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Fecal Incontinence as a Marker of Multisystem Cardiopulmonary-Kidney Disease and Mortality in US Adults:

A Nationally Representative Cohort Study

Chukwuemeka Ogbu, MD; Ifeanyi Momodu, MD, MPH, FACP; Chinazor Umerah, MD, MBA, FACP; & Prashant Raj Bhatt, M.D.

Department of Internal Medicine, Cape Fear Valley Health, Fayetteville, NC

Abstract

Objective. To determine whether fecal incontinence is associated with cardiovascular, pulmonary, and kidney disease, individually and in combination, and with all-cause mortality among community-dwelling US adults. **Methods.** Nationally representative cohort study using the National Health and Nutrition Examination Survey (NHANES) 2005–2010 with linkage to the National Death Index through December 31, 2019. The analytic cohort comprised 14,731 adults aged 20 years or older, with 14,718 eligible for mortality analysis. Fecal incontinence, defined as accidental leakage of mucus, liquid stool, or solid stool during the prior 30 days. A composite cardiopulmonary–kidney (CPK) burden was calculated as the count of three affected systems: cardiovascular disease (self-report), pulmonary disease (current asthma, emphysema, or chronic bronchitis), and kidney disease markers (estimated glomerular filtration rate <60 mL/min/1.73 m², urine albumin–creatinine ratio ≥30 mg/g, or self-reported kidney disease). Multisystem CPK burden was defined as ≥2 affected systems. Survey-weighted prevalence ratios (PRs) for each cardiopulmonary–kidney outcome from modified Poisson regression and hazard ratios (HRs) for all-cause mortality from Cox proportional hazards models, with sequential adjustment for sociodemographic, cardiometabolic, and shared functional/mood/urinary factors. **Results.** Among 14,731 adults (weighted mean age 46.8 years; 51.2% women), the weighted prevalence of fecal incontinence was 8.4% (95% CI, 7.8%–9.0%). Adults with fecal incontinence were nearly a decade older than those without (mean age 55.6 vs 46.0 years) and had higher prevalences of urinary incontinence (62.0% vs 32.6%), depressive symptoms (17.4% vs 6.2%), and functional limitation (30.3% vs 13.8%) (all P<.001). After full adjustment, fecal incontinence remained associated with kidney disease markers (PR, 1.16; 95% CI, 1.02–1.32) and with simultaneous involvement of all three CPK systems (PR, 2.38; 95% CI, 1.47–3.86). During follow-up, 2,395 deaths occurred. Crude mortality was 80.8 per 1,000 person-years among adults with both fecal incontinence and multisystem CPK burden, versus 10.6 in adults with neither. After adjustment, multisystem CPK burden alone (HR, 1.80; 95% CI, 1.58–2.04) and combined fecal incontinence plus burden (HR, 2.14; 95% CI, 1.66–2.77) predicted mortality. Fecal incontinence alone did not (HR, 1.06; 95% CI, 0.87–1.28). With multisystem CPK burden as the reference, the combined group did not demonstrate a statistically significant mortality increment (HR, 1.19; 95% CI, 0.92–1.54; P=.18). **Conclusions and Relevance.** Fecal incontinence in US adults is associated with disproportionate cardiopulmonary–kidney disease, functional impairment, and mortality, but the association with mortality was related to multisystem disease rather than to bowel symptoms themselves. Disclosure of fecal incontinence is a low-cost clinical signal that warrants integrated systemic assessment, including routine kidney function testing, rather than purely anorectal evaluation.

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Introduction

Fecal incontinence which is defined as the involuntary loss of stool, affects roughly 8% of community-dwelling US adults, rises sharply with age, and is consistently underreported by patients and underdiagnosed by clinicians.[1-3] The condition is among the most common reasons older adults are admitted to long-term care, and it tracks with reduced quality of life, social isolation, and depressive symptoms.[4] Continence depends on the coordinated function of stool consistency, rectal sensation, sphincter integrity, pelvic floor coordination, central and peripheral neural control, cognition, and the ability to reach a toilet in time.[5] In older adults a defect in any of these systems can produce leakage, and defects rarely occur in isolation. Clinical guidelines therefore recommend evaluation that addresses bowel habit, neurologic function, mobility, mental health, and coexisting illness rather than focusing solely on the anorectum.[6,7]

Despite this conceptual frame, fecal incontinence is often handled in practice as an isolated bowel-control problem. Patients seldom volunteer the symptom, and clinicians seldom ask.[8,9] Whether routine inquiry could identify adults whose bowel symptom signals broader systemic disease and so guide cardiovascular, pulmonary, and renal evaluation, has not been established. Multimorbidity is now the dominant pattern of chronic illness in US adults,[10,11] and cardiovascular–kidney–metabolic frameworks recognize that disease in one organ system commonly indicates disease in others.[12,13] Whether fecal incontinence belongs within this multisystem picture, and whether it carries independent prognostic information beyond the comorbidities it accompanies, has not been characterized in a nationally representative US adult sample.

These questions carry particular weight in Delaware. Delaware is among the oldest states in the nation: adults 65 or older now outnumber children, and chronic disease accounts for at least 61% of deaths statewide, with cardiovascular disease and cancer alone responsible for 39%.[12, 13] The state ranks in the bottom fifth of all states for chronic kidney disease and carries a stroke mortality rate well above the national figure (46.5 vs 37.6 per 100,000), burdens that fall disproportionately on non-Hispanic Black Delawareans, who die of stroke and diabetes at nearly twice the rate of their White neighbors.[13] An older population layered on a heavy, unevenly distributed cardiopulmonary–kidney burden is precisely the setting in which a low-cost marker of multisystem disease would be most useful.

We used data from NHANES 2005–2010 with linked mortality follow-up to address two questions relevant to an aging US population. First, is fecal incontinence associated with cardiovascular, pulmonary, and kidney disease, individually and in combination? Second, does fecal incontinence carry independent mortality risk, or is a change in mortality associated with co-existing systemic disease? The aim was practical which is to determine whether bowel-symptom inquiry, a low-cost, scalable clinical question has value as a public health sentinel of broader vulnerability in adult US populations.

Methods

Study Design and Population

We conducted a cohort analysis of NHANES 2005–2010, a continuous, multistage probability survey of the noninstitutionalized US civilian population.[14] NHANES combines household

interviews, standardized physical examinations, and laboratory testing through mobile examination centers (MECs). The 2005–2010 cycles were chosen because the Bowel Health Questionnaire, which contains the leakage items used to define fecal incontinence, was administered to adults aged 20 years and older during these years.[15] Adults with positive MEC weights and complete data on fecal incontinence and on the cardiovascular, pulmonary, and kidney domains were eligible. The mortality cohort additionally required eligibility for National Death Index linkage and nonmissing follow-up time. NHANES protocols are approved by the NCHS Research Ethics Review Board. This secondary analysis of de-identified public-use data was deemed exempt by the Cape Fear Valley ACGME Institutional Review Board.

Exposure

The primary exposure was fecal incontinence, defined as accidental leakage of mucus, liquid stool, or solid stool at any frequency during the prior 30 days. Gas-only leakage was excluded because it is common, nonspecific, and not the focus of clinical intervention. Secondary definitions tested liquid or solid stool leakage, weekly leakage, and any leakage including gas.

Cardiopulmonary–Kidney (CPK) Burden

Three system domains were defined a priori. The cardiovascular domain was positive for self-reported diagnosis of congestive heart failure, coronary heart disease, angina, myocardial infarction, or stroke. The pulmonary domain was positive for current asthma, emphysema, or current chronic bronchitis. The kidney domain was positive for estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m², urine albumin–creatinine ratio (UACR) ≥30 mg/g, or self-reported weak or failing kidneys. eGFR was calculated using the 2021 CKD-EPI creatinine equation without race,[16] and the albuminuria threshold followed contemporary KDIGO guidance.[17] The CPK score was the count of affected domains (0–3). Multisystem CPK burden was defined as ≥2 affected domains. For mortality analysis, a four-level joint exposure was constructed: neither fecal incontinence nor multisystem CPK burden (reference), fecal incontinence only, multisystem CPK burden only, and both.

Covariates

Sociodemographic variables included age, sex, race and ethnicity, educational attainment, family income-to-poverty ratio, marital status, and NHANES cycle. Behavioral and access variables included smoking status, alcohol use, physical activity, insurance, usual source of care, recent health care visits, and overnight hospitalization. Clinical covariates included body mass index, waist circumference, obesity, diabetes (self-report, glucose-lowering medication, or hemoglobin A1c ≥6.5%), hypertension (self-report or antihypertensive medication), urinary incontinence, depressive symptoms (Patient Health Questionnaire-9 ≥10),[18] and functional limitation.

Outcomes

Cross-sectional outcomes were each cardiopulmonary–kidney domain, any CPK domain, multisystem CPK burden, and tri-domain burden. The longitudinal outcome was all-cause mortality, ascertained through the 2019 public-use linked mortality file with follow-up beginning at the MEC examination date. Cause-specific deaths were summarized descriptively and all-cause mortality served as the primary survival end point because of stable event counts.

Statistical Analysis

All analyses used NHANES sampling weights, masked variance strata, and primary sampling units. MEC weights were divided by 3 across the pooled cycles per NHANES guidance.[14] Categorical variables were summarized as unweighted counts with weighted percentages and continuous variables as weighted means with standard errors (or weighted medians with interquartile ranges for skewed distributions). Differences by fecal incontinence status were tested with Rao–Scott chi-square and survey-weighted regression.

Survey-weighted modified Poisson regression with a log link was used to estimate prevalence ratios (PRs) for each cardiopulmonary–kidney outcome.[19] Three sequential models were fit. Model 1 adjusted for age, sex, and race and ethnicity. Model 2 added educational attainment, family income-to-poverty ratio, smoking, body mass index, diabetes, hypertension, insurance, physical activity, and NHANES cycle. Model 3 added urinary incontinence, depressive symptoms, and functional limitation.

Mortality was analyzed with survey-weighted Cox proportional hazards models. The primary contrast used adults without fecal incontinence and with low CPK burden (0–1 affected domains) as the reference. A secondary contrast re-leveled the reference to multisystem CPK burden alone, isolating any incremental risk attributable to coexisting fecal incontinence. Two-sided $P<.05$ defined statistical significance. Analyses used R, version 4.3, with the survey and survival packages.[20,21]

Results

Study Population

Of 31,034 NHANES participants, 17,132 were aged ≥20 years and 16,539 had positive MEC weights. The analytic cohort included 14,731 adults, out of which 14,718 were eligible for mortality analysis. Weighted mean age was 46.8 years (SE, 0.3); 51.2% were women, and 71.0% non-Hispanic White. The weighted prevalence of fecal incontinence was 8.4% (95% CI, 7.8%–9.0%). Stool-only fecal incontinence was 6.9% (95% CI, 6.4%–7.5%), and weekly fecal incontinence was 2.5% (95% CI, 2.3%–2.8%) (Table 1).

Table 1. Weighted Prevalence of Fecal Incontinence Definitions, Cardiopulmonary–Kidney Burden, and Selected Comorbidities

Variable	Weighted %	95% CI
Fecal incontinence (primary definition)	8.38	7.75–9.01
Stool-only fecal incontinence	6.93	6.36–7.50
Weekly fecal incontinence	2.53	2.27–2.80
Any leakage including gas	47.43	45.87–48.98
Cardiovascular disease domain	8.21	7.44–8.98
Pulmonary disease domain	10.22	9.26–11.18
Kidney disease marker domain	13.60	12.73–14.47
Any CPK domain	25.51	24.33–26.68
≥2 CPK domains	5.92	5.27–6.58

All 3 CPK domains	0.60	0.44–0.75
Urinary incontinence	35.05	34.01–36.09
Depressive symptoms (PHQ-9 ≥10)	7.10	6.39–7.80
Functional limitation	15.03	13.89–16.18
Diabetes	10.56	9.83–11.28
Hypertension	30.49	29.12–31.85

CPK indicates cardiopulmonary–kidney; PHQ-9, Patient Health Questionnaire-9; CI, confidence interval.

Adults with fecal incontinence were nearly a decade older than those without (weighted mean age 55.6 vs 46.0 years; P<.001) and more often female (57.8% vs 50.6%; P<.001) (Table 2). They had higher prevalences of obesity (41.8% vs 34.3%), diabetes (18.3% vs 9.9%), hypertension (45.7% vs 29.2%), urinary incontinence (62.0% vs 32.6%), depressive symptoms (17.4% vs 6.2%), and functional limitation (30.3% vs 13.8%) (all P<.001). Loose stool form (Bristol types 6–7) was approximately threefold more common among adults with fecal incontinence (17.3% vs 5.7%; P<.001), whereas constipation-range stool was similarly distributed (6.8% vs 7.0%; P=.79).

Table 2. Baseline Characteristics of US Adults by Fecal Incontinence Status, NHANES 2005–2010

Characteristic	Total (n=14,731)	No FI (n=13,379)	FI (n=1,352)	P value
Sociodemographic				
Age, y, mean (SE)	46.8 (0.3)	46.0 (0.3)	55.6 (0.6)	<.001
Female, n (%)	7,499 (51.2)	6,736 (50.6)	763 (57.8)	<.001
Race/ethnicity, n (%)				.002
Mexican American	2,718 (8.2)	2,522 (8.4)	196 (6.1)	
Other Hispanic	1,243 (4.4)	1,148 (4.5)	95 (2.9)	
Non-Hispanic White	7,254 (71.0)	6,489 (70.5)	765 (75.9)	
Non-Hispanic Black	2,925 (11.0)	2,681 (11.0)	244 (10.0)	
Other/multiracial	591 (5.5)	539 (5.5)	52 (5.1)	
Less than high school education, n (%)	4,164 (18.4)	3,734 (18.0)	430 (21.8)	.031
Family income-to-poverty ratio <1.30, n (%)	4,040 (19.2)	3,646 (19.0)	394 (22.1)	.006
Widowed/divorced/ separated, n (%)	3,314 (18.5)	2,870 (17.7)	444 (27.7)	<.001
Bowel Characteristics				
Loose stool form (Bristol 6–7), n (%)	1,137 (6.6)	884 (5.7)	253 (17.3)	<.001
Constipation stool form (Bristol 1–2), n (%)	1,126 (7.0)	1,031 (7.0)	95 (6.8)	.79

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Behavioral and Access				
Current smoker, n (%)	3,251 (22.3)	2,947 (22.3)	304 (22.6)	.005†
Any physical activity, n (%)	10,402 (76.0)	9,563 (76.7)	839 (67.7)	<.001
Health insurance coverage, n (%)	11,212 (80.6)	10,113 (80.3)	1,099 (84.1)	.004
Overnight hospitalization, prior year, n (%)	1,876 (10.4)	1,584 (9.7)	292 (18.0)	<.001
Health care visits, prior year (categories)				<.001
0	2,442 (16.1)	2,297 (16.6)	145 (11.0)	
1	2,557 (18.4)	2,399 (18.9)	158 (13.0)	
2–3	3,838 (27.5)	3,510 (27.7)	328 (25.4)	
4–9	3,712 (24.5)	3,308 (24.0)	404 (29.9)	
10–12	1,033 (6.1)	880 (5.8)	153 (10.0)	
≥13	1,138 (7.4)	974 (7.0)	164 (10.8)	
Clinical and Functional				
Body mass index, kg/m², mean (SE)	28.7 (0.1)	28.6 (0.1)	29.8 (0.3)	<.001
Obesity, n (%)	5,394 (34.9)	4,831 (34.3)	563 (41.8)	<.001
Diabetes, n (%)	2,195 (10.6)	1,868 (9.9)	327 (18.3)	<.001
Hypertension, n (%)	5,096 (30.6)	4,421 (29.2)	675 (45.7)	<.001
Urinary incontinence, n (%)	5,338 (35.1)	4,484 (32.6)	854 (62.0)	<.001
PHQ-9 ≥10, n (%)	1,265 (7.1)	988 (6.2)	277 (17.4)	<.001
Functional limitation, n (%)	2,775 (15.2)	2,286 (13.8)	489 (30.3)	<.001
Cardiopulmonary–Kidney Domains				
Cardiovascular disease domain, n (%)	1,612 (8.3)	1,313 (7.5)	299 (17.1)	<.001
Pulmonary disease domain, n (%)	1,552 (10.2)	1,329 (9.7)	223 (15.3)	<.001
Kidney disease marker domain, n (%)	2,681 (13.7)	2,271 (12.7)	410 (25.2)	<.001
eGFR <60 mL/min/1.73 m², n (%)	1,152 (5.8)	963 (5.2)	189 (11.4)	<.001
UACR ≥30 mg/g, n (%)	1,755 (9.0)	1,507 (8.4)	248 (15.3)	<.001
Multisystem CPK burden (≥2 domains), n (%)	1,186 (6.0)	943 (5.2)	243 (14.5)	<.001
Tri-domain CPK burden, n (%)	135 (0.6)	93 (0.4)	42 (2.7)	<.001

Categorical variables: unweighted counts (weighted percentages). Continuous variables:

weighted means (SE). P values from Rao–Scott design-adjusted chi-square (categorical) and survey-weighted regression (continuous). †P value reflects overall difference across smoking status categories. Fecal incontinence (FI) = accidental leakage of mucus, liquid stool, or solid stool at any frequency in the prior 30 days. Cardiovascular disease domain: self-reported congestive heart failure, coronary heart disease, angina, myocardial infarction, or stroke. Pulmonary disease domain: current asthma, emphysema, or current chronic bronchitis. Kidney disease marker domain: estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m², urine albumin–creatinine ratio (UACR) ≥30 mg/g, or self-reported weak or failing kidneys. CPK indicates cardiopulmonary–kidney; PHQ-9, Patient Health Questionnaire-9.

Cardiopulmonary–Kidney Burden

Each cardiopulmonary–kidney domain was more common among adults with fecal incontinence: cardiovascular disease, 17.1% vs 7.5%; pulmonary disease, 15.3% vs 9.8%; kidney disease markers, 25.2% vs 12.7%. Multisystem (≥2 domains) burden was 14.5% vs 5.2%, and tri-domain burden was 2.7% vs 0.4% (Table 3).

Table 3. Weighted Prevalence of Cardiopulmonary–Kidney Burden by Fecal Incontinence Status, NHANES 2005–2010

FI status	CV, %	Pulmonary, %	Kidney, %	Any CPK, %	≥2 CPK, %	All 3 CPK, %
No fecal incontinence	7.46	9.75	12.65	24.25	5.18	0.43
Fecal incontinence	17.12	15.33	25.19	40.42	14.54	2.67

Values are weighted percentages. Domains and abbreviations as defined in Table 2.

In age-, sex-, and race/ethnicity-adjusted models (Model 1), fecal incontinence was associated with each domain individually and with multisystem and tri-domain burden (Table 4). After adjustment for socioeconomic, behavioral, and cardiometabolic factors (Model 2), associations attenuated but remained statistically significant for all outcomes. After further adjustment for urinary incontinence, depressive symptoms, and functional limitation (Model 3), most domain-specific associations were no longer statistically significant. Two persisted: kidney disease markers (PR, 1.16; 95% CI, 1.02–1.32; P=.03) and tri-domain CPK burden (PR, 2.38; 95% CI, 1.47–3.86; P<.001).

Table 4. Survey-Weighted Prevalence Ratios for the Association Between Fecal Incontinence and Cardiopulmonary–Kidney Outcomes

Outcome	Model	PR (95% CI)	P value	Unweighted n
Cardiovascular Disease	Model 1	1.43 (1.23–1.65)	<.001	14,731
	Model 2	1.22 (1.07–1.40)	.004	13,440
	Model 3	1.07 (0.92–1.23)	.38	13,367
Pulmonary Disease	Model 1	1.40 (1.21–1.62)	<.001	14,731

	Model 2	1.27 (1.07–1.50)	.007	13,440
	Model 3	1.10 (0.92–1.31)	.30	13,367
Kidney Disease Markers	Model 1	1.37 (1.22–1.54)	<.001	14,731
	Model 2	1.20 (1.07–1.35)	.003	13,440
	Model 3	1.16 (1.02–1.32)	.03	13,367
Any CPK Domain	Model 1	1.25 (1.16–1.36)	<.001	14,731
	Model 2	1.13 (1.03–1.23)	.007	13,440
	Model 3	1.04 (0.96–1.14)	.33	13,367
≥2 CPK Domains	Model 1	1.73 (1.44–2.09)	<.001	14,731
	Model 2	1.40 (1.17–1.67)	<.001	13,440
	Model 3	1.20 (0.98–1.46)	.08	13,367
All 3 CPK Domains	Model 1	3.78 (2.29–6.22)	<.001	14,731
	Model 2	2.97 (1.82–4.85)	<.001	13,440
	Model 3	2.38 (1.47–3.86)	<.001	13,367

Survey-weighted modified Poisson regression with log link. Reference category for each outcome is absence of that outcome. Model 1 adjusted for age, sex, and race/ethnicity. Model 2 added educational attainment, family income-to-poverty ratio category, smoking status, body mass index, diabetes, hypertension, insurance status, physical activity, and NHANES cycle. Model 3 added urinary incontinence, depressive symptoms, and functional limitation. CPK indicates cardiopulmonary–kidney; CI, confidence interval; PR, prevalence ratio.

Mortality

Over follow-up, 2,395 all-cause deaths occurred. Crude mortality was 10.6 per 1,000 person-years among adults without fecal incontinence and with low CPK burden, 20.3 among those with fecal incontinence only, 66.8 among those with multisystem CPK burden only, and 80.8 among those with both (Table 5). Both groups containing multisystem CPK burden remained associated with all-cause mortality after full adjustment: multisystem CPK burden only (HR, 1.80; 95% CI, 1.58–2.04) and combined fecal incontinence plus burden (HR, 2.14; 95% CI, 1.66–2.77). Fecal incontinence alone was not (HR, 1.06; 95% CI, 0.87–1.28; P=.56) (Table 6).

Table 5. Crude All-Cause Mortality and Cause-Specific Deaths by Joint Fecal Incontinence and Cardiopulmonary–Kidney Burden Group

Joint FI/CPK group	Unweighted n	All-Cause Deaths		Death rate per 1,000 PY	CV Deaths	Resp. Deaths	Renal Deaths
No FI / low CPK burden	12,426	1,480	139,411	10.6	423	58	24
FI only	1,107	239	11,756	20.3	70	17	3
Multisystem CPK burden only	942	526	7,877	66.8	200	56	14

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FI + multisystem CPK burden	243	150	1,857	80.8	43	13	5

Crude (unweighted) rates per 1,000 person-years. Cause-specific deaths shown as unweighted counts. CV indicates cardiovascular; PY, person-years; Resp., respiratory.

Table 6. Survey-Weighted Cox Proportional Hazards Models for All-Cause Mortality by Joint Fecal Incontinence and Multisystem Cardiopulmonary–Kidney Burden

Model	Joint FI/CPK Group	HR (95% CI)	P value	Unweighted n
Model 1	FI only	1.30 (1.07–1.57)	.008	14,718
	Multisystem CPK burden only	2.60 (2.30–2.94)	<.001	14,718
	FI + multisystem CPK burden	3.23 (2.67–3.91)	<.001	14,718
Model 2	FI only	1.13 (0.93–1.36)	.21	13,432
	Multisystem CPK burden only	2.00 (1.75–2.28)	<.001	13,432
	FI + multisystem CPK burden	2.41 (1.92–3.01)	<.001	13,432
Model 3	FI only	1.06 (0.87–1.28)	.56	13,359
	Multisystem CPK burden only	1.80 (1.58–2.04)	<.001	13,359
	FI + multisystem CPK burden	2.14 (1.66–2.77)	<.001	13,359

Reference group: adults with no fecal incontinence and low cardiopulmonary–kidney burden (0–1 affected domains). Multisystem CPK burden = ≥2 affected domains. Adjustment sets parallel those used in Table 3. CPK indicates cardiopulmonary–kidney; FI, fecal incontinence; HR, hazard ratio; CI, confidence interval.

With multisystem CPK burden alone as the reference, the combined group did not demonstrate a statistically significant mortality increment (HR, 1.19; 95% CI, 0.92–1.54; P=.18) (Table 7).

Table 7. Direct Comparison of Joint Fecal Incontinence and Cardiopulmonary–Kidney Groups Using Multisystem CPK Burden Alone as the Reference (Fully Adjusted Model**)**

Comparison (Reference: Multisystem CPK Burden Only)	HR (95% CI)	P value	Unweighted n
No FI / low CPK burden	0.56 (0.49–0.63)	<.001	13,359
FI only	0.59 (0.49–0.71)	<.001	13,359
FI + multisystem CPK burden	1.19 (0.92–1.54)	.18	13,359

Survey-weighted Cox proportional hazards model with full adjustment as in Table 4, Model 3. CPK indicates cardiopulmonary–kidney; FI, fecal incontinence; HR, hazard ratio; CI, confidence interval.

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Discussion

In a nationally representative cohort of US adults, fecal incontinence is associated with significantly higher chronic disease burden, functional impairment, and mortality. Adults with fecal incontinence had nearly twice the prevalence of cardiovascular disease, more than twice the prevalence of multisystem cardiopulmonary–kidney burden, and roughly six times the prevalence of disease in all three organ systems simultaneously. These differences narrowed once urinary incontinence, depression, and functional limitation were accounted for, indicating that much of the cross-sectional clustering travels along shared pelvic-floor, mood, and mobility pathways rather than through a discrete anorectal mechanism. Two associations persisted after full adjustment: kidney disease markers and the simultaneous involvement of all three organ systems. These point to settings in which fecal incontinence carries information beyond the overlap with other functional impairments.

The mortality findings clarify a long-standing question: is fecal incontinence itself a determinant of survival, or a marker of the disease burden that is? In our analysis, the mortality signal followed multisystem disease, not bowel control. Crude mortality among adults with both fecal incontinence and multisystem CPK burden was nearly eightfold that of the reference group, but after adjustment the difference between multisystem disease alone and multisystem disease with fecal incontinence was not statistically significant. This pattern is consistent with prior cohort study in older adults, in which the apparent prognostic effect of fecal incontinence largely reflected coexisting cognitive, functional, and self-rated health vulnerabilities.[22,23] The implication is not that fecal incontinence is unimportant — the symptom marks a high-risk group — but that the appropriate clinical response is to evaluate the systemic disease the symptom signals, not to treat fecal incontinence as a freestanding mortality risk factor.

The persistence of the kidney-marker association after full adjustment deserves separate consideration. Adults with fecal incontinence remained 16% more likely to have reduced eGFR, albuminuria, or self-reported kidney disease, after accounting for diabetes, hypertension, urinary incontinence, depression, and functional limitation. Several mechanisms can explain this. Diabetic and uremic autonomic neuropathy can impair both renal autoregulation and anorectal sensorimotor control.[24] Vascular injury, chronic low-grade inflammation, and skeletal muscle dysfunction which are all features of cardiorenal disease extend to pelvic and visceral function.[25,26] Polypharmacy in cardiorenal management, including agents that alter stool consistency or rectal sensation, may also contribute. Whether the kidney–fecal incontinence link reflects a discrete pathophysiologic mechanism, or a sensitive readout of multisystem frailty cannot be determined here, but the association is sufficiently consistent to warrant attention to kidney function in adults presenting with bowel-control symptoms.

A second clinically actionable finding concerns stool form. Loose stool was three times more common among adults with fecal incontinence, while constipation-range stool was equally distributed. This asymmetry mirrors population-based work identifying altered bowel habit, rather than sphincter integrity alone, as the dominant determinant of late-onset fecal incontinence.[27,28] Liquid stool overwhelms even an intact continence apparatus, particularly when rectal sensation is blunted by aging or autonomic neuropathy. Evaluation focused on stool form, transit, medication contributors, and dietary triggers therefore offers a more tractable lever for symptom control than evaluation centered on sphincter anatomy alone.

These findings sit within a broader public health response to aging. Fecal incontinence is consistently underreported by patients and underdiagnosed by clinicians,[8,9] making the moment of disclosure a high-yield clinical opportunity that bowel-focused management alone fails to exploit. Single-disease guidelines align poorly with multimorbid patients, in whom competing priorities, polypharmacy, and treatment burden complicate care.[29,30] Care models for multimorbidity emphasize patient-priority alignment, functional preservation, and longitudinal team-based management,[31,32] and fecal incontinence fits comfortably within these frameworks as both target and indicator.

Our findings also map directly onto Delaware's disease landscape. The state's aging, hypertensive, diabetic, and disproportionately kidney-affected population is the same profile that defined the highest-risk stratum in our analysis, and the racial gradient in Delaware's stroke and diabetes mortality mirrors the multisystem burden that fecal incontinence flagged nationally.[33] In a setting where clinician time is scarce and competing chronic-disease priorities crowd the primary care visit, a single question about bowel control, which may be embedded in routine chronic-disease management and in aging-services referrals, offers Delaware a cheap, scalable way to surface systemic illness that would otherwise stay hidden. The opportunity is greatest precisely where the disease burden is heaviest and least evenly shared.

Strengths of the analysis include the use of a large, nationally representative sample with standardized assessment, the inclusion of objective kidney measures alongside self-report, the sequential modeling strategy that distinguished demographic and cardiometabolic adjustment from functional and mood adjustment, and the direct mortality contrast that isolated incremental risk of fecal incontinence beyond multisystem disease. Limitations include the self-reported nature of fecal incontinence and several disease components, which would most plausibly bias estimates toward the null, the cross-sectional design of the comorbidity analysis, which precludes inference about temporal order. The absence of anorectal manometry, defecography, validated severity instruments, and detailed medication data also limits mechanistic resolution. Residual confounding from unmeasured factors including formal frailty measures, cognitive impairment, and post-baseline institutionalization is possible. Cause-specific mortality analyses were limited by sparse event counts. Quantitative bias analyses such as the E-value[33] could refine the interpretation of these associations in future longitudinal studies.

Conclusions

Among US adults, fecal incontinence identified a population with substantially elevated cardiopulmonary–kidney disease burden, functional impairment, and mortality. The mortality signal traced to coexisting multisystem disease rather than to bowel symptoms themselves, and after full adjustment fecal incontinence remained independently associated with kidney disease markers and with simultaneous tri-domain disease. These findings argue for treating disclosed fecal incontinence as a clinical signal that warrants integrated assessment of kidney function, cardiovascular and pulmonary review, mood, mobility, and urinary continence rather than as an isolated bowel-control complaint. Routine bowel-symptom inquiry is a low-cost, scalable addition to clinical practice that may help identify high-risk adults whose systemic disease would otherwise remain undisclosed.

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Dr. Ogbu may be contacted at ogbuemmanuelchukwuemeka@gmail.com

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