Table S1: Yeast SOD1 mutations allowing for suppression of *ccs*Δ lysine auxotrophy.

Mutation	**Number of isolates[a]**	**CCS Dependent SOD Activity[b]**	**CCS Independent SOD Activity[b]**	**Corresponding Residue in hSOD1[c]**	**Corresponding Residue in wSOD-1[d]**
A35T	2	++++	-	K	K
A35V	2	++++	-	K	K
A53V	2	++++	-	N	S
V113I	1	++++	-	I	V
V30I	2	++++	-	V	I
Q2K	1	++++	+	K	R
Q2R	5	++++	+	K	R
I108V,P144A	1	++++	+++	S, S	Y, A
K8R,P144L	4	++++	+++	K, S	R, A
P144L	2	++++	+++	S	A
P144Q	1	++++	+++	S	A
P144S	3	++++	+++	S	A
V16I, P144L	1	++++	+++	I, S	T, A
V81A, V113I, P144Q	1	++++	+++	E, I, S	I, V, A

[a]Number of times the identical clone was isolated in the library of 10,000 transformants. [b]Relative SOD1 activity as measure by the native gel assay. ++++ and +++ = maximal CCS dependent and independent activity respectively, as seen in Fig. 5A; + = low but detectable activity on SOD gels; "-" = no detectable activity by the native gel assay, nevertheless these mutants consistently promote lysine independent aerobic growth of *sod1*Δ *ccs1*Δ mutants. [c,d]Identity of the corresponding residue in human SOD1 or *C. elegans* SOD-1 based on multi-sequence alignments.