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Vitamina D nelle malattie cardiovascolari

Vitamina D e disturbi mentali: *update* sulle ultime evidenze e focus su autismo e anoressia

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EDITORIALE

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Cari Lettori,
in questo numero troverete un aggiornamento relativo alla discussione sul possibile ruolo della vitamina D nelle malattie cardiovascolari e in alcuni disturbi mentali, grazie ai preziosi contributi di esperti Autori.
Noterete come entrambi riconoscano la persistente discrepanza tra i risultati degli studi osservazionali e quelli di alcuni trial d'intervento o la carenza di questi ultimi. Come sapete gli studi osservazionali sono a rischio di fattori confondenti, come la "reverse causality", in particolare per quelli sulla vitamina D, la cui carenza, considerati il meccanismo di sintesi endogena e il suo metabolismo, può essere la conseguenza e non la causa di uno stato di malattia. Questo rischio può oggi essere attenuato da nuove metodiche, come la randomizzazione mendeliana, che prevede l'uso di varianti alleliche di uno o più geni coinvolti nella codifica di un certo biomarker. In studi osservazionali che utilizzano questa metodica in una popolazione osservata e seguita nel tempo per valutare l'incidenza di determinati eventi, si confrontano i soggetti con una o più varianti geniche che determinano livelli sierici maggiori o minori nel nostro caso di 25(OH)D, simulando quindi un trial d'intervento controllato e randomizzato (RCT) con vitamina D, difficile da realizzare per motivi economici ma anche direi etici. Come vedrete in questo numero gli studi sinora condotti con questa metodica supportano il rapporto causa/effetto della correlazione tra carenza di vitamina D e mortalità o morbilità.
Recentemente sono stati pubblicati i risultati di un altro approccio che secondo me, come una sorta di "controprova", può contribuire a supportare ulteriormente un beneficio clinico extra scheletrico della supplementazione con vitamina D.
Come riportato in precedenza [1] e commentato anche in questa rivista [2], lo studio randomizzato VITAL, progettato principalmente per studiare gli effetti della supplementazione di vitamina D e omega-3 sul cancro incidente e sulle malattie cardiovascolari, ha dimostrato che 5 anni di supplementazione di vitamina D sono associati a una riduzione del 22% del rischio di malattie autoimmuni. Ora i ricercatori Karen H. Costenbader et al. hanno riportato che tra i 21.592 partecipanti allo studio VITAL che hanno accettato di essere seguiti per altri 2 anni dopo la sospensione della supplementazione con 2000 UI/giorno di colecalciferolo, la protezione contro le malattie autoimmuni non è più statisticamente significativa [3]. Quindi l'interruzione della supplementazione con vitamina D si associa a una ripresa del rischio di malattie autoimmuni. I risultati dell'estensione dello studio VITAL confermano secondo me innanzitutto che la correlazione tra supplementazione di vitamina D e riduzione del rischio di incorrere in malattie autoimmuni non era casuale e suggeriscono che l'integrazione di vitamina D dovrebbe essere somministrata su base continuativa per la prevenzione a lungo termine di malattie autoimmuni, anche perché il rischio di tornare in condizioni di carenza non è oggi improbabile. Nel background della Nota 96 [4] dell'Agenzia Italiana del Farmaco in

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relazione ai risultati dello studio VITAL viene riportato questo commento: "secondo i risultati ottenuti sarebbero stati necessari 2000 anni/persona di supplementazione con vitamina D per evitare un caso tra le 32 diagnosi di malattia autoimmune". Io credo che se si esprimesse più correttamente il beneficio in termini di persone da supplementare/anno risulterebbe proponibile e *cost/effective* un intervento supplementare di popolazioni a rischio perché si ridurrebbe in maniera significativa l'incidenza di malattie autoimmuni di rilevante impatto in termini di disabilità, di mortalità e di costi sanitari e sociali.

Cosa ne pensate?

Buona lettura

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Vitamina D nelle malattie cardiovascolari

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INTRODUZIONE

Il ruolo della vitamina D nell'ambito del metabolismo calcio-fosforico e la sua importanza fondamentale per la crescita e per il mantenimento dell'integrità dello scheletro nel corso dell'intera vita sono stati da tempo riconosciuti. In aggiunta a questo, e già da molti anni ormai, una considerevole mole di evidenze di tipo sperimentale, clinico ed epidemiologico ha messo in luce altre importanti funzioni del sistema biologico della vitamina D in relazione al differenziamento e alla crescita cellulare, alla modulazione della risposta immunitaria, al controllo dell'attività di altri sistemi ormonali e, non da ultimo, alla capacità di interferire con i principali fattori di rischio cardiometabolico e di influenzare lo sviluppo e la progressione di numerosi disordini cardiovascolari [1]. In una precedente rassegna pubblicata su questa stessa rivista nel 2019 sono stati ampiamenti discussi la composizione e le funzioni del sistema biologico della vitamina D, i criteri di misurazione e di valutazione dello stato nutrizionale della vitamina, e i risultati di numerosi studi sulle possibili relazioni tra stato nutrizionale della vitamina D e alterazioni metaboliche e cardiovascolari, con una discussione delle possibili connessioni fisiopatologiche [2]. Negli ultimi anni a partire da quella data la ricerca clinica ed epidemiologica si è impegnata sia nell'ottenere ulteriori conferme di quanto osservato attraverso i precedenti studi clinici e osservazionali, sia soprattutto nel tentativo di dimostrare l'eventuale ruolo "causale" della carenza di vitamina D rispetto alle suddette condizioni patologiche attraverso trial controllati e randomizzati di elevata qualità scientifica. La presente rassegna si propone pertanto di mettere selettivamente a fuoco i risultati di questi ultimi studi e di discutere le basi scientifiche di un impiego della supplementazione della vitamina D a scopo profilattico o terapeutico.

RISULTATI DEI PIÙ RECENTI STUDI OSSERVAZIONALI

La Tabella I riporta in forma sintetica i dati essenziali forniti dalle più recenti pubblicazioni che si riferiscono a studi di tipo osservazionale: essa comprende uno studio prospettico su un ampio campione di popolazione americana, due studi di randomizzazione mendeliana e un notevole numero di meta-analisi di studi prospettici, la maggior parte delle quali concentrata sulla mortalità totale e cardiovascolare o su altri *outcome* cardiovascolari. Lo studio prospettico di Wan et al. [3], eseguito su un campione piuttosto numeroso di pazienti diabetici estratti dalla popolazione dello studio NHANES (*National Health and Nutrition Examination Survey*), con un lungo follow-up e un considerevole numero di eventi, ha evidenziato, come molti studi osservazionali precedenti, una forte e statisticamente significativa associazione inversa tra il livello plasmatico basale di 25(OH)D e il rischio di morte per cause cardiovascolari e per tutte le cause. Gli studi di Heath et al. [4], Gholami et al. [5] e Jani et al. [6] sono tutti meta-analisi di studi prospettici condotti prevalentemente su campioni di popolazione generale: di essi lo studio di Gholami et al. è il più selettivo avendo escluso i non pochi studi condotti su partecipanti affetti già in sede basale da patologie cardiometaboliche o di altro tipo che potessero favorire il fenomeno della "*reverse causation*", per il quale più bassi livelli di vitamina D sarebbero non già la causa della malattia ma una sua conseguenza dovuta a minore possibilità di esposizione ai raggi solari e/o a carenze nutrizionali. Di fatto, in tutte e tre le meta-analisi è stata riscontrata in modo consistente un'associazione inversa tra valori basali di 25(OH)D e l'*outcome* primario dello studio, che era la mortalità totale per lo studio di Heath et al., la mortalità cardiovascolare per quello di Gholami et al., e l'incidenza di un primo evento o di eventi cardiovascolari ricorrenti per lo studio di Jani et al. La meta-analisi di Wang et al. [7] mette a fuoco invece gli studi prospettici eseguiti su campioni di pazienti affetti da insufficienza cardiaca: il numero di tali studi è relativamente piccolo

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TABELLA I.
Vitamina D, *outcome* cardiovascolari e mortalità: risultati dei più recenti studi osservazionali.

Autore ref.	Tipo di studio	Caratteristiche	Risultati principali
Wan et al., 2021 [3]	Prospettico	6.329 adulti diabetici (NHANES III e NHANES 2001-2014), 55.126 anni-persona di follow-up, 2.056 eventi	Associazione inversa tra concentrazione basale di 25(OH)D, mortalità totale e mortalità CV. *Multivariate-adjusted* HR per valori di 25(OH)D rispettivamente < 25,0, 25,0-49,9, 50,0-74,9, ≥ 75,0 nmol/L = 1,00 (ref.), 0,70, 0,56, 0,59 per la mortalità totale (p-*trend* 0,003) e 1,00 (ref.), 0,62, 0,46, 0,50 per la mortalità CV (p-*trend* 0,02)
Heath et al., 2019 [4]	Meta-analisi di studi prospettici	54 studi (n = 812.646)	Associazione inversa tra livelli basali di 25(OH)D e mortalità per tutte le cause, di tipo non lineare con un *plateau* per valori compresi tra 75 e 90 mmol/L
Gholami et al., 2019 [5]	Meta-analisi di studi prospettici	25 studi (n = 98.171), 10.099 eventi CV	Associazione inversa tra livelli basali di 25(OH)D e rischio CV. Nel confronto tra valori < 30 e valori > 50 nmol/L: RR = 1,54 (95% IC: 1,29-1,84) per la mortalità e RR = 1,18 (95% IC: 1-1,39) per l'incidenza
Jani et al., 2021 [6]	Meta-analisi di studi prospettici	79 studi (n = 1.397.831), 46.713 eventi CV	Associazione inversa di tipo lineare tra livelli basali di 25(OH)D e rischio CV. Nel confronto tra categoria più bassa e categoria più alta di 25(OH)D: RR = 1,34 (95% IC: 1,26-1,43, p < 0,001) per l'incidenza di un nuovo evento e RR = 1,86 (95% IC: 1,46-2,36, p < 0,001) per eventi ricorrenti
Wang X et al., 2022 [7]	Meta-analisi di studi prospettici o retrospettici	7 studi (n = 5.941 pazienti con insufficienza cardiaca), follow-up 1-5 anni	Nel confronto tra categoria più bassa e categoria più alta di 25(OH)D: RR = 1,37 (95% IC: 1,13-1,66, p = 0,002) per la mortalità totale e RR = 1,38 (95% IC: 0,87-2,19) per la frequenza di re-ospedalizzazione
Kong et al., 2023 [8]	Meta-analisi di studi prospettici	19 studi (n = 41.916), 3.015 eventi CV fatali e morti improvvise, follow-up 2-14 anni	Associazione inversa tra livelli basali di vitamina D circolante e rischio di morte CV o morte improvvisa in un intervallo tra 10 e 100 nmol/L. Nel confronto tra categoria più bassa e categoria più alta di 25(OH)D: HR (95% IC) 1,75 (1,49-2,06)
Jayedi et al., 2023 [9]	Meta-analisi di studi prospettici	21 studi in pazienti diabetici	Nel confronto con la categoria più alta (> 50 nmol/L) di 25(OH)D: RR = 1,36 (95% IC: 1,23, 1,49) per la categoria 25 - < 50 nmol/L e RR = 1,58 (1,33-1,83) per la categoria < 25 nmol/L, per la mortalità totale. Risultati simili per morbilità e mortalità CV. L'analisi dose-risposta indica un'associazione inversa non lineare, con valore minimo di rischio a 25(OH)D ~60 nmol/L per mortalità totale e mortalità CV
Vergatti et al., 2023 [10]	Meta-analisi di studi prospettici	4 studi (n = 7.717 pazienti con ictus), 496 casi di nuovo episodio ictale, follow-up 3-86 mesi	Associazione inversa non-lineare tra livelli di 25(OH)D in occasione del primo ictus e incidenza di nuovo episodio ictale con rischio più basso per un valore di 28 ng/mL. Nel confronto con la categoria più bassa di 25(OH)D: RR = 0,20 (95% IC: 0,10-0,67, p < 0,001) per la categoria più alta
Sutherland et al., 2022 [11]	Studio di randomizzazione mendeliana	N = 307.601 partecipanti della UK Biobank (età 37-73 anni) con valori di 25(OH)D misurati e predetti sulla base di 35 varianti genetiche, follow-up 14 anni e 18.700 eventi fatali	Associazione inversa L-*shaped* tra 25(OH)D predetta geneticamente e mortalità totale e CV (p = 0,033) con ripido calo del rischio di morte per concentrazioni crescenti fino a 50 nmol/L. Incremento della mortalità totale nell'analisi genetica del 25% (95% IC = 16-35) per i partecipanti con 25(OH)D misurata pari a 25 nmol/L in confronto a quelli con 50 nmol/L
Zhou et al., 2022 [12]	Studio di randomizzazione mendeliana	N = 295.788 partecipanti della UK Biobank con valori di 25(OH)D misurati e predetti sulla base di 35 varianti genetiche, follow-up 14 anni e 44.519 casi incidenti di malattia CV	Associazione inversa L-*shaped* tra 25(OH)D predetta geneticamente e incidenza di eventi CV, con ripido calo iniziale del rischio per concentrazioni crescenti di vitamina D e *plateau* a circa 50 nmol/L

CV: cardiovascolare; IC: intervallo di confidenza; RR: rischio relativo; HR: *hazard ratio*.

(n = 7), ma il numero totale di pazienti discretamente numeroso (circa 6.000), con un follow-up tra 1 e 5 anni: lo studio ha rilevato una significativa relazione inversa tra i livelli basali di 25(OH)D e mortalità o rischio di riospedalizzazione per insufficienza cardiaca e/o sue complicanze. La meta-analisi di Kong et al. [8] ha valutato la relazione tra i livelli basali di 25(OH)D e rischio di eventi cardiovascolari fatali o morte improvvisa in 19 studi, con oltre 40 mila partecipanti e oltre 3.000 eventi in un periodo di 2-14 anni: anche in questo caso la relazione oggetto dello studio è risultata di tipo inverso, lungo un ampio range di concentrazioni di 25(OH)D, con un incremento del rischio del 75% nel confronto tra livelli < 10 e livelli > 100 nmol/L. La meta-analisi di Javedi et al. [9] ha considerato invece soltanto gli studi prospettici condotti su pazienti diabetici, dimostrando anche in questa categoria di pazienti un'associazione inversa tra livelli basali di 25(OH)D e mortalità per tutte le cause, con un *plateau* a circa 60 nmol/L e un rischio aumentato del 36% per valori compresi tra 25 e 50 nmol/L e del 56% per valori < 25 nmol/L. I risultati erano simili per la morbilità e la mortalità cardiovascolari. Infine, la meta-analisi di Vergatti et al. [10] ha preso in esame 4 studi condotti su circa 8.000 pazienti che avevano subito un ictus cerebrale, con un follow-up compreso fra 3 e 86 mesi, e 496 casi di nuovo episodio ictale. Lo studio ha evidenziato un effetto protettivo di livelli più alti di 25(OH)D basali con una riduzione del rischio di recidiva pari all'80% nella categoria più alta (> 28 nmol/L) in confronto a quella più bassa di vitamina D.

Le ultime due pubblicazioni incluse in Tabella I nel novero degli studi "osservazionali" sono due studi di randomizzazione mendeliana, condotti peraltro da due gruppi indipendenti di autori a partire da una singola popolazione. Occorre premettere che la randomizzazione mendeliana è una metodica che fa in qualche modo da ponte tra la categoria degli studi osservazionali e quella dei trial di intervento controllati e randomizzati: attraverso l'uso delle varianti alleliche di uno o più geni coinvolti nella codifica di una certa proteina, consente di acquisire elementi robusti di evidenza riguardo la possibilità di relazioni causali tra determinati fattori di rischio e *outcome* clinici di interesse. Il principale vantaggio di questa metodica è la sua capacità di neutralizzare in buona misura l'effetto dei fattori confondenti che affliggono i classici studi di osservazione e, in particolare, ridurre il rischio di "*reverse causality*". Nella pratica, contrapponendo, nell'ambito di una popolazione osservata e seguita nel tempo, i soggetti con una o più varianti geniche, che determinano rispettivamente livelli più alti o più bassi di una certa sostanza – nel nostro caso la 25(OH)D –, essa permette di confrontare nei due gruppi l'incidenza di determinati eventi al pari di quanto realizzato attraverso un RCT, ma con costi e fatica molto minori. Gli studi di Sutherland et al. [11] e di Zhou et al. [12] hanno avuto come oggetto la stessa popolazione di circa 300.000 partecipanti della UK Biobank, con valori di 25(OH)D misurati e predetti sulla base di 35 varianti genetiche e un follow-up di 14 anni. La differenza tra i due studi è nell'*outcome* costituito nel primo caso dalla mortalità totale e cardiovascolare e nel secondo dai casi incidenti di malattia cardiovascolare. In entrambi gli studi è stata rilevata una significativa associazione inversa di tipo L-*shaped* (non lineare) tra 25(OH)D predetta geneticamente e i rispettivi *outcome*, con un ripido calo del rischio di mortalità e morbilità per concentrazioni crescenti fino a 50 nmol/L, dove si osserva un *plateau*, in modo non dissimile dai tradizionali studi di osservazione.

RISULTATI DEI TRIAL DI INTERVENTO PIÙ RECENTI

La Tabella II riporta i dati essenziali dei trial d'intervento randomizzati e controllati che hanno testato l'efficacia della supplementazione di vitamina D in vari tipi di popolazione: essa include un singolo RCT e una serie di meta-analisi di RCT prevalentemente, ma non esclusivamente, orientate alla valutazione degli effetti della supplementazione sulla mortalità e la morbilità cardiovascolari.

Lo studio di Virtanen et al. [13] ha testato l'efficacia di 1.600 o 3.200 UI di vitamina D_3/die contro placebo in un campione di popolazione generale finlandese esente da malattie cardiovascolari al basale, registrando nell'arco di 5 anni 119 eventi cardiovascolari maggiori. La supplementazione non ha conferito alcuna significativa protezione rispetto al placebo riguardo l'incidenza di eventi CV totali o specifici. Limiti importanti dello studio erano gli elevati livelli basali di 25(OH)D e il basso grado di rischio cardiovascolare della maggior parte del campione, con conseguente basso numero d≠i eventi.

Le meta-analisi di Zhang [14], Pei [15], Ruiz-Garcìa [16] e Mattumpuram [17] e rispettivi collaboratori, hanno tutte avuto come oggetto studi realizzati su campioni di popolazione generale. Tre di questi studi [14,15,17] non hanno dimostrato alcun effetto della supplementazione di vitamina D sulla mortalità o la morbilità cardiovascolare; viceversa, la meta-analisi di Rui-Garcìa et al., che si differenziava per aver incluso esclusivamente trial di durata > 1 anno e con almeno 50 partecipanti, ha dimostrato una riduzione della mortalità totale, soprattutto relativamente ai trial di maggiore qualità ovvero con più basso rischio di bias. La meta-analisi di Zhang et al., pur in assenza di un risultato positivo per l'*outcome* principale, faceva rilevare tuttavia un trend più favorevole per gli studi di durata maggiore e con supplementazione di vitamina D_3 piuttosto che D_2. La meta-analisi di Jayedi et al. [9], che ha incluso esclusivamente i trial condotti in pazienti diabetici, non ha dimostrato efficacia protettiva della supplementazione verso morbilità e mortalità cardiovascolari, denotando tuttavia un livello di evidenza piuttosto basso. A sua volta lo studio di Khan et al. [18], che ha incluso i trial condotti in soggetti pre-diabetici, non ha rilevato alcuna efficacia della supplementazione nel ridurre l'incidenza di diabete o nel migliorare la resistenza all'insulina.

La meta-analisi di Yeung et al. [19], con inclusione di trial condotti in pazienti nefropatici, parimenti non dimostrava efficacia nel ridurre la mortalità totale o cardiovascolare, pur con i limiti di trial di durata molto breve, bassa numerosità e scarsa qualità. La meta-analisi di Pincombe et al. [20], che si caratterizzava per la valutazione di trial che hanno esaminato gli effetti della supplementazione di vitamina D sulla funzione endoteliale e per includere un 42% di pazienti con insufficienza o carenza di vitamina D al basale, non ha riscontrato un beneficio significativo su nessuno dei principali parametri di funzione endoteliale, se non per un trend positivo della vasodilatazione flusso-mediata.

Infine, la rassegna sistematica di Zittermann et al. [21], che ha valutato 22 studi che riportavano gli eventuali effetti avversi della somministrazione di vitamina D in dose da 3.200 a 4.400 UI /die contro placebo per almeno 6 mesi, ha dimostrato con queste dosi un maggior rischio di ipercalcemia (per quanto contenuto in 4 casi su 1.000 soggetti trattati), ma non di ipercalciuria, nefrolitiasi o mortalità totale.

DISCUSSIONE

L'analisi complessiva dei diversi tipi di studi più recenti che hanno valutato l'impatto della carenza di vitamina D e della sua eventuale supplementazione sui principali outcome cardiovascolari conferma quanto emerso in precedenza: una forte discrepanza tra i risultati degli studi osservazionali e quelli dei trial d'intervento. Laddove i primi, corroborati anche dai risultati dei più recenti studi di randomizzazione mendeliana, evidenziano con chiarezza e coerenza interna l'impatto negativo di una condizione di insufficienza e ancor di più di carenza di vitamina D, i secondi al contrario, sia pure con qualche eccezione, non supportano il potenziale beneficio derivante dalla supplementazione vitaminica e, quindi, non fareb-

TABELLA II.
Supplementazione di vitamina D, *outcome* cardiovascolari e mortalità: risultati dei trial più recenti.

Autore (ref.)	Tipo di studio	Caratteristiche	Risultati principali
Virtanen et al., 2022 [13]	RCT	RCT con 2.495 participanti ≥ 60 anni da popolazione generale finlandese, esenti da CVD al basale, stratificati in 3 gruppi: placebo, 1.600 UI vitamina D_3/die e 3.200 UI vitamina D_3/die, follow-up 5 anni con 119 eventi CV maggiori	La supplementazione di vitamina D_3 non è risultata associata a riduzione dell'incidenza di eventi CV maggiori (4,9%, 5,0% e 4,3% rispettivamente nei gruppi placebo, vitamina D 1.600 UI/die e vitamina D 3.200 UI/die), né dell'incidenza di infarto miocardico, ictus e morte CV. Limiti principali dello studio: livelli basali di 25(OH)D nei partecipanti allo studio mediamente alti e basso numero di eventi
Zhang et al., 2019 [14]	Meta-analisi di RCT	52 trial (n = 75.454) con 7.993 morti totali di cui 1.331 CV, follow-up mediano 1 anno (solo per 12/52 trial: durata > 3 anni)	La supplementazione di vitamina D_2/D_3 non è risultata associata a riduzione della mortalità totale (R-ratio = 0,98, 95% IC: 0,95-1,02) o CV (R-ratio = 0,98, 95% IC: 0,88-1,08). Altre considerazioni e limiti dello studio: vitamina D_3 più efficace della D_2, trial più lunghi, maggior efficacia, moltissimi studi consentivano la supplementazione spontanea nel gruppo di controllo, i livelli medi basali di vitamina D erano mediamente alti
Pei et al., 2022 [15]	Meta-analisi di RCT	18 trial (n = 70.278), 1.495 morti CV, follow-up 1-6 anni	La supplementazione di vitamina D_2/D_3 non è risultata associata a riduzione della mortalità CV totale (RR = 0,96, 95% IC: 0,88-1,06), l'incidenza di ictus (RR = 1,05, 95% IC: 0,92-1,20), infarto miocardico (RR = 0,97, 95% IC: 0,87-1,09) ed eventi CV totali (RR = 0,97, 95% IC: 0,91-1,04). Limiti principali dello studio: i livelli medi basali di vitamina D erano mediamente alti, il rischio CV di base piuttosto basso, il follow-up relativamente breve
Ruiz-Garcìa et al., 2023 [16]	Meta-analisi di RCT	80 studi (n = 163.131) di cui 35 a basso rischio, 34 a rischio medio e 11 ad alto rischio di bias. Esclusi i trial con meno di 50 partecipanti e di durata < 1 anno. Follow-up mediano 2 anni	La supplementazione di vitamina D_2/D_3 ha ridotto la mortalità totale (OR 0,95, 95% IC: 0,93-0,99, p < 0,02). Tale effetto è confermato per i trial a minor rischio di bias, mentre non lo è per quelli di minore qualità. Viceversa nessuna associazione tra la supplementazione di vitamina D e la mortalità CV totale, per infarto cardiaco, ictus o insufficienza cardiaca. Limiti principali dello studio: mancanza dei livelli di 25(OH)D al basale
Mattumpuram et al., 2024 [17]	Metanalisi di RCT	36 trial (n = 493.389)	La supplementazione di vitamina D non ha prodotto effetti sulla mortalità CV (RR = 1,01, 95% IC: 0,94-1,08), sul rischio di ictus cerebrale (RR = 1,03, 95% IC: 0,95-1,11) e di infarto miocardico (RR = 0,98, 95% IC: 0,91-1,06; p = 0,65)
Jayedi et al., 2023 [9]	Meta-analisi di RCT	6 trial (n = 7.316 pazienti diabetici)	La supplementazione di vitamina D_2/D_3 non ha ridotto la mortalità totale (RR = 0,96, 95% IC: 0,79-1,16) né la morbilità e mortalità CV. Limiti principali dello studio: per morbilità e mortalità CV grado di evidenza molto basso
Khan et al., 2023 [18]	Meta-analisi di RCT	7 trial (n = 6.775 pazienti prediabetici), follow-up da 3 mesi a 5 anni con 1.385 eventi	In tutti i trial tranne 1 la supplementazione di vitamina D non ha ridotto l'incidenza di diabete (20,0% vitamina D *vs* 23,3% placebo). Anche i valori di HOMA-index non sono risultati significativamente diversi in corso di trattamento
Yeung et al., 2023 [19]	Meta-analisi di RCT	128 studi (n = 11.270 pazienti nefropatici)	La supplementazione di vitamina D non ha ridotto la mortalità totale (RR = 1,04, 95% IC: 0,84-1,24) o cardiovascolare (RR = 0,73; 95% IC: 0,31-1,71). Limiti principali dello studio: inclusione di trial di durata molto breve, bassa numerosità e scarsa qualità
Pincombe et al., 2023 [20]	Meta-analisi di RCT	26 studi (n = 2.808), con 42% dei partecipanti affetti da carenza o insufficienza di vitamina D, per valutare l'effetto della supplementazione sulla funzione endoteliale	Nessuno dei tre parametri di funzione endoteliale misurati è migliorato per effetto della supplementazione: vasodilatazione flusso-mediata, FMD% (+1,17%, 95% IC: -0,20-2,54, p = 0,095), velocità dell'onda di polso, PWV (-0,09 m/s, 95% IC: -0,24-0,07, p = 0,275), indice di incremento, AIx (+0,05%, 95% IC: -0,1-0,19, p = 0,52)
Zittermann et al., 2023 [21]	Meta-analisi di RCT	22 studi (n = 12.952) che riportavano *safety* data con somministrazione di vitamina D in dose da 3.200 a 4.400 UI/die per almeno 6 mesi	La supplementazione di vitamina D alle dosi utilizzate è risultata associata a maggior rischio di ipercalcemia (RR = 2,21, 95% IC: 1,26-3,87), pur se limitata a 4 casi per 1.000 pazienti trattati. Viceversa nessun effetto sul rischio di ipercalciuria, nefrolitiasi e mortalità totale

CVD: malattia cardiovascolare; CV: cardiovascolare; RCT: studio randomizzato controllato; IC: intervallo di confidenza; RR: rischio relativo; HR: hazard ratio; OR: odds ratio; FMD: vasodilatazione flusso-mediata; PWV: velocità dell'onda di polso; AIx: indice di incremento.

bero propendere per un ruolo causale della carenza vitaminica nel determinismo delle alterazioni metaboliche e cardiovascolari. L'impossibilità di dimostrare l'atteso effetto protettivo della correzione della carenza vitaminica rischia di generare e, di fatto, ha in certa misura generato una paralisi decisionale riguardo l'eventuale supplementazione vitaminica.

Per fornire un contributo al superamento di questa impasse, potenzialmente dannosa o anche molto dannosa per la salute dei pazienti, si offrono all'attenzione tre ordini di considerazioni. La prima di queste riguarda la qualità e validità scientifica dei trial controllati e randomizzati ai fini della dimostrazione del rapporto "causale" tra carenza vitaminica e rischio cardiovascolare. A questo riguardo occorre prendere atto che già grossi trial come il VIDA (*Vitamin D Assessment Study*), il VITAL (*VITamin D and OmegA-3 TriaL*) e il D2D (*The Vitamin D and Type 2 Diabetes*) avevano fornito evidenza che la supplementazione di vitamina D, a scopo preventivo e non sostenuta dalla documentata presenza di insufficienza o carenza, non arrecava benefici convincenti: d'altra parte questi stessi studi, proprio in virtù del loro disegno sperimentale, non hanno potuto dimostrare se una supplementazione condotta in modo adeguato, in pazienti certamente carenti e con un monitoraggio nel tempo dei livelli di 25(OH)D conseguiti attraverso la supplementazione stessa, eserciti o meno un'azione protettiva. Né questo tipo di dimostrazione è stata prodotta dagli studi di intervento più recenti, considerati in questa rassegna, in quanto a loro volta affetti dallo stesso tipo di limitazioni con l'aggiunta in molti casi di follow-up eccessivamente brevi e di numerosità insufficienti: fanno eccezione, peraltro, le meta-analisi di Ruiz-Garcìa et al. e di Zhang et al. che hanno mostrato un possibile beneficio attraverso la selezione di trial di maggiore durata e con un numero più alto di partecipanti.

Il secondo ordine di considerazione riguarda la modalità di valutazione dell'esistenza o meno di una relazione causale tra un determinato fattore di rischio (nel nostro caso la carenza di vitamina D) e uno o più *outcome* predefiniti. A questo proposito è stato da alcuni autorevolmente suggerito, analogamente a quanto realizzato in relazione ad altre applicazioni importanti della medicina preventiva, che l'analisi dei risultati dei trial controllati e randomizzati non debba essere il solo strumento di valutazione, ma che questa sia affiancata dall'analisi complessiva di tutti gli elementi di conoscenza disponibili. In particolare, si è fatto riferimento ai criteri di Hill [22], che chiamano in causa, in aggiunta ai risultati dei trial, il valore degli studi di osservazione tenendo in debito conto la forza delle associazioni eventualmente osservate, la loro consistenza, la relazione dose-risposta, la plausibilità biologica e la coerenza con i dati derivanti da studi di laboratorio e su modelli animali. Nel caso della carenza di vitamina D, l'analisi critica di tutti questi fattori depone a favore di una relazione causale con gli *outcome* cardiovascolari esaminati e di questo non è ragionevole non tener conto, soprattutto alla luce della raggiunta consapevolezza della grande difficoltà economica e pratica di progettare in futuro altri trial d'intervento che superino i limiti metodologici di quelli già disponibili.

La terza e conclusiva considerazione riguarda la condotta pratica da seguire da parte del medico alla luce di quanto discusso sopra e delle conoscenze attuali. Laddove è evidente che la supplementazione di vitamina D non è da prendere in considerazione a prescindere dalla valutazione del suo stato nutrizionale, essendosi rivelata inefficace per gli *outcome* considerati in soggetti già vitamina D-repleti, le conoscenze attualmente disponibili suggeriscono la necessità di valutare l'esistenza o meno di una situazione carenziale di vitamina D, quanto meno in quella parte della popolazione che è a maggior rischio di carenza (soggetti anziani, specialmente se costretti a casa o ricoverati presso case di riposo e comunque tutti coloro che trascorrono poco tempo all'aria aperta), anche in relazione a condizioni morbose croniche, cardiovascolari, oncologiche o di altro tipo. In tutti questi soggetti, in caso di documentata carenza di vitamina D, cioè 25(OH)D < 20 ng/mL o 50 nmol/L o anche in una condizione di marcata insufficienza, è opportuno procedere a una supplementazione tenendo conto dei risultati della recente analisi di Zittermann et al., che ha documentato l'insussistenza del rischio di effetti avversi almeno fino alla dose di 4.000 UI/die [21]. Naturalmente l'indicazione alla supplementazione permane valida in particolare per i pazienti con osteoporosi documentata che necessiti di trattamento con bifosfonati e anche dei pazienti osteopenici che non riescano ad attingere valori normali della vitamina attraverso la sola alimentazione e l'esposizione ai raggi solari.

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Vitamina D e disturbi mentali: *update* sulle ultime evidenze e focus su autismo e anoressia

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Summary

La vitamina D, originariamente associata alla regolazione del calcio e alla salute ossea, sta emergendo come un elemento cruciale nella sfera della salute mentale, non soltanto in disturbi quali la depressione e la schizofrenia, ma anche nell'autismo e nei disturbi della condotta alimentare. La presenza dei recettori della vitamina D in varie regioni cerebrali suggerisce un ruolo significativo nella neuroprotezione, neurogenesi e regolazione neuroimmunologica. La carenza di vitamina D nei primi anni di vita è associata a un aumentato rischio di sviluppare schizofrenia e bassi livelli di vitamina D sono stati correlati alla depressione, con evidenze sull'utilizzo della supplementazione della stessa nella riduzione dei sintomi depressivi. Nei disturbi dello spettro autistico, bassi livelli di vitamina D sono stati osservati nei bambini e nelle madri durante la gravidanza, ma la causalità rimane complessa. Pazienti con disturbi alimentari mostrano carenza di vitamina D, con implicazioni sulla salute ossea e mentale, e la vitamina D potrebbe avere anche un legame con l'impulsività in questi casi.
La supplementazione di vitamina D può migliorare alcuni sintomi, ma ulteriori ricerche sono necessarie per comprendere appieno i meccanismi sottostanti. Questa panoramica sottolinea l'importanza della vitamina D nella salute mentale e la necessità di ulteriori studi per chiarire le relazioni causali e sviluppare terapie più efficaci per i disturbi neuropsichiatrici.

INTRODUZIONE: LA VITAMINA D IN PSICHIATRIA E I POTENZIALI MECCANISMI D'AZIONE

Nel contesto della salute mentale, la vitamina D ha acquistato negli ultimi anni una discreta rilevanza. Recenti studi hanno approfondito il suo ruolo ben oltre l'omeostasi del calcio e la salute ossea, esplorando le sue implicazioni nel campo neuropsichiatrico. La ricerca ha progressivamente illuminato la relazione tra vitamina D e varie condizioni mentali, inclusi disturbi come la depressione e l'ansia [1].

Nel contesto dei disturbi psichiatrici, la vitamina D è coinvolta nell'espressione regione-specifica dei recettori della vitamina D (VDR) in aree come la corteccia cingolata, il talamo, il cervelletto, la substantia nigra, l'amigdala e l'ippocampo. La presenza di vitamina D, VDR ed enzimi correlati in varie regioni del cervello ha chiarito il ruolo fondamentale della vitamina D come ormone neuroattivo/neurosteroide nei processi di neuroimmuno-modulazione, neuroprotezione, neurogenesi, e nella normale funzione cerebrale [1]. Il deficit di vitamina D nei primi anni di vita, infatti, influisce negativamente sui suddetti processi: bambini con bassi livelli di vitamina D presentano, ad esempio, un maggiore rischio di sviluppare disturbi quali la schizofrenia [2]. Recentemente, è stato identificato un ulteriore ruolo significativo della vitamina D nella differenziazione dei neuroni dopaminergici: uno studio del 2023 ha dimostrato che l'esposizione cronica all'ormone attivo della vitamina D aumenta la capacità dei neuroni in via di sviluppo di produrre e rilasciare dopamina, stabilendo così la vitamina D come un agente differenziatore importante per i neuroni dopaminergici in via di sviluppo [3].

Attraverso differenti meccanismi, dunque, la vitamina D influenza disturbi mentali come ansia, depressione e schizofrenia. Inoltre, recenti studi hanno esplorato il ruolo della vitamina D anche in relazione all'autismo e ai disturbi della condotta alimentare, ampliando

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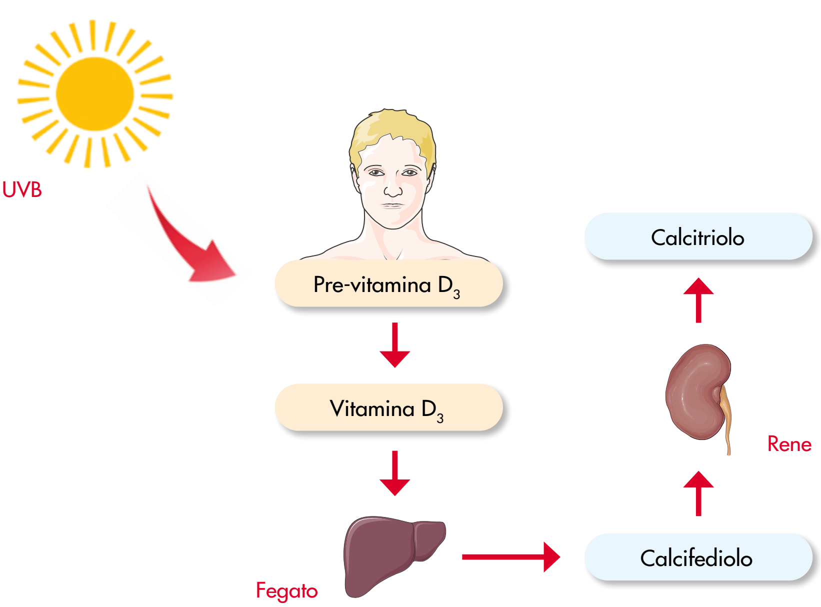
la comprensione del suo impatto sulla salute mentale.

VITAMINA D E DISTURBI PSICHIATRICI: LE EVIDENZE PIÙ RECENTI

Studi suggeriscono una relazione tra la carenza di vitamina D durante lo sviluppo e l'aumento del rischio di schizofrenia e depressione. La depressione può aggravare la carenza di vitamina D riducendo l'esposizione solare, mentre i sintomi della carenza possono a loro volta peggiorare lo stato depressivo (Fig. 1) [1].
Recentemente, una meta-analisi, che prendeva in considerazione studi controllati randomizzati con placebo, ha dimostrato che la supplementazione di vitamina D in soggetti carenti riduceva significativamente i sintomi depressivi in individui con diagnosi di disturbo depressivo maggiore e con sintomi depressivi lievi [4]. Inoltre, una recente analisi trasversale condotta negli Stati Uniti ha esaminato l'associazione tra carenza di vitamina D, età e depressione. L'analisi prendeva in considerazioni le caratteristiche demografiche, le caratteristiche dei sintomi depressivi e i livelli ematici di vitamina D, e ha rivelato un'associazione significativa tra deficit di vitamina D e rischio di depressione [5].
Analogamente, una meta-analisi ha sintetizzato le evidenze da trial controllati randomizzati, dimostrando che gli integratori di vitamina D sono significativamente superiori al placebo nel ridurre i sintomi depressivi in adulti, con un effetto particolarmente marcato in coloro che soffrono di depressione più severa e nei soggetti con livelli più bassi [6]. Altri studi hanno mostrato non solo che la supplementazione di vitamina D potrebbe ridurre lo sviluppo dei sintomi della depressione, ma anche che la presenza di livelli sierici più elevati di vitamina D potrebbe ridurre il rischio, mettendo in evidenza come soggetti con livelli ematici più bassi di vitamina D avessero maggiori probabilità di sviluppare depressione.
Inoltre, è stata evidenziata una correlazione negativa tra bassi livelli di vitamina D durante il primo trimestre di gravidanza e lo sviluppo di sintomi depressivi nel secondo trimestre, così come un rischio aumentato di sintomi depressivi peri-partum in seguito a ipovitaminosi D nel secondo trimestre [7]. Un recente trial randomizzato controllato ha peraltro mostrato che la supplementazione di vitamina D nei primi due anni di vita riduceva il rischio di disturbi come ansia e depressione all'età di 6-8 anni [8].

Nei pazienti schizofrenici si assiste a una prevalenza del 70% di carenza di vitamina D, a fronte di una prevalenza generale del 37,6% nella popolazione.
Le persone nate in inverno e primavera hanno un leggero aumento del rischio di sviluppare la schizofrenia: questo fenomeno potrebbe essere dovuto a fattori ambientali stagionali, come infezioni più comuni nei mesi freddi, ma anche a una minore esposizione alla luce solare. In particolare, è stata osservata una correlazione tra la carenza di vitamina D nelle donne incinte e nei neonati durante questi mesi e un aumento del rischio di schizofrenia: la radiazione ultravioletta durante l'inverno in siti ad alta latitudine può essere difatti insufficiente per innescare la reazione necessaria alla produzione del precursore della vitamina D [9]. Il rischio di schizofrenia è inoltre più alto nella prole di migranti dalla pelle scura in alcuni paesi. Fattori legati alla marginalizzazione sociale e allo stress migratorio sono collegati a un aumento del rischio di disturbi mentali in generale, inclusa la schizofrenia; tuttavia, gli individui con pelle pigmentata che vivono in climi freddi sono a maggior rischio di carenza di vitamina D, poiché la pelle pigmentata agisce come una protezione solare naturale e riduce la produzione del precursore della vitamina D [10]. Inoltre, è stato dimostrato che coloro che sono migrati nei Paesi Bassi da bambini hanno un rischio aumentato di schizofrenia successiva (rispetto a coloro che migrano da adulti): ciò può suggerire la presenza di una finestra critica di esposizione, ovvero di un intervallo di età in cui l'esposizione alla carenza di vita-



- Pazienti con depressione maggiore hanno livelli ematici più bassi di vitamina D
- La supplementazione di vitamina D in pazienti depressi carenti può migliorare i sintomi depressivi

- Nei pazienti con schizofrenia si riporta una prevalenza del 70% di carenza di vitamina D
- Esiste una complessa sovrapposizione genetica tra la deficienza di vitamina D e la schizofrenia

FIGURA 1.
La vitamina D, ottenuta attraverso l'esposizione ai raggi UVB e la successiva biotrasformazione a livello epatico e renale, potrebbe essere coinvolta in differenti disturbi mentali. La sua carenza, infatti, può essere correlata a depressione maggiore e schizofrenia.

mina D può aumentare il rischio di disturbi neuroevolutivi [10].
Infine, un'analisi del 2023 ha mostrato un'architettura genetica condivisa tra la schizofrenia e i livelli di vitamina D, identificando nuovi loci di rischio e sottolineando un complesso meccanismo di sovrapposizione genetica tra la deficienza di vitamina D e la schizofrenia. Questi risultati suggeriscono che varianti genetiche condivise possano contribuire alla coesistenza di schizofrenia e carenza di vitamina D, influenzando il quadro clinico [11].

VITAMINA D E AUTISMO

L'eziologia e la patogenesi dei disturbi dello spettro autistico (ASD) sono complesse e non completamente chiarite. Dall'inizio degli anni '80, la ricerca sull'autismo ha superato la teoria dell'"inadeguata cura parentale", concentrandosi sulle cause biologiche. Si è scoperto che l'ASD è un disturbo neuroevolutivo causato dall'interazione di fattori genetici e ambientali. Oltre 1.000 geni sono stati collegati all'ASD e c'è una maggiore concordanza tra gemelli monozigoti rispetto ai dizigoti, suggerendo un forte ruolo genetico. Tuttavia, solo il 25-30% dei bambini con ASD mostra geni correlati all'ASD, evidenziando l'importanza dei fattori ambientali. Fattori come nutrizione, farmaci, sostanze tossiche, infezioni materne durante la gravidanza, stress e vaccinazioni sono stati associati all'ASD. Alcuni bambini con ASD presentano livelli elevati di serotonina e anomalie nel funzionamento della dopamina, oltre a disordini nella struttura e nelle connessioni cerebrali. Studi immunologici indicano anche un'alterazione dell'equilibrio immunitario. La carenza di vitamina D, collegata a fattori come l'inquinamento atmosferico, le condizioni climatiche e la latitudine, è stata proposta come possibile causa dell'ASD [12].
Una revisione sistematica e di meta-analisi ha dimostrato che i bambini con ASD presentano livelli sierici significativamente più bassi di vitamina D rispetto ai controlli senza diagnosi di ASD [13]. Inoltre, sia bassi livelli di vitamina D nel sangue materno che bassi livelli di vitamina D nel sangue del neonato si correlano significativamente con un rischio maggiore di successiva diagnosi di ASD [13].
Anche per questo disturbo esiste un rapporto di casualità col deficit di vitamina D ambiguo: i bambini con ASD hanno abitudini di vita diverse, inclusa una dieta più selettiva e meno varia, che porta a un minore introito di vitamina D. Inoltre, tendono a trascorrere meno tempo in attività all'aperto, riducendo l'esposizione ai raggi UV-B solari e, di conseguenza, la sintesi cutanea di vitamina D. Un altro fattore che può influenzare i livelli di vitamina D è genetico, legato a varianti dei geni del metabolismo e del recettore della vitamina D associati al rischio di ASD. Infine, l'uso di alcuni farmaci, come quelli antiepilettici, può anche causare la riduzione dei livelli di vitamina D.
In ogni caso, il potenziale terapeutico della supplementazione di vitamina D nei bambini con ASD è stato esplorato in vari studi: in particolar modo, si è evidenziato che la supplementazione nei soggetti carenti può migliorare alcuni sintomi dell'ASD, in particolare i comportamenti stereotipati, ma non incide significativamente su altri sintomi principali e condizioni coesistenti [14].
I meccanismi sottostanti la relazione tra vitamina D e ASD devono ancora essere pienamente chiariti: la vitamina D è nota per svolgere ruoli nello sviluppo cerebrale, nella funzione immunitaria e nell'infiammazione, che sono rilevanti per l'ASD. È stato dimostrato che modula le citochine infiammatorie, influenza le vie antiossidanti e regola neurotrasmettitori come la serotonina, tutti elementi fondamentali nel contesto dell'ASD [13]. Inoltre, la vitamina D interagisce con vari geni associati all'ASD e la sua carenza può interrompere i processi neuroevolutivi [13].
Sussistono tuttavia limitazioni nella ricerca attuale, tra cui l'eterogeneità nei disegni di studio, nei regimi di dosaggio della vitamina D e nelle caratteristiche dei partecipanti, che sfidano la formulazione di conclusioni definitive. La variabilità nella risposta alla supplementazione di vitamina D tra gli individui con ASD suggerisce che fattori genetici e ambientali potrebbero influenzarne l'efficacia.

VITAMINA D, DISTURBI DELLA CONDOTTA ALIMENTARE E IL RUOLO DELL'IMPULSIVITÀ

I pazienti con anoressia nervosa (AN) hanno livelli significativamente più bassi di vitamina D nel siero, sia nella forma di 25-idrossivitamina D [25(OH)D] che di 1,25-diidrossivitamina D [1,25(OH)D], rispetto ai controlli [15].
I bassi livelli di 25(OH)D nel siero possono portare alla perdita ossea tipica dell'AN, con conseguente riduzione della densità minerale ossea e una maggiore frequenza di fratture cliniche e non cliniche rispetto agli adolescenti sani. È pertanto importante tener di conto dei valori di vitamina D, non soltanto per la salute del tessuto scheletrico, ma anche per il ruolo che la vitamina D riveste anche negli altri disturbi mentali, che spesso affliggono i pazienti con AN [15].
Una meta-analisi ha rivelato che i pazienti con AN mostravano livelli sierici di vitamina D significativamente più bassi rispetto ai controlli nonostante l'introito di vitamina D fosse simile. Diversi elementi possono essere presi in considerazione per giustificare questi dati: i pazienti con AN tendono a sovrastimare il loro consumo di cibo, il che potrebbe portare a una valutazione incoerente dell'assunzione di micronutrienti. Inoltre, non tutte le attività fisiche hanno effetti simili nel mantenere livelli ottimali di 25(OH)D. Potrebbe accadere che i pazienti con AN trascorrano più tempo in attività indoor piuttosto che all'aperto o indossino abiti che coprono più il corpo, riducendo così l'esposizione alla luce e la sintesi cutanea di vitamina D.
Sebbene bassi livelli sierici di 25(OH)D siano tipici nelle persone obese a causa della maggiore massa grassa, ricerche crescenti hanno mostrato che bassi livelli sierici di 25(OH)D sono associati anche a stati di magrezza, come la malnutrizione, la cachessia neoplastica e l'AN [15].
Infine, i pazienti con AN hanno anche livelli sierici più bassi della forma attiva della vitamina D, 1,25(OH)D. I livelli di quest'ultima hanno poca relazione con le riserve di 25(OH)D e sono regolati principalmente dai livelli di ormone paratiroideo (PTH). In condizioni di bassi livelli sierici di 25(OH)D, la forma attiva di vitamina D di solito aumenta, invece di diminuire, come osservato nei pazienti con AN. Questo squilibrio tra 1,25(OH)D e 25(OH)D nell'AN potrebbe essere spiegato dai bassi livelli sierici di estrogeni in questi pazienti, ormoni che sembrano essere importanti agonisti della 1-alfa idrossilasi [15].
Un recente studio pilota ha inoltre mostrato che, in una popolazione di 236 pazienti con disturbi della condotta alimentare, i livelli di vitamina D correlavano con la presenza di comportamenti impulsivi [16]. L'impulsività è considerata un elemento implicato nell'insorgenza e nell'esito di diversi disturbi alimentari: in pazienti affetti da queste patologie, infatti, attraverso indagini di neuroimaging si riscontra uno squilibrio tra l'area frontale e mesolimbica [16].
La supplementazione con vitamina D potreb-

be essere considerata come parte dell'approccio terapeutico per il controllo dei sintomi e la prevenzione delle ricadute in individui con disturbi alimentari, come già testato in pazienti con diagnosi di disturbo da deficit di attenzione/iperattività (ADHD) o con comportamenti suicidari [16].

KEY MESSAGE SU AUTISMO E ANORESSIA

Recentemente è stato ipotizzato il coinvolgimento della vitamina D e della sua carenza anche in disturbi quali l'autismo e l'anoressia nervosa.

- Bassi livelli di vitamina D nel sangue materno e nel sangue del neonato correlano con un rischio maggiore di successiva diagnosi di autismo
- La supplementazione nei soggetti carenti può migliorare i comportamenti stereotipati

- Pazienti con anoressia nervosa mostravano livelli di vitamina D più bassi rispetto ai controlli nonostante l'introito di vitamina D fosse simile
- I livelli di vitamina D correlano con la presenza di comportamenti impulsivi

CONCLUSIONI

L'analisi della letteratura recente ha delineato un quadro in cui la vitamina D si configura come un elemento potenzialmente influente in diversi disturbi mentali. Oltre alle più studiate correlazioni con la depressione e la schizofrenia, la letteratura degli ultimi anni sta producendo evidenze anche sul rapporto che intercorre tra la vitamina D e patologie, quali l'autismo e i disturbi della condotta alimentare. Sebbene i risultati suggeriscano una correlazione tra la carenza di vitamina D e la manifestazione e la severità di questi disturbi, la relazione causale non è ancora stata chiaramente delineata. In particolare, nei disturbi come l'autismo e l'anoressia nervosa la vitamina D sembra avere un ruolo sia nello sviluppo che nell'esacerbazione dei sintomi. Tuttavia, è cruciale considerare che questa associazione potrebbe non essere univoca e ulteriori ricerche sono necessarie per comprendere se la carenza di vitamina D sia un fattore causale, una conseguenza o un elemento concomitante di questi disturbi. Questa revisione mette anche in evidenza come interventi terapeutici basati sulla supplementazione di vitamina D possano aver beneficio sui disturbi mentali.
Il crescente numero delle evidenze sul rapporto che intercorre tra disturbi mentali, quali schizofrenia e depressione, e la vitamina D pone le basi per un approfondimento della relazione tra quest'ultima e altre patologie psichiatriche, nonché per l'utilizzo della supplementazione della stessa in pazienti affetti da disturbi mentali.

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