



Does the Healthy Eating Index and Mediterranean Diet Score Identify the Nutritional Adequacy of Dietary Patterns in Chronic Pancreatitis?

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Abstract

Background Chronic pancreatitis (CP) is a progressive, irreversible disease characterized by maldigestion and frequently accompanied by epigastric pain, exocrine insufficiency, and/or endocrine insufficiency. There is limited information about the dietary patterns in CP from which to guide medical nutrition therapy recommendations.

Methods Study design was a cross-sectional, case–control study comparing subjects with CP ($n=52$) to healthy controls ($n=48$). Vioscreen™ food frequency questionnaire was used to assess the dietary pattern and nutrient intake in both groups. Dietary quality scores (the Healthy Eating Index, Mediterranean Diet score), and daily energy, macronutrient, and micronutrient intake levels were compared between groups.

Analysis Two sample t tests and Wilcoxon rank sum tests were used to evaluate differences in continuous variables, and Chi-squared tests were used for categorical variables.

Results CP was associated with a lower body mass index (BMI) (24 vs. 31 mg/kg²; $p<0.001$), lower HEI (57 vs. 65; $p=0.002$), and aMED scores (29 vs. 32; $p=0.043$) compared to healthy controls. Subjects with CP in the highest BMI quartile had the highest median aMED score compared to those in the lowest BMI quartile. There were no differences in kilocalories, macronutrients, or fat-soluble vitamin intake between groups, with the exception that vitamin K intake was lower in the CP group.

Conclusions The overall quality of dietary intake is lower in subjects with CP compared to controls when assessed by two independent nutritional measurement tools. Further research is needed to examine contributing factors, such as food insecurity and coexisting endocrine or exocrine insufficiency, to dietary patterns in patients with CP from which to guide evidence-based recommendations for medical nutritional therapy.

Keywords Chronic pancreatitis · Nutrition · Dietary patterns · Healthy eating index · Mediterranean diet

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Introduction/Background

Chronic pancreatitis (CP) is a complex disease that affects approximately 50 per 100,000 in the developed world [2]. In CP, there is an uncontrolled inflammatory cascade that produces a low level of local and systemic inflammation that ultimately contributes to pancreatic fibrosis [3–6]. The clinical manifestations and disease-related complications include abdominal pain, exocrine pancreatic insufficiency (EPI), diabetes mellitus (i.e., endocrine insufficiency), metabolic bone disease, and pancreatic cancer [7]. From a nutritional perspective, the varying combinations of abdominal pain, EPI, and diabetes can lead to reduced oral intake and maldigestion leading to unintentional weight loss, nutrient deficiencies, and malnutrition [6, 8–13].

There is very little known concerning the diet quality of patients with CP, and it is likely this is influenced by disease severity. Diet quality is a multidimensional term incorporating nutrient intake (i.e., micronutrients—vitamins/minerals, macronutrients—carbohydrate/protein/fat) and dietary patterns (i.e., consumption of food groups such as dairy, fruits, and vegetables) as compared to national recommendations [14]. Although nutrient intake and dietary pattern data are largely unknown in CP, there is an association with a Western-style dietary pattern in those with pancreatic cancer indicating a poor diet quality [15, 16]. An examination of nutrient intake and dietary patterns will make it possible for clinicians to target problem areas to prevent malnutrition and improve overall diet quality. Dietary patterns are typically assessed using numerous tools such as the food frequency questionnaire (FFQ). There are several limitations to the use of a FFQ including recall bias and difficulty in portion size estimation—both of which may impact the accuracy of information. Though still having recall bias, a more accurate alternative FFQ is the validated, online web-based dietary assessment tool. Vioscreen™ Graphical Food Frequency System (GraFFQ) generates an analysis of 90-day diet patterns and nutrient intake [17]. Each GraFFQ, compiled of food items selected from over 1200 photos and 3–6 portion sizes, is analyzed using branching logic, an analytics engine (Nutrition Data System for Research (NDSR)), and a participant-friendly interface, which generates an analysis of 90-day diet patterns and estimations of nutrient intakes [17]. Kristal et al. [17] and Thompson et al. [18] compared the Vioscreen™ to pencil and paper FFQs and found a significant reduction in time to complete the FFQ (30 vs 45 min) with similar reliability and validity.

Interpretation of dietary information gathered by an FFQ can be compared to population reference standards including the Healthy Eating Index (HEI) and Mediterranean diet score (aMED). The HEI and aMED scores are known dietary indices indicative of dietary patterns associated with reduced risk for chronic diseases [1]. Assessment of dietary patterns contrasted to these dietary indices provides a method for analyzing the overall dietary intake. The HEI scoring system is an assessment created by the US Department of Agriculture to measure diet quality in terms of compliance with the Dietary Guidelines for Americans (DGA) (see Supplemental Fig. 1) [19]. The DGA translates current scientific evidence to provide dietary recommendations constitutive of healthy diet patterns conferring health promotion and reduces chronic disease risk. The HEI provides quantification of an individual's eating patterns, potential areas of nutritional improvement, and allows for comparison to other populations. The HEI score ranges from 0 to 100; scores > 80 rated as “good,” 51–80 rated as “needs improvement,” and scores < 50 indicate a “poor” diet [20]. The HEI score is generated by assessing 13 components (see Supplemental

Table 1) each having a value (weighted equally) with maximum of 5 or 10 points; the summed scores maximum value of 100 points [20]. Alternatively, the aMED score provides the ability to determine level of adherence to a Mediterranean dietary intake pattern. The scoring system was developed by Tricopoulou et al. for the Greek population and was subsequently adapted for the US population (see Supplemental Table 2) [21, 22]. The Mediterranean dietary pattern has gained much interest for prevention of chronic diseases due to the decades of research linking this dietary pattern to a reduced risk of certain chronic diseases that are associated with chronic inflammation (e.g., cardiovascular disease and cancer) [23–29]. This diet includes daily intake of the following: non-refined cereals (whole grain products), fruits, vegetables, legumes, potatoes, fish, and olive oil. It also consists of rare consumption of the following: meat and meat products, poultry, and full fat dairy products, as well as moderate alcohol intake. The aMED score provides an estimate of adherence to these principles and also assesses the ratio of monounsaturated fat to saturated fat. The overall aMED score ranges from 0–9 with 7–9 indicating a high agreement with the Mediterranean diet pattern [21].

Historically, medical nutrition therapy interventions for CP have primarily focused on the coordination of dietary intake with pancreatic enzyme replacement therapy (PERT) and management of CP-related diabetes mellitus. Additionally, medium-chain triglyceride oil (MCT) is prescribed to supplement fat calories to those with CP experiencing steatorrhea. However, the high expense and unpleasant taste of MCT oil minimizes its use in clinical practice [30]. Those with steatorrhea are at an increased risk for fat-soluble vitamin deficiencies and therefore, require biochemical and physical assessments to determine the need for supplementation. Others have attempted low-fat dietary interventions (< 50 g/day) to minimize stimulation of the pancreas and lessen abdominal pain. However, this approach has not been successful in prevention of pancreatic stimulation and may lead to worsening of nutritional status due to inability to meet metabolic demand. Furthermore, it has not been routine for patients with CP to be referred to a registered dietitian nutritionist (RDN) except during hospitalization. More recent consensus guidelines support the need for consistent RDN involvement with the multi-disciplinary team management and that this should continue in the outpatient setting [9, 31, 32]. Personalized nutrition counseling by physicians is hindered by limited training (i.e., typically < 20 h of dedicated training in nutrition for medical school graduates) as well as insufficient time to perform a comprehensive nutritional assessment and counseling [33]. Comprehensive nutrition assessment for CP must be addressed in order to determine appropriate nutritional interventions. The goal of this study was to evaluate the dietary patterns of subjects with CP to determine if patterns vary from those of a

healthy population. Elucidating the dietary pattern and nutrient inadequacies will assist in the development of medical nutrition therapies to prevent long-term complications of malnutrition. The objectives of this study were to (1) utilize the VioScreen™ FFQ to describe the dietary patterns and nutritional intake of subjects with CP compared to healthy controls and population standards using the HEI and aMED scores, and (2) evaluate the mean nutrient intake for at-risk nutrients such as total dietary fat and fat-soluble vitamins A, D, E, and K.

Methods

Following review and approval from the Institutional Review Board at The Ohio State University Wexner Medical Center (OSUWMC), we performed a prospective case–control study. Cases were defined as subjects with CP diagnosed by a gastroenterologist, using recently published diagnostic guidelines from the American Pancreatic Association [34]. Healthy controls included patients presenting for an average risk screening colonoscopy or accompanying family members. Controls were excluded if there was a self-reported previous diagnosis of pancreatitis or other gastrointestinal disease. All study subjects were > 18 years of age. Medical records were retrospectively reviewed to obtain clinical data, including: demographics, data regarding CP (including radiology findings), and anthropometric data.

Study materials included the VioScreen™ FFQ, Two-Item Food Security Screener, Malnutrition Screening Tool (MST), and a demographic/health questionnaire. The HEI and aMED scores were calculated from FFQ data, representing the dietary pattern consumed over the previous 3 months. The Two-Item Food Security Screener is a validated tool developed from the USDA 18-item Household Food Security Survey as described by Hager et al. [35]. The questions: “Within the past 12 months, we worried whether our food would run out before we got money to buy more” and “Within the past 12 months the food we bought just didn’t last and we didn’t have money to get more” are used to identify at-risk individuals [35]. MST was routinely collected by the nursing staff at the time of clinic visits (see Supplemental Fig. 1). Those who scored ≥ 2 are considered at risk of malnutrition and were identified by study personnel. The VioScreen™ FFQ, Two-Item Food Security Screener, and study questionnaire were administered by study personnel. Those subjects who reported consuming < 500 kcals/day who were unable to complete the entire FFQ were excluded from analysis which is consistent with standard practice in dietary assessment.

Descriptive statistics were used to characterize the study population. Categorical variables were compared with Chi-squared tests, and continuous variables were compared

using t tests or Wilcoxon rank sum tests, as appropriate. All analyses were conducted with SAS 9.4 (Cary, NC), and a p value < 0.05 was considered to be statistically significant.

Results

Study Population

A total of 57 eligible subjects with CP and 51 eligible control participants agreed to participate in the study. After exclusions for incomplete or inaccurate data from the FFQ, the final study population consisted of 52 subjects with CP and 48 controls (see Fig. 1).

The study population had a mean age of 52 ± 14 years (Table 1). There was a male preponderance (67 vs. 40%, $p = 0.006$) and increased frequency of active cigarette smoking (40 vs. 6%, $p < 0.001$) in the CP group. In regard to body composition, the CP group had a significantly lower BMI compared to controls (24 vs. 31 mg/kg²; $p < 0.001$). Subjects in the CP group were also at increased risk for food insecurity compared to controls (17% vs. 2%, $p = 0.001$).

Although alcohol consumption was the most common etiology for CP (Table 2), the current alcohol consumption for both groups was low at a median of 0.02 g/day for those with CP and 1.71 g/day in controls (data not shown). Approximately 30% of CP subjects underwent prior surgical treatment, and nearly 73% were utilizing pancreatic enzyme replacement therapy (Table 2). According to MST, 16% of subjects with CP were at risk for malnutrition (Table 2).

Quality of Dietary Intake

Both HEI and aMED scores were significantly lower in the CP group than in the controls (HEI 57 vs. 65; $p = 0.002$ and aMED 29 vs. 32; $p = 0.043$; Table 3) indicating a less favorable dietary pattern in CP subjects. A post hoc analysis was performed by stratifying the CP group into BMI quartiles to assess the change in HEI score across the BMI classifications. This showed the highest HEI score (61.8) occurred in subjects within the second quartile of BMI classifications (including those of a normal body weight) (Fig. 2). Conversely, the lowest HEI scores were found in the third quartile and first quartile, 52.7 and 54.5, respectively.

Vioscreen™ FFQ Dietary Intake

Macronutrient and micronutrient intakes were also assessed in both groups using the Vioscreen™ FFQ (Table 4). Despite the lower BMI in the CP group, the caloric intake was similar to controls (1549 vs. 1664 kcal/day, $p = 0.692$). There was a trend toward higher vitamin D intake (5.88 vs. 4.03 mcg/day, $p = 0.054$) and lower intake

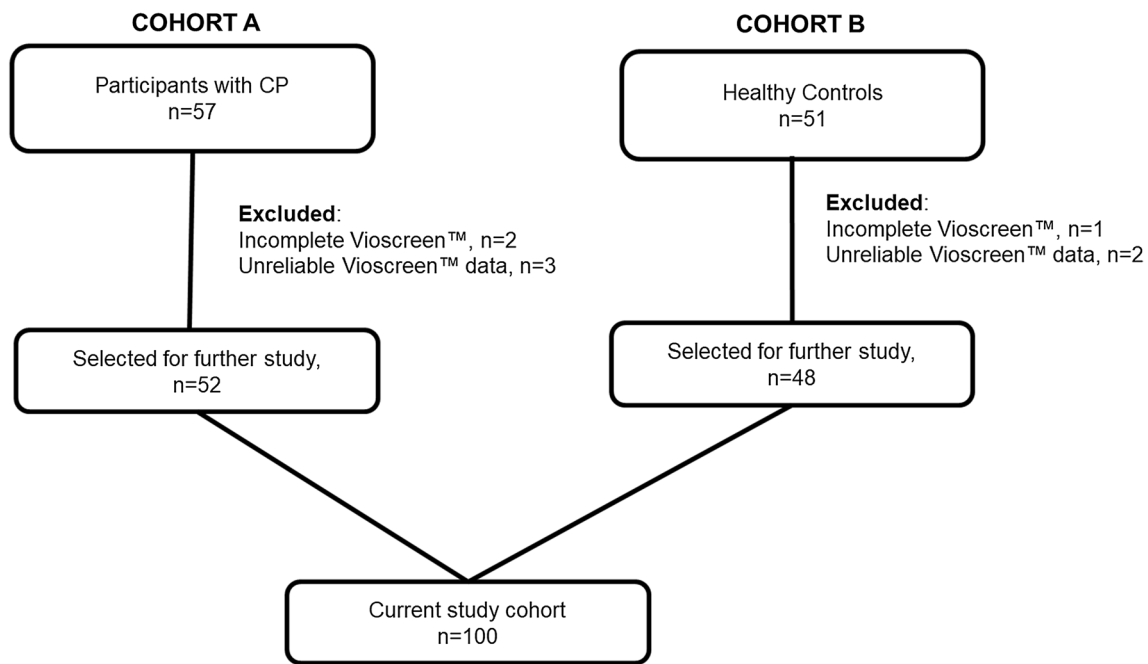


Fig. 1 Flowchart indicated participant accrual

Table 1 Study population characteristics (n = 100)

Variable	Total (n = 100)	CP (n = 52)	Controls (n = 48)	p value
Age, year, mean (SD)	52 ± 14	50 ± 15	54 ± 13	0.160
Male sex	54 (54)	35 (67)	19 (40)	0.006
Ongoing heavy alcohol use	10 (10)	7 (13)	3 (6)	0.230
Current smoker	24 (24)	21 (40)	3 (6)	< 0.001
Body composition (according to BMI)				
Body mass index, mean (SD)	28 ± 8	24 ± 6	31 ± 8	< 0.001
Underweight	9 (9)	8 (15)	1 (2)	
Normal	36 (36)	28 (54)	8 (17)	
Overweight	24 (24)	7 (14)	17 (35)	
Obese	31 (31)	9 (17)	22 (46)	
Food insecure	10 (10)	9 (17)	1 (2)	0.011

All data are presented as n(%) unless otherwise noted. CP chronic pancreatitis

of vitamin K (64 vs. 99.5 mg/day, $p = 0.01$) in CP subjects compared to controls. Otherwise, there were no difference in macronutrients (carbohydrate, protein, and fat) or vitamins A and E.

Finally, dietary patterns were assessed for differences in consumption of servings from the various food groups (Table 4). There were no differences in consumption of fruits, whole grains, vegetable-based protein, and sweet food. As reflected in the lower vitamin K intake levels, consumption of vegetables, including salads and salad

vegetables, was lower in the CP group. Lastly, estimated current alcohol usage was lower in CP subject compared to controls.

Discussion

In the current study, CP subjects are at increased risk for food insecurity and malnutrition, using standardized screening tools. Despite these risks, there is a knowledge gap in our understanding of dietary intake patterns in these patients. We demonstrate the feasibility of using the VioScreen™ FFQ to comprehensively characterize dietary patterns and nutritional intake in CP. The overall quality of dietary intake in CP is worse than study controls and the general population using both the HEI and aMED scores. Despite differences in body composition (estimated by BMI), the daily energy intake and macronutrient composition are similar in CP and controls. This study represents an initial step toward gaining a better understanding of the complexities of nutrition assessment and therapies in CP.

Current data to support the extent of outpatient medical nutrition therapy in those with CP are limited. So, the current focus during an RDN consultation is primarily limited to management of EPI- and CP-related diabetes mellitus. Subsequently, the medical nutrition therapy is focused on carbohydrate counting for those with diabetes and improvements in fat digestion in those with EPI. Other nutritional recommendations, such as vitamin/mineral/MCT

Table 2 Clinical data for study subjects with chronic pancreatitis

Variable	Chronic pancreatitis (n = 52)
Etiology of CP	
Alcoholic	21 (40)
Idiopathic	12 (23)
Genetic	3 (6)
Autoimmune	6 (12)
Recurrent and severe acute pancreatitis	5 (10)
Obstructive	2 (4)
Pancreas imaging	
Pancreatic calcifications	33 (63)
Dilation of main pancreatic duct	17 (33)
Calcifications and atrophy	26 (50)
Parenchymal atrophy	30 (58)
Previous pancreatic surgery	
Total pancreatectomy	5 (33)
Whipple	5 (33)
Distal pancreatectomy	3 (20)
Other (includes Frey or Peustow)	2 (14)
Current PERT usage	38 (73)
Mean PERT dosage (lipase units per meal)	56,868
Malnutrition screening tool^a	
Recently lost weight without trying	
No	40 (80)
Yes	10 (20)
Eating poorly due to decreased appetite	
No	35 (72.92)
Yes	13 (27.08)
MST score	
0	33 (67.35)
1	8 (16.33)
2	6 (12.24)
3	2 (4.08)
At risk of malnutrition (MST ≥ 2)	
No	42 (84)
Yes	8 (16)

All data presented as n (%) unless otherwise noted. PERT, pancreatic enzyme replacement therapy

^aMalnutrition screening data were missing for two CP subjects

Table 3 Comparison of Healthy Eating Index (HEI) and Mediterranean Diet (aMED) scores in those with and without Chronic Pancreatitis using the VioScreen™ Food Frequency Questionnaire (n = 100)

Dietary scores	Chronic Pan-creatitis (n = 52)	Controls (n = 48)	p value
HEI, mean ± SD	56.87 ± 13.56	65.42 ± 12.48	0.002
aMED, mean ± SD	29.44 ± 7.57	32.23 ± 5.85	0.043

supplementation and the use of a low-fat diet, vary widely. Despite this heterogeneity, the ultimate goal is to provide a nutrient-rich diet that is both calorically adequate and nutrient rich, which can be challenging due to a variety of previously discussed factors. Until the nutritional needs of those with CP are better understood, meeting the defined nutritional needs outlined by the dietary reference intakes (DRIs) provide initial target goals.

In this study, daily caloric intake was similar between those with CP and healthy controls; however, diet quality was worse in the CP group when measures according to adherence to DGAs (using HEI) and a Mediterranean diet (using aMED), indicating a suboptimal dietary pattern. The lower HEI and aMED scores in CP are partially attributed to lower consumption of vegetables, salad vegetables, and olive oil which contribute significantly to the overall HEI and aMED scoring system. Both groups had HEI scores in the “needs improvement” range indicating that the dietary pattern does not meet the DGA. Interestingly, there was no difference in the total dietary fat intake between subjects with both groups consuming a median intake consistent with the DGA. All fat-soluble vitamin intake was below that of the RDA indicating inadequate intake in a high-risk group with EPI. The implications of this are not addressed with this study. However, future study of blood vitamin levels and a nutrition-focused physical exam would permit further understanding of this observation. Overall, improving the dietary pattern via measurements of HEI and aMED might provide anti-inflammatory benefits that have been speculated in other studies of chronic disease [23–29]. Consultation with a RDN is imperative to providing this education since assessment of gastrointestinal tolerance to dietary fiber, fat, and carbohydrate needs to be individualized to improve patient compliance.

Poor appetite, abdominal pain, and/or steatorrhea inhibited adequate kilocalorie consumption in addition to disease-related metabolic derangements [36] may be the contributing factors associated with the lower BMI in CP. In our study, there was a ninefold increase in the prevalence of underweight classification (BMI < 18.5 kg/m²) in those with CP as well as a 2.5-fold decrease in the prevalence of overweight and obesity in those with CP. Interestingly, lower body mass distribution in CP was not associated with differences in caloric or fat intake; this discordance suggests potential contributions from either maldigestion or increased metabolic processes in the CP group. Unfortunately, body composition was not measured in this cohort, which would provide further insights into the metabolic demand of those with CP. Olesen and colleagues used indirect calorimetry to measure resting energy expenditure and bioelectrical impedance to measure body composition in stable CP (n = 28). A significant correlation between resting energy expenditure and fat-free mass was detected in those without nutritional

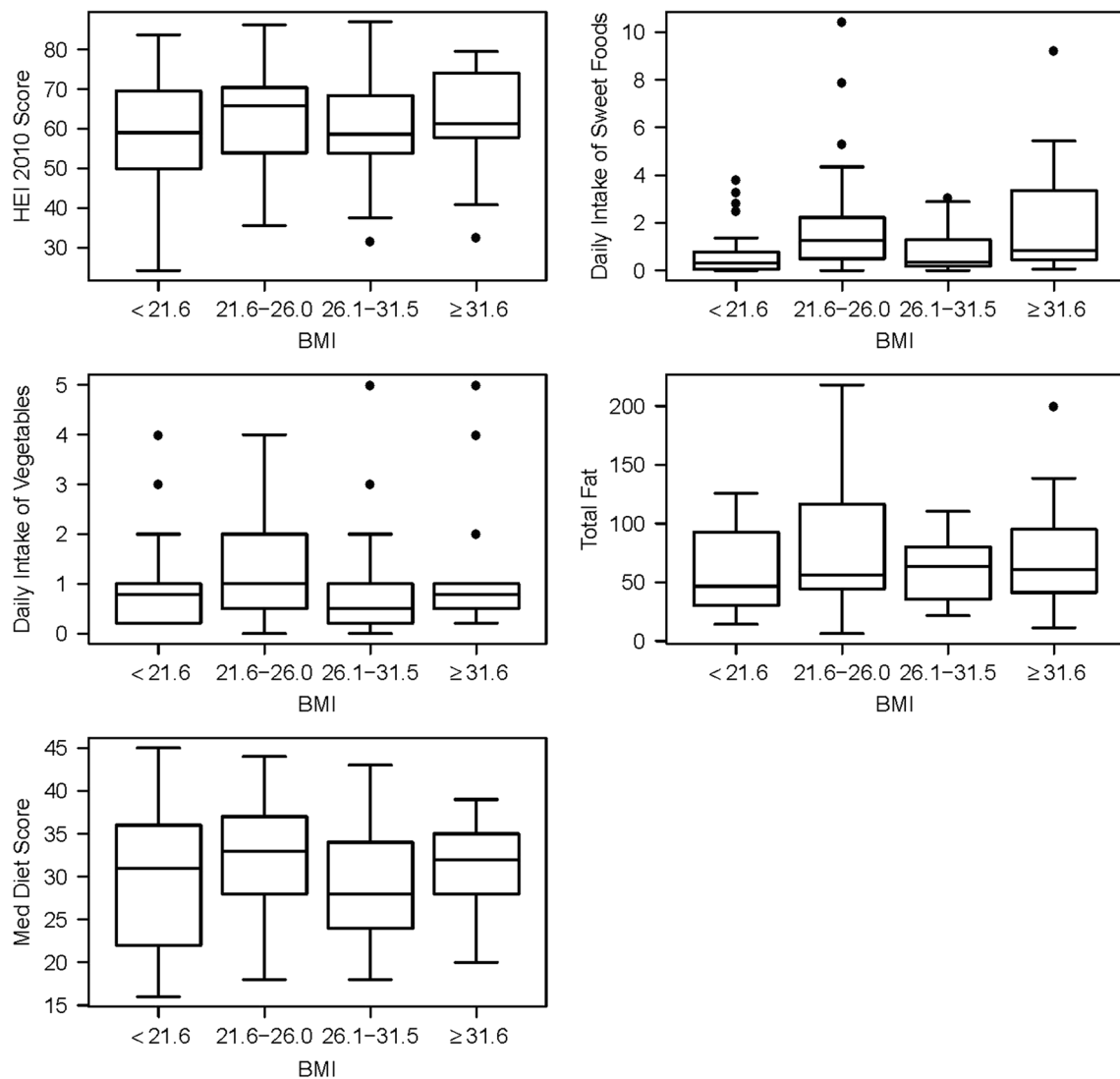


Fig. 2 Dietary characteristics among subjects ($n=52$) with CP stratified by BMI. Data represents the median, IQR, and outliers for each characteristic

risk ($\rho=0.45$; $p=0.02$), yet no significance was seen in those with nutritional risk ($\rho=0.45$; $p=0.2$). Furthermore, Hebuterne et al. [37] found an elevation in resting energy expenditure in 60% of underweight ($BMI < 20 \text{ mg/kg}^2$) subjects with alcohol-induced CP. Measurement of REE and body composition would enhance the understanding of dietary patterns in those with CP given that 16% of those with CP in this cohort were also identified to be at nutritional risk.

The MST was selected for identifying subjects at nutritional risk. This tool has been validated in a variety of patient populations and can be used to assist in identification of patients requiring a RDN referral to determine the presence of malnutrition. The Academy of Nutrition and Dietetics and American Society of Parenteral and Enteral Nutrition concur that the diagnosis of malnutrition can be made in the context of acute illness/injury, chronic illness/

injury, or social/behavioral/environmental situations when two of the following characteristics are met: weight loss, inadequate energy intake, depletion of body fat, depletion of muscle mass, fluid accumulation, or reduced handgrip strength (see Supplemental Table 1). The application of this malnutrition diagnostic criteria in an outpatient pancreatology clinic in correlation with dietary assessment data from a FFQ would provide the necessary foundation to elucidate the impact of dietary intake on the nutritional status of those with CP.

Dietary intake can also be affected by patient characteristics such as smoking, alcohol consumption, and food security. Within the present cohort, those with CP consumed less alcohol (even if alcohol was not the etiology for their disease), but smoked more cigarettes than the control population. Interestingly, a greater risk for food insecurity was

Table 4 Energy and nutrient intake from Vioscreen™ FFQ in the study population ($n = 100$)

	CP ($n = 52$)	Controls ($n = 48$)	<i>p</i> value
Total daily caloric intake (kcal), median	1549	1664	0.692
Total dietary fat (grams), median	51.89	62.14	0.899
Total dietary protein (grams), median	66.01	60.44	0.131
Total dietary carbohydrate (grams), median	208.2	181.36	0.176
Multivitamin usage, yes n (%)	29 (56)	23 (48)	0.432
Median Fat-soluble vitamin intake and levels			
Dietary Vitamin A intake (RAE), and % RDA*	744.41, 83%	651.25, 72%	0.692
Dietary Vitamin D intake (mcg), and % RDA*	5.88, 40%	4.03, 27%	0.054
Dietary Vitamin E intake (IU), and % RDA	11.77, 53%	11.54, 52%	0.937
Dietary Vitamin K intake(mg), and % AI*	64, 53%	99.5, 83%	0.010
Food group daily intake (mean servings/day)			
Fruits	1.78	1.74	0.265
Vegetables	1.78	2.99	<0.001
Whole grains	1.16	1.21	0.213
Vegetable-based protein (grams/day)	24.87	22.82	0.580
Sweet foods	1.83	0.99	0.068
Alcohol	0.23	0.91	0.002

*RDA was adjusted for the subject's sex for the vitamins that have an age-adjusted RDA

CP chronic pancreatitis, RAE retinol activity equivalent, RDA recommended dietary allowance, AI adequate intake

identified in the CP group. This finding was slightly higher than the national average for households experiencing food insecurity in 2015 (12.7%) but was comparable to the state of Ohio (16.1%) [38, 39]. Food insecurity leads to a disruption in eating patterns and reduced food intake for at least one member of the household which could also cause decreased intake of high-cost, yet nutrient-rich, foods such as fruits and vegetables [38]. This combination of food insecurity with a chronic disease such as CP leads to an even greater risk for nutritional deficiencies and malnutrition. Hager et al. stressed that when individuals and households are identified at risk for food insecurity, it allows providers to target services that combat the health risks associated with food insecurity [35]. Using this methodology to identify food insecurity as a component of the nutrition assessment protocol may help to mitigate the risk for nutritional deficiencies and malnutrition in those with CP.

This study demonstrates the feasibility of assessing the dietary pattern in those with CP. Given the complex endocrine and exocrine dysfunction, understanding the impact of dietary intake on body composition, energy expenditure, and the development of malnutrition is needed to identify appropriate targets for nutrition intervention. Future studies should be designed to complete a complex, nutritional assessment to determine the relationship to dietary patterns and nutrient intake. Lastly, screening for food insecurity and identification of patient resources may impact adherence to nutritional recommendations.

Conclusion

Given the high rate of malnutrition, abnormal body mass, and poor dietary pattern, patients with CP require personalized nutrition education to optimize their nutritional status. The heterogeneity of the CP population in terms of disease activity and dietary intake contributes to the challenge of implementing this in clinical practice. Evaluating energy intake and energy expenditure is needed to further clarify the relationships identified in the current study. For example, dietary fat is the most calorically dense nutrient and functions to transport fat-soluble vitamins. Dietary assessment can quantify intake, however, the optimal intake varies based on the degree of fat maldigestion. Therefore, dietary intake must be evaluated in conjunction with biochemical levels and a nutrition-focused physical exam to identify intake goals. Therefore, an objective measure of fat maldigestion would provide the RDN with the necessary tools to optimize the dietary pattern and initiate supplementation earlier. Assessing energy intake is not enough to describe the metabolic demand of the CP population. Future research will provide insights into the complex interactions of CP with dietary intake, energy expenditure, body composition, and food security and will lead to development of the optimal standard for nutritional management in CP.

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Compliance with ethical standards

Conflict of interest Marcia Nahikian-Nelms has received funding from ChiRhoClin Research Institute and the Medical Nutrition Therapy Practice Group of the Academy of Nutrition and Dietetics. Peter Madril is a consultant for Viocare®. Paige Golian, Kristen Roberts, Alice Hinton, Kathleen Basch, Darwin Conwell, and Phil Hart have no conflicts of interest.

References

1. Agriculture, U.S.D.O., *A Series of Systematic Reviews on the Relationship Between Dietary Patterns and Health Outcomes*. 2014: Virginia.
2. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013;144:1252–1261.
3. Gubergrits NB, Linevskiy YV, Lukashevich GM, et al. Morphological and functional alterations of small intestine in chronic pancreatitis. *JOP*. 2012;13:519–528.
4. Pezzilli R. Chronic pancreatitis: maldigestion, intestinal ecology and intestinal inflammation. *World J Gastroenterol*. 2009;15:1673–1676.
5. Stevens T, Conwell DL, Zuccaro G. Pathogenesis of chronic pancreatitis: an evidence-based review of past theories and recent developments. *Am J Gastroenterol*. 2004;99:2256–2270.
6. Nahikian-Nelms M. *Nutrition Therapy and Pathophysiology*. 3rd ed. Boston: Cengage Learning; 2014.
7. Ramsey ML, Conwell DL, Hart PA. Complications of chronic pancreatitis. *Dig Dis Sci*. 2017;62:1745–1750. <https://doi.org/10.1007/s10620-017-4518-x>.
8. Lindkvist B, Phillips ME, Dominguez-Munoz JE. Clinical, anthropometric and laboratory nutritional markers of pancreatic exocrine insufficiency: prevalence and diagnostic use. *Pancreatology*. 2015;15:589–597.
9. Rasmussen HH, Irtun O, Olesen SS, et al. Nutrition in chronic pancreatitis. *World J Gastroenterol*. 2013;19:7267–7275.
10. Afghani E, Sinha A, Singh VK. An overview of the diagnosis and management of nutrition in chronic pancreatitis. *Nutr Clin Pract*. 2014;29:295–311.
11. Widmaier EP, Raff H, Strang KT. *Vander's Human Physiology the Mechanisms of Body Function*. 11th ed. New York: McGraw Hill; 2008.
12. Lieberman, P.M., Marks A.D., *Mark's Basic Medical Biochemistry: A Clinical Approach*. 3rd ed. 2009, Baltimore: Lippincott, Williams, and Wilkins.
13. Duggan SN, Smyth ND, O'Sullivan M, et al. The prevalence of malnutrition and fat-soluble vitamin deficiencies in chronic pancreatitis. *Nutr Clin Pract*. 2014;29:348–354.
14. Alkerwi A. Diet quality concept. *Nutrition*. 2014;30:613–618.
15. Turner RC, Brazionis LB, McDermott R. Intake patterns of food nutrients and other substances associated with chronic pancreatitis. *Pancreatology*. 2013;13:33–37.
16. Salem AA, Mackenzie GG. Pancreatic cancer: a critical review of dietary risk. *Nutr Res*. 2018;52:1–13.
17. Kristal AR, Kolar AS, Fisher JL, et al. Evaluation of web-based, self-administered, graphical food frequency questionnaire. *J Acad Nutr Diet*. 2014;14:613–621.
18. Thompson FE, Kipnis V, Midthune D, et al. Performance of a food-frequency questionnaire in the US NIH-AARP (National Institutes of Health-American Association of Retired Persons) Diet and Health Study. *Public Health Nutr*. 2008;11:183–195.
19. Huffman FG, De La Cera M, Vaccaro JA, et al. Healthy eating index and alternate healthy eating index among haitian Americans and African Americans with and without Type 2 diabetes. *J Nutr Metab*. 2011;2011:398324.
20. Basiotis P, Carlson, A., Gerrior S, Juan W, Lino M, *The Healthy Eating Index: 1999–2000*. 2002: Washington, DC: USDA, Center for Nutrition Policy and Promotion.
21. Trichopoulou A, Costacou T, Bamia C, et al. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med*. 2003;348:2599–2608.
22. Fung TT, McCullough ML, Newby PK, et al. Diet-quality scores and plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr*. 2005;82:163–173.
23. Tyrovolas S, Panagiotakos DB. The role of Mediterranean type of diet on the development of cancer and cardiovascular disease, in the elderly: a systematic review. *Maturitas*. 2010;65:122–130.
24. Kontogianni MD, Tileli N, Margariti A, et al. Adherence to the Mediterranean diet is associated with the severity of nonalcoholic fatty liver disease. *Clin Nutr*. 2014;33:678–683.
25. Schroder H. Protective mechanisms of the Mediterranean diet in obesity and type 2 diabetes. *J Nutr Biochem*. 2007;18:149–160.
26. Panagiotakos DB, Pitsavos C, Arvaniti F, et al. Adherence to the mediterranean food pattern predicts the prevalence of hypertension, hypercholesterolemia, diabetes and obesity, among healthy adults; the accuracy of the MedDietScore. *Prev Med*. 2007;44:335–340.
27. Panagiotakos DB, Pitsavos C, Stefanadis C. Dietary patterns: a Mediterranean diet score and its relation to clinical and biological markers of cardiovascular disease risk. *Nutr Metab Cardiovasc Dis*. 2006;16:559–568.
28. Panagiotakos DB, Miliatis GA, Pitsavos V, et al. MedDietScore: a computer program that evaluates the adherence to the Mediterranean dietary pattern and its relation to cardiovascular disease risk. *Comput Methods Programs Biomed*. 2006;83:73–77.
29. Bamia C, Lagiou P, Buckland G, et al. Mediterranean diet and colorectal cancer risk: results from a European cohort. *Eur J Epidemiol*. 2013;28:317–328.
30. Shah ND, Limketkai B. The use of medium-chain triglycerides in gastrointestinal disorders. *Nutri Issues Gastroenterol*. 2017;160:20–28.
31. Tenner S, Baillie J, DeWitt J, et al. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol* 2013;108:1400–15, 1416.
32. Forsmark CE. Management of chronic pancreatitis. *Gastroenterology*, 2013;144:1282–91 e3.
33. Adams KM, Lindell KC, Kohlmeier M, et al. Status of nutrition education in medical schools. *Am J Clin Nutr*. 2006;83:941S–944S.
34. Conwell DL, Lee LS, Yadav D, et al. American pancreatic association practice guidelines in chronic pancreatitis: evidence-based report on diagnostic guidelines. *Pancreas*. 2014;43:1143–1162.
35. Hager ER, Quigg AM, Black MM, et al. Development and validity of a 2-item screen to identify families at risk for food insecurity. *Pediatrics*. 2010;126:e26–e32.
36. Olesen SS, Holst M, K hler M, et al. Can we rely on predicted basal metabolic rate in chronic pancreatitis outpatients? *Clin Nutr ESPEN*. 2015;10:e66–e70.
37. Hebuterne X, Hastier P, P roux JL, et al. Resting energy expenditure in patients with alcoholic chronic pancreatitis. *Dig Dis Sci*. 1996;41:533–539. <https://doi.org/10.1177/0148607189013002124>

38. Coleman-Jensen A, R.M., Gregory C, Singh A. *Household Food Security in the United States in 2015*. 2016; <https://www.ers.usda.gov/webdocs/publications/err215/err-215.pdf?v=42636>.
39. Lu Y, García Rodríguez LA LA, Malgerud L, et al. New-onset type 2 diabetes, elevated HbA1c, anti-diabetic medications, and risk of pancreatic cancer. *Br J Cancer*. 2015;113:1607–1614.

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