VITAMIN D
UpDates

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Managing Editor
Patrizia Pacini

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Via Gherardesca 1 • 56121 Pisa
Tel. 050 313011 • Fax 050 3130300
Info@pacinieditore.it
www.pacinieditore.it

Editorial Coordinator
Lucia Castelli
Tel. 050 3130224
lcastelli@pacinieditore.it

Graphics and Layout
Massimo Arcidiacono
Tel. 050 3130231
marcidiacono@pacinieditore.it

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Editorial

Maurizio Rossini
Department of Medicine,
Section of Rheumatology, University of Verona

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Vitamin D is still surprising. Notice how in this issue we go from a possible role for vitamin D in psychiatric disorders to application to Oncology. We know that vitamin D can have pleiotropic effects, but what might the common biological mechanism that determines them be? Could it be the ubiquitous nature of its receptors? Perhaps its effects on the immune system? Maybe its ability to modulate certain enzyme activities? Or its genomic effects?
It will be interesting to see what you think in view of the authors' contributions in this issue.
The stated aim of the article on vitamin D and psychiatric disorders is to identify its causal relationship, because this would make it possible to understand whether, and to what extent, vitamin D supplementation could prevent the onset of mental disorders or reduce their symptoms. Initially, we begin by describing vitamin D's possible mechanisms of action at a neurological level, before describing the current findings from observational or interventional studies in this field. Specifically, it is noted that vitamin D is involved in the region-specific expression of vitamin D receptors (VDR) in areas such as the cingulate cortex, thalamus, cerebellum, substantia nigra, amygdala and in the hippocampus. Most of these regions express 1α-hydroxylase enzymes capable of metabolising 25(OH)D in $1,25(OH)_2D_3$. This means that vitamin D also has both an autocrine and a paracrine function in the human brain, both of which could play a relevant role in neuroimmune modulation or protection and normal brain development and function. Given the involvement of vitamin D in the control of the inflammatory response, in the case of depression, it is hypothesised that it acts as a modulating mechanism by regulating the over-expression of pro-inflammatory cytokines associated with depression. However, it has also been pointed out that vitamin D is involved in regulating the activity of enzymes such as tyrosine-hydroxylase and the rate-limiting enzyme in the biosynthesis of dopamine, norepinephrine and epinephrine, which are all mechanisms that may justify a positive association between vitamin D deficiency and depression.
Our oncologist colleague, who has also followed the same path, describes the possible biological mechanisms of action, before summarising current clinical findings. He points out that a first level of interaction between vitamin D and neoplastic transformation or progression may refer to the local biosynthetic capacity of the enzyme CYP27B1, whose expression is reduced in some tumours depending on the stage and degree of differentiation. In this context, variations in VDR expression at the intra-tumour level may also influence the biological aggressiveness of the neoplasm by modulating the autocrine, paracrine and intracrine action of vitamin D.
Therefore, I would like to point out that only cholecalciferol and not the different vitamin D metabolites can ensure complete physiological local effects at the level of the various organs and tissues. In addition, the vitamin's potential anti-tumour action can also be

Correspondence
Maurizio Rossini
maurizio.rossini@univr.it

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expressed mainly through genomic mechanisms as well as through non-genomic mechanisms.
With the usual caution that characterises leading experts' assertions, including our authors, the conclusions regarding the clinical effects of vitamin D supplementation in psychiatry and oncology are similar. The results are still conflicting, probably also due to the multifactorial nature of the pathogenesis, the different evaluations of the outcomes (e.g., impact on the incidence of cancer, compared with the more convincing impact on cancer mortality) and the uncertainties about dosages.

However, to me, it already seems advisable to also include the assessment of vitamin D levels in the diagnostic pathway of these diseases as well as to avoid vitamin deficiency among these patients.

Happy reading!