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Journal Name:	International Journal of TROPICAL DISEASE & Health
Manuscript Number:	Ms_IJTDH_29371
Title of the Manuscript:	Variation of Mice Immune Responses After Infection with Lethal Strain Plasmodium yoelii: Role of Inflammatory and Anti-inflammatory Cytokines
Type of the Article	Original Research Article

General guideline for Peer Review process:

This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of '**lack of Novelty**', provided the manuscript is scientifically robust and technically sound.

To know the complete guideline for Peer Review process, reviewers are requested to visit this link:

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PART 1: Review Comments

	Reviewer's comment	Author's comment (if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)
Compulsory REVISION comments		
Minor REVISION comments	Page 14, Figure 6, histology: this figure would benefit from specific labelling (e.g., arrows pointing to lymphocyte infiltration in Panel F) and more description on the nature of the liver cell damage (b,c,e,f). Many readers may not be familiar enough with liver histology to note the differences.	Arrows is added to fig.6 please check the text. Description of liver cell damage was added in the text, highlighted by yellow colour (line 226-230).
Optional/General comments	This is a well-written paper seeking to elucidate the relationship between infection severity and mouse strain-specific cytokine production. The comments that follow are suggestions that might improve the mss, but are not required. p.2, line 35: the reference cited surely is not the most up -to-date; results from the RTS,S vaccine trials would be more relevant. Page 3, line 64-66: The term TCR^{int} does not appear to be much used in recent literature. Could the Authors expand upon what is meant? Ther is an intriguing paper by Hebesian, 2013 (DOI:10.3389/fimmu.2913.00154) arguing that low affinity T-cell clones may be more important for some kinds of responses. Page 10, Figure 2: it is unfortunate that there were no time points between Day 7 and Day 20 to more fully	TCR^{int} is an intermediate T cells considered as unconventional T cells expanded after malaria infection and have compensatory function to conventional T cell which have TCR^{high} and decrease in number due to thymic atrophy, This issue was well discussed in our previous work. Reference 11, 12. The authors did the kinetics of parasitemia in Figure 1 to identify the acute stage and severe stages for B6 mice and acute phase, peak and recovery phases for DBA2 mice. So in figure 2 and the rest of figures, the authors paid their full attention about the changes occur in these important phases.



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	characterize the differences between these strains. Page 15, line 246: there is a 2007 paper by Charavarty and coworkers (DOI: 10.1038/um1628) that discusses IFNγ in infected BALB/c mice which might add to the discussion. Page 16, line 259: do the Authors mean macrophages or dendritic cells? Page 18, line 298: the induction of auto antibodies has been noted previously, but is an intriguing observation in that it suggests a loss of tolerance similar to that observed with of check point inhibitors in treatment of certain cancers. It would be interesting to understand (speculate!) how the timing of this might affect outcome of disease/limitation of disease severity in the case of malaria.	In discussion the authors mentioned the production of cytokine (IFNγ and TNF alpha) in different strains of mice like susceptible A/J mice after infection with Plasmodium chabaudi AS Reference 32 line 260, 261 The authors meant macrophages according to reference 34 The production of auto-antibodies in malaria occurred because plasmodium is considered as an intracellular organism, and auto-antibodies are a form of innate immunity which are stimulated in malaria infection and have an important role in malaria infection. Authors' comments The authors would like to thank the reviewer for the nice and valuable comments.
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