Mortality remains one of the most important outcomes in dialysis, from the point of view of patients, caregivers, and health service sector stakeholders.1-4 Due to the lack of randomized controlled trials, the effect of dialysis modality on mortality has been uncertain. Up until the turn of the last century, observational studies suggested that peritoneal dialysis (PD) was associated with worse outcomes than hemodialysis (HD) in terms of mortality for the average patient, as well as uncertain benefits in terms health-related quality of life.4 More recent observational studies have suggested that PD has a reasonably similar survival to HD5-10 with better health-related quality of life.11-17 Consequently, modality choice (where choice is available) is often made largely on the basis of “hard” medical contraindications and lifestyle preference alone.18

We have previously analyzed mortality rates by modality in the Australia and New Zealand (ANZ) dialysis population. These early observational studies showed home HD to be associated with better survival than facility HD, and PD with worse survival.19-22 However, our subsequent observational studies have shown that mortality rates are improving faster for PD and home HD than for facility HD.23,24 In the current observational study, we compare mortality risk by dialysis modality in a cohort from 2013-2017, contrasting the findings with historical cohorts. We used data from the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA), accounting for patient characteristics, treating center, and kidney transplantation as either a censoring or competing event. We tested the hypothesis that all forms of home dialysis are currently associated with a lower mortality risk than facility HD.

Methods
A more detailed description of methods is contained Item S1. We examined outcomes for all adult patients in ANZDATA who commenced dialysis in the 20 years to December 31, 2017. The National (NZ) Health and Disability Ethics Committee (IORG0000895) approved the study protocol and waived the need for patient consent under the provisions for observational research.

The primary exposure in this study is dialysis modality. The exposure of facility HD was defined as dialysis at a staffed dialysis HD facility, and the exposures of continuous ambulatory peritoneal dialysis (CAPD), automated PD (APD), and home HD, defined as dialysis in an unstaffed setting of a domiciliary or communal nature.25-28 Dialysis
Table 1. Clinical Characteristics of the Study Cohort by Era of Dialysis Inception (the Start of Dialysis)

| Variable                        | 1998-2002 | 2003-2007 | 2008-2012 | 2013-2017 | P* |
|---------------------------------|-----------|-----------|-----------|-----------|----|
| No. of patients                 | 10,459    | 12,716    | 14,072    | 14,850    |    |
| Age, y                          | 61 [48-71]| 62 [50-72]| 62 [51-72]| 62 [51-72]| <0.001 (<0.01) |
| Male sex                        | 6,119 (58.5%) | 7,667 (60.3%) | 8,611 (61.2%) | 9,236 (62.2%) | <0.001 (0.03) |
| Ethnicity                       |           |           |           |           |    |
| ANZ European                    | 7,783 (74.4%) | 9,318 (73.3%) | 9,817 (69.8%) | 9,080 (61.1%) | <0.001 (0.2) |
| Aboriginal/TSI                  | 783 (7.5%) | 1,027 (8.1%) | 1,143 (8.1%) | 1,342 (9.0%) |    |
| Asian                           | 732 (7.0%) | 946 (7.4%) | 1,280 (9.1%) | 1,486 (10.0%) |    |
| NZ Maori                        | 682 (6.5%) | 778 (6.1%) | 880 (6.3%) | 947 (6.4%) |    |
| Pacific People                  | 419 (4.0%) | 548 (4.3%) | 736 (5.2%) | 985 (6.6%) |    |
| Other                           | 60 (0.6%) | 99 (0.8%) | 216 (1.5%) | 1,010 (6.8%) |    |
| Late referral                   | 2,634 (25.2%) | 3,141 (24.7%) | 3,068 (21.8%) | 2,586 (17.4%) | <0.001 (0.08) |
| eGFR, mL/min/1.73 m²            | 5.8 [4.3-8.0] | 6.6 [4.7-9.3] | 7.2 [5.1-10.1] | 7.0 [5.1-9.9] | <0.001 (<0.01) |
| Smoking                         | 1,463 (14.0%) | 1,732 (13.6%) | 1,845 (13.2%) | 1,872 (12.6%) | 0.007 (0.02) |
| Diabetes mellitus               |           |           |           |           |    |
| Type 1                          | 422 (4.0%) | 424 (3.3%) | 523 (3.7%) | 752 (5.1%) |    |
| Type 2                          | 3,465 (33.1%) | 5,146 (40.5%) | 6,380 (45.3%) | 7,169 (48.3%) |    |
| Primary kidney disease          |           |           |           |           |    |
| Diabetic nephropathy            | 2,969 (28.4%) | 4,287 (33.7%) | 5,451 (38.7%) | 6,147 (41.4%) |    |
| Glomerulonephritis              | 2,921 (27.9%) | 3,030 (23.8%) | 3,019 (21.5%) | 2,782 (18.7%) |    |
| Hypertension/ischemic           | 1,410 (13.5%) | 1,878 (14.8%) | 1,955 (13.9%) | 2,028 (13.7%) |    |
| Polycystic kidney disease       | 621 (5.9%) | 781 (6.1%) | 800 (5.7%) | 839 (5.7%) |    |
| Reflux nephropathy              | 396 (3.8%) | 347 (2.7%) | 305 (2.2%) | 268 (1.8%) |    |
| Other                           | 2,142 (20.5%) | 2,393 (18.8%) | 2,542 (18.1%) | 2,786 (18.8%) |    |
| Comorbidity at baseline         |           |           |           |           |    |
| Coronary artery                 | 4,205 (40.2%) | 5,228 (41.1%) | 5,822 (41.4%) | 5,224 (35.2%) | <0.001 (0.05) |
| Peripheral vascular             | 2,909 (27.8%) | 3,192 (25.1%) | 3,638 (25.9%) | 3,103 (20.9%) | <0.001 (0.03) |
| Cerebrovascular                 | 1,608 (15.4%) | 1,942 (15.3%) | 2,147 (15.3%) | 1,788 (12.0%) | <0.001 (0.04) |
| Lung                            | 1,597 (15.3%) | 2,105 (16.6%) | 2,549 (18.1%) | 2,319 (15.6%) | <0.001 (0.06) |
| Comorbidity at end of FU        |           |           |           |           |    |
| Coronary artery                 | 4,355 (41.6%) | 5,396 (42.4%) | 6,134 (43.6%) | 5,416 (36.5%) | <0.001 (0.06) |
| Peripheral vascular             | 3,059 (29.3%) | 3,326 (26.2%) | 3,887 (27.6%) | 3,266 (22.0%) | <0.001 (0.06) |
| Cerebrovascular                 | 1,711 (16.4%) | 2,024 (15.9%) | 2,331 (16.8%) | 1,905 (12.8%) | <0.001 (0.04) |
| Lung                            | 1,652 (15.8%) | 2,168 (17.1%) | 2,662 (18.9%) | 2,454 (16.5%) | <0.001 (0.03) |
| BMI (kg/m²)                     | 25.6 [22.5-29.4] | 26.6 [23.2-31.0] | 275 [23.8-32.2] | 28.1 [24.3-33.1] | <0.001 (0.02) |
| BMI ≥ 30 kg/m²                  | 2,328 (22.3%) | 3,723 (29.3%) | 4,962 (35.3%) | 5,862 (39.5%) | <0.001 (0.15) |

N = 52,097. Continuous variables are shown as median [interquartile range]; categorical variables are shown as number (percentage). Abbreviations: ANZ, Australian and New Zealand; BMI, body mass index; eGFR estimated glomerular filtration rate; FU, follow-up; TSI, Torres Strait Islander.

*Effect size in parentheses. Effect size statistics describe the likely magnitude of differences; a value of 0.2 might be considered a small effect size, 0.5 a medium one, and 0.8 and greater a large effect size. For effect size calculations, see Item S1.

was modeled as the modality at 90 days after dialysis inception in one set of models, and as varying modality throughout the period of observation in another set of models but with a 90-day lag in the attribution of death to a given modality. Both these modeling approaches have the effect of excluding patients who were not alive on dialysis at 90 days.

We justified this approach with the following 3 reasons. First, this sampling frame reduces contamination of the sample from acute kidney injury patients on dialysis who had been inadvertently entered into ANZDATA. Second, this sampling frame allows a reasonable time for patients to initiate training and transition onto home dialysis (~62% of those in ANZ who perform PD do so for the first time by 90 days, and about ~40% of corresponding home HD patients). Last, it is unlikely that modality has an impact upon early (ie, <90 days) mortality in incident dialysis patients; studies of early mortality identified other modifiable factors of more relevance to this often older and highly comorbid patient group.

The primary exposure of dialysis modality was defined to 2 ways. First, it was modeled as time varying, referring to a patient’s time-updated treatment modality over the entire study period. This is consistent with an as-treated framework (“did the exposure that the patient actually received affect mortality?”). Second, it was modeled as fixed from 90 days, referring to a patient’s initial treatment modality at baseline. This is consistent with an intention-to-treat approach (“did exposure that the patient initially...
received affect mortality, irrespective of subsequent changes that occurred along the way”). These are referred to as the “as-treated” (AT) and “intention-to-treat” (ITT) approaches, respectively.

The primary outcome was patient death.

We stratified analyses by era, defined by year of dialysis inception. In the main analysis, era was arbitrarily defined in 5-year windows (ie, 1998-2002, 2003-2007, 2008-2012, 2013-2017). Two other strategies were used as sensitivity analyses to address the well-known time-varying relative risk of mortality between PD and facility HD. One strategy used era defined in 7-year windows (1997-2003, 2004-2010, 2011-2017), and another used 10-year windows (1998-2007, 2008-2017). In all these different analyses, the hazard (or subhazard) ratio at time $T$ largely ignores the distribution of events before (and after) time $T$. The 5-year window can therefore be regarded as the best-case scenario for PD, with the mortality risk dominantly reflecting the early survival benefit for PD. By contrast, the 10-year window can be regarded as the worst-case scenario dominantly reflecting the late benefit for facility HD.

We adjusted estimates of the effect of modality on mortality using available patient-related risk factors: age, sex, ethnicity, primary kidney disease, estimated glomerular filtration rate (eGFR) at dialysis inception, late referral for nephrology predialysis care (<3 months before dialysis inception), diabetes mellitus (none, type 1, type 2), body mass index (BMI), medical comorbidity (coronary artery disease, peripheral vascular disease, cerebrovascular disease, chronic lung disease), and smoking.

Two modeling approaches were used. First, we used cause-specific proportional hazards models, censoring for kidney transplantation, return of kidney function, and loss to follow-up. We included a gamma-distributed shared frailty using the center of initial dialysis treatment as the random effect. Second, we used subdistribution proportional hazards (Fine and Gray) models, treating

Table 2. Clinical Characteristics of the Study Cohort by Intention-to-Treat (90-Day) Modality

| Variable                        | Facility HD | CAPD  | APD   | Home HD | $P^*$ |
|---------------------------------|------------|-------|-------|---------|-------|
| No. of patients                 | 29,548     | 11,136| 5,374 | 1,236   |       |
| Age, y                          | 63 [51-72] | 63 [51-71] | 59 [46-69] | 50 [42-59] | <0.001 (0.02) |
| Male sex                        | 18,142 (61.4%) | 6,274 (56.3%) | 3,336 (62.1%) | 931 (75.3%) | <0.001 (0.06) |
| Ethnicity                       |            |       |       |         | <0.001 (0.17) |
| ANZ European                    | 20,660 (69.9%) | 7,146 (64.2%) | 3,747 (69.7%) | 935 (75.7%) |       |
| Aboriginal/TSI                  | 3,034 (10.3%) | 612 (5.5%) | 265 (4.9%) | 29 (3.4%) |       |
| Asian                           | 1,942 (6.6%) | 1,310 (11.8%) | 749 (13.9%) | 89 (7.2%) |       |
| NZ Maori                        | 1,628 (5.5%) | 1,147 (10.3%) | 221 (4.1%) | 73 (5.9%) |       |
| Pacific People                  | 1,557 (5.3%) | 679 (6.1%) | 158 (2.9%) | 72 (5.8%) |       |
| Other                           | 727 (2.5%) | 242 (2.2%) | 234 (4.4%) | 38 (3.1%) |       |
| Late referral                   | 7,143 (24.2%) | 2,117 (19.0%) | 861 (16.0%) | 61 (4.9%) | <0.001 (0.10) |
| eGFR, mL/min/1.73 m²            | 6.5 [4.7-9.2] | 6.8 [4.9-9.6] | 7.3 [5.4-10.2] | 6.1 [4.6-8.1] | <0.001 (<0.01) |
| Smoking                         | 4,077 (13.8%) | 1,503 (3.5%) | 613 (11.4%) | 145 (11.7%) | <0.001 (0.02) |
| Diabetes mellitus               |            |       |       |         | <0.001 (0.09) |
| Type 1                          | 1,052 (3.6%) | 488 (4.4%) | 346 (6.4%) | 46 (3.7%) |       |
| Type 2                          | 13,374 (45.3%) | 4,631 (41.6%) | 1,936 (36.0%) | 318 (25.7%) |       |
| Primary kidney disease          |            |       |       |         | <0.001 (0.12) |
| Diabetic nephropathy            | 11,101 (37.6%) | 4,125 (37.0%) | 1,793 (33.4%) | 271 (21.9%) |       |
| Glomerulonephritis              | 6,258 (21.2%) | 2,609 (23.4%) | 1,418 (26.4%) | 435 (35.2%) |       |
| Hypertension/ischemic           | 4,122 (14.0%) | 1,608 (14.4%) | 736 (13.7%) | 84 (6.8%) |       |
| Polycystic kidney disease       | 1,613 (5.5%) | 570 (5.1%) | 349 (6.5%) | 225 (18.2%) |       |
| Reflux nephropathy              | 644 (2.2%) | 323 (2.9%) | 174 (3.2%) | 59 (4.8%) |       |
| Other                           | 5,810 (19.7%) | 1,901 (17.1%) | 904 (16.8%) | 162 (13.1%) |       |
| Comorbidity at baseline         |            |       |       |         |       |
| Coronary artery                 | 12,262 (41.5%) | 4,286 (38.5%) | 1,697 (31.6%) | 229 (18.5%) | <0.001 (0.10) |
| Peripheral vascular             | 7,633 (25.8%) | 2,779 (25.0%) | 1,037 (19.3%) | 125 (10.1%) | <0.001 (0.08) |
| Cerebrovascular                 | 4,403 (14.9%) | 1,633 (14.7%) | 653 (12.2%) | 62 (5.0%) | <0.001 (0.05) |
| Lung                            | 5,196 (17.6%) | 1,881 (15.1%) | 706 (13.1%) | 109 (8.8%) | <0.001 (0.06) |
| BMI, kg/m²                      | 27.5 [23.6-32.5] | 26.5 [23.2-30.4] | 26.5 [23.1-30.2] | 28.4 [24.6-34.4] | <0.001 (0.02) |
| BMI ≥ 30 kg/m²                  | 10,505 (35.6%) | 3,001 (27.0%) | 1,405 (26.1%) | 514 (41.6%) | <0.001 (0.10) |

Continuous variables are shown as median [interquartile range]; categorical variables are shown as number (percentage). These descriptions are derived from the risk set for analyses of initial modality. Corresponding descriptions at the time of modality inception from the risk set for analyses of as-treated modality are provided in Table S2.

Abbreviations: ANZ, Australian and New Zealand; APD, automated peritoneal dialysis; BMI, body mass index; CAPD, continuous ambulatory peritoneal dialysis; eGFR, estimated glomerular filtration rate; HD, hemodialysis; TSI, Torres Strait Islander.

*Effect size in parentheses. For effect size calculations, see Item S1. Effect size statistics describe the likely magnitude of differences; a value of 0.2 might be considered a small effect size, 0.5 a medium one, and 0.8 and greater a large effect size.
transplantation as a competing risk. For all comparative analyses, facility HD was the reference category.

Twelve strategies were used for computations, as illustrated in Figure S1. These strategies all have different combinations of how modality was defined (AT vs ITT), what follow-up windows were used (5 years vs. 7 years vs. 10 years), and which type of models were implemented (cause-specific vs subdistribution). In the main analysis, we used AT dialysis modality with a 5-year follow-up window and a cause-specific proportional hazards model owing to our hypothesis comparing etiological risks.37-40 Of note, medical comorbidity was modeled as time-varying when the modality was similarly defined (AT), and modeled at 90 days when the modality was defined at baseline (ITT).

We used 3-way interaction terms in the main-effects model in a dataset containing patients from all eras, exploring potential effect modification by age, sex, diabetes mellitus, and presence of any comorbidity, based on plausibility and previous findings from our group and others.41-47

### Results

#### Cohort Description

The inception cohort for the main analysis contained 53,662 adult patients with 95,942 patient-years of follow-up. There were 52,097 people with 93,947 patient-years with 125,822 discrete periods of modality treatment without missing data: Table S1 summarizes the study

---

**Table 3.** Key Clinical Characteristics of the Study Cohort by Intention-to-Treat (90-Day) Modality and Era

| Facility HD | 1998-2002 | 2003-2007 | 2008-2012 | 2013-2017 | P* |
|-------------|----------|----------|----------|----------|----|
| No. of patients | 5,377 | 7,323 | 8,456 | 8,392 | 0.001 (<0.01) |
| Age, y | 60 [48-71] | 63 [51-73] | 63 [52-73] | 63 [52-73] | 0.001 (<0.01) |
| Diabetes mellitus | 1,927 (35.8%) | 3,329 (45.5%) | 4,380 (51.8%) | 4,790 (57.1%) | 0.001 (0.15) |
| Comorbidity at baseline | 2,117 (39.4%) | 3,167 (43.3%) | 3,765 (44.5%) | 3,213 (38.3%) | 0.001 (0.05) |
| Coronary artery | 1,429 (26.6%) | 1,900 (26.0%) | 2,387 (28.2%) | 1,917 (22.8%) | 0.001 (0.05) |
| Cerebrovascular | 790 (14.7%) | 1,149 (15.7%) | 1,357 (16.1%) | 1,107 (13.2%) | 0.001 (0.03) |
| Lung | 837 (15.6%) | 1,274 (17.4%) | 1,621 (19.2%) | 1,464 (17.5%) | 0.001 (0.03) |

**CAPD**

| No. of patients | 3,422 | 2,885 | 2,461 | 2,368 |
| Age, y | 62 [50-70] | 63 [52-72] | 63 [52-72] | 63 [52-71] |
| Diabetes mellitus | 1,380 (40.3%) | 1,263 (43.8%) | 1,220 (49.6%) | 1,256 (53.0%) |
| Comorbidity at baseline | 1,439 (42.1%) | 1,112 (38.5%) | 951 (38.6%) | 784 (33.1%) |
| Peripheral vascular | 1,007 (29.4%) | 693 (24.0%) | 599 (24.3%) | 480 (20.3%) |
| Cerebrovascular | 550 (16.1%) | 414 (14.4%) | 382 (15.5%) | 287 (12.1%) |
| Lung | 504 (14.7%) | 445 (15.4%) | 413 (16.8%) | 319 (13.5%) |

**APD**

| No. of patients | 432 | 1,097 | 1,555 | 2,290 |
| Age, y | 60 [48-70.5] | 59 [45-70] | 59 [47-70] | 58 [46-69] |
| Diabetes mellitus | 171 (39.6%) | 446 (40.7%) | 621 (39.9%) | 1,044 (45.6%) |
| Comorbidity at baseline | 177 (41.0%) | 393 (35.8%) | 497 (32.0%) | 630 (27.5%) |
| Peripheral vascular | 139 (32.2%) | 239 (21.8%) | 294 (18.9%) | 365 (15.9%) |
| Cerebrovascular | 75 (17.4%) | 157 (14.3%) | 194 (12.5%) | 227 (9.9%) |
| Lung | 64 (14.8%) | 159 (14.5%) | 226 (14.5%) | 257 (11.2%) |

**Home HD**

| No. of patients | 275 | 229 | 299 | 433 |
| Age, y | 47 [38-54] | 48 [41-56] | 52 [44-59] | 53 [45-61] |
| Diabetes mellitus | 40 (14.6%) | 39 (17.0%) | 98 (32.8%) | 187 (43.2%) |
| Comorbidity at baseline | 43 (15.6%) | 38 (16.6%) | 57 (19.1%) | 91 (21.0%) |
| Peripheral vascular | 25 (9.1%) | 20 (8.7%) | 27 (9.0%) | 53 (12.2%) |
| Cerebrovascular | 21 (7.6%) | 3 (1.3%) | 15 (5.0%) | 23 (5.3%) |
| Lung | 17 (6.2%) | 15 (6.6%) | 27 (9.0%) | 50 (11.6%) |

Continuous variables are shown as median [interquartile range]; categorical variables are shown as number (percentage). These descriptions are derived from the risk set for analyses of initial modality. Corresponding descriptions at the time of modality inception from the risk set for analyses of as-treated modality are provided in Table S3. Abbreviations: APD, automated peritoneal dialysis; CAPD, continuous ambulatory peritoneal dialysis; HD, hemodialysis. *Effect size in parentheses. For effect size calculations, see Item S1. Effect size statistics describe the likely magnitude of differences; a value of 0.2 might be considered a small effect size, 0.5 a medium one, and 0.8 and greater a large effect size.
cohort and excluded cohort due to missing data at dialysis inception. The excluded cohort was a small proportion of the potential study cohort, with very small differences when compared with the study cohort.

Descriptive Data by Modality and Era

Table 1 summarizes the clinical characteristics of the study cohort by era of dialysis inception from the main analysis. Over the years, the key changes over time are (1) increases in type 2 diabetes mellitus and diabetic nephropathy, (2) decreases in recorded cardiovascular comorbidity at dialysis inception, (3) decreases in “late referrals” for dialysis, and (4) increases in overweight and obese patients.

Table 2 summarizes the clinical characteristics of the study cohort by ITT modality (ie, baseline modality at 90 days). The largest difference in modifiable risk factors between modalities can be found in the proportion of patients who are late referrals for dialysis, which is lowest with home HD, greatest with facility HD, and intermediate with PD. In addition, patients initially treated with home HD were younger compared with those treated with facility HD and PD, more likely to have kidney failure secondary to single-organ disease (eg, glomerulonephritis) rather than systemic disease, more likely to have a higher BMI, and less likely to have diabetes mellitus or medical comorbidity. Patients on CAPD and APD were generally similar to each other, and together were similar to those treated with facility HD. Table S2 replicates Table 2 but according to AT (ie, time-varying) modality, meaning that patients might be classified in multiple categories depending on the modalities that they were exposed to during the period of observation. The findings are generally similar to Table 2.

Table 3 summarizes key clinical characteristics of the study cohort at 90 days by by ITT modality (ie, baseline modality at 90 days) for each era. The following generalizations can be made. Over the years, there has been a small increase in age in the patients with an initial modality of facility HD or CAPD, no change in age in those with an initial modality of APD, but a comparatively large increase in age in those starting home HD. In all modalities, there has been an increase in the proportion of patients with diabetes mellitus, although this increase is approximately twice as large for home HD as it is for the other modalities. For facility HD, CAPD, and APD, there has been no change or a decrease in the proportion of patients with comorbidity. By contrast, for home HD there has been a directional increase in most types of comorbidity. Table S3 replicates Table 3 but according to AT (ie, time-varying) modality. The findings are generally similar to Table 3.

Deaths and Censoring Events

The unadjusted mortality rates for the cohort overall and by modality are illustrated from the main analysis in Figure 1. There has been an improvement in crude death rates in the cohort from ~15 deaths per 100 patient-years in 1998-2002 to ~11 in 2013-2017, although this improvement is limited to those treated with facility HD and CAPD and APD. The number of deaths (and causes of death) are provided in Table S4 for 5-year, 7-year, and 10-year follow-up windows.

Overall, CVD was the largest attributed cause of death across all eras, followed by withdrawal from dialysis, and then infectious mortality. Over the years, it is notable that there has been a slight decrease in cardiovascular mortality as a cause of death, but a proportionately larger decrease in infectious mortality. In terms of cause of death by modality, cardiovascular mortality accounts for a greater proportion of deaths on home HD compared with other modalities, infectious mortality a greater proportion of deaths on PD, and patient withdrawal a greater proportion on facility HD.

The number of censoring events (and causes of events) are also provided in Table S5 for the 5-year, 7-year, and 10-year follow-up windows. As expected, the transplantation rate was highest for those on home HD and was stable across era. It has been lowest for facility HD throughout, with a small decrease across era. It has been stable for CAPD, but rapidly rising for those on APD.

Main Results

Unadjusted nonparametric estimates of survival by era and modality from the main analysis are illustrated in
Figure S2. Qualitatively, there is generally a lower risk of death in more recent eras, with the exception of home HD where the risk of death in the most recent era is similar to that in previous ones. Quantitatively, as expected, there is significant modification of the effect of modality on mortality risk by era for the individually specified era terms ($P = 0.008$) and for the model overall ($P < 0.001$). Separate estimates were therefore computed for each era. There is subjectively good fit between the modeled and observed data for survival in these models for the main analysis (Fig S3), and no violation of the proportional hazards assumption for era ($P$ values all $\sim0.2$) (Fig S4). As expected, there was time dependency for modality ($P < 0.001$ for CAPD and APD) (Fig S5), justifying analyses with different follow-up time windows.

The fully adjusted models from the main analysis (ie, for eras defined by 1998-2002, 2003-2007, 2008-2012, 2013-2017) are presented in Figure S6. For comprehensibility, a summary illustration is presented in Figure 2, showing the adjusted effects of modality on mortality risk, by era. The figure also shows corresponding effects from the other supplementary analyses that use cause-specific proportional hazards models with shared frailty but with different follow-up time windows.

The numerical values for estimates and 95% confidence intervals from all the illustrated effects are provided in Table S6. Overall, all models demonstrate consistent results: the mortality rate of facility HD, which was previously observed to be lower than CAPD and APD is now higher than CAPD and APD, and the mortality rate with home HD continues to be lower than all other modalities and reasonably stable. From the main analysis, mortality with CAPD and APD relative to facility HD have adjusted hazard ratios in 2013-2017 of 0.88 (95% CI, 0.78-0.99) and 0.91 (95% CI, 0.82-1.00), respectively. The corresponding estimate for home HD is 0.50 (95% CI, 0.40-0.64).
Other Results

There were no 3-way interactions between dialysis modality, era, and diabetes mellitus or comorbidity ($P = 0.5$, and 0.9, respectively) in a model derived from the main analysis but containing patients from all eras. There was a borderline interaction with sex ($P = 0.06$) and a weak one with age ($P = 0.03$). This effect modification is illustrated in Figure 4, which shows the fully adjusted effects of modality for 3 subgroups of age (18-54, 55-64, and ≥65 years), and for female and male patients. Broadly, the trends over era in each age group are directionally similar to the main analysis although more marked for older patients. The same applies for both female and male patients, although the trend is more marked for male patients.

Discussion

There are 3 major insights from this observational study. First, we show that PD is associated with a lower adjusted mortality risk than facility HD in the contemporary ANZ population, contrary to our previous studies that demonstrated the opposite. These findings are directionally similar to studies from Taiwan, the United States, Canada, Europe, and South Korea. Recent systematic reviews of mortality risk by modality report similar survival for HD and PD, but synthesize mainly older data without formal testing for effect modification by era.

The reasons for the improved mortality risk with PD are not clear. One contribution could be from the marked decrease in PD peritonitis that has occurred within ANZ over the last decade (Fig S7). PD peritonitis will result directly in death in 2% to 5% of cases, and indirectly to several-fold more in the period immediately following apparent full recovery. Another consideration is increased selection bias over the years, with an increasing propensity for lower-risk patients to receive PD rather than HD. This situation is in contrast with other health jurisdictions that have reported corresponding secular trends in which it is apparent that increasingly higher risk patients are being treated with PD. In our study, patients on PD in 2013-2017 had a lower prevalence of diabetes mellitus when compared with facility HD, whereas for those in previous times it was comparable or even higher (Table 3). Mortality estimates in our models already include adjustments for this difference, as well as any other differences between groups arising for the variables in Table 2. The estimates are not adjusted for unmeasured variables, however, and it is likely that socioeconomic, medication-related, and health services factors are also more
favorable to PD compared with facility HD. The observed secular improvement in mortality risk with PD might therefore reflect this residual confounding.

A final consideration for the differential improvement of mortality risk with PD is the increased icodextrin uptake in ANZ, which has increased from 0 to ~50% over the period of observation (Fig S8). This intervention probably decreases mortality risk in clinical trials.59-62

Although differences in survival according to subgroups were not among our a priori objectives, in the exploratory analyses we noted that the mortality rates of elderly and diabetic patients on PD were similar to those of HD patients. Once again, this finding must be regarded in the light of possible selection bias from imbalance of unmeasured confounders as described previously. Notwithstanding, our findings suggest that careful consideration of the role of PD in the elderly and those with diabetes mellitus may be needed and reinforce the paradigm of “patient-centered” shared modality decision making for all patients.

Finally, there has been a marked change in home HD epidemiology, with an increasing number of patients with diabetes and medical comorbidity using this modality. We cannot definitively pinpoint the reason for this expanded use of home HD, but it is known that this increase coincides with the wider adoption of “intensive hemodialysis” in ANZ (Figs S9 and S10). It is likely that this submodality of home HD is regarded by many practitioners in ANZ as being safe or even appropriate for patients at higher clinical risk.63 Despite this secular change in demographics and comorbidity, the adjusted mortality risk with home HD has not changed relative to facility HD. We cannot identify the exact reason for the stable mortality rates in the home HD population, but it is possible that the potential benefits of intensive HD may have contributed. Although this submodality has not been shown definitively to improve the mortality risk in clinical trials, it does improve left ventricular structure and function. In a previous analysis, we showed intensive dialysis in the home setting to be associated with lower mortality risk in ANZ.29

Our study is limited by the usual foibles of observational studies, such as ascertainment error in the recording of data, the potential for model misspecification, and residual confounding as noted previously. In addition, we assumed that patients did not change their dialysis center during treatment, although almost 9% of patients do change centers after day 90 of dialysis.
Finally, the clinical and organizational culture of dialysis delivery in ANZ is distinctive, wherein there is strong emphasis across all stakeholder groups on the primacy of home dialysis. As such, health care systems are well resourced for home dialysis, with health care workers who are well versed and confident with the modalities. In addition, there are evident practices in this study that are not customary elsewhere, such as the increasing trend to treat lower risk patients with PD rather than HD. The results observed in ANZ may therefore not be entirely generalizable elsewhere, although we note the directional similarities between our findings and those from other health jurisdictions.

In summary, we have identified that the survival for patients on PD now appears to be better than the survival for patients on facility HD. The relevance of this important finding will vary by health jurisdiction, and health care workers and funding agencies should be assessing the role of home dialysis in their own contemporary settings.

Supplementary Material

Supplementary File (PDF)

Figure S1: Analytical strategies.

Figure S2: Unadjusted nonparametric estimates of survival from the main analysis, by modality and era.

Figure S3: Kaplan-Meier observed survival curves by modality and era for the main analysis, compared with those predicted by cause-specific proportional hazards models with shared frailty.

Figure S4: Scaled Schoenfeld residuals plots from the main analysis showing no time dependency of effects for era.

Figure S5: Scaled Schoenfeld residuals plots from the main analysis showing time dependency of effects for CAPD and APD, justifying the sensitivity analyses with different follow-up time windows.

Figure S6: HRs for mortality by modality from the main analysis in each of the 4 eras, fully adjusted for the confounders listed in Table 2, with shared frailty.

Figure S7: Peritonitis rates from Australian episodes collected in ANZDATA and from NZ episodes collected in the NZ PD registry.

Figure S8: Icodextrin point prevalence amongst PD patients at end of each year in ANZDATA.

Figure S9: Median frequency of dialysis in home HD patients at end of each year in the study cohort.

Figure S10: Median treatment session length of dialysis in home HD patients at end of each year in the study cohort.

Item S1: Detailed methods.

Table S1: Clinical characteristics of the inception cohort.

Table S2: Clinical characteristics of the study cohort at the time of modality inception, by as treated modality.

Table S3: Key clinical characteristics of the study cohort at the inception of each episode of modality, by modality and era.

Table S4: Number and causes of death in analyses, by follow-up window, era, and modality.

Table S5: Number and causes of censoring events in analyses, by follow-up window, era, and modality.

Table S6: Numerical estimates from the main and supplementary models as defined in Figure S1 and illustrated in Figures 2-4.

Article Information

Authors' Full Names and Academic Degrees: Mark R. Marshall, MBChB, MPH(Hons), FRACP, Kevan R. Polkinghorne, BHB, MBChB, MclinEpi, PhD, FRACP, Neil Boudville, MB, BS, MMedSci (Clin Epi), DMed, FRACP, and Stephen P. McDonald, MB, BS(Hons), PhD, FRACP.

Authors’ Affiliations: Faculty of Medical and Health Sciences, University of Auckland, and Department of Renal Medicine, Counties Manukau Health, Auckland, New Zealand (MRM); Department of Nephrology, Monash Health, and Department of Medicine, Department of Epidemiology and Preventive Medicine, and Department of Nursing and Health Sciences, Monash University, Clayton, Australia (KRP); Australia and New Zealand Dialysis and Transplant Registry (ANZDATA), South Australia Health and Medical Research Institute, Adelaide, Australia (KRP, NB, SPM); Medical School, University of Western Australia, Nedlands, Australia (NB); Department of Renal Medicine, Sir Charles Gairdner Hospital, Nedlands, Australia (NB); School of Medicine, University of Adelaide, Adelaide, Australia (SPM).

Address for Correspondence: Mark R. Marshall, MBChB, MPH(Hons), FRACP, PO Box 29, Helensville 0840, New Zealand. Email: markrogermarshall@icloud.com

Authors’ Contributions: Research idea and study design: MRM; data acquisition: MRM, SPM; data analysis/interpretation: MRM, NB, KRP, SPM; statistical analysis: MRM, KRP, SPM, NB. Each author contributed important intellectual content during manuscript drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

Support: This work was supported in part by the Jacquot Family and unrestricted educational grants made through the Research Foundation of Royal Australasian College of Physicians. ANZDATA receives funding from the Australian Government Department of Health and Ageing, the New Zealand Ministry of Health, and Kidney Health Australia. General support for registry activities has been received from AMGEN Australia Pty Ltd, Novartis Pharmaceuticals Australia Pty Ltd, Janssen-Cilag Pty Ltd, Fresenius Medical Care-Australia Pty Ltd, Roche Products (Australia) Ltd, and Wyeth Australia Pty Ltd. The funders had no role in the study design, data collection, analysis, reporting, or decision to submit the manuscript for publication.

Financial Disclosure: Dr Marshall has received honoraria as an advisor to Abbott Australia Pty Ltd and travel grants from Roche Products NZ Ltd, Novartis NZ Ltd, and Fresenius Medical Care—Asia-Pacific Pty Ltd. Within the last 2 years, he was previously employed by Baxter Healthcare (Asia-Pacific) Ltd. Dr Polkinghorne has received speaking honoraria and travel grants from AMGEN Australia. Dr Boudville has received speaking honoraria and travel and research grants from Fresenius Medical Care—Australia Pty Ltd and Baxter Healthcare. Dr McDonald has received speaking honoraria from AMGEN Australia, Fresenius Medical Care—Australia Pty Ltd, Baxter Healthcare, and Solvay Pharmaceuticals and travel grants from AMGEN Australia, Genzyme Australia, and Jansen-Cilag Pty Ltd.

Acknowledgements: ANZDATA exists because of the tireless work of the nephrology community throughout Australia and New Zealand in collecting the information.

Peer Review: Received July 23, 2020. Evaluated by 2 external peer reviewers, with direct editorial input from a Statistics/Methods Editor, an Associate Editor, and the Editor-in-Chief. Accepted in revised form March 3, 2021.
References

1. Tong A, Manns B, Hemmelgarn B, et al. Establishing core outcome domains in hemodialysis: report of the Standardized Outcomes in Nephrology-Hemodialysis (SONG-HD) Consensus Workshop. *Am J Kidney Dis*. 2017;69(1):97-107.

2. Manera KE, Johnson DW, Craig JC, et al. Establishing a core outcome set for peritoneal dialysis: report of the SONG-PD (Standardized Outcomes in Nephrology-Peritoneal Dialysis) Consensus Workshop. *Am J Kidney Dis*. 2020;75(3):404-412.

3. Sautenet B, Tong A, Manera KE, et al. Developing consensus-based priority outcome domains for trials in kidney transplantation: a multinational Delphi survey with patients, caregivers, and health professionals. *Transplantation*. 2017;101(8):1875-1886.

4. Ross S, Dong E, Gordon M, et al. Meta-analysis of outcome studies in end-stage renal disease. *Kidney Int*. 2000;57:S28-S38.

5. Ishani A, Slinin Y, Greer N, et al. Comparative Effectiveness of Home-Based Kidney Dialysis Versus In-Center or Other Outpatient Kidney Dialysis Locations: A Systematic Review. Department of Veterans Affairs; 2015.

6. Dialysis Modalities for the Treatment of End-Stage Kidney Disease: A Review. CADTH Optimal Use Report, vol 6, no. 2a. Canadian Agency for Drugs and Technologies in Health; 2011.

7. Dialysis Modalities for the Treatment of End-Stage Kidney Disease: Recommendations, CADTH Optimal Use Reports. Canadian Agency for Drugs and Technologies in Health; 2017.

8. Pike E, Hamidi V, Ringerike T, et al. Health Technology Assessment of the Different Dialysis Modalities in Norway, NIPH Systematic Reviews. Knowledge Centre for the Health Services at the Norwegian Institute of Public Health (NIPH); 2013.

9. Pike E, Hamidi V, Ringerike T, Wsloff T, Klemt M. More use of peritoneal dialysis gives significant savings: a systematic review and health economic decision model. *J Clin Med Res*. 2017;9(2):104-116.

10. Mehrrota R, Devuyst O, Davies SJ, Johnson DW. The current state of peritoneal dialysis. *J Am Soc Nephrol*. 2016;27(11):3238-3252.

11. Boateng EA, East L. The impact of dialysis modality on quality of life: a systematic review. *J Ren Care*. 2011;37(4):190-200.

12. Ho YF, Li IC. The influence of different dialysis modalities on the quality of life of patients with end-stage renal disease: a systematic literature review. *Psychol Health*. 2016;31(12):1435-1465.

13. Liem YS, Bosch JL, Arends LR, Heijenbrok-Kal MH, Hunink MG. Quality of life assessed with the Medical Outcomes Study Short Form 36-Item Health Survey of patients on renal replacement therapy: a systematic review and meta-analysis. *Value Health*. 2007;10(5):390-397.

14. Liem YS, Bosch JL, Hunink MG. Preference-based quality of life of patients on renal replacement therapy: a systematic review and meta-analysis. *Value Health*. 2008;11(4):733-741.

15. Purnell TS, Auguste P, Crews DC, et al. Comparison of life participation activities among adults treated by hemodialysis, peritoneal dialysis, and kidney transplantation: a systematic review. *Am J Kidney Dis*. 2013;62(5):953-973.

16. Wyld M, Morton RL, Hayen A, Howard K, Webster AC. A systematic review and meta-analysis of utility-based quality of life in chronic kidney disease treatments. *PLoS Med*. 2012;9(9):e1001307.

17. Zazzeroni L, Pasquinelli G, Nanni E, Cremonini V, Rubbi I. Comparison of quality of life in patients undergoing hemodialysis and peritoneal dialysis: a systematic review and meta-analysis. *Kidney Blood Press Res*. 2017;42(4):717-727.

18. Lee MB, Bargman JM. Survival by dialysis modality—who cares? *Clin J Am Soc Nephrol*. 2016;11(6):1083-1087.

19. McDonald SP, Marshall MR, Johnson DW, Polkinghorne KR. Relationship between dialysis modality and mortality. *J Am Soc Nephrol*. 2009;20(1):155-163.

20. Kasja J, Polkinghorne KR, Marshall MR, McDonald SP, Wolfe R. Clustering and residual confounding in the application of marginal structural models: dialysis modality, vascular access, and mortality. *Am J Epidemiol*. 2015;182(6):535-543.

21. Kasja J, Wolfe R, McDonald SP, Marshall MR, Polkinghorne KR. Dialysis modality, vascular access and mortality in end-stage kidney disease: a bi-national registry-based cohort study. *Nephrology (Carlton)*. 2016;21(10):878-886.

22. Marshall MR, Hawley CM, Kerr PG, et al. Home hemodialysis and mortality risk in Australian and New Zealand populations. *Am J Kidney Dis*. 2011;58(5):782-793.

23. Marshall MR, Polkinghorne KR, Kerr PG, Agar JW, Hawley CM, McDonald SP. Temporal changes in mortality risk by dialysis modality in the Australian and New Zealand dialysis population. *Am J Kidney Dis*. 2015;66(3):489-498.

24. Marshall MR, Polkinghorne KR, McDonald SP. Temporal changes in mortality risk by sub-modalities of peritoneal dialysis. *Nephrology*. 2016;21(suppl 2). 123 [abstract].

25. Marshall MR, van der Schrieck N, Lilley D, et al. Independent community house hemodialysis as a novel dialysis setting: an observational cohort study. *Am J Kidney Dis*. 2013;61(4):598-607.

26. Marley JV, Dent HK, Wearne M, et al. Haemodialysis outcomes of Aboriginal and Torres Strait Islander patients of remote Kimberley region origin. *Med J Aust*. 2010;193(9):516-520.

27. Villarba A, Warr K. Home haemodialysis in remote Australia. *Nephrology (Carlton)*. 2004;9(suppl 4):S134-S137.

28. Kneipp E, Murray R, Warr K, Fitzclarence C, Wearne M, Maguire G. Bring me home: renal dialysis in the Kimberley. *Nephrology (Carlton)*. 2004;9(suppl 4):S121-S125.

29. Marshall MR, Polkinghorne KR, Kerr PG, Hawley CM, Agar JW, McDonald SP. Intensive hemodialysis and mortality risk in Australian and New Zealand populations. *Am J Kidney Dis*. 2016;67(4):617-628.

30. McQuillan R, Trpeski L, Fenton S, Lok CE. Modifiable risk factors for early mortality on hemodialysis. *Int J Nephrol*. 2012;2012:435736-435736.

31. Bradbury BD, Fissell RB, Albert JM, et al. Predictors of early mortality among incident US hemodialysis patients in the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Clin J Am Soc Nephrol*. 2007;2(1):89-99.

32. Hole B, Casula A, Caskey F. Outcomes in the first 90 days after starting dialysis—transition of the UK Renal Registry to capture all acute and chronic dialysis sessions. *UK Kidney Week*. Deposited March 21, 2019. https://research-information.bris.ac.uk/ws/portal/files/212191786/Survival_in_the_three_months_after_first_dialysis_V3.pdf

33. Vonesh E, Schauble D, Hao W, Collins A. Statistical methods for comparing mortality among ESRD patients: Examples of regional/international variations. *Kidney Int*. 2000;57(suppl 74):S19-S27.
44. Mehrotra R, Chiu YW, Kalantar-Zadeh K, Bargman J, Vonesh E.
45. Liem YS, Wong JB, Hunink MG, de Charro FT,
46. Marshall MR. The benefit of early survival on PD versus HD—why this is (still) very important. Perit Dial Int. 2020;40(4):405-418.
47. Austin PC, Lee DS, Fine JP. Introduction to the analysis of survival data in the presence of competing risks. Circulation. 2016;133(6):601-609.
48. Li L, Yang W, Astor BC, Greene T. Competing risk modeling: time to put it in our standard analytical toolbox. J Am Soc Nephrol. 2019;30(12):2284-2286.
49. Allison P. For causal analysis of competing risks, don’t use Fine & Gray’s subdistribution method. Statistical Horizons, March 24, 2018. Accessed May 20, 2021. https://statisticalhorizons.com//for-causal-analysis-of-competing-risks
50. Noordzij M, Leffondré K, van Stralen KJ, Zoccali C, Dekker FW, Jager KJ. When do we need competing risks methods in nephrology? Nephrol Dial Transplant. 2013;28(11):2670-2677.
51. Couchoud C, Bolignano D, Nistor I, et al. Dialysis modality choice in diabetic patients with end-stage kidney disease: a systematic review of the available evidence. Nephrol Dial Transplant. 2015;30(2):310-320.
52. Huang CC, Cheng KF, Wu HD. Survival analysis: comparing peritoneal dialysis and hemodialysis in Taiwan. Perit Dial Int. 2008;28(suppl 3):S15-S20.
53. Kim H, Kim KH, Park K, et al. A population-based approach indicates an overall higher patient mortality with peritoneal dialysis compared to hemodialysis in Korea. Kidney Int. 2014;86(5):991-1000.
54. Liem YS, Wong JB, Hunink MG, de Charro FT, Winkelmaier WC. Comparison of hemodialysis and peritoneal dialysis survival in the Netherlands. Kidney Int. 2007;71(2):153-158.
55. Mehrrota R, Chiu YW, Kalantar-Zadeh K, Bargman J, Vonesh E. Similar outcomes with hemodialysis and peritoneal dialysis in patients with end-stage renal disease. Arch Intern Med. 2011;171(2):110-118.
56. Schaubel DE, Morrison HI, Fenton SS. Comparing mortality rates on CAPD/CCPD and hemodialysis. The Canadian experience: fact or fiction? Perit Dial Int. 1998;18(5):478-484.
57. Vonesh EF, Snyder JJ, Foley RN, Collins AJ. The differential impact of risk factors on mortality in hemodialysis and peritoneal dialysis. Kidney Int. 2004;66(6):2389-2401.
58. Chang YK, Hsu CC, Hwang SJ, et al. A comparative assessment of survival between propensity score-matched patients with peritoneal dialysis and hemodialysis in Taiwan. Medicine. 2012;91(3):144-151.
59. Saran R, Robinson B, Abbott KC, et al. US Renal Data System 2018 annual data report: epidemiology of kidney disease in the United States. Am J Kidney Dis. 2019;73(3):Svii-Sxii, S1-S772.
60. Perl J, Wald R, McFarlane P, et al. Hemodialysis vascular access modifies the association between dialysis modality and survival. J Am Soc Nephrol. 2011;22(6):1113-1121.
61. Yeates K, Zhu N, Vonesh E, Trpeski L, Blake P, Fenton S. Hemodialysis and peritoneal dialysis are associated with similar outcomes for end-stage renal disease treatment in Canada. Nephrol Dial Transplant. 2012;27(9):3568-3575.
62. Head JG, Wehberg S. Relative survival of peritoneal dialysis and haemodialysis patients: effect of cohort and mode of dialysis initiation. PloS One. 2014;9(3):e90119.
63. Van de Luijgaard WM, Jager KJ, Segelmark M, et al. Trends in dialysis modality choice and related patient survival in the ERA-EDTA Registry over a 20-year period. Nephrol Dial Transplant. 2015;31(1):120-128.
64. Ryu J-H, Kim H, Kim KH, et al. Improving survival rate of Korean patients initiating dialysis. Yonsei Med J. 2015;56(3):666-675.
65. Lee SW, Lee NR, Son SK, et al. Comparative study of peritoneal dialysis versus hemodialysis on the clinical outcomes in Korea: a population-based approach. Sci Rep. 2019;9(1). 5905.
66. Li PK, Szeto CC, Piraino B, et al. ISPD peritonitis recommendations: 2016 update on prevention and treatment. Perit Dial Int. 2016;36(5):481-508.
67. Fried LF, Bernardini J, Johnston JR, Piraino B. Peritonitis influences mortality in peritoneal dialysis patients. J Am Soc Nephrol. 1996;7(10):2176-2182.
68. Boudville N, Kemp A, Clayton P, et al. Recent peritonitis associates with mortality among patients treated with peritoneal dialysis. J Am Soc Nephrol. 2012;23(8):1398-1405.
69. Goossen K, Becker M, Marshall MR, et al. Icodextrin versus glucose solutions for the once-daily long dwell in peritoneal dialysis: an enriched systematic review and meta-analysis of randomized controlled trials. Am J Kidney Dis. 2020;75(6):830-846.
70. Ahn SV, Vonesh E, Han SH. Survival advantage of icodextrin peritoneal dialysis solution in a time-dependent model. Am J Kidney Dis. 2013;61(2):351-362.
71. Wang IK, Li Y-F, Chen J-H, et al. Icodextrin decreases technique failure and improves patient survival in peritoneal dialysis patients. Nephrol Dial. 2015;20(3):161-167.
72. Yang JY, Chen L, Peng YS, Chen YY, Huang JW, Hung KY. Icodextrin is associated with a lower mortality rate in peritoneal dialysis patients. Perit Dial Int. 2019;39(3):252-260.
73. Agar JWM, Hawley CM, Kerr PG. Home hemodialysis in Australia and New Zealand: how and why it has been successful. Semin Dial. 2011;24(6):658-663.
# Home Versus Facility Dialysis and Mortality in Australia and New Zealand

## Setting & Participants

52,097 adults starting dialysis in Australia and NZ 1998-2017

Mortality risk by modality compared between
- 1998-2002
- 2003-2007
- 2008-2012
- 2013-2017

Estimates computed using cause-specific and sub-distribution regression analysis

## Results

![Graph showing mortality risk comparison between different dialysis settings over time.](image)

*Unadjusted Deaths per 100 Person-Years (Per 1000 person-years)*

*Adjusted HR for mortality vs facility HD (Model Estimate, 95% CI)*

*Effects insensitive to competing risks, and not modified by diabetes, comorbidity, or sex. Secular changes / differences were greatest for older patients.*

CONCLUSION: Survival for patients treated with PD is greater than for those treated with facility HD in Australia and New Zealand.

---

Mark R Marshall, Kevan R Polkinghorne, Neil Boudville et al (2021)
@AJKDonline [DOI: 10.1053/j.ajkd.2021.03.018]