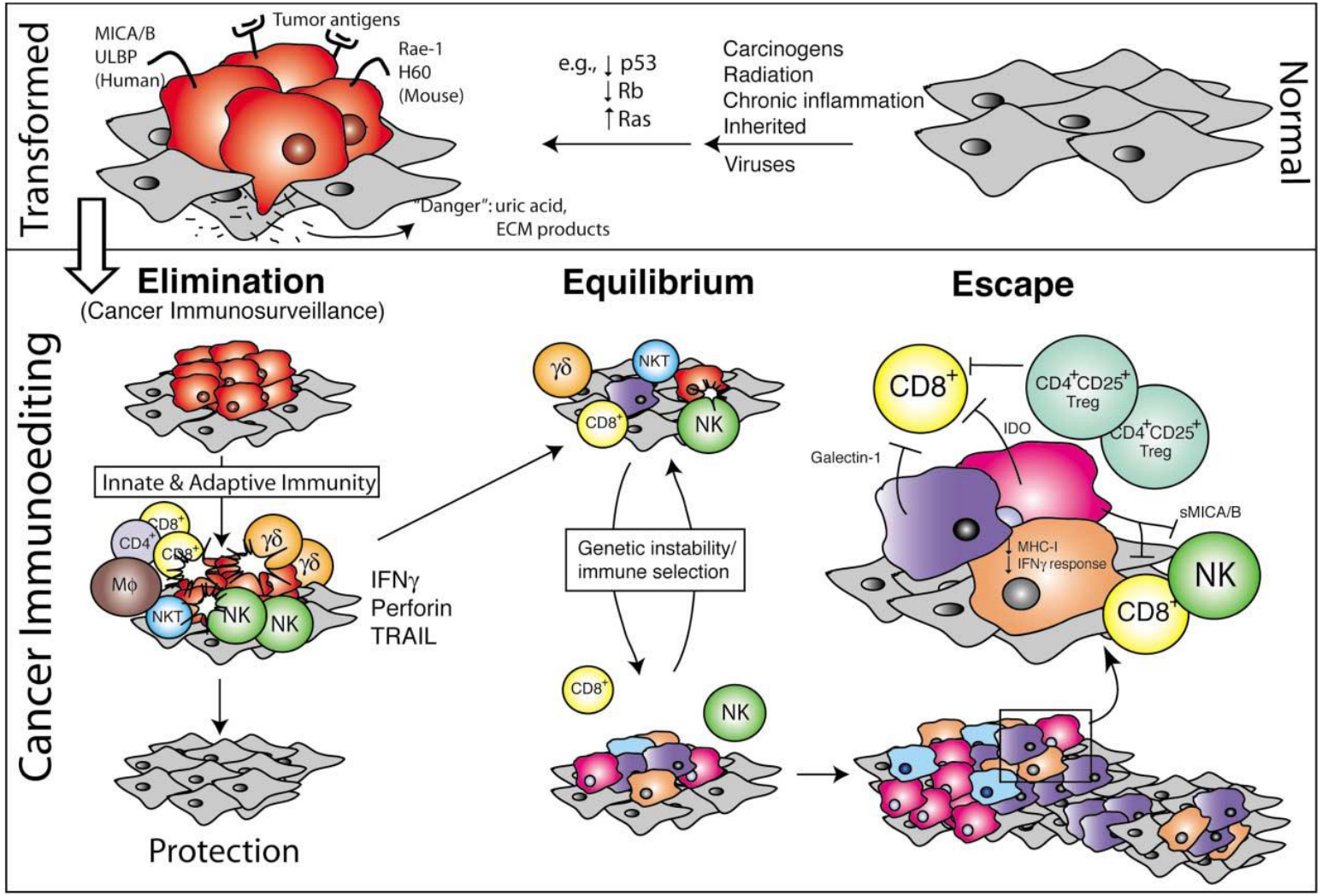
Θεραπεία ρευματικών νοσημάτων σε ασθενείς με ιστορικό καρκίνου

Διαπανεπιστημιακά μαθήματα
Ρευματολογίας

Δαούσης Δημήτρης
Αναπλ. Καθηγητής
Παθολογίας/Ρευματολογίας
Ιατρική Σχολή Πανεπιστημίου Πατρών

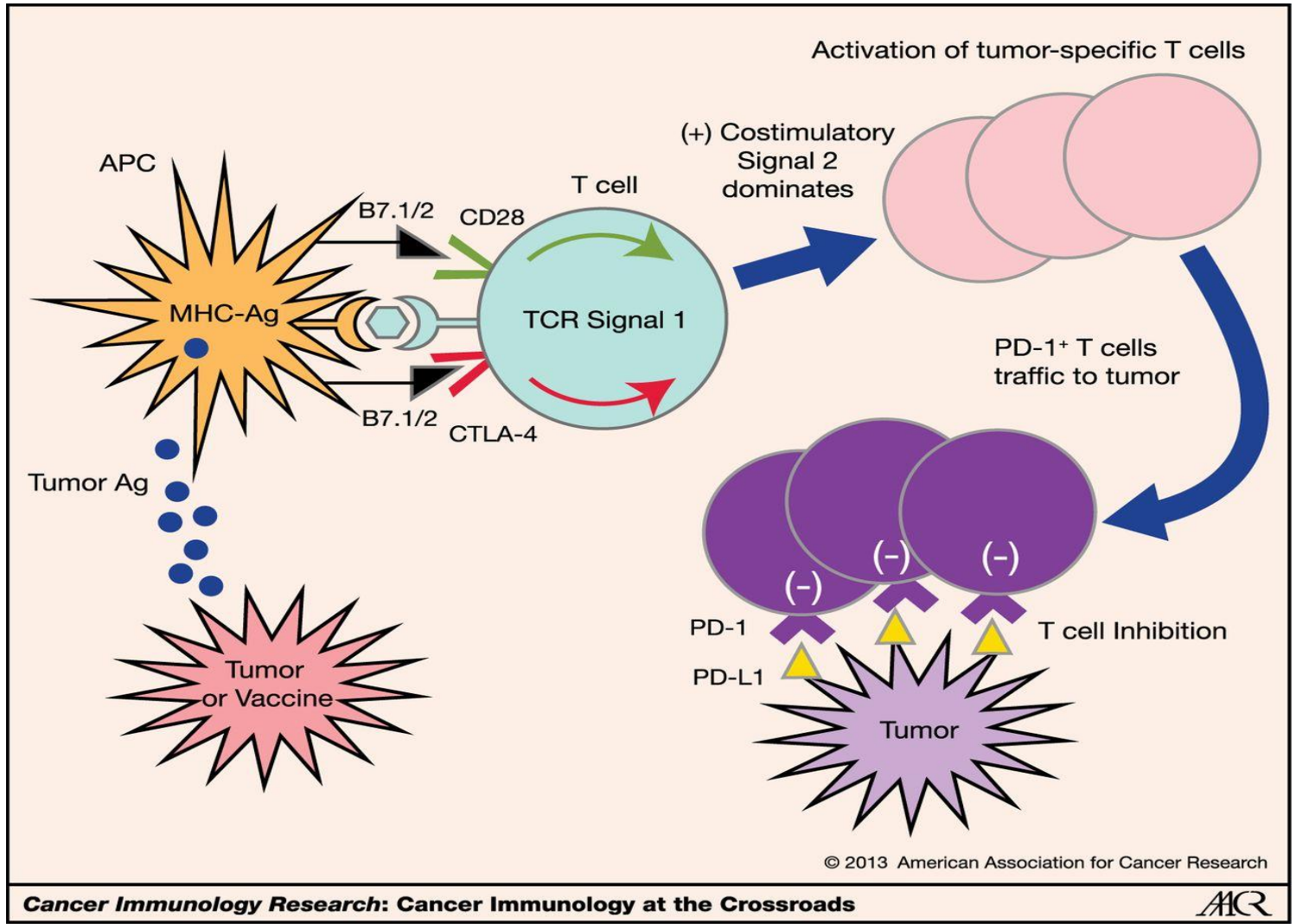
- Σχέση ανοσολογικού συστήματος-καρκίνου
- Σχέση χρόνιας φλεγμονής-καρκίνου
- Ο κίνδυνος για εμφανιση κακοήθειας σε ασθενείς με PA
- Ο κίνδυνος για εμφανιση κακοήθειας με χρήση DMARD
- Ο κίνδυνος για εμφανιση κακοήθειας με χρήση στοχευμένων θεραπειών
- **Χορήγηση στοχευμένων θεραπειών σε ασθενείς με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

Ανοσολογικό σύστημα και καρκίνος
Μια περίπλοκη, στενή σχέση

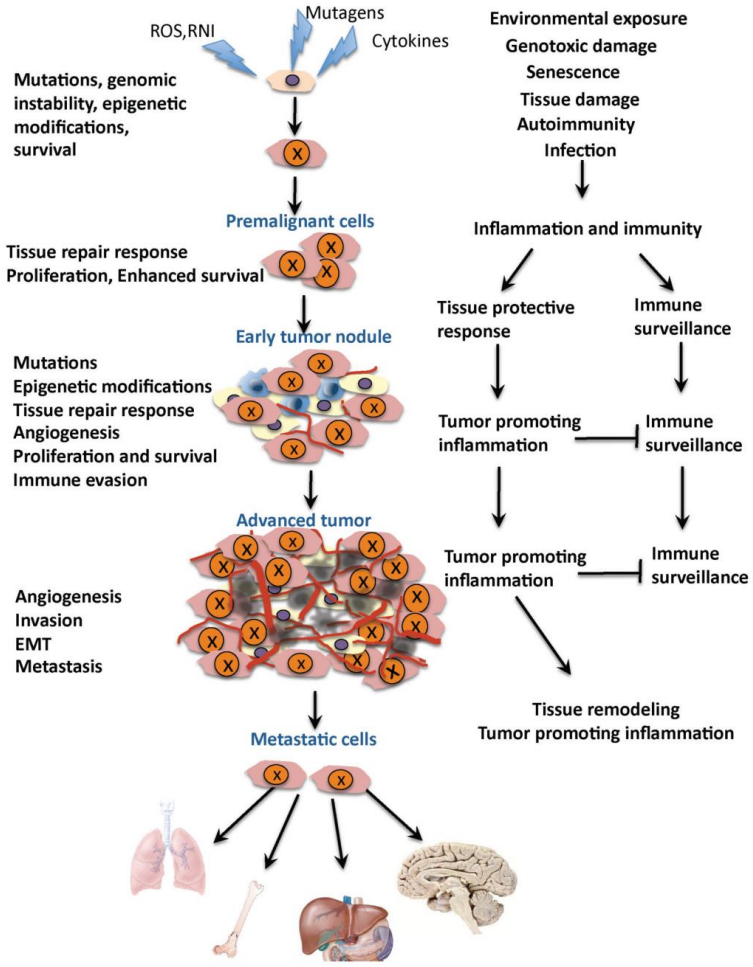


Adv Immunol 2006,90:1

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

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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

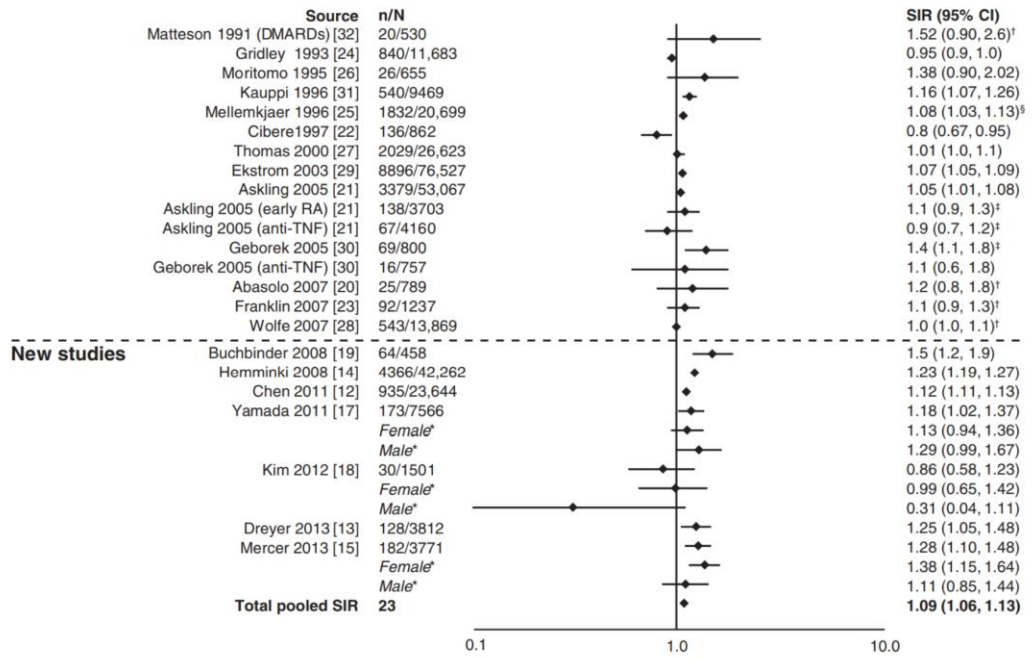

Fig. 2 Relative risk of overall malignancy in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; DMARD, disease-modifying antirheumatic drug; n, number of malignancies; N, population size; RR, relative risk; SIR, standardized incidence ratio; TNF, tumor 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

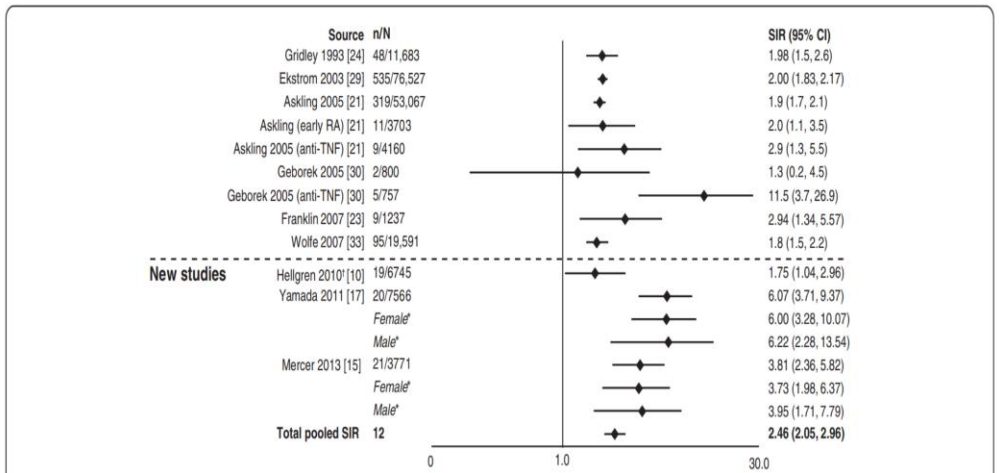

Fig. 3 Relative risk of malignant lymphoma in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; n, number of malignancies; N, population size; OR, odds ratio; SIR, standardized incidence ratio; TNF, tumor necrosis factor. *SIRs by sex are not included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

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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]

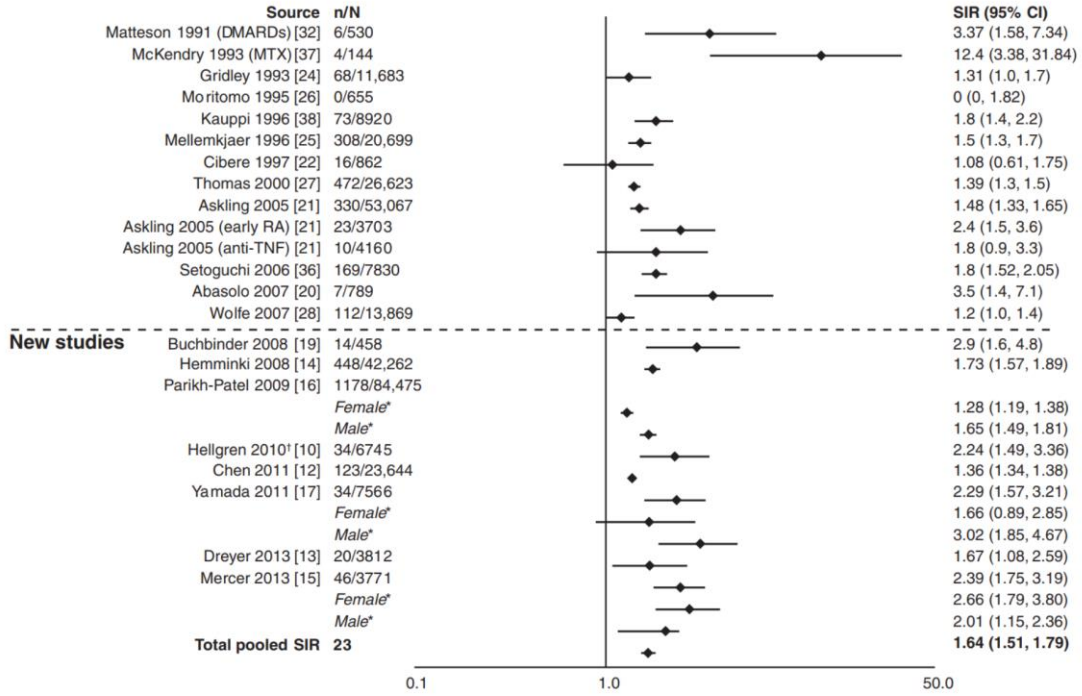
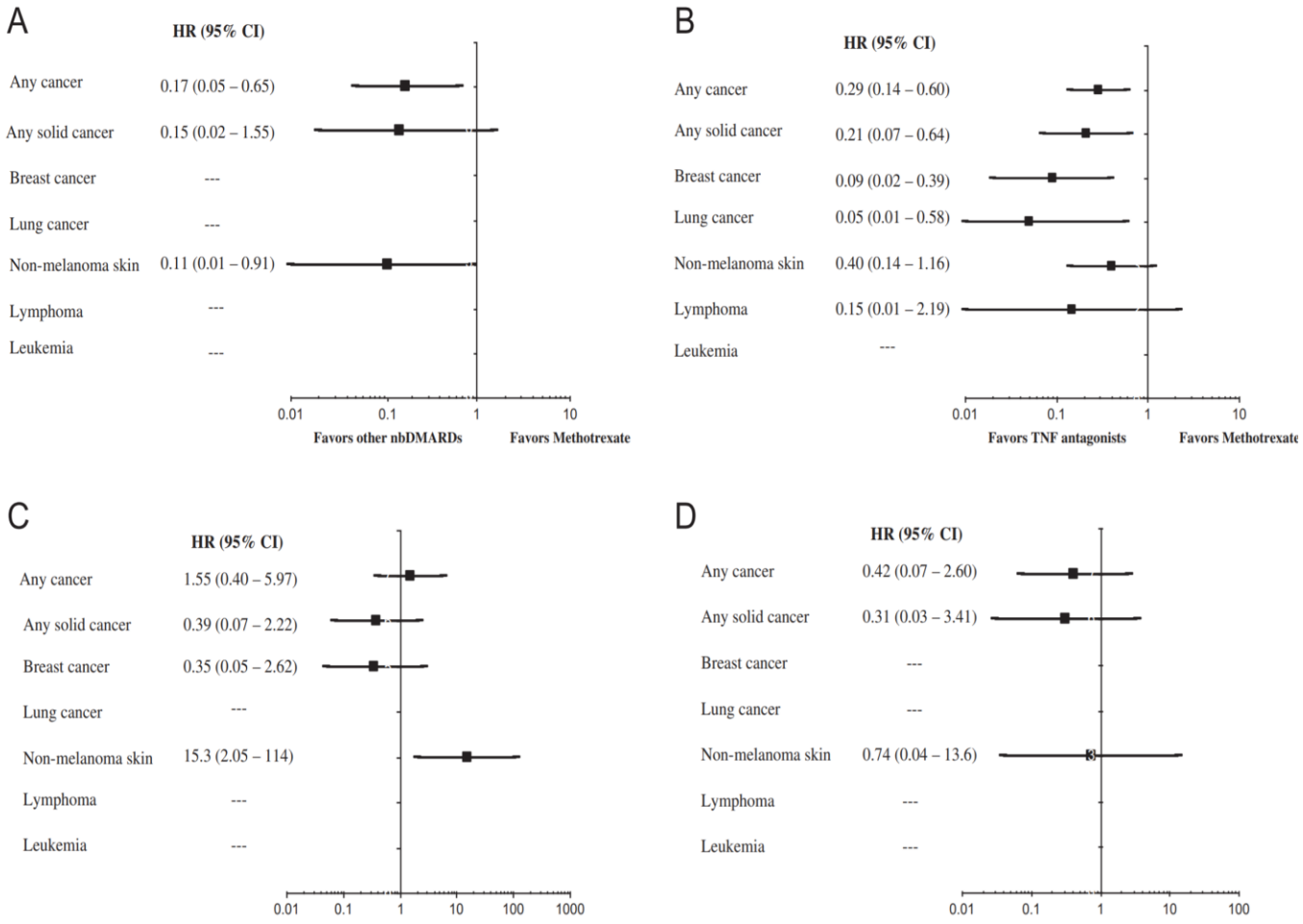


Fig. 6 Relative risk of lung cancer in patients with rheumatoid arthritis (RA) compared with the general population. CI, confidence interval; DMARD, disease-modifying antirheumatic drug; MTX, methotrexate; n, number of malignancies; N, population size; SIR, standardized incidence ratio; TNF, tumor necrosis 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
Ο ρόλος της MTX



- A <u>Other DMARD</u>
- B <u>anti-TNF</u>
- C ABA
- D RTX

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

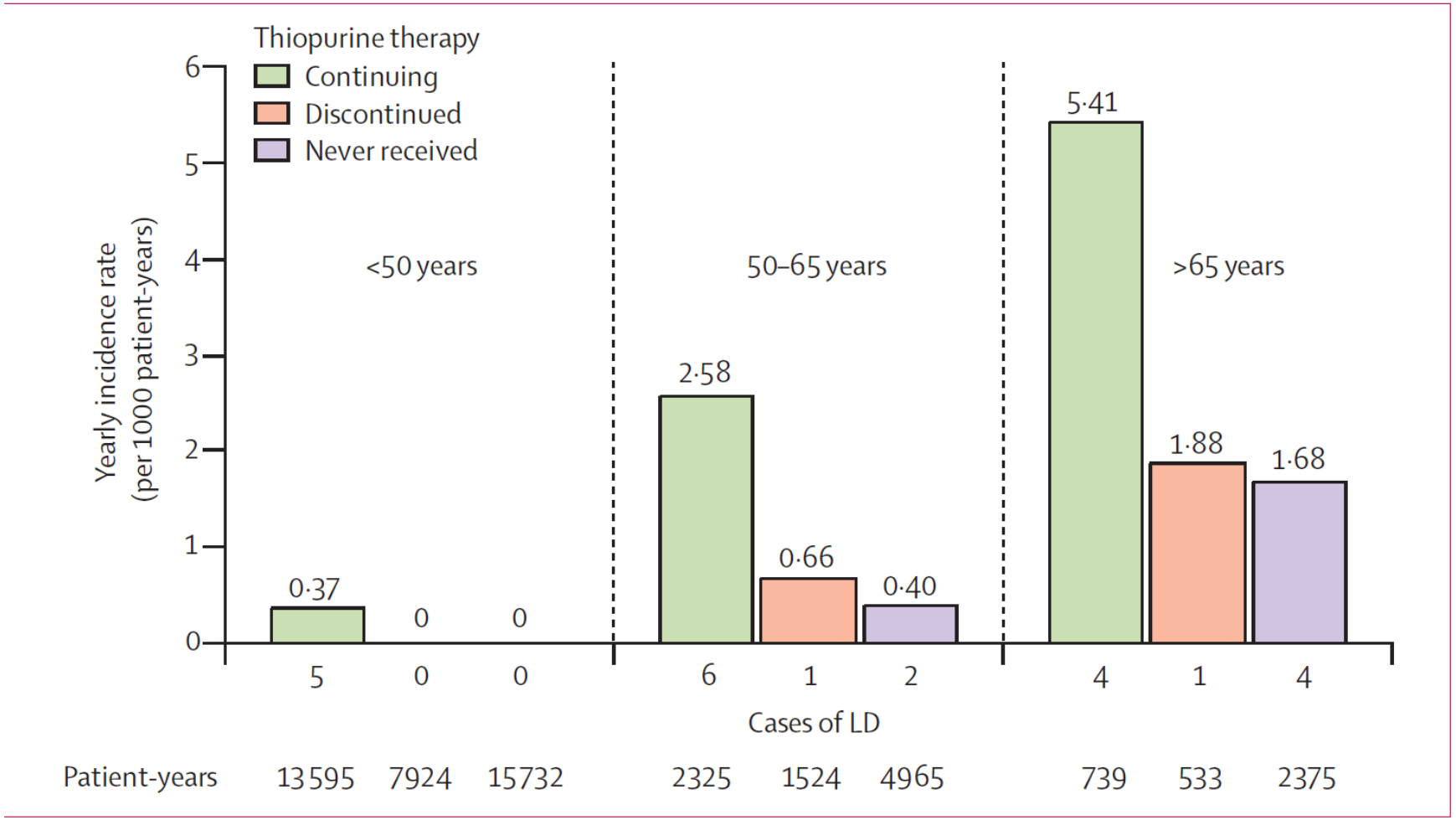
Comparative cancer risk associated with methotrexate, other non-biologic and biologic disease-modifying anti-rheumatic drugs

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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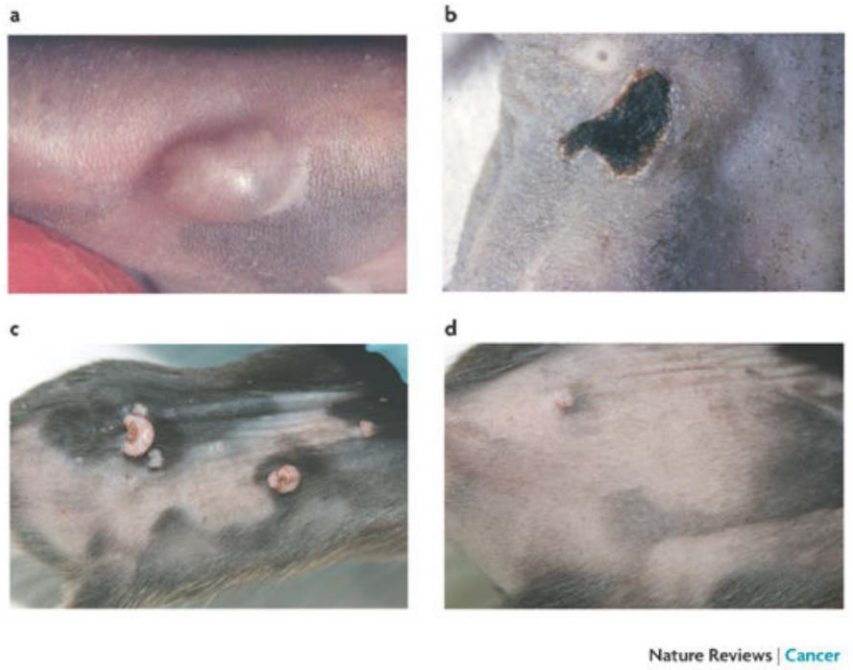
Timeline

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 Reviews | Cancer

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]

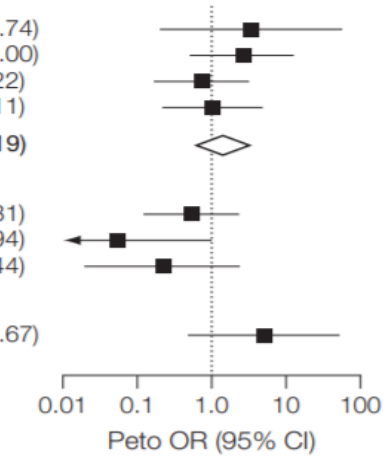
Table 7 Rates of adverse events in placebo and recommended dose anti-TNF groups of randomised controlled trials in RA

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.

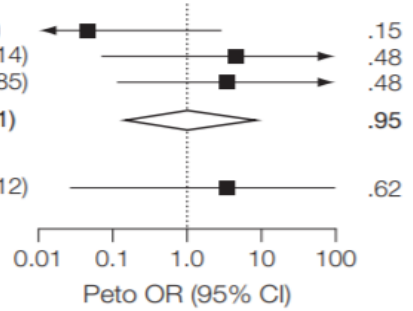
Skin cancer, nonmelanoma[a]

BRMs	No. of Studies	Peto OR (95% CI)	*P* Value
TNF inhibitors			
Certolizumab[53]	1	3.52 (0.21-58.74)	.38
Etanercept[56-61]	3	2.68 (0.55-13.00)	.22
Golimumab[62-66]	5	0.76 (0.18-3.22)	.70
Infliximab[25, 37, 38, 70-72]	3	1.07 (0.22-5.11)	.93
Overall	12	1.37 (0.59-3.19)	.46
Non-TNF inhibitors			
Abatacept[20, 25, 75]	3	0.54 (0.13-2.31)	.41
Anakinra[80]	1	0.06 (0.00-0.94)	.05
Tocilizumab[87, 88]	2	0.22 (0.02-2.44)	.22
Combination			
Abatacept + etanercept/BRM[20]	1	5.16 (0.50-53.67)	.17



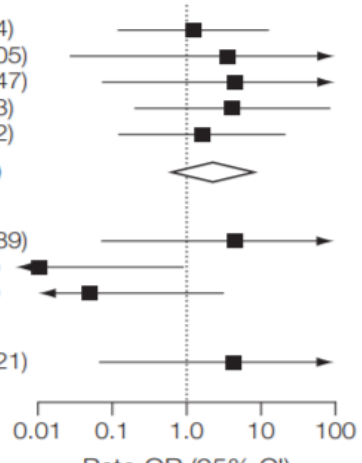
Skin cancer, melanoma[a]

BRMs	No. of Studies	Peto OR (95% CI)	*P* Value
TNF inhibitors			
Adalimumab[44]	1	0.04 (0.00-2.97)	.15
Etanercept[58, 60, 61]	1	4.50 (0.07-286.14)	.48
Infliximab[70-72]	1	3.51 (0.11-111.85)	.48
Overall	3	1.08 (0.11-10.21)	.95
Non-TNF inhibitors			
Anakinra[80]	1	3.50 (0.03-464.12)	.62



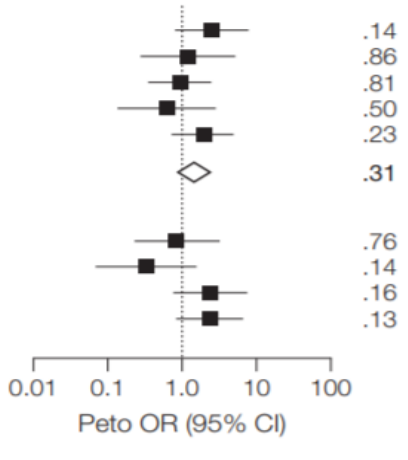
Lymphoma

BRMs	No. of Studies	Peto OR (95% CI)	*P* Value
TNF inhibitors			
Adalimumab[44, 45, 49]	3	1.23 (0.12-13.14)	.86
Certolizumab[53]	1	3.51 (0.03-458.05)	.61
Etanercept[55]	1	4.59 (0.07-284.47)	.47
Golimumab[62, 65]	2	4.18 (0.20-88.53)	.36
Infliximab[37, 38, 68, 70-72]	3	1.64 (0.12-21.92)	.71
Overall	10	2.14 (0.55-8.38)	.28
Non-TNF inhibitors			
Abatacept[76]	1	4.51 (0.07-285.89)	.48
Anakinra[80]	1	0.01 (0.00-0.94)	.05
Tocilizumab[88]	1	0.05 (0.00-3.19)	.16
Combination			
Anakinra + etanercept[21]	1	4.45 (0.07-287.21)	.48



Solid tumors[b]

BRMs	No. of Studies	Peto OR (95% CI)	*P* Value
TNF inhibitors			
Adalimumab[36, 43, 44, 47, 49]	5	2.35 (0.75-7.36)	.14
Certolizumab[51, 53]	2	1.14 (0.26-4.98)	.86
Etanercept[55, 58-61]	3	0.89 (0.33-2.39)	.81
Golimumab[62-66]	5	0.59 (0.13-2.76)	.50
Infliximab[25, 37, 38, 68-72]	5	1.82 (0.69-4.78)	.23
Overall	20	1.31 (0.78-2.20)	.31
Non-TNF inhibitors			
Abatacept[20, 25, 76, 77]	4	0.82 (0.22-3.01)	.76
Anakinra[78-81]	4	0.31 (0.07-1.49)	.14
Rituximab[26, 32, 82, 83]	4	2.28 (0.72-7.25)	.16
Tocilizumab[41, 86, 88, 89]	4	2.22 (0.79-6.20)	.13



- JAMA 2012

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

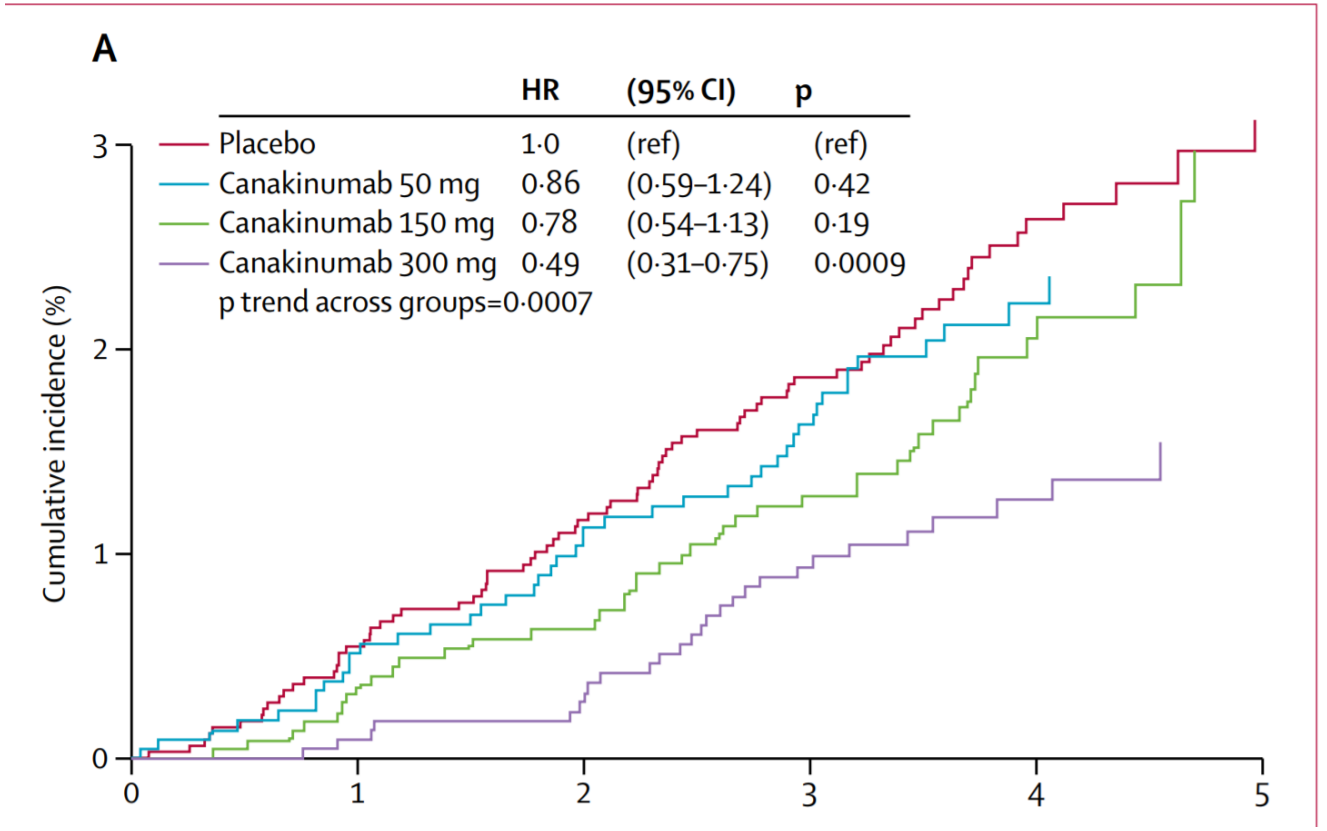
Δεδομένα από αρχεία...

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

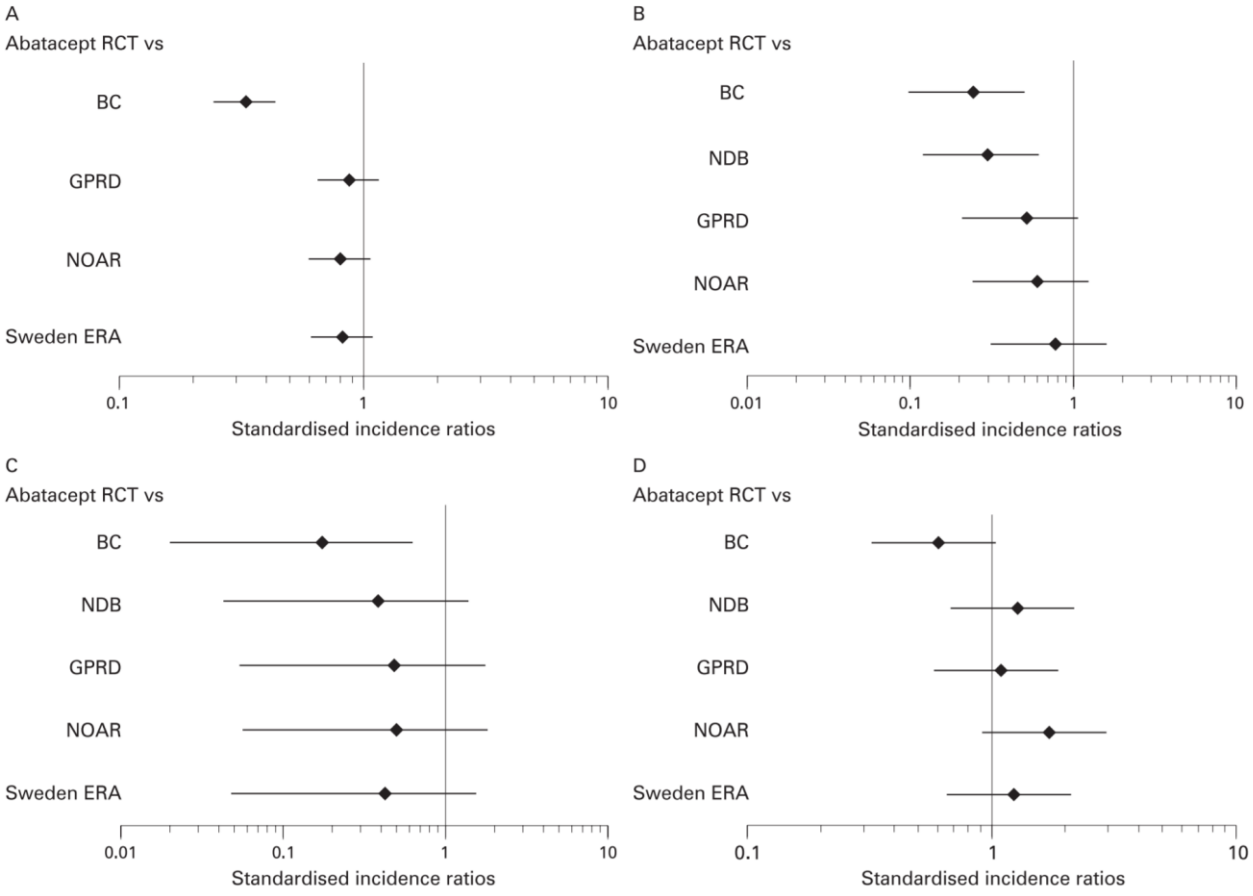
Paul M Ridker, Jean G MacFadyen, Tom Thuren, Brendan M Everett, Peter Libby, Robert J Glynn*, on behalf of the CANTOS Trial Group†*

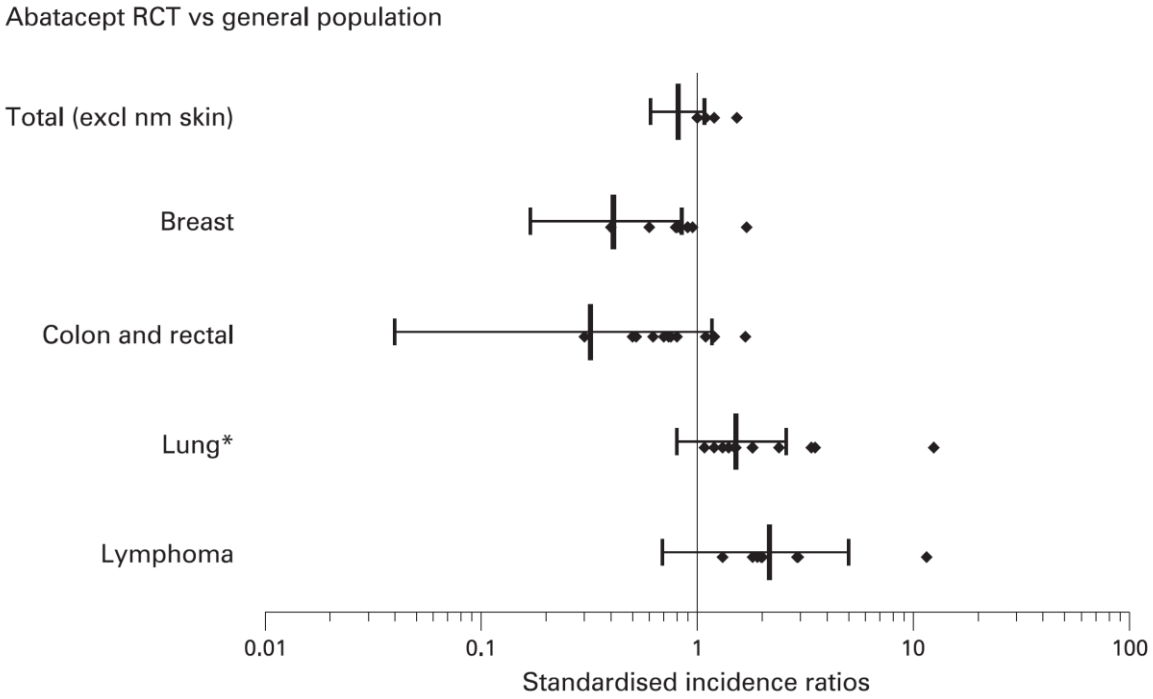
- Η αναστολή της IL-1 μειώνει την πιθανότητα καρκίνου πνεύμονα!



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

RHEUMATOLOGY

Rheumatology 2019;58:683–691
doi:10.1093/rheumatology/key352
Advance Access publication 7 December 2018

Original article

Abatacept initiation in rheumatoid arthritis and the risk of cancer: a population-based comparative cohort study

François Montastruc[1,2], Christel Renoux[1,3,4], Sophie Dell'Aniello[1], Teresa A. Simon[5], Laurent Azoulay[1,3,6], Marie Hudson[1,7] and Samy Suissa[1,3]

Initial treatment	Number of patients	Number of events	Person-years	Rate per 100 person-years	Crude HR	Adjusted HR[b] (95% CI)
Any cancer						
Other bDMARD	59 860	4197	123 254	3.41	1.00	1.00 (Reference)
Abatacept	4328	409	8596	4.76	1.39	1.17 (1.06, 1.30)

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.

Το rituximab?

- Πρόκειται ουσιαστικά για αντικαρκινικό φάρμακο
- Υπάρχει 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

VOLUME 29 · NUMBER 7 · MARCH 1 2011

JOURNAL OF CLINICAL ONCOLOGY ORIGINAL REPORT

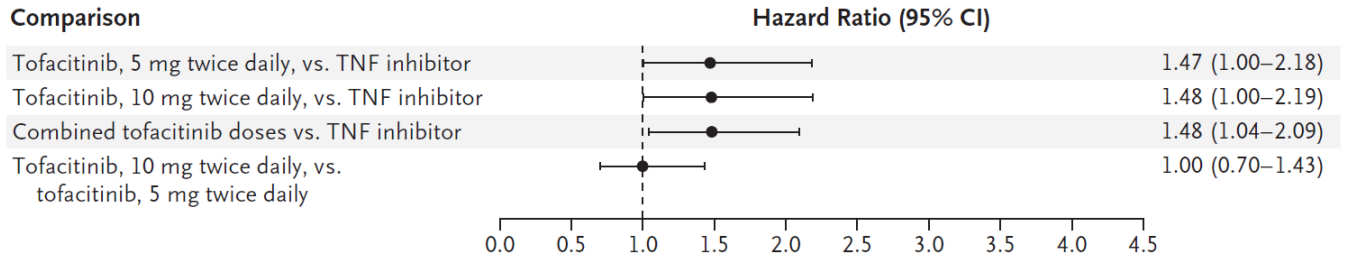
Risk Factors for the Development of Secondary Malignancy After High-Dose Chemotherapy and Autograft, With or Without Rituximab: A 20-Year Retrospective Follow-Up Study in Patients With Lymphoma

The NEW ENGLAND JOURNAL of MEDICINE

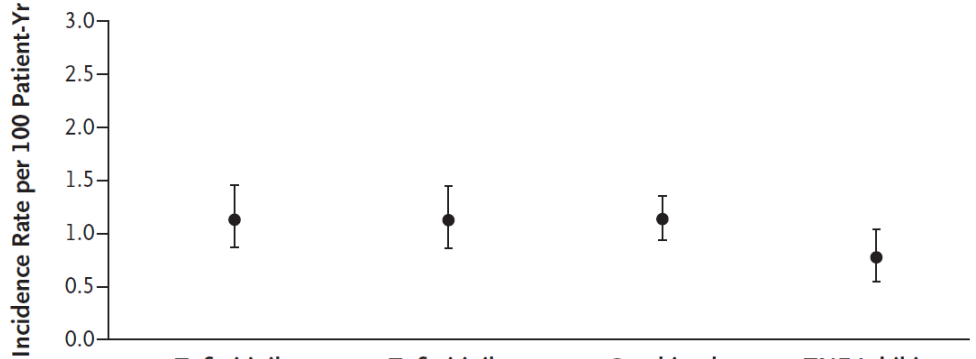
ORIGINAL ARTICLE

Cardiovascular and Cancer Risk with Tofacitinib in Rheumatoid Arthritis

A Hazard Ratio for Cancers, Excluding NMSC



B Incidence Rate for Cancers, Excluding NMSC

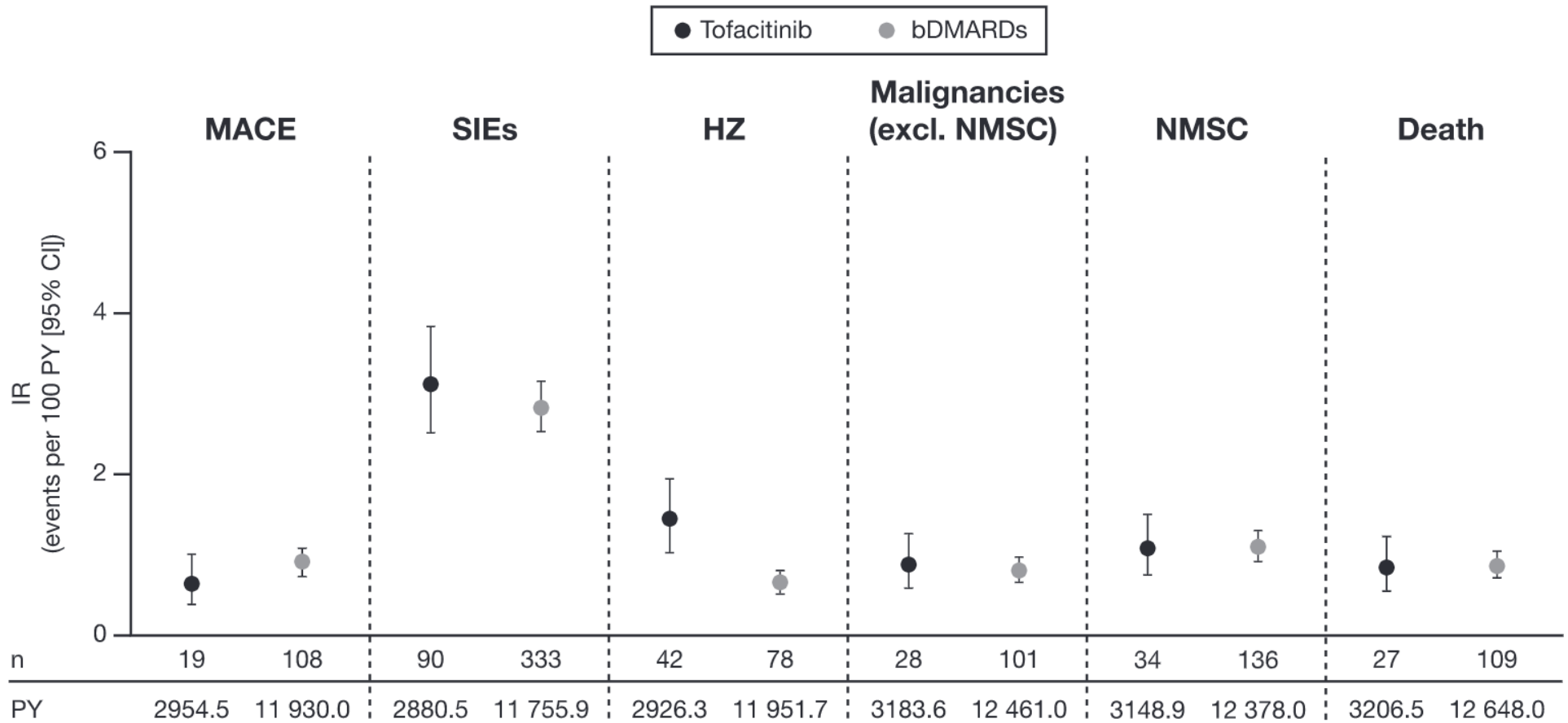


	Tofacitinib, 5 mg Twice Daily	Tofacitinib, 10 mg Twice Daily	Combined Tofacitinib Doses	TNF Inhibitor
No. of Patients with First Event/Total No. (%)	62/1455 (4.3)	60/1456 (4.1)	122/2911 (4.2)	42/1451 (2.9)
No. of Patient-Yr	5491.48	5311.71	10,803.19	5482.30
Incidence Rate per 100 Patient-Yr (95% CI)	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



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

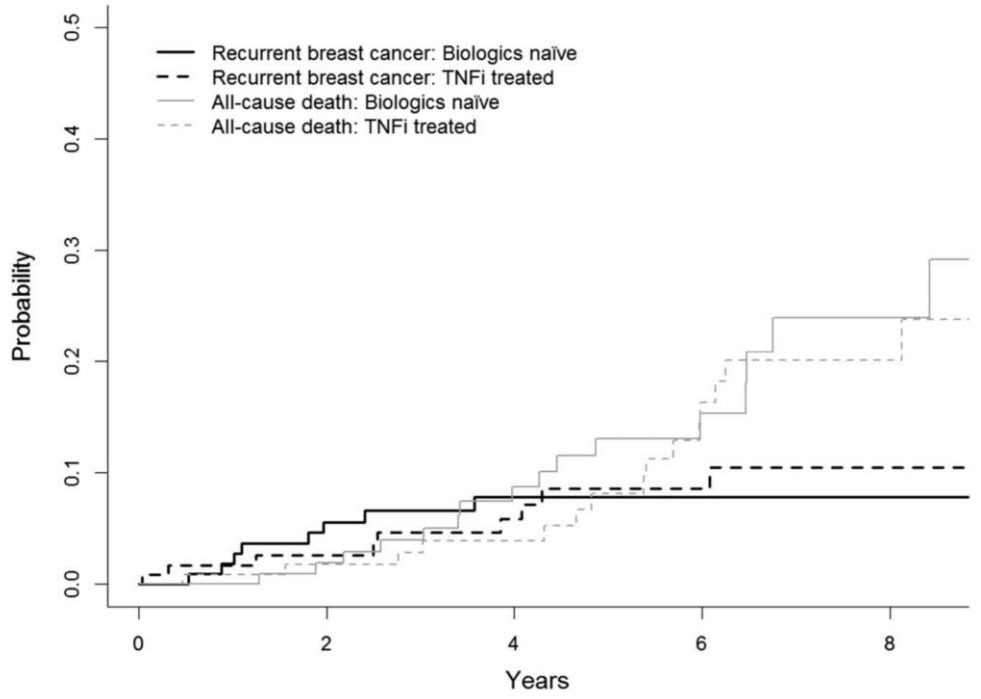
Clinical and epidemiological research

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

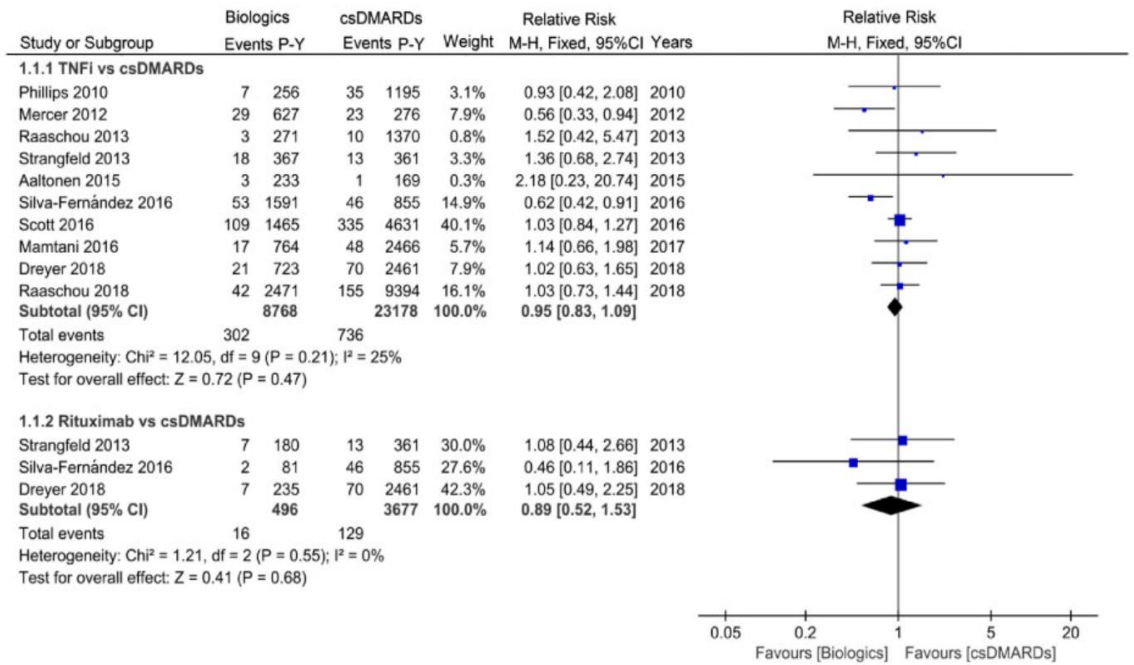
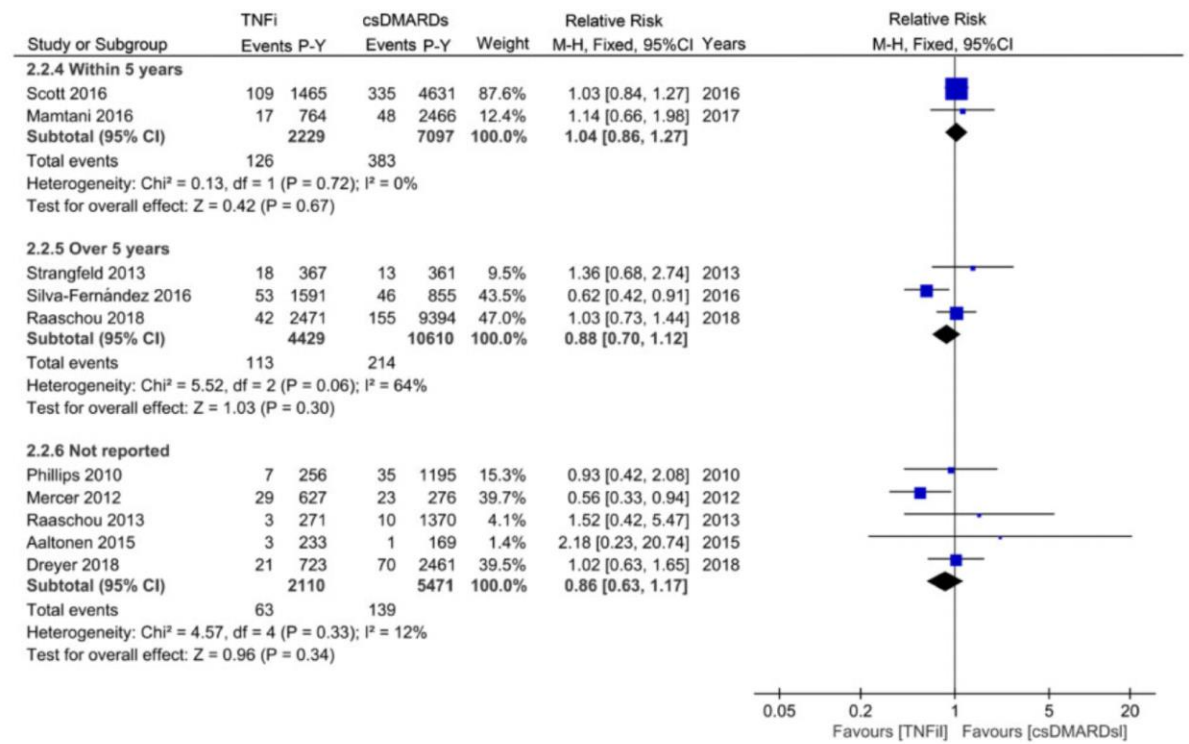


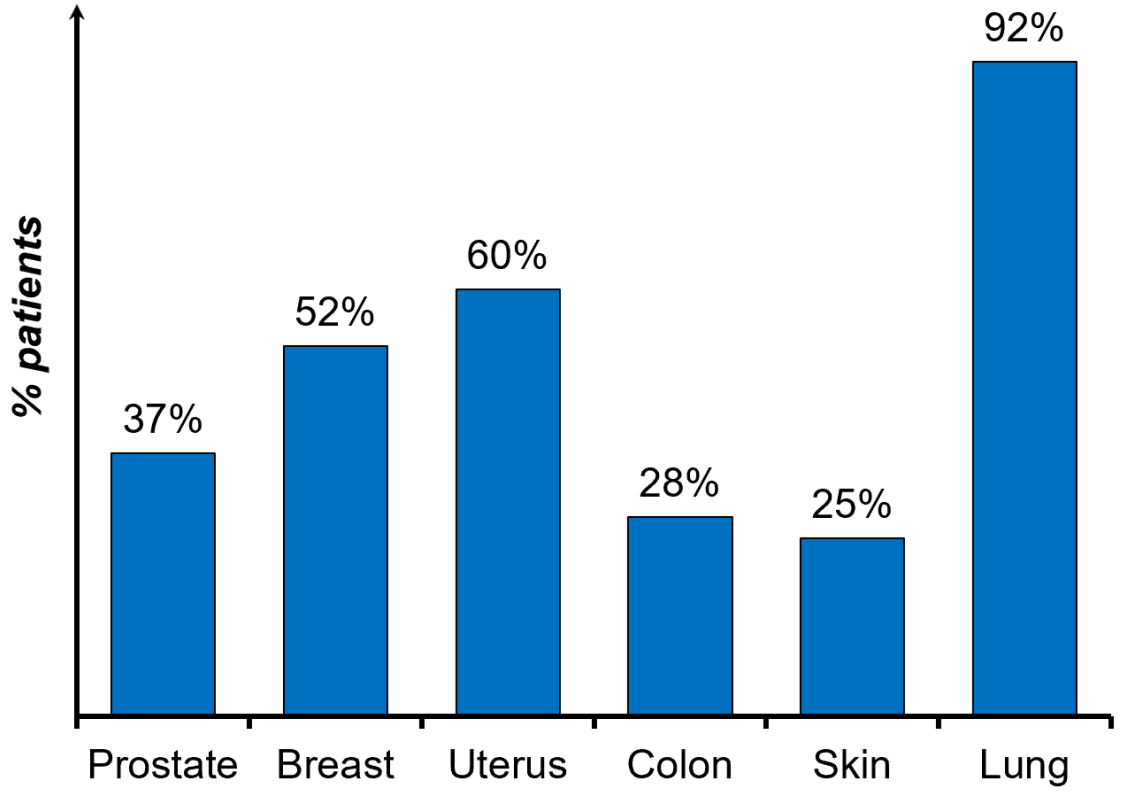
FIG. 4 Relative risk of cancer recurrence between TNFi and csDMARDs, stratified by intervals between prior cancer and TNFi (re-)initiation



Γενικές οδηγίες για μείωση του ρίσκου…

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

Screening με βαση τις οδηγίες
Δεν τα καταφέρνουμε τόσο καλά.....



Dougados M, et al. *Ann Rheum Dis* 2014;73:62–68

- **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.

Scientific Abstracts
Oral Presentations From hearts to lungs: comobidities in RA

PDF

OP0045 EULAR POINTS TO CONSIDER ON THE INITIATION OF TARGETED THERAPIES IN PATIENTS WITH INFLAMMATORY ARTHRITIDES AND A HISTORY OF CANCER FREE

E. Sebbag [1], K. Lauper [2], J. Molina Collada [3], D. Aletaha [4], J. Askling [5], K. Benesova [6], H. Bertheussen [7], S. Bitoun [8], E. C. Bolek [9], G. R. Burmester [10], H. Canhão [11], K. Chatzidionysiou [5], J. Curtis [12], F. X. Danlos [13], V. Guimaraes [14], M. L. Hetland [15], F. Iannone [16], M. Kostine [17], T. W. Kragstrup [18], T. K. Kvien [19], A. Regierer [10], H. Schulze-Koops [20], L. Silva-Fernández [21], Z. Szekanecz [22], M. H. Buch [23], A. Finckh [2], J. E. Gottenberg [24]

Results The group formulated 5 overarching principles and 8 PTC relevant to the initiation of targeted therapies in patients with IA and a history of cancer. Major themes included a) the need to assess.

the individualized risk of cancer recurrence based on the characteristics of the patient, the cancer and the underlying disease; b) the importance of engaging with specialists caring for the cancer and.

to define treatment based on a shared decision between the patient and the rheumatologist;

c) the possibility to initiate without delay an appropriate targeted therapy for the treatment of.

the IA in patients in remission of their cancer; d) the proposal to prefer anti-cytokine bDMARDs.

over other treatment options in patients with history of solid cancer and to prefer.

B cell depleting therapy in patients with a history of lymphoma; e) the proposal to use JAK inhibitors and abatacept with caution, and only in the absence of therapeutic alternatives, based on.

the significant increase in cancer incidence in patients without a history of cancer, with tofacitinib compared to anti-TNF in a randomized clinical trial, and a modest but significant increase.

with abatacept compared to other bDMARDs in some observational studies.

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