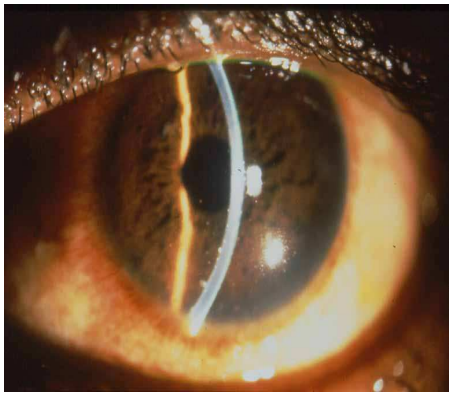
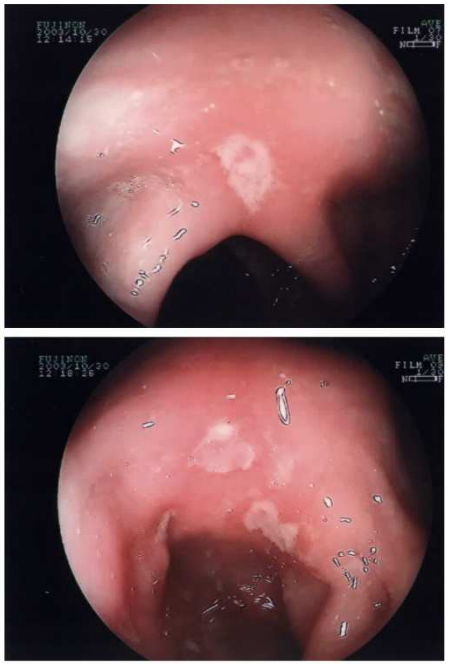
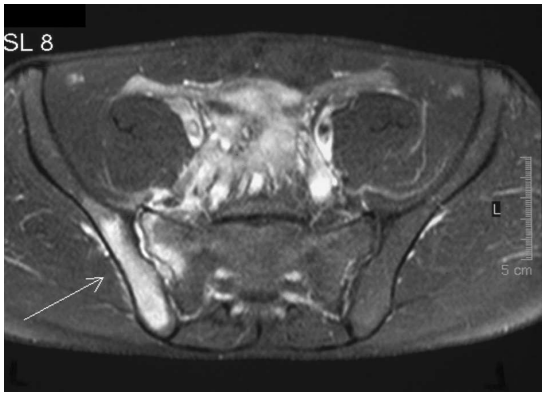
Les arthrites réactionnelles







Pr A Saraux
Chu Brest, France

Cas clinique

- Homme 60 ans
- Consulte pour fièvre brutale avec arthrite des deux chevilles et du genou droit et lombalgie inflammatoire 15 jours après un épisode de diarrhée traitée par antibiotique
- Antécédent
  - cancer de vessie in situ suivi depuis 2 ans
  - Colopathie
  - Syndrome inflammatoire modéré et asthénie depuis 1 an
- Quel diagnostic évoquez-vous?

Les arthrites réactionnelles

- Arthrite réactionnelle des spondyloarthrite
- Mais aussi
  - Rhumatisme streptococcique
  - Rhumatisme de Poncet et BCGthérapie
  - Rhumatisme des *clostridium difficile*
  - La maladie de Whipple
  - rare:
    - *Cryptosporidia, Lambliase, Amibiase, Strongyloïdose*
    - *Propionibacterium acnes?*
    - *Borrelia Burgdorferi?*
    - *Capnocytophaga canimorsus?*

Plan

- Historique
- Le concept
- L’arthrite réactionnelle des spondyloarthrites
  - Fréquences
  - Capacité des critères à les intégrer dans les SpA
  - Physiopathologie
  - Traitement
- Les autres

Arthrite réactionnelle des spondyloarthrite

- Jusqu'au milieu du XXe siècle: rhumatismes inflammatoires regroupés au sein des arthrites rhumatoïdes.

- La mise en évidence du facteur rhumatoïde a été à l'origine de la distinction progressive de plusieurs rhumatismes inflammatoires.

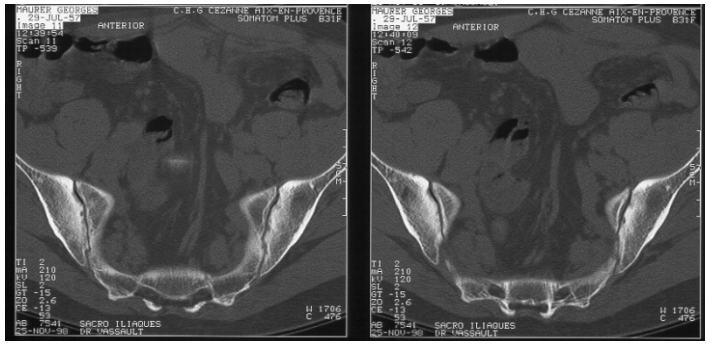
- 1970: concept de « seronegative spondarthritides » consolidé par la mise en évidence de l'association de ces différentes entités cliniques avec la présence de l'antigène HLA B27, dont les manifestations cliniques combinent de façon variable un syndrome pelvi-rachidien (ou axial), un syndrome enthésopathique périphérique, un syndrome articulaire périphérique et un syndrome extra-articulaire (uvéite, psoriasis, balanite, urétrite, diarrhée).

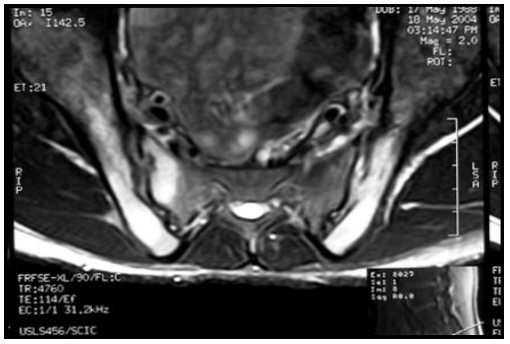
- Spondyloarthrites aussi fréquentes que la polyarthrite rhumatoïde dans la population générale 0,30 % [IC 95 % : 0.17 – 0.46], identique dans les deux sexes, les formes masculines étant souvent plus bruyantes cliniquement

- Cinq entités sont définies : spondylarthrite ankylosante, rhumatisme psoriasique, arthrites réactionnelles, arthrites associées aux maladies inflammatoires chroniques de l'intestin et spondylarthrites indifférenciées.

Le concept

Avant	Maintenant
Le groupe de maladie Les Spondylarthropathies	Les Spondyloarthrites
Les sous-groupes de maladie Spondylarthrite ankylosante arthrite réactionnelle des entérocolopathies rhumatisme posriasique indifférencié Sapho	axiales avec sacroiliite (radiographique, magnétique) sans sacroiliite périphériques





Critères ASAS pour les SPA axiales

(chez les patients avec lombalgie ≥ 3 mois et âge au début < 45 ans)

≥ 1 signe de SPA*

+ sacro-iliite**

ou

HLA-B27+

≥ 2 signes de SPA*

*Signes de spondylarthropathies

- Rachialgie inflammatoire
- Enthésite
- Uvéite
- Dactylite
- Psoriasis
- Maladie de Crohn
- Bonne réponse aux AINS
- Histoire familiale de SPA
- HLA-B27
- CRP élevée

**Inflammation hautement compatible avec une sacro-iliite à l'IRM ou une sacro-iliite radiographique définie suivant les critères de New York modifiés

N = 649 lombalgiques

Sensibilité 82,9%
Spécificité 84,5%

EULAR 2009 - D'après une communication de M. Rudwaleit. Ann Rheum Dis 2009;68(Suppl3):127. OP-0168.
Rudwaleit M et al. Ann Rheum Dis 2009;68:770-6; Rudwaleit M et al. Ann Rheum Dis 2009;68:777-83.

Critères ASAS pour les SPA périphériques (chez les patients avec arthrite ou enthésite ou dactylite)

≥ 1 signe de SPA*

ou

≥ 2 signes complémentaires**

*Signes de spondylarthropathies
- Psoriasis
- Maladie inflammatoire du côlon ou de l'intestin
- Infection récente
- HLA-B27
- Uvéite
- Sacroiliite radiographique ou IRM
- N = 992 patients

**Signes complémentaires
- Arthrites
- Enthésite
- Dactylite
- Maladie inflammatoire du côlon et de l'intestin
- Antécédents familiaux de SPA

Sensibilité 78%
Spécificité 83,7%

EULAR 2009 - D'après une communication de M. Rudwaleit. Ann Rheum Dis 2009;68(Suppl3):127. OP-0168.

Proportion d'arthites reactionnelles au sein des SpA axiales

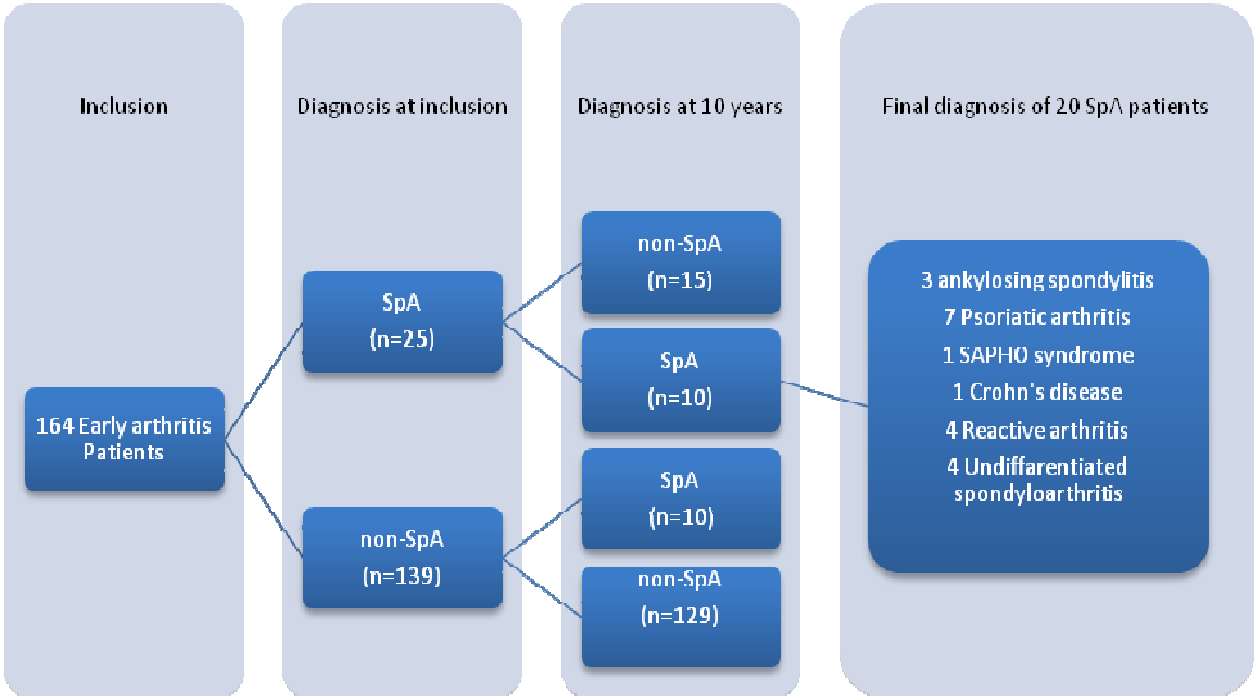
Critères d'Amor

	Critère	N	%
1.	Douleurs rachidiennes – 1 pt	705	100
2.	Oligoarthrite asymétrique ou Arthrite prédominant aux membres inférieurs – 2 pts	127	18
3.	Douleurs fessières sans précision sans douleurs fessières à bascule – 1 pt	178	25,3
4.	Douleurs fessières à bascule – 2 pts	370	52,5
5.	Doigt ou orteil en saucisse – 2 pts	91	12,9
6.	Talalgie ou toute autre enthésiopathie – 2 pts	353	50,1
7.	Iritis - 1 pt	49	7
8.	Urétrite non gonococcique ou cervicite ou diarrhée aiguë datant de moins d'un mois avant le début de l'arthrite – 1 pt	14	2
9.	Psoriasis cutané ou balanite ou Entérocolopathie chronique – 2 pts	146	20,7
10.	Histoire familiale de spondyloarthrite ou d'uvéite ou d'entéro-colopathie ou HLA B 27 + - 2 pts	458	65
11.	Amélioration en 48 h des douleurs par les AINS et/ou rechute rapide (48 h) des douleurs à leur arrêt – 2 pts	558	79,2
12.	Sacroiliite radiologique présente – 3 pts	187	26,5
	Score ≥ 6 78,6		554

Evolution de 164 artrhites débutantes après 10 ans

Patients with swelling of at least one joint were included in a cohort of early arthritis. Standardized data were collected at inclusion [8]. The diagnosis of SpA at inclusion was based on all clinical, biological, and radiological data. Ten years after, a questionnaire was sent to the physician of each patient to collect the final diagnosis. X-ray of the pelvis performed at inclusion was blindly reviewed. The statistical tests used were chi-square test and Cohen's kappa coefficient.

SpA is a relatively rare and highly difficult to establish diagnosis at the onset of a peripheral inflammatory rheumatic disease. Few peripheral arthritis progress to an axial spondylarthropathy. Diagnostic tests have low predictive value, especially PPV.



Dewi Guellec

Letters to the editor / Joint Bone Spine 80 (2013) 341–347

Brief report

Evolution over thirty years of the profile of inpatients with reactive arthritis in a tertiary rheumatology unit

Anne Brinster, Xavier Guillot, Clément Prati, Daniel Wendling*

Reactive arthritis (ReA) is sterile arthritis occurring after extra articular bacterial infection. The aim of this study was to analyze, over 30 years, clinical, biological and imaging characteristics as well as therapeutic management of new cases of ReA, comparing two periods.

Methods: retrospective monocentric study, data of all the patients followed in our unit between January 1st 1984 and April 2014 with the diagnosis or ReA were analyzed (clinical and biological features, management and outcome), and compared between two periods: from January 1984 to December 1993, and from January 2004 to December 2013.

Results: Sixty two patients fulfilling international diagnosis criteria were analyzed. There was no significant difference between the two periods in number of new cases, clinical presentation, biological data or outcome. Changes in therapeutic management were obvious with occurrence of anti TNF in the recent period.

Conclusion: Reactive arthritis is still a current rheumatologic problem in a developed country, with a need of early and tailored rheumatologic management.

Brief report

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Anne Brinster, Xavier Guillot, Clément Prati, Daniel Wendling*

Table 1
Distribution of infectious agents.

Infectious agent	Number of patients positive	Serology	PCR	Culture
Chlamydia trachomatis	18	14	2	
Ureaplasma urealyticum	5	0	5	
N. gonorrhoea	4	3	1	
Mycoplasma pneumoniae	2	2	0	
Chlamydia + Yersina	1	1		
Chlamydia + Strepto B	1		1	
Chlamydia + N. gonorrhoea	1	X	X	
Salmonella	1			1
Neisseria meningitidis	1			1
Anguillulosis	1	1		
Streptococcus B	1		1	
Yersina	1	1		
S. Aureus + Strepto B	1		1	
Not found	22			

PCR: polymerase chain reaction.

Brief report

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Table 2
"Global" comparison of the 2 cohorts.

	1984–1993	2004–2013	*p*
Number of patients included	15	31	0.4
Number of patients hospitalized	7438	11 823	
Men	13	28	0.6
Median age at diagnosis	37	30	0.9
HLA B27+ (%)	91	63	01
Delay between infection/articular symptoms (days) median	5.5	9	0.6
mean	14.2	14	
Patients with fever (%)	20	19	1
Leucocytes (giga/l)	9.8	10.6	0.4
CRP (mean) (mg/l)	87.4	90.1	0.9
Evidence of infectious agent (%)	53	61	0.2
Antibiotic treatment (%)	77	93	0.3
Treatement by azithromycin (%)	0	47	0.006
TJC/SJC	2.8/1.8	3.2/2	ns
Dactylitis (%)	13	29	0.3
Enthésitis (%)	40	26	0.5
Extra articular features (%)	47	35	0.4
Axial symptoms (%)	33	29	1
DMARDs use (%)	36	62	0.1
Median delay of DMARD introduction (days)	210	50.5	ns
Biologic agents use (%)	0	45	0.005
Remission at last follow-up (%)	57	47	0.6

(ns = non significant) TJC: tender joint count; SJC: swollen joint count, DMARDs: disease modifying anti rheumatic drugs.

Brief report

Evolution over thirty years of the profile of inpatients with reactive arthritis in a tertiary rheumatology unit

Anne Brinster, Xavier Guillot, Clément Prati, Daniel Wendling*

Table 3
Distribution of infective agents found in cases of reactive arthritis between the two periods.

Infectious agent (%)	1984–1993 ($N = 15$)	2004–2013 ($N = 31$)	p
Chlamydia	33	31	1
N. gonorrhoea	7	7	1
Mycoplasma	0	7	0.54
Ureaplasma	0	7	0.54
Salmonella	0	3	1
Anguillulosis	0	3	1
Yersinia	7	3	1
Not found	53	61	ns

Clin Rheumatol (2016) 35:2639–2648
DOI 10.1007/s10067-016-3364-0

REVIEW ARTICLE

The incidence of sexually acquired reactive arthritis: a systematic literature review

Hayley J. Denison[1] · Elizabeth M. Curtis[2] · Michael A Clynes[2] · Collette Bromhead[3] · Elaine M. Dennison[1,2] · Rebecca Grainger[4]

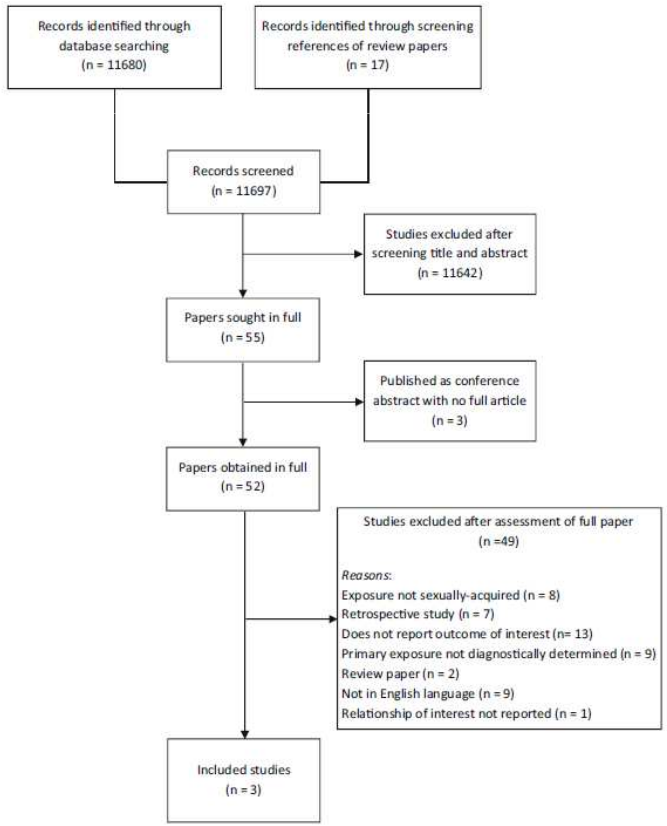


Fig. 2 Flow diagram depicting the identification and selection of relevant papers

Clin Rheumatol (2016) 35:2639–2648
DOI 10.1007/s10067-016-3364-0

REVIEW ARTICLE

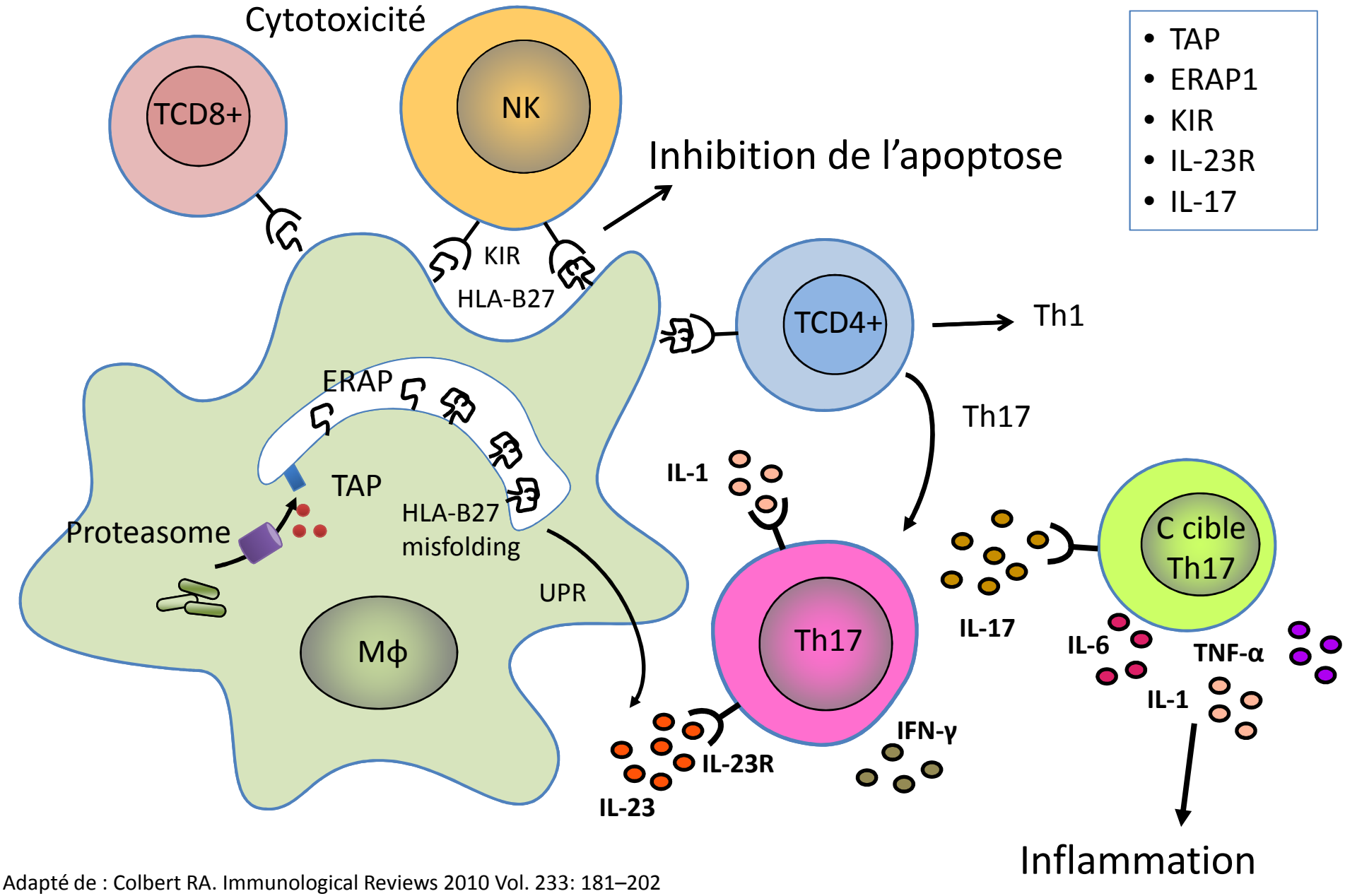
The incidence of sexually acquired reactive arthritis: a systematic literature review

Hayley J. Denison[1] · Elizabeth M. Curtis[2] · Michael A Clynes[2] · Collette Bromhead[3] · Elaine M. Dennison[1,2] · Rebecca Grainger[4]

Table 1 Characteristics of included studies

First author	Year of publication	Study location	Type of study	Study size	Study population	Primary exposure(s) (infection)	Infection diagnosis	Incidence of ReA	Proportion of ReA cases with asymptomatic infection
Carter [31]	2013	FL, USA	Prospective	149 (from 365 enrolled)	Adults attending communicable disease clinic testing positive for *Chlamydia trachomatis*	*Chlamydia trachomatis*	Laboratory diagnosis by gram stain, cell culture or NAAT	12/149 (8.1 %)	8/12 (66.7 %)
Rich [30]	1996	AL, USA	Prospective	217	Adults attending a sexually transmitted disease clinic being treated with doxycycline for a possible or proven *Chlamydia trachomatis* infection	Genital infection/ inflammation	Laboratory diagnosis by cervical cell culture for *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. In men, a gram-stained urethral smear and a urethral *Neisseria gonorrhoeae* culture were obtained. *Chlamydia trachomatis* genital cultures were obtained for every patient who had objective ReA features	9/217 (4.1 %)	7/9 (77.8 %)
Keat [29]	1978	UK	Likely prospective	531	Heterosexual men attending sexually transmitted disease clinic with new episodes of urethritis	Non-specific urethritis. Cultures for *Chlamydia trachomatis* taken to investigate association with ReA	Urethral smear. Non-specific urethritis if over 10 polymorphs found in ≥3 consecutive high-power fields (magnification ×600), if microscopy, culture and serology excluded gonorrhoea and syphilis	16/531 (3.0 %)	No details

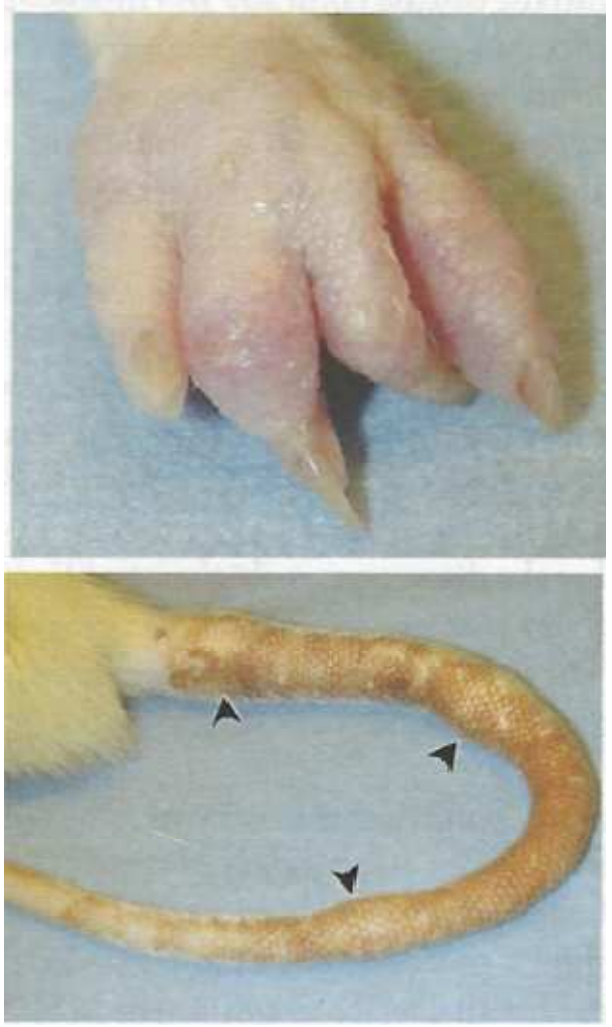
Une part génétique



Adapté de : Colbert RA. Immunological Reviews 2010 Vol. 233: 181–202

Transférable au rats transgéniques B27

- Rats Lewis, lignées 21-4H ou 33-3 :
  - Maladie inflammatoire spontanée :
    - Arthrites
    - Colite
    - Epididymite, balanite
    - Uvéite
    - Hyperkératose cutanée
  - Seuls ceux ayant incorporé un nombre important de copies des transgènes B27/b2m (Taurog 1993)
  - Fond génétique protecteur : toutes les lignées transfectées ne développent pas la maladie



Hammer RE. Cell. 1990 Nov 30;63(5):1099-112

Selon la flore intestinale

- Les rats transgéniques germ-free:
  - ne développent pas la maladie
  - sauf dermatite et épididymite
- Ajout de bactéries dans l'alimentation :
  - Restauration de l'ensemble du phénotype pathologique
  - Une flore commensale suffit…
- réintroduction d'une flore sélectionnée:
  - Seuls certains cocktails bactériens aboutissent à l'induction d'une colite
  - Rôle des *Bacteroides*

Taurog JD. J Exp Med. 1994 Dec 1;180(6):2359-64
Rath HC. J Clin Invest. 1996 Aug 15;98(4):945-53

Rôle des lymphocytes CD4+

- lymphocyte CD4+ et CD8+ restreints à HLA-B27
- rats transgéniques dépourvus de lymphocytes CD8+ développent la maladie
- Les rats transgéniques athymiques ne développent ni colite ni arthrites
- Le transfert de lymphocytes CD4+ restore le phénotype colite et arthrite

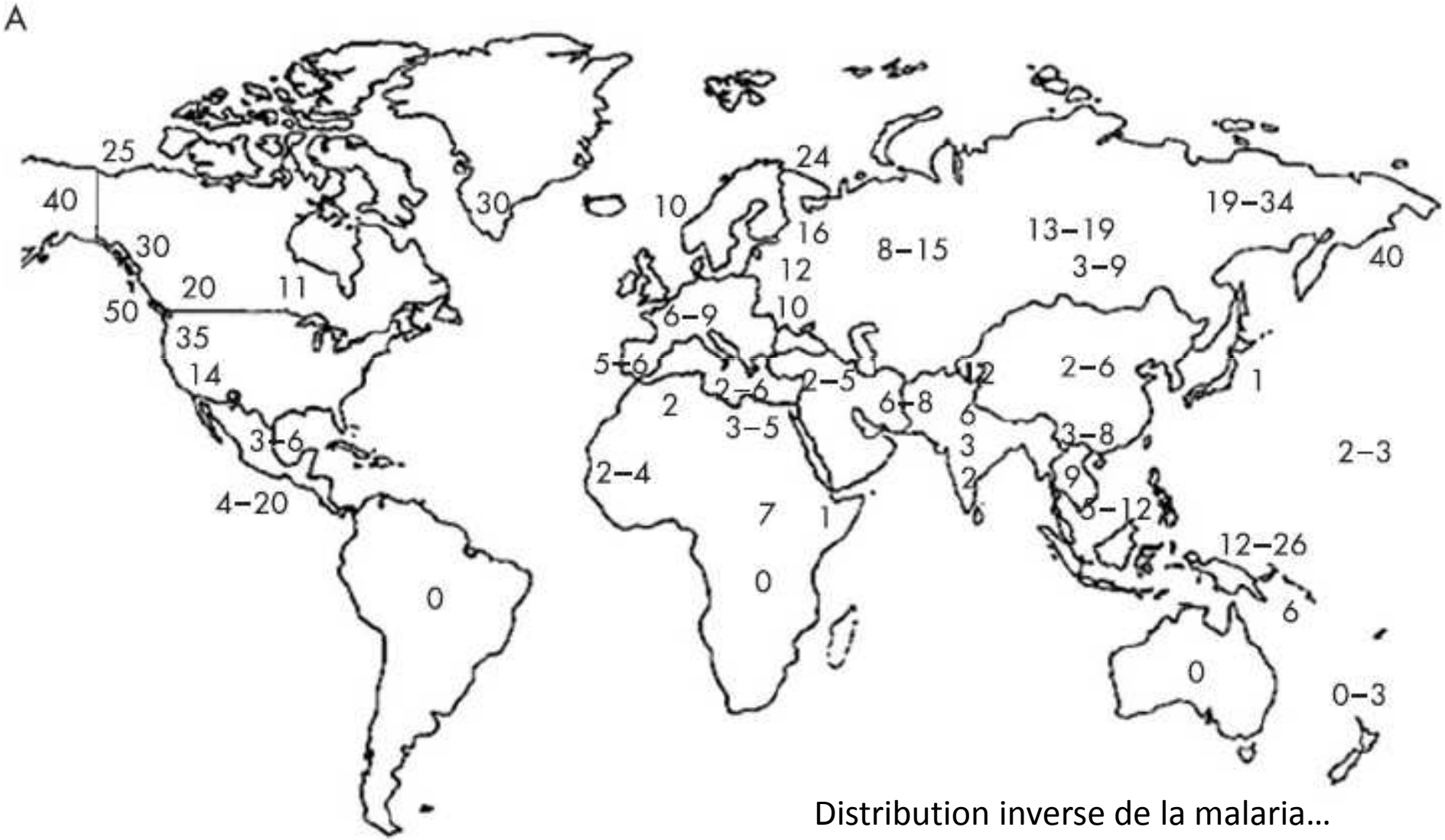
Roddis M. J Immunol. 2004 Jan. 1;172(1):155–161 Colbert RA. Immunol Rev. 2010;233(1):181–202
Taurog JD. Arthritis Rheum 2009;60:1977–1984.18 Hoentjen F. Inflamm Bowel Dis 2007;13:317–324

Mais B27 favorable à la réponse anti-virale

- B*27:05 = allèle protecteur d'infections virales :
  - Influenza A
  - VIH
  - Hépatite C
- Réponse cytotoxique CD8+ particulièrement rapide et efficace
- Diminution de la charge virale, et persistance de taux de CD4+ normaux au cours de l'infection par le VIH
- La réponse CD8+ cytotoxique reste parfaitement fonctionnelle chez les patients SA

de Groot NG. PNAS 2010; 24,107(34):15175-80
Ammaranond P. J Immunol. 2011; 1, 186(1):479-88

HLA-B27 dans le monde

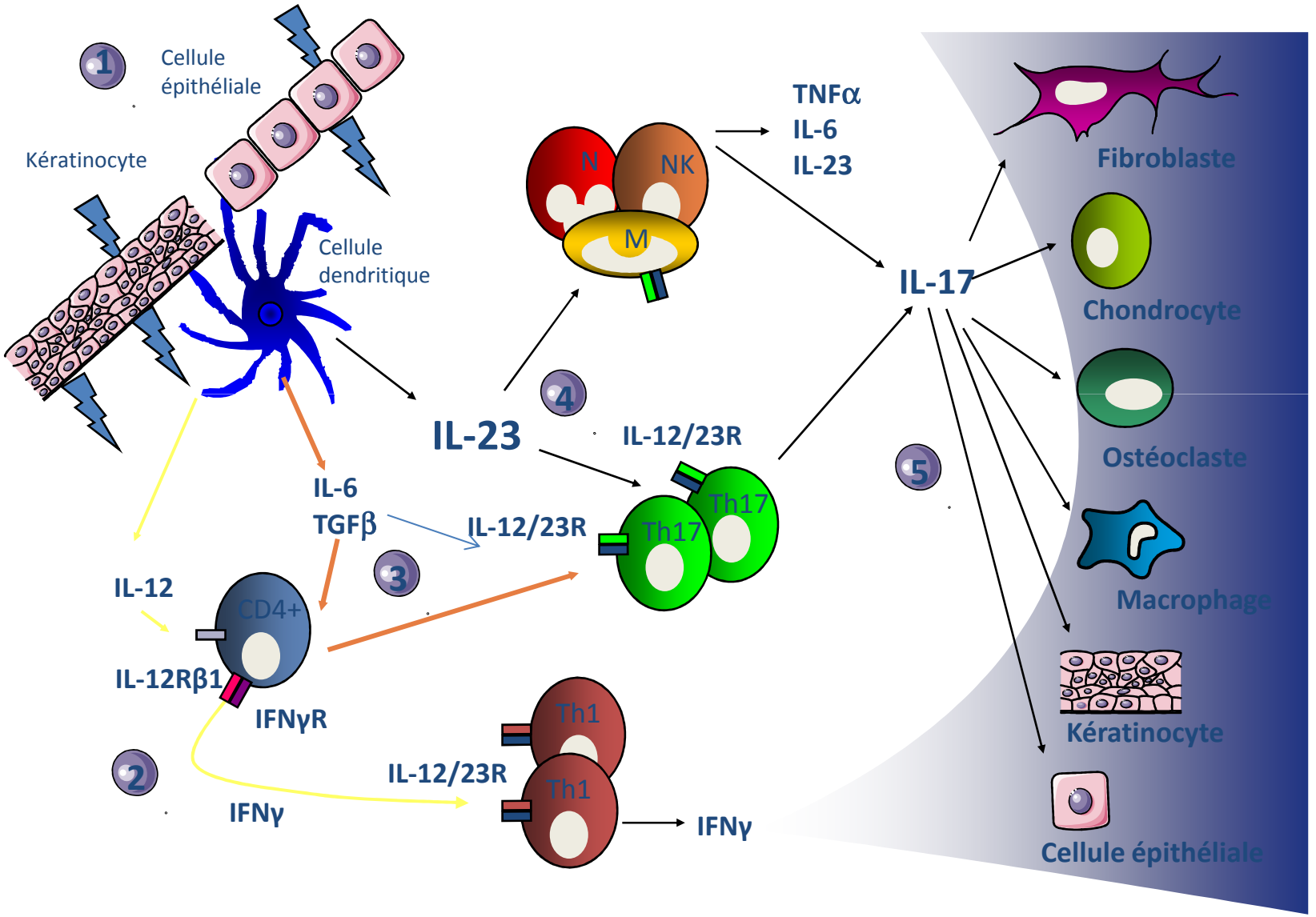


Source: Khan MA, ref. 1

Distribution inverse de la malaria…

Mathieu A. Autoimmunity Reviews 7 (2008) 398–403

Une réponse cytokinique

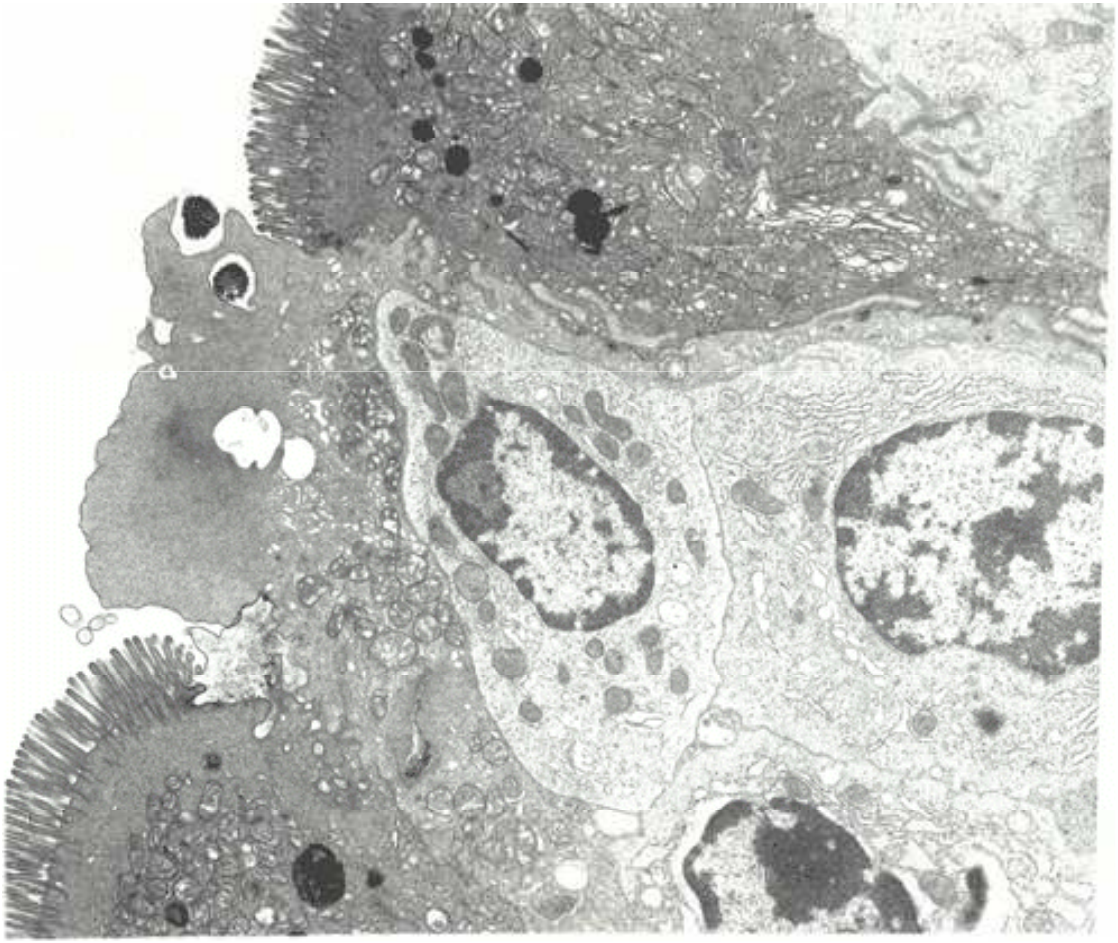


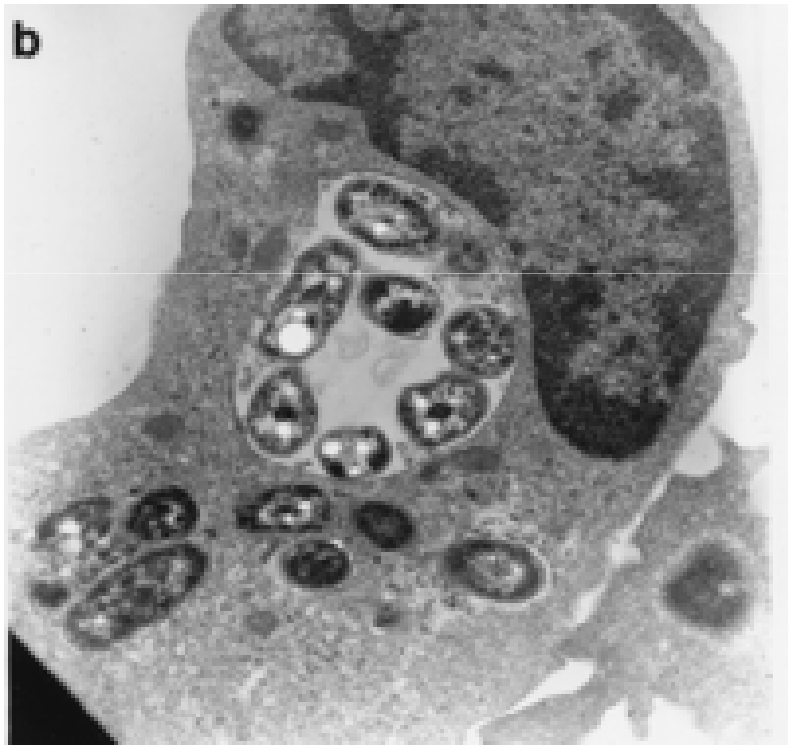
Détection d'éléments bactériens dans la synoviale au cours de l'arthrite réactionnelle

	Antigènes	ADN	ARNm	Culture
Chlamydia trachomatis	+	+	+	±
Chlamydia pneumoniae	+	+	+	-
Ureaplasma urealyticum	ND	+	ND	-
Yersinia enterocolitica	+	+	ND	-
Yersinia pseudotuberculosis	+	+	+	-
Shigella flexneri	+	?	ND	-
Shigella sonnei/enteridis	+	+	ND	-
Campylobacter jejuni	?	?	ND	-

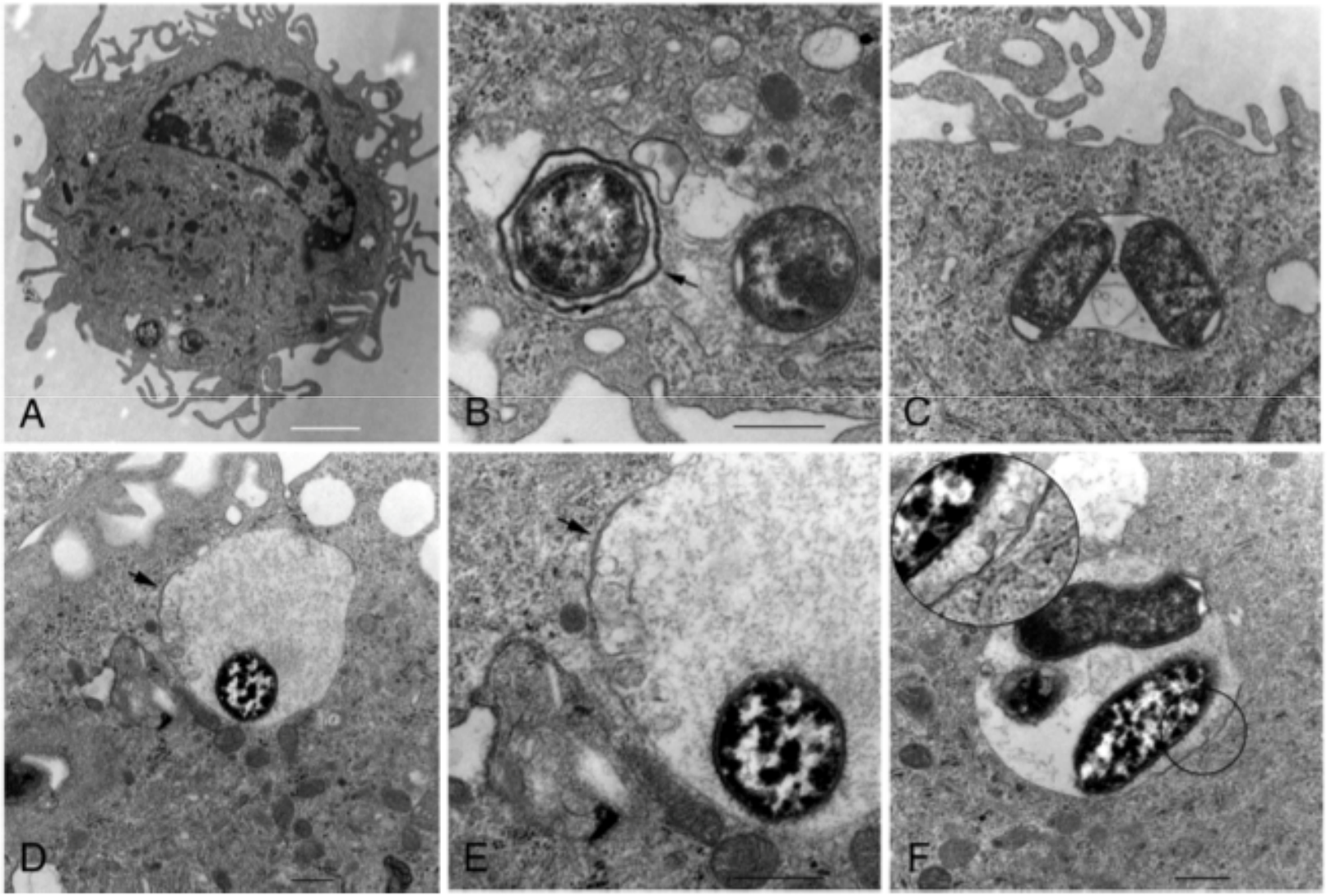
Demi-vie des ARNm = quelques minutes……

Salmonelles intra-cytoplasmiques



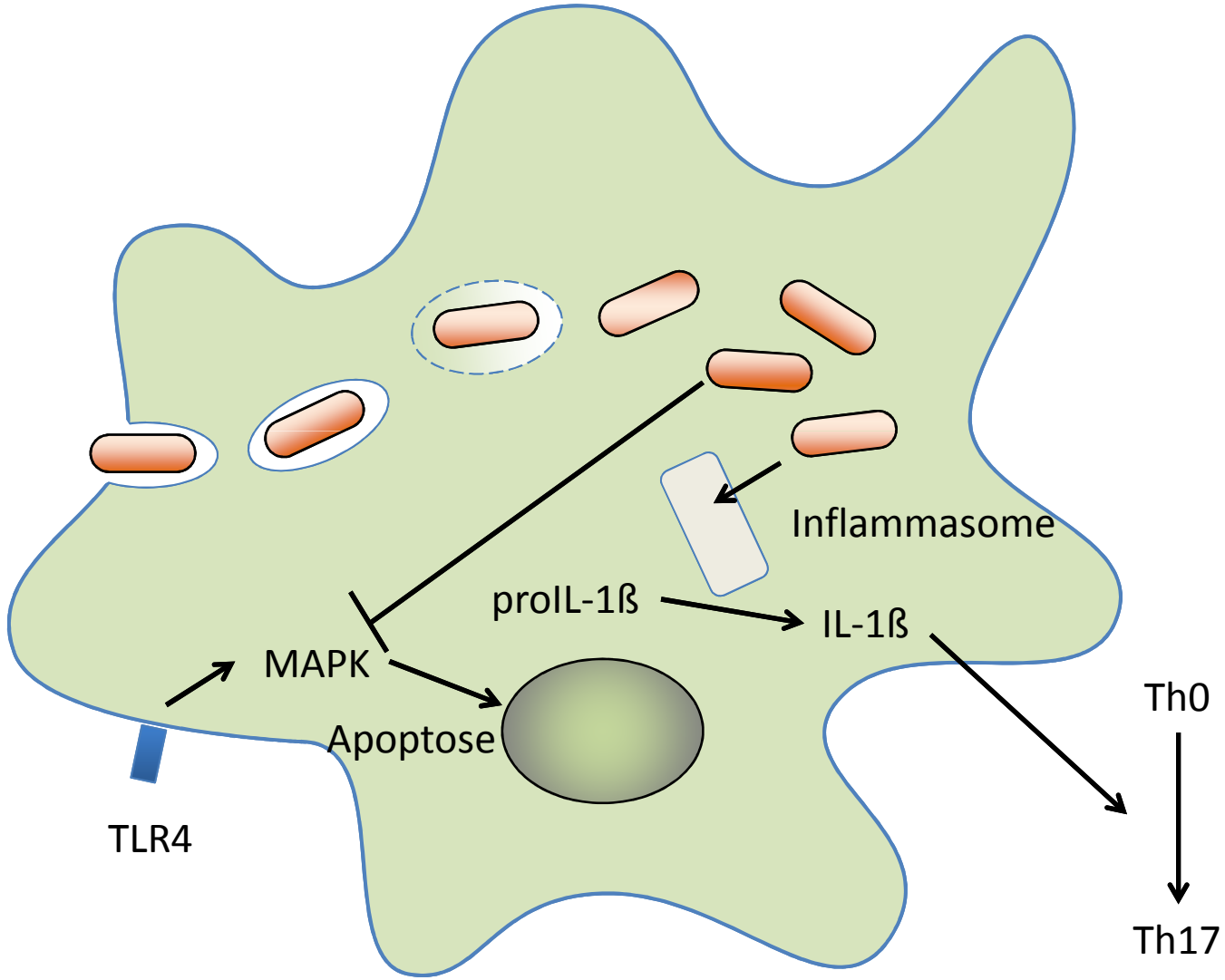


Yersinia



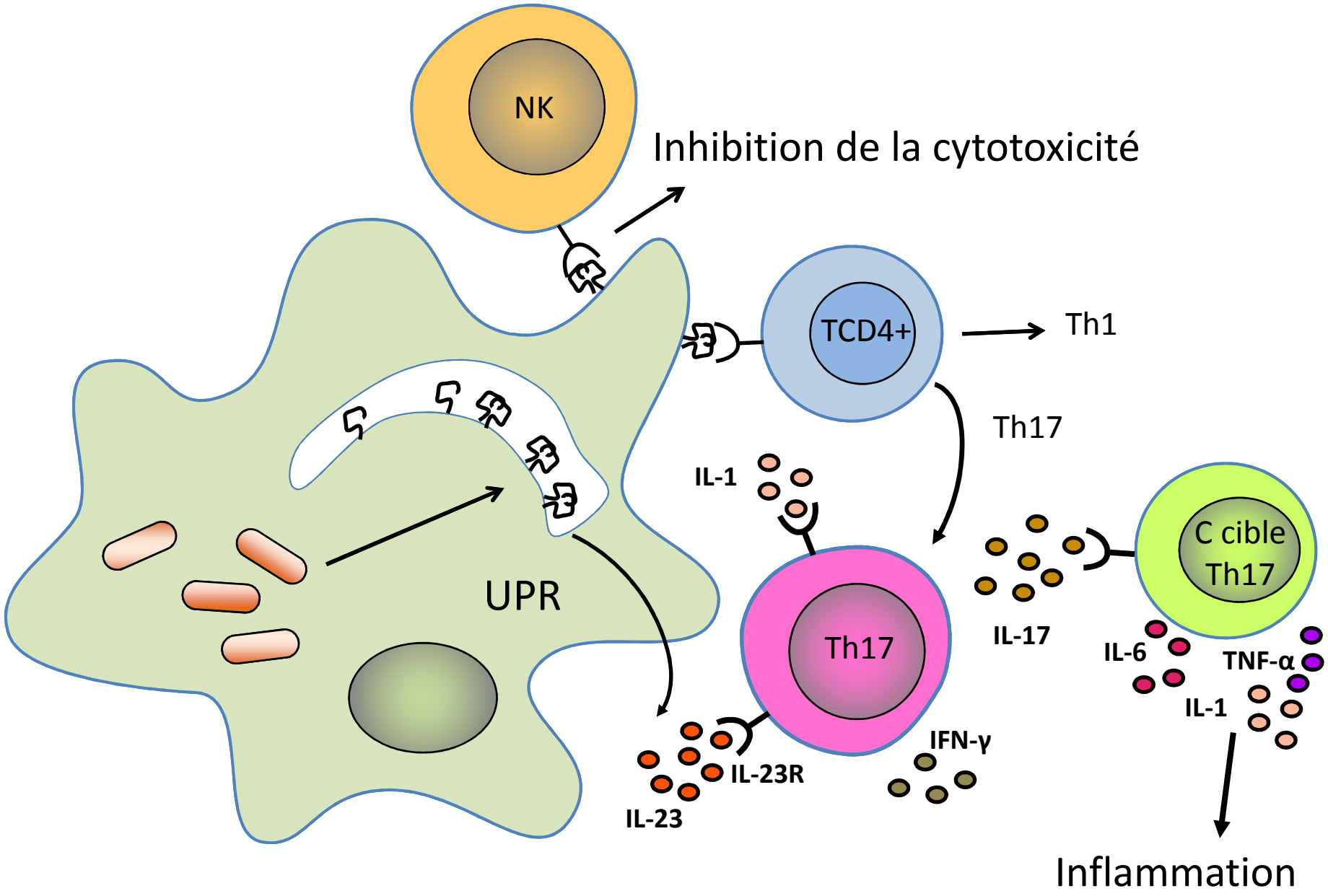
Pujol C. Infection and Immunity. 2009 Jun.;77(6):2251–2261.

Salmonelles, Shigelles, yersinia

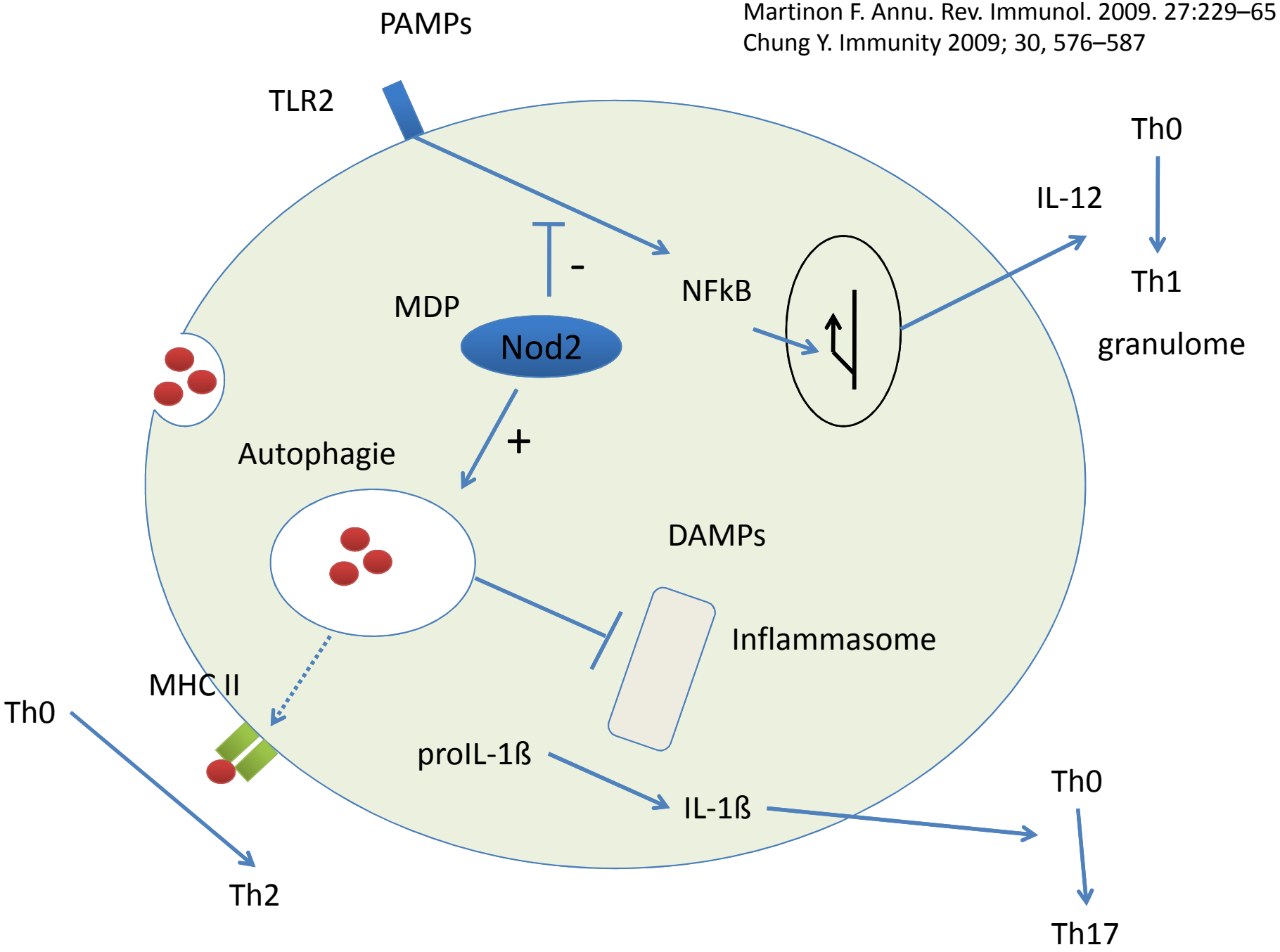


Tam MA. Immunol Rev. 2008;225(1):140–162.

Salmonelles et misfolding B27



Saitoh T. Nature 2008; 456,13: 264-9
Martinon F. Annu. Rev. Immunol. 2009. 27:229–65
Chung Y. Immunity 2009; 30, 576–587



AINS

- Pas d'études spécifiques, mais empiriquement, les AINS exercent un effet positif sur la douleur. ({Palazzi 2004 862} {Carlin 2001 783}.
- Le profil de tolérance des AINS dans les spondylarthrites semble similaire à celui noté au cours des autres affections rhumatologiques chroniques.
- On a toutefois signalé
  - possible aggravation d'un psoriasis, voire une induction d'un psoriasis *de novo* suite à l'administration d'AINS {Abensour 1988 ID}.
  - pourraient favoriser les poussées des maladies inflammatoires chroniques de l'intestin (MICI) telles que la maladie de Crohn et la rectocolite.

Les corticoïdes

- Pas d’étude propre.
- Préconisés par certains, par voie orale ou intra-articulaire {Toivanen 1995 820} dans les formes "résistantes" au traitement par AINS.
- Suivant les auteurs, la posologie préconisée varie de 15 à 40mg/j de prednisone per os ou équivalent {Palazzi 2004 862}.

Traitements de fond

- Les revues générales {Palazzi 2004 862}{Leirisalo-Repo 2005 3415}{Colmegna 2005 2801}{Petersel 2005 3192} ainsi que deux études en double aveugle randomisées comparatives au placebo {Clegg 1996 3568} {Egsmose 1997 3567} qui ont évalué 213 patients traités par sulfasalazine 2g /j ou placebo pendant 6 mois ne montrent pas de bénéfice de la sulfasalazine sur les paramètres évalués. Les sorties d'études sont nombreuses. La sulfasalazine ne semble pas modifier l'évolution naturelle de la maladie.

Antibiotiques

- Ciprofloxacine 3 mois à la posologie 1g/j en 2 prises dans 4 études et 1,5 g/j en 2 prises, dans 1 étude.
  - Quatre études ne montrent pas de différences entre les deux groupes,
  - Un essai {Yli-kerttula 2003 2823} montre en revanche une différence sur le nombre d'arthrites réactionnelles évoluant vers une forme chronique (2/26 sous ciprofloxacine versus 11/27 sous placebo (p = 0,006), mais ce travail manque de précision sur les critères d'inclusion, les données bactériologiques ou l'analyse en fonction de la zone infectée.

- Un essai {Kvien 2004 3452} randomisé, en double aveugle, a comparé l'azithromycine 1g /j pendant 12 semaines au placebo avec un suivi de 6 mois.
  - 152 patients inclus.
  - Aucune différence entre les deux groupes.

- Astraukle a réalisé une étude {astrauskle 2003 2979} ouverte sur 138 enfants de moins de 16 ans présentant des arthtrites suite à une infection. Dans 2/3 des cas ces infections étaient d'origine ORL. Les patients ont été traités soit par amoxicilline seule, soit par amoxicilline et acide clavulanique soit par placebo. La posologie d'amoxicilline était de 40mg/kg sans dépasser 2g/j. La durée du suivi était de 3 mois, la durée de l'antibiothérapie de 14 à 28 j. concernant le nombre de patients en rémissions les auteurs montrent une différence significative entre le groupe traitement et le groupe témoin :
  - Amoxicilline: 24/50 (48%) - placebo : 6/46 (13%) $p<0,01$
  - Amoxicilline et acide clavulanique: 24/50 (58,5%) - placebo : 6/46 (13%) $p<0,001$.
  - pas de différence entre les deux antibiotiques, ni sur la durée du traitement antibiotique (14 ou 28 jours).

- Dans sa revue de littérature {Palazzi 2004 862}, Palazzi conclut que le traitement antibiotique ne semble pas justifié une fois l'arthrite installée. En revanche, l'administration précoce de tétracycline ou d'érythromycine pourrait prévenir la survenue d'arthrites chez les patients.

Les biologiques

Curr Rheumatol Rep (2012) 14:390–394
DOI 10.1007/s11926-012-0280-4

SERONEGATIVE ARTHRITIS (MA KHAN, SECTION EDITOR)

Reactive Arthritis: Developments and Challenges in Diagnosis and Treatment

Davina Morris · Robert D. Inman

Abstract Reactive arthritis (ReA) has traditionally been described as a nonseptic arthritis occurring in the joint following an extra-articular bacterial infection. This concept became clinically associated with antecedent infections of either the gastrointestinal or genitourinary tract. Yet this operational definition of ReA has led to diagnostic uncertainty in different clinical settings. There are several scenarios in which the ReA has been complex. One is in the SAPHO syndrome, which shares many features with ReA. Another is the development of arthritis after infection with atypical organisms such as *Clostridium difficile* and *Giardia lamblia*. Treatment of ReA remains an area of ongoing investigation. There has been a randomized controlled trial of combination antibiotics in Chlamydia-induced ReA, which reported a positive result. There are several uncontrolled reports of anti-TNF agents being used successfully in refractory ReA. These studies in treatment modalities require validation on larger samples but do provide some encouraging preliminary findings from which to develop new therapeutic approaches.

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SERONEGATIVE ARTHRITIS (MA KHAN, SECTION EDITOR)

Reactive Arthritis: Developments and Challenges in Diagnosis and Treatment

Davina Morris · Robert D. Inman

Table 1 Use of anti-TNF agents in ReA

Author, Year	No. patients	Rx	Clinical infection	Microbial infection	Efficacy
Oili 2003 [20]	2	IFX	Diarrhea	Yersinia	Yes
Gaylis 2003 [21]	1	IFX	HIV	HIV	Yes
Flagg 2008 [18]	10	ETA	6 wk<ReA	6 wk<ReA	56 %
Gill 2008 [22]	1	IFX	Urethritis	None	Yes
Abdelmoula 2008 [23]	1	IFX	None	Chlamydia	Yes
Schafranski 2010 [24]	1	IFX	Urethritis	Chlamydia	Yes
Meyer 2011 [19•]	10	IFX, ETA, ADA	4 wk<ReA	4 wk<ReA	Yes

Les autres

- Rhumatisme streptococcique
- Rhumatisme de Poncet et BCG
- Rhumatisme des clostridium difficile
- La maladie de Whipple
- Mais aussi :
  - Cryptosporidia, Lambliase, Amibiase,Strongyloïdose,
  - Propionibacterium acnes?
  - Borrelia Burgdorferi
  - Capnocytophaga canimorsus

Post-streptococcal reactive arthritis: where are we now

Himanshu Pathak, Tarnya Marshall

- Post-streptococcal reactive arthritis (PSRA) has now emerged as a different clinical entity to acute rheumatic fever.
- PSRA should be considered as one of the differentials for acute polyarthritis in adults.
- There is no agreement about the need and duration of penicillin prophylaxis for PSRA in current literature.

Table 1 Differences between ARF and PSRA

	ARF	PSRA
Age	Single peak at 12 years	Bimodal peaks 8–14 years and 21–37 years
Genetics	Increased expression of HLA DRB1*16 alleles	Increased expression of HLA DRB1*01 alleles
Gender	No difference	No difference
Arthritis	2–3 weeks post-streptococcal infection migratory, flitting, large joints improves in 2–3 weeks, self-limiting	7–10 days post-streptococcal infection non-migratory, additive, small joints, axial, large joints median duration 2 months or more, can be recurrent
Treatment	Good response to Aspirin or NSAIDs	Moderate response to Aspirin/ NSAIDs

ARF, acute rheumatic fever; HLA, human leucocyte antigen; NSAIDs, non-steroidal anti-inflammatory drugs; PSRA, post-streptococcal reactive arthritis.

Pathak H, Marshall T. *BMJ Case Rep* 2016.

Poncet's disease: unusual presentation of a common disease

Shalabh Arora, Turaka Vijay Prakash, Ronald Albert Carey, Samuel George Hansdak

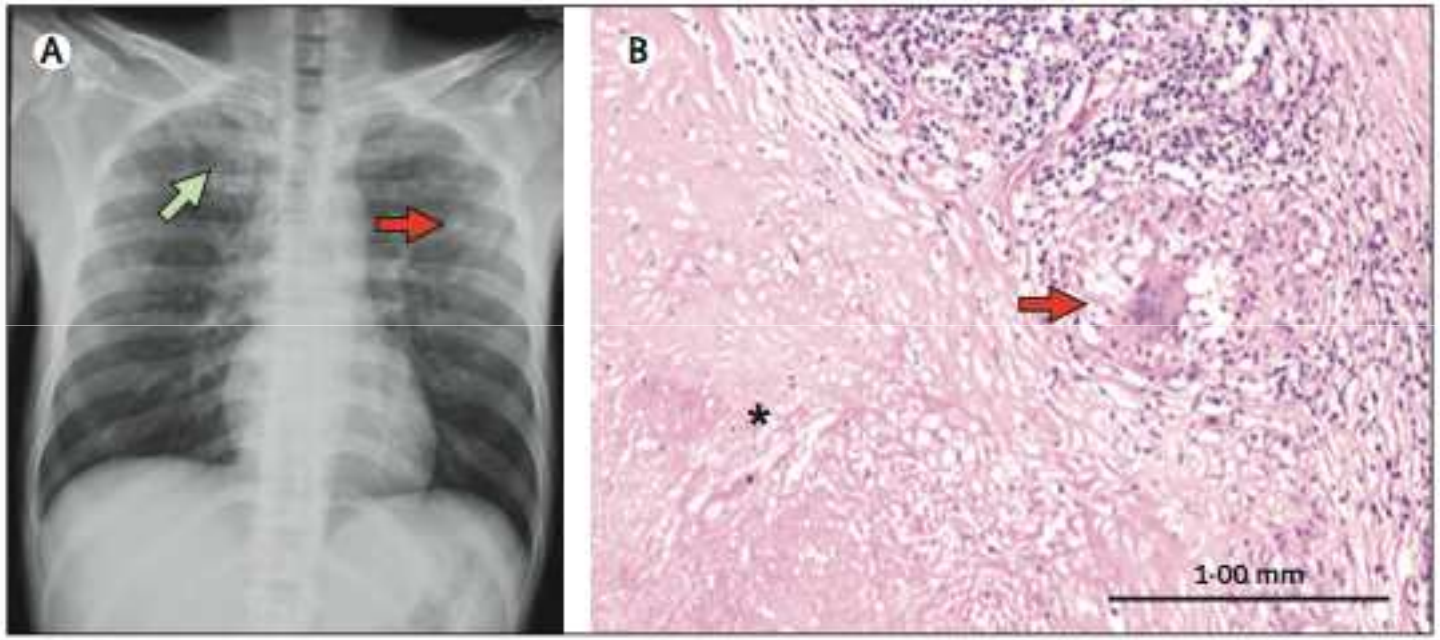


Figure: **Poncet's disease**
(A) Chest radiograph showing bilateral upper and mid-zone parenchymal infiltrates (green arrow) and nodular lesions (red arrow). (B) Haematoxylin and eosin stain of lymph node biopsy sample showing areas of necrosis (asterisk) rimmed by epithelioid cell granulomas with occasional Langhans' multinucleated giant cells (arrow).

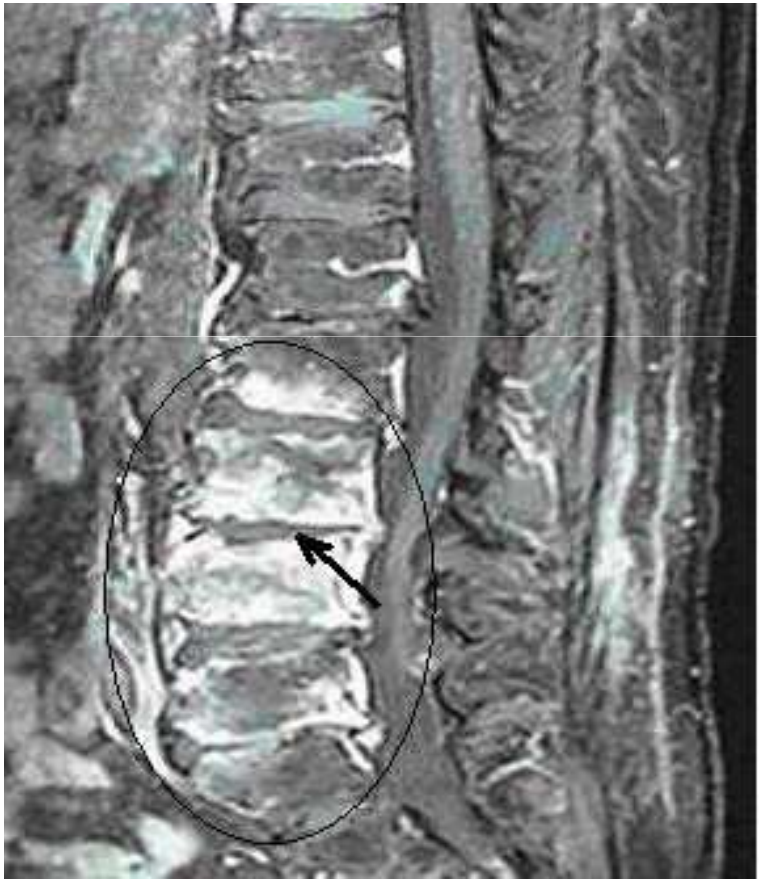
Case Report Comment

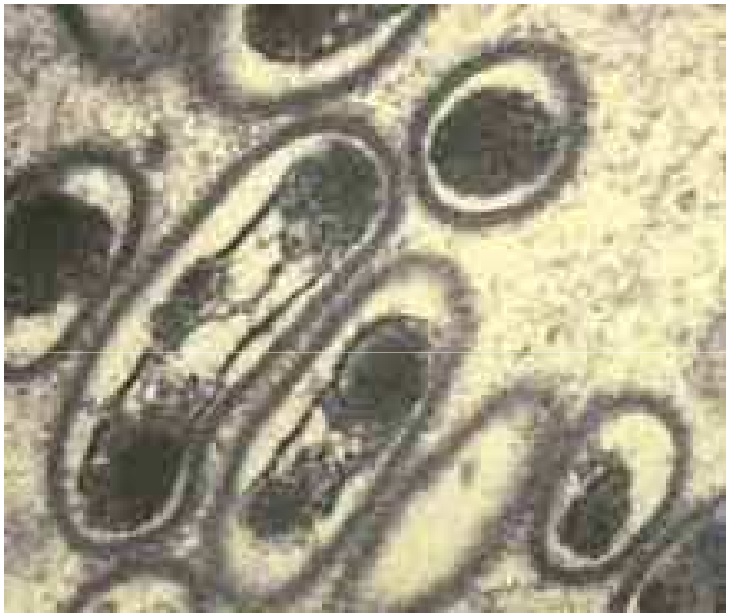
Tuberculosis and Poncet's disease: the many faces of an old enemy

Keertan Dheda

Poncet's disease, a reactive arthritis thought to be immune-mediated, is an extremely rare presentation of tuberculosis.[4,5] Detection occurs disproportionately more frequently in Asia and South America, which might reflect genetic predisposition.[4] Misclassification bias might also play a part; some presumed cases of Poncet's disease were associated with direct mycobacterial joint involvement when synovial biopsy was done. The use of automated DNA-based diagnostics such as GeneXpert MTB/RIF with synovial fluid can reduce such bias.[2]

Maladie de Whipple





"Beware Man's Best Friend"

Caroline Cleuziou, MD, Aymeric Binard, MD, Valérie Devauchelle-Pensec, MD, PhD,
Genevieve Héry-Arnaud, PharmD, PhD, Sandrine Jousse-Joulin, MD,
and Alain Saraux, MD, PhD

Pathologie

- Maladie **rare**
- Décrite en 1907 par Georges whipple
- due à une **bactérie** nommée *Tropheryma whipplei*
- Les **manifestations cliniques** sont **variées** et **rarement spécifiques**
- Toujours **mortelle** en l'absence de traitement
- **L'évolution** est **longue**, marquées par des épisodes de **rémission** et de **rechutes**

Tropheryma whipplei

- longtemps considérée comme non cultivable
- **cultivée** pour la première fois à Marseille, **en 1999**
- bactéries à **Gram positif** à haut GC%
- **séquençage** entier de son génome
- proche de 2 espèces pathogènes chez l'homme, *Actinomyces pyogenes* et *Rothia dentocariosa*
- Sa **source** et sa **transmission** sont **inconnues**
- Présence dans les **eaux d'égouts**

Epidémiologie

- **maladie rare** incidence annuelle : un par million d'habitant
- **ubiquitaire**
- plus fréquente **Suisse et Allemagne, Rhône-Alpes**
- **Transmission par voie orale**
- **hommes** d'environ **50 ans**
- plus fréquente chez les **agriculteurs**
- anomalies immunologiques ?

Signes cliniques

- Longtemps considérée comme une maladie purement digestive.
- les **manifestations cliniques sont très variées** et non spécifiques
- 3 grands tableaux classiques:
  - La maladie de **Whipple classique**, **polyviscérale**
  - L'**endocardite à hémoculture négative** isolée
  - Les **formes neurologiques isolées**

Quand l'évoquer

- **Terrain** : syndrome de malaborption digestive chez un homme de plus de 50 ans.
- Pour le rhumatologue: Oligoarthrite seronegative chronique
- les **signes biologiques d'orientation** :
  - L'hyperleucocytose (inconstante)
  - L'anémie microcytaire hypochrome (plus fréquente)
  - la thrombocytose, l'hyperéosinophilie (très inconstante)
  - élévation de la VS et de la CRP
  - Les signes biologiques de la malabsorption digestive associent : hypoalbuminémie, hypocholesterolémie et carences vitaminiques.

Comment établir le diagnostique?

- Le **diagnostic direct** le plus fréquent , il y a quelques années, était **anatomo-pathologique** : présence d'histiocytes spumeux PAS +
- L'**amplification génique** (PCR) du gène codant pour l'ARNr 16S est la plus utilisée actuellement.
- Cette **technique** est **utilisée sur toute biopsie**, **liquides de ponction, sang**.....
- La **culture** à partir de biopsies, de liquides de ponction ou du sang

Nombre de patients ayant au moins une PCR et un diagnostic selon la ville

Caractéristiques des patients ayant une maladie de Whipple classique, focale ou « réactionnelle »

CASE	PAS on duodenal biopsy	PCR on stool	PCR on saliva	PCR on duodenal biopsy	PCR on joint fluid	PCR on blood	Pattern of Whipple disease
BREST							
1	negative	**positive**	**positive**	negative	not made	not made	CTWAA
2	negative	**positive**	**positive**	negative	not made	negative	CTWAA
3	negative	**positive**	**positive**	negative	**positive**	negative	FWD
4	**positive**	**positive**	**positive**	**positive**	not made	negative	CWD
5	negative	negative	negative	negative	**positive**	negative	FWD
6	negative	**positive**	negative	negative	not made	negative	CTWAA
7	negative	**positive**	**positive**	**positive**	not made	negative	CTWAA
ANGERS							
8	negative	**positive**	**positive**	**positive**	not made	negative	CWD
9	**positive**	**positive**	**positive**	**positive**	not made	**positive**	CWD
10	negative	**positive**	**positive**	not made	not made	negative	CTWAA
11	negative	**positive**	**positive**	not made	not made	negative	CTWAA
12	negative	**positive**	negative	negative	negative	negative	CTWAA
13	negative	**positive**	**positive**	**positive**	negative	negative	CWD

Le traitement

- **Pas d'antibiogramme** possible, le traitement antibiotique reste empirique.
- **Whipple sans atteinte neurologique (PCR dans le LCR négative)**
  - **Doxycycline (Vibramycine®)** cp 100mg, 1 cp 2 fois/j.
  - **Hydroxychloroquine (Plaquenil®)** cp 200mg, 1 cp 3 fois/j.
- Cette association est **administrée pendant une période de 18 mois.**

Le traitement

- **Whipple avec une atteinte neurologique**
  - **Doxycycline (Vibramycine®)** cp 100mg, 1 cp 2 fois/j.
  - **Hydroxychloroquine (Plaquenil®)** cp 200mg, 1 cp 3 fois/j.
  - **Sulfaméthoxazole**: 800 mg et Triméthoprime: 160 mg = Bactrim Forte® : 2 cp 3 fois/j
  - Cette association est administrée pendant une **période minimale de 18 mois**.
  - De plus : Ajout de l'acide folique (Spéciafoldine®): 1 cp à 10 mg/j chez les patients > à 65 ans et les patients carencés

Le traitement

- **contre-indications :**
  - **Doxycycline (Vibramycine®)**
    - Allergie aux tétracyclines.
    - Enfant de moins de 8 ans.
    - Femme enceinte ou qui allaite.
  - **Hydroxychloroquine (Plaquénil®)**
    - Allergie à l'hydroxychloroquine.
    - Rétinopathie.
  - **Cotrimoxazole (Bactrim Forte®)**
    - Allergie aux sulfamides.
    - Déficit en G6PD : risque de déclenchement d'une hémolyse.

Le traitement

- **effets indésirables :**
  - **Doxycycline (Vibramycine®)**
    - **Photosensibilisation** Troubles digestifs, réactions allergiques, troubles hématologiques, pigmentation cutanée.
  - **Hydroxychloroquine (Plaquénil®)**
    - **Rétinopathie,** photosensibilisation, acouphènes, vertiges, troubles digestifs, réactions cutanéo-muqueuses, céphalées, dépôts cornéens régressifs à l'arrêt du traitement, troubles hématologiques**,** psychose.
  - **Cotrimoxazole (Bactrim Forte®)**
    - Allergies cutanées, troubles hématologiques, troubles digestifs, hépatite, colite pseudomembraneuse, altération de la fonction rénale, lithiase urinaire, méningite aseptique**.**

Le suivi des patients

- En **l'absence** de traitement **antibiotique** adapté, l'évolution est **mortelle**.
- **diarrhée** disparaît en **moins d'une semaine**.
- **arthralgies** régresse en **2 à 3 semaines**.
- **signes neurologiques** régressent en plus de **3 semaines**
- **séquelles neurologiques fréquentes**
- **rechutes** sont à redouter, en particulier, au niveau **cérébral**. **Fréquence** estimée entre **20 et 35%**.
- **Suivi à vie** conseillé.

Arthrite réactionnelle à clostridium

Table 1: Case reports in the literature on *Clostridium difficile* enterocolitis-associated reactive arthritis in children.

Authors/articles	Cron and Gordon 1997 [3]	Löffler et al. 2004 [5]		Durand and Miller 2009 [7]	Finger and Neubauer 1997 [4]	Dacheux et al. 2012 [6]
Age	*2,5*	*11*	*6*	*10*	*3*	*7*
Sex	M	M	F	F	M	M
Previous antibiotic therapy	Amoxicillin	Lincomycin	ND	Erythromycin and penicillin V	Cefixime	Amoxicillin-clavulanate
Joints involved	Left knee and shoulder	Left and right ankles; right knee; both hips; both elbows; left wrist	Both knees, both elbows, and both hips	Left hips	Right hip, shoulder, and knee	Twelve joints
Symptoms associated		Skin nodules		Aseptic peritonitis		
Treatment	Vancomycin	Vancomycin	Vancomycin	Metronidazole	Ibuprofen	Metronidazole
Disease's duration (days)	25	588	14	7	7	7

(1) Appearance of arthritis together with or following the onset of diarrhea and/or colitis.

(2) Diarrhea appearing some time after a course of systemic antimicrobial therapy.

(3) Microbiologic proof of CD involvement (either positive stool culture or assay for toxin).

(4) No reasonable alternative diagnosis for arthritis or diarrhea (i.e., no other identified infectious agent).

C. Putterman and A. Rubinow, "Reactive arthritis associated with *Clostridium difficile* pseudomembranous colitis," *Seminars in Arthritis and Rheumatism*, vol. 22, no. 6, pp. 420–426, 1993.

Case Rep Pediatr. 2016;2016:1591753. doi: 10.1155/2016/1591753. Epub 2016 Apr 14. **Clostridium difficile Enterocolitis and Reactive Arthritis: A Case Report and Review of the Literature.** Cappella M[1], Pugliese F[1], Zucchini A[1], Marchetti F[1].

Bacillus Calmette-Guérin (BCG) Infection Following Intravesical BCG Administration as Adjunctive Therapy For Bladder Cancer

Incidence, Risk Factors, and Outcome in a Single-Institution Series and Review of the Literature

María Asunción Pérez-Jacoiste Asín, MD, Mario Fernández-Ruiz, MD, Francisco López-Medrano, MD, PhD, Carlos Lumbreras, MD, PhD, Ángel Tejido, MD, Rafael San Juan, MD, PhD, Ana Arrebola-Pajares, MD, Manuel Lizasoain, MD, Santiago Prieto, MD, PhD, and José María Aguado, MD, PhD

Abstract: Bacillus Calmette-Guérin (BCG) is the most effective intravesical immunotherapy for superficial bladder cancer. Although generally well tolerated, BCG-related infectious complications may occur following instillation. Much of the current knowledge about this complication comes from single case reports, with heterogeneous diagnostic and therapeutic approaches and no investigation on risk factors for its occurrence. We retrospectively analyzed 256 patients treated with intravesical BCG in our institution during a 6-year period, with a minimum follow-up of 6 months after the last instillation. We also conducted a comprehensive review and pooled analysis of additional cases reported in the literature since 1975. Eleven patients (4.3%) developed systemic BCG infection in our institution, with miliary tuberculosis as the most common form (6 cases). A 3-drug antituberculosis regimen was initiated in all but 1 patient, with a favorable outcome in 9/10 cases. There were no significant differences in the mean number of transurethral resections prior to the first instillation, the time interval between both procedures, the overall mean number of instillations, or the presence of underlying immunosuppression between patients with or without BCG infection. We included 282 patients in the pooled analysis (271 from the literature and 11 from our institution). Disseminated (34.4%), genitourinary (23.4%), and osteomuscular (19.9%) infections were the most common presentations of disease. Specimens for microbiologic diagnosis were obtained in 87.2% of cases, and the diagnostic performances for acid-fast staining, conventional culture, and polymerase chain reaction (PCR)-based assays were 25.3%, 40.9%, and 41.8%, respectively. Most patients (82.5%) received antituberculosis therapy for a median of 6.0 (interquartile range: 4.0–9.0) months. Patients with disseminated infection more commonly received antituberculosis therapy and adjuvant corticosteroids, whereas those with reactive arthritis were frequently treated only with nonsteroidal antiinflammatory drugs ($p < 0.001$ for all comparisons). Attributable mortality was higher for patients aged ≥65 years (7.4% vs 2.1%; $p = 0.091$) and those with disseminated infection (9.9% vs 3.0%; $p = 0.040$) and vascular involvement (16.7% vs 4.6%; $p = 0.064$). The scheduled BCG regimen was resumed in only 2 of 36 patients with available data (5.6%), with an uneventful outcome. In the absence of an apparent predictor of the development of disseminated BCG infection after intravesical therapy, and considering the protean variety of clinical manifestations, it is essential to keep a high index of suspicion to initiate adequate therapy promptly and to evaluate carefully the risk-benefit balance of resuming intravesical BCG immunotherapy.

(*Medicine* 2014;93: 236–254)

Bacillus Calmette-Guérin (BCG) Infection Following Intravesical BCG Administration as Adjunctive Therapy For Bladder Cancer

Incidence, Risk Factors, and Outcome in a Single-Institution Series and Review of the Literature

María Asunción Pérez-Jacoiste Asín, MD, Mario Fernández-Ruiz, MD, Francisco López-Medrano, MD, PhD, Carlos Lumbreras, MD, PhD, Ángel Tejido, MD, Rafael San Juan, MD, PhD, Ana Arrebola-Pajares, MD, Manuel Lizasoain, MD, Santiago Prieto, MD, PhD, and José María Aguado, MD, PhD

TABLE 3. Demographics, Chronic Comorbidities and Underlying Factors in 282 Patients Diagnosed With BCG Infection Following Intravesical BCG Instillation (Pooled Analysis of Institutional Series and Case Reports From the Literature)

Variable*	No. of Patients (%)
Age, yr (mean ± SD) [276]	66.6 ± 11.0
Sex, male [279]	268 (96.1)
Active smoking [112]	19 (16.9)
Active alcohol use [112]	4 (3.6)
Chronic comorbidities [112]	
Hypertension	26 (23.2)
Diabetes mellitus	16 (14.3)
Renal insufficiency	8 (7.1)
Liver cirrhosis	0 (0.0)
COPD	14 (12.5)
Other non-bladder malignancy [282]	21 (7.4)
Immunosuppression [282]	5 (1.8)
Previous active tuberculosis [282]	16 (5.7)
Previous positive TST [282]	3 (1.1)

*Values in brackets represent number of patients for whom data were available.

TABLE 4. Type of BCG Infection in 282 Patients

Type of Complication	No. of Patients (%)
Systemic	
Disseminated BCG infection*	97 (34.4)
Persistent fever (as isolated manifestation)	4 (1.4)
Osteomuscular	56 (19.9)
Arthritis	20 (7.1)
Reactive arthritis	16 (5.7)
Spondylodiscitis	10 (3.5)
Prosthetic joint infection	5 (1.8)
Muscle abscess (as isolated manifestation)†	5 (1.8)
Vascular	19 (6.7)
Mycotic aneurysm or pseudoaneurysm‡	13 (4.6)
Mycotic aneurysm with fistulization	2 (0.7)
Aorto-enteric fistula	2 (0.7)
Infection of vascular bypass graft	2 (0.7)
Ocular (with no articular involvement)	9 (3.2)
Uveitis	7 (2.4)
Endophthalmitis	1 (0.4)
Autoimmune retinopathy	1 (0.4)
Hepatitis (as isolated manifestation)	16 (5.7)
Cutaneous§	4 (1.4)
Pulmonary (other than miliary tuberculosis)	2 (0.7)
Meningitis	1 (0.4)
Other¶	6 (2.1)
Mixed complications**	2 (0.7)
Local (genitourinary)	66 (23.4)
Bladder involvement	17 (5.9)
Penile lesions	17 (5.9)
Prostatitis	11 (3.5)
Kidney parenchymal involvement	10 (3.5)
Epididymo-orchitis	10 (3.5)
Pyeloureteral stenosis	1 (0.35)

*See definition in text.

†Overall, 11 patients (3.9%) were diagnosed with muscle abscess: 3 additional cases were secondary to spondylodiscitis; 2 were secondary to mycotic aneurysm or pseudoaneurysm; and 1 was secondary to infection of hip prosthesis.

‡Overall, 16 patients (5.7%) were diagnosed with mycotic aneurysm; 1 additional case was secondary to spondylodiscitis.

§Includes subcutaneous nodules (2 cases), plaque and abscess (1 case each).

¶Includes peritoneal tuberculosis (2 cases), parotid gland tuberculosis, enteritis, rhabdomyolysis, and prevesical abscess (1 case each).

**Includes 1 case of pleuropericardic effusion with arthritis and penile lesion, and 1 case of cold abscess of the chest wall with rash and penile lesion.

TABLE 6. Drug Regimens Used in 226 Patients Who Received Antituberculosis Therapy

Regimen	No. of Patients (%)	Duration, Mo [mean (Q_1–Q_3 Range)]
INH	13 (5.8)	3 (2–4)
INH + RIF	64 (28.3)	6 (3–6)
INH + EMB	5 (2.2)	8 (5–12)
RIF + EMB	3 (1.3)	6 (6–12)
INH + RIF + EMB	74 (32.7)	6.5 (6–9)[†]
INH + RIF + PZA	16 (7.1)	6 (5–6)[†]
INH + EMB + PZA	1 (0.4)	3
INH + RIF* + EMB + PZA	17 (7.5)	6.5 (3.75–12)[†]
INH + RIF + aminoglycoside	5 (2.2)	9 (6–12)
INH + RIF + quinolone	2 (0.9)	6
INH + RIF + EMB + aminoglycoside	1 (0.4)	12[†]
INH + RIF + EMB + quinolone	3 (1.3)	12 (9–15)[†]
Other combinations	13 (5.8)	
Not specified	9 (4.0)	

*Includes 1 case treated with rifabutin.

[†]In most cases comprises a 3- or 4-drug regimen during the first 2 months, followed by INH + RIF until completion of therapy.

TABLE 1. Underlying Conditions, Clinical Characteristics, Therapeutic Approaches and Outcome in 11 Patients With Systemic BCG Infection, Present Report

Patient	Age/ Sex	Underlying Condition	IS	No. of TUR/ Time Interval Between TUR and Instillation	No. of Intravesical BCG Instillations	Traumatic Instillation	Time Interval Between BCG Instillation and Diagnosis	Type of BCG Infection	Miliary Pattern	Isolation of *M. Bovis* on Culture	Therapy	Outcome
1	73 y/M	Hypertension, diabetes, ESRD, kidney carcinoma	No	1/34 d	7	NR	6 d	Lymphocytic meningitis	No	Yes (urine)	INH + RIF + LEV (6 mo)	Resolution
2	56 y/M	Hypertension, active smoking and alcohol use	No	1/16 d	6	Yes	13 d	Miliary tuberculosis	Yes	No	INH + RIF + EMB (6 mo)	Resolution
3	74 y/M	Previous tuberculosis	No	1/37 d	7	NR	13 d	Arthritis, conjunctivitis	No	No	INH + RIF + EMB (6 mo)	Resolution
4	70 y/M	None	No	3/35 d	2	NR	1 d	Persistent fever	No	Yes (urine)	INH + RIF + EMB (6 mo)	Resolution
5	56 y/M	Hypertension	No	1/40 d	10	NR	16 d	Miliary tuberculosis, hepatitis	Yes	No	INH + RIF + EMB (6 mo)	Resolution
6	71 y/M	Hypertension, diabetes, ESRD	No	6/49 d	14	NR	2 d	Persistent fever	No	No	INH + RIF + EMB (6 mo)	Resolution
7	72 y/M	Hypertension, active smoking, COPD	No	3/24 d	11	NR	1 d	Miliary tuberculosis	Yes	Yes (urine)	INH + RIF + EMB (6 mo)	Resolution
8	58 y/M	Active smoking	No	1/33 d	14	NR	1 d	Miliary tuberculosis	Yes	Yes (urine)	INH + RIF + EMB (6 mo)	Resolution
9	73 y/M	Active smoking, COPD	No	2/22 d	9	NR	10 d	Miliary tuberculosis	Yes	No	INH + EMB + LEV (6 mo)	Resolution
10	76 y/M	Hypertension	No	2/29 d	9	NR	19 d	Tubulointerstitial nephritis[120]	No	No†	Corticosteroids (3 mo)	ESRD (stage 4)
11	76 y/M	Active smoking and alcohol use, colorectal carcinoma	Yes*	2/42 d	12	NR	9 d	Miliary tuberculosis	Yes	No‡	INH + RIF + EMB	Multiorgan failure and dea

*Splenectomy.
†Kidney biopsy showed a diffuse and dense interstitial cellular infiltrate composed of lymphocytes, plasmatic cells, and eosinophils, with no interstitial or perivascular granulomas.
‡Necropsy revealed miliary granulomatosis in the lungs.

TABLE 2. Demographics, Underlying Conditions, and Bladder Cancer Treatment-Related Variables in Patients With and Without Systemic BCG Infection After Intravesical BCG Instillation

Variable	BCG Infection (n = 11)	No BCG Infection (n = 245)	P
Age, yr (mean ± SD)	68.6 ± 8.1	67.9 ± 8.8	0.753
Sex, male (%)	11 (100.0)	216 (88.2)	0.619
Active smoking (%)	5 (45.5)	133 (54.3)	0.565
Active alcohol use (%)	2 (18.2)	11 (4.5)	0.100
Chronic comorbidities (%)			
Hypertension	6 (54.6)	105 (42.9)	0.539
Diabetes mellitus	2 (18.2)	45 (18.4)	1.000
Renal insufficiency	2 (18.2)	19 (7.8)	0.225
Liver cirrhosis	0 (0.0)	2 (0.8)	1.000
COPD	2 (0.0)	33 (13.5)	0.650
Other non-bladder malignancy (%)	2 (18.2)	31 (12.7)	0.638
Immunosuppression (%)*	1 (9.1)	10 (4.1)	0.389
Previous active tuberculosis (%)	1 (9.1)	4 (1.6)	0.199
Previous positive TST (%)	0 (0.0)	5 (2.1)	1.000
No. of previous TURs (mean ± SD)	2.1 ± 1.5	2.3 ± 1.6	0.772
Time interval between the last TUR and the first BCG instillation, d (mean ± SD)	32.8 ± 9.6	40.6 ± 24.4	0.169
No. of intravesical BCG instillations (mean ± SD)	9.2 ± 3.6	9.9 ± 5.6	0.863
Total leukocyte count, x 10^3 cells/μL (mean ± SD)*	8.2 ± 2.3	7.7 ± 1.9	0.468
Total lymphocyte count, x 10^3 cells/μL (mean ± SD)*	2.5 ± 0.6	2.3 ± 1.0	0.123

*At the time of the first intravesical BCG instillation.

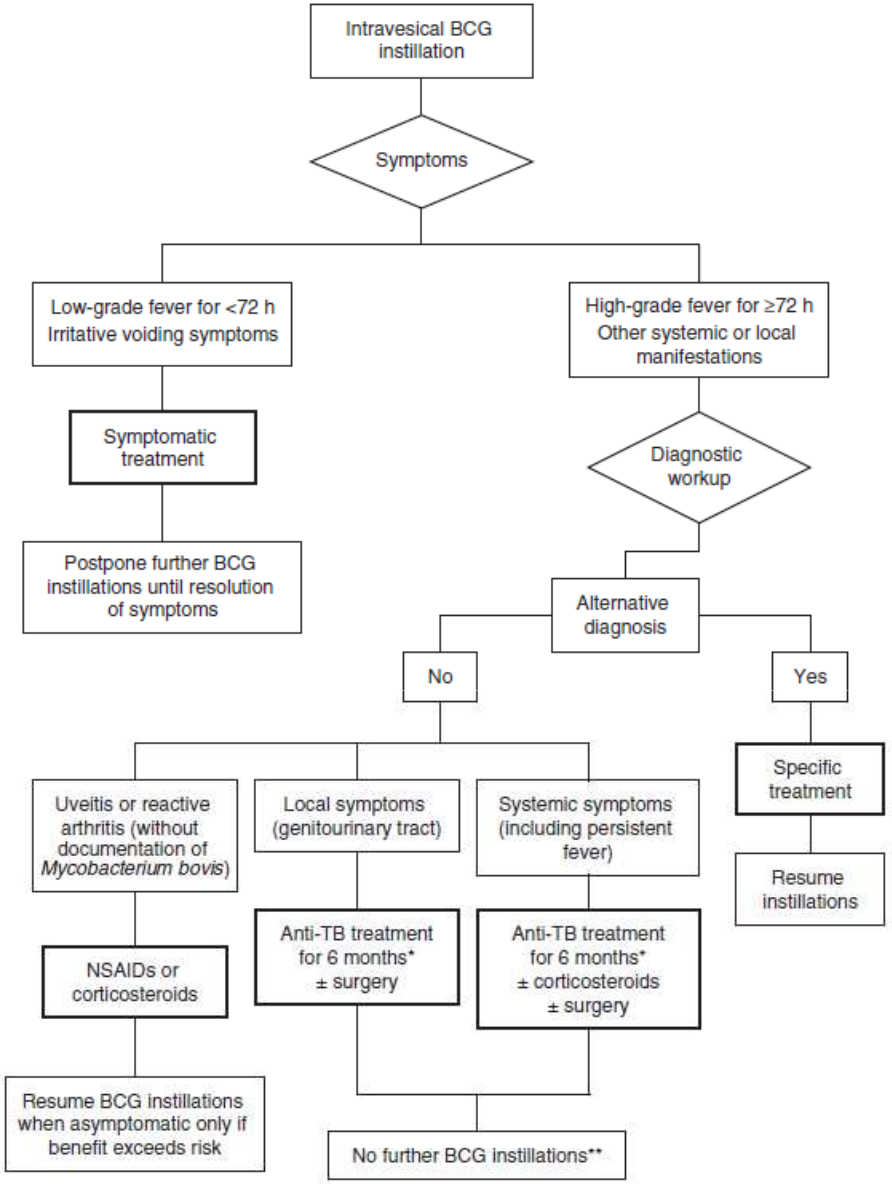


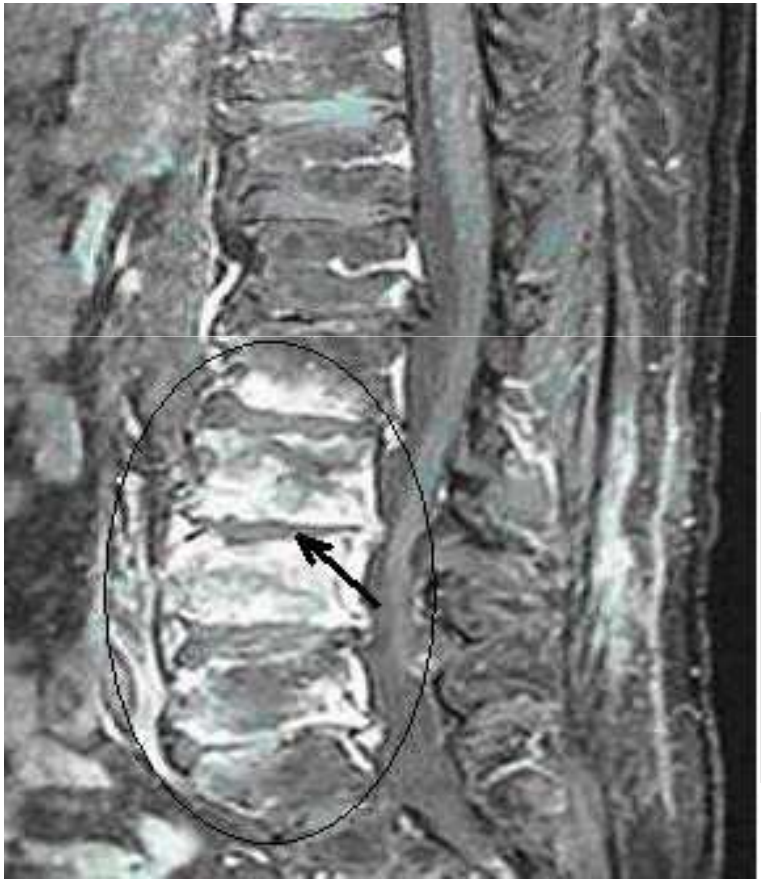
FIGURE 1. Proposal of a diagnostic and therapeutic algorithm for patients with suspected BCG infection following BCG instillation. The terms "low-grade" and "high-grade fever" refer to body temperature <37.9°C and ≥38°C, respectively. *Antituberculosis treatment should include INH, RIF, and EMB for 2 months, and INH and RIF for 4 more months. **Continuation of BCG instillations could be considered in patients with persistent fever and no miliary pattern on chest imaging, once antituberculosis treatment has been completed, and only if the expected benefits of BCG therapy clearly exceed the risks (that is, high-grade carcinoma).

Association between Giardia and arthritis or joint pain in a large health insurance cohort: could it be reactive arthritis?

J. E. PAINTER [a1], S. A. COLLIER [a2] and J. W. GARGANO [a2]

- This study aimed to assess the association between giardiasis and subsequent development of arthritis or joint pain using a retrospective cohort of individuals from a large administrative claims database in the United States.
- Using 2006–2010 data from MarketScan Commercial Claims and Encounters,
- we conducted a retrospective cohort study in people with an ICD-9-CM code for giardiasis (n = 3301) and persons without giardiasis (n = 14 612) individually matched on age, sex, and enrolment length.
- We used conditional logistic regression to model the association between giardiasis and arthritis or joint pain documented in the 6 months following initial giardiasis diagnosis or index date for matched controls.
- After adjusting for healthcare utilization rate, giardiasis was associated with a 51% increase in claims for arthritis or joint pain (odds ratio 1·51, 95% confidence interval 1·26–1·80).
- In age- and sex-stratified adjusted analyses, the association remained significant across all subgroups (age 0–19 years, age 20–64 years, males, and females). Findings from this study lend epidemiological support for the association between giardiasis and subsequent development of arthritis. Reactive arthritis might occur more frequently than has been reported in the literature. Further research is necessary to determine the mechanisms by which giardiasis could lead to arthritis.

Capnocytophaga canimorsus



"Beware Man's Best Friend"

Caroline Cleuziou, MD, Aymeric Binard, MD, Valérie Devauchelle-Pensec, MD, PhD, Genevieve Héry-Arnaud, PharmD, PhD, Sandrine Jousse-Joulin, MD, and Alain Saraux, MD, PhD

■ Key Points

- Broad-range PCR provided the microbiological diagnosis of culture-negative discitis in our patient.
- Broad-range PCR should be considered in patients with culture-negative osteoarticular infections.

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