Supplementary Information

1. Plasmid construction

1) Modification of pAIO-Cas9-yDHOD(-) to remove the preexisting *EcoR*I site.

To switch *BtgZ*I to a more convenient restriction enzyme for cloning gRNAs, we intended to use *EcoR*I. However, an *EcoR*I site was present in pAIO-Cas9-yDHOD(-) upstream of the Cam 5'UTR. To remove it, we digested the plasmid with *Xba*I and *Nco*I (and also with *EcoR*I). A 58 bp synthetic oligo (Cas9RmvEcoAddNhe) with the sequence attttaatttttttttacaaaatgTCTAGAgcatgagaGCTAGCtggcgcctgatgcgg (TCTAGA, *Xba*I; GCTAGC, *Nhe*I) was joined with the digested pAIO-Cas9-yDHOD(-) vector via an NEBuilder DNA Assembly reaction (New England Biolabs). This procedure eliminated the preexisting *EcoR*I, *Nco*I and *Avr*II (adjacent to *Nco*I) sites, regenerated *Xba*I and introduced *Nhe*I, yielding a modified pAIO-Cas9-yDHOD(-) designated pAIO*-Cas9-yDHOD(-).

2) Modification of pAIO*-Cas9-yDHOD(-) to yield M-Cas9-yDHOD(-)

To switch *BtgZ*I to *EcoR*I for cloning gRNAs and to achieve enhanced binding of the sgRNA to Cas9, we modified the gRNA cloning segment in pAIO*-Cas9-yDHOD(-) through a 3-way NEBuilder DNA Assembly reaction. Since there was no restriction site at the end of the gRNA segment, pAIO*-Cas9-yDHOD(-) was digested with *BtgZ*I and *Xho*I, which removed the old gRNA segment, U6 3'UTR and PbDT 3' UTR. The new gRNA segment with new restriction enzymes (*EcoR*I-*Hind*III-*EcoR*I) and extra nucleotides that enhance gRNA binding to Cas9 was synthesized as a 128 bp DNA fragment (ERHdERenhsgRNA), with the sequence

atatttcatattaagtatataatattGAATTCtAAGCTTtGAATTCGTTTcAGAGCTAtgctgGAAAcagcaTAGCAAGTTgAAATA AGGCTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCG . The red nucleotides including c, g, and two 5-base insertions were designed to strengthen a stem structure in the gRNA to enhance its binding to Cas9 (1,2). The segment including the U6 3'UTR and PbDT 3' UTR was amplified with primers P1 and P2 from the original plasmid, pAIO*-Cas9-yDHOD(-). The digested vector, new 128 bp gRNA segment and the PCR product containing the U6 3'UTR and PbDT 3' UTR were then joined with an NEBuilder DNA Assembly reaction. These procedures yielded a new construct named M-Cas9-yDHOD(-).

3) Removal of the Flag tag from M-Cas9-yDHOD(-)

To avoid Flag tag interference in future studies, we removed it from M-Cas9-yDHOD(-)(3) that contained a Cas9-Flag fusion protein. To achieve this, M-Cas9-yDHOD(-) was digested with *Sac*I and *Xho*I to release the entire Flag tag and a C-terminal part of Cas9 (~1.9 kb), due to unavailability of proper restriction sites to release the Flag tag CDS alone. The C-terminal ~1.9 kb part of Cas9 was amplified from the original M-Cas9-yDHOD(-) with primers P3 and P4, which included short homologous sequences matching the ends of the digested vector with *Sac*I/*Xho*I sites. The PCR product and the digested vector were joined via NEB DNA Assembly reactions. Colonies were screened using PCR with primers P5 and P6. Since P6 is in the Flag CDS region, this PCR would give amplicon in the original vector but not with the desired clones which have lost the Flag tag. Therefore, PCR negative colonies were grown up and their plasmid DNAs were sequenced using primer P5 to confirm the loss of Flag tag. These procedures yielded a new pre-gRNA construct without the Flag-tag, namely NF-Cas9-yDHOD(-).

4) gRNA constructs

The Eukaryotic Pathogen CRISPR guide RNA Design Tool (http://grna.ctegd.uga.edu/) was used to search the best gRNA for Cas9. The genomic region of the *P. falciparum* putative mitochondrial ribosomal protein S12 (PfmtRPS12, PF3D7_0412100) is 1011 bp without introns. A fragment between +909 and +1059 (151 bp) was used for searching gRNAs. The genomic region of the *P. falciparum* putative mitochondrial ribosomal protein S17 (PfmtRPS17, PF3D7_1365100) is 649 bp, with two introns (+80 to +241, +330 to +468). A fragment between +582 and +690 (109 bp) was uses for gRNA searching. The genomic region of the *P. falciparum* putative mitochondrial ribosomal protein S18 precursor (PfmtRPS18, PF3D7_1211500) is 1164 bp without introns. A fragment between +1096 and +1381 (286 bp) was analyzed for gRNA searching. Based on high efficiency scores and zero off-target matches, two best gRNAs for each gene, located near the stop codon were chosen to be cloned into the NF-Cas9-yDHOD(-) vector by NEB DNA Assembly reactions. Briefly, the vector was linearized by *EcoR*I overnight and gel purified. The synthetic oligoes (P7-P12) are 60 bp in length, containing a 20 or 21 bp gRNA (N20 /N21) sequence flanked by homologous sequences on both sides that match the ends of the vector digested by *EcoR*I. These linear 60 bp oligos and the linearized vector were individually joined by NEB DNA Assembly reactions, yielding gRNA expressing plasmids.

The N21or N20 of each gRNA (P13-P18) and a vector primer (P19) were used to perform diagnostic PCR during cloning. Another oligo (P20), 75 bp downstream of *EcoR*I site, was used to sequence the cloned gRNA.

5) Cloning template constructs for conditional knockdown studies

In the modified pMG75 vector as described previously (4), we cloned two homologous regions (3'HR and 5'HR) of each SSU gene via NEB DNA Assembly reactions, yielding three new template vectors, pMG75noP-PfmtRPS12-8apt-3HA, pMG75noP-PfmtRPS17-8apt-3HA, and pMG75noP-PfmtRPS18-8apt-3HA. The original pMG75noP-ATP4-8apt-3HA was linearized by *Afl*II and *BstE*II, to remove the ATP4 inserts. To make pMG75noP-PfmtRPS12-8apt-3HA, the 3'HR (680 bp) was immediately downstream of the stop codon and was amplified from *P. falciparum* genomic DNA via primers (P21+ P22), which harbors a 27 bp sequence homologous to the segment immediately upstream of the *Afl*II site on the vector, and a 20 bp sequence homologous to the upstream part of 5'HR. The 5'HR (659 bp) was amplified via primers (P23+ P24), which harbors a 20 bp sequence homologous to the downstream part of 3'HR and a 29 bp sequence homologous to the segment immediately downstream of the *BstE*II site on the vector. Since the gRNAs of PfmtRPS12 were located at the end of 5'HR, to avoid repetitive cutting by Cas9 in the expected transgenic parasites, the reverse primer of the 5'HR (P24) included synonymous mutations within the gRNA region. The linearized vector, the 3'HR and the 5'HR were then joined together by NEB DNA Assembly reactions. One oligo (P25), 111 bp downstream of *BstE*II site of the vector, was used to sequence the cloned 5'HR, another oligo (P26), 73 bp upstream of *Afl*II site of the vector, was used to sequence the cloned 3'HR. A similar three-piece DNA assembly strategy was used to make the other two constructs. For pMG75noP-PfmtRPS17-8apt-3HA, the 3'HR (656 bp) was amplified via P27+P28, the 5'HR (642 bp) was amplified via P29+P30. For pMG75noP-PfmtRPS18-8apt-3HA, the 3'HR (761 bp) was amplified via P31+P32, the 5'HR (683 bp) was amplified via P33+P34. The 5'HR reverse primer of PfmtRPS18 (P34) did not include any synonymous mutations since the gRNAs were located downstream of the stop codon, not in the homologous regions of 5'HR or 3'HR.

All primers and oligos were purchased from Eurofins Genomics (Supplementary Table S1). All infusion cloning steps in this study were done by NEBuilder® HiFi DNA Assembly. DNA fragments used for cloning were amplified with high fidelity DNA polymerases (New England Biolabs®, Inc) and confirmed by sequencing (Genewiz LLC). All restriction endonucleases and DNA modifying enzymes were from New England Biolabs®, Inc. Cloning steps involving pMG75-derived vectors were transformed into stable competent *E. coli* (New England Biolabs®, Inc) and bacteria were grown at 30 ℃. Cloning steps involving pAIO-derived gRNA constructs were transformed into DH5-alpha Electrocompetent *E. coli* (New England Biolabs®, Inc) and bacteria were grown at 37 ℃.

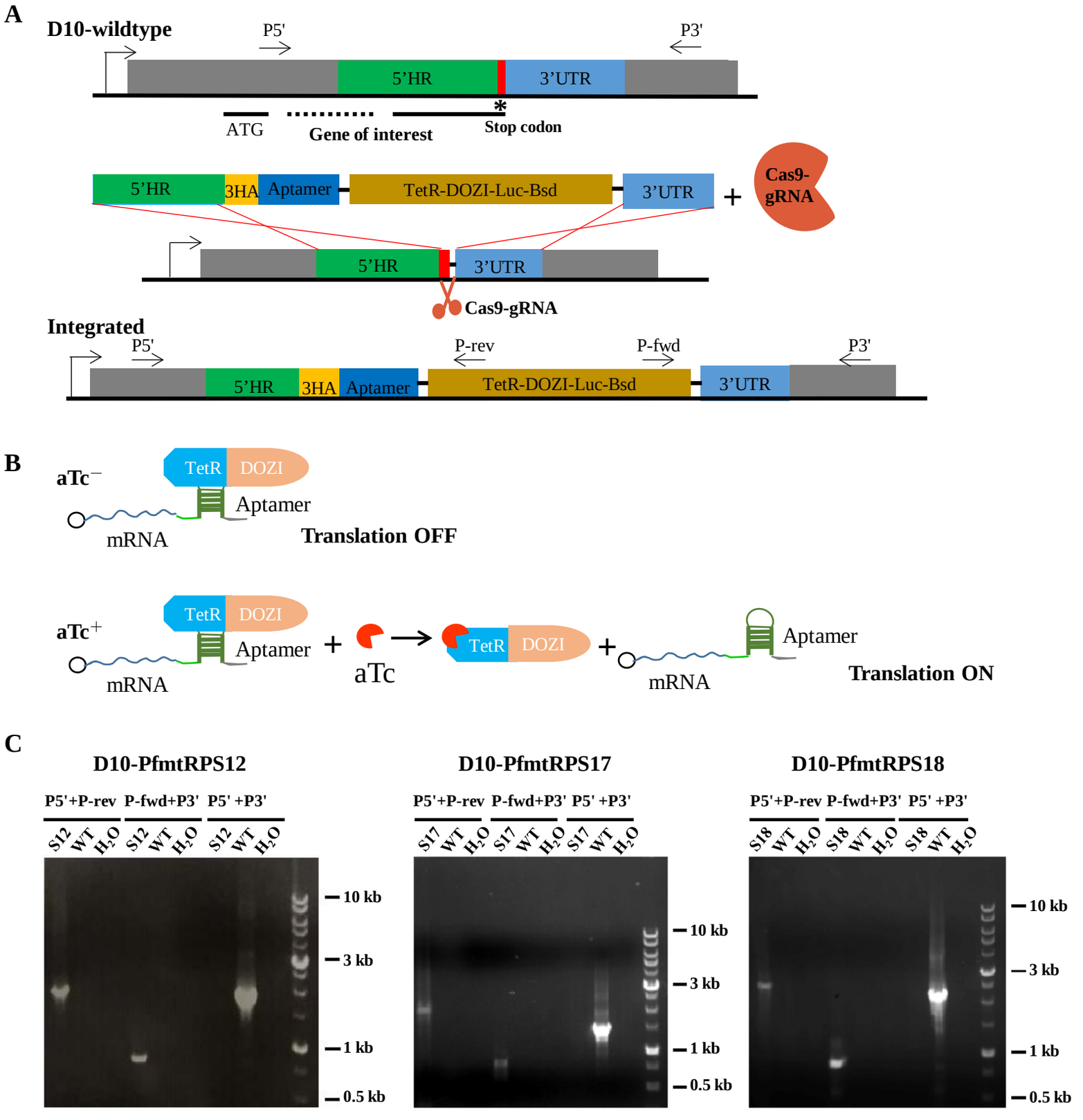
2. Drug sensitivity assay (The malaria SYBR Green I-based fluorescence, MSF)

All compounds tested were dissolved in DSMO to make concentrated stocks (mM). Drugs were diluted in 96-well culture plates (rows, A-H × columns 1-12) starting from the second column. The highest concentration of each drug was as following: 333 μM for proguanil, 33333 nM for chloroquine, 6667 nM for artemisinin, ELQ-300, HDQ or CK-2-68, 3333 nM for DSM-265, 333 nM for atovaquone. From the second column, drugs were diluted by 3-fold serial dilutions across 10 columns (2 to 11) in aTc^+ or aTc^- medium. The first column was loaded with uninfected RBCs in the absence of drugs, which served as a negative control. The last column was loaded with infected RBCs (0.5% ring stage) without drugs, which served as a positive control. Wells in columns 2 to 11 were loaded with infected RBCs (0.5% ring stage). For each well (uninfected or infected), the total volume was 200 μL of RBC suspension at 2% hematocrit. The culture plates were incubated for 3 days (72 h) at regular culture conditions and immediately frozen at −80 °C until analysis. The plates were thawed at 37 ℃ for 1 h, added 150 μL of lysis buffer containing SYBR Green directly to the wells, followed by gentle mixing and incubated at room temperature in dark for 2-3 h. The plates were examined for the relative fluorescence units (RFU) using the fluorescence plate reader (Tecan Infinite F Plex). The average RFU of all wells in column 12 of aTc^+ plates was considered as the highest RFU, maxi RFU; and the average RFU of all wells in column 1 of all plates was considered as the lowest RFU, mini RFU. The percentage growth of other wells (Y) from column 2 to column 11, was calculated by using Y_i = (Y RFU - mini RFU) / (maxi RFU - mini RFU) × 100%. The compound concentrations (X values) were transformed by using X_i = Log[X_i] and plotted against the growth percentage (Y values). The data was then analyzed in GraphPad Prism 6 (GraphPad Software, Inc., San Diego, CA) by nonlinear regression (sigmoidal dose-response/variable slope equation) to yield the IC50.

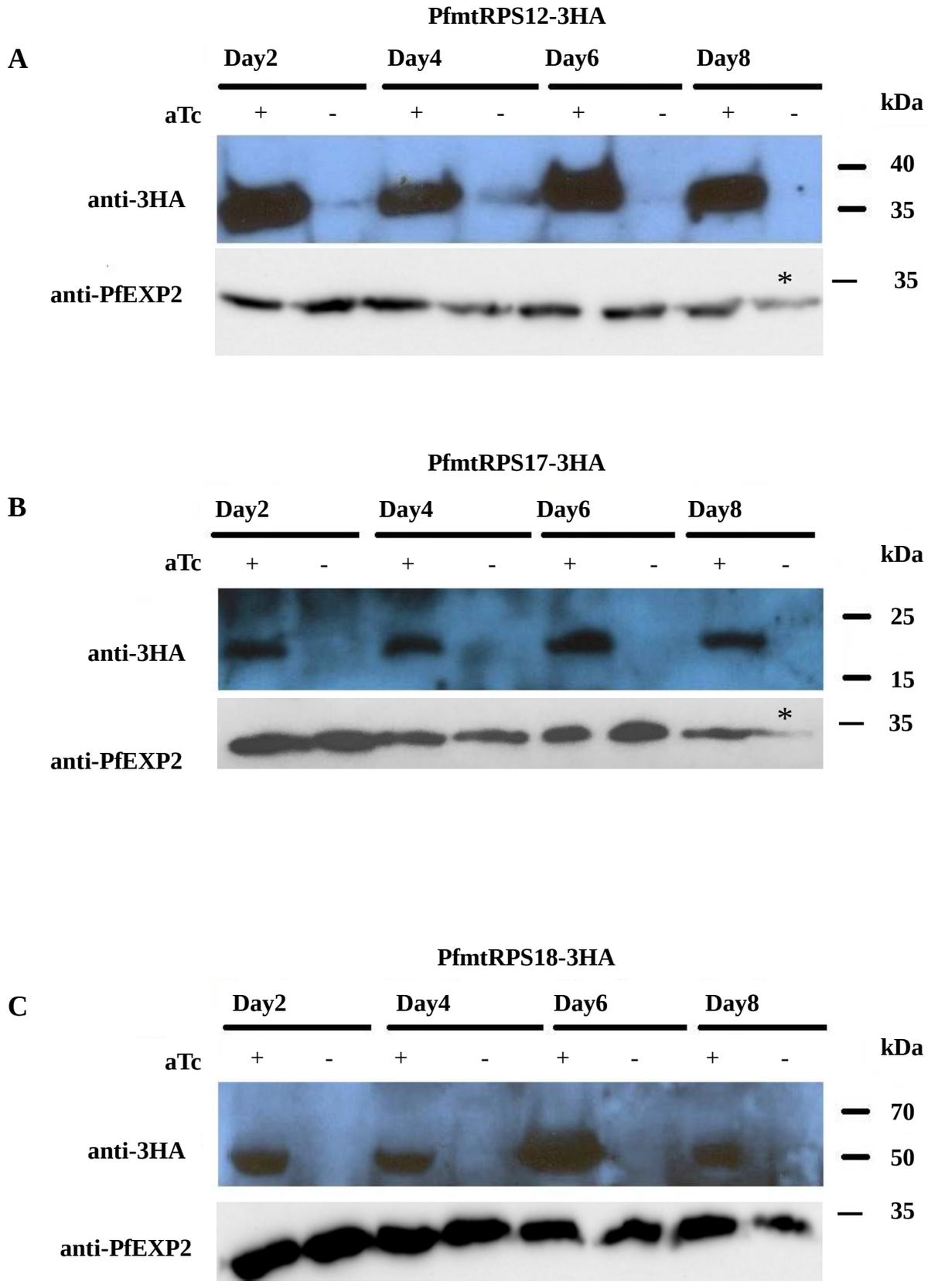
3. Metabolomic profiling of D10-PfmRPS17

D10-PfmRPS17 parasites were tightly synchronized with alanine and expanded to 2-4 T175 flasks in aTc^+ medium. Trophozoite / schizont stages were isolated using a percoll gradient (89428-524, GE Healthcare Life Sciences). The highly enriched parasites were washed 3 times with regular medium and split into two cultures maintained with or without aTc. Each flask contained ~140 µL of purified parasites supplemented with 160 mL of medium and 4 mL of fresh RBC, leading to a culture of 1.25% hematocrit and 7% parasitemia. Typically, the parasitemia reached to 20-25% in both aTc^+ and aTc^- conditions after 24 h. Starting at this timepoint and in intervals of 12 h for 7 timepoints in total, a portion of the culture was removed from both conditions and metabolites were extracted following the established protocol(5). Briefly, at timepoints of 24, 36, and 48 h, an aliquot of 150 µL of packed infected RBCs was removed from aTc^+ and aTc^- flasks, resuspended in prechilled PBS, and equally split into 3 microcentrifuge tubes to obtain 3 technical replicate samples for metabolite extraction. After collecting metabolites from these timepoints (24, 36, and 48 h), the aTc^+ culture was supplemented with fresh blood and medium to reach 1.25% hematocrit and 7% parasitemia. To prevent growth defects observed after removal of aTc for longer than 48 h, the aTc^- culture was supplemented with a lower amount of blood to yield a culture with the same hematocrit at 1.25% but a higher parasitemia at 10%. At timepoints of 60, 72, 84 and 96 h, parasites were collected, and metabolites were extracted as described above. For samples at all timepoints, thin blood smears were made to monitor parasitemia to ensure the number of parasites collected from aTc^+ and aTc^- conditions were equal. In aTc^+ condition at the 96 h time point, a portion of the culture (150 µL of packed infected RBCs) was treated with 10 nM atovaquone for 4 h followed by metabolite extraction in triplicate. A 150 µL aliquot of packed uninfected RBCs was also extracted in triplicate for metabolite analysis. All metabolite samples were dried by a nitrogen flow and resuspended in 100 µL of 3:97 methanol:water containing 0.25 µM chlorpropamide as an internal standard. A pooled sample was generated by combining 5 µL of each sample. These samples were run using a reverse-phase HPLC method using a Waters Xselect HSS T3 C18 column with 2.5 µm particle diameter. The mobile phase conditions were composed of 3% methanol, 15 mM acetic acid, and 10 mM tributylamine (mobile phase A) and 100% methanol (mobile phase B). The chromatography was coupled to a Thermo Exactive Plus orbitrap operated in negative mode scanning from 85 to 1000 m/z. The injection volume was 10 µL and the pooled sample was injected three times to determine technical compound variation. Other details of metabolite extraction and analysis are available in the previously published work (5). For each metabolite detected, a fold change of the metabolite is determined by dividing the metabolic intensity in aTc^- samples by that in aTc^+ samples.

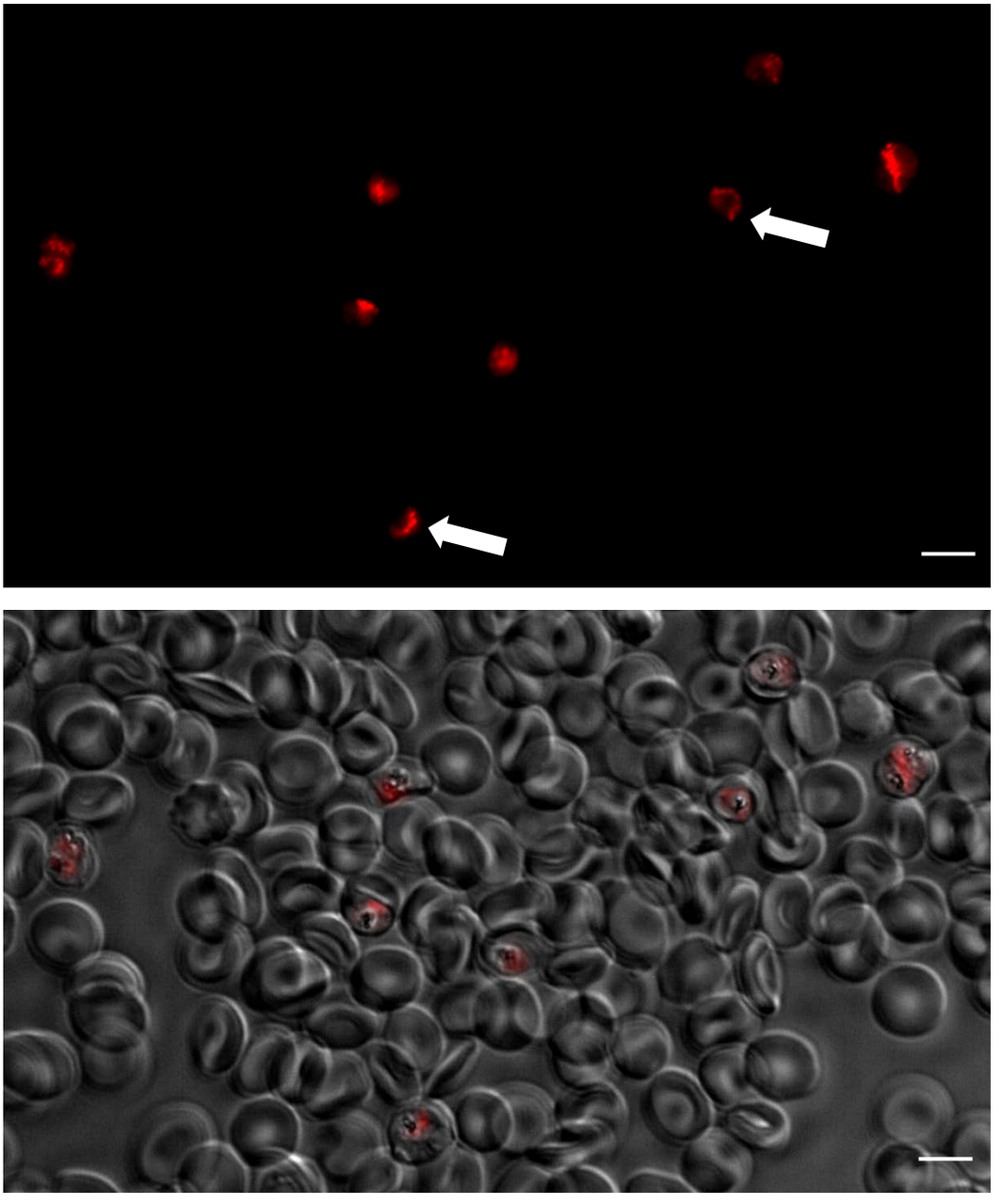
1. Dang, Y., Jia, G., Choi, J., Ma, H., Anaya, E., Ye, C., Shankar, P., and Wu, H. (2015) Optimizing sgRNA structure to improve CRISPR-Cas9 knockout efficiency. *Genome Biol* **16**, 280
2. Chen, B., Gilbert, L. A., Cimini, B. A., Schnitzbauer, J., Zhang, W., Li, G. W., Park, J., Blackburn, E. H., Weissman, J. S., Qi, L. S., and Huang, B. (2013) Dynamic imaging of genomic loci in living human cells by an optimized CRISPR/Cas system. *Cell* **155**, 1479-1491
3. Ke, H., Ganesan, S. M., Dass, S., Morrisey, J. M., Pou, S., Nilsen, A., Riscoe, M. K., Mather, M. W., and Vaidya, A. B. (2019) Mitochondrial type II NADH dehydrogenase of Plasmodium falciparum (PfNDH2) is dispensable in the asexual blood stages. *PLoS One* **14**, e0214023
4. Ke, H., Dass, S., Morrisey, J. M., Mather, M. W., and Vaidya, A. B. (2018) The mitochondrial ribosomal protein L13 is critical for the structural and functional integrity of the mitochondrion in Plasmodium falciparum. *J Biol Chem* **293**, 8128-8137
5. Allman, E. L., Painter, H. J., Samra, J., Carrasquilla, M., and Llinas, M. (2016) Metabolomic Profiling of the Malaria Box Reveals Antimalarial Target Pathways. *Antimicrob Agents Chemother* **60**, 6635-6649



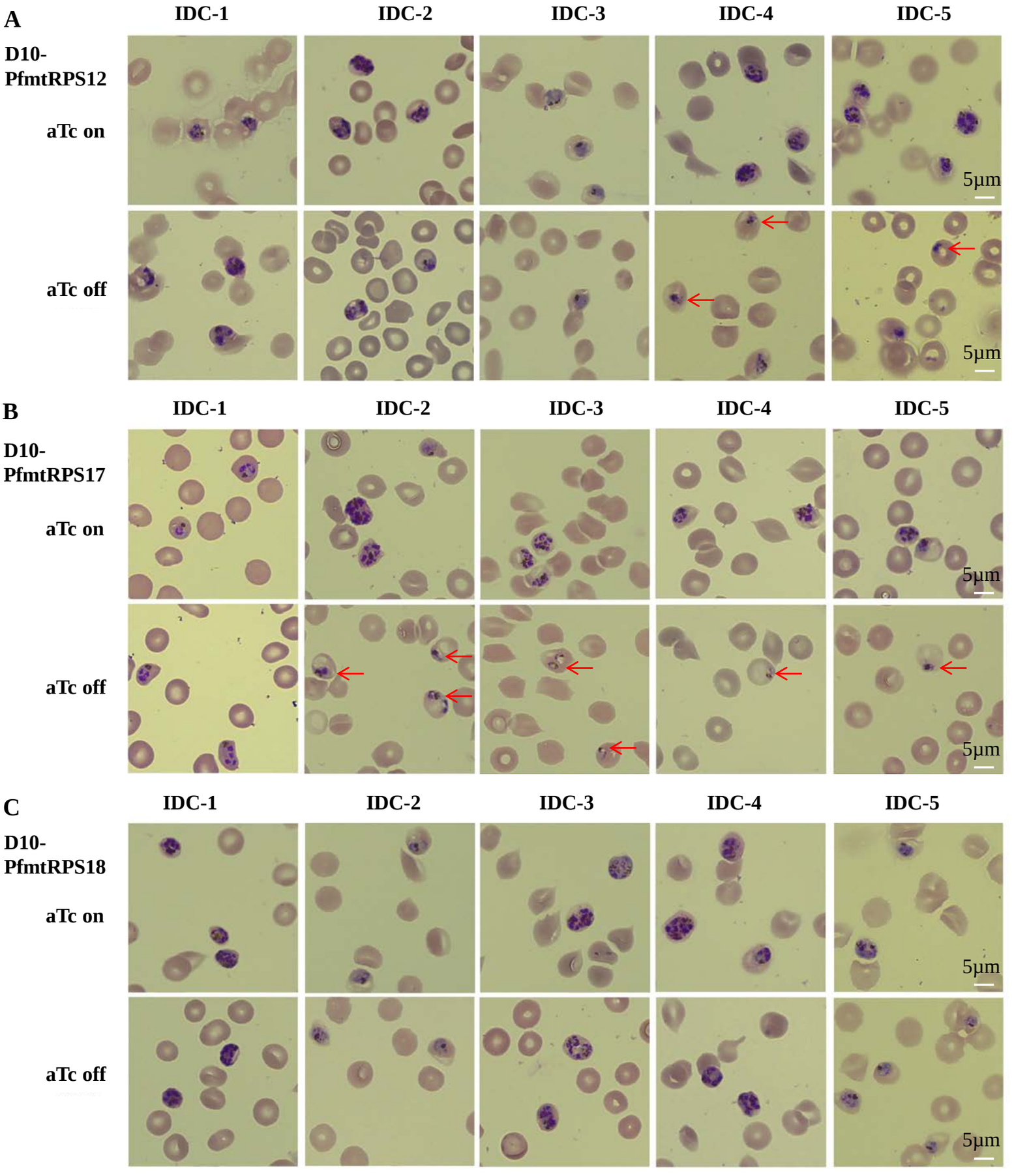
Supplementary Figure 1. Genetic tagging PfmtRPS12, PfmtRPS17 and PfmtRPS18 via CRIPSR/Cas9.
(A) A schematic diagram of genetic tagging PfmtRPS12, PfmtRPS17, and PfmtRPS18 with 3HA and aptamers at the C-terminus. The gRNAs were located near the end of target gene locus and were cloned into NF-Cas9-yDHOD(-). (B) A diagram showing how translation of the tagged mRNA is conditionally regulated by addition or removal of aTc. (C) Diagnostic PCRs showing the correct genotype of individual transgenic parasite line. Primer positions are demonstrated in (A). P5' and P-rev are used to check 5' integration whereas P-fwd and P3' are used to check 3' integration. P5' and P3' only work for D10-WT DNA since the integrated DNA was too long to be amplified (> 10 kb). H_2O was used as negative control in PCR reactions. P5' and P3' are P37 and P38 for D10-PfmRP-S12, or P39 and P40 for D10-PfmRP-S17, or P41 and P42 for D10-PfmRP-S18 (see Table S1).



Supplementary Figure 2. Knockdown efficiency was confirmed by Western blot.
Expression of PfmtRPS12-3HA (A), PfmtRPS17-3HA (B), or PfmtRPS18-3HA (C) in the knockdown parasites was examined by Western blot. PfEXP2, exported protein 2 (~33 kDa), served as a loading control. In both PfmtRPS12-HA and PfmtRPS17-HA lines, when aTc was removed for 8 days, the parasitemia dropped so expression levels of PfEXP2 also decreased in comparison to those of aTc plus controls (black stars).

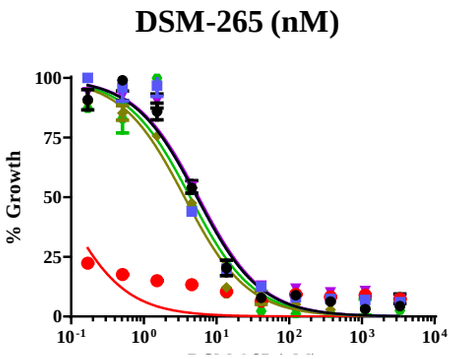


Supplementary Figure 3. The mitochondrial morphologies of D10 wildtype parasites after fixation.
The D10 wildtype parasites were pre-labelled with 60 nM mitoTracker for 30 min then fixed with 4% paraformaldehyde /0.0075% glutaraldehyde for 1 h at 37ºC . The mitochondria exhibited different morphologies, some appeared “fragmented” (white arrows). Bar, 5 µm.

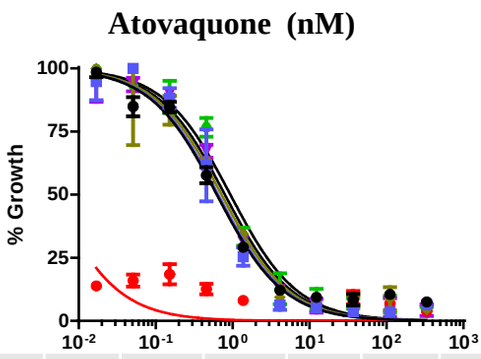


Supplementary Figure 4. Morphological changes of SSU knockdown parasites on Giemsa stained smears.

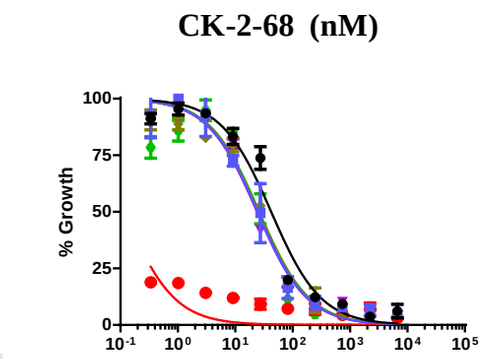
(A) Knockdown of PfmtRPS12. Red arrows indicate PfmtRPS17 knockdown parasites were sick as compared to control parasites. (B) Knockdown of PfmtRPS17. Red arrows indicate PfmtRPS17 knockdown parasites were sick as compared to control parasites. Red arrows indicate morphologically sick parasites.
(C) Knockdown of PfmtRPS18.



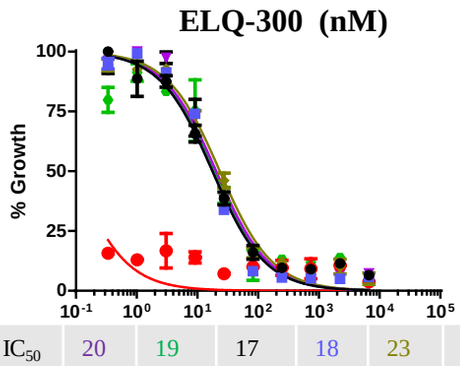
IC_{50}	5.7	5.3	5.4	5.3	3.6	0.1



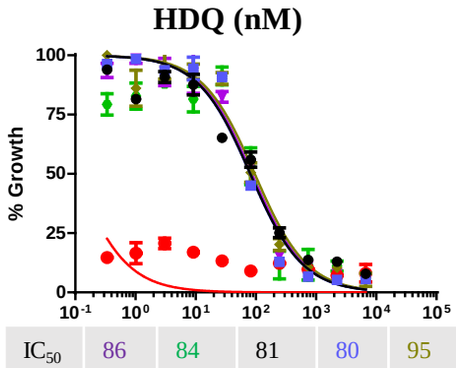
IC_{50}	0.8	0.9	0.6	0.6	0.7	0.0 05



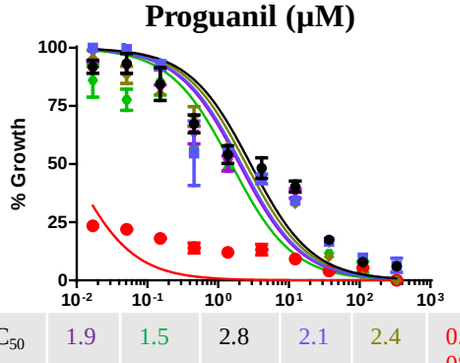
IC_{50}	24	26	29	24	25	0.1



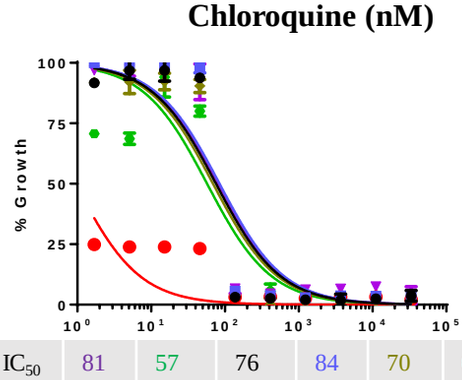
IC_{50}	20	19	17	18	23	0.1



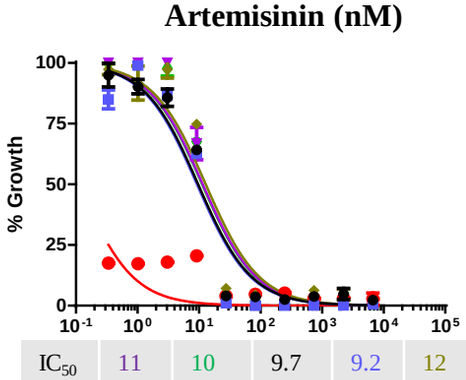
IC_{50}	86	84	81	80	95	0.1



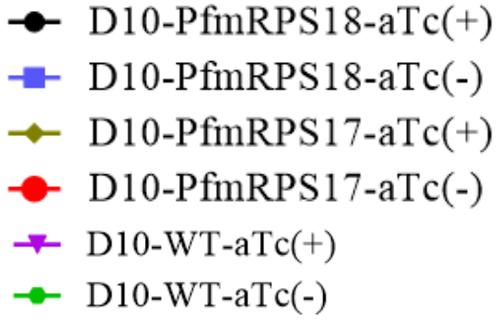
IC_{50}	1.9	1.5	2.8	2.1	2.4	0.0 08



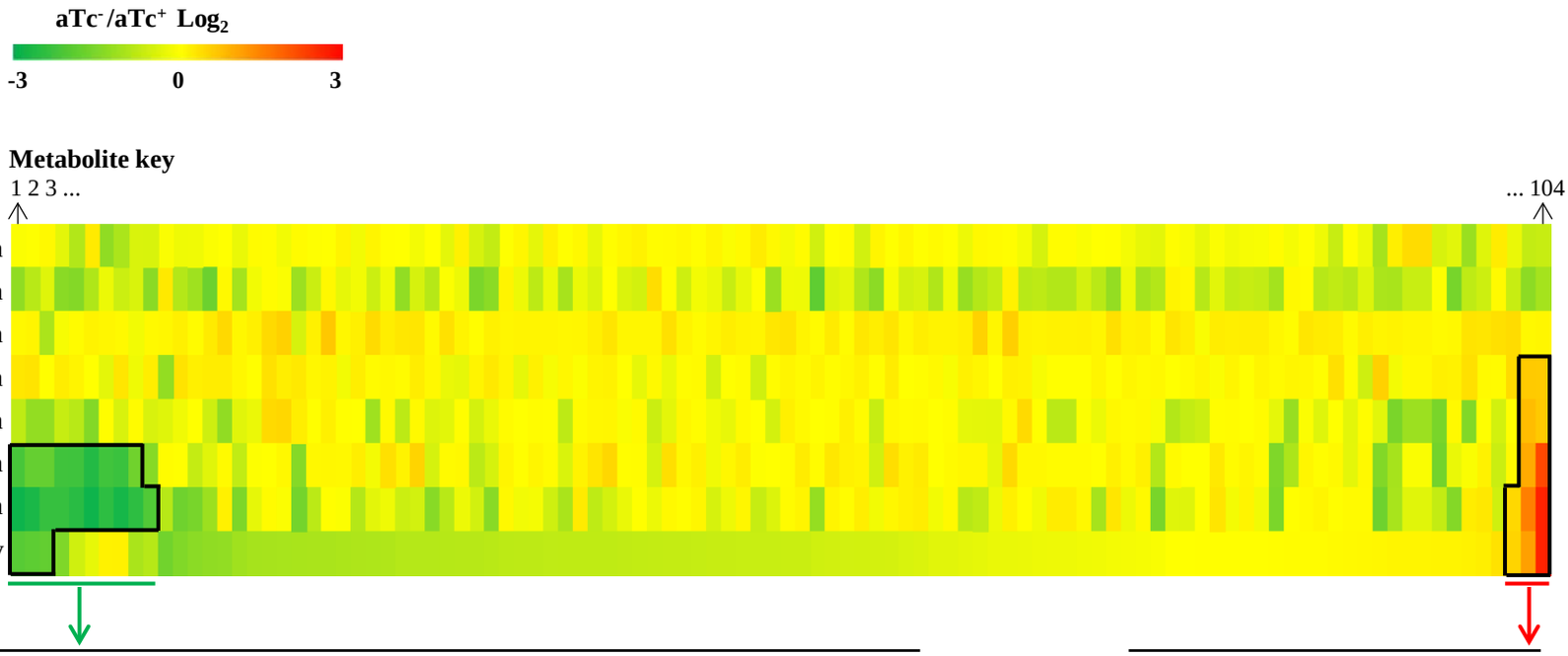
IC_{50}	81	57	76	84	70	0.9



IC_{50}	11	10	9.7	9.2	12	0.1



Supplementary Figure 5. Responses to antimalarial drugs in PfmtRPS17 and PfmtRPS18 knockdown parasites. Knockdown of PfmtRPS17 renders the parasite become hypersensitive to drugs targeting mitochondrial functions (DSM-265, atovaquone, CK-2-68, ELQ-300, HDQ, proguanil) and other pathways (chloroquine and artemisinin). Knockdown PfmtRPS18 did not render the parasites become hypersensitive to drugs tested in this study. Data shown are the mean± s.d. of duplicates and are representative of n = 3 independent expriments.



1	dTDP	6	dCDP			102	Xanthosine-5-phosphate
2	CDP	7	dCMP			103	N-carbamoyl-L-aspartate
3	Citrate/isocitrate	8	dTMP	...	...	104	Dihydroorotate
4	Succinate/Methylmalonic acid	9	CMP				
5	UMP	10	4-phosphopantothenate				

Supplementary Figure 6. Profiling of 104 metabolites in the PfmtRPS17 knockdown parasites in a time-dependent manner.

In total, 104 metabolites were detected in this study. Among them, the levels of ten metabolites decreased by more than 50% in aTc⁻ compared to aTc⁺ parasites (shown in the left Table) and three of them (dTDP, CDP, citrate/isocitrate) also showed > 2-fold decrease in atovaquone treated parasites compared to untreated ones. At the timepoint of 96 hrs, the levels of three metabolites increased more than 2-fold in aTc⁻ compared to aTc⁺ parasites (shown in the right Table). All of these metabolites also accumulated in atovaquone treated samples. The other 91 metabolites oscillated throughout the time-course but their abundance did not increase or decrease more than 2-fold in PfmtRPS17 knockdown or atovaquone treated samples. Data shown are the logistic mean of 3 independent expriments. The metabolites from left to right are listed as following: 1, dTDP; 2, CDP; 3, Citrate/isocitrate; 4, Succinate/Methylmalonic acid; 5, UMP; 6, dCDP; 7, dCMP; 8, dTMP; 9, CMP; 10, 4-phosphopantothenate; 11, adenine; 12, GDP; 13, cyclic-AMP; 14, 4-phosphopantetheine; 15, sedoheptulose bisphosphate; 16, UDP-N-acetyl-glucosamine; 17, N-acetyl-glutamine; 18, octulose bisphosphate; 19, phosphoenolpyruvate; 20, CDP-ethanolamine; 21, UDP-D-glucose; 22, fructose-1-6-bisphosphate; 23, glutamine; 24, dGDP/ADP; 25, trehalose/sucrose; 26, D-glucarate; 27, NAD^+; 28, $NADP^+$; 29, α-ketoglutarate; 30, glutathione disulfide; 31, serine; 32, xanthosine; 33, folate; 34, 4-aminobutyrate; 35, alanine/sarcosine; 36, N-acetyl-glutamate; 37, asparagine; 38, N-Acetyl-L-alanine; 39, N-acetyl-glucosamine; 40, FAD; 41, ADP-D-glucose; 42, hydroxyproline/aminolevulinate; 43, threonine/homoserine; 44, chlorpropamide; 45, NADH; 46, ribose; 47, glutamate; 48, citrulline; 49, acetyl-aspartate; 50, citraconic acid; 51, taurine; 52, histidine; 53, 3-phosphoglycerate; 54, 2-hydroxy-2-methylbutanedioic acid; 55, GMP; 56, 2-oxo-4-methylthiobutanoate; 57, cytidine; 58, 2-dehydro-D-gluconate; 59, nicotinamide mononucleotide; 60, 6-phospho-D-gluconate; 61, 1-methyladenosine; 62, pantothenate; 63, arginine; 64, D-gluconate; 65, hypoxanthine; 66, D-glucosamine-1/6-phosphate; 67, proline; 68, sn-glycerol-3-phosphate; 69, phenylalanine; 70, 4-hydroxyphenyllactate; 71, 5-aminopentanoic acid; 72, valine; 73, tyrosine; 74, aconitate; 75, glycerate; 76, fumarate; 77, L-arginino-succinate; 78, dGMP/AMP; 79, indole; 80, tryptophan; 81, thiamine; 82, methionine; 83, maleic acid; 84, isoleucine; 85, malate; 86, IMP; 87, lactate; 88, N-acetyl-glucosamine-1/6-phosphate; 89, leucine; 90, nicotinate; 91, glutathione; 92, lysine; 93, dAMP; 94, DL-pipecolic acid; 95, fructose-6-phosphate; 96, glucose-1-phosphate; 97, pyruvate; 98, guanine; 99, riboflavin; 100, pyridoxine; 101, pyroglutamic acid; 102, Xanthosine-5-phosphate; 103, N-carbamoyl-L-aspartate; 104, Dihydroorotate.

Supplementary Table 1. Primers used in the study

	primer name	Sequence
P1	U6-PbDT3prFwd	GAAAAAGTGGCACCGAGTCG
P2	U6-PbDT3prRev	TACAAGGACGACGACGACAAGTAACTCGAGGGATATGGCAGCTTAATGTTC
P3	1.9kbCas9-F	GTGAAGGTGGTGGACGAGCTC
P4	1.9kbCas9-R	GAAAAACGAACATTAAGCTGCCATATCCCTCGAGTTACACTTTCCGCTTTTTCTTAGGATCTTCC
P5	Cas9end-F	GATCCTGGCCGACGCTAATC
P6	Flag-rev	CTTGTCGTCGTCGTCCTTGTA
P7	S12gRNA1	CATATTAAGTATATAATATTGTCCTGTAGTAAGTATTAAAAGTTTCAGAGCTATGCTGGA
P8	S12gRNA2	CATATTAAGTATATAATATTGTTAAAATGGAAGATATTAATGTTTCAGAGCTATGCTGGA
P9	S17gRNA1	CATATTAAGTATATAATATTGAAGGAAAATAAAGATTAAGCGTTTCAGAGCTATGCTGGA
P10	S17gRNA2	CATATTAAGTATATAATATTGCCTTGTATAAAATTTTTACAGTTTCAGAGCTATGCTGGA
P11	S18gRNA1	CATATTAAGTATATAATATTATCATATGAAAAAATCAGTGGTTTCAGAGCTATGCTGGAA
P12	S18gRNA2	CATATTAAGTATATAATATTATATCATATGAAAAAATCAGGTTTCAGAGCTATGCTGGAA
P13	S12gRNA1N21	GTCCTGTAGTAAGTATTAAAA
P14	S12gRNA2N21	GTTAAAATGGAAGATATTAAT
P15	S17gRNA1N21	GAAGGAAAATAAAGATTAAGC
P16	S17gRNA2N21	GCCTTGTATAAAATTTTTACA
P17	S18gRNA1N20	ATCATATGAAAAAATCAGTG
P18	S18gRNA2N20	ATATCATATGAAAAAATCAG
P19	N20CheckR	ATATGAATTACAAATATTGCATAAAGA
P20	ReverseOligo	TAGGAAATAATAAAAAAGCACCGACTCG
P21	S12-3UTRFwd	ATGGCCCCTTTCCGGGCGCGCCTTAAGATTTATGTATATTTGTGTATGTATG
P22	S12-3UTRRev	TCCGGAGATATCCCGCGGGTATTTTCCTTTCTTTTGCCTAAG
P23	S12-5HRFwd	ACCCGCGGGATATCTCCGGAGTACACATACATCTTATCATTCC
P24	S12-5HRRev	AAAATGTTTATCAAACCGGGGGTAACCTGGGCATCACTATTTTGTTGAAATTTTTTATTTCTATAATAT GTGTTGAAGTGAAATATGTTTATTGGTACATTTTCTTCAGGGTCT
P25	PMG75BBHA-rev	TGGGCCCGAATTCTCATCATTGTGC
P26	PMG75seqF	CTTTAAATTCATGCAAAAATTTAC
P27	S17-3UTRFwd	ATGGCCCCTTTCCGGGCGCGCCTTAAGGCAGGAAAGATATCCATATGTG
P28	S17-3UTRRev	TCCGGAGATATCCCGCGGAACAAGGATTGTGACGTACG
P29	S17-5HRFwd	TTCCGCGGGATATCTCCGGATAAGGCTAAACAAAACTTATTC
P30	S17-5HRRev	AAAATGTTTATCAAACCGGGGGTAACCTGGTCCTTGTTCTCTTTATACAATATCTTAACTAGGATATA ATTTTTCCAAGGTCCA
P31	S18-3UTRFwd	ATGGCCCCTTTCCGGGCGCGCCTTAAGCTTCACCCATATGTTGTTCTC
P32	S18-3UTRRev	TCCGGAGATATCCCGCGGTTATTATGGTTCCTCCCAGAA
P33	S18-5HRFwd	AACCGCGGGATATCTCCGGAAAATGGCAAGTATTGTTGAAT
P34	S18-5HRRev	AAAATGTTTATCAAACCGGGGGTAACCTGATATTTATAAAAATTTTCATAGTAATTTTTC
P35	3'TetRCheck	ATATTTCATGTCTCAGTAAAGTCTTTCAATAC
P36	PMG75seqF	CTTTAAATTCATGCAAAAATTTAC
P37	S12-FAvrII	TACCTAGGATGTTTTTTACCTTATCCAATAAGTTTAG
P38	S12-3fout	TCCTTATATTTTTCTGCCTTCTTGT
P39	S17-5fout	AGATTTATGGCTTATGGGAA
P40	S17-3fout	GATAAAGATGGTCATGAATACT
P41	S18-FSpeI	TCACTAGTATGATTGTTTTGTTAAGAAAAAATAATTTAAAAAAC
P42	S18-3fout	TTTACCCAATTGATTTATCAC