POTENTIAL MECHANISMS OF ACTION OF VITAMIN D AFFECTING SARS-COV-2

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INTRODUCTION

Since the outbreak of the pandemic, vit D has been in focus as a potential molecule for prevention and treatment of COVID-19.

METHOD/DESIGN

- PubMed and Google Scholar (2020 and 2021)
- ClinicalTrials.gov: “COVID-19” and “vitamin D” (October 13, 2021)

OBJECTIVES

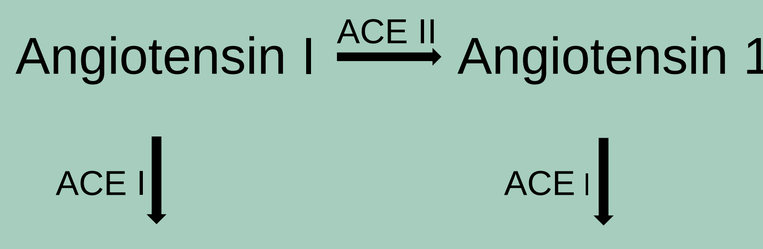
- present the potential mechanisms of action of vit D that were reported as potentially beneficial against SARS-CoV-2
- identify ongoing clinical trials regarding COVID-19 and vit D

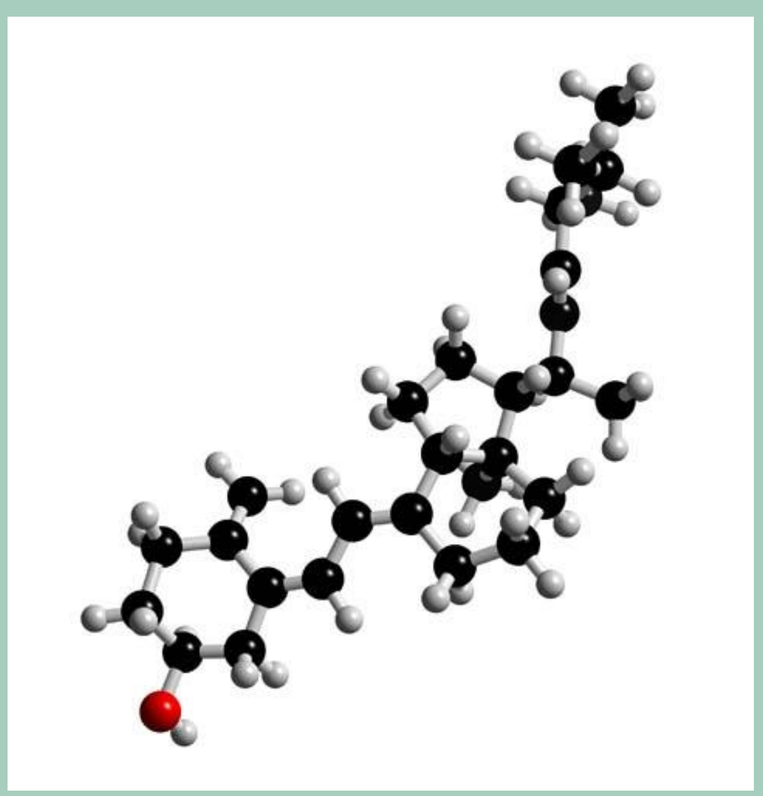
RESULTS

Vit D ⬆ cathelicidin and β-defensin 2 (antimicrobial and antiviral peptides)

Vit D ⬇ IL-6, IL-8, IL-12, TNF, INF-γ and IL-17

- COVID-19 infection down-regulates ACE2, which in turn could lead to excessive accumulation of angiotensin II
- vit D induces expression of ACE2 in the lungs in experimental animals in specific experimental conditions. ACE2 thus expressed more as a consequence of vit D supplementation might reduce lung injury
- although vit D increases the expression of ACE2, which indeed promotes the binding of the virus, it prevents pulmonary vasoconstriction response in COVID-19 cases
- ACE2 has potentially contradictory roles. On one hand, greater expression of it would be assumed as a potential risk since it is the receptor for SARS-CoV-2, but on the other, it has an important protecting role against acute lung injury and ARDS in experimental models





Vitamin D molecule, source: www.3dchem.com

Mentioned mechanisms of action are still an area of research and need to be clinically confirmed.

110 clinical trials regarding vit D and COVID-19 were registered in the ClinicalTrials.gov registry

Status	Type
72 active/not yet started 32 completed	78 interventional 32 observational

CONCLUSION

As the exact molecular mechanisms are being clarified, new clinical trials are designed in order to confirm the link between vit D deficiency and COVID-19 severity and the potential benefits of vit D administration for prevention and treatment of COVID-19.