As of 12/01/2007, 36 kidney Ts took place at our center since the rule change and were compared to the 36 sequential Ts preceding the rule change. There were no statistically significant differences in recipients mean age (12 vs. 14 years), gender (61 vs. 64% male), or ethnicity (67 vs. 75% Caucasian) before vs. after the rule change. Moreover, the percentage of pre-emptive Ts and re-Ts were identical in both groups (27% and 16.7%, respectively, and the percentage of patients with high ≥(40%) panel-reactive antibodies was not significantly different (19.5% before vs. 27.8% after). There was a significant difference in mean donor age (32.4 years before vs. 22.8 years after, p<0.002). Patient survival (100% in both groups) and 1-year graft survival were comparable (100% before vs. 90% after, p=0.06). Significant increases were found in the percentage of deceased donor (DD) Ts and HLA mismatches (MMs) after the rule change (14% DDs before vs. 57% DDs after, p=0.002; 2.8 MMs before vs. 3.6 MMs after, p=0.03). We next compared the mean wait time for DD kidneys in the 20 DD Ts since the rule change with the wait time of the most recent 20 DD kidney recipients before the rule change and found that recipients under the age of 18 years had a significantly longer wait time before the rule change (315 vs. 119 days, p=0.04). Mean donor age in the 2 DD T cohorts was not significantly different (15.3 years before vs. 18.4 years after).

We conclude that at our center, the current allocation rules are associated with a substantial decrease in wait time for DD Ts and an increase in their relative number. Overall HLA matching decreased, but there was no statistically significant worsening in short-term patient or graft survival. These data suggest that overall access to kidney transplantation has improved for children. However, the accompanying decreases in LD Ts and HLA matching may affect long-term outcomes and possibly the overall donor pool unfavorably. Larger studies are needed to evaluate these possibilities.

We conclude that accelerated renal senescence in ECD results in glomerulopenia. Those subjects in the bottom quartile receiving a single allograft would have less than 15% of the normal complement of glomeruli and could lead to the remnant kidney phenomenon with progressive allograft failure.

We evaluated glomerular function and number in 43 subjects, who were studied post-Tx after SCr had reached a stable, nadir level (1.2 ±0.3 in aging vs 1.0 ±0.3 in youthful, p<0.05). GFR, renal plasma flow and oncotic pressure were determined by standard methods. The allograft ultrafiltration coefficient (Kf) was calculated by the model of Deen et. al. GFR at follow-up was lower in the aging than the youthful group by 30% (48 ±21 vs 69 ±17 ml/min, p<0.001). Neither MAP nor plasma oncotic pressure differed between the groups. The computed value for Kf in recipients of ECD was depressed 44% below youthful donors (3.7 ±2.2 vs 6.6 ±3.4 ml/min/mmHg; p<0.001). Thus, the extent of GFR depression seen in the aging donor group can entirely be accounted for by a decrease in Kf alone. Light and morphometric analysis revealed the glomerulosclerosis be higher in the aging (17 ±11 vs 2 ±2%, p=0.002), which fails to account for the difference in allograft Kf or GFR. Enlarged glomerular volume and surface area resulted in higher SNKf in the aging group (11.1 ±3.5 vs 8.6 ±2.6 ml/min/mmHg, p=0.07) and is consistent with glomerulosclerosis, but fails to account for the low Kf. Dividing Kf by SNKf provides an estimate of the number of functioning glomeruli, which was profoundly depressed in the aging compared to the youthful group (555 ±21 vs 847 ±344 x 106, p<0.05; fig 1).

We conclude that accelerated renal senescence in ECD results in glomerulopenia. Those subjects in the bottom quartile receiving a single allograft would have less than 15% of the normal complement of glomeruli and could lead to the remnant kidney phenomenon with progressive allograft failure.