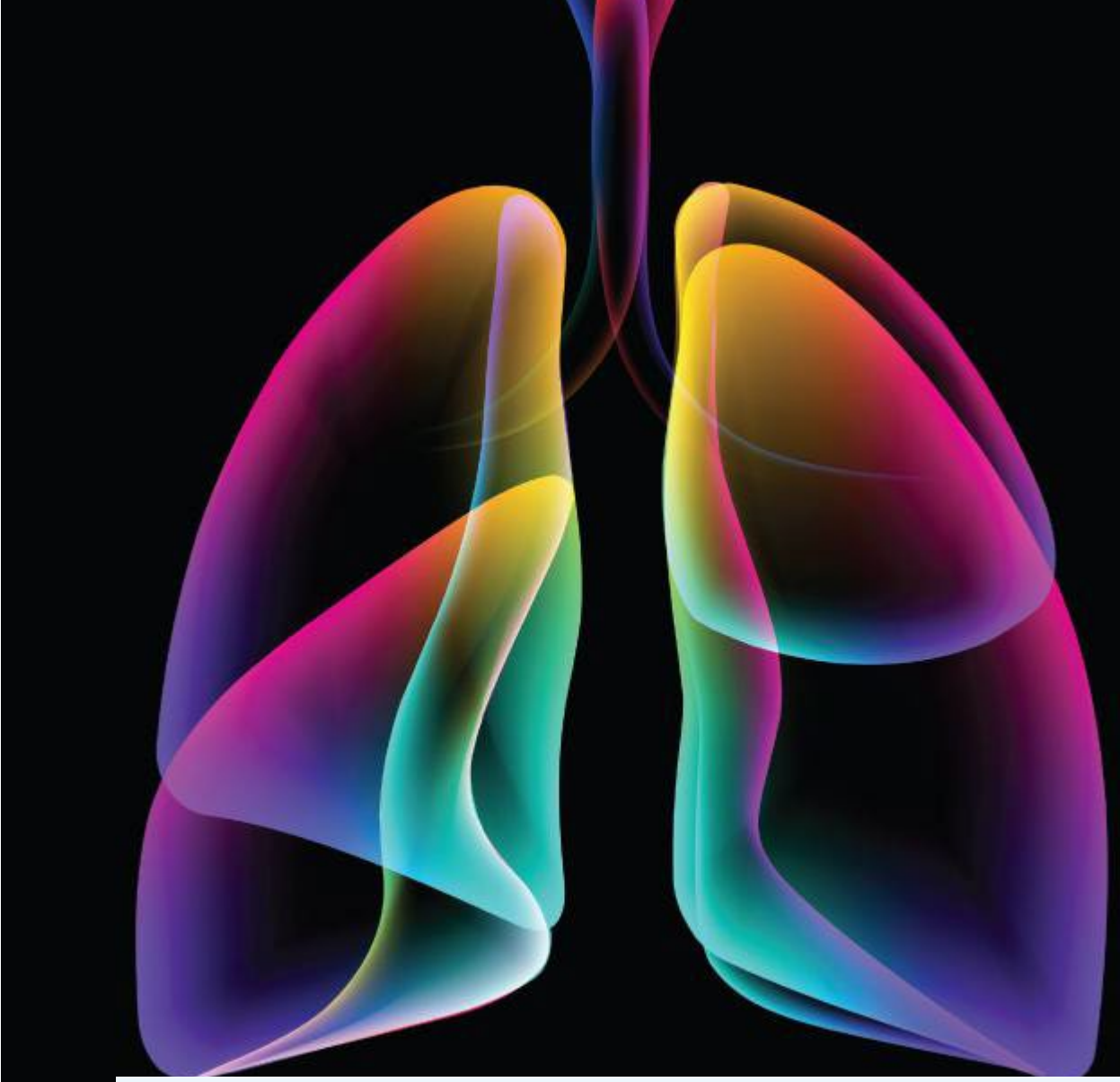


22 & 23
ΜΑΪΟΥ
2025

ΠΡΟΣΚΛΗΣΗ
σε Εκπαιδευτικό Σεμινάριο

Η Αρχή Αντιμετώπιση Εξαρτήσεων Κύπρου (ΑΑΕΚ)
και η Πνευμονολογική Εταιρεία Κύπρου
σας προσκαλούν σε Εκπαιδευτικό σεμινάριο
για την πρόληψη και διακοπή του καπνίσματος
στις **22 Μαϊου** (15:00-18:00)
και **23 Μαϊου** (9:00-15:00) **2025**
Πανεπιστήμιο Λευκωσίας,
Αμφιθέατρο Jean Monnet (M 203)

ΑΡΧΗ ΑΝΤΙΜΕΤΩΠΙΣΗΣ
ΕΞΑΡΤΗΣΕΩΝ ΚΥΠΡΟΥ

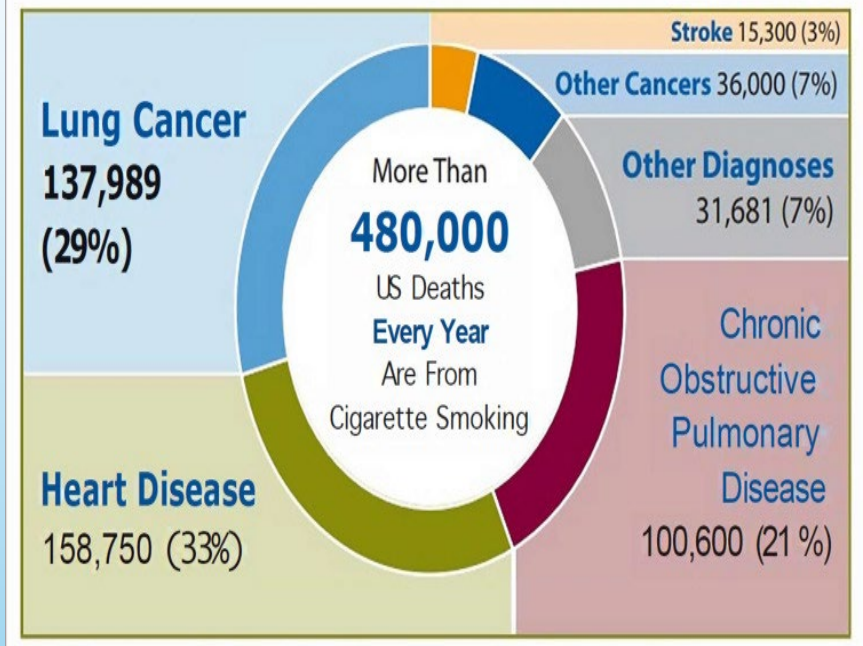
ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ ΚΥΠΡΟΥ
1993
CYPRUS RESPIRATORY SOCIETY

Άμεσα αποτελέσματα διακοπής καπνίσματος
Κλεομένης Δ. Μπενίδης MD PhD
Πνευμονολόγος Φυματιολόγος

World Health Organization

Tobacco is an important public health issue and the single most preventable cause of illness and death in the world. The latest research suggests that smoking-related mortality has risen to 7.2 million lives annually, killing more people than AIDS, malaria and tuberculosis combined.

Globally, the WHO European Region has the highest prevalence of tobacco smoking among adults (28%), including one of the highest smoking prevalence rates among women (19%). In addition to causing illness and death, tobacco is a driver of health inequities.



- Η 31η Μαΐου ορίστηκε από τον Παγκόσμιο οργανισμό υγείας ως παγκόσμια ημέρα κατά του καπνίσματος. Είναι μία ημέρα υπενθύμισης για τις βλαβερές συνέπειες της καπνιστικής συνήθειας και την ανάγκη διακοπής της.

- Πέρα από το αρνητικό περιβαλλοντικό αποτύπωμα της καλλιέργειας του καπνού ο λόγος είναι ότι το κάπνισμα ευθύνεται για περισσότερους θανάτους από πολλούς άλλους παράγοντες καθώς προκαλεί καρδιοπάθειες, χρόνια αποφρακτική πνευμονοπάθεια και καρκίνο του πνεύμονα που αποτελούν τις κύριες αιτίες θανάτου παγκοσμίως.

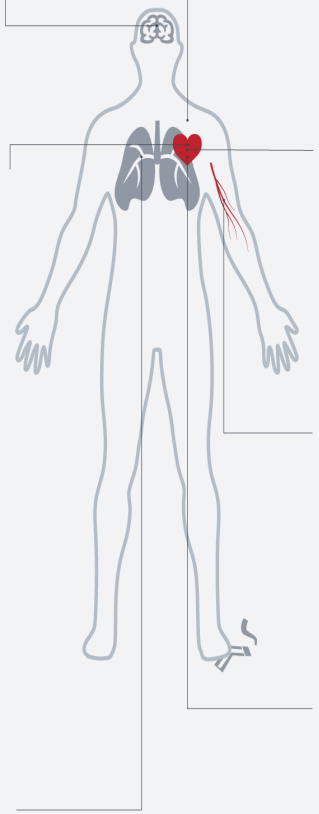
- Ο καπνός του τσιγάρου είναι εκνέφωμα με πάνω από 4.000 διαφορετικές χημικές ενώσεις εκ των οποίων τουλάχιστον 50 είναι καρκινογόνες. Το κάπνισμα σχετίζεται με ένα θάνατο κάθε 6 δευτερόλεπτα και συνολικά με 7.2 εκατομμύρια θανάτους / έτος.

- Στην Ελλάδα το κάπνισμα ευθύνεται άμεσα ή έμμεσα για 25% των θανάτων των ανδρών και 7,5% των θανάτων των γυναικών. Το παθητικό κάπνισμα δεν εξαιρείται από τις αρνητικές επιπτώσεις στην υγεία των μη καπνιστών.

World Health Organization

The effects of quitting smoking

Health improvements and time required



20 minutes
Heart rate, blood pressure drop

1 year
Risk of coronary heart disease half of that of a smoker

12 hours
Carbon monoxide level in blood drops to normal

2-12 weeks
Circulation improves, lung function increases

1-9 months
Coughing and shortness of breath decrease

10 years
The risk of lung cancer is halved

15 years
Risk of coronary heart disease reduced to level of person who has never smoked

5-15 years
Risk of stroke reduced to that of non-smoker

Source: WHO

- **20 λεπτά**
  Η πίεση του αίματος και οι σφυγμοί σας επιστρέφουν σε φυσιολογικά επίπεδα.
- **8 ώρες**
  Τα επίπεδα οξυγόνου στο αίμα επιστρέφουν σε φυσιολογικά επίπεδα και τα επίπεδα της νικοτίνης και του μονοξειδίου του άνθρακα μειώνονται πάνω από το μισό.
- **24 ώρες**
  Το μονοξείδιο του άνθρακα έχει αποβληθεί πλήρως από το σώμα σας. Οι πνεύμονες αρχίζουν να αποβάλλουν τα κατάλοιπα του τσιγάρου μέσω της βλέννης.
- **48 ώρες**
  Η νικοτίνη έχει αποβληθεί πλήρως από το σώμα σας. Η αίσθηση της γεύσης και της όσφρησης βελτιώνονται σημαντικά.
- **72 ώρες**
  Η αναπνοή γίνεται ευκολότερη. Οι βρόγχοι αρχίζουν να χαλαρώνουν και τα επίπεδα ενέργειας αυξάνονται.
- **2 έως 12 εβδομάδες**
  Βελτιώνεται η κυκλοφορία του αίματος σε όλο το σώμα διευκολύνοντας σημαντικά το βάδισμα και το τρέξιμο.
- **3 έως 9 μήνες**
  Ο βήχας, η δύσπνοια και τα αναπνευστικά προβλήματα υποχωρούν αισθητά, καθώς η λειτουργία των πνευμόνων σας έχει αυξηθεί μέχρι και 10%.
- **1 χρόνος**
  Ο κίνδυνος καρδιακής προσβολής μειώνεται κατά το ήμισυ.
- **5 χρόνια**
  Ο κίνδυνος καρδιακής προσβολής μειώνεται σχεδόν στο μισό σε σύγκριση με τον κίνδυνο που αντιμετωπίζει ένας καπνιστής.
- **10 χρόνια**
  Ο κίνδυνος εμφάνισης καρκίνου του πνεύμονα μειώνεται στο μισό σε σύγκριση με τον κίνδυνο που αντιμετωπίζει ένας καπνιστής. Οι πιθανότητες καρδιακής προσβολής είναι ίδιες με αυτές ενός ανθρώπου που δεν έχει καπνίσει ποτέ.

Επίσης, με τη διακοπή του καπνίσματος θα παρατηρήσετε τις εξής διαφορές στο σώμα σας:[2]

- Βελτιωμένη γονιμότητα
- Πιο υγιές δέρμα
- Λευκότερα δόντια και πιο δροσερή και ευχάριστη αναπνοή
- Απαλότερα και πιο λαμπερά μαλλιά

The effects of quitting smoking

Health improvements and time required

20 minutes
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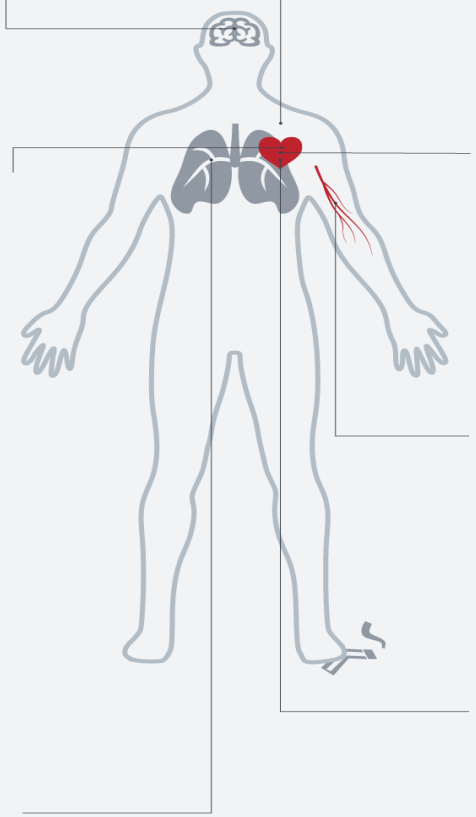


Source: WHO

ΕΛΛΗΝΙΚΗ ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ
HELLENIC THORACIC SOCIETY

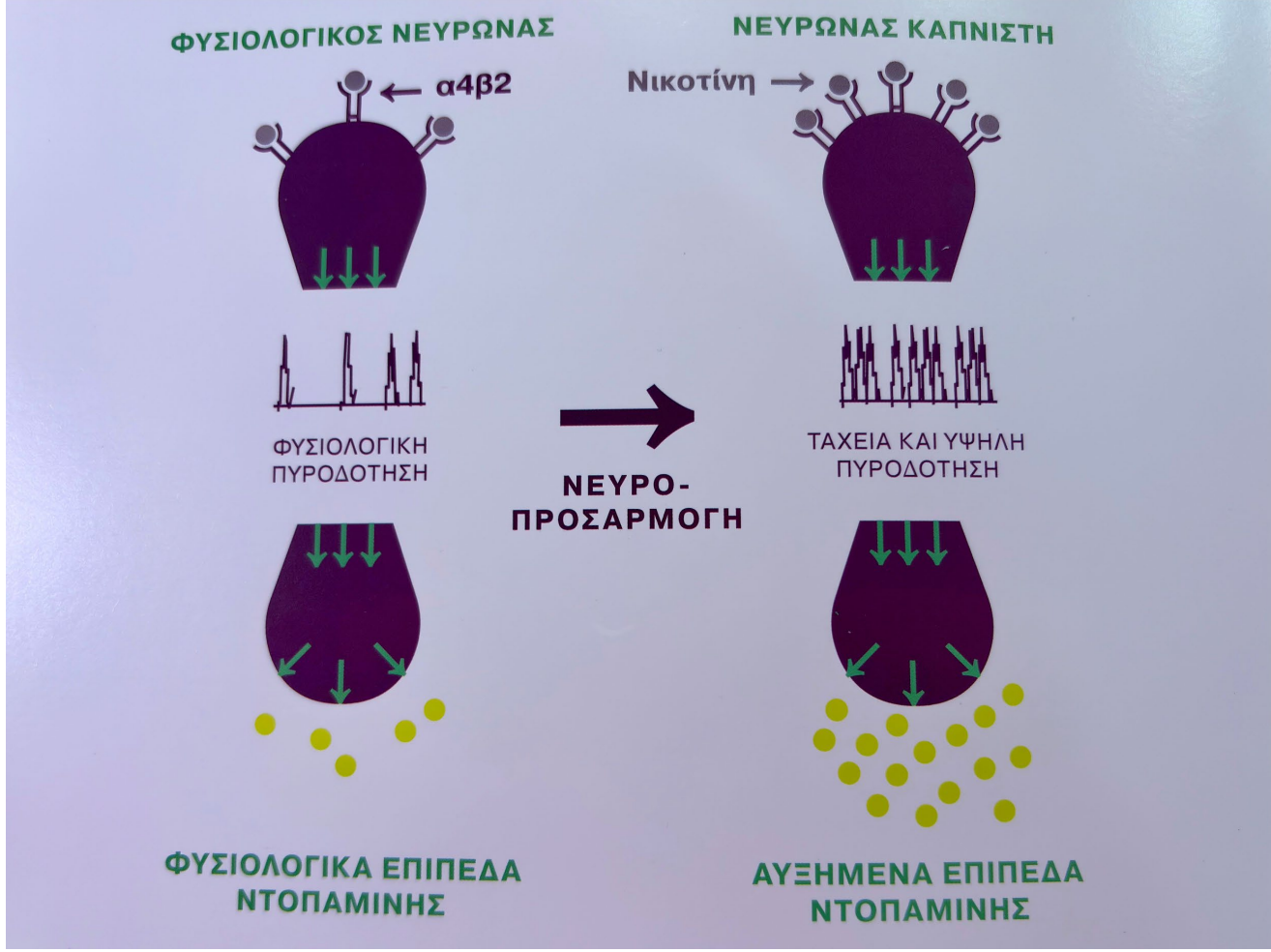
ΔΕΝ ΚΑΠΝΙΖΩ
Ζω ελεύθερα!

Κέρδος ζωής με όριο τα 80 χρόνια

Για τους μη καπνιστές:	11-12 χρόνια
Διακοπή στα 25-34:	10 χρόνια
Διακοπή στα 35-44:	9 χρόνια
Διακοπή στα 45-54:	6 χρόνια
Διακοπή στα 55-64:	4 χρόνια

Στερητικό σύνδρομο

Λαμβάνοντας υπόψη ότι το κάπνισμα, σύμφωνα με τον Παγκόσμιο Οργανισμό Υγείας, είναι μια χρόνια νόσος, η λήψη οργανωμένης βοήθειας για τη διακοπή του καπνίσματος είναι ιδιαίτερα σημαντική. Υπάρχουν διεθνείς οδηγίες για τη διακοπή του καπνίσματος, που πρέπει να εφαρμόζονται από ιατρούς και ψυχολόγους που έχουν αποκτήσει ειδική εκπαίδευση και γνώση. Είναι σημαντικό να γνωρίζετε πως η διακοπή του καπνίσματος που γίνεται με τη βοήθεια εξειδικευμένων επαγγελματιών υγείας έχει 40-60% αποτελεσματικότητα, ενώ ένας καπνιστής που προσπαθεί να απαλλαγεί μόνος του τα καταφέρνει μόνο σε ποσοστό 3-5%

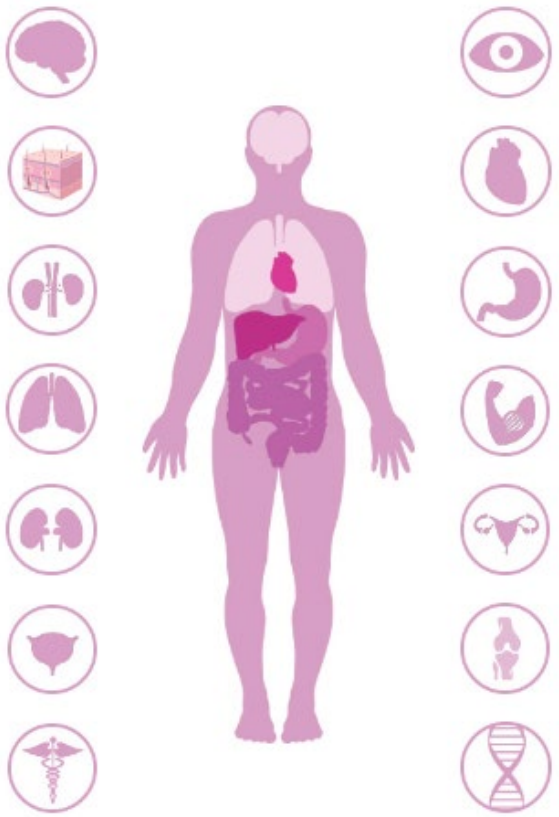


World Health Organization

Σύμφωνα με τον Παγκόσμιο Οργανισμό Υγείας, όλα τα προϊόντα καπνού είναι εξαρτησιογόνα και βρίσκονται στην ίδια ευρύτερη κατηγορία με ουσίες όπως η κοκαΐνη και τα οπιοειδή στο διεθνές σύστημα κατάταξης νοσημάτων ICD-10. Ο λόγος που το κάπνισμα είναι τόσο εθιστικό έχει σχέση με τη νικοτίνη που είναι ψυχοτρόπος ουσία και συνδέεται με ειδικούς υποδοχείς στον εγκέφαλο. Η νικοτίνη εισχωρεί στον εγκέφαλο σε 10 sec, έχει χρόνο ημίσειας ζωής περίπου 2 ώρες και χρησιμοποιώντας το σύστημα ανταμοιβής του εγκεφάλου (Brain reward system) μας κάνει να αισθανόμαστε έντονο ευχάριστο συναίσθημα αντικαθιστώντας ήπια ευχαρίστηση που λαμβάνουμε από άλλα ερεθίσματα όπως η τροφή. Το ευφορικό συναίσθημα είναι μεγαλύτερο από αυτό της κοκαΐνης, της μορφίνης και της αμφεταμίνης

ΔΕΝ ΚΑΠΝΙΖΩ
Ζω ελεύθερα!

Επιπτώσεις στην Υγεία



Νευρικό Σύστημα

- Άνοια
- Αγγειακά εγκεφαλικά επεισόδια
- Ανευρύσματα
- Πολλαπλή σκλήρυνση
- Νευροπαθητικό άλγος

Οφθαλμοί

- Καταρράκτης
- Γλαύκωμα
- Εκφύλιση ωχράς κηλίδας
- Οπτική νευροπάθεια
- Τύφλωση
- Οφθαλμοπάθεια Graves

Πεπτικό Σύστημα

- Καρκίνος πεπτικού σωλήνα
- Γαστροοισοφαγική παλινδρόμηση
- Οξεία και χρόνια παγκρεατίτιδα
- Νόσος Crohn

Ενδοκρινικό Σύστημα

Σακχαρώδης διαβήτης τύπου ΙΙ

Αυτοάνοση θυρεοειδοπάθεια

Αυξημένα επίπεδα ορμονών stress

ΕΛΛΗΝΙΚΗ ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ - ΟΜΑΔΑ ΔΙΑΚΟΠΗΣ ΚΑΠΝΙΣΜΑΤΟΣ

ΕΛΛΗΝΙΚΗ ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ
HELLENIC THORACIC SOCIETY

ΔΕΝ ΚΑΠΝΙΖΩ
Ζω ελεύθερα!

Αναπνευστικό Σύστημα

Χρόνια αποφρακτική πνευμονοπάθεια

Επιδείνωση άσθματος

Καρκίνος πνεύμονα

Κυκλοφορικό Σύστημα

Αρτηριοσκλήρυνση

Στεφανιαία νόσος

Αρτηριακή υπέρταση

Υπερλιπιδαιμία

Αυτοάνοσα Νοσήματα

Ρευματοειδής αρθρίτιδα

Συστηματικός και δερματικός λύκος

Πρωτοπαθή χολική κίρρωση

Θυρεοειδοπάθεια Graves'

Ουροποιητικό Σύστημα

Καρκίνος ουροδόχου κύστεως

Επιθετικός καρκίνος προστάτη

Νεφρά

Χρόνια νεφρική νόσος

Οστά

Οστεοπόρωση

Δέρμα

Ψωρίαση

Δερματικές μετεγχειρητικές επιπλοκές

Αισθητικές διαταραχές

Πρόωρη γήρανση επιδερμίδας

Φλυκταίνωση παλαμών-πελμάτων

Ca χειλέων

Αλλεργική δερματίτιδα

Μυϊκό Σύστημα

Μυϊκή δυσλειτουργία

Γεννητικό Σύστημα

Στυτική δυσλειτουργία

Υπογονιμότητα

Διαταραχές ορχικής και ωοθηκικής λειτουργίας

Διάφορα

Κοιλιακό και θωρακικό ανεύρυσμα

Λευχαιμία

Σύνδρομο άπνοιας στον ύπνο

Μεταβολικό σύνδρομο

Αποφρακτική αγγειίτιδα

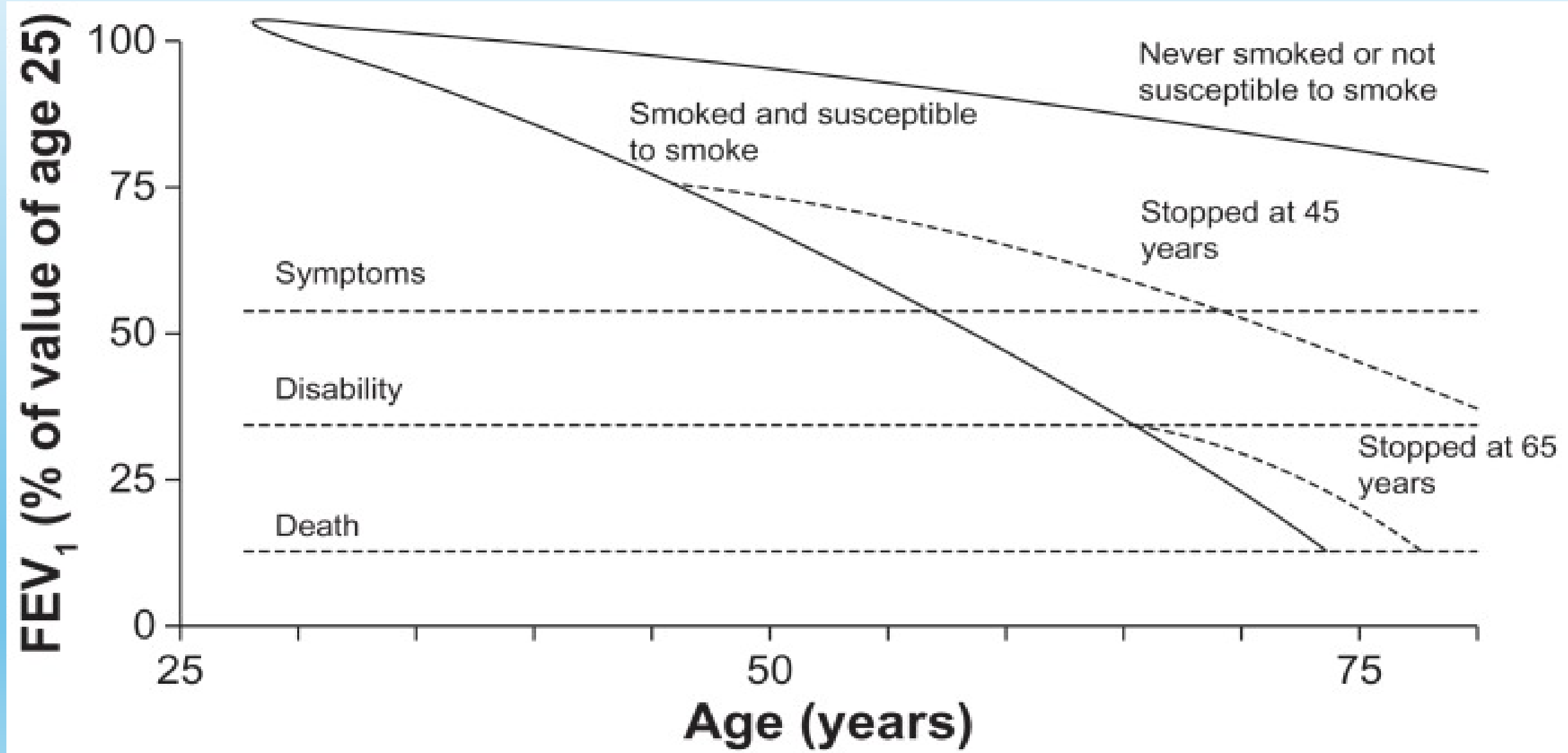
Διαταραχές ακοής

Περιοδοντίτιδα

ΕΛΛΗΝΙΚΗ ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ - ΟΜΑΔΑ ΔΙΑΚΟΠΗΣ ΚΑΠΝΙΣΜΑΤΟΣ

ΕΛΛΗΝΙΚΗ ΠΝΕΥΜΟΝΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ
HELLENIC THORACIC SOCIETY

Modified version of Fletcher and Peto's1 graph showing the decline in the forced expiratory volume in the first second (FEV1)



Eur Respir J 2004; 23: 464–476
DOI: 10.1183/09031936.04.00012704

European Respiratory Journal
ISSN 0903-1936

REVIEW

The impact of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness and inflammation

B.W.M. Willemse*,#, D.S. Postma#, W. Timens*, N.H.T. ten Hacken#

The impact of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness and inflammation. B.W.M. Willemse, D.S. Postma, W. Timens, N.H.T. ten Hacken. ©ERS Journals Ltd 2004.

ABSTRACT: Smoking is the main risk factor in the development of chronic obstructive pulmonary disease (COPD), and smoking cessation is the only effective treatment for avoiding or reducing the progression of this disease.

Despite the fact that smoking cessation is a very important health issue, information about the underlying mechanisms of the effects of smoking cessation on the lungs is surprisingly scarce. It is likely that the reversibility of smoke-induced changes differs between smokers without chronic symptoms, smokers with nonobstructive chronic bronchitis and smokers with COPD. This review describes how these three groups differ regarding the effects of smoking cessation on respiratory symptoms, lung function (forced expiratory volume in one second), airway hyperresponsiveness, and pathological and inflammatory changes in the lung.

Smoking cessation clearly improves respiratory symptoms and bronchial hyperresponsiveness, and prevents excessive decline in lung function in all three groups.

Data from well-designed studies are lacking regarding the effects on inflammation and remodelling, and the few available studies show contradictory results. In chronic obstructive pulmonary disease, a few histopathological studies suggest that airway inflammation persists in exsmokers. Nevertheless, many studies have shown that smoking cessation improves the accelerated decline in forced expiratory volume in one second, which strongly indicates that important inflammatory and/or remodelling processes are positively affected.

Eur Respir J 2004; 23: 464–476.

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Keywords: Chronic obstructive pulmonary disease
inflammation
review
smoking cessation

Received: February 5 2003
Accepted after revision: October 29 2003

Table 2.—Longitudinal data on lung function decline in smokers, exsmokers, quitters and nonsmokers without chronic respiratory symptoms

First author [ref.]	Subjects n	Sex	Age[§] yrs	Cumulative cigarette consumption pack-yrs			Follow-up yrs	Decline in FEV1 mL·yr^{-1}			
				Sm	Exsm	Quitter		Sm	Exsm	Quitter	Nonsm
BURCHFIEL [45]	4451[¶¶]	M	54				6	34*	22	23[#], 30[ff]	22
SHERRILL [44]	477[¶¶]	M	46 (45–47)	49	41		7	17	27		16
		F	46 (41–52)	43	19		7	14	15		12
BOSSE [46]	850[¶¶]	M	42 (39–44)	21		22	5	85		57[#]	53
CAMILLI [47]	1705[¶¶]	M	49	32	32	32	9.4	19[+]	4	6	6
		F	49	28	17	18	9.4	7[+]	-0.7	-4	0.4
TASHKIN [25]	2401[¶¶]	M	45	41	27	39	5	70[+]	52	62	56
		F	45	31	16	27	5	54[+]	38	38	42
SHERMAN [16]	3948[¶¶]	M	48	32	24		12	44[+]	35		33
		F	49	22	12		12	34[+]	27		28
XU [49]	4554[¶¶]	M	15–54				24	19*, 26*, 33*,[++]	20	6[#]	6
		F	15–54				24	15*, 20*, 30*,[++]	19	3[#]	15
XU [39]	5572[¶¶]	M	25–78				6	53[+]	34	41[#]	38
		F	25–78				6	38[+]	30	29[#]	29
LANGE [8]	7764[¶¶]	M	<55				5	22, 42[§§]	27	17, 36[§§]	21
			>55				5	52, 56[§§]	36	11, 43[§§]	34
		F	<55				5	17, 30[§§]	18	15, 9[§§]	13
			>55				5	39, 48[§§]	32	28, [§§]	32
TOWNSEND [55]	4926	M	47				6–7	59[+]	44	50	51
PELKONEN [51][f]	411[¶¶]	M	47				30	52[+]	36	40	35
	1007[¶¶]	M	49	27	18	25	15	66[+]	49	56	46
KRZYZANOWSKI [52]	1824[¶¶]	M	40 (19–70)				13	60*,[¶]	50	68	47
		F	40 (19–70)				13	42	38	37	38
KRZYZANOWSKI [56][##]	640[¶¶]	M	45				12	11.7		6.8	6.3
		F	48				12	10.5		1.6	7.6
	1738[¶¶]	M	40				12	14		16.5	8.7
		F	40				12	6.6		1.4	6.1
TAYLOR [53]	227[¶¶]	M	51–61				7.5	11[+]	8		6.6

FEV1: forced expiratory volume in one second; Sm: smokers without chronic respiratory symptoms; Exsm: exsmokers who quitted smoking before the start of the study; Quitter: healthy smoker at start of study, exsmoker at end of study; Nonsm: nonsmoker; M: male; F: female. [§]: mean, range or mean (range); [f]: part of population followed for 30 yrs (n=411), duration of smoking rather than cumulative cigarette consumption described and FEV0.75 rather than FEV1 measured; [##]: two populations studied: Tucson, AZ, USA (n=640), and Cracow, Poland (n=1,738); [¶¶]: general population study; [++]: light (<15 cigarettes·day^{-1}), moderate (15–24 cigarettes·day^{-1}), heavy (>24 cigarettes·day^{-1}) smokers; [§§]: light (<15 cigarettes·day^{-1}), heavy (>15 cigarettes·day^{-1}) smokers; [ff]: >2 ys, <2 yrs. *: $p<0.05$ *versus* nonsmokers; [#]: $p<0.05$ *versus* smokers; [¶]: $p<0.05$ *versus* exsmokers; [+]: $p<0.05$ *versus* all other groups.

Table 3. – Effects of smoking cessation on decline in lung function in smokers with chronic bronchitis (CB) or chronic obstructive pulmonary disease (COPD)

First author [ref.]	Subjects n	Sex	Age yrs	CB/COPD	Cumulative cigarette consumption pack-yrs			Follow-up yrs	Decline in FEV1 mL·yr^{-1}		
					Sm	Exsm	Quitter		Sm	Exsm	Quitter
COMSTOCK [21]	670	M	40–59	CB[#]				5	82		34
FLETCHER [63]	792[¶]	M	50–59	Mild				8	62, 80[+]	37	
				None				8	42, 55[+]	30	
ANTHONISEN [3, 64]	5887	M/F	48	Mild–moderate	40			5	63		34[§]
SCANLON [65]	3818	M/F	49	Mild–moderate	41		40	5	62		31
MURRAY [66]	5887	M/F	48	Mild	42, 36[f]		42, 36[f]	5	1.2% pred		0.33% pred
POSTMA [67]	81[##]	M/F	48	Moderate–severe	40			2–21	85		49[¶¶]
POSTMA [68]	81[++]	M	48	Moderate	40			2.8–20	85	49	
BARTER [69]	34[§§]	M	56	Mild				5	56	16	
HUGHES [70]	56[ff]	M	54, 57	Mild–moderate	37	35		3–13	54	16	
ANTHONISEN [71]	4517	M	61	Mild–moderate				11	66		30
		F							54		22
LEADER [72]	25[###]	M/F	50	Mild–moderate	61		71	0, 8 and 28 weeks			0

FEV1: forced expiratory volume in one second; Sm: smokers; Exsm: exsmokers who quitted smoking before the start of the study; Quitter: someone who quits smoking at start of study and is still not smoking at end of study; M: male; F: female; % pred: per cent predicted. [#]: 90% of the smokers had symptoms of CB; [¶]: general population study; [+]: light (<15 cigarettes·day^{-1}), heavy (>15 cigarettes·day^{-1}) smokers; [§]: +57 mL·yr^{-1}after 1 yr of smoking cessation; [f]: M, F; [##]: 59 smokers, 22 quitters; [¶¶]: quitters defined as those who smoked at the start of the study but quitted smoking at some point during the study and did not start smoking again; [++]: FEV1 63% pred without steroids; [§§]: five exsmokers; [ff]: 37 smokers, 19 exsmokers; [###]: 18 smokers, seven quitters.

Table 4. – Cross-sectional data on airway hyperresponsiveness (AHR) in smokers, exsmokers and nonsmokers without chronic respiratory symptoms

First author [ref.]	Subjects n	Sex	Age yrs	Method	AHR response[#] %	Results	Comments
KABIRAJ [54]	18 Nsm	M	48	FEV1 fall after 10 mg·mL^{-1} MCh	9.8	Sm>Exsm=Nsm	Atopy unk; symptoms in both Sm and Exsm, but not in all
	20 Exsm	M	48		9.5		
	22 Sm	M	48		19.5		
CERVERI [74][¶]	295 Nsm	M/F	39 (15–64)	PD15 <7.9 mg MCh	11	Sm>Exsm=Nsm	All asymptomatic and NA
	50 Exsm	M/F			15		
	70 Sm	M/F			43[+]		
PAOLETTI [75][¶]	693 Nsm	M/F	8–73	PD20 <4.8 mg MCh	34, 23[§]	Sm>Exsm=Nsm (F)	NI
	369 Exsm	M/F			31, 21[§]	Sm=Exsm=Nsm (M)	
	496 Sm	M/F			40, 25[§]		
SUNYER [76][¶]	387 Nsm	M/F	32 (20–44)	PC20 <100 mg·mL^{-1} MCh	13, 5, 25, 21[##]	Sm>Exsm=Nsm (NA)	
	163 Exsm	M/F			14, 8, 14, 31[##]	Sm=Exsm=Nsm (A)	
	619 Sm[f]	M/F			24, 18, 20, 18[##]		
SPARROW [77][¶]	129 Nsm	M	50–59	PD20 <8.6 µmol MCh	9.3	Sm>Exsm=Nsm	Age range selected (n=914 in study)
	172 Exsm				8.7		
	66 Sm				22.7		
BURNEY [78][¶]	259 Nsm	M/F	41	PD20 <8 µmol HA	10	Sm>Exsm=Nsm	
	116 Exsm	M/F			12		
	136 Sm	M/F			24		
TAYLOR [53][¶]	39 Nsm	M	51–61	PC20 <16 mg·mL^{-1} HA	5	Sm=Exsm>Nsm	NI
	71 Exsm	M			24	Sm=Exsm[¶¶]	
	117 Sm	M			29		
LIM [73]	16 Exsm	M	53	PC20 HA mg·mL^{-1}	6.7	Sm=Exsm	NI
	27 Sm	M	59		7.1		
XU [79][¶]	Nsm	M/F	>8	PC10 <8 mg·mL^{-1} HA	16	Sm=Nsm	NI
	Exsm				18		
	Sm (2684)				20, 33[++]		
RIJCKEN [15][¶]	574 Nsm	M/F	32.7	PC10 <16 mg·mL^{-1} HA	24	Sm=Exsm=Nsm	NI
	252 Exsm	M/F			18		
	1013 Sm	M/F			28		

Nsm: nonsmoker; Sm: smokers without chronic respiratory symptoms; Exsm: exsmoker; M: male; F: female; FEV1: forced expiratory volume in one second; MCh: methacholine; unk: unknown; PD15: provocative dose of drug causing a 15% fall in FEV1; NA: nonatopic; PD20: provocative dose of drug causing a 20% fall in FEV1; NI: no information about atopy or symptoms; PC20: provocative concentration of drug causing a 20% fall in FEV1; A: atopic; HA: histamine; PC10: provocative concentration of drug causing a 10% fall in FEV1. [#]: severity or prevalence (see *Method* column); [¶]: general population study; [+]: 27% heavy plus 16% moderate (<18 pack-yrs) smokers; [§]: F, M; [f]: cumulative cigarette consumption 15 pack-yrs; [##]: NA-F, NA-M, A-F, A-M; [¶¶]: subjects aged <35 yrs (n=30); [++]: <24 cigarettes·day^{-1}, >25 cigarettes·day^{-1}.

Table 5.–Effect of smoking cessation (SC) on airway hyperresponsiveness (AHR) in smokers without chronic respiratory symptoms

First author [ref.]	Subjects n	Sex	Age yrs	Cumulative cigarette consumption pack-yrs	Sympt	SC period	Method	AHR#		
								Before SC %	After SC %	SC effect
BUCZKO [23]	17	M/F	35	20	14/17	99±43 days¶	MCh TC	1.0 mg·mL^{-1}	1.6 mg·mL^{-1}	None
SIMONSSON [61]	10	M/F	42	25	Some	1 week; 1, 6, 12 months	>15% FEV1 fall (MCh+)	30	20§	None
ISRAEL [27]	10	M/F	36	20	Some	2, 6 months	PD35	20	40, 50f	None
							PD20	60	60, 70f	

Sympt: symptoms (occasional cough or sputum production); M: male; F: female; MCh: methacholine; TC: threshold concentration (baseline volume 40% vital capacity above residual volume minus 2.8 SD); FEV1: forced expiratory volume in one second; PD35: provocative dose of carbachol (various doses used) causing a 35% fall in specific airway conductance; PD20: provocative dose of carbachol (various doses used) causing a 20% fall in FEV1. #: severity or prevalence (see *Method* column); ¶: mean±SD; +: various concentrations used; §: at 12 months; f: at 2 months, 6 months.

Table 6. – Cross-sectional data on airway hyperresponsiveness (AHR) in smokers and exsmokers with chronic bronchitis (CB) or chronic obstructive pulmonary disease (COPD)

First author [ref.]	Subjects[#] n	Age yrs	Cumulative cigarette consumption pack-yrs	CB/COPD	Method	AHR response	Results
TASHKIN [80]	5877 Sm	49	41	Mild COPD	PC_{20} <25 mg·mL^{-1} MCh %	59, 85[¶]	
OOSTERHOFF [81]	12 Sm	57	20	Moderate COPD	PC_{20} MCh/AMP mg·mL^{-1}	>16, >320[+]	Sm<Sm COPD=Exsm COPD (MCh)
	19 Sm COPD	60	13			0.35, 7.2[+]	Sm<Exsm COPD<Sm COPD (AMP)
	11 Exsm COPD	63				0.5, 58[+]	
POSTMA [82]	5 Sm	53	24	Moderate/severe COPD		No AHR	Sm<Sm COPD
	5 Exsm		15			No AHR	Exsm<Sm COPD
	14 Sm COPD	57	35		PC_{20} HA mg·mL^{-1}	6.73	Sm COPD=Exsm COPD
	14 Exsm COPD	55	26			5.58	
BAHOUS [83]	24 Sm COPD/CB 4 Exsm COPD/CB	50	37	CB (n=14), mild COPD (n=4), moderate COPD (n=10)	PC_{20} <8 mg·mL^{-1} HA/MCh %[§]	CB 7, COPD 100	Sm CB<Sm COPD
WOOLCOCK [84]	10 Sm COPD	56	53	Mild COPD (n=2), moderate	PD_{20} MCh µmol	1.0–10	Nsm<Sm COPD
	2 Nsm			COPD (n=8)	PD_{20} HA µmol	0.5–5.9	

Sm: smoker; Exsm: exsmoker; Nsm: nonsmoker; PC_{20}: provocative concentration of drug causing a 20% fall in forced expiratory volume in one second (FEV_1); MCh: methacholine; AMP: adenosine 5′-monophosphate; HA: histamine; PD_{20}: provocative dose of drug causing a 20% fall in FEV_1. [#]: male and female subjects in all studies; [¶]: males, females; [+]: MCh, AMP; [§]: response identical for HA and MCh.

Table 7.—Effect of smoking cessation (SC) on inflammation in blood, sputum and bronchoalveolar lavage fluid (BALF) in smokers without chronic respiratory symptoms

First author [ref.]	Subjects n	Age yrs	Cumulative cigarette consumption pack-yrs	Specimen	Validation of quitters	Follow-up months	Effects of SC
ROBBINS [105]	10 Q	38	30¶	eNO	Exhaled CO	1, 8 weeks	Increase in eNO (5.7 to 10.3 ppb) at 1/8 weeks
MILLER [91]	20 Q	22–71		Blood		6 weeks	No change in % lym, CD3, CD4 or CD8 or CD4/CD8 ratio in lSm/mSm
	5 lSm		10–19				Decr in % CD8, CD4/CD8 ratio norm in hSm
	6 mSm 9 hSm		19–49 50–120				
SKOLD [106]#	18 Q	41	23	Blood		1, 3, 6, 9, 15	Decr in leuc and [Hb] at 9 months Inc in IgG (trend)
JENSEN [107]	160 Q 92 Q	43	25	Blood	Exhaled CO	6, 12	Decr in leuc, neut, lym; No change in baso
JENSEN [108]	92 Q	44	23	Blood	Exhaled CO	6, 12	Inc in sIgE at 6 months, esp. in Q <40 yrs; Decr in sIgE at 12 months
MELISKA [109]	28 Q (M)	21–35	21¶	Blood (lym)	Cotinine	31 days	Inc in NK cytotoxic activity; No change in T-cell activation; Decr in serum cortisol
SCOTT [110]	30 Q	43	25¶	Plasma	Exhaled CO, cotinine	12	Decr in sICAMs (307 to 241 $ng \cdot mL^{-1}$) towards normal
SCOTT [111]	30 Q	43	25¶	Plasma	Exhaled CO, cotinine	12	No change in sCD44; Decr in sCD44v5 and sCD44v6
JENSEN [112]	50 Q	39	30	Blood	Exhaled CO	3, 6, 12	Decr in sECP at 6/12 months (-13.1 $\mu g \cdot L^{-1}$); Decr in sLF at 6/12 months (-230.7 $\mu g \cdot L^{-1}$)
HERSEY [113]	35 Q	38		Blood		3	Decr in lym; Trend Decr in neut, plat Inc in NK activity; Inc in IgG, IgM
SWAN [114]	46 Q	49	49	Sputum (spont)	Cotinine in sputum	12	Decr in neut (-7.9%), MP (-14%), pigm MP (-4%); No change in columnar cells, mucus, metaplasia, dysplasia
SKOLD [115]#	18 Q	41	23	BALF		1, 3, 6, 9, 15	Decr in [cells] at 1 month; Decr in oedema, erythema, mucus+, norm at 6 months; Inc in MP Fl at 1 month; Decr in MP Fl at 6 months
SKOLD [116]#	18 Q	41	23	BALF		1, 3, 6	Decr in [cells] at 1 month; Decr in MP (91 to 83%) at 6 months; Inc in MP activity at 6 months; Inc in lym (6.6 to 14.5%) at 6 months; No change in % neut, eos, baso at 6 months
SKOLD [106]#	18 Q	41	23	BALF		1, 3, 6, 9, 15	Decr in neut, MP, lym, eos (total cells) at 9 months; No changes in L-fibronectin, L-hyaluronan Inc after SC, norm at 12 months; L-albumin Inc at 6 months SC, norm at 12 months
ANDERSSON [117]#	8 Q	37	20	BALF		1, 3, 6, 9, 15	CCSP levels lower in Sm, Inc till 9 months, norm at 15 months

Q: quitters; lSm: light smokers (Sm); mSm: moderate smokers; hSm: heavy smokers; eNO: exhaled nitric oxide; Inc: increase; Decr: decrease; ppb: parts per billion; lym: lymphocytes; norm: normalised; leuc: leukocytes; Hb: haemoglobin; Ig: immunoglobulin; neut: neutrophils; baso: basophils; sIg: serum Ig; esp.: especially; NK: natural killer [cell]; sICAM: soluble intercellular adhesion molecule; sCD44: soluble CD44; sECP: serum eosinophil cationic protein; sLF: serum lactoferrin; Plat: platelets; spont: spontaneous; MP: macrophages; pigm: pigmented; Fl: fluorescence; eos: eosinophils; CCSP: Clara cell secretory protein. #: same population (subpopulation used in [117]); ¶: cigarettes·day^{-1} (pack-yrs not given); +: macroscopic endobronchial findings.

Journal of Comparative Effectiveness Research

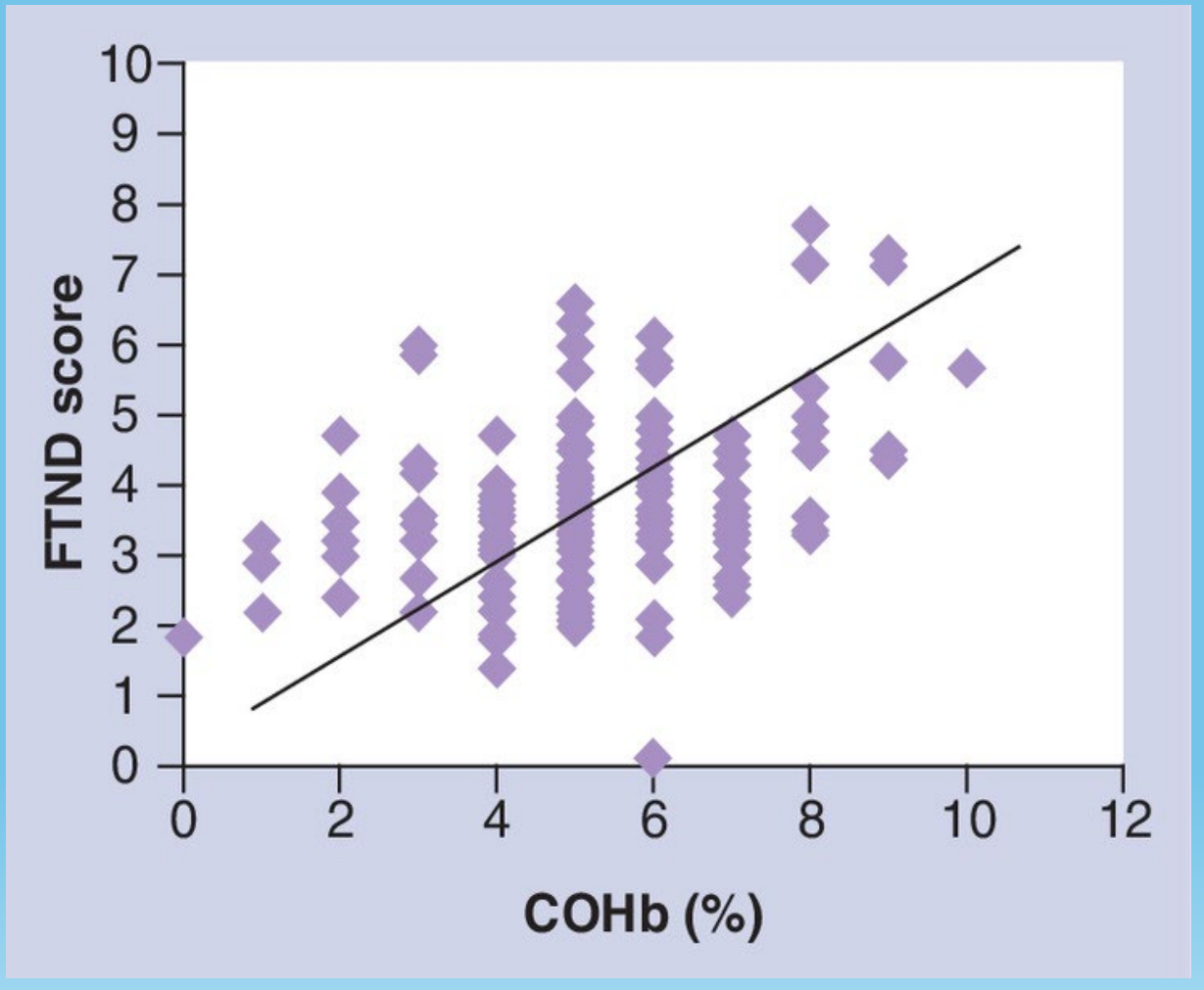
Short-term effectiveness of smoking-cessation treatment on respiratory function and CEA level

Authors: Aldo Pezzuto, Chiara Spoto, Bruno Vincenzi, and Giuseppe Tonini g.tonini@unicampus.it

Changes in clinical and laboratory outcomes at the end of treatment, a comparison between quitters and reducers

Clinical and laboratory parameters	**Reducers**			**Quitters**		**p-value**
	Basal, mean (SD)	***3 months, mean (SD)***		***Basal, mean (SD)***	***3 months, mean (SD)***	
FTND score (0–10)	5.0 (2.1)	4.4 (1.2)		5.0 (2.2)	3.1 (1.2)	<0.001
CEA (ng/ml)	5.8 (3.5)	4.3 (3.9)		5.8 (2.1)	4.0 (1.2)	<0.001
Dyspnea score (1–5)	1.7 (0.8)	0.9 (0.6)		1.7 (0.8)	0.6 (0.4)	<0.0001
COHb (%)	3.6 (1.4)	2.7 (0.9)		3.6 (1.5)	2.1 (0.9)	<0.0001

Correlation between carboxyhemoglobin and the Fagerström Test for Nicotine Dependence score



Japanese Journal of Radiology (2018) 36:165–180
https://doi.org/10.1007/s11604-017-0713-0

INVITED REVIEW

Smoking-related lung abnormalities on computed tomography images: comparison with pathological findings

Tae Iwasawa[1] · Tamiko Takemura[2] · Takashi Ogura[3]

Received: 30 June 2017 / Accepted: 7 December 2017 / Published online: 15 December 2017

Abstract
Smoking-related lung abnormalities are now an increasing public health concern. According to the findings of large-cohort studies, approximately 8% of smokers have interstitial lung abnormalities, which are associated with a relatively high risk of all-cause mortality. We reviewed the radiological and pathological findings of smoking-related interstitial lung diseases, such as respiratory bronchiolitis-interstitial lung disease, desquamative interstitial pneumonia, and airspace enlargement with fibrosis. We have also discussed the histological basis of unclassifiable interstitial pneumonia in smokers, which exhibits airway-centered cystic lesions with fibrosis. A variety of radiological findings coexist in the lungs of a smoker. This overlapping of multiple pathological conditions might cause the radiological patterns of diseases to become unclassifiable. Therefore, diagnosis should be performed not on the basis of a single radiological finding, but in a comprehensive manner, by including clinical symptoms and disease behavior. Among interstitial abnormalities in smokers, the usual interstitial pneumonia (UIP) pattern is correlated with a worse prognosis than others. Basal-predominant subpleural reticulation is a clue for accurate diagnosis of UIP, which can be achieved by computer-aided quantitative analysis.

Table 1 Smoking-related interstitial lung abnormalities

Smoking-related interstitial lung abnormalities	Abbreviations	Histological findings
Langerhans cell histiocytosis	LCH	Infiltration of Langerhans cells and cyst formation
Respiratory bronchiolitis-interstitial lung disease	RB-ILD*	Respiratory bronchiolitis
Desquamative interstitial pneumonia	DIP*	Macrophage accumulation within alveoli
Airspace enlargement with fibrosis	AEF	Pathological findings of emphysema with collagenous-type fibrosis, without obvious fibroblastic foci

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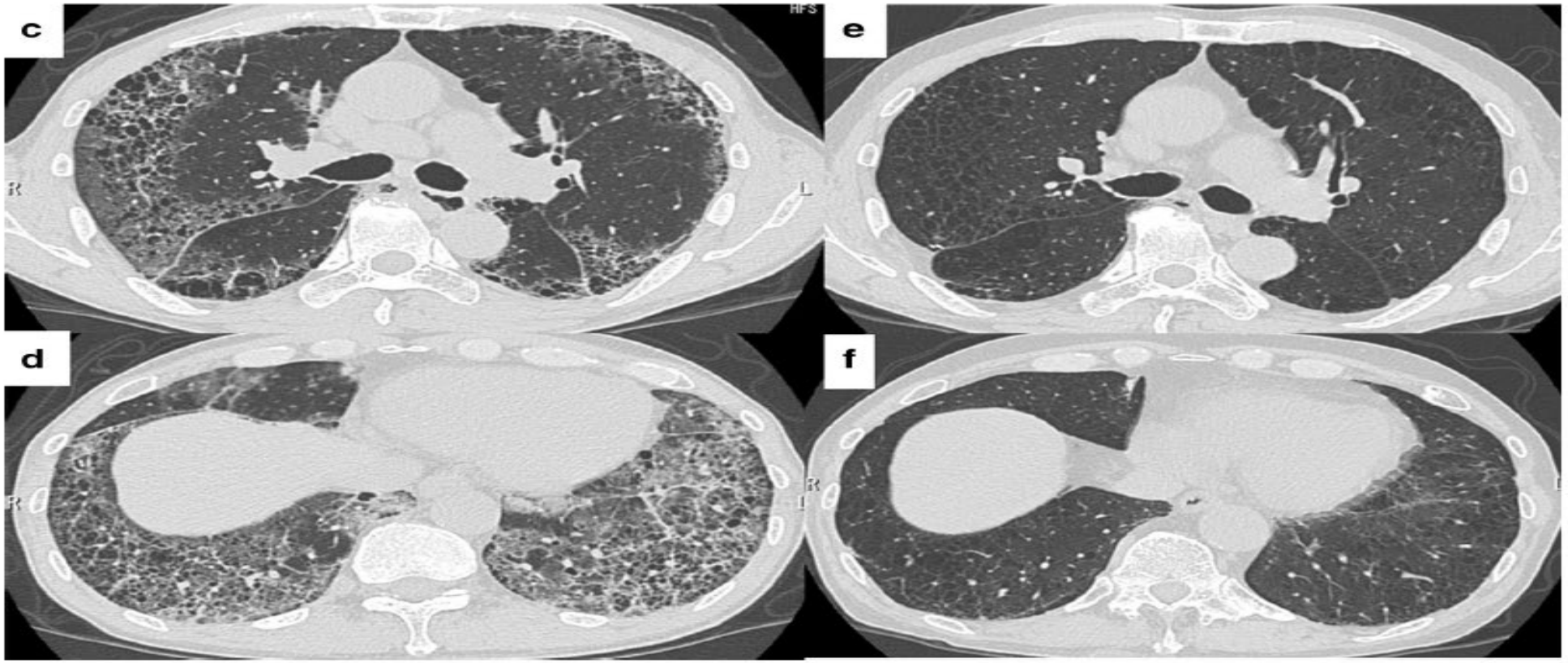

Fig. 4 DIP in a 52-year-old male smoker. Histological specimens (H & E staining) of the upper lobe (**a**, **b**) (original magnification, × 5 and × 20, respectively). **c**, **d** Initial and **e**, **f** 11-year follow-up HRCT findings. Right upper-lobe biopsy findings reveal diffuse, sheet-like, intra-alveolar macrophage accumulation (**a**, **b**). Pigmented macrophages (occasionally multinucleated) and eosinophil infiltration are noted. Mild interstitial fibrosis is also present (**b**). Initial HRCT images show widespread GGO in the middle and lower zones, with peripheral or patchy distribution. Cystic spaces are also noted (**c**, **d**). In follow-up CT images, the GGOs have disappeared after steroid therapy and cessation of smoking. However, emphysema-like cysts remain (**e**, **f**)

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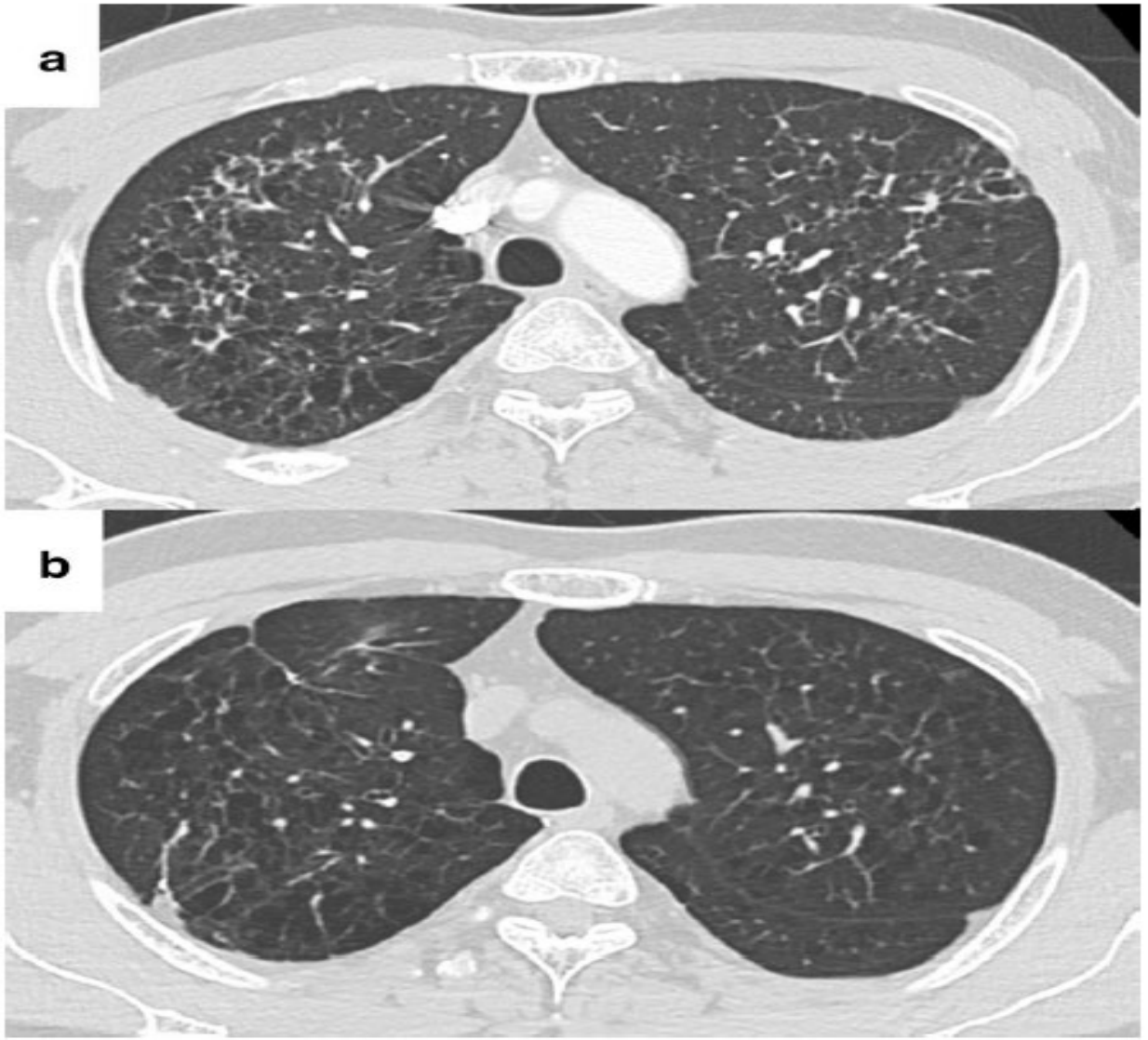
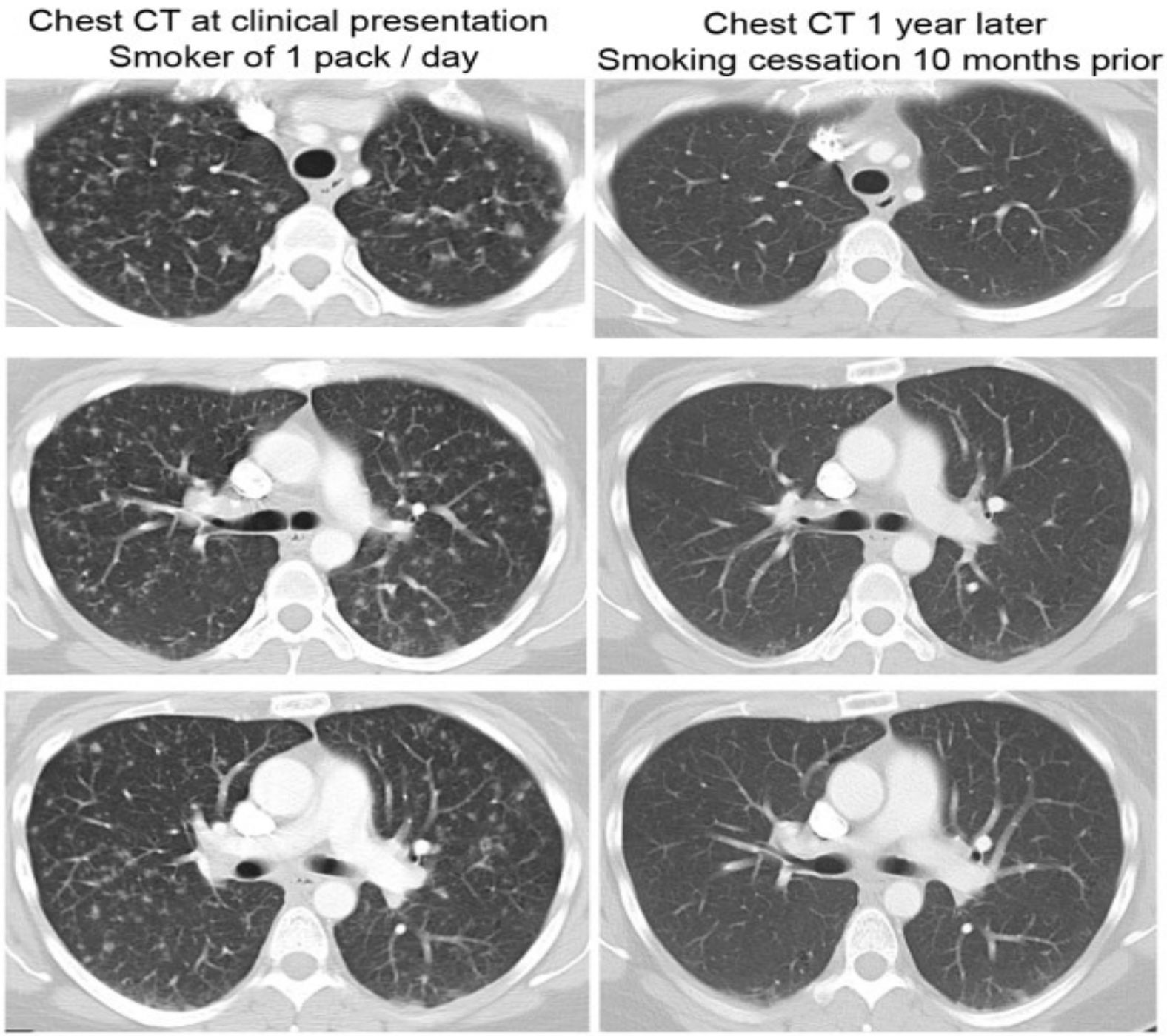


Fig. 2 Follow-up CT findings of a 33-year-old male smoker with LCH. HRCT findings at initial presentation (**a**), and (**b**) 10-year follow-up. Nodules and cysts are found mainly in the upper lobe (**a**). After cessation of smoking, the nodules have disappeared, and the air space has enlarged (**b**)

Suri et al. Orphanet Journal of Rare Diseases 2012, http://www.ojrd.com/content/7/1/16

Pulmonary langerhans cell histiocytosis
Harpreet S Suri1, Eunhee S Yi2, Gregorz S Nowakowski3 and Robert Vassallo1,4*

Radiographic improvement following smoking cessation. The chest CT images on the left side were performed in an active one pack/day 37 year old smoker with biopsy-proven PLCH. The representative chest CT images demonstrate diffuse nodular infiltrates in both upper and lower lung fields. The patient quit smoking 2 months after the first chest CT was performed. The representative chest CT images on the right side were performed one year after the first chest CT was obtained, and show considerable improvement in the nodular infiltrates following smoking cessation. The patient did not receive corticosteroid or other immunosuppressive therapy



THE LANCET
Public Health

COMMENT Volume 8, Issue 9 E664-E665 September 2023 *Open access*

Smoking cessation and lung cancer: never too late to quit

Rachael L Murray[a] rachael.murray@nottingham.ac.uk · Emma O'Dowd[b]

Although smoking rates in high-income countries have decreased since 2000, smoking remains a key modifiable risk factor for premature mortality and is the number-one risk factor for lung cancer. Continued smoking is associated with a substantially increased risk of all-cause mortality and tumour recurrence in patients with a diagnosis of lung cancer;[1] previous studies have shown improved recurrence-free and overall survival in former smokers with lung cancer compared with current smokers. A2022 meta-analysis by Caini and colleagues[2] showed that quitting smoking at or around the time of lung-cancer diagnosis (ie, within 12 months) was associated with improved overall survival.

The analysis by Aline F Faers and colleagues[3] in this issue of *The Lancet Public Health* is the first to look at the effects of duration of smoking cessation before a diagnosis of lung cancer on overall and non-small cell lung cancer (NSCLC)-specific survival across four continents (ie, Asia, Europe, North America, and South America). It highlights the benefits of stopping smoking, even in the year before diagnosis, and shows increasing benefit with longer durations of abstinence (particularly for people who quit more than 5 years before diagnosis). Their findings add to the evidence that quitting smoking is beneficial at any time. However, because the greatest gains in NSCLC-specific survival come with a longer duration of quitting, all health-care professionals should be reinforcing the benefits of smoking cessation in all patient interactions and effective, evidence-based, stop-smoking interventions should be available for all people who smoke and are willing to stop. Early intervention will maximise the duration from the timepoint that an individual quits smoking to the timepoint of a potential lung-cancer diagnosis and therefore maximise the benefits.

Original Investigation |Oncology Cancer Risk. Following Smoking Cessation in Korea

Eunjung Park, PhD[1,2]; Hee-Yeon Kang, PhD[1,2]; Min Kyung Lim, PhD[3]; et alByungmi Kim, PhD[2]; Jin-Kyoung Oh, PhD[1,2]

February 6, 2024, JAMA Network Open

Abstract

Importance Tobacco smoking is associated with increased risk of various cancers, and smoking cessation has been associated with reduced cancer risks, but it is still unclear how many years of smoking cessation are required to significantly reduce the cancer risk. Therefore, investigating the association of smoking cessation with cancer is essential.

Objective To investigate the time course of cancer risk according to the time elapsed since smoking cessation and the benefits of smoking cessation according to the age at quitting.

Design, Setting, and Participants This population-based, retrospective cohort study included Korean participants aged 30 years and older who underwent 2 or more consecutive health examinations under the National Health Insurance Service since 2002 and were followed-up until 2019. Data analysis was performed from April to September 2023.

Main Outcomes and Measures The primary cancer was ascertained using the cancer registry data: all-site cancer (*International Statistical Classification of Diseases and Related Health Problems, Tenth Revision [ICD-10]* codes C00-43, C45-96, or D45-D47), lung cancer (*ICD-10* code C34), liver cancer (*ICD-10* code C22), stomach cancer (*ICD-10* code C16), and colorectal cancer (*ICD-10* codes C18-20). Hazard ratios (HRs) and 95% CIs were estimated using a Cox proportional hazards regression model with follow-up years as the timescale.

Results Of the 2 974 820 participants, 1 727 340 (58.1%) were men (mean [SD] age, 43.1 [10.0] years), and 1 247 480 (41.9%) were women (mean [SD] age, 48.5 [9.9] years). Over a mean (SD) follow-up of 13.4 (0.1) years, 196 829 cancer cases were confirmed. Compared with continuous smokers, complete quitters had a lower risk of cancer, with HRs of 0.83 (95% CI, 0.80-0.86) for all cancer sites, 0.58 (95% CI, 0.53-0.62) for lung, 0.73 (95% CI, 0.64-0.82) for liver, 0.86 (95% CI, 0.79-0.93) for stomach, and 0.80 (95% CI, 0.72-0.89) for colorectum. The cancer risk exhibited a slightly higher value for 10 years after quitting compared with continued smoking and then it decreased over time, reaching 50% of the risk associated with continued smoking after 15 or more years. Lung cancer risk decreased 3 years earlier than that of other cancer types, with a larger relative reduction. Regardless of quitting age, a significant reduction in the cancer risk was observed. Quitting before the age of 50 years was associated with a greater reduction in lung cancer risk (HR, 0.43; 95% CI, 0.35-0.53) compared with quitting at age 50 years or later (HR, 0.61; 95% CI, 0.56-0.66).

Conclusions and Relevance In this population-based retrospective cohort study, sustained smoking cessation was associated with significantly reduced risk of cancer after 10 years since quitting. Quitting at any age helped reduce the cancer risk, and especially for lung cancer, early cessation before middle age exhibited a substantial risk reduction.

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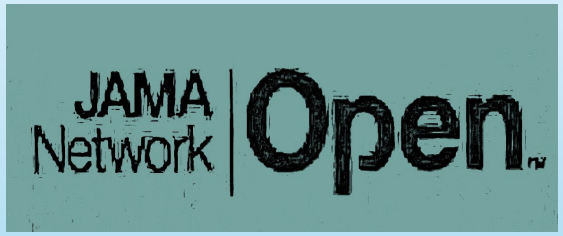


Risk of lung cancer significantly decreases after smoking is stopped

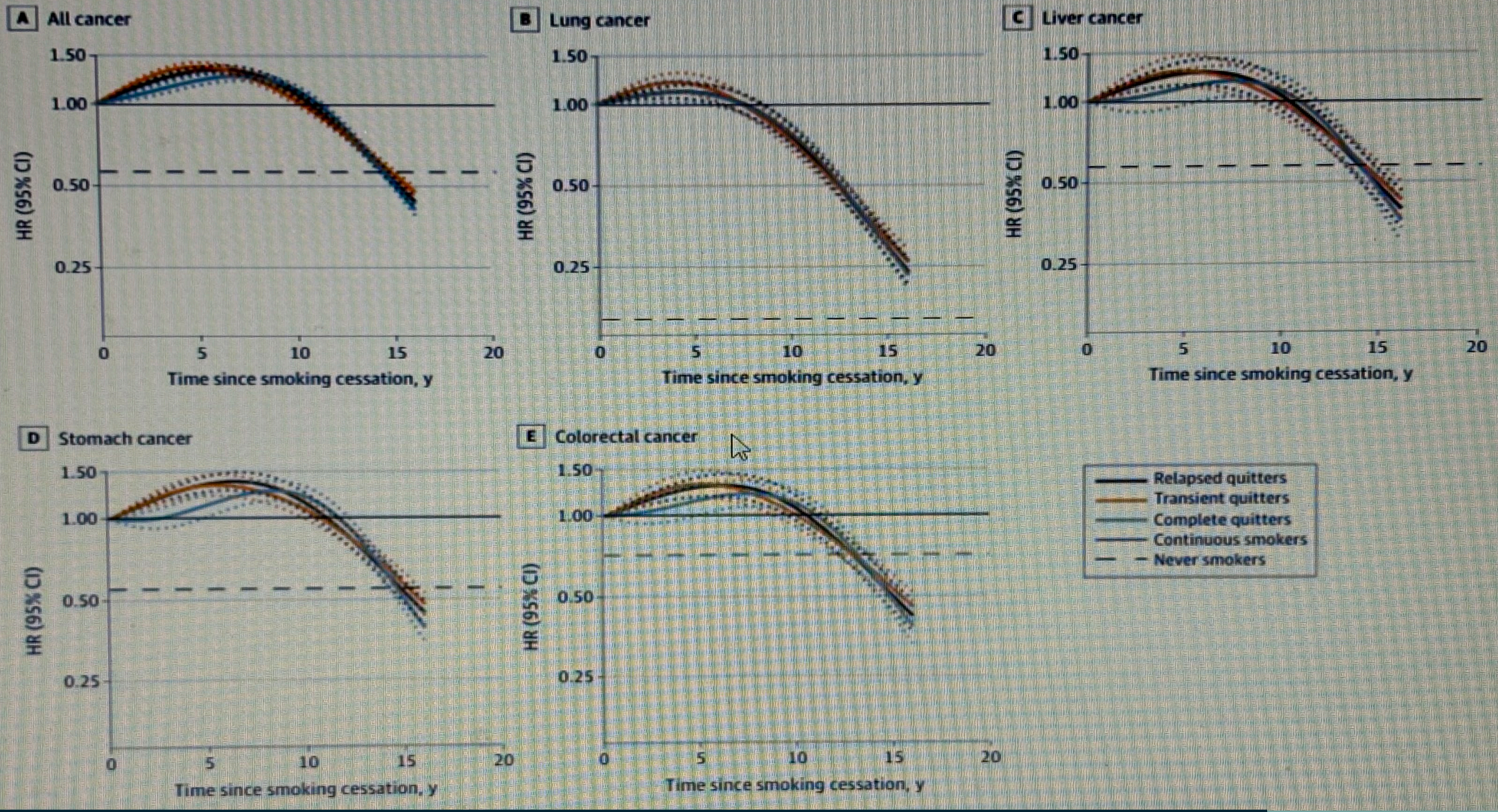
- **Stopping smoking reduces cancer risk at any age, especially after 10 years, according to a new study.**
- **Researchers report that former smokers' risks of developing lung cancer drop the fastest after stopping smoking.**
- **Quitting when you're younger has stronger benefits in cancer risk reduction, but the benefits of quitting after age 50 are still significant, the researchers said**

For their study, researchers looked at a cohort of nearly 3 million people over the age of 30 with an average follow-up of 13 years.

In their findings, researchers reported that those who stopped smoking entirely had a 17% lower overall risk of cancer than those who continued to smoke. That included a 42% lower risk of lung cancer incidence, a 27% lower risk of liver cancer, a 14% lower risk of stomach cancer, and a 20% lower risk of colorectal cancer.

Of all cancers, lung cancer risks declined the most quickly following smoking cessation, falling three years earlier than other cancers. And while quitting before age 50 was better for improving your odds against a lung cancer diagnosis — averaging a 57% lower risk of lung cancer — even quitting after age 50 reduces lung cancer risk by 39% compared to continued smokers, the researchers reported.

Figure 1. Association of Duration of Smoking Cessation With Cancer Incidence Among Men



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GLOBAL HEART
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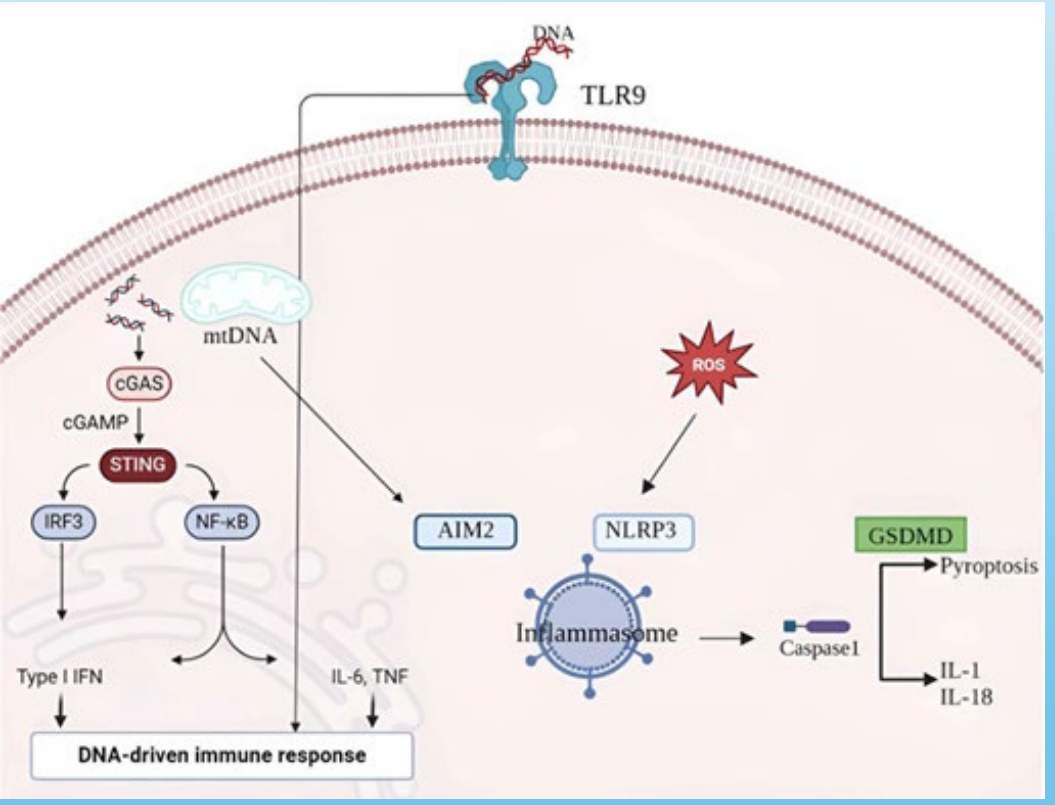
Cardiovascular Effects of Smoking and Smoking Cessation: A 2024 Update



Smoking is one of the most common modifiable risk factors for the development of cardiovascular disease (CVD). Despite clear evidence provided since the 1950s, smoking remains a major risk factor for CVD, which is the leading cause of death among adults worldwide. According to the World Health Organization (WHO) and Global Heart Journal, smoking is responsible for more than 8 million deaths annually, and one in every five cardiovascular patients dies because of smoking. Conditions such as coronary artery disease, cerebrovascular disease, aneurysms, and peripheral artery diseases are among the numerous diseases caused by smoking. Both active and passive exposure to smoke inevitably predisposes to cardiac and vascular disorders.

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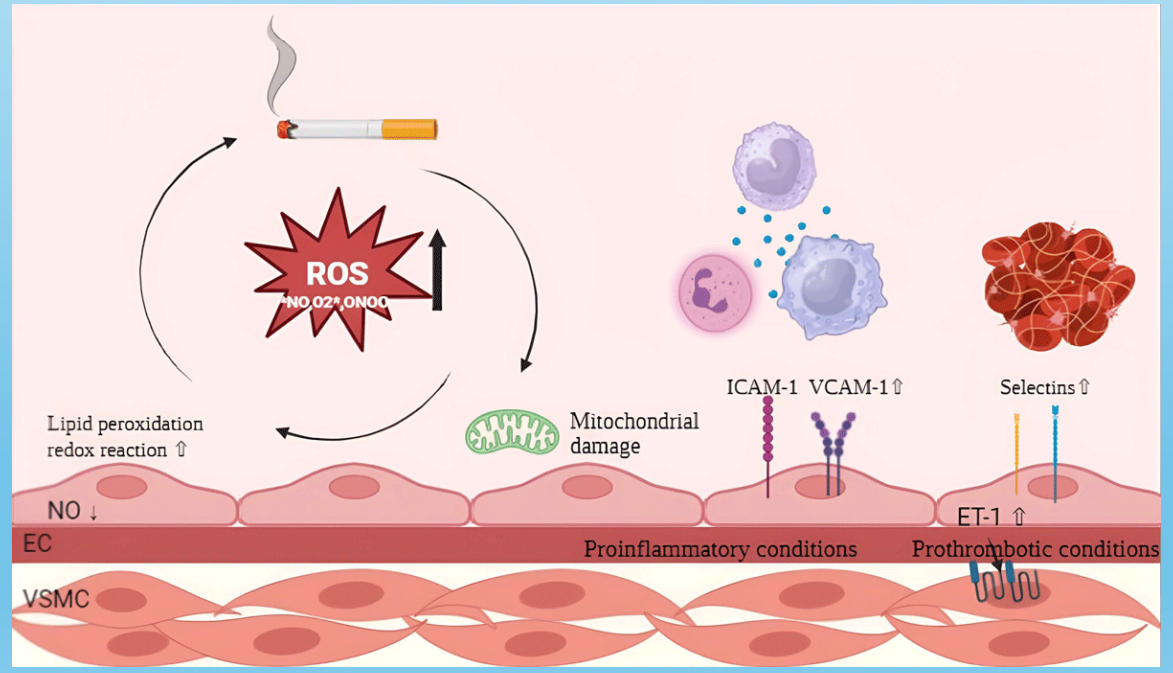
Nicotine effects seem to be induced by stimulation of the nicotinic acetylcholine receptors (nAChRs), located in the Central Nervous System and in other organs in the body that are component of the parasympathetic autonomic nervous system. The increased cardiovascular risk seems to be related to the adrenergic effects of nicotine that result in an increased heart rate, increased inotropic status, increased coronary microvascular resistance and reduced insulin sensitivity

In 2004 the Surgeon General's report listed six pathogenetic mechanisms of smoking-induced heart disease: (I) endothelial damage, (II) prothrombotic effect, (III) inflammation, (IV) abnormal lipid metabolism, (V) increased myocardial oxygen and blood demand and (VI) decreased myocardial blood and oxygen supply



But it is important to focus on the main target of the toxic effects of smoking: the endothelium, a "multitask" organ with anti-inflammatory, antithrombotic and vasomotor properties that regulate and maintain vascular tone and haemostasis. Smoking affects all these important properties and the ensuing endothelial "dysfunction" is the first step of the atherogenic process

Journal of Thoracic Disease

Cardiovascular risk of smoking and benefits of smoking cessation, 2020

Giuseppina Gallucci [1], Alfredo Tartarone [2,✉], Rosa Lerose [3], Anna Vittoria Lalinga [4], Alba Maria Capobianco [2]

Smoking and thrombogenicity

A dysfunctioning endothelium loses its antithrombotic properties, but smoking itself influences platelet aggregability. The balance between proaggregatory thromboxane A2 produced by platelets and antiaggregatory prostacyclin secreted by vascular cells has been studied in habitual smoker; it has been shown that smoking induces proaggregatory conditions thus leading to a hypercoagulable state. Smoking elicits an increased generation of von Willebrand factor, and prothrombotic factors, while impairing the process of fibrinolysis. The activation of platelet and of the coagulation cascade, with a reduction of fibrinolysis in smokers have been confirmed in more recent studies. Sudden cardiac deaths and AMI are frequently caused by acute rupture of a coronary atheromatous plaque with acute coronary artery thrombosis. Cigarette smoking is the leading factor for acute coronary thrombosis, as a consequence sudden cardiac death induced by acute thrombosis are frequent in cigarette smokers. The link between smoking and acute thrombosis has been documented in both active and passive cigarette smoking. Barua *et al.* examined *in vitro* the effects of smoke exposure on clot and fibrin architecture, using thromboelastography, GP IIb/IIa inhibition and electron microscopy. They showed that acute cigarette smoke exposure is associated with shortening of the time for fibrin formation and augmented clot strength, these two mechanisms can explain heightened thrombogenicity of the atheromatous plaques of smokers.

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Smoking and blood pressure

Whereas the influence of smoking on lipid levels and insulin resistance is well documented, data on the effect of smoking on blood pressure are conflicting. In 2010 Virdis *et al.* stated that cigarette smoking has an acute hypertensive effect mediated by the stimulation of the sympathetic nervous system. For chronic smoking available data do not prove that smoking directly induces hypertension, and smoking cessation does not lead to a reduction of blood pressure values, either. Nevertheless, the effect of smoking on arterial stiffness may have a greater impact on central blood pressure that is related to target organ damage more closely if compared to brachial blood pressure. In a more recent study Saladini *et al.* investigated the effect of smoking on peripheral and central blood pressure in a group of young stage I hypertensives. Central systolic blood pressure and pulse pressure were higher in smokers than in nonsmokers, thus implying a predominant effect on central blood pressure.

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Smoking and the immune system

Another intriguing issue is the link between smoking and the immune system. Smoking activates the immune system systemically and locally, increasing white blood cell counts and the level of neutrophils, lymphocytes and monocytes. Increased levels of proinflammatory cytokines are also found in smokers, along with increased level of C-reactive protein. The immunologic alterations induced by smoking affect local inflammatory processes in the vascular wall with increased expression of matrix metalloproteinase, leukocyte recruitment and increased plasma concentration of soluble vascular cell adhesion molecules.

We now know that chronic inflammatory conditions such as rheumatoid arthritis and other autoimmune disorders are risk enhancers for cardiovascular diseases (16) and that there are increasing data that relate a state of low-grade inflammation both to stable cardiovascular disease and to cancer, therefore it can be speculated that the effects of smoking on immune system does have an impact on CVDs

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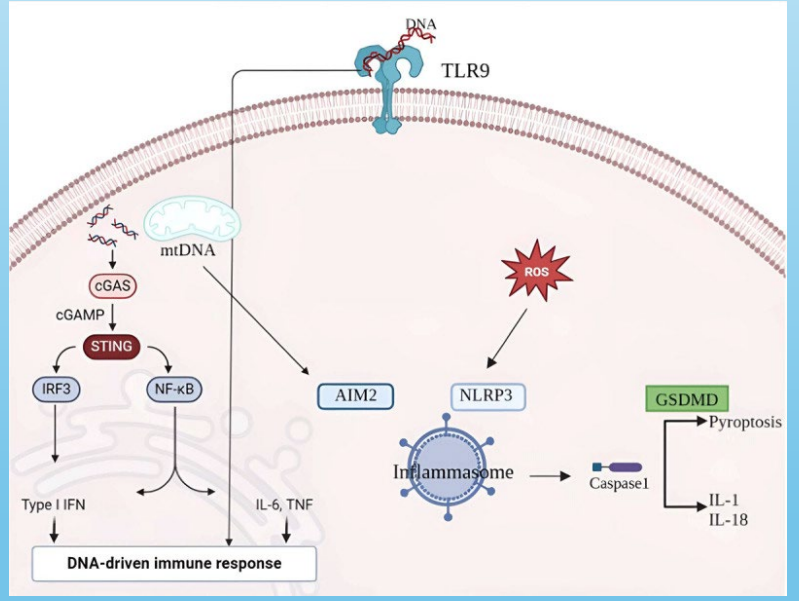

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Giuseppina Gallucci [1], Alfredo Tartarone [2,✉], Rosa Lerose [3], Anna Vittoria Lalinga [4], Alba Maria Capobianco [2]

The “lipid effect”

Besides the direct effect on endothelial function, smoking has an effect on serum lipids that can enhance endothelial damage. Craig and colleagues. showed that smoking increases, in a statistically significant way, total cholesterol, very-low and low- density lipoprotein, and triglyceride serum levels. The Authors also found decreased concentrations of high density lipoprotein (HDL) and apolipoprotein A1 in smokers. Other subsequent clinical studies documented a proatherogenic modification of serum lipid profiles induced by smoking (49,50). Moreover, smoking induces lipid oxidation, oxidatively modified LDLs are captured by macrophages that become foam cells, thus initiating the process of plaque formation (51-53). An interesting observation has been made by Neufeld and colleagues. in children with a high propensity for early onset heart disease (due to a genetic form of dyslipidemia), the Authors found in these children a significant reduced levels of HDL after secondhand smoke in the household, this effect did not show the reversibility with smoking cessation that has been described with the impaired FMD (54). In a population of ~3,000 healthy women (30–70 years) passive smoking was linked to negative effects on glucose and lipid profiles, increasing the risk of diabetes and cardiovascular disease (55).

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The “diabetic effect”

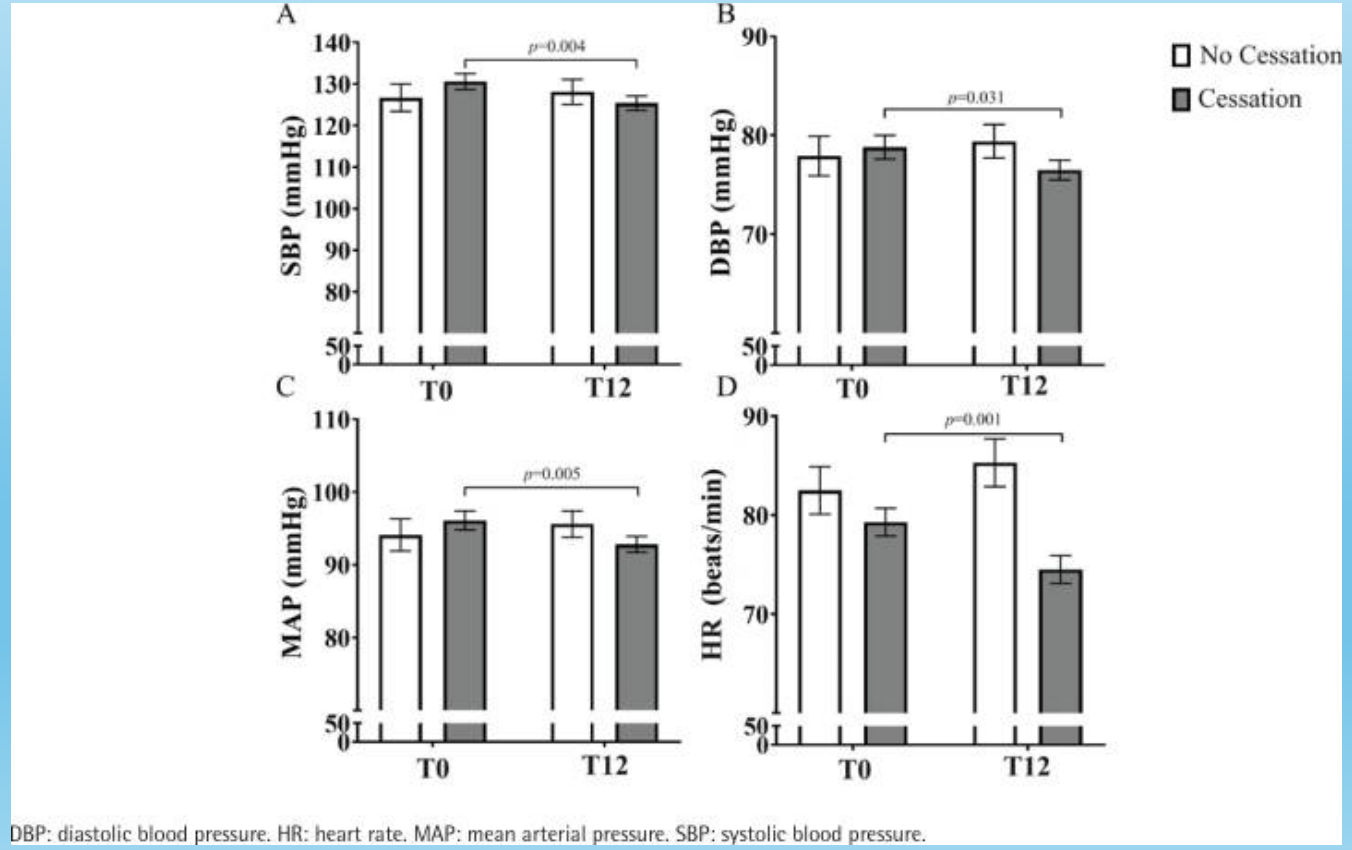
A large body of literature have suggested that smoking increases the risk of type 2 diabetes mellitus (T2DM) with pathogenetic studies supporting the liaison between smoking and T2DM. The first data of a link between smoking and diabetes are from early 90’s of last century, however, T2DM is multifactorial in etiology and the molecular mechanisms of increased incidence of both insulin resistance and T2DM in smokers are not well defined, yet. Smoking has a detrimental effect of endothelial function, increases inflammation/oxidative stress and directly damage β-cell function. Tobacco products contain thousands of chemical compounds and many free radicals that may be responsible of the pathogenetic process that leads to smoke-related insulin resistance or diabetes. In the British Regional Heart Study, active cigarette smokers were confronted with people who never smoked, current cigarette smokers had a higher risk of diabetes, even after adjustments for age, BMI, and other potential confounders. Other studies have linked smoking to insulin resistance. Smoking can affect insulin sensitivity also through epigenetic mechanisms: smoking-induced diabetes susceptibility may be due to aberrant methylation of DNA. Moreover, smoking has an impact on pancreatic β cell function, but the mechanisms are less well known.

.2024 May 16;22:10.18332/tid/186853. doi: 10.18332/tid/186853

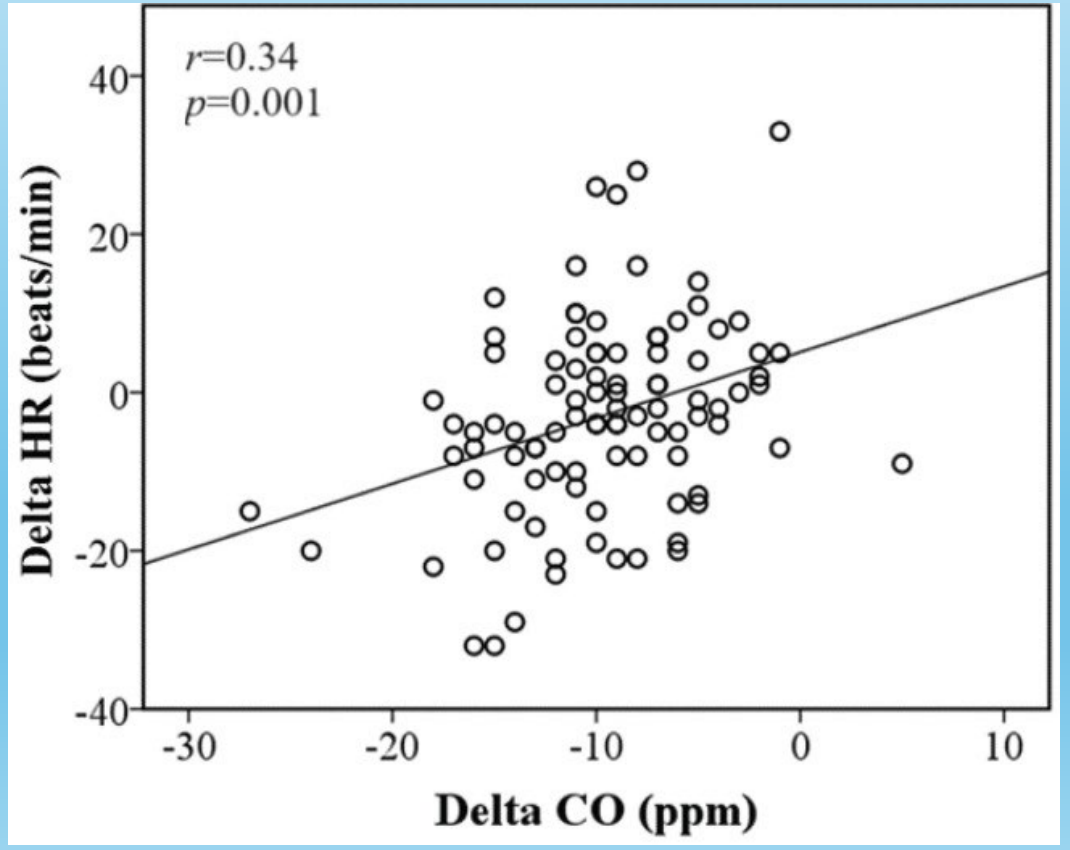
Smoking cessation decreases arterial blood pressure in hypertensive smokers: A subgroup analysis of the randomized controlled trial GENTSMOKING

Patricia V Gaya [1,✉], Guilherme Wesley P Fonseca [2], Lucas Tsuyoshi Tanji [3], Tania O Abe [1], Maria Janieire N N Alves, Gaya [1,✉], Guilherme Wesley P Fonseca [2], Lucas Tsuyoshi Tanji [3], Tania O Abe [1], Maria Janieire N N Alves [4], Paulo Caleb Junior de Lima Santos [5], Fernanda M Consolim Colombo [6], Jaqueline R Scholz

High blood pressure in hypertensive smokers is affected by nicotine consumption. This study aimed to evaluate the effect of smoking cessation treatments on blood pressure in hypertensive smokers



DBP: diastolic blood pressure. HR: heart rate. MAP: mean arterial pressure. SBP: systolic blood pressure.



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Cerebral blood flow

Cigarette smoking has an impact on cerebral blood flow too, increasing the risk of stroke and the severity of stroke. Smoking impairs nitric oxide (NO)-mediated flow increase by reducing NO synthesis in cerebrovascular endothelial cells, thus interfering with blood flow and glucose metabolism in the cerebral circulation. In the brain, as in other vascular bed, the endothelial damage is the “primum movens” of the effect. (NO)-mediated vasodilation is reduced through inhibition of nitric oxide synthase of the endothelium (eNOS) and of the neurons (nNOS) and by the overproduction of oxygen radicals. Nicotine has an acute and chronic effect on the eNOS and negatively affect nitrergic nerve function. These effects induce the synthesis of amyloid beta that speeds up the reduction of blood flow and may be causally related to Alzheimer disease (77). For passive smoking there are many studies that prove a causal relationship between secondhand smoke and increased risk of stroke as documented in the 2014 Surgeon General’ s Report

frontiers in Neurology

SYSTEMATIC REVIEW
published: 25 January 2022
doi: 10.3389/fneur.2021.772373

Cigarette Smoking and Risk of Different Pathologic Types of Stroke: A Systematic Review and Dose-Response Meta-Analysis

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Objectives: To quantify the association of cigarette smoking, including cigarettes per day and quitting duration, with the risk of different types of stroke morbidity and mortality in the general population, and to clarify the shape of the dose-response relations.

Study Selection: Prospective cohort studies and reported on the association between smoking, quitting and the incidence or mortality of stroke were included.

Data Extraction and Synthesis: All available data were converted uniformly to odds ratios (ORs) and were pooled using random-effects meta-analysis with inverse variance weighting. A dose-response meta-analysis was performed to explore the quantitative relationship between different smoking characteristics and the risk of different pathologic types of stroke incidence.

Results: Twenty-five studies with 3,734,216 individuals were included. Compared to never smokers, the pooled ORs of stroke morbidity and mortality were 1.45 (1.24–1.70) and 1.44 (1.23–1.67) among ever smokers and 1.90 (1.55–2.34) and 1.70 (1.45–1.98) among current smokers. The risk of different pathologic types of stroke was also increased among ever and current smokers. There was a significant non-linear dose-response association between the number of cigarette smoking and the risk of stroke incidence. Comparing no smoking, the ORs for smoking five and 35 cigarettes per day were 1.44 (1.35–1.53) and 1.86 (1.71–2.02). Other pathologic types of stroke have a similar dose-response relationship. There was also non-linear dose-response association between the length of time since quitting and risk of stroke. The risk of stroke decreased significantly after quitting for 3 years [OR = 0.56 (0.42–0.74)].

Conclusion: The risk of different types of stroke among smokers is remarkably high. Our findings revealed a more detailed dose-response relationship and have important implications for developing smoking control strategies for stroke prevention.

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Specialty section:
This article was submitted to Stroke, a section of the journal Frontiers in Neurology

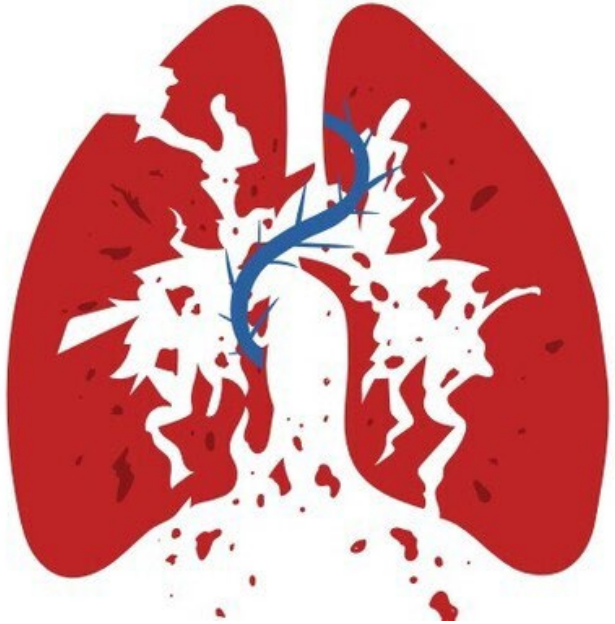
Received: *09 September 2021*
Accepted: *06 December 2021*
Published: *25 January 2022*

Citation:
Luo J, Tang X, Li F, Wen H, Wang L, Ge S, Tang C, Xu N and Lu L (2022) Cigarette Smoking and Risk of

- Ανασκόπηση 25 μελετών
- Συμμετέχοντες 3.734.216 άτομα (αξιολόγηση συχνότητας ΑΕΕ, θανάτου από ΑΕΕ μεταξύ NS, ES, CS)
- Αυξημένο ρίσκο ΑΕΕ x1.45 και θανάτου από ΑΕΕ x1.44 σε ES
- Αυξημένο ρίσκο ΑΕΕ x1.90 και θανάτου από ΑΕΕ x1.70 σε CS

Δήλωση της Ευρωπαϊκής Πνευμονολογικής Εταιρείας (European Respiratory Society) σχετικά με τα νέα προϊόντα νικοτίνης και καπνού, το ρόλο τους στον έλεγχο του καπνού και τη "μείωση της βλάβης"

23 Δεκ 2024



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Ευχαριστώ πολύ