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Gut microbiota and kidney function in autosomal dominant polycystic kidney disease participants in Cameroon: a cross-sectional study

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Abstract

Background and hypothesis Gut dysbiosis characterized by an imbalance in pathobionts (*Enterobacter*, *Escherichia* and *Salmonella*) and symbionts (*Bifidobacterium*, *Lactobacillus* and *Prevotella*) can occur during chronic kidney disease (CKD) progression. We evaluated the associations between representative symbionts (*Bifidobacterium* and *Lactobacillus)* and pathobionts (Enterobacteriaceae) with kidney function in persons with autosomal dominant polycystic kidney disease (ADPKD).

Methods In this cross-sectional study, 29 ADPKD patients were matched to 15 controls at a 2:1 ratio. Clinical data and biological samples were collected. The estimated glomerular filtration rate (eGFR) was calculated from the serum creatinine concentration using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. Microbial DNA extracted from stool specimens and amplified by qPCR was used to quantify Enterobacteriaceae, *Bifidobacterium* and *Lactobacillus* abundance. Differences between ADPKD subgroups and controls were assessed using nonparametric tests.

Results The mean age (SD) of the 44 participants was 40.65 (± 11.9) years. Among the participants with ADPKD, 62.1% experienced flank pain, and 48.3% had hypertension. Their median eGFR [IQR] was 74.4 [51.2–94.6] ml/min/1.73m^2. All stool samples had Enterobacteriaceae. *Lactobacillus* abundance was lower in ADPKD participants with more pronounced kidney function decline (CKD G3-5: 0.58 ng/µL) than in those with milder damage and controls (G1-2: 0.64 ng/µL, $p=0.047$; controls: 0.71 ng/µL, $p=0.043$), while Enterobacteriaceae abundance was greater in ADPKD patients with lower kidney function (CKD G3-5: 78.6 ng/µL) than in those in the other two groups (G1-2: 71.6 ng/µL, $p=0.048$; controls: 70.5 ng/µL, $p=0.045$).

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Conclusion Decreased kidney function was associated with decreased symbiont and increased pathobiont abundance in ADPKD patients, suggesting a potential role for the microbiota in disease progression and possible targets for further research.

Keywords ADPKD, CKD, Dysbiosis, Gut microbiota

Background

The gut microbiota, which is the collection of all microbes cohabitating and living symbiotically within the gastrointestinal tract, plays several physiological roles [1]. It protects against pathogenic microbes, participates in energy metabolism, regulates the immune system and produces short-chain fatty acids (SCFAs), which exert renoprotective effects [1–4]. The composition and function of the microbiota are modulated by several factors, such as age, diet, drugs and disease [5–8].

Chronic kidney disease (CKD) is a major public health problem affecting approximately 15% of the adult population [9, 10], with autosomal dominant polycystic kidney disease (ADPKD) being the first genetic cause [11]. CKD is associated with biochemical and biophysical changes, notably, the retention of uremic toxins, an alteration in intestinal pH and an increase in colonic transit time [3]. Furthermore, CKD involves dietary restrictions and specific therapeutic interventions. Hence, these factors may lead to gut microbiota alterations with the growth of proteolytic pathobionts at the detriment of saccharolytic symbionts as well as metabolic, immune and endocrine disorders [3, 4, 12–15]. The uremic milieu in CKD leads to the proliferation of proteolytic bacteria, which produce uremic toxins such as p-cresyl sulfate and indoxyl sulfate; the latter accumulate in the plasma with declining kidney function, leading to enhanced oxidative stress, inflammation and fibrosis, thus exacerbating CKD [16]. Hence, it has been suggested that low-protein diets of pro-, pre- and synbiotics (oligofructose-enriched inulin, *B. infantis*, and *L. acidophilus*), which promote symbiont growth, could reduce inflammation in CKD patients and slow its progression [13, 14, 16, 17].

Individuals with CKD due to conditions such as diabetic kidney disease or glomerulonephritis often present with comorbidities and may require medications such as antimicrobial or anti-inflammatory therapies, which have the potential to alter the gut microbiota [3, 18]. This premise prompted the US study on microbiota changes in a polycystic kidney disease population, which demonstrated stepwise changes associated with kidney function decline [18].

To the best of our knowledge, little is known about CKD-induced microbiota changes in sub-Saharan Africa (SSA). In this region, where the prevalence of CKD is high and access to kidney replacement therapy is limited, nephroprotection is a crucial aspect of CKD management [20]. Moreover, differences in the dietary intake of populations from SSA could influence changes in the gut microbiota compared to that in other regions [21].

Therefore, we conducted a cross-sectional study to evaluate the gut microbiota pattern with respect to kidney function in a Cameroonian ADPKD population. To achieve this goal, we analysed the faecal microbiota of ADPKD patients and healthy controls by quantitative polymerase chain reaction (qPCR) and compared bacterial abundance considering kidney function.

Methods

Design and setting

This cross-sectional study ran from November 1, 2022, to May 31, 2023, at Yaoundé University Teaching and General Hospitals. The study included ADPKD and control groups of participants matched for sex and age ± 5 years at a ratio of 2:1.

We included persons aged 18 to 65 years who agreed to participate in the study. ADPKD patients were diagnosed by nephrologists using ultrasound-based Ravine's criteria. The controls had no kidney cysts on abdominal ultrasound, normal kidney function, and negative urine dipstick results.

Subjects were excluded from the ADPKD group if they (1) had diabetes, chronic diarrhea, or chronic infections (HIV, hepatitis B or C); (2) had undergone hemodialysis; (3) were on antibiotics, prebiotics, probiotics, laxatives or NSAIDs in the last four weeks prior to enrolment, or (4) did not provide all samples. The participants were excluded from the control group if they met conditions (1), (3), and (4).

Dietary assessment was performed using a modified food frequency questionnaire (FFQ) and scoring method described elsewhere [19, 20], and 24-h diet recall with dietary intake evaluated from Cameroonian and Tanzanian food databases [21, 22]. For each individual, the calculation of the food consumption frequency score corresponding to two groups (Group I and Group II) was obtained. Group I was formed by foods considered to be protective for the gut microbiota and Group II was formed by foods considered non-protective for the gut microbiota.

Group I: fruits, vegetables, fermented foods, yoghurt, seeds, grains (pumpkin seed, sesame), popcorn, fish, palm oil, resistant starch (cocoyam, cassava). Group II: meat (beef, pork, poultry), egg, white rice, bread, margarine, mayonnaise, ripe plantains, sugar, alcoholic beverages.

Table 1 Sequences and specifications of the primers used in this study

Bacteria	Sequence (5′ – 3′)	Product size (bp)	Reference
Lactobacillus	F: GCAGCAGTAGGGAATCTTC R: AGTTACTACCTCTATCCTTCTTC	119	[23]
Bifidobacterium	F: CGCGTCCGGTGTGAAAG R: CCCCACATCCAGCATCCA	244	[23]
Enterobacteriaceae	F: GGGGATAAC(T/C)ACTGGAAACGGT(A/G)GC R: GCATGGCTGCATCAGG(C/G)TT(G/T)C		[24]

F = forward; R = reverse

Consumption frequency scores were calculated by simple summation, in which the sum of the consumption frequency weighting values for the foods corresponding to each group was calculated. Score I was represented by the sum of the weighting values for the foods that made up Group I and score II, by the sum of the weighting values for the foods that made up Group II [25].

We collected clinical (blood pressure, urine dipstick) data and biological (urine, blood, stool) samples during fasting in the morning between 8 and 10 am.

Assessment of parameters

Blood pressure (BP) was measured by means of an automated blood pressure measuring device with a cuff following the WHO specifications [26]. BP was measured in both arms, and the measurement was performed twice, with a two-minute interval. The average of the two readings was recorded. The highest BP value among the arms was considered.

Dipstick test was performed on the second morning midstream urine samples following standard recommendations for urinalysis strip tests [27]. Dipstick reactive areas were read at their respective time intervals, and the presence of proteins, leucocytes, blood and nitrites in urine was noted after a control one week later.

Samples of 3–5 mL of venous blood were collected in plain tubes. The samples were centrifuged at 5000 revolutions/minute for five minutes, and serum was collected and used to measure serum creatinine according to the kinetic Jaffé method [28]. Serum creatinine was standardized by calibration against the National Institute of Standards and Technology (NIST) standard [29] with the Kenza MAX BioChemisTry analyser (BIOLABO, France). The 2009 CKD Epidemiology Collaboration (CKD-EPI) equation was used to estimate the glomerular filtration rate (eGFR/in ml/min/1.73m^2) from the serum creatinine values.

Sampling and DNA extraction

Each participant provided a fresh stool sample in a sterile 40 mL container after receiving verbal instructions on how to collect the sample. The samples were transported to the National Veterinary Laboratory (LANAVET) Annex of Yaoundé in a cooler with ice packs and stored immediately in a -80 °C freezer until analysis.

Table 2 Real-time PCR protocol used in this study

Cycle step	Temperature	Time (in seconds)	Cycles
Initial denaturation	95 °C	60	1
Denaturation	95 °C	15	40–45
Extension	60 °C	30 (+ plate read)	
Melt curve	60–95 °C	Various	1

DNA extraction from stool was performed using the DNeasy Blood & Tissue Kit (Qiagen, Dusseldorf, Germany) according to the manufacturer's instructions. The purity and quantity of the extracted DNA were evaluated using a spectrophotometer (DeNovix, DE, USA). All DNA samples were stored at -80 °C until further analysis.

Quantitative real-time PCR (qPCR)

Primer sets for Enterobacteriaceae, *Bifidobacterium* and *Lactobacillus* from previous studies were used for real-time PCR. Their specificity was verified using the Basic Local Alignment Search Tool (BLAST) program. The primer sets used are listed in Table 1. The SYBR Green qPCR assays were performed on a CFX96 Touch Real-Time PCR Detection System (Bio-Rad, California, USA). The reaction mixture contained 10 µL of Luna universal qPCR (New England BioLabs) master mix (2 × Conc.), 0.5 µL of forward primer (10 µM), 0.5 µL of reverse primer (10 µM), 2 µL of DNA template and 7 µL of nuclease-free water. The PCR conditions are summarized in Table 2 [30]. Positive and negative controls were run alongside the DNA template. The positive controls included a single colony of *S. typhi* ATCC 14,028 for Enterobacteriaceae and yogurt-isolated *Lactobacillus* and *Bifidobacterium* spp. DNA for *Lactobacillus* and *Bifidobacterium*.

Melt curve analysis was performed to verify the specificity of the amplifications, with melting peaks at 86.50, 87.00 and 82.50 °C for Enterobacteriaceae, *Bifidobacterium* and *Lactobacillus*, respectively. For each target bacterium, the quantification cycle (Cq) of each DNA specimen was used to estimate the initial target DNA quantity in ng/µL using the positive controls' DNA yield (spectrophotometric determination) in ng/µL and the linear equation derived during the study (supplemental file).

Chronic kidney disease (CKD) was staged using eGFR [calculated by software using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) 2021 without

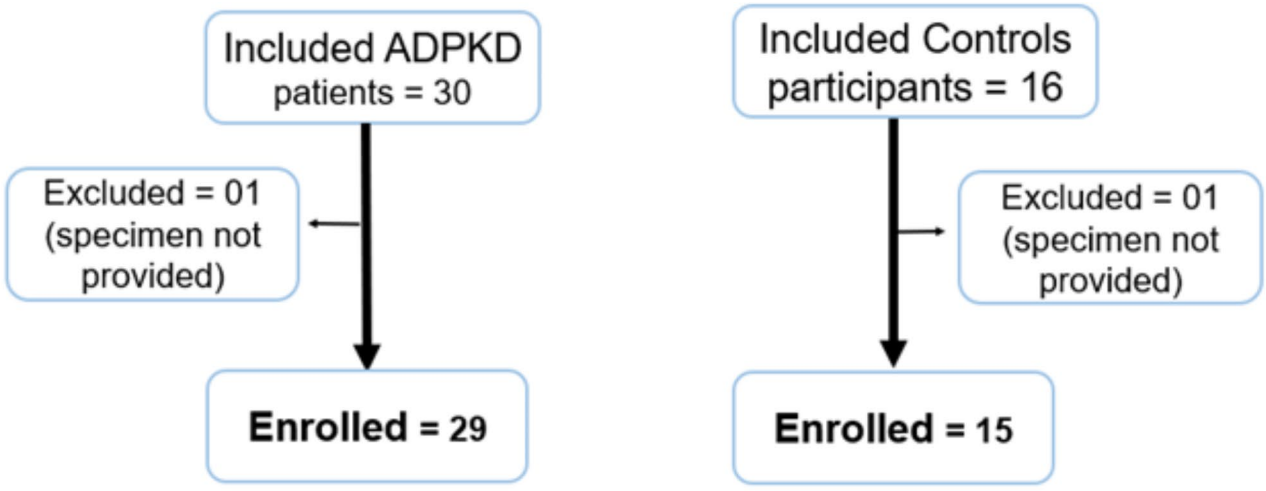

Fig. 1 Enrolment flow chart

Table 3 Demographic, clinical and paraclinical characteristics of the study participants ($n=44$)

Characteristic	ADPKD ($n=29$)	Control ($n=15$)	*p* value
Age †	40.2 ± 11.2	41.3 ± 13.4	0.795
Female sex, n (%)	20 (69%)	9 (60%)	0.738
BMI (kg/m^2) †	21.6 ± 3.4	23.3 ± 3.5	0.136
Blood pressure (mm/Hg)			
SBP †	128.9 ± 22.7	127.7 ± 16.2	0.709
DBP †	83.8 ± 13.7	78.9 ± 13.3	0.241
Serum creatinine(mg/L)*	11.6[8.7–16.3]	8.9[8.0-10.6]	**0.022**
eGFR(ml/min/1.73m^2)*	74.4[51.2–94.6]	94.5[77.3-111.7]	**0.022**

BMI = body mass index; DBP = diastolic blood pressure, eGFR = estimated glomerular filtration rate; SBP = systolic blood pressure, † mean ± standard deviation, * median [interquartile range]

racial facto] according to KDIGO 2012 (Kidney Disease Improving Global Outcomes) guidelines [31]. Stage G1 (eGFR ≥ 90 ml/min/1.73m^2) corresponds to normal or high, stage G2 (eGFR 60–89 ml/min/1.73m^2) corresponds to mildly decreased, stage G3 (eGFR 30–59 ml/min/1.73m^2) corresponds to mildly to severely decreased, stage G4 (eGFR 15–29 ml/min/1.73m^2) corresponds to severely decreased and stage G5 (eGFR < 15 ml/min/1.73m^2) corresponds to kidney failure. The presence of protein, blood and leucocytes in urine samples was noted if the colour change corresponded to at least 0.3 g/l, 10 RBC/µL and 70 WBC/µL, respectively.

Statistical analysis

Data analysis was performed using the software IBM-SPSS (Statistical Package for Social Sciences) version 26.0 (IBM Corp., Armonk, NY, USA). The results are expressed as proportions for categorical variables and means ± standard deviations or medians [interquartile ranges] for continuous variables. The chi-squared test was used for comparing categorical data. For continuous variables, the independent t test and Mann–Whitney test were used for normally distributed and skewed data, respectively. Statistical significance was inferred at a *p* value less than 0.05.

Results

Study participants

Of the 44 participants enrolled, 29 (65.9%) had autosomal dominant polycystic kidney disease (ADPKD), and 15 (34.1%) were controls (Fig. 1). The sociodemographic, clinical, dietary and biochemical characteristics of the 2 groups were similar apart from the gut microbiota-protective diet pattern (Score I), which was significantly lower in controls ($p=0.041$) (Tables 3 and 4). The main clinical manifestations of the ADPKD participants were flank pain and hypertension (Fig. 2). We collected general antihypertensive data of ADPKD participants who were on antihypertensive medication (41.4%) with most participants on angiotensin II converting enzyme inhibitors/angiotensin II receptors blockers, calcium channel blockers and beta-blockers. Close to two-thirds had a family history of ADPKD. The eGFR of ADPKD patients ranged from 12.5 to 122.0 ml/min/1.73m^2. There were 11 (38%) ADPKD participants at stage G3-5 of CKD with eGFR ranging from 12.5 to 59.7 ml/min/1.73m^2. Meanwhile, 18 (62%) ADPKD participants were CKD G1-2 with eGFR ranging from 61.2 to 122 ml/min/1.73m^2 (Fig. 3).

Bacterial quantification by qPCR

All specimens had Enterobacteriaceae, whereas *Lactobacillus* was present in 79% of ADPKD specimens and 73% of control specimens. *Bifidobacterium* was present in 93.1% of ADPKD specimens and in all healthy controls (Fig. 4). We observed no significant differences in bacterial abundance between the groups.

Table 4 Dietary characteristics of study population ($n=44$)

	ADPKD (n=29)	Control (n=15) $Q_2[Q_1\text{-}Q_3]$	*p* value
Nutrient intake (24 h recall)			
Energy (Kcal)	1544.1 [1014.5-1517.9]	1520.0 [1015.9-1822.8]	0.757
Proteins (g)	40.3 [24.9–56.5]	38.9 [32.8–50.6]	0.853
Carbohydrates(g)	114.6 [68.1-163.4]	114.1 [100.2-159.4]	0.421
Fibres(g)	19.4 [4.8–20.4]	16.9 [14.9–27.7]	0.052
Fats (g)	69.1 [36.2-127.1]	64.2 [44.8-131.9]	0.738
Dietary pattern (FFQ)			
Score I	5.2 [4.4-6.0]	4.1 [3.2–5.1]	**0.041***
Score II	2.8 [2.0-3.4]	3.7 [2.7–3.9]	0.134

Score 1: « Protective » of gut microbiota, Score II: « Non - protective » of gut microbiota; Q2[Q1-Q3] = median [interquartile range]

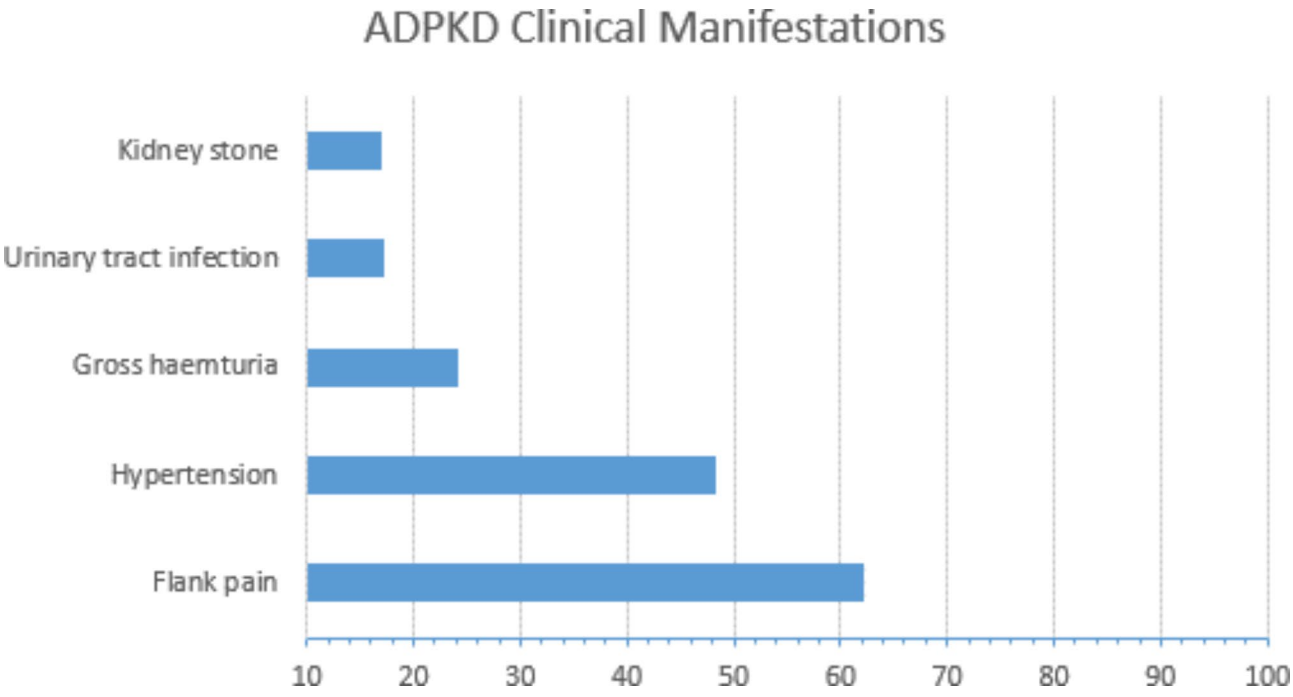

Fig. 2 ADPKD clinical manifestations

Associations between kidney function and the gut microbiota

Enterobacteriaceae were significantly more abundant in ADPKD participants with CKD G3-5 than in those with CKD G1-2 (median, 78.6 [72.7–80.0] vs. 71.6 [65.7–77.9] ng/µL; $p=0.048$). Similarly, they were also more abundant in ADPKD participants with CKD G3-5 than in healthy controls (median, 78.6 [72.7–80.0] vs. 70.5 [72.1–73.6] ng/µL; $p=0.045$). *Lactobacillus* was significantly less abundant in ADPKD participants with CKD G3-5 than in those with CKD G1-2 (median, 0.58 [0.00-0.62] vs. 0.64 [0.60–0.79] ng/µL; $p=0.047$). Additionally, *Lactobacillus* was less abundant in ADPKD participants with CKD G3-5 than in controls (median, 0.58 [0.00-0.62] vs. 0.71 [0.32–0.81] ng/µL; $p=0.043$) (Fig. 5& Fig. 6). There were no significant differences in the abundance of *Bifidobacterium* between the CKD G3-5, CKD G1-2, and healthy control groups. (Fig. 7).

Discussion

The uremic milieu in CKD promotes the growth of proteolytic bacteria that generate uremic toxins such as p-cresyl sulfate and indoxyl sulfate. The accumulation of the latter in the plasma, as kidney function declines, contributes to increased oxidative stress and inflammation, which exacerbate CKD [16]. The research presented in this article evaluated the gut microbiota pattern and kidney function of Cameroonian ADPKD participants. We investigated the associations between the abundances of Enterobacteriaceae, *Bifidobacterium* and *Lactobacillus* and kidney function in the ADPKD and control groups. As kidney function decreased, Enterobacteriaceae abundance increased, while *Lactobacillus abundance*

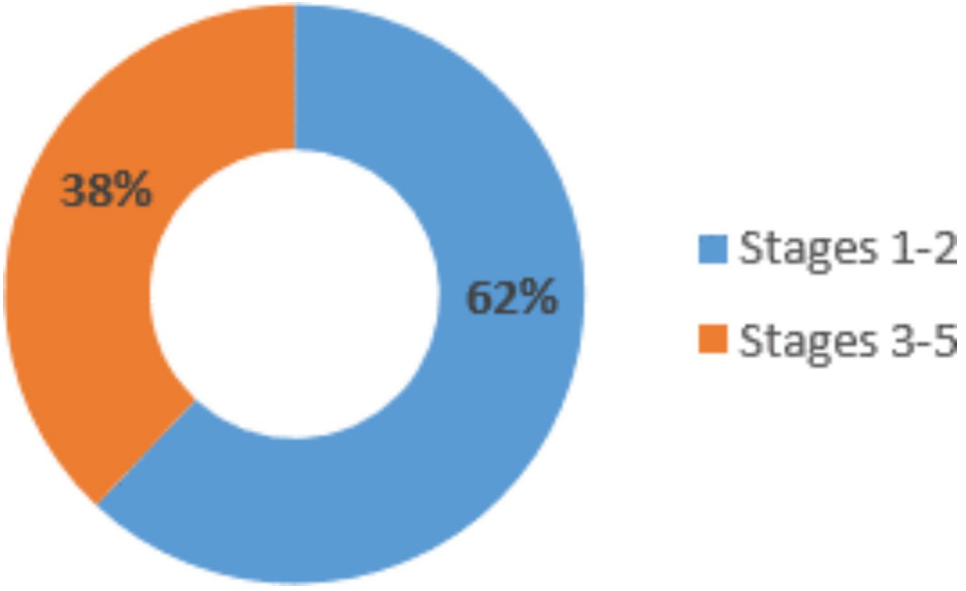

Fig. 3 CKD stage distribution in our study population

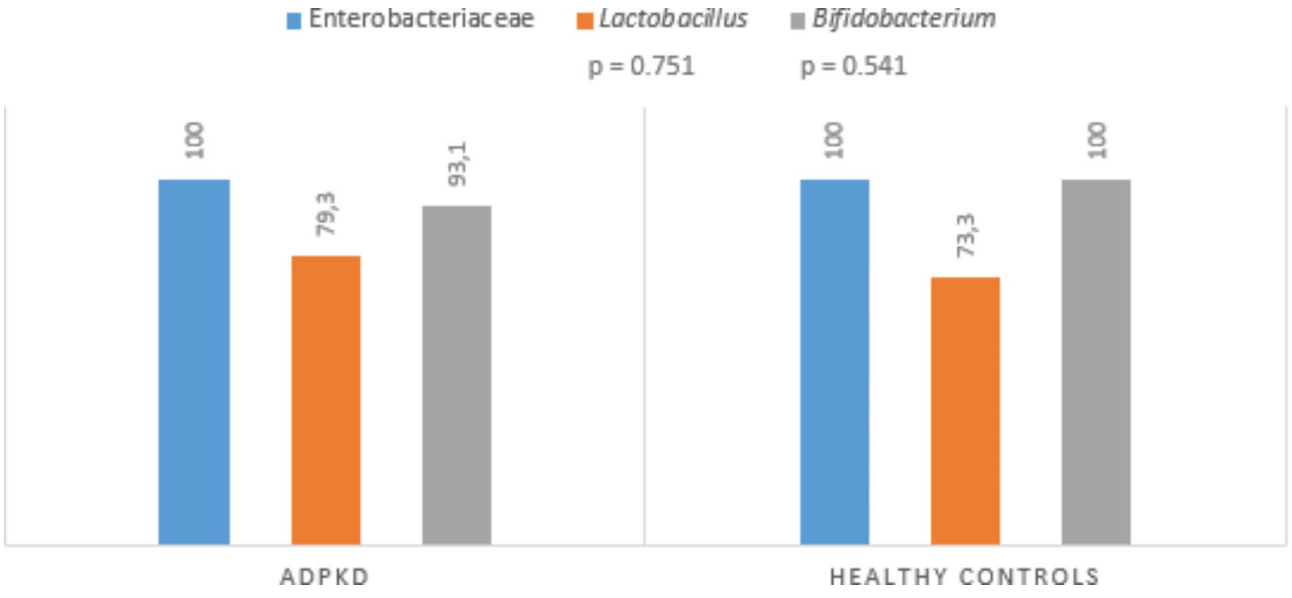

Fig. 4 Comparison of gut microbiome species in ADPKD patients and healthy controls

decreased in the study population. This finding aligns with the literature [3, 13, 15]. This study stands out as one of the few that employed locally available molecular methods to profile the microbiota and demonstrate the gut–kidney axis within a specific CKD subpopulation. This indicates that alterations in the gut microbiota occur with CKD progression and suggests that the microbiota could serve as a potential target for nephroprotective interventions.

The median *Lactobacillus* DNA abundance was lower in the ADPKD CKD G 3–5 group than in the control group ($p = 0.043$). This indicates that individuals with ADPKD who experienced a greater decrease in kidney function had a lower average abundance of *Lactobacillus* than did healthy individuals serving as controls. The accumulation of uremic toxins in CKD patients creates an unfavourable milieu for the growth of symbionts such as *Lactobacillus* while favouring the growth of proteolytic bacteria. Therefore, a decrease in the abundance of beneficial bacteria *such as Lactobacillus*, which produce short-chain fatty acids (SCFAs) through the fermentation of complex carbohydrates, may lead to reduced SCFAs, which have renoprotective anti-inflammatory, immunomodulatory and antifibrotic properties. These changes may lead to increased inflammation and impaired immune and gut barrier function [2]. Our finding is

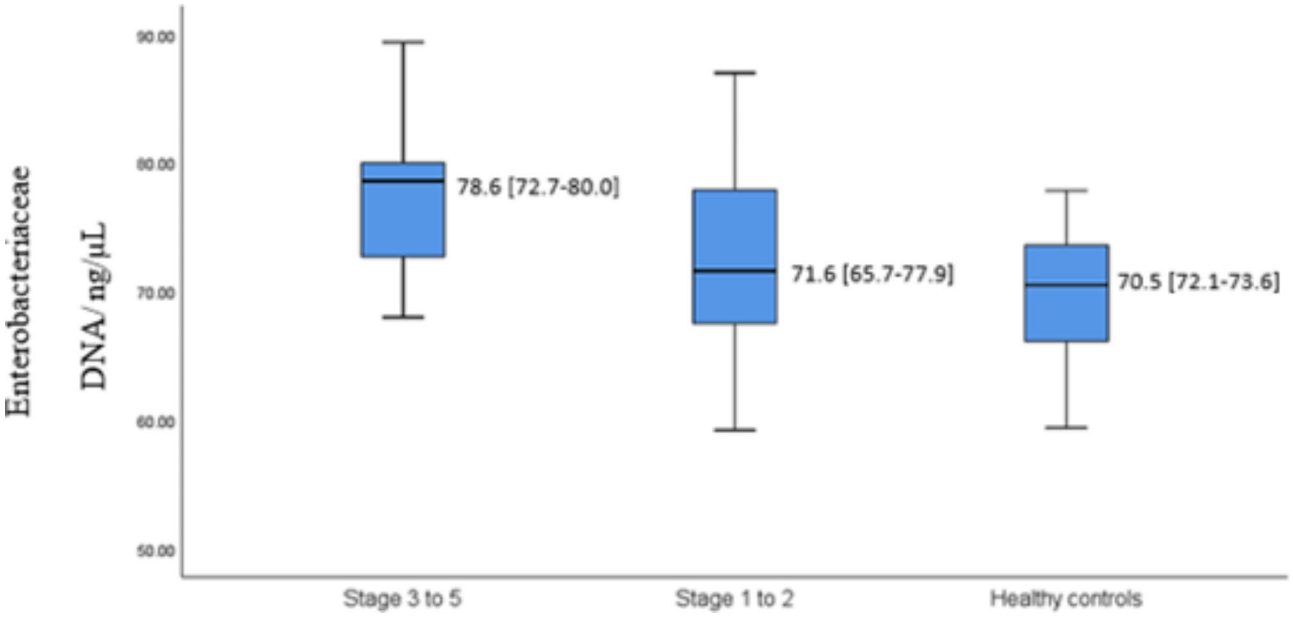

Fig. 5 Comparison of Enterobacteriaceae abundance in CKD patients and healthy controls

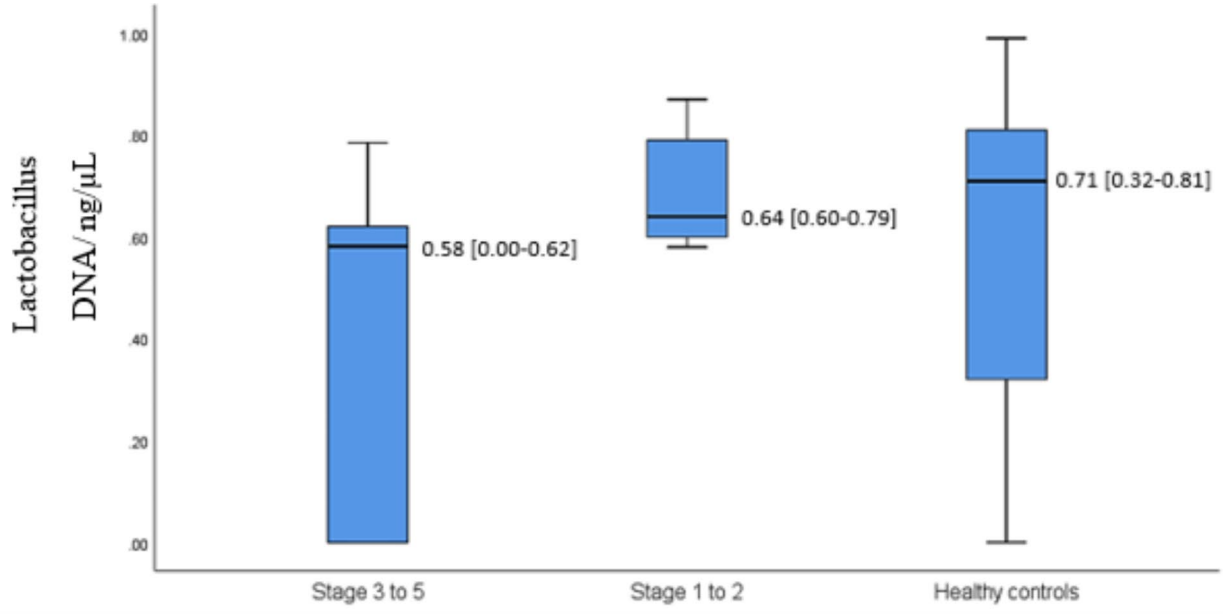

Fig. 6 Comparison of Lactobacillus relative abundance

similar to that of Hobby et al. [32], who described a reduced abundance of *Lactobacillus plantarum* in adults on peritoneal dialysis compared to controls. Butyrate-producing Lactobacillaceae were reported to have a lower abundance in CKD patients than in controls [15] and a lower abundance in postnephrectomy rats than in controls [33]. The reduction in the abundance of *Lactobacillus* in the gut with declining kidney function highlights a potential target for therapeutic interventions to improve dysbiosis, such as synbiotic supplementation or dietary modifications. This could equally be relevant for preventive strategies to delay CKD progression, especially in a setting with limited access to renal replacement therapy [14]. Moreover, *Lactobacillus* abundance was significantly lower in ADPKD CKD G 3–5 patients than in ADPKD CKD G 1–2 patients. Similarly, Gryp et al. reported that the abundance of *Lactobacillus* decreased with decreasing kidney function [14]. Our result is unlike that of Yacoub et al. [18], who reported an increase in *Lactobacillus iners* with respect to kidney function in PKD patients. This difference could be explained by the

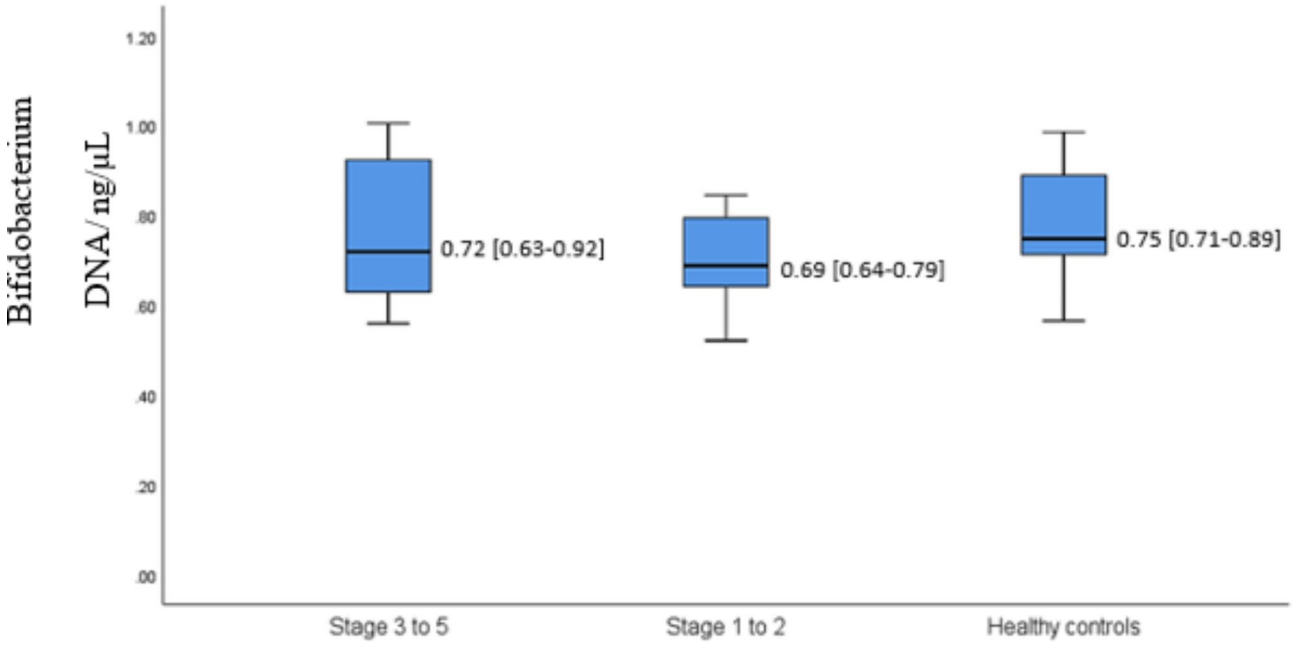

Fig. 7 Comparison of the relative abundance of Bifidobacterium

fact that our study targeted the genus while theirs targeted a species.

The median DNA abundance of Enterobacteriaceae was greater in the ADPKD CKD G 3–5 group than in the control group ($p = 0.045$). This suggests that individuals with ADPKD who experienced a greater decrease in kidney function had a greater average abundance of Enterobacteriaceae than did healthy individuals serving as controls. An impaired kidney function is associated with the growth of proteolytic bacteria such as those of the Enterobacteriaceae family, which leads to an increase in uremic toxins such as p-cresyl sulfate and indoxyl sulfate. These toxins promote oxidative stress, inflammation and/or fibrosis, potentially exacerbating systemic complications and furthering the progression of CKD. Our finding is on par with that of Vaziri et al., who reported a greater abundance of Enterobacteriaceae in end-stage kidney disease (ESKD) patients than in controls [33]. Chung et al. [4] reported similar results in patients with progressing and nonprogressing immunoglobulin A nephropathy. Other studies yielded findings that aligned with these results [15, 32]. Because of the implications of elevated Enterobacteriaceae levels in CKD patients, such as an increased risk of gastrointestinal and systemic complications related to inflammation, this result provides opportunities for targeted interventions to mitigate inflammation and potentially slow the progression of CKD [14]. Additionally, Enterobacteriaceae abundance was significantly greater in ADPKD CKD G 3–5 patients than in ADPKD CKD G 1–2 patients, highlighting the association of kidney function with aerobic Enterobacteriaceae. This relationship was also reported by Gryp et al. for CKD patients with CKD stages 1–5, and they explained that it was due to elevated pH from the accumulation of ammonia in CKD, which favours urease-expressing bacteria [14]. The latter study equally reported an increased abundance of Enterobacteriaceae in haemodialysis patients compared to patients in the early stages of CKD.

The median *abundance of Bifidobacterium* DNA in the ADPKD CKD 3–5 group was not significantly different from that in the control group ($p = 0.751$). *Bifidobacterium* contributes to host health through the biosynthesis of vitamins and essential amino acids and generates renoprotective short-chain fatty acids (SCFAs) through the fermentation of complex carbohydrates. However, unlike *Lactobacillus*, its abundance did not decrease as kidney function decreased. This could be explained by the fact that *Bifidobacterium* is able to switch to amino acid metabolism, the main substrate in a uremic milieu, in the absence of carbohydrates [14], or this could be due to the sample size of this study. Our finding is unlike that of Chung et al., who carried out a systematic review on the gastrointestinal microbiota in patients with CKD. They reported that the abundance of Bifidobacterium was lower in adult patients undergoing peritoneal dialysis than in healthy controls and that the abundance of Bifidobacteriaceae was lower in patients who had nonprogressing immunoglobulin A nephropathy. Moreover, they reported an experimental study that revealed a lower baseline abundance of *Bifidobacterium* in haemodialysis patients than in healthy controls [4]. Other reviews

reported findings similar to those of Chung et al. [15, 32, 34]. This may be attributed to the increase in intestinal urea associated with CKD, which is not favourable for the growth of normal flora saccharolytic bacteria such as *Bifidobacterium*, *Lactobacillus*, *Prevotella* and *Roseburia*.

This study is one of the few that has employed molecular techniques to quantitatively evaluate the gut microbiota composition in a Sub-Saharan African population while also assessing changes across CKD stages within a specific disease population. The results provide novel insights into microbiota-kidney function associations in the Cameroonian ADPKD population. A limitation of this study was the absence of duplicate PCR assays and absolute bacteria quantification. Additional samples and technique optimization in future studies can help validate the alterations across CKD stages observed here.

Conclusion

These exploratory findings revealed a decrease in symbionts and an increase in pathobionts as kidney function worsened. This trend aligned with previous reports and may indicate that the microbiota could play a contributory role in CKD progression. For instance, a longitudinal study could assess the impact of the gut microbiota on CKD progression in ADPKD. Overall, this study sets the stage in an understudied area and population using molecular tools. The observed dysbiotic patterns lay the foundation for larger follow-up studies to elucidate mechanisms and therapeutic targets related to the gut–kidney axis in ADPKD.

Abbreviations

ADPKD	Autosomal dominant polycystic kidney disease
BMI	Body mass index
BP	Blood presssure
CKD	Chronic kidney disease
CKD-EPI	Chronic kidney disease epidemiology collaboration
eGFR	Estimated glomerular filtration rate
GIT	Gastrointestinal tract
IBM-SPSS	International business machines- statistical package for the social sciences
KDIGO	Kidney disease improving global outcomes
MRC	Maladie rénale chronique
PCR	Polymerase chain reaction
PKD	Polycystic kidney disease
PKRAD	Polykystose rénale autosomique dominante
SCFA	Short chain fatty acids

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-03942-6.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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Author contributions

ION: conceived and designed the protocol, collected and analysed the data, and drafted and reviewed the manuscript. VBN, MM: refined the protocol, provided the data and critically reviewed the manuscript. RNB: critically reviewed the manuscript. ID and CC: analysed the data and critically reviewed the manuscript. EN, AN, JCM, FAA: reviewed the manuscript. VAM and WFM supervised and critically reviewed the manuscript. FFK: provided the data and supervised and critically reviewed the manuscript.

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Data availability

The clinical, biological, and microbiota abundance data and summary statistics supporting the findings of this study are openly available in the Gut-Microbiota-ADPKD repository at https://github.com/InesObolo/Gut-Microbiota-ADPKD. The data that support the findings are included within the manuscript.

Declarations

Ethics approval and consent to participate

This cross-sectional study was approved by the Institutional Review Board of the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé 1 (Ref N° 0025/UY1/FMSB), Cameroon. This study was equally approved by the Yaoundé University Teaching Hospital (03/AR/UY1) and the Yaoundé General Hospital (022–023/HGY/DG). All subjects provided written informed consent for inclusion in the study, which was conducted in accordance with the Declaration of Helsinki.

Consent for publication

All authors have given their consent for the publication of this article.

Competing interests

The authors declare no competing interests.

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