Dear Sir,

We have read with interest the article of Paczek et al. [1] about the inhibitory effect of dialysis membranes on β2-microglobulin synthesis and release from human lymphocyte culture. Nowadays, dialysis-amyloidosis represents a frequent and invalidating complication of patients with chronic renal failure on hemodialysis treatment, specially after long-term period. The pathogenesis remains unknown and the significance of β2-microglobulin serum levels and intradialysis production also remain questioned [2]. In a similar study to that of Paczek et al., trying to determine the role of dialysis membranes on β2-microglobulin synthesis, we obtained close results, confirming the inhibitory effect of dialysis membranes on β2-microglobulin release from lymphocyte culture.

Twenty-four patients (mean age 52 ± 11 years) affected with chronic renal failure and on hemodialysis treatment for a mean time of 5.7 ± 3.6 years, and a control group of 6 subjects with normal renal function and a mean age of 39 ± 5 years old, took part in the study. β2-Microglobulin was determined in the supernatant using commercially available EIA(Phadezym β2-Microtest; Pharmacia Diagnostics) with a sensitivity between 3.3 and 500 ng/ml.

![Fig. 1. Inhibitory effect on β2-microglobulin synthesis and release by dialysis membranes.](image-url)

Dialysis membranes sterilized with gamma rays.

Peripheral blood lymphocyte cultures were performed with conventional methodology [3] during an incubation period of 9 days. Two different short tubes of dialysis membranes were incubated in lymphocyte cultures: cuprophane (ST-15; Travenol) and polyacrylonitrile (AN-69; Hospal). Dialysis membranes were sterilized in two different ways, i.e., with ethylene oxide and with...
gamma rays (1,000 rad). The lymphocyte cultures were incubated with different amounts of dialysis membranes: 5, 10, 20 and 40 mg of each type of dialysis membrane.

Table 1. Amount of dialysis membranes

<table>
<thead>
<tr>
<th>Conditions</th>
<th>5 mg</th>
<th>10 mg</th>
<th>20 mg</th>
<th>40 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>500 ± 228</td>
<td>138 ± 77</td>
<td>234 ± 148</td>
<td>309 ± 142*</td>
</tr>
<tr>
<td>AN-69</td>
<td>95 ± 56</td>
<td>77 ± 32</td>
<td>68 ± 33</td>
<td>49 ± 13</td>
</tr>
<tr>
<td>Cuprophane</td>
<td>147 ± 108</td>
<td>242 ± 143</td>
<td>303 ± 101*</td>
<td>242 ± 143</td>
</tr>
</tbody>
</table>

Gamma ray-sterilized membranes.

Fig. 2. Progressive inhibitory effect for different amounts of dialysis membranes on β2-microglobulin synthesis.
In the first experiment, both types of membranes developed an intense and statistically significant inhibitory effect on the release and synthesis of \( \beta_2 \)-microglobulin into the supernatant. No differences were observed between the inhibitory effect exerted by the two membranes (table 1). When lymphocyte cultures were incubated with dialysis membranes sterilized with gamma rays, the inhibitory effect was similar but slightly less intensive, and without differences between the two incubated membranes (fig. 1). When different amounts of dialysis membranes were incubated, we observed a progressive inhibitory effect on the \( \beta_2 \)-microglobulin synthesis and release, the strongest inhibition being when the largest amount of membrane (40 mg) was incubated. This effect was similar with cuprophone and polyacrylonitrile membranes (fig 1).

crease and during polyacrylonitrile dialysis serum levels drop, confirming a \( \beta_2 \)-microglobulin synthesis during dialysis. The inhibitory effect observed by Paczek et al. and our group must be explained as a lessive or toxic effect of dialysis membranes on lymphocyte function. Paczek et al. and our group (data not reported) demonstrated that adsorption of \( \beta_2 \)-microglobulin to cuprophone membranes was negligible, excluding this hypothesis as the reason for the decreased concentration of \( \beta_2 \)-microglobulin on the supernatant. In our experiment a direct toxic effect exerted by the ethylene oxide could also be excluded, since membranes sterilized by gamma rays developed a similar inhibitory effect. We agree with Paczek et al. that these results can be excluded as a direct contribution of dialysis membranes on \( \beta_2 \)-microglobulin serum levels, but cannot be excluded as an indirect stimulation (cellular and/or humoral) of \( \beta_2 \)-microglobulin synthesis and release during dialysis treatment.

References

Since Hauglustaine et al. [4] proposed that cuprophone dialysis membrane would actively contribute to \( \beta_2 \)-microglobulin synthesis and dialysis-amyloidosis, large discussions have developed. Our group (data not reported), using \( ^{131} \)I-labelled \( \beta_2 \)-microglobulin and endogenous \( \beta_2 \)-microglobulin, demonstrated that during cuprophone dialysis \( \beta_2 \)-microglobulin plasma levels in-