**Table S1.** Characteristics of the patients, mean age  $54.66 \pm 5.08$ ; range 23-79 yrs.\*Patients with a diagnosis of cancer were free of metastatic disease and peritoneal involvement.

Lorazepam	
Lorazepam	
Lorazepam	
Lorazepam	
Lorazepam	
No	
No	
No	

## Cell culture

HPMCs were isolated from omental tissue from 10 different donors (free of any cardiovascular or peritoneal disease and non-taking anti-inflammatory drugs or antioxidants) undergoing nonurgent, non-septic abdominal surgery, using previously described methods (Chung-Welch et al., 1997). HPMCs were routinely cultured in M199 containing 1 g l<sup>-1</sup> of D-glucose and supplemented with 10% FCS, 100 mgml<sup>-1</sup> streptomycin, 100Uml<sup>-1</sup> penicillin, and 2.5 mgml<sup>-1</sup> amphotericin. At confluence, HPMC were passaged using a 0.02% EDTA–0.05% trypsin solution and split in a 1:2 ratio.

HPMCs characterization was based on both cell morphology (immediately prior to and at confluence, cells adopted the polygonal cobblestone-like appearance characteristic of epithelial cells and formed a monolayer) and indirect immunofluorescence staining of several human mesothelial markers (Chung-Welch et al., 1997). In brief, HPMCs showed a diffuse positive staining with an anti-von Willebrand factor antibody (Dakopatts, Glostrup, Denmark) and a marked staining with anti-cytokeratins 8 and 18, anti-E-cadherin, and anti-vimentin antibodies (all of them from Sigma Chemical Co.). HPMCs failed to express the endothelial marker PECAM-1 (CD31) (see Supplemental data, Table 2).

Table S2. HPMC characterization.

Markers	НРМС	HAEC	HUVEC	HASMC
Factor VIII (vWF)	+/-	+++	+++	negative
(, ,, = )	(diffuse)	(granular)	(granular)	
α-actin	negative	negative	negative	+++
E-Cadherin	++	negative	negative	
VE-Cadherin	+++	++	++	
Vimentin	+++	++	++	
Cytokeratin 8	++	++	++	
Cytokeratin 18	++	++	++	
PECAM-1(CD31)	negative	++		

HPMC, <u>Human Peritoneal Mesothelial Cells</u>; HAEC, <u>Human Aortic Endothelial Cells</u>; HUVEC, <u>Human Umbilical Vascular Endothelial Cells</u>; HASMC, <u>Human Aortic Smooth Muscle Cells</u>. The morphologic and immunofluorescence-staining features of the cells remained stable throughout the passages used.

## Preparation of Amadori adducts

Lyophilised human haemoglobins, nonenzymatically glycosylated at either elevated or normal levels, containing 11.1% and 5.4% HbA1, respectively, were purchased from Sigma. Briefly, haemoglobins were dissolved in deionised water and subsequently reduced by incubation with an excess of sodium dithionite. The haemoglobin solutions were then extensively dialysed using a 0.25A° pore diameter (approximately 12 kDa mol wt) dialysis membrane (Viskings, Serva, Heidelberg, Germany) against deionised water containing 10mgl<sup>-1</sup> EDTA and continuously bubbled with N2. Oxyhaemoglobins were then aliquoted and stored at -70 °C until used.

The absence of AGEs in the glycated oxyhaemoglobin solutions was assessed by measuring fluorescence in a Fluostar fluorometer (BMG Labtechnologies, Offenburg, Germany) at excitation maximum of 370 nm and emission maximum of 440 nm, which allows quantifying total AGEs (Sell & Monnier, 1989). A standard curve (r½0.99) was carried out using AGE-modified BSA (0.5–5 mgml<sup>-1</sup>), prepared following a previously described method (Bucala et al., 1991). Glycated preparations did not contain significative bacterial endotoxin contamination (p0.5U endotoxin ml<sup>-1</sup>), as measured with Pyrogents plus kit (Biowhittaker Europe SPRL, Verners, Belgium).