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 Vitamina D e muscolo

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Cari Lettori, come state? Spero bene.

Anche in questo numero raccogliamo importanti contributi su due tematiche di grande attualità nel campo dei possibili effetti extra-scheletrici della vitamina D: un update sulla relazione tra vitamina D e muscolo e un altro sulla sua relazione con l'asma e una delle maggiori cause della sua esacerbazione rappresentata dalle infezioni respiratorie. Quest'ultimo topic non poteva non fare riferimento anche alla tematica del momento rappresentata dall'infezione da COVID-19.

Come potete vedere dalla selezione bibliografica di questo numero abbiamo ritenuto opportuno dedicare uno spazio riservato alle pubblicazioni in tema di vitamina D e COVID-19, tante erano. La discussione tuttora in corso è relativa in particolare a due aspetti: è possibile che lo stato vitaminico D possa condizionare il rischio di infettarsi e/o la manifestazione clinica del COVID-19?

Come sapete i presupposti razionali ci sono e allo stato delle attuali conoscenze sono qui sintetizzabili e differenziabili in generici e specifici:

- generici:
 - studi in vitro hanno evidenziato che la vitamina D migliora la risposta immune innata, quale la risposta macrofagica, e può aumentare le difese antivirali, favorendo la produzione di peptidi antimicrobici come la catelicidina e la β -defensina;
 - studi osservazionali hanno documentato un'associazione tra bassi livelli sierici di 25(OH)D e la suscettibilità alle infezioni respiratorie;
 - una recente metanalisi ha mostrato che la supplementazione giornaliera o settimanale di vitamina D riduce significativamente il rischio di incorrere in un'infezione acuta delle vie respiratorie, non sorprendentemente in particolare nei soggetti carenti;
 - alla vitamina D è riconosciuto, anche da AIFA, un effetto "immunomodulante"; la vitamina D è risultata in grado di attenuare in particolare la risposta immunitaria adattativa e in particolare citochinica (tra cui soprattutto IL-6), riducendo la reazione di fase acuta post-virale, che, se esagerata, contribuisce alla patogenesi delle più gravi manifestazioni cliniche dell'infezione virale (il cosiddetto danno da "fuoco amico");
 - la supplementazione con vitamina D di pazienti sottoposti a ventilazione meccanica per varie cause è risultata in grado di ridurre la durata del ricovero e i livelli di PCR e IL6;
- specifici per COVID-19:
 - maggiore mortalità nelle Nazioni del Sud Europa (Italia, Spagna), notoriamente con una maggiore prevalenza del deficit di vitamina D, rispetto a Nazioni del Nord Europa (Germania, Norvegia, Finlandia, Islanda) che hanno un introito alimentare di vitamina D maggiore, grazie anche alla usuale fortificazione dei cibi;
 - maggiore prevalenza dell'infezione da COVID-19 nelle Regioni del Nord Italia, rispetto alle più soleggiate Regioni del Sud o alle popolazioni sotto il 35° parallelo;
 - particolare incidenza e gravità dell'infezione nella popolazione anziana nella quale l'ipovitaminosi D è notoriamente e storicamente endemica, specie nei mesi invernali e nella prima primavera, e drammaticamente e cronicamente presente in condizioni di lungodegenza;

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- l'obesità, notoriamente associata spesso a ipovitaminosi D, è risultata un fattore di rischio significativo per morbilità e mortalità da COVID-19;
- correlazione inversa tra i livelli sierici di vitamina con incidenza e mortalità da COVID-19;
- associazione inversa tra livelli di 25(OH)D e severità dell'infiammazione sistemica e delle manifestazioni cliniche in ricoverati per COVID-19, anche se va onestamente considerato che è noto che la flogosi di per sé riduce i livelli di 25(OH)D dosabili;
- modulazione da parte della vitamina D del sistema renina-angiotensina e dell'espressione del recettore ACE2, noto come punto d'ingresso del virus nelle cellule umane.

Come vedete la plausibilità biologica di un ruolo protettivo della vitamina D nei confronti del rischio e/o della gravità delle manifestazioni cliniche dell'infezione da COVID-19 c'è.

Ciò tuttavia non basta.

Proclami anticipati e generici di un ruolo protettivo della vitamina D, non supportati da adeguata documentazione scientifica specifica, ne hanno determinato la classificazione tra le bufale da parte del Ministero della Salute. Io non ne sarei tuttavia così sicuro perché non vi è neppure evidenza scientifica che ciò non sia vero... e scoraggiare, in attesa dei risultati dei trial specifici in corso, una supplementazione sicura ed economica con vitamina D, in particolare negli anziani o se costretti a casa o lungodegenti, non mi sembra opportuno in condizioni di emergen-

za da COVID-19, specie se si considerano perlomeno i riconosciuti benefici scheletrici. Mi preoccupa tra l'altro la riduzione di oltre il 30% della supplementazione con vitamina D osservata nei primi mesi dell'anno anche negli anziani in seguito alla pubblicazione della nota ALFA 96, che ignora l'età avanzata come fattore di rischio di ipovitaminosi D, non considerando la documentata progressiva riduzione dai 60 anni (sic...la mia età...) della capacità della pelle di sintetizzare vitamina D e il nadir stagionale. Trattasi di una perlomeno sfortunata coincidenza con il picco dell'infezione da COVID-19 in Italia... e comunque di un ulteriore motivo di preoccupazione per la salute dello scheletro dell'anziano.

Cosa ne pensate?

Buona lettura!

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VITAMINA D E MUSCOLO

Negli ultimi decenni si è assistito a un crescente interesse scientifico per gli effetti extrascheletrici del sistema della vitamina D, incluso il suo ruolo sulla funzione muscolare. L'ipotesi che questo ormone secosteroideo fosse coinvolto nell'attività muscolare è nata dall'osservazione che i bambini con carenza vitaminica, affetti da rachitismo, sperimentano un grave *impairment* muscolare, definito come "miopatia rachitica" ¹. Per comprendere meglio i meccanismi fisiopatologici del danno muscolare, nella prima metà del secolo scorso è stata formulata una teoria basata sul ruolo chiave delle alterazioni della concentrazione di estere fosforico nel tessuto muscolare ². Tuttavia, tale teoria non teneva conto degli effetti dell'iperparatiroidismo secondario all'ipovitaminosi D sul muscolo scheletrico, poiché è noto che l'iperparatiroidismo porta a un significativo danno muscolare, caratterizzato da atrofia dovuta a una perdita preferenziale delle fibre di tipo II ³, che si differenzia dalle alterazioni patognomoniche della miopatia primaria, in cui si assiste alla degenerazione, fino alla necrosi, delle fibre muscolari accompagnata dalla proliferazione del tessuto connettivo endomisiale.

La scoperta dei recettori della vitamina D (VDR) è una pietra miliare dello studio degli effetti pleiotropici della vitamina D ⁴. A livello del target muscolare, i VDR sono stati identificati non solo in mioblasti, miotubi e fibrocellule muscolari di modelli animali, ma anche nei miociti umani ^{5,6}. La vitamina D agirebbe, di conseguenza, su molteplici componenti cellulari del muscolo scheletrico durante le diverse fasi della vita dell'individuo, a partire dallo sviluppo embrionale, particolarmente nella riparazione tissutale post-danno che avviene durante tutta la vita, fino alla modulazione dell'involuzione legata all'invecchiamento.

Gli effetti della vitamina D sul tessuto muscolare si realizzano principalmente attraverso due meccanismi: uno a lungo termine, che coinvolge una via genomica, e un altro a breve termine, che coinvolge una via non genomica ⁷. Entrambi i meccanismi agiscono in modo sinergico sia sulla contrazione musco-

lare in risposta ai flussi intracellulari di calcio (risposta rapida), sia sulla massa e forza muscolare (risposta a lungo termine).

Attraverso il primo meccanismo, la vitamina D stimola la proliferazione e la differenziazione delle cellule muscolari modulando la trascrizione genica nei mioblasti, con conseguente aumento della sintesi di specifiche proteine muscolari, come la miosina e la proteina legante il calcio (*calcium-binding protein*, CBP). Questo meccanismo comporta il legame diretto della vitamina D attivata dal complesso VDR nucleare/recettore retinoide (*retinoid-X receptor*, RXR) a specifiche sequenze di DNA, note come elementi di risposta alla vitamina D (*vitamin D response element*, VDRE), con conseguente regolazione della trascrizione. Oltre a modulare l'assorbimento di calcio, la vitamina D regola il metabolismo del fosfato a livello muscolare, per far fronte alle esigenze strutturali ed energetiche cellulari. Shirvani et al. ⁸ hanno recentemente dimostrato che il meccanismo genomico attivato dalla vitamina D è proporzionale in quantità alla dose di vitamina assunta come supplemento, infatti viene regolata l'espressione di 162 geni (86 up-regolati e 76 down-regolati) nelle cellule ematiche della serie bianca di individui adulti normali che assumono 600 UI/die per 6 mesi, mentre il numero di tali geni raddoppia se vengono assunte 4.000 UI/die e addirittura viene moltiplicato per un fattore di 8 se la dose sale a 10.000 UI/die.

Attraverso il meccanismo a breve termine, invece, la vitamina D regola l'azione calcio-mediata di secondi messaggeri, che interviene sia nell'interazione tra citosol e mitocondri per modulare il metabolismo energetico muscolare, sia influenzando i meccanismi alla base della contrazione muscolare ⁹. I meccanismi non genomici, che si realizzano in seguito al legame della vitamina D con il VDR nucleare (nVDR) e/o con quello di membrana (mVDR) legato alla caveolina 1, includono l'attivazione di molecole di segnale intracellulari come PKC, PI3K, MAPK, CaMKII e PLA2 ¹⁰. Nella sua forma attiva, il calcitriolo, influenza la funzione muscolare agendo sui canali voltag-

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Conflitto di interessi

Gli Autori dichiarano nessun conflitto di interessi.

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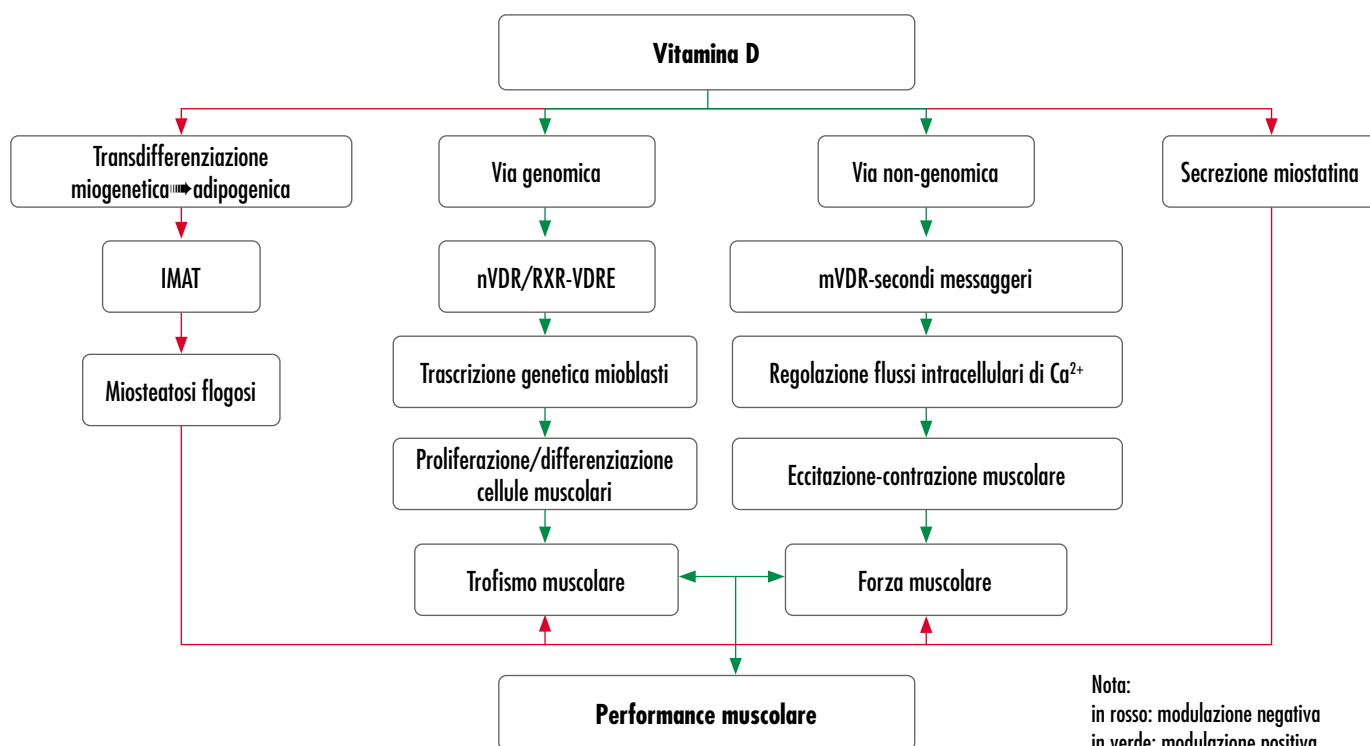


FIGURA 1.

Meccanismi biologici alla base degli effetti della vitamina D sul muscolo scheletrico.

gio dipendenti SOC/TRPC3 per regolare i livelli intracellulari di calcio e conseguentemente l'accoppiamento eccitazione-contrazione delle fibre muscolari scheletriche. Più recentemente è stato ipotizzato un terzo meccanismo mediante il quale la vitamina D eserciterebbe effetti benefici sulla funzione muscolare, ovvero inibendo la transdifferenziazione dei precursori miogenici nelle cellule adipogeniche riducendo così l'accumulo di tessuto adiposo intra- e intermuscolare (*intermuscular adipose tissue*, IMAT) ¹¹. Un altro ipotetico effetto indiretto della vitamina D deriva dall'inibizione della secrezione di miostatina, regolatore negativo chiave della massa muscolare, da parte delle cellule muscolari, come dimostrato da studi in vitro su mioblasti esposti al calcitriolo ¹² (Fig. 1). Dal punto di vista clinico, l'evidenza che la vitamina D possa giocare un ruolo essenziale sulla funzione muscolare deriva soprattutto dalla mole di studi condotti sulla controversa associazione tra ipovitaminosi D e rischio di caduta, in particolare nel periodo dell'invecchiamento. Come è noto, nella popolazione anziana, specialmente in quella istituzionalizzata, sia l'espressione di VDR a livello muscolare sia le concentrazioni sieriche di 25(OH)D sono ridotte ¹³. È stato ipotizzato che la carenza di vitamina D svolga un ruolo fondamentale

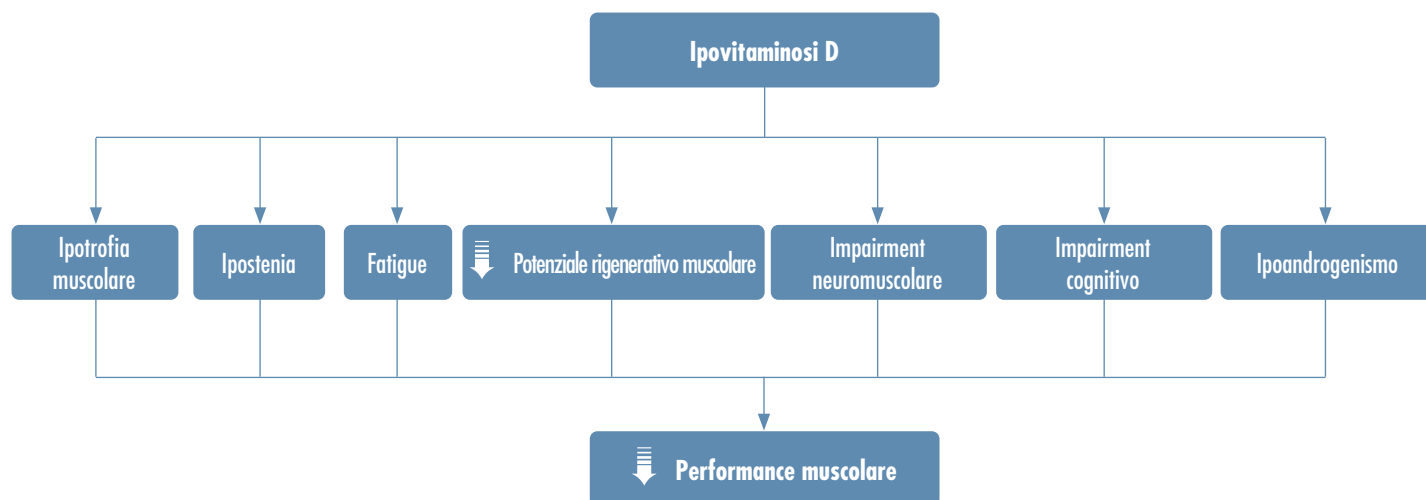
nella perdita di massa muscolare legata all'età e che l'*impairment* muscolare preceda la comparsa dei segni biochimici di osteomalacia ¹⁴. L'ipovitaminosi D si associa in modo significativo sia alla riduzione del trofismo muscolare sia al peggioramento della forza appendicolare, in particolare dei muscoli antigravitari, e della performance fisica ^{15,16}. È interessante notare che negli anziani con una carenza di vitamina D prolungata nel tempo si assiste allo stesso pattern miopatico riscontrato in corso di rachitismo, con una riduzione preferenziale delle fibre di tipo II, peraltro riscontrato anche in corso di sarcopenia, sindrome caratterizzata dalla riduzione della massa e della performance muscolare. Tale condizione aumenta il rischio di cadute e di fratture da fragilità ¹⁷, eventi che a loro volta, contribuiscono a ridurre la mobilità e l'autonomia, esacerbando lo stato di ipovitaminosi D, a causa della riduzione delle attività all'aperto e quindi dell'esposizione alla luce solare ¹⁸.

Il sistema vitamina D, attraverso i numerosi recettori ed enzimi presenti ubiquitariamente nel corpo umano, contribuisce a modulare la performance muscolare regolando diverse funzioni legate alle prestazioni fisiche, alla sintesi di androgeni, alle prestazioni cognitive e alla neuroprotezione. La deplezione di vitamina D attiva e disponibile

per espletare le sue funzioni fisiologiche nell'organismo condiziona la comparsa di una sequela di eventi patologici che caratterizzano la compromissione della funzione muscolare (Fig. 2).

Anche se è noto che condizioni di ipovitaminosi, e di alterata funzionalità muscolare, sono più comuni nell'età avanzata, è da sottolineare che la vitamina D ha un ruolo importante nella performance fisica e cognitiva anche in soggetti più giovani. Recentemente, infatti, è stato preconizzato l'uso di supplementazione di vitamina D anche in popolazioni selezionate nelle quali è richiesta una prestazione fisica e psico-emozionale di notevole impatto, come quella dei militari coinvolti in scenari di guerra. Wentz et al. ¹⁹ sostengono che la supplementazione di vitamina D ai soldati ipovitaminosici costituisce un intervento non invasivo e a basso costo per l'implementazione della performance di combattimento.

È indubbio che tra le tante azioni extrascheletriche attribuite al sistema vitaminico D, quella sulla muscolatura striata rappresenta un intrigante elemento nella conoscenza dei meccanismi alla base della sua complessa attività biologica. Gli studi preclinici, gli studi clinici e gli studi osservazionali sembrerebbero confermare uno stretto rapporto

**FIGURA 2.**

Meccanismi patoclinici dell'ipovitaminosi D nella compromissione della performance muscolare.

tra tasso sierico di 25(OH)D e attività muscolare, in particolare nel migliorare la prestazione funzionale e nel ridurre il rischio di caduta e di disabilità.

Al momento, comunque, non vi è consenso in merito ai livelli sierici ottimali da raggiungere, alla dose da supplementare e alla frequenza di somministrazione per ottenere i potenziali effetti benefici della vitamina D sulla funzione muscolare.

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Vitamina D e asma

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L'asma è un complesso disturbo respiratorio di origine infiammatoria la cui insorgenza e il cui decorso sono influenzati da molteplici elementi tra cui fattori genetici, ambientali ed etnici, oltre che da condizioni socio-economiche¹. È caratterizzata da una eterogeneità individuale che si manifesta con diverse risposte a diversi trigger e a diverse terapie. Riconoscere l'eterogeneità dell'asma e la relativa caratterizzazione aiuta a selezionare un percorso terapeutico specifico per ogni paziente².

Per spiegare la crescente prevalenza dell'asma e delle malattie allergiche nei paesi occidentali a partire dagli anni '70 sono state formulate l'ipotesi dell'igiene, l'ipotesi del microbioma intestinale e altre teorie che hanno associato la vitamina D allo sviluppo di asma, wheezing, rinite allergica, allergie alimentari e dermatite atopica³. Il ruolo dell'occidentalizzazione, di uno stile di vita in cui si trascorre più tempo al chiuso nella carenza di vitamina D, e nell'aumento di asma e allergie è stato analizzato dall'*International Study of Asthma and Allergies in Childhood* (ISAAC) che ha evidenziato l'altissima incidenza dei sintomi dell'asma in paesi quali la Gran Bretagna, l'Australia, la Nuova Zelanda e l'Irlanda^{4,5}. Alcuni studi condotti in diverse città cinesi con vari profili socioeconomici hanno riscontrato la prevalenza dell'asma e dei sintomi allergici a Hong Kong, la città più occidentalizzata tra quelle studiate⁶.

In anni recenti la vitamina D, in virtù delle sue proprietà immunomodulanti tramite la regolazione delle funzioni del sistema immunitario adattivo e innato, è stata considerata come un nuovo fattore potenzialmente in grado di inibire l'infiammazione delle vie aeree; in effetti la carenza di vitamina D è correlata a un aumento delle infiammazioni delle vie aeree, a una compromessa funzionalità polmonare, a un aumento delle esacerbazioni e delle prognosi infauste nei pazienti asmatici^{7,8}. In particolare, l'interesse per la possibile funzione immunomodulante della vitamina D nell'asma è scaturito dalla considerazione della presenza del recettore per la vitamina D (VDR) sulle cellule immunitarie e su vari

tessuti delle vie respiratorie. I recettori VDR si ritrovano nelle cellule epiteliali del tratto respiratorio e nelle cellule immunitarie (cellule B, cellule T, macrofagi e monociti) e la forma attiva della vitamina D [1,25(OH)₂D₃] esplica i suoi effetti fisiologici legandosi ai recettori VDR^{9,10}.

EFFETTI DELLA VITAMINA D SUL SISTEMA IMMUNITARIO

Le attività biologiche più note della vitamina D sono l'omeostasi del calcio e il metabolismo osseo. Ma considerato che il recettore della vitamina D, un membro della famiglia dei recettori nucleari, è stato localizzato in diversi tessuti e cellule del corpo umano, incluse le cellule dendritiche (DC), importanti cellule che presentano l'antigene, è lecito supporre che le attività della vitamina D siano numerose^{11,12}. Queste attività hanno sede nel sistema immunitario, dal momento che la vitamina D svolge un ruolo ben preciso nel decorso delle patologie immuno-mediate, inibendo la risposta e la proliferazione dei linfociti Th1 e Th17¹³. La vitamina D ha un ruolo determinante nel differenziamento delle cellule T regolatorie (Treg)¹⁴. Diversi studi riportano gli effetti positivi della vitamina D sulle patologie che comportano una iperattivazione dei linfociti Th1, come l'artrite reumatoide, la sclerosi multipla, la psoriasi¹⁵.

È stato riportato un effetto benefico della vitamina D sul decorso delle patologie allergiche in cui i linfociti Th2 svolgono un ruolo fondamentale, a prescindere dai meccanismi patogenetici soggiacenti¹⁶. Uno studio di Pichler et al. ha preso in esame il ruolo della 1,25(OH)₂D₃ sui linfociti T CD4+ helper naïve, e sui linfociti T CD8+ citotossici in colture cellulari isolate dal cordone ombelicale umano, riscontrando gli effetti inibitori della 1,25(OH)₂D₃ sulla produzione di IFN-γ indotta da IL-12 e sulla produzione di IL-4 e IL-13 indotta da IL-4 nelle cellule naïve¹⁷. Oltre a essere coinvolta in questi meccanismi, la vitamina D ha la facoltà di inibire IL-17A e di prevenire la conversione dei linfociti T CD8+ da cellule che produco-

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no IFN- γ a cellule che producono IL-13 documentata nei pazienti asmatici resistenti ai corticosteroidi^{18,20}.

Nel valutare il possibile ruolo della vitamina D nella risposta immunitaria eosinofila, è stata documentata la sua capacità di prolungare la sopravvivenza delle cellule che esprimono il recettore della vitamina D¹¹. Inoltre, la vitamina D riduce la necrosi eosinofila e il rilascio citolitico della perossidasi. Sono stati riscontrati inoltre una ridotta produzione dell'immunoglobulina E (IgE) e un incremento nell'espressione di IL-10²¹. Filho et al., analizzando una popolazione non selezionata, non dominata da pazienti allergici, riportano che i pazienti affetti da carenza di vitamina D presentano un aumento nella conta di eosinofili nel sangue. È stata riscontrata una correlazione inversa della vitamina D con i livelli sierici di basofili e neutrofilii²².

MODULAZIONE GENETICA DELLA VITAMINA D

Il legame della vitamina D e dei suoi analoghi al recettore VDR regola l'espressione di numerosi geni associati con l'infiammazione e l'immunomodulazione²³ (Fig. 1). Gli effetti biologici della vitamina D possono essere alterati da polimorfismi a singolo nucleotide (SNP) del gene VDR e potrebbero essere coinvolti negli effetti biologici della

vitamina D. Nello specifico, molti studi ipotizzano che i polimorfismi a singolo nucleotide (SNP) nel gene VDR quali rs2228570 (FokI), rs731236 (TaqI), rs1544410 (BsmI) e rs7975232 (ApaI), rappresentino dei fattori di rischio per l'insorgenza dell'asma. Masoud Hassanzadeh Makoui et al. hanno riscontrato un'associazione statisticamente rilevante tra i polimorfismi genici FokI e TaqI e il rischio asmatico^{24,25}. In aggiunta, nello stesso studio, l'analisi dei sottogruppi è stata stratificata al fine di valutare un potenziale effetto specifico correlato all'etnia, rivelando che la presenza del polimorfismo FokI in una popolazione africana riduce il rischio asmatico sotto il modello dominante (OR = 0,60) e il modello allelico (OR = 0,54). Il genotipo TT del polimorfismo TaqI è stato associato all'aumento del rischio di asma in una popolazione asiatica (OR = 2,94) e a una diminuzione del rischio nelle popolazioni americane (OR = 0,64). Questo risultato ha evidenziato una differenza di outcome tra le etnie che potrebbe essere causata da modelli nutrizionali diversi, discrepanza geografica, diversità etnica e da una significativa influenza dell'etnia sull'espressione del gene VDR e sui livelli sierici di vitamina D^{25,26}. Ognuno di questi quattro SNP può disturbare la stabilità mRNA del VDR, che a sua volta induce uno squilibrio tra Th1

e Th2, con la conseguente riduzione della produzione di IL-12 e di IFN- γ , con una produzione predominante delle citochine Th2, quali IL-4 e IL-13²⁵.

IL RUOLO DELLA VITAMINA D E LA RISPOSTA ALLA TERAPIA NEI PAZIENTI ASMATICI

La vitamina D potrebbe svolgere un ruolo anche nella risposta alla terapia anti-infiammatoria nei pazienti asmatici, in particolare ai glucocorticoidi (GC)²⁷. Valutando la risposta ai GC nei pazienti asmatici, fino al 50% dei pazienti potrebbe non avere una buona risposta ai corticosteroidi inalati (ICS), e fino al 25% dei pazienti con asma difficile da controllare potrebbe non rispondere positivamente ai GC orali, con elevata morbilità e un decorso della malattia potenzialmente mortale^{28,29}. I difetti nella trascrizione genica GC-indotta dei mediatori anti-infiammatori come IL-10 e le proteinchinasi fosfatasi attivate da mitogeni (MKP-1) potrebbero avere un ruolo nei pazienti affetti da asma resistente agli steroidi^{30,31}.

A conferma di quanto appena detto, Xystrakis et al. hanno constatato che l'aggiunta della vitamina D e del desametasone (Dex) in colture di cellule Treg CD4+ di pazienti affetti da asma resistente agli steroidi ha aumentato la secrezione di IL-10 a livelli comparabili a quelli secreti dalle cellule di pazienti sensibili agli steroidi trattati solo con il Dex³⁰. Zhang et al. hanno confermato che la vitamina D ha aumentato l'induzione da GC di MKP-1 e IL-10 nelle cellule mononucleate da sangue periferico di bambini asmatici³².

IL RUOLO DELLA VITAMINA D CONTRO LE INFEZIONI RESPIRATORIE

La vitamina D svolge un ruolo fondamentale anche nel proteggere dalle infezioni respiratorie e dunque nella prevenzione delle esacerbazioni asmatiche. Studi osservazionali hanno documentato un'associazione tra basse concentrazioni sieriche di 25(OH)D e una suscettibilità alle infezioni respiratorie acute e alle esacerbazioni nei pazienti asmatici^{33,34}. In particolare, gli studi in vitro in linee cellulari epiteliali e nelle colture primarie di cellule epiteliali bronchiali infettate da virus del genere Rhinovirus dimostrano che la vitamina D può aumentare le difese antivirali, migliorando la produzione di peptidi antimicrobici (AMP) come la catelicidina e la β -defensina^{35,36}. L'attivazione

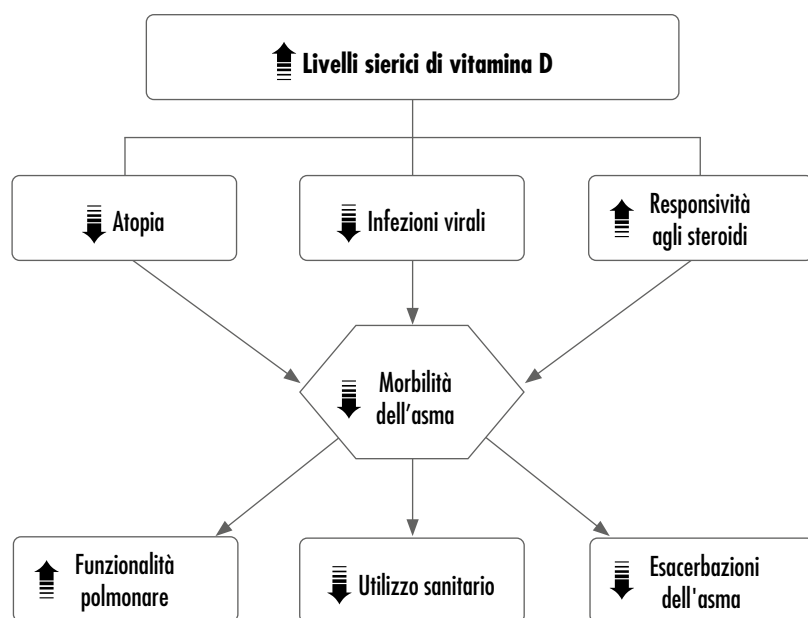


FIGURA 1.

La vitamina D e il suo network.

dei recettori dell'immunità innata *Pathogen Recognition Receptor* (PRR) sulle cellule epiteliali dell'apparato respiratorio upregola la secrezione di peptidi antimicrobici tramite le cellule epiteliali, la morte cellulare programmata e altre vie di risposta intracellulare, rilasciando mediatori proinfiammatori, citochine e chemochine. La vitamina D può interferire con molte azioni svolte dai recettori PRR^{37,38}.

Diversi studi riportano che i supplementi di vitamina D assunti per via orale da bambini soggetti a infezioni ricorrenti del tratto respiratorio (RRTI) riduce il numero delle infezioni e il loro impatto socioeconomico globale, con un ruolo di prevenzione³⁹. Martineau et al., in una meta-analisi dei dati individuali dei partecipanti (*individual participant data IPD*) condotta nell'ambito di studi controllati randomizzati, hanno evidenziato che la supplementazione con vitamina D induce una riduzione del rischio di sperimentare almeno una infezione acuta delle vie respiratorie. Le somministrazioni giornaliere o settimanali di vitamina D hanno offerto grandi benefici ai pazienti affetti da una grave carenza di vitamina D al basale, e anche i pazienti con più alte concentrazioni di 25(OH)D ne hanno tratto evidenti vantaggi³⁴. Diversi studi documentano una connessione tra la carenza di vitamina D e una elevata suscettibilità e gravità della tubercolosi (TB)⁴⁰.

VITAMINA D ED ESACERBAZIONI

Alcune meta-analisi hanno evidenziato il ruolo della supplementazione con vitamina D nel ridurre la frequenza di esacerbazioni, soprattutto nei pazienti affetti da carenza di vitamina D⁴¹. In effetti, riacutizzazioni di moderata entità sono state riscontrate dopo il trattamento mediante supplementazioni con vitamina D solo nei pazienti con livelli di 25(OH)D al basale inferiori a 25 nmol/l e non nei pazienti che presentavano livelli più elevati di 25(OH)D in circolo³³.

Alcuni studi condotti sulle cellule mononucleari da sangue periferico (*peripheral blood mononuclear cell*, PBMC) dei pazienti affetti da asma di grado severo hanno riportato una inibizione della produzione di citochine espresse dalle cellule Th17 (IL-17 e IL-22) a seguito del trattamento con 1,25(OH)₂D₃. L'importanza di questo risultato è evidente data l'impossibilità dei corticosteroidi a inibire le citochine espresse dalle Th17, in quanto suggerisce che la vitamina possa migliorare la risposta alla terapia steroidea nei pazienti asmatici^{33,42}.

VITAMINA D E SARS-COV-2

Alla luce del ruolo protettivo della vitamina D riportato in molte condizioni associate alla polmonite, l'ipercitochinemia e la sindrome da distress respiratorio acuto (ARDS) e dunque dei suoi effetti antivirali che interferiscono direttamente con la replicazione virale, è lecito supporre che la vitamina D potrebbe avere effetti decisivi sull'infezione da SARS-CoV-2. Il virus SARS-CoV-2 inizialmente adotta meccanismi di evasione della risposta immunitaria, innescando l'iperreazione immunitaria e la tempesta citochinica^{43,46}.

Il trattamento preventivo con vitamina D ha effetti positivi documentati nei modelli animali di ARDS, riducendo la permeabilità dei polmoni, modulando l'attività del sistema renina-angiotensina e l'espressione del recettore ACE2, noto come punto d'ingresso nelle cellule umane per il virus SARS-CoV-2^{47,48}.

Pertanto andrebbe valutato l'utilizzo della vitamina D come possibile terapia adiuvante o come profilassi⁴⁹.

CONCLUSIONI

Alla luce dei diversi meccanismi attivati nella patologia respiratoria, dei diversi *pathway* che possono influenzare la capacità di un individuo di produrre adeguati livelli di vitamina D attiva a livello locale, della variabilità del siero "benefico" in ogni paziente (considerati anche gli effetti collaterali della supplementazione con vitamina D quali l'ipercalcemia, l'ipercalciuria e i calcoli renali), il messaggio principale è l'importanza di diagnosticare, prevenire e trattare la carenza di vitamina D. Queste considerazioni dovrebbero portarci a considerare la vitamina D non tanto come un fattore "universale" e indipendente dell'asma, ma come un importante "regolatore" nel nostro sistema immunitario.

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