# VITAMIN D

UpDates Vol. 6 - N. 4- 2023

Sito Web www.vitamind-journal.it



www.pacinimedicina.it

ISSN: 2611-2876 (online)

Registrazione presso il Tribunale di Pisa n. 2/18 del 23-2-2018


# EDITORIALE

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VITAMIN D
UpDates
2023;6(4):130-131

Cari Colleghi
il tessuto osseo e quello muscolare scheletrico sono intimamente connessi tra loro da un punto di vista biomeccanico ed è stato ipotizzato che la vitamina D possa essere considerata una molecola "regista" del *cross-talk* intertissutale che governa l'efficienza strutturale e funzionale dell'apparato muscolo-scheletrico.
In questo numero troverete un *update* sul rapporto tra ipovitaminosi D e l'osteosarcopenia e in particolare sui meccanismi attraverso i quali la vitamina D sembra condizionare la forza muscolare. Anche il muscolo è dotato di recettori per la vitamina D ed è stato osservato, in studi condotti su animali, che la loro delezione comporta sarcopenia e deficit di funzione muscolare [1]. È peraltro noto da tempo che una condizione di grave carenza di vitamina D può manifestarsi con una grave debolezza muscolare, specie a livello dei muscoli prossimali, e quindi con un aumentato rischio di cadute. Tuttavia, è ancora dibattuto se la supplementazione con vitamina D negli adulti viventi in comunità determini un aumento della forza muscolare e riduca il rischio di cadute o se addirittura un eccesso di supplementazione possa peggiorare questi *outcome*. Una metanalisi del 2014 concludeva che la supplementazione con vitamina D migliorava la forza muscolare [2], ma ciò non è stato confermato da due metanalisi più recenti [3,4]. Due recenti metanalisi, incluse nella selezione bibliografica di questo numero, non hanno trovato una significativa riduzione del rischio di cadute e di fratture con la supplementazione di vitamina D [5,6]. Due precedenti metanalisi avevano dimostrato una significativa riduzione del rischio di cadute in soggetti carenti, ma non, comprensibilmente, in soggetti non carenti [7,8]. Due recenti RCTs [9,10], tra cui il VITAL, non hanno osservato un effetto sul rischio di cadute, ma i partecipanti erano in gran parte repleti di vitamina D! D'altra parte, come ricorderete, la somministrazione di un bolo di 500.000 UI di vitamina D, peraltro in gran parte in soggetti non carenti, era stato visto associarsi a un aumento del rischio di cadute dopo 3 mesi [11]. Anche in altri due studi è stato osservato un aumento del rischio di cadute in chi raggiungeva elevati livelli sierici di 25(OH)D [12,13] e nello Stop-it trial, è stata osservata una "curva a U" nella relazione tra livelli sierici di 25(OH)D e rischio di cadute, indicando una concentrazione ottimale tra i 20 e i 40 ng/ml [14]. Probabilmente le diversità nello stato vitaminico D e nel profilo clinico (ad es. performance muscolare, *body mass index* e comorbilità) dei soggetti trattati, la variabilità degli schemi di trattamento e la mancanza di *endpoints* primari ben definiti giustificano la discordanza nei risultati e così purtroppo generano incertezze e confusione.
L'altro articolo di questo numero è dedicato al possibile ruolo antinfiammatorio della vitamina D. La regolazione dell'infiammazione e dell'espressione delle citochine è di cruciale importanza non solo per le molteplici patologie infiammatorie ma anche in considerazione della recente ipotesi dell'"inflammaging": con l'aumentare dell'età si verificherebbe, infatti, lo spostamento verso uno stato proinfiammatorio che creerebbe e manterrebbe un'infiammazione cronica di base, cui conseguono danni d'organo e la progressione verso varie malattie croniche tipiche dell'invecchiamento (ad es. reumatologiche, metaboliche, cardiovascolari e tumorali). Ebbene

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**How to cite this article:** Rossini M. Editoriale. Vitamin D - UpDates 2023;6(4):130-131.

recentemente è stato osservato che anziani con deficit di vitamina D hanno più elevati livelli ematici di proteina C reattiva [15]. Pochi e talora discordanti sono gli studi che hanno valutato l'effetto della somministrazione di colecalciferolo, in particolare in soggetti carenti, sullo stato infiammatorio e spesso sono presenti bias che ne limitano l'interpretazione, specie in condizioni patologiche. In un gruppo di soggetti giovani e sani ma carenti di vitamina D abbiamo recentemente osservato che la supplementazione con colecalciferolo determina una progressiva riduzione dei livelli di IL-6 e IL-17 [16], due citochine chiave nella patogenesi rispettivamente dell'artrite reumatoide e delle spondiloartriti. La carenza di vitamina D potrebbe quindi accelerare l'*inflammaging* e aumentare il rischio, la progressione o ridurre la risposta al trattamento di patologie infiammatorie.
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# Ipovitaminosi D e osteosarcopenia

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VITAMIN D
UpDates
2023;6(4):132-135
https://doi. org/10.30455/2611-2876-2023-7

## INTRODUZIONE

L'apparato muscolo-scheletrico può essere considerato uno dei prodotti anatomo-funzionali più vantaggiosi comparsi nel corso della storia evolutiva del mondo animale. Tale complesso sistema vede l'interazione di più organi e tessuti, per lo più della stessa derivazione embriogenetica, che integrano diverse funzioni vitali, che vanno oltre lo scopo primario della locomozione, in un unico "organo" che si sviluppa nel periodo della crescita, e si modella e rimodella durante tutta la vita della persona [1,2]. Almeno tre tessuti fondamentali sono coinvolti in questa interazione anatomo-funzionale: il tessuto osseo, il tessuto muscolare striato e il tessuto adiposo. I tre tessuti hanno una stessa derivazione embriologica, sviluppandosi dallo strato germinale mesodermico, che può essere suddiviso in tre regioni fondamentali: mesoderma parassiale, intermedio e laterale. La somitogenesi è un passaggio fondamentale che avviene nel mesoderma parassiale dove le cellule si dividono in somiti. Ogni somite contiene specifici precursori per lo sviluppo dello scheletro assiale (sclerotomo), dei tendini (sindotomo), dei muscoli scheletrici (miotomo) e del derma (dermatomo) [3]. Lo sclerotomo si sviluppa in precartilagine, poi in cartilagine che infine va incontro a ossificazione. I precursori derivati dal mesoderma parassiale che vireranno verso la miogenesi sono sotto il controllo di Pax3/7 (*Paired Box 3/7*), seguito dall'attivazione della differenziazione e della fusione nei sincizi multinucleati, cioè miotubi, guidati dall'espressione di fattori miogenici, come Myf5 (*Myogenic Factor 5*) e MyoD (*Myogenic Differentiation*).

La fusione dei miotubi dà origine alle fibre muscolari, che poi si raggruppano in fasci e i fasci si uniscono per formare il muscolo. Alcune di queste cellule, le cosiddette "cellule satellite", sono precursori muscolari Pax7$^+$ che si localizzano sotto la lamina basale delle fibre muscolari in uno stato latente e agiscono come fonte di mionuclei durante la crescita postnatale e dopo le lesioni muscolari [4].

Il tessuto osseo e quello muscolare scheletrico sono intimamente connessi tra loro da un punto di vista biomeccanico. Mentre le ossa svolgono un ruolo di supporto, i muscoli consentono l'attività motoria attraverso l'interazione delle proteine contrattili all'interno dei sarcomeri e grazie alla loro inserzione attraverso i tendini sulle strutture scheletriche; entrambi i tessuti, inoltre, regolano il metabolismo energetico attraverso la produzione e il rilascio di varie molecole, in particolare citochine. Molecole prodotte dal tessuto osseo e immesse in circolo per svolgere attività biologica locale o a distanza vengono dette "osteochine" e tra di esse Wnt, sclerostina, RANK-L (*Receptor Activator of Nuclear Kappa B Ligand*), osteocalcina, FGF-23 (*Fibroblast Growth Factor-23*), BMP (*Bone Morphogenetic Protein*), PGE-2 (*Prostaglandin E2*), IGF-1 (*Insuline like Growth Factor-1*). Tali molecole hanno tutte una o più funzioni modulanti l'attività biologica e funzionale del muscolo. Allo stesso tempo il muscolo produce altre citochine, note come miochine, tra cui irisina, miostatina, diverse interleuchine, e fattori neurotrofici, che agiscono in modo autocrino, paracrino ed endocrino. Il *cross-talk* tra i tessuti componenti l'apparato locomotorio è dovuto proprio alla produzione e immissione in circolo di queste varie sostanze [5].

Una conoscenza approfondita della funzione delle molecole coinvolte in questo complesso sistema tissutale interconnesso è necessaria per identificare strategie terapeutiche utili nella gestione dei disturbi muscoloscheletrici, in particolare dell'osteosarcopenia.

È stato ipotizzato che la vitamina D possa essere considerata una molecola "regista" del *cross-talk* intertissutale che governa l'efficienza strutturale e funzionale dell'apparato muscolo-scheletrico [6] (Fig. 1).

## VITAMINA D E OSTEOSARCOPENIA

È da tempo nota la relazione tra la bassa concentrazione di vitamina D [25(OH)$D_3$] nel sangue e le condizioni patologiche età-correlate, come l'osteoporosi e la sarcopenia, come anche lo stretto rapporto tra ipovitaminosi D e incremento del rischio di cadute, legata alla

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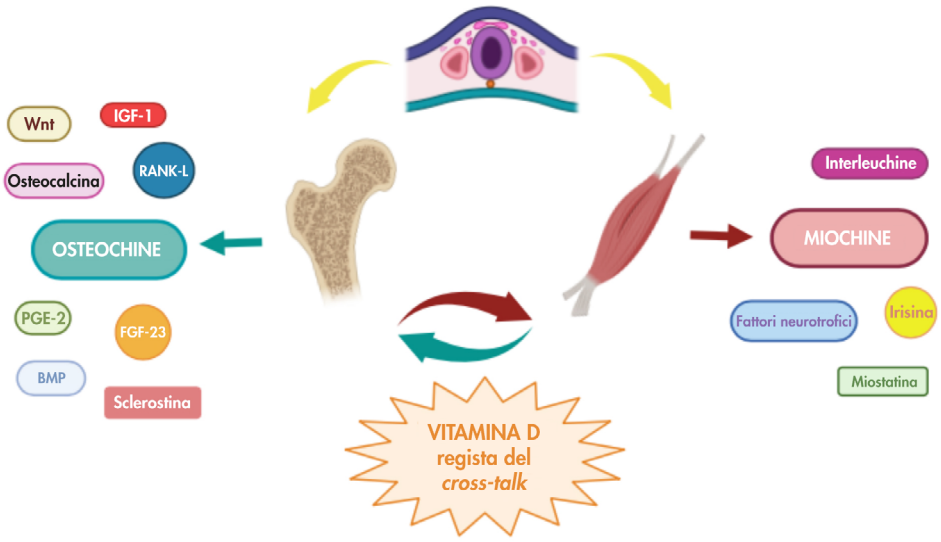
***Conflitto di interessi***
Gli Autori dichiarano di aver ricevuto finanziamenti o hanno in atto contratti o altre forme di finanziamento con Abiogen, Amgen e UCB.

**How to cite this article:** Iolascon G, Moretti A. Ipovitaminosi D e osteosarcopenia. Vitamin D–Updates 2023;6(4):132-135. https://doi.org/10.30455/2611-2876-2023-7

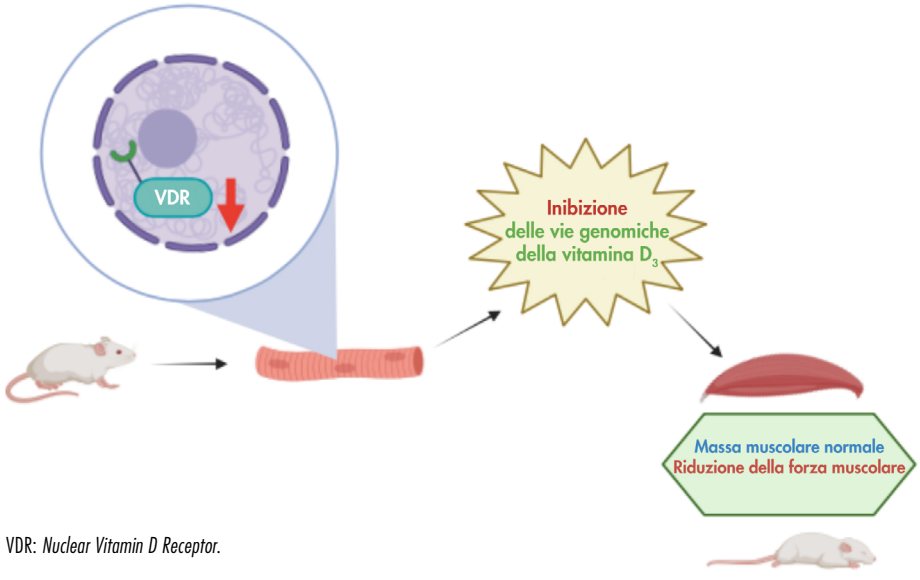



IGF-1: *Insuline like Growth Factor-1*; RANK-L: *Receptor Activator of Nuclear Kappa B Ligand*; PGE-2: *Prostaglandin E2*; FGF-23: *Fibroblast Growth Factor-23*, BMP: *Bone Morphogenetic Protein.*

**FIGURA 1.**
Ruolo della vitamina D nello sviluppo muscolare scheletrico embrionale.

consistente diminuzione della forza muscolare, dovuta principalmente alla deplezione di fibre muscolari di tipo 2 che vengono reclutate soprattutto nelle variazioni posturali [7-9]. La loro diminuzione causa necessariamente un aumento importante del rischio di cadere quando si passa, ad esempio, dalla posizione seduta a quella eretta.
D'altronde il reintegro del livello sierico di 25(OH)$D_3$ in pazienti con accertata ipovitaminosi D, attraverso una supplementazione, può indurre un significativo recupero di forza muscolare che può determinare una riduzione importante del rischio di caduta nel paziente anziano [10].
Come è noto, la vitamina D agisce principalmente con un percorso genomico, che è mediato dal legame con i recettori nucleari della vitamina D (VDR).
In presenza di una significativa diminuzione del livello sierico di vitamina D, frequente se non addirittura costante nel paziente anziano, si verificano effetti negativi sui muscoli, con segni istologici di atrofia muscolare età-correlata, caratterizzati soprattutto da deplezione delle fibre rapide di tipo 2. Evidenze sperimentali mostrano che, in topi maturi con deficit di VDR specifico per le fibre muscolari, l'inibizione della via genomica della vitamina D porta a una debolezza muscolare senza intaccare la massa muscolare [11] (Fig. 2).

Il dato sembra essere confermato da uno studio epidemiologico longitudinale condotto su residenti in comunità, nei quali il livello sierico di 25(OH)$D_3$ non aveva effetti significativi sulla massa muscolare mentre invece era significativamente correlato alla forza muscolare [11]. I suddetti dati, sperimentali ed epidemiologici, porterebbero alla conclusione che l'ipovitaminosi D sulle fibre muscolari mature esplica i suoi effetti negativi principalmente sulla forza muscolare. Si è pertanto ipotizzato che dal momento che bassi livelli sierici di vitamina D sono strettamente correlati alla debolezza muscolare età-correlata, il dosaggio della 25(OH)$D_3$ possa essere considerato un buon predittore di debolezza muscolare e quindi un biomarker di sarcopenia.
È presumibile, inoltre, che l'ipovitaminosi D non agisca principalmente attraverso una deplezione della massa muscolare, ma probabilmente in maggior misura mediante una ridotta funzione contrattile delle singole fibre, un *impairment* nell'attività dell'unità motoria per una minore frequenza di scarica dei motoneuroni, una ridotta velocità di conduzione nervosa, un disaccoppiamento eccitazione-contrazione. Inoltre, un ruolo non secondario nella genesi del deficit di forza potrebbe giocare anche l'incremento del tessuto grasso e fibroso all'interno del muscolo stesso.
Un recente studio su animali ha rivelato che la via genomica regola la forza muscolare modulando l'espressione dell'ATPasi calcio-dipendente [12]. SERCA è una pompa del calcio presente nella membrana del reticolo sarcoplasmatico che concentra il calcio nel lume del reticolo sarcoplasmatico. Tre geni distinti codificano SERCA 1, 2 e 3, che sono noti per produrre più di 10 isoforme. Le isoforme tipiche sono le se-




VDR: *Nuclear Vitamin D Receptor.*

**FIGURA 2.**
Ruolo della vitamina D sulla massa e sulla forza muscolare.

guenti: SERCA1 è l'isoforma del muscolo a contrazione rapida, SERCA2a è l'isoforma del muscolo a contrazione lenta.
Il deficit di VDR riduce l'attività della SR $Ca^{2+}$ ATPasi nelle miofibre mature, che si ipotizza essere indotta dalla ridotta espressione dei geni SERCA. La vitamina D altererebbe quindi la dinamica della contrazione muscolare diminuendo la ricaptazione di $Ca^{2+}$ nel SR, prolungando così la fase di rilassamento della contrazione muscolare. In conclusione, la diminuzione dei livelli sierici di vitamina D porta a una riduzione del segnale VDR nelle miofibre e causa un disaccoppiamento eccitazione-contrazione.
Il percorso non genomico in cui la vitamina D entra direttamente attraverso le caveole presenti sulla membrana cellulare è modulato da un'interazione della molecola con un pool separato di VDR (mVDR) o con un diverso recettore legato alla membrana o intracellulare. Un candidato proposto per tale proteina legata alla membrana che media gli effetti rapidi non genomici della vitamina D è la PDIA3 (proteina disolfuro isomerasi) chiamata anche 1$\alpha$,25D3-MARRS. Questa proteina, che è associata a diverse membrane cellulari, tra cui la membrana plasmatica e il reticolo endoplasmatico, è anche nota per il suo importante ruolo nel ripiegamento delle proteine. È stato riportato che alcuni dei nuovi idrossimetaboliti non classici della vitamina D, formati dal CYP11A1, interagiscono sia con il VDR nucleare che con l'1$\alpha$,25$D_3$-MARRS legato alla membrana [13].
Le interazioni tra la vitamina D e i suddetti recettori di membrana realizzano l'attivazione di una pletora di vie intracellulari di trasduzione del segnale. Si ipotizza che l'azione non genomica della vitamina D attivi una cascata della proteina chinasi attivata dal mitogeno (MAPK), chinasi extracellulare regolata dal segnale (ERK) 1 e 2 per mezzo di diversi effettori intermedi, che si attivano quando la vitamina D si lega al VDR. Il VDR attivato stimola l'afflusso di calcio, che, a sua volta, attiva le vie intracellulari guidate dal calcio, come la proteina chinasi C (PKC). Inoltre, la vitamina D potrebbe attivare i recettori accoppiati alle proteine G (GPCR), che, a loro volta, stimolano diversi percorsi a valle, tra cui la fosfatidilinositolo 3-chinasi (PI3K), l'adenilato ciclasi (AC), la Ras e la fosfolipasi C gamma (PLC$\gamma$). Ciascuno di questi percorsi potrebbe convergere mediante segnali diversi sull'attivazione di ERK-MAPK 1/2, che potrebbe interagire con il classico percorso genomico guidato dal VDR, per modulare l'espressione genica.

## NUOVI TARGET DELLA VITAMINA D NELLA FIBRA MUSCOLARE

La funzione contrattile del muscolo scheletrico è regolata dal calcio citosolico, che viene fornito dal trasporto dal reticolo sarcoplasmatico e alimentato dall'idrolisi dell'ATP prodotto da SERCA. La vitamina D causerebbe un'upregulation dell'espressione di SERCA fornendo calcio ionizzato nel citosol e contribuendo, in tal modo, a mantenere la forza muscolare. Risulta pertanto evidente che la vitamina D agisca nelle cellule muscolari promuovendo il consumo di ATP. Si è inoltre ipotizzato che la vitamina D sovraregoli l'espressione di connessina 43 in modo dose-dipendente, favorendo il rilascio di fosfati inorganici, come il pirofosfato, nelle nicchie della superficie cellulare laddove svolgono un ruolo importante nel metabolismo dell'ATP [14]. I pirofosfati extrascheletrici, infatti, sopprimono la calcificazione ectopica nel tessuto muscolare. La calcificazione ectopica nel muscolo scheletrico è stata osservata in modelli murini che mostravano una funzione muscolare compromessa, come la distrofia muscolare di Duchenne o una lesione muscolare scheletrica focale [15]. La vitamina D avrebbe un'attività di controllo sulla calcificazione dei muscoli scheletrici che risulta essenziale per il mantenimento di una corretta attività locomotoria.

## CONCLUSIONE

Tra le azioni extra-scheletriche della vitamina D, quella sul muscolo striato ha sicuramente un considerevole impatto sulla condizione di salute della persona. Numerose sono le prove scientifiche che confermano l'attività della vitamina D nel favorire lo sviluppo della struttura muscolare durante la vita embrionale e fetale nonché la rigenerazione e la riparazione del muscolo scheletrico durante la vita adulta. Inoltre, la vitamina D ha un ruolo fondamentale nella capacità funzionale della fibra muscolare favorendo la massima efficienza dell'accoppiamento eccitazione/contrazione e nell'opporsi all'impairment strutturale e funzionale del muscolo correlato all'invecchiamento e ad altre condizioni di sarcopenia.

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VITAMIN D
UpDates
2023;6(4):136-139
https://doi. org/10.30455/2611-2876-2023-8

# Effetti della carenza di vitamina D sulle citochine infiammatorie

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Oltre a svolgere un ruolo essenziale nel mantenimento della salute delle ossa, la vitamina D è anche riconosciuta per le sue azioni antibatteriche, antiproliferative, immunomodulatorie e antinfiammatorie [1-2]. In particolare, le funzioni immunomodulatorie sono di crescente interesse scientifico. Sono infatti stati pubblicati negli ultimi anni dati sia clinici che epidemiologici a supporto del legame tra lo stato della vitamina D e l'incidenza e la gravità di condizioni immunocorrelate, come la sclerosi multipla, la psoriasi, il diabete, l'artrite reumatoide, le malattie infiammatorie intestinali e le malattie infettive [1,2]. Se l'associazione tra questi eventi patologici e la carenza di vitamina D è stata largamente dimostrata, non altrettanto si può dire dell'effetto della supplementazione con colecalciferolo sugli stessi fenomeni. A complicare il quadro, gli studi pubblicati sono estremamente eterogenei per popolazione considerata, per livelli basali di 25(OH)D, per entità della supplementazione e per la modalità (quotidiana piuttosto che a boli) con cui è stata somministrata.

L'attenzione circa l'effetto della supplementazione con colecalciferolo sulle cellule immunitarie e sulle citochine infiammatorie è stata sicuramente riaccesa dalla pubblicazione lo scorso anno dello studio VITAL. In questo studio sono stati arruolati 25.571 soggetti, randomizzati all'assunzione per 5 anni di 2.000 UI di colecalciferolo al giorno (con o senza aggiunta di omega-3) rispetto a placebo, dimostrando una riduzione dell'incidenza di malattie autoimmuni, tra cui artrite reumatoide, polimialgia reumatica e psoriasi, del 22% [3].

La regolazione dell'infiammazione e l'espressione delle citochine è di cruciale importanza anche per la recente ipotesi dell'"inflammaging": con l'aumentare dell'età si verificherebbe, infatti, lo spostamento verso uno stato proinfiammatorio che creerebbe e manterrebbe un'infiammazione cronica di basso grado (solo parzialmente rilevabile da biomarcatori sierici quali la proteina C reattiva [PCR]) con un successivo lento accumulo di danno. Questo invecchiamento guidato dall'infiammazione cronica sarebbe la base della progressione verso varie malattie croniche [4]. Questo sarebbe confermato anche da un recente studio su una biobanca anglosassone su 397.737 soggetti, di età compresa tra 37 e 73 anni. La carenza di vitamina D è risultata associata a maggiore mortalità per varie cause anche se non ai classici marcatori sierici di infiammazione. Se questo è valido nella popolazione generale potrebbe tuttavia essere diverso nelle popolazioni di pazienti con elevata infiammazione, come individui con cancro, diabete mellito o malattie cardiovascolari acute, in cui la supplementazione in soggetti carenti ha evidenziato una riduzione della PCR ad alta sensibilità [5].

## MECCANISMO D'AZIONE DELLA VITAMINA D

La vitamina D può agire con meccanismo endocrino (la tipica azione di regolazione del metabolismo osseo), ma anche autocrino-paracrino grazie alla presenza all'interno delle singole cellule dell'enzima 1 $\alpha$-idrossilasi in grado di produrre il metabolita attivo $1,25(OH)_2D$. È l'azione autocrina-paracrina quella responsabile dell'effetto sulle cellule del sistema immunitario e di conseguenza sulla produzione di citochine proinfiammatorie. L'azione del metabolita attivo così prodotto è modulata dal legame con il suo recettore (VDR). Il VDR presente all'interno del nucleo di numerosissimi tipi cellulari media due tipi di azioni [1,6]:

- percorso non genomico: il legame del ligando ai VDR presenti nel citosol innesca molteplici percorsi a cascate di segnalazione intracellulare, portando a risposte immediate indipendenti dalla trascrizione genica nelle cellule;
- percorso genomico: il recettore dell'acido retinoico forma un eterodimero con il VDR legato a $1,25(OH)_2D$. L'eterodimero trasloca nel nucleo cellulare e si lega a degli specifici elementi di risposta alla vi-

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L'Autrice dichiara nessun conflitto di interessi.

**How to cite this article:** Viapiana O. Effetti della carenza di vitamina D sulle citochine infiammatorie. Vitamin D – Updates 2023;6(4):136-139. https://doi.org/10.30455/2611-2876-2023-8

tamina D (VDRE) su geni bersaglio, regolando di conseguenza la trascrizione nucleare.

Sia il VDR che l'1-α-idrossilasi sono espressi da diversi tipi di cellule immunitarie, tra cui macrofagi, cellule T, cellule dendritiche, monociti e cellule B, e l'evidenza di studi preclinici ha dimostrato che la vitamina D esercita effetti biologici sia sul sistema immunitario innato che su quello adattativo (Tab. I). L'1-α-idrossilasi extra-renale non è sovraregolata dal PTH (ormone paratiroideo); pertanto, la produzione di $1,25(OH)_2D_3$ dipende dai livelli del substrato $25(OH)D_3$ e può essere regolata da segnali infiammatori, come il polisaccaride (LPS) e le citochine stesse [1-2,6].

La vitamina D avrebbe un effetto diretto sulla produzione di citochine i cui meccanismi principali sono riassunti nella Tabella II [6].

## CARENZA DI VITAMINA D E CITOCHINE PRO-INFIAMMATORIE

La carenza di vitamina D è associata a un aumento dei livelli sierici di mediatori pro-infiammatori, tra cui l'IL-6 e il fattore di necrosi tumorale-alfa (TNF-α), che sono correlati sia allo sviluppo che alla progressione di patologie infiammatorie reumatiche e vascolari [1,2].

Oltre alle evidenze ormai datate che hanno osservato un'associazione tra deficit di vitamina D e citochine pro-infiammatorie nelle classiche patologie reumatologiche infiammatorie, come l'artrite reumatoide o le connettiviti, è stato recentemente pubblicato uno studio che ha documentato una correlazione lineare tra entità del deficit di vitamina D e incremento dei livelli di IL-6 e IL-8 nella fibromialgia. In particolare, livelli ridotti di vitamina D erano associati a maggiori punteggi sia per il dolore diffuso che per gli *scores* di attività di malattia [7].

In maniera analoga in un altro studio degli stessi autori in pazienti affetti da osteoartrosi di ginocchio è stata osservata una correlazione tra deficit di vitamina D e livelli più elevati di IL-6, e i livelli di IL-6 a loro volta sono risultati associati allo stadio radiografico della patologia e alla scala di funzionalità del paziente [8].

Infine, uno studio condotto su pazienti obesi ha rivelato che le ridotte concentrazioni sieriche di 25(OH)D erano solitamente correlate a livelli aumentati di altri biomarcatori di infiammazione vascolare, come la PCR ad alta sensibilità e il fibrinogeno.

**TABELLA I.**
Principali effetti della vitamina D sull'attività delle cellule coinvolte nell'immunità innata e adattativa.

| Immunità innata | Immunità adattativa |
|---|---|
| Aumento della differenziazione dei macrofagi | Riduzione delle citochine Th1 |
| Azione battericida | Aumento delle citochine Th2 |
| Inibizione della maturazione delle cellule dendritiche | Riduzione della differenziazione a Th17 |
| Inibizione della presentazione dell'antigene | Aumento della differenziazione dei T-regs |
| | Riduzione della proliferazione delle cellule B |
| | Induzione dell'apoptosi delle cellule B |
| | Inibizione della produzione di plasmacellule |
| | Inibizione della secrezione di immunoglobuline |

Th1: T Helper 1; Th2: T Helper 2; Th17: T Helper 17, T-regs: cellule T regolatorie.

**TABELLA II.**
Principali meccanismi con cui la vitamina D esplica il suo effetto antinfiammatorio.

| Bersaglio molecolare | Meccanismo | Effetto |
|---|---|---|
| MAP kinasi fosfatasi 5 | Attivazione dell'enzima che a sua volta inibisce p38 | Blocco del processo di amplificazione della cascata infiammatoria mediato da p38 |
| NF-kB | Tramite legame al VDR inibizione del fattore di trascrizione NF-kB | Riduzione della trascrizione/produzione di TNF-α, IL-1β e di conseguenza IL-6 |
| Cicloossigenasi 2 | Inibizione diretta della produzione di prostaglandine | Riduzione della proliferazione cellulare e dell'angiogenesi |

TNF-α: fattore di necrosi tumorale-alfa; NF-kB: fattore nucleare kappa B; IL-1β: interleuchina-1 beta; IL-6: interleuchina-6.

Conclusioni simili sono state raggiunte per i bambini gravemente obesi [1].

Tutti questi studi supportano l'ipotesi che nei soggetti carenti di vitamina D sia presente un contemporaneo incremento delle citochine pro-infiammatorie indipendentemente che si tratti di soggetti sani o di soggetti affetti da varie patologie reumatologiche e non.

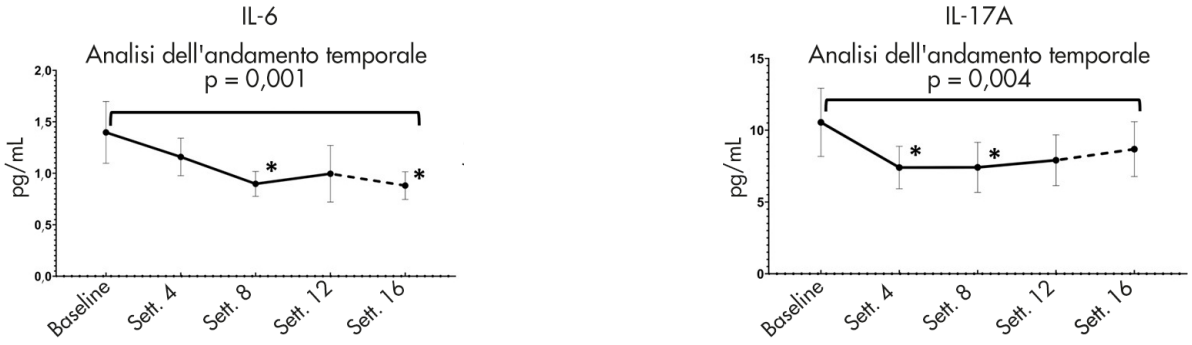
## EFFETTO DELLA SOMMINISTRAZIONE DI COLECALCIFEROLO NEI SOGGETTI CARENTI

Se esistono molte evidenze dell'associazione tra deficit di vitamina D e incremento delle citochine infiammatorie, pochi sono invece gli studi che hanno valutato l'effetto della somministrazione di colecalciferolo sullo stato infiammatorio e spesso sono presenti bias che ne limitano l'interpretazione.

In un gruppo di soggetti giovani e sani, ma carenti di vitamina D, abbiamo recentemente valutato l'effetto del colecalciferolo somministrato in 12 settimane sulla produzione di IL-17A, IL-6, IL-8, IL-10, IL-23 e TNF-α. Abbiamo osservato una progressiva riduzione dei livelli di IL-6 e IL-17A, mentre non sono state riscontrate differenze significative nelle concentrazioni sieriche delle altre citochine (Fig. 1) [9].

IL-6 e IL-17 sono due citochine chiave rispettivamente nell'artrite reumatoide e nelle spondiloartriti. La riduzione dei livelli sierici osservati in questo studio potrebbe supportare un possibile ruolo dell'integrazione di vitamina D nei pazienti affetti da malattie reumatologiche per ottimizzare la risposta terapeutica ai farmaci specifici. A supporto di questa opzione è stato inoltre osservato che nei pazienti con artrite reumatoide a seconda dei livelli sierici di 25(OH)D, l'integrazione di vitamina D avrebbe effetti diversi (positivi) sul dolore e sull'attività della malattia [10].

In un altro studio su soggetti sani ma an-




IL-6: interleuchina-6; IL-17A: interleuchina-17A; Sett.: settimana.

**FIGURA 1.**
Effetti della supplementazione con colecalciferolo sui livelli sierici di IL-6 e IL-17A in soggetti giovani, sani e carenti di vitamina D [9].

ziani (età media sopra i 70 anni) la somministrazione di colecalciferolo non ha invece modificato l'espressione genica e i livelli sierici di IL-6, IL-8, IL-10, TNF-α e IFN-γ. Da sottolineare tuttavia come i livelli di 25(OH)D sierici basali fossero più alti rispetto allo studio precedente e il dosaggio di colecalciferolo variabile a seconda dei gruppi di trattamento [11].
L'effetto sulla riduzione delle citochine è stato studiato anche in un piccolo gruppo di uomini sani sottoposti a intensa attività fisica di resistenza. Rispetto al placebo i soggetti supplementati hanno mostrato effetti positivi in termini di aumento dei livelli ematici di 25(OH)D, rapporto CD4+/CD8+ (risposta immunitaria) e capacità aerobica, inibendo le citochine infiammatorie (IL-6 e in misura minore TNF) e CK (creatina chinasi) e LDH (lattato deidrogenasi) (indicatori del danno muscolare) [12].
In Tabella III sono riassunte le caratteristiche principali degli studi che hanno valutato gli effetti della supplementazione con colecalciferolo sui livelli sierici delle citochine infiammatorie. L'effetto è più controverso invece in condizioni patologiche.
Una metanalisi di qualche anno fa su oltre 80 studi in condizioni patologiche differenti non ha evidenziato effetti significativi dell'integrazione di vitamina D sui biomarcatori infiammatori, tra cui proteina C-reattiva, IL-6 e TNF-α. Oltre all'eterogeneità delle condizioni morbose e della loro patogenesi, c'è da sottolineare che solo in 22 di questi studi è stata dosata l'IL-6 e solo in 25 il TNF-α [13].
Valutando alcune condizioni cliniche specifiche, Corrado et al. hanno recentemente dimostrato che l'esposizione in vitro a dosi crescenti di 1-25$(OH)_2$D in soggetti carenti era associata a una significativa riduzione di IL-17A e delle citochine profibrotiche (FGF2, TGF-β, CTGF) sia nei pazienti con sclerosi sistemica che nei soggetti sani, con un effetto dose-dipendente [14].
Invece in 44 pazienti affetti da sclerosi multipla e carenti in vitamina D, dopo 12 mesi di supplementazione con 500-1000 UI/die [a seconda dei livelli basali di 25(OH)D] di colecalciferolo, è stato osservato un aumento dei livelli sierici di citochine antinfiammatorie (IL-10, TGF-β) e dell'IFN-γ regolatorio, mentre l'IL-17 (proinfiammatoria) è rimasta invariata [15].
In pazienti affetti da patologia cardiovascolare l'integrazione di vitamina D, in soggetti carenti, è stata in grado di ridurre l'espressione di citochine pro-infiammatorie e proaterogeniche come IL-2 e in-

**TABELLA III.**
Studi che hanno valutato l'effetto della somministrazione di colecalciferolo sulle citochine pro-infiammatorie

| Autore | N. pazienti | Età media (anni) | 25(OH)D (ng/mL) | Dose somministrata | Durata | Effetto |
|---|---|---|---|---|---|---|
| Fassio et al. | 75 | 34 | 13,7 | • 10.000 UI/die per 8 settimane poi 1.000 UI/die per 4 settimane<br>• 50.000 UI/settimana per 12 settimane<br>• 100.000 UI a settimane alterne per 12 settimane | 12 settimane | Riduzione IL-6 e IL-17A |
| Berlanga et al. | 305 | 72 | 20 | • 4.000 UI/die<br>• 2.000 UI/die<br>• Placebo | 1 anno | Non effetto significativo |
| Liu et al. | 18 | 22 | 22 | • 5.000 UI/die<br>• Placebo | 4 settimane | Riduzione IL-6 |

IL-6: interleuchina-6; IL-17A: interleuchina-17A.

terferone-γ (IFN-γ), che sono responsabili dell'attivazione delle cellule T-helper-1 e dell'infiammazione vascolare [1].

Un discorso a parte merita lo stato di obesità. L'infiammazione cronica di basso grado sembra giocare un ruolo cruciale nello sviluppo delle comorbilità associate all'obesità, come l'insulino-resistenza, le malattie cardiovascolari e il cancro. La risposta infiammatoria sistemica nell'obesità avrebbe origine principalmente dal tessuto adiposo, promuovendo l'infiltrazione di cellule infiammatorie (macrofagi) e il rilascio di mediatori pro-infiammatori, portando a un'infiammazione sistemica di basso grado. A sostegno di ciò, studi precedenti hanno mostrato correlazioni positive tra il volume del tessuto adiposo e la secrezione di citochine pro-infiammatorie [4]. Un recente studio ha valutato l'effetto della supplementazione con probiotici (ceppi di lattobacilli e bifidobatteri), omega-3 e omega-6 e vitamina D sull'infiammazione di basso grado in individui con sovrappeso e obesità. Lo studio non ha mostrato differenze sull'outcome primario che erano i livelli di hs-CRP (proteina C-reattiva ad alta sensibilità). Tuttavia, nei soggetti trattati i livelli sierici di IL-6 sono diminuiti dopo la somministrazione a indicare un effetto seppur modesto sull'infiammazione [16]. I limiti principali di questo studio sono oltre la limitata casistica, la somministrazione contemporanea di probiotici omega-3-6 e colecalciferolo che non permettono di distinguere l'effetto dei singoli elementi e la bassa dose di vitamina D somministrata (200 UI/die, ben al di sotto delle dosi che finora hanno dimostrato effetti extrascheletrici).

Sebbene il razionale sia molto forte, solo uno studio ha documentato una riduzione della concentrazione sierica di IL-6 dopo la sola somministrazione di colecalciferolo nei soggetti obesi [17].

## CONCLUSIONI

Gli studi che hanno valutato l'effetto della supplementazione con vitamina D sulle citochine infiammatorie sono ancora pochi, talvolta con risultati discordanti e spesso non confrontabili tra loro in quanto condotti su popolazioni a volte carenti, a volte no e con comorbilità differenti. Tuttavia negli studi condotti su soggetti giovani, sani e carenti di vitamina D, dove i fattori confondenti sono ridotti, ed è possibile così valutare l'effetto "puro" del colecalciferolo, si evidenzia un effetto della supplementazione nel ridurre le citochine pro-infiammatorie. Se questi dati si confermassero, la vitamina D potrebbe diventare un trattamento complementare nella prevenzione e nel trattamento di numerose patologie reumatiche e infiammatorie.

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