ANKARA MEDICAL JOURNAL

Research Article

Ankara Med J, 2025;(3):368-379 // 10.5505/amj.2025.78476

EXOCRINE PANCREATIC FUNCTION IN ERECTILE DYSFUNCTION: A PROSPECTIVE STUDY BASED ON FECAL ELASTASE MEASUREMENT

Atilla Satır[1], Hakan Demirci[2]

[1]Department of Urology, University of Health Sciences, Bursa Yuksek Ihtisas Training and Research Hospital, Bursa, Türkiye

[2]Department of Family Medicine, University of Health Sciences, Bursa Yuksek Ihtisas Training and Research Hospital, Bursa, Türkiye

Correspondence:

Sorumlu Yazar (e-mail: atillasatir@gmail.com)

Submitted: 25.08.2025 // Accepted: 17.09.2025

Ankara Yıldırım Beyazıt University Faculty of Medicine

Department of Family Medicine

Abstract

Objectives: Exocrine pancreatic function, assessed via fecal pancreatic elastase (FPE), could contribute to the pathophysiology of erectile dysfunction (ED); however, supporting evidence is limited. The present study aimed to prospectively evaluate FPE levels in men and investigate the association between exocrine pancreatic insufficiency (EPI) and erectile function.

Methods: This prospective observational study was carried out between June and August 2025 at the urology outpatient clinic of the hospital. Male patients aged between 40 and 65 years presenting with any complaint were enrolled. Erectile function was assessed using the International Index of Erectile Function-5 (IIEF-5). Stool samples were analyzed for FPE by ELISA. Demographic, clinical, and laboratory data were collected. Group comparisons were performed using non-parametric tests, and predictors of erectile function were assessed with multivariate regression.

Results: A total of 153 patients were included. Median FPE levels demonstrated nominal variation across erectile function groups ($p=0.048$, Kruskal–Wallis), yet no statistically significant post-hoc differences after Bonferroni correction. Multivariate regression analysis revealed that diabetes mellitus ($\beta=-0.234$, $p=0.016$) and hypertension ($\beta=-0.167$, $p=0.040$) were independent negative predictors of erectile function, whereas FPE ($\beta=0.184$, $p=0.017$) was a positive predictor. Other variables, including age, BMI, testosterone, and prolactin, did not significantly contribute.

Conclusions: To our knowledge, this is one of the first studies to explore the link between exocrine pancreatic function and ED. Findings suggest that preserved pancreatic exocrine function may be beneficial for erectile health, while diabetes and hypertension remain major negative determinants. Exocrine pancreatic function deserves further investigation as a potential diagnostic and therapeutic target in the multifactorial management of ED.

Keywords: Erectile dysfunction, pancreatic elastase, exocrine pancreatic insufficiency, diabetes mellitus, hypertension

Introduction

Erectile dysfunction (ED) is one of the most prevalent male sexual health disorders, with its frequency increasing significantly with advancing age.[1] Community-based studies conducted in Turkey have reported that nearly half of men over 40 years of age experience some degree of ED, with prevalence rising steadily in older age groups.[2] Beyond its impact on sexual health, ED substantially affects psychological well-being, interpersonal relationships, and overall quality of life. The etiology of ED is multifactorial, with contributions from vascular, hormonal, neurogenic, and psychogenic factors. Systemic diseases such as diabetes mellitus (DM), hypertension, obesity, and cardiovascular disease are consistently associated with higher ED risk.[3,4]

While the traditional pathophysiological framework of ED has centered on vascular and endocrine mechanisms, recent studies have broadened this perspective to include the gastrointestinal system. Emerging evidence highlights the role of the gut–pancreas axis and the gut microbiome in modulating metabolic, hormonal, and endothelial pathways that may affect erectile function.[5,6] In this context, the exocrine pancreas has gained attention due to its critical role in digestion, nutrient absorption, and metabolic regulation.

Fecal pancreatic elastase (FPE) is a widely used non-invasive biomarker for assessing exocrine pancreatic function. Reduced FPE levels indicate exocrine pancreatic insufficiency (EPI), which is associated with malabsorption, malnutrition, and metabolic alterations.[7] Given that nutritional status and metabolic disturbances are key determinants of erectile function, investigating FPE in men with ED may provide new insights into its complex pathophysiology.

Several studies in Turkey have underscored the interplay between metabolic health and ED. Yaman et al. (2006) reported that men with metabolic syndrome had significantly higher rates of ED compared to healthy controls.[8] Similarly, Demir et al. (2008) showed that Turkish men with type 2 diabetes exhibited higher prevalence and severity of ED, reflecting the strong association between glycemic dysregulation and sexual dysfunction.[9] Notably, to date, no study has examined whether exocrine pancreatic function, as measured by fecal elastase, contributes to ED risk or severity in this population. Although only a limited number of studies have directly addressed this issue, prior reports on exocrine insufficiency, nutritional status, and gut microbiota strongly support the plausibility of this link.[10,11]

This study prospectively evaluates FPE levels in men with sexual dysfunction to investigate the potential link between EPI and ED. Understanding this relationship may enhance the multifactorial framework of ED and inform novel diagnostic and therapeutic strategies.

Materials and Methods

Study Design and Setting

This prospective observational study was carried out between June and August 2025at the Urology Outpatient Clinic of the Hospital. The study protocol was reviewed and approved by the institutional ethics committee (protocol number: 2024-TBEK 2025/06-16, date: June 4, 2025), and written informed consent was obtained from all participants before enrollment. The study was carried out in accordance with the principles of the Declaration of Helsinki.

Participants

Men aged between 40 and 65 years presenting with any complaint were considered for inclusion. Inclusion criteria: Patients with self-reported sexual function and willingness to participate. Exclusion criteria: Prior diagnosis of chronic pancreatitis, pancreatic surgery, malignancy, inflammatory bowel disease, severe hepatic or renal impairment, or current use of pancreatic enzyme replacement therapy.

Assessment of Erectile Dysfunction

Erectile function was evaluated using the International Index of Erectile Function-5 (IIEF-5) questionnaire, which has been validated in Turkish populations.[12] Participants completed the IIEF-5 either via a face-to-face interview or by self-administration under supervision.

Measurement of Fecal Pancreatic Elastase

Fresh stool samples were collected from all participants in sterile containers during admission and labeled with unique study codes. Specimens were immediately stored in a freezer at −20 °C until analysis. After thawing, a 15 mg portion was taken from each stool using a standardized stool preparation system. The pancreatic elastase levels were measured using an enzyme-linked immunosorbent assay (ELISA) with the IDK Pancreatic Elastase ELISA Kit (Immundiagnostik AG, Bensheim, Germany). Results were expressed in μg/g stool and calculated from optical density values against a standard curve. According to established thresholds, fecal elastase <200 μg/g was considered indicative of exocrine pancreatic insufficiency (EPI), with values <100 μg/g indicating severe EPI.[13] Diarrheal or urine-diluted stool specimens were excluded because they may yield falsely low results. Participants were instructed to maintain their usual diet and to avoid initiating any new medications, probiotics, or supplements during the week prior to stool collection. Although no specific dietary restrictions were applied, participants were advised to refrain from alcohol intake for 48 hours before sampling. All samples were collected under these standardized conditions to minimize variability.

Additional Data Collection

A structured questionnaire was used to obtain demographic and clinical data, including age, body mass index (BMI), smoking status, comorbidities (diabetes, hypertension, cardiovascular disease), medication history, and relevant laboratory findings (fasting glucose, hormone levels where available).

Statistical Analyses

Data obtained in the study were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA) and the Python programming language. The following Python libraries were used: pandas (data organization and cleaning), numpy (numerical computations), matplotlib and seaborn (graphical visualizations), scipy (basic statistical tests), and statsmodels (regression models and advanced statistical analyses).

For continuous variables, the assumption of normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. When normality was not met or the sample size was <30, continuous data were presented as median (interquartile range: Q1–Q3), and group comparisons were performed using the Kruskal–Wallis H test. For variables with significant differences, pairwise comparisons with Bonferroni correction were applied.

Categorical variables were expressed as counts and percentages (%). Group differences were evaluated using the Pearson Chi-square test or Fisher's exact Chi-square test when expected cell frequencies were low. Distribution of FPE values across erectile function groups was assessed with the Kruskal–Wallis test, followed by Bonferroni-adjusted pair-wise comparisons.

The group variable was treated as ordinal, and the linear relationship with FPE was examined using simple linear regression analysis and trend plots. To evaluate the impact of multiple independent variables on EF prediction, multiple linear regression analysis was carried out. The model included age, BMI, hypertension, DM, Benign Prostate Hypertrophy (BPH), Prostate Specific Antigen (PSA), smoking status, total testosterone, prolactin, fasting blood glucose (FBG), and FPE. Hypertension, DM, and BPH were coded as binary variables (0 = absent, 1 = present). Model significance was assessed using the F-test, while the significance of coefficients was examined with t-tests. The possibility of multicollinearity was evaluated using tolerance and VIF values. A p-value <0.05 was considered the threshold for statistical significance in all tests.

Results

In the comparison of demographic and clinical characteristics across erectile function groups, diabetes mellitus showed a strong association with ED severity, being most prevalent in men with severe dysfunction (62.5%) and least common in those with normal function (6.9%, $p < 0.001$) (Table 1).

Table1. Comparison of demographic and clinical characteristics by erectile function

Variable	Group 1	Group 2	Group 3	Group 4	Group 5	p-value
Age, median (Q1–Q3)	59.0 (51.5–61.5)	56.5 (52.0–60.0)	55.5 (50.5–60.0)	53.0 (51.0–57.0)	52.0 (50.0–58.0)	0.287¶
Hypertension, n (%)	11 (34.4)	8 (26.7)	6 (18.8)	3 (10.3)	3 (10.3)	0.083†
Diabetes Mellitus, n (%)	20 (62.5)	8 (26.7)	9 (28.1)	8 (27.6)	2(6.9)	<0.001†
Alcohol, n (%)	1 (3.1)	2 (6.7)	1 (3.1)	0 (0.0)	0 (0.0)	0.670‡
BMI, median (Q1–Q3)	29.0 (26.2–30.6)	28.5 (26.9–31.4)	29.2 (27.5–31.4)	27.6 (25.3–30.3)	28.4 (26.7–30.1)	0.472¶
BPH, n(%)	23 (71.9)	19 (63.3)	20 (62.5)	19 (65.5)	18 (62.1)	0.924†
Surgical History, n (%)	9 (28.1)	8(26.7)	4(12.5)	6 (20.7)	6(20.7)	0.585†
Smoking, n (%)	9 (28.1)	9(30.0)	6(18.8)	7 (24.1)	8(27.6)	0.863†
PSA, median (Q1-Q3)	1.23(0.54-2.47)	0.89(0.70-1.55)	0.99(0.49-3.68)	0.88(0.69-2.32)	1.03(0.70-1.61)	0.908¶
Total Testosterone, median (Q1-Q3)	432.50(318.50-524.50)	382.50 (285.00-495.00)	406.00 (312.00-506.50)	425.00(328.00-495.00)	417.00(379.00-450.00)	0.715¶
Prolactin, median (Q1-Q3)	9.93(7.64-12.20)	11.00(8.17-14.00)	11.30(9.00-14.12)	12.00(10.40-15.30)	11.00(9.84-12.20)	0.123¶
Fasting Blood Glucose, median (Q1-Q3)	125.00 (96.00-182.00)	100.50 (96.00-127.00)	102.00 (96.00-119.00)	100.00 (93.00-115.00)	98.00 (92.00-109.00)	0.044¶[a]

Values are presented as median (interquartile range: Q1–Q3) or number (percentage), as appropriate. † Pearson Chi-square test; ‡ Fisher's exact test; ¶ Kruskal–Wallis H test.[a] Pair-wise comparisons were performed after the Kruskal–Wallis H test using Bonferroni-adjusted significance values (1 > 2, 3, 4, 5).

Fasting blood glucose levels were also significantly higher in men with severe ED compared to those with better function (p = 0.044). Although hypertension tended to be more frequent in lower erectile function groups, this difference did not reach statistical significance (p = 0.083). Other variables, including age, BMI, smoking, alcohol

use, benign prostatic hypertrophy, surgical history, prostate-specific antigen, testosterone, and prolactin, did not differ significantly between groups.

The median FPE values were 194.5 (139.5–463.0) in Group 1, 216.0 (116.0–401.0) in Group 2, 317.5 (147.0–503.0) in Group 3, 424.0 (233.0–624.0) in Group 4, and 385.0 (287.0–466.0) in Group 5 (Figure 1). According to the Kruskal–Wallis H test, FPE values differed significantly across the five groups ($p = 0.048$). Unadjusted pairwise tests indicated nominal differences between Group 2 and Group 5 ($Z = -2.02$, $p = 0.044$), Group 2 and Group 4 ($Z = -2.49$, $p = 0.013$), and Group 1 and Group 4 ($Z = -2.31$, $p = 0.021$). Notably, none of these differences reached statistical significance following Bonferroni correction (all adjusted $p > 0.05$). Descriptive results suggested that mean ranks tended to increase from Group 1 (66.03) to Group 4 (92.09), with Group 5 (86.69) also showing relatively higher scores; nevertheless, these trends did not translate into statistically significant differences.

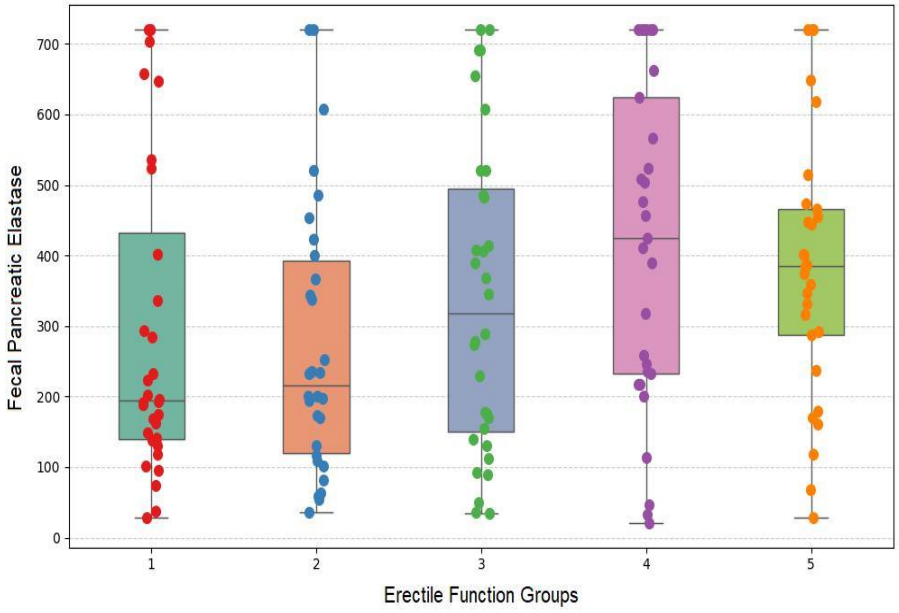
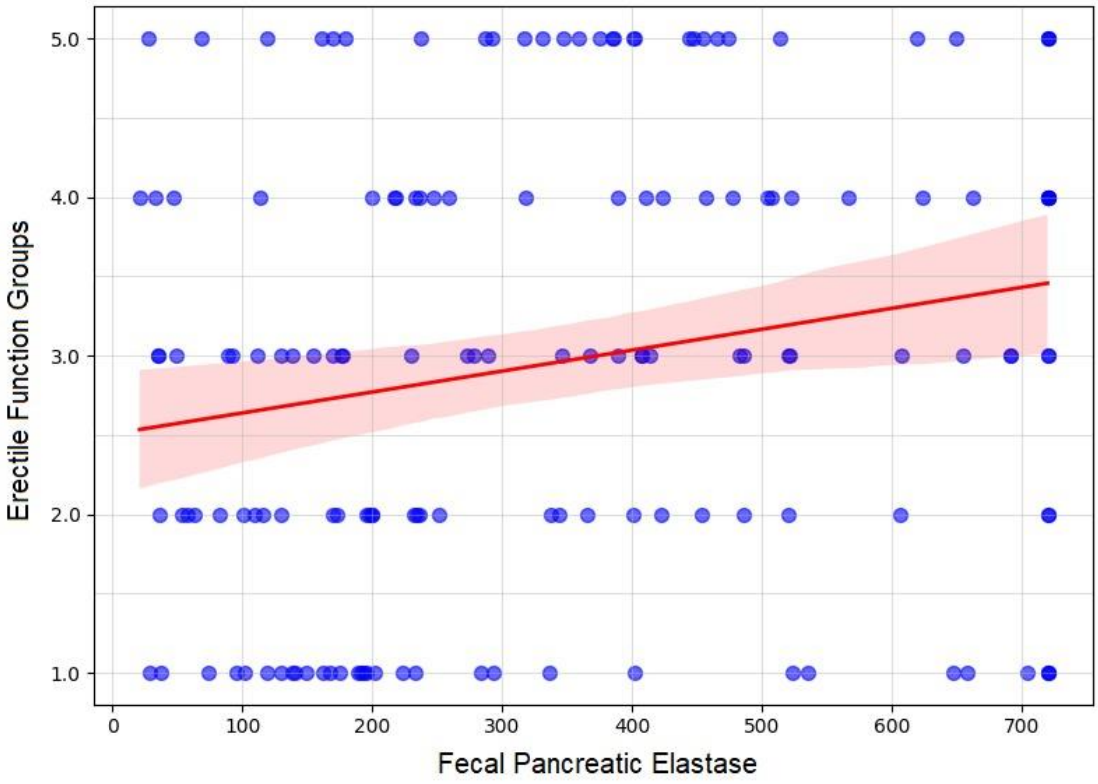


Figure 1. Comparison of fecal pancreatic elastase values between erectile function groups

Simple linear regression analysis demonstrated a modest but statistically significant positive association between fecal pancreatic elastase levels and erectile function groups ($y = 2.51 + 0.0013x$, $p = 0.0127$, $R^2 = 0.040$), indicating that higher elastase values were associated with better erectile function (Figure 2).



($y = 2.51 + 0.0013x$, $p = 0.0127$, $R^2 = 0.040$)

Figure 2. Regression analysis showing the effect of fecal pancreatic elastase values on erectile function groups 1–5

The results of the multiple linear regression analysis demonstrated that the model significantly predicted EF ($R^2 = 0.21$, $F = 3.43$, $p < 0.001$). Among the predictors, hypertension (B = –0.586, SE = 0.282, β = –.167, t = –2.08, p = .040, 95% CI [–1.144, –0.028]) and DM (B = –0.714, SE = 0.294, β = –.234, t = –2.43, p = .016, 95% CI [–1.295, –0.134]) were identified as significant negative predictors of EF. In contrast, FPE was a significant positive predictor of EF (B = 0.001, SE = 0.001, β = 0.184, t = 2.41, p = 0.017, 95% CI [0.000, 0.002]) (Table 2).

Table2. Predictors of erectile function

Variable	B	SE	Beta	t	p	95% CI
Age	-0.024	0.022	-0.110	-1.116	0.266	-0.067 to 0.019
BMI	0.014	0.032	0.035	0.428	0.669	-0.049 to 0.077
Hypertension	-0.586	0.282	-0.167	-2.077	0.040	-1.144 to -0.028
Diabetes Mellitus	-0.714	0.294	-0.234	-2.434	0.016	-1.295 to -0.134
Benign Prostate	0.369	0.297	0.125	1.242	0.216	-0.218 to 0.956
PSA	-0.189	0.251	-0.059	-0.752	0.453	-0.686 to 0.308
Smoking	-0.017	0.014	-0.096	-1.223	0.223	-0.043 to 0.010
Total Testosterone	0.000	0.001	-0.023	-0.295	0.768	-0.002 to 0.001
Prolactin	0.002	0.001	0.020	0.984	0.984	-0.004 to 0.004
Fasting Blood Glucose	-0.005	0.003	-0.136	-1.459	0.147	-0.011 to 0.002
Fecal Pancreatic	0.001	0.001	0.184	2.409	0.017	0.000 to 0.002

Multiple Linear Regression Analysis: $R^2 = 0.21$, $F = 3.43$, $p < 0.001$. CI = Confidence Interval. Hypertension, Diabetes Mellitus, and BPH were coded as binary variables (0 = absent, 1 = present).VIF values (1.04–1.70) indicated no evidence of multicollinearity in the model.

Discussion

This prospective study is, to our knowledge, one of the first to evaluate exocrine pancreatic function via FPE in men with ED. Our findings showed that although fecal elastase levels varied across erectile function groups, post-hoc comparisons revealed no statistically significant differences. Notably, regression analyses identified DM and hypertension as independent negative predictors of erectile function, whereas fecal elastase emerged as a positive predictor. These results highlight the complex interplay between metabolic comorbidities, pancreatic exocrine function, and male sexual health. In addition to diabetes and hypertension, other potential confounding factors such as hyperlipidemia, neurological and psychiatric conditions, medication use (e.g., antidepressants, antihypertensives), exercise habits, and nutritional status are also known to affect erectile function. These factors were not comprehensively assessed in the present study and may partly account for the observed variability in erectile function outcomes.

Although the role of the pancreas in sexual function has traditionally been overlooked, growing evidence suggests that digestive enzymes and gut–pancreas interactions may influence metabolic and vascular pathways relevant to ED.[14-16] Fecal elastase is a sensitive biomarker of EPI, with low levels reflecting impaired digestion

and nutrient absorption.7 Malnutrition and metabolic disturbances, in turn, can exacerbate vascular and hormonal mechanisms underlying ED. Our results support the hypothesis that preserved exocrine pancreatic function could contribute to better erectile function, even after adjusting for major comorbidities.

Consistent with prior studies, DM and hypertension were found to be strong independent risk factors for ED in this cohort. A meta-analysis by Corona et al. reported that diabetic men had nearly a threefold higher risk of ED compared to healthy controls.[17] Similarly, Turkish studies have shown increased ED prevalence among men with metabolic syndrome and type 2 DM.[18,19] Vascular endothelial dysfunction, neuropathy, oxidative stress, and chronic low-grade inflammation are the proposed mechanisms linking these metabolic disorders to ED. Our findings reaffirm these associations while also suggesting that pancreatic insufficiency may represent an additional metabolic contributor.

To date, very few studies have examined the direct relationship between fecal elastase and sexual dysfunction. Our findings, albeit with modest effect sizes, indicate a potential role of pancreatic exocrine function in the multifactorial etiology of ED. This aligns with emerging evidence that the gut microbiota and digestive enzymes may affect hormonal balance and endothelial health.[20-22] Incorporating fecal elastase assessment in selected ED patients, particularly those with diabetes or unexplained symptoms, may provide a non-invasive adjunct tool for risk stratification. Furthermore, studies focusing on pancreatic exocrine insufficiency and nutritional status in chronic disease10 and diabetes-related sexual dysfunction11 also support this hypothesis.

The clinical implications of this study are twofold. First, routine evaluation of pancreatic exocrine function could be considered in men with ED who also present with metabolic disorders, malnutrition, or gastrointestinal symptoms. Second, identifying subclinical EPI in such patients may open avenues for targeted interventions, including dietary modifications, enzyme replacement therapy, and microbiota-oriented strategies. Further multicenter studies with larger sample sizes are warranted to validate these findings and to explore whether treating EPI can improve sexual function outcomes. Notably, the lack of access to FPE testing in primary healthcare settings limits its integration into routine clinical practice. Broader access at the family medicine level could facilitate early diagnosis and management, thereby enhancing the translation of such research into clinical applications.

This study has several limitations. The single-center design and limited sample size reduce generalizability. The cross-sectional nature precludes causal inference. Fecal elastase, while a reliable marker, may be influenced by stool consistency and collection variability. Moreover, detailed dietary assessments, gastrointestinal symptom profiles, and advanced imaging markers of pancreatic function were not addressed. Future prospective longitudinal studies should include these factors and consider interventional designs.

Although the Turkish-validated International Index of Erectile Function-5 (IIEF-5) was used to assess ED, recommending FPE as a diagnostic test for clinical application based on a baseline questionnaire exceeds the scope of the present study. Therefore, it would be reasonable for future research to evaluate the predictive value of FPE in ED using objective measures such as Penile Color Doppler Ultrasonography and Night Penile Tumescence and Rigidity Assessment, in addition to validated ED scales. Furthermore, the mode of questionnaire administration (face-to-face interview vs. self-administration) may have influenced participants' responses and scale scores, introducing an additional source of variability.

Overall, this prospective study demonstrated that DM and hypertension were independent negative predictors of erectile function, while FPE emerged as a positive predictor, suggesting that exocrine pancreatic function could contribute to the multifactorial etiology of erectile dysfunction and deserves further investigation as a potential diagnostic and therapeutic target.

Conflict of Interest: The authors declare that they have no commercial or financial relationships that could be construed as conflicts of interest. No funding was received for the preparation of this study, and there are no competing interests among the authors.

Ethics Approval: This study was reviewed and approved by the Ethics Committee of SBU Bursa Yüksek İhtisas University (protocol number: 2024-TBEK 2025/06-16, date: June 4, 2025). All procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki.

References

1. Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. J Urol. 1994;151(1):54-61.
2. Akkus E, Kadioglu A, Esen A, Doran S, Ergen A, Anafarta K, et al. Prevalence and correlates of erectile dysfunction in Turkey: a population-based study. Eur Urol. 2002;41(3):298-304.
3. Bacon CG, Hu FB, Giovannucci E, Glasser DB, Mittleman MA, Rimm EB. Association of type and duration of diabetes with erectile dysfunction in a large cohort of men. Diabetes Care. 2002;25(8):1458-63.
4. Doumas M, Tsakiris A, Douma S, Gravas S, Papadopoulos A, Hatzichristou D. Factors affecting the increased prevalence of erectile dysfunction in Greek hypertensive compared with normotensive subjects. J Androl. 2006;27(3):469-77.
5. Khoo J, Piantadosi C, Duncan R, Worthley S, Jenkins A, Noakes M, et al. Comparing effects of a low-energy diet and a high-protein low-fat diet on sexual and endothelial function, urinary tract symptoms, and inflammation in obese diabetic men. J Sex Med. 2011;8(10):2868-75.
6. Moya A, Ferrer M, Zabala JL, Blas-García A, Esplugues JV. The role of gut microbiota in the development and progression of erectile dysfunction. Int J Mol Sci. 2020;21(19):7069.
7. Loser C, Mollgaard A, Folsch UR. Faecalelastase 1: a novel, highly sensitive, and specific tubeless pancreatic function test. Gut. 1996;39(4):580-6.
8. Yaman O, Soygur T, Zumrutbas AE, Ciftci H, Anafarta K. Erectile dysfunction in men with metabolic syndrome. Int J Impot Res. 2006;18(4):368-74.
9. Demir O, Akgul K, Akar Z, Ciftci H, Yazici C, Kanbay A, et al. Association between severity of erectile dysfunction and left ventricular diastolic dysfunction in men with type 2 diabetes. Urology. 2008;72(1):97-101.
10. Madeddu C, Gramignano G, Floris C, Massa E, Astara G, Cherchi MC, et al. Pancreatic exocrine insufficiency and nutritional status in advanced cancer patients. Support Care Cancer. 2011;19(9):1521–1525.
11. Maiorino MI, Bellastella G, Esposito K. Diabetes and sexual dysfunction: current perspectives. Diabetes MetabSyndrObes. 2014;7:95–105.
12. Rosen RC, Cappelleri JC, Smith MD, Lipsky J, Peña BM. Development and evaluation of an abridged, 5-item version of the International Index of Erectile Function (IIEF-5) as a diagnostic tool for erectile dysfunction. Int J Impot Res. 1999;11(6):319-26.
13. Lankisch PG, Schmidt I, König H, Lehnick D, Knollmann R, Löhr M, et al. Faecalelastase 1: not helpful in diagnosing chronic pancreatitis associated with mild to moderate exocrine pancreatic insufficiency. Gut. 1998;42(4):551-4.

14. Keller J, Layer P. Human pancreatic exocrine response to nutrients in health and disease. Gut. 2005;54Suppl 6:vi1-28.
15. Domínguez-Muñoz JE. Pancreatic enzyme therapy for pancreatic exocrine insufficiency. CurrGastroenterol Rep. 2007;9(2):116-22.
16. Witt H, Apte MV, Keim V, Wilson JS. Chronic pancreatitis: challenges and advances in pathogenesis, genetics, diagnosis, and therapy. Gastroenterology. 2007;132(4):1557-73.
17. Corona G, Mannucci E, Schulman C, Petrone L, Mansani R, Cilotti A, et al. Psychobiological correlates of hypoactive sexual desire in patients with erectile dysfunction. Int J Impot Res. 2004;16(4):275-81.
18. Esposito K, Giugliano D. Obesity, the metabolic syndrome, and sexual dysfunction. Int J Impot Res. 2005;17(5):391-8.
19. Bal K, Özkara A, Sönmez Y, Can E, Yılmaz H, Bozkurt A, et al. Prevalence of metabolic syndrome and its association with erectile dysfunction among urology patients. Int J Impot Res. 2007;19(3):358-63.
20. Khoo J, Tian HH, Tan B, Chew K, Ng CS, Leong D, et al. Erectile dysfunction and hypogonadism in patients with obstructive sleep apnea: efficacy of continuous positive airway pressure. Int J Impot Res. 2010;22(1):38-43.
21. Turnbaugh PJ, Ley RE, Mahowald MA, Magrini V, Mardis ER, Gordon JI. An obesity-associated gut microbiome with increased capacity for energy harvest. Nature. 2006;444(7122):1027-31.
22. Dinan TG, Cryan JF. The impact of gut microbiota on brain and behavior: implications for psychiatry. CurrOpinClinNutrMetab Care. 2015;18(6):552-8.