VITAMIN D IMMUNOLOGY: COVID 19 DISEASE

Defence & Adjunct Therapy

Dr. Renu Mahtani MD FMNM

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Vitamin D deficiency is now recognized as a **PANDEMIC**

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The American Journal of
CLINICAL NUTRITION


Rickets is just the tip of the vitamin D deficiency iceberg
RICKETS
ALLERGIES
ASTHMA
AUTISM
CANCER
CARDIOVASCULAR DISEASE
CHRONIC PAIN
COLDS & FLU
DENTAL CAVITIES
DIABETES - TYPE 1
ECZEMA
FETAL GROWTH IMPAIRMENT
GESTATIONAL DIABETES
GROWTH & DEVELOPMENT PROBLEMS
PREECLAMPSIA
PRENATAL INFECTIONS
PRETERM BIRTH
The lifelong impact of deficiency on pregnancy and the developing child
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The Harvard Gazette

HEALTH & MEDICINE

Study confirms vitamin D protects against colds and flu

A recent study by a global team of researchers has found that Vitamin D supplements, already widely prescribed for a variety of ailments, are effective in preventing respiratory diseases.

Credit: iStock

nutrients

MDPI

Review

Evidence that Vitamin D Supplementation Could Reduce Risk of Influenza and COVID-19 Infections and Deaths

William B. Grant [1,*], Henry Lahore [2], Sharon L. McDonnell [3], Carole A. Baggerly [3], Christine B. French [3], Jennifer L. Aliano [3] and Harjit P. Bhattoa [4]

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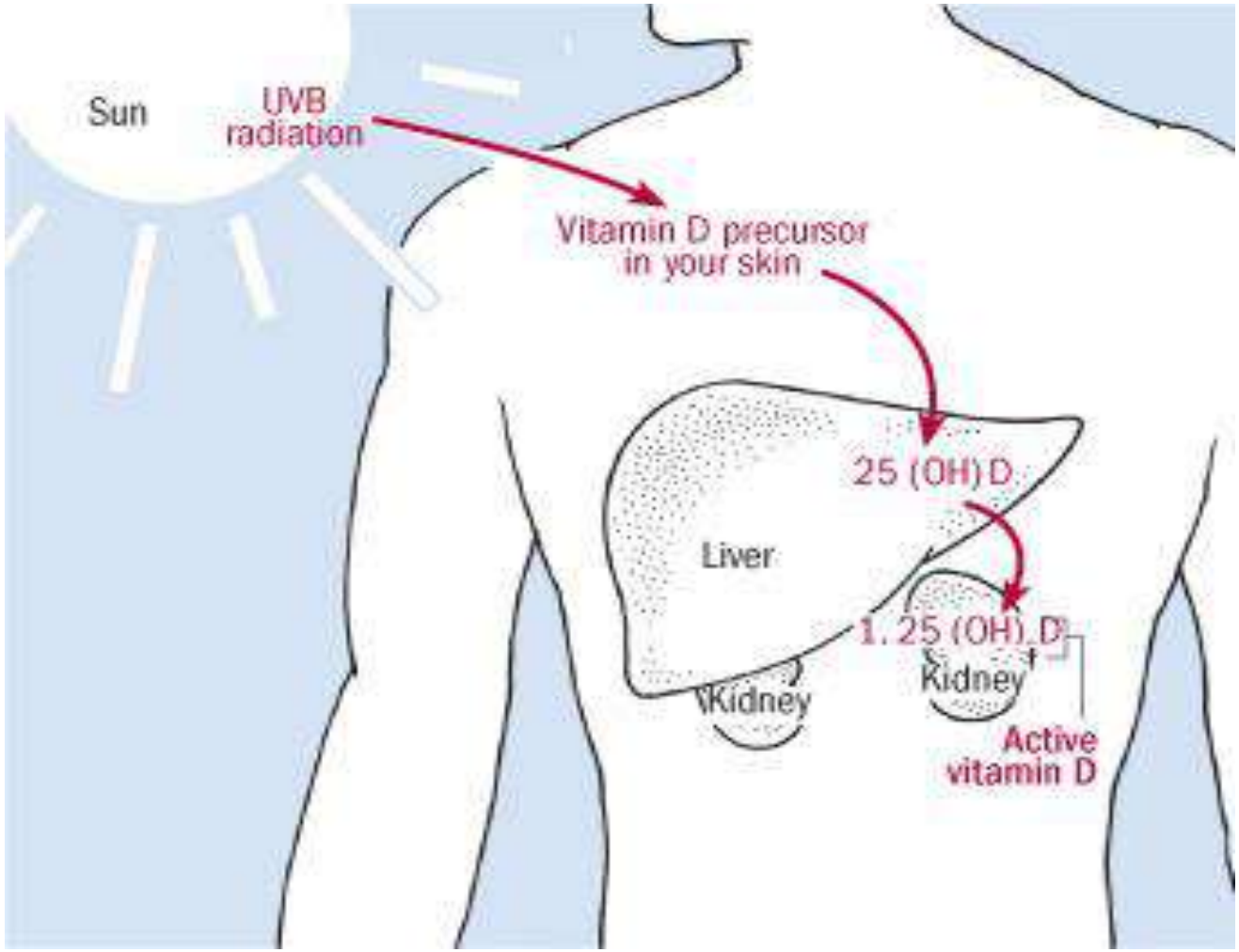
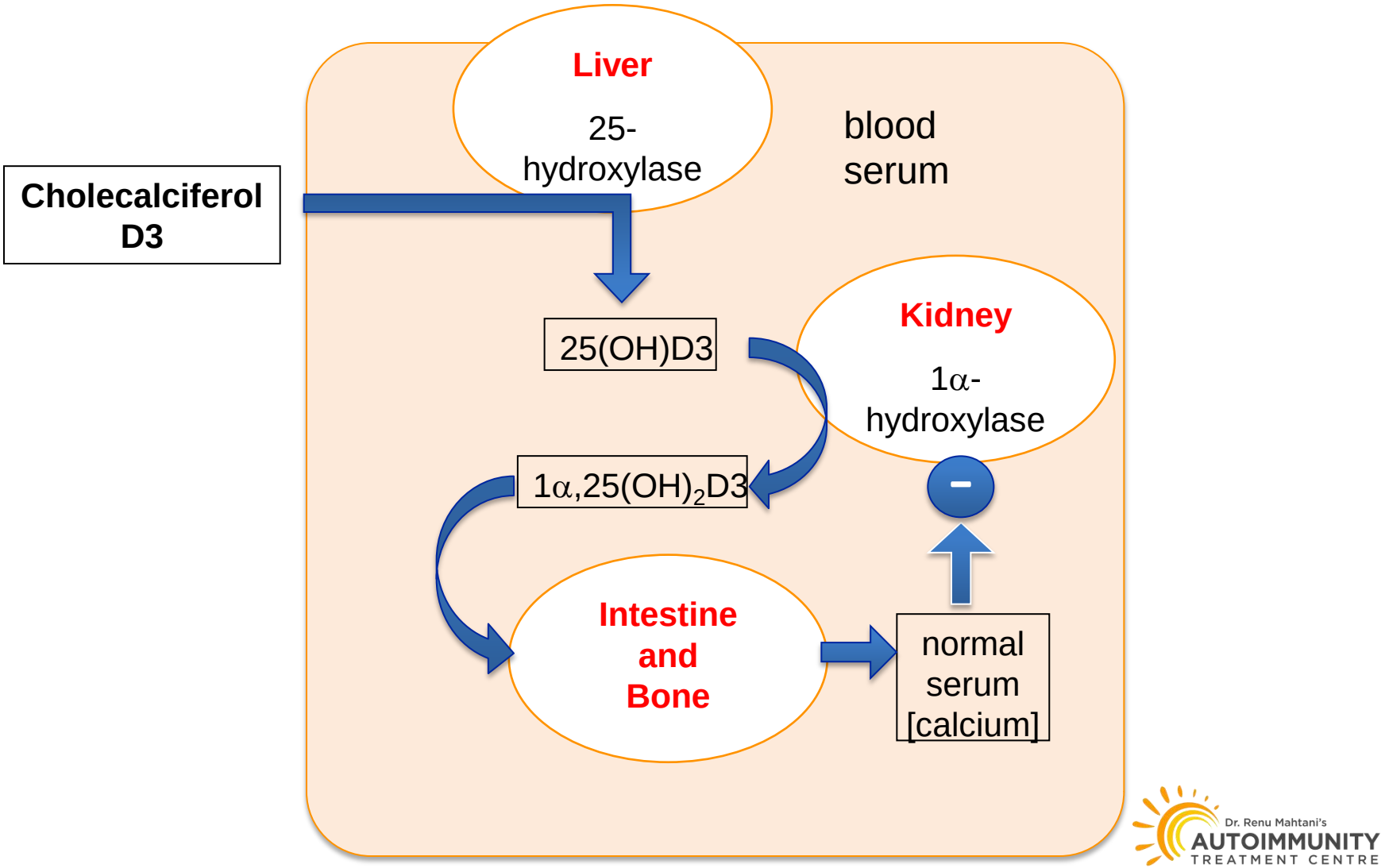
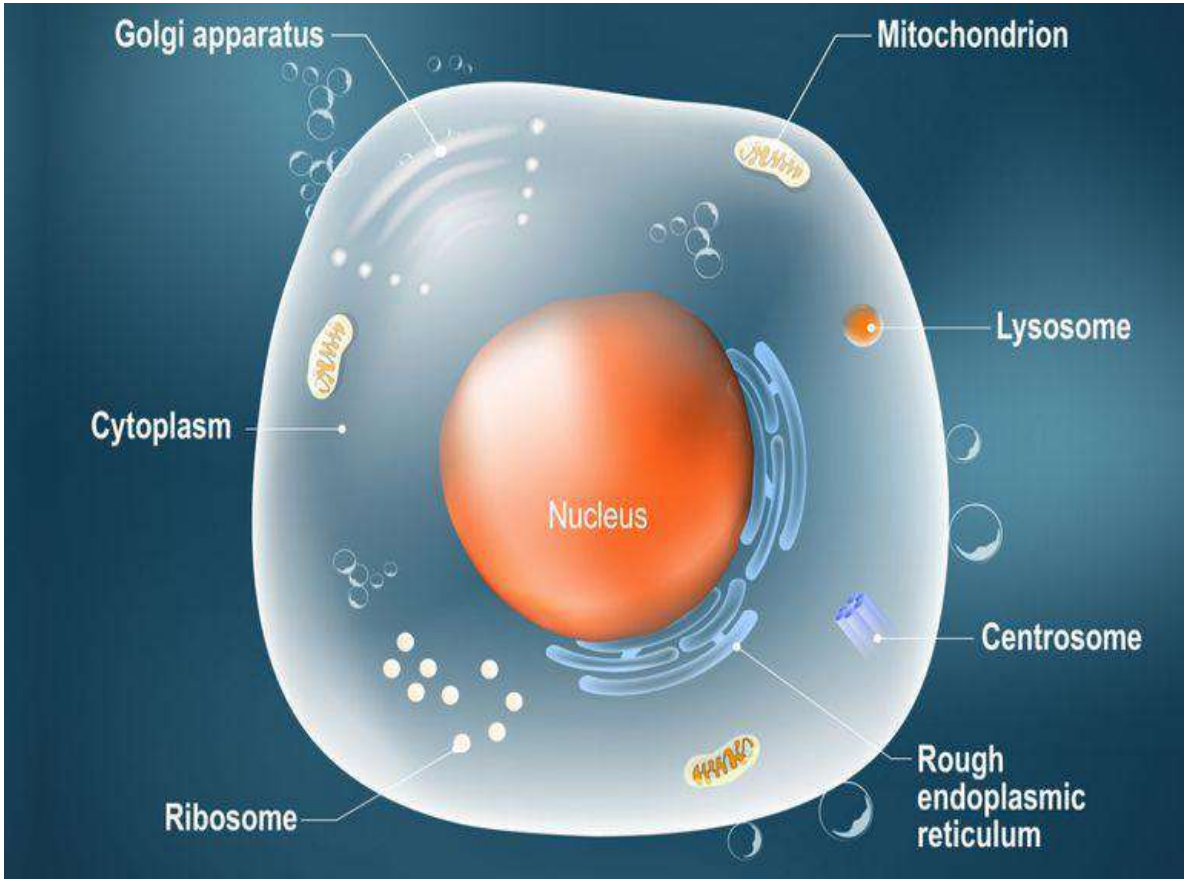
Vitamin D
Biological Activation



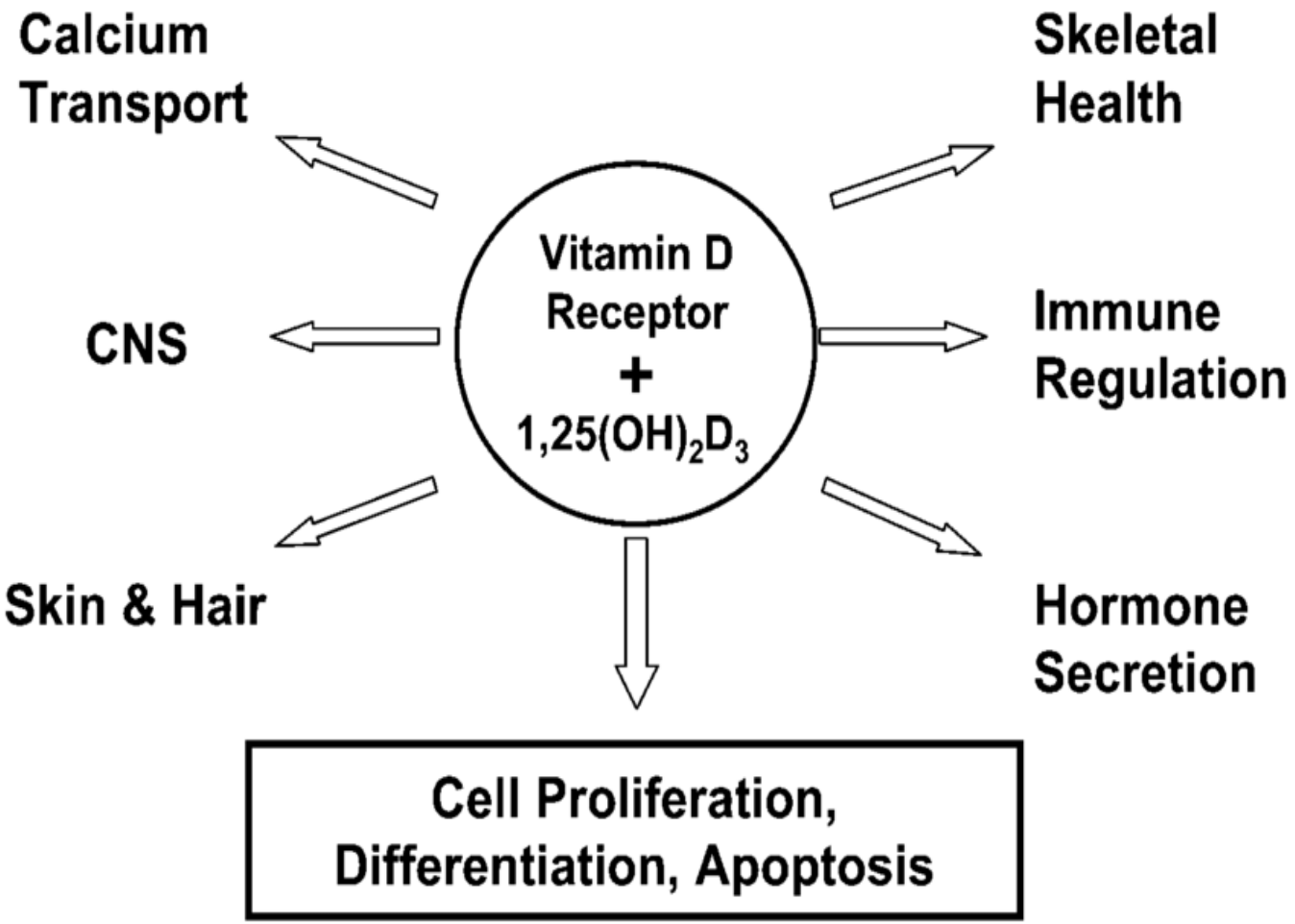
How Vitamin D works – 1 Hormone / Endocrine Action



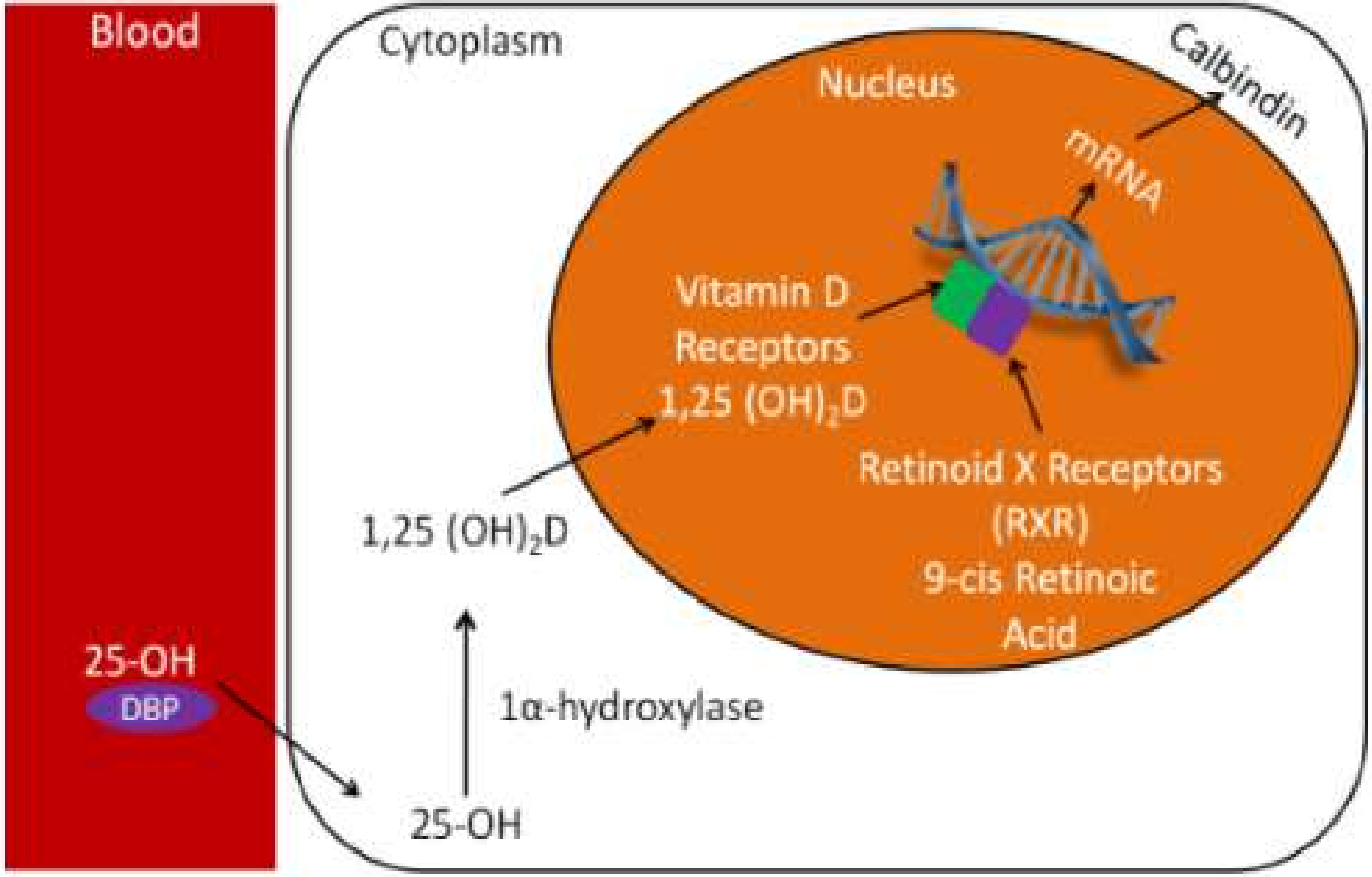
How Vitamin D works – 2
VDR – Vitamin D Receptor
Nuclear Receptor in every cell



VDR - Vitamin D Receptor



How Vitamin D works – 3
Local (intracellular) activation of Vitamin D
Autocrine / Paracrine action



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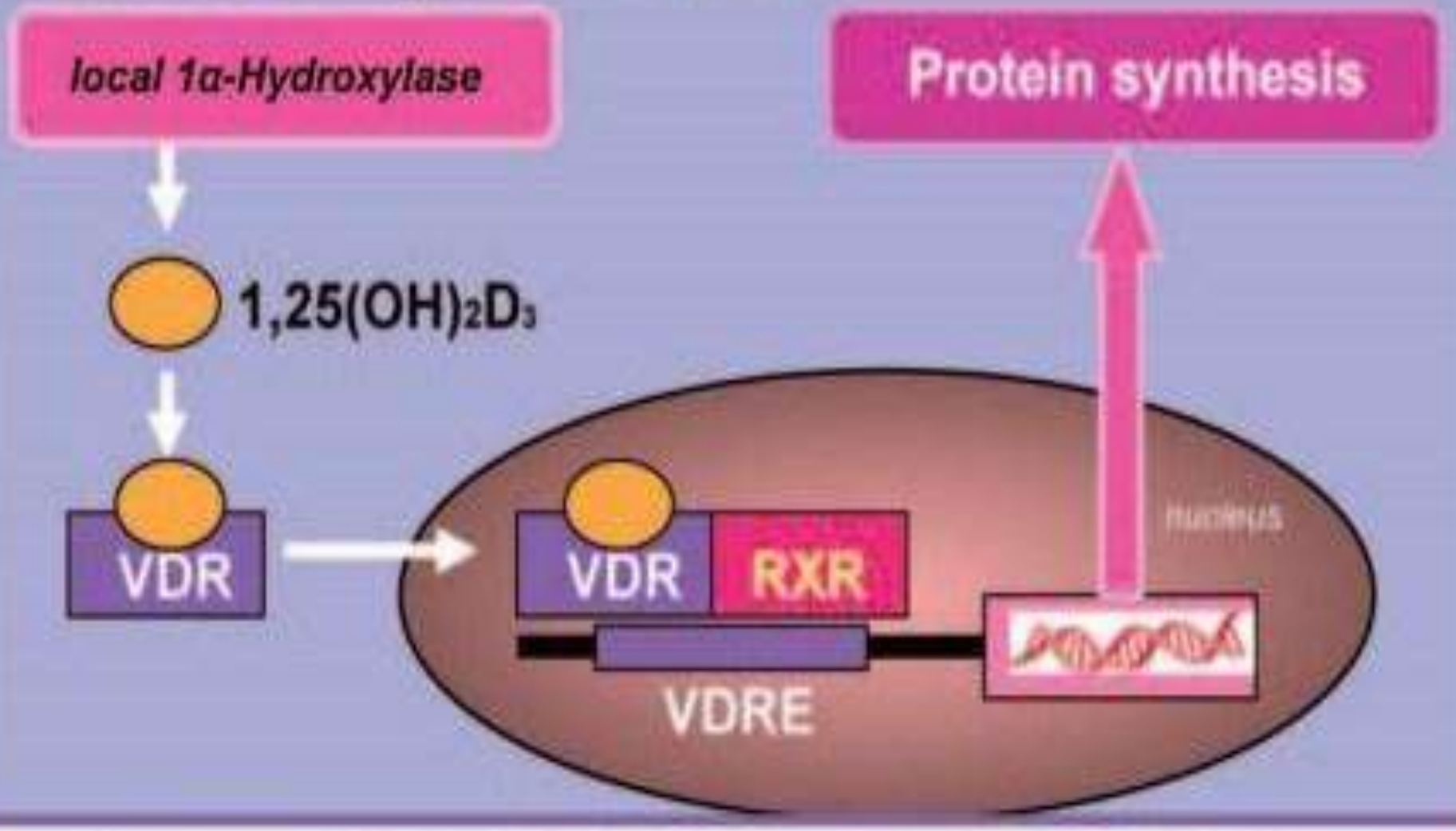
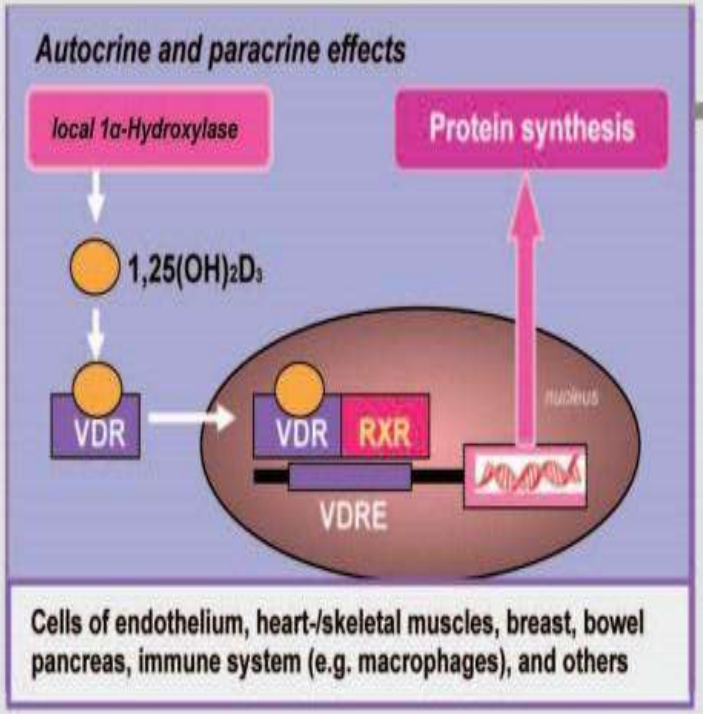
Autocrine and paracrine effects



Cells of endothelium, heart-/skeletal muscles, breast, bowel pancreas, immune system (e.g. macrophages), and others

Autocrine & Paracrine Effects



- Immune system: modulation
- Endothelial function ↑
- Renin angiotensin system ↓
- Secretion of insulin ↑
- Cell differentiation ↑
- Epigenetic signaling
- Induction of apoptosis ↑
- Tumor induced angiogenesis ↓

Extra-skeletal Actions of Vitamin D Beyond the Bones

- **Local intra-cellular activation** of vitamin D by 1 alpha hydroxylase, the enzyme that converts 25(OH)D to 1,25(OH)2D
- **Vitamin D receptor (VDR)** is expressed in all cells
  - Immune cells - Macrophages, monocytes, T cells, B cells, dendritic cells
  - Brain - microglia
  - Lungs, Intestines, Kidneys, Heart, Prostate
- **Gene modulator** - more than 2000 genes

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The Gene Modulator Vitamin D 2000 genes through the nuclear receptors (VDR)

Research

A ChIP-seq defined genome-wide map of vitamin D receptor binding: Associations with disease and evolution

Sreeram V. Ramagopalan,[1,2,3,6,7] Andreas Heger,[4,6] Antonio J. Berlanga,[1,2] Narelle J. Maugeri,[1] Matthew R. Lincoln,[1,2] Amy Burrell,[1,2] Lahiru Handunnetthi,[1,2] Adam E. Handel,[1,2] Giulio Disanto,[1,2] Sarah-Michelle Orton,[1,2] Corey T. Watson,[5] Julia M. Morahan,[1,2] Gavin Giovannoni,[3] Chris P. Ponting,[4] George C. Ebers,[1,2,7] and Julian C. Knight[1,7]

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VITAMIN D IMMUNOLOGY

- **COVID – 19 Disease**
  - For Prevention
  - As Adjunct Therapy
- **Mechanisms**
  - Genomic
  - Non-genomic

nutrients

Review

Vitamin D: Nutrient, Hormone, and Immunomodulator

Francesca Sassi, Cristina Tamone and Patrizia D'Amelio *

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* Correspondence: patrizia.damelio@unito.it; Tel.: +39-011-6335533

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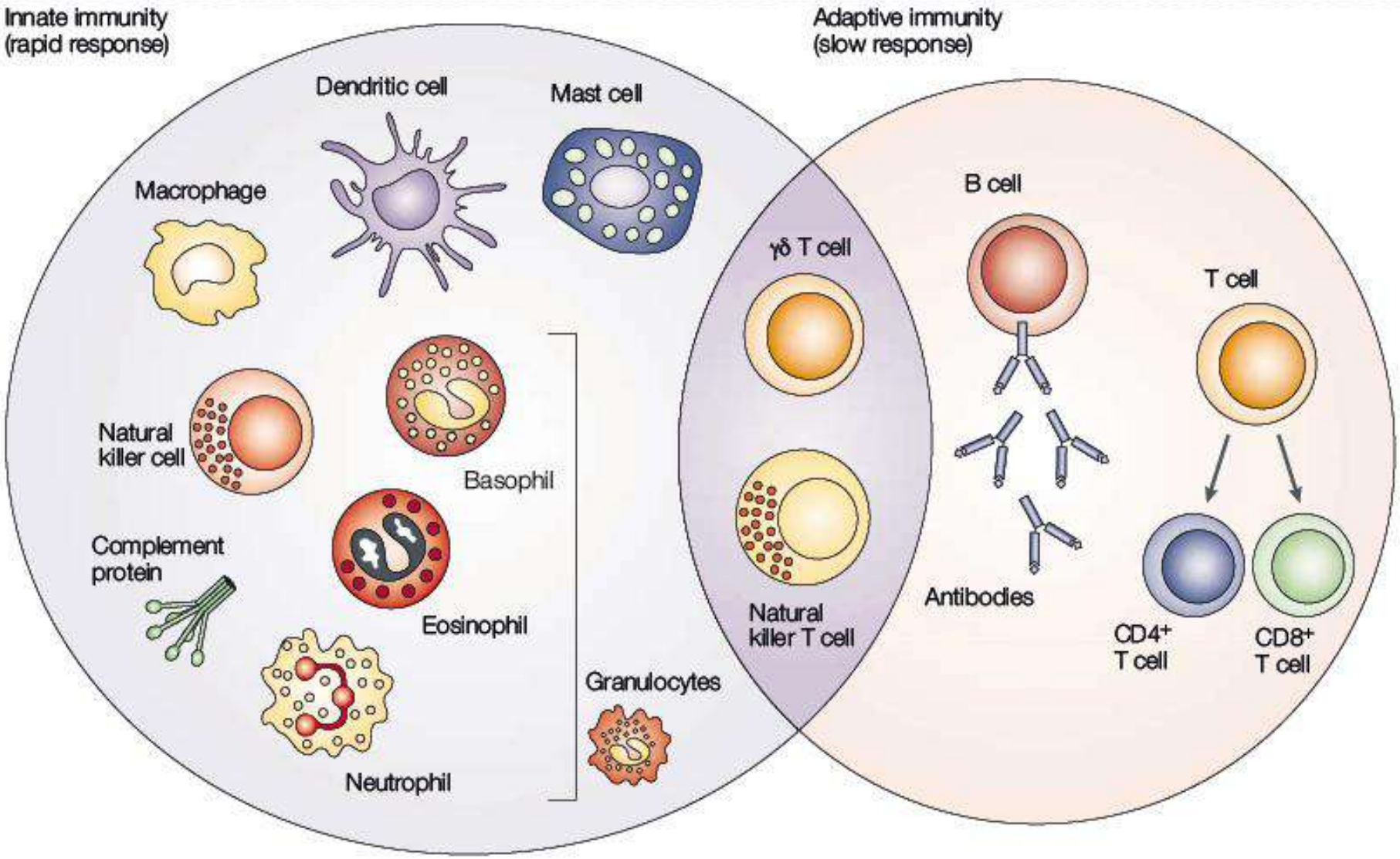
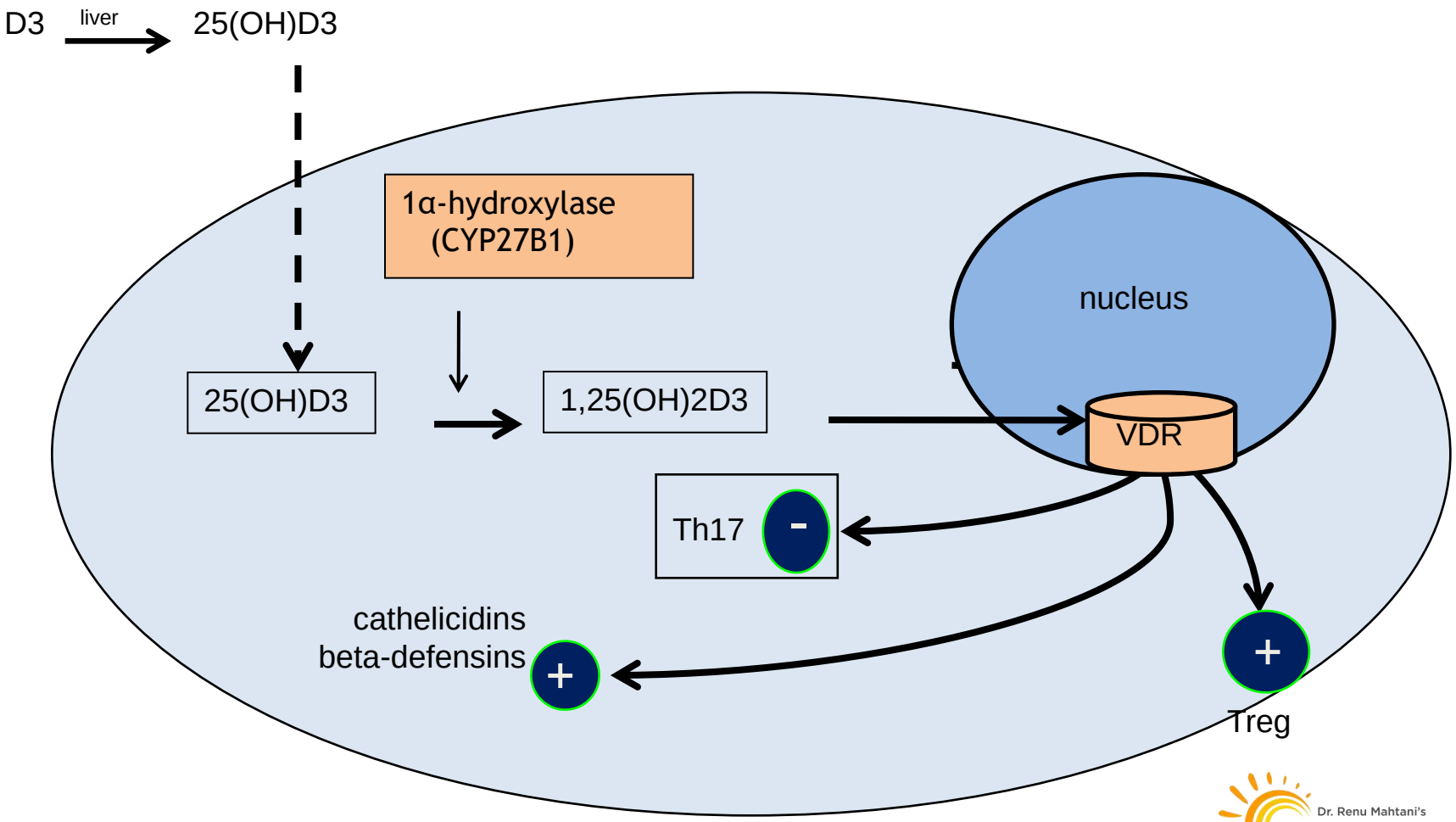
Abstract: The classical functions of vitamin D are to regulate calcium-ph
control bone metabolism. However, vitamin D deficiency has been re
conditions associated with increased inflammation and deregulation of th
diabetes, asthma, and rheumatoid arthritis. These observations, together
suggest a critical role for vitamin D in the modulation of immune fu
hypothesis of a disease-specific alteration of vitamin D metabolism
vitamin D in maintaining a healthy immune system. Two key observatio

VDR are present in all immune cells



Vitamin D gets activated inside the Immune Cells



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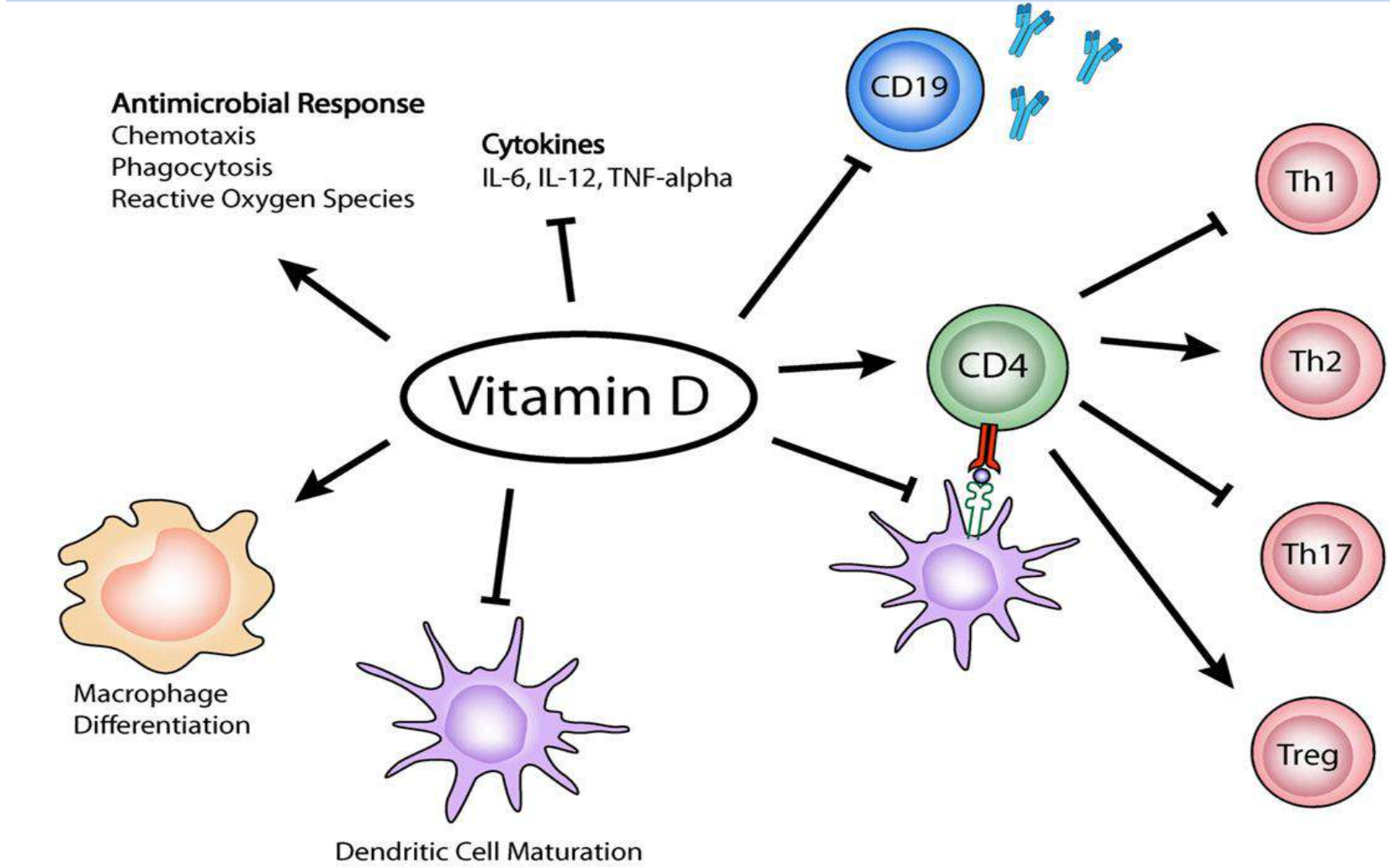
Vitamin D – Innate & Adaptive Response



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Review

Modulation of the Immune Response to Respiratory Viruses by Vitamin D

Claire L. Greiller * and Adrian R. Martineau *

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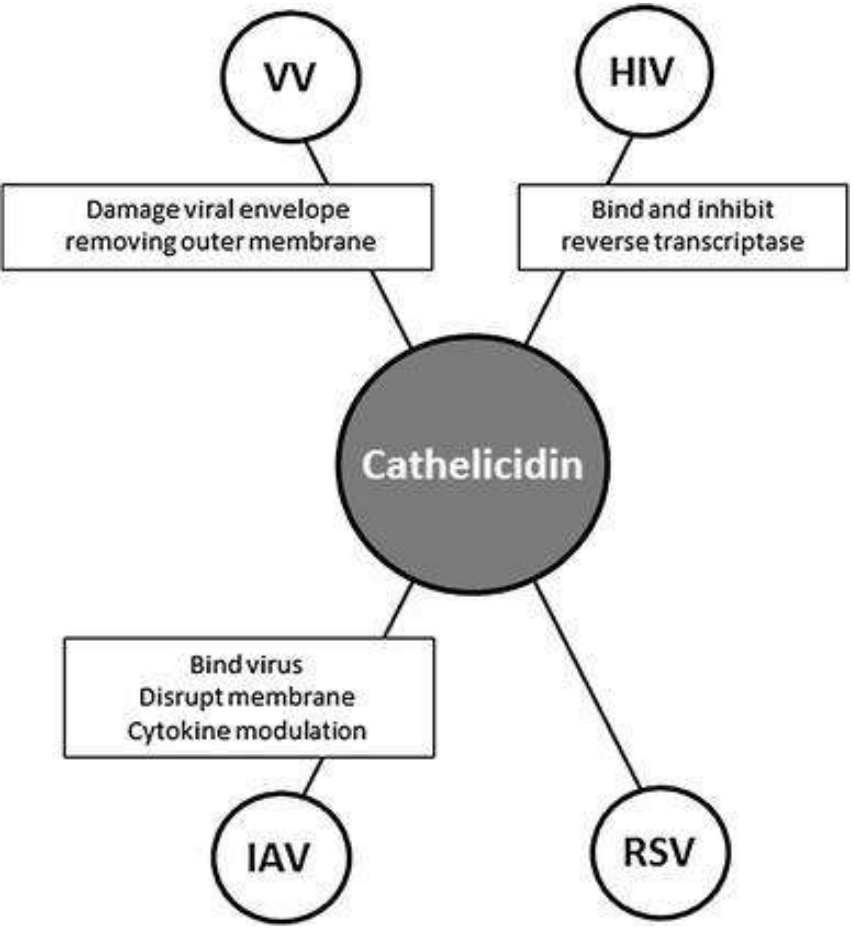
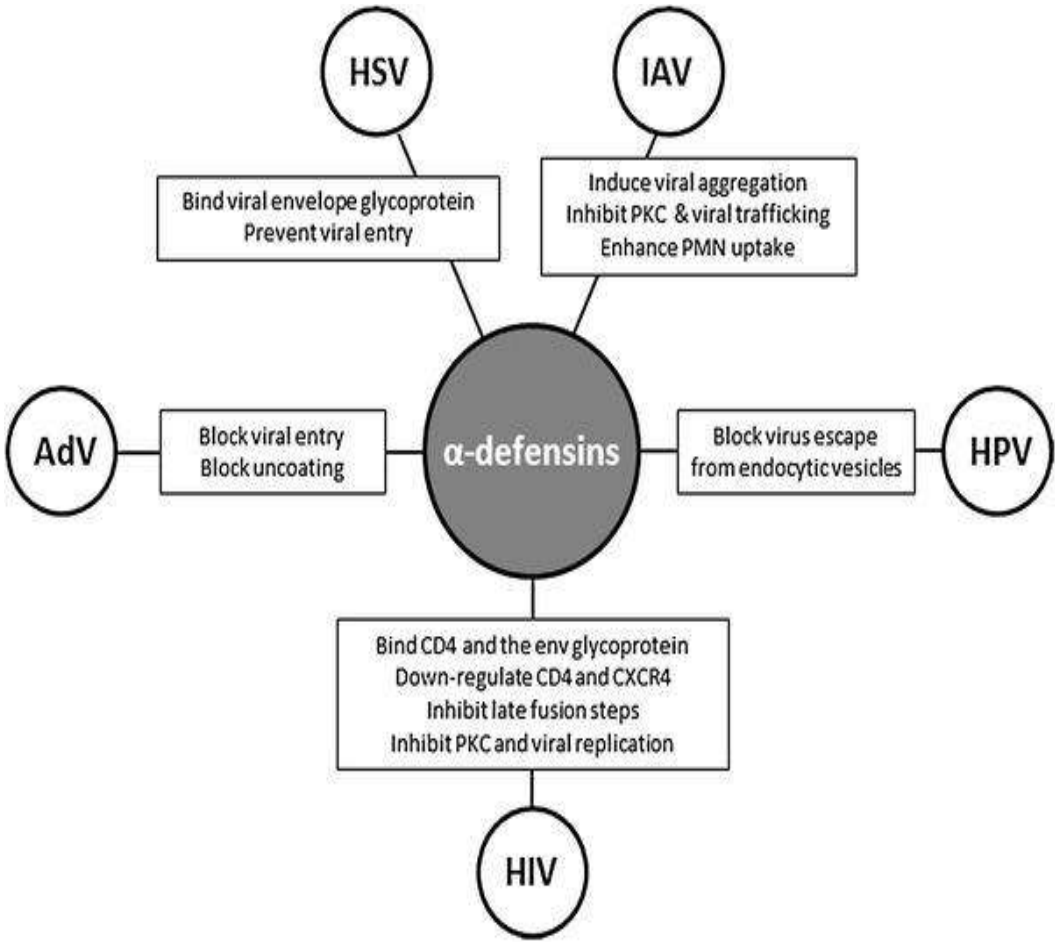
Innate immune response or **First line of Defense** - Defensins & Cathelicidins - anti-microbial and immuno-modulatory capabilities.

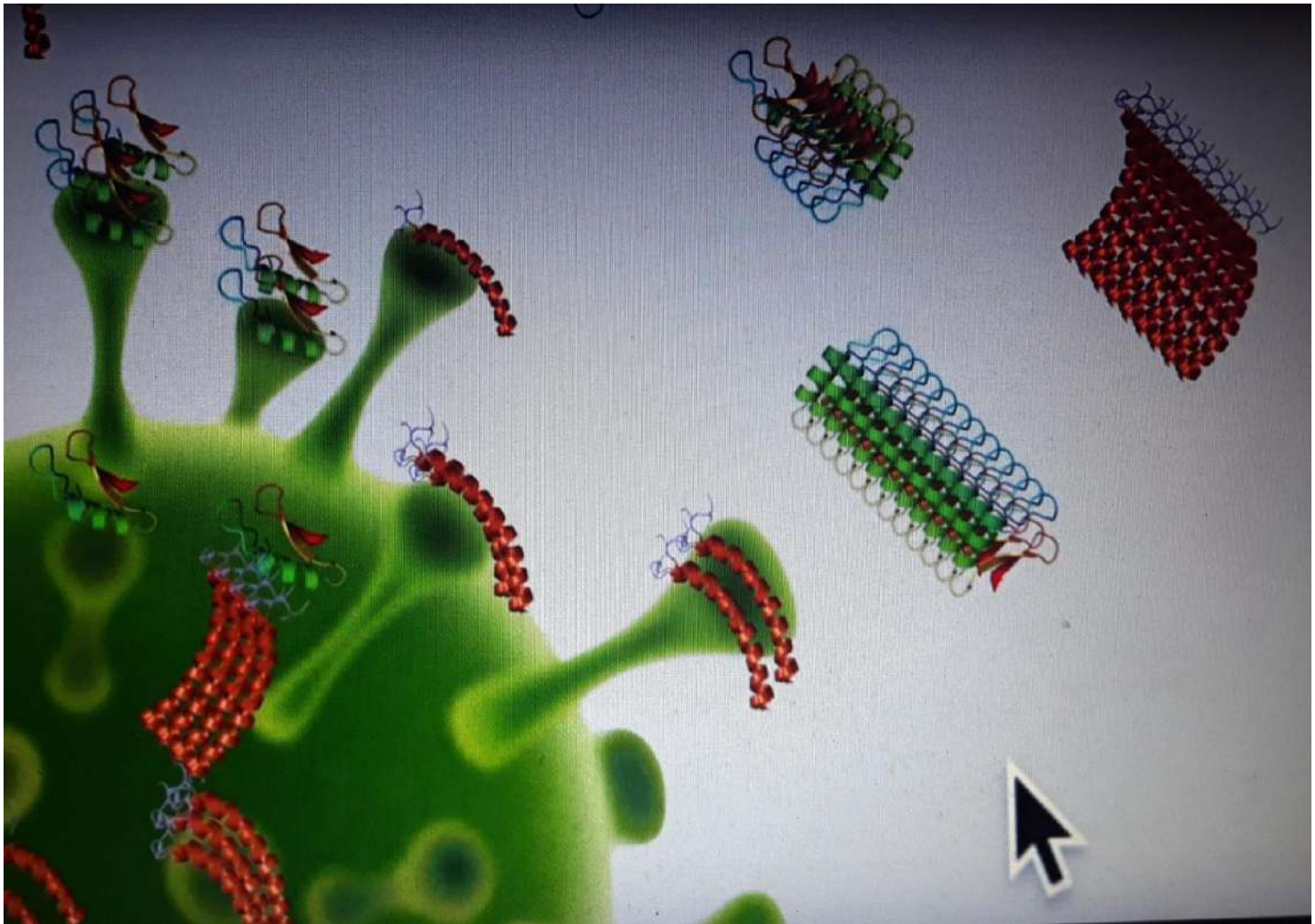
Natural, broad spectrum anti-viral agents against both enveloped and non-enveloped viruses.

Natural antibiotics & antivirals
Cathelicidins & Defensins

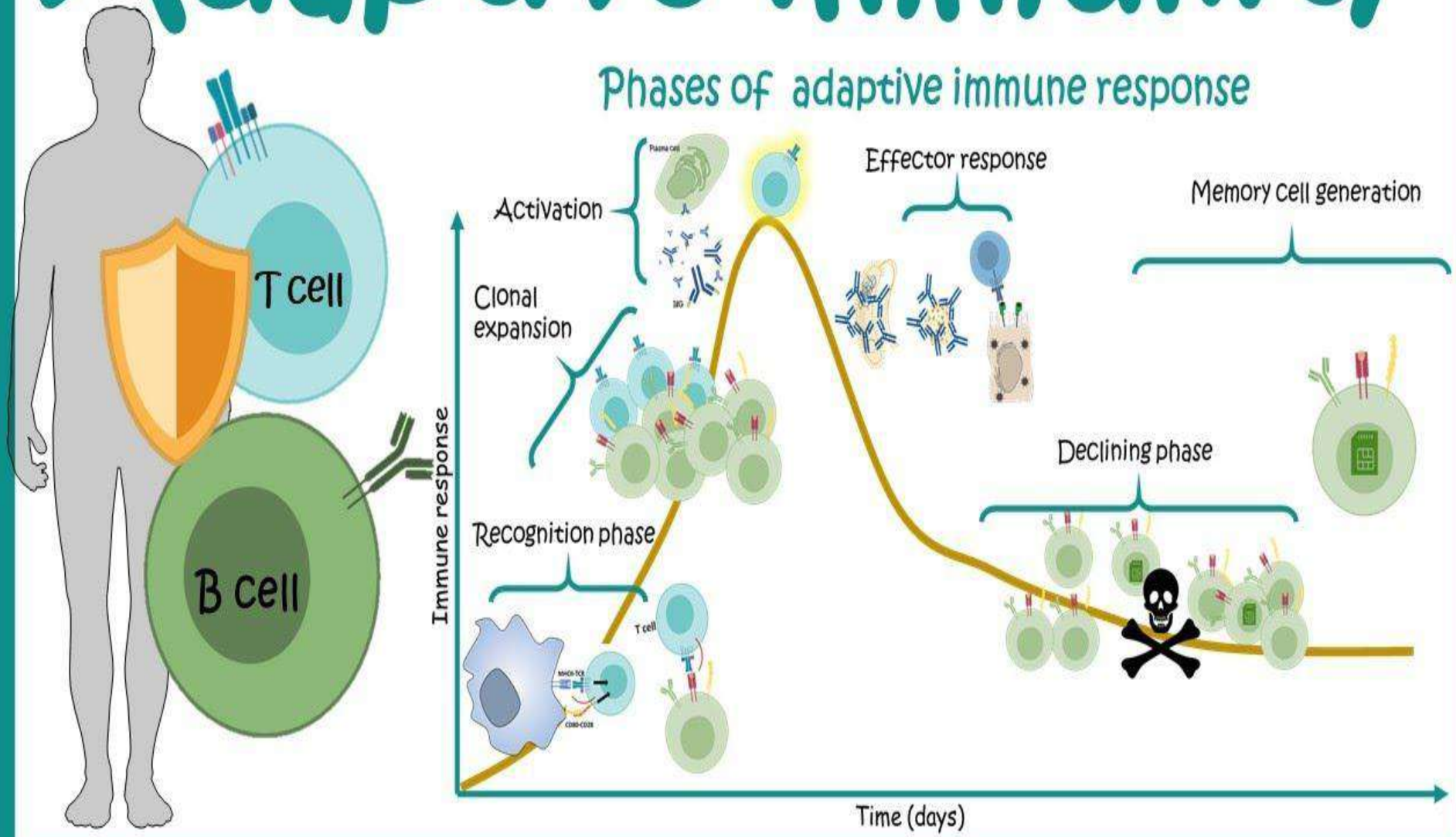


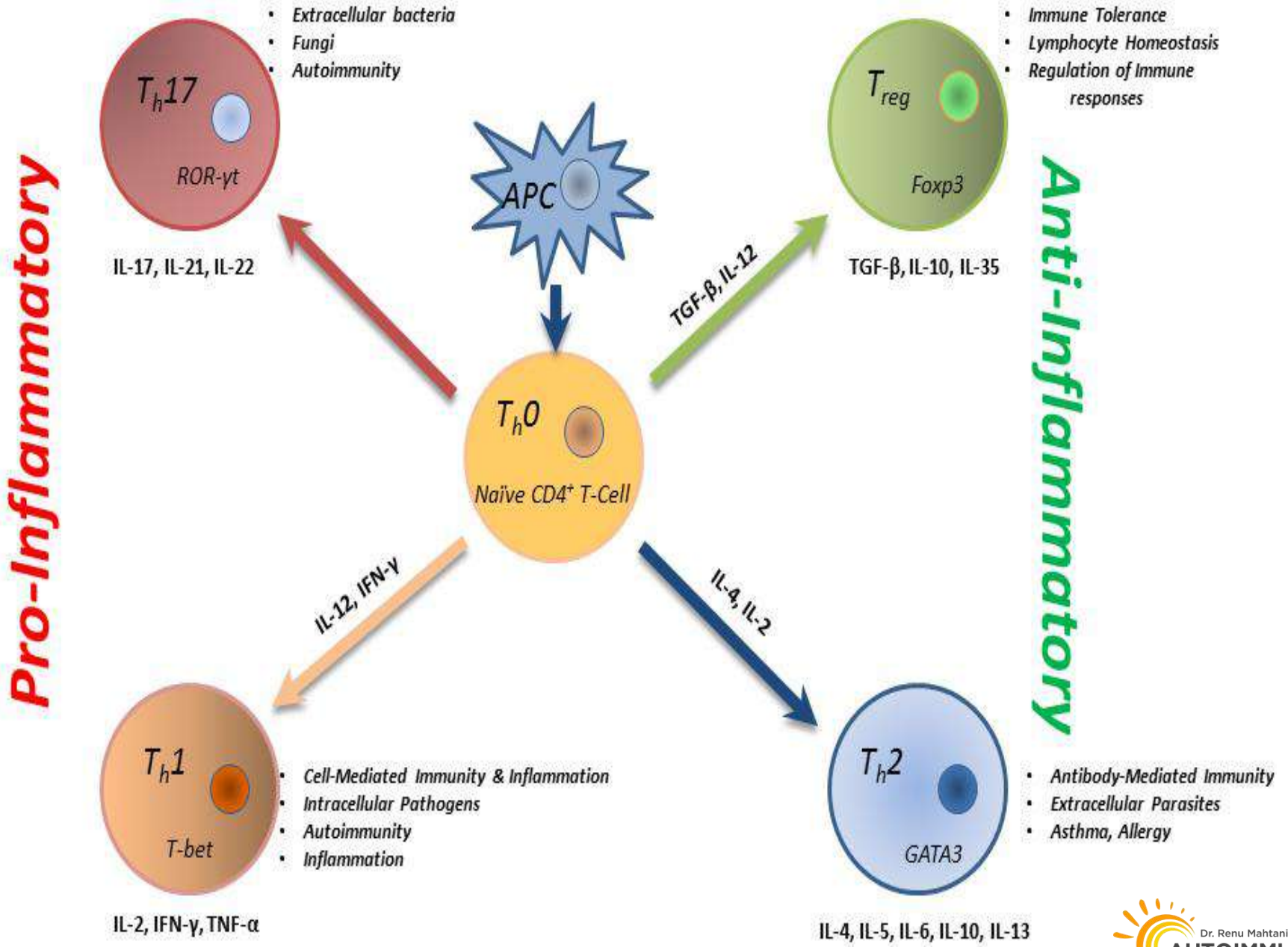


Vitamin D Innate Immune Response
Defensins & Cathelecidins
(Anti Spike Protein)



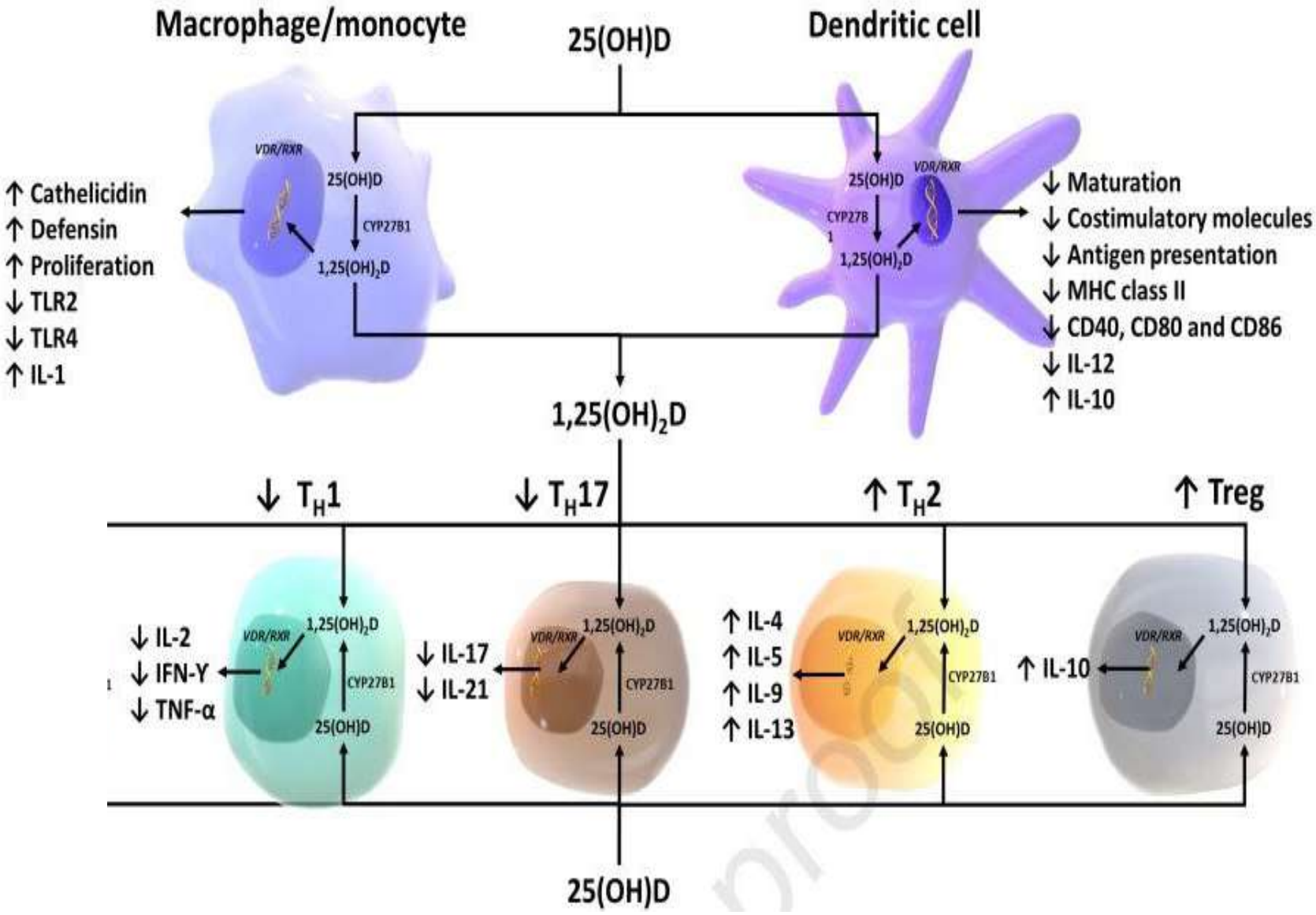
Adaptive immunity

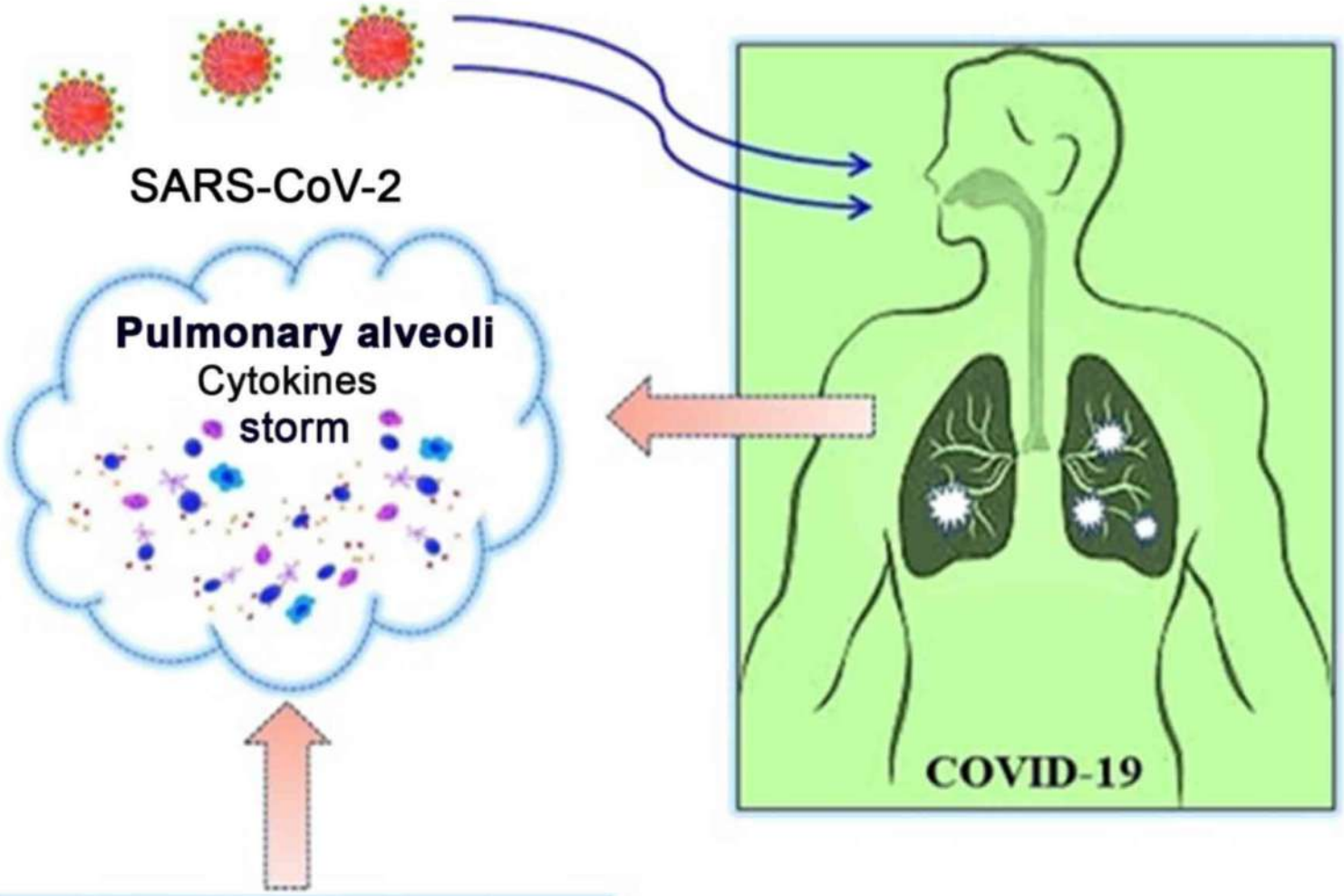




Vitamin D on Adaptive Immune Response

- Vitamin D - **powerful regulator** of the immune system modulates this process by -
  - **Promotes a timely shift from Th1 to Th2** cell immune profile (Cell-mediated to Antibody-mediated immunity)
  - It **suppresses the Th17 reaction** caused by over production of the 'immune messenger' cytokine called interleukin 17
  - It **facilitates differentiation of the T regulatory cells** that balance the immune response.


Macrophage/monocyte
25(OH)D
Dendritic cell
↑ Cathelicidin
↑ Defensin
↑ Proliferation
↓ TLR2
↓ TLR4
↑ IL-1
VDR/RXR
25(OH)D
CYP27B1
1,25(OH)2D
↓ Maturation
↓ Costimulatory molecules
↓ Antigen presentation
↓ MHC class II
↓ CD40, CD80 and CD86
↓ IL-12
↑ IL-10
1,25(OH)2D
↓ TH1
↓ TH17
↑ TH2
↑ Treg
↓ IL-2
↓ IFN-Y
↓ TNF-α
↓ IL-17
↓ IL-21
↑ IL-4
↑ IL-5
↑ IL-9
↑ IL-13
↑ IL-10
25(OH)D



Vitamin D

↓ Viral replication
↑ Physical barrier
↑ Cellular natural immunity
↑ Adaptive immunity: ↓ Th1/Th17
$CD4^+$ T cells, ↓TNF-α, ↓IFN-γ

ARDS & Multiple Organ Failure

Acute Respiratory Distress Syndrome

Renin-Angiotensin System (RAS)

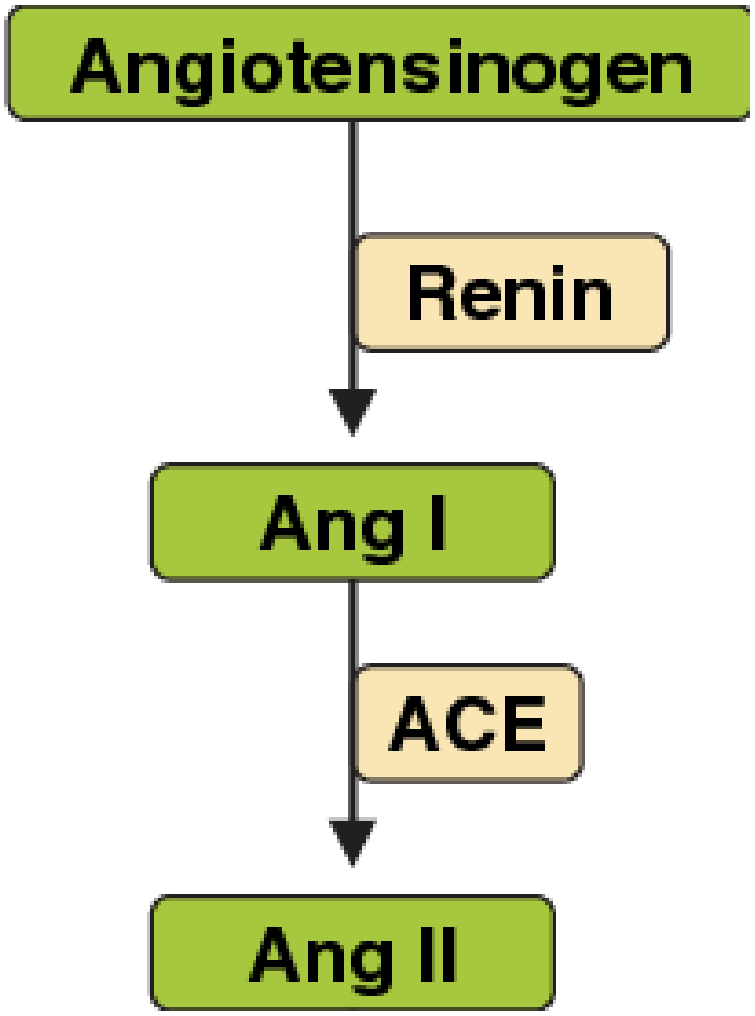
ELSEVIER

Pulmonary Pharmacology & Therapeutics

Volume 58, October 2019, 101833

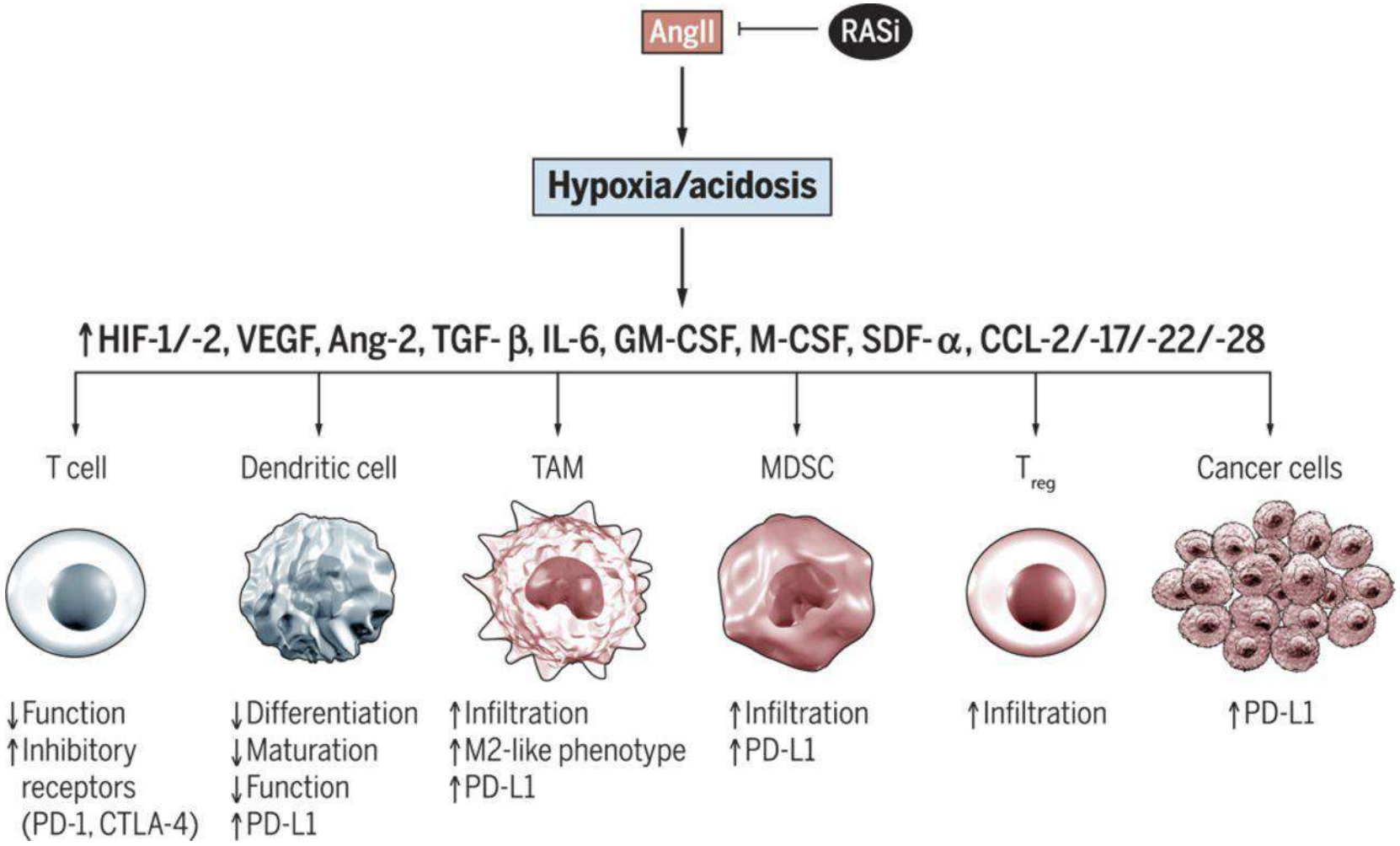
Renin-angiotensin-system, a potential pharmacological candidate, in acute respiratory distress syndrome during mechanical ventilation

Renin-Angiotensin System (RAS)

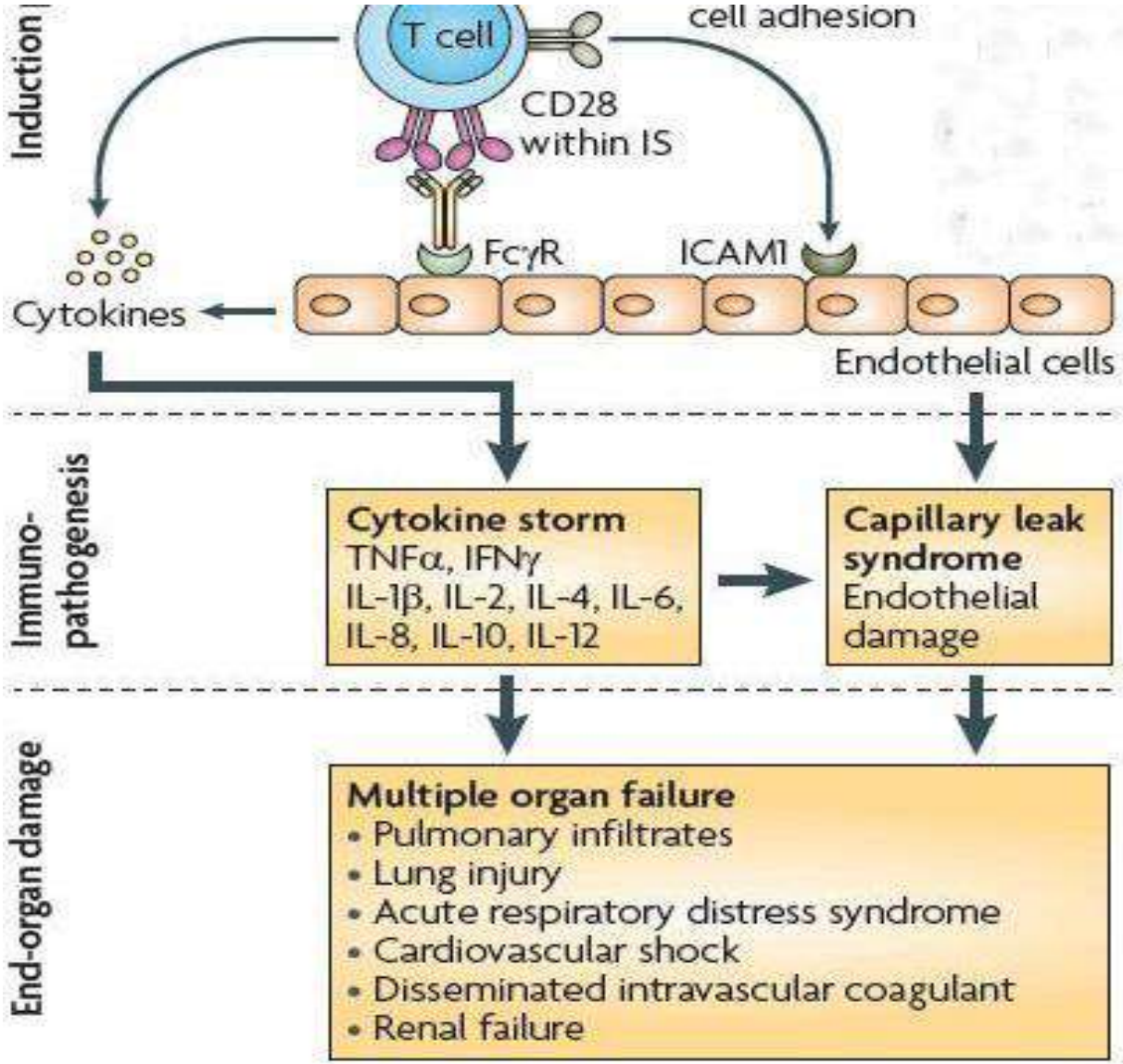


- RAS is a complex network that plays a major role in maintaining blood pressure, fluid and salt balance.
- Angiotensin II is the central biological effector of the RAS and the most potent constrictor of blood vessels.
- Angiotensin II is inflammatory in nature and its levels are linked to disease severity

Cytokine storm
Uncontrolled Angiotensin II



Cytokine storm & consequences



Molecular Medicine Reports

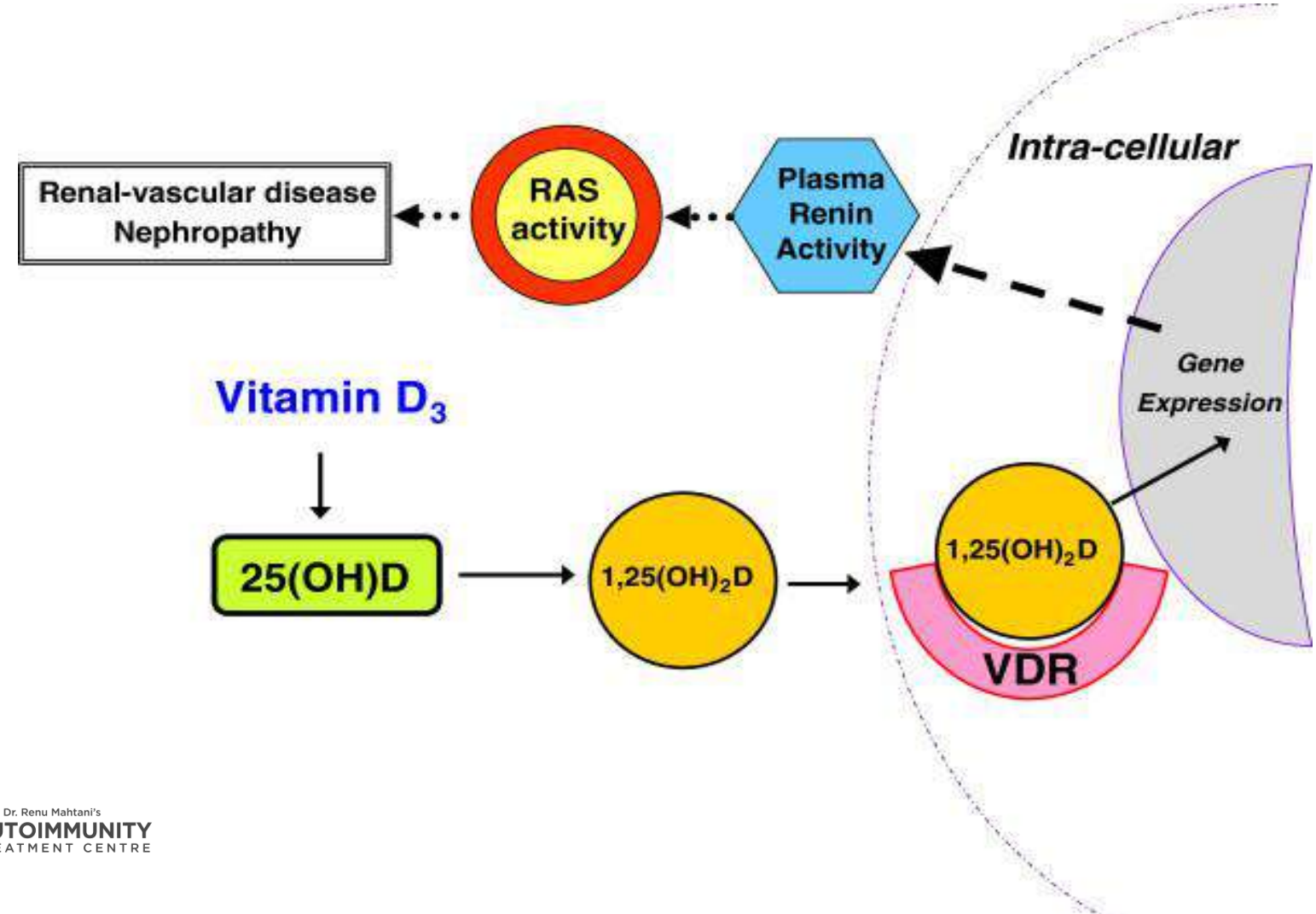
Vitamin D alleviates lipopolysaccharide-induced acute lung injury via regulation of the renin-angiotensin system

Open Access

Authors: Jun Xu, Jialai Yang, Jian Chen, Qingli Luo, Qiu Zhang, Hong Zhang

View Affiliations

Vitamin D suppresses Renin production through it's nuclear receptors (VDR)



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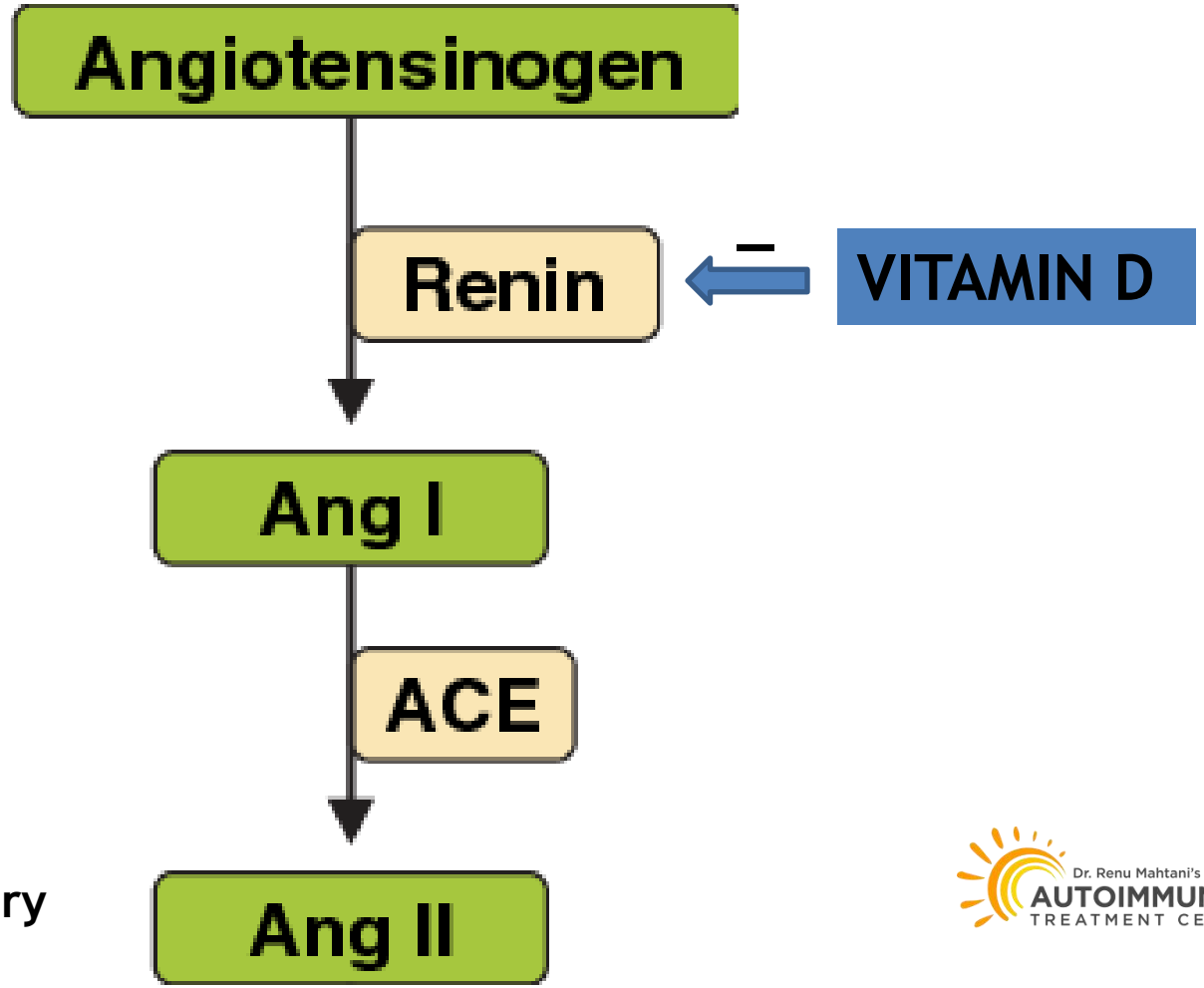
Vitamin D down regulates the RAS



Pro-inflammatory
Vasoconstrictor

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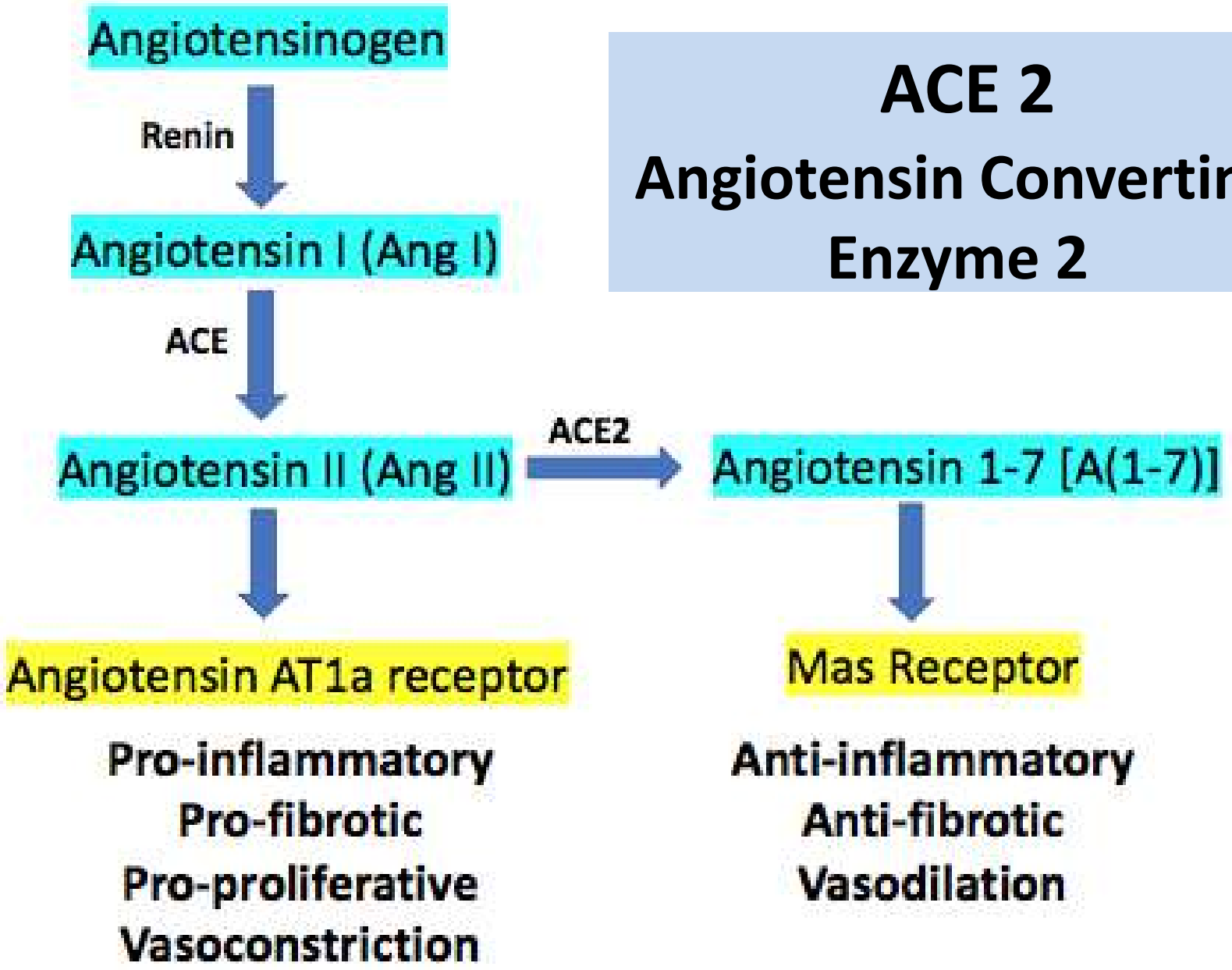
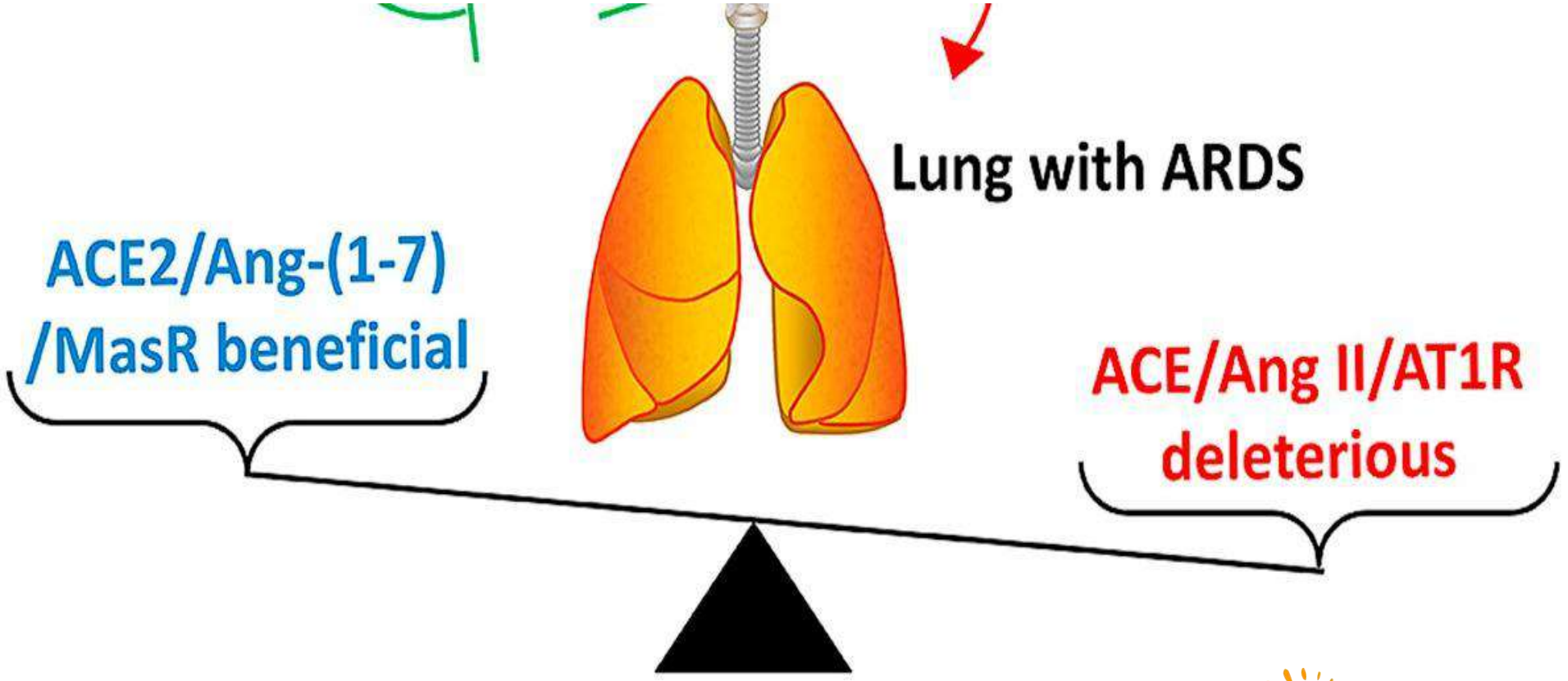

Angiotensinogen
Renin
Angiotensin I (Ang I)
ACE
Angiotensin II (Ang II)
ACE2
Angiotensin 1-7 [A(1-7)]
Angiotensin AT1a receptor
Mas Receptor
Pro-inflammatory
Pro-fibrotic
Pro-proliferative
Vasoconstriction
Anti-inflammatory
Anti-fibrotic
Vasodilation
ACE 2
Angiotensin Converting
Enzyme 2

ACE2 nullifies Angiotensin II

Is protective towards ARDS and lung injury



Angiotensin-converting enzyme 2 protects from severe acute lung failure

Yumiko Imai[1]*, Keiji Kuba[1]*, Shuan Rao[2], Yi Huan[2], Feng Guo[2], Bin Guan[2], Peng Yang[2], Renu Sarao[1], Teiji Wada[1], Howard Leong-Poi[3], Michael A. Crackower[4], Akiyoshi Fukamizu[5], Chi-Chung Hui[6], Lutz Hein[7], Stefan Uhlig[8], Arthur S. Slutsky[9], Chengyu Jiang[2] & Josef M. Penninger[1]

Acute respiratory distress syndrome (ARDS), the most severe form of acute lung injury, is a devastating clinical syndrome with a high mortality rate (30–60%) (refs 1–3). Predisposing factors for ARDS are diverse[1,3] and include sepsis, aspiration, pneumonias and infections with the severe acute respiratory syndrome (SARS) coronavirus[4,5]. At present, there are no effective drugs for improving the clinical outcome of ARDS[1–3]. Angiotensin-converting enzyme (ACE) and ACE2 are homologues with different key functions in the renin–angiotensin system[6–8]. ACE cleaves angiotensin I to generate angiotensin II, whereas ACE2 inactivates angiotensin II and is a negative regulator of the system. ACE2 has also recently been identified as a potential SARS virus receptor and is expressed in lungs[9,10]. Here we report that ACE2 and the angiotensin II type 2 receptor (AT_2) protect mice from severe acute lung injury induced by acid aspiration or sepsis. However, other components of the renin–angiotensin system, including ACE, angiotensin II and the angiotensin II type 1a receptor (AT_1a), promote disease pathogenesis, induce lung oedemas and impair lung function. We show

(Fig. 1b) and the development of pulmonary oedema (Fig. 1c). Acid aspiration resulted in increased alveolar wall thickness, oedema, bleeding, inflammatory cell infiltrates and formation of hyaline membranes (Fig. 1d). Notably, acid-treated *Ace2* knockout mice[9] showed significantly greater lung elastance compared with control wild-type mice, but there were no differences in lung elastance between saline-treated *Ace2* knockout and wild-type mice (Fig. 1a). Moreover, loss of *Ace2* resulted in worsened oxygenation (Fig. 1b), massive lung oedema (Fig. 1c), increased inflammatory cell infiltration and hyaline membrane formations (Fig. 1d) in response to acid aspiration. It should be noted that ACE2 protein expression is typically downregulated in wild-type mice following acid challenge (Fig. 1e).

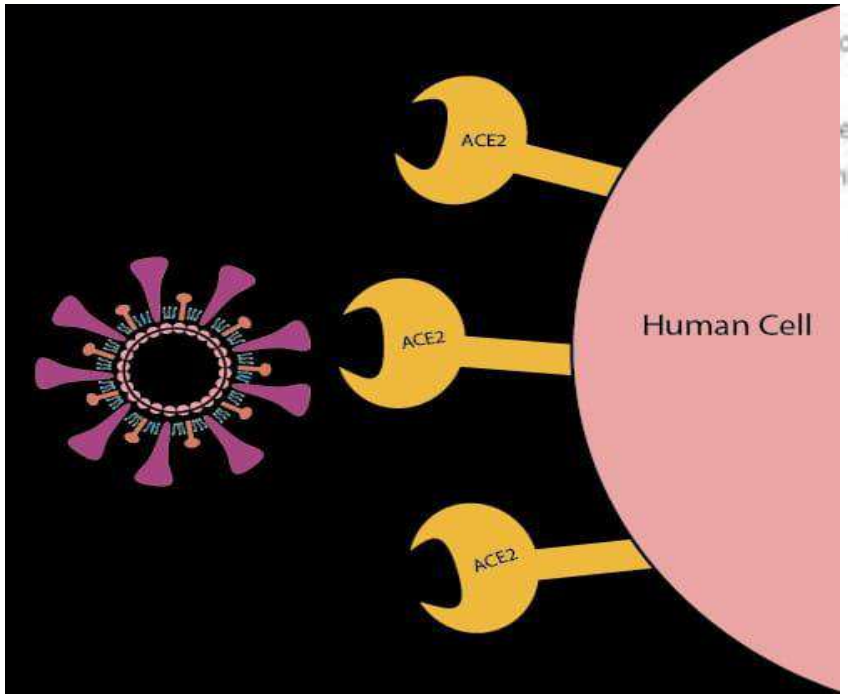
Sepsis is the most common cause of acute lung injury/ARDS[1–3]. We therefore examined the effect of *Ace2* gene deficiency on sepsis-induced acute lung injury using caecal ligation and perforation (CLP)[20]. CLP causes lethal peritonitis and sepsis, a polymicrobial infection that is accompanied by acute lung failure[20].

AMERICAN SOCIETY FOR MICROBIOLOGY | Journal of Virology®

Coronavirus targets ACE2

Entry of Human Coronavirus NL63 into the Cell

Aleksandra Milewska,[a,b] Paulina Nowak,[a,b] Katarzyna Owczarek,[a,b] Artur Szczepanski,[a,b] Miroslaw Zarebski,[c] Agnieszka Hoang,[c] Krzysztof Berniak,[c] Jacek Wojarski,[d] Slawo Krzysztof Pyrc[a,b]



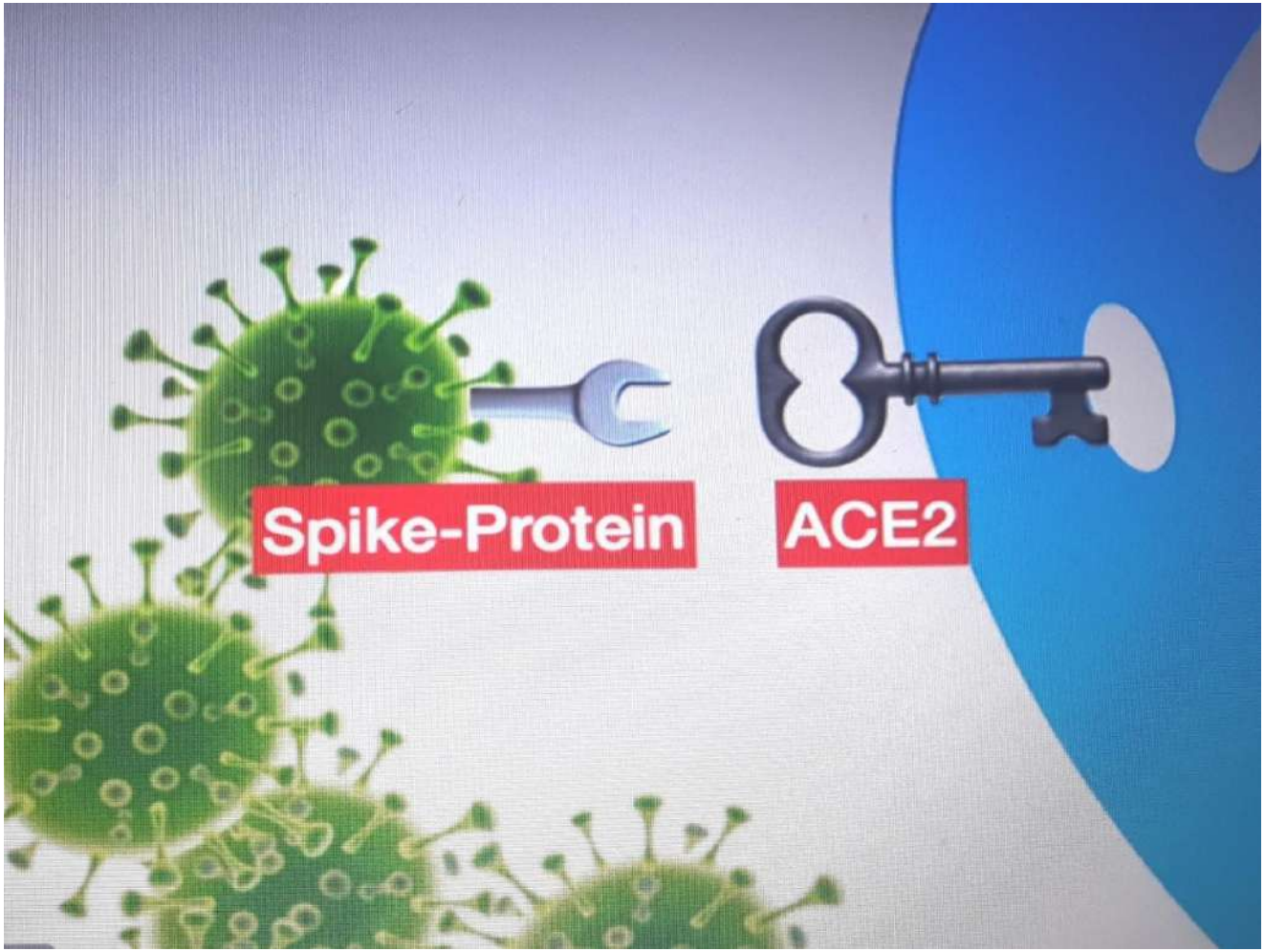
Angiotensin-converting enzyme 2 is a functional receptor for the SARS coronavirus

Wenhui Li[1], Michael J. Moore[1], Natalya Vasilieva[2], Jianhua Sui[3], Swee Kee Wong[1], Michael A. Berne[4], Mohan Somasundaran[5], John L. Sullivan[5], Katherine Luzuriaga[6], Thomas C. Greenough[5], Hyeryun Choe[2] & Michael Farzan[1]

[1]*Partners AIDS Research Center, Brigham and Women's Hospital, Department of Medicine (Microbiology and Molecular Genetics),* [2]*Perlmutter Laboratory, Pulmonary Division, Children's Hospital, Department of Pediatrics,* [3]*Dana-Farber Cancer Institute, Department of Medicine, Harvard Medical School, Boston, Massachusetts 02115, USA*
[4]*Tufts University Core Facility, Tufts University School of Medicine, Boston, Massachusetts 02111, USA*
[5]*Program in Molecular Medicine, University of Massachusetts Medical School, Worcester, Massachusetts 01605, USA*

Spike (S) proteins of coronaviruses, including the coronavirus that causes severe acute respiratory syndrome (SARS), associate with cellular receptors to mediate infection of their target cells[1,2]. Here we identify a metallopeptidase, angiotensin-converting enzyme 2 (ACE2)[3,4], isolated from SARS coronavirus (SARS-CoV)-permissive Vero E6 cells, that efficiently binds the S1 domain of the SARS-CoV S protein. We found that a soluble form of ACE2, but not of the related enzyme ACE1, blocked association of the S1 domain with Vero E6 cells. 293T cells transfected with ACE2, but not those transfected with human immunodeficiency virus-1 receptors, formed multinucleated syncytia with cells expressing S protein. Furthermore, SARS-CoV replicated efficiently on ACE2-transfected but not

Spike Protein inactivates ACE2



ACE2 & ARDS

- The corona virus through its S spike – protein uses the ACE2 receptor of the cell surface to enter the cell. This deactivates ACE2.
- ACE2 is the enzyme that inactivates the inflammatory AT2 (angiotensin 2).
- Virus-blocked ACE2 with a storm of Angiotensin 2 is the cause of pulmonary edema with shock lung.
- In order to avoid ARDS (acute respiratory distress syndrome) regulation of the angiotensin system is necessary.

[Lessons From SARS: A New Potential Therapy for Acute Respiratory Distress Syndrome (ARDS) With Angiotensin Converting Enzyme 2 (ACE2)]

Effect of vitamin D on ACE2 and vitamin D receptor expression in rats with LPS-induced acute lung injury

DOI

VernacularTitle: 维生素D对脂多糖致急性肺损伤大鼠肺组织血管紧张素转化酶2和维生素D受体表达水平的影响

Author: Jialai YANG[1]; Jun XU; Hong ZHANG
Author Information

Keywords: Vitamin D (Vit D); Calcitriol; Vitamin D receptor (VDR); Lipopolysaccharide (LPS); Acute lung injury; Angiotensin converting enzyme 2 (ACE2)

From:

Country

Language:

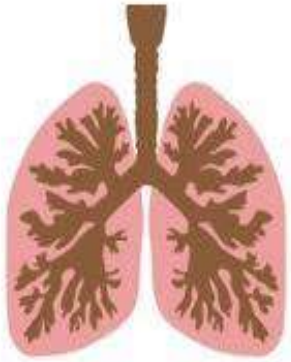
Abstract:

Calcitriol (Vitamin D) increases the expression of ACE2 suggesting that ACE2 plays a role in protection against the development of ALI (Acute Lung Injury)

shallow breathing;listlessness;the oral and nose hemorrhage) in LPS group were obvious,and the clinical manifestations and

Vitamin D
How can it protect

ENZYME	QUALITY & TARGET	CAN VITAMIN D HELP
Angiotensin II	Bad - reduce it	YES
ACE2	Good – Increase it	YES
Spike Protein	Bad – nullify it	YES



Angiotensinogen

Renin

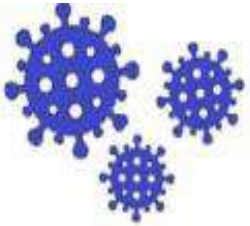
Angiotensin-(1-9)

Angiotensin I

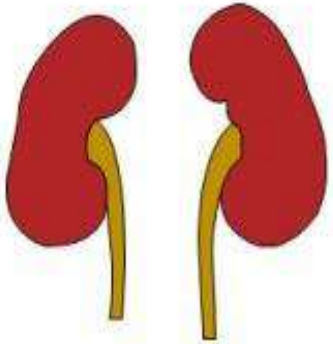
$1,25(OH)_2D$

ACE

Bradykinin storm



SARS-CoV-2



Kidney injury

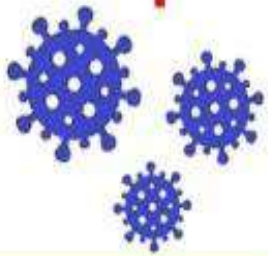
Angiotensin II

Angiotensin-(1-7)

ACE2

AT_1 receptor

Mas receptor



ARDS
Myocarditis
Cardiac injury

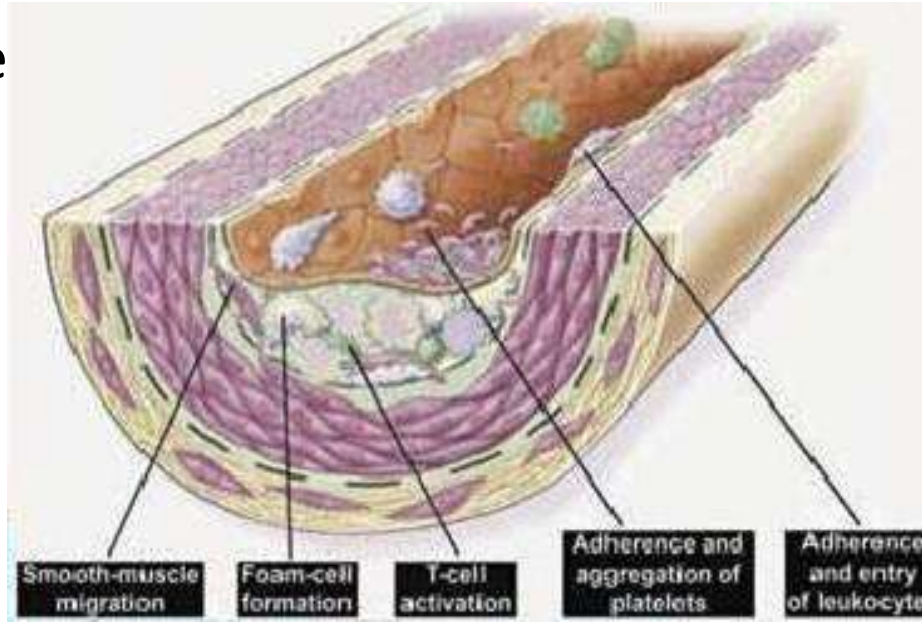
↑ Inflammation
↑ Apoptosis
Vasoconstriction

↓ Inflammation
↓ Apoptosis
Vasodilatation

SARS-CoV-2

Vitamin D – Effects on Vascular Endothelium

- VDR present in the endothelium, vascular smooth muscles and cardiomyocytes
- Protects via -
  - Inhibition of macrophage cholesterol uptake & foam cell formation
  - Reduced vascular smooth muscle proliferation
  - Reduced expression of adhesion molecules
  - Reduce cytokine release from lymphocytes



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biomolecules

MDPI

Review

Emerging Role of Vitamin D and its Associated Molecules in Pathways Related to Pathogenesis of Thrombosis

Syed Mohammad, Aastha Mishra and Mohammad Zahid Ashraf *

Department of Biotechnology, Faculty of Natural Sciences, Jamia Millia Islamia, New Delhi 110025, India; mahmudaga@gmail.com (S.M.); aastha0602@gmail.com (A.M.)

* Correspondence: zashraf@jmi.ac.in

PLOS ONE

RESEARCH ARTICLE

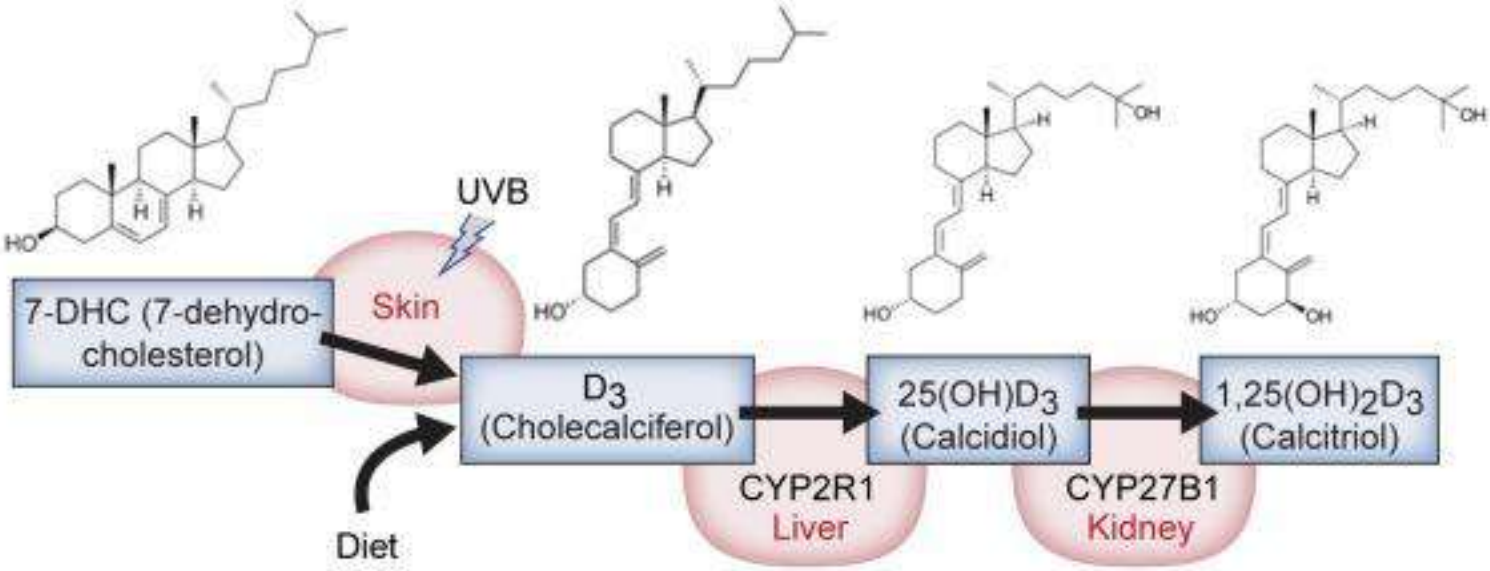
Dietary Vitamin D and Its Metabolites Non-Genomically Stabilize the Endothelium

Christopher C. Gibson[1,2,3☯], Chadwick T. Davis[1,3,4☯], Weiquan Zhu[1], Jay A. Bowman-Kirigin[1], Ashley E. Walker[5], Zhengfu Tai[6], Kirk R. Thomas[1,3], Anthony J. Donato[5], Lisa A. Lesniewski[5], Dean Y. Li[1,3,4,6,7,8,9]*

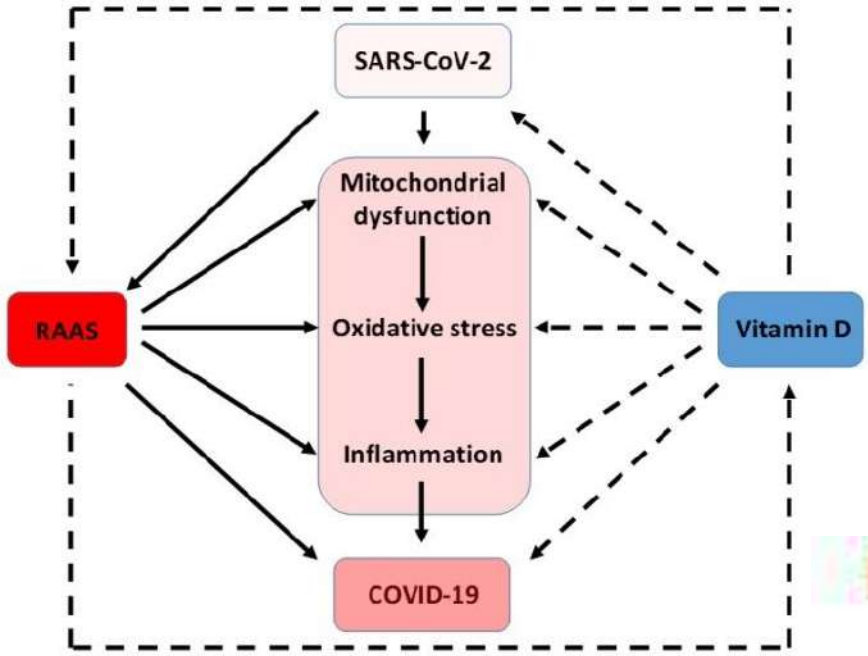
1 Program in Molecular Medicine, University of Utah, Salt Lake City, Utah, 84112, United States of America, **2** Department of Bioengineering, University of Utah, Salt Lake City, Utah, 84112, United States of America, **3** Recursion Pharmaceuticals, LLC, Salt Lake City, Utah, 84108, United States of America, **4** Department of Human Genetics, University of Utah, Salt Lake City, Utah, 84112, United States of America, **5** Division of Geriatrics, Department of Medicine, University of Utah, Salt Lake City, Utah, 84112, United States of America, **6** The Key Laboratory for Human Disease Gene Study of Sichuan Province, Institute of Laboratory

Endothelial stabilisation

Vit D3 > 25 OH D3 > 1,25 (OH)2 D3



Normal circulating levels*	NA	1-90 nM	30-80 nM	50-100 pm
Enhancement of endothelial stability (minimum active dose)	NA	<100 pM	100 nM	1 nM
Enhancement of endothelial stability	Inactive	Active	Active	Active



Mitochondrial dysfunction & Oxidative Stress

Vitamin D Mechanisms Prevent Covid-19 Disease

- Immune Response
  - Innate Immunity
  - Adaptive Immunity
- Renin-Angiotensin System Regulation
  - Cytokine Storm
  - Bradykinin Storm
- Endothelial Vascular Stabilisation
- Lung Fibrosis & Inflammation Suppression
- Oxidative Stress & Mitochondrail Dysfunction

Vitamin D is not an optional supplement. It is a non negotiable cellular necessity. It is your life health support system.

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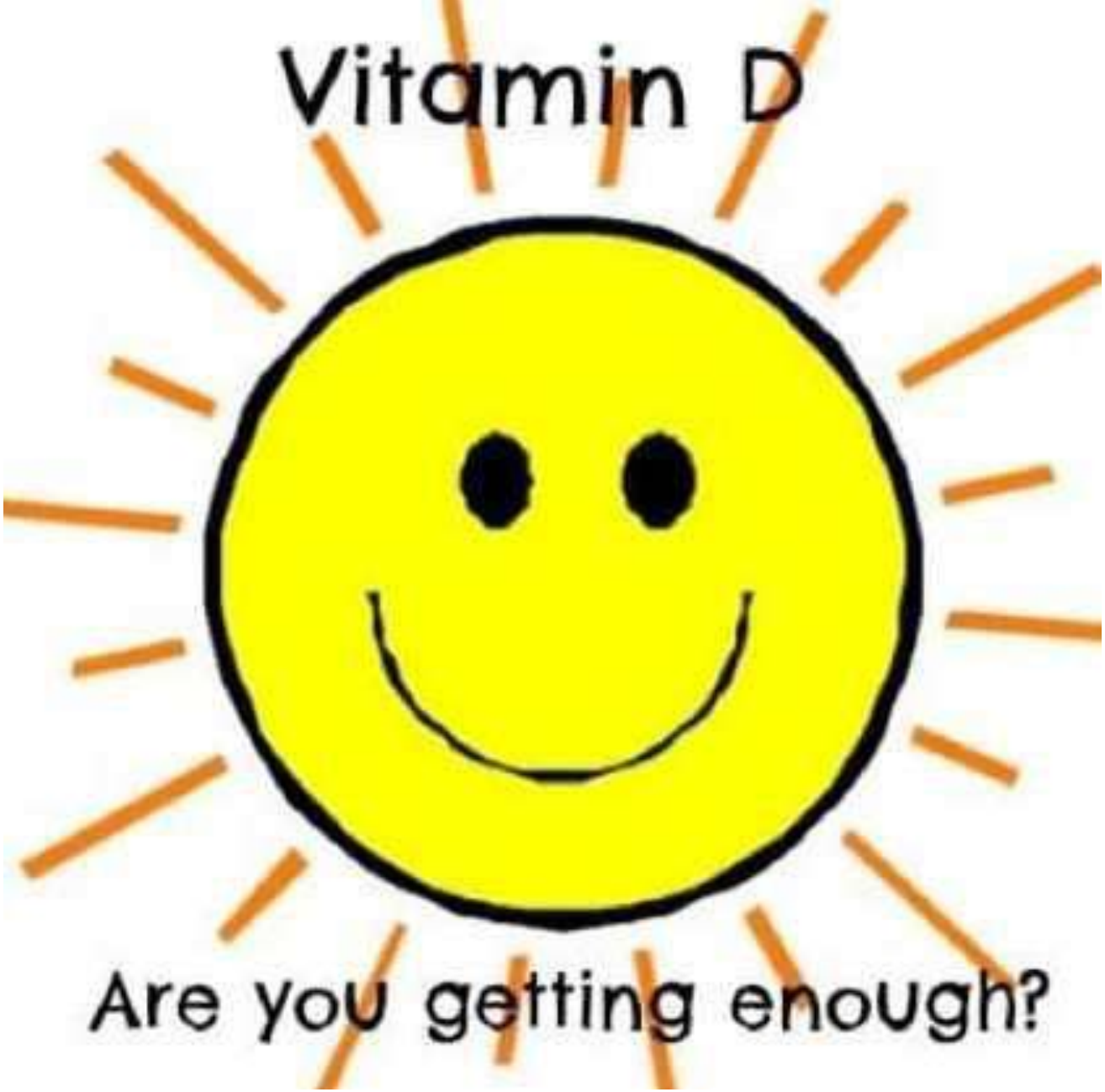
AUTOIMMUNITY
TREATMENT CENTRE



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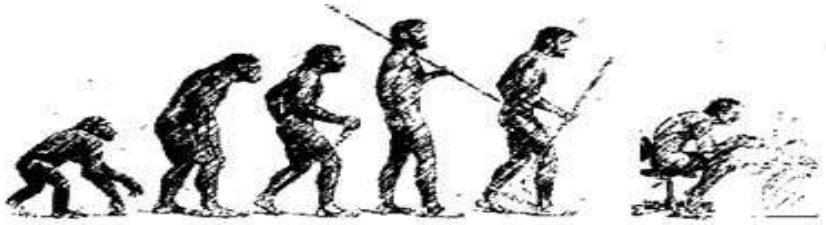
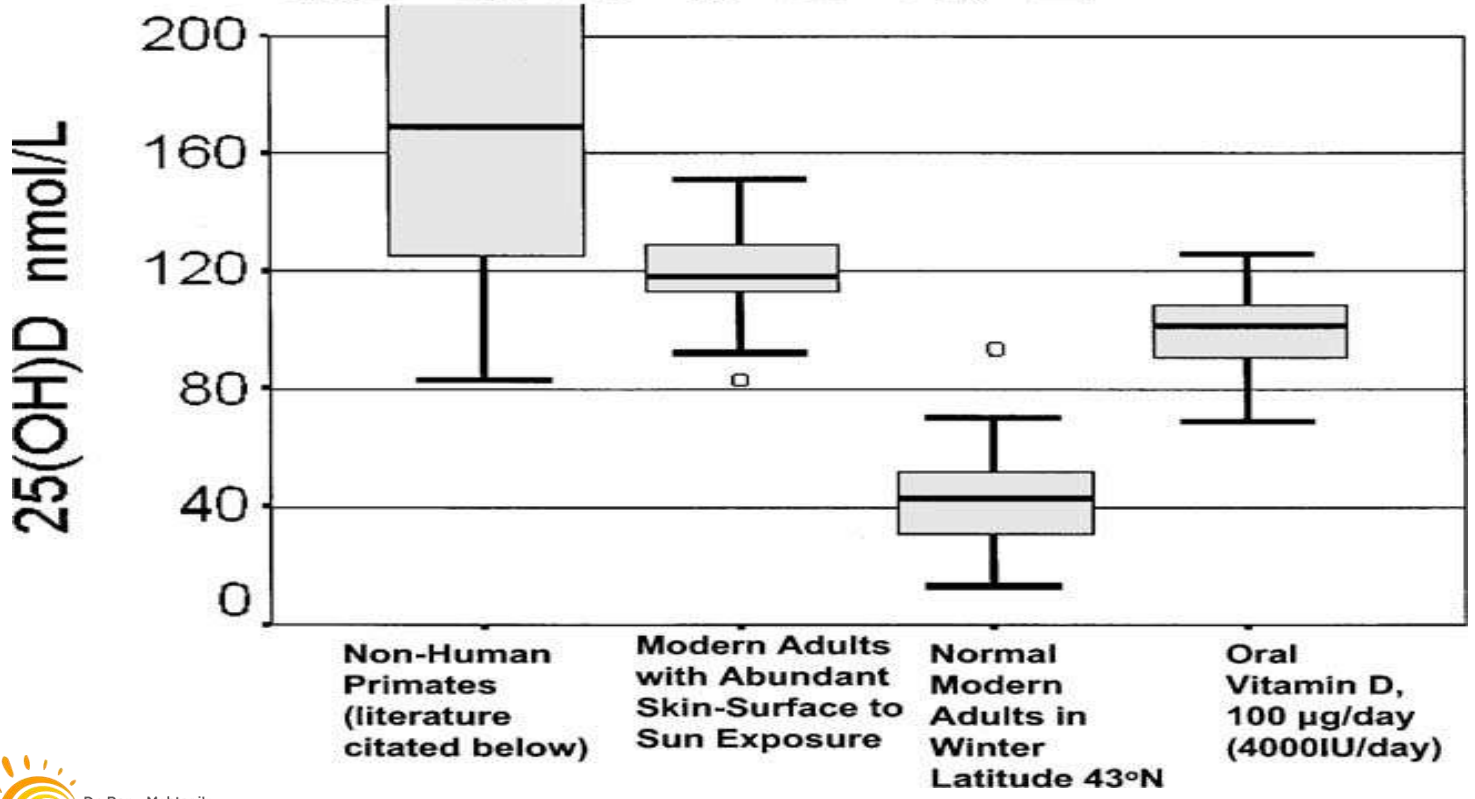
The physiological range for circulating D3 in humans extends to beyond 80 ng/ml





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Vitamin D levels (ng/ml)

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Causes of Vitamin D Deficiency

- Lack of adequate healthy sun exposure – Lifestyle & Pollution
- Lack of vitamin D in diet (vegetarian food is devoid of Vitamin D)
- Inability to absorb adequately from intestines
- Inability to process vitamin D
  - Unhealthy liver & kidney
  - Widespread Magnesium deficiency
  - Endocrine disruptors
- Genetic polymorphism s in VDR (Vitamin D receptor)
- Low RDA's

Perspective

J Prev Med Public Health 2017;50:278-281 • https://doi.org/10.3961/jpmph.16.111

pISSN 1975-8375 eISSN 2233-4521

Journal of Preventive Medicine & Public Health

The Big Vitamin D Mistake

Dimitrios T. Papadimitriou[1,2]

[1]*Third Department of Pediatrics, Division of Pediatric Endocrinology, Attikon University Hospital, University of Athens School of Medicine, Athens;*
[2]*Pediatric-Adolescent Endocrinology and Diabetes, Athens Medical Center, Athens, Greece*

Since 2006, type 1 diabetes in Finland has plateaued and ther
with cholecalciferol. The role of vitamin D in innate and adapt
mended dietary allowance (RDA) for vitamin D was recently d
cine, it was found that 8895 IU/d was needed for 97.5% of in
6201 IU/d was needed to achieve 75 nmol/L and 9122 IU/d w
ed of studies published between 1966 and 2013 showed that
associated with higher all-cause mortality, demolishing the p
D levels. Since all-disease mortality is reduced to 1.0 with se

20 ng/ml blood levels are sub-optimal

40 ng/ml is the minimal desired level

BMJ open

Vitamin D_3 supplementation in patients with frequent respiratory tract infections: a randomised and double-blind intervention study

Peter Bergman,[1,2,3] Anna-Carin Norlin,[2,4] Susanne Hansen,[2] Rokeya Sultana Rekha,[5] Birgitta Agerberth,[5] Linda Björkhem-Bergman,[6] Lena Ekström,[6] Jonatan D Lindh,[6] Jan Andersson[3]

To cite: Bergman P, Norlin A-C, Hansen S, *et al*. Vitamin D_3 supplementation in patients with frequent respiratory tract infections: a randomised and double-blind intervention study. *BMJ Open* 2012;**2**: e001663. doi:10.1136/bmjopen-2012-001663

ABSTRACT

Background: Low serum levels (D_3 are associated with an increas tract infections (RTIs). Clinical tri against various infections have be data are so far not conclusive. Th additional randomised controlled vitamin D_3 on infections.

Objective: To investigate if supp vitamin D_3 could reduce infectiou

Maintenance of Vit D serum concentration of 40 ng/ml or higher reduces acute viral respiratory tract infections

European Review for Medical and Pharmacological Sciences 2020; 24: 4048-4052

Editorial – Vitamin D status: a key modulator of innate immunity and natural defense from acute viral respiratory infections

A. FABBRI[1], M. INFANTE[1,2], AND C. RICORDI[2]

[1]Endocrine Unit, CTO Hospital – ASL Roma 2, Department of Systems Medicine, University of Rome "Tor Vergata", Rome, Italy
[2]Diabetes Research Institute (DRI), University of Miami Miller School of Medicine, Miami, FL, USA

Key Words:
Vitamin D, Vitamin D deficiency, Innate immunity, Viral respiratory infections, Covid-19.

'Maintenance of circulating 25-hydroxyvitamin D levels of 40-60 ng/mL would be optimal…..

What is the healthy vitamin D level for 2021

	Recommendations by Vitamin D Council	Recommendations by The Endocrine Society	Recommendations by Testing Laboratories
Deficient	0-30 ng/ml	0-20 ng/ml	0-31 ng/ml
Insufficient	31-39 ng/ml	21-29 ng/ml	
Sufficient	40-80 ng/ml	30-100 ng/ml	32-100 ng/ml
Toxic	>150 ng/ml	Aim for this!	VitSupp

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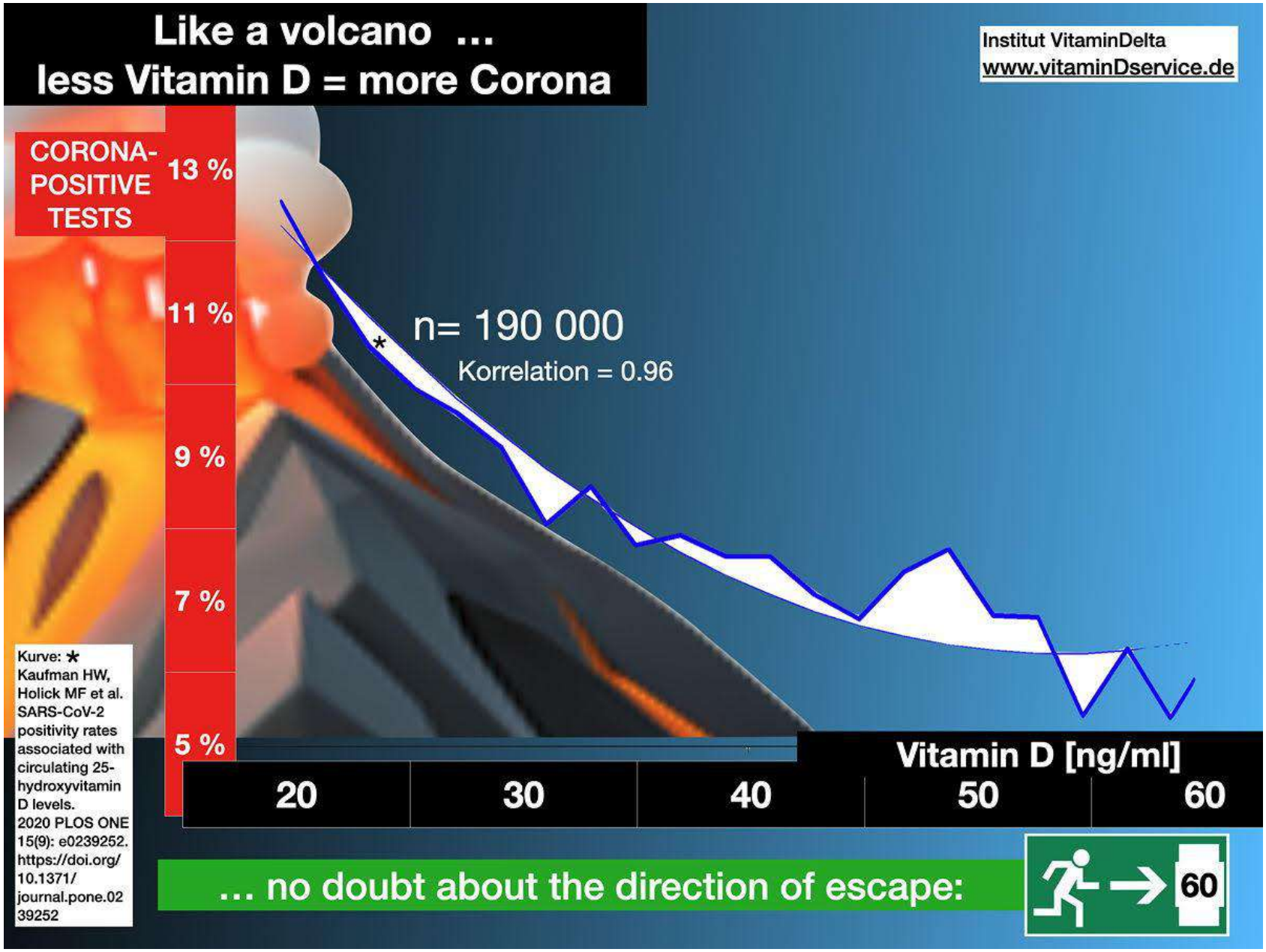
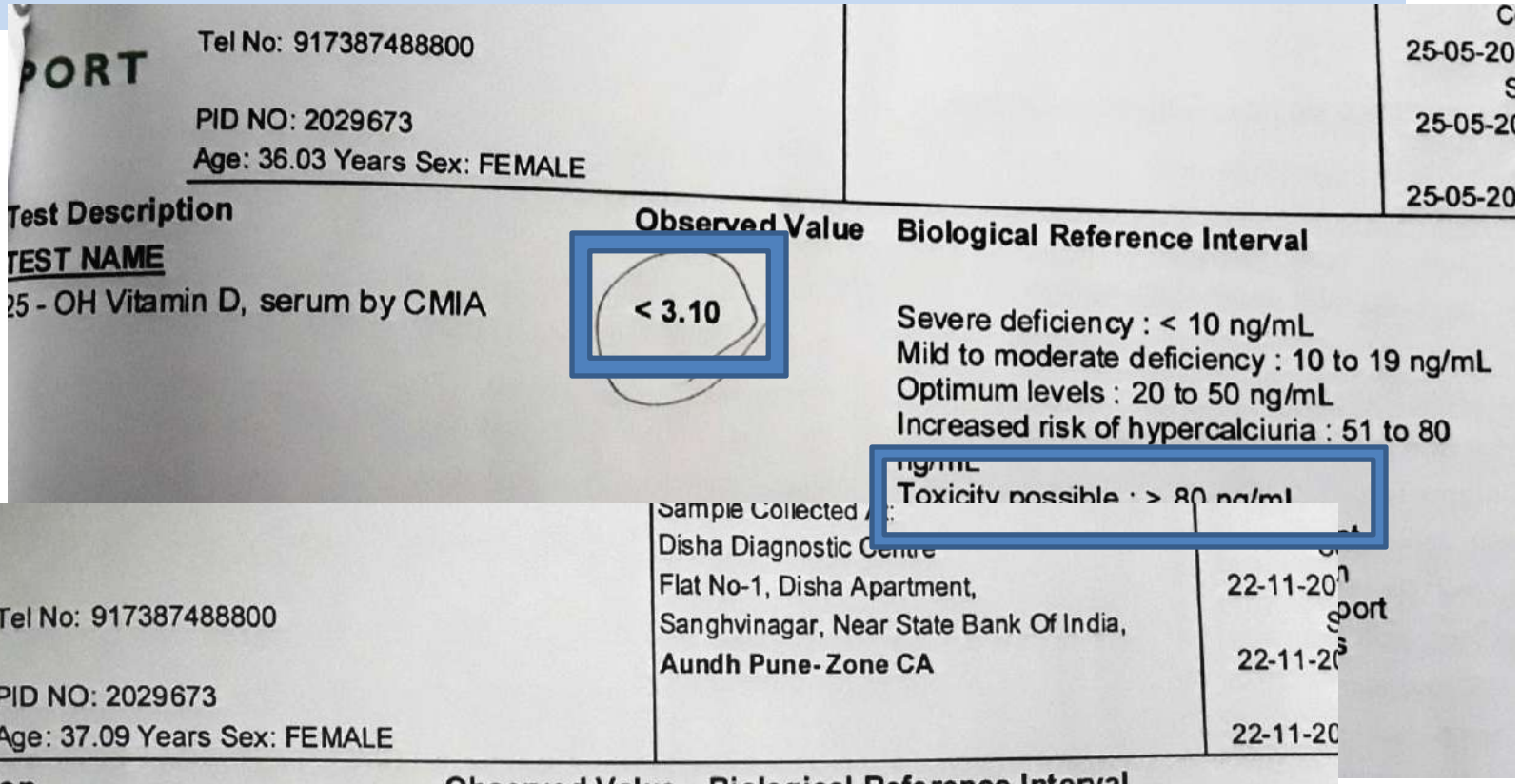

Like a volcano ...
less Vitamin D = more Corona
Institut VitaminDelta
www.vitaminDservice.de
CORONA-POSITIVE TESTS
13 %
11 %
9 %
7 %
5 %
n= 190 000
Korrelation = 0.96
*
Kurve: * Kaufman HW, Holick MF et al. SARS-CoV-2 positivity rates associated with circulating 25-hydroxyvitamin D levels. 2020 PLOS ONE 15(9): e0239252. https://doi.org/10.1371/journal.pone.0239252
Vitamin D [ng/ml]
20
30
40
50
60
... no doubt about the direction of escape:
60

Reports seen commonly in 2021....



PORT

Tel No: 917387488800

PID NO: 2029673

Age: 36.03 Years Sex: FEMALE

25-05-20

25-05-20

25-05-20

Test Description	Observed Value	Biological Reference Interval
TEST NAME		
25 - OH Vitamin D, serum by CMIA	< 3.10	Severe deficiency : < 10 ng/mL Mild to moderate deficiency : 10 to 19 ng/mL Optimum levels : 20 to 50 ng/mL Increased risk of hypercalciuria : 51 to 80 ng/mL Toxicity possible : > 80 ng/mL

Sample Collected At:

Disha Diagnostic Centre

Flat No-1, Disha Apartment,

Sanghvinagar, Near State Bank Of India,

Aundh Pune-Zone CA

22-11-20

22-11-20

22-11-20

Tel No: 917387488800

PID NO: 2029673

Age: 37.09 Years Sex: FEMALE

Test Description	Observed Value	Biological Reference Interval
TEST NAME		
25 - OH Vitamin D, serum by CMIA	8.90	Severe deficiency : < 10 ng/mL Mild to moderate deficiency : 10 to 19 ng/mL Optimum levels : 20 to 50 ng/mL Increased risk of hypercalciuria : 51 to 80 ng/mL Toxicity possible : > 80 ng/mL These reference ranges represent clinical decision values, based on the 2011 Institute of Medicine report

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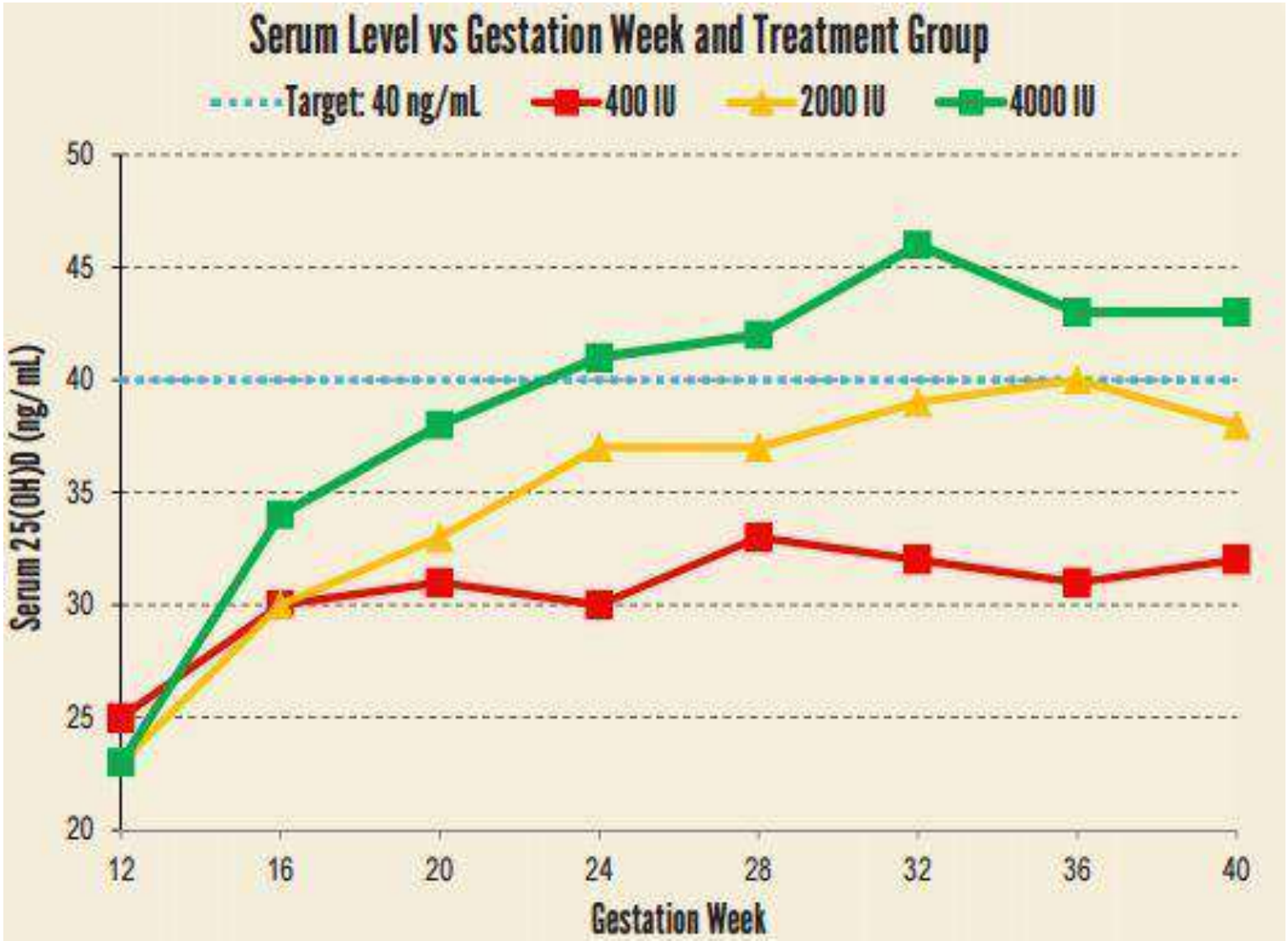
Without loading dose it takes 5 months (20 weeks) to get adequate vitamin D levels



Perspective

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Dimitrios T. Papadimitriou[1,2]

[1]Third Department of Pediatrics, Division of Pediatric Endocrinolo
[2]Pediatric-Adolescent Endocrinology and Diabetes, Athens Medic

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6201 IU/d was needed to achieve 75 nmol/L and 9122 IU/d
of studies published between 1966 and 2013 showed th
associated with higher all-cause mortality, demolishing the
D levels. Since all-disease mortality is reduced to 1.0 with serum vitamin D levels ≥100 nmol/L, we call public health authorities to

40 ng/ml is the minimal desired level

9000 IU/day intake is needed to reach 40 ng/ml

Vitamin D intake observed to produce noted 25(OH)D serum levels in 90% of adults (age 18 years and older), weighing 150 lbs. (N=7324)

RECOMMENDED RANGE: 40-60 ng/ml

WHAT TO DO

1. **Test**
2. **Establish recommended intake level**
3. **Test again in 3-6 months**

(For supplements, vitamin D3, cholecalciferol may be used.)

Individuals should consult with a health care practitioner to develop a custom plan.

Change in Serum Level Based on Intake (IU/day) for 90% of Adults* (N=7324)

Current Level (ng/ml) ▼ \ Expected Level (ng/ml) ▶	20	30	40	50	60
10	2000	4000	6000	10,000	10,000
15	1000	3000	6000	9000	10,000
20		2000	5000	8000	10,000
25		1000	4000	7000	10,000
30			3000	6000	10,000
35			1000	5000	9000
40				3000	8000
45				2000	6000
50					4000

** values rounded to the nearest 1000 IU; highest recommended intake is 10,000 IU/day*

Example: With a starting serum level of 20 ng/ml, an additional intake of approximately 5000 IU/day would be sufficient for 90% of adults (age 18 years and older, weighing 150 lbs) to achieve a serum level of at least 40 ng/ml.

Nutrition 28 (2012) 344–356

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Review

A 21st century evaluation of the safety of oral vitamin D

Michael J. Glade Ph.D.*

The Nutrition Doctor, Skokie, Illinois, USA

Outdoor workers can make 10000 to 25000 IU vitamin D daily

Long-term intakes of up to 10 000 IU/day of vitamin D maximize physiologic benefits and are safe.

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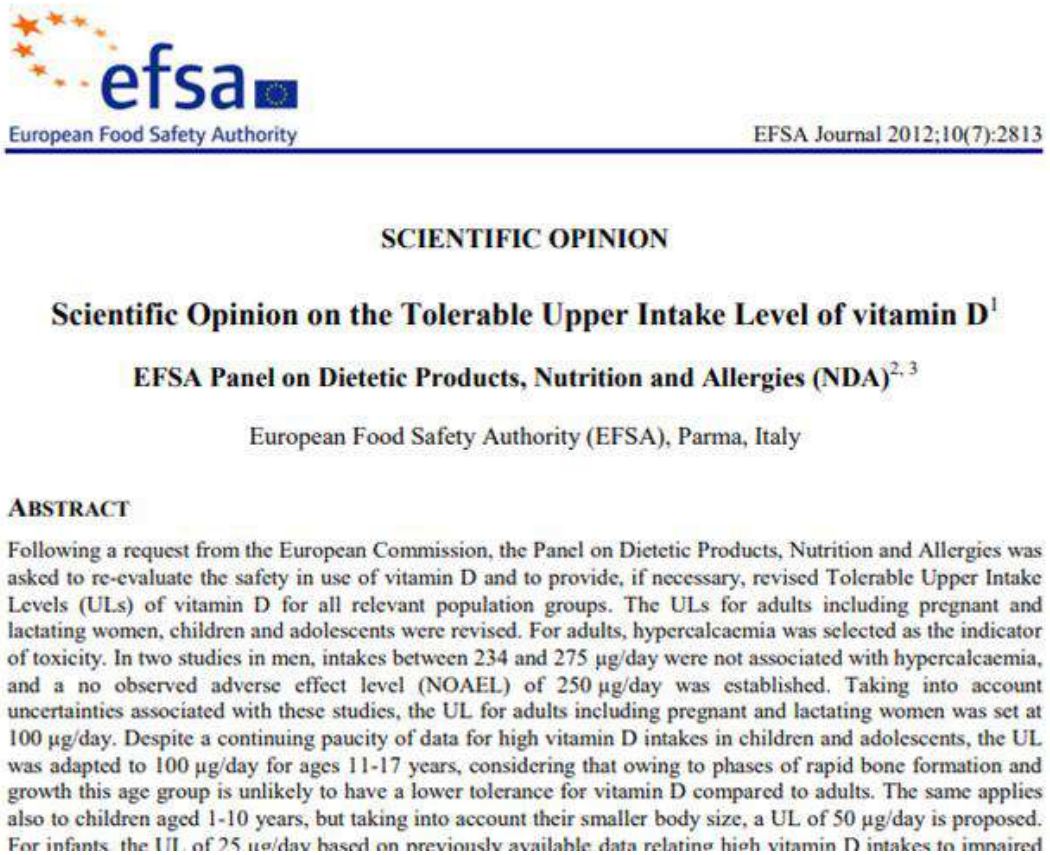
The NOAEL – No Observed Adverse Effect Level for Vitamin D

- 10000 IU/day for adults
- 4000 IU/day for young adolescents (11 – 17 yrs)
- 2000 IU/day for children (1-10 yrs)



efsa
European Food Safety Authority

EFSA Journal 2012;10(7):2813

SCIENTIFIC OPINION

Scientific Opinion on the Tolerable Upper Intake Level of vitamin D[1]

EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA)[2, 3]

European Food Safety Authority (EFSA), Parma, Italy

ABSTRACT

Following a request from the European Commission, the Panel on Dietetic Products, Nutrition and Allergies was asked to re-evaluate the safety in use of vitamin D and to provide, if necessary, revised Tolerable Upper Intake Levels (ULs) of vitamin D for all relevant population groups. The ULs for adults including pregnant and lactating women, children and adolescents were revised. For adults, hypercalcaemia was selected as the indicator of toxicity. In two studies in men, intakes between 234 and 275 µg/day were not associated with hypercalcaemia, and a no observed adverse effect level (NOAEL) of 250 µg/day was established. Taking into account uncertainties associated with these studies, the UL for adults including pregnant and lactating women was set at 100 µg/day. Despite a continuing paucity of data for high vitamin D intakes in children and adolescents, the UL was adapted to 100 µg/day for ages 11-17 years, considering that owing to phases of rapid bone formation and growth this age group is unlikely to have a lower tolerance for vitamin D compared to adults. The same applies also to children aged 1-10 years, but taking into account their smaller body size, a UL of 50 µg/day is proposed. For infants, the UL of 25 µg/day based on previously available data relating high vitamin D intakes to impaired

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No fear of toxicity with vitamin D supplementation @ 10000 IU/day (adults)

- Level - 40 – 60 ng/ml blood levels
- Dose – 10000 IU daily or 60000 IU once a week for 3 months followed by maintenance of half the dose

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CFP·MFC
Official Publication of
The College of
Family Physicians of Canada

Can Fam Physician. 2015 Jun; 61(6): 507.

PMCID: PMC4463890
PMID: 26071153

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AUTOIMMUNITY
TREATMENT CENTRE

Vitamin D for influenza

Gerry Schwalfenberg, MD CCFP FCFP

· Author information · Copyright and License information Disclaimer

Vitamin D for influenza

I thank Dr Korownyk and colleagues for their interesting review on the neuraminidase inhibitors.[1] Having spent some time looking at the reviews on these drugs myself, I agree that they are not very useful and the risk

A colleague of mine and I have introduced vitamin D at doses that have achieved greater than 100 nmol/L in most of our patients for the past number of years, and we now see very few patients in our clinics with the flu or influenzalike illness. In those patients who do have influenza, we have treated them with the *vitamin D hammer*, as coined by my colleague. This is a 1-time 50000 IU dose of vitamin D3 or 10000 IU 3 times daily for 2 to 3 days. The results are dramatic, with complete resolution of symptoms in 48 to 72 hours. One-time doses of vitamin D at this level have been used safely and have never been shown to be toxic.[8] We urgently need a study of this intervention. The cost of vitamin D is about a penny for 1000 IU, so this treatment costs less than a dollar.

—*Gerry Schwalfenberg MD CCFP FCFP*
Edmonton, Alta

Competing interests

Treated influenza with 50000 IU vitamin D daily for 2 to 3 days

Immediate Protection
Target – 40-60 ng/ml blood levels

- Not taking any Vit D for more than 6 months
- Blood levels – 10 - 20 ng/ml

– Vitamin D hammer - 50-60k IU daily for 3 days. After 1 week, 60k IU weekly or 10k IU daily for 3 months

– Blood test ideally to decide maintenance dose (usually 5k IU)

- Taking any Vit D 1000 – 5000 IU per day
- Blood levels – 25 - 35 ng /ml or less

– Replace with 50-60K IU once a week or 10k IU daily for 3 months and then the maintenance dose (5k IU)

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TREATMENT CENTRE

nutrients

MDPI

Article

Vitamin D Deficiency and Outcome of COVID-19 Patients

Aleksandar Radujkovic [1], Theresa Hippchen [2], Shilpa Tiwari-Heckler [2], Saida Dreher [2], Monica Boxberger [2] and Uta Merle [2,*]

1 Department of Internal Medicine V, University of Heidelberg, 69121 Heidelberg, Germany; aleksandar.radujkovic@med.uni-heidelberg.de

2 Department of Internal Medicine IV, University of Heidelberg, 69121 Heidelberg, Germany; theresa.hippchen@med.uni-heidelberg.de (T.H.); shilpa.tiwari-heckler@med.uni-heidelberg.de (S.T.-H.); saida.dreher@med.uni-heidelberg.de (S.D.); monica.boxberger@med.uni-heidelberg.de (M.B.)

Journal of Endocrinological Investigation
https://doi.org/10.1007/s40618-020-01370-x

ORIGINAL ARTICLE

Check for updates

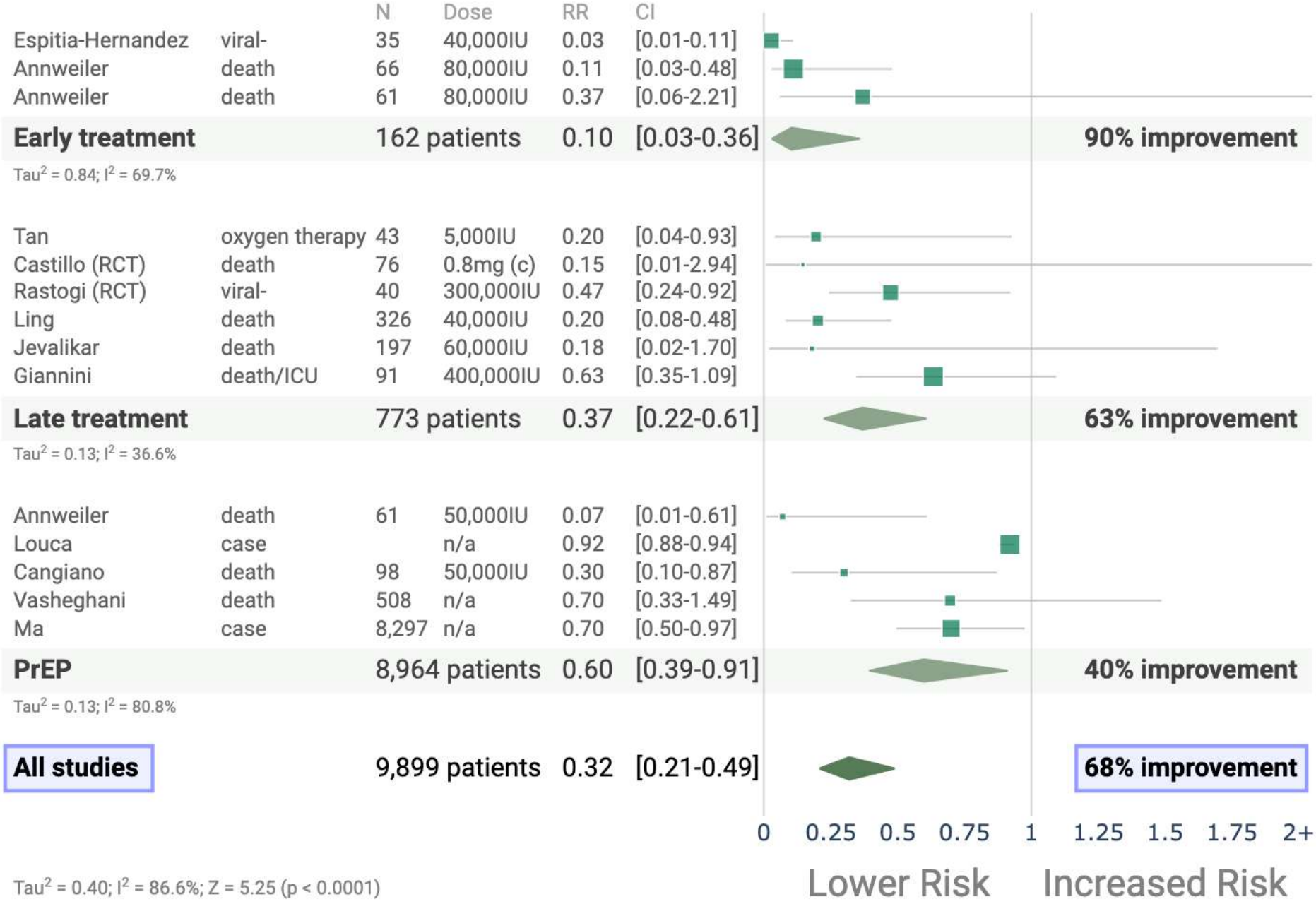
Vitamin D deficiency as a predictor of poor prognosis in patients with acute respiratory failure due to COVID-19

G. E. Carpagnano[1] · V. Di Lecce[1] · V. N. Quaranta[2] · A. Zito[3] · E. Buonamico[1] · E. Capozza[1] · A. Palumbo[1] · G. Di Gioia[1] · V. N. Valerio[1] · O. Resta[1]

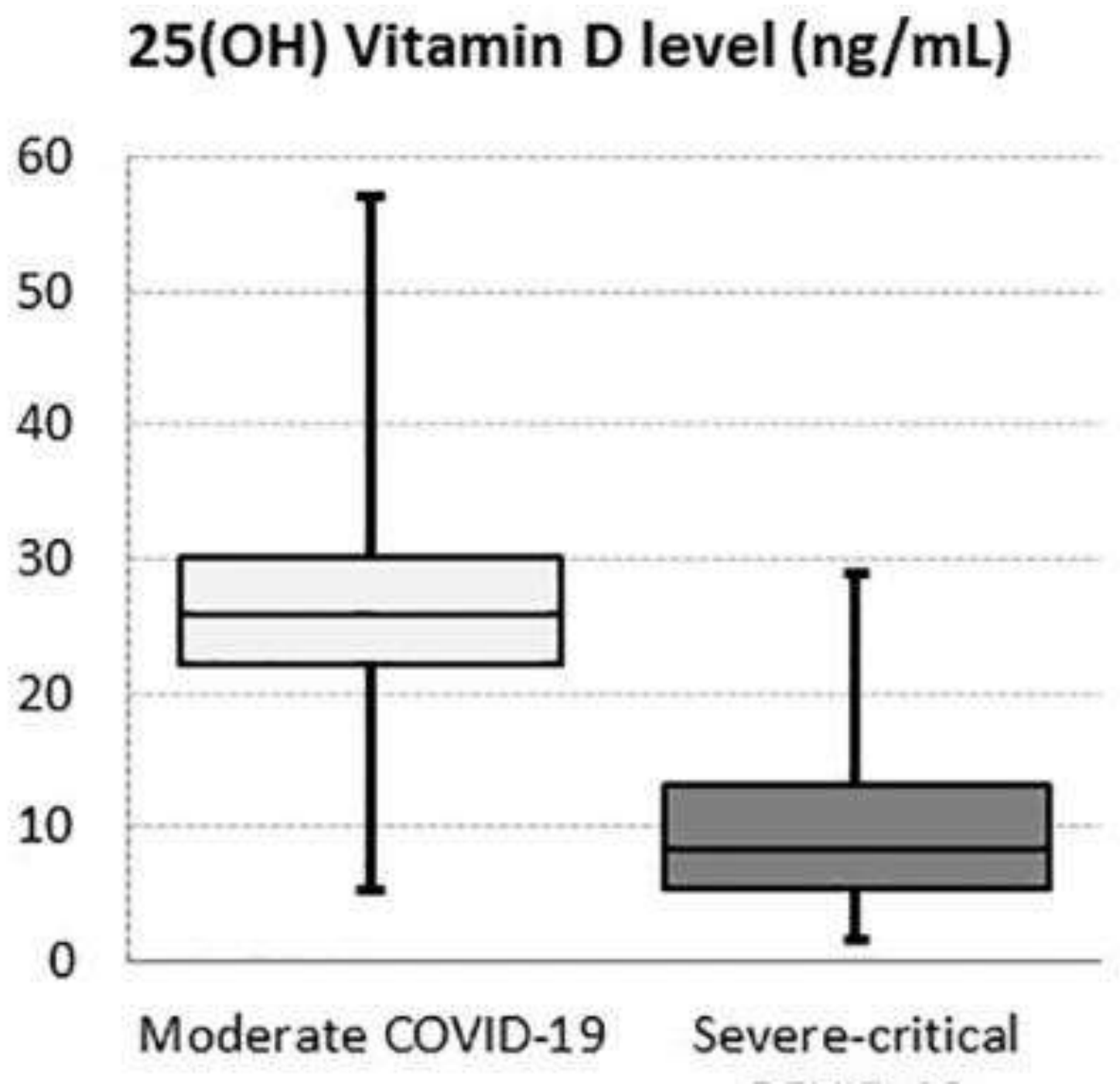
Received: 7 June 2020 / Accepted: 18 July 2020

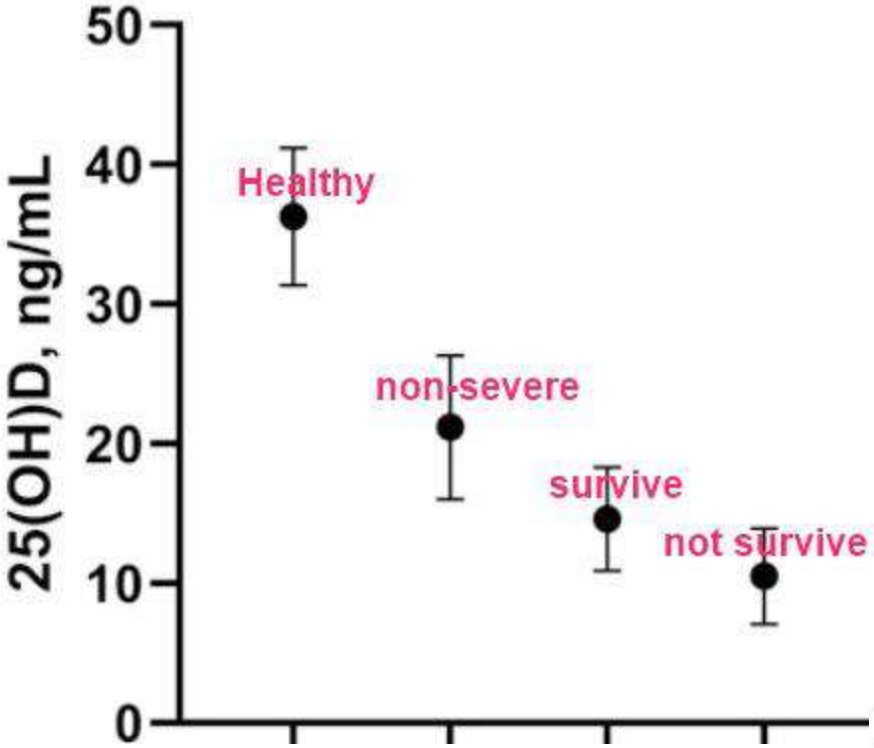
Vitamin D COVID-19 treatment studies with exclusions

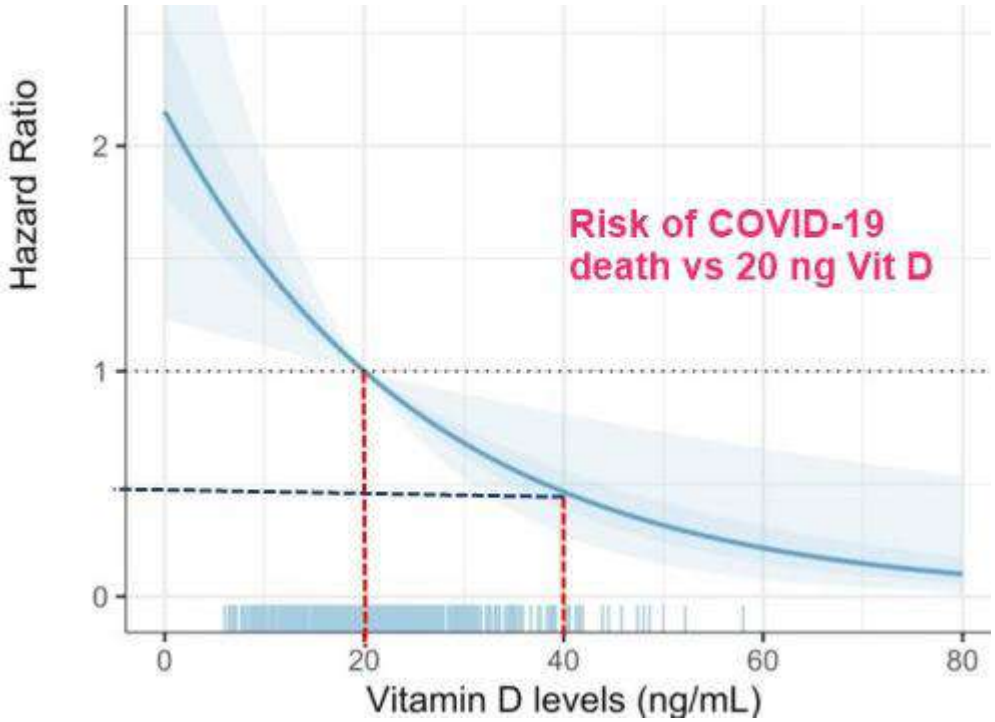
vdmeta.com 2/7/21

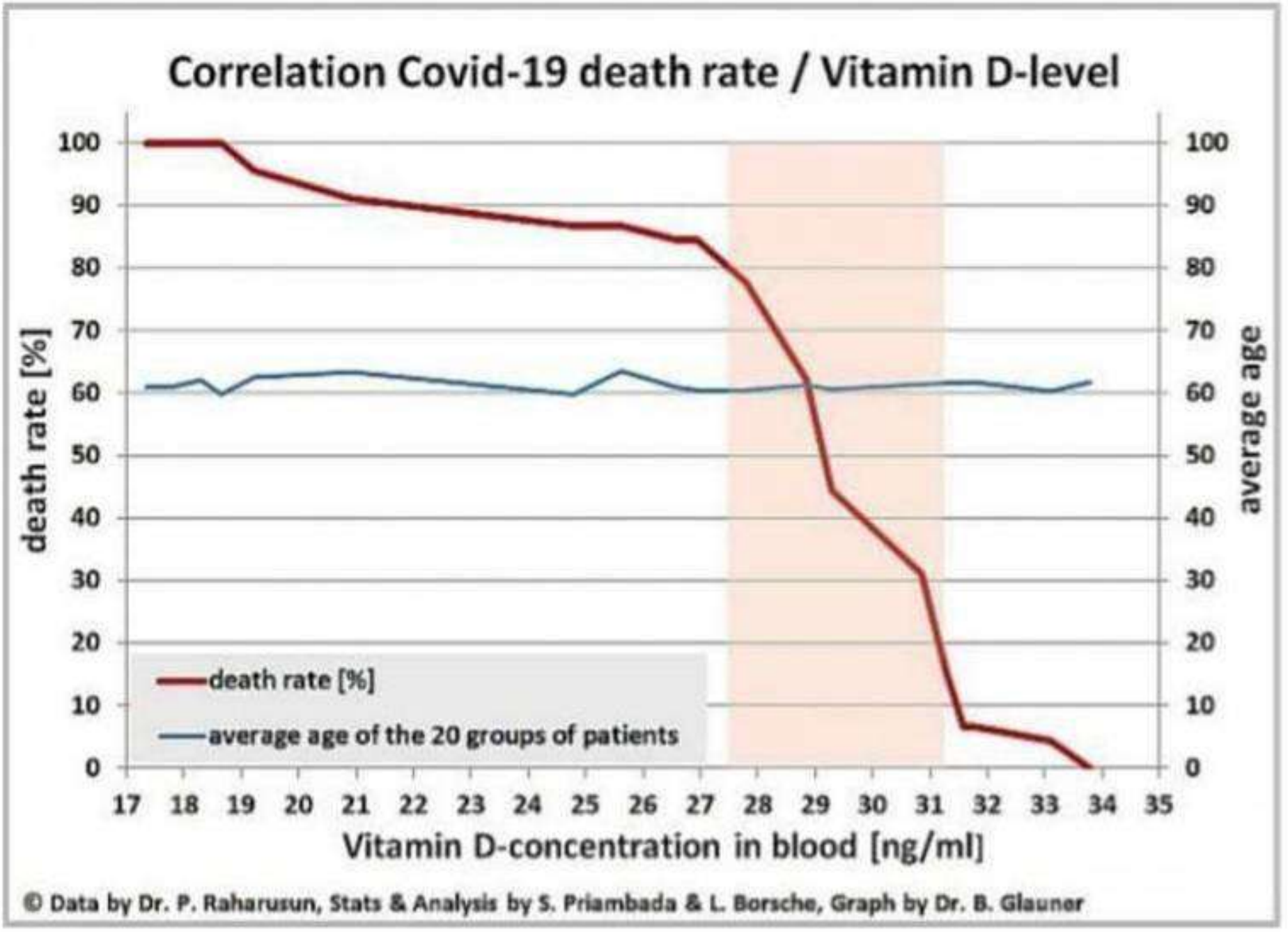


Tau² = 0.40; I² = 86.6%; Z = 5.25 (p < 0.0001)

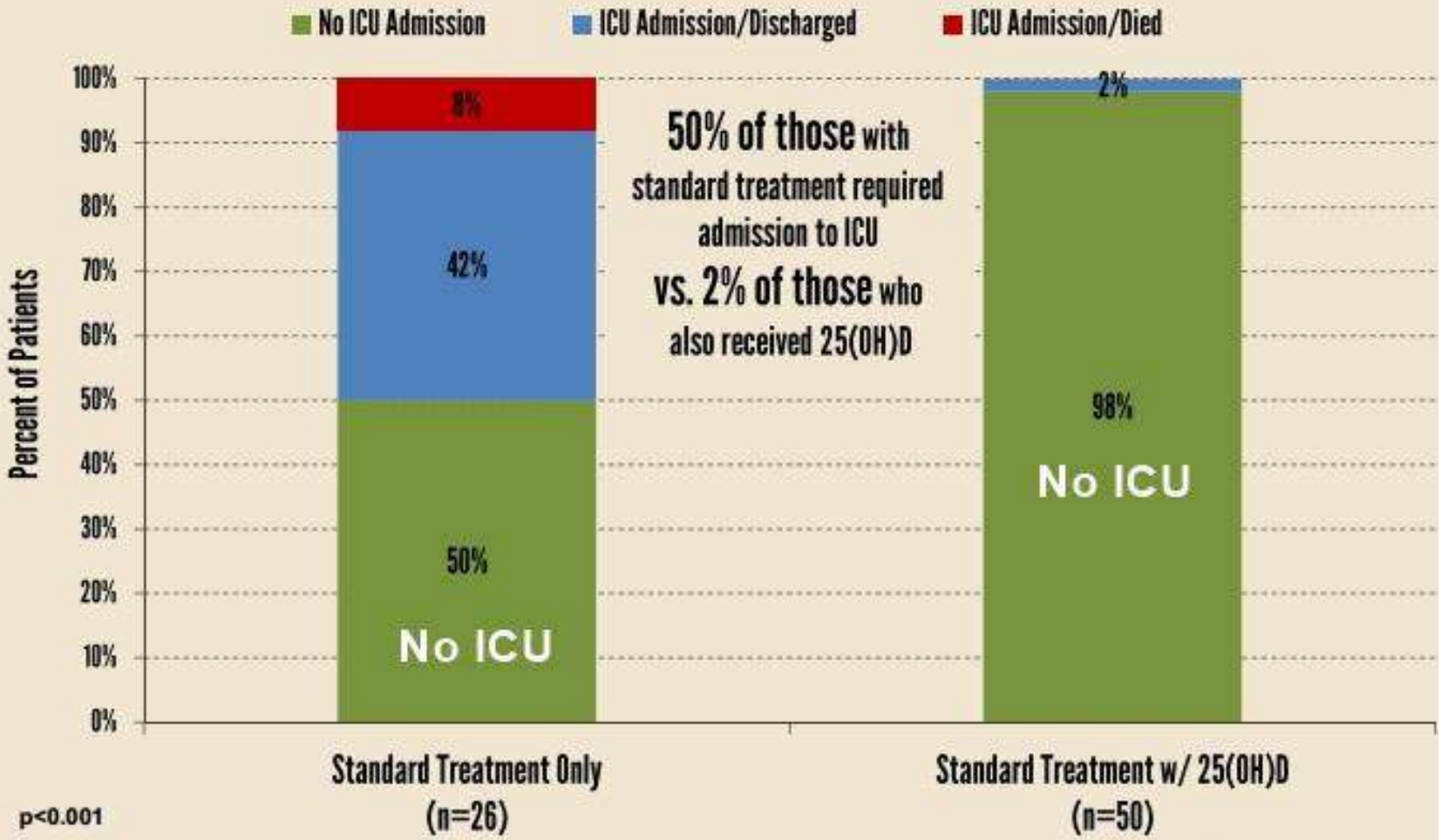

25(OH) Vitamin D level (ng/mL)
60
50
40
30
20
10
0
Moderate COVID-19
Severe-critical


25(OH)D, ng/mL
50
40
30
20
10
0
Healthy
non-severe
survive
not survive


Hazard Ratio
2
1
0
Risk of COVID-19
death vs 20 ng Vit D
0
20
40
60
80
Vitamin D levels (ng/mL)


Correlation Covid-19 death rate / Vitamin D-level
death rate [%]
average age
100
90
80
70
60
50
40
30
20
10
0
death rate [%]
average age of the 20 groups of patients
17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35
Vitamin D-concentration in blood [ng/ml]
© Data by Dr. P. Raharusun, Stats & Analysis by S. Priambada & L. Borsche, Graph by Dr. B. Glauner

Intensive Care Unit Admission and Death Among Hospitalized COVID-19 Patients



p<0.001

Chart Date 9/2/2020

Castillo et al., *Journal of Steroid Biochemistry and Molecular Biology*, 2020.

GrassrootsHealth Nutrient Research Institute | Moving Research Into Practice

www.grassrootshealth.net

	COVID-19 Trials using Vitamin D			VitaminDWiki					https://is.gd/COVIDMedia	Jan 5, 2021
	Country - City	IU	Dose	Place	# people	Placebo	Treat	Age	Completion	Comments, results highlighted	Trial ID
38	India - Mumbai	180,000	Vit D & 45 mg Zinc daily	Hospital	700	yes	Treat	>18	Jan 2022	Far too late	NCT04641195
37	Belgium - Liege	100,000	total in 5 days, then maint. dose	Hospital	100	yes	Treat	>18	Feb 2021	might be enough	NCT04636086
36	UK - Manchester	300,000	none, maint, loading	3 hospitals	986	no	Obs.	senior	Oct 2020	2X fewer deaths	NCT04386044
35	Spain -	21,000	only if <30 ng	Hospital	108	yes	Treat	all	Dec 2020	far too litte	NCT04621058
34	UK -London	no loading	3,200 IU daily	Home	5,440	800 IU	Treat	>16	Jul 2021	far too little, too late Martineau	NCT04579640
33	Jordan	no loading	weekly 50,000 IU (8 weeks)	Hospital	100		Treat		Dec 2020		NCT04476745
32	Spain, Oviedo	100,000	Given when diagnosed	Hospital	80		Treat	>18	Jan 2021	too few, too late	NCT04552951
31	Mexico- Mexico	4,000	for 30 days to workers	Hospital	400	yes	Prevent		Jul 2021	far too little	NCT04535791
30	Switzerland - Basel	140,000		Hospital	80	yes	Treat		Jul 2021	too little, too late, too few	NCT04525820
29	UK - London	-	Observe, no intervention	Hospital	27,000	-	Obs.	1-100	Dec 2020	a little late, should be interesting	NCT04519034
28	Iran - Mashhad	50,000	weekly	Hospital	210		Treat	30-60	Jan 2021	too little, too late	IRCT20110726007117N11
27	US - Columbus	100,000	Load + Resveratrol 4X daily	Home	200	yes	Treat	>45	Nov 2020	Res. gets more D into cells	NCT04400890
26	US - Cleveland	200,000	Load + 200K in 2 week	Home	110	yes	Treat		Aug 2020	monitor at home	NCT04489628
25	Mexico - Mecico City	2,000	daily (1,000 IU if <1 year)	Hospital	40	no	Treat	0-17	April 2021	too late, too little, too few	NCT04502667
24	Canada - Montreal	100,000	Load +10,000 .week (too little)	Hosp - staff	2,414	yes	Prevent	18-69	April 2021	too late	NCT04483635
23	Iran - Sabzevar	350,000	50,000 daily for a week	Hospital	30	no	Treat	15+	?		
22	Iran - Shahroud	1,200,000	Injection, repeated in 1 week	Hospital	100	no	Treat		?	injection has slow response	IRCT20200411047024N1
21	Iran - Mashhad	50,000	then 10,000 IU daily	Hospital	70	no	Treat	30-60	?		
20	Iran - Tehran	600,000		ICU	60	no	ICU	20-60	Aug?	1 week	46838
19	India - Chandigarh	420,000	60,000 daily nano for a week	Home?	30	yes	Treat	18+	Jul 2020	no longer test positive 2X faster	NCT04459247
18	Brazil - Sao Paulo	200,000	once	Hospital	200	yes	Treat	18+	Nov 2020	NOT help if wait 10 days	NCT04449718
17	Spain - Cordoba	100,000	532 ug, + 532 ug in first week **Calcidiol**	Hospital	1,008	yes	Treat	18-90	Aug 2020	pre-trial for 50. Aug 29 10.1016/j.jsbmb.2020.10575	NCT04366908
16	Iran - Tehran	~5,000	25 ug Calcidiol daily	Home	1,500	yes	Prevent	18-75	March 2021	too late; too little, Holick	NCT04386850
14	Iran - Tehran	1,000	daily 2 months	Hospital	1,500		Treat	adult	Mar 2021	too late*, too little	NCT04386850
13	France - Lille	2,000	daily for 2 mon. +2 Zinc daily	Nurse Home	3,140	no	Treat	>59	?	too little, Test positive?	NCT04351490
12	Argentina - Mendoza	500,000	5 capsules of 100,000	Hospital	200	yes	Treat	>45	Dec 2020	too late*	NCT04411446
11	US - New Orleans	50,000	weekly, +daily aspirin	Hospital	1,080	Aspirin	Treat	adult	Dec 2020	too little,too late	NCT04363840
9	Canada - Aberta	100,000	50K twice in first week	Hospital	64	1,000	Treat	adult	Dec 2020	too late*	NCT04385940
8	US - Arizona	10,000	daily, but 5,000 when > 30 ng	Hospital	100	No	Treat	adult	May 2021	too little, too late*	NCT04407286
7	US - Boston	19,800	plus maint = 3,200 IU daily	Home	2,700	Yes	Treat	30-50	Jan 2021	Manson, too little, too late	NCT04536298
5	France - Angers	400,000	once	Hospital	260	50,000	Treat	>70	May 2021	too late, Annweiler	NCT04344041
4	Spain	25,000	once	Hospital	200	?	Treat	40-70	July 2020	too little	NCT04334005
3	US - South Carolina	60,000	over 3 days, then 6,000 daily	home	140	yes	Prevent	>50	Dec 2021	Hollis, too few, too late	NCT04482673
2	UK - Leeds	1,000	daily - blood test every 3 wk	College	4,400	Yes	Prevent	18-30	May 2021	too little, too late*	NCT04476680

Short term, high-dose vitamin D supplementation for COVID-19 disease: a randomised, placebo-controlled, study (SHADE study)

Ashu Rastogi,[1] Anil Bhansali,[1] Niranjan Khare,[2] Vikas Suri,[2] Narayana Yaddanapudi,[3] Naresh Sachdeva,[1] G D Puri,[3] Pankaj Malhotra [2]

ABSTRACT

Background Vitamin D has an immunomodulatory role but the effect of therapeutic vitamin D supplementation in SARS-CoV-2 infection is not known.

Aim Effect of high dose, oral cholecalciferol supplementation on SARS-CoV-2 viral clearance.

Design Randomised, placebo-controlled.

Participants Asymptomatic or mildly symptomatic SARS-CoV-2 RNA positive vitamin D deficient (25(OH) D<20 ng/ml) individuals.

Intervention Participants were randomised to receive daily 60 000 IU of cholecalciferol (oral nano-liquid droplets) for 7 days with therapeutic target 25(OH) D>50 ng/ml (intervention group) or placebo (control group). Patients requiring invasive ventilation or with significant comorbidities were excluded. 25(OH)D levels were assessed at day 7, and cholecalciferol

millions of individuals globally and severely

significantly decreased with cholecalciferol supplementation (intergroup difference 0.70 ng/ml; P=0.007) unlike other inflammatory biomarkers.

Conclusion Greater proportion of vitamin D-deficient individuals with SARS-CoV-2 infection turned SARS-CoV-2 RNA negative with a significant decrease in fibrinogen on high-dose cholecalciferol supplementation.

Trial register number NCT04459247.

Check for updates

To cite: Rastogi A, Bhansali A, Khare N, *et al*. *Postgrad Med J* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/postgradmedj-2020-139065

INTRODUCTION

Coronavirus-2019 (COVID-19) caused by severe acute respiratory syndrome-associated coronavirus-2 (SARS-CoV-2) has affected the lives of

Impact of Pulse D Therapy on The Inflammatory Markers in Patients With COVID-19.

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D is a known
ulse D therapy in reducing

ere evaluated for

inflammatory markers (N/L ratio, CRP, LDH, IL6, Ferritin) along with vitamin D on 0^{th} day and 9^{th} / 11^{th} day as per their respective BMI category. Subjects were randomised into VD and NVD groups. VD group received Pulse D therapy (targeted daily supplementation of 60,000 IUs of vitamin D for 8 or 10 days depending upon their BMI) in addition to the standard treatment. NVD group received standard treatment alone. Differences in the variables between the two groups were analysed for statistical significance.

Results: Eighty seven out of one hundred and thirty subjects have completed the study (VD:44, NVD:43). Vitamin D level has increased from 15.65 ± 5.54 ng/ml to 88.96 ± 31.55 ng/ml after Pulse D therapy in VD group and highly significant ($p<0.01$) reduction of all the measured inflammatory markers was noted. Reduction of markers in NVD group was insignificant ($p>0.05$) . The difference in the reduction of markers between the groups (NVD vs VD) was highly significant ($p<0.01$).

Conclusions: Therapeutic improvement in vitamin D to 80-100 ng/ml has significantly reduced the inflammatory markers associated with COVID-19 without any side effects. Hence, adjunctive Pulse D therapy can be added safely to the existing treatment protocols of COVID-19 for improved outcomes.

Variable	Pre (n=44)		Post (n=44)		Pre vs Post	
	Mean ± SD or Median (IQR)	95% CI of Mean / Median	Mean ± SD or Median (IQR)	95% CI of Mean / Median	t or z statistic	p value
Vit.D (ng/ml)	15.65 ± 5.54#	13.96-17.33*	88.96 ± 31.55#	79.40-98.52*	15.53	<0.0001
CRP (mg/L)	81.31 ± 66.38#	61.13-101.49*	16.48 ± 41.99#	3.72-29.26*	-5.98	<0.0001
LDH (U/L)	369.46 ± 159.34#	321.02-417.91*	274.4 ± 114.8#	239.50-309.30*	-4.58	<0.0001
IL6 (pg/ml)	15.2 (5.30-56.65)	8.95-28.62	2.95 (0.90-7.55)	1.70-4.79	4.29	<0.0001
Ferritin (ng/ml)	430.65 (189.9-835.7)	261.76-708.01	333.95 (153.8-508.0)	202.77-432.69	3.52	0.0004
N/L Ratio	5.49 (3.08-10.99)	4.14-7.72	3.32 (2.35-5.26)	2.69-4.78	3.66	0.0003

TABLE 2. PREDICTORS ASSOCIATED WITH DEATH FROM COVID-19, UNIVARIATE ANALYSIS.

	OR (95% CI)	*p-value (unadjusted)*	*OR_{adj} (95% CI)*	*p-value (adjusted)*	*n*
Age >74 years	2·84 (2·13-3·79)	$1{\cdot}51 \times 10^{-12}$	2·88 (2·16-3·85)	$9{\cdot}43 \times 10^{-13}$	935
Treatment with vitamin D	0·59 (0·43-0·80)	0·001	0·48 (0·35-0·67)	$1{\cdot}36 \times 10^{-5}$	904
High-flow O_2	3·88 (2·82-5·34)	$9{\cdot}7 \times 10^{-17}$	5·96 (4·10-8·66)	$7{\cdot}32 \times 10^{-21}$	927
Asthma	0·29 (0·16-0·50)	1·3E-05	0·41 (0·23-0·74)	0·003	940
IHD	2·63 (1·85-3·74)	8·28E-08	1·90 (1·31-2·75)	0·001	940
Vitamin D booster therapy	0·49 (0·29-0·84)	0·010	0·46 (0·26-0·81)	0·006	338
Vitamin D maintenance therapy	2·02 (1·18-3·44)	0·010	2·16 (1·24-3·77)	0·006	338
Admission SpO_2 <96%	1·62 (1·21-2·18)	0·001	1·52 (1·11-2·09)	0·009	846
CRP >77·5 mg/L	1·67 (1·26-2·21)	$3{\cdot}84 \times 10^{-4}$	1·72 (1·27-2·33)	4×10^{-4}	914
Creatinine >83 µmol/L	2·42 (1·82-3·22)	$1{\cdot}38 \times 10^{-9}$	1·76 (1·28-2·41)	$4{\cdot}6 \times 10^{-4}$	928
Glucose >6·8 mmol/L	1·36 (1·02-1·82)	0·035	1·39 (1·02-1·89)	0·035	848

2X fewer deaths

2X more deaths

Vitamin D Toxicity

"WORRYING ABOUT VITAMIN D TOXICITY IS LIKE WORRYING ABOUT DROWNING WHEN YOU'RE DYING OF THIRST."

-DR. JOHN CANNELL, VITAMIN D RESEARCHER

JOURNAL OF BONE AND MINERAL RESEARCH
Volume 22, Supplement 2, 2007
doi: 10.1359/JBMR.07S221

Hypercalcemia is the hazard criterion for vitamin D

Vitamin D Toxicity, Policy, and Science

Reinhold Vieth

ABSTRACT: The serum 25-hydroxyvitamin D [25(OH)D] concentration that is the threshold for vitamin D toxicity has not been established. Hypercalcemia is the hazard criterion for vitamin D. Past policy of the Institute of Medicine has set the tolerable upper intake level (UL) for vitamin D at 50 μg (2000 IU)/d, defining this as "the highest level of daily nutrient intake that is likely to pose no risks of adverse health effects to almost all individuals in the general population." However, because sunshine can provide an adult with vitamin D in an amount equivalent to daily oral consumption of 250 μg (10,000 IU)/d, this is intuitively a safe dose. The incremental consumption of 1 μg (40 IU)/day of vitamin D_3 raises serum 25(OH)D by ~1 nM (0.4 ng/ml). Therefore, if sun-deprived adults are to maintain serum 25(OH)D concentrations >75 nM (30 ng/ml), they will require an intake of more than the UL for vitamin D. The mechanisms that limit vitamin D safety are the capacity of circulating vitamin D–binding protein and the ability to suppress 25(OH)D-1-α-hydroxylase. Vitamin D causes hypercalcemia when the "free" concentration of 1,25-dihydroxyvitamin D is

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MAYO CLINIC
PROCEEDINGS

Vitamin D Is Not as Toxic as Was Once Thought: A Historical and an Up-to-Date Perspective

In the current issue of *Mayo Clinic Proceedings* rheumatoid arthritis and massive doses of

See also page 577

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D (25(OH)
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history of

There is enough evidence that vitamin D toxicity is one of the rarest medical conditions and is typically due to intentional or inadvertent intake of extremely high doses of vitamin D (usually in the range of >50,000-100,000 IU/d for months to years) without monitoring for hypercalcemia

Facts and Rationale 2021

- Vitamin D deficiency is a global pandemic
- Vitamin D is a powerful immuno-modulator
- When checked first time without any supplementation, very low vitamin D levels found in 90% (3 – 20 ng/ml)
- Nobody has been found with the minimal desired level 40 ng/ml unless taking Vit D supplements
- We lose nothing by improving the global vitamin D status
- <u>Correcting a deficiency is our duty, responsibility & obligation as a doctor</u>

Dr. Renu Mahtani's AUTOIMMUNITY TREATMENT CENTRE

Points to Consider….

- It is advisable to maintain serum 25-hydroxy vitamin D in the range of **40 – 60 ng/mL** to minimize the risk of COVID-19 infection and its severity.
- We must **check the vitamin D (25 OH D3) blood levels in Covid 19 positive patients** (hospitalised and those advised tests for inflammatory markers)
- We should not hesitate in giving **bolus dose** to immediately boost up the levels
- **If not now, then when????**

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