PARKINSON DISEASE: THE NUTRIENTS USEFUL FOR PREVENTION AND CURE

Prof.ssa Mariangela Rondanelli

Dipartimento di Scienze Sanitarie Applicate e Psicocomportamentali, Sezione di Scienza dell'Alimentazione e Nutrizione Umana, Facoltà di Medicina e Chirurgia, Servizio Endocrino-Nutrizionale. Azienda di Servizi alla Persona di Pavia

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Parkinson disease: the enteric nervous system spills its guts.

Derkinderen P, Rouaud T, Lebouvier T, Bruley des Varannes S, Neunlist M, De Giorgio R.

Inserm U913, 1 place Alexis Ricordeau, CHU Nantes, 44093 Nantes Cedex 1, France. pascal.derkinderen@chu-nantes.fr

Lewy pathology in Parkinson disease (PD) extends well beyond the CNS, also affecting peripheral autonomic neuronal circuits, especially the enteric nervous system (ENS). The ENS is an integrative neuronal network also referred to as "the brain in the gut" because of its similarities to the CNS. We have recently shown that the ENS can be readily analyzed using routine colonic biopsies. This led us to propose that the ENS could represent a unique window to assess the neuropathology in living patients with PD. In this perspective, we discuss current evidence which indicates that the presence of ENS pathology may by exploited to improve our understanding and management of PD and likely other neurodegenerative disorders.

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Treatment approaches of gastrointestinal dysfunction in Parkinson's disease, therapeutical options and future perspectives.

Woitalla D, Goetze O.

Department of Neurology, St. Josef Hospital, Ruhr-University-Bochum, Germany, Dirk, Wolfalla@rub.de

Gastrointestinal (GI) dysfunction is a common but underestimated feature in Parkinson's disease (PD). Out of the multimodal spectrum of treatment options, there currently are only a few pharmacological treatments available to improve gastrointestinal motility and symptoms. Because enteric nervous function is mainly regulated by transmitters different from those involved in the brain, dopamine replacement is not a treatment option in PD patients. This article focuses on the known regulative mechanism of GI function and presents known and upcoming treatment options for GI dysfunction in PD.

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Prospective study of dietary pattern and risk of Parkinson disease.

Gao X, Chen H, Fung TT, Logroscino G, Schwarzschild MA, Hu FB, Ascherio A.

BACKGROUND: Several studies have shown associations between Parkinson Disease (PD) risk and individual foods and nutrients with inconsistent results.

OBJECTIVE: We examined associations between dietary patterns and risk of PD in the Health Professionals Follow-Up Study (1986-2002) and the Nurses' Health Study (1984-2000)

DESIGN: We included 49 692 men and 81 676 women free of PD at baseline and used principal components analysis to identify major dietary patterns and the Alternate Healthy Eating Index (AHEI) and the alternate Mediterranean Diet Score (aMed) to assess diet quality. Relative risks (RRs) were computed by using Cox proportional hazards models within each cohort and were pooled by using a random-effects model.

RESULTS: We documented 508 new PD cases after 16 y of follow-up. The principal components analysis identified 2 dietary patterns: prudent and Western. The prudent dietary pattern, characterized by high intakes of fruit, vegetables, and fish, was inversely associated with PD risk, but the Western pattern was not. The pooled multivariate-adjusted RR for the top compared with the bottom quintiles of the prudent score was 0.78 (95% Cl: 0.56, 1.07; P for trend = 0.04; For the AHEI, the pooled multivariate-adjusted RR for the top compared with the bottom quintile was 0.70 (95% Cl: 0.51, 0.94; P for trend = 0.01) and for aMED was 0.75 (95% Cl: 0.57, 1.00; P for trend = 0.07).

CONCLUSIONS: Dietary patterns with a high intake of fruit, vegetables, legumes, whole grains, nuts, fish, and poults low intake of saturated fat and a moderate intake of alcohol may protect against PD. Benefits of a plant-based dietar pattern including fish to PD merit further investigation.

JAMA Neurology

Prevalence of vitamin d insufficiency in patients with Parkinson disease and Alzheimer

disease.

EvatiMi. Delong MR. Khazai N. Rosen A. Triche S. Tanopricha V.

EvatiMi. Delong MR. Khazai N. Rosen A. Triche S. Tanopricha V.

EvatiMi. Delong MR. Allanta, GA 30329, USA. mevati@emory.edu

BACKGROUND: A role for vitamin D deficiency in Parkinson disease (PD) has recently been prop

CLINICAL NUTRITION

OBJECTIVE: To compare the prevalence of vitamin D deficiency in a research database cohort of patients with PD with the prevalence in age-matched healthy controls and patients with Alzheimer disease (AD).

DESIGN: Survey study and blinded comparison of plasma 25-hydroxyvitamin D (25[OH]D) concentrations of stored samples in a clinical research database at Emory University School of Medicine.

SETTING: Referral center (PD and AD patients), primary care clinics, and community setting (control

PARTICIPANTS: Participants were recruited into the study between May 1992 and March 2007. Every fifth consecutively enrolled PD patient was selected from the clinical research database. Unrelated AD (n = 97) and control (n = 99) participants were randomly selected from the database after matching for age, see, race, APOE genotype, and geographic locations. MAIN OUTCOME MEASURES: Prevalence of suboptimal vitamin D and mean 25(OH)D concentrations.

CONCLUSIONS: This report of 25(OH)D concentrations in a predominantly white PD cohort demonstrates a significantly higher prevalence of hyportaminosis in PD vs both healthy controls and patients with AD. These data support a possible of vitamin D insufficiency in PD. Further studies are needed to determine the factors contributing to these differences and elucidate the potential role of vitamin D in pathogenesis and clinical course of PD.

Nutrition Reviews



Nutr Rev. 2011 Sep;69(9):520-32. doi: 10.1111/j.1753-4887.2011.00413.x.

Prevalence of malnutrition in Parkinson's disease: a systematic review.

Sheard JM, Ash S, Silburn PA, Kerr GK,

Movement Neuroscience Program, Institute of Health and Biomedical Innovation, Queensland University of Technology, Brisbane, Queensland, Australia. jamie.sheard@qut.edu.au

Parkinson's disease (PD) patients may be at higher risk of malnutrition because of the symptoms associated with the disease and the side effects of the medication used to manage it. A decline in nutritional status is associated with many adverse outcomes related to health and quality of life. It is not clear, however, to what extent this population is currently affected by malnutrition. The objective of this review was to systematically assess the methodology and outcomes of studies reporting the prevalence of malnutrition in PD patients. Studies that attempted to classify participants with PD into nutritional risk and/or malnutrition categories using body mass index, weight change, anthropometric measures, and nutritional screening and assessment scores were included. The prevalence of palnutrition ranged from 0% to 24% in PD patients, while 3-60% of PD patients were reported to be at risk of malnutrition. There was a large degree of variation among studies in the methods chosen, the definition of malnutrition using those methods, and the detail in which the methodological protocols were reported. The true extent of malnutrition in the PD population has yet to be accurately quantified. It is important, however, to screen for abutrition at the time of PD diagno

Farmacocinetica della Levodopa





La Levodopa viene assorbita nell'intestino tenue

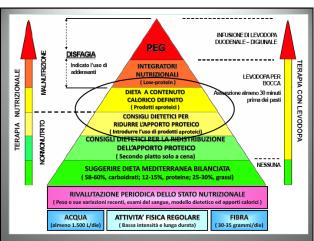
ma deve transitare nello stomaco dove può venire degradata dagli enzimi gastrici

Più tempo resta nello stomaco e più viene degradata

Fattori che rallentano lo svuotamento gastrico:

- presenza di grassi (e fibre) di un pasto
- farmaci anticolinergici
- acidità gastrica (eccesso o difetto)





Dual energy X-ray Absorptiometry (DEXA)

La densitometria con metodologia DEXA (Dual energy X-ray Absorptiometry: assorbimento a raggi X a doppia energia) è attualmente a livello mondiale il "gold-standard" nella valutazione della densità minerale ossea per la diagnosi di osteoporosi ed il "gold-standard" nella valutazione della composizione corporea.





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Curcumin-glucoside, a novel synthetic derivative of curcumin, inhibits α-synuclein

oligomer formation: relevance to Parkinson's disease.

Gadad BS, Subramanya PK, Pullabhatla S, Shantharam IS, Rao KS.

Department of Biochemistry and Nutrition, Central Food Technological Research Institute, Mysore-570020

Abstract

α-Synuclein aggregation is centrally implicated in Parkinson's disease (PD). It involves multi-step nucleated polymerization process via the formation of dimers, soluble toxic oligomers and insoluble fibrils. In the present study, we synthesized a novel compound viz., Curcumin-glucoside (Curc-gluc), a modified form of curcumin and studied its anti-aggregating potential with a-synuclein. Under aggregating conditions in vitro, Curc-gluc prevents oligomer formation as well as inhibits fibril formation indicating favorable stoichiometry for inhibition. The binding efficacies of Curc-gluc to both α-synuclein monomeric and oligomeric forms were characterized by micro-calorimetry. It was observed that titration of Curc-gluc with a-synuclein monomer yielded very low heat values with low binding while, in case of oligomers, Curc-gluc showed significant binding. Addition of Curc-gluc inhibited aggregation in a dosedependent manner and enhanced α-synuclein solubility, which propose that Curc-gluc solubilizes the oligomeric form by disintegrating preformed fibrils and this is a novel observation. Overall, the data suggest that Curc-gluc binds to α-synuclein oligomeric form and prevents further fibrillization of α-synuclein; this might aid the development of disease modifying agents in preventing or treating PD.