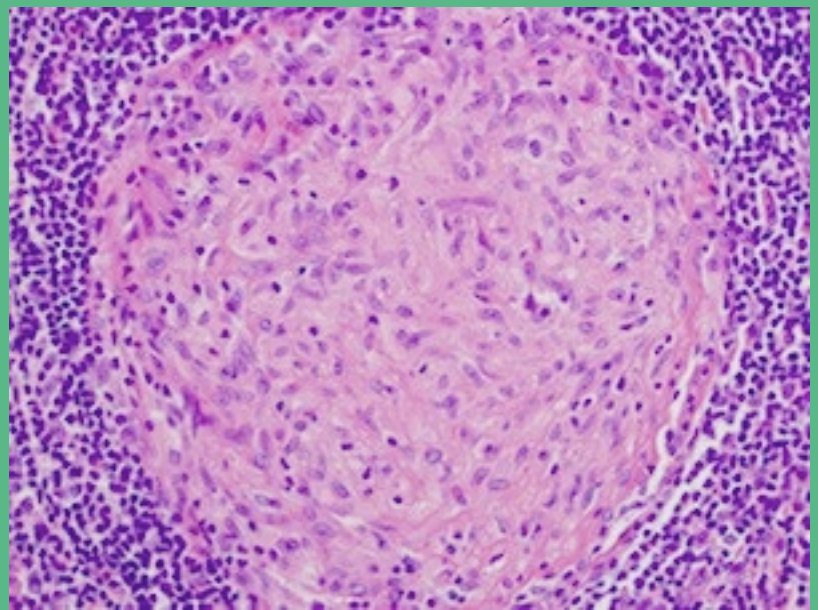
Sarcoïdose et tabac

Dr. Catherine Charpentier
Service de Pneumologie
CHL, Luxembourg

SFT, Ajaccio, 21 novembre 2019



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Sarcoïdose et tabac

Je n'ai pas de conflit d'intérêt.

Je n'ai aucun lien avec l'industrie du tabac, de l'alcool ou des jeux, ni avec l'industrie de la cigarette électronique

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Sarcoïdose et tabac

- La sarcoïdose
- Tabac et sarcoïdose
- Hypothèses de cette relation
- Conclusion

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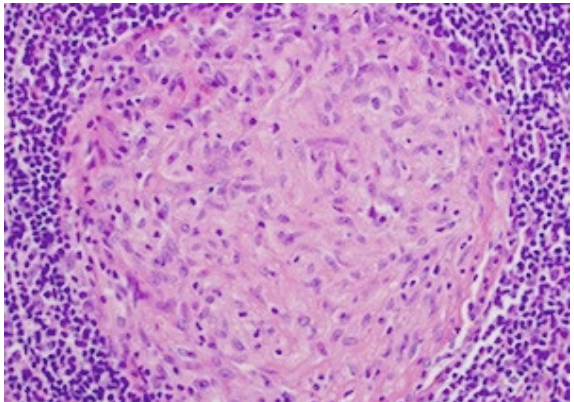
Sarcoïdose

- Maladie rare, multisystémique: touchant en général 2 ou 3 organes, avec atteinte pulmonaire interstitielle et lymphatique (95% des cas), de découverte fortuite souvent et d'étiologie indéterminée
- Caractérisée par des granulomes épithélioïdes non nécrosants, non caséeux avec une accumulation de T-lymphocytes et de macrophages
- 2 phénotypes: inflammatoire et fibrotique



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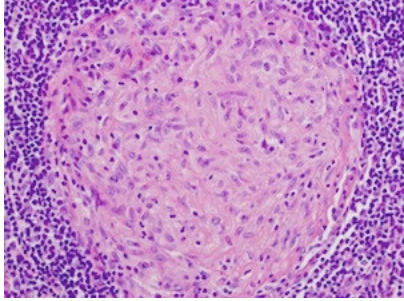
Sarcoïdose



- Prévalence: 30-40 / 100 000 hab. en France
- Incidence: 1 à 5 /100 000 hab. (Jamilloux, ERJ 2016)
- Sexe ratio F>H
- Durée d'évolution généralement < 2-3 ans
- D'évolution favorable dans plus de 2/3 des cas
- Mais aussi responsable de complications graves pulmonaires (fibrose, HTAP, greffe aspergillaire, ..) et d'autres localisations (cardiaque, ophtalmo, rénale,…)
- Mortalité de 0.5 à 5%.

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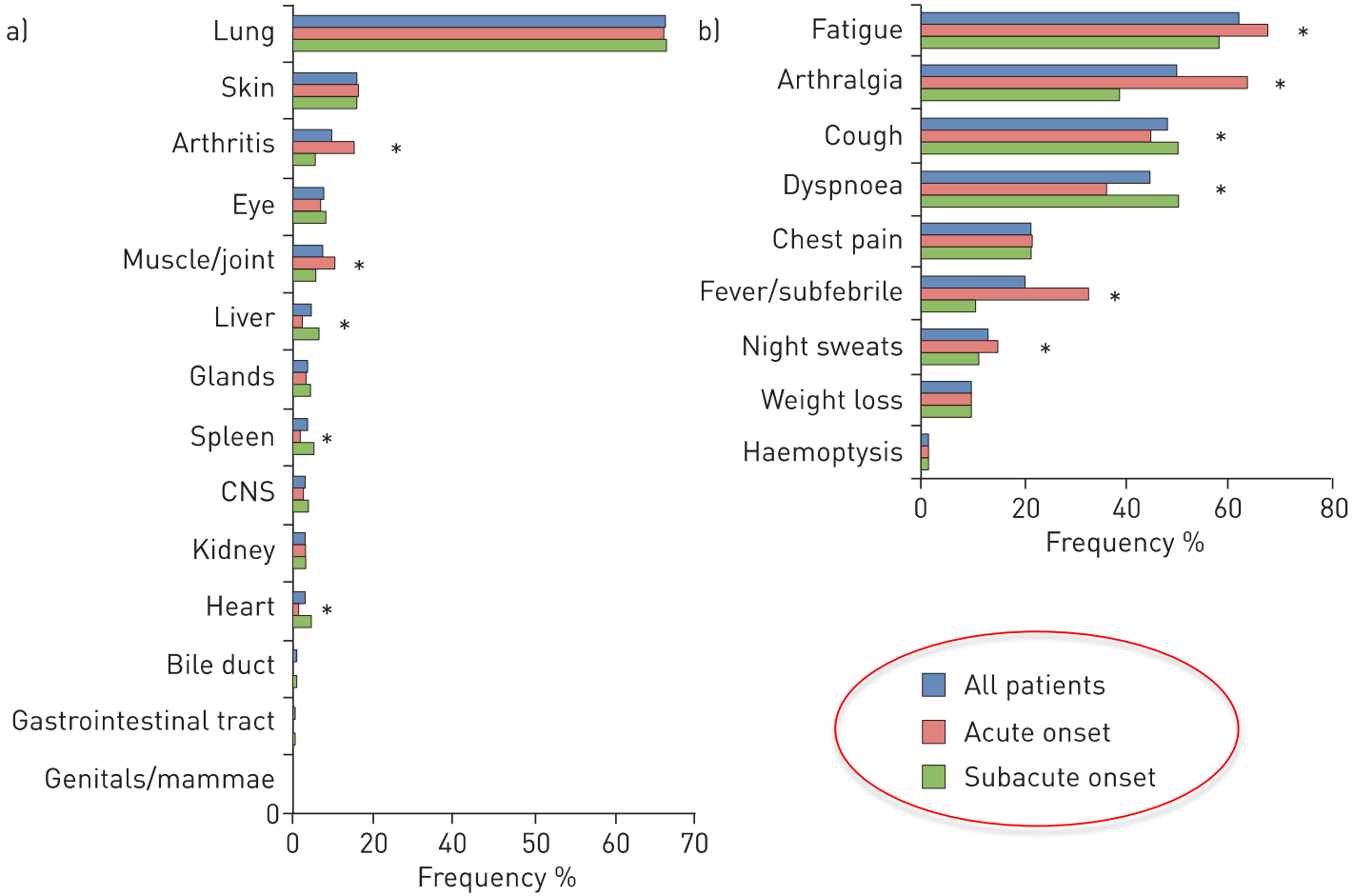
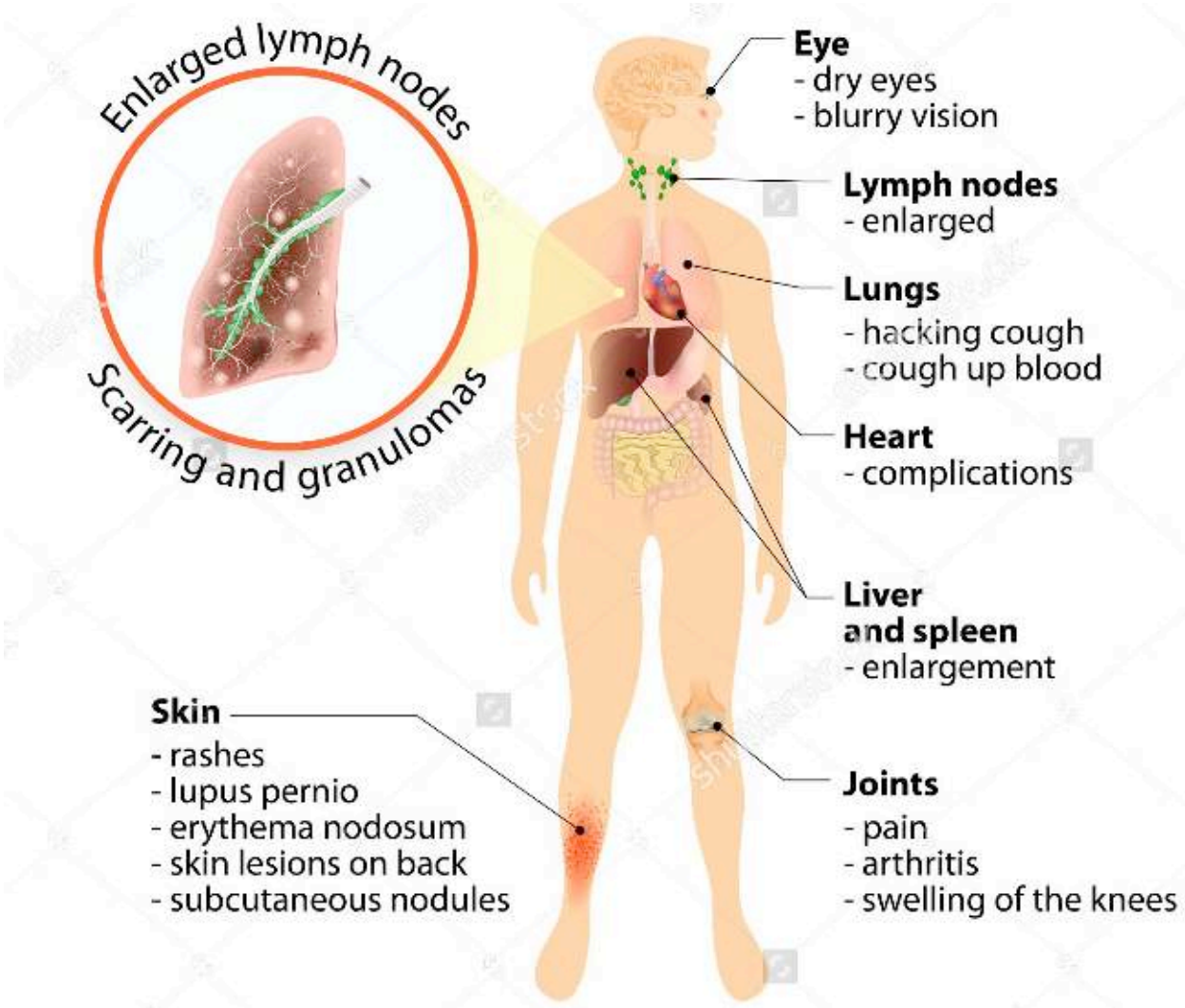
Sarcoïdose: formes cliniques



Schupp, ERJ 2018

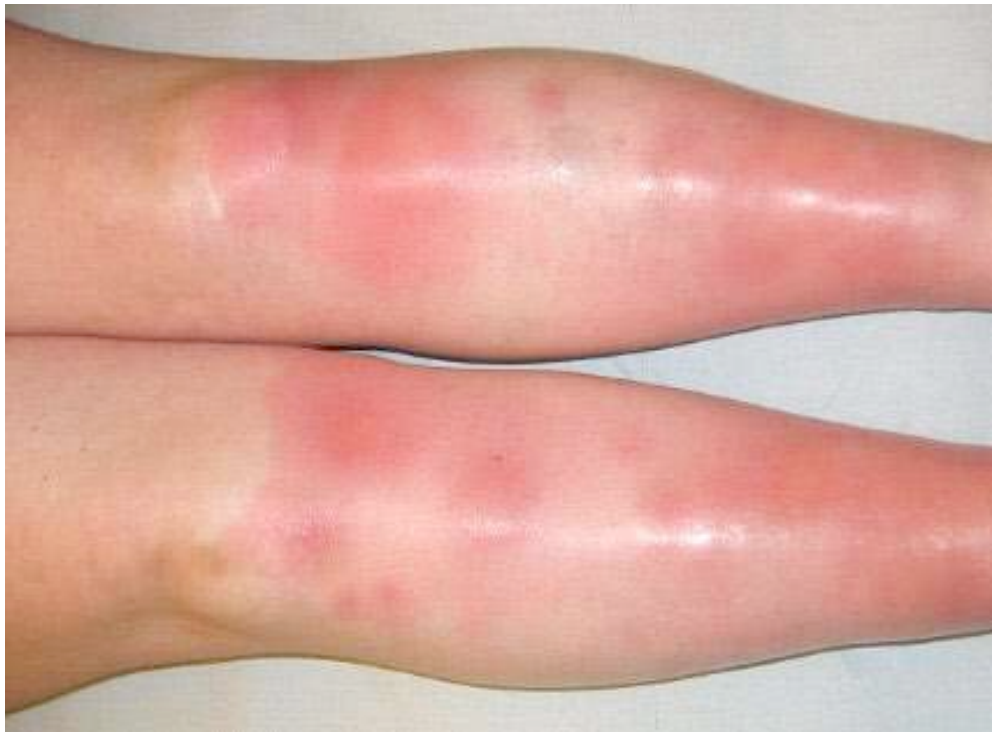
Sarcoïdose

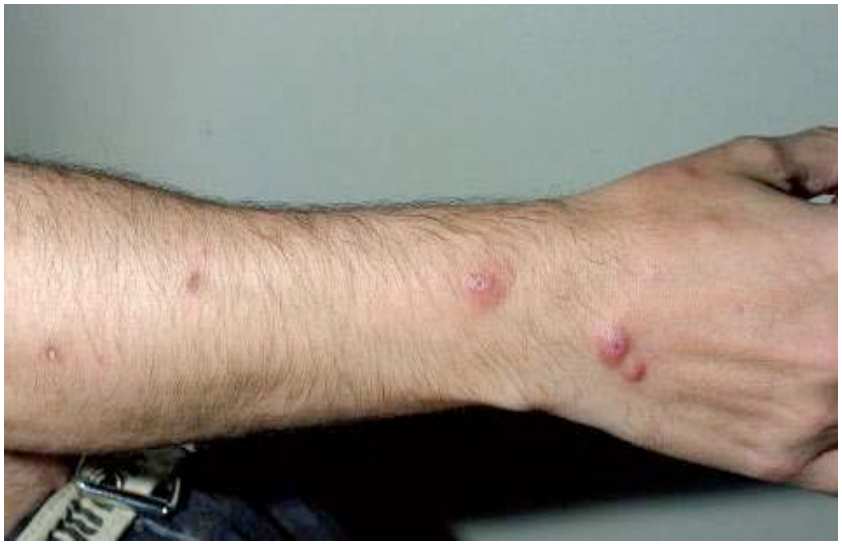

Enlarged lymph nodes
Scarring and granulomas
Eye
- dry eyes
- blurry vision
Lymph nodes
- enlarged
Lungs
- hacking cough
- cough up blood
Heart
- complications
Liver
and spleen
- enlargement
Skin
- rashes
- lupus pernio
- erythema nodosum
- skin lesions on back
- subcutaneous nodules
Joints
- pain
- arthritis
- swelling of the knees

Sarcoïdose

Atteintes cutanées

Erythème noueux





Sarcoïdes

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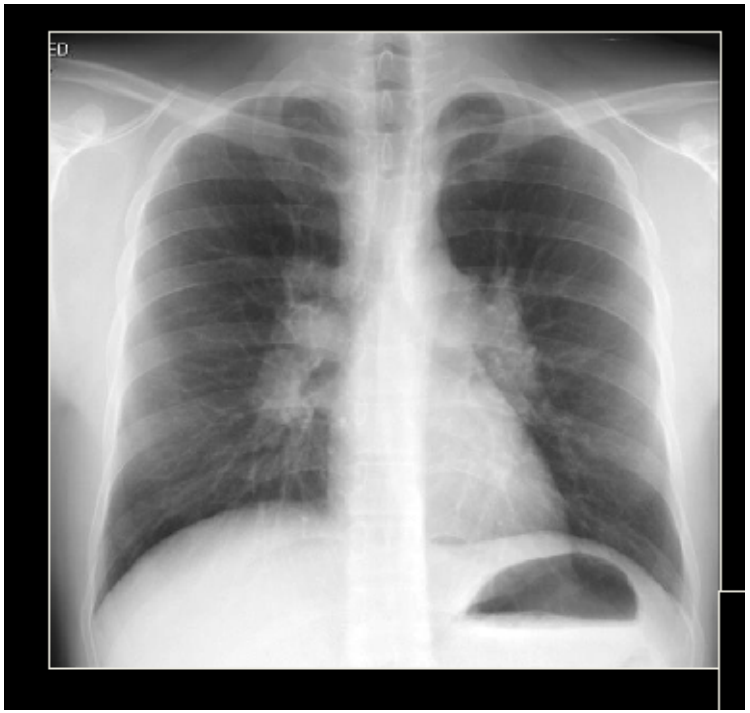
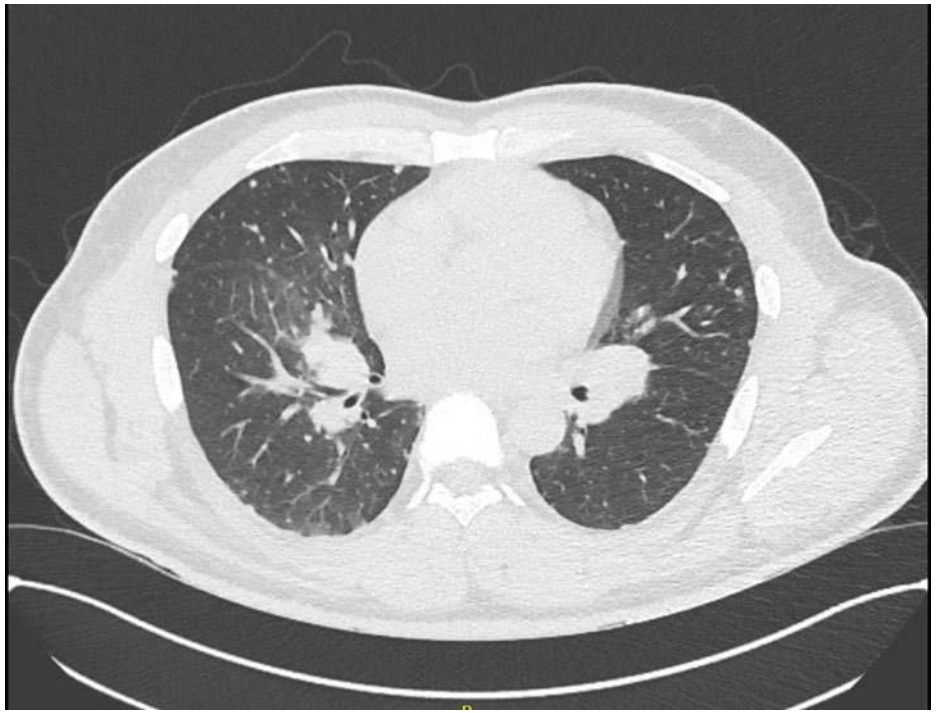
Classification de la sarcoïdose

Stade I : Adénopathies hilaires bilatérales

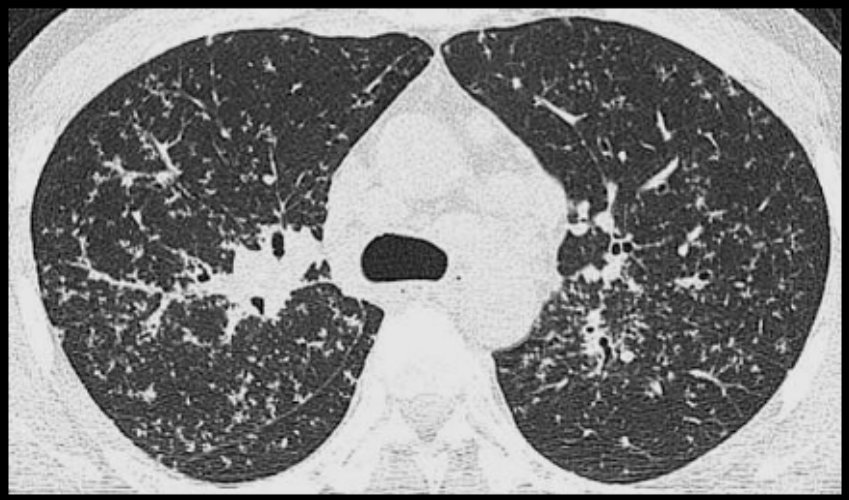


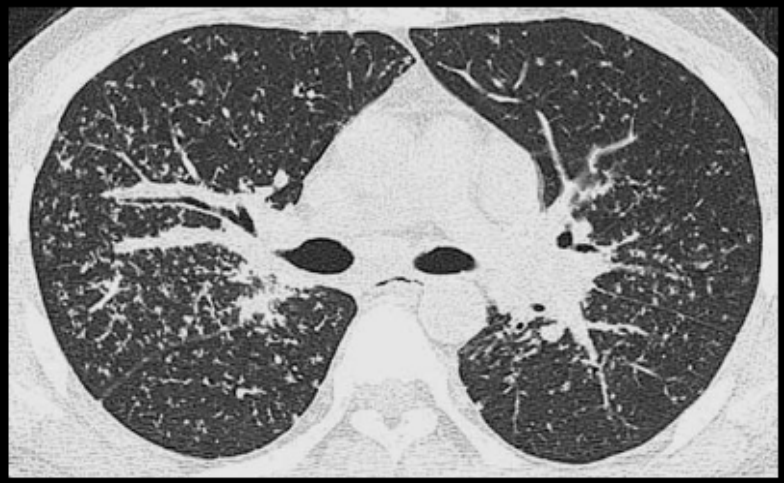


Classification de la sarcoïdose

Stade I : Adénopathies hilaires bilatérales

Stade II: Adénopathies hilaires bilatérales et infiltrat interstitiel



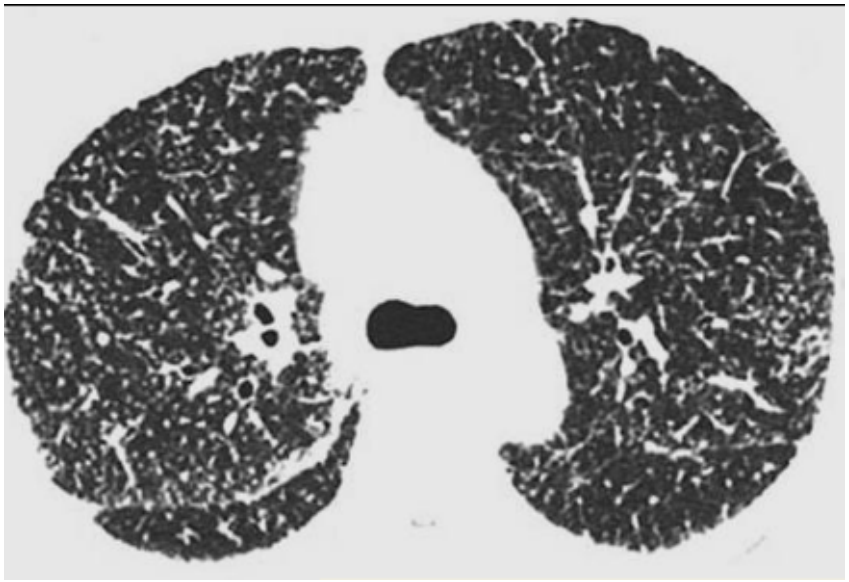


Classification de la sarcoïdose

Stade I : Adénopathies hilaires bilatérales

Stade II: Adénopathies hilaires bilatérales et infiltrat interstitiel

Stade III : Infiltrat interstitiel seul



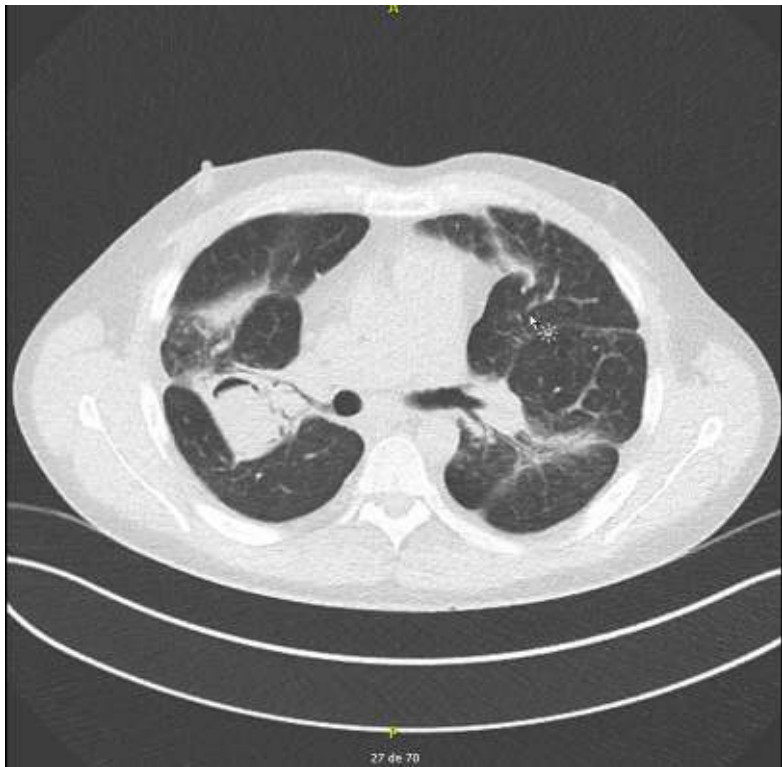
Classification de la sarcoïdose

Stade I : Adénopathies hilaires bilatérales

Stade II: Adénopathies hilaires bilatérales et infiltrat interstitiel

Stade III : Infiltrat interstitiel seul

Stade IV: Fibrose pulmonaire



Sarcoïdose:
Facteurs génétiques

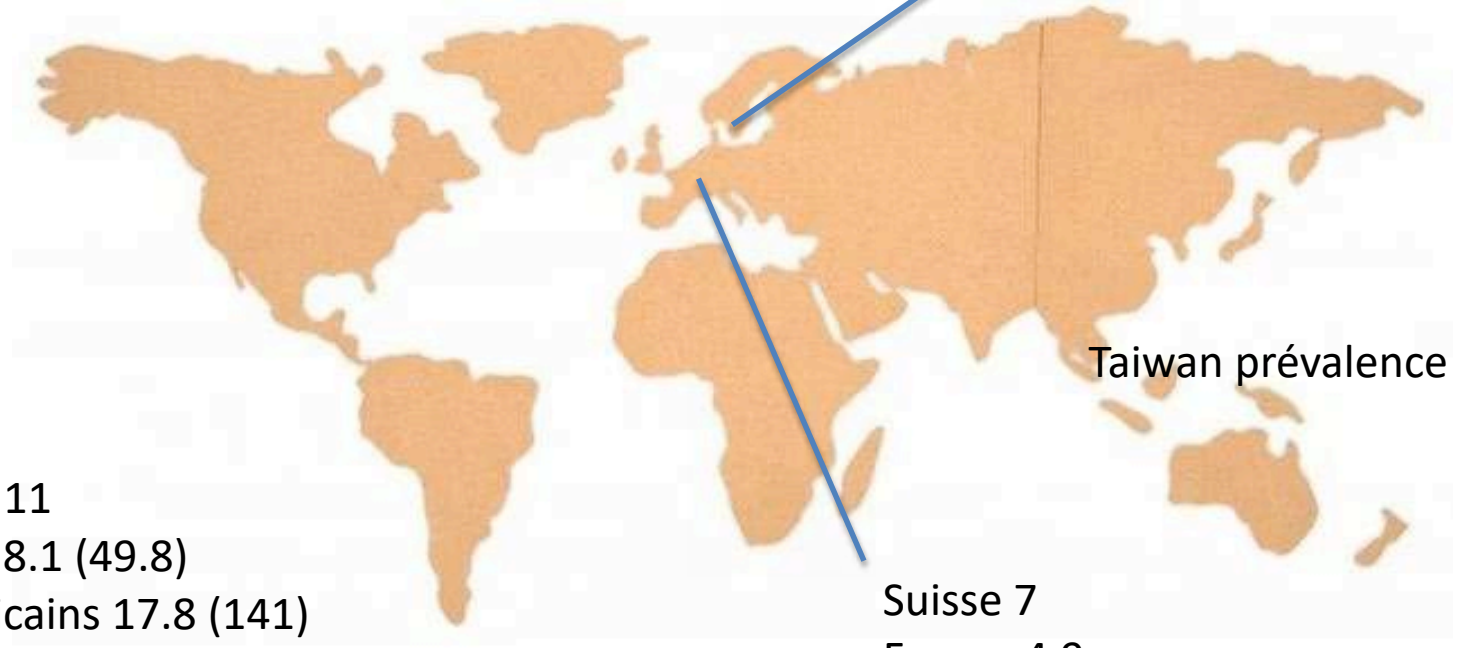
- Incidence variable selon les régions, selon les populations, existence de cas familiaux:

  ->Facteurs génétiques HLA classe 2

Sarcoïdose

Incidence (Prévalence): 4.7-64/100.000 hab

Etudes 2010-2017



Américains 11
Caucasiens 8.1 (49.8)
Noirs américains 17.8 (141)
Hispaniques 3
Asiatiques 4
Noirs antillais 2.3

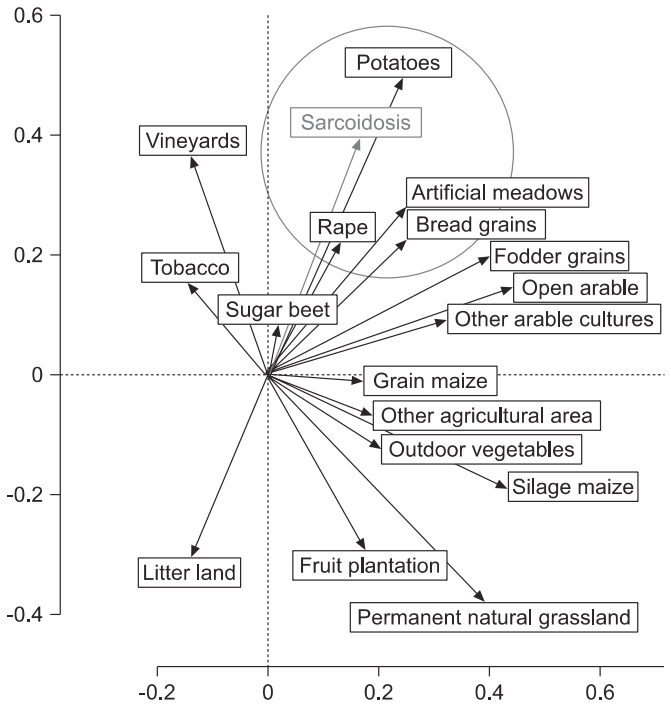
Arkema, Ther Adv Chronic Dis 2018
Deubelbeiss, ERJ 2010

Sarcoïdose:
Facteurs environnementaux

. Rôle des insecticides, de l'environnement agricole et de l'exposition à des aérosols biologiques microbiens
Newman, AJRCCM 2004

. Pompiers du WTC
Miller, Chest 2007

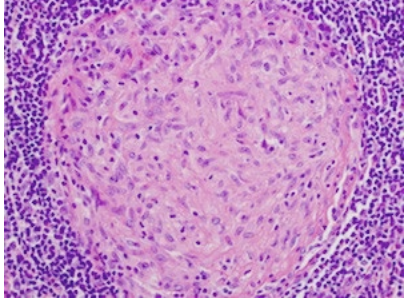
. Ile de Man: contagiosité?



-> Influences génétiques (sexe, ethnie) et environnementales (climat, industries, cultures,…)

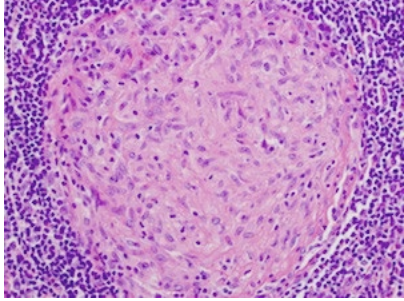
Deubelbeiss, ERJ 2010
Ramos-Casals, Lung 2019

Sarcoïdose
Diagnostic



- Faisceau d'arguments cliniques, RX et CT
- Fibroscopie bronchique:
  - Stade 1 et 2 : EBUS et Biopsies TB
  - Stade 3 et 4 : LBA et Biopsies
- Histologie: granulome non caséeux
- Absence d'autre diagnostic
- Différentiel: tuberculose, lymphome, lymphangite

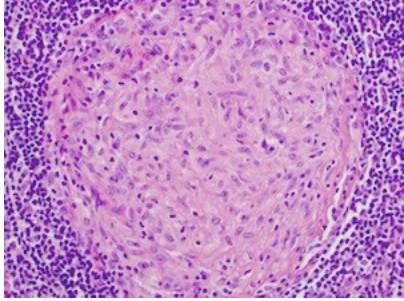
Sarcoïdose



- Mesure de l'activité de la maladie:
  - Clinique: majoration de la dyspnée, toux,
  - Imagerie: CT, Pet
  - LBA: lymphocytose
  - Biologie: angiotensine convertase, IL2 récepteurs, calcium urinaire de 24 heures
  - Tests fonctionnels respiratoires

Sarcoïdose

Traitement

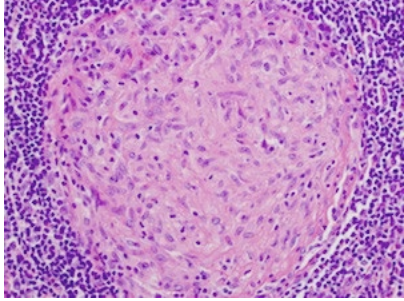


- Corticoïdes
- Cytotoxiques: Méthotrexate, Azathioprine
- Anti-TNF : Infliximab, Retuximab
- TTT pour HTAP, Fibrose, Infectieux
- Transplantation
- Epargner au max les corticoïdes

Deubelbeiss, ERJ 2010

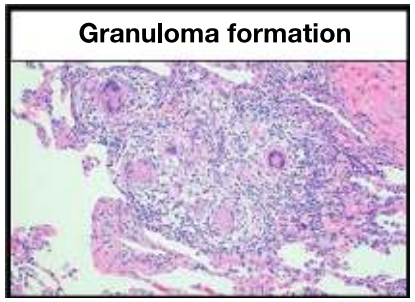
Sarcoïdose:

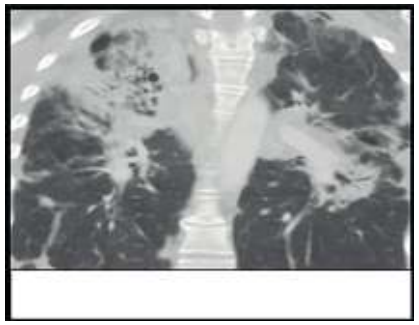
Hypothèses pathogéniques



- Réaction immunitaire exagérée au niveau de différents territoires cibles, particulièrement les poumons.
- Accumulation locale des macrophages qui se transforment progressivement en cellules épithélioides auxquelles s'associent des lymphocytes et des cellules géantes multinucléées.

Pathogénie de la sarcoidose





Patterson, Chest 2018

"activity." Cigarette smoking was associated with a significant increase in the serum angiotensin converting enzyme activity (SACE), and patients with very high SACE and pulmonary gallium-67 uptake were smokers. Furthermore, more $CD8^+$ (but not $CD4^+$) lymphocytes were recovered by lavage from smoking than from non-smoking patients, giving a lower CD4 :CD8 ratio in smokers. Fewer alveolar macrophages were recovered by lavage from smokers with sarcoidosis than from normal subjects with a similar smoking history. These findings support the possibility that smokers, particularly those with a prominent accumulation of alveolar macrophages in the lower respiratory tract, may be less likely to develop sarcoidosis.

Introduction

Pulmonary sarcoidosis is a granulomatous lung

alterations in the number, type, and functional activity of lung immune and inflammatory cells.[3,4] We and others have suggested that the incidence of sarcoidosis

. Etude de 64 patients avec sarcoïdose suivis 1 an avec groupe contrôle
. 30% vs 46% de fumeurs sarcoïdose vs contrôle
. 2 hypothèses: fumer diminue le risque de sarcoïdose ou fumer modifie l'évolution de la maladie qui est moins sévère
. Pas d'argument pour corrélation tabac-sévérité de la maladie
. Critères d'activité + élevés chez les fumeurs (SACE, scinti au gallium*, rapport CD4/CD8 (plus bas chez les fumeurs), Moins de macrophages au LBA

Thorax 1988;43:516-24

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ournal homepage: www.elsevier.com/locate/rmed

Smoking, obesity and risk of sarcoidosis: A population-based nested case-control study

Patompong Ungprasert [a, *], Cynthia S. Crowson [a, b], Eric L. Matteson [a, c]

Characteristics of sarcoidosis cases and controls at sarcoidosis incidence/index date.

	Cases (N = 345)	Controls (N = 345)	P value
Mean age in years (±SD)	45.6 (±13.6)	45.4 (±13.7)	0.87
Female sex	174 (50%)	174 (50%)	1.0
Ethnicity			<0.001
Caucasian	301 (90%)	327 (95%)	
African-American	18 (5%)	2 (1%)	
Asian	6 (2%)	0 (0%)	
Native American	2 (1%)	0 (0%)	
Other	7 (2%)	14 (4%)	
Unknown	11	327 (95%)	
Mean length of follow-up in years (±SD)	15.1 (±10.5)	16.8 (±10.8)	–
Smoking status			<0.001
Never	198 (60%)	132 (42%)	
Ex-smoker	71 (21%)	70 (22%)	
Current smoker	63 (19%)	115 (36%)	
Unknown	13	28	

Sarcoïdose et tabac
Hypothèses d'une relation

Nombre de macrophages alvéolaires dans le LBA en fonction du tabagisme

Sujets contrôle

Patients sarcoidose

Smoking and pulmonary sarcoidosis

Table 2 *Numbers and types of cells recovered by lavage fr*
as a function of smoking history (means with standard devi

Study group	*n*	*Cells recovered by lav* *Total cells*	*Macr*
SARCOIDOSIS			
Non-smokers	45		
Cells × 10^3/ml		280 (13)††	160 (
%			59 (
Smokers	19		
Cells × 10^3/ml		394 (184)*†	269 (
%			68 (
NORMAL SUBJECTS			
Non-smokers	15		
Cells × 10^3/ml		155 (68)	136 (
%			88 (
Smokers	8		
Cells × 10^3/ml		535 (327)***	488 (

Hypothèse: les macrophages alvéolaires plus nombreux chez le fumeur protégeraient de la sarcoïdose

Valeyre, Thorax 1988;43:516-24

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Sarcoïdose et tabac:
Hypothèses d'une relation

smoking patients had clearly increased serum immunoglobulin concentrations ($p > 0{\cdot}2$ for smokers *v* non-smokers with sarcoidosis by χ^2 analysis).

Pulmonary gallium uptake

No significant difference in ^{67}Ga accumulation was observed between non-smoking and smoking patients (fig 2*a*). The two patients with very high ^{67}Ga indices were both heavy smokers (>20 cigarettes/day).

Serum angiotensin converting enzyme activity

In the patients with sarcoidosis SACE activity was

RELATION OF CIGARETTE SMOKING TO THE EVOLUTION OF SARCOIDOSIS

Treatment with corticosteroids

Information on the clinical course during a follow up period of one year was available for 46 (72%) of the 64 patients with sarcoidosis initially evaluated. There was no significant difference in the number of non-smoking patients (10/33) and smoking patients (5/13) who received corticosteroids ($p > 0{\cdot}2$). The primary indication for steroid treatment in both groups was usually the presence of extrapulmonary sarcoidosis or severe systemic symptoms (9/10 non-smokers and 4/5

Valeyre, Thorax 1988;43:516-24

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"activity." Cigarette smoking was associated with a significant increase in the serum angiotensin converting enzyme activity (SACE), and patients with very high SACE and pulmonary gallium-67 uptake were smokers. Furthermore, more $CD8^+$ (but not $CD4^+$) lymphocytes were recovered by lavage from smoking than from non-smoking patients, giving a lower CD4 :CD8 ratio in smokers. Fewer alveolar macrophages were recovered by lavage from smokers with sarcoidosis than from normal subjects with a similar smoking history. These findings support the possibility that smokers, particularly those with a prominent accumulation of alveolar macrophages in the lower respiratory tract, may be less likely to develop sarcoidosis.

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. **Hypothèse 1**: Les macrophages alvéolaires plus nombreux chez le fumeur protégeraient de la sarcoïdose

. **Hypothèse 2**: Alvéolite de la sarcoïdose est due à une prolifération de lymphocytes dans le poumon qui sont inhibés par l'excès de macrophages chez le fumeur

.

Valeyre, Thorax 1988;43:516-24
Douglas, Thorax 1986;41:787-91

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Sarcoïdose et tabac

- Facteurs environnementaux positifs associé à la sarcoïdose: pas de consensus
- Un seul facteur environnemental négatif associé à la sarcoïdose: le tabagisme actif ou passé
- Odds ratio de 0.65 (0.51-0.82, $p<0.001$)
- Retrouvé dans plusieurs études
- Démontré également dans la pneumonie d'hypersensibilité et la bérylliose chronique

Newman, Am J Respir Crit Care Med, 2004; vol170:1324-1330

Sarcoïdose et tabac

Auteurs	Effectif	
Douglas *Thorax 1986*	183	0.36 (0.25-0.52) vs 1.00
Valeyre *Thorax 1988*	133	30% Tabac+Sarc vs 46% Témoins ($p<0.05$)
Newman Am J Respir Crit Care Med 2004	1442	0.65 (0.51-0.82)
Ungprasert *Respiratory medicine 2016*	690	0.34 (0.23-0.50) vs 1.00
1 étude japonaise *Respirology 2013*		Plus de fumeurs chez les patients avec sarcoïdose

Probable influence de facteurs géographiques Ramos-Casals, Lung 2019

Sarcoïdose: mortalité et tabac

Les fumeurs ont-ils un meilleur pronostic?

Treated (intention to treat)	328	6·20 (3·13–12·27)	0·001
Not treated (intention to observe)	175	6·19 (2·44–10·64)	<0·0001
Smoker	172	3·62 (1·68–7·79)	0·001
Non smoker	331	9·76 (4·44–15·27)	<0·0001
Emphysema	149	2·85 (1·35–6·01)	0·006
No emphysema	354	10·61 (4·80–20·41)	<0·0001
Biopsy	302	5·89 (2·99–11·57)	<0·0001
No biopsy	201	7·74 (2·98–16·07)	<0·0001

Table 4: **Mortality expressed as hazard ratios on univariate analysis for**

2 processus pathologiques de mauvais pronostic: fibrose interstitielle et HTAP

Walsh, Lancet Respir 2014

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Sarcoïdose et tabac : résumé

- Sarcoïdose généralement moins fréquente chez le fumeur
- mais persiste plus longtemps et
- évolue plus souvent vers la fibrose
- avec une mortalité plus élevée toutes causes confondues

Pereira, Current Opin Pulm Med 2014

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Smoking, Use of Moist Snuff, and Risk of Chronic Inflammatory Diseases

Cecilia Carlens[1], Maria-Pia Hergens[2], Johan Grunewald[3], Anders Ekbom[4], Anders Eklund[3], Caroline Olgart Höglund[3,5], and Johan Askling[1,4]

[1]Rheumatology Unit, Department of Medicine, Karolinska University Hospital and Institute; [2]Department of Medical Epidemiology and Biostatistics,

	Rheumatoid Arthritis	Ulcerative Colitis	Crohn's Disease	Sarcoidosis	Multiple Sclerosis
Current (or ever) smoking	Increased risk (1–3)	Decreased risk (4, 9–11)	Increased risk (4, 10, 11)	Decreased risk (12–15)	Increased risk (6–8, 24)
Smoking cessation	Attenuated increased risk (1–3)	Increased risk (4, 9–11)	Attenuated increased risk (4, 10, 11)		Attenuated increased risk (24)

	Rheumatoid Arthritis: RR (95% CI); n = cases	Ulcerative Colitis: RR (95% CI); n = cases	Crohn's Disease: RR (95% CI); n = cases	Sarcoidosis: RR (95% CI); n = cases	Multiple Sclerosis: RR (95% CI); n = cases
Never-users of tobacco	1.0 (reference); n = 129	1.0 (reference); n = 284	1.0 (reference); n = 157	1.0 (reference); n = 145	1.0 (reference); n = 37
Ever-smoker	2.1 (1.7–2.5); n = 641	1.3 (1.1–1.5); n = 616	1.5 (1.2–1.8); n = 405	0.5 (0.4–0.5); n = 135	1.9 (1.4–2.6); n = 150
Ever-user of moist snuff	1.0 (0.9–1.2); n = 168	1.1 (0.9–1.2;) n = 305	0.9 (0.8–1.1); n = 174	1.1 (0.9–1.4); n = 103	1.0 (0.8–1.4); n = 64

	Rheumatoid Arthritis		Ulcerative Colitis		Crohn's Disease		Sarcoidosis		Multiple Sclerosis	
	RR (95% CI)	IR	RR (95% CI)	IR	RR (95% CI)	IR	RR (95% C)	IR	RR (95% C)	IR
Never-user of tobacco	1.0 (reference); n = 129	8	1.0 (reference); n = 284	16	1.0 (reference); n = 157	9	1.0 (reference); n = 145	9	1.0 (reference); n = 37	2
Ever-smoker, never-use of moist snuff	2.3 (1.9–2.7); n = 500	21	1.2 (1.1–1.4); n = 425	19	1.5 (1.3–1.9); n = 297	13	0.5 (0.4–0.6); n = 94	4	2.5 (1.7–3.6); n = 113	6
Ever-use of moist snuff, never-smoker	1.2 (0.8–1.8); n = 27	7	1.0 (0.8–1.2); n = 114	15	1.0 (0.8–1.4); n = 66	9	1.1 (0.8–1.5); n = 62	10	1.8 (1.1–2.9); n = 27	4
Ever-smoker and ever-user of moist snuff	2.0 (1.6–2.6); n = 141	17	1.4 (1.1–1.6); n = 191	22	1.4 (1.1–1.8); n = 108	12	0.5 (0.4–0.8); n = 41	5	1.9 (1.2–3.1); n = 37	4

Carlens, AJRCCM, 2010

Smoking, Use of Moist Snuff, and Risk of Chronic Inflammatory Diseases

Cecilia Carlens[1], Maria-Pia Hergens[2], Johan Grunewald[3], Anders Ekbom[4], Anders Eklund[3], Caroline Olgart Höglund[3,5], and Johan Askling[1,4]

[1]Rheumatology Unit, Department of Medicine, Karolinska University Hospital and Institute; [2]Department of Medical Epidemiology and Biostatistics,

TABLE 4. RELATIVE RISKS WITH 95% CONFIDENCE INTERVALS AND INCIDENCE RATES OF RHEUMATOID ARTHRITIS, ULCERATIVE COLITIS, CROHN'S DISEASE, SARCOIDOSIS, AND MULTIPLE SCLEROSIS AMONG EVER-SMOKERS NEVER USING SNUFF, EVER–SNUFF USERS WHO WERE NEVER-SMOKERS, AND AMONG EVER-SMOKERS AND SNUFF USERS IN COMBINATION

	Rheumatoid Arthritis		Ulcerative Colitis		Crohn's Disease		Sarcoidosis		Multiple Sclerosis	
	RR (95% CI)	IR	RR (95% CI)	IR	RR (95% CI)	IR	RR (95% C)	IR	RR (95% C)	IR
Never-user of tobacco	1.0 (reference); n = 129	8	1.0 (reference); n = 284	16	1.0 (reference); n = 157	9	1.0 (reference); n = 145	9	1.0 (reference); n = 37	2
Ever-smoker, never-use of moist snuff	2.3 (1.9–2.7); n = 500	21	1.2 (1.1–1.4); n = 425	19	1.5 (1.3–1.9); n = 297	13	0.5 (0.4–0.6); n = 94	4	2.5 (1.7–3.6); n = 113	6
Ever-use of moist snuff, never-smoker	1.2 (0.8–1.8); n = 27	7	1.0 (0.8–1.2); n = 114	15	1.0 (0.8–1.4); n = 66	9	1.1 (0.8–1.5); n = 62	10	1.8 (1.1–2.9); n = 27	4
Ever-smoker and ever-user of moist snuff	2.0 (1.6–2.6); n = 141	17	1.4 (1.1–1.6); n = 191	22	1.4 (1.1–1.8); n = 108	12	0.5 (0.4–0.8); n = 41	5	1.9 (1.2–3.1); n = 37	4

Definition of abbreviations: CI = confidence interval; IR = incidence rate per 100,000 person-years standardized to age distribution of person-years experienced by all workers, using 5-year age categories; RR = relative risk.

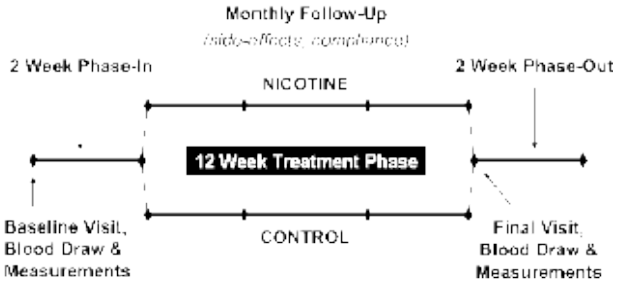
. Des études expérimentales ont montré que la nicotine diminue la cellularité du LBA et la production des cytokines: effet immunomodulateur de la nicotine

. mais la consommation de snus ne diminue pas le risque de sarcoïdose.

Cette étude est en faveur du rôle protecteur d'un constituant de la fumée de cigarette

Carlens, AJRCCM, 2010

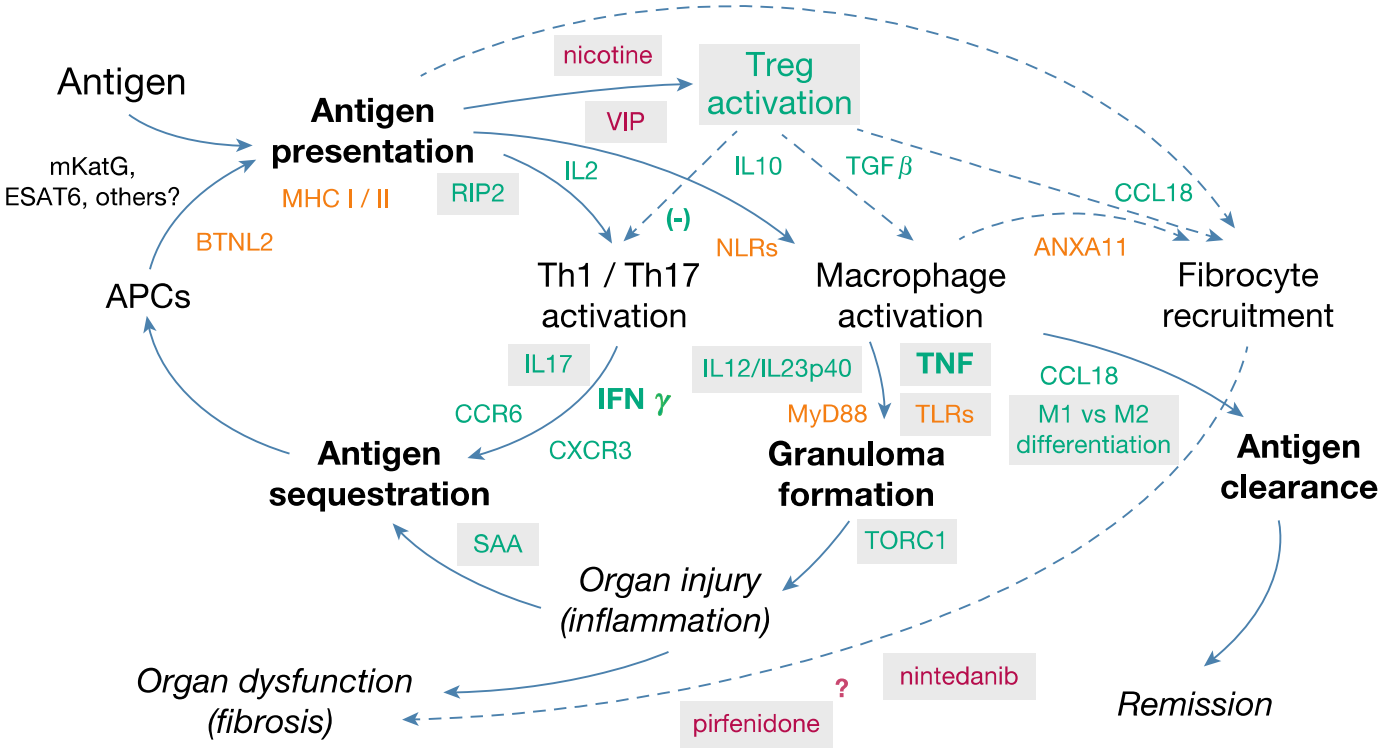
- Nicotine joue un rôle dans la régulation de l'inflammation médiée par les cellules T via l'activation du récepteur Alpha 7 nicotinique.
- Petite étude de 13 patients consentants, randomisés traités par Nicotine et tt conventionnel ou tt conventionnel seul



- Nicotine est bien tolérée.
- Pas de bénéfice clinique
- Augmentation et restauration des Tregs responsable des pathologies autoimmunes avec retour à un phénotype immun identiques à celui des patients asymptomatiques.
- Problème éthique de la prescription de nicotine

Julian, Chest 2013

Pathogénie de la sarcoidose



Patterson, Chest 2018

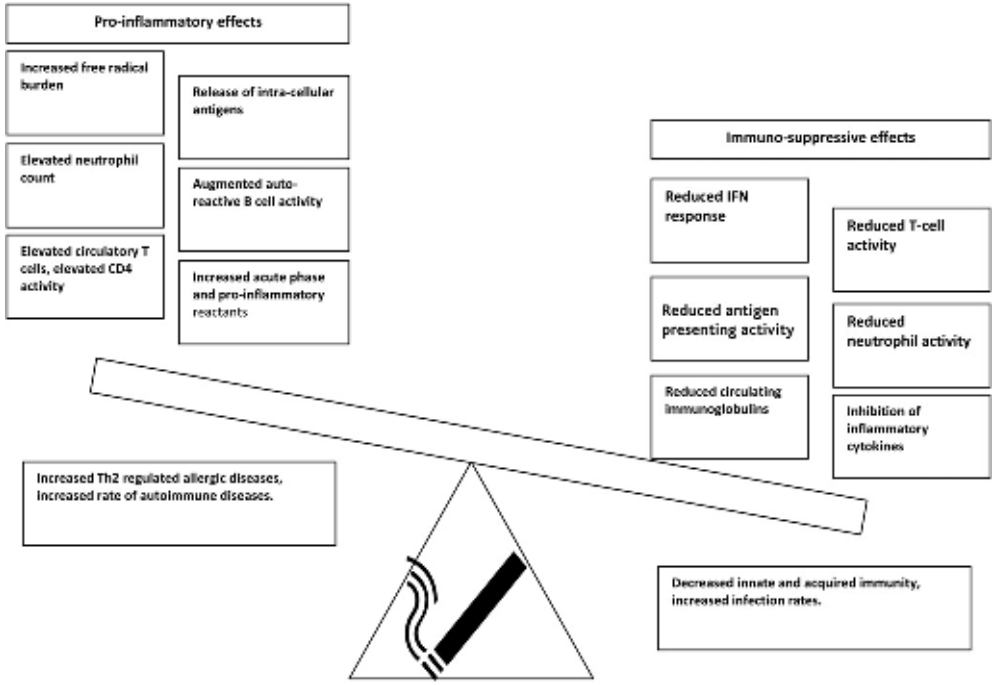
Sarcoïdose et nicotine

- Nicotine
  - Pour certains: augmente les cytokines proinflammatoires
  - Pour d'autres: effet protecteur et antinflammatoire en se liant à la sous-unité alpha7 du récepteur acétylcholine
  - Etudes lupus, vascularites,…

Nicotine and autoimmunity: The lotus'flower in tobacco
Gomes, Pharmacological Research, 2018

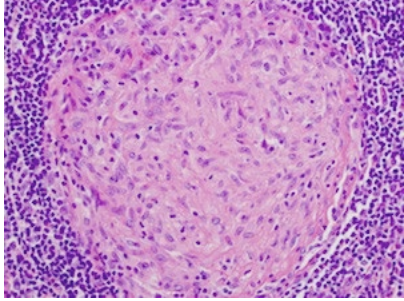
Effet du tabac sur l'immunité, l'inflammation et l'autoimmunité

- Expériences sur des rats: phase particulaire de la fumée qui est immunosuppresive



Arnson, J of Autoimmunity, 2010

En résumé



- L’agent causal de la sarcoïdose reste insaisissable
- Facteurs génétiques
- Facteurs environnementaux
- Rôle fondamental des lymphocytes T CD4, des lymphocytes B et des cellules de l’immunité

Kaiser, ERJ 2019

Conclusion

- La plupart des études montrent une incidence moindre de la sarcoidose chez les fumeurs comme pour les pneumonies d'hypersensibilité ou certaines formes de beryllioses
- De nombreuses explications sont plausibles et le mécanisme n'est pas connu
- Quel est l'agent responsable? Nicotine? Un composant de la fumée de cigarette?

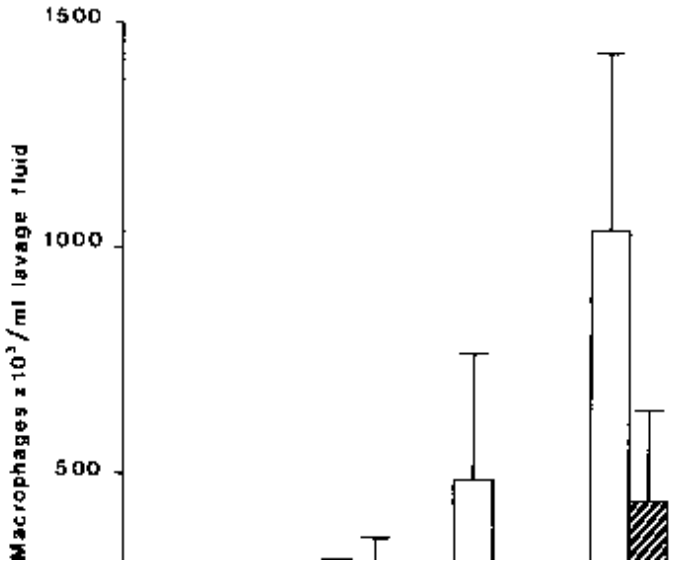
« The truth is rarely pure et never simple »

Oscar Wilde cité par Kaiser ERJ 2019

- **Le tabac est un facteur de risque majeur pour les maladies respiratoires, en particulier la BPCO et le cancer du poumon**

Sarcoïdose et tabac:
Hypothèses d'une relation

(table 2; $p < 0{\cdot}001$ for control *v* sarcoidosis group).



subdivided according to cigarette consumption the number of macrophages recovered increased progressively as a function of cigarette consumption (fig 1). In contrast, only those patients with sarcoidosis who smoked over 20 cigarettes a day had more alveolar macrophages than non-smokers with sarcoidosis, and the total number of macrophages recovered from these patients with a heavy smoking history was significantly less than that of controls with a similar smoking history (fig 1; $p < 0{\cdot}001$).

The numbers of lymphocytes recovered/ml lavage fluid in the patients with sarcoidosis was not significantly different in non-smokers and smokers (table 2), nor did smoking modify the total number of $CD4^+$ T lymphocytes recovered by lavage (table 3). Smoking, however, was associated with a twofold

Valeyre, Thorax 1988;43:516-24

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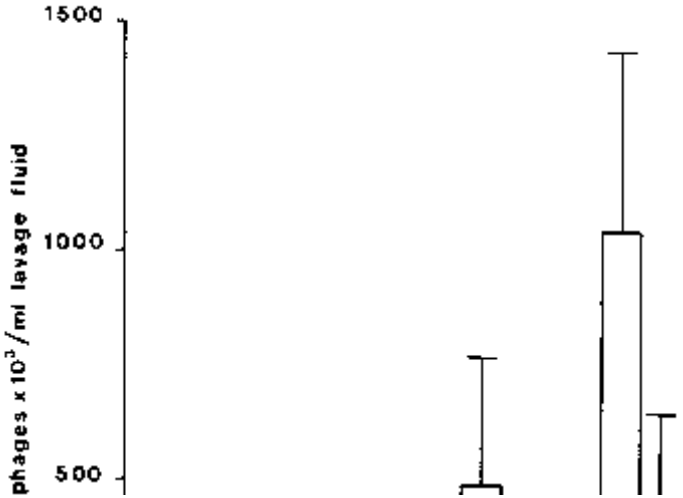
recovered by lavage from smoking patients than from non-smoking patients. Among smokers the number of macrophages obtained from patients with sarcoidosis was less than that recovered from control subjects (table 2; $p < 0{\cdot}001$ for control *v* sarcoidosis group).



The reduced number of alveolar macrophages recovered from sarcoid smokers compared with control smokers was not entirely explained by differences in tobacco consumption. When control subjects were subdivided according to cigarette consumption the number of macrophages recovered increased progressively as a function of cigarette consumption (fig 1). In contrast, only those patients with sarcoidosis who smoked over 20 cigarettes a day had more alveolar macrophages than non-smokers with sarcoidosis, and the total number of macrophages recovered from these patients with a heavy smoking history was significantly less than that of controls with a similar smoking history (fig 1; $p < 0{\cdot}001$).

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Thorax 1988;43:516-24

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"activity." Cigarette smoking was associated with a significant increase in the serum angiotensin converting enzyme activity (SACE), and patients with very high SACE and pulmonary gallium-67 uptake were smokers. Furthermore, more $CD8^+$ (but not $CD4^+$) lymphocytes were recovered by lavage from smoking than from non-smoking patients, giving a lower CD4 :CD8 ratio in smokers. Fewer alveolar macrophages were recovered by lavage from smokers with sarcoidosis than from normal subjects with a similar smoking history. These findings support the possibility that smokers, particularly those with a prominent accumulation of alveolar macrophages in the lower respiratory tract, may be less likely to develop sarcoidosis.

Introduction

Pulmonary sarcoidosis is a granulomatous lung

alterations in the number, type, and functional activity of lung immune and inflammatory cells.[3 4] We and others have suggested that the incidence of sarcoidosis

. Etude de 133 patients atteints de sarcoïdose suivis 1 an

. 30% vs 46% de fumeurs sarcoïdose vs contrôle

. 2 hypothèses: fumer diminue le risque de sarcoïdose ou fumer modifie l'évolution de la maladie qui est moins sévère

. Pas d'argument pour corrélation tabac-sévérité de la maladie

. Critères d'activité + élevés chez les fumeurs (SACE, scinti au gallium*, rapport CD4/CD8 (plus bas chez les fumeurs), Moins de macrophages au LBA

Hypothèse: les macrophages alvéolaires plus nombreux chez le fumeur protégeraient de la sarcoïdose

Thorax 1988;43:516-24

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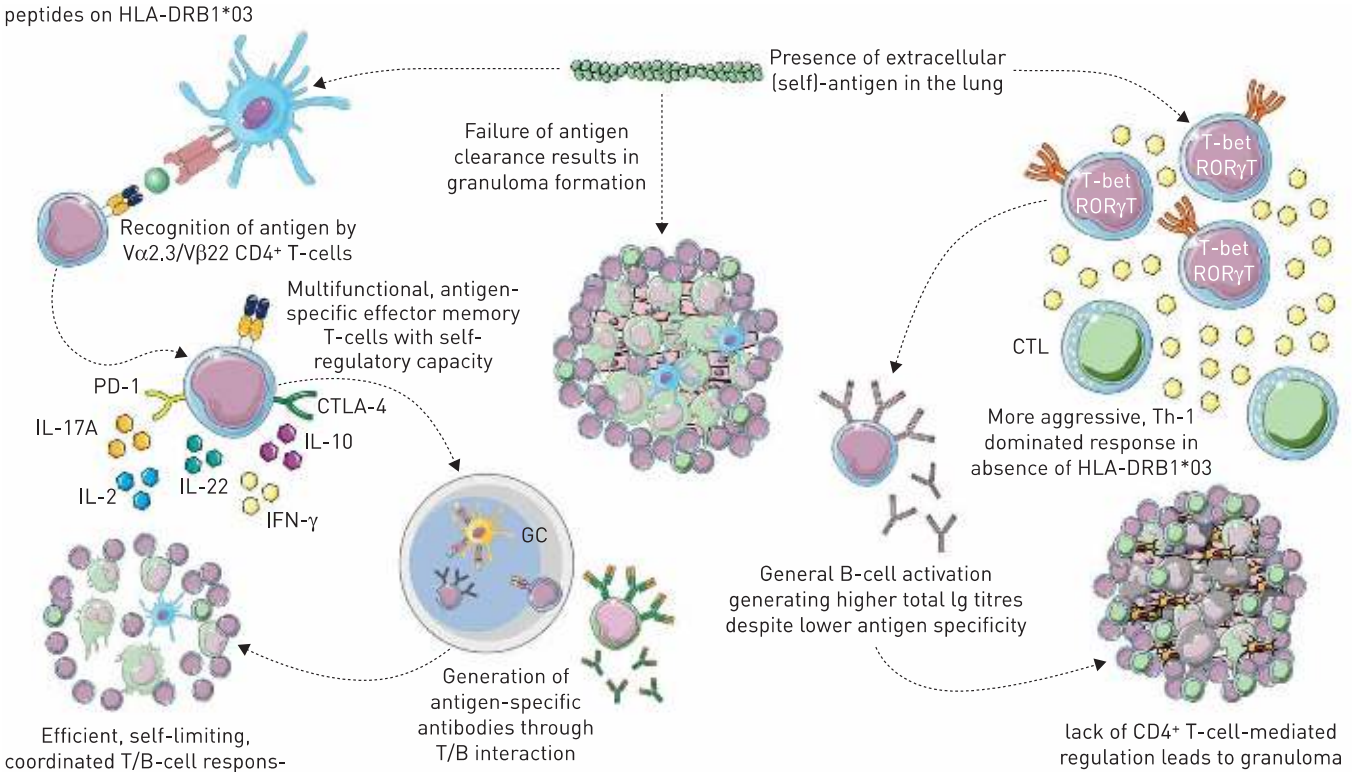
Sarcoïdose

- Incidence variable selon les régions, selon les populations, existence de cas familiaux:

  ->Facteurs génétiques

- Facteurs environnementaux: bactéries, virus ou Poussières: Industries du métal (beryllium), WTC ou agricoles (cultures de pdt, céréales ou prairies artificielles)



Kaiser, ERJ 2019

Sarcoïdose,

caméléon des maladies systémiques

- Atteintes cliniques:
- . Poumon
- Yeux
- Rein
- Cœur
- Système nerveux: ex: paralysie faciale

- Signes cliniques:
- Généraux: fatigue, perte de poids, adénopathies, arthralgies, érythème noueux
- Spécifiques d'un organe atteint

2010

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