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OMICS Group has been instrumental in taking the knowledge on Science & technology to the doorsteps of ordinary men and women. Research Scholars, Students, Libraries, Educational Institutions, Research centers and the industry are main stakeholders that benefitted greatly from this knowledge dissemination. OMICS Group also organizes 500 International conferences annually across the globe, where knowledge transfer takes place through debates, round table discussions, poster presentations, workshops, symposia and exhibitions.

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OMICS International is a pioneer and leading science event organizer, which publishes around 500 open access journals and conducts over 500 Medical, Clinical, Engineering, Life Sciences, Pharma scientific conferences all over the globe annually with the support of more than 1000 scientific associations and 30,000 editorial board members and 3.5 million followers to its credit.

OMICS Group has organized 500 conferences, workshops and national symposiums across the major cities including San Francisco, Las Vegas, San Antonio, Omaha, Orlando, Raleigh, Santa Clara, Chicago, Philadelphia, Baltimore, United Kingdom, Valencia, Dubai, Beijing, Hyderabad, Bengaluru and Mumbai.

OMICS International

3rd International Conference On Integrative Biology

The role of microRNA-125b in calcitriol-induced differentiation of human leukemia and lymphoma cells

Justyna Trynda

Ludwik Hirszfeld Institute of Immunology and Experimental Therapy
Polish Academy of Sciences
Wroclaw, Poland

4-6 August 2015
Valencia, Spain

Adults leukemia and lymphoma incidents in 2014

Leading New Cancer Cases and Deaths – 2014 Estimates

Estimated New Cases*		Estimated Deaths	
Male	**Female**	**Male**	**Female**
Prostate 233,000 (27%)	Breast 232,670 (29%)	Lung & bronchus 86,930 (28%)	Lung & bronchus 72,330 (26%)
Lung & bronchus 116,000 (14%)	Lung & bronchus 108,210 (13%)	Prostate 29,480 (10%)	Breast 40,000 (15%)
Colon & rectum 71,830 (8%)	Colon & rectum 65,000 (8%)	Colon & rectum 26,270 (8%)	Colon & rectum 24,040 (9%)
Urinary bladder 56,390 (7%)	Uterine corpus 52,630 (6%)	Pancreas 20,170 (7%)	Pancreas 19,420 (7%)
Melanoma of the skin 43,890 (5%)	Thyroid 47,790 (6%)	Liver & intrahepatic bile duct 15,870 (5%)	Ovary 14,270 (5%)
Kidney & renal pelvis 39,140 (5%)	Non-Hodgkin lymphoma 32,530 (4%)	Leukemia 14,040 (5%)	Leukemia 10,050 (4%)
Non-Hodgkin lymphoma 38,270 (4%)	Melanoma of the skin 32,210 (4%)	Esophagus 12,450 (4%)	Uterine corpus 8,590 (3%)
Oral cavity & pharynx 30,220 (4%)	Kidney & renal pelvis 24,780 (3%)	Urinary bladder 11,170 (4%)	Non-Hodgkin lymphoma 8,520 (3%)
Leukemia 30,100 (4%)	Pancreas 22,890 (3%)	Non-Hodgkin lymphoma 10,470 (3%)	Liver & intrahepatic bile duct 7,130 (3%)
Liver & intrahepatic bile duct 24,600 (3%)	Leukemia 22,280 (3%)	Kidney & renal pelvis 8,900 (3%)	Brain & other nervous system 6,230 (2%)
All sites 855,220 (100%)	All sites 810,320 (100%)	All sites 310,010 (100%)	All sites 275,710 (100%)

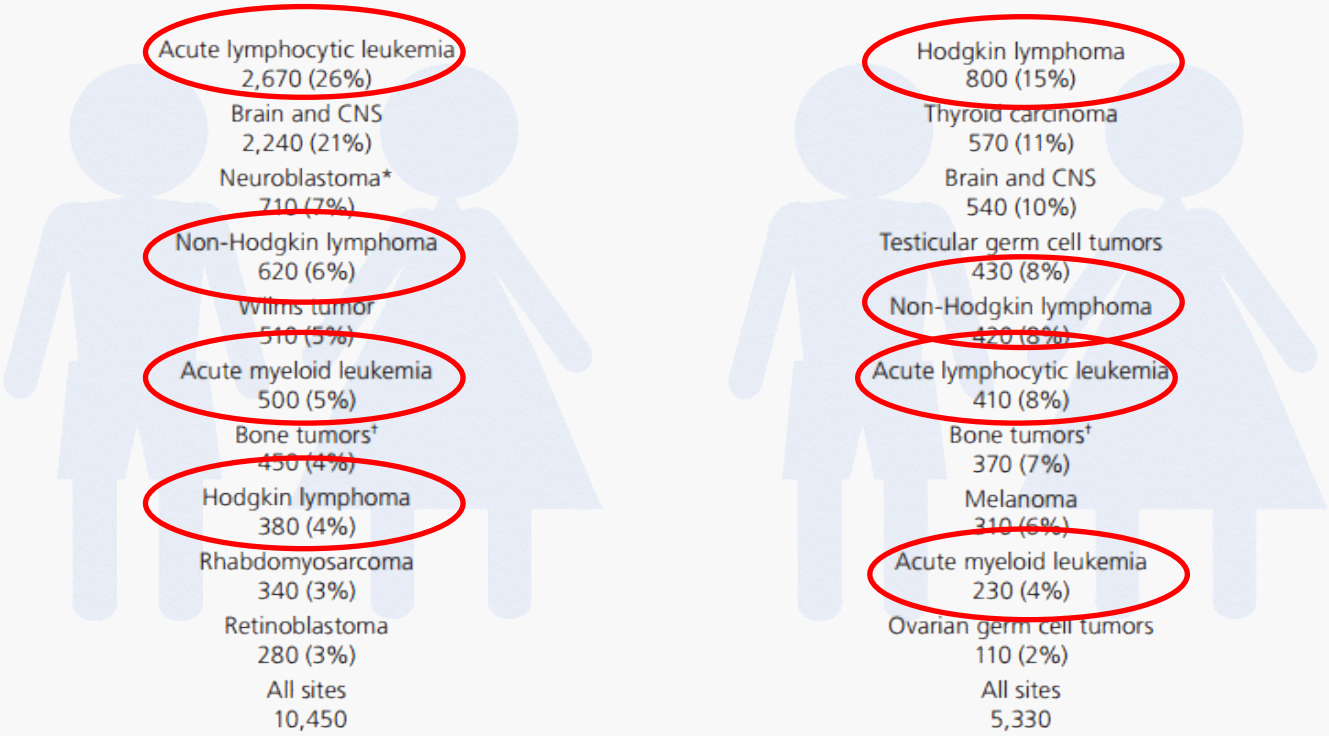
*Excludes basal and squamous cell skin cancers and in situ carcinoma except urinary bladder.

From 2006 to 2010, overall leukemia incidence rates increased slightly (by 0.5% per year).

http://www.cancer.org/acs/groups/content/@research/documents/webcontent/acspc-042151.pdf

The most common cancers among children and adolescents

Estimated Cases for Childhood and Adolescent Cancers, US, 2014

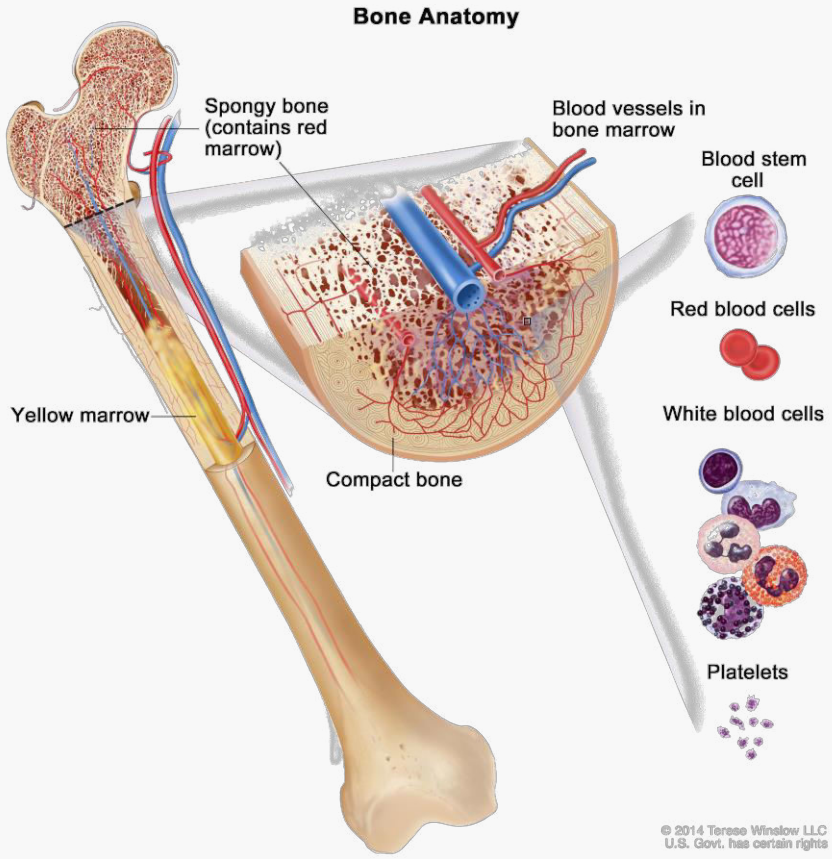


Estimates are for malignant cancers only and are rounded to the nearest 10. In addition, 730 children and 630 adolescents will be diagnosed with benign and borderline brain tumors in 2014.
CNS = central nervous system
* Includes ganglioneuroblastoma.
†Bone tumors include osteosarcoma and Ewing sarcoma.

About 40 % of all cancer incidents in children are leukemias and lymphomas

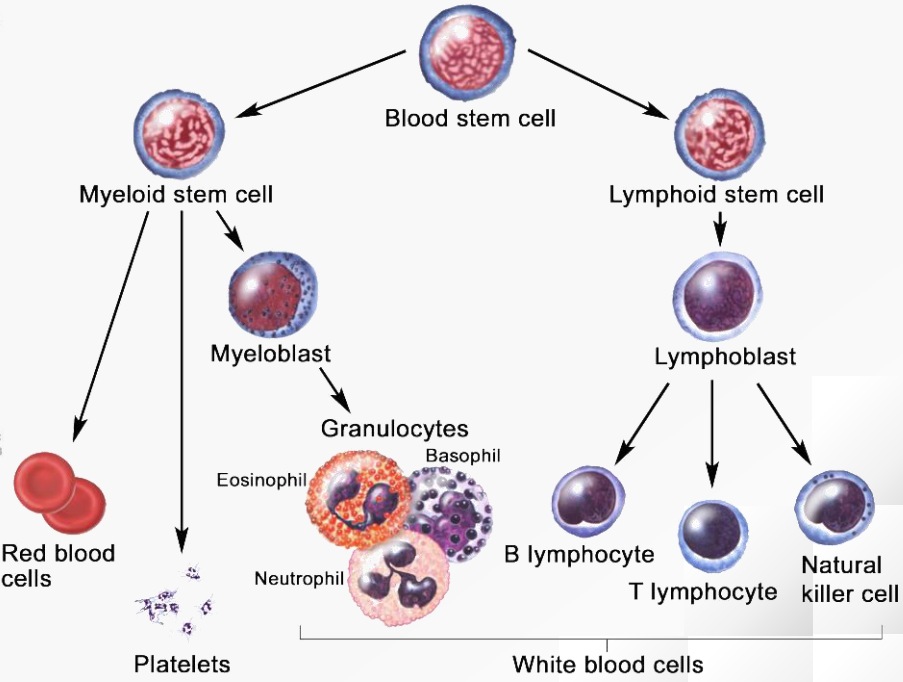
http://www.cancer.org/acs/groups/content/@research/documents/webcontent/acspc-042151.pdf

Hemopoiesis is the proliferation and differentiation of the formed elements of blood



Classification of leukemia and lymphoma:

- ✓ acute lymphocytic (ALL)
- ✓ chronic lymphocytic (CLL)
- ✓ acute myeloid (AML)
- ✓ chronic myeloid (CML)



Winslow T: Chronic Lymphocytic Leukemia Treatment. *Cancer* December 30, 2014

The human leukemia and lymphoma WHO classification

Table 2. WHO classification of myeloid neoplasms and acute leukemia

Myeloproliferative neoplasms (MPN)
- Chronic myelogenous leukemia, *BCR-ABL1*–positive
- Chronic neutrophilic leukemia
- Polycythemia vera
- Primary myelofibrosis
- Essential thrombocythemia
- Chronic eosinophilic leukemia, not otherwise specified
- Mastocytosis
- Myeloproliferative neoplasms, unclassifiable

Myeloid and lymphoid neoplasms associated with eosinophilia and abnormalities of *PDGFRA*, *PDGFRB*, or *FGFR1*
- Myeloid and lymphoid neoplasms associated with *PDGFRA* rearrangement
- Myeloid neoplasms associated with *PDGFRB* rearrangement
- Myeloid and lymphoid neoplasms associated with *FGFR1* abnormalities

Myelodysplastic/myeloproliferative neoplasms (MDS/MPN)
- Chronic myelomonocytic leukemia
- Atypical chronic myeloid leukemia, *BCR-ABL1*–negative
- Juvenile myelomonocytic leukemia
- Myelodysplastic/myeloproliferative neoplasm, unclassifiable
  - *Provisional entity: refractory anemia with ring sideroblasts and thrombocytosis*

Myelodysplastic syndrome (MDS)
- Refractory cytopenia with unilineage dysplasia
  - Refractory anemia
  - Refractory neutropenia
  - Refractory thrombocytopenia

Acute myeloid leukemia and related neoplasms
- Acute myeloid leukemia with recurrent genetic abnormalities
  - AML with t(8;21)(q22;q22); *RUNX1-RUNX1T1*
  - AML with inv(16)(p13.1q22) or t(16;16)(p13.1;q22); *CBFB-MYH11*
  - APL with t(15;17)(q22;q12); *PML-RARA*
  - AML with t(9;11)(p22;q23); *MLLT3-MLL*
  - AML with t(6;9)(p23;q34); *DEK-NUP214*
  - AML with inv(3)(q21q26.2) or t(3;3)(q21;q26.2); *RPN1-EVI1*
  - AML (megakaryoblastic) with t(1;22)(p13;q13); *RBM15-MKL1*
  - *Provisional entity: AML with mutated NPM1*
  - *Provisional entity: AML with mutated CEBPA*
- Acute myeloid leukemia with myelodysplasia-related changes
- Therapy-related myeloid neoplasms
- Acute myeloid leukemia, not otherwise specified
  - AML with minimal differentiation
  - AML without maturation
  - AML with maturation
  - Acute myelomonocytic leukemia
  - Acute monoblastic/monocytic leukemia
  - Acute erythroid leukemia
    - Pure erythroid leukemia
    - Erythroleukemia, erythroid/myeloid
  - Acute megakaryoblastic leukemia
  - Acute basophilic leukemia
  - Acute panmyelosis with myelofibrosis
- Myeloid sarcoma
- Myeloid proliferations related to Down syndrome
  - Transient abnormal myelopoiesis
  - Myeloid leukemia associated with Down syndrome
- Blastic plasmacytoid dendritic cell neoplasm

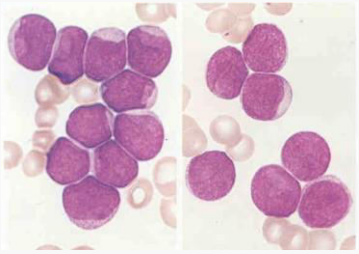


Table 2. WHO classification of myeloid neoplasms and acute leukemia (continued)

Acute leukemias of ambiguous lineage
- Acute undifferentiated leukemia
- Mixed phenotype acute leukemia with t(9;22)(q34;q11.2); *BCR-ABL1*
- Mixed phenotype acute leukemia with t(v;11q23); *MLL* rearranged
- Mixed phenotype acute leukemia, B-myeloid, NOS
- Mixed phenotype acute leukemia, T-myeloid, NOS
- *Provisional entity: natural killer (NK) cell lymphoblastic leukemia/lymphoma*

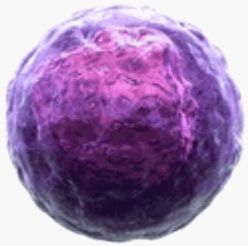
B lymphoblastic leukemia/lymphoma
- B lymphoblastic leukemia/lymphoma, NOS
- B lymphoblastic leukemia/lymphoma with recurrent genetic abnormalities
  - B lymphoblastic leukemia/lymphoma with t(9;22)(q34;q11.2);*BCR-ABL 1*
  - B lymphoblastic leukemia/lymphoma with t(v;11q23);*MLL* rearranged
  - B lymphoblastic leukemia/lymphoma with t(12;21)(p13;q22) *TEL-AML1* (*ETV6-RUNX1*)
  - B lymphoblastic leukemia/lymphoma with hyperdiploidy
  - B lymphoblastic leukemia/lymphoma with hypodiploidy
  - B lymphoblastic leukemia/lymphoma with t(5;14)(q31;q32) *IL3-IGH*
  - B lymphoblastic leukemia/lymphoma with t(1;19)(q23;p13.3);*TCF3-PBX1*

T lymphoblastic leukemia/lymphoma

The WHO classification system is based on recently published, peer-reviewed data

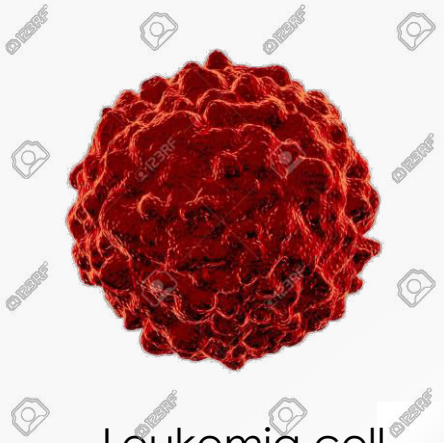
Vardiman J. et al.: The 2008 revision of the World Health Organization (WHO) classification of myeloid neoplasms and acute leukemia: rationale and important changes Blood Jul 2009, 114 (5) 937-951; DOI: 10.1182/blood-2009-03-209262

What causes leukemia and lymphoma ?



Progenitor cell

- Genetic aberrations
- Signal transduction
- Transcription factors
- Growth factors
- Epigenetic disorder
- **miRNA**



Leukemia cell

miRNAs have important role in the development of chemosensitivity or chemoresistance in cancer including leukemia and lymphoma

Treatments of human leukemia and lymphoma

AML Acute myeloid leukemia	CML Chronic myelogenous leukemia	Lymphoblastic leukemias and lymphomas
• idarubicin • cytosine arabinoside	• tyrosine kinase inhibitors imatinib (Gleevec), dasatinib	• antimetabolites; 6 - mercaptopurine, fludarabine, methotrexate • topoisomerase inhibitors; idarubicin, daunorubicin and doxorubicin, etoposide • alkylating agents; cyclophosphamide • allkaloidy vinca; vincristine and vinblastine • monoclonal antibodies; rituximab , alemtuzumab, gemtuzymab

When there are no signs of leukemia for 5 years, a person is usually considered cured. But if the leukemia doesn't go into remission, or if it comes back within the first few years, treatments may include more chemotherapy, a stem cell transplant, or joining a clinical trial for new treatments

The role of vitamin D in the inhibition of malignant cell proliferation in hematological malignancies is indicative of its future use in cancer therapy

Clinical trials of vitamin D, derivatives, analogs in high dose treatments and combination treatments.

Treatment	Malignancy	No. of patients	Administration	Conclusion	Ref.
Vitamin $1,25(OH)_2D_3$	MDS	18	Oral, 2 μg/day	Seven patients developed leukemia by end of 12 weeks; eight patients developed hypercalcemia; no enduring therapeutic effect	[119]
Low dose ara-C, interferon-α, Vitamin $1(OH)D_3$, retinoic acid combination	MDS, AML	62		A 50% responded favorably to the combination of IFN, Vit D_3 and retinoic acid; marrow hypoplasia seen only in 5 out of 27 patients; treatment potentially toxic	[118]
Vitamin $1(OH)D_3$	MDS, AML, CML	8, 2, 1	Oral, 0.25–10 μg/day	Three patients had partial response, three patients had minor response, rest of the patients did not respond; hematological improvement of 6 responders lasted 1 to 2 months; no hypercalcemia seen	[121]
Low dose ara-C, 13-cis-retinoic acid (13-CRA), Vitamin $1(OH)D_3$	MDS, AML	63, 15		18 (26.1%) Responded to therapy; disease progressed from MDS to AML in 12 out of 27 patients receiving only ara-C; disease progressed in 6 out of 29 patients receiving 13-CRA and $1(OH)D_3$; therapeutic effects of 13-CRA and $1(OH)D_3$ on MDS not supported by this study	[122]
Vitamin $1(OH)D_3$ (Alfacalcidol)	NHL	34	Oral, 1 μg/day	Complete response seen in four patients, partial response seen in four patients; 24% overall response rate; median duration of response 14 months; stabilization of disease in 10 patients (29%), progression to tumor in 16 patients (47%)	[123]
Low doses of ARA-C, Vitamin $1(OH)D_3$	AML			A 17% complete remission, 45% reached only a partial remission; cell differentiation seen in 7 out of 11 patients	[115]
Prednisone, Vitamin $1,25(OH)_2D_3$, 13-cis-retinoic acid	MDS			Long-lasting hematological remission	[124]
Vitamin D_3	MDS	30	Oral, 4–6 μg/day	7 out of 15 in nontreatment group developed acute leukemia, versus only 1 out of 15 receiving treatment progressed into leukemia	[125]
Cytarabine, hydroxyurea, Vitamin $1,25(OH)_2D_3$	AML	29	Oral	13 Patients (45%) obtained complete remission, 10 patients (34%) had a partial response, overall 79% response rate; median remission 9.8 months, overall	[116]

Kim M. et al.: Application of vitamin D and derivatives in hematological malignancies. *Cancer Letters,* 319; 8–22; 2012

First therapy using retinoic acid to differentiate the human leukemia cells

Proc. Natl. Acad. Sci. USA
Vol. 77, No. 5, pp. 2936–2940, May 1980
Medical Sciences

Induction of differentiation of the human promyelocytic leukemia cell line (HL-60) by retinoic acid*

(retinoids/myeloid differentiation/hematopoiesis)

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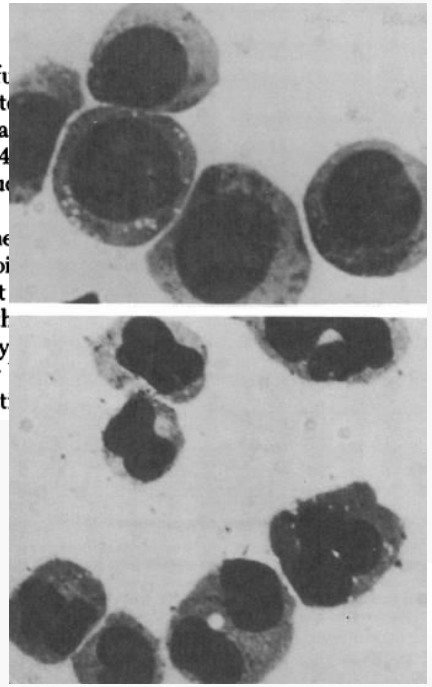
Laboratory of Tumor Cell Biology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20205

Communicated by Marshall Warren Nirenberg, January 31, 1980

ABSTRACT **The HL-60 cell line, derived from a patient with acute promyelocytic leukemia, proliferates continuously in suspension culture and consists predominantly (>90%) of promyelocytes. These cells can be induced to differentiate to morphologically and functionally mature granulocytes by incubation with a wide variety of compounds, including butyrate and hypoxanthine and polar planar compounds such as dimethyl sulfoxide and hexamethylene bisacetamide. We have now found that retinoic acid (all-*trans*-retinoic acid) induces differentiation (as measured morphologically and by the ability to reduce nitroblue tetrazolium) of HL-60 at concentrations as low as 1 nM. Maximal differentiation (approximately 90%) occurs at 1 μM, a concentration 1/500th to 1/160,000th the concentrations of butyrate (0.5 mM) and dimethyl sulfoxide (160 mM) that promote a similar increase in differentiation. Continuous exposure to**

induced HL-60 cells have many of the fu
of normal peripheral blood granulocyt
tosis, complement receptors, chemota
reduce nitroblue tetrazolium (NBT) (14
line provides a unique system for stu
differentiation *in vitro*.

In the present report we describe the
differentiation of HL-60 cells by retinoi
induces differentiation of HL-60 cells at
to 1/160,000th the concentrations of oth
suggests a new approach to the therapy
mias and indicates that retinoids may
ferentiation of certain hematopoietic t



Witamin D active form calcitriol induces differentiation of human leukemia cells

Proc. Natl. Acad. Sci. USA
Vol. 78, No. 8, pp. 4990–4994, August 1981
Cell Biology

Differentiation of mouse myeloid leukemia cells induced by 1α,25-dihydroxyvitamin D_3

(vitamin D_3/1α-hydroxyvitamin D_3/macrophage/lysozyme activity/phagocytic activity)

ETSUKO ABE*, CHISATO MIYAURA*, HIROSHI SAKAGAMI†, MINORU TAKEDA†, KUNIO KONNO†, TOHRU YAMAZAKI‡, SHUSAKU YOSHIKI‡, AND TATSUO SUDA*§

*Department of Biochemistry, School of Dentistry, †Department of Biochemistry, School of Medicine, and ‡Department of Oral Pathology, School of Dentistry, Showa University, 1-5-8 Hatanodai, Shinagawa-ku, Tokyo 142, Japan

Communicated by Susumu Hagiwara, April 21, 1981

ABSTRACT Mouse myeloid leukemia cells can be induced to differentiate into macrophages *in vitro* by 1α,25-dihydroxyvitamin D_3, the active form of vitamin D_3. The minimal concentration of 1α,25-dihydroxyvitamin D_3 to induce the cell differentiation was 0.12 nM. The degree of cell differentiation in various markers induced by 12 nM 1α,25-dihydroxyvitamin D_3 was nearly equivalent to that induced by 1 μM dexamethasone, the most potent known stimulator. Among several markers of the differentiation by 1α,25-dihydroxyvitamin D_3, phagocytic activity was induced within 24 hr, and this was followed by induction of lysozyme and locomotive activities. Similar changes were also induced by 0.01–1 μM 1α-hydroxyvitamin D_3. 25-Hydroxyvitamin D_3 and 24*R*,25-dihydroxyvitamin D_3 showed only weak inducing activity. These results suggest the possibility that, in addition to its well-known biological activities in enhancing intestinal calcium transport and bone mineral mobilization, 1α,25-dihydroxyvitamin D_3 is involved in the differentiation of bone marrow cells.

MATERIALS AND METHODS

Vol. 102, No. 3, 1981 BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS
October 15, 1981 Pages 937-943

1α,25-DIHYDROXYVITAMIN D_3 INDUCES DIFFERENTIATION OF HUMAN MYELOID LEUKEMIA CELLS

Chisato Miyaura, Etsuko Abe, Takeo Kuribayashi, Hirofumi Tanaka, Kunio Konno†, Yasuho Nishii¶ and Tatsuo Suda*

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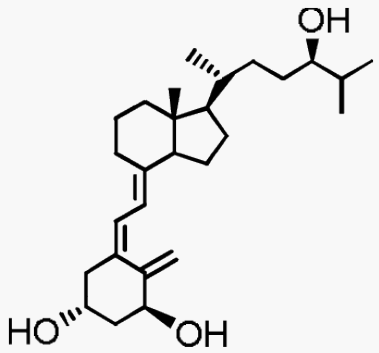
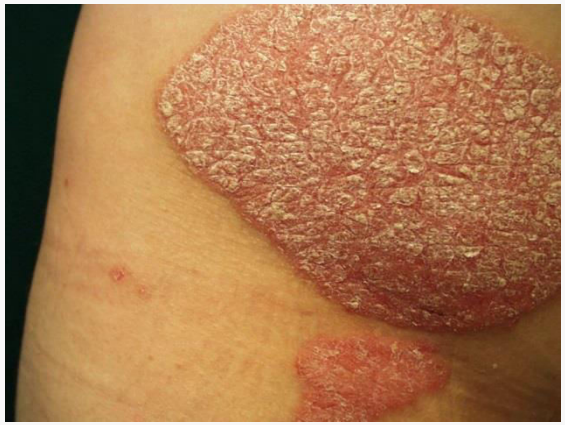
Received August 27, 1981

One of such active and less toxic vitamin D analog is 1,24-dihydroxyvitamin D_3, tacalcitol (1,24-$(OH)_2D_3$) PRI-2191

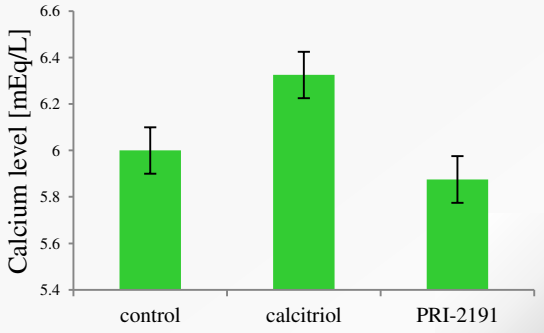


tacalcitol- PRI-2191



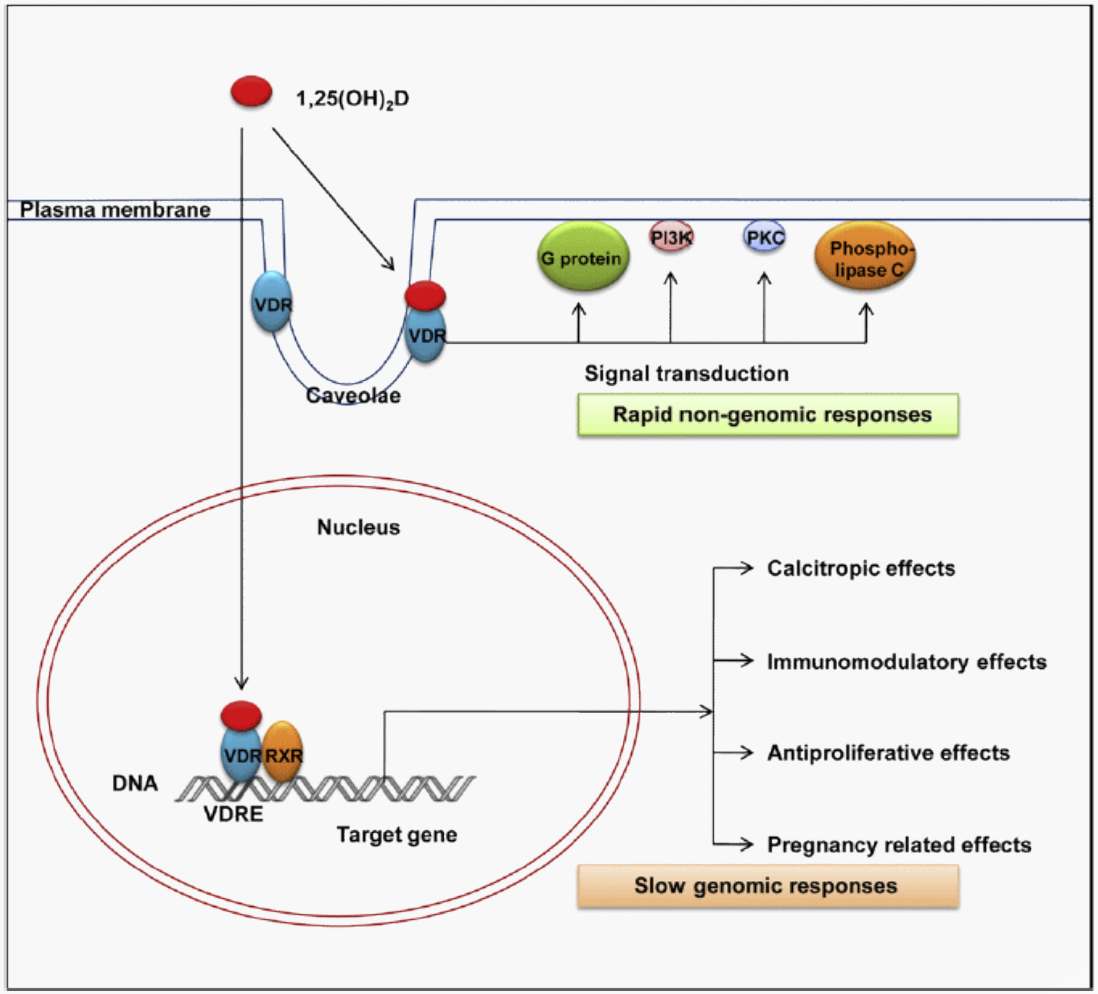
http://www.huidziekten.nl/zakboek/dermatosen/ptxt/Psoriasis.htm

Dose (μg/kg/day)	Calcitriol		$1,24\text{-}(OH)_2D_3$	
	N	Calcium level (mEq/L) ± S.D.	*N*	Calcium level (mEq/L) ± S.D.
Subcutaneous route				
1	3	6.1 ± 0.2	3	5.2 ± 0.3[a]
1	3	5.6 ± 0.06	2	4.9 ± 0.07
10	3	7.6 ± 0.1	3	6.8 ± 0.5[a]
10	3	6.0 ± 0.1	3	5.6 ± 0.3
80% propylene glycol	4		4.6 ± 0.1 mEq/L	
Oral route				
1	2	4.6 ± 0.0[c]	2	4.6 ± 0.1
1	3	4.6 ± 0.1[c]	3	4.4 ± 0.2
10	4	6.4 ± 0.5	4	5.3 ± 0.05[ac]
10	4	5.3 ± 0.05[bc]	4	5.0 ± 0.2[bc]
80% propylene glycol	6		4.1 ± 0.4 mEq/L	
Untreated	5		3.6 ± 0.1 mEq/L	



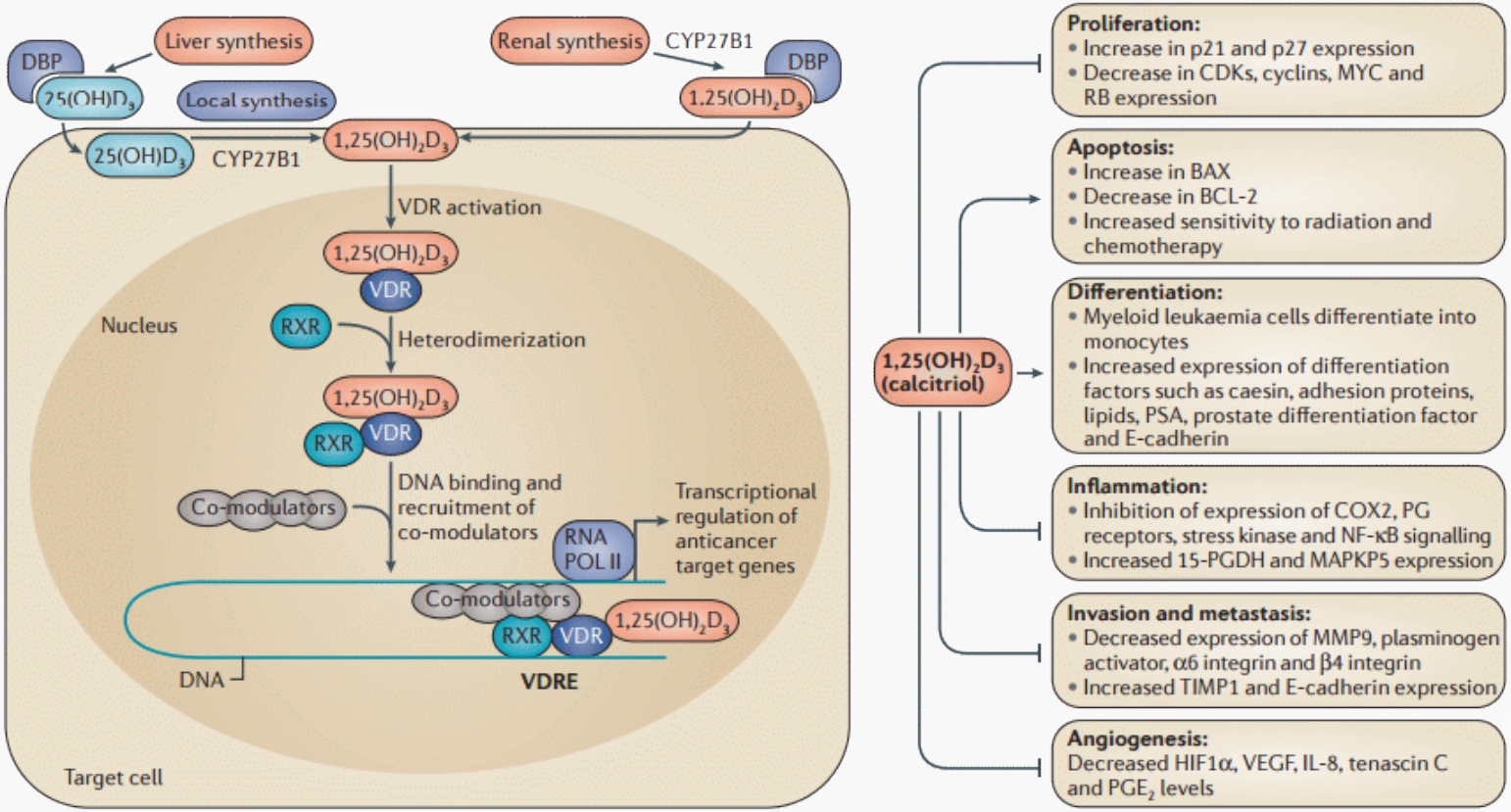
Wietrzyk J. et al.: Toxicity and antineoplastic effect of (24*R*)-1,24-dihydroxyvitamin D3 (PRI-2191), Steroids 69 , 629–635, 2004

Genomic and non-genomic responses of vitamin D receptor binding to $1,25(OH)_2D_3$



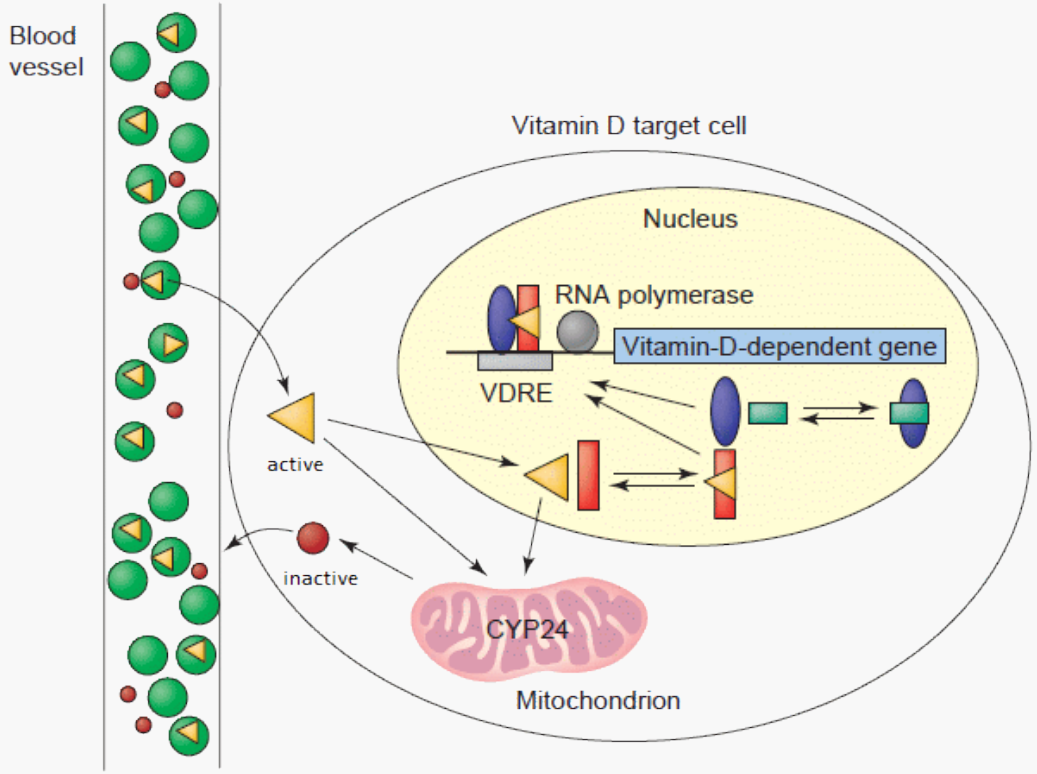
Shin J. et al.: Vitamin D effects on pregnancy and the placenta. *Placenta* 31; 1027-1034; 2010

Vitamin D target genes are involved in diverse molecular pathways



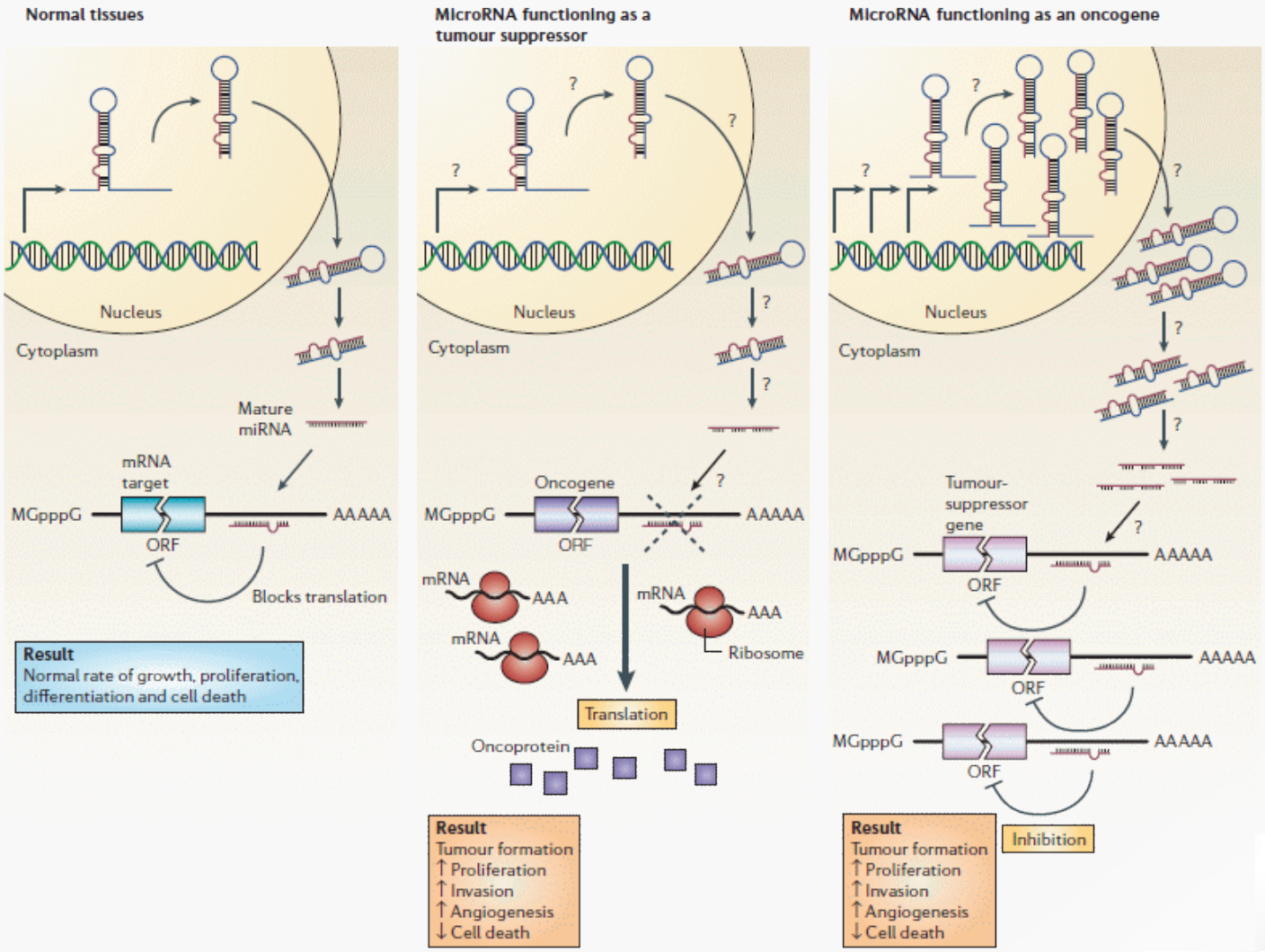
Feldman D. et al.: The role of vitamin D in reducing cancer risk and progression. *Nature Reviev*, 12 (314-320), 2014

The intracellular concentration of calcitriol is determined by cytochrome P450 enzyme CYP24A1



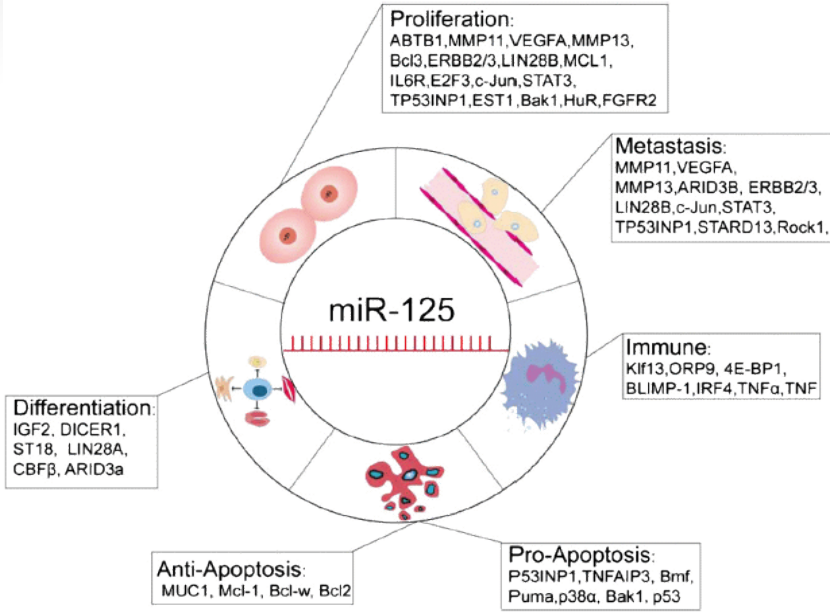
Prosser D. and Jones G.: Enzymes involved in the activation and inactivation of vitamin D. *Trends in Biochemical Sciences, 29*, 12:2004

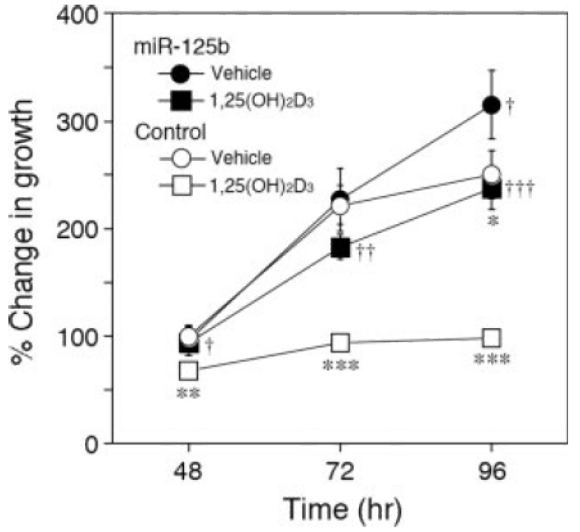
MicroRNAs can function as tumour suppressors and oncogenes



Esquela-Kerscher A. and Slack F.: Oncomirs — microRNAs with a role in cancer. *Nature Rewievs, Cancer* 6, 2006

The targets of miR-125 are involved in different types of disease pathogenesis





Antiproliferative effects of $1,25(OH)_2D_3$ in MCF-7 cells

miR-125 consists of three homologs miR-125a, miR-125b-1 hsa-miR-125-2 and **miR-125b** regulates vitamin D receptor and CYP24 expression

Sun Y. et al. Diverse functions of miR-125 family in different cell contexts. *Journal of Hematology & Oncology* 2013, 6:6
Mohri T. et al. :MicroRNA regulates human vitamin D receptor. *Int. J. Cancer*: 125, 1328–1333 (2009)

Differences between sensitivity of leukemia and lymphoma cells to calcitriol and its analog

Human myeloid leukemia and lymphoma cells

- **HL-60** – acute promyelocytic leukemia (M2)
- **KG-1** - acute promielocytic leukemia (M1)
- **K562** – chronic promielocytic leukemia (M6)
- **MV-4-11** - bifenotype mielomoncytic leukemia (M5)
- **Thp-1** – acute monocytic leukemia (M5)

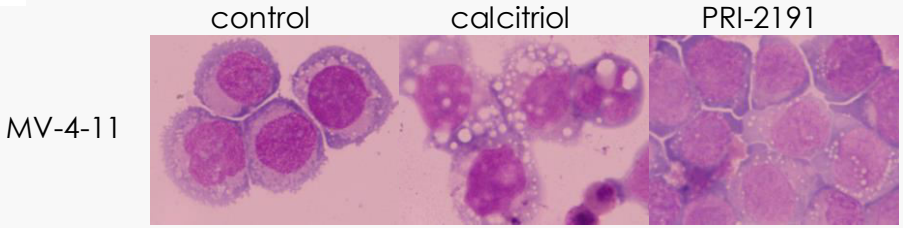
- **Daudi** – Burkitt's lymphoma
- **Raji** – Burkitts lymphoma
- **Jurkat** – acute T cells lymphoma
- **U2932** - diffuse large B-cell lymphoma

CYP24
VDR
miR-125b

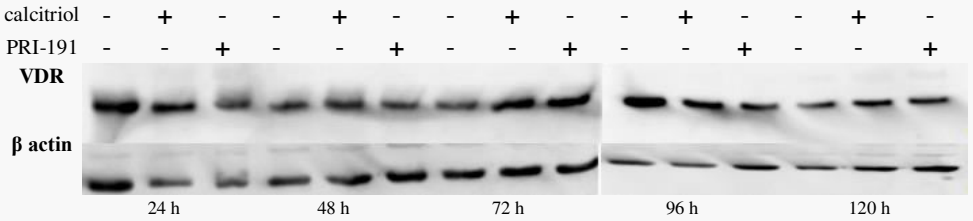
IC50 nM	HL-60	Thp-1	MV-4-11
calcitriol	42,40 ± 14,47	39,22 ± 19,50	21,49 ± 6,32
tacalcitol (PRI-2191)	8,7 ± 2,4	6,43 ± 2,3	2,77 ± 0,99

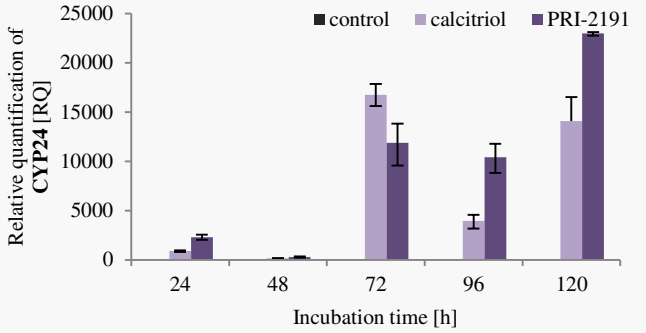
Proliferation inhibiton (%)	K562	KG-1	U_{2932}	Jurkat	Daudi	Raji
calcitriol 1000 nM	30,12 ± 13,83	1,25 ± 1,77	6,09 ± 8,61	11,24 ± 7,71	19,75 ± 14,40	23,55 ± 0,44
tacalcitol (PRI-2191) 1000 nM	32,16 ± 10,06	18,27 ± 6,24	0,48 ± 0,68	17,72 ± 2,14	11,70 ± 13,99	27,64 ± 13,11

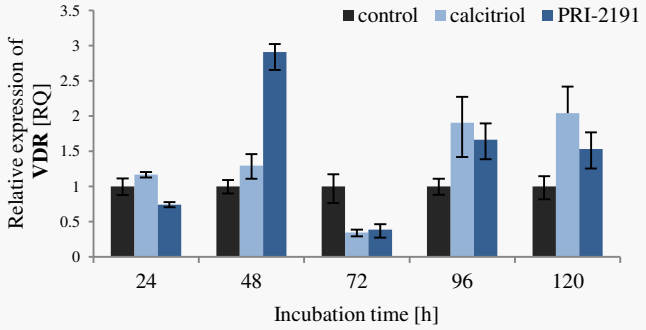
The expression level of CYP24, VDR and miR-125b in MV-4-11 bifenotype mielomoncytic leukemia sensitive to calcitriol and its analog

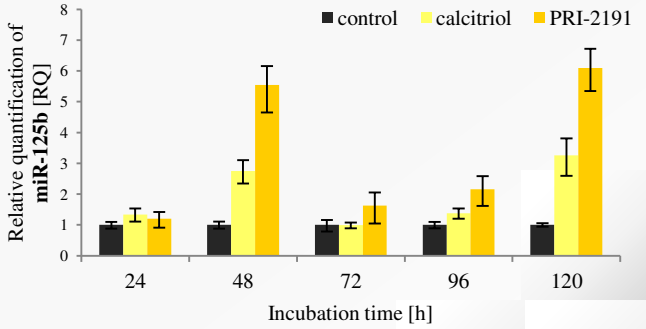


Cell morphology after 120 h with 10 nM calcitriol and analog treatment. May- Grunwald Giemsa staining, 100 x.



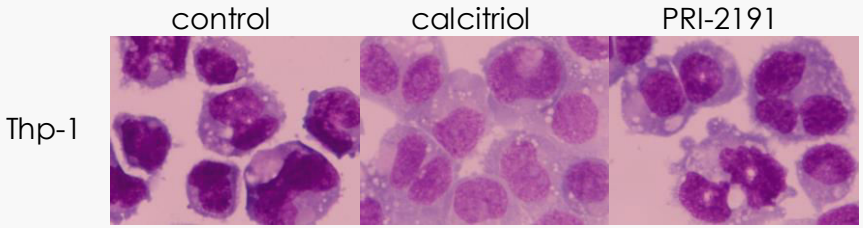




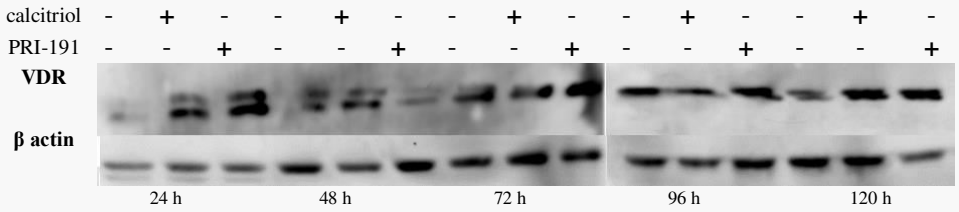


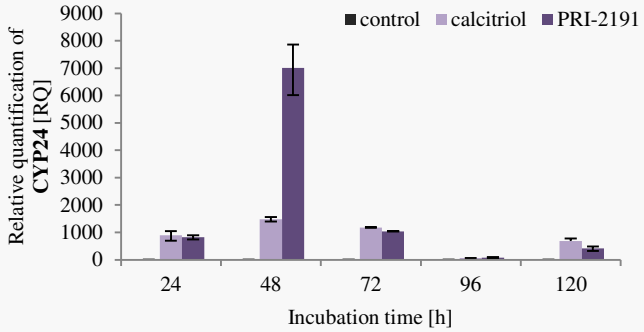
Real Time PCR analysis of **VDR** and **CYP24** mRNA and **miR-125b** expression using TaqMan Gene Expresion Assay TaqMan MicroRNA Expresion Assay, $\Delta\Delta C_T$ method

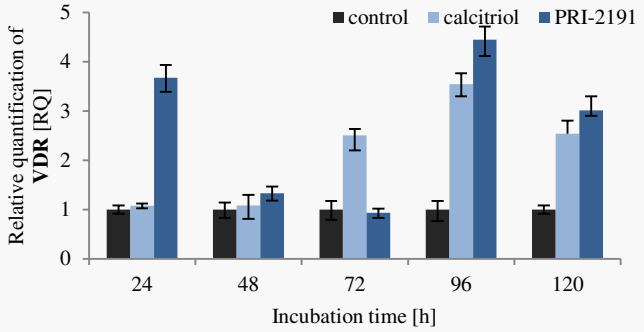
The expression level of CYP24, VDR and miR-125b in Thp-1 acute monocytic leukemia sensitive to calcitriol and its analog

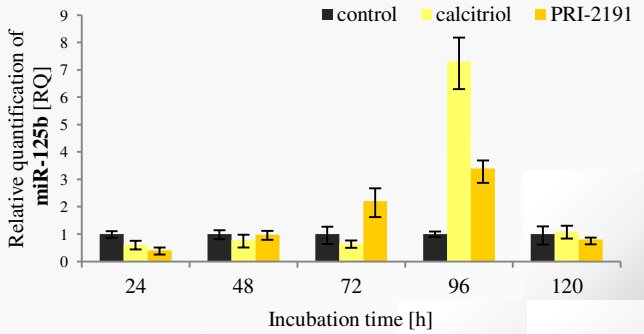


Cell morphology after 120 h with 10 nM calcitriol and analog treatment.
May- Grunwald Giemsa staining, 100 x.





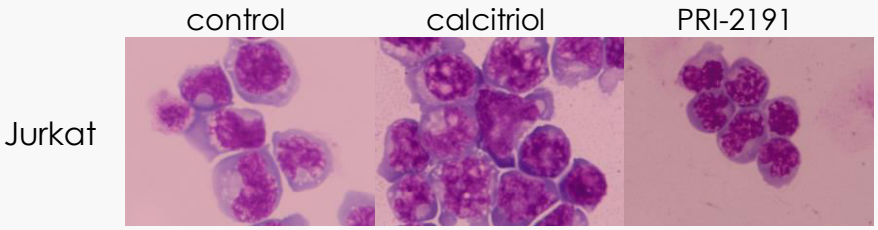




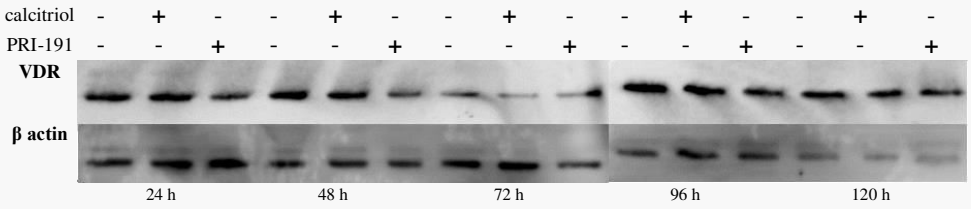
Real Time PCR analysis of **VDR** and **CYP24** mRNA and **miR-125b** expression using TaqMan Gene Expresion Assay TaqMan MicroRNA Expresion Assay, $\Delta\Delta C_T$ method

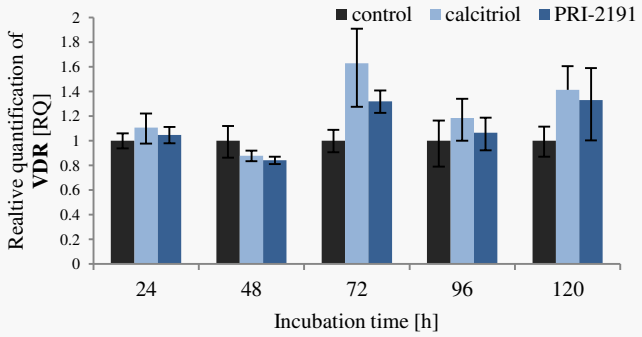
The expression level of CYP24, VDR and miR-125b in Jurkat acute T cells lymphoma cells unsensitive to calcitriol and its analog

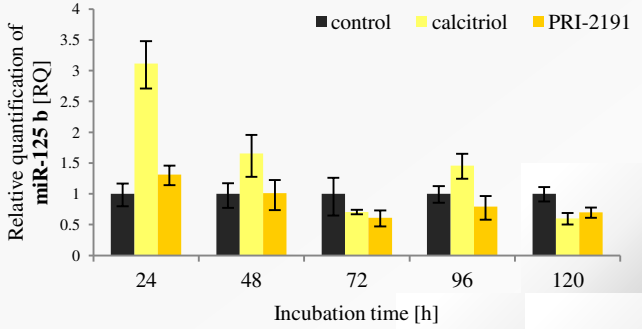
CYP24 expression was undetectable before and after calcitriol and its analog treatmetnt



Cell morphology after 120 h with 10 nM calcitriol and analog treatment.
May- Grunwald Giemsa staining, 100 x.



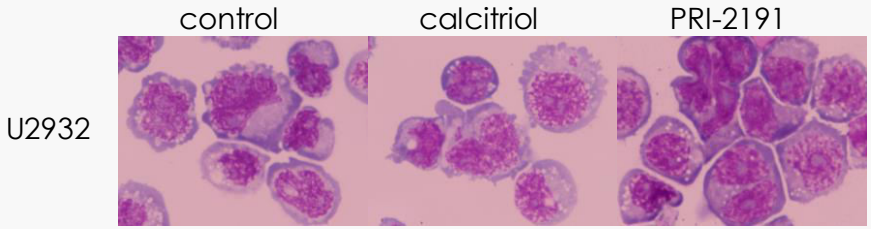




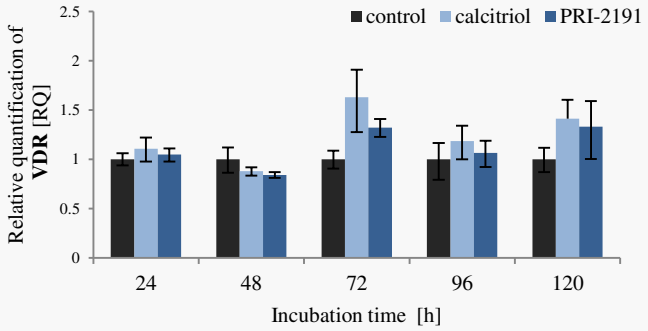
Real Time PCR analysis of **VDR** and **CYP24** mRNA and **miR-125b** expression using TaqMan Gene Expresion Assay TaqMan MicroRNA Expresion Assay, $\Delta\Delta C_T$ method

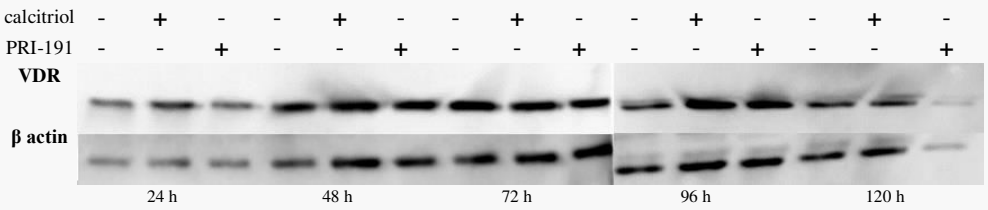
The expression level of CYP24, VDR and miR-125b in U2932 diffuse large B-cell lymphoma unsensitive to calcitriol and its analog

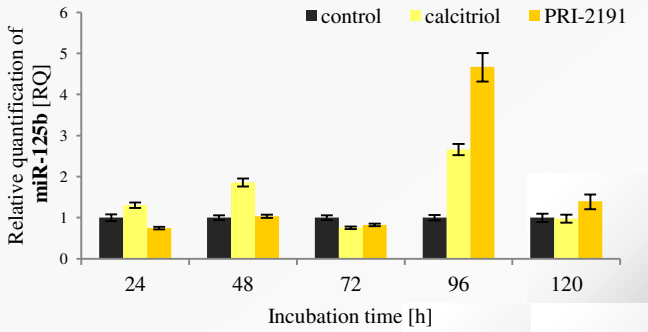
CYP24 expression was undetectable before and after calcitriol and its analog treatmetnt



Cell morphology after 120 h with 10 nM calcitriol and analog treatment.
May- Grunwald Giemsa staining, 100 x.







Real Time PCR analysis of **VDR** and **CYP24** mRNA and **miR-125b** expression using TaqMan Gene Expresion Assay TaqMan MicroRNA Expresion Assay, $\Delta\Delta C_T$ method

Conclusions

- ✓ The most sensitive to calcitriol and its analog human leukemia and lymphoma cells express CYP24, VDR and miR-125b
- ✓ In Daudi, Jurkat and U2932 human lyhmpoma cell lines calcitriol and its analog didn't induce-CYP24 expression despite of VDR expression
- ✓ The highest changes in the expression of CYP24 was observed in the most sensitive cell line MV-4-11 (fold change about 25 000)
- ✓ In human leukemia cells the VDR expression was similar both on mRNA and protein level independently of treatment

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