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Advances in murine models for studying parasite immunology.

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Introduction

Murine models have long stood as the cornerstone for unraveling the complexities of immune responses to parasitic infections. Their genetic tractability, short reproductive cycles, and similarity to human immune mechanisms make them ideal tools for probing host-parasite interactions. With the advent of cutting-edge techniques and refined transgenic mouse lines, researchers have expanded the utility of murine models in parasite immunology, offering nuanced insights into pathogenesis, immunity, and therapeutic development [1, 2].

Tools like CRISPR/Cas9 and Cre-lox systems enable precise gene editing to evaluate host factors in immunity. Key components of the adaptive and innate immune systems mirror those in humans, including T and B cells, macrophages, and cytokine signaling. Controlled infections allow reproducible studies of dose-response relationships and immune kinetics. Rodent malaria models like *Plasmodium berghei*, *P. chabaudi*, and *P. yoelii* have elucidated mechanisms of: The *P. berghei ANKA* model has been pivotal in identifying $CD8^+$ T-cell involvement in cerebral malaria and endothelial dysfunction [3, 4].

Used to delineate Th1 vs Th2 roles, Deficient in T and B cells, vital for studying innate immunity and adoptive transfers. Allow Treg-specific investigations via diphtheria toxin administration. These models enable exploration of cell-specific contributions, cytokine dependencies, and immunopathology. Modern imaging tools have transformed murine studies: Allows real-time visualization of immune cell trafficking during infection [5, 6].

Enable non-invasive tracking of parasite burden and distribution. Reveals transcriptomic heterogeneity of immune cells post-infection. To bridge translational gaps, humanized mice with human immune components offer novel perspectives: Used to study *Plasmodium falciparum* infection and immune clearance**.** Reveal human-specific cytokine responses and pathology. Despite their promise, limitations include incomplete immune maturation and restricted tissue distribution [7, 8].

Murine models, while powerful, have constraints: Not all human-parasite dynamics are faithfully replicated. Variations in cytokine networks and cell markers can skew interpretation. Many human parasites lack natural infectivity in mice without modification. Therefore, complementary models (e.g., nonhuman primates, organoids) and cross-validation are essential. Murine models continue to evolve: Gnotobiotic mice illuminate how gut flora modulate parasite immunity. Fuse 3D human tissue systems with murine hosts for dual-layer insights [9, 10].

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