p62 plays a specific role in interferon-γ-induced presentation of a Toxoplasma vacuolar antigen.

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p62 (also known as Sqstm1) is a selective autophagy adaptor with an ubiquitin-binding domain. However, the role of p62 in the host defense against *Toxoplasma gondii* infection is unclear. Here, we show that interferon γ (IFN-γ) stimulates ubiquitin and p62 recruitment to *T. gondii* parasitophorous vacuoles (PVs). Some essential autophagy-related proteins, but not all, are required for this recruitment. Regardless of normal IFN-γ-induced *T. gondii* clearance activity and ubiquitination, p62 deficiency in antigen-presenting cells (APCs) and mice diminishes the robust IFN-γ-primed activation of CD8+ T cells that recognize the *T. gondii*-derived antigen secreted into PVs. Because the expression of Atg3 and Irgm1/m3 in APCs is essential for PV disruption, ubiquitin and p62 recruitment, and vacuolar-antigen-specific CD8+ T cell activation, IFN-γ-mediated ubiquitination and the subsequent recruitment of p62 to *T. gondii* are specifically required for the acquired immune response after PV disruption by IFN-γ-inducible GTPases.