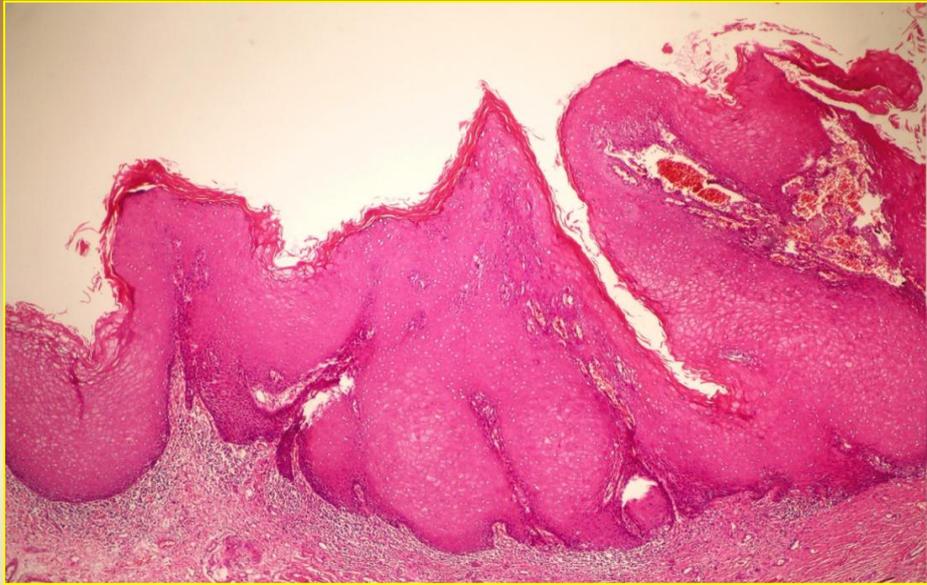
fibroma



fibrosi

leucoplachia

Risposta al danno
lesioni reattive

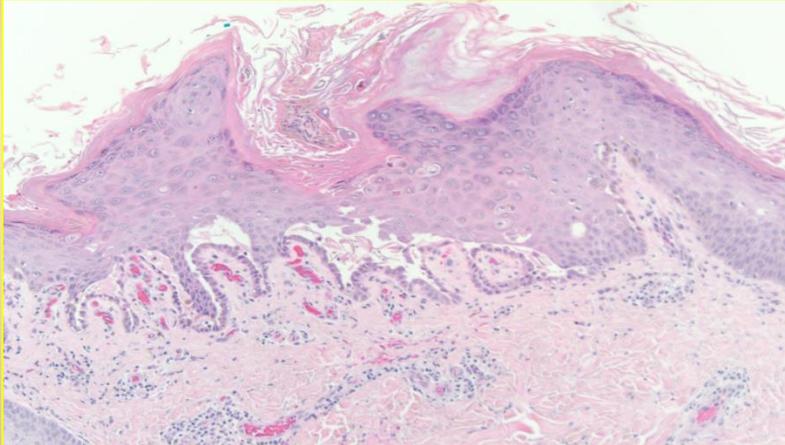


Condizioni ereditarie:

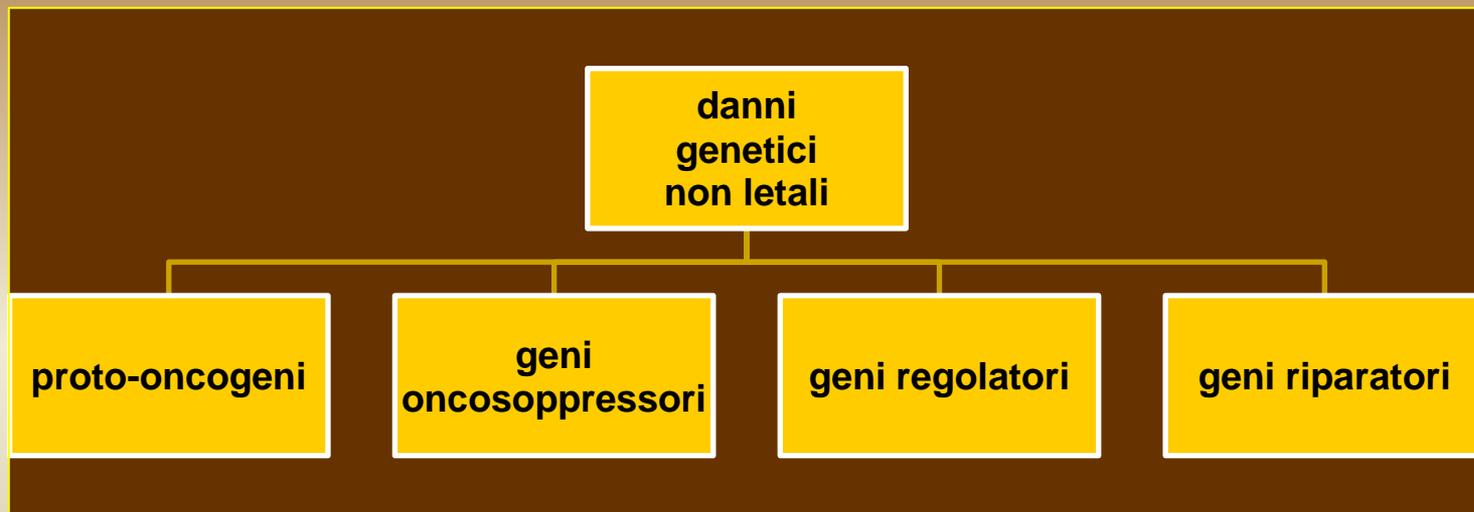
- Nevo epiteliale
- Pachionichia congenita
- Discheratosi congenita
- Tilosi
- Discheratosi ereditaria
- Cheratosi follicolare (m. di Darier)



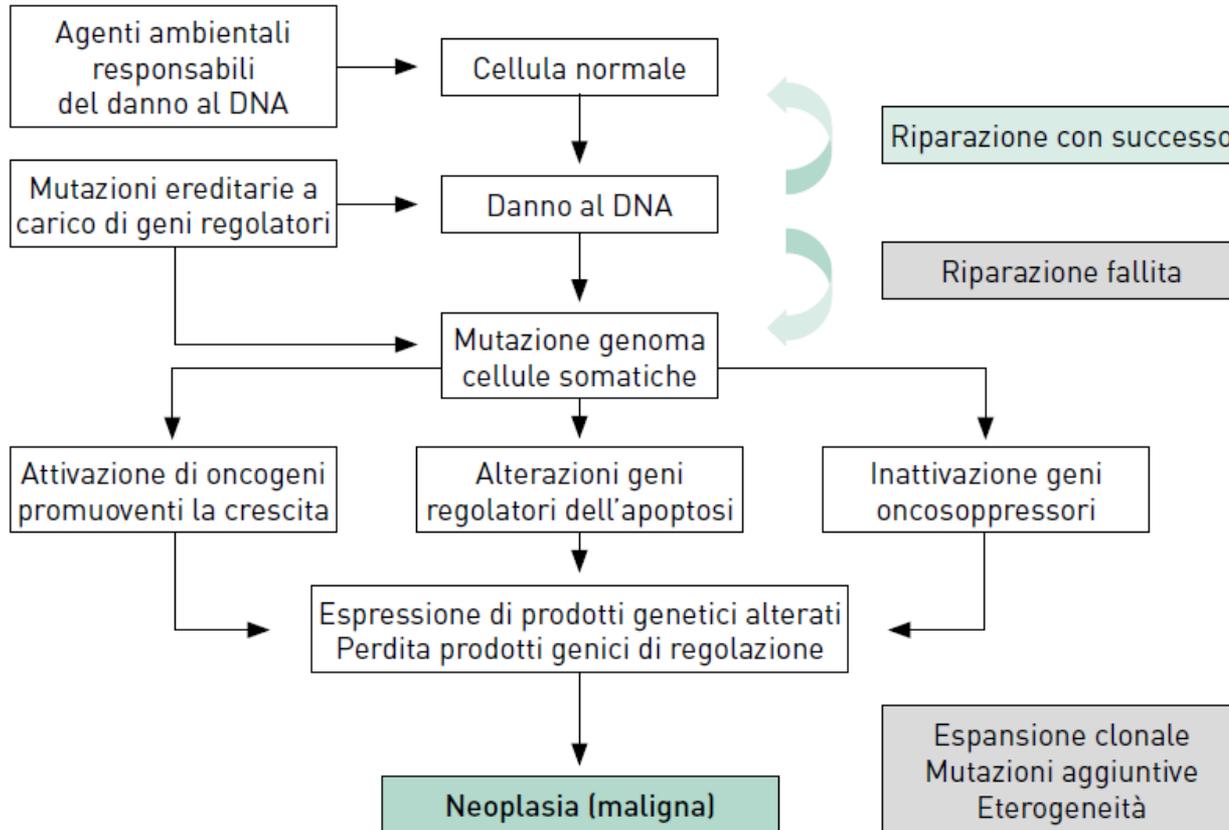
Condizioni ereditarie generalità



Basi molecolari delle neoplasie

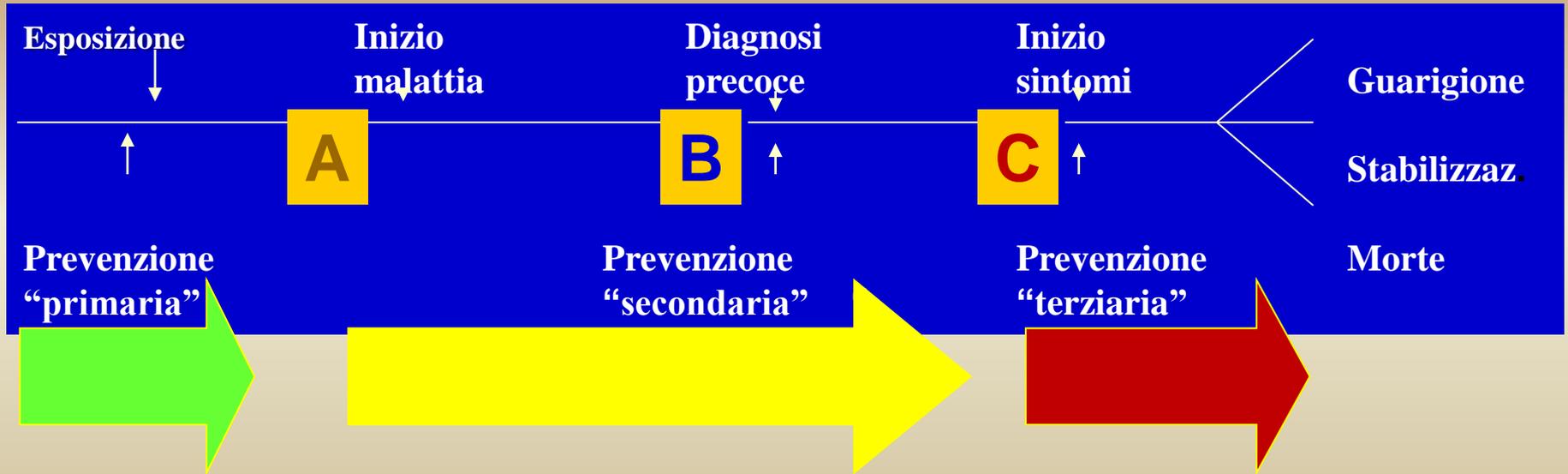


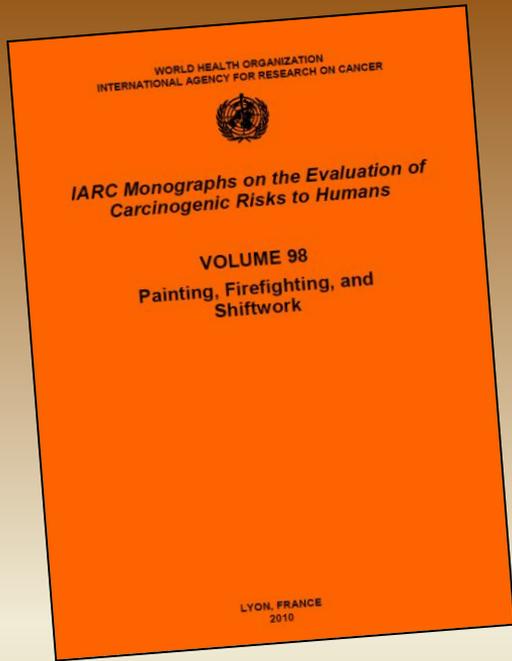
Cancerogenesi basi molecolari



Da: Cotran, Kumar, Collins 2005 (modificato)

Cancerogenesi fasi e tempistica





Important occupational agents or work processes considered by IARC (WHO) as being human carcinogens

Substance of process	Site(s) of cancer
Acrylonitrile	Lung
Aluminum production	Lung, bladder
4-Aminobiphenyl	Bladder
Arsenic and certain arsenic compounds	Lung, skin
Asbestos	Gastro-intestinal tract, mesothelioma of pleura and peritoneum
Auramine manufacture	Bladder
Benzene	Haemopoietic tissue
Benzidine	Bladder
Beryllium and beryllium compounds	Lung
Bis(chloromethyl) ether and chloromethyl methyl ether	Lung
Boot and shoe manufacture and repair	Nasal cavity
1,3-Butadiene	Haemopoietic tissue
Cadmium and cadmium compounds	Lung
Coal gasification	Lung
Coal-tars and pitches	Skin
Coke production	Lung
Chromium and certain chromium compounds	Lung
Diesel exhaust	Lung
Dioxins	Soft-tissue sarcoma, non-Hodgkin's lymphoma
Ethylene oxide	Haemopoietic tissue
Formaldehyde	Nose and nasopharynx
Glass manufacture	Lung
Hairdresser or barber	Bladder
Underground hematite mining	Lung
Iron and steel founding	Lung
Magenta, manufacture of	Bladder
Mineral oils, treated and mildly treated	Skin
Mustard gas	Pharynx, lung
2-Naphthylamine	Bladder
Nickel and nickel compounds	Nose and nasal sinus
Nonarsenical pesticides, spraying of	Lung
Painter	Lung
Petroleum refining, occupational exposure	Skin, haemopoietic tissue
Polychlorinated biphenyls	Liver, skin
Radon	Lung
Rubber industry	Bladder, haemopoietic tissue
Shale-oils	Skin
Silica	Lung
Soots	Skin
Sulphuric acid mist	Nasal cavity, larynx, lung
Talc-containing asbestiform fibres	Lung
Trichloroethylene	Liver, biliary tract
Vinyl chloride	Liver
Wood dust	Nasal cavity

Cancerogenesi fattori di rischio

Sources of ionizing radiation exposure

Source	Amount (mSv per year)
<i>Natural</i>	
Radon	3.0
Cosmic	2.0
Terrestrial	0.3
Internal	0.4
<i>Artificial</i>	
Medical	0.6
Consumer	0.5
<i>Other</i>	
Work, nuclear power generation, fallout	<0.01
Total	3.6

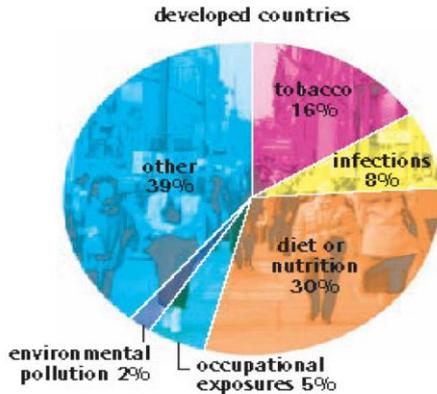
- ## Classificazione dei carcinogeni per l'uomo
- Gruppo 1 - **cancerogeni**
 - Gruppo 2 - **probabilmente cancerogeni**
 - Gruppo 3 - **possibilmente cancerogeni**
 - Gruppo 4 - **non classificabili per cancerogenicità**
 - Gruppo 5 - **probabilmente non cancerogeni**

Infectious agents associated with human cancers

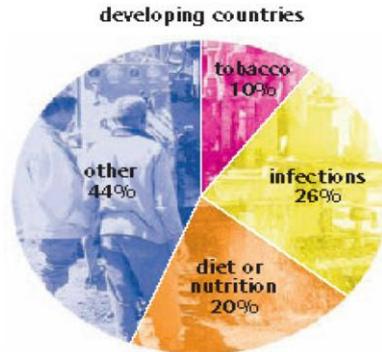
Infectious agents	Types of cancer	No. of cancer cases attributable to infections (1990, world total)	
Epstein-Barr virus (1964)	Burkitt's lymphoma	6100	
	Nasopharyngeal carcinoma	56200	
	Hodgkin's disease	26200	
	Post-transplant lymphomas		
Hepatitis B (1965)	Hepatocell	228900	
	Adult T-cell leukaemia	2600	
HLTV-1 (1980)	Cancer of the cervix	327000	
Human papilloma viruses (1983)	Ano-genital cancer	26400	
	Skin		
	Head and neck		
Human immunodeficiency virus (1983) associated with:			
	Human herpes virus-8 (1994)	Kaposi's sarcoma	43600
	Epstein-Barr virus	B-cell lymphoma	8800
	Human papilloma viruses (2000)	<i>In situ</i> cancer of the cervix, vulva/vagina and penis	
Hepatitis C (1988)	Hepatocell lar carcinoma	109700	
<i>Helicobacter pylori</i> (1983)	Gastric cancer	337800	
	Gastric lymphoma	8300	
Schistosomes	Bladder cancer	9500	
Liver flukes	Cholangiocarcinoma	800	
	Total	1 191 900	

Cancerogenesi fattori di rischio

Proportion of cancers caused by major risk factors



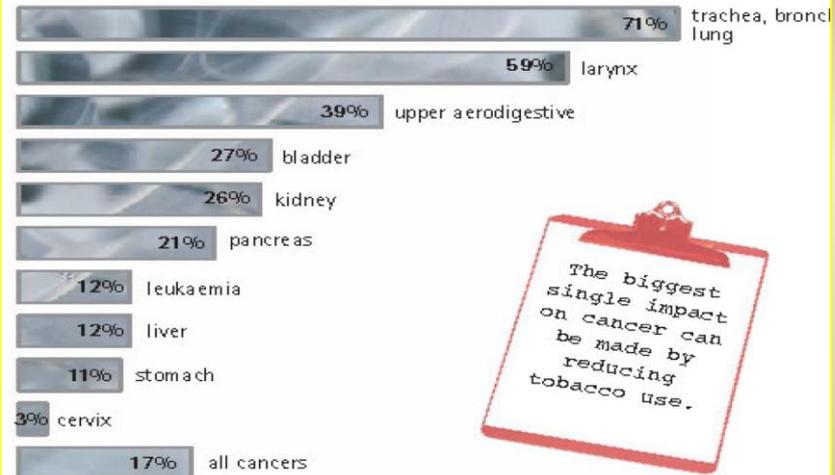
In developing countries, chronic infections are a major cause of cancer and occupational carcinogens pose a substantial risk.



About 1 in 5 cancer deaths worldwide is caused by tobacco.

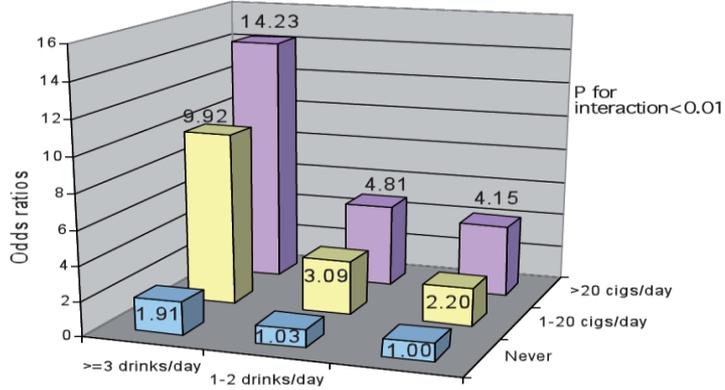
Cancer deaths caused by smoking

Percentage by site of cancer
2005 or most recent estimate



The biggest single impact on cancer can be made by reducing tobacco use.

tabacco

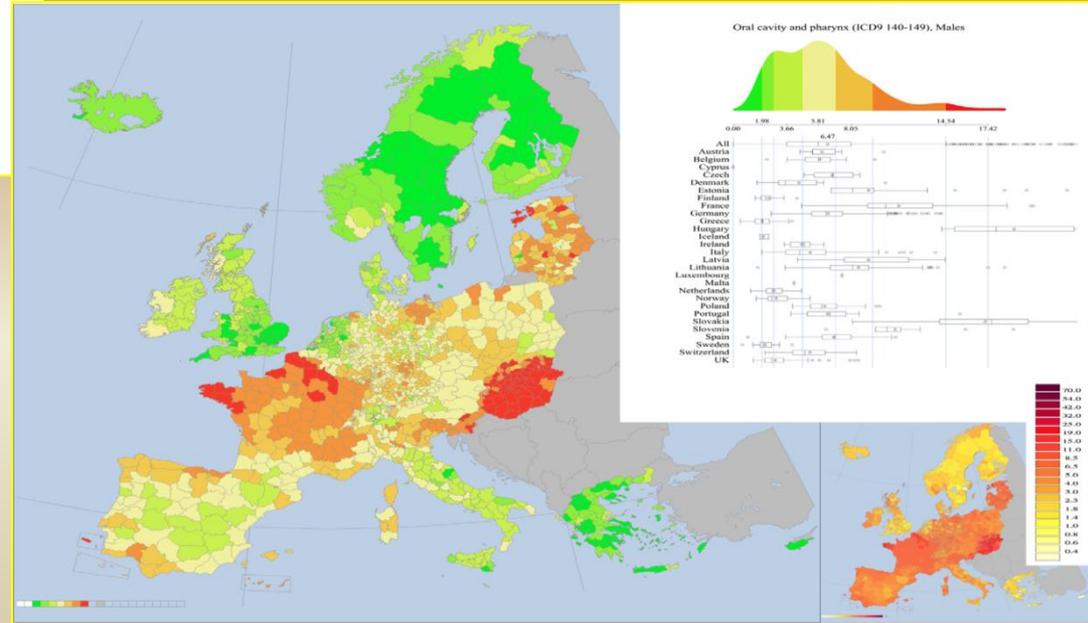


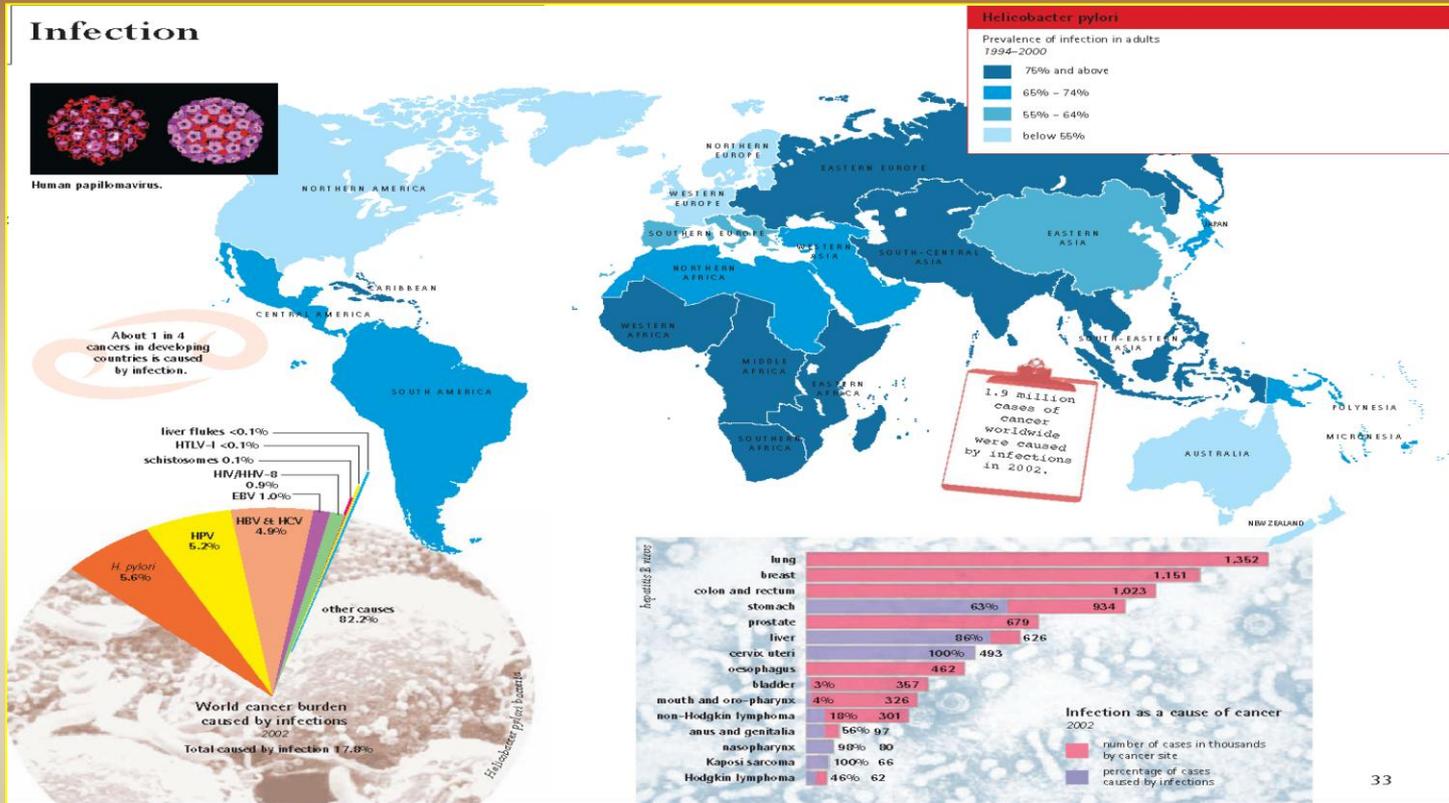
tabacco

**Cancerogenesi
fattori di rischio**

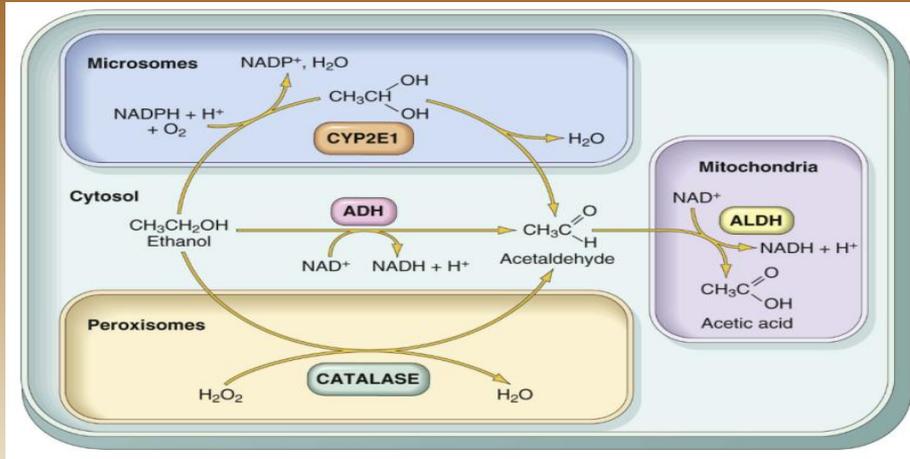
Summary

- > Tobacco smoking, alone and in combination with alcohol, is the most important risk factor
- > Tobacco chewing is also an important risk factor in some populations
- > Infection of human papilloma virus is a recognised cause of some head and neck cancers
- > Genes that metabolise alcohol appear to influence the risk of developing head and neck cancers





Cancerogenesi fattori di rischio



alcool

Metaboliti	Effetti biologici
Acetaldeide	<ul style="list-style-type: none"> • Prodotto diretto dell'ossidazione alcolica • Responsabile degli effetti tossici acuti e cronici
$\text{NAD} \gg \gg \text{NADH}$	<ul style="list-style-type: none"> • L'aumento di NADH/NAD causa acidosi lattica • La diminuzione di NAD compromette l'ossidazione degli acidi grassi nel fegato ($\gg \gg \gg$steatosi)
Fattori ossidanti	<ul style="list-style-type: none"> • Perossidazione lipidica (membrane epatociti)
Endotossine	<ul style="list-style-type: none"> • Rilascio lipopolisaccaridi da Gram- della flora intestinale • Produzione di TNF • Danno epatico

Cancerogenesi fattori di rischio

alcool

Intossicazione acuta:

SNC: deficit intellettivi, motori, coma

Stomaco: gastrite acuta (ulcera)

Fegato: steatosi

Pancreas: pancreatite acuta

Rene: necrosi tubulare tossica, IRA

Intossicazione cronica:

Fegato: steatosi, epatite cronica, cirrosi HCC

Pancreas: pancreatite cronica

App. G.I.: ulcerazioni, emorragie (varici)

SNC-SNP: Neuropatie (Def. B1), S. Wernicke-

Korsakoff, atrofia cerebrale, neuropatia oculare

Feto: s. alcolica fetale (ritardo crescita, anomalie scheletriche)

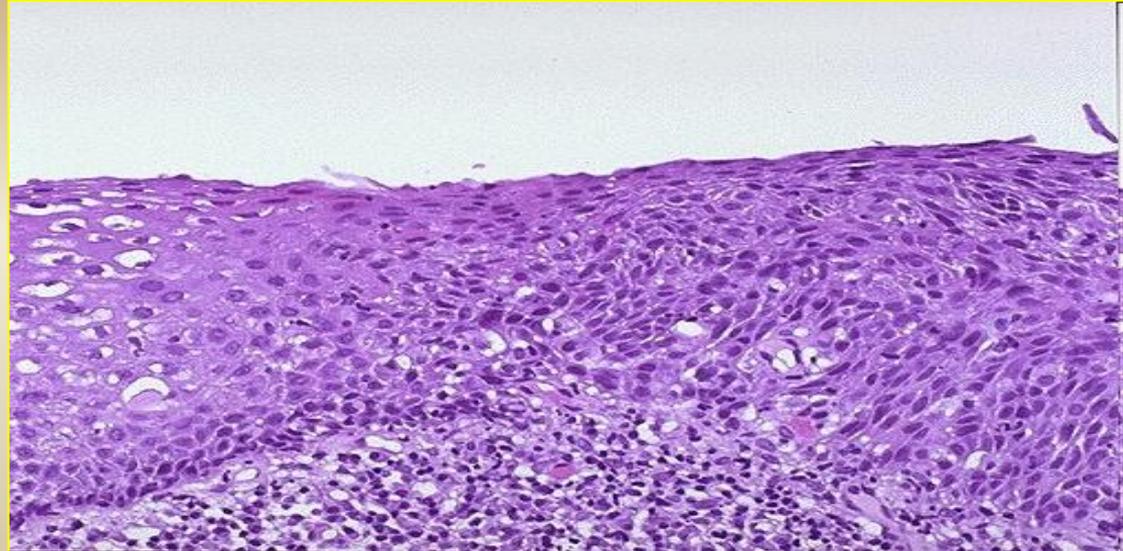
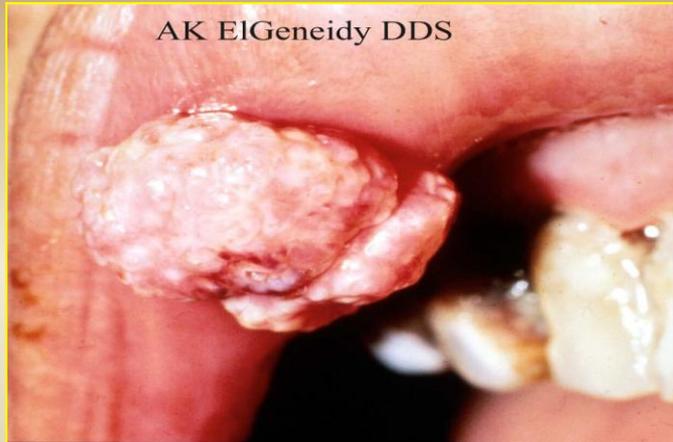
Testa-collo, fegato, (mammella): carcinomi

Clinica:

- Induzione di sonnolenza dai 100mg/dl, coma a livelli più alti
- Ossidazione ad acetaldeide dal cit. P450
- Deplezione di NAD (acidosi, steatosi ep.)
- Epatite alcolica, cirrosi, ipertensione portale, HCC
- Emorragie gastrointestinali
- Neuropatie periferiche
- Pancreatite acuta e cronica
- Cardiomiopatie
- Carcinomi del distretto testa-collo

HPV types 16, 18

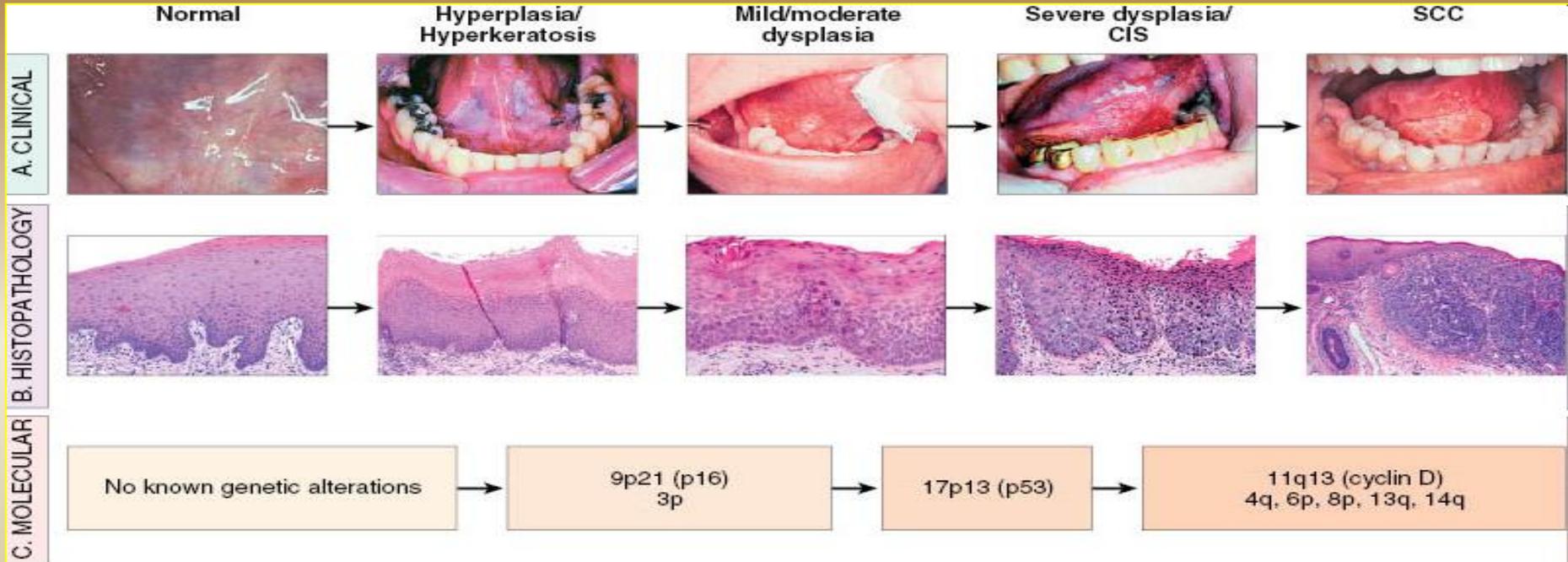
**Cancerogenesi
fattori di rischio**



Cancerogenesi alterazioni tissutali

Architettura	Citologia
Stratificazioni epiteliali irregolari	Variazioni anomale delle dimensioni dei nuclei (anisonucleosi)
Perdita di polarità delle cellule basali	Variazioni anomale di forma dei nuclei (pleomorfismo)
Creste epiteliali a goccia	Variazioni anomale delle dimensioni cellulari
Mitosi superficiali anomale	Aumento del rapporto nucleo/citoplasma
Cheratinizzazione prematura (discheratosi)	Aumento delle dimensioni del nucleo
Perle cornee	Figure mitotiche atipiche; aumento di numero e dimensioni dei nucleoli

Cancerogenesi cavo orale basi molecolari della progressione



(Clinical photographs courtesy of Sol Silverman, MD, from Silverman S: Oral Cancer. Hamilton, Ontario, Canada, BD Dekker, 2003.)

leucoplachia

Cancerogenesi cavo orale
condizioni precancerose

lesione prevalentemente bianca della mucosa orale che non può essere identificata con nessun'altra lesione riconoscibile

Fattori di rischio

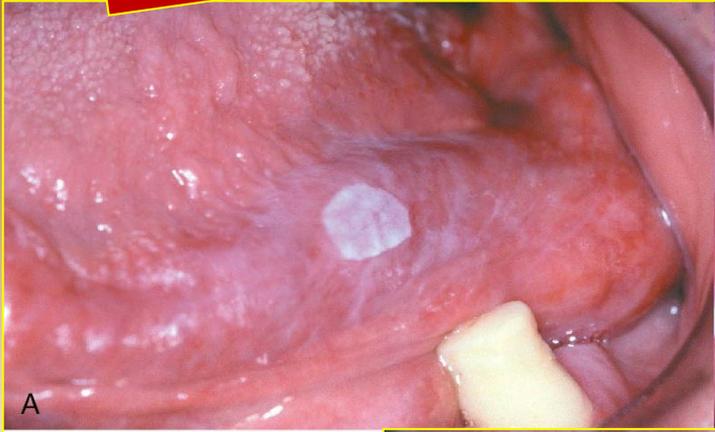
- Tabacco
- Alcol
- Candida
- Virus (HIV EBV)
- Atrofie (carenziali, infettive)
- Anomalie p53

Incidenza e prevalenza
•Variabili

Progressione
•4% a 10 anni

leucoplachia

Cancerogenesi cavo orale condizioni precancerose



(Courtesy of Drs. Neville, Damm, Allen, Bouquet [eds], Oral and Max

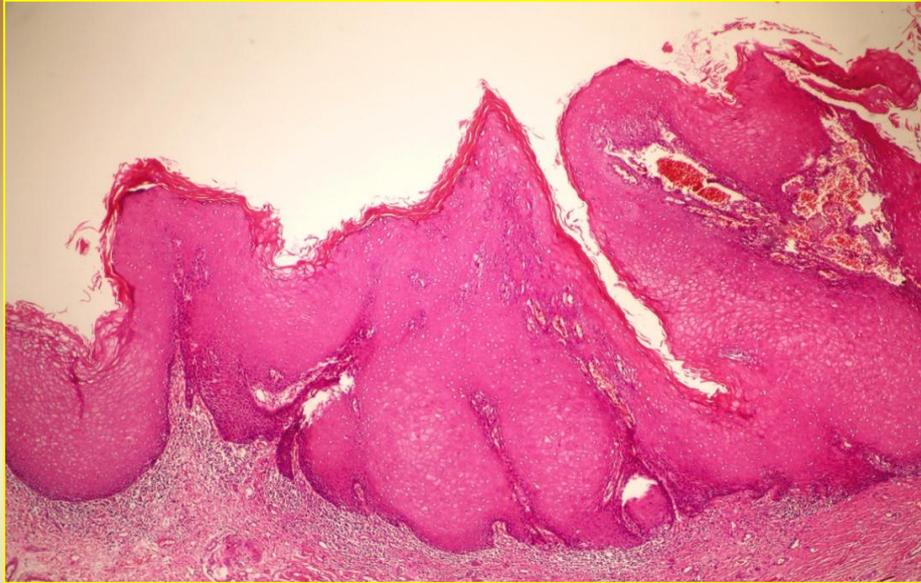


(Courtesy of Drs. Neville, Damm, Allen, Bouquet [eds], Oral and Maxillofacial Pathology, Philadelphia, WB Saunders, 2008.)

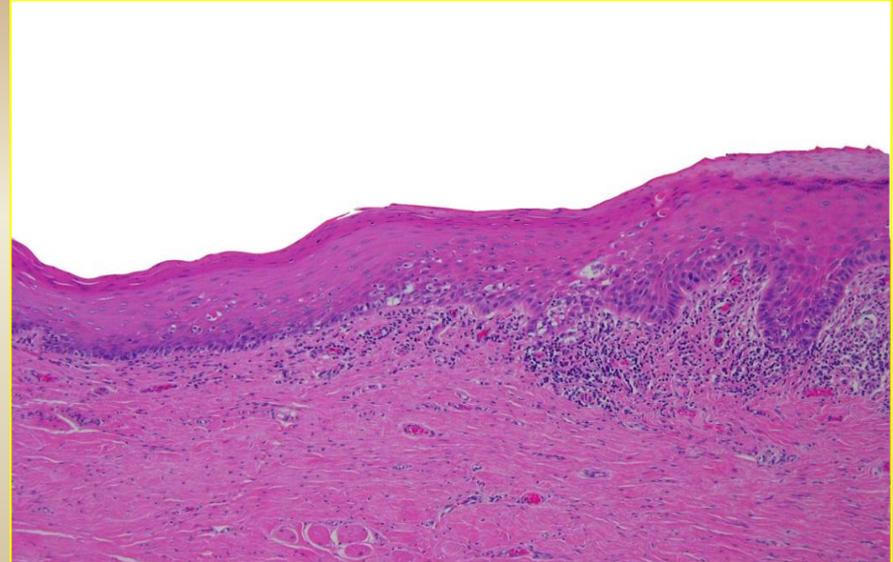


(Courtesy of Drs. Neville, Damm, Allen, Bouquet [eds], Oral and Maxillofacial Pathology, Philadelphia, WB Saunders, 2008.)

leucoplachia



Cancerogenesi cavo orale condizioni precancerose

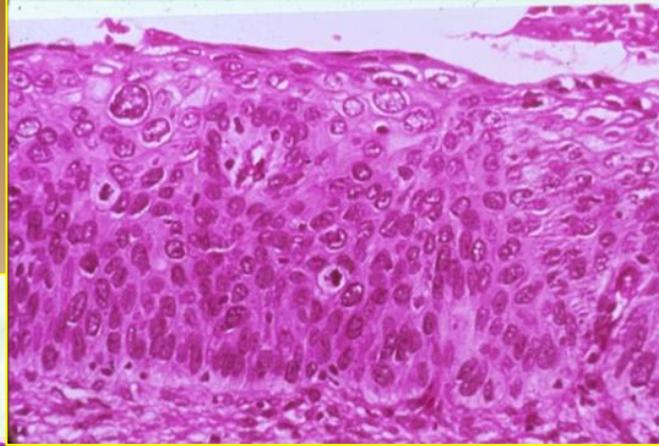


eritroplachia

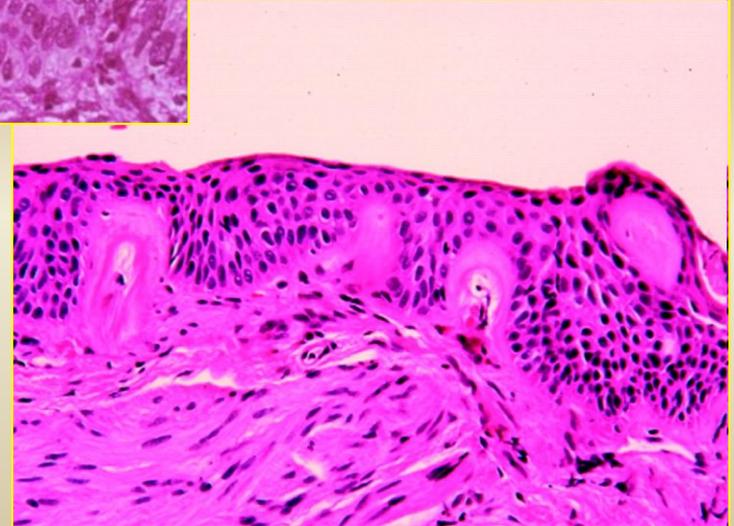
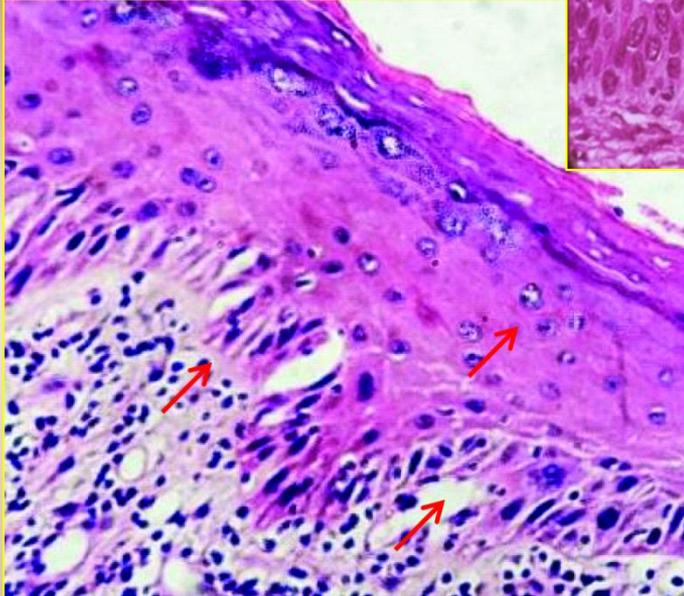
Cancerogenesi cavo orale
lesioni precancerose



eritroplachia

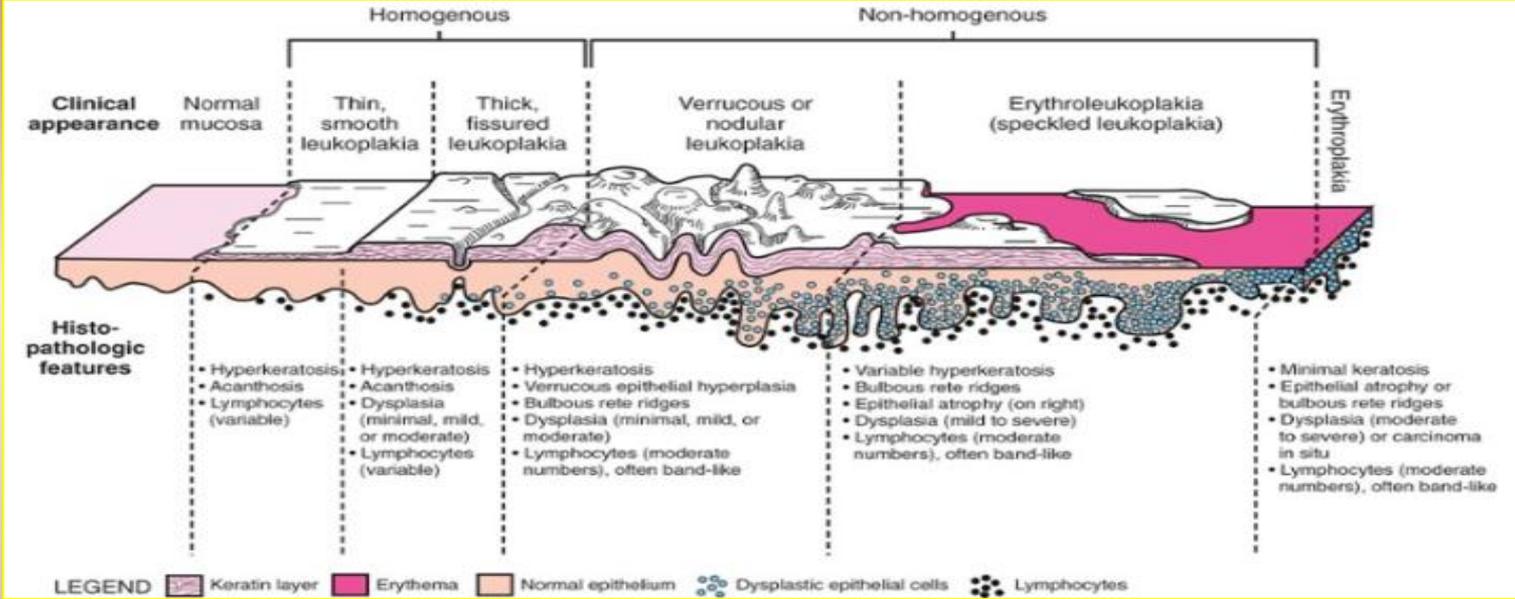


Cancerogenesi cavo orale
lesioni precancerose



leuco/eritroplachia

Cancerogenesi cavo orale condizioni/lesioni precancerose



Cancerogenesi cavo orale condizioni/lesioni precancerose

Lesioni di partenza	Sesso	Casi studio	Anni/pers. studio	Casi RT*	Anni/pers. RT*	IRR grezzi ^o	IC 95%	IRR agg. ^{oo}	IC 95%
Leucoplachia	maschi	3	1 215	410	3.040.167	18,31	3,8; 53,9	20,60	4,2; 60,2
	femmine	4	1 086	163	3.315.786	74,93	20,2; 195,5	27,05	7,4; 69,3
	totale	7	2 301	573	6.355.953	33,74	13,5; 70,0	23,85	9,6; 49,1
Eritroplachia	maschi	2	122	410	3.040.167	121,56	14,7; 441,9	75,38	9,1; 272,2
	femmine	2	69	163	3.315.786	589,63	70,8; 2164,4	220,77	26,8; 797,2
	totale	4	191	573	6.355.953	232,30	63,1; 598,0	112,39	30,6; 287,7
Lichen	maschi	5	719	410	3.040.167	51,57	16,7; 121,3	65,89	21,4; 153,8
	femmine	3	947	163	3.315.786	64,44	13,2; 191,7	23,64	4,9; 23,6
	totale	8	1 666	573	6.355.953	53,26	22,9; 105,7	39,45	17,0; 77,7
Polipi	maschi	1	4 115	410	3.040.167	1,80	0,1; 10,1	2,32	0,1; 12,9
	femmine	0	6 494	163	3.315.786	0,00	-	0,00	-
	totale	1	10 609	573	6.355.953	1,05	0,3; 5,9	0,86	0,0; 4,8
Papillomi	maschi	1	2 863	410	3.040.167	2,58	0,1; 14,5	4,64	0,1; 25,9
	femmine	0	2 520	163	3.315.786	0,00	-	0,00	-
	totale	1	5 383	573	6.355.953	2,06	0,1; 11,5	2,23	0,1; 12,4
Aspecifiche	maschi	6	2 125	410	3.040.167	20,94	7,7; 46,0	23,23	8,5; 50,6
	femmine	3	2 647	163	3.315.786	23,01	4,7; 68,6	9,12	1,9; 26,6
	totale	9	4 772	573	6.355.953	20,92	9,5; 40,0	15,32	7,0; 29,1
Totali	maschi	18	11 159	410	3.040.167	11,96	7,0; 19,1	15,61	9,3; 24,7
	femmine	12	13 763	163	3.315.786	17,74	9,0; 31,8	7,61	3,9; 13,3
	totale	30	24 922	573	6.355.953	13,35	8,9; 19,3	10,99	7,4; 15,7

(*) Rif. Registro tumori provincia Ferrara 1991-2008

(^o) Incidence Rate ratio grezzo e (^{oo}) aggiustato per età

Neoplasie del cavo orale carcinoma squamoso

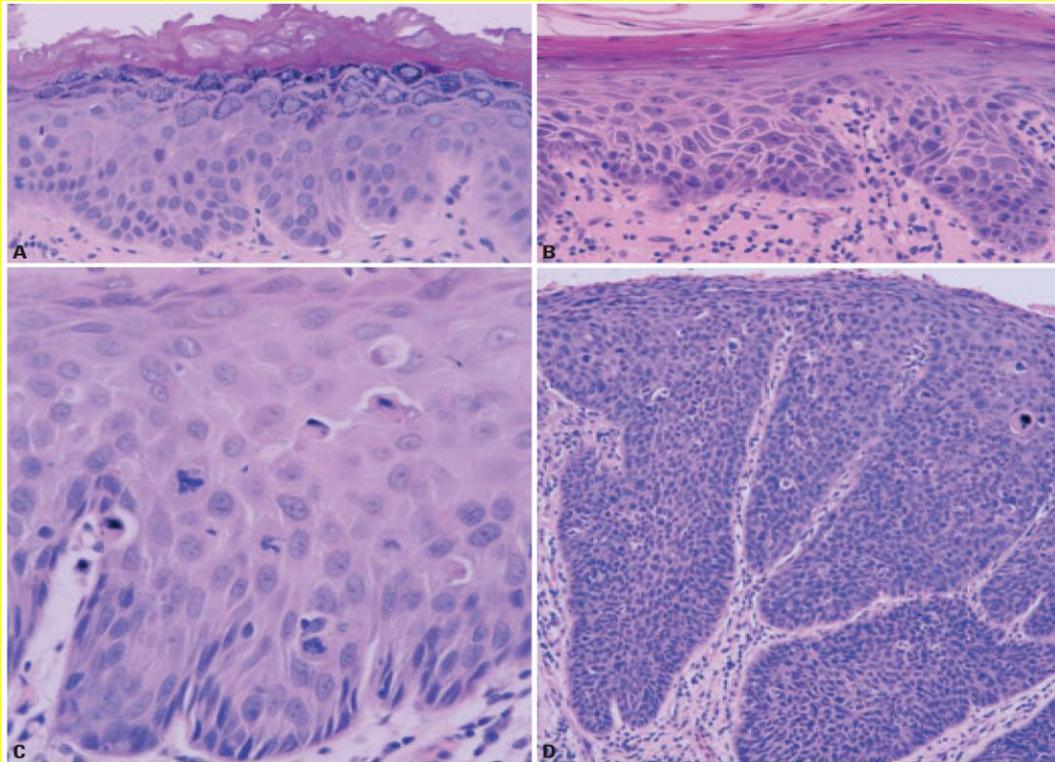
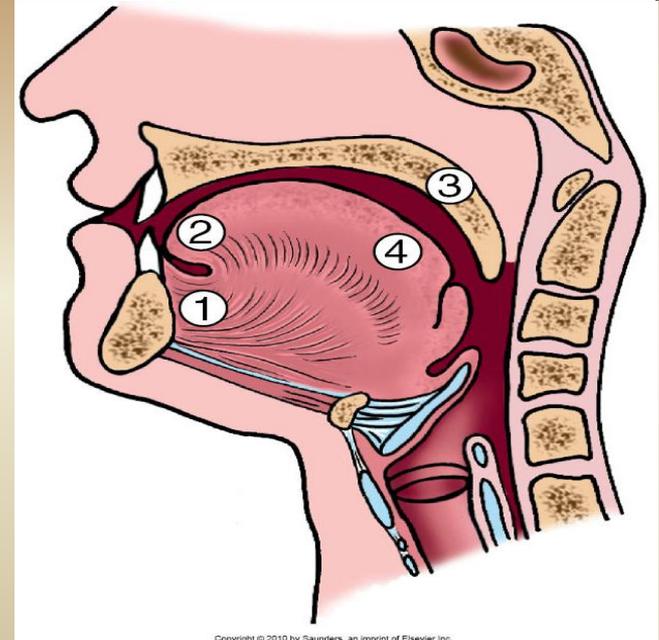


Fig. 4.15 **A** Moderate dysplasia. Drop shaped rete ridges, dysplasia extending to mid-third and moderate cytological changes **B** Severe dysplasia into upper third of epithelium with marked cytological change **C** Severe dysplasia into upper third of epithelium with prominent cytological change including abnormal mitoses. **D** Carcinoma in-situ. Abnormal cells seen throughout the full thickness of epithelium.



Eziopatogenesi:

- Autonomia proliferativa
- Insensibilità ai fattori inibenti la crescita
- Alterazione metabolismo cellulare
- Blocco apoptosi
- Immortalizzazione
- Acquisizione attività angiogenetica
- Capacità di invasione e metastasi
- Elusione del contrasto immunitario

Neoplasie del cavo orale carcinoma squamoso

Fattori di rischio:

- Fumo
- Alcool
- Consumo di bethel
- Radiazione attinica
- HPV (16, 18, ...)

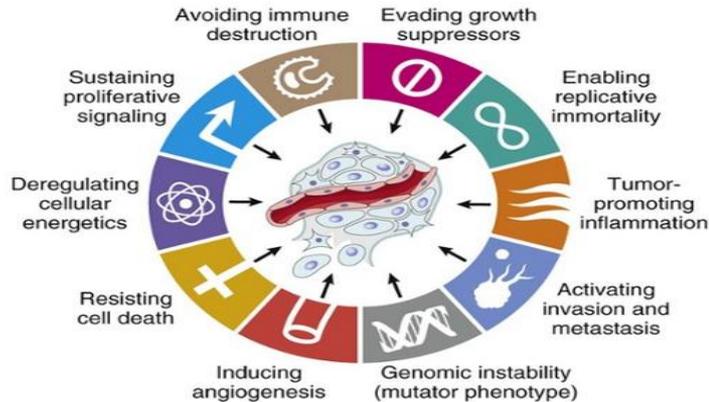


FIGURE 7-24 Hallmarks of cancer. (Adapted from Hanahan D, Weinberg RA. Hallmarks of cancer...

Fattori prognostici:

- Differenziazione
- Dimensioni
- Sede
- Metastasi
 - Contiguità
 - Continuità
 - Linfonodali
 - A distanza

Istotipi:

- Carcinoma verrucoso
- Carcinoma HPV associato
- Carcinoma squamoso «convenzionale»
- Carcinoma squamoso sarcomatoide
- Carcinoma basaloide
- Carcinoma squamoso adenoide
- Carcinoma adenosquamoso
- Carcinoma cunicolato

Neoplasie del cavo orale carcinoma squamoso



pT

- 1: dimensione max ≤ 2 cm e invasione ≤ 5 mm
- 2: dimensione max ≤ 2 cm e invasione >5 e ≤ 10 mm oppure
dimensione max > 2 e ≤ 4 cm e invasione ≤ 10 mm
- 3: dimensione max > 4 cm o invasione > 10 mm
- 4a (labbro): invasione corticale ossea, nervo alveolare inf., pav. bocca, cute mento/naso
- 4a (c. orale): invasione corticale ossea mand., seno mascellare, cute volto
- 4b: invasione sp. masticatorio, lamine pterigoidee, base cranica, inglob. carotide int.

pN

- 0: assenza di metastasi LGH regionali (cervicali omolat.)
- 1: metastasi ≤ 3 cm, singolo linfonodo, no inv. extranod.
- 2a: meta 1 LGH omolat ≤ 3 cm + inv extranodale oppure
meta 1 LGH omolat > 3 e ≤ 6 cm, senza inv extranodale
- 2b: meta più LGH omolaterali, nessuna > 6 cm, senza inv. extranodale
- 2c: meta LGH bilaterali/controlaterali, nessuna > 6 cm, senza inv. extranodale
- 3a: meta > 6 cm in 1 LGH, senza estensione extranodale
- 3b: meta > 3 cm in 1 LGH, con est. extranodale oppure
meta più LGH omolaterali o più LGH omo/controlat., con est. extranodale

M

- 0: assenza di metastasi a distanza
- 1: metastasi a distanza

UICC TNM staging system VIII ed.

Stage	TNM classification
Stage I	T1 N0 M0
Stage II	T2 N0 M0
Stage III	T3 N0 M0, or T1, T2, or T3 N1 M0
Stage IV	
Stage IVA	T4a N0 or N1 M0, or T1, T2, T3, or T4a N2 M0
Stage IVB	Any T N3 M0, or T4b any N M0
Stage IVC	Any M1 lesion

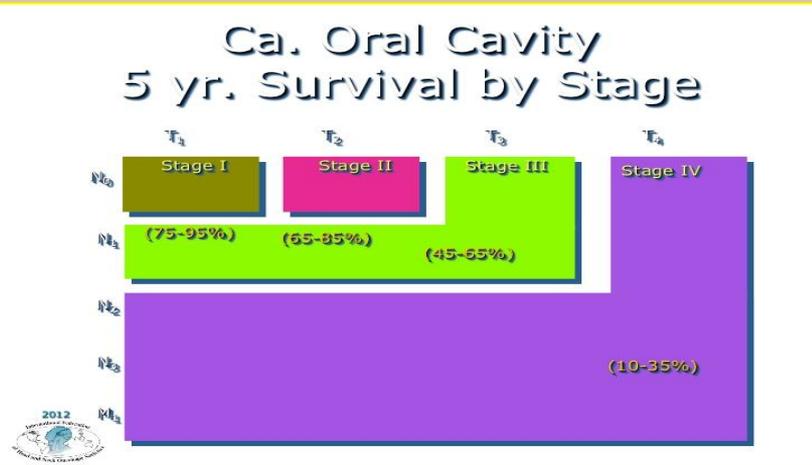
Carcinoma del labbro e cavità orale stadiazione

Tabella dei fattori prognostici

Fattori prognostici per sopravvivenza in carcinoma laringeo e ipofaringeo

Fattori prognostici	Correlati al tumore	Correlati al paziente	Correlati all'ambiente
Essenziali	Classi T, N, M Estensione extracapsulare (ECE)	Comorbilità Età >70 anni Condizioni generali	Possibilità di fornire il trattamento standard (risorse) Qualità del trattamento Margini di resezione chirurgica
Addizionali	Regioni/sottosedi coinvolte Linfonodi della parte inferiore del collo Volume del tumore Compromissione delle corde vocali Tracheostomia	Sesso Funzione laringea	Nutrizione Fattori sociali/ambientali (per esempio stazioni anatomiche) Durata complessiva del trattamento
Innovativi e promettenti	Marker tumorali: TP53, VEGF, amplificazione ciclina D1, EGFR, Bcl-2 Stato HPV del tumore Geni responsabili di chemioresistenza	Qualità della vita al basale	Imaging ottico Nuovi sensibilizzanti per terapia fotodinamica

Fonte: UICC Manual of Clinical Oncology, Ninth Edition. Edited by Brian O'Sullivan, James D. Brierley, Amil K. D'Cruz, Martin F. Fey, Raphael Pollock, Jan B. Vermorken and Shao Hui Huang. © 2015 UICC. Published 2015 by John Wiley & Sons, Ltd.



Localizzazione

Ubiquitaria

AP macroscopica

Massa rilevata/ulcerata

AP Microscopica

Invasione diffusa «single cells»

Reazione desmoplastica

Presenza di linfociti ed eosinofili

Cheratosi nelle forme differenziate (perle)

Talora aspetto indifferenziato

Frequente sovrainfezione candidosica

Trattamento

Chirurgia

SLNB

Radioterapia

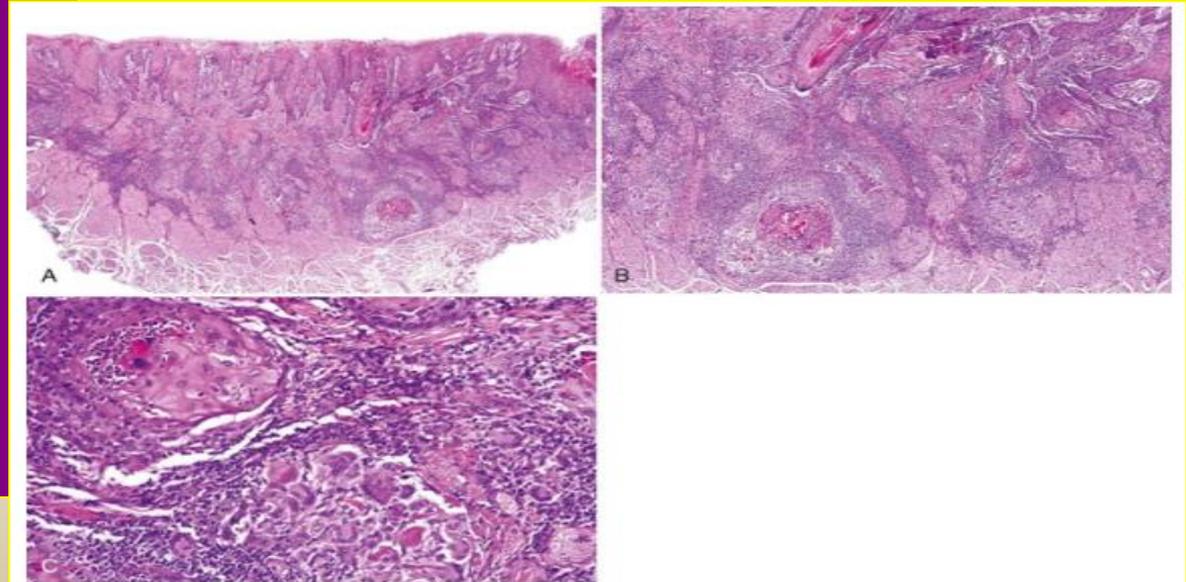
Chemioterapia

Target therapy (EGFR)

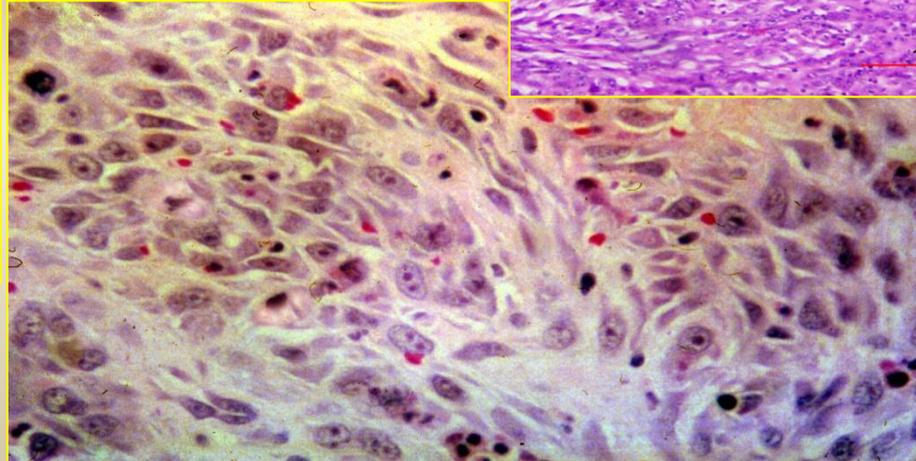
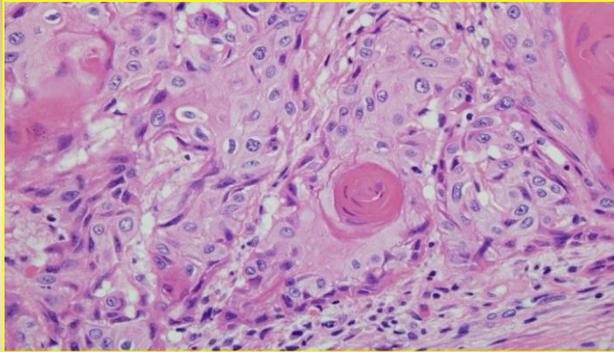
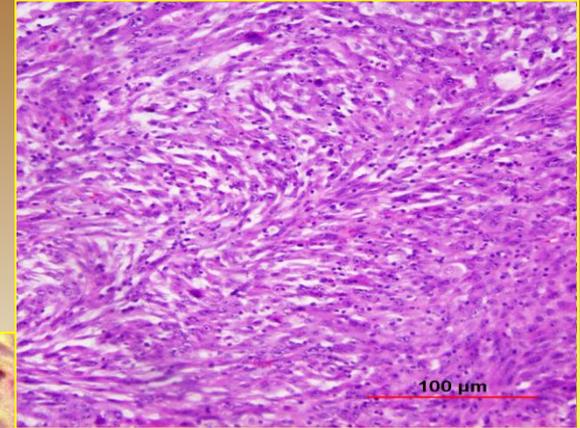
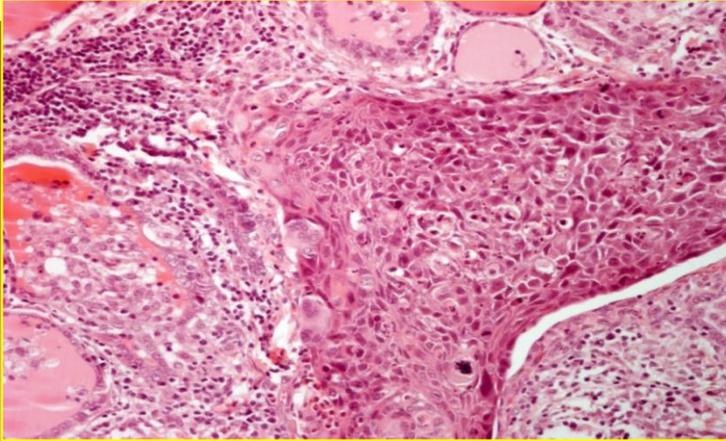
Prognosi

Mediocre (OS 3 yr: 57%)

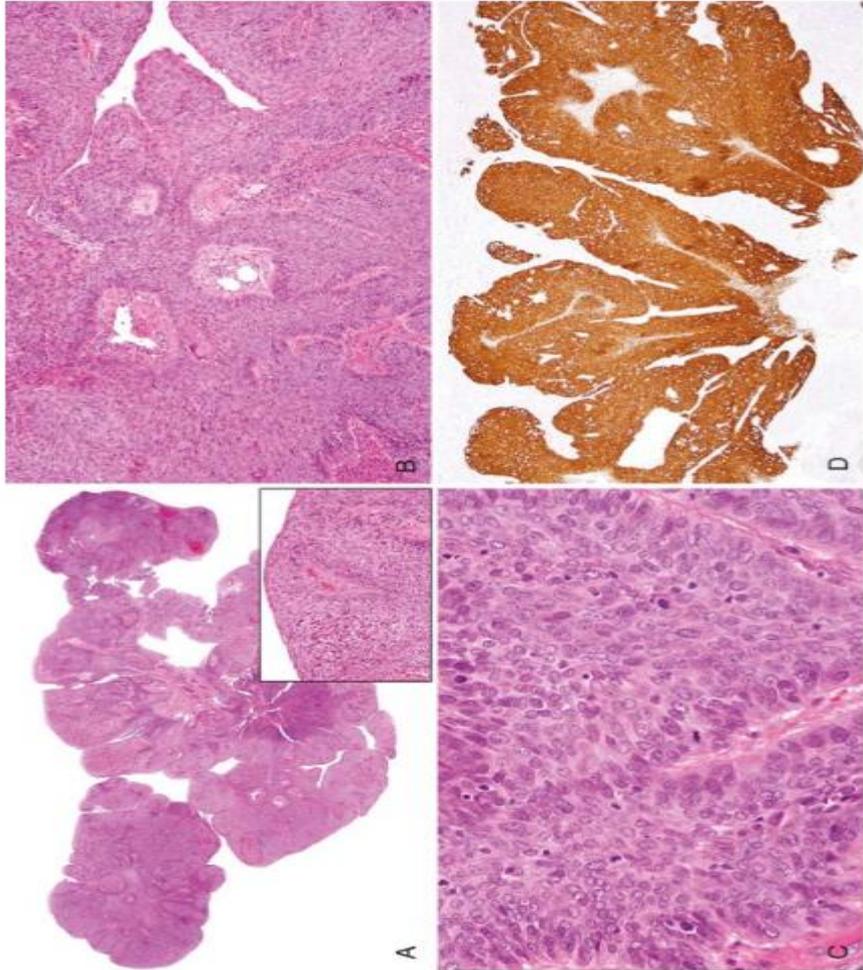
Neoplasie del cavo orale carcinoma squamoso NAS



**Neoplasie del cavo orale
carcinoma squamoso NAS**



Neoplasie del cavo orale carcinoma squamoso HPV-associato



Localizzazione

Cavo orale

Orofaringe (frequente)

Tonsille

AP Microscopica

Lesione non cheratinizzante

Cellule a citopl. indistinto, chiaro

Nuclei ovoidi, piccoli nucleoli, cromatina dispersa

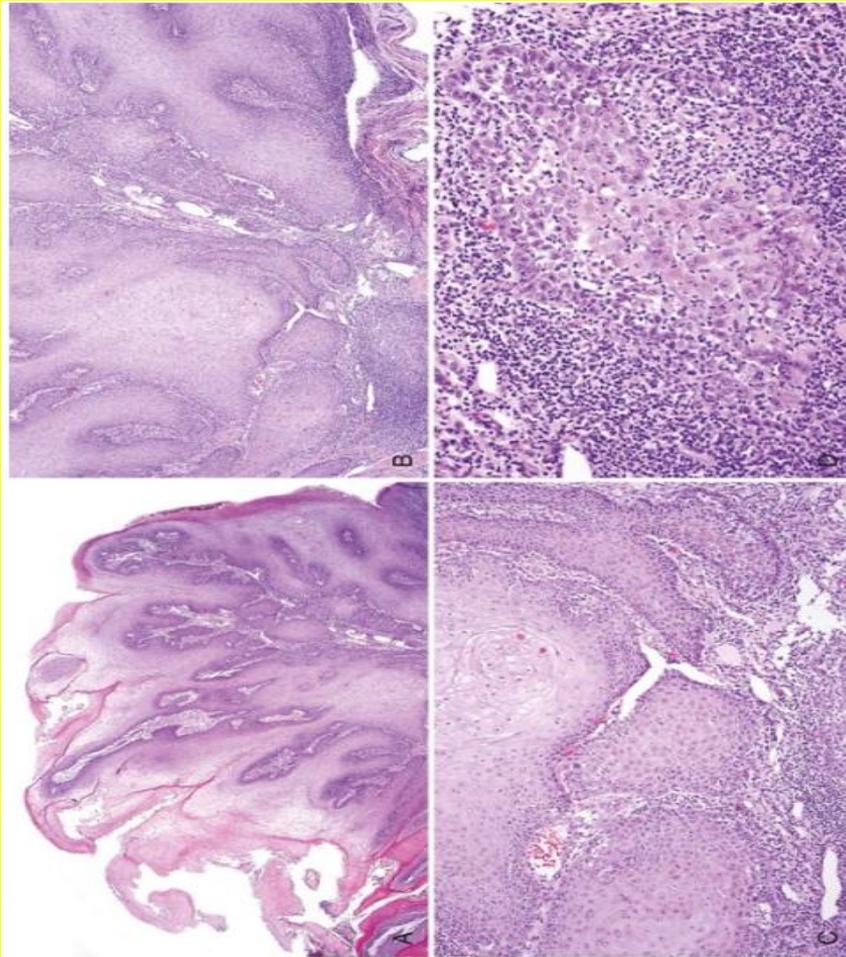
Positività HPV (16, 18, 31, 33, 35,...)

p16+

Prognosi

Buona (OS 3 yr: 82%)

Neoplasie del cavo orale carcinoma squamoso verrucoso



Localizzazione

Cresta dentale
Solco mandibolare
Mucosa bocca

AP macroscopica

Formazione papillare esofitica

AP Microscopica

Papille con paracheratosi
Fronte invasivo regolare (pushing)
Minime atipie citologiche
Talora infiltrazione linfocitica basale

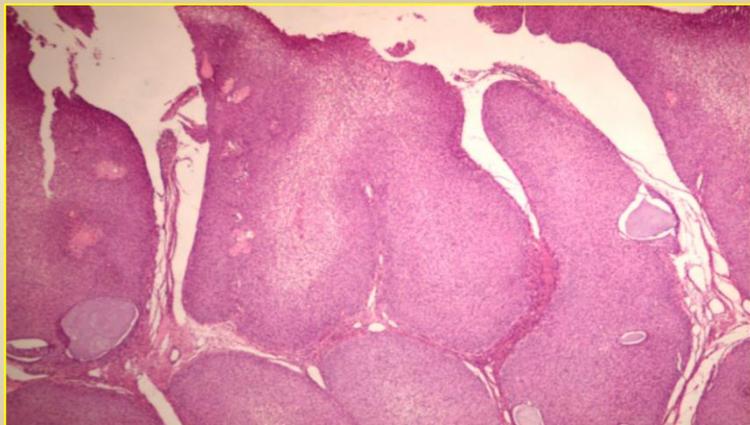
Biologia

Talora HPV positivo

Trattamento

Chirurgico
Radioterapia controversa (trasformazione anaplastica)

Neoplasie del cavo orale
carcinoma squamoso verrucoso



Localizzazione

Cavo orale posteriore
Talora post-radioterapia

AP macroscopica

Massa polipoide ulcerata

AP Microscopica

Popolazione bifasica epitelioid/fusata
Pattern storiforme
Cellule fusate p63+
Aspetti cheratinizzanti marginali

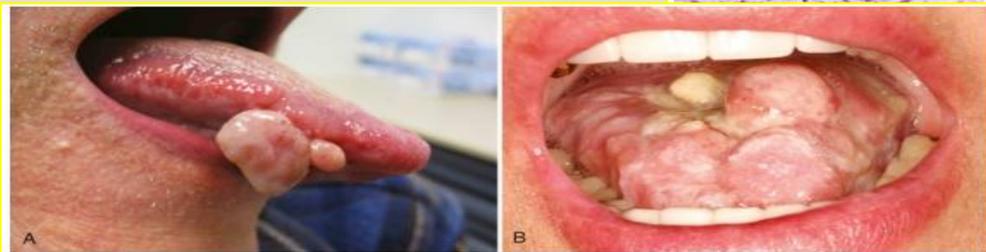
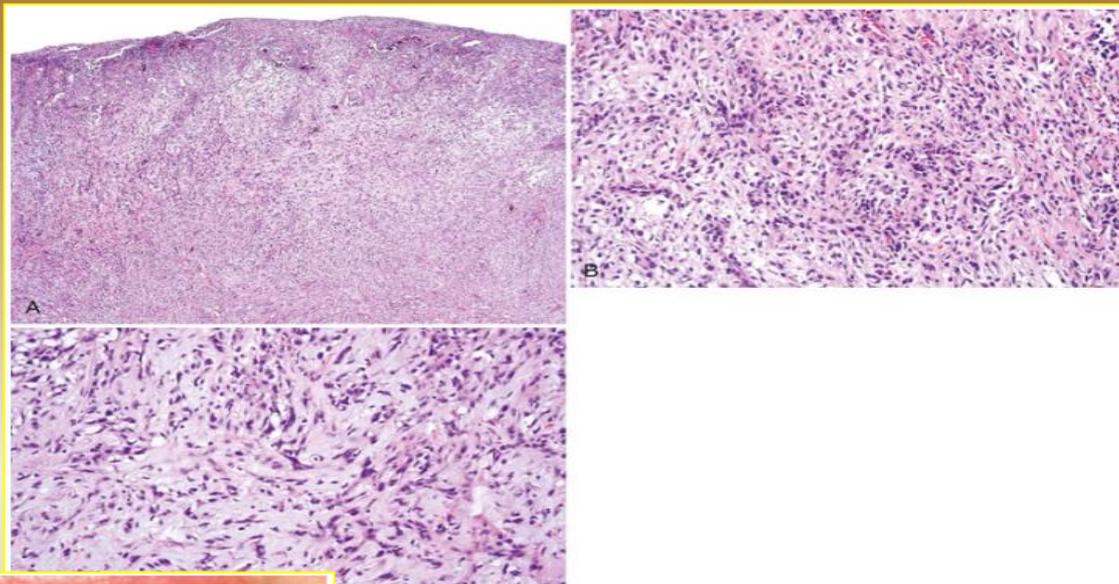
DD

Melanoma desmoplastico
Fascite nodulare
Modificazioni post-radioterapia

Prognosi

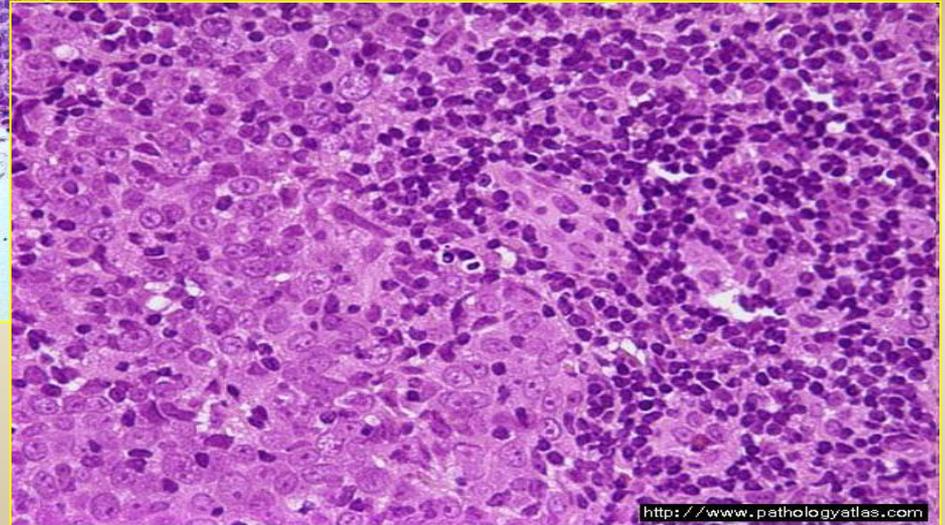
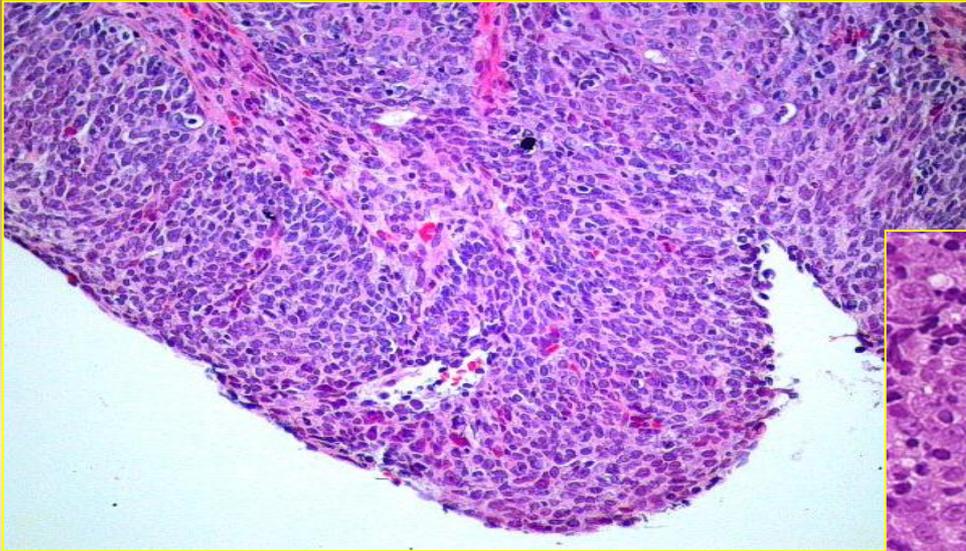
Severa (OS 3 yr: 25-30%)

Neoplasie del cavo orale carcinoma squamoso sarcomatoide



EBV associato

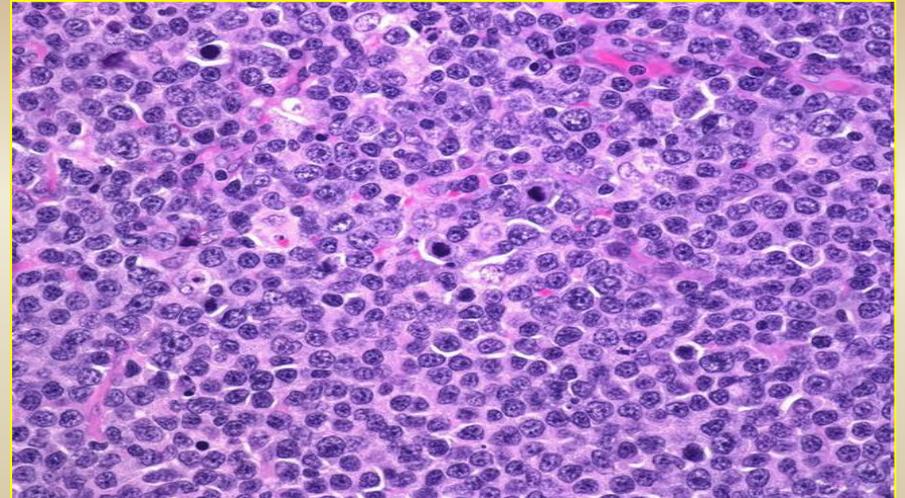
**Neoplasie dell'orofaringe
carcinoma nasofaringeo**



EBV associated



**Neoplasie dell'orofaringe
linfoma di Burkitt**

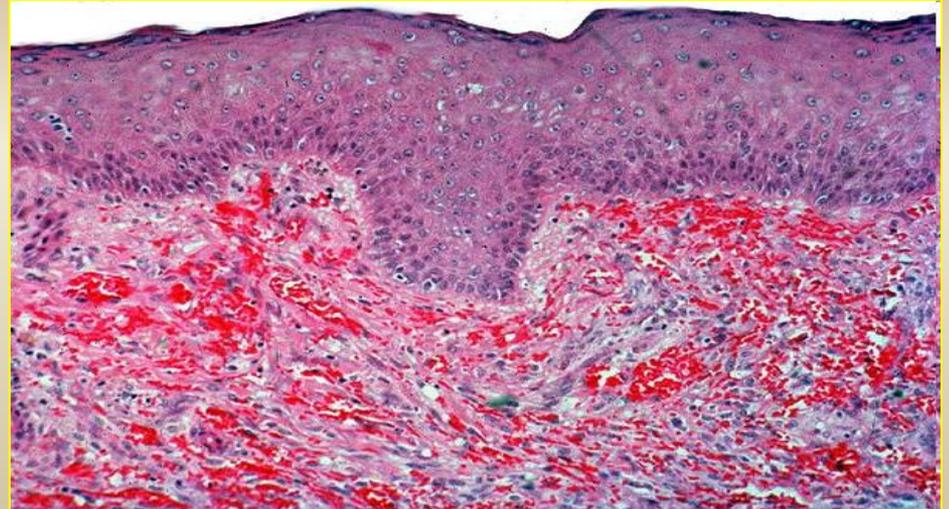




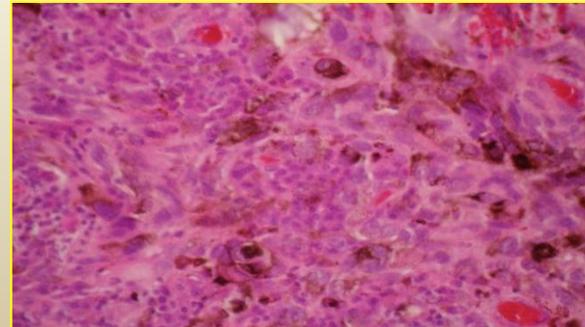
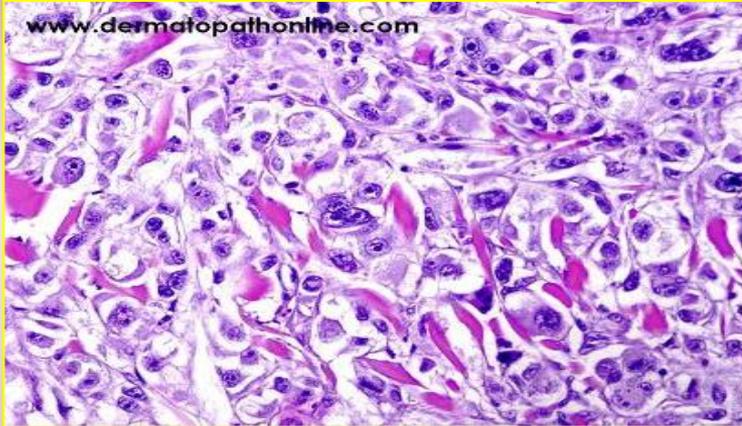
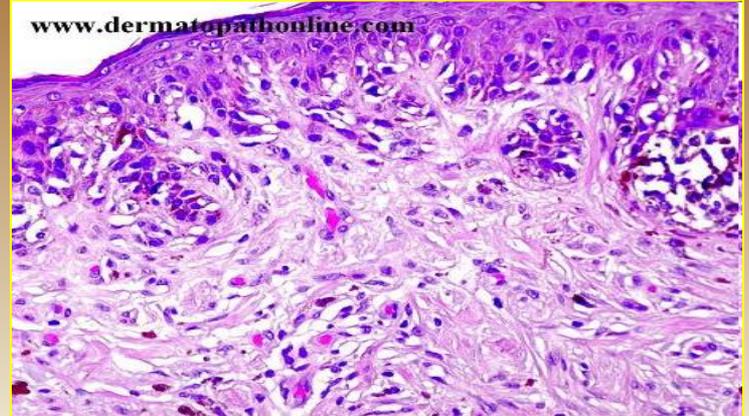
Neoplasie dell'orofaringe
sarcoma di Kaposi

Agente eziologico:
HHV- 8

Variante
HIV associated



Neoplasie dell'orofaringe melanoma



Carcinomi

Carcinomi in cisti odontogene
Carcinoma odontogeno
Carcinoma intraosseo primitivo
Carcinoma ameloblastico
Ameloblastoma maligno

Sarcomi

Fibrosarcomi

Neoplasie del cavo orale neoplasie “maligne” odontogene

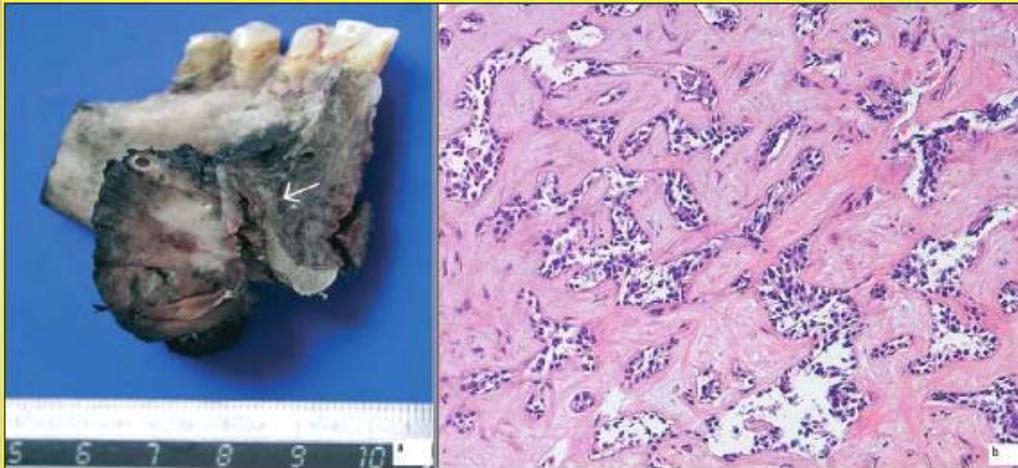
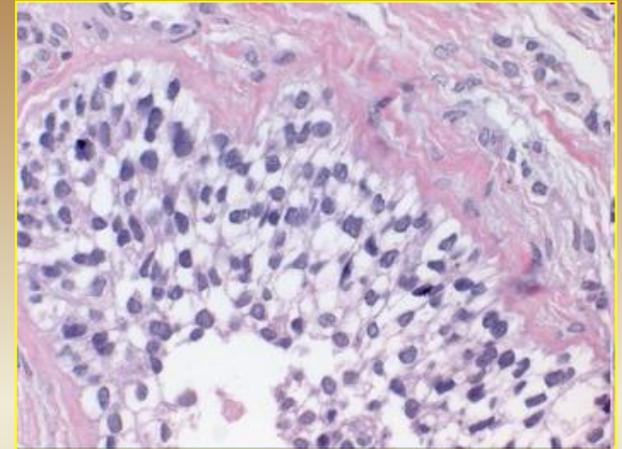
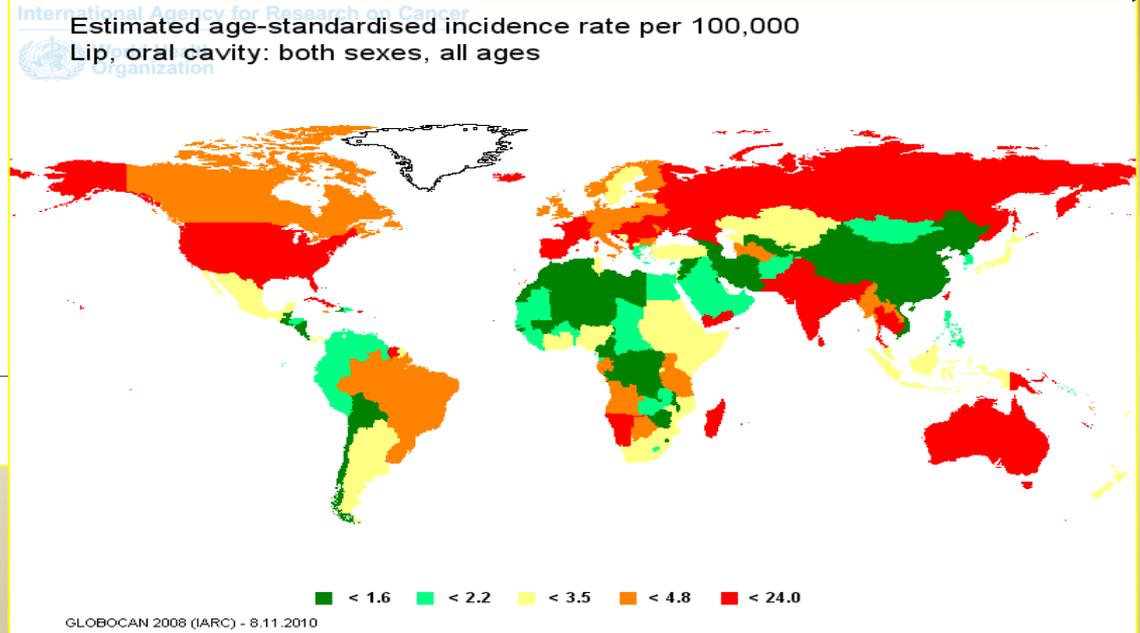
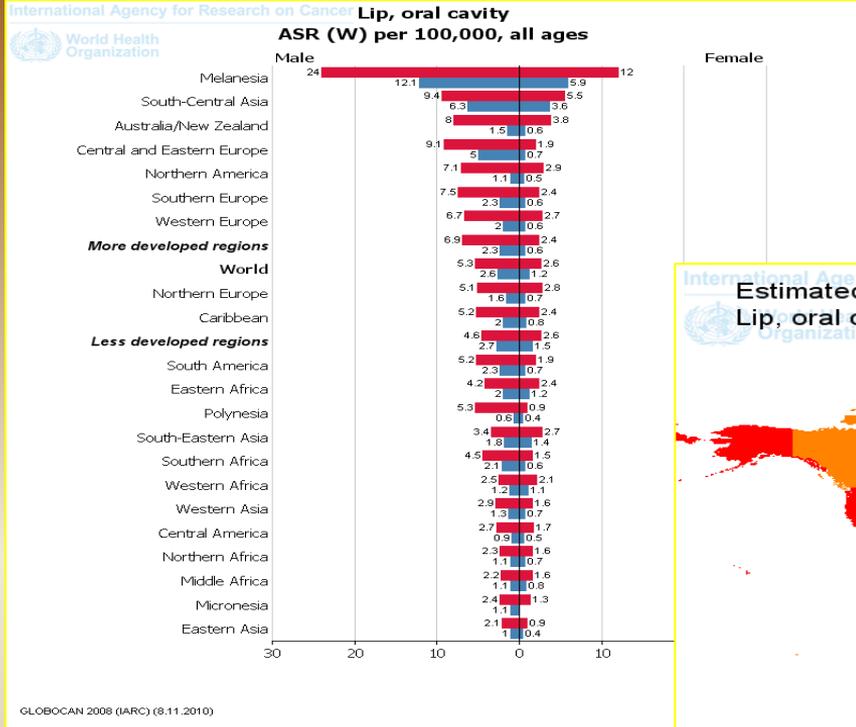
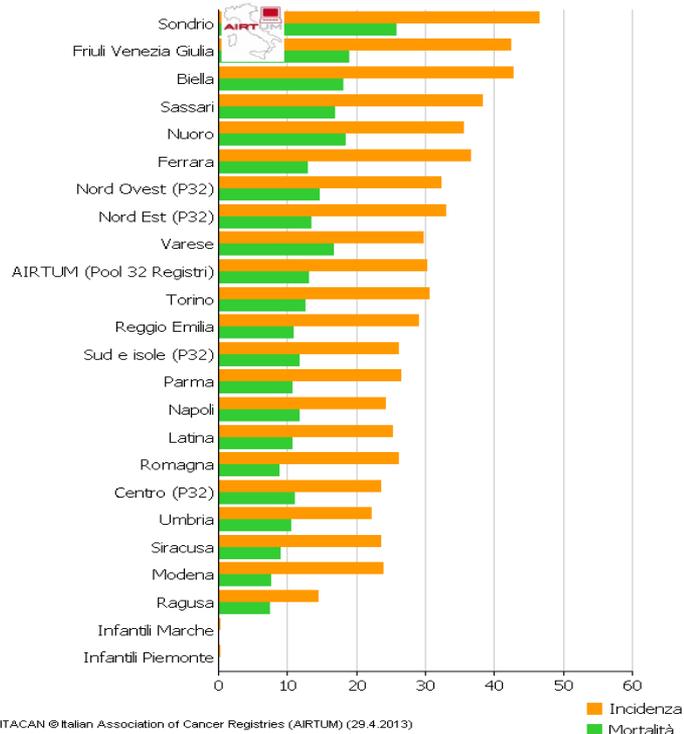


Figura 2. a: Hemimandibulectomia inferior que muestra lesión tumoral blanco grisácea, de bordes mal delimitados, que destruye el hueso (cuerpo mandibular) e infiltra el tejido muscular esquelético. b: Ameloblastoma maligno variedad folicular, con extensas zonas de infiltración perineural y destrucción ósea del cuerpo de la mandíbula.

Neoplasie del settore testa-collo incidenza



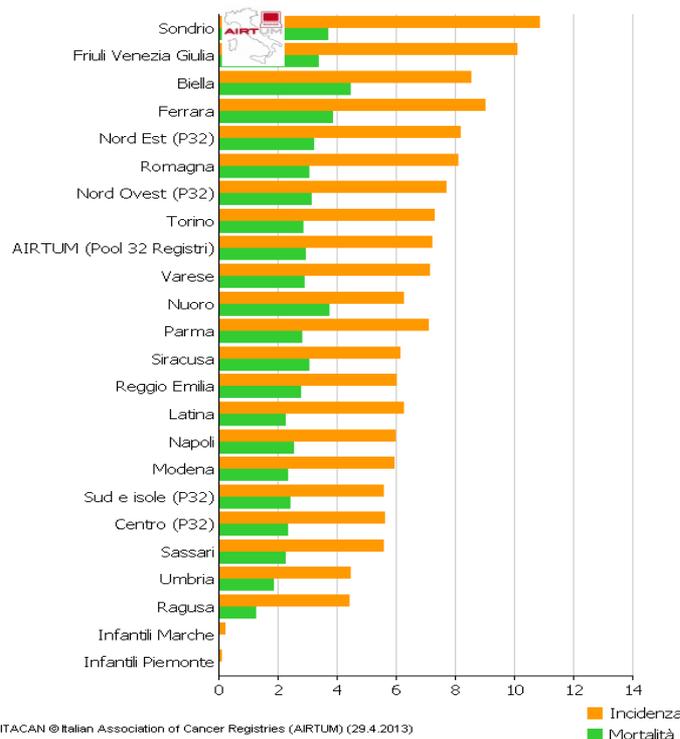
Testa e collo (2005-2007)
Maschi: TSE (Italiana) età (0-85+)



ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

Neoplasie del settore testa-collo incidenza e mortalità in Italia

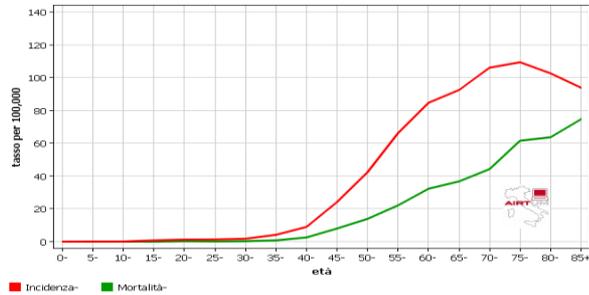
Testa e collo (2005-2007)
Femmine: TSE (Italiana) età (0-85+)



ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

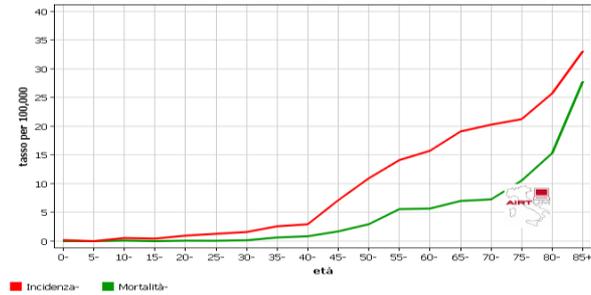
Neoplasie del settore testa-collo incidenza e mortalità in Italia

AIRTUM (Pool 32 Registri) (2005-2007)
Testa e collo: Maschi



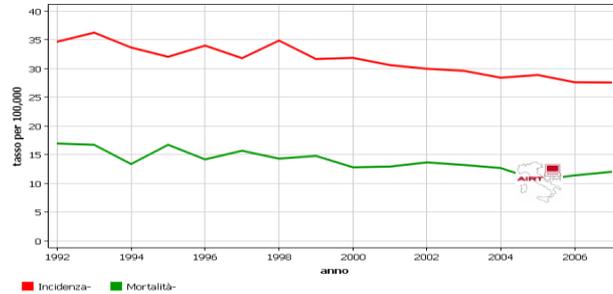
ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

AIRTUM (Pool 32 Registri) (2005-2007)
Testa e collo: Femme



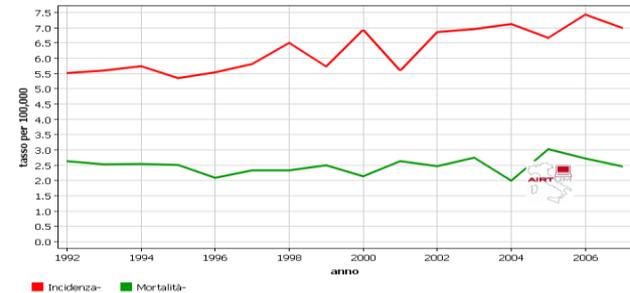
ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

AIRTUM (Pool 9 Registri)
Testa e collo
TSE (Italiana), Maschi età (0-85+)



ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

AIRTUM (Pool 9 Registri)
Testa e collo
TSE (Italiana), Femme età (0-85+)



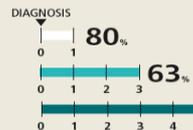
ITACAN © Italian Association of Cancer Registries (AIRTUM) (29.4.2013)

SUMMARY

NET SURVIVAL

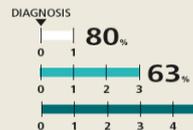
1 YEAR AFTER DIAGNOSIS

80%



3 YEARS AFTER DIAGNOSIS

63%



5 YEARS AFTER DIAGNOSIS

57%

7 percent points higher in 15 years

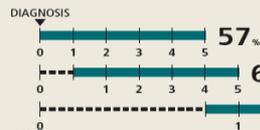


MALES & FEMALES, PERIOD OF DIAGNOSIS: 2005-2009

CONDITIONAL 5-YEAR NET SURVIVAL

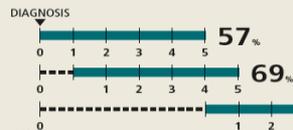
5 YEARS AFTER DIAGNOSIS

57%



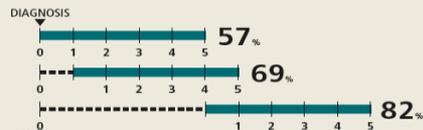
CONDITIONED ON HAVING SURVIVED 1 YEAR AFTER DIAGNOSIS

69%



CONDITIONED ON HAVING SURVIVED 5 YEARS AFTER DIAGNOSIS

82%



5-YEAR NET SURVIVAL - GEOGRAPHICAL COMPARISONS

Source: graph A

NORTH WEST

M 57%
F 59%

NORTH EAST

M 56%
F 59%

CENTRE

M 57%
F 54%

SOUTH

M 56%
F 57%

NO SIGNIFICANT DIFFERENCES in cancer survival across ITALY

5-YEAR NET SURVIVAL BY GENDER

MALES

57%

NO SIGNIFICANT DIFFERENCE in cancer survival

FEMALES

58%

LIFE EXPECTANCY AT A GIVEN AGE OF DIAGNOSIS

GENERAL POPULATION (light blue bar), CANCER (dark blue bar)



5-YEAR NET SURVIVAL - BY REGION

Source: graph E

REGIONS

- ABOVE THE NATIONAL MEAN
- AROUND THE NATIONAL MEAN
- BELOW THE NATIONAL MEAN
- WITHOUT DATA



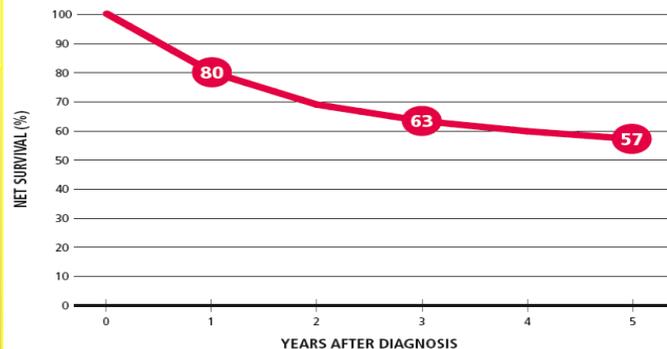
Neoplasie del settore testa-collo sopravvivenza in Italia



HEAD AND NECK CANCER

MALES & FEMALES

AGE-STANDARDIZED NET SURVIVAL (%), 2005-2009
POOL OF 42 CANCER REGISTRIES (25259 OBSERVED CASES)

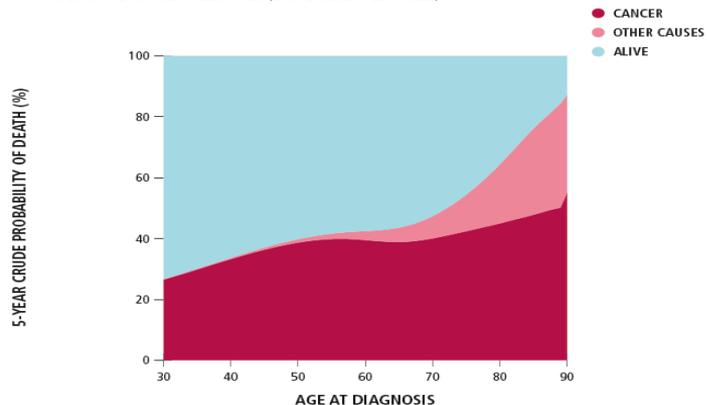


HEAD AND NECK CANCER

MALES & FEMALES

D CRUDE PROBABILITY OF DEATH (%), 2005-2009

POOL OF 42 CANCER REGISTRIES (24872 OBSERVED CASES)

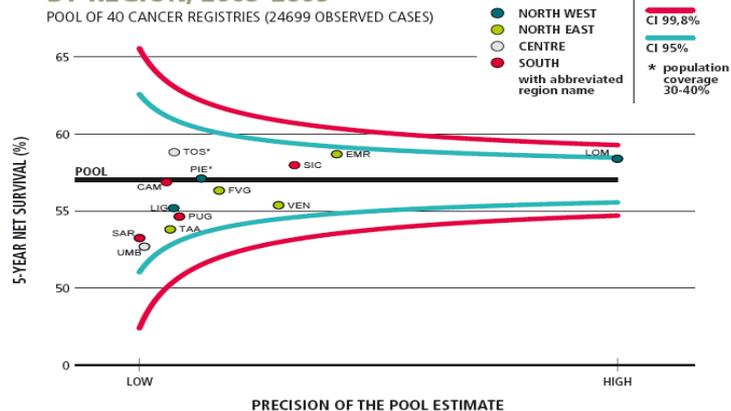


HEAD AND NECK CANCER

MALES & FEMALES

E 5-YEAR AGE-STANDARDIZED NET SURVIVAL (%) BY REGION, 2005-2009

POOL OF 40 CANCER REGISTRIES (24699 OBSERVED CASES)



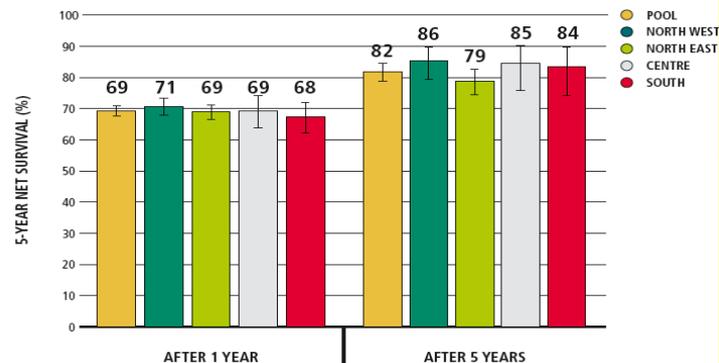
Neoplasie del settore testa-collo sopravvivenza in Italia

HEAD AND NECK CANCER

MALES & FEMALES

F CONDITIONAL 5-YEAR AGE-STANDARDIZED NET SURVIVAL AFTER SURVIVING 1, 5 YEARS (%)

POOL OF 26 CANCER REGISTRIES (21463 OBSERVED CASES)

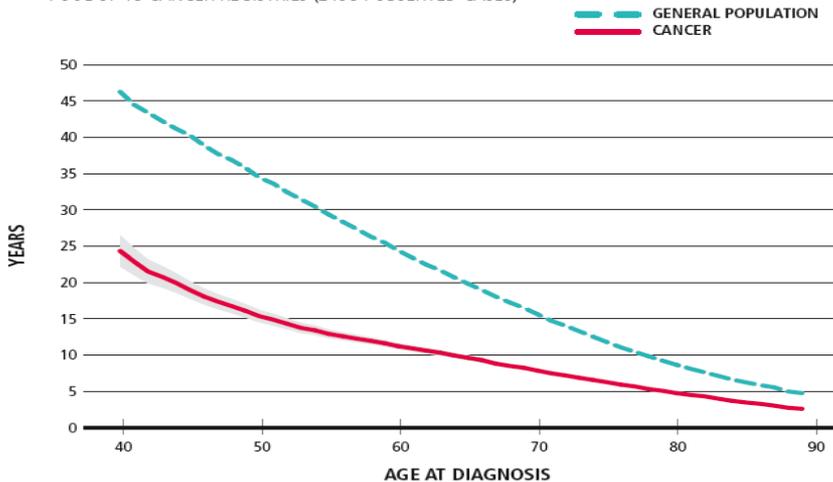


HEAD AND NECK CANCER

MALES & FEMALES

G LIFE EXPECTANCY

POOL OF 18 CANCER REGISTRIES (2 1954 OBSERVED CASES)



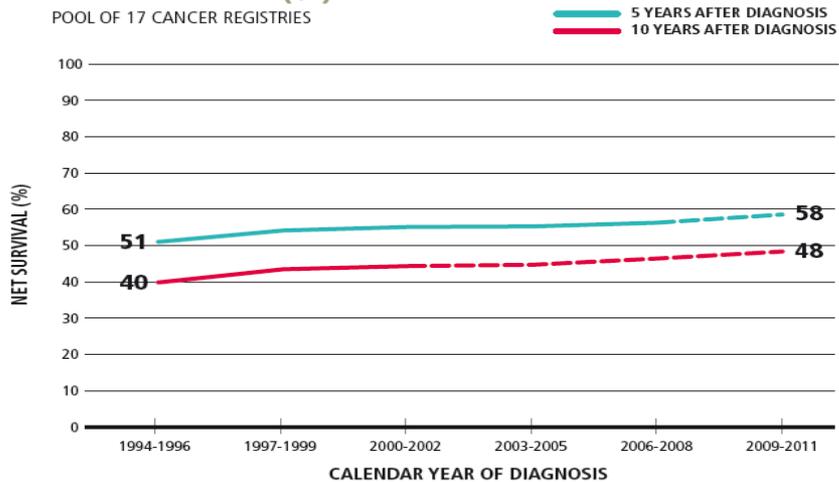
Neoplasie del settore testa-collo sopravvivenza in Italia

HEAD AND NECK CANCER

MALES & FEMALES

H TIME TREND OF 5-, 10-YEAR AGE-STANDARDIZED NET SURVIVAL (%)

POOL OF 17 CANCER REGISTRIES





Pool of Italian Cancer Registries - 1 January 2010

BOCCA MOUTH (ICD-10 C03-06)

COMPLETE PREVALENCE BY YEARS SINCE DIAGNOSIS

YEARS →	≤ 2	(2 - 5]	(5 - 10]	(10 - 15]	(15 - 20]	> 20
No. →	2 509	2 073	2 595	1 569	832	1 171
% →	23%	19%	24%	15%	8%	11%
PROPORTION PER 100 000 →	5	4	5	3	2	2
	MALE 60%			FEMALE 40%		

2 587 347 CANCER SURVIVORS

10 749
MOUTH
CANCER SURVIVORS

ALREADY CURED
at 1 JANUARY 2010

564 (5%)

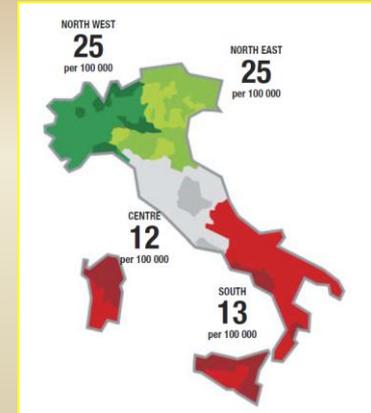
Neoplasie del settore testa-collo sopravvivenza e guarigione in Italia



CURE FRACTION AND TIME TO CURE BY AGE AT DIAGNOSIS AND SEX FOR CANCER PATIENTS DIAGNOSED IN ITALY IN 1985-2009

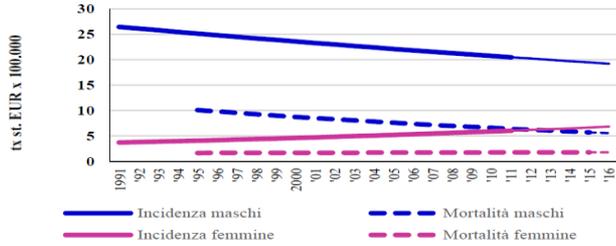
AGE AT DIAGNOSIS	CURE FRACTION %	
	MALE	FEMALE
0 - 44	19%	51%
45 - 59	14%	36%
60 - 74	9%	21%
75+	7%	14%

AGE AT DIAGNOSIS	TIME TO CURE YEARS	
	MALE	FEMALE
0 - 44	>25	15
45 - 59	>25	18
60 - 74	>25	22
75+	>25	23



Neoplasie del settore testa-collo in Emilia-Romagna

Andamenti temporali



Anni	maschi		femmine	
	incidenza	mortalità	incidenza	mortalità
Anni 1991-2011				
1995-2015				
APC	-1.27*	-2.82*	+2.50*	+0.38

APC: variazione percentuale annua * p<0,05

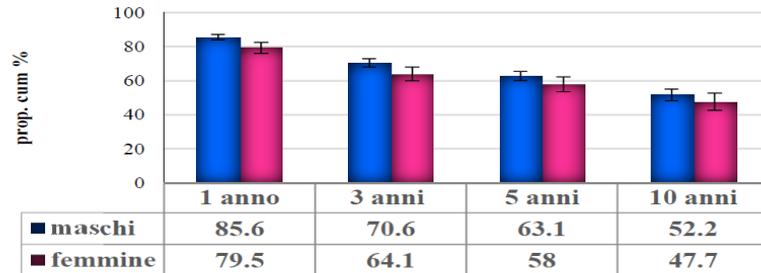
Casi prevalenti residenti in Emilia-Romagna

Stima al 1.1.2012, per anni trascorsi dalla diagnosi

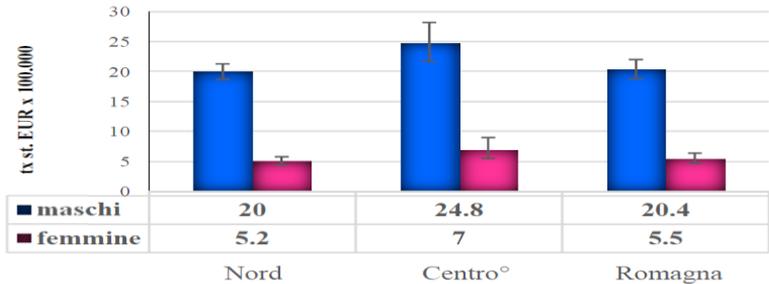
Anni	casi	% prop.x10 ⁵	M/F
<=2	234	32%	5.2 / 1.5
2-5	182	25%	4.1 / 1.4
5-10	202	27%	4.5 / 1.1
10-15	119	16%	2.7 / 1.2
<=15	737	100%	16.5 / 1.3



Sopravvivenza relativa



Confronti incidenza tra Aree Vaste



^oProvincia di Ferrara



Continua...

