Effects of two low-flux cellulose acetate dialysers on plasma lipids and lipoproteins—a cross-over trial

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Abstract

Background. Studies have shown a beneficial effect of high-flux dialysis on lipids, lipoproteins and lipoprotein lipase (Lpl) activity. This has been attributed to improved clearance of Lpl-inhibitory molecules of middle molecular weight, but differences in flux or biocompatibility have not been addressed. We conducted a blinded cross-over trial of two cellulose acetate dialysers (AN140, Althin Medical Inc. and CA210, Baxter Inc.) of similar flux (11 ml/h/mmHg transmembrane pressure) but with different clearances of larger molecules [AN140 sieving coefficient at mol. wt 11 000 Da (β₂-microglobulin) 0.6; CA210 sieving coefficient negligible].

Methods. Sixteen patients were divided into two groups to receive dialysis with AN140 for 1 week followed by CA210 or vice versa. Before and after the third dialysis with each membrane, plasma lipid and lipoprotein concentrations were measured. Post-dialysis post-heparin lipase activity was measured in six patients.

Results. Fifteen patients completed the study. No difference between dialysers was found for apolipoprotein (apo) A1, B or total cholesterol measurements. The rise in triglyceride post-dialysis was attenuated by AN140 (rise 0.05 ± 0.4 mmol/l vs CA210 0.44 ± 0.54 mmol/l, P = 0.03), while high density lipoprotein (HDL) cholesterol was increased by AN140 (rise 0.18 ± 0.12 mmol/l vs CA210 0.06 ± 0.14 mmol/l, P < 0.02). ApoE rose with AN140 during dialysis but declined with CA210 (1.10 ± 1.06 mg/dl and −0.77 ± 0.63 mg/dl, P = 0.002) as did apoCIII (HDL) (AN140 rise 1.33 ± 2.06 mg/dl; CA210 fall −0.67 ± 0.73 mg/dl, P = 0.001). Lpl activity, measured in six patients, tended to be higher for AN 140 (45.3 ± 10.5 mmol FFA/ml plasma/h vs CA 210 (37.2 ± 7.9 mmol FFA/ml plasma/h) (P = 0.16).

Conclusions. We conclude that low-flux dialysis using a cellulose acetate membrane with good clearance of higher molecular weight molecules may be associated with beneficial changes in plasma lipids and lipoproteins.

Key words: apolipoprotein; cellulose acetate; dialysis; lipids; lipoprotein lipase

Introduction

It is well established that chronic haemodialysis patients experience excessive cardiovascular mortality when compared with the general population [1,2]. Lipid abnormalities have been demonstrated in this population for >25 years [3], most consistently elevations in triglyceride and decrements in high density lipoprotein (HDL) concentrations [4,5]. Uraemic serum has been shown to impair lipoprotein lipase (Lpl) activation [6], and manoeuvres that increase Lpl activity, such as exercise [7] and fibric acid medications [8] were found to ameliorate these lipid abnormalities.

Apolipoprotein (apo) concentrations have also been found to differ in uraemic [9] and dialysis patients on either haemo- or peritoneal dialysis [10], although the atherogenicity of lipoprotein profiles in peritoneal dialysis patients has been suggested to be greater than that of haemodialysis patients [11]. Both uraemic and dialysis populations display lower levels of apop A1, AII and E in concert with markedly elevated values for apoCIII [12,13]. Markedly increased apoCIII:apoCII and apoCIII:apoE ratios in heparin-precipitable particles (primarily very low density lipoproteins; VLDL) have also been reported [12,14].

There has been some recent interest in the effects of different dialysis membranes on these lipid and lipoprotein abnormalities [15–17]. Patients undergoing chronic haemodialysis with high-flux biocompatible polysulfone membranes were demonstrated to have lower pre-dialysis triglyceride concentrations [15,17]. The role of post-heparin Lpl activity was also examined; one study found an increase with high-flux membranes [15], while another did not [17]. It was proposed that superior clearance of a hypothetical ‘middle molecule’ that inhibited Lpl activity could account for the improvement in Lpl activity and lipid profiles in one study, but issues of biocompatibility were not addressed [15]. It is certainly conceivable that other inhibitors of Lpl may exist in uraemic patients, and
two that have been proposed are increases in the number of triglyceride-rich particles [18] and increases in the particle content of apoCIII [19]. Another study found that apoCIII and Lp activity both independently predicted triglyceride concentration in multiple regression analysis in pre-dialysis patients [20]. High-flux haemodialysis has been demonstrated to lower apoCIII concentrations [21], although which lipoprotein particles were affected was not examined.

Accordingly, we undertook a study to examine the effects on lipid and lipoprotein profiles of two cellulose dialysis membranes, between which the only advertised difference lay in clearance of higher molecular weight substances, as measured by the sieving coefficient for \( \beta_2 \)-microglobulin. A cross-over design was employed, allowing greater precision when compared with past studies with similar sample size [22]. Pre- and post-dialysis lipids and lipoproteins were measured in all patients, and post-heparin lipolytic activity in six.

**Subjects and methods**

**Subjects and samples**

Characteristics of the patients studied and dialysis treatment details are provided in Table 1. In brief, patients with chronic renal failure of various aetiologies, excluding diabetes, that had been stable on haemodialysis for a minimum of 6 months (mean 3.5 years) were studied. Patients who were taking lipid-lowering agents, had a coagulopathy or who were unable to give informed consent or refused to participate were excluded. The study protocol was approved by the institutional review board of St. Joseph's Hospital, Hamilton, Ontario, Canada. All subjects gave signed informed consent. Patients were randomized to receive dialysis for four sessions with AN140 (Althin Medical Inc., Miami Lakes, FL) followed immediately by CA210 (Baxter Healthcare Corp., Deerfield, IL). The AN 140 is a 1.4 m² cellulose acetate haemodialyser. At a blood flow of 300 ml/min, the in vivo urea and creatinine clearances are 193 and 174 ml/min respectively. The in vivo ultrafiltration rate is 7.1 ml/h/mmHg and the sieving coefficient, after exposure to human plasma, is 0.6 for \( \beta_2 \)-microglobulin [27]. The CA210 is a 2.1 m² cellulose acetate haemodialyser. At a blood flow rate of 300 ml/min, the in vitro urea and creatinine clearances are 247 and 212 ml/min respectively. The ultrafiltration rate is 7.9 ml/h/mmHg. The sieving coefficient for \( \beta_2 \)-microglobulin is negligible.

**Sample analysis**

All samples were assayed together and therefore subjected to the intra-assay coefficient of variance only. These are provided at the bottom of the tables for the respective assays. Triglyceride (TG) and cholesterol (chol) were determined by colorimetric assay [23] using the respective kits (Diagnostic Chemicals Ltd., Monroe, CT). The phosphotungstic acid–magnesium chloride precipitation method was used to measure HDL cholesterol.

Apolipoproteins AI, AII, B, CIII, CIII (HDL) and E were measured by enzyme immunoassay [24]. In brief, for each apolipoprotein, 100 \( \mu \)l of the respective rabbit-raised anti-

Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Initials</th>
<th>Sex</th>
<th>Age</th>
<th>Aetiology of CRF</th>
<th>Haematocrit Pre-dialysis</th>
<th>Haematocrit Post-dialysis</th>
</tr>
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<tbody>
<tr>
<td>ES</td>
<td>M</td>
<td>52</td>
<td>Hypertensive</td>
<td>0.34</td>
<td>0.36</td>
</tr>
<tr>
<td>JM</td>
<td>F</td>
<td>61</td>
<td>Glomerulonephritis</td>
<td>0.32</td>
<td>0.35</td>
</tr>
<tr>
<td>JL</td>
<td>F</td>
<td>55</td>
<td>Hypertensive</td>
<td>0.29</td>
<td>0.29</td>
</tr>
<tr>
<td>KR</td>
<td>M</td>
<td>46</td>
<td>Polycystic</td>
<td>0.31</td>
<td>0.34</td>
</tr>
<tr>
<td>KD</td>
<td>M</td>
<td>68</td>
<td>Vascular</td>
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</tr>
<tr>
<td>MF</td>
<td>F</td>
<td>64</td>
<td>Hypertensive</td>
<td>0.30</td>
<td>0.31</td>
</tr>
<tr>
<td>MD</td>
<td>M</td>
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</tr>
<tr>
<td>CD</td>
<td>M</td>
<td>53</td>
<td>Glomerulonephritis</td>
<td>0.31</td>
<td>0.35</td>
</tr>
<tr>
<td>GR</td>
<td>F</td>
<td>61</td>
<td>Hypertensive</td>
<td>0.37</td>
<td>0.38</td>
</tr>
<tr>
<td>VS</td>
<td>F</td>
<td>62</td>
<td>Hypertensive</td>
<td>0.33</td>
<td>0.36</td>
</tr>
<tr>
<td>AR</td>
<td>F</td>
<td>64</td>
<td>Analgesic</td>
<td>0.36</td>
<td>0.38</td>
</tr>
<tr>
<td>TC</td>
<td>M</td>
<td>32</td>
<td>Reflux</td>
<td>0.29</td>
<td>0.32</td>
</tr>
<tr>
<td>BP</td>
<td>M</td>
<td>36</td>
<td>Reflux</td>
<td>0.32</td>
<td>0.35</td>
</tr>
<tr>
<td>JA</td>
<td>M</td>
<td>62</td>
<td>Vascular</td>
<td>0.31</td>
<td>0.31</td>
</tr>
<tr>
<td>AD</td>
<td>M</td>
<td>66</td>
<td>Polycystic</td>
<td>0.38</td>
<td>0.39</td>
</tr>
<tr>
<td>NK</td>
<td>F</td>
<td>57</td>
<td>Hypertensive</td>
<td>0.31</td>
<td>0.33</td>
</tr>
</tbody>
</table>
body (15 μg/ml) was pipetted into a microtitre plate well (Dynatech Labs Inc., Alexandria, VA). This was incubated overnight at 4 °C in sodium phosphate buffer (10 mmol/l, pH 7.4 with NaCl 0.15 mmol/l). Plates were then washed three times with washing buffer (sodium phosphate buffer with Tween 0.5 g/l) and blocked with albumin [sodium phosphate buffer with bovine serum albumin (BSA) 10 g/l] (Sigma Chemical Inc., St. Louis, MO). Serum samples were diluted 20 000-fold and then 100 μl was added to each well. Plates were covered and incubated at 37 °C for 2 h. Remaining liquid was then aspirated and plates washed five times with sodium phosphate buffer. Then 100 μl of anti-rabbit peroxidase antibody conjugate (Vector Laboratories, Burlingame, CA) diluted 7500-fold in assay buffer was added to each well. Plates were again incubated for 2 h and aspirated and washed as above. Subsequently, 100 μl of freshly prepared o-phenylenediamine dihydrochloride substrate (Sigma Chemical Inc., St. Louis, MO) at 3 g/l in sodium phosphate citrate buffer (0.1 mmol/l, pH 5.6, with 0.2 g of H₂O₂/l) was then added to each well and allowed to stand for 30 min at room temperature in the dark. The reaction was then stopped by adding 100 μl of 2.5 mmol/l H₂SO₄. The absorbance of each well was then read at 490 nm (Bio-Tek Instruments Ltd., Burlingame, CA).

Post-heparin lipolytic activity was determined with a detergent-solubilized trioleoylglycerol emulsion as described previously [25]. Briefly, the substrate was prepared by adding 200 mg of trioleoylglycerol in chloroform and 200 μCi of tri-[1-¹⁴C]-oleoylglycerol to a 50 ml conical glass tube and evaporated to dryness. Next, 15 ml of 1 M Tris–HCl pH 8.0, 15 ml of 20% BSA and 15 ml of 0.2% Triton N-101 (all Sigma Chemical Inc., St. Louis, MO) was added. The mixture was emulsified by sonication for 4 min at 4 °C. Post-heparin lipolytic activity was then determined. Assays were performed in triplicate tubes, being a no-plasma blank, plasma and plasma plus 1 M NaCl.

The blood sample used as a standard was from a normal human volunteer 10 min after the injection of 50 U/kg heparin. Reactions were performed on ice and contained 75 μl of substrate, 50 μl of normal human plasma and 50 μl of the post-heparin plasma. For hepatic lipase activity, 50 μl of 5 M NaCl was added. The final volume was brought up to 250 μl and the samples incubated for 15 min at 37 °C. Reactions were stopped by the addition of 3 ml of methanol/chloroform/heptane (140:125:100) and 1 ml of 140 mM potassium carbonate and 140 mM boric acid at pH 10.6. After mixing and centrifugation (2000 g) at room temperature, 1 ml of the top fraction was removed and radioactivity determined. Activity was expressed as micromoles of oleic acid released per hour per ml of plasma. Lpl activity was determined by subtracting hepatic lipase activity from the total.

Data analysis

Comparison of pre-dialysis data for the two dialysers for each treatment order (commencing with AN140 and ending with CA210 or commencing with CA210 and ending with AN140) by analysis of variance revealed no statistical difference, indicating no order effect. The data for each membrane was therefore pooled irrespective of the treatment order. The statistical analysis was performed on differences between post- minus pre-dialysis values for each dialyser using either the paired t-test (for normally distributed data) or Wilcoxon’s signed rank test (for non-normally distributed data).

Results

Patients and protocol

Fifteen of 16 patients completed the study (Table 1). One patient dropped out after two dialyses with the initial dialyser (CA210) as he received a renal transplant. There were no other drop-outs, deviation from the protocol or mortality.

Cholesterol and triglycerides

No difference between dialysers was seen for total cholesterol. There was, however, a significantly greater intradialytic rise in HDL cholesterol with the AN140 compared with the CA210 dialyser (P=0.007). In addition, the observed intradialytic rise in triglyceride was significantly blunted by the AN140 dialyser (P=0.02) (Table 2, Figure 1).

Apolipoproteins

No differences between dialysers were seen for apoAI or for apoB. Likewise, there was no difference in the apoAI:apoB ratios between the two dialysers. Whilst there was no difference in total apoCIII, more of the apoCIII was apportioned to the non-heparin-precipitable (HDL) fraction (P=0.002). ApoE was also significantly increased intradialytically by AN140 when compared with CA210 (P=0.002) (Table 3, Figure 2). The results did not differ when corrected for volume change as determined by pre- and post-dialysis haematocrit (data not shown).

Lipoprotein lipase activity

Lpl activity did not differ significantly after dialysis in the six patients in whom it was measured, although Differences yielding P-values of <0.05 were labelled significant.

Fig. 1. Lipid differences across dialysis with AN140 and CA210.
Table 2. Triglycerides and cholesterol

<table>
<thead>
<tr>
<th></th>
<th>Triglyceride (mmol/l)</th>
<th>Total chol (mmol/l)</th>
<th>HDL chol (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA210</td>
<td>pre- 1.6 ± 0.7</td>
<td>4.24 ± 0.71</td>
<td>1.0 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>post- 2.0 ± 1.0</td>
<td>4.92 ± 1.06</td>
<td>1.1 ± 0.3</td>
</tr>
<tr>
<td>Difference (post–pre)</td>
<td>0.4 ± 0.5</td>
<td>0.68 ± 0.59</td>
<td>0.1 ± 0.1</td>
</tr>
<tr>
<td>AN140</td>
<td>pre- 1.7 ± 0.8</td>
<td>4.36 ± 0.73</td>
<td>1.0 ± 0.4</td>
</tr>
<tr>
<td></td>
<td>post- 1.7 ± 0.8</td>
<td>4.94 ± 0.92</td>
<td>1.2 ± 0.3</td>
</tr>
<tr>
<td>Difference (post–pre)</td>
<td>0.0 ± 0.4</td>
<td>0.58 ± 0.32</td>
<td>0.2 ± 0.2</td>
</tr>
</tbody>
</table>

Each lipid parameter was determined in a single assay. The intra-assay coefficient of variance was 0.6% for triglycerides, 0.8% for cholesterol and 1.1% for HDL cholesterol.

Table 3. Apolipoproteins

<table>
<thead>
<tr>
<th></th>
<th>apoE (g/l)</th>
<th>apoAI (g/l)</th>
<th>apoB (g/l)</th>
<th>apoCIII (mg/dl)</th>
<th>CIII-HDL (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA210</td>
<td>pre- 1.22 ± 0.2</td>
<td>1.00 ± 0.25</td>
<td>13.0 ± 5.1</td>
<td>7.1 ± 2.9</td>
<td>6.6 ± 2.4</td>
</tr>
<tr>
<td></td>
<td>post- 1.39 ± 0.3</td>
<td>1.17 ± 0.36</td>
<td>13.3 ± 4.8</td>
<td>6.4 ± 2.9</td>
<td>5.9 ± 2.2</td>
</tr>
<tr>
<td>Difference (post–pre)</td>
<td>0.17 ± 0.13</td>
<td>0.17 ± 0.18</td>
<td>0.3 ± 1.4</td>
<td>−0.7 ± 0.7</td>
<td>−0.6 ± 0.7</td>
</tr>
<tr>
<td>AN140</td>
<td>pre- 1.26 ± 0.21</td>
<td>1.05 ± 0.25</td>
<td>13.8 ± 4.8</td>
<td>7.9 ± 2.6</td>
<td>7.6 ± 4.3</td>
</tr>
<tr>
<td></td>
<td>post- 1.21 ± 0.24</td>
<td>1.21 ± 0.33</td>
<td>14.3 ± 4.9</td>
<td>9.2 ± 2.7</td>
<td>8.6 ± 4.5</td>
</tr>
<tr>
<td>Difference (post–pre)</td>
<td>0.05 ± 0.08</td>
<td>0.16 ± 0.13</td>
<td>0.5 ± 1.3</td>
<td>1.3 ± 1.1</td>
<td>1.0 ± 1.1</td>
</tr>
<tr>
<td>P for differences</td>
<td>0.233</td>
<td>0.450</td>
<td>0.499</td>
<td>0.002</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Each apolipoprotein parameter was measured in a single assay. The intra-assay coefficient of variance was 1.1% for apoAI, 1.9% for apoB, 3% for apoCIII, 3.8% for apoCIII (HDL) and 2.4% for apoE.

Discussion

This is the first study to examine the effects of two different dialysis membranes on lipid and lipoprotein parameters whilst controlling for flux and, presumably, as the membranes are constructed of the same material, biocompatibility. A cross-over design allowed direct intra-patient comparisons of changes in lipoprotein and lipid parameters.

Baseline lipid and lipoprotein levels were similar in our subjects to previously reported chronic stable haemodialysis patients in the literature [26], and similar to subjects enrolled in other studies assessing effects of dialysis membranes on lipoprotein and lipid metabolism [15,17]. The magnitude of the changes observed in the lipid and lipoprotein measurements common to all studies was also similar [15,17]. No studies have heretofore corrected post-dialysis lipid and lipoprotein parameters for volume change across dialysis [15,17]. When pre- and post-haematocrits were used as indicators of volume change, the results did not differ in this study. In this report, haemodialysis with low-flux cellulose acetate-based dialysers was shown to have different effects on lipids and lipoproteins. This was associated with different sieving coefficients of the two dialysers for β-2-microglobulin (AN140 higher). There was no other advertised difference between the two membranes [27].
We have shown that dialysis with the AN140 membrane was associated with post-dialysis elevations in HDL cholesterol and attenuation of the post-dialysis rise in triglyceride. In conjunction with this, apoE and the HDL-associated apoCIII also rose. No differences were observed in total cholesterol, apoAI, apoB or total apoCIII. Lpl activity tended to be higher post-dialysis with the AN140 membrane in those patients in whom it was measured (n=6). Virtually all confounding factors may be excluded by virtue of the cross-over design. There did not appear to be a carry-over effect as judged by the lack of effect of either dialyser on pre-dialysis values.

Other studies of the effects of dialysis membrane on lipid parameters have concentrated on the apparent benefits of high-flux dialysers, which have been shown consistently to lower triglyceride across dialysis [15–17]. The effect on triglyceride with low-flux dialysers has been more variable, with some workers showing a decrease across dialysis [15,17], and some an increase [21], as we observed. The reasons for these discrepancies are not clear, as heparin was used in all cases. Total cholesterol has generally not been affected, and effects on HDL cholesterol were variable [15–17].

The major changes in lipoproteins we observed included post-dialysis elevations in apoE and apoCIII (HDL) with the AN140 membrane. The lack of difference in total CHI suggests that less CHI was associated with heparin-precipitatable particles (VLDL) with the AN140 membrane. This may be of importance. Lpl has been reported to be non-competitively inhibited by apoCIII [28], and mice transgenic for apoCIII have a remarkable hypertriglyceridaemia [29]. Heparin-precipitatable apoCIII was the strongest risk factor for angiographic progression of coronary disease in humans [30]. Subjects with chronic renal failure have elevated apoCIII levels, and a lower apoAI:apoCIII ratio predicted vascular disease in this group [12]. In pre-dialysis patients, the apoCIII level was shown to predict hypertriglyceridaemia in multiple logistic regression analysis [20].

In this study, Lpl activity was measured in six patients only. Whilst it tended to be higher after AN140 dialysis, this difference did not reach statistical significance. This may be due to lack of power to detect a difference in this small sample, or it may indicate that the observed changes are not due to altered Lpl activity. In this respect, it is noteworthy that changes in heparin-precipitatable apoCII or CHI may not be associated with changed in vitro Lpl activity, despite rendering the affected particles less susceptible to Lpl-mediated lipolysis [31]. A fibric acid derivative was observed to increase the apoCII:apoCIII ratio and improve dyslipidaemia in continuous ambulatory peritoneal dialysis (CAPD) patients without affecting Lpl or hepatic lipase activity [32]. ApoCIII transgenic mice do not have reduced Lpl activity in spite of marked hypertriglyceridaemia [32]. Consequently, elevations in apoCIII in VLDL may result in abnormalities of disposition of these triglyceride-containing particles without affecting Lpl activity.

Others have postulated this mechanism to account for an increase in triglyceride in pre-dialysis patients without a change in measured Lpl activity [20].

The other major lipoprotein difference found across dialysis with these two dialysers was an increase in apoE with the AN140 dialyser, whilst apoE declined across dialysis with the CA210 dialyser. Increases in apoE content of VLDL have also been associated with enhanced lipolysis [18]. Furthermore, apoE can enhance apoCII-dependent lipolysis of artificial substrates, and so may be a co-activator of Lpl [33]. Lack of apoE-mediated uptake of triglyceride was postulated as the mechanism leading to hypertriglyceridaemia in apoCIII transgenic mice, who show reduced VLDL-associated apoE in concert with increased apoCIII [34].

Our results, and those of others [35,36] support a construct in which abnormal VLDL composition, specifically increased apoCII and decreased apoE results in impaired non-enzymatic removal of these particles, particularly by the liver. ApoCII retards apoE-mediated remnant VLDL uptake [32,37]. Decrements in Lpl activity or the ability of Lpl to metabolize abnormally constructed VLDL particles may play a role, although this does not appear to be indispensable.

This study does not explain how the AN140 dialyser might yield these changes, although the difference in sieving coefficient and, presumably, clearance of higher molecular weight molecules is likely to be responsible, as this is the only apparent difference between the two dialysers used in this study. Similar effects on lipids were found with high-flux dialysers in the studies of Josephson and co-workers and Seres and colleagues. Both suggested that an Lpl inhibitor might only be cleared by high-flux dialysis [15,16]. Clearly, however, increased biocompatibility of the polysulfone high-flux membranes used could have led to the same effect by decreasing production of cytokines that might also inhibit Lpl or otherwise alter lipid metabolism [38]. Cytokine mediators of inflammation, especially interleukin-1 (IL-1) have been demonstrated to impair Lpl activity [39]. This study would tend to support the contentions in these previous works that clearance of higher molecular weight molecules is important, as biocompatibility should not differ between the two membranes used given that they are constructed of the same material. There are, however, no published data on biocompatibility of the AN140 membrane. It is important to note that none of these works, including ours, have measured putative markers of biocompatibility [40].

The role of membrane in the treatment of the dyslipidaemia of end-stage renal disease is uncertain. Given the very high cardiovascular mortality of patients on haemodialysis [1], efforts to improve lipid abnormalities must remain a therapeutic goal. While beneficial changes can be demonstrated across dialysis with some membranes, pre-dialysis lipid parameters may [17] or may not be affected [15]. We detected no change in pre-dialysis lipids in this short-term trial. Therefore, the role of membrane in the treatment of uraemic dyslipidaemia remains unclear.
Comparison of two low-flux cellulose acetate dialysers

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