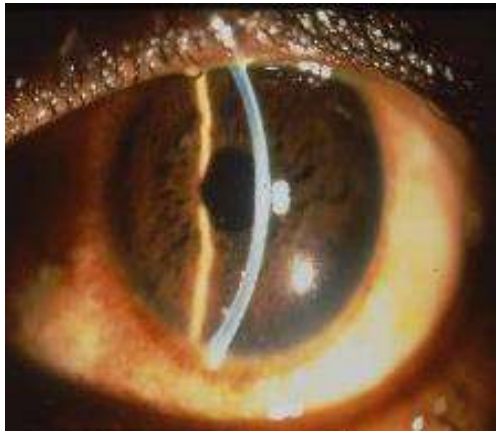
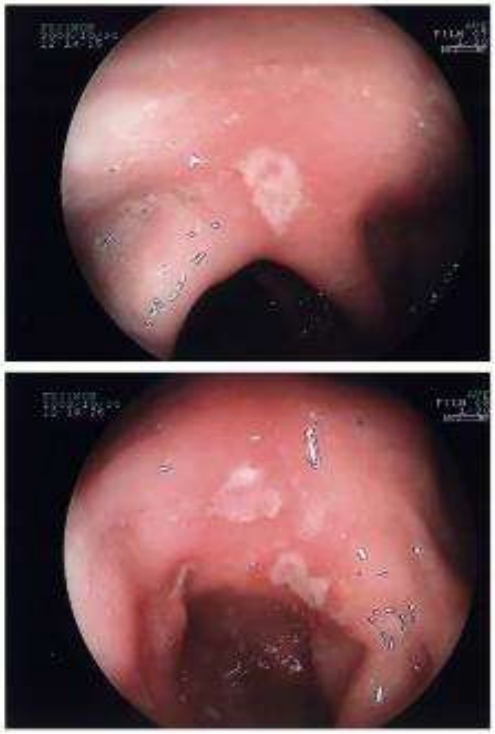
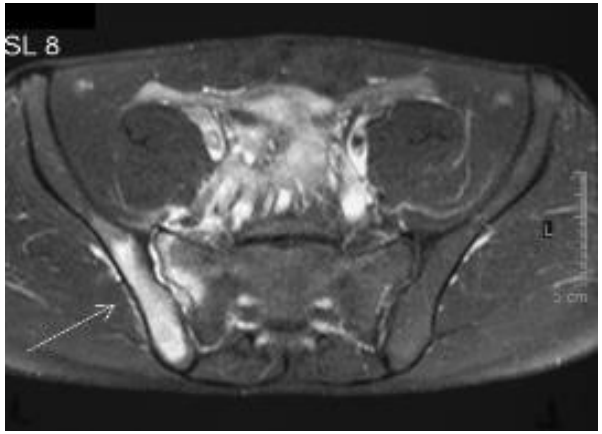
Que reste-t-il des arthrites réactionnelles?







Pr A Saraux
Chu Brest, France

Cas clinique

- Homme 60 ans
- Consulte pour fièvre brutale à 40° 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
- Quels diagnostics sont envisageables?

Cas clinique

1. Arthrite réactionnelle de spondyloarthrite
2. Arthrite septique mycobactérienne
3. Arthrite réactionnelle à mycobactérie
4. Arthrite septique à Salmonelle
5. Arthrite réactionnelle a clostridium difficile

Cas clinique

1. Arthrite réactionnelle de spondyloarthrite
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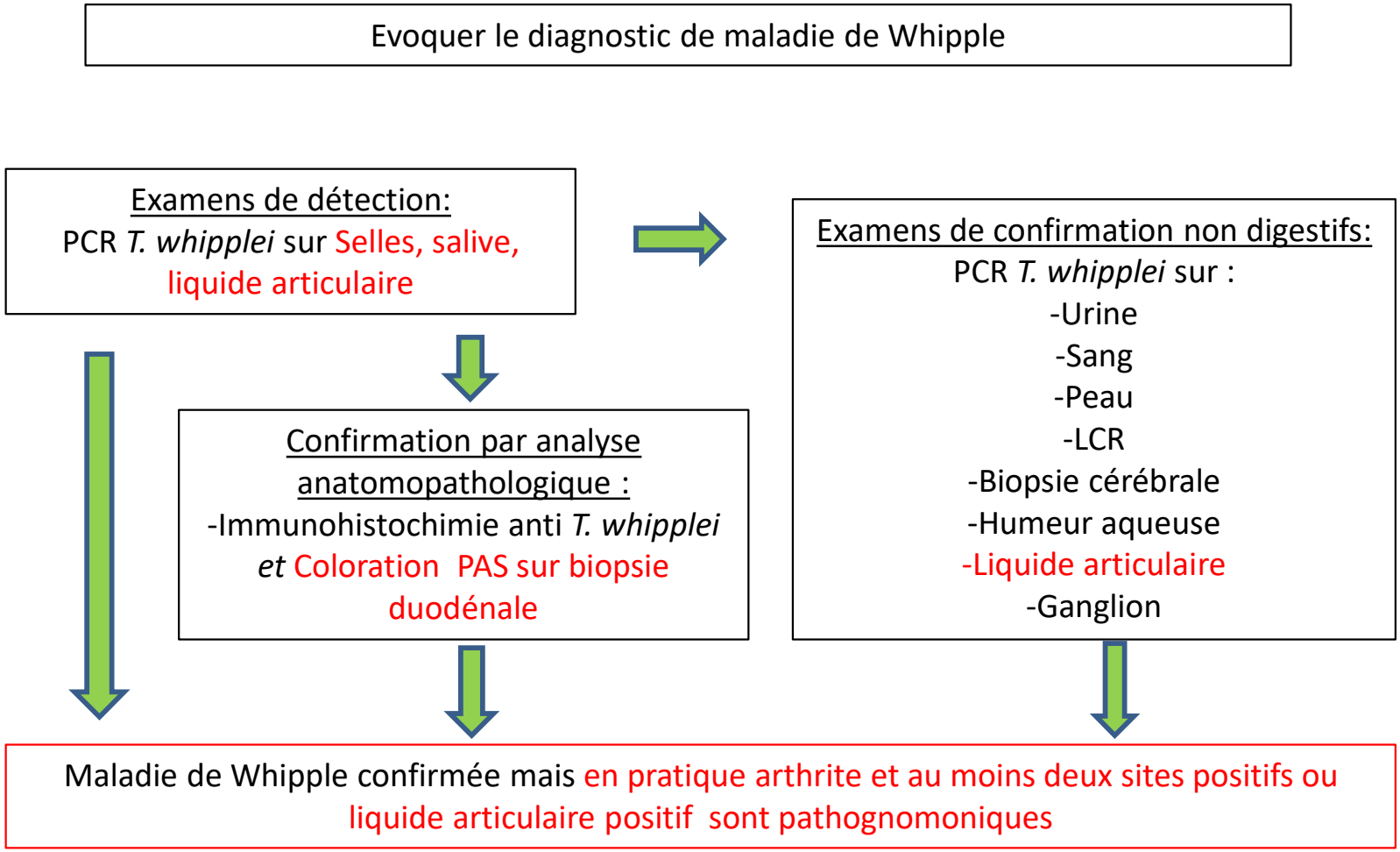
3. Arthrite réactionnelle à mycobactérie
4. Arthrite septique à Salmonelle
5. Arthrite réactionnelle a clostridium difficile

Lien infection-articulation

- Pour la WHO
  - 1- germe dans l'articulation
  - 2- post infectieux avec antigène dans l'articulation
  - 3- Arthrite réactionnelle urogénitale ou digestive
  - 4- Déclenchée par un microbe qui n'est pas, ni ses antigènes, dans l'articulation (ex: post streptococcique)

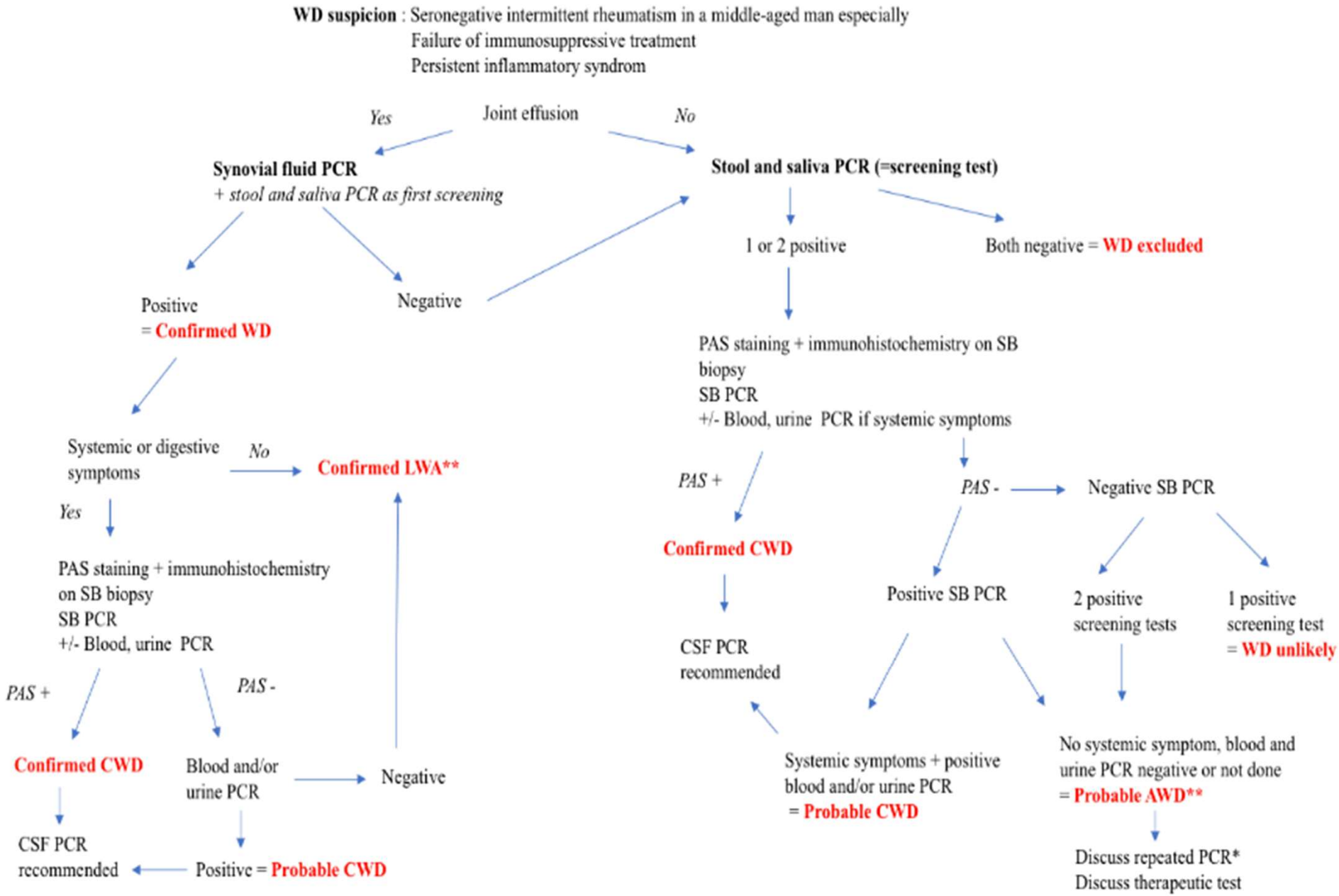
L’exemple de la maladie de Whipple

- Décrite en 1907 par George Whipple
- due à une *Tropheryma whipplei*
  - **cultivée** pour la première fois à Marseille, **en 1999**
  - bactéries à **Gram positif**
  - **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**
- Classiquement **mortelle** en l’absence de traitement
- **L'évolution** est **longue**, marquées par des épisodes de **rémission** et de **rechutes**



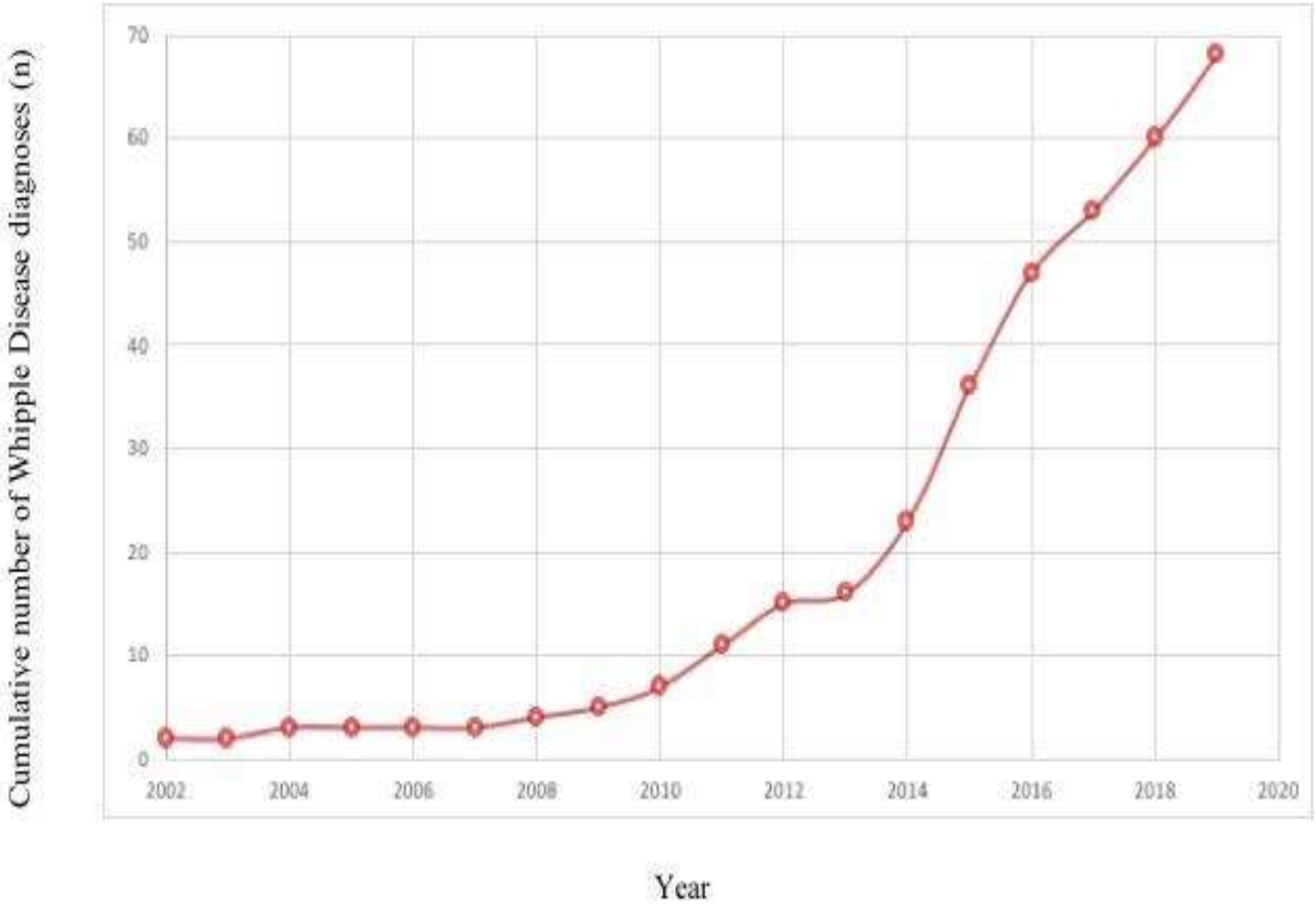
Tison A. Scientific report 2021

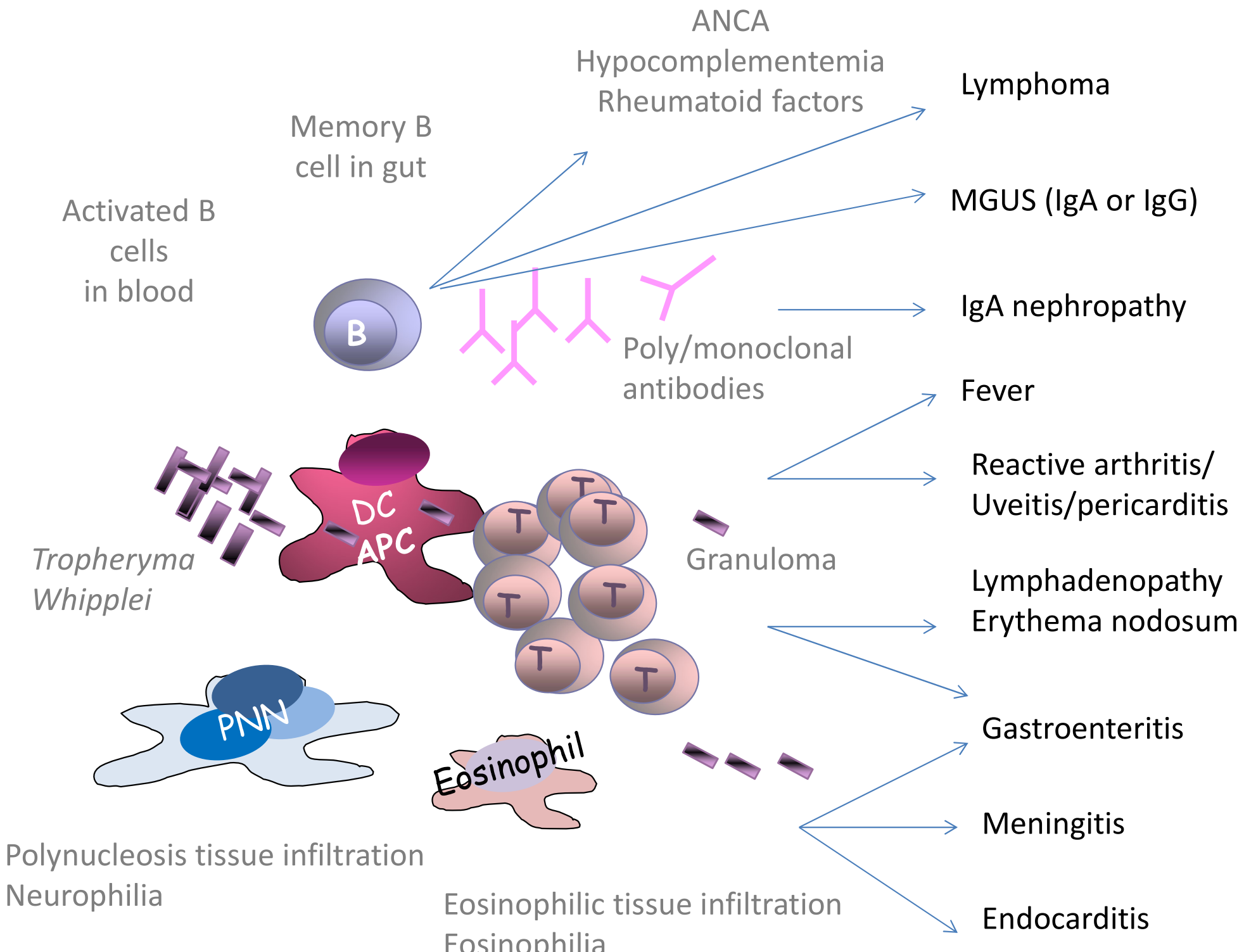
Figure 2 – Proposed algorithm for Whipple disease in patients in whom rheumatism is suspected



*Legend : CSF = Cerebro-spinal fluid ; CWD = Classic Whipple Disease ;LWA = Localized Whipple Arthritis ; PAS = Periodic Acid-Schiff ; SB = Small Bowel; WD = Whipple disease ; † Cases of spondylodiscitis not considered in this algorithm ; ** Discuss CSF PCR in case of neurologic symptoms ; * In case of chronic arthritis with negative synovial fluid PCR, discuss synovial biopsy. NB : Data on cutaneous biopsy PCR were to scarce in our cohort to integrate it on the algorithm.*

Supplementary Figure 1 – Evolution of number of Whipple disease diagnoses across years in the cohort




ANCA
Hypocomplementemia
Rheumatoid factors
Lymphoma
Memory B
cell in gut
Activated B
cells
in blood
MGUS (IgA or IgG)
B
IgA nephropathy
Poly/monoclonal
antibodies
Fever
Reactive arthritis/
Uveitis/pericarditis
DC
APC
T
Tropheryma
Whipplei
Granuloma
Lymphadenopathy
Erythema nodosum
PNN
Gastroenteritis
Eosinophil
Meningitis
Endocarditis
Polynucleosis tissue infiltration
Neurophilia
Eosinophilic tissue infiltration
Eosinophilia

Traitement et surveillance

- Doxycycline = 100 mg X 2/ jour
- Hydroxychloroquine = 200 mg X 3/jour

Une **protection solaire** car photosensibilisation due à la doxycycline.

Une **consultation ophtalmologique**

Si impossible Bactrim

Au total maladie de Whipple

Chez un **homme âgé de 40 à 75 ans**,

Ayant une **polyarthrite** chronique classiquement non érosive (mais pas si ancien) intermittente (ou persistante…) séronégative (ou positive…) touchant les grosses articulations **inexpliquée**

Une recherche de maladie de Whipple doit être effectuée par **PCR dans la salive, les selles et si possible le liquide articulaire**

Si **PCR positive, une recherche de la forme classique de la maladie par biopsie duodénale** doit être faite.

Si doute sur une endocardite PCR sanguine et **si doute** sur atteinte neurologique PCR dans le LCR

Lien infection-articulation

- Pour la WHO
  - 1- germe dans l'articulation
  - 2- post infectieux avec antigène dans l'articulation
  - 3- Arthrite réactionnelle urogénitale ou digestive
  - 4- Déclenchée par un microbe qui n'est pas, ni ses antigènes, dans l'articulation (ex: post streptococcique)

Poncet's disease: unusual presentation of a common disease

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

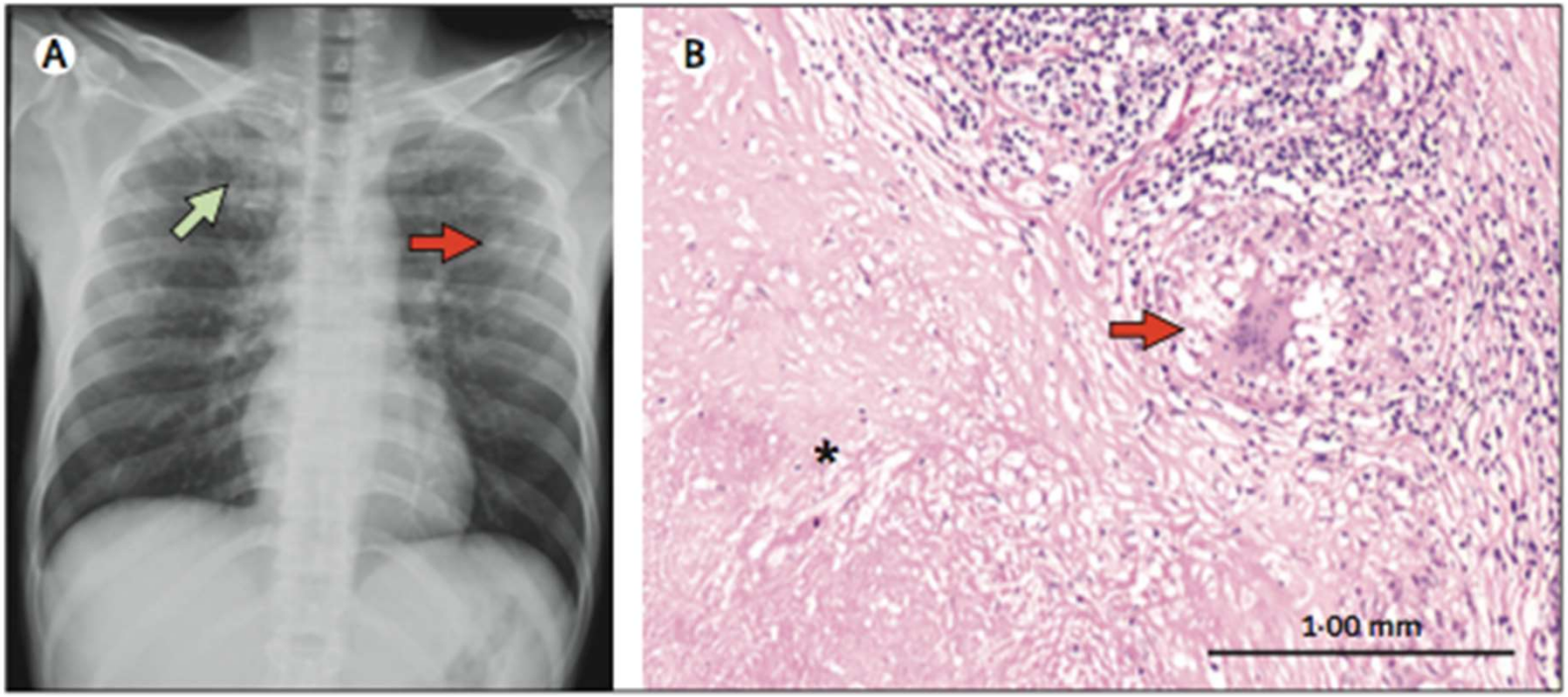


Figure: **Poncet's disease**
(A) Chest radiograph showing bilateral upper and mid-zone parenchymal infiltrates (green arrow) and nodular lesions (red arrow). (B) Haematoxylin and eosin stain of lymph node biopsy sample showing areas of necrosis (asterisk) rimmed by epithelioid cell granulomas with occasional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

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)

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.

Lien infection-articulation

- Pour la WHO
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  - 3- Arthrite réactionnelle urogénitale ou digestive
  - 4- Déclenchée par un microbe qui n'est pas, ni ses antigènes, dans l'articulation (ex: post streptococcique)

Si la majorité sont des spondyloarthrites….

Table 1 Bacterial species/groups known to be primary causes of HLA-B27 positive ReA, and identification of bacteria and bacterial products in the joint by various methods [modified from 10–12]

Entry site	Bacteria	Bacterial products
Urogenital tract	*Chlamydia trachomatis*	Antigens, DNA, RNA, short-lived primary ribosomal RNA transcripts (viability), aberrant organism by electron microscopy
Gastrointestinal tract	*Yersinia enterocolitica O3, O8, and O9*	Antigens, RNA, DNA
	Y. pseudotuberculosis	Antigens, RNA
	Salmonella enterica serovars *Typhimurium enteritidis, Paratyphi B and C, and others*	Antigens, DNA
	Shigella flexneri, S. sonnei and *S. dysenteriae*	Antigens, DNA
	Campylobacter jejuni	DNA
Respiratory tract	*Chlamydia pneumoniae*	Antigens, DNA, RNA

Table 2 Rare infectious agents implicated to cause ReA [modified from 11, 24, 25] (see supplement for additional references reporting microbial products in the joint and HLA-B27 positivity)

Entry site	Microbial agents	Microbial products in the joint	HLA-B 27 positive
Urogenital tract	*Gardnerella vaginalis*	ND	Positive
	Human immunodeficiency virus	Virus isolated in one patient	Positive in Caucasians
	Mycoplasma genitalium/hominis/orale	DNA, coinfections	Single case
	*Neisseria gonorrhoea**	DNA	No
	Ureaplasma urealyticum	DNA, coinfections	No
Gastrointestinal tract	*Blastocytosis**	ND	Single case
	Clostridium difficile	ND	Yes
	*Cyclospora cayetanensis**	ND	No
	Echerichia coli	ND	Some cases
	Hafnia alvei	ND	No
	Helicobacter pylori	ND	Yes
	Microsporidia	Antigens, DNA, culture positive	Single case
	*Strongyloides stercoralis**	Larva and antigen found in SM (one case)	Single case
	*Tropheryma whippelii***	DNA, rRNA, culture positive	Some cases
	Vibrio parahaemolyticus	ND	ND
Amoebae	*Cryptosporidium*	ND	No
	*Entamoeba histolytica**	Culture positive in one case	No
	*Entamoeba hartmanni**	ND	Single case
	*Giardia lamblia**	ND	Some cases

Table 2 Rare infectious agents implicated to cause ReA [modified from 11, 24, 25] (see supplement for additional references reporting microbial products in the joint and HLA-B27 positivity)

Entry site	Microbial agents	Microbial products in the joint	HLA-B 27 positive
Respiratory tract	*β-haemolytic Streptococci*	ND	Some cases
	*Mycobacterium tuberculosis**	65 kDa mycobacterial heat shock protein reactive T-cells	Some cases
	Mycoplasma pneumoniae	ND	Some cases
	Neisseria meningitidis	Immune complexes	Single case
Other (skin, soft tissue)	*Bartonella henselae*	ND	No
	*Borrelia burgdorferi***	DNA	No
	Brucella abortus/mellitensis	Culture negative by definition	No
	Calmette-Guerin Bacillus	*Mycobacteri bovis* culture positive in one case	Yes
	Coxiella buenetii	ND	ND
	Leptospira	ND	ND
	*Orientia tsutsugamushi**	ND	ND
	Propionibacterium acnes	Culture positive	Single case
	Pseudomonas aeruginosa	DNA	No
	*Rickettsia conorii**	Immune complexes	No
	*Rickettsia rickettsii**	Culture negative (single case)	Single case
	Staphylococcus aureus	ND	Some cases
	Staphylococcus epidermidis	ND	Single case
	Staphylococcus haemolyticus	ND	Single case
	Staphylococcus lugdunensis	ND	ND
Vaccination	Hepatitis B	ND	Two cases
	Influenza	ND	Single case
	Measles plus mumps	ND	ND
	Tetanus	ND	Positive
	Typhoid	ND	No

*Effective antimicrobial therapy for ReA documented in case reports

**Effective antimicrobial therapy for ReA documented in double-blind controlled studies

Lien infection-articulation

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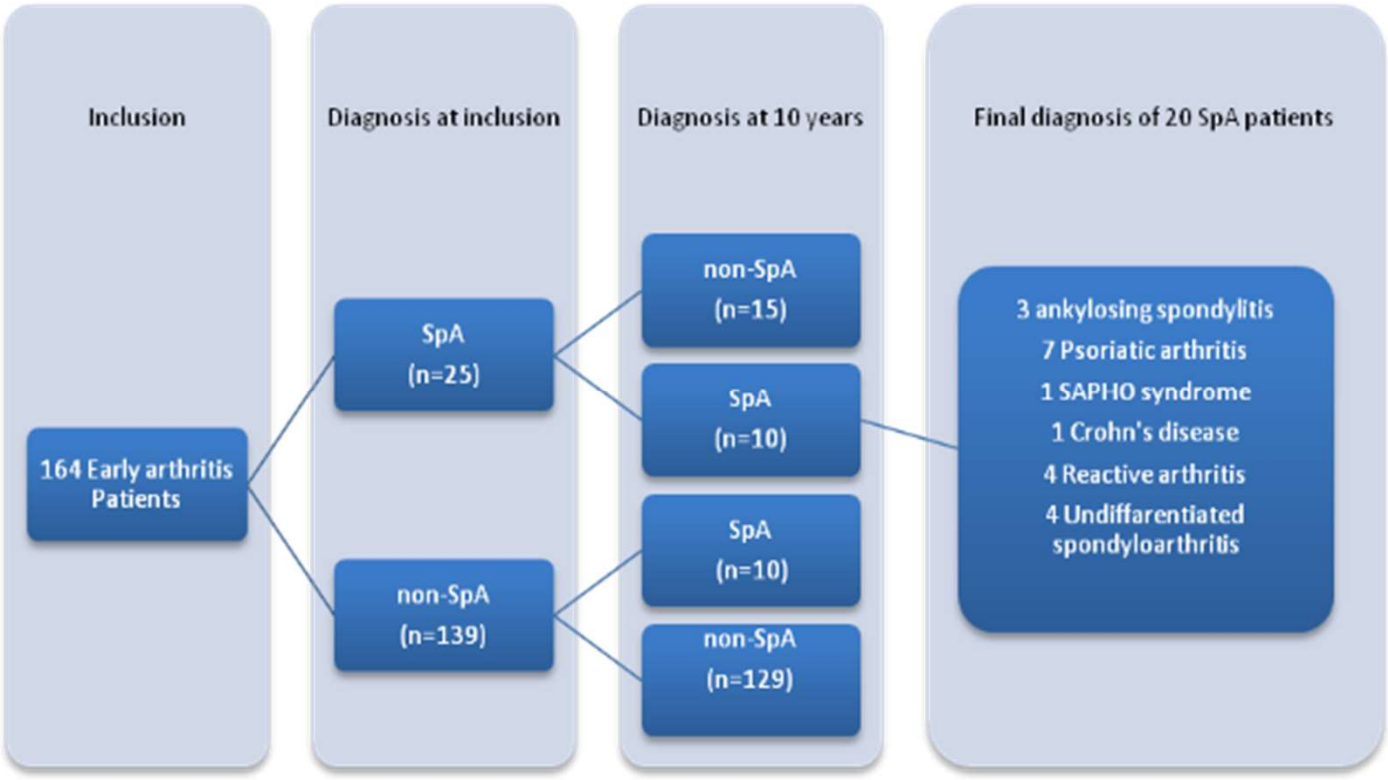
Arthrite réactionnelle= spondyloarthrites?

- Jusqu'au milieu du XXe siècle: rhumatismes inflammatoires = arthrites rhumatoïdes
- Mi XXe siècle
  - Facteurs rhumatoïdes a été à l'origine de la distinction progressive de plusieurs rhumatismes inflammatoires.
  - 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
    - Prévalence 0,30 %
    - Cinq entités: spondylarthrite ankylosante, rhumatisme psoriasique, arthrites réactionnelles, arthrites associées aux maladies inflammatoires chroniques de l'intestin et spondylarthrites indifférenciées.

Un petit nombre d'arthrites sont réactionnelles

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

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

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

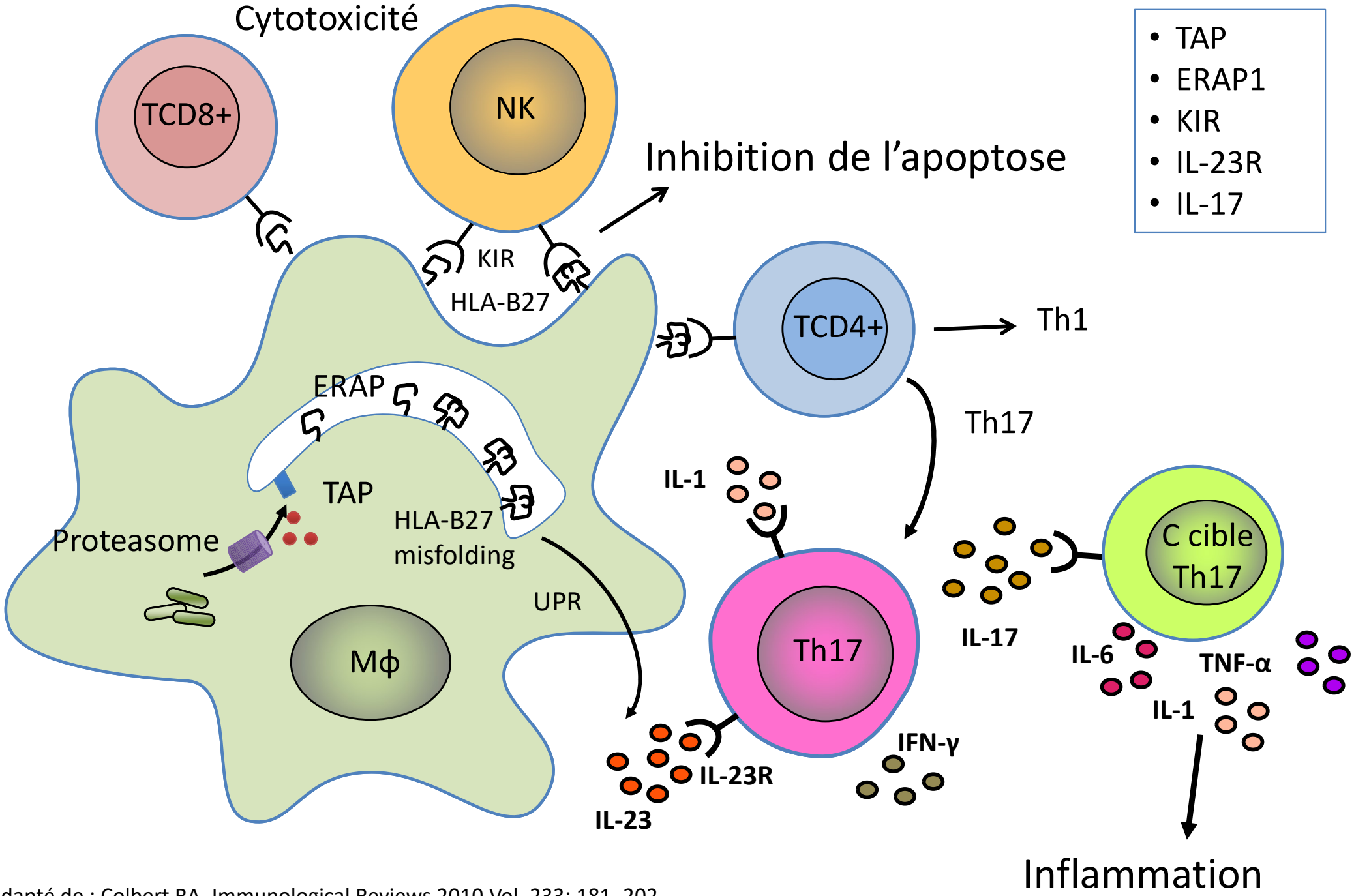
Evolution over thirty years of the profile of inpatients with reactive arthritis in a tertiary rheumatology unit

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

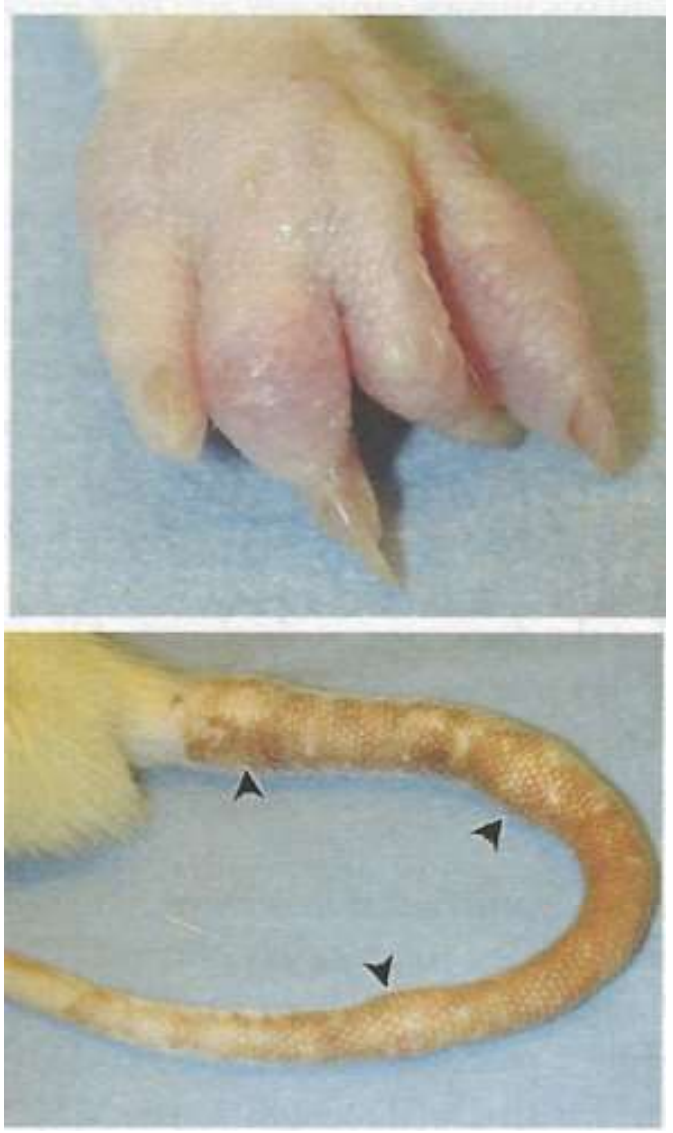
Une part génétique



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

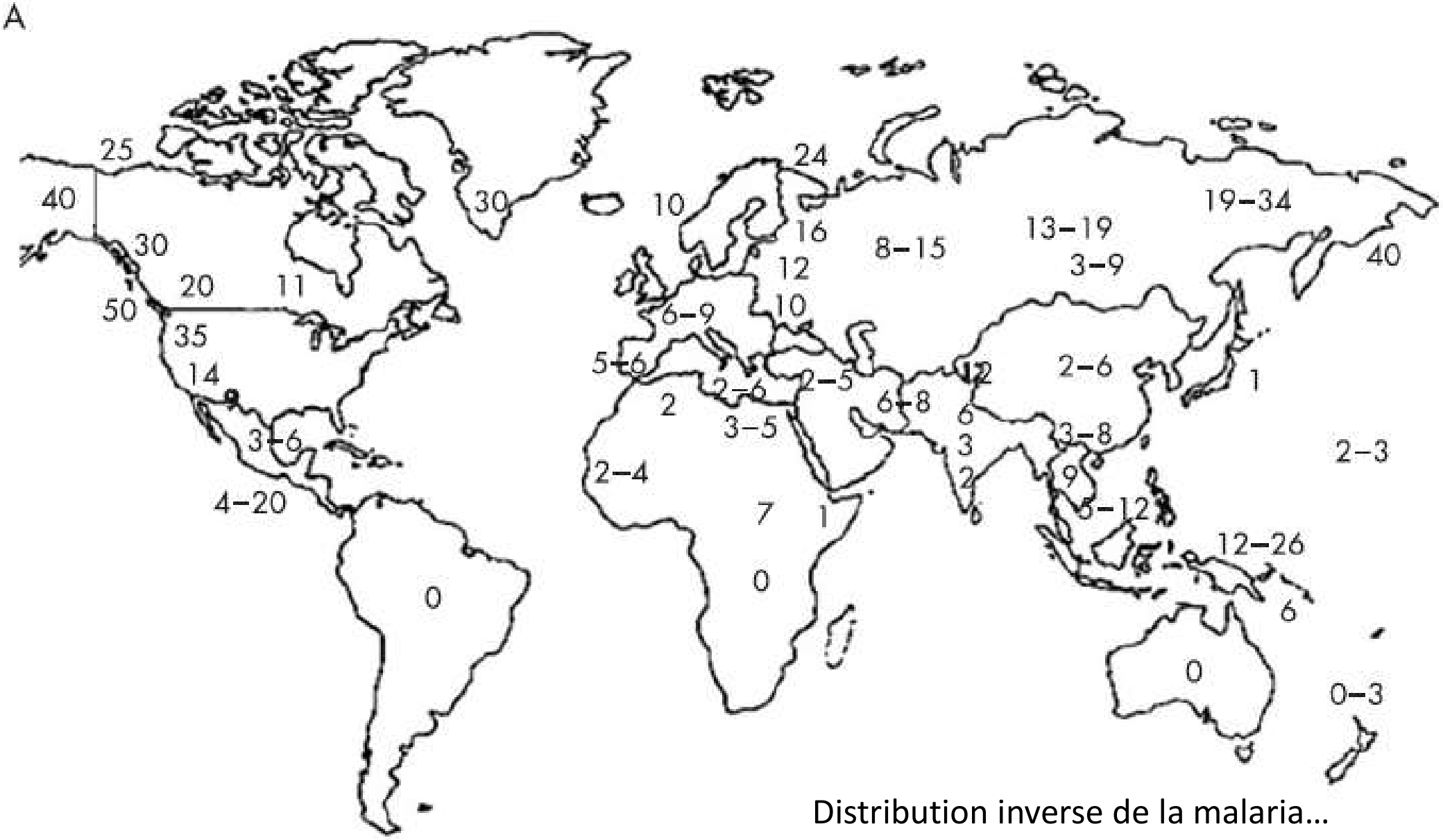
Roddis M. J Immunol. 2004 Jan. 1;172(1):155–161 Colbert RA. Immunol Rev. 2010;233(1):181–202
Taurog JD. Arthritis Rheum 2009;60:1977–1984.18 Hoentjen F. Inflamm Bowel Dis 2007;13:317–324

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

Traitement

- AINS (sans étude)
- Corticoïdes (sans études)
- Sulfasalazine (deux études plutôt négatives)
- Antibiotique (plusieurs études négatives)
- Biologiques

Lien infection-articulation

- Pour la WHO
  - 1- germe dans l'articulation
  - 2- post infectieux avec antigène dans l'articulation
  - 3- Arthrite réactionnelle urogénitale ou digestive
  - 4- Déclenchée par un microbe qui n'est pas, ni ses antigènes, dans l'articulation (ex: post streptococcique)

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.

Conclusion

- L’approche pasteurienne est révolue
- Les germes sont nos amis
- Mais on ne les apprécie pas tous
- Il faut s’adapter à son environnement en fonction de sa génétique
- Heureusement que nous sommes tous différents