PATRAS UNIVERSITY HOSPITAL

Στοχευμενες θεραπειες σε ασθενεις με ρευματικα νοσηματα και ιστορικο καρκινου

Δαούσης Δημήτρης
Αναπλ. Καθηγητής Ρευματολογίας
Ιατρική Σχολή Πανεπιστημίου Πατρών
Προεδρος Ελληνικης Ρευματολογικης Εταιρείας

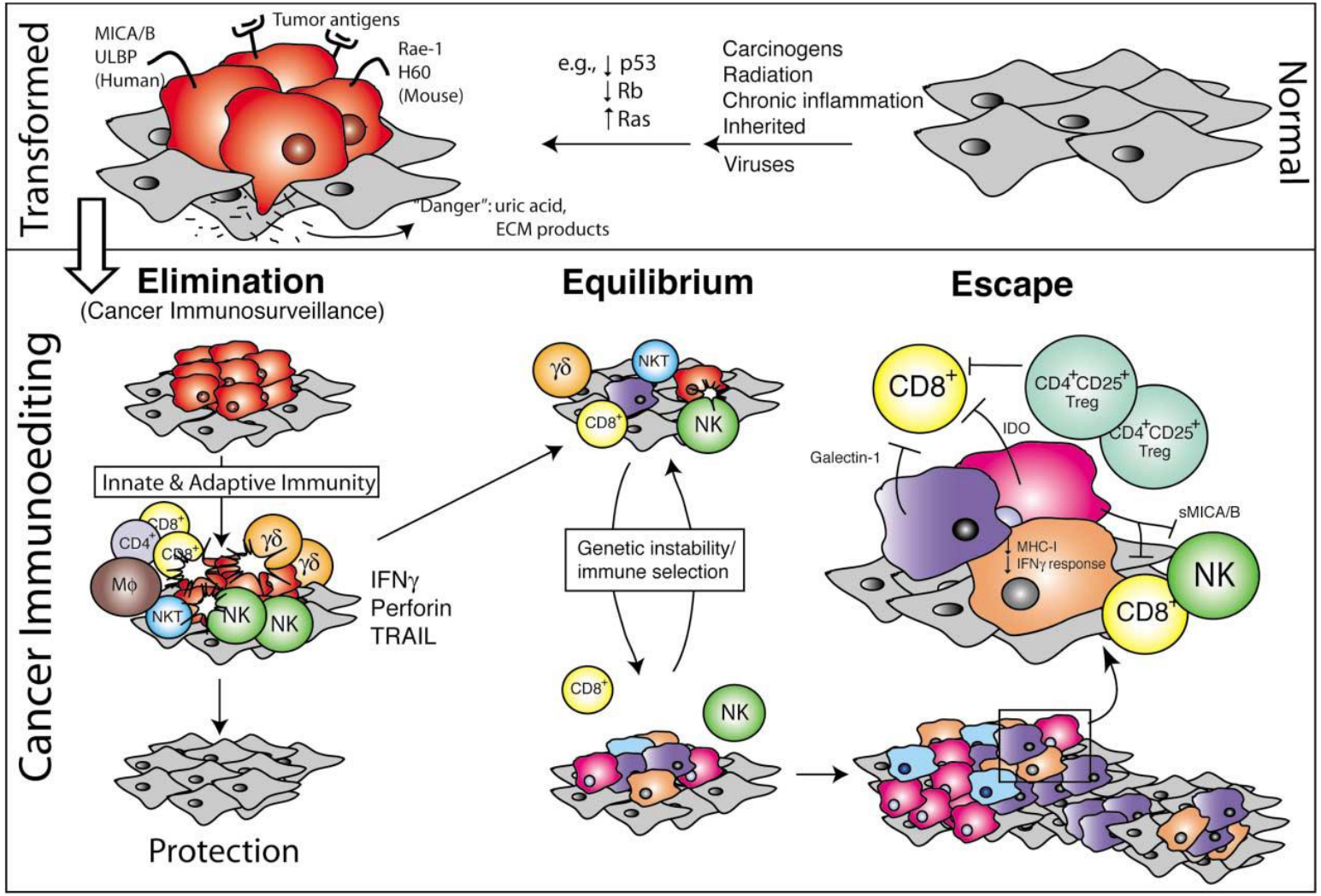
Συνεδριο ΕΠΕΝΜΥ, Πορτο Χελι 2025

Σύγκρουση συμφερόντων

- Τιμητική αμοιβή για ομιλίες και συμμετοχή σε advisory boards από τις εταιρείες UCB, Pfizer, Novartis, BMS, MSD, Janssen, Abbvie,Lilly, Aenorasis, Boehringer, Astra Zeneca, GSK

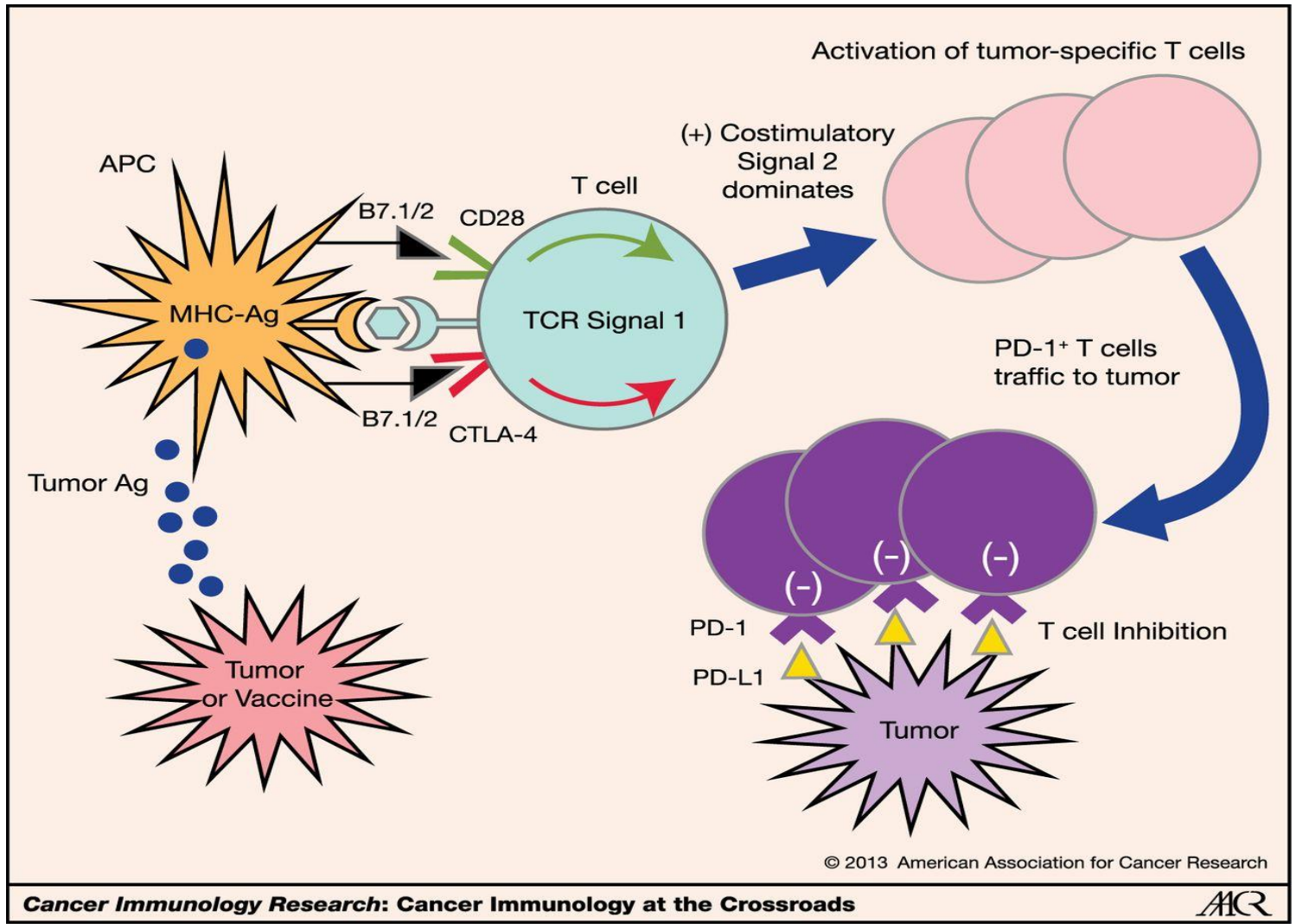
- Σχέση ανοσολογικού συστήματος-καρκίνου
- Σχέση χρόνιας φλεγμονής-καρκίνου
- Ο κίνδυνος για εμφανιση κακοήθειας σε ασθενείς με PA
- Ο κίνδυνος για εμφανιση κακοήθειας με χρήση DMARD
- Ο κίνδυνος για εμφανιση κακοήθειας με χρήση στοχευμένων θεραπειών
- **Χορήγηση στοχευμένων θεραπειών σε ασθενείς με ιστορικό κακοήθειας**

Ανοσολογικό σύστημα και καρκίνος
Μια περίπλοκη, στενή σχέση

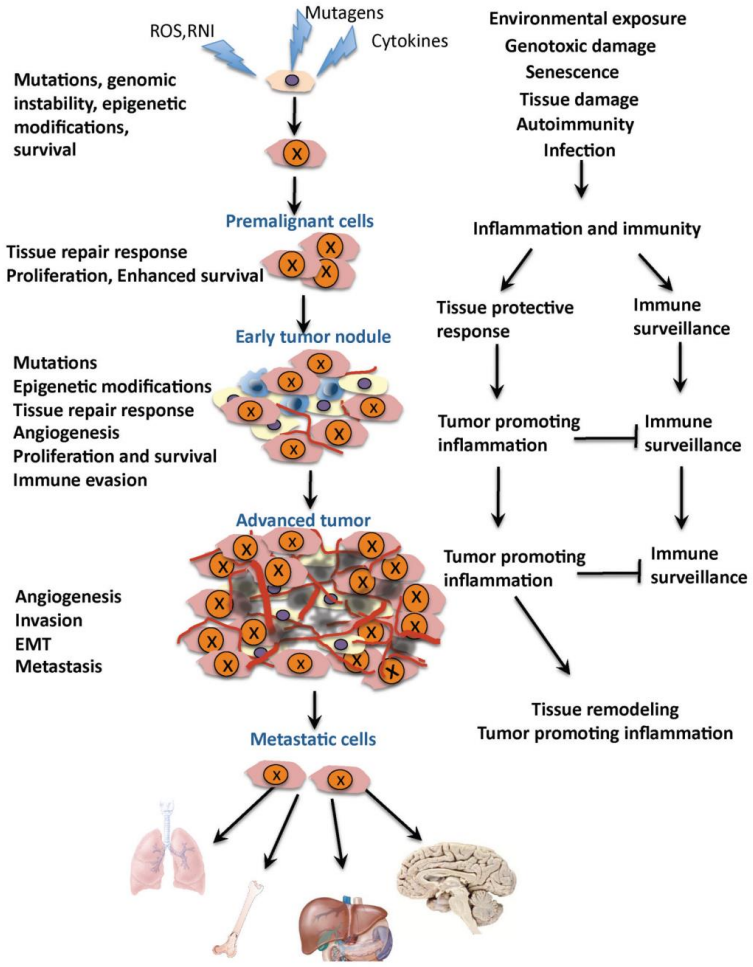


Adv Immunol 2006,90:1

Θα μπορούσαμε να ενισχύσουμε την ανοσολογική απάντηση έναντι του όγκου?
Η εποχή της αντικαρκινικής ανοσοθεραπείας έχει ξεκινήσει



Χρόνια φλεγμονή και καρκίνος…



- Χρόνια φλεγμονή ωε προδιαθεσικός παράγοντας καρκίνου
  - Hepatitis B/C
  - Helicobacter pylori
  - IBD

Published in final edited form as:
Cell. 2010 March 19; 140(6): 883–899. doi:10.1016/j.cell.2010.01.025.

Immunity, Inflammation, and Cancer

Sergei I. Grivennikov[1], **Florian R. Greten**[2], and **Michael Karin**[1]
[1]Laboratory of Gene Regulation and Signal Transduction, Departments of Pharmacology and Pathology, School of Medicine, University of California, San Diego, 9500 Gilman Drive, La Jolla, CA, 92093, USA.
[2]2nd Department of Medicine, Klinikum rechts der Isar, Technical University Munich, Munich, Germany.

Η ΡΑ είναι τυπική χρόνια φλεγμονώδης νόσος ενώ για την θεραπεια της δινουμε ανοκατασταλτικά. Αυξάνεται η πιθανότητα κακοήθειας?

- Ναι αλλά λίγο. Σχετικός κίνδυνος 1.1-1.3
- Η μικρή αυτή αύξηση αφορά κυρίως αιματολογικές κακοήθειες

Arthritis and Rheumatol, 2011, p352
ART 2015 17:212

ARTHRITIS & RHEUMATISM
Vol. 63, No. 2, February 2011, pp 352–358
DOI 10.1002/art.30134

The Risk of Cancer in Patients With Rheumatoid Arthritis

A Nationwide Cohort Study in Taiwan

Yi-Ju Chen,[1] Yun-Ting Chang,[2] Chang-Bi Wang,[3] and Chun-Ying Wu[4]

- Η PA ως νόσος σχετίζεται με αυξημένη πιθανότητα εμφάνισης λεμφώματος

Table 2. SIRs and 95% CIs for cancers, according to age, sex, and duration of followup in Taiwanese patients with rheumatoid arthritis*

Characteristic	All cancers†				Hematologic cancers			
	No. observed	No. expected	SIR	95% CI	No. observed	No. expected	SIR	95% CI
All patients	935	762.19	1.23	1.22–1.23	75	27.35	2.74	2.68–2.81
Women	634	535.40	1.18	1.17–1.19	39	18.90	2.06	2.00–2.13
Men	301	228.13	1.32	1.30–1.33	36	8.23	4.38	4.23–4.52
Age, years								
15–39	43	18.33	2.35	2.28–2.42	2	1.30	1.54	1.33–1.77
40–69	698	442.85	1.58	1.56–1.59	52	14.90	3.49	3.40–3.59
≥70	194	188.66	1.03	1.01–1.04	21	7.22	2.91	2.79–3.04
Followup, years								
<1	160	2.71	58.96	58.13–59.96	14	0.10	139.08	132.76–147.53
1–2	150	8.25	18.19	17.89–18.48	13	0.30	42.67	41.01–45.75
2–4	246	102.85	2.39	2.36–2.42	17	3.71	4.59	4.37–4.81
4–6	165	156.57	1.05	1.04–1.07	18	5.62	3.21	3.06–3.35
6–8	128	165.15	0.78	0.76–0.79	7	5.88	1.19	1.10–1.28
≥8	86	273.97	0.31	0.31–0.32	6	9.72	0.62	0.57–0.67

* SIRs = standardized incidence ratios; 95% CI = 95% confidence interval.
† Includes hematologic cancers.

Table 4. SIRs for hematopoietic malignancies in Taiwanese patients with rheumatoid arthritis*

Cancer type	No. observed	No. expected	SIR	95% CI
All	75	27.35	2.74	2.68–2.81
Leukemia	15	10.12	1.48	1.41–1.56
Hodgkin's lymphoma	1	0.56	1.76	1.45–2.17
Non-Hodgkin's lymphoma and others†	59	16.66	3.54	3.45–3.63

* SIRs = standardized incidence ratios; 95% CI = 95% confidence interval.

Simon et al. Arthritis Research & Therapy (2015) 17:212
DOI 10.1186/s13075-015-0728-9

arthritis research&therapy

RESEARCH ARTICLE Open Access

Incidence of malignancy in adult patients with rheumatoid arthritis: a meta-analysis

Teresa A. Simon[1*], Adam Thompson[1], Kunal K. Gandhi[1], Marc C. Hochberg[2] and Samy Suissa[3]

Overall malignancy

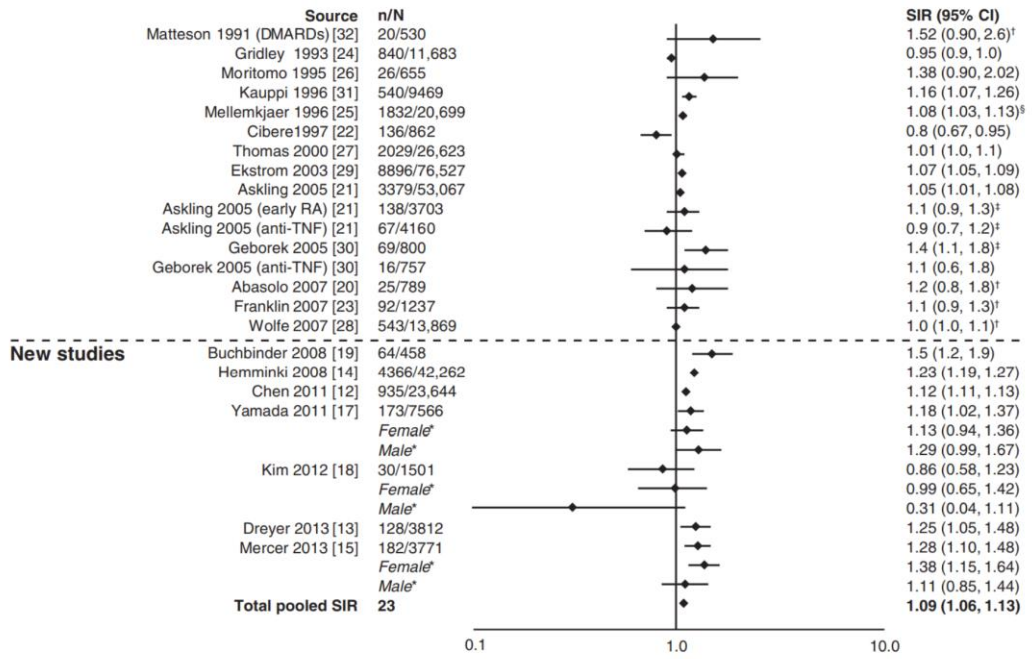


Fig. 2 Relative risk of overall malignancy in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; DMARD, disease-modifying antirheumatic drug; n, number of malignancies; N, population size; RR, relative risk; SIR, standardized incidence ratio; TNF, tumor necrosis factor. *SIRs by sex are not included in the total pooled SIR. †Excluding non-melanoma skin cancer. ‡All solid tumors. §Excluding lymphatic and hematopoietic

lymphoma

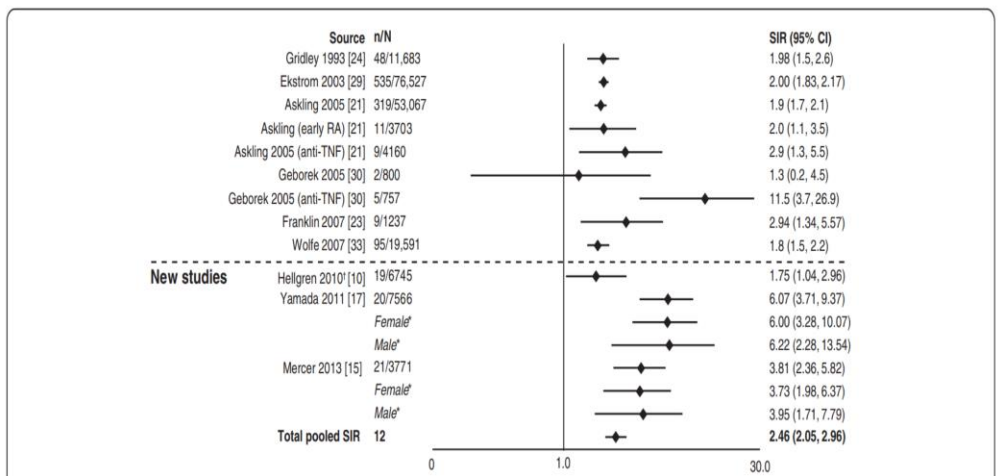


Fig. 3 Relative risk of malignant lymphoma in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; n, number of malignancies; N, population size; OR, odds ratio; SIR, standardized incidence ratio; TNF, tumor necrosis factor. *SIRs by sex are not included in the total pooled SIR. †Reported as odds ratio

Simon et al. Arthritis Research & Therapy (2015) 17:212
DOI 10.1186/s13075-015-0728-9

arthritis research&therapy

RESEARCH ARTICLE Open Access

Incidence of malignancy in adult patients with rheumatoid arthritis: a meta-analysis

Teresa A. Simon[1*], Adam Thompson[1], Kunal K. Gandhi[1], Marc C. Hochberg[2] and Samy Suissa[3]

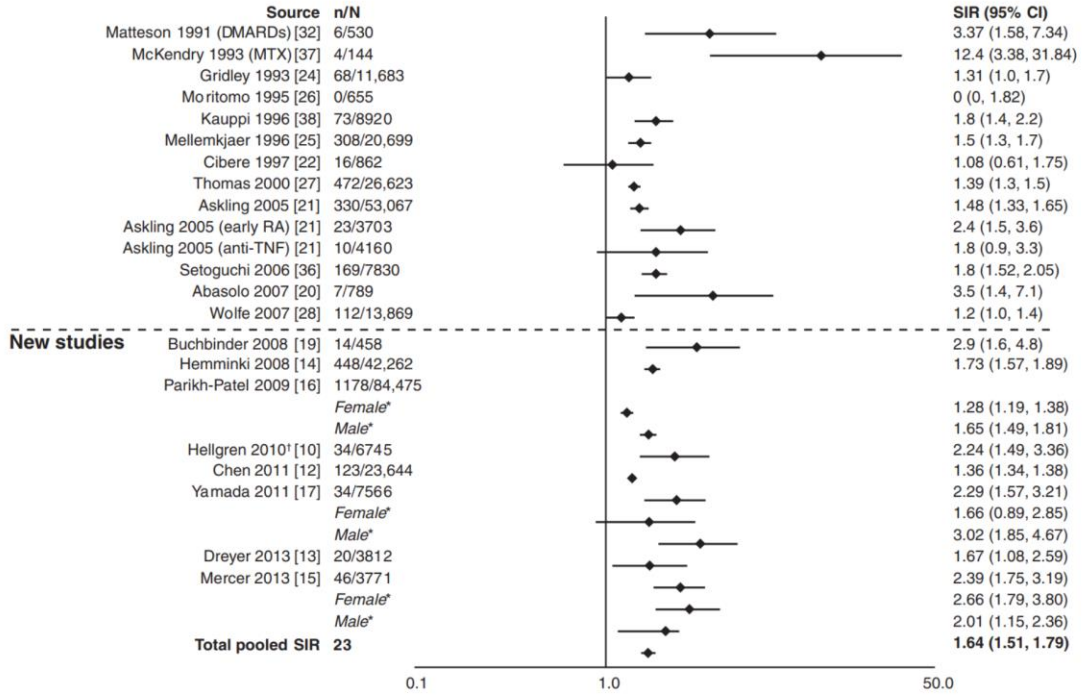


Fig. 6 Relative risk of lung cancer in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; DMARD, disease-modifying antirheumatic drug; MTX, methotrexate; n, number of malignancies; N, population size; SIR, standardized incidence ratio; TNF, tumor necrosis factor. *SIRs by sex are included in total pooled SIR only if overall SIR was not available. †Reported as odds ratio

- Αυξημένη πιθανότητα για καρκίνο πνεύμονα?
- Κοινός προδιαθεσικός παράγοντας το κάπνισμα

Ανοσοτροποποιητικά φάρμακα και καρκίνος

- Τα κλασσικα DMARD θεωρουνται γενικως ασφαλη

Seminars in Arthritis and Rheumatism 43 (2014) 489–497

Contents lists available at ScienceDirect

Seminars in Arthritis and Rheumatism

journal homepage: www.elsevier.com/locate/semarthrit

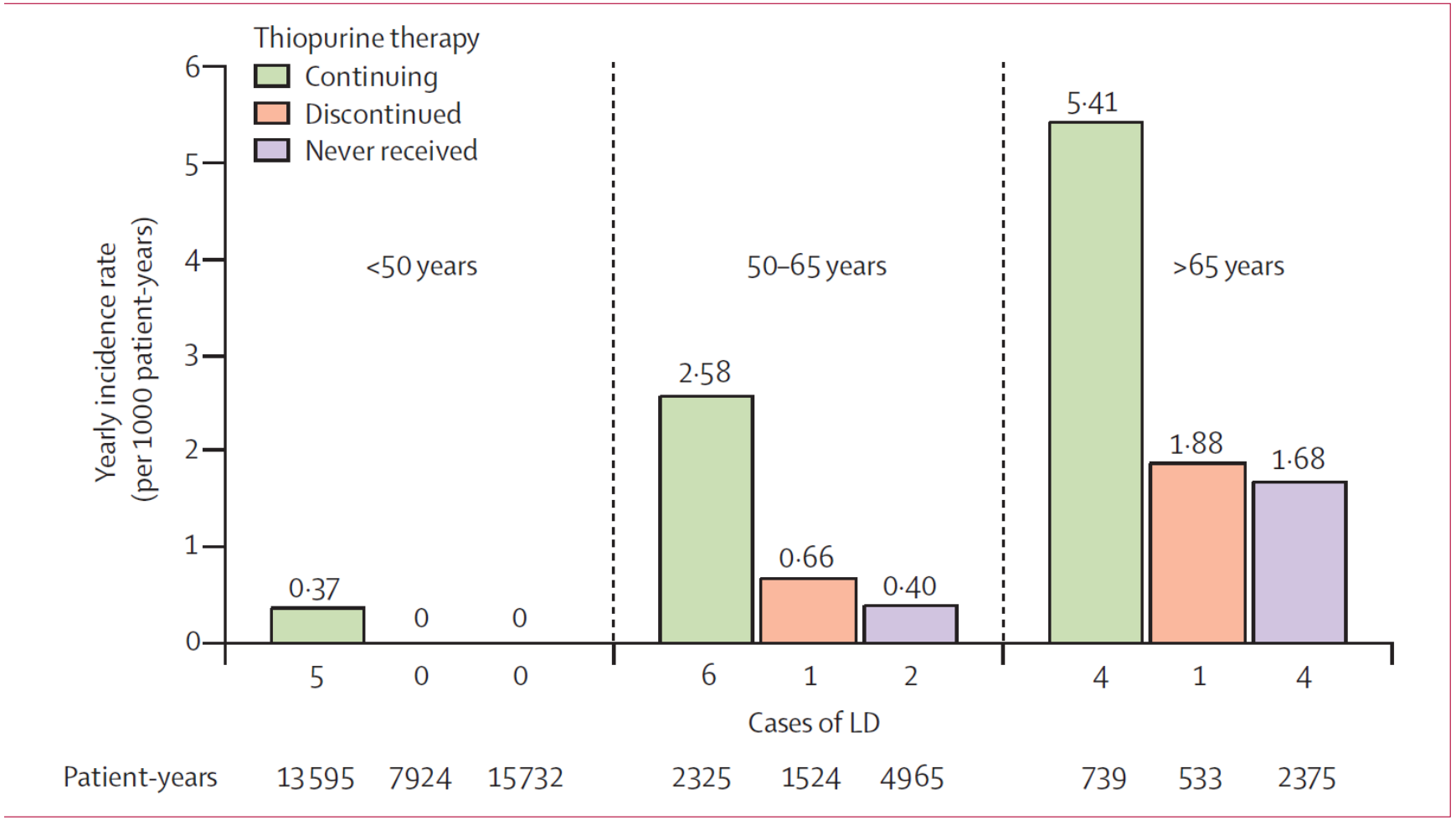
Comparative cancer risk associated with methotrexate, other non-biologic and biologic disease-modifying anti-rheumatic drugs

Daniel H. Solomon, MD, MPH[a,b,*], Joel M. Kremer, MD[c], Mark Fisher, MD[d], Jeffrey R. Curtis, MD, MPH[e], Victoria Furer, MD, MPH[i], Leslie R. Harrold, MD, MPH[f], Marc C. Hochberg, MD, MPH[g], George Reed, PhD[h], Peter Tsao, MSc[a], Jeffrey D. Greenberg, MD, MPH[i]

Lymphoproliferative disorders in patients receiving thiopurines for inflammatory bowel disease: a prospective observational cohort study

Laurent Beaugerie, Nicole Brousse, Anne Marie Bouvier, Jean Frédéric Colombel, Marc Lémann, Jacques Cosnes, Xavier Hébuterne, Antoine Cortot, Yoram Bouhnik, Jean Pierre Gendre, Tabassome Simon, Marc Maynadié, Olivier Hermine, Jean Faivre, Fabrice Carrat, for the CESAME Study Group

AZA



- Στα ρευματικά νοσήματα δεν φαίνεται να προκύπτει κάποιος ιδιαίτερος κίνδυνος

Βιολογικοί παράγοντες και καρκίνος...

- Περισσότερα δεδομένα για τους TNF αναστολείς
- In vitro ο TNF καταστρέφει καρκινικά κύτταρα
- In vivo όμως τα πράγματα είναι πιο περίπλοκα....

MENU nature REVIEWS CANCER

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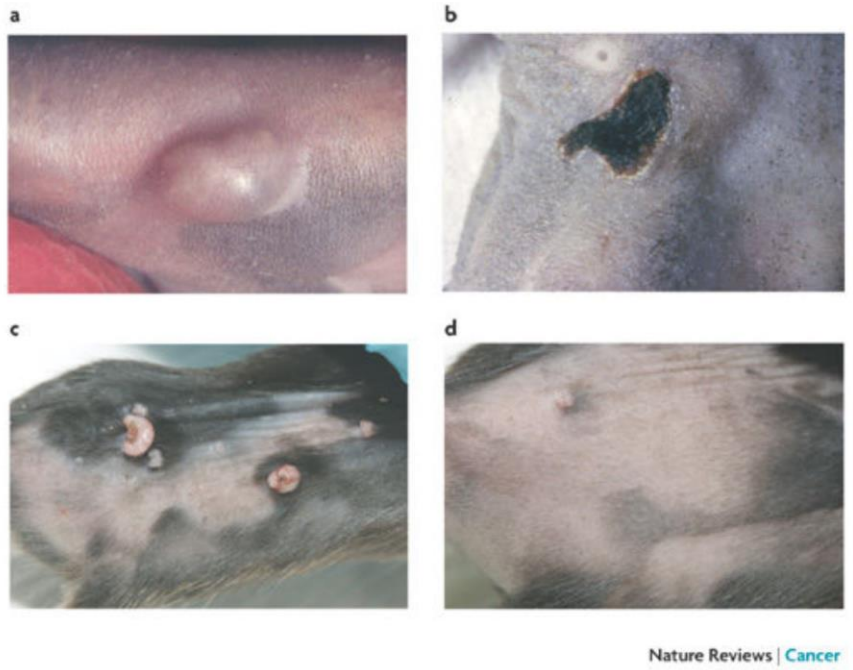
Altmetric: 6 Citations: 751 More detail »

Timeline

Tumour necrosis factor and cancer

Frances Balkwill

Figure 2: The pro- and anti-tumour actions of tumour necrosis factor (TNF) in mouse models of cancer.



Direct TNF administration to tumor

TNF-/- mice resistant to tumor development

Nature Rev Cancer, 2009, 361

The safety of anti-tumour necrosis factor treatments in rheumatoid arthritis: meta and exposure-adjusted pooled analyses of serious adverse events

J P Leombruno,[1] T R Einarson,[1] E C Keystone[2]

Table 7 Rates of adverse events in placebo and recommended dose anti-TNF groups of randomised controlled trials in RA

Outcome	Unadjusted event rate per 1000 subjects (controlled portions of trials only)		Meta-analysis OR (95% CI)	Exposure-adjusted event rate per 1000 subject/years (controlled portions of trials only)		Meta-analysis RR (95% CI)	Exposure-adjusted event rate per 1000 subject/years (controlled and uncontrolled portions of trial)		Simple pooled RR (95% CI)
	Placebo	Biological		Placebo	Biological		Placebo	Biological	
Death	4.1	5.6	1.39 (0.74 to 2.62)	5.2	6.0	1.23 (0.66 to 2.29)	5.2	5.9	1.13 (0.57 to 2.27)
Serious adverse event	118.0	139.3	1.11 (0.94 to 1.32)	177.0	164.6	0.94 (0.77 to 1.15)H	177.0	177.4	1.00 (0.87 to 1.16)
Serious infection	27.5	32.5	1.21 (0.89 to 1.63)	34.0	35.7	1.07 (0.81 to 1.43)	34.0	36.7	1.08 (0.81 to 1.43)
Lymphoma	0.4	1.0	1.26 (0.52 to 3.06)	0.5	1.2	1.26 (0.53 to 3.01)	0.5	1.2	2.41 (0.37 to 15.59)
Non-cutaneous cancers and melanoma	3.7	5.4	1.31 (0.69 to 2.48)	5.1	6.4	1.21 (0.63 to 2.32)	5.1	7.1	1.40 (0.69 to 2.83)
Non-melanoma cutaneous cancer	1.4	2.1	1.27 (0.67 to 2.42)	1.8	1.9	1.01 (0.43 to 2.38)	1.7	2.4	1.41 (0.41 to 4.91)

H, Evidence of heterogeneity with fixed effects method, random effects analysis shown. OR, odds ratio; RA, rheumatoid arthritis; RR, risk ratio; TNF, tumour necrosis factor.

CONCISE REPORT

Incidences of overall and site specific cancers in TNFα inhibitor treated patients with rheumatoid arthritis and other arthritides – a follow-up study from the DANBIO Registry

Lene Dreyer,[1] Lene Mellemkjær,[2] Anne Rødgaard Andersen,[3] Philip Bennett,[4] Uta Engling Poulsen,[5] Torkell Juulsgaard Ellingsen,[6] Torben Høiland Hansen,[7] Dorte Vendelbo Jensen,[8] Louise Linde,[3] Hanne Merete Lindegaard,[9] Anne Gitte Rasmussen Loft,[10] Henrik Nordin,[11] Emina Omerovic,[12] Claus Rasmussen,[13] Annette Schlemmer,[14] Ulrik Tarp,[15] Merete Lund Hetland[3 16]

- Τα registries πιθανά δίνουν καλύτερες πληροφορίες
  - Περισσότεροι ασθενείς
  - Μακροχρόνια παρακολούθηση

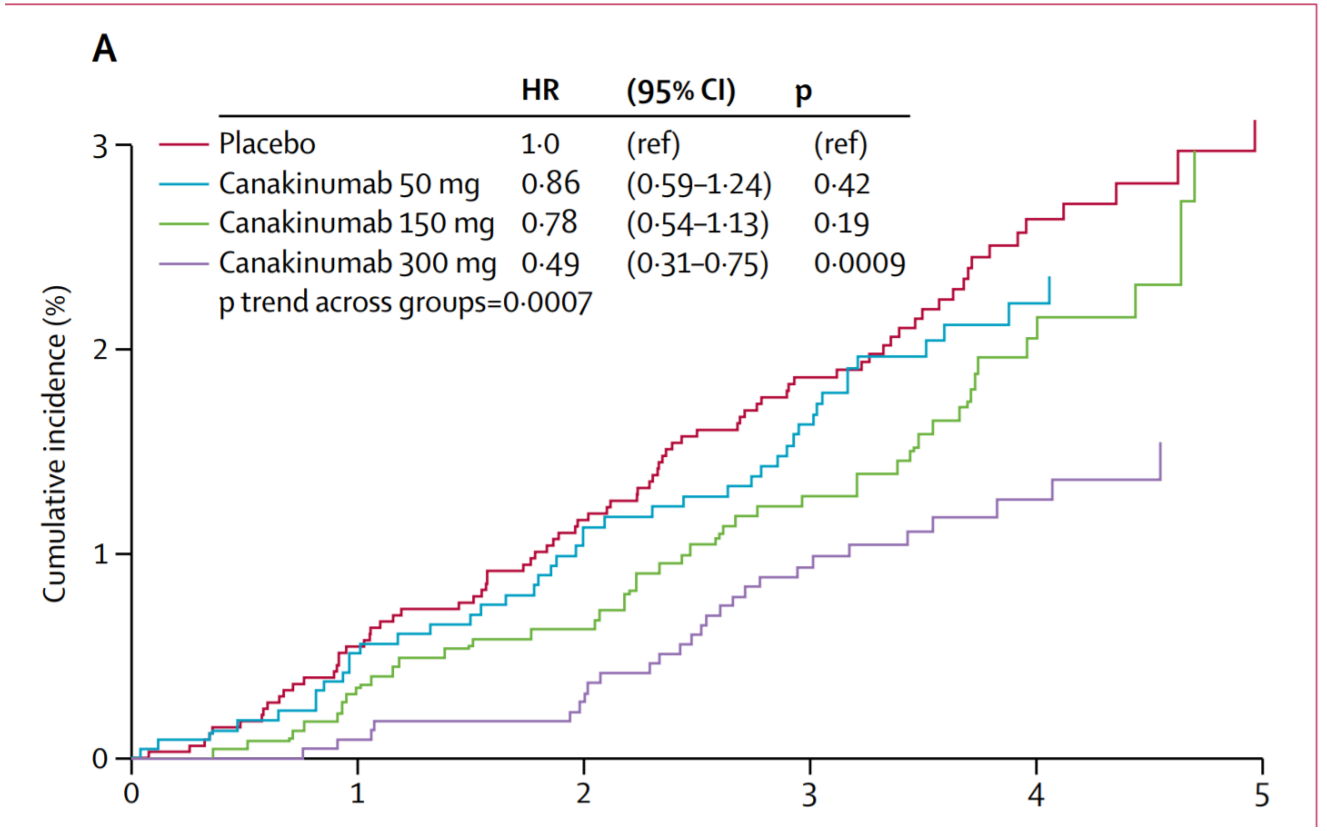
Δεδομένα από αρχεία...

Variable	No of cancers among treated†	TNF-I treated versus non-treated HR‡ (95% CI)	p Value*
Ever TNF-I treatment			
Overall effect	152	1.02 (0.80 to 1.30)	
Plus adjustment for HAQ§		0.95 (0.74 to 1.22)	
Plus adjustment for CRP§		0.99 (0.77 to 1.26)	
Plus adjustment for DAS28§		0.96 (0.74 to 1.24)	
Men	48	0.83 (0.55 to 1.26)	p=0.24
Women	104	1.13 (0.83 to 1.53)	
Time since treatment initiation, years			
<1	41	1.04 (0.72 to 1.50)	
1–4	97	1.03 (0.79 to 1.35)	p=0.86
5+	14	0.88 (0.51 to 1.54)	
1+	111	1.01 (0.78 to 1.30)	
Cumulative duration of treatment, years			
<1	43	1.04 (0.73 to 1.48)	
1–2	39	1.19 (0.83 to 1.71)	p=0.69
2–3	29	1.09 (0.72 to 1.63)	
4+	41	0.86 (0.60 to 1.22)	
Age at treatment start, years			
<50	12	0.83 (0.38 to 1.82)	
50–64	69	0.97 (0.68 to 1.37)	p=0.76
≥65	71	1.10 (0.80 to 1.50)	

Effect of interleukin-1β inhibition with canakinumab on incident lung cancer in patients with atherosclerosis: exploratory results from a randomised, double-blind, placebo-controlled trial

Paul M Ridker, Jean G MacFadyen, Tom Thuren, Brendan M Everett, Peter Libby, Robert J Glynn*, on behalf of the CANTOS Trial Group†*

- Η αναστολή της IL-1 μειώνει την πιθανότητα καρκίνου πνεύμονα!



Schiff et al. Arthritis Research & Therapy 2011, 13:R141
http://arthritis-research.com/content/13/5/R141

arthritis research&therapy

RESEARCH ARTICLE Open Access

Integrated safety in tocilizumab clinical trials

Michael H Schiff[1*], Joel M Kremer[2], Angelika Jahreis[3], Emma Vernon[4], John D Isaacs[5] and Ronald F van Vollenhoven[6]

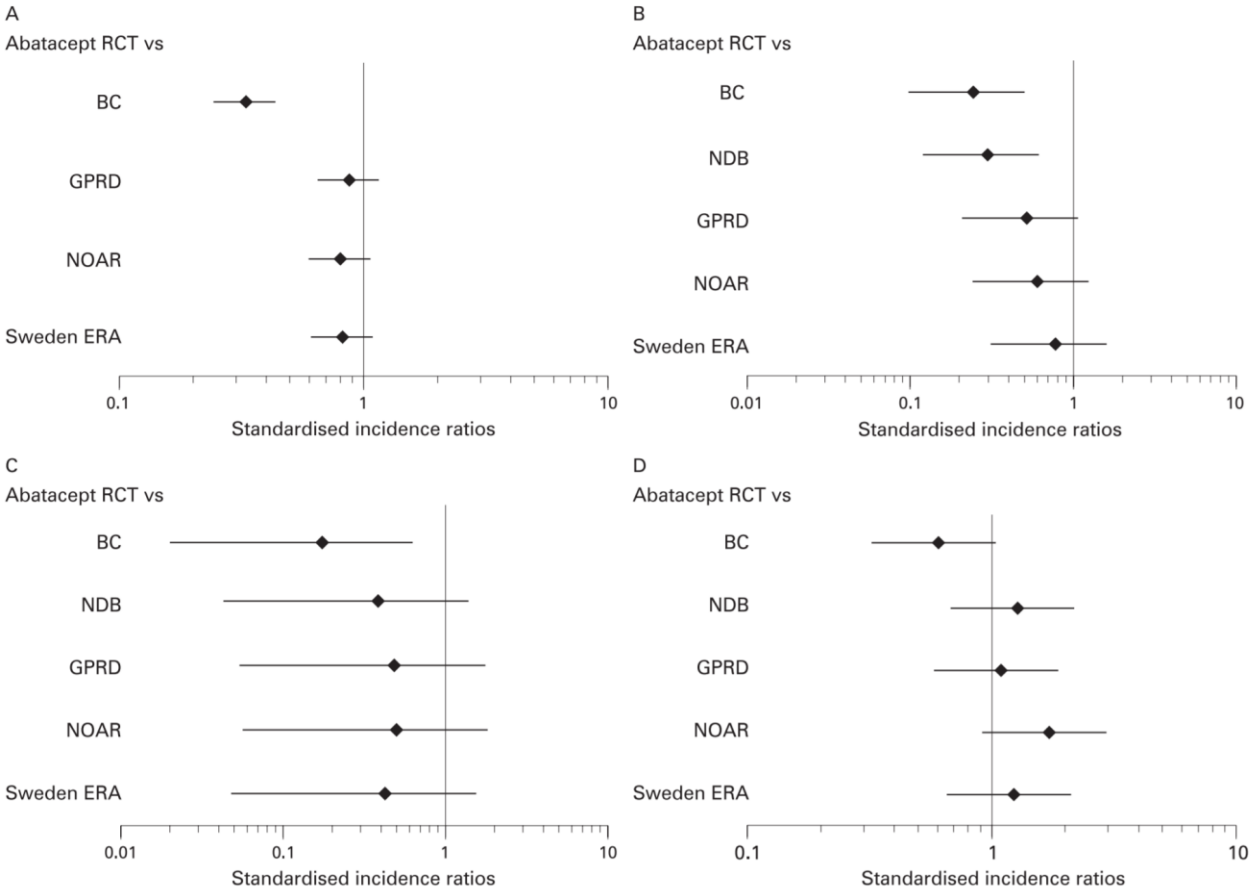
Table 1 Serious adverse events reported at a rate of ≥ 0.3 per 100 patient-years in any group (all-control population)

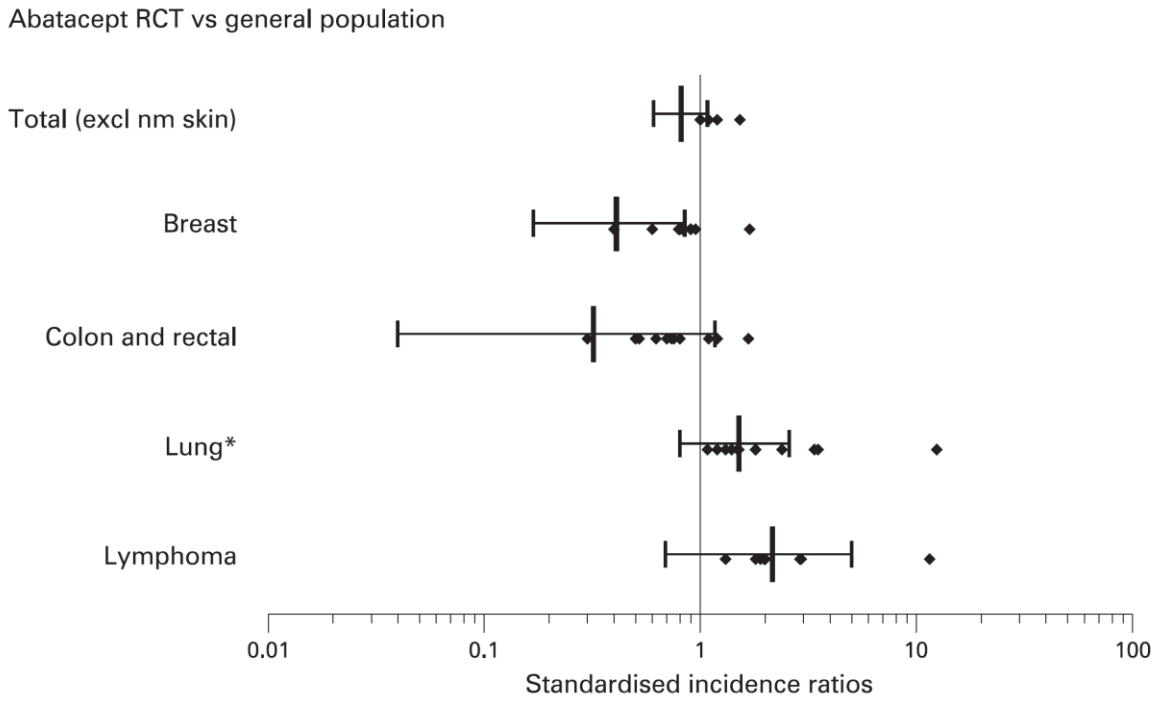
	All-control population *n* = 4,199		
	Control *n* = 1,555	**Tocilizumab 4 mg/kg + DMARDs *n* = 774**	**Tocilizumab 8 mg/kg + DMARDs *n* = 1,870**
Rate per 100 PY (number of events)			
Pneumonia	0.6 (5)	0.7 (4)	0.9 (11)
Cellulitis	0.2 (2)	–	0.9 (11)
Gastroenteritis	0.2 (2)	0.5 (3)	0.1 (1)
Urinary tract infection	0.5 (4)	0.2 (1)	0.1 (1)
Sepsis	0.1 (1)	0.4 (2)	0.2 (2)
Herpes zoster	0.1 (1)	–	0.3 (4)
Fall	0.1 (1)	–	0.3 (4)
Pulmonary embolism	0.2 (2)	–	0.3 (3)
Basal cell carcinoma	0.1 (1)	0.4 (2)	0.1 (1)
Spinal compression fracture	0.1 (1)	–	0.3 (3)
Coronary artery disease	–	0.2 (1)	0.3 (3)
Back pain	0.1 (1)	–	0.3 (3)
Rheumatoid arthritis	0.4 (3)	–	–
Gastroenteritis viral	0.1 (1)	0.4 (2)	–
Prostate cancer	0.1 (1)	0.4 (2)	–
Neutropenia	–	0.4 (2)	0.1 (1)
Syncope	–	0.4 (2)	–
Tendon rupture	–	0.4 (2)	–
Interstitial lung disease	–	0.4 (2)	–
Anaphylactic reaction	–	0.4 (2)	–

DMARD, disease-modifying antirheumatic drug; PY, patient-years.

Malignancies in the rheumatoid arthritis abatacept clinical development programme: an epidemiological assessment

T A Simon,[1] A L Smitten,[1] J Franklin,[2] J Askling,[3] D Lacaille,[4] F Wolfe,[5] M C Hochberg,[6] K Qi,[7] S Suissa[8]





- A Total
- B Breast
- C Colorectal
- D Lung

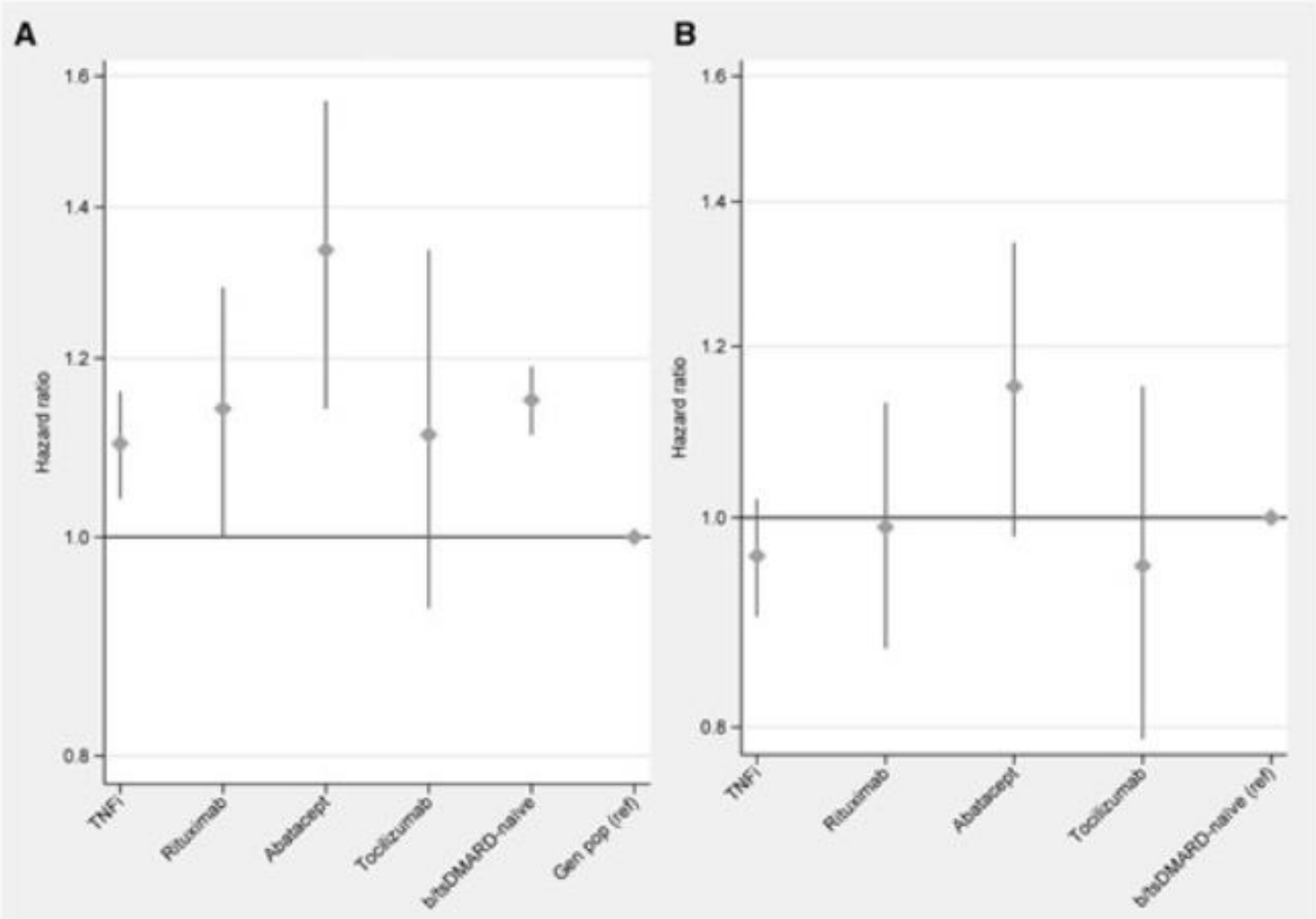
JOURNAL ARTICLE

Short- and longer-term cancer risks with biologic and targeted synthetic disease-modifying antirheumatic drugs as used against rheumatoid arthritis in clinical practice

Viking Huss ✉, Hannah Bower, Hjalmar Wadström, Thomas Frisell, Johan Askling, the ARTIS group Author Notes

Τα νεότερα δεδομενα...

In each of the RA treatment cohorts vs. the general population.



n bDMARD-treated RA vs. bDMARD-naïve RA.

Το rituximab?

- Πρόκειται ουσιαστικά για αντικαρκινικό φάρμακο
- Υπηρχε τάση προτίμησης RTX σε ασθενείς με ιστορικό καρκίνου, χωρίς όμως να υπάρχουν ισχυρές αποδείξεις

Longterm Safety of Rituximab: Final Report of the Rheumatoid Arthritis Global Clinical Trial Program over 11 Years

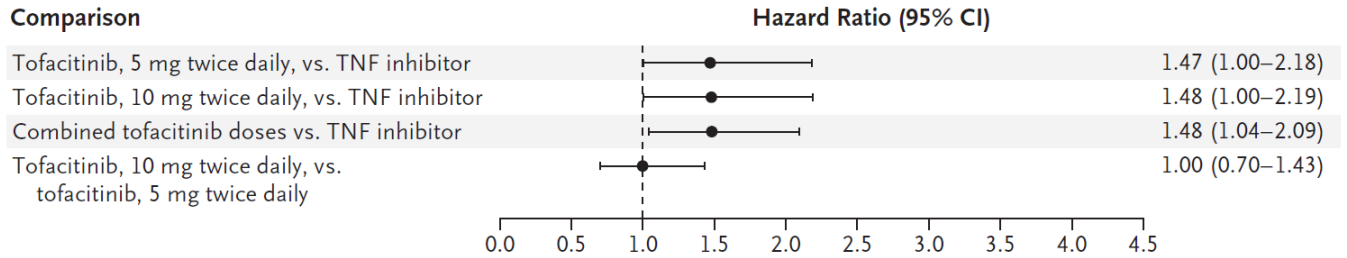
Ronald F. van Vollenhoven, Roy M. Fleischmann, Daniel E. Furst, Stuart Lacey, and Patricia B. Lehane

The NEW ENGLAND JOURNAL of MEDICINE

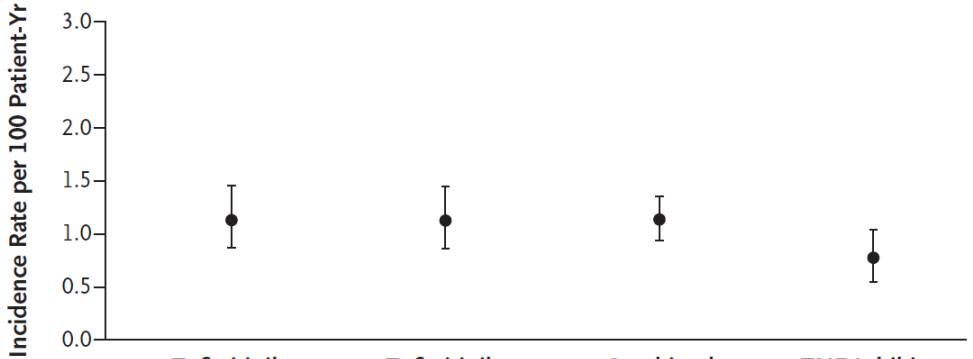
ORIGINAL ARTICLE

Cardiovascular and Cancer Risk with Tofacitinib in Rheumatoid Arthritis

A Hazard Ratio for Cancers, Excluding NMSC



B Incidence Rate for Cancers, Excluding NMSC

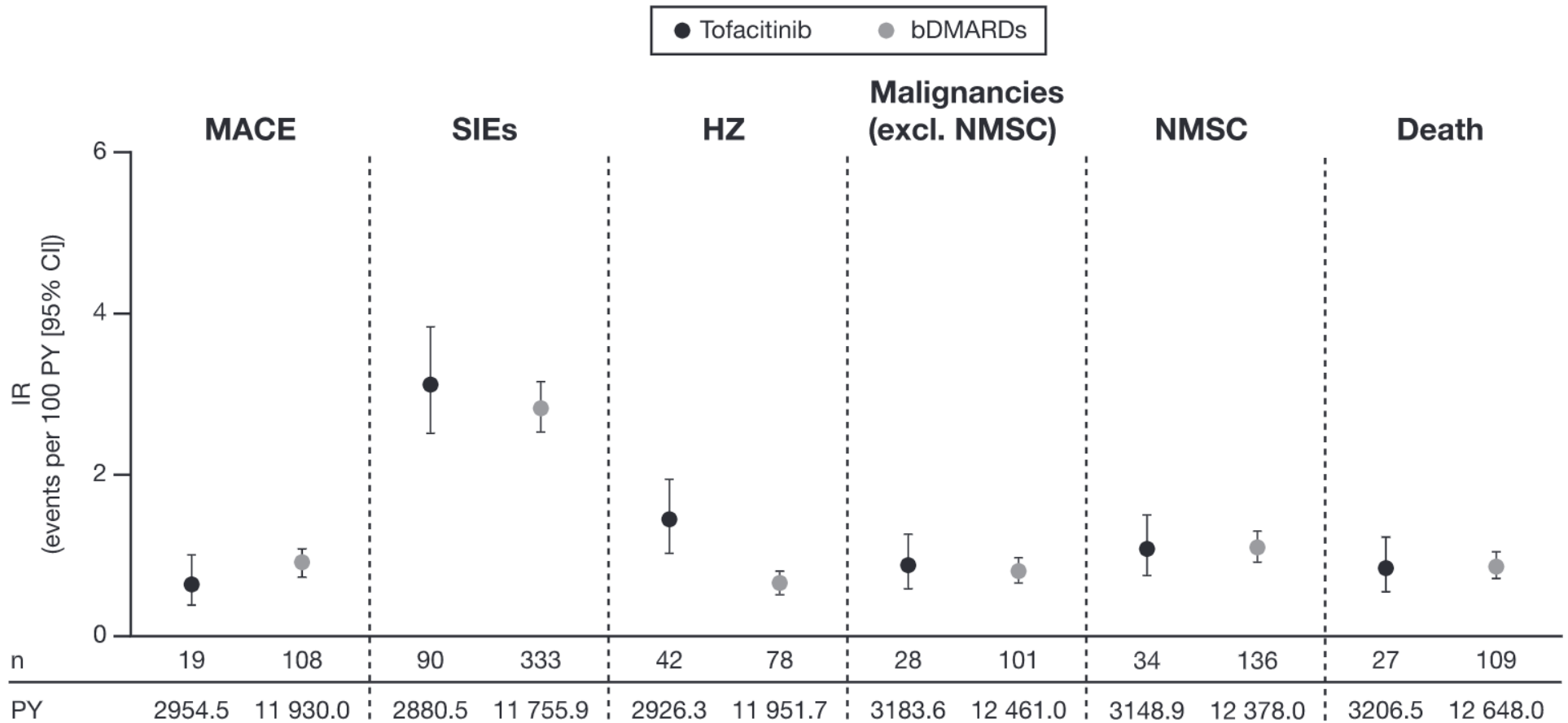


	Tofacitinib, 5 mg Twice Daily	Tofacitinib, 10 mg Twice Daily	Combined Tofacitinib Doses	TNF Inhibitor
No. of Patients with First Event/Total No. (%)	62/1455 (4.3)	60/1456 (4.1)	122/2911 (4.2)	42/1451 (2.9)
No. of Patient-Yr	5491.48	5311.71	10,803.19	5482.30
Incidence Rate per 100 Patient-Yr (95% CI)	1.13 (0.87–1.45)	1.13 (0.86–1.45)	1.13 (0.94–1.35)	0.77 (0.55–1.04)
NNH (patient-yr) vs. TNF Inhibitor	276	275	—	—
NNH (over 5-yr period) vs. TNF Inhibitor	55	55	—	—

ACR Open Rheumatology
Vol. 3, No. 3, March 2021, pp 173–184
DOI 10.1002/acr2.11232

American College of Rheumatology
Empowering Rheumatology Professionals

Postapproval Comparative Safety Study of Tofacitinib and Biological Disease-Modifying Antirheumatic Drugs: 5-Year Results from a United States–Based Rheumatoid Arthritis Registry



Characteristics of

Cancer

Active inflammatory arthritis

Individualized risk of cancer recurrence

Risk of complications associated with undertreated inflammatory disease activity

Specialist caring for cancer

Patient

Rheumatologist

Shared decision

No initiation of a targeted therapy

Initiation **without delay** of a targeted therapy

Μπορούμε να δώσουμε anti-TNF σε ασθενείς με ιστορικό κακοήθειας?

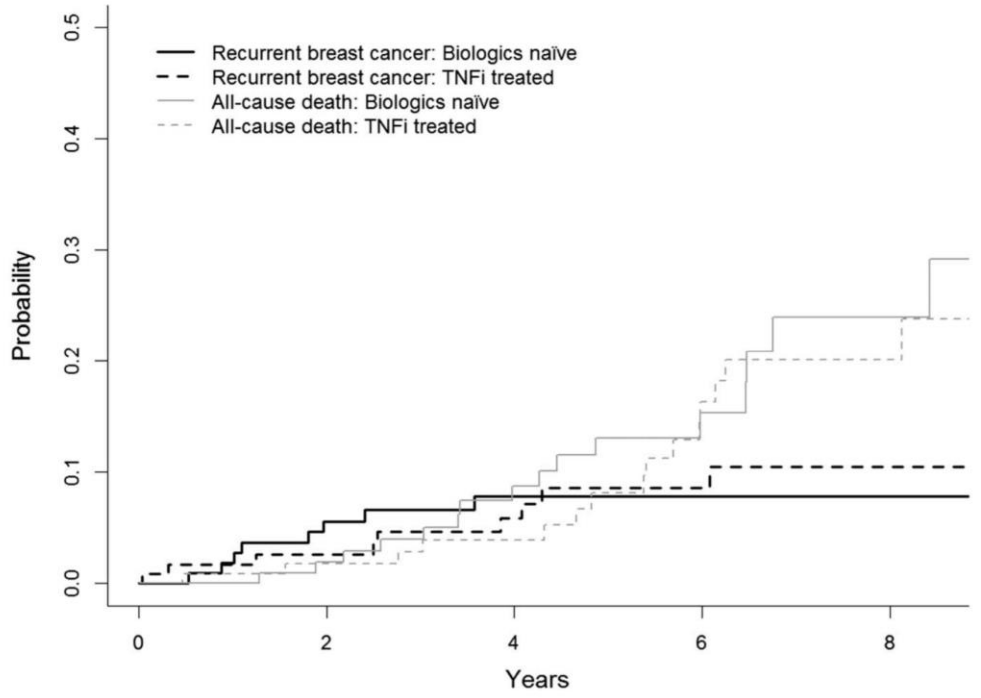
Clinical and epidemiological research

EXTENDED REPORT

TNF inhibitor therapy and risk of breast cancer recurrence in patients with rheumatoid arthritis: a nationwide cohort study

Pauline Raaschou,[1,2] Thomas Frisell,[1] Johan Askling,[1,3] for the ARTIS Study Group

- Η χρήση TNF αναστολέων σε ασθενείς με ιστορικό καρκίνου μαστού δεν φαίνεται να αύξησε τον κίνδυνο υποτροπής (χορήγηση μετά από 9.4 έτη από την διαγνωση)



RHEUMATOLOGY

Rheumatology 2020;59:930–939
doi:10.1093/rheumatology/kez475
Advance Access publication 17 October 2019

Systematic Review and Meta-analysis

A meta-analysis of biologic therapies on risk of new or recurrent cancer in patients with rheumatoid arthritis and a prior malignancy

FIG. 2 Relative risk of cancer recurrence between biologic and csDMARDs

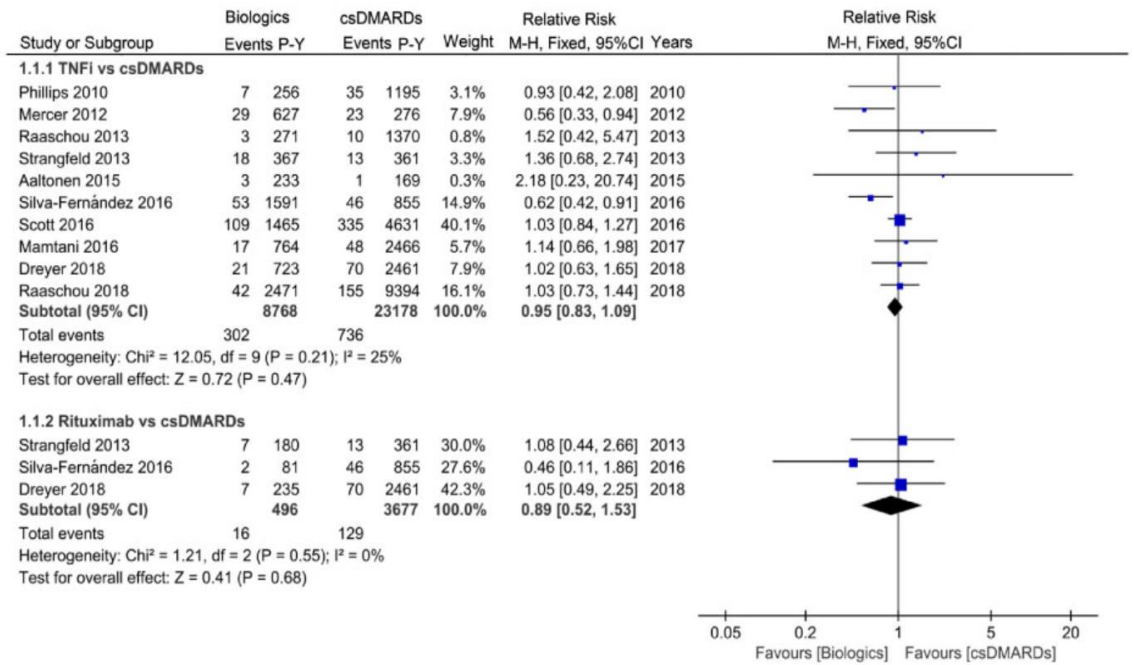
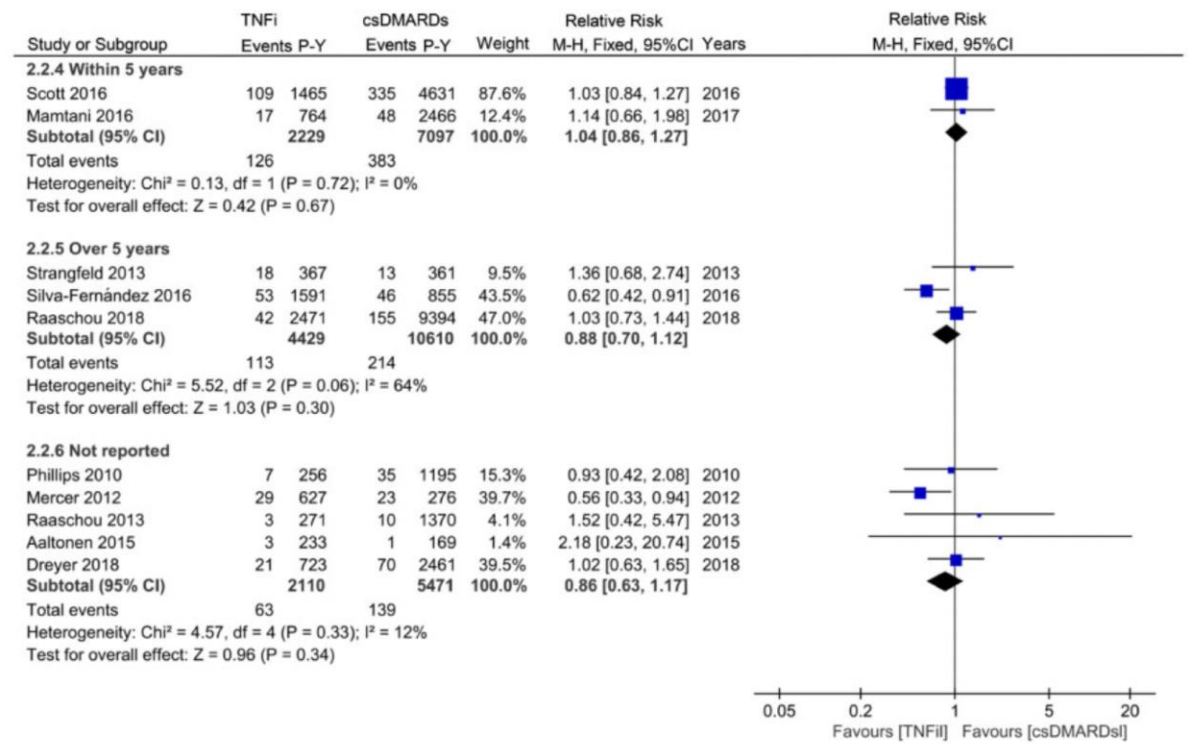


FIG. 4 Relative risk of cancer recurrence between TNFi and csDMARDs, stratified by intervals between prior cancer and TNFi (re-)initiation



- **Melanoma skin cancer** – In patients with a history of melanoma, we use conventional DMARDs over biologic DMARDs or JAK inhibitors. Approaches including monoclonal antibody treatments that activate T cells have shown benefit in treating melanoma (see "Systemic treatment of metastatic melanoma lacking a BRAF mutation"); therefore, some clinicians avoid the use of abatacept in patients with a prior history of melanoma [85]. Routine skin cancer surveillance is indicated. (See "Screening for melanoma in adults and adolescents".)

- **History of lymphoproliferative disorder** – In patients with a history of a lymphoproliferative disorder, we prefer conventional DMARDs, and if a biologic agent is needed, the first choice would be rituximab, given its use in the treatment of lymphoproliferative disorders and a lack o evidence for increased cancer risk with its use.

- **Solid organ malignancy** – In patients with a treated solid organ malignancy within the past five years, we use conventional DMARDs over biologic DMARDs. If a biologic agent is needed, the first choice would be rituximab, given the lack of evidence for increased cancer risk with its use.

In patients who are more than five years out from a treated solid organ malignancy, excluding melanoma, RA treatment is no different than from those without malignancy.

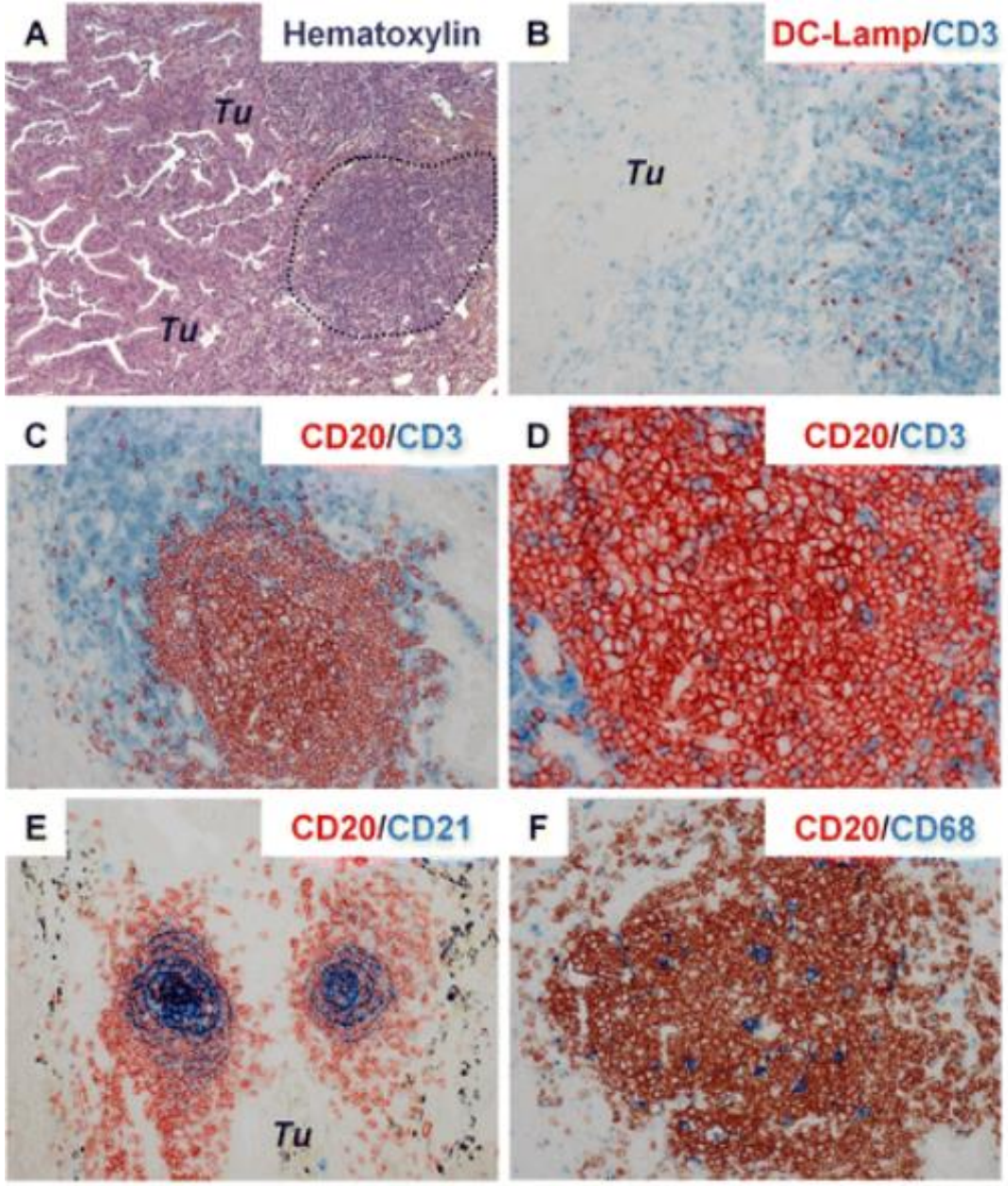
American Journal of Respiratory and Critical Care Medicine

Presence of B Cells in Tertiary Lymphoid Structures Is Associated with a Protective Immunity in Patients with Lung Cancer

Claire Germain [1,2,3], Sacha Gnjatic [4,5], Fella Tamzalit [1,2,3], Samantha Knockaert [1,2,3], Romain Remark [1,2,3], Jérémy Goc [1,2,3], Alice Lepelley [1,2,3], Etienne Becht [1,2,3], Sandrine Katsahian [6,7], Geoffray Bizouard [6], Pierre Validire [1,8],

What This Study Adds to the Field

Here, we demonstrate that B cells organized into tertiary lymphoid structures exhibit features of an ongoing humoral immune response, and that their high density is associated with the long-term survival of patients with non–small cell lung cancer. The presence of both types of antigen-presenting cells, mature dendritic cells and B cells, in tertiary lymphoid structures strongly predicts the outcome of patients. The low density of both follicular B cells and mature dendritic cells allows the identification of patients at high risk of poor survival.



Clinical/translational cancer immunotherapy
Original research

Modeling resistance of colorectal peritoneal metastases to immune checkpoint blockade in humanized mice

Emre Küçükköse [1], Balthasar A Heesters [2], Julien Villaudy [3, 4], André Verheem [1], Madalina Cercel [4], Susan van Hal [4], Sylvia F Boj [5], Inne H M Borel Rinkes [1], Cornelis J A Punt [6], Jeanine M L Roodhart [1, 7], Jamila Laoukili [1], Miriam Koopman [7], Hergen Spits [4, 8] and Onno Kranenburg [1, 9]

WHAT THIS STUDY ADDS

- We have developed a novel humanized mouse model in which the clinically observed organ site-dependent benefit of ICB is faithfully recapitulated. Using this model we provide empirical evidence for a critical role for B cells in ICB-induced clearance of primary tumors and liver metastases. In the same mice, peritoneal metastases were therapy-resistant and lacked B cells and TLS. We identified very high levels of immunosuppressive cytokines in ascites as a potential cause for the observed resistance of peritoneal metastases to ICB.

2024 EULAR points to consider on the initiation of targeted therapies in patients with inflammatory arthritis and a history of cancer

Points to consider		
1	Treating inflammatory arthritis effectively in patients with a history of cancer is important to reduce the potential associated risk of malignancy	5
2	The risk of complications associated with undertreated inflammatory activity should be balanced against the potential risk of targeted antirheumatic therapy-related cancer recurrence	5
3	The rheumatologist should engage with other specialists caring for cancer for the co-management of patients with inflammatory arthritis and a history of cancer	5
4	Appropriate targeted antirheumatic treatment can be initiated without delay in patients with cancer in remission	4
5	In patients with a history of cancer, JAK inhibitors and Abatacept may be used with caution and only in the absence of therapeutic alternatives	5
6	When targeted antirheumatic therapy is indicated in patients with a history of solid cancer*, TNF inhibitors may be preferred over other treatment options	4
7	When targeted antirheumatic therapy is indicated in patients with a history of lymphoma, B-cell-depleting therapy may be preferred over other treatment options	5
8	In patients with a malignancy not in remission and active inflammatory arthritis, the decision to start targeted antirheumatic therapy should be based on a shared decision between the patient, the specialist caring for cancer and the rheumatologist	5

Γενικές οδηγίες για μείωση του ρίσκου…

- Καλός έλεγχος ενεργότητας νόσου
- Διακοπή καπνίσματος
- Περιορισμός ηλιακής ακτινοβολίας
- Μείωση βαρους

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