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 PFAS e correlazione
con il metabolismo
della vitamina D

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In questo numero spaziamo dalla demenza all'inquinamento. Lo possiamo fare in tema di vitamina D perché vi sono recenti evidenze anche qui di un possibile ruolo.

Gli esperti Autori a cui abbiamo affidato l'approfondimento sul possibile rapporto tra carenza di vitamina D e demenza ci fanno notare come da studi in ambito di fisiopatologia neurologica vi siano indicazioni che la vitamina D possa esercitare numerose azioni a livello del sistema nervoso centrale e periferico, sintetizzabili in quattro effetti principali: supporto neurotrofico, neurotrasmissione, neuroprotezione, e neuroplasticità. Inoltre i dati epidemiologici disponibili sulla relazione tra stato vitaminico D e malattie neurologiche degenerative, come la demenza, sembrano supportare le evidenze descritte nei modelli animali perché generalmente descrivono una relazione inversa, tra l'altro di tipo "dose-risposta", tra i livelli sierici di 25(OH)D e il rischio di demenza. Tuttavia gli Autori ammettono che attualmente non esistono evidenze solide a supporto di un effetto preventivo o comunque positivo della supplementazione con vitamina D in questo campo, anche se non si può escludere considerate le importanti e svariate limitazioni degli studi sino a ora condotti. Concludono tuttavia saggiamente che trattandosi di soggetti generalmente anziani, dovrebbero trovare comunque giustificazione alla supplementazione considerati i riconosciuti benefici scheletrici, decisamente superiori ai costi e ai rischi di effetti indesiderati, ed, aggiungo io, la nota incapacità di produrre in età senile la fisiologica dose giornaliera di vitamina D.

Gli Autori del secondo articolo ci forniscono invece un originale contributo, anche sulla base di loro recenti studi, sulla possibile correlazione tra inquinamento e alterazioni del metabolismo della vitamina D. Hanno infatti osservato in particolare che le sostanze perfluoroalchiliche (PFAS), utilizzate principalmente per rendere resistenti ai grassi e all'acqua diversi tipi di materiali come tessuti, tappeti, rivestimenti, ecc. e causa di un diffuso e preoccupante inquinamento alimentare, specie in alcune aree del Veneto, possano interferire, per la loro similarità con gli ormoni steroidei, con i recettori per la vitamina D. Ne deriverebbe una ridotta risposta delle cellule scheletriche alla vitamina D, che si manifesterebbe con minor mineralizzazione ossea e con una risposta alterata dei geni sensibili alla vitamina D. Clinicamente lo testimonierebbe la maggior prevalenza di osteoporosi nelle popolazioni esposte a PFAS e il riscontro di livelli sierici mediamente superiori di paratormone, espressione di ipovitaminosi D funzionale. Capite anche perché, al contrario, una carenza subclinica e diffusa di vitamina D, che caratterizza la nostra popolazione, potrebbe rappresentare un fattore di suscettibilità agli effetti negativi sulla salute dell'esposizione a PFAS. Questi temi sono stati oggetto di una recente Commissione del Consiglio Superiore di Sanità, cui ho avuto il piacere di partecipare, che ha prodotto un documento di specifiche raccomandazioni che dovrebbe essere tra poco pubblicato nel sito del Ministero della Salute. Tra queste il consiglio di promuovere nelle popolazioni esposte all'inquinamento da PFAS il dosaggio dei livelli circolanti di 25(OH)D, dei suoi principali metaboliti

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e di biomarker della loro funzionalità nel metabolismo fosfo-calcico e osseo, uno screening densitometrico e la valutazione dell'incidenza di fratture da fragilità e di malattie extrascheletriche potenzialmente anch'esse correlate con il deficit assoluto o funzionale di vitamina D (in particolare cardiovascolari e immunologiche, la cui prevalenza pare effettivamente aumentata in soggetti esposti a PFAS) e l'eventuale realizzazione di studi che prevedano la supplementazione con vitamina D.

Il problema dell'inquinamento come sape- te è di grande attualità e di preoccupa- zione per le future generazioni. La nostra

Scuola ha recentemente osservato una correlazione tra inquinamento atmosferico (il particolare il particolato) e la prevalen- za di osteoporosi ¹ o la riattivazione di malattia ² e una minor risposta ai tratta- menti in pazienti affetti da artrite reumatoi- de ³. Che il deficit assoluto o funzionale di vitamina D abbia un ruolo anche nel giustificare queste correlazioni? Buon Natale e Buon Anno.

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Riassunto

Numerose evidenze derivanti da studi sperimentali, perlopiù condotti su modelli *in vitro* o modelli animali, sembrerebbero indicare come la vitamina D giochi un ruolo nella fisiologia e nella fisiopatologia del sistema nervoso, potenzialmente determinante anche nella patogenesi di alcune malattie degenerative, quali la demenza. La vitamina D sembrerebbe infatti esercitare un effetto neurotrofico, neuroprotettivo, e neuroplastico, e sarebbe coinvolta anche nella sintesi di alcuni neurotrasmettitori. I dati derivanti dagli studi prospettici osservazionali hanno confermato chiaramente le osservazioni sperimentali, dimostrando un'associazione inversa tra stato vitaminico D (concentrazione di 25-idrossi-vitamina D) e incidenza di demenza, con una relazione di tipo dose-risposta. A oggi, gli studi di intervento con colecalciferolo nella riduzione del rischio di demenza non hanno riportato risultati positivi, prevalentemente in relazione a significativi limiti in termini di disegno sperimentale, regimi terapeutici, numerosità della popolazione in esame e durata del follow-up. Studi disegnati *ad hoc* e metodologicamente più appropriati sono necessari per definire il potenziale effetto benefico del colecalciferolo nella prevenzione del rischio di demenza.

LA VITAMINA D E IL SISTEMA NERVOSO CENTRALE E PERIFERICO

Numerose evidenze scientifiche suggeriscono come la vitamina D giochi un ruolo nella fisiologia e nella fisiopatologia del sistema nervoso centrale (SNC) e periferico. È stato infatti ipotizzato come la deficienza di vitamina D possa giocare un ruolo nella patogenesi di alcune malattie neuro-degenerative, tra cui la demenza, la malattia di Parkinson, la sclerosi multipla e la sclerosi laterale amiotrofica¹⁻³. Numerose osservazioni supporterebbero un coinvolgimento della vitamina D nei processi fisiologici e fisiopatologici del SNC. Il recettore della vitamina D (VDR) è distribuito in maniera ubiquitaria nel SNC e periferico¹. La topografia della distribuzione del VDR, inizialmente definita nei ratti/criceti, è stata infatti successivamente confermata e precisata anche negli esseri umani¹. Il VDR sarebbe infatti espresso a livello dei neuroni e delle cellule gliali di numerose aree del sistema nervoso, tra cui la corteccia (ad es. temporale, frontale, parietale), il cervelletto, il midollo spinale e i nuclei della base¹. All'interno del SNC è stata anche identificata l'attività della 25-idrossilasi e della 1 α -idrossilasi,

indicative di una produzione paracrina di 1,25-di-idrossi-vitamina D [1,25(OH)₂D]¹⁻³. Gli stessi metaboliti della vitamina D sono stati identificati nel fluido cerebro-spinale¹⁻³. Infine, un'ulteriore dimostrazione dell'esistenza di un'attività produttiva paracrina della 1,25(OH)₂D all'interno del sistema nervoso deriva dall'osservazione che la concentrazione della 1,25(OH)₂D all'interno del SNC correla positivamente con la concentrazione plasmatica di 25-idrossi-vitamina D [25(OH)D], mentre non correla con la concentrazione plasmatica di 1,25(OH)₂D¹⁻³. Sulla base di queste e altre evidenze, è stato pertanto ipotizzato che la vitamina D possa esercitare numerose azioni a livello del SNC e periferico, sintetizzabili in quattro effetti principali: supporto neurotrofico, neurotrasmissione, neuroprotezione e neuroplasticità¹⁻³. La vitamina D eserciterebbe funzioni neurotrofiche correlate con la differenziazione, la maturazione e la crescita neuronale, attraverso ad esempio la stimolazione della sintesi di fattori neurotrofici quali il *nerve growth factor* (NGF), il *glial cell line-derived neurotrophic factor* (GDNF) o la *neurotrophin 3* (NT-3)¹⁻³. Analogamente avrebbe l'effetto sui livelli

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Conflitto di interessi

Gli Autori dichiarano nessun conflitto di interessi.

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(downregulation) di *neurotrophin 4* (NT-4) e la regolazione della espressione genica del recettore neurotrofico a bassa affinità del NGF (p75^{NTR})¹⁻³. A supporto di queste ipotesi e del ruolo neurotrofico della vitamina D, alcuni studi morfologici condotti in anziani sani o affetti da vario grado di deterioramento cognitivo hanno dimostrato una correlazione tra stato vitaminico D [definito dalla concentrazione plasmatica della 25(OH)D] e/o la deficienza di vitamina D, e il volume della sostanza grigia e dell'ippocampo^{4,5}.

La vitamina D e i suoi metaboliti sembrerebbero in grado di mediare anche la sintesi di una varietà di neurotrasmettitori, tra cui l'acetilcolina, le catecolamine, la serotonina e la dopamina¹. Questo effetto della vitamina D sembrerebbe essere duraturo nel tempo e soprattutto transgenerazionale. Infatti, una precoce esposizione a insufficienti/deficienti livelli di vitamina D potrebbe indurre alterazioni epigenetiche che a loro volta sarebbero in grado di influenzare l'espressione genica e con il tempo incrementare la suscettibilità a numerose malattie neurodegenerative (*metabolic imprinting*)^{1,6-10}.

L'effetto neuroprotettivo della vitamina D è stato oggetto di numerosi studi sperimentali condotti su modelli animali, dove la somministrazione di vitamina D o dei suoi metaboliti ha dimostrato di esercitare un effetto protettivo sui neuroni attraverso una riduzione del danno cellulare e della neurotossicità mediato da alcune sostanze notoriamente

neurotossiche^{1,11,12}. In uno studio *in vitro*, condotto su colture di cellule neuronali corticali, Annweiler et al. hanno dimostrato come la combinazione di memantina (farmaco utilizzato nel trattamento del deterioramento cognitivo) e vitamina D (ma anche la sola vitamina D) fosse in grado di attenuare e prevenire la degenerazione assonale prodotta dalla beta-amiloide e dal glutammato¹¹. I meccanismi alla base di questo effetto neuroprotettivo della vitamina D sono stati solo in parte chiariti e sono tuttora oggetto di discussione (ad es. regolazione flussi di calcio, effetto anti-infiammatorio, azione anti-ossidante)^{11,12}.

La vitamina D sembrerebbe essere in grado di influenzare la neuroplasticità attraverso la regolazione di geni aventi un impatto rilevante sullo sviluppo neuronale e su numerose funzioni neuronali (probabilmente già durante la gravidanza)^{1,13}. Una deficienza di vitamina D, ad esempio, sembrerebbe in grado di alterare il profilo trascrizionale di geni coinvolti nel mantenimento del citoscheletro, nella funzione mitocondriale, nella plasticità neuronale e nella proliferazione e crescita cellulare¹. Inoltre, come già descritto, una deficienza di vitamina D durante alcune fasi della gravidanza, potrebbe determinare alterazioni della regolazione della funzione neuronale (su base molecolare), capaci di influenzare la suscettibilità ad alcune malattie degenerative nell'età adulta¹³.

ESISTE UNA RELAZIONE TRA LA CONCENTRAZIONE DI 25(OH)D E IL RISCHIO DI DEMENZA?

I dati epidemiologici relativi alla relazione tra lo stato vitaminico D e le malattie neurodegenerative, in particolare la demenza, sembrano supportare pienamente le evidenze descritte derivanti dai modelli animali. Una recente *overview* (Tab. I) ha analizzato i risultati dei principali studi di revisione/meta-analisi relativi alla relazione tra lo stato vitaminico D e il rischio di demenza e/o malattia di Alzheimer (AD)³. Sebbene i risultati dei differenti studi presi in considerazione non fossero sempre facilmente interpretabili in relazione, prevalentemente, alla mancata standardizzazione delle valutazioni sierologiche e cliniche, complessivamente due aspetti di rilievo sono emersi in modo piuttosto omogeneo e consistente^{3,14-19}:

- esiste una relazione inversa tra la concentrazione di 25(OH)D e il rischio di demenza o AD;
- la relazione inversa tra la concentrazione di 25(OH)D e il rischio di demenza o AD sembrerebbe seguire il principio della "dose-risposta".

Ad esempio, Chen et al.¹⁴, hanno elaborato una meta-analisi di 10 studi di coorte, includendo circa 28.000 pazienti. Gli autori hanno identificato una correlazione inversa tra la concentrazione di 25(OH)D e il rischio di demenza [rischio relativo 0,72 comparando la categoria con la più alta

TABELLA I. Meta-analisi degli studi di coorte¹⁴⁻¹⁹ che hanno investigato la relazione tra stato vitaminico D [definito dalla concentrazione sierica della 25(OH)D] e declino cognitivo (da Maretzke et al., 2020, mod.)³.

Referenza	Studi inclusi	N. pazienti (età)	Cut-off 25(OH)D (nmol/l)	Outcome	Risultati principali (95% IC)
Chen (2018) ¹⁴	10 prospettici	28.640 (56-85 aa)	Alta vs bassa concentrazione 25(OH)D	Demenza e AD	RR demenza 0,72 (0,59-0,88) RR AD 0,78 (0,60-1,00)
Jayed (2018) ¹⁵	7 prospettici + 1 retrospettivo	28.354 (≥ 18 aa)	Insufficienza: 25-50 Deficienza: < 25	Demenza e AD	HR demenza per deficienza 1,33 (1,08-1,58) HR AD per deficienza 1,31 (0,98-1,65)
Goodwill (2017) ¹⁶	14 prospettici	30.000 (≥ 18 aa)	Alta vs bassa concentrazione 25(OH)D	Declino cognitivo	OR declino cognitivo 1,14 (1,06-1,23)
Cao (2016) ¹⁷	3 prospettici	12.702 (≥ 20 aa)	Alta vs bassa concentrazione 25(OH)D	Declino cognitivo	RR declino cognitivo 1,52 (1,17-1,98)
Shen (2015) ¹⁸	2 prospettivi	8086 (media 74 aa)	Deficienza: < 50	Demenza e AD	OR demenza 1,63 (1,09-2,16) OR AD 1,21 (1,01-1,40)
Annweiler (2013) ¹⁹	3 prospettivi	4095 (media 75 aa)	Alta vs bassa concentrazione 25(OH)D	Funzioni esecutive	OR per declino incidente 1,25 (1,05-1,48)

RR: rischio relativo; 95% IC: intervallo di confidenza 95%; HR: hazard ratio; OR: odd ratio; 25(OH)D: 25-idrossi-vitamina D sierica; AD: malattia di Alzheimer; aa: anni.

TABELLA II. Incidenza di demenza o malattia di Alzheimer in funzione della concentrazione basale di 25(OH)D (da Littlejohns et al., 2014, mod.)²⁰.

Demenza	N. partecipanti	N. casi	25(OH)D sierica (nmol/l)			P
			≥ 50	≥ 25 - < 50 HR (95% IC)	< 25 HR (95% IC)	
Demenza (qualunque tipo)						
Modello A*	1658	171	1	1,51 (1,06-2,16)	2,22 (1,23-4,02)	,002
Modello B**	1615	168	1	1,53 (1,06-2,21)	2,25 (1,23-4,13)	,002
Malattia di Alzheimer						
Modello A*	1589	102	1	1,67 (1,06-2,62)	2,27 (1,06-4,84)	,006
Modello B**	1547	100	1	1,69 (1,06-2,69)	2,22 (1,02-4,83)	,008

25(OH)D: 25-idrossi-vitamina D sierica; HR: hazard ratio; 95% IC: intervallo di confidenza 95%.

* Modello A: correzione (*Cox proportional hazards regression model*) per età e stagione in cui è stata dosata la 25(OH)D. ** Modello B: correzione (*Cox proportional hazards regression model*) per età, stagione in cui è stata dosata la 25(OH)D, scolarità, genere, indice di massa corporea, fumo, consumo di alcol e sintomi depressivi.

concentrazione di 25(OH)D con la categoria con la più bassa concentrazione di 25(OH)D e di AD [rischio relativo 0,78, comparando la categoria con la più alta concentrazione di 25(OH)D con la categoria con la più bassa concentrazione di 25(OH)D]. Inoltre, analizzando l'effetto dose [concentrazione di 25(OH)D] risposta, gli autori hanno anche dimostrato come il rischio di demenza o di AD diminuisse rispettivamente del 5 e del 7% per ogni incremento di 10 nmol/l della concentrazione della 25(OH)D¹⁴.

In linea con quanto descritto da Chen et al. e in altri studi analoghi (Tab I)¹⁴⁻¹⁹, uno studio longitudinale meno recente (Tab. II)²⁰, che ha considerato 1.658 anziani ambulatoriali che non presentavano (al momento dell'arruolamento) demenza, malattie cardiovascolari o cerebrovascolari, ha dimostrato un'incidenza di demenza e/o AD (durante un periodo di osservazione medio di 5,6 anni, range 0,1-8,4 anni) superiore nei soggetti con stato vitaminico D deficiente (< 50 nmol/l) o severamente deficiente (< 25 nmol/l), al momento dell'arruolamento, rispetto ai soggetti considerati avere una concentrazione di 25(OH)D nel range della sufficienza²⁰. Altri studi hanno confermato questi rilievi, evidenziando risultati consistenti soprattutto per valori di 25(OH)D < 25 nmol/l (deficienza severa di vitamina D)³. Per valori superiori a tale cut-off (ad es., compresi tra 25 nmol/l e 50 nmol/l) i risultati in favore della 25(OH)D sembrerebbero meno omogenei e consistenti.

SUPPLEMENTAZIONE CON COLECALCIFEROLO

Alla luce dei dati sperimentali (modelli animali) e di quelli derivanti dagli studi epidemiologici è stato ipotizzato un ruolo, ovviamente non di primo piano, della supplementazione con colecalciferolo nella prevenzione delle malattie neurodegenerative e in particolare della demenza³. Gli studi randomizzati e controllati, quelli osservazionali pre-post e le loro meta-analisi non hanno dimostrato un effetto significativo della supplementazione con colecalciferolo sui principali parametri cognitivi presi in esame. Questi studi presentavano tuttavia delle limitazioni significative e determinanti nell'interpretazione dei risultati³. Sia gli studi randomizzati e controllati che quelli osservazionali pre-post erano estremamente eterogenei in termini di disegno sperimentale e di regime terapeutico impiegato: i dosaggi di colecalciferolo impiegati variavano infatti da 400 UI al giorno (dosaggio verosimilmente troppo basso) a 5.000 UI al giorno, con anche bolus da 600.000 UI (inappropriati). La durata della supplementazione e il periodo di follow-up erano piuttosto brevi nella maggior parte degli studi, non sufficienti in considerazione della complessa fisiopatologia di demenza/AD e pertanto inappropriati per testare il potenziale effetto protettivo del colecalciferolo sul rischio di demenza/AD. Infine, in alcuni *trial* la numerosità dei pazienti ridotta risultava inadeguata per testare l'ipotesi oggetto dello studio. In considerazione delle suddette limitazioni, sebbene al momento non esistano evidenze solide in supporto di un effetto preventivo

o comunque benefico della supplementazione con colecalciferolo nella demenza/AD, non si può escludere completamente tale potenziale beneficio e studi randomizzati e controllati disegnati *ad hoc* saranno necessari nel futuro per chiarire il potenziale della supplementazione con colecalciferolo nelle malattie neurodegenerative e in particolare nella demenza.

In conclusione, un'ultima considerazione merita di essere enfatizzata: i pazienti anziani sono quelli a maggior rischio di deterioramento cognitivo/demenza e ugualmente sono la popolazione con la più alta prevalenza di ipovitaminosi D; pertanto, questa categoria di pazienti fragili sono meritevoli sempre e comunque di essere trattati con colecalciferolo in considerazione del suo basso costo, della totale sicurezza e tollerabilità, e della grande efficacia nella prevenzione delle cadute e delle fratture, al di là dei potenziali, ma verosimili, benefici extra-scheletrici. Una dose giornaliera di mantenimento con 1.000 UI o 2.000 UI al giorno di colecalciferolo preceduta, ove indicato, da una dose di carico, sembrerebbe rappresentare la strategia più fisiologica per ottimizzare gli effetti scheletrici ed extra-scheletrici del colecalciferolo²¹.

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PFAS e correlazione con il metabolismo della vitamina D

VITAMIN D

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Riassunto

Le sostanze perfluoroalchiliche (PFAS) sono una classe di composti largamente utilizzata nell'industria e nei prodotti di consumo. Sono resistenti alla degradazione e tendono ad accumularsi nell'ambiente e negli esseri viventi con possibili effetti tossici. Questi inquinanti sono un problema di sanità pubblica soprattutto in alcune zone della Regione Veneto, ma sono state recentemente isolate in acque provenienti da altre zone d'Italia. L'acido perfluorooctanoico (PFOA) è la forma predominante nei campioni umani ed è stato dimostrato che induce gravi conseguenze sulla salute, come alterazioni neonatali, neurotossicità e immunotossicità. Studi tossicologici indicano che gli PFAS si accumulano nel tessuto osseo e ne alterano lo sviluppo. Studi epidemiologici hanno riportato una relazione inversa tra i livelli ematici di PFAS e la salute delle ossa, soprattutto in termini di densità minerale ossea (DMO). Osteopenia e osteoporosi sono state evidenziate in più coorti: dalle donne in post-menopausa fino ai giovani uomini. Essendo già dimostrata l'interazione tra questa classe di composti e alcuni recettori ormonali nucleari (come il recettore degli ormoni tiroidei e il recettore androgenico), è stata ipotizzata un'interazione anche con il recettore della vitamina D, il quale è fondamentale per una corretta regolazione del metabolismo fosfocalcico, il principale determinante della densità ossea. In questo studio vengono sintetizzate le evidenze sperimentali e cliniche a supporto dell'interferenza del PFOA sulla via di segnalazione della vitamina D.

INTRODUZIONE

Le sostanze perfluoroalchiliche sono molecole in grado di interferire con il sistema endocrino, ovvero appartengono alla categoria degli EDs (*endocrine disruptors*). Si classifica come interferente endocrino qualunque entità chimica o miscela di composti che sia in grado di interferire con un qualsiasi aspetto dell'azione ormonale e che sia quindi responsabile di variazioni dell'omeostasi¹. Gli EDs esercitano la loro tossicità promuovendo la crescita e lo sviluppo tissutale. È ben conosciuto il meccanismo di interazione col sistema riproduttivo attraverso il legame di queste sostanze con il recettore androgenico (AR) ed estrogenico (ER). A seguito del legame tra l'interferente endocrino e il recettore si avrà come risposta un'azione di agonismo o antagonismo recettoriale, che si manifesta con aumento o diminuzione della risposta cellulare allo stimolo ormonale fisiologico².

LE SOSTANZE PERFLUOROALCHILICHE

Le sostanze perfluoroalchiliche (PFAS) sono una classe molto ampia di molecole organiche che appartengono alla categoria dei composti polifluorinati. Sono molecole prodotte artificialmente e non presenti in natura. Nella struttura dei PFAS è presente uno scheletro idrocarburico nel quale tutti gli atomi di idrogeno sono sostituiti con atomi di fluoro. La presenza del fluoro permette loro di acquisire delle caratteristiche fisico-chimiche particolari, prima fra tutte l'anfipilicità: presentano un lato apolare e uno polare, essendo contemporaneamente idrocarburi e acidi forti. Il lato polare contiene il gruppo funzionale che può essere un carbossile, un gruppo solforico, un gruppo alcoolico o svariati altri. Il gruppo funzionale polare e la lunghezza della catena fluorocarburica definiscono il singolo PFAS. Ad esempio, i due composti maggiormente studiati, perché più frequenti nelle aree inquinate, sono: l'acido perfluorooctanoico

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Conflitto di interessi

Gli Autori dichiarano nessun conflitto di interessi.

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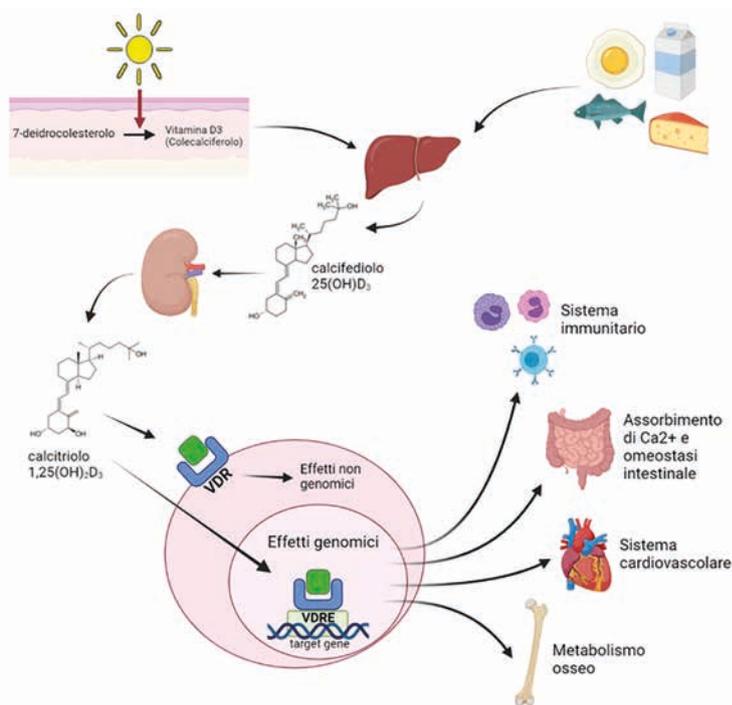


FIGURA 1.

Metabolismo della vitamina D: la vitamina D viene prodotta per via endogena dalla cute in seguito a esposizione solare o per via esogena tramite ingestione con la dieta. Il precursore della vitamina D (colecalfiferolo) viene quindi convertito in 25-idrossi-vitamina D (25(OH)D, calcifediolo) dall'enzima 25-idrossilasi a livello epatico. A sua volta, la 25(OH)D viene idrossilata a livello renale in posizione 1- α nel suo metabolita biologicamente attivo, 1,25-idrossi-vitamina D (1,25(OH)₂D, calcitriolo) esplica le sue funzioni biologiche su diversi organi agendo sul proprio recettore nucleare, che in seguito a legame con l'agonista migra nel nucleo e si lega a specifiche sequenze di riconoscimento (VDRE) a livello dei promotori dei geni bersaglio.

(PFOA) e l'acido perfluorottansolfonico (PFOS). Le principali fonti di esposizione possono essere l'ingestione di acqua potabile contaminata o di cibi con alti livelli di questi composti (ad esempio, pesce e frutti di mare). Svariati composti perfluoroalchilici sono stati ritrovati nei fluidi corporei: siero, liquido seminale, latte materno e persino nel cordone ombelicale, suggerendo che l'esposizione a tali composti duri tutta la vita fin dal suo principio. PFOA e PFOS inducono gravi conseguenze sulla salute umana come mortalità neonatale, neurotossicità e immunotossicità. Da dati sulla sorveglianza sanitaria negli Stati Uniti è emerso che i PFAS sono rintracciabili nel siero del 95% della popolazione³.

Nell'uomo, i livelli sierici di PFAS variano a seconda del livello di esposizione. Nella popolazione generale non esposta si ritrovano concentrazioni medie di 5,5 ng/mL per lo PFOA e 2,1 ng/mL per lo PFOS⁴. Nella popolazione residente nelle aree inquinate del-

la regione Veneto le concentrazioni di PFOA sono comprese tra i 54 e i 540 ng/mL⁵.

TOSSICITÀ SUL SISTEMA SCHELETRICO

Da circa un decennio è noto che il rischio di osteoporosi e fratture patologiche si associa a esposizione ad alcuni inquinanti ambientali (piombo, cadmio e mercurio). Tra gli inquinanti che interagiscono con il metabolismo osseo troviamo anche i PFAS. Pochi studi sono a oggi disponibili riguardo l'interazione di queste sostanze con il metabolismo osseo. Nei roditori sono stati riportate malformazioni ossee fetali con l'esposizione prenatale allo PFOS e, sempre nei topi, l'esposizione ambientale allo PFOS ne determina un rapido accumulo nel tessuto osseo. Nell'uomo è stata dimostrata la presenza di composti perfluoroalchilici (in prevalenza PFOS) a livello osseo mediante analisi di reperti scheletrici provenienti da autopsie di soggetti esposti alla contaminazione⁶.

Le analisi più recenti riguardano quelle ef-

fettuate dai due studi sulla salute della popolazione americana, in cui viene messa in evidenza la correlazione tra gli elevati livelli sierici di PFAS delle zone contaminate e la ridotta densità mineraria ossea, che variava in accordo al tipo di sostanza perfluoroalchilica considerata. Considerando più nello specifico i singoli PFAS, si nota una prevalenza più elevata di osteoporosi e una più bassa densità ossea a livello della tibia e del femore e un'alta prevalenza di osteoporosi tra le donne associata a PFOA, PFNA, e PFHxS. Più recentemente questi risultati sono stati confermati in altri studi sempre su soggetti adolescenti o giovani adulti^{7,8}.

Questi ultimi dati sono stati confermati in una coorte di giovani uomini (tra i 18 e 21 anni) provenienti dalle aree inquinate della Regione Veneto. Nei soggetti esposti si dimostra l'associazione tra l'esposizione a PFAS e il rischio di frattura⁹.

METABOLISMO DELLA VITAMINA D

La maggior parte della vitamina D3 è prodotta nella cute a partire dalla provitamina 7-deidrocolesterolo attraverso una reazione fotochimica che richiede l'intervento della componente UV della radiazione solare. Una volta sintetizzata nella cute, o assorbita dall'intestino, la vitamina D si trova in circolo legata alla sua proteina legante sierica (*D binding protein*, DBP), un' α -globulina sintetizzata dal fegato. Per essere biologicamente attiva la vitamina D3 necessita di una doppia idrossilazione (Fig. 1). La prima idrossilazione, in posizione 25, è svolta da enzimi mitocondriali e microsomiali che appartengono alla super famiglia del citocromo P450. La seconda idrossilazione è svolta nel rene da un enzima presente nel tubulo contorto prossimale, l'1 α -idrossilasi, anch'essa appartenente alla famiglia del citocromo P450 (CYP27B1). Questa idrossilasi, a differenza della precedente, è sottoposta a stretta regolazione: il paratormone e l'ipofosforemia l'attivano; il calcio, il fosfato, l'FGF-23 e il calcitriolo (meccanismo di feedback negativo) la inibiscono. Si ottiene così l'1,25-diidrossi-colecalfiferolo, o calcitriolo, il vero ormone derivato dalla vitamina D.

Il recettore della vitamina D (VDR) è espresso in numerosi tipi cellulari e tessuti. Il calcitriolo, la forma attiva della vitamina D, regola direttamente o indirettamente più di 200 geni coinvolti nella proliferazione cellulare, differenziazione, apoptosi e neoangiogenesi. È conveniente suddividere gli effetti biologici della vitamina D in scheletrici, ovvero

che riguardano il metabolismo fosfo-calcio, ed extra-scheletrici ¹⁰ (Fig. 1).

INTERFERENZA DEI PFAS SUL METABOLISMO DELLA VITAMINA D

Sebbene il numero di studi epidemiologici a conferma di un effetto negativo di queste sostanze sul metabolismo scheletrico sia sempre maggiore, non sono ancora del tutto dimostrati i meccanismi che possono indurre questa associazione. Un ormone fondamentale nello sviluppo scheletrico è la vitamina D, ormone steroideo che agisce stimolando il riassorbimento intestinale di calcio a favore di un'azione anabolica sull'osso. Diversi fattori esogeni sono noti influenzare i livelli di vitamina D circolanti, quali l'obesità, la dieta e l'inquinamento. L'omeostasi della vitamina D infatti può essere influenzata anche dagli interferenti endocrini poiché il metabolita biologicamente attivo, la 1,25-idrossivitamina D, è molto simile in struttura ai classici ormoni steroidei e il suo recettore nucleare è anch'esso paragonabile ai recettori degli ormoni tiroidei o steroidei. In due studi epidemiologici è stata, ad esempio, riportata un'associazione inversa tra bisfenolo A e ftalati rispetto ai livelli di vitamina D ¹¹. Data la similarità tra ormoni steroidei, e in particolare il testosterone, e la vitamina D, e tra i rispettivi recettori steroidei, in particolare il recettore per l'androgene, e il recettore per la vitamina D, si può ipotizzare che la già riportata interferenza dei PFAS sulla funzionalità degli ormoni steroidei possa essere estesa anche al metabolismo della vitamina D. Questo meccanismo potrebbe dimostrare le precedentemente riportate associazioni tra esposizione ai PFAS e alterato sviluppo scheletrico e osteoporosi. Sulla base di queste evidenze, è stato ipotizzato un ruolo dei PFAS nell'alterare il metabolismo della vitamina D. Questo meccanismo potrebbe rappresentare una delle possibili modalità di alterazione scheletrica indotta da queste sostanze. L'omeostasi della vitamina D potrebbe essere influenzata dagli interferenti endocrini, poiché questo ormone ha origine steroidea ed è già stata dimostrata interferenza endocrina dei PFAS nei confronti dei recettori steroidei, come ad esempio il recettore androgenico ¹².

Un recentissimo studio del gruppo del prof. Foresta ¹³ ha dimostrato che i PFAS interferiscono con il recettore della vitamina D, inducendo una ridotta risposta delle cellule scheletriche alla vitamina D stessa, che si manifesta con una minor mineraliz-

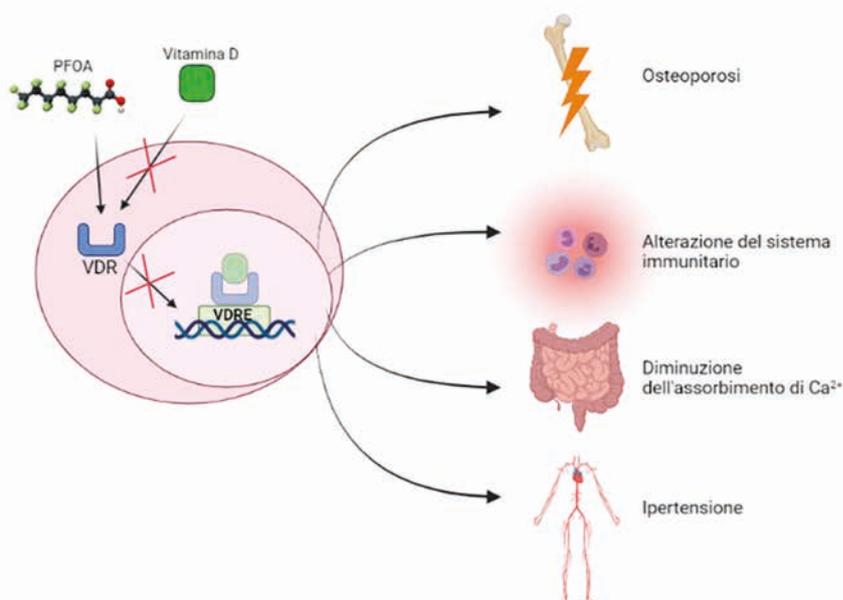


FIGURA 2.

Meccanismo di interferenza endocrina dei PFAS sulla vitamina D: i PFAS inibiscono il legame della vitamina D al suo recettore (VDR), impedendogli di legarsi ai promotori dei geni bersaglio. Questa interferenza comporta uno stato di ipovitaminosi D funzionale in cui, anche a fronte di normali livelli di vitamina D, questa non è in grado di svolgere la sua funzione biologica nelle cellule bersaglio. Questo meccanismo può spiegare le diverse manifestazioni cliniche osservate in popolazioni esposte a inquinamento da PFAS e connesse all'attività della vitamina D stessa, come osteoporosi, ridotta risposta immunitaria, ridotto assorbimento del calcio e problemi cardiovascolari.

zazione ossea (Fig. 2). In primo luogo, il PFOA compete con il calcitriolo nello stesso sito di legame del recettore della vitamina D (VDR), portando a un'alterazione della flessibilità strutturale del recettore. In secondo luogo, questa interferenza porta a una risposta alterata dei geni sensibili alla vitamina D in due popolazioni cellulari bersaglio di questo ormone, osteoblasti e cellule epiteliali del tratto coloretale. In terzo luogo, la mineralizzazione negli osteoblasti umani si riduce, in caso di coincubazione, con PFOA e calcitriolo. Infine, in una coorte di giovani uomini sani, la vitamina D non era diminuita nel gruppo esposto, ma i livelli di PTH erano più alti in associazione con l'esposizione a PFAS, suggerendo un meccanismo compensativo in risposta all'ipovitaminosi funzionale D. Complessivamente, questa evidenza trova un importante coinvolgimento fisiopatologico nella carenza di vitamina D associata all'esposizione ambientale a sostanze chimiche che alterano il sistema endocrino, e potrebbe spiegare le osservazioni epidemiologiche della ridotta massa ossea in questo contesto. Questi risultati, oltre a chiarire i meccanismi attraverso i quali i PFAS inter-

feriscono con l'attività di questo importante ormone, suggeriscono un possibile ruolo per questi inquinanti nella patogenesi dell'osteoporosi, la principale patologia correlata ai ridotti livelli di vitamina D.

CONCLUSIONI

Le evidenze epidemiologiche e sperimentali dimostrano che i PFAS alterano l'omeostasi della vitamina D e rappresentano quindi un fattore di rischio per il tessuto osseo in tutte le fasce di età, dall'età evolutiva (fase di crescita) alla post-menopausa, fase ad alto rischio per l'osteoporosi. Il monitoraggio dello status della vitamina D e della salute del sistema scheletrico è altamente raccomandato nelle popolazioni esposte. Nel contempo, la carenza subclinica di vitamina D (un problema diffuso nelle società occidentali) rappresenta un fattore di suscettibilità agli effetti dell'esposizione a PFAS. Pertanto, è particolarmente importante sviluppare campagne di sensibilizzazione e di prevenzione non farmacologica nelle popolazioni esposte, basate sulla promozione dell'attività fisica, della corretta esposizione alla luce solare e l'alimentazione.

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