Supplemental Methods

Genotyping of single nucleotide polymorphisms in the promoter of HO-1 gene

The promoter region (up to 1.4 kb) was analyzed by direct sequencing. Primer sequences have been published elsewhere (1). Briefly, PCR reactions carried out in Amplitaq Gold Buffer (Applied Biosystems, Courtaboeuf, France) with 2.5 mM $MgCl_2$, 200 µM dNTPs (Roche Diagnotics, Mannheim, Germany), 10 pmol of each oligonucleotide primers, approximately 200 ng genomic DNA and 1 U AmpliTaq Gold Taq polymerase (Applied Biosytems, Couratboeuf, France). After initial denaturation for 10 min at 95°C, 35 cycles were carried out at 95°C for 30 s, 55°C for 30 s and 72°C for 1 min. Amplicons were then prepared for sequencing by incubation with Shrimp alkaline phosphatase and exonuclease I (EXOSAP®, Amersham Biosciences, Orsay, France). Nucleotide sequencing was carried out using CEQ Dye Terminator Cycle Sequencing (Beckman Coulter, Villepinte, France) and analyzed using a capillary sequencer CEQ 8000 (Beckman Coulter). All sequence changes were confirmed on both strands.

1. Ono K, Goto Y, Takagi S, Baba S, Tago N, Nonogi H, et al. A promoter variant of the heme oxygenase-1 gene may reduce the incidence of ischemic heart disease in Japanese. Atherosclerosis. 2004 Apr;173(2):315-9.

Supplemental table

eTable 1: Allele and genotype frequencies of –1135 and –413 polymorphisms

	Severe hemophilia A patients	
	Inhibitor-positive – N (%)	**Inhibitor-negative – N (%)**
-1135 A>G		
Genotype AA	18 (29.5%)	46 (26.4%)
Genotype GG	19 (31.1%)	57 (30.5%)
Genotype AG	24 (34.9%)	84 (44.9%)
Allele A	60 (49.2%)	176 (47.1%)
Allele G	62 (50.8%)	198 (52.9%)
-413 A>T		
Genotype AA	18 (36.7%)	62 (35.2%)
Genotype TT	19 (38.8%)	82 (46.6%)
Genotype AT	12 (24.5%)	32 (18.2%)
Allele A	55 (56.1%)	206 (58.5%)
Allele T	43 (43.9%)	146 (41.5%)
N of patients	61	187