Review of: "Aspirin-triggered resolvin D1 reduces parasitic cardiac load by decreasing inflammation through N-formyl peptide receptor 2 in a chronic murine model of Chagas disease"

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The research article entitled "Aspirin-triggered resolvin D1 reduces parasitic cardiac load by decreasing inflammation through N-formyl peptide receptor 2 in a chronic murine model of Chagas disease" by Ileana Carrillo et al., investigates the role of Aspirin-triggered resolvin D1 (AT-RvD1) as a pro-resolving mediator of inflammation that acts through N-formyl peptide receptor 2 (FPR2) using a FPR2 knockout murine model of chronic *Trypanosoma cruzi* infection. The data provided in the manuscript is important and interesting in connecting a link between AT-RvD1 as an attractive therapeutic activity due to its regulatory effect on the inflammatory response at the pathophysiology to cardiac pathology during *T. cruzi* infection. The manuscript is an original research article and I recommend it, although it suffers with the following minor concerns:

Materials and methods section

1. Explain the rationale for the use of Dm28c strain of *T. cruzi*. What is the rationale to select this strain and provide more data concerning its virulence and susceptibility level to benznidazol?

Results and discussion sections

1. Include the data of eletrocardiographic variables in the manuscript, even in the absence of diferences among the groups of mice. Discussion about eletrocardiography abnormalites in Chagas cardiomyopathy needs to explore more parameters than only QT interval, for example: PR interval (iPR) and atrial block, axis of QRS, evidences of atrial fibrillation and right bundle branch block.