VITAMIN D

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

Divisione Pacini Editore Medicina
Andrea Tognelli
Medical Project - Marketing Director
Tel. 050 3130255
atognelli@pacinieditore.it

Redazione
Lucia Castelli
Tel. 050 3130224
lcastelli@pacinieditore.it

Grafica e impaginazione
Massimo Arcidiacono
Tel. 050 3130231
marcidiacono@pacinieditore.it

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EDITORIALE

Maurizio Rossini
Dipartimento di Medicina, Sezione di Reumatologia, Università di Verona

Carissimi, non so voi ma io comincio a chiedermi se nell'acritica applicazione della metodologia statistica alla base dell'*Evidence Based Medicine* abbiamo dimenticato il presupposto che dovrebbe guidarla: il razionale fisiopatologico e clinico. Mi spiego meglio. Sul *Journal of the American Medical Association* (JAMA) è stata pubblicata recentemente la raccomandazione dell'*United States Preventive Service Task Force* (USPSTF) sull'uso della supplementazione con vitamina D e/o calcio per la prevenzione primaria delle fratture negli adulti viventi in comunità [1]. Si conclude che sulla base degli studi disponibili non vi sono evidenze sufficienti in termini di bilancio rischi/benefici per raccomandare la supplementazione con calcio e vitamina D e anzi si sconsiglia nelle donne in postmenopausa la supplementazione con dosi di vitamina D e calcio ≤ 400 UI o 1000 mg/die rispettivamente perché aumenta il rischio di calcolosi renale. Peccato che queste raccomandazioni non siano applicabili a persone con una storia di fratture osteoporotiche, con aumentato rischio di cadute, o con diagnosi di osteoporosi o di carenza di vitamina D (!), visto che queste erano in gran parte escluse dagli studi considerati. Visto che il buon senso ci dice, sulla base delle conoscenze di fisiopatologia, che la vitamina D serve solo quando manca … secondo me è come aver dimostrato che è inutile (se non dannoso) accendere una lampadina in una stanza nella quale non manca la luce! Per raggiungere questa conclusione c'era bisogno di una task force e di una complessa analisi?
Mi preoccupa anche l'impatto mediatico del messaggio conclusivo, che immagino sarà talvolta semplificato e comunicato o recepito in maniera acritica per esigenze editoriali od incompetenza.
E nelle persone a rischio di carenza? Smettiamo di fare prevenzione e attendiamo di documentarla, con relativi costi, o magari interveniamo solo quando la persona diventa paziente cioè sintomatica? Va anche considerato infatti che la stessa Task Force, secondo me giustamente, definisce insufficienti le evidenze in termini di bilancio benefici/rischi di uno screening del deficit di vitamina D in adulti asintomatici.
Credo d'altra parte che sia giustificato cercare di ridurre i costi esorbitanti della supplementazione con vitamina D ridimensionando le aspettative, migliorando l'appropriatezza dell'intervento, semplificandolo anche ricorrendo al solo buon senso ed evitando il ricorso alle soluzioni inutilmente più costose.
Una novità è rappresentata dalla recente autorizzazione all'immissione in commercio da parte dell'Agenzia Italiana del Farmaco (AIFA) di una nuova formulazione del calcifediolo in capsule molli. Ben vengano nuove soluzioni, specie se a basso costo, che allargano le opzioni terapeutiche dei medici nell'interesse dei pazienti, anche se va ricordato che è il colecalciferolo a rappresentare la forma della vitamina D fisiologicamente prodotta e metabolizzata. Quello che mi lascia perplesso è l'RCP (riassunto delle caratteristiche del prodotto) del nuovo prodotto a base di calcifediolo. In particolare mi preoccupa:

- L'espressione inappropriata del contenuto in UI di vitamina D, quando è noto che il calcifediolo non è assolutamente comparabile al colecalciferolo in termini di farmacocinetica e forse anche di farmacodinamica e vi sia tuttora discussione sull'entità del rapporto di equivalenza tra i due [2]. Ciò potrà generare un ulteriore motivo di confusione sui dosaggi di vitamina D, peraltro pericolosa in termini di safety;
- L'indicazione e la dose raccomandata per il "trattamento della carenza di vitamina D nei

Corrispondenza
MAURIZIO ROSSINI
maurizio.rossini@univr.it

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casi in cui risulta necessaria la somministrazione iniziale di dosi elevate ...": 0,266 mg di calcifediolo una volta al mese è da considerarsi una dose elevata se risulta la metà di quella ritenuta necessaria in recenti studi della Scuola del prof. Minisola [3,4] e considerato che l'emivita del calcifediolo è limitata a 2-3 settimane [5]?

- L'indicazione del "trattamento della carenza di vitamina D nei casi ... in cui sia preferibile una somministrazione dilazionata nel tempo, come nelle seguenti condizioni: come coadiuvante nel trattamento dell'osteoporosi, nei pazienti affetti da sindrome da malassorbimento, osteodistrofia renale, nelle patologie ossee indotte dal trattamento con corticosteroidi". Quali sono le evidenze a sostegno di trattamenti con calcifediolo preferibilmente dilazionati nel tempo in queste patologie?
- L'esigenza più volte ribadita di un "regolare controllo delle concentrazioni sieriche del 25-OH-colecalciferolo": questa prudenza potrebbe derivare dal fatto che l'incremento dei livelli sierici del 25-OH-colecalciferolo in seguito all'uso del calcifediolo, a differenza di quanto avviene con il colecalciferolo, non è fisiologicamente regolato. Peccato che l'uso di questa formulazione di calcifediolo, effettivamente poco costosa, potrà essere così gravato da elevati costi di gestione nella pratica clinica;
- L'affermazione che "in caso di insufficienza epatica, l'assenza di produzione di sali biliari darà luogo a incapacità di assorbire il calcifediolo", quando invece è riportato che l'assorbimento intestinale del calcifediolo, a differenza di quello del colecalciferolo, si realizza largamente tramite la vena porta [6] e non dipende dalla presenza di acidi biliari [7], tanto da giustificare, considerato anche il possibile deficit di 25OH-idrossilasi in condizioni di grave insufficienza epatica, l'uso preferibile del calcifediolo proprio in questa condizione [2].

Voi cosa ne pensate?
Buona lettura.

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Vitamina D: nulla di nuovo sotto il sole

Davide Gatti, Angelo Fassio
Dipartimento di Medicina, Università di Verona

Una vitamina è un elemento che, seppur presente in piccole quantità, ha un ruolo indispensabile per il normale svolgimento di uno o più processi fisiologici. In genere l'organismo non è in grado di sintetizzare queste sostanze da solo e pertanto esse devono venire introdotte regolarmente con la dieta. Questa definizione di vitamina, in realtà, viene in gran parte non soddisfatta proprio dalla vitamina D. Il calore e l'azione dei raggi ultravioletti solari sono in grado infatti di trasformare il 7-deidrocolesterolo presente sulla pelle proprio in vitamina D_3 (Fig. 1). Pertanto, la vitamina D diventa una "vera vitamina" solo quando l'uomo (e qualsiasi altro mammifero) non presenta un'adeguata esposizione alla luce solare e deve di conseguenza assicurarsi l'approvvigionamento tramite la dieta [1].

Un altro aspetto interessante è che la vitamina D (sia quella endogena sintetizzata dall'organismo a livello cutaneo che quella esogena introdotta con l'alimentazione) è un composto biologicamente inattivo.

Non ci sono mai stati dubbi, fin dalla scoperta della vitamina D all'inizio del secolo scorso, che la luce solare fosse in grado di correggere e prevenire il rachitismo proprio tramite la produzione di questa molecola, ma il reale meccanismo con cui questa sostanza (anche quando somministrata come integratore) agisse è stato a lungo sconosciuto. Solamente negli anni '60-70 si è finalmente chiarito che la vitamina D agisce in realtà da substrato per un processo metabolico complesso che, attraversando diverse tappe di idrossilazione e coinvolgendo diversi organi (primariamente fegato e rene), dà origine a un gran numero di metaboliti (Fig. 1). Fu poi ben presto dimostrato come il metabolita idrossilato in posizione 1 e 25 (calcitriolo) fosse oltre 400 volte più potente della vitamina D (substrato) nell'indurre a livello intestinale il trasporto attivo di calcio, chiarendo come esso rappresentasse in realtà la tappa metabolica finale e biologicamente attiva della vitamina D (Fig. 1) [2].

La storia tuttavia non finì a quel punto, perché l'identificazione dell'esistenza di una specifica proteina legante e quindi di un recettore (*vitamin D receptor* o VDR) [3] aprì nuovi e inattesi campi di ricerca. Infatti, divenne rapidamente chiaro che il recettore VDR era praticamente ubiquitario. In realtà, sono stati identificati due tipi di recettori per la vitamina D. Il primo, localizzato nel nucleo delle cellule, è in grado di stimolare direttamente la trascrizione di geni e quindi la sintesi ex-novo di proteine (meccanismo genomico). Il secondo invece si trova sulla membrana cellulare e agisce inducendo la formazione di secondi messaggeri cellulari (quali l'AMP ciclico, l'acido arachidonico e altri) o fosforilando alcune proteine cellulari. Quest'ultimo meccanismo è di tipo non genomico e assicura una risposta cellulare molto più rapida [4]. A questo punto, se consideriamo che la struttura del calcitriolo è quella di un ormone steroideo e che il suo recettore è distribuito in una gran quantità di tessuti, non possiamo non applicare il paradigma endocrinologico secondo il quale se una cellula esprime un recettore ormonale, quella cellula deve per forza avere in sé la capacità di produrre effetti biologici conseguenti al legame ormone-recettore (in questo caso quindi calcitriolo-VDR).

Tutto questo spiega perché l'interesse per la vitamina D non fu più solo limitato al metabolismo osseo ma anche ai cosiddetti effetti extra-scheletrici, legati all'importante ruolo fisiologico che essa svolge in numerose altre funzioni dell'organismo.

Se andiamo a ricercare su PubMed il termine "vitamin D" ci accorgiamo come la mole dei lavori pubblicati sia enorme e come il numero annuale di lavori sia andato incontro in questi ultimi 25 anni ad una rapida crescita. Fino al 1994 venivano pubblicati meno di 1000 lavori all'anno sulla vitamina D. Questo numero è raddoppiato nei successivi 15 anni per arrivare nel 2009 a superare i 2000 lavori/anno. In seguito, sono bastati solo 5 anni perché questo numero raddoppiasse ulterior-

Corrispondenza
DAVIDE GATTI
davide.gatti@univr.it

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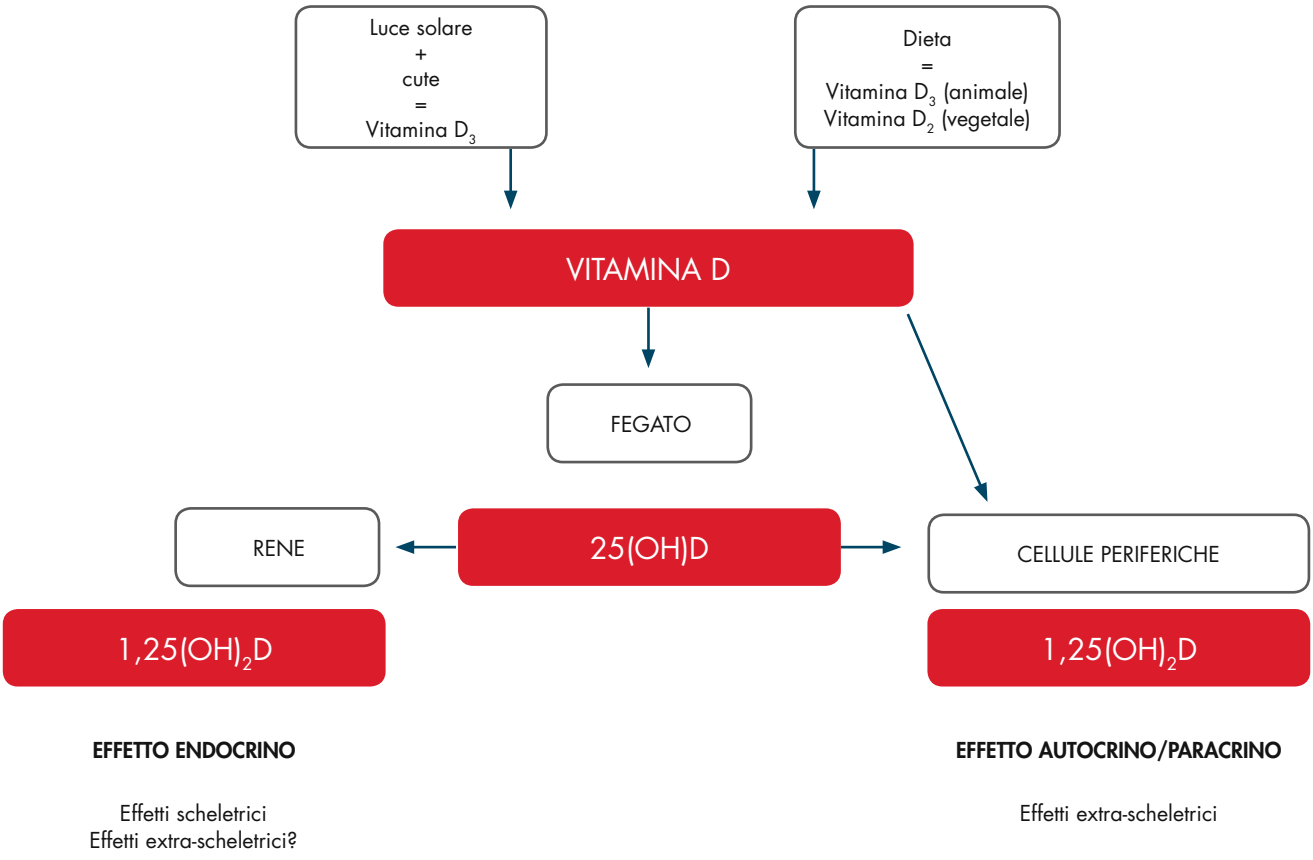

VITAMINA D: substrato inattivo; 25(OH)D: metabolita inattivo, marker delle stato vitaminico D; $1,25(OH)_2D$: metabolita attivo che produce gli effetti biologici mediante suo legame al VDR.

FIGURA 1.
Tappe metaboliche dell'attivazione della vitamina D e azioni biologiche.

mente (dal 2014 si pubblicano oltre 4000 articoli/anno su questo tema!). Nel 2018, considerando solo i primi sei mesi, siamo già a 2500 nuovi lavori.
Il grande interesse tuttavia non ha, come spesso accade, creato una cultura condivisa basata su dati oggettivi. Questo pullulare di studi, spesso di scarsa qualità e riguardanti aspetti marginali, ha finito per produrre ancora più confusione generando posizioni spesso contraddittorie anche tra esperti e società scientifiche. Purtroppo, ci si trova spesso a dover gestire posizioni basate su pregiudizi, estreme e fortemente contrapposte, tra chi vuole riconoscere a questa vitamina un ruolo quasi di panacea per tutti i mali (sopravvalutando gli effetti extra-scheletrici) e coloro che invece ne riconoscono un ruolo esclusivo e per di più solo per limitate problematiche metaboliche ossee (rachitismo e osteomalacia).
In realtà, non vi possono essere dubbi sul fatto che la vitamina D abbia ben altre azioni oltre all'assorbimento del calcio. La vitamina D interviene nella regolazione del 3% dei geni umani e molte cellule hanno un apparato enzimatico in grado di convertire localmente la vitamina D nel metabolita 25(OH)D e/o il 25(OH)D in calcitriolo con effetti regolatori paracrini e autocrini sulla proliferazione, differenziazione e funzione cellulare [5]. Detto ciò, va sottolineato che per ora non abbiamo ancora alcun dato certo che ci orienti sui livelli ideali indispensabili per poter accedere a questi effetti positivi né alcun studio interventistico convincente che ci orienti su schemi, dosi e durata di trattamento eventualmente ottimali. Pertanto, in completo accordo con quanto affermato recentemente dall'*European Society for Clinical and Economic aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases* (ESCEO) in un interessante *position paper* [6], al momento non è assolutamente possibile raccomandare né la supplementazione, né l'uso di dosi farmacologiche di prodotti a base di vitamina D per la prevenzione di patologie croniche extra-scheletriche. L'incertezza riguardo i vari aspetti gestionali e scientifici della vitamina D è presente anche in Italia come ha recentemente confermato un incontro tra esperti clinici a Verona (D...battito: *Mito o realtà. Opinione degli esperti italiani nella real life*). Di questo meeting, che ha coinvolto 50 specialisti di ambito differente (internisti, reumatologi, endocrinologi, geriatri, pediatri, dermatologi, ginecologi, nefrologi) su specifici temi relativi alla vitamina D, è in corso di stesura un documento dove verranno riportati i principali spunti emersi dalla discussione dei vari quesiti posti. Tuttavia, da una prima visione dell'orientamento espresso dai singoli partecipanti, viene fuori un'immagine molto eterogenea su quasi tutti i temi trattati. Alcune questioni in particolare sono risultate essere davvero divisive. Ad esempio, il dosaggio sierico per orientare la supplementazione viene ritenuto sempre indispensabile per

TABELLA I.
Soglie di livelli di 25(OH)D circolante proposte come ideali. Livelli di consenso e conseguenze negative dello stato carenziale cronico.

Livello di 25(OH)D considerato ideale	Grado di consenso	Conseguenze negative dello stato carenziale (se cronico)
> 10-12 ng/ml	Consenso generale	• Ridotto assorbimento intestinale di calcio • Iperparatiroidismo secondario • Livelli di calcemia e fosforemia ridotti o ai limiti inferiori della norma • Mancata mineralizzazione del tessuto osteoide → osteomalacia e riduzione della BMD (nell'adulto); rachitismo (in età pediatrica) • Anomalie scheletriche e radiografiche da rachitismo/osteomalacia • Anomalie extra-scheletriche con miopatia dei muscoli prossimali degli arti e possibile cardiomiopatia
> 20 ng/ml	Consenso largo	• Assorbimento intestinale di calcio ai limiti inferiori della norma • Iperparatiroidismo secondario • Aumento del turnover osseo • Aumento della perdita ossea • Osteoporosi accelerata
> 30 ng/ml	Consenso scarso	La *Endocrine Society* concorda sul limite di 20 ng/ml per la popolazione generale, ma nei soggetti a rischio o fragili raccomanda livelli > 30 ng/ml

circa il 40% dei partecipanti, mentre viene considerato da riservare solamente a limitati casi per il restante 60%. Una suddivisione simile emerge anche riguardo al target ideale da raggiungere: per il 38% la soglia era 20 ng/ml, mentre il 62% sostiene che si debbano superare i 30 ng/ml. La maggioranza (60%) ritiene che i dati attuali siano già convincenti riguardo gli effetti extra-scheletrici della vitamina D, d'altra parte il 78% richiede studi clinici controllati (RCT) e non solo osservazionali per dare sostegno alla terapia.

È evidente quanto sia necessario avviare un processo che possa portare a un po' più di chiarezza sul tema, dal momento che, se questo è lo spaccato del mondo specialistico, è evidente quale possa essere la confusione tra i "non addetti ai lavori". Questo diventa un problema particolarmente scottante visto che l'ipovitaminosi D non è per niente un problema limitato. L'ipovitaminosi D è infatti una condizione così diffusa da coinvolgere l'intero pianeta [7], anche se la gravità e la prevalenza dello stato carenziale varia molto da Paese a Paese, per effetto delle differenti abitudini di vita. In Italia la carenza vitaminica D è particolarmente frequente, specialmente negli anziani e nei mesi invernali. Quasi l'80% delle donne italiane sopra i 70 anni presenta infatti livelli ematici di 25(OH)D < 12 ng/ml alla fine dell'inverno [8], al punto da rendere del tutto scontato l'esito di un eventuale dosaggio ematico. Se consideriamo poi i soggetti istituzionalizzati o con comorbidità il dato diventa ancor più drammatico [9].

TABELLA II.
Valutazione critica di 2 recenti pubblicazioni di studi con risultati negativi sugli effetti muscoloscheletrici della vitamina D. La prima (Khaw et al., 2017) [10] è un vasto studio clinico controllato (RCT), la seconda (Zhao et al., 2017) [11] una meta-analisi di studi clinici in cui è stata utilizzata la vitamina D. Notare come nella selezione dei pazienti trattati solo una minima parte fosse realmente carente.

Khaw et al., 2017 [10]				
Casistica	Dose utilizzata	Livelli basali 25(OH)D dei pazienti:		
		% pz < 10 ng/ml	% pz 10-20 ng/ml	% pz > 20 ng/ml
5.110 soggetti (50-84 anni)	200.000 UI il 1° mese Poi 100.000 UI/mese	2%	22%	76%
Zhao et al., 2017 [11]				
		Livelli basali medi 25(OH)D degli studi sulla vitamina D		
		% < 10 ng/ml	% 10-20 ng/ml	% > 20 ng/ml
27.631 (58-82 età media)	Oltre il 50% degli studi con 800 UI o meno	0%	28%	57%
NB: nel 15% dei casi non era neanche prevista una valutazione basale dello stato vitaminico.				

Risulta quindi necessario fare chiarezza, per evitare che emergano dubbi sul fatto che possa essere davvero rilevante, dal punto di vista della salute personale e pubblica, correggere questo deficit. È vero: a oggi non c'è ancora un accordo generale su quali siano i livelli ottimali di vitamina D, nemmeno per la salute del tessuto osseo (Tab. I). Tuttavia, siamo tutti d'accordo che avere una grave carenza di vitamina D (< 12-10 ng/ml) non faccia bene alle ossa e che livelli > 30 ng/ml sarebbero ideali, ma quasi tutti riteniamo che sia preferibile portare questi valori almeno al di sopra dei 20 ng/ml. Questo rappresenta già un punto fondamentale per gestire l'ondata di ulteriore incertezza generata dagli esiti di alcuni studi e meta-analisi, spesso prodotte dagli stessi gruppi neozelandesi, per dimostrare che la supplementazione con vitamina D non abbia in realtà effetti rilevanti e sia pertanto del tutto inutile. L'interpretazione di questi studi merita, come sempre dovrebbe essere, un'analisi critica che non può fermarsi al mero risultato finale o addirittura al solo titolo. Negli studi (e nelle meta-analisi che ne derivano) dobbiamo considerare alcuni aspetti tutt'altro che secondari, come le caratteristiche della popolazione arruolata, le dosi utilizzate, la durata del follow-up, l'aderenza al trattamento, l'eventuale interferenza prodotta dalla presenza di altre fonti di vitamina D (dieta, esposizione solare). Un trial clinico non diviene automaticamente credibile solo perché controllato e in doppio cieco; la sua validità dipende fortemente anche da questi altri aspetti. Somministrare dosi elevate di vitamina D per lungo tempo non fornisce necessariamente la certezza di avere eseguito uno studio adeguato. Se selezioniamo una popolazione non carente di vitamina D, della quale pertanto non ne ha alcun bisogno (Tab. II), cosa dobbiamo aspettarci? Se sbagliamo a selezionare i pazienti nessuna analisi statistica potrà risolvere l'errore commesso alla base!

In conclusione: la vitamina D vive di un grande interesse scientifico e pubblico. Le potenzialità che la correzione dell'ipovitaminosi D può realizzare sono notevoli. Il nostro Paese da anni è capofila della gestione di questa problematica e i risultati iniziano a venire alla luce da più parti. Il clima di confusione e di incertezza che sta crescendo in questi ultimi anni non deve fermare gli interventi validi e razionali. Tutti, compresi specialisti, medici, pazienti, dobbiamo chiedere con forza l'intervento delle società scientifiche più autorevoli per fare chiarezza, cercando di raggiungere maggiori livelli di *consensus* e di pretendere studi clinici costruiti fin dall'inizio su fondamenta credibili.

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Vitamina D e psoriasi

Paolo Gisondi

Dipartimento di Medicina, Sezione di Dermatologia e Venereologia, Università di Verona

INTRODUZIONE

Il ruolo principale della vitamina D è quello di regolare il metabolismo del calcio e del fosfato e preservare la mineralizzazione del tessuto osseo. Tuttavia, sono molto valorizzate anche le sue funzioni extra-scheletriche incluse quella immunomodulante, anti-proliferativa e anti-infettiva. La vitamina D è d'interesse anche per il dermatologo perché essa è sintetizzata nella cute dopo esposizione ai raggi ultravioletti e la sua carenza è stata ripetutamente dimostrata in alcune malattie della pelle quali la psoriasi, la dermatite atopica e la vitiligine. Inoltre, dei derivati di sintesi della vitamina D inclusi il calcipotriolo e il tacalcitolo sono utilizzati comunemente come farmaci per uso topico nel trattamento della psoriasi e di altre malattie della cute immuno-mediate, in virtù delle loro proprietà antinfiammatorie. Nel presente articolo sarà trattato il ruolo della vitamina D nella psoriasi.

LA CUTE COME ORGANO DI SINTESI DELLA VITAMINA D

Vitamina D e paratormone regolano l'omeostasi del calcio e del fosfato, agendo su intestino, tessuto osseo e rene. In seguito all'esposizione solare, il 7-deidrocolesterolo (7-DHC o provitamina D) è convertito a livello cutaneo in previtamina D_3 la quale entro poche ore subisce l'isomerizzazione a vitamina D_3 (colecalciferolo) [1]. La sintesi cutanea di vitamina D_3 è influenzata da vari fattori quali il fenotipo cutaneo, l'età, l'uso di schermanti solari, la stagionalità, la latitudine, il tempo d'esposizione alla luce solare e l'entità della superficie corporea a essa esposta (Tab. I) [2]. La vitamina D_3 sintetizzata a livello cutaneo e quella assunta con gli alimenti è metabolizzata a livello epatico dall'enzima 25-idrossilasi in 25(OH)D_3 o calcifediolo, che rappresenta il principale metabolita in circolo della vitamina D e il miglior indicatore dello stato generale di vitamina D. Valori di 25(OH)D_3 compresi tra 30 e 100 ng/ml indicano uno stato vitaminico D adeguato. Valori di 25(OH)D_3 inferiori a 20 ng/ml configurano uno stato di deficit di vitamina D che può essere associato a debolezza muscolare, dolore osseo e aumentato rischio di fratture ossee. Valori compresi tra 20 e 30 ng/ml indicano uno stato di insufficienza [3]. La 25(OH)D_3 viene a sua volta convertita a livello renale, grazie all'enzima 25(OH)D-1α-idrossilasi, nella sua forma metabolicamente attiva, l'1,25$(OH)_2D_3$. La produzione renale di 1,25$(OH)_2D_3$ è regolata principalmente dal paratormone. Una volta attivata a 1,25$(OH)_2D_3$, la vitamina D esplica la sua attività biologica attivando il suo recettore nucleare ad alta affinità. I recettori per la vitamina D sono ubiquitari nell'organismo e presenti anche nei cheratinociti, e questo spiega perché la vitamina D possa svolgere funzioni anti-proliferative. Alla nostra latitudine, l'80% del fabbisogno di vitamina D è fornito dall'esposizione solare e solo il 20% dall'alimentazione. La vitamina D è poco presente nei cibi, la maggiore fonte alimentare di vitamina D è costituita dai grassi animali contenuti soprattutto nei pesci grassi, quali il salmone e l'aringa [4].

FUNZIONI IMMUNI DELLA VITAMINA D

La vitamina D svolge un ruolo importante nella regolazione dell'immunità innata e acquisita. La vitamina D può agire sulle cellule del sistema immunitario inclusi i linfociti, i macrofagi, le cellule dendritiche e i cheratinociti, in modo endocrino, autocrino e paracrino.
Per quanto riguarda gli effetti sull'immunità in-

Tabella I. Fattori che influenzano la sintesi cutanea di vitamina D.

Fenotipo
Età
Superficie cutanea esposta
Uso di schermi solari
Stagionalità
Latitudine
Tempo di esposizione alla luce solare

Corrispondenza
PAOLO GISONDI
paolo.gisondi@univr.it

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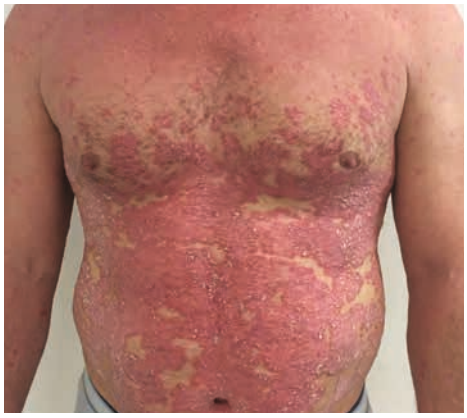

FIGURA 1.
Placche eritemato-squamose di psoriasi localizzate a livello del torace e addome in un paziente di 55 anni.

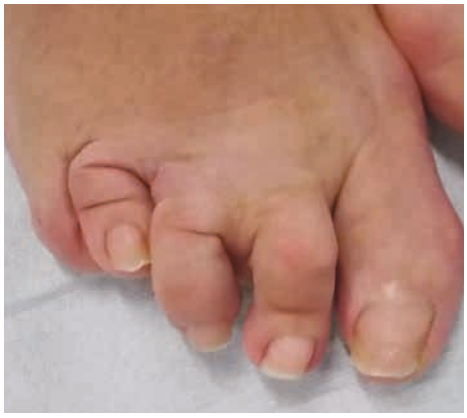

FIGURA 2.
Artrite psoriasica delle articolazioni interfalangee del piede con fenomeni di accorciamento "a cannocchiale" del III e IV dito del piede.

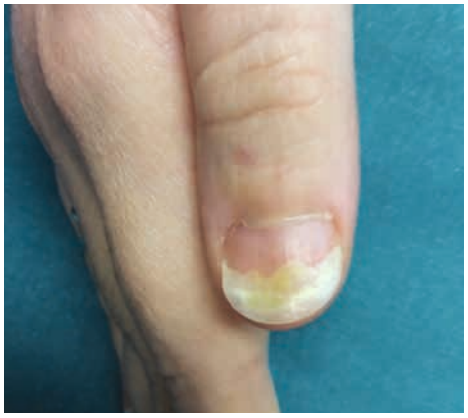

FIGURA 3.
Onicolisi psoriasica del I dito della mano in una paziente di 19 anni. La porzione distale della lamina è parzialmente sollevata dal letto ungueale.

nata, la vitamina D promuove la produzione da parte dei macrofagi e cellule epiteliali di peptidi antimicrobici quali defensine e catelicidine inclusa la hCAP18/LL-37 e nei macrofagi l'espressione di *Toll Like Receptor 2* e CD14 [5]. I peptidi antimicrobici esercitano la loro azione microbicida formando dei pori destruenti sulla membrana batterica e inibendo l'attività enzimatica, mitocondriale e la sintesi degli acidi nucleici e delle proteine batteriche. Inoltre, il legame tra la vitamina D e il suo recettore stimola il meccanismo dell'autofagia mediante la produzione di LL-37 che media la fusione del fagolisosoma con il lisosoma. L'autofagia serve a rimuovere proteine o organuli danneggiati ed esercita un'azione microbicida contro patogeni intracellulari [6].
La vitamina D modula alcune importanti funzioni dell'immunità acquisita [7]. In particolare, la vitamina D inibisce l'attività di presentazione l'antigene delle cellule dendritiche, riduce l'espressione di membrana del complesso maggiore d'istocompatibilità di classe II e delle molecole co-stimolatorie (CD40, CD80, CD86) e ne favorisce il fenotipo tolerogenico mediato dall'aumentata espressione di IL-10, MCP-1 e MIP-1α e ridotta espressione di IL-12. La vitamina D promuove il fenotipo T-regolatorio mediante produzione di IL-10, TGF-β, Foxp3 e CTLA4. In questo modo, la vitamina D potrebbe ridurre lo sviluppo di risposte auto-immunitarie [8]. Nel topo, l'applicazione topica di calcipotriolo (derivato della vitamina D) fa aumentare il numero di cellule T regolatorie nella cute. Un'altra importante azione della vitamina D sull'immunità acquisita è la regolazione della funzione e del fenotipo dei linfociti. In particolare, essa è in grado di inibire la produzione linfocitaria dell'interleuchina IL-1, IL-6, TNF-α e interferone-γ (INF-γ), potenti mediatori della risposta infiammatoria [9]. Studi in vitro mostrano, inoltre, che la vitamina D inibisce l'espressione di citochine Th1 (IL-2, TNF-α, INF-γ) e promuove quella di citochine Th2 (IL-3, IL-4, IL-5, IL-10).

VITAMINA D E PSORIASI

La psoriasi è una malattia infiammatoria cronica della cute che interessa circa il 3% della popolazione italiana e si manifesta con placche eritemato-squamose localizzate presso le sedi estensorie come gomiti, ginocchia e cuoio capelluto; in una parte dei pazienti la psoriasi è più diffusa e può interessare gran parte della superficie cutanea (Fig. 1). La psoriasi può essere associata nel 30% dei casi a una forma di spondiloartropatia nota come artropatia psoriasica che può presentare un decorso clinico molto sintomatico per il paziente e causare delle gravi alterazioni delle ossa (Fig. 2). Frequente è anche il coinvolgimento dell'apparato ungueale che rappresenta un'importante causa d'imbarazzo per il paziente (Fig. 3). La psoriasi insorge in persone geneticamente predisposte, come conseguenza di un'alterata reattività del sistema immunitario. La psoriasi è mediata dalle cellule T, in particolare dalle sottopopolazioni Th1 e Th17. La cute psoriasica è infiltrata, inoltre, dalle cellule dendritiche mieloidi (CDM) e plasmocitoidi (CDP), che vengono attratte nella cute psoriasica dalla chemerina prodotta soprattutto dai fibroblasti. I fibroblasti rilasciano pro-chemerina convertita in chemerina ad opera di elastasi rilasciate dai granulociti neutrofili [10]. Le CDM e CDP esprimono i recettori TLR-7 e -9 che riconoscono sostanze rilasciate dai cheratinociti danneggiati, come ssDNA, dsDNA e il peptide antimicrobico catelicidina/LL-37. I complessi di LL37 e DNA sono potenti attivatori delle CDP che rilasciano IFN-α e TNF-α attivatori della risposta immune adattativa. LL-37 svolge un ruolo fondamentale nella patogenesi della psoriasi in quanto rappresenta anche un importante autoantigene [11]. La maggior parte dei pazienti con psoriasi esprime linfociti T, inclusi Th17, reattivi verso LL-37 [11]. Le CDM attivate migrano ai linfonodi drenanti, determinando la differenziazione delle cellule T naïve in cellule effettrici Th1 e Th17. Popolazioni sensibilizzate di linfociti Th1 e Th17 CD4+ e cellule T effettrici CD8+ attivate penetrano e si accumulano secondo un gradiente chemiotattico nella cute. I cheratinociti attivati dalle citochine prodotte dai linfociti Th17 e Th1 (INF-γ, IL-22, IL-17, TNF-α) rilasciano chemochine come CCL20, CCL2, CCL5, IL-8, che reclutano leucociti e di conseguenza amplificano il processo infiammatorio. L'accumulo di linfociti T nella cute ha come conseguenza la secrezione di citochine pro-infiammatorie e di fattori di crescita che stimolano la proliferazione dei cheratinociti, provocando la comparsa delle lesioni psoriasiche.
La vitamina D può avere delle implicazioni rilevanti nel meccanismo patogenetico della psoriasi. Il gene che controlla l'espressione del recettore della vitamina D (VDR), sito sul cromosoma 12q13.11, presenta più di 200 SNPs (*single-nucleotide polymorphism*). In particolare, i quattro SNPs più

studiati (FokI, BsmI, ApaI, TaqI) sono stati associati a diverse malattie immuno-mediate inclusa la psoriasi, dermatite atopica e l'asma [12]. I polimorfismi del VDR possono predisporre alla psoriasi in quanto regolano la risposta immunitaria locale e il pathway infiammatorio legato a NF-kB. La vitamina D inibisce NF-kB perché aumenta i livelli di IkBα, riduce la capacità di NF-kB di legarsi al DNA, sopprime la trascrizione di NF-kB e reprime l'espressione di IL-1, IL-6, IL-8 e TNF-α che sono citochine pro-infiammatorie che hanno un ruolo importante nella patogenesi della psoriasi. Inoltre, la vitamina D stimola l'espressione di CTLA-4 e Foxp3 che in presenza di IL-2 inducono la formazione di linfociti T-regolatori. Il signaling del VDR agisce anche sul pathway JNK/c-Jun inibendo la proliferazione cellulare [13]. Studi in vitro e su modelli animali indicano che esiste un effetto immunomodulante della vitamina D, che è dimostrato dallo switch da Th1/Th17 a Th2/Treg. La vitamina D è un importante regolatore positivo dell'espressione di catelicidina da parte dei cheratinociti. Al contrario, la vitamina D e i suoi analoghi riducono l'espressione di altri peptidi antimicrobici quali psoriasina (S100A7) e koebnerisina (S100A15) da parte dei cheratinociti attivati da IL17A, IL-22 e TNF-α. Psoriasina e koebnerisina agiscono come potenti agenti chemiotattitici e alarmine che amplificano l'infiammazione della psoriasi.

Il deficit di vitamina D è stato riportato nei pazienti con psoriasi in diversi studi osservazionali. In particolare, in uno studio condotto a Firenze, Ricceri et al. ha osservato che il 97% dei pazienti con psoriasi presentava livelli di vitamina D inferiori a 30 ng/ml [14]. In un altro studio condotto a Verona su 145 pazienti affetti da psoriasi cronica a placche, 112 da artrite reumatoide e 141 controlli sani abbiamo osservato che il deficit di vitamina D [livelli di 25(OH)D < 20 ng/ml] era significativamente maggiore nei pazienti affetti da psoriasi rispetto a quelli affetti da artrite reumatoide o nei controlli [15]. Tale carenza nella popolazione psoriasica era particolarmente frequente nel periodo invernale rispetto a quello estivo (81% *vs* 37%). Un altro studio condotto in Spagna ha mostrato che il deficit di vitamina D interessa più frequentemente i pazienti con psoriasi e sindrome metabolica che i controlli e vi è una correlazione inversa tra i valori sierici di 25(OH)D_3 e glicemia e lipidi [16]. Sono stati condotti diversi trial clinici in aperto che hanno studiato l'efficacia della supplementazione della vitamina D_3 nella psoriasi e nell'artrite psoriasica. Nell'unico trial randomizzato, placebo-controllato, 9 su 20 pazienti (45%) trattati con 1 grammo di 1-idrossivitamina D_3 hanno mostrato un debole miglioramento rispetto agli 8 su 21 (38%) trattati con placebo. Tale differenza non era statisticamente significativa [17]. È stato riportato che il trattamento biologico con farmaci biologici anti TNF-α riduce i livelli sierici di vitamina D a differenza degli altri trattamenti sistemici inclusi ciclosporina ed acitretina, attraverso un meccanismo non ancora noto [18].

CONCLUSIONI

La vitamina D è prodotta dai cheratinociti a seguito dell'esposizione alla luce solare e regola molteplici funzioni immunologiche, oltre a quelle scheletriche. Gli effetti extra-scheletrici della vitamina D sono immunomodulanti, di controllo della proliferazione cellulare, anti-infettivi, anti-infiammatori e anti stress-ossidativo. Il deficit di vitamina D è stato descritto in diverse malattie immunomediate, inclusa la psoriasi che è una comune malattia della cute che può interessare anche l'apparato muscolo-scheletrico. Il significato clinico dell'ipovitaminosi D nella psoriasi, il ruolo e la modalità della sua supplementazione sono oggetto di studio. Non è noto se mantenere livelli adeguati di vitamina D possa prevenire l'insorgenza di malattie autoimmunitarie o comunque avere un effetto favorevole sulla storia naturale della malattia, inclusa la psoriasi, anche se vi sono i presupposti biologici per poterlo ipotizzare.

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