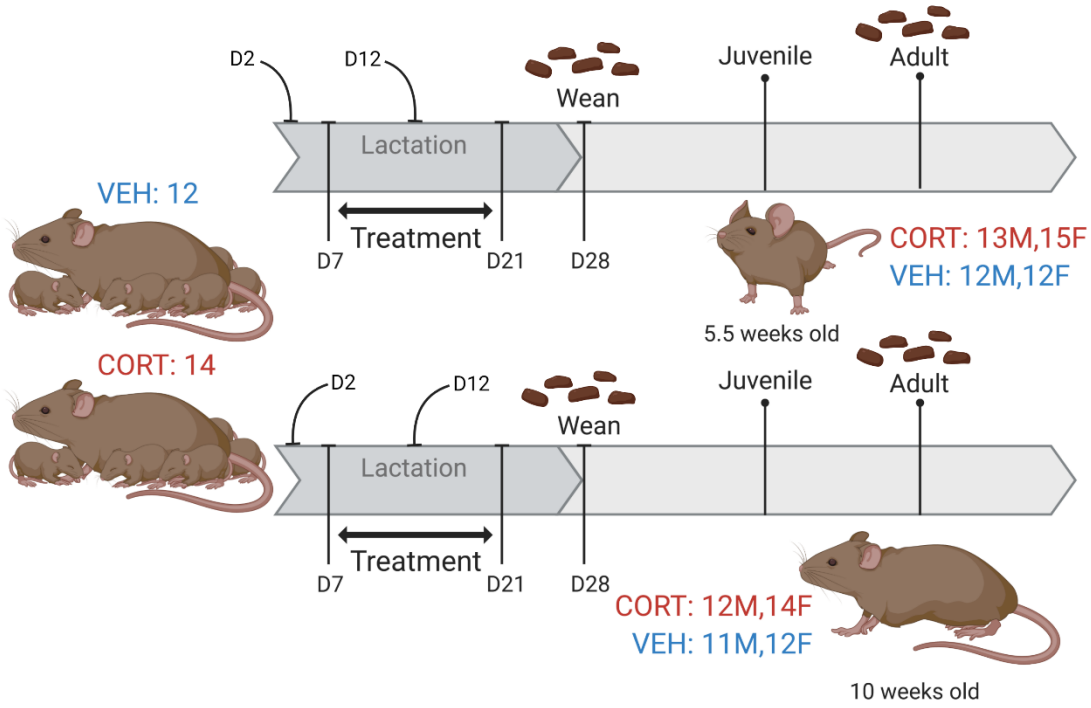

Fig. S1. Experimental timeline of vehicle (VEH: 12) and corticosterone-treated (CORT: 14) reproducing dams, as well as their Juvenile (CORT: 13M, 15F; VEH: 12M, 12F) and Adult (CORT: 12M, 14F; VEH: 11M, 12F) offspring. Units are indicated as days post parturition (D#). Litter mass was taken at day 2 post-partum (D2), day 12 post-partum (D12) and weaning (D28). Body mass of dams were measured at sacrifice (10 days post-weaning). Juvenile (5.5 weeks) and Adult (10 weeks) offspring mass were also measured at sacrifice. Fecal samples were collected on the day of sacrifice from dams at weaning (D28) and Adult offspring (10 weeks).

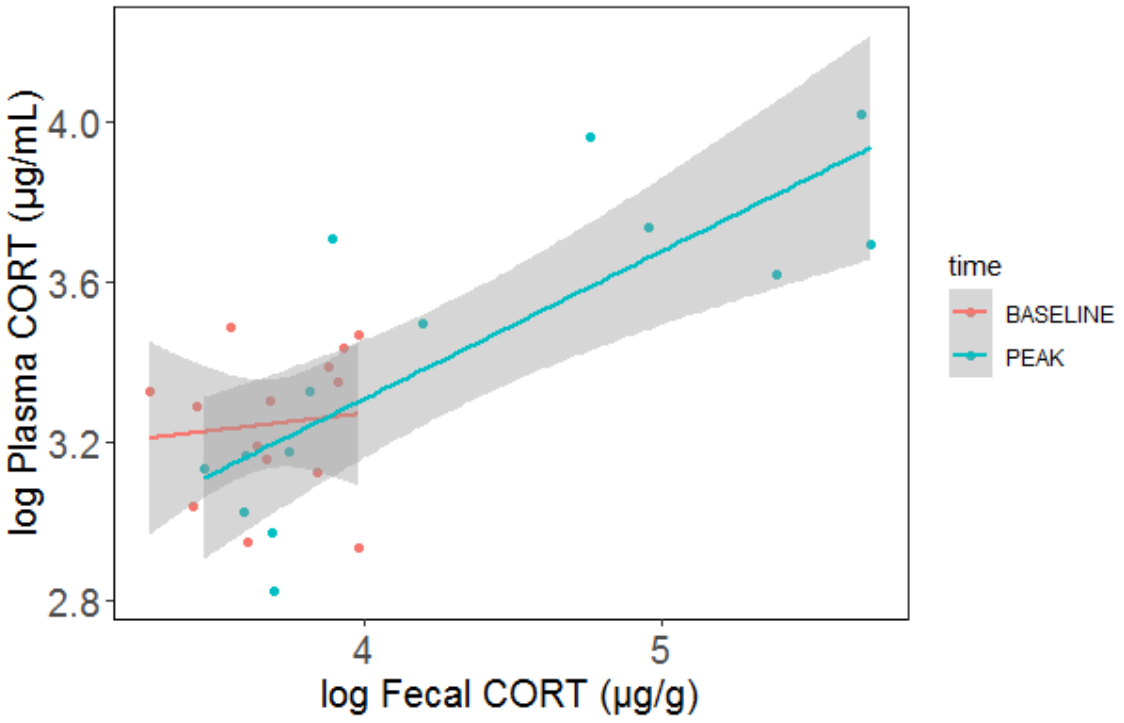

Fig. S2. To confirm that fecal CORT metabolites were correlated with circulating CORT, a separate validation study, 14 female mice were evenly divided into VEH (n=7) and CORT (n=7) groups. Experimental females were bred and given the exact same CORT treatment described above starting on day 7 post-partum. A blood sample of approximately 70-100 µl was collected from each mouse on day 12 post-partum from the facial vein (Hoff, 2000). Once blood was obtained, each female was immediately returned to her pups in a clean box. Feces were then collected from the box the next morning, approximately 12 hours later. The CORT content of these blood and fecal samples were analyzed as described above, except plasma was extracted from blood samples through a single centrifugation event. Dissociation reagent (5 µl) from the ELISA kit was vortexed gently with plasma (5 µl) and incubated at room temperature for 5 minutes. Samples were then diluted 1:100 with 490 µl of assay buffer before plate loading. Our results showed a positive correlation between corticosterone (CORT) metabolites (pg/g) found in both plasma and fecal samples of reproducing female mice (Pearson correlation, $r=0.55$, $p<0.001$), when baseline and peak lactation stages were combined. When stages were assessed separately, there was a positive correlation between corticosterone (CORT) metabolites (pg/g) found in both plasma and fecal samples during peak lactation (Pearson correlation, $r=0.80$, $p<0.001$) but not during baseline (Pearson correlation, $r=0.10$, $p>0.05$).

Table S1. General linear model comparisons of body mass, fecal corticosterone, and litter mass (D2, D12, D28 post-partum) in corticosterone-treated (CORT) or vehicle (VEH) dams. Data shown are least-squared means ± s.e. and units are indicated as days post parturition (D#). Fecal corticosterone metabolites (µg/gm fecal solid) across baseline (before pairing with male), peak lactation (D12 post-partum), and weaning (D28 post-partum) timepoints. Death date and mass were included as covariates and interactions between groups were explored. The p-values are given for all comparisons. Significant findings are reported in bold.

	VEH	**CORT**		**Statistics**			
	$\bar{x}$**+se**	$\bar{x}$**+se**	**num DF**	**den DF**	**Main effect/Int-eraction**	**F-value**	**p-value**
Body mass (g)	20.5±0.61	21.7±0.56	1	24	Group	-1.43	0.17
Fecal CORT metabolites (µg/g)	B: 0.155±0.32 P: 0.0514±0.31 W: 0.118±0.31	B: 0.122±0.29 P: 2.62±0.30 W: 0.104±0.30	2	45	**Group xTime**	12.48	**<0.0001**
D2 Litter mass (g)	8.11±0.47	7.82±0.43	1	24	Group	0.42	0.68
D12 Litter mass (g)	29.9±1.8	27.7±1.6	1	24	**Group**	0.83	0.42
D28 Litter mass (g)*	68.1±1.7	59.3±1.5	1	16	**Group**	3.68	**0.003**

For each model: parent = random factor; B = baseline; P = peak; W = weanling
*Dams with final litter size < 5 pups removed

Table S2. General linear model comparisons of mitochondrial physiology and oxidative damage of liver and skeletal muscle in corticosterone-treated (CORT) or vehicle (VEH) dams. Data shown are least-squared means ± s.e. Maternal death date and mass were included as covariates and interactions between groups were explored. The p-values are given for all comparisons and significant findings are reported in bold.

	VEH	CORT	Statistics				
	$\bar{x}$+se	$\bar{x}$+se	num DF	den DF	Main effect/Int-eraction	F-value	p-value
LIVER:							
Citrate synthase (nM/min/mg protein)	1.95±0.13	1.77±0.12	1	24	Group	0.95	0.35
H_2O_2 emission (pmols/mg protein/min)	34.5±4.1	34.9±3.8	1	24	Group	-0.065	0.95
Complex I substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	124±8.8	137±7.6	1	23	Group	-1.03	0.31
State 4 respiration (nmole O_2/mg/ protein/min)	25.7±2.0	29.4±1.8	1	24	Group	-1.24	0.23
Respiratory control ratio (state 3/state 4)	5.23±0.36	4.86±0.33	1	24	Group	0.69	0.50
Complex II substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	226±11.6	224±10.5	1	24	Group	0.79	0.38
State 4 respiration (nmole O_2/mg/ protein/min)	50.5±3.3	55.2±3.0	1	24	Group	-0.94	0.36
Respiratory control ratio (state 3/state 4)	4.53±0.13	4.18±0.24	1	24	Group	0.87	0.39
Oxidative damage markers							
4HNE adducts (fold-change)	0.993±0.035	0.996±0.032	1	24	Group	-0.059	0.95

Protein carbonyls (fold-change)	0.970±0.063	0.923±0.057	1	24	Group	0.49	0.63
SKELETAL MUSCLE:							
Citrate synthase (nM/min/mg protein)	1.61±0.18	1.70±0.16	1	24	Group	-0.35	0.73
H_2O_2 emission (pmols/mg protein/min)	361±54.6	168±49.7	1	24	**Group**	2.36	**0.03**
Complex I substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	351±31.5	271±28.7	1	24	Group	1.70	0.10
State 4 respiration (nmole O_2/mg/ protein/min)	81.8±8.1	65.9±7.4	1	24	Group	1.26	0.22
Respiratory control ratio (state 3/state 4)	4.45±0.48	4.39±0.44	1	24	Group	0.08	0.94
Complex II substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	449±39.9	383±36.3	1	24	Group	1.11	0.28
State 4 respiration (nmole O_2/mg/ protein/min)	167±17.1	148±15.5	1	24	Group	0.72	0.48
Respiratory control ratio (state 3/state 4)	2.70±0.13	2.67±0.12	1	24	Group	0.17	0.87
Oxidative damage markers							
4HNE adducts (fold-change)	0.984±0.032	0.995±0.029	1	24	Group	-0.26	0.83
Protein carbonyls (fold-change)	0.995±0.063	1.078±0.060	1	23	Group	-0.85	0.41

For each model: parent = random factor

Table S3. General linear mixed effect model comparisons of body mass and fecal corticosterone in the offspring of corticosterone-treated (CORT) or vehicle (VEH) mice. Offspring were evaluated at Juvenile (5.5 weeks) and Adult (10 weeks) timepoints. Body mass of offspring was measured at sacrifice. Fecal corticosterone metabolites (µg/gm fecal solid) were evaluated in Adult offspring only. Maternal death date and mass were included as covariates and interactions between groups were explored. The p-values are given for all comparisons and significant findings are reported in bold.

	VEH	**CORT**		**Statistics**			
	$\bar{x}$+se	**$\bar{x}$+se**	**num DF**	**den DF**	**Main effect/Int-eraction**	**F-value**	**p-value**
Body mass (g)	J: 16.2±0.57 A: 18.3±0.60	J: 15.6±0.52 A: 16.8±0.56	1	24	Group	1.55	0.23
Fecal CORT metabolites (µg/g)*	0.096±0.0101	0.075±0.0089	1	22	Group	2.10	0.16

For each model: parent = random factor; J = juvenile; A = adult;
*Adults only

Table S4. General linear model comparisons of mitochondrial physiology and oxidative damage of liver and skeletal muscle in the offspring of corticosterone-treated (CORT) or vehicle (VEH) dams. Data shown are least-squared means ± s.e. Maternal death date and mass were included as covariates and interactions between groups were explored. The p-values are given for all comparisons and significant findings are reported in bold.

	VEH	CORT	Statistics				
	$\bar{x}$+se	$\bar{x}$+se	num DF	den DF	Main effect/Int-eraction	F-value	p-value
LIVER:							
Citrate synthase (nM/min/mg protein)	1.03±0.046	0.97±0.043	1	24	Group	0.73	0.40
H_2O_2 emission (pmols/mg protein/min)	36.4±2.9	37.2±2.7	1	24	Group	0.033	0.86
Complex I substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	142±8.0	150±7.4	1	24	Group	0.48	0.50
State 4 respiration (nmole O_2/mg/ protein/min)	26.9±1.1	26.8±0.97	1	24	Group	0.0090	0.93
Respiratory control ratio (state 3/state 4)	5.28±0.31	5.74±0.28	1	24	Group	1.043	0.32
Complex II substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	224±8.4	226±7.8	1	24	Group	0.0097	0.92
State 4 respiration (nmole O_2/mg/ protein/min)	52.9±2.3	53.5±2.2	1	24	Group	0.030	0.86
Respiratory control ratio (state 3/state 4)	4.30±0.17	4.32±0.16	1	24	Group	0.0038	0.95
Oxidative damage markers							
Juvenile:							
4HNE adducts (fold-change)	1.05±0.034	0.97±0.032	1	22	Group	2.48	0.13

Protein carbonyls (fold-change)	1.04±0.056	0.95±0.051	1	23	Group	1.23	0.28
Adult:							
4HNE adducts (fold-change)	1.047±0.036	0.95±0.033	1	23	Group	3.26	0.08
Protein carbonyls (fold-change)	1.04±0.044	0.94±0.041	1	23	Group	2.41	0.13
SKELETAL MUSCLE:							
Citrate synthase (nM/min/mg protein)	1.38±0.14	1.51±0.13	1	24	Group	0.42	0.52
H_2O_2 emission (pmols/mg protein/min)	J: 247±47.7 A: 398±39.0	J: 289±34.2 A: 209±45.6	1	70	**Group ×Age**	14.39	**0.0003**
Complex I substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	382±16.8	377±15.2	1	24	Group	0.056	0.81
State 4 respiration (nmole O_2/mg/ protein/min)	J: 67.7±7.0 A: 100.2±5.5	J: 79.4±5.0 A: 77.9±6.5	1	67	**Group ×Age**	16.56	**0.0001**
Respiratory control ratio (state 3/state 4)	4.81±0.30	4.96±0.28	1	24	Group	0.12	0.73
Complex II substrates							
State 3 respiration (nmole O_2/mg/ protein/min)	J: 452±29.4 A: 507±25.1	J: 486±21.7 A: 440±28.5	1	67	**Group ×Age**	6.18	**0.02**
State 4 respiration (nmole O_2/mg/ protein/min)	J: 157±13.2 A: 211±10.9	J: 173±9.6 A: 155±12.4	1	67	**Group ×Age**	18.58	**0.0001**
Respiratory control ratio (state 3/state 4)	J: 2.93±0.10 A: 2.45±0.089	J: 2.84±0.077 A: 2.73±0.10	1	66	**Group ×Age**	6.10	**0.02**
Oxidative damage markers							
Juvenile:							
4HNE adducts (fold-change)	1.00±0.062	0.98±0.057	1	22	Group	0.039	0.85

Protein carbonyls (fold-change)	1.01±0.046	0.973±0.042	1	21	Group	0.39	0.54
Adult:							
4HNE adducts (fold-change)	1.03±0.032	0.995±0.029	1	23	Group	0.624	0.44
Protein carbonyls (fold-change)	1.07±0.049	0.91±0.045	1	23	**Group**	4.64	**0.04**

For each model: parent = random factor; J = juvenile; A = adult