Reviewer's report

Title: A redundant role for dectin-1 in experimental colitis models.

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Reviewer: Satish Keshav

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Review of: A redundant role for dectin-1 in experimental colitis models
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When assessing the work, please consider the following points:

1. Is the question posed by the authors well defined? Yes
2. Are the methods appropriate and well described? Yes
3. Are the data sound? Yes
4. Does the manuscript adhere to the relevant standards for reporting and data deposition?
5. Are the discussion and conclusions well balanced and adequately supported by the data? Mainly yes, see below
6. Are limitations of the work clearly stated? Yes
7. Do the authors clearly acknowledge any work upon which they are building, both published and unpublished? Yes
8. Do the title and abstract accurately convey what has been found? See below
9. Is the writing acceptable? Yes

Comments (all are discretionary revisions):

Overall this is a well-conducted study, concentrating on two out of many possible models of intestinal inflammation. There seems to be little or no effect of genetic deficiency of dectin-1 in these models.

Title and abstract:

The term “redundant role” implies that there is some effect that is possibly compensated for. At the moment, however, this is speculative. An alternative title that describes the main findings might be: “Genetic deletion of dectin-1 does not affect the course of murine experimental colitis”

Results:

Controls for immunohistochemistry, such as isotype-matched antibody, and dectin-1 -/- should be shown and mentioned in legend.
The TNF response by isolated macrophages to fecal extracts is much lower than then IL-10 response, and this difference is not seen with zymozan. This merits some discussion.

In figure 3, the levels of IL-10 are much lower in dectin1 -/- than wild-type, although not statistically significant. In two instances, the levels are zero. This seems like an experiment to repeat to see if indeed there is some biological effect.

Discussion:

The antigens that elicit ASCA reactivity are not definitively known, although Candida albicans is suspected/assumed, see, for example: Gastroenterology. 2006 May;130(6):1764-75.


The summary statement “we showed that dectin-1 is able to induce a cytokine response towards mouse faeces” is based on indirect evidence, i.e. that the genetic absence results in change in the response of macrophages in vitro, rather than a formal demonstration, for instance by blocking dectin-1 signalling, and probably should be modified. For the rest, the conclusion that this does not substantially affect at least two models of intestinal inflammation seems fair, and is an important contribution to the literature on the subject.

**Level of interest:** An article whose findings are important to those with closely related research interests

**Quality of written English:** Acceptable

**Statistical review:** No, the manuscript does not need to be seen by a statistician.

**Declaration of competing interests:**

I declare that I have no competing interests