Table S1 Sequence polymorphism in mtDNA coding region of suppressor and non-suppressor strains compared with Refseq

Nucleotide change	Gene	Coding change	BER1	QI2	CO3	Reids-1	*tko*^{25t} (original)	Oregon R-C	Canton S	Oregon R
671C>T	ND2	silent	X						X	
710G>A	ND2	silent	X						X	
735C>T	ND2	silent	X						X	
791A>G	ND2	silent	X						X	
838TA>AT	ND2	L>Y	X	X	X	X	X	X	X	X
1068A>C	ND2	I>L	X						X	
1154T>C	ND2	silent							X	
1478G>A	COI	R>Q					X			
1512T>C	COI	silent							X	
1674G>A	COI	silent	X						X	
1779A>T	COI	silent	X						X	
1836A>G	COI	silent	X						X	
1861T>C	COI	silent	X						X	
1917A>G	COI	silent	X							
1929G>A	COI	silent	X						X	
2071C>T	COI	silent	X							
2136G>A	COI	silent		X	X					
2160T>C	COI	silent	X						X	
2187C>T	COI	silent	X						X	
2661C>T	COI	silent	X							
2863C>T	COI	silent	X	X	X	X	X	X	X	X
2928A>T	COI	Q>H				X				
2964G>A	COI	silent	X						X	
3517T>C	COII	silent	X						X	
3583T>C	COII	silent	X							
3685G>A	COII	silent	X						X	
4088C>T	A6	silent					X	X		X
4096T>C	A6	L>S	X	X	X	X	X	X	X	X
4247A>T	A6	silent	X						X	
4592T>A	A6	N>K					X	X		X
4599T>C	A6	S>P	X						X	
4616A>T	A6	silent							X	
4620A>G	A6	M>V	X						X	
4694G>A	A6	silent				X				
4762C>T	COIII	silent	X						X	
4898G>A	COIII	V>M				X				

5212A>G	COIII	silent	X						X	
5347C>T	COIII	silent	X							
5396T>C	COIII	silent		X	X	X	X	X	X	X
5419C>T	COIII	silent		X	X	X	X	X		X
5524A>G	COIII	silent		X	X	X				
5644C>T	ND3	silent		X	X	X	X	X		X
5964delAT	intergenic			X	X					
5966delAT	intergenic			X	X	X	X	X	X	X
5966ATT>TTATA	intergenic		X							
6050insTTAAT	intergenic								X	
6301A>C	tRNA-glu	TΨCloop							X	
6305C>T	tRNA-glu	TΨCloop	X	X	X	X	X	X	X	X
6620A>T	ND5	I>M		X	X	X	X	X		X
6982A>G	ND5	silent		X	X	X	X	X	X	X
7130delCAATTC	ND5	delIG	X	X	X	X	X	X		X
7180G>A	ND5	silent	X						X	
7870G>A	ND5	silent	X							
7888G>A	ND5	silent	X						X	
7906T>C	ND5	silent					X	X		X
8153T>A	tRNA-his	DHU loop	X							
8875G>A	ND4	silent							X	
8944AC>GA	ND4	Y>H	X	X	X	X	X	X	X	X
8981C>T	ND4	silent	X							
9005A>G	ND4	silent	X						X	
9044T>A	ND4	silent	X						X	
9064C>A	ND4	V>L							X	
9187A>G	ND4	silent	X						X	
9783G>A	ND4L	silent	X				X	X		X
9886delT	tRNA-thr	TΨCloop					X	X		X
9990C>T	ND6	silent	X						X	
10225C>T	ND6	silent	X							
10289C>A	ND6	S>Y				X				
10389C>T	ND6	silent	X						X	
10905A>G	Cyt b	Silent	X							
10925T>G	Cyt b	V>G	X	X	X	X	X	X	X	X
10935T>A	Cyt b	silent	X	X	X	X	X	X	X	X
10952AC>CA	Cyt b	Y>S	X	X	X	X	X	X	X	X
11265C>T	Cyt b	silent	X		X				X	
11279C>A	Cyt b	T>N	X	X	X	X	X	X	X	X
11560T>C	Cyt b	silent	X						X	
11893T>A	ND1	I>F	X	X	X	X	X	X	X	X

11916GG>CC	ND1	P>G	X	X	X	X	X	X		
11987C>A	ND1	L>F	X	X	X	X	X	X	X	X
12060T>G	ND1	N>T	X	X	X	X	X	X	X	X
12062C>A	ND1	silent					X	X		X
12091C>T	ND1	V>M	X						X	
12131T>C	ND1	silent		X	X				X	
12316A>G	ND1	silent		X	X					
12344A>C	ND1	silent							X	
12380C>T	ND1	silent							X	
12626T>C	ND1	silent	X	X	X	X	X	X	X	X
12804T>C	LSU rRNA			X	X	X	X	X		X
13065delA	LSU rRNA			X	X	X	X	X		X
13289T>A	LSU rRNA		X	X	X	X	X	X	X	X
13295delA	LSU rRNA		X	X	X	X	X	X	X	X
13561C>T	LSU rRNA								X	
13587G>A	LSU rRNA							X		X
14698T>C	SSU rRNA			X	X					

Notes:

All numbering based on Refseq (NCBI accession NC_001709). Oregon R sequence from NCBI accession AF200828

New database submissions: *tko*^{25t} original strain, full genome sequence, NCBI accession JQ686693 ; BER1 full genome sequence, NCBI accession JQ686694; Oregon R-C full genome sequence, NCBI accession JQ686698; CO3 coding region sequence, NCBI accession JQ686695;
QI2 coding region sequence, NCBI accession JQ686696; Reids-1 coding region sequence, NCBI accession JQ686697; Canton S coding region sequence, NCBI accession JQ686699

Yellow background denotes strain-specific polymorphisms

Blue background denotes polymorphisms over-represented in suppressor cytoplasms

Grey background denotes polymorphisms over-represented in nonsuppressor cytoplasms

Significant heteroplasmy is excluded, based on the following: our sequencing strategy involved pooling of multiple PCR products, followed by sequencing on both strands using an overlapping primer set. All novel polymorphisms were verified by additional PCR and sequencing. In no case did we find any evidence for heteroplasmy, within the limits of detection by Sanger sequencing (10-15%). Disease-associated heteroplasmy in humans is easily detected by such sequencing, with relative levels of pathological mutant mtDNA in the 50-90% range. Thus we can categorically rule out any significant contribution of heteroplasmy to the *tko*^{25t} phenotype, in any of the strains tested. Moreover, *tko*^{25t} breeds true phenotypically of over many generations in either the suppressor or non-suppressor background, showing standard X-linked recessive inheritance, which would not be expected if the phenotype were subject to influences of heteroplasmy. Conversely, the suppressor itself shows standard maternal inheritance, which would also not be expected if heteroplasmy were a significant factor in suppression.