Reviewer's report

Title: Effects of short-term fructooligosaccharide intake on equol production in Japanese postmenopausal women consuming soy isoflavone supplements: A pilot study

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Reviewer: Michael Hall

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Yuko Tousen et al.

Minor Essential Revisions

Abstract
Page 2, line 3: Recent studies suggest that [some of] the clinical effectiveness…
Page 2, line 5: Equol….is produced in the GIT by [certain] intestinal microflora [where present].

Introduction
Page 3, line 5: I suggest replacing ‘suggesting oestrogenic activity..’ with ‘eliciting mild oestrogenic-like activity..’
Page 3, line 8: The clinical effectiveness of isoflavones cannot be solely attributed to their ability to be converted to equol, as not every isoflavone is converted to equol.
Page 3, line 18: Suggest that the sentence reads ‘Thus, FOS has a prebiotic activity as well as being a dietary fibre’.
Page 3, line 19: suggest insert the word ‘certain’ before ‘Lactobacillus and Bifidobacteria species’

Methods
Page 4, line 8: Suggest: [Equol producer: Log (urinary equol / daidzein) > -1.70]
Page 4, line 10: Delete ‘of’
Page 4, line 17: Justify use of sucrose as control. Digestive products may stimulate growth of certain microflora, while FOS may selectively stimulate the growth of certain (different) microflora.

Results and Discussion:
Page 5, line 15: The similarly increased serum equol concentrations in FOS and Control groups among equol producers may have been stimulated by the isoflavone supplement. It would have been interesting to determine if a
non-carbohydrate used as control influenced equol production.

Page 5, line 20: It is perhaps more correct to state that for equol non-producers FOS intervention did not affect serum equol production compared to Sucrose...

Major Compulsory Revisions

Page 6, line 9: This is a critical statement. What is the typical isoflavone load in the Japanese diet, and with respect to it, what is the relative contribution of 25mg of isoflavones to this diet. If the Japanese diet contains sufficient isoflavones to saturate equol producer capacity, then the supplement cannot elicit a response. Conversely, where a diet had low isoflavone content, the same experiment may provide quite different results. This point must be emphasised in the paper.

Page 6, line 19: It is evident that the equol producers were hosts to microflora that did not respond to the intervention. It holds that ‘non-producers’ remain non-producers due to the specific microflora in their gastrointestinal tract. Conversion of non-producers to producers would appear to require a change in gastrointestinal microflora, rather than an increase in the existing population, which was not elicited by provision of FOS as a dietary supplement.

Page 7, line 2: I suspect the 2-week study was sufficient to determine what response could be elicited by the intervention. The limitations of the intervention are the treatments and the non-control of dietary isoflavones in addition to those provided in the supplement. This must be discussed in the paper. A very different response may have been elicited in subjects on a typical ‘western’ diet.

Conclusion:

Further conclusions are necessary. The final statement contends that it may be possible to stimulate equol production by dietary conditions (alone). The possibility of microflora supplementation should be mentioned.

**Quality of written English:** Needs some language corrections before being published

**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.