Hepatocytes corrected by gene therapy are selected *in vivo* in a murine model of hereditary tyrosinaemia type I

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Tables 1 & 2 were inadvertantly omitted from the original publication. Also, the last sentence of the Fig. 2 legend should read: "The number below lanes 9–15 indicates the % of total hybridization signal contributed by the wild-type (lower band) allele".

Table 1 Biochemical measurements in transplanted and retrovirus treated FAH deficient mice

				Transplanted		Retrovirus treated	
		wild type	mutant	on NTBC	off NTBC	Single injection	Multiple injection
FAH enzyme	µmol/g/min	54±12 (10)	0.18±0.2 (10)	2.25±1.6 (5)	41±20.2 (7)	7±4.5 (5)	32±14 (5)
Succinylacetone	µg/L	21.4±13 (6)	932±131 (4)	63.7±23.5 (5)	42.1±21 (7)	166±62 (18)	44.4±15.5 (4)
AST	U/L	70±8 (7)	969±583 (3)	140±51 (4)	146±68 (5)	307±112 (21)	73±8 (4)
Conjugated bilirubin	mg/dL	0 (6)	4.5±3.2 (9)	0.04±0.05 (5)	0.03±0.05 (6)	1.3±1.9 (23)	0 (5)

The measurements are given with 1 standard deviation and the number of animals tested in brackets.

Human choroideremia protein contains a FAD-binding domain

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Fig. 1 was improperly aligned. The correct version appears below.

Table 2 Transplantation with limiting numbers of donor cells

Number of cells transplanted	Number of mice transplanted	Survivors
100	6	1
400	1	0
600	2	1
800	1	1
1,000	6	5
10,000	9	9

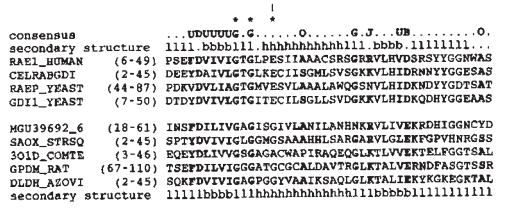

Fig. 1 Human choroideremia proteins and other REPs and GDIs contain a conserved FAD-binding domain The alignment is an excerpt of the output of the database search with the MoST program. A multiple alignment block was constructed from the BLASTP output for the CHM protein with the CAP program, and a position-dependent weight matrix derived from this block was used for iterative database scanning with the MoST program. A ratio of the expected number of detected sequence segments to the number actually extracted from the database of 0.001 was used as the cut-off for this search[14]. The original block included only the N-terminal segments of REPs and GDIs. The sequences were from the SWISS-PROT (an underline before the organism name) or GenBank databases, and the names are exactly as in the respective database. The upper block of aligned sequences includes REPs and GDIs; RAE1 is the human choroideremia gene product. CELRABGDI is the putative *C. elegans* GDI. The bottom block shows selected sequences of dehydrogenases extracted from the database by the MoST search (MGU39692_6, putative *M. genitalium* dTDP-4-dehydrorhamnose reductase; SAOX, *Streptococcus* sp. sarcosine oxidase; 3O1D, *Comamonas testosteroni* 3-oxosteroid 1-dehydrogenase; GPDM, rat mitochondrial glycerol-3-phosphate dehydrogenase; DLDH, *Azotobacter Vinelandii* dihydrolipoamide dehydrogenase). The consensus shows amino acid residues conserved in at least 8 out of the 9 aligned sequences; U indicates a bulky hydrophobic residue (I, L, V, M, F, Y, W); O indicates a small residue (G, A, S); J indicates a positively charged residue (K, R); B indicates a negatively charged residue (D, E); dot indicates any residue. The P-loop comprising the FAD-binding motif is overlined, and the 3 positions typically occupied by glycines, are marked by asterisks. The position containing glutamic acid in REPs and GDIs in contrast to glycine in most FAD(NAD)-binding proteins is additionally denoted by an exclamation mark. The secondary structure prediction for CHM and the experimentally observed secondary structure elements of DLDH are shown above and below the alignment, respectively (a indicates α-helix; b indicates β-strand, and l indicates loop).

Loss of NF1 results in activation of the Ras signaling pathway and leads to aberrant growth in haematopoietic cells

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