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EDITORIALE

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Cari Lettori,
è noto che solo il 20% dello stato vitaminico D deriva dall'alimentazione ma sino ad ora erano scarse le informazioni relative al contributo effettivo dell'introito alimentare quotidiano di vitamina D nella popolazione italiana. In questo numero troverete una sintesi dei risultati principali di un recente studio in merito coordinato dalla Scuola Senese. Noterete che l'introito giornaliero alimentare di vitamina D è risultato pari a sole 200-250 UI, marcatamente ridotto rispetto alla quantità di vitamina D di 800 UI al giorno ritenuta essere quella minima sufficiente per soddisfare le esigenze metaboliche di un giovane adulto sano. Peraltro è stata anche osservata una progressiva diminuzione di assunzione dalla quinta alla ottava decade di vita, a fronte di un aumentato fabbisogno con l'invecchiamento. Il pesce, tra i pochi alimenti con un alto contenuto di vitamina D, è risultato avere una frequenza di assunzione molto bassa. L'introito alimentare di vitamina D è risultato particolarmente ridotto nei vegetariani e nei vegani ed inferiore rispetto alla popolazione sana nei pazienti affetti da patologie croniche, tra cui in particolare l'osteoporosi o le malattie renali, per le quali non si può escludere un ruolo nella loro patogenesi. Dopo regressione logistica multipla anche la residenza nella macro-area del Nord Italia ed il basso livello educazionale sono risultate associate ad un basso apporto alimentare di vitamina D. I messaggi principali che derivano da questo studio sono che in condizioni di scarso irraggiamento solare in Italia le attuali abitudini alimentari non consentono di fornire un significativo apporto alternativo di vitamina D e che andrebbero intraprese campagne educazionali in merito o strategie di fortificazione degli alimenti con vitamina D. A quest'ultimo proposito vi invito a leggere anche la recente fotografia in Europa [1].
L'altro contributo in questo numero della rivista riguarda l'interferenza del frequente e diffuso uso cronico degli inibitori di pompa (PPI) sul metabolismo osseo e della vitamina D in particolare. L'uso a lungo termine dei PPI è oggi riconosciuto come fattore di rischio per lo sviluppo di profonde alterazioni del metabolismo minerale, contribuendo all'insorgenza di osteoporosi. I meccanismi patogenetici sono molteplici e qui ben descritti dagli Autori: 1) i PPI interagiscano con molecole regolatorie centrali nel rimodellamento osseo, come i regolatori della proliferazione osteoblastica, della sensibilità estrogenica e dell'attività osteoclastica; 2) l'ipomagnesiemia cronica rappresenta una delle complicanze meglio documentate del trattamento protratto con PPI, attribuibile in gran parte a disregolazione dell'assorbimento intestinale. Poiché il magnesio rappresenta un cofattore essenziale per le reazioni di 25-idrossilazione epatica e 1-idrossilazione renale, la sua carenza determina una condizione di "resistenza funzionale" alla vitamina D, con ridotta produzione del metabolita attivo e conseguente compromissione dell'assorbimento intestinale del calcio, iperparatiroidismo secondario ed aumento del riassorbimento osseo; 3) la soppressione dell'acidità gastrica indotta dai PPI riduce la solubilizzazione dei sali di calcio, in particolare del carbonato, aggravando ulteriormente il deficit di assorbimento e contribuendo al quadro di iperparatiroidismo secondario; 4) l'ipocloridria indotta dai PPI non altera soltanto la fisiologia digestiva, ma modifica profondamente anche l'ecosistema

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microbico gastrointestinale per la perdita della normale barriera acida, consentendo la sopravvivenza, la proliferazione e la migrazione di batteri verso il tenue e così predisponendo allo sviluppo di *Small Intestinal Bacterial Overgrowth*, la quale causa malassorbimento di micronutrienti liposolubili, tra cui la vitamina D, e deficit enzimatici tra cui l'intolleranza al lattosio.
Mi sembra evidente che entrambi i contributi di questo numero della rivista indicano ulteriori motivazioni alla supplementazione con vitamina D nella pratica clinica.
Buona lettura.

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Alimentazione e vitamina D: introito di vitamina D in soggetti adulti in Italia

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INTRODUZIONE

La vitamina D presente nell'organismo deriva per circa l'80% dalla sintesi cutanea di colecalciferolo (D_3) dopo irraggiamento solare e per il restante 20% dall'introduzione di cibi contenenti in prevalenza vitamina D_3 e, in misura minore, D_2 (ergocalciferolo), quest'ultima di esclusiva provenienza vegetale. Il 25(OH)D (calcifediolo) rappresenta il primo metabolita della vitamina D: viene sintetizzato a livello epatico e, per la sua lunga emivita, viene utilizzato per esprimere lo stato vitaminico D dell'organismo. Il 25(OH) D viene a sua volta metabolizzato a livello renale in $1,25(OH)_2D_3$, noto anche come calcitriolo, considerato il metabolita finale biologicamente attivo della vitamina.

Il bersaglio principale della vitamina D è rappresentato dall'intestino dove, grazie alla sintesi della *calcium binding protein*, è in grado di promuovere l'assorbimento di calcio e fosfato [1]. Da questa essenziale funzione biologica deriva l'azione della vitamina D a livello osseo, tessuto in cui è in grado di favorire il meccanismo di mineralizzazione della matrice organica. La prolungata carenza di vitamina D a livello scheletrico si sostanzia nel rachitismo del bambino e nell'osteomalacia del soggetto adulto, condizioni in cui è prevalente la presenza di tessuto osteoide non mineralizzato, causa di deformità ossee e di fratture patologiche; laddove l'insufficienza di vitamina D è meno marcata, si può realizzare, per motivi omeostatici, una condizione di iperparatiroidismo secondario e osteoporosi, da cui ancora fratture da fragilità. Grazie all'identificazione dei recettori per la vitamina D in numerosi organi e tessuti, è stato ipotizzato un ruolo della vitamina D anche nell'ambito di malattie del sistema autoimmunitario, nelle malattie cardiovascolari, respiratorie e gastrointestinali, in alcune malattie endocrine come il diabete mellito e in oncologia.

Molte sono le condizioni in grado di indurre uno stato di ipovitaminosi D: alcune da riferire specificatamente a un inadeguato irraggiamento solare, come la vita in ambienti chiusi, l'uso di abiti che coprono estesamente la superficie cutanea, l'impiego eccessivo di creme solari protettive e l'inquinamento atmosferico; altre da considerare come fisiopatologiche, come l'invecchiamento, la gravidanza, la pigmentazione scura della pelle e l'obesità; altre ancora da ricondurre a un carente apporto alimentare di vitamina D, come l'intolleranza al lattosio, le condizioni socio-economiche e lo scarso utilizzo di cibi fortificati con vitamina D [2]. In quest'ottica dobbiamo considerare che solo pochi cibi sono in grado di fornire un adeguato apporto di vitamina D: tra questi vanno ricordati il pesce, il latte e i suoi derivati, l'uovo e la carne, mentre altri alimenti come il pane e la frutta sono del tutto privi di vitamina D. Ed effettivamente numerosi studi hanno documentato come l'ipovitaminosi D sia una condizione diffusa in età pediatrica, nelle donne in gravidanza e, in particolare, tra i soggetti intolleranti al lattosio [2]. In popolazioni del nord Europa, la prevalenza della ipovitaminosi D è stata dimostrata non soltanto nella popolazione anziana, ma anche in quella di età compresa tra 50 e 70 anni, in particolare dopo il periodo invernale [3]. Per quanto riguarda l'Italia, l'ipovitaminosi D è una condizione diffusa, che tende ad aumentare con l'invecchiamento. Non sono tuttavia disponibili informazioni scientifiche su quanto l'apporto alimentare possa contribuire alla realizzazione di un carente stato vitaminico D. Per rispondere a quest'ultimo quesito, abbiamo ritenuto opportuno effettuare uno studio epidemiologico osservazionale nella popolazione italiana, comprensivo

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di differenti realtà regionali e di diverse condizioni socio-economiche, finalizzato ad analizzare specificatamente l'entità dell'apporto alimentare di vitamina D nei soggetti adulti.

LO STUDIO SAD

Lo studio SAD (Studio sull'introito alimentare di vitamina D) è stato condotto presso gli ambulatori medici dei centri GISMO (Gruppo Italiano Studio Malattie dell'Osso) e GIBIS (Gruppo Italiano per lo studio dei Bisfosfonati) su 870 soggetti, 627 donne e 243 maschi, di età compresa da 40 a 80 anni, provenienti da tutte le regioni italiane con l'eccezione della Valle d'Aosta e del Molise. Le regioni più rappresentate sono state Sicilia, Campania e Basilicata. Sotto il profilo educazionale e lavorativo, il 39,7% dei soggetti era laureato, il 36% aveva un diploma di scuola superiore, mentre il 16,2 e l'8,2% avevano rispettivamente un diploma di scuola media ed elementare; il 32,5% della popolazione era rappresentato da pensionati, mentre il 24%, il 13,2%, il 12,8% e l'11,4% erano rispettivamente impiegati, liberi professionisti, artigiani e casalinghe.

L'analisi dell'apporto alimentare di vitamina D è stata condotta utilizzando uno specifico questionario (FFQ, *Food Frequency Questionnaire*), precedentemente validato attraverso uno studio comparativo con un diario alimentare (FFD, *Food Frequency Diary*)[4]. Il questionario conteneva 11 differenti domande, riguardanti il tipo e la quantità di alimento contenente vitamina D consumato nei 14 giorni precedenti l'intervista. La tipologia degli alimenti comprendeva: latte (intero, scremato e parzialmente scremato, addizionato con vitamina D); *corn flakes* addizionati con vitamina D; yogurt (intero, scremato, parzialmente scremato, addizionato con vitamina D); formaggio (camembert, mozzarella, fontina, *gruyère*, parmigiano, provolone, pecorino, ricotta); carne (vitello, maiale, faraona, pollo, tacchino, agnello); pesce (pesce azzurro, merluzzo, platessa, nasello, pesce spada, trota, tonno, salmone, stoccafisso, baccalà, orata, spigola, branzino; suddiviso, quando necessario, in fresco, congelato, affumicato, essiccato, conservato in olio); uovo (intero, cotto, crudo, tuorlo); cibo a base di uovo (omelette, sformato, fritto, polpette, maionese, pasta); salumi (prosciutto crudo e cotto, salame, mortadella, bresaola); dolci contenenti uovo, latte e yogurt (torta, biscotti, gelato, pasticceria); funghi. Per quanto riguarda la frequenza di assunzione, nel questionario veniva riportato quante volte un determinato cibo veniva assunto nel periodo di 14 giorni. Le informazioni relative al contenuto in vitamina D degli alimenti sono state tratte in prevalenza dalla banca dati USDA (*United States Department of Agriculture*) e, in misura minore, CREA (Consiglio per la Ricerca in agricoltura e l'analisi dell'Economia Agraria) [5]. Lo studio è stato approvato dal Comitato Etico Regionale (Regione Toscana, Sezione Area Vasta Sud Est).

Della popolazione studiata, il 31,6% era in apparente stato di buona salute, mentre il 68,4% presentava differenti patologie, di cui l'osteoporosi e le malattie cardiovascolari risultavano le più rappresentate (rispettivamente 50,1 e 41,3%). Soltanto il 3,4% della popolazione esaminata riferiva particolari abitudini alimentari: il 3,0 e lo 0,4% rispettivamente un'alimentazione vegetariana e vegana [6].

La Tabella I riporta per ciascun alimento la percentuale di utilizzo nella popolazione studiata e la sua frequenza di assunzione nel periodo di 14 giorni. Il latte viene assunto nel 63,6% dei soggetti esaminati (di cui latte parzialmente scremato nel 51,9%), il formaggio nel 93,6% (di cui parmigiano nel 23,1%), la carne nel 95,3% (di cui pollo nel 32,7%) e il pesce nel 88,7% (di cui tonno nel 18,5%). Tuttavia, se andiamo a valutare la frequenza di assunzione, il latte viene assunto quotidianamente nel 56,4% della popolazione, mentre il formaggio e la carne rispettivamente soltanto nel 3,7 e nello 0,4%. Emblematico è l'utilizzo del pesce: l'88,7% dei soggetti ha dichiarato di mangiare pesce, che per altro è l'alimento con il più elevato contenuto di vitamina D, tuttavia la sua frequenza di assunzione è molto bassa, in quanto il 45,8 e il 36,7% della popolazione dichiara di assumerlo rispettivamente soltanto 1 oppure 2 volte nell'arco dei 14 giorni del rilevamento. Anche le uova, i cibi preparati con uova e i salumi vengono assunti in misura prevalente 1 o 2 volte in 14 giorni.

Correlando per ciascun alimento il contenuto in vitamina D con la quantità ingerita nel periodo di 2 settimane, è stato possibile calcolare l'introito globale e successivamente giornaliero di vitamina D nella popolazione oggetto del presente studio. Nella popolazione femminile e maschile, l'introito globale di vitamina D si è rivelato essere rispettivamente 2832 UI ± 87 SE e 3502 UI ± 146 SE, con una differenza statisticamente significativa ($p < 0,001$): ciò corrisponde a un introito giornaliero di 202 UI ± 6,2 SE e di 250 UI ± 10,4 SE rispettivamente nei soggetti di sesso femminile e maschile. Questo dato risulta essere marcatamente ridotto rispetto alla quantità di vitamina D di 800 UI al giorno, ritenuta essere quella minima sufficiente per soddisfare le esigenze metaboliche dell'organismo [7].

Suddividendo l'introito globale della vitamina D per sesso e per decadi di età, abbiamo potuto apprezzare una graduale diminuzione di assunzione dalla quinta all'ottava decade di vita: nelle donne da 3128 UI ± 224 SE nella decade 40-49 anni a 2362 UI ± 162 SE nella decade 71-80 anni; negli uomini da 3774 UI ± 428 SE a 3029 UI ± 187 SE nelle analoghe decadi. La diminuzione è risultata statisticamente significativa nella popolazione femminile ($p < 0,01$).

La Figura 1 riporta l'introito medio giornaliero della vitamina D suddiviso per sesso e per decadi di età. L'assunzione media giornaliera diminuisce gradualmente dalla quinta all'ottava decade, passando da 269 UI ± 30 SE a 216 UI ± 13 SE nella popolazione maschile, e da 223 UI ± 16 SE della decade 40-49 anni a 168 UI ± 1 SE della decade 71-80 anni nella popolazione femminile. Sulla base di questi dati, il 76,4% dei soggetti mostrava un introito di vitamina D di 100 UI/die o inferiore, e soltanto l'11,3% della popolazione raggiungeva un introito giornaliero di almeno 400 UI/die o superiore.

Suddividendo l'apporto giornaliero di vitamina D in funzione delle tre macro-aree nord, centro e sud, l'introito medio giornaliero è risultato essere rispettivamente 197 UI ± 11 SE, 216 UI ± 18 SE e 222 U ± 36 SE, senza alcuna differenza statisticamente significativa ($p < 0,17$).

Relativamente ai soggetti con particolari abitudini alimentari, l'introito medio giornaliero nei soggetti vegetariani e vegani è risultato essere marcatamente e significativamente ($p < 0,0005$) ridotto rispetto alla popolazione del presente studio con normali abitudini alimentari: rispettivamente 112 UI ± 23 SE e 84 UI ± 31 SE *vs* 220 UI ± 5 SE.

In una successiva estensione dello studio, nella quale sono stati valutati 1372 soggetti, di cui 997 donne e 375 uomini,

TABELLA I.
La tabella riporta per ciascun alimento la percentuale di assunzione (seconda colonna da sinistra) e la sua frequenza nell'arco di 2 settimane (da Nuti et al., 2024, mod.) [6].

Cibo	Utenti (%)	Frequenza giornaliera di assunzione in 14 giorni (% dei soggetti)													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14
Latte	63,60%	3,4%	3,6%	1,2%	6,8%	1,1%	5,1%	7,7%	3,8%	0,6%	3,9%	0,2%	2,8%	0,9%	56,4%
Panna	16,30%	53,4%	28,7%	2,8%	7,2%	0,5%	1,0%	/	/	/	/	/	/	/	1,0%
Yogurt	58,60%	5,1%	15,4%	4,7%	24,5%	6,6%	9,5%	9,3%	3,3%	0,4%	5,0%	0,4%	1,5%	/	13,6%
Formaggio	93,60%	18,9%	29,5%	10,1%	15,8%	5,7%	6,3%	3%	2,9%	0,4%	3,8%	0,1%	0,7%	0,2%	3,7%
Carne	95,30%	21,9%	40,5%	10,2%	16,4%	4,7%	3,2%	1,0%	0,7%	/	0,3%	/	0,1%	/	0,4%
Pesce	88,70%	45,8%	36,7%	6.3%	6,7%	3,1%	2,2%	0,1%	0,2%	/	/	/	/	/	/
Uova	89,00%	33%	41,5%	5,7%	13,7%	1,7%	2,3%	0,5%	0,9%	/	0,2%	0,1%	/	0,1%	0,6%
Cibi preparati con uova	79,40%	50,5%	34,5%	7,0%	5,5%	1,6%	0,6%	0,3%	0,6%	/	/	/	0,4%	5,7%	1,6%
Salumi	80,90%	38,5%	38,1%	9,5%	8,8%	1,8%	1,4%	0,6%	0,6%	/	0,4%	/	0,1%	/	0,3%
Dolci preparati con uova-latte-yogurt	85,10%	15,5%	22,7%	8,6%	11,7%	5,3%	4,1%	3,1%	3,0%	0,4%	4,4%	0,4%	0,5%	0,7%	17,7%
Funghi	41,30%	56,6%	27,5%	6,2%	6,7%	1,4%	0,5%	0,5%	0,3%	/	0,6%	/	/	/	/

è stato specificatamente analizzato l'introito alimentare in pazienti affetti da specifiche patologie [8]: 429 soggetti riferivano assenza di malattia, mentre 943 la presenza di malattie croniche (399 osteoporosi, 396 malattie cardiovascolari, 136 gastrointestinali, 81 respiratorie, 210 oncologiche, 61 neurologiche, 247 endocrine e 31 renali). Nella popolazione affetta da patologie croniche, l'introito globale di vitamina D si è rivelato essere inferiore rispetto alla popolazione sana (2879 UI ± 2510 SD *vs* 3151 ± 2481 SD). In particolare, abbiamo osservato valori significativamente più bassi rispetto alla popolazione sana nei pazienti con osteoporosi (2543,1 UI ± 1904,4 SD; 95% CI: 2293,9-2793,2, p = 0,017) e con malattie renali (2210,7 UI ± 1711,6 SD; 95% CI: 1929,2-2492,2, p = 0,008) (Fig. 2). Inoltre, nelle donne con osteoporosi, l'introito di vitamina D diminuiva con l'età in modo statisticamente significativo (p <0,01). Per mezzo della regressione logistica multipla, è stato possibile rilevare che la macro-area nord (OR 1,61, 95% CI 1,20-2,16, = 0,002), il basso livello educazionale (OR 1,45, 95% CI 1,06-1,97 = 0,021), la dieta vegetariana (OR 1,86, 95% CI 1,14-3,04, = 0,012) e vegana (OR 3,89, 95% CI 2,15-7,15, p < 0,001) erano associate a un apporto di vitamina D molto basso. Relativamente ai fattori socio-economici, i soggetti con laurea oppure con diploma di scuola media superiore erano caratterizzati da un apporto di vitamina D significativamente superiore (p < 0,002) rispetto ai soggetti con licenza di scuola elementare o media; così come gli impiegati, i liberi professionisti e i manager (p < 0,03) rispetto agli artigiani, contadini e casalinghe.

DISCUSSIONE E CONCLUSIONI

I risultati del presente studio dimostrano chiaramente che in Italia, nei soggetti adulti di entrambi i sessi, l'apporto alimentare giornaliero di vitamina D è estremamente basso, mediamente 202 UI e 250 UI rispettivamente nelle donne e negli uomini. Valori questi che risultano molto inferiori rispetto a quelli raccomandati da organismi sanitari internazionali e da società scientifiche [7,9,10]. In particolari condizioni alimentari, come ad esempio la dieta vegetariana oppure vegana, l'apporto vitaminico risulta addirittura più compromesso, con valori che oscillano mediamente da 84 a 112 UI al giorno. Altra importante osservazione riguarda i pazienti affetti da patologie croniche. Rispetto ai soggetti normali inclusi nello studio, i pazienti con osteoporosi, malattie oncologiche e dell'apparato

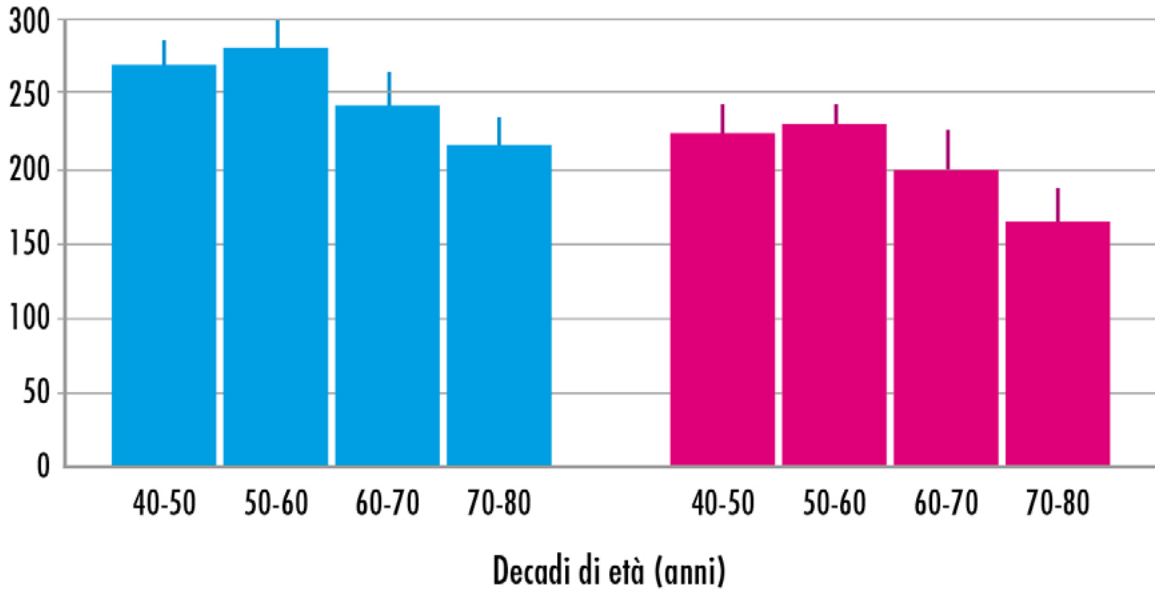

FIGURA 1.
Apporto alimentare giornaliero di vitamina D espresso in UI nei soggetti di sesso maschile (colonne blu) e di sesso femminile (colonne rosse), suddiviso per decadi di età (da Nuti et al., 2024, mod.) [6].

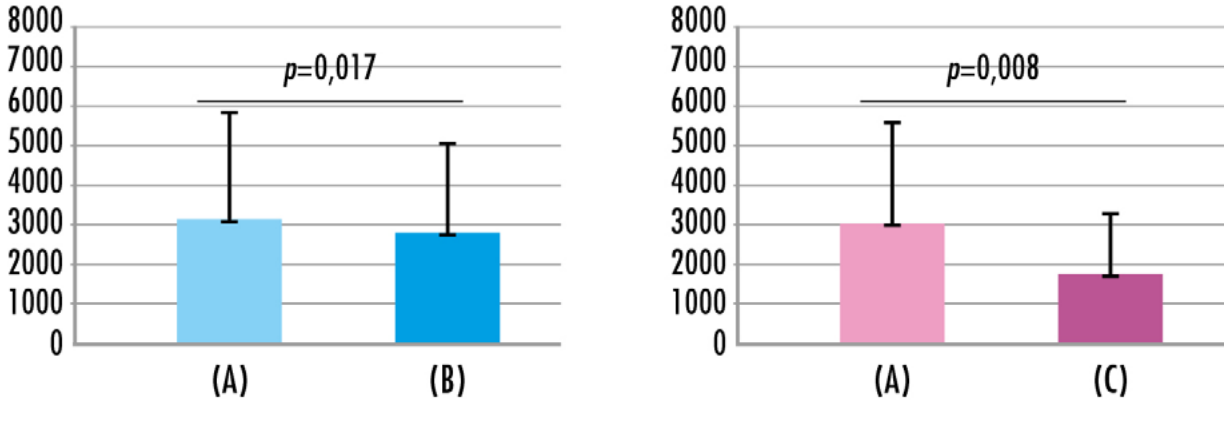

FIGURA 2.
Apporto alimentare globale di vitamina D espresso in UI nei soggetti affetti da osteoporosi (B) e malattie renali (C) rispetto a soggetti sani (A) (da Nuti et al., 2025, mod.) [8].

cardiovascolare, gastrointestinale, neurologico, respiratorio, endocrino e renale presentano valori di assunzione di vitamina D mediamente inferiori, con differenze in alcuni casi statisticamente significative (osteoporosi e malattie renali). Questi dati supportano a nostro parere il ruolo che il carente apporto alimentare di vitamina D può esercitare nel determinismo di una condizione di ipovitaminosi D. Come è noto, il principale meccanismo che regola lo stato vitaminico D dell'organismo è rappresentato dall'irraggiamento solare. Tuttavia, molte situazioni possono ostacolare una regolare esposizione ai raggi UVB: dalla latitudine al fototipo, dal non regolare e corretto svolgimento di attività fisica all'aria aperta all'abituale utilizzo di creme protettive, oltre all'età avanzata e alla vita in ambienti protetti [2]. Di conseguenza, un adeguato e costante apporto alimentare

di vitamina D potrebbe senza dubbio essere determinante nel mantenimento di un fisiologico stato vitaminico D, cosa questa che, almeno nella popolazione adulta italiana da noi esaminata, non si realizza. L'utilizzo di alimenti contenenti vitamina D nella popolazione studiata appare scarso e marginale: ad esempio il latte, che viene assunto giornalmente in oltre il 60% dei soggetti esaminati, contiene in 100 g poche decine di UI di vitamina D (52 UI) e comunque la quantità assunta risulta essere modesta; per altro, il pesce, che rappresenta l'alimento contenente il quantitativo più elevato di vitamina D (ad esempio, sgombro fresco 652 UI, salmone affumicato 692 UI, dentice 408 UI) viene mangiato dalla maggioranza dei soggetti soltanto una o due volte nell'arco di 14 giorni, contribuendo così in misura inadeguata al raggiungimento di un fisiologico apporto vitaminico. Tuttavia, se analizziamo separatamente per ogni singolo alimento il contributo che ciascuno di essi è in grado di fornire nell'arco di 2 settimane, il pesce seppur ingerito saltuariamente (1 o 2 volte) si dimostra il cibo capace di garantire una quantità di vitamina D enormemente superiore (2038,95 UI) rispetto agli altri alimenti contenenti vitamina D (Fig. 3).

Considerando in ogni caso che assai spesso, per i motivi che abbiamo già ricordato, l'esposizione ai raggi solari si rivela del tutto inadeguata, l'apporto alimentare di vitamina D può quindi svolgere un ruolo cruciale nel determinismo dell'ipovitaminosi D. Emblematico quanto osservato in un recente studio condotto in Marocco, nel quale è stata documentata un'elevata presenza di ipovitaminosi D (circa il 90% delle donne esaminate) da riferire sia a una moderata esposizione ai raggi solari, sia a un basso apporto alimentare di vitamina D (oltre l'80% della popolazione studiata assumeva vitamina D in misura inferiore rispetto a quella raccomandata) [11]. Per altro, i dati da noi osservati risultano del tutto in accordo con quanto pubblicato da altri Autori in altre realtà geografiche, dall'Australia, al Regno Unito, al Nord America: in particolare, negli USA l'apporto medio giornaliero di vitamina D risulta essere mediamente 140 UI, e rispettivamente nella popolazione canadese femminile e maschile mediamente 168 UI e 204 U [12,13]. Anche nei Paesi europei dell'area mediterranea, l'apporto medio alimentare giornaliero di vitamina D è stato dimostrato essere spesso al di sotto di 160 UI [14]. Questi dati supportano senza dubbio la crescente diffusione dell'ipovitaminosi D, da alcuni considerata una vera e propria pandemi [2], con conseguenze non soltanto limitate all'apparato scheletrico e muscolare, da cui osteomalacia, osteoporosi da iperparatiroidismo secondario, miopatia generalizzata con aumentato rischio di cadute, ma estese anche ad altri organi e apparati. Recenti ricerche supportano il ruolo essenziale della carenza di vitamina D nella fisiopatologia delle malattie cardiovascolari, neurologiche, respiratorie, del diabete mellito di I e II tipo e delle malattie autoimmuni. I nostri risultati sotto il profilo patogenetico supportano queste osservazioni, dato che nelle popolazioni affette da varie patologie l'introito di vitamina D si è dimostrato sensibilmente ridotto rispetto alla popolazione sana (in particolare nei pazienti con osteoporosi e con patologie renali) e soprattutto rispetto agli standard di riferimento. Strettamente connessa con questi aspetti è la relazione osservata tra ipovitaminosi D e mortalità. Numerosi studi osservazionali hanno documentato una correlazione statisticamente significativa tra l'ipovitaminosi D e la mortalità per tutte le cause [15]: inoltre, basandosi su studi controllati randomizzati condotti su oltre 30.000 partecipanti, è stato possibile

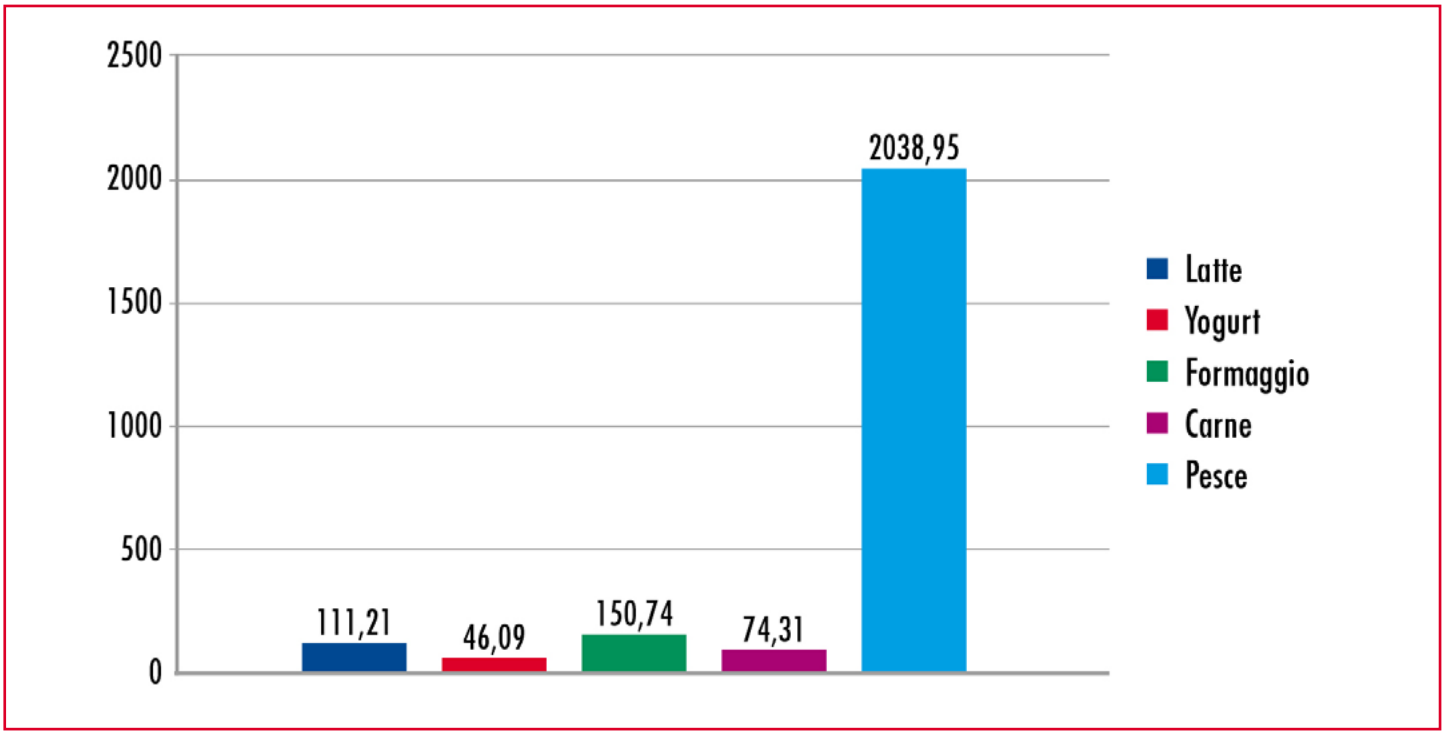

FIGURA 3.
Apporto globale di vitamina D in 14 giorni espresso in UI, suddiviso per i principali alimenti.

dimostrare che la supplementazione di vitamina D è in grado di ridurre la mortalità per tutte le cause del 11%. Come è noto la "dieta mediterranea" si caratterizza per l'assunzione in prevalenza di cibi di origine vegetale quali verdura, ortaggi, frutta fresca e secca, legumi, pane e pasta da farina integrale, olio di oliva: l'impiego di alimenti contenenti vitamina D, come latte e derivati, carne e soprattutto pesce, è prevista a frequenza settimanale e sostanzialmente non è in grado di fornire un fisiologico apporto di vitamina D. Riteniamo quindi, sulla base dei risultati dello studio SAD, che sia opportuna una campagna educazionale a partire dall'epoca infantile e adolescenziale, da proseguire successivamente sino alle età più avanzate per favorire l'introduzione di alimenti contenenti vitamina D, in particolare latte e derivati, e soprattutto pesce. Questo porterebbe a una diminuzione dell'incidenza dell'ipovitaminosi D e delle sue conseguenze cliniche. Come alternativa a questo approccio, consideriamo proponibile una fortificazione con colecalciferolo di alcuni alimenti, in particolare latte e derivati, come per altro avviene in altri Paesi. Infine, laddove questo non sia possibile, la supplementazione di vitamina D, soprattutto in popolazioni o condizioni a rischio, come indicato dalla SIOMMMS (Società Italiana dell'Osteoporosi, del Metabolismo osseo e delle Malattie dello Scheletro) [10], rimane comunque una strategia del tutto adeguata e corretta.

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Vitamina D e (ab)uso di prazoli

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VITAMIN D
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INTRODUZIONE

Gli inibitori della pompa protonica presente sulle cellule parietali gastriche (*proton pump inhibitor*, PPI) rappresentano una delle classi di farmaci più prescritte al mondo e costituiscono un pilastro nella gestione delle patologie acido-correlate. La loro maggiore efficacia rispetto agli anti-H_2 (quali cimetidina, ranitina, famotidina, ecc.) nell'indurre una totale e prolungata soppressione della secrezione acida gastrica ne ha favorito un uso sempre più esteso. Quest'ultimo è stato alimentato dall'impatto epidemiologico che l'infezione da *Helicobacter pylori*, principale causa di malattia peptica, aveva in passato nei Paesi occidentali e continua ad avere nei Paesi in via di sviluppo. Inoltre, la possibilità di un acquisto diretto da parte dell'utente senza una prescrizione medica ha determinato un impiego spesso improprio, che va oltre le indicazioni cliniche formalmente riconosciute. In Italia i PPI rappresentano il 5-10% delle prescrizioni farmaceutiche, in linea con quanto osservato in altri Paesi [1], con una quota crescente di utilizzo in assenza di reali fattori di rischio o di valide indicazioni. Ciò ha fatto sì che negli ultimi anni emergesse un crescente interesse nei confronti degli effetti collaterali conseguenti all'impiego prolungato di questi farmaci. Una cospicua letteratura ha, infatti, documentato come la soppressione cronica della secrezione acida gastrica induca profonde modificazioni dell'ecosistema intestinale, del metabolismo dei micronutrienti e dell'omeostasi minerale e ormonale. Tra le complicanze più frequentemente riportate figurano un aumentato rischio infettivo, la contaminazione batterica del tenue, la riduzione dell'assorbimento di ferro, vitamina B_{12} e magnesio, e alterazioni significative del metabolismo della vitamina D e del calcio. Ricerche più recenti hanno, inoltre, messo in evidenza la possibilità che i PPI esercitino anche degli effetti negativi diretti sul tessuto osseo, interferendo con i meccanismi regolatori la proliferazione osteoblastica e l'attività osteoclastica. In particolare, l'associazione tra ipomagnesiemia, ridotta attivazione della vitamina D, ipocalcemia lieve e incremento del turnover osseo configura un quadro fisiopatologico complesso, che va ben oltre la tradizionale spiegazione basata sulla ipocloridria. Queste osservazioni suggeriscono che l'uso cronico dei PPI rappresenti un fattore di rischio multidimensionale, capace di agire attraverso vie metaboliche, endocrine e microbiologiche convergenti verso una compromissione dell'equilibrio osseo. In tale prospettiva, la valutazione critica dell'appropriatezza prescrittiva e la comprensione dei meccanismi alla base delle complicanze associate all'uso cronico di PPI assumono un ruolo fondamentale per una prescrizione e per un uso consapevoli e personalizzati di questi farmaci.

IL MECCANISMO D'AZIONE DEGLI INIBITORI DELLA POMPA PROTONICA E IL LORO IMPIEGO CLINICO

Introdotti in commercio nel 1989, i PPI attualmente utilizzabili sono sei – omeprazolo, lansoprazolo, dexlansoprazolo, pantoprazolo, rabeprazolo, esomeprazolo – e ogni molecola ha diversi nomi commerciali in base all'azienda produttrice. Tutti esercitano la loro azione farmacologica inibendo in modo irreversibile la pompa protonica H^+/K^+-ATPasi presente sulla membrana apicale delle cellule parietali gastriche, bloccando così la secrezione di acido gastrico [2-3]. Inoltre, i PPI sono basi deboli, per cui possono essere protonati e resi attivi solo in un ambiente fortemente acido come quello gastrico [4]. Una volta attivati tramite protonazione, si legano a uno o più residui di cisteina della pompa H^+/K^+-ATPasi, rendendola inattiva [4]. Nonostante un'emivita media relativamente breve (compresa tra 0,6 e 1,9 ore), l'irreversibilità del legame fa sì che l'effetto farmacologico duri quanto l'emivita delle cellule parietali, pari a 48 ore in media. L'attività ottimale dei PPI si ottiene quando vengono somministrati a stomaco vuoto, preferibilmente 30-60 minuti prima dei pasti.

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L'Autore dichiara nessun conflitto di interessi.

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L'assunzione a digiuno non solo ne migliora l'assorbimento, ma garantisce anche che il picco plasmatico del farmaco coincida con la stimolazione massimale della pompa protonica espressa sulle cellule parietali gastriche conseguente all'ingestione di cibo [5-6]. Per quanto attiene le indicazioni terapeutiche, queste sono molteplici e comprendono: le emorragie digestive alte, la malattia da reflusso gastroesofageo, l'esofagite, le varici esofagee e/o la gastropatia congestizia conseguenti all'ipertensione portale, la malattia peptica, la prevenzione delle ulcere indotte da farmaci antinfiammatori non steroidei nei pazienti a rischio, la gastrite uremica, la sindrome di Zollinger-Ellison, nonché nei cicli di terapia eradicante l'infezione da *Helicobacter pylori* [7]. In passato, prima che si scoprisse l'*Helicobacter pylori* quale agente eziologico della malattia peptica, l'evidenza che la riduzione dell'acidità gastrica riducesse il rischio di sviluppare ulcera gastrica e/o duodenale e che la curasse in una larga maggioranza di casi aveva alimentato l'idea – poi rivelatasi inesatta – che i PPI e qualunque altra molecola in grado di ridurre/bloccare la secrezione acida gastrica fossero "gastroprotettori" [7]. L'erroneo concetto di "gastroprotezione" contribuisce ancora oggi a molte prescrizioni inappropriate da parte della classe medica e dei farmacisti e alla autoprescrizione. Un importante studio inglese del 2022 ha mostrato come il 50% delle prescrizioni dei PPI da parte del medico di medicina generale avvenisse senza un'indicazione clinica adeguata [8]. I principali predittori di un uso improprio erano: la terapia antibiotica, quella steroidea o la politerapia anche in assenza di uno specifico fattore di rischio, ovvero il ricovero in ambiente non intensivo e l'assenza di rischio di ulcere da stress [8]. Altrettanto comune è la prescrizione a chi assume farmaci antinfiammatori non steroidei senza avere fattori di rischio per malattia peptica, che ricordiamo essere: età >65 anni, storia di ulcera gastro/duodenale, infezione da *Helicobacter pylori*, oppure in coloro che assumono anti-aggreganti come prevenzione di incidenti vascolari. In quest'ultimo caso, si ricorda che l'acido acetilsalicilico a una dose inferiore a 250 mg ha solo un effetto anti-aggregante e non impatta sulla istoprotezione della barriera gastrica, in quanto inibisce la ciclo-ossigenali e non la lipo-ossigenasi [9]. Inoltre, circa un terzo delle terapie con PPI avviate per malattia da reflusso gastroesofageo e circa un decimo di quelle intraprese a scopo profilattico vengono proseguite per un tempo indeterminato, anche dopo la cessazione dell'indicazione originaria, evidenziando una tendenza al mantenimento ingiustificato del trattamento, nonostante tutte le linee guida, comprese quelle italiane più recenti [10], raccomandino una durata di 4-8 settimane, seguita da un tentativo di sospensione o di riduzione (*step-down*) secondo la minima dose efficace per il controllo dei sintomi del paziente.

L'UTILIZZO A LUNGO TERMINE DEGLI INIBITORI DI POMPA PROTONICA

Una crescente messe di evidenze ha messo in luce le conseguenze cliniche associate a un uso cronico di PPI e derivanti proprio dalla riduzione dell'acidità gastrica [11]. Diversi studi, infatti, hanno dimostrato un aumento significativo del rischio infettivo: in particolare, l'associazione con la colite da *Clostridium difficile* risulta tra le più solide, con un rischio fino a 2,9 volte maggiore nei pazienti in terapia con PPI rispetto ai non utilizzatori. Similmente, è stata dimostrata una correlazione con un aumentato numero di casi di polmonite acquisita in comunità e di infezione da *Streptococcus pneumoniae*, soprattutto durante le prime settimane dall'inizio della terapia con PPI, suggerendo come la soppressione della secrezione acida gastrica possa facilitare la colonizzazione e l'aspirazione di batteri patogeni dalle prime vie digestive [12]. Parallelamente, la modifica del pH gastrico determina importanti alterazioni della barriera antimicrobica intrinseca rappresentata proprio dalla secrezione acida, favorendo la sopravvivenza e la proliferazione delle specie microbiche provenienti dal cavo orale e presenti negli alimenti non sottoposti a cottura. In particolare, un ambiente gastrico meno acido permette il transito e la colonizzazione di microrganismi nel tenue, predisponendo allo sviluppo della contaminazione batterica del tenue (*Small Intestinal Bacterial Overgrowth*, SIBO) e della sindrome che ne consegue (malassorbimento di micro-macronutrienti, diarrea, dolori addominali, meteorismo, flatulenza, calo ponderale, anemia, osteoporosi, ecc.). La più recente metanalisi ha infatti evidenziato una prevalenza di SIBO pari al 36,8% nei pazienti trattati con PPI, significativamente superiore al 19,9% dei controlli, con un incremento del rischio del 4,265% per ogni mese aggiuntivo di terapia [13]. Ciò in quanto la mancanza di acido nell'ambiente gastrico compromette l'assorbimento di micronutrienti essenziali, quali il ferro nella forma non-eme, presente nelle fonti di origine vegetale e in molte preparazioni alimentari, che richiede un ambiente fortemente acido per essere convertito nella forma ferrosa assorbibile, e la vitamina B_{12}, la cui dissociazione dai complessi proteici alimentari e il legame con gli *R-bindings* dipendono dall'acidità gastrica [14]. Infine, l'uso prolungato dei PPI favorisce anche la disbiosi intestinale, ossia la presenza di specie patobionte rispetto a quelle eubionte che, a lungo andare, sono la concausa di un'alterata permeabilità intestinale e della perdita della tolleranza immunologica periferica alla base di tante patologie infiammatorie croniche che affliggono la nostra società [15].

Questi elementi di fisiopatologia anticipano e giustificano le conseguenze endocrine, minerali e scheletriche di un uso improprio e prolungato di PPI, che verranno approfondite nei paragrafi seguenti.

ALTERAZIONI DEL METABOLISMO MINERALE E VITAMINICO

L'uso a lungo termine dei PPI è oggi riconosciuto come un fattore di rischio per lo sviluppo di profonde alterazioni del metabolismo minerale, contribuendo all'insorgenza di osteoporosi [16]. Le più recenti evidenze provengono da studi multidisciplinari, che hanno chiarito sia i meccanismi endocrini sia gli effetti diretti dei PPI sul metabolismo osseo. Uno dei contributi più completi è rappresentato dal lavoro di Mu e collaboratori che, attraverso approcci integrati di *network toxicology*, *molecular docking* e *dynamic*, ha dimostrato come i PPI interagiscano con molecole regolatorie centrali nel rimodellamento osseo, quali *Epidermal Growth Factor Receptor*, *Estrogen Receptor-1* e *Src-family kinasee*, noti regolatori rispettivamente della proliferazione osteoblastica, della sensibilità estrogenica e dell'attività osteoclastica [17]. I complessi che si formano a seguito dell'interazione tra queste molecole con i loro ligandi hanno una stabilità conformazionale sufficiente a suggerire un potenziale effetto *off-target* sul metabolismo scheletrico, mediato dall'alterazione di vie rilevanti quali la segnalazione *Human Epidermal Growth Factor Receptor* e le vie di resistenza

endocrina, entrambe essenziali per il mantenimento dell'equilibrio tra formazione e riassorbimento osseo [17]. Sebbene tali evidenze molecolari rappresentino un contributo significativo alla comprensione dei meccanismi attraverso cui i PPI possano indurre l'osteoporosi, esse devono essere integrate con le ben note perturbazioni del metabolismo minerale e vitaminico associate al loro uso prolungato [16]. L'ipomagnesiemia cronica rappresenta una delle complicanze meglio documentate del trattamento protratto con PPI ed è attribuibile sia a una ridotta biodisponibilità intestinale sia a una disregolazione dell'assorbimento transcellulare mediato dai canali TRPM6/TRPM7 [18]. Tale deficit minerale, spesso silente nelle fasi iniziali, può manifestarsi clinicamente con dei disturbi neuromuscolari e cardiaci e risulta frequentemente refrattario alla supplementazione orale, ma tende a normalizzarsi rapidamente dopo la sospensione del trattamento. Poiché il magnesio rappresenta un cofattore essenziale per le reazioni di 25-idrossilazione epatica e 1-idrossilazione renale, la sua carenza determina una condizione di "resistenza funzionale" alla vitamina D, con una ridotta produzione di $1,25(OH)_2D$ e la conseguente compromissione dell'assorbimento intestinale del calcio [18]. Tale quadro può addirittura palesarsi con ipocalcemia lieve ma persistente e iperparatiroidismo secondario, che a sua volta determina un'accelerazione del turnover osseo. Contestualmente, la soppressione dell'acidità gastrica indotta dai PPI riduce la solubilizzazione dei sali di calcio, in particolare del carbonato, aggravando ulteriormente il deficit di assorbimento e contribuendo al quadro di iperparatiroidismo secondario. La copresenza di ridotta disponibilità di vitamina D attiva e di scarso assorbimento di calcio amplifica la vulnerabilità del tessuto osseo e può spiegare perché l'integrazione esclusiva di calcio non si traduca in un recupero significativo della densità minerale ossea nei pazienti in terapia cronica con PPI. La concomitanza dei meccanismi di interferenza diretta con punti chiave del *signaling* osteoblastico e osteoclastico, di disfunzioni endocrine calcio-vitamina D-dipendenti e di ipomagnesiemia iatrogena configura un fenotipo osteoporotico complesso e multifattoriale, che supera la semplice ipotesi dell'ipocloridria come unico meccanismo patogenetico associato ai PPI. È pertanto plausibile che la maggiore incidenza di fratture osservata nei pazienti in trattamento cronico con PPI derivi da una combinazione di effetti diretti, mediati da interazioni *ligand-target*, e indiretti, dovuti alla perturbazione dell'omeostasi minerale (Fig. 1).

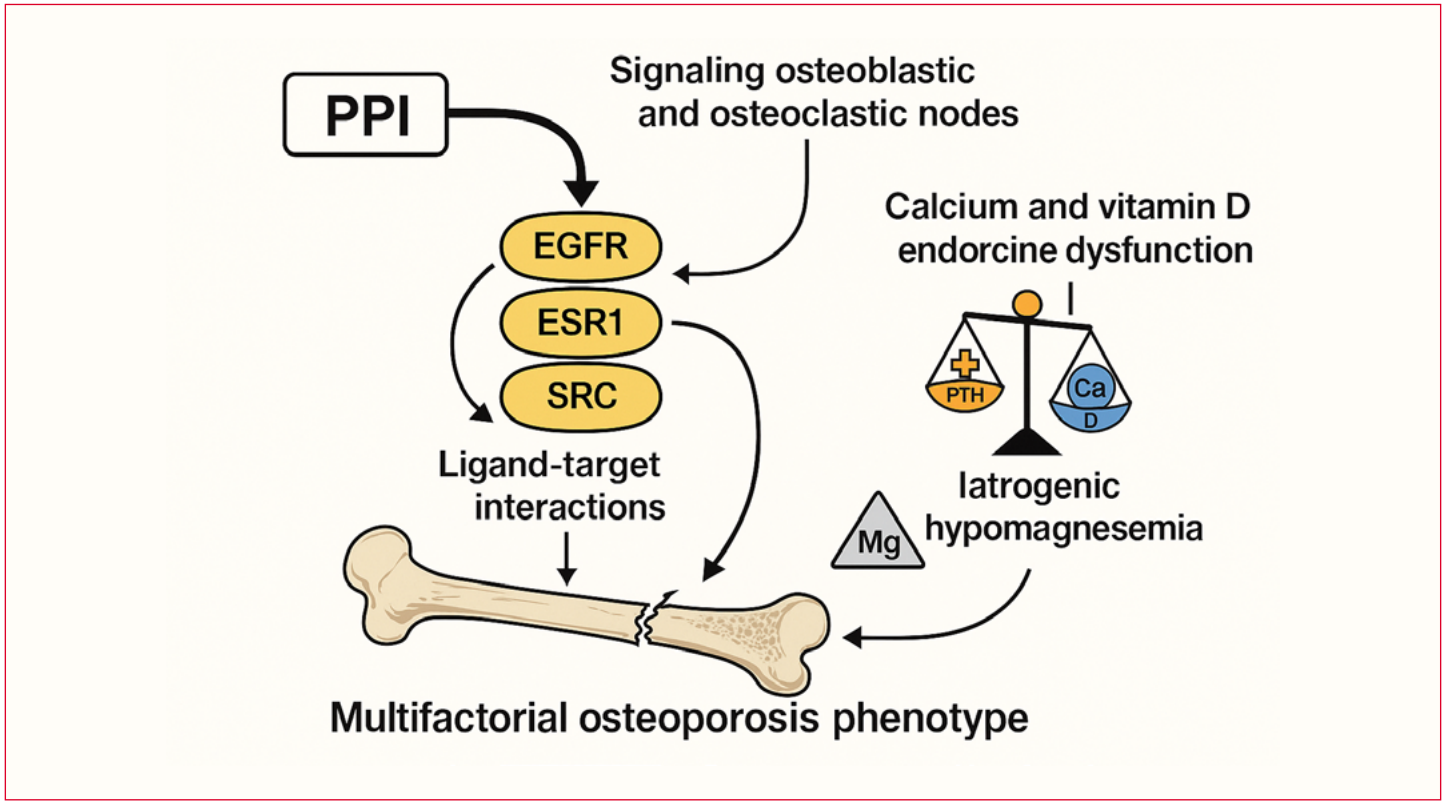

FIGURA 1.
Effetti degli inibitori di pompa protonica (PPI) sul metabolismo minerale e osseo. La ridotta disponibilità di magnesio compromette i canali di assorbimento TRPM6/TRPM7 (*Transient Receptor Potential Melastatin 6/7*) e ostacola la 25-idrossilazione epatica e la 1-idrossilazione renale, generando una "resistenza funzionale" alla vitamina D e il conseguente ridotto assorbimento del calcio. La soppressione dell'acidità gastrica riduce inoltre la solubilizzazione dei sali di calcio. È anche ipotizzata un'interferenza *off-target* di tali farmaci su EGFR (*Epidermal Growth Factor Receptor*), ESR1 (*Estrogen Receptor 1*) e SRC (*Proto-oncogene tyrosine-protein kinase Src*), interferendo con i processi di rimodellamento osseo e contribuendo a un fenotipo osteoporotico multifattoriale.

IL RUOLO DELLA SIBO – INDOTTA DA PPI – SUL MALASSORBIMENTO DELLA VITAMINA D

L'ipocloridria indotta dai PPI non altera soltanto la fisiologia digestiva, ma modifica profondamente anche l'ecosistema microbico gastrointestinale. La perdita della normale barriera acida, infatti, consente la sopravvivenza, la proliferazione e la migrazione di batteri verso il tenue, predisponendo allo sviluppo di SIBO [13]. Già nelle prime descrizioni della SIBO, veniva evidenziato come i pazienti sviluppassero deficit di micronutrienti liposolubili, inclusa la vitamina D, con delle manifestazioni cliniche quali osteomalacia e tetania. Il meccanismo fisiopatologico principale attraverso cui la SIBO interferisce con l'assorbimento dei lipidi è rappresentato dalla deconiugazione precoce dei sali biliari, operata dai batteri. Tale processo, fisiologicamente confinato al colon, viene patologicamente anticipato al digiuno e all'ileo, compromettendo la capacità dei sali biliari di formare micelle stabili e determinando una riduzione della emulsificazione dei lipidi. La vitamina D assunta attraverso la dieta necessita di essere incorporata nelle micelle di lecitina e sali biliari, per poter essere assorbita dagli enterociti dove viene trasportata attraverso la membrana apicale tramite trasportatori lipidici (NPC1L1, SR-B1, CD36), incorporata nei chilomicroni e, quindi, rilasciata nel sistema linfatico. La deconiugazione anticipata dei sali biliari ostacola queste fasi, riducendo in modo significativo l'assorbimento della vitamina D [19]. Oltre al meccanismo bile-dipendente, contribuiscono al deficit di vitamina D ulteriori alterazioni indotte dalla SIBO, come l'effetto diretto dei metaboliti batterici sulla mucosa intestinale e l'inattivazione degli enzimi espressi sulla membrana enterocitaria. Quest'ultimo meccanismo comporta una perdita o una riduzione del patrimonio enzimatico, compresa la lattasi, e il conseguente sviluppo di sintomi da intolleranza al lattosio, il cui assorbimento intestinale dipende dalla idrolisi operata dall'enzima beta-galattosidasi presente sull'orletto a spazzola degli enterociti e la cui mancata digestione determina delle importanti ripercussioni nutrizionali, metaboliche e immunologiche, dovute alla conseguente carenza di vitamina D [20]. Nel complesso, i dati disponibili supportano l'esistenza di un nesso fisiopatologico tra l'ipocloridria indotta dai PPI e la SIBO, e tra quest'ultima e il metabolismo scheletrico, attraverso dei meccanismi diretti (molecolari) e indiretti (digestivi e metabolici) sull'assorbimento lipidico, compresa la vitamina D, il calcio e il magnesio (Fig. 2).

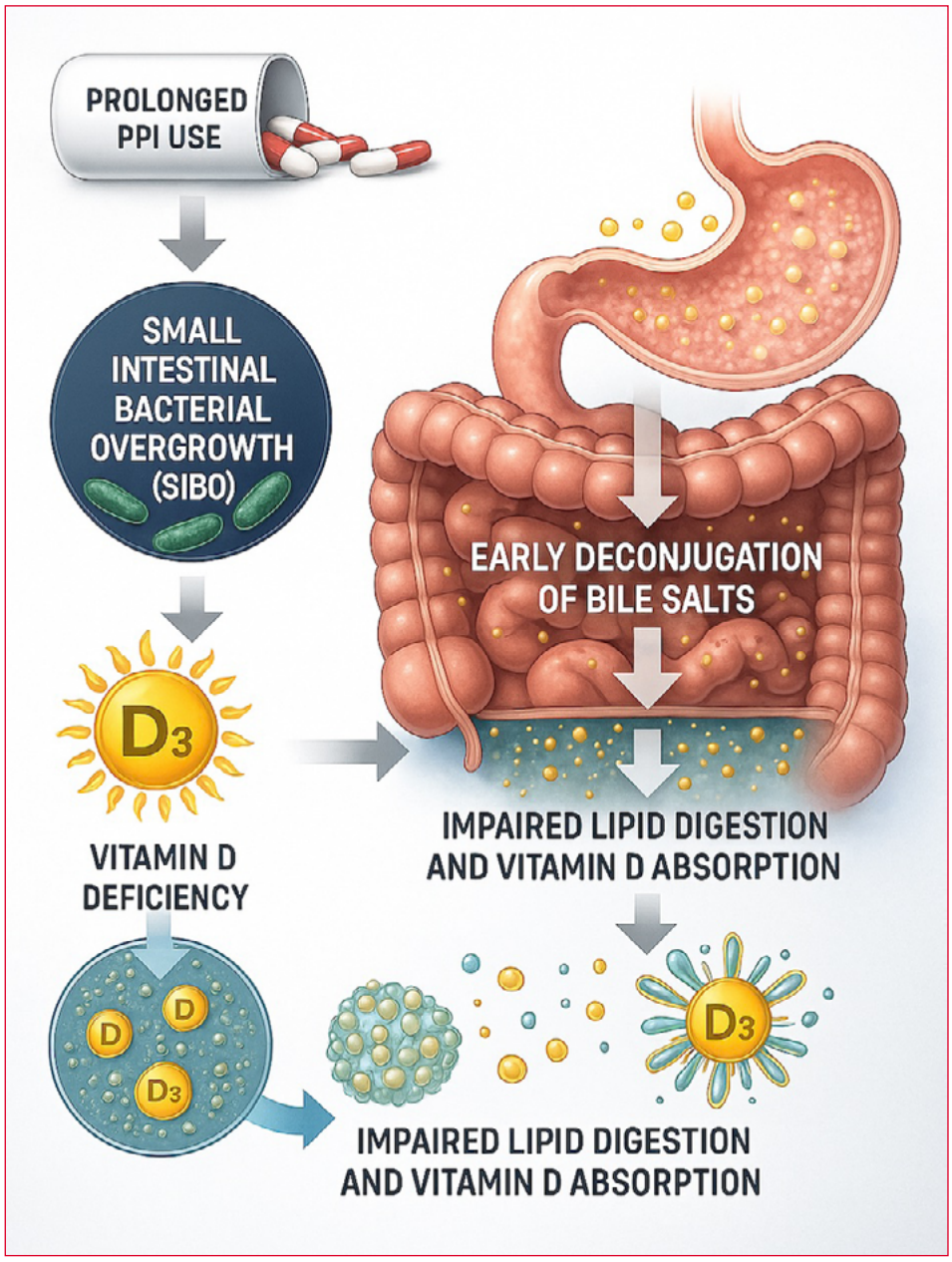

FIGURA 2.
Meccanismo attraverso cui l'ipocloridria indotta dagli inibitori di pompa protonica (PPI) favorisce la SIBO (*Small Intestinal Bacterial Overgrowth*) e compromette l'assorbimento della vitamina D. La proliferazione batterica determina deconiugazione precoce dei sali biliari, ostacolando la formazione delle micelle lipidiche e l'assorbimento enterocitario tramite i trasportatori NPC1L1 (*Niemann-Pick C1-Like 1*), SR-B1 (*Scavenger Receptor Class B Type 1*) e CD36 (*Cluster of Differentiation 36*). Le alterazioni dell'orletto a spazzola e la riduzione degli enzimi enterocitari, tra cui la lattasi, contribuiscono ulteriormente alla carenza di vitamina D, delineando una rilevante interferenza indiretta dei PPI sull'omeostasi minerale.

CONCLUSIONI

L'analisi integrata delle evidenze sino a oggi disponibili conferma che l'impiego cronico dei PPI rappresenta un fattore di rischio importante nell'indurre

un'alterazione dell'omeostasi intestinale, minerale ed endocrina. Emerge infatti un modello fisiopatologico complesso, in cui l'interferenza diretta dei PPI con nodi critici del *signaling* osteoblastico e osteoclastico si combina con profonde disfunzioni endocrine – in particolare del metabolismo di vitamina D e calcio – e con alterazioni sistemiche dell'omeostasi minerale – soprattutto di ferro, calcio e magnesio. Inoltre, la prevalenza di SIBO nei pazienti in trattamento cronico con PPI, con conseguente malassorbimento lipidico, la documentata ipomagnesiemia spesso refrattaria alla supplementazione e la ridotta attivazione della vitamina D delineano un quadro in cui la perdita di massa ossea rappresenta la convergenza di molteplici vie patogenetiche, la cui coesistenza crea un circolo vizioso che favorisce la demineralizzazione ossea, lo sviluppo di osteoporosi e l'aumentato rischio di fratture. Alla luce di tali evidenze, l'appropriatezza prescrittiva dei PPI diventa un elemento centrale, a cui deve seguire anche un controllo periodico sulla necessità di proseguire il trattamento e, ove possibile, stabilire strategie di *step-down* o di sospensione. Inoltre, l'identificazione dei pazienti a rischio – anziani, soggetti con carenze minerali, osteoporosi nota, politerapia – assume un ruolo prioritario nella prevenzione delle conseguenze di una terapia inappropriata con PPI. In conclusione, una maggiore consapevolezza delle implicazioni a lungo termine di tale terapia può contribuire a migliorare la loro appropriatezza prescrittiva e a ridurre le conseguenze sullo stato di salute.

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