

Table S2 Host pathways altered by *T. gondii* infection

Target	Findings
Apoptosis	Infection of human foreskin fibroblasts (HFF) with type I RH strain parasites blocks multiple pathways of activating apoptosis ¹ .
	Infection of human HL-60 or U937 cells with the type II strain NTE blocks apoptosis induced by actinomycin D, TNF- α , and cyclohexamide ² .
	Infection of NIH 3T3 cells with type I RH strain parasites blocks caspase activation and inhibits apoptosis ³ .
Cell cycle	Infection of HFF cells with the type I RH strain parasites stimulates G1/S transition followed by G2/M arrest, associated with sustained ERK activation ⁴ .
	Infection of human trophoblast (BeWo) and normal human dermal fibroblasts (NHDF) with the type I RH strain parasites results in G2 arrest via cyclin B1 down regulation ⁵ .
DC maturation and trafficking	Type II PRU strain parasites preferentially infect immature murine bone-marrow derived DCs and blocks maturation induced by TLR ligands or CD40L ⁶ .
	Infection of human monocyte-derived or murine bone marrow-derived DCs with type I RH strain parasites enhances motility and barrier migration <i>in vitro</i> , and <i>in vivo</i> dissemination in the mouse with DCs pulsed with the type II strain parasite PTG ⁷ .
	DC are activated and expand in response to type II strains but this is reduced in Type I strains ⁸ .
Host gene transcription	Infection a clone of the type II strain ME49 induces transcription of many genes in HFF cells, including immune response pathways, glycolysis, and lipid metabolism ⁹ ; similar responses were seen in HFF cells infected with the type I RH strain ¹⁰ .
	Infection with type I RH strain induces Hif1a in HFF cells and this response is important for parasite growth under hypoxic conditions ¹¹ .
	Induction of host EGR2 transcriptional response is due to rhoptry discharge following infection of HFF cells by the type I RH strain ¹² .
	Differential expression of host cell transcriptional responses during infection of HFF cells by type II and III strain parasites drives development of immune responses including STATs and IL-12 ¹³ .
P38, JNK, ERK1/2	Infection of human THP-1 monocyte cell line with type I strain RH induces phosphorylation of ERK1/2, P38, and JNKs ¹⁴ .
	Transient activation of MAPK pathways in murine bone marrow derived macrophages cells infected with the type I RH strain is followed by suppression of LPS triggered responses ¹⁵ .
	Soluble tachyzoite antigen from type I RH strain activates MAPK through TRAF6 that are required for IL-12 production ¹⁶ .
NF κ B	Proinflammatory cytokines triggered by LPS are blocked by inhibition of nuclear translocation of NF κ B in response to infection with type I RH strain ¹⁷ .
	The ability of LPS to induce NF κ B activation is blocked by infection of bone marrow derived murine macrophages infected with type I RH strain ¹⁸ .
IL-12	IL-12 production is strongly induced by type II, but not type I or III strains in murine bone marrow derived and peritoneal macrophages ¹⁹ .
	Infection of bone marrow derived murine macrophages with type II strain ME49 induces higher levels of IL-12 than infection with type I RH strain ²⁰ .
IFN- γ	Induction of gene expression by treatment of HFF cells with IFN- γ is blocked by infection with each of three strain types (I,II, III) ²¹ .
	Induction of MHC I and MHC II by treatment of murine bone marrow derived macrophages cells with IFN- γ is blocked by infection with type II NTE strain ²² .
	Infection of murine bone marrow derived macrophages or RAW264 macrophages with the type II NTE strain down regulated induction of inducible nitric oxide by treatment with IFN- γ ²³ .

	Infection of RAW264 macrophages with type I RH and type II ME49 strain parasites inhibited upregulation of MHC II by IFN- γ , but only type I RH strain parasites were able to block induction of nitric oxide production ²⁴ .
STAT1	Infection of bone marrow derived macrophages with type II NTE strain blocked IFN- γ induced transcriptional changes by altering chromatin and disrupting STAT1 binding to nuclear promoters ²⁵ .
SOCS-1	Induction of SOCS-1 expression contributes to blocking of IFN- γ signaling through STAT1 in murine BMM and RAW264 macrophages infected with type I BK strain parasites ²⁶ .
	SOCS1 expression in RAW264 macrophages cells is upregulated during infection with type I strain but not type II ME49 strain parasites; this pathway independent of TLR signaling but is regulated by EGR2 and p38 Map kinase ²⁴ .
SOCS-3	Infection with type I and II strains induces SOCS3 that limits IL-6 signaling and protects from immune pathology ²⁷ .
TNF- α	Infection with type I RH strain suppresses TNF- α mediated transcription in murine BMM and this occurs through chromatin modification ²⁸ .
STAT3	Infection with live type I RH strain, but not treatment with lysates, induces STAT3 activation in murine bone marrow derived macrophages and suppresses IL-12 and TNF- α ²⁹ .

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