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High dietary fiber intake prevents stroke at a population level

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SUMMARY

Background & aims: This research was aimed at clarifying whether high dietary fiber intake has an impact on incidence and risk of stroke at a population level.
Methods: In 1647 unselected subjects, dietary fiber intake (DFI) was detected in a 12-year population-based study, using other dietary variables, anagraphics, biometrics, blood pressure, heart rate, blood lipids, glucose, insulin, uricaemia, fibrinogenaemia, erytrosedimentation rate, diabetes, insulin resistance, smoking, pulmonary disease and left ventricular hypertrophy as covariables.
Results: In adjusted Cox models, high DFI reduced the risk of stroke. In analysis based on quintiles of fiber intake adjusted for confounders, HR for incidence of stroke was lower when the daily intake of soluble fiber was >25 g or that of insoluble fiber was >47 g. In multivariate analyses, using these values as cut-off of DFI, the risk of stroke was lower in those intaking more that the cut-off of soluble (HR 0.31, 0.17–0.55) or insoluble (HR 0.35, 0.19–0.63) fiber. Incidence of stroke was also lower (−50%, $p < 0.003$ and −46%, $p < 0.01$, respectively).
Conclusions: Higher dietary DFI is inversely and independently associated to incidence and risk of stroke in general population.
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1. Introduction

The term «dietary fiber» was initially used to indicate non-digestible constituents making up the cell wall. More recently, an *ad hoc* committee[1] stated that dietary fiber must be considered as the remnants of the edible part of plants and analogous carbohydrates that are resistant to digestion and absorption in the human small intestine with complete or partial fermentation in the human large intestine, making fermentation the key factor for potential fiber activity.

The Western dietary pattern is disliked today, and some scientists openly sustain that the high prevalence of cardiovascular disease detected in Western societies is partially attributable to lifestyle, including low ingestion of fruit, vegetables and - more in general - fiber.[2,3] The belief that dietary fiber intake (DFI) is inversely associated to some diseases (the so-called «dietary fiber hypothesis») was postulated in the '70s and then enormously fuelled and kept alive by a great number of publications. The most important scientific societies warmly supported this hypothesis with recommendations having the value of clinical guidelines. Today it is therefore generally believed that high DFI prevents obesity, diabetes and cardiovascular disease.[4]

Nevertheless, many questions remain open. The fiber hypothesis, although substantiated by a limited number of controlled trials and epidemiological studies,[5–13] is mainly based on theoretical considerations and is still view *de facto* as a puzzle. Some studies on this topic are not population-based[5,14,15] or have poor phenotype,[10–12] and their results should be considered conditionally. It has also been whispered that the benefits could be different for soluble fiber (pectins) and insoluble fiber (mainly lignin and cellulose).[16] Furthermore, the recommended doses of DFI per day are different across countries and are often more theoretical than evidence-based. Finally, the recommendations derived from epidemiological studies are based on prevention of coronary disease,[17] cerebrovascular events were limitedly analyzed in this respect.[5,14,15] Consequently, information about a possible association between DFI and stroke is scanty.

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We therefore decided to tackle the topic of DFI and its relation with cerebrovascular disease in a population-based epidemiological context and in a longitudinal view. For this purpose, we tried to establish 1) whether in multivariate models DFI might be considered as an independent risk factor for stroke, 2) whether higher DFI is associated with lower risk of stroke and with lower incidence of stroke, 3) whether soluble fiber is different from insoluble fiber in this putative action, and 4) whether evidence-based longitudinally-derived cut-off values may be detected for soluble and insoluble DFI from population data, and used as recommendable dose.

2. Methods

2.1. Study population

All subjects aged ≥18 years living in the two Italian towns of Torrebelvicino and Valli del Pasubio were invited by letter for a screening in the frame of the Last Evidences Of Genetic Risk factors in the Aged (LEOGRA) study, an epidemiological project already largely described elsewhere.[18–20] In details, 1663 unselected subjects representative of the general population were recruited in 1989–2000 (participation rate 76%) and followed-up on visits at 3-year intervals for 12 years or until death by a staff of specialists. Of the original cohort, 16 subjects were excluded due to missing values on DFI, resulting in a representative sample of 1647 participants. No drop out was observed during the follow up. People who died for causes different from stroke were considered in the stroke free survival category.

The investigation conformed with the principles outlined in the Declaration of Helsinki and with institutional guidelines, and was approved by the Ethics Committees of the University of Padova and of the Local Health Unit No. 4 of the Veneto Region (Italy). Each subject gave and signed an informed consent.

Clinical evaluation and definitions. At baseline and at each follow-up visit, all subjects were invited to attend physical examination and underwent a Rose's questionnaire[21] about medical history, smoking and drinking habits. Menopausal status was determined in women by a detailed questionnaire asking for the age of cessation of menses.[22]

Blood pressure (BP) was measured in triplicate by means of a validated oscillometric device (Omron 705 IT, Omron Europe, Hoofddorp, Netherlands), using the appropriate cuff size, with the participants in the supine position. Average of the last two measurements was taken into account for the analysis of data. Body mass index was estimated as the body weight in kg divided by the squared height in m. Serum blood glucose, serum total cholesterol and the low-density lipoprotein fraction (LDLC), serum triglycerides, serum uric acid and erytrosedimentation rate were measured at fast by automated methods. Subjects then received a 75 g oral glucose load, and blood was taken again 1 h later for measurement of serum peak glucose and serum insulin.[23] The homeostasis model assessment index (HOMA-IR) was calculated (in arbitrary units) from [circulating insulin (in μU/ml) × fasting blood glucose (in mmol/l)]/22.5. Cut off values and definitions of diseases are summarized in Table 1.

All subjects underwent a standard 12-leads electrocardiogram and a 2-dimensional guided M-mode echocardiogram with a Megas device (Esaote, Firenze, Italia). Left ventricular end-diastolic diameter (LVEDD), end-diastolic inter-ventricular septum (EDIVS) and left ventricular posterior wall thickness (LVEDPWT) were measured according to the American Society of Echocardiography and Penn convention. Recordings were analyzed automatically during the exam using the inner software, and then analyzed off-line separately by an independent operator who did not know the aim and design of the study. A preliminary Bland–Altman analysis demonstrated a very good agreement between the two measurements (data not shown), so that the average of the two measurements was used for analysis of data. Left ventricular mass (in g) was calculated from $0.832 \times [(\text{EDIVS} + \text{LVEDD} + \text{LVEDPWT})^3 - (\text{LVID})^3] + 0{,}6$ and indexed for body surface area (in m^2) calculated from $71.84 \times \text{weight}_{kg}^{0.425} \times \text{height}^{0.725}$. Men having left ventricular mass index >125 g/m^2 and women >110 g/m^2 were considered as having left ventricular hypertrophy.

Dietary questionnaire. A 138-item food frequency detailed questionnaire previously validated for the Mediterranean diet[24] was administered at the initial screening. The reported frequencies of food intake per week were converted to number of intakes per day and multiplied by the weight of the portion size indicated. The mass of each dietary item was calculated. Each food was then resolved into its chemical components according to composition tables conceived for Italian food, where data were expressed as percent of food actually consumed after eliminating the scrap. Daily DFI was obtained, and expressed for total, soluble and insoluble components as g/day.

Daily alcohol intake was calculated in g ethanol from a detailed questionnaire asking for daily consumption of wine (ethanol 10–12%), beer (ethanol 3–7%), aperitifs (16–24%) and spirits (ethanol 33–46%).

Daily caffeine intake was calculated from number of cups of coffee and tea per day after ascertaining experimentally that 1 cup of espresso Italian coffee contains 80 mg.[25]

Assessment of events. The vital status of the LEOGRA participants and the International Classification of Diseases (ICD) codes, including causes of death, were obtained from the Italian Register's Office. The incidence of fatal diseases was then double-checked for causes of death by referring to hospitals, retirement homes or physicians' files. The incidence of nonfatal events was obtained *via* follow-up visits with repeated administration of the same standardized questionnaire used at baseline and *via* consultation of physicians and retirement homes records based on the ICD codes. In terms of coding, stroke was defined at baseline and at follow-up as ICD-9 codes 430–434 or 436 also based on clinical history or positive computer tomography or magnetic resonance imaging. Fatal and nonfatal stroke did not include transient ischemic attacks.

2.2. Statistical analysis

For database management and statistical analysis, the Statistical Analysis System (SAS) software, version 9.1 (SAS Institute, Cary, NC), was used. *A priori* power analysis based on previous experience of the same laboratory[18–21] indicated that 236 participants per group in equality for two proportions test and 19 participant per cell in analysis of variance were sufficient to show effects, if any, avoiding β error with a power of 0.95 and a test level of 0.05. Average difference (δ) between means and its standard deviation (σ) were used to calculate the centrality parameter $\Phi = 5 \times \delta/\sigma$. The magnitudes of the effect size were 17.8 (standardized 1.53) and 35.4 (standardized 1.56) for soluble and insoluble fiber intake, respectively. Linearity assumption was ascertained for each variables by the residuals method, and normality assumption by the Kolmogorov–Smirnov one-sample test.

Continuous variables were expressed as mean and standard deviation. After log-transformation, analysis of variance was used to compare grouped continuous variables, the χ^2 test to compare the prevalence of categorical variables, and the log rank of Wilcoxon to compare stroke-free survival between groups. Differences were considered statistically significant when p value was <0.05. Kaplan–Meier analysis was performed to compare stroke-free survival in groups of subjects.

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