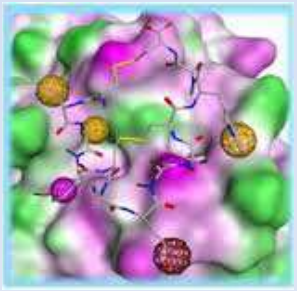
Università degli Studi di Padova
Dipartimento di Scienze Ginecologiche e della Riproduzione Umana
Scuola di Specializzazione in Ginecologia e Ostetricia
Direttore Prof. Giovanni Battista Nardelli

INNOVATIONS IN GYNECOLOGICAL MALIGNANCIES

STATIN USE

IN ADVANCED STAGES OVARIAN / ENDOMETRIAL CANCER



- ***Dott. S. Gizzo***

Estimated New Cases*

Females

Breast	232,670	29%
Lung & bronchus	108,210	13%
Colorectum	65,000	8%
Uterine corpus	52,630	6%
Thyroid	47,790	6%
Non-Hodgkin lymphoma	32,530	4%
Melanoma of the skin	32,210	4%
Kidney & renal pelvis	24,780	3%
Pancreas	22,890	3%
Leukemia	22,280	3%
All Sites	**810,320**	**100%**

Estimated Deaths

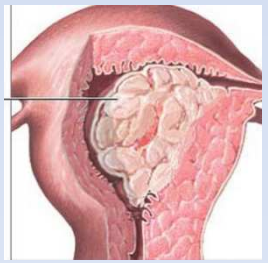
Females

Lung & bronchus	72,330	26%
Breast	40,000	15%
Colorectum	24,040	9%
Pancreas	19,420	7%
Ovary	14,270	5%
Leukemia	10,050	4%
Uterine corpus	8,590	3%
Non-Hodgkin lymphoma	8,520	3%
Liver & intrahepatic bile duct	7,130	3%
Brain & other nervous system	6,230	2%
All Sites	**275,710**	**100%**

Survival Rate and Diagnosis for Varied Stages (2002-2008)

Stage at Diagnosis	Five-year Relative Survival Rate	Percentage of Total Women Diagnosed
Localized (cancer is limited to organ from which it originated)	91.5%	15%
Regional (cancer has spread to nearby lymph nodes or organs and tissue)	71.9%	17%
Distant (cancer has spread to distant organs or lymph nodes)	26.9%	61%
Unstaged (not enough information to identify a stage)	22%	7%

Stage	Distribution (%)	5 year survival
Confined to uterus	69%	95.5%
Regional spread (lymph nodes)	19%	67.5%
Distant	8%	17.1%
Unknown (unstaged)	4%	7.5%



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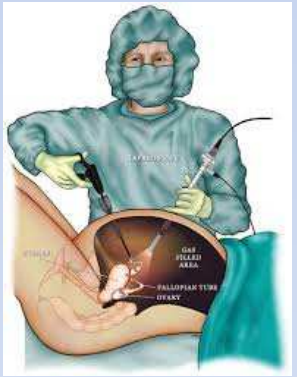

"It doesn't matter if care is cutting-edge and technologically advanced; if it doesn't take the patient's goals into account, it may not be worth doing."

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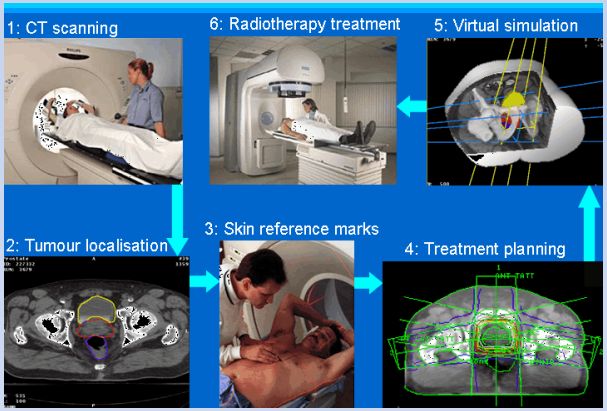
1: CT scanning
6: Radiotherapy treatment
5: Virtual simulation
2: Tumour localisation
3: Skin reference marks
4: Treatment planning


Chemotherapy
cancer
drugs
cell
growth
division
tumors
effectively
treatment


KILLER FREED,
DOCTOR JAILED

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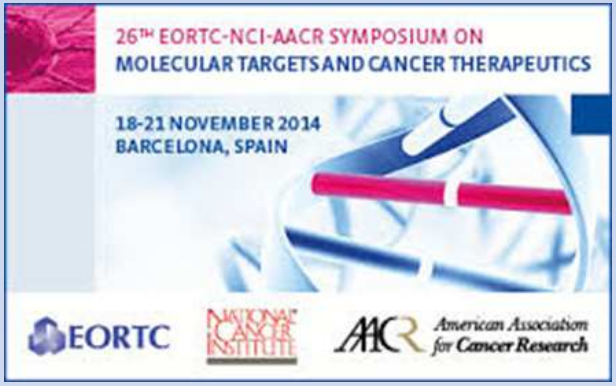
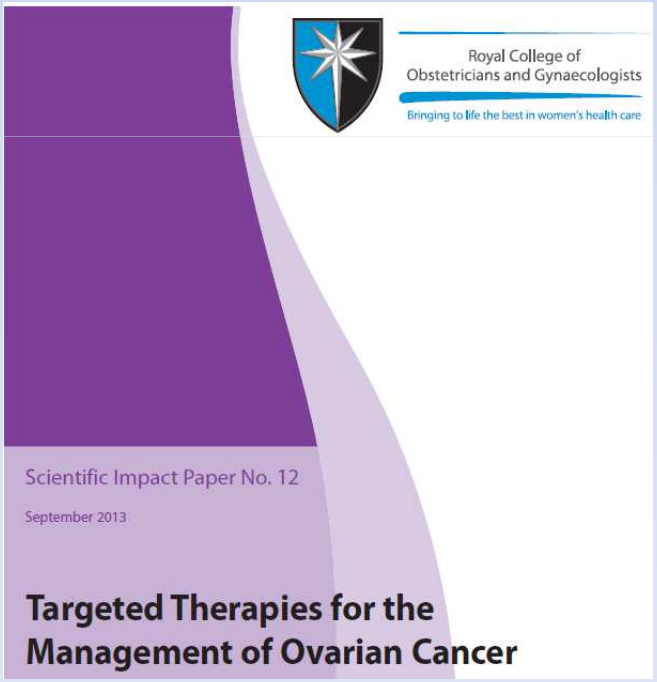
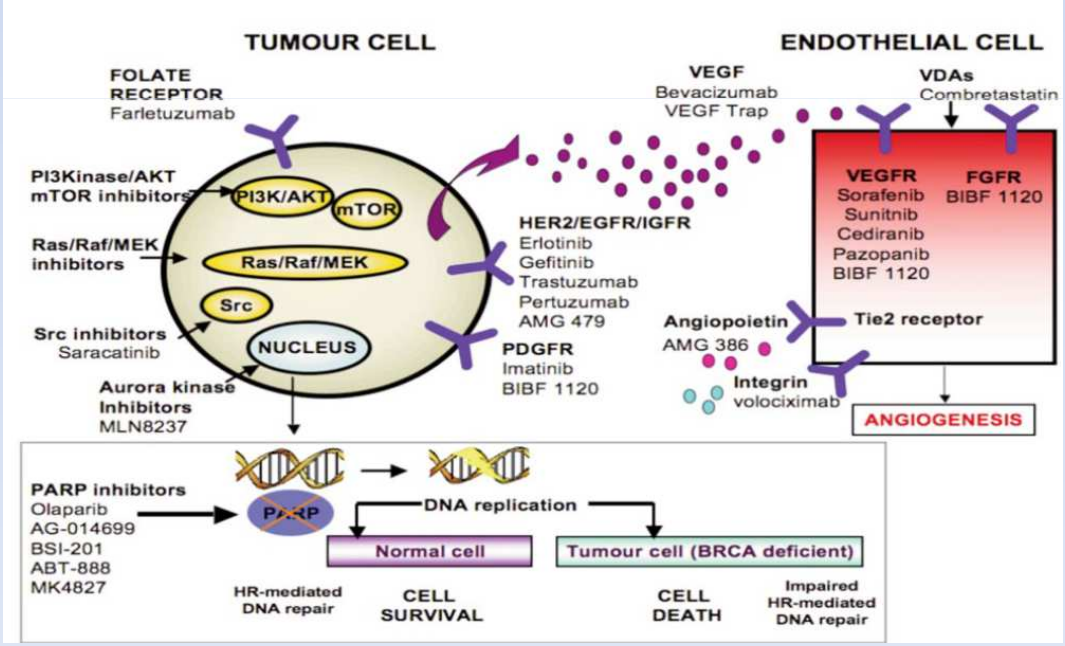




Targeted approaches in Ovarian Cancer.[2]



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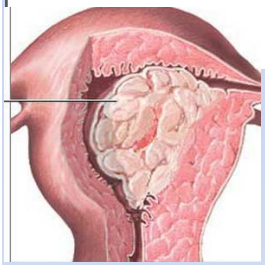
REVIEW

Molecular targets and targeted therapeutics in endometrial cancer



Britta Weigelt[a] *and Susana Banerjee*[b]

Table 3. Phase II trials of targeted therapies for patients with recurrent or metastatic endometrial cancer

Molecular target	Drug
mTOR	Everolimus [98]
	Temsirolimus [99▪]
	Temsirolimus [99▪]
	Deferolimus [100]
	Ridaforolimus [101]
	Ridaforolimus [102]
HER2	Trastuzumab [103]
EGFR	Erlotinib [104]
	Gefitinib [105]
VEGF	Bevacizumab [106▪]
	Thalidomide [107]
VEGFR	Sunitinib [108]
	Sunitinib [109]
	Sorafenib [110]

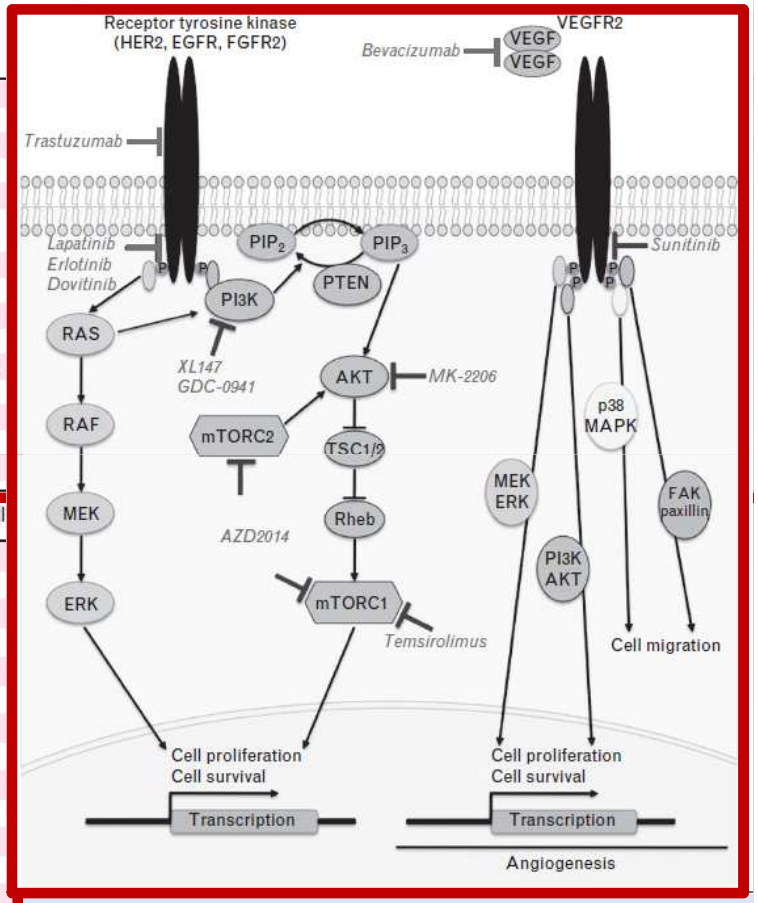


Table 2. Molecular genetic alterations in type I and II endometrial cancer

Alteration	Prevalence in type I (endometrioid)	Prevalence in type II (serous and clear cell
PTEN mutation [22–24,25▪▪]	57–83%	Up to 10%
PIK3CA mutation [25▪▪,26▪▪]	Up to 50%	30%
PIK3R1 mutation [26▪▪,27▪▪]	20–40%	5–12%
PIK3CA amplification [28–30]	2–14%	45%
AKT mutation [31–33]	2%	0%
KRAS mutation [21,25▪▪,34–38]	22–43%	2–5%
TP53 mutation [36,39–46]	2–20%	9–54% (>90% serous)
FGFR2 mutation [47–49]	16%	2%
HER2 amplification [50–55]	Up to 3%	17–50%
EGFR overexpression [51,56,57]	38–46%	35–56%
EGFR mutation/amplification [56]	0%	0%
E-cadherin loss [58–61]	6–57%	41–87%
Nuclear β-catenin [60,62,63]	14–47%	0–3%
BAF250a (*ARID1A*) loss [64▪]	29–39%	18–26%
Microsatellite instability [36,39,65–70]	20–45%	0–14%

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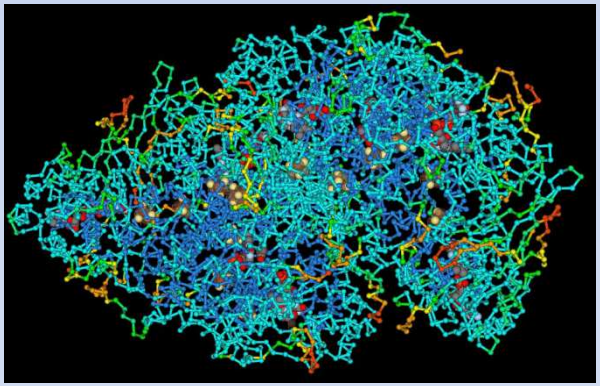
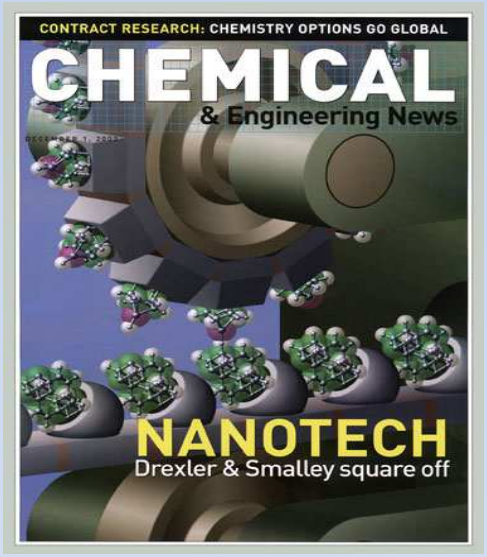
LOST
CONFUSED
UNSURE
UNCLEAR
PERPLEXED
DISORIENTED
BEWILDERED




CONTRACT RESEARCH: CHEMISTRY OPTIONS GO GLOBAL
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MIRACLES
HAPPEN


Nature's cancer antidote
'Turkey Tail Mushroom'
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Friday, November 30, 2012
FDA Approves Bastyr Turkey Tail Trial for Cancer Patients
Researchers study how a traditional Chinese mushroom helps cancer patients strengthen their immune systems in a $5.4 million investigation.
Turkey tail mushrooms, named for their colorful stripes, have been brewed for thousands of years in Chinese medicinal teas.

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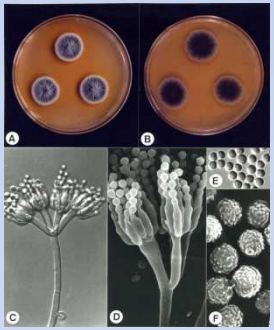


Eur. J. Biochem. 87, 313–321 (1978)

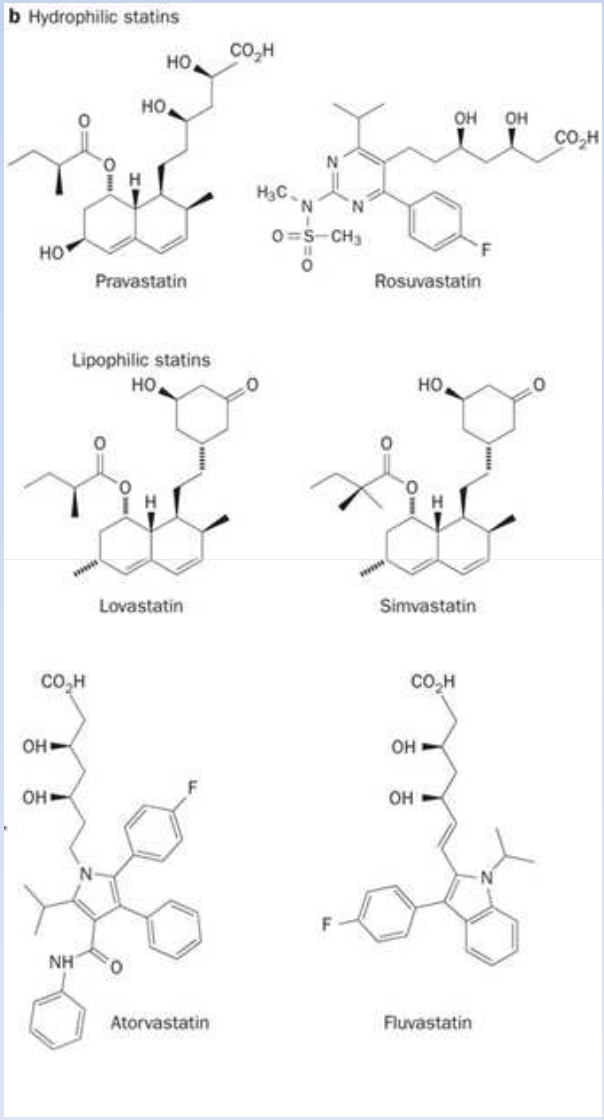
Inhibitory Effects on Lipid Metabolism in Cultured Cells of ML-236B, a Potent Inhibitor of 3-Hydroxy-3-methylglutaryl-Coenzyme-A Reductase

Isao KANEKO, Yoko HAZAMA-SHIMADA, and Akira ENDO
Fermentation Research Laboratories, Sankyo Co. Ltd, Tokyo

(Received January 4, 1978)

inhibition of growth was prevented by the presence in the culture medium of mevalonate, but not by acetate, thus indicating that ML-236B inhibits cell growth by specifically interferring with mevalonate synthesis. It is further concluded that slight activity of endogenous sterol synthesis, which provides endogenous compounds generated from mevalonate, may be essential to the growth of cells even when sufficient amounts of cholesterol are available to the cells.

Akira Endo



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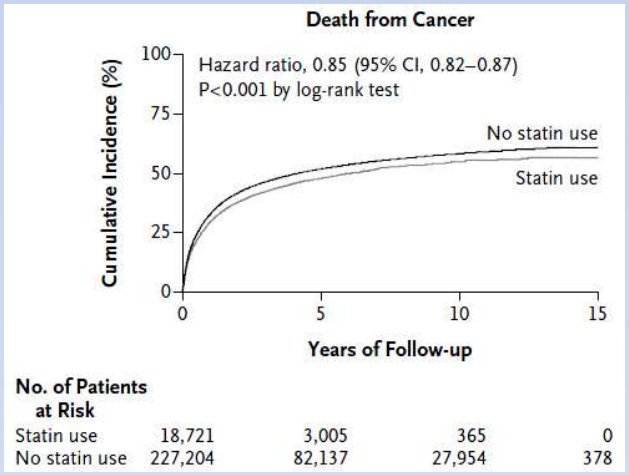
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

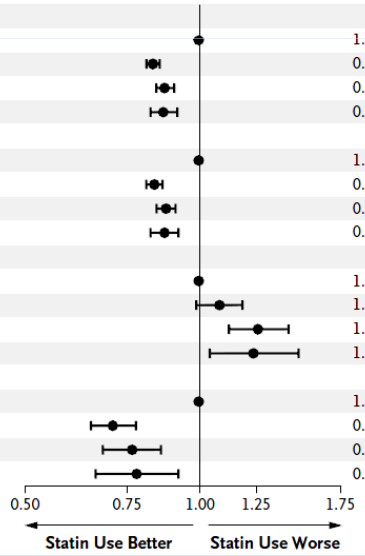
Statin Use and Reduced Cancer-Related Mortality

Sune F. Nielsen, Ph.D., Børge G. Nordestgaard, M.D., D.M.Sc.
and Stig E. Bojesen, M.D., Ph.D., D.M.Sc.



A Nationwide Study

	No. of Patients	No. of Deaths	Hazard Ratio (95% CI)
Any cause			
0.00	277,204	184,895	1.00
0.01–0.75	9,780	5,730	0.82 (0.81–0.85)
0.76–1.50	6,181	3,438	0.87 (0.83–0.89)
>1.50	2,760	1,531	0.87 (0.81–0.91)
Cancer			
0.00	277,204	153,327	1.00
0.01–0.75	9,780	4,680	0.83 (0.81–0.86)
0.76–1.50	6,181	2,810	0.87 (0.83–0.91)
>1.50	2,760	1,250	0.87 (0.81–0.92)
Cardiovascular cause			
0.00	277,204	13,512	1.00
0.01–0.75	9,780	529	1.08 (0.99–1.19)
0.76–1.50	6,181	314	1.25 (1.21–1.41)
>1.50	2,760	134	1.24 (1.03–1.48)
Other cause			
0.00	277,204	18,056	1.00
0.01–0.75	9,780	521	0.70 (0.64–0.77)
0.76–1.50	6,181	314	0.76 (0.68–0.86)
>1.50	2,760	147	0.77 (0.66–0.92)



Published OnlineFirst November 13, 2013; DOI: 10.1158/1055-9965.EPI-13-1101

Cancer Epidemiology, Biomarkers & Prevention

Long-term Statin Use and Risk of Breast Cancer

Salvatore Gizzo, Emanuele Ancona, Marco Noventa, Donato D'Antona, and Giovanni Battista Nardelli

To solve the dilemma, an answer to the lingering questions about the association between gynecologic cancer risk and statin use, a meta-analysis will be necessary combining the existing large-scale studies and further prospective ones. In the same way, further prospective studies, even if conducted on breast cancer and statins, should also consider outcomes about ovary and endometrium to increase the amount of data available in this field.

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ARTICLE IN PRESS

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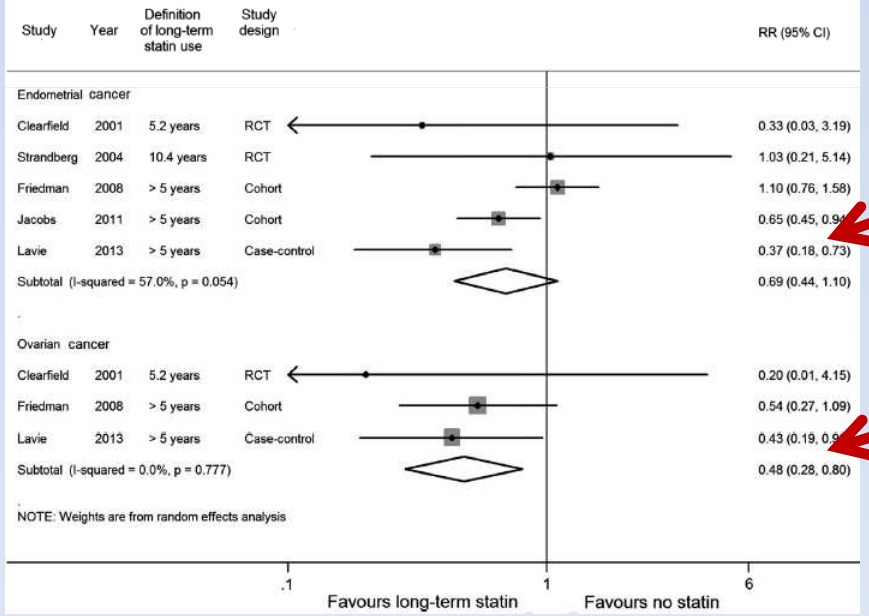
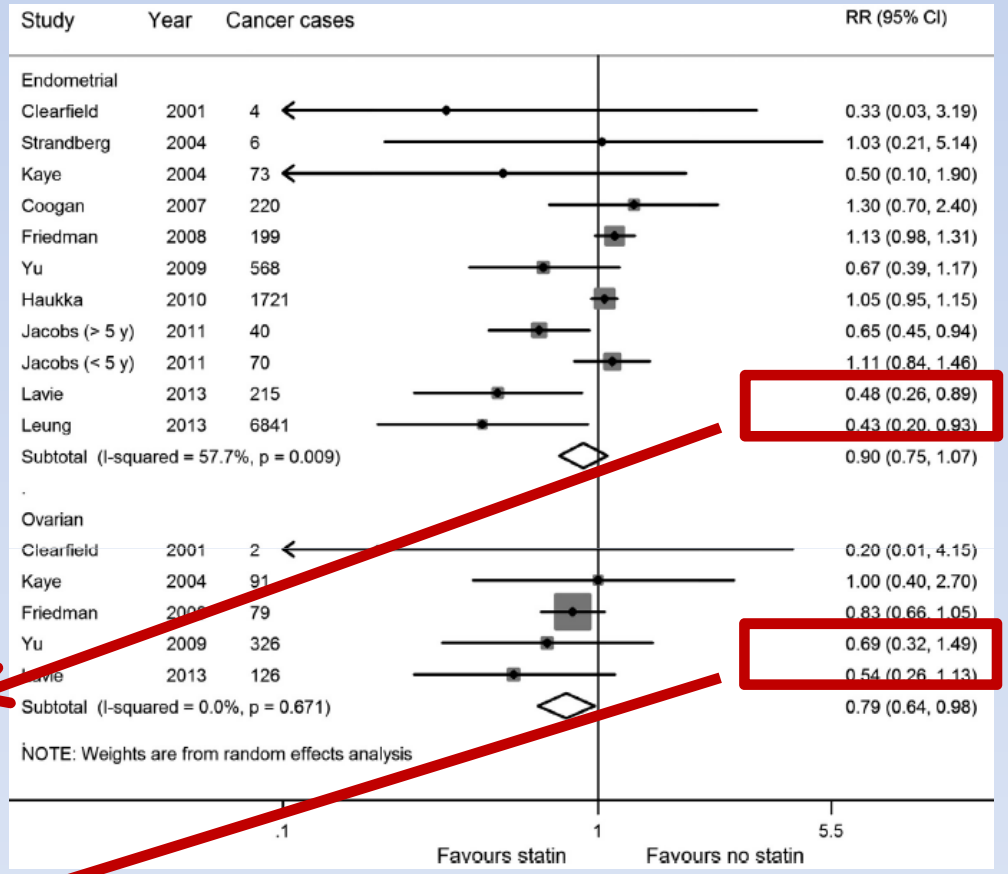
Review

Effect of statin on risk of gynecologic cancers: A meta-analysis of observational studies and randomized controlled trials

Yanqiong Liu [a], Aiping Qin [b], Taijie Li [a], Xue Qin [a], Shan Li [a,*]

[a] Department of Clinical Laboratory, First Affiliated Hospital of Guangxi Medical University, Nanning, Guangxi, China
[b] Department of Obstetrics and Gynecology and Reproductive center, First Affiliated Hospital of Guangxi Medical University, Nanning, Guangxi, China





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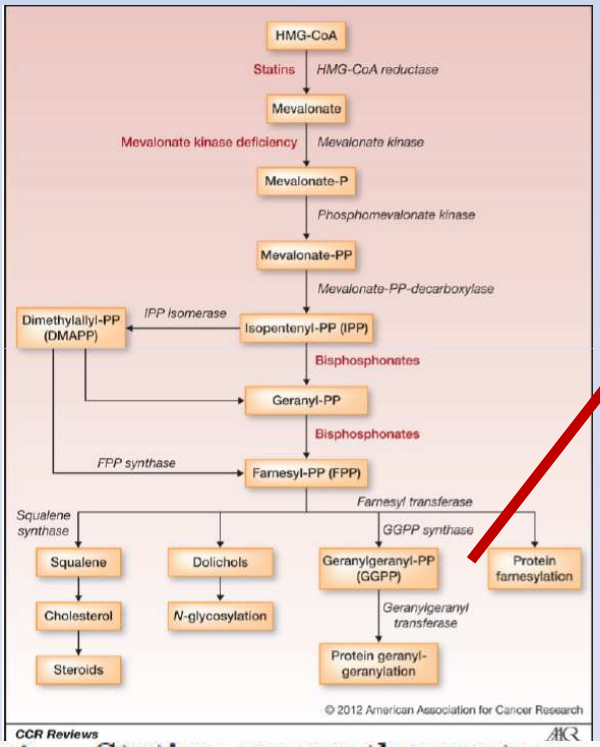
ASSOCIATE EDITOR: MICHAEL M. GOTTESMAN

Pharmacological Actions of Statins: A Critical Appraisal in the Management of Cancer

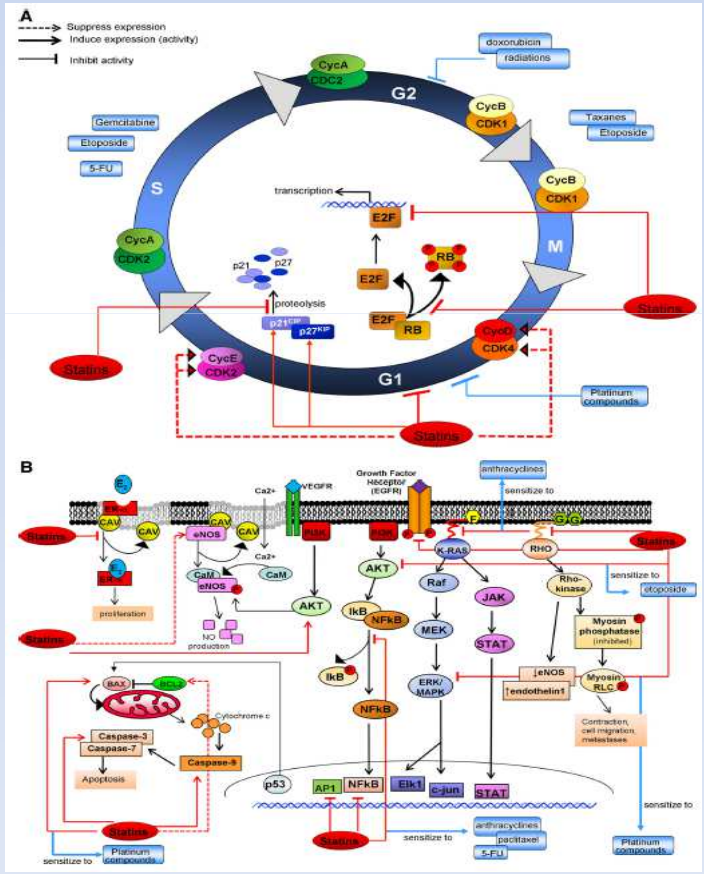
Patrizia Gazzerro, Maria Chiara Proto, Giuseppina Gangemi, Anna Maria Malfitano, Elena Ciaglia, Simona Pisanti, Antonietta Santoro, Chiara Laezza, and Maurizio Bifulco

Department of Pharmaceutical and Biomedical Sciences, University of Salerno, Fisciano, Italy (P.G., M.C.P., G.G., A.M.M., E.C., S.P., A.S., M.B.); and Istituto di Endocrinologia e Oncologia Sperimentale, Consiglio Nazionale delle Ricerche, Napoli, Italy (C.L.)



***Abstract*——Statins, among the most commonly prescribed drugs worldwide, are cholesterol-lowering agents used to manage and prevent cardiovascular and coronary heart diseases.**

Statins have been shown to act through cholesterol-dependent and -independent mechanisms and are able to affect several tissue functions and modulate specific signal transduction pathways that could account for statin pleiotropic effects.



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THE PERFECT ANTICANCER AGENT

MUST BE ABLE TO:

inhibit cell growth
induce apoptosis
inhibit neo-angiogenesis
inhibit cells migration and implantation
strengthen the immune system
avoid drug resistance
no side effects

AND PREFERABLY TO

potentiate the effects of radiation
potentiate the effects of other anticancer agents

The Oncologist®
Prevention

The Role of Statins in Cancer Therapy

KATJA HINDLER,[a] CHARLES S. CLEELAND,[b] EDGARDO RIVERA,[c] CHARLES D. COLLARD[a]

[a]Division of Cardiovascular Anesthesiology, Texas Heart® Institute, St. Luke's Episcopal Hospital, Houston, Texas, USA; [b]Department of Symptom Research and [c]Department of Breast Medical Oncology, University of Texas M.D. Anderson Cancer Center, Houston, Texas, USA

Key Words. HMG-CoA reductase inhibitor • Tumor • Chemotherapy • Inflammation • Prevention

LEARNING OBJECTIVES

After completing this course, the reader will be able to:

1. Explain how statins, used in the treatment of hypercholesterolemia, may be applicable to cancer prevention.
2. Discuss how statins potentially interfere with biologic processes relevant to cancer etiology.
3. Outline the gaps in our understanding in this area of theoretical versus applied medicine.

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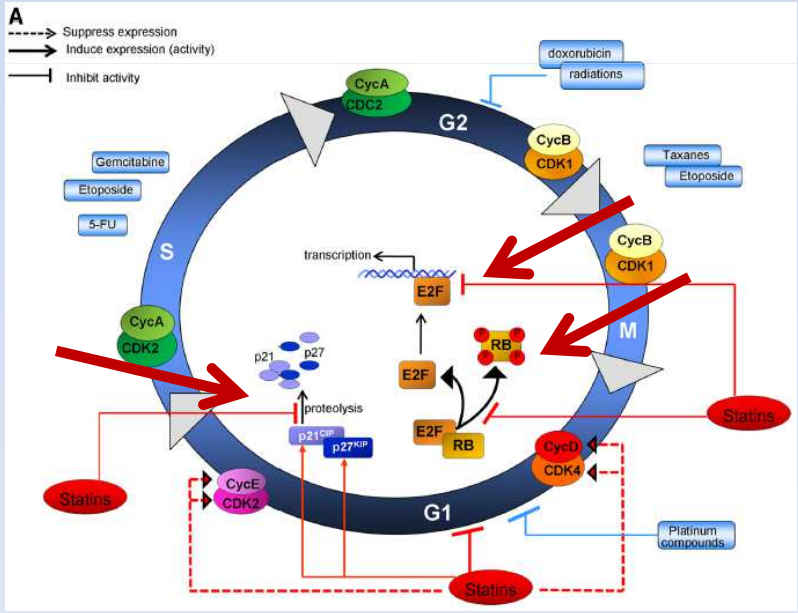

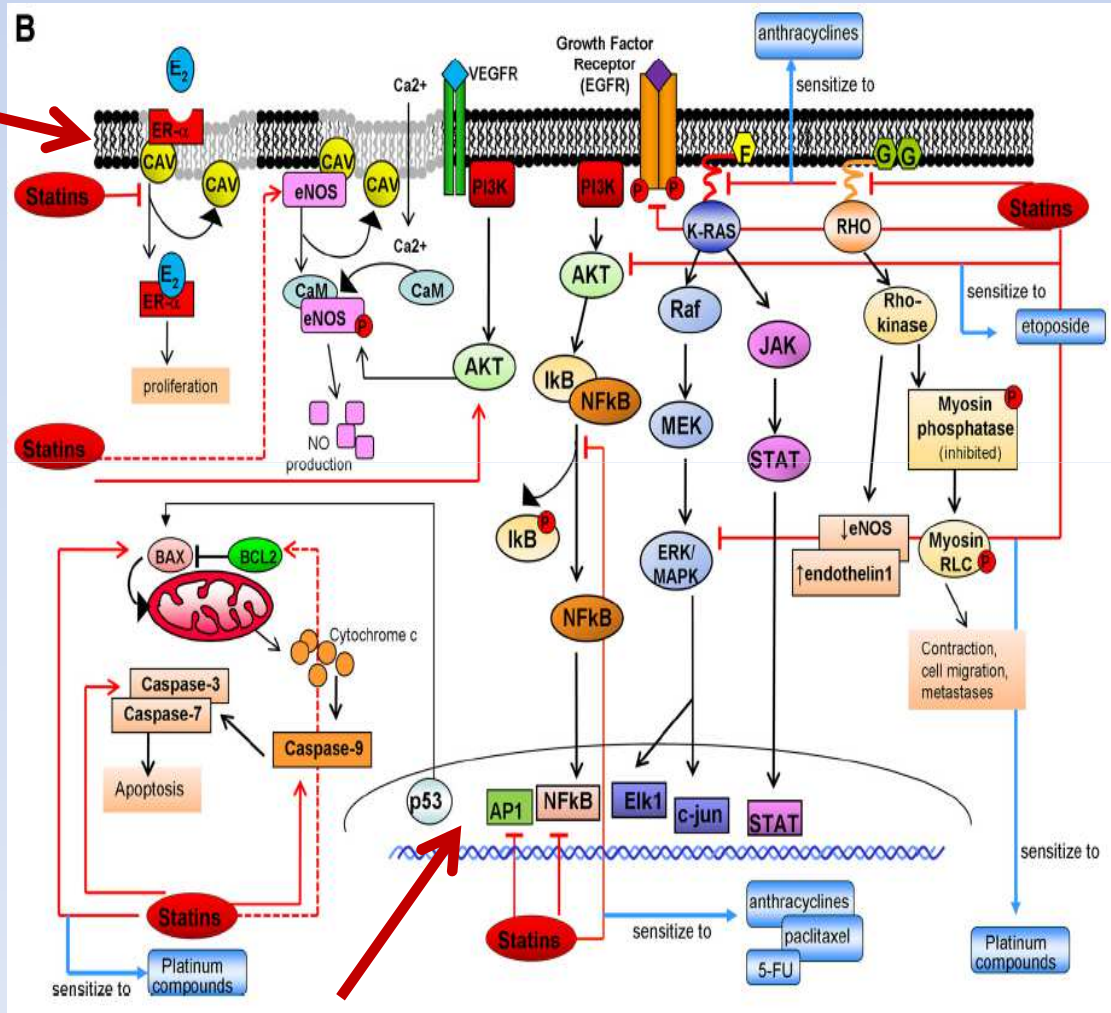

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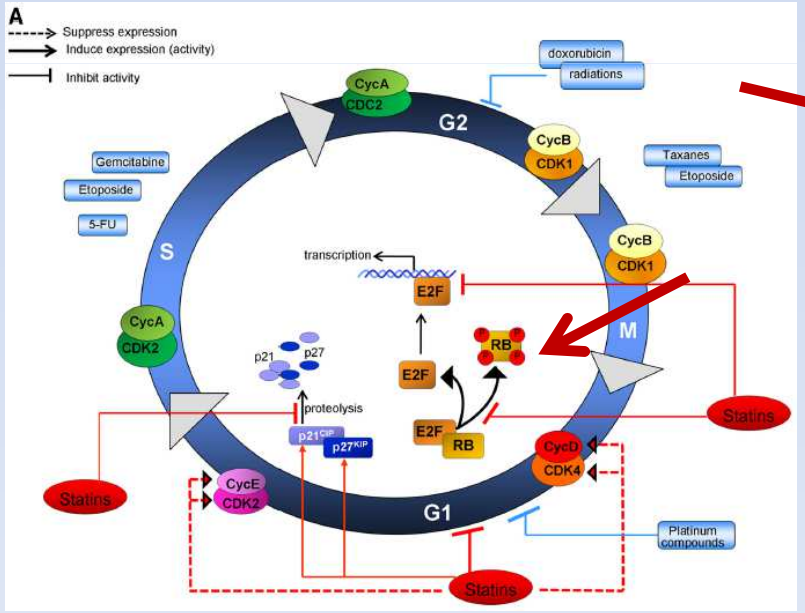

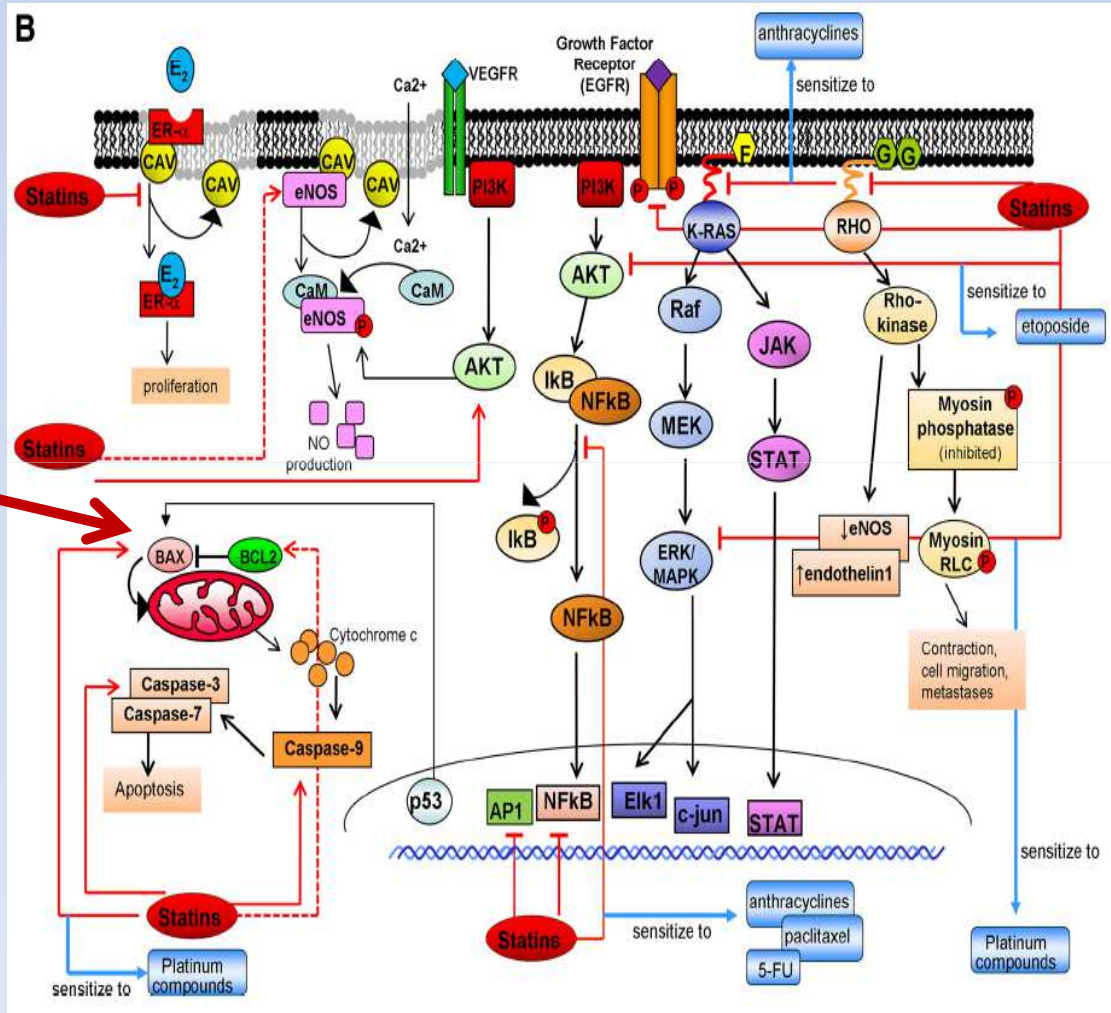

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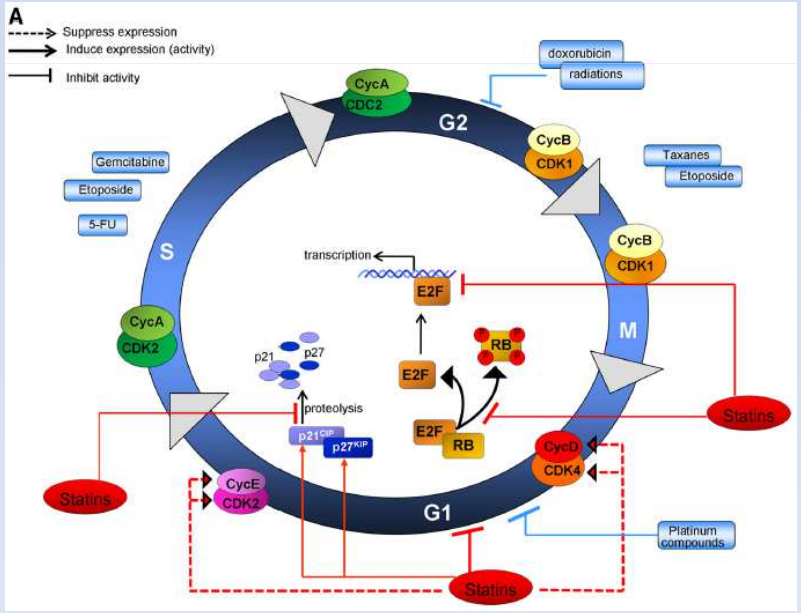

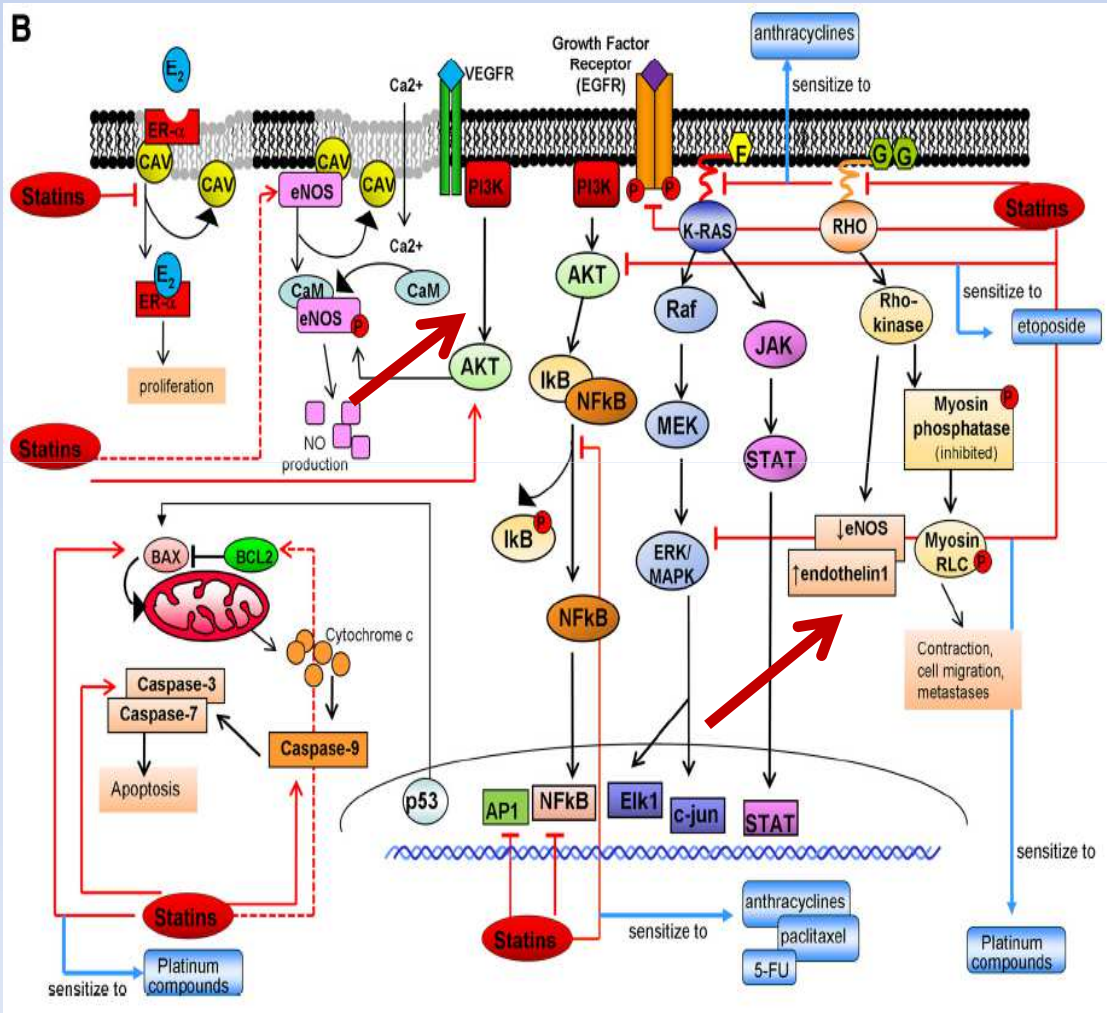

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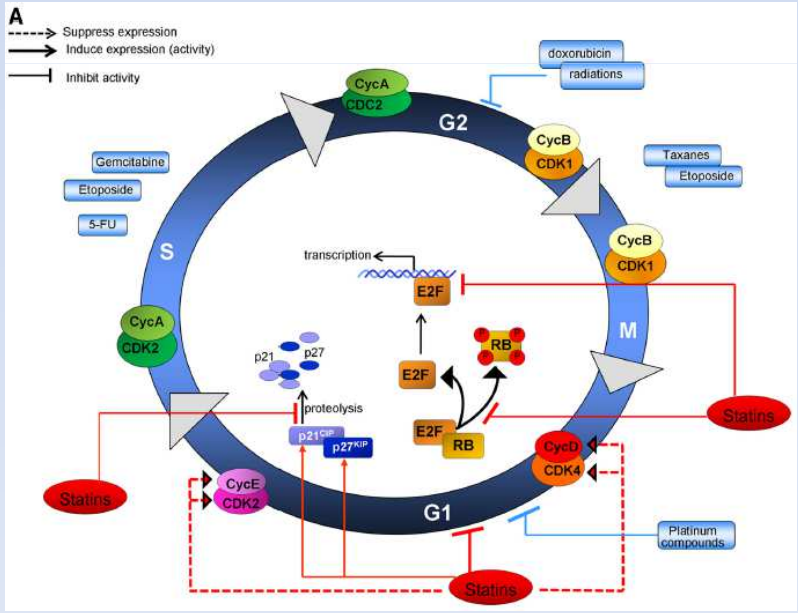

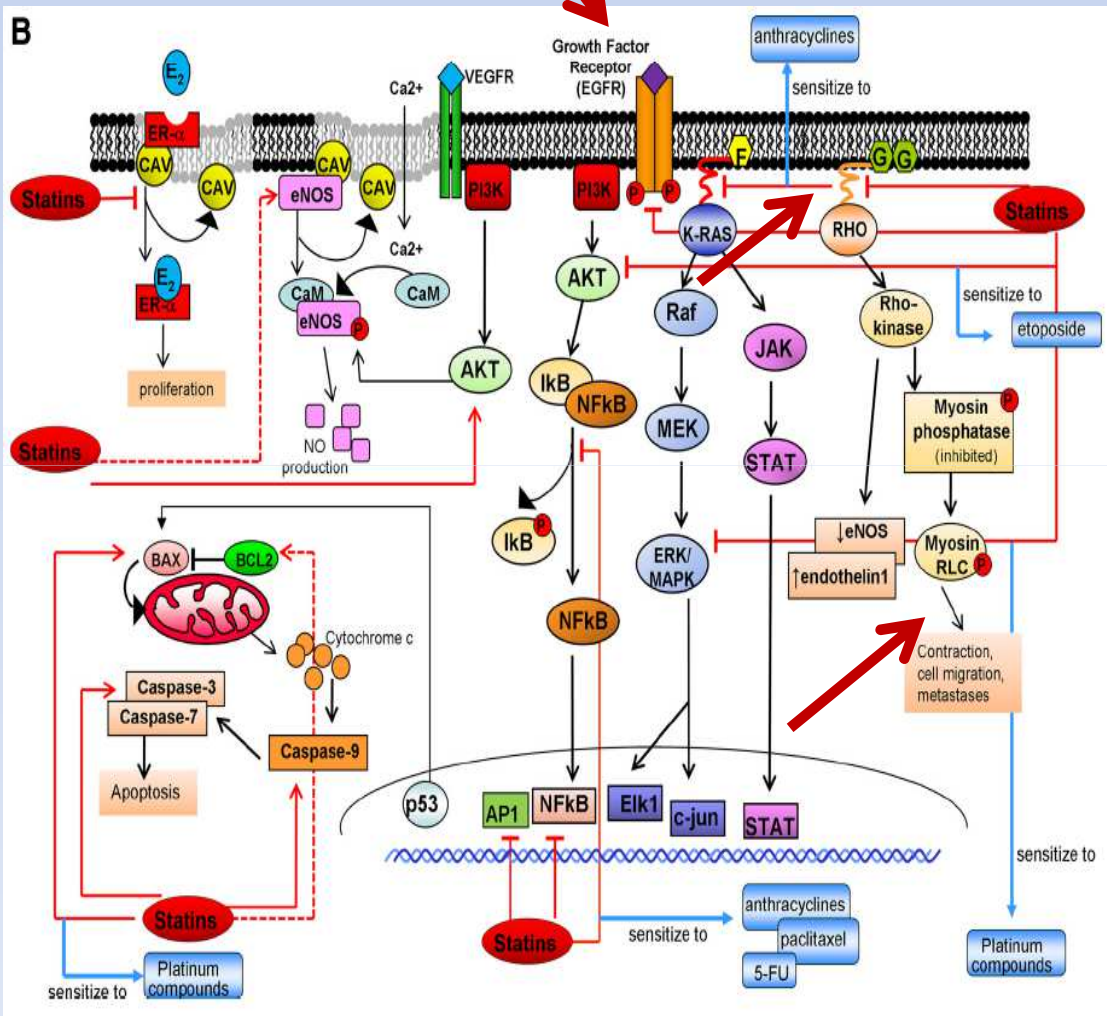

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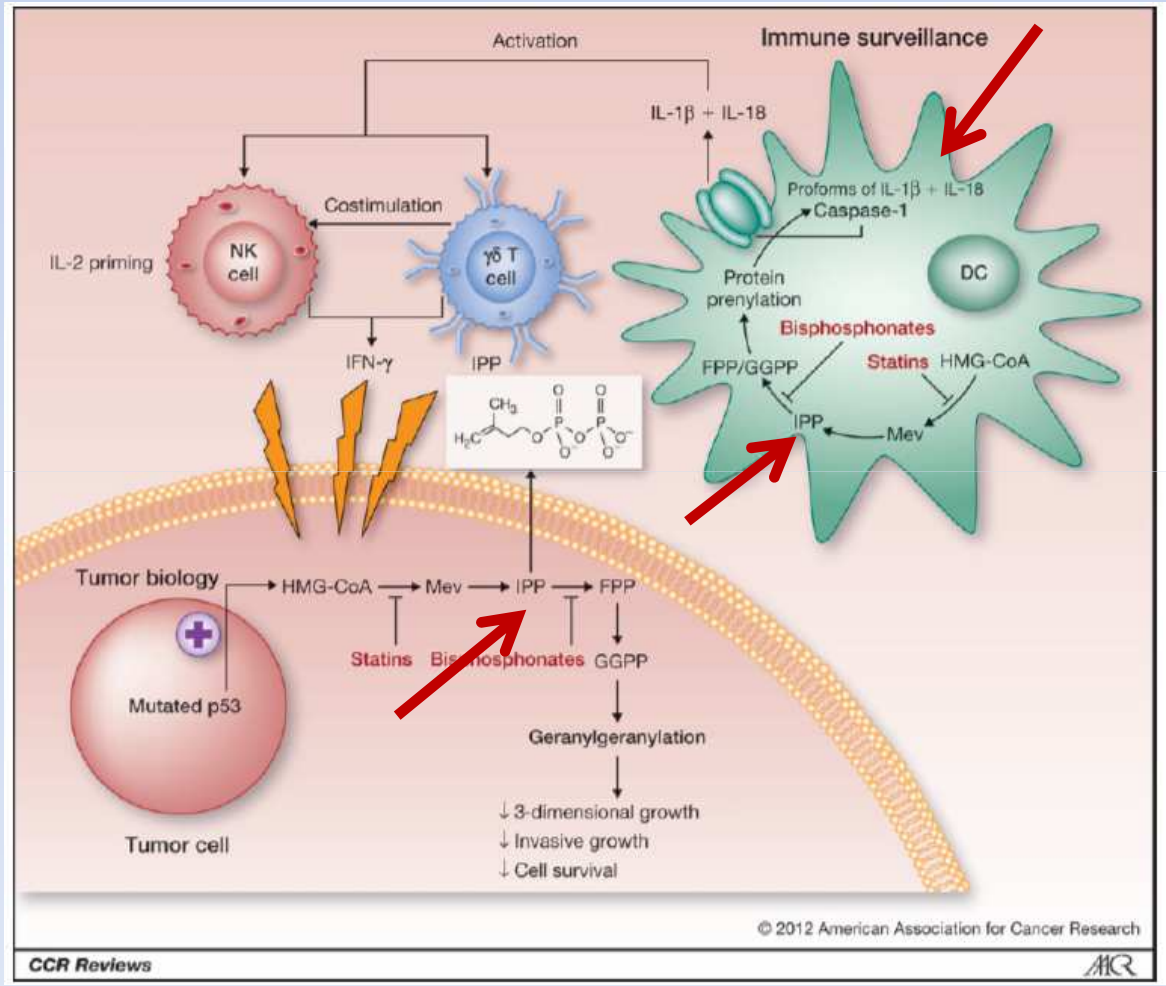
SIMILAR MECHANISM

BUT

ANTITHETICAL EFFECT

RESPECT TO

ATHEROMATOUS PLAQUE



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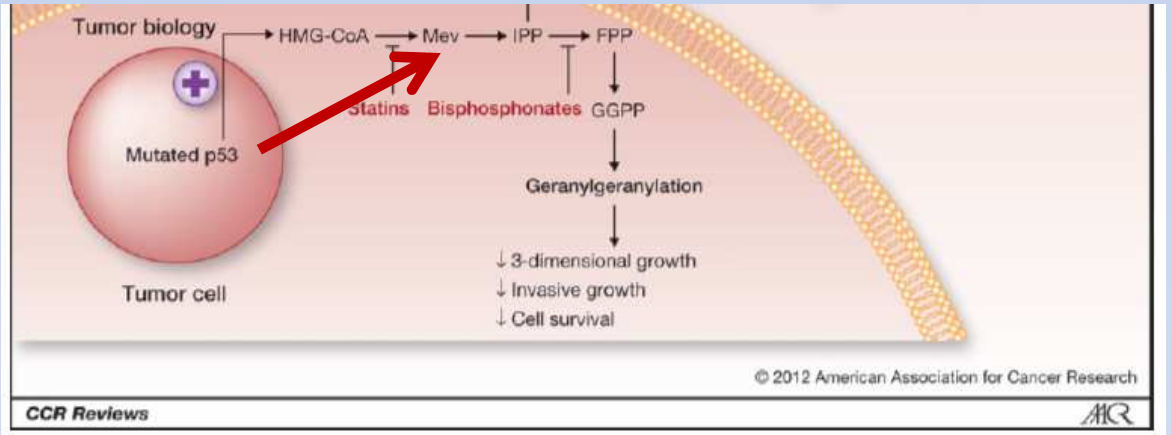

Enhanced mevalonate metabolism and statin sensitivity in cancer cells carrying mutant p53

Recent work showed that mutant p53, which is present in more than half of all human cancers, can significantly upregulate mevalonate pathway activity in cancer cells (Fig. 2), which contributes to maintenance of the malignant phenotype (13). Simvastatin used at clinically achievable

phenotype (13). Simvastatin used at clinically achievable concentrations was shown to reduce 3-dimensional growth of cancer cells expressing a single mutant p53 allele. Moreover, simvastatin was able to induce extensive cancer cell death in these cells and a significant reduction of their invasive phenotype. Intriguingly, the morphologic changes

not observed in wild-type p53-expressing cells. In isoprenoid add-back experiments, supplementation with GGPP was sufficient to restore the invasive phenotype in the presence of HMG-CoA reductase inhibition, showing

Dott. S. Gizzo

THE PERFECT ANTICANCER AGENT

MUST BE ABLE TO:

inhibit cell growth
induce apoptosis
inhibit neo-angiogenesis
inhibit cells migration and implantation
strengthen the immune system
avoid drug resistance
no side effects

AND PREFERABLY TO

potentiate the effects of radiation
potentiate the effects of other anticancer agents

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Eur J Prev Cardiol. 2014 Apr;21(4):464-74. doi: 10.1177/2047487314525531. Epub 2014 Mar 12.

What proportion of symptomatic side effects in patients taking statins are genuinely caused by the drug? Systematic review of randomized placebo-controlled trials to aid individual patient choice.

Finegold JA[1], Manisty CH, Goldacre B, Barron AJ, Francis DP.

Author information

Abstract

OBJECTIVE: Discussions about statin efficacy in cardiovascular prevention are always based on data from blinded randomized controlled trials (RCTs) comparing statin to placebo; however, discussion of side effects is not. Clinicians often assume symptoms occurring with statins are caused by statins, encouraging discontinuation. We test this assumption and calculate an evidence-based estimate of the probability of a symptom being genuinely attributable to the statin itself.

METHODS: We identified RCTs comparing statin to placebo for cardiovascular prevention that reported side effects separately in the two arms.

RESULTS: Among 14 primary prevention trials (46,262 participants), statin therapy increased diabetes by absolute risk of 0.5% (95% CI 0.1-1%, p = 0.012), meanwhile reducing death by a similar extent: -0.5% (-0.9 to -0.2%, p = 0.003). In the 15 secondary prevention RCTs (37,618 participants), statins decreased death by 1.4% (-2.1 to -0.7%, p < 0.001). There were no other statin-attributable symptoms, although asymptomatic liver transaminase elevation was 0.4% more frequent with statins across all trials. Serious adverse events and withdrawals were similar in both arms.

CONCLUSIONS: Only a small minority of symptoms reported on statins are genuinely due to the statins: almost all would occur just as frequently on placebo. Only development of new-onset diabetes mellitus was significantly higher on statins than placebo; nevertheless only 1 in 5 of new cases were actually caused by statins. Higher statin doses produce a detectable effect, but even still the proportion attributable to statins is variable: for asymptomatic liver enzyme elevation, the majority are attributable to the higher dose; in contrast for muscle aches, the majority are not.

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Radiotherapy and Oncology 92 (2009) 492–499

Contents lists available at ScienceDirect

Radiotherapy and Oncology

journal homepage: www.thegreenjournal.com

Experimental radiobiology

Lovastatin attenuates ionizing radiation-induced normal tissue damage *in vivo*

Christian Ostrau [a,1], Johannes Hülsenbeck [a,1], Melanie Herzog [a], Arno Schad [b], Michael Torzewski [c], Karl J. Lackner [c], Gerhard Fritz [a,*]

[a] Department of Toxicology, University Medical Center of the Johannes Gutenberg University Mainz, Germany
[b] Institute of Pathology, University Medical Center of the Johannes Gutenberg University Mainz, Germany
[c] Institute of Clinical Chemistry and Laboratory Medicine, University Medical Center of the Johannes Gutenberg University Mainz, Germany

ABSTRACT

Background and purpose: HMG-CoA-reductase inhibitors (statins) are widely used lipid-lowering drugs. Moreover, they have pleiotropic effects on cellular stress responses, proliferation and apoptosis *in vitro*. Here, we investigated whether lovastatin attenuates acute and subchronic ionizing radiation-induced normal tissue toxicity *in vivo*.

Materials and methods: Four hours to 24 h after total body irradiation (6 Gy) of Balb/c mice, acute pro-inflammatory and pro-fibrotic responses were analyzed. To comprise subchronic radiation toxicity, mice were irradiated twice with 2.5 Gy and analyses were performed 3 weeks after the first radiation treatment. Molecular markers of inflammation and fibrosis as well as organ toxicities were measured.

Results: Lovastatin attenuated IR-induced activation of NF-κB, mRNA expression of cell adhesion molecules and mRNA expression of pro-inflammatory and pro-fibrotic marker genes (i.e. TNFα, IL-6, TGFβ, CTGF, and type I and type III collagen) in a tissue- and time-dependent manner. γH2AX phosphorylation stimulated by IR was not affected by lovastatin, indicating that the statin has no major impact on the induction of DNA damage *in vivo*. Radiation-induced thrombopenia was significantly alleviated by lovastatin.

Conclusions: Lovastatin inhibits both acute and subchronic IR-induced pro-inflammatory and pro-fibrotic responses and cell death in normal tissue *in vivo*. Therefore, lovastatin might be useful for selectively attenuating acute and subchronic normal tissue damage caused by radiotherapy.

 Radiotherapy and Oncology 92 (2009) 492–499

ORIGINAL ARTICLE

Lovastatin Sensitizes Lung Cancer Cells to Ionizing Radiation

Modulation of Molecular Pathways of Radioresistance and Tumor Suppression

Toran Sanli, MSc,† Caiqiong Liu, MSc,* Ayesha Rashid, MSc,*† Sarah N. Hopmans, MSc,‡ Evangelia Tsiani, PhD,§ Carrie Schultz, BSc,# Thomas Farrell, PhD,‖# Gurmit Singh, PhD,‡ James Wright, MD,#† and Theodoros Tsakiridis, MD, PhD*†#*

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Overcoming Tumor Multidrug Resistance Using Drugs Able to Evade P-Glycoprotein or to Exploit Its Expression

Stefania Nobili, Ida Landini, Teresita Mazzei, and Enrico Mini

Department of Preclinical and Clinical Pharmacology, University of Florence, Florence, Italy, Viale Pieraccini, 6-50139, Firenze, Italy

Published online 3 March 2011 in Wiley Online Library (wileyonlinelibrary.com).
DOI 10.1002/med.20239

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International Journal of Cancer

Differential interactions between statins and P-glycoprotein: implications for exploiting statins as anticancer agents

Carolyn A. Goard[1,2], Richard G. Mather[3], Balpreet Vinepal[3], James W. Clendening[1,2], Anna Martirosyan[2], Paul C. Boutros[4], Frances J. Sharom[3] and Linda Z. Penn[1,2]

[1] Department of Medical Biophysics, University of Toronto, Toronto, ON, Canada
[2] Division of Cancer Genomics and Proteomics, Ontario Cancer Institute, Toronto, ON, Canada
[3] Department of Molecular and Cell Biology, University of Guelph, Guelph, ON, Canada
[4] Bioinformatics and Biocomputing Platform, Ontario Institute for Cancer Research, Toronto, ON, Canada

Statins, prescribed for decades to control cholesterol, have more recently been shown to have promising anticancer activity. Statins induce tumor-selective apoptosis by inhibiting the mevalonate (MVA) pathway. In addition, we have recently demonstrated that lovastatin modulates drug accumulation in a MVA-independent manner in multidrug-resistant (MDR) tumor cells overexpressing the P-glycoprotein (P-gp) multidrug transporter. P-gp-mediated drug efflux can contribute to chemotherapy failure.

Martirosyan *et al. BMC Cancer* 2010, **10**:103
http://www.biomedcentral.com/1471-2407/10/103

BMC Cancer

RESEARCH ARTICLE Open Access

Lovastatin induces apoptosis of ovarian cancer cells and synergizes with doxorubicin: potential therapeutic relevance

Anna Martirosyan[†1], James W Clendening[†1,2], Carolyn A Goard[1,2] and Linda Z Penn[*1,2]

Dott. S. Gizzo

The International Journal of Biochemistry & Cell Biology 45 (2013) 745–752

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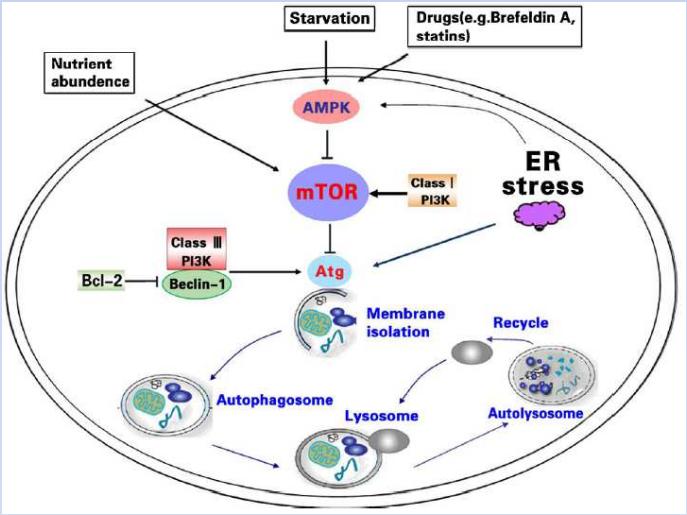
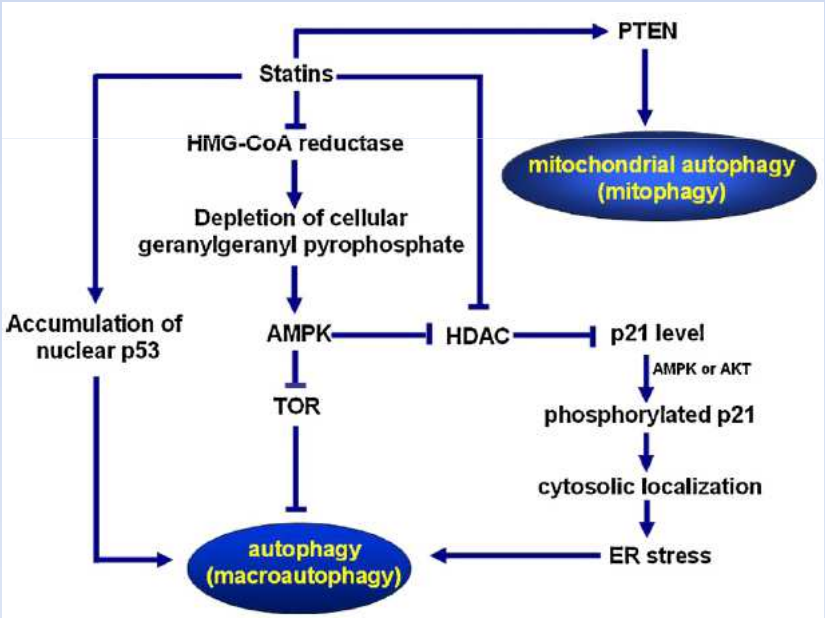
journal homepage: www.elsevier.com/locate/biocel

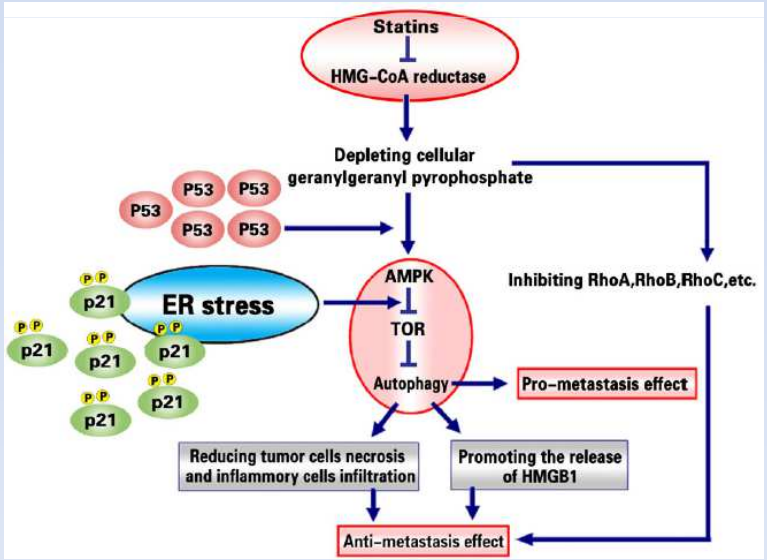
Review

Statins, autophagy and cancer metastasis

Jing Zhang[a,1], Zuozhang Yang[a,*,1], Lin Xie[b,1], Lei Xu[a], Da Xu[a], Xuefeng Liu[a]







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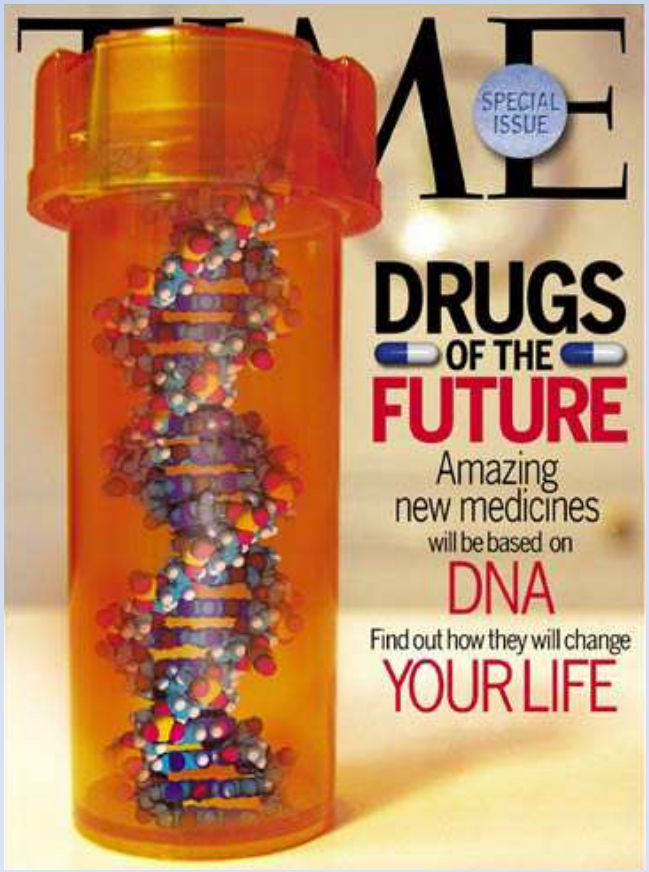
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Cancer Chemother Pharmacol (2009) 63:997–1005
DOI 10.1007/s00280-008-0830-7

ORIGINAL ARTICLE

Statins induce apoptosis in ovarian cancer cells through activation of JNK and enhancement of Bim expression

Hongli Liu · Shu-Ling Liang · Sheetal Kumar · Crystal M. Weyman · Wendy Liu · Aimin Zhou

J. Cell. Mol. Med. Vol 14, No 5, 2010 pp. 1180-1193

Lipophilic but not hydrophilic statins selectively induce cell death in gynaecological cancers expressing high levels of HMGCoA reductase

S. Kato [a], S. Smalley [a], A. Sadarangani [b], K. Chen-Lin [a], B. Oliva [b], J. Brañes [a], J. Carvajal [a], R. Gejman [c], G. I. Owen [b], M. Cuello [a, *]

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[b] Department of Physiology, Faculty of Biological Sciences, Pontificia Universidad Católica de Chile, Santiago, Chile
[c] Department of Pathology, Faculty of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile

Overexpression of RhoA enhances peritoneal dissemination: RhoA suppression with Lovastatin may be useful for ovarian cancer

Akiko Horiuchi,[1,4] Norihiko Kikuchi,[1] Ryosuke Osada,[1] Cuiju Wang,[1] Akiko Hayashi,[1] Toshio Nikaido[2] and Ikuo Konishi[3]

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University, Faculty of Medicine, Toyama 930-0194, [3]Department of Gynecology and Obstetrics, Kyoto University Gradua

Brennan et al. BMC Cancer 2010, 10:125
http://www.biomedcentral.com/1471-2407/10/125

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Research article

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Activity of mevalonate pathway inhibitors against breast and ovarian cancers in the ATP-based tumour chemosensitivity assay

Louise A Knight[1], Christian M Kurbacher[2], Sharon Glaysher[1], Augusta Fernando[1], Ralf Reichelt[2], Susanne Dexel[2], Uwe Reinhold[3] and Ian A Cree*[1]

RESEARCH ARTICLE Open Access

Tumour-specific HMG-CoAR is an independent predictor of recurrence free survival in epithelial ovarian cancer

Donal J Brennan[1,2*], Jenny Brändstedt[3], Elton Rexhepaj[2], Michael Foley[4], Fredrik Pontén[5], Mathias Uhlén[6], William M Gallagher[2], Darran P O'Connor[2], Colm O'Herlihy[4], Karin Jirstrom[3,7]

Dott. S. Gizzo

Science AAAS

Title Page

It's time to consider the lipophilic statins a cost-effective tool to improve post-surgical outcome of advanced stage ovarian and endometrial malignancies?

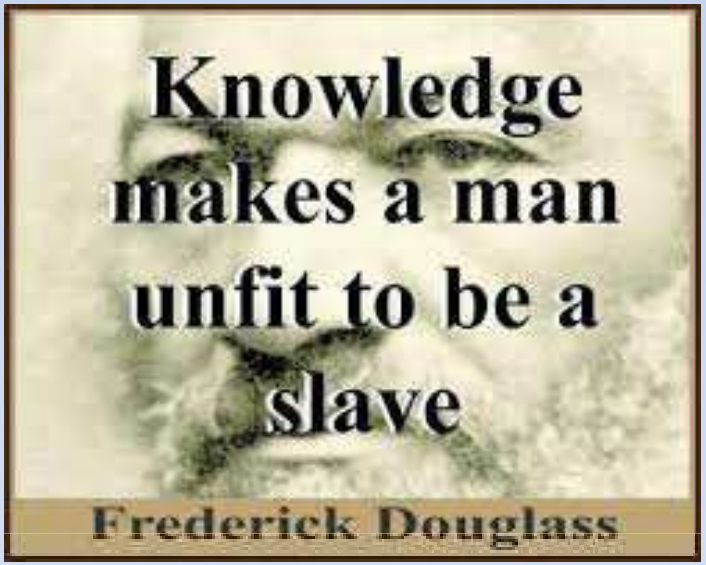
Hypothesis, rationale and study proposal.

Salvatore Gizzo[1]M.D; Marco Noventa[1]M.D; Emanuele Ancona[1]M.D; Giovanni Battista Nardelli[1]M.D.

1 - Department of Woman and Child Health – University of Padua, Padua, Italy

Running Title: statin use as anticancer agent

Keywords: statin treatment, anticancer agents, adjuvant therapy, oncological outcome, targeted therapy, gynaecological malignancy, advanced stage.



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