Supplementary Information

Urolithin-A attenuates neurotoxoplasmosis and alters innate response towards predator odor

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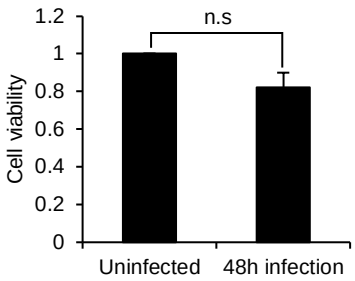
Content:

This file contains the Supplementary Figures S1-2 and the accompanying figure legends.

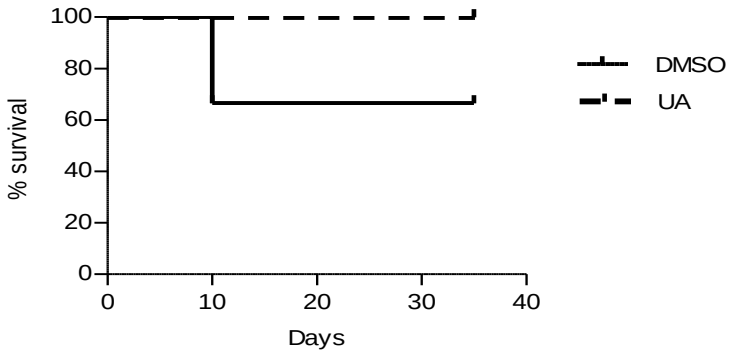
Supplementary Figure 1



48h *T. gondii* infection does not induce significant cell death in differentiated neural cells. Differentiated ReNcell were infected with wild-type Pru *T. gondii* for 48h or left uninfected. MTS assay was performed to examine cell viability. All values are mean ± S.E.M (n=3). Difference against uninfected cells was not significant (n.s).

Supplementary Figure 2

a



b

Table depicting the $2^{\Delta Ct}$ values of *SAG1* expression in infected mice. Majority of the *SAG1* expression in the DMSO and UA experimental animals were lower than the uninfected control animal.

	Animal Identity	$2^{\Delta Ct}$	~ Fold change (against uninfected)
	Uninfected	1.09e-4	1
	Positive control	1.47	> 10,000
Experimental animals	DMSO #1	6.14e-5	< 1
	DMSO #2	4.43e-7	< 0.01
	DMSO #3	1.69e-6	< 0.1
	DMSO #4	7.13e-6	< 0.1
	UA #1	6.65e-7	<0.01
	UA #2	8.81e-4	8
	UA #3	3.13e-5	< 1
	UA #4	1.50e-6	< 0.1
	UA #5	1.03e-4	< 1
	UA #6	7.55e-5	< 1

Mortality and *SAG1* expression in *T. gondii* infected mice. (a) Survival graph of the DMSO and UA-injected mice throughout the experimental duration. 6 DMSO and 6 UA mice were present at the start of the experiment. (b) *SAG1* mRNA levels in the DMSO and UA-injected experimental animals that survived till the end of the experiment, normalized against the *GAPDH* mRNA and expressed as fold-change against the uninfected control. The positive control animal was acutely infected with type II *Pru T. gondii* strain. Here, it is used as a control to show that the *SAG1* qPCR amplification was working.