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 Editoriale

 Vitamina D e malattie
psichiatriche:
analisi delle possibili
relazioni di causalità

 Il ruolo della vitamina D
in oncologia:
a che punto siamo?

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Pacini Editore srl

Direttore Responsabile

Patrizia Pacini

Edizione

Pacini Editore Srl

Via Gherardesca 1 • 56121 Pisa

Tel. 050 313011 • Fax 050 3130300

Info@pacineditore.it - www.pacineditore.it

Divisione Pacini Editore Medicina

Manuela Amato

Business Development Manager

Tel. 050 3130255

mamato@pacineditore.it

Redazione

Lucia Castelli

Tel. 050 3130224

lcastelli@pacineditore.it

Grafica e impaginazione

Massimo Arcidiacono

Tel. 050 3130231

marcidiacono@pacineditore.it

Stampa

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Maurizio Rossini

Dipartimento di Medicina,
Sezione di Reumatologia, Università di Verona

La vitamina D continua a sorprendere. Notate come in questo numero si passi da un possibile ruolo della vitamina D nelle malattie psichiatriche a quello in ambito oncologico. Sappiamo che la vitamina D può avere effetti pleiotropici, ma quale può essere il comune meccanismo biologico principale che li determina? L'ubiquitarità dei suoi recettori? Gli effetti sul sistema immunitario? La capacità di modulare alcune attività enzimatiche? Gli effetti genomici?

Vediamo che idea vi fate a riguardo considerati i contributi degli Autori in questo numero.

Lo scopo dichiarato dell'articolo sulla vitamina D e le malattie psichiatriche è l'identificazione della relazione di causalità, perché questa consentirebbe di poter comprendere se, e in che misura, la supplementazione di vitamina D possa prevenire l'insorgenza di disturbi mentali o ridurre la sintomatologia. Si parte quindi descrivendo innanzitutto i possibili meccanismi di azione della vitamina D a livello neurologico, prima di descrivere le attuali evidenze derivanti da studi osservazionali o di intervento in questo ambito. Si fa in particolare notare che la vitamina D è coinvolta nell'espressione regione-specifica dei recettori della vitamina D (VDR) in aree quali la corteccia cingolata, talamo, cervelletto, *substantia nigra*, nell'amigdala e nell'ippocampo, e che la maggior parte di queste regioni esprime enzimi 1 α -idrossilasi in grado di metabolizzare 25(OH)D in 1,25(OH)₂D₃: ciò significa che la vitamina D svolge anche nel cervello umano sia una funzione autocrina che paracrina, che potrebbero entrambe avere un ruolo rilevante nella neuro-immuno-modulazione o protezione e nel normale sviluppo e funzione cerebrale. Nel caso della depressione, dato il coinvolgimento della vitamina D nel controllo della risposta infiammatoria, si ipotizza che essa agisca come meccanismo di modulazione regolando la sovra espressione di citochine pro-infiammatorie associate alla depressione. Tuttavia si fa anche notare che la vitamina D è coinvolta nella regolazione dell'attività di enzimi, quali la tirosina-idrossilasi e l'enzima limitante la velocità di biosintesi di dopamina, norepinefrina ed epinefrina, tutti meccanismi che possono giustificare un'associazione positiva tra deficit di vitamina D e depressione.

Anche il collega oncologo segue la stessa strada e prima di sintetizzare le attuali evidenze cliniche descrive i possibili meccanismi d'azione biologici. Fa notare come un primo livello di interazione tra vitamina D e trasformazione o progressione neoplastica possa far riferimento alla capacità biosintetica locale da parte dell'enzima CYP27B1, la cui espressione è ridotta in alcuni tumori in maniera

Corrispondenza**Maurizio Rossini**

maurizio.rossini@univr.it

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dipendente dallo stadio e dal grado di differenziazione. In questo contesto anche variazioni nell'espressione del VDR a livello intra-tumorale possono influenzare l'aggressività biologica della neoplasia, modulando l'azione autocrina, paracrina e intracrina della vitamina D. Vi faccio notare come pertanto solo il colecalciferolo e non i vari metaboliti della vitamina D possa garantire i fisiologici e completi effetti locali a livello dei vari organi e tessuti. La potenziale azione anti-tumorale

della vitamina potrebbe inoltre esplicarsi attraverso meccanismi prevalentemente genomici, ma anche attraverso meccanismi non genomici.

Con la consueta prudenza che contraddistingue i maggiori esperti, tra cui i nostri Autori, le conclusioni relative agli effetti clinici della supplementazione con vitamina D in ambito psichiatrico e oncologico sono simili: i risultati sono tuttora contrastanti, probabilmente anche in seguito alla multifattorialità della patogenesi, alle

diverse valutazioni degli outcome (ad es. impatto sull'incidenza di tumori, rispetto al più convincente impatto sulla mortalità per tumore) e alle incertezze sulle posologie.

Mi pare comunque già consigliabile inserire la valutazione dei livelli di vitamina D anche nel percorso diagnostico di queste patologie ed evitare condizioni carenziali anche in questi pazienti.

Buona lettura!

Vitamina D e malattie psichiatriche: analisi delle possibili relazioni di causalità

VITAMIN D

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**Alessandro Cuomo, Bruno Beccarini Crescenzi,
Maria Nitti, Pietro Carmellini, Andrea Fagiolini**

Università di Siena e Azienda Ospedaliera Universitaria Senese,
Dipartimento di Salute Mentale e Organi di Senso, Siena

INTRODUZIONE

Secondo l'Organizzazione Mondiale della Sanità oltre un miliardo di persone soffre di un disturbo mentale o comportamentale. Si stima che oltre 300 milioni di persone al mondo, ossia il 4,4% della popolazione mondiale, soffrano di depressione. La schizofrenia ha un tasso di prevalenza che varia da 4 a 7 persone ogni 1000 individui, interessando pertanto circa 20 milioni di soggetti. Ne deriva che trattare un così elevato numero di persone, oltre che rappresentare un notevole sforzo economico per l'intero sistema sanitario, rappresenti una sfida per l'intero settore medico, dato l'impatto di tali patologie su diversi settori clinici.

Numerose evidenze scientifiche hanno dimostrato l'esistenza di un'associazione tra la deficienza di vitamina D e la depressione o schizofrenia. La vitamina D è presente nel cervello umano ed è stata identificata come uno dei fattori chiave nella regolazione di numerosi percorsi di neurotrasmissione, inclusi quelli di dopamina, serotonina, noradrenalina e glutammina. Studi recenti hanno dimostrato come il deficit di vitamina D sia associato a disfunzioni dell'ippocampo – regione coinvolta nella patogenesi dei disturbi mentali – e come sia positivamente correlato al volume della sostanza grigia.

Tuttavia, la relazione di causalità tra la vitamina D e i disturbi mentali rimane ancora poco chiara. Sebbene sia evidente il connubio tra una deficienza di vitamina D e i disturbi mentali maggiori, la letteratura in merito non è ancora riuscita a comprendere se la deficienza di vitamina D sia la causa, la conseguenza o il fattore concomitante del disturbo mentale osservato¹.

Lo scopo di questo studio è riassumere le principali evidenze scientifiche riguardanti l'associazione tra il deficit di vitamina D e i disturbi mentali, così da aumentare il livello di cono-

scenza dei clinici di ogni settore medico e stimolare la produzione scientifica e l'osservazione sperimentale al riguardo. L'identificazione della relazione di causalità consentirebbe infatti di poter comprendere se e in quale misura, la supplementazione di vitamina D possa prevenire l'insorgenza di disturbi mentali o ridurre la sintomatologia.

VITAMINA D: MECCANISMI DI AZIONE PER I DISTURBI MENTALI

La vitamina D è un ormone steroideo che svolge un ruolo fondamentale nel bilanciamento minerale dell'organismo, nel corretto funzionamento del sistema immunitario e nella patogenesi di vari disturbi, come il cancro e le malattie autoimmuni.

Nei disturbi psichiatrici la vitamina D stessa è coinvolta nell'espressione regione-specifica dei recettori della vitamina D (VDR) in aree quali la corteccia cingolata, il talamo, il cervelletto, la *substantia nigra*, nell'amigdala e nell'ippocampo². La maggior parte di queste regioni esprime enzimi 1 α -idrossilasi, in grado di metabolizzare 25(OH)D in 1,25(OH)₂D₃; ciò significa che la vitamina D svolge nel cervello umano sia una funzione autocrina sia una funzione paracrina.

La presenza di vitamina D, di VDR e di enzimi correlati (CYP27B1, CYP24A1) in varie regioni del cervello ha permesso di comprendere il ruolo fondamentale della vitamina D quale ormone neuroattivo/neurosteroidale nei processi di neuro-immuno-modulazione, di neuro-protezione, di sviluppo del cervello e nella normale funzione cerebrale. Il deficit di vitamina D nei primi anni di vita influenza negativamente la differenziazione neuronale, la connettività assonale, la struttura e la funzione del cervello, tutti meccanismi che spiegano l'associazione tra la deficienza di vitamina D nell'infanzia e il maggiore rischio di schizofrenia³. Allo stesso modo, sono sta-

Corrispondenza

Alessandro Cuomo

alessandrocuomo86@gmail.com

Conflitto di interessi

Andrea Fagiolini è/è stato consulente e/o relatore e/o ha ricevuto assegni di ricerca da Allergan, Angelini, Apsen, Boehringer Ingelheim, Daiichi Sankyo Brasil Farmacêutica, Doc Generici, FB-Health, Italfarmaco, Janssen, Lundbeck, Mylan, Otsuka, Pfizer, Recordati, Sanofi Aventis, Sunovion, Vifor.

Alessandro Cuomo è/è stato consulente e/o relatore e/o ha ricevuto assegni di ricerca da Angelini, Doc Generici, Janssen, Lundbeck, Mylan, Otsuka, Pfizer, Recordati, Sanofi Aventis.

Maria Nitti, Bruno Beccarini Crescenzi, Pietro Carmellini dichiarano nessun conflitto di interessi.

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te segnalate alterazioni dell'espressione del trasportatore della dopamina, espressione della catecol-O-metil transferasi neonatale e del metabolismo della dopamina, evidenze che legano la vitamina D e una sua deficienza alla schizofrenia.

Nel caso della depressione, dato il coinvolgimento della vitamina D nel controllo della risposta infiammatoria, si ipotizza che essa agisca come meccanismo di modulazione, regolando la sovra espressione di citochine pro-infiammatorie associate alla depressione e la risposta infiammatoria stessa ⁴. La vitamina D è inoltre coinvolta nella regolazione dell'attività di enzimi, quali la tirosina-idrossilasi e l'enzima limitante la velocità di biosintesi di dopamina, norepinefrina ed epinefrina, tutti meccanismi che possono giustificare un'associazione positiva tra il deficit di vitamina D e la depressione.

VITAMINA D E DEPRESSIONE

La depressione è la malattia psichiatrica più comunemente debilitante, i cui meccanismi eziopatogenetici sono patologici e correlati a molteplici aspetti della funzionalità neurale ¹. Negli anziani la depressione colpisce principalmente coloro che sono affetti da malattie croniche e da un deterioramento cognitivo, in quanto i processi legati all'invecchiamento e alla malattia cronica e i cambiamenti infiammatori a essi connessi, sia endocrini che immunitari, compromettono l'integrità dei circuiti frontostriatali, dell'amigdala e dell'ippocampo, aumentando dunque la vulnerabilità alla depressione.

L'associazione tra la scarsa esposizione al sole e i cambiamenti dell'umore è riconosciuta da oltre 2000 anni ⁵. Studi recenti hanno messo in evidenza la correlazione tra i bassi livelli ematici di vitamina D e l'aumento del rischio di depressione dall'8 al 14%, con una percentuale di rischio di suicidio pari al 30% ^{6,8}, senza una particolare differenza tra la popolazione dei giovani adulti e degli anziani ⁷.

Allo stesso modo, Sherchand et al. (2018), nell'analizzare la relazione tra bassi livelli di vitamina D e il rischio di depressione hanno dimostrato una probabilità 3,8 volte maggiore di sviluppare depressione negli individui con un deficit di vitamina D rispetto a quelli con dei normali livelli di vitamina D ⁹. Il *Third National Health and Nutrition Examination Survey*, studiando un campione di 7.970 individui di età compresa tra 15 e 39 anni, ha identificato un più alto rischio di sviluppo di depressione nei pazienti con livelli

di vitamina D < 50 nmol/L rispetto a quelli con livelli di vitamina D > 75 nmol/L ⁶.

Inoltre, l'ipovitaminosi D sembra essere connessa allo sviluppo della depressione post-partum ^{10,11}. Studi recenti hanno infatti evidenziato una correlazione negativa tra i bassi livelli di vitamina D durante il primo trimestre di gravidanza e lo sviluppo di sintomi depressivi durante il secondo trimestre ^{12,13}; allo stesso modo è stata dimostrata una correlazione tra l'ipovitaminosi D nel secondo trimestre e un più alto rischio di sviluppo di sintomi depressivi peri-partum sei mesi dopo il parto ¹⁴.

Un'altra importante osservazione riguarda l'associazione tra il deficit di vitamina D e lo sviluppo di depressione in età tardiva e nelle popolazioni geograficamente più a nord ¹⁵. A tal proposito, uno studio condotto su 1.282 pazienti olandesi (di età compresa tra i 65 e i 95 anni) ha osservato una deficienza di vitamina D nel 14% dei pazienti depressi; percentuali nettamente inferiori di ipovitaminosi D sono state evidenziate nel gruppo di controllo ⁷.

In uno studio trasversale, condotto su 80 pazienti anziani (di età compresa fra i 60 e i 92 anni), di cui 40 affetti da demenza di Alzheimer e 40 senza alcuna forma di demenza, l'ipovitaminosi D è stata riscontrata in circa il 58% del campione (Wilkins, 2006).

Infine, i pazienti affetti da malattie croniche come la fibromialgia o quelli affetti da depressione in comorbidità presentano una più alta incidenza di deficit o un'insufficienza di vitamina D (*Hospital and Anxiety Depression Scale* = 31) ¹⁶.

VITAMINA D E SCHIZOFRENIA

La schizofrenia è un disturbo mentale dal decorso cronico, caratterizzato da un anomalo comportamento sociale e con un grave *impairment* sul contenuto del pensiero e sul piano comportamentale dell'individuo affetto. La schizofrenia riconosce una patogenesi multifattoriale: la vulnerabilità genetica, le alterazioni del neuro sviluppo, le infezioni virali, il fumo, il coefficiente intellettivo, l'uso di cannabis o i traumi infantili rappresentano degli importanti fattori di rischio.

Secondo una recente meta-analisi, il rischio di sviluppare schizofrenia è 2,14 volte maggiore negli individui con un deficit di vitamina D rispetto agli individui con dei normali livelli ematici ¹⁷. Si riconoscono alcuni fattori di rischio ambientali, quali la stagionalità di nascita, il luogo di nascita, la latitudine e

la migrazione, che legano la deficienza di vitamina D alla schizofrenia ^{3,18}.

In particolare, secondo una revisione eseguita su 86 studi, per un totale di 437.710 individui, il rischio di schizofrenia è risultato del 5-8% più alto nei soggetti nati tra dicembre e maggio, con un picco di maggiore vulnerabilità nei soggetti nati tra gennaio e febbraio, data la minore presenza di raggi UVB idonei a stimolare la produzione di vitamina D ¹⁹.

In uno studio condotto su 424 pazienti schizofrenici e su altrettanti controlli selezionati per età, genere e data di nascita, i pazienti sono stati suddivisi in quintili, a seconda dei livelli di vitamina D misurati entro un anno dalla nascita; negli individui appartenenti ai due quintili più bassi e al quintile più alto, è stato riscontrato un rischio maggiore (il doppio) di sviluppo di schizofrenia rispetto ai soggetti classificati secondo gli altri quintili ³. Analogamente, uno studio condotto su una coorte di 12.058 bambini finlandesi, ha dimostrato che la supplementazione di vitamina D durante il primo anno di vita si associa a una riduzione significativa del rischio di sviluppo di schizofrenia in età adulta, specialmente nei maschi ²⁰.

A corroborare tali ipotesi, in uno studio recente condotto da Okasha et al. (2020) su 20 pazienti schizofrenici e 20 pazienti di controllo, è stato osservato che i livelli di vitamina D risultano essere statisticamente più bassi nei pazienti con schizofrenia (55%) rispetto ai pazienti non schizofrenici ²¹.

Inoltre, il deficit di vitamina D sembra essere correlato con un più alto rischio di sintomi psicotici isolati. In uno studio svedese, condotto su 33.623 donne, è stata evidenziata una significativa associazione tra i bassi livelli di vitamina e lo sviluppo di sintomi psicotici isolati, permettendo dunque di ipotizzare che l'ipovitaminosi D rappresenti un possibile fattore rischio per lo sviluppo di psicosi in età adulta ²².

SUPPLEMENTAZIONE DI VITAMINA D

La supplementazione di vitamina D potrebbe rappresentare un approccio valido per il miglioramento dei sintomi depressivi e psicotici. In un trial clinico randomizzato, condotto in doppio cieco su 441 soggetti (di età compresa fra i 21 e i 70 anni) suddivisi in tre gruppi e sottoposti per un anno a un trattamento con dosi di 20.000 UI (primo gruppo) e 40.000 UI (secondo gruppo) di vitamina D o con placebo (terzo gruppo), è stato riscontrato un miglioramento dei valori

della *Back Depression Inventory Scale* (BDI) dopo un anno di trattamento²³. In un trial clinico randomizzato di 8 settimane, condotto su 42 pazienti affetti da un disturbo depressivo maggiore, l'uso concomitante di fluoxetina (20 mg) e vitamina D (1500 IU) ha condotto al conseguimento di un miglioramento significativo dei sintomi depressivi rispetto al gruppo fluoxetina-placebo, dopo appena quattro settimane di trattamento²⁴. Tuttavia, i risultati sull'efficacia della supplementazione appaiono ancora contrastanti. Secondo le meta-analisi condotte da Gowda et al. (2015) e da Shaffer (2014), la supplementazione di vitamina D non produrrebbe in generale alcun miglioramento dei sintomi depressivi, salvo un moderato effetto nei pazienti con dei sintomi depressivi clinicamente significativi^{25,26}.

Nel caso della schizofrenia, un trial clinico randomizzato open label condotto da Sheikhmoonesi (2016) su un campione di pazienti schizofrenici trattati con supplementi di vitamina D in aggiunta alla terapia standard di base, non ha evidenziato alcun cambiamento dei sintomi²⁷.

CONCLUSIONE

Dalla breve revisione narrativa condotta emergono molteplici spunti di riflessione. Numerosi studi hanno messo in evidenza la relazione tra l'ipovitaminosi D durante lo sviluppo embrionale e nell'età infantile e l'insorgenza di schizofrenia in età adulta, sebbene questa correlazione non abbia ancora trovato definitivi consensi. Per quanto concerne la depressione, la relazione resta non del tutto chiara e comunque di tipo multifattoriale. Oltre ai meccanismi di azione che legano la carenza di vitamina D ai meccanismi biologici tipici della depressione, altri fattori di tipo comportamentale rendono la comprensione della relazione ancora più complessa. Da un lato la depressione è associata a una riduzione dell'attività fisica all'aperto, con conseguente riduzione dell'assorbimento di luce solare. Dall'altro, i sintomi del deficit di vitamina D, quali senso di fatica e dolore, sono di per sé elementi in grado di generare un abbassamento dell'umore e quindi un conseguente stato depressivo.

Sono necessarie ulteriori ricerche per definire dei protocolli appropriati per il test e l'integrazione della vitamina D nella pratica clinica e per stabilire se, quando e quanto l'integrazione di vitamina D può migliorare il decorso della depressione o ridurre l'inci-

denza della schizofrenia. Per i clinici è consigliabile inserire la valutazione dei livelli di vitamina D nel proprio percorso decisionale-diagnostico: dall'analisi di tali livelli è possibile prevedere con un buon margine di probabilità le possibili conseguenze psicofisiche connesse ai bassi livelli di vitamina D e dunque prevenirle.

Stili di vita sani che comprendono l'alimentazione con cibi ad alto contenuto di vitamina D, integratori alimentari per ridurre la deficienza o l'insufficienza e un aumento dell'esposizione alla luce solare sono indicazioni indispensabili per migliorare il benessere mentale e devono rientrare nell'ordinario percorso educativo di ogni paziente, a prescindere dalla specifica condizione patologica.

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Il ruolo della vitamina D in oncologia: a che punto siamo?

VITAMIN D

UpDates

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Michele Milella

Sezione di Oncologia, Dipartimento di Medicina, Università di Verona – Scuola di Medicina e Azienda Ospedaliera Universitaria Integrata (AOUI) di Verona

INTRODUZIONE

Studi preclinici in modelli *in vitro* e *in vivo* dimostrano che la vitamina D (vitD) è in grado di inibire la trasformazione e progressione neoplastica, attraverso l'induzione della differenziazione cellulare, l'inibizione della proliferazione del clone neoplastico e molteplici altre attività biologiche di tipo anti-infiammatorio, immunomodulante, pro-apoptotico e anti-angiogenico.

Da un punto di vista clinico, i livelli circolanti di vitD e dei suoi metaboliti attivi sono stati collegati a una maggiore sopravvivenza dei pazienti oncologici e numerosi studi randomizzati sono stati condotti, sebbene con risultati contrastanti, sul possibile impatto della supplementazione di vitD sull'incidenza, sulla mortalità e sulla sopravvivenza per cancro nell'uomo.

Questa breve revisione della letteratura si propone l'obiettivo di fare il punto sui più recenti dati preclinici e clinici e sul possibile ruolo della vitD in ambito oncologico.

MECCANISMI BIOLOGICI D'AZIONE

La vitD, prodotta per conversione del 7-deidro-colesterolo a opera delle radiazioni UV a livello cutaneo, è il precursore del potente ormone multifunzionale calcitriolo [1,25-diidrossi-vitamina D₃ (1,25(OH)₂D₃)], prodotto per di-idrossilazione a livello epatico e renale a opera del citocromo P450¹⁻⁴. Attraverso il legame con il suo recettore (VDR), il calcitriolo regola, direttamente o indirettamente, il 3-5% del genoma umano.

Un primo livello di interazione tra la vitD e la trasformazione e progressione neoplastica fa riferimento alla capacità biosintetica locale da parte dell'enzima CYP27B1, la cui espressione è ridotta in alcuni tumori in maniera dipendente dallo stadio e dal grado di differenziazione; in questo contesto, anche le variazioni nell'espressione del VDR a livello intra-tumorale possono influenzare l'aggressività biologica della neoplasia, modulando l'azione autocrina, paracrina e intracrina della vitD¹⁻⁴.

La potenziale azione anti-tumorale della vitD si esplica attraverso meccanismi prevalentemente genomici, ma anche attraverso dei meccanismi non genomici, che coinvolgono, ad esempio, il VDR e la *endoplasmic reticulum stress protein 57* (ERP57; 5). Le azioni genomiche della vitD coinvolgono la modulazione di una vasta serie di mediatori, che regolano *pathways* di proliferazione, apoptosi, e differenziazione delle cellule tumorali. Ad esempio, nelle tre neoplasie con maggior evidenza, anche clinica, di potenziale sensibilità agli effetti anti-neoplastici di vitD/VDR (carcinoma mammario, prostatico e coloretale), tale azione si esplica attraverso la modulazione dei *pathways* proliferativi regolati da estrogeni, androgeni e dal sistema WNT/ β -catenin, rispettivamente sia a livello di popolazioni cellulari tumorali parzialmente differenziate, che di popolazioni neoplastiche staminali (CSC). Inoltre, la segnalazione attraverso l'asse vitD/VDR può influenzare in senso anti-tumorale l'interazione tra le cellule tumorali e il microambiente circostante (*tumor microenvironment*, TME); tale azione si esplica attraverso la modulazione della capacità invasiva e metastatica, l'inibizione dei *pathways* pro-infiammatori e pro-angiogenetici¹⁻⁴. Tra i meccanismi molecolari implicati nella regolazione delle attività anti-tumorali della vitD si può ipotizzare un ruolo bi-direzionale di un ampio pannello di micro-RNA (miRNA), che da un lato sono regolati dal sistema vitD/VDR, mediandone a valle gli effetti anti-tumorali, e dall'altro possono regolare l'espressione di VDR e CYP24A1, modulando la sensibilità delle cellule tumorali all'azione di vitD¹⁻⁴.

Nella maggior parte, sebbene non in tutti gli studi in modelli animali, la supplementazione dietetica di vitD e/o la somministrazione di calcitriolo e di suoi analoghi ritarda la trasformazione e inibisce la progressione neoplastica; tali modelli includono quelli di progressione di lesioni pre-neoplastiche, xenotrapianti di tumori umani, modelli di cancerogenesi spon-

Corrispondenza

Michele Milella

michele.milella@univr.it

Conflitto di interessi

Michele Milella dichiara nessun conflitto di interessi.

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tanea o indotta dall'alimentazione, modelli di cancerogenesi chimica o indotta da cancerogeni noti e modelli transgenici di sviluppo tumorale¹⁴.

EVIDENZE CLINICHE

Sebbene le evidenze dagli studi epidemiologici e dagli studi clinici randomizzati non abbiano documentato in maniera conclusiva un impatto clinicamente rilevante dei livelli di vitD sugli esiti oncologici più significativi, i dati a oggi disponibili indicano complessivamente un effetto maggiore sulla mortalità per cancro piuttosto che sulla sua incidenza, suggerendo un possibile effetto biologico sui meccanismi di progressione/promozione piuttosto che su quelli di trasformazione/iniziazione neoplastica. Tali evidenze (brevemente ricapitolate di seguito) collocherebbero gli interventi basati sulla supplementazione dietetica o sulla somministrazione farmacologica di vitD, calcitriolo e molecole correlate nell'ambito concettuale della chemioprevenzione.

Impatto sull'incidenza di tumori

In tre recenti revisioni sistematiche della letteratura con metanalisi dei dati cumulati⁶⁻⁸ (Tab. I) il rischio relativo (RR) di sviluppare una patologia neoplastica maligna, nel gruppo sottoposto a intervento con supplementazione con vitD, varia dallo 0,98 allo 1,03, senza una significativa eterogeneità; tali dati, come del resto i risultati dei tre singoli studi più importanti (RECORD, ViDA e VITAL⁹⁻¹¹), non suppor-

tano un'associazione significativa tra una supplementazione di vitD e l'incidenza del cancro. In generale, negli studi analizzati, non vi è evidenza di un effetto differenziale in particolari sottogruppi.

Impatto sulla mortalità per tumore

Sebbene nei singoli studi non sempre la riduzione della mortalità per cancro abbia raggiunto la significatività statistica, in tre dei quattro studi principali^{9,11,12} vi è una sorprendente uniformità nella stima della riduzione del rischio di morte per cancro, che varia dal 14 al 18%, con l'eccezione dello studio ViDA¹⁰, nel quale tale riduzione è minima (7%). Conseguentemente, le quattro metanalisi disponibili^{6-8,13} documentano un RR di mortalità per cancro variabile dallo 0,85 allo 0,88, in favore dell'intervento basato sulla supplementazione di vitD, senza una significativa eterogeneità, che raggiunge la significatività statistica in 3 studi di metanalisi (Tab. I)^{6,7,13}. Alcune analisi di sottogruppo indicano una maggiore probabilità di vantaggio in termini di riduzione di mortalità, per gli studi che hanno incluso soggetti di entrambi i sessi e senza storia precedente di cancro, per gli studi che hanno utilizzato una somministrazione quotidiana di vitD e per gli studi con dosi di vitD relativamente basse e che hanno raggiunto livelli di 25(OH)D circolanti < 100 nmol/L⁶⁻⁸. Un'ulteriore analisi di sottogruppo suggerisce che la riduzione della mortalità per cancro è ristretta agli interventi che utilizzano vitD₃, ma non è evidente per gli interventi che utilizzano vitD₂¹³.

INTERPRETAZIONE DEI DATI DISPONIBILI E LINEE DI SVILUPPO FUTURE

Le evidenze cliniche precedentemente citate suggeriscono, come già accennato, un effetto prevalente della vitD sui meccanismi di progressione/promozione, piuttosto che su quelli di trasformazione/iniziazione neoplastica. In questo senso vanno anche i risultati di una sottoanalisi dello studio VITAL, che indicano una riduzione significativa nell'incidenza di cancro avanzati (metastatici o fatali, hazard ratio – HR – 0,83, 95% CI 0,69-0,99, P = 0,04) nel gruppo trattato con vitD, particolarmente nel sottogruppo di soggetti con *body mass index* normale (P per l'interazione = 0,03)¹⁴. In linea con questi risultati, il primo studio randomizzato, condotto in pazienti con neoplasia coloretta avanzata in trattamento chemioterapico, ha rilevato un *trend* in favore delle alte dosi di vitD₃ rispetto alle dosi standard, con un vantaggio di circa 2 mesi nella mediana di sopravvivenza libera da progressione (PFS; 13 vs 11 mesi, *log-rank* P = 0,07) e un HR in analisi multivariata di 0,64 (1-sided 95% CI, 0-0,90; P = 0,02)¹⁵.

Va, infine, ricordato l'impatto tutt'altro che trascurabile della supplementazione di vitD nel contesto della prevenzione delle complicanze scheletriche e della palliazione dei sintomi in fasi avanzate di malattia^{16,17}.

Nonostante l'interesse e il notevole numero di studi sia preclinici che clinici sinora riportati, lacune importanti rimangono nelle conoscenze relative al potenziale effetto della vitD nel ridurre la progressione tumorale e

Tabella I. Principali metanalisi condotte sull'impatto di vitD sull'incidenza e mortalità per cancro negli anni recenti.

Incidenza

Autore	N. trial	N. pazienti	Casi (vitD)	Casi (cont)	RR	95% IC	P	Eterogeneità	Ref
Zhang et al.	10	81.362	3716 (9,16%)	3799 (9,26%)	0,99	0,94-1,03	0,532	No	6
Keum et al.	10	-	6.537		0,98	0,93-1,03	0,420	No	7
Goulão et al.	24	18.440	540 (5,66%)	521 (5,85%)	1,03	0,91-1,15	n.s.	No	8

Mortalità

Autore	N. trial	N. pazienti	Casi (vitD)	Casi (cont)	RR/HR	95% IC	P	Eterogeneità	Ref
Zhang et al.	7	77.653	821 (2,11%)	942 (2,43%)	0,87	0,79-0,95	0,003	No	6
Keum et al.	5	-	1.591		0,87	0,79-0,96	0,005	No	7
Goulão et al.	7	11.202	150 (2,67%)	170 (3,04%)	0,88	0,70-1,09	n.s.	No	8
Zhang et al.	5	39.197	397 (2,02%)	468 (2,39%)	0,85	0,74-0,97	0,01	No	13

la mortalità per cancro¹⁸. Da un punto di vista preclinico la letteratura recente dimostra un ruolo importante di vitD nel revertire fenomeni di *multidrug resistance*, attraverso l'interferenza con i meccanismi di transizione epitelio-mesenchimale (EMT), che supportano la resistenza farmacologica e favoriscono la diffusione metastatica, e attraverso la modulazione di specifici miRNA legati alla progressione neoplastica¹⁹, suggerendone quindi l'uso in contesti di malattia avanzata e in combinazione con altre strategie terapeutiche. Da un punto di vista clinico, tuttavia, ulteriori studi sono necessari non solo per confermare l'effetto di riduzione della mortalità per cancro, ma soprattutto per chiarire il potenziale ruolo di vitD in tumori di specifici distretti anatomici, le possibili interazioni con specifiche alterazioni genetiche tumorali *driver*, la possibile modulazione degli effetti protettivi in specifici contesti genetici individuali (ad esempio polimorfismi di VDR)¹⁸, al fine di ricondurre l'utilizzo terapeutico o preventivo di vitD a un contesto di Oncologia di precisione.

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