Risk of Post-Lung Transplant Renal Dysfunction in Adults With Cystic Fibrosis

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Background: Cystic fibrosis (CF) is one of the leading indications for lung transplantation. The incidence and pre-lung transplant risk factors for posttransplant renal dysfunction in the CF population remain undefined.

Methods: We conducted a cohort study using adults (≥ 18 years old) in the CF Foundation Patient Registry from 2000 to 2008 to determine the incidence of post-lung transplant renal dysfunction, defined by an estimated glomerular filtration rate of < 60 mL/min/1.73 m². Multivariable Cox proportional hazards modeling was used to identify independent pretransplant risk factors for post-lung transplant renal dysfunction.

Results: The study cohort included 993 adult lung transplant recipients with CF, with a median follow-up of 2 years. During the study period, 311 individuals developed renal dysfunction, with a 2-year risk of 35% (95% CI, 32%-39%). Risk of posttransplant renal dysfunction increased substantially with increasing age (25 to < 35 years vs 18 to < 25 years: hazard ratio [HR], 1.60; 95% CI, 1.15-2.23; vs ≥ 35 years: HR, 2.45; 95% CI, 1.73-3.47) and female sex (HR, 1.56; 95% CI, 1.22-1.99). CF-related diabetes requiring insulin therapy (HR, 1.30; 95% CI, 1.02-1.67) and pretransplant renal function impairment (estimated glomerular filtration rate, 60-90 mL/min/m² vs > 90 mL/min/m²: HR, 1.58; 95% CI, 1.19-2.12) also increased the risk of posttransplant renal dysfunction.

Conclusions: Renal dysfunction is common following lung transplant in the adult CF population. Increased age, female sex, CF-related diabetes requiring insulin, and pretransplant renal impairment are significant risk factors.

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Abbreviations: CF = cystic fibrosis; CFRD = cystic fibrosis-related diabetes; CF Registry = Cystic Fibrosis Foundation Patient Registry; eGFR = estimated glomerular filtration rate; HR = hazard ratio; KDOQI = Kidney Disease Outcome Quality Initiative

Cystic fibrosis (CF) is the third most common indication for lung transplantation worldwide and, alongside COPD, is the most common indication for bilateral lung transplantation.¹ Lung transplant

survival has improved significantly over the past 2 decades, and currently 50% of CF lung transplant recipients can expect to live > 7 years.¹ Improved post-lung transplant survival has been largely attributed to increased 1-year survival due to advances in surgical technique and postoperative care and improvements in immunosuppressive regimens leading to reduced rates of acute rejection.^{1,2}

As a result of improved post-lung transplant survival, CF lung transplant recipients can expect to experience multiple medical complications over time related to transplant.³ Renal dysfunction is a well-recognized complication of nonrenal organ transplantation.^{4,6} Although its cause is often multifactorial,

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calcineurin-inhibitor immunosuppressive medications are believed to play an important role.⁶⁻⁸ In the overall lung transplant population, the cumulative incidence of posttransplant renal dysfunction has been reported to range between 4% and 35%, depending on how renal dysfunction is defined and the severity of renal dysfunction.^{1,9-12} Renal dysfunction complicates posttransplant management, as it usually prompts a reduction in the dosing of immunosuppressive medications or, if severe enough, necessitates a change in the immunosuppressive regimen.⁷ It can also progress to require dialysis and/or kidney transplantation.¹³ Most importantly, a posttransplant diagnosis of end-stage chronic renal failure has been shown to be associated with reduced survival.^{2,8}

To our knowledge, no previous studies have looked at the incidence or predictors of posttransplantation renal dysfunction in the CF population. Previous studies of post-lung transplant renal dysfunction have been single center¹⁰ and/or have not looked specifically at the CF population.^{8,9,11} We suspect individuals with CF might be at heightened risk for developing renal dysfunction post-lung transplant because of increased risk for dehydration¹⁴ and repeated exposure to nephrotoxic antibiotics used to treat chronic pulmonary infections.¹⁵ Furthermore, previously identified pretransplant risk factors for post-lung transplant renal dysfunction, including increased age, hypertension, and diabetes mellitus, may not be applicable to the much younger CF lung transplant recipient population.⁸ Identifying pretransplant factors associated with renal dysfunction post-lung transplant is important, as it can identify individuals at highest risk and lead to more vigilant monitoring of renal function in such individuals.

MATERIALS AND METHODS

Study Population and Data Sources

Data from the US Cystic Fibrosis Foundation Patient Registry (CF Registry) were used for this study, which contains longitudinal data on 34,937 patients from > 110 CF-accredited care centers spanning the years 2000 to 2008.¹⁶ We studied adults with CF (≥ 18 years old) who underwent their first lung transplantation from January 1, 2000, to December 31, 2008, and had at least one estimated glomerular filtration (eGFR) measurement over this time period. Individuals entered the study cohort following lung transplantation and were followed to the earliest of the following: renal dysfunction diagnosis, second lung transplantation, death, or last follow-up.

Outcome Definition

Renal function was estimated using the Cockcroft-Gault formula standardized for body surface area, and the following variables were required to perform this calculation: serum creatinine, age, sex, weight, and height.^{17,18} Posttransplant serum creatinine values

were obtained from encounter-level CF Registry data, and age, sex, weight, and height were obtained from annual-level CF Registry data. Renal dysfunction was defined by eGFR measured < 60 mL/min/1.73 m² on at least one occasion at least 1 month posttransplant. This eGFR threshold corresponds to stage 3 chronic kidney disease severity based on National Kidney Foundation Kidney Disease Outcome Quality Initiative (KDOQI) guidelines and is the earliest stage that can be diagnosed using serum creatinine alone, without concomitant evidence of kidney damage.¹⁹ Our definition of renal dysfunction was not referred to synonymously as chronic kidney disease, as KDOQI guidelines require at least two eGFR measurements of < 60 mL/min/1.73 m² separated by ≥ 3 months. Our definition did not require two measurements of < 60 mL/min/1.73 m² separated by at least 3 months, as this could have selected for cases among individuals who lived longer, resulting in a healthy survivor bias. We did not count transient renal dysfunction that was identified only in the first month posttransplant as cases, as these most likely represented acute kidney injury related to perioperative hemodynamic insults. Patients were excluded from the analysis if they had renal dysfunction (eGFR < 60 mL/min/1.73 m²) prior to lung transplantation.

Classification of Exposures

Pretransplantation predictors of posttransplant renal dysfunction were characterized using annual-level CF Registry data from the year prior to transplant. Predictors of interest included recipient age, sex, BMI, evidence of CF-related diabetes (CFRD), evidence of hypertension, evidence of infection by *Pseudomonas aeruginosa*, era of lung transplant being performed, and evidence of impaired renal function (eGFR 60-90 mL/min/1.73 m²) prior to transplant. Age was categorized as 18 to < 25 years old (reference), 25 to < 35 years old, and ≥ 35 years old. BMI was categorized as < 18 kg/m², 18 to < 22 kg/m² (reference), 22 to < 25 kg/m², and ≥ 25 kg/m². To ensure a consistent case definition of CFRD, it was defined as CFRD requiring insulin therapy. Era of lung transplant was categorized by the approximate midpoint of the time period spanned by the cohort: 2000 to 2003 vs 2004 to 2008.

Statistical Analyses

Descriptive analyses were used to summarize baseline characteristics in the year prior to lung transplant stratified by renal dysfunction status. Differences, 95% CIs for differences, and *P* values between groups were reported for continuous variables using the Student *t* test. Percent of nonmissing data and *P* values were reported for categorical variables between groups using the χ^2 test. Kaplan-Meier estimator was used to plot cumulative incidence over time. Multivariable Cox proportional hazard modeling was used to identify independent predictors of posttransplant renal dysfunction. Pretransplant exposures were treated as time-fixed covariates.

Three sensitivity analyses were defined a priori to assess the robustness of our results (e-Appendix 1 and e-Table 1). A two-sided *P* value $< .05$ was considered statistically significant for all analyses. Analyses were performed using Stata 10.0 (StataCorp). The institutional review board at the University of Washington approved this study (IRB Application No. 38862).

RESULTS

Cohort Selection

Among the > 110 CF accredited care centers in the United States, there were 1,188 lung transplantations performed in 29,376 adults (≥ 18 years of age) during

the study period of January 1, 2000, to December 31, 2008 (Fig 1). Following the exclusion of 148 lung transplant recipients without renal function data, we were left with 1,040 transplants. Forty-seven individuals had renal dysfunction prior to transplant and were subsequently excluded from the posttransplant renal dysfunction analysis. The final number of lung transplant recipients at risk for posttransplant renal dysfunction was 993. The cohort was followed for a median of 2 years, for a total follow-up of 2,284 person-years. Twenty-five percent of the cohort did not have renal function measurements available beyond the year of transplant.

Risk of Post-Lung Transplant Renal Dysfunction

There were 311 cases of post-lung transplant renal dysfunction defined by eGFR < 60 mL/min/1.73 m² (≥ stage 3 chronic kidney disease) detected over the observation period of 2000 to 2008. Based on Kaplan-Meier estimates, the risk of renal dysfunction was 22.5% (95% CI, 19.6%-25.7%), 35.4% (95% CI, 31.7%-39.3%), and 57.6% (95% CI, 51.9%-63.3%) at 1, 2, and 5 years post-lung transplant, respectively

FIGURE 1. Diagram of study population at risk for post-lung transplant renal dysfunction along with cohort follow-up time and number of renal dysfunction events. CF = cystic fibrosis.

(Fig 2). Fifty-five percent of renal dysfunction cases occurred within the first year posttransplant and 95% of cases were diagnosed within 5 years post-lung transplant. When analyzed by calendar year of lung transplant, the 1-, 2-, and 5-year risks of post-lung transplant renal dysfunction decreased from 2001 to 2002 but has remained fairly stable since 2002 (Table 1).

Ninety-eight of 311 (31.5%) post-lung transplant renal dysfunction cases progressed to ≥ stage 4 chronic kidney disease (eGFR of < 30 mL/min/1.73 m² and/or required dialysis). Thirty-seven of 311 (11.9%) progressed to stage 5 chronic kidney disease (eGFR < 15 mL/min/1.73 m² and/or required hemodialysis).

Cohort Characteristics

Characteristics of the study cohort in the year prior to lung transplant comparing those who developed renal dysfunction with those who did not are shown in Table 2. CF lung transplant recipients who developed posttransplant renal dysfunction were older (32.8 vs 28.2 years; mean difference, 4.6 years; 95% CI for difference, 3.4-5.8 years), more likely to be female (56.3% vs 43.4%; mean difference, 12.9%; 95% CI for difference, 6.2%-19.5%), and to be culture positive for *P. aeruginosa* (91.3% vs 87.1%; mean difference, 4.2%; 95% CI for difference, 0.2%-8.2%). The posttransplant renal dysfunction group was also more likely to have renal impairment defined by an eGFR between 60 and 90 mL/min/1.73 m² prior to transplant (29.0% vs 15.8%; mean difference, 13.2%; 95% CI for difference, 7.2%-19.2%) and be transplanted prior to the year 2004 (59.5% vs 33.6%; mean difference, 25.9%; 95% CI for difference, 19.4%-32.4%). Other pretransplant factors, including BMI, prevalence of CFRD requiring insulin, and hypertension, were similar between groups. Individuals with posttransplant renal dysfunction were more likely to require hemodialysis (10.3% vs 2.8%; mean difference, 7.5%; 95% CI for difference, 3.9%-11.1%).

Multivariable Association Between Exposures of Interest and Post-Lung Transplant Renal Dysfunction

Based on multivariable Cox proportional hazards regression, the risk of posttransplant renal dysfunction increased significantly with increasing recipient age, with a hazard ratio (HR) of 1.60 (95% CI, 1.15-2.23) for recipients aged 25 to < 35 years and an HR of 2.45 (95% CI, 1.73-3.47) for recipients aged ≥ 35 years, compared with recipients aged 18 to < 25 years (Table 3). The risk of posttransplant renal dysfunction was higher for women relative to men, with an HR of 1.56 (95% CI, 1.22-1.99). A diagnosis of

FIGURE 2. Kaplan-Meier estimates of the risk of post-lung transplant renal dysfunction over time in years.

insulin-requiring CFRD significantly increased the risk of renal dysfunction with an HR of 1.30 (95% CI, 1.02-1.67) compared with recipients without this diagnosis.

Pretransplant BMI was not independently associated with posttransplant renal dysfunction (BMI < 18 kg/m² vs 18 to < 22 kg/m²: HR, 1.01; 95% CI, 0.77-1.33; BMI 22 to < 25 kg/m²: HR, 0.78; 95% CI, 0.52-1.17; BMI ≥ 25 kg/m²: HR, 1.01; 95% CI, 0.77-1.33). A pretransplant diagnosis of hypertension (HR, 0.84; 95% CI, 0.21-3.40) and growth of *P. aeruginosa* from sputum culture (HR, 1.28; 95% CI, 0.80-2.05) were also not independently associated with posttransplant renal dysfunction.

The risk of posttransplant renal dysfunction was significantly higher for individuals with renal function impairment (eGFR, 60-90 mL/min/1.73 m²) in the year prior to transplant, with an HR of 1.58 (95% CI,

1.19-2.12) compared with recipients with normal renal function (eGFR > 90 mL/min/1.73 m²) pre-transplant. Although receiving a lung transplant between 2000 and 2003 increased the risk of post-transplant renal dysfunction compared with 2004 to 2008, with an HR of 1.34 (95% CI, 1.05-1.72), this analysis might be biased because of a shorter follow-up and thus incomplete case ascertainment for transplants performed in more recent years.

Sensitivity Analysis

First, as a conservative estimate of the risk of post-lung transplant renal dysfunction, we assumed that all individuals censored because of loss to follow-up were followed until the end of the study period and did not develop renal dysfunction. The risk of renal dysfunction was lower than the primary analysis but remained high and was 17.4% (95% CI, 15.2%-19.9%), 23.9% (95% CI, 21.4%-26.7%), and 29.7% (95% CI, 27.0%-32.7%) at 1, 2, and 5 years post-lung transplant, respectively (e-Figure 1). Second, a multivariable analysis was performed to examine the potential influence of informative censoring for individuals dying in the first year post-lung transplant. Individuals dying within the first year post-lung transplant were assumed to have renal dysfunction. Our risk estimates were very similar to our primary analysis (e-Table 2). Third, a restricted multivariable analysis from 2002 to 2008 was performed to determine the potential influence of miscoding or misdiagnosis of CFRD-requiring insulin in earlier CF Registry years. The relative risk estimate of CFRD requiring insulin increased slightly and remained statistically significant (e-Table 3).

DISCUSSION

Our cohort study is the first, to our knowledge, to examine the incidence and predictors of renal dysfunction in the adult CF population following lung transplantation. Previous observational studies have used nonstandardized definitions of renal dysfunction^{1,9-11} or focused on renal dysfunction in the overall lung transplant population.^{4,8,9,11} The CF lung transplant recipient population is unique, as patients are younger and may have different pretransplant predictors of posttransplant renal dysfunction.

Renal dysfunction (equivalent to ≥ stage 3 chronic kidney disease) was very common following lung transplantation in the adult CF population, with a 2-year risk of 35% and a 5-year risk of 58%. Thirty-two percent of cases progressed to stage 4 disease or greater, 12% progressed to stage 5 disease, and 10% progressed to require hemodialysis. Interestingly, the risk of posttransplant renal dysfunction decreased stepwise

Table 1—1-, 2-, and 5-y Kaplan-Meier Risk Estimates of Post-Lung Transplant Renal Dysfunction Stratified by Year of Lung Transplant

Year of LTx	Kaplan-Meier Risk Estimate of Renal Dysfunction (95% CI)		
	1 Year Post-LTx ^a	2 Years Post-LTx ^b	5 Years Post-LTx ^c
2000	26 (18-37)	52 (41-64)	68 (56-80)
2001	38 (29-49)	51 (41-62)	66 (54-78)
2002	18 (11-28)	29 (20-41)	52 (40-65)
2003	21 (13-32)	33 (23-46)	56 (42-70)
2004	18 (12-28)	25 (17-35)	...
2005	18 (12-28)	26 (18-37)	...
2006	19 (13-30)	25 (17-35)	...
2007	15 (9-25)	...	...

Data are presented as %. LTx = lung transplant.

^a2008 excluded because < 1 y of posttransplant follow-up.

^b2007 and 2008 excluded because < 2 y of posttransplant follow-up.

^c2004 to 2008 excluded because < 5 y of posttransplant follow-up.

Table 2—Pre-Lung Transplant Characteristics of Recipients Who Did and Did Not Develop Post-Lung Transplant Renal Dysfunction

LTx Recipient Characteristic ^a	No RD (n = 682)	RD (n = 311)	Difference ^b (95% CI)	P Value ^c
Mean age, y	28.2	32.8	4.6 (3.4 to 5.8)	< .001
Age category, %				
18 to < 25 y	39.3	18.8	...	...
25 to < 35 y	40.0	44.2	...	< .001
≥ 35 y	20.7	37.0	...	...
Female sex, %	43.4	56.3	12.9 (6.2 to 19.5)	< .001
Mean BMI, kg/m ²	19.3	19.6	0.3 (0.2 to 0.7)	.3
BMI category, %				
< 18	32.7	28.9	...	...
18 to < 22	53.9	56.1	...	.6
22 to < 25	10.4	11.5	...	...
≥ 25	3.0	3.6	...	...
Impaired renal function, ^d %	15.8	29.0	13.2 (7.2 to 19.2)	< .001
CFRD requiring insulin, %	33.4	37.6	4.2 (−2.3 to 10.7)	.2
Hypertension, %	1.5	1.6	0.1 (−1.5 to 1.8)	.9
<i>Pseudomonas aeruginosa</i> culture positive, %	87.1	91.3	4.2 (0.2 to 8.2)	.04
Lung transplant prior to 2004, %	33.6	59.5	25.9 (19.4 to 32.4)	< .001

CFRD = cystic fibrosis-related diabetes; eGFR = estimated glomerular filtration rate; RD = renal dysfunction. See Table 1 legend for expansion of other abbreviation.

^aFrom the year prior to lung transplant.

^bAbsolute difference with 95% CI.

^cTwo-sided *P* value for *t* test or χ^2 test.

^dDefined by eGFR 60-90 mL/min/1.73 m².

from 2001 to 2002 but has remained fairly stable since 2002. Temporally, the stepwise decrease might be related to the switch from cyclosporine to tacrolimus as the calcineurin inhibitor of choice. Tacrolimus has been shown to be less nephrotoxic than cyclosporine.^{8,20,21} Although we do not have data on the type of calcineurin inhibitor used following transplant to examine trends in usage over time, cyclo-

sporine use was reported in two very large studies to be 80% from 1990 to 2000⁸ compared with 15% from 2002 to 2009.¹

Despite the younger age of CF lung transplant recipients relative to other pretransplant diagnoses, such as COPD and idiopathic pulmonary fibrosis, increased age was a strong risk factor for posttransplant renal dysfunction, similar to previous studies on the overall lung transplant population.^{8,9,12} Recipients between 25 and 35 years old had a 1.6-fold increase in risk compared with recipients between 18 and 25 years old. In a previous study, increased risk with age was believed to be due to a greater burden of atherosclerotic vascular disease.⁹ A similar explanation would not seem likely in the 25- to 35-year old population with CF. However, increased age in the pretransplant CF population likely correlates with lifetime exposure to nephrotoxic IV aminoglycoside antibiotics. In a previous study, renal function was inversely associated with lifetime IV aminoglycoside exposure.¹⁵

Interestingly, insulin-requiring CFRD was a significant risk factor for posttransplant renal dysfunction, suggesting that CFRD may have similar posttransplant renal consequences as diabetes mellitus observed in the non-CF population.⁸ A recent study by our group demonstrated that CFRD requiring insulin is a strong risk factor for chronic kidney disease in the nontransplanted adult CF population.²² This study builds on our previous finding and raises the importance of aggressively managing

Table 3—Final Multivariable Cox Proportional Hazard Model

Pre-LTx Exposure ^a	Post-LTx Renal Dysfunction, HR (95% CI)
Age, y	
18 to < 25	Ref
25 to < 35	1.60 (1.15-2.23)
≥ 35	2.45 (1.73-3.47)
Female sex	1.56 (1.22-1.99)
BMI, kg/m ²	
< 18	1.01 (0.77-1.33)
18 to < 22	Ref
22 to < 25	0.78 (0.52-1.17)
≥ 25	1.00 (0.48-2.08)
Renal function impairment ^b	1.58 (1.19-2.12)
CFRD (insulin-requiring)	1.30 (1.02-1.67)
Hypertension	0.84 (0.21-3.40)
<i>Pseudomonas aeruginosa</i> culture positive	1.28 (0.80-2.05)
LTx performed prior to 2004	1.34 (1.05-1.72)

HR = hazard ratio; Ref = reference. See Table 1 legend for expansion of other abbreviations.

^aFrom the year prior to lung transplant.

^bDefined by eGFR 60-90 mL/min/1.73 m².

CFRD to minimize the risk of long-term complications such as posttransplant renal dysfunction.

Female transplant recipients exhibited a higher risk of posttransplant renal dysfunction. This unexpected finding was also found in the largest study of chronic renal dysfunction in nonrenal transplants performed to date, but the study authors did not speculate on cause.⁸ Although the Cockcroft-Gault estimating equation for GFR used in our study may have underestimated renal function in women because of the built-in correction factor of 0.85, we believe this is unlikely, as both men and women had a similar mean eGFR prior to lung transplant (eGFR men, 116 mL/min/1.73 m² vs women, 115 mL/min/1.73 m²; $P = .75$). We speculate that posttransplant renal function monitoring, with overreliance on serum creatinine alone, may lead to discrepancies in care and renal outcomes for women. Women can have “normal” serum creatinine values despite depressed renal function, because of lower muscle mass compared with males.²¹ Missed diagnoses of renal impairment or renal dysfunction in women can lead to inappropriate dosing of nephrotoxic immunosuppressive agents and antibiotics, thus leading to further renal damage. Furthermore, women have reduced nephron mass relative to men and therefore might be more vulnerable to the nephrotoxic effects of calcineurin inhibitors.^{23,24}

Our study is subject to a few notable limitations. By requiring at least one post-lung transplant renal function measurement, the survival of our post-lung transplant cohort was slightly better than the overall CF lung transplant population, because of immortal time bias (e-Figure 2). This bias is more likely to lead to an underestimate of the incidence of renal dysfunction in our cohort population given the worse clinical outcome of those without serum creatinine values recorded. Second, our definition of renal dysfunction only required a single eGFR measurement of < 60 mL/min/1.73 m² compared with the National Kidney Foundation KDOQI guidelines definition for chronic kidney disease, which requires at least two eGFR measurement of < 60 mL/min/1.73 m² separated by at least 3 months. Our definition did not require repeat measurement after 3 months because of concern about selecting for cases in those individuals living longer (akin to the healthy survivor effect), which could have led to spurious associations between our potential predictors and outcomes. Although our definition of renal dysfunction could have represented cases of acute kidney injury that might have subsequently resolved in follow-up, this did not appear to be the case. Following exploratory analysis, 86% of our cases of renal dysfunction had eGFR measurements of < 60 mL/min/1.73 m² after at least 3 months of follow-up and thus represented cases of chronic

kidney disease. Third, the median follow-up time of our cohort was relatively short at 2 years, as 25% of transplant recipients did not have serum creatinine values recorded in the registry beyond the year of lung transplant. This is likely a result of the majority of patient care being transferred to transplant clinics and/or a lack of recording of serum creatinine by accredited CF care centers in the CF Registry post-transplant. However, we do not believe our results were biased by informative censoring, as the baseline characteristics of individuals censored vs not censored within 1 year of lung transplant were roughly similar (e-Table 4). Fourth, we could not directly assess the impact of pretransplant IV aminoglycoside exposure due to a lack of this variable or a reliable surrogate in the CF Registry. However, the increased risk of posttransplant renal dysfunction with increased age, even among younger age groups, is compelling and suggestive of increased risk due to an age-related exposure, such as lifetime IV aminoglycoside use. Fifth, there was a gradual change in the creatinine assay technique over time from the Jaffe method to isotope-dilution mass spectrometry, with the new methodology resulting in slightly lower serum creatinine values.²⁵ As we have selected to focus on moderate to severe renal dysfunction, the impact of subtle changes in creatinine are unlikely to have meaningfully changed our results. Last, we could not assess the risk of other posttransplant factors, such as calcineurin inhibitor use, because of the absence of such variables in the CF Registry. Nevertheless, a better understanding of pretransplant risk factors is important for counseling patients on the risks of posttransplant renal dysfunction and for identifying those individuals who require the closest monitoring post transplant.

In conclusion, to assess the incidence and predictors of post-lung transplant renal dysfunction in adults with CF, we have longitudinally analyzed 993 lung transplants from 2000 to 2008. During that period, the 2-year risk of posttransplant renal dysfunction was 35%. Despite the uniqueness of the CF lung transplant recipient population, our findings support a previous study of all lung transplant recipients, as we have similarly identified age, female sex, pretransplant renal impairment, and diabetes as important risk factors for post-lung transplant renal dysfunction.⁸

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Dr Mayer-Hamblett: contributed to study conception and design, analysis and interpretation, and drafting the manuscript for important intellectual content.

Dr Aitken: contributed to drafting the manuscript for important intellectual content.

Dr Goss: contributed to study conception and design, analysis and interpretation, and drafting the manuscript for important intellectual content.

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Additional information: The e-Appendix, e-Figures, and e-Tables can be found in the "Supplemental Materials" area of the online article.

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